Complications of third stage of labor & Post Partum Hemorrhage:

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The third stage of labor involves the separation and expulsion of placenta.

The third stage is most important because ***it is a primary factor in determining whether postpartum hemorrhage (PPH) will occur.*** The duration of the 3rd stage normally lasts anywhere between 5 minutes to an hour, depending upon whether it is actively managed or allowed to progress spontaneously.

Separation of placenta occurs because of the reduction of volume of the uterus due to uterine contraction and the retraction of the myometrial muscle fibers. A cleavage plane develops within the decidua basalis and the separated placenta lies free in the lower segment of the uterine cavity. Management of the 3rd stage of labor can be described as "active" and "physiological".

Physiological management:

- ***It is where the placental delivered by maternal effort and no uterotonic drugs are given to assist this process***.it is associated with heavier bleeding, but women who are not under risk of PPH should be supported if they choose this option.
- in the event of hemorrhage (estimated blood loss >500ml) or if the placenta remains undelivered after 60 minutes of physiological management, active management should be recommended.

Active management:

It should be recommended to all women because high quality evidence shows that it reduces the incidence of PPH from 15% to 5%.

It includes the following:

- Intramuscular injection of 10 units of oxytocin, given as the anterior shoulder o the baby is delivered, or immediately after delivery of the baby.
- Early clamping and cutting of the umbilical cord.
- Controlled cord traction, when the signs of placental separation are recognized:
  - Apparent lengthening of the cord.
  - A small gush of blood from the placental bed.
  - Rising of the uterine fundus to above the umbilicus.
  - Uterine contraction resulting in firm globular feel on palpation.

It is now recognized that ***a modified approach to active management of the 3rd stage*** may be preferable with ***delayed cord clamping for between 1 and 3 minutes***. This approach allows

autotransfusion of placental blood to the neonate while maintaining the benefit of a reduced risk of PPH. It is a particular importance in preterm labor.

- So the most common complication of this stage is the PPH with all its sequel as it will be discussed below.
- In approximately 2% of cases, the placenta will not be expelled by this method. If no bleeding occurs, a further attempt at CCT should be made after 10 minutes. If this fails, ***the placenta is "retained"*** and manual removal should be done under LA or GA. After completion of the 3rd stage of labor, the placenta should be inspected for any missing cotyledons or succenturate lobe. If these are suspected examination and manual removal of placental tissue (MROP) under anesthesia should be arranged, because in this situation the risk of PPH is high.
- ***The vulva should be inspected for any tears or lacerations.*** Minor tears do not require suturing, but tears extended into ***the perineal muscles (or an episiotomy) or injuries extending to urethra or rectum*** (2nd &3rs degree perineal tears) will require careful repair.
- ***Uterine inversion*** is another but rare complication happened in the 3rd stage of labor, which may be occur if the uterus is not adequately controlled with left hand and excessive traction is exerted on the cord in the absence of complete separation and a uterine contraction.
- ***Coagulopathy, amniotic fluid embolism and obstetric shock*** are other 3rd stage of labor complications with high maternal mortality and morbidity

Postpartum Hemorrhage (PPH):

It is defined as blood loss in excess of 500 ml, at the time of vaginal delivery or blood loss in excess of 1000 ml, following C/S. it is a leading cause of maternal mortality.

The excessive blood loss is ***usually occurs in the immediate postpartum period, but it can occur slowly over the first 24 hours and it is known as primary PPH.***

Delayed PPH or secondary PPH can occasionally occur, with the excessive bleeding commencing more than 24 hours after delivery. This is usually due to subinvolution of the uterus and disruption of placental site "scab" several weeks postpartum or to the retention of placental fragments that separate several days after delivery.

The most common causes of PPH are:

1. Uterine atony (commonest cause of hemorrhage) may resulted from: macrosomia, multiple pregnancy, prolonged labor, oxytocin use, induction of labor, grand multiparity, polyhydramnious, APH (due to placenta Previa or accrete), placental abruption.

2. Retained placental tissues. (Both of these conditions result in retained blood clots & placental fragments, causing uterine stretching and prevention of uterine contractions).
3. Genital tract trauma (there are 3 main areas from which hemorrhage may occurs: uterus& placenta, cervix and vagina.
4. Uterine rupture or inversion.
5. Coagulation disorder (e.g. factor VIII deficiency) or DIC.
6. Amniotic fluid embolism.
7. Retained dead fetus.

The four T's mnemonic can be used to identify and address the most common causes of PPH (uterine atony {Tone}; laceration, hematoma, inversion, rupture {Trauma}; retained tissue or invasive placenta {Tissue}; and coagulopathy {Thrombin}.

Since 1996, there has been a gradual increase in the incidence of PPH in US & other developed countries and this has been related to uterine atony. The cause of this is not known and is currently under intense investigation.

- ***Uterine atony:*** the majority of PPH cases (75-80%) are due to uterine atony.
  Factors predisposing to postpartum uterine atony:
  - History of PPH.*
  - Prolonged labor*
  - Grand multiparity (a parity of 5 or more).*
  - Overdistension of the uterus (multiple gestation, polyhydramnious or fetal macrosomia)^
  - Oxytocic augmentation of labor^
  - Precipitated labor (lasting less than 3 hours)
  - Magnesium sulfate treatment of preeclampsia^
  - Chorioamnionitis^
  - Halogenated anesthetics
  - Uterine leiomyoma^.
  - Vit. D deficiency (vit. D is known to play an important role in muscle function, and muscle is component of both the uterus and vascular system.
  - Genetic and epigenetic factors (maternal, environmental and fetal)

*high-risk patients ^medium-risk patient

Most of blood loss due to uterine atony is from myometrial spiral arterioles and decidual veins that previously supplied and drained the intervillous space of placenta. As the contractions of the partially empty uterus cause placental separation, bleeding occurs and continues until the uterine musculature contract around the blood vessels and act as a physiologic-anatomic ligature. ***Failure of the uterus to contract after placental separation (uterine atony) leads to excessive placental site bleeding.***

**Management of uterine atony:*

- When uterine atony is determined the cause of the PPH, ***a rapid continuous IV infusion of dilute oxytocin (40-80U in 1L of N/S)*** should be given to increase uterine tone. If the uterus remains atonic, and the placental site bleeding continues, ***0.2 mg of ergonovine maleate or methylergonovine may be given intramuscularly***. The ergot drugs are relatively contraindicated in hypertensive patients because the smooth muscle-constricting effect of these drugs may also increase vascular tone and also increase blood pressure to dangerous levels.
- ***Analogues of prostaglandin F2alpha given IM*** are quite effective in controlling PPH caused by uterine atony. ***The 15-methyle analogue carboprost (Haemobate)*** has a more potent uterotonic effect and longer duration of action than the parent compound. The expected time of onset of the uterotonic effect when carboptost is given IM (0.25 mg) is 5 minutes, with a peak effect around 15 -20 minutes. When injected into the myometrium, its effect may be more rapid. An alternative next level drug is misopristol 800-1000microgram.
- If these pharmacologic treatment s fail, ***a bimanual compression and massage of uterine corpus*** may control the bleeding and cause the uterus to contract.
- ***Uterine cavity packing is no longer widely practiced***, but it may occasionally control PPH and obviate the need for surgical intervention. Alternatively, ***a large –volume balloon catheter*** has been developed that perform a similar function.
- If uterine bleeding persists in a stable patient, an interventional radiologist may be able to place ***a percutaneous catheter into the uterine arteries for injection of thrombogenic material*** to control hemorrhage.
- Hysterectomy is a treatment of last resort, if the patient has completed her childbearing, a supracervical or total abdominal hysterectomy, is the definitive therapy for intractable PPH caused by uterine atony. If reproductive potential is important to the patient, ligation of the uterine arteries adjacent to the uterus will be effective.

- ***Genital tract (GT) trauma:*** **trauma during delivery is the second most common cause of PPH.** During vaginal delivery, laceration of the cervix and vagina may occur spontaneously, but they are more common, but they are more common following instrumental delivery using forceps or ventous extractor. The vascular bed in the GT are engorged during pregnancy, and bleeding can be profuse. Lacerations are particularly prone to occur over the perineal body, in the periurethral area, and over the ischial spines along posterolateral aspects of the vagina. The cervix may be lacerate at the two lateral angles while rapidly dilating in the first stage of labor. Uterine rupture may occasionally occur. At the time of delivery by low transverse cesarean, a lateral extension of the incision can damage the ascending branches of the uterine arteries; an extension inferiorly can damage the cervical branches of the uterine artery.

`* ***Management of GT trauma:***

When PPH is related to GT trauma, surgical intervention is necessary. When repairing GT lacerations, the first suture must be placed well above the apex of the laceration to incorporate any retracted bleeding arterioles into the ligature. Repair of vaginal lacerations require good light and good exposure, and the tissue should be approximated without dead space. A running locked suture technique provides the best hemostasis.

- ***Cervical lacerations*** need not to be sutured unless they are actively bleeding.
- ***Large, extended hematomas*** of the GT require surgical evacuation of clots and search for bleeding vessels that can be ligated. Stable hematomas can be observed and treated conservatively. ***A retroperitoneal hematoma*** generally begins in the pelvis. If the bleeding cannot be controlled using a vaginal approach, a laparotomy may be necessary.
- ***An intraoperative laceration of the ascending branch of the uterine artery*** during C/S can be easily controlled by the placement of a large suture ligature through the myometrium and broad ligament below the level of the laceration.
- ***A uterine rupture*** usually necessitates subtotal or total hysterectomy, although, small defect may be repaired.

Retained placental tissue:

- About one-half of patients with delayed PPH, placental fragments are present. The uterus is unable to maintain a contraction and involute normally around a retained placental tissue mass.
- When the ***placenta cannot be delivered in the usual manner, manual removal is necessar***y, and should be done urgently if the bleeding is profuse. Otherwise, it is reasonable to delay for 30 minutes to await spontaneous separation. GA may be required.
- If ***retained placental fragments*** are suspected, ***ultrasonic assessment of the uterus*** should be performed.
- If placental fragments are identified, ***manual exploration of the uterine cavity*** should be performed, with the patient under GA if necessary. With fingertips together, a gloved hand be slipped through the opened cervix and the hand inserted into the uterus. The endometrial surface should be palpated carefully to identify any retained products of conception, uterine wall lacerations, or partial uterine inversion.
- Following manual removal of placenta or its fragments, ***the uterus should be scraped with a large curette.***
- If ***no cause*** for the bleeding is found, ***coagulopathy*** must be considered.

Uterine inversion:

It is "turning" inside out" of the uterus in the third stage of labor. It is quite rare, occurring only about 1 in 200,000 pregnancies. Just after the second stage of labor, the uterus is somewhat atonic, the cervix open, and the placenta attached. Improper management of the third stage of labor can cause iatrogenic uterine inversion. If the inexperienced physician exerts fundal pressure while pulling on the umbilical cord before complete separation of placenta (particularly with a fundal implantation of the placenta), uterine inversion may occur. As the fundus of the uterus moves through the vagina, the inversion exerts traction on peritoneal structures, which can elicit a profound vasovagal response. The resulting vasodilatation increases the bleeding and the risk of hypovolemic shock. If the placenta is completely or partially separated, the uterine atony may cause profuse bleeding, which compounds the vasovagal shock.

***Management of uterine inversion:**

- The management of uterine inversion requires quick thinking. The patient is rapidly goes in shock, and immediate intravascular volume expansion with IV crystalloids is required.
- An anesthesiologist should be present.
- When the patient's condition is stable, the partially separated placenta should be completely removed and an attempt is made to replace the uterus by placing a cupped hand into the inverted fundus from below and elevating it in the long axis of the vagina.
- If this is unsuccessful, a further attempt should be made using IV nitroglycerine (100microgram) or GA to relax the uterine muscles.
- Once replaced, a dilute infusion of oxytocin should be started to cause the uterus to contract before removing the intrauterine hand.
- Rarely, the uterus cannot be replaced from below, and a surgical procedure may be required. At laparotomy, a vertical incision through the posterior portion of the cervix to inside the constriction ring and allow the fundus to be replaced into the peritoneal cavity. Suturing of the cervical incision completes this procedure.

Coagulation disorders:

Peripartum coagulation disorders are high-risk factors for PPH, but fortunately they are quite rare.

Patients with thrombotic thrombocytopenia have a rare syndrome of unknown etiology characterized by thrombocytopenic purpra, microangiopathic hemolytic anemia, transient and fluctuating neurologic signs renal dysfunction and a febrile course. In pregnancy, the disease is usually fatal.

Patients with idiopathic thrombocytopenic purpura have platelets with abnormal function or a short ended lifespan. This cause thrombocytopenia and a tendency to bleed. Circulating

antiplatelet antibodies of IG G type may occasionally cross the placenta and result in fetal and neonatal thrombocytopenia as well

Von Willebrand disease (VWD), is an inherited coagulopathy characterized by prolonged bleeding time due to factor VIII deficiency. During pregnancy, the patients are likely to have a decreased diathesis because pregnancy elevates factor VIII levels. In the postpartum period, they are susceptible to delayed bleeding as factor VIII levels fall.

An amniotic fluid embolus is rare but associated with 80% mortality rate. It is characterized by a fulminating consumption of coagulopathy, intense bronchospasm, and vasomotor collapse. It is triggered by an intravascular infusion of a significant quantity of amniotic fluid during rapid labor in the presence of ruptured membranes. During the process of placental abruption, a small amount of amniotic fluid may leak into the vascular system, and the thromboplastin in the amniotic fluid may trigger a consumption coagulopathy.

***management of amniotic fluid embolus:

The principle objectives of treatment for amniotic fluid embolism are to support the respiratory system, correct the shock, and replace the coagulation factors. This type of embolism requires immediate cardiopulmonary resuscitation, usually with mechanical ventilation, rapid volume expansion with an electrolyte solution, positive inotropic cardiac support, placement of bladder catheter to monitor urine output, correction of the red cell deficit by transfusion with packed RBC, and reversal of coagulopathy with the use of platelets, fibrinogen and other blood components.

*Management of coagulopathy:

When PPH is associated with coagulopathy, the specific defect should be corrected by the infusion of blood products

Laboratory evaluation of DIC:

- ***Platelet count*** (normal range 150-450*10^9 /L): 1 L of platelets will raise the platelets count by 5-10*10^9/L.
- ***Plasma fibrinogen*** (normal range 175-600mg/dl): fresh frozen plasma (FFP): 1 U=1gm of fibrinogen; 4U of FFP will rise the plasma fibrinogen by 5-10mg/dl.
- ***Cryoprecipitate:*** 1 bag = 0.25g of fibrinogen; 16 bags raise the plasma fibrinogen 5-10mg/dl.
- ***Fibrin split products:*** normal range<0.05 microgram/ml (D-dimer method).

Patients with thrombocytopenia require platelet concentrate infusion; those with VWD require factor VIII concentrate or cryoprecipitate.

- A packed red cell infusion is given to patient who has bleed sufficiently to compromise the delivery of oxygen to the tissues. Therefore, institution of blood transfusion is best judged by symptoms of oxygen deprivation rather than by some empirical Hb level.
- No important physiologic impairment has been noted at Hb levels as low as 6 to 8 g/dl (hematocrit of 18-24%).
- In general, a 1-U transfusion of packed red cells will increase the Hb level by 1gm/dl (and the hematocrit by 3-4%).
- Massive blood replacement (when total blood volume is replaced in 24-hour period) may be associated with thrombocytopenia, prolonged PT, and hypofibrinogenemia.
- Thrombocytopenia is the most common abnormality; so platelets transfusion following determination of a low platelet count is not an uncommon scenario.
- FFP may be transfused for prolonged PT or hypofibrinogenemia.

Maternal mortality and morbidity have been reduced when the protocol of packed RBC, FFP, and platelets, given in a ratio of 6:4:1, is implemented.

- Treatment should not be delayed while awaiting laboratory results or blood product cross matching.

Blood products used to correct coagulopathy defect:

Blood product	***Volume (ml) in 1 U***	***Effect of transfusion***
Platelet concentrate	*30-40*	*Increase platelet count by about 5000-10.000*
cryoprecipitate	*15-25*	*Supplies fibrinogen, VWF, factor VIII, and fibronectin*
Fresh frozen plasma (FFP)	*200*	*Supplies all factors except platelets (1gm of fibrinogen)*
Packed red blood cells	*200*	*Raises hematocrit 3-4%*

Management of patients with risk for PPH:

Because the major cause of PPH is uterine atony, the initial focus should be in prevention of uterine atony by considering the following steps:

1) All women in early labor who have risk factors for PPH should be identified and their Hb checked. For medium-risk women, their blood should be typed and Rh status. For high-risk group, 2 units of blood should be typed and cross matched.
2) As soon as the fetus has been delivered, an infusion of oxytocin (Pitocin) 10 to 40 U/LIV should be stated and maintained during the first 6 hours postpartum.
3) The vagina and perineum should be inspected to rule out any lacerations that could cause excessive bleeding.

4) The placenta should be carefully assessed at delivery to make certain there is no missing cotyledons (lobules of placenta).
5) The uterus should be evaluated by abdominal palpation during the first 1 to 2 hours before transfer to the postpartum unit. The nurse on the postpartum should frequently assess the status of uterine contractility, instructing the patient on how to assess uterine firmness and reporting any excessive bleeding. For high-risk patients, continuation of the oxytocin IV infusion during the early postpartum hours should be considered.

Obstetric shock without external bleeding:

Hypotension without significant external bleeding can be happened in obstetric patient. This condition is called obstetric shock.

- The causes of obstetric shock include concealed hemorrhage within the uterus, uterine inversion, and amniotic fluid embolism.
- An improperly sutured episiotomy can lead to concealed PPH. If the first suture at the vaginal apex of the episiotomy incision dose not incorporate the cut and retracted arterioles, these can continue to bleed, creating hematoma that can be dissect cephalad into the retroperitoneal space. This may cause shock without external evidence of blood loss.
- A soft tissue hematoma, usually of the vulva, may occur following delivery in the absence of any laceration. Uterine rupture can also occur secondary to blunt trauma at the time of automobile accident.

Management of established PPH and obstetric shock:

During the diagnostic workup pf an established hemorrhage:

- The patient's vital signs must be monitored closely. However, young healthy women may tolerate and mask hypovolemia well. The sensitivity and specificity of vital signs are not absolute.
- The estimated blood loss is commonly underestimated, and it should be replaced by quantitated blood loss, where sponges and pads are weighed and measured.
- Multiple units of packed RBC must be typed and cross matched, and IV crystalloids (such as N/S or lactate Ringer solution) infused to restore intravascular volume.
- Resuscitation with N/S usually requires a volume of three timed the estimated blood loss to replace the intravascular volume.
- During a massive hemorrhage, morbidity and/or mortality are reduced with an emphasis on early blood product replacement rather than crystalloid-based resuscitation.

Secondary PPH:

- ***Secondary PPH is defined as fresh bleeding from the genital tract between 24hrs and 6 weeks after delivery***.
- The most common time for secondary PPH is between ***days 7 and 14***.
- ***The cause*** is usually either ***endometritis or retained placental tissue***, and it is often very difficult to distinguish between them.
- Classically, women with ***endometritis*** have ***constant low abdominal pain and a tender uterus with a closed internal os***.
- In contrast, women with ***retained products of conception have crampy lower abdominal pain, a uterus larger than appropriate, with an open internal os, and a history of prolonged labor or sometimes passage of bits of placental tissue or tissues***.
- Both endometritis and retained products of conception may have signs and symptoms of infection as low- grade fever, pungent lochia, and uterine tenderness.
- Those bleeding heavily will require a circulatory support with fluid and blood along with strong oxytocic s (e.g. ergometrine) and uterine evacuation.
- Antibiotics should be given if placental tissue is found, even without evidence of overt infection.
- If blood loss is not excessive, the use of pelvic U/S to exclude retained products is sometimes used, but is only helpful if the uterus is seen to be empty. Debris, clots and fluids are commonly found even the normal postpartum uterus and their presence does not mean that there is retained products.
- ***In the absence of clear diagnosis,*** expectant management with an empirical antibiotics is often used.
- ***Other causes*** of Secondary PPH are including:

- ***Infection (infected C/S wound, infected genital tract lacerations, infected placental site, or separation of infected retained parts)***
- ***Hormonal contraception***
- ***bleeding disorders (e.g. VW disease)***
- ***Fibroid polyp: necrosis of its tip***
- ***Subinvolution of the uterus.***
- ***Puerperal inversion of the uterus.***
- ***Choriocarcinoma.***
- ***Local gynecological lesions: as cervical ectropian or malignancy.***