

# OBSTETRICS

## PREGNANCY

### Physiology of Pregnancy:

- CO input increases 30-50% (max 20-24 weeks) (mostly due to increase in stroke volume)
- SVR and arterial bp decreases (**likely due to increase in progesterone**)
  - decrease in systolic blood pressure of 5 to 10 mm Hg and in diastolic blood pressure of 10 to 15 mm Hg that **nadir at week 24**.
- Increase tidal volume 30-40% and **total lung capacity decrease by 5% due to diaphragm**
- Increased red blood cell mass
- **GI: nausea – due to elevations in estrogen, progesterone, hCG** (resolve by 14-16 weeks)
- Stomach – prolonged gastric emptying times and decreased GE sphincter tone → reflux
- Kidneys increase in size and ureters dilate during pregnancy → increased pyelonephritis
- GFR increases by 50% in early pregnancy and is maintained, RAAS increases = increase aldosterone, but no increased sodium bc GFR is also increased
- **RBC volume increases by 20-30%**, plasma volume increases by 50% → decreased crit (dilutional anemia)
- Labor can cause WBC to rise over 20 million
- Pregnancy = **hypercoagulable state** (increase in fibrinogen and factors VII-X); clotting and bleeding times do not change
- Pregnancy = hyperestrogenic state
- hCG double 48 hours during early pregnancy and reach peak at 10-12 weeks, decline to reach steady stage after week 15
- **placenta produces hCG which maintains corpus luteum in early pregnancy**
- corpus luteum produces progesterone which maintains endometrium
- increased prolactin during pregnancy
- **elevation in T3 and T4**, slight decrease in TSH early on, but overall euthyroid state
- linea nigra, perineum, and face skin (melasma) changes
- **increase carpal tunnel (median nerve compression)**
- increased caloric need 300cal/day during pregnancy and 500 during breastfeeding
- should gain 20-30 lb
- increased caloric requirements: protein, iron, folate, calcium, other vitamins and minerals

<b>TABLE 1-1</b> Signs and Symptoms of Pregnancy
<b>Signs</b>
Bluish discoloration of vagina and cervix (Chadwick sign)
Softening and cyanosis of the cervix at or after 4 wk (Goodell sign)
Softening of the uterus after 6 wk (Ladin sign)
Breast swelling and tenderness
Development of the linea nigra from umbilicus to pubis
Telangiectasias
Palmar erythema
<b>Symptoms</b>
Amenorrhea
Nausea and vomiting
Breast pain
Quickening—fetal movement

### Testing:

In a patient with **irregular menstrual cycles** or **unknown date of last menstruation**, the last date of intercourse should be used as the marker for repeating a urine pregnancy test. A **urine pregnancy test 14 days after last intercourse** would minimize the possibility of a false negative. False negatives are most likely caused by performing the test too soon after conception. A **serum test** is the most **sensitive test**, but **results are not immediately** available and there is an increased cost associated with the serum test. A repeat urine test is readily available and may eliminate the need for the serum test.

### Prenatal Care

- Naegle's rule: LMP – 3 months + 7 days
- Gravida (# total pregnancies), parity (# deliveries), P(TPAL) – term, preterm, abortion, live
- first visit: between 6-10 weeks, usually 6-8 weeks after LMP and examined every 4 weeks until 32 weeks, every 2 weeks until 36 weeks then weekly
- 1. get a history: patient's history includes the present pregnancy, the LMP, and symptoms during the pregnancy. After this, an obstetric history of prior pregnancies, including date, outcome (e.g., SAB

[spontaneous abortion], TAB [therapeutic abortion], ectopic pregnancy, term delivery, mode of delivery, length of time in labor and second stage, birth weight, and any complications, should be obtained. Finally, a complete medical (GYN history = pap smear/cysts, fibroids, STIs, CV hx, asthma, autoimmune disorders, bleeding disorders, seizure disorders), surgical (abdominal / pelvic), family (chromosomal abnormalities, mental retardation, diabetes), and social history (alcohol, drugs, diet) should be obtained.

- Each prenatal visit: focused history and exam, assessment of general health, diet, activity, complaint with vitamins, maternal weight gain, edema, fetal movement (begins 18-20 weeks in primigravida / 14-18 in multi), blood pressure
  - **Check fundal height at 20 weeks – 20 weeks = umbilicus**
  - Fetal heart tones (120-160bpm) + UA for g/c at 10 weeks (can detect cardiac activity at week 6)
  - **Assess cervical dilation at 36 weeks**
- The most accurate method of determining estimated delivery date is **crown-rump length**. **Transvaginal ultrasound** is more accurate than abdominal ultrasound. The results are **most accurate early in the pregnancy**, ideally before the 22+0 week, and less accurate as the pregnancy progresses, due to biological differences in development and variations in fetal position. When assessing fetal development and gestational age using sonogram, there are milestones that should be noted during the first trimester. **Fetal cardiac activity** can be detected by week six. The **fetal yolk sac should be visible at around five weeks**, but degrades between weeks 10 and 12. The second and third trimester assessment relies on biometric markers such as biparietal diameter and head circumference, the more reliable of the markers to estimate gestational age.
- Pelvic exam: pap (if not in past 6 mo), G/C
- Bimanual: size should be consistent with gestational age from LMP
- 1<sup>st</sup> trimester testing: CBC, crit, blood type, ab screen, RPR, rubella ab, hep B surface antigen, UA, urine culture, VZV ab, PPD, sometimes toxoplasma titers (debated), nuchal translucency
  - Nuchal translucency screening / nuchal fold: performed at 10-13 weeks for trisomies 13, 18, 21, Turner syndrome → if wide measurement detected CVS or amniocentesis offered (CVS allows for first trimester termination), risk of CVS = amnio
- 2<sup>nd</sup> trimester: maternal serum alpha protein (15-18 weeks) – elevation = increased risk neural tube defects, decrease in down syndrome
  - B-HCG + estriol + MSAFP = triple screen (+ inhibin A = quad screen)
    - **Low unconjugated estriol and AFP + high inhibin A = trisomy 21 risk**
    - **High AFP = neural tube defects**
  - 18 – 20 weeks = screen for anomalies on US
  - **Amnio performed 15-18 weeks if AMA**, previous child with chromosomal abnormality, pt. or father of baby with chromosomal anomaly, family hx chromosomal anomaly, neural tube defect risk, abnormal 1<sup>st</sup>/2<sup>nd</sup> trimester maternal serum screening tests, two previous pregnancy losses, abnormal US
- 3<sup>rd</sup> trimester: fetus = viable, prenatal visits q2-3weeks from 28-36, q week >36 weeks; 37 weeks = cervix examination at each visit (stripping / sweeping membranes also performed to decrease needing induction of labor)
  - GLT: 50g oral glucose after 1 hr - >140 → **3 hour OGTT (fasting: 95, 1 hr: 180), 2 hr: 155, 3 hr: 140**
  - High risk: vaginal culture gonorrhea / chlamydia repeated
  - Latent HSV = antiviral prophylaxis at 36 weeks
  - GBS: 36 weeks

■ **TABLE 1-3** Routine Tests in Prenatal Care

Initial Visit and First Trimester	Second Trimester	Third Trimester
Hematocrit	MSAFP/triple or quad screen	Hematocrit
Blood type and screen	Obstetric ultrasound	RPR/VDRL
RPR/VDRL	Amniocentesis for women interested in prenatal diagnosis	GLT
Rubella antibody screen		Group B streptococcal culture
Hepatitis B surface antigen		
Gonorrhea culture		
Chlamydia culture		
PPD		
Pap smear		
Urinalysis and culture		
VZV titer in patients with no history of exposure		
HIV offered		
Early screening for aneuploidy (NT plus serum markers)		

- External doppler + external stress gauge for uterine contractions = nonstress test (NST) – baseline HR = 120-160, normal NST = 2 accels in 20 min of 15bpm from baseline for 15 seconds; persistent late decels (**decline in fetal HR 15bpm lasting more than 15 seconds or slow return to baseline**)
- BPP: NST, amniotic fluid level, gross fetal movements, fetal tone, fetal breathing (each worth 2 points)
  - Asphyxia risk rises with score
- All subsequent visits: bp, weight, urine dipstick (protein, glucose, blood, leuks), measurement of uterus, auscultation of the FH
- Measurement of uterine fundal height in cm corresponds to weeks gestation: 12 weeks = above pubic symphysis, 14-16 weeks = btwn pubic symphysis and umbilicus, 20 weeks = umbilicus, 20-38 corresponds roughly +/-2cm to weeks of gestational age, 38-40: 2-3cm below xiphoid

#### Multiple Gestation: twins = 1 in 80 births

- All same symptoms of pregnancy occur, but more severe → more frequent prenatal visits
- Twins: 2/3 dizygotic / fraternal; mono occur randomly and associated with fetal transfusion syndrome and discordant fetal growth
- MC complications: spontaneous abortion and preterm birth, preeclampsia, anemia
- Fetal complications: intrauterine growth restriction, cord accidents, death of one twin, congenital anomalies, abnormal or breech presentation, placental abruption or previa
- Monozygotic twins carry identical genetic material, whereas dizygotic twins are from separate ova and sperm.
- Multiple gestations are at increased risk for preterm labor and delivery, placenta previa, postpartum hemorrhage, pre-eclampsia, cord prolapse, malpresentation, and congenital abnormalities.
- There is a genetic predisposition for dizygotic twinning, whereas the rate of monozygotic twinning is the same throughout all races and families.
- Monozygotic twins are at risk for TTTS and should have frequent ultrasound examinations to diagnose this early.
- Vaginal delivery of vertex/vertex presenting twins is preferred and is possible with vertex/nonvertex twins under the right circumstances. Nonvertex presenting twins and Mo-Mo twins are delivered by cesarean section.
- Mo-Mo twins: single placenta, one chorion, one amnion
- Mo-Di: single placenta, single chorion, two amniotic sacs
- Di-Di: two placentae, two chorion, two amniotic sacs
- **Twin-twin transfusion syndrome** occurs in **monochorionic twins** and describes a phenomenon whereby the **blood of one twin is transfused to the other through vascular anastomoses in their shared placenta**. The donor twin can become **anemic** and suffer **intrauterine growth retardation**, while the recipient twin can become plethoric and acquire neurologic damage. The **donor** twin will exhibit **oligohydramnios** on ultrasound, and the **recipient** twin will demonstrate **polyhydramnios**. The stages of disease progression in twin-twin transfusion syndrome are calculated according to the Quintero system. **Stage one** refers to polyhydramnios/oligohydramnios, **visualized bladders in both** fetuses, and evidence of normal umbilical Doppler flow. **Stage two** is entered when **the bladder of the donor fetus is not visualized** on ultrasound. Further, if **Doppler flow is abnormal** in either fetus, **stage three** has begun. **Stage four** is when one or both of the fetuses show **signs of hydrops**, and **stage five** refers to the **death** of one or both fetuses. **Stage one** twin-twin transfusion syndrome may be managed **expectantly**, but **stages two through five** require **clinical intervention**. The intervention of choice in **Quintero stage two** is **fetoscopic laser ablation of placental anastomoses**. This intervention utilizes a laser, inserted fetoscopically, to ablate the anastomotic vessels and halt the transfusion of blood from one twin to the other. Complications of the procedure include preterm premature rupture of membranes (PPROM), rupture of the inter-twin membranes, twin anemia polycythemia sequence, and intra-amniotic bleeding. Despite the risks involved, the procedure is recommended for **Quintero stage two to four** in pregnancies from **16 to 26 weeks of gestation** due to evidence of a two-fold increase in perinatal survival as compared to expectant management alone. The patient in the above vignette is in Quintero stage two because Doppler flow is good but there is oligohydramnios/polyhydramnios sequence, and no bladder is visualized in the donor twin. As the mother is 20 weeks pregnant, the best intervention would be fetoscopic laser ablation.

- **Multiple gestation pregnancies** put patients at increased risk of nearly **all pregnancy complications**, such as **premature labor, preeclampsia, congenital birth defects, and anemia**. Fetal complications in twin pregnancies include increased risk of **congenital anomalies, low birth weight, and twin-twin transfusion syndrome**.
- Recommendations to give a patient who is pregnant with twins include (but are not limited to): aim for total **weight gain of 37–54 pounds** during pregnancy, take **one** prenatal vitamin with iron during the **first trimester** and **two** prenatal vitamins with iron during the **second and third trimesters, exercise as tolerated**, supplement the diet with **1 mg of folate** and **1000 mg of vitamin D**, expect and keep appointments for **more frequent ultrasounds** than a singleton pregnancy, and sleep on the **left side** during the **second and third trimesters**. For anemia, take two prenatal vitamins with iron instead of one.

#### Routine problems of pregnancy:

- back pain: massage, heating pad, Tylenol, PT, sometimes: muscle relaxant / narcotic
- constipation - avoid laxatives 3<sup>rd</sup> trimester bc can induce labor
- contractions: dehydration can cause, contractions q10min = preterm labor
- dehydration: due to expanded IV space and increased third spacing – increased fluids!!
- Edema: compression of IVC and pelvic veins by uterus → increased hydrostatic pressure – elevate feet / sleep on side
- GERD: antacids, eat multiple small meals per day, avoid lying down after 1 hour of eating, H2blocker / PPI
- Hemorrhoids: topical anesthetics and steroids for pain and swelling / prevent constipation
- PICA: order tox screen / encourage to stop
- Round ligament pain: late 2<sup>nd</sup> trimester / early 3<sup>rd</sup> – pain in adnexa / low abdomen – self-limited (warm compress / Tylenol)
- Urinary frequency: r/o UTI
- Varicose veins: elevate lower extremities, compression stockings, refer if still there 6 mo postpartum

#### Pregnancy Complications:

#### Classification of Spontaneous Abortion

##### Spontaneous abortion

- Termination of pregnancy **before 20 weeks** – occurs in 15-20% of pregnancies
- **r/f:** smoking, infection, maternal systemic disease, immunologic parameters, drug use
- **s/s:** bleeding = variable, fundus of uterus may be boggy or tender
- **dx:** serial hCG titers, serum progesterone, serial US to confirm; US: inappropriate development or interval growth, poorly formed / unformed fetal pole, fetal demise; blood type and Rh status necessary tests to preclude Rh sensitization in mother
- **tx:** empty uterus, follow up with pelvic exams, serial hCG titers, transvaginal US, dilation and curettage to ensure complete emptying of uterus; immunoglobulin administered to Rh negative women, septic / infected abortion requires complete evacuation of uterine contents, medical support, abx

Type	Vaginal Bleeding	Cervix Open	POC passed
Threatened	Yes	No	No
Inevitable	Yes	Yes	No
Incomplete	Yes	Yes	Partial
Complete	Yes	Yes	Yes
Missed	No	No	No

Types of Spontaneous Abortions			
Abortion Classification	Characteristics	Cervical Os	Passage of Fetal Tissue
Threatened	Abdominal pain or bleeding in the first 20 weeks of gestation	Closed	None
Inevitable	Abdominal pain or bleeding in the first 20 weeks of gestation	Open	None
Incomplete	Abdominal pain or bleeding in the first 20 weeks of gestation	Open	Yes (Some products of conception remain in uterus)
Complete	Abdominal pain or bleeding in the first 20 weeks of gestation	Closed	Complete passage of fetal parts and placenta and contracted uterus
Septic	Infection of uterus during miscarriage, fever and chills. Usually due to <i>Staph aureus</i>	Open with purulent cervical discharge and uterine tenderness	None or may be incomplete

##### Ectopic Pregnancy:

- Implantation of pregnancy anywhere but endometrium, 95% in fallopian tube (55% in ampulla of tube)
- **MC cause = occlusion of tube secondary to adhesions**
- **r/f:** hx of previous ectopic, previous salpingitis (caused by PID), previous abdominal or tubal surgery, use of IUD, assisted reproduction
- **s/s:** unilateral adnexal pain, amenorrhea / spotting, tenderness or mass on pelvic exam, dizziness, syncope, GI distress

- **ruptured ectopic:** severe abdominal or **shoulder pain**, peritonitis, tachycardia, syncope, orthostatic HTN
- **dx:** serial increases of hCG are less than expected - >1500 should show evidence of developing intrauterine gestation on ultrasound → if not, suspect ectopic, transvaginal US >90% sensitive,
- **tx:**
  - **methotrexate** (if ectopic measures <4cm, bhCG <5000, hemodynamically stable, no blood disorders, no pulmonary disease, no peptic ulcer, normal renal function, normal hepatic function, compliant pt that can return for follow up
    - **Administration of methotrexate** is the appropriate treatment for an ectopic pregnancy unless there are contraindications to the use of the drug. These **contraindications** include current breastfeeding, active pulmonary disease, immunodeficiency, or hypersensitivity to methotrexate. The drug is a **folic acid antagonist that inhibits DNA replication**. The effectiveness of administration is similar to surgical treatment without the risk of surgical complications. **Indications** for methotrexate therapy include a **hemodynamically stable** patient, **hCG levels below 5,000 IU/L**, mass <3.5 cm, **no fetal cardiac activity**, and the ability to **comply with post-treatment follow-up**. Methotrexate can be administered intravenously, intramuscularly, or orally. It can also be injected into the ectopic pregnancy directly, although this route of administration is not commonly used. Intramuscular administration is the route of administration that is most commonly used for treatment of ectopic pregnancy.
  - **surgical treatment:** laparoscopy → emergent situations / not meeting methotrexate criteria
  - **follow-up testing** = crucial

#### Gestational Trophoblastic Disease:

- spectrum of diseases arising from placental and includes complete and partial hydatidiform moles, placental site-invasive moles, trophoblastic tumors, choriocarcinomas
- molar pregnancy = benign
- **r/f:** AMA, hx of previous mole
- **s/s** complete molar pregnancy: empty egg, no viable fetus, abnormal vaginal bleeding, uterine size greater than gestation age based on LMP, hyperemesis gravidarum, high blood pressure during first 20 weeks pregnancy, snowstorm pattern / grapelike vesicle on US (20% progress to malignancy)
- partial hydatidiform moles have fetus present, but fetus is nonviable
- **dx:** **hCG level >100,000** and persistently elevated, US shows grapelike vesicles or snowstorm appearance consistent with swelling of chorionic villi
- **tx:** depends on tumor classification
  - benign / low-risk tumors treated with chemotherapy; high-risk require combination of chemo with or without adjuvant radiation / surgery
  - **surgical:** suction curettage (to preserve fertility) or hysterectomy – 80-100% cure rate
  - **monitor with serial hCG to assure return to baseline and dx and manage sequelae;** contraception recommended 6-12 months afterwards
- This patient presents with vaginal bleeding and an ultrasound consistent with a **hydatidiform mole** or **molar pregnancy** requiring **obstetrics consultation**. Molar pregnancy is a spectrum of diseases characterized by abnormal chorionic villi proliferation. A **complete hydatidiform mole** refers to the situation in which there is **no fetal tissue**. In an **incomplete mole**, there is some fetal tissue along with trophoblastic hyperplasia. Patients with molar pregnancy may present with nausea, vomiting, abdominal pain, and vaginal bleeding. Without ultrasound, it is difficult to differentiate these patients from a threatened miscarriage or ectopic pregnancy. Often, the **uterine size is larger than the expected for dates in molar pregnancy** and the **beta-hCG is higher than expected for dates**. Diagnosis is based on characteristic findings on ultrasound. Hydropic vessels within the uterus cause a **“snowstorm” appearance**. Because of the potential for complications and the non-viability of the pregnancy, dilation and curettage is recommended. Once a hydatidiform mole is diagnosed, a chest X-ray should be obtained as trophoblastic tumors metastasize to lung, liver, and brain.
- **Quantitative beta-human chorionic gonadotropin** level is the best method to monitor for recurrent gestational trophoblastic disease. **Gestational trophoblastic disease (GTD)** is a collective term for disorders that include partial and complete hydatidiform mole, invasive mole, choriocarcinoma, and placental site trophoblastic tumor. GTD can be non-neoplastic or neoplastic. The non-neoplastic form of GTD includes exaggerated placental site, placental site nodules, and hydatidiform moles. Neoplastic GTD includes invasive mole, choriocarcinoma, and trophoblastic tumors.

Hydatidiform mole is the most common form of GTD. Risk factors include prior molar pregnancy, advanced maternal age, and Asian, Native American, or African ancestry. Patients with GTD typically present with uterine bleeding, excessive nausea and vomiting, and a uterus that is larger than expected. Serum beta-human chorionic gonadotropin (hCG) levels generally range from high normal to in the millions. **Ultrasound** is the mainstay of diagnostic studies. A **“snowstorm appearance”** is a pathognomonic finding on ultrasound. **Uterine evacuation with suction curettage** is the treatment of choice. However, trophoblastic tissue remains in about 20% of patients. All patients with GTD should be monitored with **serial serum hCG levels** to identify persistent disease. Patients should be advised to avoid pregnancy for at least six months, so that hCG can return to undetectable level. Chemotherapy is indicated for neoplastic GTD. GTD is curable in 85-100% of cases, even with metastases.

- **Placental site trophoblastic tumors** cause **very low, persistent levels of hCG**. These are **malignant** tumors that most commonly occur after a non-molar abortion or pregnancy. They can be diagnosed months to years after pregnancy and tend to remain in the uterus for long periods of time before spreading regionally or metastasizing. Patients typically present with **abnormal uterine bleeding and pelvic pressure**. When hCG levels are tested, they will be elevated at lower levels than other subtypes or gestational trophoblastic neoplasms. Pelvic ultrasound will demonstrate a hyperechoic-intrauterine mass which may invade the myometrium. Chest radiographs should be obtained to help rule out metastatic disease. Treatment in patients who do not desire fertility preservation includes **hysterectomy**. These types of neoplasms are generally **resistant to chemotherapy**.

### Placenta Previa:

- Complete previa: placenta completely covers internal os
- Partial previa: placenta covers portion of internal os
- Marginal previa: edge of placenta reaches margin of the os
- Low-lying placenta: implanted in the lower uterine segment in close proximity but not extending to the internal os
- Vasa previa: fetal vessel may lie over the cervix
- Bleeding from placenta previa results from small disruptions in placental attachment during normal development and thinning of lower uterine segment during third trimester → may stimulate further uterine contractions → further placental separation and bleeding
- Placenta accreta: **superficial attachment of the placenta to uterine myometrium** → causes inability of placenta to properly separate from uterine wall after delivery of fetus → profuse hemorrhage
  - **Average blood loss with accreta = 3000-5000 mL → 2/3 women require hysterectomy at time of delivery esp. with uterine atony**
- Increta: placenta invades myometrium
- Percreta: placenta invades through myometrium to uterine serosa
- Fetal complications associated with previa: preterm delivery and its complications, preterm PROM, intrauterine growth restriction, malpresentation, vasa previa, congenital abnormalities
- Painless vaginal bleeding!!! Usually occurs after 28 weeks of gestation
- Placenta accreta and increta usually asymptomatic
- **r/f:** prior c-section, multiple gestations, multiple induced abortions, AMA
- Vaginal exam contraindicated!!! Digital exam can cause further separation
- **Dx: ultrasound** (can be over diagnosed with full bladder) – **transvaginal sonography** > transabdominal sonography
- **Tx:** strict pelvic rest (**no intercourse**) and modified bed rest
  - **Blood transfusion may be necessary so get a type and screen if you discover previa via U/S**
  - C-section is preferred delivery
  - Give Rhogam if Rh-
- Some studies show that deliver btwn 34-37 weeks may be optimal

<b>Circumvallate placenta</b>	Occurs when the membranes double back over the edge of the placenta, forming a dense ring around the periphery of the placenta. Often considered a variant of placental abruption, it is a major cause of second-trimester hemorrhage
<b>Placenta previa</b>	Occurs when the placenta develops over the internal cervical os. Types include complete, partial, and marginal
<b>Placenta accreta</b>	Abnormal adherence of part or all of the placenta to the uterine wall. May be associated with a placenta in normal locations, but incidence increases in placenta previa
<b>Placenta increta</b>	Abnormal placentation in which the placenta invades the myometrium
<b>Placenta percreta</b>	Abnormal placentation in which the placenta invades through the myometrium to the uterine serosa. Occasionally, placentas may invade into adjacent organs such as the bladder or rectum
<b>Vasa previa</b>	Occurs when a velamentous cord insertion causes the fetal vessels to pass over the internal cervical os. Also seen with velamentous and succenturiate placentas
<b>Velamentous placenta</b>	Occurs when blood vessels insert between the amnion and the chorion, away from the margin of the placenta, leaving the vessels largely unprotected and vulnerable to compression or injury
<b>Succenturiate placenta</b>	An extra lobe of the placenta that is implanted at some distance away from the rest of the placenta. Fetal vessels may course between the two lobes, possibly over the cervix, leaving these blood vessels unprotected and at risk for rupture

- Course of action: 1. Stabilize patient 2. Prepare for catastrophic hemorrhage 3. Prepare for preterm delivery
- **For accreta/increta/percreta: plan for total abdominal hysterectomy at the time of c-section, schedule delivery at 34-37 weeks, plan ahead and have back-up available (counsel pt. on hysterectomy and blood transfusion)**
- Accounts for 20% of antepartum hemorrhage
- Complications of placenta previa include **hemorrhage, preterm labor with rupture of membranes and fetal malpresentation.**

#### **Placental abruption (abruptio placentae):**

- Premature separation of normally implanted placenta from uterine wall after 20<sup>th</sup> week, resulting in hemorrhage between uterine wall and placenta – 50% occur before labor and after 30 weeks gestation
- **MC cause third trimester bleeding**
- **r/f:** hypertension, cocaine use, trauma, multiparity, smoking
- Primary cause: unknown – maternal HTN, prior history of abruption, maternal cocaine use, external maternal trauma, rapid decompression of overdistended uterus
- **Sx:** third trimester vaginal bleeding with severe abdominal pain and / or frequent strong contractions (30% have no symptoms)
- **Presentation:** vaginal bleeding > uterine tenderness / abdominal or back pain > abnormal contractions / increased uterine tone > fetal distress > fetal demise
- **Physical exam:** vaginal bleeding and firm, tender uterus with small frequent contractions, 20% present with no bleeding (concealed hemorrhage)
- Couvelaire uterus - life threatening condition that occurs where there is enough blood from abruption that markedly infiltrates myometrium to reach the serosa, especially at the cornea, that gives the myometrium a bluish purple tone that can be seen on the surface of uterus (seen on c-section)
- **Dx:** clinical
- Negative findings on US don't exclude placental abruption
- Confirmed by inspection of placenta at delivery → presence of retroplacental clot with overlying placental destruction confirms diagnosis
- Additional findings: hypovolemic shock, consumptive coagulopathy
- **Tx:** stabilize pt., prepare for possibility of future hemorrhage (blood products, crystalloid fluids, prompt delivery to control hemorrhage), prepare for preterm delivery, deliver if bleeding is life threatening or fetal testing is non-reassuring
- 30% of all third-trimester hemorrhage – seen most often with chronic HTN, preeclampsia, cocaine, methamphetamines, or hx of abruption
- **Abruptio placentae** is a condition of premature separation of the placenta from the uterus. Abnormal placenta-uterus separation may lead to significant fetal and maternal stress. One of the most common maternal complications is a **consumptive coagulopathy**. Placental separation results in intravascular and retroplacental coagulation. This excessive coagulation depletes platelets, fibrinogen and other clotting factors, leading to **thrombocytopenia** and **hypofibrinogenemia**, as well as an increase in the INR and the activated partial thromboplastin time. If placental abruption is a suspected cause of third trimester bleeding, laboratory evaluation of the above values should be obtained early in the management plan. If abnormalities are found, component therapy should be initiated via transfusions of platelets and fresh frozen plasma.

#### **Augmentation / Induction**

- Augmentation: increase already present contractions → prostaglandins, oxytocic agents, mechanical dilation, artificial ROM
- Success of induction (achieving vaginal delivery) is higher with favorable cervical status defined by Bishop score

- Bishop score 5 or less may lead to failed induction as often as 50% of the time → PGE2 gel, pessary or miso often used to ripen cervix (reduce risk of c-section)

Bishop Score to Assess Cervical Favorability					
Cervix	Score				Bishop Score Modifiers
	0	1	2	3	
Position	Posterior	Mid-position	Anterior	N/A	<b>Add 1 point for:</b> <ul style="list-style-type: none"> <li>• Preeclampsia</li> <li>• Each previous vaginal delivery</li> </ul>
Consistency	Firm	Medium	Soft	N/A	
Effacement	0 - 30%	30 - 50%	60 - 70%	> 80%	<b>Subtract 1 point for:</b> <ul style="list-style-type: none"> <li>• Postdate pregnancy</li> <li>• Nulliparity</li> <li>• PPROM</li> </ul>
Dilation	Closed	1 - 2 cm	3 - 4 cm	> 5 cm	
Station	-3	-2	-1	+1, +2	

- c/i: asthma, glaucoma, prior section, non reassuring fetal testing
- done via Pitocin or amniotomy
- augmentation: inadequate contractions or prolonged phase of labor measured via intrauterine pressure catheter that determines absolute change in pressure during contraction and estimates strength of contractions
  - oxytocin and amniotomy
- >160hr fetus: fetal distress secondary to infection hypoxia, anemia are of concern
- Decel >2 min with HR <90 = immediate action

#### Fetal HR Tracing:

- Early deceleration: begin and end at same time as contractions d/t increased vagal tone secondary to head compression during contraction
- Variable deceleration: occur at any time – drop more precipitously than either early or late decelerations d/t cord entrapment under fetal shoulder / around neck and compressed with each contraction
- Late deceleration: start at peak of contraction and return to baseline after contraction finished → uteroplacental insufficiency (MOST WORRISOME)
- **Repetitive decels: fetal scalp electrode often used** (c/i: hx of maternal hepatitis or HIV or fetal thrombocytopenia)
- Category I: normal fetal heart rate tracing characterized by normal baseline, moderate variability, and no variable or late decelerations
- Category II: indeterminate fetal heart tracing – many variety of fetal heart tracings (variable and late decelerations, bradycardia, tachycardia, minimal variability, marked variability, absent variability without decel)
- Category III: abnormal fetal heart rate tracing – absent fetal heart variability and recurrent late or variable decels or bradycardia OR sinusoidal pattern consistent with fetal anemia
- Uterine contractions = Montevideo units
- IUPC: determines timing and strength of contractions – **adequate with >200MV units**
- If fetal HR monitor not reassuring, use fetal scalp pH to assess fetal hypoxia and acidemia, **reassuring when pH >7.25**, bad when <7.20; normal fetal pulse ox >30%
- VEAL CHOP
  - Variables → cord compression
  - Early decels → head compression
  - Accels → OK
  - Late decels → placental insufficiency (bad!!)

#### Preterm Labor: MC cause of infant morbidity and mortality in the industrialized world

- Before week 37 due to preterm contractions or cervical insufficiency: silent, painless dilation and effacement of cervix or both



- **Preterm labor** refers to **sustained, progressive uterine contractions** which lead to **cervical dilatation** and **effacement from 20 to 37 weeks of gestation**. Before 20 weeks, such contractions would be termed inevitable spontaneous abortion. After 37 weeks, such contractions are considered full-term labor. Many causative factors have been studied in regard to preterm labor, and they can be generally classified into four main groups: pathologic uterine distention, decidual hemorrhage and abruption, exaggerated response to infection or inflammation, and premature activation of the hypothalamic-pituitary-adrenal (HPA) axis secondary to maternal or fetal stress. **Uterine distention** can be pathologic in the presence of multiple gestation or polyhydramnios. **Damaged decidual blood vessels** (which can occur in maternal hypertension) can lead to placental abruption and preterm labor. **Inflammation** and **infection** can lead to a cascade which activates tissue necrosis factor, leading to increased apoptosis of the amniotic epithelial cells and premature rupture of membranes (PROM). Maternal or fetal stress can activate the **HPA axis**, which will increase adrenocorticotrophic hormone. This hormone will increase levels of circulating **cortisol**. Cortisol increases the release of **corticotropin-releasing hormone**, which activates **prostaglandins** and can lead to **cervical ripening** and **rupture of membranes**.
- **4 primary processes:** premature activation of the maternal or fetal HPA axis, exaggerated inflammatory response or infection, abruption (decidual hemorrhage), pathological uterine distention
  - **Ehlers-Danlos syndrome** is a congenital risk factor for cervical insufficiency.
  - **Cervical insufficiency** is defined by the American College of Obstetricians and Gynecologists (ACOG) as the **inability of the uterine cervix to retain a pregnancy throughout the second trimester, in the absence of uterine contractions**. Cervical insufficiency can be congenital or, more commonly, acquired. Congenital risk factors include genetic collagen disorders, such as Ehlers-Danlos syndrome, uterine anomalies, and in utero diethylstilbestrol exposure. Acquired risk factors include cervical trauma during labor or delivery, rapid mechanical cervical dilation, and treatment of cervical intraepithelial neoplasia. Patients with cervical insufficiency may present with mild symptoms, such as pelvic pressure or vaginal discharge, or be asymptomatic. Uterine contractions are mild to absent. During late presentation, pelvic exam may reveal a soft, effaced and dilated cervix with grossly prolapsed or ruptured membranes. The diagnosis of cervical insufficiency is usually based on history of **recurrent mid-trimester loss**, risk factors, and a transvaginal ultrasound measurement of cervical length. **Cerclage placement** is recommended at **12-14 weeks** for with a history suggestive of cervical insufficiency.
  - **Avoiding coitus** should be recommended to pregnant women who have an ultrasound-confirmed shortened cervix and are at risk for preterm birth secondary to cervical insufficiency. **Cerclage placement** is recommended in pregnant women who have a prior history of preterm birth or pregnancy loss (before 28 weeks gestation) and have a **shortened cervical length (less than 25 mm)** before 24 weeks gestation. Avoiding coitus is the only restriction recommended for pregnant women who undergo cerclage or have ultrasound-confirmed cervical insufficiency. Diagnosis of cervical insufficiency is made through physical exam, ultrasound, or obstetrical history.
- Low birth weight = <2500g
- **r/f:** PROM, chorioamnionitis, multiple gestations, uterine anomalies, previous preterm delivery, maternal pre-pregnancy weight <50kg, placental abruption, maternal dz, infections, intra-abdominal dz or surgery, low SES, smoking, cocaine use, uterine malformations, cervical incompetencies, infection, low pre-pregnancy weight
- **s/s:** regular uterine contractions >4-6/hr between 20-36 weeks and: cervical dilation 2cm at presentation, 1cm or greater on serial exams, effacement of >80%; late sx: painful or painless contractions, pressure, menstrual like cramps, watery or bloody discharge, low back pain
- **dx:** US; normal cervix length = 4cm, 2cm at 24 weeks increases risk premature delivery; look for fetal fibronectin (absence = low risk delivery in next 2 weeks); vaginal cultures and UA
- **tx:** bed rest, oral or IV hydration, abx for infection, steroids for lung maturity, tocolytics, surgical cerclage
  - **hydration:** (dehydrated = increased ADH which is similar to oxytocin → bind to oxytocin receptors → contractions)
  - **Betamethasone (celestone):** reduce incidence of RDS
  - **TOCOLYSIS:** prevent contractions and labor / halt cervical change: IT'S NOT MY TIME → INDOMETHACIN, NIFEDIPINE, MAGNESIUM SULFATE, TERBUTALINE
    - **1. magnesium sulfate:** decrease uterine tone and contractions by acting as calcium antagonist and membrane stabilizer
      - s/e: flushing, headaches, fatigue, diplopia, nausea, muscle weakness, but not as severe as terbutaline and ritodrine

- **toxic levels >10mg/dL** – resp. depression, hypoxia, cardiac arrest
- with the aim to reach a therapeutic level of 3.5 to 7 mEq/L, which corresponds to **4.2 to 8.4 mg/dL**
- < 10: decreased deep tendon reflexes
- Give 6g bolus over 15-30 min then maintained at 2-3g/hr continuous infusion
- Give calcium gluconate for toxicity
- **2. Calcium Channel Blockers:** inhibit smooth muscle contractility to relax uterine muscles
  - **ss/e:** maternal hypotension, tachycardia
- **3. Prostaglandin inhibitors:** indomethacin – decrease intracellular levels of Ca and enhance myometrial gap junction function, increasing myometrial contractions
  - **s/e:** fetal complications – premature constriction of ductus arteriosus, pulmonary HTN, oligohydramnios secondary to renal failure, increased risk necrotizing enterocolitis and intraventricular hemorrhage – **MC used before 32 weeks gestation for 48-72 hours**
- **4. Oxytocin antagonist:** atosiban – decrease uterine myometrial contractions but not demonstrated an improvement in outcomes
- **5. B-mimetic agents: terbutaline / ritodrine:** may cause maternal death and cardiac events (tachycardia, transient hyperglycemia, hypokalemia, cardiac arrhythmias, pulmonary edema, MI) → black box warning beyond 24-28 hours
  - s/e both drugs? Tachycardia, headaches, anxiety, pulmonary edema, maternal death
  - infrequently used d/t complications
- **Cerclage:** for known cervical incompetence or hx or preterm birth
- **Prevention:** weekly injections of **17 alpha hydroxyprogesterone caproate** from 16-36 weeks can reduce rate of recurrent preterm death

### Premature Rupture of Membranes

- pPROM = preterm rupture of membranes = before 37 weeks – most women go into spontaneous labor within 24 hours after PROM
- PROM = before onset of labor
- Prolonged rupture of membranes = >18 hours
- Major risk = infection or cord prolapse
- **Pooling of amniotic fluid in the vaginal fornix on physical exam** would be expected to be seen in this woman who is likely presenting with **premature rupture of membranes (PROM)**. PROM refers to membrane rupture before the onset of labor or regular uterine contractions. The classic clinical presentation of PROM is a **sudden “gush” of clear or pale yellow fluid** from the vagina that occurs after 38 weeks of gestation. Direct observation of amniotic fluid coming out of the cervical canal or pooling in the vaginal fornix is pathognomonic of PROM. The diagnosis of premature rupture of membranes is clinical and is generally based on visualization of amniotic fluid in the vagina of a woman who presents with a history of leaking fluid. Laboratory tests are used to confirm the clinical diagnosis when it is uncertain. If PROM is not obvious after visual inspection, the diagnosis can be confirmed by **testing the pH of the vaginal fluid**, which is easily accomplished with **Nitrazine paper**. **Amniotic fluid usually has a pH range of 7.0 to 7.3** compared to the normally acidic vaginal pH of 3.8 to 4.2. The strips will turn blue if the pH is greater than 6.0 and indicates ruptured membranes. A second confirmatory test is the **presence of arborization, or ferning, on microscopic evaluation of the vaginal fluid**. Fluid from the posterior vaginal fornix is swabbed onto a glass slide and allowed to dry for at least 10 minutes. Amniotic fluid produces a delicate ferning pattern. In equivocal cases, ultrasound can be performed to look for a reduction in amniotic fluid volume, or oligohydramnios. If the patient has a normal amniotic fluid volume, it is very unlikely that she has experienced rupture of membranes, even with a seemingly convincing history.

### Preterm Rupture of Membranes

- **Dx:** gush of fluid from vagina
  - r/o incontinence,
  - pooling on sterile speculum exam

- positive nitrazine and fern test
  - ultrasound to examine amniotic fluid
  - Amnisure test
  - amniocentesis dye test (tampon test)
  - AVOID DIGITAL EXAM
- Tx: 32-36 weeks → risk of prematurity = risk of infection (MC to **deliver at 34 weeks**)
  - 37 weeks: hospitalize and monitor, induce labor via prostaglandin cervical gel or oxytocin
  - **20-36 weeks:** steroids, Use of abx in PPROM leads to longer latency period prior to onset of labor → ampicillin +/- erythromycin +/- tocolysis, +/- steroids recommended for PPROM
    - MC concern is **chorioamnionitis**, abx recommended for women with prolonged ROM and women with unknown GBS status
    - Daily BPP / NST, amniocentesis to monitor lung maturity
- What to do first? Get a microscopic eval of vaginal fluid → nitrazine testing or fern test

### Fetal Complications

- Small for gestational age: explore underlying etiology; no indication to expedite delivery in SGA fetuses who have been consistently small throughout pregnancy; if they've fallen off growth curve, do BPP
- LGA: EFW greater than 90<sup>th</sup> percentile, **birth weight >4500g** – treat by inducing labor before attainment of macrosomia – avoid gaining too much weight and getting PDM; maternal obesity = r/f
- Oligohydramnios: in absence of rupture of membranes associated with 40 fold increase perinatal mortality
  - Cause: either decreased production or increased withdrawal (fluid produced by fetal lung and kidneys)
  - Dx: when **AFI <5** measured by ultrasound
  - Tx: depends on etiology – **induce labor, dilute meconium in amniotic fluid to decrease risk meconium aspiration syndrome**
- Polyhydramnios: **AFI >20 or 25** – fetal structural and chromosomal abnormalities more common in polyhydramnios – associated with maternal diabetes, hydrops, multiple gestation, neural tube defects, obstruction of fetal alimentary canal
  - Dx: ultrasound
  - Tx: increased risk cord prolapse, **ROM should be done**
- RH Incompatibility and alloimmunization:
  - woman is Rh negative and fetus is Rh positive → antibodies cross placenta and cause hemolysis of fetal RBCs → erythroblastosis fetalis / fetal hydrops → hyperdynamic state, heart failure, diffuse edema, ascites, pericardial effusion due to serious anemia
  - fetal hydrops: **accumulate of fluids in extracellular space in at least two body compartments**
  - tx: RhoGAM if there's any chance she could mix with fetal blood supply ( 300 mg of Rh Ig
  - MC time blood mixing = delivery, also give during amniocentesis – any time blood could mix
  - MC IN WOMEN FROM BASQUE COUNTRY
  - **Negative Rh(D) on blood typing and antibody screen** in a woman experiencing a spontaneous abortion is an indication of receiving Rh(D) immunoglobulin. Rh incompatibility occurs when an Rh negative mother develops antibodies against her Rh positive fetus. A mother who is Rh negative can develop antibodies **against the infant's blood** if the fetus is Rh positive. This antibody development can result in **hemolysis**. Because of this mismatch, Rh negative women are routinely given Rh(D) immunoglobulin at **28 weeks of gestation** and then again at **delivery** if the fetus is found to be Rh positive. Women undergoing miscarriage, ectopic pregnancy, uterine procedures while pregnant, or who experience any abdominal/pelvic trauma are at risk of developing maternal-fetal blood mixing and should receive Rh(D) immunoglobulin. Infants can experience anemia and **fetal hydrops** if maternal antibodies develop. **Type and screen** should be used to determine the mother's blood type. In instances of severe maternal fetal hemorrhage, **Kleihauer-Betke** test can measure the occurrence and severity of hemorrhage.
  - This medication is administered to any **Rh-negative female** whose fetus may be **Rh-positive** at **28 weeks gestation** and within **72 hours of delivery**. Patients also receive **anti-D immunoglobulin** as soon as possible within **72 hours** of the event when there is an increased risk of **fetomaternal hemorrhage**.
- Fetal Demise:

- Delivery: >20 weeks = induction of labor, <20 weeks: dilation and evacuation with mifepristone and misoprostol and test for reason why (RPR, CMV, HSV)
- Post-term pregnancy is defined as greater than 42 weeks' gestational age → increased risk for fetal demise, macrosomia, meconium aspiration, and oligohydramnios.
  - Increased fetal surveillance and labor induction are the most common management options for post-term pregnancies.

### **Maternal Complications of Pregnancy:**

**Hypertension in Pregnancy:** BP routinely decreases during pregnancy, **reaches nadir, mid-second trimester, during third, BP will slowly increase back to baseline**

- **Preeclampsia:** triad – nondependent edema, hypertension, proteinuria
  - **Cause:** arteriolar constriction and intravascular depletion secondary to generalized transudative edema – imbalance in prostacycline and thromboxane
  - **r/f:** those related to manifestations of the dz (chronic HTN or renal disease) and those related to immunogenic nature of preeclampsia – MC risk factor = nulliparity
    - primary dz: chronic HTN, chronic renal dz, collagen vascular disease (SLE), pregestational diabetes, African American, maternal age
    - **autoimmune disease puts patient at high risk**
    - primary immunogenic related: nulliparity, previous preeclampsia, multiple gestation, abnormal placentation, new paternity
    - family hx: female relatives of parturient, mother-in-law, cohabitation <1
  - **gestational HTN:** BPs > 140/90 on at least 2 occasions 4-6 hours apart when pt. is seated and 24 hour urine >300mg
  - **mild:** third- trimester BP >140 systolic or 90 diastolic on two occasions at least 6 hours apart with proteinuria >300mg/24 hours and nondependent edema (face and/or hands) – edema is not essential to be diagnosis of preeclampsia, proteinuria of 1+ or greater on clean catch urine dipstick on two occasions or 2+ greater
  - **severe:** severe headache not relieved by acetaminophen, visual changes, scotoma, SBP>160 or DBP >110, pulmonary edema, acute renal failure with rising creatinine, oliguria <500mL/24h, RUQ pain, elevation of AST and ALT, hemolytic anemia, <100,000platelets, DIC, IUGR, abnormal umbilical dopplers
    - **goal is to prevent eclampsia, control maternal BP and delivery baby**, stabilize using magnesium sulfate for seizure prophylaxis and hydralazine or labetalol, try to hold off delivery <32 weeks, if >32 weeks – deliver
    - **can have lingering effects for weeks!!!**- continue seizure prophylaxis 24 hours postpartum or until patient improves markedly – use labetalol and nifedipine and continue for a few weeks if needed
    - **if HELLP syndrome is present, use corticosteroid tx**
    - **LOW DOSE ASPIRIN OR CALCIUM SUPPLEMENTATION IN SUBSEQUENT PREGNANCIES TO DECREASE RISK PREECLAMPSIA**
  - **tx:** delivery, induction of labor, magnesium given for seizure prophylaxis and continued 12-24 hours after, hydralazine (5mg q20min prn max: 20mg or nifedipine)
  - **eclampsia: seizures – if post-partum woman comes in with seizures, give Mag ASAP!**
- **HELLP Syndrome:** rapidly deteriorating liver function and thrombocytopenia, some develop DIC

- **Dx:** hemolytic anemia (schistocytes on peripheral blood smear, elevated lactate, elevated bilirubin), elevated AST/ALT, low platelets
- Need >2 of the following: **abnormal peripheral blood smear (schistocytes, burr cells), elevated bilirubin >1.2, low haptoglobin, significant drop in Hgb level unrelated to blood loss, AST / ALT >2x upper limit normal, platelet <100K**
- **Tx: delivery**
- **Acute Fatty Liver Disease:** elevated ammonia, blood glucose <50mg/dL, reduced fibrinogen and antithrombin III
- **Eclampsia:** grand mal seizures in preeclamptic patient (tonic clonic), 25% before labor, 50% during labor, 25% after
  - Eclampsia refers to seizures that develop as a complication of severe preeclampsia. The clinical manifestations of preeclampsia are hypertension after 20 weeks of pregnancy plus proteinuria. Severe preeclampsia is evidenced by marked hypertension (blood pressure  $\geq 160$  mm Hg systolic or  $\geq 110$  mm Hg diastolic) with evidence of end-organ dysfunction, such as visual disturbances, mental status changes, pulmonary edema, epigastric or right upper quadrant pain, elevated liver function tests, thrombocytopenia, proteinuria, oliguria, or impaired fetal growth. Most cases of eclampsia occur in the **3rd trimester**, with approximately **80% occurring during delivery or within the first 48 hours after delivery**, though **seizures may occur as late as several weeks postpartum**. Seizures are most commonly tonic-clonic and last 60 to 90 seconds. **Magnesium sulfate** is the drug of choice for eclamptic seizures. A loading dose of 4-6 g of magnesium sulfate should be administered over 15-20 minutes followed by a maintenance infusion of 1-2 g per hour. Most eclamptic seizures terminate with magnesium.
  - **Tx:** seizure management, BP control, prophylaxis against further convulsions
    - **Seizures:** ABCs, hydralazine to lower BP, magnesium sulfate to decrease seizure threshold and **continued 12-24 hours after delivery**
    - Delivery initiated after eclamptic pt. stabilized and convulsions controlled or c-section
- **Chronic HTN:** HTN present before conception, before 20 weeks gestation, or persisting >6 weeks after baby, 1/3  $\rightarrow$  preeclampsia
  - **Tx: consistent BP 140/90** – labetalol and nifedipine (methyldopa hasn't been shown to be effective), do baseline 24 hour urine collection for CrCl and baseline ECG
  - **Pregnancy induced HTN:** presents after 20 weeks but has no other symptoms

Serum Concentration MgSO <sub>4</sub> (mg/mL)	Clinical Response
4.8–8.4	Therapeutic seizure prophylaxis
8	CNS depression
10	Loss of deep tendon reflexes
15	Respiratory depression/ paralysis
17	Coma
20–25	Cardiac arrest

### Asthma in Pregnancy

- **The patient's asthma is the treatment priority. Standard therapy should be administered even in the setting of pregnancy.** Albuterol, ipratropium, prednisone are category C and are safe to use in pregnancy

### Diabetes during Pregnancy:

- **Gestational diabetes mellitus:** impairment in carbohydrate metabolism that first manifests during pregnancy – hormones act as anti-insulin agents  $\rightarrow$  increased insulin resistance / carb intolerance = elevated postprandial and occasionally fasting glucose ??beta cell hypertrophy??
  - Increased risk macrosomia and birth injuries, neonatal hypoglycemia, hypocalcemia, hyperbilirubinemia, polycythemia, 4-10fold increased chance developed T2DM later in life
  - Maternal complications: preeclampsia, hyper-acceleration of general diabetic complications, traumatic birth (shoulder dystocia)
  - Fetal complications: macrosomia, prematurity, fetal demise, delayed fetal lung maturity
  - **s/s:** usually asymptomatic, r/f: hx of previous LGA infant, obesity, age older 25 years, glucosuria, family hx diabetes, AA, asian, hispanic, American Indian
  - **Dx:** screened 24-28 weeks
    - 50g glucose load: 1hr - >130-140 = positive  $\rightarrow$  100g 3 hour oral GTT
    - Fasting: 90, 1hr: 165-180, 2hr: 145-155, 3hr: 125-140
    - BPPs and NSTs often used beginning at 34 weeks
    - **Screen at 6 weeks post partum** for diabetes + yearly intervals after that
  - **Tx:** 2200 calories/day diet with 200-220g carb + mild exercise, walking
    - **Diet and exercise controlled = class A1 (medical nutrition therapy = 1<sup>st</sup> line therapy)**

- Check levels daily after fasting overnight and every meal and each office visit
- **>105 or 2 hr >120 may require insulin**; orals not routinely used in US
- Induce labor at 40 weeks if well controlled; poorly controlled = 38 weeks (risk macrosomia)
- Doesn't work → insulin / oral hypoglycemic agent indicated – short acting insulin + intermediate insulin
  - Short acting = Humalog, intermediate = NPH
- Schedule delivery at 39 weeks – long acting hypoglycemic agents discontinued and blood glucose monitored every hour – maintain blood glucose <120 (macrosomia / increased risk shoulder dystocia)
- **25-35% will develop T2DM in subsequent years (lifetime risk is >50%)**
- **Recurrence = 60-90%**
- **Pregestational Diabetes:** 4x more likely to get preeclampsia or eclampsia than women without diabetes
  - Tx: pt. education, control maternal glucose, careful maternal and fetal monitoring and testing
  - **Strict control glucose levels during pregnancy** → decrease rate of maternal and neonatal complications → diet, insulin, exercise
  - Increased risk congenital anomalies
- **T1DM:** maintain A1c 6-6.5%, and monitor thyroid fn because these people at risk for autoimmune issues
  - autoimmune destruction of pancreatic islet cells → diminished / absent insulin production
  - first half of pregnancy – dosing regiment usually increased slightly
  - second half: increased significantly
  - better control using insulin pump
- **T2DM:** peripheral insulin resistance – managed prior to pregnancy with oral hypoglycemic agents or diet
  - **Most pregnancies require insulin**
  - Oral hypoglycemic agents not generally used during pregnancy bc of concerns for fetal hypoglycemia or potential teratogenicity (research hasn't really shown this tho)
  - Insulin can be substituted or supplemented
  - Weekly NSTs from 32-36 weeks and weekly modified BPP to assess amniotic fluid measurement
  - No complications: induce labor at 39 weeks
  - Goal: maintain blood glucose 100-120

#### Intrahepatic Cholestasis of Pregnancy:

- MC in third trimester
- Increased serum bile acids, elevated LFTs
- Pruritus on palms and soles
- **Tx: ursodeoxycholic acid** → increase hepatic bile flow and decrease bile acid levels
- Recurs in over ½ of pregnancies
- It is characterized by **pruritus** which is often **concentrated in the palms of the hands and soles of the feet**. Serum bile acids are almost always elevated and there is a significant **increase in intrauterine fetal demise**. The pathophysiology of fetal death is poorly understood, but may be related to fetal dysrhythmias or placental vasospasms from high bile acid levels. There is no ideal antepartum testing strategy, but regular non stress tests and biophysical profiles looking for signs of fetal compromise are often performed. A widely accepted management approach is induction of labor between 36 and 37 weeks gestation as most fetal deaths occur after 37 weeks.

#### Infectious Diseases in Pregnancy:

- 5% pregnant women have ASB and are at increased risk cystitis and pyelonephritis
- Lower UTIs treated with oral abx, **pyelonephritis treated with IV abx** and change to oral after 24-48 hours afebrile
- Pyelo can be complicated by septic shock and ARDS
- Symptomatic BV associated with preterm delivery
- BV treatment = oral metronidazole for 7 days
- **GBS = leading cause neonatal sepsis** – screening decreases neonatal sepsis → do at 35 and 37 weeks

- Chorioamnionitis diagnosed by maternal fever, uterine tenderness, elevated maternal WBC count, and fetal tachycardia – treat with IV abx and delivery
- Infections in first trimester = more likely to cause serious infections and SAB
- GBS colonization has high correlation with chorio and neonatal sepsis

#### Hyperemesis Gravidarum:

- **Dx:** woman has persistent vomiting, weight loss of >5% of pre-pregnancy body weight, ketonuria – common in molar pregnancies
- **Tx: Phenergan is 1<sup>st</sup> therapy**, followed by adding reglan, Compazine or etigan – if these fail → droperidol and Zofran
  - Persistent nausea → vitamin B6 and doxylamine, ginger and B12 also helpful
  - **Rehydrate and give electrolyte solution** – 5% dextrose
  - **If this doesn't work** → corticosteroids, acupuncture, acupressure, hypnotherapy, nerve stimulation OR feeding tubes

#### Other Medical Complications of Pregnancy:

- **Seizures:** taper down to lowest possible dose monotherapy; no seizure 2+ years, consider stopping meds – MANY congenital side effects with seizure meds: NTD, IUGR, microcephaly, low IQ, distal digital hypoplasia, low set ears, epicanthal fold, short nose, long philtrum, lip abnormalities, hypertelorism, developmental delay - phenytoin = drug of choice
- **DVT:** heparin
- **Thyroid disease:** hyperthyroid – test with serial NSTs, treat with PTU and methimazole but can cross placenta so use minimum daily dose / taper off meds; hypothyroid: increase levothyroxine supplement
- **SLE:** continue aspirin, heparin and steroids, stop using cyclophosphamide and methotrexate
- **Alcohol:** growth retardation, CNS effects, abnormal facies → aggressive coulsing, barbiturates for withdrawal (**benzo = teratogens**)
- **Caffeine:** <150mg a day is fine
- **Cigarettes:** associated with spontaneous abortion, preterm birth, abruptio placenta, decreased birth weight, SIDS, resp distress → smoking cessation programs
  - **Decreased risk preeclampsia oddly enough**
- **Cocaine:** abruptio placentae, IUGR, increased risk preterm labor and delivery
- **Opiates:** no known teratogenic risks, risk of withdrawal = miscarriage, preterm delivery, fetal death – use suboxone during pregnancy or methadone and then taper post partum – withdrawal effects = more dangerous than drug itself
- **No Depakote or valproic acid!!!!** Very very low IQs / mental retardation (Keppra is drug of choice, or lamictil)

### LABOR:

#### Normal Labor and Delivery:

- Labor: contractions that cause cervical change in either effacement or dilation or station
  - Regular uterine contractions that cause cervical change
  - Station: location of presenting part (head) in relation to maternal ischial spines
    - At the spines = 0, above the spines = -1cm, -2, below the spines = +1, +2cm
- Prodromal labor: “false labor” – irregular contractions that vary in duration, intensity, and intervals and yield little or no cervical change
- Assessed by progression of cervical effacement, cervical dilation and descent of fetal presenting part
- **Cardinal Movements:** engagement, descent, flexion, internal rotation, extension, external rotation (restitution / resolution)
  - **Engagement:** fetal presenting part enters pelvis → fetal head **descends** into pelvis → **flexion** – smallest diameter to present to the pelvis → fetal vertex undergoes **internal rotation** from OT position so that sagittal suture is parallel to anteroposterior diameter of pelvis, commonly to

occiput anterior position → vertex passes beneath and beyond pubic symphysis and **extends** to deliver → **external rotation** after head delivers so shoulders can be delivered

- **Stage 1:** onset of labor and lasts until dilation and effacement of cervix
  - Lasts ~10-12 hours in nulliparous pt. and 6-8 hrs in multiparous
  - Latent phase: onset of labor until 2-4cm of dilation (slow cervical change)
  - Active phase: extends until >9cm of dilation – time when slow of cervical change against time increases (1-1.2cm/hr)
    - A patient in **active labor** should be admitted to the labor unit. The determination of active labor compared to false labor is based on multiple criteria. **Vaginal mucus plug discharge** is a sign that membranes could have ruptured and the patient should be further evaluated. Other criteria include **cervical dilation of > 4 cm, uterine bleeding, abnormal fetal heart rate pattern, regular uterine contractions** that require the woman's focus and attention, and significant **effacement** (>80 percent). Women who are not in active labor at the time of admission are at increased risk of iatrogenic intervention such as epidural, oxytocin augmentation, and cesarean section.
  - Deceleration / transition phase: cervix completes dilation
  - Powers, passenger, pelvis all affect transit time
    - Powers – determined by strength and frequency of uterine contraction
    - If passenger is too large for pelvis, cephalopelvic disproportion (CPD) results
  - >200MV units = adequate
  - If no change for 2 hrs during active phase → c-section (**should wait up to 4 hours**)
  - Amniotic fluid rupture
  - Repeat **digital cervical examinations** should be done at varying intervals during labor to confirm that **dilation of cervix is progressing** at an appropriate rate and to determine **cervical effacement** and **fetal station**. The **first** cervical examination should be performed at the time of **admission**. During the **first stage** of labor, the examinations should be performed **every two to four hours**. During **stage two**, **every one to two hours** is sufficient. **When the woman feels the urge to push**, a cervical examination is done to **determine whether the cervix is completely dilated**. If the woman has chosen to have anesthesia, a cervical examination should be done **prior to its administration**. Additionally, if the **fetal heart rate becomes irregular**, a cervical exam is indicated to **evaluate for complications** such as cord prolapse or uterine rupture. The results of the examination can be documented on a **partogram**, a graphical representation of the progression of dilation, in addition to the medical record.
- **Stage 2:** time of full dilation until delivery of infant
  - Cervix is completely dilated
  - Prolonged if >2hrs in nulliparous, >1 in multiparous
  - Epidurals have profound effect on length of second stage (due to decreased sensation to push)
  - Early and variable decelerations are common during second stage
  - Forceps: blades placed around fetal head and shaped with cephalic curve to accommodate head → use with full dilation of cervix, ruptured membranes, engaged head at least +2 station, absolute knowledge of fetal position, no evidence CPD, adequate anesthesia, empty bladder
  - Vacuum: vacuum cup that is placed on fetal scalp and suction device that is connected to cup to create vacuum
  - Forceps vs vacuum: vacuums associated with higher rate cephalohematomas and shoulder dystocias, forceps associated with higher rate of facial nerve palsies
  - Forceps = higher rate 3<sup>rd</sup> / 4<sup>th</sup> degree perineal lacerations
- **Stage 3:** begins after delivery of infant and ends with delivery of placenta
  - Placental separation occurs within 5-10 minutes of delivery of infant – up to 30 min = wnl
  - 3 signs placental separation: cord lengthening, gush of blood, uterine fundal rebound as placenta detaches from uterine wall – gentle traction on cord – must put pressure on suprapubic to avoid perineal trauma / uterine inverting or prolapse
  - Retained placenta: placenta doesn't deliver within 30 min after infant (common in preterm) – remove by manual extraction – hand in intrauterine cavity and fingers used to shear placenta from surface of uterus OR curettage
  - Laceration repair:
    - 1<sup>st</sup> degree: mucosa / skin



- 2<sup>nd</sup> degree: extend into perineal body but not involving anal sphincter
- 3<sup>rd</sup> degree: extend into or through anal sphincter → repair anal sphincter with several interrupted sutures
- 4<sup>th</sup> degree: anal mucosa itself is entered → repair anal sphincter
  - “buttonwall” – laceration through rectal mucosa into vagina, but with sphincter still intact
- Lacerations:
  - **Vaginal tears** that occur during labor and delivery fall into one of four categories. A **first-degree tear** involves only the skin in the perineal area. A **second-degree tear** involves the superficial perineal area as well as deeper tissues. **Third and fourth-degree** lacerations go beyond the perineal area and into the muscle of the rectal sphincter. A **fourth-degree** laceration will breach the **mucosa of the rectum** increasing the risk of infection. A fourth-degree laceration repair that has become separated and is infected should be treated with **antibiotics, debridement, and secondary repair**. Patients with separation of a first or second-degree laceration repair can be managed expectantly and the wound can be left to heal by secondary intention. In the case of a third or fourth-degree laceration separation, secondary repair is indicated. Any infection should be treated with systemic antibiotics and evaluated for possible debridement.
  - **Third-degree lacerations** are perineal lacerations that involve the **perineal fascia, musculature**, and involve **the external and internal anal sphincter**. Obstetric perineal lacerations are graded depending on the severity and involvement of the perineal structures. First and second-degree lacerations are easily repaired in the delivery room with suture material, but depending on the extent of a third or fourth-degree laceration, more extensive repair in an operating room may be necessary for adequate anesthesia and management of aseptic conditions. Patients with third and fourth-degree lacerations are started on **broad-spectrum antibiotics** to help prevent postoperative infection. In the past an **episiotomy** was used to allow for easier delivery of the fetus, however, it is used less frequently now and reserved for patients who are at greater risk of more severe perineal lacerations during delivery, such as nulliparous woman requiring vacuum-assisted or forcep delivery. Complications of third- and fourth-degree lacerations include wound breakdown, **infection, incontinence, and prolapse**.
- Repetitive late decels, bradycardia, loss of variability = non-reassuring fetal status
- **Give pt. face mask O2, turn on left side to decrease IVC compression and increase uterine perfusion and discontinue oxytocin**
- If a prolonged deceleration is felt to be the result of uterine hypertonus (a single contraction lasting 2 minutes or longer) or tachysystole (greater than five contractions in a 10-minute period), which can be diagnosed by palpation or examination of the tocometer, the patient can be given a dose of terbutaline to help relax the uterus.
- Episiotomy: incision made in perineum to facilitate delivery (hasten delivery and prevent shoulder dystocia) – median or mediolateral

#### **Pain Management:**

- Morphine sulfate used for pain (don't use too close to delivery)
- Pudendal nerve: travels posterior to ischial spine – used in case of operative vaginal delivery with forceps or vacuum
- Local anesthesia: use for those requiring episiotomy, or repair of vaginal, perineal, periurethral laceration
- Epidural / spinal: **placed in L3-L4** when in active phase of labor – given as bolus for epidural
- General anesthesia: urgent c-section; risks: risk of maternal aspiration and risk of hypoxia to mother and fetus

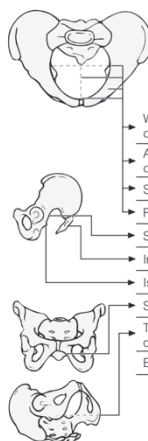
#### **C-section:**

- Reasons: biologic (higher rates multiples gestations, older population with more medical disorders, overweight; patient preference for election section; clinician preference
- **MC reason:** failure to progress in labor – pelvis too small / fetus too large

- **Vaginal birth after caesarean:** has to be **Kerr (low transverse) or Kronig (low vertical incision)** – greatest risk is rupture of prior uterine scar

### Obstruction, Malrepresentation, malposition

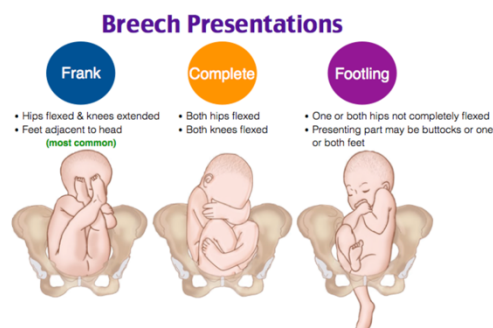
- MC form of delivery = spontaneous vertex vaginal
- **Cephalopelvic Disproportion (CPD):** MC indications for c-section = failure to progress (aka CPD) – due to pelvis too small, fetal presenting part too large, contractions inadequate
- **Gynecoid, android, anthropoid, platypelloid**
- Obstetric conjugate: distance between sacral promontory and midpoint of symphysis pubis – shortest anteroposterior diameter of pelvic inlet
- Tx: attempt labor → c-section



	<b>Gynecoid</b>	<b>Android</b>	<b>Anthropoid</b>	<b>Platypelloid</b>
Widest transverse diameter of inlet	12 cm	12 cm	12 cm	12 cm
Anteroposterior diameter of inlet	11 cm	11 cm	>12 cm	10 cm
Side walls	Straight	Convergent	Narrow	Wide
Forepelvis	Wide	Narrow	Divergent	Straight
Sacrosciatic notch	Medium	Narrow	Backward	Forward
Inclination of sacrum	Medium	Forward (lower 1/3)	Wide	Narrow
Ischial spines	Not prominent	Not prominent	Not prominent	Not prominent
Suprapubic arch	Wide	Narrow	Medium	Wide
Transverse diameter of outlet	10 cm	<10 cm	10 cm	10 cm
Bone structure	Medium	Heavy	Medium	Medium

### Breech

- 3-4% of all singleton deliveries
- **Frank:** flexed hips, extended knees, feet near fetal head
- **Complete:** flexed hips, one or both knees flexed, with at least one foot near the breech
- **Incomplete / footling:** one or both hips not flexed so foot or knee lies below breech in birth canal
- **Dx:**
  - abdominal exam using Leopold maneuvers – fetal head palpated near fundus while breech is palpated in pelvis
  - vaginal exam: palpated gluteal cleft and anus or lower extremity
  - confirmed with US
  - bedside doppler: fetal heart heard in upper uterus
- **Tx: deliver past 37 weeks in case baby turns or try turning them at 39 weeks with anesthesia – if secone time fails, pt. often has section**
  - External cephalic versions of breech
  - Trial of vaginal delivery
  - Elective section
  - Complications: cord prolapse, entrapment of fetal head, fetal neurologic injury
  - **Trial:** favorable pelvis, flexed head, est. fetal weight between 2000-3800g, frank or cpmlete breech
  - **c/i:** nulliparity, est fetal weight >3800g, incomplete breech presentation
- **External cephalic version** is an elective procedure used to **reorient a fetus who is in breech position**, with the goal of **successful vaginal delivery** and **avoidance of cesarean section**. In women who opt to undergo external cephalic version, the rate of cesarean sections is decreased, although it remains higher than the rate of cesarean sections performed in patients with cephalic presentations who did not require external cephalic version. The procedure involves **pharmacologic uterine relaxation** and **external bimanual manipulation of the fetus** to cause the fetus to perform a forward or backward somersault and reorient cephalically. The procedure should **not** be attempted in cases of **placenta previa** and **previous cesarean section**. Likelihood of successful external cephalic version is increased in patients who are **black**, who present with **nonlongitudinal fetal lie**, and whose **fetus has an unengaged presenting part**. **Nonlongitudinal lie aids in external cephalic version** because the fetus need not be rotated 180 degrees, and because transverse lie is an inherently unstable lie. Risks of external cephalic version include stillbirth, placental



abruption, emergency cesarean section, cord prolapse, vaginal bleeding, rupture of membranes, maternofetal transfusion, and transient abnormal changes in fetal heart rate. The rate of serious complications in large studies was found to be very low.

#### Other Malpresentation:

- face can labor
- brow can labor if brow convert to vertex or face to deliver
- compound = fetal extremity presenting alongside vertex or breech – common complication = umbilical cord prolapse – use US to determine if hand or feet are presenting
  - **Umbilical cord prolapse** refers to an umbilical cord which presents **outside the cervix** before the fetus has been delivered. It represents an obstetrical emergency, as pressure on the cord from the descending fetus can inhibit blood flow and lead to fetal harm or demise. Presenting signs of umbilical cord prolapse are often **changes on fetal heart rate tracings**, such as **variable late decelerations** or **prolonged bradycardia**. On vaginal exam, the umbilicus may be visible (overt umbilical cord prolapse) or not visualized (occult prolapse). **Manual elevation of the fetal head with two fingers** is the most common intervention employed to keep the umbilical cord free from compression. Placing the patient in **Trendelenburg position** or in a **knee-to-chest** position with her face towards the floor may change the relationship between the fetus and the umbilical cord and alleviate cord compression. **Emergency cesarean section** is the standard obstetrical management. If a delay in cesarean section is anticipated, such as waiting for an open surgical suite or transporting the patient from a remote location, the **tocolytic terbutaline 0.25 mg** can be given subcutaneously to **decrease uterine contractions** and **alleviate pressure on the cord**.
- shoulder: delivered via section bc of increased risk of cord prolapse, uterine rupture, difficulty of vaginal delivery – diagnose with US
- **transverse fetal lie: ordering an abdominal ultrasound to confirm a transverse fetal lie** is the next best step in caring for this patient. Ultrasound allows the practitioner to determine the precise position of the fetus. Imaging also allows for determination of potential underlying causes including maternal pelvic abnormalities, uterine abnormalities, and placenta previa. A vaginal examination should not be performed until underlying abnormalities are excluded, particularly, placenta previa. External version can be attempted in cases of a singleton gestation without any underlying abnormality. **External version is typically attempted at 37 weeks gestation** which allows for ample amniotic fluid while restricting the time period for recurrence.

#### Malposition:

- OA = optimal (occiput anterior)
- **OT/ OP = malposition** – particularly in first part of labor → seen more in epidural use (more likely to stay in OT/OP rather than move)
  - Women who are previously **nulliparous** are at **greater risk of an occiput posterior fetal malposition at birth**. This is the most common fetal malposition and puts the fetus and mother at increased risk. Occiput posterior delivery is associated with **greater risk of lower Apgar scores, umbilical artery acidemia, and neonatal intensive care admission**. Maternal risk includes **anal sphincter injury** or failed vacuum or forceps delivery. The normal fetal position at delivery is occiput anterior. Diagnosis of fetal malposition is typically made during the second stage of labor and can be confirmed with ultrasound. Manual rotation may be attempted before delivery although operative vaginal delivery or cesarean delivery are also options.
  - r/f: nullparity, maternal age >35 years, obesity, previous OP delivery, small pelvic outlet, gestational age >41 weeks, birthweight >4000 grams, anterior placenta, epidural anesthesia
- **MC** position at onset of labor LOT or ROT but then converts to OA
- OT position with platypeloid pelvis = try to rotate or vacuum

#### Obstetric Emergencies:

- Fetal Bradycardia: FHR <100-110, prolonged decel: longer than 10 minutes = bradycardia
  - Preuterine (maternal hypotension or hypoxia – seizure, AFE, PE, MI resp. failure, recent epidural or spinal placement leading to hypertension), uteroplacental (placental abruption, infarction, hemorrhaging previa, uterine hyperstimulation), postplacental (cord prolapse, cord compression, rupture of fetal vessel i.e. vasa previa)

- Dx: look at mom for signs of resp. compromise, assess maternal BP and heart rate, look to see how much vaginal blood is passing (placental abruption / uterine rupture), examine pt
- Tx:
  - 1. move pt. to left or right lateral decubitus position to resolve FHR decel secondary to compressing IVC → decreased preload
  - 2. Put on oxygen face mask
  - 3. Treat underlying cause – IV hydration / ephedrine for mom hypotension- time all of these closely
- Shoulder Dystocia:
  - Risks: fetal macrosomia (>4000g), preconceptional and gestational diabetes, previous shoulder dystocia, maternal obesity, post-term pregnancy, prolonged second stage labor
  - Dx: diagnosis made when routine obstetric maneuvers fail to deliver fetus
  - Preparation: place patient in dorsal lithotomy position having adequate anesthesia – incomplete delivery of head and chin tucking against maternal perineum
  - Tx: two individuals hold patients leg, one person give suprapubic pressure
    - Mcroberts maneuver: sharp flexion of maternal hips that decreases inclination of pelvis increasing AP diameter
    - Suprapubic pressure: dislodge anterior shoulder from behind pubic symphysis
    - Rubin maneuver: pressure on accessible shoulder toward anterior chest all of fetus to decrease bisacromial diameter and free impacted shoulder
    - Wood's corkscrew maneuver: pressure behind posterior shoulder to rotate baby and dislodge anterior shoulder
    - Delivery of posterior arm/shoulder: sweep posterior arm across chest to allow bisacromial diameter to rotate to oblique diameter of pelvis and anterior shoulder to be freed
    - Zavenelli maneuver: if all above maneuvers failed: place infant head back into pelvis and perform c section – reserve for true emergency – increased risk infection
  - **Shoulder dystocia** is defined as **failure of the shoulders to spontaneously traverse the pelvis** after delivery of the fetal head, requiring **extra maneuvers** to enable delivery of the **fetal shoulders**. In a normal labor and delivery, the head is expelled first, followed by the shoulders. In normal circumstances, **gentle downward traction on the head of the fetus** is all that is needed for delivery of the shoulders. As the infant proceeds down the pelvis, the shoulders enter at an oblique angle, with the posterior shoulder in front of the anterior one. At the pelvic outlet, when the fetal head externally rotates, the shoulders switch position, becoming anterior-posterior, and the anterior shoulder slides out after the head is expelled. In **shoulder dystocia**, the shoulders descend simultaneously into the pelvis or descend anterior-posterior before rotation of the fetal head, and the shoulders become **impacted**, either anteriorly (on the symphysis pubis) or posteriorly (on the sacral promontory). Subjectively, the provider notices **gentle traction on the fetal head does not produce delivery of the shoulders**, and **extra manipulation** is needed for full delivery of the fetus. Initial approach should involve **McRoberts' maneuver** without followed by with suprapubic pressure to release the impacted shoulder. If this is unsuccessful, **delivery of the posterior fetal arm** should be tried next. **McRoberts' maneuver** is performed by two assistants who each **flex one of the mother's legs** until her thighs touch her abdomen, thus flattening the sacrum and rotating the symphysis pubis cephalically. Several other noninvasive, invasive, and surgical maneuvers exist for shoulder dystocia, but the aforementioned are the most commonly used. The goal of management is to prevent fetal asphyxia, **permanent brachial plexus palsy**, death, and fetal or maternal physical injury.

**APGAR score:**

- Performed at 1 min, 5 min

Apgar refers to a scoring method used to assess neonatal status at one minute and five minutes after birth. The five elements assessed are **appearance, pulse, reflex crying to unpleasant stimuli, color, and respirations**. Two, one, or zero points are awarded for each category. A total Apgar score of **seven or more** at one minute indicates that the infant is **not in distress** and can be returned to his mother for skin-to-skin contact. Apgar scores of **six or less** at one minute indicate **neonatal distress** and **call for clinical intervention**. If

SCORE	0 points	1 point	2 points
Appearance - Skin colour	Cyanotic/ Pale all over	Peripheral cyanosis only	Pink
Pulse (Heart rate)	0	<100	100-140
Grimace - Reflex irritability)	No response to stimulation	Grimace (facial movement)/ weak cry when stimulated	Cry when stimulated
Activity - Tone	Floppy	Some flexion	Well flexed and resisting extension
Respiration	Apnoeic	Slow, irregular breathing	Strong cry

a neonate has a pulse, but it is less than 100 beats per minute, this is most often indicative of respiratory distress, not cardiac pathology. A neonate who demonstrates a **weak cry with gasping** and a **pulse less than 100 beats per minute** should receive **positive airway pressure within one minute of birth**. After 30 seconds of positive airway pressure, the heart rate is reassessed. If the heart rate is over 100 beats per minute and spontaneous breathing has begun, positive airway pressure can be discontinued. If the pulse continues to be under 100 beats per minute, the neonate should be reassessed for adequacy of ventilation. Repositioning, suction, mask adjustment, or pressure increases may need to be made. While Apgar scores at one minute and five minutes after birth are not accurate predictors of neonatal morbidity and mortality, they are useful in **assessment of the neonate's need for further clinical intervention**.

## POSTPARTUM

**Postpartum Care / Complications:** first 6 weeks after delivery

- **Routine Care:**
  - **Vaginal:** NSAIDs or Tylenol for pain, low dose opioids for sleep, perianal care: ice packs for 24 hours for pain and edema, inspect for hematomas, hemorrhoidal meds, stool softeners, ice packs
  - **C-Section:** local wound care, look for signs of infection, wound dehiscence, manage pain with opioids (stool softener + laxative), NSAIDs for cramping pain,
  - **Breastfeeding:** oxytocin release from pituitary gland with breastfeeding stimulates postpartum uterine contractions → increasing uterine tone and decreasing risk of bleeding – passive immunity via immunoglobulins, more likely to lose weight / decreased risk T2D; letdown happens 24-72 hours after birth – warmer, firmer, tender breasts
    - **Not breastfeeding:** ice packs, tight bra, analgesics, anti-inflammatory meds
  - **Contraception:** begin OCPs 3-6 weeks postpartum (don't start until after 3 weeks breastfeeding)
  - **Normal puerperium:** immediately after delivery, uterus = at umbilicus, after 2 days → shrinks / involutes, 2 weeks → descend into pelvic cavity, 6 weeks = normal size
    - **Lochia = bleeding** can last for 4-5 weeks
    - **Menses resume 6-8 weeks postpartum** – breastfeeding = amenorrhea until finish
    - First postpartum visit = 6 weeks after delivery, perineum should be healed
    - Get hemoglobin and crit, and fasting blood glucose if GDM, do Edinburgh postnatal depression scale
    - EMPHASIZE CONTRACEPTION, vitamin supplementation for nursing mother, atrophic vaginitis treat with vaginal estrogen
- **Complications:**
  - **Post Partum Hemorrhage:** >500mL for vaginal and >1000 c-section – uterine atony, retained POCs, placenta accrete, cervical lacerations, vaginal lacerations
    - Within first 24 hours = early postpartum hemorrhage
    - **Investigate cause, start fluids and blood transfusion at the same time**
    - **>2-3 L blood loss** = coagulation factors and platelets
    - If retained placenta is a highly suspected cause of postpartum hemorrhage, immediate **digital extraction should be performed** by inserting fingers thru the cervix and into the uterus, then using them to direct and maneuver any remaining intrauterine tissue out through the vagina. If

this is unsuccessful, and bleeding continues or the patient decompensates, **curettage with a suction device or sharp curette** is recommended. Adjuvant interventions include establishing large-bore intravenous access, uterine massage, oxytocin

- Sheehan syndrome – absence of lactation secondary to lack of prolactin or failure to restart menstruation secondary to absence of gonadotropins
- **Blood loss = 10% drop in crit → get hemoglobin and crit numbers / US to show placenta fragments**
- Initially: fundal massage, IV access, IV oxytocin, hemabate, methergine, misoprostol
- Uterine atony > birth trauma > retained products of conception
- Uterine atony, trauma to birth canal, retention of fetal or placental tissue, coagulopathy or thrombin disorder
- Oxytocin is typically the first agent used, with carboprost and methylergonovine typically used in cases of continued bleeding after oxytocin use. **Carboprost, a prostaglandin analog that stimulates uterine contractility, may cause significant bronchospasm and is contraindicated in patients with asthma. Methergine = c/I in hypertension**

This can be due to prolonged labor or rapid, forceful labor, especially if the labor was induced. Maternal obesity and large-for-gestational-age newborns are also linked to atony. Other etiologies for hemorrhage include **retention of a portion of the placenta, trauma, and coagulation disorders**. Presentation of symptoms is often dramatic, with **heavy vaginal bleeding that can quickly progress to hypovolemic shock**. Because of this rapidity, diagnostic procedures are often limited to physical examination findings. Uterine atony should be first suspected and can be diagnosed by physical examination of the uterus. The **presence of a boggy uterus upon palpation with heavy vaginal bleeding or increasing uterine size is indicative of atony**. Once postpartum hemorrhage has been diagnosed clinically, a complete blood count, coagulation studies, and cross-match for possible blood transfusion should be performed. An ultrasound can be helpful in evaluating uterine size and shape or to evaluate for any remaining placental portions or blood clots. However, this method can be time-consuming and unnecessarily delay treatment which involves quickly identifying the underlying cause of hemorrhage and resuscitation and management of hemorrhage and shock. **Fluid resuscitation** should be commenced immediately, and blood transfusions should be ordered if the patient continues to show signs of shock despite aggressive resuscitation. **Oxytocin is the drug of choice for both prophylaxis and treatment of postpartum hemorrhage caused by uterine atony**. **Bimanual massage** is also indicated to help uterine contraction. If the uterus is properly contracted, evaluation and removal of placental fragments or clots must be done by manual exploration and the presence of lacerations must be ruled out. Ongoing bleeding caused by an atonic uterus, ruptured uterus or large cervical laceration may require surgical intervention, with total hysterectomy being curative for bleeding from uterus and cervix

- **Vaginal Lacerations / Hematomas:**
  - Trauma of delivery injure blood vessel without disrupting epithelium above it → hematoma
- **Endomyometritis:** polymicrobial infection of the uterine lining that often invades underlying muscle wall – MC after section but may occur after vaginal delivers as well – esp in manual extraction of placenta
  - **Sx: High fever, elevated WBC, uterine tenderness, higher suspicion after section, adnexal tenderness, peritoneal irritation, decreased bowel sounds**
  - **Dx: WBC >20K, anaerobic streptococci = MC agent, UA**
  - **Usually 5-10 days after delivery**
  - **Broad spectrum IV abx (clindamycin or gentamycin) and add ampicillin if no response in 24-48 hours, then add metronidazole if sepsis persists**
  - D&C if part of placenta retained on US
  - Single dose abx at time of cord clamping reduces risk
- **Mastitis:** regional infection of breast from skin flora or oral flora of breastfeeding baby – organisms enter erosion or cracked nipple; main cause is clogged milk ducts
  - **Focal tenderness, erythema, differences in temperature from one region of breast to another**
  - **Dx: physical exam, fever, elevated WBC**

- **Tx:** I&D + oral abx (dicloxacillin; **clindamycin with allergy**) – if unresponsive to oral – admitted for IV abx, decrease time in between feeding (feed every 45 minutes / completely empty feeds)
- **Prevent with lubricating ointments**
- **Postpartum Depression:** usually within 2-3 days after delivery, peaking at 5<sup>th</sup> and resolving within 2 weeks – sadness, disinterest
  - **Dx:** low energy level, anhedonia, anorexia, apathy, sleep disturbance, extreme sadness, other depressive symptoms > few weeks
  - **Tx:** determine if having SI/HI, involve social worker / counselor, SSRIs have good efficacy

## GYN

### Amenorrhea

#### Def:

1. no menses by age 14 + no other pubertal dvlt OR no menses by age 16 w/other pubertal dvlt → PRIMARY amenorrhea
3. Previous menstrual cycles but now absent for 3 cycles or 6 mos. → SECONDARY amenorrhea
4. Physiologic amenorrhea: seen prepubescently, during pregnancy and lactation, and post-menopause

#### **Etiology**

-Pregnancy MCC of secondary amenorrhea

Anatomic disorders → inherited or acquired

- Mullerian agenesis aka vaginal agenesis (Mayer-Rokitansky-Kuster-Hauser syndrome) – congenital absence of all or part of uterus and vagina – cervix and fallopian tubes; pt. are phenotypically and genotypically female; dx: US/MRI; tx is creation of a neovagina via dilation/surgery
- Imperforate Hymen or Transverse vaginal septum – results in distal outflow tract obstruction; tx is hymenectomy/excision
- Vaginal Atresia – lower vagina fails to develop and upper vagina is normal → primary amenorrhea / cyclic pelvic pain; p/e: vaginal dimple, pelvic imaging with US/MRI; tx: surgery or serial vaginal dilators – normal sexual intercourse is possible
- Intrauterine Synechiae (Asherman Syndrome) – scarring of endometrium that results from vigorous uterine curettage that interferes w/normal endometrial growth and shedding; tx: hysteroscopic lysis of adhesions + estrogen to stimulate the endometrium
- Cervical stenosis – can result from D&C, cone bx, or infection; tx: cervical dilation (vaginal pull through procedure)

Endocrine disorders → hypergonadotropic hypogonadism, hypogonadotropic hypogonadism, or eugonadotropic hypogonadism

- *Hypergonadotropic hypogonadism (premature ovarian failure)* → Primary ovarian dysfunction (not from hypothalamus or pituitary)
  - Def: loss of oocytes before age 40
  - **Dx: two FSH levels >40 drawn at least 1 mos apart**
  - Phys: Gonadal dysgenesis = no synthesis of ovarian hormones d/t absence of follicles
  - Etio: Turner syndrome (45, XO; accts for >50%), other inherited chromosomal abnormalities, acquired via chemotherapy, radiation, infection, or autoimmune processes
    - Sxs of Turner syndrome: short, no secondary sex features, infantile genitalia, shield-like chest, webbed neck
  - Tx: hormone replacement therapy (helps breast dvlt and bone strength)
- *Hypogonadotropic hypogonadism* → primary hypothalamic-pituitary dysfx
  - Def: decr in gonadotropic stimulation of ovaries = loss of follicles = low ovarian hormones
    1. Hypothalamic d/o: inherited d/o (Kallman syndrome –assoc with midline facial abn and loss of smell), acquired d/o (brain tumor, physical stress, weight loss, exercise, pseudocyesis, etc)
      - tx: hormone replacement therapy, behavior modification/therapy if acquired
    2. Pituitary d/o: inherited d/o's resulting in hypoplasia, acquired (most often from prolactinoma, metastatic tumor, Sheehan's syndrome)
      - tx: hormone replacement therapy, resection (if tumor)
    3. Chronic illnesses: end-stage CKD, AIDS, adv liver ds – can cause hypogonadotropic amenorrhea
- *Eugonadotropic hypogonadism* → amenorrhea w/out abnormal gonadotropic levels
  - Etio: PCOS, congenital adrenal hyperplasia, hyperprolactinemia, hypothyroidism
  - Tx: hormone replacement therapy, tx underlying illness

Additional way to think about secondary amenorrhea

Hypothalamus dysfx (35%) → disrupted normal GnRH secretion = less FSH or LH secretion

Etio: hypothalamic d/o, anorexia, exercise, stress, nutritional deficiency, systemic ds (celiac's)

Dx: normal or low FSH/LH, low estradiol, normal prolactin

Mng: stimulate gonadotropin secretion → clomiphene citrate, Menotropin

Pituitary dysfx (19%) → MC is a prolactin secreting pituitary adenoma

Dx: low FSH/LH, high prolactin; MRI of pituitary sella may show tumor; prolactin inhibits GnRH secretion

Mng: transsphenoidal surgery

Ovarian d/o: PCOS, premature ovarian failure (follicular failure or follicular resistance to LH/FSH), Turner's syndrome

Sxs: signs of estrogen deficiency – hot flashes, sleep/mood disturbances, dyspareunia, dry/thin skin, atrophic vaginitis

Dx: High FSH/LH and low estradiol = ovarian abn (primary d/o)

Normal or low FSH/LH = pituitary or hypothalamus d/o (secondary or tertiary d/o)

-Progesterone challenge test: 10mg medoxyprogesterone x 10 days

+ withdrawal bleeding = ovarian d/o (pt is anovulatory but estrogen is present so endometrial

lining thickens)

-withdrawal bleeding = hypoestrogenic state or Uterine anomaly (Asherman's syndrome or

outflow tract obstruction)

Uterine d/o → Asherman's syndrome (scarring of endometrium 2/2 PPH, D&C, or endometrial infxn)

Dx: pelvic US (abn uterine stripe), Hysteroscopy (diagnostic and therapeutic)

Mng: Estrogen therapy – stimulates endometrial regeneration of denuded area

W/u

Pelvic exam – if no uterus, do genetic testing → 46 xx – Mullerian agenesis; 46 XY – androgen insensitivity syndrome

Labs – TSH, Prolactin, FSH, pregnancy test!

-**elevated prolactin warrants Brain MRI**

- Normal FSH = eugonadotropic hypogonadism; High FSH = hypergonadotropic hypogonadism; low =

hypogonadotropic hypogonadism

### Complications

Infertility → anatomic disorders usually corrected; hypergonadotropic pts may conceive w/donor egg + IVF, hypogonadotropic pts may be tx w/pulsatile GnRH, Eugonadotropic pts may conceive w/fertility aid (clomiphene citrate)

- **Secondary amenorrhea** is the absence of menses for at least **three** months in a woman who previously had regular menstrual cycles, or for **six** months in a woman who previously had irregular menstrual cycles. Secondary amenorrhea can result from hypothalamic dysfunction, pituitary dysfunction, or ovarian dysfunction. The most common cause of secondary amenorrhea is **pregnancy**, which must be ruled out before proceeding with further evaluation. Focused history and exam should reveal **risk factors** such as **stress**, significant change in **weight, diet** or **exercise**. **Hirsutism, acne**, or a history of **irregular menses** suggests **hyperandrogenism**.
- **Visual field defects** and **polyuria** suggests **hypothalamic-pituitary disease**. **Galactorrhea** suggests **hyperprolactinemia**. **Hot flashes** and **vaginal dryness** indicate **estrogen deficiency**. Medications known to cause amenorrhea, such as **oral contraceptives, danazol, or metoclopramide**, should be reviewed with a patient. Initial laboratory evaluation should include **follicle-stimulating hormone (FSH)** to rule out primary ovarian insufficiency, **serum prolactin** to rule out hyperprolactinemia, and **thyroid-stimulating hormone (TSH)** to test for thyroid disease. In general, secondary amenorrhea is managed by correcting the etiology if possible, helping the woman achieve fertility if desired, and preventing any complications of the etiology (for example, preventing osteoporosis in a woman with estrogen deficiency).
- **Hypothalamic amenorrhea** caused by increased exercise is managed by encouraging the patient to **consume adequate calories** and **reduce exercise**, although affected women are often hesitant to do so. This type of amenorrhea is common in female athletes caused by the suppression of the hypothalamic-pituitary-ovarian axis due to an **energy deficit stemming from stress, excessive exercise, or disordered eating**. Treatment of this condition involves nutritional rehabilitation as well as a reduction in stress and exercise levels. Menses typically return after correction of underlying nutritional deficit (female athlete triad)

### Benign Epithelial Disorders of the Vulva and Vagina

- **Lichen sclerosis**: inflammatory dermatosis with most significance postmenopausal women – clobetasol 1-2x/d for 6-12 weeks then maintenance schedule of topical steroid



- **Lichen planus:** uncommon inflammatory skin condition that can affect nails, scalp, skin – chronic eruption of shiny purple papules with white striae on the vulva; can also be associated with vaginal adhesions; clobetasol 1-2x/d 6-12 weeks
- **Lichen simplex chronicus:** thickened skin with accentuated skin markings and excoriations due to chronic itching and scratching; medium to high potency topical steroid 2x/d for 6 more weeks
- **Vulvar psoriasis:** silvery-red scaly patches on genital area; topical steroids, UV light
- **Hx:** vulvar itching, irritation, burning, dysuria, dyspareunia, vulvar pain
- **p/e:** range: erythematous plaques, hyperkeratotic white plaques
- **dx:** biopsy
- **tx:** healthy vulvar and vaginal hygiene practices, **loose fitting clothing, unscented detergent and soap; high potency topical steroid (Clobetasol)**, no role for topical estrogens or progesterone

#### Benign Cysts / Tumors

- **Epidermal Inclusion Cysts:** MC tumor found on vulva dt occlusion of pilosebaceous duct or blocked hair follicle
- **Sebaceous cyst:** sebum accumulation
- **Apocrine sweat gland cysts: fox-fordyce disease / hidradenitis suppurativa**
- Skene's gland cysts: paraurethral glands located next to urethra meatus
- Bartholin's duct cyst and abscess (4 o'clock and 8 o'clock) – mucus secreting glands with ducts that open to hymenal ring; obstruction → cystic dilation of Bartholin's duct; tx: I&D (recurrence), word catheter placement (balloon left in place 4-6 weeks), marsupialization – for recurrent cysts 0 entire abscess / cyst is incised and cyst wall is sutured to vaginal mucosa

#### Benign Solid Tumors of the Vulva and Vagina

- Lipomas: soft pedunculated or sessile tumors composed of mature fat cells and fibrous strands – don't require removal unless large and symptomatic
- Cherry hemangiomas: elevated soft red papules aka Campbell de Morgan spots or senile angiomas (contain abnormal proliferation of blood vessels)
- Urethral caruncles / urethral prolapse: small, red, fleshy tumors found at distal urethral meatus

#### Dysfunctional uterine bleeding

##### Definitions

- Menorrhagia: prolonged/heavy bleeding (>7 days or >80 mL); regular intervals
- Metrorrhagia: variable amounts of bleeding at irregular, frequent intervals
- Polymenorrhea: short intervals (<21 days)
- Oligomenorrhea: long intervals (>35 days)

##### Etiology

##### Organic causes

- Reproductive tract disease → pregnancy, gestational trophoblastic disease, uterine lesions, iatrogenic causes (IUDs, contraception, HRT, psychotropic agents)
  - Uterine lesions: menorrhagia or metrorrhagia d/t increase in endometrial surface area/distortion of vasculature/having friable or inflamed surface → includes endometrial CA/sarcoma, endometrial hyperplasia, submucosal fibroid, endometrial polyps, endometritis, adenomyosis
- Systemic disease → blood dyscrasias (vW disease, prothrombin deficiency, leukemia, severe sepsis), Hypothyroidism, hyperthyroidism, cirrhosis
  - Hypothyroidism assoc w/menorrhagia or metrorrhagia; hyperthyroidism assoc with oligomenorrhea and amenorrhea
  - Cirrhosis can cause excess bleeding d/t low platelets and less metabolism of estrogens

##### Endocrine causes → anovulatory vs ovulatory DUB

- Anovulatory
  - continuous production of estradiol-17 beta without corpus luteum formation and no progesterone release
  - unopposed estrogen = continuous proliferation of endometrium which eventually outgrows its blood supply and sloughs off in an irregular unpredictable pattern
- Ovulatory

- Mid-cycle spotting after LH surge

#### Dx

1. Rule out pregnancy
2. Med reconciliation
3. PE → thyromegaly, hepatomegaly, GU infections, GI problems (hemorrhoids), pelvic structural abnormalities (polyps, fibroids)
4. Labs → CBC, iron levels, TSH, coags
5. Eval of uterus → endometrial bx or hysteroscopy, pelvic US

#### Management

Structural problems can be corrected surgically → D&C (therapeutic and dx), hysteroscopy, endometrial ablation, hysterectomy  
OCPs can regularize cycles

IV estrogen can be used acutely if pt is presenting with acute hemorrhage d/t DUB

NSAIDs reduce menstrual blood loss

#### **Dysmenorrhea**

-commonly found in those who ovulate regularly

-pain usually lasts 1-2 days and is relieved by NSAIDs and OCPs

Assoc w/endometriosis: pain begins prior to menses

**Pain isn't relieved by NSAIDs and OCPs**

Often have dyspareunia as well

**Primary Dysmenorrhea: begins w/in 6-12 mos of menarche**

Patho: d/t excess PG and leukotriene production at menstruation → incr uterine contraction

-blood vessels are vasoconstricted = decr blood flow

-ischemia from contractions can cause pain

Sxs: severe cramp that start w/menses and last 2-3 days (highest in first day), lower abd pain that radiates to back/thighs, h/a, nausea, diarrhea

PE: normal

Tx: **NSAIDs = first line**, OCPs (prevent ovulation), menstrual suppression, surgical (endometrium resection)

**Primary dysmenorrhea is crampy midline lower abdominal or pelvic pain during menses without another disease process** that could account for those symptoms. **Secondary dysmenorrhea** involves the same symptoms but occurs in **women with a disorder** that could account for their symptoms, such as endometriosis, adenomyosis, or uterine fibroids. **Primary dysmenorrhea** is caused by **prostaglandins** release from the endometrium at the beginning of menses, which cause intense uterine contractions. Pain usually occurs a day or two before menstrual cycle and typically resolves within 72 hours. Women with primary dysmenorrhea commonly also complain of **diarrhea, nausea, headache, fatigue, and general malaise**. Primary dysmenorrhea usually begins in adolescence, when ovulatory cycles begin. The diagnosis of primary dysmenorrhea is based upon the presence of characteristic clinical features in the absence of demonstrable disease that could account for the pain. **Secondary dysmenorrhea** may present as **dysmenorrhea with onset at age 25 years or older, abnormal uterine bleeding (oligomenorrhea, menorrhagia, intermenstrual bleeding), non-midline pelvic pain, presence of dyspareunia, progression in symptom severity, and absence of nausea, vomiting, diarrhea, back pain, dizziness, or headache during menstruation**. Evaluation should exclude other causes of lower abdominal cramping pain and determine how severe the symptoms are to help guide the treatment. The presence of risk factors or any abnormal physical exam findings, such as a palpable pelvic mass, should prompt imaging or laboratory investigation for secondary dysmenorrhea. The goal of treatment is to provide adequate pain relief. First-line treatment for primary dysmenorrhea is with **non-steroidal anti-inflammatories (NSAIDs)** or **hormonal contraceptives**. Women who do not respond to initial treatment should be evaluated further for secondary dysmenorrhea; sometimes exploratory laparoscopy is necessary for definitive diagnosis and treatment.

#### **Menopause**

Def: cessation of menstruation for 12 mos d/t termination of ovarian follicle dvlt + elevated gonadotropin levels (FSH and LH)

-avg onset is ~51 yrs; RF for early menopause include smoking and surgery (hysterectomy)

Laboratory studies are not routine but may be helpful. Typically with menopause, **follicle-stimulating hormone will be elevated, estradiol will be low**, prolactin and thyroid-stimulating hormone levels will be normal.

**An increase in estrone, decrease in estradiol, and no change in testosterone**

Phys: ovaries stop producing estrogen and only estrogen source is now from peripheral conversion of androgens; elevated FSH and LH d/t lack of estrogen – these drive ovarian stroma to produce androgens

Perimenopausal period

-Climacteric phase of menopause: transition pd before menopause; can begin as early as 35 but most women notice changes in their mid 40's

Sxs: weight gain (thought to be body's defense mechanism to retain as much estrogen as possible bc estrogen is fat soluble), menstrual irregularities

### Complications – d/t hypoestrogenemia

- Vasomotor instability → hot flashes (can be from hypoestrogenemia or from rapid w/d of estrogen); get less frequent the further along in menopause
  - Tx: estrogen + progesterone, **clonidine**, botanical products
- Osteoporosis (estrogen slows bone resorption);
  - tx: bisphosphonates, calcium supplements, SERMs, Calcitonin
- Genital atrophy: lower vagina, labia, urethra, trigone are all estrogen dependent → results in vaginismus, dysuria, urgency, incontinence
  - Premarin cream, estring, vagifem, lubrication
- Mood disturbances – fatigue, anxiety, h/a, insomnia, depression, irritability; clear link between menopause and mood isn't established
  - Counseling, botanical agents, antidepressants

### Hormone replacement therapy

-Need for HRT is based on sx's of menopause

Pros: treats hot flashes, osteoporosis, genital atrophy, and mood disturbances; may decr risk of alzheimers, osteoarthritis, colon CA, tooth loss, skin aging

Risks: endometrial hyperplasia/adenocarcinoma (if given w/out progesterone), incr risk of breast cancer

SE: nausea, erratic vaginal bleeding, headaches, breast tenderness

Regimens: Premarin (equine estrogen) on days 1-25 + Provera (days 13-25); or Premarin/estradiol/transdermal estrogen + Provera continuously

-compliance can be an issue bc of long term course and no immediate benefits

-Recent concerns that HRT increases risk of breast cancer, thromboembolism and stroke → HRT has fallen out of use slightly

Adjunct/alternative meds:

-Raloxifene – SERM w/estrogen agonist effects on bones and cholesterol but antagonistic effects on breast and endometrium

-Gabapentin, paroxetine, and venlafaxine can help w/hot flashes

-Botanicals (soy products, isoflavones, **St. Johns Wort, black cohosh**) can help w/hot flashes and depression (potential interactions and efficacy not well known)

### **Normal Physiology**

#### **HPOU axis**

Hypothalamus releases GnRH → Pituitary releases FSH and LH → FSH and LH stimulate the ovaries and uterus

-this axis drives menses and reproduction

GnRH: stimulated by dopamine, E&P, NE, Serotonin, and Endorphins

-released in a pulsatile fashion (this is important for normal physiology)

-stimulates release of FSH and LH which stimulates the ovaries and testes

FSH: causes follicle to develop into a dominant follicle

LH: the surge in LH is what allows for the rupturing of the follicle and the ovum to be released

Estrogen: released from granulosa cells of the follicle (released more in follicular phase of cycle); helps build endometrium

Progesterone: released from corpus luteum after ovulation (higher in the luteal phase of cycle); helps stabilize the uterine lining

-any time there is chronic estrogen exposure that isn't balanced by Progesterone, the endometrial lining becomes unstable and is at risk for developing CA

#### **Menstrual Cycle**

##### Ovulation

FSH: causes the follicle to develop into dominant follicle

LH: surge is needed for rupture of the follicle and ovulation to occur

-LH release from pituitary increases ~2 days just before ovulation

-LH converts the granulosa cells to release progesterone (estrogen falls ~1day before ovulation)

-as the follicle is developing, granulosa cells around it produces estrogen

-the mature follicle is technically a cyst and can be seen on US (cysts up to 3cm are normal)

-as the follicle matures, it eventually ruptures to release the ovum

Corpus Luteum: remainder of the ruptured follicle

-full of cholesterol and fat and makes progesterone

-if pregnancy occurs, **the corpus luteal cysts persists for ~13 weeks to support the pregnancy**

Abnormal Uterine Bleeding	
Type	Characteristics
Oligomenorrhea	Cycle length > 35 days
Polymenorrhea	Cycle length < 21 days
Hypomenorrhea	Scanty menstruation
Amenorrhea	Absence of menses for 6 months
Menorrhagia	Regular cycles; excessive flow, duration
Metrorrhagia	Irregular cycles
Menometrorrhagia	Irregular cycles; excessive flow, duration

-if there is no HCG present, the corpus luteal cyst gets reabsorbed

Phases:

Follicular phase (aka Proliferative phase): phase of follicle development and growth, endometrium thickens d/t estrogen release

Luteal phase (aka Secretory phase): after ovulation; ruptured follicle becomes corpus luteum which releases progesterone which enhances uterine lining (prep for implantation); without implantation the corpus luteum degenerates which decreases E&P which leads to menstruation

Uterus

Goes through phases of endometrial growth→

1. Menstrual Phase (days 0-5): shedding of the endometrium

2. Proliferative phase (days 5-13ish): early and advanced

-uterine lining grows in response to estrogen

3. Ovulation (usually ~day 14)

3. Secretory phase (days 14-28ish): Early, advanced, and late (pre-menstrual)

-uterine lining growth stabilizes with progesterone exposure → long term Progesterone thins lining

-lasts ~14 days

Normal Menstruation

Cycles occur every ~21-35 days → varies by 1-2 days each month

Cycles last ~4-6 days

Lose ~30cc of blood (20-80 is considered normal)

-base loss on pad/tampon use (heavy bleeding = changing one super tampon/pad every hour)

-can base blood loss off of CBC

### **Premenstrual Dysphoric Disorder**

#### **Premenstrual syndrome**

Def: cyclic appearance of myriad sx's that affect lifestyle/work

Epi: common; generally mild

Sx's: bloating, weight gain, constipation, anxiety, breast tenderness, depression, sugar/salt cravings, irritability

Tx: support, exercise, diet modification

SSRIs – fluoxetine, sertraline can reduce depression, anger, and anxiety = **first line**

Oral contraceptives with drospirenone or GnRH if severe

According to the American Psychiatric Association DSM-5, **mood swings, anger, irritability, sense of hopelessness or tension, and anxiety or feeling on edge** associated with severe premenstrual syndrome symptoms is defined as **premenstrual dysphoric disorder**; must be present in most of the menstrual cycles for the past year, and symptoms must be associated with significant distress or interference with usual activities (work, school, social life)

Ineffective tx: progesterone, vitamin supplements, dietary restrictions, **Exercise and relaxation techniques (A)** are first-line therapy for women with premenstrual syndrome without socioeconomic dysfunction.

### **Cervicitis**

#### **Chlamydia**

-most often an asxs infection (men and women)

If sx's: Females – mucopurulent discharge, post-coital bleeding, friable cervix, urinary sx's

Males – dysuria, urethral discharge

Dx: NAAT – high sn/sp, done via endocervical/intraurethral swab or via urine

-CDC recommends annual screening of sexually active women <26 and all others at risk

Tx: Azithromycin 1 gram PO x 1 or Doxycycline 100 mg PO x 7 days

-expedited partner tx (EPT): give written rx to patient who can give it to partner to fill at any pharmacy

-should tx all partners in last 6 mos

#### **Gonorrhea**

-caused by Neisseria gonorrhea

-can cause cervicitis/urethritis, pharyngitis, anorectal infection, disseminated gonococcal infection (septic arthritis)

Sx's and Dx: same as CT

Tx: Ceftriaxone 250 mg IM x 1 AND zith 1g PO

-zith increases the efficacy of tx

-if GC is present, the NAAT test may produce a false (-) CT, so we treat for CT anyways

#### **Herpes simplex**

-One of the most prevalent STIs

-caused by HSV1 or HSV2 (either can cause oral or genital); majority are HSV-1

-Asxs shedding is common. More shedding occurs if recent acquisition or frequent recurrence

Primary Initial Infection	Initial non-primary infection	Recurrent Infection	Subclinical Infection
No prior HSV antibody; 1 <sup>st</sup> infection with either HSV 1 or HSV 2.	Prior antibody to one HSV, new infection with the other.	Prior antibody to one HSV, recurrent symptoms to reactivation of that HSV.	Asymptomatic shedding of HSV virus.
Usually asymptomatic. If symptoms: severe pain, dysuria, fevers. Can last 2-6 weeks.	Clinically indistinguishable from recurrent →	Mild pain or tingling. Rare systemic symptoms. Shorter course (3-7 days). Triggered by stress, trauma, menses.	No symptoms (primary means of transmission).
Antivirals and topical lidocaine; bladder catheterization if retention.	Antivirals	Antivirals, either episodic or suppressive (if 4+ episodes per year).	Consider suppressive if discordant couple.

-s/s: flulike symptoms, malaise, myalgias, nausea, diarrhea, fever, vulvar burning and pruritus precede multiple vesicles

-Primary initial may have so much pain that they retain urine → catheterize

-Can do IG testing to determine if its 1 or 2 → not usually done bc tx is the same

Dx: Viral cx → obtained from unroofing an unruptured vesicle; high false – rate; test of choice

-Serum HSV ab – very sn but doesn't necessarily tell you what's going on in the genitals (may have latent HSV + another problem)

-examine vesicles, viral cultures – gold standard but sensitivity is low; tzanck smear

Tx: antivirals →

**Primary infection: valacyclovir 1 g BID x 10 days**

-if severe, IV antivirals (acyclovir 10 mg/kg IV q8 hours x 2-7 days) and bladder cath

**Recurrent HSV: topical lido + valacyclovir (1 g qd x 5 d)**

Suppressive therapy: done if >4-6 episodes p/year

-Valacyclovir 500 mg qd

-makes outbreaks less frequent/less severe

-decreases transmission rate (still possible)

### Chancroid

Chancroid – not common here

Caused by *Haemophilus ducreyi*

-forms tender papules that become seriginous friable ulcers – painful, demarcated, nonindurated ulcer located anywhere in anogenital region

-caused by *haemophilus ducrei*

Dx: R/o other causes, gram stain

Tx: Zith, ceftriazone 250mg IM, cipro 500mg bid 3 days, erythromycin 500mg 4x/day

**Chancroid** is a sexually transmitted disease which results in **painful genital ulcers**. The causative pathogen is ***Haemophilus ducreyi***, a gram-negative rod which is very contagious but rarely found in the developed world. Cases of chancroid in the developing world may be underreported due to the difficulty of definitive diagnosis. Chancroid begins as **one or several erythematous papules** on areas of the genitals most susceptible to friction, such as the glans penis or the vaginal introitus. The papules become **pustules** which **ulcerate** and form **painful open sores**, usually of one to two centimeters in diameter. The ulcers have an **erythematous base** covered by a **gray or yellow purulent exudate**. In about half of patients with chancroid, there will also be marked **lymphadenopathy** in the inguinal chain. The definitive diagnosis of chancroid is elusive because it requires a specific culture medium which is not readily available. Therefore, the vast majority of chancroid cases are diagnosed based on the presence of **all four** of the following criteria: **one or more painful genital ulcers, a negative polymerase chain reaction (or culture) for herpes simplex virus, no evidence of *Treponema pallidum* on darkfield microscopy of the ulcer exudate or a negative venereal disease research laboratory test (or other serologic test for syphilis), and clinical signs and symptoms consistent with chancroid**. Treatment of chancroid is with **ceftriaxone 250 mg** injected intramuscularly or with **one gram of oral azithromycin**.

### Lymphogranuloma Venereum (LGV)

LGV – not common here

-herpes like ulcers at first then becomes grooved inguinal mass (“groove sign”) – primary stage is local lesion that may either be papule or shallow ulcer and is often painless, transient, and can go unnoticed

-caused by Chlamydia L 1, 2, or 3

Dx: NAT (by CDC or DPH)

Tx: Doxycycline

It is most commonly found in tropical and subtropical areas of the world (e.g., **Caribbean**, East and West Africa). Risk factors include HIV infection, previously diagnosed sexually transmitted infections, **recent travel abroad**, and unprotected anal sex. There are three stages of infection, each with a specific characteristic to identify it. **Primary infection** occurs 3 to 12 days following exposure, and is characterized by a **genital ulcer** or an inflammatory reaction of the mucosa at the site of inoculation, which heals within a few days. **Secondary infection** occurs two to six weeks later and is characterized by local direct extension of the infection to regional lymph nodes, most commonly the **inguinal and femoral nodes**. At this stage, there can be severe inflammation and invasive infection, indicated by the associated systemic symptoms a patient experiences. The “**groove**” **sign** is characteristic and is caused by inflammation of the superficial and deep inguinal lymph nodes. An anorectal syndrome can also occur, which results in an inflammatory mass present in the rectum and retroperitoneum. **Late infection** results in fibrosis and strictures in the anogenital tract, which are a result of untreated infection. Diagnosis can be difficult due to the lack of standardized laboratory tests, but involves either culture, direct immunofluorescence, or nucleic acid detection of *Chlamydia trachomatis* using genital, rectal, or lymph node specimens (e.g., lesion swabs or bubo aspirate). First line treatment is **doxycycline** 100 mg orally twice daily for 21 days. Second line treatment is erythromycin 500 mg orally four times daily for 21 days. Buboec may require needle aspiration or incision and drainage.

#### PID

-ascending infection of the female GYN tract

Sxs: potentially asxs

-abdominal/pelvic/LBP

-abnormal vaginal discharge

-inter-menstrual bleeding or post-coital bleeding

-fever

-N/V if severe

PE findings:

uterine/adnexal tenderness

Cervical motion tenderness w/chandelier sign

Mucopurulent discharge from cervix

Friable cervix

Fever (<1/3)

Sequelae:

Chronic pelvic pain d/t adhesions

Infertility d/t tubal occlusion

Ectopic pregnancy → d/t salpingitis

Fitz-Hugh Curtis syndrome

RF: Age <25, Multiple partners/partners w/multiples, Hx of STD, Inconsistent condom use

Dx: → CDC guidelines

Sexually active and <= 25 OR hx of STI/multiple partners

AND

Tenderness on pelvic exam

AND

No other etio

-dx and tx if these are met (err on the side of overtx d/t high incidence of adverse outcomes)

Tx

**Outpatient: Ceftriaxone IM x1 (or Cefoxitin IM/probenecid PO) AND Doxycycline (14 days)**

**+/- add metronidazole if BV is present**

-if not getting better in 48 hours, they should return for inpatient tx w/IV abx

Inpatient: 24 hours of IV abx then sent home on 14 day course

-Cefotetan IV or Cefoxitin IV AND doxycycline (PO or IV)

**TABLE 16-1** Infectious Causes of Ulcerated Lesions

	<b>Syphilis</b>	<b>Herpes</b>	<b>Chancroid</b>	<b>LGV</b>
Incubation period (days)	7–14	2–10	4–7	3–12
Primary lesion	Papule	Vesicle	Papule/pustule	Papule/vesicle
Number of lesions	Single	Multiple	1–3, occasionally more	Single
Size (mm)	5–15	1–3	2–20	2–10
Painful	No	Yes	Yes	No
Diagnostic test	Dark-field microscopy RPR/MHA-TP/ FTA-ABS	Viral culture	Gram stain with “school of fish” appearance	Complement fixation
Treatment	Penicillin	Acyclovir	Ceftriaxone or azithromycin	Doxycycline

Indications: other condition can't be r/o, pt is pregnant, doesn't respond to OP tx, pt has severe N/V/high fever

#### Tubo-Ovarian Abscess

- Persistent PID → tubo-ovarian abscess
- **Dx:** clinical setting of PID and appreciation of adnexal or posterior cul-de-sac mass or fullness, abdominal/pelvic pain, fever, leukocytosis, left shift
- **Tx: broad spectrum abx / same for PID treatment**
- **Cefoxitin, doxycycline, and metronidazole** are an appropriate antibiotic regimen for a patient with a **tubo-ovarian abscess (TOA)**. A TOA usually follows **pelvic inflammatory disease (PID)** and infection with sexually transmitted infections, particularly ***N. gonorrhoeae*** and ***C. trachomatis***. Antibiotic choices should target these organisms. Adequate **anaerobic** coverage should be provided as well. Recurrent pelvic infections and damaged adnexal tissue can predispose individuals to formation of an abscess. Pain may be accompanied by nausea, vomiting, or fever. A pelvic ultrasound is the radiographic imaging study of choice and can show a complex multiloculated fluid collection or mass. A CT scan may be preferred in some patients to rule out other causes of a surgical or acute abdomen. Treatment includes **gynecologic consultation, hospital admission, and intravenous antibiotics**. Large abscess may require surgical drainage.

#### Endometritis:

- Infection of uterine endometrium (if invades into myometrium = endomyometritis)
- **r/f:** retained products of placenta, STIs, IUD placement
- **chronic = asymptomatic**
- **dx:** bimanual exam with uterine tenderness, fever, elevated WBC
- **Tx:** same tx for PID – clindamycin, gentamicin, cephalosporins – continue until clinically improved and afebrile

#### Syphilis

-Incidence is increasing

- Treponema pallidum

-Part of routine STI screening panel and prenatal work-up (RPR replaced by Treponema 3 test)

-Dx: nontreponemal anticardiolipin antibodies; **Fluorescent treponemal antibody absorption (FTA-ABS)**, a treponemal test, **will remain positive after a person's first infection with syphilis leading to a false positive results and inaccuracy in diagnosing syphilis.**

Stages:

-Primary: painless ulcer (chancre), highly infectious; lasts 3-6 weeks

-Secondary: maculo-papular rash (palms/soles), condyloma latum (flat genital wart thing), systemic sx; lasts 2-6 weeks

-Latent syphilis: no sx; can last 1-60 years (this is when its often dx w/screening)

-Tertiary syphilis: affects skin/bones (gummas), CV (aortic aneurysms/aortic insufficiency), Neuro (tabes dorsalis, Argyll-Robertson pupil, blindness, paresis); lasts until treated – symptoms may be permanent - For late syphilis, penicillin **G benzathine IM once weekly for three weeks** is standard therapy. Neurosyphilis should be treated with **penicillin G intravenously for 10 to 14 days.**

**Tabes Dorsalis: demyelination of dorsal columns → lose proprioception, vibration, and fine touch (lose DTR, high step gait)**

Argyll-Robertson pupil: pupil is accommodating but not reactive (d/t dorsal midbrain lesion)

Tx: PCN G → allergy to PCN requires desensitization

#### Vaginitis

##### Bacterial vaginitis

-lack of lactobacilli = low hydrogen peroxide = high pH (>4.5)

-infection is polymicrobial (mostly Gardnerella vaginalis)

Sxs: thin discharge, fishy odor

Dx: milky vaginal discharge

pH >4.5

Amine "whiff" test → drop KOH on smear and assess for fishy smell

A common office test is the KOH "**whiff**" **wet preparation test**, in which a secretion sample is mixed with saline and 10-20% **potassium hydroxide**. The presence of a "**fishy**" **amine odor** represents a **positive test**, while the absence of this abnormal amine-like odor represents a negative result.

Clue cells on microscopy, **gram negative**

BD affirm: DNA test that looks for specific microbes

Tx: Metronidazole (PO or intravaginal) or clindamycin

-recurrence is common d/t biofilm production → may need prolonged tx (6 mos.)

-educate vulvovaginal health (no douching, soaps, etc)

AMSEL criteria: thin, white homogenous discharge, presence of clue cells in microscopic exam (stippled epithelial cell), pH >4.5, fishy odor – must have 3/4

### Candidiasis

-infection of the vaginal tract w/candida (MC is albicans)

Sxs: itching/burning, dyspareunia, thick white discharge, beefy red vaginal mucosa

Dx: wet mount → KOH and saline + microscopy

pH <4.5 (normal)

BD affirm → DNA test

Yeast cx if recurrent (look for different type of yeast)

Tx: Fluconazole (150 mg PO x1) – if its really bad, can give another dose 72 hours later

Vaginal cream (Miconazole, terconazole, clotrimazole, etc) – 7 day course typically works better

-educate vulvovaginal health

**Diabetes mellitus** is a predisposing factor for **recurrent vulvovaginal candidiasis** since hyperglycemia enhances the ability of *Candida albicans* to bind to vaginal epithelial cells. The patient above should be tested for **diabetes** due to recurrent **vulvovaginal candidiasis**, especially since she has other known risks (elevated body mass index and history of hypertension).

**Glycated hemoglobin** (also called A1C) is used to screen, diagnose and monitor prediabetes and diabetes. Patients with vaginitis may present with **vulvar pruritus** and **burning**, as well as **erythema** and **edema** of the labia majora and minora.

### Trichomonas

-caused by parasitic protozoan *Trichomonas Vaginalis*

-can affect fertility so we want it treated well

Sxs: Men are usually asxs

Females: itching, burning, post-coital bleeding, dysuria, frothy white/grey discharge

Dx: saline wet mount → must look at very quickly (parasites die fast)

pH >= 4.5

BD affirm

Strawberry cervix on spec exam (only present ~10% of time but is pathognomonic)

Tx: Metronidazole (PO only)

Partner tx

-warrants full STI screening

-Pt should be retested in 2 weeks- 3mos. To ensure successful tx ("test of cure")

### Atrophic vaginitis

Def: atrophy of vaginal and vulvar tissues d/t hypoestrogenic state (often seen in menopause)

Sxs: dryness, burning, irritation, low lubrication, pain/discomfort w/sex, urinary urgency, dysuria, recurrent UTIs


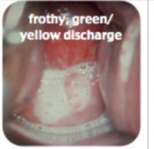

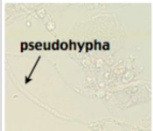

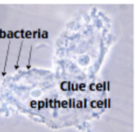
PE: fragile tissue, fissures, petechiae, labia minora resorption, loss of moisture and rugae and elasticity, prominent meatus, urethral eversion or prolapse

Dx: clinical

Tx:

1, vaginal moisturizing agents + water-based lubricants during intercourse

2. Low-dose vaginal estrogen → twice weekly via insert, ring, or cream; depending on dose may need concurrent tx with progestin - **Prescribing a vaginal ring that contains 2 mg of estradiol** to be placed once every 3 months is an appropriate initial

	Candidiasis	Trichomoniasis	Bacterial vaginosis
Discharge			
	White	Green/yellow	Gray/white
Color			
pH	< 4.5	> 5	> 4.5
Amine odor w/ KOH	Negative	Positive	Positive
Wet mount	<ul style="list-style-type: none"><li>• WBC</li><li>• Spores</li><li>• Pseudohyphae</li></ul>	<ul style="list-style-type: none"><li>• WBC</li><li>• Motile trichomonads</li></ul>	<ul style="list-style-type: none"><li>• Few WBCs</li><li>• Clue cells</li></ul>
			



intervention for patients with symptoms of **post-menopausal vaginal atrophy**. A daily intravaginal estradiol tablet is also effective. Vaginal atrophy is a very common problem in post-menopausal women that occurs due to thinning of the vaginal mucosa in the setting of decreased estrogen secretion.

----First-line therapy for symptomatic relief of vulvovaginal atrophy is with **non-hormonal vaginal moisturizers and lubricants**. If therapy does not result in symptom relief, low-dose vaginal **estrogen** (insert, ring, cream) therapy may be used if the woman has no contraindications (estrogen-dependent malignancy). Sexual activity and/or use of vaginal dilators can help maintain healthy vaginal epithelium.

-caution use in pts w/concurrent breast CA

### **Breast cancer**

Epi: >1M new cases p/yr; MC cancer in females (1 in 8 women by age 80)

RF:

Inherited → BRCA1 and BRCA2 (55-85% incr risk)

Increasing age

Previous breast CA

FHx in two close relatives

Radiation exposure

Nulliparity or first birth at age  $\geq$  35

North American/northern European

Early menarche, late menopause

Obesity

Urban residence, upper SES

Primary cancer in ovary/endometrium

Screening

Mammogram q2 years ages 50-74

-high risk pts (BRCA carriers or Fhx) may be screened earlier/more frequently and with MRI

Histopathology: **MC type is infiltrating ductal carcinoma**, then lobular carcinoma

Staging: TNM system

Dx:

FNA and **core bx** (office procedures) used to dx a palpable breast mass

**Excisional bx** allows for complete histology

Needle-localized excisional bx and stereotactic mammography used for non-palpable lesions

Tx

Lumpectomy: most common breast-conserving therapy

Modified radical mastectomy: removal of breast, fascia of pec major, and axillary lymph nodes

Post op radiation

Adjuvant chemo: depends on pt age, estrogen/progesterone receptor status, and HER-2 expression

Agents: doxorubicin, cyclophosphamide, paclitaxel, tamoxifen, aromatase inhibitors

Once the diagnosis is established, the affected woman must be tested for hormone receptor presence, including **estrogen and progesterone receptor** expression as well as **human epidermal growth factor receptor** overexpression. These receptors are both prognostic indicators and targets for specific therapies. Multidisciplinary management with surgical oncology, radiation oncology, and medical oncology has led to reduced mortality. Breast cancer management guidelines recommend that both estrogen receptor (ER) and progesterone receptor (PR) analysis should be routinely performed in all invasive breast cancers because **tumor expression can best predict which women will benefit from endocrine therapy**. Tumors that are ER-negative and PR-negative are unlikely to respond to endocrine therapy. Progesterone receptor status is heavily dependent on estrogen receptor status and does not seem to have independently predictive value when the estrogen receptor status is unknown. **Endocrine therapy**, chemotherapy, or biologic therapy are all **systemic adjuvant therapy** options which can be used to treat breast cancer in addition to surgical therapy and radiation. **Tamoxifen**, an estrogen receptor modulator, significantly reduces the risk of recurrence and death in patients with ER-positive disease.

**Breast Cancer** is the second most common female cancer in the United States. The most common type of invasive breast cancer is **infiltrating ductal carcinoma**. Patients may present with a **hard, nontender, immobile breast mass**, although with the incorporation of screening mammography, patients often present due to an abnormal mammogram. The most common location is the **upper outer quadrant** of the breast. Additionally, breast skin changes such as **erythema, thickening, or dimpling (peau d'orange)** may be present on exam. Classic mammographic findings of breast cancer include the presence of a soft tissue mass or density and clustered **microcalcifications**. The most specific mammographic feature for breast cancer is a **spiculated soft tissue mass**. Women with **abnormal imaging** findings alone should undergo **biopsy guided by mammogram** (stereotactic

biopsy), **ultrasound**, or **breast magnetic resonance imaging (MRI)**. Women with a **breast mass** should undergo a **fine needle aspiration** or **core needle biopsy**. The diagnosis of breast cancer requires the presence of malignant epithelial cells (carcinoma) showing evidence of stromal invasion. Treatment is based on the extent and stage of breast cancer. In the early stage, treatment options often include **lumpectomy** or mastectomy, followed by radiation therapy. Chemotherapy may also be recommended. Patients with **hormone receptor-positive** (e.g., estrogen receptor-positive, progesterone receptor-positive) breast cancer should receive endocrine therapy with **tamoxifen** or **aromatase inhibitors** like letrozole or **anastrozole**. Post-treatment follow-up should include frequent history and physical examinations, patient education regarding the signs and symptoms of recurrence, and mammograms. It is recommended that a baseline **bone density scan** (i.e., dual energy X-ray absorptiometry) be performed on postmenopausal women who are taking an **aromatase inhibitor**, as they are at an increased risk for **osteoporosis**.

### Cervical carcinoma, dysplasia, and HPV

#### HPV

**Cervical dysplasia**, also known as cervical intraepithelial neoplasia (CIN), is a **premalignant condition** of the **squamous** cell of the cervix. The major risk factor for the development of cervical dysplasia is infection with **human papillomavirus**. Terminology and histology classifications vary based on the degree of dysplasia present. **Cytology** results garnered from Papanicolaou smears can be classified as one of the following: atypical squamous cells of undetermined significance (ASC-US), **atypical squamous cells: cannot exclude high-grade squamous intraepithelial lesion (ASC-H)**, low-grade squamous intraepithelial lesions (LSIL), atypical glandular cells of undetermined significance (AGC) and high-grade squamous intraepithelial lesion. Follow-up testing varies depending on the age of the patient and the degree of dysplasia. For patients who have a cervical cytology reporting ASC-H, the next diagnostic study is always a **colposcopy** regardless of HPV status. **Pregnant women** with cervical cytology showing **ASC-H** should be evaluated with **colposcopy during pregnancy**. Colposcopy provides a microscopic examination of the cervix, which aids in the determination of the need for a cervical biopsy. The cervical biopsy results provide **histologic information** and are classified as either cervical intraepithelial neoplasia 1 (i.e. mild dysplasia), cervical intraepithelial neoplasia 2 (moderate dysplasia), and cervical intraepithelial neoplasia 3 (severe dysplasia). Management depends on the cervical intraepithelial neoplasia classification as well as other clinical factors and includes the treatment options observation, loop electrosurgical procedure, cold knife cervical conization, and ablative therapies.

**Atypical squamous cells of undetermined significance (ASC-US)** is the most common cervical cytologic abnormality. The risk of invasive cervical cancer in women with ASC-US is low because one to two thirds are not associated with high risk human papillomavirus (HPV). Women with **human papillomavirus (HPV)** are at higher risk for cervical precancer or cancer. **HPV strains 16 and 18** are the most commonly isolated strains in cervical cancer. **Multiple sexual partners** and **unprotected sex** increase a woman's risk of contracting HPV and therefore increase the risk of developing cervical dysplasia. The pathologic cellular changes most commonly occur at the cervical **transformation zone**, an anatomic landmark where the **squamous** epithelium of the **ectocervix** transitions to the **glandular epithelium** of the **endocervix**. Cervical dysplasia is most often discovered when women are **in their 20s** (shortly after their sexual debut). Women with cervical dysplasia are usually asymptomatic, and detection of pathologic changes usually occurs during routine cervical cytology screening. For women with **ASC-US cytology**, the preferred next step is **HPV testing**. Since one-third to two-thirds of ASC-US cases are not associated with high risk (HPV), HPV testing prior to colposcopy prevents unnecessary colposcopy. Any women with **ASC-US** and **positive** HPV testing should undergo **colposcopy**. Women with **ASC-US** and **negative** HPV testing may proceed with **routine follow-up**.

-more than 100 types of the virus – 30-40 infect the genital tract and ~20 are oncogenic (high risk HPV)

-high risk viruses are the ones tested for on HPV test

-Types 16 and 18 acct for 70% of cervical dysplasia/CA

Tx: local excision, cryotherapy, topical trichloroacetic acid, topical podophyllin, 5-fluorouracil cream

-cervarix and Gardasil vaccine

-HPV virus is necessary for cervical CA to develop (you do not get dysplasia w/out exposure to the virus)

-HPV can also cause oropharyngeal, anal, vulvar/vaginal, and penile CA

-this is why Gardasil is important for MEN and women

-Virus is sexually transmitted (skin-skin or bodily fluid contact)

-condoms don't fully protect

-should still screen "virgins" bc they may have gotten it through skin contact

-RF for HPV infection: STI RFs, immunosuppression, smoking, young age (lack acquired immunity)

-Most sexually active people get the virus at some point in their life (but then clear it)

Oncogenic properties:

-has oncogenes E6 and E7 – E6 inhibits P53 and E7 inhibits p53 and other proteins

- integrates itself into the host genome bc it is a double stranded-DNA virus
- Natural hx: most HPV infections are transient (not a “forever” virus)
  - pts are usually asxs and it resolves spontaneously (70-90% clear by 2 years)
  - pt then maintains an immunity to protect from re-infection of the same HPV type
- “Persistent Infection”: infection by same HPV type after 2 years → can lead to cervical CA through progressive neoplasia (takes 10-25 years to develop invasive CA)**
  - happens ~10% of the time
  - most women are able to eventually clear the virus and stop the progressive neoplasia
  - because its so slow growing, there are opportunities to intercept the virus/neoplasia
- Epi (F): young women are more likely to have HPV but are also more likely to clear it rapidly
  - we tend to be less aggressive with tx in young women because of complications the tx can have on childbearing and they have a higher likelihood of clearing the virus
- Epi (M): prevalence is as high/higher in men but has a lower likelihood of persistence (men clear it faster)
  - circumcision helps reduce risk of persistent infection
  - There are no screening guidelines for men (except for MSM and IC men – anal paps)

#### Screening for cervical CA

1. Pap Smear: sample of superficial epithelial cells from cervix – fixated and evaluated by cytopathologist for any abnormal cell growth/dysplasia

- want to sample from the SCJ → rotate 5x and put in liquid solution
- can do conventional cytology (rub on slide) or liquid base cytology (more accurate and then have sample for HPV if needed)

2. HPV screening: looks for 13ish of the HR HPV types (16 and 18 included)

- can be done as a co-test with pap or as a primary screen (US usually does pap then reflex HPV)
- HPV test may be + with no cell change (body likely to clear it)
- involves doing a viral screen on the fluid from the pap test
- doesn't come back with the type of virus, but just if there is a HR type present

Screening guidelines:

- <21: no screening
- 21-29: pap q3 years (reflex HPV)
- 30-65: Cotest (pap + HPV q 5 years) or pap q3 years (reflex HPV)
- >65 or s/p hysterectomy: no screening (if no hx of CIN 2+ in past 20 years)

-if Pap returns as low grade dysplasia/atypical – get reflex pap

**-if Pap returns as high grade change, pt will get a colpo (don't need reflex HPV)**

Location:

SCJ moves caudally with age → area between old SCJ and new SCJ is the “transformation zone” and this is where most cervical CA occurs

- Pap should include endocervical cells; if it doesn't, get a repeat one in a year or so to qualify it as satisfactory

#### Pap results

HPV effect: low grade squamous intraepithelial lesion (LSIL)

Mild dysplasia: CIN 1; LSIL

Moderate dysplasia: CIN2; LSIL

Severe dysplasia: CIN3, High grade squamous intraepithelial lesion

Carcinoma in situ: HSIL

Colposcopy: done for any high grade cervical changes

Magnification and illumination to help visual inspection of the cervix/vagina/anogenital area

Solutions used to highlight area of concern (usually acetic acid)

Used to help clinician bx the area of highest concern

Tools: bx forceps, endocervical curettage, acetic acid, Lugol's solution

- end up doing curettage if you can't see where to bx

What you're looking for: raised areas, sharp borders, white and coarse areas (these are most concerning); if the area is red/yellow and peeling/rolling – this is most concerning for invasive CA

- look for enlargement of the vessels/mosaicism

Tx (Preinvasive – CIN 1, 2, 3)

-use the ASCCP.org guidelines (shows how to tx and screen pts)

1. CIN 1

- Follow up with cytology and HPV testing in 12 mos. – if negative, resume routine age-based testing
- never tx at this stage bc immune system is likely to clear it

2. CIN2:

-**Young women: observe w/cytology q6 mos; colpo recommended**

-may tx with excision/ablation – many women are in childbearing years at this stage, so f/u may be the only recommended thing

3. Tx of CIN2/3: excision or ablation

-recommend tx if done w/ child bearing or if dx is CIN 3

LEEP procedure: Loop electrical excision procedure – done in office w/local anesthesia; very little blood loss – can then analyze what you excise

Ablation: doesn't allow you to further analyze the tissue

#### Invasive Cervical CA

Sxs: bleeding (often post-coital), late signs are back pain, anorexia, wt. loss

RF: hx of inadequate screening

Natural hx: spreads directly; late stage can spread to pelvic lymph nodes

-can be exophytic (protrusion) or endophytic (deep invasion)

-Staging depends on pelvic exam and is modified by further imaging – we don't stage operatively

Staging: most important prognostic factor

1. confined to cervix -- 80-95% 5 yr survival

2. beyond cervix, upper 2/3 of vagina – 64% 5 yr survival

3. lower 1/3 vagina or pelvic sidewall -- 38% 5 year survival

4: adjacent/distant organs – 14% 5 year survival

**Tx: radical hysterectomy (take everything at the root) – only early stage**

**Late stage gets chemo/radiation (brachytherapy an option where radiation is localized to uterus/vagina/cervix)**

#### Cervical CA prevention

HPV prevention: abstinence/delay sexual activity, less sexual partners, condoms

**Vaccination: yeast + capsid protein → basically give the virus w/no oncogene**

-protects against 7 HR types (including 16/18) and against 6 and 11

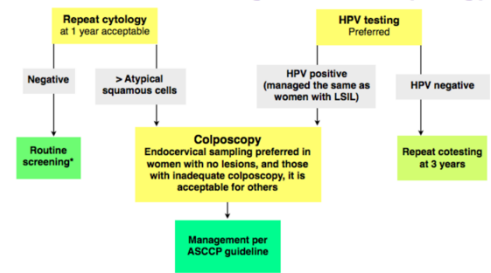
(genital warts)

-ideal age is 11-12 yo bc immune system is most robust and they

likely haven't been exposed yet

-encourage vaccination at any patient visit

#### **Management of Women with Atypical Squamous Cells of Undetermined Significance on Cytology**



#### **Molluscum Contagiosum**

- Caused by pox virus, aka water warts, can occur anywhere, often asymptomatic and resolve on own,
- Dx: clinical / confirmed with lesion biopsy for histologic or electron microscopic examination – remove with local excision and/or treat nodule base with trichloroacetic acid

#### **Endometrial cancer**

Epi: **MC GYN CA in US – best survival of the GYN cancers**

-most are dx early on bc it usually presents as heavy bleeding

-because it presents early on with sxs, there is no screening test for it (vs. cervical CA which doesn't usually present w/sxs)

-the only exception to this is women w/Lynch syndrome – they get routine biopsies .

#### RF (hyperplasia)

Increasing age

**Lynch syndrome – 80% lifetime risk of colon CA, 30-50% risk of endometrial, incr risk of breast CA**

Excess estrogen – PCOS, overweight/obese, unopposed ERT, Estrogen tumor, **Tamoxifen therapy**

Tamoxifen: SERB used to tx Breast CA → stimulates estrogen receptors in endometrium

Nulliparity (possibly if d/t anovulation)

**-Early menarche, nulliparity, late menopause, obesity and family history of endometrial cancer**

#### Protective factors

OCPs -- ~50% RR; protection lasts ~10yrs after d/c

Parity/Breast feeding

Early menopause/oophorectomy

Lean habitus

Smoking

Sxs: Suspect with any of these → recommend bx if on DDx

- Heavy, prolonged, or frequent menses
- any postmenopausal bleeding

**-Remember that PCOS is a RF**

Tamoxifen, a medication used to treat breast cancer, acts to increase estrogen in the uterus and would increase the risk of developing uterine cancer. Diagnosis is made by biopsy. **Adenocarcinoma is most common**

Dx

**Transvaginal ultrasound** is the **preferred initial diagnostic test** of choice to evaluate **painless vaginal bleeding in a postmenopausal patient** in order to rule out endometrial (uterine) carcinoma. Transvaginal ultrasonography is used to measure the endometrial **thickness**, which should be less than 5 mm in a healthy patient. **An endometrial thickness less than 3-4 mm excludes most endometrial pathology in women with postmenopausal vaginal bleeding.** A transvaginal ultrasound can identify other causes of vaginal bleeding such as **polyps and fibroids**. An alternative to transvaginal ultrasound would be an endometrial biopsy which has a high sensitivity, low cost and low risk of complications. If either test is inconclusive, further testing is warranted. **Endometrial biopsy** should be performed if **endometrial thickness** on ultrasound is greater than **four mm** in **postmenopausal women** or if a patient is **> 35 years of age** with **risk factors** for endometrial cancer (diabetes, obesity, PCOS). This patient is likely in the perimenopausal stage due to her age and history, and does not require endometrial biopsy given her ultrasound findings.

Endometrial Bx using suction curette for endometrial sampling: evaluates for malignancy and hyperplasia

- can confirm ovulation/anovulation
- simple and quick, low-risk office procedure

Hyperplasia: larger glands and little to no stroma

4 categories → will progress if not intervened

- simple hyperplasia, complex hyperplasia, simple atypical hyperplasia, complex atypical hyperplasia
- complex atypical hyperplasia is most concerning and has highest assoc w/CA (we tx it as CA → hysterectomy)
- all other categories are treated w/progesterone

Cancer progression:

1% of simple hyperplasia progresses, 3% of complex hyperplasia, 10% of simple atypical hyperplasia, 30% of complex atypical hyperplasia

Tx (hyperplasia)

The **management is determined by staging** of the endometrial cancer. **Stage 1 cancers are treated with hysterectomy and bilateral salpingo-oophorectomy with or without radiation.** Stage 2 cancers are treated with hysterectomy and bilateral salpingo-oophorectomy, lymph node excision and radiation. The depth of the tumor determines prognosis.

Progestin therapy: thins the endometrium

- MPA/norethindrone PO, DMPA, implant, LNG IUD
- should rebx (3 mos) and monitor these pts

Hysterectomy: done for complex atypical hyperplasia

- traditional is usually enough (don't need radical)
- if its full blown endometrial CA, pt gets total hysterectomy w/bilateral salpingo-oophorectomy**
- staging is based on surgery, so get the whole thing the first operation**

Endometrial CA Staging

-staging is done w/operation → want to make sure you get the whole thing

Stage 1: confined to uterus

Stage 2: invades cervix

Stage 3: serosa/adnexa, vagina/parametrium, lymph nodes

Stage 4: invades bladder/bowel or distant metastasis

**-Stage 1 just needs surgery, everything else needs chemo/XRT**

Endometrial CA tx

Surgery: hysterectomy + bilateral salpingo-oophorectomy, +/- pelvic/para-aortic lymph node sampling

Adjuvant therapy (advanced ds): chemo, radiation

### **Ovarian neoplasms**

Normal anatomy: 3 areas of the ovary → epithelium, stroma, and germ cells

- CA can arise in any of these areas (epithelial ovarian CA, sex-cord stromal tumor, germ cell tumor)
- most ovarian CA is referring to epithelial ovarian CA

**Epithelial ovarian CA = MC**

Epi: 1 in 70 lifetime risk

5 yr survival overall is 20-30% → most dx at late stage (doesn't show sx's until more advanced)

-Most deadly GYN malignancy

RF

Infertility/Nulliparity

Early menarche/late menopause

Hereditary: BRCA and Lynch

BRCA 1 or 2 gene → get TVUS q6 mos, stay on OCPs and recommend BSO when done childbearing (risk is higher w/BRCA1)

Lynch: 5-10% risk

Environmental exposure – high fat diet

Protective factors

Tubal ligation (RR of 0.3)

-**data suggests that ovarian CA starts in fallopian tubes and “drops” onto ovaries**

Pregnancy – subsequent pregnancies have added risk reduction

OCPs (RR of 0.6)

-pregnancy and OCPs reduce ovulation which is thought to be protective

**Sxs: often asxs!**

-may have pain, abdominal bloating, early satiety/anorexia, weight loss (VAGUE sxs)

-should warn people who have RF of these sxs to get checked out

-Sex cord tumors can be hormonally active → estrogen excess or virilization (T producing) – pt would show sxs of having excess hormones

Eval: hx and PE are very important!

Abdominal exam: fluid wave, subQ nodules/masses

Pelvic exam: pelvic mass, cul-de-sac nodularity

Extremities: evidence of DVT → pts are hypercoagulable

Labs: potentially if concerned for germ cell or sex cord tumor (AFP, LDH, HcG, Testosterone)

-labs and imaging and suggest or reassure → want to do these before bx

-tissue/pathology is diagnostic (can do surgery or paracentesis)

CA-125: never order as a screening test (often elevated w/out CA)

-only order if you see a cyst on US that is concerning

Screening

US + CA-125 aren't recommended for screening of general population

-studies showed no benefit to screening for ovarian CA and false+ had high rate of complications

The only screening test is a bimanual exam

Ovarian cysts v CA

-we do a lot of unnecessary sx because of ovarian cysts thinking that they are CA → we may overtest for ovarian CA but we also really don't want to miss it

ACOG recommendation: cysts <10cm in pre-menopausal women have a 0% CA risk – don't need more surveillance/intervention

-cysts >5cm in women age 40+ or complex cysts or bilateral cysts should be followed

-persistent concerning cysts or those w/suggestive sxs or if CA-125 is high should be eval'd surgically

**-MC type of ovarian cyst is follicular**

Ovarian CA spread

Spreads via direct extension to pelvic/abdominal viscera

Can spread via lymphatic dissemination (iliac nodes, pelvic nodes, para-aortic nodes)

-able to travel through peritoneal circulation – often see first deposits on liver and diaphragm

Staging

Stage 1: ovaries only → rarely found at this stage

Stage 2: pelvic extension only

Stage 3: extrapelvic extension

Stage 4: intraparenchymal liver mets, pleural effusion

Tx

Surgery/debulking – washings, omentectomy, remove all tumor to <2cm (take out as much as we can)

Chemo – platinum based, done after sx for stage 1C +

**-Carboplatin and paclitaxel intravenously** is the preferred first-line chemotherapy in women who have suboptimally cytoreduced disease after surgical cytoreduction for **epithelial ovarian cancer**. Platinum and taxane agents are used first-line. A **dose-dense schedule** is typically used with carboplatin administered every three weeks and paclitaxel administered once a week. This regimen is continued for a total of 15 weeks. Dose-dense therapy has shown either equivalent or, in some studies, improved outcomes when contrasted with conventional chemotherapy dosing. Usually, chemotherapy begins within two to four weeks after primary surgical cytoreduction. Delaying chemotherapy beyond four weeks after surgery has been shown in some studies to result in worse outcomes.

Prognosis: depends on pt age and stage

>50 yo: 5 yr survival 15%

<50 yo: 5 yr survival 40%

Stage 1: 76-93%, stage 2: 60-74%, Stage 3: 23-41%, stage 4: 11%

#### Germ Cell Tumors

Most are benign, almost all are mature teratomas (dermoids)

Dermoids aka "monster balls" – can have skin/hair/etc inside tumor + lots of sebaceous material

Malignant Germ cell tumors: immature teratoma, dysgerminoma, endodermal sinus tumor, choriocarcinoma, embryonal carcinoma

-all have different tumor markers → should order markers if you see any abnormal cyst on US

-tumor markers can be used to help dx and follow response to therapy and detect recurrence

Dermoid tumors are large and heavy → potential for causing ovarian torsion

-will often take them out and leave ovary behind

#### Sex Cord-Stromal Tumors

Arise from gonadal stroma (Females: granulosa cells, Males: Sertoli-Leydig cells)

-In most cases, the ds is confined to the ovary

-They do cause hormonal changes because they are releasing E/T → female sex cord-stromal tumors put female at greater risk for endometrial CA d/t high estrogen

#### **Vaginal/vulvar neoplasms**

-**Vulvar cancer** is the most likely diagnosis based on the patient's age, clinical presentation and history of previous HPV infection.

Vulvar cancer typically occurs in **postmenopausal women** with the average age of diagnosis being 65 years. Women typically present with a singular **pruritic lesion to the labia majora**. Rarely, vulvar cancer will present with multiple lesions or lesions located on the labia minora, clitoris or mons. Women may also present with vulvar discharge, vulvar bleeding or local lymph node enlargement, however these symptoms typically indicate more advanced disease. Risk factors include prior **HPV infection**, **cigarette smoking**, northern European ancestry, vulvar dystrophy, immunocompromised state, prior cervical cancer, and vulvar or cervical intraepithelial neoplasia. Diagnosis is confirmed with biopsy which typically shows **squamous cell carcinoma**.

-**Vulvar cancer** is the fourth most common gynecologic malignancy in the United States. The most common histologic type of vulvar cancer is **squamous cell carcinoma (SCC)**. Risk factors for squamous cell carcinoma of the vulva are **human papillomavirus** (types 16, 18, and 33), early age at first intercourse, multiple sexual partners, human immunodeficiency virus infection, and cigarette smoking. In the United States, the average age of women diagnosed with vulvar cancer is 68 years. Clinical presentation includes the presence of a **unifocal vulvar ulcer**, plaque, or mass, predominantly on the **labia majora**.

Patients commonly complain of vulvar **pruritus** and may also have **bleeding** and **pain**. Diagnosis involves a thorough history and physical, a complete pelvic examination, and a biopsy of the lesion. Colposcopy of the vulva may also aid in the identification of additional lesions not appreciated on gross examination. Definitive diagnosis is via **histologic evaluation** of the biopsy.

Treatment varies based on the histology and stage. For squamous cell carcinoma of the vulva, surgical resection with adjuvant radiation to the vulva, groin, and involved lymph nodes is recommended for most patients. Chemotherapy is recommended for patients who have evidence of metastatic disease or patients with unresectable locally advanced disease.

#### **Breast abscess**

Def: local pus collection in breast tissue

Micro: most from Staph aureus (MRSA increasing); recurrent abscesses more likely to have mixed flora/anaerobic infxn

Sxs: localized, painful infm, +/- fever, malaise

PE: tender, fluctuant mass

Dx: clinical + US

-blood cultures if severe infection

-culture of breast milk if lactating can help guide abx selection

Tx:

1. Drainage – needle aspiration or surgical drainage

2. Antibiotics – coverage for S. aureus; usually 10-14 days

-Dicloxacillin or Cephalexin; Clindamycin if B-L allergy

-Bactrim or clinda if MRSA risk

-vanco if severe infection

3. If lactating – should continue breast feeding if possible to help resolve infection

#### **Breast fibroadenoma**

Def: firm, rubbery, mobile solid mass (usually solitary)

-2<sup>nd</sup> MC benign breast ds

Sxs: painless, slow growing mass (often discovered by pt during SBE)

Dx

PE → usually solitary lesion (can find multiple); avg size is 2.5 cm; will be firm, rubbery, mobile, and nonpainful

US – can help differentiate it from cyst

Mammogram – if pt is >30

Tx:

FNA

Surgical removal – if histopathology not determined via FNA

-any rapidly growing mass should be removed and any mass found in female >30 yo

**fibroadenoma** which is the **most common breast mass in adolescents and young adults**. The peak incidence is between 25 and 40 years and decreases after age 40. Fibroadenomas are characterized by a **painless, firm, solitary, mobile, breast mass**.

Fibroadenomas are partially hormone-dependent and may enlarge in pregnancy. They often involute at menopause.

The **lesions are not fixed** to the surrounding parenchyma and slip under the palpating hand; this has led to fibroadenomas being referred to as a **breast “mouse.”** Because there is a very small risk for malignancy, these patients should undergo monitoring for changes in size of the mass, have an ultrasound, or fine needle aspiration. Treatment is supportive, but sometimes requires surgical excision.

### Fibrocystic breast ds

Def: exaggeration of normal changes in breast tissue in response to cyclic levels of ovarian hormones, noncancerous breast lumps; estrogen = primary cause

**-MC benign breast condition**

Sxs: cyclic bilateral breast pain, incr engorgement/density of breasts, excess nodularity, rapid change/fluctuation in size of cystic areas, tenderness, occasional spontaneous clear nipple discharge

-intermittent breast pain and tenderness, sx peak before menses and improve after menopause

Dx:

PE → tender, well delineated slightly mobile cystic nodules or thickened areas

Definitive through bx → variety of histopathologic changes (cysts, adenosis, fibrosis, duct ectasia)

-mammography, US, aspiration

There are many different radiographic findings for fibrocystic breast disease since there are so many variants in normal tissue, but a typical ultrasound would show **dense, prominent, fibroglandular tissue with solitary or grouped cysts but no discernable mass**.

Tx:

**Supportive → well-fitting bra**, light loose clothing, decr coffe/chocolate/tea intake, smoking cessation

OCPs or progestins help in up to 90%

**Fibrocystic breast disease** is the most likely cause of this patient's presentation. Fibrocystic breast disease commonly presents in **women age 30-50 years** as multiple, small, circular, mobile, tender cystic masses. After the cysts rupture, scar tissue forms resulting in firm breasts with rope-like structures that patients can often palpate during self breast exams. Common symptoms include breast pain, breast masses or increased breast firmness. These **symptoms are often worse leading up to menses and then resolve after menses conclude**. There is **no association with increased risk of developing breast cancer**. Cysts are able to be aspirated revealing benign straw-colored fluid. Aspiration can be both diagnostic and therapeutic. Use of a **supportive bra** is often the only treatment needed. **Caffeine restriction** has mixed data supporting its effectiveness at decreasing symptoms associated with fibrocystic breast disease. Other novel treatments include **vitamin E supplementation, low-salt diets and premenstrual hydrochlorothiazide therapy**.

**Danazol for severe symptoms → suppresses LH and FSH**

### Mastitis

Def: infection of the breast → can be lactational or non-lactational

- Lactational: most common in first six weeks postpartum
- Non-lactational includes periductal mastitis and idiopathic granulomatous mastitis
  - Periductal: infm of subareolar ducts; presents w/periareolar infm
  - Idiopathic granulomatous mastitis: rare benign infm breast ds w/unknown etio; dx via core needle bx

Micro: MC from **S. Aureus**

Sxs: firm, red, tender, swollen area of breast, fever, myalgia, chills, malaise, flu-like sxs

Dx: clinical

Tx:

1. Symptomatic – NSAIDs, cold compresses, emptying of breast



2. Antibiotics if persistent symptoms >24 hours

- Dicloxacillin or Cephalexin if no risk of MRSA; Clindamycin if BL allergy
- Bactrim or Clinda if MRSA risk → avoid Bactrim in breastfeeding moms of baby <1 mos old
- Vanco if severe infxn

#### Complications

Breast abscess

Fistula

#### Cystocele

Damage to the anterior vaginal wall pubocervical fascia can result in herniation of the bladder (cystocele) and/or urethra (urethrocele) into the vaginal lumen. Injuries to the endopelvic fascia of the rectovaginal septum in the posterior vaginal wall can result in herniation of the rectum (rectocele) into the vaginal lumen. Cystocele is the herniation of the anterior vaginal wall, or anterior compartment prolapse, often associated with descent of the bladder. Patients with a cystocele may be asymptomatic or present with urinary symptoms such as urgency, frequency and incontinence. While many women eventually choose surgical correction of the prolapse, **first-line treatment is conservative and includes both pessary and pelvic floor muscle training.**

#### Rectocele

A **rectocele** is a **posterior vaginal wall defect associated with anterior prolapse of the rectum**. It is a defect in the rectovaginal septum rather than the rectum. Patients typically complain that they feel **increased pelvic pressure** or the **sensation that something is “falling out” of the vagina**. In addition, **patients often utilize splinting of the vagina, perineum or rectum in order to achieve a bowel movement**. Splinting is the firm application of pressure on a particular anatomical location. Other symptoms include constipation, fecal incontinence or sexual dysfunction. Diagnosis can usually be made during vaginal and rectal examination by observing a bulge in the posterior vaginal area upon bearing down. Imaging is rarely needed. **Nonsurgical management** involves **medications for constipation** to minimize straining that may make the rectocele worse and **pessary placement**. Surgical repair with a **posterior colporrhaphy** has an anatomic cure rate of up to 96%. **Dyspareunia** is a potential complication that patients should be educated about when undergoing surgical repair of a rectocele.

- **Rectocele is the anterior protrusion of the rectum into the posterior vaginal wall. Observation with yearly examinations is appropriate management for asymptomatic women** or for women with mild symptoms and a mild prolapse because the majority of these women may never experience prolapse progression. The Women’s Health Initiative estrogen and progestin trial found that the majority of women with Baden Walker grade I pelvic organ prolapse (distal descent of prolapse halfway to the hymen with Valsalva maneuver) would experience prolapse regression, while the majority of women with more advanced prolapse would experience prolapse progression. **When indicated, treatment for symptomatic women may consist of directly treating bowel symptoms (constipation or defecatory dysfunction), supportive or space-occupying pessary, pelvic floor muscle training, or surgery with traditional posterior colporrhaphy or site-specific defect repair.**

#### Uterine Prolapse

##### Risk Factors

#1 is pregnancy/childbirth – developing pregnancy puts weight on pelvic floor and birth can cause trauma

#2 is advancing age: d/t **lower amounts of estrogen which causes weakening/deterioration of ligaments**

-**lactating women are also low in estrogen → can be a problem after birth**

Others: obesity, hysterectomy, family history, genetics, connective tissue d/o, chronic straining / constipation

Hx: pelvic pressure, vaginal bulge, urinary dysfunction, defecatory issues, sexual dysfunction

Anatomy: Can have prolapse of any reproductive organ → uterus, vagina, or both

##### Epi

11% lifetime risk by age 80

30% need reoperation for recurrence

##### W/u

PE: isolate each compartment to determine what is prolapsing

-Quantify prolapse with the POPQ (Pelvic Organ Prolapse Quantification / Baden-Walker Halfway Scoring System) , split-speculum exam

-  
Management: kegals, pessaries, PT with biofeedback

**Pessary: plastic device fitted to patient to hold organs in = first line**

-have to accommodate for intercourse

-good to try before surgery – may be used as a bridge or if patient doesn’t want/qualify for sx

Surgery:

Anterior or posterior colporrhaphy: "tighten up" fascia to create better support

Complications: injury to other organs, incontinence/voiding dysfx, bleeding, recurrence, constipation/defecation dysfx, pain, rectovaginal fistula

Sacrospinous ligament suspension: open posterior vagina and Tac it to the sacrospinous ligament

Complication: injury to pudendal nerve/rectum

**Sacrocolpopexy: the gold standard; attach mesh to anterior and posterior vagina and tether it to ligament on sacrum**

LeFort Colpocleisis: basically, sew vaginal wall closed (recreate perineum and shrink vaginal canal)

option for someone who can't tolerate an operation / doesn't want penetration

Pt can never have penetration again

### Contraceptive methods

-About 1/2 of pregnancies are unintended in U.S.

#### Barrier methods

-items that physically prevent sperm from reaching egg

##### Condoms

-Condoms are the only method that work to prevent pregnancy and STIs

Goals of use: correct use, consistent use, availability

Key steps for efficacy: "each time, every time, start to finish", reservoir needs to be at tip, withdraw the penis while erect and hold at base during withdrawal

-Perfect use: ~98% effective

##### Diaphragms

-low cost and low SE profile but not super reliable

Use: insert <6 hours before sex w/spermicide and remove >6 hours (but <24) after sex

-no STI protection

-Can't be prescribed anymore (out of production)

-the Caya cup is a "one size fits all" diaphragm that can be prescribed and the pt buys spermicide

#### Periodic abstinence

AKA "Natural Family Planning"

-accounts for the sxs of fertile phase, viability of sperm in GYN tract (2-7 days), and lifespan of ovum (1-3 days)

Methods: Rhythm/calendar, Cervical Mucus, Symptothermal

-typically requires about 17 days of abstinence/protection per cycle (→ high failure rates)

-Can be very effective if pts are adherent

#### Hormonal Contraception

##### Hormones

Estrogens: estradiol + Ethinyl (prevents inactivation when given orally)

Dosing: 10/20/30/35/50 mcg

-doses have slowly been decreasing to lessen SE (10mcg is newest dose)

-20-35mcg are the mainstay doses → may increase if pt is having breakthrough bleeding

##### **Progestins: derived from Testosterone**

-19 nortestosterones are the "traditional" progestones. Newer ones have less androgenic SE

-Yaz and Yasmin have an anti-androgen effect (hormone is Gesperidone) → can be good for pts w/acne or PCOS

MOA: provide negative feedback to hypothalamus/anterior pituitary to decrease FSH and LH which inhibits follicle development/ovulation

-Progesterone also thickens cervical mucous, thins endometrium, and decreases tubal peristalsis

-Estrogen has more of an effect on FSH and Progesterone has more of an effect LH

-Progesterone can be used alone because it works to prevent ovulation

-Estrogen helps prevent cyst growth → can be used to protect a remaining ovary in a patient who only has one

-Bc progesterone decreases tubal peristalsis, if a pt does become pregnant on bc, it has higher chance of being ectopic

##### CHC

Combined OCPs: has 3 weeks of hormonal pills and 4-7 days of placebo pills

-can use continuously (skip the placebo week)

-types of pills vary in EE dose/type of progesterone

Transdermal patch: wear weekly for 3 weeks, then 1 week off

-can use continuously

-2x the risk of VTE (unclear why)

Transvaginal Ring: insert vaginally, leave in 3 weeks, 1 week off

-can be used continuously

#### **Progesterone-only methods**

**-these will always be “safer” than CHCs**

Injectable contraception: Depot-medoxyprogesterone acetate (DMPA)/**Depo-Provera**

-IM injection q3mos.

-Highest assoc w/weight gain and decrease in BMD

-Can disrupt ovulation/menses after cessation for up to one year

-BMD loss is a black box warning buuutt...not associated with an increased risk of fracture and once you stop DMPA, the density loss is reversible (ACOG says its okay to continue for >2 years) – studies show osteopenia based on pts having lower density than others in their age group

**Implant (Nexplanon): lasts 4 years**

**-statistically the most effective BC method (> than sterilization)**

-may cause light, irregular bleeding for first 3-12 mos.

Levonorgestrel IUD: thickens cervical mucus, partially inhibit ovulation, and thins endometrium

-approved for 3-7 years (depending on type)

r/f for expulsion: prior expulsion, hx of menorrhagia or severe dysmenorrhea, postpartum / post-second trimester abortion, <25 yo (3-10% chance expulsion)

#### **Non-Hormonal Methods**

Copper IUD: release free copper ions which creates inflammatory response and makes the intrauterine environment very spermicidal

-approved for 12 years

-copper does degrade over time

#### **Sterilization**

Male: vasectomy; Female: tubal ligation, Essure, etc.

-Male sterilization has advantage of no general anesthesia and lower rates of failure

Essure: polyester coil placed in proximal tube hysteroscopically

-stimulates rxn causing fibrosis and occlusion (original intention was to keep tube patent)

-repeat HSG to look for occlusion—has ~99% effective rate

-has fallen out of favor d/t massive law suits for chronic pain

Salpingectomy: removal of fallopian tubes

-has increased in popularity

-thought to be protective against ovarian CA

#### **Emergency Contraception**

Plan B (levonorgestrel)

Progesterone surge blocks LH surge to inhibit ovulation → doesn't do anything if ovulation has already occurred

-can take w/in 72 hours of UPI

-reduces risk of pregnancy by ~75%

-not harmful to embryo if conception has occurred

Ella (Ulipristal Acetate)

Anti-progesterone agent → inhibits ovulation and makes the endometrium inhospitable to implantation

-take w/in 5 days of UPI

-More effective than plan B (98%)

-Theoretical risk to pregnancy if already implanted (but would have to take >1 pill to cause harm)

Copper IUD

Can be used as EC if placed within 5 days of UPI → most effective form of EC

#### **Efficacy and Safety**

-LARC methods shown to be 20x more effective than OCPs/patch/ring

-Women tend to continue LARC methods longer than non-LARC methods

Safety: CDC has a Medical eligibility Criteria for contraceptive use chart that outlines medical conditions and which BC methods can and can't be used

**\*Absolute Contraindications to EE use\***

-Thromboembolic d/o

-known/suspected breast CA

-Smokers >35 y.o.

-Uncontrolled HTN

-Migraine w/aura

-SLE w/antiphospholipid ab's (also shouldn't get Progesterone – Copper IUD is best choice)

### Endometriosis

- may be related to genetics / altered immune system
- presence of endometrial tissue outside endometrial cavity – MC found in ovary and pelvic peritoneum; sometimes transported to lymphatic system (retrograde menstruation)
- severity of symptoms does not equate to amount of endometriosis

Theories of ds:

- Retrograde menstruation (most likely): endometrium floats back out of fallopian tubes onto ovary/into cul-de-sac
- Hematogenous/lymphatogenous spread
- Coelomic metaplasia

- ds is estrogen dependent (decreases in low estrogen states)
- can happen as a result of obstructive anomaly (imperf. Hymen, transverse septum/longitudinal septum, cervical agenesis)
  - once the obstruction is removed, the endo usually resolves

**RF:** early menarche, short cycles, heavy/prolonged cycles, early menarche, prolonged menses, Mullerian anomalies, family history, autoimmune hx

Protective: multiparity, longer lactation, regular exercise

**Sxs:** cyclic pelvic pain peaking 1-2 days before onset of menses

- dysmenorrhea, chronic pelvic pain, dyspareunia, adnexal mass, infertility (dysmenorrhea, dyspareunia, dyschezia)**
- many women are asxs
- amount of endo doesn't correlate to pain (depth of implantation correlates better)
- decrease in pain w/surgical excision/ablation/cauterization

**P/e:** could be subtle / nonexistent

**Dx:** clinical + laparoscopy for direct visualization – surgical confirmation necessary for dx

- imaging can only see it w/in the ovary
- surgical dx: 2 of – endometrial epithelium, endometrial glands, endometrial stroma, hemosiderin-laden macrophages (seen on patho report)
- surgical staging

Complications: chronic pain, infertility, chronic infm d/o, chronic infm state

**Tx:** Medical or surgical

- Medical: NSAIDs, progestins, OCPs, Danazol, GnRH agonist (put in state of pseudopregnancy)
  - can only give Danazol for 6 mos bc of bone loss (can do longer if you do add-back therapy)

**Increasing consumption of long-chain omega-3 fatty acids** can **decrease** a woman's **risk** of endometriosis. Having multiple childbirths, extended periods of lactation, and use of oral contraception may also decrease risk. Some protective factors include race, late menarche, and early menopause.

### PCOS

The patient has manifestations of **hyperandrogenism** and menstrual abnormalities that are suspicious for **polycystic ovary syndrome (PCOS)**. PCOS is characterized by the **triad of oligo-ovulation or anovulation**, clinical or biochemical **hyperandrogenism**, and **ovarian cysts** (greater than or equal to 12 follicles). There is gonadotrophic dysregulation with **increased luteinizing hormone (LH)** pulsatility and abnormally high ratios of circulating LH to follicle-stimulating hormone (FSH). Serum androgens may be elevated from increased ovarian production and findings of androgen excess are common. Obesity is also common although a subset of patients may present with "lean" PCOS phenotype; therefore absence of excess weight should not preclude PCOS diagnosis. Management is composed of lifestyle changes for weight loss in overweight and obese patients, hormonal contraceptives for managing hyperandrogenism and menstrual dysfunction, and metformin to reduce insulin resistance and levels of androgens. Antiandrogenic medications may also be added to other therapies or used alone for the treatment of hirsutism.

### Adenomyosis

- Extension of endometrial tissue into uterine myometrium
- Cause: ??? – high levels estrogen stimulate hyperplasia of basalis layer of endometrium or metaplastic transformation
- Causes uterus to become diffusely enlarged and globular due to hypertrophy and hyperplasia – most extensive in fundus and posterior uterine wall
- r/f: endometriosis, uterine fibroids
- hx: asymptomatic, secondary dysmenorrhea, menorrhagia, or both, increasingly heavy or prolonged menstrual bleeding

- p/e: diffusely enlarged globular uterus
- dx: MRI is most accurate imaging tool but US used first – indistinct endometrial-myometrial junction or glandular tissue within myometrium
- tx: minimal symptoms: NSAIDs, OCPs, progestins, endometrial ablation
  - mirena = most effective temporary means of managing
  - hysterectomy = only definitive treatment

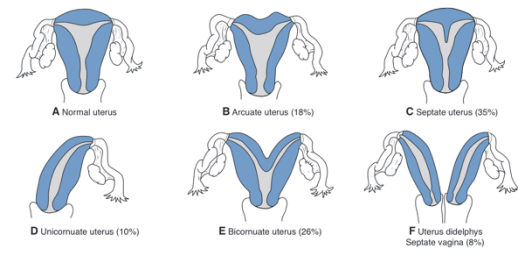
## Infertility

### Leiomyoma

Uterine fibroids (benign monoclonal tumors, shiny/white masses) – MC indication for surgery, but most require no treatment  
**-grow in response to estrogen** – respond to both estrogen and progesterone; need good blood supply → can get larger during pregnancy (stop growing during menopause)  
 -benign monoclonal tumors

#### Locations:

- Submucosal: beneath the endometrium – these are most likely to cause **heavy bleeding**
- Intramural: within myometrium; **this is MC**
- Subserosal: beneath the serosa – these are most space occupying and most likely to cause bladder P or tenesmus
- Parasitic: pedunculated fibroid that attaches to pelvic viscera or omentum and develops own blood supply



**Epi:** common (present in ~25% of women), higher incidence in AA women (80% lifetime risk vs 70%)

- if you have one, you're likely to have another
- MC associations: AA heritage, nonsmoking, early menarche, nulliparity, perimenopause, increased alcohol use, HTN
- low dose OCPs = protective against

r/f: advancing age, hyperestrogen state, African-americans, hypertension, nulliparity

**Sxs: most are asxs** – sxs depend on location and size (may be large enough to cause dysfunctional uterine bleeding; only problematic with weird location causing irregular bleeding or reproductive difficulties / cause mass effect of other organs)

-menorrhagia, pelvic pain, palpable uterus above pubic symphysis

-others have abnormal uterine bleeding / chronic iron-deficiency anemia / pressure related symptoms (urinary frequency / retention)

- most can conceive without difficulty

**Dx:** nontender irregularly enlarged uterus, pelvic US = MC means, MRI also helpful

**Tx:** only if sxs and depends on location

- nonhormonal options: NSAIDs and **anti-fibrinolytics (tranexemic acid)**
- hormonal: combined OCPs, progestins, mifepristone, androgenic steroids, gonadotropin-releasing hormone agonist

#### → work to decrease circulating estrogen levels

Submucosal: tx for AUB, hysteroscopic resection

-hysteroscopy: “shave down” the fibroid

Large/Intramural/subserosal: myomectomy, Hysterectomy = definitive treatment, artery/fibroid embolization (expensive and not widely available)

- may tx with a **GnRH agonist (leuprolide) to shrink before surgery** (given in a non-pulsatile fashion which shuts down FSH release and decreases the HPO axis) → basically puts pt through menopause and has a lot of SE
- Myomectomy puts pt at higher risk for uterine rupture bc of the damage to the integrity of the myometrium
- Vaginal hysterectomy is preferred (may not be able to bc of size of fibroid – would do laparoscopically)

**-hormonal, myomectomy, embolization, hysterectomy**

**TABLE 14-2 Clinical Symptoms of Uterine Leiomyomas (Mnemonic: FIBROIDS)**

F	Frequency and retention of urine, hydronephrosis
I	Iron deficiency anemia
B	Bleeding abnormalities (menorrhagia, metrorrhagia, menometrorrhagia, postcoital spotting), bloating
R	Reproductive difficulties (dysfunctional labor, premature labor/delivery, fetal malpresentation, increased need for cesarean delivery)
O	Obstipation and rectal pressure
I	Infertility (failed implantation, spontaneous abortion)
D	Dysmenorrhea, dyspareunia
S	Symptomless (most common)

## Abnormal Uterine Bleeding

**Abnormal uterine bleeding** is a **diagnosis of exclusion** once all other causes of abnormal uterine bleeding are excluded. It is most common at beginning stages and end stages of reproductive age being **most common in perimenopausal women**. In adolescents, abnormal uterine bleeding is frequently caused by anovulatory cycles. In these patients, treatment is typically achieved with oral contraceptives. In premenopausal women, endometrial cancer should be considered before a diagnosis of

AUB is made. If hormones cannot control the symptoms, surgical intervention should be considered. A **dilation and curettage (D&C)** procedure may stop the bleeding but could offer only temporary relief. Hormone-releasing intrauterine devices (IUD) can be used or an endometrial ablation is also a treatment option. Hysterectomy is often used as a last resort if all other treatment options fail and fertility is not a concern.

**Acute abnormal uterine bleeding** refers to an episode of heavy bleeding that may require immediate intervention to prevent further blood loss. Initial management should include the following: establishing hemodynamic stability, excluding pregnancy, identifying the source of bleeding, and providing appropriate therapeutic intervention. In a patient with signs of hypovolemia and hemodynamic instability, fluid resuscitation and blood transfusion along with intrauterine tamponade performed to **stabilize** the patient. **Uterine curettage** is the **treatment of choice** in hemodynamically unstable patients with heavy uterine bleeding. If bleeding persists, the next step in treatment would be the use of **intravenous conjugated equine estrogen**. Estrogen in high doses is used to reduce sudden, heavy uterine bleeding by stabilizing the endometrial lining and stopping the bleeding within one to two hours.

- **Oral contraceptives** are used as first-line conservative treatment to suppress endometrial overdevelopment and promote predictable cycles. **Nonsteroidal anti-inflammatory drugs (NSAIDs)** used to decrease blood loss. In patients with coagulation disorders, **desmopressin** can be used to control bleeding. Uterine curettage, endometrial ablation and hysterectomy can be used in patients who have failed hormonal therapy or have symptomatic anemia.

#### Endometrial Polyp

- Benign overgrowth of endometrial glands, **most common in women 40-50 yo taking tamoxifen for breast cancer prevention**
- **Hx:** abnormal vaginal bleeding / bleeding between periods
- **Dx:** US, sonohysterogram, hysteroscopy
- **Tx:** determine malignant or non-malignant – recommended to remove any

#### Ovarian Cyst

- Divided into functional cyst and neoplastic growths
- Functional: normal physiologic functioning of ovaries → follicular / corpus luteum
  - **Follicular = MC** – arise from failure of follicle to rupture during follicular maturation phase (3-8cm); most resolve spont.
  - Corpus luteum = luteal phase of menstrual cycle – can delay menstruation
- Hx: follicular tend to be asymptomatic, larger = pelvic pain; corpus luteum = local pelvic pain, amenorrhea, or delayed menses
- p/e: ruptured = pain, torsion = waxing and waning pain, n/v
  - **Abdominal and pelvic ultrasound** is the first imaging study of choice for suspected ovarian torsion because it is less expensive than and has similar diagnostic performance as computed tomography (CT) and magnetic resonance imaging (MRI). Definitive diagnosis is direct visualization of a torsed ovary during surgery, and prompt operative evaluation is the mainstay of treatment to preserve ovarian function.
- dx: pelvic US
- tx: palpable ovary or adnexal mass in premenarchal or postmenopausal patient is suggestive of ovarian neoplasm; **cysts >7cm that persist should be monitored with MRI; <7cm = observe with fu US + OCPs to suppress ovulation and cystectomy**

#### Sexual assault and intimate partner violence

##### Sexual Assault

Components of care:

- treat life-threatening injuries
- get informed consent before examination
- get detailed hx/recollection of account from the patient → document the details
- inspect for trauma (head-to-toe exam)
- examine external genitalia → document/photograph
- Speculum exam – test for semen/STI screening
- Baseline bloodwork for HIV, syphilis, HepB/HepC
- Provide abx and ARV prophylaxis
- Provide EC
- If rectal penetration – do a rectal exam

-Counsel, arrange f/u (medical and psychosocial)  
-have a witness throughout the exam and specimen collection. Witness must sign chain of custody statement if transporting/managing specimens → always best to hand it off personally

#### Epi

1 in 6 women, 1 in 33 men are victims of sexual assault

80% are <30

~75% know their assailant

AA and Native Americans are at higher risk

#### RF

Alcohol/drug use

Impulsive/anti-social tendencies

Personal hx of abuse

Hostility towards women

Poverty

Tolerance of violence in the community

Societal norms around male sexual dominance

#### Prognosis

Puts patient at risk for: alcohol/drug abuse, eating disorders, depression, PTSD, anxiety, sexual dysfx, chronic pain, GI problems, H/a

#### **Intimate Partner violence**

##### Warning signs

Partner overly attentive/critical

Psych changes, anxiety, sleep disturbances

Suspicious bruises – arms

Multiparous/sexually active but difficulty w/pelvic exam

##### Screening tools

HITS – how often does your partner hit, insult, threaten, or scream at you?

STAT – have you ever been slapped, threatened, or thrown?

RADAR – remember to ask, document, assess, and refer

-everyone should be screened!

Most at risk:

Age 16-24

Low SES

Low education

Hx of violence

Immigrant

Transgender

Hx of alcohol/drug abuse

#### What to do

Kids: called the police

Adults: offer support but patient needs to call the police

#### **Urinary incontinence**

Definition: involuntary leakage of urine

-Not technically a disease; may be a symptom of an underlying ds (ex: MS)

-Most cases are multifactorial and require a very thorough hx to get to underlying problem

Epi: common but likely underreported

##### Normal Micturition

Bladder filling stage → sensation to void (detrusor relaxes, urethra and pelvic floor contract)

→ Desire to void (detrusor contracts, urethra relaxes, pelvic floor relaxes, you pee)

Staying continent: requires healthy fascia, attachments, and strength/connection/coordination of levator ani muscles

#### Dx

Screening: ask questions about leaking (situation? Amount?)

Types: Stress, Urge, Mixed, or Other

#### **Urge Incontinence**

-incontinence secondary to uninhibited detrusor contractions (leak w/out provocation)

Aka “Overactive Bladder”

#### **Stress Incontinence**

-leakage d/t increased intrabdominal pressure + laxity of the internal urethral sphincter

### **Mixed Incontinence**

-a mix between stress and urge IC

### **“Other” Incontinence**

Overflow IC: can be from underactive detrusor muscle or outlet obstruction → urine retention to a point where it leaks out

-this is what happens in MS patients

-pt feels like they have to pee but can't and will have dribbling

-do NOT give muscle relaxants to these patients (will make it worse)

Fistula: can connect any part of the urinary tract with vagina or rectum (will have leakage)

### **Urethral Diverticula: classic triad of sx: dysuria, dribbling, and discharge**

-can collect urine that will occasionally come out (dribbling), get infected (Discharge and dysuria)

Insensible urine loss: leakage w/out trigger or sensation

Functional: inadequate toileting/ physical or mental impairments

Psychogenic: secondary gain

### Work-up

Need good hx and PE to clarify the type of IC

Key questions: onset and course, leakage frequency/volume/timing/assoc sx

-abrupt onset w/no UTI = specialty referral

PE: cough stress test, look for prolapse and atrophy, neuro exam (MS, S2-4 sensation, sacral reflex, Motor, strength/tone of levator ani and anal sphincter)

-abnormal neuro exam = referral

UA and cx to rule out UTI

Infection can cause urge IC or worsen stress IC

Hematuria can be sign of intra-vesical patho → if no UTI = refer for CT

Have pt keep bladder diary (help classify the type)

Refer to Urogynecology as needed

-can perform cystometrogram (measure strength of detrusor contraction) and Urodynamic studies (compare abdominal P to bladder P)

### Treatment options

Stress UIC: PT, OTC devices, Pessary, Midurethral sling

-need more support to bladder neck

Midurethral sling: mesh under urethra for added support (can be done retropubic or transobturator)

Sx complications: bladder perf or urethral perf

Urge IC: behavior modification, meds, posterior tibial nerve stimulation, Botox

Behavior: avoid caffeine/alcohol (irritate) – First line

Antimuscarinics: act on bladder to inhibit involuntary detrusor contractions

SE: dry mouth/eyes, constipation, retention, GERD, blurry vision, cognitive SE

CI: narrow angle glaucoma, gastroparesis, urine retention

Mirabegron (beta-agonist): acts on B receptor in detrusor to relax it and increase capacity

SE: HTN

CI: uncontrolled HTN, ESRD, liver ds

Posterior Tibial nerve stimulation: stimulate afferent nerves which blocks abn signals and prevents hyperactivity

Multiple tx over 12 weeks

Intravesical Botox: blocks release of ACH to cause flaccid paralysis; repeated q6-9 mos

Could lead to urinary retention and need for self-catheterization

Interstim: “Pacemaker” for the bladder – targets S2-S4 and calms the nerve down/synchronizes input

Doesn't work for everybody; much more effective for fecal incontinence

### **UTIs**

- MC infection of lower genitourinary tract treated by clinicians
- **Dx:** hematuria, leukocytes, leukocyte esterase, nitrate in absence of vaginal infection → UTI (get a UA)
  - E.coli, staph saprophyticus, proteus mirabilis, klebsiella pneumoniae, enterococcus
- **Tx:** most can be treated with oral abx (Bactrim, FQ, nitrofurantoin 3-7 days), ampicillin / cephalexin also used