Past Gyne/Obs Summary

Obstetrics

I. Physiological Changes in Pregnancy (Dr. Oqba)*(Q49, 66, 100, 101, 108, 154, 225, 271, 331, 431, 456, 526, 531, 556, 613, 624, 660, 694, 695, 696, 725, 726, 727, 789, 791)*

Pregnancy isn't just a growing belly; it's a systemic overhaul. If you don't get these changes, you won't understand the complications.

- **Cardiovascular System – The Engine Ramps Up:**
  - **Cardiac Output (CO) INCREASES:** By 30-50%. This is crucial. It starts early and peaks mid-pregnancy. *(Q49E, Q225A implies CO increases, which is true)*.
  - **Heart Rate (HR) INCREASES:** By 10-20 bpm. *(Q49A is correct)*.
  - **Stroke Volume (SV) INCREASES:** Especially early on.
  - **Systemic Vascular Resistance (SVR) DECREASES:** Progesterone is a vasodilator! This is why BP often drops slightly in the 2nd trimester. *(Q49D says peripheral resistance increases – this is DEAD WRONG for most of pregnancy, only rising back towards term or in pathology like pre-eclampsia)*.
  - **Blood Pressure (BP):** Typically drops in the first and second trimesters (lowest mid-pregnancy), then gradually returns to pre-pregnancy levels by term. *(Q108C correctly states the nadir is around 20 weeks. Q100A is wrong to say BP increases above pre-pregnancy levels in the 3rd trimester as a normal finding; it returns to normal, or increases pathologically)*.
  - **Blood Volume INCREASES:** Plasma volume expands more than red cell mass, leading to **physiological anemia**. *(Q49E refers to increased blood volume, which is true)*.
  - **Physical Exam Findings:** Expect an ejection systolic murmur (due to increased flow), exaggerated S1 split, and a third heart sound (S3). A diastolic murmur is ALWAYS pathological. *(Q331E nails this - diastolic murmur is NOT normal)*.
  - **Supine Hypotension Syndrome:** The gravid uterus compresses the IVC when supine, reducing venous return, CO, and BP. Advise left lateral tilt. *(Q449E correctly identifies IVC compression as the cause)*.
- **Respiratory System – Breathing for Two (Sort Of):**
  - **Tidal Volume (TV) INCREASES:** Deeper breaths, not necessarily faster (initially).
  - **Minute Ventilation INCREASES:** Due to increased TV.
  - **Respiratory Rate:** May increase slightly or stay the same. *(Q225C, Q456C says RR increases – this is a subtle point; TV increase is more significant, but a slight RR increase can occur. However, Q154D states RR is unchanged, which is also a common teaching point. Focus on the increased minute ventilation driven by progesterone acting on the respiratory center)*.
  - **Functional Residual Capacity (FRC) DECREASES:** Uterus pushes the diaphragm up. *(Q154C is correct, FRC decreases)*. Residual Volume (RV) also decreases *(Q154A is correct)*.
  - **Chronic Respiratory Alkalosis:** Progesterone stimulates the respiratory center, leading to hyperventilation, ↓PaCO2, and compensatory renal excretion of bicarbonate. pH is slightly alkalotic.
- **Hematological System – Diluted and Sticky:**
  - **Plasma Volume ↑↑↑, Red Cell Mass ↑:** Leading to dilutional "physiological anemia." Hb/Hct drop. *(Q431E implies this expansion)*.
  - **Iron Requirements INCREASE:** Fetus and placenta are greedy. Iron deficiency is common. *(Q81A states iron demand increases, which is true)*.
  - **White Cell Count (WCC) INCREASES:** Mild leukocytosis is normal. *(Q100E, Q225D, Q456D are correct)*.
  - **Platelets:** May decrease slightly (gestational thrombocytopenia) but usually stay within normal range.

- **Coagulation Factors INCREASE:** Pregnancy is a **hypercoagulable state** (↑fibrinogen, factors VII, VIII, IX, X, von Willebrand). This is protective against PPH but increases VTE risk. *(Q613: Factors II, V, IX, XII all increase. Factor XI (Q613D) and XIII tend to DECREASE or stay the same – so this is the likely exception).*
- **Erythrocyte Sedimentation Rate (ESR) INCREASES:** Due to increased fibrinogen. *(Q725E is correct).*

- **Renal System – Working Overtime:**
  - **Renal Blood Flow (RBF) and Glomerular Filtration Rate (GFR) INCREASE:** By up to 50%. This leads to ↓BUN and ↓Creatinine. *(Q531A, Creatinine clearance increases, is correct).*
  - **Ureters & Renal Pelvis DILATE:** (Hydronephrosis/hydroureter) due to progesterone (smooth muscle relaxation) and mechanical compression by the uterus, more common on the right. This increases risk of pyelonephritis. *(Q556E, ureteric dilatation, is correct).*
  - **Glycosuria:** Common due to increased GFR overwhelming tubular reabsorption capacity. *(Q531C, glycosuria decreases, is WRONG).*
  - **Proteinuria:** Small amounts can be normal, but significant proteinuria is a red flag.
- **Gastrointestinal System – Slow and Sluggish:**
  - **Progesterone relaxes smooth muscle:** Leading to ↓esophageal sphincter tone (reflux - Q101C), ↓gastric motility (delayed emptying - Q225E), and ↓bowel motility (constipation). *(Q101A, increased transit time, is WRONG – it's decreased motility leading to longer transit time).*
  - **Nausea/Vomiting ("Morning Sickness"):** Very common, related to hCG.
  - **Ptyalism (excessive salivation):** Can occur.
  - **Dental Caries:** Increased incidence is mentioned (Q101B), possibly due to changes in saliva pH or dietary habits.
- **Metabolic & Endocrine Changes:**
  - **Insulin Resistance INCREASES:** Due to human placental lactogen (hPL), progesterone, cortisol. This ensures adequate glucose for the fetus. This is the basis for Gestational Diabetes. *(Q66A states relative insulin resistance is normal in late gestation - this is TRUE. Q271B correctly identifies hPL as the main diabetogenic factor).*
  - **Thyroid:** TBG increases (estrogen effect), so Total T4/T3 increase, but Free T4/T3 remain normal. TSH may slightly decrease in 1st trimester (hCG cross-reactivity) then normalizes. *(Q621E, TSH does not cross placenta, is correct).*
  - **Basal Metabolic Rate (BMR) INCREASES.**
  - **Weight Gain:** Average 12.5kg (Q66D).
  - **Calcium Metabolism:** Increased intestinal absorption of calcium (Q66E). Maternal total calcium may decline due to hemodilution and decreased albumin (Q727A), but ionized calcium (the active form) remains unchanged (Q727C is correct). PTH levels increase to meet fetal demand (Q727D is correct). Calcitonin levels also rise (Q727B).
  - **Albumin Production:** May decrease (Q101D).
  - **Hormones:** hCG, hPL, estrogen, progesterone all skyrocket from placental/corpus luteum origin. Aldosterone (Q791A), Estriol (Q791B), MSH (Q791C), hPL (Q791D) all increase. Albumin (Q791E) decreases or stays same due to hemodilution.

II. Obstetrics Ultrasound Scan (Dr. Asmaa)*(Q7, 58, 158, 247, 654)*

Ultrasound is our window to the womb. Knowing *when* and *why* is key.

- **First Trimester Scan (Dating & Viability):**
  - **Timing:** Typically 8-14 weeks.
  - **Purpose:**
    - **Confirm viability:** Fetal heart beat (detectable by transvaginal US around 5.5-6 weeks - *(Q7A, 6 weeks is correct)*; by transabdominal US a bit later).
    - **Accurate dating: Crown-Rump Length (CRL)** is the most accurate biometric parameter for dating in the 1st trimester. *(Q58A, first trimester CRL is the best for dating, is correct).*
    - **Determine number of fetuses & chorionicity/amnionicity** in multiples.

    - **Detect major structural abnormalities** (e.g., anencephaly).
    - **Nuchal Translucency (NT) screening:** (11-13+6 weeks) for aneuploidy risk assessment, often combined with maternal serum markers.
- **Second Trimester Scan (Anomaly Scan):**
    - **Timing:** Typically 18-22 weeks. *(Q158, measurement of choice for GA at 9 weeks is CRL, is correct - though this question also fits 1st trimester).*
    - **Purpose:** Detailed anatomical survey to detect fetal structural abnormalities. This is your "Level II scan" often mentioned *(Q58E refers to measurements at Level II scan).* Assess placental location, amniotic fluid volume.
- **Third Trimester Scan (Growth & Wellbeing):**
    - **Purpose:** Assess fetal growth (biometry: BPD, HC, AC, FL), estimate fetal weight, assess amniotic fluid volume (AFI), placental location, and fetal wellbeing (Biophysical Profile, Doppler studies if indicated). *(Q247 refers to scanning a low placenta at 20 weeks – the advice is that most migrate up).*
- **Biophysical Profile (BPP):** Assesses fetal wellbeing. Components (each scores 0 or 2, max 10):
    1. Fetal breathing movements
    2. Gross body movements
    3. Fetal tone
    4. Amniotic fluid volume (AFI or single deepest pocket)
    5. Non-Stress Test (NST) - reactive CTG

    (Q69, BPP components, is testing this. Fetal rapid eye movements (Q69C) are NOT a standard component).
- **Doppler Ultrasound:**
    - **Umbilical Artery Doppler:** Assesses placental resistance. Abnormal (absent or reversed end-diastolic flow - AEDF/REDF) indicates placental insufficiency and fetal compromise.
    - **Middle Cerebral Artery (MCA) Doppler:** Assesses for fetal anemia (increased peak systolic velocity - PSV) or fetal hypoxia/compromise (cerebral redistribution/brain-sparing effect – decreased pulsatility index).

III. Preterm Labor (Dr. Shawqi)*(Q14, 28, 38, 79, 123, 190, 242, 285, 287, 317, 365, 368, 574, 671, 751, 756)*

Labor before 37 weeks. Major cause of neonatal morbidity/mortality.

- **Definition:** Regular uterine contractions PLUS cervical change (dilatation and/or effacement) at <37 weeks gestation.
- **Risk Factors:** Previous preterm birth (strongest!), multiple gestation *(Q28A correct)*, infections (UTI, BV - *Q14D implies UTI is a major risk factor, true*), cervical incompetence, uterine anomalies, polyhydramnios, smoking, low socioeconomic status, short interpregnancy interval.
- **Diagnosis:**
    - Clinical: Contractions + cervical change.
    - **Fetal Fibronectin (fFN):** A "glue-like" protein. If present in cervicovaginal secretions between 22-34 weeks, it indicates increased risk of PTL. High negative predictive value (NPV) is its main strength – if negative, PTL is unlikely in the next 1-2 weeks. *(Q38: "Considered abnormal if detected before 20 weeks" (Q38B) is INCORRECT; it's normally present before 20 weeks and after 34-36 weeks. Its utility is detection between 22-34 weeks. "Predicts 40% of patients at risk" (Q38C) is too high for positive predictive value; NPV is better. "Negative testing reduce risk to <1%" (Q38E) is a strong claim, but NPV is indeed high).*
    - **Transvaginal Ultrasound (TVUS) for Cervical Length:** Short cervix (<25mm, or even <15mm) is a significant risk factor.
- **Management Aims:**
    1. Delay delivery to allow for corticosteroid administration.
    2. Administer corticosteroids for fetal lung maturity.

3. Administer Magnesium Sulfate for fetal neuroprotection (if <32 weeks).
4. Transfer to a center with NICU capabilities if needed.
5. Treat underlying causes if identified (e.g., UTI).

- **Key Interventions:**
  - **Corticosteroids (Betamethasone or Dexamethasone):** CRUCIAL. Given to mother IM. Accelerates fetal lung maturation, reduces RDS, IVH, NEC. Benefit greatest if delivery is >24h but <7 days after administration. Indicated usually between 24-34 weeks (sometimes up to 36+6). *(Q287E refers to uterine stretch/multiple gestations as a cause of PTL - True)*.
  - **Tocolytics:** Drugs to suppress uterine contractions. Aim to delay delivery for 48h to allow steroids to work.
    - **Beta-mimetics (e.g., Ritodrine, Terbutaline):** Many side effects (tachycardia, hyperglycemia, hypokalemia, pulmonary edema). Less used now.
    - **Magnesium Sulfate:** Also used for neuroprotection. Side effects: flushing, lethargy, respiratory depression. Check reflexes, urine output. Calcium gluconate is antidote. *(Q79B β-Adrenergic antagonist is WRONG – agonists are tocolytics. Antagonists would worsen things)*.
    - **Calcium Channel Blockers (e.g., Nifedipine):** Commonly used. Side effects: hypotension, headache, flushing. *(Q368C Calcium channel blockers are listed as safe tocolytics)*.
    - **NSAIDs (e.g., Indomethacin):** Prostaglandin synthetase inhibitor. Risk of premature closure of ductus arteriosus (especially if used >32 weeks or for >48-72h), oligohydramnios. *(Q79C Prostaglandin synthetase inhibitors are tocolytics)*.
    - **Oxytocin Antagonists (e.g., Atosiban):** Fewer side effects but expensive. *(Q368D Oxytocin receptor antagonists are tocolytics)*.
    - **Contraindications to Tocolysis:** Severe pre-eclampsia/eclampsia, chorioamnionitis, significant APH, fetal demise, lethal fetal anomaly, mature fetus, non-reassuring fetal status.
  - **Magnesium Sulfate for Neuroprotection:** Given if delivery is anticipated <32 weeks. Reduces risk/severity of cerebral palsy.
  - **Antibiotics for GBS Prophylaxis:** If GBS status unknown or positive.
- **Preterm Premature Rupture of Membranes (PPROM):** ROM <37 weeks before labor onset. Management depends on GA:
  - Expectant management with antibiotics (to prolong latency and reduce maternal/neonatal infection) and corticosteroids if <34 weeks.
  - Delivery often considered around 34 weeks or if signs of infection/fetal compromise. *(Q190 discusses PPROM management factors - amount of fluid lost (D) is arguably less important than GA, fetal wellbeing, and signs of infection)*.
- **Prognosis:** Pulmonary immaturity and intraventricular hemorrhage are major causes of morbidity/mortality *(Q14E correctly states this)*.

IV. Gestational Trophoblastic Diseases (GTD) (Dr. Naser)*(Q22, 57, 115, 141, 161, 224, 290, 323, 329, 416, 455, 535, 669, 707)*

A spectrum of proliferative disorders of placental trophoblast. Think HIGH hCG.

- **Spectrum:**
  - **Hydatidiform Mole (Molar Pregnancy):**
    - **Complete Mole:** Diploid, entirely paternal in origin (e.g., empty ovum fertilized by one sperm that duplicates, 46XX; or two sperm, 46XX or 46XY). NO fetal tissue. Diffuse trophoblastic hyperplasia, diffuse hydropic villi ("bunch of grapes" or "snowstorm" on US). Higher risk of malignant transformation (Gestational Trophoblastic Neoplasia - GTN). *(Q323A - 46XX all paternal origin is the most common karyotype. Q161A is correct. Q115A most common chromosomal pattern 46XY is also possible, but 46XX paternal is classic. Q161B no fetal tissue, correct. Q161C 75-80% from single sperm duplication, correct. Q115C embryonic tissue often present is WRONG for complete mole)*.

        - **Partial Mole:** Triploid (usually 69XXX, 69XXY, 69XYY – ovum fertilized by two sperm or a diploid sperm). Fetal tissue IS present. Focal trophoblastic hyperplasia, focal hydropic villi. Lower risk of GTN. *(Q57A karyotype triploid, correct. Q57D embryonic tissue present, correct. Q57E uterine size large is less common than in complete mole but possible; usually small or normal for dates. The exception is Q57E if it means "always large". The question asks for "except" - focal swelling (Q57C) and focal hyperplasia (Q57B) are hallmarks).*
    - **Gestational Trophoblastic Neoplasia (GTN):** Malignant forms.
        - **Invasive Mole:** Molar tissue invades myometrium.
        - **Choriocarcinoma:** Highly malignant, metastasizes early (lungs common - Q115B). Can follow mole, normal pregnancy, abortion, or ectopic. *(Q22 Choriocarcinoma most likely after complete mole (C) is correct).*
        - **Placental Site Trophoblastic Tumor (PSTT):** Rare, from intermediate trophoblast.
- **Clinical Presentation:**
    - Vaginal bleeding (often "prune juice" like).
    - Uterus larger than dates (especially complete mole).
    - hCG levels markedly elevated (often >100,000 mIU/mL). *(Q141 refers to β-hCG 150,000 in a molar pregnancy scenario).*
    - Hyperemesis gravidarum (due to high hCG).
    - Early pre-eclampsia (<20 weeks).
    - Theca lutein cysts on ovaries (due to hCG overstimulation).
    - Hyperthyroidism (hCG can mimic TSH).
    - Passage of vesicular tissue.
- **Diagnosis:**
    - **Ultrasound:** "Snowstorm appearance" for complete mole. *(Q141, Q224B, Q455C all refer to this presentation. Q161E definitive diagnosis by TVU is correct).*
    - **Quantitative β-hCG:** Markedly elevated.
- **Management:**
    - **Suction Evacuation (D&C):** Treatment of choice for molar pregnancy. *(Q141A Suction evacuation is correct. Q336 patient with β-hCG 7500, no adnexal mass – this is tricky. If ectopic is suspected, TVUS and serial hCGs. If GTD is suspected and products are in utero, suction evacuation. Given "thickened endometrium" and no adnexal mass, a very early IUP or resolving GTD/miscarriage could be considered. Further β-hCG in 48hrs (B) is a reasonable step to assess viability/trend before jumping to D&C if diagnosis isn't molar from the start).*
    - **Hysterectomy:** Option for older women who have completed childbearing.
    - **β-hCG Follow-up:** CRITICAL. Weekly until undetectable for 3 consecutive weeks, then monthly for 6-12 months. Rising or plateauing hCG indicates GTN. *(Q416C hCG is sensitive marker for treatment response, correct).*
    - **Contraception:** Essential during follow-up (usually OCPs, avoid IUD initially).
    - **Chemotherapy for GTN:**
        - **Low-risk GTN:** Single-agent (Methotrexate or Actinomycin D). *(Q416E MTX for low-risk GTN, correct).*
        - **High-risk GTN (or metastatic):** Multi-agent chemotherapy (e.g., EMA-CO).
        - Indications for chemo after mole evacuation: Plateauing/rising hCG, hCG >20,000 >4wks post-evac, histological choriocarcinoma, evidence of metastases. *(Q416D Chemo for plateaued hCG after evacuation, correct). (Q329 Poor prognosis factors for GTN: hCG >100,000 (B), prior normal delivery (C), previous chemo (D), longer duration (E). Age younger than 39 (A) is NOT a poor prognostic factor; older age can be.*
- **Key Differentiators:**
    - **Q290 Least likely clinical data to suggest hydatidiform mole:** Bleeding 1st half (A), lower abdo pain (B), toxemia <24wks (C), hyperemesis (D) are all classic. Nervousness, anorexia, tremors (E) are symptoms of hyperthyroidism, which CAN be associated, but are less direct/common than the others.

Brutal Honesty Checkpoint:

- **Physiological Changes:** Don't just memorize lists. Understand *why* SVR drops (progesterone), *why* GFR increases (increased RBF), *why* there's physiological anemia (plasma volume > RBC mass expansion). These "whys" explain normal symptoms and predispositions to pathology.
- **Ultrasound:** Know *what* you're looking for at *each* stage. CRL for 1st tri dating is bread and butter. Anomaly scan is a critical safety net.
- **Preterm Labor:** Steroids, tocolysis (indications/contraindications), MgSO4 neuroprotection – these are non-negotiable. Understand fFN's role (high NPV).
- **GTD:** Complete vs. Partial mole differences (karyotype, fetal parts, GTN risk). hCG is your guiding star for diagnosis and follow-up. Suction D&C is the first step for moles.

Obstetrics: Navigating Hypertensive Crises, Bleeding, and the Drama of Delivery

High blood pressure in pregnancy can kill. Bleeding before delivery is an emergency. Labor is a physiological marvel but can go wrong. And sometimes, we need to help the baby out. Get these concepts nailed down.

V. Hypertensive Disorders of Pregnancy (Dr. Naser)*(Q11, 42, 70, 117, 147, 202, 249, 321, 330, 350, 383, 457, 462, 468, 549, 591, 647, 661, 662, 749, 753, 780, 783, 798)*

This isn't just "high BP." It's a spectrum from relatively benign to life-threatening. Definitions are EVERYTHING.

- **The Spectrum – Know Your Definitions COLD:**
  - **Chronic Hypertension:** BP ≥140/90 mmHg *before* pregnancy OR *before 20 weeks* gestation, or persisting >12 weeks postpartum. *(Q661B defines it correctly as before 20 weeks or pre-existing).*
  - **Gestational Hypertension:** New onset BP ≥140/90 mmHg at or *after 20 weeks* gestation, WITHOUT proteinuria or signs of end-organ damage. Resolves by 12 weeks postpartum.
  - **Pre-eclampsia (PET):** New onset BP ≥140/90 mmHg at or *after 20 weeks* gestation PLUS one or more of:
    - **Proteinuria** (≥300mg/24hr or PCR ≥30mg/mmol or 2+ on dipstick).
    - **Signs of end-organ damage:** Renal insufficiency (↑creatinine), liver involvement (↑LFTs, epigastric/RUQ pain), neurological complications (headache, visual disturbances, hyperreflexia, clonus), hematological complications (thrombocytopenia, DIC, hemolysis), uteroplacental dysfunction (IUGR, abnormal Doppler).

      (Q70 Pre-eclampsia is more common in certain conditions. Multigravid women (A) are generally at lower risk than primigravids, unless they have other risk factors. So Q70A is the likely exception. Congenital heart disease (B), multiple pregnancy (C), diabetes (D), renal disease (E) are all risk factors.)(Q468 Sign of severe pre-eclampsia: Thrombocytopenia (D) is a key one. BP 140/90 (A) is the diagnostic threshold for any PET, not necessarily severe. Edema (B) is non-specific. Proteinuria 200mg (C) is less than the diagnostic threshold for PET. Dizziness (E) is vague. Severe features are BP ≥160/110, massive proteinuria, severe headache, visual changes, epigastric pain, HELLP, eclampsia, pulmonary edema.)(Q783 asks for a sign of severe PET: Thrombocytopenia (D) is a definite one. Proteinuria 200mg (C) is not even diagnostic for PET. Edema (B) is not specific. BP 140/90 (A) defines PET but not severity. Dizziness (E) is non-specific.)
  - **Eclampsia:** Pre-eclampsia PLUS new-onset generalized tonic-clonic seizures that cannot be attributed to other causes. A true obstetric emergency. *(Q321 Drug of choice for eclampsia is Magnesium Sulphate (A) - absolutely critical to know this for prophylaxis and treatment of seizures).*
  - **HELLP Syndrome: H**emolysis, **E**levated **L**iver enzymes, **L**ow **P**latelets. A severe variant/complication of pre-eclampsia. *(Q647 Most common association with maternal death in HELLP: Cerebral hemorrhage (B) or stroke is a major one, hepatic rupture/hemorrhage (C) also. DIC (A) is part of the cascade. ARDS (D) and Renal failure (E) are also severe complications. Often, it's cerebrovascular events or liver rupture that are cited as leading causes of death).*
  - **Superimposed Pre-eclampsia:** Chronic HTN with new development of proteinuria or other features of pre-eclampsia after 20 weeks.
- **Risk Factors for Pre-eclampsia:** Primigravidity, previous PET, family history of PET, multiple gestation, extremes of maternal age, obesity, pre-existing medical conditions (chronic HTN, renal disease, diabetes, autoimmune diseases like SLE/APS). *(Q330 Risk factors for PET: Family history (A), Diabetes (B), Renal disease (C), Antiphospholipid antibodies*

(E) are all true. Multiparity (D) is generally protective compared to primiparity, unless other risk factors are present. So Q330D is the exception.) (Q462 Sex of the baby (E) is NOT a risk factor for PET).

- **Pathophysiology (Simplified):** Abnormal placental development → poor trophoblastic invasion of spiral arteries → placental hypoperfusion/ischemia → release of anti-angiogenic factors & inflammatory mediators → widespread maternal endothelial dysfunction → vasospasm, increased capillary permeability, activation of coagulation cascade → multi-organ damage.
- **Management Principles:**
  - **Only CURE is DELIVERY of the placenta.**
  - **Monitoring:** BP, urine protein, maternal symptoms, fetal growth (IUGR risk!), fetal wellbeing (NST, BPP, Dopplers).
  - **Antihypertensives:** Used if BP persistently severe (e.g., ≥160/110 mmHg) to prevent maternal stroke. Goal is not to normalize BP but to reduce it to a safer range (e.g., 140-150 / 90-100 mmHg).
    - **Labetalol (beta-blocker with alpha-blocking activity):** Common first-line.
    - **Nifedipine (calcium channel blocker):** Common first-line. *(Q664 Main mechanism of Nifedipine is Calcium channel blocker (D)).*
    - **Methyldopa (centrally acting alpha-agonist):** Slower onset, good for chronic HTN but less for acute severe HTN. *(Q591 Drug of choice for mild HTN at 13wks: Methyldopa (C) is traditionally a safe choice in early pregnancy, though Labetalol is also used.)*
    - **Hydralazine (direct vasodilator):** IV for acute severe HTN, but can cause reflex tachycardia, headache.
    - **AVOID:** ACE inhibitors, ARBs, direct renin inhibitors (teratogenic!). Diuretics generally avoided unless specific indication (e.g., pulmonary edema).
  - **Magnesium Sulfate (MgSO4):**
    - **Indication:** Seizure prophylaxis in severe PET; treatment of eclamptic seizures.
    - **Mechanism:** CNS depressant, exact mechanism unclear but likely involves NMDA receptor antagonism and vasodilation.
    - **Monitoring for Toxicity:** Loss of deep tendon reflexes (patellar) is first sign *(Q202D correctly identifies this)*, respiratory depression, cardiac arrest. Therapeutic range: 4-7 mEq/L.
    - **Antidote:** Calcium gluconate.
  - **Timing of Delivery:** Balances maternal and fetal risks. Depends on GA, severity of PET, maternal/fetal condition.
    - Gestational HTN/Mild PET at term (≥37 weeks): Usually induce.
    - Severe PET: Often deliver regardless of GA once maternal stabilization achieved, especially if ≥34 weeks. If <34 weeks, may consider expectant management in tertiary center with steroids if stable.
    - *(Q662 Indications to deliver lady with uncontrolled high BP: Platelet count <50,000 (A), persistent headache (C), oliguria (D), fetal distress (E) are all valid. BP 140/90 (B) itself is diagnostic of PET but not an automatic indication for delivery if otherwise stable and preterm; severity and end-organ damage dictate this.*
- **Complications of Chronic HTN in Pregnancy:** Superimposed PET, IUGR, placental abruption, preterm delivery, C-section. *(Q42 lists pregnancy complications NOT associated with chronic HTN. Superimposed PET (A), Abruptio (B), IUGR (D), Preterm delivery (E) are all associated. Placenta previa (C) is not directly caused by chronic HTN, its risk factors are different (e.g., prior C/S, multiparity). So Q42C is the answer).*

VI. Antepartum Hemorrhage (APH) (Dr. Asmaa)*(Q25, 61, 125, 129, 146, 170, 174, 194, 218, 264, 268, 307, 360, 361, 371, 411, 432, 593, 637, 655, 709, 718, 721)*

Vaginal bleeding from 24 weeks gestation until delivery. This is an EMERGENCY until proven otherwise.

- **Definitions & Key Causes:**
  - **Placenta Previa:** Placenta implanted in the lower uterine segment, partially or completely covering the internal cervical os.
    - **Presentation:** Classically **painless** bright red vaginal bleeding. Uterus usually soft, non-tender. Fetal parts may be high/malpresentation.

- **Risk Factors:** Previous C-section (MAJOR!), multiparity, advanced maternal age, smoking, multiple gestation, prior uterine surgery/D&C. *(Q61 Predisposing factors for previa: Repeated induced abortion (A), Multi-fetal gestation (B), IVF (C), Congenital uterine anomalies (E - though less strongly than others). Malposition (D) is a consequence, not a cause. Q61D seems the best "except" if malposition refers to fetal lie which can be caused by previa). (Q194 Conditions LESS likely to be associated with previa: Primigravida (C) is less likely than multigravida (E), previous C/S (B), previous previa (D), or large placenta (A)).*
- **Diagnosis:** Ultrasound (transvaginal is safe and accurate). **NO DIGITAL VAGINAL EXAM** if previa suspected until location confirmed by US.
- **Management:** Depends on GA, severity of bleeding, maternal/fetal status.
  - If preterm and stable: Expectant management, corticosteroids, tocolysis if contracting (controversial).
  - If term, significant bleeding, or maternal/fetal compromise: Delivery by C-section.
  - *(Q170 Patient G2P0+1 at 37wks with painless bleeding, stable, Hb 11.5. Next step: Perform ultrasound examination (E) to confirm placental location and rule out previa before any other intervention. Vaginal exam (B) is contraindicated if previa is suspected. Induction (D) or C/S (C) depends on diagnosis and stability).*

- **Placental Abruption (Abruptio Placentae):** Premature separation of a normally implanted placenta from the uterine wall.
  - **Presentation:** Classically **painful** vaginal bleeding (can be dark red or concealed if retroplacental clot), uterine tenderness/rigidity ("woody" uterus), high-frequency contractions, fetal distress/demise. Maternal shock may be out of proportion to visible bleeding.
  - **Risk Factors:** Hypertension (chronic or PET - MAJOR!), trauma, smoking, cocaine use, previous abruption, PPROM, multiparity. *(Q129 Risk factors for abruption: Twins (A), Maternal thrombophilias (B - though association isn't as strong as for VTE), IVF pregnancy (C - can be associated with other placental issues), Intrauterine infections (E). Low BMI (D) is generally NOT a risk factor; if anything, severe malnutrition could be, but "low BMI" itself is vague.) (Q307 Primary cause of abruption: Often unknown (E) or multifactorial. Hypertension (B) is a major predisposing factor. Hypofibrinogenemia (A) is a consequence (DIC), not a primary cause. Acute toxemia (C - PET) is a risk factor. Trauma (D) is a known cause.)*
  - **Diagnosis:** Primarily clinical. Ultrasound may show retroplacental clot but is often normal (not sensitive).
  - **Management:** ABCs for mother, continuous fetal monitoring.
    - If severe abruption, maternal/fetal compromise: Immediate delivery (often emergency C-section).
    - If mild and preterm: May consider expectant management with close monitoring in a tertiary center.
    - *(Q125 G3P0+3 at 32wks with bleeding, pain, BP 130/80, +1 protein. This screams abruption (C).)*
    - *(Q268 G3P2 at 36wks in shock, rigid uterus, dead fetus, cervix 2cm. After resuscitation: Induce labor with prostaglandin/oxytocin (E) is often preferred for fetal demise if mother is stable and no contraindication to vaginal delivery. Proceeding immediately to C/S (B) for a dead fetus is less common unless for maternal reasons. Amniotomy (A) might be part of induction. Clexane (C) for DIC is specialized and not first-line. Stabilization (D) is part of initial resuscitation, then definitive management.)*
    - *(Q411 Suggests abruption: Abdominal tenderness (B). Abnormal fetal lie (A), profuse bleeding (C), low placenta (D), no pain (E) are more suggestive of previa or other causes).*
  - **Vasa Previa:** Fetal blood vessels run unprotected in the membranes over the cervical os. Catastrophic fetal bleeding occurs with ROM. Rare but deadly. High index of suspicion with painless bleeding after ROM and acute fetal distress/bradycardia. Emergency C-section.
  - **Local Causes:** Cervicitis, polyps, cervical cancer, vaginal trauma. Usually less severe bleeding.

- **General APH Management:**
  - **ABCs, IV access (2 large-bore cannulas), crossmatch blood.**
  - **History and Examination (NO digital VE if previa suspected).**
  - **Ultrasound** to locate placenta.
  - **Continuous fetal monitoring.**

- Decision on mode and timing of delivery based on cause, GA, severity, and maternal/fetal condition.
- *(Q25 Regarding APH: Vaginal bleeding after viability (A) is definition. Passage of show (B) is NOT APH. Increased perinatal morbidity (C) is true. Fetal bleeding (E) can occur (vasa previa, fetomaternal hemorrhage in abruption). "Patients with APH should be delivered by C/S" (D) is an EXCEPTION - this is not always true; depends on cause, severity, GA. For example, marginal previa with minimal bleeding at term might deliver vaginally, or a minor local cause.* So Q25D is the "except".)*

VII. Labor (Dr. Kameel)*(Q1, 27, 29, 46, 63, 72, 77, 78, 80, 91, 120, 122, 142, 163, 183, 187, 195, 198, 203, 206, 210, 212, 215, 222, 227, 230, 251, 252, 260, 270, 300, 304, 309, 320, 326, 341, 351, 352, 358, 362, 364, 370, 401, 417, 423, 435, 436, 448, 472, 476, 490, 498, 519, 536, 541, 547, 551, 552, 569, 570, 576, 579, 581, 585, 588, 618, 620, 623, 626, 683, 685, 724, 8, 34, 47, 363, 424, 438,446, 458, 466, 467, 477, 478, 491, 492, 515, 517, 544, 617, 631, 634, 665, 704, 708, 741)*

The process of childbirth. Understand the stages, mechanisms, and what's normal vs. abnormal.

- **Definitions:**
  - **Lie:** Relationship of the long axis of the fetus to the long axis of the mother (longitudinal, transverse, oblique). *(Q63A Longitudinal lie is appropriate). (Q210 Relationship of fetal parts to one another is Attitude (C)).*
  - **Presentation:** Fetal part entering the maternal pelvis first (cephalic, breech, shoulder).
  - **Position:** Relationship of a defined point on the presenting part (denominator) to the maternal pelvis (e.g., Left Occipito-Anterior - LOA). *(Q63B Position flexed – this is attitude, not position. So Q63B is the "except." Position describes rotation, e.g., OA, OP, OT).*
  - **Denominator:** Arbitrary point on the presenting part used to define position (Occiput for vertex, Sacrum for breech, Mentum for face). *(Q47 Denominator with face presentation is Mentum (D)).*
  - **Attitude:** Degree of flexion or extension of the fetal head and limbs. Normally fully flexed. *(Q618 An abnormal attitude is illustrated by Face presentation (B) - which is extended. Breech (A) and Transverse (C) are malpresentations (lie/presentation). Occiput posterior (D) is a malposition.)*
  - **Station:** Level of the presenting part in relation to the maternal ischial spines (0 station = at spines; -1, -2, -3 cm above; +1, +2, +3 cm below). *(Q63C Station at level of spines is appropriate if it means 0 station).*
  - **Engagement:** Largest diameter of the presenting part (biparietal for cephalic) has passed through the pelvic inlet. Clinically, head is 0 station or lower, and <2/5 palpable abdominally. *(Q63D Engagement two-fifths: This means 2/5 of the head is palpable abdominally, meaning 3/5 is in the pelvis – engaged. Q491 Head engaged when biparietal diameter (BPD) descends below pelvic inlet (C). Q626 Engagement is descent of BPD below pelvic inlet (A).)*
  - **Most favorable position for vaginal delivery:** Well flexed Occipito-Anterior (OA). *(Q1D Well flexed occipitoanterior is correct).*
- **Stages of Labor:**
  - **First Stage:** Onset of regular painful contractions to full cervical dilatation (10 cm).
    - **Latent Phase:** Cervix dilates slowly from 0 to ~4-6 cm. *(Q122 Primigravida, 2cm dilated, 1cm long, contractions q5min. This is Latent phase of labor (A)).*
    - **Active Phase:** More rapid cervical dilatation from ~4-6 cm to 10 cm.
  - **Second Stage:** Full dilatation to delivery of the baby.
  - **Third Stage:** Delivery of the baby to delivery of the placenta and membranes. *(Q448 During 3rd stage, pulling cord immediately after delivery (D) is contraindicated and can cause uterine inversion or cord avulsion. Gentle traction (A) is done with signs of separation. Check completeness (B) and wait for separation signs (C) are correct. Uterine massage (E) is after placental delivery usually.)*
  - **Fourth Stage:** First 1-4 hours postpartum (monitoring for PPH).
- **Mechanisms of Labor (Cardinal Movements) for Vertex OA:**
  1. **Engagement**
  2. **Descent**

3. **Flexion**
4. **Internal Rotation** (occiput rotates anteriorly)
5. **Extension** (head delivers by extending at the neck)
6. **Restitution** (head rotates back to align with shoulders)
7. **External Rotation** (shoulders rotate into AP diameter of outlet)
8. **Expulsion** (delivery of body)

   (Q251 Fetal head delivered by Extension (E). Q320 Rotation of head to become perpendicular to shoulders' axis after delivery of head is Restitution (C).)

- **Assessment During Labor:**
  - **Partogram:** Graphical record of labor progress (cervical dilatation, descent, contractions, maternal/fetal vitals). Essential for identifying abnormal labor.
  - **Fetal Monitoring:**
    - **Intermittent Auscultation:** For low-risk labors.
    - **Continuous Cardiotocography (CTG):** For high-risk labors or if abnormalities detected.
      - **Baseline FHR:** Normal 110-160 bpm.
      - **Variability:** Normal 5-25 bpm (indicator of fetal oxygenation).
      - **Accelerations:** Reassuring (≥15bpm rise for ≥15s).
      - **Decelerations:**
        - **Early:** Mirror contractions, due to head compression (vagal), usually benign. *(Q191 Classic cause of early decels is Head compression (B)).*
        - **Variable:** Abrupt onset/offset, variable shape, due to cord compression. *(Q283 Commonest cause of variable decels is Cord compression (E).)*
        - **Late:** Onset after peak of contraction, due to uteroplacental insufficiency/fetal hypoxia. Ominous. *(Q6 Repetitive late decels indicate Fetal hypoxia (B).)*
        - **Prolonged:** >2 min (if >10 min = baseline change/bradycardia).
      - *(Q259 Normal CTG parameters: Small decels <20bpm could be normal (B) if variable and not repetitive/severe. No sinusoidal (C) is normal. Basal HR 116-160 (E) is normal. At least one fetal movement or three accels (A) – usually 2 accels in 20 min for reactive NST. Short term variation (beat-to-beat) is different from long term variation (cycling). "Short term variation should be 2 minutes or less" (D) is an odd phrasing; variability is measured in bpm, not duration.)*
      - *(Q421 Positive Contraction Stress Test (CST) means Late decelerations with at least 50% of contractions (E).)*
- **Induction of Labor (IOL):** Artificial initiation of labor.
  - **Indications:** Post-term gestation (common - *Q8B, Q198B*), PET, PROM, IUGR, fetal demise, maternal medical conditions (e.g., diabetes).
  - **Contraindications:** Placenta/vasa previa *(Q8A, Q198A)*, cord presentation *(Q8C, Q198C)*, prior classical C-section *(Q8D, Q198D)*, active genital herpes *(Q8E, Q198E)*, transverse lie, cephalopelvic disproportion (CPD). *(Q222 Absolute contraindications for IOL except: Umbilical cord presentation (C) IS an absolute contraindication, often needing C/S. Active herpes (A), previa grade II posterior (B), transverse lie (D), oblique lie (E) are also generally contraindications. This question is tricky. Perhaps "umbilical cord presentation" here implies it's diagnosed before IOL is considered, making other options relatively "less" absolute if conditions could change or if specific IOL methods were considered. However, typically all listed are strong contraindications).*
  - **Bishop Score:** Assesses cervical favorability for induction (dilatation, effacement, station, consistency, position). Higher score = higher success. *(Q215 Bishop score components: Station (A), Length (C), Dilatation (D), Consistency (E). Position of presenting part (B) is not a direct component, but cervical position (anterior/mid/posterior) is). (Q438 Bishop score parameters except: Pelvic capacity (C) is not part of Bishop score).*

    - **Methods:** Prostaglandins (PGE2 gel/pessary, PGE1 Misoprostol), amniotomy (ARM), oxytocin, mechanical (balloon catheter).
    - *(Q34 Contraindicated for ARM: Genital active herpes (E) is a contraindication for vaginal delivery in general, thus ARM. Occipitoposterior (A), polyhydramnios (B), accidental hemorrhage (C), twins (D) are not absolute contraindications to ARM, though care is needed, especially with polyhydramnios due to cord prolapse risk).*
- **Abnormal Labor (Dystocia):**
    - **Protracted Labor:** Slower than normal progress.
    - **Arrest of Labor:** No progress over a defined period (e.g., 2-4 hours in active phase).
    - *(Q46 Cervix from 6 to 9 cm in 2 hours in G2P1. This is Normal labor (C) progression (active phase usually 1-1.5cm/hr for multip, >1cm/hr for primip).)*
    - Causes: 3 P's - Powers (contractions), Passenger (fetus - size, position, presentation), Passage (pelvis).
- **Fetal Skull & Pelvis:**
    - **Sutures & Fontanelles:** Allow moulding. Anterior fontanelle (diamond) closes ~18mo. Posterior (triangle) closes ~2-3mo. *(Q72B Sutures are NOT ossified, allowing moulding; this statement is false and thus the "except").*
    - **Diameters:** Suboccipitobregmatic (SOB) 9.5cm (well-flexed vertex). Occipitofrontal (OF) 11.5-12cm (deflexed). Occipitomental (OM) 13.5cm (brow). Submentobregmatic (SMB) 9.5cm (face). *(Q72D OM diameter is indeed too large. Q341 Fetal skull diameters: Mentovertical (B) is 13.5cm, not 11.5cm, so this is incorrect and the "except". SOB 9.5cm (A), OF 11.5cm (C), SMB 9.5cm (D), Biparietal 9.5cm (E) are correct.)*
    - **Moulding:** Overlapping of skull bones. Normal physiological process. *(Q72E correct).*
    - *(Q402 Shortest distance between sacral promontory and symphysis pubis is Obstetric Conjugate (D). True conjugate is from SP to sacral promontory. Diagonal conjugate is measured clinically from lower border of SP to sacral promontory.)*
    - *(Q492 Plane of least pelvic dimensions is Midpelvis (C), at the level of ischial spines.)*

VIII. Operative Vaginal Delivery (OVD) (Dr. Islam Al-awamleh)*(Q2, 13, 35, 36, 155, 156, 179, 333, 356, 493, 582, 616, 630, 659)*

Use of forceps or vacuum extractor to assist vaginal birth.

- **Indications:**
    - Prolonged second stage of labor (maternal exhaustion). *(Q333B)*
    - Non-reassuring fetal status in second stage. *(Q333D)*
    - Maternal conditions where pushing is contraindicated (e.g., certain cardiac diseases, risk of ICP).
- **Prerequisites (CRITICAL - "FORCEPS" or similar mnemonic):**
    - **F**ull cervical dilatation (10 cm). *(Q2 Pre-requisites for forceps except: At least 8cm dilatation (B) is WRONG. Must be FULLY dilated).*
    - **R**uptured membranes. *(Q2A correct).*
    - **E**ngaged presenting part (usually 0 station or lower).
    - **P**osition of head known. *(Q2C correct).*
    - **C**ephalic presentation (vertex ideal).
    - **E**mpty bladder.
    - **P**ain relief adequate.
    - **S**killed operator, consent obtained. *(Q2E correct).*
    - *(Q2D +1 station or below is generally acceptable for outlet/low forceps).*
- **Contraindications:**
    - Unengaged head.
    - Incompletely dilated cervix.

    - Unknown fetal position.
    - Non-cephalic presentation (generally).
    - Cephalopelvic disproportion (CPD). *(Q333A CPD is a contraindication, not an indication, so this is the "except").*
    - Certain fetal conditions (e.g., bone demineralization disorders, bleeding diathesis).
    - *(Q493 Contraindications for forceps: Brow (C) and Face (D) presentations are generally contraindications. Occipito-posterior (B) can sometimes be delivered with specific forceps/maneuvers or rotated. Occipito-anterior (A) is ideal. After-coming head in breech (E) can be delivered with Piper forceps.) So C or D are strong contraindications.*
    - *(Q630 Contraindications for forceps: Brow (C) and Face (D) are strong contraindications.)*
- **Types:**
    - **Forceps:** Outlet, low, mid (mid-forceps rarely used now due to higher morbidity). Specific types for rotation (e.g., Kielland's).
    - **Vacuum Extractor (Ventouse):** Cup applied to fetal scalp.
- **Complications:**
    - **Maternal:** Perineal trauma (3rd/4th degree tears - *Q13B Episiotomy is an intentional second-degree perineal tear*), PPH, infection, urinary/fecal incontinence.
    - **Fetal/Neonatal:** Scalp/facial bruising or lacerations, cephalohematoma (especially vacuum), subgaleal hemorrhage (rare, dangerous - vacuum), facial nerve palsy (forceps), intracranial hemorrhage (rare), shoulder dystocia. *(Q156 Dangers of vacuum extraction, incorrect: APH (A) can be a complication if trauma occurs. Ruptured uterus (B) is rare but possible with difficult OVD. IUFD (C) - OVD is done on a live fetus. PPH (D) is a risk. Acute fetal distress (E) is an INDICATION for OVD, not usually a direct danger of it, unless the procedure is prolonged/traumatic. Q156A APH is the least direct/common "danger" listed as a primary event caused by vacuum itself, compared to fetal trauma).*
        - *(Q616 Risks of forceps: Intracranial hemorrhage (A), Skull fracture (B), Scalp injury (C), Facial palsy (E) are all known. Brachial palsy (D) is more associated with shoulder dystocia, which can occur with or without OVD, but OVD doesn't directly cause it in the same way as head/facial injuries.)*
- **Episiotomy:** Intentional incision of perineum to enlarge vaginal outlet. Mediolateral or midline. *(Q13 Episiotomy is an intentional second-degree perineal tear (B)).*
    - *(Q155 Midline vs. Mediolateral episiotomy: Mediolateral is more likely to bleed (A) due to more vascular tissue, but less likely to extend to anal sphincter (C is wrong for mediolateral). Midline is less painful (B is wrong for mediolateral) and easier to repair but higher risk of sphincter extension.)*
- **Specific Scenarios:**
    - *(Q35 Impacted shoulder (dystocia) of a living fetus is managed by: upper segment Cesarean section (D) is an option if maneuvers fail or it's diagnosed before full engagement. Internal podalic version (A) is for a second twin usually. ECV (B,C) is for breech before labor.)* This question likely refers to maneuvers first (McRoberts, suprapubic pressure etc.), and if those fail, heroic measures or C/S.*
    - *(Q36 A G1P0 with flexed breech, membranes intact, prior C/S, keen for VBAC. Management: Caesarean Section (C) is generally the safest for a breech with prior C/S. Vaginal breech delivery (D) carries higher risks in this scenario. ECV (A) might be an option if no contraindications, but prior C/S can be a relative contraindication.)*

Brutal Honesty Checkpoint:

- **Hypertension:** Know the definitions. MgSO4 for seizure prophylaxis/treatment is gold. Delivery is the cure.
- **APH:** Painless (previa) vs. Painful (abruption) is the classic starting point. NO VEs with suspected previa until US. Abruption is a clinical diagnosis.
- **Labor:** Stages, cardinal movements, CTG interpretation basics (especially late decels). Partogram is your friend for identifying dystocia.
- **OVD:** Prerequisites are a MUST-KNOW. Don't attempt if not met. Know the main maternal/fetal risks.

Obstetrics: The Aftermath – Bleeding, Recovery, Special Cases

The baby is out, but the drama isn't always over. Postpartum hemorrhage is a leading cause of maternal mortality. Recovery has its own challenges. And some pregnancies have unique immunological or numerical complexities.

IX. Postpartum Hemorrhage (PPH) & Maternal Injuries (Dr. Ayman)*(Q12, 121, 186, 205, 253, 291, 384, 437, 502, 739)*

Losing too much blood after delivery. Know the causes and management cold.

- **Definition:**
  - **Primary PPH:** Blood loss ≥500ml from genital tract within 24 hours of delivery (or ≥1000ml for C-section, or any amount causing hemodynamic instability).
  - **Secondary PPH:** Abnormal or excessive bleeding from genital tract between 24 hours and 12 weeks postpartum.
- **The 4 T's – Your PPH Bible (Causes of Primary PPH):**
  1. **Tone (Atony):** Most common cause (70-80%). Uterus fails to contract effectively.
     - **Risk factors:** Prolonged labor, augmented labor, grand multiparity, overdistended uterus (macrosomia, multiple pregnancy, polyhydramnios), fibroids, chorioamnionitis, retained products, general anesthesia.
     - *(Q253 PPH, uterus felt 2cm below umbilicus and firm, after forceps delivery. This points away from atony. Likely Genital tract trauma (E). Q12 PPH unresponsive to oxytocin and uterine massage: most likely Lacerations (A) if atony is ruled out by effective massage/uterotonics and a firm uterus.*
  2. **Trauma:** Lacerations of cervix, vagina, perineum; uterine rupture; uterine inversion.
     - Suspect if bleeding persists despite a well-contracted uterus.
     - *(Q121, Q253 above highlight trauma).*
  3. **Tissue (Retained Products):** Retained placenta, placental fragments, or membranes.
     - Prevents uterus from contracting effectively. Inspect placenta for completeness.
     - *(Q12C Retained placenta is a cause of PPH unresponsive to oxytocin/massage).*
  4. **Thrombin (Coagulopathy):** Pre-existing (e.g., von Willebrand's) or acquired (e.g., DIC from abruption, severe PET, amniotic fluid embolism, sepsis).
     - *(Q12E Coagulopathy is a cause).*
- **Management of Primary PPH (Simultaneous Actions – "Call for Help" is First!):**
  - **ABCs:** Airway, Breathing, Circulation. High-flow oxygen.
  - **IV Access:** Two large-bore cannulas. Send blood for FBC, coagulation screen, crossmatch.
  - **Uterine Massage:** "Rub up a fundus."
  - **Uterotonics:**
    - **Oxytocin (Syntocinon):** First-line IV infusion.
    - **Ergometrine:** IM/IV (contraindicated in HTN).
    - **Misoprostol (PGE1):** Rectal, sublingual, oral.
    - **Carboprost (Hemabate, PGF2α):** IM (contraindicated in asthma).
  - **Tranexamic Acid:** Antifibrinolytic, reduces bleeding.
  - **Identify and Treat Cause:**
    - **Atony:** Massage, uterotonics.
    - **Trauma:** Inspect, repair lacerations.
    - **Tissue:** Manual removal of placenta/products, D&C.
    - **Thrombin:** Correct coagulopathy (FFP, cryoprecipitate, platelets).
  - **Surgical/Interventional (if medical management fails):**
    - **Balloon Tamponade (e.g., Bakri balloon).**
    - **Compression Sutures (e.g., B-Lynch).**

        - **Uterine Artery Ligation/Embolization.**
        - **Internal Iliac Artery Ligation.**
        - **Hysterectomy (last resort).**
        - *(Q291 Lady with PPH, G3P3, estimated blood loss 2L, tachycardic, hypotensive. All are part of plan EXCEPT: Giving her iron supplement intra venous (A) is for treating anemia LATER, not for acute massive hemorrhage management. Repetition of labs (B), transfusion (C), B-Lynch (D), uterine artery ligation (E) are all appropriate considerations in massive PPH.)*
- **Maternal Injuries:**
    - **Perineal Tears:**
        - 1st degree: Skin only.
        - 2nd degree: Perineal muscles (episiotomy is a 2nd degree).
        - 3rd degree: Involves anal sphincter complex (3a, 3b, 3c subdivisions).
        - 4th degree: Involves rectal mucosa.
        - *(Q741 Primigravida, paraurethral tear and tear involving sphincter, rectum intact. This is a Third degree tear (B)).*
    - **Uterine Inversion:** Uterus turns inside out. Obstetric emergency. Associated with fundal pressure, excessive cord traction. Leads to shock, PPH.
        - *(Q205 Complete uterine inversion associated with: Excessive umbilical cord traction (D) is a classic cause. Placenta accreta (A) can also predispose. Short umbilical cord (B) can contribute to traction. Posterior placenta (C) less directly linked. Grandmultiparity (E) is a risk for atony, not directly inversion as much as the others).*
    - **Uterine Rupture:** Can occur with prior uterine scar (especially classical C/S), obstructed labor, trauma.

X. Postpartum Care (Dr. Oqba)*(Q67, 94, 237, 238, 239, 308, 344, 548, 565, 625, 648, 692, 784, 794)*

The "puerperium" – typically the first 6 weeks after delivery. A period of significant physiological and psychological adjustment.

- **Uterine Involution:** Uterus returns to pre-pregnant size.
    - Immediately after delivery: Fundus at umbilicus or just below.
    - Descends ~1 cm/day.
    - By ~2 weeks: No longer palpable abdominally.
    - By ~6 weeks: Back to normal size.
    - *(Q237 Uterine involution: Uterus becomes normal non-pregnant size by 12 days postpartum (E) is TOO EARLY. It's typically around 6 weeks. Contraction of interlacing myometrial muscle bundles (C) is key for hemostasis. Large vessels at placental site thrombose (D) also key. Myometrial retraction (B) is a unique characteristic. Uterus involutes immediately after delivery (A) is true – the process starts then.)*
    - *(Q238 Examining uterine fundus immediately post-delivery: Soft (A) is FALSE; it should be firm. More globular (B) is true. Not tender (C) is usually true unless infection/retained products. Near umbilicus (D) is true. Midway between SP and umbilicus within one week (E) is also typical progression.)*
    - **Subinvolution:** Uterus fails to involute properly. Causes: Retained products, infection (endometritis). *(Q239 Causes of subinvolution except: Breastfeeding (C) promotes involution (oxytocin release). Multiparity (A), C/S (B), Full bladder (D), Endometritis (E) can all impair involution).*
- **Lochia:** Postpartum vaginal discharge.
    - Lochia rubra: Red, first few days.
    - Lochia serosa: Pinkish-brown, up to ~10 days.
    - Lochia alba: Whitish-yellow, up to ~2-6 weeks.

- **Postpartum Blues, Depression, Psychosis:**
  - **Blues:** Common (~50-80%), mild, transient mood disturbance, tearfulness, anxiety. Onset 2-3 days postpartum, resolves within ~2 weeks. Reassurance, support.
  - **Depression (PPD):** More severe, persistent (>2 weeks) depressive symptoms. Affects ~10-15%. Needs active treatment (counseling, SSRIs). Edinburgh Postnatal Depression Scale (EPDS) for screening.
  - **Psychosis:** Rare (~0.1-0.2%), severe psychiatric emergency. Delusions, hallucinations, disorganized behavior, risk to self/baby. Onset often early (days to weeks). Requires urgent psychiatric admission, antipsychotics. *(Q794 Puerperal psychosis: Usually responds well to treatment (A) with appropriate intervention. Often occurs in patients with a history of mental illness (B), especially bipolar. Can be schizophrenic or affective (C is too simplistic). CAN recur (D is false). Can take various forms including mania/depression (E is true). Q794B is the most accurate "correct" statement in the context of risk factors.)*
- **Contraception:** Discuss before discharge.
  - Progestogen-only methods (POP, implant, injection) can be started immediately.
  - Combined hormonal contraceptives (COCP, patch, ring): Delay ~3-6 weeks due to VTE risk (especially if breastfeeding, delay till 6 weeks).
  - IUD/IUS: Can be inserted immediately post-placentally (higher expulsion risk) or at ~4-6 weeks postpartum.
- **Breastfeeding:** Benefits for mother and baby. Support and education crucial. Common issues: sore nipples, engorgement, mastitis.
- **Other Postpartum Issues:**
  - **Puerperal Infection (Sepsis):** Endometritis (most common), UTI, wound infection, mastitis. *(Q67 Risk factors for puerperal infection except: Prolonged pregnancy (B) itself is not a direct risk factor for infection as much as prolonged ROM (A), prolonged 2nd stage (C), C/S (D), or retained products (E)).*
  - **Thromboembolism (VTE):** Risk significantly elevated postpartum.
  - **Urinary issues:** Retention, incontinence.
  - **Anemia.**
- **Routine Checks:** Lochia, fundal height, perineum, vital signs, mood, breastfeeding.

XI. Rh Isoimmunization (Dr. Oqba)*(Q17, 83, 269, 366, 691, 754, 782, 787)*

A preventable cause of Hemolytic Disease of the Fetus and Newborn (HDFN).

- **Pathophysiology:**
  1. RhD-negative mother.
  2. RhD-positive fetus (father is RhD-positive).
  3. Sensitization event: Fetal RhD-positive RBCs enter maternal circulation (e.g., delivery, miscarriage, abortion, APH, invasive procedures, abdominal trauma - *Q83A trauma can trigger it*).
  4. Mother produces anti-D IgG antibodies.
  5. In subsequent RhD-positive pregnancies, maternal anti-D IgG crosses placenta *(Q17C IgG crosses placenta, correct)*, attacks fetal RBCs → hemolysis → HDFN.
- **HDFN Spectrum:** Fetal anemia, jaundice, hepatosplenomegaly, hydrops fetalis (severe edema, ascites, effusions), kernicterus (bilirubin encephalopathy), fetal/neonatal death.
- **Key Points:**
  - Usually does NOT affect the first pregnancy (unless sensitization occurred before that pregnancy, e.g. prior mismatched transfusion, or very early unrecognized miscarriage of Rh+ fetus). *(Q83B "Never affects the first pregnancy" is generally true for the first RhD-positive pregnancy if no prior sensitization).*
  - Severity tends to increase with successive sensitized pregnancies *(Q17D Tends to become more severe, correct).*
- **Prevention with Anti-D Immunoglobulin:**

- **Mechanism:** Anti-D given to mother binds to any fetal RhD-positive RBCs in her circulation, preventing her immune system from recognizing them and forming her own anti-D antibodies.
- **Routine Antenatal Anti-D Prophylaxis (RAADP):** Given to all RhD-negative unsensitized women at ~28 weeks gestation. *(Q269, Q366, Q691, Q787 often allude to this timing or need for prophylaxis)*.
- **Postpartum Anti-D:** Given to RhD-negative mother within 72 hours of delivering an RhD-positive baby.
- **After Potentially Sensitizing Events (PSEs):** E.g., miscarriage, abortion, APH, CVS, amniocentesis, ECV, abdominal trauma. Dose depends on GA and event. *(Q17B 500 IU anti-D can eliminate up to ~8-10ml of fetal blood – this is generally true for standard doses, though exact neutralization capacity varies by preparation. Q83E 50 microgram dose for ~5mL is a reasonable approximation for smaller amounts/earlier GAs)*.
- **Screening:** All pregnant women for blood type and antibody screen at booking *(Q83C Should be screened at booking, correct)* and often again at ~28 weeks.
- **If sensitized (anti-D antibodies present):** Anti-D prophylaxis is USELESS. Pregnancy requires close monitoring by specialist team (serial antibody titers, fetal MCA Dopplers for anemia, intrauterine transfusions if severe). *(Q83D Anti-D indicated at 28 weeks if mother is SENSITIZED is FALSE. Anti-D is for unsensitized women to prevent sensitization)*.

- **Genetics:** RhD-negative means genotype dd. RhD-positive can be DD or Dd. *(Q17A "All rhesus negative people have "d" in each half of the genotype" is TRUE (dd))*.

XII. Multiple Pregnancy (Dr. Amal)*(Q32, 62, 65, 85, 87, 159, 272, 286, 453, 508, 693, 711, 781, 785)*

More than one fetus. Higher risks for almost everything.

- **Types & Zygosity/Chorionicity:**
  - **Dizygotic (DZ, "Fraternal"):** Two ova fertilized by two sperm. Always dichorionic-diamniotic (DC/DA). Genetically like siblings. *(Q159B All DZ twins are DC/DA, correct)*.
  - **Monozygotic (MZ, "Identical"):** Single ovum fertilized by single sperm, then splits.
    - Timing of split determines chorionicity/amnionicity:
      - **Days 0-3 (morula):** Dichorionic-diamniotic (DC/DA) – ~30%. *(Q159A "All monochorionic diamniotic twins are monozygotic" is TRUE. Q159C "All dichorionic diamniotic twins are dizygotic" is FALSE, as MZ can be DC/DA). (Q159E DC/DA twins are more common than MC/MA twins - this is generally true as DZ are more common overall, and MZ can also be DC/DA)*.
      - **Days 4-8 (blastocyst):** Monochorionic-diamniotic (MC/DA) – ~70%. Most common MZ type. Share placenta, separate amniotic sacs.
      - **Days 8-13 (implanted blastocyst):** Monochorionic-monoamniotic (MC/MA) – ~1%. Share placenta and amniotic sac. High risk of cord entanglement. *(Q159D MC/MA twins are always same sex, true, as are all MZ twins)*.
      - **Days >13 (embryonic disc):** Conjoined twins – rare.
    - *(Q32 Ovulation-cleavage interval 9-12 days: This timing corresponds to splitting after implantation of blastocyst but before formation of embryonic disc, leading to Monochorionic-Monoamniotic (MC/MA) twins (A). Diamniotic monochorionic (C) is earlier split (4-8 days).)*
    - *(Q286 Cleavage 9-12 days results in MC/MA twins (A).)*
- **Chorionicity is KEY:** Determines risks and management. Best determined by US in 1st trimester (lambda/twin-peak sign for DC, T-sign for MC).
- **Maternal Complications (Increased Risk Of):**
  - Hyperemesis gravidarum
  - Anemia
  - Gestational HTN / Pre-eclampsia *(Q87 Not an increased risk in multiple pregnancy: Diabetes mellitus (B). While GDM can occur, the risks of PET (C), APH (A - from previa/abruption), Malpresentation (D), IUGR (E) are often more emphasized as direct consequences of multiplicity. However, GDM risk IS increased in multiples. This*

question is tricky. Perhaps diabetes mellitus refers to pre-existing type 1 or 2, which isn't caused by multiple pregnancy but can complicate it.)

- Gestational Diabetes Mellitus (GDM)
- Antepartum hemorrhage (previa, abruption)
- Polyhydramnios
- Preterm labor & delivery (MAJORITY deliver preterm - *Q62A PTL common, true*)
- Operative delivery (C-section more common)
- Postpartum hemorrhage (uterine atony due to overdistension) *(Q62E PPH can occur with 2nd stage, true, and is more common with twins).*

- **Fetal/Neonatal Complications (Increased Risk Of):**
  - Prematurity and its sequelae (RDS, IVH, NEC, ROP)
  - Congenital anomalies (slightly higher in MZ)
  - IUGR (placental sharing/crowding)
  - Cord accidents (especially MC/MA)
  - Stillbirth/neonatal death
  - **Complications Specific to Monochorionic Twins (due to shared placenta & vascular anastomoses):**
    - **Twin-to-Twin Transfusion Syndrome (TTTS):** Unbalanced blood flow between twins. Donor anemic, oliguric, IUGR, oligohydramnios. Recipient polycythemic, polyuric, cardiomegaly, polyhydramnios, hydrops. *(Q693 Antenatal diagnosis of TTTS includes all except: Polyhydramnios in the smaller twin (C) is WRONG. Smaller twin is donor, has oligohydramnios. Same sex (A) is true for MC. Intertwin birth weight difference >20% (B), Hb difference >5g/dl (D), Large bladder in recipient (E) are all features.) (Q781 TTTS all true except: "Occur in diamniotic dichorionic twins" (B) is FALSE. It's a MC complication.)*
    - **Twin Anemia-Polycythemia Sequence (TAPS):** Chronic, slow inter-twin transfusion without fluid imbalance.
    - **Selective IUGR (sIUGR):** One twin significantly smaller.
    - **Twin Reversed Arterial Perfusion (TRAP) Sequence:** Acardiac twin perfused by normal "pump" twin.
- **Antenatal Management:**
  - Early US for chorionicity.
  - More frequent visits and US surveillance (growth, AFI, Dopplers, cervical length).
  - Nutritional advice (increased caloric/iron/folate needs).
  - Early recognition and management of complications.
- **Delivery Considerations:**
  - Timing: Depends on chorionicity, complications. MC twins often delivered earlier (~36-37w for uncomplicated MC/DA, ~32-34w for MC/MA). DC/DA ~37-38w.
  - Mode: Cephalic/cephalic twins can often deliver vaginally if no other contraindications. Non-cephalic presentation of first twin usually C/S. Malpresentation of second twin after vaginal delivery of first may need ECV, internal podalic version, or C/S. *(Q62 Lady G3P2 with twins, worried. False statement: "Always delivered by cesarean section" (C) is FALSE. Vaginal delivery is possible and often attempted.) (Q65 Reproductive history: G3P2 with previous twin delivery and a stillbirth at 27/40. A twin delivery counts as ONE parity event but TWO live/stillborn children (depending on outcome). A stillbirth at 27/40 counts as a para. So, 3 pregnancies. First twin delivery = P1 (but 2 babies). Second stillbirth = P1. So G3P2. If the twins were live, it could be G3 P1+2 (one term, two preterm/term depending on GA of twins). The notation is often complex. Assuming the G3P2 is the "summary" including the current pregnancy as G1, then two prior para events. A previous twin delivery (P1 resulting in 2 babies) and a stillbirth (P1 resulting in 1 baby). So she's had 3 babies from 2 deliveries. So G3P2 is plausible under some systems. However, typically parity refers to pregnancies carried to viability. If the twin delivery was one parity event, and the stillbirth another, then it should be G3P2. If the question implies current pregnancy is the 3rd (G3), then prior: Twin delivery (Para 1, producing 2 fetuses), Stillbirth (Para 1, producing 1 fetus). This question depends heavily on the specific notation system being used. Q85 asks for G/P for a woman at 12/40 with previous twin delivery and a stillbirth. Current preg = G1. Previous twin = G1P1 (2 fetuses). Previous stillbirth = G1P1 (1 fetus).*

Total: G3 P2 (resulting in 3 fetuses). The options (G3P3, G3P4 etc.) are confusing. If G3P3 (A?) means 3 pregnancies, 3 delivered beyond viability (twins as one, stillbirth as one, plus one other not mentioned or this is current and P counts will be 2). This is very notation-dependent. Let's assume "P" refers to number of deliveries past viability, and any number after "+" refers to abortions/previable losses. If she is *currently* G3, she had 2 prior deliveries. If one was twins and one was a stillbirth, she is P2. The number of babies from those deliveries would be 3. If the question meant she's *had* 3 pregnancies in total and two delivered, then G3P2 is correct. Q65/Q85 seem to be the same question stem. Assuming "P" counts number of *deliveries* past 20-24 weeks. Twin delivery is one P event. Stillbirth is one P event. So P2. If she's had 3 pregnancies in total (including current if it's counted in "G"), then G3 P2. The options are messy for standard G_P(TPAL) notation.)*

Brutal Honesty Checkpoint:

- **PPH:** 4 T's! Know them, know how to manage each. This is an emergency.
- **Postpartum Care:** Involution timeline, red flags for infection/mood disorders. Contraception advice is key.
- **Rh Isoimmunization:** Understand sensitization and HOW Anti-D works (prevention, not treatment of sensitization). Know who gets it and when.
- **Multiple Pregnancy:** Chorionicity dictates everything. Know the specific risks of monochorionic twins (TTTS!). Most will deliver preterm.

Obstetrics: When Things Go Sideways – Ectopics, Sugar Troubles, Severe Sickness, Hormones, and Fluid Imbalances

Pregnancy isn't always straightforward. Sometimes the implantation is wrong, metabolic stress reveals underlying issues, or the body's own processes go into overdrive. Understanding these common but potentially serious conditions is vital.

XIII. Ectopic Pregnancy (Dr. Kameel)*(Q126, 178, 265, 298, 316, 336, 380, 388, 543, 644, 728)*

Implantation outside the uterine cavity. A leading cause of first-trimester maternal mortality. THINK RUPTURE!

- **Commonest Site:** Ampulla of the fallopian tube (overwhelmingly). *(Q543A Ampullary part is correct).*
- **Risk Factors:**
  - Previous ectopic pregnancy (highest risk factor).
  - History of Pelvic Inflammatory Disease (PID) / Salpingitis (causes tubal damage).
  - Previous tubal surgery (e.g., tubal ligation, reanastomosis).
  - Use of Intrauterine Device (IUD) – if pregnancy occurs *with* an IUD in situ, it's more likely to be ectopic.
  - Assisted Reproductive Technologies (ART) like IVF.
  - Smoking.
  - Advanced maternal age.
  - Endometriosis.
  - *(Q265 Risk factors for ectopic except: Combined oral contraceptive pills (E). COCPs prevent ovulation and thus pregnancy overall; they are protective against ectopic pregnancy, not a risk factor. PID (A), pelvic surgery (B), fertility drugs (C), smoking (D) are all risks).*
  - *(Q178 Concerning ectopic: Incidence has increased (A) - true, due to more PID, ART etc. Diethylstilbestrol (B) is more linked to clear cell vaginal adenoCa, not a primary ectopic risk. Most occur in fimbrial end (C) - ampulla is more common than fimbrial. Mildly febrile (D) - not typical unless secondary infection/inflammation. MTX interferes with DNA synthesis (folate antagonism) (E) - this statement about MTX is correct).*
- **Clinical Presentation – The Classic Triad (but often incomplete/atypical):**
  1. Amenorrhea (missed period).
  2. Vaginal bleeding (often scanty, dark, "prune juice").
  3. Abdominal/pelvic pain (can be unilateral, colicky, or sudden severe if ruptured).
  - **Signs of Rupture:** Severe pain, shoulder tip pain (referred diaphragmatic irritation from hemoperitoneum), signs of shock (tachycardia, hypotension, dizziness, syncope). THIS IS A SURGICAL EMERGENCY. *(Q380 Signs of ruptured*

fallopian tube ectopic except: Diarrhea (D) is not a typical sign. Sudden severe pain (A), faintness/dizziness (B), N/V (C due to pain/peritonism), shoulder tip pain (E) are all consistent.)

- **Diagnosis:**
  - **Quantitative β-hCG:** In a normal IUP, hCG doubles approx. every 48 hours. In ectopics, hCG levels often rise suboptimally, plateau, or fall slowly. There's a "discriminatory zone" (hCG level, e.g., 1500-2000 mIU/mL) above which an IUP should be visible on transvaginal ultrasound (TVUS).
    - *(Q126 Pregnant lady, 6 weeks missed period, minimal bleeding/pain, hCG 630, TVUS showed thickened endometrium but no IUP. Suitable treatment: Repeat β-hCG after 48 hours (A) is the standard next step for a stable patient with a "pregnancy of unknown location" (PUL) at this hCG level to assess the trend before more invasive measures like laparoscopy or empiric methotrexate, unless symptoms worsen or suspicion is very high for rupture.)*
    - *(Q316 Lady with missed period, spotting, prior ectopic, hCG 800, TVUS unremarkable. Next best step: Repeat serum β-hCG after 48 hours (D). Similar to Q126, assessing the trend is crucial in stable PUL.)*
    - *(Q336 Patient with pain/spotting, hCG 7500, TVUS: thickened endometrium, normal ovaries, no adnexal mass/free fluid. Next step: Further β-hCG in 48 hours (B). At hCG 7500, an IUP should be visible. If not, strong suspicion for ectopic or complete miscarriage. Serial hCGs will differentiate. Suction evacuation (D) is for molar or incomplete miscarriage if confirmed in utero.)*
  - **Transvaginal Ultrasound (TVUS):** Key. Look for IUP. Absence of IUP with hCG above discriminatory zone is highly suggestive of ectopic. May see adnexal mass (the "blob sign," "ring of fire" with Doppler), or free fluid/hemoperitoneum (suggests rupture).
  - **Laparoscopy:** Gold standard for diagnosis and often treatment.
- **Management:**
  - **Expectant:** For very select, asymptomatic cases with low and falling hCG, no adnexal mass, no rupture. Requires close monitoring.
  - **Medical (Methotrexate - MTX):** For stable, unruptured ectopics meeting specific criteria (e.g., hCG <5000, adnexal mass size <3.5-4cm, no fetal cardiac activity, hemodynamically stable, reliable for follow-up, normal LFTs/renal function). MTX is a folate antagonist, inhibiting trophoblast cell division.
  - **Surgical:**
    - **Laparoscopy** is preferred over laparotomy unless hemodynamically unstable.
    - **Salpingostomy:** Incision in tube, removal of ectopic, tube preserved (for future fertility, but risk of persistent trophoblast).
    - **Salpingectomy:** Removal of affected tube (if tube damaged, recurrent ectopic in same tube, completed family, or uncontrolled bleeding). *(Q644 Patient with 6wks amenorrhea, pain, US: 5cm unruptured ectopic. Options for therapeutic operative procedures: Salpingectomy (A), Salpingotomy (C), Salpingostomy (D) are all standard. Milking pregnancy from tube (E) is sometimes done but less common/reliable. Salpingo-oophorectomy (B) - removing the ovary - is NOT a standard option unless ovary is involved/damaged or other pathology exists. So Q644B is the one "not an option" for a simple unruptured ectopic.)*

XIV. Gestational Diabetes Mellitus (GDM) (Dr. Shawqi)*(Q40, 41, 102, 103, 209, 271, 305, 503, 525, 605, 643, 672, 776)*

Carbohydrate intolerance with onset or first recognition during pregnancy. Pregnancy is a diabetogenic state (hPL, progesterone leading to insulin resistance).

- **Risk Factors:** Previous GDM, family history of diabetes, obesity (high BMI), advanced maternal age, previous macrosomic baby, PCOS, certain ethnic groups. *(Q643 Predisposing factors for GDM except: Family history of type 1 diabetes (C). While any family history of diabetes might slightly increase awareness, GDM is more akin to Type 2 diabetes in its pathophysiology (insulin resistance). Advanced maternal age (A), PCOS (B), previous LGA baby (D), high BMI (E) are all strong risk factors.)*
- **Screening:** Usually at 24-28 weeks gestation (earlier if high risk).
  - **One-step:** 75g Oral Glucose Tolerance Test (OGTT).
  - **Two-step:** 50g Glucose Challenge Test (GCT) followed by 100g OGTT if GCT abnormal.

- **Diagnosis:** Based on OGTT values (thresholds vary by guidelines).
- **Maternal Complications:** Pre-eclampsia, polyhydramnios, increased risk of operative delivery (C/S), birth trauma (due to macrosomia), increased risk of developing Type 2 DM later in life.
- **Fetal/Neonatal Complications:**
  - **Macrosomia (LGA):** Maternal hyperglycemia → fetal hyperglycemia → fetal hyperinsulinemia (insulin is a growth factor). Leads to increased fat deposition. *(Q40 Patient with T1DM. Fetal macrosomia (E) is a risk.)*
  - **Neonatal Hypoglycemia:** After delivery, maternal glucose supply ceases, but fetal hyperinsulinemia persists, leading to rapid drop in neonatal blood glucose.
  - **Respiratory Distress Syndrome (RDS):** Fetal hyperinsulinemia can delay surfactant production.
  - **Polycythemia & Hyperbilirubinemia (Jaundice).**
  - **Hypocalcemia, Hypomagnesemia.**
  - **Birth Trauma** (due to macrosomia, e.g., shoulder dystocia).
  - **Congenital Anomalies:** Risk increased if mother has pre-existing, poorly controlled diabetes *before and during early pregnancy*. Less so for GDM diagnosed later. Common ones: cardiac, neural tube defects (caudal regression is classic but rare). *(Q40B, Q102A congenital anomalies/NTD are risks, especially with pre-existing DM).*
  - *(Q103 With regards to GDM: No increased risk of miscarriages (D) is generally TRUE, especially if GDM is well-controlled and diagnosed later in pregnancy. Maternal islet cell damage (A) isn't the primary mechanism; it's insulin resistance. Congenital anomalies (B) are more with pre-gestational DM. Maternal insulin requirements increase after delivery (C) is FALSE; they plummet. Neonatal hyperglycemia (E) is FALSE; hypoglycemia is the concern.)*
- **Management:** Goal is euglycemia.
  1. **Dietary modification & Exercise:** First-line. *(Q41 Once GDM diagnosed, next step: Have blood sugar checks four times daily (C) is essential for monitoring. Diet and exercise are initiated simultaneously. Insulin (D) or Glyburide (E - oral hypoglycemic, less preferred now) are if diet/exercise fail.)*
  2. **Blood Glucose Monitoring:** Fasting and postprandial.
  3. **Pharmacotherapy (if targets not met):**
     - **Insulin:** Gold standard. Does NOT cross placenta. *(Q102C "Insulin crosses placenta" is INCORRECT.)*
     - **Oral hypoglycemics (e.g., Metformin, Glyburide):** Use varies by guideline; Metformin increasingly accepted. Glyburide crosses placenta, risk of neonatal hypoglycemia.
- **Intrapartum & Postpartum:**
  - Monitor glucose during labor.
  - Insulin requirements drop dramatically after delivery. *(Q102B Insulin requirements fall after delivery, correct).*
  - Screen for overt diabetes 6-12 weeks postpartum and periodically thereafter.
- **Preconception Counseling for Pre-existing Diabetes:** CRITICAL. Optimize glycemic control (HbA1c) *before* conception to reduce risk of congenital anomalies. Folic acid supplementation. *(Q40 T1DM preconception visit: Breech presentation (C) is not directly and significantly increased by well-controlled T1DM itself, unlike PET (A), anomalies (B), C/S risk (D), macrosomia (E).)*

XV. Anemia & Hyperemesis Gravidarum (Dr. Narges)*(Q19, 31, 81, 335, 404, 422, 474, 673, 745, 799)*

- **Anemia in Pregnancy:**
  - **Definition:** Hb <11 g/dL in 1st/3rd trimesters, <10.5 g/dL in 2nd trimester (due to maximal hemodilution). *(Q335 Anemia in 3rd trimester is Hb <11 g/dL (E)).*
  - **Iron Deficiency Anemia (IDA):** Most common type. Increased maternal/fetal demand.
    - **Diagnosis:** Low Hb, low MCV (microcytic), low serum ferritin (BEST indicator of iron stores - *Q31C Serum ferritin is most sensitive for iron depletion*), low serum iron, high TIBC.

        - **Treatment:** Oral iron (ferrous sulfate). Side effects: constipation, nausea. Take with Vitamin C for better absorption, avoid with antacids/milk. IV iron if severe, intolerant to oral, or poor response. Blood transfusion for acute severe anemia/hemorrhage. *(Q81 Regarding IDA: Blood transfusion may be necessary towards term (E) if severe/symptomatic. Iron demand increases (A). MCV <85fL confirms IDA (B) is too high a cut-off for MCV to confirm IDA; low ferritin is better. 10mg daily supplement (C) is for prevention, not treatment of established IDA. IV iron can cause allergic reaction (D), true.)*
    - **Folate Deficiency Anemia:** Macrocytic. Increased demand. Supplementation prevents neural tube defects.
    - **B12 Deficiency Anemia:** Macrocytic. Rare unless malabsorption/vegan.
    - **Hemoglobinopathies:** Sickle cell, thalassemia.
- **Hyperemesis Gravidarum (HG):** Severe, intractable nausea and vomiting of pregnancy leading to:
    - Weight loss (>5% of pre-pregnancy weight).
    - Dehydration.
    - Electrolyte imbalance (hypokalemia, hyponatremia, hypochloremia, metabolic alkalosis).
    - Ketonuria.
    - **Pathophysiology:** Not fully understood. hCG plays a role. Risk factors: multiple gestation, molar pregnancy, female fetus, history of HG.
    - **Differential Diagnosis:** Gastroenteritis, UTI, pancreatitis, cholecystitis, hepatitis, peptic ulcer, thyroid disease, neurological causes.
    - **Management (Stepwise):**
        1. **Dietary/Lifestyle:** Small frequent meals, avoid triggers, ginger.
        2. **Antiemetics:**
            - First-line: Pyridoxine (B6) +/- Doxylamine.
            - Others: Promethazine, Metoclopramide, Ondansetron (use in 1st tri debated due to small risk of cardiac defects, but often used for severe cases).
        3. **IV Fluids:** For dehydration, electrolyte correction.
        4. **Thiamine:** To prevent Wernicke's encephalopathy if prolonged vomiting.
        5. **Enteral/Parenteral Nutrition:** For severe, refractory cases.
        6. **Corticosteroids:** Short course for refractory HG (after 10 weeks usually).
    - *(Q474 Regarding hyperemesis, biochemical disturbances are common: Metabolic acidosis (E) is WRONG. Due to loss of gastric acid (HCl) and volume contraction, it's typically a **hypochloremic metabolic alkalosis** with hypokalemia. Hyponatremia (A) can occur. Hyperkalemia (B) is unlikely, hypokalemia is common. Raised urea (C) from dehydration. Raised calcium (D) not typical.)*

XVI. Thyroid Disorders in Pregnancy (Dr. Naser)*(Q88, 233, 621, 731)*

Thyroid function changes in pregnancy. Both hyper- and hypothyroidism can affect maternal/fetal outcomes.

- **Physiological Changes (Recap):**
    - Estrogen → ↑TBG → ↑Total T4/T3.
    - hCG (1st trimester) → mimics TSH → transient ↓TSH, slight ↑Free T4.
    - Free T4/T3 generally remain within normal (trimester-specific) ranges.
    - Increased iodine demand. *(Q233B half-life of TBG is not minutes, it's days. Q233C Total thyroid hormones INCREASE, not decrease. Q233A Maternal thyroxine (T4) does cross placenta prior to 12 weeks, this is correct. fT3 crossing is less clear/significant. Q233D Increased iodine demand is true. Q233E Untreated hypothyroidism requires urgent treatment, true).*
- **Hypothyroidism:**
    - **Causes:** Hashimoto's thyroiditis (most common), post-thyroidectomy/radioiodine, iodine deficiency.
    - **Maternal Risks:** Miscarriage, pre-eclampsia, placental abruption, PPH.

    - **Fetal/Neonatal Risks:** Impaired neurodevelopment (cretinism if severe untreated congenital hypothyroidism), preterm birth, low birth weight, stillbirth.
    - **Diagnosis:** ↑TSH, ↓Free T4. *(Q731 Best screen for hypothyroidism is TSH (A)).*
    - **Management:** Levothyroxine. Dose usually needs to be increased by ~25-30% in pregnancy. Monitor TSH every 4-6 weeks.
- **Hyperthyroidism:**
    - **Causes:** Graves' disease (most common).
    - **Maternal Risks:** Miscarriage, pre-eclampsia, heart failure, thyroid storm.
    - **Fetal/Neonatal Risks:** IUGR, preterm birth, stillbirth, fetal/neonatal hyperthyroidism (due to transplacental TSH receptor antibodies - TRAb), fetal goiter. *(Q88 Main complications for fetus in maternal hyperthyroidism: Growth restriction and fetal tachycardia (not bradycardia - D is wrong).)*
    - **Diagnosis:** ↓TSH, ↑Free T4/T3. TRAb positive in Graves'.
    - **Management:**
        - **Antithyroid Drugs (ATDs):**
            - **Propylthiouracil (PTU):** Preferred in 1st trimester (methimazole associated with embryopathy - aplasia cutis, choanal/esophageal atresia).
            - **Methimazole (MMI)/Carbimazole:** Preferred in 2nd/3rd trimesters (PTU has higher risk of hepatotoxicity).
        - Goal: Maintain Free T4 in upper normal range using lowest possible ATD dose. *(Q88E Therapy should maintain free T4 and T3 in high-normal or mildly elevated range to avoid fetal hypothyroidism from over-treatment. "Low normal" is incorrect target for maternal hyperthyroidism treatment).*
        - *(Q88A Surgical treatment is rarely first-line. Q88B Diagnosis by total T4 is wrong, use Free T4/TSH. Q88C More than half due to Graves' is true).*
- **Postpartum Thyroiditis:** Inflammation of thyroid within 1 year postpartum. Can cause transient hyperthyroidism, then hypothyroidism, then often recovery.

XVII. Polyhydramnios and Oligohydramnios (Dr. Amal)*(Q86, 104, 243, 355, 433, 434, 504, 505, 578, 603, 755)*

Imbalance in amniotic fluid volume.

- **Amniotic Fluid Dynamics:** Produced by fetal urine, lung secretions. Removed by fetal swallowing, intramembranous absorption.
- **Polyhydramnios (Too Much Fluid):**
    - **Definition:** Amniotic Fluid Index (AFI) >24-25 cm, or single deepest vertical pocket (SDP) >8 cm.
    - **Causes (Think Fetus Can't Swallow or Produces Too Much Urine):**
        - **Maternal:** Diabetes Mellitus (GDM/pre-existing - fetal polyuria), Rh isoimmunization (hydrops).
        - **Fetal:**
            - **GI obstruction:** Esophageal atresia, duodenal atresia *(Q433C Esophageal atresia IS a cause, not an exception)*, tracheo-esophageal fistula *(Q243D IS a cause)*.
            - **Neuromuscular disorders affecting swallowing:** Anencephaly, myotonic dystrophy *(Q243E Fetal muscular dystrophy IS a cause)*.
            - **Chromosomal abnormalities** (e.g., Trisomy 18, 21).
            - **High cardiac output states:** Sacrococcygeal teratoma, fetal anemia, chorioangioma of placenta *(Q243C Placental angioma IS a cause)*.
            - **Multiple gestation** (especially with TTTS recipient).
        - **Idiopathic** (most common, ~50-60%).
        - *(Q243 Causes of polyhydramnios except: Polycystic kidneys (B). Fetal polycystic kidney disease typically causes OLIGOhydramnios or anhydramnios due to impaired urine production.)*

    - *(Q755 Risk factor for polyhydramnios: History of maternal DM (C) is a classic one. HTN (A), thrombophilia (B), IUGR baby (D), steroid therapy (E) are not typical causes.)*
  - **Complications:** Preterm labor (uterine overdistension), PPROM, malpresentation, cord prolapse, placental abruption (rapid decompression), PPH (uterine atony).
- **Oligohydramnios (Too Little Fluid):**
  - **Definition:** AFI <5 cm, or SDP <2 cm.
  - **Causes (Think Fetus Can't Produce Urine or There's a Leak):**
    - **Uteroplacental Insufficiency:** Pre-eclampsia, chronic HTN, IUGR. *(Q355E Uteroplacental insufficiency is a cause).*
    - **PROM:** Premature rupture of membranes. *(Q355A Chronic amniotic leak is a cause).*
    - **Fetal Renal Abnormalities:** Bilateral renal agenesis (Potter's syndrome - *Q86E Renal agenesis is a cause, Q104A Potter's syndrome, Q104B Bilateral renal agenesis, Q434D Renal agenesis*), posterior urethral valves (PUV - *Q104D PUV*), bilateral multicystic dysplastic kidneys.
    - **Post-term pregnancy.** *(Q433A Postdate pregnancy is a cause).*
    - **Drugs:** NSAIDs, ACE inhibitors.
    - *(Q104 Congenital anomalies NOT associated with oligohydramnios: Duodenal atresia (C). This causes POLYhydramnios due to impaired swallowing. Q434 Malformation that does NOT induce polyhydramnios: Renal agenesis (D) causes OLIGO. Duodenal atresia (A), Anencephaly (B), Omphalocele (C - can be associated with swallowing issues), Spina bifida (E - often normal fluid or poly) are more likely with poly or normal fluid.)*
    - *(Q355 Conditions associated with oligohydramnios except: Chronic amniotic leak (A), Fetal renal agenesis (B), Urinary tract obstruction (D), Uteroplacental insufficiency (E) all cause oligo. Pulmonary hypoplasia (C) is a CONSEQUENCE of severe, prolonged oligohydramnios, not a cause of it. So C is the "except" as a cause.)*
  - **Complications:**
    - **Pulmonary Hypoplasia** (if severe and early onset).
    - **Fetal growth restriction.**
    - **Cord compression** (leading to variable decelerations, fetal distress).
    - **Limb contractures/Potter's facies** (if severe, prolonged).
    - **Increased risk of intrapartum complications.**

Brutal Honesty Checkpoint:

- **Ectopic:** Know risk factors, triad (pain, amenorrhea, bleeding), role of hCG/TVUS. Rupture = EMERGENCY.
- **GDM:** Insulin resistance is key. Macrosomia/neonatal hypoglycemia are classic fetal effects. Diet/exercise first, then insulin. Preconception control for pre-existing DM is VITAL.
- **Anemia/HG:** IDA is common, ferritin is best test. HG is severe N/V with weight loss/ketonuria/electrolyte imbalance; rule out other causes.
- **Thyroid:** Pregnancy state increases Total T4/T3, Free hormones usually normal. Screen with TSH. Know PTU for 1st tri hyperthyroidism, Levothyroxine for hypo.
- **Fluid Imbalances:** Polyhydramnios = fetus can't swallow/too much urine. Oligohydramnios = fetus can't make urine/leak. Know key causes for each. Pulmonary hypoplasia is a dread of oligo.

Obstetrics: Watching for Danger – Fetal Compromise and Monitoring

XVIII. Fetal Compromise (Dr. Fidaa) & XIX. Intrapartum Fetal Monitoring (Dr. Amal)*(Q6, 18, 69, 93, 106, 134, 148, 191, 196, 199, 200, 244, 259, 262, 266, 280, 283, 296, 347, 357, 359, 369, 406, 412, 419, 421, 429, 454, 501, 513, 557, 562, 563, 580, 584, 668, 690, 697, 698, 732, 750, 761)*

Fetal compromise (often loosely termed "fetal distress") means the fetus is not tolerating the intrauterine environment, usually due to hypoxia and/or acidosis. Intrapartum fetal monitoring aims to detect this early.

- **Causes of Fetal Compromise:**

    - **Uteroplacental Insufficiency:** Chronic (e.g., IUGR, pre-eclampsia, post-term pregnancy) or acute (e.g., placental abruption, uterine hyperstimulation). This is a big one.
    - **Cord Compression:** Prolapse, nuchal cord, oligohydramnios.
    - **Maternal Hypotension:** Epidural, supine hypotension, hemorrhage.
    - **Uterine Rupture.**
    - **Maternal Hypoxia/Acidosis.**
    - **Fetal Conditions:** Anemia, infection, cardiac arrhythmias.
- **Signs of Fetal Compromise:**
    - **Reduced Fetal Movements (RFM):** A crucial maternal observation. Always take seriously.
    - **Intrauterine Growth Restriction (IUGR):** Suggests chronic compromise.
    - **Abnormal Cardiotocography (CTG):** This is the mainstay of intrapartum monitoring.
    - **Abnormal Doppler Studies:** (e.g., absent/reversed umbilical artery end-diastolic flow).
    - **Meconium-Stained Liquor:** Can be a sign of past or ongoing fetal stress, especially in term/post-term infants. Risk of Meconium Aspiration Syndrome (MAS). *(Q18, Q93, Q191 link meconium with fetal compromise).*
    - **Fetal Scalp Blood pH < 7.20:** Indicates significant acidosis (more invasive, used when CTG is suspicious but not definitively abnormal). *(Q196 Fetal scalp pH 7.20-7.25 is Borderline (D), needs repeating. <7.20 is abnormal/acidotic.)*
- **Intrapartum Fetal Monitoring Tools & Interpretation:**
    - **Intermittent Auscultation (IA):** For low-risk women. Listen with Pinard or Doppler before, during, and after contractions.
    - **Continuous Cardiotocography (CTG):**
        - **Indications:** High-risk pregnancy, meconium, abnormal IA, augmentation of labor, epidural, etc.
        - **DR C BRAVADO (or similar mnemonic) for Interpretation:**
            - **D**efine **R**isk.
            - **C**ontractions: Frequency, duration, intensity (palpation/toco).
            - **B**aseline **RA**te: Normal 110-160 bpm. Tachycardia >160, Bradycardia <110 (or <100). *(Q200 Fetal tachycardia could result from: Maternal febrile illness (A) is a common cause. Hypothyroidism (B) more likely brady. Labetalol (C) can cause brady. Postmaturity (D) can be associated with distress which might manifest as tachy or brady. Pethidine (E) can reduce variability or cause transient sinusoidal patterns.)*
            - **V**ariability: Fluctuations around baseline. Normal 5-25 bpm. Reduced (<5bpm) or absent variability is concerning. Increased (>25bpm, saltatory) also can be.
            - **A**ccelerations: Reassuring. Rise of ≥15bpm for ≥15s.
            - **D**ecelerations:
                - **Early:** Benign, head compression. *(Q191 Classic cause is head compression (B)).*
                - **Variable:** Cord compression. Can be mild/moderate/severe depending on depth and duration. "Shouldering" (small accelerations before/after) can be reassuring. *(Q283 Commonest cause is cord compression (E)).*
                - **Late:** Uteroplacental insufficiency. Always concerning. *(Q6 Repetitive late decelerations indicate fetal hypoxia (B). Q370 Most common type of decelerations in laboring patient is often Variable (B) due to frequent cord compression. Early (A) are common but less significant. Late (D) are most ominous but hopefully less frequent.)*
                - **Prolonged:** ≥2 min. If >10 min = baseline change.
            - **O**verall assessment & Plan: Categorize (Normal, Suspicious, Pathological) and manage accordingly.
        - **Sinusoidal Pattern:** Smooth, sine-wave like. Ominous. Associated with severe fetal anemia (e.g., vasa previa, feto-maternal hemorrhage), severe hypoxia, or certain drugs. *(Q148 Sinusoidal trace associated with: Severe uteroplacental insufficiency (A) leading to hypoxia/anemia, Vasa previa (B) with bleeding, Placental abruption*

(C) with fetal anemia/hypoxia. Cord prolapse (D) usually causes profound bradycardia or severe variables. Congenital heart block (E) causes baseline bradycardia, not typically sinusoidal.)

- *(Q421 Positive Contraction Stress Test (CST) means: Late decelerations with at least 50% of contractions (E). This indicates uteroplacental insufficiency under stress.)*

- **Fetal Scalp Blood Sampling (FSBS):** Measures pH or lactate from a small scalp sample.
  - **Indications:** Suspicious or pathological CTG where delivery is not immediately indicated or feasible.
  - **Interpretation (pH):**
    - 7.25: Normal, continue monitoring.
    - 7.20-7.24: Borderline, repeat in 30 min or deliver if deteriorating. *(Q196: 7.20-7.25 is Borderline (D)).*
    - <7.20: Abnormal, expedite delivery.
  - **Contraindications:** Maternal infections (HIV, Hep B/C, active herpes), fetal bleeding disorders, prematurity <34 weeks. *(Q584 Regarding FSBS: PH 7.10 is NOT reassuring (D), it's severely acidotic. Indicated for fetal distress on CTG (A). PH 7.15 suggests fetal distress (B) - acidosis. PH 7.27 is reassuring (C). Contraindicated in HIV (E) is true.)*
- **Fetal Pulse Oximetry, ST-Segment Analysis (STAN):** Less commonly used, ancillary tools.

- **Management of Suspected Fetal Compromise (Intrauterine Resuscitation):**
  - **Call for help.**
  - **Maternal position change:** Left lateral (to relieve IVC/aortic compression).
  - **IV fluid bolus:** To improve hydration/placental perfusion.
  - **Oxygen to mother (by mask):** Limited evidence but often done.
  - **Stop oxytocin infusion (if running):** To reduce uterine activity.
  - **Tocolysis (e.g., terbutaline):** For uterine hyperstimulation or to buy time for C-section.
  - **Correct maternal hypotension.**
  - **If non-reassuring fetal status persists:** Expedite delivery (OVD or C-section). *(Q252 Primigravida, 40wks, fully dilated, +1, ROA, recurrent late decels. Best management: Instrumental delivery (D) if prerequisites met, as she's fully dilated with head at +1. Emergency C/S (B) if OVD not feasible/fails or more severe distress. Fetal scalp pH (A) might be considered if not immediately deliverable but late decels are ominous. Oxytocin (E) is contraindicated.)*
- **Non-Stress Test (NST):** Antenatal CTG monitoring for 20-30 min.
  - **Reactive (Reassuring):** ≥2 accelerations in 20 min.
  - **Non-Reactive:** Insufficient accelerations. May need further assessment (e.g., BPP, extend NST). Common causes: fetal sleep, medications, prematurity, fetal compromise. *(Q697 Commonest cause for non-reactive NST in a 37wk breech: Baby might be in a period of inactivity or quiet sleep (D). Hypoglycemia (C) or congenital malformations (E) are possible but sleep is more common. Previous C/S (A) or LGA (B) are not direct causes of non-reactivity.)*
- **Contraction Stress Test (CST) / Oxytocin Challenge Test (OCT):**
  - Induce contractions (oxytocin or nipple stimulation) and monitor for late decelerations.
  - **Negative (Reassuring):** No late decelerations.
  - **Positive (Non-reassuring):** Late decelerations with ≥50% of contractions. Suggests UPI.
  - **Equivocal/Suspicious:** Intermittent late decels or significant variables.
  - *(Q357 Routine antenatal care for primigravida at 34 weeks. Tests for assessment except: Ultrasound sonography (E). While US is used throughout pregnancy, for routine fetal wellbeing assessment at 34wks in an uncomplicated primigravida, NST (A), BPP (C), Kick counts (D) are more typical. CST (B) is less routine and more for specific concerns or post-dates.)*

XX. Intrapartum Maternal Monitoring (Dr. Amal)

As noted, this is deeply intertwined with "Labor" (#12). Key aspects of maternal monitoring include:

- **Vital Signs:** BP, HR, temperature, respiratory rate.

- **Labor Progress:**
  - **Cervical dilatation, effacement, station** (vaginal exams).
  - **Uterine contractions:** Frequency, duration, strength (palpation, tocodynamometer). *(Q183 Pressure by uterine contractions greatest at: Second stage (C), during active pushing, or transition. Active phase (B) also has strong contractions. Late phase (A) implies late first stage/transition.) (Q417 Optimal frequency of uterine contractions in 1st stage: 3-4 contractions/10 minutes (B) is generally considered adequate for active labor. 0-1 (A) is latent. 6-8 (C) or more (D, E) risks hyperstimulation.)*
- **Pain Relief:** Assessment of pain, effectiveness of analgesia.
- **Hydration & Nutrition:** IV fluids if needed, encourage oral intake if appropriate.
- **Bladder Care:** Encourage regular voiding, catheterize if needed (full bladder can impede descent/contractions).
- **Psychological Support.**
- **Monitoring for Complications:** Hemorrhage, infection, hypertensive crisis, etc.

Brutal Honesty Checkpoint:

- **Fetal Compromise is a Process:** Understand the cascade from placental issues or cord compression to hypoxia/acidosis and CTG changes.
- **CTG Interpretation is a SKILL:** Don't just look for "lates." Understand baseline, variability, accelerations, and the *types* of decelerations and what they mean. DR C BRAVADO is your friend.
- **Intrauterine Resuscitation:** Know the steps. They are logical if you understand the physiology.
- **FSBS:** Know when it's indicated and how to interpret pH – <7.20 is bad.
- **Maternal Monitoring:** Is about ensuring maternal safety and optimizing labor progress. The partogram is a key tool to track both maternal and fetal parameters in labor.

Gynecology

I. Premenstrual Syndrome (PMS) & Dysmenorrhea (Dr. Asmaa) (*Q15, 56, 124, 246, 255, 278, 343, 399, 469, 571, 653, 722, 723, 779)*

- **Dysmenorrhea – Painful Periods:**
  - **Primary Dysmenorrhea:** Painful menstruation in the ABSENCE of pelvic pathology. Starts within 6-12 months of menarche, once ovulatory cycles are established.
    - **Pathophysiology:** Excess prostaglandins (PGF2α, PGE2) in the endometrium → uterine hypercontractility, ischemia, and pain.
    - **Symptoms:** Crampy, colicky lower abdominal/pelvic pain, often radiating to back/thighs. Starts just before or with onset of menses, lasts 1-3 days. May have associated N/V, diarrhea, headache, fatigue. *(Q15 "Which is NOT a characteristic of primary dysmenorrhea": Symptoms start 3-4 days before menses (C) is too early for classic primary dysmenorrhea; it's typically just before or with onset. Improves with analgesics (A), normal pelvic exam (B), cause of school absence (D), bowel disturbances (E) are all consistent. Q56 "Regarding primary dysmenorrhea, one is true": It is spasmodic pain (C) is the hallmark. Q124 "Regarding primary dysmenorrhea, one is true": Can be relieved by contraceptive pills (D) - OCPs suppress ovulation and reduce prostaglandin production, thus effective. Q278 "Symptoms except Headache (E)": Headache can be associated, but the other symptoms (N/V (A), Fatigue (B), Constipation (C), Lower backache (D)) are very common. If "except" means least common or not directly caused, then headache might be the answer in some contexts, but it's often listed.)*
    - **Management:**
      1. **NSAIDs:** First-line (e.g., ibuprofen, naproxen, mefenamic acid). Inhibit prostaglandin synthesis. Start at onset of pain or just before, continue regularly for 2-3 days.
      2. **Hormonal Contraceptives (OCPs):** Suppress ovulation, thin endometrium, reduce prostaglandins. Very effective.

3. Lifestyle: Heat, exercise.

- **Secondary Dysmenorrhea:** Painful menstruation DUE TO underlying pelvic pathology. Often starts later in life (>25 yrs), pain may be longer, progressively worsen, or have different character.
  - **Causes:** Endometriosis (common!), adenomyosis, fibroids, PID, ovarian cysts, cervical stenosis, IUDs.
  - *(Q255 "Secondary dysmenorrhea can be caused by all except Genital prolapse (D)": Prolapse causes pressure/discomfort but isn't a classic cause of cyclical dysmenorrhea like fibroids (A), adenomyosis (B), pelvic congestion (C - though controversial), or PID (E).)*

- **Premenstrual Syndrome (PMS) / Premenstrual Dysphoric Disorder (PMDD):**
  - **Definition:** Cyclical recurrence of physical, mood, and/or behavioral symptoms during the **luteal phase** of the menstrual cycle, resolving within a few days of menses onset, with a symptom-free period in the follicular phase. Symptoms must cause distress or functional impairment.
  - **PMDD:** Severe form of PMS with prominent mood symptoms (e.g., marked depression, anxiety, irritability, affective lability).
  - **Pathophysiology:** Not fully understood. Likely abnormal neurotransmitter (especially serotonin) response to normal cyclical ovarian hormone fluctuations. NOT a hormone imbalance per se.
  - **Symptoms (Many!):**
    - **Mood:** Irritability, mood swings, depression, anxiety, tension, difficulty concentrating.
    - **Physical:** Bloating, breast tenderness (mastalgia), headache, fatigue, fluid retention, appetite changes/cravings.
    - *(Q399 "Components of premenstrual syndrome except Vaginal spotting (B)": Spotting is not a core PMS symptom. Breast tenderness (A), mood swings (C), bloating (D), headaches (E) are all classic.)*
    - *(Q469 PMS characteristics except Menorrhagia (B)": Menorrhagia (heavy bleeding) is a menstrual disorder, not a PMS symptom.)*
  - **Diagnosis:** Symptom diary for ≥2 cycles to confirm timing and impact. Rule out other medical/psychiatric conditions.
  - **Management (Stepwise):**
    1. **Lifestyle:** Regular exercise, stress reduction, balanced diet, limit caffeine/salt/alcohol.
    2. **Supplements:** Calcium, Vitamin B6 (pyridoxine) - evidence is mixed.
    3. **SSRIs:** First-line pharmacotherapy for moderate-severe PMS/PMDD (e.g., fluoxetine, sertraline). Can be given continuously or just during luteal phase. *(Q723 Management of PMS except: GnRH agonist is the best first line (C) is FALSE. SSRIs or lifestyle/OCPs are first-line. GnRH agonists are for very severe, refractory cases due to side effects.)*
    4. **Hormonal Contraceptives (OCPs):** Especially drospirenone-containing OCPs. Suppress ovulation.
    5. **GnRH Agonists:** For severe, refractory PMDD. Induce a "medical menopause." Add-back therapy needed if used long-term.
    6. **Danazol, Bromocriptine:** Older treatments, more side effects, rarely used now.

II. Subfertility (Dr. Nadia) *(Q139, 193, 257, 263, 276, 284, 306, 311, 324, 334, 342, 348, 386, 393, 409, 460, 464, 480, 507, 516, 587, 650, 746, 769, 797)*

Inability to conceive after 12 months of regular unprotected intercourse (or 6 months if female age ≥35).

- **Causes (The Big Categories):**
  1. **Male Factor (~30-40%):** Abnormal semen parameters (oligospermia, asthenospermia, teratospermia, azoospermia). Causes: varicocele, infections, endocrine disorders, genetic, ejaculatory dysfunction, idiopathic.
     - *(Q393 Regarding male factor infertility except: Oligozoospermia means total sperm number <5 million/ejaculate (B) is WRONG. WHO criteria define oligospermia as <15 million/mL or <39 million/ejaculate. Little to gain from exam if normal semen analysis (A) is often true but exam can still find varicocele etc.*

Variability in sperm quality (C) is true. Increased pregnancy rates after varicocele treatment (D) is controversial but often claimed. Retrograde ejaculation common in diabetics (E) is true.)

2. **Ovulatory Dysfunction (~20-30%):** Anovulation or oligo-ovulation.
    - **Causes:** PCOS (most common), hypothalamic amenorrhea (stress, weight loss, exercise), hyperprolactinemia, premature ovarian insufficiency (POI), thyroid disorders.
    - *(Q507 With regard to infertility, all true except: Ovulation induction is first-line for amenorrhea and ANORREXIA NERVOSA (D) - this is tricky. For anorexia, weight restoration and psychological support are primary. Ovulation induction is dangerous if severely underweight. PCOS accounts for 80-90% of anovulatory infertility (B) - true. Anovulation contributes to ~1/4 cases (A) - true. Mid-luteal progesterone to assess ovulation (C) - true. Idiopathic hypogonadotropic hypogonadism treated with gonadotropins (E) - true.)*
3. **Tubal Factor (~20-30%):** Tubal blockage or damage.
    - **Causes:** PID (chlamydia!), previous ectopic, endometriosis, adhesions from prior surgery.
4. **Uterine Factor (~5-10%):** Congenital anomalies (e.g., septate uterus, bicornuate uterus - *Q193 Bicornuate uterus diagnosed by HSG, screen Urinary system (C) for associated anomalies due to shared embryological origin (Mullerian and Wolffian ducts)*), fibroids (especially submucosal), polyps, Asherman's syndrome.
5. **Cervical Factor (Rare):** Stenosis, abnormal mucus.
6. **Unexplained Infertility (~10-15%):** All investigations normal.

- **Initial Investigations (Couple-Based):**
    - **Female:**
        - **History:** Menstrual cycle, prior pregnancies, PID, surgery, medical conditions.
        - **Ovulation Assessment:** Mid-luteal (Day 21 of 28-day cycle) serum progesterone. Basal body temperature (BBT) charting, LH kits less reliable for *confirming* ovulation.
        - **Ovarian Reserve:** Day 3 FSH/Estradiol, AMH, antral follicle count (AFC) – especially if >35 or risk factors for POI. *(Q92 Blood test best for ovarian reserve is AMH (E). FSH (A) is also used (Day 3).)*
        - **Tubal Patency: Hysterosalpingogram (HSG)** is first-line. Laparoscopy with dye test (chromopertubation) is gold standard if HSG equivocal or high suspicion of pelvic pathology. *(Q365 Most reliable method for cervical incompetence is vaginal ultrasound in index pregnancy (E), not HSG (B) or Hegar dilator (A). Q619 HSG can diagnose hydrosalpinx (B), subserous fibroids distorting cavity (C - though US better for fibroids), minimal adhesions (D - though laparoscopy better), ovarian cyst (E - NO, HSG is for tubes/uterus).)*
        - **Uterine Cavity Assessment:** TVUS, HSG, sonohysterography, hysteroscopy.
    - **Male:**
        - **Semen Analysis:** CRUCIAL. Volume, concentration, motility, morphology. If abnormal, repeat. *(Q516 Normal seminal fluid analysis except: Ideal result includes 60% sperm viability, 60% normal shape, 60% motility (E). WHO criteria are lower (e.g., motility >40% total or >32% progressive, morphology >4% normal forms). Low pH with low volume/sperm count (A) can suggest ejaculatory duct obstruction/seminal vesicle issues. Liquefaction within 20-30 min (B) is normal. Fluid from testes, seminal vesicles, prostate (C) is true. Azoospermia = no sperm (D) is true. So E likely incorrect by modern strict criteria standards.)*
- **Management (Depends on Cause):**
    - **Lifestyle:** Weight optimization, smoking cessation, reduce alcohol/caffeine.
    - **Ovulation Induction:**
        - **Clomiphene Citrate (CC):** SERM. First-line for anovulation (e.g., PCOS). Blocks estrogen receptors at hypothalamus/pituitary → ↑GnRH → ↑FSH/LH. Risk of multiple pregnancy, ovarian hyperstimulation syndrome (OHSS - rare with CC). *(Q257 Treatment of choice for hypogonadotropic hypogonadism is FSH and LH therapy (B - gonadotropins), not clomiphene (E) which requires intact HPO axis.)*
        - **Letrozole:** Aromatase inhibitor. Off-label, increasingly used as first-line for anovulation, especially PCOS. Similar/better efficacy than CC, lower multiple pregnancy risk.
        - **Gonadotropins (FSH +/- LH):** For hypothalamic amenorrhea or CC/Letrozole failure. Higher risk of multiples/OHSS. Requires close monitoring.

- **Intrauterine Insemination (IUI):** Prepared sperm placed directly into uterus. For mild male factor, cervical factor, unexplained infertility.
- **In Vitro Fertilization (IVF):** Eggs retrieved, fertilized in lab, embryo transferred to uterus. For tubal blockage, severe male factor, failed OI/IUI, advanced maternal age, POI (with donor eggs). *(Q157 Infertility secondary to endometriosis: Any endometriomas should be surgically removed prior to IVF (B) is controversial; depends on size/symptoms, risk of ovarian reserve damage. Surgical treatment can reduce ovarian reserve (C) is true. IVF is treatment of choice if ART needed (D) is true. Tubal patency confirmation doesn't exclude other fertility problems (E) is true. Surgical treatment improves spontaneous pregnancy rates (A) is true for moderate/severe.)*
- **Surgical Correction:** Tuboplasty, myomectomy, adhesiolysis, varicocelectomy.

- **Timing Intercourse for Conception:** Ovulation occurs ~14 days *before* next menses (or ~36 hrs after LH surge). Sperm viable for ~3-5 days, ovum for ~12-24 hrs. Fertile window is ~5 days before ovulation and day of ovulation. *(Q139 Lady with regular 30-day cycle, optimal time for conception intercourse: Ovulation around day 16 (30-14). So intercourse around Day 14-16. Day 16 (C) is best single option.)*

III. Polycystic Ovary Syndrome (PCOS) (Dr. Nadia)*(Q92, 145, 152, 228, 234, 313, 509, 561, 601, 615, 736)*

Common endocrine disorder in reproductive-aged women. Heterogeneous.

- **Rotterdam Criteria (Need ≥2 of 3):**
  1. **Oligo- and/or Anovulation:** (e.g., irregular cycles, amenorrhea).
  2. **Clinical and/or Biochemical Signs of Hyperandrogenism:** Hirsutism, acne, androgenic alopecia; elevated serum testosterone/DHEAS.
  3. **Polycystic Ovaries on Ultrasound:** ≥12 follicles (2-9mm) in each ovary and/or ovarian volume >10mL (in one ovary). (One PCO-morphology ovary is enough if other criteria met).
  - *(Q313 All true about PCOS except: LH-FSH ratio is always elevated (B) is FALSE. While often elevated (>2 or >3), it's not a diagnostic criterion and not always present. Increased risk of endometrial cancer (A) true. Each ovary may contain 20 cystic follicles (C) - fits PCO morphology (≥12 is criteria). Free estradiol increased (D) - often normal or slightly elevated due to peripheral aromatization of androgens. Androgen excess common (E) true.)*
  - *(Q736 In PCOS, all true except: Raised LH/FSH is necessary for diagnosis (C) is FALSE. It's a common finding but not a diagnostic criterion.)*
- **Pathophysiology (Complex, Not Fully Elucidated):**
  - **Insulin Resistance & Compensatory Hyperinsulinemia:** Central. Insulin stimulates ovarian androgen production and reduces SHBG production by liver → ↑free androgens.
  - **Abnormal GnRH Pulsatility:** Leads to ↑LH relative to FSH → ↑ovarian androgen production, impaired follicle development.
  - Genetic predisposition, environmental factors.
- **Clinical Features:**
  - Menstrual dysfunction (oligomenorrhea, amenorrhea, DUB).
  - Infertility (anovulatory).
  - Hyperandrogenism (hirsutism, acne, alopecia).
  - Obesity (common, but not universal; "lean PCOS" exists).
  - Acanthosis nigricans (skin marker of insulin resistance).
  - Polycystic ovaries on US.
  - *(Q152 Single female, secondary amenorrhea, increased facial hair, weight gain, US: "string of pearl." Shouldn't counsel her that: She is at increased risk of breast cancer (B). PCOS is primarily linked to endometrial cancer risk due to unopposed estrogen from anovulation. Endometrial hyperplasia risk (A) true. Sleep apnea risk (C) true due to obesity. Weight reduction improves symptoms (D) true. Glucose tolerance test needed (E) true due to insulin resistance.)*
- **Associated Long-Term Risks:**

    - Type 2 Diabetes Mellitus *(Q228 PCOS risk factors except: Type 1 diabetes (B). PCOS is strongly linked to Type 2 DM due to insulin resistance. Sleep apnea (A), impaired glucose tolerance (C), HTN (D), dyslipidemia (E) are all associated metabolic risks.)*
    - Gestational Diabetes.
    - Cardiovascular Disease (HTN, dyslipidemia).
    - Endometrial Hyperplasia & Cancer (due to chronic anovulation → unopposed estrogen).
    - Sleep Apnea.
    - Depression/Anxiety.
- **Investigations:**
    - Hormonal profile: Testosterone, DHEAS, LH, FSH, prolactin, TSH (to rule out other causes).
    - OGTT / HbA1c (screen for IGT/DM).
    - Lipid profile.
    - TVUS.
    - *(Q615 Serum endocrinology of PCOS except: Increased sex hormone binding globulin (B) is FALSE. SHBG is typically DECREASED by hyperinsulinemia, leading to higher free androgen levels. Raised fasting insulin (A) common. Normal FSH (C) common. Raised estrone (D) from peripheral aromatization. Raised or normal LH (E) common.)*
- **Management (Tailored to Symptoms & Goals):**
    - **Lifestyle Modification:** WEIGHT LOSS (if overweight/obese) is cornerstone. Diet, exercise. Improves insulin sensitivity, can restore ovulation.
    - **Menstrual Irregularity / Endometrial Protection:**
        - Combined OCPs: Regulate cycles, provide progestin to protect endometrium, reduce androgens (↑SHBG, ↓LH).
        - Progestins (cyclical or continuous).
    - **Hirsutism/Acne:**
        - OCPs.
        - Anti-androgens (e.g., spironolactone, cyproterone acetate - often with OCPs).
        - Topical treatments, cosmetic measures.
    - **Infertility (Ovulation Induction):**
        - Letrozole or Clomiphene Citrate: First-line.
        - Metformin: May improve insulin sensitivity and ovulatory function, often adjunct.
        - Gonadotropins.
        - Ovarian drilling (laparoscopic).
        - IVF.
    - *(Q234 PCOS, all true except: Hirsutism found in 50% of patients (D) is an underestimate, it's usually higher (60-80%). Endometrial thinning (E) is FALSE; chronic anovulation leads to endometrial hyperplasia due to unopposed estrogen.)*

IV. Amenorrhea (Dr. Islam Al-awamleh)*(Q16, 60, 112, 131, 137, 181, 192, 201, 349, 382, 392, 396, 463, 487, 510, 542, 594, 602, 622, 670, 734, 735, 742, 774, 778)*

Absence of menstruation.

- **Primary Amenorrhea:**
    - No menses by age 15 (or 13 if no secondary sexual characteristics).
    - **Causes (Workup based on presence/absence of uterus & breasts):**

- **Chromosomal abnormalities:** Turner Syndrome (45,XO - streak gonads, no breasts/uterus normal or small, primary amenorrhea). *(Q131 Regarding Turner syndrome, incorrect: Incidence rises with increasing maternal age (B) is FALSE; it's usually sporadic. Karyotype 45,XO (A) true. Cystic hygroma (C) true. Intelligence normal (D) true. Estrogen therapy increases bone mineral density (E) true.)*
- **Hypothalamic/Pituitary (Hypogonadotropic Hypogonadism):** Kallmann syndrome (anosmia + GnRH deficiency - *Q181 Kallmann's characterized by Amenorrhea (C) and often anosmia, due to GnRH deficiency, leading to hypogonadotropic hypogonadism. Optic atrophy (D) is not classic.*), constitutional delay, CNS tumors, chronic illness, stress/anorexia.
- **Ovarian (Hypergonadotropic Hypogonadism):** Turner syndrome, POI, gonadal dysgenesis (e.g., Swyer syndrome 46,XY).
- **Anatomical (Outflow Tract Obstruction):** Imperforate hymen, transverse vaginal septum, Mullerian agenesis (MRKH syndrome - absent uterus/upper vagina, normal ovaries/secondary sexual characteristics). *(Q112 16yo, Tanner V breasts/pubic hair, blind vagina, 46XX. Most likely diagnosis: Mullerian agenesis (C).) (Q353 Most common anomaly from Mullerian duct development/canalization: Imperforate hymen (C) is a possibility for outflow obstruction. Vaginal septum (A) also. Uterine anomalies like bicornuate uterus (B) or vaginal agenesis (E) are Mullerian. Hermaphroditism (D) is a DSD, not purely Mullerian.) (Q602 Regarding imperforate hymen except: Secondary sexual development is usually absent (E) is FALSE. Ovarian function is normal, so secondary sexual characteristics develop normally. Presents typically at 14-16 (A) true. Normal karyotype (B) true. Primary amenorrhea (C) true. Urinary retention (D) possible due to hematocolpos pressure.)*
    - **Androgen Insensitivity Syndrome (AIS) (46,XY):** Testes produce androgens, but receptors are insensitive. Female external genitalia, blind vagina, absent uterus, intra-abdominal testes. Develop breasts (peripheral aromatization of androgens).

- **Secondary Amenorrhea:**
    - Cessation of menses for ≥3 months (if previously regular) or ≥6 months (if previously irregular).
    - **ALWAYS RULE OUT PREGNANCY FIRST!**
    - **Causes (Categorize by HPO Axis + Uterus):**
        - **Hypothalamic:** Functional hypothalamic amenorrhea (stress, excessive exercise, low body weight/anorexia nervosa - *Q16 Most common pathologic cause of amenorrhea in adolescent females is Anorexia nervosa (A) among the options. Q542 Causes of secondary amenorrhea except: Mullerian agenesis (C) causes PRIMARY amenorrhea.*), CNS tumors.
        - **Pituitary:** Prolactinoma (hyperprolactinemia → ↓GnRH), Sheehan's syndrome (postpartum pituitary necrosis - *Q548 Most common cause of Sheehan is Massive postpartum hemorrhage (C)*), empty sella syndrome *(Q137 Empty sella syndrome: Induction of ovulation is contraindicated (C) is FALSE; ovulation can often be induced if pituitary function is otherwise okay or with gonadotropins if deficient)*, other pituitary tumors.
        - **Ovarian:** PCOS (common!), Premature Ovarian Insufficiency (POI - menopause <40 yrs). *(Q392 Premature ovarian failure associated with all except: Early Menarche (E). POI is associated with things like autoimmune disease, genetic predispositions (Fragile X premutation), chemo/radio. Early menarche isn't a risk.)*
        - **Uterine:** Asherman's syndrome (intrauterine adhesions, often post-D&C/endometritis). *(Q146 P0+3, 6mo amenorrhea after 3 curettages. Not at risk of: Premature menopause (B). Repeated curettages put her at high risk for Asherman's (infertility (A), abnormal placentation if she gets pregnant (C, D), early pregnancy loss (E)).)*
        - **Other Endocrine:** Thyroid disorders (hypo/hyper), Cushing's, CAH.
- **Workup of Amenorrhea:**
    1. Pregnancy test (hCG).
    2. History & Physical exam (inc. secondary sexual characteristics, signs of hyperandrogenism/virilization, pelvic exam).
    3. Initial labs: FSH, LH, estradiol, prolactin, TSH.
    4. Progestogen challenge test: (If uterus present and initial labs non-diagnostic). Withdrawal bleed → adequate estrogen, patent outflow tract (suggests anovulation, e.g., PCOS). No bleed → hypoestrogenic or outflow obstruction.

5. Further investigations based on suspected cause: Karyotype (if primary/POI), imaging (pelvic US, brain MRI), androgen levels, etc.

Brutal Honesty Checkpoint:

- **PMS/Dysmenorrhea:** Know primary vs. secondary dysmenorrhea (prostaglandins vs. pathology). PMS/PMDD is luteal phase, serotonin-linked. NSAIDs/OCPs for dysmenorrhea; SSRIs/OCPs/lifestyle for PMS/PMDD.
- **Subfertility:** Think male, ovulatory, tubal, uterine. Semen analysis, ovulation check (Day 21 progesterone), HSG are key initial steps.
- **PCOS:** Rotterdam criteria. Insulin resistance is central. Manage based on goals (cycle control, fertility, hirsutism). Long-term DM/endometrial Ca risk.
- **Amenorrhea:** Primary vs. Secondary. PREGNANCY TEST FIRST for secondary. Then categorize by HPO axis + uterus.

Gynecology: When Support Fails, Control is Lost, Skin Issues Arise, and Family Planning

Now we're dealing with structural integrity, bladder control, concerning skin changes, and the crucial aspect of contraception. These are common clinical scenarios requiring a solid understanding of anatomy, physiology, and patient counseling.

V. Pelvic Organ Prolapse (POP) (Dr. Ayman)*(Q219, 302, 339, 415, 555, 597, 635, 638, 737, 738)*

Descent of one or more pelvic organs (bladder, uterus/vaginal vault, rectum, bowel) into or out of the vagina. It's a hernia through the pelvic floor.

- **Anatomy of Pelvic Support – The Layers Matter:**
  - **Level I Support (Apical):** Uterosacral-cardinal ligament complex. Supports uterus and vaginal apex. Defects → uterine/vault prolapse, enterocele. *(Q737 Level 1 support is Cardinal and Uterosacral complex (C)).*
  - **Level II Support (Mid-vaginal):** Paravaginal attachments (fascia to arcus tendineus fasciae pelvis). Supports lateral vaginal walls. Defects → cystocele, rectocele.
  - **Level III Support (Distal):** Perineal body, urogenital diaphragm. Supports distal vagina and perineum.
  - **Pelvic Diaphragm:** Levator ani muscles (pubococcygeus, iliococcygeus, ischiococcygeus/coccygeus) and coccygeus muscles. Crucial for overall support. *(Q219 Pelvic diaphragm composed of all except: Obturator internus muscle (C). This is a lateral wall muscle of the pelvis, not part of the diaphragm. Q339 Pelvic diaphragm composed of all except Obturator internus (C) - same concept.)*
- **Types of Prolapse (Named by Descending Organ):**
  - **Anterior Vaginal Wall Prolapse:**
    - **Cystocele:** Bladder descent.
    - **Urethrocele:** Urethral descent.
  - **Posterior Vaginal Wall Prolapse:**
    - **Rectocele:** Rectal descent.
    - **Enterocele:** Small bowel descent (often into upper posterior vagina, especially post-hysterectomy).
  - **Apical Prolapse:**
    - **Uterine Prolapse:** Uterine descent.
    - **Vaginal Vault Prolapse:** Post-hysterectomy apical descent.
  - **Procidentia:** Complete uterine prolapse where cervix and uterus descend outside the introitus. *(Q738 Procedentia is Stage 4 prolapse (A) by POP-Q staging, or generally the most severe form.)*
- **Risk Factors:**
  - Vaginal childbirth (parity, traumatic delivery, macrosomia, forceps).
  - Advancing age.

    - Menopause/estrogen deficiency (tissue atrophy).
    - Obesity.
    - Chronic increased intra-abdominal pressure (chronic cough, constipation, heavy lifting).
    - Connective tissue disorders.
    - Family history / genetics.
    - Previous pelvic surgery (especially hysterectomy for vault prolapse risk).
    - *(Q638 Cause for uterovaginal prolapse in a 29yo P2 with inguinal hernia repair and smoker: Multiparity (A) is a direct risk. Age (B) - 29 is young, but parity matters more. Inguinal hernia repair (C) isn't a direct cause of POP itself, though weak connective tissue could underlie both. Collagen deficiency (D) is a plausible underlying factor. Smoking (E) can worsen tissue quality/cough.) Given the options, multiparity is the most direct obstetric risk factor listed.*
- **Symptoms:**
    - Vaginal bulge/pressure/"something coming down."
    - Urinary: Stress incontinence, urgency, frequency, incomplete emptying, need to splint.
    - Bowel: Constipation, incomplete emptying, need to splint (digitation).
    - Sexual dysfunction: Dyspareunia, altered sensation.
    - Backache/pelvic pain.
- **Diagnosis:** Pelvic exam, often with Valsalva. POP-Q staging system is the objective standard.
- **Management:**
    - **Conservative:**
        - **Observation:** If asymptomatic or mild.
        - **Pelvic Floor Muscle Training (PFMT/Kegels):** First-line for mild-moderate.
        - **Pessaries:** Intravaginal devices to support prolapsed organs. Good for women who are poor surgical candidates or wish to avoid surgery. Many types (ring, Gellhorn, etc.).
        - **Lifestyle:** Weight loss, treat constipation/cough.
    - **Surgical:** For symptomatic prolapse failing conservative management.
        - **Anterior Repair (Colporrhaphy):** For cystocele.
        - **Posterior Repair (Colporrhaphy):** For rectocele.
        - **Apical Suspension Procedures:**
            - **Vaginal:** Sacrospinous fixation, uterosacral ligament suspension.
            - **Abdominal/Laparoscopic/Robotic:** Sacrocolpopexy (gold standard for vault prolapse, uses mesh).
        - **Hysterectomy:** Often performed concurrently if uterine prolapse and no desire for future childbearing/uterine preservation.
        - **Obliterative Procedures (Colpocleisis):** Vaginal closure. For frail, elderly women not desiring future sexual function. Highly effective.
        - *(Q555 Ligaments providing most support to uterus (preventing prolapse): Cardinal ligaments (D) and uterosacral ligaments form the main Level I support.)*

VI. Urinary Incontinence (UI) (Dr. Ayman)*(Q9, 98, 185, 221, 267, 295, 407, 540)*

Involuntary leakage of urine. Common, distressing.

- **Types (KNOW THESE DIFFERENTLY!):**
    - **Stress Urinary Incontinence (SUI):** Leakage with increased intra-abdominal pressure (cough, sneeze, laugh, exercise).

        - **Pathophysiology:** Urethral hypermobility (weak pelvic floor/fascial support) OR intrinsic sphincter deficiency (ISD - damaged sphincter).
        - *(Q9 Regarding UI: Genuine Stress Incontinence can be diagnosed on history alone (A) - History is suggestive, but exam (cough stress test) and sometimes urodynamics are needed for confirmation and to rule out other types. This is likely considered false by strict criteria.)*
        - *(Q98 68yo lady with leakage on coughing/sneezing: Stress incontinence (E) is the classic description.)*
    - **Urgency Urinary Incontinence (UUI) / Overactive Bladder (OAB):** Leakage preceded by or associated with urgency (sudden, compelling desire to void that is difficult to defer). OAB includes urgency, frequency, nocturia, +/- UUI.
        - **Pathophysiology:** Detrusor overactivity (DO) - involuntary bladder contractions during filling. Can be idiopathic or neurogenic.
        - *(Q9B Detrusor instability (overactivity) can be treated with anticholinergic medication - TRUE, these are first-line pharmacotherapy. Q9C TVT is ideal for detrusor instability - FALSE, TVT is for SUI. Q48 Commonest cause of detrusor overactivity: Idiopathic (D) is very common. Q407 Commonest cause of detrusor overactivity is Idiopathic (D).)*
    - **Mixed Urinary Incontinence (MUI):** Combination of SUI and UUI symptoms.
    - **Overflow Incontinence:** Leakage due to bladder overdistension (chronically full bladder).
        - **Pathophysiology:** Impaired detrusor contractility (e.g., neurogenic, drugs) OR bladder outlet obstruction (e.g., severe prolapse, stricture, BPH in men).
        - Symptoms: Dribbling, weak stream, hesitancy, feeling of incomplete emptying.
    - **Functional Incontinence:** Cognitive or physical impairment preventing timely toileting.
- **Investigations:**
    - History (type of leakage, triggers, pads used, fluid intake, voiding diary).
    - Pelvic exam (atrophy, prolapse, cough stress test).
    - Urinalysis & culture (rule out UTI).
    - Post-void residual (PVR) volume (assess bladder emptying).
    - **Urodynamic Studies (UDS):** For complex cases, diagnostic uncertainty, prior to surgery, failed conservative treatment. Measures bladder pressures during filling/voiding, identifies DO, SUI, voiding dysfunction. *(Q9D Urodynamics only required if surgery fails - FALSE, often done before surgery to confirm diagnosis and plan procedure, or if diagnosis unclear.)*
- **Management:**
    - **Lifestyle (All Types):** Fluid management, timed voiding, bladder training, weight loss, caffeine reduction, treat constipation.
    - **SUI:**
        - **Conservative:** PFMT (first-line), continence pessaries, vaginal cones.
        - **Medical:** Duloxetine (SNRI - modest efficacy, side effects).
        - **Surgical:** Mid-urethral slings (TVT, TOT - very effective - *Q9E TVT only under GA - FALSE, can be done under regional/local*), Burch colposuspension (older, abdominal), bulking agents (for ISD).
    - **UUI/OAB:**
        - **Conservative:** Bladder training (first-line).
        - **Medical:**
            - **Antimuscarinics/Anticholinergics** (e.g., oxybutynin, tolterodine, solifenacin): First-line pharmacotherapy. Block muscarinic receptors on detrusor → reduce contractility. Side effects: dry mouth, constipation, blurred vision, cognitive issues. *(Q639 Oxybutynin (anticholinergic) contraindicated in all except: Ulcerative colitis (D). Contraindicated in urine retention (A), myasthenia gravis (B - can worsen weakness), narrow-angle glaucoma (E). Liver disease (C) needs caution but not absolute CI like the others. Thus D is the best exception.)*

        - **Beta-3 Agonists** (e.g., mirabegron): Relax detrusor. Fewer anticholinergic side effects.
    - **Other:** Botox injections into detrusor, sacral neuromodulation, percutaneous tibial nerve stimulation (PTNS).

VII. Benign & Malignant Conditions of the Vulva & Vagina (Dr. Naser)*(Q20, 23, 135, 235, 439, 440, 608, 658, 677, 703, 775, 792)*

Itches, lumps, bumps, and scary stuff. Biopsy is your friend for suspicious lesions.

- **Benign Vulval Conditions:**
    - **Lichen Sclerosus:** Chronic inflammatory skin condition.
        - **Symptoms:** Intense pruritus (classic!), soreness, dyspareunia.
        - **Signs:** Thin, white, crinkled ("cigarette paper" or "parchment-like") skin; erosions, fissures, ecchymoses. Can lead to scarring, introital stenosis, loss of labia minora/clitoral hood (architectural changes).
        - **Diagnosis:** Clinical, biopsy to confirm and rule out malignancy (small risk of SCC).
        - **Management:** Ultrapotent topical corticosteroids (e.g., clobetasol propionate) are mainstay. Emollients. Long-term follow-up. *(Q677 Regarding lichen sclerosus, true except: Surgical excision is the main treatment (C) is FALSE. Topical steroids are main. Pruritus (A), affects children/adults (B), potent steroids help (D), increased vulval cancer risk (E) are all true.)*
    - **Lichen Planus:** Inflammatory, can affect vulva, vagina, mouth. Erosive form is painful.
    - **Vulval Dermatitis (Eczema):** Irritant or allergic.
    - **Bartholin's Gland Cyst/Abscess:** Blockage of Bartholin's duct → cyst. Infection → abscess.
        - **Management:** Marsupialization or Word catheter for recurrent cysts/abscesses. Antibiotics for abscess.
    - **Vulval Vestibulitis/Vestibulodynia:** Pain on touch/pressure to vestibule. Cause of dyspareunia.
- **Vaginal Conditions:**
    - **Bacterial Vaginosis (BV), Candidiasis, Trichomoniasis:** (Covered also under Lower Genital Infections). *(Q4 Bacterial Vaginosis: Causes fishy discharge from bacterial amine production (D) is correct. Not rare (A). Often asymptomatic (B false). Little inflammatory reaction (C false - clue cells, not WBCs). Clotrimazole (E) is for candida.) (Q113 Regarding Trichomonas vaginalis, incorrect: No vulvovaginal irritation (B) is FALSE; it causes significant irritation/pruritus. Yellow, frothy discharge (A), pH >4.5 (C), motile flagellates on wet mount (D), treat partner (E) are all true.) (Q184 26yo, itching/burning, pH 5.5. Most likely: Bacterial vaginosis (E) or Trichomonas (D). Yeast (C) usually normal pH. Given options, BV is very common with elevated pH. Trichomonas also fits with elevated pH and irritation.)*
    - **Atrophic Vaginitis (Genitourinary Syndrome of Menopause - GSM):** Due to estrogen deficiency. Symptoms: dryness, itching, dyspareunia, urinary symptoms. Signs: pale, thin, dry vaginal mucosa, loss of rugae, petechiae. Treatment: topical estrogen. *(Q20 Most effective treatment of vulvar pruritus with atrophic vulvitis is Topical estrogen therapy (D)).*
- **Anatomy:** *(Q23 Main blood supply of vulva is Pudendal artery (A). Q135 Anatomy of vulva, incorrect: Labia Minora contain numerous sweat glands (C) is FALSE; they are rich in sebaceous glands but lack sweat glands and hair follicles.)*
- **Pre-malignant & Malignant Vulval Conditions:**
    - **Vulval Intraepithelial Neoplasia (VIN):** Dysplastic changes. HPV-related (usual VIN) or differentiated VIN (dVIN, often associated with lichen sclerosus). Risk of progression to SCC.
    - **Squamous Cell Carcinoma (SCC):** Most common vulval cancer (>90%).
        - **Risk Factors:** HPV infection (for usual type), chronic inflammatory conditions (lichen sclerosus for dVIN type), smoking, immunodeficiency.
        - **Presentation:** Pruritus (commonest - *Q439, Q792 Most common symptom of vulvar Ca is Pruritus (E)*), lump, ulcer, bleeding, pain.
        - **Diagnosis:** BIOPSY of suspicious lesion.

        - **Management:** Surgery (wide local excision, radical vulvectomy) +/- lymph node dissection (inguinofemoral) +/- radiotherapy/chemotherapy. *(Q235 En bloc radical vulvectomy... modification due to all except: Minimize need for postoperative radiotherapy (A) is a GOAL of modification (e.g., sentinel node biopsy, less radical surgery for smaller tumors), not a factor driving modification itself. Disease affecting younger women (B), awareness of psychosexual consequences (C), concern about morbidity (D), long-term hospitalization (E - reducing this is a goal) are reasons for less radical approaches.) (Q440 45yo, 3cm solid vulval lesion, moderate SCC, no mets. Proper next step: Radical vulvectomy with ipsilateral inguinofemoral lymphadenectomy (C) is a standard approach for Stage IB/II depending on depth/location if unilateral. Bilateral LND (E) if midline or bilateral disease. Wide local excision (B) may be for VIN or very early invasive.)*
- **Vaginal Intraepithelial Neoplasia (VAIN) & Vaginal Cancer (Rare):** Mostly SCC, HPV-related.

VIII. Contraception (Dr. Fidaa)*(Q10, 50, 52, 75, 114, 127, 138, 177, 189, 207, 250, 310, 325, 414, 473, 484, 485, 514, 520, 524, 627, 646, 667, 681, 710, 729, 730, 758, 770)*

Preventing pregnancy. Know types, mechanisms, efficacy, key CIs and side effects.

- **Highly Effective (LARCs - Long-Acting Reversible Contraceptives):**
    - **Implants (e.g., Nexplanon):** Progestogen-only. Subdermal. Lasts 3 years. Mechanism: inhibits ovulation, thickens cervical mucus. Side effect: irregular bleeding common.
    - **Intrauterine Devices (IUDs):**
        - **Copper IUD (Cu-IUD):** Non-hormonal. Lasts 5-10 years. Mechanism: spermicidal (copper ions), inhibits implantation. Can cause heavier/more painful periods. *(Q52 Discontinuing IUCD mostly due to Abnormal uterine bleeding (C) or pain.) (Q127 Failure rate of Copper IUCD is 1-2 per 100 women year (B) is in the correct range, though modern ones are closer to <1.)*
        - **Levonorgestrel IUS (LNG-IUS, e.g., Mirena, Kyleena):** Progestogen-releasing. Lasts 3-7 years (depending on type). Mechanism: thickens cervical mucus, thins endometrium, +/- inhibits ovulation. Reduces menstrual bleeding (often leads to amenorrhea). *(Q485 Mirena contains Levonorgestrel (C).)*
    - **Contraindications to IUD/IUS:** Current PID, unexplained vaginal bleeding, uterine cavity distortion, cervical/endometrial cancer. *(Q681 Contraindications to IUD use: Active pelvic inflammatory disease (C). Smoking (A), multiparity (B), ovarian cyst (D), previous hysteroscopy (E) are not absolute CIs unless other specific issues.)*
- **Hormonal Methods:**
    - **Combined Oral Contraceptives (COCPs):** Estrogen + Progestogen.
        - **Mechanism:** Inhibit ovulation (main), thicken cervical mucus, thin endometrium.
        - **Benefits:** Regulate cycles, reduce dysmenorrhea/menorrhagia, reduce risk of ovarian/endometrial/colorectal cancer, improve acne. *(Q114 COCPs do NOT reduce risk of Cervical cancer (E); may slightly increase it. Reduce benign breast disease (A), colorectal Ca (B), salpingitis (C), PMS (D) are known benefits or plausible effects.)*
        - **Risks/Side Effects:** VTE (MAJOR!), stroke, MI (especially if smoker >35), HTN, breast tenderness, nausea, mood changes, breakthrough bleeding. *(Q10 OCs may increase risk of Hepatic adenoma (B). They are protective against ovarian (D) and endometrial (E) cancer. They reduce fibrocystic breast disease (A) and salpingitis (C).) (Q50 Side effects of COCPs except: Functional ovarian cyst (A). COCPs suppress ovulation and typically reduce functional ovarian cysts.) (Q325 Main mode of action of OCPs is Inhibition of LH surge (B) thus preventing ovulation.)*
        - **Absolute Contraindications (UKMEC 4):** Current/history of VTE, known thrombogenic mutation, ischemic heart disease, stroke, migraine with aura, active liver disease, current breast cancer, uncontrolled HTN, smoker ≥35 and ≥15 cigs/day. *(Q138 Not an indication for discontinuing COCPs: Diagnosis of breast cancer in a first-degree relation (C). Personal history of breast ca is a CI, but family history is not an absolute CI for starting/continuing in many cases, though needs assessment. Hypertension (A), focal migraine (B), jaundice (D - if due to liver disease/cholestasis), personal hx of DVT (E) are reasons to stop/not start.) (Q250 Absolute CIs for COCPs except: Medically treated gall bladder disease (E). This is often a relative CI or needs caution, not usually absolute like complicated valvular heart disease (A), active viral hepatitis (B), migraine with aura (C), or current breast cancer (D).)*

        - *(Q646 Regarding COCP, one is true: Ovulation is prevented by the progestogen (D) - Progestogen primarily suppresses LH surge, estrogen suppresses FSH. Both contribute. "Relatively safe up to 40 in smoker" (A) - risk increases significantly. "20-30% women in 40s use COCP" (B) - prevalence varies. "0.2-0.5mg ethinyl estradiol" (C) - doses are usually 20-35 micrograms. "More effective in epilepsy on medication" (E) - enzyme-inducing AEDs reduce COCP efficacy.) Q646 is tricky, D is the closest to "true" regarding mechanism.*
    - **Progestogen-Only Pills (POP, "Mini-pill"):**
        - **Mechanism:** Thicken cervical mucus (main), +/- inhibit ovulation (less consistent than COCPs). Must be taken at same time daily.
        - **Suitable if estrogen CI** (e.g., migraine with aura, VTE risk, breastfeeding).
        - Side effect: Irregular bleeding common.
        - *(Q207 Progesterone-only pills work as contraceptives by: Altering the cervical mucus (B). Suppressing ovulation (A) is less consistent than COCPs. Not endometrial hyperplasia (C), not reducing libido (D), not spermicide (E).) (Q627 Contraindications to POPs except: Mild hypertension (D). POPs are generally safe with HTN where COCPs might be CI. Depression (A) - caution. Unexplained uterine bleeding (B) - investigate first. Acne (E) can be worsened by some POPs. Migraine headaches (C) - POPs are generally safe, unlike COCPs with aura.)*
    - **Injectable (e.g., Depo-Provera - DMPA):** Progestogen-only. Every 12-13 weeks.
        - **Mechanism:** Inhibits ovulation, thickens mucus.
        - Side effects: Irregular bleeding then often amenorrhea, weight gain, mood changes, delayed return to fertility, bone mineral density reduction (reversible).
    - **Patch, Vaginal Ring:** Combined hormonal, similar to COCPs.
- **Emergency Contraception (EC):**
    - **Levonorgestrel ("Morning-after pill"):** Up to 72 hrs (some efficacy up to 120 hrs).
    - **Ulipristal Acetate:** Up to 120 hrs (5 days). More effective than levonorgestrel.
    - **Copper IUD:** Most effective. Up to 5 days after unprotected sex or 5 days after earliest estimated ovulation.
- **Barrier Methods:** Condoms (male/female), diaphragms, caps. Protect against STIs (condoms).
- **Sterilization (Permanent):**
    - **Female:** Tubal ligation/occlusion.
    - **Male:** Vasectomy.
- **Failure Rates:** *(Q127 Regarding failure rates per 100 women year, not correct: Mirena 3 (C) is too high; typical use failure is <1 (e.g., 0.2-0.7). COCPs 0.1-1 (A) is a wide range (perfect vs typical use). Copper IUCD 1-2 (B) - modern ones closer to 0.6-0.8. Diaphragm 1-15 (D) plausible. Vasectomy 0.02 (E) very low, plausible for perfect use.)*
- **Natural Family Planning/Fertility Awareness:** Higher failure rates.
- **IUCDs and PID:** *(Q189 Method associated with lowest incidence of PID: Condom (A). IUCD (B) has small transient risk at insertion. OCPs (C) are protective. Vaginal foam (D) no protection. Norplant (E - implant) no direct effect on PID risk. Condoms actively prevent STIs which cause PID. OCPs also reduce PID risk by thickening mucus.) Q189 is asking for lowest incidence, which implies prevention. Condom or OCPs are good candidates. If the question implies inherent property of the method not leading to PID, then OCPs or implants are good. If it means preventing STIs that lead to PID, then condoms. OCPs are often cited as reducing PID risk.*

Brutal Honesty Checkpoint:

- **POP:** Know the different compartments and what causes them to descend. Pessaries are good non-surgical options. Surgery aims to restore anatomy.
- **UI:** Stress vs. Urge is the key distinction. PFMT for SUI, bladder training & antimuscarinics for UUI are first-line.
- **Vulvovaginal:** Itch in older woman could be lichen sclerosus (biopsy!), risk of SCC. Pruritus is the commonest symptom of vulval cancer.
- **Contraception:** LARCs are most effective. Know absolute CIs for COCPs (VTE, migraine with aura, smoker >35). Progestogen-only methods for when estrogen is out. Understand basic mechanisms.

Gynecology: Lumps, Bumps, Bleeding, and Bad Bugs

IX. Fibroids (Uterine Leiomyomas) (Dr. Amal)*(Q143, 216, 223, 261, 303, 441, 590, 599, 614, 628, 636, 651)*

Benign smooth muscle tumors of the uterus. Extremely common. Estrogen-sensitive.

- **Types (Location Matters!):**
  - **Submucosal:** Protrude into uterine cavity. Most likely to cause **heavy menstrual bleeding (HMB/menorrhagia)** and subfertility/miscarriage.
  - **Intramural:** Within the uterine wall. Can cause HMB if large, pressure symptoms.
  - **Subserosal:** Protrude from uterine surface. Often asymptomatic unless large (pressure symptoms) or pedunculated (risk of torsion).
  - **Cervical Fibroids:** Less common. *(Q441E Fibroids are uncommon in the cervix - TRUE, compared to uterine body.)*
- **Clinical Features:**
  - Often **asymptomatic.**
  - **Heavy Menstrual Bleeding (HMB):** Most common symptom.
  - **Pressure Symptoms:** Pelvic pain/heaviness, urinary frequency/retention, constipation (if large).
  - **Dysmenorrhea.**
  - **Subfertility / Recurrent Miscarriage** (especially submucosal).
  - **Acute Pain:** Torsion of pedunculated fibroid, or red degeneration (hemorrhagic infarction, common in pregnancy).
  - *(Q143 True regarding uterine leiomyomas: May cause hydronephrosis (C) if very large and compressing ureters. Not common until after menopause (A) is FALSE, common in repro years, often regress after menopause. More common in multiparous (B) - nulliparity is a risk factor. GnRH analogue effects are long-lasting (D) is FALSE, fibroids regrow after stopping. Always require surgery (E) is FALSE, many are managed expectantly or medically.)*
  - *(Q216 Lady with secondary dysmenorrhea, HMB, deep dyspareunia. Logic first diagnosis: Adenomyosis (E) or Endometriosis (B) fit this pain profile well. Fibroids (D) can cause HMB & dysmenorrhea. Ectopic (A) and Endometrial Ca (C) are less likely for this chronic picture of dysmenorrhea and deep dyspareunia, though HMB can be in Ca.)*
  - *(Q599 Fibroids can result in all except: Menorrhagia in all cases (A) is FALSE. Many are asymptomatic. Polycythemia (B) - rarely, some fibroids can produce erythropoietin. Constipation (C), acute urinary retention (D), infertility (E) are all possible.)*
  - *(Q614 In regards to uterine fibroids: May protrude through the cervix (D) - a prolapsing submucosal fibroid. Affect 5% (A) is an underestimate. Always symptomatic (B) false. True capsule (C) false, they have a pseudocapsule. Sarcomatous change (E) is rare, <1%.)*
  - *(Q628 Regarding uterine fibroids, all true except: 75% of women with myomas are infertile (C) is FALSE. While they can cause subfertility, it's not such a high percentage.)*
- **Diagnosis:**
  - Pelvic exam: Enlarged, irregular uterus.
  - **Ultrasound (Transvaginal/Transabdominal):** Primary imaging modality.
  - Hysteroscopy (for submucosal), Sonohysterography, MRI (for complex cases/mapping).
- **Management (Tailored to Symptoms, Size, Location, Age, Fertility Desires):**
  - **Expectant:** If asymptomatic or mildly symptomatic.
  - **Medical:**
    - **Tranexamic Acid, Mefenamic Acid (NSAIDs):** For HMB.
    - **Hormonal (OCPs, LNG-IUS, Progestins):** Control HMB, may not shrink fibroids significantly.
    - **GnRH Agonists (e.g., Leuprolide):** Induce medical menopause, shrink fibroids significantly. Used pre-operatively or for temporary relief. Side effects: menopausal symptoms, bone loss (limit use to 3-6 months

without add-back therapy). *(Q303 Treatment of fibroid with GnRH analogue: This shrinkage is achieved in 6 weeks of treatment (B) is plausible (max shrinkage often by 3 months). Amenorrhea and hypoestrogenic side effects UNLIKELY (C) is FALSE, they are very likely. Osteoporosis if >6mo (D) true. Makes dissection easier (E) true. Decreases size by 10% max (A) is an underestimate, often 30-50%.)*

- **Selective Progesterone Receptor Modulators (SPRMs - e.g., Ulipristal Acetate):** Can shrink fibroids and control bleeding. Concerns about endometrial changes with long-term use.

- **Surgical:**
  - **Myomectomy:** Surgical removal of fibroids, preserves uterus. For women desiring fertility or uterine preservation. Can be hysteroscopic (submucosal), laparoscopic, or open.
  - **Hysterectomy:** Definitive treatment. For women with severe symptoms who have completed childbearing.
  - **Uterine Artery Embolization (UAE):** Interventional radiology procedure. Blocks blood supply to fibroids → shrinkage. For women who wish to avoid surgery and preserve uterus, but impact on future fertility is debated.
  - **MRI-guided Focused Ultrasound Surgery (MRgFUS):** Non-invasive thermal ablation.

X. Endometrial Cancer (Dr. Kamil)*(Q43, 110, 236, 282, 289, 297, 328, 373, 374, 375, 481, 483, 528, 534, 566, 649, 762, 763, 764)*

Most common gynecological malignancy in developed countries. Primarily a disease of postmenopausal women.

- **Types:**
  - **Type I (Endometrioid Adenocarcinoma, ~80%):** Estrogen-dependent. Arises from endometrial hyperplasia. Generally better prognosis.
  - **Type II (Serous, Clear Cell, Carcinosarcoma, ~20%):** Estrogen-independent. Arise in atrophic endometrium. More aggressive, poorer prognosis.
- **Risk Factors (Think Unopposed Estrogen for Type I):**
  - **Unopposed Estrogen Therapy (ERT without progestin in women with a uterus).** *(Q43B is true).*
  - **Obesity:** Peripheral conversion of androgens to estrone in adipose tissue. (MAJOR risk factor - *Q282 Highest incidence will be in Obesity (A)*).
  - **Nulliparity.** *(Q110 Not a risk factor: Women on combined HRT (E). Combined HRT (estrogen + progestin) is protective against endometrial Ca if progestin is adequate. Nulliparity (A), late menopause (B), PCOS (C), Diabetes (D) are all risk factors.)*
  - **Early Menarche / Late Menopause** (longer lifetime estrogen exposure).
  - **PCOS** (chronic anovulation → unopposed estrogen).
  - **Tamoxifen Therapy** (estrogenic effect on endometrium).
  - **Diabetes Mellitus, Hypertension.**
  - **Lynch Syndrome (HNPCC).**
  - Family history of endometrial/ovarian/breast/colon cancer.
  - *(Q373 Risk factors, most common one is Obesity (A).)*
  - *(Q649 Risk factors except: Late menarche and early menopause (B). These are PROTECTIVE. Early menarche/late menopause are risk factors.)*
- **Clinical Presentation:**
  - **Postmenopausal Bleeding (PMB):** HALLMARK. Any PMB needs investigation.
  - Abnormal uterine bleeding in perimenopausal women (intermenstrual, heavy, prolonged).
  - Pyometra, hematometra (if cervical stenosis).
  - Late symptoms: Pelvic pain, pressure.
  - *(Q43A Often presents with PMB, true. Q43D Commonly develops from leiomyomas is FALSE. Q43E PCOS is risk factor, true.)*
- **Diagnosis:**

- **Transvaginal Ultrasound (TVUS):** Measure endometrial thickness (ET).
  - In PMB: ET ≤4mm has high negative predictive value for cancer. If >4mm (or persistent bleeding), needs endometrial sampling.
  - *(Q328 55yo obese, PMB, ET 10mm. Most important DDx is Endometrial carcinoma (E).)*
- **Endometrial Biopsy (Pipelle):** Outpatient procedure. High accuracy.
- **Hysteroscopy with D&C:** If biopsy inconclusive or focal lesion seen on US.
- *(Q43C Screening with CA125 and TV scanning - CA125 is NOT a screening tool for endometrial Ca; TVS for ET is used in symptomatic PMB, not general screening.)*
- *(Q763 Least important investigation in workup: Computed tomography scanning (E). Hysteroscopy (A), endometrial biopsy (B), MRI (C - for staging/myometrial invasion), TVUS (D) are more primary. CT is more for metastatic workup if high risk disease.)*

- **Staging (FIGO Surgical Staging):** Based on hysterectomy, bilateral salpingo-oophorectomy (BSO), lymph node assessment, peritoneal washings.
  - **Stage I:** Confined to uterine corpus. (IA: <50% myometrial invasion; IB: ≥50% myometrial invasion). *(Q376 Tumor size 48mm, invading 7/12mm (>50%) myometrium, glandular cervical involvement. This is Stage IB (FIGO 2009/2023 depending on cervical stromal vs glandular). Given options, IB1/IB2 used to be for cervical cancer. For endometrial, it would be IB due to >50% myometrial invasion. If cervical glandular involvement is considered II, then it's II. The options provided (IA1, IA2, IB1, IB2, II) are more typical of cervical cancer staging. Assuming a typo and it should be endometrial staging options, IB is likely with >50% myo invasion. If Stage II is meant due to cervical involvement, that's another option. Without clear endometrial staging options, Q376 Stage IB is a plausible interpretation focusing on myometrial invasion if cervical involvement isn't stromal.)*
- **Management:**
  - **Surgery:** Total hysterectomy + BSO is cornerstone. Lymphadenectomy (pelvic +/- para-aortic) for staging/treatment depending on risk factors (grade, depth of invasion, histological type). *(Q289 Formal surgical staging should be performed for all except: Presence of an adnexal mass (A). This is an indication for full staging. High grade (C), deep invasion (D), possible cervical extension (E) also warrant full staging. Serous/clear cell histology (B) always gets full staging. So this question is tricky as all listed usually warrant full staging. Perhaps "adnexal mass" if it's clearly benign and separate, but this is unlikely to be the intent.)*
  - **Adjuvant Therapy (based on stage and risk factors):**
    - **Radiotherapy:** Vaginal brachytherapy (to vault) or external beam pelvic radiotherapy (EBRT).
    - **Chemotherapy:** For advanced or high-risk disease (e.g., serous/clear cell types, Stage III/IV).
    - **Hormonal Therapy (Progestins):** For low-grade, ER/PR positive, recurrent/metastatic disease, or fertility-sparing in select young patients with hyperplasia/Grade 1 cancer.
  - *(Q375 Surgical staging recommended for all except: Patients with grade 2 tumors >2cm in diameter (B). Tumor size itself in grade 2 is not an absolute contraindication to omitting full lymphadenectomy IF other features are very low risk (e.g. <50% myometrial invasion, no LVSI). Grade 3 (A), clear cell/serous (C), ovarian mets (D), >50% myo invasion (E) all typically require full staging. B is the least absolute indication among these for full staging with lymphadenectomy if all other factors are favorable.)*
  - *(Q481 Most important prognostic factor: Stage of the disease (B).) (Q762 Most important prognostic factor is Stage of the disease (B).)*
  - *(Q483 Well-diff endometroid tumor, limited to endometrium & endocervical glandular region. Next step: Vault radiotherapy followed by vault smear (A) or observation if very low risk and glandular involvement is minimal and not stromal. Pelvic RT (C) might be overtreatment if truly low risk. Chemo (B) not for low-risk. Combined RT (D) for higher risk. If it's only glandular involvement without stromal, often considered Stage I still, and vault brachytherapy or observation are options.)*

XI. Benign Ovarian Tumors & XII. Ovarian Cancer (Dr. Amal)*(Benign: Q37, 95, 160, 166, 167, 169, 171, 172, 220, 229, 231, 254, 277, 288, 340, 376, 377, 378, 379, 430, 451, 538, 539, 567, 568, 583, 640, 642, 676, 680, 682, 757, 768, 777, 800)*

(Malignant: Q172, 231, 377, 378, 379, 430, 451, 538, 539, 567, 568, 640, 642, 680, 757, 768, 777, 800)

Adnexal masses are common. Differentiating benign from malignant is critical.

- **Benign Ovarian Tumors:**
  - **Functional Cysts (Commonest in Reproductive Age):**
    - **Follicular Cysts:** Failed ovulation, follicle continues to grow. Usually <3cm, resolve spontaneously.
    - **Corpus Luteum Cysts:** Form after ovulation. Can be hemorrhagic, cause pain. Usually resolve. *(Q160 Regarding corpus luteum lesions, wrong: Spontaneously resolved in 7-10 days (E). Usually resolve over 1-3 cycles, not 7-10 days, though some small ones might. Thin-walled, unilocular (A) true. Can cause pain/tenderness (B) true. Associated with amenorrhea (C) if progesterone production maintained (as in early pregnancy or persistent CL). May cause torsion (D) if large, true.)*
  - **Benign Neoplasms:**
    - **Serous Cystadenoma:** Most common benign epithelial tumor.
    - **Mucinous Cystadenoma:** Can be very large. *(Q167 Pseudomyxoma peritonei can be seen with Mucinous tumors (B) of appendix or ovary.)*
    - **Dermoid Cyst (Mature Cystic Teratoma):** Most common germ cell tumor. Contains various tissues (hair, teeth, bone, sebum). Risk of torsion, rupture (chemical peritonitis), rare malignant transformation. *(Q676 Mature cystic teratomas (Dermoids): Are rarely malignant (B) is true (malignant transformation <1-2%). Epithelial tumors (A) false, they are germ cell. Bilateral in 40% (C) is too high, more like 10-15%. Oophorectomy always performed (D) false, cystectomy is common to preserve ovary. Laparotomy not laparoscopy (E) false, laparoscopy is often preferred.)*
    - **Endometrioma ("Chocolate Cyst"):** Endometriosis on the ovary.
    - **Fibroma/Thecoma:** Benign stromal tumors. Fibromas can be associated with Meigs' syndrome (fibroma, ascites, pleural effusion).
  - **Presentation:** Often asymptomatic. Pelvic pain/pressure, dyspareunia, menstrual irregularities. Acute pain if torsion, rupture, hemorrhage.
  - **Management:**
    - **Observation:** For simple cysts <5-7cm in premenopausal women, especially if asymptomatic. Repeat US.
    - **Surgery (Cystectomy or Oophorectomy):** For persistent/large cysts, symptomatic, suspicious features, or complications (torsion). Laparoscopy preferred. *(Q166 25yo, asymptomatic 5cm unilocular ovarian cyst. Best plan: Re-examination in 6 weeks (E) with repeat US is appropriate for an asymptomatic simple cyst. OCPs (A) don't resolve functional cysts. Laparotomy (B) or laparoscopy (C) is overtreatment. CA125 (D) is not routinely needed for simple cysts in premenopausal women.) (Q254 22yo student, dull mid-cycle pain, 5cm bilocular cyst. Next step: Pain killer, re-examination in 6 weeks time (E). This suggests a functional cyst. Tumor markers (D) are not first-line for this presentation.)*
- **Ovarian Cancer ("Silent Killer"):**
  - **Epidemiology:** 5th most common cancer in women. Highest mortality of Gynae cancers (late diagnosis). Peak incidence >60 yrs.
  - **Types (Histological):**
    - **Epithelial (Most Common, ~90%):** Serous (most frequent), mucinous, endometrioid, clear cell, Brenner. *(Q229 Mucinous type resembles endocervical glands (A). Q377 Commonest type of ovarian cancer resembles glandular epithelium of fallopian tube (C) for high-grade serous, or endocervical glands (B) for mucinous. Q538 Epithelial ovarian tumors except: Dysgerminoma (D) is a germ cell tumor.) (Q231 Low risk group criteria for epithelial ovarian cancers involve all except: Ascites (A). Low risk typically means early stage, well-differentiated, intact capsule (B), no surface excrescences (C), negative washings (D), diploid tumor (E). Ascites often indicates more advanced disease.)*
    - **Germ Cell Tumors:** Younger women. Dysgerminoma, teratoma (immature), yolk sac tumor, choriocarcinoma. Often curable even if advanced.

        - **Sex Cord-Stromal Tumors:** Granulosa cell tumor (estrogen-producing), Sertoli-Leydig cell tumor (androgen-producing), fibroma/thecoma. *(Q37 58yo, PMB, estrogen-secreting ovarian tumor. Most likely: Thecoma (E) or Granulosa cell tumor. Dysgerminoma (A) is germ cell. Mucinous/Serous (B,C) are epithelial, usually not hormonally active in this way. Sertoli cell (D) produces androgens.)*
    - **Risk Factors:**
        - Advancing age.
        - Nulliparity / low parity.
        - Early menarche / late menopause.
        - Family history of ovarian/breast cancer (BRCA1/BRCA2 mutations, Lynch syndrome).
        - Endometriosis (for some types like clear cell, endometrioid).
        - *(Q451 Risk factors for ovarian cancer except: Fertility drugs (D). Link is controversial and weak if it exists, mostly related to underlying subfertility. Low parity (A), early menarche/late meno (B), high BMI (C - less strong than others), estrogen replacement (E - unopposed, or for prolonged periods, link is complex) are more established or plausible for some types. D is the best "except".)*
        - *(Q640 Risk factor for ovarian cancer: Nulliparity (B).)*
    - **Protective Factors:** OCP use, multiparity, breastfeeding, tubal ligation, hysterectomy, BSO.
    - **Clinical Presentation:** Often vague, late.
        - Abdominal bloating/distension, pelvic/abdominal pain, early satiety/difficulty eating, urinary urgency/frequency. ("BEAT" symptoms: Bloating, Eating difficulty, Abdominal/pelvic pain, Toilet changes). *(Q172 Uncommon presentation of ovarian cancer: Chest pain (B). Abdo pain (A), weight loss (C), abdo distension (D), SOB (E - from ascites/pleural effusion) are more typical.)*
        - *(Q379 Not a component of Ovarian Cancer Symptom Index: Elevation of tumor markers (E). The index focuses on symptoms like pain (A), urinary issues (B), increased abdo size (C), bloating/eating difficulty (D). Tumor markers are investigations.)*
        - *(Q430 Ovarian Cancer Symptom Index parameters except: Abnormal vaginal bleeding (B). This is more typical of endometrial/cervical cancer. Pelvic/abdo pain (A), urinary frequency/urgency (C), bloating (D), increased abdo contour (E) are classic ovarian Ca symptoms.)*
    - **Diagnosis & Workup:**
        - Pelvic exam (adnexal mass, ascites).
        - **TVUS:** Assess mass characteristics (size, solid/cystic, septations, papillary projections, vascularity - Doppler).
        - **Tumor Markers:**
            - **CA-125:** Elevated in ~80% epithelial ovarian Ca (especially serous). Not specific (also elevated in endometriosis, fibroids, PID, liver disease, pregnancy, menstruation - *Q168 CA125 elevated, not associated with: Anovulation (B). Cirrhosis (A), Menses (C), Ectopic (D), Lung cancer (E - some non-gynae cancers) can elevate it. Anovulation itself doesn't directly raise CA125 unless associated with something like endometriosis.)* Not for general screening. Useful for monitoring response to treatment and detecting recurrence. *(Q444 Tumor marker useful for screening epithelial cancer: CA125 (B). "Screening" is used loosely here; it's not for general population screening but for risk assessment in high-risk women or evaluating a mass.)*
            - Other markers for specific types: AFP, hCG (germ cell), Inhibin (granulosa cell), HE4.
            - *(Q378 Useful tumor markers except: Alpha fetoprotein (AFP) (D) and Cancer antigen 15.3 (CA 15.3) (E). AFP is for germ cell tumors (yolk sac). CA15.3 is more for breast cancer. CA125 (A), HE4 (C), CA19.9 (B - can be up in mucinous) are used for epithelial ovarian Ca.)*
        - CT/MRI/PET for staging.
    - **Management:**
        - **Surgery (Staging Laparotomy):** Hysterectomy, BSO, omentectomy, lymph node sampling/dissection, peritoneal washings, biopsies of suspicious areas. Cytoreduction (debulking) for advanced disease. *(Q169 55yo, radical*

surgery for ovarian tumor, psammoma bodies. Most likely diagnosis: Serous tumors (D) are classically associated with psammoma bodies.)

- **Chemotherapy:** Platinum-based (carboplatin) + Taxane (paclitaxel) is standard for most epithelial ovarian Ca.
- *(Q539 Bilateral ovarian conservation is correct surgical step in management of: Early non-endometrioid adenocarcinoma in young newly married lady (E) - if fertility sparing is desired for very early stage, low grade specific histologies. For A, B, C, D, oophorectomy (often bilateral) is usually part of standard surgical treatment if ovarian involvement or high risk of mets/synchronous primaries.)*
- *(Q800 Staging essentials for ovarian cancer except: The condition of both kidneys (A). While renal function is important for chemo, direct assessment of kidneys isn't a primary staging component like malignant ascites (B), omental mets (C), inguinal node mets (D - though pelvic/para-aortic are more typical for ovarian spread), or sub-diaphragmatic smears (E).)*

XIII. Lower Genital Infections (Dr. Amal)*(Q4, 24, 113, 164, 184, 213, 232, 256, 273, 338, 397, 418, 470, 532, 589, 608, 633, 657, 715, 786, 796)*

Commonly BV, Candidiasis, Trichomoniasis. Discharge, itch, odor.

- **Bacterial Vaginosis (BV):**
  - **Cause:** Polymicrobial overgrowth of anaerobes (e.g., Gardnerella vaginalis, Mycoplasma hominis), decrease in lactobacilli. Not an "infection" in classic sense, but dysbiosis.
  - **Symptoms:** Thin, greyish-white, homogenous discharge; **fishy odor** (especially after intercourse/menses). Often asymptomatic. Minimal/no inflammation.
  - **Diagnosis (Amsel Criteria - ≥3 of 4):** 1. Characteristic discharge, 2. Vaginal pH >4.5, 3. Positive Whiff test (amine odor with KOH), 4. Clue cells on microscopy. *(Q4D correct. Q273 40yo, frothy discharge, clue cells. Cells represent: Epithelial squamous cell covered with bacteria (B).)*
  - **Treatment:** Metronidazole (oral/vaginal) or Clindamycin (oral/vaginal). *(Q715 Treatment of choice for BV is Metronidazole (C))*.
- **Vulvovaginal Candidiasis (VVC, "Yeast Infection"):**
  - **Cause:** Overgrowth of Candida species (C. albicans most common - *Q397 Most common Candida species is C. albicans (A)*).
  - **Risk Factors:** Diabetes, antibiotic use, pregnancy, OCPs, immunosuppression, tight/occlusive clothing. *(Q164 Regarding vulvovaginal candidiasis, incorrect: IUCD is a risk factor (B) - not a strong/direct risk factor like the others. Most common cause of discharge in pregnant women (A) - it's common, but BV might be more prevalent overall. 80% caused by C. albicans (C) - true. Intense pruritus (D) - true. Vulvar burning/dyspareunia (E) - true.) (Q213 40yo, vaginal Candida, least known risk factor: Intrauterine contraceptive device (D). DM (A), Broad spectrum Abx (B), Corticosteroids (C), OCPs (E) are all well-known.)*
  - **Symptoms:** Thick, white, "cottage cheese" discharge; intense vulvovaginal pruritus, burning, soreness, dyspareunia.
  - **Diagnosis:** Vaginal pH usually normal (≤4.5). Microscopy (KOH prep): pseudohyphae, spores. Culture if recurrent/atypical.
  - **Treatment:** Topical azoles (clotrimazole, miconazole) or oral fluconazole.
- **Trichomoniasis:**
  - **Cause:** Trichomonas vaginalis (flagellated protozoan). STI.
  - **Symptoms:** Profuse, yellow-green, frothy, malodorous discharge; vulvovaginal irritation, pruritus, dysuria, dyspareunia. "Strawberry cervix" (colpitis macularis) in ~10%. Can be asymptomatic (especially in men).
  - **Diagnosis:** Vaginal pH >4.5. Microscopy (wet mount): motile trichomonads. NAATs highly sensitive.
  - **Treatment:** Metronidazole or Tinidazole (oral). **TREAT PARTNERS.** *(Q113B "No vulvovaginal irritation" is incorrect for Trichomonas). (Q338 25yo, profuse frothy discharge, strawberry cervix. Treatment: Metronidazole (A).)*
- **Other:**
  - **Atrophic Vaginitis:** (See Vulva/Vagina section).

- **Foreign Body:** Especially in children with foul discharge. *(Q24 5yo girl, foul vaginal discharge. Most likely: Foreign body (A).)*
- **Chlamydia/Gonorrhea:** Can cause cervicitis with discharge, but often PID is the bigger concern. *(Q232 26yo, florid vaginitis, profuse yellow, frothy, offensive discharge. Next step: Perform (KOH) smear and wet smear (D). This describes Trich or severe BV. Culture (B,C) takes time. Imidazole (A) is for yeast. Pap (E) is for cervical screening.)*

XIV. Pelvic Inflammatory Disease (PID) (Dr. Oqba)*(Q33, 68, 82, 96, 119, 162, 279, 387, 702)*

Ascending infection of upper female genital tract (endometrium, fallopian tubes, ovaries, pelvic peritoneum).

- **Cause:** Most commonly STIs (Neisseria gonorrhoeae, Chlamydia trachomatis - *Q162 Chlamydia trachomatis (E) is a major cause*). Can also be polymicrobial (anaerobes, Gardnerella, Mycoplasma).
- **Risk Factors:** Young age, multiple sexual partners, new partner, history of STIs/PID, IUD insertion (small transient risk), douching, instrumentation.
- **Clinical Features (Can be subtle to severe):**
  - Lower abdominal/pelvic pain (bilateral).
  - Abnormal vaginal discharge (often mucopurulent).
  - Intermenstrual or postcoital bleeding.
  - Dyspareunia.
  - Fever, chills (less common in mild PID).
  - **On Exam:** Cervical motion tenderness ("chandelier sign"), adnexal tenderness, uterine tenderness.
  - *(Q33 Which is NOT criteria for PID: ESR 10 mm/hr (C). While ESR can be elevated, a low ESR doesn't rule it out and specific cutoffs are not universal diagnostic criteria like temp >38 (A), WBC >15000 (B - though often >10,000 is used), cervical motion tenderness (D), bilateral lower abdo pain (E). The CDC minimum criteria are lower abdominal/pelvic pain PLUS one of: cervical motion tenderness, uterine tenderness, or adnexal tenderness.)*
  - *(Q82 Regarding PID, true: Bimanual examination is the most important examination (D) for eliciting tenderness which is key to diagnosis. Vaginal discharge common (A) but not specific. Perihepatic adhesions (Fitz-Hugh-Curtis) (B) indicate disseminated infection, not just active local PID. Cultures necessary (C) - often clinical diagnosis, cultures help guide specific Rx but not always essential to start Rx. Does not occur in pregnancy (E) - rare but possible, especially early.)*
- **Diagnosis:** Primarily clinical. High index of suspicion.
  - Minimum criteria (CDC): Pelvic/lower abdominal pain + cervical motion tenderness OR uterine tenderness OR adnexal tenderness.
  - Additional criteria: Fever, abnormal discharge, positive GC/Chlamydia, elevated ESR/CRP, WBCs on microscopy.
  - Laparoscopy: Gold standard (visualize inflamed tubes, pus), but invasive, not routine.
- **Differential Diagnosis:** Ectopic pregnancy *(Q68 PID differentials except Hepatitis (D). Ectopic (A), Ovarian cyst torsion (B), Endometriosis (C), Appendicitis (E) are all common DDx for pelvic pain)*, appendicitis, ovarian cyst accident, UTI, endometriosis, IBS.
- **Complications (Serious!):**
  - **Tubo-ovarian Abscess (TOA).**
  - **Chronic Pelvic Pain.**
  - **Infertility** (tubal factor).
  - **Ectopic Pregnancy** (increased risk due to tubal damage).
  - **Fitz-Hugh-Curtis Syndrome:** Perihepatitis (RUQ pain, adhesions between liver capsule and peritoneum).
- **Management:**
  - **Broad-spectrum antibiotics** to cover GC, Chlamydia, and anaerobes.
    - **Outpatient regimens (mild-moderate PID):** e.g., Ceftriaxone IM + Doxycycline PO +/- Metronidazole PO.

        - **Inpatient regimens (severe PID, TOA, pregnant, failed outpatient):** e.g., Cefoxitin/Cefotetan IV + Doxycycline IV/PO; or Clindamycin IV + Gentamicin IV.
    - Treat sexual partners.
    - Remove IUD if present (controversial, but often done if severe).
    - Drainage of TOA if large or not responding to antibiotics.
    - *(Q119 Not used in management of acute PID: Dilatation and curettage (D&C) (E). This is for uterine contents, not for treating upper tract infection. Oral doxycycline (A), Removal of IUD (B - if present and severe), Clindamycin (C), Laparoscopy (D - for diagnosis/TOA drainage) are all relevant.)*

Brutal Honesty Checkpoint:

- **Fibroids:** Location dictates symptoms (submucosal = bleeding!). Asymptomatic = observe. Medical Rx for symptoms, GnRH for shrinkage (temporary). Myomectomy for fertility, hysterectomy for definitive.
- **Endometrial Ca:** PMB = Cancer until proven otherwise in postmenopausal woman. ET >4mm needs biopsy. Unopposed estrogen is the enemy for Type I. Surgery is mainstay.
- **Ovarian Masses:** Is it functional, benign neoplastic, or malignant? US features, age, CA-125 (in postmeno) guide. "BEAT" symptoms for ovarian cancer. Surgery often needed for diagnosis/treatment of suspicious masses.
- **Lower Genital Infections:** BV (fishy, clue cells, pH >4.5, Metronidazole). Candida (itch, cottage cheese, normal pH, Azoles). Trich (frothy, strawberry cervix, pH >4.5, Metronidazole, treat partner).
- **PID:** STI bugs ascending. Pelvic pain + tenderness (CMT!). Treat empirically & broadly. Sequelae (infertility, ectopic, chronic pain) are devastating.

Gynecology: Life's Transitions, Bleeding Puzzles, and Cervical Concerns

This section covers the beginning and end of reproductive life (puberty, menopause), the common problem of abnormal bleeding, the preventable threat of cervical cancer, and the unfortunate reality of early pregnancy loss.

XV. Menopause (Dr. Nadia)*(Q26, 99, 116, 140, 144, 165, 226, 312, 332, 459, 495, 496, 497, 564, 596, 631, 632)*

Permanent cessation of menstruation due to loss of ovarian follicular activity.

- **Definitions:**
    - **Menopause:** Diagnosed retrospectively after 12 consecutive months of amenorrhea for which no other obvious pathological or physiological cause is apparent. *(Q144D, Q332A correctly define this.)*
    - **Average Age:** ~51 years in Western countries. *(Q116B "Average age is 55" is INCORRECT; it's around 50-52.)*
    - **Perimenopause (Climacteric):** Transition period before menopause (and first year after). Characterized by hormonal fluctuations, irregular cycles, and onset of menopausal symptoms.
    - **Premature Ovarian Insufficiency (POI):** Menopause before age 40.
    - **Predominant Estrogen Postmenopause:** Estrone (A), derived from peripheral conversion of adrenal androstenedione in adipose tissue. Estradiol levels plummet. *(Q497A, Q632A are correct.)*
- **Physiology:** Ovaries run out of follicles → ↓estrogen & inhibin → ↑FSH & LH (loss of negative feedback). FSH elevation is a key biochemical marker.
- **Symptoms:**
    - **Vasomotor Symptoms (VMS):** Hot flushes, night sweats. HALLMARK. *(Q26A Most distressing symptom is Hot flushes. Q116D Hot flashes is an early symptom, true.)*
    - **Genitourinary Syndrome of Menopause (GSM) / Vulvovaginal Atrophy:** Vaginal dryness, itching, dyspareunia, urinary urgency/frequency, recurrent UTIs.
    - **Mood/Cognitive:** Irritability, anxiety, depression, sleep disturbance (often linked to VMS), memory/concentration issues.
    - **Musculoskeletal:** Joint pain (arthralgia), osteoporosis risk increases. *(Q116C Increased risk of osteoporosis, true.)*
    - **Other:** Skin changes, hair thinning, changes in libido.

- **Long-Term Consequences of Estrogen Deficiency:**
  - **Osteoporosis:** Increased bone resorption → decreased bone mineral density → fracture risk.
  - **Cardiovascular Disease (CVD):** Risk increases after menopause (loss of estrogen's protective effects on lipid profile and vasculature).
  - **Cognitive Decline/Dementia:** Association less clear-cut, but estrogen has neuroprotective roles.
- **Management of Menopausal Symptoms:**
  - **Lifestyle:** Diet, exercise, smoking cessation, stress management, layered clothing, trigger avoidance for VMS.
  - **Hormone Replacement Therapy (HRT) / Menopausal Hormone Therapy (MHT):**
    - **Indications:**
      1. **Moderate-severe VMS:** Most common indication. *(Q99 Indications for HRT except: Perimenopausal cycle irregularities (E). While HRT can regulate cycles, it's not a primary indication for HRT if VMS aren't the main issue; OCPs or progestins might be used for cycle control in perimenopause. Hot flushes (A), vaginal dryness (B), night sweats (C), bone/joint pains (D - though less strong indication than VMS/osteoporosis prevention) are more typical reasons.) (Q596 Main current indication to give HRT: Symptomatic relief of hot flushes (C).)*
      2. **Prevention/Treatment of Osteoporosis:** Especially if <60 yrs or within 10 yrs of menopause and at risk.
      3. **Premature Ovarian Insufficiency (POI):** HRT usually recommended until average age of natural menopause.
      4. **Genitourinary Syndrome of Menopause (GSM):** Low-dose topical/vaginal estrogen is highly effective and has minimal systemic absorption/risks. *(Q140 Management options for postmenopausal symptoms: Estrogen patches with Mirena IUD (D) - Mirena provides progestin for endometrial protection if uterus is present, making this a valid combined HRT approach. Combined continuous (A), topical estrogen for dryness (B), estrogen tablets (C - needs progestin if uterus intact), combined sequential (E) are all forms of HRT.)*
    - **Regimens:**
      - **Estrogen-Only Therapy (ET):** ONLY for women WITHOUT a uterus (post-hysterectomy). Unopposed estrogen increases endometrial cancer risk.
      - **Combined Estrogen-Progestogen Therapy (EPT):** For women WITH a uterus. Progestogen protects endometrium.
        - **Sequential EPT:** Estrogen daily, progestogen for 10-14 days/month → withdrawal bleed. For perimenopausal or early postmenopausal women.
        - **Continuous Combined EPT:** Estrogen + progestogen daily → no bleed (amenorrhea after initial spotting). For postmenopausal women (>1 yr since LMP).
          - **Risks (from WHI study, primarily in older women starting HRT later):** VTE, stroke, breast cancer (with EPT >5 yrs), gallbladder disease.
          - **Benefits (especially if started early around menopause):** VMS relief, osteoporosis prevention, improved GSM, potential mood/sleep benefits. CVD risk is complex: may be neutral or beneficial if started early, potentially harmful if started late in older women with pre-existing CVD.
          - **Contraindications:** Current/past breast cancer (or estrogen-dependent cancer), undiagnosed vaginal bleeding, current/past VTE, active liver disease, known CAD/stroke, porphyria. *(Q564 Absolute CIs for HRT except: Well differentiated endometroid uterine carcinoma (B). This IS an estrogen-sensitive cancer and a CI. Undiagnosed vaginal bleeding (A), Pregnancy (C), Coronary artery disease (D), Venous thrombosis (E) are all CIs.)*
      - **Non-Hormonal Options for VMS:** SSRIs/SNRIs (e.g., venlafaxine, paroxetine), gabapentin, clonidine.
- **Other Considerations:**
  - Smoking induces earlier menopause. *(Q116A True)*.
  - Delayed menopause is a risk factor for endometrial cancer (longer estrogen exposure). *(Q116E True)*.

XVI. Puberty (Dr. Nadia)*(Q5, 206, 274, 400, 413, 418, 445, 537, 606, 678)*

Transition from childhood to reproductive maturity. Driven by HPG axis activation.

- **Normal Sequence & Timing (Tanner Staging):**
  - **Girls (Onset ~8-13 yrs):**
    1. **Thelarche (Breast Budding):** First sign. *(Q606A)*
    2. Pubarche (Pubic Hair).
    3. Peak Height Velocity (PHV).
    4. **Menarche (First Menses):** ~2-2.5 years after thelarche, typically Tanner stage 3-4 breast development. *(Q400 Median age of menarche: 13-16 years (B) is a reasonable range, though 12-13 is often cited as average in Western countries. Q678 Regarding puberty, one is true: Pubertal changes are completed faster in girls than in boys (B).)*
  - **Boys (Onset ~9-14 yrs):**
    1. **Testicular Enlargement (≥4mL or ≥2.5cm):** First sign.
    2. Pubarche.
    3. Penile Growth, Voice Deepening.
    4. Peak Height Velocity (later than girls).
- **Precocious Puberty:** Onset of secondary sexual characteristics before age 8 (girls) or 9 (boys).
  - **Central (GnRH-dependent):** Premature activation of HPG axis. Idiopathic (more common in girls), CNS tumors/lesions.
  - **Peripheral (GnRH-independent):** Excess sex steroids from gonads/adrenals or exogenous sources. CAH, gonadal/adrenal tumors, McCune-Albright syndrome. *(Q678E GnRH analogues are mainstay for Central precocious puberty, not peripheral.)*
- **Delayed Puberty:** No signs by age 13 (girls) or 14 (boys).
  - **Causes:** Constitutional delay (most common, "late bloomers"), hypogonadotropic hypogonadism (Kallmann, chronic illness, malnutrition), hypergonadotropic hypogonadism (Turner, Klinefelter, gonadal damage).
- **Physiological Leukorrhea:** *(Q418 12yo girl, slight odorless clear vaginal discharge, normal pubertal development, not sexually active. Most likely: Physiologic discharge (C). This is normal, due to estrogen effect on vaginal mucosa.)*
- **Ambiguous Genitalia:** *(Q5 Conditions causing ambiguous external genitalia in neonate: Adrenogenital syndrome (D) (i.e., CAH) is a classic cause of virilization in 46,XX females. Turner's (A) is 45,XO, usually normal female external genitalia but gonadal dysgenesis. Testicular feminization (AIS) (B) is 46,XY with female external genitalia. Cryptorchidism (C) is undescended testes, not ambiguous genitalia itself. Klinefelter's (E) is 47,XXY, male phenotype, often diagnosed later.) (Q622 Associated with ambiguous genitalia: Adrenogenital syndrome (D).)*

XVII. Dysfunctional Uterine Bleeding (DUB) / Abnormal Uterine Bleeding (AUB) (Dr. Asmaa)*(Q51, 109, 118, 153, 173, 258, 315, 381, 395, 405, 408, 479, 550, 553, 714, 740, 793)*

AUB is bleeding outside normal parameters (frequency, duration, volume, regularity). DUB is AUB with no identifiable structural/organic pathology (often anovulatory). PALM-COEIN classification for AUB.

- **PALM (Structural Causes):**
  - **P**olyp (endometrial/cervical).
  - **A**denomyosis.
  - **L**eiomyoma (fibroid - especially submucosal).
  - **M**alignancy & Hyperplasia (endometrial, cervical).
- **COEIN (Non-structural Causes):**
  - **C**oagulopathy (e.g., von Willebrand's).
  - **O**vulatory Dysfunction (anovulation/oligo-ovulation → unopposed estrogen → irregular shedding. Common at extremes of reproductive age: adolescents due to immature HPO axis, perimenopause due to declining ovarian

function). *(Q109 40yo lady, abnormal uterine bleeding. Incorrect: If due to ovulatory dysfunction, she will have combination of irregularity AND same bleeding volume (C). Ovulatory dysfunction usually causes variable volume, often heavy if prolonged unopposed estrogen then breakthrough bleed.) (Q145 Most common cause for menstrual irregularities in adolescent girls: Anovulatory cycles (C) due to immature HPO axis.)*

  - **E**ndometrial (local endometrial factors, e.g., infection, inflammation).
  - **I**atrogenic (e.g., medications - anticoagulants, hormonal contraception breakthrough bleeding).
  - **N**ot Yet Classified.

- **Specific Bleeding Patterns:**
  - **Menorrhagia (Heavy Menstrual Bleeding - HMB):** Excessive cyclical bleeding (>80mL or impacting QoL). *(Q153 Not a cause of menorrhagia: Hyperprolactinemia (D). This usually causes oligo/amenorrhea or anovulatory DUB, not typically cyclical heavy bleeding. Fibroids (A), PID (B - can cause intermenstrual or HMB), Warfarin (C), vWD (E) can all cause HMB.) (Q479 All cause regular heavy periods except: Cervical cancer (B). This usually causes intermenstrual or postcoital bleeding, not typically regular heavy menses. Endometrial polyp (A), Hypothyroidism (C - can cause HMB), Fibroids (D), Adenomyosis (E) can cause HMB.) (Q118 Not used in treatment of menorrhagia: Progestogen only pills (C). While some POPs can reduce bleeding or cause amenorrhea, they are not primary licensed treatments for HMB like COCPs (A), mefenamic acid (B), tranexamic acid (D), or endometrial ablation (E). LNG-IUS is very effective but that's different from POPs.)*
  - **Metrorrhagia:** Irregular bleeding between periods.
  - **Menometrorrhagia:** Heavy AND irregular bleeding. *(Q395 "Heavy uterine bleeding...at irregular intervals". My name is: Menometrorrhagia (C) or Metromenorrhagia (D) - these are synonymous.)*
  - **Oligomenorrhea:** Cycles >35 days.
  - **Polymenorrhea:** Cycles <21 days.
  - **Hypomenorrhea:** Scanty bleeding. *(Q51 Asherman syndrome typically presents with: Hypomenorrhea (A) or amenorrhea due to intrauterine adhesions.)*
- **Diagnosis & Management:**
  - History (bleeding pattern, associated symptoms, medical/gynae history).
  - Pelvic exam.
  - Pregnancy test!
  - Labs: FBC, coagulation screen, TSH, prolactin, androgen profile (if PCOS suspected).
  - TVUS (assess endometrium, ovaries, uterus). Saline infusion sonohysterography.
  - Endometrial biopsy (especially if >45 yrs, or younger with risk factors for endometrial Ca, or persistent AUB). Hysteroscopy.
  - Management depends on cause, severity, age, fertility desires. Can range from reassurance, medical (NSAIDs, tranexamic acid, hormonal Rx), to surgical (D&C, endometrial ablation, myomectomy, hysterectomy).
  - *(Q408 Regarding DUB, all correct except: May be caused by adenomyosis (A). DUB is bleeding without organic pathology. Adenomyosis IS an organic pathology. So A is the "except" in the strict definition of DUB. Is common at perimenopause (B) - true (anovulation). Can be endometrial dysfunction (C) - true (part of COEI*N*). Commonly treated with progestogens (D) - true (for anovulatory). Can be due to bleeding tendency (E) - true (Coagulopathy in COEI*N*).)*

XVIII. Cervical Cancer & Pre-cancer (CIN) (Dr. Kamil)*(Q21, 54, 175, 197, 208, 275, 327, 372, 443, 444, 494, 499, 572, 674, 765)*

Largely preventable through screening (Pap smear/HPV testing) and HPV vaccination.

- **Etiology:** Persistent infection with **High-Risk Human Papillomavirus (HR-HPV)** types (especially HPV 16, 18). *(Q54 Most common STD leading to cervical Ca is HPV (B). Q275 Direct causal relationship between cervical Ca and sexual activity, all correct except: The frequency of sexual activity (C). It's more about number of partners and early age of onset. Young age at first pregnancy (A - often proxy for early coitarche), multiple partners (B), high parity (D - can be a confounder), low socioeconomic status (E - access to screening/healthcare) are all associated.)*

- **Risk Factors:** Early coitarche, multiple sexual partners, high-risk male partners, smoking, immunosuppression, history of STIs, DES exposure (for clear cell adenocarcinoma). *(Q175 Not a risk factor for cervical cancer: Using Mirena coil (D). Mirena may even have some local protective effects. Early coitus (A), multiple partners (B), immunocompromise (C), smoking (E) are risks.)*
- **Cervical Intraepithelial Neoplasia (CIN) / Squamous Intraepithelial Lesion (SIL):** Pre-malignant changes.
  - CIN 1 (LSIL): Mild dysplasia. High regression rate. Often managed expectantly.
  - CIN 2/3 (HSIL): Moderate/severe dysplasia, carcinoma in situ. Higher risk of progression to invasive cancer. Usually requires treatment.
  - *(Q21 33yo with HSIL on Pap. Next step: Colposcopic directed biopsy (B). This is standard. Repeat Pap (A) is inadequate. Conization (C) is for treatment or if colpo unsatisfactory/lesion not fully seen. Radical hysterectomy (D) is for invasive cancer. CT scan (E) not for initial workup of HSIL.)*
  - *(Q443 Definite final diagnosis of CIN III is made by: Histopathology (C) from colposcopic biopsy or LLETZ/cone.)*
- **Screening:**
  - **Pap Smear (Cytology):** Detects abnormal cells.
  - **HPV Testing:** Detects HR-HPV DNA. Can be primary screening or co-testing with Pap.
  - **Colposcopy:** Magnified view of cervix, acetic acid/Lugol's iodine to highlight abnormal areas for biopsy.
  - *(Q197 Concerning carcinoma of the cervix: The histological type adenocarcinoma occurs more frequently in young women (B) is true relative to older women where SCC dominates. Peak incidence ~40yrs (A) is a bit young, often bimodal (30s-40s and older). Negative smear excludes diagnosis (C) FALSE, false negatives occur. Commonly presents with menorrhagia (D) FALSE, usually PCB/IMB. More common in obese (E) FALSE, obesity is risk for endometrial Ca.)*
- **Invasive Cervical Cancer:**
  - **Histology:** Squamous cell carcinoma (SCC, ~70-80%), Adenocarcinoma (~20-25%).
  - **Symptoms:** Often asymptomatic in early stages. Postcoital bleeding (PCB), intermenstrual bleeding, abnormal vaginal discharge, pelvic pain (late), dyspareunia.
  - **Staging (FIGO Clinical Staging):** Based on clinical exam, imaging (MRI, CT, PET) for extent.
  - **Management:**
    - **Early Stage (e.g., IA1, some IA2/IB1):** Cone biopsy, simple hysterectomy, or radical hysterectomy/trachelectomy (fertility-sparing) +/- lymph node dissection.
    - **Locally Advanced (e.g., IB2-IVA):** Chemoradiation (external beam RT + brachytherapy + concurrent cisplatin).
    - **Metastatic (IVB):** Palliative chemotherapy +/- radiotherapy.
    - *(Q372 Management for Stage Ia cervical Ca (assuming Ia1 without LVSI): Total abdominal hysterectomy (B) is an option, or conization if fertility desired and margins clear. Radical hysterectomy (D) is for more advanced stage. Conization + chemo (C) not standard for Ia1. Chemoradiation (E) not for Ia1.)*
    - *(Q499 Prognosis of cervical Ca, least important factor: The age of the patient (E). Stage (A), tumor volume (B), vascular invasion (C), histologic type (D) are all more significant prognostically.)*
    - *(Q674 Facts about cervical cancer except: Positive association of high gravidity and adenocarcinoma (B). High gravidity is more linked to SCC, and association is complex/possibly confounding.)*
    - *(Q766 Radical hysterectomy involves resection of: The uterus, cervix, uterine artery, parametrial and cervical ligaments, and vagina (upper part) (D). Ovaries/tubes (A,C,E) are not always removed unless indicated (salpingo-oophorectomy).)*

XIX. Miscarriages (Spontaneous Abortion) (Dr. Fidaa)*(Q45, 151, 188, 322, 346, 353, 394, 471, 488, 489, 511, 518, 521, 522, 527, 629, 720)*

Pregnancy loss before viability (usually <20-24 weeks or <500g).

- **Incidence:** Common (~15-20% of clinically recognized pregnancies).
- **Causes:**

    - **Chromosomal Abnormalities of Fetus:** Most common cause of early miscarriages (~50-60%). *(Q151 Most likely cause for 10wk loss with no fetal heart: Chromosomal abnormalities of the conceptus (B). Q322 Most probable cause of first trimester spontaneous abortion: Chromosomal abnormalities (A).)*
    - **Maternal Factors:** Uterine anomalies, Asherman's, fibroids, cervical incompetence, infections (TORCH, listeria, BV), endocrine (uncontrolled DM, thyroid disease, luteal phase defect), thrombophilias (e.g., APS), autoimmune (SLE), advanced maternal age, trauma, environmental toxins.
    - *(Q488 Etiology of abortion, all true except: Structural abnormalities of chromosomes are more common than number abnormalities (B) is FALSE. Numerical abnormalities (aneuploidy like trisomies) are far more common than structural (translocations etc.).)*
    - *(Q489 Causes of first trimester miscarriage except: Incompetent cervix (D). This typically causes SECOND trimester loss. Fetal abnormalities (A), Listeria (B), Ovarian surgery (C - if corpus luteum removed early), Autosomal trisomy (E) are all possible causes of first trimester loss.)*
- **Types of Miscarriage (Based on Clinical & US Findings):**
    - **Threatened:** Vaginal bleeding, cervix closed, viable IUP on US.
    - **Inevitable:** Vaginal bleeding, cervix open, +/- products at os.
    - **Incomplete:** Vaginal bleeding, cervix open, some products passed, some remain in utero. *(Q346 Criteria for incomplete miscarriage except: The scan showed empty uterus (D). This would be a complete miscarriage. Bleeding (A), history of passage of tissue (B), proven pregnancy (C), abdominal pain (E) are consistent.)*
    - **Complete:** All products passed, cervix often closed, empty uterus on US.
    - **Missed:** No bleeding (or minimal old spotting), cervix closed, non-viable IUP (no fetal heart, or empty sac).
    - **Septic:** Any of above with signs of uterine infection (fever, tender uterus, purulent discharge). *(Q45 Most likely mechanism of septic abortion: Ascending infection (C). Q518 Septic miscarriage causative organism except: Staphylococcus (B). E.coli (A), Bacteroides (C), Streptococci (D), Clostridium (E) are all classic. Staph can cause toxic shock but less common as direct uterine pathogen in septic abortion.)*
    - **Recurrent Miscarriage (RM):** ≥3 consecutive pregnancy losses <20 weeks. Needs detailed workup. *(Q188 Not a possible cause of recurrent miscarriage: Pituitary microadenoma (A). While prolactinomas can cause infertility/anovulation, they are not a direct cause of recurrent miscarriage once pregnancy established. PCOS (B - hormonal milieu), Progesterone deficiency (C - luteal phase defect), Cervical incompetence (D), APS (E) are all implicated in RM.)*
- **Diagnosis:** History, exam (speculum for cervical os, bimanual), TVUS (key for viability, retained products, location), quantitative β-hCG (serial levels).
- **Management:**
    - **Threatened:** Expectant, pelvic rest. Progesterone sometimes used (evidence varies).
    - **Inevitable/Incomplete/Missed:**
        - **Expectant:** Allow spontaneous passage. Success varies.
        - **Medical:** Misoprostol (PGE1 analogue) vaginally or orally. Induces uterine contractions.
        - **Surgical:** Suction curettage (D&C). Quickest, most predictable.
    - **Septic:** IV broad-spectrum antibiotics, prompt uterine evacuation.
- **Complications:** Hemorrhage, infection, Asherman's (post-D&C), psychological distress. Rh-negative unsensitized women need Anti-D.

Brutal Honesty Checkpoint:

- **Menopause:** VMS are hallmark. HRT is for symptom relief primarily (VMS, GSM) and osteoporosis prevention in right candidates. ET for women with uterus, EPT if uterus intact. Risks/benefits depend on age/timing of initiation.
- **Puberty:** Know the first signs (Thelarche girls, Testicular enlargement boys) and average menarche timing. Differentiate central vs. peripheral precocious puberty.
- **AUB/DUB:** PALM-COEIN helps frame causes. Pregnancy test always. Biopsy if >45 or risk factors. Anovulatory bleeding is common at extremes of reproductive age.

- **Cervical Ca:** HPV is the villain. Screening (Pap/HPV) saves lives. Colposcopy for abnormal screen. Staging is clinical.
- **Miscarriage:** Chromosomal abnormalities are #1 cause early on. Know the types based on cervix and US. Manage based on type, stability, patient preference.

XX. Endometriosis (Dr. Shawqi)*(Q3, 30, 97, 128, 149, 157, 182, 211, 216, 294, 299, 354, 389, 398, 442, 533, 546, 600, 607, 679, 713)*

- **Definition:** Presence of endometrial-like glands and stroma *outside* the uterine cavity.
- **Common Sites:** Ovaries (forming endometriomas/"chocolate cysts"), pelvic peritoneum (uterosacral ligaments, pouch of Douglas, bladder serosa), rectovaginal septum, bowel, and rarely distant sites (lungs, umbilicus). *(Q3E The commonest site is the pelvic peritoneum is debatable; ovaries are extremely common. Both are very frequent. Given Q3A says "inflammatory condition" - which is true - and Q3E is about location, the question might be looking for the most encompassing initial description. If forced to pick one commonest, ovaries are often cited, but peritoneum is also very common.) (Q679 All true regarding endometriosis except: More common in multiparous women (B) is FALSE. It's often associated with nulliparity or delayed childbearing, though it can occur in multiparous women.)*
- **Pathogenesis Theories (Still Debated):**
  - **Retrograde Menstruation (Sampson's Theory):** Most widely accepted. Endometrial cells flow back through fallopian tubes and implant in pelvis. *(Q299 Factors in initiation/spread of endometriosis except: The monthly rupture ovarian follicle theory (B). While ovarian involvement is common, follicular rupture itself isn't a primary pathogenesis theory like retrograde menstruation (C), Mullerian metaplasia (D), or lymphatic/vascular spread (E). Genetic predisposition (A) is also recognized.)*
  - **Coelomic Metaplasia:** Peritoneal cells transform into endometrial-like cells.
  - **Lymphatic/Vascular Spread:** Explains distant endometriosis.
  - **Immune Dysfunction:** Impaired clearance of ectopic endometrial cells.
  - **Genetic Predisposition.**
- **Clinical Features:**
  - **Pain (Classic Triad, but variable):**
    1. **Dysmenorrhea:** Cyclical pelvic pain, often starting before menses and lasting longer.
    2. **Dyspareunia:** Deep pain during intercourse (especially with uterosacral/pouch of Douglas involvement).
    3. **Chronic Pelvic Pain (CPP):** Non-cyclical pain lasting >6 months.
  - **Subfertility/Infertility:** Due to adhesions, distorted anatomy, inflammation, altered peritoneal fluid, impaired ovulation/implantation.
  - **Dyschezia:** Painful defecation, especially during menses (if bowel involvement).
  - **Urinary Symptoms:** Dysuria, urgency (if bladder involvement).
  - **Abnormal Uterine Bleeding:** Less common as a primary symptom but can occur.
  - **Asymptomatic:** Can be an incidental finding at laparoscopy.
  - *(Q3C Commonest symptom is excessive menstrual loss is FALSE. Pain (dysmenorrhea, CPP, dyspareunia) is far more common. Q97 Which is most likely a symptom of adenomyosis: Dysmenorrhea (C). Adenomyosis and endometriosis share dysmenorrhea as a key symptom, though adenomyosis often has menorrhagia too. Q216 (also listed under fibroids) 45yo lady, severe secondary dysmenorrhea, HMB, deep dyspareunia. Endometriosis (B) or Adenomyosis (E) are strong contenders. Q294 Characteristic triad (dysmenorrhea, dyspareunia, dyschezia) typical guide to Endometriosis (E). Q391 Common causes of dyspareunia except: Dysmenorrhea (C). Dysmenorrhea is painful periods; dyspareunia is painful intercourse. While a woman with endometriosis might have both, dysmenorrhea isn't a cause of dyspareunia. Endometriosis (A), Leiomyoma (B - if pressing), Lichen sclerosus (D), PID (E) can all cause dyspareunia. Q398 Most common symptom of endometriosis: Pain that gets worse during the menstrual period (B) - i.e., dysmenorrhea. Q571 Common causes of dyspareunia except: Dysmenorrhea (C) - as above. Q354 Types of pain related to endometriosis except: Postcoital (D). While dyspareunia (pain during coitus) is classic, specifically isolated postcoital pain is less typical than menstrual, premenstrual, or midcycle pain if ovulation is affected.)*

- **Diagnosis:**
    - **History & Pelvic Exam:** May reveal tenderness, fixed retroverted uterus, adnexal mass (endometrioma), nodules on uterosacral ligaments. Exam often normal.
    - **Ultrasound (TVUS):** Primarily to detect endometriomas (characteristic "ground glass" appearance). May not show superficial peritoneal implants.
    - **MRI:** Better for deep infiltrating endometriosis (DIE) and complex cases.
    - **CA-125:** May be elevated, especially with endometriomas, but non-specific (not for diagnosis or screening). *(Q389 Regarding endometriosis, all correct except: Measuring serum CA125 is useful as screening test (A) is FALSE. It's not a screening test.)*
    - **Laparoscopy with Histological Confirmation: GOLD STANDARD** for diagnosis. Allows direct visualization of implants, adhesions, and biopsy for confirmation. *(Q3D Biopsy is a must for diagnosis - generally true for definitive diagnosis, usually via laparoscopy.)*
- **Management (Tailored to Symptoms, Severity, Age, Fertility Desires):**
    - **Expectant:** If asymptomatic or mild.
    - **Medical (Pain Relief & Suppression of Lesions):**
        - **NSAIDs:** For pain.
        - **Hormonal Contraceptives (COCPs, Progestins - oral, injectable, LNG-IUS):** First-line. Induce endometrial atrophy, reduce inflammation. Can be used continuously to achieve amenorrhea. *(Q389E Combined oral contraceptive pills are suitable for long-term use - TRUE for suppression.)*
        - **GnRH Agonists (e.g., Leuprolide, Goserelin):** Induce medical menopause (hypoestrogenic state) → atrophy of implants. Very effective for pain. Side effects (menopausal symptoms, bone loss) limit use to ~6 months unless add-back therapy (low-dose estrogen/progestin) is given.
        - **Danazol:** Androgenic steroid. Suppresses ovarian function. Significant androgenic side effects limit use.
        - **Aromatase Inhibitors (e.g., Letrozole):** Reduce estrogen production. Used for severe/refractory cases, often with progestin or GnRH agonist.
    - **Surgical (Laparoscopy):**
        - **Conservative Surgery:** Excision or ablation (laser, diathermy) of endometriotic implants, adhesiolysis, removal of endometriomas. Aims to relieve pain and/or improve fertility. *(Q30 Women seeking immediate fertility with Endometriosis would MOST likely benefit from: Laparoscopic ablation of the lesion (C). Danazol (A) and GnRH agonist (D) suppress ovulation. Aromatase inhibitor (B) is not first-line for fertility. Laparoscopic staging (E) is diagnostic, not therapeutic for fertility per se.) (Q157 Surgical treatment of moderate to severe endometriosis improves spontaneous pregnancy rates (A) - TRUE. Any endometriomas should be surgically removed prior to IVF (B) - Controversial, depends on size/symptoms, risk to ovarian reserve. Surgical treatment can lead to reduction of ovarian reserve (C) - TRUE, especially with ovarian cystectomy. IVF is treatment of choice if ART required (D) - TRUE. Confirmation of tubal patency during laparoscopy does not exclude fertility problems (E) - TRUE, as endometriosis can affect fertility in other ways.)*
        - **Definitive Surgery:** Total hysterectomy with bilateral salpingo-oophorectomy (TAH-BSO). For severe disease in women who have completed childbearing and failed other treatments. May still require HRT if BSO performed premenopausally.
- **Endometriosis and Infertility:**
    - Management options: Expectant, ovulation induction +/- IUI (if mild endo), laparoscopic surgery to restore anatomy/remove lesions, IVF (often most effective if other methods fail or severe disease/tubal factor).

Brutal Honesty Checkpoint:

- **Endometriosis is Painful:** Dysmenorrhea, dyspareunia, chronic pelvic pain are key. Don't forget subfertility.
- **Diagnosis:** Laparoscopy is gold standard. US is good for endometriomas.
- **Management is Symptom-Driven & Goal-Oriented:**
    - Pain? NSAIDs, Hormonal (OCPs/progestins first, then GnRH agonists).
    - Fertility? Surgery to restore anatomy, then consider OI/IUI or IVF.

- **It's Chronic:** No definitive "cure" other than TAH-BSO (which has its own consequences). Focus on managing symptoms and achieving patient goals.

MISC QUESTIONS.

The question numbers listed for MISC QUESTIONS are:

(Q39, 44, 48, 53, 55, 59, 64, 71, 73, 74, 76, 84, 89, 90, 105, 107, 111, 130, 132, 133, 135, 136, 150, 168, 176, 180, 204, 214, 217, 240, 241, 245, 248, 281, 292, 293, 301, 314, 318, 319, 337, 345, 367, 385, 390, 391, 402, 403, 410, 420, 425, 426, 427, 428, 447, 449, 450, 452, 461, 465, 475, 482, 486, 500, 506, 512, 523, 529, 530, 545, 554, 558, 559, 560, 573, 575, 577, 586, 592, 595, 604, 609, 610, 611, 612, 619, 639, 641, 645, 652, 656, 663, 664, 666, 675, 684, 686, 687, 688, 689, 699, 700, 701, 705, 706, 712, 716, 717, 719, 733, 743, 744, 747, 752, 759, 760, 766, 767, 771, 772, 773, 788, 790, 795)

Gynecology & Obstetrics: Miscellaneous High-Yield Points & Rapid Review

Alright, this is the "grab bag" section. These questions jump around, hitting various fundamental concepts, anatomy, definitions, and a few clinical pearls. Don't underestimate these – they often test your ability to recall key facts quickly and apply basic principles.

Key Miscellaneous Takeaways & Rapid Review Points (with illustrative Q numbers):

Basic Anatomy & Embryology – You Can't Escape It:

- **Vulva Blood Supply:** Pudendal artery is the main player. *(Q23)*
- **Ovarian Anatomical Relations:** Know what's anterior, posterior, superior. The broad ligament is posterior to the fallopian tube. *(Q39B Broad ligament posterior to tube is correct, Fallopian tube anteriorly (A) is also correct. Bowel/omentum superiorly (C) is true. External iliacs posteriorly (D) is true. Pelvic sidewall laterally (E) is true. This question's "except" might be subtle or depend on specific definitions of anterior/posterior relations).*
- **Mullerian Duct Derivatives:** Fallopian tubes, uterus, cervix, upper 2/3 of vagina. Ovaries are NOT Mullerian. *(Q150 Not derived from Mullerian ducts: Distal third of the vagina (E) arises from urogenital sinus. Fallopian tubes (A), Uterine body (B), Cervix (C), Proximal two-thirds of vagina (D) are Mullerian.)*
- **Urogenital Ridge Derivatives:** Does NOT give rise to the vulva. Gives rise to kidneys, gonads, genital ducts. *(Q136 Urogenital ridge doesn't give rise to: Vulva (E). Uterus (A), Fallopian tube (B), Ureter (C - from ureteric bud, part of urogenital system), Cervix (D) are related to structures from this ridge system.)*
- **Genital Tubercle Origin:** Clitoris in females, penis in males. *(Q217 Genital tubercle gives rise to Clitoris (C)).*
- **Genital Folds/Swellings:** Folds → labia minora. Swellings → labia majora. *(Q611 Genital swellings give rise to Labia majora (D). Q706 Genital folds give rise to Labia minora (B).)*
- **Para-mesonephric (Mullerian) Duct Derivatives:** Fallopian tubes, uterus, cervix, upper vagina. *(Q612 All from para-mesonephric except: Ovary (C) - arises from germinal epithelium/gonadal ridge).*
- **Fetal Hematopoiesis Origin:** Yolk sac first, then liver, then bone marrow. *(Q717 Fetal hematopoiesis first occurs in Yolk sac (B)).*
- **Decidua:** Modified endometrium of pregnancy. *(Q426 True: It is modified endometrium (A).)*
- **Chorion:** Forms chorionic villi. *(Q427 True: Forms the chorionic villi (C).)*
- **Uterine Cavity Obliteration:** Fusion of decidua capsularis and parietalis around 10-14 weeks. *(Q486, Q700: 10 weeks (C) or 14 weeks (D) are in the ballpark. Let's take the average/commonly cited, which is around 12-16 weeks. So, Q486C/Q700D might be reasonable approximations depending on source.)*
- **Canalization of Vagina:** Occurs around 20 weeks gestation. *(Q529A)*
- **Ovarian Vein Drainage:** Right ovarian vein → IVC. Left ovarian vein → Left renal vein. *(Q95 Left ovarian vein to Renal vein (C). Q530 Right ovarian vein to Inferior vena cava (B).)*
- **Pelvic Support Ligaments:** Cardinal and uterosacral ligaments are key for apical support (Level 1). *(Q555, Q737C)*
- **Mid Cavity of Pelvis Boundaries:** *(Q240 Except: Bounded posteriorly by junction of L4 S1 (D). Sacrum forms posterior boundary, L4/S1 is too high for mid-cavity.)*

- **Ligament providing most support to uterus:** Cardinal ligaments. *(Q420D)*
- **Sensory Pain Fibers to Uterus:** Pass through uterosacral ligaments (among others like inferior hypogastric plexus). *(Q214D Uterosacral ligament is plausible.)*

Physiology Snippets:

- **Oogenesis:** Highest number of oocytes at ~20 weeks gestation (fetal life). *(Q771B)* Secondary oocyte arrested in metaphase II until fertilization. *(Q461C Second meiotic division completed only at ovulation is FALSE, it's at fertilization).*
- **Fertilization Window:** Ova usually fertilized within 12-24 hours of ovulation. *(Q284B 12 hours is a good window. Q733A 12-24 hours.)*
- **Implantation:** Blastocyst implants around 6-7 days after fertilization. *(Q132B 6.5 days.)*
- **Endometrial Phase After Ovulation:** Secretory phase. *(Q747C)*
- **Basal Body Temperature:** Rises after ovulation due to progesterone. *(Q746C Thermogenic effect of progesterone.)*
- **Day After Ovulation Changes:** *(Q348 Except: Plasma progesterone concentration falls (C) is FALSE; it rises.)*
- **Glycoprotein Hormones:** FSH, LH, TSH, hCG. HPL is NOT a glycoprotein. *(Q701E HPL is a polypeptide.)*
- **Hormones in Adolescent Growth Spurt:** GH, Estradiol, IGF-I, Somatomedin-C. Progesterone is NOT primarily for linear growth. *(Q719E Progesterone.)*
- **Normal Menstrual Blood Loss:** Average ~30-40mL, up to 80mL. *(Q405C 25-50mL is a good average range.)*

Definitions & Clinical Scenarios:

- **Kallmann's Syndrome:** Hypogonadotropic hypogonadism + anosmia → Amenorrhea. *(Q181C)*
- **Turner's Syndrome:** 45,XO. Streak gonads, short stature, webbed neck, etc. *(Q131B Incidence rises with maternal age is INCORRECT.)*
- **Androgen Insensitivity Syndrome (AIS):** 46,XY. Female phenotype, absent uterus, present testes. Normal pubic/axillary hair if some androgen receptor function for hair follicles, or if it's partial AIS. Complete AIS may have sparse/absent hair. *(Q201E "Normal pubic/axillary hair as they have male testosterone level" - This is FALSE for Complete AIS where receptors are absent; hair development is often sparse/absent despite normal male testosterone levels. Partial AIS is more complex.) (Q463 Karyotype of AIS is 46,XY (B)).*
- **Asherman's Syndrome:** Intrauterine adhesions → Hypomenorrhea/Amenorrhea. *(Q51A)*
- **Empty Sella Turcica Syndrome:** *(Q137C Induction of ovulation is contraindicated is FALSE.)*
- **Precocious Puberty:** *(Q606 First sign of puberty (girls) is Budding of breasts (A).)*
- **Primordial Germ Cells Origin:** Wall of yolk sac close to allantois. *(Q687C)*
- **Prostaglandin Synthesis:** Occurs in many tissues. Red cells do NOT synthesize prostaglandins. *(Q688C Red cell.)*
- **Oogenesis - First Maturation Division:** Completed just before ovulation (most sources) or starts in fetal life and arrests, completes just before ovulation. *(Q689B Few hours before ovulation.)*
- **Ovarian Reserve Test:** AMH is best. *(Q92E)*
- **Bishop Score Components:** *(Q215B Position of presenting part is NOT a component. Q326E Presenting part position is NOT a component.)* (Cervical: Dilatation, Effacement, Consistency, Position; Station of presenting part).
- **Apgar Score Components:** *(Q91C Good eye opening is NOT part of APGAR.)* (Appearance, Pulse, Grimace, Activity, Respiration).
- **Bicornuate Uterus - Associated Anomalies:** Urinary tract. *(Q193C, Q464C)*
- **Normal Sexual Differentiation:** *(Q487B If gonads removed before differentiation, development will be female regardless of chromosomal sex is a complex statement and depends on timing and what is considered "default". Generally, without testes/androgens, female external development occurs. The "depending on chromosomal sex" part makes it tricky. Often the female pathway is considered default if no SRY/testicular influence.)*
- **Intermenstrual Bleeding (Metrorrhagia):** *(Q109C Ovulatory dysfunction causes irregular bleeding, often with variable volume, not "same bleeding volume".)*

- **Post-term Pregnancy Complications:** *(Q93C Failure of induction is a risk/complication of managing post-term, not a direct complication of post-term itself.)*
- **Death related to pregnancy:** *(Q94B Indirect maternal death definition fits.)*
- **Antenatal Care Booking Visit Time:** Optimum 8-10 weeks or earlier. *(Q73C 11-14 weeks is acceptable, especially if including NT scan. Earlier is better. Given options, 8-10 (B) or 11-14 (C) are reasonable. Q73's key says C?)*
- **28 Week Visit Routine Checks:** *(Q74E Fetal presentation is not always routinely and definitively assessed by exam at 28wks as it can change; US would be definitive if needed. Urine protein/sugar, BP, FHR are standard.)*
- **Single Umbilical Artery (SUA):** Associated with increased risk of congenital anomalies. *(Q401C)*
- **Ovarian Cancer Symptom Index:** *(Q220A Abnormal uterine bleeding is NOT a typical ovarian cancer symptom. Q430B Abnormal vaginal bleeding. Q568C Elevated CA125 is an investigation, not a symptom in the index.)*
- **Twin-to-Twin Transfusion Syndrome (TTTS):** *(Q693C Polyhydramnios in the smaller (donor) twin is FALSE; donor has oligo.)*
- **Renal Changes in Pregnancy:** GFR increases, ureteric dilatation, increased excretion of some solutes. Folate excretion is *not* decreased. *(Q556C Decreased excretion of folate is FALSE.)*
- **Hepatic Changes in Pregnancy:** *(Q726C Cholesterol level is elevated 4 times is an overestimate; it increases but not usually 4-fold.)*
- **Calcium Metabolism in Pregnancy:** *(Q727D PTH levels increase is a response to increased demand/lower ionized Ca if it tends to drop, or increased Vit D metabolism. However, some sources state PTH is normal or slightly decreased due to increased calcitriol. This is complex. "Maternal total calcium levels decline" (A) is true due to hemodilution/low albumin.)*
- **Thrombophilia & Pregnancy Complications:** *(Q59E Placenta previa is NOT directly associated with thrombophilia. Q656C Post date is NOT associated.)*
- **Maternal Self-Assessment of Fetal Well-being:** Kick counting. *(Q296B)*
- **Hematological Changes in Pregnancy:** *(Q725A "Accelerate starvation state in first few weeks" and E "Recurrent early morning hypoglycemia" are NOT typical physiological changes. B, C, D are more plausible or complex. The provided answer options for Q725 are B?D?, suggesting ambiguity or error in keys.)*
- **Plasma Hormone Increases in Pregnancy:** *(Q696 TSH (A), Albumin (B), Growth Hormone (C - hPL has GH-like effects but pituitary GH is suppressed), Catecholamines (E) do not typically show a marked sustained increase. Melanocyte Stimulating Hormone (D) does increase.)*
- **Shoulder Dystocia Complications:** Brachial plexus injury, clavicle fracture. *(Q362B Injury of brachial plexus is commonest.)*
- **Chronic HTN Definition:** *(Q661B BP >140/90 before 20 weeks or pre-existing.)*
- **Fetal Demise - Highest Modifiable Risk Factor:** Maternal overweight/obesity. *(Q266D)*
- **Complete Moles - Genetics:** Usually 46,XX (paternal monospermic empty ovum duplication) or 46,XY (paternal dispermic). *(Q161E TVU diagnosis is correct; Q535A 46,XX is commonest.)*
- **Fetal Circulation:** *(Q429A Two umbilical arteries & one umbilical vein is correct, so the statement is FALSE. Q788E Blood from IVC is largely directed through foramen ovale is TRUE.)*
- **Placenta Accreta Management:** Often hysterectomy. *(Q593C)*
- **Uterine Involution Postpartum:** *(Q237E Uterus normal size by 12 days is FALSE, takes ~6 weeks.)*
- **Oligohydramnios - Congenital Anomalies:** *(Q504C Duodenal atresia causes POLYhydramnios.)*
- **Cephalopelvic Disproportion (CPD):** *(Q29A Commonest cause of non-engagement at term in primigravida.)*

Treatment & Management Pearls:

- **Iron Deficiency Anemia Tx:** Therapeutic doses, not just 10mg. *(Q81C)*
- **Rubella in 1st Trimester:** High risk of Congenital Rubella Syndrome (CRS). *(Q106 CRS does not typically include Intra-cranial calcification (D) - that's more Toxo/CMV. Q412B First trimester is highest risk.)*
- **Classical C-Section Indications:** *(Q107E Anterior uterine wall fibroids are NOT an indication for classical C/S; usually for transverse lie back down, some premies, placenta previa anterior.)*

- **Pre-eclampsia Management - Antihypertensives:** *(Q591C Methyldopa is a safe choice for mild HTN in early pregnancy.)*
- **Amniocentesis after 16 weeks:** *(Q501B Not associated with fetal postural deformities is generally true, risk is low with experienced operator.)*
- **Post-term Pregnancy Management:** *(Q580B Determination of true length of gestation is first step/part of initial assessment.)*
- **Breech at Term - Not in Labor, Ruptured Membranes:** Vaginal exam to assess cervix and rule out cord prolapse. *(Q351C)*
- **Assisted Breech Delivery - Mandatory Step:** Rotation of fetus to sacro-anterior. *(Q352E)*
- **Placenta Accreta Diagnosis:** Ultrasound, MRI. *(Q361C Ultrasound evaluation.)*
- **Cervical Incompetence Diagnosis:** Vaginal US in index pregnancy. *(Q365E)*
- **Painful Bladder Syndrome (PBS) Symptoms:** *(Q295D Urine retention is NOT a symptom; frequency/urgency (B), pain (A), dysuria (C), dyspareunia (E) are.)*
- **Acute Pyelonephritis in Pregnancy:** Requires hospitalization and IV antibiotics. *(Q577C Oral antibiotics is FALSE for initial treatment of pyelo.)*
- **Hydatidiform Mole Suggestion:** *(Q290E Nervousness, anorexia, tremors (signs of hyperthyroidism due to hCG) is LEAST specific compared to bleeding, large uterus, hyperemesis, early toxemia.)*
- **Lichen Sclerosus Tx:** Ultrapotent topical steroids. *(Q677C Surgical excision is FALSE as main tx.)*
- **True Hermaphroditism Dx:** Presence of both testicular and ovarian tissue. *(Q778B)*
- **Ovarian Cancer Screening Tool (for evaluating mass/risk):** CA-125 (not for general population screening). *(Q444B)*
- **Imperforate Hymen Presentation:** *(Q445C Hirsutism is NOT a feature. Q602E Secondary sexual development is usually ABSENT is FALSE, it's normal.)*
- **Amniotic Fluid Embolism:** Causes hypofibrinogenemia. *(Q447D)*
- **Supine Hypotension in Pregnancy:** Compression of descending aorta by gravid uterus. *(Q449E is true; the question asks for "except" - all listed contribute or are features.)*
- **Down's Syndrome Diagnostic Testing (16 wks):** Amniocentesis. *(Q450C)*
- **Magnesium Sulfate Toxicity Antidote:** Calcium gluconate. (Related to Q202, though not explicitly asked).
- **Early Fetal Heart Decelerations Cause:** Head compression. *(Q191B)*
- **Persistent Mullerian Duct Syndrome (Male with Uterus/Tubes):** Lack of Mullerian-Inhibiting Factor (AMH) or its receptor. *(Q192A)*
- **Chorionic Villus Sampling (CVS) Timing:** 10-13 weeks. *(Q248B Between 10-12 weeks.)*
- **Hypothyroidism Screening in Pregnancy:** TSH. *(Q731A)*
- **IUGR Fetal Biometry:** Serial Abdominal Circumference (AC). *(Q732C)*
- **HELLP Syndrome - Maternal Death Association:** Cerebral hemorrhage or hepatic rupture. *(Q647B or C.)*
- **Anticoagulation Duration Postpartum for DVT:** Typically 6 weeks to 3-6 months depending on specifics. *(Q648B Six weeks is minimum.)*

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