

The background features a light blue gradient with scattered small stars in shades of blue and green. On the left, there are illustrations of three pills (one white, two blue) and a syringe. On the right, there is a green thermometer and a blue drip chamber with a tube. The title 'Obstetric shock' is centered in a large, bold, dark blue font.

Obstetric shock

Done by:

Hamza Tabanja
Omar abu Al saod
Hiba Makawi
Emran Younis
Obada Alayed

Definition of shock

- Shock-- is an acute circulatory failure leading to inadequate perfusion and delivery of oxygen to vital organs.

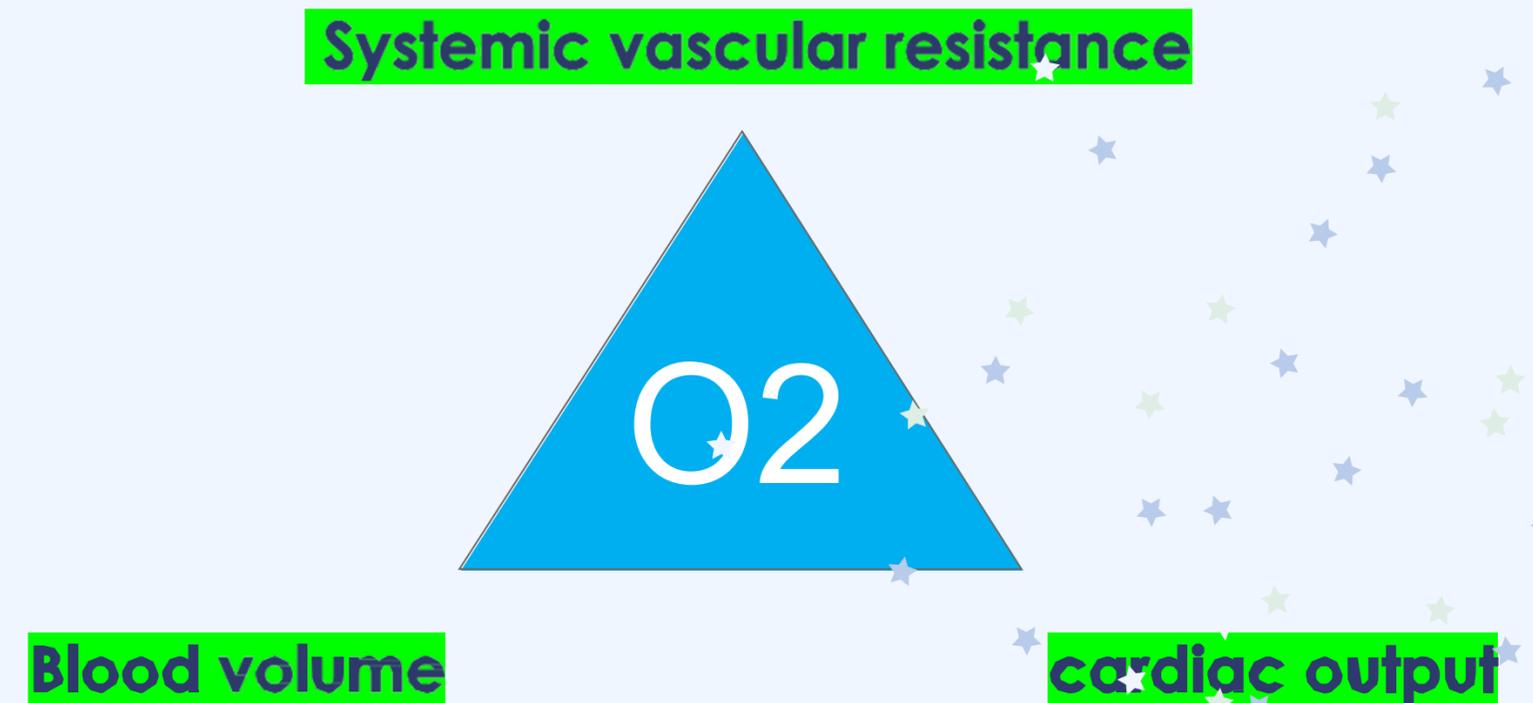
- **Types and causes**

- 1) Hypovolemic Shock

- 2) Cardiogenic Shock

- 3) Obstructive Shock

- 4) Distributive Shock
(septic & neurogenic)



□ **In Obstetric cases shock is most commonly due to either hemorrhage or sepsis**

1) Hypovolemic Shock

- **Definition:** medical or surgical condition in which ----->
rapid fluid loss results in **multiple organ failure** due to

**inadequate circulating volume_ and subsequent
inadequate perfusion**

Hypovolemic Shock

Can be classified according to the causes into

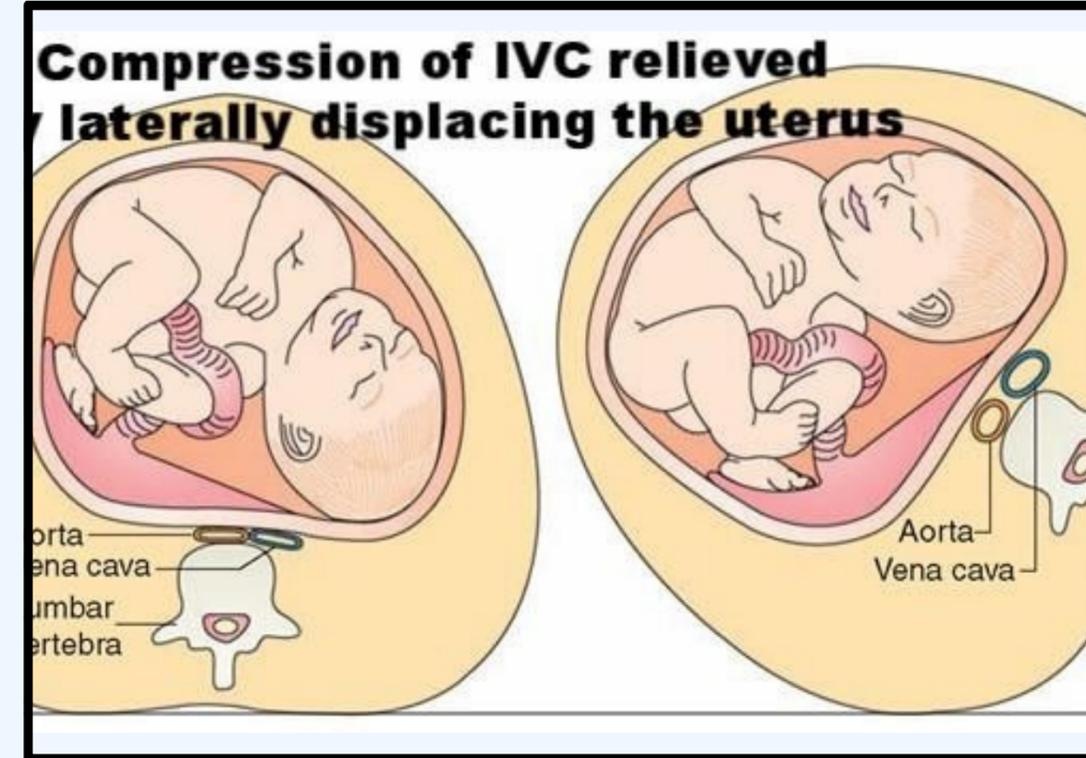
Hemorrhagic

Non-Hemorrhagic

Causes in pregnancy

1. **Blood loss (obstetric hemorrhage)**
In Jordan , **HEMORRHAGE** is the leading cause of **Maternal mortality**.
 - a. **Bleeding in early pregnancy**
 - b. **Antepartum hemorrhage**
 - c. **Postpartum hemorrhage**
 - d. **Trauma**
2. **Fluid loss : hyperemesis gravidarum**
3. **Supine hypotension syndrome .**
4. **Splanchnic shock** : sudden drop in intrauterine pressure :
 - Precipitate delivery
 - ROM in polyhydramnios baby

The most common form of hypovolemic shock in obstetrics is **hemorrhagic shock**



*Phases of Hemorrhagic Shock

o Phase of compensation (pre_shock state):

- **Sympathetic stimulation:** It is the initial response to blood loss leading to peripheral Vasoconstriction to maintain blood supply to the vital organs (redistribution to heart & brain)
- **Clinical picture: Pallor, tachycardia, tachypnoea**

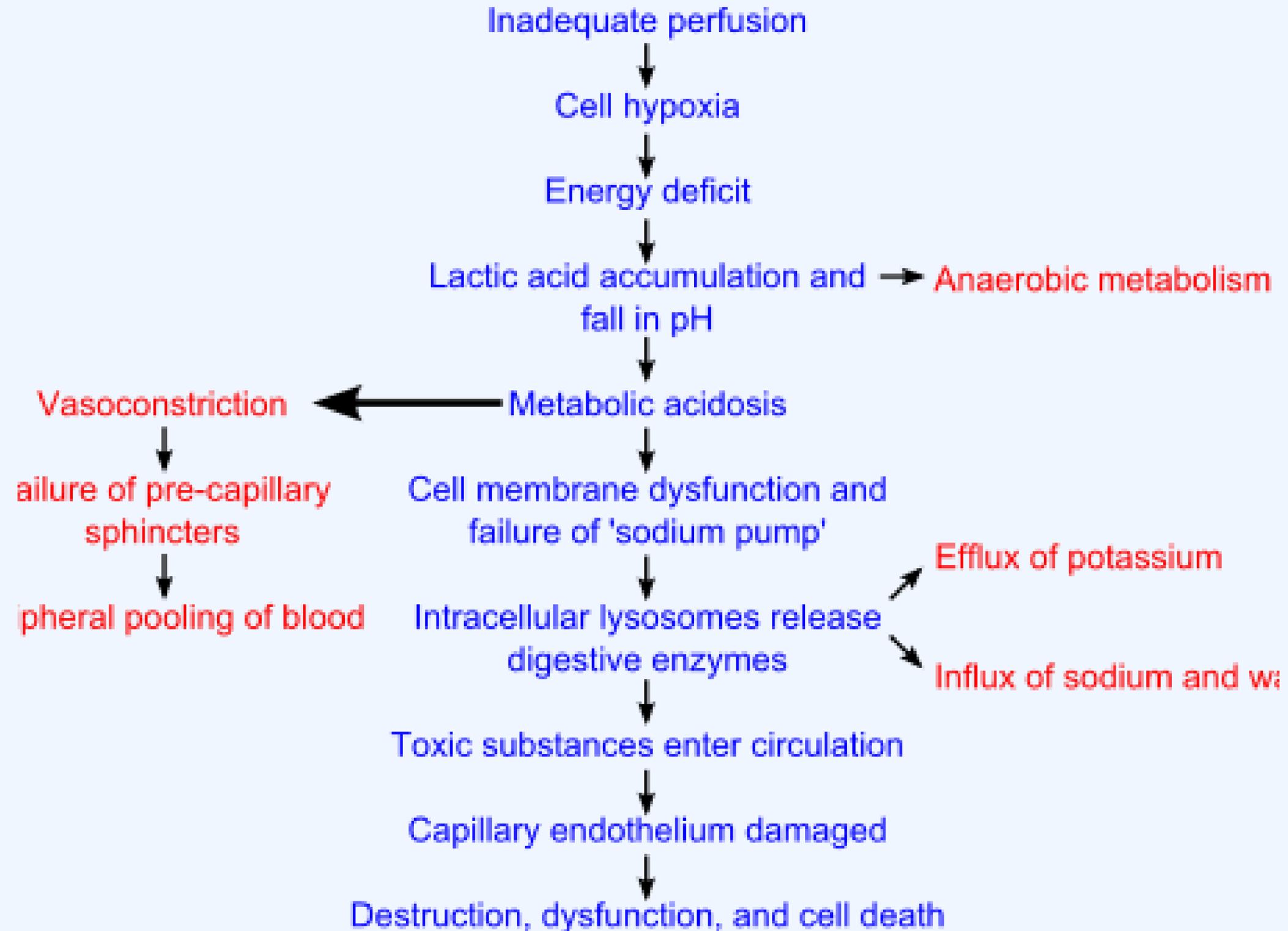
o Phase of decompensation (Shock state): (reversible dysfunction)

- Blood loss exceeds 1000 ml in normal patients or less if other adverse factors are operating.
- **Clinical picture: the classic clinical picture of shock.**
- Adequate treatment at this phase improves the condition rapidly without residual adverse effects.
- **The normal pregnant woman can withstand blood loss of 500 ml and even up to 1000 ml during delivery without obvious danger due to physiological cardiovascular and hematological adaptations during pregnancy.**

**o Phase of cellular damage and danger of death:
(irreversible dysfunction)**

- Metabolic acidosis: due to anaerobic metabolism initiated after lack of oxygen.**
- Arteriolar dilatation: caused by accumulation of metabolites leading to pooling and stagnation of blood in the capillaries and leakage of fluid into the tissues.**
- Disseminated intravascular coagulation: caused by the release of thromboplastin from the damaged tissues.**
- Cardiac failure: due to diminished coronary blood flow.**
- In this phase death is imminent, transfusion alone is inadequate and if recovery from the acute phase occurs, residual tissue damage as renal and/ or pituitary necrosis will occur.**

Mechanism of shock



Classic Clinical Picture of Shock

- **Low blood pressure (not an absolute requirement)**
 - SBP <90 mmHg, not seen in preshock state**
- **Rapid weak (thready) pulse (not in all type)**
- **Cool Skin**
 - Vasoconstriction mechanisms to redirect blood from periphery to vital organs.**
 - Exception is warm skin in early distribution shock.**
- **Oliguria or anuria (decrease kidney perfusion)**
- **Metabolic Acidosis (Lactic acid due to anaerobic metabolism in body tissues)**
- **Altered Mental Status . (decrease brain perfusion)**

Approach to any hemorrhage:

History:

- How much bleeding?
- Triggering factors (e.g. postcoital bleed).
- Associated with pain or contractions?
- Is the baby moving?
- Risk factors and symptoms according to the cause.

Examination:

- **Vitals:** Pulse, blood pressure, o2 sat, Temperature.
- Is the uterus soft or tender and firm?
- Fetal CTG.
- Speculum vaginal examination, **Visualizing the cervix** (having established that placenta is **not a Previa**, by using ultrasound).

- **Investigations:**

- **Blood sample for:**

CBC, RH and cross-matching , electrolytes, LFT , KFT, blood sugar Coagulation profile (PT . PTT . INR . Fibrinogen . FDP)

- **Ultrasound :**

Mainly placental site and Retroplacental hematoma(concealed abruption placenta)

fetal size, Presentation , amniotic fluid

Management:

- Any mother presented with bleeding and change in vitals: **AB**
- Adequate ventilation providing oxygen
- Stop bleeding
- two large bore cannulas
- Labs (CBC, PT, PTT, cross-match)
- Start IV FLUID (NS + RL) // or colloids in hemodynamic collapse
- At least 4 Blood Units and FFP
- Folly's catheter
- Monitoring (vitals, urine output, CVP, mental status, temperature , HB, PH)

Treatment:

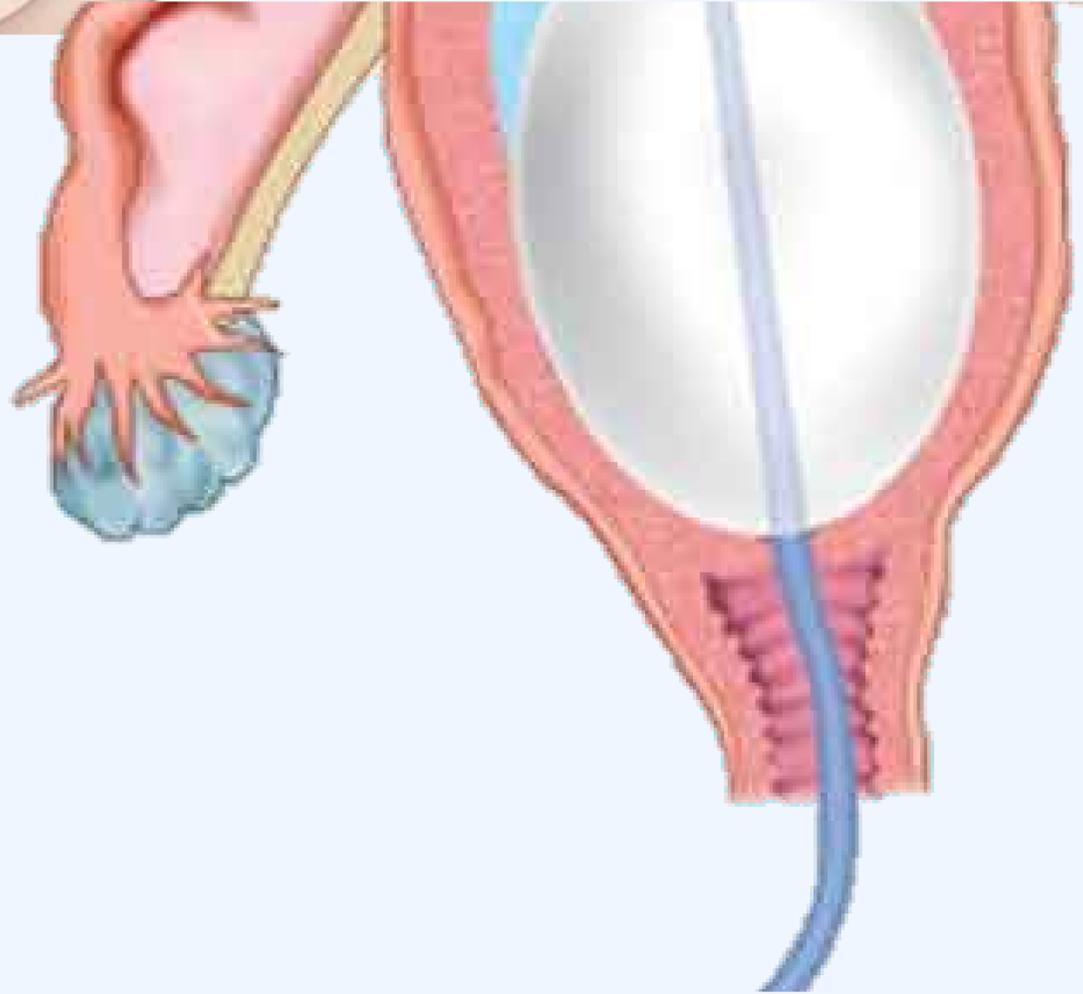
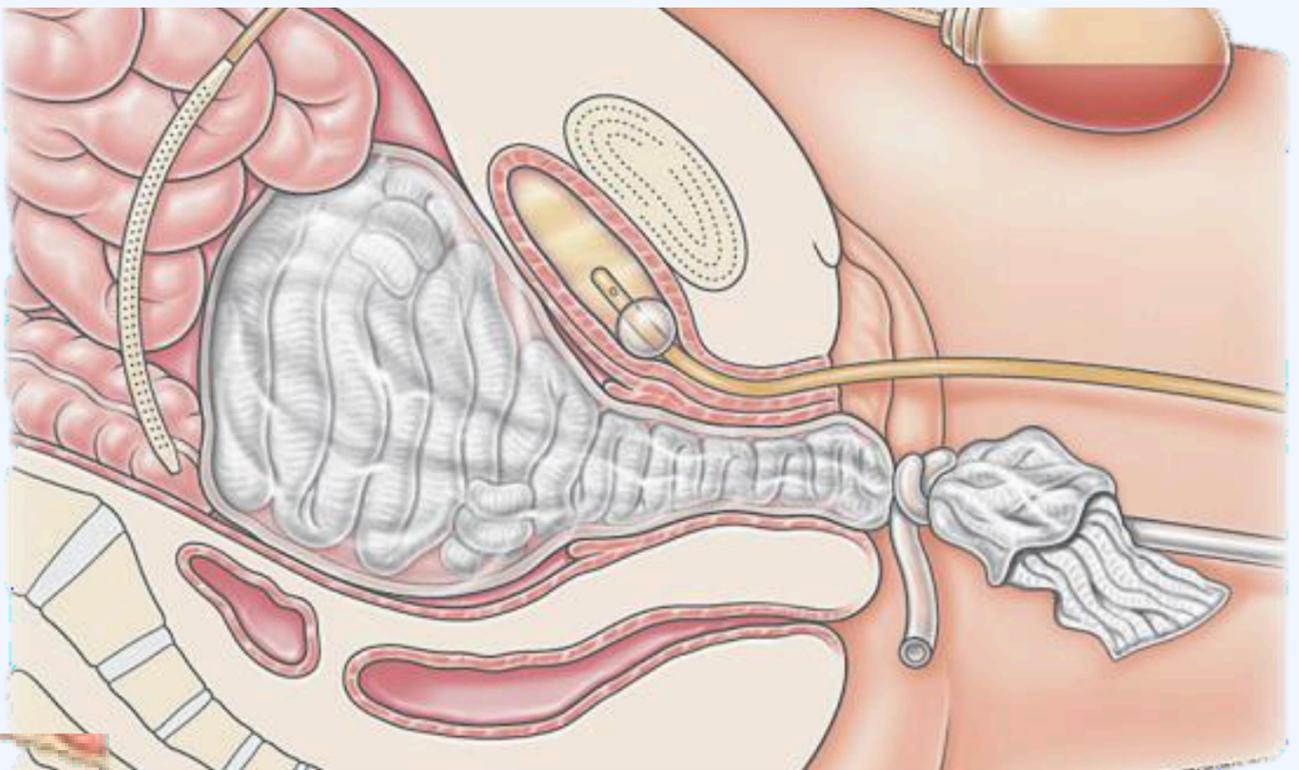
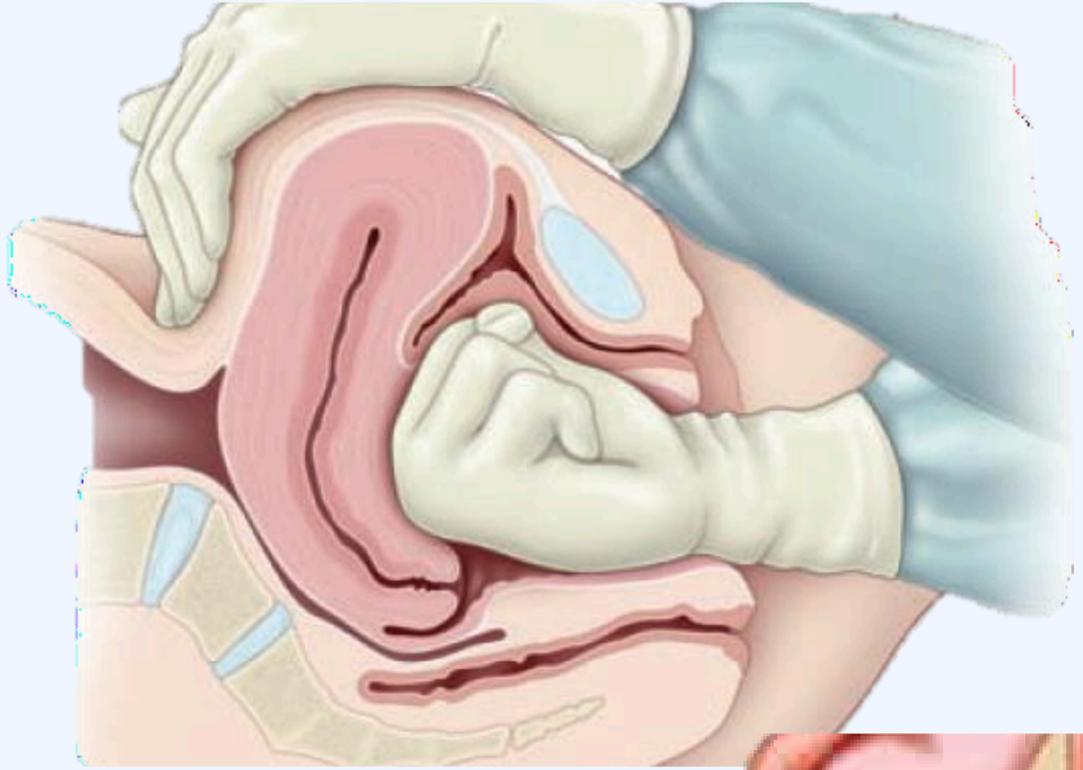
- **Stabilization if unstable with fluid and blood transfusion if needed**
- **TREAT the**
- **Laceration** □ repair
- **Placental causes** □ CS, expectant until safe to deliver
- **Retained product** □ manual removal under GA
- **Atony** □ massage
- **Early pregnancy** □ D/C...
- **Vasopressors/ inotropes** : to increase blood pressure to maintain renal perfusion-- **Dopamine**: drug of choice
- **Analgesia**

What If the uterus still soft (Atony

)

- **Massage** the uterus
- **Oxytocin** infusion or IM **prostaglandins**
- **Shift to theatre**, with : **aortic compression** , **bimanual compression**
- **balloon or uterine packing** **after exclusion of retained tissue and trauma**
- **IV Tranexamic acid** (antifibrinolytic agent)
- Apply **compression sutures** on the uterus (**B-Lynch** or modified technique)
- **Interventional radiology** and, if appropriate, **uterine artery embolization**

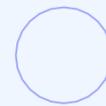
Finally , If nothing work -- **Subtotal or total abdominal hysterectomy**



Complications of Hypovolemic shock

1. **Acute renal failure. (pre-renal)**
2. **Pituitary necrosis (Sheehan's syndrome) in PPH**
3. **Disseminated intravascular coagulation (DIC)**
4. **Death**

Cardiogenic Shock

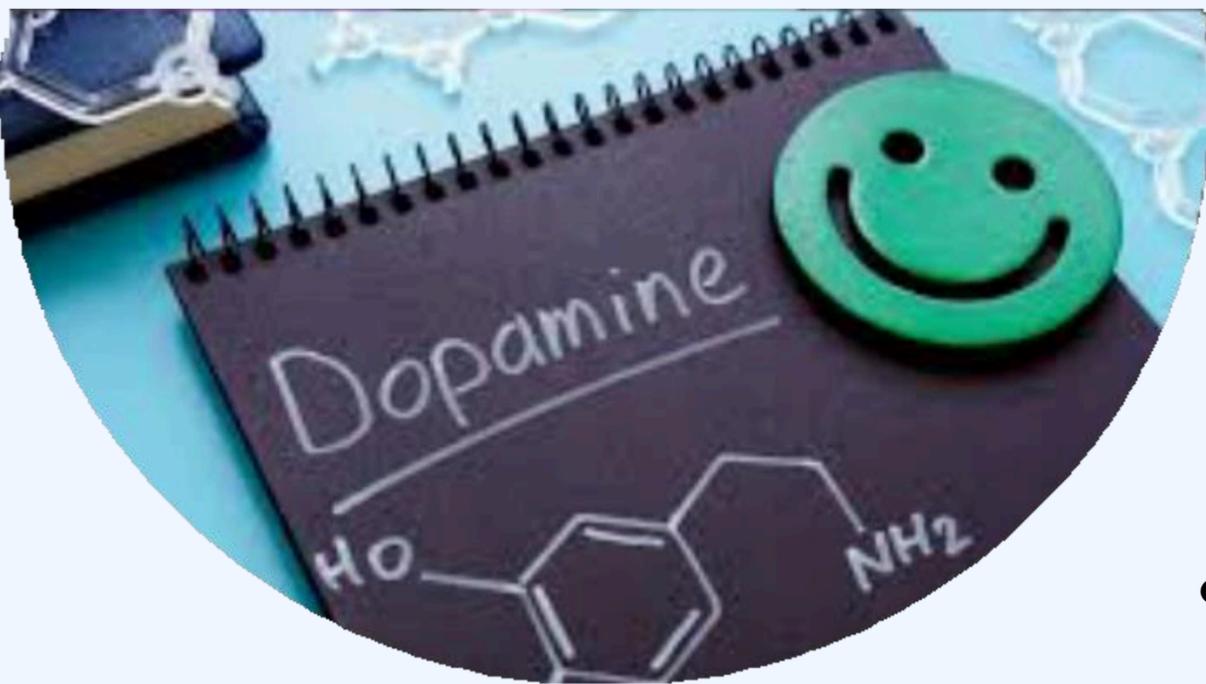


- **Life-threatening condition resulting from inadequate cardiac output.**
What precipitates this condition?
- Pregnancy puts a progressive strain on the heart as progresses.
- Preexisting heart disease increases the risk
- **Incidence:** Cardiac-related mortality is the second most common cause of death in pregnancy.

Signs and symptoms:

- 1. Chest pain**
- 2. Hypotension**
- 3. Palpitation, tachycardia**
- 4. Pale cold mottled skin (low capillary refill)**
- 5. Raised JVP**
- 6. Bilateral basal pulmonary crackles**
- 7. SOB**
- 8 Profuse sweating**
- 8. Nausea and vomiting.**
- 9. Confusion**
- 10. Third and fourth heart sounds**
- 11. Heave thrill, murmurs**
- 12. Syncope**
- 13. Oliguria**





How to treat?

- Reestablish the circulation
- O2 supply
- Cardiac drugs as needed (**dopamine, dobutamine, epinephrine, NE**)



Sepsis

The background features a central light green circular glow surrounded by concentric, fainter circles in shades of blue and purple. The overall color palette is soft and pastel. There are several decorative elements: a small pink circle with a dashed outline in the lower-left, a blue circle in the lower-right, a blue circle in the upper-right, and three blue wavy lines on the right side. The word 'Sepsis' is centered in a large, bold, black sans-serif font.

Remains an important cause of maternal death with deaths due to group A streptococcal infection contributing significantly.

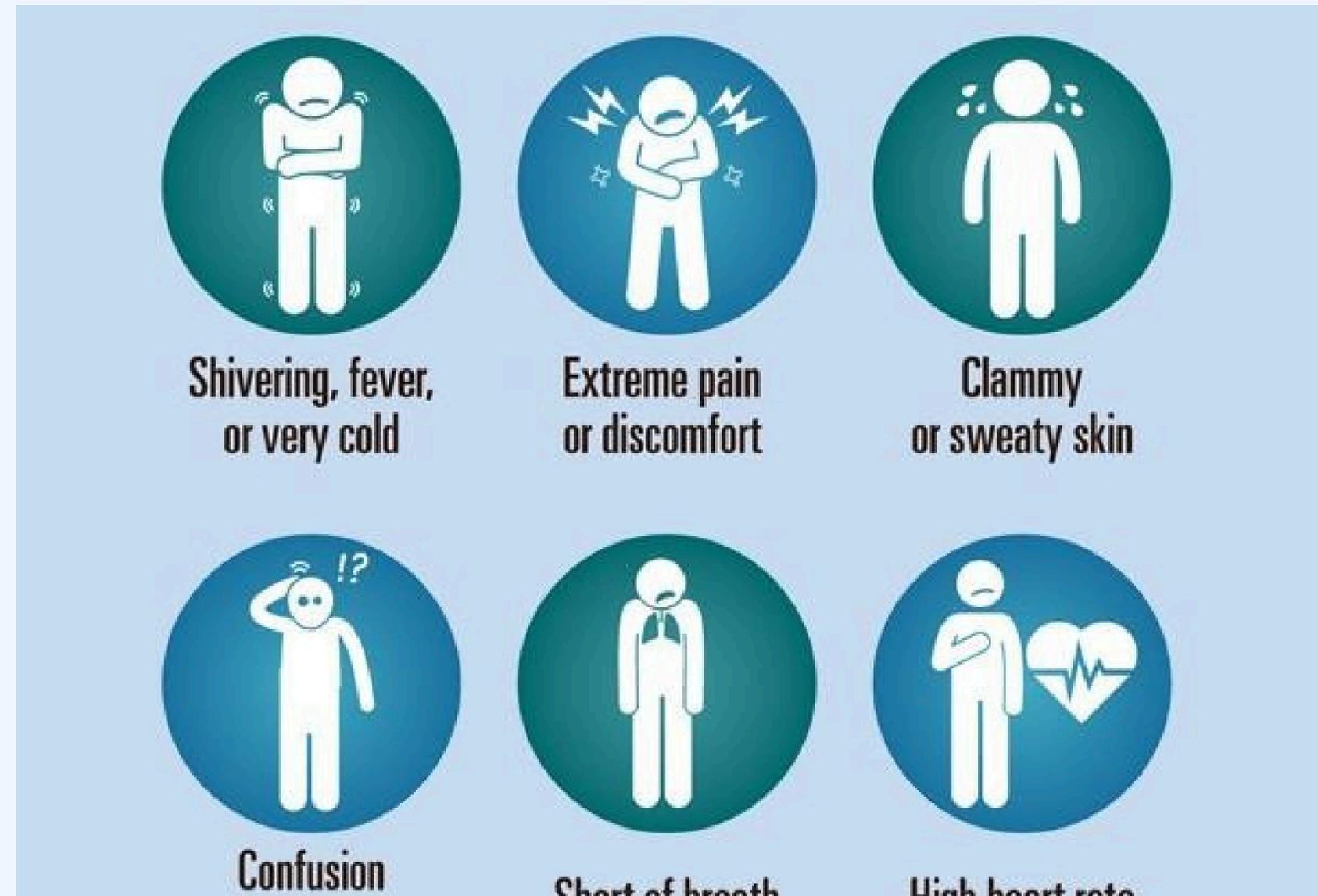
- **Risk factors:**

- ruptured membranes, amniocentesis and other invasive procedures, cervical cerclage
- immunocompromised patients, obesity, diabetes, anemia.
- urinary tract infections, vaginal discharge, previous pelvic infection, group B streptococcal infection, group A streptococcal infection in close contacts.
- minority ethnic group origin.



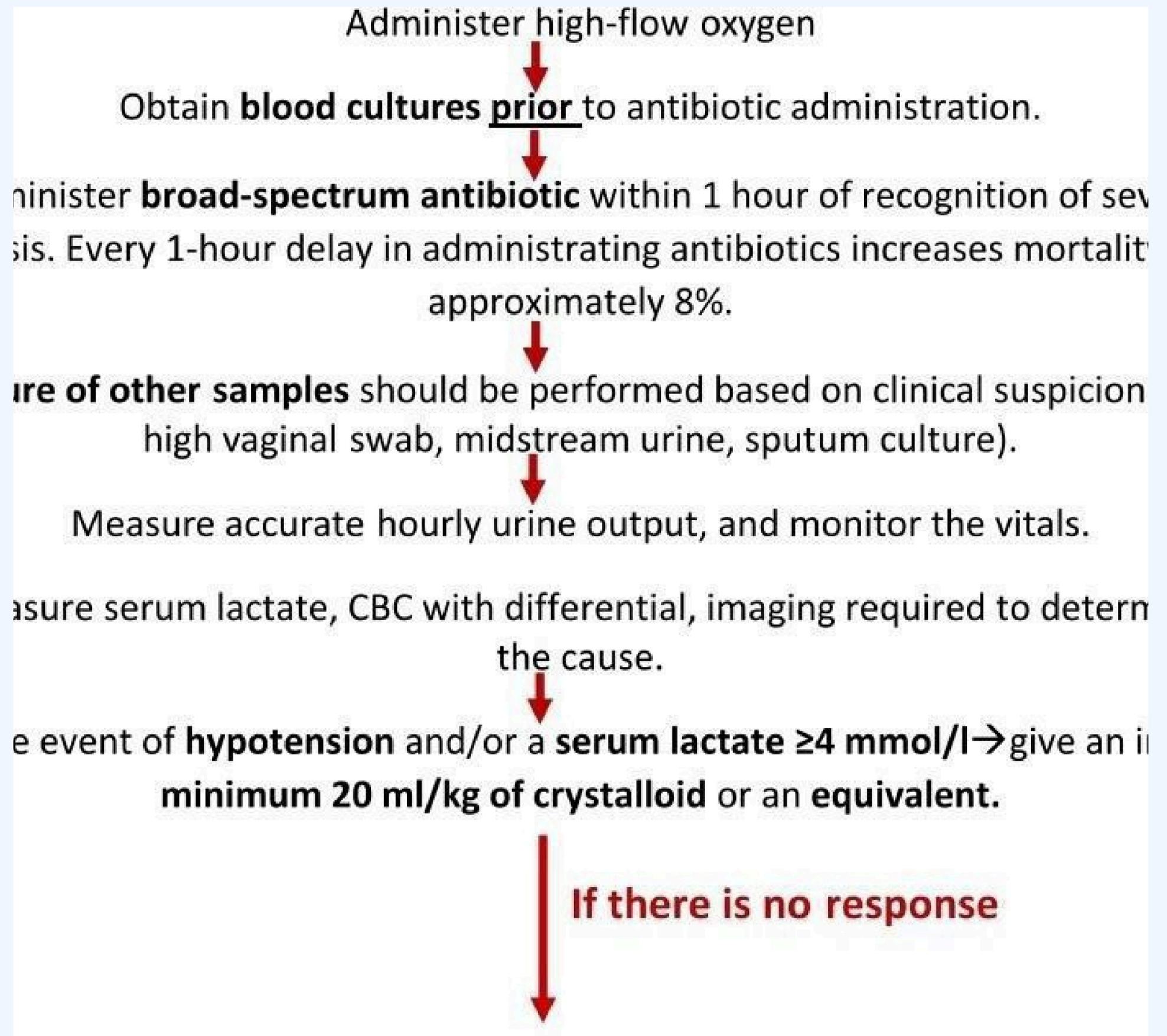
Warning signs:

- pyrexia
- tachycardia
- increased respiratory rate,
- hypotension,
- oliguria.
- Patients may rigor, have a rash
-reduced levels of
consciousness and not respond
to initial treatment



MANAGEMENT

The Surviving Sepsis Campaign Resuscitation bundles have key points in the timely management of sepsis:



vasopressors for hypotension that is not responding to
citation to maintain mean arterial pressure (MAP) ≥ 65 mmHg

ent of **persistent hypotension** despite fluid resuscitation
lactate ≥ 4 mmol/l

to achieve a central venous pressure (CVP) of ≥ 8 mmHg
or a central venous oxygen saturation $\geq 70\%$

Antibiotic use

The selection of antibiotics should be guided by **risk factors** and **potential sources** of sepsis.

A combination of either **piperacillin/tazobactam** or a **carbapenem plus clindamycin** provides very broad coverage for the treatment of severe sepsis.

Other:

- **Co-amoxiclav:** provides gram-positive and anaerobe cover.
- **Metronidazole:** provides anaerobic cover.
- **Clindamycin:** covers streptococci and staphylococci including MRSA.
- **Gentamicin:** provides gram-negative cover against coliforms and Pseudomonas.



Anaphylactic shock:

- Anaphylaxis is a rare event during pregnancy, but its onset may trigger maternal hypotension leading to intrapartum asphyxia and, eventually, the risk of severe fetal brain damage. Furthermore, the risk of cesarean delivery in anaphylactic patients in pregnancy is high (74%).
- **Symptoms of anaphylaxis:**
 - respiratory (e.g., wheeze, dyspnea)
 - gastrointestinal (e.g., vomiting, abdominal pain)
 - skin and mucosal (e.g., urticaria, itchy rash, swelling of lips)
 - cardiovascular and central nervous systems (e.g., reduced blood pressure, feeling faint, headache).
 - **Potential pregnancy-related symptoms** and signs of anaphylaxis are lower
 - back pain, fetal distress, uterine cramps, preterm labor, and vulvar or vaginal itching

Initial treatment includes

1. improving maternal airway, breathing, and circulation, removing causative agents, and injecting **epinephrine in the mid**-outer thigh.
2. It is important to re-evaluate the allergic symptoms of the patient, to prevent permanent damage to the fetus.
If allergy symptoms recur or do not improve, epinephrine injection can be needed repeatedly every 5–15 min.
3. Patients may require cesarean delivery to avoid fetal hypoxemia and prevent severe fetus damage.
4. The epinephrine auto-injector should be prepared for pregnant women who have potential risk of anaphylaxis.

ETRIC

OCK



OBSTETRIC

SHOCK

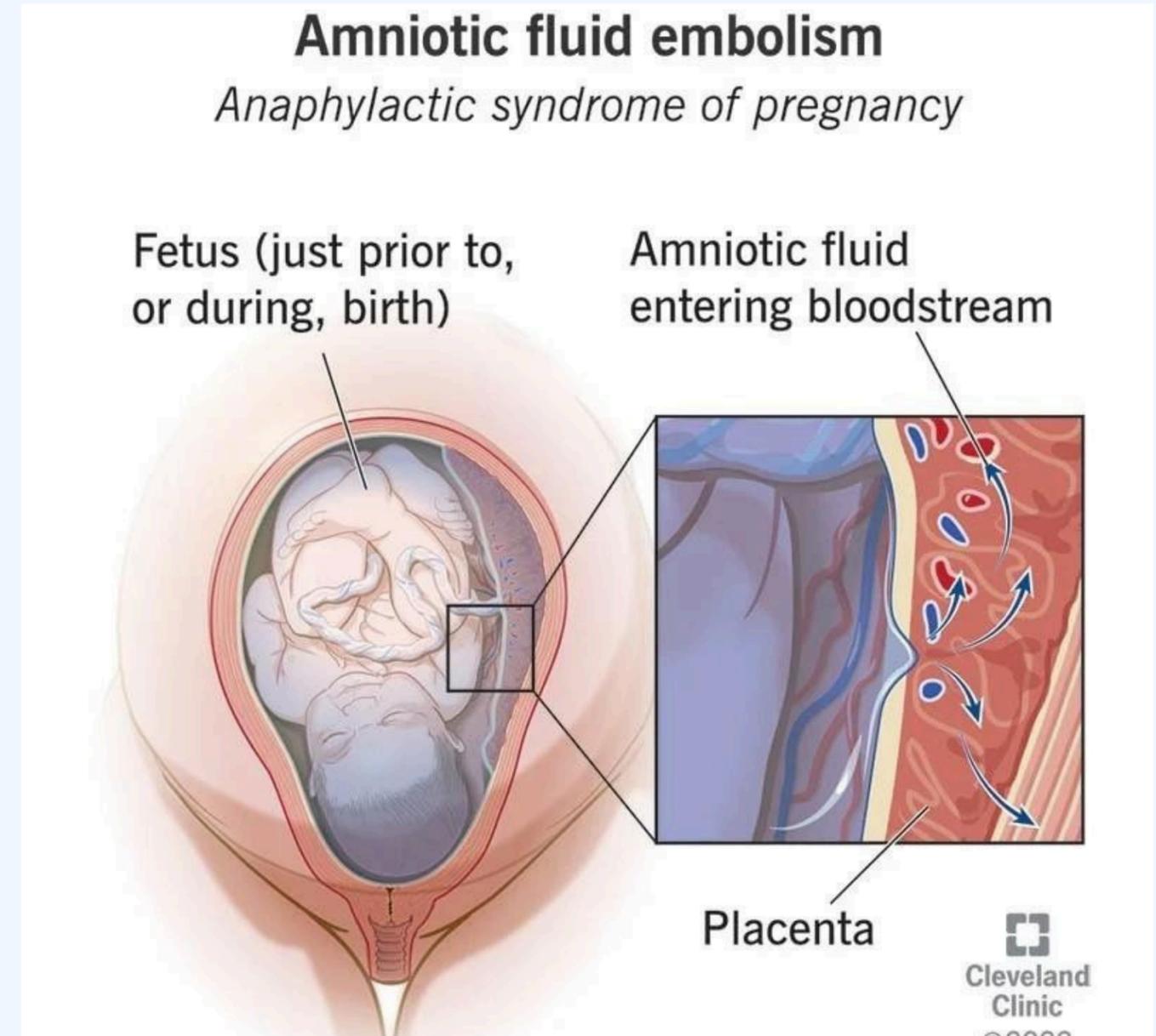
OBSTO

SHO

- Acute circulatory failure during pregnancy, childbirth, or the postpartum It requires immediate intervention to prevent maternal mortality.
- Some causes of obstetric shock
 1. concealed hemorrhage within the uterus
 2. uterine inversion
 3. amniotic fluid embolism.
 4. An improperly sutured episiotomy can lead to a concealed PPH

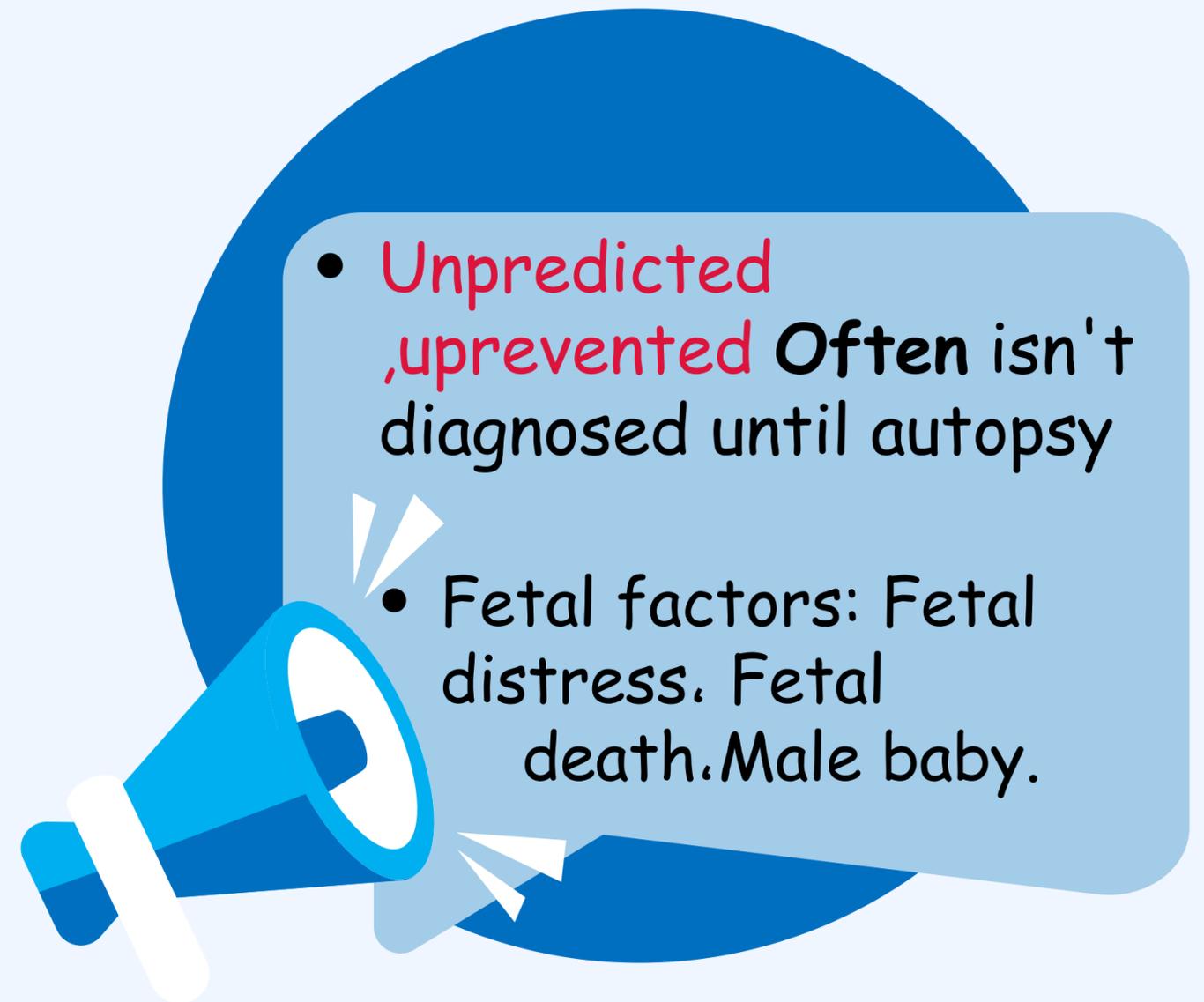
Amniotic fluid embolism

- Amniotic fluid embolism (AFE) is a life-threatening obstetric emergency
- It happens due to the entering of amniotic fluid, fetal cells, hair, and amniotic debris into the maternal circulation causing **cardiopulmonary** collapse.
- Previous studies revealed mortality rates as high as 61-86%, but recent estimates suggest a case fatality of 13-26%. This decrease in risk for maternal mortality from AFE may be the result of **early diagnosis and better resuscitative care as well as changes in case inclusion criteria**



R i s k f a c t o r s :

- Older maternal age.
- Multiparty.
- Intense contractions during labor.
- Abdominal trauma
- Cesarean section.
- Induction of labor.
- Placenta Previa.
- Eclampsia.
- Multiple pregnancy
- invasive procedures:amniocentesis



- **Unpredicted**,**uprevented** Often isn't diagnosed until autopsy

- Fetal factors: Fetal distress. Fetal death. Male baby.

Pathophysiology

- the exact pathophysiology is **still unknown**.
- **The process is similar to anaphylaxis than to embolism, so also termed as anaphylactic syndrome of pregnancy** because fetal tissue or amniotic fluid components are not universally found in women who present with signs and symptoms attributable to AFE.
- In some women, AFE may lead to a mild degree of organ dysfunction while in others it may lead to coagulopathy, cardiovascular collapse, and death.

- This theory is supported by the fact that amniotic fluid contains **vasoactive and pro-coagulant products** including platelet-activating factor, cytokines, bradykinin, thromboxane, leukotrienes, and arachidonic acid.
 - Concentrations of **tissue factor and tissue factor pathway inhibitor**, which trigger intravascular coagulation, are higher in amniotic fluid than in maternal serum
 - The direct pro-coagulant property of amniotic fluid may explain the prevalence of disseminated intravascular clotting (DIC) in AFE.
- It is speculated that maternal plasma **endothelin** concentrations are increased by the **entry of amniotic** fluid into the systemic vasculature. Endothelin acts as a Broncho constrictor as well as a pulmonary and coronary vasoconstrictor, which may contribute to respiratory and **cardiovascular collapse**

Hypotension + (CVS collapse) :

Bradykinin + cytokines -> systemic vasodilation ↓ BP.

Thromboxane + leukotrienes -> pulmonary vasospasm -> right heart strain

Hypoxia +(RS collapse):

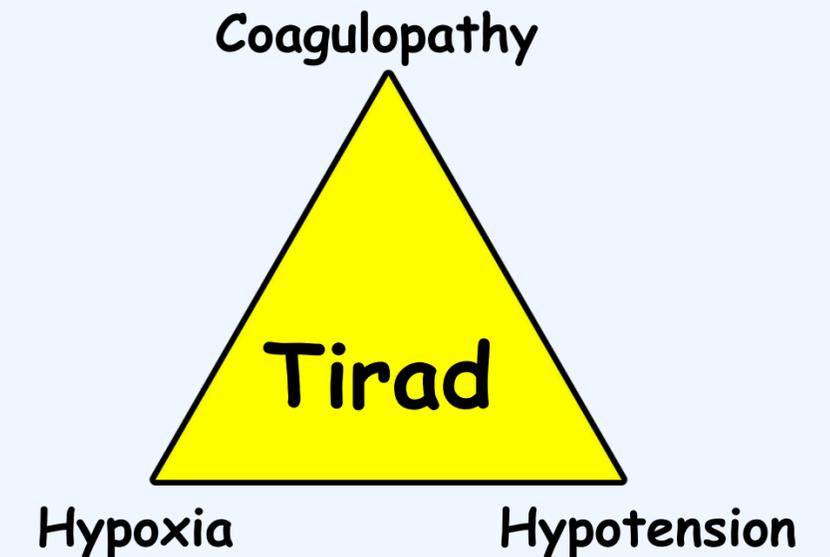
Leukotrienes -> bronchospasm.

Cytokines -> capillary leak -> pulmonary edema.

Coagulopathy (DIC):

PAF + tissue factor -> platelet activation/thrombin generation

-> thrombosis -> consumptive coagulopathy



The cause of AFE-associated reactions is explained by two hypothesis: The effect of amniotic fluid itself or a host idiosyncrasy ("hypersensitivity" reaction)

There are two clinical forms of AFE

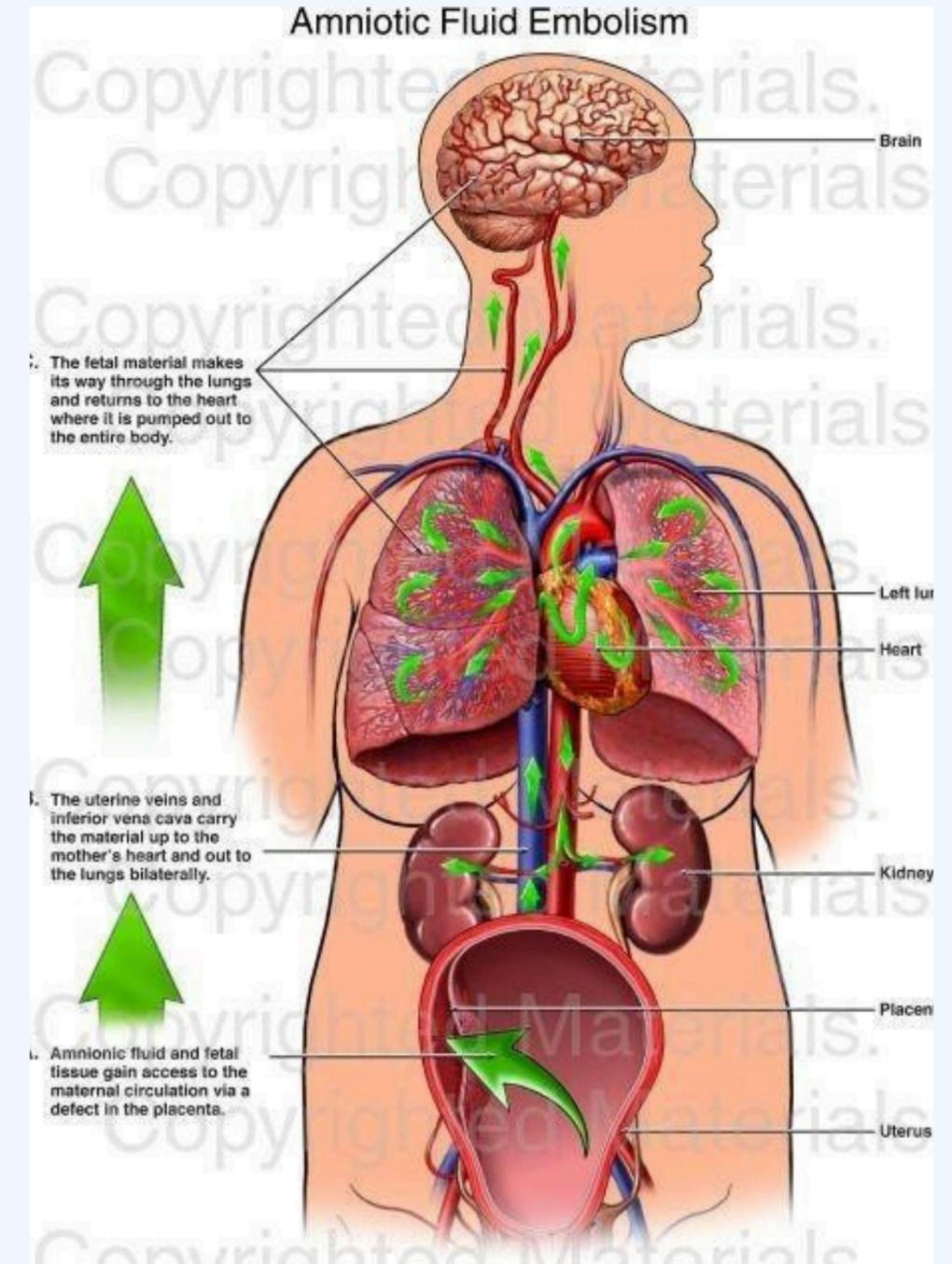
- Typical, (classic) with three phases:

- Phase 1- respiratory and circulatory disorders
- Phase 2- coagulation disturbances of maternal hemostasis
- Phase 3- acute renal failure and acute respiratory distress syndrome (ARDS), leading to cardiopulmonary collapse.

- Atypical:

In contrast to typical embolism

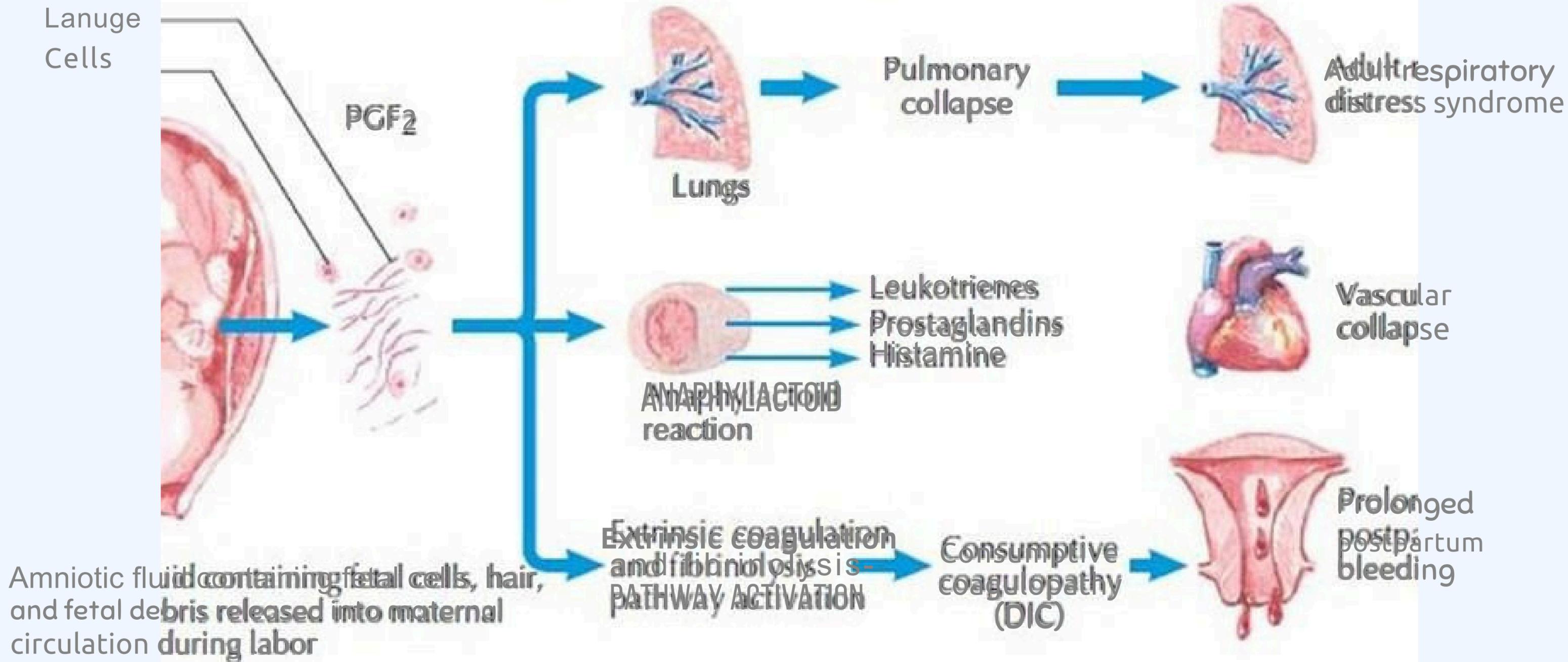
- cardiopulmonary collapse does not occur in atypical embolism but the first symptom is life threatening **hemorrhage** due to DIC.
- atypical **embolism** was observed during cesarean section or immediately after it, in cases of profound rupture of uterine cervix, as well as in the course of placenta abruption and in association with induced mid-trimester abortion.



Clinical presentation

- sudden chills, shivering, sweating
- **Cyanosis:** Ventilation perfusion mismatching , may explain sudden hypoxia and respiratory arrest.
- Altered mental status.
- **Hypotension: the most common (100%).**
- Acute dyspnea (manifested as cough)
- **Acute pulmonary hypertension and vasospasm** results in right ventricular failure, hypoxia, and cardiac arrest
- **Coagulopathy or severe hemorrhage:** Coagulation disorders are a prominent feature of the amniotic fluid syndrome. DIC is present in more than 83% of patients with AFE. The onset can occur as quickly as 10-30 min from the onset of symptoms or may be delayed by as long as 4 h.
- Fetal bradycardia: In response to the hypoxic insult
- **Encephalopathy** associated with AFE is thought to be secondary to hypoxia and includes a spectrum of symptoms ranging from **altered mental state to seizures**. Tonic-clonic seizures are seen in 10-50% of patients
- Uterine atony :hypofibrinogenemia

Clinical Features of Amniotic Fluid Embolism



DIC, disseminated intravascular coagulation; PT, prothrombin time; PTT, partial thromboplastin time.

- **Four criteria must be present to make the diagnosis of AFE:**
- Acute **hypotension** or cardiac arrest.
- Acute **hypoxia**.
- **Coagulopathy** or severe hemorrhage in the absence of other explanations.
- All of these occur during **labor**, cesarean delivery, dilation, and evacuation, or within 30 min postpartum with no other explanation of findings.

Rule of Thumb



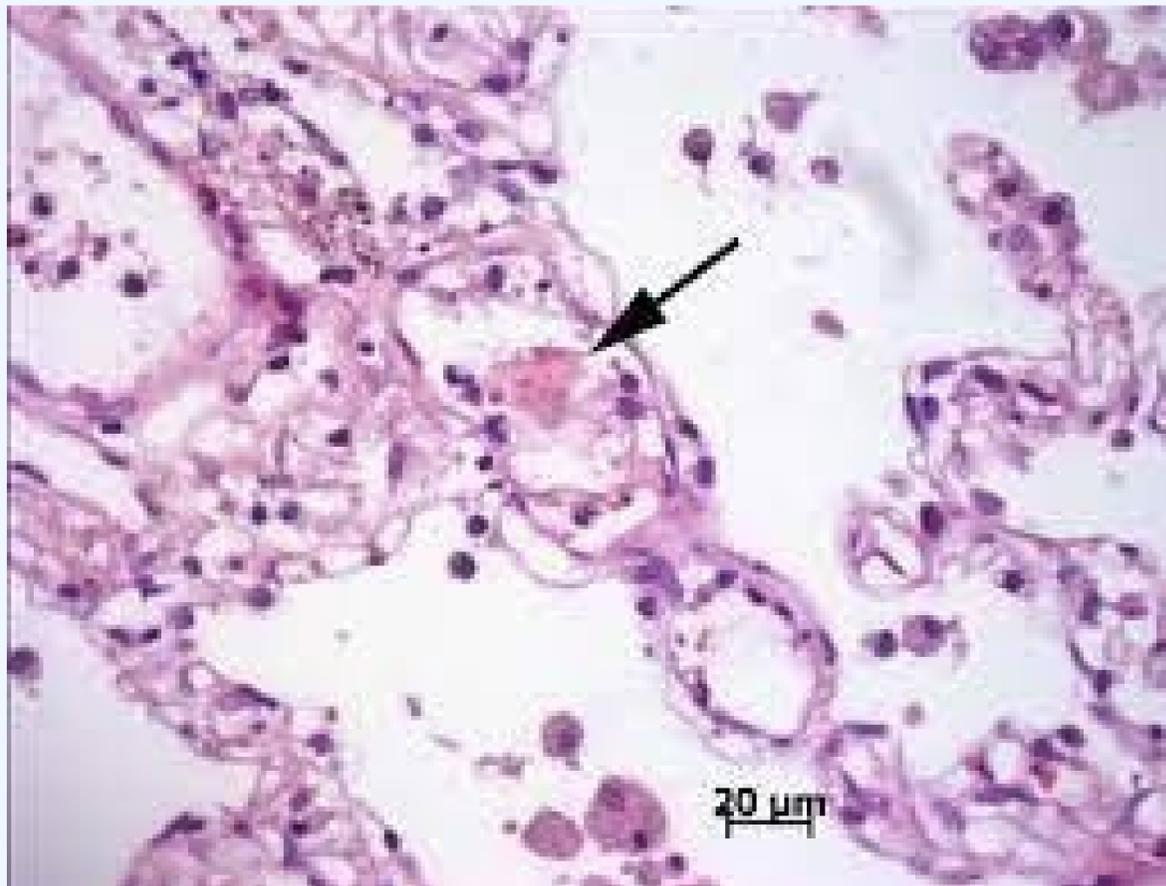
During labor: Suspect AFE in any sudden collapse.

Postpartum: Suspect AFE in unexplained DIC

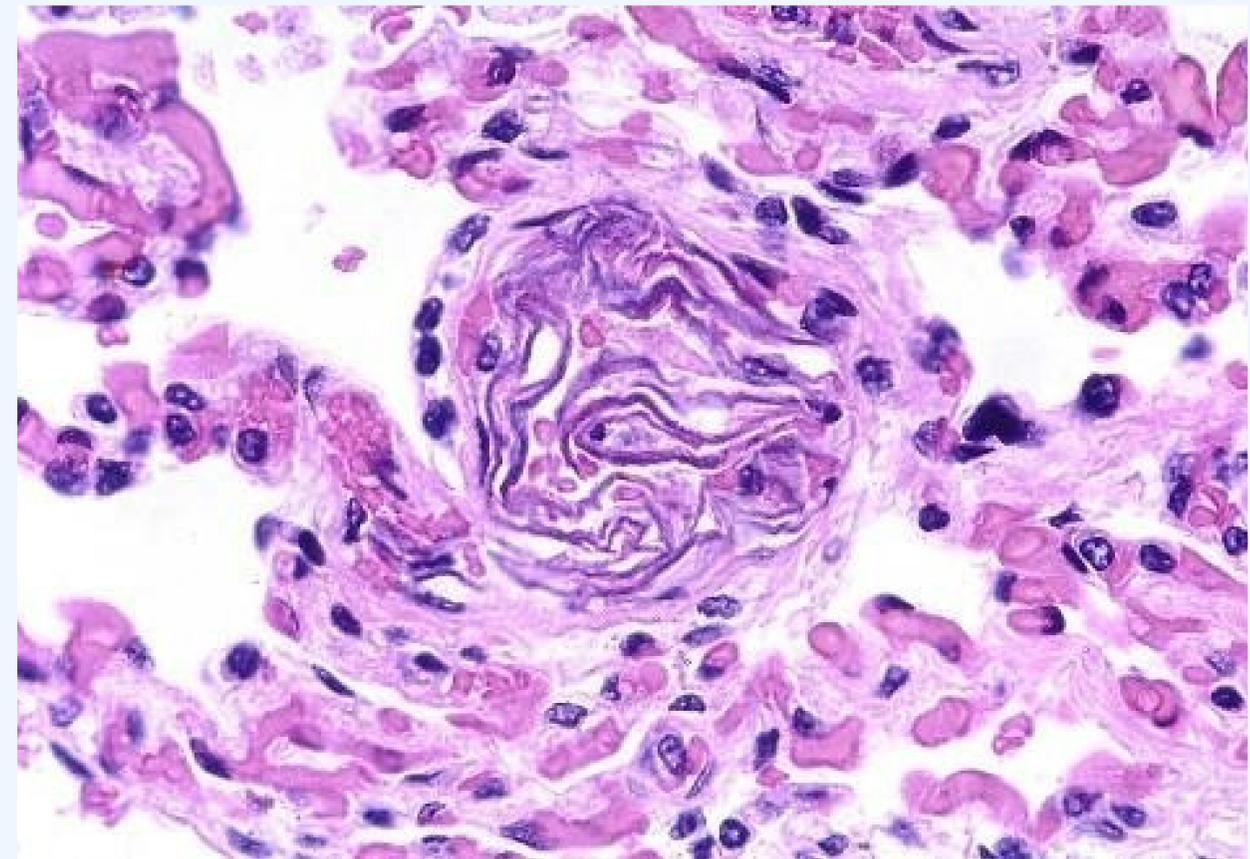
Diagnosis

- **continuous pulse oximetry and arterial blood gas (ABG)**
- **Serial CBC and coagulation studies**
- **Blood type and screen**
- **CXR -- postero-anterior and lateral findings are usually nonspecific.**
- **ECG -- may show tachycardia, ST-segment, and T-wave changes, and findings consistent with right ventricle strain.**
- **Lung scan – may demonstrate some areas of reduced radioactivity in the lung field.**
- **Increased serum tryptase, urinary histamine concentrations, and significantly lower complement concentrations suggest an anaphylactoid process.**
- **Decreased serum levels of C3 and C4 complement had sensitivities between 88% and 100% and a specificity of 100%.**
- **Bedside trans-esophageal echocardiography may aid early diagnosis by showing acute pulmonary vasoconstriction, right ventricular dilation, and a collapsed left ventricle with a leftward deviation of the intra-ventricular septum**

- The most significant pathologic findings at autopsy are limited to the lungs.
- Grossly, the lungs show evidence of **pulmonary edema (in 70% of the cases)**.
- **Alveolar hemorrhage and pulmonary embolism of amniotic fluid materials are present; the presence of embolic particles is essential for diagnosis, but on histology they may be missed because of their small size.**



Alveolar tissue section. A fetal squamous epithelial cell (arrow) derived from amniotic fluid is seen in the pulmonary vasculature



Squamous within a pulmonary arteriole.

Management tips:

1. Stable, non-life-threatening conditions → usually treat after diagnosis is reasonably confirmed.
2. Unstable or life-threatening conditions → often treat based on suspicion, not waiting for complete diagnosis.
3. Amniotic fluid embolism can lead to death within 5 - 10 mins because of cardiorespiratory collapse.
4. Amniotic fluid embolism → resuscitation starts immediately; diagnosis is retrospective.
5. Any bleeding emergency is managed using the trauma-style ABCDE principles.
 - * **Adequate ventilation providing oxygen**
 - * **Two large bore cannulas**
 - * **Start IV Fluids (NS + RL) // or colloids in hemodynamic collapse**
 - * **Folys catheter**
 - * **prepare at least 4 Blood Unit and FFP**

Management goals:

- Restoration of cardiovascular and pulmonary equilibrium immediately
- Maintain systolic blood pressure > 90 mm Hg
- Urine output > 25 ml/hr.
- Arterial pO₂ > 60 mm Hg.
- Re-establishing uterine tone
- Correct coagulation abnormalities

Immediate measures:

Set up IV Infusion, O2 administration.

- **Airway control : endotracheal intubation**
 - **maximal ventilation and oxygenation.**
-
- **LABS : CBC , ABG , PT , PTT , fibrinogen, Fibrin degradation products (FDP)**

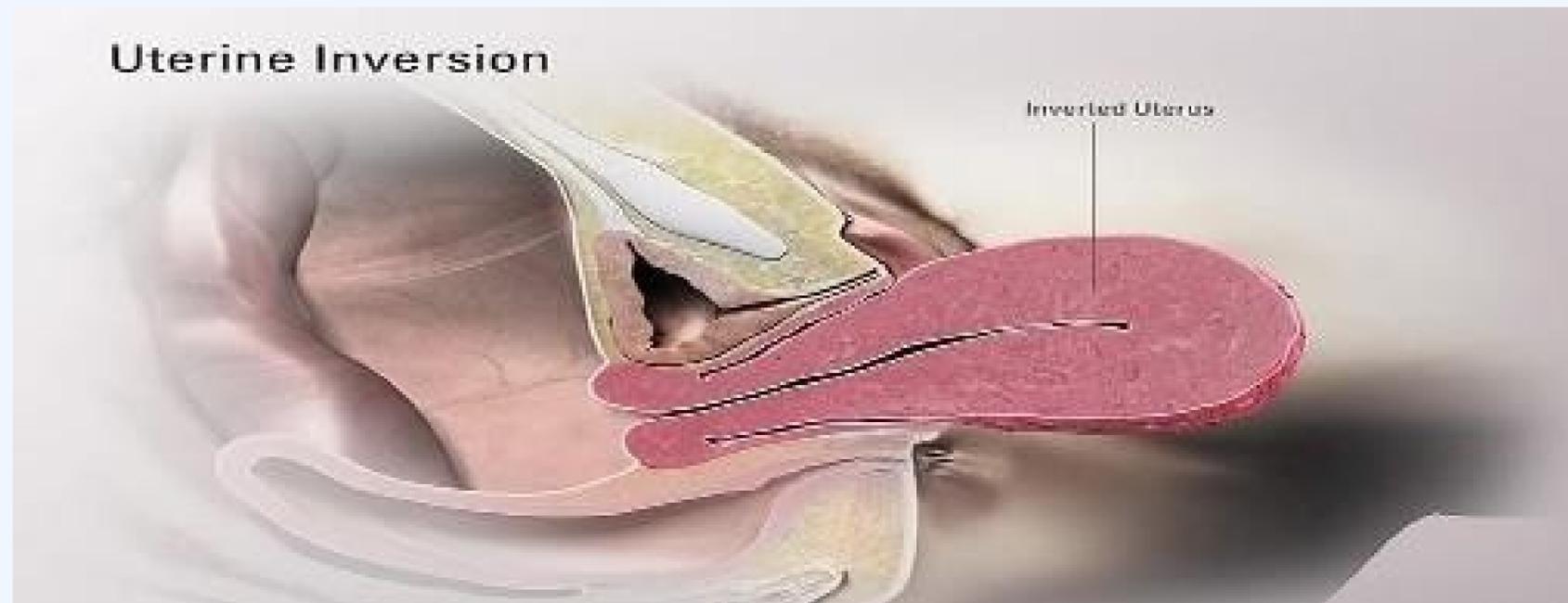
Management :

- **Oxygen**: endotracheal intubation and positive pressure respiration is usually indicated as the patient is often unconscious
- **Aminophylline**: to reduce bronchospasm.
- **Isoprenaline**: to improve pulmonary blood flow and cardiac activity.
- **Digoxin and atropine**: if central venous pressure is raised and pulmonary secretions are excessive
- **Hydrocortisone**: to cause vasodilatation and improves tissue perfusion
- **Bicarbonate solution**: if there is respiratory acidosis.
- **Low molecular weight dextran**: reduces platelets aggregation in vital organ.
- **Heparin**: for treatment of DIC if there is no active bleeding.
- **Vaginal delivery**: is safer than C.S. if the baby is not yet delivered

Uterine Inversion

Definition: it is a rare life-threatening obstetric emergency, occurs when the uterine fundus collapses and turns partially or completely inside out the endometrial cavity protruding through cervix into or beyond the vaginal canal.

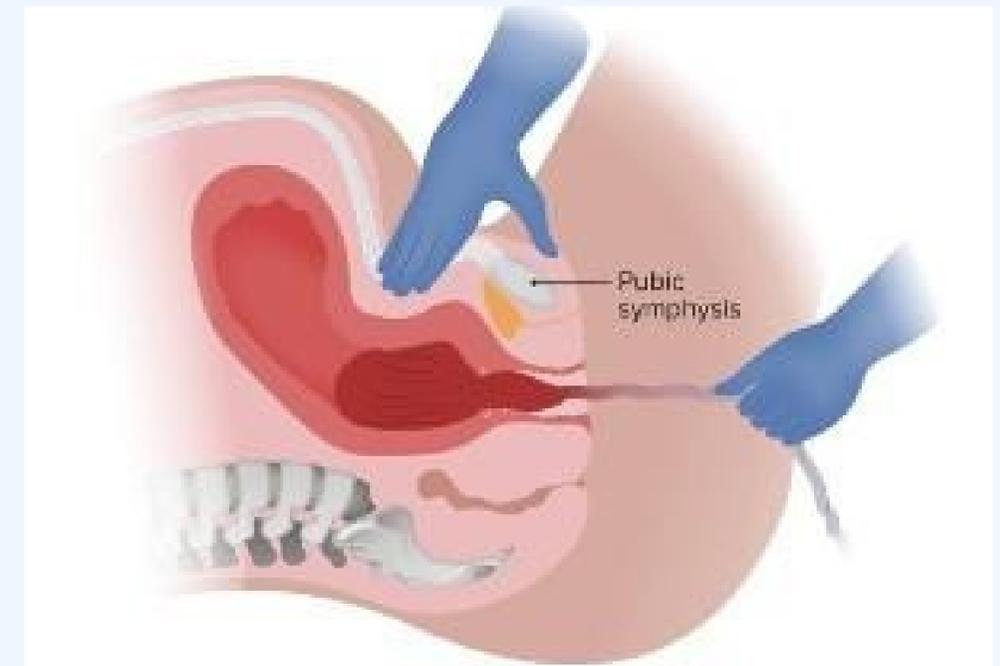
- Incidence: It's estimated to occur in 1 in 2500 deliveries.
- It may occur with Vaginal or CS deliveries??



Etiology: the most important precipitating factor is mismanagement of third stage of labor, particularly excessive traction on the umbilical cord before placental separation.

Risk factors:

- Uterine atony
- previous uterine inversion
- Usage of uterine relaxant (Tocolytics)
- Multiparity
- Fundal placental implantation
- placenta accreta
- short umbilical cord
- precipitous or prolonged labor
- Macrosomia



- * It is likely that other factors play role since
 1. These risk factors are present in fewer than 50 percent of inversion cases.
 2. spontaneous inversions sometimes occur
 3. inversion is rare even though cord traction and the Credé maneuver are used.

Pathophysiology

The inversion begins when the uterine fundus is depressed, often by traction or pressure (Credé maneuver), and collapses into the endometrial cavity.

→

As the inversion progresses, the uterus may protrude through the cervix and vagina. This condition is dangerous and can lead to maternal death for two reasons:

1. Massive hemorrhage due to uterine atony and exposed sinuses.
2. Neurogenic shock caused by stretching of the uterine ligaments and parasympathetic nerves causing vagal stimulation, which can make the patient's hemodynamic state disproportionately worse than the visible blood loss.

Clinical features:

sudden onset during 3rd stage of labor.

Symptoms

- Acute mild to severe lower abdominal pain
- Mild to severe vaginal bleeding
- dizziness, collapse, features of shock.

Signs

- A smooth, round mass protruding from the cervix or vagina (beefy-appearing bleeding).
- On abdominal examination, absence of uterine fundus by palpation.
- urinary retention.
- maternal shock, often out of proportion to blood loss??

- The most common presentation is a complete uterine inversion with severe postpartum hemorrhage, often leading to hypovolemic shock.

Incomplete uterine inversion:

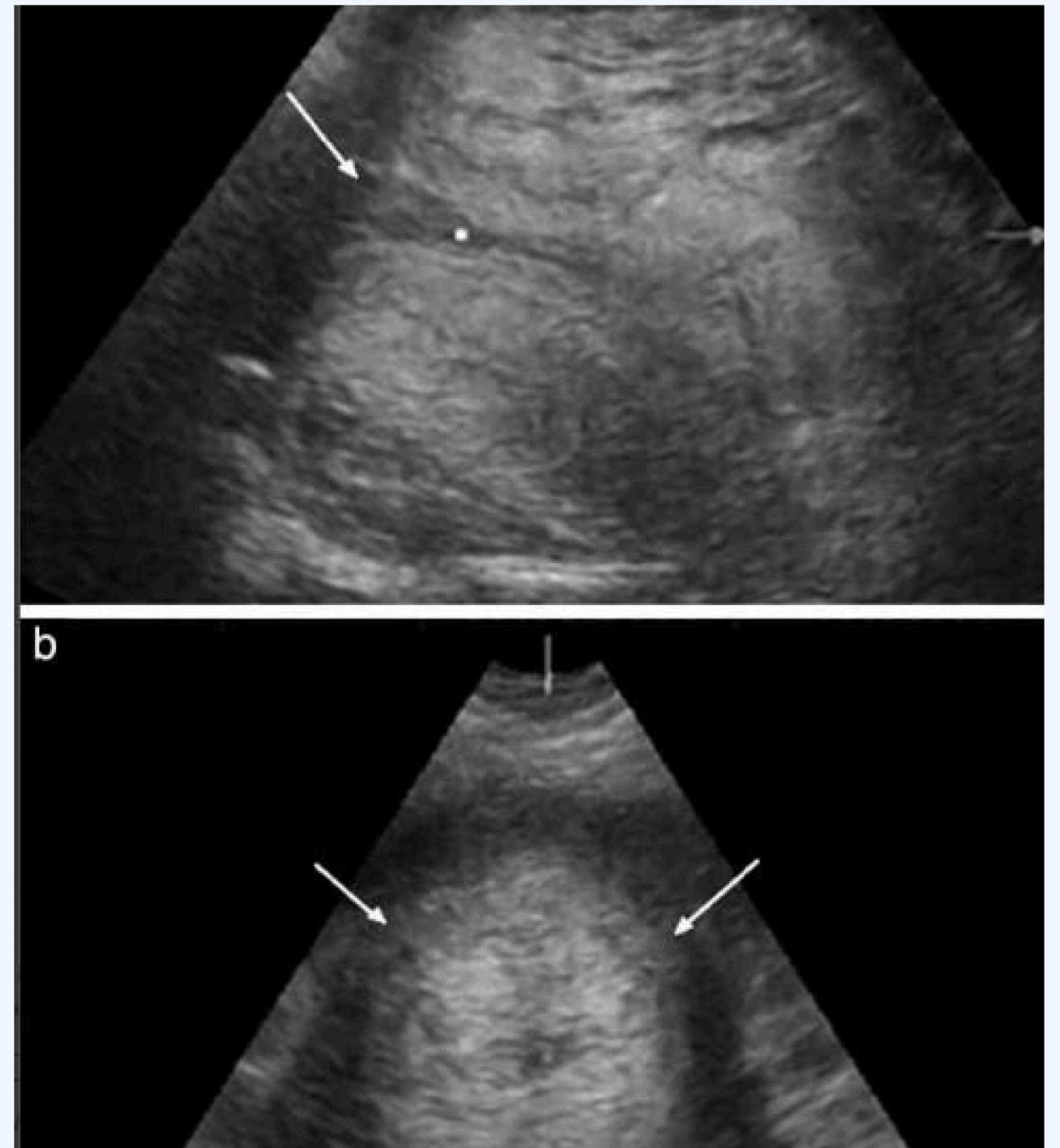
- occurs in approximately 10 % of cases and is associated with more subtle findings.
- blood loss may be **minimal**
- **Vaginal examination:**reveals a mass (fundus) in the uterine cavity
- **Abdominal examination:**a cup-like defect (fundal notch).
- a mass may not be seen externally and **postpartum hemorrhage** is usually the most striking symptom.
- These patients may not be identified for days or weeks.
- Because of increasing cervical constriction over time, delayed recognition of inversion is more likely to require surgical exploration



Figure 2 Intraoperative finding showing uterine inversion with lateral fallopian tubes.

Diagnosis

- Mainly **Clinically**
- Radiographic imaging (ultrasound, magnetic resonance) is rarely necessary, but has been used to confirm inversion when the diagnosis was uncertain and the patient was hemodynamically stable.
- Importantly, in women with significant vaginal bleeding, treatment **should not** be delayed for radiologic confirmation



Ultrasound images of the uterus in the sagittal plane showing the inverted fundus as a **central crater-like depression** (arrow) (a) and in the axial plane showing the **constriction ring** (arrows) (b).

Management



- 1- MANAGE POSTPARTUM HEMORRHAGE AND SHOCK, IF PRESENT
- 2- REPLACE THE UTERUS TO ITS CORRECT POSITION
- 3- MANAGEMENT OF THE PLACENTA
- 4- PREVENT RECURRENT INVERSION

In general, because obstetric shock considered as obstetric emergency, and because management of emergency cases is the same in pregnant and non-pregnant, so you have to start with ABCDE approach, or approach in the figure.

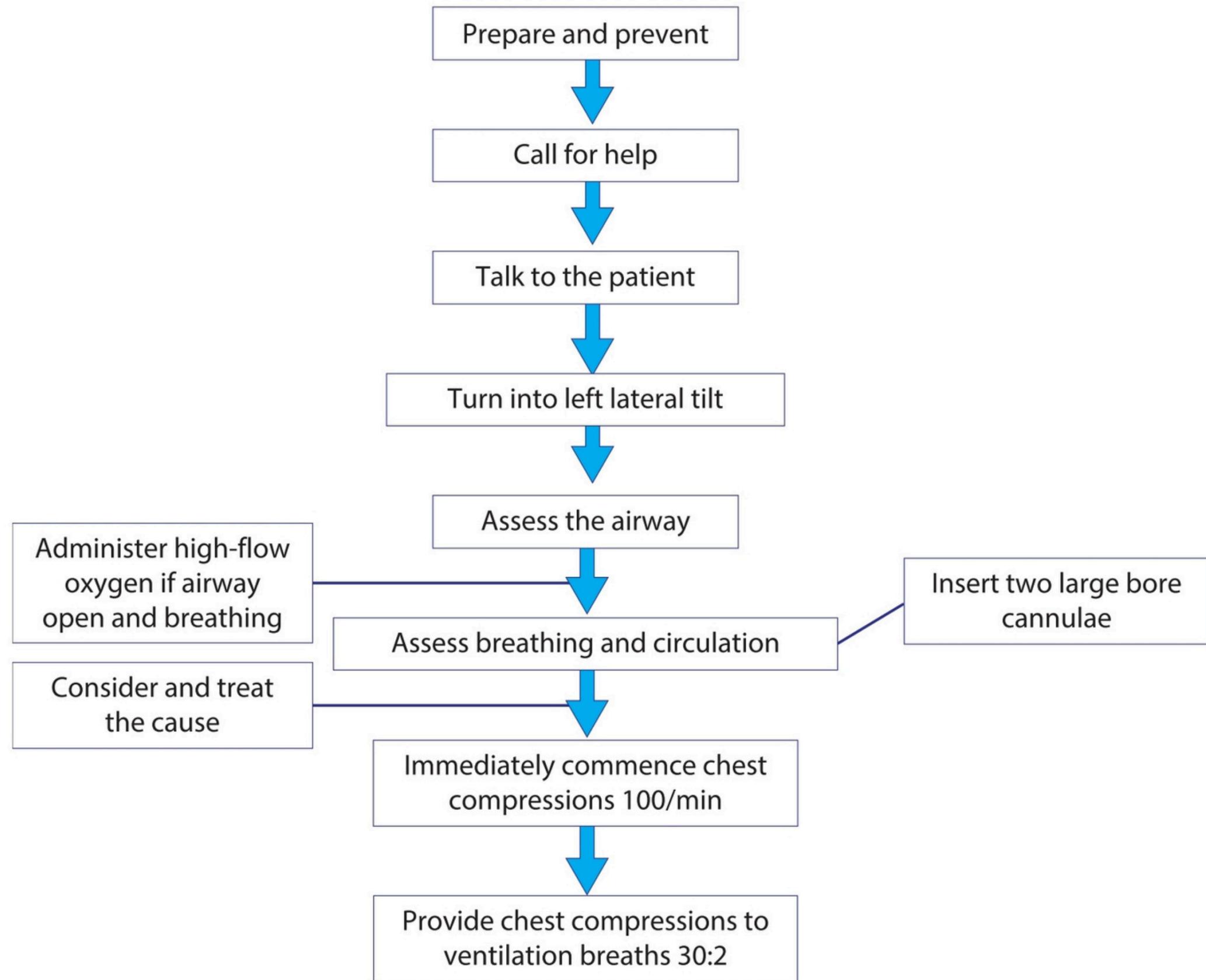
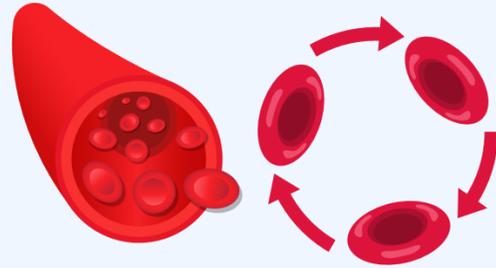


Figure 14.1 The structured approach to managing obstetric emergencies.

1) Manage postpartum hemorrhage and shock, if present.



You have to resuscitate the patient.

- 1) placement of two large bore intravenous lines (at least one intravenous catheter should be 16-gauge) and begin infusion of crystalloid to support blood pressure.
- 2) Draw blood for baseline laboratory tests, including (CBC, PT, PTT, cross-matching, urine monitoring).
- 3) Blood should be administered, as needed, to treat hypovolemia and prevent cardiovascular collapse

Q??

2) Replace the uterus to its correct position

In cases while the cause of shock is uterus inversion, you have to replace uterus in its correct position. Follow these points in management:

1. Discontinue uterotonic drugs (oxytocin) and give uterine relaxants (Tocolytic).
2. As soon as the diagnosis is made, manual replacement should be attempted.
3. If unsuccessful, transfer the patient to the operating theatre for replacement of the uterus under anaesthetic.
4. If unsuccessful, attempt hydrostatic replacement by running 2-3 L of warm saline via tubing into the vagina using your hands to create a seal around the vulva

1. Tocolytics

Nitroglycerin (glyceryl trinitrate administered IV), It has an extremely short half-life, which could be advantageous in women with severe hemorrhage and hemodynamic instability.

Also IV terbutaline & magnesium sulfate may be used.

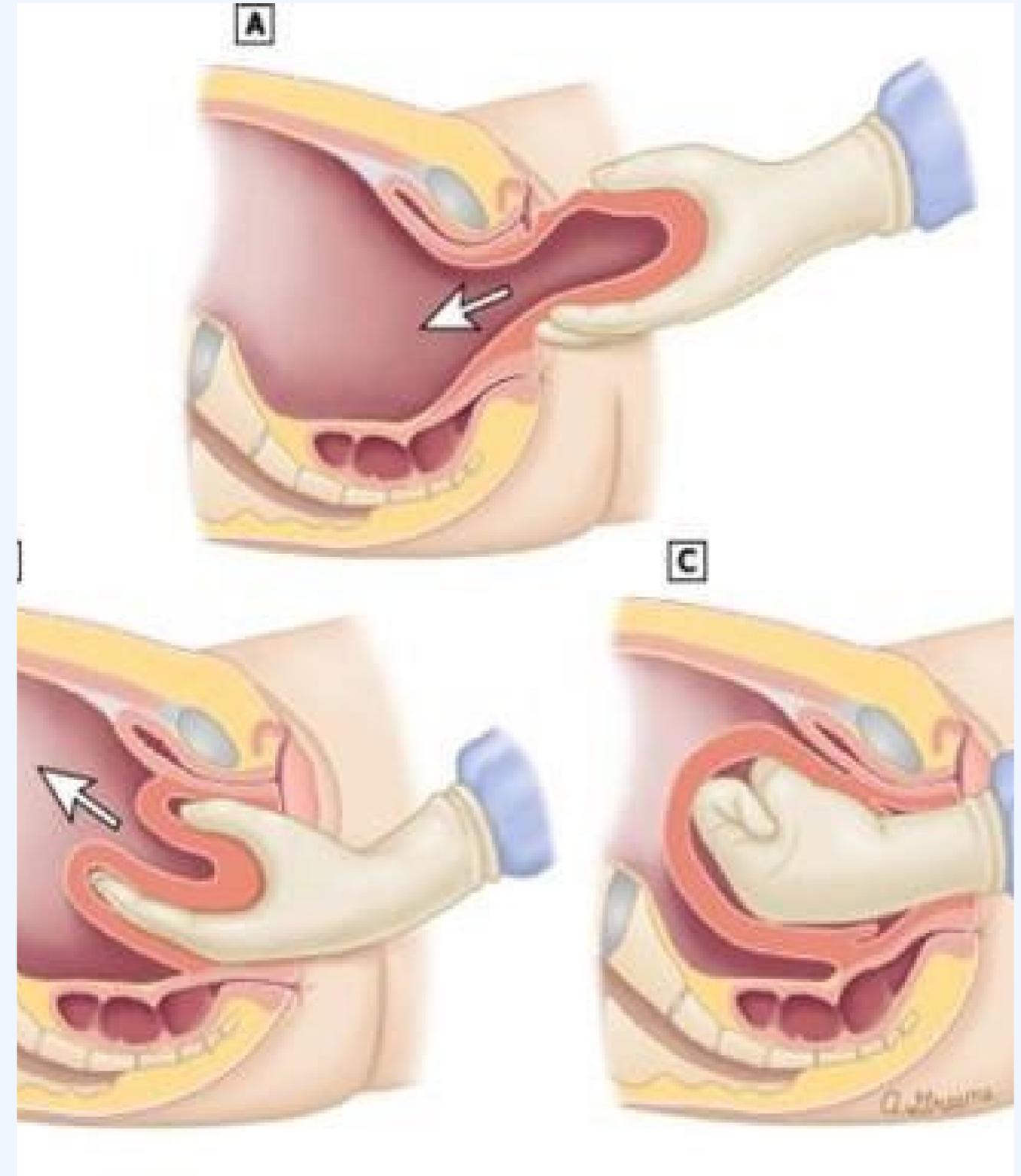
2. Manual replacement

- As soon as the diagnosis is made, manual replacement should be attempted.

-This is best accomplished by placing a hand inside the vagina and pushing the fundus with the fist along the long axis of the vagina toward the umbilicus (Johnson maneuver).

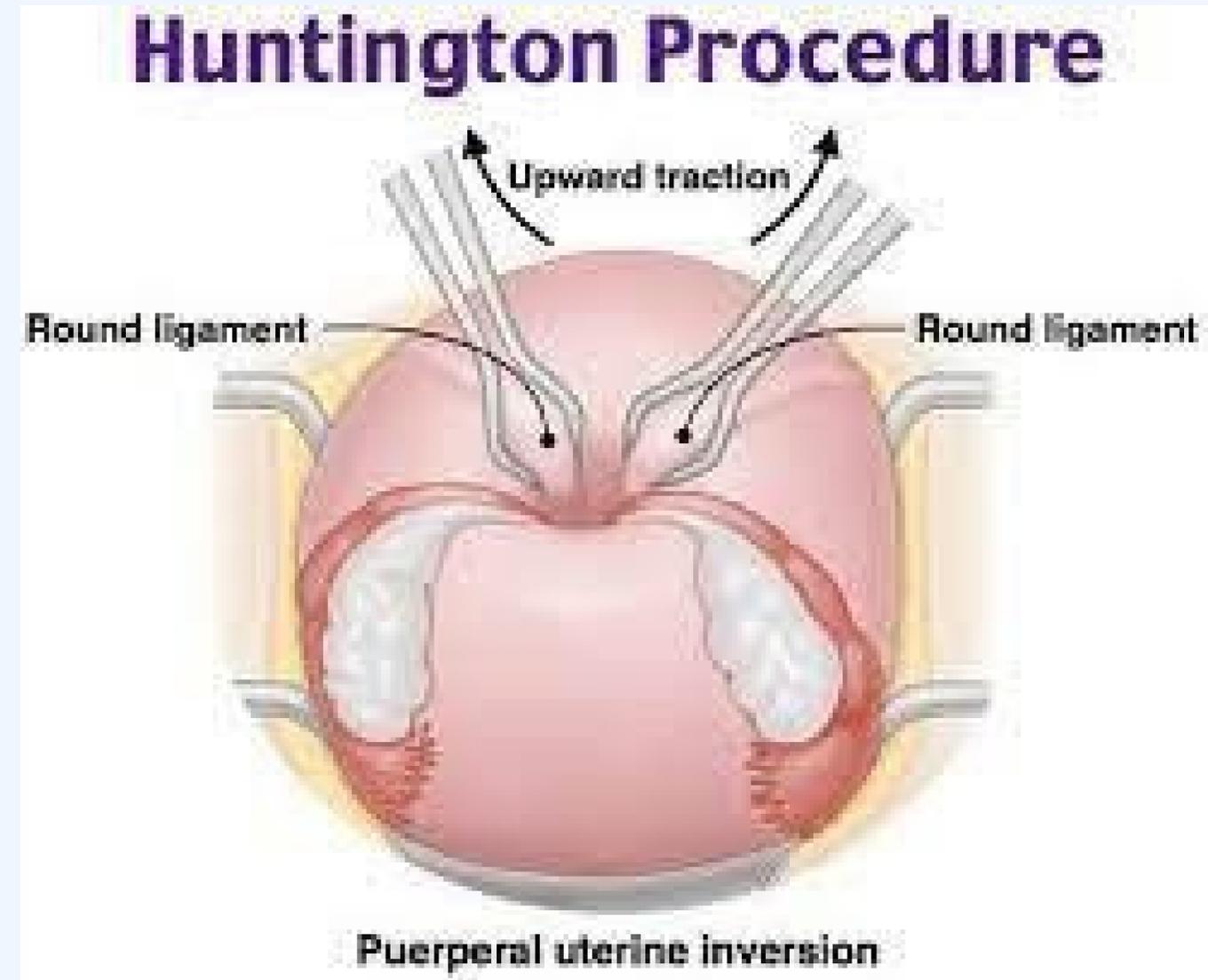
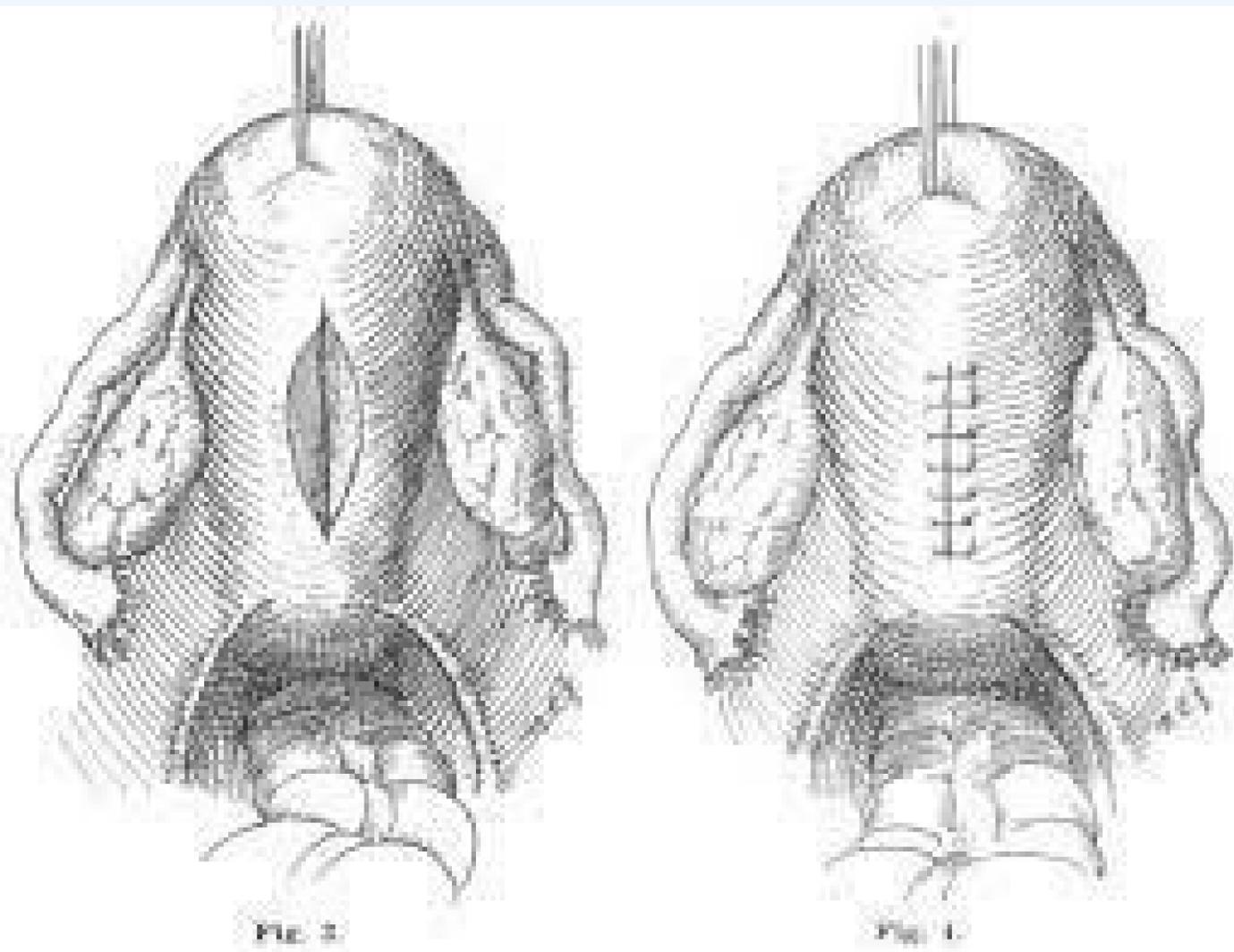
- the management of a uterine inversion requires quick thinking.

- The patient rapidly goes into shock, and immediate intravascular volume expansion with IV crystalloids is required



Secondary interventions

1. Huntington procedure.
2. Haultain procedure.
3. Hydrostatic reduction (if both are not possible)

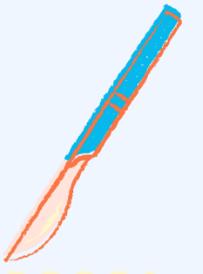


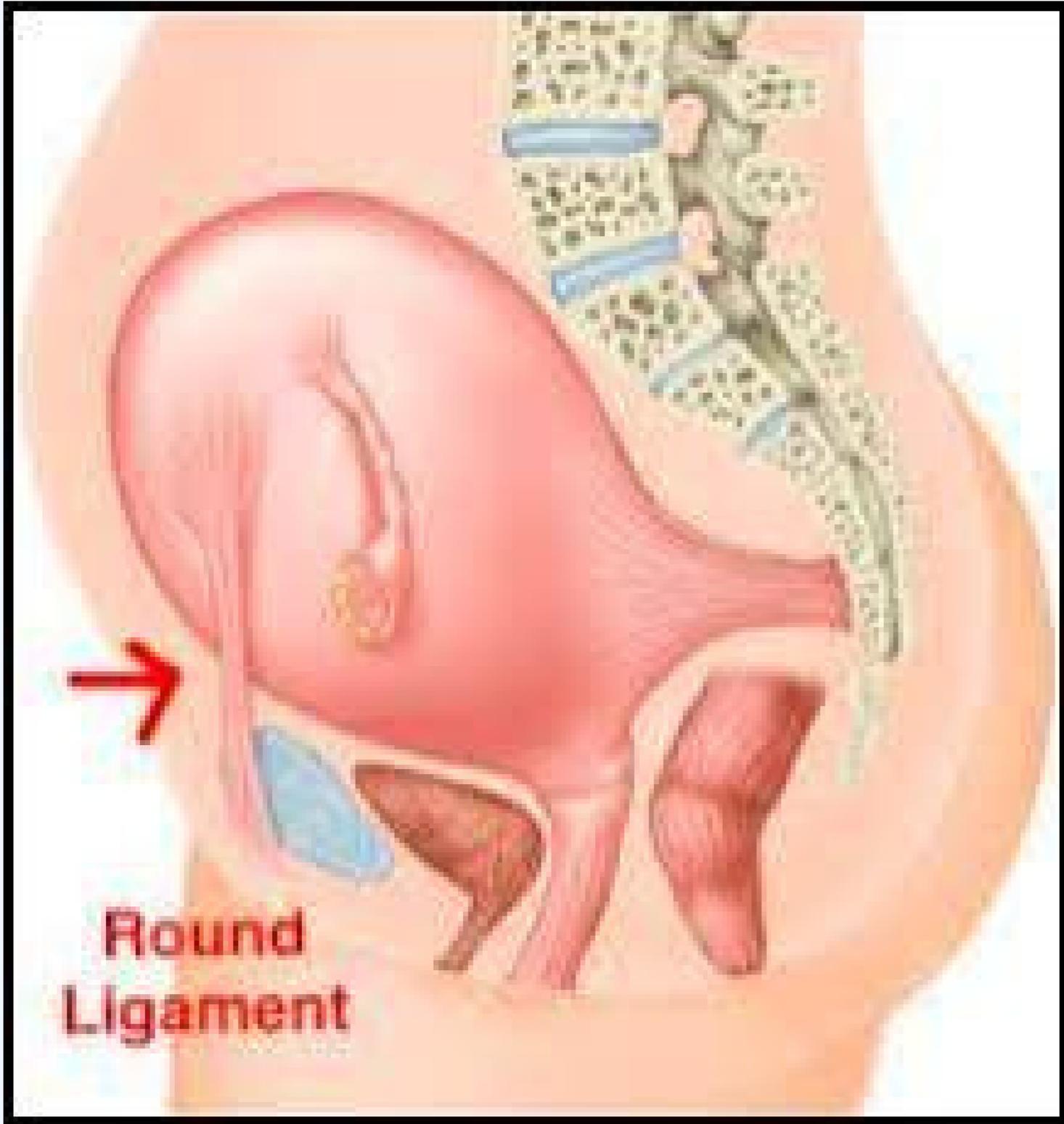


3. Surgical options

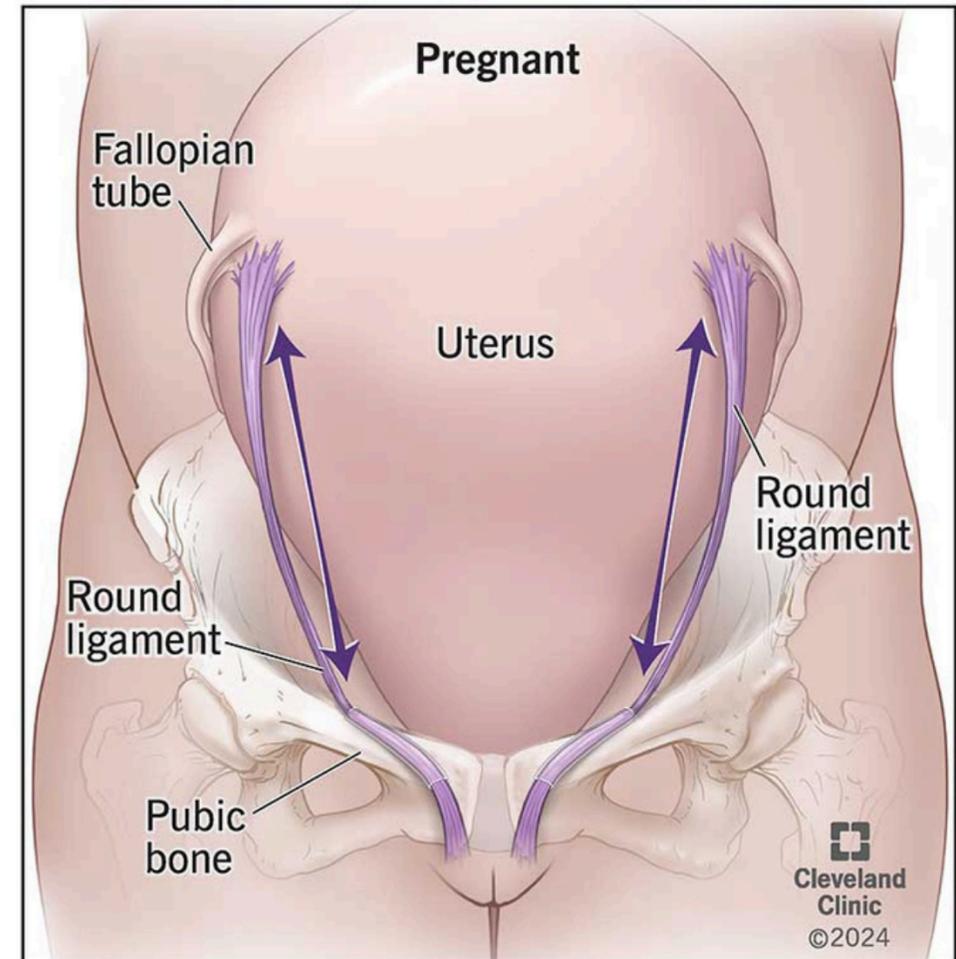
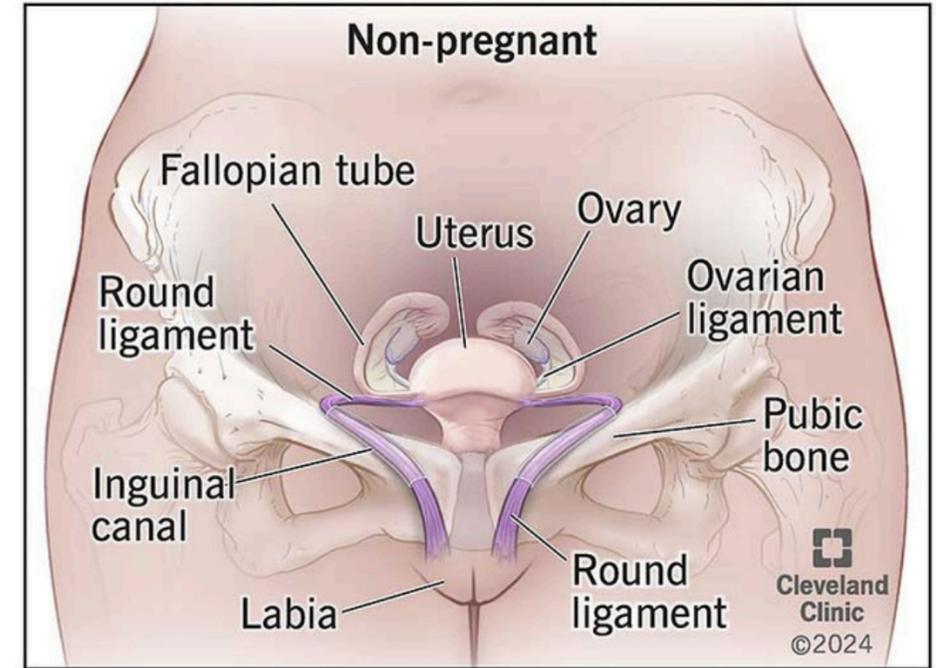
Include Huntington and Haultain procedures:

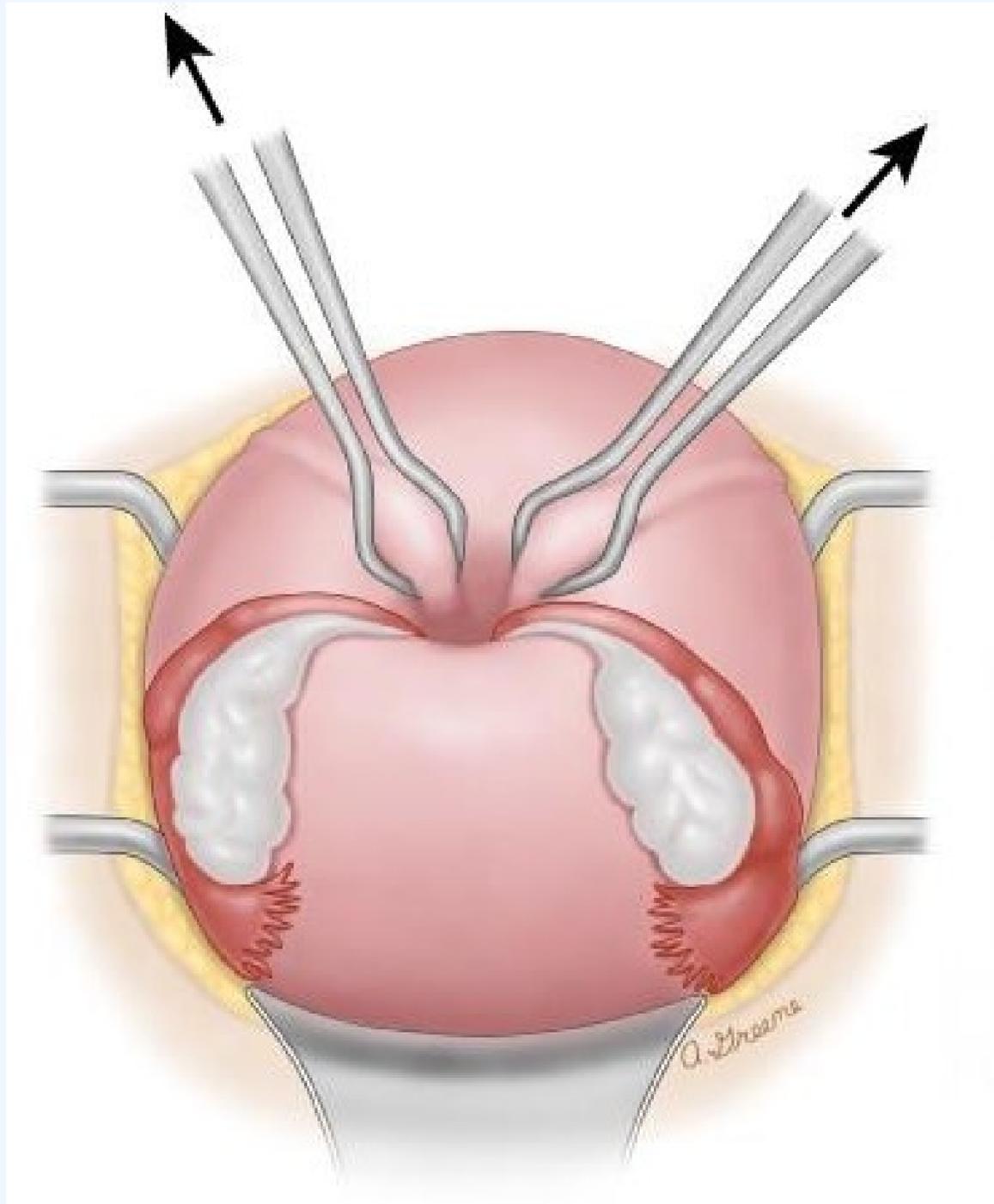
- laparoscopic-assisted repositioning, and cervical incisions with manual uterine repositioning.
 - **The Huntington procedure involves laparotomy by gradually pulling on the round ligaments** to restore the uterus to its proper position.
 - If the cervical ring is very tight, repositioning may be more easily achieved by incising the ring posteriorly with a vertical incision and manual pushing of the fundus. **(Haultain procedure)**
 - As with manual repositioning, after replacing the fundus, the anesthetic agent used to relax the myometrium is stopped, and uterotonic therapy is administered immediately, followed by repair of uterine incision.
 - **If these procedures are performed, then pregnancies in the future will require a cesarean delivery.**





Round Ligament





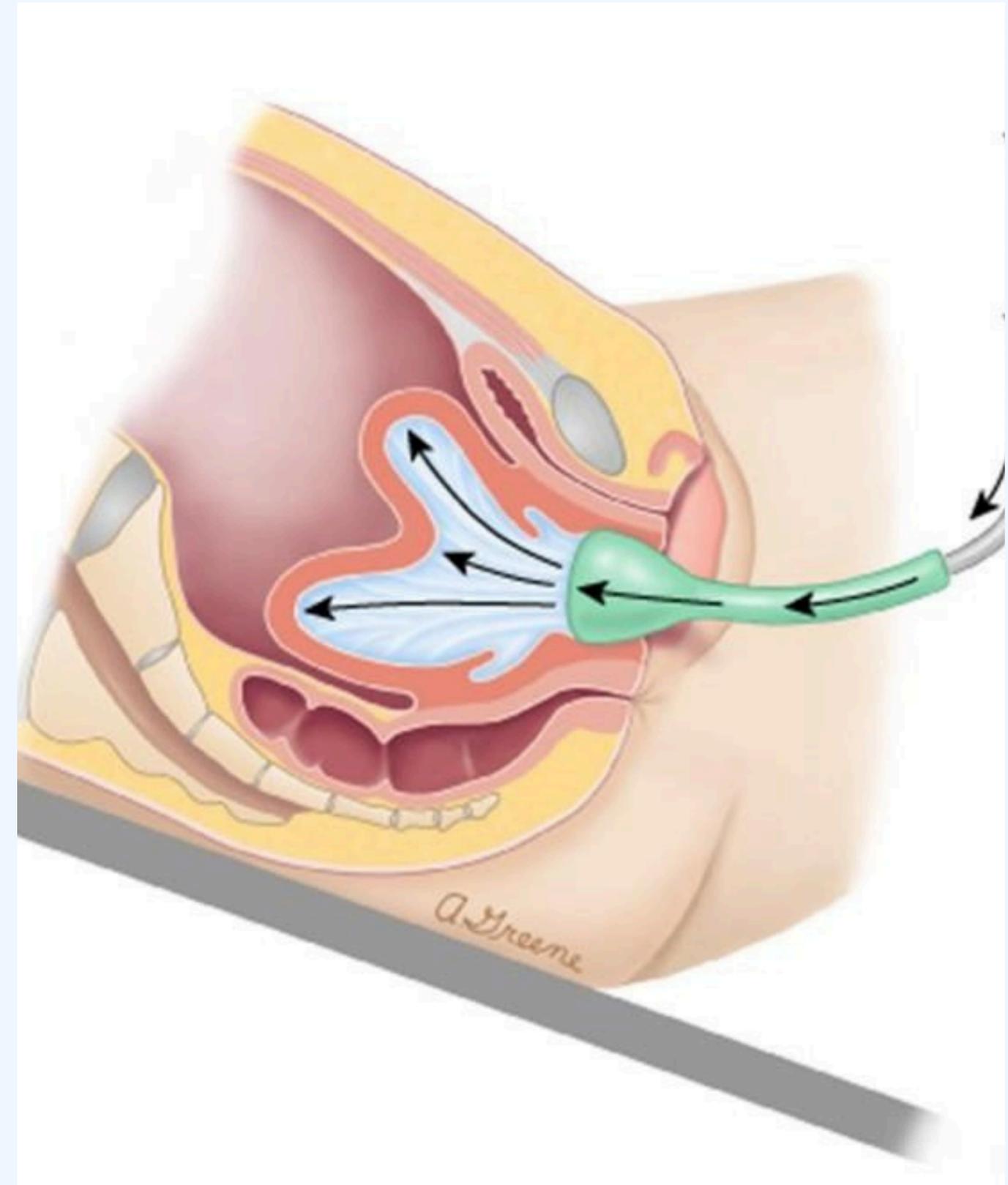
- **Locate the cup formed by the inversion.**
- **Place a clamp, such as an Allis or Babcock clamp, on each round ligament entering the cup, approximately 2 cm deep in the cup. Clamp the myometrium if the round ligaments cannot be identified.**
- **Gently pull on the clamps to exert upward traction on the inverted fundus.**
- **Repeatedly clamp in 2 cm increments along the ligament and exert traction until the inversion is corrected. This procedure is similar to the hand-over-hand movements used when pulling a line. If available, a second operator can place a hand in the vagina and apply upward pressure on the fundus to facilitate the procedure, or they can pull one of the clamps while the first operator pulls the other clamp.**

4. Hydrostatic reduction

- If manual reduction alone is not successful and surgical intervention is not possible, simple hydrostatic pressure may be of great assistance in pushing the fundus back to its normal anatomical position
- The main problem with hydrostatic replacement is developing a good enough water seal to allow generation of adequate hydrostatic pressures to allow uterine replacement.
- 2 to 5 liters of fluid may be needed
- also known as "O'Sullivan's method"

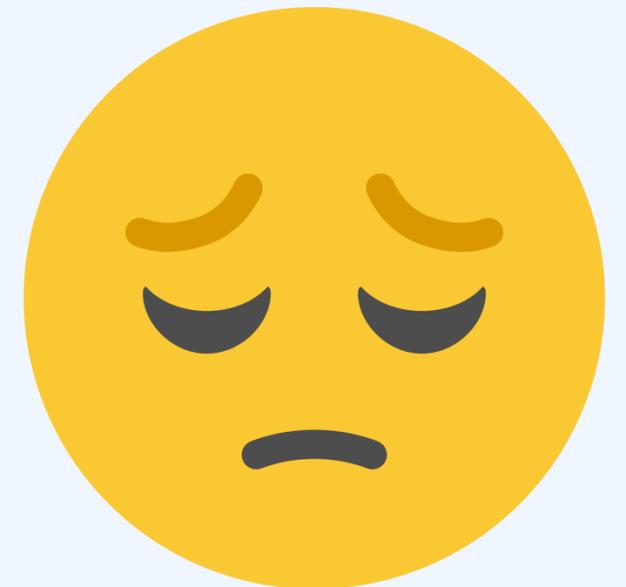


- The patient is placed in reverse Trendelenburg - lithotomy position.
- Warmed sterile saline is infused into the vagina.
- The clinician's hand or a silicone ventouse cup is used as a fluid retainer to generate intravaginal hydrostatic pressure and resultant correction of the inversion.
- The bag of fluid should be elevated about 100 to 150 cm above the vagina to guarantee sufficient pressure for insufflation.
- The resulting intravaginal hydrostatic pressure may force the inverted fundus back to its normal position.
- The possible complications associated with the procedure include **infection** , **failure of the procedure**, and **saline embolus**.





**If all methods was
unsuccessful, surgical
procedures including
hysterectomy may be
necessary.**



3) Management of the placenta

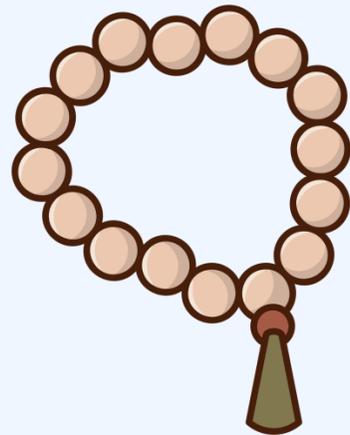
- After the uterus has been replaced, the most conservative approach is to wait a spontaneous separation of the placenta, and performing manual extraction for usual obstetric indications (hemorrhage, prolonged third stage)
- If the placenta is not separated from the uterus, then a hysterectomy may be necessary.
- do not remove the placenta until the uterus has been replaced. Removing the placenta before replacing the uterus increases blood loss, which may be severe.

4) Prevent recurrent inversion

- Atony is common after restoration of the normal uterine position.
- Once placental removal has been successfully accomplished, uterotonic agents are administered to induce myometrial contraction and maintain uterine involution, thereby impeding reinversion and reducing the risk of hemorrhage.

يقول ابن القيم رحمه الله تعالى: **"إذا حملت على القلب هموم الدنيا وأثقالها، وتهاونت بأوراده التي هي قوته وحياته، كنت كالمسافر الذي يحمل دابته فوق طاقتها ولا يوفيتها علفها، فما أسرع ما تقف به!"**

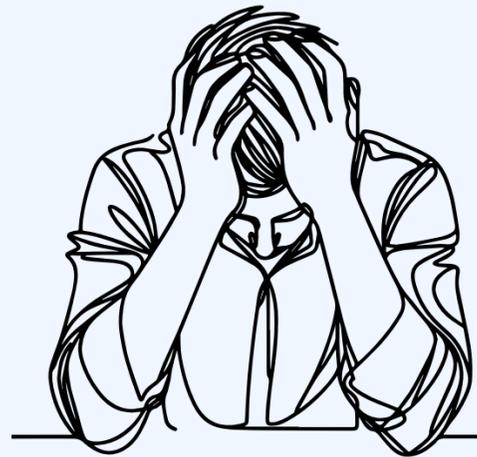
الأوراد



إنسان مستأنس بذكر
الله والعبادات، فتكون
حياته طيبة بهذا الأُنس
بالطاعة وبذوقه حلاوة
الإيمان



بدون أوراد

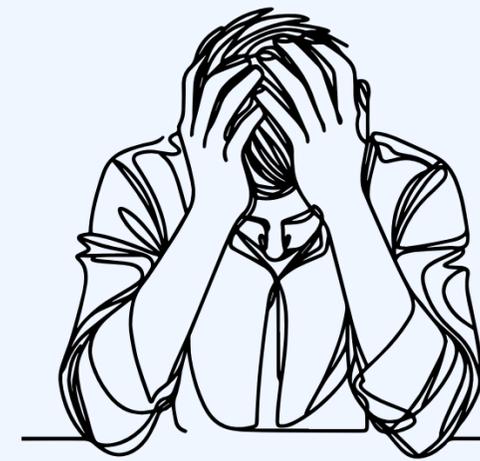
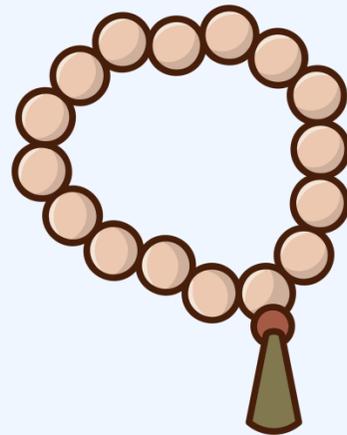


قال تعالى: ﴿مَنْ عَمِلَ صَالِحًا مِنْ ذَكَرٍ أَوْ أَنْتَى وَهُوَ مُؤْمِنٌ فَلَنُحْيِيَنَّهٗ حَيَاةً طَيِّبَةً وَلَنَجْزِيَنَّهُمْ أَجْرَهُمْ بِأَحْسَنِ مَا كَانُوا يَعْمَلُونَ﴾ [النحل: 97]

عجبا لمن يحمل أثقال الهموم ومشاكل الدنيا ومتاعبها وقلبه لا يملك القوة التي تمكنه من حمل تلك الأثقال!
قوة قلبك = أوراك.

الأوراد

إنسان مستأنس بذكر
الله والعبادات، فتكون
حياته طيبة بهذا الأُنس
بالطاعة وبذوقه حلاوة
الإيمان



Thank
you

