Obstetrics

BY

Dr. Haya Yanes
Dr. Noor Hammad

Obstetrics

Contents:

☒ The fetoplacental unit largely controls the endocrinologic events of the pregnancy

✓ HCG levels: it's a glycoprotein

- Begins to rise 8 days after ovulation
- Appears in maternal blood 10 days after fertilization
- In the first trimester, it should double every 72 hours.
- Peaks at 9-10 weeks .
- And then decline to a moderate, more constant level.
- After normal pregnancy, it should return to normal within 2-4 weeks.

✓ Human placental lactogen (HPL):

- Antagonizes the cellular action of insulin and decreases maternal glucose utilization, which increases glucose availability to the fetus. (Play a role in the pathogenesis of gestational diabetes)
- Maternal serum concentrations of hPL parallel placental weight

✓ Corticotropin-Releasing Hormone (CRH)

- important role in the activation and amplification of labor, both preterm and term.

✓ Prolactin

- from the maternal and , in late pregnancy , fetal anterior pituitary For stimulation of postpartum milk production.

☒ Physiological Changes in Pregnancy

- **Skin:** Increased Vascularity and Increased Pigmentation
  - ✓ Spider angioma/ Palmar erythema/ Chadwick sign
  - ✓ Linea Nigra /Chloasma/ melasma / Darkening of the nipple & areola
- **Hair Changes:** Mild hirsutism is common.
- **Ocular Changes:** Increase thickness of the cornea/ Decreases intraocular pressure: Glaucoma improves

- **Hematologic changes:**

✓ **Blood volume: Increases by 50%: Increase of the plasma volume is greater than the increase in erythrocytes, which leads to dilutional anemia (normal physiological anemia).**

✓ **Iron:**

- **Iron Absorption increases with pregnancy.**
- **Requirements increase to 1000 mg/day.**
- **Most iron is used in hematopoiesis in the 2nd half of pregnancy**
- **Average Hb = 12g/dl**

✓ **RBC mass Increases by 30%**

✓ **ESR: CRP & ALP è increases**

✓ **WBC: increases up to 16,000/mm3 in the 3rd trim.**

✓ **Platelets: Mild decrease**

✓ **Coagulation factors: Increase in all factors (mainly I.VII, VIII,IX, X), except factos (XI and XIII)/Increase resistance to activated protein C**

☒ **Pregnancy is a hypercoagulable state which increases the risk for stroke/DVT/ PE.**

- **Cardiovascular Changes:**

Arterial BP decreased : Diastolic more than systolic

Venous BP: Central stable , Femoral increased

Peripheral Vascular resistance PVR decreased : by 30%

Stroke volume and HR increased

increase in cardiac output .

☒ **Diastolic murmurs are NEVER Normal and must be investigated if found.**

- **Gastrointestinal Changes:**

✓ **GERD**

✓ **constipation**

✓ **cholethiasis**

✓ **Increase in protein synthesis (estrogen effect).**

✓ **Decrease in albumin concentration due to dilutional effect.**

✓ **Normal AST, ALT, GGT and bilirubin.**

• Respiratory Changes:

- ✓ The main change in the lungs is Tidal volume (VT): increases by 40%.
- ✓ Respiratory rate (RR) is unchanged.
- ✓ Residual volume: decreased by 20% due to the pressure of the uterus on the
- ✓ Lungs/ Total lung capacity è decreases.

• Renal Changes:

- ✓ Kidney size (increase in blood flow) + Renal pelvis Volume + ureteral
- ✓ volume è All increase.
- ✓ Renal plasma flow + GFR + Cr Clearance Increase
- ✓ BUN + Serum Cr + Serum Uric Acid (UA) decrease

• Endocrine Changes:

- ✓ Pituitary: Increase in size by 100% by term due to increase in blood flow. This makes it susceptible to ischemic injury, Sheehan syndrome, from Postpartum hypotension and hemorrhage.
- ✓ Adrenals: No change in size, but increase in the production of cortisol
- ✓ Thyroid: Thyroid gland increases in size due to increase in blood flow.
  - Thyroid binding globulin TBG increases
  - Total T3,T4 increases due to increase in TBG.
  - TSH, TRH, Free T3,T4 are unchanged!

☒Antenatal Care :

✓ FIRST VISIT and 1st trimester:

- ❑ History and P/E
- ❑ Labs:
  - CBC
  - Blood group, Rh, Antibody screen (Indirect Coombs)
  - UA and urine culture
  - Pap smear
  - Blood sugar
  - Gonorrhea and chlamydia cultures and PCR
  - Infection screen: Rubella/ Syphilis/ HBV/ HIV/ TB

❑ Ultrasound for :

- Accurate dating of gestational age by CRL (crown-rump length)

- **Fetal viability by FHA (fetal heart activity)**
- **Visualization of gestational sac**
- **Site of implantation to rule out ectopic pregnancy**
- **Number of gestational sacs**
- **Nuchal translucency (NT): normal NT is <3mm, it is abnormal when >3mm**

✓ 2nd trimester:

❑ **Labs : Triple marker test: MS-AFP, HCG, Estriol OR Quadruple marker test: MS-AFP, HCG, Estriol, Inhibin-A**

- **DDx for increased MS-AFP (>2.5)**
  - **Wrong date (most common)**
  - **Twin pregnancy**
  - **NTD (Neural Tube Defect)**
  - **Ventral Wall Defects (VWD): (Gastroschisis or omphalocele)**
  - **Renal disease**
  - **Sacrococcygeal teratoma**
- **DDx for decreased MS-AFP (<0.75-0.85)**
  - **Wrong date**
  - **Trisomy**

❑ **Ultrasound :**

- **Rule out congenital anomalies by detailed US between 18-22 weeks (typically at the 20th week)**
- **Amniotic fluid index (AFI)**
- **Cervical length and changes**
- **Cervical incompetence (funneling)**

✓ 3rd trimester

❑ **Ultrasound for**

- **Placental location in relation with the internal os**
- **Biometric measurements in relation to gestational age to rule out Intrauterine growth restriction (IUGR)**

- **Fetal weight**
- **Presentation**
- **Biophysical profile (BPP)**
- **Postmature placental signs (calcifications)**

☒ ***Medical Conditions Complicating Pregnancy***

- **ENDOCRINE DISORDERS:**
- ***THYRIOD DISEASES:***

✓ **Thyroid diseases are relatively common disorders in pregnancy, complicating up to 2% of pregnancies.**

✓ **Thyroid function tests:**

✓ **During pregnancy, the ↑ in Estrogen → ↑ TBG →**

✓ **The serum levels of free T3, free T4 usually remains in normal range.**

✓ **When interpreting serum levels of (TSH) and free T4 and T3, it is best to use a laboratory that has trimester-specific values.**

✓ Complications:

1. increased incidence of prematurity

2. intrauterine growth restriction (IUGR)

3. superimposed preeclampsia

4. stillbirth

5. neonatal morbidity and mortality

6. abortion

☒ Managment :

- **Medical treatment is the first line treatment.**
- **Significant maternal tachycardia: B-blocker**

- **Thioamides are the mainstay of antithyroid therapy: PTU and methimazole are both effective, but they have different safety profiles.**

- **PTU should be used to treat overt hyperthyroidism in the first trimester, and methimazole should be used in the second and third trimesters.**

- **Radioactive iodine is contraindicated in pregnancy**

Surgical management of a pregnant patient with hyperthyroidism during the *second trimester* is recommended only when medical treatment fails

❖ Hypothyroidism

➢ **Complications: if left untreated**

1. Spontaneous abortion
2. Preeclampsia
3. Abruption
4. Low-birth-weight or stillborn infants
5. Lower cognitive function in offspring.

➢ **Diagnosis:**

- **The most important laboratory finding of hypothyroidism is an elevated TSH level, to differentiate between overt and subclinical, we look for free T4 levels. If it is decreased then this is an overt hypothyroidism.**

➢ **Treatment:**

- **Once diagnosed, therapy such as levothyroxine should be started, and serum *TSH levels should be measured monthly* with appropriate adjustments in levothyroxine dosage.**

☒ Heart Disease (HD):

➢ ***1- RHEUMATIC HEART DISEASE:***

The most common lesion associated with rheumatic heart disease is mitral stenosis(MS),
followed by mitral regurgitation.

- ✓ **During pregnancy, the mechanical obstruction associated with MS worsens as cardiac output increases which lead to increase in preload (due to ↑ blood volume), and that increase LA overload, leading to pulmonary HTN. Asymptomatic patients may develop symptoms of cardiac decompensation or pulmonary edema as pregnancy progresses. Atrial fibrillation is more common in patients with severe mitral stenosis, and nearly all women who develop atrial fibrillation during pregnancy experience congestive heart failure.**
- ✓ **25% of patients with MS develop HF for the 1st time during pregnancy.**
- ✓ **MS increase the risk of IUGR.**
- ✓ **Management of MS: ↓ tachycardia, ↓ excessive volume load, considers intrapartum SBE prophylaxis.**

➢ **2-CONGENITAL HEART DISEASE**

- **Patients with persistent atrial or ventricular septal defects and those with tetralogy of Fallot with complete surgical correction generally tolerate pregnancy well.**

Patients with primary pulmonary hypertension or cyanotic heart disease with residual pulmonary hypertension are in danger of experiencing de-compensation during pregnancy

❑ **In general, significant pulmonary hypertension with Eisenmenger syndrome is a contraindication to pregnancy due to the high maternal mortality that accompanies this condition.**

- **Antepartum care in heart disease :**

✓ **AVOIDANCE OF EXCESSIVE WEIGHT GAIN AND EDEMA By rest in left lateral decubitus position at least for 1 hour daily, avoid excessive salt intake, give diuretics if HF is present)**

✓ **AVOIDANCE OF STRENUOUS ACTIVITY**

✓ **AVOIDANCE OF ANEMIA.**

- ✓ **AVOIDANCE OF INFECTION. This is by routine screening for sexually transmitted infections and urinary tract infections, the timely administration of appropriate immunizations**
- ✓ **FETAL ECHOCARDIOGRAPHY. Women with congenital heart disease have an increased risk of having children with heart disease.**

➢ **Management of Delivery and the Immediate Postpartum Period**

- ✓ **Cardiac patients should be delivered vaginally unless obstetric indications for cesarean delivery are present**
- ✓ **labor in the lateral decubitus position**
- ✓ **Frequent assessment of vital signs, urine output, and pulse oximetry. Adequate pain relief is important**
- ✓ **Pushing should be avoided during the second stage of labor (assisted delivery by outlet forceps, vacuum extractor)**
- ✓ **The immediate postpartum period presents special risks to the cardiac patient.**
- ▪ **Monitor fluid balance (avoiding overload)**
- ▪ **Prevention of uterine atony (avoiding depletion from blood loss) with oxytocin and uterine massage.**

Prevent endocarditis : prophylactic antibiotic is given to high risk patients (prosthetic valves, unrepaired or incompletely repaired congenital heart disease, or a previous history of bacterial endocarditi

☒ Gastrointestinal Disorders:

➢ HYPEREMESIS GRAVIDARUM

persistent nausea and vomiting in pregnancy that is associated with ketosis and loss of more than 5% of pre-pregnancy body weight

❖ Risk factors:

1. first pregnancies

2. multiple pregnancies

3. Trophoblastic disease

❖ **Clinical presentation:**

- **A history of intractable vomiting, beginning in the first trimester, significant abdominal pain and tenderness are generally absent.**

Signs: weight loss, dry and coated tongue, and decreased skin turgor

❖ **Work up:**

- **urine tests for ketonuria**
- **blood tests for electrolytes and acetone (electrolyte disturbances usually seen : <u>hypokalemia, hyponatremia, and hypochloremic alkalosis</u>)**
- **blood urea nitrogen (BUN), creatinine, lipase,**
- **liver function and amylase (usually elevated)**

❖ **Treatment is symptomatic.**

- ***<u>If outpatient management fails, patients must be admitted for intravenous administration of fluids, electrolytes, glucose, vitamins, and medical therapy</u>***

➢ GASTROESOPHAGEAL REFLUX DISEASE

- **70% of pregnant women**
- **Clinical presentation:**

✓ **Discomfort aggravated by meals and the recumbent position**

✓ **water brash, which is a salty taste and produces nausea.**

- **Treatment**

▪ **Symptomatic: Upper GI endoscopy is usually not necessary, unless there is significant GI bleeding**

▪ **Give instruction to patient: refrain from eating large and late meals; avoid the recumbent position, especially after meals use an extra pillow to elevate the head when sleeping**

Antacids If failed : H2 receptor antagonist (cimetidine) or a proton pump inhibitor (omeprazole) is indicated

➢ INTRAHEPATIC CHOLESTASIS OF PREGNANCY

❑ **Features**

✓ **cholestasis and pruritus in the second half of pregnancy without other major liver dysfunction**

✓ **tendency for recurrence with each pregnancy**

✓ **An association with oral contraceptives and multiple gestations**

✓ **There are usually no maternal hepatic sequelae**

✓ **an increased rate of meconium-stained amniotic fluid and fetal demise**

❑ **Clinical presentation:**

- **1. Itching (most intense on the palms and soles and it is the MAIN SYMPTOM), without abdominal pain or a rash**

☒ Pelvic Anatomy

- **The pelvis is divided into the following four imaginary planes for descriptive purposes.**

  1. The pelvic inlet: where the fetal head enters the pelvis in the transverse position/The transverse diameter of this plane is = 13.5 cm and it is the wider than the AP diameter which is 11 cm.

  2. The plane of greatest diameter: where the fetal head rotates to the anterior position.

  3. The plane of least diameter (Midplane): the most important from a clinical standpoint because most instances of arrest of descent occur at this level.

  4. The pelvic outlet: this plane is the site of low pelvic arrest.

- ## Pelvic shapes:

1. Gynecoid: The classic female type. Found in approximately 50% of women. It is round at the inlet, with the widest transverse diameter only slightly greater than the anteroposterior diameter. The fetal head generally rotates into the occipito-anterior position in this shape.

2. Android: The typical male type of pelvis. It is found in less than 30% of women. It has a triangular inlet. The fetal head is forced to be in occipito-posterior position to conform to this narrow anterior pelvis.

3. Anthropoid: It is found in 20% of women. The AP diameter is much larger than the transverse diameter. Fetal head can engage only in the AP diameter.

4. Platy pelloid: Described as flattened gynecoid. It is found in 3% of women. It has a short AP and wide transverse diameter, giving it an Oval-shaped inlet. The fetal head has to engage in the transverse diameter.

☒ Fetal Head

- ✓ **The cranial bones at birth are thin, weakly ossified, easily compressible, and interconnected only by membranes. These features allow them to overlap under pressure and to change shape to conform to the maternal pelvis, a process known as molding.**
- ✓ **The membrane-occupied spaces between the cranial bones are known as sutures.**
- ✓ **The membrane-filled spaces located at the point where the sutures intersect are known as fontanelles, the most important of which are the anterior and posterior fontanelles**

✓ Labor

- ✓ **Labor is defined as progressive cervical effacement and dilation resulting from regular uterine contractions that occur at least every 5 minutes and last 30 to 60 seconds each**
- ✓ **Presentation: the part of the fetus in the lower pole of the uterus overlying the pelvic brim: types are**
  - **Cephalic or vertex : Occiput/ widest diameter is biparietal (9.5 cm)**

- **Face: mentum**
- **Breech: sacrum/inter-trochanteric (10 cm)**
- **Transverse: shoulder/ Bi-acromial diameter (11cm)**
- **Shoulder: olecranon process**

✓ **Engagement: when the widest diameter of the presenting part of the fetus enters the pelvic inlet.**

✓ **Station: relation between the presenting part and the ischial spines of the mother (at which station is considered ZERO)**

✓ **Position: is the relation of the presenting part and the mother's pelvis**

✓ **Vertex: area between the fontanelles and bounded laterally by the parietal eminences**

✓ **Attitude: is the posture of the fetus**

- **- Normal attitude : full flexion**
- **- Other attitudes; deflexion, extension**

✓ **Lie: describes the orientation of the long axis of the fetus with respect to the long axis of the uterus. Lie can be transverse, longitudinal or oblique.**

➢ **In a longitudinal lie, both axes are aligned.**

- **This is considered the normal lie.**
- **both breech and cephalic presentations have normal lies.**

➢ **In a transverse lie, the long axis of the fetus lies perpendicular to the long axis of the uterus.**

- **In an oblique lie, the fetal long axis is at an angle to the bony inlet. This lie usually is transitory and occurs during fetal conversion between other lies.**

☒ **Both the transverse and oblique lies predispose to *shoulder presentation***

• Position

- ✓ **Refers to position of presenting part of the fetus relative to the maternal pelvis.**
- ✓ **Description of position depends on presentation of fetus.**
- ✓ **In vertex presentation, the occiput is the reference point. Think of fetal occiput and**

its relation to the mother's pelvis (is the occiput at the anterior or posterior of pelvis):

- ❖ **Occipitoanterior (OA): most common position; normal. Occiput of fetus towards symphysis pubis (occiput at anterior of pelvis, hence, occipitoanterior).**
- **Any other position is considered malposition.**
- **** Left OA (LOA) is most common.***
- **Left means the occiput of the fetus is against the left side of the anterior pelvis.**

❖ ***Occipitoposterior (OP):***

- ✓ ***most OP rotate spontaneously to OA.***
- ✓ **May cause prolonged second stage of labor**
- ✓ **Arrested labor may occur when the head does not rotate and/or descend.**
- ✓ **Delivery may be complicated by perineal tears or extension of an episiotomy**

☒ Malpresentation

✓ **Presentation refers to the part of the fetus that is overlying the maternal pelvic inlet.**

- ✓ **Malpresentation encompasses any fetal position other than vertex and includes breech, face, brow, compound, and shoulder presentations.**
- ✓ **In decreasing incidence: breech, face and brow.**
- ✓ **Compound and shoulder presentations are rare**

☒ Breech (MC malpresentation):

- **Occurs when the fetal buttocks or lower extremities present into the maternal pelvis.**

• Etiology:

- ➢ **1- Fetal:**
- **Prematurity : Before 28 weeks, 25% of fetuses are presenting as a breech. However, as the fetus grows and occupies more of the uterus, it tends to assume a vertex presentation to accommodate best to the confines and shape of the uterus.**

☒ ***prematurity is a major factor predisposing to breech presentation.***

- **Fetal structural anomalies (e.g., hydrocephaly) may restrict the ability of the fetus to present as a vertex.**
- **Multiple gestation.**
- ➢ **2- Maternal:**
- **Uterine anomalies (e.g., bicornuate uterus).**
- **Oligo/polyhydramnios.**
- **Contracted maternal pelvis.**
- **Uterine surgery.**
- **Pelvic tumors that obstruct the birth canal.**
- ➢ **3- Placental: placenta previa.**

☒ *Classification:*

- ➢ **Frank (extended):65%**
- ➢ **complete (flexed):25%**
- ➢ **incomplete/footling:10%**

- **In frank breech, the buttocks come first, legs are flexed at hip and extended at knee.**

- **In complete breech, legs are flexed at both hip and knee.**
- **In footling breech, one or both feet come first and the buttocks are at a higher position**

☒ Diagnosis of breech presentation can often be made by the

- ✓ **Leopold examination.**
- **The firm fetal head is palpated in the fundal region and the softer, smaller breech occupies the lower uterine segment above the symphysis pubis.**
- ✓ **Vaginal examination:**
- **In a frank breech, the fetal buttocks, anus, sacrum, and ischial tuberosities can be palpated.**
- ✓ **Breech presentation is confirmed using ultrasound.**

▪ Management

- ❖ **The standard of care now in most practices is to deliver all breeches by cesarean**
- **Principle in breech delivery is masterly inactivity (Hands-off). Let the mother expel the fetus by her own effort with uterine contractions, never pull from below.**

❖ **The following points are criteria for vaginal delivery of a breech presentation:**

- **Gestational age should be at least 36 weeks.**
- **Must be frank or complete breech.**
- **Estimated fetal weight should be between 2.5 and 3.8 kg.**
- **Fetal head must be flexed.**
- **There must be no indication for cesarean delivery.**
- **Anesthetist should attend the delivery.**
- **Obstetrician must be experienced**

☒ Complications of breech presentation:

- ✓ **Umbilical cord prolapse**
- ✓ **entrapment of aftercoming head**

- ✓ **asphyxia.**
- ✓ **Increased risk of CTG abnormalities.**
- ✓ **Birth trauma due to forceful traction on the fetus and can involve the brachial plexus (Erbs palsy).**

☒ ***Face (2nd MC malpresentation)***

- **Occurs when the fetal head is hyper-extended such that the fetal face, between the chin and orbits, is the presenting part.**
- **The presenting diameter is the *submento-bregmatic (9.5 cm).***

➢ **Management**

- ✓ **The reference point for position in face presentation is the fetal chin (mentum).**
- ✓ **If the mentum is facing the symphysis pubis (mento-anterior), vaginal delivery should be expected/ Forceps, but not vacuum, can be applied to assist Mentum posterior cases and mentum transverse must be delivered by cesarean delivery.**

➢ ***Complications***

- **Prolonged labor is common.**
- **When spontaneous vaginal delivery or forceps delivery occurs, perinatal morbidity and mortality for face presentations are similar to those for vertex presentations.**

☒ Brow

- **Occurs when the presenting part of the fetus is between the facial orbits and anterior fontanelle resulting from the extension of the fetal head such that it is midway between flexion (vertex presentation) and hyperextension (face presentation).**
- **The *presenting diameter is the supra-occipito-mental diameter*, which is the *longest anterior-posterior fetal diameter (13.5 cm).***

➢ **Management**

- **Brow presentation is unstable.**
- **50-75% will convert to either a face presentation or vertex and will**

- **subsequently deliver vaginally.**
- **With a persistent brow presentation, the large presenting diameter makes vaginal delivery impossible and cesarean section is required.**

➢ *Stages of labor*

- ***The first stage consists of two phases:***

✓ **Ranges from (6-18 hours) in nulliparous women/(2-10)hours in multiparous women((AVG is 10 hours))**

- ***latent phase*, during which cervical effacement and early dilation (up to 4cm) occur/ The length of the latent phase is highly variable due to possible overlap with preparatory period, and depends largely on the parity (it is longer in primi-parous women.)**
- ***active phase*, during which more rapid cervical dilation occurs(from the end of latent phase until 10cm of dilation)./The active phase progresses by 1-1.2 cm/hr in primiparous and multiparous women, respectively.**

☒ **Three P's affect the active phase:**

✓ **Power: uterine contraction > 200 Montevideo unit are adequate.**

✓ **Passenger: fetus size & position**

✓ **Pelvis: size & shape**

- **In the active phase, at least 1 cm/ hr of dilation in nulliparous**
- **At least 1.2 cm/hr of dilation in multipara**
- **If the rate of dilation is less than this or no cervical changes in station for 2 hours, evaluate the patient for**

✓ **uterine dysfunction**

✓ **Fetal malposition**

✓ **Cephalopelvic disproportion**

☒ **If there is a problem in the passenger or pelvis, cephalopelvic disproportion may appear ,Then the following could be evident:**

- **Fetal caput/ extensive molding/ palpable overlapping sutures.**
- **-If no cervical changes occur or no station changes occur within 2 hours in the setting of adequate uterine contractions (Active phase arrest)common indication for C-section**

☒ ***The Second Stage of Labor:***

- **Begins after the cervix is completely dilated (10cm) to the delivery of the newborn.**
- **The second stage generally takes from 30 minutes to 3 hours in primigravid women and from 5 to 30 minutes in multigravida.**
- **Uterine contractions and the mother's desire to bear down with every contraction aid in the decent of the fetus that should be monitored regularly Six movements of the baby enable it to adapt to the maternal pelvis**

➢ These movements are described below.

1. Descent: Completed by the force of uterine contractions and bearing down.

2. Flexion: During descent, resistance from the cervix and pelvic walls and floor causes the fetal cervical spine to be further flexed

3. Internal rotation.

4. Extension:."Crowning" occurs when the largest diameter of fetal head is encircled by the vulvar ring. At this time, the vertex has reached station +5 and an episiotomy may aid in reducing perineal resistance.

5. External rotation: Returns the head to its original position at the time of engagement to allow the head to align itself with the fetal back and shoulders.

6. Expulsion: Delivery of the anterior shoulder (under symphysis pubis), followed by the posterior shoulder (over the perineal body), and finally the body of the newborn.

➢ ***The Third stage of labor***

- **Starts immediately after the baby's delivery. The cervix and vagina should be inspected thoroughly for lacerations and surgical repair should be done when necessary.**
- **The *delivery of the placenta* is the main part of the third stage of labor.**

➢ **Separation of the placenta usually occurs *within 2-10 minutes. Signs* of placental separation include:**

1. A fresh show of blood from the vagina.
2. Umbilical cord lengthening outside the vagina.
3. Rising of the fundus of the uterus.
4. The uterus becomes firm and globular.

- **Only after these signs have appeared, gentle cord traction and counter pressure between the fundus and symphysis are applied.**
- **Finally, the placenta should be examined to ensure its complete removal (no missing cotyledons) and to detect placental abnormalities**

➢ ***The Fourth Stage of labor***

- **Extends up to 4-6 hours after the delivery of the placentas and stabilization of the mother**
- **Continuous close monitoring of the patients' vital signs and uterine blood loss is essential to prevent postpartum hemorrhage.**
  Most cases of serious postpartum hemorrhage occur during this stage

☒ Shoulder dystocia

- **Shoulder dystocia occurs when either the *anterior,* or less commonly the posterior fetal shoulder impacts on the maternal symphysis, or sacral promontory**

➢ Risk factors:

1. Fetal macrosomia
2. Maternal diabetes.
3. obesity

4. multiparty
5. post term gestation
6. short stature
7. previous history of macrosomic birth
8. Previous history of shoulder dystocia.
9. labor induction
10. epidural analgesia
11. prolonged labor
12. operative vaginal delivery

➢ Complications:

- The major neonatal complication of shoulder dystocia is Erb palsy: caused by excessive traction on the brachial plexus by the delivery attendant
- Klumpke's paralysis is a variety of partial palsy of the lower roots of the brachial plexus
- clavicular fracture, humeral fracture
- hypoxia, brain injury, and death

❖ Management:

- Shoulder dystocia is recognized at delivery by retraction of the fetal head, which is called the "turtle sign."
- Shoulder dystocia is not overcome by traction on the fetal head but, instead, by one or more maneuvers designed to displace the anterior shoulder from behind the symphysis pubis.
- An initial maneuver that can be attempted is Supra-pubic pressure, which involves downward or lateral pressure with the hand over the maternal suprapubic region in an effort to guide the anterior shoulder under or away from the symphysis pubis.
- Other options: (McRoberts maneuver, Gaskin" or "all-fours", Wood maneuver).
- If none of these maneuvers is successful, one or both clavicles must be fractured, preferably by pressure on the clavicle directed away from the pleural cavity to prevent traumatic puncture of the lungs.

Last-resort procedure, when all previous methods failed is Zavanelli maneuver: the fetal head is manually returned to its pre-situation position, and then slowly replaced into the vagina and then into the uterus by steady upward pressure against the head. Delivery is then accomplished by cesarean delivery. A uterine relaxant may be required to carry out this procedure

❑ Puerperium

✓ **It's the period of time in which organ systems return to their pre-pregnant state (6 weeks postpartum).**

✓ **Normal puerperium (physiological changes)**

1. Reproductive System

• Uterus

- Involution occurs 10-12 days postpartum

- Uterine contractions are present (to keep venous placental sinuses closed)

• Lochia

- Superficial layers of the endometrial decidua that are shed through the vagina during the 1st 3 postpartum weeks:

•1st few days : Red Lochia (Rubra)

•up to 2nd week : Pink Lochia (Serosa)

•after 2 weeks : White Lochia (Alba)

- **Vagina : NEVER return to prepartum size**

2. Breast

• Colostrum production starts in the latter part of pregnancy until 3 days postpartum

• Milk production starts on the 3rd-4th day postpartum (under the effect of prolactin)

3. CVS

- **Risk of VTE is higher postpartum**

❑ Postpartum Complications (Abnormal Puerperium)

1. Breast disorders

- **Engorgement**

- Over-distention of the breast due to milk collection

- 3rd day postpartum

- Physical exam: hard and tender with nodules of enlarged breast tissue; enlarged breast covered by dilated veins.

- Treatment: breast care (cleaning); frequent emptying by suction (manual/pump) (Not indication to stop breastfeeding)

- **Puerperal mastitis**

- Source of infection: the baby's mouth flora has Staphylococcus aureus

- Signs and symptoms: fever, tachycardia, pain and tenderness with localized areas of hotness and erythema

- Complications: breast abscess

- Treatment: continued breast feeding should be encouraged; analgesia, antipyretics, and broad spectrum antibiotics; breast care.

2. Postpartum fever : postpartum pyrexia

- **Causes are**

✓ **Atelectasis : day 1**
✓ **UTI : day 2-3**
✓ **Endometritis : day 3-4**
✓ **wound infection : day 5-7**
✓ **superficial thrombophlebitis**
✓ **breast disorders (abscess/mastitis)**

❑ Postpartum Hemorrhage :(PPH)

✓ **Blood loss >500 cc in vaginal delivery and >1000 cc in cesarean section**

✓ **Primary (early): within 24 hours of delivery**

- The most common cause is uterine atony

- 2nd most common cause is infection

✓ **Secondary (late): after 24 hours of delivery**

- Most common cause is infection

✓ **It is the most common cause of death in developing countries**

✓ **Causes of primary PPH are :**

1. Tone: Uterine atony

• The most common cause (50%)

• Risk factors:

- Overworked uterus:

Rapid labor and increased oxytocin/ Prolonged labor

- Overdistended uterus:

Multiple pregnancy/Polyhydramnios/Macrosomia

- Infected uterus: chorioamnionitis

- Relaxed uterus (drugs):

MgSO4/ B-adrenergic agonists/Halothane

• Clinical presentation: doughy uterus, above umbilicus

• Management:

- Uterine massage (1st step)

- Uterotonics: Oxytocin, Methergine, PG F2α (Hemabate)

- B-lynch suture can be done if failed massage and drugs

2. Trauma: Genital laceration

• Risk factors: Traumatic vaginal delivery /Operative vaginal delivery

• Clinical presentation: lacerations and contracted uterus (rule out atony)

• Management: surgical repair (suturing)

3. Tissue: Retained products of conception (POC)

• Risk factors: Accessory placental lobe /Placenta accrete

• Physical examination: missing cotyledons and contracted uterus

• Management: manual removal of placenta or curettage under ultrasound guidance

4. Thrombosis: Obstetric DIC

• Risk factors:

- Abruptio placenta (common)

- Severe PET

- Amniotic fluid embolus

- Fetal demise

• Physical examination: Generalized oozing and Petechiae

• Management: Remove POC, ICU, blood products

5. Turned over: Uterine inversion

- **Risk factors: myometrial weakness (most common), previous history of inversion, fundal placenta, placenta accrete, too much traction of the cord & fundal pressure.**

- **Physical examination: beefy bleeding mass (fundus coming through the vagina), uterus not palpable**
- **Management: replace the uterus into its normal place (by elevating the vaginal fornices) then give IV oxytocin.**

6. Unexplained hemorrhage: Need Ligation of the uterine vessels /internal iliac or hysterectomy.

❑ Episiotomy

- ✓ **Incision in the perineum (skin, vagina, perineal muscles) to increase space**
- ✓ **available for delivery during second stage of labor.**
- ✓ **Indications:**
- **Suspected maternal and/or fetal compromise**
- **Delivery needs to be expedited**
- **Suspected fetal compromise**
- **Shoulder dystocia to aid with performing internal maneuvers**
- **Anticipation of significant perineal and or rectal trauma**
- ✓ **Types:**

A) Midline: from fourchette down the perineal midline raphe towards the anal verge..

- **Disadvantages: risk of anal sphincter & anal muscles injury (incontinence).**
- **Advantages: less bleeding, better healing , less pain**

B) Mediolateral

Incision at a 45 angle inferiorly from midline of the fourchette

- **Disadvantages: more bleeding and pain, less healing**
- **Advantages: no risk of anal sphincter injury**

Amniotic Fluid:

- **It is the fluid surrounding the growing fetus in utero. It's produced at first (before 14weeks gestation) mainly from maternal serum, while fetal kidneys start excreting it beyond 14 weeks.**
- **The amniotic fluid compartment allows**

1-The fetus room for growth , movement and development. Without amniotic fluid, the uterus would contract and compress the fetus. In cases of leakage of amniotic fluid early in the first trimester, the fetus may develop structural abnormalities including facial distortion, limb reduction, and abdominal wall defects secondary to uterine compression

2-important for fetal pulmonary development.. The absence of adequate amniotic fluid during mid-pregnancy is associated with pulmonary hypoplasia at birth, which is often incompatible with life.

3- The amniotic fluid also has a protective role for the fetus. It contains antibacterial activity and acts to inhibit the growth of potentially pathogenic bacteria.

4- During labor and delivery, the amniotic fluid continues to serve as a protective medium for the fetus, aiding dilation of the cervix

☒ The volume of AF is assessed antenatally:

- **measurements of AF present in all 4 quadrants is referred to as AF index (AFI.) AFI values ranges normally from 5-23, depending on the gestational age at measurements.**
- **single deepest pocket of AF measurement between 2 and 8.**

☒ Oligohydramnios

- **is defined by an AFI < 5. It can be a result of many conditions**
- **causes are:**

✓ **mother (Dehydration),**

- ✓ **the fetus (Renal anomalies, Potter syndrome (bilateral renal agenesis) Chromosomal Trisomy),**
- ✓ **the placenta or other factors (Placental insufficiency, Post-term pregnancy, Medications like ACEIs and Indomethacin, PROM.)**

☒The most common cause is ROM

- **On physical examination, the size of the uterus is lower than the expected to it's GA, and Fetal movements may be reduced.**
- **Complications: (usually if occurred <24 weeks)**

✓ **Malpresentation**

✓ **fetal asphyxia and hypoplastic lung**

✓ **Intrauterine Growth Restriction (IUGR)**

✓ **Fetal anomalies (CHD, Potter face) and Contractures (arthrogryposis**

✓ **Management of oligohydramnios largely depends on the gestational age (GA) and the degree of severity..**

- **If<24wk and severe : termination**
- **If> 24wks: Mild: follow up for signs of chorioamnionitis, AFI, fetal movement Severe: hydration, dexamethasone, amnioinfusion (transcervical intrauterine catheter)**
- **In severe cases of oligohydramnios especially when cardiac tracing of the fetus shows cord compressions, Amnioinfusion may be considered; a process through which AF is replaced with normal saline through transcervical Intrauterine pressure catheter**

☒ Polyhydramions

- **Amniotic fluid index >23 OR Largest single pocket >8**
- **Classification:**

- ✓ **Amniotic Fluid Index :Mild 25-30/Moderate 30-40/ Severe >40**
- ✓ **Largest single pocket :Mild 8-12/ Moderate 12-15/ Severe >15**

- **<u>Causes</u> of polyhydramnios:**

- ✓ **Idiopathic (most common cause)**
- ✓ **Maternal: DM, cardiovascular diseases, infections (CMV, toxoplasmosis)**
- ✓ **Fetal: multiple gestations, GI obstruction, CNS (anencephaly) –because baby can't swallow and there's no ADH secretion in this case- , CVS (hydrops fetalis), Trisomy Placenta: chorioangioma**

- **<u>Symptoms:</u>**

-abdominal distention and discomfort

-Indigestion/ vomiting

-Shortness of breath (diaphragm)

-Surrounding veins (varicose veins, hemorrhoids, lower limbs edema

- **<u>Signs:</u>**

-Distended abdomen (large for gestational age)

-Shiny skin /Stria

-Faint fetal heart sound and difficulty feeling fetal movement

- **Complications**

- ✓ **Antepartum: Preeclampsia (PET), placental abruption, <u>preterm labor (most important),</u> PROM**
- ✓ **Intrapartum: poor presentation, poor uterine contractions, prolapse of the cord, supine hypotension syndrome**
- ✓ **Postpartum: Postpartum hemorrhage (caused by uterine atony)**
- ✓ **Fetal complications: prematurity, perinatal morbidity and mortality, cord prolapse**

• Management

- ✓ **For mild to moderate cases, outpatient management to the cause (if identified) with regular follow ultrasound assessment done every 2 weeks would be appropriate.**
- ✓ **For severe cases, patients should be admitted to ensure bed rest. Indomethacin or/and AF reduction is considered, beside further investigations (including OGTT, Ig titers, Rh status) to identify the cause, and fetal assessment for any anomaly**

☒Small for gestational age

- ✓ **SGA is when Estimated Fetal Weight (EFW) <10th**
- ✓ **Majority are healthy/Familial in 85% of case**
- ✓ **SGA is classified into: Normal (no structural abnormalities, normal liquor and normal UAD) vs Abnormal (IUGR)**
- ✓ **IUGR classified into:**
  - **Symmetrical (due to fetal causes) –decreased POTENTIAL Both head and body are equally affected**
  - **Asymmetrical: M.C/ (due to maternal/placental causes) –decreased SUBSTRATES the body (abdominal circumference) is small while head (head circumference) is preserved**

☒ **Complications of IUGR :**

- ✓ **Respiratory: meconium aspiration, asphyxia (PROTECTED AGAINST RDS)**
- ✓ **Electrolytes/ blood: Polycythemia, increased Bilirubin, decreased Calcium, decreased Glucose**
- ✓ **Life manifestations : mental retardation, fetal distress, Intrauterine fetal death**

  - **Causes of IUGR:**

- ✓ **Fetal : Trisomies, Turner, infections (TORCH, syphilis, malaria, varicella), polyhydramnios, oligohydramnios, uterine overcrowding (due to fibroids or multiple gestations) /here the baby has decreased potential to grow, increase feeding will not help**

- ✓ **Maternal :HTN, preeclampsia, DM, CVD, Respiratory, Renal, SLE, Sickle cell anemia poor, malnutrition (low BMI), infections, smoking, alcohol Drug history: Chemotherapy, radiotherapy, teratogenicity drugs (antiepileptic, Beta-blockers**

- ✓ **Placental :placenta previa, placental abruption, thrombosis, calcifications, TTTs (twin-twin transfusion syndrome)**

- **Investigations: SERIAL U/S (the most effective way in detecting growth restriction) (Biometry, NST, BPP, umbilical Doppler, MCA Doppler)**

- **Management:**
  - **-Lifestyle changes (nutrition, rest, stop smoking)**
  - **-Baby Aspirin (for prevention)**
  - **-Fetal monitoring (weekly) NST, Biometry, BPP)**
  - **Biometric measurement :BPD (biparietal diameter) /HC (head circumference) / AC (abdominal circumference) /FL(femoral length)**

☒ Large for Gestational Age (LGA)

- ✓ **When EFW< 90-95th percentile for a given GA**
- ✓ **Risk factors:**
- **-Gestational DM, overt DM**
- **-prolonged gestation (postdate pregnancy)**
- **-increased BMI (obesity) , increased weight gain during pregnancy**
- **-Multiparity /Male fetus**

✓ **Complications:**

☒ **Maternal: -operative vaginal delivery -perineal lacerations, pelvic floor injury leading to urinary incontinence and pelvic organ prolapse - emergency C/S -Postpartum hemorrhage (due to atony)**

☒ **Fetal: -shoulder dystocia -asphyxia -birth injury**

☒ **Neonatal: NICU admission/Erbs palsy /hypoglycemia**

✓ **Management :**

☒ **Elective C/S if EFW >4.5kg in diabetic mother or >5kg in non-diabetics**

☒ **Early induction (increased failed induction)**

❑ Rh Isoimmunization

✓ **The Rh complex on the surface of the red blood cell is made up of a number of antigens, including C, D, E, c, d, e, and other variants, such as partial D antigens.**

✓ **Rh(D)antigen is expressed by 30 days of gestation. More than 90% of cases of Rh alloimmunization are due to antibodies to the D antigen, and this is the only form of alloimmunization that can be prevented with Rh immune globulin prophylaxis.**

✓ **A person who lacks the D antigen on the surface of the red blood cells is regarded as being-negative," and an individual with the D antigen is considered to be "RhD-positive."**

✓ **When RhD-negative patients are exposed to the RhD antigen, they may become sensitized (start to recognize D antigen as immunogenic and want to destroy it).**

✓ **In general, two exposures to the RhD antigen are required to produce any significant sensitization, unless the first exposure is massive. The first exposure leads to primary sensitization(the production of immunoglobulin M (IgM) antibodies (which cannot cross the placenta) for a short period of time)**

✓ **The second causes rapid production of immunoglobulins (IgG antibodies) that are capable of crossing the placenta.**

✓ **If the fetus has the RhD antigen, these antibodies will coat the fetal red blood cells, causing them to be destroyed, or hemolyzed, in the spleen**

✓ Causes of sensitization :

- **Most cases of sensitization are caused by a placental leak of fetal red blood cells into the maternal circulation (fetomaternal hemorrhage) during pregnancy. With advancing gestational age, the incidence and size of these transplacental (fetomaternal) hemorrhages increase, with the largest hemorrhages usually occurring at delivery.**
- **Most immunizations occur at the time of delivery, and antibodies appear either during the postpartum period or following exposure to the antigen in the next pregnancy.**
- **Antepartum event that may cause so:**

✓ **- Invasive prenatal diagnostic or therapeutic procedures**
✓ **- Blunt abdominal trauma**
✓ **-external cephalic version**
✓ **- Miscarriage or induced abortion.**
✓ **- Ectopic pregnancy**
✓ **- Fetal death in the second or third trimester**
✓ **- Antepartum hemorrhage in the second or third trimester**
✓ **- Hydatidiform mole only if partial, as a complete mole does not contain fetal red cells..**

➢ **Consequences of the destruction of the fetal red blood cells:**

✓ **If the hemolysis is mild, the fetus can compensate by increasing the rate of erythropoiesis.**
✓ **If the hemolysis is severe, it can lead to profound fetal anemia, resulting in extramedullary hematopoiesis, portal hypertension, hypoalbuminemia, hyperbilirubinemia, and heart failure(hydrops fetalis), as well as intrauterine fetal death. High bilirubin levels can damage the central nervous system and lead to neonatal encephalopathy and kernicterus (deposition in basal ganglia).**

✓ **The disease gets worse in subsequent pregnancies in Rh-D antigen alloimmunization**

❖ **Approach:**

- **All D-negative pregnant women should undergo an antibody screen at the first prenatal visit of each pregnancy. In RhD-negative patients whose anti-D antibody titers are positive (i.e., those who are RhD-sensitized), the RhD status of the father of the baby should be determined. If the father was RhD negative, we won't worry about the baby as it's going to be RhD negative as well.**
- **The diagnosis of Rh(D) alloimmunization is based upon detection of anti-Rh(D) antibody in maternal serum. In patients with a positive titer less than 1:16, repeat titers should be obtained every 2 to 4 weeks. If the titer rises to 1:16 or greater, a detailed ultrasound to detect hydrops and Doppler studies of the MCA are indicated.**

❑ **TO prevent alloimmunization we treat mother with anti-D Ig (RhoGAM) during and after each pregnancy to prevent anti-D IgG formation. So we give her the Anti-D antibody before her body forms it. What's the point? When a mother is given a dose of anti-D IgG, the antibodies bind to the fetal RBCs that have the D antigen on them and clear them from the maternal circulation.**

- **The goal is to prevent the mother's immune system from recognizing the presence of the D antigen forming antibodies against it.**
- **The routine prophylactic administration of Rh immune globulin at 28 weeks' gestation is now the standard of care.**
  RhoGAM is not indicated for patients who already have anti-D antibodies and are sensitized. It prevents but does NOT treat alloimmunization

❑ **Testing for fetomaternal hemorrhage**

- Routinely testing all D-negative women for excessive fetomaternal bleeding at the time of delivery to ensure that they receive an adequate dose of anti-D immunoglobulin.
- At delivery is at the limit of effective prophylaxis from a single 300 microgram dose of anti-D immunoglobulin.
- Rosette test; a qualitative, yet sensitive test for fetomaternal hemorrhage/The rosette test is a screening test for FMH that detects fetal D+ red cells in maternal Rh negative blood. If the rosette test is positive, follow-up testing is done to quantitate : Kleihauer-Betke test or flow cytometry : Quantitative

❑ No need for prophylaxis if

- ✓ The biologic father of the baby is known with certainty to be D-negative
- ✓ Cell-free DNA (cfDNA) results on maternal plasma suggest that the fetus is D-negative.
- ✓ Fetal blood sampling confirmed the fetus to be Rh –ve.
- ✓ Amniocentesis/CVS confirmed the fetus to be Rh –ve.
- ✓ Complete mole

❑ Management:

- ✓ Serial Doppler assessments of peak systolic velocity in the fetal MCA have proven to be the most valuable tools for detecting fetal anemia..
- ✓ Amniotic fluid bilirubin levels (obsolete)
- ✓ - Fetal blood sampling (not routine)
- ✓ - Severe fetal anemia near term is treated by delivery for neonatal treatment;
- ✓ - Intrauterine fetal transfusions are performed. – Intravascular/-Intraperitoneal
- ✓ - Serial combined maternal plasmapheresis

- Intravenous immune globulin therapy is a promising approach for decreasing the severity of fetal disease, but is investigational

☒Multiple pregnancy

- It's defined as the development of more than one fetus inside the uterus.
- Number of twin pregnancies increased due to increased use of assisted reproduction techniques

Types:

- Note: dizygotic twins have identifiable CAUSES while monozygotic twins have NO identifiable CAUSES

1. Monozygotic (identical= SAME sex and genetic):1/3
   - ✓ 30% - Dichorionic diamniotic DCDA (2 placenta, 2 amniotic sacs) / (0-3)DAYS
   - ✓ 69% -Monochorionic diamniotic MCDA (1 placenta, 2 amniotic sacs) /(4-8)DAYS
   - ✓ -Monochorionic monoamniotic MCMA (1 placenta, 1 amniotic sac /(9-12)DAYS
   - ✓ Conjoined twins (AFTER DAY 12)

2. Dizygotic :more common than monozygotic (2/3)

- ALWAYS dichorionic diamniotic (DCDA) /May or may not be the same sex./ different genetics

**Risk factors /causes are:

1-Race (more common in blacks).

2-Age and parity (increases with age and parity).

3-Family history.

4-Ovulation induction and conception after cessation of contraception

- **Symptoms: exaggerated pregnancy symptoms, increased fetal activity/Uterus size more than date of pregnancy,**
- **Lab findings: 1. Increased B-hCG 2. Increased MSAFP**
- **Determination of zygosity is the most important next step after multifetal pregnancy has been first diagnosed, and this can be helpful by ultrasound which can be made as early as 6 weeks' gestation.**
- **U/S findings: T- sign seen in monochorionic/ lambda (λ) sign) seen in Dichorionic**
  - **-A thick amnion-chorionic septum is suggestive of dizygotic twins**
  - **-A thin dividing membrane is suggestive of monochorionic twins.**
- **Definitive diagnosis of zygosity may require detailed examination of the placenta after delivery.**
- **Complications:**

- **Maternal: 1. Hyperemesis Gravidarum 2. Nutritional Anemia (iron and folate deficiency)**

  3. PET / Gestational DM 4. Preterm labor/ cervical incompetence and increased risk of abortion

  5. Increased C/S (MCMA is a relative indication for C/S because of risk of cord prolapse /95% of twins are delivered by C/S)

  6. Postpartum hemorrhage/ Antepartum hemorrhage

Fetal complications:

- **1-Malpresentation. 2- Placenta previa/ Abruptio placentae. 3-PROM/Prematurity/SGA.**
- **4-poly hydramnios. 5-Umbilical cord prolapse. 6- Intrauterine growth restriction.**
- **7-Congenital anomalies. 8-Increased perinatal morbidity and mortality. 9-TTTS-only in monochorionic.**

❑ Management:

☒ **Antepartum:**

- **First and second trimesters:- Between 16 and 22 weeks, the patient is seen every 2 weeks for ultrasonic cervical length assessment. /-Increase the supplements (Iron, Folate, vitamins).**
- **Third trimester:-Prevention of prematurity; bed rest, serial uterine activity monitoring, hospitalization./-The patient should be monitored closely for signs of preeclampsia.**

☒ **Intrapartum: -NVD if both cephalic. /Delivery of 1st twin ---> avoid giving oxytocin until after second baby.**

- ❑ **Indications for C/S: >2 fetuses / malpresentation of the first twin / cephalopelvic disproportion or conjoined twins.**
- ❑ **MCMA twins is a relative indication for C/S (34-36 weeks) because of risk of cord prolapse.**

❑ **Average of GA: 1. Twins :35 weeks 2. Triplets : 32-33 weeks 3. Quadruplets : 28-29 weeks**

☒ **Postpartum:-Watch for PPH (risk of atony), active management of third stage**

❖ **Complications are More common with monozygotic twins, and they include:**

1-Cojnjoined twins.

2-Interplacental vascular anastomosis.

3-Twin-twin transfusion syndrome (TTTS).

4-Fetal malformations; Twin reverse arterial perfusion sequence (TRAP).

5-Umbilical cord abnormalities.

6-Retained fetus syndrome

❑ Hypertensive disorders of pregnancy

☒ **Hypertensive disorders of pregnancy include:**

- **Preeclampsia/eclampsia**
- **Chronic hypertension**
- **Chronic hypertension with superimposed preeclampsia**
- **Gestational hypertension.**

☒ Preeclampsia

✓ **It is a multisystem disorder . Characterized by high blood pressure and signs of damage to other organ systems after the 20th week of gestation.**

✓ **Because of the resolution of preeclampsia after delivery, inadequate uteroplacental perfusion leading to placental ischemia or hypoxia seem to be central to the development of the disease.**

✓ **HELLP syndrome: a variant of severe preeclampsia with particularly high morbidity. Occurs in women with preeclampsia with evidence of Hemolysis, Elevated Liver enzymes, Low Platelets (thrombocytopenia). More likely to be multiparous, >25 years old and less than 36 weeks of gestation.**

✓ Risk factors:

a) Primigravida
b) Extremes of age
c) Methamphetamine use
d) History of preeclampsia (family or personal)
e) Chronic hypertension
f) Obesity/DM/Thrombophilia
g) Multiple gestation/polyhydramnios/macrosomia
h) gestational trophoblastic disease

✓ Clinical manifestations

- **Weight gain and edema**
- **Oliguria/frothy urine(proteinuria).**
- **Thrombocytopenia**
- **Right upper quadrant pain/ epigastric pain/elevated live enzymes**
- **due to vasospasm resulting in focal hemorrhages and infarctions.**
- **IUGR/Oligohydramnios/fetal heart rate abnormalities**

✓ Assessment:

- **History and physical exam.**
- **Fetal evaluation**
  Gestational age, U/S to evaluate growth, amniotic fluid index, Nonstress test to determine if there is evidence of fetal compromise.
- **Initial labs for evaluating a patient with preeclampsia:**
  CBC, platelet count, LDH (if abnormal order a D-dimers, coagulation panel and smear) /renal studies: serum bun creatinine and uric acid, urinalysis, 24-hour urine collection or protein/creatinine ratio.
  LFTs.

✓ Complications

☒ **Maternal : seizures, cerebral hemorrhage, stroke/HF, renal failure/ HELPP/pulmonary edema/death (most likely from CNS hemorrhage)**

☒ **Fetal: Preterm/infection/abruption/IUGR/Oligohydramnios/death**

✓ Management

- **Delivery is the only definitive cure for preeclampsia.**
- **ADMIT THE PATIENT : Fetal and maternal monitoring**
- **Give the mother antihypertensive / MgSo4 in case of severe PET**

- **If PET is mild with no evidence of fetal compromise:**
  <36 weeks – expectant management vs >37 weeks – deliver
- **If severe:**
- **>34 weeks – delivery after stabilization**
- **<34 weeks – may be stabilized and with careful monitoring of the mother and fetus, delivery delayed until the pregnancy has reached 34 weeks.**
- **Mode of delivery should be vaginal unless there is an indication for C/S**

✓ **Magnesium sulfate: seizure prophylaxis for patients with severe preeclampsia . Serial assessments of urine output, deep tendon**

reflexes, and respirations are important for detecting signs of magnesium toxicity.

- ✓ **The safest drugs for control of acute control of severe hypertension are labetalol and hydralazine.**

☒ **Gestational hypertension**

Hypertension without proteinuria or other signs of organ dysfunction appearing after 20 weeks of gestation. The diagnosis can only be made in retrospect.

☒Diabetes mellitus in pregnancy

- ✓ **Gestational diabetes mellitus (GDM): glucose intolerance with onset after 20 weeks of pregnancy.**
- ✓ **Pre-gestational DM: diabetes present before pregnancy and may be either type 1 or 2**
- ✓ **Diabetogenic hormones are :**
- **HPL (which is secreted from the placenta in the 2nd half of pregnancy and explains the development of DM at the same time period)**
- **Placental insulinase**
- **Cortisol**
- **Progesterone**
- **Prolactin**

✓ Risk factors

1. Age (<25 years old)
2. Obesity
3. Family history of GDM or DM
4. PCOS
5. Recurrent infections

6. HTN/PET

7. Previous history of GDM/macrosomia/polyhydramnios/

8. History of unexplained fetal death/neonatal death/congenital anomalies/IUGR

9. Current pregnancy: polyhydramnios/macrosomia.

✓ Complications

- **The majority of complications are due to hyperglycemia. Glucose crosses the placenta easily by facilitated diffusion, causing fetal hyperglycemia and eventually fetal hyperinsulinemia.**
- **Pregestational DM is generally associated with a higher rate of maternal and fetal complications**
- **Maternal complications :**

  1. Difficulty in controlling diabetes due to:

  2. Recurrent hospitalization

  3. Increased risk of abortion

  4. Increased risk of preterm labor and PROM

  5. Increased risk of traumatic delivery

  6. Increased risk of developing DM (type 2), metabolic syndrome, obesity and CVS disease after disease.

  7. Increased risk of vascular or end organ involvement or deterioration.
- **Fetal complication**

  1. Congenital anomalies: this only occurs in pregestational DM.
  - ✓ **Most common: cardiac anomalies.**
  - ✓ **Most specific and pathognomonic for DM: Caudal regression.**

  2. Macrosomia and traumatic delivery

  3. Neonatal hypoglycemia, hypocalcemia, hypomagnesemia.

  4. IUGR

  6. Intrauterine fetal death

  7. Increased risk to develop DM, HTN, CVS disease.

☒ **JU Protocol for DX:**

- Perform a FBS test for all patients at booking

- FBS >126: preexisting DM

FBS (92-126): 75g OGTT immediately

FBS <92 – 75g OGTT at 24-28 weeks.

☒ **Management**

1. Control glucose levels 3 months prior to conception.

2. Diet and exercise are important.

3. Pharmacological therapy: Insulin is the mediation of choice.

4. Antepartum care :

- Ophthalmology, cardiac, endocrine and nephrology consults are recommended for the mother.

5. Timing of delivery:

✓ **In general, well-controlled GDM without complications, we can wait for spontaneous delivery.**

✓ **Earlier intervention is indicated if complications arise or GDM is no longer controlled.**

✓ **If the fetus weighs more than 4500 g, C-section decrease the risk of a traumatic delivery.**

✓ **If there isn't an indication for C-section, normal vaginal delivery**