SMFM Clinical guidelines No. 9: Amniotic Fluid Embolism: Diagnosis and Management

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Amniotic Fluid Embolism: Diagnosis and Management

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Abstract

OBJECTIVE: We sought to provide evidence-based guidelines regarding the diagnosis and management of amniotic fluid embolism (AFE).

METHODS: A systematic literature review was performed using MEDLINE, PubMed, EMBASE, and the Cochrane Library. The search was restricted to English-language articles published from 1966 through March 2015. Priority was given to articles reporting original research, in particular randomized controlled trials, although review articles and commentaries were consulted. Abstracts of research presented at symposia and scientific conferences were not considered adequate for inclusion. Evidence reports and published guidelines were also reviewed, and additional studies were located by reviewing bibliographies of identified articles. Grade (Grading of Recommendations Assessment, Development, and Evaluation) methodology was employed for defining strength of recommendations and rating quality of evidence. Consistent with US Preventive Task Force guidelines, references were evaluated for quality based on the highest level of evidence.

RESULTS AND RECOMMENDATIONS:

We recommend the following: (1) we recommend consideration of AFE in the differential diagnosis of sudden cardiorespiratory collapse in the laboring or recently delivered woman. (GRADE 1C); (2) we do not recommend the use of any specific diagnostic laboratory test to either confirm or refute the diagnosis of AFE; at the present time, AFE remains a clinical diagnosis. (GRADE 1C); (3) we recommend the provision of immediate high quality cardiopulmonary resuscitation with standard BCLS and ACLS protocols in patients who develop cardiac arrest associated with AFE. (GRADE 1C); (4) we recommend that the involvement of a

multidisciplinary team including anesthesia, respiratory therapy, critical care, and maternal-fetal medicine should be involved in ongoing care of such women. (Best Practice); (5) following cardiac arrest with AFE, we recommend immediate delivery in the presence of a fetus ≥ 23 weeks of gestation. (GRADE 2C); (6) we recommend the provision of adequate oxygenation and ventilation and, when indicated by hemodynamic status, the use of vasopressors and inotropic agents in the initial management of AFE. Excessive fluid administration should be avoided. (GRADE 1C); and (7) since coagulopathy may follow cardiovascular collapse with AFE, we recommend early assessment of clotting status and early aggressive management of clinical bleeding with standard massive transfusion protocols. (GRADE 1C)

KEYWORDS: Amniotic fluid embolism, pregnancy, cardiorespiratory arrest

Introduction

Amniotic fluid embolism (AFE) is a rare but potentially lethal condition. Due to a lack of international consensus regarding diagnostic criteria, estimates of both incidence and mortality rates associated with AFE vary widely. [1-4] These issues have recently been reviewed in detail and are not the focus of this manuscript. [2,5] Rather we emphasize that despite its low incidence in the general population of pregnant women, both maternal and perinatal morbidity and mortality are significant with AFE, even in cases ideally managed. Due to the rarity of this condition, most physicians and institutions have limited experience with the management of AFE. The purpose of this document is to provide clinicians with information that may improve the ability to make a timely diagnosis and establish appropriate supportive treatment to patients suffering from AFE in order to improve maternal and perinatal outcomes.

What is AFE and what are its clinical features?

A detailed review of the pathophysiology of AFE is beyond the scope of this document, but may be found elsewhere and is summarized in figures 1 and 2. [1,2,5] It appears to involve a complex sequence of events triggered in certain women by entrance into the maternal circulation of material from the fetal compartment, resulting in abnormal activation of proinflammatory mediator systems similar to the systemic inflammatory response syndrome.

The typical presentation of AFE includes a triad of sudden hypoxia and hypotension, followed in many cases by coagulopathy, all occurring in relation to labor and delivery. The diagnosis of AFE is clinical, based upon the presence of these elements and the exclusion of other likely causes. AFE should be considered in the differential diagnosis in any pregnant or

immediately postpartum woman who suffers sudden cardiovascular collapse or cardiac arrest, seizures, severe respiratory difficulty or hypoxia, particularly if such events are followed by a coagulopathy that cannot be otherwise explained. The analysis of the national registry reveals that 70% of cases of AFE occur during labor, 11% after a vaginal delivery, and 19% during a cesarean delivery.[1] These figures suggest that mode of delivery may alter the timing of AFE but not its occurrence. Rarely, AFE may occur during the first or second trimesters of pregnancy at the time of pregnancy termination or amniocentesis.[6]

The clinical presentation of AFE is, in its classic form, dramatic. A period of anxiety, change in mental status, agitation, and a sensation of "doom" may precede the event.[7] Patients may progress rapidly to cardiac arrest, with pulseless electrical activity (PEA), asystole, ventricular fibrillation, or pulseless ventricular tachycardia. In cases occurring prior to delivery, electronic fetal monitoring will demonstrate decelerations, loss of variability, and terminal bradycardia as oxygenated blood is shunted away from the uterus, and catecholamine-induced uterine hypertonus causes a further decline in uterine perfusion.[1-2]

DIC is present in up to 83% of cases.[1] The coagulopathy of AFE may occur in conjunction with the cardiopulmonary manifestations, be manifest only after initial cardiopulmonary resuscitation has been completed, or in very rare cases may be the only finding in women without cardiorespiratory compromise.[8-10] DIC is commonly manifested by hemorrhagic complications including bleeding from venipunctures or surgical sites, hematuria, gastrointestinal hemorrhage, and vaginal bleeding. As with any condition involving diminished uterine perfusion, co-existence with uterine atony is not uncommon. However, bleeding from

incompletely controlled atony followed by hypovolemic shock and either a consumptive or dilutional coagulopathy cannot be attributed to AFE, nor does AFE occur as a mild coagulopathy followed hours later by sudden cardiovascular collapse in the absence of interval hemorrhage and hypovolemia.

Reported risk factors for AFE include situations in which the exchange of fluids between the maternal and fetal compartments is more likely, such as operative delivery (cesarean or vaginal), placenta previa, placenta accreta and abruption. An association between induction of labor and AFE is inconsistently reported. Abnormalities of uterine tone (hypo- or hypertonous) described commonly in cases of AFE may be the consequence of uterine hypoperfusion secondary to profound maternal shock and hypoxia with massive catecholamine release, rather than the cause.[1] Other putative risk factors include cervical lacerations, uterine rupture, eclampsia, polyhydramnios, and multiple gestations; as outlined above, a tendency to overdiagnose AFE in cases actually involving other causes of primary hemorrhage may contribute to these reports. Sociodemographic risk factors such as maternal age and race/ethnicity, are also reported in some series.[11-16] However, given the rare and unpredictable nature of AFE, there are no risk factors sufficiently established to justify any alteration in standard obstetric care.

How should you manage a patient with sudden cardiac arrest in whom AFE is suspected?

AFE should be considered in the differential diagnosis of sudden cardiorespiratory compromise in any pregnant or recently post-partum patient. (GRADE 1C) Initial

resuscitation of cardiac arrest does not require a specific diagnosis of AFE, as initial maternal treatment (with BCLS and ACLS protocols) is similar regardless of the exact etiology. **We do not recommend the use of any specific diagnostic laboratory test to either confirm or refute the diagnosis of AFE; at the present time, AFE remains a clinical diagnosis. (GRADE 1C) We recommend the provision of immediate high quality cardiopulmonary resuscitation with standard BCLS and ACLS protocols in patients who develop cardiac arrest associated with AFE. (GRADE 1C) We recommend that the involvement of a multidisciplinary team including anesthesia, respiratory therapy, critical care, and maternal-fetal medicine should be involved in ongoing care of such women. (Best Practice)** The most critical immediate action is to start chest compressions before rescue breathing is administered.[17]

Chest compressions should be performed similarly to non-pregnant individuals. The hands of the provider should be placed in the lower half of the sternum.[17] Chest compressions should be performed "hard and fast", achieving a depth of at least 2 inches and allowing complete chest recoil between compressions. Patients who are undelivered should be tilted to the left lateral decubitus position, or preferably have the uterus displaced laterally by an assistant to prevent aorto-caval compression by the gravid uterus.[17] The use of vasopressors, anti-arrhythmic agents, and defibrillating doses is not different than those utilized in non- pregnant individuals. Although concerns that electric arcing may occur if fetal monitors are in place at the time of cardioversion or defibrillation are largely theoretical, it is reasonable to remove such monitors while CPR is in progress. However, the presence of such monitors should not delay defibrillation when indicated. [17] The components of high quality cardiopulmonary resuscitation are summarized in Table 1.

If the patient is undelivered at the time of cardiac arrest, expeditious delivery is indicated if the fetus has reached an age of potential viability (≥ 23 weeks.) Not only may this be life saving for the fetus, but in theory may assist in maternal resuscitation by removing venacaval compression. An operative vaginal delivery (forceps or vacuum assisted) should be performed in laboring patients in whom obstetrical conditions support such an intervention. If a vaginal delivery is not an option, emergency cesarean delivery is generally indicated. Classically, the indication for a perimortem cesarean delivery has been failure to obtain spontaneous circulation after 4 minutes of CPR to reduce the profound fetal hypoxia occurring during maternal cardiac arrest. [17] This timeframe is ideal, but is rarely achievable when cardiac arrest is unexpected. We recommend that preparations for emergent perimortem cesarean be initiated simultaneously with initiation of CPR – if the cardiac arrest is still ongoing as the instruments become available, proceed with cesarean delivery. The dismal prognosis of adult cardiac arrest not amenable to, or unresponsive to immediate DC countershock suggests that maternal prognosis will not be significantly compromised by such an operation. Some authors recommend moving this threshold to 20 weeks in order to improve maternal perfusion – no evidence exists however that such pre-viable cesarean improves the outcome in cases of AFE related maternal cardiac arrest.[17] **Following cardiac arrest with AFE, we recommend immediate delivery in the presence of a fetus ≥ 23 weeks of gestation. (GRADE 2C)** In cases of maternal hemodynamic instability which does not involve one of the lethal dysrhythmias, cases must be individualized based on fetal age and degree of compromise, maternal condition and the availability of anesthetic support. No data exists to guide delivery decisions under these circumstances.

The literature contains innumerable case reports in which various novel therapeutic modalities have been used in women with presumptive AFE, and the patient did not die.[7] Unfortunately, evidence of a causal, as opposed to an anecdotal connection between most of these and survival from AFE is lacking. We focus here only upon the better supported ancillary treatment options.

The use of veno-arterial extracorporeal membrane oxygenation (ECMO) has been described in cases of AFE refractory to conventional resuscitation maneuvers.[7] However, the use of anticoagulation during ECMO may worsen bleeding in the profoundly coagulopathic patient with active hemorrhage. Because of these concerns, as well as lack of adequate evidence of benefit, ECMO is controversial and not routinely recommended in the management of AFE.

After successful resuscitation, post cardiac arrest management is of paramount importance.[18] Hemodynamic instability is common and patients may require fluids, vasopressors, and inotropes. The goal is to maintain a mean arterial blood pressure of 65 mmHg.[18] Fever may worsen ischemia-reperfusion injury to the brain and should be aggressively treated. Hyperoxia will also worsen ischemia-reperfusion injury, and administration of 100% oxygen after survival of cardiac arrest should be avoided. The inspired fraction of oxygen should be weaned to maintain a pulse oxymetry value of 94%-98%.[18] Serum glucose should be maintained between 140-180 mg/dL with the use of insulin intravenous infusions if needed.[19]

Mild therapeutic hypothermia (TH), defined as cooling the patient to a temperature between 32 and 34 °C for 12-24 hours, has been recommended by the American Heart

Association to increase the rate of a favorable neurologic outcome and reduce mortality.[18] A recent study, however, found no differences in outcomes between targeted temperatures of 33°C versus 36 °C among survivors of cardiac arrest who were treated with mild therapeutic hypothermia.[20] Current guidelines recommend targeted temperature management of cardiac arrest victims aiming at temperatures between 32-36°C.[18] The data on TH during pregnancy is limited to case reports.[21,22] Most survivors of AFE will have been delivered during the course of successful resuscitation. The main concern limiting use of TH in these patients is concern that this may increase the risk of hemorrhage. In patients not demonstrating significant DIC and bleeding, TH should be considered. Targeting a temperature of 36°C (as opposed to lower temperatures with a concomitant increased risk of hemorrhage) is an option. Such decisions must be made in conjunction with the available medical critical care team.

What are the considerations for hemodynamic support in a patient with AFE?

Immediate survivors of AFE require multidisciplinary management, including maternal-fetal medicine subspecialists and intensive care specialists. The management of suspected AFE is supportive and focuses on rapid maternal hemodynamic stabilization.

The initial phase of AFE consists mainly of right ventricular failure. If available, transthoracic and/or transesophageal echocardiography may provide valuable information. Immediately after presentation, the echocardiography will commonly reveal a severely dilated hypokinetic right ventricle (acute cor pulmonale) with deviation of the inter-ventricular septum into the left ventricle. Hypoxia, acidosis, and hypercapnia should be avoided as they increase

pulmonary vascular resistance and lead to further right heart failure.[13] Right ventricular output may be improved by using inotropes such as dobutamine and milrinone. These agents also will lead to pulmonary vasodilation. Other specific interventions aimed at decreasing the pulmonary vascular resistance include sildenafil, inhaled or intravenous prostacyclin, and inhaled nitric oxide. Hypotension in this phase should be mainly treated with vasopressors such as norepinephrine or vasopressin.[23] The commonly used doses of these agents are provided in Table 2. **We recommend the provision of adequate oxygenation and ventilation and, when indicated by hemodynamic status, the use of vasopressors and inotropic agents in the initial management of AFE. Excessive fluid administration should be avoided. (GRADE 1C)** After an initial phase of right ventricular failure, left ventricular failure predominates. Excess fluid administration should be especially avoided in the setting of a massively dilated right ventricle, as this will increase over-distention of the ventricle and increase the risk of a right-sided myocardial infarction. Increased distention of the right ventricle will also displace the inter-ventricular septum to the left, further compromising the cardiac output due to left ventricular obliteration.

Minutes to hours following the initial presentation, the right ventricular function usually improves and left ventricular failure with cardiogenic pulmonary edema becomes the prominent finding.[7] In patients who are not intubated, non-invasive mechanical ventilation or endotracheal intubation should be considered. Left sided heart failure should be treated by optimizing cardiac preload, the use of vasopressors in cases of hypotension in order to maintain coronary perfusion pressure, and inotropes (dobutamine or milrinone) to increase left ventricular contractility.

Severe pulmonary congestion not responsive to diuretic therapy may require fluid removal through dialysis.

Later in the clinical course, some patients with persistent severe inflammation and prolonged care in the intensive care unit may develop nosocomial infections and distributive shock with non-cardiogenic pulmonary edema from severe sepsis.[24] No evidence exists to justify the routine use of steroids in cases of AFE. The overall management of AFE is summarized in Figure #2.

How is the coagulopathy associated with amniotic fluid embolism managed?

DIC is present in most cases of AFE. The onset is variable; DIC may be manifest either immediately following cardiovascular collapse, or in the later phases of the syndrome. Severe hemorrhage may require simultaneous medical and surgical approaches. Medical management classically includes administration of blood products to maintain a platelet count above 50,000/mm^3 and normal (or close to normal) activated partial thromboplastin time (aPTT) and international normalized ratio (INR). In the setting of massive hemorrhage, blood product administration should not be delayed while awaiting the results of laboratory tests. Instead, early aggressive resuscitation with packed red blood cells, fresh-frozen plasma, and platelets at a ratio of 1:1:1 (hemostatic resuscitation) results in improved outcomes.[25] **Since coagulopathy may follow cardiovascular collapse with AFE, we recommend early assessment of clotting status and early aggressive management of clinical bleeding with standard massive transfusion protocols. (GRADE 1C)**

Administration of recombinant activated factor VII has been described in cases of AFE.[26-28] However, some authors believe that this treatment, in patients with DIC and elevated levels of tissue factor (as occurs in AFE), could lead to excessive diffuse thrombosis and multi-organ failure. The use of this agent may be considered as a last resort in cases where hemorrhage cannot be stopped with massive blood component replacement and surgical interventions.[27]

Both plasminogen activators and plasminogen activator inhibitors have been identified in amniotic fluid.[29] The presence of hyperfibrinolysis has been described in AFE related coagulopathy and should be considered in the management of AFE. When available, bedside thromboelastography may aid in identifying bleeding patients who might benefit from the use of anti-fibrinolytics such as tranexamic acid or epsilon aminocaproic acid.[30]

Uterine atony is common with AFE and should be managed aggressively.[31] The use of uterotonics such as oxytocin, ergot derivatives, and prostaglandins is appropriate when indicated. Refractory cases may require uterine tamponade with the use of packing or commercially available intra-uterine balloons. Extreme cases may need bilateral uterine artery ligation, B-Lynch stitch, or hysterectomy. We caution however against making the diagnosis of AFE based exclusively on hemorrhage from persistent atony with secondary coagulopathy – in our experience, this is a common diagnostic error.

In patients delivered vaginally, a thorough inspection of the cervix and vagina is warranted to rule out lacerations as the cause, or as a contributing factor to profuse bleeding in a patient with DIC. In patients with diffuse bleeding after or during a cesarean delivery that is not amenable to

surgical control, consideration should be given to packing the pelvis and transfer to the intensive care unit for further medical therapy with delayed closure.

What other differential diagnoses should be considered when amniotic fluid embolism is suspected?

In the absence of the classic triad of hypotension, hypoxia and subsequent coagulopathy, AFE often remains a diagnosis of exclusion. The list of conditions that may result in either acute cardiac or respiratory or hematologic failure in pregnancy is relatively long, and includes myocardial infarction, pulmonary embolism, air embolism, anesthetic complications, anaphylaxis, and eclampsia and in some cases, sepsis. Providers caring for pregnant women with an acute clinical event and cardiorespiratory failure should narrow this list to clinically relevant diagnoses that require specific treatment strategies. Importantly, an exact diagnosis is not required to start treatment for presumed AFE since immediate interventions, such as good quality cardiorespiratory resuscitation, are supportive in nature.

Risk factors for myocardial infarction, such as advanced maternal age, diabetes, chronic hypertension, smoking, obesity, dyslipidemia, and previous history of coronary artery disease may suggest this diagnosis. Cardiac troponins and a 12 lead electrocardiograph should be obtained as soon as possible. A bedside echocardiography may be useful in making a diagnosis of cardiogenic shock secondary to myocardial ischemia. Echocardiography will also aid in ruling out conditions such as a peripartum dilated cardiomyopathy.[32]

Pulmonary embolism is a recognized complication of pregnancy. Computed tomography angiography or a ventilation perfusion scan may be useful in evaluating this potential diagnosis. In cases complicated by profuse bleeding, however, thromboembolism is unlikely.

A high spinal anesthesia can result in apnea but is unlikely to cause a dramatic drop in cardiac output or hemorrhagic manifestations. Inadvertent intravascular injection of local anesthetics may cause seizures and cardiovascular collapse.[33] Timing between injection and onset of symptoms may suggest this diagnosis or make it less likely. If local anesthetic toxicity is likely, consideration should be given to the use of intravenous lipids (20% Intralipid) in addition to other supportive measures.[34]

Air embolism may also cause acute cardiorespiratory compromise. If venous air embolism is high on the list of potential diagnoses, normobaric 100% oxygen should be used. The patient should be turned to the left lateral decubitus to prevent air from migrating to the pulmonary artery. If a central line is in place, then aspiration of blood may be performed in an attempt to aspirate bubbles of air. If an arterial air embolism is suspected (e.g if neurologic symptoms are present), hyperbaric oxygen therapy should be considered, if available.

Eclampsia is obviously a possibility in a patient in the latter half of pregnancy with new onset seizures, although eclampsia is not commonly associated with cardiorespiratory arrest and acute profound coagulopathy. Transfusion reactions may cause acute pulmonary edema (transfusion related acute lung injury) and coagulopathy when incompatible blood is administered. This is an uncommon event in modern practice.

Anaphylactic shock is a possibility, particularly in the setting of urticarial rash, and laryngospasm or bronchospasm immediately following the administration of medication known to cause anaphylaxis. Bronchospasm has been reported in about 15% of cases of AFE. However, anaphylaxis is not usually accompanied by coagulopathy and cardiac dysfunction is not commonly severe, as hypotension associated with anaphylaxis is due primarily to vasodilation and increased vascular permeability. If anaphylaxis is suspected, treatment with epinephrine, steroids, and inhaled bronchodilators is indicated.

Bedside echocardiography demonstrating right ventricular dysfunction favors the diagnosis of AFE over anaphylaxis and most of the other conditions that may mimic AFE.

What is the prognosis and recurrence risk for patients who survive an episode of amniotic fluid embolism?

The recurrence rate of AFE is difficult to define due to the rarity of the condition and high mortality rate. Multiple cases of uneventful subsequent pregnancies, and no cases of recurrence have been reported.[35,36] Patients should be cautioned however that the available sample size precludes definitive conclusions regarding recurrence risk.

Conclusions

AFE is a rare but often lethal condition. Maternal and perinatal mortalities appear to have decreased during the last decades likely due to improvements in delivery of critical care,

recognition of atypical or milder cases with no cardiorespiratory collapse and the likely inclusion of patients with conditions other than AFE, particularly in series based upon administrative data. The diagnosis remains clinical and is often one of exclusion, as no single specific diagnostic test is currently available. Treatment is mainly supportive and involves delivery of the fetus when indicated, respiratory support (usually in the form of endotracheal intubation and mechanical ventilation), and hemodynamic support with judicious use of fluids, vasopressors, inotropes, and pulmonary vasodilators. Rapid initiation of treatment, aided by a high index of clinical suspicion, is essential. The recurrence rate of AFE is unknown but appears to be low. Much of the published literature regarding AFE is of poor quality and likely includes a significant number of patients with other conditions. Uniform diagnostic criteria for AFE cases reported in research publications are badly needed, and may accelerate our understanding of this condition.

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374 **Summary of Recommendations**

	Recommendations	**GRADE**
1	We recommend consideration of AFE in the differential diagnosis of sudden cardiorespiratory collapse in the laboring or recently delivered woman.	1C Strong recommendation Weak quality evidence
2	We do not recommend the use of any specific diagnostic laboratory test to either confirm or refute the diagnosis of AFE; at the present time, AFE remains a clinical diagnosis.	1C Strong recommendation Weak quality evidence
3	We recommend the provision of immediate high quality cardiopulmonary resuscitation with standard BCLS and ACLS protocols in patients who develop cardiac arrest associated with AFE.	1C Strong recommendation Weak quality evidence
4.	We recommend that the involvement of a multidisciplinary team including anesthesia, respiratory therapy, critical care, and maternal-fetal medicine should be involved in ongoing care of such women.	Best Practice
5.	Following cardiac arrest with AFE, we recommend immediate delivery in the presence of a fetus $\geq$ 23 weeks of gestation.	2C Weak recommendation Weak quality evidence
6.	We recommend the provision of adequate oxygenation and ventilation and, when indicated by hemodynamic status, the use of vasopressors and inotropic agents in the initial management of	1 C Strong recommendation, Weak quality evidence

	AFE. Excessive fluid administration should be avoided.	
7.	Since coagulopathy may follow cardiovascular collapse with AFE, we recommend early assessment of clotting status and early aggressive management of clinical bleeding with standard massive transfusion protocols.	1C Strong recommendation, Weak quality evidence

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476 Table 1

477 Components of high quality cardiopulmonary resuscitation in pregnancy

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Rapid chest compressions (100 x minute)
Perform hard compressions, achieving a depth of at least 2 inches
Assure adequate chest recoil between compressions
Minimize interruptions of chest compressions
Avoid prolonged pulse checks (no more than 5-10 seconds)
Resume chest compressions immediately after defibrillating
Switch provider of compressions every 2 minutes to avoid fatigue
Lateral displacement of uterus during resuscitation

479 Reference: Jeejeebhoy FM, Zelop CM, Lipman S, Carvalho B, Joglar J, Mhyre JM, et al. Cardiac
480 arrest in pregnancy: A scientific statement from the American Heart Association.
481 Circulation 2015;132:1747-1773.
482
483

484 **Table 2.** Recommended doses for agents commonly used in cases of acute right ventricular
485 failure.
486

Agent	Dose
Sildenafil	20 mg tid PO or through nasogastric/orogastric tube.
Dobutamine	2.5-5.0 micrograms/kg/minute. Higher doses may compromise right ventricular filling time due to tachycardia.
Milrinone	0.25-0.75 micrograms/kg/minute. Most common side effect is systemic hypotension.
Inhaled nitric oxide	5-40 ppm. Follow methemoglobin levels every 6 hours, avoid abrupt discontinuation.
Inhaled prostacyclin	10-50 nanograms/kg/minute.
Intravenous prostacyclin	Start at 1-2 nanograms/kg/minute through a central line and titrate to desired effect. Side effects include systemic hypotension, nausea, vomiting, headache, jaw pain, and diarrhea.
Norepinephrine	0.05-3.3 micrograms/kg/minute

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Figure 1. Proposed pathophysiology of amniotic fluid embolism

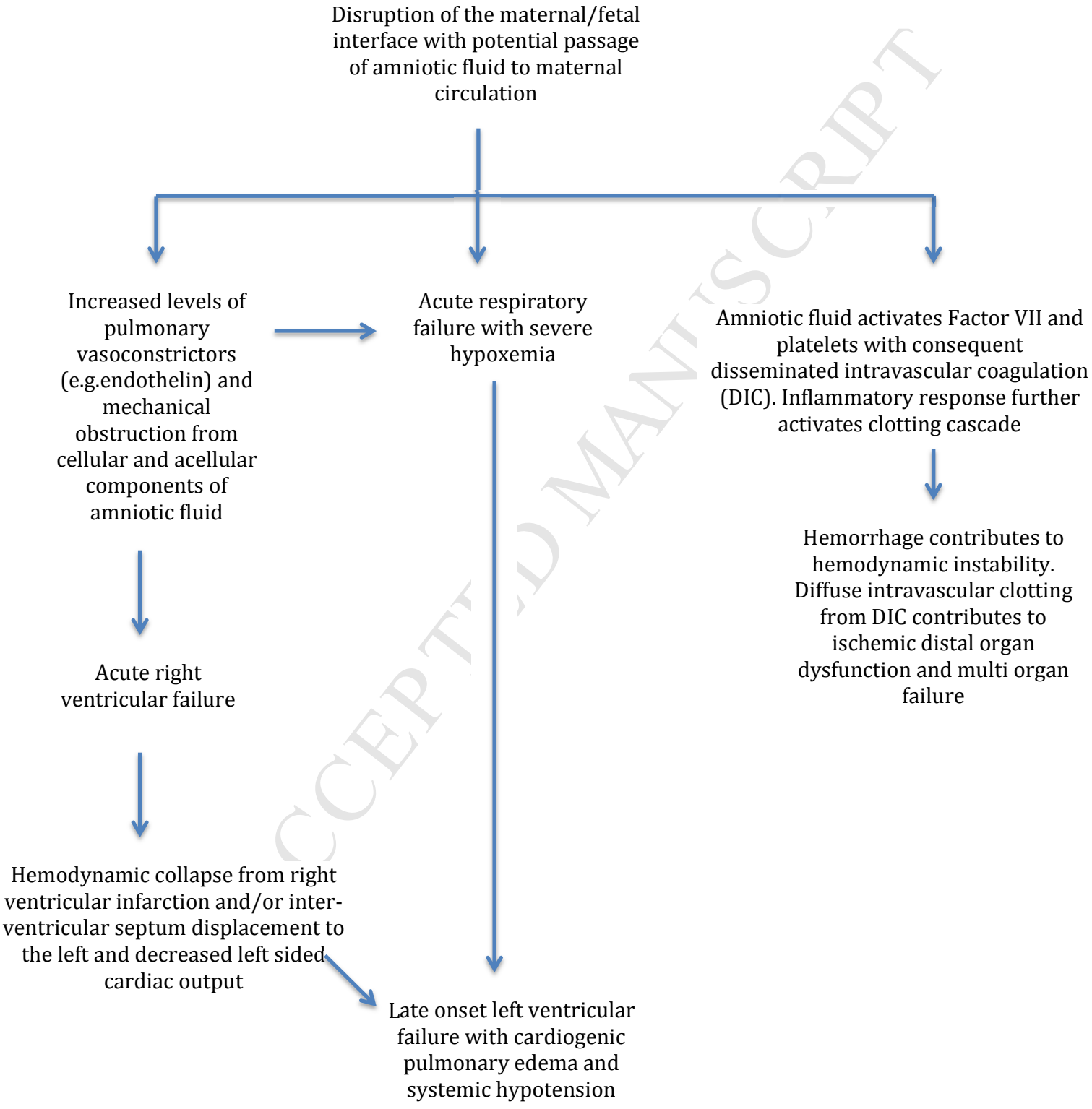
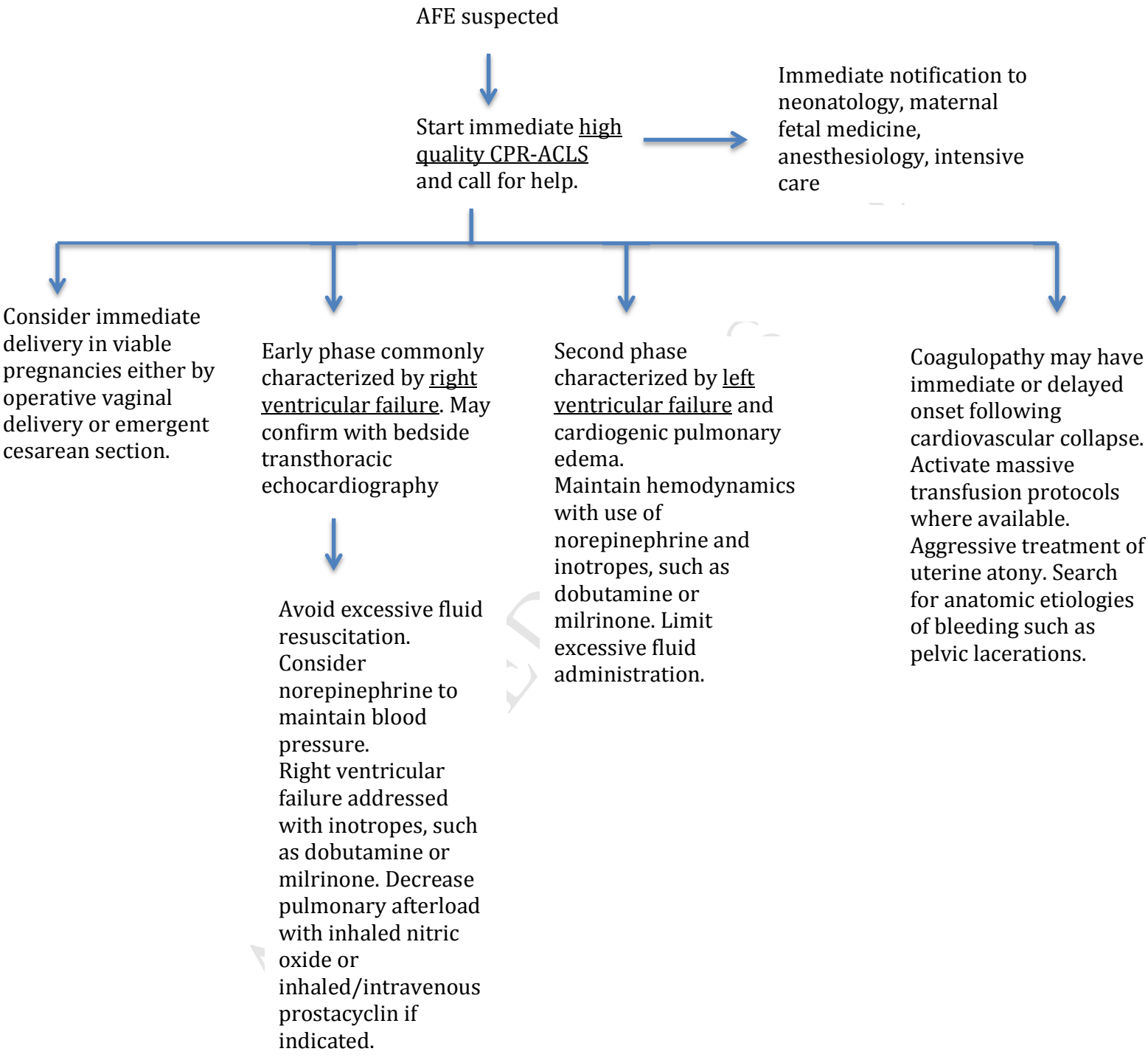


Figure 2. Immediate supportive treatment in suspected cases of amniotic fluid embolism.



CPR: Cardiopulmonary resuscitation; ACLS: Advanced cardiac life support; AFE: Amniotic fluid embolism.