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INVITED REVIEW ARTICLE

Critical Care in Obstetrics: Where are We

Sushil Chawla[1] · **T. Jose**[1] · **Manish Paul**[2]

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About the Author

Dr. Sushil Chawla is working as a dedicated teacher in Indian Navy at Armed Forces Medical College, Pune. He has a special interest in high-risk obstetrics and fetal medicine.

Abstract Maternal mortality is disastrous news for the society, family, newborn, and the obstetrician. Yet, we all who are care providers to these apparently healthy women carrying another life within them are dumbfounded by the clinical conditions arising due to the pregnancy or the effects of the pregnancy, that it becomes difficult to provide an ideal care to them. The rapid uprising of a condition and the worsening of commonly occurring benign conditions—preeclampsia, hemorrhage, etc., necessitates that all obstetricians are well versed with the physiological changes and should be able to not only provide the best of obstetric care to the mother and the newborn but also perform or assist in performance of life-saving procedures.

keywords Maternal mortality · Critical Care in obstetrics · Amniotic fluid embolism · Physiological changes in pregnancy · Perimortem cesarean section

Surg Capt Sushil Chawla is an Associate Professor in Department of Obstetrics and Gynaecology at AFMC, Pune, India; Col T. Jose is an Associate Professor in Department of Obstetrics and Gynaecology at AFMC, Pune, India; Lt Col Manish Paul is an Associate Professor in Department of Anaesthesiology and Critical Care at AFMC, Pune, India.

✉ Sushil Chawla
chawla_sushil@rediffmail.com

[1] Department of Obstetrics and Gynaecology, AFMC, Pune, India

[2] Department of Anaesthesiology, Pain & Critical Care, AFMC, Pune, India

Introduction

Pregnancy, with its potential hazards, has the opportunity to produce life-threatening complications. Hypertensive disorders, hemorrhage, and infection all occur frequently

during pregnancy and require special care, but they do not become critical illnesses, and the same was cited by *World Health Organization* in the fact book on maternal mortality. Lack of systematic surveillance for the points of critical care in these conditions and the outcomes have led to poor development of parameters for critical care during pregnancy [1–3]. The preexisting medical diseases, changing age of gravidas, availability of assisted reproductive techniques, pregnancy post-organ transplant, and complications due to obesity complicate the care and necessitate the need for critical care in the obstetric population.

1–3% of the obstetric population require critical care. The majority of these are for respiratory support antenatally and for hemodynamic instability in the postpartum period. Obstetric and medical complications of pregnancy share equal proportions in all admissions to the ICU. The level of care provided and the place to be provided may vary from center to center depending on the organ systems requiring support [2–4].

This article focuses on the effect of pregnancy on cardiopulmonary resuscitation (CPR) and several important disorders of pregnancy. The objective of CPR is to maintain adequate vital organ perfusion and oxygenation. Sudden cardiac arrest (SCA) in pregnancy is usually associated with peripartum events like obstetrical hemorrhage and infection, and the delivery of high-quality CPR has a significant impact on the survival rates. Thus, obstetrical units should have proper resuscitative equipment and personnel trained in CPR.

If breathing or the heart stops, by 4 min, brain damage may occur, and after 6 min, brain damage will almost always occur. The goals of CPR are achieved by remembering the "ABCDs" of the primary and secondary survey. The primary survey consists of airway management using noninvasive techniques, breathing with positive-pressure ventilations, and performing CPR. A secondary survey requires the use of advanced, invasive techniques to resuscitate, stabilize, and transfer the patient if indicated.

The mechanical and physiologic changes of pregnancy impact every phase of the resuscitation process as mentioned [5–8] in Table 1.

Airway

The aim is to facilitate the airway access with simple head tilt and chin lift and removing any foreign body. The airway obstruction in the first half of pregnancy can be relieved with the abdominal thrusts, but in the latter half of pregnancy, the gravid uterus necessitates the use of chest thrusts instead. Chest thrusts require placing the thumb side

Table 1 Physiologic changes of pregnancy

Characteristics	Change	Effect on resuscitation method
GIT		
Motility	Decrease	Increased risk of aspiration
LES tone	Decrease	Need to protect airway
Hematologic		
Clotting factors	Increase	Increased VTE
Cardiovascular		
Cardiac output	Increase	Increases circulatory demand
Blood volume	Increase	Physiological anemia with decreased oxygen carrying capacity
Heart rate	Increase	Pulmonary edema
SVR	Decrease	
COP	Decrease	
Aortocaval compression	Increase	Lateral uterine displacement required to maintain venous return and hence cardiac output
Respiratory		
Pharyngeal edema	Increase	Smaller endotracheal tube
		Difficulty with intubation
Minute ventilation	Increase	Increased development of hypercarbia
Oxygen consumption	Increase	More rapid development of hypoxia
FRC	Decrease	
PaCO2	Decrease	
Chest Wall		
Compliance	Decrease	More difficult intubation
Bicarbonate	Decrease	Decreased acid buffering capability

of the fist on the middle of the sternum, avoiding the xiphoid and the ribs. The rescuer then grabs his fist with the other hand and performs chest thrusts. Up to five abdominal or chest thrusts are given followed by repetition of the jaw-lift, foreign body removal, and attempted ventilation. These steps are repeated until effective or until a surgical airway can be obtained. If, after clearing obstruction, the patient is unresponsive but breathing spontaneously, she is placed on her left side and head tilted back.

Enlarged breasts and pharyngeal edema in pregnancy can make the airway access and maintenance difficult in pregnancy. Endotracheal intubation is the preferred method for maintaining airway patency, although laryngeal mask airway may be used if endotracheal intubation fails [9]. It may become necessary to use a smaller endotracheal tube than normal due to edema and increased chances to reflux and aspirate due to lowered tone of esophageal sphincter. Effective preoxygenation helps to avoid hypoxia.

Breathing

Rescue breathing may occur mouth to mouth, mouth to nose, mouth to mask or by endotracheal intubation. The current guidelines call for a ratio of 2 ventilations to 30 compressions in one- or two-person CPR, pausing for ventilations in the absence of an advanced airway.

The expanding breast tissue, displaced diaphragm, and progesterone cause decreased chest wall compliance, decrease in the functional residual capacity (FRC), and increased minute ventilation. The decrease in FRC and increase oxygen demand predispose her to rapid decrease in arterial and venous oxygen tension during periods of hypoxia and thus increasing maternal carbon dioxide levels and promote fetal acidosis. This makes resuscitation of the mother and fetus difficult.

Circulation

In late pregnancy, aortocaval compression by the uterus exerts pressure on the inferior vena cava, common iliac vessels, and abdominal aorta causes sequestration of circulating blood volume, decreasing venous return, causing supine hypotension, and posing an obstruction to forward blood flow and decreasing effectiveness of thoracic compressions. Thus simple displacement of the uterus by the resuscitative team manually or using a wedge helps in resuscitation. One should avoid using lower limb veins for infusing drugs as it does not reach the heart until the baby is delivered.

Chest compressions should be given at a rate of approximately 100/min. Defibrillators can be used with same dose and voltage without significant effect on the fetus. An effective CPR requires adequate quality and quantity of blood in the circulatory system, and this is ensured by transfusion and control of hemorrhage.

The change in the circulating blood volume and increased GFR may alter the distribution and metabolism of various drugs thus necessitating the use of standard or a higher dose of the drugs. Certain drugs which might be contraindicated for chronic use in pregnancy may be life-saving in this acute situation.

It is necessary to assess the fetal heart status along with the assessment of maternal circulation, and if the fetus is not alive, one should give all efforts for maternal survival and ignore the fetus [9].

Acute Fatty Liver of Pregnancy

Acute fatty liver of pregnancy (AFLP) or acute yellow atrophy is a rare, yet potentially fatal complication of late pregnancy, occurring 1 in 10,000 pregnancies. The condition occurs more commonly in primigravida and multiple gestations [10].

The presentation is non-specific with nausea, vomiting, anorexia, tachycardia, fever, headache, pruritus, and abdominal pain. Preeclampsia is seen in 70% cases. Some women present with decreased fetal movements, uterine contractions, and vaginal bleeding. In AFLP, neurological dysfunction begins early and rapidly progress from restlessness, confusion, and disorientation to psychosis and coma. It can cause fulminant hepatic failure, acute renal failure, infection, pancreatitis, gastrointestinal hemorrhage, coagulopathy, and hypoglycemia. Serum transaminases are usually between 100 and 1000 U/L and bilirubin generally > 5 mg/dL. Liver function tests usually return to normal 4–8 weeks after delivery.

These women need intensive care setting, and there is no role of expectant management. The route of delivery is based on the fetomaternal condition and the Bishop's score. Epidural anesthesia is the best option as it preserves hepatic blood flow without any hepatotoxic effects Supportive care includes providing 2000 calories per day, mainly as glucose. Enemas are given for colonic emptying, and one omits the drugs requiring hepatic metabolism. H2 blockers and antibiotics help to reduce the GI bleeding and concomitant infections. Presence of hemorrhagic complications or if surgery is contemplated requires correction of coagulation abnormalities with blood components [10].

Amniotic Fluid Embolism (AFE)

The term "amniotic fluid embolism" is a misnomer and should be designated as "anaphylactoid syndrome of pregnancy." AFE is characterized by hypoxia, hemodynamic collapse, and coagulopathy. There is a 10-fold variation in estimates of incidence and mortality, because

the condition is rare, and there is no gold standard for diagnosis [11].

In a typical case, a patient in labor or having just undergone Cesarean delivery or immediately following vaginal delivery or pregnancy termination presents with acute profound hypoxia and hypotension followed by cardiopulmonary arrest and consumptive coagulopathy, leading to exsanguination. Maternal outcome is dismal with mortality of 80% and only 15% survive neurologically intact.

The diagnosis of AFE remains clinical. Other markers for AFE, such as serum tryptase, pulmonary mast cell antitryptase, serum TKH-2 antibody to fetal antigen sialyl Tn, serum complement, and plasma zinc coproporphyrin provide no definitive means of diagnosing or excluding AFE.

The initial treatment for AFE is supportive. CPR should be provided. Oxygen is given at high concentrations. One has to ensure adequate cardiac preload using fluids and vasopressors as LVF is commonly seen. High-dose steroids, extracorporeal membrane oxygenation with intra-aortic balloon, cardiopulmonary bypass, and nitric oxide have been used and reported in survivors [11, 12].

Severe Preeclampsia

The preeclampsia syndrome due to its pathophysiology of increased capillary permeability, extensive vasospasm, microangiopathy, and generalized inflammatory response causes damage to maternal cardiovascular, renal, hematologic, neurologic, and hepatic systems, thereby resulting in eclampsia, HELLP syndrome, pulmonary edema, ARF, etc.

The basic principles in the management of eclampsia include nursing the patient in lateral position or placing a wedge under the right hip, oxygen administration after securing the airway of the patient, and putting an indwelling urinary catheter and monitoring urine output. The involvement of the senior obstetrician and intensive monitoring of the vital signs helps in favorable outcome in this condition.

1. Crystalloid infusions of normal saline or lactated Ringer's solution at 80–100 mL/h. Carefully controlled crystalloid infusions appear to be the safest mode of fluid therapy in severe preeclampsia. Close monitoring of fluid intake and output and hemodynamic parameters must be undertaken to prevent an imbalance of hydrostatic and oncotic forces which may lead to pulmonary edema. Thus, putting it simply—"Keep the patient dry."
2. Injection of magnesium sulfate prevents eclamptic seizures via neuromuscular blockade or central action. The retinal vessels form an easy access for the clinician to see the status of all vessels. Belfort et al. studied magnesium sulfate's effect on small vessels in the cerebral circulation and found that it causes vasodilatation, reduces cerebral perfusion pressure, and helps in maintaining cerebral blood flow. This led to the formulation of Pritchard's, Sibai, Zuspan, and other commonly practiced regimens. We employ a regimen of a 4-g IV loading dose over 20 min, followed by a 1 g/h continuous IV infusion. The Eclampsia Trial Collaborative Group compared anticonvulsant regimens of magnesium sulfate, phenytoin, and diazepam and concluded that women given magnesium sulfate had a lower risk of recurrent convulsions, requirement of mechanical ventilation and ICU admissions. Cochrane review found magnesium superior to lytic cocktail, diazepam, and phenytoin for prevention of eclampsia. A randomized clinical trial compared standard 24-h treatment of magnesium with discontinuation upon the onset of maternal diuresis and showed no untoward outcomes or need for re-initiation of treatment [13]. The use of MgSO4 requires the monitoring for the toxicity by simple instructions of monitoring the urine output, patellar reflex, and respiratory rate.
3. Sharma et al. compared intravenous labetalol with intravenous hydralazine and found that labetalol had a quicker onset of action. An initial dose of 10 mg is given and is followed by progressively increasing doses (20, 40, 80 mg) every 10 min, to a total dose of 220 mg. Alternately, intravenous infusion may be started at 1–2 mg/min until therapeutic goals are achieved and then decreased to 0.5 mg/min or completely stopped. [17] Oral nifedipine 10 mg repeated if required after 30 min and given every 3–6 h is the alternate choice. When using nifedipine in patients receiving concomitant magnesium sulfate, one should watch for the possibility of an exaggerated hypotensive response [14].

Severe systemic and pulmonary hypertension during endotracheal intubation and extubation, in patients undergoing Cesarean section may be watched for while patients undergoing epidural anesthesia for Cesarean section maintained stable systemic and pulmonary arterial pressures. Deleterious hypotension may be avoided by lateral maternal tilt and preloading with crystalloid solution.

Pulmonary edema

This appears to be multifactorial as elaborated in Table 2. The condition is best explained at the tissue level (pulmonary capillaries) by Starling's equation. The imbalance

Table 2 Pulmonary edema causes

Category	Risk factors
Pregnancy related causes	Sepsis
	Preterm labor
	Amniotic fluid embolism
	Pulmonary embolism
Pre-pregnancy medical causes	IHD, RHD
Drugs	β-Agonists tocolytics
	Corticosteroids
	Magnesium sulfate

of hydrostatic pressure, colloid oncotic pressure, and capillary permeability is the end cause of pulmonary edema.

The primary interventions are aimed at reducing the preload and afterload and prevent ischemia of heart. It is important and difficult to differentiate cardiogenic from non-cardiogenic pulmonary edema [15]. Management includes upright positioning and oxygen administration along with fluid restriction with echocardiographic assessment of the heart. The management differs depending on hypertensive or normotensive patient. In a hypertensive patient, the cause is generally pre-eclampsia with fluid overload and/or drugs and thus the management involves nitroglycerine with frusemide along with magnesium sulfate and calcium channel blockers. In a hypotensive or normotensive patient, the cause is generally a preexisting cardiac condition along with other conditions like amniotic fluid embolism, sepsis, and tocolysis. The management in these cases involves vasopressor support and frusemide. Invasive mechanical ventilation may be considered in both scenarios if stability is not maintained. Close monitoring with arterial blood gas analysis, an indwelling catheter to follow urine output, and serum electrolytes is necessary [16].

HELLP syndrome

The term was coined by Weinstein in 1982, but the hematologic and hepatic abnormalities were described by Pritchard in 1954. HELLP syndrome may be the imitator of a variety of non-obstetric medical entities. The majority of patients present with malaise, nausea (with or without vomiting), and epigastric pain. Peripheral smears show schistocytes with polychromasia, and thrombocytopenia. Clotting parameters are generally normal. SGOT and SGPT are rarely in excess of 1,000 IU/L; however, HELLP syndrome progressing to liver rupture is associated with markedly elevated hepatic transaminases. An upward trend in platelet count and a downward trend in lactate dehydrogenase concentration should occur in patients without complications by the fourth postpartum day. Corticosteroids have been proposed for the treatment of postpartum HELLP syndrome; a metanalysis found that women given dexamethasone demonstrated significantly better outcome.

Septic Shock

Septic shock results from the systemic inflammatory response to an infectious insult and is characterized by an inability to maintain vascular integrity and fluid homeostasis causing inadequate tissue oxygenation and circulatory failure. Physiologic changes in pregnancy such as an increase in pelvic vascularity promote maternal survival after infection, but may also predispose the gravid female to more significant infectious morbidity as is the case with urinary stasis. The fetus is more resistant to the endotoxin, but changes in uteroplacental flow cause hypoxia, acidosis, cerebral injury, and fetal demise. The data regarding septic shock and ICU management in pregnancy are limited. Predisposing maternal factors are described in Table 3.

A high index of clinical suspicion in the appropriate clinical setting is needed as there is no definitive diagnostic test [17]. The staff managing the patient must be conversant with MEOWS charting of vital signs (pulse, respiratory rate, blood pressure, and temperature). Signs and symptoms suggestive of peripartum sepsis are described in Table 4. It is worth mentioning that the severity of sepsis may not be indicated by the level of derangement of signs and symptoms. Laboratory tests are sent in an effort to identify potential causes for shock and early signs of organ failure. Leukocytosis with a left shift may suggest the

Table 3 Risk factors for peripartum sepsis

Diabetes
Obesity
Immunosuppression
Anemia
Vaginal discharge
History of PID
Invasive procedures like amniocentesis
Cervical encerclage
Prolonged PROM

Table 4 Features of peripartum sepsis

Tachycardia and tachypnea
Hyperpyrexia/hypothermia
Hypoxemia
Hypotension
Reduced urinary output
Delirium

diagnosis of bacterial infection, but overwhelming septic shock may be associated with leucopenia. Thrombocytopenia may be an early sign of DIC and is an independent predictor of multiorgan failure and poor outcome. An elevated D-dimer levels and increasing circulating lactate levels are associated with the development of septic shock and death, while declining levels in response to therapy are a good prognostic feature. These patients should have at least two blood cultures obtained from separate sites and provisional results from these help in the selection of antimicrobial therapy. High concentration of inflammatory mediators as serum procalcitonin are usually increased in sepsis, but may also increase in non-septic conditions, the high negative predictive value of a normal serum procalcitonin level (< 0.25 μ g/L) can be used to exclude a diagnosis of septic shock and thereby avoid unnecessary use of antibiotics. The trend of serial procalcitonin monitoring is utilized in the escalation/de-escalation of antimicrobials. Serum lactate has been described as a potential biomarker for recognition of early sepsis in maternal sepsis (Tables 5, 6).

The initial management focuses on maternal resuscitation as this has a positive effect on the fetal condition. A Cesarean in patient with hemodynamic instability, increases the risk of maternal mortality with the exception of chorioamnionitis, where emergency Cesarean to drain the infection is indicated. One should aim for CVP 8–12 mmHg, MAP $\geq$ 65 mmHg, and urine output $\geq$ 0.5 mL/kg/h with the aggressive use of volume replacement and inotropic support to treat hypotension. Blood transfusion, inotropic agents, and supplemental oxygen help to meet the increased metabolic needs for oxygen. Noradrenaline is preferred over dopamine as a first line therapy of maternal septic shock.

Two most common bacterial etiologies of fatal maternal sepsis include E Coli and Group A and Beta hemolytic streptococci. Empiric antimicrobial therapy should include cover for Gram-negative and Gram-positive bacteria and anaerobic bacteria. Culture results and organism sensitivities are used to more selectively guide subsequent antimicrobial therapy. Uterine evacuation in septic abortion, hysterectomy in postpartum sepsis, and aggressive surgical debridement in necrotizing fasciitis may be required in obstetric practice with supportive care in an ICU. If septic pelvic thrombophlebitis is entertained, treatment with heparin with antibiotics is indicated.

Adequate nutrition to prevent translocation of bacteria from the gut to the systemic circulation and stress ulcer prophylaxis help in early recovery. The Surviving Sepsis Campaign Guidelines for the management of severe sepsis and septic shock recommend the use of moderate doses of hydrocortisone [18]. Steroid therapy should be, however, restricted to severe septic shock requiring high dose of inotrope/vasopressor therapy and should be promptly tapered off once the inotropes have been withdrawn [20].

Hypovolemic shock

Hypovolemic shock—hemorrhagic or non-hemorrhagic—is not infrequent in obstetric critical care practice. The altered physiology of an obstetric patient warrants early intervention. The causes could be antepartum (ruptured ectopic pregnancy, placental abruption) or postpartum (atonic uterus). The patient may present in a fully compensated, partly compensated, or non-compensated and irreversible shock, depending on the amount and the rate of blood loss.

The blood loss leads to acute sympathetic activation thereby causing peripheral vascular constriction, poor perfusion, and increased myocardial contractility. This increases the cardiac oxygen demand and deranged oxygen carrying capacity leads to worsening of vital organ perfusion and metabolic acidosis. The triggering of humoral and cellular inflammation further worsens the cellular injury. The clinical evaluation is aided with laboratory and radiological assessment. The baseline samples for cross-match have to be taken along with samples for hematology, renal assessment, and liver status. The arterial blood gas analysis (ABG) and lactate samples will aid in the assessment of response to therapy.

An echo evaluation of left ventricle filling and IVC collapsibility index can be done. In moderate-to-severe

Table 5 Classification of hemorrhage

Parameter	I	II	III	IV
Blood loss (ml)	< 750	750–1500	1500–2000	> 2000
Blood loss (%)	< 15%	15–30%	30–40%	> 40%
Pulse rate (beats/min)	< 100	> 100	> 120	> 140
Blood pressure	Normal	Decreased	Decreased	Decreased
Respiratory rate (breaths/min)	14–20	20–30	30–40	>35
Urine output (ml/h)	> 30	20–30	5–15	Negligible
CNS symptoms	Normal	Anxious	Confused	Lethargic

Table 6 Clinical features of hypovolemic shock

Organ system involved	Early shock	Late shock
CNS	Normal or altered mental status	Obtunded
CVS	Tachycardia	Cardiac failure and arrhythmias
Renal	Oliguria	Anuria
Respiratory	Tachypnea	Respiratory failure
Hematological	Anemia	Coagulopathy
Metabolic	Normal	Acidosis

Hypovolemic shock "kissing sign" of left ventricle may be seen. These could be repeated to assess the response to therapy.

Management of such a patient will include crystalloids, colloids, and blood (including blood products as indicated). Initial resuscitation is done with crystalloids at a rate of 10–20 ml/kg. This is followed by reassessment after which a decision of using colloids or blood may be taken. Threshold of use of blood and blood products is generally lower in obstetric population due to the already altered coagulation status during pregnancy. The principle to be followed is "*don't be too late or too less.*"

Peripartum cardiomyopathy

Cardiomyopathy develops in the last month of pregnancy or the first 5 months postpartum in a woman with no previous cardiac disease and after exclusion of other causes of cardiac failure and hence is a diagnosis of exclusion. Other peripartum complications, such as amniotic fluid embolism, arrhythmogenic, corticosteroid or sympathomimetic-induced pulmonary edema, need to be ruled out.

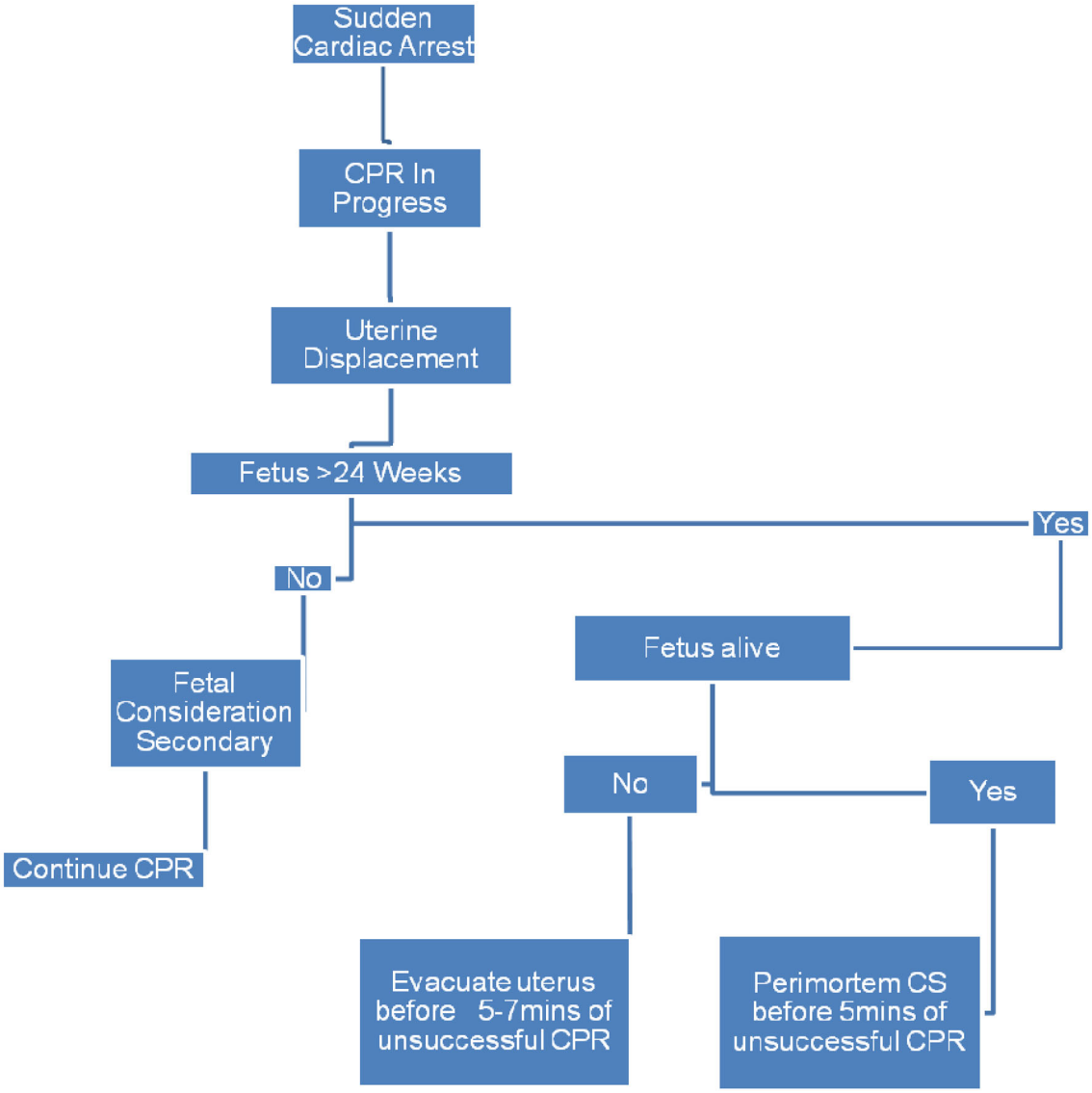
The incidence is estimated to be between 1 in 3000 and 1 in 4000 live births. Multiple gestations, multiparity, and preeclampsia are few risk factors with genetic predisposition. The entity of this condition is questioned due to non-specific clinical and pathologic findings. There is gradually increasing fatigue, dyspnea and with evidence of congestive heart failure—raised JVP, rales, and S3 gallop. ECG shows left ventricular and atrial dilatation, and chest X-ray shows features of cardiomegaly and pulmonary edema, and the mortality ranges from 25 to 50%. Echo also shows the dilation of the Lt side of the heart with decreasing ejection fraction.

The standard line of treatment includes oxygen, diuretics, afterload reduction with angiotensin-converting enzyme inhibitors (ACE)/angiotensin II receptor blockers (ARB)/hydralazine, β-blockers, and digoxin. The ACE/ARB agents should be avoided prenatally, but are a mainstay of therapy otherwise. Anticoagulation should be given if the ejection fraction is $< 35\%$ to prevent intracardiac thromboembolism.

Sixty-six percent of patients with peripartum cardiomyopathy will have normal LV size and function at the end of the year, while the rest will have varied degrees of persistently depressed function. These patients with cardiomegaly have 80% mortality rate in future pregnancies and counseled regarding contraceptive methods. Bromocriptine is the new drug being researched for use in this condition [19].

Perimortem cesarean section

In normal circumstances, Cesarean is done for obstetric indications and is performed at gestational age consistent with fetal viability. Several clinical circumstances may necessitate perimortem Cesarean during unsuccessful maternal CPR because even correctly performed CPR provides less than 30% of normal cardiac output and fraction of blood going to the uterus approaches nil and the fetus is anoxic at all times following maternal cardiac arrest, even during ideal CPR [20]. Uterine evacuation may be indicated for either maternal or fetal reasons, or both. An algorithm for perimortem cesarean section is listed.



Conclusion

The management of critically ill young obstetric patient, who suffered an unanticipated illness requires many skills and is stressful to the obstetrician, family and the patient. An optimal successful outcome requires close collaboration between intensivists and obstetricians. Each ICU in the present day receiving obstetric patients must be ready for perimortem Cesarean section for maternofetal benefit.

Compliance with Ethical Standards

Conflict of interest We declare that we have no conflict of interest.

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