Resuscitating the Crashing Pregnant Patient

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KEYWORDS

• Critical care • Pregnancy • Obstetrics • Maternal resuscitation • Respiratory failure • Shock

KEY POINTS

- The physiologic changes of pregnancy can mask severe illness and early shock.
- Early specialist consultation is crucial. If needed, transfer for obstetric or pediatric specialist care should be initiated immediately or as soon as the patient is stabilized.
- The pregnant patient has an inherently difficult airway and desaturates quickly because of decreased pulmonary reserve and increased oxygen consumption.
- Most medications used in standard resuscitation can be used in pregnancy.
- Remember the 5-minute rule for perimortem cesarean section and be prepared to deliver quickly in the event of a maternal cardiac arrest.

INTRODUCTION

Critical illness in pregnancy is rare[1] and may be attributed either to an independent cause of illness that is complicated by pregnancy or to a cause associated with the pregnancy itself (**Table 1**). Caring for an unstable obstetric patient is a stressful clinical situation for the emergency physician, who must consider the welfare of two patients because the fetus is also at risk. An understanding of the physiologic changes associated with pregnancy and a solid knowledge base for treating the critically ill pregnant patient can improve care in this high-risk population.

PHYSIOLOGIC CHANGES OF PREGNANCY

Pregnancy is a dynamic state. The placenta not only connects the maternal and fetal circulation but is itself an endocrine organ, and the hormones of pregnancy set the

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Table 1
Most commonly identified causes of pregnancy-related morbidity and mortality in the United States

Etiology	Percentage
Cardiovascular conditions	15.7
Sepsis/infection	12.5
Hemorrhage	11
Cardiomyopathy	11
Pulmonary embolism	9
Cerebrovascular accident	7.7
Preeclampsia/eclampsia	6.9
Amniotic fluid embolism	5.6
Other noncardiovascular causes	13.9
Unknown	6.4

Data from [CDC Pregnancy Mortality Surveillance System. Causes of pregnancy-related death in the United States: 2011-2016. 2019. Available at: https://www.cdc.gov/reproductivehealth/maternal-mortality/pregnancy-mortality-surveillancesystem.htm/www.cdc.gov/reproductivehealth/maternalinfanthealth/pregnancy-mortality-surveilance-system.htm#causes. Accessed December 1, 2019].

stage for the numerous physical changes undergone by the pregnant patient (**Table 2**). The gestational age of the fetus aids understanding of expected physiologic changes and is estimated using point-of-care ultrasound (POCUS) if unknown.

UTEROPLACENTAL PHYSIOLOGY

The two maternal uterine arteries provide approximately 700 mL/min of uteroplacental blood flow. The placental vessels have no autoregulatory capability and are maximally dilated because of circulating progesterone and nitric oxide, leaving maternal blood pressure as the primary factor in uteroplacental blood flow. Maternal hypotension, uterine contractions, alkalemia, and vasoconstricting agents (vasopressors, nicotine, cocaine) decrease uteroplacental blood flow.[8]

Transplacental gas exchange is dependent on a maternal-fetal concentration gradient; maternal respiratory alkalosis facilitates diffusion of fetal CO_2 into maternal circulation for removal. It also increases maternal hemoglobin oxygen saturation, whereas fetal hemoglobin's stronger affinity for oxygen, in comparison with maternal hemoglobin, enables oxygen transfer to the fetus.[8,9]

In the setting of acidemia the hemoglobin-oxygenation curve shifts to the right, decreasing hemoglobin-oxygen binding and decreasing oxygenation of distal tissues. In fetal hemoglobin, which already exists in a hypoxemic environment, lack of sufficient oxygen delivery quickly proceeds to anaerobic metabolism and metabolic acidosis, which in turn can lead to bradycardia and poor fetal perfusion.[9]

INITIAL STEPS

Identify Illness

Because the physiologic changes of pregnancy can mask illness and early shock, it is important to assess clinical signs of end-organ perfusion, such as mental status, capillary refill, and urine output. Physicians should notice subtle changes in vital signs and maintain a lower threshold for aggressive care, because these patients are at risk for

Table 2
Physiologic changes of pregnancy

System	Change	Clinical Relevance
Cardiovascular	↓ Systemic vascular resistance ↑ Heart rate (15–20 bpm above baseline) ↑ Cardiac output (by 30%–50%) Aortocaval compression by the uterus (≈20 wk)	↓ Blood pressure (nadirs at 24–28 wk) Supine hypotension syndrome
Respiratory	↑ Mucosal vascularity and edema Upward shift of diaphragm, decreased FRC ↑ Respiratory rate ↑ Tidal volume (caused by widening of chest wall)	Difficult airway (narrower, distorted, friable) ↓ Functional reserve and rapid desaturations Physiologic respiratory alkalosis
Renal	↑ Renin-angiotensin-aldosterone system activity ↑ Renal blood flow and glomerular filtration rate ↑ Bicarbonate secretion	↑ Total body water and circulating volume (1.5–2L) ↓ "Normal" creatinine, ↑ drug clearance Relatively normal pH
Hematologic	↑ Erythropoiesis <↑ total plasma volume ↑ White blood cell count ↑ Circulating coagulation factors ↓ Fibrinolysis	Physiologic relative anemia Hypercoagulable state
Gastrointestinal	↓ Gastric emptying/ gastrointestinal motility ↓ Lower esophageal sphincter tone	Increased risk of vomiting and aspiration

FRC, functional residual capacity. *Data from* Refs.[2–7]

rapid decompensation and have narrower targets for optimal oxygenation and ventilation.

Call Consultants

As soon as severe maternal illness is recognized it is crucial to involve the obstetric team, particularly if the gestational age of the fetus has reached the age of viability, typically considered to be 24 weeks. It is similarly prudent to notify the neonatal intensive care team so that they can prepare for a precipitous delivery. In cases where the mother has comorbid chronic disease or a high-risk pregnancy, or there is fetal distress, emergent delivery may be required. These considerations are indications for urgent transfer, once the patient is stabilized, if there is no specialized obstetric or neonatal care available in-house.

Immediate Resuscitation

A sick pregnant patient with abnormal vital signs requires at least two large-bore intravenous (IV) lines. If IV access cannot be promptly achieved, intraosseous access should be pursued. Around 20 weeks of gestation and beyond, the gravid uterus can compress the inferior vena cava when the patient is lying supine, therefore IV, intraosseous, and if required, central lines, should be placed above the diaphragm to avoid a delay in fluids, blood products, and medications reaching central circulation.[10]

Similarly, supine positioning should be avoided in any patient where aortocaval compression and subsequent decrease in venous return, cardiac output, and blood pressure by an enlarged uterus is a reasonable concern. In the setting of maternal hypotension, immediately displace the uterus by moving the patient into the left lateral decubitus position or providing at least 15° of leftward tilt.[11–13] If the patient is unable to be repositioned, the uterus should be displaced leftward manually.

Any obstetric patient with hypoxia should be placed immediately on supplemental oxygen to maintain a goal oxygen saturation of greater than 95% while steps are taken to provide the patient with the additional respiratory support needed.

Specific Considerations for the Fetus

Continuous fetal heart rate and tocometry monitoring should be performed in all pregnancies beyond 24 weeks, with consideration given to continuous monitoring in pregnancy beyond 20 weeks. If cardiotocometry monitoring is unavailable, POCUS should be used for regular evaluation of fetal heart rate.

In preterm deliveries the administration of antenatal corticosteroids to the mother is strongly associated with decreased fetal morbidity and mortality.[14] The American College of Obstetricians and Gynecologists recommends administration of either dexamethasone 6 mg or betamethasone 12 mg to all pregnant women between 24 and 34 weeks of gestation who are at substantial risk of preterm delivery within the next 7 days. Administration in the previable period, between 23 and 24 weeks, can be considered.[14]

MANAGEMENT OF CRITICAL ILLNESS

Acute Respiratory Failure

The pregnant patient with respiratory illness is at risk of rapid decompensation and hypoxemia caused by increased oxygen use and decreased pulmonary reserve. Intubation of the pregnant patient presents a significant risk because of the physiologic changes of pregnancy (see **Table 2**) and should be performed in the most controlled setting possible. To this end, it is prudent to have a low threshold to intubate the pregnant patient with respiratory distress, because a crash intubation is less than ideal and even slight maternal hypoxia puts the fetus at risk.[5,9] Still, there remains a role for noninvasive ventilation in pregnant patients who may not yet require intubation. Although evidence regarding the use of noninvasive ventilation modalities in pregnancy is limited, a few case reports and data on its use in nonpregnant patients indicate that high-flow nasal cannula is a suitable option for obstetric patients with mild to moderate respiratory distress who do not require significant ventilatory support.[15–17] Although noninvasive positive pressure ventilation has been historically avoided in pregnant patients because of concerns of aspiration, there are several case reports of successful use and its initiation is reasonable in alert pregnant patients who are protecting their airway.[18] As in nonobstetric patients, it is important to monitor the patient's status because delays to intubation when needed are associated with worsened outcomes.

When preparing for intubation, ensuring appropriate patient positioning and peri-intubation oxygenation is key. To decrease the chances of desaturation, preoxygenate and provide apneic oxygenation via nasal cannula,[19,20] and keep the patient's gravid uterus off of the diaphragm by maintaining them in reverse Trendelenburg or positioned with the head of bed slightly elevated.[19,20] Limit ventilation with a bag-valve mask if possible, to prevent overdistention of the stomach and increased chance of aspiration.

The airway of a pregnant patient is more likely to be difficult, with narrowing and landmark distortion caused by edema and a high risk of bleeding caused by increased vascularity and friability.[2] The most experienced emergency clinician should intubate, use a smaller endotracheal tube to improve chances of first-pass success, and should have clear backup plans understood by the entire team to reduce the risk of complications. Nasotracheal intubation is not recommended unless absolutely necessary because of likelihood of epistaxis and secondary obscuration of the vocal cords.

Ventilator settings

As in the nonpregnant population, the maintenance of lung-protective ventilator settings including a plateau pressure less than 30 mm Hg, higher positive end-expiratory pressure strategies to maintain a driving pressure less than 15 mm Hg, and avoidance of unnecessary levels of fraction of inspired oxygen (F_{IO_2}) remains key.[21–24]

The oxygenation goals in pregnancy are higher than in the nonpregnant population. Emergency clinicians should target a goal maternal Pa_{O_2} of greater than 70 mm Hg, corresponding to a saturation of greater than 95%, because levels less than 60 mm Hg are associated with fetal hypoxemia.[25] As with obese patients, a slightly higher positive end-expiratory pressure may be needed to account for upward pressure on the diaphragm and increased atelectasis.

As opposed to the permissive hypercapnia widely accepted in the general population,[26] the goal in pregnancy is an average Pa_{CO_2} of 30 mm Hg (**Table 3**).[4,5,20] This lower Pa_{CO_2} level preserves the maternal-fetal CO_2 gradient and the appropriate level of hemoglobin-oxygen affinity.[9,20] Although lung-protective tidal volumes of 6 to 8 mL/kg of ideal body weight have become standard in ventilated patients, in pregnant patients not suffering from acute respiratory distress syndrome (ARDS), relative liberalization of tidal volumes may be reasonable as long as not at the expense of higher plateau pressures.[21] Use of sedation and even paralytics to achieve ventilator synchrony may be required in patients with severe hypoxia, hypercarbia, bronchospasm, or ARDS.[21,22]

In the intubated obstetric patient with a persistent Pa_{O_2} less than 70 mm Hg or Pa_{O_2}:F_{IO_2} ratio less than 150 despite appropriate settings and therapy, early referral should be made for potential venovenous extracorporeal membrane oxygenation (ECMO).[27] Of note, proning has successfully been performed in pregnant patients with ARDS,[28] and may be worth initiating in severe refractory hypoxemia if there is a delay to intensive care unit or outside hospital transfer.

Table 3
Arterial blood gas changes in pregnancy

Arterial Blood Gas Measurement	Nonpregnant Mean ± SD	Pregnant Mean ± SD
pH	7.43 ± 0.02	7.45 ± 0.02
Pa_{O_2} (mm Hg)	93 ± 9	98.5 ± 10
Pa_{CO_2} (mm Hg)	40 ± 2.5	32 ± 3
Serum HCO_2 (mEq/L)	25.3 ± 1.2	19.9 ± 1.3

Data from [McAuliffe F, Kametas N, Krampl E, et al. Blood gases in pregnancy at sea level and at high altitude. BJOG 2001;108:980-5].

Medications

Selecting appropriate medications for the critically ill obstetric patient is daunting because most commonly used medications cross the placenta and are not well studied in pregnancy. It is important to keep in mind that resuscitating the mother optimizes fetal status and that in the limited studies that exist, most sedatives and resuscitative medications have not been demonstrated to have long-term fetal effects.[29,30] In the event of a precipitous delivery, it is key to inform the neonatal care team of the medications administered to the mother for appropriate support of the neonate.

Use of etomidate for rapid sequence induction has been associated with transient (6 hour) decreases in neonatal cortisol levels without clinical effect[31] and therefore could be considered safe, but effects of its repeated or prolonged use has not been studied. However, the use of propofol during pregnancy is well-studied. It freely crosses the placenta but overall is considered to be safe without teratogenic effects, with caveats in relation to the side effect of lowering maternal blood pressure. In higher doses it may have short-term effects on the fetus.[29,30] Although controlled studies on the fetal effects of dexmedetomidine are lacking, multiple case series and reports have demonstrated no adverse effects in human pregnancy, it has minimal uteroplacental transfer to the fetus, and is generally considered to be safe for use.[30,32] Benzodiazepines are not ideal as first-line agents unless there is another indication, such as seizure or alcohol withdrawal syndrome, because they are known to cause respiratory depression, withdrawal symptoms, and floppy baby syndrome in the neonate.[29,30] Although ketamine was used in pregnancy before the availability of neuroaxial anesthesia, and low doses may be safe, there are conflicting data regarding potential fetal neurotoxic effects, and widespread use at this time is not recommended (**Table 4**).[33]

The commonly used paralytic agents succinylcholine, vecuronium, rocuronium, and cisatracurium are used in pregnancy. All have been noted to cross the placenta, although in small quantities that are proportional to doses given to the mother. For the purposes of rapid sequence induction, a longer-acting paralytic, such as rocuronium, may be preferred because it allows the clinician more time to manage a difficult airway. However, shorter acting neuromuscular blockade with succinylcholine may be preferred if delivery of the fetus is imminent.[30]

With respect to pain management, opioids are used as needed with the understanding that they may cause withdrawal symptoms and respiratory depression in an infant who has had prolonged exposure in utero. Nonsteroidal anti-inflammatory drugs must be avoided to prevent miscarriage, malformations, and premature closure of the fetal patent ductus arteriosus.[34]

Circulatory Shock

Despite the relative hypotension of pregnancy, hypotension should not be presumed to be normal, especially in the third trimester. If possible, a comparison of blood pressures at a patient's obstetric clinic visits and current presentation may be helpful. Management of hypotension should be tailored as soon as possible to the underlying cause. Stabilization of undifferentiated shock may be initiated with IV crystalloids, but it is crucial to limit dilutional coagulopathy in hemorrhage and overload in cardiogenic shock. Even in sepsis, the generally recommended 30 mL/kg of IV crystalloid[35] may be too much given lower colloidal pressures in pregnancy. The Society for Maternal-Fetal Medicine recommends starting with 1 to 2 L and transitioning quickly to vasopressors if the patient does not seem to be fluid responsive.[36] Hemorrhage should be managed with blood products rather than crystalloids and is specifically discussed next.

Table 4
Commonly used medications for intubation and sedation

Sedative Agent	Use in Pregnancy
Etomidate	RSI only
Propofol	Yes
Dexmedetomidine	Yes
Benzodiazepines	If necessary
Ketamine	Not advised

RSI, rapid sequence induction.

A mean arterial pressure greater than 65 mm Hg is a good general goal, although slightly lower mean arterial pressure may be tolerable in pregnant women if end-organ perfusion is not compromised and the fetal heart tracing is reassuring.[36] Although there is the possibility of uterine vessel vasoconstriction with the use of vasopressors or inotropes, the emergency physician should not be afraid to initiate these therapies, because without adequate maternal perfusion fetal hypoxemia and acidosis occurs.[9]

Although studies comparing norepinephrine with other vasopressors in pregnancy are limited, it has been demonstrated to be safe[37] and is recommended as the first-line vasopressor in pregnancy-associated sepsis.[36] The use of ephedrine and phenylephrine in pregnant patients is well-documented, generally in the setting of reversing hypotension because of spinal anesthesia.[38] Both are considered as adjunctive vasopressors. Phenylephrine has more recently been favored over ephedrine because of a lower risk of fetal acidosis,[38,39] but its potential effects of maternal bradycardia and lower cardiac output may limit its utility in critical illness. Finally, vasopressin is used if needed but carries the theoretic risk of stimulating contractions via its action on myometrial oxytocin and vasopressin receptors and is not recommended as a sole or first-line agent.[36,40]

There are no studies comparing inotropes in pregnancy, but cardiogenic shock should be treated with an inotropic agent, preferably dobutamine.[36,40,41] Depending on the underlying cause, the patient with profound cardiogenic shock on maximum medical therapy should be referred for potential intra-aortic balloon pump, mechanical circulatory assist device (eg, Impella, Abiomed, Danvers, MA), or venoarterial ECMO, because there are reports of pregnant patients doing well on these therapies.[42,43] A multidisciplinary team should determine the best course of action for the patient and fetus. Depending on circumstances, imminent delivery may be advisable, and for any procedural therapies it is crucial to consult the appropriate clinicians early so as to not delay care.

CRITICAL ILLNESS SPECIFIC TO PREGNANCY

Peripartum Hemorrhage

Peripartum hemorrhage is common and a major contributor to maternal mortality worldwide.[44] The causes of major hemorrhage are remembered using the "four Ts" (**Table 5**).

Pregnant and postpartum women are at higher risk of developing disseminated intravascular coagulation (DIC) and therefore require aggressive therapy to prevent decompensation and hemorrhage. In cases of postpartum hemorrhage, the obstetrician should be consulted immediately as the emergency physician focuses on patient

Table 5
The 4 Ts of peripartum hemorrhage and initial management

Tone	Uterine atony	Bimanual uterine massage Oxytocin infusion
Trauma	Uterine laceration or rupture Vaginal laceration Uterine inversion	Repair of lacerations Attempt manual reduction (must stop oxytocin, if running, first)
Tissue	Retained placenta Placenta accreta, increta, percreta Placenta previa	Manual removal of retained placenta Transfusion support while awaiting obstetrics arrival
Thrombin	Coagulopathy	Massive transfusion (including cryoprecipitate)

stabilization (Fig. 1). If an obstetrician is unavailable, the general surgeon should be consulted. The interventional radiologist should also be consulted early, because transcatheter arterial embolization can effectively manage several types of peripartum hemorrhage, potentially preserving future fertility.

Clinicians often perform poorly at estimating the volume of blood loss by visual cues,[45] and the physiologic changes to heart rate and cardiac output seen in pregnancy can hide the vital sign abnormalities typically associated with significant

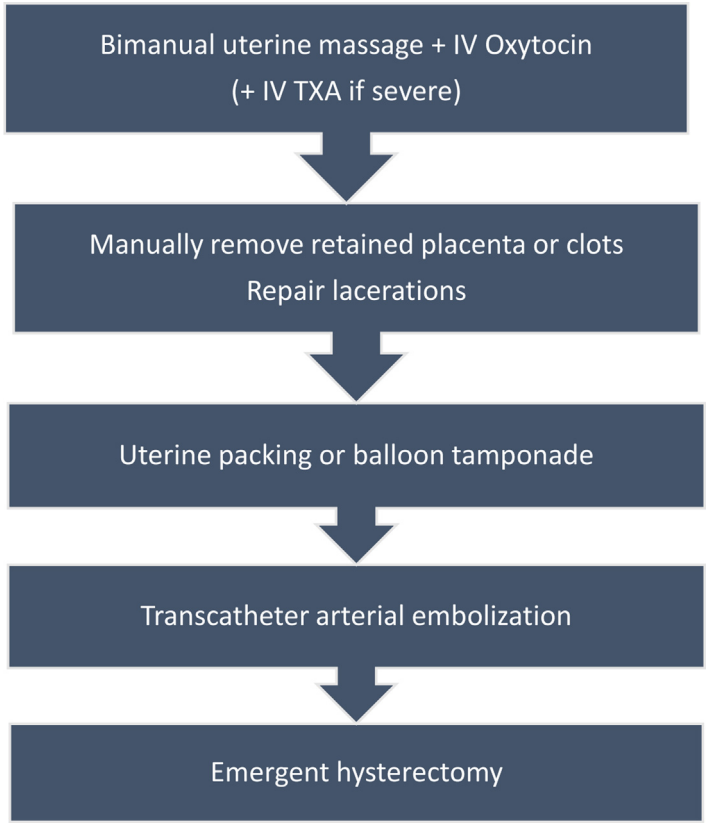

Fig. 1. Steps to control postpartum hemorrhage. IV, intravenous; TXA, tranexamic acid.

hemorrhage. More than a liter of blood may be lost before the pregnant patient becomes hypotensive.[46] Alternately, pregnant women with underlying conditions, such as anemia or preeclampsia, can experience hemorrhagic shock with smaller volumes of blood loss.[45] In general, severe peripartum hemorrhage is managed much the same way as hemorrhage from other causes, using massive transfusion, including cryoprecipitate, and avoiding actions, such as large-volume crystalloid infusion that exacerbate coagulopathy and bleeding.[47,48] Early activation of the massive transfusion protocol, with specific requisition for cryoprecipitate if not included, ensures blood products are rapidly available. If there is no time to appropriately crossmatch blood, Rhesus factor (Rh)-negative products should be used. Rh-negative individuals experiencing hemorrhage should be treated with Rhogam. Additional immediate steps to be taken include:

- Administration of tranexamic acid within the first 3 hours of life-threatening bleed, because it may decrease bleeding-related death in postpartum hemorrhage without increasing thromboembolic events.[49]
- Repair of identified uterine or vaginal lacerations and, in uterine atony, removal of any retained placenta and bimanual uterine massage in concert with oxytocin administration to promote uterine contraction. Dosing is 60 ~ 200 milliunits/min IV, titrating to appropriate uterine contraction.
- If hemorrhage persists, uterine or vaginal packing with Kerlix or gauze should be placed to provide direct pressure. Balloon tamponade with a specialized balloon catheter or even large Foley can also be attempted.[50] A Foley catheter should be inserted into the bladder before packing is placed to prevent compressive urethral obstruction and urinary retention.
- Use of thromboelastography, if available, to guide targeted management of coagulopathy, because early data have shown it to be associated with decreased bleeding and requirement of fewer transfusions.[51]

Preeclampsia with Severe Features and Eclampsia

Preeclampsia is defined as a systolic blood pressure greater than or equal to 140 mm Hg or diastolic pressure greater than or equal to 90 mm Hg, associated with proteinuria, which occurs anytime between 20 weeks of gestation and 6 weeks postpartum.[52] Occurring in approximately 4% of pregnancies, it can advance to preeclampsia with severe features (**Box 1**, or eclampsia, and must be managed aggressively to prevent poor maternal and fetal outcomes.[53] The American College of Obstetricians and Gynecologists recommends management with IV labetalol, IV hydralazine, or oral immediate-release nifedipine as the preferred agents in pregnancy.[53,54] Animal studies have found teratogenic effects with high-doses, but nifedipine has been used in the management of hypertension in pregnancy for decades without signs of fetal harm, although there are no placebo-controlled human studies investigating its use.[55]

In the setting of eclampsia, defined as the presence of seizure activity in the setting of preeclampsia, first ensure the airway is maintained. First-line treatment is IV magnesium sulfate 4 to 6 g over 15 minutes followed by a 1-2 g/h infusion, closely monitoring for signs of magnesium toxicity: vision changes, loss of reflexes, decreased mental status, difficulty breathing. If seizures remain uncontrolled, lorazepam or diazepam may also be used. Consultation with obstetrics and gynecology is essential, because if the seizures are not able to be quickly controlled imminent delivery may be indicated.

Box 1
Signs and symptoms of preeclampsia with severe features

- SBP ≥160 mm Hg or DBP ≥110 mm Hg taken twice at least 4 hours apart while resting
- Abnormal liver function tests (≥2 × the upper limit of normal)
- Renal insufficiency (serum creatinine >1.1 mg/dL or 2 × baseline without other cause)
- Thrombocytopenia (platelet count <100 × 10^9/L)
- Unexplained RUQ or epigastric pain
- New-onset headache unresponsive to medication and not otherwise explained
- Visual disturbance
- Pulmonary edema

Abbreviations: DBP, diastolic blood pressure; RUQ, right upper quadrant; SBP, systolic blood pressure.

Data from [ACOG Practice Bulletin No. 202: Gestational Hypertension and Preeclampsia. Obstet Gynecol 2019;133(1):e1-25].

Amniotic Fluid Embolism

Amniotic fluid embolism (AFE) is a rare cause of maternal critical illness but has a mortality rate of 60%.[56] The underlying pathophysiology of AFE is not clear, but it is thought that amniotic fluid or other debris, such as fetal cells, enters the maternal circulation and triggers a release of inflammatory mediators, leading to cardiopulmonary dysfunction and activation of the clotting cascade, often progressing to DIC. Early recognition and aggressive resuscitative measures are key to improving survival.

The presentation of AFE is usually sudden. Initial symptoms can include shortness of breath, coughing, rigors, diaphoresis, agitation, or acute anxiety, but AFE often presents with cardiopulmonary collapse or even cardiac arrest. Although not distinguishable from high-risk pulmonary embolism, POCUS can help identify potential AFE as a cause of hemodynamic instability, because it usually demonstrates right ventricular strain with an empty left ventricle and septal bowing caused by secondary pulmonary hypertension.[56]

Management of AFE is supportive and focused on treating hypoxemia, supporting circulation, and transfusing blood products, including cryoprecipitate, if the patient is hemorrhaging with DIC.[56,57] Because there is acute right heart strain, use of IV crystalloid should be minimized to avoid further worsening right ventricular overload, and it is prudent to initiate hemodynamic resuscitation with early vasopressor or inotropic support before initiating preload-reducing positive pressure ventilation to avoid peri-intubation decompensation.[58] Norepinephrine remains the vasopressor of choice, whereas dobutamine and milrinone offer pulmonary vasodilation and are effective inotropes.[56] Pulmonary vasodilation with inhaled epoprostenol or nitric oxide, if available, should be initiated to decrease right ventricular afterload.[56] Use of circulatory assist devices and ECMO has been reported in the literature with successful outcomes and is worth serious consideration.[57]

Cardiac Arrest

Maternal cardiac arrest requires rapid action by clinicians if either the pregnant woman or fetus are to survive. The first step is to activate a maternal code, which depending on hospital resources may bring additional support from obstetrics, neonatology, anesthesia, or others.

In patients more than 20 weeks of gestation or with a uterine fundus at or above the umbilicus, the uterus should be manually displaced leftward. Of note, the most recent American Heart Association guidelines removed the recommendation for placement of a wedge or positioning the patient at a tilt, because these practices compromise the effectiveness of chest compressions.[10] Chest compressions should be performed as in the nonpregnant patient, because there are insufficient data to recommend otherwise.[10] Appropriate ventilatory support and high-quality compressions should be continued while addressing the underlying cause of arrest (See **Table 6**).

Table 6
Mnemonics for underlying causes of maternal cardiac arrest

B	Bleeding	A	Anesthetic complications
E	Embolism	B	Bleeding
A	Anesthetic complications	C	Cardiovascular
U	Uterine atony	D	Drugs
C	Cardiac disease	E	Embolism
H	Hypertension (preeclampsia/eclampsia)	F	Fever
O	Other (Hs and Ts, magnesium toxicity)	G	General nonobstetric causes
P	Placenta abruptio/previa	H	Hypertension
S	Sepsis		

Data from [Jeejeebhoy FM, Zelop CM, Lipman S, et al. Cardiac arrest in pregnancy: A scientific statement from the American Heart Association. Circulation 2015;132(18):1747-73] and [Hui D, Morrison LJ, Windrim R, et al. The American Heart Association 2019 guidelines for the management of cardiac arrest in pregnancy: consensus recommendations on implementation strategies. J Obstet Gynaecol Can 2011;33(8):858–63].

Perimortem Cesarean Section

In the event of maternal cardiac arrest, the decision to perform a perimortem cesarean section must be made immediately to obtain the appropriate supplies and deliver within 5 minutes of maternal loss of pulses. This “5-minute rule” exists because it is the time frame in which the chance of maternal and fetal survival is the highest with the most preserved neurologic function, although the mortality for both remains high.[59] It should be performed in patients presenting in cardiac arrest with a pregnancy of known gestation age greater than 24 weeks, or in a pregnancy of unknown gestational age in which the uterine fundus is above the umbilicus.

The benefit of the perimortem cesarean section is two-fold. First, after 24 weeks gestation there is a possibility of fetal survival. Second, with delivery, aortocaval compression is relieved and blood volume previously serving the uteroplacentofetal unit is shunted back to the maternal circulation, increasing cardiac output up to 80%,[3] which may increase the chance of return of spontaneous circulation.

The procedure for a perimortem cesarean section differs from the more common low transverse cesarean section because it is designed to be performed rapidly with easy visualization of structures (**Fig. 2**).[60] Using a scalpel, a vertical incision is made from the top of the uterine fundus to the pubic symphysis. Once the uterus is visualized the bladder is identified and retracted to avoid injury. The scalpel is used to quickly incise the uterus, but scissors should be used to extend the incision to avoid fetal injury, with rupture of the amniotic sac to deliver the fetus, and then clamping and cutting of the umbilical cord and delivery of the placenta. Chest compressions are held briefly while incisions are being made but should be limited to less than 10 seconds.[10,61]

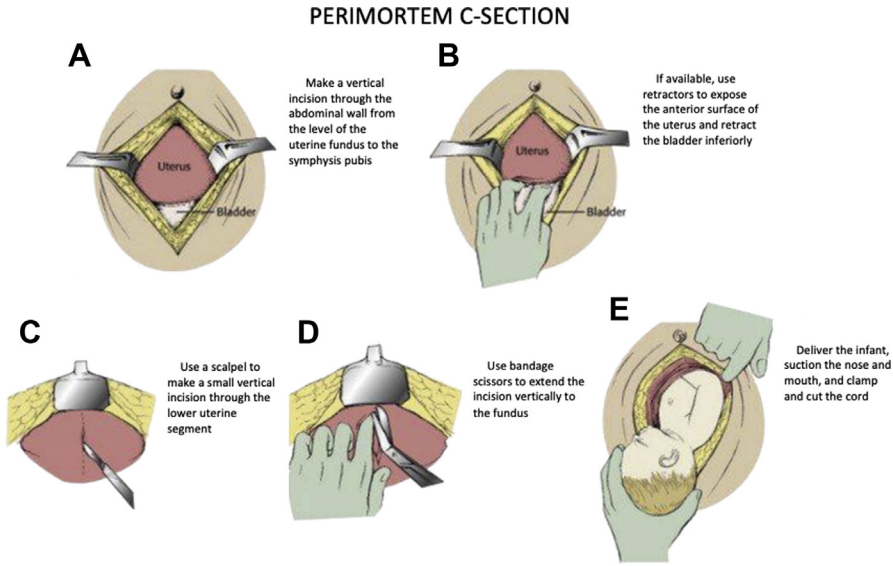

Fig. 2. Perimortem cesarean section. (*A*) Vertical incision through the abdominal wall. (*B*) Retraction of bladder inferiorly. (*C*) Incision through low uterine segment. (*D*) Extension of incision superiorly. (*E*) Delivery of fetus. (*From* Healy ME, Kozubal DE, Horn AE, et al. Care of the critically ill pregnant patient and perimortem cesarean delivery in the emergency department. J Emerg Med 2016;51(2):172-7; with permission.)

SUMMARY

Resuscitation of the critically ill obstetric patient mandates early consultation of a multidisciplinary team because two lives are at stake. Emergency clinicians should appreciate the potential difficulties in intubating the pregnant patient, target pregnancy-specific goals for oxygenation and ventilation, and aggressively support maternal blood pressure to optimize fetal oxygenation and perfusion. Although some medications may be safer in pregnancy than others, stabilizing the pregnant patient is paramount. In cardiac arrest in pregnancies beyond 24 weeks or with a fundal height above the umbilicus, a perimortem cesarean section should be performed with delivery of the fetus within 5 minutes of maternal pulselessness to maximize benefit to patient and fetus.

DISCLOSURE

The authors have nothing to disclose.

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