Chapter

30

Medical Resuscitation in Pregnancy

T CAMPBELL, S GALWANKAR, KP O'KEEFE

INTRODUCTION

Any clinician can be confronted with the task of resuscitating a pregnant patient. Approximately 1 in 30,000 live births are complicated by maternal cardiac arrest[1]. In addition to the causes of cardiac arrest in the general public, pregnancy adds other risks to both the mother and the fetus. Changes in the anatomy and physiology during pregnancy affect both the incidence of certain diseases, as well as the mother's ability to compensate for illness. Additionally, there is a second life which must also be considered. Trauma, pulmonary embolism, hemorrhage, hypertension, and infection are the leading causes of maternal death in pregnancy. If a pregnant patient suffers from cardiopulmonary arrest, the best predictor of fetal survival is rapid maternal resuscitation. Prompt assessment of the maternal-fetal unit and initiation of appropriate management are essential to the survival of both patients. Since cardiopulmonary arrest can occur abruptly, all physicians need to be aware of the alterations in resuscitation procedures necessary during pregnancy. Furthermore, specialists in obstetrics and neonatology should be consulted early in the resuscitative efforts.

ANATOMY AND PHYSIOLOGY OF PREGNANCY

Many changes in normal anatomy and physiology occur throughout pregnancy which alters both the presentation and treatment of disease. A sound knowledge of these changes will allow the physician to determine the severity of illness and the appropriate treatment early in the course of management.

Pregnancy induces numerous hemodynamic changes that can make patient assessment challenging. The heart rate will typically rise throughout pregnancy, reaching a maximum increase of 10-15 beats per minute by the end of the third trimester[1,2]. Blood pressure decreases by 2-3 mm Hg systolic and 10-15 mm Hg diastolic during the first two trimesters, returning to normal during the third trimester[1,2]. Progesterone induced smooth muscle relaxation results in decreased vascular resistance, and a 5 mm Hg decrease in central venous pressure by the third trimester[2,3]. These changes combine to lead the naïve physician into believing the pregnant mother to be hypotensive, where in fact the blood pressure may be actually adequate for sustaining normal cellular functions. Additionally pregnant patients have a dilutional anemia due to a 50% increase in plasma volume, with only a 30% increase in red blood cell mass[1,2]. At term, the placenta receives 10% of this maternal systemic volume. This increase in circulating volume allows for a much greater hemorrhage before signs of maternal hypovolemia become apparent.

At end of the second trimester, cardiac output increases by 30-50% to provide for the increasing demand of the growing uterus[1]. There is a 10 fold increase in the blood flow to the pregnant uterus[1,2] with the mother's total blood volume flowing through the uterus every 8-11 minutes.[2] Disruption of the placenta or trauma to the uterus or pelvis can result in extensive maternal hemorrhage.

By 20 weeks gestation the gravid uterus can cause vena caval compression resulting in the supine hypotension that is commonly seen in pregnancy. This compression causes a decrease in preload, resulting in a 10 to 30 percent decrease in cardiac output, and decreased arterial blood pressure[1]. The uterus also begins to compress the aorta in the latter half of pregnancy. Placing the patient in the left lateral decubitus position relieves the aorto-caval compression, and is considered

the optimal position if the patient is greater the 20 weeks' gestation. If the patient is hypotensive and repositioning is not an option, the uterus can be manually displaced to the left allowing for greater blood return to the heart. This maneuver is valuable during cardiopulmonary resuscitation (CPR) to improve cardiac return while allowing more efficient chest compressions.

Compression of pelvic veins by the enlarging uterus, in addition to that of the inferior vena cava, can cause an increase in venous pressure below the uterus. This increase in pressure leads to dependant edema, venous stasis, varicose veins, and hemorrhoids of pregnancy. The increased venous pressure results in rapid blood loss from injuries to the pelvis or lower extremities. Due to the increased pressure and poor venous return to the heart, intravenous lines in the lower extremities should be avoided if possible. When intravenous access below the uterus is necessary, any medication administered through that IV will have a limited return to the heart and the arterial circulation.

The large increase in plasma volume during pregnancy can result in extravasation of fluid into the surrounding tissue. This is most noticeable in the lower extremities due to the added affect of increased venous pressure. However, the edema can also occur in the upper extremities, face, and oropharynx. It is important to note that while edema may indicate a disease process, it can also be present in the absence of disease. Edema can also make ventilation of the pregnant patient more difficult.

Thromboembolism is more prevalent in pregnancy. Venous stasis, expanded venous volume, and an increase in fibrinogen and coagulation factors are the causes of thromboembolism during the latter part of pregnancy. Immobility in a pregnant patient increases this risk, and can further complicate the illness.

In addition to the hemodynamic changes seen in pregnancy (Table 1), there are also alterations in respiration. These changes can have an effect on the patient's ability to compensate for respiratory distress, as well altering values on blood gas. The gravid uterus slowly pushes the diaphragm more cephalad as it enlarges. Furthermore, the growing fetus puts many new demands on the maternal system. There is an increase in metabolic rate and a 15-20% increase in oxygen requirements.[1] The combination of these changes causes a 40% increase in tidal volume, with a resultant 25% decrease in residual volume and functional residual capacity[1, 3]. Hypoxia can occur quickly with respiratory arrest. The increase in tidal volume causes increased minute ventilation and a respiratory alkalosis. However, renal compensation is typically able to maintain pH values near normal. These changes can be seen in the arterial blood gas as an increase in pO_2, and a decrease in both pCO_2 and bicarbonate. Consequently, the patient is less able to buffer pH changes or to compensate for respiratory compromise. This increases the risk of injury to the fetus due to maternal hypoxemia and acidemia.

Table 1: Mean values for hemodynamic changes seen throughout pregnancy

	Pre-pregnancy	*1st trimester*	*2nd trimester*	*3rd trimester*
Heart rate (beats/min)	70	78	82	85
Systolic blood pressure (mm Hg)	115	112	112	115
Diastolic blood pressure (mm Hg)	70	60	63	70
Central venous pressure (mm Hg)	9.0	7.5	4.0	3.8
Femoral venous pressure (mm Hg)	6	6	18	18
Cardiac output (L/min)	4.5	4.5	6.0	6.0
Blood volume (mL)	4000	4200	5000	5600
Uterine blood flow (mL/min)	60	600	600	600
Hematocrit (%)	40	36	34	36

Table modified from Marx: Rosen's Emergency Medicine: Concepts and Clinical Practice, 6th ed. Ch 35 Trauma in pregnancy

Changes in the gastrointestinal system also affect the management of the pregnant patient. The slow stretching of the abdominal wall due to uterine enlargement, desensitizes it to peritoneal irritation. Abdominal tenderness, rebound, and guarding may be absent or significantly reduced even in the presence of peritoneal irritation. Thus, injury or infection in the abdominal cavity may be more easily overlooked. Gastrointestinal motility slows and the gastric sphincter response is reduced. This results in an increased likelihood of aspiration with altered level of consciousness or resuscitative efforts. Additionally, aspiration can be more damaging to the lungs due to the increased production of gastric acid during pregnancy.

Routine laboratory tests are an important part of the management of any critically ill patient. Due to the vast change in maternal physiology, laboratory normal values change in pregnancy as well. This must be considered in the interpretation of any results (see Table 2 for normal values in pregnancy). The CBC will often demonstrate an elevated white blood cell count. This coupled with an increased erythrocyte sedimentation rate (ESR) might suggest an infectious process in

the non-pregnant patient, but is entirely normal in pregnancy. Special attention should be given to the platelet value and the fibrinogen level. If these values are low, the possibility of DIC should be considered and further evaluation accomplished.

An arterial blood gas can provide valuable information about a patient's respiratory status. A PCO_2 of 40 is within the normal range for the non-pregnant patient, but is concerning in a pregnant patient. This value is indicative of poor ventilation and possible respiratory acidosis which can lead to fetal distress[6].

Table 2: Laboratory values in pregnancy compared to controls

	Pregnancy values	*Normal values*
Hematocrit (%)	32-42	35-47
White blood cell count (/μ)	5,000-12,000	4,500-11,000
ESR (mm/hr)	78	<20
Arterial pH	7.40-7.45	7.35-7.44
Bicarbonate (mEq/L)	17-22	21-28
PCO_2 (mm Hg)	25-30	35-45
Fibrinogen (mg/dL)	> 400	200-400
Prothrombin Time (sec)	11.2	13.5

Table from information obtained in ch. 35 Trauma in Pregnancy - Marx: Rosen's Emergency Medicine: Concepts and Clinical Practice, 6th Ed.

Pre-hospital Care

General treatment of the pregnant patient is the same as that for a non-pregnant patient with a few exceptions. Any female of child bearing age should be assessed for the possibility of pregnancy. If greater than 20 weeks gestation, the possibility of supine hypotension must be considered. These patients should be placed in the left lateral decubitus position to maximize blood return to the heart. Even in the absence of respiratory distress, the patient should be given supplemental oxygen to compensate for the increased oxygen demand of the fetus. IV access should be established for the administration of fluids and medication if necessary.

If possible, the patient should be transported to a medical center that can handle both the mother and the fetus. A quick assessment of fetal viability can be obtained by palpating the fundal height. When the fundus reaches the level of the umbilicus appropriate arrangements should be made prior to arrival to the hospital in the event that emergent delivery is required.

Cardiopulmonary Resuscitation

Evaluation of airway, breathing, and circulation remains the primary focus. The first priority should be resuscitation of the mother before evaluation of the fetus is initiated. Estimation of gestational age is beneficial in determination of fetal viability. If the fetus is deemed non-viable, treatment should be directed exclusively toward maternal well-being (Fig. 1).

Fetal age can be estimated by the fundal height of the uterus (Table 3). Once the fundus reaches the level of the umbilicus, fundal height in centimeters is approximately equivalent to gestational age (i.e. 24 cm, 30 cm and 34 cm measured from the symphysis pubis to the fundus is approximately 24, 30 and 34 weeks respectively). A fundal height at the level of the umbilicus is estimated to be 20 weeks' gestation. Twenty-three to 24 weeks' gestation is considered the minimum age for fetal viability. With this in mind, a fundal height greater than 3 to 4 cm above the umbilicus should be considered a potentially viable fetus.

Table 3: Fundal height related to gestational age

Fundal height	*Average gestational age*
Pubic symphysis	12 weeks
Umbilicus	20 weeks
Xiphoid process	36 weeks

If oxygen supplementation was not initiated prior to arrival, it should be initiated immediately, and continued until hypoxemia, hypovolemia and fetal distress have all been ruled out. Hypoxia can occur rapidly due to the decreased oxygen reserve, and the increased oxygen consumption seen in pregnancy. The threshold for intubation should be low. Securing the airway will maintain proper oxygenation and reduce the risk of aspiration.

The American Heart Association recommends following standard resuscitation algorithms with a few modifications to compensate for the altered anatomy and physiology of pregnancy. The primary ABCD survey requires no alteration to the assessment of airway or breathing. Angling the patient at 15 to 30 degrees to the left or having someone pull the gravid uterus to the left will relieve compression on the inferior vena cava and allow for optimal cardiac return. No additional changes are needed in positioning or dosage for defibrillation. Defibrillation does not carry any increase risk for the fetus, but the fetal monitor should be removed prior to defibrillation[7].

The secondary survey requires the greatest number of modifications for the pregnant patient. Airway management in the pregnant patient can be difficult. Obesity is prevalent in the obstetric population and in the supine position the neck is more extended than in a

non-obese patient. This results in improper alignment of the larynx, and difficulty intubating. If possible, the mother should be placed in the sniffing position to facilitate intubation. In the sniffing position the neck is flexed onto the chest and extended at the alantooccipital joint. The head should be elevated slightly with the shoulders remaining on the table. This allows optimal alignment of the pharynx and larynx and the least amount of tongue obstruction. Breast engorgement in pregnancy can also interfere with intubation, by making insertion of the laryngoscope more difficult.

Changes in gastrointestinal motility and sphincter control place patients at increased risk of aspiration. Rapid sequence intubation with cricoid pressure should be considered early in the resuscitative efforts to prevent aspiration and to ensure adequate ventilation. The edematous soft tissue of the oropharynx during the latter part of pregnancy can make both mask ventilation and intubation challenging. Pregnant patients are at greater risk of developing hypoxemia, so adequate preoxygenation is essential. There is a greater rate of failed intubation in the obstetric population than in the general population[9]. As pregnancy progresses and edema of the oropharynx intensifies, the rate of Mallampati class III (for Mallampati classification see Table 4) airway increases.[1] In pregnancy, the airway is more prone to injury, and trauma from attempted intubation can cause bleeding and increased swelling of the already edematous tissue.

Table 4: Mallampati classification of difficult airways

Mallampati classification	*Visualization*
Class I	Soft palate, fauces, anterior pillar and posterior pillar
Class II	Soft palate, fauces and entire uvula
Class III	Soft palate and base of uvula
Class IV	Hard palate only soft palate not visible

The special circumstances surrounding airway management in a pregnant patient should be kept in mind when intubation is considered. Initially, the same induction medications and dosage should be administered in both the pregnant and nonpregnant patient. As the mother is in a hypervolemic state, higher doses may be necessary. A short handle laryngoscope will allow easier insertion into the oropharynx. If cervical injury is not suspected, the patient should be placed in the sniffing position. Cricoid pressure should be held both to aid in intubation and to prevent aspiration. There may be a need for a smaller tube to compensate for a smaller airway patency and prevent potential complications. The use of nasotracheal intubation should be avoided due to the increased risk of injury to the edematous tissue. Likewise, an orogastric tube is preferred over a nasogastric tube for gastric decompression. The airway should be checked closely for blood following intubation. It may be necessary to resort to alternative measures to secure the patient's airway. Laryngeal mask airway can be considered if orotracheal intubation is unsuccessful. However, this does not protect against aspiration. Broncosopic intubation may be necessary, so the appropriate equipment should be readily available. If all attempts fail and an airway can not be secured, it may be necessary to use more invasive measures to prevent hypoxia in both the mother and the fetus. Percutaneous transtracheal jet ventilation or cricothyrotomy have both been proven to be lifesaving maneuvers[10].

Since the mother's oxygen reserve is low and hypoxia can occur rapidly, ventilator settings should selected to compensate for this. Both functional residual capacity and functional residual volume are decreased, and minute ventilation and tidal volume are increased in pregnancy. Maternal oxygen saturation needs to remain above 60 mm Hg to prevent hypoxia in the fetus.

Intravenous access should be obtained with administration of sufficient replacement fluids (preferably lactated ringer's solution) and appropriate medication. With vena caval compression, intravenous medication delivered through access below the uterus may be delayed in reaching the heart or arterial circulation. Obtaining access above the uterus is essential. Pregnancy causes a baseline hypervolemia and this must be considered in the calculation of replacement fluids. Since circulating maternal volume is dramatically increased, signs of hypovolemia may not become apparent until significant hemorrhage has occurred. In a hypovolemic state the body responds with self preservation, and the maternal blood supply is shunted to essential organs and away from non-essential organs. The uterus is non-essential, and extensive fetal compromise can occur before the mother shows signs or symptoms of shock. Most ACLS medications can be given during resuscitation in pregnancy. However, vasopressors impair uterine perfusion, and should be avoided if possible. If, after crystalloid infusion, vasopressors are still warranted, they should not be withheld.

Following maternal stabilization, the condition of the fetus should be assessed. Verification of fetal heart tones is the first step in evaluation. In the absence of fetal heart beat upon presentation to the emergency department, the chance of fetal resuscitation is poor. In this situation,

full attention should be focused on the mother and treatment of her condition regardless of gestational age. If fetal heart tones are present, and the mother is stable, continued evaluation of the fetus is warranted. Fetal heart rate should be between 120 and 160 beats per minute[1,6]. Placental disruption or insufficiency, as well as maternal hypoxia, hypovolemia, or hypotension can all result in fetal hypoxia. Hypoxia will lead to fetal bradycardia. Fetal tachycardia may be due to hypoxia as well, but fetal hypovolemia should also be considered. Bedside ultrasound is ideal for assessment of the fetus.

Effects on the Fetus

The effects on the fetus from maternal cardiopulmonary arrest depend on the initiating factor. Maternal resuscitation offers the best prognosis for the fetus no matter what the cause. Fetal demise is typically due to hypoxia and can be minimized by maintaining adequate perfusion and oxygenation to the placenta.

The fetus does have some protective measures against severe hypoxia. The fetus can alter cardiac output, resulting in increased blood flow to the placenta and increased gas exchange. The fetus can redistribute blood volume to vital organs, protecting them from severe damage. Fetal hemoglobin has a greater affinity for oxygen than maternal hemoglobin. In comparison to the maternal oxyhemoglobin dissociation curve, the fetal oxyhemoglobin dissociation curve is shifted to the left. Thus, even at a lower partial pressure of oxygen, fetal hemoglobin will bind to oxygen more strongly, allowing for greater oxygen saturation. Additionally, there is more fetal hemoglobin in each fetal red blood cell than maternal hemoglobin in maternal red blood cells, further allowing oxygenation of the fetus. In contrast to the mother the fetus is slightly acidotic. This academia results in better off loading of oxygen to the fetal tissues. As long as the maternal oxygen saturation remains above 60 mmHg, the fetus is able to use these mechanisms to compensate for hypoxia. When the maternal oxygen saturation falls below 60 mm Hg, fetal oxygen saturation falls dramatically[8,9].

Fetal Monitoring

After the initial assessment of the fetus and determination of gestational age, any fetus estimated to be greater than 20 weeks' gestation (potentially viable) should be observed with fetal tocodynamometry. Fetal monitoring should be initiated immediately even if treatment of the mother is not complete. Early signs of fetal distress include tachycardia, loss of beat-to-beat or long term variability, or late decelerations. The fetus is more vulnerable to adverse conditions, and fetal distress could be an indication of impending maternal destabilization.

If conditions of fetal distress persist, and the fetus is greater than 23 weeks, emergent cesarean delivery may be required. Often, delivery of the fetus will result in maternal stabilization due to the resolution of cardiovascular compromise caused by aortocaval compression by the gravid uterus.

Perimortem Cesarean Delivery

In the event that all attempts at maternal resuscitation have failed, a decision to perform a cesarean section must be made rapidly. Gestational age greater than 23 weeks is the recommended cut-off for immediate delivery. These instances of perimortem delivery are rare, and chances of fetal survival are poor in general. The earlier the fetus is delivered following maternal arrest, the greater the chance of fetal survival. Delivery should take place in the current location; transportation to an operating room will cost valuable time. Currently it is suggested that cesarean section be performed within 4 minutes after initial maternal arrest. This is based on the evidence that a fetus delivered within 5 minutes from initiation of CPR had the best chance of survival. Those deliveries occurring more than 5 minutes after cardiopulmonary arrest were unlikely to result in a normal, viable infant.[1,2] Resuscitative efforts should continue during preparation for, and throughout delivery of the fetus. In some circumstances, delivery of the fetus can result in maternal recovery due to release of vena caval compression and improved cardiac return.

Management

After initial assessment and stabilization, further testing should be performed based on clinical findings. Diagnostic tests should not be withheld due to potential adverse effects on the fetus. The mother is the primary patient, and her survival is the best predictor of fetal survival. However, if an alternative test with less potential to cause harm to the fetus is available, it should be considered. A sterile speculum examination should be performed for injury, vaginal bleeding, rupture of membranes, cervical dilation and effacement. If vaginal bleeding is noted on external exam, an ultrasound to rule out placenta previa should be done prior to speculum or bimanual examination. If the placenta is over the cervical os, trauma can occur to the placenta during digital or speculum exam, resulting in massive hemorrhage.

Ultrasound is standard for the evaluation of the fetus throughout pregnancy. It is non-invasive and has no deleterious effects on the fetus. It is used at the bedside to evaluate both the condition of the mother and the fetus. Fetal movement, size, gestational age, heart rate, placental location, and amniotic fluid volume can all be assessed. Radiography is often required for the diagnosis and treatment of many diseases. Although the developing fetus is more susceptible to the effects of radiation, the risks are minimal, and are directly affected by gestational age and level of exposure. Fetal loss is seen with excessive radiation exposure during the first 2 weeks of pregnancy. The greatest potential for malformation secondary to radiation occurs during organogenesis (2 to 8 weeks of development)[5,6]. Beyond 20 weeks normal radiation exposure has little consequence on the developing fetus. When exposure is kept to a minimum, the related risks are trivial. A cumulative radiation dose less than 10 rads during development has not shown an increase in fetal loss, malformation, or mental retardation[5,6]. However, it is possibly associated with a small increase in the number of childhood neoplasms. A radiation dose of 15 rads or more has shown a slightly higher risk of all of these complications. A V / Q scan delivers less then 0.5 radsto the developing fetus. Plain films are estimated to deliver less than 1 rad per film to the fetus[5,6]. Shielding of the uterus when possible can minimize this exposure. Computed tomography (CT) represents a higher exposure risk, but again appropriate shielding can reduce this risk significantly. CT of the head and chest can easily be performed with a fetal exposure of less than 1 rad. Abdominal and pelvic CT scans should be done judiciously and if possible should be avoided in early pregnancy as they can deliver anywhere from 3 to 9 rads depending on variation in individual scanners, and technique used[5,6]. MRI is without use radiation and is safe in pregnancy, but the effects of contrast have not been studied.

Causes of Maternal Arrest

Although cardiopulmonary arrest in pregnancy is rare, it does occur, and identification of the underlying cause will guide further treatment (Table 5). There are

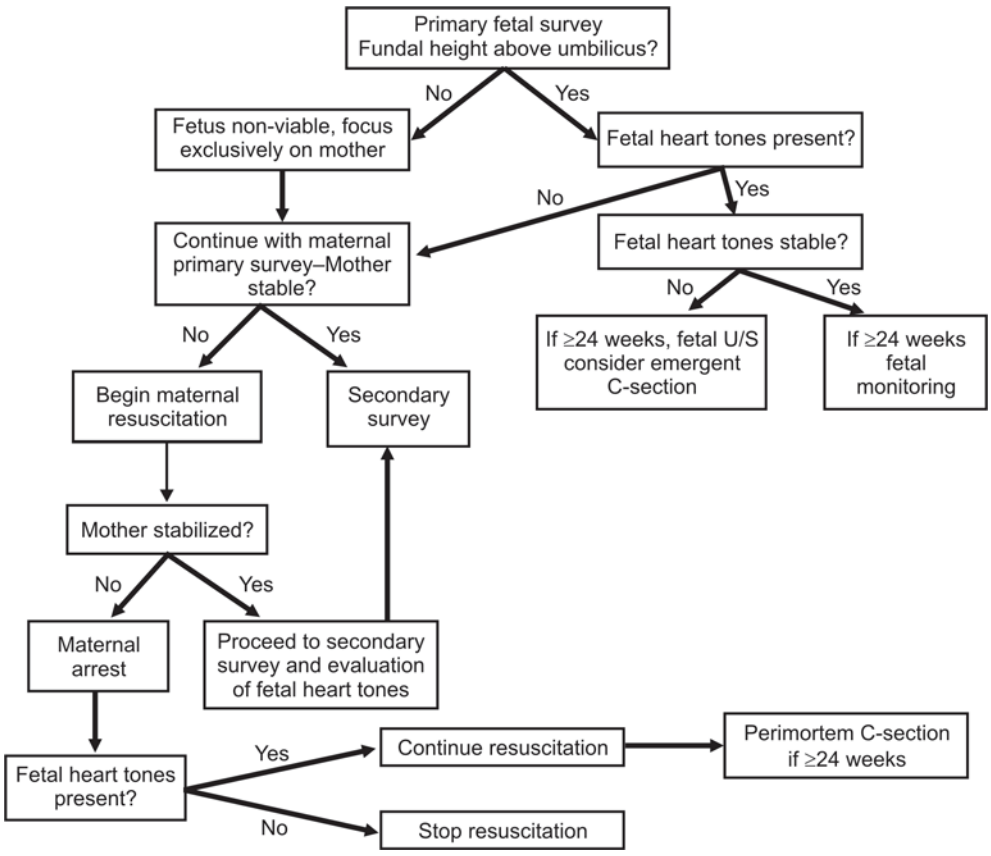

Fig. 1: Decision making algorithm for resuscitation in pregnancy - adapted from Marx: Rosen's Emergency Medicine: Concepts and Clinical Practice, 6th ed. Ch 35, Trauma in Pregnancy

Table 5: Resuscitative medications in pregnancy

Drug	*Indications*	*Drug class*
Epinephrine	Cardiac arrest	Category C - may induce uteroplacental vasoconstriction.
Lidocaine	Ventricular ectopy, V-tach, and V-fib	Category C - may cause fetal bradycardia.
Atropine	Bradycardia, asystole	Category B - can cause fetal tachycardia.
Sodium bicarbonate	Cardiac arrest, metabolic acidosis	Category C
Dopamine	Hypotension	Category C - use only when clearly indicated.
Dobutamine	Depressed myocardial contractility	Category C - use only if clearly indicated
Amiodarone	V-fib tachycardia, and SVT	Category D - serious fetal adverse effects have been observed.
Adenosine	SVT	Class C
Magnesium sulfate	AMI, torsades de pointes, toxemia, tocolysis	Class B - neonatal neurologic depression may occur

Table adapted from Datner, E. Promes, S. Tintinalli's Emergency Medicine. Resuscitation in Pregnacy: The McGraw-Hill Co. 2006. ch 16.

many causes of maternal arrest, both as a direct complication of the pregnancy, and preexisting disease (Table 6). The following is a list of the causes of cardiopulmonary arrest in pregnancy. The more common causes will be discussed in further detail.

Table 6: Obstetric and non-obstetric causes of cardiac arrest in pregnancy

Obstetric causes	*Non-obstetric causes*
Hemorrhage (17%)	Pulmonary embolism (19%)
Pregnancy induced hypertension (16%)	Infection/sepsis (13%)
Idiopathic peripartum cardiomyopathy (8%)	Stroke (5%)
Anesthetic complications (2%)	Myocardial infarction
Amniotic fluid embolism	Trauma

Hemorrhage

Obstetric hemorrhage is responsible for an estimated 25 percent of maternal death in pregnancy[9]. While hemorrhage is most common during delivery and immediately postpartum, antepartum hemorrhage is also prevalent. Causes of antepartum hemorrhage include abruptio placenta, placenta previa, and uterine rupture. Although the obstetric patient is well prepared for hemorrhage due to the physiologic changes of pregnancy, there is also a greater risk of extensive blood loss from the gravid uterus. Pregnancy induces a hypervolemic state and a mother can experience massive blood loss before she shows any significant changes in vital signs. If these conditions are not recognized and treated early, it can result in loss of both the fetus and the mother. Rapid assessment and initiation of appropriate resuscitative measures can greatly improve prognosis.

Placental abruption is the premature separation of the placenta from the uterine wall. Increased risk is associated with prior abruption, trauma, hypertension, cocaine abuse, smoking, premature rupture of membranes, polyhydraminos, and high parity. Abruption is thought to occur because the elasticity of the uterus allows it to deform easily without injury. However, when the uterus changes shape, the inelastic placenta is unable to conform, and is torn from the uterine wall. This causes hemorrhage between the uterine wall and the placenta. As a result, fetal oxygen and nutrient supply is reduced, and waste removal can be inadequate. Intrauterine hemorrhage leads to irritation of the myometrium and the uterus begins contracting. These contractions cause constriction of uterine blood vessels resulting in a greater decrease in blood flow to the already distressed fetus. There does not appear to be a correlation with abruption, and the location of the placenta, but the extent of abruption does correlate with the rate of fetal loss. Even a small abruption can induce premature labor, but the larger the abruption, the greater the risk to the fetus. Signs and symptoms of abruption include vaginal bleeding, uterine tenderness, abdominal cramps, maternal signs of hypovolemia, and fetal tachycardia. Although hemorrhage is usually apparent, up to 2.5 L of blood may be concealed between the myometrium and the placenta.[9] Thus, the patient can become hemodynamically unstable with what appears to be a minimal loss of blood volume.

Placenta previa is the implantation of the placenta near or over the cervical os. It can result in maternal hemorrhage, but rarely becomes life threatening. Risk factors include prior previa, uterine scars, multi parity, and advanced maternal age. Bleeding occurs near the end of pregnancy with cervical effacement and dilation. As the lower uterine segment elongates, the placenta is separated from the uterine wall and bleeding occurs.

Diagnosis is made by transabdominal or transvaginal ultrasound which should be done prior to performing a pelvic examination. If the placenta previa is partial, the uterus will stretch, and previa will resolve. As long as the mother remains hemodynamically stable, the pregnancy can be carried to term. Delivery must be via cesarean section to prevent massive hemorrhage at delivery. If the mother can not be stabilized, replacement of fluids and immediate cesarean is indicated.

Uterine rupture is rare, but is associated with nearly a 100% fetal mortality rate[2]. The probability of rupture increases as the pregnancy progresses and the uterine walls are stretched. Women with a history of prior cesarean section are at highest risk of rupture from separation of the scar. The most common cause of uterine rupture is trauma. Uterine rupture should be considered when there is an inability to palpate the top of the uterus, and fetal parts are easily felt through the abdominal wall. Abdominal tenderness may or may not be present. Management is considered on an individual basis. If the mother desires more children in the future, repair of the uterus can occasionally be accomplished. However, with extensive damage to the uterus or inability to repair damaged vessels, hysterectomy is the optimal treatment.

Any time there is maternal hemorrhage, there is a potential for fetomaternal transfusion. Every patient with the potential of fetomaternal hemorrhage should have the blood type with Rh status determined immediately. When fetomaternal hemorrhage is suspected in an unsensitized Rh negative patient, the Kleihauer-Betke (KB) or the Apt tests may help in the diagnosis and management. The KB test provides a quantitative estimate of fetal blood cells present in the maternal circulation. This is used for appropriate dosing of Rh-immune globulin. Acid elution of the sample causes the loss of hemoglobin in the maternal red blood cells, leading them to appear pale (ghost cells). Fetal red blood cells are stained in the process and have a bright pink appearance on microscopic examination. Fetal cells are counted, and an estimate of fetal to maternal blood transfusion can be made. The standard dosing of Rh-immune globulin is an initial 300 µg followed by an additional 300 µg for every 30 mL of estimated fetal blood transfused. This test should be repeated in 24-48 hours to evaluate for continued fetomaternal hemorrhage[1,2]. The KB test is very advantageous when significant fetomaternal hemorrhage occurs, but the sensitivity of the test drops substantially when there is less than 5 µL of fetal blood. The presence of as little as 0.1 µL of fetal blood can lead to isoimmunization of the mother[1,2]. The Apt test is a qualitative test used for the identification of any amount of fetal blood in the maternal circulation. When the Apt test is positive in an Rh negative mother, a single 300 µg dose of Rh-immune globulin should be administered to prevent sensitization[1].

Pulmonary Embolism

Pulmonary embolism accounts for approximately 20 percent of maternal deaths[10]. The majority of patients with pulmonary emboli are asymptomatic. Nonetheless, it is still the most common cause of acute hemodynamic and respiratory collapse in pregnancy[10]. The most common forms of emboli seen in pregnancy are thrombotic and amniotic fluid. These can enter the pulmonary circulation and result in ventilation / perfusion (V / Q) mismatch and hypoxia, with resultant cardiac arrest. Rapid assessment and initiation of resuscitative measures will reduce morbidity and mortality.

The risk of thromoembolism is increased by 5 to 10 fold as a result of venous stasis, hypercoagubility, and vascular damage seen in pregnancy (virchows triad). Contributing risk factors include; prior thromboembolism or family history, advancing maternal age, increased parity, obesity, immobility, trauma, or recent surgery[10]. When present, signs and symptoms of pulmonary embolism include pleuritic chest pain, cough, dyspnea, tachypnea, and tachycardia.

Diagnostic studies are ordered based on physical exam findings and clinical suspicion. Ultrasound of the lower extremities is safe for the fetus. CT angiography (CTA) of the pulmonary arteries in the chest is now the preferred method for the diagnosis of pulmonary embolism. CT pulmonary angiography delivers a minimum radiation dose of 2.0 rad[12]. Appropriate shielding can protect the fetus from unwanted exposure. When facilities for CTA are not available or there is allergy to intravenous contrast, V / Q scan becomes the modality of choice. V / Q scans deliver a minimal amount of radiation to the fetus, and as such are safe to use in pregnancy. However, the majority of V / Q scans are intermediate risk or indeterminate. In these cases, there is a 20% chance the patient has a pulmonary embolus[10]. If venography is necessary to make a definitive diagnosis, the pelvis should be shielded to protect the fetus from radiation. It should also be noted that venography has the potential to induce venous thrombosis, and so its use in pregnancy should be weighed against the potential complications to both mother and fetus. Magnetic Resonance Imaging (MRI) may show pulmonary emboli by using standard or special gated techniques. MR Angiography can also be performed by

using intravenous administration of gadolinium. Overall MRI has a sensitivity of 85% and specificity of 96% but is not adequate to diagnose sub segmental emboli[14].

The goal of treatment of pulmonary embolism in pregnancy is maintaining adequate oxygenation and circulation. In addition to supportive treatment, heparin should be started prior to a definitive diagnosis when pulmonary embolism is suspected. Pregnancy is a relative contraindication to thrombolytic therapy. However, it has been shown to be beneficial in some cases. It should only be used in severe cases and after careful evaluation of the risks and benefits.

Amniotic Fluid Embolism

Amniotic fluid embolus is most common immediately following delivery, but it has the potential to complicate pregnancy at any time. It is not a common complication, but is associated with a high mortality rate. It is estimated that 50 percent of patients die within one hour of initial onset, and of those who survive, less than half are neurologically intact[10]. Risk factors for developing amniotic fluid embolism include: difficult labor, advancing maternal age, multiparity, rupture of membranes, amnioinfusion, trauma, placental abruption, ruptured uterus, and fetal death. Unlike many complications in pregnancy, prior amniotic fluid embolism does not appear to increase future risk.

Amniotic fluid embolism is actually an anaphylactoid reaction, and not a true embolic event. When foreign debris from the amniotic fluid enters the maternal circulation, a systemic inflammatory response is initiated. This typically presents with maternal respiratory distress and hypotension, but can also present with shock, pulmonary edema, seizure, confusion, or coma. Those patients who survive the initial insult often go on to develop disseminated intravascular coagulopathy (DIC), and ultimately multi system organ failure. Treatment is supportive, with concentration on maintenance of oxygenation and circulation to minimize long term sequelae.

Disseminated Intravascular Coagulopathy

Disseminated intravascular coagulopathy (DIC) is a potentially life threatening complication in pregnancy. Normal hemostasis is accomplished by a balance between coagulation factors and their inhibitors as well as thrombus formation and lysis. DIC is caused by a systemic activation of the coagulation cascade disrupting normal hemostasis. Instead of the typical local reaction to vascular damage, the body begins to uncontrollably form and lyse clots throughout the system. This results in a consumptive coagulopathy, and leads to hemorrhage. Platelets are destroyed and fibrin plugs are formed which can block small vessels leading to ischemia. The ischemic changes cause further vascular damage and continuation of the coagulation cascade. Common triggers for DIC include placental abruption, fetal demise, amniotic fluid embolism, septic shock, and transfusion reactions. When diagnosing DIC, special attention should be given to the platelet value and the fibrinogen level. If these values are low, the suspicion of DIC should be high and further evaluation is needed. Treatment should focus on the underlying etiology. Most cases of DIC in pregnancy are self-limited and resolve with elimination of the trigger. If hemorrhage is extensive, patients will often benefit from replacement of platelets, clotting factors, fibrinogen, and red blood cells.

Eclampsia

Severe hypertension, preeclampsia, and eclampsia are a continuum of potential complications in pregnancy. Risk factors for pregnancy-induced hypertension include a family history, teen pregnancy, first pregnancy, twin pregnancy, molar pregnancy, and obesity. Preeclampsia is hypertension (usually after 20 weeks' gestation) with the presence of protein in the urine (greater than 300 mg / 24 hours)[4]. Eclampsia adds seizure activity. The cause of these various scenarios in pregnancy is unknown, but is thought to be related to vasospasm induced by maternal hormones (Marx). Patients can present with a wide variety of signs and symptoms. Rapid weight gain, headache, visual disturbances, seizure, abdominal pain, nausea, vomiting, edema, hypertension and hyperreflexia can all be seen in the continuum of this disease. Eclampsia should be considered in any patient 20 weeks gestation or greater who presents with seizures. Initial management is similar to management of any seizure patient. Secure the airway, and obtain intravenous access for administration of medication. Although the mechanism of action is unknown, magnesium sulfate is the drug of choice for eclamptic seizures. Other possible causes of seizure activity such as hypoglycemia or intoxication should be ruled out as well. Once the patient is stabilized further testing is necessary to document the severity of the disease. Laboratory tests should be drawn to evaluate possible end organ damage, and look for signs of HELLP syndrome (H – hemolysis, EL – elevated liver enzymes and LP – low platelets). Hypertension typically resolves with the cessation of the seizure. If the diastolic blood

pressure remains higher then 105 mmHg, it may be necessary to administer an antihypertensive. Care must be taken not to lower the pressure too much as this will cause placental hypoperfusion and fetal compromise. Hydralazine in the drug of choice in pregnancy, but nimodipine and labetalol can also be used[11].

Aortic Dissection (AD) in Pregnancy[13]

Aortic dissection during pregnancy is potentially lethal. Both the mother and the fetus are at risk. The complex management is dependent upon the type of dissection and the gestational age of the fetus. The Stanford classification divides aortic dissections into two types: Type A involves the ascending aorta and type B, involves the aorta distal to the origin of the left subclavian artery. Type B dissections in pregnancy are rare. The treatment of Type B dissections is medical, with strict control of blood pressure. Type A dissections require emergent surgery. In a 12-year review of management of acute aortic Type A dissections complicating pregnancy, researchers have concluded treatment should be aimed at saving the life of the mother and the fetus. Prior to 28 weeks of gestation, aortic repair is recommended with the fetus in utero. If the fetus is more than 32 weeks of gestation a primary cesarean section with simultaneous aortic repair is advised. There is a dilemma when the fetus is between 28 and 32 weeks gestation. Here the strategy is determined by the fetal condition.

The diagnosis of AD is often missed in pregnancy. The most common presenting feature of AD in pregnancy is acute onset of severe back or chest pain. This chest pain is characteristically stabbing, tearing or ripping. The pain migrates in the path of propagation of the dissection. The dissection and the signs of presentation can mark the path of propagation. End organ ischemia can occur from obstruction of arteries originating from the aorta. The diagnosis of AD must be confirmed by investigations like chest radiography, echocardiography, contrast-enhanced computed tomography, aortography and magnetic resonance imaging. Chest radiography lacks specificity but it can be used for the prediction of dissection when combined with the history and clinical findings. The radiographic feature of mediastinal widening occurs in less than half of the patients. Transesophageal Echocardiography can be used very effectively to diagnose AD in the chest. CTA of the chest and aortography have known doses of radiation exposure. Proper shielding can help to protect the fetus.

Approximately 50% of aortic dissections in women under 40 years of age occur in pregnancy or the puerperium. Pregnancy is considered as an independent risk factor for aortic dissection. The commonest site of pregnancy-associated AD is proximal aorta. Aortic rupture is found to occur in third trimester or the first stage of labor.

Disposition

Obstetrics and neonatology specialists should be involved early in the resuscitation. Once the patient is stabilized, care can be turned over to the appropriate specialist. If these specialties are not available at your hospital, the patient should be transferred to a nearby facility that can manage both patients. Most patients will be admitted to the obstetric service or the intensive care unit. Either continuous or intermittent fetal monitoring should be done throughout the hospital stay.

Summary

Cardiopulmonary arrest in pregnancy can be due to nonpregnancy related causes as well as pregnancy related causes. Resuscitation is a challenging task. The basic resuscitative measures used in any patient apply in pregnancy. There are several modifications that need to be made. A few important points to remember are as follows. Anatomy and physiology are altered in pregnancy which change the signs and symptoms of disease and interfere with resuscitative measures. Making stabilization of the mother the primary focus will offer the best fetal outcome. Fetal distress can offer early warning signs of deteriorating maternal status. No necessary test should be withheld from the mother for fear of fetal compromise. Specialty services should be consulted early in the process. With a basic knowledge of the changes that occur in pregnancy, appropriate treatments can be instituted, and two lives can be saved.

REFERENCES

1. Datner E, Promes S. Tintinalli's Emergency Medicine. Resuscitation in Pregnacy: The McGraw-Hill Co. 2006. ch 16 and 254.
2. Neufeld J. Trauma in Pregnancy: Marx editor. Rosen's Emergency Medicine: Concepts and Clinical Practices. 6th ed. Mosby; 2006. ch 35.
3. Morrison L. General approach to the pregnant patient: Marx editor. Rosen's Emergency Medicine: Concepts and Clinical Practices. 6th ed. Mosby; 2006. ch 176.
4. Houry D, Abbott J. Acute complications of Pregnancy: Marx editor. Rosen's Emergency Medicine: Concepts and Clinical Practices. 6th ed. Mosby; 2006. ch 177.
5. Shah A, Kilcline, B. Trauma in Pregnancy. Emerg Med Clin N Am 2003;21:615-29.

6. Mattox K, Goetzi L. Trauma in Pregnancy. Crit Care Med 2005; 33(10):385-9.
7. American Heart Association. 10-8 Cardiac arrest associated with pregnancy. Circulation Journal of the American Heart Association 2005;112:150-3.
8. Clark S. Critical Care Obstetrics: In: James S, et al (Eds). Danforth's Obstetrics and Gynecology. Lippincott Williams and Wilkins; 2003 Ch 25.
9. Crochetiere C. Obstetric Emergencies. Anesthesiology Clin N Am 2003; 21:111-25.
10. Gei A, Vadhera R, Hankins G. Embolism during pregnancy: thrombi, air and amniotic fluid. Anesthesiology Clin N Am 2003;21:165-82.
11. Martin J, et al. Understanding and managing HELLP syndrome: The integral role of aggressive glucocorticoids for mother and child. American Journal of Obstetrics and Gynecology 2006; 95:914-34.
12. Parker MS, Ferdinand K Hui, Marc A Camacho, Jiyearn K Chung, Dean W Broga, Narinder N Sethi. Female Breast Radiation Exposure During CT Pulmonary Angiography. AJR 2005;185:1228-33.
13. Lewis S, Ryder I, Lovell AT. Peripartum presentation of an acute aortic dissection. British Journal of Anaesthesia 2005; 94(4):496-9.
14. Sharma S. Pulmonary Embolism. E-Medicine website. Web address: http://www.emedicine.com/med/topic1958.htm