# Gynecology Final

🔊 **Podcast Style Review (Experimental Feature)**

attachment:1b8fab64-c361-4a00-8599-5c83dd01eab3:Gynecology_Final_Dossier_Key_Concepts.m4a

👨‍⚕️ **Made by: Ibrahim Al-Khatib**

- Topics are arranged in order of most to least commonly tested
- Check the table of contents below for easier navigation
- Good luck 🍀



## Gynecological Malignancies

### Ovarian Cancer

- **Risk Factors:**
  - Low parity
  - Early menarche and late menopause
  - High body mass index (BMI)
  - Fertility drugs (potential association)
  - Estrogen replacement therapy
  - Family history of breast/ovarian cancer
  - Factors NOT associated: Multiparity, use of combined oral contraceptives (COCPs are protective).
- **Symptoms & Presentation (Ovarian Cancer Symptom Index - OCSI):**
  - Pelvic or abdominal pain
  - Urinary frequency or urgency
  - Increased abdominal size or bloating
  - Difficulty eating or feeling full quickly
  - Symptoms NOT typically part of OCSI: Abnormal uterine bleeding, elevation of tumor markers (marker elevation is a *finding*, not a symptom).
  - Uncommon presentation: Chest pain. Common: Abdominal pain, weight loss, abdominal distention, shortness of breath.
- **Epithelial Ovarian Cancer:**
  - **Types & Resemblance:**
    - Serous: Resembles fallopian tube epithelium. Psammoma bodies may be present in serous tumors. High-grade serous tumors are common in Type II.
    - Mucinous: Resembles endocervical glands. Can be associated with Pseudomyxoma peritonei.
    - Endometrioid: Resembles proliferative endometrium.
  - **Type I vs Type II:**
    - Type I: Lower grade, often arise from borderline tumors or endometriosis, associated with specific mutations (e.g., KRAS, BRAF), often present at an early stage, have multistep pathway, develop less rapidly.
    - Type II: High grade, develop rapidly, present at advanced stage, more common, arise from ovarian surface epithelium/mullerian inclusions (or fallopian tube), associated with p53 mutations. Accounts for most ovarian cancer deaths.
  - **Tumor Markers:**
    - CA-125: Useful marker, but elevated in many benign conditions (menstruation, PID, fibroids, endometriosis, pregnancy, cirrhosis) and some non-ovarian cancers (pancreatic, lung). NOT useful for screening general population. Elevated level is NOT part of OCSI parameters for initial symptom check.
    - Human epididymis protein 4 (HE4).
    - CA 19.9.
    - CA 15.3 (Breast cancer marker, less specific for ovarian).
    - Alpha-fetoprotein (AFP) is NOT typically useful for epithelial ovarian cancer (more for germ cell tumors).
  - **Staging & Prognosis:**
    - Formal surgical staging is crucial.
    - Low-risk criteria (for preserving fertility/less aggressive treatment): Intact capsule, no surface excrescences, negative washings, diploid tumor. Ascites indicates higher risk/stage. Dense adhesions and surface excrescences are high-risk features.
    - Bilateral ovarian conservation may be considered in specific early-stage, low-grade tumors (e.g., early non-endometrioid adenocarcinoma) in young patients desiring fertility.
    - Prognostic factors: Stage of the disease (most important), histologic grade, histologic type, nuclear grade, vascular space invasion, patient age, residual tumor size after debulking. Age of the patient is the least

important among major factors.
    - Omental cake on CT suggests advanced malignancy.
- **Germ Cell Tumors:**
    - Dysgerminoma: An epithelial tumor type.
    - Mature cystic teratomas (Dermoid cysts): Usually benign, rarely malignant. Bilaterality occurs, though rates vary in literature. Treatment is often cystectomy (laparoscopy preferred over laparotomy if possible), not necessarily oophorectomy unless indicated.
- **Screening:**
    - No effective screening method for the general population.
    - Routine annual pelvic exams, CA-125, and transvaginal ultrasound are NOT validated screening tools for low-risk individuals but may be used for high-risk or symptomatic patients. The best practical method for evaluating symptomatic patients is debated, often involving symptom index awareness and appropriate workup (e.g., TVUS/Transabdominal US + CA-125, not routine exams or CT scans alone).

## Endometrial Cancer

- **Risk Factors:**
    - Obesity (most common risk factor)
    - Nulliparity (risk is higher compared to women with children)
    - Late menopause (and early menarche)
    - Unopposed estrogen therapy (Estrogen only HRT)
    - Tamoxifen therapy
    - Polycystic ovary syndrome (PCOS)
    - Diabetes mellitus
    - History of anovulation
    - Lynch syndrome (HNPCC)
    - Factors NOT increasing risk: Smoking, COCP use (protective), combined HRT, multiparity, early menopause (Late menarche is also not a risk factor).
- **Symptoms & Diagnosis:**
    - Postmenopausal bleeding (most common presenting symptom).
    - Abnormal uterine bleeding in premenopausal women.
    - Diagnosis often involves endometrial biopsy or D&C. Transvaginal ultrasound showing thickened endometrium (>4-5 mm in PMB) warrants investigation. An endometrial thickness of 10mm in a postmenopausal lady is highly suspicious.
    - Hysteroscopy can aid diagnosis.
    - CT scanning may be less important for initial workup compared to biopsy/US.
- **Pathology & Staging:**
    - Type I: Estrogen-dependent, often arise from hyperplasia, typically endometrioid adenocarcinoma, lower grade, better prognosis (~85% 5-yr survival), associated with risk factors like obesity, diabetes.
    - Type II: Estrogen-independent, arise in atrophic endometrium, higher grade (e.g., papillary serous, clear cell), poorer prognosis, not typically associated with hyperplasia.
    - Staging is surgical. Includes TAH+BSO, pelvic/para-aortic lymphadenectomy, peritoneal washings.
    - Surgical staging is recommended for: Grade 3 lesions, large Grade 2 tumors, clear cell/papillary serous types, known ovarian mets, deep myometrial invasion. It may NOT be required for Grade 1 tumors confined to endometrium without adverse features.
    - Stage IB involves invasion <50% of myometrium. Stage II involves cervical stromal invasion (involvement of the glandular component of the cervix places the stage at least at Stage II).

- Prognostic factors: Stage (most important), histologic grade, depth of myometrial invasion, lymphovascular space invasion, histologic type, nuclear grade.

- **Treatment:**
  - Total Abdominal Hysterectomy and Bilateral Salpingo-oophorectomy (TAH-BSO) is the cornerstone.
  - Adjuvant therapy (e.g., radiotherapy) depends on stage and risk factors. For tumor limited to endometrium/endocervical glands (early stage, low risk), TAH-BSO alone may suffice (Vault radiotherapy/chemo not needed).

## Cervical Cancer

- **Risk Factors:**
  - Human Papillomavirus (HPV) infection (primary cause). HPV 16 & 18 are most common high-risk types. HPV 18 more associated with adenocarcinoma.
  - Early age at first coitus.
  - Multiple sexual partners.
  - High parity (association with adenocarcinoma noted in some resources).
  - Smoking.
  - Immunocompromised status.
  - Lower socioeconomic status.
  - Factors NOT directly related or proven: Frequency of sexual intercourse, alcohol consumption. Using Mirena coil is NOT a risk factor.
- **Screening & Diagnosis:**
  - Pap smear (Cervical cytology) is a screening test for pre-cancerous lesions (CIN) and early cancer. A negative smear does NOT completely exclude cancer.
  - Colposcopy with directed biopsy is used to evaluate abnormal Pap smears.
  - Conization may be needed for diagnosis (if biopsy inconclusive) or treatment (for CIN).
- **Pathology & Staging:**
  - Squamous cell carcinoma is most common type. Adenocarcinoma frequency increasing, especially in younger women.
  - Cervical Intraepithelial Neoplasia (CIN) is the precursor lesion. CIN III is carcinoma in situ. Definite diagnosis of CIN III requires histopathology (biopsy/colposcopy).
  - Staging is primarily clinical (FIGO staging).
  - Stage Ia requires microscopic diagnosis. Stage Ia management might involve conization or simple hysterectomy (sometimes with ovarian conservation options depending on specifics).
- **Cervical Ectropion (Erosion):**
  - Columnar epithelium present on ectocervix.
  - Often asymptomatic, can cause post-coital bleeding.
  - Benign condition. It involves columnar epithelium, not squamous epithelium, on the ectocervix.
  - Can be treated by cryotherapy if symptomatic.

## Vulvar Cancer

- **Presentation:**
  - Pruritus (most common symptom).
  - Vulvar bleeding, discharge, pain, palpable lesion.
- **Risk Factors:**
  - HPV infection (especially for VIN-related cancers).
  - Lichen sclerosus (associated with differentiated VIN/non-HPV related cancers).

    - Smoking.
    - Immunodeficiency.
- **Diagnosis & Staging:**
    - Biopsy is required for diagnosis.
    - Staging is surgical (FIGO). Example: A 3cm lesion on the labia majora with 1cm vaginal affection and no nodal involvement would be Stage II.
- **Treatment:**
    - Historically, en bloc radical vulvectomy with bilateral groin node dissection was standard.
    - Current trend towards less radical surgery: Wide local excision or radical vulvectomy with separate groin node dissection (often sentinel node biopsy first).
    - Modification towards less radical surgery is due to minimizing morbidity (lymphedema, psychosexual issues), earlier detection of smaller tumors. The need for post-op radiotherapy is *considered* based on findings, not the primary reason for less radical surgery itself. Long-term hospitalization is a potential consequence, not a driver for modification.
    - For a localized, moderately differentiated squamous cell carcinoma, radical vulvectomy + ipsilateral inguinofemoral lymphadenectomy is appropriate treatment.

## Gestational Trophoblastic Neoplasia (GTN)

- **Molar Pregnancy (Hydatidiform Mole):**
    - **Complete Mole:** Diploid, entirely paternal origin (46XX most common, 46XY possible via dispermy). No fetal tissue. "Snowstorm" appearance on ultrasound is characteristic but definitive diagnosis often requires histology. Higher risk of malignant GTN (15-20%). Karyotype is typically 46XX or 46XY.
    - **Partial Mole:** Triploid (e.g., 69XXY, 69XXX). Fetal tissue often present. Focal trophoblastic hyperplasia and villous swelling. Lower risk of malignant GTN (<5%). Chromosomal pattern is triploid, not diploid. Diagnosed by histology, often suspected after miscarriage. Uterine size may or may not be large.
    - **Risk Factors:** Extremes of maternal age (<20 or >40).
    - **Presentation:** Vaginal bleeding (most common), uterine size large for dates (more common in complete mole), hyperemesis gravidarum, early pre-eclampsia (<20 weeks), hyperthyroidism. Nervousness, anorexia, and tremors are less specific symptoms.
    - **Diagnosis:** Ultrasound, very high β-hCG levels. Definitive diagnosis often post-evacuation histology. Transvaginal ultrasound is often key for initial diagnosis, though histology confirms the type.
    - **Management:** Suction evacuation is standard therapy. Rh D immunoglobulin for Rh-negative women. β-hCG monitoring post-evacuation is crucial.
- **Malignant GTN (Includes Choriocarcinoma, Invasive Mole):**
    - Develops after molar pregnancy (most common), normal pregnancy, ectopic, or abortion.
    - **Diagnosis:** Plateauing or rising β-hCG levels after evacuation (e.g., plateau for 3 consecutive weeks). Serum β-hCG is a sensitive marker.
    - **Prognosis Factors (Poor):** High pre-treatment β-hCG (>100,000), long duration since pregnancy (>4-6 months), metastatic disease (brain, liver), antecedent term pregnancy, prior failed chemotherapy. Age > 39/40 is adverse, so younger age (<39) is generally considered a *good* prognostic factor.
    - **Treatment:** Chemotherapy (e.g., Methotrexate for low-risk, multi-agent chemo for high-risk). Hysterectomy may be considered. Methotrexate is used for low-risk malignant GTN.
    - **Most common feature of GTN tumor:** Vaginal bleeding.

## General Malignancy Concepts

- **Tumor Markers:** Useful for monitoring treatment response and detecting recurrence, less so for screening (e.g., CA-125). AFP is primarily for germ cell/hepatocellular ca.
- **Most common Gyn Malignant in Jordan:** Endometrial Cancer.
- **Worldwide 3rd most common cancer affecting women:** Cervical Cancer.

- **Bimodal age representation:** Cervical cancer is noted to have bimodal peaks in incidence.

## Benign Gynecological Conditions

### Uterine Fibroids (Leiomyomas)

- **General:** Most common benign tumor of the uterus. Originate from smooth muscle. Contain muscle and connective tissue. Commonest tumor found in the uterus.
- **Epidemiology:** Affect many women, incidence increases with age up to menopause. Peak incidence is often in the 40s-50s, not typically highest in the 7th decade. They are common in reproductive years. Estrogen plays a role in growth.
- **Symptoms:** Often asymptomatic. Can cause Heavy Menstrual Bleeding (HMB)/Menorrhagia, pelvic pain/pressure, dysmenorrhea, urinary frequency, constipation. Infertility (especially submucosal/intramural distorting cavity). Rarely cause oligomenorrhea. Associated with polycythemia (rarely).
- **Diagnosis:** Pelvic exam, Ultrasound (transvaginal/abdominal).
- **Fibroids & Pregnancy:** Often increase in size during pregnancy. Degeneration (e.g., red degeneration) can cause pain. Can obstruct labor. Fibroids found at C-section are generally NOT removed unless pedunculated and easily accessible due to bleeding risk.
- **Complications:** Torsion of pedunculated fibroid, degeneration, compression symptoms, infertility, recurrent pregnancy loss. Anemia due to HMB.
- **Treatment:**
  - Observation if asymptomatic.
  - Medical: Hormonal therapies (COCPs, progestins, GnRH agonists), tranexamic acid. GnRH analogues cause shrinkage (max effect ~3 months, not 6 weeks), hypoestrogenic side effects (amenorrhea, hot flushes, bone loss if >6 months). GnRH analogues can make surgical dissection easier.
  - Surgical: Myomectomy (preserves fertility), Hysterectomy (definitive).
  - Uterine Artery Embolization (UAE).
- **Pathology:** Usually multiple. Can be submucosal, intramural, subserosal, pedunculated. Parasitic fibroids lose uterine attachment and gain secondary blood supply. Necrobiosis (degeneration) due to impaired blood supply, especially in pedunculated fibroids. Fibroids have a pseudocapsule, not a true capsule distinct from surrounding myometrium. Malignant change (leiomyosarcoma) is rare (<1%).

### Endometriosis

- **Definition:** Presence of endometrial glands and stroma outside the uterine cavity.
- **Epidemiology:** Affects reproductive-age women. Common cause of pelvic pain and infertility. More common in nulliparous women, potentially higher socioeconomic groups. Not typically more common in multiparous women.
- **Etiology Theories:** Retrograde menstruation (Sampson's theory), Mullerian metaplasia, lymphatic/hematogenous spread, genetic predisposition, immunological factors. Monthly ovarian follicle rupture is not a primary theory.
- **Symptoms:** Dysmenorrhea (secondary, progressive), deep dyspareunia, chronic pelvic pain, dyschezia (painful defecation), infertility, abnormal bleeding (intermenstrual spotting). Characteristic triad: dysmenorrhea, dyspareunia, dyschezia. Can be asymptomatic. Least common GI symptom might be intermittent rectal bleeding compared to cramping or pain with defecation. Pain related to inflammation.
- **Sites:** Pelvic peritoneum, ovaries (endometriomas/"chocolate cysts"), uterosacral ligaments, pouch of Douglas, rectovaginal septum, bladder, bowel. Ovaries are the most common site.
- **Diagnosis:**
  - History and pelvic examination (nodularity in pouch of Douglas/uterosacral ligaments, fixed retroverted uterus, adnexal tenderness/masses). Bimanual exam is important.
  - Laparoscopy is the gold standard for diagnosis and staging. Allows direct visualization and biopsy. Biopsy is ideal for confirmation but not always mandatory if classic lesions seen.
  - Ultrasound may detect endometriomas.
  - CA-125 may be elevated but is NOT useful as a screening or primary diagnostic test.

- **Histology:** Requires presence of endometrial glands AND stroma. Evidence of old/recent hemorrhage (hemosiderin-laden macrophages) is supportive. Decidual reaction in surrounding tissue is not a diagnostic criterion.
- **Treatment:**
  - Analgesia (NSAIDs).
  - Hormonal: COCPs (suitable for long-term use), progestogens, Danazol, GnRH agonists (effective treatment, though with side effects).
  - Surgical: Laparoscopic ablation or excision of implants, adhesiolysis, removal of endometriomas. Hysterectomy +/- BSO for severe cases not responding to other treatments.
- **Endometriosis & Infertility:** Common cause. Mechanisms include pelvic adhesions distorting anatomy, altered tubal motility, peritoneal inflammation affecting sperm/egg/embryo, Luteinized Unruptured Follicle (LUF) syndrome, potential oocyte maturation defects. Surgical treatment (moderate/severe) *may* improve spontaneous pregnancy rates but can also reduce ovarian reserve. IVF is often needed. Confirmation of tubal patency doesn't exclude endometriosis as cause of infertility.

### Adenomyosis

- **Definition:** Presence of endometrial glands and stroma within the myometrium.
- **Symptoms:** Heavy menstrual bleeding (menorrhagia), secondary dysmenorrhea. Uterus often bulky, globular, and tender on examination. Dysmenorrhea is a very likely symptom. Can be associated with deep dyspareunia.
- **Diagnosis:** Often clinical suspicion. Transvaginal ultrasound or MRI can be suggestive. Definitive diagnosis is histological after hysterectomy.
- **Treatment:**
  - Hormonal treatments (similar to endometriosis) may help symptoms.
  - Hysterectomy is the definitive treatment.

### Ovarian Cysts

- **Functional Cysts:** Most common type in reproductive years. Usually regress spontaneously over 1-3 cycles. Includes follicular and corpus luteum cysts. Management often involves observation and re-examination/ultrasound after 6 weeks or a couple of months. COCPs may prevent *new* functional cysts but don't hasten resolution of existing ones. A 5-8cm simple cyst noted on exam that regresses is likely functional.
- **Corpus Luteum Cysts:** Thin-walled, unilocular. Can cause local pain/tenderness. May be associated with delayed menstruation (but not typically amenorrhea). Can cause torsion. Usually resolve spontaneously in <2-3 months (reports of 7-10 days seem too short for typical resolution).
- **Endometriomas:** "Chocolate cysts" containing old blood. Associated with endometriosis.
- **Dermoid Cysts (Mature Cystic Teratomas):** See Ovarian Cancer section (usually benign).
- **Malignant Potential:** Features concerning for malignancy on ultrasound include: large size, solid components, thick septations, papillary projections, ascites, bilaterality. Simple cysts <5cm in postmenopausal women are often benign.
- **Management:** Depends on size, characteristics, patient age, symptoms. Observation, surgical removal (cystectomy vs oophorectomy) via laparoscopy or laparotomy. For suspected simple cyst in young asymptomatic woman, re-examination/US in 6 weeks or a couple of months is appropriate. Tumor markers (CA-125, AFP, hCG etc.) may be checked if malignancy suspected.

### Polycystic Ovary Syndrome (PCOS)

- **Diagnostic Criteria (Rotterdam):** Need 2 out of 3: Oligo/anovulation, clinical/biochemical hyperandrogenism, polycystic ovaries on ultrasound (>12-20 follicles 2-9mm or volume >10ml depending on criteria/US tech).
- **Features:** Menstrual irregularity (oligomenorrhea/amenorrhea), infertility (due to anovulation), hirsutism, acne, obesity, insulin resistance. Bilateral enlarged ovaries with smooth surface and multiple small follicles ("string of pearls" on US). Sclerosis may also be present.
- **Endocrinology:**
  - Elevated LH/FSH ratio is common but NOT always present and NOT required for diagnosis. FSH level is often normal.
  - Elevated androgens (testosterone, DHEAS). Increased free estradiol.

    - Insulin resistance and hyperinsulinemia common. Raised fasting insulin.
    - Sex Hormone Binding Globulin (SHBG) is often decreased, not increased.
- **Associated Risks:** Endometrial hyperplasia and cancer (due to chronic anovulation/unopposed estrogen), type 2 diabetes mellitus, metabolic syndrome, dyslipidemia, hypertension, cardiovascular disease, sleep apnea. Not associated with Type 1 Diabetes. Risk of breast cancer is NOT clearly increased.
- **Management:**
    - Lifestyle: Weight loss (improves insulin sensitivity, hyperandrogenism, ovulation).
    - Menstrual regularity/Endometrial protection: COCPs, progestogens.
    - Hyperandrogenism: COCPs, anti-androgens (spironolactone, cyproterone acetate).
    - Infertility: Ovulation induction (Clomiphene citrate, Letrozole, gonadotropins).
    - Insulin resistance: Metformin.
- **Patient Counseling:** Address risks of endometrial hyperplasia, diabetes, cardiovascular disease, sleep apnea. Encourage weight loss. Counsel on fertility options. Discuss management of hirsutism/acne. Glucose tolerance test should be performed.

### Uterine Polyps

- **Symptoms:** Often asymptomatic. Can cause intermenstrual bleeding (most common symptom), heavy menstrual bleeding, postmenopausal bleeding. Rarely cause infertility.

## Menstrual Cycle Disorders

### Amenorrhea

- **Primary Amenorrhea:** Absence of menstruation by age 15/16 (or by 13/14 if no secondary sexual characteristics).
    - Causes: Gonadal dysgenesis (e.g., Turner syndrome 45,XO), Mullerian agenesis (MRKH syndrome - 46,XX, normal female development but absent uterus/upper vagina), androgen insensitivity syndrome (AIS - 46,XY, female phenotype, absent uterus), constitutional delay, imperforate hymen, PCOS, hypothalamic/pituitary dysfunction, outflow tract obstruction. The most common pathologic causes in adolescents are often cited as gonadal failure/dysgenesis or anorexia nervosa/hypothalamic causes.
- **Secondary Amenorrhea:** Cessation of menses for >3-6 months after previous menstruation.
    - Most common cause: Pregnancy.
    - Other Causes: Hypothalamic dysfunction (stress, weight loss, excessive exercise, anorexia nervosa), pituitary causes (hyperprolactinemia - due to adenoma or drugs, Sheehan's syndrome), ovarian causes (PCOS, premature ovarian insufficiency/failure), uterine causes (Asherman's syndrome), thyroid dysfunction, systemic illness. Empty sella syndrome can cause amenorrhea/galactorrhea; ovulation induction is not contraindicated, though underlying cause needs assessment.
- **Asherman's Syndrome:** Intrauterine adhesions/scarring, typically after D&C (especially postpartum or post-abortion). The commonest cause is endometrial curetting after abortion or postpartum. Leads to secondary amenorrhea or hypomenorrhea.
- **Premature Ovarian Insufficiency (POI)/Failure (POF):** Ovarian failure before age 40. Causes secondary amenorrhea, infertility, menopausal symptoms. Associated with autoimmune disorders, genetic factors (e.g., Fragile X premutation), chemotherapy/radiotherapy. Not typically caused by using fertility drugs (though ovarian hyperstimulation is a risk of treatment). Bilateral oophorectomy causes surgical menopause, not POF. Early menarche is not associated.
- **Evaluation:** History, exam, pregnancy test (hCG), FSH, LH, Prolactin, TSH, Progesterone challenge test, estrogen/progesterone challenge, karyotype (if primary amenorrhea/POI), imaging (pelvic US, brain MRI if pituitary suspected).

### Dysmenorrhea

- **Primary Dysmenorrhea:** Painful menstruation without underlying pelvic pathology. Typically starts 1-2 years after menarche. Caused by excess prostaglandins leading to uterine contractions/ischemia. Pain is usually spasmodic, starts with or just before menses (not typically 3-4 days before), lasts 1-3 days. Associated with normal pelvic exam. Major cause of school/work absence. Can be associated with nausea, vomiting, diarrhea, headache, fatigue

(constipation is less typical). Relieved by NSAIDs, COCPs. Not typically relieved after marriage. Usually improves with age/childbirth. Not usually due to underlying pathology.

- **Secondary Dysmenorrhea:** Painful menstruation due to underlying pelvic pathology. Causes include endometriosis, adenomyosis, fibroids, PID, pelvic congestion, cervical stenosis, IUD use. Onset often later in reproductive life. Pain may start before menses and last longer.

### Abnormal Uterine Bleeding (AUB) / Menorrhagia

- **Definitions:**
  - Menorrhagia (Heavy Menstrual Bleeding - HMB): Excessive (>80ml) or prolonged (>7 days) regular menstrual bleeding.
  - Metrorrhagia: Irregular bleeding between periods.
  - Menometrorrhagia: Heavy and irregular bleeding.
  - Polymenorrhea: Regular cycles <21 days apart. (Definition in Q173 was incorrect).
  - Oligomenorrhea: Cycles >35 days apart. (Definition in Q173 was incorrect).
  - Hypomenorrhea: Scant menstrual flow.
  - Hypermenorrhea: Excessive regular menstrual loss. (This term is often used interchangeably with menorrhagia).
- **Causes (PALM-COEIN classification):**
  - Structural (PALM): Polyps, Adenomyosis, Leiomyoma (Fibroids), Malignancy/Hyperplasia.
  - Non-structural (COEIN): Coagulopathy (e.g., Von Willebrand's disease), Ovulatory dysfunction (e.g., PCOS, thyroid disorders, hyperprolactinemia), Endometrial causes, Iatrogenic (e.g., anticoagulants - Warfarin), Not yet classified.
  - Specific examples from Qs: Fibroids, adenomyosis, endometrial polyps, PID, endometrial hyperplasia/cancer, Von Willebrand's, ovulatory dysfunction (e.g., irregular ripening), endometriosis, cervical cancer, hypothyroidism. Hyperprolactinemia typically causes amenorrhea/oligomenorrhea, not usually HMB.
- **Dysfunctional Uterine Bleeding (DUB):** AUB with no identifiable organic pathology (diagnosis of exclusion). Often due to anovulation. Essential defect is hormonal imbalance affecting the endometrium. Diagnosis relies on ruling out other causes, may involve assessing hormonal status. Can be associated with hypothyroidism.
- **Evaluation:** History (pattern, amount), pelvic exam, CBC, TSH, Prolactin, coagulation studies (if indicated), Pap smear, transvaginal ultrasound, endometrial biopsy (especially if >40 or risk factors), hysteroscopy.
- **Treatment of Menorrhagia (HMB):**
  - Medical: Tranexamic acid (antifibrinolytic), Mefenamic acid (NSAID), COCPs, Progestogen-only pills (less commonly first line but can be used), Levonorgestrel-releasing IUS (Mirena), GnRH agonists. Copper IUDs (like Nova-T) often *increase* bleeding, so are NOT used for treatment.
  - Surgical: Endometrial ablation, Hysterectomy.

### Premenstrual Syndrome (PMS) / Premenstrual Dysphoric Disorder (PMDD)

- **Definition:** Cyclical physical, emotional, and behavioral symptoms occurring in the luteal phase and resolving with menstruation.
- **Symptoms:** Mood swings, irritability, depression, anxiety, bloating, breast tenderness, headache, fatigue, appetite changes, sleep disturbance. Vaginal spotting is NOT a typical PMS symptom. Menorrhagia is NOT a PMS symptom.
- **Management:** Lifestyle changes (diet, exercise, stress reduction), SSRIs (selective serotonin reuptake inhibitors) are effective first-line medical treatment. Vitamin B6/Calcium/Magnesium supplements may help. COCPs can regulate symptoms. GnRH agonists are used in severe, refractory cases, not first line. Stopping smoking/caffeine is advised.

## Contraception

- **Methods & Failure Rates (Typical Use, per 100 woman-years):**
  - Combined Oral Contraceptive Pills (COCPs): Ranges vary, but typical use failure is higher than perfect use (~0.3%). Failure rate of 5 per 100 woman-years means 5 pregnancies in 100 women using method for 1 year.
  - Progestogen-Only Pills (POPs): Similar failure rate range to COCPs in typical use.
  - Copper IUD (e.g., Nova-T): ~0.8.

- Levonorgestrel IUS (Mirena): ~0.2. (Value of 3 cited previously is incorrect). Contains Levonorgestrel.
- Implanon (Progestogen implant): ~0.05. Lowest failure rate among reversible methods.
- Injectable Progestins (e.g., Depo-Provera): ~0.2-6.
- Diaphragm: ~12-18.
- Condoms: ~18 (male), ~21 (female).
- Vasectomy: ~0.15. (Value of 0.02 seems too low for typical use).
- Tubal Ligation: ~0.5.

- **Combined Oral Contraceptives (COCPs):**
  - **Mechanism:** Primarily inhibit ovulation (by suppressing GnRH/FSH/LH surge), thicken cervical mucus, alter endometrium. The progestogen component is key for preventing ovulation.
  - **Benefits (Non-contraceptive):** Reduce risk of ovarian and endometrial cancer, benign breast disease, colorectal cancer, salpingitis (PID). Regulate cycles, reduce HMB, dysmenorrhea, PMS symptoms. Improve acne/hirsutism.
  - **Side Effects:** Nausea, headache, breast tenderness, fluid retention, weight gain, mood changes, loss of libido, breakthrough bleeding, chloasma. Estrogen-related: Nausea, breast tenderness. Progestogen-related: Acne, mood changes. Functional ovarian cysts are *decreased* with COCPs, not a side effect. Migraine can be worsened or triggered.
  - **Risks:** Venous thromboembolism (VTE), stroke, myocardial infarction (risk increased in smokers >35), hypertension, hepatic adenoma, gallstones. Do NOT reduce risk of cervical cancer (may slightly increase).
  - **Contraindications (Absolute):** Current/history of VTE/Stroke/MI, complicated valvular heart disease, uncontrolled hypertension, migraine with aura, current breast cancer, active liver disease/tumor, pregnancy, undiagnosed vaginal bleeding, major surgery with prolonged immobilization, smokers >35 years (>15 cigarettes/day). Relative CI: controlled HTN, smokers <35, diabetes. Medically treated gall bladder disease is generally NOT an absolute CI. Active viral hepatitis IS an absolute CI.
  - **Discontinuation Indications:** Development of focal migraine, severe hypertension, jaundice, VTE/MI/Stroke. Diagnosis of breast cancer in first-degree relative is NOT an indication to stop. Personal history of DVT IS a contraindication.
  - **Safety:** Relatively safe to continue up to age 40 in non-smokers, but risks increase, especially VTE. Use in 40s is common but requires careful consideration. Standard Ethinyl estradiol doses are usually 20-35mcg (values like 0.2-0.5mg are incorrect). May be less effective with enzyme-inducing drugs (e.g., some antiepileptics; they do not make COCPs more effective).
- **Progestogen-Only Pills (POPs / Mini-pill):**
  - **Mechanism:** Primarily thicken cervical mucus, alter endometrium; may inhibit ovulation inconsistently. Do NOT reliably suppress ovulation. Do NOT cause endometrial hyperplasia. Do NOT reduce libido as primary action. Not primarily spermicidal.
  - **Side Effects:** Irregular bleeding/spotting, absent menses, functional ovarian cysts, acne, breast tenderness. Do NOT inhibit lactation.
  - **Contraindications:** Current breast cancer, severe liver disease, undiagnosed vaginal bleeding. Mild hypertension is NOT a contraindication. Suitable for breastfeeding women.
- **Intrauterine Devices (IUDs):**
  - **Copper IUD:** Mechanism: spermicidal, inhibits fertilization, prevents implantation via sterile inflammatory reaction. Side effects: Heavier, longer, more painful periods; risk of expulsion, perforation, PID (risk highest near insertion). Can cause abnormal uterine bleeding, leading to discontinuation. NOT associated with lowest PID risk. Contraindications: Pregnancy, unexplained vaginal bleeding, active PID/STI, uterine distortion, copper allergy. History of ectopic pregnancy is a *relative* CI/precaution, not absolute. History of valvular/rheumatic heart disease is generally NOT a contraindication.
  - **Levonorgestrel IUS (Mirena):** Mechanism: Thickens cervical mucus, thins endometrium, inhibits sperm function, may inhibit ovulation. Benefits: Reduces HMB, dysmenorrhea; provides endometrial protection. Side effects: Irregular bleeding/spotting initially, amenorrhea later, hormonal side effects (mood, acne - less than systemic), ovarian cysts. Contains Levonorgestrel.

    - **IUCD & PID:** Overall PID risk low after first few weeks post-insertion. IUD use is a risk factor for PID. Lowest incidence of PID seen with methods preventing STIs (Condoms) or suppressing ovulation (OCPs). Active PID is a contraindication to insertion.
- **Injectable Progestins (e.g., Depo-Provera):**
    - **Mechanism:** Inhibits ovulation, thickens mucus, thins endometrium.
    - **Side Effects:** Menstrual irregularities (spotting, amenorrhea), weight gain, headache, mood changes, bone mineral density loss (reversible), delayed return to fertility. Does NOT typically cause flare-up of endometriosis (often used to treat it).
- **Implants (e.g., Implanon/Nexplanon):** Long-acting reversible contraception (LARC). Progestogen-only. Highly effective. Side effects similar to other progestogen methods, mainly irregular bleeding.
- **Barrier Methods (Condoms):** Protect against STIs. Higher failure rates than hormonal/IUD methods. Associated with the lowest PID incidence among common methods.
- **Emergency Contraception (EC):** Levonorgestrel pills, Ulipristal acetate pills, Copper IUD.
- **Causes of Failure:** Incorrect use (most common for pills, barriers), method failure, drug interactions (enzyme inducers reduce COCP efficacy).

## Pelvic Anatomy and Physiology

- **External Genitalia (Vulva):**
    - Mons Veneris: Pad of fatty tissue over pubic bone.
    - Labia Majora: Fatty folds, usually covered with pubic hair. Develop from genital swellings.
    - Labia Minora: Hairless folds medial to majora. Contain sebaceous glands, not primarily sweat glands. Develop from genital folds.
    - Clitoris: Erectile tissue, highly sensitive. Develops from genital tubercle.
    - Vestibule: Area enclosed by labia minora, contains urethral/vaginal openings. The genital swellings give rise to the labia majora, not the vestibule.
    - Hymen: Membrane partially covering vaginal opening. Imperforate hymen causes primary amenorrhea, cyclic pain, bulging mass (hematocolpos). Secondary sexual characteristics are normal. Associated with normal karyotype. Can cause acute urine retention. Surgery is mainstay treatment. Usually presents at 14-16 years. Secondary sexual development is usually present.
- **Internal Genitalia:**
    - Vagina: Fibromuscular tube. Upper 2/3 derived from Mullerian (paramesonephric) ducts, lower 1/3 from urogenital sinus. Canalization occurs around 20 weeks gestation.
    - Uterus: Pear-shaped muscular organ. Body, fundus, cervix. Derived from fused Mullerian ducts.
    - Cervix: Lower, narrow part of uterus.
    - Fallopian Tubes: Derived from unfused Mullerian ducts. Site of fertilization.
    - Ovaries: Produce eggs and hormones (estrogen, progesterone). NOT derived from paramesonephric duct. Develop from gonadal ridge.
- **Pelvic Supports:**
    - Level 1 (Apical Support): Cardinal ligaments (most important for uterus) and Uterosacral ligaments complex. Supports cervix/upper vagina.
    - Level 2 (Mid-vaginal Support): Attachments to arcus tendineus fascia pelvis (Pubocervical/Rectovaginal fascia).
    - Level 3 (Distal Support): Perineal body, superficial/deep perineal muscles.
    - Broad ligaments: Double layer of peritoneum, provides minimal support.
    - Round ligaments: Maintain uterine anteversion, minimal support.
    - Ligaments providing most support against prolapse: Cardinal ligaments.
- **Pelvic Diaphragm:** Formed by Levator ani (Pubococcygeus, Iliococcygeus, Puborectalis) and Coccygeus (Ischiococcygeus) muscles. Obturator internus is a lateral wall muscle, NOT part of diaphragm.

- **Pelvic Nerves:** Pudendal nerve provides sensory innervation to perineum and motor function to external anal sphincter/urethral sphincter. Sensory pain fibers from uterus travel via uterosacral ligaments and inferior hypogastric plexus.
- **Pelvic Vasculature:** Ovarian artery (from aorta), Uterine artery (from internal iliac). Left ovarian vein drains to left renal vein; Right ovarian vein drains to IVC.
- **Pelvic Inlet/Outlet/Cavity:**
  - Inlet Bounded by: Sacral promontory, ala of sacrum, arcuate line, pectineal line, pubic crest, symphysis pubis.
  - Midpelvis: Plane of least dimensions. Level of ischial spines.
  - Outlet Bounded by: Symphysis pubis, ischiopubic rami, ischial tuberosities, sacrotuberous ligaments, coccyx.
  - True Pelvis: Lies below pelvic brim.
  - Mid cavity bounded anteriorly by mid-symphysis, laterally by pubic bone/obturator fascia/ischial bone/spines, posteriorly by sacrum (around S2-S4 level; the L4/S1 junction is too high). Cavity is roughly round.
  - Diagonal conjugate: Measured clinically from lower symphysis pubis to sacral promontory (~12.5 cm minimum). Obstetric conjugate: shortest AP diameter of inlet (~11cm). True conjugate: upper symphysis to promontory (~11.5cm). The shortest distance between the promontory and the symphysis is the Obstetric Conjugate.
- **Male vs Female Pelvis:** Male pelvis is narrower, deeper, heart-shaped inlet, narrow subpubic arch (<90 degrees). Female pelvis is wider, shallower, round/oval inlet, wide subpubic arch (>90 degrees). Male pelvic cavity is cone-shaped, female is cylindrical. Anteroposterior diameter of inlet and transverse diameter of outlet are smaller in males.
- **Embryology:**
  - Genital tubercle → Clitoris (female) / Penis (male).
  - Genital folds → Labia minora (female) / Ventral penile shaft (male).
  - Genital swellings → Labia majora (female) / Scrotum (male).
  - Urogenital sinus → Lower vagina, vestibule, urethra (female) / Prostate, urethra (male). The urogenital ridge gives rise to gonads and parts of the urinary/genital tracts, but not specifically the vulva itself.
  - Paramesonephric (Mullerian) ducts → Fallopian tubes, uterus, cervix, upper vagina (female). Regress in males due to Anti-Mullerian Hormone (AMH) from Sertoli cells. Absence of AMH leads to persistent Mullerian structures (uterus/tubes) in a 46,XY individual.
  - Mesonephric (Wolffian) ducts → Epididymis, vas deferens, seminal vesicles (male). Regress in females. Testosterone drives Wolffian development.
  - Sexual differentiation depends on chromosomes (XX/XY) and hormones (Testosterone, DHT, AMH). If gonads are removed before differentiation, the default phenotype is female, regardless of chromosomal sex. Male external genitalia differentiation requires fetal testosterone action.
  - Primordial germ cells originate in yolk sac wall near allantois and migrate to gonadal ridges.

# Gynecological Infections

## Pelvic Inflammatory Disease (PID)

- **Definition:** Infection and inflammation of the upper female genital tract (uterus, fallopian tubes, ovaries, pelvic peritoneum).
- **Microbiology:** Often polymicrobial. Common pathogens include *Neisseria gonorrhoeae*, *Chlamydia trachomatis* (major cause), anaerobes, *Mycoplasma genitalium*. *E. coli* is less common primary cause.
- **Risk Factors:** Multiple sexual partners, young age, previous PID, IUD insertion (risk highest initially), vaginal douching, bacterial vaginosis.
- **Symptoms:** Lower abdominal pain (usually bilateral), deep dyspareunia, abnormal vaginal discharge, abnormal uterine bleeding, fever.
- **Signs:** Cervical motion tenderness ("chandelier sign"), adnexal tenderness, uterine tenderness, purulent cervical discharge. Bimanual examination is a most important diagnostic step. Perihepatic adhesions (Fitz-Hugh-Curtis syndrome) indicate active/past infection.
- **Diagnosis:** Primarily clinical. Criteria include pelvic pain plus CMT/uterine tenderness/adnexal tenderness. Supporting criteria: fever (>38C), elevated ESR/CRP, positive GC/CT test, purulent discharge. WBC count > 10,000 is supportive.

ESR > 15 mm/hr is supportive (value of 10 is low). Vaginal discharge is common. Bacterial cultures are useful but not always necessary for treatment initiation. Laparoscopy can confirm diagnosis but not routinely required. PID does occur in pregnancy, although less commonly.

- **Differential Diagnosis:** Ectopic pregnancy, appendicitis, ovarian cyst accident (torsion/rupture), endometriosis, UTI, diverticulitis. Hepatitis is NOT a typical differential diagnosis.
- **Complications:** Tubo-ovarian abscess (TOA), chronic pelvic pain, infertility (tubal factor), ectopic pregnancy.
- **Treatment:** Broad-spectrum antibiotics covering GC, CT, anaerobes (e.g., Ceftriaxone + Doxycycline +/- Metronidazole). Removal of IUD may be considered but not always necessary. Hospitalization if severe/pregnant/TOA suspected. D&C is NOT used for management. Laparoscopy for diagnosis/drainage if needed. Oral doxycycline and clindamycin are treatment options.
- **Prevention:** Condom use, STI screening/treatment. Lowest incidence associated with condoms or OCPs.

## Vaginitis

- **Bacterial Vaginosis (BV):**
  - Overgrowth of anaerobic bacteria (e.g., *Gardnerella vaginalis*), replacing normal *Lactobacillus* flora. NOT considered a true infection. Common, not rare. Usually minimal inflammatory reaction.
  - Symptoms: Fishy-smelling (amine) discharge, thin, grayish-white discharge. Often minimal/no inflammation or itching. Can be asymptomatic.
  - Diagnosis (Amsel Criteria - need 3/4): Homogeneous discharge, vaginal pH >4.5, positive Whiff test (amine odor with KOH), Clue cells on microscopy (epithelial cells coated with bacteria). Gram stain (Nugent score) is gold standard.
  - Treatment: Metronidazole (oral/vaginal) or Clindamycin (oral/vaginal). Clotrimazole is for yeast, not BV.
- **Vulvovaginal Candidiasis (Yeast Infection):**
  - Caused by *Candida* species, usually *C. albicans* (most common). Not a protozoal infection.
  - Risk Factors: Diabetes mellitus, antibiotic use, pregnancy, immunosuppression, OCP use, tight clothing. IUD is NOT a typical risk factor.
  - Symptoms: Vulvar itching (pruritus - predominant symptom) and burning, thick, white, cottage cheese-like discharge, dyspareunia, dysuria. Can cause vulvar erythema/edema.
  - Diagnosis: Clinical picture, normal vaginal pH (<4.5), microscopy (KOH prep) shows hyphae/yeast buds. Culture if recurrent/atypical. Vaginal acidity usually maintained or increased.
  - Treatment: Topical or oral azole antifungals (e.g., Clotrimazole, Fluconazole). Imidazole cream (Clotrimazole) is appropriate first step for suspected Candida vaginitis.
- **Trichomoniasis:**
  - Caused by protozoan *Trichomonas vaginalis*. Sexually transmitted.
  - Symptoms: Profuse, yellow-green, frothy, malodorous discharge, vulvovaginal irritation/itching/burning (is present), dysuria, dyspareunia. "Strawberry cervix" (punctate hemorrhages) seen in minority. Can be asymptomatic. Offensive discharge common.
  - Diagnosis: Elevated vaginal pH (>4.5), microscopy (wet mount) shows motile, flagellated trichomonads, rapid antigen tests, NAATs (most sensitive). KOH smear is not the primary diagnostic test.
  - Treatment: Metronidazole or Tinidazole (oral). Treat patient AND sexual partners concurrently. For suspected trichomoniasis, performing a wet mount and KOH smear first is appropriate before empiric treatment. A discharge pH of 5.5 is suggestive of Trichomoniasis or BV.

## Cervicitis

- Inflammation of the cervix.
- Causes: *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, HSV, *Trichomonas*.
- Symptoms: Purulent/mucopurulent discharge, intermenstrual/post-coital bleeding, dysuria. Often asymptomatic.
- Signs: Cervical erythema, friability, discharge from os.
- Diagnosis: Clinical exam, tests for GC/CT (NAATs).

## Menopause and Hormone Replacement Therapy (HRT)

- **Menopause:** Permanent cessation of menstruation due to loss of ovarian follicular activity. Diagnosed retrospectively after 12 months of amenorrhea. Average age ~51 years (value of 55 is high). Premature if <40 years. Delayed menopause (>55) is risk factor for endometrial cancer. Caused by ovarian follicular activity cessation, leading to low estrogen/progesterone and high FSH. Loss of basal endometrium does NOT cause menopause. Ovarian vessel sclerosis occurs. Smoking induces earlier menopause.
- **Perimenopause:** Transition period before menopause. Characterized by irregular cycles, fluctuating hormone levels, onset of symptoms. In the last 4-5 years of reproductive life, fertility declines, anovulatory cycles increase, FSH/LH levels begin to rise (not decrease), and follicular development failure becomes gradual and progressive.
- **Symptoms of Estrogen Deficiency:**
  - Vasomotor: Hot flushes (most common/distressing symptom), night sweats. Hot flushes are an early symptom.
  - Urogenital Atrophy: Vaginal dryness, dyspareunia, urinary frequency/urgency, recurrent UTIs.
  - Psychological: Mood swings, irritability, insomnia, depression, memory changes.
  - Other: Osteoporosis (increased risk), joint pains, skin changes.
- **Hormone Replacement Therapy (HRT):**
  - **Indications:** Primarily for management of moderate-severe vasomotor symptoms (main current indication). Treatment of vulvovaginal atrophy. Prevention/treatment of osteoporosis (but not first-line solely for this). Not indicated solely for preventing cardiovascular disease (WHI showed potential harm). Perimenopausal cycle irregularities, bone/joint pains, vaginal dryness, hot flushes ARE potential indications. Strong family history of osteoporosis alone is not a primary indication without other risk factors or low bone density. Bilateral oophorectomy at a young age (e.g., 38) IS an indication. Intractable hot flushes ARE an indication. Vaginal dryness/dyspareunia post-menopause IS an indication. Premature menopause IS an indication.
  - **Types:** Estrogen-only therapy (ET - only for women without a uterus), Estrogen + Progestogen therapy (EPT - for women with a uterus to protect endometrium). Continuous combined (daily E+P), Sequential combined (E daily, P for part of cycle). Topical estrogen for urogenital symptoms. Patches available (e.g., combined patch with Mirena IUD for progestogen).
  - **Risks (WHI Findings):** Increased risk of VTE, stroke, invasive breast cancer (EPT > ET), possibly coronary heart disease (initial harm in older women starting HRT). Increased risk of pulmonary embolism. Does NOT increase risk of bowel cancer (may decrease).
  - **Contraindications (Absolute):** Undiagnosed vaginal bleeding, known/suspected breast cancer, known/suspected estrogen-dependent tumor (e.g., well-differentiated endometrial cancer), active/history of VTE/stroke/MI, active liver disease, pregnancy. Relative: Migraine, fibroids, endometriosis, gallstones. Coronary artery disease is often considered a relative or absolute contraindication depending on severity. Venous thrombosis IS an absolute CI.
  - **Initiation:** Not indicated *per se* just because menopause diagnosed. Decision based on symptoms and risk/benefit assessment. Not considered premature if started after age 45.
- **Postmenopause Physiology:** Predominant estrogen is Estrone (A), derived from peripheral conversion of adrenal androgens. FSH/LH levels are high.

## Infertility

- **Definition:** Failure to conceive after 1 year of regular unprotected intercourse. Fecundity: chance of conception per cycle (~20% normal).
- **Causes:**
  - Female Factor (~40-50%): Ovulatory dysfunction (PCOS most common cause of anovulatory infertility, hypothalamic amenorrhea, POI, hyperprolactinemia), Tubal factor (PID, endometriosis, surgery), Uterine factor (fibroids distorting cavity, polyps, Asherman's, congenital anomalies), Cervical factor (stenosis, mucus hostility), Endometriosis. Anovulation contributes significantly (~25%).
  - Male Factor (~30-40%): Abnormal semen parameters (oligo-, astheno-, teratozoospermia), varicocele, infections, obstruction, genetic factors, erectile/ejaculatory dysfunction.
  - Unexplained (~10-20%).
- **Evaluation:**

    - Female: History (cycles, past pregnancies, STIs, surgery), exam, assessment of ovulation (basal body temp, LH kits, mid-luteal progesterone), TSH, prolactin, pelvic ultrasound, hysterosalpingogram (HSG - checks tubal patency, can diagnose hydrosalpinx, but not endometriosis/adhesions reliably), laparoscopy (if endometriosis/adhesions suspected). Mid-luteal progesterone level IS part of initial assessment guidelines.
    - Male: Semen analysis (volume, count, motility, morphology). Normal semen analysis parameters include count >15-20 million/mL, motility >40% (progressive+non-progressive) or >32% (progressive), morphology >4% normal forms (WHO criteria). If semen analysis normal, extensive male physical exam adds little. Oligozoospermia definitions vary (<15 or <5 million/mL used in different contexts). Sperm quality varies over time. Retrograde ejaculation is common in diabetic patients. Varicocele treatment benefit on pregnancy rates is debated.
- **Treatment:** Treat underlying cause. Ovulation induction (Clomiphene, Letrozole, Gonadotropins - treatment for hypothalamic amenorrhea/hypogonadotropic hypogonadism). Intrauterine Insemination (IUI). In Vitro Fertilization (IVF). Surgery (myomectomy, tuboplasty, endometriosis excision).
- **Specific Conditions & Infertility:**
    - Endometriosis: See Endometriosis section. IVF often required. Surgical treatment for moderate/severe endometriosis may improve spontaneous rates but can also reduce ovarian reserve. Any endometriomas should ideally be managed before IVF, though removal isn't always mandatory and carries risk.
    - PCOS: See PCOS section. Ovulation induction is key.
    - Hypogonadotropic Hypogonadism: Treatment is FSH & LH (gonadotropins). Idiopathic cases treated with gonadotrophins.
    - Cervical Mucus Hostility: Relatively viscous mucus can impair sperm penetration. Post-coital test assesses this. Treatment may involve IUI or improving mucus (e.g., estrogen). Performing immunological tests for sperm antibodies may be considered. Giving ovulation induction for IVF, performing IUI, or prescribing estrogen to improve mucus are potential next steps after a poor post-coital test, depending on the full clinical picture (repeating the test might also be valid).

## Screening, Diagnosis, and Procedures

- **Pap Smear (Cervical Cytology):** Screens for cervical pre-cancer (CIN) and cancer. Not designed for infection, polyps, or erosion.
- **Colposcopy:** Magnified view of cervix, vagina, vulva. Used to evaluate abnormal Pap smears, guide biopsies. Acetic acid and Lugol's iodine used to highlight abnormal areas.
- **Endometrial Biopsy:** Samples endometrial tissue. Used to investigate AUB, postmenopausal bleeding, suspected hyperplasia/cancer.
- **Hysterosalpingogram (HSG):** X-ray with contrast to visualize uterine cavity and fallopian tubes. Assesses tubal patency, uterine shape (e.g., bicornuate uterus). Can diagnose hydrosalpinx, submucous fibroids, polyps, adhesions. Cannot reliably diagnose endometriosis or minimal adhesions. Can diagnose Ovarian cyst if it communicates with tube somehow? Unlikely. Cannot diagnose endometriosis or subserous fibroids.
- **Pelvic Ultrasound (Transvaginal/Transabdominal):** Visualizes uterus, ovaries, adnexa. Assesses fibroids, ovarian cysts, endometrial thickness, ectopic pregnancy, monitors follicular development. Key tool in evaluating adnexal masses, AUB, pelvic pain. Can suggest molar pregnancy. Used in infertility workup.
- **Hysteroscopy:** Direct visualization of uterine cavity. Diagnostic (polyps, fibroids, adhesions, septa) and operative (polypectomy, myomectomy, adhesiolysis). Complications: Uterine perforation, fluid overload (water intoxication, pulmonary edema if distension media absorbed), air embolism, bleeding, infection. Does NOT cause cervical incompetence. Septicemia is a recognized, though rare, complication.
- **Laparoscopy:** Direct visualization of pelvic/abdominal organs. Gold standard for endometriosis diagnosis. Used for tubal ligation, ovarian cystectomy, myomectomy, ectopic pregnancy treatment, adhesiolysis, staging malignancies, investigating pelvic pain/infertility. Key diagnostic tool for adnexal torsion, ruptured ovarian cyst, ectopic pregnancy, PID confirmation/TOA drainage. Less useful for fibroid degeneration diagnosis. Diagnostic laparoscopy is key tool in ectopic pregnancy, PID, adnexal torsion, ruptured ovarian cyst, but not typically for fibroid degeneration.
- **Dilatation and Curettage (D&C):** Dilating cervix and scraping uterine lining. Used for diagnosis (AUB) or treatment (miscarriage, abortion, removing polyps). Complications: Perforation, hemorrhage, infection, Asherman's syndrome. Does NOT cause Rh isoimmunization (unless performed for pregnancy-related issue in Rh neg woman without Anti-D) or directly cause cervical incompetence. Endometritis IS a risk.

- **Hysterectomy:** Surgical removal of uterus. Can be abdominal, vaginal, laparoscopic. Indications: Fibroids, AUB, endometriosis, adenomyosis, prolapse, malignancy. Total hysterectomy removes uterus and cervix. Often includes BSO (removal of tubes/ovaries) depending on age/indication. Resection usually involves uterus & cervix; tubes/ovaries optional. Complications: Hemorrhage, wound infection (most common, occurs ~5 days post-op), urinary tract infection (occurs usually within 24h post-op), injury to bladder/ureter/bowel, VTE (thrombophlebitis ~7-12 days, PE ~7-12 days), atelectasis, vaginal vault dehiscence. Wound disruption ~4-8 days post-op.

## Vulvar Conditions

- **Lichen Sclerosus:** Chronic inflammatory condition. Causes vulvar itching, soreness, dyspareunia, scarring ("figure-of-eight" pattern), narrowing of introitus. Affects all ages, including children. Most common presentation is pruritus. Increased risk of squamous cell carcinoma. Treatment: Ultrapotent topical steroids (mainstay). Surgical excision is NOT main treatment and often leads to recurrence.
- **Vulvar Pruritus:** Itching of the vulva. Common causes: Candidiasis, Lichen sclerosus, dermatitis (eczema, contact), psoriasis, VIN, Paget's disease, nerve irritation. Less common: Trichomonas, Gonorrhea. Atrophic vulvitis (due to estrogen deficiency) causes pruritus, treated effectively with topical estrogen.

## Congenital Anomalies & Disorders of Sexual Development (DSD)

- **Turner Syndrome (45,XO):** Features: Short stature, webbed neck, broad chest, ovarian dysgenesis (streak gonads), primary amenorrhea, infertility, lack of secondary sexual characteristics, associated cardiac/renal anomalies, lymphedema. Intelligence usually normal. Risk does NOT rise with maternal age. Can present with cystic hygroma in utero. Estrogen therapy needed for development and bone density. Karyotyping has greatest importance in diagnosis.
- **Androgen Insensitivity Syndrome (AIS):** 46,XY karyotype. Defect in androgen receptor. Phenotypically female with normal breast development, blind-ending vagina, absent uterus/tubes, intra-abdominal/inguinal testes. Primary amenorrhea. Scant/absent pubic/axillary hair (due to receptor defect). No prostate/internal male genitalia. Gonadectomy needed AFTER puberty due to malignancy risk (seminoma). Testosterone levels are normal/high male range.
- **Mullerian Agenesis (MRKH Syndrome):** 46,XX karyotype. Congenital absence of uterus, cervix, upper vagina. Normal ovaries and secondary sexual characteristics (Tanner V breast/pubic hair). Primary amenorrhea. Associated with renal/skeletal anomalies. Blind ending vagina on exam.
- **Mixed Gonadal Dysgenesis:** Usually mosaic karyotype (e.g., 45,X/46,XY). Ambiguous genitalia, asymmetric gonadal development (streak gonad + dysgenic testis). Can feature ambiguous genitalia.
- **Congenital Adrenal Hyperplasia (CAH):** Enzyme defect (usually 21-hydroxylase) leads to cortisol deficiency, excess adrenal androgen production. 46,XX females present with ambiguous genitalia at birth (virilization). Can cause salt wasting. Adrenal hyperplasia causes ambiguous genitalia.
- **Ambiguous Genitalia Causes:** CAH (most common cause in newborns), AIS, mixed gonadal dysgenesis, true hermaphroditism, 5-alpha-reductase deficiency. Turner's syndrome does NOT cause ambiguous genitalia. Cryptorchidism is undescended testes, not ambiguous genitalia. Klinefelter's (47,XXY) affects males, not ambiguous genitalia. Testicular feminization is AIS. Adrenogenital syndrome (CAH) causes ambiguous genitalia.
- **Imperforate Hymen:** See External Genitalia section.
- **Bicornuate Uterus:** Congenital Mullerian anomaly. Associated with recurrent pregnancy loss, preterm labor, malpresentation. Not associated with menorrhagia or primary amenorrhea typically. Associated with renal anomalies (screen urinary system). Anomalies resulting from Mullerian duct development issues include vaginal septum, unicornuate uterus, vaginal agenesis. Imperforate hymen is a urogenital sinus issue.

## Pelvic Organ Prolapse and Urinary Incontinence

- **Pelvic Organ Prolapse (POP):** Herniation of pelvic organs into vagina.
  - Types: Cystocele (bladder prolapse - anterior wall), Rectocele (rectum prolapse - posterior wall), Enterocele (small bowel prolapse - apical), Uterine prolapse, Vaginal vault prolapse (after hysterectomy).
  - Risk Factors: Multiparity, vaginal delivery, aging, menopause (estrogen deficiency), obesity, chronic cough/constipation (raised intra-abdominal pressure), connective tissue disorders, previous pelvic surgery (e.g., hysterectomy). Smoking, possibly prior inguinal hernia repair history (as marker of tissue weakness), age, multiparity, collagen deficiency are all potential contributors.

    - Cystocele: Prolapse of bladder into anterior vaginal wall. Common after menopause. Can cause stress urinary incontinence (SUI), urinary tract infections (UTIs), incomplete bladder emptying. Uncommon in nulliparous women. Prolapse is of bladder into *anterior* wall, not specifically upper part.
    - Procedentia: Complete eversion of the vagina with uterus outside the body (Stage 4 prolapse).
- **Urinary Incontinence:**
    - Stress Urinary Incontinence (SUI): Involuntary leakage on effort/exertion (coughing, sneezing). Due to urethral hypermobility or intrinsic sphincter deficiency. Common cause is pelvic floor weakness. Cystocele can contribute. Stress incontinence is the likely explanation for leakage on coughing/sneezing in a 68-year-old.
    - Urge Urinary Incontinence (UUI): Leakage associated with urgency. Due to detrusor overactivity (DO).
    - Detrusor Overactivity (DO): Commonest cause is idiopathic. Other causes: neurological conditions (upper motor neuron lesion), bladder irritation (infection, stones, tumor), previous incontinence surgery. NOT typically caused by low estrogen level (though atrophy can worsen symptoms). Characteristic symptom is urgency and frequency. Treated with anticholinergic medication or beta-3 agonists.
    - Overflow Incontinence: Due to chronic urinary retention, bladder outflow obstruction.
    - Mixed Incontinence: Combination of SUI and UUI.
- **Evaluation:** History, pelvic exam (assess prolapse, cough stress test), urinalysis, post-void residual volume, urodynamics (especially if surgery planned or diagnosis unclear). History alone is often insufficient to diagnose Genuine Stress Incontinence definitively.
- **Management:**
    - Conservative: Pelvic floor muscle training (Kegels), lifestyle changes (weight loss, fluid management), pessaries.
    - Medical: Anticholinergics/Beta-3 agonists for UUI/DO (Oxybutynin - CI in urine retention, myasthenia gravis, narrow angle glaucoma, severe ulcerative colitis). Topical estrogen for atrophy.
    - Surgical: Mid-urethral slings (TVT/TOT) for SUI. TVT can be done under local/regional/general anesthesia. TVT is for SUI, NOT for detrusor instability. Prolapse repair (anterior/posterior repair, sacrocolpopexy, etc.). Complications of slings: postoperative voiding problems, bladder perforation, erosion (vaginal/urethral), retropubic bleeding/hematoma, ureteric injury (rare).

## Puberty

- **Definition:** Transition from childhood to reproductive maturity.
- **Sequence of Events (Girls):** Thelarche (breast budding - first sign) → Pubarche (pubic hair) → Growth spurt → Menarche (onset of menstruation). Changes in voice occur but later.
- **Timing:** Median age of menarche ~12.5-13 years (Range often cited as 10-16). Pubertal changes occur faster in girls than boys. Growth as measured by height slows significantly and stops ~1-2 years after menarche.
- **Precocious Puberty:** Onset before age 8. Central (GnRH-dependent) or Peripheral (GnRH-independent - e.g., McCune-Albright syndrome). Gonadotrophin-releasing hormone analogues are the mainstay of treatment for *central* precocious puberty. McCune-Albright involves *peripheral* precocious puberty.
- **Delayed Puberty:** Absence of thelarche by age 13 or menarche by age 15/16. Causes include constitutional delay, hypogonadism (hyper- or hypo-gonadotropic).