EMERGENCIES AFTER 20 WEEKS OF PREGNANCY

THROMBOEMBOLIC DISEASE OF PREGNANCY:

PATHOPHYSIOLOGY:

- Pregnancy induces a HYPERCOAGULABLE STATES → pregnant woman is 5-10 more likely to develop DVT/PE than nonpregnant woman
    - Hypercoagulable state likely due to haematologic and physiologic changes

CLINICAL FEATURES:

- Maintaining a high clinical suspicion is essential for the evaluation and diagnosis of DVT and PE, because delayed diagnosis is associated with significant morbidity and mortality
- Traditional signs are nonspecific as they also occur in pregnancy → SOB, tachycardia, tachypnoea, lower extremity oedema
- RF are outlined below:

Table 104-1 Risk Factors for Thromboembolic Disease in Pregnancy
Black race
Heart disease
Diabetes
Lupus erythematosus
Smoking
Obesity
Advanced maternal age
Assisted reproduction with ovarian hyperstimulation
Multiparity
Hypercoagulable states
Antiphospholipid syndrome
Factor V Leiden mutation,
Antithrombin deficiency
Protein C deficiency
Protein S deficiency

DIAGNOSIS:

- DVT:
    - Up to 24% of DVT are complicated by PE, so early diagnosis is essential
    - Compression US is the test of choice (sensitivity and specificity for detecting DVT is 95-96%) → less accurate for isolated calf and iliac vein thromboses.
    - MR direct thrombus imagines is an alternative
    - Utility of D-dimer remains controversial
- PE:
    - Pregnant women with symptoms suggestive of PE for whom findings on compression US are positive should receive anticoagulation without waiting for confirmatory diagnostic studies
    - Patients with normal compression US → further studies mandated and should not be withheld due to fear of radiation exposure to the foetus

- CXR to exclude other causes of chest pain → pneumothorax and pneumonia
- Options are CTPA and VQ
  - CTPA exposes foetus to less radiation than VQ at ALL STAGES OF PREGNANCY
  - Risk of subsequent childhood cancer is higher with VQ (1 case per 280,000) than with CT (1 case per million), but risk of maternal breast cancer is higher with CT

TREATMENT:

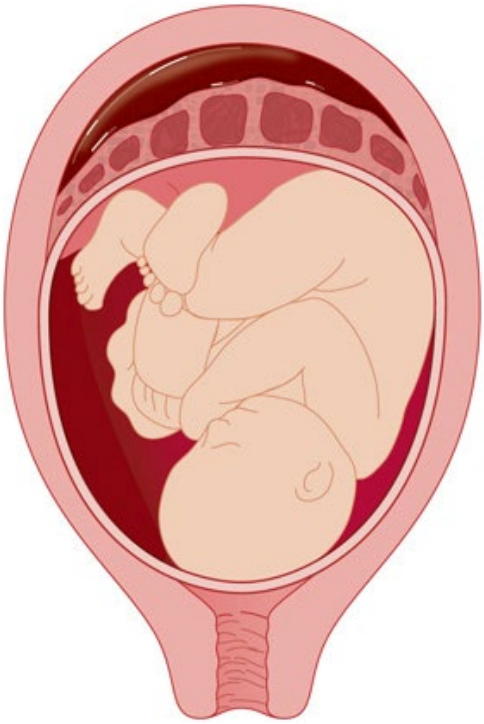
- DVT/PE are treated with either unfractionated heparin or LMWH
- UFH does not cross the placenta and does not result in foetal haemorrhage or teratogenesis
- Adverse effects of UFH → HIITS, haemorrhage, heparin-induced osteopenia.
- UFH preferred over LMWH in massive PE, those likely to bleed, patients in labour, those receiving regional anaesthesia, those undergoing LSCS
- LMWH is otherwise safe → does not cross the placenta and is associated with less HIITS/osteopenia than UFH → monitor with Anti-Xa levels
- IVC filters should be considered in those who cannot be anticoagulated
- WARFARIN SHOULD BE AVOIDED IN PREGNANCY BECAUSE IT CROSSES THE PLACENTA AND IS TERATOGENIC
- Thrombolytic therapy has not been extensively studied and is thus reserved for dire cases in which the mother's life is endangered → risk of foetal loss in these circumstances is high

<u>VAGINAL BLEEDING IN THE SECOND HALF OF PREGNANCY:</u>

- Causes of vaginal bleeding in the second half of pregnancy include:
  - ABRUPTIO PLACENTAE
  - PLACENTA PREVIA
  - PREMATURE LABOUR
  - LESIONS OR LACERATIONS OF THE VAGINA OR LOWE RGENITAL TRACT
- Severe haemorrhage can occur that can cause significant morbidity and mortality to mother and foetus

ABRUPTIO PLACENTAE:

- PREMATURE SEPARATION OF A NORMALLY IMPLANTED PLACENTA FROM THE UTERINE LINING



- ABRUPTION occurs spontaneously but can be associated with trauma (even minor)
- Most common RF is HT → postulated to cause placental inflammation and ischaemia that may lead to abruption
- Other risk factors are outlined below:

Table 104-8 Risk Factors Associated with Abruptio Placentae
Hypertension
Chronic or pregnancy induced
Trauma
Even minor trauma may be associated with abruptio placentae
Smoking
Advanced maternal age
Cocaine abuse
Black race
History of cesarean or other uterine surgery
Previous abruptio placentae

- Abruption can be COMPLETE, PARTIAL OR CONCEALED
- Clinical features → painful vaginal bleeding, severe uterine pain or tenderness, uterine hypertonicity and hypotension
    - High index of suspicion required as symptoms may be subtle
    - The diagnosis must be considered in all pregnant females near term who present with painful vaginal bleeding

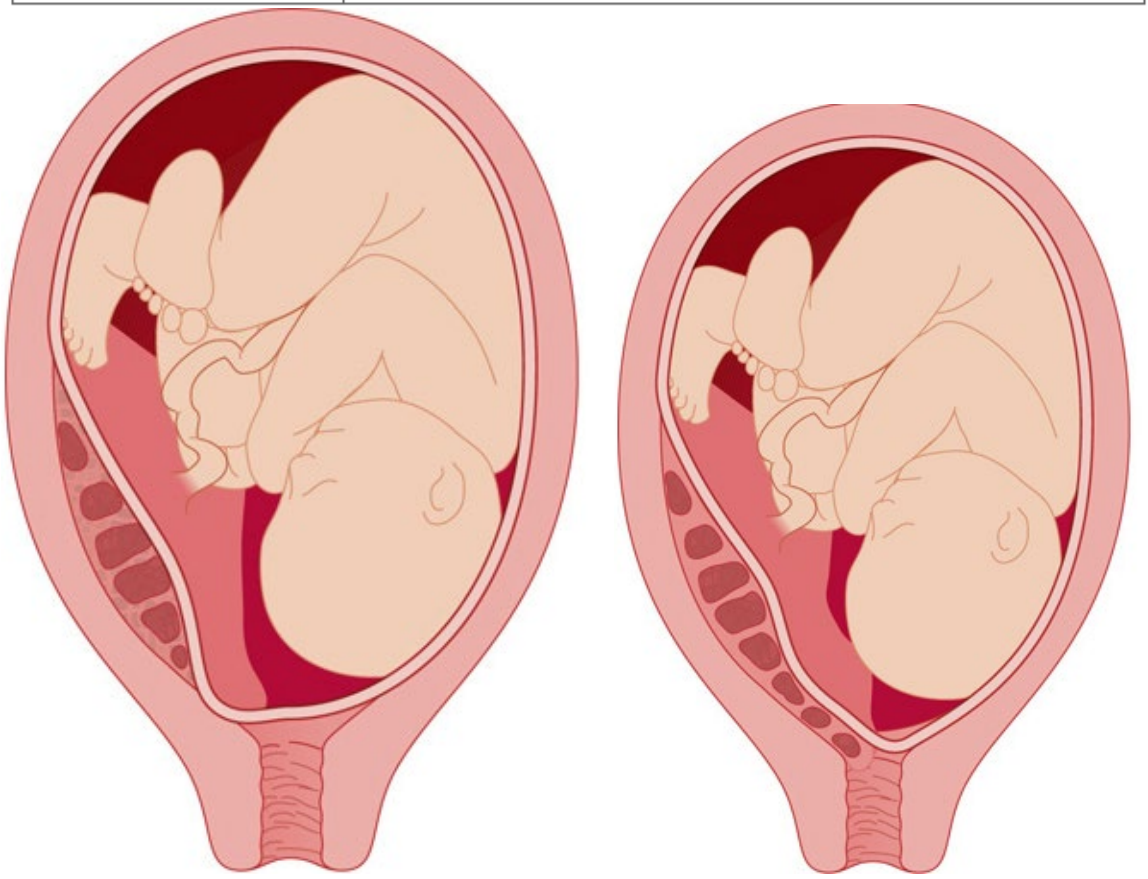
- Maternal complications → haemorrhagic shock DIC, uterine rupture, multi-organ failure
- Neonatal effects include neurodevelopmental abnormalities and death
- If abruption considered → two large bore IV and FBC/G+H
    - Administer blood products as appropriate
    - Fluid resuscitation and foetal monitoring if beyond 24 weeks
- US is fairly specific, but less so for detection of retroplacental clot
- IMMEDIATE O&G CONSULT REQUIRED → immediate delivery warranted in patients with severe abruption and haemorrhage

PLACENTA PREVIA:

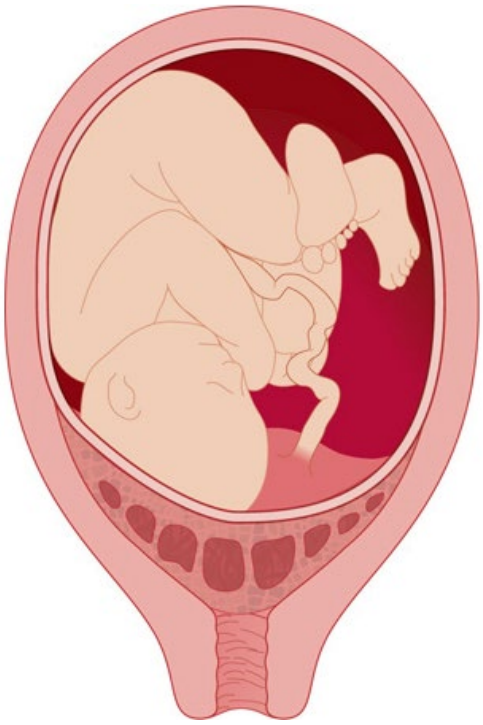
- Defined as a placenta that extends near, partial over or beyond the internal cervical os

Table 104-9 Subclasses of Placenta Previa

Subclass	Description
Marginal placenta previa	Placenta is near the os but does not cover it (**Figure 104-2**).
Partial placenta previa	Placenta partially covers the os (**Figure 104-3**).
Complete placenta previa	Placenta covers the internal os (**Figure 104-4**).



Marginal (left) and partial (right) placenta previa



Complete placenta previa

- If placenta previa is suspected, insert tow large-bore IVC → avoid speculum or PV exam if there is third-trimester bleeding, because this may precipitate catastrophic haemorrhage and death
- URGENT O&G CONSULT

VASA PREVIA:

- A rare cause of late-pregnancy bleeding
- Incidence is ~1 in 2500 births
- Reefers to insertion of the umblical cord into the lower uterine segment in such a way that it traverses foetal membranes before it inserts into the placenta
- Haemorrhage associated with vasa previa usually occurs with spontaneous rupture of membranes and is seldom recognised prior to vessel disruption → perinatal mortality ranges from 30-100%

PREMATURE RUPTURE OF MEMBRANES AND PRETERM BIRTH:

- Preterm birth may occur spontaneously (as result for PROM) or may be intentionally induced for medical reasons
    - PROM is defined as rupture of membranes prior to onset of labour, if this occurs before 37 weeks, it is called preterm PROM (pPROM)
- Keys to diagnosis of PROM are shown below:

Table 104-10 Keys to Diagnosis of Premature Rupture of Membranes	
Information Obtained	**Comments**
History	
Gush of fluid and continued leakage of fluid	
Details of contractions	Determine if active labor is in process.
Date of last menstrual period	Use to calculate estimated date of delivery and gestational age. Gestational age is number of weeks from first day of last menstrual period.
Vaginal bleeding	Raises concern for placenta praevia.
Recent intercourse	
Fever	Infection raises fetal and maternal risk.
Physical Examination	
Measurement of fundal height	
Auscultation of fetal heart tones	
Sterile speculum examination	Check for:
	Cervical dilatation and effacement
	Pooling in vagina of fluid leaking from cervix
	If no fluid is noted, apply fundal pressure or ask patient to perform a Valsalva maneuver or cough
Laboratory Evaluation	
Vaginal fluid	Test with nitrazine paper
	Blue color indicates pH >6.5 signaling presence of amniotic fluid
	Note: Blood, semen, and antiseptics may cause false positive result
Swab of vaginal walls or posterior fornix	Examine glass slide preparation for ferning.
	Ferning indicates presence of amniotic fluid.
	Blood may obscure ferning.
	Mucus results in a false positive fern test finding.
	Test for *Chlamydia*, *Neisseria gonorrhoeae*, group B streptococci, bacterial vaginosis.

- TREATMENT OF PROM:
  - All women suspected of PROM/preterm labour require O&G input
  - Conservative therapy, combined with corticosteroids to hasten infant lung maturity and antibiotics to prevent infection has been associated with pregnancy prolongation and reduction in infant and maternal complications
  - A dose of BETAMETHASONE 12mg IM, or DEXAMETHASONE 6mg IM Q12H FOR 2 DAYS → NOT GIVEN AFTER 34 WEEKS GESTATION

- Administration of IV antibiotics can decrease neonatal infeciotns, prolong latency and reduce postpartum endometritis, chorioamnionitis, neonatal sepsis, neonatal pneumonia and intraventricular haemorrhage → BENZYLPENICILLIN
- Role of TOCOLYSIS REMAINS CONTROVERSIAL → magnesium, CCB → candidates are women in preterm labour at between 24-36 weeks gestation → decision should be made by an obstetrician

POSTPARTUM HAEMORRHAGE:

- The most common cause of maternal mortality worldwide and remains among the tope three of maternal mortality in developed nations
- Usually occurs within the FIRST 24 HOURS → PRIMARY PPH:
  - UTERINE ATONY
  - RETAINED PLACENTAL FRAGMENTS
  - LOWER GENITAL TRACT LACERATIONS
  - UTERINE RUPTURE
  - UTERINE INVERSION
  - HEREDITARY COAGULOPATHY
- Initial treatment of PPH should focus on aggressive fluid resuscitation and severe haemorrhage may require transfusion of blood products to correct anaemia/coagulopathy
- Apply bimanual compression/uterine massage as required
- US to identifty retained products and genital tract lesions should be repaired
- Pharmacological treatment options are outlined above

Table 104-11 Pharmacologic Treatment of Postpartum Hemorrhage

Drug	Comments
Oxytocin, 10 milligrams IM or slow IV push	First-line treatment
	Uterotonic agent
	Rapid administration may cause hypotension
Methylergonovine, 0.2 milligram IM	Ergot
	Contraindicated in patients with hypertension or preeclampsia
Misoprostol, 600 micrograms SL[47]	Prostaglandin
	Side effects: nausea, vomiting, diarrhea
Carboprost, 250 micrograms IV[47]	Prostaglandin
	Side effects: nausea, vomiting, diarrhea, hypertension, bronchospasm
	Avoid in patients with hypertension or asthma

- Uterine atony can occur from overdistention of the uterus due to foetal macrosomia, polyhydramnios or multifoeatl gestation → patients may require large doses of oxytoxin and fluid resusciations
- Abnormal placental implantation may also lead to severe haemorrhage and may require emergency peripartum hysterectomy (placenta increta or eccreta)

UTERINE RUPTURE:

- A rare complication of labour in an unscarred uterus (rate of 0.01%)
- Today, previous LSCS is the primary risk factor for uterine rupture → rate of intrapartum uterine rupture secondary to prior LSCS is estimated at 0.2-0.8% → uterine rupture has significant high maternal and foetal mortality and morbidity rates
- Other risk factors associated with uterine rupture are malpresentation, labour dystocia and hypertensive disorders
- RISK OF RUPTURE IS TEN TIMES HIGHER IN THE SCARRED UTERUS THAN IN THE UNSCARRED UTERUS
- Clinical signs associated with uterine rupture are:
    - Persistent abdominal pain
    - Vaginal bleeding
    - Loss of foetal station
    - Palpable uterine defect
        - Foetal monitors may show foetal distress and bradycardia
        - Diagnosis needs to be made clinically and quickly
- TREATMENT:
    - Blood product replacement
    - Emergent delivery of the foetus
    - Frequently → hysterectomy is required for stabilisation

AMNIOTIC FLUID EMBOLUS:

- AN UNEXPECTED COMPLICATION OF PREGNANCY THAT OCCURS WHEN AMNIOTIC FLUID, FOETAL CELLS, HAIR OR OTHER DEBRIS ENTER THE MATERNAL CIRCULATION
- Presenting signs:
    - Respiratory distress
    - Pulmonary oedema
    - Hypoxia
    - Altered mental status
    - Seizures
    - Sudden maternal CV collapse
    - DIC
    - MATERNAL DEATH
- REMAINS A DIAGNOSIS OF EXCLUSION
- Mortality rates are between 60-80% → over 85% of survivors have neurologic sequelae → RF include LSCS, advanced maternal age, multiparity, abnormal placental implantation, uterine rupture and eclampsia
- Death can occur withn an hour of symptosm → rapid initiation of treatment crucial
    - Main aim is to prevent/treat hypoxia
    - Place mum in left lateral decubitus position
    - Pressors and blood products may be needed
    - Case reports of continous haemofiltration and ECMO being used → usually not enough time

- IMMEDIATE DELIVERY OF THE INFANT IS CRUCIAL → consider perimortem LSCS within 5 minutes of cardiac arrest

PERIPARTUM CARDIOMYOPATHY:

- A dilated cardiomyopathy that can occur at any stage of gestation → most classically occurs in last month of gestation or within first 5 months after delivery → cause is unknown
- Present with symptoms of CHF → ECG, CXR, TSH, EUC to exclude other causes of CCF
- ECHO → will show EF <45% and increased LV end-diastolic dimension → standard treatment for CHF and pulmonary oedema is instituted → except NITROPRUSSIDE IS RELATIVELY CONTRAINDICATED IN THE PREGNANT WOMAN BECAUSE IT CAN CAUSE THIOCYANATE AND CYANIDE ACCUMULATION

POSTPARTUM ENDOMETRITIS:

- Most postpartum infections are identified after hospital discharge
- A postpartum woman who presents with a persitant fever is assumed to have a genital tract infection until proven otherwise → pelvic infeciotn is the most common serious complication of the puerperium
- Risk factors are outlined below:

Table 104-12 Risk Factors for Postpartum Endometritis
Cesarean section*
Multiple gestation
Younger maternal age
Long duration of labor and membrane rupture
Internal fetal monitoring
Low socioeconomic level
Digital examination after 37 wk of gestation
Maternal human immunodeficiency virus infection

- Most common pathogens are those that reside in the bowel and colonise the perineum, vagina and cervix, most infections are polymicrobial
- Patients typically present with persistent fever, foul-smelling lochia, leukocytosis, tachycardia and uterine tenderness. Only scant discharge may be present, especially in GBS infection
- MAINSTAY OF TREATMENT IS ANTIBIOTICS, DRAINAGE OF ABSCESSES AND PURULENT MATERIAL AND DEBRIDEMENT OF NECROTIC TISSUE
- ANTIBIOTICS ARE OUTLINED BELOW:

Table 104-13 Inpatient Treatment Regimens for Postpartum Endometritis
Cefoxitin (2 grams IV every 6 h)
or
Cefotetan (2 grams IV every 12 h)
or
Cefotaxime (2 grams IV every 6 h)
or
Clindamycin (500 milligrams IV every 6 h) *plus* gentamicin (4.2 milligrams/kg IV daily)
or
Amoxicillin (2 grams IV every 4 h) *plus* gentamicin (4.2 milligrams/kg IV daily)
or
Metronidazole (500 milligrams IV every 8 h) *plus* ampicillin (2 grams IV every 4 h) *plus* an aminoglycoside

- Mild infections can be treated with oral antibiotics → clindamicin (doxycycline is an option if NOT BREASTFEEDING)
- Any patient who appears toxic, has had a LSCS or has underlying comorbid infection should be hospitalised for parenteral therapy → combination of ampicillin or clindamicin plus gentamicin is sufficient for 90% of patients
- COMPLICATIONS OF ENDOMETRITIS:
    - Parametrial phlegmons
    - Surgical, incisional and pelvic abscesses
    - Infected haematomas
    - Septic pelvic thrombophlebitis
    - Necrotising fasciitis → high mortality, associated with obesity, DM and hypertension
    - Peritonitis