Obstetrics & Gynecology Comprehensive Summary

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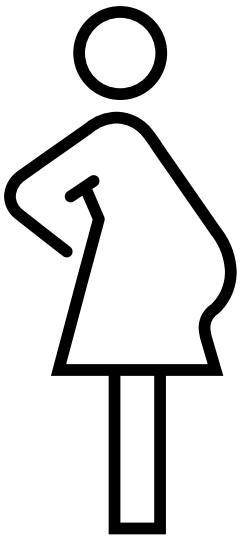


REFERENCES

SLIDES AND PP

OUTLINES

OBSTETRICS

Obstetrics Ultrasound Scan

I. Antenatal Care Basics and Booking Visit

- **Optimum Booking Time:** The ideal time for the first booking visit is between **11 and 14 weeks** gestation.
- **Routine Investigations:** Standard booking tests include a **Complete Blood Count (CBC)**, **Blood Grouping and Rh typing**, **Rubella titer (IgG and IgM)**, **Hepatitis screen**, **HIV**, **TSH**, **Random Blood Sugar**, and **Urinalysis**.
- **Excluded Routine Tests: Liver function tests (LFT)**, the **Glucose Tolerance Test (GTT)** at booking, and the **Direct Coombs test** are not part of routine booking investigations.
- **Screening Conditions:** Routine screening is offered for Down syndrome, Hepatitis C, Rubella, and HIV, but not typically for Cystic Fibrosis.
- **Calculating EDD:** The expected date of delivery (EDD) is based on an average pregnancy length of **280 days**. The **Last Menstrual Period (LMP)** is only reliable if cycles were regular for at least **three cycles** prior to conception. Ultrasound is generally more accurate than LMP for dating.
- **Symphysis–Fundal Height (SFH):** Measured from **24 weeks** gestation. The mean height is ~20 cm at 20 weeks. A large SFH may indicate polyhydramnios or twins, while a small SFH suggests growth restriction (IUGR).

II. First Trimester Ultrasound (Up to 13+6 Weeks)

- **Indications:** Suspected miscarriage, vaginal bleeding, uncertain gestational age, multiple gestation, hydatidiform mole, and IUD localization.
- **Visualization:** An intrauterine gestational sac is visible via transvaginal ultrasound (TVS) when **-hCG levels are >1000–1500 IU/L**. The fetal heartbeat can be detected by TVS as early as **6 weeks**.
- **Dating Methods:**
    - **Mean Sac Diameter (MSD):** Used when no fetal pole is visible.
    - **Crown-Rump Length (CRL):** The measurement of choice at **9 weeks** and up to 14 weeks.
- **Chromosomal Screening (11–13+6 Weeks):**

    - **Markers:** Includes **Nuchal Translucency (NT)**, maternal age, weight, smoking status, and serum levels of **PAPP-A and free -hCG**.
    - **Biochemical Trends:** In Trisomy 21, **free -hCG is high** (not low). Smoking and diabetes decrease -hCG and PAPP-A levels, which can increase screen-positive results.
    - **Aneuploidy Markers:** Increased NT (>3mm), an **absent nasal bone**, tricuspid regurgitation, and reverse flow in the ductus venosus are all markers for aneuploidy.
    - **Triple Screening:** Used primarily for Down syndrome and Edward syndrome.

III. Ultrasound Physics and Safety

- **Frequencies:** Ultrasound uses mechanical vibrations **above 20 kHz**.
    - **Transabdominal (TA):** Uses **2–5 MHz** probes.
    - **Transvaginal (TV):** Uses **5–9 MHz** probes.
- **Resolution vs. Depth:** Higher frequency increases resolution but provides a **shallower depth** of view.
- **Safety:** There is no evidence that ultrasound causes neonatal injury through cavitation, heating, or cell death. This is due to low total sound exposure, a short "duty cycle," and the distance of the fetus from the energy source.

IV. Second Trimester (18–23 Week Anomaly Scan)

- **Purpose:** This is the best window for anatomical evaluation because organ systems are mature enough to visualize, yet early enough for intervention if anomalies are found.
- **Assessments:** Includes fetal number, cardiac activity, anatomy (head, face, spine, heart, etc.), placental position, and amniotic fluid.
- **Head Biometry (BPD/HC):** The **Biparietal Diameter (BPD)** and **Head Circumference (HC)** are used for dating from 14 weeks.
    - **Correct BPD Plane:** Must show the falx cerebri, cavum septum pellucidum, and thalamus; it must **not** show the cerebellum or orbits.
- **Cervical Length:** A closed cervical length should be **>2.5 cm**.

V. Third Trimester (Growth and Welfare)

- **Indications:** IUGR, macrosomia, fluid abnormalities (oligo/polyhydramnios), decreased fetal movements, and PIH.
- **Assessment:** Focuses on fetal presentation, size (BPD, HC, AC, FL), placental localization, and wellbeing.
- **Amniotic Fluid Volume:**
    - **Single Deepest Pool:** Normal is 2–8 cm; <2 cm is oligohydramnios, >8 cm is polyhydramnios.
    - **Amniotic Fluid Index (AFI):** Normal range is **5–25 cm**.
- **Doppler Studies:** Assesses fetal blood flow in sites like the **umbilical artery**, **middle cerebral artery (MCA)**, and **ductus venosus**. Normal ductus venosus flow is antegrade; reverse flow indicates hypoxemia.

VI. Diagnostic and Invasive Procedures

- **Most Common Invasive Test: Amniocentesis** is the most frequent invasive diagnostic procedure.
- **Timing:** Amniocentesis is typically performed at **14–16 weeks**.
- **Accuracy:** For advanced maternal age, **Chorionic Villus Sampling (CVS)** is considered the most accurate test for Down syndrome early in pregnancy.
- **Capabilities:** Amniocentesis can diagnose chromosomal issues (Turner, Edward syndrome) and infections like toxoplasmosis.

VII. Key Clinical Findings to Memorise

- **Rubella:** Infection in the first trimester increases the risk of microcephaly, congenital cataracts, heart disease, and deafness, but not typically intracranial calcification.
- **Abdominal Palpation:** Used to assess fetal number, size, presentation, and engagement, but not the **station** of the presenting part (which requires a vaginal exam).
- **Adnexal Mass Malignancy:** Ultrasound scores depend on multilocular cysts, solid areas, bilateral lesions, and ascites, but **not endometrial thickness**.
- **Pelvic Exam:** A bimanual exam can evaluate uterine size, direction, and masses, but it cannot reliably evaluate the size of **normal adnexae**.

Physiological Changes In Pregnancy

1. Cardiovascular System & Hemodynamics

- **Cardiac Output (CO):** Increases by **40%** during pregnancy, with 20% of that increase occurring by 8 weeks. Max CO is reached at **20–28 weeks**.
    - **Labor Impact:** CO increases further by **15% in the first stage** and **50% in the second stage**. It returns to pre-pregnancy levels within 2 weeks of delivery.
- **Components:** The increase in CO is driven by rises in both **stroke volume** and **heart rate**.
- **Vascular Resistance: Systemic Vascular Resistance (SVR)** and **Peripheral Vascular Resistance (PVR) decrease** due to peripheral vasodilation (PVD).
- **Blood Pressure (BP):** BP falls from the start of pregnancy, reaching its **nadir (lowest point) at 20–24 weeks** before returning to pre-pregnancy levels at term.
    - **Measurement:** Use Phase V (disappearance) rather than Phase IV (muffling) Korotkoff sounds. The patient should be sitting or lying with a **30-degree tilt**.
- **Normal Physical & ECG Findings:**
    - Physiological heart dilation and increased myocardial contractility.
    - **Normal sounds:** Ejection systolic murmur, loud first heart sound, and a third heart sound. **Diastolic murmurs are NOT normal**.
    - **Normal ECG:** Q wave in lead III, inverted T in lead III, premature atrial/ectopic beats, and a slight left axis deviation. **ST segment elevation is NOT normal**.

2. Respiratory System

- **Ventilation:** Minute ventilation increases by **40–50%**, primarily due to a **30–40% increase in tidal volume**.
- **Lung Capacities:**
    - **Decreased:** Functional residual capacity (FRC) and residual volume (by ~20%).
    - **Unchanged: Respiratory rate**, vital capacity, FEV1, peak expiratory flow rate (PEFR), and total lung capacity.
- **Blood Gases:** Hyperventilation causes a **reduction in PaCO2** and a compensatory fall in serum bicarbonate. **PaO2 remains unchanged**.

- **Metabolism:** O2 consumption increases by 20% and the metabolic rate rises by 15%.

3. Renal System

- **Anatomy:** The kidneys increase in size. There is dramatic dilation of the renal collecting system (ureters/pelvicalyceal system), which is **more prominent on the right side** due to progesterone and uterine dextrorotation.
- **Function:**
    - **GFR increases by 50%**.
    - **Renal Plasma Flow (RPF):** Increases 60–80% by the second trimester, then decreases slightly but remains 50% higher than pre-pregnancy levels at term.
- **Biochemical Changes:**
    - **Serum urea and creatinine fall** due to increased clearance.
    - **Glucosuria:** The renal threshold for glucose falls, making trace glucose in urine a normal finding.
    - **Proteinuria:** Protein excretion increases (up to 300mg is normal vs 150mg non-pregnant).
    - **Excretion:** Increased excretion of urate; however, excretion of **folate increases** (it does not decrease).

4. Hematology & Coagulation

- **Blood Volume:** Plasma volume increases significantly (50%), while Red Cell Mass increases proportionately less (20–30%), leading to **physiological hemodilution (decreased hematocrit)**.
- **White Blood Cells:** Polymorphonuclear leukocytes (WBC count) increase.
- **Platelets:** Count may decrease slightly; at term, it is approximately 10% less than pre-pregnancy levels.
- **Coagulation (Hypercoagulable State):**
    - **Increased Factors:** Fibrinogen (dramatic rise), Factors II, VII, VIII, IX, and X. **Factor XI and Factor V do not increase** (Factor XI actually decreases).
    - **Decreased Anticoagulants:** Anti-Thrombin III and Protein S.
    - **Fibrinolysis:** Activity is reduced.

    - **Risks:** Venous stasis in lower limbs (worse on the left) increases DVT risk, lasting until 6 weeks postpartum.

5. Hepatobiliary & Gastrointestinal (GIT)

- **GIT Motility:** Progesterone causes **decreased lower esophageal sphincter pressure** (causing reflux), decreased gut peristalsis, and delayed gastric emptying (causing **constipation**).
- **Liver Function:**
    - **Total serum protein and albumin decrease** due to dilution.
    - **Alkaline Phosphatase:** Rises **3–4 times** the normal value.
    - **ALT/AST:** Slightly fall or remain normal.
    - **Biliary:** Increased biliary cholesterol saturation.
- **Symptoms:** Nausea, vomiting, and common palmar erythema or spider nevi (due to estrogen).

6. Endocrine & Metabolism

- **Thyroid:** TBG increases. Total T3/T4 rise, but **Free T3/T4 remain constant**. hCG mimics TSH, causing **TSH to fall in the 1st trimester**. Pregnancy is a state of relative **iodine deficiency** due to active transport to the fetus and increased renal loss.
- **Pituitary:** Anterior pituitary volume increases by 35%. **Prolactin increases 10x**. **LH and FSH are suppressed**.
- **Adrenal:** Cortisol (free and bound), ACTH, Angiotensin II, and Renin all increase. **Aldosterone increases 10-fold** by the 3rd trimester.
- **Glucose Control:** Pregnancy is a state of **physiological insulin resistance** (driven by placental hormones: **hPL**, glucagon, cortisol). Fasting glucose levels decrease, while post-prandial levels increase.
- **Calcium:** Calcium is actively transported across the placenta. While maternal total calcium levels decline, **serum ionized calcium stays stable**.
- **Weight & Fluid:** Average weight gain is **12.5 kg** (range 10–15 kg). Total body water increases by approximately **3L** (mostly extracellular).

7. Skin & Uterus

- **Pigmentation:** Increases (Melasma/face, darkened areola) due to Melanocyte-stimulating hormone.
- **Striae Gravidarum:** New stretch marks are pink; old ones are white.
- **Hirsutism:** Increased hair growth is a normal skin change. Hair fall typically occurs 2–20 weeks postpartum.
- **Uterus:** Generally **rotates to the right** (dextrorotation) because of the rectosigmoid colon.

Miscarriages

1. Core Definitions and Diagnostic Classifications

A **miscarriage** is the spontaneous loss of pregnancy at or before **24 weeks** of gestation. It is a common occurrence, affecting approximately **20% of clinical pregnancies**, with the highest frequency in the first trimester.

The clinical classification depends heavily on the state of the cervical os and ultrasound findings:

- **Biochemical Pregnancy:** A loss that occurs so early it is only documented by a positive pregnancy test that subsequently becomes negative before any evidence of pregnancy is visible on a scan.
- **Pregnancy of Unknown Location (PUL):** A scenario where the pregnancy test is positive, but no pregnancy is visible inside or outside the uterus on ultrasound.
- **Threatened Miscarriage:** Vaginal bleeding occurs, but the pregnancy remains viable as confirmed by ultrasound.
- **Inevitable Miscarriage:** This is diagnosed when a patient presents with vaginal bleeding and abdominal pain, and clinical examination reveals an **opened cervical os** while the pregnancy tissue is still entirely present within the uterus.
- **Incomplete Miscarriage:** Characterized by ongoing vaginal bleeding and a history of passing tissue. Ultrasound shows **retained products of conception (RPOC) >15 mm** in diameter. It is critical to note that an "empty uterus" on a scan is a counter-indicator for this diagnosis.
- **Complete Miscarriage:** Bleeding has ceased, the **cervical os is closed**, and the uterus is either empty or contains RPOC <15 mm.
- **Missed Miscarriage (Early Fetal Demise):** Often presents with minimal or no symptoms. It is diagnosed via ultrasound when the gestational sac is **>25 mm** with no yolk sac or embryo, or the **Crown Rump Length (CRL) is >7 mm with no fetal heart activity**.
- **Septic Miscarriage:** A miscarriage complicated by infection, most commonly following an induced abortion via **ascending infection**. Common causative organisms include **E. coli, Bacteroides, Streptococci, and Clostridium welchii**. Notably, **Staphylococcus is NOT** typically a causative organism in this clinical context.

2. Etiology and Risk Factors

The causes of miscarriage are multifaceted, ranging from genetic to structural factors.

- **Genetic Factors:** Roughly **50% of cases** are due to chromosomal abnormalities. **Numerical abnormalities** (such as autosomal trisomy) are significantly more common than structural abnormalities. In most instances, the parents possess a normal chromosomal pattern.
- **Maternal Health and Lifestyle:**
    - **Age:** Maternal age is a dominant factor; the risk is 20% at age 20 but rises to **93% by age 45**.
    - **Medical Conditions:** Poorly controlled diabetes, thrombophilia, and certain infections like **Listeria monocytogenes** or Rubella.
    - **Lifestyle:** Obesity (BMI >30), smoking, heavy alcohol use, and certain medications like methotrexate.
- **Uterine and Structural Factors:**
    - **Septate Uterus:** Specifically linked to **recurrent first-trimester** pregnancy loss.
    - **Intrauterine Adhesions (Asherman Syndrome):** The most frequent cause is **endometrial curetting** performed after a previous abortion or postpartum.
    - **Cervical Weakness:** This is a classic cause of **second-trimester** losses, often presenting as painless cervical dilatation.
    - **Note on Risk:** A previous large loop excision of the transformation zone (LLETZ) of the cervix is generally **not** considered a risk factor for first-trimester loss.

3. Advanced Diagnostic Evaluation

Diagnosis relies on a combination of clinical history, serial hormone monitoring, and Transvaginal Ultrasound (TVS).

- **Ultrasound Milestones:** A gestational sac should be visible at 5 weeks, the yolk sac and embryo at 6 weeks, and the amnion at 7 weeks.
- **hCG Dynamics:** In a healthy pregnancy, beta-hCG levels typically **double every 48 hours** (or increase by >63%).
    - An hCG ratio (48hr/0hr) **<0.8** is strongly suggestive of a missed miscarriage.

    - A ratio between **0.8 and 1.66** indicates a high likelihood of an ectopic pregnancy or PUL.

- **Biopsy Evidence:** The presence of **chorionic villi** in uterine curetting is the only conclusive evidence of an intrauterine pregnancy.
- **Differential Diagnosis:** The **passage of decidual casts** is a specific sign associated with **tubal (ectopic) abortion**, rather than a standard miscarriage.

4. Management Protocols

Expectant and Medical Management

- **Expectant:** "Nature taking its course" is most successful in cases of incomplete miscarriage (70% success rate).
- **Medical: Misoprostol (Cytotec)** is the primary drug used for missed miscarriages between 8–10 weeks. It is a prostaglandin analogue that binds to myometrial cells to cause contractions and cervical ripening. Vaginal administration is often preferred to reduce gastrointestinal side effects.

Surgical Management (ERPC/D&C)

Surgical intervention is indicated for excessive bleeding, infection, or hemodynamic instability.

- **Instrumentation:** Standard tools include a **Sound**, **Hegar dilators** (used to dilate up to 8–10 mm), **Vulsellum**, and **blunt or suction curettes**. **Sharp curettes are NOT used** due to increased trauma risks.
- **Complications:** Uterine perforation occurs in 5 per 1000 cases; if suspected, **hysteroscopy and laparoscopy** are performed to assess visceral damage.
- **Infection Protocol:** In cases of septic abortion, surgery should be **delayed for 12 hours** to allow for the administration of intravenous antibiotics.

Rhesus Prophylaxis

Anti-D immunoglobulin must be administered to non-sensitized RhD-negative women in the following scenarios:

- Any miscarriage at **>12 weeks** gestation.
- A miscarriage at **<12 weeks** IF **surgical instrumentation** (curettage) is performed.
- Threatened miscarriage below 12 weeks if the bleeding is heavy, repeated, or associated with pain.

5. Recurrent Miscarriage (3+ Consecutive Losses)

Recurrent loss affects roughly 1% of couples and requires specialized investigation.

- **Antiphospholipid Syndrome (APS):** A major, treatable cause of recurrent loss.
    - **Diagnostic Criteria:** Requires **two abnormal readings** (lupus anticoagulant, anticardiolipin, or anti-β2 glycoprotein-1) separated by **at least 12 weeks**. A single reading is insufficient.
    - **Mechanism:** It causes pregnancy loss through **placental thrombosis and infarction**.
    - **Pregnancy Outcomes:** Associated with IUGR, early-onset pre-eclampsia, and thrombosis, but **NOT polyhydramnios**.
    - **Treatment:** A combination of **low-dose Aspirin and LMWH** reduces the risk of further miscarriage by 45%.
- **Endocrine Factors:** PCOS is linked to insulin resistance and miscarriage; **weight loss** is a highly recommended simple management step.
- **Factors NOT linked to recurrent loss:** Research indicates that pituitary microadenomas and the age of the male partner are **not** significant risk factors for recurrent miscarriage.

Ectopic Pregnancy

Definition and Epidemiology

An ectopic pregnancy occurs when a blastocyst implants **outside of the uterine endometrium**. This condition accounts for approximately **0.5% to 1.5%** of all first-trimester pregnancies. Early diagnosis is critical for maternal survival and is primarily achieved through **transvaginal sonography** and **serum β-hCG assays**.

Locations of Implantation

The vast majority of ectopic pregnancies (**95%**) occur within the fallopian tube.

- **Ampullary part:** The **most common site**, accounting for 70% of cases.
- **Isthmic:** 12%.
- **Fimbrial:** 11%.
- **Interstitial:** 2%.
- **Nontubal sites:** These include the ovary, cervix, peritoneal cavity, and prior C-section scars.
- **Heterotopic pregnancy:** A rare occurrence (1:30,000 naturally) where one pregnancy implants normally in the uterus while another is in an ectopic location.

Risk Factors

Understanding risk factors is essential for clinical suspicion:

- **Strongest Risk Factor:** A **previous ectopic pregnancy** increases the risk fivefold.
- **Tubal Factors:** History of pelvic inflammatory disease (PID), salpingitis (which distorts anatomy), tubal ligation, or previous pelvic surgery.
- **Lifestyle and Others: Smoking**, use of fertility drugs or assisted reproductive technology (ART), and endometriosis.
- **Contraception and Myths: Combined oral contraceptive pills and fibroids do NOT increase the risk** of ectopic pregnancy. While intrauterine devices (IUDs) do not increase the absolute risk compared to the general population, they do increase the *relative* risk that a pregnancy, if it occurs, will be ectopic.

Clinical Manifestations and Symptoms

The presentation can vary from asymptomatic to life-threatening:

- **Most Common Symptom: Amenorrhea** (missed period).
- **The Classic Triad:** Delayed menstruation, pelvic/abdominal pain, and vaginal bleeding or spotting.
- **Signs of Rupture:** Sudden, **severe, sharp stabbing pain** in the lower abdomen.
- **Referred Pain: Shoulder tip pain** or neck pain can occur due to diaphragmatic irritation from blood in the peritoneal cavity (hemoperitoneum).
- **Systemic Symptoms:** Feeling faint, dizzy, nausea, and vomiting. Notably, **diarrhea is NOT a typical symptom** of a ruptured ectopic pregnancy.
- **Mortality:** The leading cause of death associated with ectopic pregnancy is **hypovolaemia** due to hemorrhage.

Diagnostic Findings

- **Sonography: Identification of an intrauterine pregnancy (IUP)** is the single most important finding to **exclude** ectopic pregnancy.
- **Ultrasound Markers:** Findings may include an empty uterus, a complex adnexal mass, or a "trilaminar" endometrial pattern. For cervical pregnancies, ultrasound is the primary diagnostic tool.
- **Endometrial Changes:** The specific change seen in the endometrium during an ectopic pregnancy is **decidual transformation**.
- **Physical Exam:** Patients may exhibit cervical excitation pain or a bulging posterior vaginal fornix if blood has collected in the rectouterine cul-de-sac.
- **Hemoperitoneum:** Free fluid in Morison's pouch (near the liver) suggests significant internal bleeding of at least 400 ml.

Management Strategies

1. Medical Management (Methotrexate)

Methotrexate (MTX) is a folic acid antagonist that arrests DNA, RNA, and protein synthesis.

- **Indications:** The patient must be hemodynamically stable, asymptomatic, and compliant.

- **Contraindications:**
    - **β-hCG > 5000 IU/L**.
    - Presence of **fetal heart activity**.
    - Gestational sac **greater than 4 cm**.
    - Patient is breastfeeding, immunodeficient, or has hepatic/renal dysfunction.
- **Monitoring:** Serum β-hCG levels are checked on days 4 and 7; if the level does not decline by at least 15%, a second dose or surgery may be required.

2. Surgical Management

The choice of surgery depends on the patient's stability and desire for future fertility:

- **Laparoscopy:** The preferred route for **hemodynamically stable** patients.
- **Laparotomy:** Reserved for **hemodynamically unstable** patients or those with significant intraperitoneal bleeding.
- **Salpingostomy:** A 10–15 mm incision is made in the tube to remove the pregnancy, leaving the tube to heal. This is often the best choice for a viable ectopic pregnancy in the remaining tube if the other has already been removed.
- **Salpingectomy:** Complete removal of the fallopian tube, often used for ruptured pregnancies or to prevent recurrence in the tubal stump.
- **Inappropriate Options: "Milking" the tube** to remove the pregnancy is **NOT a recommended surgical option**.

Differential Diagnosis

Clinicians must distinguish ectopic pregnancy from other conditions with similar symptoms, such as:

- Threatened or incomplete abortion.
- Ruptured corpus luteal cyst.
- Adnexal torsion or degenerating leiomyoma.
- **Acute pelvic inflammatory disease (PID)** is considered the **least likely differential** to mimic the acute pain of a pregnancy-related problem like ectopic pregnancy in its early stages.

Gestational Trophoblastic Diseases

Classification and Pathogenesis

Gestational Trophoblastic Disease (GTD) represents a group of disorders ranging from **complete and partial molar pregnancies** to malignant conditions known as **Gestational Trophoblastic Neoplasia (GTN)**, which include invasive mole, choriocarcinoma, and the rare placental site trophoblastic tumour (PSTT).

- **Complete Molar Pregnancy (CM):**
    - **Genetics:** Most commonly **diploid (46, XX)** and entirely **androgenic** in origin (paternal).
    - **Origin:** 75–80% result from a single sperm duplicating within an 'empty' ovum; 20–25% result from dispermic fertilisation of an 'empty' ovum.
    - **Features: No fetal tissue** is present. It has a higher risk of malignant transformation (15–20%) compared to partial moles.
- **Partial Molar Pregnancy (PM):**
    - **Genetics:** Usually **triploid** (69, XXX or 69, XXY) with two paternal and one maternal haploid sets.
    - **Origin:** Almost always dispermic fertilisation of a normal ovum.
    - **Features:** Often contains **evidence of a fetus**, fetal red blood cells, or embryonic tissue. It demonstrates focal (rather than diffuse) trophoblastic hyperplasia.

Epidemiology and Risk Factors

- **Incidence:** In the UK, the incidence is **1/714 live births**. **Asian women** have a higher incidence than non-Asian women.
- **GTN after pregnancy:** The incidence of GTN after a live birth is roughly **1/50,000**.
- **Recurrence:** The risk of a repeat molar pregnancy in future gestations is increased **10-fold** (approximately 1/80).

Clinical Presentation

- **Classic Symptoms:** Irregular **vaginal bleeding** (the most common symptom), hyperemesis, excessive uterine enlargement (large-for-dates), and early failed pregnancy.

- **Other Features:** Pelvic pressure/pain (though less common than bleeding), **early-onset pre-eclampsia** (toxaemia before 24 weeks), and hyperthyroidism.
- **Rare/Severe:** Abdominal distension from **theca lutein cysts**, acute respiratory failure, or neurological symptoms (seizures) due to metastatic disease.
- **Metastasis:** The most common sites for metastases are the **lungs and vagina**.

Diagnosis

- **Ultrasound (US):** Useful for early diagnosis; often shows a **"snowstorm appearance"** or cystic spaces in the placenta. For partial moles, a ratio of transverse to anteroposterior dimension of the gestation sac > 1.5 is a marker.
- **hCG Levels:** Extremely high levels (e.g., >100,000 or >230,000 IU/L) are suggestive. However, **high hCG is more associated with complete** than partial moles.
- **Definitive Diagnosis:** Histological examination of the **products of conception** is the gold standard.
- **Note:** If a patient has persistent bleeding after any pregnancy event (miscarriage, termination, or birth), a **urine pregnancy test** must be performed to exclude GTN.

Management

- **Primary Treatment: Suction curettage** is the mainstay for both complete and partial moles.
    - **Medical evacuation** is only used for partial moles where fetal parts are too large for suction.
    - **Anti-D prophylaxis:** Required for partial moles but **not required for complete moles** (due to lack of fetal red cells/D antigen), though it may be given if the diagnosis is uncertain.
- **Surgical Safety:**
    - Avoid oxytocics before evacuation as they increase uterine sensitivity and the risk of **tumour embolisation**.
    - Cervical ripening is safe immediately prior to surgery but prolonged preparation should be avoided.
    - **Routine second evacuations are not recommended** but may be considered in selected cases if hCG < 5000 units/litre.

- **Pre-Surgical Investigations:** Include Chest X-ray, LFTs, and coagulation profile. (A pregnancy test is already done for diagnosis).

Follow-up and Registration

- **Registration:** In the UK, all cases (CM, PM, twin molar pregnancies, choriocarcinoma, PSTT, and atypical placental site nodules) must be registered at centres like **Sheffield, London (Charing Cross), or Dundee**.
- **hCG Monitoring:** Serial blood or urine tests every 2 weeks until normal.
    - If hCG normalises within 56 days: Follow-up for **6 months from the date of evacuation**.
    - If hCG takes >56 days to normalise: Follow-up for **6 months from the date of normalisation**.
- **Future Pregnancies:** Women must notify screening centres at the end of *any* future pregnancy; hCG is measured **6–8 weeks post-partum** to ensure no recurrence.

Gestational Trophoblastic Neoplasia (GTN) & Chemotherapy

- **Risk of Needing Chemo: 15% after a complete mole** and **0.5% after a partial mole**.
- **Indications for Chemo:** Includes a plateau in hCG for 3 consecutive weeks, rising hCG, or histological diagnosis of choriocarcinoma.
- **FIGO 2000 Scoring:**
    - **Low Risk (Score ≤ 6):** Treated with **single-agent methotrexate** (alternated with folinic acid). Cure rate is almost 100%.
    - **High Risk (Score ≥ 7):** Treated with **multi-agent chemotherapy** (EMA-CO: etoposide, methotrexate, dactinomycin, cyclophosphamide, vincristine). Cure rate is 95%.
- **Special Case: PSTT** is less sensitive to chemotherapy and is often treated with **surgery**.

Prognosis and Long-term Outcomes

- **Poor Prognosis Factors:** Age > 39, hCG > 100,000, long interval since index pregnancy, occurrence after a **normal term delivery** (worse than after miscarriage), and previous failed chemotherapy.
- **Late Effects of Chemo:**
    - **Early Menopause:** Advanced by 1 year (single-agent) or 3 years (multi-agent).

    - **Secondary Cancers:** Increased risk of AML, colon cancer, melanoma, and breast cancer if multi-agent treatment (with etoposide) lasts > 6 months.

Future Pregnancy and Contraception

- **Wait Time:** Conceptions should be avoided for **6–12 months** of documented hCG remission. If chemotherapy was received, wait **1 year**.
- **Contraception:**
    - **Barrier methods** until hCG reverts to normal.
    - **COCP** is safe once hCG is normal. (Early use has a very low risk of increasing GTN).
    - **IUCDs** should be avoided until hCG is normal to prevent **uterine perforation**.
- **HRT:** Safe to use once hCG is normal.
- **Future Risks:** No increased risk of congenital malformations or placenta accreta, but there is a slightly higher risk of **spontaneous abortion** and **postpartum haemorrhage**.

Summary Table: Complete vs. Partial Mole

Feature	Complete Mole (CM)	Partial Mole (PM)
Karyotype	46, XX (Paternal)	69, XXX or XXY
Fetal Tissue	Absent	Often Present
Trophoblast	Diffuse Hyperplasia	Focal Hyperplasia
Uterine Size	Often Large for Dates	Often Small/Normal for Dates
hCG Levels	Markedly Elevated	Less Elevated
Theca Lutein Cysts	Common	Rare
Malignant Risk	15–20%	< 1%

Multiple Pregnancies

Multiple pregnancy, defined as a gestation with more than one fetus, is a high-risk obstetric condition that requires careful classification, monitoring, and management to mitigate significantly increased maternal and fetal risks.

Classification and Zygosity

Multiple pregnancies are primarily classified by **zygosity** (the number of fertilized ova) and **chorionicity/amnionicity** (the number of placentas and amniotic sacs).

- **Dizygotic (DZ) Twins:** These result from the fertilization of **two separate ova** by two separate sperm. They are always **dichorionic-diamniotic (DCDA)**, meaning they have two placentas (which may fuse but lack vascular connections) and two sacs. DZ twins can be of different genders.
- **Monozygotic (MZ) Twins:** These result from a **single fertilized ovum** that subsequently divides. Placentation in MZ twins is strictly determined by the **timing of the cleavage**:
    - **0–3 days (Morula stage):** Results in **DCDA** twins (approx. 29% of MZ twins).
    - **4–8 days (Blastocyst stage):** Results in **monochorionic-diamniotic (MCDA)** twins (approx. 70%).
    - **9–12 days (Implanted blastocyst):** Results in **monochorionic-monoamniotic (MCMA)** twins (approx. 1%).
    - **13 days or later (Embryonic disc):** Results in **conjoined twins**, an extremely rare condition.

Key Exam Note: While all dizygotic twins are DCDA, **not all DCDA twins are dizygotic**, as early-cleaving monozygotic twins also present this way.

Epidemiology and Risk Factors

The incidence of multiple gestations is increasing, now accounting for over 3% of live births. While the rate of MZ twins remains stable worldwide (3–5 per 1000), DZ twin rates vary significantly based on:

- **Fertility Treatments:** IVF and ovulation induction are major drivers; up to 24% of IVF procedures result in multiple births.
- **Maternal Demographics:** Increased maternal age (due to higher FSH levels), high parity, high BMI, and maternal height (taller women) are associated with higher DZ rates.

- **Geography:** Incidence ranges from 1.3/1000 in Japan to 50/1000 in Nigeria.
- **Genetics:** DZ twins have a familial trait, whereas MZ twins do not run in families and are not influenced by paternal factors.

Clinical Diagnosis and Ultrasound Monitoring

A "large for dates" uterus may suggest multiple gestation, but must be differentiated from incorrect dating, macrosomia, polyhydramnios, fibroids, obesity, or molar pregnancy.

Ultrasound is the definitive diagnostic tool:

- **First Trimester (10–13 weeks):** The most critical step after diagnosis is determining **chorionicity**, as it is the main determinant of outcome.
    - **Lambda (λ) or Twin-Peak Sign:** Indicates a **DCDA** pregnancy, where a triangular projection of placental tissue extends into the intertwin membrane.
    - **T-sign:** Indicates a **monochorionic** pregnancy, characterized by a thin membrane meeting the placenta at a 90-degree angle without intervening chorion.
- **Fetal Gender:** Identifying discordant (different) gender confirms a **dichorionic** pregnancy with 100% predictive value.
- **Anatomy Survey (16–24 weeks):** Necessary as congenital anomalies are 3–5 times more common in multiple gestations.

Complications of Multiple Pregnancy

Maternal Complications

Multiple gestations carry a 2.5 times higher risk of maternal mortality compared to singletons.

- **Hypertensive Disorders:** Preeclampsia (26% in twins, risk increases 9-fold in triplets) and HELLP syndrome.
- **Other Risks:** Anemia (24%), gestational diabetes (14%), placental abruption/previa, acute fatty liver, and postpartum hemorrhage (PPH).

Fetal and Neonatal Complications

- **Prematurity:** Occurs in >50% of twins and 75% of triplets. The mean gestational age for twins is approximately 35 weeks.
- **Growth Restriction:** Significant growth discordance (>20%) is a predictor of adverse outcomes.

- **Stillbirth and Morbidity:** Increased risk of neurodevelopmental disabilities and chronic lung disease.

Specific Monochorionic Complications

Monochorionic twins face unique risks due to placental vascular communications:

1. **Twin-to-Twin Transfusion Syndrome (TTTS):** Occurs in 15% of MCDA twins due to an imbalance in blood flow.
    - **Donor Twin:** Hypoperfused, anemic, growth-restricted, and has **oligohydramnios**.
    - **Recipient Twin:** Hyperperfused, hypertensive, has a large bladder, and has **polyhydramnios**.
    - **Management: Fetoscopic Laser Photocoagulation (FLP)** is the best treatment (16–26 weeks) to block communicating vessels.
2. **Monoamniotic Complications:** High risk of **cord entanglement**, which can cause sudden fetal loss.
3. **Intrauterine Demise of One Fetus:** In monochorionic pregnancies, the death of one twin leads to a 15% risk of co-twin death and significant neurologic morbidity in the survivor due to acute blood loss into the dead twin. This risk is much lower in dichorionic gestations.

Management and Delivery

- **Antenatal Care:** Serial ultrasounds monitor growth and cervical length. A cervical length <20mm between 20–24 weeks strongly predicts preterm birth. A single course of **corticosteroids** is recommended between 24 and 34 weeks if preterm delivery is imminent.
- **Intrapartum Care:** Proper equipment, an experienced team, and two CTG monitors are essential. Epidural anesthesia is recommended.
- **Timing of Delivery:**
    - **DCDA:** 37–38 weeks.
    - **MCDA:** 36–37 weeks.
    - **MCMA:** 32–34 weeks (delivered by **Cesarean section** to avoid cord accidents).

- **Mode of Delivery:** Vaginal delivery is possible if the first twin is in a **cephalic presentation**. Cesarean section is required if the first twin is non-cephalic, for higher-order multiples (triplets+), or for conjoined twins.

Multifetal Pregnancy Reduction (MFPR)

MFPR is a procedure performed between 10 and 13 weeks to reduce the number of fetuses and improve the survival chances of the remaining ones.

- **Technique:** Potassium chloride (KCl) is usually injected into the fetal thorax under ultrasound guidance.
- **Caution:** KCl must **not** be used in monochorionic pairs due to the risk of co-fetal demise through vascular connections; instead, vascular occlusion (e.g., radiofrequency ablation) is used.

Rh Isoimmunization

1. Overview of Rh Isoimmunisation

Rh isoimmunisation occurs when a **Rhesus (Rh)-D negative woman** is exposed to Rh(D) positive red blood cells, leading to the development of **anti-D antibodies**. These IgG antibodies can cross the placenta, opsonising fetal RBCs for destruction in the fetal spleen, resulting in **Hemolytic Disease of the Fetus and Newborn (HDFN)**.

- **Prevalence:** Varies by population; for example, 15% in Caucasians, 9.8% in Jordan, and as low as 0.3% in China and Thailand.
- **Zygosity:** Roughly 40% of Rh-positive individuals are homozygous (DD), while the rest are heterozygous (Dd).
- **Common Causes:** Transplacental fetomaternal hemorrhage (FMH) is the primary cause. Other causes include contaminated needle injections, inadvertent blood transfusions, or stem cell transplants.

2. Pathogenesis and Fetal Risks

The Rh(D) antigen is expressed as early as **30 days of gestation**. Maternal sensitization can occur during any pregnancy event where fetal and maternal blood mix.

Findings Suggestive of Erythroblastosis Fetalis (Fetal Anemia): Severe anemia can lead to **Hydrops Fetalis**, defined by two or more of the following findings on ultrasound:

- Fetal ascites
- Placental thickening
- Body wall edema
- Pleural or pericardial effusion
- *Note: Small chest size is **not** a typical finding of erythroblastosis fetalis.*

3. Diagnosis and Monitoring

Diagnosis is confirmed by detecting anti-Rh(D) antibodies in maternal serum via the **Indirect Coombs Test (ICT)**.

Methods to Assess Fetal Risk of Anemia

Method	Description/Notes
Serial Maternal Titers	A critical value of **15 IU/mL** (or a low titer in some contexts) warrants further investigation.
MCA-PSV Ultrasound	Ultrasound of the fetal **middle cerebral artery peak systolic velocity** is a primary method for monitoring anemia.
Fetal Blood Sampling	Provides a definitive diagnosis but is not performed routinely due to risks.
Amniotic Fluid Bilirubin	Historically used but now considered largely obsolete.
Methods NOT Used	**Umbilical artery pulsatility index (PI)** does NOT assess the risk of anemia.

4. Anti-D Prophylaxis (Prevention)

Anti-D immunoglobulin is derived from the plasma of alloimmunised donors and is highly effective at preventing sensitization.

Dosage and Neutralization Power

- **300 micrograms:** Neutralizes **15 mL** of Rh-positive red cells or **30 mL** of fetal whole blood.
- **50 micrograms:** Neutralizes **2.5 mL** of Rh-positive red cells or **5 mL** of fetal whole blood.
- **Elimination Power:** 500 IU (100 micrograms) does **not** eliminate 8 mL of blood; it neutralizes approximately 5 mL of whole blood.

Indications and Contraindications for Anti-D

Indicated (Give Anti-D)	NOT Indicated (Do NOT Give)
Miscarriage (spontaneous/induced)	**Already sensitized mother** (ICT positive)
Ectopic pregnancy (including methotrexate treatment)	**Complete molar pregnancy** (no fetal RBCs)

Abdominal trauma at any stage	Spontaneous complete miscarriage at **11 weeks**
Invasive procedures (Amniocentesis/CVS)	Biological father is known to be **Rh-D negative**
External cephalic version	Painless vaginal spotting **< 8 weeks**
Fetal death/demise in 2nd or 3rd trimester	Fetus confirmed Rh-negative via cfDNA or sampling

5. Management of Affected Pregnancies

If a mother is already sensitized, management focuses on the fetus:

- **Pre-term/Severe Anemia:** Treated with **Intrauterine Fetal Transfusions** (intravascular or intraperitoneal).
- **Hydrops at 36 Weeks:** The most appropriate treatment is **delivery by Cesarean section** rather than observation or further transfusions.
- **Investigational Treatments:** Maternal plasmapheresis and intravenous immune globulin therapy are being explored to decrease disease severity.

6. Post-Delivery Testing for Fetomaternal Hemorrhage (FMH)

All Rh-negative women should be tested at delivery to ensure the Anti-D dose is sufficient for the volume of hemorrhage.

1. **First Screening Test:** The **Rosette Test** is a qualitative, sensitive test performed first.
2. **Follow-up (if Rosette is positive):** Quantitative tests like the **Kleihauer-Betke acid elution test** or **flow cytometry** are used to calculate the exact dose of Anti-D needed.
3. **Routine Postpartum Care:** Anti-D should be administered within **72 hours** of delivery if the baby is confirmed Rh-positive.

Gestational DM

1. Classification of Diabetes in Pregnancy

Diabetes encountered during pregnancy is categorised into several types:

- **Type 1 and Type 2 Diabetes:** Pre-existing (pregestational) conditions.
- **Gestational Diabetes (GDM):** Diabetes first diagnosed during pregnancy, affecting 2–3% of pregnancies.
- **Monogenetic Diabetes (MODY):** An autosomal dominant single-gene mutation affecting pancreatic B-cell secretion; it is not associated with obesity.
- **Mitochondrial Diabetes:** A mutation in mitochondrial DNA associated with sensorineural deafness and a tendency for strokes; it usually develops in the mid-thirties without obesity.
- **Secondary Diabetes:** Associated with other conditions like pancreatitis, cystic fibrosis, or use of drugs like glucocorticoids.

2. Pathophysiology: Why Pregnancy is Diabetogenic

Pregnancy naturally increases insulin resistance to ensure the fetus receives adequate glucose.

- **Key Hormones:** The main factor responsible for this state is **Human Placental Lactogen (HPL)**. Other contributing factors include estrogen, progesterone, cortisol, and the degradation of insulin by the placenta.
- **Insulin Resistance:** Resistance increases as the pregnancy progresses. While insulin secretion may double to compensate, if the body cannot meet this demand, GDM develops.

3. Screening and Diagnosis

Universal screening and specific diagnostic tests are crucial for identifying GDM.

- **Timing:** The standard window for screening for GDM is **24–28 weeks** of gestation.
- **Indications for Early Testing (GTT):** Testing may be done at "booking" (first visit) if risk factors are present, such as a BMI >30, previous baby weighing ≥4.5kg, family history of diabetes (1st degree), previous GDM, or history of congenital anomalies/stillbirth.
- **Diagnostic Criteria:**

- **O'Sullivan Test:** 50g non-fasting; if 1-hour blood glucose is >7.8 mmol/L, a full Glucose Tolerance Test (GTT) is required.
- **75g OGTT (24-32 wks):** Fasting 5.1 mmol/L; 1 hour 10 mmol/L; 2 hours 8.5 mmol/L.
- **100g 3-hour GTT:** Fasting <5.3 mmol/L (95 mg/dl); 1 hour 10 mmol/L (180 mg/dl); 2 hours 8.6 mmol/L (155 mg/dl); 3 hours 7.8 mmol/L (140 mg/dl).

4. Impact of Diabetes on the Mother and Fetus

Diabetes significantly increases the risk of complications throughout the pregnancy.

Maternal Complications

- **Infections:** Increased risk of UTIs, asymptomatic bacteriuria, and recurrent monilial vulvovaginitis due to high glucose in vaginal epithelium.
- **Obstetric Risks:** Increased incidence of **Preeclampsia (PET)**, **Polyhydramnios** (seen in 25% of cases, often due to fetal polyuria), and higher rates of Caesarean sections.
- **Diabetes Progression:** Diabetic retinopathy and retinal detachment may occur more frequently.

Fetal and Neonatal Complications

- **First Trimester:** Increased risk of miscarriage and congenital defects. The **most specific anomaly** associated with overt maternal diabetes is **Caudal Regression Syndrome**.
- **Second/Third Trimester:** Fetal hyperinsulinemia (triggered by maternal hyperglycemia) leads to accelerated growth (macrosomia), fat deposition, and **delayed lung maturation**.
- **Neonatal Risks:** At birth, infants are at risk for **Hypoglycemia**, **Polycythemia**, **Neonatal Jaundice**, and **Respiratory Distress Syndrome (RDS)**. They are also at risk for birth trauma such as **Shoulder Dystocia**.
- **Long-term Risks:** Intrauterine exposure predisposes the child to obesity and Type 2 diabetes in adulthood.

5. Clinical Management

Preconception Care

- Women should achieve a normal **HbA1c** before conception.
- **Folic acid (5 mg/day)** should be started before conception and continued for 12 weeks.
- Medications should be modified to pregnancy-safe alternatives **before** the pregnancy begins.
- Existing retinopathy should be treated before conception.

Antenatal Care

- **First Trimester:** Focus on dating scans and assessment of glycemic control (Target: Fasting 6 mmol/L, 1-hour postprandial 7.8 mmol/L). **Glycosylated Hb (HbA1c)** is the best method to assess potential fetal damage during this period.
- **Second/Third Trimester:** Monitoring fetal growth via ultrasound and screening for anomalies. Antenatal testing (e.g., at 34 weeks) specifically aims to prevent **stillbirth**.
- **GDM Initial Management:** Once diagnosed, the first step is to check blood sugar **four times daily**.

Delivery and Postpartum

- **Timing:** Delivery is ideally at **40 weeks** if well-controlled, but may be moved to **38 weeks** if there is a poor obstetric history.
- **Mode of Delivery:** C-section is not universally indicated but is recommended if **macrosomia** is suspected in a GDM patient to prevent shoulder dystocia.
- **Intrapartum:** Use insulin infusion and 5% dextrose with hourly blood glucose monitoring.
- **Postpartum: Insulin requirements fall significantly** after delivery; the mother should return to her pre-pregnancy dose.
- **Breastfeeding:** Encouraged, but requires an additional 50g of carbohydrates per day; oral hypoglycemics are generally contraindicated during this period.

6. Important Past Paper Highlights (For Quick Review)

- **Insulin and Placenta:** Maternal insulin does **not** cross the placenta; fetal hyperinsulinemia is caused by maternal glucose crossing the placenta.
- **Polyhydramnios:** This is associated with GDM, particularly if poorly controlled.
- **Stillbirth:** A primary goal of late-pregnancy testing is to prevent stillbirth.
- **Corticosteroids:** Drugs like **Dexamethasone** (given for preterm labour) can cause a significant spike in blood sugar levels.
- **Oligohydramnios:** This is **not** typically a complication of diabetes; rather, polyhydramnios is the concern.
- **Neonatal Exclusions:** Infants of diabetic mothers are **not** typically at higher risk for Down's Syndrome, neonatal anemia, hypercalcemia, or hypermagnesemia.

Hypertensive Disorders

1. Definitions and Classification

- **Pre-eclampsia (PET)** is defined as **hypertension (≥140/90 mmHg)** on two occasions at least 4 hours apart, combined with **proteinuria (≥300 mg in 24 hours)**, arising **after 20 weeks** of gestation in a previously normotensive woman.
- **Chronic Hypertension** is distinguished by the presence of high blood pressure **before 20 weeks** of gestation.
- **Gestational Hypertension** occurs after 20 weeks but lacks the proteinuria associated with PET, though 50% of these cases do not necessarily progress to pre-eclampsia.
- **HELLP Syndrome** is a severe variant of PET involving **H**aemolysis, **E**levated **L**iver enzymes, and **L**ow **P**latelets.

2. Pathophysiology: The Placental Origin

- In a normal pregnancy, **trophoblast cells** invade the maternal spiral arterioles by 20 weeks, converting them into large, **low-resistance vessels** to increase blood flow to the placenta.
- In pre-eclampsia, there is a **failure of trophoblast invasion**, causing spiral arteries to remain narrow and high-resistance, leading to **placental under-perfusion**.
- This under-perfusion releases factors that target the **maternal vascular endothelium**, making PET a truly **multisystem disease**.
- A characteristic renal lesion called **'glomeruloendotheliosis'** occurs, which reduces glomerular filtration and causes the selective loss of proteins like albumin.

3. Epidemiology and Risk Factors

- **Primary Risk Factors:** PET is most common in **primigravid women** (first pregnancy). Other risks include a family history (3-4 fold increase), BMI ≥ 35, age ≥ 40, and pre-existing conditions like **diabetes**, renal disease, or antiphospholipid antibodies.
- **Historical Risk:** A woman with a history of PET has a **20% recurrence risk**, which increases if the previous episode was severe or occurred at an early gestation.
- **Past Paper Insights:** According to the sources, **smoking** and the **sex of the baby** are **NOT** considered risk factors for pre-eclampsia. Furthermore, a history of a macrosomic baby is not a recognized risk factor.

4. Clinical Presentation and Warning Signs

- Many women are **asymptomatic**, making regular screening vital.
- **Classic Symptoms of Severity:** These include **frontal headaches**, **visual disturbances** (blurred vision), and **epigastric pain**.
- **Physical Signs:** Rapidly progressive oedema of the face and hands, **epigastric tenderness** (suggesting liver involvement), and neurological signs like **hyperreflexia or clonus** are worrying indicators.
- **Blurred vision** is specifically attributed to the effects of the disease on the **brain** (hypertensive encephalopathy) rather than the eyes alone.

5. Management and Pharmacology

The primary goals are to prevent maternal cerebrovascular accidents (strokes) and seizures while timing the delivery to avoid complications.

- **Antihypertensive Agents:**
    - **Methyldopa:** Centrally acting with a long safety record; however, it takes 24 hours to act and can cause sedation.
    - **Labetalol:** An alpha and beta-blocker that can be given orally or intravenously.
    - **Nifedipine:** A **calcium-channel blocker** with a rapid onset.
    - **Hydralazine:** Often used as an intravenous infusion for acute management.
    - **Contraindications: Enalapril** (and other ACE inhibitors) must be switched (e.g., to methyldopa) if a patient becomes pregnant.
- **Seizure Prophylaxis (Magnesium Sulfate - MgSO4):**
    - MgSO4 is the **drug of choice** to prevent and treat eclamptic seizures.
    - It reduces the risk of eclampsia by half and decreases maternal death.
    - **Toxicity Monitoring:** It is excreted by the kidneys (not the liver), so **urine output**, **respiratory rate**, and **deep tendon reflexes** (patellar reflex) must be monitored.
    - Loss of deep tendon reflexes is an **early sign of toxicity**, while respiratory depression indicates excessive dosage.

6. Complications and Delivery

- **Leading Causes of Death: Intracranial haemorrhage** is the most common cause of death in PET, while **hepatic haemorrhage** is frequently associated with death in HELLP syndrome.
- **Indications for Delivery:** Delivery is the only "cure." Immediate delivery is indicated for **uncontrolled hypertension**, low platelet counts (<50,000), persistent headache, or **fetal distress**.
- **Mode of Delivery:** For pregnancies less than 34 weeks, a **Caesarean section** is often preferred. **Ergometrine** is strictly contraindicated for third-stage management in these patients.
- **Prevention: Low-dose aspirin (75 mg)** is recommended for high-risk women to modestly reduce PET risk. Calcium supplementation may help those with low dietary intake, but Vitamins C and E have been shown not to lower the risk.

Anemia in Pregnancy & Hyperemesis Gravidarum

I. Anemia in Pregnancy

Definition and Thresholds Anemia is defined as a Hemoglobin (Hb) concentration below specific cut-off points, which vary by physiological state.

- **Non-pregnant women:** < 12 g/dl.
- **1st Trimester:** < 11 g/dl.
- **2nd & 3rd Trimesters:** < 10.5 g/dl (Note: Past paper identifies < 11 g/dl as the 3rd-trimester threshold).
- **Postpartum:** < 10 g/dl.

Physiological Changes

- **Haemodilution:** Plasma volume expands by **50%**, starting in the 1st trimester and plateauing in the 3rd. This expansion exceeds the increase in red blood cell production, causing a natural fall in Hb.
- **Iron Requirements:** Total iron requirement during pregnancy is approximately **1000 mg**. Daily requirements increase from 1–2 mg to **6 mg per day** (Recommended Dietary Allowance is 27 mg).

1. Iron Deficiency Anemia (IDA)

IDA is the **most common hematological problem** in pregnancy.

- **Causes:** Low dietary intake, impaired absorption, or loss (e.g., multiple pregnancy, malaria, or postpartum hemorrhage).
- **Diagnosis:**
    - **Serum Ferritin:** The **most sensitive test** for detecting iron depletion; a level **< 30 µg/L** is diagnostic.
    - **CBC Indices:** Characterised as **microcytic hypochromic**. **MCV is the first index to become abnormal**.
    - **Past Paper Note:** A high level of serum ferritin does *not* confirm IDA; it is the low level that does.
- **Maternal & Fetal Effects:** Fatigue (most common symptom), dizziness, pica, low birth weight, and preterm birth.

- **Management:**
    - **Diet:** Absorption is enhanced by Vitamin C (ascorbic acid) and inhibited by tea, coffee, chocolate (tannins), and phytic acid (bread).
    - **Oral Iron:** The first-line treatment. Supplements (e.g., Ferrous Sulphate) should be taken on an empty stomach.
    - **IV Iron:** Considered after 34 weeks if Hb < 100 g/l or if oral iron is not tolerated. It carries a risk of allergic reactions.
    - **Blood Transfusion:** Necessary if Hb < 8 g/dl in late pregnancy.

2. Folate Deficiency (Megaloblastic Anemia)

This is the second most common cause of anemia in pregnancy.

- **Key Indicator:** The earliest morphological evidence is the **hypersegmentation of neutrophils**.
- **Prevention:** Standard dose is 400 µg/day. A **high dose (5 mg/day)** is required for women with a previous child with a neural tube defect, BMI > 30, diabetes, or those on anti-epileptic drugs.

II. Hemoglobinopathies

These are autosomal recessive genetic disorders diagnosed via **Hb electrophoresis**.

1. Thalassemia

- **Nature:** The **most common genetic blood disorder**, involving reduced production of normal Hb.
- **Diagnosis:** HbA2 > 3%.
- **Management:** Screen the partner; if both are carriers, offer prenatal genetic diagnosis (PGD). Iron chelation (Desferrioxamine) is safe from 20 weeks.

2. Sickle Cell Anemia

- **Nature:** Result of an **amino acid substitution** (valine for glutamic acid) at position 6 of the β-chain.
- **Complications:** Red cells "sickle" in deoxygenated states, leading to painful, hemolytic, or sickle cell crises, especially in the last trimester.

- **Maternal Risks:** Acute chest syndrome, pre-eclampsia, and venous thromboembolism (VTE).
- **Management:** Multidisciplinary team, serial growth scans from 20 weeks, folate (5 mg/day), and postnatal thrombo-prophylaxis (LMWH).

III. Hyperemesis Gravidarum (HG)

HG is severe nausea and vomiting affecting 0.3–3% of pregnancies, distinct from common morning sickness.

- **Clinical Features:** Excessive vomiting leading to **dehydration**, **weight loss (>5% of pre-pregnancy weight)**, and **ketonuria**.
- **Key Diagnostic Signs:** Early signs include **ketonuria**. Other signs are an anxious look, acetone breath, tachycardia (not slow pulse), and oliguria.
- **Risk Factors:** Multiple pregnancy and molar pregnancy (gestational trophoblastic disease). It is **not** typically associated with grand multiparity.
- **Investigations:**
    - **CBC:** Raised Hematocrit (HCT) due to dehydration.
    - **Electrolytes:** Hyponatremia, **hypokalemia**, and metabolic alkalosis.
    - **Ultrasound:** Essential to rule out molar or multiple pregnancies.
- **Complications:**
    - **Wernicke's Encephalopathy:** Caused by **Vitamin B1 (thiamine) deficiency**; symptoms include confusion, ataxia, and nystagmus.
    - **Central Pontine Myelinolysis:** Caused by rapid correction of sodium.
    - **Mallory-Weiss tears**.
- **Management:**
    - **In-patient:** NPO (nothing by mouth) and IV fluids (Normal Saline or Ringer's Lactate). **Dextrose-containing fluids are inappropriate** as they can precipitate Wernicke's.
    - **Pharmacotherapy:** Anti-emetics (Promethazine, Metoclopramide) and potentially corticosteroids (e.g., prednisolone) for severe cases.
    - **Thiamine supplementation** is vital.

IV. Memory Aids & Exam Essentials

- **Most Common Hematological Disorder:** Iron Deficiency Anemia.
- **HG vs. Morning Sickness:** HG requires >5% weight loss and ketonuria.
- **Sickle Cell vs. Thalassemia:** Sickle cell is an *amino acid substitution*; Thalassemia is *reduced production*.
- **IV Fluid Rule:** Use Saline/Ringer's for HG; avoid Dextrose until thiamine is replaced.
- **IDA Screening:** Serum ferritin is the gold standard.
- **Constipation Management:** Increase fiber, fluids, and exercise; do **not** avoid fruits and vegetables.

Labor & Intrapartum Materanl Monitoring

1. Definitions and Diagnosis of Labor

- **True Labor Pain:** Defined as **regular, rhythmic, progressive, and painful** uterine contractions. These result in progressive cervical dilation and effacement (thinning) and the eventual expulsion of the fetus. True labor is **not relieved** by rest or hydration.
- **False Labor (Braxton-Hicks Contractions):** These are **irregular**, infrequent, unpredictable, and **non-rhythmic**. They are more uncomfortable than painful and do not increase in frequency or intensity. They can start as early as the 20th week.
    - **Exam Alert:** Past papers clarify that Braxton-Hicks do **not** cause prolongation of the second stage of labor. They often resolve with ambulation (walking).

2. Anatomy of the Maternal Pelvis

The pelvis is divided into the inlet (brim), midpelvis (midcavity), and outlet.

- **Pelvic Shapes:**
    - **Gynecoid:** The **most common** (50%) and **most favourable** for labor.
    - **Android:** Similar to the **male pelvis**; predisposes to **failure of rotation** and deep transverse arrest. It is the most common cause of **Occipito-posterior (OP) position**.
    - **Anthropoid:** Encourages an **Occipito-posterior (OP)** position.
    - **Platypelloid:** Associated with an increased risk of **obstructed labor** due to failure to engage.
- **Key Landmarks and Measurements:**
    - **Inlet:** Wider transversely than anterior-posteriorly (AP diameter is 11 cm).
    - **Midpelvis:** Where the fetal head typically rotates from transverse to AP position.
    - **Ischial Spines:** The landmark for **0 station**. They are the narrowest transverse diameter of the maternal pelvis.
    - **Pudendal Nerve:** Passes **behind and below** the ischial spine; this is the site for a pudendal block (sensory to the perineum).
    - **Pelvic Outlet:** The plane of **least pelvic dimensions**.

3. Anatomy of the Fetal Skull

- **Structure:** The vault (compressible) and sutures (not ossified at term) allow for **moulding** to accommodate the maternal pelvis.
- **Key Diameters (High Exam Frequency):**
    - **Vertex Presentation:** Suboccipito-bregmatic (**9.5 cm**) – the longitudinal diameter in a well-flexed head.
    - **Face Presentation:** Submento-bregmatic (**9.5 cm**) – measured from below the chin to the anterior fontanelle.
    - **Occipito-posterior (OP) Position:** Occipito-frontal (**11.5 cm**).
    - **Brow Presentation:** Mento-vertical (**13-14 cm**) – this diameter is normally **too large** to pass through the maternal pelvis.
- **Fontanelles:** The **Anterior fontanelle** (Bregma) is diamond-shaped; the **Posterior fontanelle** is triangular and indicates the occiput.

4. Fetal Lie, Presentation, and Position

- **Lie:** The relationship between the longitudinal axis of the fetus and the mother (e.g., **Longitudinal**, Transverse, Oblique).
- **Presentation:** The part of the fetus entering the pelvis first (e.g., **Vertex**, Face, Brow, Breech, Shoulder).
    - **Attitude:** The relation of fetal parts to one another (e.g., **Flexion** is normal; Face/Brow are abnormal attitudes).
- **Denominator:** The fixed point on the presenting part used to describe position.
    - **Vertex:** Occiput.
    - **Face:** Mentum (**Chin**).
    - **Breech: Sacrum**.
    - **Transverse Lie:** Shoulder.
- **Position:** The orientation of the denominator to the maternal pelvis (e.g., Occipito-anterior is ideal).
- **Station:** Measured in cm relative to the **ischial spines** (0 station).

- **Engagement:** Occurs when the widest part of the presenting part passes the pelvic inlet.
    - Abdominally, the head is engaged if **2/5 or less** is palpable.
    - **Non-engagement** in primigravidae is most commonly caused by **Cephalopelvic disproportion (CPD)**.

5. The Mechanism of Labor (Cardinal Movements)

The fetal head enters the inlet transversely and rotates in the midpelvis. **The correct sequence is:**

1. **Descent** (occurs throughout).
2. **Flexion**.
3. **Internal Rotation**.
4. **Extension** (leads to delivery of the head).
5. **Restitution** (head rotates to become perpendicular to the shoulders).
6. **External Rotation**.
7. **Expulsion**.

6. The Four Stages of Labor

- **First Stage:** From the onset of labor to full cervical dilation (10 cm).
    - **Latent Phase:** Dilation up to 4 cm (or 5-6 cm depending on guidelines). Averages 8 hours in nulliparas and 5 hours in multiparas.
    - **Active Phase:** Regular contractions and progressive dilation beyond 4-6 cm. Typically dilates at **1 cm/hour**.
- **Second Stage:** From full dilation to delivery of the fetus.
    - **Duration (Nulliparous):** 2 hours without epidural, 3 hours with.
    - **Duration (Multiparous):** 1 hour without epidural, 2 hours with.

- **Third Stage:** From delivery of the baby to delivery of the placenta.
    - **Signs of Placental Separation:** Cord lengthening, gush of blood, rising fundus, and firm globular uterus.
    - **Active Management:** Includes 10 IU oxytocin, **delayed cord clamping (1-3 mins)**, and controlled cord traction.
- **Fourth Stage:** The first two hours postpartum; monitor vitals, bleeding, and uterine firmness.

7. Cervical Assessment: The Bishop Score

Used to assess the "ripeness" of the cervix for induction.

- **Five Parameters: Dilation** (best predictor), **Effacement** (length), **Consistency**, **Position**, and **Station**.
- **Simplified Score:** Uses Dilation, Effacement, and Station (Range 0-9); a score **>5 is favourable**.

8. Induction and Augmentation of Labor

- **Induction:** Starting labor artificially (Indications: Post-term, Preeclampsia, Chorioamnionitis, IUFD).
    - **Contraindications:** Placenta previa, active genital herpes, transverse lie, or prior classical C-section.
- **Augmentation:** Speeding up slow labor.
- **Pharmacological Methods:**
    - **Oxytocin (Syntocinon):** Stimulates contractions. **Side effects:** Water intoxication (hyponatremia), uterine rupture, and fetal distress.
    - **Prostaglandins (PGE1, PGE2):** Used for cervical ripening. Side effects: Vomiting, diarrhea, and uterine hyperstimulation.
- **Mechanical Methods:** Amniotomy (Artificial Rupture of Membranes) and Intracervical balloons.
    - **Amniotomy Alert:** Contraindicated in **floating head** (risk of cord prolapse) or **active HIV/Hep B/Hep C**.

9. Monitoring and Abnormal Labor

- **The 3 "Ps": Power** (contractions), **Passage** (pelvis), and **Passenger** (fetus).
- **Partogram:** A tool to detect prolonged labor by recording dilation, descent, and vitals.
- **Dystocia (Obstructed Labor):**
    - **Causes:** CPD, malpresentation (Brow/Mentum posterior), or inefficient contractions.
    - **Bandl's Ring:** A pathological retraction ring between the upper and lower uterine segments; a sign of **obstructed labor**.
- **CPD Diagnosis:** In the absence of gross pelvic deformity, the best diagnosis is a **trial of labor**.

10. Special Presentations and Complications

- **Breech Presentation:**
    - **Types: Frank** (hips flexed, knees extended - most common for vaginal delivery), **Complete** (hips/knees flexed), **Footling** (one/both feet presenting).
    - **Risk Factors: Prematurity** (major factor), multiple gestation, uterine anomalies, and placenta previa.
    - **Management:** External Cephalic Version (ECV) can be offered at 36-37 weeks.
- **Face and Brow Presentations:**
    - **Face:** If **Mento-anterior**, it can be delivered vaginally. If **Mento-posterior**, it **requires a C-section**.
    - **Brow:** Usually requires a C-section because the presenting diameter is too large.
- **Cord Prolapse:** Risk factors include **multiparity**, **footling breech**, polyhydramnios, and a non-engaged presenting part. **Post-maturity** is **not** a risk factor.

11. Analgesia and Anaesthesia

- **Epidural:** Can prolong the second stage of labor.
- **Contraindications to Regional Anaesthesia: Hypovolemia** (greatest), skin infection at the site, or patient refusal. **Pseudotumor cerebri** is **not** an absolute contraindication.

Intrapartum Fetal Monitoring (CTG)

1. Core Modalities of Fetal Monitoring

Fetal wellbeing during labour is monitored through two primary methods:

- **Structured Intermittent Auscultation:** Periodic listening to the fetal heart rate.
- **Continuous Electronic Fetal Monitoring (EFM):** This is performed via a **Cardiotocograph (CTG)**, which can be **external** (transducers on the maternal abdomen) or **internal** (fetal scalp electrode).

2. Systematic Approach to Reading a CTG

To ensure a complete assessment, clinical practitioners should use the following checklist:

1. **Patient Details:** Confirm the name and date.
2. **Clinical Risk:** Define the specific risks associated with the pregnancy.
3. **Contractions:** Assess the frequency and duration.
4. **Baseline Fetal Heart Rate (FHR):** Normal range is **110–160 bpm**.
5. **Variability:** Normal beat-to-beat variation is **5–25 bpm**.
6. **Accelerations:** Check for their presence or absence.
7. **Decelerations:** Identify types (Early, Late, Variable, or Prolonged).
8. **Overall Impression:** Categorise as **reassuring, suspicious, or abnormal** and create a management plan.

3. Uterine Activity Assessment

The CTG monitors the strength, frequency, and duration of contractions.

- **Resting Tone:** The pressure between contractions (normal: 5–10 mmHg; up to 15 mmHg in labour). A resting tone **above 20 mmHg** is concerning as it decreases uterine perfusion.
- **Montevideo Units (MVUs):** A standardized way to quantify contraction strength calculated internally. **Above 200 MVUs** is generally required for adequate labour in the active phase.
- **Hyperstimulation:** Defined as **more than 5 contractions in 10 minutes**. This is the most frequent cause of a pathological CTG and can lead to fetal hypoxia and acidosis.

4. Fetal Heart Rate (FHR) Characteristics

Baseline Rate and Deviations

- **Normal:** 110–160 bpm.
- **Tachycardia (>160 bpm for >10 mins):** Causes include maternal infection/pyrexia, fetal hypoxia, prematurity, or drugs (e.g., beta-agonists like Ritodrine or Terbutaline).
- **Bradycardia (<110 bpm for >10 mins):** Causes include maternal hypotension, hypothermia, beta-blockers, or fetal metabolic acidosis. Values of 100–110 bpm can be normal in postdate pregnancies.

Variability

Variability represents the interaction between the fetal nervous system and heart, indicating an **intact neurological system**.

- **Normal:** 5–25 bpm.
- **Reduced/Minimal (<5 bpm):** The most common cause is **fetal sleep** (should not exceed 40 minutes). Other causes include fetal acidosis, tachycardia, prematurity, or drugs (opiates, benzodiazepines, magnesium sulphate).

Accelerations

Abrupt increases in FHR (≥15 bpm for 15s if ≥32 weeks; ≥10 bpm for 10s if <32 weeks). Their presence is **highly reassuring**.

5. Decelerations: Types and Causes

Decelerations are abrupt drops in FHR (>15 bpm for >15s).

- **Early (Type I):** Mirror the contraction (lowest point of FHR matches peak of contraction). Caused by **fetal head compression**; they are physiological and not a sign of distress.
- **Variable (Type III):** V-shaped with abrupt onset and recovery. Caused by **umbilical cord compression**.
    - **"Shoulders":** Small accelerations before and after a variable deceleration are reassuring signs of adaptation.
    - **Absence of shoulders** or "60 x 60" severity (dropping below 60 bpm for >60s) is worrying.

- **Late (Type II):** Start at the peak of a contraction and recover after it ends. Caused by **uteroplacental insufficiency** (e.g., maternal hypotension, IUGR, or placental abruption). Repetitive late decelerations often indicate **fetal metabolic acidosis**.
- **Prolonged:** Last 2–3 minutes (non-reassuring) or **longer than 3 minutes (abnormal)**. Causes include cord prolapse, uterine rupture, or maternal seizures.
- **Sinusoidal Pattern:** A smooth, wave-like pattern that is very concerning. It indicates **severe fetal hypoxia, severe anaemia, or haemorrhage**.

6. Clinical Categorisation and Management

Category	Definition	Management
Category I (Normal)	110–160 bpm baseline, moderate variability, no late/variable decelerations.	No specific management needed.
Category II (Intermediate)	Tachycardia, minimal variability, or recurrent decelerations with moderate variability.	Continued surveillance; correct underlying causes (e.g., maternal hypotension).
Category III (Abnormal)	Absent variability PLUS recurrent late/variable decelerations, bradycardia, or sinusoidal pattern.	**Urgent action:** Scalp blood sampling, or immediate delivery.

Conservative Management Strategies:

- Change maternal position to **left lateral**.
- Increase **IV hydration** and administer oxygen (though oxygen is the least useful).
- **Stop oxytocin** infusion and consider **tocolytic drugs** to halt contractions.
- Perform a **vaginal examination** to check for cord prolapse or rapid descent.

7. Secondary Tests (Fetal Scalp Sampling)

When a CTG is abnormal, **Fetal Scalp pH** may be assessed to reduce unnecessary caesarean sections.

- **Normal:** pH ≥ 7.25.
- **Borderline:** pH 7.21–7.24 (repeat in 30 minutes).
- **Abnormal:** pH ≤ 7.20 (expedite birth); **pH < 7.15 (urgent delivery)**.

Note: Fetal scalp sampling is contraindicated in cases of maternal infections (HIV, Hepatitis B/C) or suspected fetal blood disorders.

Operative Vaginal Delivery

1. Instruments and Classifications

A. Forceps

- **Types:**
    - **Wrigley's:** Used for **outlet/low cavity** deliveries.
    - **Simpson's and Neville Barnes:** Used for **mid-cavity, non-rotational** deliveries.
    - **Kielland's:** Specifically designed for **mid-cavity rotational** deliveries. They feature a **sliding lock** and a **cephalic curve**, but **lack a pelvic curve** to allow for rotation and correction of asynclitism.
    - **Piper's:** Specifically used for the **after-coming head in breech** delivery.
- **Anatomy:** Consists of blades (with cephalic and/or pelvic curves), shanks, a lock, and a handle.

B. Vacuum (Ventouse)

- **Types:** Includes metal cups (Malmstrom), **Silastic (soft) cups** (associated with lower scalp trauma but higher failure rates), and the **Kiwi OmniCup** (standard or OP versions).
- **Key Distinction:** Vacuum extraction is associated with **less maternal morbidity** but has a **higher failure rate** compared to forceps.

C. Classification of Station (Assisted Birth)

- **Outlet (+3):** Fetal scalp visible without separating labia; skull has reached the perineum; rotation ≤ 45°.
- **Low (+2):** Fetal skull at station +2 cm but not on the perineum.
- **Mid (0 to +1):** Head ≤ 1/5th palpable per abdomen; leading point of skull is at station 0 or +1 cm.

2. Indications and Contraindications

Indications for OVD:

1. **Fetal Compromise:** Suspected fetal distress in the second stage (e.g., pathological CTG, abnormal fetal blood sample).
2. **Maternal Exhaustion/Fatigue:** Inability to push effectively.
3. **Medical Conditions:** Where maternal pushing is contraindicated (e.g., **cardiac disease Class III/IV**, cerebral aneurysm, proliferative retinopathy, myasthenia gravis).
4. **Delayed Second Stage:** Inadequate progress (e.g., lack of progress for 3 hours in nulliparous women with regional anesthesia).

Contraindications:

- **Vacuum: Gestational age < 32 weeks** (due to risk of subgaleal/intracranial hemorrhage), **face or breech presentation**, and suspected fetal bleeding disorders.
- **Forceps: Brow presentation** and **hydrocephalus**.
- **General: Cephalopelvic disproportion (CPD)** or unknown fetal position.

3. Prerequisites for Procedure

Before attempting any OVD, the following must be confirmed (**mandatory**):

- **Abdominal/Vaginal Exam:** Fetal head must be **≤ 1/5th palpable** per abdomen (engaged), **vertex presentation**, cervix **fully dilated**, and membranes **ruptured**.
- **Preparation:** The **maternal bladder must be empty**, appropriate **analgesia** must be in place (regional block for mid-cavity), and informed **consent** obtained.
- **Safety:** The operator must be skilled, a **back-up plan** (emergency CS) must be available within 30 minutes, and neonatal resuscitation staff should be present.

4. Risks and Complications

Maternal Risks:

- **Perineal Tears:** 3rd and 4th-degree tears are more common with forceps (8–12%) than vacuum (1–4%).
- **Vaginal/Vulval Trauma:** Occurs in 1 in 5 forceps deliveries and 1 in 10 vacuums. **Episiotomy** is considered an intentional **second-degree tear**.
- **Post-Delivery:** Increased risks of PPH, urinary retention, and pelvic floor dysfunction.

Fetal Risks:

- **Common/Innocuous:** Forceps marks on the face or a **chignon** (scalp swelling) from vacuum.
- **Serious: Cephalohematoma** (the most common vacuum complication), **subgaleal hematoma**, and **intracranial hemorrhage**.
    - *Note:* Clinically significant ICH is actually most common in CS performed during labor.
- **Nerve Injuries:** Facial nerve palsy (rare with forceps). **Brachial plexus injury** (Erb's palsy) is a risk specifically during difficult deliveries/shoulder dystocia.

5. Procedure Management and Failure

- **Traction:** Pull only during contractions and stop if there are **3 pulls without progress** or **2 "pop-offs"** of the vacuum cup.
- **Abandonment:** The procedure must be abandoned if there is no progressive descent or if delivery is not imminent after 3 contractions with correct instrument application.
- **Failure Factors:** High maternal BMI (>30), fetal weight >4000g, **occipito-posterior (OP) position**, and mid-cavity station.

6. Shoulder Dystocia

Shoulder dystocia is a major obstetric emergency often associated with a **prolonged first stage of labor**.

- **Predictors:** Previous shoulder dystocia and macrosomia, though **ultrasound is not an accurate predictor of fetal weight**.
- **Management:**
    - **DO NOT** apply fundal pressure (can cause uterine rupture or further impaction).
    - **Use McRoberts position** (hyperflexion of maternal hips) and **suprapubic pressure**.
    - Secondary maneuvers include **internal rotation (Corkscrew)** or delivery of the posterior arm.
- **Complications: Brachial plexus injury** (Erb's Palsy, involving **C5-C6** nerve roots) and fractures of the clavicle or humerus.

7. Post-Procedure Aftercare

- **Medical:** Provide paracetamol/diclofenac for pain, **prophylactic antibiotics** (recommended for CS and OVD), and reassess for **thromboprophylaxis (VTE risk)**.
- **Bladder Care:** Monitor the first void to detect retention.
- **Future Pregnancies:** Women should be encouraged that they have an **80% chance of a successful spontaneous vaginal delivery** in subsequent pregnancies.
- **Psychological:** Address potential **tocophobia** (fear of childbirth) resulting from a traumatic delivery experience.

Cesarean Section

Definition and Epidemiology

- **Definition:** A surgical procedure under anaesthesia where the fetus, placenta, and membranes are delivered through incisions in the abdominal wall and uterus.
- **Incidence:** While the ideal rate is considered **10-15%**, global rates have risen two- to three-fold in the last decade.
    - In the US (2017), the rate was approximately **32%**.
    - In Jordan (University Teaching Hospitals), it rose from **18.2% in 2002 to 30.3% in 2012**.
- **Most Common Indication:** In recent years, the most common indication for C-section is a **previous Caesarean section**.

Surgical Anatomy and Incisions

1. Anatomical Layers (External to Internal)

Skin Fat Rectus sheath Rectus abdominis Parietal peritoneum Visceral peritoneum Uterine muscles.

2. Skin Incisions

- **Low Transverse (Pfannenstiel/"Bikini line"):** Offers improved cosmetic results, decreased pain, and superior wound strength.
- **Vertical Midline Infraumbilical:** Provides easier access to organs and can be enlarged easily; however, it carries a higher risk of **wound dehiscence and incisional hernia**. It is the standard skin incision for a Classical C-section.

3. Uterine Incisions

- **Lower Uterine Segment C/S (LUSCS):** The most common type (>95%).
    - **Advantages:** Easier to repair, reduced blood loss, faster healing (due to more fibrous tissue), and lower risk of rupture in future pregnancies.
- **Classical C/S (Upper Vertical):** A longitudinal incision in the upper segment, rarely performed.
    - **Major Concern:** High risk of **scar rupture** in subsequent pregnancy and labour.

- **Indications:** Fibroids occupying the lower segment, dense adhesions, certain placenta previa cases, **transverse lie (especially back down)**, fetal abnormalities (e.g., conjoined twins), constriction ring, cervical carcinoma, and peri-mortem operations.
- *Note:* Anterior uterine wall fibroids (not occupying the LUS) are generally **not** considered an indication for a classical section.

Indications for Caesarean Section

- **Labour Issues: Failed induction of labour** (the most frequent indication in labour), failure to progress (labour dystocia), and Cephalopelvic Disproportion (CPD).
- **Fetal Concerns:** Fetal distress (hypoxia, bradycardia), cord prolapse, IUGR, macrosomia, and malpresentations (breech, face, brow).
- **Placental/Vascular:** Placenta previa, vasa previa, and Placenta Accreta/increta/percreta.
    - *Note:* While major abruptio placenta is listed as an indication, some past paper sources suggest it may not be an absolute indication depending on the clinical context.
- **Maternal Health:** Active maternal infections (HIV, HSV, Hep C + HIV) to reduce transmission, and medical disorders like Hypertension or Diabetes.
- **Previous Surgery:** Two previous LUSCS, one previous classical C-section, previous uterine rupture, or **previous myomectomy where the endometrial cavity was entered**.

Pre-operative Management and Preparation

- **Timing:** Elective C-sections should **not** be performed before **39 weeks** to avoid neonatal respiratory morbidity.
- **Screening:** Full blood count (to identify anaemia), blood grouping, and antibody screening.
- **Prophylaxis:**
    - **Antibiotics:** Given **before** the skin incision to prevent endometritis and wound/UTI infections.
    - **Skin Prep:** Use alcohol-based chlorhexidine.

    - **Vaginal Prep:** Use aqueous povidone-iodine if membranes are ruptured.
    - **Gastric Prep:** Antacids (H2-blockers/PPIs) and anti-emetics to reduce gastric acidity and nausea.
    - **Thromboprophylaxis:** Essential, as C-sections have a **4-fold higher risk of DVT** than vaginal delivery. Methods include hydration, early mobilisation, stockings, or LMWH.
- **Anaesthesia: Regional anaesthesia** is preferred over general anaesthesia, even in cases of placenta previa.

Complications

1. Maternal Complications

- **Intraoperative:** Bleeding (atony, vessel injury), bladder/bowel injury, and anaesthesia complications.
    - **Caesarean Hysterectomy:** Most commonly performed due to **uncontrollable haemorrhage**.
- **Postoperative/Early:** Pain, lung atelectasis, paralytic ileus, DVT, and infections (UTI, endometritis, wound infection).
- **Fever after C/S (Causes):** Atelectasis, wound infection, endometritis (risk is 5-20 fold higher than vaginal delivery), bacteremia, UTI, breast abscess, and DVT.
- **Long-term:** Adhesions, increased risk of future placenta previa/accreta, and uterine rupture.

2. Fetal Complications

- **Respiratory:** Increased risk of **Transient Tachypnea of the Newborn (TTN)**.
- **Other:** Surgical nicks (rare), iatrogenic prematurity, and low APGAR scores.
- **Neonatal Care:** A trained practitioner skilled in resuscitation must be present for general anaesthesia or fetal compromise cases. Early **skin-to-skin contact** and breastfeeding support should be offered.

Trial of Labour After Caesarean (TOLAC) and VBAC

- **Success Rate:** Up to **70%** of women can achieve a vaginal birth after C-section (VBAC).
- **Best Predictors of Success:**
    - **Previous vaginal birth** (specifically a previous VBAC) is the best predictor.
    - Success is also high if the indication for the previous C-section was **failure to progress**.
- **Candidates for TOLAC:**
    - Previous **one** LUSCS (not classical or vertical scars).
    - Inter-pregnancy interval months.
    - Non-recurring previous indication and spontaneous labour.
- **Contraindications:** Previous **classical (upper segment) scar** or previous myomectomy breaching the uterine cavity.
- **Management Requirements:**
    - **Continuous fetal heart rate monitoring** is strongly recommended.
    - Facility must have the capability for an **emergency C-section**.
    - **Prostaglandins should NOT be used** for induction in these patients.
    - Low-dose oxytocin or mechanical dilation (balloon) may be used.

Preterm Labor

1. Definitions and Epidemiology

- **Preterm Birth:** Defined by the WHO as birth occurring **before 37 completed weeks** of gestation.
- **Preterm Labour (PTL):** The occurrence of **regular uterine contractions** (typically 4 every 20 minutes) associated with **cervical changes** (dilatation ≥ 2 cm or effacement > 80%).
- **Threatened PTL:** Regular uterine contractions occurring without evidence of cervical changes.
- **Categories of Preterm Birth:**
    - **Moderately Preterm:** 33–36 weeks.
    - **Very Preterm:** < 32 weeks.
    - **Extremely Preterm:** < 28 weeks.
- **Significance:** PTL affects 7–12% of all deliveries but accounts for **over 85% of perinatal mortality and morbidity**. The primary causes of morbidity and mortality are **pulmonary immaturity (RDS)** and **intraventricular haemorrhage**.

2. Etiology and Risk Factors

The majority of preterm births are **spontaneous** (40–45% in high-income countries), followed by PPROM and iatrogenic causes.

- **Common Pathways:** Infection (cervical, vaginal, urinary), uterine stretch (multiple gestations), placental-vascular issues, and psychosocial stress.
- **Risk Factors:**
    - **Maternal Characteristics:** Age (<15 or >35), Black race (double the risk), low maternal weight (<50kg or low BMI), and habits such as smoking, alcohol, or coitus.
    - **Obstetric History: Previous preterm delivery** is a major risk factor for recurrence. Second-trimester abortions and a **short interval between pregnancies** (1–5 months) also double the risk.

    - **Pregnancy Complications: Multiple pregnancy** is the single biggest risk factor (60% of twins are born preterm). Other factors include polyhydramnios, uterine abnormalities, and antepartum haemorrhage (APH).
    - **Infection: Genital tract infections** are the most common single known cause of PTL. **Urinary tract infections (UTIs)** are also a major risk factor.
- **Cervical Incompetence:** While a cause of PTL, it is considered the **least common cause** among major obstetric emergencies like APH or PPROM. It is best diagnosed via **vaginal ultrasound** in the index pregnancy and is usually treated in the second trimester. Note: Multiple pregnancy does *not* cause cervical incompetence.

3. Prediction and Diagnosis

- **Ideal Timing for Risk Assessment:** Risk factors should ideally be assessed **before conception**.
- **Diagnostic Tests:**
    - **Fetal Fibronectin (fFN):** A glycoprotein normally low between 22–37 weeks. A positive test predicts PTL, while a **negative test reduces the risk to less than 1%**. It is considered abnormal if detected between 22-34 weeks (not before 20 weeks).
    - **Cervical Length (TVUSS):** A cervical length of **≤ 1.5 cm** indicates a high risk (26%) of delivery before 34 weeks.
    - **Combined Testing:** Using fFN and cervical length together provides the most accurate prediction.
    - **Other markers:** Salivary oestriol, cervical swabs for pathogens, and various inflammatory markers (IL-6, IL-8) in amniotic fluid or serum.

4. Prevention and Management of PTL

- **Prevention:** Includes **cervical cerclage**, treating infections, and **progesterone therapy**.
    - **Vaginal Progesterone (200mg daily):** Significantly reduces PTB before 34 weeks in women with a **short cervix (< 15mm)**.
- **General Management:**
    - Initial evaluation includes history, physical exam, and mid-stream urine (MSU).
    - **Digital vaginal examination is generally indicated** to assess cervical status in suspected PTL (unlike PPROM).

- **Absolute Contraindications to Delaying Delivery:** Fetal death, congenital anomalies incompatible with life, **chorioamnionitis**, and fetal or maternal distress requiring immediate delivery. Preeclampsia *without* severe features is a relative, not absolute, contraindication.
- **Corticosteroids (Dexamethasone/Betamethasone):** Indicated between **24–36 weeks** to promote surfactant production and reduce RDS.

5. Tocolytic Agents

Tocolytics are used for up to 48 hours to allow for steroid administration or in-utero transfer.

- **Nifedipine (Calcium Channel Blocker):** Associated with **better neonatal outcomes** and fewer adverse effects compared to beta-agonists.
- **Atosiban:** An **oxytocin antagonist** used to suppress labor.
- **Magnesium Sulphate (MgSO4):** Used for **neuroprotection** (reduces cerebral palsy) and as a tocolytic. The antidote is **calcium gluconate**. Contraindicated in Myasthenia Gravis and renal failure.
- **Prostaglandin Synthetase Inhibitors (e.g., Indomethacin):** Side effects include **premature closure of the ductus arteriosus** and oligohydramnios.
- **Beta-Adrenergic Agonists (e.g., Ritodrine):** Should be **avoided in women with hypotension** or cardiac disease. They have a high frequency of adverse effects, including palpitations, pulmonary edema, and neonatal hypoglycemia.
- **Contraindication Note:** Tocolysis should **not be used if intrauterine infection (chorioamnionitis) is present**.

6. Preterm Premature Rupture of Membranes (PPROM)

Defined as rupture of membranes before 37 weeks and before the onset of labour.

- **Diagnosis:**
    - **Speculum Examination:** The **initial and most important test** to confirm a gush of fluid, see the cervix, and take swabs.
    - **Digital vaginal exams should be avoided** in PPROM due to infection risk.
    - **Nitrazine Test:** pH 7–7.5 (turns paper from yellow to blue).
    - **Ferning/Arborization:** Microscopic crystallization of dried fluid.

- **Chorioamnionitis:** A major complication. Key diagnostic criteria include **fever, maternal/fetal tachycardia, uterine tenderness**, and foul-smelling discharge. It requires immediate delivery regardless of gestational age. Diagnosis can be clinical, microbiological, or histopathological.
- **Management Strategy:**
    - **< 34 weeks:** Expectant management (hospitalisation, vitals, steroids, and prophylactic antibiotics).
    - **> 34 weeks:** Delivery is generally recommended.
    - **Antibiotics:** Prophylactic antibiotics are given to increase the latent period and reduce infection, though some (like Co-amoxiclav) are linked to increased risks of cerebral palsy in specific contexts.

Antepartum Hemorrhage

1. Overview of Antepartum Hemorrhage (APH)

- **Definition:** Bleeding from or into the genital tract occurring from **24 weeks of pregnancy** until the birth of the baby.
- **Impact:** Complicates 3–5% of pregnancies and is a **leading cause of perinatal and maternal mortality** worldwide.
- **Classification of Severity:**
    - **Spotting:** Staining or streaking on underwear.
    - **Minor:** Blood loss **< 50 ml** that has settled.
    - **Major:** Blood loss of **50–1000 ml** without signs of clinical shock.
    - **Massive:** Blood loss **> 1000 ml** and/or **signs of clinical shock**.
- **Note:** The amount of visible blood often **underestimates** total loss (e.g., in concealed abruption). Signs of shock and fetal compromise are better indicators of volume depletion. In fluid replacement, **urine output** is the best parameter to indicate adequate volume.

2. Placenta Previa

Definition: The placenta is inserted partially or fully in the **lower segment of the uterus**.

- **Grades:**
    1. **Grade 1 (Low Implantation):** Reaches lower segment but not the internal os.
    2. **Grade 2:** Reaches the internal os but does not cover it.
    3. **Grade 3 (Partial):** Covers the internal os asymmetrically.
    4. **Grade 4 (Complete):** Centrally covers the internal os.
- **Risk Factors:**
    - Previous placenta previa.
    - **Uterine scars** (Previous C-sections, curettage, endometritis).
    - Multiparity and advanced maternal age (>40).
    - Multiple pregnancy, smoking, and **Assisted Conception (IVF)**.

    - **Note:** Primigravida is *less likely* to be associated with previa compared to multigravida.

- **Clinical Presentation:**
    - **Painless, recurrent vaginal bleeding** (70% of cases).
    - **Soft uterus** on examination (tenderness usually rules out previa).
    - **High presenting part** or fetal malpresentation (breech/transverse).
- **Diagnosis & Management:**
    - **Ultrasound (U/S):** The primary diagnostic tool. Transvaginal scan is 100% accurate.
    - **Contraindication: Digital vaginal examination is strictly contraindicated** until previa is ruled out.
    - **Migrating Placenta:** Most placentas found "low" at 20 weeks will migrate upwards as the pregnancy progresses.
    - **Outpatient Care:** Patients with **major** placenta previa should **not** be managed as outpatients; they require hospitalization.
    - **Delivery:** Heavy bleeding with a viable fetus often requires **emergency Caesarean Section**.

3. Placental Abruption

Definition: Separation of the placenta from the implantation site after 24 weeks but before delivery.

- **Types:** Total or partial; **Concealed or revealed**.
- **Risk Factors:**
    - **Previous abruption** (The **most predictive factor**).
    - Pre-eclampsia/Hypertension.
    - Trauma (Accidents or domestic violence).
    - **Cocaine/Amphetamine use** and smoking.
    - Polyhydramnios, low BMI, and intrauterine infection.

- **Clinical Presentation:**
    - **Painful vaginal bleeding** with **uterine tenderness** or back pain.
    - **Hypertonic uterus** ("woody" hard) and high-frequency contractions.
    - Fetal distress or Intrauterine Fetal Death (IUFD).
- **Complications:**
    - **Consumptive Coagulopathy/DIC:** Abruption is the **commonest cause of blood clotting defects** in pregnancy.
    - Maternal shock, Renal tubular necrosis, and **Sheehan syndrome**.
- **Diagnosis & Management:**
    - Diagnosis is primarily **clinical**; U/S only detects about 2% of abruptions.
    - **Mode of Delivery:** CS is **not absolute**; vaginal delivery may be possible. If the fetus is dead, **forewater amniotomy** and vaginal birth are recommended.

4. Vasa Previa

Definition: Fetal vessels from the umbilical cord traverse the membranes in the lower uterine segment, ahead of the presenting part.

- **Risks:** Rupture of these vessels leads to **fetal exsanguination**.
- **Diagnosis:**
    - Suspected if bleeding occurs **immediately upon rupture of membranes** with concomitant fetal heart abnormalities (e.g., a **sinusoidal trace**).
    - **Apt Test:** Differentiates fetal from maternal blood. Fetal hemoglobin **stays pink** when mixed with NaOH, while adult blood turns yellow-brown.
- **Management:** Hospitalization in the third trimester, corticosteroids for lung maturity, and **elective CS at 35–36 weeks**.

5. Uterine Rupture

Definition: Complete separation of the uterine musculature through all layers.

- **Risk Factors:** Primarily a **previous uterine scar** (C-section incisions—classical/T-shape carry higher risk than low transverse).

- **Presentation:** Sudden **acute abdominal pain**, vaginal bleeding, **loss of fetal station** (head recedes), and easily palpable fetal parts.
- **Management:** Maternal stabilization and prompt **Caesarean section** with either uterine repair or **hysterectomy**.

6. General Management of APH

- **Immediate Action:** The **mother is the priority** and must be stabilized before assessing the fetus.
- **Initial Investigations:**
    - Vitals, abdominal palpation, and U/S to confirm fetal heart and **exclude previa**.
    - **Labs:** CBC, Blood Group/Rh, Cross-match, Coagulation profile, LFT/KFT.
    - **Kleihauer Test:** To quantify feto-maternal hemorrhage.
- **Treatment Protocols:**
    - **Corticosteroids:** Offered between **24+0 and 34+6 weeks** if preterm birth is a risk.
    - **Tocolysis:** Contraindicated in abruption or major APH.
    - **Anti-D Ig:** Must be given to all **non-sensitized RhD-negative women** after any APH episode.
- **Delivery Timing:**
    - Immediate delivery if there is maternal or fetal compromise.
    - If the woman is >37 weeks with minor/major APH, **induction of labour** is advised (unless it is placenta previa, which requires CS).

7. Specific Pathologies (MCQ Key Points)

- **Placenta Accreta:** Usually treated via **hysterectomy** or hypogastric artery ligation.
- **Placenta Percreta:** May present with bluish tissue adherent between the uterus and bladder.
- **Show:** Maternal "bloody show" is a common obstetric cause of APH.
- **Local Causes:** Cervical polyps, cervicitis, or trauma.

Postpartum Hemorrhage

I. Postpartum Hemorrhage (PPH): Fundamentals

- **Definition:** Blood loss **>500 cc** in vaginal deliveries or **>1,000 cc** in abdominal (caesarean) deliveries. It is also defined as any blood loss causing **hemodynamic instability**.
- **Classification:**
    - **Primary:** Occurs within the first **24 hours** of delivery.
    - **Secondary:** Occurs after 24 hours up to 6 weeks postpartum. Causes include retained products of conception, infection (endometritis), uterine sub-involution, or myoma.
- **Significance:** PPH is often **underestimated**, is generally well-tolerated until a "breaking point," and significantly impacts **maternal mortality**.
- **Mechanism of Normal Delivery:** The main mechanism for placental separation is the **reduction in placental site surface area** as the uterus shrinks.

II. Etiology: The "4 Ts"

Category	Specific Causes & Risk Factors
Tone (Atony)	**Commonest cause (90%)**. Risk factors: Overdistended uterus (multiple gestation, large baby, polyhydramnios), prolonged labor, high parity, and general anesthesia. **Primigravida is NOT a risk factor**.
Tissue	Retained placenta, membranes, or clots. Higher incidence in patients with **previous multiple Caesarean sections**.
Trauma	Lacerations (cervical/vaginal), episiotomy, uterine rupture, or inversion. **If the uterus is FIRM but bleeding is heavy, suspect genital tract trauma**.
Thrombin	Coagulopathies or bleeding tendencies.

III. Management of PPH

1. Immediate Steps (ABC)

- Assess the patient, call for help, and establish **large-bore IV access** for crystalloid resuscitation.
- Order CBC, cross-match, and type.

- Assess the **uterine fundus** and perform **bimanual massage**.

2. Medical Management (Uterotonics)

- **Oxytocin:** The first-line treatment. Administered as 5-10 units IV/IM or diluted in N/S.
- **Ergometrine:** (0.25 mg IM). **Contraindication: Hypertension**.
- **Hemabate (Carboprost/PGF2α):** (0.25 mg IM). **Relative Contraindication: Asthma**.
- **Cytotec (Misoprostol):** Classified as **PGE1**. Dose: 800–1000 mcg per rectum.
- **Note: Prostaglandin E2 (PGE2) is NOT used** for PPH management.

3. Surgical & Advanced Management

- **Manual exploration** of the uterus to rule out retained tissue, rupture, or inversion.
- **Refractory PPH:** Options include B-Lynch sutures, uterine artery ligation, or uterine artery embolization. **IV Iron supplements are NOT appropriate** for acute, massive (2L+) blood loss.

IV. Maternal Injuries: Episiotomy & Tears

1. Episiotomy Types

- **Midline (Median):**
    - **Pros:** Less blood loss, easier to repair, and more comfortable post-op.
    - **Cons:** Higher risk of extension into the **anal sphincter** or mucosa. It does NOT increase instrumental delivery rates (it is used to facilitate them).
- **Mediolateral:**
    - **Pros: Reduces the risk of extension** into the anal sphincter compared to midline.
    - **Cons: More bleeding** and more painful than midline.

2. Perineal Tear Classification

- **1st Degree:** Vaginal mucosa/epithelial layer only.
- **2nd Degree:** Mucosa and **muscularis/fascia** layer.
- **3rd Degree:** Includes the **external anal sphincter** fibers (rectal mucosa remains intact).
- **4th Degree:** Extends through the anal sphincter into the **rectal mucosa**.

V. Major Obstetric Complications

1. Uterine Rupture

- **Leading Risk Factor:** Previous **Caesarean section scar**, especially when labor is augmented with **Syntocinon/Oxytocin**.
- **Clinical Presentation:** Sudden **severe fetal heart rate abnormalities** (most common finding), cessation of labor, recession of the presenting part, and easily palpable fetal parts.
- **Outcome:** Fetal mortality can reach **50%**.
- **Myomectomy History:** Also increases the risk of uterine rupture in subsequent pregnancies.

2. Uterine Inversion

- **Primary Cause: Excessive umbilical cord traction**.
- **Management: Manual replacement** of the uterus immediately ("last out, first in") **before** removing the placenta. Shock in these cases is often due to increased **vagal tone**.

3. Paragenital Hematomas

- **Infralevator (Vulvar):** Painful; small ones are treated with ice packs, large ones require surgical drainage.
- **Supralevator (Paravaginal):** Dangerous as they can involve **massive blood loss** into the broad ligament/retroperitoneal space; may be initially less painful.

Easy Memorisation Summary (The "Don't Forget" List)

- **Commonest PPH Cause:** Uterine Atony.
- **Medication to Avoid in PPH:** PGE2.
- **Firm Uterus + Bleeding:** Suspect Trauma/Lacerations.
- **Misoprostol Identity:** It is PGE1.
- **Uterine Rupture Warning:** Sudden fetal distress is the hallmark.
- **Mediolateral Episiotomy:** Design choice to avoid the anal sphincter.
- **Post-Maturity:** Is NOT a significant risk factor for PPH.

Postpartum Care

1. The "Big Reset": Physical Changes

- **Uterine Involution:** The uterus contracts to prevent hemorrhage. The fundus (top of the uterus) is near the **umbilicus at 24 hours**, midway to the pubic bone at one week, and no longer felt by 12 days.
- **Lochia (Discharge):** This is the shedding of blood and tissue. It follows three stages: **Rubra** (red-brown, first few days), **Serosa** (pink-brown, 2-3 weeks), and **Alba** (yellow-white).
- **Cervix & Vagina:** The cervix remains 2–3 cm dilated for the first few days and returns to less than 1 cm by one week. The vaginal opening changes permanently, and rugae (folds) return by the third week.
- **Weight Loss:** Most women lose about **6 kg immediately** (baby, placenta, fluid) and another **2–7 kg** over the following weeks as fluids and the uterus return to normal.

2. Hormones & Healing

- **Hormones:** Pregnancy hormones (hCG) usually disappear within **2 to 4 weeks**. Non-lactating women typically see menstruation return in 45–64 days, while **breastfeeding delays ovulation** by inhibiting the hormones that trigger it.
- **Skin & Hair:** "Mask of pregnancy" (chloasma) fades. Hair loss (**telogen effluvium**) is common 1–5 months after delivery but usually resolves within a year.
- **Breasts:** Breast milk is the optimal feeding choice. **Engorgement** (swelling/pain) can be managed with tight bras, avoiding stimulation, and cool compresses; drug therapy for suppression is not recommended due to risks.

3. Immediate Care & Comfort

- **Skin-to-Skin:** Encouraged immediately to improve the baby's temperature regulation, blood sugar, and maternal attachment.
- **Shivering:** Chills affect 25–50% of women within 30 minutes of delivery. They are normal and treated simply with **warm blankets**.
- **Pain Management:** "Afterpains" from uterine contractions are common during nursing and are best treated with **NSAIDs (like ibuprofen)** rather than opioids.

- **Urinary Health:** Retention (difficulty peeing) occurs in 4% of women due to nerve injury. It is usually self-limiting and clears within a week.

4. Safety & Complications (The Warning Signs)

- **Infection/Fever:** A temperature of **38.0°C or higher** on any two days (excluding the first 24 hours) may indicate a UTI, wound infection, or mastitis.
- **Thrombosis (VTE):** The risk for blood clots is **highest in the first two weeks** and is significantly higher after a C-section.
- **Preeclampsia:** This can occur **after delivery**, most often within the first 48 hours.
- **Excessive Bleeding:** While lochia is normal, heavy bleeding persisting beyond eight weeks or sudden heavy bleeding several weeks later requires medical evaluation.

Summary Table for Quick Memory

Category	Key Fact to Remember
Uterus	Fundus non-palpable by day 12; normal size by week 8.
Lochia	Changes from Red (Rubra) to Pink (Serosa) to White (Alba).
Pain	NSAIDs are the first-line choice for uterine and perineal pain.
Fever	38.0°C+ after the first 24 hours is a red flag for infection.
Clots	Peak risk is in the first 14 days postpartum.
Rh- Mothers	Must receive anti-D immune globulin if the baby is Rh+.

Gynecology

Puberty

1. The Physiology of Puberty

- **Definition:** The process of reproductive and sexual development changing a child into an adult.
- **The HPO Axis:** Puberty represents the activation of the **Hypothalamic-Pituitary-Ovarian (HPO) axis**.
    - **Prepubertal State:** Characterised by an **inactive HPO axis**, low gonadotropins, and maximal hypothalamic suppression.
    - **Fetal Development:** GnRH is detectable by 10 weeks; FSH/LH are produced by 10–13 weeks, peaking at 20–28 weeks of intrauterine life.
    - **Activation:** Pulsatile GnRH secretion begins at age 8–9, stimulating the pituitary to release FSH and LH, which then trigger ovarian oestrogen production.
- **Key Hormones:**
    - **Adrenarche:** Elevation of DHEA and DHEAS beginning at age 6–8.
    - **Interactions:** Menstrual cycles require the interaction of the hypothalamus, pituitary, ovaries, and endometrium (the thyroid is not a primary component of this specific axis).

2. Physical Landmarks and Chronology

The first physical sign of puberty is typically **breast budding (thelarche)**. The standard sequence is:

1. **Thelarche:** Breast development.
2. **Adrenarche/Pubarche:** Growth of pubic and axillary hair.
3. **Growth Spurt:** Height acceleration.
4. **Menarche:** Onset of menstruation.
5. **Menarche:** The median age is **10–14 years** (mean 12.8). Initially, cycles are often **anovulatory, irregular, and unpredictable**; anovulation is the **most common cause** of adolescent menstrual irregularities.
6. **Growth Velocity:** Maximum growth velocity occurs during **Tanner Stage 3**. Height generally stops increasing by age 16.

3. Tanner Staging (The Sexual Maturity Rating)

Stage	Breast Development	Pubic Hair Development
Stage 1	Prepubertal; papilla elevation only.	Villus hair only.
Stage 2	**Breast buds** palpable; areolae enlarge.	Minimal coarse, pigmented hair on labia.
Stage 3	Further elevation; areolae enlarge.	Dark, curly hair spreads over mons pubis.
Stage 4	Areola forms a **secondary mound**.	Adult quality hair; no spread to medial thigh.
Stage 5	Adult contour; areola recesses.	Adult distribution; **spreads to medial thigh**.

4. Precocious Puberty

Defined as the onset of puberty before **age 8 in girls** or age 9 in boys.

- **Central (Gonadotropin-dependent):** Early activation of the HPO axis. 75% are idiopathic; 25% are due to CNS malformations/tumours or meningitis.
- **Peripheral (Gonadotropin-independent):** Driven by external hormones or tumours (e.g., **McCune-Albright syndrome** or hormone-secreting ovarian tumours).
    - *Note:* In ovarian tumours, oestrogen is high, but **gonadotropins are low**.
- **Workup:** Includes FSH/LH levels, **Hand and wrist X-ray (bone age)**, CNS radiography, and Pelvic Ultrasound.
- **Treatment Goals:** Treat underlying causes, allow for psychological maturity, decrease social stigma, and **maximize adult height** (improving cognitive function is not a goal). **GnRH analogues** are the mainstay for central precocious puberty.
- **Premature Thelarche:** Isolated breast development before age 8. Crucially, **bone age is not advanced**, growth is not accelerated, and **surgical biopsy is contraindicated**.

5. Delayed Puberty and Clinical Conditions

Defined as no secondary sexual characteristics by **age 14**.

- **Hypogonadotropic (Low FSH/LH):** Caused by constitutional delay, **anorexia nervosa**, excessive exercise, or Kallman syndrome.
- **Hypergonadotropic (High FSH/LH):** Indicates ovarian failure (e.g., **Turner syndrome**, autoimmune failure, or chemotherapy).
- **Imperforate Hymen:** A common cause of primary amenorrhea.
    - **Presentation:** Typically age 14–16 with cyclical abdominal pain and potentially **acute urinary retention**.
    - **Findings:** Normal karyotype and **normally developed secondary sexual characteristics**. Hirsutism is not a feature.
- **Anorexia Nervosa:** Often affects higher socio-economic groups; can occur before puberty and may be associated with binge eating. It is **not** typically associated with antisocial behaviour.
- **Virilisation:** Facial hair and clitoromegaly can be caused by adrenal/ovarian tumours, CAH, or exogenous androgens, but **not hyperthyroidism**.
- **Treatment:** Target the underlying cause, Watchful waiting, Gonadal hormone replacement, Growth hormone therapy.

Menopause

I. Definitions and Classifications

- **Menopause:** The permanent cessation of menstruation resulting from the loss of ovarian follicular activity.
- **Clinical Diagnosis:** It is recognized after **12 consecutive months of amenorrhea** with no other pathological or physiological cause.
- **Perimenopause:** The period immediately prior to menopause (when endocrinological and clinical features begin) and the first year after.
- **Climacteric:** The transition phase from the reproductive to the non-reproductive state.
- **Premature Menopause:** Defined as menopause occurring before the **age of 40**. It is associated with chromosomal abnormalities, radiotherapy, and pernicious anemia, but **not** with polycystic ovarian syndrome (PCOS).
- **Induced Menopause:** Follows surgical removal of both ovaries (oophorectomy) or iatrogenic ablation via chemotherapy or radiation.
- **Postmenopause:** The period dating from the FMP, regardless of whether the menopause was induced or spontaneous.

II. Pathophysiology and Hormonal Changes

- **Ovarian Decline:** There is a steady decline in oocytes; remaining follicles become resistant to FSH and fail to mature.
- **Hormonal Profile:**
    - **Decreased:** Ovarian **estradiol** and **inhibin** production.
    - **Increased:** Gonadotrophin levels (**FSH and LH**) significantly increase due to loss of negative feedback. FSH > 30 IU/L is a typical investigative finding.
    - **Post-menopausal Estrogen:** The predominant estrogen becomes **Estrone**.
- **Late Reproductive Phase:** During the last 4-5 years of reproductive life, fertility declines rapidly, anovulatory cycles increase, and follicular failure is progressive.

III. Clinical Manifestations (Symptoms)

Symptoms are categorized by their onset following the final menstrual period (FMP):

- **Short-Term (0–5 years):**
    - **Vasomotor Symptoms:** Hot flushes (the most distressing symptom) and night sweats.
    - **Psychological:** Insomnia, irritability, mood disturbances, and loss of concentration.
    - **Physical:** Joint aches, dry/itchy skin, and hair changes.
    - **Note:** Visual changes and weight loss are **not** classical menopausal symptoms.
- **Intermediate (3–10 years):**
    - **Urogenital Atrophy:** Vaginal dryness, **dyspareunia** (painful intercourse), and urogenital prolapse.
    - **Urinary:** Sensory urgency and recurrent UTIs.
- **Long-Term (>10 years):**
    - **Skeletal: Osteoporosis** and decreased bone density.
    - **Systemic:** Cardiovascular disease and dementia.

IV. Investigations

- FSH > 30 IU/L, preferably 2 measurements, 2 weeks to 3 months apart.
- Breast screening and mammography
- Endometrial assessment of unscheduled bleeding
- Cardiovascular disease risk assessment
- Skeletal assessment

IV. Management and Hormone Replacement Therapy (HRT)

A. Indications and Benefits

- The primary current indication for HRT is the **symptomatic relief of hot flushes**.
- It is also indicated for vaginal dryness, premature menopause (e.g., at age 32), or following a bilateral oophorectomy at a young age.

- **Benefits:** Relief of vasomotor/urogenital symptoms and prevention of osteoporosis and colon cancer.

B. Regimens and Routes

- **Routes:** Oral, topical (patches, gels, creams), or subcutaneous implants.
- **Regimens:**
    - **Estrogen-only:** Prescribed **only** for women who have undergone a **hysterectomy**.
    - **Combined HRT (Estrogen + Progesterone):** Required for women with an intact uterus to prevent endometrial cancer.
    - **Duration:** Generally the minimum effective dose for the shortest duration (average 2–3 years); however, in premature menopause, it should continue at least until age 50.

C. Non-Hormonal and Lifestyle Options

- **Prescription:** SSRIs, Beta-blockers, and Alpha-adrenergic agonists (e.g., Clonidine).
- **Lifestyle:** Smoking cessation (smoking induces earlier menopause), diet, exercise, and evening primrose oil.

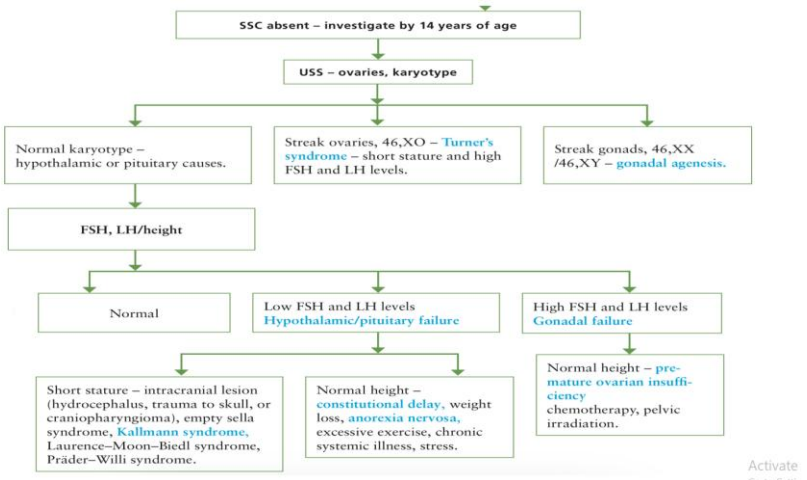
V. Risks and Contraindications (Crucial for Exams)

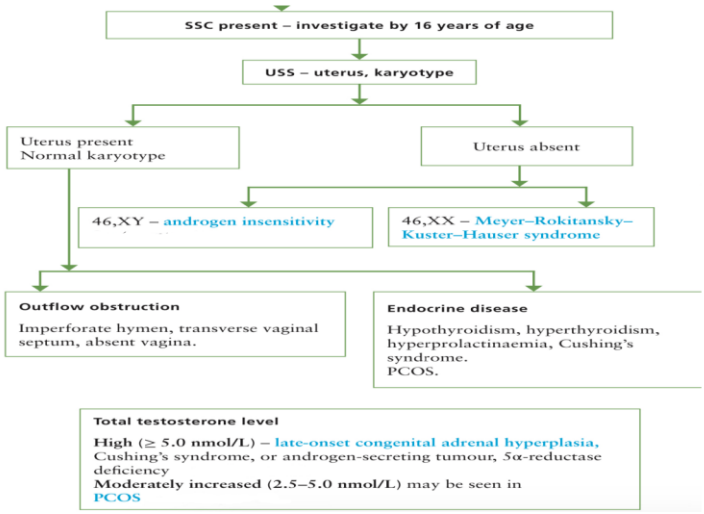
- **Absolute Contraindications:** Active liver disease, undiagnosed vaginal bleeding, breast cancer, endometrial cancer, known venous thromboembolism (VTE), and coronary artery disease.
    - *Note:* While pregnancy is an absolute contraindication in clinical practice, some exam formats may list it as an "exception" in specific context-based questions.
- **Risks (WHI Study):** HRT is associated with an increased risk of **Myocardial Infarction (MI), Stroke, Pulmonary Embolism (PE), and invasive breast cancer**.
    - **Important:** HRT is **not** associated with an increased risk of bowel cancer; it actually decreases it.
- **Osteoporosis Risk Factors:** Family history, slender body composition, alcohol, smoking, and long-term steroids. **Obesity** is actually protective against osteoporosis and is not a risk factor.

Amenorrhea

I. Definitions and Classifications

- **Amenorrhea:** The absence of menstruation in a woman of childbearing age.
- **Primary Amenorrhea:** Failure to establish menstruation by **15 years of age** with normal secondary sexual characteristics (SSC), or by **13 years of age** if no SSC are present.
- **Secondary Amenorrhea:** Absence of menstruation for **6 consecutive months** (if cycles were previously regular) or **12 months** (if cycles were previously oligomenorrheic).
- **Hypogonadotropic Hypogonadism:** Characterised by **low FSH/LH**; causes include Anorexia Nervosa, Kallmann syndrome, and constitutional delay.
- **Hypergonadotropic Hypogonadism:** Characterised by **high FSH/LH** (gonadal failure); examples include Turner syndrome and Premature Ovarian Failure (POF).





II. Outflow Tract Disorders

Disorders here often present with primary amenorrhea despite normal secondary sexual characteristics (Tanner Stage V) and a normal 46,XX karyotype.

1. **Imperforate Hymen:**
    - The **most common anomaly** resulting from abnormal Müllerian duct development.
    - **Presentation:** Cyclical lower abdominal pain, a **bulging purple/blue mass** at the introitus (haematocolpos), and potential urinary retention.
    - **Key Fact:** It is associated with a **normal karyotype** and **normal SSC**; secondary amenorrhea is *not* a typical presentation.
2. **Mayer-Rokitansky-Küster-Hauser (MRKH) Syndrome (Müllerian Agenesis):**
    - Second most common cause of primary amenorrhea.
    - **Features:** 46,XX karyotype, **absent uterus** and upper two-thirds of the vagina, but **normal ovaries** and SSC (breasts and pubic hair).
    - **Associations:** Often linked to renal and skeletal anomalies.
3. **Transverse Vaginal Septum:** Failure of fusion between the Müllerian tubercle and sinovaginal bulb; presents with cyclical pain and a "pink" bulge.
4. **Asherman Syndrome (Secondary Amenorrhea):**
    - Uterine synechiae (adhesions) often following **uterine curettage** for miscarriages.
    - Typically presents with **hypomenorrhea** or secondary amenorrhea.

III. Disorders of Sexual Development (DSD)

These conditions often involve a mismatch between chromosomal sex and phenotypic appearance.

1. **Androgen Insensitivity Syndrome (AIS):**
    - **Karyotype: 46,XY** (genetically male).
    - **Phenotype:** Female with **well-developed breasts** but **scanty/absent pubic and axillary hair**.
    - **Anatomy:** Undescended testes, **blind-ending vaginal pouch**, and **absent uterus** (due to intact Anti-Müllerian Hormone).

    - **Management:** Gonadectomy is performed **after puberty** (age 16-18) to allow for natural breast development while mitigating the risk of malignancy in cryptorchid testes.

2. **Turner Syndrome:**
    - **Karyotype: 45,XO**; the most common cause of gonadal dysgenesis.
    - **Features:** Short stature, webbed neck, widely spaced nipples, and **streak ovaries**.
    - **Note:** Incidence **does NOT rise** with maternal age (unlike Down Syndrome).
3. **Swyer Syndrome:** 46,XY karyotype but female phenotype because streak gonads produce neither AMH nor androgens; the **uterus is present**.
4. **True Hermaphroditism:** Requires the **presence of both testicular and ovarian tissue**.

IV. Hypothalamic and Pituitary Causes

1. **Anorexia Nervosa:**
    - The **most common pathologic cause** of amenorrhea in adolescent females.
    - **Symptoms:** Amenorrhea, dry skin, **hypothermia**, constipation, and **bradycardia** (Note: it does *not* cause tachycardia).
2. **Kallmann Syndrome:** Characterised by GnRH deficiency, primary amenorrhea, and **anosmia** (loss of smell).
3. **Sheehan Syndrome:** Pituitary infarction following severe **postpartum haemorrhage**; earliest symptom is **failed lactation**.
4. **Empty Sella Syndrome:** Can follow surgery or radiotherapy; it typically does **not** progress to pituitary failure, and induction of ovulation is **not** contraindicated.
5. **Prolactinoma:** Elevated prolactin inhibits GnRH; **dopamine inhibitors** (like bromocriptine) suppress secretion, while phenothiazines and stress increase it.

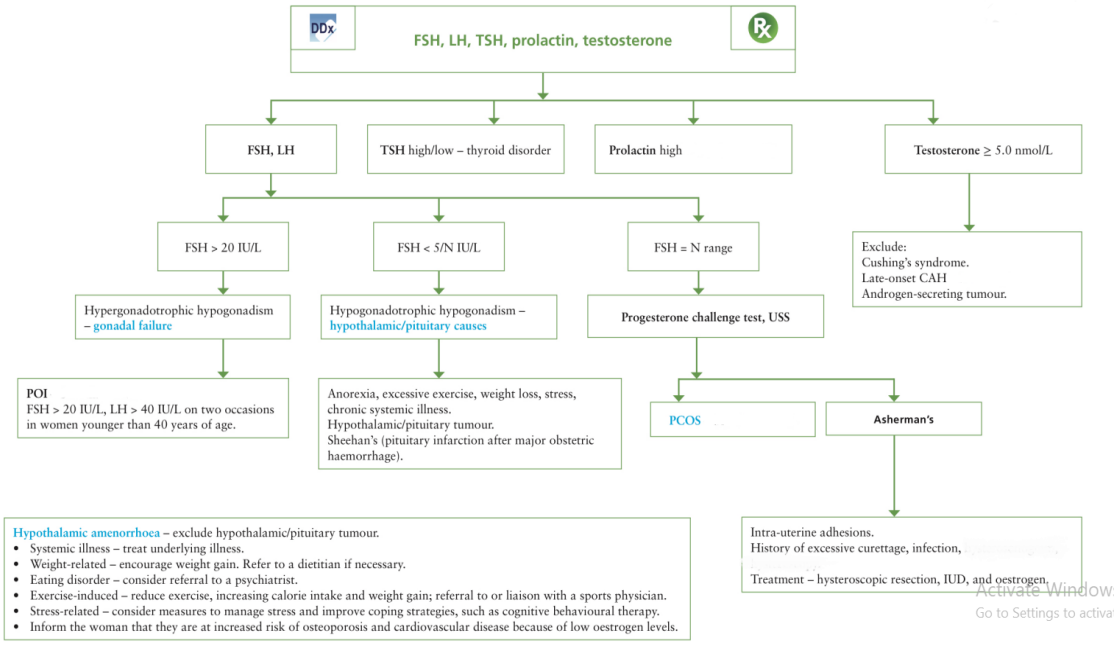
V. Endocrine and Other Causes

- **Polycystic Ovary Syndrome (PCOS):** The most common cause of adult-onset anovulation; causes amenorrhea due to a **thickened endometrium** and hormonal imbalance.
- **Congenital Adrenal Hyperplasia (CAH):** Most commonly a **21-hydroxylase deficiency**, leading to ambiguous genitalia in girls and salt-wasting.

- **Primary Ovarian Insufficiency (POI):** Ovarian failure before age 40; symptoms include **vaginal dryness, dyspareunia, and insomnia**.

VI. Diagnostic and Investigation Summary (Easy Memorisation)

- **Karyotyping:** Essential for differentiating AIS (46,XY) from MRKH (46,XX) and identifying Turner Syndrome (45,XO).
- **Progesterone Withdrawal Test:** Used to assess endogenous estrogen levels and outflow tract patency.
- **Hysterosalpingogram (HSG):** Used to diagnose conditions like **hydrosalpinx** and Asherman syndrome.
- **Ovulation Tests:** Include mid-luteal progesterone, basal body temperature, and LH kits; **oestradiol levels** are *not* a reliable test for ovulation.
- **Normal Sexual Differentiation:** If testes are absent, the fetus defaults to a female phenotype; male genitalia require fetal testosterone. If Müllerian-inhibiting factor is lacking in a male, a **uterus and fallopian tubes** will develop.



Dysmenorrhea and Premenstrual Syndrome

I. Dysmenorrhea (Painful Menstruation)

Dysmenorrhea is defined as pain associated with menstruation, affecting 50%–70% of women, with 10% being incapacitated by it.

1. Primary (Spasmodic) Dysmenorrhea

- **Definition:** Painful menses in the **absence of pelvic pathology**.
- **Profile:** Typically starts **1–2 years after menarche** (associated with ovulatory cycles) and is most common in younger women.
- **Etiology (Theories):**
    - **Prostaglandins:** The most accepted theory. High levels of **PGF2α** (vasoconstrictor/contractor) and **PGE2** (nerve sensitiser) lead to uterine hyperactivity and ischemia.
    - **Hormonal:** Depends on progesterone; therefore, **anovulatory cycles are usually painless**.
- **Risk Factors:** Early menarche, long/heavy flow, heavy smoking, and a **sedentary life**. Lower consumption of fish, eggs, and fruits also increases risk.
- **Clinical Features:**
    - **Pain Type:** Spasmodic/cramping pain in the lower abdomen, groins, and thighs.
    - **Timing:** Starts just before or at the onset of bleeding and lasts about 2 days.
    - **Associated Symptoms:** Nausea, vomiting, fatigue, headache, and backache. **Note:** Constipation is *not* a typical symptom; bowel disturbances (like diarrhea) are more common.
    - **Examination:** Pelvic examination is typically **normal**.
- **Management:**
    - **General:** Reassurance, exercise, and dietary improvements.
    - **Medical (First-line): NSAIDs** (e.g., Ibuprofen, Mefenamic acid) are the mainstay as they inhibit prostaglandins. **Combined OCPs** are also highly effective as they abolish ovulation.

- **Surgical:** Reserved for severe cases; includes cervical dilation or nerve ablation (Cotte's operation).

2. Secondary (Congestive/Organic) Dysmenorrhea

- **Definition:** Painful menses due to **underlying organic pelvic disease**.
- **Profile:** Usually seen in older women (3rd–4th decade) after years of painless cycles.
- **Common Causes: Endometriosis** (most common), adenomyosis, fibroids, Pelvic Inflammatory Disease (PID), and endometrial polyps.
- **Note:** OCPs are a *treatment*, not a cause. Genital prolapse is also *not* a cause.
- **Clinical Features:** Dull, aching pain that begins days before menses and often lasts throughout the cycle. Often associated with **dyspareunia** and **infertility**.
- **Diagnosis:** The **first logical test** for acute excessive bleeding in these patients is a **pregnancy test** to rule out complications. Investigations include Ultrasound, Hysteroscopy, and Laparoscopy.

II. Premenstrual Syndrome (PMS) & PMDD

PMS involves cyclical physical and emotional symptoms during the **luteal phase** that remit after menses starts. **PMDD** is the severe form with dominant emotional symptoms like anger and depression.

1. Etiology & Diagnosis

- **Cause:** Likely a **neuroendocrine disorder** involving **serotonergic dysfunction** triggered by hormonal changes.
- **Diagnostic Criteria:**
  - Symptoms must occur in the **luteal phase** and remit with menses.
  - They must **interfere with daily life**.
  - They must *not* be an exacerbation of an underlying psychiatric disorder.
  - Prospective charting for **2–3 cycles** is required for diagnosis.

2. Symptoms

- **Somatic (Physical): Abdominal bloating** (most common, 90%), breast tenderness, headache, and edema. **Note:** Vaginal spotting is *not* a symptom of PMS.
- **Affective (Emotional):** Irritability, anxiety, depression, and social withdrawal.

3. Management

- **Conservative:** Exercise, smoking cessation, and reducing caffeine/salt.
- **Supplements:** Vitamin B6 and Vitamin E.
- **Medical:**
    - **SSRIs (e.g., Fluoxetine):** Considered **first-line treatment** for moderate to severe cases.
    - **Hormonal:** GnRH agonists are effective for severe/resistant cases but are **not the best first-line** option due to side effects.
    - **Specific Symptoms:** Spironolactone for bloating/edema; Dostinex for breast tenderness.

III. Key Past Paper "Pearls" for Memorisation

- **History Taking:** When assessing gynecological history, the **regularity of the cycle**, amount of bleeding, and history of dysmenorrhea/spotting are relevant. In a single 40-year-old, HRT history is often least relevant.
- **Mittelschmerz Syndrome:** If a patient describes **mid-cycle pain** (ovulation pain) that resolves within 24 hours, the likely diagnosis is Mittelschmerz.
- **Primary vs. Secondary:** Primary dysmenorrhea starts **after** menarche (1–2 years) and is **spasmodic**. Secondary dysmenorrhea starts **later in life** and is **dull/aching**.
- **Primary Dysmenorrhea Facts:** It is **relieved by OCPs** and NSAIDs. Psychological states can **intensify** the symptoms but do not cause them. It is **not** caused by fibroids (that would be secondary).

Dysfunctional Uterine Bleeding

I. Fundamentals of the Menstrual Cycle

- **Normal Parameters:**
    - **Menarche/Menopause:** Starts at ~12.5 years; ends at 51–52 years.
    - **Frequency:** Every 21 to 35 days (average 28).
    - **Duration:** 2 to 7 days.
    - **Volume:** Clinically ≥5 to ≤80 mL. **Average loss is 25 to 50 mL**.
- **Key Definitions:**
    - **Abnormal Uterine Bleeding (AUB):** Any symptomatic variation from normal, including intermenstrual bleeding.
    - **Dysfunctional Uterine Bleeding (DUB):** AUB not due to organic disease, pregnancy, or systemic causes. It is common at perimenopausal ages and can be due to endometrial dysfunction.
    - **Menorrhagia:** Heavy regular periods.
    - **Metrorrhagia:** Bleeding at irregular intervals.
    - **Menometrorrhagia:** Heavy uterine bleeding at irregular intervals.
    - **Polymenorrhea:** Menses at intervals <21 days.
    - **Oligomenorrhea:** Infrequent periods (>35-day intervals).
    - **Hypermenorrhea:** Excessive regular menstrual loss.

II. Etiology: PALM-COEIN Classification

The causes of AUB in non-pregnant reproductive-age women are categorized as structural (**PALM**) or non-structural (**COEIN**):

Structural Causes (PALM)

- **Polyp (AUB-P):** Localized epithelial tumors in the endometrium or cervix. **Most common symptom is intermenstrual bleeding (spotting between periods)**.
- **Adenomyosis (AUB-A):** Endometrial tissue within the myometrium. Presents with **painful, heavy, regular periods** and an **enlarged, boggy, tender uterus**.

- **Leiomyoma (Fibroids) (AUB-L):** Benign smooth muscle neoplasms; primary symptom is **heavy regular periods**.
- **Malignancy & Hyperplasia (AUB-M):** Diagnosed via endometrial sampling; presents as heavy or irregular bleeding.

Non-Structural Causes (COEIN)

- **Coagulopathy (AUB-C):** Most commonly **von Willebrand's disease**. Also caused by drugs like Warfarin or Aspirin.
- **Ovulatory Dysfunction (AUB-O):** Occurs at extremes of reproductive age (puberty and perimenopause). **Anovulation is the most common cause in 13-year-olds**.
- **Endometrial (AUB-E):** Includes **endometritis** (e.g., from Chlamydia).
- **Iatrogenic (AUB-I):** Caused by steroids, GnRH analogues, anticoagulants, or drugs affecting dopamine (e.g., antipsychotics, methyldopa).
- **Not Otherwise Classified (AUB-N):** Arteriovenous malformations or Cesarean scar defects.

III. Important Clinical Distinctions

- **Causes of Regular Heavy Periods:** Fibroids, Hypothyroidism (Myxedema), Adenomyosis, and Ovulatory DUB.
- **Causes of Irregular/Intermenstrual Bleeding:** Endometrial polyps, Cervical cancer, and Endometritis.
- **Associations:** Thyroid disease, PID, and Polyps are associated with menstrual disorders; **Diabetes is NOT**.
- **Infrequent Periods (Oligomenorrhea):** Caused by PCOS, Hyperprolactinemia, and Premature ovarian failure; **Idiopathic hirsutism is NOT a cause**.
- **Irregular Ripening of Endometrium:** Due to a corpus luteum defect; presents as premenstrual spotting; biopsy shows patchy progestational appearance.

IV. Post-Menopausal Bleeding (PMB)

- **Rule 1:** Must exclude endometrial and cervical cancer.
- **Rule 2:** Can **never** be ignored, even on the first occasion.
- **Most Common Cause: Vaginal (atrophic) atrophy**.
- **Diagnostic Pathway:**
    1. **Transvaginal Ultrasound (TVS):** The **next best step** for a woman with PMB.
    2. If TVS shows **1mm thickness** and pale mucosa (atrophy), the management is **topical estrogen**.
    3. TVS is mandatory, but hysteroscopy is not mandatory for every patient unless indicated.

V. Diagnostic Approach

1. **History:** Detailed menstrual history, endocrine symptoms, and emotional stress.
2. **General Examination:** Check for fever, thyroid issues, hyperandrogenism (hirsutism, acne), or acanthosis nigricans.
3. **Local Examination:** Inspect the vulva, vagina, and cervix (for masses/polyps/lacerations) and palpate the uterus for size/tenderness.
4. **Investigations:**
    - **Initial: BhCG to exclude pregnancy** and CBC.
    - **Others:** Cervical smear (Pap smear is essential in AUB), Endocrine tests (Thyroid, Prolactin), and TVS.
    - **Endometrial Biopsy:** Indicated in older women (e.g., age 45+) with AUB.

VI. Management & Treatment

Treatment must be individualized based on age, parity, and severity.

Medical Management

- **NSAIDs (Mefenamic Acid):** Used for menorrhagia; specifically **reduces prostaglandin E2 production**.
- **Antifibrinolytics (Tranexamic Acid):** Used to reduce heavy flow.
- **Hormonal:**
    - **Mirena (Levonorgestrel IUS):** Often the **most suitable treatment** for women with heavy periods and a normal uterus (especially those with high BMI/smokers who cannot take estrogen).
    - **Combined Oral Contraceptive Pill (COCP):** Effective for regularizing cycles and reducing flow.
    - **Oral Progestogens:** Used for DUB. **Note: Progestogen-only pills (POP) are generally NOT listed as a primary treatment for menorrhagia** in these sources.

Surgical Management

- **Endometrial Ablation/Resection:** Options for women who have completed childbearing.
- **Hysterectomy:** A definitive surgical option.
- **Uterine Curettage:** Both diagnostic and therapeutic.

Contraceptive Methods

1. Fundamental Concepts & Efficacy

- **Contraception** is the voluntary control of fertility, with the **"ideal" method** being 100% effective, completely reversible, free of side effects, and independent of intercourse.
- **Efficacy** is measured using the **Pearl Formula**, defined as the number of pregnancies per **100 women-years** (Total pregnancies / total months of use × 1,200).
- A failure rate of 5 per 100 woman-years indicates that five pregnancies will occur in **1,200 cycles**.
- Failure rates depend on the method's mechanism and ease of use; methods like the IUS and Implanon have high effectiveness because they do not require regular user action.

2. Natural and Lactational Methods

- **Lactational Amenorrhoea Method (LAM):** Delaying fertility via breastfeeding is over 98% effective if the woman is fully breastfeeding, remains amenorrhoeic, and is within the first 6 months postpartum. Note that progestogen-only methods do **not inhibit lactation**, making them suitable for breastfeeding women.
- **Natural Family Planning:** Relies on predicting ovulation via **Basal Body Temperature (BBT)**, tracking cycle days (rhythm method), and monitoring **cervical mucus** (which becomes clear, thin, and slippery like raw egg white during ovulation).

3. Combined Hormonal Contraception (CHC)

- **Composition:** Contains both **Estrogen** (typically ethinyl estradiol) and **Progestogen** (available in four generations).
- **Mechanism of Action:** Primarily **inhibits ovulation** by suppressing the **LH surge** and inhibiting FSH to prevent follicular development.
- **Positive Health Benefits:** Regular/lighter periods, improved PMS, and long-term protection against **ovarian and endometrial cancers**. It also treats acne and reduces the formation of benign ovarian cysts.
- **Risks and Side Effects:**
    - **Cardiovascular:** Increased risk of **Venous Thromboembolism (VTE)** (highest in the first year and with 3rd generation pills and **cyproterone acetate**), and ischemic stroke. It does **not** increase the risk of hemorrhagic stroke.

    - **Malignancy:** Small increased risk of breast and cervical cancer.
    - **Common Side Effects:** Headache, fluid retention, nausea, and loss of libido. **Functional ovarian cysts** are generally *not* a side effect of CHC (unlike POCs).
- **Contraindications:** Smoking (especially if >35 years old), history of VTE/TIA, hypertension (>160/100), active liver disease, and estrogen-dependent neoplasms. Ovarian cancer is **not** a contraindication.
- **Drug Interactions:** Efficacy is reduced by **liver enzyme inducers** such as **barbiturates**, anticonvulsants, and certain antibiotics. CHC also reduces the serum level of Lamotrigine.
- **Missed Pill Management:** If one pill is missed, take it as soon as remembered and continue normally. If two or more are missed, additional protection (condoms) is needed for 7 days.

4. Progestogen-Only Contraception (POC)

- **Types:** Includes the Progestogen-Only Pill (POP), injectables (Depo-Provera), subdermal implants (Implanon), and the Intrauterine System (IUS/Mirena).
- **Mechanism:** Primarily works by **altering cervical mucus** (making it thick and hostile to sperm) and thinning the endometrium; higher doses (implants/injectables) also inhibit ovulation.
- **Advantages:** No VTE risk, minimal impact on lipids, and safe for lactating women.
- **Disadvantages:** Menstrual disturbances, weight gain, and **functional ovarian cysts**.
- **Injectables (Depo-Provera):** Associated with **amenorrhoea** (70% of users), a small reduction in bone mineral density, and a significant **delay in the return of fertility** (6–7 months).

5. Intrauterine Contraception (IUCD & IUS)

- **Copper IUCD:** Creates a sterile inflammatory reaction toxic to sperm and ova; it is a long-term (5–10 years) reversible method.
- **Hormonal IUS (Mirena):** Releases **levonorgestrel**; it is highly effective for treating **heavy menstrual bleeding** (menorrhagia) and lasts 5 years.

- **Clinical Management:**
    - **Lost Threads:** If threads are not visualized, the first diagnostic step is a **pelvic ultrasound**.
    - **Contraindications:** Suspected pregnancy, Pelvic Inflammatory Disease (PID), and unexplained vaginal bleeding. A **history of ectopic pregnancy** is **not** a contraindication.
    - **Complications: Abnormal uterine bleeding** is the most common reason for discontinuation. While the absolute risk is low, if a pregnancy occurs with an IUD, there is a higher relative chance it will be **ectopic**.

6. Barrier Methods and Emergency Contraception

- **Barrier Methods:** Male and female condoms, diaphragms, and caps used with spermicides (Nonoxynol-9). Male condoms provide the best protection against **STIs and HIV**.
- **Emergency Contraception (EC):**
    - **Copper IUD:** The most effective method; can be used up to 5 days after unprotected intercourse or predicted ovulation.
    - **Pills:** Ulipristal acetate (UPA) is effective up to 5 days; Levonorgestrel (LNG) is effective up to 72 hours.

7. Sterilisation (Permanent Methods)

- **Male Sterilisation (Vasectomy):** Involves dividing the vas deferens. It is cheaper and safer than female sterilization but **not effective immediately**. Use of other contraception must continue until **two consecutive semen analyses** show azoospermia.
- **Female Sterilisation:** Usually involves blocking fallopian tubes with **Filshie clips** or via the Pomeroy technique. Hysteroscopic methods (Essure/Adiana) use micro-inserts to induce fibrosis.
- **Regret:** Roughly 10% of couples regret sterilization, particularly those under 30 or those who chose it immediately following delivery.

Subfertility

Overview of Subfertility and Reproductive Medicine

Subfertility is defined as the inability of a couple to conceive after **12 months** of regular unprotected intercourse for women under 35, or after **6 months** for women aged 35 and older.

- **Fecundability:** The probability of achieving pregnancy in a single menstrual cycle; the normal rate is approximately **20%**.
- **Success Rates:** 85% of couples conceive within one year, and 95% within two years.
- **Classification: Primary subfertility** occurs when a woman has never achieved pregnancy; **secondary subfertility** occurs when a woman has achieved pregnancy before, regardless of the outcome.
- **Distribution of Causes:** Causes are roughly divided into **1/3 male factors, 1/3 female factors, and 1/3 combined or unexplained factors**.

Female Subfertility: Causes and Characteristics

Female factors are categorized by the anatomical or physiological system involved:

1. Ovarian Factors (Ovulatory Dysfunction)

- Includes **oligoovulation** (infrequent) and **anovulation** (absent).
- **WHO Classifications of Anovulation:**
    - **Class I:** Hypogonadotropic hypogonadal anovulation (e.g., Kallmann syndrome, anorexia nervosa).
    - **Class II:** Normogonadotropic normoestrogenic anovulation (e.g., **PCOS**, which accounts for 80-90% of anovulatory infertility).
    - **Class III:** Hypergonadotropic hypoestrogenic anovulation (e.g., Premature Ovarian Failure).
- **Premature Ovarian Failure:** Commonly caused by chemotherapy, radiation, or autoimmune disorders; however, **fertility drugs** are **not** a cause.

2. Tubal and Pelvic Factors

- Causes include **Pelvic Inflammatory Disease (PID)**, pelvic tuberculosis, and adhesions from previous surgeries.
- **Pathogens: Chlamydia** is a primary pathogen associated with bilateral tubal blockage.
- **Pseudo-obstruction:** Can be caused by mucus plugs or tubal spasms during testing.

3. Uterine and Cervical Factors

- **Uterine:** Bicornuate uterus, fibroids (submucosal or intracavitary), and intrauterine adhesions.
    - **Clinical Note:** If a **bicornuate uterus** is diagnosed, the **urinary system** must be screened for associated congenital anomalies.
- **Cervical:** Congenital malformations or injury. Viscous cervical mucus with non-motile sperm in a post-coital test suggests a need for **Intrauterine Insemination (IUI)**.

4. Endometriosis

- Causes infertility through **anatomic distortion**, damage to ovarian tissue, and the production of inflammatory substances.

Ovulation: Diagnosis and Physiology

Monitoring and diagnosing ovulation is critical for assessing female fertility:

- **LH Surge:** Ovulation typically occurs **12 hours** after the LH peak and **36h** after LH surge.
- **Signs of Ovulation:**
    - **Basal Body Temperature (BBT):** Rises the day after ovulation due to the **thermogenic effect of progesterone**.
    - **Cervical Mucus:** Becomes viscous and scanty.
    - **Endometrium:** Undergoes **secretory changes** (diagnosed via day 23 biopsy).
- **Diagnosis Methods:** Includes clinical symptoms, follicular tracing via ultrasound, and mid-luteal serum progesterone levels. **Endometrial thickness of 6.0 mm** is **not** a reliable parameter to diagnose ovulation.

Male Subfertility and Seminal Fluid Analysis (SFA)

Male infertility factors are classified as **pre-testicular** (hormonal), **testicular** (sperm production), or **post-testicular** (ductal obstruction or sexual dysfunction). **Retrograde ejaculation** is a common cause in diabetic patients.

Normal WHO Parameters for Semen Analysis:

- **Volume:** ≥ 1.5 mL.
- **pH:** ≥ 7.2.
- **Concentration:** ≥ 15 million/mL.
- **Morphology:** ≥ 4% normal forms.
- **Motility:** ≥ 32% progressive motility.
- **Liquefaction:** Should occur within 20 to 30 minutes of ejaculation.
- **Azoospermia:** The complete absence of sperm in the seminal fluid.

Evaluation and Diagnostic Investigations

The evaluation must involve **both partners** and include a complete history and physical exam.

- **Ovarian Reserve:** Best evaluated by the **Anti-Mullerian Hormone (AMH)** blood test, which is released from granulosa cells and declines with age.
- **Tubal Patency:** Often assessed via **Hysterosalpingogram (HSG)**. If HSG shows bilateral cornual blockage, the next step is **Laparoscopy and Hysteroscopy** to confirm.
- **Genetic Testing: Karyotyping** is the most important test for diagnosing **Turner Syndrome** (45 XO), which features rudimentary streak gonads.
- **Ambiguous Genitalia:** The most common cause in newborns is **Adrenal Hyperplasia**.

Hyperprolactinemia and Galactorrhea

Elevated prolactin can cause anovulatory infertility.

- **Causes:** Pituitary adenomas, hypothalamic tumors, renal failure, and medications like **phenothiazines**, alpha-methyldopa, and high-dose estrogens.
- **Note: Androgens** do **not** cause hyperprolactinemia.
- **Treatment: Bromocriptine** is used to treat galactorrhea and hyperprolactinemia, but for macro-adenomas (>10mm), the first line is medical management, while surgery is reserved for specific cases.

Ovarian Hyperstimulation Syndrome (OHSS)

OHSS is a serious complication of ovulation induction:

- **Risk Factors:** Women with a **higher ovarian reserve** (e.g., PCOS) are at an **increased risk** compared to those with lower reserve.
- **Symptoms:** Abdominal bloating and pain, nausea, vomiting, diarrhea, and reduced urine output. **Vulval swelling** is **not** a standard symptom.
- **Management:** Requires a multidisciplinary team, correction of intravascular hypovolemia, paracentesis for severe ascites, and **thromboprophylaxis**.

Treatment Strategies for Subfertility

Factor	Recommended Treatment
Ovulatory Disorders	Correct underlying causes, ovulation induction (e.g., **FSH/LH therapy** for hypogonadotropic hypogonadism), or oocyte donation.
Tubal Factors	**IVF** is the treatment of choice for bilateral blockage.
Endometriosis	Surgical intervention or IVF.
Uterine Anomalies	Surgical correction, surrogacy, or uterine transplant.
Male Factor	**ICSI** (Intracytoplasmic Sperm Injection) or sperm donation.
Unexplained	IVF.

PCOS

Overview and Prevalence

Polycystic Ovary Syndrome (PCOS) is a major public health issue with reproductive, metabolic, and psychological implications. It affects **8–13% of reproductive-aged women**, though up to 70% of cases remain undiagnosed. It is also known as **Stein-Leventhal syndrome**.

Diagnostic Criteria (Rotterdam Consensus)

To diagnose PCOS, at least **two of the following three** criteria must be met, provided other etiologies (like CAH, Cushing syndrome, or thyroid dysfunction) are excluded:

1. **Oligo/anovulation:** Irregular or absent menstrual cycles.
2. **Hyperandrogenism:** Can be **clinical** (hirsutism, male pattern alopecia, acne) or **biochemical** (raised testosterone or Free Androgen Index).
3. **Polycystic Ovaries on Ultrasound:** 12 or more small antral follicles (2–9 mm) per ovary or an ovarian volume **>10 cm^3**.

Important Diagnostic Clarifications (Past Paper Pearls):

- **BMI >30 kg/m^2 is NOT a diagnostic criterion**, even though obesity is common.
- A **raised LH/FSH ratio is NOT necessary** for a diagnosis.
- Ovaries typically show **bilateral enlargement with sclerosis**, NOT a smooth surface.

Clinical Presentation and Phenotypes

PCOS presents through various systems:

- **Reproductive:** Irregular cycles (defined by age post-menarche), subfertility, and **endometrial hyperplasia/thickening** (NOT thinning).
- **Metabolic:** Obesity, hypertension, and impaired glucose tolerance.
- **Psychological:** Anxiety, depression, and eating disorders.

NIH Phenotypes (2012):

- **A:** Androgen excess + Ovulatory dysfunction + PCO morphology.
- **B:** Androgen excess + Ovulatory dysfunction.
- **C:** Androgen excess + PCO morphology.
- **D:** Ovulatory dysfunction + PCO morphology.

Laboratory and Imaging Findings

- **Endocrinology:** Typically shows **raised fasting insulin**, raised estrone, normal FSH, and **decreased sex hormone-binding globulin (SHBG)**.
- **Best Indicator of Ovulation:** Serum **progesterone**.
- **Ultrasound Limitations:** Ultrasound is **not reliable** for diagnosis in adolescents or within **8 years of menarche**, as up to 70% of young women may have polycystic-looking ovaries naturally.

Complications and Risks

Patients with PCOS face several long-term health risks:

- **Increased Risks:** Type 2 Diabetes, **Cardiovascular disease (CVD)**, Dyslipidemia, Hypertension, **Sleep apnea**, and Endometrial cancer.
- **Misconceptions:** PCOS is **NOT** associated with an increased risk of **Breast cancer**, **Osteoporosis**, or **Type 1 Diabetes**.

Management Strategies

Management focuses on symptoms, fertility, and long-term prevention.

1. Lifestyle Modification (The Cornerstone)

- **Weight loss** is the first-line treatment.
- Losing even **5% of body weight** can restore menstrual regularity and ovulation, halve diabetes risk, and improve mental wellbeing.

2. Medical Treatment

- **Irregular Cycles:** The **Combined Oral Contraceptive Pill (COCP)** is first-line for cycle regularity and acne/hirsutism. Intermittent progestin every 3 months can be used to protect the endometrium from hyperplasia.
- **Anti-Androgens:** Used for hirsutism. Options include **Spironolactone**, **Cyproterone acetate**, **Flutamide**, and **Drospirenone**. **Finasteride** works by inhibiting the enzyme 5-alpha-reductase. *Note: Tamoxifen is NOT an anti-androgen.*
- **Metformin:** An insulin-sensitizing agent used for metabolic and hormonal outcomes. The most common cause of non-compliance is **gastrointestinal side effects**.

3. Fertility and Ovulation Induction

- **Letrozole** is now the **first-line** pharmacological treatment (superior to Clomiphene).
- **Clomiphene Citrate (Clomid):** Common side effects include **hot flushes**, mood changes, visual changes, and a risk of **multiple births** (NOT heavy periods).
- **Surgical:** Laparoscopic ovarian drilling is a second-line option.

4. Cardiovascular Risk Monitoring

- **Lipid profile:** Every 2 years if normal; annually if abnormal/overweight.
- **Blood pressure:** Annually if BMI <25; every visit if BMI >25.
- **Glucose:** Assess for prediabetes using the **oral glucose tolerance test (oGTT)**.

Lower Genital tract infections & Sexually Transmitted Diseases

I. The Normal Vaginal Ecosystem

Understanding the healthy vaginal environment is crucial for diagnosing infections.

- **Vaginal pH:** The normal range for women of reproductive age is **4.0 to 4.5**. It is slightly higher in premenarchal and postmenopausal women.
- **Primary Flora:** ***Lactobacillus acidophilus*** is the primary stabilizer. It ferments glycogen into **lactic acid**, maintaining the protective acidic environment.
- **Normal Secretions:** Clear, odorless, and floccular in consistency.
    - *High-Yield Point:* A 12-year-old with clear, odorless discharge and no symptoms likely has **physiologic discharge** related to normal pubertal development.
- **Defense Mechanisms:** Natural defenses include acidic pH, Doderlein's bacilli (*Lactobacillus*), and stratified squamous epithelium. The physical apposition of the pudendal cleft is *not* considered a primary defense mechanism against infection.

II. The "Big Three" Causes of Vaginitis

These account for the majority of vaginal infections: Bacterial Vaginosis (40–50%), Candidiasis (20–25%), and Trichomoniasis (15–20%).

Feature	Bacterial Vaginosis (BV)	Vulvovaginal Candidiasis (VVC)	Trichomoniasis
Pathogen	Overgrowth of anaerobes (e.g., *Gardnerella vaginalis*).	***Candida albicans*** (80–90% of cases).	*Trichomonas vaginalis* (flagellated protozoan).
Discharge	**Homogenous, thin, milky/grey** with a **fishy odor**.	Thick, white, **"cottage cheese"** or curd-like.	**Profuse, frothy, greenish-yellow**, foul-smelling.
Vaginal pH	**> 4.5** (usually 4.7–5.7).	**Normal (< 4.5)**.	**Elevated (> 5.0 or even > 6.5)**.
Whiff Test	**Positive** (fishy odor with 10% KOH).	Negative.	Can be positive.
Microscopy	**Clue Cells** (epithelial squamous cells covered in bacteria).	**Hyphae, pseudohyphae**, or budding yeast.	**Motile flagellated organisms** and leukocytes.
Key Sign	Often asymptomatic; no profound inflammation.	Intense **vulvar pruritus** (itching), soreness, and erythema.	**"Strawberry Cervix"** (patchy redness/petechiae).
Treatment	**Metronidazole** (oral or gel) or Clindamycin.	Vaginal imidazoles/triazoles or **Oral Fluconazole**.	**Metronidazole** or Tinidazole.
Partner RX	Not recommended; doesn't affect relapse.	Not routinely required.	**Mandatory** (it is an STD).

III. Key Diagnostic Criteria & Sequelae

Bacterial Vaginosis (BV)

- **Amsel Criteria:** (Need 3 of 4): 1. Thin, homogenous discharge; 2. pH > 4.5; 3. Positive Whiff test; 4. Clue cells (>20% of epithelial cells).
- **Important Note:** BV does **not** cause vaginal ulceration.
- **Pregnancy Risks:** Associated with **preterm labour**, mid-trimester miscarriage, rupture of membranes, and endometritis.

Vulvovaginal Candidiasis (VVC)

- **Risk Factors:** Pregnancy (commonest infection), Diabetes Mellitus, broad-spectrum antibiotics, corticosteroids, and oral contraceptive pills. Intrauterine devices are a *less* known risk factor.
- **Phases:** It is *less* common during the proliferative phase of the menstrual cycle.

IV. Viral Infections

Genital Warts (Condyloma Acuminata)

- **Cause: HPV types 6 and 11**. (Note: They are *not* caused by HSV).
- **Spread:** Highly contagious (>75%) via skin-to-skin contact.
- **Treatment:** Aims to remove visible warts (cryotherapy, laser, imiquimod, trichloroacetic acid) but **cannot eradicate the virus**.
- **Pregnancy:** Proliferate due to altered immunity. Can cause **Juvenile-onset recurrent respiratory papillomatosis (JORRP)** in the offspring. C-section may be indicated to prevent neonatal infection.

Genital Herpes (HSV)

- **Cause:** HSV-1 or HSV-2.
- **Symptoms:** Clusters of **painful vesicular lesions/ulcers**, fever, and swollen lymph nodes.
- **Treatment: Acyclovir** (400 mg 3x/day for 7–10 days).
- **Pregnancy:** High risk of neonatal transmission during primary infection. **C-section** is recommended for women with active lesions or prodromal symptoms at the time of delivery. Suppressive therapy starts at 36 weeks.

Molluscum Contagiosum

- **Cause: Pox virus**.
- **Appearance:** Small, smooth, round, **pearly lumps with a central core**.

V. Cervicitis and Other STIs

- **Diagnosis:** Finding of **purulent/mucopurulent endocervical discharge** (yellow or green).
- **Gonorrhea (*N. gonorrhoeae*):** Often asymptomatic (50%). Diagnosis via culture (Thayer Martin) or NAAT. **Treatment:** Ceftriaxone 250mg IM or Cefixime 400mg oral.
- **Chlamydia (*C. trachomatis*):** Often asymptomatic (75%). Diagnosis via **NAAT** (most sensitive). **Treatment:** Azithromycin 1g oral (single dose) or Doxycycline.
    - *High-Yield Case:* A patient with dysuria, mucopurulent cervical discharge, and negative routine urinary cultures likely has **urethritis associated with Chlamydia**.

VI. Infections in Pregnancy and General OB-GYN

- **Urinary Tract Infections (UTIs):**
    - **Asymptomatic Bacteriuria:** Defined as **>100,000 colony-forming units/mL**. It can lead to pyelonephritis if untreated.
    - **Acute Pyelonephritis:** A leading cause of **preterm labor**. Most common organism is ***E. coli***. Requires **hospitalization** and IV antibiotics.
- **Group B Streptococcus (GBS):** 20–25% of women are carriers. Screening at 28 weeks does *not* eliminate infection risk (screening is typically later). Intrapartum antibiotics reduce neonatal sepsis.
- **Postpartum Endometritis:** Foul-smelling discharge and retained products 10 days postpartum require **IV antibiotics followed by evacuation**.
- **HIV and Delivery:** Elective C-section is recommended for women **not taking HAART** to reduce vertical transmission. If on HAART with undetectable loads, the benefit of C-section is less significant.
- **Genital Tuberculosis:** Most commonly affects the **fallopian tubes** and causes infertility; it is usually *not* sexually transmitted.

Pelvic Inflammatory Disease

1. Definition and Scope

Pelvic Inflammatory Disease (PID) refers to **acute and subclinical infection** of the female **upper genital tract**. It involves the **uterine body (endometritis), fallopian tubes (salpingitis), and ovaries (oophoritis)**, and often involves neighbouring pelvic organs. It can manifest as peritonitis, perihepatitis, or a tubo-ovarian abscess.

2. Microbiology and Pathogens

- **Major Cause: *Chlamydia trachomatis*** is a primary pathogen.
- **Common Pathogens:** *Neisseria gonorrhoeae* is also frequently identified. PID is clinically considered a **mixed infection**.
- **STIs vs. Non-STIs:** Approximately **85% of cases** are caused by sexually transmitted or bacterial vaginosis-associated pathogens.
- **Rare Causes:** Fewer than **15% of cases** involve enteric pathogens (e.g., *E. coli, Bacteroides fragilis*) or respiratory pathogens.
- **Specific Groups:** In **post-menopausal women**, *E. coli* and colonic anaerobes are more likely. *Actinomycosis* is occasionally associated with indwelling **IUDs**.

3. Clinical Features and Risk Factors

- **Patients at Risk:** Sexually active females, particularly those under **25 years old**, those with multiple partners, or those with a history of PID or STDs.
- **Protective Factors:** The use of **barrier contraception** is protective.
- **Pregnancy:** PID is rare in pregnancy due to the mucus plug but can occur in the **first 12 weeks**.
- **Primary Symptoms: Bilateral lower abdominal pain** is the cardinal symptom, often of less than two weeks' duration. Pain may worsen during coitus or jarring movements.
- **Chronic Symptoms:** Chronic infection symptoms include deep dyspareunia, pelvic pain, infertility, and dysmenorrhea (**Oligomenorrhea is NOT a typical symptom**).

4. Diagnosis and Evaluation

- **Defining Characteristic: Uterine and adnexal tenderness** on **bimanual pelvic examination** is the most important finding.
- **Diagnostic Criteria:**
    - Temperature **> 38°C**.
    - WBC count of **15,000**.
    - **Cervical motion tenderness**.
    - **Note:** An **ESR of 10 mm/hr is NOT a criterion** for PID, as the rate is typically elevated.
- **Laboratory Tests:** Routine tests include a **pregnancy test** (to rule out ectopic pregnancy), NAATs for Chlamydia/Gonorrhea, and HIV/Syphilis screening. **Saline microscopy** of vaginal discharge is used to detect increased WBCs.
- **Imaging and Procedures:**
    - **Ultrasound** is the most studied imaging technique.
    - **Laparoscopy** is highly specific but has a sensitivity as low as 50%. It is useful if a patient fails to improve after 72 hours of treatment.
    - **Endometrial biopsy** is not routine due to processing delays and patchy inflammation.

5. Differential Diagnosis

PID must be distinguished from other conditions. **Common differentials** include:

- **Ectopic pregnancy** (history of missed menses, positive pregnancy test).
- **Appendicitis** (pain in the right iliac fossa, vomiting).
- **Ovarian cyst torsion or rupture** (sudden onset of severe pain).
- **Endometriosis** (cyclical or chronic pain).
- **Note: Hepatitis is NOT a differential diagnosis** for PID.

6. Complications

- **Perihepatitis (Fitz-Hugh Curtis Syndrome):** Occurs in 10% of acute PID cases. It involves inflammation of the liver capsule, causing **right upper quadrant pain** and pleuritic chest pain. Laparoscopy reveals **"violin string" adhesions**.
- **Tubo-ovarian Abscess (TOA):** An inflammatory mass involving the tube and ovary. Acute management of PID is primarily aimed at **preventing TOA**.
- **Chronic Pelvic Pain:** Common causes include chronic infection, IBS, and endometriosis (**Uterine retroversion is NOT a common cause of pelvic pain**).

7. Management and Treatment

Hospitalization Indications

Patients should be hospitalised if they are **pregnant**, have **nausea/vomiting**, have severe clinical illness (fevers/chills), or have a **suspected pelvic abscess**. **Uterine tenderness alone is NOT an indication for hospitalisation**.

Antibiotic Regimens

Treatment must cover *N. gonorrhoeae, C. trachomatis*, and various vaginal flora.

- **Outpatient Treatment:** Includes **Ceftriaxone** (IM), **Doxycycline**, and **Metronidazole**.
- **Inpatient Treatment (Severe Cases):**
    - **Second-generation Cephalosporin** (Cefoxitin or Cefotetan) intravenously PLUS **Doxycycline**.
    - **Alternative: Clindamycin** plus **Gentamicin**.
- **General Rules:**
    - The optimal duration of therapy is **14 days**.
    - Outpatients must be evaluated for clinical improvement within **72 hours**.
    - **Male sex partners** from the previous 60 days should be examined and treated.
    - **D&C (Dilatation and curettage) is NOT used** in the management of acute PID.

Fibroids

Uterine fibroids, also known as **leiomyomas** or **myomas**, are the most common benign gynaecological tumours, arising from the smooth muscle tissue of the myometrium. They are noncancerous, monoclonal growths composed of disordered myofibroblasts and extracellular matrix. Their growth is strictly **hormone-dependent**, relying on estrogen and progesterone; consequently, they often increase in size during pregnancy and typically shrink after menopause.

1. Epidemiology and Risk Factors

Fibroids affect a significant portion of the population, with an incidence of 40% to 60% by age 35 and up to **70% to 80% by age 50**. They are the leading indication for hysterectomy worldwide.

- **Increased Risk Factors:** Advancing age (peak incidence between 40–50), **African American race** (more common than in white populations), obesity, family history, high blood pressure, nulliparity, early menarche, and Vitamin D deficiency.
- **Decreased Risk Factors:** The risk of developing fibroids decreases with an increasing number of **pregnancies**.

2. FIGO Classification and Types

The International Federation of Gynecology and Obstetrics (FIGO) uses a nine-point system based on the fibroid's location relative to the uterine layers:

- **Submucosal (Types 0, 1, 2):** These protrude into the uterine cavity. **Type 0** is pedunculated and entirely intracavitary. Submucosal fibroids are the type **most commonly associated with abnormal uterine bleeding**.
- **Intramural (Types 3, 4, 5):** Located within the muscular wall. **Heavy menstrual bleeding** is the most common symptom for this type.
- **Subserosal (Types 6, 7):** Located on the outer surface. **Type 7** is pedunculated.
- **Other (Type 8):** Includes cervical fibroids or **parasitic fibroids** (pedunculated fibroids that lose their uterine attachment and gain a secondary blood supply from another organ).

3. Clinical Presentation and Complications

While 50% to 80% of fibroids are **asymptomatic**, they can cause significant morbidity when symptomatic.

- **Abnormal Uterine Bleeding (AUB):** Typically presents as **menorrhagia** (heavy or prolonged periods). They do **not** typically cause oligomenorrhea.
- **Bulk Symptoms:** Large fibroids (palpable if >12 weeks gestation size) can cause pelvic pressure, urinary frequency/urgency, **acute urinary retention**, and bowel issues like **constipation**.
- **Pain:** Can result from torsion, impaction, or degeneration.
- **Reproductive Issues:** While most women with fibroids conceive normally, they can be associated with **infertility due to impaired implantation** or recurrent pregnancy loss.
- **Systemic/Rare:** Fibroids may be associated with **polycythemia** or **hydronephrosis** due to ureteric compression.

4. Fibroid Degeneration

Degeneration occurs when a fibroid outgrows its blood supply.

- **Hyaline Degeneration:** The **most common** type of degeneration (63%).
- **Red Degeneration (Necrobiosis):** A hemorrhagic infarction most common **during pregnancy**. It presents with acute abdominal pain, low-grade fever, and uterine tenderness. It is managed **conservatively** with bed rest, IV hydration, and analgesics.
- **Other types:** Calcific, cystic, fatty, myxomatous, and necrotic.

5. Diagnosis

- **Physical Exam:** Bimanual examination may reveal an enlarged, mobile uterus with an irregular contour.
- **Imaging:**
    - **Ultrasonography:** The first-line, most widely used, and cost-effective modality.
    - **MRI:** The **most accurate** method for assessing size, number, and exact location.
    - **Sonohysterography/Hysteroscopy:** Excellent for identifying submucosal/intracavitary lesions.

6. Management Strategies

Treatment must be individualised based on symptoms, age, and desire for future fertility.

- **Expectant Management:** Appropriate for asymptomatic women.
- **Medical Therapy:**
    - **NSAIDs/Anti-prostaglandins:** Often the first step to manage heavy menstrual loss.
    - **LNG-IUS (Mirena):** Highly effective at reducing menstrual blood loss and uterine volume.
    - **GnRH Analogues (Agonists):** Induce a "menopause-like" state to **shrink fibroids (up to 50%)** within 3 months. They are useful preoperatively to reduce anemia and surgical time. However, they are not long-lasting (regrowth occurs after cessation) and can cause **osteoporosis** if used for more than 6–12 months without "add-back" therapy.
- **Surgical and Non-Excisional Therapy:**
    - **Uterine Artery Embolization (UAE):** A non-surgical option that may compromise ovarian reserve; thus, it is generally discouraged for those desiring pregnancy.
    - **Myomectomy:** Surgical removal of fibroids while preserving the uterus. **Hysteroscopic resection** is the best option for submucosal fibroids in women seeking pregnancy. Myomectomy has a 15% recurrence rate.
    - **Hysterectomy:** The **definitive treatment** for symptomatic fibroids in women who have completed childbearing.

7. Fibroids in Pregnancy

- **Complications:** Potential risks include early pregnancy loss, **preterm labor**, malpresentation, placental abruption, and postpartum hemorrhage. Notably, **placenta previa** and **oligohydramnios** are generally **not** considered standard complications of fibroids.
- **Surgical Warning:** Performing a myomectomy during a **Caesarean section is generally avoided** due to the high risk of severe, uncontrollable hemorrhage.

8. Malignancy (Leiomyosarcoma)

Malignant change occurs in only about **0.25% (1 in 400)** of cases. A high index of suspicion is required if a fibroid grows **after menopause**. Laparoscopic **morcellation** (cutting the tumor into small pieces for removal) carries a risk of spreading an undiagnosed sarcoma, which can worsen the patient's prognosis.

9. Related Condition: Adenomyosis

Adenomyosis is the presence of endometrial glands and stroma within the myometrium, leading to a **"globular," uniformly enlarged uterus**.

- **Symptoms:** Menorrhagia, dysmenorrhea, and dyspareunia.
- **Findings:** Ultrasound may show asymmetrical wall thickening and small myometrial cysts.
- **Management:** Definitive diagnosis and treatment is **total hysterectomy**.

10. Basic Anatomy: Uterine Blood Supply

- **Primary supply: Uterine arteries**, which originate from the anterior division of the **internal iliac arteries**.
- **Collateral supply:** Ovarian arteries (arising from the abdominal aorta).
- **Venous drainage:** The **right ovarian vein** drains into the inferior vena cava, while the **left ovarian vein** drains into the left renal vein.

Endometriosis and Adenomyosis

1. Core Definitions and Pathology

- **Endometriosis (Endometriosis Externa):** The presence of tissue similar to normal endometrium (glands and stroma) outside the uterine cavity.
    - **Common Sites:** The **ovaries** are the most common site. Other sites include the uterosacral ligaments, rectovaginal septum, and pelvic peritoneum.
    - **Macroscopic Appearance:** Small black dots (**powder burns**), large cystic masses (**chocolate cysts**/endometriomas), and atypical red, serous, or white plaque lesions.
    - **Microscopic Diagnosis:** Requires the presence of endometrial glands, stroma, and evidence of old hemorrhage.
- **Adenomyosis (Endometriosis Interna):** Endometrial glands and stroma located within the myometrium, surrounded by muscle fibres. It typically results in a **symmetrically enlarged uterus**, rarely exceeding 12-14 weeks in size.

2. Pathogenesis and Risk Factors

- **Theories of Endometriosis:**
    - **Implantation (Sampson's) Theory:** Retrograde menstruation.
    - **Coelomic Metaplasia:** Transformation of peritoneal cells.
    - **Dissemination:** Lymphatic and vascular spread.
    - **Genetic:** Risk is 7% if a first-degree relative is affected.
    - **Note:** Autoimmune factors are considered a theory, but monthly rupture of ovarian follicles is **not** a cause.
- **Patient Profiles:**
    - **Endometriosis:** Typically occurs in the 4th decade, often in women of **higher socio-economic groups** and those who are **nulliparous** or delayed their first pregnancy.
    - **Adenomyosis:** Characteristically seen in **multiparous** women at the end of their reproductive life.

3. Clinical Manifestations

- **The Classic Triad (Endometriosis): Dysmenorrhea** (secondary), **Deep Dyspareunia**, and occasionally **Dyschezia** (painful defecation).
- **Pain Patterns:** Pain typically occurs premenstrually, menstrually, and midcycle; **postcoital pain** is less characteristic.
- **Physical Findings (Endometriosis):** Fixed retroversion of the uterus, tender pelvic masses, adnexal enlargement, and tender **cul-de-sac nodules**.
- **Adenomyosis Symptoms:** Menorrhagia (heavy periods), secondary dysmenorrhea, and deep-thrust dyspareunia. It can also be asymptomatic.

4. Infertility Mechanisms

Endometriosis affects 40-60% of patients' fertility through several mechanisms:

- **Anatomic:** Pelvic adhesions and distortion of anatomy.
- **Biochemical:** Alteration of tubal motility by prostaglandins (PGF2) and the presence of "ovum capture inhibitor".
- **Cellular:** Increased peritoneal fluid with high concentrations of **activated macrophages**.
- **Endocrine/Ovulatory:** Luteinized Unruptured Follicle (**LUF**) syndrome and oocyte maturation defects.
- **Note:** Endometriosis does **not** typically cause infertility through anovulation.

5. Diagnosis

- **Laparoscopy:** The **gold standard** and definitive investigation for endometriosis.
- **Histology:** Provides definitive confirmation; negative histology does not entirely exclude the disease, but confirmation of at least one lesion is ideal.
- **CA-125:** While it may be elevated in endometriosis, it is **not** useful as a screening test.

6. Management Strategies

Medical Treatment

- **Analgesia:** NSAIDs are used, though evidence of effectiveness is inconclusive.
- **Hormonal Suppression:**
    - **Combined Oral Contraceptives:** Suitable for long-term use.
    - **Progestogens:** Depot Provera or the Mirena IUD (reduces associated pain).
    - **Danazol:** An androgen derivative that inhibits the midcycle FSH/LH surge; side effects include weight gain, facial hair, and irreversible deepening of the voice.
    - **GnRH Agonists:** Creates a "pseudomenopause." **Add-back therapy** should be considered at **6 months** of treatment to mitigate bone calcium loss and menopausal symptoms.
    - **Aromatase Inhibitors:** (e.g., Letrozole) Suppress local estrogen production in endometriotic tissues.
- **Note:** Medical treatment is **not effective** for improving fertility in minimal-mild disease.

Surgical and Assisted Treatment

- **Conservative Surgery:** Laparoscopic ablation or excision of lesions improves pregnancy rates in minimal-mild disease.
- **Radical Surgery:** Hysterectomy with bilateral oophorectomy; **hysterectomy** is the definitive treatment for **adenomyosis**.
- **Assisted Reproduction:**
    - **IUI:** Improves fertility in minimal-mild endometriosis.
    - **IVF:** The treatment of choice if tubal function is compromised or other treatments fail.
    - **Pre-IVF:** GnRH agonists for 3-6 months before IVF can increase clinical pregnancy rates.

Endometrial Cancer

Endometrial cancer (EC) is the **most common gynecologic malignancy worldwide**, with a lifetime risk of approximately 3%. While the average age at diagnosis is in the **early 60s**, most patients (67%) are diagnosed early at Stage I, which generally allows for better outcomes.

1. Classification: The Dualistic Model

According to the sources, endometrial cancer is categorized into two main types based on pathogenesis:

- **Type I (Endometrioid Adenocarcinoma):** This accounts for **80–90% of cases**. It is **estrogen-dependent**, typically low-grade, and often develops from a precursor lesion called atypical endometrial hyperplasia.
- **Type II (Serous or Clear Cell):** These are **not dependent on estrogen**, have no precursor lesions, and follow a much more **aggressive clinical course**.

2. Risk and Protective Factors

Understanding the factors that influence risk is essential for early identification and prevention.

- **Risk Factors (Excess Estrogen):** Type I EC is strongly linked to an environment of unopposed estrogen. Specific factors include **obesity**, Polycystic Ovary Syndrome (**PCOS**), **unopposed estrogen therapy**, early menarche, late menopause, nulliparity, and the use of **Tamoxifen**.
- **Other Risk Factors:** A family history of **Lynch syndrome** (HNPCC) carries a 40–60% risk of EC. While Diabetes Mellitus (DM) and Hypertension (HTN) are associated with EC, they are not considered causal.
- **Protective Factors:** Combined Oral Contraceptives (**COCs**) are protective due to their progesterone component. **Smoking** is also associated with a lower risk because it induces a hypo-estrogenic state.

3. Signs and Symptoms

The most hallmark sign of endometrial cancer is **postmenopausal bleeding (PMB)**; notably, while only 5–10% of women with PMB will have EC, it remains a critical red flag. Other symptoms include:

- **Premenopausal women:** Prolonged heavy menstruation or intermenstrual spotting.
- **Older women:** Abnormal vaginal discharge.
- **Advanced disease/Type II:** Pelvic pressure, pain, bloating, and early satiety.

4. Diagnostic Evaluation

Diagnosis follows a step-by-step approach utilizing imaging and sampling.

- **Imaging: Transvaginal Ultrasound (TVUS)** is the first-line method.
    - In **postmenopausal women**, an endometrial thickness **> 4mm** warrants a biopsy, while 4mm indicates low risk.
    - In **premenopausal women**, there is no single cutoff due to cycle changes.
    - **MRI** is used to assess cervical or myometrial invasion, while **CT** helps assess lymph nodes or metastatic disease.
- **Pap Test Findings:** While primarily for cervical cancer, finding **benign endometrial cells** in an asymptomatic postmenopausal woman or **atypical glandular cells** in women over 35 (or younger with risk factors) should prompt endometrial sampling.
- **Sampling:** The **Pipelle biopsy** is the preferred initial tool. If it fails to provide enough information, a **Dilation and Curettage (D&C)** is required. **Hysteroscopy** is specifically sensitive for focal lesions.
- **Laboratory Testing: CA125** is the only clinically useful marker, primarily used to monitor response to therapy in advanced disease or serous subtypes.

5. Pathology and Grading

The histologic grade, determined by architectural growth, dictates prognosis:

- **Grade 1:** Indolent; unlikely to spread outside the uterus.
- **Grade 2:** Intermediate prognosis.
- **Grade 3:** High potential for myometrial invasion and nodal metastasis.
- **Histologic Types: Endometrioid Adenocarcinoma** is the most common. **Serous Carcinoma** (5–10%) is highly aggressive with a propensity for intraperitoneal spread, resembling ovarian cancer. Other types include Clear Cell, Carcinosarcoma, and Mucinous carcinoma.

6. FIGO Staging

Staging is **surgical and pathological**. Based on the 2023 FIGO criteria:

- **Stage I:** Confined to the uterine corpus and ovary.
- **Stage II:** Invasion of the cervical stroma.
- **Stage III:** Local or regional spread (e.g., serosa, adnexa, vagina, or lymph nodes).
- **Stage IV:** Spread to the bladder/bowel mucosa or distant metastasis (e.g., lung, liver, bone).

7. Management Strategies

The primary treatment for most patients is a **Hysterectomy and Bilateral Salpingo-Oophorectomy (BSO)**.

- **Surgical Approach:** Laparoscopic or robotic approaches are standard for disease confined to the uterus, while laparotomy was the traditional standard. A **simple (Type I) hysterectomy** is usually sufficient, but **Radical hysterectomy** may be needed for cervical extension.
- **Chemotherapy:** The **TAP** regimen (Paclitaxel, Adriamycin, and Cisplatin) or a less toxic combination of **Paclitaxel and Carboplatin** are used, often combined with radiotherapy.
- **Radiotherapy: Vaginal brachytherapy** is the common adjuvant RT to reduce recurrence risk in high-risk subgroups. Primary RT is reserved for those who are poor surgical candidates.
- **Hormonal Therapy:** Since EC is hormone-responsive, **progestins** (like the LNG-IUD) can be used for non-surgical candidates or as adjuvant therapy for recurrent disease.

8. Follow-up and Prevention

- **Follow-up:** Post-surgery patients should have a pelvic exam every **3–6 months for 2 years**, then every 6–12 months for the next 3 years.
- **Prevention:** Women with **PCOS** benefit from weight loss and progestin therapy. Those with **Lynch syndrome** should undergo genetic testing, biopsies every 1–2 years starting at age 30–35, and **prophylactic hysterectomy/BSO** in their early 40s.

Cervical Cancer

1. Epidemiology and Risk Factors

Cervical cancer has seen a significant downtrend in incidence, with a current **lifetime risk of 0.7% in developed countries**, though it remains more common in developing nations.
The **average age of diagnosis is 50**. Risk factors are primarily divided into two categories:

- **HPV-related Factors:** Persistent infection with **Human Papillomavirus (HPV)** is responsible for almost all cases. Risks include early onset of sexual activity, multiple sexual partners, having a high-risk partner, a history of STIs, increasing parity, and immunosuppression.
- **Non-HPV-related Factors:** These include **cigarette smoking**, lower socioeconomic status, and the use of **combined oral contraceptives (COCs)**, which are specifically associated with adenocarcinoma.

2. Pathogenesis and Histopathology

HPV plays a central role and is detected in **99.7% of cervical cancers**. While over 40 genital types exist, **subtypes 16 and 18** are found in over 77% of all cases. The progression from initial oncogenic HPV infection of the metaplastic epithelium to persistent infection, precancer, and finally invasive carcinoma typically takes an average of **15 years**. Most women clear the infection naturally, but persistence leads to clonal epithelial progression.

The two most common histologic types are:

- **Squamous Cell Carcinoma (SCC):** Accounts for **70%** of cases, affects the ectocervix, and is mostly due to HPV.
- **Adenocarcinoma:** Accounts for **25%** of cases, affects the endocervix, is often **occult and diagnosed at advanced stages**, and is over 80% HPV-related.

3. Clinical Presentation and Diagnosis

Early cervical cancer is **frequently asymptomatic**, highlighting the importance of screening. When symptoms do occur, the most common is **irregular, heavy, or postcoital vaginal bleeding**. Some patients may experience mucoid or watery discharge. **Advanced disease** may present with back pain, pressure symptoms, lower limb edema (due to lymphatic compression), or signs of uremia and hydronephrosis (due to ureteral compression).

Diagnosis involves:

- **Physical Examination:** A pelvic and rectovaginal exam is mandatory. The cervix may look normal or display visible growth (exophytic, polypoid, or ulcerative).
- **Biopsy:** A **cervical biopsy** must be performed on any visible or symptomatic lesion; a Pap smear is not a diagnostic tool for visible lesions.
- **Investigations:** CBC (to check for anemia), creatinine (to check for obstruction), and LFTs (to check for metastasis).
- **Imaging:** Incorporating **MRI** to measure tumor size and local invasion, and **CT** to exclude metastatic disease in lymph nodes or distant organs.

4. FIGO Staging (2018)

Staging is now surgical and radiological.

- **Stage I:** Confined to the cervix.
    - **IA:** Microscopic (IA1 <3mm depth; IA2 3–5mm depth).
    - **IB:** Clinical lesions (IB1 <2cm; IB2 2–4cm; IB3 ≥4cm).
- **Stage II:** Extends beyond the uterus but not to the pelvic wall or lower third of the vagina.
    - **IIA:** No parametrial invasion.
    - **IIB:** Obvious parametrial involvement.
- **Stage III:** Extends to the pelvic wall, involves the lower third of the vagina, causes hydronephrosis, or involves **pelvic (IIIC1) or para-aortic (IIIC2) lymph nodes**.
- **Stage IV:** Extends beyond the true pelvis or involves the bladder/rectum mucosa (IVA) or distant organs (IVB).

5. Management Strategies

The choice of treatment depends on the stage and fertility desires:

- **Early Stage (IA1 to IIA1):** Surgery is generally preferred over radiation.
    - **Stage IA1 (no LVSI):** Simple hysterectomy or conization if fertility is desired.
    - **Stage IA1 (with LVSI) to IB2:** Radical hysterectomy with pelvic lymph node dissection (PLND) or **radical trachelectomy** if seeking to preserve fertility.
- **Locally Advanced (IB3 to IVA): Chemoradiation (CCRT)** is the standard. Evidence shows that concurrent **Cisplatin-based chemotherapy** and radiation improve survival compared to radiation alone.
- **Advanced/Metastatic (IVB):** Palliative chemoradiation.

6. Complications and Prevention

The most common complication of a radical hysterectomy is **urinary dysfunction** due to partial denervation of the detrusor muscle. Other risks include a shortened vagina, hemorrhage, infection, and rectovaginal fistulas.

Prevention is achieved through the **Gardasil-9 vaccine**, which protects against nine HPV types (6, 11, 16, 18, 31, 35, 45, 52, and 58).

- **Ages 9–14:** 2-dose schedule.
- **Ages 15–26:** 3-dose catch-up schedule.
- **Ages 27–45:** May be given for some benefit.

Benign Ovarian Masses

Overview and Classification of Ovarian Masses

Ovarian masses are a common finding in general gynaecology, affecting approximately **5–15% of patients**. The vast majority of these masses are **cystic** rather than solid tumours. They are generally classified into two main categories: **functional ovarian cysts**, which result from disruptions in the normal ovulation process, and **ovarian cystic neoplasms**, which represent benign neoplastic growth.

Clinical Presentation and Diagnosis

Most ovarian cysts are **asymptomatic** and are often discovered incidentally during imaging for unrelated reasons. When symptoms do occur, they typically manifest as:

- **Pain:** Chronic pressure or aching caused by the stretching of the ovarian capsule, or **dysmenorrhea**, which may suggest an endometrioma.
- **Acute Pain:** Sudden, severe pain accompanied by vomiting is a clinical red flag for **ovarian torsion**.
- **Hormonal Effects:** Some tumours may cause menstrual disruption or **virilisation**.

Initial investigations focus on rule-outs and markers:

- **-hCG:** Crucial to exclude ectopic pregnancy and serve as a marker for certain germ cell tumours.
- **Tumour Markers: CA125** is used for epithelial tumours, **CEA and CA19-9** for mucinous types, **AFP** for endodermal sinus tumours, **Inhibin A and B** for granulosa cell tumours, and **LDH** for dysgerminomas.
- **Imaging: Sonography** is the first-line tool, typically using both transabdominal and transvaginal approaches to ensure large masses are not missed. Features concerning for malignancy include **thick septa, increased vascularity, papillary growths, and solid components**. **CT scans** are reserved for cases where malignancy is suspected to check for metastasis or ascites.

Functional Ovarian Cysts

These cysts originate from ovarian follicles during maturation or after ovulation.

- **Follicular Cysts:** Caused by hormonal dysfunction before ovulation, leading to fluid expansion in the follicle.
- **Corpus Luteal Cysts:** Formed when excessive haemorrhage occurs within the follicle centre after ovulation.
- **Theca Lutein Cysts:** These are uncommon, typically **bilateral**, and multiple. They are triggered by **elevated -hCG or LH levels**, often associated with gestational trophoblastic disease (GTD), multiple gestations, or fertility treatments.
- **Management:** Most functional cysts **regress within 6 months**; while high-dose oral contraceptives (OCPs) can prevent new ones, they do not speed up the resolution of existing cysts.

Benign Neoplastic Cysts

Neoplastic cysts are categorized by their cell type of origin:

1. **Epithelial Tumours:**
    - **Serous Cystadenomas:** Typically thin-walled, unilocular, and filled with serous fluid; they are bilateral in 20% of cases.
    - **Mucinous Cystadenomas:** Generally thicker-walled and contain mucoid fluid.
2. **Germ Cell Tumours (Teratomas):**
    - **Mature Cystic Teratoma (Dermoid Cyst):** These account for **60% of benign ovarian neoplasms**. They arise from a single germ cell and can contain tissues from all three layers (ectoderm, endoderm, and mesoderm), though **ectodermal tissues** (like hair and sebaceous glands) usually predominate.
    - A characteristic internal growth known as a **Rokitansky protuberance** is often present.
    - On ultrasound, they are identified by **fat-fluid or hair-fluid levels** and hyperechoic nodules.

Management and Surgical Principles

Management strategies depend on the risk of malignancy and the severity of symptoms:

- **Expectant Management:** Reasonable for asymptomatic functional cysts, as many resolve spontaneously.
- **Surgical Intervention:** Indicated if the mass is **larger than 7 cm**, symptomatic, or shows concerning features on imaging or elevated CA125.
- **Procedure Selection:** The choice between **cystectomy** (removing only the cyst) and **oophorectomy** (removing the entire ovary) depends on the patient's age, menopausal status, and intraoperative findings. **Laparoscopy** is the preferred approach for benign-appearing masses.
- **Key Precautions: Cyst aspiration is avoided** to prevent the "seeding" of potentially malignant cells. If a dermoid cyst spills during surgery, **intrabdominal irrigation** is vital to prevent chemical peritonitis.

Ovarian Carcinoma

Overview and Epidemiology

Ovarian cancer affects approximately **1 in 78 women (1.3%)** during their lifetime. **Epithelial ovarian carcinomas** comprise **90%** of cases, including more indolent borderline types. The average age at diagnosis is in the **early 60s**, and the overall 5-year survival rate remains relatively low at **45%**.

Risk and Protective Factors

Understanding these factors is key to identifying at-risk patients:

- **Risk Factors:** Increasing age, **family history** of breast or ovarian cancer, personal history of breast cancer, **nulliparity**, early menarche, late menopause, and pelvic inflammatory disease (PID).
- **Protective Factors:** Use of **oral contraceptive pills (OCPs)**, tubal ligation, and hysterectomy.

Clinical Presentation

While often called a "silent killer," most patients experience symptoms even in early stages:

- **Common Symptoms:** Bloating, **increased abdominal size**, urinary urgency, pelvic pain, constipation, fatigue, and loss of appetite.
- **Physical Findings:** A palpable **solid, nodular, and fixed** mass is common; however, very large masses are paradoxically often benign or borderline.
- **Advanced Signs: Ascites** (abdominal fluid wave), **omental caking** (upper abdominal mass), pleural effusion, and rarely, the **Sister Mary Joseph sign** (umbilical nodule).

Diagnostic Testing

A combination of blood work and imaging is used for diagnosis and management planning:

- **Blood Tests: CA125** is elevated in most patients but can yield false negatives in Stage I or false positives in conditions like PID. Other markers include **HE4**, and **CA19.9/CEA** for mucinous tumors. Some patients may show **thrombocytosis** or **hyponatremia** (SIADH).
- **Imaging: Transvaginal sonography** is the primary tool for differentiating benign from malignant masses (malignant signs include size **>5 cm**, thick septa, and solid components). **CT scans** are vital for planning surgical management.

Pathology and Spread

- **Histologic Types: Serous** is the most common (>50%) and often features psammoma bodies. Other types include **Endometrioid** (15-20%), **Mucinous** (5-10%), and **Clear Cell** (5-10%, characterized by hobnail cells).
- **Metastasis:** The primary mode of spread is **exfoliation** into the peritoneal cavity. It also spreads via lymphatic dissemination, direct extension to nearby organs, and occasionally hematogenous spread to the liver, brain, or lungs. **Krukenberg tumors** are a specific type of metastatic mucinous cancer originating from the GI tract, usually the stomach.

Staging and Management

Ovarian cancer is **surgically staged**, though only **one-third** of cases are caught in Stage I or II.

- **Early Stage:** Treatment involves **extrafascial hysterectomy with bilateral salpingo-oophorectomy (BSO)**, omental biopsy, and platinum-based chemotherapy depending on the stage.
- **Advanced Stage:** Focuses on **cytoreductive (debulking) surgery** to remove all gross disease, followed by six courses of **platinum-based chemotherapy**. Surgery is considered "optimal" if residual disease is **less than 1 cm**.
- **Follow-up:** Patients require a pelvic exam and CA125 test every **2–4 months for the first two years**.

Prognosis and Prevention

- **Favorable Prognosis:** Linked to **younger age**, early-stage disease, well-differentiated tumors, and **minimal residual disease** after surgery. Mucinous and clear cell types generally have a poorer prognosis.
- **Prevention:** There is no general screening method for the public. For high-risk **BRCA1/2 carriers**, **prophylactic BSO** is the only proven prevention method, typically recommended between ages **35–45**.

Pelvic Organ Prolapse

I. Anatomy of Pelvic Support

The normal support of the uterus and vagina relies on the interaction between pelvic muscles and connective tissue.

- **Primary Muscle Support:** The **Levator Ani muscle group** provides the main support for the pelvic floor, creating a firm elastic base. This group includes the **pubococcygeus, iliococcygeus, and puborectalis muscles**. The ischiococcygeus is also part of the pelvic diaphragm, but the **obturator internus is NOT**.
- **Connective Tissue & Ligaments:**
    - **Cardinal Ligament:** Recognized as the **main ligament** supporting the uterus.
    - **Uterosacral Ligament:** Provides apical support.
    - **Pubocervical Ligament/Fascia:** Supports the anterior compartment.
    - **Rectovaginal Fascia/Septum:** The structure where a defect leads to a **rectocele**.
    - **Ligaments NOT providing uterine support:** The **ovarian ligament** does not support the uterus (the round, uterosacral, and cardinal ligaments do).
- **Levels of Vaginal Support:**
    - **Level I (Apical):** Cardinals and Uterosacrals suspend the vaginal apex.
    - **Level II (Lateral):** Arcus tendineus (white line) and fascia overlying the levator ani.
    - **Level III (Distal):** Perineal membrane and perineal body.

II. Pelvic Organ Prolapse (POP): Definitions & Types

POP is a common gynaecological problem, though it is **rare in nulliparous women** and more common after menopause. Surgical correction is **not needed in all cases**.

- **Anterior Wall Prolapse (Cystocele):** Pathologic descent of the bladder base into the vagina. It is common after menopause and may lead to URIs, but it is **not the primary cause of stress incontinence** (though often associated).
- **Apical Prolapse:** Involves the uterus or vaginal vault (post-hysterectomy) due to damage to the **uterosacral-cardinal ligament complex**.
- **Posterior Wall Prolapse:**
    - **Rectocele:** A defect in the rectovaginal septum.
    - **Enterocele:** A hernia where the peritoneum contacts the vaginal mucosa, often occurring when endopelvic fascia is absent. risk increases with colposuspension.
- **Procidentia:** Defined as **Stage 4 prolapse**, representing complete eversion of the vagina.

III. Etiology and Risk Factors

- **Parity:** The **strongest risk factor** for developing prolapse.
- **Increased Abdominal Pressure:** Chronic cough, heavy lifting, obesity, and straining due to **constipation** (61% of women with chronic straining develop POP).
- **Surgery:** Total hysterectomy carries an 11.6% risk of subsequent prolapse.
- **Collagen Abnormalities:** Women with prolapse often have a higher proportion of **Type III collagen** (weaker/flexible) compared to Type I, and may exhibit joint hypermobility.
- **Other Factors:** Age, menopause, and smoking. In young patients (e.g., 29 years old), a history of hernias may suggest an underlying **collagen deficiency**.

IV. Clinical Presentation and Symptoms

- **General:** Bulge, heaviness, dragging sensation, backache, and vaginal irritation.
- **Urinary:** Frequency, urgency, and stress incontinence.
- **Bowel (Rectocele):** Constipation, incomplete bowel emptying, and the need to manually reduce the prolapse to defecate.
- **Obstetric Trauma:** The **most likely cause of fecal incontinence** in otherwise healthy middle-aged women.

V. Staging and Grading Systems

Traditional Grading

- **1st Degree:** Prolapse reaches halfway to the hymen.
- **2nd Degree:** Prolapse reaches the level of the hymen.
- **3rd Degree:** Prolapse extends beyond the hymen.

Standardized POP-Q System

- **Stage 0:** No prolapse.
- **Stage 1:** Most distal portion is **>1 cm above** the hymen.
- **Stage 2:** Most distal portion is **between 1 cm above and 1 cm below** the hymen.
- **Stage 3:** Most distal portion is **>1 cm below** the hymen but remains 2 cm less than the total vaginal length.
- **Stage 4:** Complete eversion; the most distal portion is at least (total vaginal length - 2) cm.

VI. Key Clinical and Anatomical Facts for Exams

- **Diagonal Conjugate Diameter:** Should be at least **12 cm** in a normal female pelvis.
- **Pelvic Cavity:** The mid-cavity is bounded posteriorly by the **junction of S2 and S3** (it is NOT L4-S1).
- **Uterine Retroversion:** This occurs in **20% of normal women**.
- **Anatomical Relations:**
    - The **right ureter** is in close proximity to the **infundibulo-pelvic ligament**.
    - The ureter is **NOT** an intraperitoneal structure.
    - The middle portion of the Fallopian tube is the **ampulla**.
    - The ovary is attached to the uterus by the **ovarian ligament**, not the round ligament.

Urinary Incontinence

1. Epidemiology and Definitions

- **Prevalence:** Approximately 13% of the general population is affected by Stress Urinary Incontinence (SUI). In Jordanian women aged 50–65, one-third suffer from urinary incontinence (UI), with 23.1% having stress UI, 26.4% having urge UI, and 18.1% having a mixed type.
- **Stress Urinary Incontinence (SUI):** The involuntary loss of urine during activities that increase abdominal pressure, such as coughing, laughing, or sneezing. It is often caused by laxity of the **pubourethral ligament**.
- **Urge Urinary Incontinence (UUI):** Involuntary urine loss associated with an abrupt, strong desire to void (urgency).
- **Overactive Bladder (OAB):** A symptom-based condition characterised by increased frequency and urgency, with or without urge incontinence. It is also associated with **nocturia**.
- **Overflow Incontinence:** Involuntary loss that occurs when the bladder overfills and never empties completely. Note that this is **not** a characteristic of OAB syndrome.
- **Detrusor Overactivity (DO):** A urodynamic observation of involuntary detrusor contractions during the filling phase. The most common cause is **idiopathic**.

2. Risk Factors and Pathophysiology

- **Major Risk Factors for SUI:** The **most important risk factor** is the use of **forceps during delivery**. Other promoting factors include obesity, chronic cough/smoking, chronic constipation, menopause, and previous pelvic surgery.
- **Mechanism of Voiding:** Urine is expelled from the bladder through the **contraction of the detrusor smooth muscle** and the **relaxation of the bladder neck**.
- **Pregnancy:** Urinary frequency in early pregnancy is most commonly caused by **decreased renal tubular reabsorption of water** and increased GFR, rather than just mechanical pressure.
- **Estrogen:** Low estrogen levels (menopause) decrease urethral resting pressure, facilitating leakage.

3. Clinical Evaluation

- **Initial Assessment:** History alone is **insufficient** to diagnose Genuine Stress Incontinence (GSI). For a 50-year-old woman presenting with UI, **urodynamic studies** should be carried out to confirm the diagnosis.
- **Bladder Diary:** This is an objective three-day record of food/fluid intake, output, and leakage incidents. It **does not** require hourly documentation of weight.
- **Urodynamic Observations:** A normal flow curve is smooth with a Qmax >15 ml/s. **Detrusor overactivity** shows an abnormally high flow rate with increased voiding pressure, while **outlet obstruction** presents as a long, flat plateau with low flow.

4. Conservative and Lifestyle Management

- **First-Line Therapy:** Pelvic Floor Muscle Training (PFMT) should be offered as the first-line therapy for all patients with mixed or urge incontinence.
- **Weight Reduction:** Obesity is an independent risk factor. A **5–10% weight loss** is effective in reducing symptoms for both SUI and OAB in moderately obese women.
- **Caffeine and Tea:** High caffeine intake is an independent risk factor for detrusor overactivity. Tea drinking (specifically) is epidemiologically associated with all forms of incontinence.
- **Bladder Drill:** This involves setting target voiding times and using a fluid balance chart to retrain the bladder; it should be considered for all OAB patients.

5. Pharmacotherapy (Antimuscarinics)

- **Mechanism:** Antimuscarinics (anticholinergics) reduce intra-vesical pressure, increase bladder compliance, and raise the volume threshold for micturition. They are **not used for SUI** because they have no effect on the urethral sphincter.

- **Common Medications:** Oxybutynin, Darifenacin, Tolterodine, and Solifenacin. **Mirabegron** is used for OAB but is **not** an anti-muscarinic (it is a beta-3 agonist).

- **Contraindications:** Antimuscarinics are **contraindicated** in patients with:
    - Closed-angle glaucoma.
    - Myasthenia gravis.
    - Ulcerative colitis.
    - Urinary retention.
    - *Note: They are generally **not** contraindicated in thrombophilias or liver disease*.
- **Side Effects:** Common reasons for stopping therapy include dry mouth, constipation, blurred vision, somnolence, and insufficient efficacy.

6. Surgical Management

- **Burch Colposuspension:** Historically a gold standard with high cure rates (~84%), but largely replaced by minimally invasive tapes unless laparotomy is required for other reasons.
- **Mid-Urethral Slings (TVT and TOT):**
    - **TVT (Tension-free Vaginal Tape):** Ideal for stress incontinence, with an 80% cure rate. It can be performed under local, regional, or general anaesthetic.
    - **TOT (Transobturator Tape):** The tape runs through the obturator foramina to support the mid-urethra in a "hammock" fashion. Bladder perforation is unlikely, so cystoscopy is sometimes considered unnecessary.
- **Complications of TVT:** These include postoperative voiding dysfunction, retropubic bleeding/hematoma, bladder perforation, and vaginal erosion. **Ureteric injury** is **not** a typical complication of TVT.
- **Urethral Bulking Agents:** These are expensive and have a transient cure rate, often falling to 50% after two years.

Benign and malignant conditions of the vulva and vagina

1. Vulvovaginal Anatomy

- **Location:** Between genitocrural folds (lateral), mons pubis (anterior), and anus (posterior).
- **Components:** Labia majora/minora, clitoris, vestibule, urinary meatus, vaginal orifice, hymen, Bartholin glands, and Skene ducts.
- **Key Vascular Supply:** The **pudendal artery** is the main blood supply to the vulva.
- **Anatomical Notes:**
    - The **Mons Veneris** is the fatty tissue pad covering the pubic bone.
    - The **clitoris** is highly sexually sensitive spongy tissue.
    - The **hymen** is a membrane partially covering the vaginal opening.

2. Benign Non-Neoplastic Epithelial Disorders

These are common causes for primary care visits and include the following "Lichen" conditions:

Lichen Simplex Chronicus (Squamous Cell Hyperplasia)

- **Cause:** Local thickening of the epithelium due to **prolonged itching**.
- **Presentation:** Pain and itching; white plaques or dark red areas with a **leathery raised surface**.
- **Treatment:** Intermediate potency topical corticosteroids.

Lichen Sclerosus (LS)

- **Profile:** Most common in the anogenital area of **midlife women**.
- **Symptoms:** Intense pruritus (most common), dyspareunia, burning pain, and painful bleeding fissures.
- **Signs:** Thin, white, inelastic skin with a **"tissue paper" appearance**; shrinkage of labia minora and a buried clitoris.
- **Diagnosis:** Skin biopsy showing loss of rete ridges and atrophic epithelium.
- **Management: High/ultrapotent topical corticosteroids**. **Surgical excision is NOT the main treatment**.

- **Pathology:** Heat shock protein-70 (HSP-70) is expressed more often in LS than in healthy controls.
- **Risk:** These women are at an **increased risk of vulval cancer**. LS is **not** an ulcerating lesion.

Lichen Planus

- **Type:** Inflammatory autoimmune process involving the vulva, vagina, and/or mouth.
- **Signs:** Erythema and erosions on the vulva surrounded by **white striae**.
- **Vaginal Involvement:** Causes pain and, if untreated, **vaginal stenosis** (narrowing). Treatment includes steroids and vaginal trainers (dilators).
- **Past Paper Tip:** Itching white plaques not responsive to antifungals suggest **Lichen Sclerosus** over Lichen Planus or Candidiasis.

3. Benign Cysts and Solid Masses

1. **Epidermal Inclusion Cysts:** Form in obstructed hair follicles; non-tender, mobile, and contain sebum/epithelium.
2. **Bartholin Cysts:** Located below the hymenal ring at **4 or 8 o'clock**. Ducts open into the **posterior vaginal vestibule**. Treated with marsupialization or I&D if symptomatic.
3. **Skene's Duct Cysts:** Located in the paraurethral area; can cause urinary obstruction or dyspareunia.
4. **Genital Warts (Condylomata):** Caused by **HPV 6 and 11**; usually found at the posterior fourchette and lateral walls.
5. **Hidradenitis Suppurativa:** Chronic inflammation of the apocrine glands.
6. **Urethral Caruncle:** Small, red, benign fleshy outgrowth at the urethral meatus.

4. Premalignant Lesions (VIN and Paget's)

- **Vulval Intraepithelial Neoplasia (VIN):**
    - **Squamous VIN (Bowen's Disease/VIN 3):** Mean age is **45 years** (Note: "40 years" is considered false in past papers). 50% are asymptomatic; **itching** is the most common symptom. Lesions are elevated and varied in color (white, red, pink, brown, grey).
    - **Non-squamous VIN:** Includes Paget's disease and noninvasive melanocyte tumors.

- **Paget's Disease of the Vulva:**
    - **Profile:** Elderly, white postmenopausal women.
    - **Presentation:** Well-demarcated eczematous lesions with white plaques.
    - **Pathology:** Characteristic large, pale **"Paget's cells"** in the epidermis.
    - **Associated Risk: 20% are associated with an underlying adenocarcinoma**.

5. Vulval Malignancies

Represent only 5% of gynecological malignancies.

Squamous Cell Carcinoma (SCC)

- **Frequency:** Constitutes **90%** of all vulval cancers.
- **Age Distribution:** Notably follows a **bimodal age distribution**.
- **Types:**
    1. **More common:** Older women, related to long-standing **Lichen Sclerosus**.
    2. **Less common:** Younger women, related to **HPV infection**.
- **Presentation: Pruritus (most common)**, vulval lump, or ulcer.
- **Spread:** Commonly spreads to the **vagina, urethra, or anus**; 30% have inguinal lymph node metastasis.
- **Risk Factors:** Smoking, HPV, Lichen Sclerosus, and cervical cancer. **Molluscum contagiosum is NOT a risk factor**.

Malignant Melanoma

- **Frequency:** 2nd most common vulvar cancer.
- **Profile:** Postmenopausal white women; involves labia minora or clitoris.
- **Prognosis:** Correlates to the **depth of penetration** into the dermis (30% 5-year survival).

6. FIGO Staging and Management (Vulva)

Stage	Description
Stage I	Tumor limited to vulva/perineum, 2 cm, no nodes.
Stage IA	Stromal invasion 1 mm.
Stage IB	Stromal invasion > 1 mm.
Stage II	Tumor limited to vulva/perineum, **> 2 cm**, no nodes. Includes spread to lower 1/3 of vagina.
Stage III	Adjacent spread (urethra/vagina/anus) OR **unilateral** regional node metastasis.
Stage IV	Invades upper urethra, bladder/rectal mucosa, pelvic bone, or **bilateral** nodes (IVA); Distant metastasis (IVB).

Management Summary:

- **Stage IA:** Radical local excision (1 cm margins); **no groin dissection** needed.
- **Stage IB & II:** Radical local excision + **ipsilateral** inguinal/femoral lymphadenectomy (if lesion is unilateral).
- **Stage III:** Radical vulvectomy + bilateral groin dissection. Preoperative radiation/chemo-radiation may be used to shrink tumors.
- **Prognosis:** Significantly correlates with **Lymph Node (LN) status**. Negative nodes = 90% survival; Positive nodes = 50%.

7. Diseases of the Vagina

- **Infections:** Bacterial vaginosis, Candida (fungal), and Trichomonas (protozoal) cause inflammation/discharge.
- **Erosive Lichen Planus:** Causes pain and narrowing; treated with trainers and steroids. Note: **Lichen Sclerosus does NOT affect the vagina**.
- **Vaginal Intraepithelial Neoplasia (VAIN):** Usually an extension of **Cervical Intraepithelial Neoplasia (CIN)** and shares the same risk factors. HPV plays a **major role** in its pathogenesis.
- **Vaginal Cancer:**
    - **General:** Rare; cause unknown. HPV is a risk factor.
    - **Presentation:** Often advanced stage; symptoms include bleeding, discharge, and pain (if nerves are infiltrated).
    - **Management:** Surgery is rarely an option due to advanced state; **Radiotherapy and chemotherapy** are first-line.
    - **Complications:** Advanced stages may develop **rectovaginal or vesicovaginal fistulae**.
- **Cervical Erosion Fact:** They are **benign** and often asymptomatic but **covered by columnar epithelium** (the past paper says "covered by squamous" is false).