Obstetrics Final

🔊 Podcast Style Review (Experimental Feature)

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

Labor & Delivery (Normal & Abnormal)

Fetal Monitoring During Labor

- **Cardiotocography (CTG):**
    - **Baseline:** Normal 110-160 bpm.
    - **Variability:** Normal short-term variation indicates fetal well-being. Sinusoidal pattern is ominous (associated with severe fetal anemia, hypoxia, e.g., vasa previa).
    - **Accelerations:** Presence is reassuring.
    - **Decelerations:**

        - *Early:* Mirror contractions, gradual onset/return. Due to head compression (vagal stimulation). Usually benign. Classic cause is head compression.
        - *Variable:* Abrupt onset/return, variable shape and timing relative to contractions. Most common type in labor. Due to cord compression.
        - *Late:* Gradual onset/return, nadir after peak of contraction. Indicate uteroplacental insufficiency, fetal hypoxia. Repetitive late decelerations most commonly indicate fetal hypoxia.
        - *Prolonged:* Last >2 min, <10 min. Concerning for fetal hypoxia.
    - **Non-Stress Test (NST):** Assesses fetal well-being antenatally. Reactive (normal) = 2+ accelerations (15bpm for 15s) in 20 min. Non-reactive requires further assessment (e.g., BPP). Commonest cause of non-reactivity is fetal sleep state.
    - **Contraction Stress Test (CST):** Assesses fetal response to uterine contractions (spontaneous or induced). Positive CST (late decelerations with >=50% contractions) indicates uteroplacental insufficiency, high risk. Negative CST (no late/significant variable decels) is reassuring.
    - **Computerized CTG:** Normal parameters include baseline 116-160, presence of accelerations, absence of significant decelerations, normal short-term variation (e.g., >2-3 ms). At least one fetal movement or three accelerations may be criteria for normality in some systems. Small decelerations (<20 beats) might be normal. Short term variation should not be below a certain threshold (e.g., <2 ms).
- **Fetal Scalp Blood Sampling (pH):**
    - Used when CTG is suspicious/pathological.
    - Normal pH: >7.25 (Reassuring).
    - Borderline pH: 7.20-7.25 (Repeat within 30 mins).
    - Acidotic pH: <7.20 (Requires urgent delivery). pH 7.15 indicates fetal distress. pH 7.10 is severe acidosis.
    - Contraindicated in: Maternal HIV, Hepatitis B/C, fetal bleeding disorders, prematurity <34 weeks.
- **Fetal Biophysical Profile (BPP):** Antenatal assessment. Components include NST, fetal breathing movements, gross body movements, fetal tone, amniotic fluid volume. Each scored 0 or 2. Score 8-10 is normal. Does NOT include fetal rapid eye movements. Umbilical artery Doppler is not part of the standard BPP score itself but may be done alongside.

Normal Labor & Delivery

- **Definition:** Spontaneous onset at term (37-42 weeks), vertex presentation, culminating in vaginal delivery of a healthy baby and placenta without complications. Delivery may be assisted by episiotomy. Labor started spontaneously or induced does not fit the strict definition of 'normal' spontaneous labor. Delivery of singleton baby, longitudinal lie, term outcome are parts of definition.
- **Stages of Labor:**
    - First stage: Onset of regular contractions to full cervical dilatation (10 cm). Latent phase (slow dilatation up to ~4-6 cm), Active phase (faster dilatation). Duration longer in nulliparous women.
    - Second stage: Full dilatation to delivery of baby. Duration longer in nulliparous, with epidural. Increased duration is indication for assisted delivery.
    - Third stage: Delivery of baby to delivery of placenta. Active management (oxytocics) shortens duration, reduces PPH risk. Pulling cord before placental separation is contraindicated. Wait for signs of separation (gush of blood, cord lengthening, uterus rises). Check placenta for completeness.
- **Mechanism of Labor (Vertex Presentation):**
    - Engagement: Passage of widest diameter (biparietal) through pelvic inlet. Head is engaged when biparietal diameter is at or below ischial spines / pelvic inlet. Defined by descent below pelvic inlet.
    - Descent: Continuous throughout labor.
    - Flexion: Occurs as head meets pelvic floor resistance. Allows smaller suboccipitobregmatic diameter to present.
    - Internal Rotation: Occiput rotates (usually anteriorly) to lie under symphysis pubis.
    - Extension: Head extends as it passes under symphysis pubis. Fetal head delivered by extension.
    - Restitution: Head rotates back briefly to align with shoulders.
    - External Rotation: Shoulders rotate into AP diameter of outlet, head rotates further.

- Delivery of shoulders and body.

- **Fetal Position/Presentation/Lie:**
  - Lie: Relationship of fetal long axis to maternal long axis (longitudinal, transverse, oblique).
  - Presentation: Fetal part entering pelvis first (cephalic, breech, shoulder).
  - Attitude: Relationship of fetal parts to each other (usually flexion). Abnormal attitude = deflexion (e.g., face, brow). Face presentation is an abnormal attitude.
  - Position: Relationship of presenting part's denominator to maternal pelvis (e.g., LOA, ROP). Denominator: Occiput (vertex), Mentum (face), Sacrum (breech), Acromion/Scapula (shoulder). Fetal part mostly related to symphysis pubis is not the definition of position. The relation of fetal parts to one another defines Attitude. Relationship between presenting part and maternal pelvis defines Position.
  - Vertex presentation denominator = Occiput. Face presentation denominator = Mentum. Brow presentation denominator = Frontum (though often described by occipitomental diameter). Shoulder presentation denominator = Acromion/Scapula. Breech presentation denominator = Sacrum.
  - Most favorable position for vaginal delivery: Well-flexed Occipitoanterior (OA).
- **Uterine Contractions:** Increase in frequency, duration, intensity during labor. Optimal frequency in active labor ~3-5 in 10 min. Pressure greatest during second stage.

Abnormal Labor & Delivery

- **Malpresentations:**
  - *Breech Presentation:* Incidence ~3-4% at term. Risk factors: Prematurity (major factor), multiple gestation, polyhydramnios, uterine anomalies (septa, bicornuate), placenta previa, fetal anomalies (hydrocephalus, anencephaly), previous breech. Conjoined twins, contracted pelvis, septate uterus are predisposers. Fibroids can predispose. Short umbilical cord is NOT associated.
    - Types: Frank (hips flexed, knees extended), Complete (hips & knees flexed - 'squat position'), Footling (one/both feet below buttocks).
    - Management: External Cephalic Version (ECV) offered ~36-37 weeks if no contraindications (e.g., ruptured membranes, APH, fetal compromise). ECV should not be done before ~36 weeks. Requires non-engaged breech, intact membranes, adequate fluid. May lead to fetal bradycardia, placental abruption. General anesthesia not required.
    - Mode of Delivery: Planned Cesarean Section often recommended, especially for non-frank breech or estimated large baby. Vaginal breech delivery requires experienced operator, specific criteria (adequate pelvis, frank/complete breech, average EFW, no hyperextension of head). Assisted breech delivery steps include allowing spontaneous delivery to umbilicus, assisting arms (Loveset's maneuver), delivering head (Mauriceau-Smellie-Veit maneuver or forceps). Rotation to sacro-anterior is key. Gentle traction on legs may be needed but excessive traction avoided.
    - Complications: Cord prolapse (esp. footling), head entrapment, birth trauma, hypoxia.
  - *Face Presentation:* Head hyperextended. Denominator: Mentum. Presenting diameter: Submentobregmatic (9.5cm). Vaginal delivery possible if mentum anterior (MA). Persistent mentum posterior (MP) requires C-section. Lowermost part felt on exam: fetal jaw/mouth.
  - *Brow Presentation:* Head partially extended. Denominator: Frontum. Presenting diameter: Occipitomental (or mentovertical if fully extended brow) - largest diameter (~13.5cm). Vaginal delivery usually impossible unless converts to vertex/face. Requires C-section. Presenting diameter is Occipitomental. Lowermost part felt on exam: frontal bones/anterior fontanelle/supraorbital ridges.
  - *Transverse/Oblique Lie:* Fetal long axis perpendicular/oblique to maternal axis. Common causes: Grand multiparity, prematurity, polyhydramnios, placenta previa, uterine anomalies, pelvic tumor. Abruptio is NOT a typical cause. Requires C-section if persists in labor. Risk of cord prolapse high with ROM. Management of dead fetus in active labor with transverse lie: Cesarean section (internal version/decapitation rarely done now).
  - *Occipitoposterior Position (OP):* Common malposition. Can lead to longer labor, back pain, increased risk of assisted delivery/C-section. May rotate spontaneously or require manual/instrumental rotation. Increases average duration of labour.
- **Shoulder Dystocia:** Impaction of anterior shoulder behind symphysis pubis after delivery of head. Obstetric emergency.

- Risk factors: Fetal macrosomia (esp. diabetic mothers), maternal diabetes, previous shoulder dystocia, prolonged second stage, assisted delivery.
- Management Maneuvers: Call for help (neonatologist, anaesthetist, extra staff), McRoberts maneuver (hyperflex maternal legs - flattens lumbar spine, rotates symphysis), suprapubic pressure, delivery of posterior arm, Woods screw/Rubin maneuvers (internal rotation), Gaskin maneuver (all-fours), Zavanelli (cephalic replacement → C/S), symphysiotomy, intentional clavicle fracture. Vigorous fundal pressure is CONTRAINDICATED. Attendance of expert neonatologist is required.
- Complications: Fetal - Brachial plexus injury (Erb's palsy most common C5-C6), clavicle/humerus fracture, hypoxia, death. Maternal - PPH, perineal trauma (3rd/4th degree tears). Erb's palsy involves C5, C6 (sometimes C7). Resolution often occurs in 6-12 months, but can be permanent. Fractured clavicles often heal well. Seizures related to hypoxia can occur.

- **Obstructed Labor:** Failure to progress despite adequate contractions due to mechanical obstruction. Causes: Cephalopelvic disproportion (CPD), malpresentation/malposition, fetal macrosomia, pelvic tumors (ovarian, fibroids), contracted pelvis. NOT caused by cystocele/rectocele or distended bladder directly (though bladder distension can impede descent).
- **Labor Dystocia (Abnormal Progress):**
  - Protracted active phase: Slow dilatation (<1-1.5 cm/hr).
  - Arrest of active phase: No change in dilatation for >=2-4 hours with adequate contractions.
  - Prolonged latent phase: >20 hrs (nullipara), >14 hrs (multipara).
  - Arrest of descent: No fetal descent in second stage.
  - Example: Cervix changing 6 → 9 cm over 2 hrs is normal progress in active phase.
- **Induction of Labor (IOL):**
  - Indications: Post-term pregnancy (>41 weeks), PROM at term, pre-eclampsia, fetal growth restriction, fetal demise, maternal diabetes, Rh isoimmunization, other maternal/fetal conditions. Active genital herpes is indication for C-section, NOT IOL. Placenta previa, cord presentation, prior classical C-section are CONTRAINDICATIONS. Post-term gestation IS an indication.
  - Contraindications: Placenta previa, vasa previa, cord presentation, transverse lie, previous classical C-section, active genital herpes, prior uterine rupture, invasive cervical cancer. Polyhydramnios, twins, occipitoposterior position, accidental hemorrhage are NOT absolute contraindications (may require careful consideration). Previous LSCS is NOT an absolute CI (VBAC possible). Fetal renal anomaly/hydrocephaly are not standard CIs unless causing obstruction. Macrosomia is relative CI.
  - Methods: Membrane sweep, amniotomy (ARM), Prostaglandins (PGE2 gel/pessary, PGE1 Misoprostol), Oxytocin infusion, Balloon catheter.
  - Bishop Score: Assesses cervical favorability for induction. Components: Dilatation, Effacement, Station, Consistency, Position. Score >6-8 suggests favorable cervix. Length of cervix is implicitly part of effacement. Position of presenting part is NOT a component.
  - Risks: Uterine hyperstimulation, fetal distress, uterine rupture (esp. with previous scar), failed induction, increased C-section rate, PPH, infection. Increases risk of PPH, infection, uterine rupture, cord accidents. Does NOT necessarily increase risk of premature ROM (often done *after* ROM).
- **Assisted Vaginal Delivery (Forceps/Vacuum):**
  - Indications: Prolonged second stage, fetal distress in second stage, maternal exhaustion, maternal medical conditions (e.g., cardiac disease - shorten second stage). Cephalopelvic disproportion is a CONTRAINDICATION. Occipitoposterior position may require rotational forceps.
  - Prerequisites: Fully dilated cervix (NOT 8cm), ruptured membranes, engaged head, known position, adequate pelvis, informed consent, adequate analgesia, skilled operator, empty bladder. Adequate personnel needed. Station +1 or below usually required (maybe 0 station for outlet).
  - Contraindications: Face/brow presentation, unengaged head, CPD, incomplete dilatation, fetal bleeding disorder (vacuum). Delivery after coming head in breech CAN use specific forceps (Piper). Occipito-anterior positions are suitable. Occipito-posterior may require rotation or specific forceps.
  - Vacuum vs Forceps: Vacuum associated with less maternal trauma but more neonatal cephalhematoma, retinal hemorrhages. Forceps associated with more maternal trauma but fewer neonatal complications like

cephalhematoma. Apgar scores not significantly different typically. Vacuum associated with *more* cases of cephalhematoma. Maternal morbidity may be less with vacuum.

- Dangers of Vacuum: Scalp laceration/bruising, cephalhematoma, subgaleal hemorrhage, intracranial hemorrhage, retinal hemorrhage, fetal distress (if prolonged). Does not typically cause APH, ruptured uterus, PPH directly (these relate more to indication/labor itself).

- **Cesarean Section (C/S):**
  - Indications: Previous classical C/S, placenta previa, malpresentation (breech, transverse lie), cord prolapse, fetal distress unresponsive to resuscitation, failure to progress, active herpes, HIV (high viral load), maternal request, prior uterine surgery (e.g., extensive myomectomy). Most frequent indication in recent years: previous C/S or failure to progress. Previous C/S is commonest indication. Fetal malpresentation is a major indication. Non-reassuring FHR pattern is an indication. Antepartum hemorrhage (previa/abruption) is an indication.
  - Classical C/S: Vertical incision in upper uterine segment. Higher risk of rupture in subsequent pregnancy. Indications: Preterm breech (<28-32 wks), transverse lie with back inferior, structural anomaly, anterior placenta previa, post-mortem C/S, cervical cancer requiring hysterectomy. Anterior uterine wall fibroids are NOT necessarily an indication for classical incision. Previous classical IS indication for repeat C/S. Perimortem operation IS an indication.
  - Lower Segment C/S (LSCS): Transverse incision in lower uterine segment. Lower risk of rupture.
  - VBAC (Vaginal Birth After Cesarean): Trial of labor after one previous LSCS. Success depends on reason for previous C/S, favorable cervix, spontaneous labor. Best success rate if previous C/S was for non-recurring reason (e.g., breech) and patient has had previous vaginal delivery. Poorer success if prior C/S for CPD/failure to progress.
  - Complications: Hemorrhage, infection (endometritis, wound), injury to bladder/ureter/bowel, VTE, anesthetic complications, longer recovery, respiratory distress in newborn, risk of uterine rupture/placenta accreta in future pregnancies. Maternal mortality slightly higher than vaginal delivery. Prophylactic antibiotics reduce infection risk.
- **Postpartum Hemorrhage (PPH):** Blood loss >500ml (vaginal) or >1000ml (C/S) within 24h (primary) or >24h up to 6-12wks (secondary).
  - Causes (4 T's): Tone (Atony - most common cause), Trauma (lacerations, rupture), Tissue (Retained placenta/products), Thrombin (Coagulopathy).
  - Primary PPH Commonest Cause: Uterine atony.
  - Risk factors for Primary PPH: Prolonged labor, induced/augmented labor, multiple gestation, polyhydramnios, macrosomia, grand multiparity, fibroid uterus, history of PPH, chorioamnionitis, general anesthesia, operative delivery. Premature labor is NOT a typical risk factor for primary PPH itself. Placental abruption IS a risk factor.
  - Management: Call for help, ABCs (IV access, fluids, oxygen), uterine massage, oxytocics (Oxytocin, Ergometrine, Carboprost, Misoprostol), examine for trauma/retained products, manual removal of placenta, balloon tamponade, B-Lynch suture, uterine artery ligation/embolization, hysterectomy. Consider blood transfusion early. Give iron supplements after stabilization.
  - PPH unresponsive to oxytocin/massage: Suspect retained placenta, lacerations, uterine rupture, coagulopathy. If uterus firm, check for lacerations/trauma (most likely cause if uterus firm immediately post-delivery).
- **Puerperal Infection:** Infection of genital tract within 6 weeks postpartum.
  - Risk Factors: Prolonged ROM, prolonged labor, multiple vaginal exams, manual removal of placenta, C-section, retained products, anemia, diabetes. Prolonged pregnancy itself is NOT a direct risk factor.
- **Shoulder presentation impacted, living fetus:** Managed by Cesarean section.

Pregnancy Complications

Hypertensive Disorders

- **Chronic Hypertension:** BP >=140/90 mmHg before pregnancy or <20 weeks gestation.
- **Gestational Hypertension:** BP >=140/90 mmHg after 20 weeks gestation, no proteinuria.
- **Pre-eclampsia (PET):** BP >=140/90 mmHg after 20 weeks gestation PLUS Proteinuria (>=300mg/24hr or PCR >=30mg/mmol or dipstick 1+).

- **Risk Factors:** Nulliparity (primigravida), previous PET, chronic hypertension, renal disease, diabetes mellitus, multiple pregnancy, molar pregnancy, antiphospholipid syndrome, advanced maternal age (>40), obesity, family history. Previous polycystic ovary syndrome is NOT a direct risk factor. Positive history of macrosomic baby is NOT a risk factor. Sex of baby is NOT a risk factor. Multiparity is associated with *lower* risk (unless other factors present).
- **Pathophysiology:** Vasospasm, endothelial dysfunction. Affects multiple organs.
- **Symptoms/Signs (Severe PET):** BP >=160/110, heavy proteinuria (>5g/24hr), headache, visual disturbances, epigastric/RUQ pain, oliguria (<500ml/24hr), pulmonary edema, fetal growth restriction (IUGR), elevated liver enzymes, low platelets (thrombocytopenia <100,000), hyperreflexia/clonus. Proteinuria 200mg/24h is NOT severe. Dizziness is non-specific. Thrombocytopenia IS a sign of severe disease. Platelet count >100,000 is NOT severe PET. Retinal hemorrhage/papilledema, severe hypertension, pulmonary edema ARE severe manifestations.
- **Complications (Maternal):** Eclampsia, HELLP syndrome, placental abruption, stroke, renal failure, liver rupture/failure, DIC, pulmonary edema, death. Increased risk of postpartum hemorrhage, venous thromboembolism.
- **Complications (Fetal):** IUGR, prematurity (iatrogenic), placental abruption, oligohydramnios, fetal distress, stillbirth.
- **Management:** Monitoring (BP, urine, labs, fetal well-being), antihypertensives (Labetalol, Nifedipine, Methyldopa - aim <150/100), Magnesium Sulphate (MgSO4) for seizure prophylaxis/treatment in severe PET/eclampsia (drug of choice), delivery (definitive cure). MgSO4 toxicity monitored by reflexes, respiratory rate, urine output; loss of deep tendon reflexes is early sign; calcium gluconate is antidote. Delivery timing based on severity, gestational age. Delivery warranted before 37 weeks for severe PET.

- **Eclampsia:** Seizures in a woman with pre-eclampsia. Obstetric emergency. Management: ABCs, MgSO4, control BP, deliver.
- **HELLP Syndrome:** Hemolysis, Elevated Liver enzymes, Low Platelets. Variant of severe PET. Associated with high maternal/fetal morbidity/mortality. Cerebral hemorrhage is a common cause of maternal death.
- **Chronic HTN in Pregnancy Complications:** Superimposed pre-eclampsia, IUGR, placental abruption, preterm delivery. Placenta previa is NOT associated.

Antepartum Hemorrhage (APH)

- **Definition:** Bleeding from genital tract after viability (~24 weeks) until delivery. Passage of show is cervical mucus plug, not true APH. Can be due to fetal bleeding (e.g. vasa previa), but usually maternal. Associated with increased perinatal morbidity/mortality.
- **Placenta Previa:** Placenta implanted partially or wholly in lower uterine segment.
  - **Risk Factors:** Previous C-section (risk increases with number of scars), previous previa, multiparity, multiple gestation, advanced maternal age, smoking, previous uterine surgery/curettage, IVF. Nulliparity is NOT a risk factor. Congenital anomalies of uterus ARE a risk factor. Malposition is NOT a typical risk factor. Large placenta IS a risk factor. Prior classical C-section increases risk significantly.
  - **Presentation:** Painless, bright red vaginal bleeding. Often recurrent episodes. Uterus usually soft, non-tender. Fetal heart often normal initially.
  - **Diagnosis:** Ultrasound (transvaginal US is safe and accurate). Clinical exam (vaginal exam contraindicated until previa excluded by US). MRI may be used for suspected accreta.
  - **Management:** Depends on gestational age, severity of bleeding, maternal/fetal condition. Conservative management if preterm and stable (hospitalization, steroids). Delivery (usually C-section) if term, significant bleeding, or fetal/maternal compromise. Patients with APH generally require delivery by C-section, especially with previa.
  - **Complications:** PPH (due to poor lower segment contractility, accreta), placenta accreta spectrum, preterm delivery, fetal hypoxia/death. Associated with IUGR. Post C-section complication: Amenorrhea (Sheehan's or Asherman's).
- **Placental Abruption:** Premature separation of normally implanted placenta.
  - **Risk Factors:** Chronic hypertension, pre-eclampsia, previous abruption, trauma, smoking, cocaine use, PPROM, multiple gestation, polyhydramnios, thrombophilias (Factor V Leiden - heterozygous noted), advanced maternal age, increasing parity. Low body mass index is NOT a risk factor. IVF pregnancy may have slightly increased risk. Intrauterine infections ARE a risk factor. Maternal anxiety is NOT a direct cause.

- **Presentation:** Painful vaginal bleeding (can be concealed), uterine tenderness/rigidity ('woody' feel), fetal distress/death. Symptoms depend on degree of separation. Abdominal tenderness is suggestive. Fetal lie usually normal.
- **Primary Cause:** Unknown in many cases. Vascular disruption is underlying mechanism.
- **Complications (Maternal):** Hemorrhagic shock, DIC (consumptive coagulopathy), renal failure (acute tubular necrosis), Sheehan's syndrome, PPH, hysterectomy. (Fetal): Hypoxia, IUGR, prematurity, death.
- **Management:** ABCs, IV access, fluids, blood transfusion, monitoring, delivery (often urgent C-section if fetal distress/severe maternal compromise; vaginal delivery may be possible if mild/fetus deceased). Steroids if preterm. Start Clexane therapy is NOT appropriate acute management. Perform forewater amniotomy may be done to expedite delivery if vaginal birth pursued. Induce labor is an option in stable cases with dead fetus.

- **Vasa Previa:** Fetal vessels run in membranes over/near cervix, unsupported by placenta/cord. High risk of fetal exsanguination if membranes rupture. Diagnosis antenatally by US with Doppler. Requires planned C-section before labor/ROM.
- **Other Causes:** Cervical/vaginal lesions, uterine rupture (rare antenatally).

Diabetes Mellitus in Pregnancy

- **Pre-existing Diabetes (Type 1 or 2):**
  - **Risks (Maternal):** Worsening nephropathy/retinopathy, DKA, hypoglycemia, pre-eclampsia, C-section, PPH.
  - **Risks (Fetal/Neonatal):** Congenital anomalies (cardiac, NTDs, caudal regression syndrome - most specific), miscarriage, macrosomia, IUGR (if vascular disease), stillbirth, birth trauma (shoulder dystocia), neonatal hypoglycemia, hyperbilirubinemia, polycythemia, respiratory distress syndrome (delayed lung maturity). Increased risk of oligohydramnios is NOT typical (polyhydramnios more common). Fetal distress during labor is increased. Risk of fetal death highest in 3rd trimester if poor control. Breech presentation is NOT a specific risk of diabetes itself. Cesarean section rates are increased.
  - **Management:** Preconception counseling crucial (optimize glycemic control HbA1c <6.5-7%, folic acid 5mg). Tight glycemic control during pregnancy (insulin preferred). Regular fetal monitoring (US for growth/anomalies, BPP, CTG). Plan delivery timing/mode (often induction ~38-39 weeks or C/S). Treat retinopathy before conception if present. Stop smoking. Modification of some oral hypoglycemics often needed before/during pregnancy.
- **Gestational Diabetes Mellitus (GDM):** Glucose intolerance first diagnosed during pregnancy.
  - **Risk Factors:** Previous GDM, family history of DM, obesity, advanced maternal age, previous macrosomic baby (>4-4.5kg), PCOS, ethnicity (South Asian, Black, Hispanic). Previous delivery of large baby IS a risk factor. High BMI at booking IS a risk factor. Family history of Type 1 DM IS a risk factor. Polycystic ovarian syndrome IS a risk factor.
  - **Screening/Diagnosis:** Usually screened at 24-28 weeks (e.g., 50g GCT followed by 75/100g OGTT).
  - **Management:** Dietary modification, exercise, blood glucose monitoring. Insulin if targets not met (sometimes Metformin/Glyburide used). Fetal monitoring similar to pre-existing DM.
  - **Risks:** Similar to pre-existing DM but generally lower risk of congenital anomalies (as occurs after organogenesis). Increased risk of macrosomia, shoulder dystocia, neonatal hypoglycemia, pre-eclampsia. No increased risk of miscarriage typically. Neonatal hyperglycemia is rare (hypoglycemia is the concern).
  - **Postpartum:** Screen for persistent DM 6-12 weeks postpartum.
- **Maternal Islet cell damage:** is NOT causal factor in GDM. Maternal insulin requirements INCREASE during pregnancy (due to placental hormones), fall rapidly after delivery.

Preterm Labor (PTL) & Preterm Birth (PTB)

- **Definition:** Labor between 20-37 weeks gestation. PTB is birth <37 weeks.
- **Risk Factors:** Previous PTB (strongest predictor), multiple gestation, uterine anomalies, cervical incompetence, infections (UTI, BV, chorioamnionitis), PPROM, polyhydramnios, APH, smoking, low socioeconomic status, stress, short cervical length. Urinary tract infection is a major risk factor. Cervical incompetence/uterine anomalies are causes. Multiple gestation is most common single known cause. Pregnancy-associated hypertension is NOT a primary cause of *spontaneous* PTL (though may lead to indicated preterm birth). Least common cause among primary factors might be cervical incompetence/uterine anomalies compared to infection/multiple gestation.

- **Diagnosis:** Regular uterine contractions PLUS cervical change (dilatation/effacement). Fetal fibronectin testing can help predict risk in symptomatic women (negative test has high negative predictive value). Subjective assessment can be difficult.
- **Management:**
  - **Tocolysis:** Medications to suppress contractions (delay delivery to allow steroid administration). Agents: Beta-mimetics (ritodrine, terbutaline), Magnesium sulfate, Calcium channel blockers (Nifedipine), Prostaglandin inhibitors (Indomethacin - risk of ductus arteriosus closure >32wks), Oxytocin antagonists (Atosiban). Magnesium sulfate is used. Calcium channel blockers used. Prostaglandin inhibitors used. Oxytocin antagonists used. Cyclo-oxygenase inhibitors (NSAIDs like Indomethacin) are used. Methergine is an oxytocic, used for PPH, contraindicated in PTL.
  - **Antenatal Corticosteroids:** (Betamethasone/Dexamethasone) Given between 24-34 weeks to accelerate fetal lung maturity, reduce RDS, IVH, NEC. Steroids are NOT contraindicated.
  - **Magnesium Sulfate:** For neuroprotection if birth <30-32 weeks.
  - **GBS Prophylaxis:** If GBS status positive or unknown.
- **Complications of Prematurity:** Respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH), necrotizing enterocolitis (NEC), sepsis, long-term neurodevelopmental impairment. Pulmonary immaturity and IVH are main causes of morbidity/mortality. Sepsis is a major cause of morbidity.

Premature Rupture of Membranes (PROM / PPROM)

- **PROM:** Rupture before onset of labor at term (>=37 weeks).
- **PPROM:** Rupture before 37 weeks gestation.
- **Diagnosis:** History of fluid gush/leakage, sterile speculum exam (pooling of fluid, positive nitrazine test - turns blue, ferning pattern on microscopy).
- **Management:**
  - **Term PROM:** Induction of labor usually recommended if labor doesn't start spontaneously within ~12-24 hours (due to infection risk).
  - **PPROM (Preterm):** Management depends on gestational age.
    - 34 weeks: Usually deliver (induce labor).
    - <34 weeks: Expectant management (if no infection/fetal distress) - hospitalization, steroids, antibiotics (latency antibiotics to prolong pregnancy/reduce infection), fetal monitoring. Least important factor in decision making might be amount of fluid lost compared to GA, infection signs, fetal well-being. Deliver if chorioamnionitis, fetal distress, or labor ensues. Conservative management for one week to allow steroid benefit IS an option if stable before ~34 weeks. Induction at 38 weeks if no chorioamnionitis is incorrect if PPROM occurs much earlier. Admission to ward for observation is standard. IV fluid replacement does not stop leakage.

Multiple Gestation

- **Types:** Dizygotic (DZ, fraternal) from 2 eggs/2 sperm. Monozygotic (MZ, identical) from 1 egg/1 sperm splitting.
- **Chorionicity/Amnionicity:** Based on timing of MZ split. Dichorionic Diamniotic (Di/Di - split days 0-3), Monochorionic Diamniotic (Mo/Di - split days 4-8), Monochorionic Monoamniotic (Mo/Mo - split days 9-12), Conjoined (split >12 days).
  - All DZ twins are Di/Di.
  - Most MZ twins (~70%) are Mo/Di. Mo/Mo twins are rare (~1%). Di/Di MZ twins ~30%.
  - Di/Di twins are more common than Mo/Mo twins.
  - Mo/Mo twins always same sex. Mo/Di twins always same sex. Di/Di twins can be same or different sex (if DZ).
- **Risks:** Increased risk of almost all pregnancy complications - preterm labor/birth, PPROM, pre-eclampsia, GDM, anemia, APH (previa, abruption), PPH, C-section, congenital anomalies, IUGR, twin-twin transfusion syndrome (TTTS - Mo/Di only), cord entanglement (Mo/Mo only). Diabetes mellitus risk IS increased. Placenta previa risk IS increased. Pre-eclampsia risk IS increased. IUGR risk IS increased. Malpresentation IS increased.
- **Management:** Increased surveillance (ultrasound for growth, TTTS screening in monochorionic), nutrition advice, plan delivery timing/mode (often earlier delivery recommended, mode depends on presentation/chorionicity). Second twin delivery can be assisted by vacuum. PPH common after 2nd stage. Delivery usually by C-section if first twin non-

vertex or monochorionic complications exist, but vaginal delivery possible if first twin vertex and criteria met. Always deliver by C-section is FALSE. CTG monitoring during labor is standard.

- **TTTS:** Vascular anastomoses in monochorionic placenta lead to unbalanced blood flow. Recipient twin polycythemic, polyhydramnios; Donor twin anemic, oligohydramnios, IUGR. Diagnosis: Ultrasound criteria (discordant fluid/growth, bladder visibility). Includes same sex fetuses, intertwin birth weight difference >20%, polyhydramnios in recipient, oligohydramnios in donor. Hemoglobin difference >5 g/dl is also a criterion. Large bladder in recipient is typical. Polyhydramnios in the *smaller* twin is incorrect (it's in the recipient, usually larger).

Intrauterine Growth Restriction (IUGR) / Fetal Growth Restriction (FGR)

- **Definition:** Fetus fails to reach growth potential. Often defined as EFW <10th centile or abdominal circumference (AC) <10th centile.
- **Causes:** Maternal (hypertension, smoking, malnutrition, substance abuse, chronic disease), Fetal (chromosomal abnormalities, congenital anomalies, infections), Placental insufficiency (most common). Controlled DM is NOT a cause (uncontrolled can cause IUGR or macrosomia). Systemic lupus IS a cause. Thrombophilia IS a cause. Steroid therapy can be associated.
- **Diagnosis:** Serial ultrasound measurements (EFW, AC). AC is most sensitive single biometric parameter. Doppler velocimetry (umbilical artery, MCA, ductus venosus) assesses placental function/fetal adaptation.
- **Management:** Identify cause, serial monitoring (growth, Doppler, BPP/CTG), timely delivery.
- **Complications (Neonatal):** Hypoglycemia, hypothermia, polycythemia, birth asphyxia, increased perinatal mortality. Respiratory distress syndrome IS a complication. Neonatal hypoglycemia IS a complication. Sudden infant death syndrome (SIDS) risk may be increased. Cerebral palsy risk increased. Atherosclerosis in adulthood (fetal programming) is NOT a direct neonatal complication.

Oligohydramnios & Polyhydramnios

- **Amniotic Fluid Index (AFI):** Sum of deepest vertical pockets in 4 quadrants. Oligo <5cm, Poly >20-25cm.
- **Oligohydramnios (Low Fluid):**
  - Causes: PPROM, placental insufficiency (IUGR), post-term pregnancy, fetal renal anomalies (bilateral renal agenesis - Potter's syndrome, posterior urethral valves), certain drugs (NSAIDs, ACE inhibitors). Duodenal atresia causes POLYhydramnios. Polycystic kidney disease can cause oligo. Chronic amniotic leak IS a cause. Fetal renal agenesis IS a cause. Urinary tract obstruction IS a cause. Uteroplacental insufficiency IS a cause.
  - Complications: Pulmonary hypoplasia, cord compression, fetal distress, limb deformities.
- **Polyhydramnios (Excess Fluid):**
  - Causes: Maternal diabetes, multiple gestation, fetal anomalies (GI obstruction - esophageal/duodenal atresia; CNS - anencephaly; cardiac failure), fetal infections, placental chorioangioma, idiopathic (most common). Polycystic kidneys cause OLIGOhydramnios. Fetal muscular dystrophy IS a cause. Tracheo-esophageal fistula IS a cause. Renal agenesis causes OLIGO. Open spina bifida usually normal fluid or oligo. Anencephaly IS a cause.
  - Complications: Preterm labor, PPROM, cord prolapse, placental abruption, maternal respiratory distress, PPH (uterine atony).

Fetal Demise / Stillbirth

- **Definition:** Fetal death after 20-24 weeks gestation.
- **Causes:** Often unexplained. Known causes: Placental abruption, chromosomal/congenital anomalies, infections, severe IUGR, cord accidents, maternal disease (diabetes, hypertension, APLAS). Modifiable risk factor with highest ranking: Maternal overweight/obesity.
- **Diagnosis:** Absence of fetal heart activity on ultrasound. Clinical signs (late): absent fetal movements, regression of pregnancy symptoms. Spalding's sign (overlapping skull bones), Robert's sign (gas in circulation), marked spine curvature, fetal maceration are late signs on X-ray/US. First reliable sign is absent fetal heart activity.
- **Management:** Expectant vs Induction of labor. Emotional support. Investigation for cause.

Miscarriage (Spontaneous Abortion)

- **Definition:** Pregnancy loss before viability (<20-24 weeks).

- **Causes:** Chromosomal abnormalities (most common cause in 1st trimester, ~50%), maternal factors (infection, endocrine disorders, thrombophilias, uterine anomalies, cervical incompetence), environmental factors. Aging gametes increase risk. Structural abnormalities of chromosomes less common than numerical (trisomy most common finding). Abnormal zygote/placenta development accounts for many cases. Most parents have normal chromosomes.
- **Types:** Threatened, Inevitable, Incomplete, Complete, Missed, Septic.
- **Incomplete Miscarriage Criteria:** Vaginal bleeding, passage of tissue history, abdominal pain, open cervix on exam, retained products on ultrasound. Empty uterus on scan suggests complete miscarriage.
- **Septic Miscarriage:** Miscarriage complicated by uterine infection. Organisms: Polymicrobial, E.coli, Staphylococcus, Streptococcus, Bacteroides, Clostridium welchii (Perfringens). Management: IV antibiotics, uterine evacuation.
- **Recurrent Miscarriage:** 3+ consecutive losses. Causes: Parental chromosomal abnormalities, uterine anomalies, APLAS, endocrine disorders, thrombophilias, cervical incompetence. Pituitary microadenoma is NOT a typical cause. PCOS is associated. Progesterone deficiency (luteal phase defect) is controversial cause. Cervical incompetence IS a cause. Antiphospholipid syndrome IS a cause.

Ectopic Pregnancy

- **Definition:** Implantation outside uterine cavity.
- **Sites:** Fallopian tube (Ampulla > Isthmus > Fimbria > Interstitial - most common overall is ampulla), ovary, cervix, abdomen.
- **Risk Factors:** Previous ectopic, previous tubal surgery, PID, IUD use (esp. progestogen-only), history of infertility/ART, smoking, advanced maternal age. Combined OCP use is PROTECTIVE. History of pelvic inflammatory disease IS a risk factor. History of pelvic surgery IS a risk factor. Use of fertility drugs IS a risk factor. Smoking IS a risk factor. Diethylstilbestrol exposure not a major risk factor.
- **Presentation:** Amenorrhea, vaginal bleeding, lower abdominal pain. Shoulder tip pain (diaphragmatic irritation from blood). Can present with collapse if ruptured.
- **Diagnosis:** Transvaginal ultrasound (empty uterus +/- adnexal mass/free fluid), serial β-hCG measurements (suboptimal rise or plateau). Laparoscopy is gold standard for diagnosis and treatment. Positive pregnancy test + clinical suspicion.
- **Management:**
  - Expectant (if hCG low/falling, asymptomatic).
  - Medical: Methotrexate (if stable, unruptured, hCG <5000, no fetal heartbeat, compliant patient). Interferes with folate metabolism / DNA synthesis (not RNA).
  - Surgical: Laparoscopy preferred. Salpingectomy (remove tube) or Salpingotomy (incise tube, remove pregnancy - risk of persistent trophoblast).
- **Incidence:** Has increased in recent decades.

Infections in Pregnancy

- **Rubella:** Risk highest in 1st trimester (Congenital Rubella Syndrome - deafness, cataracts, cardiac defects, microcephaly). Vaccination is live attenuated - contraindicated in pregnancy. Intracranial calcification is NOT typical of rubella (seen in CMV, Toxo).
- **Chickenpox (Varicella Zoster Virus):** Exposure in non-immune pregnant woman: Give Varicella Zoster Immunoglobulin (VZIG) ASAP (ideally within 96h). Acyclovir treatment if develops chickenpox (safe in 3rd trimester). Risk of Fetal Varicella Syndrome (skin scarring, limb hypoplasia, eye/brain damage) highest if infection <20 weeks (~1-2% risk). Risk is NOT 15%. Vaccine (live) contraindicated.
- **Group B Streptococcus (GBS):** Colonizes vagina/rectum in 10-30% women. Leading cause of neonatal sepsis (non-iatrogenic). Risk factors for neonatal disease: Preterm birth, prolonged ROM (>18h), intrapartum fever, previous GBS baby, GBS bacteriuria. Screening ~35-37 weeks. Intrapartum antibiotic prophylaxis (Penicillin) for carriers or risk factors reduces early-onset disease. Mortality rates high (~5-10%) but not 20%. Screening at 28 weeks is too early.
- **Urinary Tract Infection (UTI):** Asymptomatic bacteriuria common (~2-10%). Screen at booking. Treat to prevent pyelonephritis. Acute pyelonephritis risk: Preterm labor, sepsis. Treatment requires hospitalization, IV antibiotics. Commonest adverse outcome is preterm labor.
- **Congenital Infections (TORCH):** Toxoplasmosis, Other (Syphilis, VZV, Parvo), Rubella, CMV, Herpes. Can cause IUGR, anomalies, neurodevelopmental problems. HIV is congenital but not usually grouped under TORCH acronym. HIV is not

classically congenital intrauterine infection like Rubella/CMV/Toxo.

Medical Disorders & Pregnancy

- **Epilepsy:** Preconception counseling vital. Aim for monotherapy at lowest effective dose. Folic acid 5mg recommended. Some anti-epileptic drugs (AEDs) are teratogenic (Valproate highest risk - NTDs; Phenytoin - fetal hydantoin syndrome; Carbamazepine - NTDs). Carbamazepine IS associated with NTDs. Newer AEDs generally safer. Risk of congenital malformations needs explanation. Stress compliance. Alter medication based on seizure control BEFORE pregnancy. Reduce to monotherapy if possible. Stop medication during embryonic phase is generally NOT advised unless seizure-free for years. Breastfeeding usually safe (monitor infant). Vitamin K from 36 weeks recommended if on enzyme-inducing AEDs. IV Magnesium Sulphate is treatment for eclamptic seizures, NOT status epilepticus in labor (use IV benzodiazepines/phenytoin).
- **Thrombophilia:** Inherited or acquired tendency to VTE. Increased risk of VTE in pregnancy/puerperium. Associated with recurrent miscarriage, IUGR, placental abruption, severe pre-eclampsia. Requires thromboprophylaxis (e.g., LMWH) depending on risk factors/history. Placenta previa is NOT associated.
- **Thyroid Disease:**
  - Hypothyroidism: Needs thyroxine dose increase (often 30-50%). Untreated associated with miscarriage, pre-eclampsia, low birth weight, cognitive impairment in child. TSH is best screening test. Treat urgently if diagnosed.
  - Hyperthyroidism (Graves' most common): Controlled with Propylthiouracil (PTU - preferred 1st trimester) or Carbimazole/Methimazole (risk of aplasia cutis/embryopathy). Aim for maternal free T4/T3 in high-normal range. Fetal risks: goiter, hyper/hypothyroidism. Therapy should maintain free T4/T3 in low normal range is incorrect target. Surgical treatment rarely needed. Total T4 levels increase due to TBG rise, free levels better guide. More than half are due to Graves' disease. Main fetal complications: IUGR, tachycardia (not bradycardia).
- **Cardiac Disease:** Risk depends on specific lesion and functional class. High risk conditions (contraindications): Pulmonary hypertension, severe aortic/mitral stenosis, Marfan with dilated aorta, Eisenmenger syndrome. Mitral stenosis commonest acquired lesion. Shorten second stage (assisted delivery). Epidural preferred pain relief. Prophylactic antibiotics for prosthetic valves/prior endocarditis. Labor should be managed carefully, induction only if indicated (not routine at 38wks unless specific reason).
- **Iron Deficiency Anemia:** Common in pregnancy. Iron demand increases (total ~1000mg). Diagnosis: Low Hb, low ferritin, low MCV (<85 fL). Oral iron supplement (e.g., 100-200mg elemental iron/day) is treatment. 10mg daily is prophylaxis, not treatment. IV iron if severe/intolerant/late gestation. Blood transfusion if severe/symptomatic near term. Definition of anemia in 3rd trimester: Hb <11 g/dl.

Antenatal Care & Fetal Assessment

- **Booking Visit:** Ideally by 10-12 weeks. Includes history, exam, dating scan, screening tests (blood group, antibodies, Hb, infections - HIV, Syphilis, Hep B, Rubella immunity), urine culture. Optimum time considered 8-10 or 10-12 weeks. 11-14 weeks allows nuchal translucency scan.
- **Subsequent Visits:** Monitor BP, urine (protein/glucose), fetal growth (SFH), fetal heart auscultation, fetal movements. 28-week visit: Repeat Hb, antibodies (if Rh neg), offer Anti-D, screen for GDM. Routine checks include urine protein, sugar, maternal BP, fetal heart. Fetal presentation assessed later (~36 weeks), not routinely at 28 weeks.
- **Dating Pregnancy:**
  - Last Menstrual Period (LMP): Reliable if cycles regular, known LMP. Naegele's rule (LMP + 9 months + 7 days) estimates EDD. Average pregnancy 280 days (40 weeks) from LMP.
  - Ultrasound: Most accurate dating in 1st trimester using Crown-Rump Length (CRL). Biparietal diameter (BPD), Head Circumference (HC), Femur Length (FL), Abdominal Circumference (AC) used later. Best measurement for dating is 1st trimester CRL. LMP dates less accurate than early USS, especially if cycles irregular. Head circumference useful up to ~20-24 weeks. Measurement of choice at 9 weeks: CRL. Most accurate time overall: First trimester scan.
- **Calculating EDD:** Based on reliable LMP or early USS. Example: LMP May 7th → EDD Feb 14th.
- **Screening for Chromosomal Abnormalities (e.g., Down Syndrome):**
  - Combined Test (11-14 weeks): Nuchal Translucency (NT) scan + maternal serum (free β-hCG, PAPP-A).
  - Quad Test (15-20 weeks): Maternal serum (AFP, hCG, uE3, Inhibin-A).
  - Triple Test (older): AFP, hCG, uE3. Screening test for Down syndrome and Edward syndrome.

    - Low PAPP-A associated with: Impending fetal death, preterm labor, low birth weight, IUGR. NOT typically postdate pregnancy.
    - Non-Invasive Prenatal Testing (NIPT): Cell-free fetal DNA in maternal blood. High sensitivity/specificity.
    - Diagnostic Tests (if high risk): Chorionic Villus Sampling (CVS ~11-14 weeks), Amniocentesis (~15+ weeks). Amniocentesis after 16 weeks not associated with fetal postural deformities. Can diagnose infections (Toxo PCR), assess lung maturity, diagnose Turner's. Associated with small risk respiratory distress if early.
- **Screening for Neural Tube Defects (NTDs):**
    - Maternal Serum Alpha-Fetoprotein (MSAFP): Measured ~15-20 weeks (most sensitive at 16-18 weeks). Elevated levels suggest NTD (or multiple gestation, incorrect dates, ventral wall defect).
    - Detailed Ultrasound (Anomaly Scan): ~18-20 weeks.
- **Assessment of Fetal Well-being:**
    - Fetal Kick Counts (Maternal): Subjective, useful screening from ~28 weeks.
    - Non-Stress Test (NST): See Fetal Monitoring section.
    - Biophysical Profile (BPP): See Fetal Monitoring section. Components: NST, breathing, movement, tone, fluid.
    - Ultrasound assessment of fetal breathing movements IS part of BPP.
    - Amniotic fluid volume IS part of BPP.
- **Congenital Anomalies:**
    - Causes: Chromosomal, single gene, multifactorial, teratogens.
    - Screening: Ultrasound (nuchal translucency, anomaly scan), maternal serum screening.
    - Diagnosis: Invasive testing (CVS, amnio), detailed ultrasound, fetal MRI.
    - Specific Anomalies & Associations: See relevant sections (Oligo/Polyhydramnios, Diabetes, Epilepsy). Gastroschisis is NOT typically associated with chromosomal abnormalities. Diaphragmatic hernia, Exomphalos, Duodenal atresia, Cystic hygroma ARE associated.

Physiological Changes in Pregnancy

- **Cardiovascular:**
    - Cardiac Output: Increases 30-50%. Due to increased Stroke Volume and Heart Rate.
    - Blood Pressure: Decreases in 1st/2nd trimester (nadir ~20 weeks), returns towards baseline in 3rd. Diastolic decreases more than systolic. Increase above pre-pregnancy levels in 3rd trimester is abnormal.
    - Systemic Vascular Resistance: Decreases significantly. Peripheral resistance decreases.
    - Heart Sounds: Exaggerated splitting of S1, loud S1, S3 gallop common. Ejection systolic murmur common (flow murmur). Diastolic murmur is always PATHOLOGICAL.
    - ECG: Left axis deviation. Ectopic beats may increase.
- **Hematological:**
    - Plasma Volume: Increases 40-50%.
    - Red Cell Mass: Increases 20-30%. Disproportionate increase leads to hemodilution ('physiological anemia').
    - Hemoglobin/Hematocrit: Decrease. Anemia defined as Hb <11 (1st/3rd trimesters), <10.5 (2nd).
    - White Blood Cells: Increase (esp. neutrophils). Increase in polymorphonuclear leukocytes occurs. T helper 1 / NK cells decrease (relative shift to Th2).
    - Platelets: May decrease slightly (gestational thrombocytopenia).
    - Coagulation Factors: Increase in fibrinogen, Factors VII, VIII, IX, X, Von Willebrand factor. Decrease in Protein S. Factor XI decreases. Pregnancy is hypercoagulable state. Activated Protein C resistance increases.
    - ESR: Markedly elevated. Normal ESR is not expected.
    - Iron: Increased requirement (~1000mg total). Absorption increases but insufficient. Supplementation often needed. About 10% ingested iron absorbed.
- **Renal:**

- Kidneys enlarge. Dilatation of renal pelves/ureters (progesterone effect, uterine pressure).
- Renal Blood Flow & GFR: Increase 50%. Leads to decreased BUN, Creatinine. Creatinine clearance increases.
- Glycosuria: Common due to increased filtered load exceeding tubular reabsorption capacity.
- Proteinuria: Slight increase normal (<300mg/24hr).
- Excretion of folate increases (hence need for supplement). Excretion of urate decreases initially then normalizes.

- **Metabolic:**
  - Diabetogenic state: Increased insulin resistance (due to hPL, progesterone, cortisol). Post-prandial glucose higher. Fasting glucose lower. Accelerated starvation state (esp. early pregnancy), increased ketone production.
  - Weight Gain: Average 11-16 kg (average ~12.5 kg).
  - Basal Metabolic Rate: Increases.
  - Calcium: Increased intestinal absorption (Vit D mediated). Total serum calcium decreases (due to low albumin). Ionized calcium remains stable. PTH levels decrease initially then rise. Calcitonin levels rise. Calcium actively transported across placenta.
  - Lipids: Increased cholesterol, triglycerides. HDL cholesterol increases.
- **Gastrointestinal:**
  - Nausea/Vomiting: Common in 1st trimester (hCG).
  - Appetite: Increases.
  - Reduced Esophageal Sphincter Tone: → Reflux/Heartburn.
  - Decreased Motility/Transit Time: → Constipation. Transit time increases.
  - Gallbladder: Stasis → increased risk gallstones. Biliary cholesterol saturation increases.
  - Liver: Increased alkaline phosphatase (placental origin). AST/ALT normal. Albumin decreases. Binding globulins increase. Increased protein synthesis occurs, but serum albumin *decreases* due to hemodilution/altered synthesis ratios.
- **Respiratory:**
  - Increased Minute Ventilation (due to increased Tidal Volume, Respiratory Rate unchanged). Decreased Functional Residual Capacity, Residual Volume.
  - Chronic compensated respiratory alkalosis (decreased PCO2, decreased bicarbonate). Increased PO2.
- **Endocrine:**
  - Increased Total Thyroxine (T4)/T3 (due to increased TBG). Free T4/T3 normal or slightly low. TSH suppressed initially (hCG effect), then normal. TSH does NOT cross placenta.
  - Increased Cortisol (total & free).
  - Increased Aldosterone, Prolactin, Sex Hormone Binding Globulin. FSH/LH suppressed.
  - Increased Melanocyte Stimulating Hormone (→ skin pigmentation).
  - Increased Human Placental Lactogen (hPL - causes insulin resistance).
  - Increased Estrogen, Progesterone (placental production).
- **Uterus:** Rotates to the right (dextrorotation) due to rectosigmoid colon on left.
- **Other:** Total body water increases (~6-8L, not 3L). Edema common. Palmar erythema/spider veins common.

Obstetric Terminology & Fetal Anatomy

- **Gravidity & Parity:**
  - Gravidity (G): Total number of pregnancies (regardless of outcome).
  - Parity (P): Number of births after viability (usually >20-24 weeks). Often written as TPAL (Term, Preterm, Abortions, Living children).
  - Examples: G3P3 = 3 pregnancies, 3 births past viability. G5P4+1ab = 5 pregnancies, 4 births past viability, 1 abortion. G5P5 (one twin) = 5 pregnancies, 5 births (twins count as one parity event but 2 living children), total 6 children. G1P1 (triplet) = 1 pregnancy, 1 birth event, 3 babies delivered. Molar pregnancy counted as abortion.

- **Lie, Presentation, Position, Attitude:** See Labor & Delivery section.
- **Fetal Skull:**
  - Sutures (membranous gaps) and Fontanelles (junctions) allow moulding. Sutures are NOT ossified at term. Moulding is normal physiological process.
  - Anterior Fontanelle: Diamond shaped, junction of sagittal, coronal, frontal sutures. Closes ~18 months.
  - Posterior Fontanelle: Triangular, junction of sagittal, lambdoid sutures. Closes ~2-3 months.
  - Diameters (Approximate):
    - Suboccipitobregmatic (SOB): 9.5 cm (Vertex presentation, well flexed).
    - Occipitofrontal (OF): 11.5-12 cm (Vertex, less flexed).
    - Occipitomental (OM): 13.5 cm (Brow presentation). Too large to pass through pelvis.
    - Submentobregmatic (SMB): 9.5 cm (Face presentation).
    - Biparietal (BPD): 9.5 cm (Widest transverse diameter).
    - Mentovertical diameter (13.5-14cm) is largest AP diameter, associated with brow presentation. Value 11.5cm incorrect.
- **Placenta & Umbilical Cord:**
  - **Placental Anomalies:**
    - *Succenturiate Lobe:* Accessory lobe connected by vessels. Risk of retained lobe → PPH, infection. May lead to PPH.
    - *Placenta Accreta Spectrum:* Abnormal adherence to uterine wall. Accreta (villi attach to myometrium), Increta (villi invade myometrium), Percreta (villi penetrate through myometrium, potentially into bladder/rectum). Risk factor: previous C/S, previa. Diagnosis by US/MRI. Often requires hysterectomy. Best method for diagnosis: Ultrasound evaluation (MRI if complex). Can cause PPH. Bluish tissue adherent between uterus and bladder suggests percreta.
    - *Vasa Previa:* Fetal vessels in membranes over cervix. High risk fetal bleeding with ROM. Requires C/S delivery.
    - *Circumvallate Placenta.*
  - **Umbilical Cord Anomalies:**
    - *Single Umbilical Artery (SUA):* (~1% births). Associated with increased risk of congenital anomalies (renal, cardiac, chromosomal). Requires careful fetal assessment. Not insignificant finding. Equally common in diabetic/non-diabetic newborns is likely false. Not present in 5% of births.
    - *Velamentous Insertion:* Vessels insert into membranes before reaching placenta. Risk of rupture, vasa previa. More common in monochorionic twins.
    - *True Knot:* Can tighten, cause fetal compromise/death. Occurs earlier in pregnancy, not typically late.
    - *Cord Coiling:* Normal finding. Achordia (absent cord) is rare. Rightward coiling may have higher risk than leftward.
- **Fetal Circulation:** Umbilical vein (1) carries oxygenated blood from placenta. Umbilical arteries (2) carry deoxygenated blood to placenta. Ductus venosus shunts blood from umbilical vein to IVC (bypassing liver). Foramen ovale shunts blood from Right Atrium to Left Atrium. Ductus arteriosus shunts blood from Pulmonary Artery to Aorta (bypassing lungs). Blood returning from lung has low oxygen saturation. Inferior vena cava contains mix of oxygenated (ductus venosus) and deoxygenated blood. Prostaglandins maintain patency of ductus arteriosus. Two umbilical *arteries* & one umbilical *vein*.
- **Embryonic/Fetal Periods:** Embryonic period = fertilization to end of 8th week post-conception (10 weeks gestation LMP). Fetal period = 9th week to birth. Organogenesis largely complete by end of embryonic period. Teratogenic period highest during organogenesis (~weeks 3-8 post-conception / 5-10 weeks LMP). Classic teratogenic period in 28-day cycle often cited as day 17-56 post conception (approx week 5-10 LMP).

Puerperium

- **Definition:** Period after delivery (usually 6 weeks) when maternal physiological changes return to non-pregnant state.
- **Uterine Involution:** Uterus contracts, decreases in size. Immediately after delivery, fundus at umbilicus or slightly below. Descends ~1 cm/day. Returns to pelvis by ~2 weeks, normal non-pregnant size by ~6 weeks (not 12 days).

Myometrial retraction (sustained contraction) is key. Contraction impedes blood flow (main mechanism preventing PPH). Thrombosis of large placental site vessels is secondary mechanism. Fundus immediately postpartum is firm, globular, near umbilicus. Subinvolution: Failure of uterus to return to normal size. Causes: Retained products, infection (endometritis), fibroids, full bladder. Breastfeeding promotes involution (oxytocin release), Caesarean delivery may slightly delay it. Multiparity can sometimes lead to less efficient involution.

- **Lochia:** Postpartum vaginal discharge (blood, tissue, mucus). Rubra (red, first few days) → Serosa (pink/brown, ~day 3-10) → Alba (yellow/white, ~day 10 up to 6 weeks).
- **Menstruation Return:** Variable. Non-breastfeeding women: usually resumes within 6-12 weeks (often by 10 weeks). Breastfeeding women: delayed due to prolactin suppressing ovulation (lactational amenorrhea), can be months.
- **Lactation:** Breast milk production stimulated by prolactin, let-down reflex by oxytocin. Frequent suckling maintains production. COCPs containing >30-35mcg estrogen can suppress lactation (Q625). Progestogen-only methods (mini-pill, Depo-Provera, implants) generally considered safe.
- **Postpartum Collapse (Sudden):** Differential Diagnosis: PPH, amniotic fluid embolism, pulmonary embolism, eclampsia, anesthesia complications (toxicity), uterine rupture, septic shock, cardiac event. Ruptured ectopic pregnancy would not cause *postpartum* collapse.
- **Puerperal Infections:** See Labor & Delivery section.
- **Maternal Self Assessment of Fetal Wellbeing:** This is an ANTENATAL assessment, usually daily fetal kick counts in the third trimester.