MATERNAL911

Amniotic Fluid Embolism

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Course Description:

Amniotic Fluid Embolism (AFE) is a high acuity, low occurring process that when recognized may be life-saving. The course will help by giving understanding of the disease and its management. AFE is a detrimental disease process that is life threatening for the women it effects. The AFE module will provide knowledge for this low occurring process.

Approximate Time to Complete: 40 minutes

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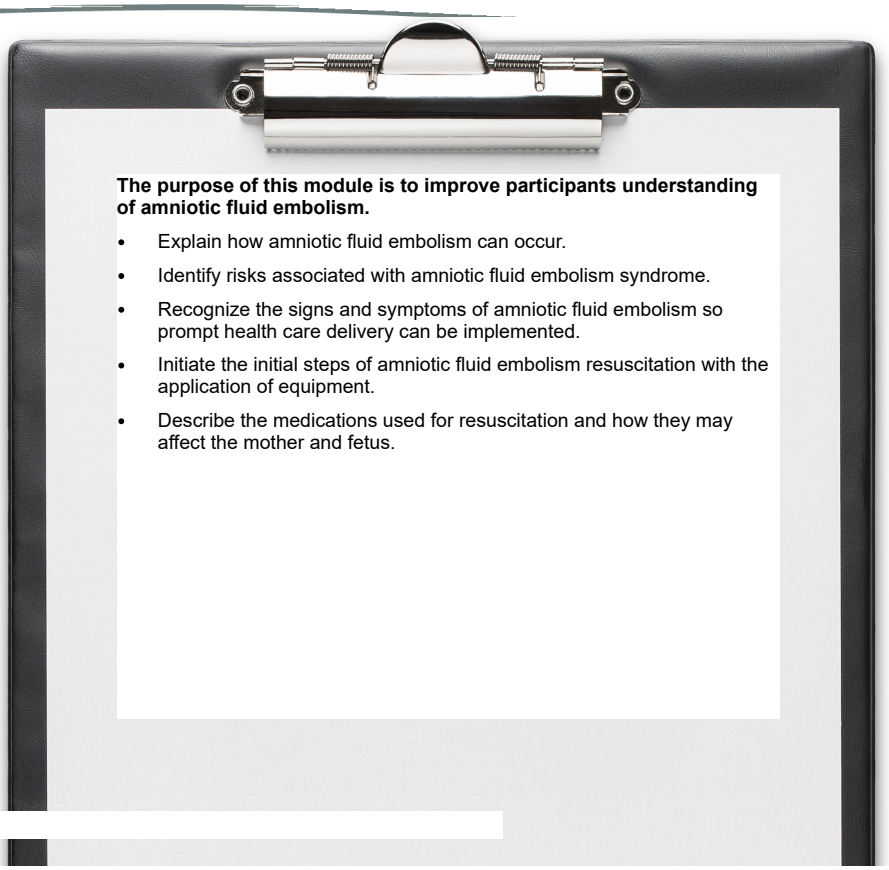
Introduction



The purpose of this module is to improve participants understanding of amniotic fluid embolism.

- Explain how amniotic fluid embolism can occur.
- Identify risks associated with amniotic fluid embolism syndrome.
- Recognize the signs and symptoms of amniotic fluid embolism so prompt health care delivery can be implemented.
- Initiate the initial steps of amniotic fluid embolism resuscitation with the application of equipment.
- Describe the medications used for resuscitation and how they may affect the mother and fetus.

Amniotic fluid embolism syndrome (AFES)

- *Also called anaphylactiod syndrome of pregnancy.*
- *A catastrophic condition that occurs during pregnancy or shortly after delivery [1-3].*

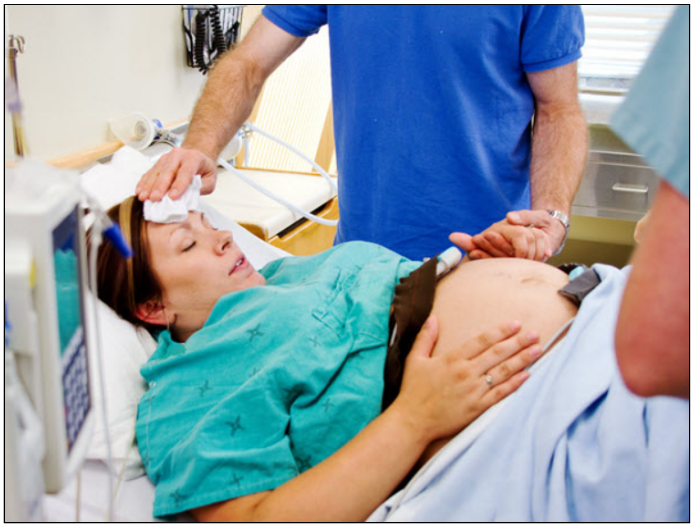
- First reported in 1926
- Not widely recognized until 1941.
- In 1941 autopsy's in a series of eight women dying from sudden shock during labor reported squamous cells and mucin of fetal origin in the maternal pulmonary vasculature [4,5].
- These same fetal squamous cells have been described in the vascular beds of maternal renals, liver, spleen, pancreas and brain [6].

- AFES is different from venous thromboembolism and is reviewed in a separate module.
- The incidence rate is between 1 - 12 cases per 100,000 deliveries in most studies [7-12].

Occurance

Several factors have been associated with AFES [7,12,13] they include:

- precipitous or tumultuous labor
- advanced maternal age
- cesarean and instrumental delivery
- placenta previa, placental abruption, placenta acreta, percreta, or increta
- grand multiparity (≥ 5 live births or stillbirths)
- cervical lacerations
- fetal distress
- eclampsia
- medical induction of labor
- uterine rupture
- polyhydramnios
- miscarriage or abortion
- amniocentesis

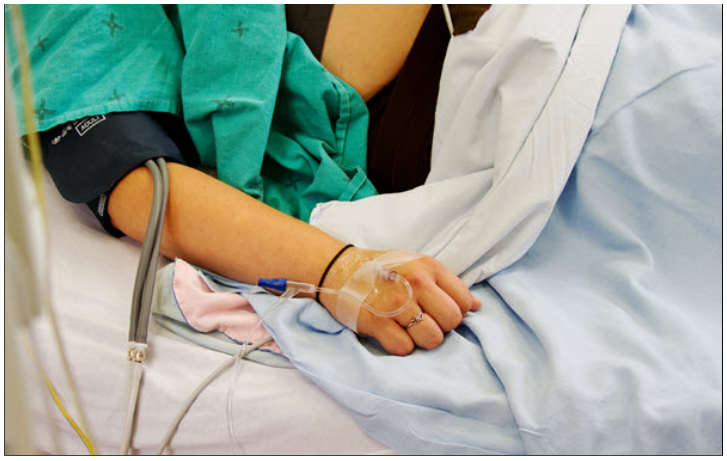


These factors are associated with the pathogenesis of AFES but not the direct cause.

AFES is best considered unpreventable and unpredictable.

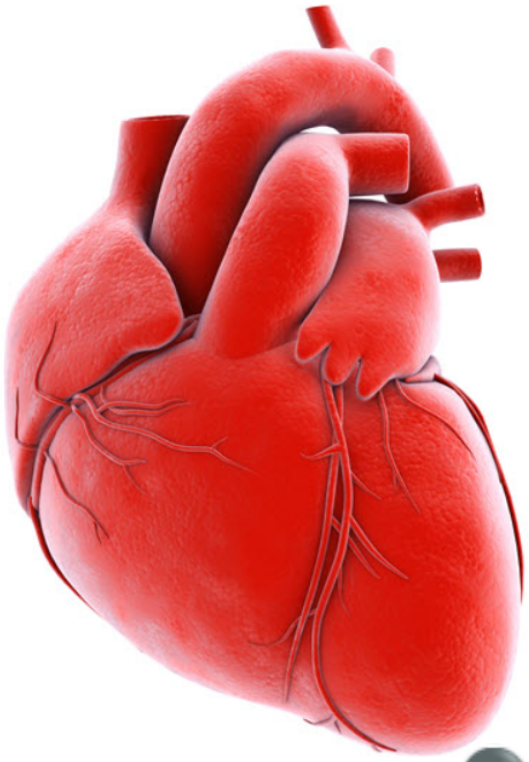
Stage or duration of labor has not been associated with risk for AFE.

- The pathogenesis of AFES is thought to involve the amniotic fluid entering the maternal circulation through endocervical veins, the placental insertion site or a site of uterine trauma [14].
- Once the amniotic fluid reaches the maternal circulation it can precipitate cardiogenic shock, respiratory failure and most likely, an inflammatory and anaphylactoid response.



- Amniotic fluid has been shown to cause occlusion and vasospasm of the maternal pulmonary vasculature with animal studies, resulting in rapid development of pulmonary hypertension, acute cor pulmonale and systemic hypotension [15-17].
  - In women with AFES, invasive hemodynamic studies seem to contradict these animal studies [18,19].
  - When elevated pulmonary arterial pressure, elevated pulmonary capillary wedge pressure, decreased cardiac output and decreased cardiac index are combined it suggests the principal hemodynamic alteration in humans is left ventricular failure, rather than pulmonary hypertension and right ventricular failure.

- To reconcile the human and animal observations, a biphasic pattern of cardiogenic shock in AFES has been proposed [2,15,19].
- Initial acute pulmonary hypertension and right ventricular failure (usually lasting 15-30 minutes), according to the hypothesis, is followed by left ventricular
dysfunction [20,21].
- Studies utilizing transesophageal echocardiography to non-invasively measure the hemodynamic parameters early during AFES report vasospasm of the pulmonary vasculature, elevated pulmonary arterial pressure and right ventricular failure, thus supporting the bi-phasic hypothesis [20,21].
- The mechanism is unclear with the later phase left ventricular failure.



Mouse over the heart to see what the mechanism may involve.

- Among patients with AFES, hypoxemia is the most common manifestation of the resultant respiratory failure.
- Hypoventilation can also occur.
- The primary cause of hypoxemia involves severe ventilation/perfusion (V/Q) mismatching.

Factors contributing to the V/Q mismatch [2] :

- Acute pulmonary hypertension during the first phase of cardiogenic shock
- Cardiogenic pulmonary edema during the second phase

Other contributors may include [8,22] :

- Bronchospasm (about 15 percent of patients)
- Noncardiogenic pulmonary edema

- In 70% of patients who survive the first several hours, noncardiogenic pulmonary edema occurs [22].
- It generally develops as left ventricular dysfunction improves.
- Damage to the endothelial-alveolar membrane and capillary leak syndrome are evidence leading to high protein concentration in edematous fluid and the presence of amniotic debris in sputum and alveolar spaces.
- Widespread damage to the alveolar-capillary membrane causes non-cardiogenic edema to occur in AFES; it usually does not produce the clinical pattern typical of acute respiratory distress syndrome (ARDS).
- Women who survive the first few hours of AFES usually recover quite rapidly, whereas the course of ARDS tends to be protracted.

- Obstruction of the pulmonary vasculature seems unlikely to be the lone cause of AFES, since there is often a lag of many hours between the entry of amniotic fluid into the the maternal circulation and onset of symtoms and signs of AFES.
- Propositions have brought up how the lag may reflect evolution of AFES of an immunologic response or inflammatory reaction to the amniotic fluid.
- Support is evident by reports of decreased complement and increased inflammatory markers in some patients with AFES, including elevated serum tryptase levels and pulmonary mast cell activity [23-28].
- It is hypothesized that maternal circulation allows entry of fetal antigens via the amniotic fluid.
- The clinical manifestations and the severity may be related to the degree of immunologic stimulation or the balance of arachidonic acid metabolites such as leukotrienes in the amniotic fluid [29,30].

- The symptoms and signs associated with AFES have a typical onset during labor and delivery, or immediately postpartum [2].
- In rare instances it has been reported following first or second trimester abortion, amniocentesis, or abdominal/uterine trauma and as late as 48 hours after cesarean delivery or post partum, [8.31-35].
- In review of 272 cases, most women present with rapid cardiorespiratory collapse [10].
- Preceding the onset of dyspnea and hypotension may be nonspecific symptoms such as chills, nausea, vomiting and agitation [8,10].
- Tonic-clonic seizure activity may also occur.
- A less severe presentation of AFES, partial amniotic fluid embolism syndrome, may present with only some of the major symptoms and signs [15,36,37].
- These patients with partial amniotic fluid embolism generally present with sudden onset of milder dyspnea and hypotension.
- In this situation, the clinical course tends to be abbreviated and the prognosis much better compared to those who have the full syndrome.

Clinical Presentation

Click the terms in blue to learn more.

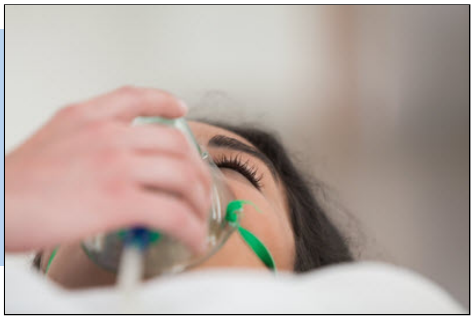
The major clinical findings are the abrupt and fulminant onset of:

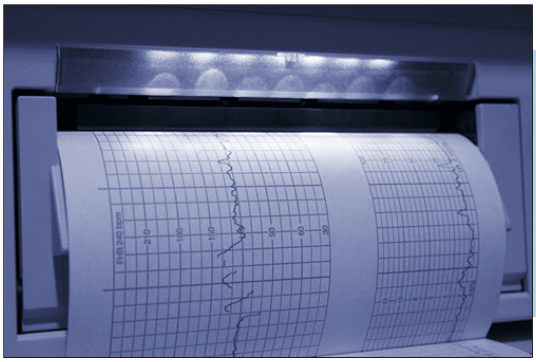
- Hypotension due to cardiogenic shock
- Disseminated intravascular coagulation (DIC)
- Hypoxemia and respiratory failure
- Coma or seizures

AFES is diagnosed from clinical findings based on the constellation of clinical findings, rather than isolated signs and symptoms.

- AFES should be suspected whenever shock and/or respiratory compromise develops during labor or immediately postpartum.
- Other causes of sudden intrapartum or postpartum cardiorespiratory failure must be excluded.
- Sometimes the identification of amniotic fluid debris (squamous cells, trophoblastic cells, mucin, and lanugo) from blood samples drawn from the distal port of a pulmonary artery catheter.
- Such debris is common in maternal circulation without AFES, so finding the amniotic fluid debris alone should not be considered diagnostic [43] but should involve the constellation of signs and symptoms.

- There is no specific treatment for AFES.
- The therapeutic goal is to correct hypoxemia and hypotension so that ischemic consequences (i.e. hypoxic brain injury, acute kidney injury) are prevented in the mother and adequate oxygen delivery occurs to the fetus.



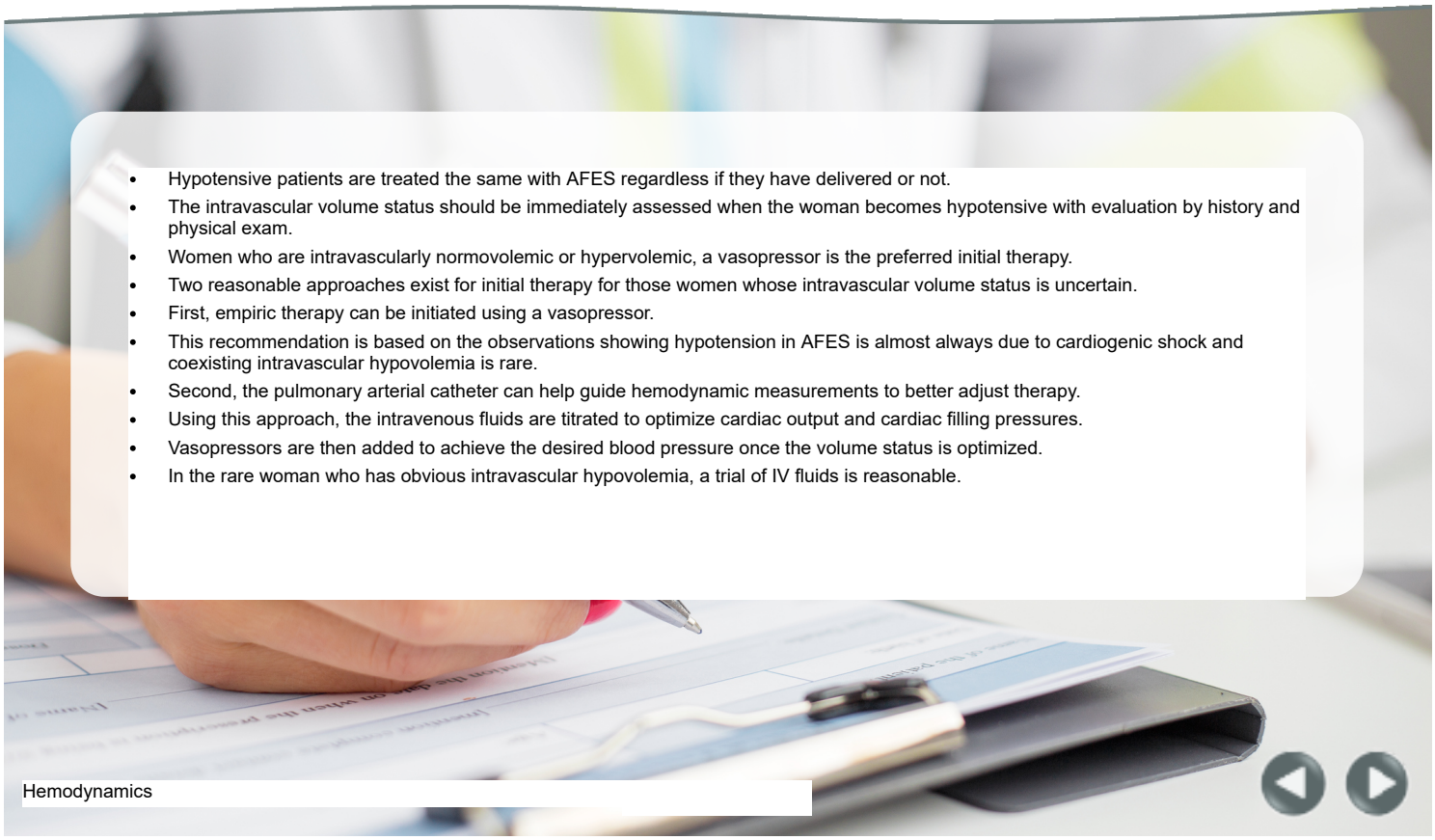


- Monitoring of maternal oxyhemoglobin saturation, heart rate and rhythm, and respiratory rate should be immediately initiated in all patients with suspected AFES.
- It is important to monitor the blood pressure non-invasively at frequent intervals until continuous blood pressure monitoring is established.
- Continuous monitoring of the fetal heart rate is desirable.

Catheters

- Following the initial assessment, plans should be made for both an arterial and a central venous catheter to be inserted.
- The initiation of therapies described below should not be delayed for catheter insertion as these procedure can be time consuming.
- The arterial catheter can be used to continuously monitor blood pressure.
- It also provides access to arterial blood for frequent measurement of arterial blood gases.

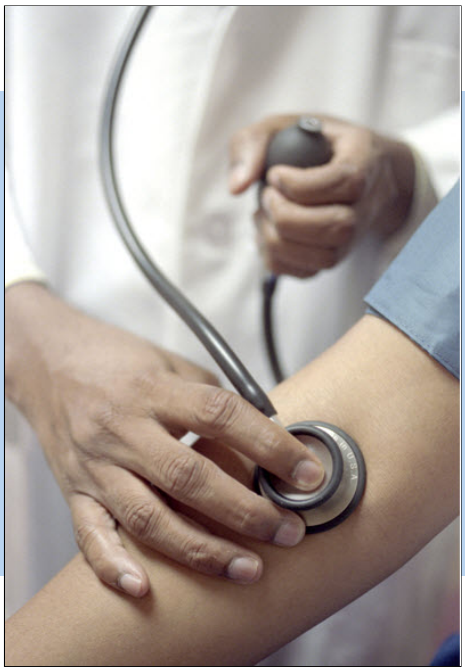
- Supplemental oxygen should be provided to all patients.
- Reaching a maternal oxygen tension (PaO2) above 65 mmHg is a reasonable goal.
- To reach this goal, it often requires high flow rates of supplemental oxygen by facemask or invasive mechanical ventilation.
- When invasive mechanical ventilation can occur it provides additional options to improve oxygenation. The options include:
- Increasing the fraction of inspired oxygen
- Increasing the positive end-expiratory pressure
- Prolonging or inversing the inspiratory to expiratory ratio.
- Due to the associated high risk of aspiration, non-invasive positive pressure ventilation should be avoided during pregnancy.



- Hypotensive patients are treated the same with AFES regardless if they have delivered or not.
- The intravascular volume status should be immediately assessed when the woman becomes hypotensive with evaluation by history and physical exam.
- Women who are intravascularly normovolemic or hypervolemic, a vasopressor is the preferred initial therapy.
- Two reasonable approaches exist for initial therapy for those women whose intravascular volume status is uncertain.
- First, empiric therapy can be initiated using a vasopressor.
- This recommendation is based on the observations showing hypotension in AFES is almost always due to cardiogenic shock and coexisting intravascular hypovolemia is rare.
- Second, the pulmonary arterial catheter can help guide hemodynamic measurements to better adjust therapy.
- Using this approach, the intravenous fluids are titrated to optimize cardiac output and cardiac filling pressures.
- Vasopressors are then added to achieve the desired blood pressure once the volume status is optimized.
- In the rare woman who has obvious intravascular hypovolemia, a trial of IV fluids is reasonable.

Hemodynamics

- When vasopressor therapy is warranted, norepinephrine and dopamine are the typical drugs of first choice.
- The inotrope, dobutamine, may be added and would likely be beneficial since it increases the low cardiac output and decreases the high afterload that are characteristic of cardiogenic shock.
- However, until the vasopressors have improved blood pressure, dobutamine should not be used.
- Dobutamine, when used alone, tends to reduce blood pressure by causing a drop in the systemic vascular resistance that is out of proportion to the increase in cardiac output.



Vasoactive Drugs

Vasopressors

Stimulates smooth muscle contraction of the capillaries & arteries

↓

Vasoconstriction

Ionotropes

Increase the force of contraction of myocardial muscle

↓

Positive ionotropism

↘ ↙

Rise in Mean Blood Pressure

▼

Improved tissue perfusion and oxygenation

- Vasopressors are used to treat hypotension in AFES even though they may diminish uteroplacental perfusion pressure.
- The rationale is found in the fact that untreated shock diminishes uteroplacental perfusion pressure and has numerous additional potential adverse consequences.

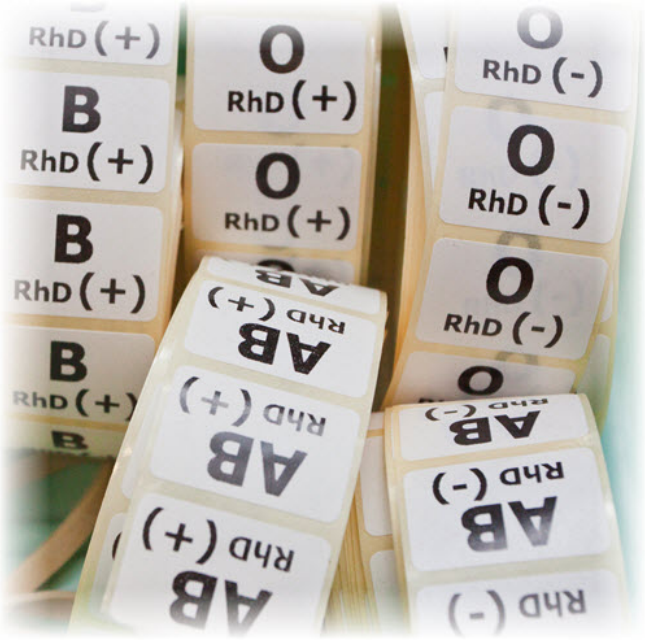
These include:

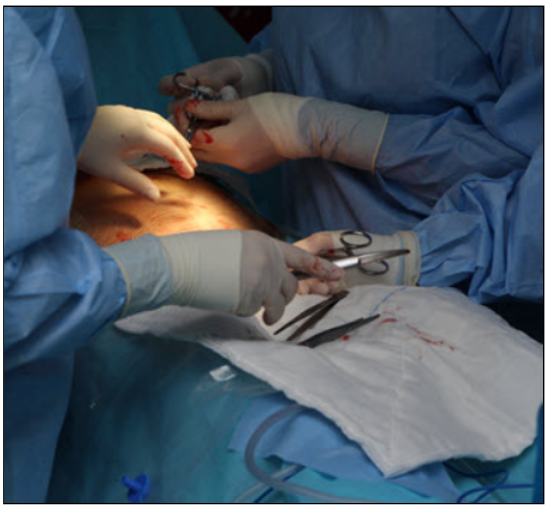
- Decreasing fetal oxygen delivery
- Increasing the mother's risk of ischemic complications
- Acute kidney injury
- Hypoxic brain injury
- Increasing mother's risk of death

IV Fluids

- Pulmonary edema is common in AFES so a cautious approach is necessary whenever a trial of IV fluid is selected.
- When IV fluid is given, it is necessary to be administered in small boluses with recurrent assessment being required.
- The IV fluids should be discontinued when the intravascular volume has been replenished.
- The IV fluids should be promptly discontinue when new or worsening pulmonary edema occurs.
- When the pulmonary edema worsens with a trial of IV fluids, these women should be regarded as having uncertain intravascular volume and may be better managed with vasoactive agents.

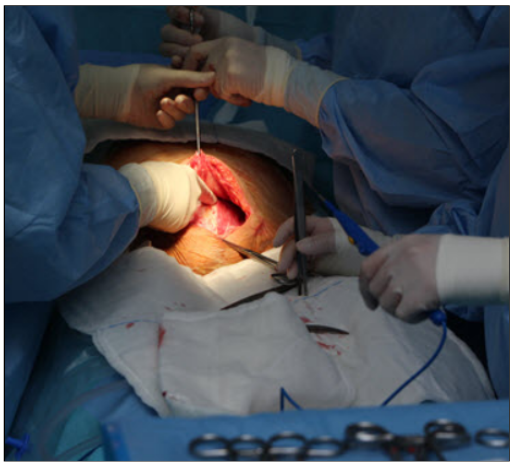
Some women with coagulopathy or bleeding due to disseminatd intravascular coagulation (DIC) may require blood product transfusion which may include the transfusion of recombinant factor VIIa.





- As discussed, AFES most commonly occurs during labor and delivery or postpartum.
- When AFES presents intrapartum, the need for immediate delivery must be determined.
- Delivery is determined mother-by-mother but factors favoring delivery include:
- Non-reassuring fetal heart race tracing
- Rapid and progressive deterioration of the mother's condition
- The opinion that delivery of the fetus may facilitate maternal resuscitative efforts
- When the cervix is fully dilated and the fetal head has descended to a station of at least +2/5, operative vaginal delivery is reasonable with a capable clinician.
- Otherwise, an emergency cesarean delivery is indicated.
- Consideration for peri-mortem cesarean may be warranted when maternal status is compromised

- Consideration for the peri-mortem cesarean is warranted when shock and cardioplumnoary resusicitative efforts fail to restore maternal circulation.
- Because brain damage begins at 5 minutes of anoxia, the procedure should be initiated at 4 minutes (the 4-minute rule) to deliver the healthiest fetus.
- If a mother has a resuscitatable cause of death, then her life may be saved as well by a prompt and timely cesarean delivery during CPR.
- Sadly, the clinicians are too often paralyzed by the horror of the maternal cardiac arrest and instinctively CPR is performed too long before turning to the peri-morten delivery.
- This quick procedure may actually improve the situation for the mother and certainly save the child's life [48].



- However, there is significant risk of major maternal morbidity or death when a cesarean is performed in the presence of coagulopathy.
- Some clinicians recommend that operative intervention not begin until the coagulopathy is corrected.
- Unfortunately, this is not always possible because the delay could lead to fetal death, further blood loss, and worsening of the coagulopathy.
- When a cesarean is performed, blood, fresh frozen plasma, platelets, cryoprecipitate should be available and administered if there is any evidence of impaired coagulation:
  - Persistent bleeding without clotting from the incision
  - Needle site bleeding

- Case reports have described novel interventions that have been successfully used in patients with AFES:
- A right ventricular device and inhaled nitric oxide have been used in patients with right ventricular failure and pulmonary hypertension [49,50].
- In patients with severe left ventricular failure and hypoxemia the following have been utilized: cardiopulmonary bypass, intraaortic balloon pump counterpulsation and extracorporeal membrane oxygenation (ECMO) [21,51].
- The use of recombinant human factor VIIa (rVIIa) may be utilized when severe coagulopathy and bleeding, especially in those undergoing surgery to control postpartum hemorrhage. [50,52].
- Unfortunately, rVIIa use in AFE may be associated with thrombotic morbidity and mortality (i.e. stroke and pulmonary embolism).

Maternal | Neonatal

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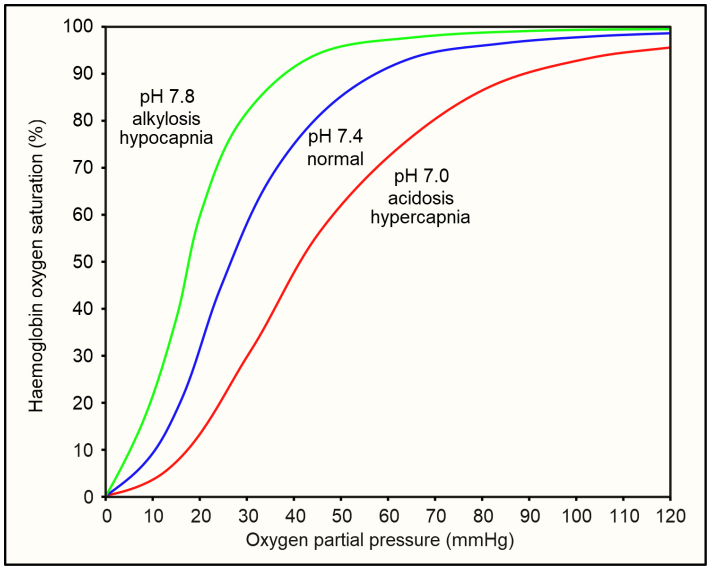

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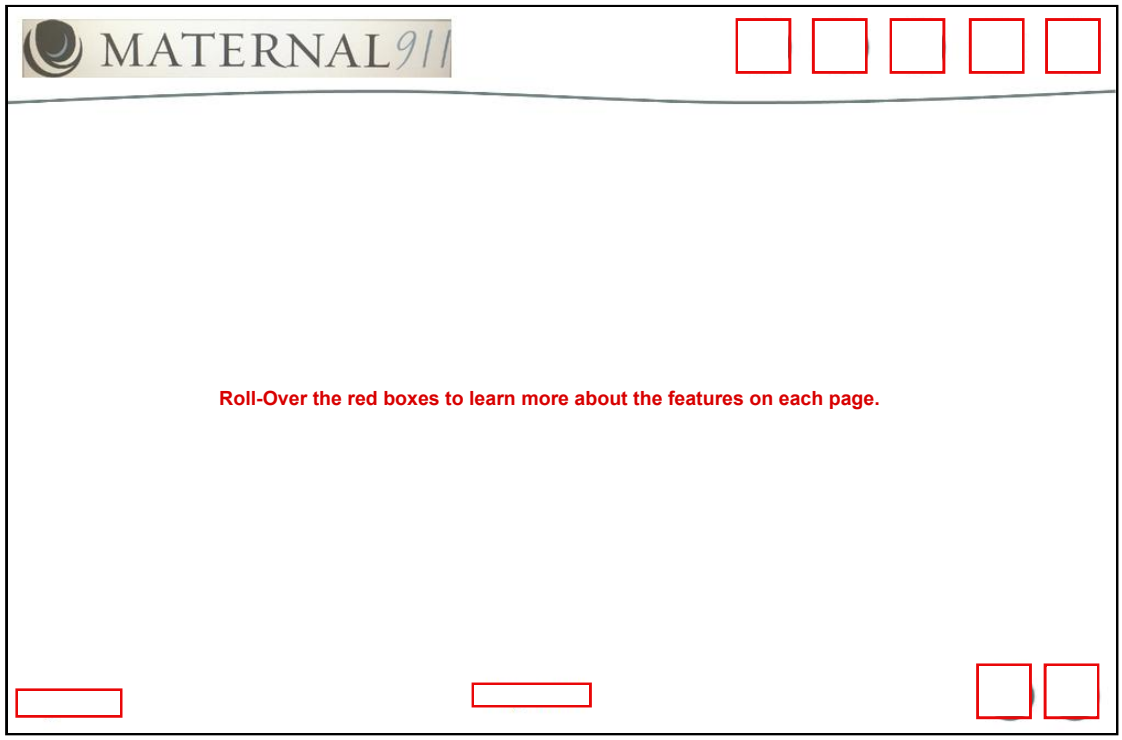

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Links

Title	Link