Obstetrics:

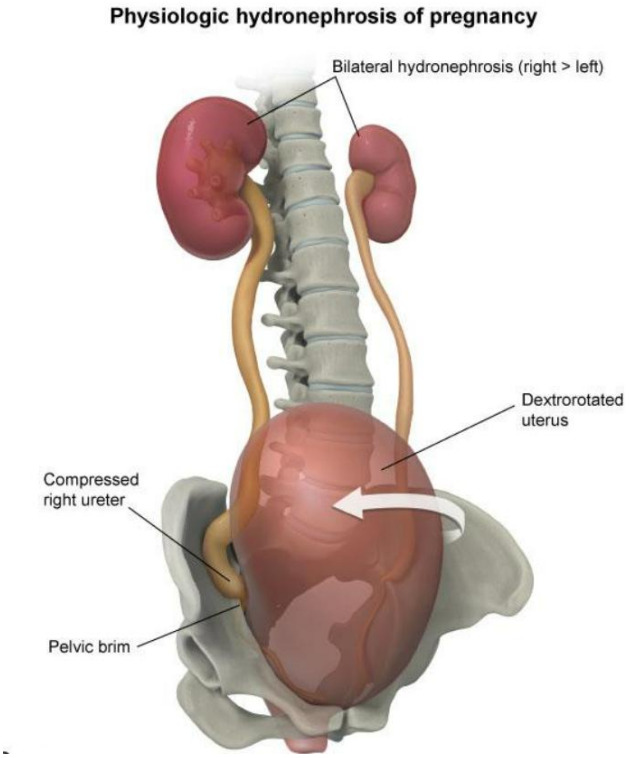
Physiologic changes in pregnancy:

Normal physiological changes during pregnancy		
System	**Clinical finding**	**Mechanism**
Renal/Urinary	↑ Glomerular filtration rate & renal size, ↓ blood urea nitrogen & serum creatinine	↑ Cardiac output & renal blood flow due to progesterone, with ↑ renal excretion
	Urinary frequency, nocturia	↑ Urine output & sodium excretion
	Mild hyponatremia	Hormones reset threshold to ↑ ADH release from pituitary
Heme	Dilutional anemia	↑ Plasma volume & red blood cell mass
	Prothrombotic state	Hormone-mediated ↓ in total protein S antigen & activity; ↑ in fibrinogen & coagulation factors
Cardiovascular	↑ Cardiac output & heart rate	↑ Blood volume, ↓ systemic vascular resistance
Pulmonary	Chronic respiratory alkalosis with metabolic compensation, ↑ PaO_2 & ↓ $PaCO_2$	Progesterone directly stimulates central respiratory centers to ↑ tidal volume & minute ventilation

- *Fall in **sodium concentration** during pregnancy closely correlates with i**nc. production of hCG***

  ***Fall <u>below 130</u>** <u>should prompt evaluation for pathologic causes of hyponatremia</u>*

Physiologic Hydronephrosis of Pregnancy:



During pregnancy, kidney enlargement occurs because there is an increase in maternal blood volume that requires increased renal filtration, resulting in greater renal vasculature and interstitial tissue. Hydronephrosis begins during the first trimester as high progesterone levels cause ureteral dilation and decreased peristalsis. Hydronephrosis becomes more pronounced in the second and third trimesters as uterine enlargement compresses the ureters at the pelvic brim, which results in **dilation of the proximal ureters** and **bilateral hydronephrosis**. Right hydronephrosis is often more pronounced due to dextrorotation of the uterus, which causes increased compression of the right ureter. Unlike pathologic hydronephrosis (eg, secondary to obstruction or infection), physiologic hydronephrosis of pregnancy requires **no additional management**.

Obstruction due to stones or other things usually presents with microscopic hematuria and unilateral imaging findings (eg, visible stone, unilateral hydronephrosis).

- **Renal pelvis** and **ureter** dilation during pregnancy occurs more commonly on the **right** side *due to **cushioning** provided by the **sigmoid colon** on the **left***

Exercise:

What exercise regimen is recommended for healthy women with uncomplicated pregnancies? 20 - 30 minutes of moderate-intensity exercise on most or all days of the week

Pregnancy & exercise	
Absolute contraindications	Amniotic fluid leak Cervical insufficiency Multiple gestation Placenta abruption or previa Premature labor Preeclampsia/gestational hypertension Severe heart or lung disease
Unsafe activities	Contact sports (eg, basketball, ice hockey, soccer) High fall risk (eg, downhill skiing, gymnastics, horseback riding) Scuba diving Hot yoga

Exercise during pregnancy can be beneficial and is associated with decreased risks of excessive gestational weight gain, gestational diabetes mellitus, preeclampsia, and cesarean delivery. Therefore, low-risk patients who exercised regularly prior to pregnancy are encouraged to continue moderate-intensity exercise during pregnancy.

Exercise during pregnancy	
Goal	• 30 minutes of moderately intense exercise most days of the week
Benefits	• ↓ Gestational diabetes mellitus risk • ↓ Preeclampsia risk • ↓ Cesarean delivery risk • Shorter postpartum recovery • Weight management
Recommended	• Walking/running • Cycling • Yoga • Swimming • Light-weight strength training

However, exercise during pregnancy may exacerbate some obstetric conditions; therefore, it is **contraindicated** in certain patients. Exercise should be avoided by:

- Patients at **high risk for preterm birth**
    - **Cervical insufficiency** or cerclage
    - Preterm labor during current pregnancy
    - Preterm prelabor rupture of membranes
- Patients at risk for **antepartum bleeding**
    - Placenta previa
    - Persistent second- or third-trimester bleeding
- Patients with an **underlying condition** that could be **exacerbated by exercise**
    - Severe anemia (eg, hemoglobin <8 g/L)
    - Hypertensive disorders of pregnancy (eg, preeclampsia)
    - Restrictive lung disease
    - Hemodynamically significant heart disease.

Pregnant patients should avoid exercises that increase the risk of abdominal trauma and maternal injury (eg, contact sports, downhill skiing). Hot yoga is also not recommended due to the risks of hyperthermia (eg, fetal neural tube defects). Most other types of exercise, including running and moderate weight lifting, may be continued in pregnancy.

Weight gain in pregnancy		
Prepregnancy BMI (kg/m^2)	**Ideal weight gain**	**Complications**
<18.5	12.7-18.1 kg (28-40 lb)	Inadequate weight gain Low birth weight Preterm delivery Excessive weight gain Gestational diabetes mellitus Fetal macrosomia Cesarean delivery
18.5-24.9	11.3-15.9 kg (25-35 lb)	
25-29.9	6.8-11.3 kg (15-25 lb)	
≥30	5-9 kg (11-20 lb)	

During pregnancy, women have increased nutritional requirements because both calories and nutrients (eg, folate, calcium, iron) are preferentially shunted to the developing fetus. To meet these metabolic demands, average women should increase their intake by approximately 350-450 kcal/day in the second and third trimesters.

The target goals for gestational weight gain depend on prepregnancy BMI, which is an indicator of baseline maternal fat and nutrient stores. Patients with an **underweight prepregnancy BMI** (ie, <18.5 kg/m^2) have low baseline stores and therefore require greater weight gain (~1 lb [0.5 kg]/week) to maintain a healthy pregnancy.

Complications of inappropriate pregnancy weight gain	
Excessive weight gain	Gestational diabetes mellitus Fetal macrosomia Cesarean delivery
Inadequate weight gain	Fetal growth restriction Preterm delivery

Vomiting:

• **Nausea and vomiting during pregnancy** affects many women in the first trimester and is relatively mild with **no** associated weight loss, hypovolemia, or electrolyte abnormalities.

• What is the *pharmacological* **treatment** (*1st and 2nd line*) of nausea and vomiting in pregnancy ("*morning sickness*")?

- *Avoid triggers, consume fluids 30 minutes before or after solid meals*
- **1st line: ginger, pyridoxine (B6)**
- **2nd line**: add **doxylamine**

• Some cases of nausea and vomiting during pregnancy can **mask an underlying eating disorder**, with potential **concerning findings**, including:

- **Weight loss** with an already relatively low BMI
- **Unpredictable appetite** (suggesting dysregulated eating habits) with skipping meals (suggesting possible caloric restriction) followed by episodes of overeating (suggesting possible binge eating)
- **Distorted view of body weight and shape**, evidenced by the perception of "looking pregnant" despite early gestational age (when the uterus is still below the pubic symphysis) and switch to maternity clothing despite weight loss
  - **Evaluate for Disordered Eating behaviour**

Hyperemesis Gravidarum:

Hyperemesis gravidarum	
Risk factors	• Hydatidiform mole • Multifetal gestation • History of hyperemesis gravidarum
Clinical features	• Severe, persistent vomiting • >5% loss of prepregnancy weight • Dehydration • Orthostatic hypotension
Laboratory abnormalities	• Ketonuria • Hypochloremic metabolic alkalosis • Hypokalemia • Hemoconcentration
Treatment	• Admission to hospital • Antiemetics & intravenous fluids

• Patients with HG can develop **prolonged hypoglycemia** due to **inadequate oral intake**; this can lead to **ketoacidosis** and **ketones on urinalysis**.

Ketonuria suggests more **severe disease** and is an indication for **hospital admission**

💡 With appropriate treatment, there are **rarely** any adverse **fetal** effects.

Vaccines:

Vaccines during pregnancy	
Recommended	Tdap Inactivated influenza Rho(D) immunoglobulin
Indicated for high-risk patients	Hepatitis B Hepatitis A Pneumococcus *Haemophilus influenzae* Meningococcus Varicella-zoster immunoglobulin
Contraindicated	HPV MMR Live attenuated influenza Varicella

HPV = human papillomavirus; **MMR** = measles-mumps-rubella; **Tdap** = tetanus toxoid–reduced diphtheria toxoid–acellular pertussis.

Maternal vaccination is beneficial to both mother and fetus, and the optimal time for administration is prior to conception. Vaccinations during pregnancy are indicated if the vaccine has minimal risk, if significant risk exists for infection exposure, and if increased morbidity and mortality are associated with infection. **Vaccines** that are **safe during pregnancy** include **Immunoglobulins** (eg, Rho(D) immunoglobulin), **toxoids** (eg, tetanus toxoid), and **inactivated vaccines** (eg, tetanus toxoid-reduced diphtheria toxoid-acellular pertussis [Tdap], injectable influenza virus). Live-attenuated vaccines (eg, intranasal influenza) are contraindicated during pregnancy due to the theoretical risk of congenital infection.

Pregnant women are at increased risk for morbidity (eg, pneumonia) and mortality from the seasonal influenza virus. **All pregnant women** without contraindications (eg, allergic response to prior vaccination) should receive the influenza vaccination as soon as it becomes available. The **inactivated influenza vaccine** is **safe during every trimester** of pregnancy and while breastfeeding. This vaccine prevents maternal influenza-related illness and provides passive neonatal immunity, thereby decreasing the risk of influenza-related illness in newborns.

- **Tdap:** Given **>26 weeks** (in every pregnancy)

  If there is wound injury, give Tdap regardless of gestational age on the basis of standard wound management guidelines (e.g if not received in last 10 years)

- What is the *recommended management* for a pregnant woman that is **rubella-nonimmune**?

  Immediate *postpartum* vaccination (e.g. MMR)

  contraindicated during pregnancy but safe during breastfeeding

> 💡 The inactivated **influenza** vaccine is **safe during every trimester** of pregnancy and while **breastfeeding.**

The *Haemophilus influenzae* type b vaccine, an inactivated vaccine, is safe in pregnancy. However, it is indicated only in unvaccinated or high-risk patients (eg, HIV, sickle cell disease, prior splenectomy).

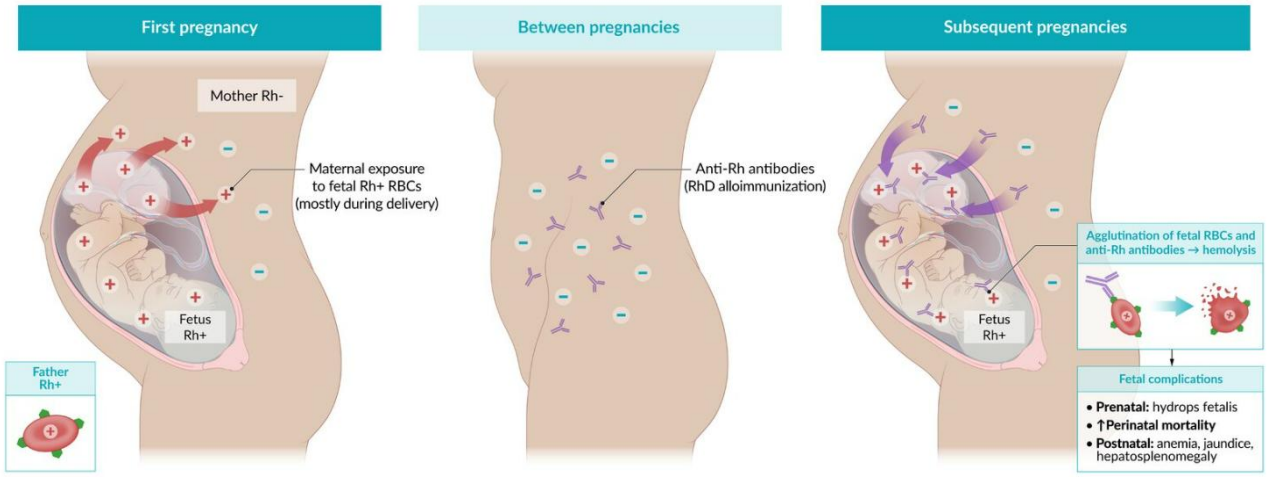
Varicella in pregnancy can be associated with maternal complications (eg, pneumonia, meningitis, encephalitis) and with congenital anomalies (eg, cicatricial lesions, cataracts, chorioretinitis). Patients who are exposed to varicella but lack evidence of varicella immunity (eg, negative IgG antibody serologic testing, no history of childhood infection) are treated with postexposure prophylaxis. In pregnancy, postexposure prophylaxis includes varicella-zoster immunoglobulin administration. The varicella-zoster vaccine, a live-attenuated vaccine, is contraindicated in pregnancy.

So if a pregnant women is exposed to varicella in pregnancy we will check her IgG and if it is negative we give ***Varicella-zoster immunoglobulin*** not a vaccine// the vaccine is not safe during pregnancy.

RhD & ABO incompatibility:

Rh incompatibility [2][4]

- **Rh-negative mother** and **Rh-positive fetus** [5]
  - Production of maternal IgM antibodies against the Rh antigen occurs after exposure of fetal blood cells to the maternal circulation (fetomaternal hemorrhage) during delivery or an antepartum Rh sensitizing event, e.g.:
    - Threatened or confirmed pregnancy loss
    - Termination of pregnancy
    - Ectopic pregnancy
    - Invasive obstetric procedures
    - External cephalic version
    - Antepartum hemorrhage
    - Abdominal trauma
  - Over time, maternal IgM antibodies seroconvert to **Rh-IgG** (antibodies are able to cross the placenta).
  - In a **subsequent pregnancy** with an **Rh-positive fetus**: rapid production of **maternal IgG anti-D antibodies** to fetal RhD antigens → Rh-IgG **agglutination of fetal RBCs** with **hemolytic anemia** → risk of HDFN with possible **hydrops fetalis**
- Rh-incompatibility in combination with ABO incompatibility: reduction of Rh(D)-isoimmunization in Rh-negative mothers



IgM at first exposure, cant cross placenta. **IgG** seroconversion by the time of second pregnancy

Now an interesting factoid:

When the baby is both ABO and Rh incompatible this decreases the risk of Rh isoimmunization.

Bcs when the mother is exposed to the fetal RBCs the immune system already has antibodies against ABO type and destroys the RBCs quickly without being able to identify the D antigen and processing it and making memory B cells against it.

Subtypes and variants

Nonimmune hydrops fetalis [9]

- **Definition**: a subgroup of hemolytic diseases of the fetus and newborn not caused by red cell alloimmunization
- Epidemiology
  - Incidence: ~ 1 in 4,000 pregnancies
  - Accounts for over 90% of all hydrops fetalis cases
- **Etiology**
  - Congenital heart defects and arrhythmias
  - Chromosomal aberrations (e.g., Turner syndrome, Down syndrome, trisomy 18)
  - Severe fetal anemia (e.g., thalassemia, twin-to-twin transfusion syndrome, fetomaternal hemorrhage)
  - Congenital TORCH infections (especially parvovirus B19 infection)
- **Pathophysiology**: severe fetal anemia → hypoxia → ↓ hepatic and renal blood flow → activation of RAAS → ↑ central venous pressure and ↓ lymphatic flow → fetal edema [10]

Clinical features

Prenatal

- Hydrops fetalis (expected in cases of Rh incompatibility and in nonimmune hydrops fetalis)

Postnatal

- Neonatal anemia
- Hepatosplenomegaly
- Neonatal jaundice
  - Usually present at birth or manifests within the first 24 hours of life
  - In Rh incompatibility, unconjugated bilirubin levels may be dangerously high, causing **kernicterus**.
- Hypoxia
- Prematurity
- Scattered petechiae (rare but associated with poor prognosis) [11]

ABO incompatibility usually has a significantly milder course of disease than Rh incompatibility.

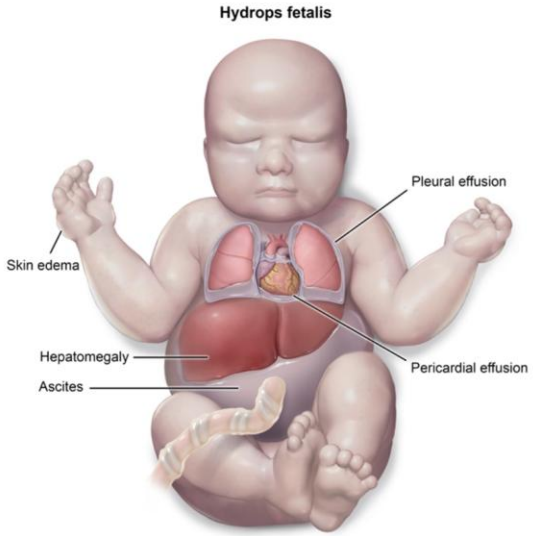
Anemia may conceal cyanosis.

Diagnosis

The diagnosis of HDFN requires evidence of hemolysis in the presence of fetomaternal blood incompatibility.

Prenatal diagnosis

- **Imaging**
  - Ultrasound: to determine hydrops fetalis
    - Fetal pleural or pericardial effusions
    - Fetal ascites
    - Fetal subcutaneous or nuchal edema
    - Placental edema
  - Doppler sonography of fetal blood vessels: Increased flow rate indicates fetal anemia.



Postnatal diagnosis

- If the newborn has signs of hemolysis, conduct a **Coombs test** (either direct or indirect).
  - Rh incompatibility: positive
  - ABO incompatibility: weak positive or negative

Treatment [12]

- **Prenatal**
  - **Intrauterine blood transfusion** via the umbilical vein, umbilical artery, peritoneal cavity, or heart (should only be performed in centers with experience in fetal transfusions.)
  - Possible IV immunoglobulin (IVIG) in severe cases
  - Early delivery in severely affected pregnancies
- **Postnatal**
  - Anemia: iron supplementation and, if necessary, RBC transfusion. [14]
  - Hyperbilirubinemia: **phototherapy**; if necessary, **exchange transfusion** with red blood cells
  - See "Treatment" in neonatal jaundice.
  - In severe cases, **IV immunoglobulin** (IVIG) may be administered.

Previous administration of RhIG may cause false-positive anti-D antibodies. [13]

This treatment if the hydrops fetalis happens of if the hemolytic disease happens.

But how do we prevent them?

Prevention

Of the causes of HDFN, only RhD incompatibility has a proven prophylactic treatment (i.e., RhIG). [15]

Screening for RhD alloimmunization in pregnancy [15][16]

- All pregnancies: Perform routine maternal ABO typing and Rh typing at the initial prenatal visit.
- RhD-positive pregnant individuals: Further screening is not indicated; provide routine prenatal care.
- RhD-negative pregnant individuals: Screen for anti-D antibodies with an indirect antiglobulin test. [12]
  - Anti-D titers positive (sensitized): Refer to maternal-fetal medicine for treatment of alloimmunization in pregnancy. [12][15]
  - Anti-D negative (not sensitized): See "Management of unsensitized RhD-negative individuals during pregnancy." [17]

Management of unsensitized RhD-negative individuals during pregnancy [12][15]

- Educate patients to seek care immediately after potentially Rh-sensitizing events.
- Give RhIG as indicated.
  - **Routine prophylaxis at 28 weeks'** gestation
  - Within **72 hours** of Rh-sensitizing events (including invasive prenatal diagnostic testing)
  - At delivery if newborn blood tests show the infant is RhD-positive
- Reassess for sensitization at 24–28 weeks' gestation and before each dose of RhIG (if feasible). [12][15]

> ! If repeat screening shows the patient is now sensitized, do not give RhIG; start treatment of alloimmunization in pregnancy. [12]

Anti-D immunoglobulin (RhIG)

- Given to **unsensitized RhD-negative** pregnant individuals to prevent Rh-sensitization
- Rapidly clears Rh-positive erythrocytes, preventing the development of anti-D antibodies [18]
- Dosages of RhIG are usually fixed, but fetomaternal hemorrhage tests may be needed to determine if larger doses are required.

> ! RhIG protects fetuses in subsequent pregnancies. [15]

Fetomaternal hemorrhage tests

- **Indications**
  - Rh-sensitizing events > 20 weeks' gestation
  - After delivery of a RhD-positive fetus
- **Initial testing**: rosette test to qualitatively detect fetal-maternal hemorrhage
- **Subsequent testing (if positive rosette test)**: Kleihauer-Betke test (most common) or flow cytometry
  - Quantitative tests that calculate fetal blood in the maternal circulation
  - Used to determine RhIG dosing

Indications for prophylactic administration of anti-D immune globulin for Rh(D)-negative patients*

- At 28-32 weeks gestation
- <72 hours after delivery of Rh(D)-positive infant
- <72 hours after spontaneous abortion
- Ectopic pregnancy
- Threatened abortion
- Hydatidiform mole
- Chorionic villus sampling, amniocentesis
- Abdominal trauma
- 2nd- & 3rd-trimester bleeding
- External cephalic version

*Antepartum prophylaxis is not indicated if the father is Rh(D) negative.

What screening test is used to determine if an Rh(D)- pregnant woman has already alloimmunized?

Antibody screen (indirect Coombs test) detects the presence of any RBC antibodies; Rh(D)- women with a negative antibody screen should still receive anti-D immune globulin at 28-32 weeks gestation.

• The **Kleihauer-Betke test** may be used to determine if a *higher dose* of **anti-D immune globulin** is needed after a procedure or delivery.

*the **standard dose (300 mg)** is enough to neutralize 30 mL of fetal blood; this test is typically used when there is an increased risk of fetal blood cells entering the maternal circulation (e.g. **placental abruption**, **amniocentesis**)*

ABO hemolytic disease	
Risk factors	• Infants with blood types A or B born to a mother with blood type O
Clinical features	• Jaundice within 24 hours of birth • Anemia • ↑ Reticulocyte count • Hyperbilirubinemia • Positive Coombs test
Management	• Serial bilirubin levels, oral hydration & phototherapy for most neonates • Exchange transfusion for severe anemia/hyperbilirubinemia

• Antibodies against A and B are **hemolytic**. However, in addition to red blood cells, A and B antigens are present on the **cells of all other fetal tissues**. The reaction of the anti-A and anti-B antibodies with the antigens on these other cells **neutralizes** the antibody response, leading to a much **milder** form of hemolytic disease.

• Non-invasive diagnosis of **fetal anemia** may be made by **middle cerebral artery peak systolic velocity** on **Doppler ultrasound**

useful for fetuses at risk of anemia due to red cell alloimmunization

Differential diagnoses of petechiae in newborns

Neonatal alloimmune thrombocytopenia

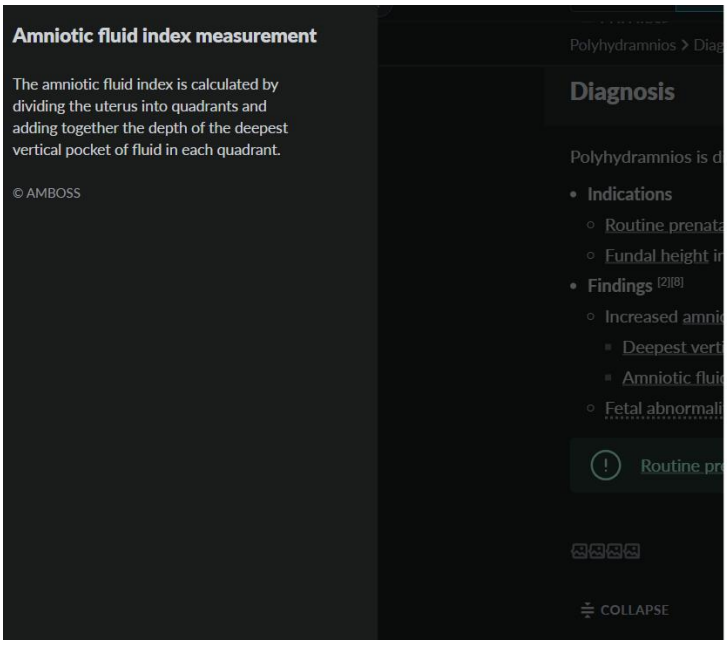
- **Description**: a rare condition in newborns characterized by maternal-fetal platelet incompatibility resulting in fetal thrombocytopenia
- **Epidemiology**: the leading cause of severe thrombocytopenia in the newborn
- **Pathophysiology**: formation of maternal antibodies against fetal platelets (most commonly targeting platelet antigen 1a) → maternal IgG cross the placenta and result in the destruction of fetal platelets → fetal and neonatal thrombocytopenia
- **Clinical features**
  - Mild: asymptomatic thrombocytopenia
  - Moderate: petechia and/or ecchymoses within a few hours after birth
  - Severe: spontaneous intracranial hemorrhage

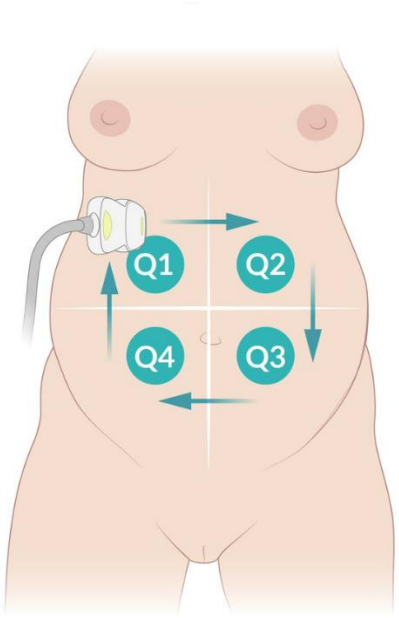
Other

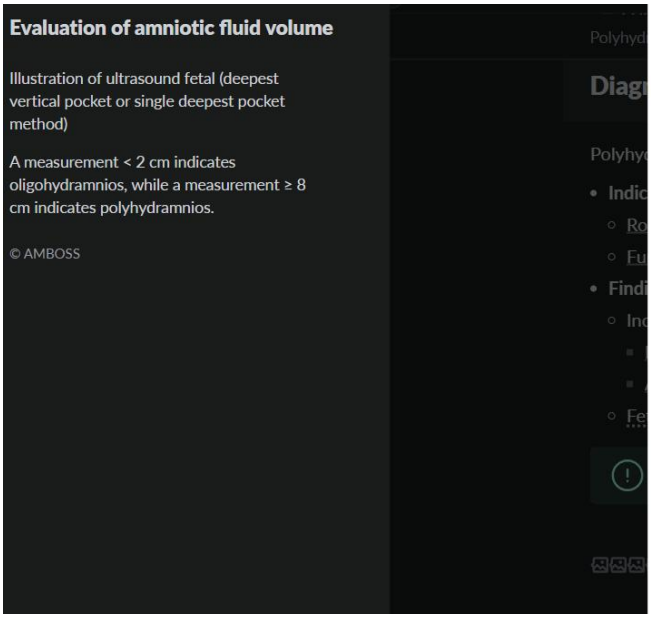
- Immune thrombocytopenic purpura
- Kasabach-Meritt syndrome
- Perinatal infections
- For an overview of thrombocytopenias and disorders of platelet function, see "Differential diagnosis of platelet disorders."

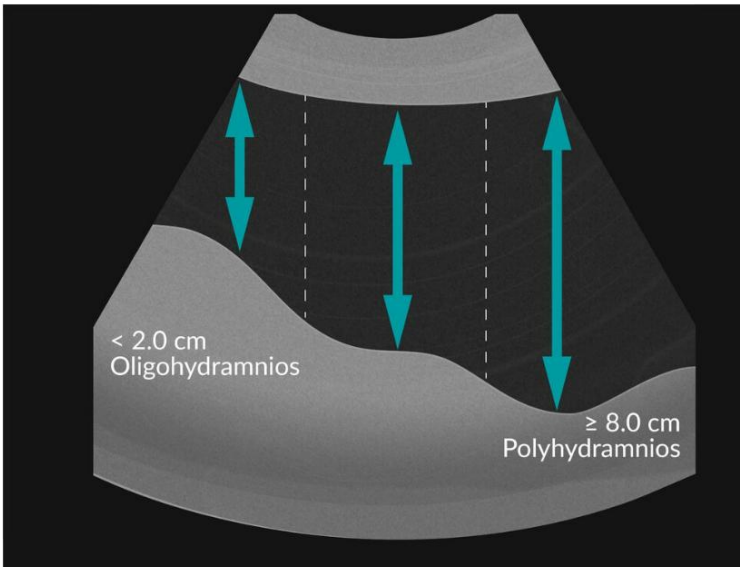
Amniotic Fluids:

Amniotic fluid index		
	Oligohydramnios (AFI <5 cm)	**Polyhydramnios (AFI ≥24 cm)**
Causes	Preeclampsia Abruptio placentae Uteroplacental insufficiency Renal anomalies NSAIDs	Esophageal/duodenal atresia Anencephaly Multiple gestation Congenital infection Diabetes mellitus
Complications	Meconium aspiration Preterm delivery Umbilical cord compression	Fetal malpresentation Umbilical cord prolapse Preterm labor Preterm prelabor rupture of membranes
AFI = amniotic fluid index; **NSAIDs** = nonsteroidal anti-inflammatory drugs.		









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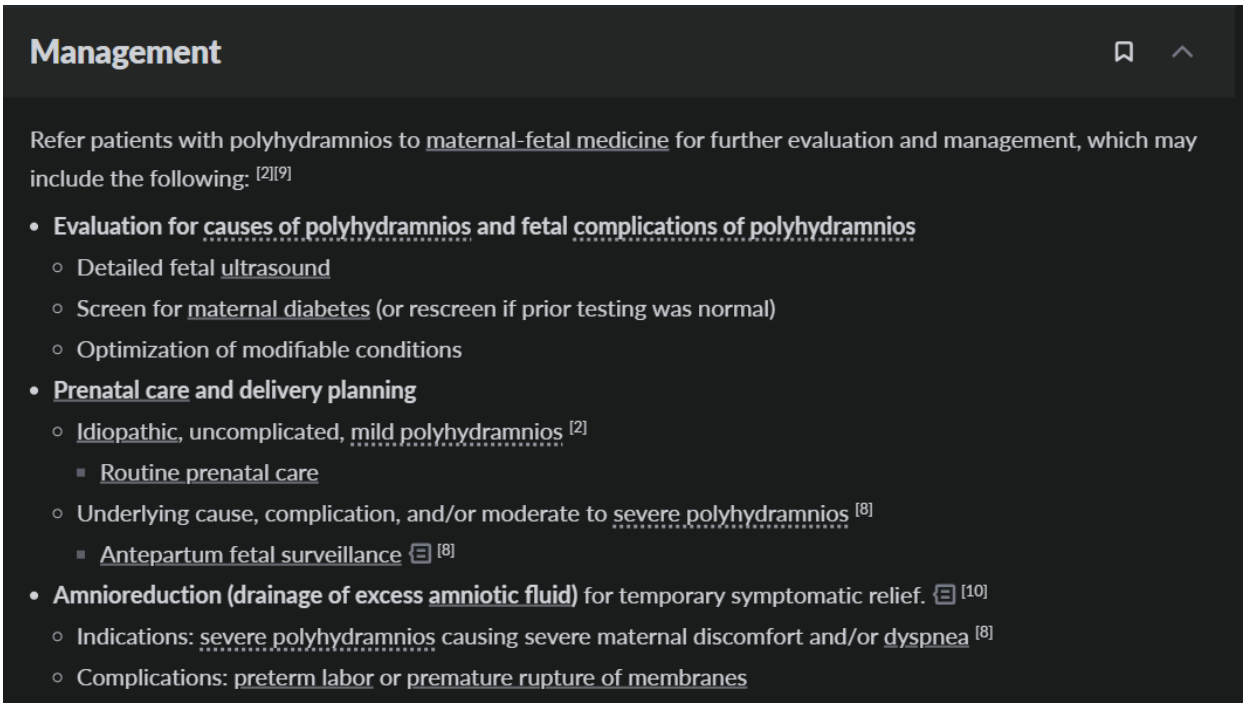


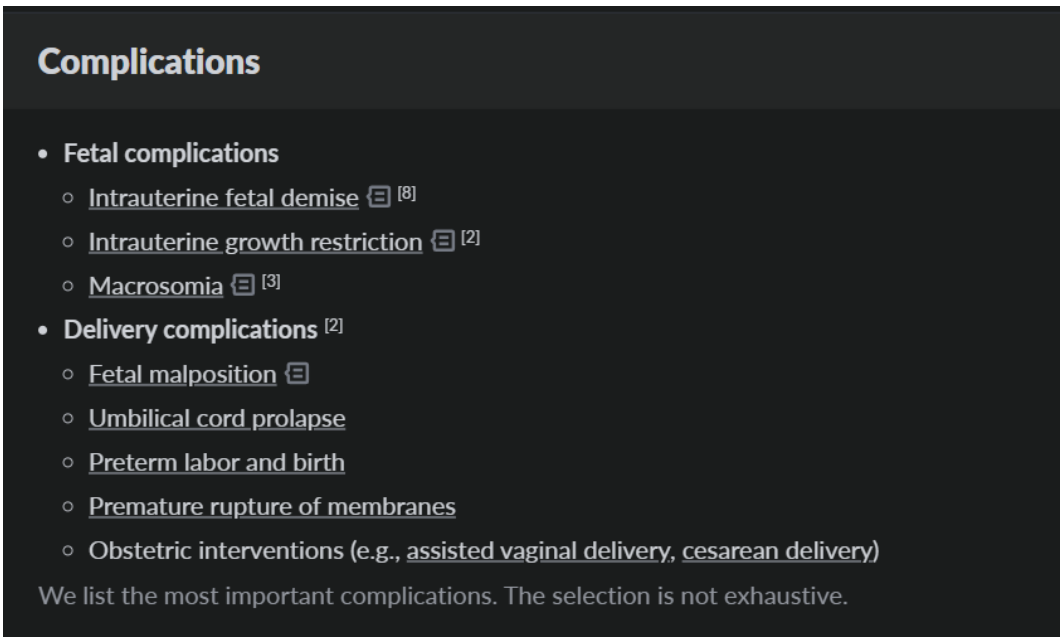
Management

Refer patients with polyhydramnios to maternal-fetal medicine for further evaluation and management, which may include the following: [2][9]

- **Evaluation for causes of polyhydramnios and fetal complications of polyhydramnios**
  - Detailed fetal ultrasound
  - Screen for maternal diabetes (or rescreen if prior testing was normal)
  - Optimization of modifiable conditions
- **Prenatal care and delivery planning**
  - Idiopathic, uncomplicated, mild polyhydramnios [2]
    - Routine prenatal care
  - Underlying cause, complication, and/or moderate to severe polyhydramnios [8]
    - Antepartum fetal surveillance [8]
- **Amnioreduction (drainage of excess amniotic fluid)** for temporary symptomatic relief. [10]
  - Indications: severe polyhydramnios causing severe maternal discomfort and/or dyspnea [8]
  - Complications: preterm labor or premature rupture of membranes

Patients with polyhydramnios may have abdominal discomfort (from increased uterine size), dyspnea (due to maternal lung compression), and preterm contractions (from increased intrauterine pressure); however, most patients are asymptomatic. Management is based on severity, maternal symptoms, and gestational age:

- Patients with severe or symptomatic polyhydramnios at preterm gestation are at increased risk for obstetric complications, including preterm labor and preterm prelabor rupture of membranes. Therefore, these patients may benefit from amnioreduction (ie, amniotic fluid removal by amniocentesis) **(Choice A)**.
- In contrast, patients with **mild**, **asymptomatic** polyhydramnios at **term gestation**, such as this patient, can undergo **expectant management** because obstetric outcomes are unchanged by increased antenatal fetal surveillance or intervention.



Complications

- **Fetal complications**
  - Intrauterine fetal demise [8]
  - Intrauterine growth restriction [2]
  - Macrosomia [3]
- **Delivery complications** [2]
  - Fetal malposition
  - Umbilical cord prolapse
  - Preterm labor and birth
  - Premature rupture of membranes
  - Obstetric interventions (e.g., assisted vaginal delivery, cesarean delivery)

We list the most important complications. The selection is not exhaustive.

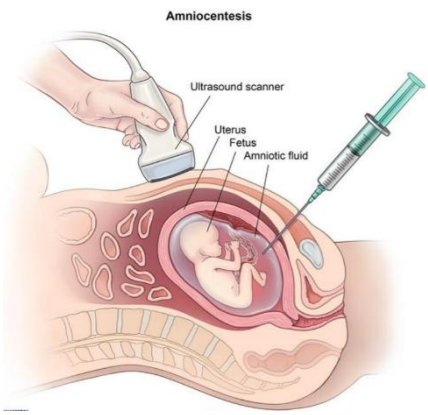
Polyhydramnios

Management is based on severity, maternal symptoms, and gestational age:

- Patients with **severe** or **symptomatic polyhydramnios** at **preterm gestation** are at increased risk for obstetric complications, including preterm labor and preterm prelabor rupture of membranes. Therefore, these patients may benefit from **amnioreduction** (ie, amniotic fluid removal by amniocentesis)
- Patients with **mild**, **asymptomatic** polyhydramnios at **term gestation**, can undergo **expectant management** because obstetric outcomes are unchanged by increased antenatal fetal surveillance or intervention.

Most patients with polyhydramnios are asymptomatic and have a **uterine size-larger-than-dates discrepancy** (eg, fundal height 32 cm at 24 weeks gestation); others may have dyspnea due to insufficient maternal lung expansion from an enlarged uterus.

Polyhydramnios, especially with increasing severity, can cause obstetric complications due to uterine overdistension and increased intraamniotic pressure. The increased tension of the fetal membranes makes them more susceptible to rupture, placing these patients at higher risk for **preterm prelabor rupture of membranes**. Uterine overdistension may also cause inflammation, prostaglandin release, uterine irritability, and an increased risk of **preterm labor**. Additional complications include fetal malpresentation (eg, breech), umbilical cord prolapse, and postpartum uterine atony. Patients with symptomatic polyhydramnios may benefit from amnioreduction (ie, amniotic fluid removal by amniocentesis).



Oligohydramnios

The etiology of oligohydramnios can vary with gestational age:

- **Early-gestation oligohydramnios** is concerning for **fetal etiologies** (eg, aneuploidy, renal agenesis, posterior urethral valves) because amniotic fluid volume is dependent on normal fetal urine production.
- **Second- and third-trimester** causes of oligohydramnios are typically due to **uteroplacental insufficiency** (with concomitant **fetal growth restriction**) or maternal causes, such as **dehydration** or **rupture of membranes** (with **normal fetal growth**).

Antepartum fetal surveillance			
Test	**Description**	**Normal result**	**Abnormal result**
Nonstress test	External fetal heart rate monitoring for 20-40 minutes	• Reactive: ≥2 accelerations • 2 points	• Nonreactive: <2 accelerations • Recurrent variable or late decelerations • 0 points
Biophysical profile	• Nonstress test plus ultrasound assessment of the following: ◦ Amniotic fluid volume ◦ Fetal breathing movement ◦ Fetal movement ◦ Fetal tone • 2 points per category if normal (maximum 10/10) • 0 points per category if abnormal	8 or 10 points	• Equivocal: 6 points • Abnormal: 0, 2, or 4 points • Oligohydramnios
Contraction stress test	External fetal heart rate monitoring during spontaneous or induced (eg, oxytocin, nipple stimulation) uterine contractions	No late or recurrent variable decelerations	Late decelerations with >50% of contractions
Doppler sonography of the umbilical artery	Evaluation of umbilical artery flow in fetal intrauterine growth restriction only	High-velocity diastolic flow in umbilical artery	Decreased, absent, or reversed end-diastolic flow

Biophysical profile*	
Component	**Normal finding**
1. Nonstress test	Reactive fetal heart rate monitoring
2. Amniotic fluid volume	Single fluid pocket ≥2 x 1 cm or amniotic fluid index >5
3. Fetal movements	≥3 general body movements
4. Fetal tone	≥1 episodes of flexion/extension of fetal limbs or spine
5. Fetal breathing movements	≥1 breathing episode for ≥30 seconds

Maximum score = 10; 0 = abnormal; 2 = normal for each component
*Performed continuous observation for ≥30 minutes

- Management?
  - **0-4: fetal hypoxia → urgent delivery**
  - **~6**: repeat in **24 hours**
  - **8-10: normal** and rules out fetal hypoxia

- What is the *recommended management* for a pregnant woman at 32 weeks gestation with gestational hypertension and a **biophysical profile score of 6** at today's visit?

  Repeat biophysical profile in 24 hours

pregnant women with gestational hypertension need weekly BPPs starting at 32 weeks gestation.

• What is the *next step* in management for a pregnant woman at **34 weeks** gestation that presents with **decreased fetal movement**? Non-stress testing reveals a normal FHR but is non-reactive despite vibroacoustic stimulation.

Biophysical profile or Contraction stress test

choice of test depends on resources available and contraindications
(e.g.
***contraction stress test** is **contraindicated** if there are contraindications to labor, such as placenta previa or prior myomectomy)*

💡 Normal fetal movements: **≥10 movements in 2 hours**

• What is the *next step* in management for a pregnant woman at **32 weeks** gestation that presents with **decreased fetal movement**? Heart tones are heard by Doppler.

Non-stress test

i.e. recording the fetal heart rate while monitoring for spontaneous perceived fetal movements

• What is the *most common cause* of a non-reactive **non-stress test**?

Fetal sleep cycle

*vibroacoustic stimulation may be used to **awaken** the fetus;*

*sleep cycles can last as long as **40 minutes** therefore a non-reactive test should be extended to 40 - 120 minutes*

• What is a **normal (reactive) nonstress test**?

Nonstress test	
Reactive	• Baseline of 110-160/min • Moderate variability (6-25/min) • ≥2 accelerations in 20 minutes, each peaking ≥15/min above baseline & lasting ≥15 seconds
Nonreactive	• Does not meet criteria for reactivity

*Due to fetal sympathetic nervous system activity, which develops around **26 - 28 weeks (no reactivity before 28 weeks)***

💡 **NST → Non-reactive → Vibroacoustic Test/BPP → Still Non reactive/ equivoval or low score → Contraction Stress Test**

• Can a **reactive non-stress test** effectively rule *out* **fetal acidemia**?

Yes

a reactive NST has a high negative predictive value for fetal acidemia

Non-reactive NST *has a low positive predictive value for fetal acidemia (**high rate of false positives**)*

CTG:

Late fetal decelerations are a sign of uteroplacental insufficiency and impending **fetal hypoxemia and acidemia**.

One cause can be due to uterine tachysystole, defined as >5 contractions/10 min. Uterine contractions temporarily interrupt intervillous blood flow; excessively frequent contractions, as in this patient, may cause fetal compromise as a result of decreased uteroplacental blood flow during contractions and inadequate recovery time (resumption of blood flow) between contractions.

Although uterine tachysystole can occur in spontaneous labor, there is an increased risk with induced or augmented labor (eg, uterotonic agents). Uterine tachysystole is managed with supportive measures (eg, lateral maternal repositioning, tocolysis) and **discontinuation of uterotonic agents** (eg, oxytocin) until the excessive uterine activity and resulting fetal decelerations resolve

Prenatal:

Routine prenatal laboratory tests	
Initial prenatal visit	• Rh(D) type & antibody screen • Hemoglobin/hematocrit, MCV, ferritin • HIV, VDRL/RPR, HBsAg, anti-HCV Ab • Rubella & varicella immunity • Urine culture • Urine dipstick for protein • Chlamydia PCR (if risk factors are present) • Pap test (if screening indicated)
24-28 weeks	• Hemoglobin/hematocrit • Antibody screen if Rh(D)-negative • 1-hr 50-g GCT
36-38 weeks	• Group B *Streptococcus* rectovaginal culture
anti-HCV Ab = hepatitis C antibody; **GCT** = glucose challenge test; **HBsAg** = hepatitis B surface antigen; **MCV** = mean corpuscular volume; **PCR** = polymerase chain reaction; **RPR** = rapid plasma reagin.	

100g 3-hour OGT to confirm !

An **indirect antiglobulin (Coombs) test** can be used to determine whether an Rh-negative patient has developed anti-Rh antibodies and is an important component of early prenatal care,
especially in patients with previous pregnancies.

Prenatal visits are recommended **every 4 weeks until 28 weeks** of gestation, **every 2 weeks from 28 to 36 weeks** of gestation and then **every week until delivery**.

High-risk sexually transmitted infection screening in pregnancy	
High-risk patients	• Age <25 • Prior sexually transmitted infection • High-risk sexual activity (eg, multiple partners, commercial sex work)

Required screening	• Performed at initial prenatal visit & 3rd trimester:
	  ◦ HIV
	  ◦ Syphilis
	  ◦ Hepatitis B & C viruses
	  ◦ Gonorrhea
	  ◦ *Chlamydia*

Patients treated for a sexually transmitted infection during pregnancy require retesting a month after completion of treatment to ensure response to therapy (ie, test of cure). Patients with a sexually transmitted infection diagnosed earlier during pregnancy are also retested in the third trimester, prior to delivery.

Prenatal testing			
Test	**Timing (weeks)**	**Advantages**	**Disadvantages**
First-trimester combined test*	9-13	Early screening	Not diagnostic
Cell-free fetal DNA	≥10	High sensitivity & specificity for aneuploidy	Not diagnostic
Chorionic villus sampling	10-13	Definitive karyotypic diagnosis	Invasive; risk of spontaneous abortion
Second-trimester quadruple screen**	15-22	Screens for neural tube defects & aneuploidy	Not diagnostic
Amniocentesis	15-20	Definitive karyotypic diagnosis	Invasive; risk of membrane rupture, fetal injury & pregnancy loss
Second-trimester ultrasound	18-20	Measures fetal growth, evaluates fetal anatomy, confirms placenta position	Cannot identify all abnormalities; some findings are of uncertain significance

*Pregnancy-associated plasma protein, β-hCG, nuchal translucency.

**Maternal serum α-fetoprotein, estriol, β-hCG, inhibin A.

1st trimester screening		
Trisomy	β-hCG	PAPP-A
21	↑	↓
18	↓	↓
13	↓	↓

2nd trimester screening				
Trisomy	β-hCG	Inhibin A	Estriol	AFP
21	↑	↑	↓	↓
18	↓	— or ↓	↓	↓
13	—	—	—	—

When is cell-free DNA testing (cffDNA) indicated?

Test used in 10 weeks gestation onwards to identify chromosomal aberrations (high specificity and sensitivity for aneuplodies); not diagnostic (must confirm with CVS or amniocentesis)

Cell-free fetal DNA testing	
Indications	• Maternal age ≥35 • Abnormal maternal serum screening test • Sonographic findings associated with fetal aneuploidy • Previous pregnancy with fetal aneuploidy • Parental-balanced Robertsonian translocation
Applications	• Screening for trisomy 21, 18, 13 & sex-chromosome aneuploidies • Fetal sex determination

Cell free DNA is also used to screen for **heritable disorders**. In white patients these include **Cystic Fibrosis**, **Tay Sachs**, **Spinal Muscular Atrophy.**

What is the next step in management for a pregnant woman at 12 weeks gestation with an abnormally elevated beta-hCG level and increased nuchal thickness on ultrasound?

Chorionic villous sampling (CVS)

this patient has a positive first-trimester combined test; diagnosis is made via CVS or amniocentesis depending on gestational age.

2nd trimester: -Triple marker test: MS-AFP, HCG, Estriol

Quadruple marker test: MS-AFP, HCG, Estriol, Inhibin-A

Maternal Serum Alpha Feto Protein (MS-AFP) → Elective NOT routine prenatal test

AFP: is a major serum glycoprotein of the embryo

Peaks at 12 weeks (in fetus and amniotic fluid)

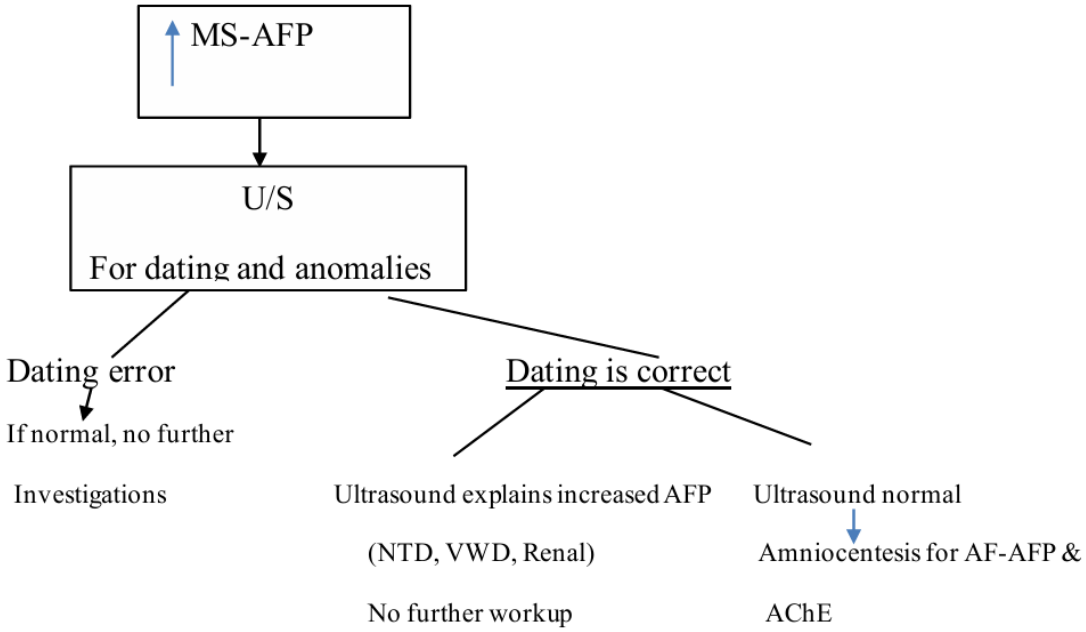
Rises until 30 weeks (in the maternal serum)

MS-AFP: Performed within 15-20 weeks GA

Fetal serum AFP	Peaks at 12 weeks
Amniotic fluid AFP	Peaks at 12 weeks
Maternal AFP	Peaks at 30 weeks

Normal: 0.75-2.5

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



Amniocentesis for AF-AFP & AChE

Because the cause is still unknown, you must look more closely at the fetal environment.

- **Procedure:** Perform an **amniocentesis** to obtain **amniotic fluid (AF)**.
- **Tests on the Amniotic Fluid:**

1. **Amniotic Fluid AFP (AF-AFP):** Directly measures the level of AFP **in the fluid surrounding the fetus**. A **high AF-AFP** confirms that the fetus is the source of the protein leak (as opposed to a maternal source).
2. **Acetylcholinesterase (AChE):** This is a **neurological enzyme**. Its presence in the amniotic fluid is a **specific marker for open neural tube defects** (like

spina bifida) that might have been **too small to see on ultrasound**. It can also be elevated in ventral wall defects (e.g., omphalocele, gastroschisis).

Interpreting the Amniocentesis Results

Result Pattern	Likely Diagnosis	Implication
AF-AFP: High **AChE: Positive**	**Open Neural Tube Defect (NTD)** or **Ventral Wall Defect (VWD)** that was missed on ultrasound.	Confirms a fetal structural anomaly. Requires detailed counselling, planning for delivery at a tertiary care center, and possible fetal surgery consultation.
AF-AFP: High **AChE: Negative**	**Other causes:** Fetal renal disease (e.g., congenital nephrosis), placental abnormalities, or other rare conditions.	Further workup needed (e.g., detailed fetal echo, renal ultrasound, genetic testing).
AF-AFP: Normal	**False-positive MSAFP.** The elevation was likely due to **maternal factors** (e.g., maternal liver disease) or a technical error.	**No fetal risk.** The pregnancy can be managed as normal.

Maternal serum α-fetoprotein screening

↑ MSAFP	↓ MSAFP
Open neural tube defects (eg, anencephaly, open spina bifida) Ventral wall defects (eg, omphalocele, gastroschisis) Multiple gestation	Aneuploidies (eg, trisomy 18 & 21)

Alpha-fetoprotein (AFP) is a major protein produced by the fetal yolk sac, liver, and gastrointestinal tract. Maternal serum AFP (MSAFP) is measured at 15-20 weeks gestation (optimally at 16-18 weeks) to screen for fetal anomalies. MSAFP is used primarily to screen for **open neural tube defects**. Increased levels are also associated with fetal **abdominal wall defects** (eg, gastroschisis, omphalocele) and **multiple gestation**. Less commonly, an increased MSAFP can be seen in fetal congenital nephrosis and benign obstructive uropathy.

An elevated MSAFP warrants careful **ultrasound** evaluation of the fetal anatomy. In addition, the number of fetuses should be clarified as multiple gestations produce more AFP. Gestational age is also confirmed as interpretation of AFP level depends on an accurate gestational age.

Fetal growth restriction:

Fetal growth restriction	
Definition	• Weight <10th percentile for gestational age
Risk factors	• Maternal hypertension • Pregestational diabetes mellitus • Genetic abnormalities • Congenital infection
Appearance	• Large anterior fontanel • Thin umbilical cord • Loose, peeling skin • Minimal subcutaneous fat
Evaluation	• Placenta histopathology • Consider karyotype, urine toxicology, serology
Neonatal complications	• Polycythemia • Hypoglycemia • Hypocalcemia • Poor thermoregulation
Management	• Monitor/treat complications (eg, hypoglycemia, hypothermia, polycythemia) • Hypoglycemia: frequent screening and frequent feedings • Hypothermia: skin to skin with mother, examinations in incubator • Polycythemia and hypocalcemia: screen if symptoms develop (eg, poor feeding, vomiting, jitteriness)

• FGR can be a complication of **maternal vascular disease** (eg, type 1 diabetes mellitus, chronic hypertension), which causes chronic placental vasoconstriction and ischemia with resultant **uteroplacental insufficiency**

Patients with FGR have a high risk for **intrauterine fetal demise** and require an immediate **umbilical artery Doppler ultrasound** to assess placental perfusion

💡 **Bicornuate uterus** with two separate endometrial cavities has less room for appropriate egg implantation, placental development, and fetal growth. Patients with this abnormality are also at increased risk for **preterm delivery**, **fetal growth restriction**, and **malpresentation**.

Fetal growth restriction		
	Symmetric	**Asymmetric**
Definition	Ultrasound estimated fetal weight <10th percentile or birth weight <3rd percentile for gestational age	
Onset	1st trimester	2nd/3rd trimester
Etiology	Chromosomal abnormalities Congenital infection	Uteroplacental insufficiency Maternal malnutrition
Clinical features	Global growth lag	Head-sparing growth lag
Management	Regular nonstress testing Weekly biophysical profiles Serial umbilical artery Doppler sonography Serial growth ultrasounds	

The definition states that **both symmetric and asymmetric FGR** are diagnosed when:

> **Ultrasound estimated fetal weight (EFW) <10th percentile**
> **OR**
> **Birth weight <3rd percentile** for gestational age.

Fetuses with growth restriction have increased risk for intrauterine demise and neonatal morbidity/mortality (eg, preterm delivery).

FGR can be characterized as either symmetric or asymmetric:

- Symmetric FGR is uniform and equal global growth impairment (eg, head, femur, abdomen) that begins in the first trimester due to an early developmental insult (eg, aneuploidy, congenital infection).
- In contrast, **asymmetric FGR** is **disproportionate** growth impairment due to **uteroplacental insufficiency** and subsequent chronic fetal hypoxia. Maternal conditions, such as hypertension and pregestational diabetes mellitus, predispose to abnormal placental development that causes progressive placental dysfunction during the late **second and/or third trimester**. The fetus adapts to impaired blood flow and reduced oxygen delivery by redistributing blood to vital organs (eg, brain) and away from nonvital organs (eg, abdomen), causing a **head-sparing growth** pattern.

Differential diagnoses

Constitutionally small fetus

- **Definition**: estimated fetal weight < 10th percentile without an identified underlying condition
- **Predisposing factors** [15]
  - Low maternal height
  - Low maternal weight before/in early pregnancy
  - Asian descent [16]
  - Parity
  - Fetal female sex
- **Diagnosis**
  - Assessment of fetal growth using customized growth charts [17]
  - Doppler velocimetry of the umbilical artery: normal systolic/diastolic ratio [18]
- **Prognosis**: constitutionally small fetuses are not at increased risk for adverse perinatal outcomes

Fetal constitutional smallness is associated with **short maternal stature, low prepregnancy weight**, and **fetal female sex**. Constitutional smallness presents with an ultrasound-estimated **fetal weight <10%** for gestational age, **normal umbilical artery Doppler evaluation**, and **appropriate interval growth**.

Constitutionally small infants have **normal long-term outcomes.**

Fetal growth restriction (FGR)

AKA **intrauterine growth restriction (IUGR)**

= Estimated fetal weight < 10th percentile OR birth weight < 3rd percentile for gestational age

0 weeks — 13 / 14 weeks — 27 / 28 weeks — 40 weeks

	Symmetrical IUGR	Asymmetrical IUGR
Onset	**1st trimester**	**2nd / 3rd trimester**
Growth lag	**Global growth restriction**	**Disproportionate growth restriction** ("Head-sparing")
Etiology	• Chromosomal abnormalities (e.g., aneuploidy) • Congenital heart disease • Congenital TORCH infection	• Uteroplacental insufficiency • Maternal systemic disease (e.g., hypertension) • Maternal malnutrition
	- Affects the **formation** of organs - ↑ risk of **neurologic sequelae**	- Affects the **growth** of organs; body and limbs are thin and small - The brain develops normally though; head dimensions are normal

TWINS:

Twin pregnancy	

Types	• Monochorionic, monoamniotic
	  ◦ 1 placenta, 1 amniotic sac
	• Monochorionic, diamniotic
	  ◦ 1 placenta, 2 amniotic sacs
	  ◦ "T-sign" at intertwin membrane
	• Dichorionic, diamniotic
	  ◦ 2 placentas, 2 amniotic sacs
	  ◦ "Lambda sign" at intertwin membrane

Maternal complications	• Hyperemesis gravidarum • Preeclampsia • Gestational diabetes mellitus • Iron-deficiency anemia

Fetal complications	• Congenital anomalies
	• Fetal growth restriction
	• Preterm delivery (MC)
	• Malpresentation (eg, breech)
	• Monochorionic twins
	  ◦ Twin-twin transfusion syndrome
	• Monoamniotic twins
	  ◦ Conjoined twins
	  ◦ Cord entanglement

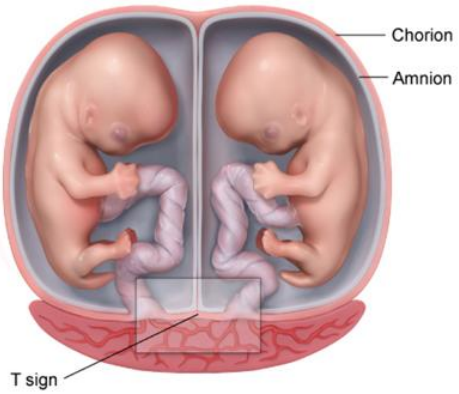
The risk of preterm labor during a multiple gestation pregnancy may be reduced with adequate early weight gain.

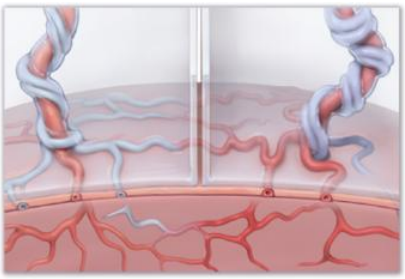
• **Monochorionic monoamniotic** twins are typically managed **inpatient** beginning at **28 weeks gestation** with frequent fetal monitoring (eg, nonstress test) and antenatal corticosteroid administration.

Patients are **delivered preterm** (**32-34 weeks gestation**) via **cesarean delivery**.

T sign for twins

Monochorionic diamniotic

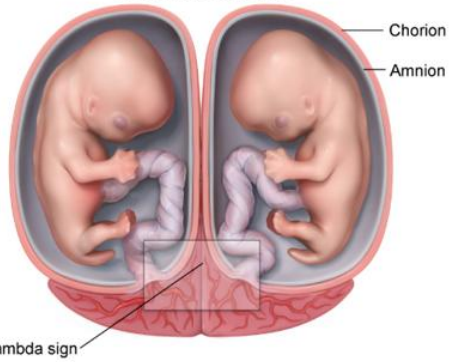


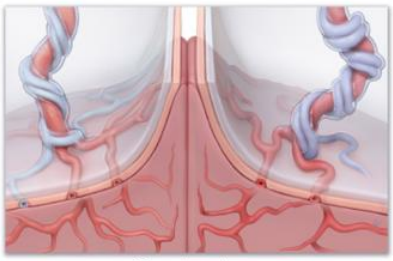


Single placenta

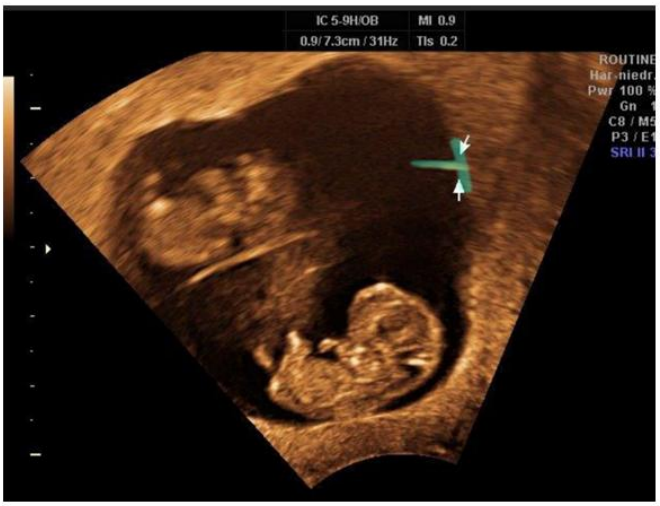
Lambda sign for twins

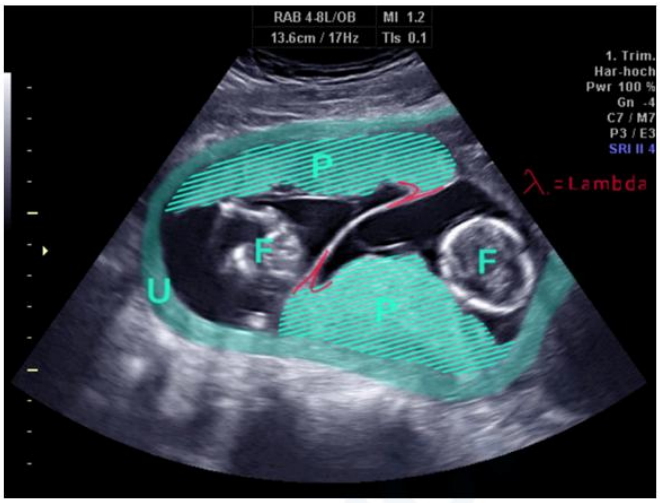
Dichorionic diamniotic





Two placentas





Hypertension:

Hypertensive disorders of pregnancy	
Chronic hypertension	• Systolic pressure **≥140 mm Hg** &/or diastolic pressure **≥90 mm Hg** prior to conception or 20 weeks gestation
Gestational hypertension	• New-onset elevated blood pressure at ≥20 weeks gestation • No proteinuria or end-organ damage
Preeclampsia	• New-onset elevated blood pressure at ≥20 weeks gestation **AND** • Proteinuria **OR** signs of end-organ damage
Eclampsia	• Preeclampsia **AND** • New-onset grand mal seizures
Chronic hypertension with superimposed preeclampsia	Chronic hypertension **AND** 1 of the following: • New-onset proteinuria or worsening of existing proteinuria at ≥20 weeks gestation • Sudden worsening of hypertension • Signs of end-organ damage

*Elevated blood pressure must be seen on 2 separate measurements taken **at least 4 hours apart***

Obstetric complications of hypertension	
Maternal	• Superimposed preeclampsia • Cesarean delivery • Abruptio placentae • Postpartum hemorrhage • Maternal mortality
Fetal	• Fetal growth restriction ± oligohydramnios • Preterm delivery • Intrauterine fetal demise • Perinatal mortality

Antihypertensive medications in pregnancy		
First-line (safe)	**Second-line**	**Contraindicated**
• **Methyldopa** • Beta blockers (**labetalol**) • **Hydralazine** • Calcium channel blockers (nifedipine)	• Thiazide diuretics • Clonidine	• ACE inhibitors • Angiotensin receptor blockers • Aldosterone blockers • Direct renin inhibitors • Furosemide

Preeclampsia	
Risk factors	• Nulliparity • Obesity • Preexisting medical condition (eg, SLE, chronic hypertension) • Multiple gestation • Advanced maternal age
Definition	• New-onset hypertension* (SBP ≥140 or DBP ≥90 mm Hg) at ≥20 weeks AND • Proteinuria OR signs/symptoms of other end-organ damage
Severe features	• Severe-range hypertension (SBP ≥160 or DBP ≥110 mm Hg) • Platelets <100,000/mm^3 • Creatinine >1.1 mg/dL or 2x normal • Elevated transaminases >2x upper limit of normal • Pulmonary edema • Vision or cerebral symptoms (eg, headache)
Management	• <37 weeks & no severe features: expectant • ≥37 weeks (or ≥34 weeks with severe features): delivery • Severe-range blood pressure: IV labetalol, IV hydralazine, PO nifedipine • Seizure prophylaxis: magnesium sulfate

Preeclampsia prevention	
Definition	• New-onset hypertension & proteinuria &/or end-organ damage at >20 weeks gestation
High risk	• Prior preeclampsia • Chronic kidney disease • Chronic hypertension • Diabetes mellitus • Multiple gestation • Autoimmune disease
Moderate risk	• Obesity • Advanced maternal age • Nulliparity
Prevention	• Low-dose aspirin at 12 weeks gestation

• High-risk patients also require a **24-hour urine collection for total protein** at the **initial prenatal visit** to establish a baseline (prepregnancy) proteinuria assessment.

• What level of proteinuria is required to make a diagnosis of **preeclampsia**?

≥ 300 mg/day (or a protein/creatinine ratio > 0.3)

Patients with preeclampsia are at increased risk of hemorrhagic and ischemic stroke due to acute elevations in cerebral perfusion pressure and intracerebral vessel rupture (hemorrhagic), as well as preeclampsia-mediated vascular endothelial damage and microthrombi formation (ischemic).
NOT Subarachnoid Hemorrhage.

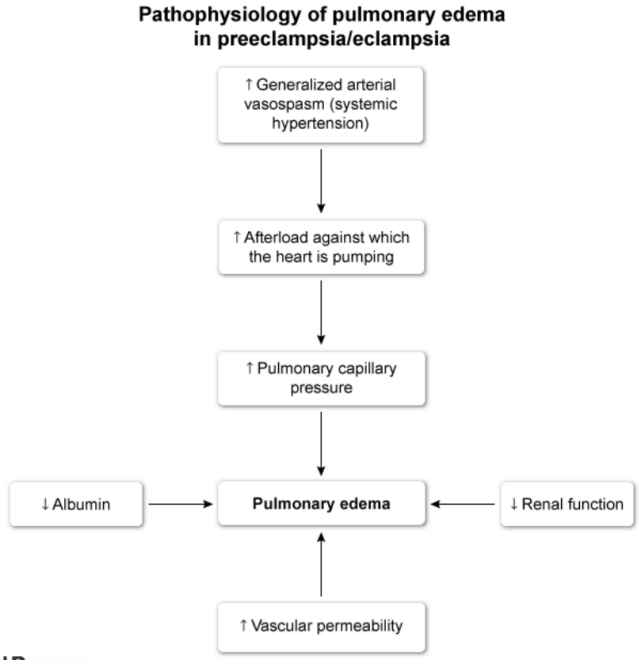
Preeclampsia can present **upto 6 weeks after delivery.**

• What is the likely *diagnosis* in a pregnant woman with preeclampsia that develops sudden-onset **dyspnea**, **hypoxia**, and **crackles**?

Pulmonary edema

rare but life-threatening complication of severe preeclampsia

*Preeclamptic patients have generalized arterial vasospasm leading to **increased systemic vascular resistance and high cardiac afterload** -- the heart becomes hyperdynamic to try to overcome the systemic hypertension.*



Treatment of preeclampsia	
Drug	**Indication**
Hydralazine IV, labetalol IV, or nifedipine PO	Lower blood pressure acutely to decrease stroke risk
Magnesium sulfate IV or IM	Prevent or treat eclamptic seizures

• In patients with Myasthenia Gravis and Preeclampsia, **magnesium sulfate** is **contraindicated** because it may trigger a **myasthenic crisis.**

In these patients, seizure prophylaxis is with **valproic acid.**

• What are the *first-line agents* (3) for **maternal hypertensive crisis**?

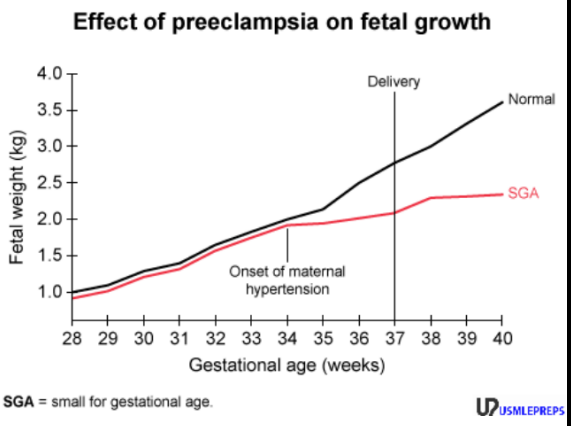
nifedipine, hydralazine, or labetalol

*Be careful with **labetalol** -- if patient has bradycardia(<60 bpm) or asthma, **do not give!***

***Nifedipine** is **oral**, thus patient may not be able to tolerate if they have emesis!*

During the first and second trimesters, the decreased placental perfusion is often subtle and not clinically noticeable because the placenta can keep up with the metabolic demands of the fetus. However, during the third trimester, there is rapid fetal growth (~0.2 kg [0.4 lb]/week), and the chronically under-perfused placenta **cannot provide adequate oxygen** to the fetus. In patients with preeclampsia, this **uteroplacental insufficiency** often coincides with the onset of **wide-spread maternal vasoconstriction**, which can manifest as **hypertension** and signs of **end-organ damage**, such as headache, proteinuria,

thrombocytopenia, and hemolysis (eg, anemia, elevated lactate dehydrogenase).



The addition of vasoconstriction on the already under-perfused placenta culminates in substantially decreased fetal oxygenation and nutrient supply. Over the course of several weeks (as in this patient with no prenatal care in a month), fetal growth can rapidly decrease, resulting in a **small-for-gestational-age infant** (potentially 1-2 lb less than average birth weight).

Eclampsia (preeclampsia + new-onset seizure)	
Clinical features	• Hypertension, typically severe (ie, SBP ≥160 or DBP ≥110 mm Hg) • Seizure, typically tonic-clonic with postictal phase • Severe headache • Visual disturbances (ie, scotoma) • Hyperreflexia • Proteinuria
Diagnosis	• Mainly clinical • Bilateral frontal lobe edema on CT scan of the head
Management	• Magnesium sulfate infusion • Antihypertensive agent for severe hypertension • Delivery

HELLP syndrome	
Clinical features	• Preeclampsia • Nausea/vomiting • Right upper quadrant abdominal pain
Laboratory findings	• Microangiopathic hemolytic anemia • Elevated liver enzymes • Low platelet count
Treatment	• Delivery • Magnesium for seizure prophylaxis • Antihypertensive drugs

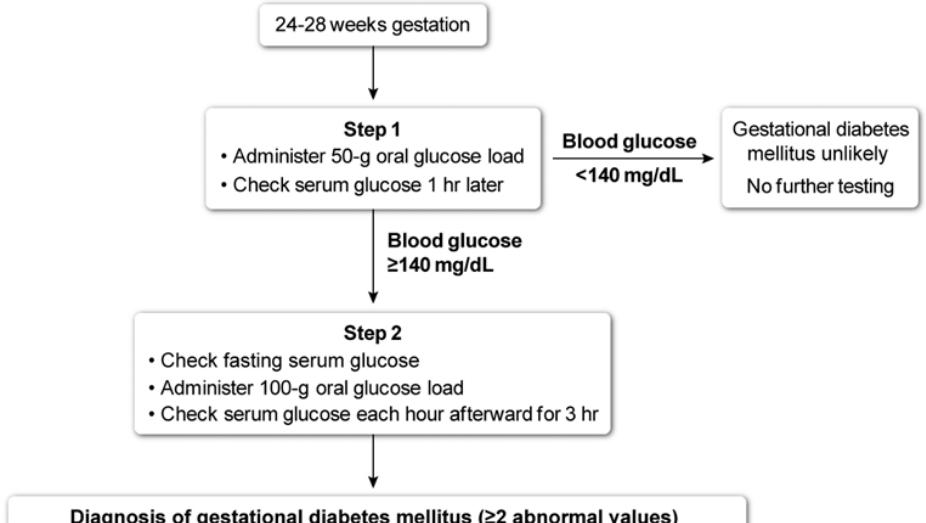
Delivery should occur at **≥ 34 weeks gestation** or with **deteriorating maternal/fetal status**

Gestational DM:

	Gestational diabetes mellitus
Pathophysiology	Human placental lactogen secretion
Screening	24-28 weeks gestation 1-hr 50-g GCT 3-hr 100-g GTT
Management	1st line: diet 2nd line: insulin, glyburide, metformin
Target blood glucose goals	Fasting: ≤95 mg/dL 1-hr postprandial: ≤140 mg/dL 2-hr postprandial: ≤120 mg/dL
Postpartum management	Fasting glucose at 24-72 hr 2-hr 75-g GTT at 6- to 12-week visit

GCT = glucose challenge test; **GTT** = glucose tolerance test.

2-step approach for screening & diagnosing gestational diabetes mellitus



Diagnosis of gestational diabetes mellitus (≥2 abnormal values)		
Blood glucose level	**Carpenter & Coustan**	**NDDG**
Fasting	≥95 mg/dL (5.3 mmol/L)	≥105 mg/dL (5.8 mmol/L)
1 hr	≥180 mg/dL (10 mmol/L)	≥190 mg/dL (10.6 mmol/L)
2 hr	≥155 mg/dL (8.6 mmol/L)	≥165 mg/dL (9.2 mmol/L)
3 hr	≥140 mg/dL (7.8 mmol/L)	≥145 mg/dL (8 mmol/L)

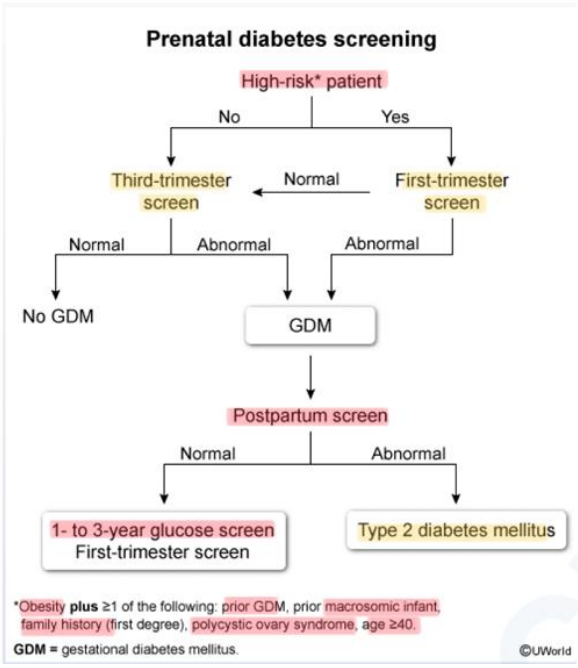
NDDG = National Diabetes Data Group criteria.

*Patients with risk factors (e.g. **obesity**, **previous GDM**) should be **screened earlier** and **re-screened** at 24-28 weeks if negative*

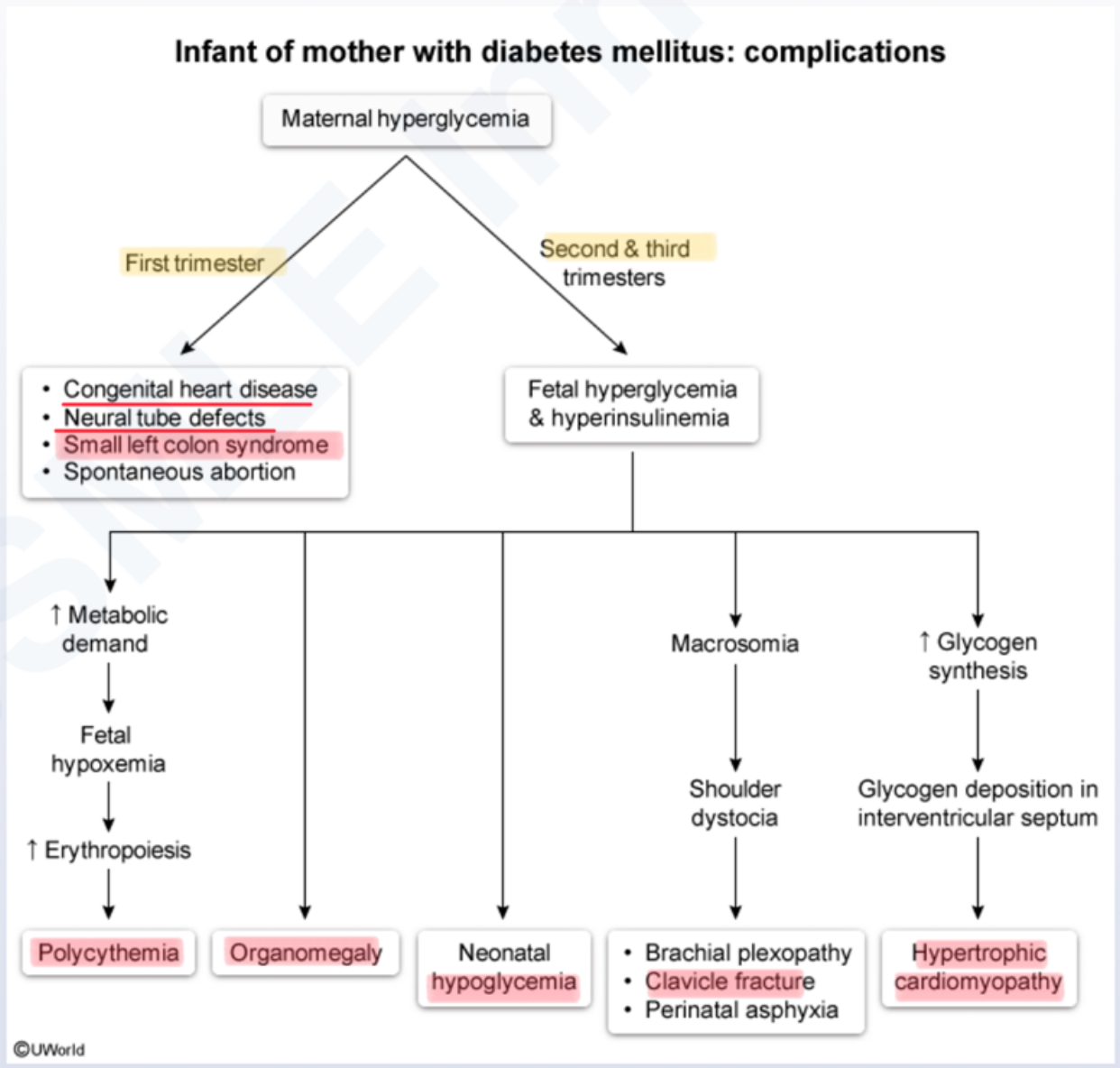
gestational diabetes mellitus (GDM), which is increasingly common due to the obesity epidemic. Other risk factors include GDM in a prior pregnancy and a prior macrosomic infant. All women require **screening at 24-28 weeks gestation** because GDM is associated with increased risk for gestational hypertension, preeclampsia, fetal macrosomia, and cesarean delivery. Patients with risk factors benefit from earlier screening (eg, early second trimester) and rescreening at 24-28 weeks if the initial screen is negative.

Patients diagnosed with GDM, such as this one, are monitored with serial **blood glucose level measurements** (ie, fasting and either 1- or 2-hr postprandial). Dietary modification (eg, low-carbohydrate diet, small meals with intermittent snacks) is the initial approach to management. However, patients with glucose levels consistently **above the target range** (ie, fasting >95 mg/dL, 1-hr >140 mg/dL, 2-hr >120 mg/dL), such as this patient, require additional pharmacotherapy because dietary modification alone has proved insufficient.

Insulin is first-line pharmacotherapy because it does not cross the placenta and dosing is easily adjustable. Oral antiglycemic medications (eg, metformin, glyburide) may be used in patients who decline insulin or have difficulty administering or adhering to insulin therapy.



All patients **at risk** for **type 2 DM** (eg, obesity, affected first-degree relative) require diabetic screening at the **initial prenatal visit** (eg, hemoglobin A1c, glucose challenge test).



*The fetus cannot produce insulin in the 1st trimester, thus hyperglycemia can disrupt **organogenesis***

The conditions that occur in the first trimester are called diabetic embryopathy.

While the ones that occur after that are called diabetic fetopathy.

Diabetic embryopathy

- **Definition**: any anomaly in an embryo associated with maternal diabetes, typically developing during the main embryonic period
- **Onset**: first trimester
- **Pathophysiology**: hyperglycemia → inhibition of myo-inositol uptake → abnormalities in the arachidonic acid-prostaglandin pathway → **congenital anomalies** and **early pregnancy loss** [14]

Manifestations of diabetic embryopathy [14]

- Early pregnancy loss and perinatal death
- **Cardiovascular defects**: congenital heart disease

And Central nervous system defects: neural tube defects

And genitourinary defects and

- **Skeletal defects**
  - Caudal regression syndrome: a congenital condition characterized by the partial or complete absence of the sacrum and often of the lower lumbar spine
    - Pathophysiology: The cause of caudal regression syndrome is unknown.
    - Maternal diabetes is a known risk factor.
    - Clinical features: based on the level of the spinal lesion and disease severity
      - Lower limb deformities or foot deformities (e.g., club foot)
      - Anorectal malformations
      - Aplasia or hypoplasia of the sacrum and/or lumbosacral spine
      - Mild to severe motor function impairment and paralysis
      - Flat buttocks and shallow gluteal clefts
      - Bowel and bladder dysfunction (e.g., neurogenic bladder, bladder incontinence)
    - May occur as part of other caudal syndromes (e.g., VACTERL, OEIS syndrome)
  - Vertebral anomalies (e.g., hemivertebrae)
- **Gastrointestinal defects**
  - Small left colon syndrome: an abrupt decrease in intestinal diameter characterized by transient intestinal obstruction due to inability to pass meconium
  - Duodenal atresia
  - Anorectal malformation
- **Other**: cleft palate

Diabetic fetopathy

- **Definition**: any anomaly in a fetus associated with maternal diabetes, caused by fetal hyperinsulinemia during gestation
- **Onset**: second and third trimesters
- **Pathophysiology**: maternal hyperglycemia → fetal hyperglycemia → stimulation of fetal pancreas → fetal hyperinsulinemia → ↑ metabolic rate, oxygen consumption, and fetal hypoxemia → metabolic, respiratory, and cardiovascular complications

Neonatal complications of diabetes during pregnancy	
Pathogenesis	• Maternal hyperglycemia → fetal hyperglycemia → β-cell hyperplasia & hyperinsulinemia • ↑ Fetal fat & glycogen stores • ↑ Fetal metabolic demand
Associated risks	• Prematurity • Congenital anomalies (eg, caudal regression syndrome) • Macrosomia & associated complications (eg, brachial plexus injury, clavicle fracture) • Respiratory distress syndrome • Hypertrophic cardiomyopathy
Laboratory findings	• Hypoglycemia • Polycythemia, low iron • Hypocalcemia & hypomagnesemia • Hyperbilirubinemia

Manifestations of diabetic fetopathy [14]

- **Growth defect**: fetal macrosomia
- **Metabolic defects**
  - Neonatal hypoglycemia: maternal hyperglycemia → fetal hyperglycemia → beta cell hypertrophy and hyperfunctioning → fetal and neonatal hyperinsulinemia → transient hypoglycemia after birth (when maternal glucose supply stops)
  - Neonatal polycythemia: maternal hyperglycemia → chronic fetal hyperglycemia → ↑ metabolic effects and oxygen demand → fetal hypoxemia → ↑ erythropoietin concentrations→ ↑ erythrocyte count
  - Neonatal hypocalcemia and neonatal hypomagnesemia: maternal hyperglycemia → abnormal maternal calcium-phosphorus metabolism → ↑ maternal urinary Mg excretion → maternal hypomagnesemia → fetal hypomagnesemia → impaired PTH synthesis in the fetus → fetal hypocalcemia and hypomagnesemia
- **Respiratory defects**
  - Acute respiratory distress syndrome
  - Transient tachypnea of the newborn
  - Perinatal asphyxia

• The pathophysiology of **hypocalcemia** in infants of diabetic mothera relates to maternal **hypomagnesemia**, which is caused by **osmotic diuresis** in the setting of poorly controlled gestational diabetes.

Low fetal magnesium concentrations, which reflect maternal levels, lead to **PTH suppression** and **low calcium levels** in the neonate.

💡 **Symptomatic hypocalcemia** can present with **jitteriness**

• What maternal pathology is associated with ***increased*** *risk* for **neonatal respiratory distress syndrome**?

Maternal diabetes

maternal diabetes results in fetal hyperinsulinism, which ***antagonizes cortisol*** *and* ***blocks maturation*** *of* ***sphingomyelin****.*

other risk factors include ***Prematurity,*** *male sex, perinatal asphyxia and C-section*

Hypertrophic cardiomyopathy in infants of diabetic mothers	
Pathogenesis	• Maternal hyperglycemia → fetal hyperglycemia & hyperinsulinemia • ↑ Glycogen & fat deposition in interventricular septum → dynamic LVOT obstruction
Clinical findings	• Often asymptomatic • May have respiratory distress and/or hypotension • Systolic ejection murmur
Imaging	• Chest x-ray: cardiomegaly • Echocardiogram: ↑ thickness of interventricular septum, ↓ LV chamber size
Treatment	• Intravenous fluids & beta blockers to increase LV blood volume
Prognosis	• Spontaneous regression by age 1
LVOT = left ventricular outflow tract.	

- **Cardiovascular defects**: transient hypertrophic cardiomyopathy
  - Definition: thickening of one or both of the ventricular walls and the interventricular septum
  - Clinical features: often asymptomatic in infants but may manifest with symptoms of heart failure (e.g., tachypnea, poor feeding, irritability)
  - Pathophysiology: maternal hyperglycemia → fetal hyperglycemia → fetal hyperinsulinemia → ↑ fat and glycogen in fetal myocardial cells → thickening of ventricular walls and the intraventricular septum in utero → ↓ ventricular size → left ventricular outflow obstruction and systolic and diastolic cardiac dysfunction
  - Diagnostics: echocardiography showing thickened ventricular walls and interventricular septum
  - Management: supportive care

Absolute contraindications to pregnancy	
Conditions in which pregnancy is contraindicated	• Pulmonary arterial hypertension • Peripartum cardiomyopathy with residual LV dysfunction • Heart failure with LVEF <30% • Severe coarctation • Severe mitral stenosis • Severe symptomatic aortic stenosis • Severe aortic dilation (eg, Marfan syndrome)
Management	• Recommend against pregnancy at preconception counseling visit • If pregnant, discuss abortion; if abortion declined, regular cardiology follow-up
LV = left ventricle; **LVEF** = left ventricular ejection fraction.	

Pregnancy contraindicated **UNLESS** the condition is fixed first.

Preterm Labour

Preterm labor	
Risk factors	• Prior spontaneous preterm delivery • Multiple gestation • Short cervical length • Cervical surgery (eg, cold knife conization) • Cigarette use
Screening & prevention	• Cervical length measurement by TVUS • Progesterone administration • Cerclage placement

Strongest: preterm delivery in a prior pregnancy

Definitions

- **Preterm labor**: regular uterine contractions with cervical effacement, dilation, or both before 37 weeks' gestation [1]
- **Preterm birth**
  - Live birth between 20 0/7 weeks' and 36 6/7 weeks' gestation
  - WHO subcategories [2]
    - Extremely preterm (< 28 weeks)
    - Very preterm (28 to < 32 weeks)
    - Moderate to late preterm (32 to < 37 weeks)

Clinical features

- Regular uterine contractions and associated symptoms of labor (e.g., lower back pain)
- Loss of mucus plug (bloody show)
- Cervical effacement and/or cervical dilation
- Rupture of membranes
- See "First stage of labor."

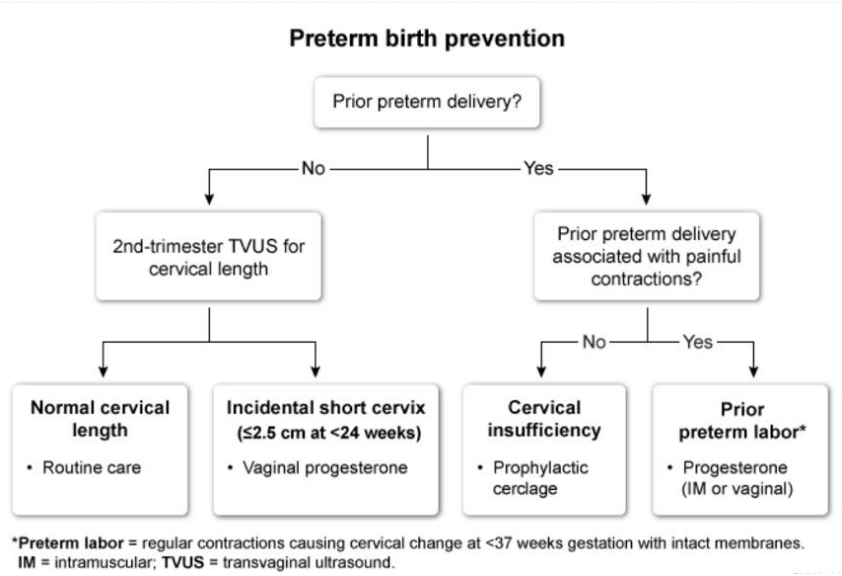
Diagnosis

The diagnosis of preterm labor is made clinically based on preterm contractions and cervical changes. The presence of risk factors for preterm labor can help establish the diagnosis. Fetal fibronectin levels and cervical length measurements can help assess the risk of impending delivery.

- Evaluate for clinical features of preterm labor.
- Perform sterile speculum examination.
  - Evaluate for cervical effacement and/or cervical dilation.
  - Assess for rupture of membranes.
- Cervicovaginal fetal fibronectin (fFN) test
  - Used to help differentiate between true preterm labor and false labor
  - Elevated levels in cervical secretions are associated with an increased risk of preterm delivery.
- Transvaginal ultrasound: **Cervical length** > 3 cm indicates a low likelihood of delivery within 14 days. [5]

A **negative fetal fibronectin test** prior to term is an indicator of **low risk** for **preterm delivery.**

***high negative predictive value**; fetal fibronectin levels are high until 20 weeks gestation and thus are not a reliable marker of preterm delivery in early pregnancy*



• What is the *first step* in evaluating risk of preterm delivery in a patient with a **history of cervical surgery** (e.g. cold knife conization)?

Transvaginal ultrasound measurement of **cervical length** in the **2nd trimester**

• What is the *recommended management* for a pregnant patient with no history of preterm labor and a **short cervix** (< 2 cm) on TVUS?

Vaginal progesterone

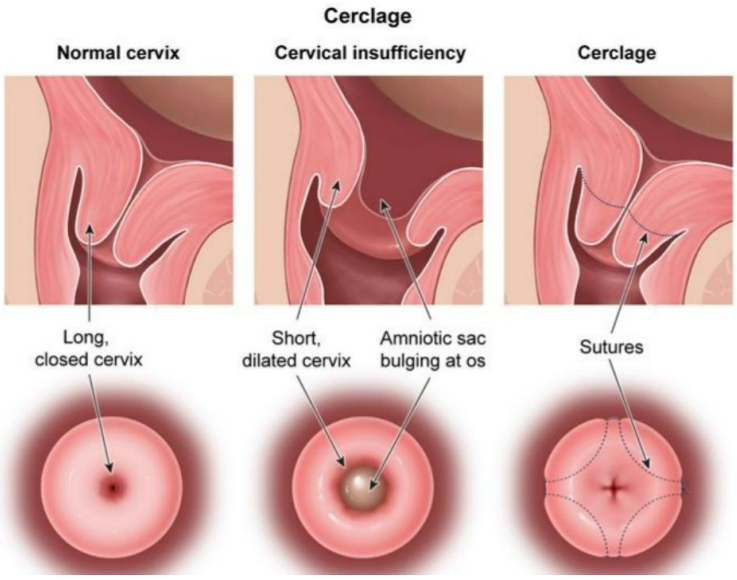
progesterone maintains uterine quiescence and protects against premature rupture of membranes

	Cervical insufficiency
Risk factors	Collagen defects Uterine abnormalities Cervical conization Obstetric injury
Clinical features	≥2 prior painless, 2nd-trimester losses Painless cervical dilation
Management	Cerclage placement

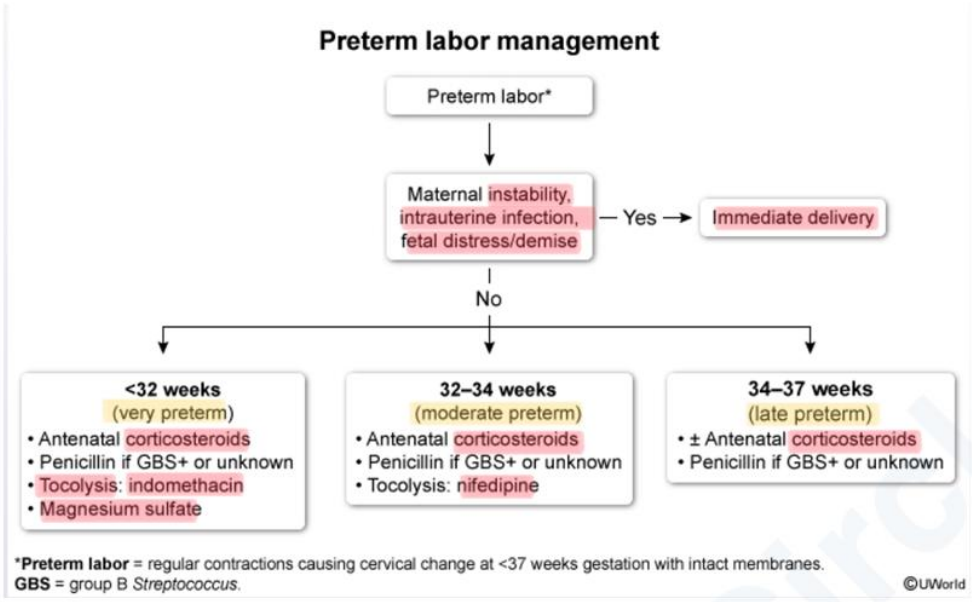
cervical insufficiency, a structural weakness of the cervix that predisposes to second-trimester pregnancy loss. It is diagnosed by any 1 of the following criteria:

- Painless cervical dilation in the current pregnancy (ie, examination-based) OR
- A second-trimester cervical length of ≤2.5 cm plus a prior preterm delivery (ie, ultrasound-based) OR
- **≥2 prior consecutive, painless, second-trimester losses** (ie, history-based), which typically present with mild symptoms (eg, vaginal discharge, light spotting) followed by precipitous delivery

Because of the high risk of recurrence, patients with **history-based cervical insufficiency** are treated with **prophylactic cerclage**, a procedure in which a suture is placed to reinforce and add tensile strength to the cervix. Prophylactic cerclage is performed in the first trimester (ie, 12-14 weeks gestation) in patients with a history of cervical insufficiency before it can recur in the second trimester. The suture is removed at term to allow vaginal delivery.



In patients who have **bulging** or **prolapsing amniotic membranes**, a rescue cerclage is **not** typically recommended due to the high risk of membrane rupture, complications (eg, intraamniotic infection, cervical trauma), and likely imminent delivery.



Magnesium sulfate given **<32 weeks** for **neuroprotection** if birth is indicated. (**Decreases** risk for **Cerebral Palsy**)

Tocolytics: ***Indomethacin*** *(preferred if given with magnesium)*, ***nifedipine, Terbutaline.***

- **Indomethacin C/I** after **32 weeks** due to risk of premature Fetal Ductus Arteriosus Closure.
- It can also cause **Decreased renal perfusion** and fetal oliguria can result in **oligohydramnios.** *Hence not given for more than **48 hours.***

• 30 year old female that has gone into preterm labor (32 weeks). The patient was given a drug to attempt to slow down the labor WHILE she is given appropriate pharmacotherapy to promote fetal lung development. An hour to 2 hours later the heart rate of the woman increases significantly. Mechanism?

Nifedipine causing arterial dilation → reflex tachycardia

Tocolysis [1][5][15]

Overview

- **Definition**: administration of tocolytics to inhibit uterine contractions [16]
- **Goal**: prolonging pregnancy to allow for induction of fetal lung maturity and/or transfer to another medical center, if necessary
- **Duration**: up to **48 hours**
- **Contraindications**
  - Maternal-specific drug contraindications
  - Nonreassuring fetal cardiotocography
  - Intrauterine fetal demise
  - Chorioamnionitis
  - Antepartum hemorrhage with hemodynamic instability
  - Severe preeclampsia or eclampsia
  - Lethal fetal anomaly

Medication	Maternal adverse effects	Fetal adverse effects
Nifedipine (calcium channel blocker)	• Hypotension • Dizziness • Flushing	• No known adverse effects
Indomethacin (NSAID)	• Esophageal reflux • Gastritis • Nausea and vomiting	• Renal dysfunction leading to oligohydramnios • Premature closure of the ductus arteriosus

Terbutaline (beta-2 adrenergic agonist)	• Potentially severe cardiovascular and metabolic conditions, e.g.:
	  ◦ Hypokalemia, hyperglycemia
	  ◦ Tachycardia, hypotension
	  ◦ Pulmonary edema
	  ◦ Myocardial ischemia
		• Tachycardia
		• Hypoglycemia

Magnesium sulfate	• Signs of magnesium toxicity, e.g.:
	  ◦ Hypocalcemia
	  ◦ Respiratory depression
	  ◦ Pulmonary edema
		• Possibly skeletal abnormalities with prolonged use [20]

Induction of fetal lung maturity [1][21]

- **Definition**: administration of antenatal steroids to promote the production of surfactant and thereby improve neonatal survival and fetal lung maturity
- **Indications**
  - Patients at 24 0/7 to 33 6/7 weeks' gestation
    - Initial course: all patients at risk of delivery within 7 days
    - Second course: patients at risk of delivery within 7 days whose prior course was > 14 days ago
- **Options**
  - Betamethasone
  - Dexamethasone

Fetal neuroprotection

- **Definition**: administration of antenatal magnesium sulfate to reduce the risk and severity of neurological disorders (e.g., cerebral palsy) [1]
- **Indication**: preterm labor at < 32 weeks' gestation [5]

Labour

Labour has three stages:

- The **first stage** is when the neck of the womb (cervix) opens to 10cm dilated.
- The **second stage** is when the baby moves down through the vagina and is born.
- The **third stage** is when the placenta (afterbirth) is delivered.

Stages of Normal Labor

- **Stage 1 Latent**: **0-6 cm**; <20 hr (nulli), <14 hr (multi)
- **Stage 1 Active**: **6-10cm**
- **Stage II**: **10cm → delivery; 3 hr (n), 2 hr (m)**
- **Stage III**: **Fetus → placental expulsion; < 30 min**

False labor versus latent labor		
Contractions	**False labor**	**Latent labor**
Timing	Irregular, infrequent	Regular, increasing frequency
Strength	Weak	Increasing intensity
Pain	None to mild	Yes
Cervical change	No	Yes

•**False labor:** *characterized by **mild**, **irregular contractions** (**Braxton Hicks** contractions) that cause no cervical change*

• What is the *recommended management* for patients that present in **false labor**?

Expectant management

Disorders of the active phase of labor		
Diagnosis	**Clinical features**	**Treatment**
Protraction	• Cervical change slower than expected • ± Inadequate contractions	Oxytocin
Arrest	• No cervical change for ≥4 hours with adequate contractions OR • No cervical change for ≥6 hours with inadequate contractions	Cesarean delivery

- *The **active stage** of labor typically begins at **> 6 cm dilation (≥1 cm dilation every 2 hour)***

  Protraction: ≤1 cm dilation in 2 hours

Adequate contractions are defined as generation of > **200 Montevideo units (MVUs)** in **10 minutes.**

*measured via an **intrauterine pressure catheter***

Second stage arrest of labor	
Definition	Insufficient fetal descent after pushing for: • ≥3 hours if nulliparous • ≥2 hours if multiparous
Risk factors	• Maternal obesity • Excessive pregnancy weight gain • Diabetes mellitus
Etiology	• Cephalopelvic disproportion • Malposition • Inadequate contractions • Maternal exhaustion
Management	• Operative vaginal delivery • Cesarean delivery

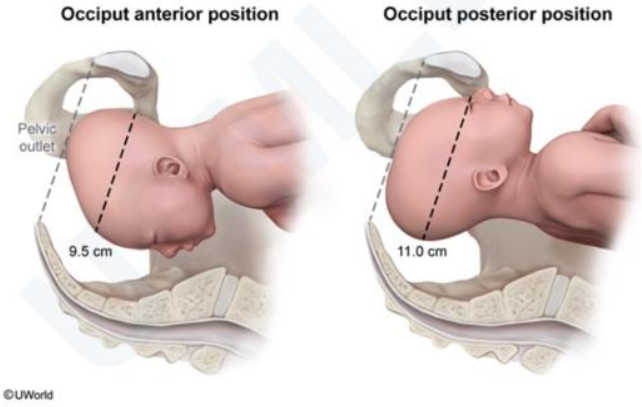
In order to **Operative Vaginal delivery** to occur, fetal head must be **fully engaged** i.e at **0 station**

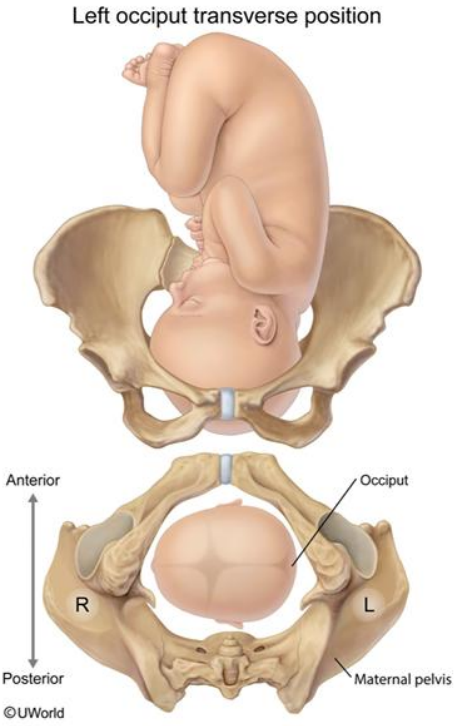
Neuraxial anesthesia (eg, epidural) can **lengthen the second stage** of labor **NOT first.**

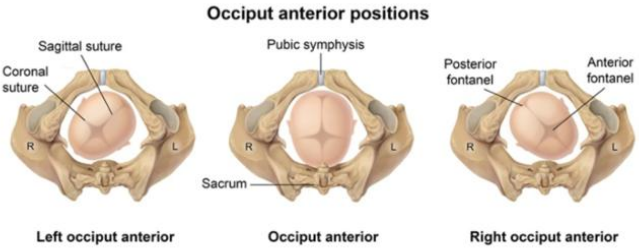
• What is the *most common cause* of **second stage arrest of labor**?

Fetal malposition (e.g. occiput transverse)

the optimal fetal position is ***Occiput anterior***







Rupture of membranes when there is **no palpable presenting fetal body part** or there is **fetal malpresentation** carries a high risk for **umbilical cord prolapse**, which can lead to **cord compression** and fetal hypoxia.

1st stage >> Latent phase abnormalities

PROLONGED
Nulliparous: **> 20 hours** (to reach 6 cm)
Multiparous: **> 14 hours** (to reach 6 cm)

Management:
Supportive, oxytocin, amniotomy, cervical ripening

2nd stage abnormalities

ARRESTED
No descent after pushing for:
Nulliparous: **> 3 hours** (> 4 hours if epidural)
Multiparous: **> 2 hours** (> 3 hours if epidural)

Management:
If baby is at 2+ station or more:
operative vaginal delivery *OR* c-section

1st stage >> Active phase abnormalities

PROLONGED
≥ 6 cm cervical dilation without adequate dilation **(< 1 cm / 2 hours)**

Management: Oxytocin, amniotomy, c-section

ARRESTED
ROM + **adequate** contractions + no cervical changes **> 4 hours**
OR
ROM + **inadequate** contractions + no cervical changes **> 6 hours**

Management: C-section

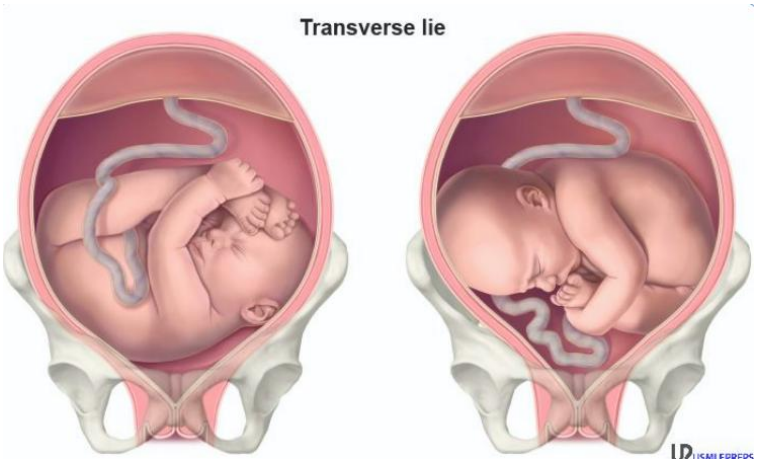
3rd stage abnormalities

Placenta has not been delivered > 30 minutes after birth

Management: - Uterotonic agents (IV/IM oxytocin)
- Controlled umbilical cord traction
- External compression / bimanual uterine massage

At an earlier gestational age, the fetus is mobile due to its small size when compared with the amniotic fluid volume and intrauterine cavity size. Therefore, abnormal fetal lie and malpresentation (eg, shoulder, breech) are common and frequently change.

With increasing gestational age, increased fetal growth and relative decreased amniotic fluid volume lead to decreased fetal mobility. By term (ie, ≥37 weeks gestation), the majority of fetuses will have **spontaneously rotated** into a longitudinal lie (ie, fetal spine is parallel to the long axis of the uterus) and cephalic presentation as this prepares the fetus for delivery and is the most effective way to occupy the intrauterine cavity. Therefore, patients with transverse lie and malpresentation at preterm gestations are **managed expectantly** with an ultrasound at term to evaluate fetal presentation.



• What is the *recommended management* for a patient at 28 weeks gestation with the fetus in **transverse lie position**? The patient desires a vaginal delivery.

Expectant management

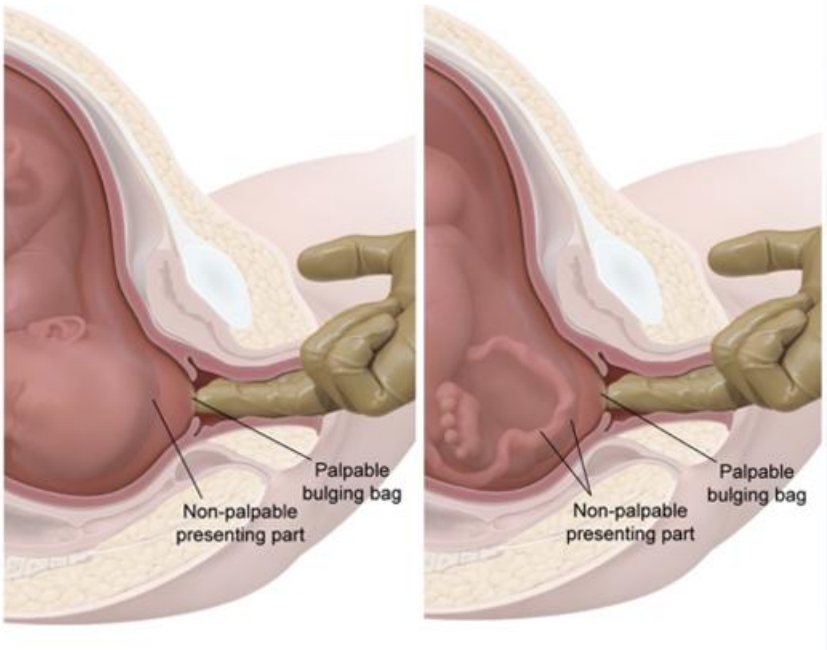
most fetuses in transverse lie ***spontaneously convert to vertex presentation*** *prior to term;*

persistent malpresentation may require ***external cephalic version*** *or* ***C-section***

Uterine surgical history & vaginal birth	
Surgery	**Trial of labor contraindicated?**
Low transverse cesarean delivery (horizontal incision)	No
Classical cesarean delivery (vertical incision)	Yes
Abdominal myomectomy with uterine cavity entry	Yes
Abdominal myomectomy without uterine cavity entry	No

• **Digital cervical examination** is used to determine cervical dilation and fetal presentation (ie, the fetal part [eg, head, buttocks] directly overlying the pelvic inlet)

If fetal presentation (eg, cephalic, breech) is uncertain on digital cervical examination, a **transabdominal ultrasound** should be performed to confirm fetal presentation and determine the safest route of delivery.



Amniotomy / Iatrogenic Rupture of Membranes are **NO** longer done.

The most commonly injured nerves in the **lithotomy position** (with feet in stirrups) are the **femoral nerve** and the **peroneal nerve**. The femoral nerve can be compressed under the inguinal ligament when the hip is flexed greater than 90 degrees, which can lead to numbness of the anterior and medial aspects of the thigh, as well as weakness of the quadriceps muscle.

Peroneal nerve injuries typically are caused by compression at the fibular head against the stirrup, leading to **foot drop** and **numbness over the dorsal foot** and lateral lower extremity

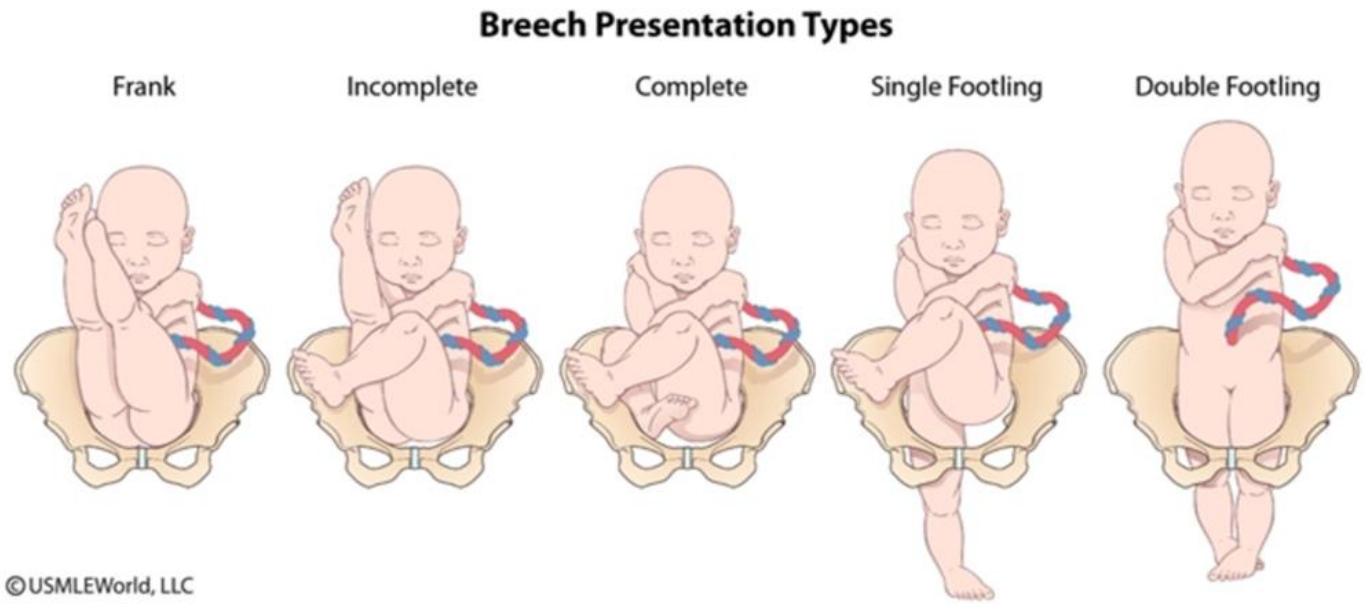
Breech:

	Breech presentation
Breech types	Frank: hips flexed & knees extended (buttock presenting) Complete: hips & knees flexed Incomplete: 1 or both hips not flexed (feet presenting)
Risk factors	Advanced maternal age (≥35) Multiparity Uterine didelphys, septate uterus Uterine leiomyomas Fetal anomalies (eg, anencephaly) Preterm (<37 weeks gestation) Oligohydramnios/polyhydramnios Placenta previa
Management	External cephalic version Cesarean delivery

Breech presentation occurs when the fetal buttocks or feet are the closest fetal part to the cervix. It is a common type of fetal malpresentation and has an increased risk of complications during vaginal delivery (eg, delivery trauma, umbilical cord prolapse, head entrapment). Variable fetal presentation, including breech, is normal during early pregnancy due to the small fetal size relative to the amniotic fluid volume; with increasing gestational age, the fetus typically develops a cephalic presentation, thereby optimizing chances of an uncomplicated vaginal delivery.

Fetal malpresentation (eg, breech) can occur when either fetal mobility or maternal anatomy (ie, fixation of presenting part in the pelvic inlet) is affected. A common **risk factor** for breech presentation (as seen in this patient) is **uterine leiomyomas**, particularly submucosal fibroids, as they can **distort the uterine cavity** and **limit fetal mobility**. In addition, large leiomyomas in the lower uterine segment can become space occupying and **prevent fetal cephalic engagement**.

Patients with breech presentation and no contraindications to vaginal delivery (eg, placenta previa, prior classical cesarean delivery) can be offered an external cephalic version (manual rotation of the fetus to cephalic presentation) at ≥36 weeks gestation.



• **Frank, Complete:** Can **try** Vaginal

• **Incomplete, Footling:** Vaginal **Contraindicated**

• What is the *recommended management* for a pregnant woman at **35 weeks gestation** that presents in **preterm labor** with the fetus in **breech presentation** on ultrasound?

Cesarean delivery

betamethasone +/- penicillin may be administered as well

• If **vertex presentation** on ultrasound?

Expectant management

> 💡 ***Active labor*** *is a* ***contraindication*** *for external cephalic version*

• What is the *management* of **Breech presentation/Transverse Lie**:

- **< 37 weeks** = **no intervention** as most fetuses spontaneously convert to cephalic
- **External cephalic version** = **>37 weeks, no active labor, no fetal distress** → **C-section** if fails

External Cephalic Version

External cephalic version	
Procedure	• Manual rotation of fetus to cephalic presentation • Decreases cesarean delivery rate
Indications	• Breech/transverse presentation • ≥37 weeks gestation

Absolute contraindications	• Contraindication to vaginal delivery
	  ◦ Prior classical cesarean delivery
	  ◦ Prior extensive uterine myomectomy
	  ◦ Placenta previa

Complications	• Abruptio placentae • Intrauterine fetal demise

Contraindications to external cephalic version
• Indications for cesarean delivery regardless of fetal lie (eg, failure to progress during labor, non-reassuring fetal status) • Placental abnormalities (eg, placenta previa or abruption) • Oligohydramnios • Ruptured membranes • Hyperextended fetal head • Fetal or uterine anomaly • Multiple gestation

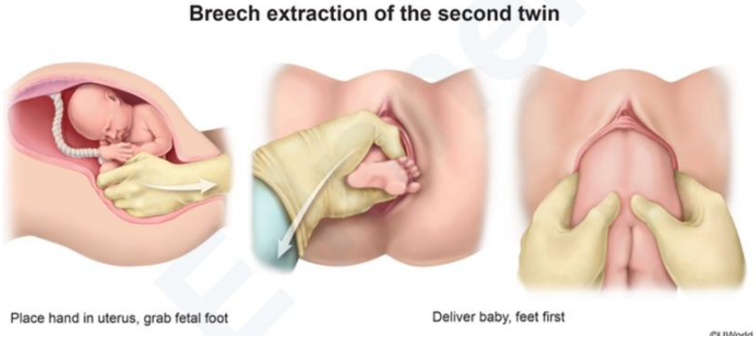
Active Labour

• What is the *next step* in management for a healthy pregnant woman at 37 weeks gestation that **desires a vaginal delivery**? Ultrasound reveals the fetus is in a **frank breech presentation**.

External cephalic version

can be attempted in women with breech pregnancies at ***> 37 weeks of gestational age*** *if there's no contraindications to vaginal delivery and the fetus is in good health*

Internal Podalic Version



Shoulder dystocia:

Shoulder dystocia	
Definition	• Failure of usual obstetric maneuvers to deliver fetal shoulders
Risk factors	• Fetal macrosomia • Maternal obesity • Excessive pregnancy weight gain • Gestational diabetes • Postterm pregnancy **>42 Weeks**
Warning signs	• **Protracted labor** • Retraction of fetal head into the perineum after delivery **(turtle sign)**

shoulder dystocia, or **impaction** of the fetal **anterior shoulder** behind the maternal pubic symphysis that impairs delivery of the shoulders with routine gentle traction. Risk factors include gestational diabetes mellitus (as in this patient) and associated fetal macrosomia (ie, birth weight ≥4.5 kg [9 lb 14 oz]). Shoulder dystocia is an **obstetric emergency** that can lead to both fetal and maternal complications. Fetal complications include brachial plexus injuries, clavicular or humeral fractures, and hypoxic encephalopathy. Maternal complications include fourth-degree (ie, rectal mucosa) perineal lacerations and postpartum hemorrhage.

	Complications of shoulder dystocia
Fractured clavicle	Clavicular crepitus/bony irregularity ↓ Moro reflex due to pain on affected side Intact biceps & grasp reflexes
Fractured humerus	Upper arm crepitus/bony irregularity ↓ Moro reflex due to pain on affected side Intact biceps & grasp reflexes
Erb-Duchenne palsy	↓ Moro & biceps reflexes on affected side Waiter's tip ◦ Extended elbow ◦ Pronated forearm ◦ Flexed wrist & fingers Intact grasp reflex
Klumpke palsy	Clawhand ◦ Extended wrist ◦ Hyperextended metacarpophalangeal joints ◦ Flexed interphalangeal joints ◦ Absent grasp reflex Horner syndrome (ptosis, miosis) Intact Moro & biceps reflexes
Perinatal asphyxia	Variable presentation depending on duration of hypoxia Altered mental status (eg, irritability, lethargy), respiratory or feeding difficulties, poor tone, seizure

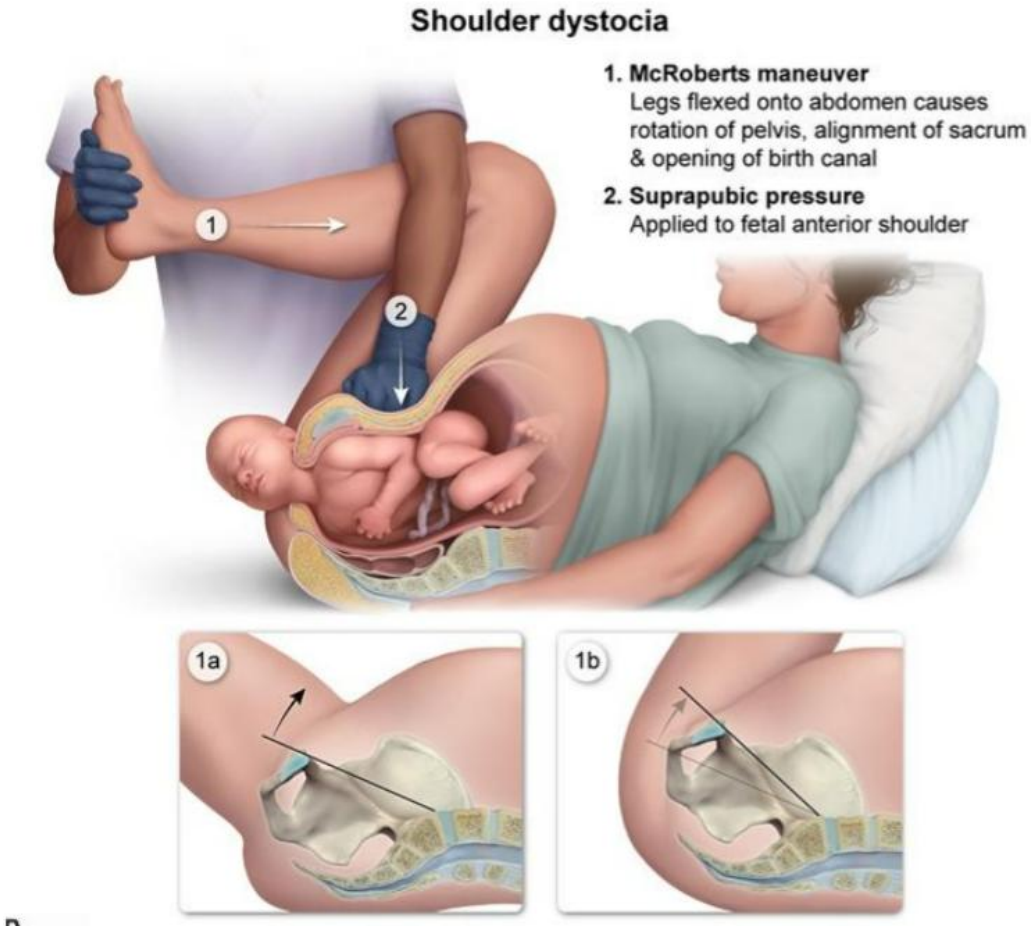
• In Erb & Klumpke Palsy, Management involves **observation alone** because most infants recover arm function spontaneously within a **few months.**

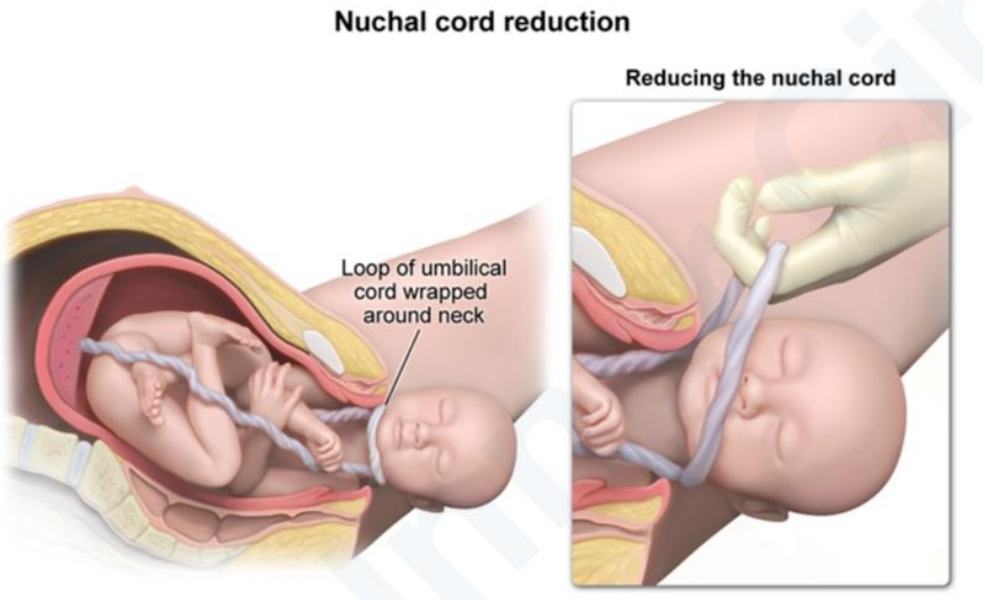
The Moro reflex is a normal reflex for an infant when he or she is startled or feels like they are falling. The infant will have a startled look and the arms will fling out sideways with the palms up and the thumbs flexed. Absence of the Moro reflex in newborn infants is abnormal and may indicate an injury or disease.

Management of shoulder dystocia (BE CALM)	
B	Breathe; do not push
E	Elevate legs & flex hips, thighs against abdomen (McRoberts)
C	Call for help
A	Apply suprapubic pressure
L	En**L**arge vaginal opening with episiotomy
M	Maneuvers: Deliver posterior arm Rotate posterior shoulder (Woods screw): apply pressure to anterior aspect of the posterior shoulder Adduct posterior fetal shoulder (Rubin): apply pressure to the posterior aspect of the posterior shoulder Mother on hands & knees: "all fours" (Gaskin) Replace fetal head into pelvis for cesarean delivery (Zavanelli)

Management of shoulder dystocia requires maneuvers to dislodge the anterior shoulder or reorient the fetus to deliver through the widest diameter of the bony pelvis. The initial step in relieving shoulder dystocia is the **McRoberts maneuver**—flexing the patient's hips back against her abdomen—and applying **suprapubic pressure**. The McRoberts maneuver flattens the sacral

promontory and decreases obstruction through the bony pelvis. Suprapubic pressure may then dislodge the anterior shoulder to allow delivery. The combination of these maneuvers relieves shoulder dystocia in nearly half of cases without further intervention.





A **nuchal cord** is a **common, usually harmless loop of umbilical cord around the fetal neck** that is easily managed at delivery. While it can sometimes cause **intrapartum heart rate decelerations** due to compression, the vast majority of babies are born healthy without any sequelae. Its discovery on ultrasound is not a reason for intervention, and its presence at delivery is a routine part of obstetrical practice.

During **shoulder dystocia**, clamping and **cutting the umbilical cord** (even a nuchal cord) is **absolutely contraindicated** because it would sever the only source of oxygen to the fetus, resulting in fetal hypoxia, hypoxic encephalopathy, and fetal **death**.

Replacement of the fetal head into the maternal pelvis (ie, Zavanelli maneuver) is performed if all other maneuvers fail and an emergency cesarean delivery must be performed.

	Late- & post-term pregnancy
Definition	Late-term: ≥41 weeks gestation Post-term: ≥42 weeks gestation
Risk factors	Prior post-term pregnancy Nulliparity Obesity Age ≥35 Fetal anomalies (eg, anencephaly)
Complications	Fetal/neonatal ◦ Macrosomia ◦ Dysmaturity syndrome ◦ Oligohydramnios ◦ Demise Maternal ◦ Severe obstetric laceration ◦ Cesarean delivery ◦ Postpartum hemorrhage
Management	Frequent fetal monitoring (eg, nonstress test) Delivery prior to 43 weeks gestation

Late-term and postterm pregnancy complications	
Fetal	**Maternal**
• Oligohydramnios • Meconium aspiration • Stillbirth • Macrosomia • Convulsions	• Cesarean delivery • Infection • Postpartum hemorrhage • Perineal trauma

Placental sulfatase deficiency is a risk factor for **post**-term delivery.

*other risk factors include **fetal adrenal hypoplasia** and **anencephaly***

3rd Trimester Bleeding:

The causes can be classified into: maternal and fetal -the only fetal one is vasa previa-.

	Placenta previa
Risk factors	Prior placenta previa Prior cesarean delivery Multiple gestation
Clinical features	Painless vaginal bleeding >20 weeks gestation
Diagnosis	Transabdominal followed by transvaginal sonogram
Management	No intercourse No digital cervical examination Inpatient admission for bleeding episodes

placenta previa, a condition in which the **placenta covers the cervix**. This patient's risk factors for placenta previa include prior cesarean delivery, multiparity, and smoking; additional risk factors include prior placenta previa and multiple gestation. Placenta previa has the potential for massive antepartum hemorrhage because cervical manipulation (even minimal manipulation from intercourse) causes partial placental detachment and **painless vaginal bleeding** (eg, nontender uterus, painless irregular contractions) from the intervillous space. This bleeding is primarily of maternal origin; therefore, many patients have **reassuring fetal monitoring** initially (eg, accelerations, no decelerations).

Treatment depends on maternal hemodynamic stability and fetal status. Stable patients are managed expectantly, as most previas resolve by the third trimester. Patients with persistent previa undergo cesarean delivery at 36-37 weeks gestation (prior to the onset of labor).

T/t:

- Stable with mild bleed: **IV fluids & Observation**
- Unstable or acti: **Immediate C/S**

> 💡 Patients who present with vaginal bleeding after 20 weeks gestation **should not** undergo a Digital Cervical examination **until USG** has been performed to determine the exact location of placenta. *(Risk of causing massive haemorrhage if it is placenta previa)*

- What *recommendations* should you make for diagnosis of **placenta previa**?

  Pelvic rest, abstinence from intercourse

  Treatment? **Schedule C/S** at **36/37 weeks**

- The **majority** (**~90%**) of cases **resolve spontaneously** due to lower uterine segment lengthening and/or placental growth toward the fundus; therefore, initial management is with **routine obstetric care**.

	Abruptio placentae
Definition	Placental detachment from the uterus before fetal delivery
Risk factors	Hypertension, preeclampsia Abdominal trauma Prior abruptio placentae Cocaine & tobacco use
Clinical presentation	Sudden-onset vaginal bleeding Abdominal or back pain High-frequency, low-intensity contractions Rigid, tender uterus
Diagnosis	Clinical Ultrasound: ± Retroplacental hematoma
Complications	Fetal hypoxia, preterm birth, mortality Maternal hemorrhage, disseminated intravascular coagulation

abruptio placentae, the premature detachment of the placenta from the uterus due to bleeding at the uteroplacental interface.

Patients with **uterine overdistension** (eg, twin gestation, **severe polyhydramnios**) are at increased risk for abruptio placentae because the sudden, uncontrolled loss of amniotic fluid causes **rapid uterine decompression**, which shears maternal placental vessels and causes placental hemorrhage and separation. The bleeding also increases intrauterine pressure, which causes constant pain, a **rigid, tender uterus**, and uterine irritability (ie, high-frequency contractions).

As more of the placenta detaches, fetal perfusion progressively declines and can lead to hypoxia. Therefore, patients with persistent bleeding and an abnormal fetal heart rate tracing (eg, bradycardia, minimal variability) require urgent delivery to prevent fetal demise and maternal complications (eg, hemorrhage, disseminated intravascular coagulation).

- **Regular, High frequency contractions** *(vs **irregular** and **decreasing** in **Uterine Rupture**)*

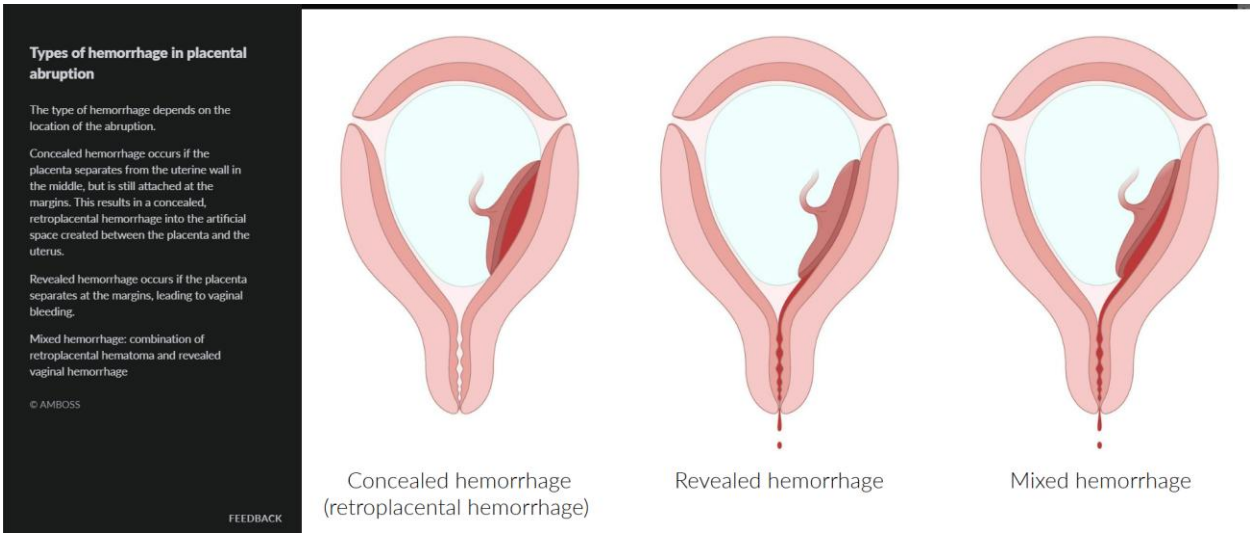
Occurs after **20 weeks gestation.**

Uterine tachysystole (eg, **>5 contractions in a 10 minute period**).

• What is the *initial management* for a hemodynamically unstable pregnant patient that presents with **abruptio placentae** following a motor vehicle accident?

IV fluid resuscitation and left lateral decubitus positioning

left lateral decubitus position displaces the uterus off the aortocaval vessels and maximizes cardiac output



Uterine Rupture

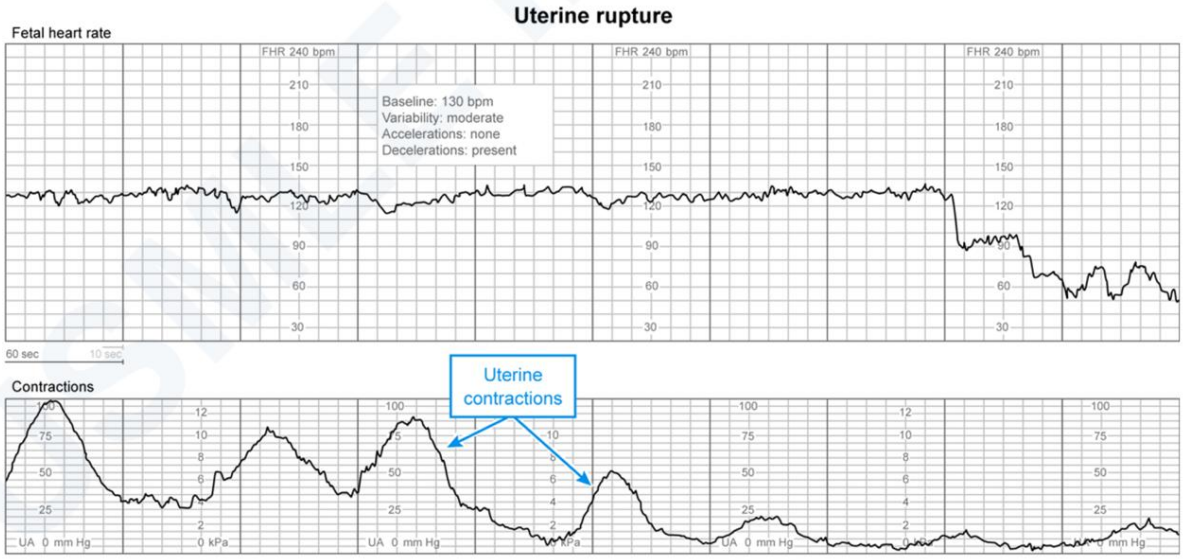
• What is *most important* risk factor for **uterine rupture**?

Previous C-section or myomectomy (*scar becomes weakest point of uterus*)

Presentation?

- **Painful 3rd trimester bleeding, sudden abdominal pain during labour**
- **Loss of fetal station (***presenting fetal part retracts***)**
- **Palpable fetal parts** on abdominal exam
- **Fetal heart decels (bradycardia)**

Laboring patients at *high risk* of **uterine rupture** require **urgent laparotomy and delivery**



Progressively decreasing contraction amplitude: **Staircase Sign**

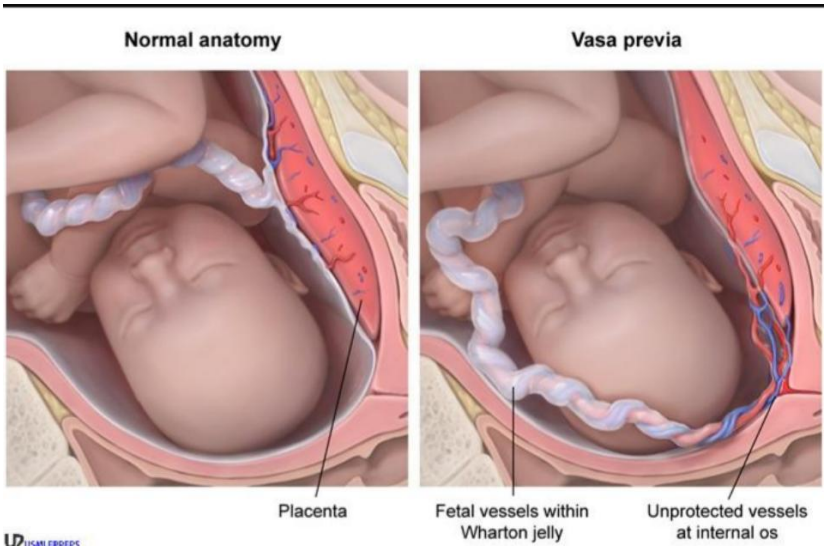
VASA PREVIA:

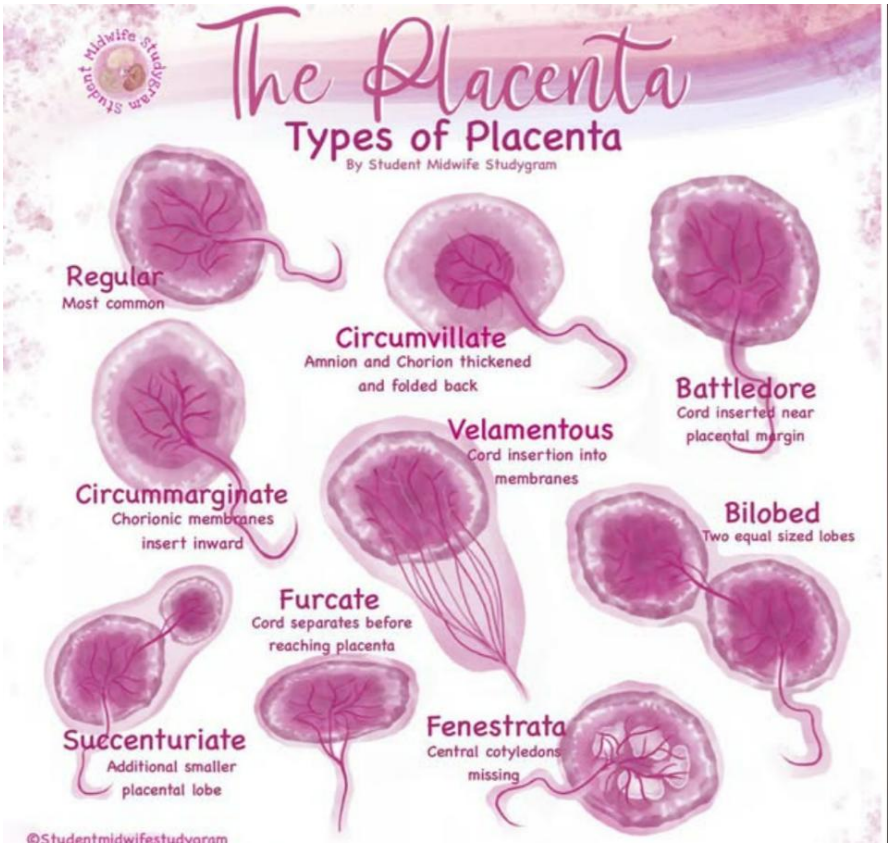
	Vasa previa
Definition	Fetal vessels overlying the cervix
Risk factors	Placenta previa Multiple gestations In vitro fertilization Succenturiate placental lobe
Clinical presentation	Painless vaginal bleeding with ROM or contractions FHR abnormalities (eg, bradycardia, sinusoidal pattern) Fetal exsanguination & demise
Management	Emergency cesarean delivery

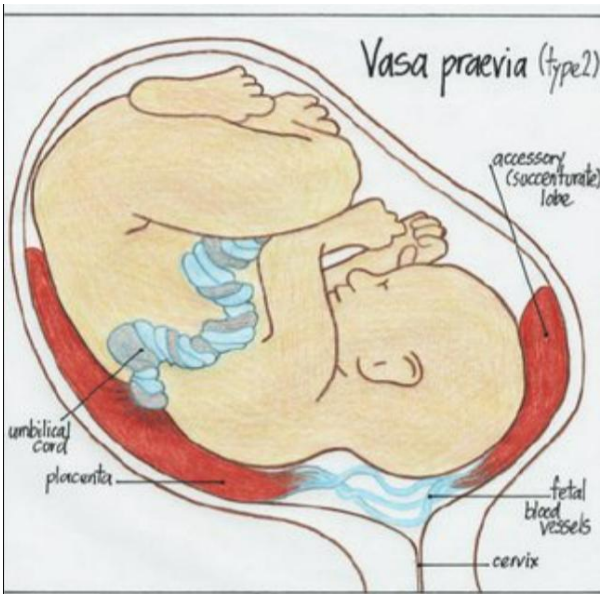
Normal fetal vessels travel in the umbilical cord surrounded by thick, gelatinous tissue (ie, Wharton jelly) that protects them. In contrast, **vasa previa** is an aberrant condition in which the **fetal vessels overlie the cervix**, surrounded only by thin fetal membranes, making them prone to **tear** with **rupture of membranes** or contractions. Risk factors include in vitro fertilization, as in this patient, and placenta previa.

Vasa previa is typically diagnosed on fetal anatomy ultrasound at 18-20 weeks and managed with planned cesarean delivery at 34-35 weeks gestation (ie, prior to spontaneous labor). Because total fetal blood volume is low (eg, ~250 mL or 1 cup), even minimal fetal bleeding can lead to rapid **exsanguination** and **fetal demise**. Therefore, these patients require third-trimester inpatient management to monitor for acute changes that require immediate delivery.

If a patient is a known case of vasa previa and present with ROM this will cause the fetal vessels to bleed and the bleeding will be very slight but it will cause a threat to the fetal life thus this case is considered an emergency and requires **emergency cesarean delivery**.


Normal anatomy
Vasa previa
Placenta
Fetal vessels within Wharton jelly
Unprotected vessels at internal os


The Placenta
Types of Placenta
By Student Midwife Studygram
Regular
Most common
Circumvillate
Amnion and Chorion thickened and folded back
Battledore
Cord inserted near placental margin
Velamentous
Cord insertion into membranes
Circummarginate
Chorionic membranes insert inward
Bilobed
Two equal sized lobes
Furcate
Cord separates before reaching placenta
Succenturiate
Additional smaller placental lobe
Fenestrata
Central cotyledons missing
@Studentmidwifestudygram


Vasa praevia (type 2)
accessory (succenturiate) lobe
umbilical cord
placenta
fetal blood vessels
cervix

PROM:

	Preterm prelabor rupture of membranes (PPROM)
Definition	Membrane rupture at <37 weeks prior to labor onset
Risk factors	Prior PPROM Genitourinary infection (eg, ASB, BV) Antepartum bleeding
Diagnosis	Vaginal pooling or fluid from cervix Nitrazine-positive (blue) fluid Ferning on microscopy
Management	<34 weeks (reassuring): latency antibiotics, corticosteroids <34 weeks (nonreassuring): delivery ≥34 weeks: delivery
Complications	Preterm labor Intraamniotic infection Placental abruption Umbilical cord prolapse

Abnormal rupture of membranes

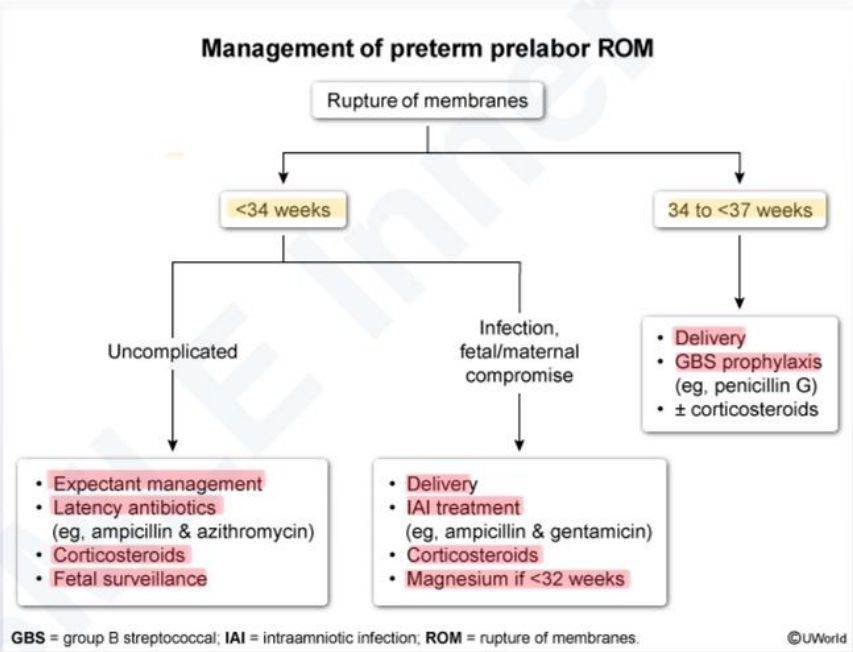
Definitions [26]

- Prelabor rupture of membranes (PROM): rupture of membranes before the onset of labor at ≥ 37 weeks' gestation
- Preterm prelabor rupture of membranes (PPROM): rupture of membranes before the onset of labor and before 37 weeks' gestation
- Prolonged rupture of membranes: rupture of membranes > 18 hours before the onset of labor [27]

Diagnostics for abnormal rupture of membranes [26]

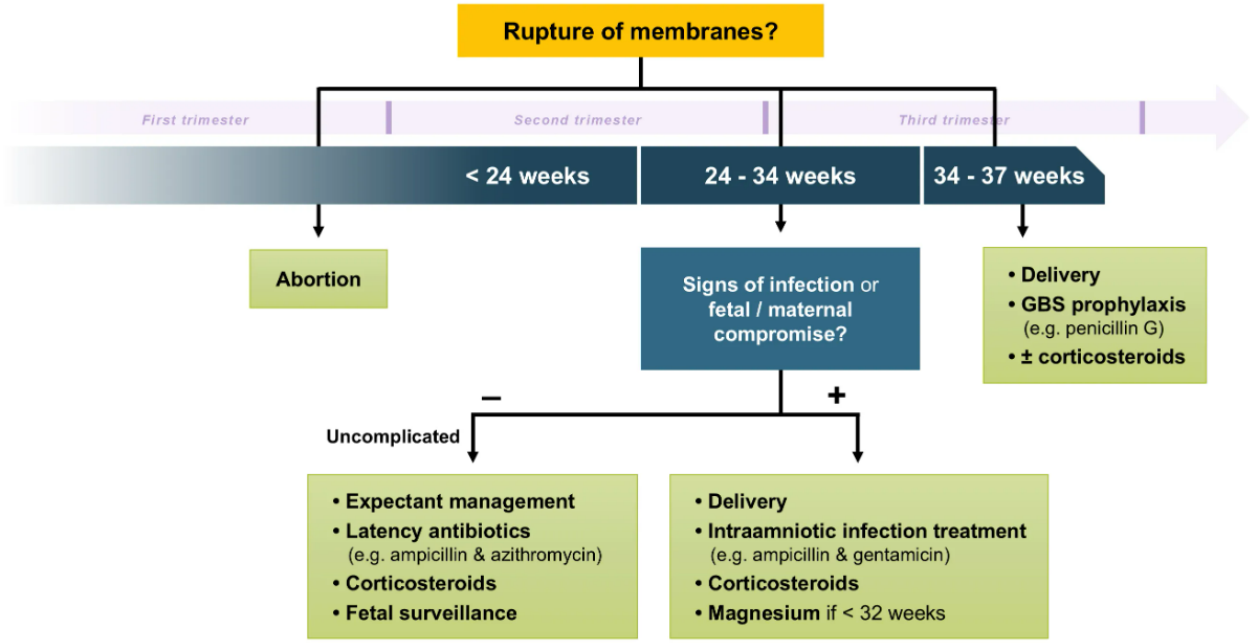
- **Clinical diagnosis**: a sudden gush of pale yellow or clear fluid from the vagina
- **Sterile speculum examination**
  - Fluid exiting the cervix and pooling in the vaginal fornix suggests rupture of membranes.
  - Detection of amniotic fluid
    - Litmus test or nitrazine test: Test strips turn blue, as amniotic fluid is alkaline.
    - Positive fern test: fern pattern on glass slide
- **Ultrasound**: Oligohydramnios may be present.

Do not perform a digital cervical examination unless the patient is in active labor because it increases the risk of infection and has minimal diagnostic utility. [26]



Tocolysis is **NOT indicated** in PROM. It is only used to preterm labour if membranes have not yet ruptured.

Management of preterm prelabor rupture of membranes (PPROM)

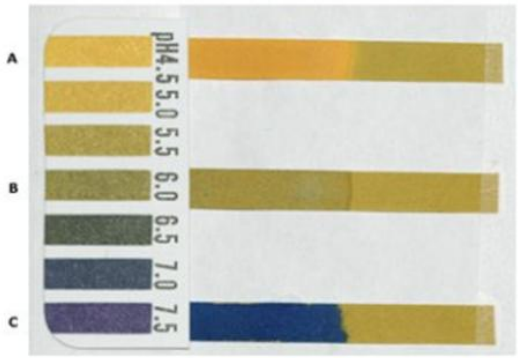


PPROM antibiotic prophylaxis

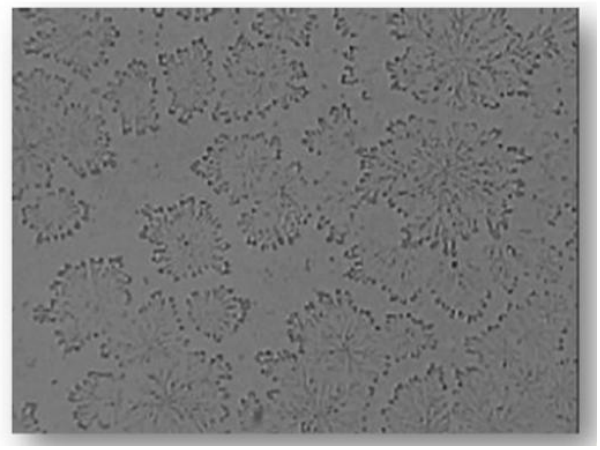
- **Goals**
  - Prolong pregnancy
  - Reduce the risk of maternal and neonatal infections
- **Treatment**
  - Initial 48 hours: ampicillin PLUS erythromycin IV
  - Subsequent 5 days: amoxicillin PLUS erythromycin PO
  - Alternative: A single dose of azithromycin may replace the 7-day course of erythromycin.

Test

- Nitrazine test
  - Fluid from vaginal exam placed on strip of nitrazine paper
  - Paper turns blue in presence of alkaline (pH > 7.1) amniotic fluid



- Fern test
  - Fluid from vaginal exam placed on slide and allowed to dry
  - Amniotic fluid narrow fern vs. cervical mucus broad fern



• What is the likely *diagnosis* in an obese, multiparous pregnant woman at 34 weeks gestation that presents with **intermittent leakage of clear fluid** and a **negative nitrazine/fern test?**

Stress urinary incontinence

A small **pool of urine in the posterior vagina** may be seen on speculum examination because urine can become trapped in the vagina (ie, **retrograde vaginal voiding**) during pregnancy

differentiated from rupture of membranes by ***negative nitrazine/fern tests*** *and* ***absence of vaginal pooling***

Complications [26][28]

- PROM: intrauterine infection (risk increases with duration of ruptured membranes)
- PPROM
  - Fetal
    - Complications of prematurity (e.g., neonatal respiratory distress, necrotizing enterocolitis)
    - Umbilical cord compression
    - Antepartum death
  - Maternal
    - Chorioamnionitis
    - Placental abruption

Chorioamnionitis:

Intraamniotic infection (chorioamnionitis)	
Risk factors	• Prolonged rupture of membranes (>18 hours) • Preterm prelabor rupture of membranes • Prolonged labor • Internal fetal/uterine monitoring devices • Repetitive vaginal examinations • Presence of genital tract pathogens
Diagnosis	Maternal fever PLUS ≥1 of the following: • Fetal tachycardia (>160/min) • Maternal leukocytosis • Purulent amniotic fluid
Management	• Broad-spectrum antibiotics • Delivery
Complications	• Maternal: postpartum hemorrhage, endometritis • Neonatal: preterm birth, pneumonia, encephalopathy

Deliver regardless of age

Definitions

- **Intra-amniotic infection** (chorioamnionitis) [1][2]
  - Confirmed microbial invasion of the amniotic cavity causing infection and inflammation of the fetus, amniotic fluid, fetal membranes, placenta, and/or endometrial decidua
  - Usually only diagnosed after delivery, from studies performed on amniotic fluid or placenta
- **Clinical chorioamnionitis:** a clinical syndrome comprising maternal fever and signs of maternal and/or fetal inflammation, usually caused by intra-amniotic infection or, less commonly, sterile intra-amniotic inflammation [2]

Although intra-amniotic infection and clinical chorioamnionitis are distinct entities, they cannot usually be distinguished from one another before delivery. [2]

A **low** amniotic fluid glucose is an *indicator* of **intra-amniotic infection**

Clinical features

The following features indicate clinical chorioamnionitis. [1][2][5]

- Fever
- Tachycardia
- Uterine tenderness
- Malodorous amniotic fluid
- Purulent cervical discharge
- Fetal tachycardia
- Maternal or neonatal complications of intra-amniotic infection

Fever is not always present in intra-amniotic infection. [5]

Criteria for suspected intra-amnitoic infection [1][2][5]

- **Maternal temperature 38.0–38.9°C** and ≥ 1 of the following:
  - **Maternal leukocytosis > 15,000 cells/µL**
  - **Purulent cervical drainage**
  - **Fetal tachycardia > 160 beats/minute**
- OR isolated maternal temperature ≥ 39.0 °C [1]
- OR clinical features of intra-amniotic infection in the absence of maternal fever [5]

Empiric antibiotic therapy for intra-amniotic infection [1][7]

- First-line therapy
  - **Ampicillin** [1]
  - PLUS **gentamicin** [1]
  - Following cesarean delivery
    - At least one additional dose of ampicillin and gentamicin
    - PLUS **clindamycin** OR metronidazole
- Nonsevere penicillin allergy [1]
  - Cefazolin [1]
  - PLUS gentamicin
- Severe penicillin allergy [1]
  - Clindamycin OR vancomycin [1]
  - PLUS gentamicin

• Management?

- **IV ampicillin + gentamicin** (+ **Clindamycin** for C/S)
- Immediate delivery via **augmentation of labour**
- **C/S generally not indicated** unless of obstetrical complications (non-reassuring tracings, breech)

• What is the *next step* in management for a pregnant woman at 38 weeks gestation with chorioamnionitis after **administration of antibiotics**? Fetal heart tracing reveals tachycardia but is otherwise reassuring.

Induction of labor

cesarean delivery is reserved for standard obstetric indications (e.g. prior uterine surgeries, breech presentation, non-reassuring fetal heart tracing)

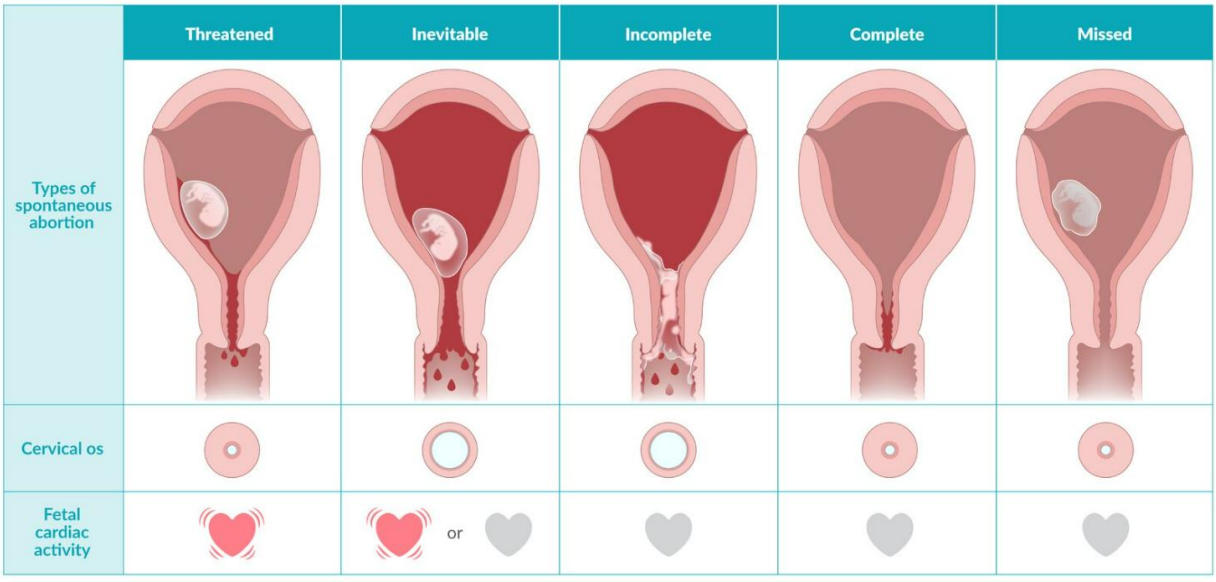
intraamniotic infection (IAI) (ie, chorioamnionitis), a fulminant polymicrobial infection of the amniotic sac, fetus, cord, and placenta from ascending vaginal flora. Additional signs may include maternal tachycardia, **uterine tenderness**, and malodorous or **purulent amniotic fluid**. IAI has an increased risk of maternal morbidity (eg, sepsis, disseminated intravascular coagulation) and mortality that outweighs the fetal benefit gained by prolonging pregnancy. Therefore, patients with PPROM complicated by IAI require **therapeutic antibiotics and immediate delivery** (eg, induction of labor), regardless of gestational age.

Miscarriage/ Abortion:

- **Spontaneous abortion** = loss **< 20 weeks**
- **Stillbirth** (*intrauterine fetal demise*) = loss **after 20 weeks**

Types of pregnancy loss [1]

Type [2][3]	Definition	Findings	Treatment
Threatened abortion	• An abortion process starting before 20 weeks' gestation that has not progressed to a state from which recovery is impossible (potentially reversible)	• **Vaginal bleeding** • **Fetal cardiac activity** • Closed cervical os	• **Expectant management** • Patient should avoid strenuous physical activity. • Weekly pelvic ultrasound • Rule out **treatable causes** of vaginal bleeding. • **Anti-D immunoglobulin** should be considered for Rh(D)-negative women.
Inevitable abortion	• A condition of vaginal bleeding and cervical dilation without expulsion of products of conception (POC) that occurs before 20 weeks' gestation • Typically followed by partial or complete passage of POC	• **Vaginal bleeding**, and **visible/palpable products of conception** • Fetal cardiac activity may be present. • **Dilated** cervical os	• Depends mostly on **patient preference** • **Expectant management** (option for women ≤ 12 weeks gestation) • Medical evacuation: combination of **mifepristone and misoprostol** • **Surgical evacuation**: if spontaneous evacuation does not occur or in cases of **septic abortion** or **heavy bleeding**
Missed abortion	• Death of a fetus before 20 weeks' gestation without expulsion of any POC	• **No bleeding** • **No expulsion** of the products of conception • **No fetal cardiac activity** • **Closed** cervical os	
Incomplete abortion	• Passage of some but not all POC before 20 weeks' gestation	• **Vaginal bleeding**; products of conception within the **cervical canal or uterus** • **Dilated** cervical os	
Complete abortion	• The complete passage of all POC before 20 weeks' gestation	• **Vaginal bleeding**; products of conception **completely outside of the uterus** • **Closed** cervical os	• No treatment required
Stillbirth	• Loss of pregnancy **after 20 weeks'** gestation (also called intrauterine fetal demise)	• **Absence of fetal movements and cardiac activity** • Cervical os variable	• Spontaneous labor usually begins within **2 weeks** of intrauterine fetal death. • Vaginal delivery is safer than cesarean delivery



Features of spontaneous pregnancy loss [3]

Type	Vaginal bleeding	Fetal cardiac activity	Products of conception (POC)	Cervical os
Threatened abortion	• Yes	• Yes	• Intrauterine	• Closed
Inevitable abortion	• Yes	• May be present	• **Visible/palpable POC**	• **Dilated**
Missed abortion	• No	• No	• **No expulsion** of the POC	• **Closed**
Incomplete abortion	• Yes	• No	• POC within the **cervical canal or uterus**	• **Dilated**
Complete abortion	• Yes	• No	• POC **completely outside of the uterus**	• **Closed**

- **Missed Abortion**

  *Patients are typically asymptomatic or have loss of pregnancy symptoms (e.g. **nausea**, **breast tenderness**) with **decreasing beta-hCG levels***

  USG findings: **No Cardiac activity**, **empty gestational sac** without **a fetal pole**

- **Complete Abortion**

  *other findings include **Unilateral Ovarian Cyst** (**Corpus luteum**) and some **fluid in the pelvis.***

  B-HCG can take **6 weeks** to go undetectable

Spontaneous abortion	
Definition	• Pregnancy loss <20 weeks
Risk factors	• Advanced maternal age • Previous spontaneous abortion • Substance use disorder
Treatment options	• Expectant • Medical induction (misoprostol) • Suction curettage if infection or hemodynamic instability
Additional management	• Rho(D) immunoglobulin • Pathology examination
Complications	• Hemorrhage • Retained products of conception • Septic abortion • Uterine perforation • Intrauterine adhesions

💡 **Spontaneous abortion** is most often due to chromosomal **trisomies**, especially **trisomy 16**

Spontaneous abortion is the general term used to describe loss of fetus before 20 weeks of gestation and the types and threatened inevitable etc.

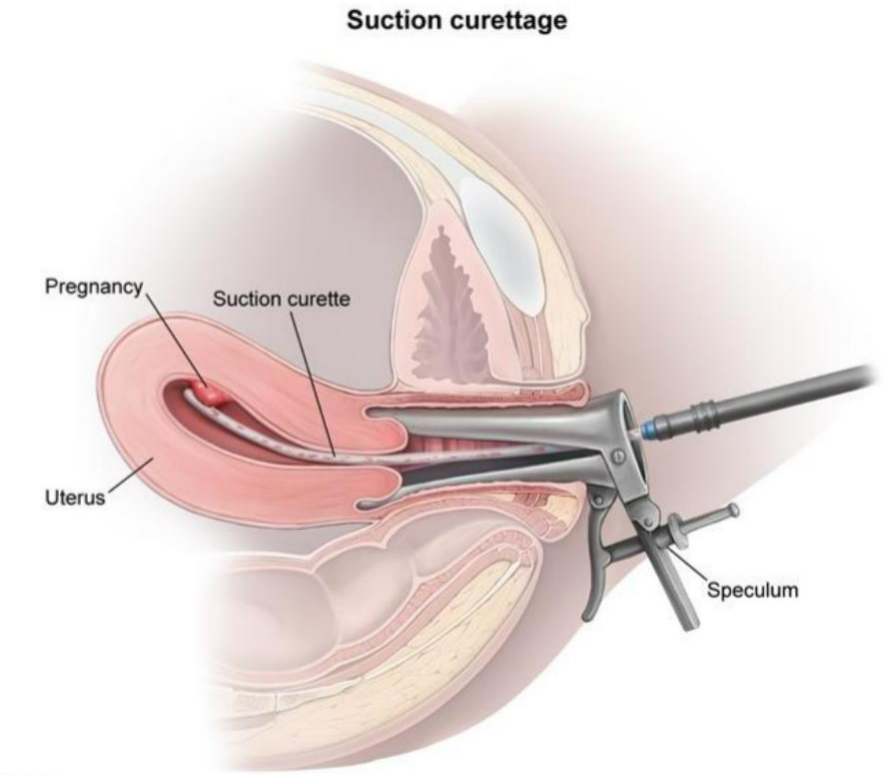
Septic abortion	
Risk factors	Retained POC from: • Elective abortion with nonsterile technique • Missed or incomplete abortion (rare)
Clinical presentation	• Fever, chills, abdominal pain • Sanguinopurulent vaginal discharge • Boggy, tender uterus; dilated cervix • Pelvic ultrasound: retained POC, thick endometrial stripe
Management	• Intravenous fluids • Broad-spectrum antibiotics • Suction curettage
POC = products of conception.	

- What is the *recommended treatment* for patients with **septic abortion**?

  Broad-spectrum antibiotics and suction curettage

  medical emergency; urgent treatment required to reduce risk of sepsis

Surgical management via **suction curettage** is indicated for **symptomatic** (eg, dizziness on standing) and **hemodynamically unstable** (eg, hypotensive, tachycardic) patients with anemia from acute blood loss. Suction curettage removes the retained products of conception, allowing the uterus to fully contract around open arterial vessels, which stops the bleeding. In addition, Rh negative patients require Rho(D) immunoglobulin to prevent isoimmunization from Rh incompatibility



So any patient that presents with hemodynamic instability needs surgical management

Other wise we treat abortion with medical management first:

Mifepristone which is a progesterone antagonist that makes the baby not adherent to the uterus

Then we use misoprostol which is a prostaglandin agonist that causes uterine contractions that expels the fetus.

Demise:

Intrauterine fetal demise	
Definition	• Absent fetal cardiac activity at ≥20 weeks gestation
Risk factors	• Aneuploidy • Fetal or placental anomalies • Fetal growth restriction • Congenital infection • Substance use (eg, tobacco, cocaine) • Maternal conditions (eg, hypertension, diabetes mellitus)
Evaluation	Fetal • Autopsy • Gross & microscopic examination of placenta & umbilical cord • Karyotype/genetic studies Maternal • Kleihauer-Betke test • Antiphospholipid antibodies • Coagulation studies*

Kleihauer-Betke: A maternal blood smear is exposed to acid, and stained afterwards. Adult hemoglobin is removed by the acid, whereas fetal hemoglobin (HbF) is not, resulting in pink color cells that indicate fetal hemoglobin on a positive test.

Women with unexplained late term still birth , should undergo **weekly non-stress testing** in the **third trimester** starting at 32 weeks gestation

Even after thorough evaluation, up to half of IUFDs have **no identifiable cause**. Therefore, patients should be counselled in advance that **testing may be negative.**

Intrauterine fetal demise	
Definition	Fetal death at ≥20 weeks
Diagnosis	Absence of fetal cardiac activity on ultrasound
Management	20-23 weeks • Dilation & evacuation OR • Vaginal delivery* ≥24 weeks • Vaginal delivery*
Complication	Coagulopathy after several weeks of fetal retention

*Cesarean delivery by maternal choice if history of prior classical cesarean/myomectomy

Induce vaginal delivery when mother is **ready**

The absence of cardiac activity must be on ultra sound doppler is not enough.

Delivery planning for a nonviable fetus	
Fetal diagnosis	• Anencephaly • Bilateral renal agenesis • Holoprosencephaly • Acardia • Thanatophoric dwarfism • Intrauterine fetal demise
Obstetric management	• Vaginal delivery • No fetal monitoring
Neonatal management	• Palliative care if not stillborn

💡 Development of **DIC** requires **urgent delivery** of the fetus, via **vaginal** or **cesarean delivery.**

• What is the *recommended management* for a pregnant woman at **28 weeks gestation** that presents in **labor** for a fetus diagnosed with **anhydramnios** and **anencephaly**? The fetus is in **breech** presentation.

Allow spontaneous vaginal delivery

*the priority for patients with lethal fetal anomalies (e.g. anecephaly) is to **minimize maternal morbidity/mortality**; C-section has an increased risk of maternal complications*

*A **breech presentation** in **lethal anomalies** does not require a C-section (vaginal delivery preferred due to increased risk of maternal complications associated with C-section)*

• What is the *next step* in management for a pregnant woman at 25 weeks gestation that presents after feeling **no fetal movement** for the past two days? Fetal heart sounds are not heard on Doppler.

Transabdominal ultrasound

*the patient likely has **intrauterine fetal demise** (fetal death at > 20 weeks), which must be confirmed by the absence of fetal cardiac activity on ultrasound*

Because parents may blame themselves for the baby's death, it is important for the physician to respond with a clear statement that it is not their fault and there is nothing they could have done to prevent the loss.

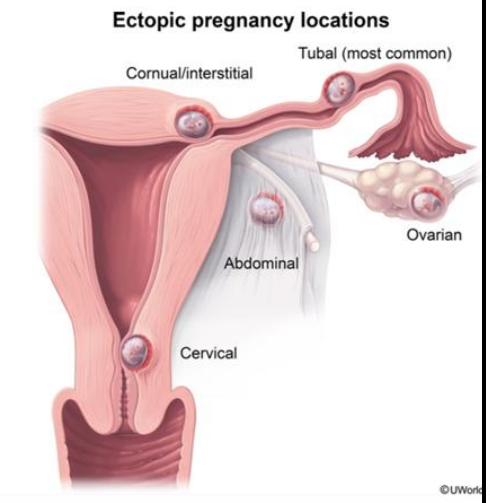
The physician should also avoid prematurely changing the focus of discussion to medical management and reassurance about future pregnancies. Referring to the fetus as their "baby" and avoiding medical terminology (eg, fetal demise) are helpful.

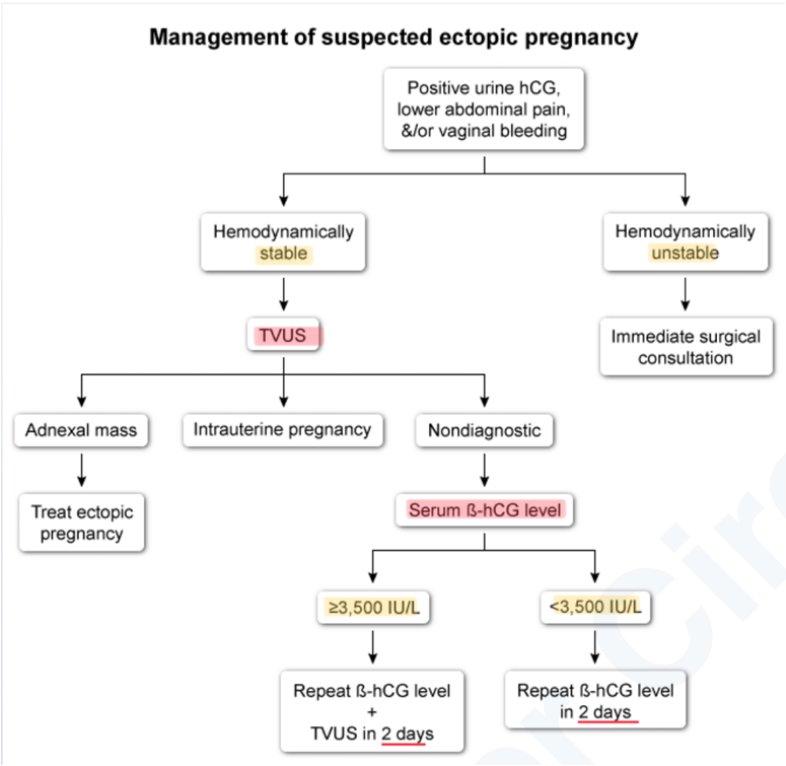
Recurrent pregnancy loss:

Causes of recurrent pregnancy loss	
Structural	• Uterine: fibroids, adhesions, polyps • Cervical insufficiency
Chromosomal	• Aneuploidy • Translocations/rearrangements • Mosaicism
Immunologic/ Hematologic	• Hypercoagulable disorders (eg, antiphospholipid syndrome) • Alloimmune intolerance
Endocrine	• Thyroid disease • Polycystic ovary syndrome • Diabetes mellitus • Hyperprolactinemia
Other	• Advancing maternal age • Defective endometrial receptivity • Decreased ovarian reserve • Celiac disease

Ectopic Pregnancy:

Ectopic pregnancy	
Risk factors	• Previous ectopic pregnancy • Previous pelvic/tubal surgery • Pelvic inflammatory disease
Clinical features	• Abdominal pain, amenorrhea, vaginal bleeding • Hypovolemic shock in ruptured ectopic pregnancy • Cervical motion, adnexal &/or abdominal tenderness • ± Palpable adnexal mass
Diagnosis	• Positive hCG • Transvaginal ultrasound revealing adnexal mass, empty uterus
Management	• Stable: Methotrexate • Unstable: Surgery





- **Early but viable intrauterine pregnancies : ≥35%-50%** rise in β-hCG every 48 hours.
- **Completed spontaneous abortions** : β-hCG levels **decrease**
- **Ectopic and nonviable intrauterine pregnancies : <35% rise**

• To distinguish between an ectopic pregnancy and nonviable intrauterine pregnancy, patients may undergo **diagnostic dilation and curettage**, a procedure that samples tissue within the endometrial cavity. The procedure confirms the abnormal pregnancy's location based on the **postprocedure β-hCG level**:

- **A negative or decreased β-hCG level** confirms that the patient had a nonviable intrauterine pregnancy; these patients require **reassurance** and **observation only** because the products of conception have been removed by the dilation and curettage.
- **A persistent rise in β-hCG level** after dilation and curettage is diagnostic for an **ectopic pregnancy** (ie, the uterus has been evacuated but an extrauterine pregnancy continues to produce β-hCG). These patients require **additional management. (Methotrexate)**

A progesterone level < 5 ng/mL suggests an abnormal or extrauterine pregnancy

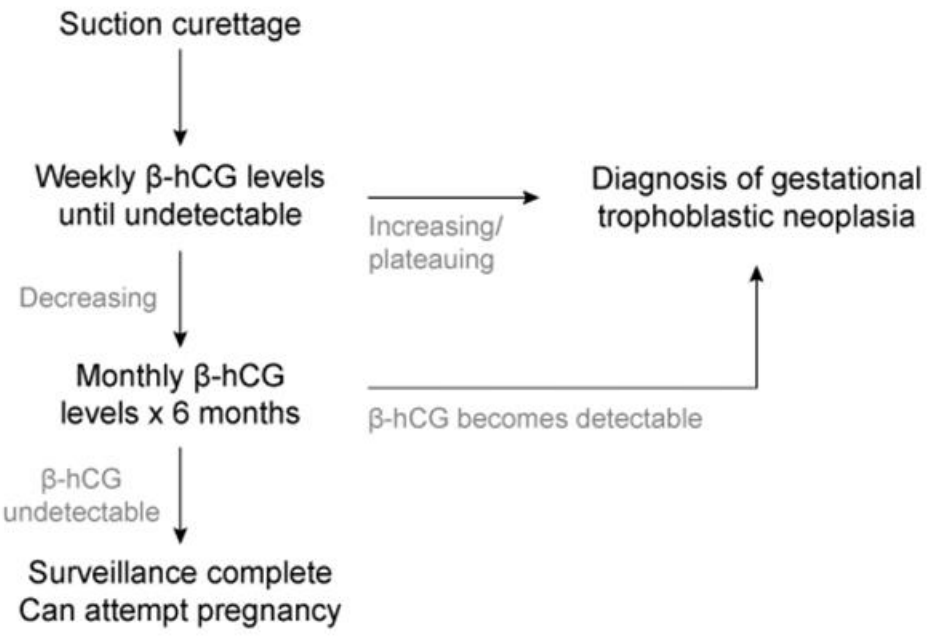
>25 ng/mL suggests a healthy intrauterine pregnancy.

> 💡 **Methotrexate** is **contraindicated** in patients with **hepatic disease** or **renal disease** (eg, diabetic nephropathy) due to decreased clearance and slower drug metabolism increasing the risk for methotrexate toxicity.

Hydatidiform Mole:

Hydatidiform mole	
Clinical presentation	• Abnormal vaginal bleeding ± hydropic tissue • Uterine enlargement > gestational age • Abnormally elevated β-hCG levels • Theca lutein ovarian cysts • Hyperemesis gravidarum • Preeclampsia with severe features • Hyperthyroidism
Risk factors	• Extremes of maternal age • History of hydatidiform mole
Diagnosis	• "Snowstorm" appearance on ultrasound • Quantitative serum β-hCG • Histologic evaluation of uterine contents
Management	• Dilation & suction curettage • Serial serum β-hCG post evacuation • Contraception for 6 months

Management of hydatidiform mole



Choriocarcinoma

Choriocarcinoma:

Gestational trophoblastic neoplasia	
Risk factors	• Hydatidiform mole • Maternal age >40
Clinical features	• Vaginal bleeding • Pelvic pain/pressure • ↑ β-hCG level • Metastasis (eg, vagina, lungs)
Evaluation	• Pelvic ultrasound • Chest x-ray • Thyroid function tests • Hepatic function tests • Renal function tests
Treatment	• Chemotherapy • Hysterectomy

• What is the likely *diagnosis* in a 6-month postpartum woman that presents with **irregular vaginal bleeding**, an **enlarged uterus**, and **dyspnea** with multiple infiltrates on CXR?

Choriocarcinoma

classically occurs after a complete hydatidiform mole, but can occur after ***normal pregnancy*** *or* ***spontaneous abortion***

Liver disorders in pregnancy:

Liver disorders unique to pregnancy		
Disorder	**Presentation**	**Laboratory abnormalities**
ICP	• Intense pruritus	• Elevated bile acids • Elevated levels of liver aminotransferases • Diagnosis of exclusion
HELLP	• Preeclampsia • Right upper-quadrant pain • Nausea/vomiting	• Hemolysis • Moderately elevated liver aminotransferases • Thrombocytopenia
AFLP	• Malaise • Right upper-quadrant pain • Nausea/vomiting • Sequelae of liver failure	• Hypoglycemia • Mildly elevated liver aminotransferases • Elevated bilirubin • Possible disseminated intravascular coagulopathy

Intrahepatic cholestasis of pregnancy	
Clinical features	• Development in 3rd trimester • Generalized pruritus • Pruritus worse on hands & feet • No associated rash • Right upper quadrant pain
Laboratory abnormalities	• ↑ Total bile acids (≥10 μmol/L) • ↑ Liver transaminases (typically <2x normal, rarely >1000 U/L) • ± ↑ Total & direct bilirubin
Obstetric risks	• Intrauterine fetal demise • Preterm delivery • Meconium-stained amniotic fluid • Neonatal respiratory distress syndrome
Management	• Ursodeoxycholic acid • Antihistamines • Delivery at 37 weeks gestation

- Pregnancy, pruritis, elevated total bile acids, +/- elevated aminotransferases

  Intrahepatic cholestasis of pregnancy

  Treatment: **ursodeoxycholic acid,** *typically resolves within weeks following delivery*

Epidemiology

- Most common pregnancy-associated liver disease [1]
- Occurs in ~ 0.4–10% of pregnancies [2]

Etiology [2][5]

- Genetic predisposition
- Increased estrogen and progesterone during pregnancy
- Certain drugs (e.g., antibiotics)

Clinical features [2][5]

- Pruritus
- Jaundice [1]
- Steatorrhea (uncommon)

Diagnostics [1][2][4]

- ↑ **Total serum bile acid levels** (> 10 mcmol/L): confirmatory [1][2][3]
- ↑ ALT, ↑ AST [2]
- Normal or mildly elevated bilirubin
- ↑ GGT, ↑ ALP [3]

> Elevated total serum bile acid level (> 10 mcmol/L) in a patient with pruritus in the second or third trimester (without other causes of pruritus) is diagnostic for intrahepatic cholestasis of pregnancy. Elevated transaminases are not required for diagnostic confirmation. [2]

Management [2][4]

- **Pharmacological treatment**
  - First line: ursodeoxycholic acid (reduces bile acid levels and pruritus) [1][2][4]
  - Alternative or adjunctive therapies (e.g., for refractory pruritus): first-generation antihistamines
- Prenatal care
  - Perform antepartum fetal surveillance.
  - Administer corticosteroids for fetal lung maturity if preterm delivery is anticipated. [1]
- **Peripartum management**: Deliver at 36–39 weeks' gestation based on severity. [4]
- **Postpartum management** [4]
  - Complete resolution typically occurs postpartum.

> Early initiation of therapy with ursodeoxycholic acid may reduce the risk of preterm birth and stillbirth. [4]

> The risk of **intrauterine fetal demise (IUFD)** is particularly high when serum bile acids are **≥100 µmol/L**

Acute fatty liver of pregnancy	
Clinical features	• Nausea, vomiting • Right upper quadrant/epigastric pain • Fulminant liver failure
Laboratory findings	• Profound hypoglycemia • ↑ Aminotransferases (2-3× normal) • ↑ Bilirubin • Thrombocytopenia • Disseminated intravascular coagulopathy
Management	• Immediate delivery

Immediate delivery irrespective of gestational age

- **Normal BP** *(vs HELLP)*

💡 **AFLP** is unique because it is an intrahepatic process due to **microvesicular fatty infiltration** of hepatocytes secondary to abnormal maternal-fetal fatty acid metabolism.

Epidemiology

- 1–3 cases per 10,000 pregnancies [7]

Etiology

- Risk factors [1][5]
  - Multiple pregnancy
  - Low BMI
- **Pathophysiology**: dysfunction of fatty acid β-oxidation [5]

Clinical features [1][3][5]

- Sudden onset of jaundice
- RUQ pain, nausea, and vomiting
- Polyuria and polydipsia
- Clinical features of complications (e.g., hepatic encephalopathy, ascites, bleeding diathesis)
- Preeclampsia (present in ~ 50% of patients) [3]

Diagnostics [1][3][5]

- Ultrasound is the preferred initial imaging modality to exclude alternative diagnoses (e.g., liver hematoma). [1]
- Liver biopsy is rarely necessary but may be considered for: [5]
  - Diagnostic uncertainty (e.g., atypical presentation) that affects management [2]
  - Persistent postpartum liver dysfunction
- Swansea criteria: a set of clinical, imaging, and/or histological findings commonly used to evaluate for AFLP
  - The presence of ≥ 6 features suggests AFLP.
- Diagnostic testing may show findings associated with complications (e.g., thrombocytopenia in patients with DIC). [1][2]

Swansea criteria for AFLP [1][2][8]	
Clinical features	• Vomiting • Abdominal pain • Polydipsia and/or polyuria • Encephalopathy
Laboratory studies	• ↑ WBC • ↑ ALT, ↑ AST • Hyperbilirubinemia • ↑ Creatinine • Hyperuricemia • Hypoglycemia • Coagulopathy (prolonged PT and/or aPTT) • Hyperammonemia
Ultrasound findings	• Bright white liver or presence of ascites
Histology	• Microvascular steatosis on liver biopsy
Presence of ≥ 6 features suggests AFLP.	

Rule out causes of acute liver injury that are unrelated to pregnancy (e.g., acute viral hepatitis, autoimmune hepatitis, drug-induced liver injury, Wilson disease). [5]

Differential diagnoses

- HELLP syndrome
- Preeclampsia with severe features
- Other causes of acute liver failure

It is often difficult to differentiate between AFLP, HELLP, and preeclampsia with severe features, and these conditions can also coexist. Renal failure, hyperuricemia, and hypoglycemia are more common and severe in AFLP than in HELLP and severe preeclampsia. [5]

Management [5][7]

- Deliver immediately regardless of gestational age. [1]
- Provide supportive care and management of complications (see "Management of acute liver failure").

Symptomatic cholelithiasis in pregnancy	
Pathophysiology	• ↑ Biliary cholesterol excretion (estrogen) • ↓ Gallbladder motility (progesterone)
Clinical features	• Recurrent, postprandial epigastric/RUQ pain • RUQ ultrasound with echogenic foci (stones or sludge)
Management	• Conservative (eg, pain control) • Cholecystectomy (for complicated, recurrent cases)
RUQ = right upper quadrant.	

Cholecystectomy usually **delayed** until postpartum

Patients who develop biliary colic while pregnant are typically treated with intravenous **fluids** and **pain control**.

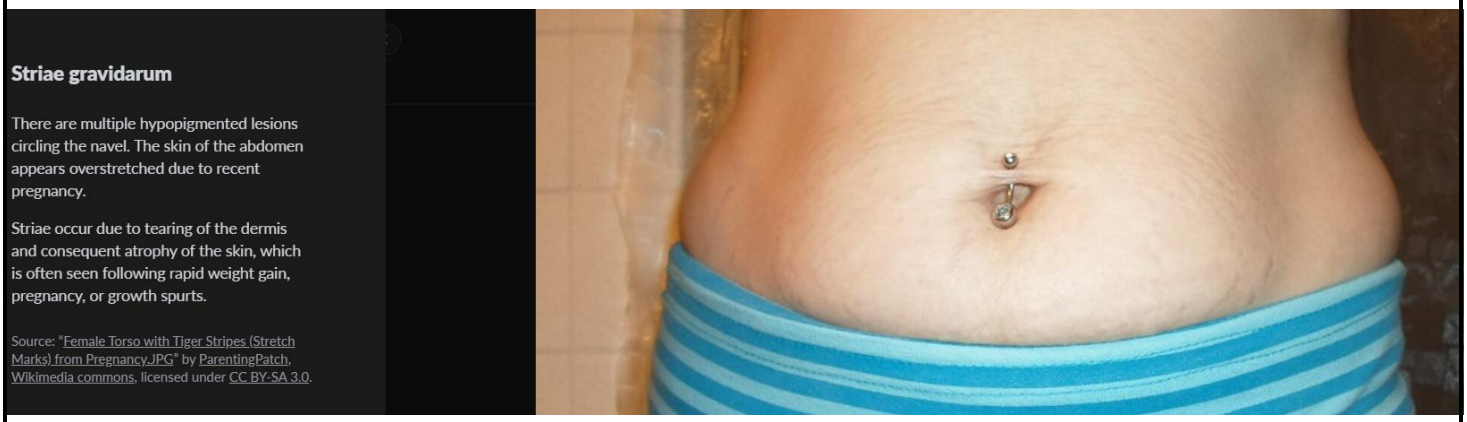
If symptoms cannot be controlled with supportive care, cholecystectomy is often performed during the **second trimester**.

Pregnancy-induced skin changes vs intrahepatic cholestasis of pregnancy		
	Pregnancy-induced skin changes	**Intrahepatic cholestasis of pregnancy**
Presentation	• Focal pruritus • No rash	• Generalized pruritus • Hands & foot involvement • No rash
Etiology	• Pregnancy hormone changes	• Intrahepatic cholestasis
Laboratory abnormalities	• None • ± Mild transaminitis	• ↑ Bile acids • Transaminitis
Obstetric risks	• None	• Intrauterine fetal demise
Obstetrical management	• Expectant	• Delivery at 37 weeks
Treatment	• Oatmeal baths • Ultraviolet light • Antihistamines	• Ursodeoxycholic acid • Antihistamines

Skin changes in pregnancy and dermatoses:

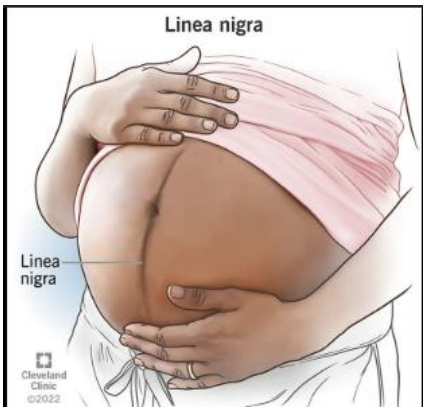
- Spider angioma
- Palmar erythema
- Striae gravidarum: scarring that manifests as erythematous, violaceous, and/or hypopigmented linear striations on the abdomen

Spider angiomas are benign, erythematous lesions that blanch with pressure. They occur frequently in pregnancy due to increased estrogen levels that cause increased blood vessel vascularity; they typically resolve postpartum.



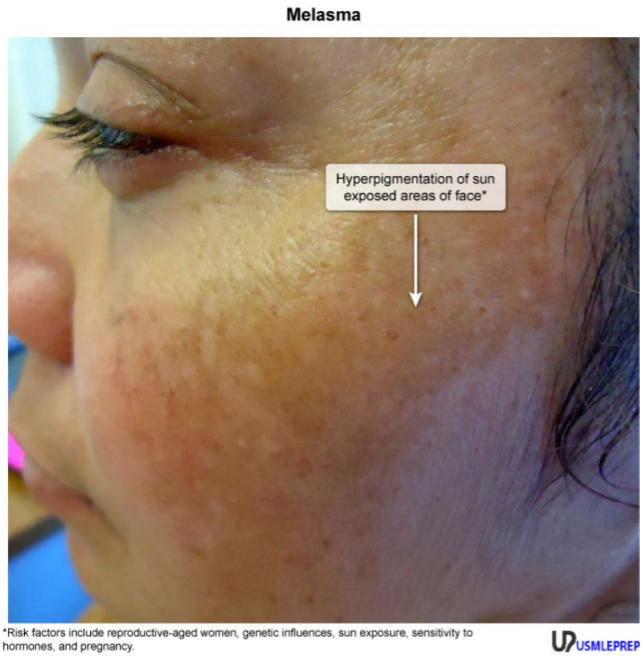
Due to increased secretion of melanocyte-stimulating hormone There will be hyperpigmentation in some areas:

chloasma, linea nigra, hyperpigmentation of the nipples.



Chloasma is an old term used to describe melasma due to hormonal changes

While melasma is the more broad term to describe A common, acquired, chronic skin condition characterized by **symmetrical, blotchy, brown to gray-brown patches** on the face (cheeks, bridge of nose, forehead, chin, and upper lip) and sometimes other sun-exposed areas.



melasma, an acquired hyperpigmentation disorder that occurs on **sun-exposed areas of the face**. Melasma likely occurs when ultraviolet radiation triggers melanocyte proliferation and pigment deposition. It is more common in women of reproductive age, particularly during **pregnancy** because estrogen and progesterone also stimulate melanocyte proliferation. Additional risk factors include darker skin tones, thyroid dysfunction, medications (eg, antiepileptics), and cosmetic use.

Patients typically develop **irregularly shaped**, **hyperpigmented macules** of varying color (eg, light to dark brown, ash/blue) that occur in a symmetric centrofacial, mandibular, or **malar distribution**. Melasma is not associated with systemic symptoms

Melasma is diagnosed clinically and **requires no further evaluation**. Management during pregnancy includes minimizing progression of the hyperpigmentation with sun exposure avoidance and broad-spectrum sunscreen use. Melasma typically resolves postpartum; areas that do not resolve can be treated with skin-lightening agents or topical retinoids after delivery.
the important thing is that it spares the nasolabial folds.

Dermatoses in pregnancy:

Pemphigoid gestationis [14]

- **Definition**: bullous, pemphigoid-like dermatosis during pregnancy of unknown cause (most likely immunological)
- **Epidemiology**: 1:50,000 pregnancies (in the US)
- **Clinical features**
  - Commonly starts in the **periumbilical** region during the 2nd and 3rd trimester
  - Intensely pruritic, mostly nonblistering lesions (eczema, urticarial or papular lesions) on extremities and mucous membranes
  - Grouped vesicles with herpetiform appearance ("gestational herpes") usually occur as the disease advances.
- **Diagnosis**: The diagnosis is confirmed via biopsy and immunofluorescence.
- **Treatment**: glucocorticoids (topical or systemic) at the lowest dose needed to control disease [15]
- **Prognosis**
  - Usually self-limited; heals spontaneously after delivery, but associated with complications (e.g., premature labor, increased lifetime risk of autoimmune disease)
  - Recurrence is possible, especially appearing:
    - Spontaneously in the postpartum period
    - In subsequent pregnancies
    - When taking contraceptives containing progestin or estrogen
    - During menstruation
  - Infants born to women with gestational pemphigoid can develop transient blistering that resolves spontaneously.

Manifestations of gestational pemphigoid

(A) Truncal urticarial papules and plaques

(B) Periumbilical plaque with two additional lesions above the navel

(C) Multiple vesicular plaques

(D) Plaques on the forearm with sporadic bullae

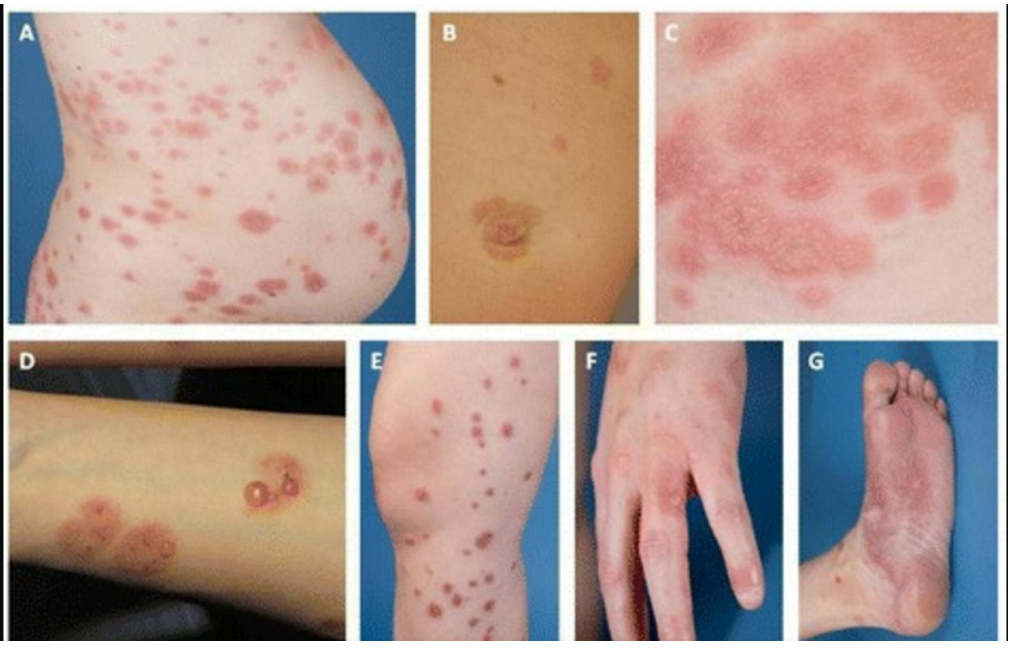
(E) Multiple, nonconfluent, erythematous plaques on the upper and lower leg

(F) Multiple, vesicular plaques on the left hand

(G) Large plaque of the left sole of the foot with sporadic bullae

Source: "Figure 1: Skin findings of gestational pemphigoid. in: "Gestational pemphigoid"" by Kaarin Mäkikallio, Kaisa Tasanen, Laura Huilaja, Orphanet Journal of Rare Diseases, licensed under CC BY 4.0.

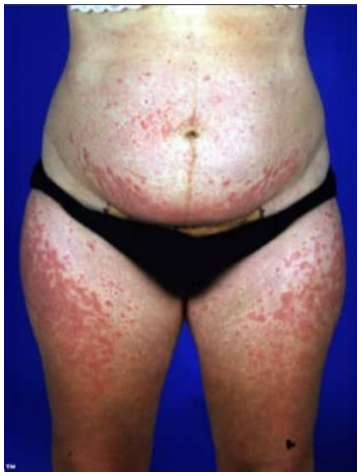
FEEDBACK

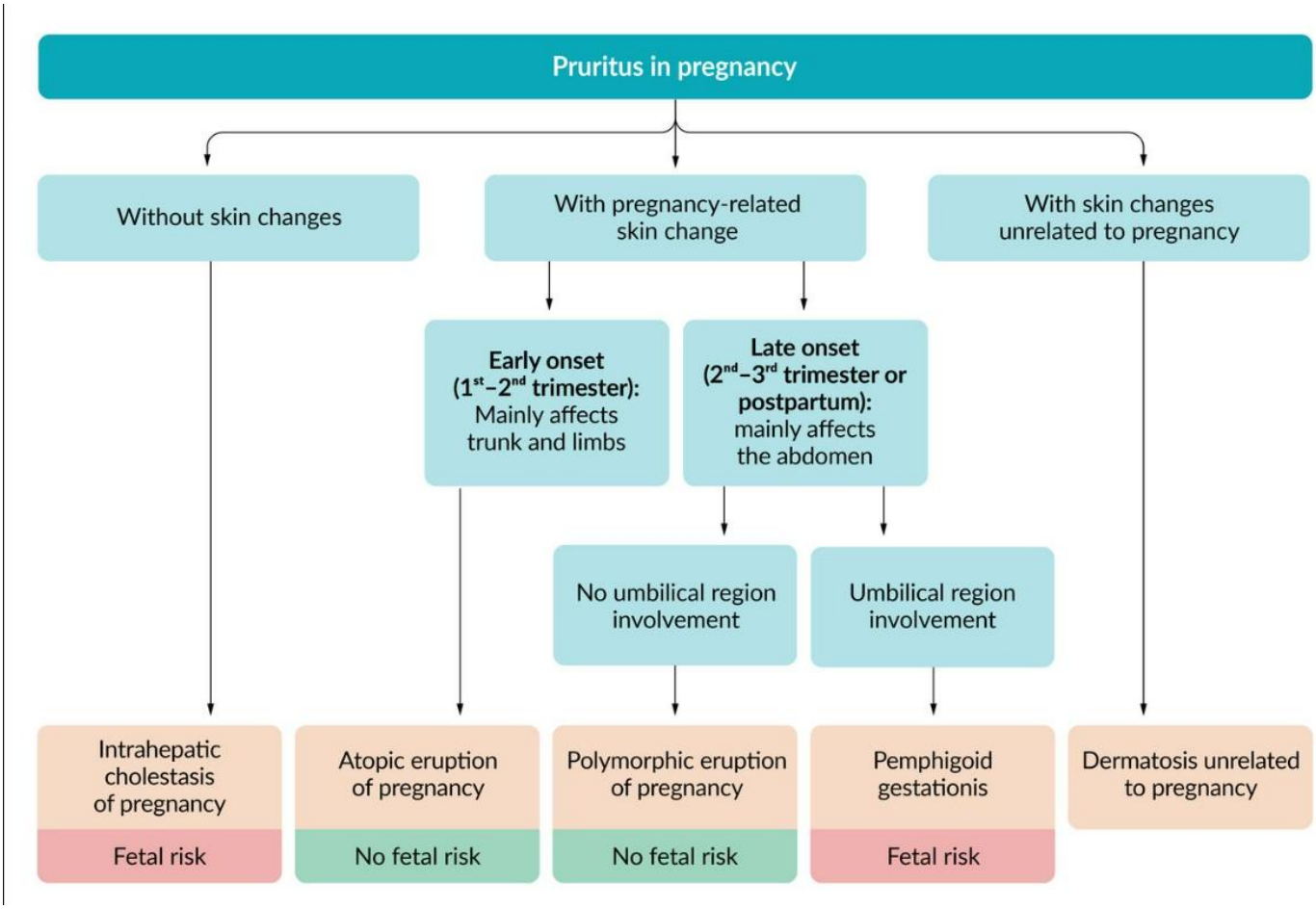


Polymorphic eruption of pregnancy (PEP)

- **Description:** A benign, inflammatory condition that most commonly affects primiparous women in the third trimester of pregnancy or immediately postpartum.
- **Clinical features**
  - Very pruritic, erythematous papules **within abdominal striae**
  - Lesions can spread to the chest, back, and extremities, and coalesce into urticarial plaques, sparing the face, palms, and soles
  - Lesions last 4–6 weeks and resolve spontaneously [17]
- **Management:** topical corticosteroids

Polymorphic eruption of pregnancy (also known as pruritic urticarial papules and plaques of pregnancy [PUPPP]) may be triggered by the skin stretching over the enlarging gravid uterus that causes inflammation and pruritus within abdominal striae. It is a benign self-limited condition that resolves postpartum.





Postpartum:

	Postpartum period
Normal findings	Transient rigors/chills Peripheral edema Lochia rubra Uterine contraction & involution Breast engorgement
Routine care	Rooming-in/lactation support Serial examination for uterine atony/bleeding Perineal care Voiding trial Pain management

The **immediate postpartum period** (ie, hours to days) is marked by physiologic changes that begin immediately after placental delivery:

- **Increased oxytocin levels** (endogenous and administered) cause **uterine contraction**, which compresses placental bed vessels and protects against postpartum hemorrhage. As the uterus involutes, it rapidly decreases in size, becoming firm and palpable 1-2 cm above or below the umbilicus. Involution also generates subsequent **lochia** (shedding of the uterine decidua and blood), which initially appears bloody with small clots and can continue for several weeks.
- **Increased prolactin levels** stimulate breast milk excretion and milk letdown over the course of hours to days. Infant suckling further increases maternal prolactin and oxytocin levels (ie, positive feedback).
- **Decreased estrogen and progesterone levels** may cause postpartum **chills and shivering**, with subsequent mild hyperthermia/low-grade fever in the first 24 hours after delivery.

Normal postpartum lochia		
	Expected duration	**Description**
Lochia rubra	• Birth to 3-4 days postpartum	• Dark or bright red (blood); odor similar to that of menstrual blood; occasional small clots; quantity decreasing each day
Lochia serosa	• 4th postpartum day to 10th or 14th postpartum day	• Serosanguineous (pink); brownish (old blood); quantity gradually decreasing in amount
Lochia alba	• 11th postpartum day to 6 weeks postpartum	• White/yellow; creamy; light quantity
Lochia may increase in quantity after breastfeeding (suckling releases oxytocin & causes uterus to contract) & 7-14 days postpartum, when scabbing on the placental site sloughs off (heavier bleeding for <2 hr); lochia may also feel increased after lying down & then standing (due to blood pooling in vagina).		

• Lochia Rubra : upto **1 Week**; Bright Red blood with small clots **(<1 pads per hour)**

• Patients often have concerns about **postpartum bleeding** because it is **heavier** and more prolonged than normal menses. These patients should be evaluated for features concerning for delayed postpartum hemorrhage, including the following:

- Passage of **large blood clots**
- **Increased pad counts** (eg, saturation of **≥1 pad/hr for ≥2 consecutive hours**)
- Signs and symptoms of **anemia** (eg, dizziness, chest pain) due to **acute blood loss**

PPH:

Postpartum hemorrhage	
Definition	• >500 mL after vaginal delivery • >1,000 mL after cesarean delivery
Risk factors	• Prolonged or induced labor • Chorioamnionitis • Multiple gestation • Polyhydramnios • Grand multiparity • Operative delivery
Causes	• Uterine atony (most common) • Retained placenta • Genital tract laceration • Uterine rupture • Coagulopathy
Treatment	• Bimanual uterine massage, oxytocin • Intravenous fluids, oxygen • Uterotonics (methylergonovine, carboprost, misoprostol) • Intrauterine balloon tamponade • Uterine artery embolization • Hysterectomy

*Oxytocin is a first-line agent; other agents, such as methylergonovine and carboprost may be administered if **oxytocin fails***

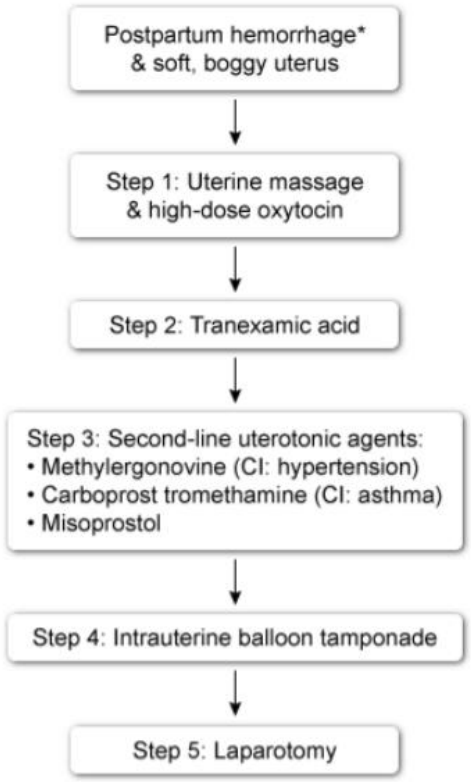
	Postpartum uterine atony
Risk factors	Uterine fatigue from prolonged, induced, or precipitous labor Intraamniotic infection Uterine overdistension (multiple gestation, macrosomia, polyhydramnios) Retained placenta Grand multiparity (≥5 prior deliveries)
Clinical features	Most common cause of postpartum hemorrhage Enlarged, soft, boggy, poorly contracted uterus
Interventions	Bimanual uterine massage Correction of bladder distension High-dose oxytocin, misoprostol Tranexamic acid Carboprost, methylergonovine Intrauterine balloon tamponade Possible surgical intervention (if atony unresolved)

uterine atony, the most common cause of **postpartum hemorrhage** (PPH) within 24 hours of delivery. Uterine atony occurs due to insufficient uterine contractility, resulting in the inability of the uterus to clamp down on bleeding placental bed vessels. Risk factors include uterine overdistension (often associated with **fetal macrosomia** [weight ≥4 kg]), uterine fatigue (eg, **prolonged induction of labor**), and **operative vaginal delivery** (eg, forceps-assisted), all of which are present in this patient.

Normally during the immediate postpartum period, contractions increase uterine tone, causing a firm uterine fundus to be palpable at or below the umbilicus. In contrast, patients with uterine atony have a **soft, boggy, enlarged uterus** with the **fundus palpable above the umbilicus** because of decreased uterine tone. Some patients may not have an immediate PPH, such as this patient whose bleeding was identified 60 minutes after delivery, because blood can gradually accumulate in the lower uterine segment (the least contractile portion of the uterus).

-precipitous labor which is rapid (less than 3 hours labor) is also a risk factor-.

Management of postpartum hemorrhage due to uterine atony

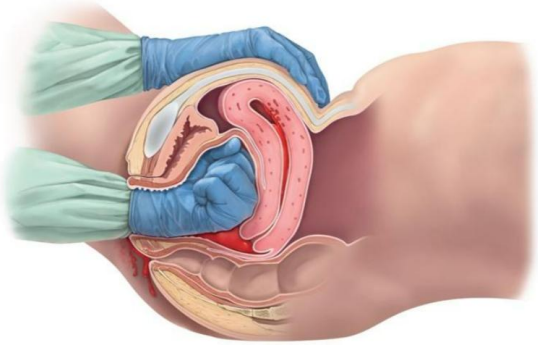


*Estimated blood loss ≥1,000 mL or bleeding + hypovolemia.

Postpartum hemorrhage (PPH) is defined as an estimated blood loss ≥1,000 mL or bleeding with signs/symptoms of hypovolemia. The management of PPH **due to uterine atony** occurs in a stepwise approach:

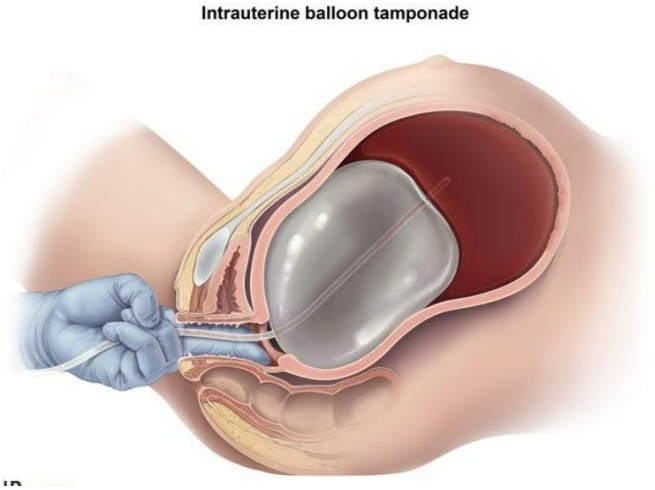
1. **Initial management** includes the placement of 2 large-bore intravenous lines for volume resuscitation. Bimanual uterine massage and bladder catheterization are performed to improve uterine tone. **High-dose oxytocin**, the **first-line uterotonic agent**, is administered.

Bimanual uterine massage



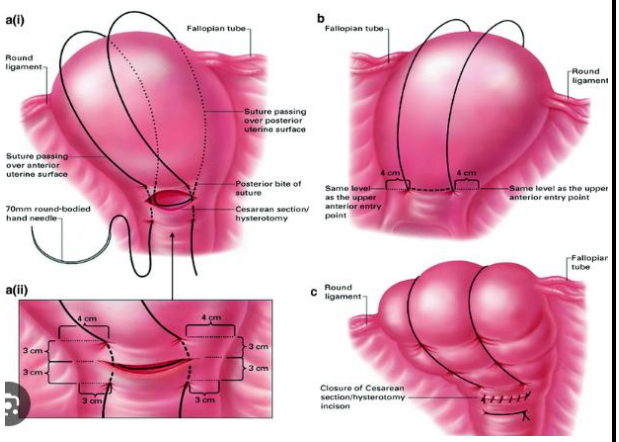
2. If uterine atony persists, the best next step in management is tranexamic acid, an antifibrinolytic agent that prevents the breakdown of blood clots to achieve hemostasis; its use reduces maternal mortality from hemorrhage.

3. Patients with continued bleeding are administered second-line uterotonic medications, including carboprost tromethamine, methylergonovine, and misoprostol. Carboprost tromethamine is contraindicated in patients with asthma due to the risk of bronchospasm. Methylergonovine is contraindicated in patients with hypertension (regardless of the patient's current blood pressure) due to an increased risk of stroke.
4. If PPH does not resolve with medical therapy, an <u>intrauterine balloon tamponade</u> can be used to compress placental bed blood vessels for hemostasis. -also called Bakri balloon-



5. Finally, patients with PPH refractory to medical and minimally invasive techniques require either uterine artery embolization or surgical management with laparotomy (and possible hysterectomy).

• **Uterine atony** *unresponsive* to medical management may respond to a **B-lynch compression** suture during exploratory laparotomy.



We do bladder cath because:

- A **full bladder pushes the uterus upward and laterally**
- This **interferes with uterine contraction**
- Poor contraction → **continued postpartum hemorrhage**

Emptying the bladder:

- Allows the uterus to **sit midline**
- Improves **effective uterine contraction**
- Enhances response to **uterotonics** (oxytocin, etc.)

Differential diagnosis of postpartum hemorrhage			
Diagnosis	**Risk factors**	**Examination**	**Management**
Uterine atony	• Prolonged labor • Chorioamnionitis • Uterine overdistension (multiples, fetal macrosomia, polyhydramnios)	• Enlarged, boggy uterus	• Bimanual uterine massage • Uterotonic medications
Retained products of conception	• Succenturiate placenta • Manual extraction of placenta • History of previous uterine surgery	• Enlarged, boggy uterus • Placenta missing cotyledons • Retained placental fragments on ultrasound	• Manual extraction
Genital tract trauma	• Operative vaginal delivery	• Laceration of cervix or vagina • Enlarging hematoma	• Laceration repair
Inherited coagulopathy	• History of abnormal bleeding in patient or family members	• Continued bleeding despite contracted uterus	• Correction of coagulopathy

Vaginal hematoma	
Risk factors	• Operative vaginal delivery • Infant ≥4000 g (8.8 lb) • Nulliparity • Prolonged 2nd stage of labor
Clinical features	• Vaginal mass • Rectal or vaginal pressure • ± hypovolemic shock
Treatment	• Nonexpanding: observation • Expanding: embolization, surgery

Because the **vagina** is supplied by **branches** of the **uterine artery** (which receives **30%** of maternal cardiac output at delivery), patients with a **vaginal laceration/hematoma can have profuse bleeding.**

Vulval Hematoma caused by damage to **Pudendal Artery.**

Secondary (late) postpartum hemorrhage		
Cause	**Clinical features**	**Management**
Retained POCs	• Heavy bleeding • ± Uterine atony	• Dilation & curettage
Placental site subinvolution	• Heavy bleeding • Uterine atony	• Uterotonics (eg, oxytocin, methylergonovine, carboprost)
Postpartum endometritis	• Fever • Uterine tenderness • Purulent lochia	• Broad-spectrum IV antibiotics (eg, clindamycin & gentamicin)
POCs = products of conception; **IV** = intravenous.		

Secondary = **>24 hours** after Delivery

💡 **Delayed placental delivery** occurs when the placenta does not deliver within **30 minutes**.

A common risk factor is
extreme preterm delivery (eg, <26 weeks gestation) because these deliveries typically require prolonged oxytocin administration and are often complicated by intraamniotic infection, **stillbirth**, and placental disorders (eg, preeclampsia, placenta accreta). In addition, the prolonged oxytocin administration can cause a paradoxical decrease in myometrial contractility, thereby inhibiting placental expulsion. Intrauterine inflammation (eg, from intrauterine fetal demise) can also increase placental adhesion to the uterine wall.

Patients with a
retained placenta usually have profuse **postpartum bleeding**, which may be immediate or delayed. Initial management includes gentle downward cord traction and oxytocin administration to promote placental separation and expulsion.

If the placenta does not deliver with these measures,
manual placental extraction or **dilation and curettage** are indicated.

• What is the *next step* in management for a mother with **post-partum hemorrhage** following a forceps-assisted vaginal delivery? The patient is afebrile and the uterus is normal-sized and firm.

Genital tract inspection

genital tract injury is a common cause of PPH after operative vaginal deliveries

Operative vaginal delivery (vacuum/forceps)	
Indications	• Protracted 2nd stage of labor • Fetal heart rate abnormalities • Maternal contraindications to pushing
Fetal complications	• Laceration • Cephalohematoma • Facial nerve palsy • Intracranial hemorrhage • Shoulder dystocia
Maternal complications	• Genitourinary tract injury • Urinary retention • Hemorrhage

• **Post-cesarean** delivery patients with **hemorrhagic shock** and **no signs of uterine atony** most likely have **intraabdominal bleeding** from uterine artery injury.

a rare but life-threatening cause of postpartum hemorrhage that typically presents with **no incisional bleeding** and **minimal abdominal or back pain**

Hemodynamically **unstable** patients with a suspected retroperitoneal hematoma require **emergency laparotomy.**

Uterine Inversion:

Uterine inversion	
Pathophysiology	• Excessive fundal pressure • Excessive umbilical cord traction
Presentation	• Lower abdominal pain • Round mass protruding through cervix • Uterine fundus not palpable transabdominally • Hemorrhage shock
Management	• Aggressive fluid replacement • Manual replacement of the uterus • Placental removal & uterotonic drugs after uterine replacement

• **Neurogenic shock, due to the traction effect** on the surrounding peritoneum, may also occur, resulting in a **paradoxical bradycardia**.

Placenta Accreta:

Placenta accreta	
Definition	• Morbidly adherent placental attachment to the myometrium
Risk factors	• Placenta previa + prior uterine surgery (eg, cesarean delivery, D&C, myomectomy)
Clinical features	• Prenatal diagnosis: US with placenta previa, numerous placental lacunae, myometrial thinning • Postpartum diagnosis: adherent placenta, postpartum hemorrhage
Management	• Cesarean hysterectomy with placenta in situ
D&C = dilation & curettage; **US** = ultrasound.	

• What is the likely diagnosis of a "**firm uterus**" in the setting of a **post-partum hemorrhage**?

Placenta accreta

Most *important* risk factor? **Previous C-section**

Presents with delayed placental detachment and massive PPH at the time of attempted manual separation of the placenta

💡 *Antenatally* diagnosed **placenta accreta** is delivered by **planned cesarean hysterectomy**.

Sheehan Syndrome:

Sheehan syndrome	
Pathogenesis	• Obstetric hemorrhage complicated by hypotension • Postpartum pituitary infarction
Clinical features	• Lactation failure (↓ prolactin) • Amenorrhea, hot flashes, vaginal atrophy (↓ FSH, LH) • Fatigue, bradycardia (↓ TSH) • Anorexia, weight loss, hypotension (↓ ACTH) • Decreased lean body mass (↓ growth hormone)

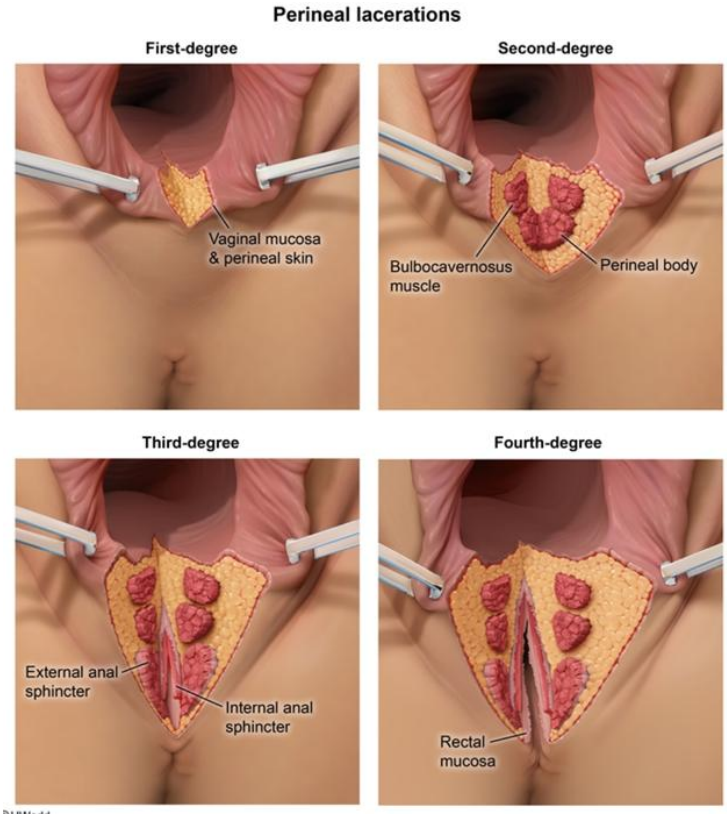
- **T/t:** Replace the hormones individually. Cant do anything else

The pituitary is physiologically hypertrophic in pregnancy and, consequently, more susceptible to hypoxic injury. **Sheehan syndrome** (postpartum **hypopituitarism**) is a rare but potentially severe complication of **massive obstetric hemorrhage** and hypovolemic shock causing ischemic **infarction and necrosis** of the anterior **pituitary**.

The resulting symptoms of hypopituitarism can occur in the immediate postpartum period or up to several years later. Manifestations include fatigue, weight loss, **hypotension**, **inability to breastfeed**. In addition, non-lactating women typically resume menses within 10 weeks of birth, hypogonadotropic hypogonadism leads to amenorrhea in these patients.

Postpartum hemorrhage can also cause iron-deficiency anemia. Management involves evaluation of the pituitary hormones and replacement as needed.

Perineal Laceration:



During vaginal delivery, constant, increased pressure (eg, fetal head, maternal pushing) on maternal tissues causes vaginal, labial, and perineal edema. The vagina has increased elasticity to accommodate the fetal head, but other structures, such as the perineum, are less elastic and more likely to lacerate. Therefore, perineal lacerations are common after vaginal delivery, particularly in a primigravidas.

second-degree perineal laceration, which disrupts the vaginal mucosa, perineal skin, and perineal body (ie, bulbocavernosus muscle). Second-degree perineal lacerations typically have no long-term sequelae because they heal quickly (due to high vascularity of vaginal tissue) and do not cause extensive muscle damage. This contrasts with more extensive lacerations, which disrupt the anal sphincter muscles (third-degree) and rectal mucosa (fourth-degree) and can lead to anal or fecal incontinence.

Although second-degree perineal lacerations heal quickly, patients in the **immediate postpartum period** often have **localized pain**, particularly with voiding (due to the proximity of the laceration to the urethra), and **perineal edema**. This pain is normal (ie, not a sign of infection) and requires only **supportive care** with nonsteroidal anti-inflammatory drugs and sitz baths.

Fistula:

Rectovaginal fistula	
Risk factors	• Pelvic radiation • Obstetric trauma • Pelvic surgery • Colon cancer • Diverticulitis • Crohn disease
Clinical features	• Uncontrollable passage of gas &/or feces from the vagina
Diagnostic studies	• Physical examination • Fistulography • Magnetic resonance imaging • Endosonography

• **Rectovaginal fistula** may occur after obstetric trauma and presents with **incontinence of flatus and feces** through the vagina.

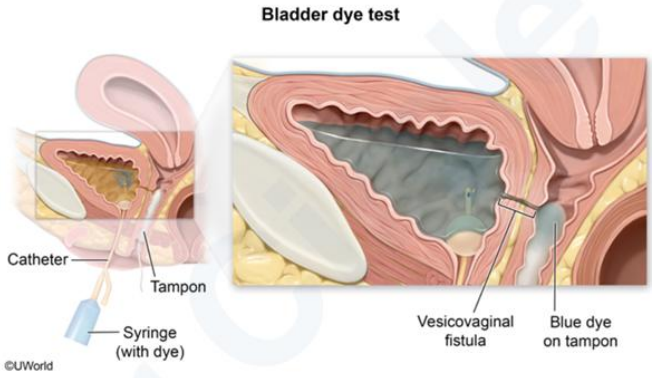
"Red, velvety (rectal) mucosa" *may be seen on* ***posterior vaginal wall***

• What is the likely *diagnosis* in a patient that presents with **foul-smelling brown discharge** from the posterior vaginal wall two weeks after a vaginal delivery complicated by third-degree laceration?

Rectovaginal fistula

Vesicovaginal fistula	
Risk factors	• Pelvic surgery • Pelvic irradiation • Prolonged labor/childbirth trauma • Genitourinary malignancy
Clinical features	• Painless, continuous urine leakage from the vagina
Diagnostic studies	• Physical examination • Dye test • Cystourethroscopy

Pelvic examination typically shows vaginal pooling of urine, a visible defect, or an area of **raised, red granulation tissue** on the anterior vaginal wall.



💡 Takes some **days** to form and **no bladder distention** *(vs* ***Bladder atony*** *sec to* ***epidural****)*

Pubic Symphysis Diastasis:

	Pubic symphysis diastasis
Risk factors	Fetal macrosomia Multiparity Precipitous labor Operative vaginal delivery
Presentation	Difficulty ambulating Radiating suprapubic pain Pubic symphysis tenderness Intact neurologic examination
Management	Conservative Nonsteroidal anti-inflammatory drugs Physical therapy Pelvic support

*typically resolves within the first **4 weeks** postpartum*

During pregnancy, increased levels of progesterone and relaxin increase pelvic mobility and promote a physiologic widening (diastasis) of the pubic symphysis to facilitate vaginal delivery. However, after a **traumatic delivery**, patients can develop a symptomatic **pubic symphysis diastasis**. Risk factors include **fetal macrosomia**, forceps-assisted vaginal delivery, and multiparity.

Symptomatic pubic symphysis diastasis typically presents with **suprapubic pain** that **radiates** to the back, hips, thighs, or legs and is **exacerbated by walking**, weight-bearing, or position changes. The diagnosis is made clinically; patients have **point tenderness to palpation** over the pubic symphysis and sometimes have a waddling gait. Management is typically conservative with supportive care (eg, pelvic support, physical therapy), and most patients recover within the first 4 weeks postpartum

Septic pelvic thrombophlebitis:

	Septic pelvic thrombophlebitis
Risk factors	Cesarean delivery Pelvic surgery Endometritis Pelvic inflammatory disease Pregnancy Malignancy
Pathophysiology	Hypercoagulability Pelvic venous dilation Vascular trauma Infection
Presentation	Fever unresponsive to antibiotics No localizing signs/symptoms Negative infectious evaluation Diagnosis of exclusion
Treatment	Anticoagulation Broad-spectrum antibiotics

septic pelvic thrombophlebitis (SPT), a complication associated with either pelvic surgery or the postpartum period. SPT is a thrombosis of the deep pelvic or ovarian veins (as seen in this patient's bilateral lower quadrant tenderness) that becomes infected. Several factors predispose postpartum patients to thrombosis:

- Hypercoagulable state of pregnancy
- Pelvic venous stasis and dilation
- Endothelial damage from infection and/or trauma during delivery

Because the most common etiology for postpartum fever is endometritis, patients are initially treated empirically with antibiotics. **Persistent fever** unresponsive to broad-spectrum antibiotic therapy and a negative infectious evaluation (eg, blood and urine cultures, urinalysis) suggest SPT, which is a **diagnosis of exclusion**. Risk factors include cesarean delivery and chorioamnionitis/endometritis. Treatment is with **anticoagulation** and **broad-spectrum antibiotics**.

Diagnosis of exclusion; *due to an infected thrombosis of the deep pelvic or ovarian veins*

Postpartum Endometritis:

Postpartum endometritis	
Risk factors	• Cesarean delivery • Intraamniotic infection • Group B *Streptococcus* colonization • Prolonged rupture of membranes • Operative vaginal delivery
Clinical features	• Fever >24 hr postpartum • Uterine fundal tenderness • Purulent lochia
Etiology	• Polymicrobial infection
Treatment	• Clindamycin & gentamicin

Infection of Endometrium lining

• **Neither blood** nor **endometrial cultures** are required for diagnosis, but further evaluation is indicated if there is **no clinical improvement** after **48 hours of antibiotic therapy**

Clindamycin covers: **Gram +ve** and **Anaerobes**

Gentamycin (**Aminoglycoside**) covers **Gram -**

• **Postpartum endometritis** is 5-10x more common in patients with **Cesarean** delivery

💡 Because of the risk for puerperal sepsis following cesarean delivery, women typically receive a **prophylactic dose of antibiotics** at the **time of surgery.**

💡 **Chronic endometritis** shows **Plasma Cells** on biopsy.

Urinary Retention:

	Postpartum urinary retention
Risk factors	Primiparity Regional neuraxial anesthesia Operative vaginal delivery Perineal injury Cesarean delivery
Clinical features	Small-volume voids or inability to void Incomplete bladder emptying Dribbling of urine
Management	Self-limited condition Intermittent catheterization

postpartum urinary retention, defined as an **inability to void ≥6 hours after vaginal delivery** or ≥6 hours after urinary catheter removal following cesarean delivery. Patients with acute urinary retention can develop a concomitant **overflow incontinence**, resulting in clinical features such as urinary dribbling and lower abdominal pressure from an **overdistended bladder**.

Postpartum urinary retention often occurs due to the following:

- Perineal trauma from a prolonged second stage of labor and/or perineal laceration that results in a **pudendal nerve injury**. Damage to the pudendal nerve can result in a decreased voiding sensation, thereby promoting urinary retention, and cause external urethral sphincter dysfunction.
- Reduced sensory and motor sacral spinal cord impulses from regional neuraxial anesthesia (eg, **epidural anesthesia**), which can suppress the micturition reflex and decrease detrusor tone, resulting in **bladder atony**.

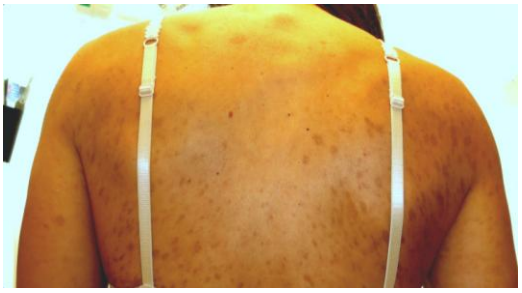
Other risk factors include primiparity and operative (forceps-assisted) vaginal delivery. Management is with intermittent urethral catheterization and reassurance as retention is typically self-limited and resolves in <1 week.

Infections:

	Syphilis manifestations
Primary	Painless genital ulcer (chancre)
Secondary	Diffuse rash (palms & soles) Lymphadenopathy (epitrochlear) Condyloma latum Oral lesions Hepatitis
Latent	Asymptomatic
Tertiary	CNS (tabes dorsalis, dementia) Cardiovascular (aortic aneurysm/insufficiency) Cutaneous (gummas)

Primary syphilis typically causes a painless genital chancre (ie, inoculation site) that can often go unnoticed. Untreated, primary syphilis progresses within weeks to months to secondary syphilis, which typically causes a **diffuse maculopapular rash** due to the widespread circulation of *Treponema pallidum*. The rash usually begins along the **skin-cleavage lines of the trunk** and extends to the extremities, **including the palms and soles**.

Because *T pallidum* crosses the placenta, patients are at increased risk for **obstetric complications**, including fetal growth restriction, as possibly demonstrated by this patient's lagging fundal height of 28 cm at 31 weeks gestation. Other complications include **congenital infection** and possible intrauterine fetal demise. Therefore, all pregnant patients are screened for syphilis at the initial prenatal visit; repeat third-trimester screening is indicated in patients with risk factors (eg, HIV) or limited or no prenatal care.

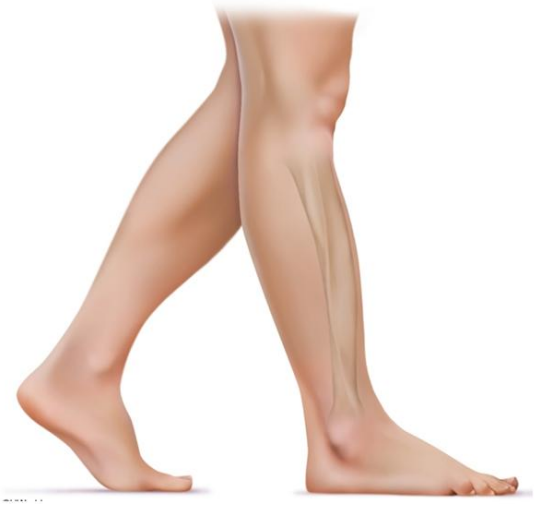


	Syphilis in pregnancy
Screening	Universal at first prenatal visit Third trimester & delivery (if high risk)
Serologic tests	Nontreponemal (RPR, VDRL) Treponemal (FTA-ABS)
Treatment	Intramuscular penicillin G benzathine
Pregnancy effects	Intrauterine fetal demise Preterm labor
Fetal effects	Hepatic (hepatomegaly, jaundice) Hematologic (hemolytic anemia, ↓ platelets) Musculoskeletal (long bone abnormalities) Failure to thrive

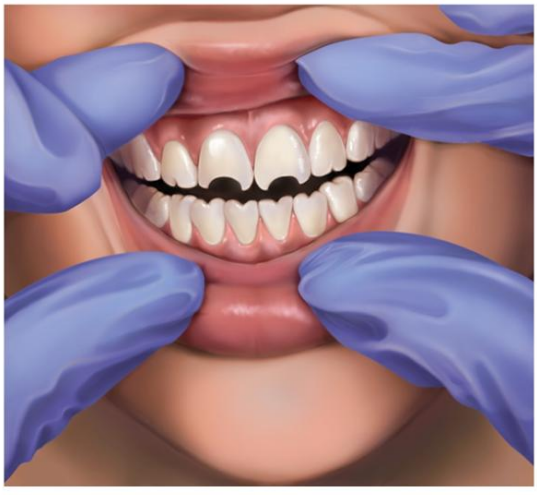
Desensitize if penicillin allergic

Congenital syphilis	
Clinical features	**Early*** • Snuffles: copious clear, purulent, or serosanguineous rhinorrhea • Maculopapular rash • ◦ Palms, soles, buttocks, legs usually involved ◦ Desquamation & hyperpigmentation • Long bone abnormalities (eg, metaphyseal lucencies) **Late** • Saddle nose, notched (Hutchinson) teeth, saber shins, sensorineural hearing loss
Diagnosis	• Serologic testing: VDRL, RPR • Darkfield microscopy: *Treponema pallidum* in infectious material (eg, nasal discharge, skin lesions, placenta)
Treatment	• Penicillin
Prevention	• First-trimester maternal screening; repeat if high risk (eg, other prenatal STI) • Prenatal penicillin
*Nonspecific findings: jaundice, hepatosplenomegaly, growth restriction, "blueberry muffin" spots.	

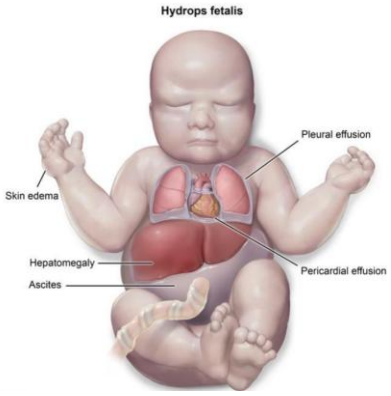
Saber shins



Hutchinson teeth



	Fetal hydrops
Pathogenesis	↑ cardiac output demand causing heart failure ↑ fluid movement into interstitial spaces (third spacing)
Clinical features	Pericardial effusion Pleural effusion Ascites Skin edema Placental edema Polyhydramnios
Etiology	Immune ○ Rh(D) alloimmunization Nonimmune ○ Parvovirus B19 infection ○ Fetal aneuploidy ○ Cardiovascular abnormalities ○ Thalassemia (eg, hemoglobin Barts)



fetal hydrops (hydrops fetalis), an excessive fluid accumulation in the interstitium (eg, **pericardial** and **pleural effusions**) that is due to high-output heart failure from either immune or nonimmune etiologies. Rh(D) alloimmunization is the most common etiology of immune fetal hydrops; in contrast, **maternal parvovirus B19 infection** causes nonimmune fetal hydrops and is likely the reason for this patient's presentation.

Parvovirus B19 infection in adults can be asymptomatic (or associated with nonspecific flulike symptoms or arthralgias); however, in children, it typically presents with a **slapped-cheek rash** that follows nonspecific prodromal symptoms (corresponding with viremia). The virus is transmitted via respiratory droplets. Pregnant women should avoid infected individuals because the virus can cross the placenta and have devastating fetal consequences.

Parvovirus B19 is highly cytotoxic to fetal red blood cell precursors, which can cause **severe fetal anemia** and result in **increased cardiac output demand** (as evidenced by fetal tachycardia with a fetal heart rate >160/min). When cardiac output can no longer compensate for worsening fetal anemia and hypoxemia, fetuses develop **high-output heart failure** and its sequalae: effusions, ascites (eg, enlarged abdominal circumference), and polyhydramnios. Fetal hydrops has a high risk of fetal demise, and affected patients require serial ultrasounds and possible intrauterine transfusion.

Preventing neonatal group B *Streptococcus* infection	
Antenatal screening	Rectovaginal culture at 36-38 weeks gestation
Indications for intrapartum prophylaxis	GBS bacteriuria or GBS urinary tract infection in current pregnancy (regardless of treatment) GBS-positive rectovaginal culture in current pregnancy Unknown GBS status PLUS any of the following: ◦ <37 weeks gestation ◦ Intrapartum fever ◦ Rupture of membranes for ≥18 hours Prior infant with early-onset neonatal GBS infection
Intrapartum prophylaxis	Intravenous penicillin
GBS = group B *Streptococcus*.	

Streptococcus agalactiae, or **group B *Streptococcus* (GBS)**, is a common colonizer of the maternal gastrointestinal and genital tract. Although GBS is typically asymptomatic in pregnant women, it can be vertically transmitted during vaginal delivery or after rupture of membranes, causing **early-onset neonatal GBS infection** (eg, sepsis, pneumonia). To prevent vertical transmission, most women are screened at 36-38 weeks gestation; those who are GBS-positive require intrapartum antibiotic prophylaxis (IAP).

Women with an **unknown GBS status**, such as this patient, are triaged based on risk factors. Most women at ≥37 weeks gestation do not require IAP as they are at low risk for vertical transmission, and indiscriminate antibiotic use can lead to bacterial resistance. In contrast, women with the following risk factors require IAP:

- **Rupture of membranes for ≥18 hours** (as in this patient) because the extended time period allows for possible GBS proliferation, increased bacterial load, and prolonged fetal exposure to infected amniotic fluid—all of which increase the **risk of vertical transmission** and neonatal infection
- Intrapartum fever, which indicates possible intraamniotic infection involving GBS
- Delivery at <37 weeks gestation, as the immature fetal immune system is more susceptible to infection

Women who meet criteria for IAP are typically administered intravenous penicillin

DRUGS:

	Magnesium toxicity
Clinical features	Mild: nausea, flushing, headache, hyporeflexia Moderate: areflexia, hypocalcemia, somnolence Severe: respiratory paralysis, cardiac arrest
Treatment	Stop magnesium therapy Give IV calcium gluconate bolus

Patients with preeclampsia with severe features are administered magnesium sulfate to prevent eclamptic seizures because magnesium acts as a calcium channel blocker on the CNS, thereby raising the seizure threshold. Therapeutic serum magnesium levels (ie, 4.8-8.4 mEq/L), which are 2-4 times higher than normal concentrations, are required to prevent eclampsia. Normally, this therapeutic range is maintained between the balance of magnesium sulfate infusion and renal excretion of magnesium. However, in patients with abnormal renal function—as evidenced by either an elevated creatinine level or decreased urine output (as in this patient with minimal, dark urine)—magnesium can be retained and increase to toxic levels.

High levels of magnesium inhibit presynaptic acetylcholine release, thereby causing neuromuscular inhibition. Therefore, the first sign of **magnesium sulfate toxicity** is often hyporeflexia. As magnesium levels increase, patients can develop **areflexia** (eg, loss of patellar reflexes) and **respiratory depression** (eg, **drowsiness**, respirations ≤12/min). If untreated, patients are at risk for respiratory paralysis and cardiac arrest. Treatment is immediate cessation of magnesium sulfate and administration of **calcium gluconate**.

- ***Hypocalcemia*** *due to temporary* ***suppression of PTH***

Magnesium is solely excreted by the **kidneys**

thus patients with renal insufficiency are at increased risk for toxicity

- **Inhaled B agonist** and **inhaled corticosteroids** are **safe** to use in pregnancy.

Illicit drug abuse in pregnancy	
Risk factors	• Adolescent pregnancy • Late/noncompliant prenatal care • Inadequate pregnancy weight gain
Obstetric complications	• Spontaneous abortion • Preterm birth • Preeclampsia • Abruptio placentae • Fetal growth restriction • Intrauterine fetal demise

Uterotonic

What are the (*4*) uterotonic agents?

- **Oxytocin**
- **Carboprost tromethamine** (*Hemabate*): PGF2 analog
  - **CI in asthma**
- **Methylergonovine** (*Methergine*): Ergot alkaloid
  - **CI in htn**

- **Misoprostol**: Prostaglandin
  - also used in abortions, cervical ripening, labor induction

Oxytocin	
Indications	• Induction or augmentation of labor • Prevention & management of postpartum hemorrhage
Adverse effects	• Hyponatremia • Hypotension • Tachysystole

💡 Oxytocin is **not effective** in stimulating uterine contractions or expelling retained products of conception during the **first** or **second trimesters** because **few oxytocin receptors** are in the uterus during early pregnancy.

- What are the (*3*) complications of **oxytocin toxicity**?

  *Released from posterior pituitary with **ADH a**nd both have similar structure*

- **Hyponatremia** (*seizures*)
  - Treat with **hypertonic saline**
- **Hypotension**
- **Uterine Tachysystole (***>5 contractions in 10 minutes***)**

Other:

	Disseminated intravascular coagulation
Major causes	Sepsis Severe traumatic injury Malignancy Obstetric complications
Pathophysiology	Procoagulant excessively triggers coagulation cascade → Formation of fibrin-/platelet-rich thrombi & fibrinolysis → Bleeding & organ damage (eg, kidneys, lungs)
Laboratory findings	Thrombocytopenia Prolonged PT & PTT ↓ Fibrinogen ↑ D-dimer Microangiopathic hemolytic anemia (schistocytes)

Obstetric complications (eg, postpartum hemorrhage, placental abruption) are a major cause of DIC due to the large volume of bleeding associated with these conditions.

Significant bleeding can cause release of tissue factor (thromboplastin), which leads to uncontrolled activation of the coagulation cascade and a **consumptive coagulopathy**. This leads to the formation of fibrin- and platelet-rich thrombi, which consume platelets (ie, **thrombocytopenia**), and coagulation factors (evidenced by **prolonged PT/INR and PTT**), and fibrinogen. Fibrinolysis is then triggered to degrade the clots, which elevates D-dimer (a fibrin degradation product) and depletes antithrombin and proteins C and S. This results in a paradoxical **thrombosis.**

The thrombi in the vasculature also often shear red blood cells, leading to a **microangiopathic hemolytic anemia** (evidenced by elevated total bilirubin levels).

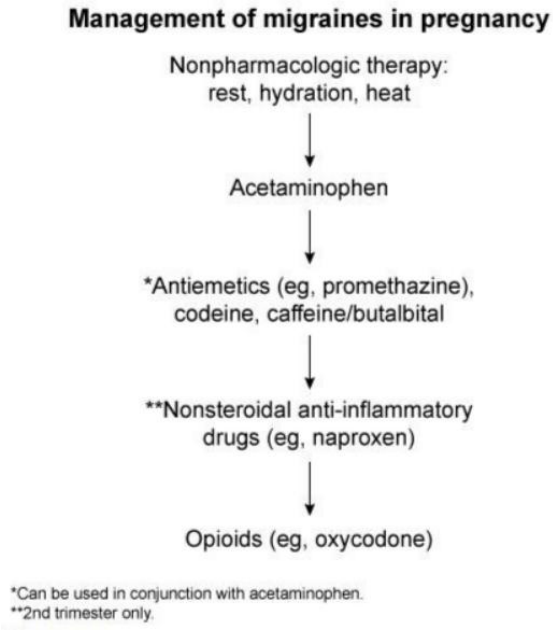
Patients with DIC require emergency supportive care, including treatment of the underlying etiology (eg, uterine atony causing postpartum hemorrhage) and resuscitation with blood products (eg, packed red blood cells, fresh frozen plasma).

	Thrombocytopenia in pregnancy
Gestational	Isolated, mild (100,000-150,000/mm^3) Asymptomatic Diagnosis of exclusion
Preeclampsia with severe features/ HELLP syndrome	Moderate to severe (<100,000/mm^3) Hypertension ± headache/scotomata ± ↑ Creatinine, ↑ AST & ALT
Immune-mediated thrombocytopenia (ITP)	Isolated, moderate to severe (<100,000/mm^3) Asymptomatic or mucosal bleeding/bruising Normal PT, aPTT
Thrombotic thrombocytopenic purpura (TTP)	Severe (<30,000/mm^3) Neurologic symptoms (eg, confusion, seizure), fever, abdominal pain, petechiae Normal PT, aPTT
Disseminated intravascular coagulopathy (DIC)	Moderate to severe (<100,000/mm^3) Bleeding (eg, oozing intravenous sites) ± thrombosis ↑ PT, ↑ aPTT, ↓ fibrinogen

gestational thrombocytopenia, a benign condition that causes an isolated, mild thrombocytopenia (ie, **platelets 100,000-150,000/mm^3**). Gestational thrombocytopenia occurs in 5%-10% of pregnancies and is likely due to physiologic increases in plasma volume during pregnancy (ie, dilutional effect). It is commonly diagnosed in the **third trimester** on a routine complete blood count, but can arise as early as the first trimester. Patients are **asymptomatic** (ie, no bruising, bleeding, or anemia) or have normal physiologic symptoms of pregnancy, such as fatigue, shortness of breath, and symmetric leg swelling.

Patients with isolated, asymptomatic, and mild thrombocytopenia in pregnancy are assumed to have gestational thrombocytopenia. Because this condition does not affect obstetric or neonatal outcomes and because platelet counts usually normalize within 6 weeks of delivery (ie, self-limited), management includes **reassurance and observation**. In contrast, symptomatic or moderate to severe thrombocytopenia (ie, <100,000/mm^3) requires evaluation for an alternate diagnosis (eg, thrombotic thrombocytopenic purpura).

During **pregnancy**, headaches are common and typically benign. Headaches with atypical features (eg, altered mental status, neurologic deficits) or in patients ≥20 weeks gestation require evaluation to exclude other etiologies, such as preeclampsia or cerebral venous thrombosis, due to risk of adverse maternal (eg, stroke, disseminated intravascular coagulation) and fetal (eg, growth restriction, demise) outcomes.



Management of migraines in pregnancy is complicated due to limited therapeutic options.

- **Acetaminophen** is the preferred **first-line** option; patients also typically benefit from caffeine and limiting exposure to light and sound (eg, resting in a dark room).
- In patients **who do not improve** with acetaminophen alone, a low-potency opioid (eg, **acetaminophen-codeine**), antiemetics (eg, promethazine), or caffeine/butalbital may be beneficial.
- More potent opioids (eg, oxycodone) are typically not used due to their tendency to worsen gastrointestinal symptoms (eg, constipation, nausea) during pregnancy; however, they can be considered if all other options fail to improve symptoms.

Parenteral antiemetics (eg, metoclopramide) are used acutely and are effective monotherapy.

Back Pain:

Low back pain during pregnancy	
Etiology	• Enlarged uterus → exaggerated lordosis • Joint/ligament laxity from ↑ progesterone/relaxin • Weak abdominal muscles → decreased lumbar support
Risk factors	• Excessive weight gain • Chronic back pain • Back pain in prior pregnancy • Multiparity
Imaging	• Not indicated
Management	• Behavioral modifications • Heating pads • Analgesics

• What is the *recommended management* for a woman in the third trimester of pregnancy that presents with **lower back pain** that radiates down the legs, especially with activity? Physical exam is benign.

Reassurance and Conservative management

The management includes:

- **Reassurance and behavioral modifications**
  - ***Lifestyle interventions*** *(wear-low heeled shoes, good back support, heat/cold/massage to the painful area)*
  - ***Rest*** *with hip flexion*
  - *Exercise*
  - *Medication: Short course analgesic →* ***acetaminophen*** *has best safety profile in pregnancy*

Round Ligament Pain

• Round ligament pain develops when the ligament is stretched by the **gravid uterus**, usually in the **late second trimester** and the **third trimester**.

It typically presents as a **sharp unilateral pain** that **radiates to the vagina**, is triggered by **movement** (ie, positional), and **resolves without treatment**.

Pseudocyesis:

What is **pseudocyesis**?

- "False pregnancy"
- Women present with classic signs of pregnancy *(e.g. morning sickness, amenorrhea, abdominal distention)* despite it being ruled out with **U/S and negative pregnancy test.**

• Pseudocyesis can occur when **psychological pressures** (eg, difficulty conceiving), social pressures, and the **somatization of stress** affect the hypothalamic-pituitary-ovarian axis, or when bodily changes (eg, weight gain, amenorrhea) are misinterpreted by the patient.

The end result is a **deeply held, nondelusional belief of being pregnant** that may be strong enough to cause a patient to misread a negative home pregnancy test result as positive

Because pseudocyesis is a form of **somatization**, management requires **psychiatric evaluation** and treatment.

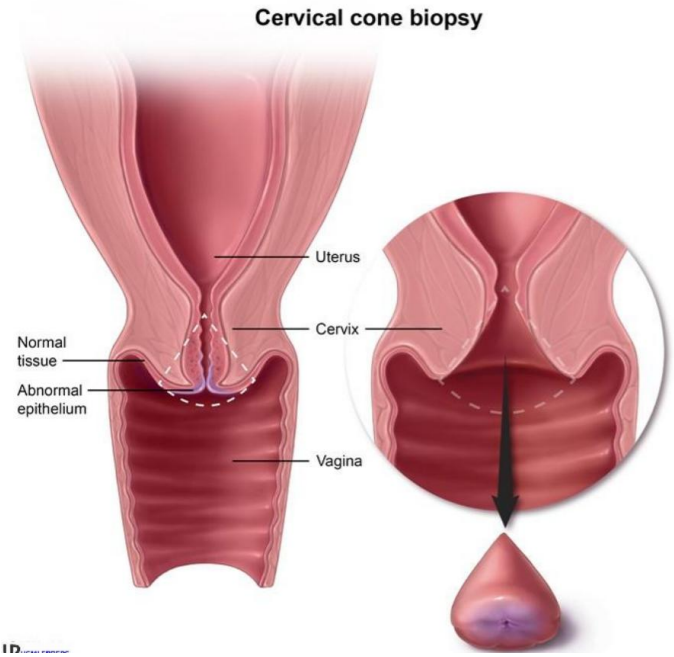
	Cervical excisional procedures
Indications	Cervical intraepithelial neoplasia grades 2 & 3*
Procedure types	Cold knife cone Loop electrosurgical excision procedure
Complications	Cervical stenosis Preterm birth Preterm prelabor rupture of membranes Second-trimester pregnancy loss
*Observation preferred for cervical intraepithelial neoplasia 2 in young women.	

Cervical excisional procedures (eg, loop electrosurgical excision procedure, CKC) excise the entirety of the transformation zone with surrounding areas of the endocervical canal. Although these procedures are highly effective cervical dysplasia treatments, they have clinically significant **late-developing sequelae**, particularly CKCs, which allow greater depth and access to the endocervical canal.

Specific pregnancy complications, including the following, occur because removal of the dysplastic tissue also excises normal cervical stroma and cervical glands:

- Loss of cervical stroma is thought to **reduce tensile strength** and cervical plasticity, which can result in either a **second-trimester pregnancy loss** or a **preterm birth**.
- Loss of cervical glands reduces cervical mucus (ie, a barrier to infection), which **increases the susceptibility to ascending infection** and increases the risk for **preterm prelabor rupture of membranes** (because infection is a trigger for rupture of membranes).

Pregnant patients with a history of CKC may undergo cervical length measurements to evaluate their risk for preterm birth.


Cervical cone biopsy
Uterus
Cervix
Normal tissue
Abnormal epithelium
Vagina