AHA Scientific Statement

Cardiac Arrest in Pregnancy

A Scientific Statement From the American Heart Association

Farida M. Jeejeebhoy, MD, Chair; Carolyn M. Zelop, MD; Steve Lipman, MD; Brendan Carvalho, MD; Jose Joglar, MD; Jill M. Mhyre, MD; Vern L. Katz, MD; Stephen E. Lapinsky, MB BCh, MSc; Sharon Einav, MD; Carole A. Warnes, MD; Richard L. Page, MD; Russell E. Griffin, LP, FP-C; Amish Jain, MD; Katie N. Dainty, PhD; Julie Arafeh, RN, MS; Rory Windrim, MD; Gideon Koren, MD; Clifton W. Callaway, MD, PhD; on behalf of the American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Diseases in the Young, and Council on Clinical Cardiology

***Abstract*—This is the first scientific statement from the American Heart Association on maternal resuscitation. This document will provide readers with up-to-date and comprehensive information, guidelines, and recommendations for all aspects of maternal resuscitation. Maternal resuscitation is an acute event that involves many subspecialties and allied health providers; this document will be relevant to all healthcare providers who are involved in resuscitation and specifically maternal resuscitation. (*Circulation*. 2015;132:00-00. DOI: 10.1161/CIR.0000000000000300.)**

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Cardiac arrest in pregnancy is one of the most challenging clinical scenarios. Although most features of resuscitating a pregnant woman are similar to standard adult resuscitation, several aspects and considerations are uniquely different. The most obvious difference is that there are 2 patients, the mother and the fetus. Caregivers must have a thorough understanding of maternal mortality to best prevent and treat cardiac arrest in pregnancy. Maternal mortality is defined as the death of a woman during pregnancy and up to 42 days after delivery or termination of pregnancy, provided that the cause of death is related to or aggravated by the pregnancy or its management. Recent data from the US Nationwide Inpatient Sample suggest that cardiac arrest occurs in 1:12 000 admissions for delivery.[1] Globally, 800 maternal deaths occur daily.[2,3] Maternal mortality trends in the United States as reported by the Centers for Disease Control and Prevention from 1989 to 2009 have documented a steady increase from 7.2 deaths per 100 000 live births in 1987 to 17.8 deaths per 100 000 live births in 2009.[4] However, maternal mortality rates are just a small representation of maternal critical events; maternal near-miss data should be considered. A maternal near miss is defined as "a woman who nearly died but survived a complication that occurred during pregnancy, childbirth, or within 42 days of termination of pregnancy."[5] Data from the Netherlands show an incidence of maternal near miss of 1:141 in delivery wards.[6] Among cases with severe maternal morbidity, there was an overall case fatality rate of 1:53.[6] Knowledge deficits[7,8] and poor resuscitation skills[9] could be major contributors to poor outcomes once cardiac arrest has occurred. Despite these problems, recent data show that the rate of survival to hospital discharge after maternal cardiac arrest may be as high as 58.9%,[1] far higher than most arrest populations, further justifying appropriate training and preparation for such events despite their rarity.

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This scientific statement addresses all of the important factors related to maternal arrest, including maternal physiology as it relates to resuscitation, pre-event planning of the critically ill pregnant patient, risk stratification during pregnancy, management of the unstable pregnant patient, basic life support (BLS) in pregnancy, advanced cardiovascular life support (ACLS) in pregnancy, neonatal considerations, emergency medical service (EMS) care, cause of maternal arrest (with a comprehensive discussion found in the online-only Appendix), point-of-care instruments, immediate postarrest care, medicolegal considerations, and knowledge translation, training, and education recommendations.

Methods

Authors with expertise in maternal resuscitation and relevant areas of specialty were selected to contribute to this statement. Selection of the writing group was performed in accordance with the American Heart Association's (AHA's) conflict-of-interest management policy. Relevant literature considered for inclusion in this statement was identified through an up-to-date search strategy of the process used for the 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations and the 2010 International Liaison Committee on Resuscitation worksheets, PubMed, Embase, and an AHA master resuscitation reference library. The search also included a review of bibliographies and manual searches of key articles. Participants volunteered to write sections relevant to their expertise and experience. Drafts of each section were written and sent to the chair of the writing group for incorporation into a single document, which was then edited. The edited document was discussed during Webinars in which participants provided feedback to the primary author of each section and discussed the document as a group. On the basis of these discussions and consensus, the sections were then edited accordingly by the primary author, returned to the chair, and reincorporated into the single document. Further edits were performed by the chair, and a final version of the document was produced. The final document was circulated among all contributors, and consensus was achieved. Recommendations were generated from this process and then assigned a class of recommendation and level of evidence (Table 1). The final document was submitted for independent peer review and has been approved for publication by the AHA Emergency Cardiovascular Care Committee and Science Advisory and Coordinating Committee.

Important Physiological Changes in Pregnancy

Fetal development and maternal maintenance of pregnancy require multiorgan physiological adaptations that are pertinent to the team responding to cardiopulmonary arrest during pregnancy.

Cardiac output rises 30% to 50% as a result of increased stroke volume and, to a lesser extent, increased maternal heart rate (15–20 bpm).[10,11] Systemic vascular resistance decreases as a result of an increase in several endogenous vasodilators, including progesterone, estrogen, and nitric oxide, leading to a decrease in mean arterial pressure, reaching a nadir in the second trimester.[12] The enlarging uterus can produce increased afterload through compression of the aorta and decreased cardiac return through compression of the inferior vena cava, starting at ≈12 to 14 weeks of gestational age.[13] As a result, the supine position, which is most favorable for resuscitation, can lead to hypotension.[13,14] A magnetic resonance imaging study comparing the maternal hemodynamics in the left lateral position with those in the supine position was performed.[15] This study found that at 20 weeks of gestational age, there was a significant increase in ejection fraction of 8% and stroke volume of 27% in the left lateral position. At 32 weeks, there was a significant increase in ejection fraction of 11%, in end-diastolic volume of 21%, in stroke volume of 35%, and in cardiac output of 24% in the left lateral position.[15] Uteroplacental blood flow increases from 50 to close to 1000 mL/min during pregnancy, receiving up to a maximum of 20% of maternal cardiac output at term.[16] Expanded intravascular volume and a decrease in uterine vascular resistance facilitate sufficient uterine placental blood. Overall, uterine vascular reactivity is altered, characterized by reduced tone, enhanced vasodilation, and blunted vasoconstriction. Systemic hypotension can overwhelm the compensatory mechanisms, which attempt to maintain uterine blood flow.

Functional residual capacity decreases by 10% to 25% during pregnancy as the uterus enlarges and elevates the diaphragm. Increased ventilation (ie, an increase in tidal volume and minute ventilation) occurs, beginning in the first trimester, reaching a level 20% to 40% above baseline by term, mediated by the elevated serum progesterone levels.[17] This produces a mild respiratory alkalosis with compensatory renal excretion of bicarbonate, resulting in an arterial carbon dioxide pressure of ≈28 to 32 mm Hg (3.7–4.3 kPa) and a plasma bicarbonate level of 18 to 21 mEq/L.[18] Oxygen consumption increases because of the demands of the fetus and maternal metabolic processes, reaching a level 20% to 33% above baseline by the third trimester.[19] The reduced functional residual capacity reservoir and increased consumption of oxygen are responsible for the rapid development of hypoxemia in response to hypoventilation or apnea in the pregnant woman.[20] The oxyhemoglobin dissociation curve is shifted to the right in the mother during pregnancy (P_{50} increases from 27 to 30 mm Hg). A higher partial pressure of oxygen is therefore required to achieve the same maternal oxygen saturation. The same curve is shifted to the left in the fetus (P_{50} is 19 mm Hg), conferring relative resilience to hypoxic conditions. Upper airway edema and friability occur as a result of hormonal effects and may reduce visualization during laryngoscopy and increase the risk of bleeding.

Pregnancy is characterized by glomerular hyperfiltration and increased renal blood flow by 40% to accommodate the maternal role of fetal detoxification of metabolic byproducts and maintenance of maternal osmoregulation in the face of increased circulatory intravascular volume. Altered tubular function prevents wasting of glucose, amino acids, and proteins required by both maternal and fetal metabolisms. On balance, Starling forces favor a narrowing of the oncotic pressure–wedge pressure gradient, increasing the tendency for pulmonary edema to develop.[21]

Progesterone relaxes gastroesophageal sphincters and prolongs transit times throughout the intestinal tract during the second and third trimesters,[22,23] predisposing the patient to aspiration of stomach contents.

Drug metabolism is altered by several different mechanisms in pregnancy.[23a–23d] In addition to changes in renal

Table 1. Applying Classification of Recommendations and Level of Evidence

ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT	SIZE OF TREATMENT EFFECT: CLASS I *Benefit >>> Risk* Procedure/Treatment **SHOULD** be performed/ administered	CLASS IIa *Benefit >> Risk* *Additional studies with focused objectives needed* **IT IS REASONABLE** to perform procedure/administer treatment	CLASS IIb *Benefit ≥ Risk* *Additional studies with broad objectives needed; additional registry data would be helpful* Procedure/Treatment **MAY BE CONSIDERED**	CLASS III *No Benefit* or CLASS III *Harm* COR III: No benefit — Procedure/Test: Not Helpful — Treatment: No Proven Benefit COR III: Harm — Procedure/Test: Excess Cost w/o Benefit or Harmful — Treatment: Harmful to Patients
LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	■ Recommendation that procedure or treatment is useful/effective ■ Sufficient evidence from multiple randomized trials or meta-analyses	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Sufficient evidence from multiple randomized trials or meta-analyses
LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is useful/effective ■ Evidence from single randomized trial or nonrandomized studies	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Evidence from single randomized trial or nonrandomized studies
LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	■ Recommendation that procedure or treatment is useful/effective ■ Only expert opinion, case studies, or standard of care	■ Recommendation in favor of treatment or procedure being useful/effective ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation's usefulness/efficacy less well established ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Only expert opinion, case studies, or standard of care
Suggested phrases for writing recommendations	should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	COR III: No Benefit: is not recommended; is not indicated; should not be performed/ administered/ other; is not useful/ beneficial/ effective COR III: Harm: potentially harmful; causes harm; associated with excess morbidity/mortality; should not be performed/ administered/ other
Comparative effectiveness phrases†	treatment/strategy A is recommended/indicated in preference to treatment B treatment A should be chosen over treatment B	treatment/strategy A is probably recommended/indicated in preference to treatment B it is reasonable to choose treatment A over treatment B		

A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as sex, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use.

†For comparative effectiveness recommendations (Class I and IIa; Level of Evidence A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

physiology, gastrointestinal absorption and gastrointestinal transit affect bioavailability. Protein binding changes also alter the free fraction of the drug available. Steroid-induced acceleration of the hepatic P450 metabolism and increased renal clearance will also lower circulating drug levels.

Estimating Gestational Age

Management decisions made during a maternal cardiac arrest may require estimation of gestational age. Symphysis fundal height is the measurement from the top of the maternal pubic bone to the top of the uterine fundus. In a singleton pregnancy, with the fetus in a longitudinal lie, this height in centimeters will approximately correspond to the gestational age in weeks when measured between 16 and 36 weeks of gestation. If a tape measure is not available, finger breadths are usually used as a surrogate for the centimeters. Classically accepted rule-of-thumb landmarks may also be used: Gestational age is 12 weeks if the uterus is palpable at above the pubic symphysis, 20 weeks if the uterus is palpable at the level of the umbilicus,[24] and 36 weeks if the uterus is palpable at the level of the xiphisternum. However, the fundus can be a poor predictor of gestational age and may reach the umbilicus between 15 and 19 weeks of gestation.[25] In the last month of pregnancy, after 36 weeks of gestation, there may be diminution of the fundal height from 36 down to ≈32 cm as the fetal head engages into the pelvis. Fundal height may also be skewed by other factors such as abdominal distention[24]

and increased body mass index; therefore, fundal height may be a poor predictor of gestational age.

Recommendation

1. **Code team members with responsibility for pregnant women should be familiar with the physiological changes of pregnancy that affect resuscitation technique and potential complications (*Class I; Level of Evidence C*).**

The Critically Ill Pregnant Patient

Pre-Event Planning

The critically ill pregnant patient may be managed in units not accustomed to managing obstetric patients such as the intensive care unit (ICU), coronary care unit, or medical or surgical ward. Teams on these units need to prepare for unexpected emergencies in these patients[26] by covering the following 4 important steps:

1. Preparation for cardiac arrest: Educate staff about the management of cardiac arrest in pregnancy.
2. Preparation for perimortem cesarean delivery (PMCD): Identify contact details or appropriate code calls to mobilize the entire maternal cardiac arrest response team, and ensure the availability of equipment for cesarean delivery and resuscitation of the neonate. In cultures that require consent for a PMCD, even in the event of a cardiac arrest, pre-event consent should be obtained.
3. Preparation for management of obstetric complications: Stock drugs and equipment commonly available in obstetric units, including oxytocin and prostaglandin $F_{2\alpha}$. Pre-event planning for power of attorney related to healthcare decisions should be done for the critically ill patient.
4. Decisions involving the resuscitation status of the neonate: Decisions about fetal viability should be made in collaboration with the obstetrician, neonatologist, and family. The decision depends on the gestational age and, to a significant degree, the neonatal facilities available. This information should be clearly documented.

Severity of Illness and Early Warning Scores

The British Center for Maternal and Child Enquiries report of 2011 (2006–2008 triennium) has stated that timely recognition of pregnant women at risk of potentially life-threatening conditions plays an important role in the appropriate institution of treatment.[27] Brief checklists are provided for the identification of a number of conditions, including sepsis, respiratory distress, and neurological complications. The report also highlights the potential value of a modified early obstetric warning score. In a more recent publication, using a large British ICU data set, Carle et al[28] described the evaluation of several preexisting obstetric early warning scores and the development and validation of a new obstetric score and demonstrated excellent discrimination between survivors and nonsurvivors for this new score (area under the receiver-operating characteristic curve, 0.995). These scores can be used to monitor patients by clinical use of an early warning score chart (Figure 1) and can accurately identify patients at high risk of mortality, although not specifically mortality resulting from cardiac arrest. Therefore, they are of value in patient management and triage.

Recommendations

1. **Pregnant women who become ill should be risk stratified by the use of a validated obstetric early warning score (*Class I; Level of Evidence C*).**
2. **Hospital units with a pregnant woman in their care should ensure that proper pre-event planning has been instituted, including preparation for maternal cardiac arrest and neonatal resuscitation (*Class I; Level of Evidence C*).**

Management of the Unstable Pregnant Patient

Rapid response to instability in the pregnant patient is essential for the prevention of cardiac arrest. Maternal hemodynamics must be optimized; hypoxemia must be treated; and intravenous access must be established.

Recommendations[29]

1. **The patient should be placed in a full left lateral decubitus position to relieve aortocaval compression (*Class I; Level of Evidence C*).**
2. **Administration of 100% oxygen by face mask to treat or prevent hypoxemia is recommended (*Class I; Level of Evidence C*).**
3. **Intravenous access should be established above the diaphragm to ensure that the intravenously administered therapy is not obstructed by the gravid uterus (*Class I; Level of Evidence C*).**
4. **Precipitating factors should be investigated and treated (*Class I; Level of Evidence C*).**

Cardiac Arrest Management

Basic Life Support

The cardiac arrest in pregnancy in-hospital BLS algorithm should be used as a guide to management (Figure 2).

First Responders

Nurses are often first responders in cardiopulmonary arrest; however, any hospital staff member may witness or discover a patient in arrest and should be able to begin basic emergency care.[30] Basic emergency care is crucial. Rapid mobilization of expert resuscitation teams and BLS performed competently until the arrival of these teams give the woman the best chance for return of spontaneous circulation (ROSC).

Accomplishing a coordinated, well-executed first response is challenging in patient care areas in which cardiopulmonary arrest rarely occurs, including obstetric units.[30–41] The unique physiology of pregnancy renders the patient vulnerable to hypoxemia and hemodynamic disadvantage, given the rapid development of desaturation with apnea and the presence of aortocaval compression when the patient is unconscious and supine. Therefore, all BLS interventions are essential and should be initiated rapidly and simultaneously once the rescuers arrive. First responders should

Date										
Time										
Systolic BP										
<80	3									
80–89	2									
91–139	0									
140–149	1									
150–159	2									
>160	3									
Respiratory rate										
<10	3									
10–17	0									
18–24	1									
25–29	2									
>30	3									
Heart rate										
<60	3									
60–110	0									
111–149	2									
>150	3									
FIO_2 to keep Sat >96%										
Room air	0									
24%–39%	1									
>40%	3									
Temperature										
<34	3									
34.1–35.0	1									
35.1–37.9	0									
38.0–38.9	1									
>39.0	3									
Consciousness										
Alert (GCS=15)	0									
Not alert (<15)	3									

Figure 1. Example of a color-coded early warning score chart based on the score developed by Carle et al.[28] A score ≥6 should trigger a call for support from the intensive care unit or rapid response team and initiation of continuous monitoring of vital signs. BP indicates blood pressure; FIO2, fraction of inspired oxygen; GCS, Glasgow Coma Scale score; and Sat, saturation.

initiate the usual resuscitation measures simultaneously, including placement of the backboard and provision of chest compressions and appropriate airway management, defibrillation when appropriate, and manual left uterine displacement (LUD). To accomplish all tasks effectively, a minimum of 4 BLS responders should be present.

Chest Compressions in Pregnancy

Adult Chest Compression Science

As with all adult resuscitations, high-quality chest compressions are essential to maximize the patient's chance of survival. For high-quality chest compressions, the patient must be supine on a hard surface, the rescuer's hands must be placed correctly, the correct rate and depth of compressions must be performed, and interruptions must be minimized. Because hospital beds are typically not firm and some of the force intended to compress the chest results in mattress displacement rather than chest compression, we have traditionally recommended the use of a backboard despite insufficient evidence for or against the use of backboards during cardiopulmonary resuscitation (CPR).[42–45] If a backboard is used, care should be taken to avoid delays in the initiation of CPR, to minimize interruptions in CPR, and to avoid line/tube displacement.[46] Air-filled mattresses should be deflated when CPR is performed.[45–49] Chest compression physiology has been best studied only with the patient in the supine position and has never been studied in a patient placed in a tilt.[50] Chest compression recommendations for the pregnant patient are the same as the most current recommendations for adult resuscitation.

Recommendations

1. **Chest compressions should be performed at a rate of at least 100 per minute at a depth of at least 2 in (5 cm), allowing full recoil before the next compression, with minimal interruptions, and at a compression-ventilation ratio of 30:2[51] (*Class IIa; Level of Evidence C*).**
2. **Interruptions should be minimized and limited to 10 seconds except for specific interventions such as insertion of an advanced airway or use of a defibrillator[51] (*Class IIa; Level of Evidence C*).**
3. **The patient should be placed supine for chest compressions (*Class I; Level of Evidence C*).**
4. **There is no literature examining the use of mechanical chest compressions in pregnancy, and this is not advised at this time.**

Factors Affecting Chest Compressions in the Pregnant Patient

Aortocaval Compression. In the pregnant patient, supine positioning will result in aortocaval compression. Relief of aortocaval compression must be maintained continuously during resuscitative efforts and continued throughout postarrest care. Manual LUD should be used to relieve aortocaval compression during resuscitation. Historically, tilt has been used as an option to relieve aortocaval compression during resuscitation. Rees and Willis[52] found that at >30° left lateral tilt, the manikin slid off the incline plane and that chest compression force was

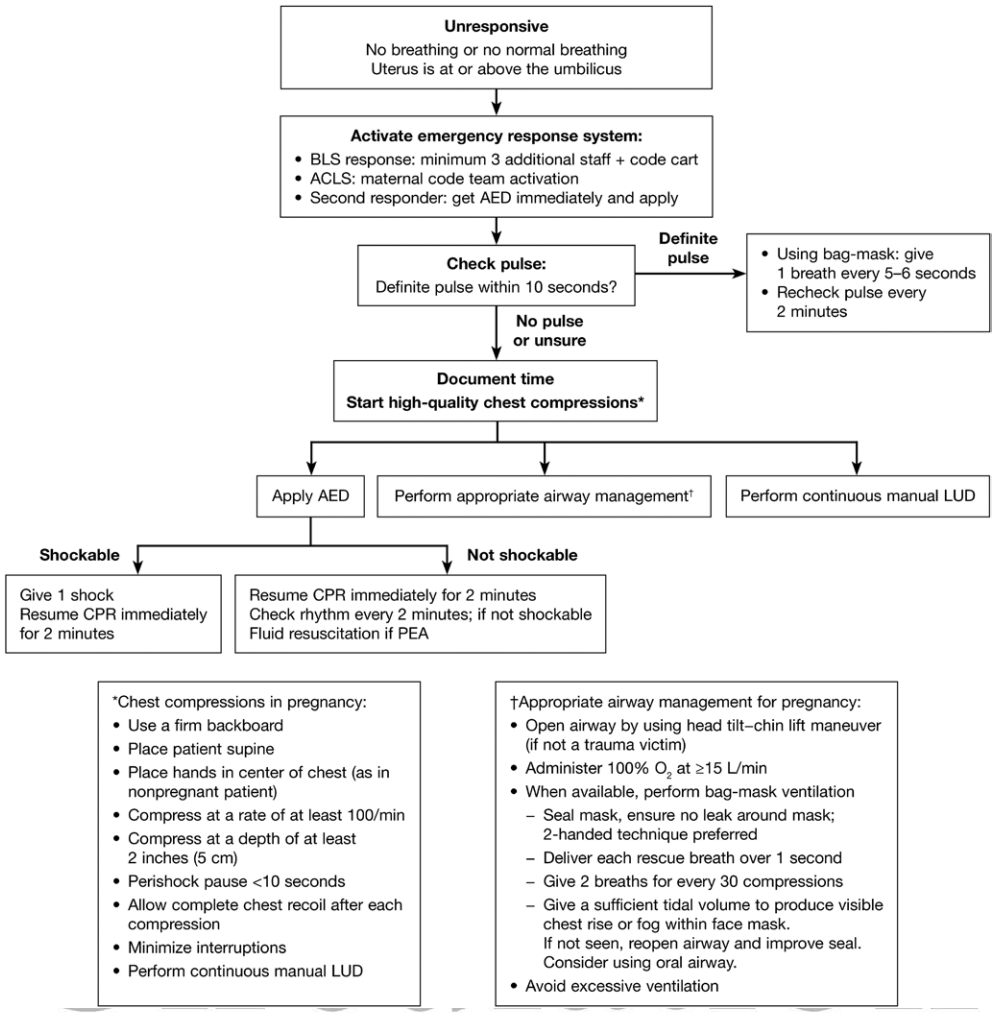

Figure 2. Cardiac arrest in pregnancy in-hospital basic life support (BLS) algorithm: simultaneous C-A-B-U (chest compressions/current-airway-breathing-uterine displacement). ACLS indicates advanced cardiovascular life support; AED, automated external defibrillator; CPR, cardiopulmonary resuscitation; LUD, left uterine displacement; and PEA, pulseless electric activity.

reduced as the angle of inclination was increased. It has also been found that at >30° lateral tilt, inferior vena cava compression can still occur.[53] In addition, the heart has been shown to shift laterally during tilt[54] compared with the supine position. Therefore, chest compressions performed with the patient in a tilt could be significantly less effective than those performed with the patient in the usual supine position, and this could have a major impact on the chance of successful resuscitation.[52,54] In the nonarrest population, manual LUD compared with 15° left lateral tilt has been shown to result in less hypotension and a significantly lower ephedrine requirement during a cesarean delivery.[55] Additional benefits of manual LUD over tilt include easier access for both airway management and defibrillation. While manual LUD is performed, the patient can remain supine and receive usual resuscitative measures, including high-quality chest compressions without hindrance (Figure 3). Manual LUD can be performed from the left of the patient (Figure 4), where the uterus is cupped and lifted up and leftward off the maternal vessels, or from the right of the patient (Figure 3), where the uterus is pushed upward and leftward off the maternal vessels. The rescuer must be careful not to inadvertently push down, which would increase the amount of inferior vena cava compression and negatively affect maternal hemodynamics.

Recommendations[29]

1. **Continuous manual LUD should be performed on all pregnant women who are in cardiac arrest in which the uterus is palpated at or above the umbilicus to relieve aortocaval compression during resuscitation (*Class I; Level of Evidence C*).**
2. **If the uterus is difficult to assess (eg, in the morbidly obese), attempts should be made to perform manual LUD if technically feasible (*Class IIb; Level of Evidence C*).**

Positioning of Hands During Chest Compressions. There is no scientific evidence to support changing the recommendation for hand placement for chest compressions in the pregnant patient compared with the nonpregnant patient. Previous guidelines recommended placing the hands slightly higher on the sternum in the pregnant patient, but there are no scientific data to support this recommendation.

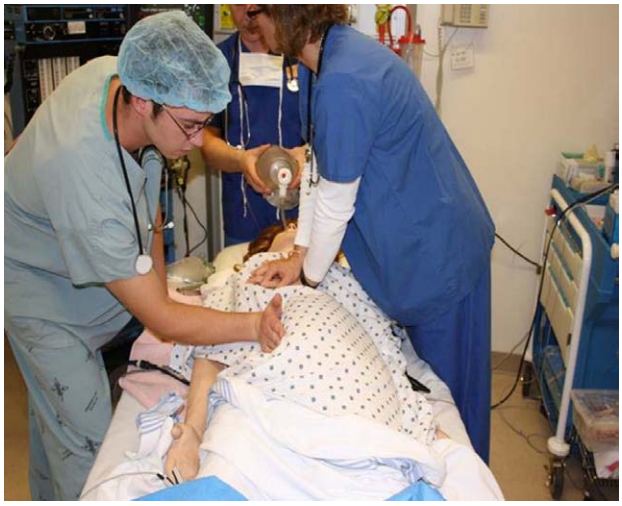

Figure 3. Manual left uterine displacement by the 1-handed technique from the right of the patient during adult resuscitation.

Recommendation

1. **The rescuer should place the heel of 1 hand on the center (middle) of the victim's chest (the lower half of the sternum) and the heel of the other hand on top of the first so that the hands overlap and are parallel (*Class IIa; Level of Evidence C*).**

Transporting Pregnant Women During Chest Compressions. Simulation of chest compressions on manikins has shown that the quality of CPR decreases during transport to the operating room.[56]

Recommendation

1. **Because an immediate cesarean delivery may be the best way to optimize the condition of the mother and fetus (see section on "PMCD"), this operation should optimally occur at the site of the arrest. A pregnant patient with in-hospital cardiac arrest should not be transported for cesarean delivery. Management should occur at the site of the arrest (*Class I; Level of Evidence C*). Transport to a facility that can perform a cesarean delivery may be required when indicated (eg, for out-of-hospital cardiac arrest or cardiac arrest that occurs in a hospital not capable of cesarean delivery).**

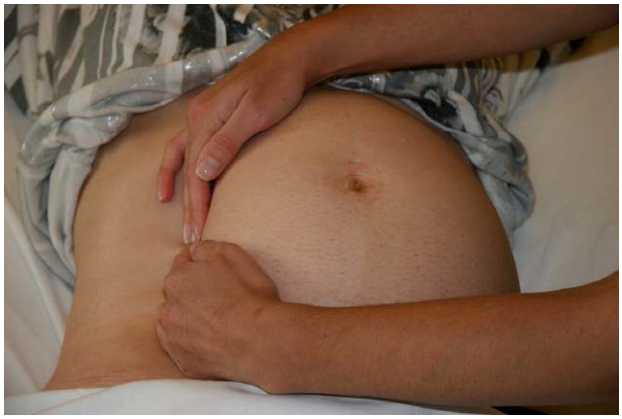

Figure 4. Manual left uterine displacement by the 2-handed technique from the left of the patient.

Defibrillation Issues During Pregnancy

Prompt application of defibrillation in the setting of ventricular fibrillation or pulseless ventricular tachycardia is critical to maximize the likelihood of survival. This is no different in the pregnant patient. Transthoracic impedance remains unchanged during pregnancy compared with the nonpregnant state; therefore, the energy required for defibrillation during cardiac arrest in pregnancy would be the same as the most current recommendations for the nonpregnant patient.[57] Application of defibrillation and cardioversion shocks to the maternal chest would be expected to pass minimal energy to the fetus and is considered safe in all stages of pregnancy.[29] Defibrillation would be unlikely to cause electric arcing to fetal monitors, and the presence of fetal monitors should not deter providers from the use of rapid defibrillation when indicated.[29] When indicated, defibrillation should be performed in the pregnant patient without hesitation or delay. The risk to the mother in delaying appropriate defibrillation would outweigh any potential concern about defibrillation in the setting of fetal monitors.

Recommendations

1. **The same currently recommended defibrillation protocol should be used in the pregnant patient as in the nonpregnant patient. There is no modification of the recommended application of electric shock during pregnancy[29] (*Class I; Level of Evidence C*).**
2. **The patient should be defibrillated with biphasic shock energy of 120 to 200 J (*Class I; Level of Evidence B*) with subsequent escalation of energy output if the first shock is not effective and the device allows this option.[58]**
3. **Compressions should be resumed immediately delivery of the electric shock[58] (*Class IIa; Level of Evidence C*).**
4. **For in-hospital settings where staff have no ECG rhythm recognition skills or where defibrillators are used infrequently such as in an obstetric unit, the use of an automated external defibrillator may be considered[58] (*Class IIb; Level of Evidence C*).**
5. **Anterolateral defibrillator pad placement is recommended as a reasonable default (*Class IIa; Level of Evidence C*). The lateral pad/paddle should be placed under the breast tissue, an important consideration in the pregnant patient.**
6. **The use of adhesive shock electrodes is recommended to allow consistent electrode placement (*Class IIa; Level of Evidence C*).**

Airway and Breathing

Hypoxemia develops more rapidly in the pregnant patient compared with the nonpregnant patient; therefore, rapid, high-quality, and effective airway and breathing interventions are essential. As discussed above, a higher partial pressure of oxygen is required to achieve the same maternal oxygen saturation, thus highlighting the importance of ensuring maternal oxygenation and ventilation concurrent with effective chest compressions in the pregnant

patient. As a result, in the 2010 AHA guidelines for CPR and emergency cardiovascular care (ECC), the recommendations for management of cardiac arrest in pregnancy note the importance of early bag-mask ventilation with 100% oxygen.[29] Airway management should always be considered more difficult in the pregnant patient; therefore, appropriate airway algorithms for pregnancy should be instituted. For first responders with minimal airway experience, bag-mask ventilation with 100% oxygen is the most rapid noninvasive strategy to initiate ventilation.[59] The standard initial compression-ventilation ratio of 30:2 will minimize interruptions in chest compressions. Hyperventilation has been shown to decrease the likelihood of survival in nonpregnant arrest victims, particularly when it interrupts chest compressions.[60] Two-handed bag-mask ventilation is more effective than a single-handed technique[61] and should be used as soon as a second provider is available to compress the self-inflating bag. If attempts at mask ventilation do not produce visible chest rise or fog within the face mask, the rescuer should try to reopen the airway and improve the seal of the mask on the patient's face. An oral airway may help relieve airway obstruction in the hypopharynx. Ideally, airway patency should be maintained continuously to optimize oxygen delivery. Obesity, sleep apnea, and airway edema all increase the difficulty of face mask ventilation.[62]

BLS Recommendations (Actions Are Simultaneous, Not Sequential)

1. **Rapid notification should be provided to the maternal cardiac arrest response team[29,63–65] (*Class I; Level of Evidence C*).**
2. **The time when pulselessness was confirmed should be documented[66] (*Class I; Level of Evidence C*).**
3. **High-quality CPR should be paired with uterine displacement, and a firm backboard should be used[42-45] (*Class I; Level of Evidence C*).**
4. **Rapid automated defibrillation should be provided whenever it is indicated as appropriate by rhythm analysis[63,65] (*Class I; Level of Evidence C*).**
5. **Appropriate BLS airway management should be initiated.**
   a. **A member of the first responder team should perform bag-mask ventilation with 100% oxygen flowing to the bag at a rate of at least 15 L/min (*Class IIb; Level of Evidence C*).**
   b. **Two-handed bag-mask ventilation is preferred (*Class IIa; Level of Evidence C*).**
6. **Hospitals need to establish first-responder roles that satisfy all of the requirements for BLS, including modifications recommended during pregnancy. A minimum of 4 staff members should respond for BLS resuscitation of the pregnant patient. All hospital staff should be able to fulfill first-responder roles (*Class I; Level of Evidence C*).**

Advanced Cardiovascular Life Support

A fast and well-coordinated response to maternal cardiac arrest is important, and the cardiac arrest in pregnancy in-hospital ACLS algorithm should be used as a guide to management (Figure 5). The ACLS maternal cardiac arrest team will continue BLS tasks and perform advanced airway management, insert an intravenous access above the diaphragm, and administer the usual ACLS drugs and doses when indicated. With the arrival of the obstetric and neonatal teams, preparation for PMCD can begin. The ACLS algorithm includes PMCD as a treatment option for the mother who has not achieved ROSC by 4 minutes after the onset of cardiac arrest and in whom the uterus extends to or above the umbilicus. The cause of the arrest needs to be considered and addressed as necessary.

The Maternal Cardiac Arrest Team

Activating and achieving prompt code team response is one of the most fundamental tasks to be completed during maternal cardiac arrest.[29] Each hospital must have a specific method to activate the maternal cardiac arrest team; for example, "maternal code blue" or "code blue maternal" could serve as a universal call to action. Creating 1 "bundled" emergency code call (eg, maternal code blue) to all necessary responders simultaneously may save time, help prevent confusion, and reduce the risk of team members not being notified. The universal call to action should also prompt rescuers to bring the necessary specialized equipment (see section on "Special Equipment Required for a Maternal Cardiac Arrest") to the scene of the arrest without delay. The composition of the code team must reflect the fact that 2 critically ill patients (mother and fetus) must be resuscitated.

Recommendations

1. **There should be 1 call to action that activates the maternal cardiac arrest team, notifies all members, and ensures that specialized equipment is brought to the scene without delay (*Class I; Level of Evidence C*).**
2. **The maternal cardiac arrest team would ideally be composed of the following[26] (*Class I; Level of Evidence C*):**
   a. **An adult resuscitation team (potentially composed of critical care physicians and nurses, and/or emergency physicians and nurses, and/or internal medicine physicians and nurses, or other service lines such as general surgery and trauma, with respiratory therapy or equivalent [ie, nurse or physician] and pharmacy representatives according to institutional policy, etc)**
   b. **Obstetrics: 1 obstetric nurse, 1 obstetrician**
   c. **Anesthesia care providers: obstetric anesthesiologist if available or staff anesthesiologist; anesthesia assistant or certified nurse anesthetist if available**
   d. **Neonatology team: 1 nurse, 1 physician, 1 neonatal respiratory therapist or equivalent (ie, nurse or physician)**
   e. **In centers without obstetric/neonatology services, it is suggested that the cardiac arrest committee and hospital emergency services discuss contingency plans in the event of maternal cardiac arrest.**
3. **Leadership during a maternal cardiac arrest is complicated, given the multiple teams involved. Leadership will depend on where the arrest occurs and may be specific to institutional practices. In**

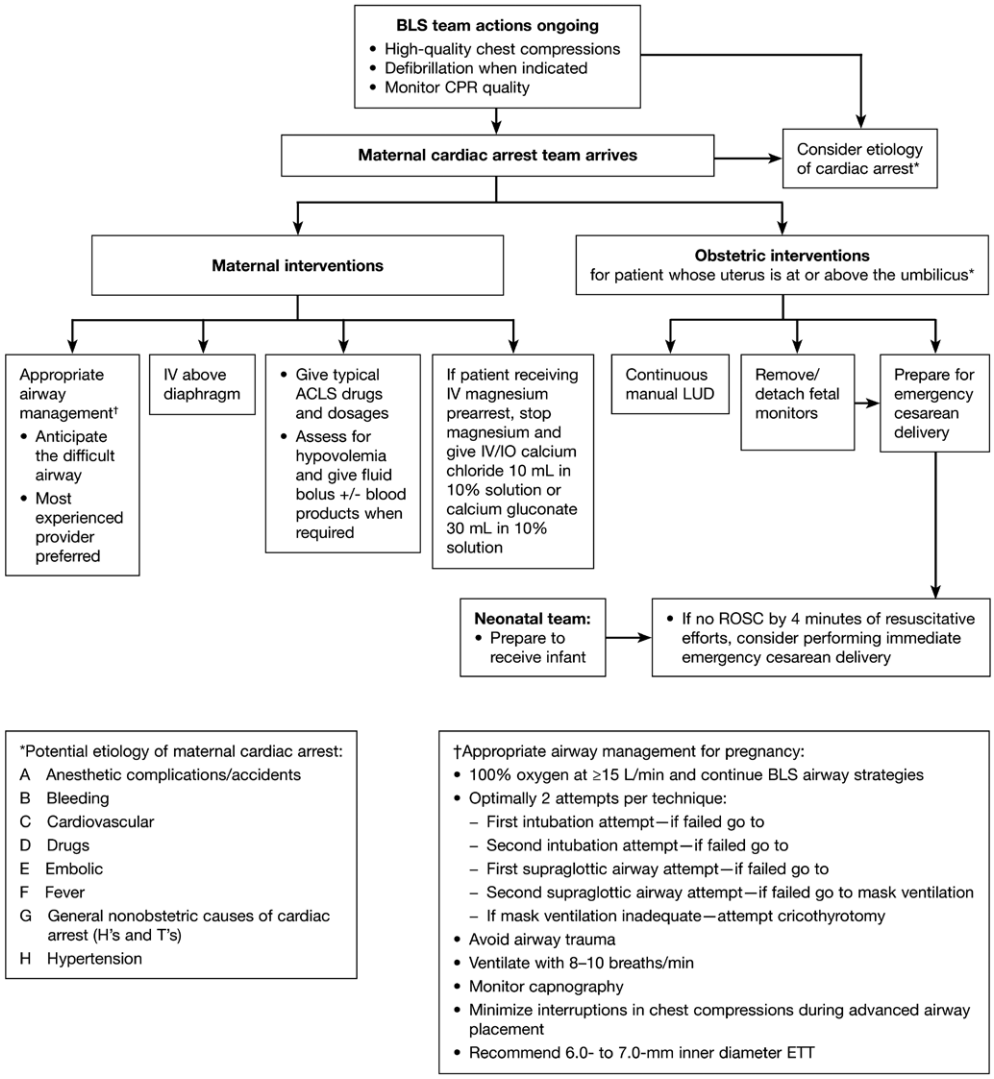

Figure 5. Cardiac arrest in pregnancy in-hospital advanced cardiovascular life support (ACLS) algorithm. BLS indicates basic life support; CPR, cardiopulmonary resuscitation; ETT, endotracheal tube; IV, intravenous; IO, intraosseous; LUD, left uterine displacement; and ROSC, return of spontaneous circulation.

general, there should be a team leader for adult resuscitation, a team leader for obstetric care, and a team leader for neonatal/fetal care. One approach to deal with multiple subspecialties is for the usual cardiac arrest team leader to delegate leadership for obstetric care, fetal/neonatal care, and airway/ventilatory management. All team leaders must communicate effectively together to make decisions about code management (*Class I; Level of Evidence C*).

Special Equipment Required for a Maternal Cardiac Arrest

Special equipment is required for a maternal cardiac arrest. Emergency response committees must ensure that there is a process for delivering this specialized equipment to the code scene without delay if it is not already located on the code cart. Options include either delegating a specific member of the code team to bring the equipment or locating it on the code cart. Specialized equipment should include a PMCD tray (Table 2) but at a minimum must include a scalpel. In addition, equipment for a difficult airway (Table 3) may be required for the mother. Neonatal resuscitation equipment will be required if the fetus is delivered and viable (Table 4).

Breathing and Airway Management in Pregnancy

Management of Hypoxia

Current guidelines for the management of cardiac arrest in adults stress that oxygen delivery to vital organs is limited by blood flow during CPR and that chest compressions should not initially be interrupted for ventilation or airway placement. The pregnant patient has a very limited oxygen reserve. Furthermore, it should be noted that cardiac arrest secondary to hypoxia (eg, severe pneumonia, aspiration, amniotic fluid embolism, acute respiratory distress syndrome, narcotic therapy, high spinal block) requires early attention to airway and ventilation. Although delayed endotracheal intubation combined with passive oxygen delivery and minimally interrupted chest compressions has been associated with a better

Table 2. Recommended Equipment for Perimortem Cesarean Section*

Equipment Contents of the Emergency Cesarean Delivery Tray
Scalpel with No. 10 blade
Lower end of a Balfour retractor
Pack of sponges
2 Kelly clamps
Needle driver
Russian forceps
Sutures and suture scissors

*The items listed in this table represent suggestions. The contents should be selected to meet the specific needs, preferences, and skills of the practitioner and healthcare facility.

outcome in witnessed ventricular fibrillation arrest,[67] this is not necessarily the case in the pregnant patient, particularly those with preexisting hypoxia.

Recommendation

1. **Hypoxemia should always be considered as a cause of cardiac arrest. Oxygen reserves are lower and the metabolic demands are higher in the pregnant patient compared with the nonpregnant patient; thus, early ventilatory support may be necessary (*Class I; Level of Evidence C*).**

Airway Management

It is essential to be familiar with airway management algorithms in maternal cardiac arrest, given the high likelihood of a difficult airway in the pregnant patient.

Because endotracheal intubation is frequently more difficult in pregnant patients compared with the nonpregnant surgical population[68,69] and is best achieved with minimal or no disruption in chest compressions, any intubation attempts should be undertaken by an experienced laryngoscopist. Forceful laryngoscopy can lead to bleeding and airway edema that interferes with ventilation.[70] Therefore, regardless of provider experience with laryngoscopy, optimally no more than 2 attempts at either direct laryngoscopy or videolaryngoscopy should be made before insertion of a supraglottic airway.[71,72] The glottis in pregnancy is often smaller because of edema; therefore, starting with a smaller endotracheal tube (ETT) may increase the likelihood of successful intubation. Face mask ventilation between laryngoscopic attempts may preserve oxygenation; any difficulty in ventilation indicates the need to avoid further laryngoscopy and to select alternative methods of airway management. Supraglottic airway placement is the preferred rescue strategy to facilitate ventilation after failed intubation.[73] Supraglottic airway devices with an esophageal drain provide access to the stomach to relieve air and stomach contents and may reduce the risk of regurgitation and aspiration pneumonitis. Subsequent exchange with a definitive airway with fiberoptic guidance may be considered for women with ROSC. If oxygenation and ventilation are not successful with a supraglottic device or ETT and mask ventilation is impossible, a "cannot intubate, cannot ventilate" situation has occurred. Help (in-house general, trauma, or otolaryngology surgeons) must be called emergently, and the final pathway steps in the difficult airway algorithm must be followed for establishing emergency invasive airway access (eg, percutaneous cricothyroidotomy). Pregnant women and those who are immediately postpartum are at increased risk of regurgitation and aspiration of stomach contents. Despite these concerns, chest compressions, oxygenation, and relief of aortocaval compression are a higher priority than techniques to limit the risk of regurgitation (eg, cricoid pressure, rapid intubation) when caring for the obstetric victim of cardiopulmonary arrest. The 2010 AHA guidelines for CPR and ECC do not recommend the use of cricoid pressure during resuscitation of nonpregnant patients,[74] and there are no data to support its use in the management of pregnant patients during cardiopulmonary arrest. Cricoid pressure may not be effective at preventing aspiration[75] and can impede ventilation and laryngoscopy. If cricoid pressure is used, it should be adjusted or released if ventilation is difficult or the laryngoscopic view is poor. In the event of regurgitation before intubation, suction may be used to remove gastric contents from the oropharynx during ongoing chest compressions.

Table 3. Recommended Airway and Breathing Equipment*

Equipment to Be Used by First Responders	Equipment to Be Used by Experts
Oxygen	Laryngoscope and assorted blades
Bag-valve-mask devices (eg, Ambu Bag with disk valve as opposed to duckbill valve preferred	Videolaryngoscope
Appropriate size face masks and oral airways	Cuffed tracheal tubes: size 6.0- to 7.0-mm inner diameter with a semirigid stylet and a range of backup sizes available
Stethoscope	Gum elastic bougie
Pulse oximeter	Airway exchange catheter
Qualitative carbon dioxide detector	Supraglottic airways in a range of sizes
Suction device	Flexible fiberoptic intubation equipment
	Equipment suitable for emergency invasive airway access (eg, cricothyrotomy)
	Exhaled carbon dioxide detector

*The items listed in this table represent suggestions. The contents should be selected to meet the specific needs, preferences, and skills of the practitioner and healthcare facility.

Continuous capnography should be used if available to assess correct placement of the ETT, the quality of chest compressions, and ROSC. Confirmation of endotracheal placement is complicated by the fact that end-tidal partial pressure of carbon dioxide (P_{ETCO_2}) may decrease to almost zero during cardiac arrest and increase only after the onset of effective CPR. The presence of a decreasing or flat capnographic tracing should prompt the physician leading the code to ensure vigorous chest compressions and LUD, to re-evaluate the location of the airway device, and to consider obstructive causes of cardiopulmonary arrest (ie, massive pulmonary embolism, cardiac tamponade, or pneumothorax). ROSC is more likely when P_{ETCO_2} can be sustained >10 mm Hg; an abrupt increase in P_{ETCO_2} by ≈10 mm Hg is consistent with ROSC. Findings

Table 4. Recommended Neonatal Resuscitation Equipment

Airway	Breathing	Circulation/Drugs	Miscellaneous
Suction apparatus: Suction bulb Mechanical suction Suction catheters (6F–12F) Meconium aspirator ET intubation supplies: Laryngoscope (blade size: 0 for preterm and 1 for term infants) Extra bulbs and batteries ET tubes (internal diameters: 2.5, 3.0, 3.5, 4.0 mm) Stylets ET-securing device Carbon dioxide detector Tapes and scissors Laryngeal mask airway size 1 (for use when ET intubation is not feasible)	Positive-pressure ventilation: Device: T-piece resuscitator/flow-inflating bag/self-inflating bag with oxygen reservoir (least preferred) Bag sizes 240–750 mL, fitted with pop-off valve Mask sizes 0 and 1 (cushioned preferred) Sources of compressed air and O_2 O_2 blender with a flowmeter (capacity up to 10 L/min) Plastic tubing	IV access: Sterile gloves Antiseptic solution Cord tie Scalpel Umbilical venous catheters (3.5F–5.0F) 3-Way stopcock Suture for securing 24-Gauge IV cannulas (for use if umbilical vessel access not feasible) IO needle (rarely needed) Drugs: Epinephrine 1:10 000 (0.1 mg/mL) Normal saline bags 10% dextrose bags 4.2% sodium bicarbonate (rarely indicated) Saline flushes	Temperature regulation: Heat source (radiant warmer preferred) Warm towels Thermometer (servo-controlled preferred) Clean plastic bag (for extremely premature neonates) Monitoring: Neonatal stethoscope Neonatal cardiac leads Pulse oximeter Neonatal/infant oximeter probes Clock (digital preferred) Other: Firm resuscitation surface (preferably height adjustable) Good light source Nasogastric tubes (6F–10 F) Sterile syringes (1, 3, 5, 20 mL) Clean, sterile gloves Sterile gauzes Bottles to collect blood samples suitable for low-volume blood testing

ET indicates endotracheal; IO, intraosseous; and IV, intravenous.

suggestive of adequate chest compressions, ROSC, or both include a rising P_{ETCO_2} level or levels >10 mm Hg.[76–80]

Recommendations

1. **Endotracheal intubation should be performed by an experienced laryngoscopist (*Class I; Level of Evidence C*).**
   a. **Starting with an ETT with a 6.0- to 7.0-mm inner diameter is recommended (*Class I; Level of Evidence C*).**
   b. **Optimally no more than 2 laryngoscopy attempts should be made (*Class IIa; Level of Evidence C*).**
   c. **Supraglottic airway placement is the preferred rescue strategy for failed intubation (*Class I; Level of Evidence C*).**
   d. **If attempts at airway control fail and mask ventilation is not possible, current guidelines for emergency invasive airway access should be followed (call for help, obtain equipment).**
2. **Prolonged intubation attempts should be avoided to prevent deoxygenation, prolonged interruption in chest compressions, airway trauma, and bleeding (*Class I; Level of Evidence C*).**
3. **Cricoid pressure is not routinely recommended (*Class III; Level of Evidence C*).**
4. **Continuous waveform capnography, in addition to clinical assessment, is recommended as the most reliable method of confirming and monitoring correct placement of the ETT (*Class I; Level of Evidence C*) and is reasonable to consider in intubated patients to monitor CPR quality, to optimize chest compressions, and to detect ROSC (*Class IIb; Level of Evidence C*). Findings consistent with adequate chest compressions or ROSC include a rising P_{ETCO_2} level or levels >10 mm Hg (*Class IIa; Level of Evidence C*).**
5. **Interruptions in chest compressions should be minimized during advanced airway placement (*Class I; Level of Evidence C*).**

Arrhythmia-Specific Therapy During Cardiac Arrest

Medical therapy during cardiac arrest is no different in the pregnant patient than in the nonpregnant patient. For patients with refractory (shock-resistant) ventricular fibrillation and tachycardia, the drug of choice is amiodarone; in 2 separate randomized studies, amiodarone has been shown to improve survival to hospital admission compared with standard of care and lidocaine.[81,82] The US Food and Drug Administration categories of fetal risk of medications do not apply in the cardiac arrest scenario because fetal concerns are overshadowed by the arrest outcome.

Recommendations

1. **For refractory (shock-resistant) ventricular fibrillation and tachycardia, amiodarone 300 mg rapid infusion should be administered with 150-mg doses repeated as needed (*Class IIb; Level of Evidence C*).**
2. **Medication doses do not require alteration to accommodate the physiological changes of pregnancy. Although there are changes in the volume of distribution and clearance of medication during pregnancy, there are very few data to guide changes in current recommendations (*Class IIb; Level of Evidence C*).**
3. **In the setting of cardiac arrest, no medication should be withheld because of concerns about fetal teratogenicity[83] (*Class IIb; Level of Evidence C*).**

4. **Physiological changes in pregnancy may affect the pharmacology of medications, but there is no scientific evidence to guide a change in current recommendations. Therefore, the usual drugs and doses are recommended during ACLS (*Class IIb; Level of Evidence C*).**

Other Drugs Used During ACLS

Historically, in the setting of cardiac arrest, vasopressors such as epinephrine and vasopressin have been used with the goal of increasing myocardial and cerebral blood flow and improving patient outcomes. However, as stated in the 2010 AHA guidelines for CPR and ECC,[74] these drugs have not been shown to improve neurologically intact long-term survival.

Epinephrine, an α-adrenergic receptor stimulant that has been used for many years, has been shown to augment cerebral and myocardial perfusion during cardiac arrest in preclinical studies.[84] However, data in support of clinical efficacy in humans are scant. The first randomized trial evaluating this drug, involving 851 patients assigned to ACLS with drug versus ACLS with no drug, demonstrated an improvement in ROSC with epinephrine but no difference in survival to hospital discharge or long-term survival.[85] These results are consistent with prior observations, and thus, in view of the short-term benefit, the 2010 AHA guidelines for CPR and ECC state that it is reasonable to administer 1 mg IV/IO epinephrine every 3 to 5 minutes (*Class IIb; Level of Evidence A*).[74]

Vasopressin is a nonadrenergic peripheral vasoconstrictor that was studied as an alternative to epinephrine in view of its powerful vasoconstrictive properties. However, in several randomized trials, vasopressin did not prove to be superior to epinephrine, either alone or in combination with epinephrine.[86,87] Because the clinical effect of vasopressin is regarded as equivalent to that of epinephrine, the 2010 AHA guidelines for CPR and ECC recommend 40 U IV/IO vasopressin as an alternative to the first or second dose of epinephrine (*Class IIb; Level of Evidence A*).[74]

A new concept under investigation is the use of a combination of drugs during vasopressor-requiring cardiac arrest. A randomized study of patients with in-hospital cardiac arrest demonstrated that the combination of vasopressin-epinephrine and methylprednisolone during CPR and stress-dose hydrocortisone in postresuscitation shock led to improved survival to hospital discharge compared with epinephrine alone.[88] Despite the promising results, additional studies are needed before recommendations can be made about combined vasopressors.

No data are available comparing the use of different vasopressors during arrest in pregnant patients, but because vasopressin can induce uterine contraction and both agents are considered equivalent in the nonpregnant patient, epinephrine is the preferred agent of the two.[89]

Atropine was removed from the 2010 AHA guidelines for CPR and ECC ACLS cardiac arrest algorithm in view of its lack of efficacy, and it is indicated only for cases of bradycardia.[74]

Recommendations

1. **Administering 1 mg epinephrine IV/IO every 3 to 5 minutes during adult cardiac arrest should be considered. In view of the effects of vasopressin on the uterus and because both agents are considered equivalent, epinephrine should be the preferred agent (*Class IIb; Level of Evidence C*).**
2. **It is recommended that current ACLS drugs at recommended doses be used without modifications[29] (*Class IIa; Level of Evidence C*).**

Fetal Assessment During Cardiac Arrest

During active CPR, the focus should remain on maternal resuscitation and restoration of maternal pulse and blood pressure with adequate oxygenation. During this time, evaluation of the fetal heart will not be helpful and carries the risk of inhibiting or delaying maternal resuscitation and monitoring. Should the mother achieve ROSC and her condition be stabilized, then fetal heart surveillance may be instituted when deemed appropriate.

Recommendations

1. **Fetal assessment should not be performed during resuscitation (*Class I; Level of Evidence C*).**
2. **Fetal monitors should be removed or detached as soon as possible to facilitate PMCD without delay or hindrance (*Class I; Level of Evidence C*).**

Delivery

This section is written on the premise that the patient's arrest occurred in an institution that has staff with the expertise to deliver a neonate. PMCD is defined as the birth of the fetus after maternal cardiac arrest, most commonly during resuscitation. Birth is almost always accomplished through cesarean delivery. A review of all published cases of PMCD up to 2010 showed that PMCD led to a clear maternal survival benefit in 19 of 60 cases (31.7%), and there were no cases in which PMCD may have been deleterious to maternal survival.[90]

There may be situations during advanced pregnancy in which noninvasive relief of inferior vena cava compression with manual LUD is not enough to provide a hemodynamic advantage to result in successful resuscitation. This is when PMCD needs to be considered as the definitive means to achieve complete relief of inferior vena cava compression and as a treatment option during ACLS measures for maternal cardiac arrest.

The purpose of timely perimortem delivery is 2-fold. The first is facilitation of resuscitation. If cardiac output has not yet been effectively established, relieving aortocaval compression by emptying the uterus significantly improves resuscitative efforts. Second, and of critical importance, early delivery of the baby, the second patient, is accomplished with a decreased risk of permanent neurological damage from anoxia. In situations in which the mother is nonresuscitatable (eg, severe trauma is present), timely delivery of the fetus is essential.

Resuscitation team leaders should activate the protocol for a PMCD as soon as cardiac arrest is identified in a pregnant

woman whose uterus extends to or above the umbilicus.[29] By the time the physician is ready to deliver the baby, standard ACLS should be underway, and immediately reversible causes of cardiac arrest should have been ruled out. When the gravid uterus is large enough to cause maternal hemodynamic changes as a result of aortocaval compression, PMCD should be considered regardless of fetal viability.[29]

What Defines a Gravid Uterus With the Potential to Cause Aortocaval Compression?

Several factors determine the weight of the gravid uterus, and these additive factors could result in a uterus that is heavy enough or positioned in such a manner to cause aortocaval compression. Determinants of uterine weight include weight of the fetus, the number of fetuses, and the weight of the fluid (ie, polyhydramnios). Other factors that may affect the degree of aortocaval compression include the size of the fetus, the relationship of the fetus to the woman's anatomy, and additional factors such as increased body mass index and morbid obesity.

Unfortunately, published evidence does not adequately address each of these contingencies. However, some general principles can be used to guide recommendations.

A study found that maternal aortocaval compression can occur for singleton pregnancies at >20 weeks of gestational age.[14] One review of PMCD in maternal cardiac arrest before the third trimester concluded that if the fundus extends above the level of the umbilicus, aortocaval compression can occur, and PMCD should be performed regardless of gestational age.[25] Two cases of maternal cardiac arrest in early pregnancy of 13 to 15 weeks were reported in which the mother was resuscitated without PMCD being performed and the pregnancy continued to successful delivery of a live infant at term.[91,92] Not every pregnant woman in cardiac arrest is a candidate for PMCD; the decision depends on whether the gravid uterus is thought to interfere with maternal hemodynamics.

Why Perform PMCD in Cardiac Arrest?

Several case reports of PMCD during a maternal cardiac arrest resulted in ROSC or improvement in maternal hemodynamic status only after the uterus had been emptied.[93–103] In a case series of 38 PMCDs, 12 of 20 women for whom maternal outcome was recorded had ROSC immediately after delivery.[93] No cases of worsened maternal status after cesarean delivery were reported.[93] The critical point to remember is that both mother and infant may die if the provider cannot restore blood flow to the mother's heart.

The Importance of Timing With PMCD

The 5-minute window that providers have to determine whether cardiac arrest can be reversed by BLS and ACLS was first described in 1986 and has been perpetuated in specialty guidelines.[93,103a] It was recommended that PMCD should begin at 4 minutes to effect delivery at 5 minutes after failed resuscitative efforts. This time interval was chosen to minimize the risks of neurological damage, which begins to develop after 4 to 6 minutes of anoxic cardiac arrest if there is no ROSC.[104] The rescue team was not required to wait 5 minutes before initiating PMCD, and there are circumstances that support an earlier start.[24] For instance, in an obvious nonsurvivable injury[30] in which the maternal prognosis is grave and resuscitative efforts appear futile, moving straight to PMCD may be appropriate, especially if the fetus is viable. In the situation of an unwitnessed arrest in which a prolonged period of pulselessness is suspected, the priority of PMCD comes to the forefront.

Many reports document long intervals between a decision for an urgent hysterotomy and actual delivery of the infant, far exceeding the obstetric guideline of 30 minutes for patients not in arrest.[105,106] Very few cases of PMCD fall within the previously recommended 5-minute period.[90,93,94] However, survival of the mother has been reported with PMCD performed up to 15 minutes after the onset of maternal cardiac arrest.[94,107–109] Therefore, if PMCD could not be performed by the 5-minute mark, it was still advisable to prepare to evacuate the uterus while the resuscitation continued. At >24 to 25 weeks of gestation, the best survival rate for the infant occurs when the infant is delivered no more than 5 minutes after the mother's heart stops beating.[104,110–112] At gestational ages >30 weeks, infant survival has been seen even when delivery occurred >5 minutes from the onset of maternal cardiac arrest.[93] In a recent retrospective cohort series, neonatal survival was documented even when delivery occurred up to 30 minutes after the onset of maternal cardiac arrest.[94] A systematic review of all published maternal cardiac arrests occurring before delivery after widespread adoption of resuscitation guidelines (1980–2010) demonstrated a good outcome in most cases; Cerebral Performance Category was assessed to have been 1 or 2 in 78.4% (40 of 51) of mothers and 52.3% (22 of 42) of neonates after the event. In cases undergoing PMCD, the average time elapsing from arrest to delivery was significantly different between surviving (27 of 57) and nonsurviving (30 of 57) mothers (10.0±7.2 minutes [median, 9 minutes; range, 1–37 minutes and 22.6±13.3 minutes [median, 20 minutes; range, 4–60 minutes], respectively; $P<0.001$; 95% confidence interval, 6.9–18.2). The area under the receiver-operating curve for the prediction of maternal death by the time that elapsed from arrest to delivery was 0.827. For neonates, the time elapsing from arrest to delivery was described in only 57 cases and was <4 minutes in only 4 cases. Mean times were 14±11 minutes (median, 10 minutes; range, 1–47 minutes) and 22±13 minutes (median, 20 minutes; range, 4–60 minutes) in surviving and nonsurviving neonates, respectively ($P=0.016$), and the area under the receiver-operating curve for the prediction of neonatal death by the time that elapsed from arrest to delivery was only 0.729, reflecting the wide range of arrest-to-delivery survival times.[90]

PMCD Technique

The procedure should be performed at the site of the maternal resuscitation. Time should not be wasted moving the patient. Simulation with a manikin of maternal transport to the operating room during cardiac arrest has found that transport increases the time to PMCD.[113] Additionally, time should not be wasted waiting for surgical equipment or doing abdominal preparation. If desired, antiseptic solution may be poured on the maternal abdomen. The only equipment needed to start a PMCD is a scalpel.

Resuscitative efforts should be continued during cesarean delivery, including manual LUD. The position of the rescuer performing the manual LUD will need to accommodate the surgical field to allow access and to prevent injury to the rescuer.

The technique used to perform the PMCD is at the discretion of the physician performing the procedure. Both the vertical incision and the Pfannenstiel incision are acceptable. The vertical incision provides better visualization of the abdomen and pelvis and may prove beneficial in treating the cause of the arrest. The vertical abdominal incision is also considered faster. However, the Pfannenstiel incision may be preferred by a provider who is more comfortable with performing this technique. Because successful PMCD has also been described with the Pfannenstiel incision, it is a reasonable alternative.

During PMCD, the fetus is delivered and given to the neonatal resuscitation team. The placenta is delivered. The uterus should then be quickly wiped clean, and the uterine incision should be closed with a running locking stitch of absorbable suture. The abdomen is closed in the regular fashion. A Foley catheter should be placed at this time if not already present. After the procedure, if maternal resuscitation has been successful, administration of antibiotics and oxytocin may be considered. However, oxytocin should be used with caution because it can precipitate rearrest (see the section on etiology in the online-only Appendix).

Teams find it difficult to perform PMCD in a timely fashion. Therefore, emergency preparedness is important for maternal cardiac arrest. Institutions with limited resources or only 1 staff member available (who, for example, may be involved in an operation when the cardiac arrest is called) should have staff respond as quickly as possible. Response times may be dictated by the reality of staff numbers and availability. Given the rarity of maternal cardiac arrest, it is not reasonable to allocate 2 in-house obstetricians to be on call 24/7 solely for the purpose of responding to a maternal cardiac arrest if the institution otherwise has a more restricted staffing protocol.

Recommendations

1. **During cardiac arrest, if the pregnant woman (with a fundus height at or above the umbilicus) has not achieved ROSC with usual resuscitation measures with manual uterine displacement, it is advisable to prepare to evacuate the uterus while resuscitation continues (*Class I; Level of Evidence C*).**
2. **Decisions on the optimal timing of a PMCD for both the infant and mother are complex and require consideration of factors such as the cause of the arrest, maternal pathology and cardiac function, fetal gestational age, and resources (ie, may be delayed until qualified staff is available to perform this procedure). Shorter arrest-to-delivery time is associated with better outcome (*Class I; Level of Evidence B*).**
3. **PMCD should be strongly considered for every mother in whom ROSC has not been achieved after ≈4 minutes of resuscitative efforts (*Class IIa; Level of Evidence C*).**
4. **If maternal viability is not possible (through either fatal injury or prolonged pulselessness), the procedure should be started immediately; the team does not have to wait to begin the PMCD (*Class I; Level of Evidence C*).**
5. **When PMCD is performed, the following are recommended:**
   a. **The woman should not be transported to an operating room for PMCD during the management of an in-hospital maternal cardiac arrest (*Class IIa; Level of Evidence B*).**
   b. **The team should not wait for surgical equipment to begin the procedure; only a scalpel is required (*Class IIa; Level of Evidence C*).**
   c. **The team should not spend time on lengthy antiseptic procedures. Either a very abbreviated antiseptic pour should be performed, or the step should be eliminated entirely (*Class IIa; Level of Evidence C*).**
   d. **Continuous manual LUD should be performed throughout the PMCD until the fetus is delivered (*Class IIa; Level of Evidence C*). Care should be taken to avoid injury to the rescuer performing the manual LUD during PMCD.**
6. **If the uterus is difficult to assess (eg, in the morbidly obese), then determining the size of the uterus may prove difficult. In this situation, PMCD should be considered at the discretion of the obstetrician by using his or her best assessment of the uterus. In these patients, bedside ultrasound may help guide decision making (*Class IIa; Level of Evidence C*).**

Vaginal Delivery During Maternal Cardiac Arrest

Few published cases describe vaginal delivery during a cardiac arrest in pregnancy.[114] Obstetric caregivers involved in an intrapartum cardiac arrest resuscitation may conduct a vaginal examination, provided that CPR is being adequately performed by the medical team. If the cervix is found to be fully dilated and the fetal head is at an appropriately low station, immediate assisted vaginal delivery can be considered. This will allow resuscitation of the fetus and will facilitate the resuscitation of the mother because of the factors discussed above.

Recommendation

1. **Assisted vaginal delivery should be considered when the cervix is dilated and the fetal head is at an appropriately low station (*Class IIb; Level of Evidence C*).**

Neonatal Resuscitation Considerations

Neonatal Resuscitation Team

It is expected that each maternity hospital will have a designated team for managing unexpected neonatal resuscitations. Because of the high likelihood of delivering a depressed neonate after maternal arrest, the team attending delivery must anticipate and be prepared for an advanced resuscitation. This includes designating a team leader, checking equipment, and

preassigning specific roles to team members. Team composition optimally should include a neonatologist/pediatrician, neonatal nurses, and respiratory therapists who should be familiar with the local neonatal resuscitation algorithms.[115] At least 1 member of the team must be skilled in emergency neonatal endotracheal intubation. In some settings, this may require accepting the urgent assistance of other subspecialty professionals, for example, an anesthesiologist, an otolaryngologist, or emergency physicians.

Recommendations

1. **The neonatal resuscitation team should be notified of the impending delivery and its circumstances as early as feasible to allow maximum preparatory time (*Class I; Level of Evidence C*).**
2. **The following critical information should be provided to the neonatal resuscitation team leader: gestational age, number of fetuses, and mode of delivery (*Class I; Level of Evidence C*).**
3. **In the event of multiple pregnancies, it is recommended that each fetus be resuscitated by a separate resuscitation team (*Class I; Level of Evidence C*).**

Neonatal Resuscitation Equipment

PMCD may be performed outside the maternity unit and will require the team to perform resuscitation in a relatively unfamiliar environment that may lack optimal equipment. Each hospital must have prestocked neonatal crash carts available, the locations of which should be clearly marked and known to the neonatal resuscitation team. Alternatively, neonatal resuscitation equipment can be prestocked in easy-to-carry bags that can be taken to the area of need by the resuscitation team on notification of impending delivery. Such carts/bags must be fully stocked, regularly checked, and accessible from all relevant clinical locations of the hospital. A comprehensive list of all equipment deemed necessary for neonatal resuscitation is presented in Table 4.

EMS Considerations

Maternal cardiac arrest that occurs out of hospital will likely have worse outcomes than cardiac arrest that occurs in hospital.[90] Therefore, a coordinated EMS response to maternal cardiac arrest is of critical importance. If possible, additional prehospital providers should respond to the location of the maternal arrest to ensure that a sufficient number of providers is available to provide BLS (Figure 6) and ACLS care, including LUD. Prehospital providers should not be expected to perform a PMCD; however, transporting the mother in cardiac arrest to a location where PMCD can be performed in a timely manner is essential. Fetal cardiac activity may be slow but present after many minutes of maternal pulselessness.[116,117] As a result, fetal survival can occur in cases when maternal vital signs are lost before arrival in the emergency department and when CPR fails to restore maternal pulses.[118]

Immediate transport of the obviously pregnant patient, identified as the uterus extending to or above the umbilicus, should be initiated. This is justified because PMCD may be required to achieve ROSC by relieving aortocaval compression, decreased time to PMCD is associated with better fetal outcomes,[90] resources to perform PMCD are usually lacking in the field,[119] and multiple teams will be required to resuscitate the neonate and the mother after PMCD.

EMS medical directors should identify appropriate receiving hospitals for obviously pregnant patients according to the resources available within the service area. Choices should include whether a specialized obstetric center or a center with a neonatal ICU is preferred over the closest destination. As a result of the geographical restrictions of rural systems, the closest appropriate receiving hospital might be used regardless of the availability of obstetric or neonatal services. For PMCD to be used as a lifesaving procedure, it is extremely time dependent; delays as short as 5 minutes affect fetal survivability. Therefore, transport should be directed toward a center that is prepared to perform PMCD rather than the closest facility, but optimally transport should not be prolonged by >10 minutes to reach a center with more capabilities (eg, neonatal ICU). Although it is possible that prehospital transport will take >5 minutes and the likelihood of PMCD success will therefore be lower, there is still less advantage to transporting a patient to a facility where PMCD cannot be offered. When trauma is the proximate cause of maternal cardiac arrest, resuscitation including PMCD can be performed at a trauma center with early activation of the hospital maternal cardiac arrest team. Bypassing the trauma bay or emergency department to arrive at the operating room is not advisable, given the evidence that CPR quality is impaired during transfer[56] and that procedures do not occur faster in the operating room compared with other settings.[113]

EMS providers in rural or other resource-limited settings may be faced with limited staff and equipment and extended transportation distances to the most appropriate receiving hospitals. EMS systems that provide care in the rural setting should consider these factors during planning and attempt to optimize care through coordinated education and training with first-responder organizations.

EMS responders should use the cardiac arrest in pregnancy out-of-hospital BLS algorithm for healthcare providers as the basis of care during a maternal cardiac arrest (Figure 6).

Recommendations

1. **If resources are available, EMS response to a maternal cardiac arrest should include the appropriate complement of staff to ensure that BLS and ACLS actions can be performed, including chest compressions, LUD, defibrillation when indicated, and management of the difficult airway (*Class I; Level of Evidence C*).**
2. **If available, transport should be directed toward a center that is prepared to perform PMCD, but transport should not be prolonged by >10 minutes to reach a center with more capabilities (*Class IIb; Level of Evidence C*).**

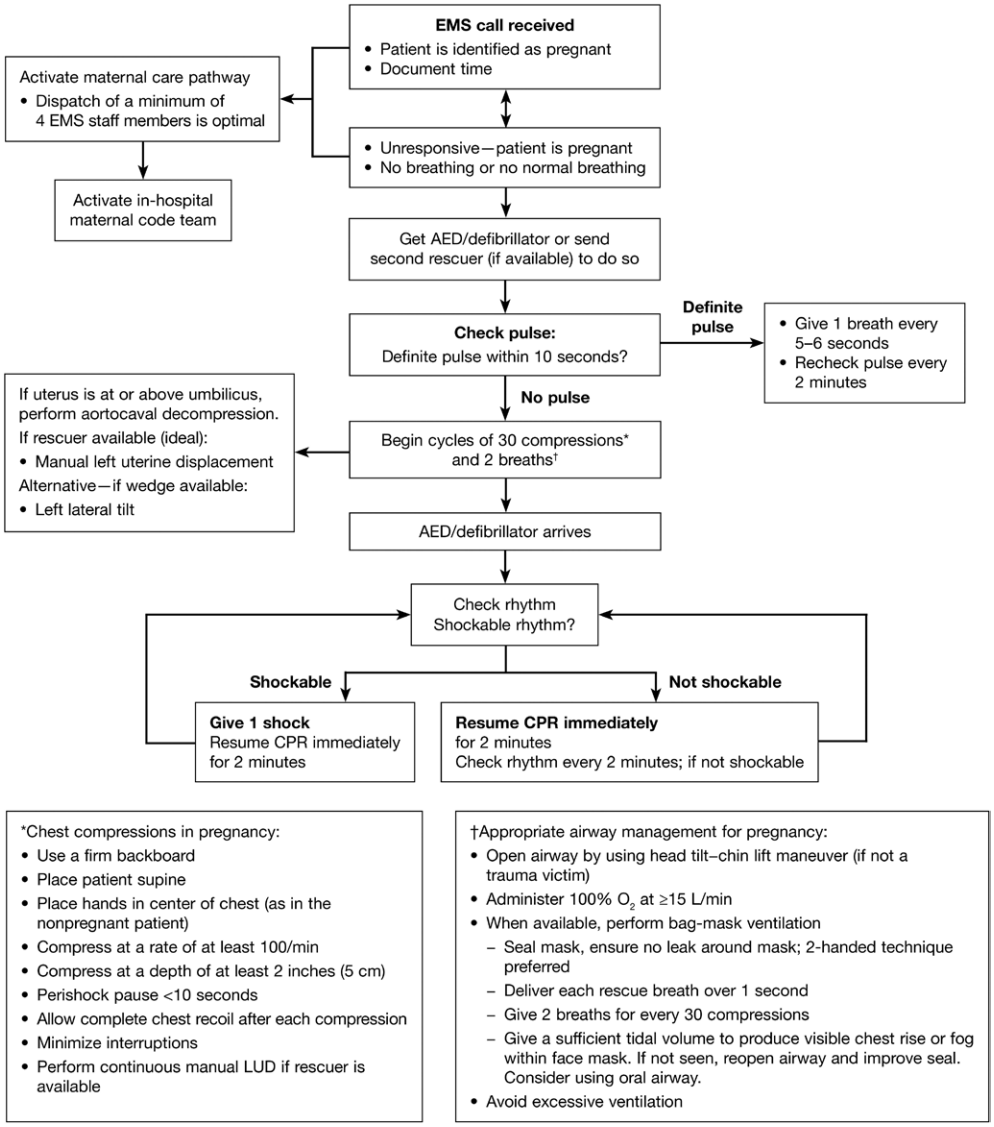

Figure 6. Cardiac arrest in pregnancy out-of-hospital basic life support (BLS) algorithm for healthcare providers. AED indicates automated external defibrillator; CPR, cardiopulmonary resuscitation; EMS, emergency medical services; and LUD, left uterine displacement.

3. **EMS and the receiving emergency department must establish optimal communication and an action plan for the transport of a maternal cardiac arrest patient. The emergency department should be able to rapidly mobilize the maternal cardiac arrest team, and specialized equipment should be available from the time the patient arrives in the emergency department (*Class I; Level of Evidence C*).**

Cause of the Cardiac Arrest

Similar to the recommendations for adult (nonpregnant) ACLS,[74] an understanding of the importance of diagnosing and treating the underlying cause or aggravating factors of the cardiac arrest is fundamental to the management of cardiac arrest in pregnancy. It is important to consider the cause of the cardiac arrest early in the management algorithm. Specific therapy directed at the cause of the cardiac arrest can be lifesaving. It is important to understand the causes of maternal mortality to have an understanding of the unique pathogenic factors that may have precipitated the maternal cardiac arrest. The most common causes of maternal cardiac arrest and mortality are listed in Table 5. Caregivers not routinely involved in high-risk pregnancy may not have experience with the presentation or frequency in which specific diagnoses can result in maternal mortality and cardiac arrest. Therefore, the online-only Appendix includes a specific robust chapter that reviews the causes of maternal mortality and highlights important diagnostic and treatment considerations.

Point-of-Care Instruments

Checklists may help individual responders access temporarily inaccessible cognitive information during periods of intense stress or task saturation. However, the use of checklists during medical emergencies is not consistent, despite the evidence of recurrent cognitive errors on the part of medical providers.[120] Checklists seem particularly well suited to the obstetric domain.[121] One study found that all critical actions for simulated obstetric cardiac arrest were performed only when a cognitive aid reader assisted the team leader.[121] Several groups

Table 5. Most Common Etiologies of Maternal Arrest and Mortality

Letter	Cause	Etiology
A	Anesthetic complications	High neuraxial block
		Hypotension
		Loss of airway
		Aspiration
		Respiratory depression
		Local anesthetic systemic toxicity
	Accidents/trauma	Trauma
		Suicide
B	Bleeding	Coagulopathy
		Uterine atony
		Placenta accreta
		Placental abruption
		Placenta previa
		Retained products of conception
		Uterine rupture
		Surgical
		Transfusion reaction
C	Cardiovascular causes	Myocardial infarction
		Aortic dissection
		Cardiomyopathy
		Arrhythmias
		Valve disease
		Congenital heart disease
D	Drugs	Oxytocin
		Magnesium
		Drug error
		Illicit drugs
		Opioids
		Insulin
		Anaphylaxis
E	Embolic causes	Amniotic fluid embolus
		Pulmonary embolus
		Cerebrovascular event
		Venous air embolism
F	Fever	Sepsis
		Infection
G	General	H's and T's
H	Hypertension	Preeclampsia
		Eclampsia
		HELLP syndrome, intracranial bleed

HELLP indicates hemolysis, elevated liver enzymes, and low platelet count.

have recommended and produced obstetric crisis–specific checklists.[26,103a,122] One such checklist for maternal cardiac arrest is provided in Figure 7. These checklists may include, but are not limited to, contact numbers, critical service lines (eg, transfusion services, neonatal/pediatric teams), locations of necessary equipment (eg, scalpel location), and critical steps in care (eg, prepare to make the incision for delivery). The key factors for optimal use of point-of-care instruments are that teams should be familiar with the content and use of checklists in general and that the checklist should be written specifically for the institution with input from providers who would respond and be involved in rendering care at that institution.

Recommendation

1. **Institutions should create point-of-care checklists to help guide and support critical interventions during obstetric crises (*Class I; Level of Evidence B*).**

Immediate Postarrest Care

It is essential that a multidisciplinary team continue care in the postarrest period. As with all postarrest patients, the pregnant patient who is successfully resuscitated will require thorough assessment, monitoring, and treatment as complications arise. For example, as perfusion improves, bleeding may become a serious issue. If the patient is not delivered, aortocaval compression could precipitate hypotension and rearrest.

Recommendations

1. **If the patient is still pregnant, she should be placed in the full left lateral decubitus position, provided that this does not interfere with additional management issues such as monitoring, airway control, and intravenous access. If the patient is not in full left lateral tilt, manual LUD should be maintained continuously (*Class I; Level of Evidence C*).**
2. **The patient should be transferred to the ICU unless an operation is required (*Class I; Level of Evidence C*).**
3. **Optimal pre-event planning should be ensured as discussed above (*Class I; Level of Evidence C*).**
4. **Multidisciplinary care must continue (*Class I; Level of Evidence C*).**
5. **The cause of the arrest should continue to be considered and treated accordingly (*Class I; Level of Evidence C*).**

Antiarrhythmic Therapy

Postarrest therapy for recurrent life-threatening arrhythmias includes consideration of placement of an implantable cardioverter-defibrillator or medication therapy in the pregnant patient as in the nonpregnant patient[124] (*Class I; Level of Evidence C*). β-Blockers are often used as first-line therapy for a diversity of arrhythmias; they are generally safe in pregnancy (metoprolol is a preferred β-antagonist used in pregnancy)[125] (*Class IIa; Level of Evidence C*). For long-QT syndrome, β-blockers were found to be effective in reducing the incidence of adverse events and therefore highly recommended during pregnancy and the postpartum period[126,127] (*Class IIa; Level of Evidence C*). In general, for recurrent primary ventricular tachycardia and ventricular fibrillation, amiodarone should be considered (*Class IIb; Level of Evidence C*). Evaluation for reversible causes of cardiac arrhythmias should be routine. Thyroid dysfunction, adverse drug effects, electrolyte disturbances, cardiac ischemia, and heart failure should all be identified and corrected when possible (*Class I; Level of Evidence C*).

Call for help **Start CPR**	☐ Call maternal code blue (Time: _____) ☐ Backboard (Time: _____) ☐ IMMEDIATE BLS ☐ AED/defibrillator ☐ Maternal airway equipment ☐ Scalpel/cesarean pack ☐ Assign timer/documenter ☐ Document time of cardiac arrest (Time: _____) ☐ Assign cognitive aid reader/recorder
C Circulation Chest Compressions	☐ Left uterine displacement (manual) (Time: _____) ☐ Hands midsternum ☐ 100 compressions/min (Time: _____) ☐ PUSH HARD, PUSH FAST ☐ Change compressors every 2 minutes ☐ Obtain IV access above diaphragm (Time: _____)
A Airway	☐ Minimize interruptions in chest compressions ☐ Chin lift/jaw thrust if not trauma victim ☐ 100% O_2 at ≥15 L/min (Time: _____) ☐ Use self-inflating bag-mask ☐ Oral airway or ☐ Experienced personnel: intubation with 6.0- to 7.0-mm inner diameter ETT or (Time: _____) ☐ Supraglottic airway (eg, laryngeal mask airway with gastric port) (Time: _____)
B Breathing	☐ If not intubated: 30 compressions to 2 breaths ☐ If intubated: 8–10 breaths/min ☐ Administer each breath over 1 second
D Defibrillate	☐ Pads front and side ☐ AED: analyze/defibrillate every 2 minutes (Time: _____) ☐ Immediately resume CPR for 2 minutes ☐ Prepare for delivery
E Extract Fetus	☐ PMCD started (Time: _____) and ☐ Fetus delivered (Time: _____)

Figure 7. Cognitive aid checklist for cardiac arrest in pregnancy. AED indicates automated external defibrillator; BLS, basic life support; CPR, cardiopulmonary resuscitation; ETT, endotracheal tube; IV, intravenous; and PMCD, perimortem cesarean delivery. Modified with permission from Lipman et al.[103a] Copyright © 2014, International Anesthesia Research Society.

Targeted Temperature Management/Therapeutic Hypothermia

Even when resuscitative efforts are successful in restoring spontaneous circulation, postarrest brain injury often limits the positive outcome for cardiac arrest.[128] One apparent beneficial intervention has been mild postarrest-induced hypothermia, which is based on scientific plausibility and the positive results of 2 randomized, clinical trials published in 2002.[129,130] On the basis of these data, targeted temperature management to 32°C to 34°C (89.6°F–93.2°F) for 12 to 24 hours has been recommended for nonpregnant comatose adult patients with ROSC after out-of-hospital ventricular fibrillation cardiac arrest (*Class I; Level of Evidence B*). There are 2 case reports with favorable maternal and fetal outcomes in which postarrest cooling was instituted in early pregnancy, the fetus was monitored, and emergency cesarean delivery was not necessary.[91,131] There is 1 case report of fetal demise in the setting of targeted temperature management, but in this case, there was a period of unsuccessful resuscitation of 22 minutes before resuscitation by EMS was started.[132] Therefore, data for hypothermia in pregnancy are scant. Pregnancy is thus not an absolute contraindication to targeted temperature management. However, given the lack of data on the use of targeted temperature management after PMCD and the risk of impaired coagulation during the lowering of systemic temperatures, targeted temperature management may be considered on an individual basis after cardiac arrest in a comatose pregnant patient.

Recommendations

1. **Targeted temperature management should be considered in pregnancy on an individual basis (*Class IIb; Level of Evidence C*).**
2. **If used in pregnancy, targeted temperature management should follow the same current protocol as for the nonpregnant patient (*Class IIb; Level of Evidence C*).**
3. **Fetal monitoring should be performed throughout targeted temperature management (*Class I; Level of Evidence C*).**

Fetal Risk of Postresuscitation Intervention

A large number of medications may be used after ROSC is achieved. Unlike in the nonpregnant state, whether medications may adversely affect the fetus must be considered, in addition to the potential compromise caused by circulatory failure, lack of adequate placental perfusion, and impaired oxygen and nutrient exchange between the mother and fetus.

Three major principles guide the decisions made by clinicians at this stage:

1. Maternal well-being is the overriding priority because maternal demise or unfavorable recovery never bodes well for the unborn baby.
2. Embryogenesis is mostly complete by 12 weeks of gestation; hence, even teratogenic drugs (eg, warfarin, phenytoin, corticosteroids) are unlikely to cause malformations if the event occurs after the first trimester.
3. Drugs may cause fetal toxicity rather than teratogenicity in late pregnancy (eg, angiotensin-converting enzyme inhibitors, which can cause fetal renal failure and oligohydramnios).

In general, most therapeutic drugs have a molecular weight of <1000, allowing them to cross the human placenta from the maternal to the fetal circulation. The exceptions are large molecules such as heparin and low-molecular-weight heparin, insulin, and other proteins. However, after 20 weeks of gestation, all biologics that are IgG can cross the placenta by using the Fc transporters. The fact that a drug crosses the placenta is, per se, not a reason for concern because the concentrations of most of these agents do not inflict fetal damage. The risks and benefits of medication use in the postarrest period should be considered on an individual basis.

Assessment of the Newborn

The majority of neonates delivered by PMCD are likely to require active resuscitation[90]; the severity of perinatal depression and the extent of resuscitation may vary. Management of the neonate after PMCD should follow the most current AHA guidelines.[115]

Fetal Assessment (If Undelivered)

In cases when maternal cardiac arrest is treated without delivery of the fetus and the pregnancy is considered potentially viable (minimum, 23 weeks of gestation), continuous fetal heart rate monitoring with cardiotocography should be started as soon as feasible after maternal ROSC and continued until clinical recovery of the mother. The goal for such monitoring is to assess for signs of nonreassuring fetal status (fetal tachycardia, bradycardia, loss of heart rate variability, variable or late decelerations) and to monitor for uterine activity.[29,133] Furthermore, because the fetus is considered to be more sensitive to changes in environment, nonreassuring fetal well-being could be the first sign of deterioration of the maternal clinical condition and may signify impending maternal decompensation. The presence of signs of nonreassuring fetal well-being on fetal heart rate monitoring should prompt an urgent obstetric and medical review because an emergency cesarean delivery may be necessary.

Recommendations

1. **Postarrest assessment of the fetus should include continuous fetal heart rate monitoring (*Class I; Level of Evidence C*).**
2. **Signs of nonreassuring fetal status should prompt a thorough maternal and fetal reassessment (*Class I; Level of Evidence C*).**
3. **Delivery could be considered if signs of nonreassuring fetal status occur (*Class I; Level of Evidence C*).**

Medical-Legal Considerations

Maternal cardiac arrest and the death of the mother, the fetus, or both are traumatic and usually unexpected events. As highlighted throughout this scientific statement, efforts of all those involved in the care of pregnant women should be directed at prevention, identification of high-risk patients, and referral to specialized care for those at risk for adverse events. However, not all events can be prevented, and if cardiac arrest does occur, EMS, institutions, and individual healthcare team members who would be involved in resuscitative efforts must be prepared. Patient safety is strengthened through this type of proactive approach to pregnancy care. One such example of a system-wide approach to pregnancy care has been shown to result in reduced claims.[134] Once a cardiac arrest has occurred during pregnancy, important steps should be taken to review the care received by the patient to improve systems of care going forward. Implementation of a quality incident notification system has shown that this type of program can help identify avoidable adverse outcomes and can be used to improve practices of care.[134,135,135a] The references highlighted above are useful tools for readers to review and potentially improve their own institutional practices.

Recommendations

1. **All cases of cardiac arrest and maternal near miss should be reviewed by the maternal cardiac arrest committee for the hospital (*Class I; Level of Evidence C*).**
2. **Identified deficiencies should be corrected (*Class I; Level of Evidence C*).**

Knowledge Translation Strategy

Knowledge translation, also referred to as dissemination and implementation, has been defined by the National Center for the Dissemination of Disability Research as "the collaborative and systematic review, assessment, identification, aggregation, and practical application of high-quality research by key stakeholders (consumers, researchers, practitioners, policy makers) for the purpose of improving the lives of individuals...."[136] This statement represents an important step in the knowledge translation process: the collaborative filtering of information by experts so that only the most valid and useful knowledge is left.[137] Clinicians are often faced with an unmanageable multitude of primary studies or information of variable quality. Comprehensive syntheses presented in statements such as this one provide a trustworthy and applicable aggregation and appraisal of the existing knowledge. This scientific statement is specifically designed to increase the likelihood of translation and uptake by using reviews of the evidence and providing direct practice recommendations.

Reading this statement cannot be where the knowledge translation process ends. An active knowledge translation strategy, which includes multidisciplinary involvement (customized information), barrier assessment, full leadership

commitment and support,[138] and a variety of ongoing dissemination approaches, is crucial to ensure routine use of evidence-based practices.

There is an entire body of literature on various approaches to ensure the routine use of knowledge, all with variable success, depending on the context and intensity with which they are applied. Some approaches that may be specifically pertinent in the case of maternal cardiac arrest include the following:

- Instituting standardized order sets, a prefilled form of evidence-based orders that can be signed by the lead physician so that there is no confusion as to what needs to be done and no time is lost starting from blank orders.
- Developing a program of mock code drills. Lifelike maternal cardiac arrest scenarios are simulated for all staff at regular intervals to make crisis situations feel more commonplace, to decrease fears and anxiety in the team, to improve communication, and to increase familiarity with resuscitation guidelines. This has been shown to directly improve resuscitation skills performance in both the adult[139] and pediatric settings.[140] Recommendations by the Confidential Enquiries Into Maternal and Child Health of the United Kingdom, The Joint Commission, and others emphasize the use of periodic emergency drills that involve both the obstetric and neonatal teams as a way to practice critical communication skills and to identify occult errors in the system.[141–145]
- Building in an audit and feedback mechanism to collect data during resuscitation situations and to provide constructive feedback to the team to identify key areas of success and areas for improvement.
- Holding case debriefing sessions, which are similar to audit and feedback, to provide an opportunity for the team to talk about how the event went, what worked well and what did not work, and to offer a chance for others who may never have been part of a maternal resuscitation to learn from their colleagues' experience.

The key factor to successful knowledge translation is an active, multifaceted approach that attends to the multidisciplinary nature of health care. A unique consideration for maternal cardiac arrest is the relatively low volume of events any one clinician will be involved in treating; this can be detrimental to the impact of knowledge translation efforts. However, strategies such as case discussions and mock drills can be very effective in ensuring that the team is ready when a pregnant patient in cardiac arrest comes through the door.

Recommendation

1. **All stakeholders/specialties involved in maternal resuscitation should form maternal cardiac arrest committees within each institution to ensure guideline implementation, training, and institution of mock code drills (*Class I; Level of Evidence C*).**

Maternal Resuscitation Training

An analysis of simulated maternal cardiac arrests involving participants trained in ACLS suggests that performance during an actual event may be suboptimal.[31] AHA ACLS courses do not routinely emphasize obstetric-specific interventions. In addition, CPR courses tend to stress knowledge and technical skills over behavioral/communication skill sets.[146,147] The rarity of maternal cardiac arrest[9] implies that participants in CPR courses could benefit from a review of obstetric-specific interventions. Although many obstetric providers are not trained in ACLS, knowledge decay and gaps in fund of information specific to the obstetric domain exist even among those who are.[7,8,148,149]

Maternal cardiac arrest demands a multidisciplinary response that requires unique coordination among teams. Such coordination is predicated on clear and succinct communication.[150] Data from The Joint Commission suggest that communication failures are the root cause of neonatal morbidity and mortality in 70% of cases[142] that occur in the obstetric domain. In addition, in an analysis of preventable maternal mortalities, facility factors contributed significantly to the fatal outcome in 75% of cases.[142a] This suggests that lack of institutional preparedness can decrease or even negate the ability of highly functioning staff to render optimal care. Moreover, ACLS courses cannot hope to address the identification and correction of institutional system issues; this suggests that multidisciplinary maternal cardiac drills are important components of institutional preparedness. Maternally oriented CPR courses are likely more relevant to the learning needs and goals of obstetric staff; these courses have been developed by groups in the United Kingdom and the United States.[94,141,151]

Recommendations

1. **Periodic multidisciplinary drills may help institutions optimize safety systems (*Class IIa; Level of Evidence C*).**
2. **Specific courses on maternal resuscitation should be available to staff if not available outside local institutions (*Class IIa; Level of Evidence C*).**
3. **The future goal should be to have national and international programs in maternal resuscitation (*Class I; Level of Evidence C*).**

Future Considerations

The questions remaining unanswered in relation to both treatment and outcomes (both maternal and neonatal) should prompt the establishment of a central registry of cases of maternal near miss and cardiac arrest.[152] Maternal cardiac arrest represents the tip of the iceberg of near-miss maternal complications. Understanding why in some cases the pregnant woman reached a state of arrest may require root-cause analysis.

Multiple issues related to maternal resuscitation remain. Some could be addressed by simple analyses of database information, as mentioned above, for example, whether PMCD improves the rate of ROSC in accordance with the theory of aortocaval decompression, the optimal timing of cesarean delivery for both maternal and neonatal outcomes, and whether these outcomes are as good as those suggested in the most recent literature.

Current guidelines advocate placement of the woman in the full supine position with manual uterine displacement to alleviate aortocaval compression. However, there are sparse data on the impact of the anterior and left lateral displacement

of the heart during pregnancy on the probability of hand placement on the cardiac apex, and data on the effect of manual uterine displacement or pelvic wedging on the position of the heart and upper back are also scant. Finally, both maternal and neonatal long-term neurological status and functional status after resuscitation remain enigmas.

Recommendations

1. **A central registry of cases of maternal near miss and cardiac arrest with documentation of both process and outcome should be established (*Class I; Level of Evidence C*).***
2. **A standardized training course in maternal resuscitation should be developed (*Class I; Level of Evidence C*).**

Conclusion

Maternal cardiac arrest is a complex clinical scenario. Resuscitation of the pregnant woman involves multispecialties and complex care decisions. It is unlikely that prospective studies on maternal resuscitation will provide additional data in the future, despite the fact that clinical equipoise remains for most treatments in this situation. Although maternal cardiac arrest is rare, it appears to be increasing in frequency. The number of high-risk women undergoing pregnancy is on the rise, as is the rate of severe complications related to pregnancy (including cardiac arrest).[153] The writing group acknowledges that scientific evidence for management of cardiac arrest in pregnancy is lacking.[154,155] The majority of the writing group's recommendations are Level of Evidence C, which underscores the need for further research. This expert panel of authors has applied a multispecialty, expert approach to develop these recommendations through experience, previous publication of direct and indirect data, and expert opinion to reach consensus. The writing group recognizes that without an organized approach to maternal cardiac arrest, chaos will likely ensue. Therefore, the development and implementation of the recommendations contained in this document will be beneficial to maternal care. This scientific statement will help healthcare providers be prepared and provide the best possible care for a maternal cardiac arrest. The newly developed in-hospital and out-of-hospital BLS and ACLS algorithms should be the backbone of the response plan to a maternal cardiac arrest. Special attention should be paid to manual LUD, the difficult airway, and appropriate use of PMCD. Lifesaving interventions such as defibrillation and medications should not be withheld in the setting of pregnancy. The healthcare community must be proactively prepared to respond to a maternal cardiac arrest. A maternal cardiac arrest committee must be formed at every institution, and emergency response plans specific to each institution must be developed and implemented. The maternal cardiac arrest committee would link adult resuscitation teams with obstetrics, neonatology, intensive care, anesthesia, the emergency department, and EMS and involve the allied healthcare teams, including nursing, respiratory therapy, social work, and clergy, as available and necessary to implement guidelines and recommendations. Training, mock code drills, and review of cases should become routine. This scientific statement has addressed all aspects of maternal resuscitation: prearrest care, BLS, ACLS, and postarrest care. In addition, the online-only Appendix has a robust chapter on the causes of maternal mortality and cardiac arrest in pregnancy with specific therapies to consider. The statement has also provided readers with resources, point-of-care instruments, and algorithms that will be useful to consider when developing institutional response plans.

*For those interested in joining the registry, please email us at eccscience@heart.org.

Disclosures

Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
Farida M. Jeejeebhoy	University of Toronto, William Osler Health System	Rescu Project grant (all funds provided through nonindustry funds)*	None	None	None	None	None	None
Julie Arafeh	Stanford University	None	None	None	None	None	None	None
Clifton W. Callaway	University of Pittsburgh	NHLBI (Research Outcome Consortium)†	None	None	None	None	None	None
Brendan Carvalho	Stanford University	None	None	None	None	None	None	None
Katie N. Dainty	St. Michael's Li Ka Shing Knowledge Institute	None	None	None	None	None	None	None
Sharon Einav	Shaare Zedek Medical Center (Jerusalem, Israel)	Covidien*; Zoll*; Diasorin*; Israel Ministry of Health†; National Institute for Health Policy Research†	None	None	None	Patent on capnography in weaning from mechanical ventilation*	None	European Society of Anaesthesia*; American College of Chest Physicians*
Amish Jain	Mount Sinai Hospital	None	None	None	None	None	None	None
Jose Joglar	UT Southwestern	None	None	None	None	None	None	None
Vern L. Katz	Oregon Health Sciences University	None	None	None	None	None	None	None
Gideon Koren	University of Toronto	Duchesnay Inc†	None	None	None	None	Duchesnay Inc*; Novartis*; Bayer*	None
Stephen E. Lapinsky	University of Toronto	None	None	None	None	None	None	None
Steve Lipman	Stanford University	None	None	None	None	None	None	None
Jill M. Mhyre	University of Arkansas for Medical Sciences	None	None	None	None	None	None	None
Richard L. Page	University of Wisconsin	None	None	None	None	None	None	None
Carole A. Warnes	Mayo Clinic Rochester	None	None	None	None	None	None	None
Rory Windrim	University of Toronto	None	None	None	None	None	None	None
Carolyn M. Zelop	The Valley Hospital	None	None	ACOG annual meeting*	None	None	UpToDate*	None
Staff								
Russell E. Griffin	American Heart Association	None	None	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

Reviewer Disclosures

Reviewer	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
Judith Finn	Curtin University	NHMRC (director of the Australian Resuscitation Outcomes Consortium [Aus-ROC], an NHMRC Center of Research Excellence)*	None	None	None	None	None	None
Kathryn J. Lindley	Washington University in St. Louis	None	None	None	None	None	None	None
Michael R. Sayre	University of Washington	None	None	None	None	None	None	None
John T. Sullivan	Northwestern University	None	None	None	Donohue Brown Mathewson & Smyth*	None	None	None

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

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Appendix:
Etiology of Maternal Cardiac Arrest and Mortality

The most frequent causes of maternal cardiac arrest include bleeding, heart failure, amniotic fluid embolism (AFE), and sepsis,[1] and the most common causes of maternal mortality that may also lead to cardiac arrest include cardiac disease, sepsis, preeclampsia/eclampsia, hemorrhage, cerebrovascular events, AFE, complications from anesthesia, and thrombosis/thromboembolism.[2,3] However, data from the British Centre for Maternal and Child Enquiries (CMACE) report illustrate that the approach to the etiology of a maternal cardiac arrest must be more inclusive,[2] and a broader approach to the possible etiology of a maternal cardiac arrest is necessary in order to have the best chance to identify and correctly treat causative factors and thereby give the patients, mother and baby, the best chance of survival. It should also be recognized that women are entering into pregnancy with more comorbidities and risk factors than seen historically.[2,4] During pregnancy, the chance exists for the additional development of maternal and fetal complications, which may result in acute deterioration. Therefore, the cause of the maternal cardiac arrest may often be multifactorial. Diagnostic testing and treatment of disease is almost always more complicated in pregnancy, given that there are 2 patients to consider, the mother and the baby. Therefore, rescuers should have a basic understanding of diagnostic approaches and treatment considerations. This appendix includes a discussion of the important etiologies of maternal mortality (listed in table 1), and, where appropriate, diagnostic testing and treatment recommendations are discussed.

Chronic Health Problems That Exist Before Pregnancy

Chronic health problems that exist before pregnancy can put the pregnant woman at risk for cardiac arrest and mortality. A list of all possible health problems and concerns with pregnancy is outside the scope of this report. However, important examples are discussed below. It is important that pregnant women who have preexisting chronic health problems receive specialized care during their pregnancy and into the postpartum period.[2]

- Women with chronic kidney disease have more adverse maternal outcomes, including maternal mortality, compared with maternal outcomes of those without chronic kidney disease.[5]
- Neurologic disorders such as epilepsy are often more difficult to manage during pregnancy because the physiological changes of pregnancy often affect antiepileptic drug levels.[6,7] Medication dosages may need to be adjusted during pregnancy to achieve therapeutic levels. CMACE data report deaths from epilepsy during pregnancy; the majority of these women were not referred to a neurologist and therefore likely did not have their medication dosages assessed.[2]
- Lung diseases such as asthma can result in death during pregnancy.[2] Asthma does not get worse during pregnancy; however, it has been found that women and caregivers often fear adverse effects of asthma medications on the fetus, which results in inadequate treatment during pregnancy, leading to worsening of the condition and sometimes death.[2] Specialized care for asthma during pregnancy will help prevent this scenario.

Obesity

At least 30% of all pregnant women are obese, and obesity is a risk factor for cardiopulmonary arrest. Obesity increases the risk of death from specific causes of maternal mortality (eg, fatal thromboembolism)[2] and the occurrence of other conditions associated with cardiopulmonary arrest, including cardiac disease, preeclampsia, peripartum hemorrhage, and perioperative infection leading to sepsis.[8]

Morbid obesity (body mass index 40 or greater) further complicates resuscitation because critical interventions (eg, mask ventilation, intubation, uterine displacement, peripheral IV access, chest compressions, perimortem cesarean delivery) are more technically challenging. Basic monitoring such as noninvasive blood pressure measurement may not be possible with standardly available cuff sizes, and measurements may be inaccurate even with the largest cuff sizes because of improper cuff fit. Lifting and positioning the morbidly obese patient may not be possible, may result in further injury to patient and/or

staff, or may require a specialized lift team that may not be immediately available. These factors may negatively affect the ability of responding teams to render optimal care during maternal cardiac arrest.

Table 1. Most Common Etiologies of Maternal Arrest and Mortality

Letter	Cause	Etiology
A	Anesthetic complications	High neuraxial block
		Hypotension
		Loss of airway
		Aspiration
		Respiratory depression
		Local anesthetic systemic toxicity
	Accidents/trauma	Trauma
		Suicide
B	Bleeding	Coagulopathy
		Uterine atony
		Placenta accreta
		Placental abruption
		Placenta previa
		Retained products of conception
		Uterine rupture
		Surgical
		Transfusion reaction
C	Cardiovascular causes	Myocardial infarction
		Aortic dissection
		Cardiomyopathy
		Arrhythmias
		Valve disease
		Congenital heart disease
D	Drugs	Oxytocin
		Magnesium
		Drug error
		Illicit drugs
		Opioids
		Insulin
		Anaphylaxis
E	Embolic causes	Amniotic fluid embolus
		Pulmonary embolus
		Cerebrovascular event
		Venous air embolism
F	Fever	Sepsis
		Infection
G	General	H’s and T’s
H	Hypertension	Preeclampsia
		Eclampsia
		HELLP syndrome, intracranial bleed

HELLP indicates hemolysis, elevated liver enzymes, and low platelet count.

A—Anesthetic Complications

Anesthesia-related maternal mortality has decreased nearly 60% over the past few decades[9]; however, a significant proportion (1% to 4%) of maternal deaths are still directly related to complications from anesthesia.[1,10-12] Although case-fatality rates for general anesthesia are decreasing, rates for regional anesthesia are increasing.[9] Many of these complications are avoidable and potentially reversible. Although complications from anesthesia accounted for 8% of maternal cardiac arrests in the 1998-2011 US Nationwide Inpatient Sample, 82% of these women survived to hospital discharge.[1]

High Neuraxial Block and Hypotension

Epidemiology: With increasing use of neuraxial anesthetic techniques in the obstetric setting,[9] neuraxial anesthesia–related complications such as high spinal blocks have increased. In an analysis of closed claims related to regional anesthesia and analgesia in England, approximately half were related to obstetric cases.[13]

Pathophysiology: The innervation to the diaphragm is C3, C4, and C5; therefore, high spinals will paralyze the diaphragm if the cephalad extent of the block reaches or exceeds this level. Paralysis will result in a respiratory arrest with the potential for full cardiopulmonary arrest if ventilation is not immediately instituted. In addition, high spinal blocks cause significant sympathectomy. As a result, decreased systemic vascular resistance may precipitate marked hypotension. Persistent severe hypotension may result in cardiac arrest if left untreated. Severe sinus bradycardia or arrest during spinal or epidural anesthesia is also described, presumably due to a Bezold-Jarisch reflex and/or blockage of cardiac accelerator fibers at T1-4 with unopposed vagal tone.[14,15]

Diagnosis: Early recognition and immediate institution of resuscitative measures is essential to optimize outcomes.[14,15] Spinal-induced cardiopulmonary arrest should be suspected if it occurs soon after administration of spinal anesthesia or after epidural bolus (because of the potential for intrathecal migration of the catheter and/or high block from

the epidural itself). Underlying and contributing causes for cardiac arrest complicating spinal anesthesia should always be investigated; one case report after an investigation of cesarean sections revealed dilated cardiomyopathy related to pregnancy.[16]

Treatment: The liberal use of phenylephrine, ephedrine (progressing to epinephrine and atropine if necessary), and vasopressin; repeated, frequent blood pressure measurement; aggressive intravascular fluid repletion; inferior vena caval decompression; and external or transvenous pacing may help prevent the hypotension associated with high or total spinal but will not prevent high or total spinal from occurring and causing paralysis of the diaphragm. If high or total spinal occurs, management should focus on oxygenation, intubation, and ventilation if apnea, loss of consciousness, or both occur; administration of vasopressors to increase systemic vascular resistance (early and aggressive administration of epinephrine, vasopressin, ephedrine, and phenylephrine); fluid resuscitation with colloid or crystalloids; adequate left uterine displacement to restore the effective circulating blood volume; inotropes (epinephrine and ephedrine) and chronotropes (atropine) if bradycardia presents; and chest compressions as necessary.

Loss of Airway and Aspiration

Epidemiology: The risk of loss of maternal airway and gastric aspiration has decreased with increasing use of regional anesthetic techniques.[9] However, airway-related problems still cause maternal death.[9-12] Aspiration pneumonitis accounted for 7% of maternal cardiac arrests in a 1998-2011 US Nationwide Inpatient Sample.[1] The reported survival to hospital discharge during maternal cardiac arrests associated with aspiration pneumonitis is 83%.[1]

Pathophysiology: The physiology of pregnancy results in increased edema and friability of the oropharyngeal mucosa as well as increased acidity of gastric contents and decreased lower esophageal sphincter tone. These physiological changes may synergize and result in more technically challenging airway control. If aspiration occurs in an unprotected airway, highly acidic gastric contents may enter the pulmonary tree and injure the lung parenchyma, causing pneumonitis.

Diagnosis: Hypoventilation, airway obstruction, or both have been reported during induction, emergence, extubation, or recovery and often in the setting of inadequate anesthesia supervision or expertise. Aspiration may be diagnosed by the presence of gastric contents in the oropharynx and may be associated with hypoxia.[10,11]

Treatment: Common strategies used to prevent aspiration include the use of nonparticulate antacids to temporarily (about 15 minutes) neutralize acidic pH before induction of anesthesia and agents (such as metoclopramide) that increase lower esophageal sphincter tone. The quality of the evidence was poor, but the findings suggest that the combination of antacids plus H2 antagonists was more effective than no intervention and superior to antacids alone in preventing low gastric pH.[17] Meticulous airway examination, documentation, and communication, in conjunction with proper equipment (videolaryngoscopic capability, supraglottic devices with orogastric ports, bougies, etc), and the presence of a difficult-airway cart and experts in airway management all confer added safety to control of the airway in the parturient. If pulmonary aspiration of gastric contents is suspected, management strategies include use of an endotracheal tube and mechanical ventilation with 100% oxygen and positive end-expiratory pressure 5 cm H_2O; suction airway; bronchoscopy if necessary; and bronchodilators, fluids, and inotropes as needed.[18] Steroids and antibiotics should not be used in the acute phase, because there is no evidence of an improved outcome.[18]

Respiratory Depression

Epidemiology: Although very rare, maternal respiratory depression and arrest is a potentially serious risk after neuraxial (epidural and intrathecal) or intravenous opioid administration.[19] In 1 report, 73% of these patients died or experienced permanent brain damage after respiratory depression involving neuraxial opioids.[20] Early recognition and effective management often result in good outcomes.[21]

Pathophysiology: Opioids may cause hypoventilation and/or apnea, particularly when administered by multiple routes (eg, oral, intravenous, neuraxial) to high-risk patients

(eg, obstructive sleep apnea), although opioid-induced respiratory arrest may occur in any patient.

Diagnosis: Opioid-induced respiratory arrest in the setting of labor should be suspected if respiratory arrest occurs within recent (within 30 minutes) administration of intravenous epidural or intrathecal neuraxial opioids.[19,21,22] Delayed (6 to 18 hours postdose) respiratory depression after use of neuraxial morphine (due to rostral spread in cerebrospinal fluid and brainstem penetration) has also been described.[19,23] Continuous infusion of short-acting opiates has also been associated with maternal apnea.[24] Therefore, monitoring for this potential anesthetic complication should be of adequate duration, depending on the opioid used.[25]

Treatment: Prevention of respiratory depression and arrest from opioids depends on appropriate monitoring of the respiratory rate and capnography, widespread education of staff, readily available reversal agents (eg, naloxone), and ventilation equipment. In the setting of suspected opioid-induced respiratory depression, treatment protocols should focus on rapid provision of supplemental oxygen and immediate administration of the opioid reversal agent naloxone. If naloxone fails to reverse respiratory depression or arrest, prompt bag-mask ventilation and/or endotracheal intubation should be performed. Treatment and intensive monitoring should be maintained until signs and symptoms of respiratory depression have resolved.[26]

Local Anesthetic Systemic Toxicity

Epidemiology: With the increasing use of neuraxial anesthesia in obstetrics, one possible anesthetic-related etiology for cardiac arrest is local anesthetic systemic toxicity (LAST).[9]

Pathophysiology: Pregnancy may enhance sensitivity to local anesthetics, and LAST-induced cardiac toxicity may be particularly resistant to conventional resuscitative interventions.

Diagnosis: LAST-induced cardiac arrest should be suspected if an arrest occurs soon after an epidural local anesthetic "top-up."[27] If LAST-induced cardiac arrest is suspected, lipid emulsion therapy should be given in addition to standard resuscitative measures.[28,29]

Treatment: There are limited data to elucidate if the lipid emulsion dosage needs to be altered in the pregnant patient; therefore, dosing and timing of lipid emulsion therapy during resuscitation of pregnant patients should follow standard algorithms (Table 2).[30,31] If LAST-induced cardiopulmonary arrest is refractory to standard resuscitative measures with lipid emulsion, cardiopulmonary bypass or extracorporeal membrane oxygenation (if available) should be considered as a temporizing intervention.[32] Although the fetal risks of lipid emulsion therapy in this setting are largely unknown, lipid administration for parenteral nutrition in cases of severe hyperemesis gravidarum appears safe for the fetus.[33] The potential maternal benefit of lipid therapy for LAST-induced cardiopulmonary arrest outweighs any theoretic fetal risks.

Table 2. Suggested Lipid Emulsion Dosing Guidelines

Protocol	Dose	Comments
Initial bolus	1.5 mL/kg IBW	100 mL for a person weighing 70 kg
Maintenance infusion	0.25 mL/kg IBW per minute	Continued for at least 10 min after ROSC
Ongoing administration *if ROSC is not attained*	Re-bolus with 1.5 mL/kg IBW and increase maintenance infusion to 0.5 mL/kg IBW per minute	Upper limit is ~10 mL/kg lipid emulsion in first 30 min

IBW indicates ideal body weight; and ROSC, return of spontaneous circulation.
Use 20% lipid emulsion (propofol is not a substitute for lipid emulsion; insufficient lipid content and cardiovascular depressant effects).

A—Accidents/Trauma

Trauma/Suicide in Pregnancy

Epidemiology: One of the largest single causes of maternal mortality is trauma.[34-38] In the developed world, trauma is the leading cause of maternal mortality. Unintentional injury (primarily motor vehicle accidents), homicide, and suicide are the 3 leading components of fatal trauma. In many parts of the United States, homicide from domestic abuse is

more common than fatal motor vehicle accidents.[37,39,40] Suicide represents up to 10% of maternal mortality but may be decreased in pregnancy compared with nonpregnant controls.[39,41] Approximately 1% to 4% of pregnant women are admitted for treatment of traumatic injury, yet there are few educational strategies targeted toward prevention/management of maternal trauma. Pregnancy should be tested for in all trauma cases involving women of childbearing age. Use of illicit drugs and alcohol, domestic abuse, and depression are all important contributors to maternal trauma; thus, a high index of suspicion should be maintained when treating young injured women.

Pathophysiology: Maternal death is associated with penetrating injury, high injury severity score, and head injury. Pregnancy-related morbidity occurs in about 25% of cases and may include placental abruption, uterine rupture, preterm delivery, and need for cesarean delivery.

Diagnosis: Diagnosis is performed by physical examination, monitoring of uterine activity and fetal heart rate, and ultrasound evaluation (depending on gestational age). Fetal-maternal hemorrhage (detected with a Kleihauer-Betke test) complicates 10% to 30% of cases.

Treatment: The response of the resuscitation team to traumatic injury includes an expedited use of perimortem cesarean delivery. If the mother has an obviously lethal injury, such as a fatal gunshot wound to the head, then continued maternal resuscitation is not indicated after the mother arrives in the emergency department. Emergency cesarean delivery should be performed. Considerations for immediate cesarean delivery include the extent of the injury and the time from presumed cardiac arrest until arrival in the emergency department. In general, >30 minutes of absent uterine blood flow is not associated with fetal survival. However, in many situations, this information may not be immediately obvious. Other considerations for immediate cesarean delivery include fetal age and the extent of maternal injury. Each situation is different. In maternal suicide, the means of the suicide may direct resuscitative efforts. A study of the California birth database found drug overdose to be the most common means of maternal suicide

attempts.[42] Rh-negative patients should receive Rh-D immunoglobulin within 72 hours of injury. Anesthetic management should be directed toward maternal oxygenation and perfusion. Airway management includes prevention of aspiration, expert help, and insertion of a smaller-than-usual endotracheal tube. Drugs (choice and dose) should be adapted primarily to suit maternal injuries and, when possible, pregnancy. If pregnancy is confirmed, gestational age should be assessed while providers adhere to advanced trauma life support guidelines. Treating the mother appropriately is beneficial for both mother and fetus. The only guideline modifications include provision of supplemental oxygen, preference for intravenous access above the diaphragm, and left lateral positioning whenever possible.

B—Bleeding

Epidemiology: Hemorrhage complicates approximately 3% to 4% of all births, but fewer than 1 in 1000 women with hemorrhage also experience cardiac arrest.[1] Regardless, antepartum or postpartum hemorrhage accounts for 38% of all cardiac arrests during hospitalization for delivery.[1]

Pathophysiology: Maternal mortality reviews have suggested that hemorrhagic cardiac arrest often follows delays in recognition, treatment, or escalation of care for women with obstetric hemorrhage.[2,43,44] In some cases, hemorrhage may be concealed (eg, abruption, retroperitoneal hemorrhage). The bleeding pregnant patient can lose 1500 mL before any clinical manifestations of compromise.[45] Occasionally, catastrophic hemorrhage can present abruptly or overwhelm standard therapies (eg, amniotic fluid embolism). Although 80% of obstetric hemorrhage may be caused by uterine atony, retained placenta, including the spectrum of morbidly adherent placentation (ie, placenta accreta, increta, and percreta), is an important etiology of massive hemorrhage and peripartum hysterectomy and has been linked to the escalation of cesarean birth rates.[46,47] Uterine infection (eg, chorioamnionitis) has also been associated with unremitting uterine atony that requires peripartum hysterectomy.[48] Additional etiologies of massive maternal hemorrhage include abruption, uterine rupture, uterine inversion, placenta previa, and coagulation defects.[49] Other risk factors for peripartum obstetric hemorrhage include

prolonged labor, episiotomy, preeclampsia, operative delivery, and uterine overdistention (ie, multiple gestations and polyhydramnios).[50] Finally, vascular catastrophe is a rare but highly lethal cause of hemorrhagic arrest (eg, aortic rupture, splanchnic arterial rupture) that appears to be more common in pregnancy (see aortic dissection in "Cardiovascular Causes").[51]

Hemorrhage typically causes hypovolemic arrest. Alternatively or additionally, blood product transfusion may generate hypocalcemic or hyperkalemic arrest, anaphylactic reaction to blood products, or respiratory arrest as a result of resuscitation-induced pulmonary or airway edema.

Diagnosis: Rescuers need to perform concurrent resuscitation and evaluation to diagnose the source of bleeding, which may guide specific intervention and treatment. History and physical examination may also help reveal the etiology. Transabdominal ultrasound at the bedside may be beneficial in localizing the diagnosis. In the arrest patient with a closed abdomen, transabdominal ultrasound may be used to diagnose concealed hemorrhage (retroperitoneal hemorrhage, hepatic rupture) and localize the source of bleeding. The Focused Assessment with Sonography in Trauma protocol should be combined with comprehensive uterine evaluation (eg, endometrial stripe, signs of partial uterine inversion). Serial arterial blood gas analysis may help identify acid base and electrolyte abnormalities as well as abnormalities of gas exchange. Hematologic measurements, including the hematocrit, platelet count, and fibrinogen level may not correlate acutely with the degree of blood loss, so transfusion should be started when hemorrhage is suspected clinically rather than waiting for laboratory results. Finally, hemostatic monitoring with thromboelastography or thromboelastometry may facilitate diagnosis of coagulopathy.

Treatment: To prevent cardiac arrest, include obstetric hemorrhage protocols that facilitate a multidisciplinary approach to ensure timely recognition and treatment to stop the bleeding and ensure end-organ perfusion. Uterotonics, uterine evacuation, and uterine tamponade constitute early intervention. Hemodynamic and hemostatic resuscitation

become increasingly important as the quantity and velocity of blood loss increase. Nevertheless, patient survival ultimately depends on the timely escalation of surgical interventions to control the source of bleeding.

Use of massive transfusion with appropriate proportions of packed red blood cells and fresh frozen plasma and cryoprecipitate requires early activation.[52] Aggressive hemostatic and volume resuscitation requires a large multiprofessional team of clinicians and careful coordination with the blood bank, clinical lab, and clinical pharmacy. Institutional massive transfusion protocols can accelerate and enhance access to large volumes of blood products and timely clinical lab turnaround. Coagulopathy is best treated by product replacement and maintenance of normothermia. Early antifibrinolytic therapy (eg, tranexamic acid) has been shown to increase survival in patients with traumatic hemorrhage, and although currently there is insufficient evidence to confirm this benefit in postpartum hemorrhage,[53] there is no evidence of harm to the maternal patient. In contrast, no trials have been attempted to evaluate the benefit of recombinant factor VIIa, a drug that has been associated with serious thrombotic complications in survivors. Exploratory laparotomy with uterine compression sutures, pelvic vascular ligation, and/or vascular embolization may provide successful treatment. Hysterectomy may be required when other measures have been unsuccessful. Exploratory laparotomy may allow ligation of vascular bleeding pedicles and gain access to the uterus. Compression sutures of the uterus and serial pelvic vessel ligation can decrease blood loss. If unsuccessful, hysterectomy should be performed. Pledgeted sutures may help achieve hemostasis when suturing highly friable vascular beds.[54]

Alternatively or additionally, abdominal packing may be necessary to temporarily control bleeding during resuscitation. Vascular radiological embolization may be a viable adjunct, but this would not be the modality of choice during cardiac arrest. It may be helpful as a postresuscitation maneuver in a more stabilized but critically ill patient in whom repetitive surgical procedures should be avoided. In the event of cardiac arrest, a vertical abdominal skin incision will facilitate rapid surgical maneuvers. Examination of the liver and retroperitoneal space for hemorrhage may be important if the cause of

cardiac arrest is unknown. The vertical incision also opens the abdomen for manual compression of the aorta, which has been described as a lifesaving temporizing maneuver for women with catastrophic pelvic hemorrhage.[55]

Both hyperkalemic and hypocalcemic arrest are best treated with calcium chloride and chest compressions. In addition, hyperkalemia should be treated with drugs that shift potassium into cells (eg, sodium bicarbonate, insulin and dextrose, and a β_2-agonist), followed by therapies that accelerate potassium clearance (eg, diuretics, dialysis). Insufficient and difficult peripheral venous access may be rescued with central line insertion or intraosseous needle insertion; any fluid, blood product, or medication can be administered by the intraosseous route while additional venous access is secured.[56] Postresuscitation hypothermia instituted for neuroprotection may exacerbate coagulopathy.

C—Cardiovascular Causes

Cardiac Risk Stratification

In the presence of maternal cardiac disease, the hemodynamic changes of pregnancy may result in decompensation or death of the mother or fetus. In western civilizations, most maternal cardiac disease is congenital in origin, due in large part to the success of cardiac surgery in the past few decades. This may cause significant maternal morbidity but not necessarily mortality. Predictors of maternal cardiac events have been studied largely retrospectively, and predictors of maternal cardiac complications have been identified (Table 3). There are significant limitations in the use of risk scores to predict maternal cardiac events because many studies are retrospective, may have different definitions of a specific lesion (heart failure, for example), and are highly dependent on the populations studied. The study by Siu et al[57] reviewed patients with both congenital and acquired heart disease, whereas the study of Drenthen et al[58] studied only patients with congenital heart disease. Known important risk factors such as pulmonary arterial hypertension and aortic dilatation were not identified in either of these studies and thus are underrepresented in these cohorts. Therefore, the use of predictive risk scores should be only a part of a prepregnancy assessment. Patients with Eisenmenger syndrome, for

example, have a maternal mortality approaching 30% and as such are usually counseled to avoid pregnancy.

The World Health Organization categorizes maternal risk as grades I to IV. Patients in class III are at significant risk of maternal mortality or severe morbidity. Patients in class IV are at extremely high risk of severe morbidity or mortality; for these patients, pregnancy is contraindicated.[59,60] Other pathologies included in class III (not mentioned in Table 1) are a systemic right ventricle, Fontan circulation, and an aorta of 40 to 45 mm in Marfan syndrome and 45 to 50 mm with a bicuspid aortic valve. Patients in class IV include those with pulmonary arterial hypertension of any cause, prior peripartum cardiomyopathy (PPCM) with any residual impairment of ventricular function, native severe coarctation, an aorta >45 mm in Marfan syndrome, and an aorta >50 mm with a bicuspid aortic valve.

Cardiac Disease in Pregnancy

Cardiac disease remains the number 1 cause of maternal mortality in developed countries, albeit a relatively rare event.[2] Leading causes include sudden adult death syndrome, aortic dissection, myocardial infarction, ischemic heart disease, and PPCM. In this context, current trends in lifestyle, including the increasing prevalence of metabolic syndrome, diabetes, smoking, hypertension, and advanced maternal age, must be taken into consideration because they undoubtedly contribute to the growing incidence of ischemic heart disease in pregnancy. In the CMACE study,[2] from 2006 to 2008, 30 of 50 women (60%) who died from cardiac disease were overweight or obese, with half having a body mass index ≥30.

Myocardial Infarction

Epidemiology: Myocardial infarction and ischemic heart disease are important causes of maternal death, with most infarcts occurring in the period 6 weeks postpartum.[61] Pregnancy is a risk factor for acute myocardial infarction (AMI), increasing risk 3- to 4-fold in comparison with the nonpregnant state. Maternal age adds to this risk, so that risk of pregnancy-related AMI is 30 times higher in women >40 years of age versus those <20

years of age.[59] AMI is more common in multigravidas (66%),[62] and approximately three-quarters of these involve the anterior wall. Almost half of acute infarcts are related to coronary dissection; others are related to coronary atheroma, reflecting the impact of lifestyle factors such as increasing maternal age, obesity, and smoking. Indeed, in the CMACE study,[2] all women who died from ischemic heart disease had identifiable risk factors. A US population–based study reported that age >35 years and black race were significant risk factors for pregnancy-related myocardial infarction, with odds >5 times higher for black women 35 years of age and older. Multivariable analysis, however, eliminated race as a risk factor, suggesting that black women have an increased prevalence of other risk factors.[63] Independent risk factors include chronic hypertension, diabetes, advanced maternal age, and eclampsia.[61] Smoking may increase risk 8-fold. Thrombophilia is also a risk factor, which is not surprising in view of the hypercoagulability of pregnancy, which is further increased in the presence of thrombophilia. Women diagnosed during the prenatal period may have a higher mortality and more pregnancy complications.[61] The risk of death from AMI in pregnancy has been reported to be as high as 37%, but recent data suggest the risk is about 5%.[63] This may be related, in part, to the rapidity with which patients with acute coronary syndromes are transported to the catheterization laboratory in the contemporary era and the widespread use of percutaneous coronary intervention (PCI). Mortality appears twice as high in women diagnosed with AMI in the peripartum period compared with the antepartum or postpartum period.[62]

Pathophysiology: Delay in diagnosis and treatment is common because chest pain in pregnancy is common and may reflect reflux disease, with a failure to perform an electrocardiogram to facilitate diagnosis and, where PCI is unavailable, a failure to administer thrombolytic therapy because of the concern for hemorrhage. Failure to proceed rapidly to open the occluded coronary artery either by PCI or thrombolysis is the most common cause of cardiac arrest with ventricular tachycardia or fibrillation. Cardiogenic shock may also occur due to acute ventricular failure with low cardiac output.

Diagnosis: The criteria for diagnosis of AMI in pregnant women are generally the same as in nonpregnant patients and consist of symptoms, electrocardiographic changes, and cardiac markers, although patients may initially have normal troponin levels. Nonetheless, elevated troponin levels should heighten the consideration of AMI as the etiology. Echocardiography is safe and can be used in a timely way to assess regional wall motion abnormalities. The differential diagnoses in this context are aortic dissection, acute pulmonary embolism, and preeclampsia; thus, appropriate investigations for a woman presenting with chest pain should include an electrocardiogram, chest x-ray, troponin levels, an echocardiogram (which may include a transesophageal echocardiogram, depending on available expertise), computed tomography, and/or magnetic resonance imaging.

Treatment: When the diagnosis of AMI is suspected, an invasive approach with prompt transfer to a cardiac catheterization laboratory for consideration of PCI is preferable to thrombolysis because coronary angiography will also diagnose coronary dissection. Morphine may be used for pain control and has no teratogenic effects. β-Adrenergic blocking agents generally can be used safely, as can antiplatelet therapy with low-dose aspirin (81 mg/d). Thienopyridine derivatives such as clopidogrel and ticlopidine may be used when necessary, but data about the safety of these drugs in the fetus are sparse. Ideally, clopidogrel should be discontinued for 1 week before regional anesthesia or cesarean section. Inotropic support may be necessary if cardiac output is impaired. In the context of profound hemodynamic instability, inotropic support may be administered and an intra-aortic balloon pump may be implanted concomitantly with the catheterization procedure. Because drug-eluting stents require prolonged dual antiplatelet treatment, bare metal stents should be used when necessary. Thrombolytic therapy should be reserved for life-threatening AMI when there is no access to a catheterization laboratory, because its use is associated with an increased risk of placental hemorrhage.[64] It may also contribute to further progression of a coronary dissection and for these reasons should be considered a second choice to primary PCI. If possible, the patient should be treated in a coronary care unit that can also provide maternal monitoring as well as a comprehensive maternal-fetal medicine service. In cases of hemodynamic instability and cardiac deterioration, a

plan should be established for urgent delivery of a potentially viable fetus. Surgical myocardial revascularization may be considered, usually when there is extensive coronary dissection and failure of PCI to reopen the vessel. Limited data are available about the safety and outcomes of these procedures, however.

Aortic Dissection

Epidemiology: Aortic dissection is another major cause of cardiac death, particularly in the context of connective tissue abnormalities such as Marfan syndrome, Ehlers-Danlos syndrome type IV, bicuspid aortic valve, familial aortic dissection, and Turner syndrome. In addition to the hemodynamic changes of pregnancy, hormonal changes render the aortic wall more vulnerable to dissection, with a fragmentation of the reticulin fibers, reduced mucopolysaccharides, and loss of the normal corrugation of elastic fibers. Type A and type B dissections occur, particularly in those with Marfan syndrome. Most dissections occur in the third trimester (50%) or the early postpartum period[60,65] during periods of maximum hemodynamic stress. Women with dilated aortas or prior aortic dissection are at particularly high risk during pregnancy. The diagnosis of dissection should always be considered in pregnant women who present with chest pain. Early mortality is high.

Dissections typically propagate in an antegrade direction but may extend retrogradely. Acute coronary compromise and severe aortic regurgitation may occur along with cardiac tamponade, which is the most common cause of cardiac arrest.

Pathophysiology: Cardiac arrest may occur from hemorrhage, acute aortic regurgitation (typically with a type A dissection), dissection of a coronary artery, and cardiac tamponade.

Diagnosis: Emergent echocardiographic imaging, transthoracic or transesophageal, is typically the fastest modality to delineate the etiology.

Treatment: Women with one of these connective tissue abnormalities and an aortic root diameter >4.5 cm should be counseled against pregnancy because of the risk of dissection.[60,65] This is particularly true when there is a family history of dissection or the aorta has undergone a period of rapid dilatation. For those with Marfan syndrome, no safe aortic diameter exists, and there is a 1% risk of dissection even with a normal aortic root diameter.[66] For those with Turner syndrome, who often are of short stature, an aortic diameter index >27 mm/m^2 is associated with a high risk of dissection, which carries a maternal mortality of approximately 11%.[60] Associated risk factors in this group include concomitant bicuspid aortic valve, coarctation of the aorta, and hypertension.

Patients with aortic pathology (ascending aorta >4 cm or less in those with a low body surface area) should be monitored throughout pregnancy by a multidisciplinary team that includes cardiologists with expertise and training in the management of high-risk patients with cardiovascular disease.[60] Monitoring should include imaging with echocardiography at regular intervals, the frequency of which is determined by the clinical context and size of the aorta, but generally at 6- to 8-week intervals and for 6 months postpartum.[59] β-Blocking agents are generally used to keep blood pressure under strict control and to reduce the shear stresses on the aortic wall, although there are scant data to support their use except in the context of Marfan syndrome. Hospitalization of high-risk patients should be considered between 28 and 32 weeks of gestation, with elective fetal lung maturation at 26 weeks of gestational age. Hospitalization should be undertaken in a center where cardiothoracic surgery is available. Prophylactic surgery should be considered during pregnancy if the aortic diameter is >50 mm and increasing rapidly.[60]

Aortic dissection in pregnancy is a surgical emergency, and the diagnosis must be made promptly, typically with transesophageal echocardiography, computed tomography, or magnetic resonance imaging, depending on local facilities and expertise. Senior cardiothoracic surgeons along with the team of cardiology, obstetric, and anesthesia staff must proceed rapidly to the cardiac operating room to repair the dissection. After 30 weeks of gestation, immediate cesarean section with delivery of the fetus in the cardiac operating room followed immediately by cardiac surgery seems to be the most promising

option to save the life of the mother and the unborn child. Cardiopulmonary bypass can be instituted simultaneously during cesarean section (femoral or axillary cannulation).[65] Cardiac surgery should be performed where neonatal intensive care facilities are available. If the fetus is not yet viable, high-pressure, high-flow (>2.4 L/min per square meter) normothermic perfusion is preferable for cardiopulmonary bypass, because hypothermia decreases placental flow and causes fetal bradycardia with an increased likelihood of intrauterine death or hypoxic-ischemic fetal insult.[65,67] If possible, maintaining uterine displacement by placing the patient in the left lateral recumbent position during cardiopulmonary bypass will help avoid aortocaval compression and minimize fetal risk.[67] Continuous fetal cardiac monitoring may decrease the risk to the fetus, as will close monitoring of serum potassium concentration (goal <5 mmol/L), because prolonged cardioplegia may increase potassium levels.

For type B dissection, medical therapy to strictly control blood pressure is the preferred strategy in the absence of rupture or malperfusion.[65] In this situation, there is a high incidence of fetal death, probably due to compromise of the internal iliac or ovarian arteries impairing placental blood flow.

Indications for surgical repair include leakage or rupture, progressive aortic dilatation, extension of the dissection, and recurrent pain.

Cardiomyopathy

Epidemiology: Cardiomyopathies are rare but remain an important cause of death.[68] This includes PPCM; patients with a prior history of PPCM should be counseled about the risk of recurrence. The incidence varies from around 1 in 4000 pregnancies in the United States to 1 in 300 pregnancies in Haiti, suggesting an environmental influence or common genetic mutation.[69] The etiology and pathophysiology are poorly understood, but inflammation and autoimmune processes may play a role. Risk factors include multiparity, race (being black), older maternal age, smoking, hypertension, and preeclampsia. Most cases present in the puerperium when the increased hemodynamic burden of pregnancy has diminished. Subsequent normalization of ventricular function

occurs in up to half of patients with PPCM and is more likely if the ejection fraction is >30% at the time of diagnosis. A subsequent pregnancy carries a 30% to 50% risk of recurrence of PPCM, which may result in further clinical deterioration and even death, particularly when ventricular function failed to normalize before the subsequent pregnancy.[68,70] Appropriate counseling before pregnancy is therefore imperative to prevent a cardiac catastrophe.

Other causes of cardiomyopathy include idiopathic dilated cardiomyopathy, familial cardiomyopathy, noncompaction, and ischemic cardiomyopathy. With all of these cardiomyopathies, women are at high risk of heart failure if the ejection fraction is <40%, and close monitoring with a multidisciplinary team in a tertiary center should be advised. When the ejection fraction is <30%, maternal mortality is increased, and termination of the pregnancy should be considered.[60] In accordance with the guidelines, patients with congestive heart failure and an ejection fraction <35% should have had implantation of an automated implantable cardioverter-defibrillator (ICD) because of the increased risk of ventricular arrhythmia and sudden cardiac death.

Hypertrophic cardiomyopathy: Pregnancy in women with hypertrophic cardiomyopathy (HCM) is usually well tolerated because the volume load of pregnancy reduces left ventricular outflow obstruction. Those at increased risk of cardiac arrest are very symptomatic before pregnancy with dyspnea, angina, syncope, and arrhythmias. Typically, these patients have a high left ventricular outflow tract gradient or clinically important diastolic dysfunction.[71,72] For patients with HCM, risk factors for sudden death include a history of out-of-hospital arrest, family history of sudden death, history of ventricular tachycardia, massive left ventricular hypertrophy on echocardiography (septal wall thickness >3 cm), presence of scar by delayed enhancement on magnetic resonance imaging, history of syncope, and either ventricular tachycardia or a decrease in blood pressure on treadmill exercise testing. When the 2 largest studies of pregnancy and HCM were combined in recent years,[71,72] only 2 deaths occurred in 470 pregnancies in 227 patients. Both had high-risk features before pregnancy (massive left ventricular hypertrophy with severe outflow obstruction, symptoms of heart failure, and a family

history of malignancy). Typically, such patients would have received an automated ICD before pregnancy. In other patients, pregnancy is usually well tolerated, and the risk of cardiac arrest is therefore very low. The decision to place an ICD for prevention of sudden death should be individualized, but in general, patients at the highest risk are considered candidates.[73]

Pathophysiology: Severe impairment of ventricular function with dilated cardiomyopathies may lead to intractable heart failure and ventricular tachycardia or fibrillation. Malignant ventricular arrhythmias may occur with HCM and high-risk features as outlined above.

Diagnosis: Diagnosis can be made with a transthoracic echocardiogram.

Treatment: Most severe heart failure in pregnancy is related to PPCM, and guidelines for the management of acute heart failure apply, except that angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are contraindicated in pregnancy. Bed rest and sodium and fluid restriction are important. Digoxin, hydralazine, oral nitrates used in combination with hydralazine, β-blockers, and diuretics can all be used with relative safety, although the latter may decrease placental perfusion and should be used judiciously. Aldosterone antagonists should be avoided because of antiandrogenic effects on the fetus. Anticoagulation with heparin should be considered in those with a very low ejection fraction, particularly those with PPCM who have a high incidence of ventricular thrombi and cerebral embolism.[68] Complex ventricular arrhythmias may precipitate cardiac arrest in this context.

In severe cases, nitrates and inotropes may be used, and in such circumstances, consideration should be given to transferring the patient to a center where intra-aortic balloon pump counterpulsation, ventricular assist devices, and transplant teams are available. Extracorporeal membrane oxygenation may be necessary for those with cardiogenic shock. Urgent cardiac transplantation may need to be considered.[68] Urgent

delivery irrespective of gestation should be considered in women with hemodynamic instability despite treatment.

For women with HCM, pregnancy should be avoided in those with high-risk features. Implantation of an automated ICD can prevent cardiac arrest in high-risk patients with HCM. Maintenance of β-blocker therapy (the mainstay of treatment) should be continued or even increased during pregnancy and through delivery to alleviate left ventricular outflow obstruction. Dehydration and anemia should be avoided, as should tocolytic therapy. Gentle incremental diuresis can be used for symptoms of heart failure. Delivery should be accomplished in high-risk centers; spinal block should be avoided in case of hypotension, and blood loss should be promptly replaced.

Mechanical Valve Prostheses

Epidemiology: Death may also occur from thrombosis of a mechanical prosthetic valve, although this is a rare cause of cardiac death. Optimum anticoagulation strategies are challenging in this situation, and there are clear data that anticoagulation with warfarin is safer for the mother then either low-molecular-weight or unfractionated heparin.[74] The use of heparin for even a relatively short time (typically in the first trimester to avoid the risk of fetal embryopathy) more than doubles the risk of mechanical valve thrombosis.[75] Because pregnancy is a markedly prothrombotic state, valve thrombosis and death can occur even in the context of therapeutic anti-Xa levels with the use of low-molecular-weight heparin.[76] The mechanical mitral valve, which is the most vulnerable to thrombus formation, is particularly problematic.[60] Optimum anticoagulation with the selected anticoagulant is essential throughout pregnancy, and patients should be managed in a tertiary care center with a multidisciplinary approach. Anticoagulant targets may need to be measured weekly.

Although cardiac arrest is uncommon, it may occur in the setting of a thrombosed mechanical prosthesis, particularly with left-sided valves (either mitral or aortic), and results from low cardiac output and/or ventricular arrhythmia.

Pathophysiology: Pregnancy is a hypercoagulable state with an increased concentration of clotting factors, decreased fibrinolysis, and increased platelet adhesiveness. Most valve thromboses occur in the setting of a tilting disc mitral prosthesis with heparin therapy, which is a less optimum anticoagulant than warfarin during pregnancy. Even with careful and meticulous monitoring in a tertiary care center, valve thrombosis may occur. Because anticoagulation is interrupted during both vaginal and cesarean delivery, prosthetic valve thrombosis may occur in the peripartum period.

Diagnosis: Emergent transthoracic echocardiography can facilitate the diagnosis to identify the thrombosed valve.

Treatment: The only interventional therapy of benefit in this situation is emergent valve replacement with concomitant delivery of the fetus if viable.

Congenital Heart Disease

Epidemiology: Patients with severely reduced function of the systemic ventricle and those with cyanotic heart disease, particularly those with pulmonary hypertension, are at most risk of adverse cardiac events. In those with severe pulmonary vascular disease and a right-to-left shunt (Eisenmenger syndrome), maternal mortality was 30% to 50% in early series, with death usually occurring in the last trimester or peripartum.[77]

Patients with severe symptomatic aortic stenosis, usually secondary to a bicuspid aortic valve, should be counseled against a pregnancy, because the increase in cardiac output and afterload reduction that accompanies pregnancy exaggerates the aortic gradient. Patients may present with syncope, angina, or heart failure, and malignant arrhythmias and death may occur.[60] Such patients also have an associated aortopathy and an increased risk of aortic dissection and rupture.

Patients with d-transposition of the great arteries after an atrial switch procedure (Mustard or Senning operation) are vulnerable to heart failure because the systemic ventricle is a morphological right ventricle. Patients with l-transposition of the great

arteries are also at risk because the systemic ventricle is a right ventricle and vulnerable to failure, particularly in the context of systemic atrioventricular valve regurgitation, which may progress as pregnancy advances. Patients with single-ventricle physiology after the Fontan procedure are also vulnerable to heart failure, and both atrial and ventricular arrhythmias may intervene, causing profound hemodynamic instability and cardiac arrest.

In Eisenmenger syndrome, cardiac arrest usually occurs with a progressive decrease in peripheral resistance with hypotension causing more right-to-left shunting and cyanosis, or abruptly with pulmonary thrombosis. Patients are at special risk of cardiac arrest particularly in the third trimester and in the early postpartum weeks. Progressive right-sided heart failure may also occur. Even with contemporary pulmonary vasomodulator drugs (nitric oxide and sildenafil), maternal mortality may occur in 33% of patients and may occur in those with minimal disability before pregnancy.[78]

Pathophysiology: Eisenmenger syndrome—right-sided heart failure, in situ pulmonary thrombosis, and progressive cyanosis with decrease in peripheral resistance; aortic stenosis—angina, syncope, heart failure, and ventricular arrhythmias.

Diagnosis: If the diagnosis is unknown, emergent echocardiography is the only modality that might facilitate diagnosis in an emergent situation.

Treatment: Aortic stenosis—severely symptomatic patients refractory to medical therapy may be considered for percutaneous valvuloplasty if the valve is noncalcified and free of regurgitation. If the fetus is viable, early delivery by cesarean section followed by emergency valve replacement may be lifesaving.[67] In women whose pregnancy is <30 weeks in duration, emergency aortic valve replacement with the precautions previously outlined for cardiopulmonary bypass should be undertaken.

Eisenmenger syndrome: Hospital admission to a tertiary care center well in anticipation of delivery is warranted, with management by a multidisciplinary care team. Treatment of

right-sided heart failure is often warranted, and concomitant therapy with sildenafil may help reduce pulmonary artery pressure. Intravenous prostacyclin analogues can also be considered. Inhaled nitric oxide may be helpful peripartum in those with unstable hemodynamics. The use of anticoagulants is controversial, because cyanotic patients are at increased risk of bleeding, but death may occur from pulmonary thrombosis.

High-Risk Cardiac Arrhythmia Substrate

Epidemiology: In addition to the cardiac conditions mentioned above, genetic conditions associated with a high risk of sudden arrhythmic death include those related to disturbances of the cardiac ion channels (channelopathies), which include the long QT syndrome, catecholaminergic polymorphic ventricular tachycardia, Brugada syndrome, and short QT syndrome. Of the channelopathies, the long QT syndrome is the most studied**.** Observational reports from registries have demonstrated an increased incidence of adverse cardiac events (syncope or ventricular arrhythmias) during the postpartum period, but not during the pregnancy, which actually was associated with decreased risk. Women with the LQT2 genotype are especially at high risk.[79,80] Reports on other channelopathies are scant and mainly limited to single case reports or very small series.[81]

Pathophysiology: There is a diversity of syndromes, but in general, these genetic mutations affect the proper function of ion channels within the heart, predisposing the patient to life-threatening arrhythmias, mainly ventricular fibrillation and torsades de pointes.

Diagnosis: The diagnosis is made based on clinical presentation and electrocardiographic and genetic testing.

Treatment: The most studied channelopathy during pregnancy is the long QT syndrome, for which β-blockers have been shown to be effective in preventing events.[79,80]

ICD Implantation in High-Risk Patients

ICD therapy has been shown to be well tolerated during pregnancy, although the number of patients reported in the literature is small. Natale et al reported no significant increase in complications due to ICD therapy or ICD shocks in 44 women who received or already had ICDs during pregnancy.[82] When ICD shocks are required, no adverse effects to the fetus have been reported. ICD therapy should therefore be considered for pregnant patients who are at high risk for sudden cardiac death or patients who plan to conceive in the future.[82,83] Related to implantation of an ICD during pregnancy, one common concern is the risk of radiation exposure to the fetus. This risk is likely overstated, as today's ICDs can be implanted safely with very small doses of fluoroscopy or even none at all. In fact, common cardiovascular interventions can be performed with relatively low radiation exposure to the fetus, well below the 50 mGy threshold generally considered associated with increased risk.[84] After serious consideration of risk versus benefit, if implantation of an ICD is deemed necessary, careful planning and additional precautions should be taken to minimize radiation exposure.[85] Similarly, in rare cases, catheter ablation of arrhythmias might be necessary. This can now be done using nonfluoroscopic 3-dimensional mapping technology, which significantly decreases the amount of radiation required during the study and in some cases can even be done with no radiation at all.[86]

D—Drugs

Oxytocin

Epidemiology: Oxytocin is commonly used for induction and augmentation of labor and prevention of uterine atony and postpartum hemorrhage after vaginal or cesarean delivery.

Pathophysiology: Oxytocin is a potent systemic vasodilator with negative inotropic effects, and its use may result in cardiovascular side effects, including hypotension, tachycardia, and myocardial ischemia.[87] In patients who develop hemodynamic instability before or immediately after cardiac arrest, administration of oxytocin may precipitate rearrest because of sudden decreases in pre- or afterload that decrease stroke volume and systemic vascular resistance, respectively.

Diagnosis: Although uterine atony is a common etiology of obstetric hemorrhage and may precipitate cardiac arrest, administration of oxytocin during chest compressions must be balanced against other medical and/or surgical interventions available to the emergency response team. Oxytocin may precipitate or contribute to cardiovascular collapse if administered in large (>5 international units) bolus doses.[88]

Treatment: To prevent oxytocin-mediated hypotension, the smallest effective dose should be used when oxytocin is administered as an uterotonic agent.[89,90] Slow infusion rather than bolus administration is preferable,[91] and a concomitant phenylephrine infusion can minimize the hemodynamic effect of oxytocin on systemic vascular resistance.[92]

Magnesium

Epidemiology: Magnesium sulfate (intravenous or intramuscular) is commonly used for seizure prophylaxis in preeclampsia, tocolysis in threatened preterm labor, and fetal neuroprotection of extreme preterm (<32 weeks of gestation) fetuses.[93]

Pathophysiology: Magnesium is a mild generalized vasodilator, a tocolytic (depresses smooth muscle contraction), and a central nervous system depressant. Both maternal (flushing, lethargy, nausea, pulmonary edema, respiratory depression, cardiac arrest, uterine atony) and neonatal symptoms of toxicity (respiratory distress, decreased cerebral perfusion, hypotonia, feeding difficulties) are potentially associated with its administration.[93,94] Although magnesium-induced uterine atony is theoretically possible, evidence that magnesium administration is associated with hemorrhage is equivocal.[95] Decreased renal excretion of magnesium is a major cause of toxicity, but maternal and fetal magnesium toxicity can present regardless of impaired renal function.

Diagnosis: Toxicity should be immediately suspected in any patient receiving magnesium in respiratory and/or cardiac arrest.

Treatment: Magnesium should be stopped immediately and empiric calcium treatment administered. Calcium gluconate IV/IO 30 mL 10% solution or calcium chloride IV/IO 10 mL 10% solution should be administered immediately.[96-98] Cardiopulmonary arrest resulting from magnesium sulfate overdose has been successfully treated with aggressive resuscitation and calcium administration.[97]

E—Embolic Causes

Amniotic Fluid Embolism

Epidemiology: AFE complicates between 2 and 8 of 100 000 deliveries.[99-101] High-quality supportive care can result in good outcomes, with survival >70% in most contemporary surveillance reports.[99-101] One in 4 women who experience AFE also experience cardiopulmonary arrest; more than half of these women survive.[1] Nevertheless, some cases of AFE may be fatal regardless of medical care.

Pathophysiology: Cardiovascular pathophysiology after AFE appears to progress from pulmonary vasospasm and hypertension to right-sided heart failure and later left-sided heart failure.[102,103] Any of these cardiovascular derangements may be sufficiently severe to cause cardiac arrest; alternatively or additionally, disseminated intravascular coagulopathy may produce massive hemorrhage resulting in hypovolemic cardiac arrest.

Diagnosis: Although cardiac arrest may be the first sign of AFE, most women present first with changes in mental status, dyspnea, hypotension, fetal bradycardia, maternal seizure, or rarely, hemorrhage with coagulopathy.[104,105] AFE most frequently presents during labor but can also manifest at the time of delivery[104] or in the immediate postpartum period.[32,106]

Treatment: Treatment for AFE-related maternal cardiopulmonary arrest follows the principles of maternal basic life support and advanced cardiovascular life support plus specific treatment for coagulopathy and hemorrhage and aggressive interventions to support the cardiovascular system. Although bleeding may be anticipated after AFE,[107] for women who experience cardiopulmonary arrest outside the operating room,

perimortem cesarean delivery should be completed at the bedside before consideration of whether or not to transport the patient to an operating room. After delivery, uterine packing and manual compression of the aorta may be effective strategies to limit any hemorrhage that may develop in the immediate postpartum period.

In anticipation of massive hemorrhage and coagulopathy after return of spontaneous circulation, providers should secure large-bore venous access, administer uterotonics, and activate the institutional massive transfusion protocol to provide both hemodynamic and hemostatic support. Point-of-care viscoelastic monitoring may reveal a profound fibrinolysis that indicates antifibrinolytic therapy (eg, tranexamic acid or α-aminocaproic acid).[108] Prothrombotic agents (eg, recombinant factor VIIa) are available but may be associated with thrombotic complications and should be considered a therapy of last resort.[109,110]

Case reports have described the successful use of cardiopulmonary bypass and extracorporeal membrane oxygenation to treat AFE.[29,100,102,108-110] These technologies may exert their greatest effect by filtering noxious substances from the blood; even continuous hemofiltration has been described as therapeutic.[111,112] Institutions should evaluate whether any of these technologies are available on an emergent basis for obstetric patients and are the most effective means of activation.

Inhaled nitric oxide or prostacyclin may be indicated to treat pulmonary hypertension.[113] Case reports also describe right ventricular support devices for right-sided heart failure[114] and intra-aortic balloon counterpulsation for management of left-sided heart failure.[115]

Finally, although hypothermia improves neurologic outcomes for survivors of cardiac arrest, its use may exacerbate coagulopathy[116,117] and should be carefully considered in women with evidence of coagulopathy induced by AFE.

Thromboembolism

Epidemiology: Thromboembolism is an important cause of maternal mortality. According to the CMACE data, thromboembolism is the fourth leading cause of maternal deaths.[2] The major cause of maternal mortality from thromboembolism is pulmonary embolism and central vein thrombosis.[2] The most recent CMACE data showed a dramatic decrease in deaths due to thromboembolism, which is attributed to more widespread thromboprophylaxis and enhanced recognition of women at risk. There are many risk factors for thromboembolism in pregnancy[118]; however, obesity is the most important of these.[2] Important strategies for preventing maternal mortality from thromboembolism include identifying at-risk women, using weight-specific guidelines for specific thromboprophylaxis when necessary, and investigating chest symptoms when necessary based on the patient's risk of thromboembolism.[2]

Pulmonary Embolism

Epidemiology: Pulmonary embolism is an important cause of cardiac arrest in women at all stages of pregnancy and into the postpartum period.

Pathophysiology: Preexisting risk factors include age >35 years, obesity with body mass index >30 kg/m^2, parity >3, previous thromboembolism, thrombophilia, gross varicose veins, paraplegia, sickle cell disease, and chronic medical conditions. Transient risk factors include hyperemesis, ovarian hyperstimulation syndrome, dehydration, long-haul travel, surgical procedures (cesarean section), infection, and immobility.

Diagnosis: If pulmonary embolism is suspected during cardiac arrest, investigation can include a transesophageal echocardiogram. But chest compressions should not be interrupted for investigation; therefore, other tests such as V/Q scan and computed tomography pulmonary angiography will not be possible while the patient is in cardiac arrest. If there is a high index of suspicion of pulmonary embolism, treatment can be considered empirically. If there is evidence of pulmonary embolism, then treatment should be rapidly initiated.

Treatment: Recommendations for the treatment of life-threatening pulmonary embolism include thrombolysis.[119,120] Although thrombolysis can result in both maternal and fetal complications, case reports of successful treatment with embolectomy with and without thrombolysis have been reported.[121,122] Risk-benefit ratio assessment should govern its use.

Cerebrovascular Events

Epidemiology: Cerebral venous thrombosis (CVT) accounts for 2% of all strokes during pregnancy.[123] Cerebral thrombosis is a cause of indirect mortality during pregnancy.[2] The incidence of CVT is estimated to be 12 per 100 000 during the peripartum period, which is almost 12 times the incidence of CVT in the general population.[124,125] The risk is highest in the third trimester and the initial 4 weeks postpartum.[123]

Pathophysiology: CVT is a cause of stroke and is defined as thrombosis of the dural sinus and/or cerebral veins.[123] The increased risk of CVT during pregnancy is due to the hypercoagulability. Risk factors for CVT during pregnancy include increasing maternal age, increasing hospital size, cesarean delivery, hypertension, infections, and excessive vomiting.[123] Volume depletion is believed to be the reason for increased risk in the postpartum period. The large hemorrhagic lesion or multiple lesions and diffuse brain edema can cause transtentorial herniation, resulting in death.[123] Other causes of death in the setting of CVT are status epilepticus, medical complications, and pulmonary embolism.[123]

Diagnosis: Diagnosis is made on clinical suspicion with confirmation by imaging. The patient usually has a headache from the increased intracranial pressure. This may be the only clinical clue to the diagnosis of CVT.[123] Other findings include focal neurological findings and papilledema.[123] Magnetic resonance imaging T2-weighted imaging plus magnetic resonance venography is the imaging modality of choice to confirm the diagnosis of CVT.

Treatment: The treatment of choice for CVT during pregnancy is low-molecular-weight heparin in full anticoagulant doses, which should be continued until at least 6 weeks postpartum.[123]

Venous Air Embolism

Epidemiology: Air embolism can cause cardiac arrest in pregnancy. Historically, venous air embolism is responsible for about 1% of all maternal deaths.[126]

Pathophysiology: Venous air embolism is caused by air that is released from ruptured veins and then travels into the central venous system and embolizes into the pulmonary artery.[127] Risk factors for venous air embolism include cesarean delivery, Trendelenburg position, placental abruption, placenta previa, exteriorization of the uterus, manual extraction of the placenta, severe preeclampsia, hemorrhage, and hypovolemia.[127]

Diagnosis: Symptoms of venous air embolism include chest tightness or shortness of breath. Findings can include hypotension, decreased oxygen saturation, decrease in P_{ETCO_2}, and increased end-tidal nitrogen prearrest. Air embolism can be diagnosed by echocardiogram (transthoracic and transesophageal); once the condition is diagnosed, treatment should be rapidly initiated.[127]

Treatment[127]: Treatment consists of a multidisciplinary approach that involves prevention of further gas entry, gas removal, and maintenance of hemodynamic stability. Hydration is an important first step in management, and in a patient undergoing an operation, the surgical field should be flooded with fluid. The team could insert a central venous catheter to extract the air. During resuscitation, inotropes may be required. Hyperbaric oxygen therapy can be considered.

F—Fever

Sepsis

Epidemiology: Sepsis in the pregnant woman is relatively infrequent but remains one of the 5 leading causes of pregnancy-related death around the world. Bacteremia occurs in

<1% of obstetric patients,[128] and only a small proportion of these develop septic shock, which has been estimated to occur in 0.002% to 0.01% of all deliveries.[129] Sepsis was the only direct cause of maternal death to increase in frequency in the most recent Confidential Enquiries report, with 26 deaths from 2006 to 2008 compared with 13 to 18 in the previous triennia,[2] largely due to community-acquired group A streptococcal genital tract infection.

Pathophysiology: Septic shock is characterized by refractory hypotension and inadequate delivery of oxygen, producing end-organ hypoperfusion and lactic acidosis. Cardiac arrest may result from myocardial ischemia (as a result of the sepsis process or inotropic drug therapy) or severe acidosis or as a result of marked hypotension.

Diagnosis: The CMACE report lists "red flag" signs and symptoms of sepsis in pregnancy. In addition to the common characteristics, such as fever, tachycardia, and dyspnea, important other concerns include renal angle tenderness or the woman who is "generally unwell, unduly anxious, distressed."[2] Sepsis may occur as a result of nonobstetric infections (commonly pyelonephritis or pneumonia) or obstetric infection (chorioamnionitis, postpartum endometritis, and wound infections).

Treatment: Deaths due to sepsis may be avoided by prompt recognition and treatment. Early administration of antibiotics has a dramatic effect on outcome, with survival (in a nonpregnant cohort) decreasing by about 7% for each hour of delay in administration of antibiotic after onset of hypotension.[130] Management of sepsis in the pregnant patient is similar to that in the nonpregnant patient. Because the fetus is at risk in the presence of maternal hypotension, this must be rapidly managed initially with volume resuscitation and left lateral positioning. Vasopressor drugs may reduce uterine and therefore placental perfusion, but the benefits of correcting maternal hemodynamics usually outweigh this concern. The usual intensive care vasopressors are typically used, despite the fact that norepinephrine, epinephrine, and dopamine may adversely affect uterine blood flow.[131] Short-term management of maternal hypotension secondary to neuraxial anesthesia with ephedrine or phenylephrine is often used without a problem in small bolus doses or

infusion.[132,133] Cardiac arrest in the patient with sepsis may be a result of inadequate fluid resuscitation, and marked hypotension may be interpreted as pulseless electrical activity. Consideration should be given to aggressive fluid resuscitation.

Influenza/Acute Respiratory Distress Syndrome

Epidemiology: Data from influenza pandemics have demonstrated an increased mortality rate in pregnant women compared with a matched nonpregnant cohort. Reports after the 2009 H1N1 influenza A pandemic described a high incidence of severe disease and respiratory failure in pregnant women, with hypoxemic respiratory failure and a significant mortality rate.[134] Influenza vaccination is an important prophylactic intervention, but a low uptake of vaccination was identified in pregnant patients, some of whom went on to develop severe respiratory failure.[135]

Pathophysiology: The pregnant woman's immune system changes to allow tolerance to paternally derived fetal antigens, with a downregulation of cell-mediated immunity balanced by an intact or upregulated humoral immune response.[136] These changes may predispose the pregnant woman to more severe manifestations of certain infections, including some viral and fungal infections. In addition, the pregnant woman may be more susceptible to the development of acute respiratory distress syndrome, related to factors such as increased circulating blood volume and hypoalbuminemia, but an immunological effect may also play a role. The pregnant state or the process of labor and delivery may produce an inflammatory change in the lungs, priming the lungs for the development of acute respiratory distress syndrome.[137] Cardiac arrest may result from the marked hypoxemia that may occur in these patients.

Diagnosis: During an influenza outbreak, a pregnant woman with pulmonary infiltrates should be considered to have influenza pneumonitis. Diagnosis can be confirmed by immunoassay or polymerase chain reaction of a nasopharyngeal swab specimen.

Treatment: Treatment with antiviral therapy within 48 hours of onset of symptoms improves outcome. Management of cardiac arrest in these patients should emphasize the importance of rapidly correcting hypoxemia.

G—General

The pregnant patient would also be at risk for causes of cardiac arrest in the nonpregnant patient, and these etiologies should be considered. Some of these etiologies listed below overlap with others highlighted in this appendix. These collective etiologies are often referred to as the "H's and T's": hypoxia, hypovolemia, hydrogen ion (acidosis), hypo-/hyperkalemia, hypothermia, toxins, tamponade (cardiac), tension pneumothorax, thrombosis (pulmonary), and thrombosis (coronary). More extensive discussion is outside the scope of this appendix.

H—Hypertension

Epidemiology: Preeclampsia and its variants complicate about 7% of all pregnancies, leading to morbidity and mortality in both mother and fetus.[138] In a British surveillance report from 2006, eclampsia occurred in 27.5 cases per 100 000 maternities.[139] US data reveal a higher rate of 82 per 100 000 deliveries between 1995 and 2004.[140] Overall, 14.5% of all cardiac arrests during hospitalization for delivery may be related to preeclampsia and its variants.[1]

Pathophysiology: Preeclampsia and its variants can lead to cardiac arrest through a variety of pathophysiological processes, including cerebral hemorrhage; eclampsia leading to hypoxia or stroke; pulmonary edema leading to hypoxia; and hepatic failure or rupture leading to profound hemorrhage. Preeclampsia itself can lead to thrombocytopenia and disseminated intravascular coagulation, resulting in massive hemorrhage.[49] Due to the complex pathophysiological presentation, multiple organ systems must be evaluated during resuscitation.

Diagnosis: The hallmark of preeclampsia and its variant is hypertension: systolic blood pressure ≥140 mm Hg more than 4 to 6 hours apart and/or diastolic blood pressure ≥90

mm Hg more than 4 to 6 hours apart in the sitting position. Because this is a multisystem disease, evaluation of liver function, renal function, and hematologic system, including coagulation parameters, is required. Proteinuria is no longer required for the diagnosis.[141] Catastrophic complications such as stroke may alter the presentation. A toxicology screen should also be included. If labetalol is given, the toxicology screen will be positive for barbiturates.

Treatment: Timely recognition and treatment of severe preeclampsia/eclampsia is the best preventive strategy to avoid arrest. Cerebral autoregulation is compromised, and women are at risk of intracerebral hemorrhage when blood pressure reaches a level of 160/110 mm Hg.[142] Treatment of systolic blood pressures ≥160 mm Hg or diastolic blood pressures ≥110 mm Hg with antihypertensive agents is essential. Two systems solutions have been proposed to ensure timely treatment for severe hypertension: (1) prompt bedside evaluation by a senior clinician and/or (2) standing orders that empower nurses to initiate antihypertensive therapy.[143] The use of magnesium sulfate to prevent or treat eclampsia can be lifesaving.[2,141] Currently, delivery of the fetus and placenta is the only intervention known to treat the underlying pathophysiology of preeclampsia. Attempting to balance perinatal prematurity against severity of maternal disease can lead to insidious disease progression, which can result in significant maternal morbidity leading to cardiac arrest.

Airway management in the setting of preeclampsia and eclampsia is fraught with danger. Airway edema associated with preeclampsia increases the risk of difficult laryngoscopy and failed intubation. In addition, if laryngoscopy and/or endotracheal intubation are undertaken without meticulous hemodynamic management, the resulting abrupt increase in blood pressure can overwhelm already injured cerebral endothelium and lead to intracerebral hemorrhage. For these reasons, endotracheal intubation in the setting of preeclampsia/eclampsia should only be attempted (1) by airway management experts, (2) after other airway management strategies fail (eg, bag-mask ventilation, supraglottic airway ventilation), or (3) after the onset of cardiopulmonary arrest. The diagnosis of

preeclampsia/eclampsia is critical information for providers called to manage the obstetric airway.

The patient with cardiac arrest and preeclampsia should be delivered within 5 minutes; uterine evacuation will facilitate resuscitation and also begin to reverse the underlying pathophysiology. Magnesium sulfate should be instituted in a 4 to 6 g intravenous loading dose over 15 to 20 minutes, with a maintenance dose of 2 g/h unless hypermagnesemia is thought to be the cause of arrest (see section on drugs). Oxygenation and ventilation are crucial to management, especially if intracranial hemorrhage is suspected; however, before return of spontaneous circulation, standard ventilation with a goal of maintaining normocarbia will help preserve cardiac output and cerebral blood flow.[144,145] After return of spontaneous circulation, neuroradiologic evaluation should be performed to identify intracranial processes, including hemorrhage and mass effect leading to intracranial hypertension. Intracranial surgical intervention may be required. Hyperventilation may be considered after return of spontaneous circulation to avoid brainstem herniation in the patient with intracranial hypertension. However, this maneuver reduces cerebral blood flow and may exacerbate anoxic brain injury.[144,145] Blood product replacement may be necessary to reverse coagulopathy.

Table 3. Risk Stratification in Cardiac Disease[57]

Prior cardiac event (heart failure, transient ischemic attack, stroke)[57]
Cardiac arrhythmia before pregnancy[57]
NYHA class III or IV or cyanosis[57]
Left-sided heart obstruction (mitral valve area <2 cm^2, aortic valve area <1.5 cm^2 or peak left ventricular outflow tract gradient >30 mm Hg)[57]
Systemic ventricular ejection fraction <40% or hypertrophic or restrictive cardiomyopathy[57]
Cardiac medication before pregnancy[58]
NYHA class II or greater[58]
Left-sided heart obstruction (peak gradient >50 mm Hg or aortic valve area <1.0 cm^2)[58]
Systemic atrioventricular valve regurgitation (moderate/severe)[58]
Mechanical valve prosthesis[58]
Cyanotic heart disease, repaired or unrepaired[58]
Severe pulmonary regurgitation and/or reduced subpulmonary ventricular systolic function[146]
History of smoking[146]

NYHA indicates New York Heart Association.

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