DOI: 10.1097/JPN.0b013e31825187f8

CE **Continuing Education**

J Perinat Neonat Nurs • Volume 26 Number 2, 117–123 • Copyright © 2012 Wolters Kluwer Health | Lippincott Williams & Wilkins

Maternal Cardiac Arrest

An Overview

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ABSTRACT

Cardiac arrest in pregnancy is a rare event, and the speed of resuscitation response is critical to the outcome of both the mother and the fetus. The management of the unresponsive pregnant woman differs from that of the traditional adult resuscitation. In this article, causes of maternal arrest, management of proper cardiopulmonary arrest in pregnancy, and implementation of perimortem cesarean delivery are discussed.

Key Words: advanced cardiac life support, maternal cardiac arrest, maternal mortality, pregnancy, perimortem cesarean delivery

Despite many attempts over the years to improve quality and safety in obstetric care, maternal morbidity and mortality in the United States continue to be significant public health issues. Although death from complications during pregnancy, childbirth, and the puerperium remains an infrequent event, the maternal mortality rate remains far above the *Healthy People 2010 and 2020* national target of fewer than 3.3 maternal deaths per 100 000 live births.[1] The current maternal mortality rate in the United States is 13.3 deaths per 100 000 live births, well over the targeted rate and thought to be underreported because of inconsistent state reporting systems.[2] Racial disparities related to maternal mortality also exist. Black women are 3.7 times more likely to die from pregnancy complications than white women.[3]

In addition, pregnancies complicated by preexisting medical conditions have increased.[4] In one recent study, severe morbidity was shown to be 50 times more common than maternal death.[5] Furthermore, several studies have concluded that 28% to 50% of maternal deaths were preventable.[6–8] Finally, in 2010, the World Health Organization reported that 49 other countries had lower maternal mortality rates than the United States.[9] These data suggest that maternal morbidity and mortality are reducible and there is a need for improved knowledge, skills, and attitude regarding early recognition of and response to deterioration in a pregnant woman's condition during pregnancy, labor, birth, and the puerperium.

Because of the increasing number of pregnancies with preexisting conditions and maternal mortality, the healthcare team has a duty to respond when the pregnant woman's condition worsens, leading to the provision of advanced cardiopulmonary life support. In situations where deterioration of maternal status results in respiratory or cardiac arrest, healthcare providers need to provide basic and advanced life support that include the necessary pregnancy alterations taking into account physiologic changes induced by the pregnancy and the unique circumstances of both the mother and the fetus. The goal of implementing pregnancy-specific changes in response to an arrest is improving survival of both the mother and the fetus. This article reviews the most common causes of maternal arrest and modifications that need to be made for proper cardiopulmonary resuscitation of the pregnant woman, including perimortem cesarean birth.

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Disclosure: The authors have disclosed that they have no significant relationships with, or financial interest in, any commercial companies pertaining to this article.

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Submitted for publication: January 16, 2012; accepted for publication: February 22, 2012.

CAUSES OF MATERNAL ARREST

Causes of cardiopulmonary arrest during pregnancy are usually nonarrhythmogenic events resulting in pulseless electrical activity.[10] Following recommended advanced cardiac life support (ACLS) resuscitation guidelines for pulseless electrical activity, determination of the cause of cardiopulmonary arrest is required to properly manage the event. For this discussion, the more common causes have been divided into obstetric, nonobstetric, and iatrogenic etiologies as noted in Table 1.

Obstetric etiologies

Hemorrhage

Expansion of maternal circulating blood volume can mask the signs and symptoms of hemorrhage until blood loss is considerable. Loss of circulating blood volume or cardiac output leads to decreased perfusion and can result in cardiopulmonary collapse. Lack of early recognition and treatment increases the risk of maternal arrest in response to hemorrhage.[11] Effective quantification of blood loss and awareness of initial changes in maternal vitals signs are strategies to identify significant blood loss and begin treatment.[12] If maternal arrest is attributed to hemorrhage, blood volume should be replaced with crystalloid solutions and blood components (packed red blood cells, fresh frozen plasma, and platelets), using low resistance, intravenous tubing. A rapid infusion device is helpful to warm and quickly administer large volume of fluids to reverse the arrest. In addition, surgical intervention may be required to control hemorrhage.

Hypertension/preeclampsia/eclampsia

Up to 12% to 22% of pregnancies are affected by hypertension, and the disorder accounts for approximately 17% to 18% of maternal deaths in the United States.[13] Life-threatening complications result from hypertensive emergencies, eclamptic seizure, hypovolemia, cerebral edema, stroke, and HELLP (hemolysis, elevated liver enzymes, and low platelets). Other complications associated with maternal mortality include hepatic rupture, pulmonary edema, placental abruption with risk of maternal hemorrhage, renal failure, and cardiac dysfunction.[14] This disease is the multisystem organ failure phenomenon of pregnancy.

Supportive treatment includes the administration of magnesium sulfate to prevent seizure activity. A 4- to 6-g loading dose is administered intravenously over 20–30 minutes, followed by a 2-g/h maintenance dose as a continuous infusion for 24 hours, is recommended.[14] Antihypertensive therapy may include hydralazine or labetalol for the treatment of diastolic pressure of 105 to 110 mm Hg or more.[14]

In addition, it is important to pay meticulous attention to fluid intake and output. If cardiac arrest occurs, there may be decreased intravascular volume, which is part of the pathophysiology of preeclampsia/eclampsia. Fluid volume resuscitation may be a treatment option for cardiac arrest in the pregnant woman.

The overall treatment of preeclampsia/eclampsia is delivery of the infant. If the infant is preterm and the woman has mild preeclampsia, observation is appropriate. The woman is evaluated weekly by monitoring 12- to 24-hour urine collection for assessment of renal function and obtaining laboratory test consistent with platelet count and liver enzymes. An astute nursing assessment of blood pressure, pulse, respirations, development of generalized edema, complaints of headache, visual changes, right upper quadrant pain, and hyperreflexia are reported to the physician. In addition, signs of neurologic changes such as mental confusion and decreased level of consciousness are reported immediately. Ongoing fetal surveillance includes weekly nonstress tests and biophysical profiles. If there are indications of worsening maternal or fetal condition, delivery of the infant is indicated regardless of gestation.[13]

Amniotic fluid embolism

Amniotic fluid embolism (also known as anaphylactoid syndrome of pregnancy) is an unpredictable, lethal condition reported to occur in 1 per 12 053 North American births, making this a rare event that is difficult to diagnose.[15] It causes a series of symptoms, with 100% of women having acute hypotension and 87% experiencing associated cardiovascular collapse and consumptive coagulopathy.[16] Treatment is supportive, with the major

Table 1. **Common causes of maternal arrest**[a]

Obstetric	Nonobstetric	Iatrogenic
Hemorrhage	Sepsis	Anesthestic complications
Preeclampsia	Pulmonary embolism	Magnesium sulfate toxicity
Anaphylactoid syndrome of pregnancy	Cardiovascular disease	
Peripartum cardiomyopathy	Stroke	

[a]From Raschke.[10]

goals of adequate oxygenation, aggressive restoration of cardiac output, and reverse coagulopathy.[17]

Nonobstetric etiologies

Sepsis

Sepsis is the leading cause of mortality in an intensive care environment.[18] The pathophysiology of the sepsis, severe sepsis, and septic shock continuum leads to alterations in vascular tone, maldistribution of circulating volume, microvascular coagulation, myocardial dysfunction, decreased cardiac output, and limited oxygen transport to organ systems.[18] Decreased tissue perfusion results in multisystem organ dysfunction and failure if the signs and symptoms of maternal compromise are not recognized and managed appropriately in a timely manner. With decreased T-cell- and humoral-mediated immunity, pregnancy is considered an altered immune state, leaving the woman vulnerable to infection. Common sources of obstetric infection are the reproductive tract, urinary tract, respiratory tract infection, wound, chorioamnionitis, and cholecystitis.[18] In response to the infectious source, an uncontrolled, pathologic inflammatory response leads to the signs and symptoms of sepsis. Treatment of sepsis to decrease the likelihood of cardiopulmonary compromise is outlined in Table 2.

Table 2. **Sepsis resuscitation bundle**[a]

Bundle element 1	Measure serum lactate levels: May reflect ↓ in tissue perfusion and organ dysfunction Mortality rates ↑ with increasing lactate >4 mmol/ L +
Bundle element 2	Obtain blood cultures prior to antibiotic administration: Blood cultures should be taken as soon as possible after the onset of fever or chills.
Bundle element 3	Administer broad-spectrum antibiotic within 3 h of ED admission and within 1 h of non-ED admission.
Bundle element 4	In the event of hypotension or serum lactate >4 mmol/L: Deliver an initial minimum of 20 mL/kg of crystalloid or an equivalent Administer vasopressors for hypotension not responding to initial fluid resuscitation to maintain mean arterial pressure >65 mm Hg
Bundle element 5	In the event of persistent hypotension despite fluid resuscitation (septic shock) and/or lactate >4 mmol/L: Maintain adequate CVP Maintain adequate O_2 saturation

Abbreviations: CVP; ED, emergency department.
[a]From Dellinger et al.[19]

Pulmonary embolism

Venous thromboembolism (VTE) is one of the leading causes of maternal mortality in the United States, complicating 1 in 2500 pregnancies.[20] Because of the normal physiologic changes that affect coagulation, pregnancy is considered a hypercoagulable state, increasing the risk of VTE 4-fold over nonpregnant states.[20] Fibrinogen levels double during pregnancy in preparation for placental separation. There is also an increase in clotting factors V, VII, VIII, IX, X, and XII. In addition to the normal physiologic changes that place the pregnant woman at increased risk, other conditions that increase the incidence for venous thrombosis and thromboembolism are listed in Table 3.

Research has not validated a specific cause and effect relationship between some of the risk factors and thrombus formation; it may be a combination of several risk factors. In the case of a deep vein thrombosis, early recognition and management decrease the incidence of VTE. In pregnant women who are adequately treated for a deep vein thrombosis, VTE occurs in approximately 4.5% of these women, resulting in death of less than 1% women. However, without diagnosis of a deep vein thrombosis or adequate treatment, the incidence of VTE increases to 24%, with a resulting mortality rate of 15%.[21]

Venous thromboembolism is also higher following cesarean birth. Among pregnant women who give birth by cesarean delivery, especially emergent cesarean delivery, the risk of VTE is increased 9-fold versus women who have vaginal delivery. A higher incidence is thought to be due to an increase in tissue trauma and subsequent disruption of the vascular endothelium.[22–24] In 2008, Clark et al[6] reviewed individual causes of maternal deaths among 1.5 million births within 125 hospitals in the previous 6 years at the Hospital Corporation of America. One of the conclusions of the Hospital Corporation of America report was to decrease maternal death, nationwide efforts must be taken to prevent VTE in women who deliver by cesarean birth.[6] Although there is debate regarding the necessity of and procedure for VTE prophylaxis, options include the use of embolic stockings or a pneumatic compression device, anticoagulant administration, or a combination of the therapies.

Stroke

Stroke during pregnancy has a serious consequence in terms of maternal mortality and fetal outcome.

Antepartum pregnancy-related stroke increased 47% from 4085 in 1994-1995 to 6293 in 2006-2007,[25] In postpartum women, the stroke rate increased 83%.[25] Most arterial strokes occur during the end of pregnancy and immediately after delivery, as opposed to venous strokes occurring anytime.[26] Risk factors strongly linked to pregnancy-related stroke include hypertension, diabetes, heart disease, sickle-cell disease, thrombophilia, smoking, and recreational drug use, particularly cocaine.[26] In addition, cesarean delivery has been associated with 3 to 12 times increased risk of puerperium and postpartum stroke.[26] This could be due to other pregnancy-related conditions such as preeclampsia or cardioembolic phenomenon. Common complaints on presentation are headache, focal neurologic deficits, seizures, or visual changes. Preeclampsia and eclampsia should not be regarded as a cause of the neurologic event during pregnancy until stroke has been ruled out. Time is of the essence when the pregnant woman exhibits signs and symptoms of a stroke.[26] For proper treatment, it is necessary to rule out cerebral ischemia or hemorrhage as soon as possible by obtaining a computed tomographic brain scan.

Cardiac disorders

It is estimated that cardiac disease complicates 4% of all pregnancies and accounts for a large percentage of maternal mortality in the United States.[27] Cardiovascular disease accounts for 12% of pregnancy-related deaths. Cardiomyopathy is responsible for an additional 11% of pregnancy-related deaths. If these 2 categories are combined, cardiac disorders are the leading cause of pregnancy-related mortality in the United States. Because of the similarity in patient assessment parameters, pregnancy can mimic cardiac disease and make the diagnosis of cardiac problems difficult. Symptoms such as dyspnea, decreased tolerance to exercise, fatigue, dependent edema, and alterations in heart sounds are normal in pregnancy.[27] Abnormal maternal cardiac symptoms include syncope, chest pain, paroxysmal nocturnal dyspnea, hemoptysis, cyanosis, shortness of breath at rest, distention of neck veins, persistent arrhythmia, summation gallop, systolic murmur (grades IV-V; VI), diastolic murmur, and a persistent maternal tachycardia of more than 100 beats per minute.[27] In general, the normal symptoms that result from pregnancy are more gradual in onset. Acute onset of symptoms should be concerning and result in a full evaluation of the woman's physiologic status.[28]

Becoming pregnant at a later age contributes to the increased chance of atherosclerotic heart disease, causing cardiac complications. Since 1991, cardiac death as a result of myocardial infarction and aortic dissection in pregnancy is rising.[29] Also adding to this group are infants born with congenital heart disease who survive into adulthood and have children.

There are 4 predictors of maternal compromise during pregnancy if cardiac disease is present as noted in Table 4.[30] The risk of a cardiac event occurring during pregnancy can be described as follows: a woman with cardiac disease but no other risk factor, as noted in Table 4, is assigned a risk rate of 5%; for a woman with 1 risk factor, the risk rate increases to 25%; and for a woman with more than 1 risk factor, this risk rate further increases to 75%.[30]

Iatrogenic etiologies

Anesthetic complications

Complications related to anesthesia include difficulty in obtaining an airway or systemic toxicity from an epidural or spinal anesthesia. Difficult or failed intubation leads to the inability to ventilate or oxygenate, resulting in cardiac arrest.[31] The laryngeal mask airway is an option that may be used as life-saving rescue for obstetrical patients with failed intubation. Systemic toxicity develops from anesthesia accidentally injected into the

Table 3. Risk for venous thombosis and thromboembolism[a]

Cesarean birth
Previous or family history of venous thromboembolism
Maternal age >35 y
Parity of ≥3
Smoking
Mechanical heart valve
African American women
Trauma
Infection
Prolonged immobilization
Antiphospholipid syndrome
Obesity—body mass index ≥30 kg/m^2
Sickle cell disease

[a]From Han and Paidas.[20]

Table 4. Predictors of maternal compromise[a]

A. Prior cardiac event (eg, heart failure, transient ischemic attack, stroke, or arrhythmia)
B. New York Heart Association Class >II prior to pregnancy
C. Left heart obstruction (eg, mitral valve area <2 cm^2, aortic valve area <1.5 cm^2, peak left outflow gradient >30 mm Hg)
D. Ejection fraction <40%

[a]From Siu et al.[30]

circulatory system causing respiratory arrest, acute hypotension, bradycardia, and ventricular arrhythmias. Immediate intervention includes discontinuing the anesthesia, obtaining an effective airway, increasing fluids, and using ACLS protocols for favorable outcomes.

Magnesium sulfate toxicity

According to the Institute of Safe Medicine Practices, there have been multiple reports of accidental magnesium sulfate overdose resulting in cardiopulmonary arrest and maternal death.[32] Although magnesium sulfate is listed as a "high-alert" medication, reports of administration errors still frequently occur. Acute care settings should have an administration procedure in place for consistent, safe administration practices. Symptoms of magnesium toxicity are cardiac effects from electrocardiographic changes, (eg, bradycardia, prolonged QT interval), loss of deep tendon reflexes, hypotension, sedation, severe muscular weakness, urinary retention, respiratory depression, nausea and vomiting, and flushing. A serum magnesium level of more than 7.5 mmol/L may result in respiratory depression. Cardiac arrest may occur with levels more than 12 mmol/L. If magnesium sulfate toxicity is suspected, the nurse should turn off the infusion, notify the physician or team, and consider the administration of 10 mL of 10% solution of calcium gluconate over 10 minutes.

ALTERATIONS IN LIFE SUPPORT

Anatomic and physiologic changes during pregnancy require several additions to ACLS algorithm. Following recognition of cardiopulmonary arrest, rapid activation of the code response team should be accomplished to gain additional assistance and guidance to follow current American Heart Association (AHA) guidelines for adults. In addition, to prepare for proper resuscitation in the pregnant woman, availability of ACLS equipment, Cesarean delivery instruments, and neonatal equipment should be brought immediately to the woman's bedside. Time is not taken to move the woman to an operating room for resuscitation, which may include perimortem cesarean delivery.[10]

Positioning

Positioning the pregnant woman to optimize resuscitation efforts is necessary. Aortocaval compression by the uterus impedes resuscitation by decreasing venous return, causing supine hypotension, and decreasing the effectiveness of thoracic compressions. The woman should be positioned supine and the uterus manually displaced on the left or right side to achieve the most effective chest compressions. There are 2 techniques to displace the uterus. If using the 2-handed technique, the hands are placed around the uterus and pulled toward the person providing this relief measure. Another technique is to manually displace the uterus by placing a firm wedge underneath the patient's buttock to elevate the left or right hip. If the uterus is not displaced, uterine compression can occlude up to 30% of circulating blood volume.[10] In addition, the uterus can prevent the forward blood flow needed in resuscitation.

Airway/breathing

Physiologic changes in pregnancy make airway management more difficult to achieve due to upper airway edema, increased vascularity resulting in hyperemia, disease processes associated with pregnancy (eg, preeclampsia), and hypersecretion.[33] Attempts are made to rapidly secure an effective airway with endotracheal intubation, since oxygen desaturation is significantly faster in pregnant patients than in nonpregnant patients.[33] Potential for rapid maternal decompensation is due to a 20% reduction in functional residual capacity and increased oxygen demand and consumption. In addition, ventilation volumes may need to be decreased because of the elevated diaphragm.

When intubating, a smaller endotracheal tube may be necessary due to laryngeal edema; usually a 6.5- or 7.0-French catheter is preferred.[10] Every effort is made to prevent aspiration during resuscitation, as can occur with prolonged bag-mask ventilation. Application of cricoid pressure during intubation and the use of rapid sequence intubation can reduce the risk of aspiration.[10]

Circulation

In pregnant women, chest compressions are performed slightly higher on the sternum. During cardiac arrest, compressions administered by the rescuer will deliver a small amount of blood flow to the vital organs. In the nonpregnant state, at best, chest compressions provide only 30% of the normal cardiac output. Effective compressions increase the likelihood for blood flow to the vital organs.[34] Since the gravid uterus and/or positioning of the woman may obstruct a portion of venous return and limit cardiac output, chest compressions in pregnancy may provide less than 30% of normal cardiac output.

Pharmacologic agents are given on the basis of electrocardiographic rhythm and maternal response. Importantly, the volume of drug distribution and metabolism may vary from nonpregnant women. If there is no response to standard doses, higher doses are considered to account for the expanded plasma volume of pregnancy. Vasopressin may be more effective than its counterpart, epinephrine, and gives rise to fewer adverse effects with fewer postresuscitation complications.[33]

Defibrillation

Defibrillation is performed according to the recommended ACLS protocol.[33] Risk factors for adverse fetal outcomes such as fetal arrhythmias or burns are due to the magnitude of the current complication, although there are no studies documenting fetal complications.[33] The only recommendation is that fetal monitors are removed prior to defibrillation.[33] This prevents the electric arcing during defibrillation; however, there is no evidence to support this.

PERIMORTEM CESAREAN BIRTH

Perimortem cesarean delivery is defined as a cesarean delivery after cardiopulmonary resuscitation has been initiated.[35] Without adequate cerebral perfusion, irreversible brain damage from anoxia occurs within 4 to 6 minutes.[36] Historically, providers have been taught to perform a perimortem cesarean delivery within 5 minutes of maternal unresponsiveness. Unfortunately, there are no case-control studies to verify the exact timing of a perimortem cesarean delivery and maternal/neonatal outcome. There are several case reports that have documented successful resuscitation of pregnant women in cardiac arrest after a perimortem cesarean delivery. It seems that the time interval from cardiac arrest to delivery is the single most important prognostic factor for fetal survival.[37] If the fetus is delivered early in resuscitation, the autotransfusion that occurs after delivery and release of aortocaval obstruction can cause enough volume to be shunted back into the systemic system, causing an increase in cardiac output and a return of sinus rhythm. Delivery may assist in cardiopulmonary resuscitative efforts and allow for a viable fetus. The fetus has multiple physiologic mechanisms to respond to hypoxic sequelae lasting more than 10 minutes, including high hemoglobin concentration, increased oxygen affinity, and the ability to transport blood supply to the brain, heart, and adrenal glands when needed.[38,39]

On the basis of physiological changes in pregnancy and according to the 2010 ACLS recommendations, "When the gravid uterus is large enough to cause maternal hemodynamic changes due to aortocaval compression, emergency cesarean section should be considered,"[33(pS837)] This is usually recommended at 24 weeks or more and no maternal response to advanced resuscitation.[33] Therefore, it is recommended that perimortem cesarean delivery should begin at 4 minutes after the onset of maternal cardiac arrest.[33,36,37]

The best place to perform a perimortem cesarean delivery is at the current location of the patient. With the increasing drills or simulations, when everything is in perfect order, performing a perimortem cesarean delivery can be hard to achieve in the recommended time frame. Transporting the pregnant woman to an operative area increases the time from cardiopulmonary arrest and perimortem cesarean delivery. In addition, transporting the woman does not allow continued adequate resuscitative efforts such as compressions and correct bag-mask ventilation.[40] This delay in care may impact maternal and fetal survival. To facilitate a timely delivery, it is necessary to have the emergency delivery equipment with the resuscitation cart. This includes an antiseptic solution, scalpel, and packs for the abdomen and uterus. A Pfannenstiel incision may be faster than a vertical midline incision; however, when gestation and the lie of the fetus are not confirmed, a vertical midline incision should be considered for a perimortem cesarean delivery.[33] The delivery occurs while cardiopulmonary resuscitation is in progress and ACLS protocols are followed.

SUMMARY

Care providers must be aware of the impact of physiologic and anatomic changes that occur during pregnancy and how these changes affect resuscitation techniques in the pregnant woman. Guided by ACLS protocols, rapid intubation, uterine displacement, fast, firm chest compressions and performing PCMS within 4 minutes must be commenced. Cause of the cardiopulmonary arrest should be considered to treat with the appropriate measures. Since cardiopulmonary arrest is rare in pregnancy, ongoing simulation or drills to improve provider readiness are necessary to optimize outcomes.

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