Hemodynamic assessment in a pregnant and peripartum patient

Shigeki Fujitani, MD; Marie R. Baldisseri, MD

Pathophysiology: Critical care in obstetrics has many similarities in pathophysiology to the care of nonpregnant women. However, changes in the physiology of pregnant woman necessary to maintain homeostasis for both mother and fetus, especially during critical illness, result in complex pathophysiology. Understanding the normal physiologic changes during pregnancy, intrapartum, and postpartum is the key to managing critically ill obstetric patients with underlying medical diseases and pregnancy-related complications.

Hemodynamic Monitoring: When the pathophysiology of critically ill obstetric patients cannot be explained by noninvasive hemodynamic monitoring and the patient fails to respond to conservative medical management, invasive hemodynamic monitoring may be helpful in guiding management. Most important, the proper interpretation of hemodynamic data is predicated on knowledge of normal values during pregnancy and immediately postpartum. Invasive hemodynamic monitoring with pulmonary artery catheterization has been used in the obstetric population, particularly in patients with severe preeclampsia associated with pulmonary edema and renal failure. (Crit Care Med 2005; 33[Suppl.]:S354–S361)

Key Words: cardiac output; oxygen consumption; tidal volume; functional residual capacity; supine hypotensive syndrome; autotransfusion; hemodynamic monitoring; cardiac comorbidity; mitral stenosis; pulmonary hypertension

Pregnancy complications contribute significantly to maternal and fetal morbidity and mortality. The overall prevalence of obstetric patients who may require critical care during their pregnancy ranges from 1 to 9 in 1,000 gestations (1, 2). Overall maternal mortality was 11.5 maternal deaths per 100,000 live births during 1991–1997 (3). The goal for maternal deaths per 100,000 live births in the Unites States is <3.3 by the National Center for Disease Prevention and Health Promotion. Most maternal deaths have been attributed to complications from hypertensive diseases, pulmonary illness, and cardiac diseases (4). The mortality rate of critically ill obstetric patients ranges from 12% to 20% (5). A review of 18 studies reporting on the main indication for intensive care unit (ICU) admission showed that hypertensive diseases (eclampsia, preeclampsia, HELLP syndrome—hemolysis, elevated liver enzymes, low platelet count, and hypertension crisis) were the most frequent cause (30.8%), followed by hemorrhage (shock, placental abruption, postpartum hemorrhage, 20.3%) and pulmonary complications (pulmonary edema, pneumonia, adult respiratory disease syndrome, asthma) (4).

Normal Cardiac Physiology in Pregnancy

Hemodynamic studies of normal findings during pregnancy have demonstrated increases in cardiac output, left ventricular stroke work, and oxygen consumption (Tables 1 and 2). Cardiac output is principally increased by stroke volume, particularly in the first two trimesters. In the third trimester, cardiac output increases only minimally primarily because of the increase in heart rate as term approaches. The failure of pulmonary artery occlusion pressure or central venous pressure to increase in pregnant patients, despite significant increases in intravascular volume, reflects the decreases in the systemic and pulmonary vascular resistances. The decreased vascular resistances allow both the systemic and pulmonary vasculature to accommodate higher volumes at normal vascular pressures (6).

Structural Remodeling of the Heart

The heart is dramatically remodeled during pregnancy. The remodeling of the heart causes enlargement of all four chambers, particularly the left atrium, which may predispose to supraventricular and atrial arrhythmias. As the uterus enlarges and the diaphragm elevates, the heart is rotated upward and to the left, which results in left axis deviation on the electrocardiogram and the appearance of cardiomegaly on the chest radiograph without underlying cardiac pathology. The valvular annular diameters increase, as does the thickness of the left ventricular wall. New murmurs often appear during pregnancy. Mild pulmonic and tricuspid regurgitation occurs in >90% of healthy pregnant women (7, 8). One third of pregnant women have evidence of clinically insignificant mitral regurgitation (8). Left ventricular end-diastolic and left atrial dimensions gradually return to normal over a 2-wk period, whereas left ventricular wall thickness returns to normal over a 24-wk period (8).

Pulmonary Adaptation to Pregnancy

During pregnancy, there is an increase in tidal volume of approximately 40%, a decrease in the functional residual capacity by 25%, and an increase in oxygen consumption as a result of increased metabolic needs of the mother and the fetus. The combination of the decreased functional residual capacity and the increased oxygen consumption diminishes the oxygen reserve of the mother and subsequently increases the hypoxic risk to both the mother and the fetus.
in response to maternal hypoventilation or apnea. Oxygen requirements increase by approximately 30–40 mL/min in pregnancy and are met by an increase in minute ventilation, primarily as a result of an increased tidal volume. The increase in minute ventilation results in a mild compensated respiratory alkalosis with a decline in the PaCO₂ to ~30 torr (4.0 kPa). The pH does not change (pH range 7.40–7.47) secondary to renal compensation that results in a decrease in the serum bicarbonate concentration (18–21 mEq/L) (5). Pregnant women who present with a PaCO₂ level of 40 torr should prompt the clinician to look for an imminent cause of respiratory failure.

**Endocrinologic Adaptation**

There are numerous endocrine and metabolic alterations during pregnancy that principally affect the hypothalamus, pituitary, and adrenal glands. Both corticotropin and cortisol levels are elevated in pregnancy. The pituitary gland in pregnancy enlarges, mainly due to proliferation of prolactin-producing cells in the anterior pituitary. The enlargement of the pituitary makes it more susceptible to alterations in blood supply and increases the risk of postpartum infarction when large amounts of blood loss occur during delivery. In patients with occult adrenal insufficiency, a life-threatening adrenal crisis may be precipitated by the stress of labor and delivery. Estrogen is a mediator that plays a role in some of the physiologic changes in pregnancy, and it predominantly affects uterine blood flow (9). Progesterone and prostaglandins E₂ and I₂ have vasodilatory effects on the uteroplacental vessels, which result in increased cardiac output in a pregnant woman (9).

**Influence of Body Position**

In the supine position, the enlarged uterus compresses the inferior vena cava, reducing venous return to the heart, which decreases stroke volume and cardiac output. However, before 24 wks the effect of the supine position on cardiac output is not observed. The effect of the supine position on cardiac output is most marked in late pregnancy. The decrease in cardiac output in the supine position compared with the lateral recumbent position can be as high as 25–30%. The “supine hypotensive syndrome” of pregnancy (severe hypoperfusion associated with hypotension and bradycardia) can further decrease cardiac output by 30–40%. The left lateral position quickly reverses compression of the inferior vena cava. An alternate position, although less optimal than the left lateral position but preferable to the supine position, is left lateral tilt to 15° or manual displacement of the gravid uterus. The latter maneuver of left uterine displacement can be performed by manually moving the uterus away from the midline to the left side when the patient is supine. A study to assess the maternal position during resuscitation showed that at an angle of 27° on an inclined board, 80% of maximum force was obtained with cardiac compressions (10).

Clark et al. (11) observed the central hemodynamic responses to position changes in ten normotensive pregnant patients in the third trimester and at 11–3 wks postpartum. It is of interest that the orthostatic response after pregnancy was much more labile than that during the third trimester (11). This implies a greater hemodynamic stability in response to orthostasis during pregnancy.

**Normal Hemodynamic Profiles During Pregnancy and Postpartum**

Although cardiac output remains relatively constant in the latter half of pregnancy, there is significant increase during active labor and immediately after delivery. Table 1 illustrates that during labor and delivery, cardiac output increases up to 50% and blood volume increases additional 300–500 mL with each
contraction in the second stage of labor. The hemodynamic changes seen during labor and delivery are influenced by anesthetic and analgesic techniques, especially if caudal anesthesia is used. Within the first 15–20 mins after delivery of the fetus and placenta, there is a substantial increase in cardiac output because blood is no longer diverted to the uteroplacental vascular bed. Approximately 500 mL is redirected to the maternal circulation in the so-called “auto-transfusion” effect of pregnancy. This effect can cause cardiac output to increase by 60–80% after aortic compression is removed and blood volume is increased. Most of the physiologic changes of pregnancy resolve and revert to normal within several days after delivery. Cardiac output remains at the high values seen during pregnancy for 2 days after delivery and then returns to normal gradually within 2 wks to 3 months after delivery as sodium and water balances normalize (12). Table 3 lists potential indications for hemodynamic monitoring in obstetric patients.

Noninvasive Hemodynamic Monitoring

Belfort et al. (13) assessed the correlation between noninvasive techniques with Doppler echocardiography and invasive hemodynamic measurements made with the pulmonary artery catheter (PAC) in 11 critically ill obstetric patients. The authors showed a high correlation between invasive and noninvasive techniques in the measurement of stroke volume and cardiac output. Ventricular filling pressures and pulmonary artery pressures also showed a similar significant correlation with invasive techniques. Belfort et al. (14) also reported on a series of 14 patients with an indication for invasive hemodynamic monitoring in whom Doppler ultrasound was used as a guide to clinical management. This pilot study concluded that the noninvasive monitoring with Doppler ultrasound had facilitated management and only two patients went on to have invasive monitoring to allow continuous monitoring (14). A study by Penny et al. (15) showed that the esophageal Doppler monitor consistently underestimated cardiac output by 40% in patients with severe preeclampsia <35 yrs old. The reason for underestimation was uncertain, but it may have been due to incorrect assumption of a fixed aortic diameter during systole and a fixed percentage of blood perfusion. Bioimpedance techniques are another noninvasive tool of central hemodynamic assessment. However, a study assessing cardiac index in normal term pregnancy with thoracic electrical bioimpedance and oxygen extraction techniques showed that the results of thoracic electrical bioimpedance were influenced by maternal position (16, 17).

Urine Sodium

One study with seven oliguric patients with preeclampsia evaluated volume status with fractional excretion of sodium and invasive hemodynamic monitoring (18). The findings suggested that if urine output or urinary diagnostic indexes were solely used to guide peripartum fluid management, the majority of these tests would indicate hypovolemia (71.4%) in patients with preeclampsia. The concordance of the reading of pulmonary artery occlusion pressure and fractional excretion of sodium was only 57.1%. Thus, fractional excretion of sodium may be misleading if used to guide fluid management in oliguric patients with preeclampsia.

Invasive Hemodynamic Monitoring

The utility of the PAC expanded from cardiac functional diagnosis to the direction of therapy when Swan et al. (19) introduced the balloon-tipped catheter in 1970. One year later, Ganz et al. (20) introduced a new technique for measurement of cardiac output by thermodilution. Since then, the PAC has been widely used in critically ill patients both for diagnosis and to guide therapy. However, the clinical value of the PAC in critically ill patients has been debated for decades. A multiple-center prospective cohort study demonstrated that patients with PAC had significantly increased 30-day, 2-month, and 6-month mortality rate and increased ICU and hospital length of stay by case matching multivariable regression analysis (21). A multiple-center, randomized, blinded, controlled trial assessed the efficacy of PAC for high-risk perioperative patients with American Society of Anesthesiologists class III or IV. A higher rate of pulmonary embolism was documented in patients with PAC; however, the survival rates at 6 and 12 months did not differ among patients in the standard care and PAC groups (22). Supportive data to evaluate the efficacy of the PAC in hemodynamically unstable patients, such as acute respiratory distress syndrome and septic shock, are still lacking.

The Pulmonary Artery Catheter in Obstetrics

In 1980s, the PAC was first used for invasive hemodynamic monitoring in critically ill pregnant patients. Studies have demonstrated the benefit of hemodynamic monitoring with placement of a PAC in patients with severe preeclampsia complicated with pulmonary edema and/or renal failure (23). A summary of 14 studies using the PAC in obstetric patients demonstrated that 64.3% of these studies used PAC for hemodynamic assessment in patients with the diagnosis of preeclampsia or eclampsia (24). Hemodynamic studies of obstetric patients suggest placement of the pulmonary artery catheter for severe valvular disease, cardiomyopathy, severe pulmonary and renal disease, and septic shock not responsive to standard therapies (25). Potential indications for hemodynamic monitoring in obstetric patients are listed in Table 4.

Complications of the Pulmonary Artery Catheter

The benefit and risks of the PAC in obstetrics have been extensively reviewed
Table 4. PAC measurements in renal failure, pulmonary edema, and eclampsia

<table>
<thead>
<tr>
<th>Physiologic Variable</th>
<th>Renal Failure (n = 53)</th>
<th>Pulmonary Edema (n = 30)</th>
<th>Eclampsia (n = 17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial PAC readings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVP, mm Hg, mean ± SE</td>
<td>4.3 ± 0.6</td>
<td>9.6 ± 1.2*p</td>
<td>6.5 ± 1.5</td>
</tr>
<tr>
<td>PAOP, mm Hg, mean ± SE</td>
<td>10.3 ± 0.7</td>
<td>21.0 ± 2.6*p</td>
<td>13.3 ± 2.4</td>
</tr>
<tr>
<td>SVR, dyne · sec · cm⁻², mean ± SE</td>
<td>1218 ± 76</td>
<td>1348 ± 103</td>
<td>1572 ± 279</td>
</tr>
<tr>
<td>CO, L/min, mean ± SE</td>
<td>7.6 ± 0.3</td>
<td>7.6 ± 0.4</td>
<td>6.6 ± 0.7</td>
</tr>
<tr>
<td>LSVI, g/(min · m²), mean ± SE</td>
<td>56.5 ± 3.5</td>
<td>50.9 ± 3.8</td>
<td>49.8 ± 7.9</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>49</td>
<td>66</td>
<td>55</td>
</tr>
<tr>
<td>Intervention before PAC placement, %</td>
<td>100</td>
<td>60</td>
<td>100</td>
</tr>
<tr>
<td>Diuretics</td>
<td>7.5</td>
<td>87</td>
<td>6</td>
</tr>
<tr>
<td>Dopamine</td>
<td>85</td>
<td>10.3</td>
<td>47</td>
</tr>
<tr>
<td>Intervention after PAC placement, %</td>
<td>90</td>
<td>40</td>
<td>88</td>
</tr>
<tr>
<td>Volume expansion</td>
<td>72</td>
<td>100</td>
<td>71</td>
</tr>
<tr>
<td>Diuretics</td>
<td>72</td>
<td>100</td>
<td>71</td>
</tr>
</tbody>
</table>

PAC, pulmonary artery catheter; CVP, central venous pressure; PAOP, pulmonary artery occlusion pressure; SVR, systemic vascular resistance; CO, cardiac output; LSVI, left ventricular stroke volume index; LVEF, left ventricular ejection fraction.

*p < .001, pulmonary edema group vs. renal failure; *p < .0001, pulmonary edema group vs. renal failure group, p < .03, pulmonary edema group vs. eclampsia group. Reproduced from Ref. 23; © 2005, with permission from Elsevier.

(1, 26–28). Complications associated with invasive hemodynamic monitoring include pneumothorax, ventricular arrhythmias, air embolism, pulmonary infarction, pulmonary artery rupture, sepsis, local vascular thrombosis, intracardiac knotting, and valve damage. Complications have decreased in part because of better physician and nursing vigilance. The prevalence of pneumothorax has decreased from 6% to 1% in the early literature to <0.1%, pulmonary infarction from 7.2% in 1974 to 0 to 1.3% in recent studies, and pulmonary artery rupture from 0.1 to 0.2% to almost <0.1%. Local vascular thrombosis has decreased with the use of heparin-bonded catheters. Septicemia has decreased from 2% to 0.5% (28).

Clinical Interpretation of Hemodynamic Data Obtained by Means of Invasive Monitoring in Pregnancy

Hemodynamic measurements obtained with the pulmonary artery catheter provide particular information about the circulation and the factors that govern the pumping action of the heart: preload, afterload, contractility, and the heart rate. Pulmonary artery occlusion pressure and the stroke volume or left ventricular stroke work (stroke volume times the pressure gradient over the left ventricle) generate a Frank-Starling curve that allows rapid and accurate bedside assessment of the patient’s hemodynamic condition and responses to treatment (29). Central venous pressure reflects the preload of the right ventricle that can fail as a result of left ventricular failure and increasing pressures in the left ventricle. In particular, in the presence of cardiopulmonary disease and in conditions in which pulmonary capillary damage with pathologic capillary wall permeability may be expected (e.g., in septic or severe hemorrhagic shock, eclampsia, or any other cause of adult respiratory distress syndrome), measured absolute values of central venous pressure and pulmonary artery occlusion pressure may be extremely disparate (29). Pregnant women without any evidence of left heart failure and with normal mitral valves have shown a marked discrepancy between central venous pressure and pulmonary artery occlusion pressure values. Wallenberg (30) concluded that with the exception of very low or high readings, central venous pressure values cannot be expected to give reliable information on hemodynamic conditions on the left side of the heart and, for that reason, should not be used in the intensive care management of critically ill pregnant patients.

Mixed venous oxygen saturation ($S\text{V}_\text{O}_2$) is a marker of the balance between oxygen delivery and oxygen consumption. The continuous measurement of oxygen saturation in mixed venous blood is performed with specialized pulmonary artery catheters equipped with fiberoptic bundles that can transmit light to and from the catheter tip. $S\text{V}_\text{O}_2$ measured by the PAC is within 1.5% of the $S\text{V}_\text{O}_2$ measured by co-oximeters (31). Continuous reading of $S\text{V}_\text{O}_2$ provides a general screening method for monitoring a group of four variables: cardiac output, hemoglobin, arterial oxygen saturation, and oxygen consumption. Changes in $S\text{V}_\text{O}_2$ are extremely sensitive but are a nonspecific marker of cardiovascular stress. Excessive oxygen consumption (shivering, spontaneous movements), decreased hemoglobin, and decreased arterial oxygen saturation all independently result in a decrease in $S\text{V}_\text{O}_2$. To the best of our knowledge, there is no study to report the use of $S\text{V}_\text{O}_2$ as a hemodynamic monitor in the obstetric population.

Preeclampsia

Preeclampsia is the most common illness diagnosed and treated in critical care obstetric medicine. Invasive hemodynamic monitoring with the PAC is most commonly used in patients with severe preeclampsia, so we will focus on the utility of the PAC in patients with severe preeclampsia. Preeclampsia is a pregnancy-related multiple-system disease that usually occurs after the 32nd week of gestation. The diagnosis of preeclampsia is confirmed by the development of hypertension with proteinuria, with or without generalized edema occurring after the 20th week of gestation although it may occur postpartum. Preeclampsia is generally categorized as mild or severe primarily on the basis of the degree of elevation in blood pressure, the degree of proteinuria, or both. The incidence of preeclampsia was about 5% in one prospective multiple-center study in the United States (32). A retrospective study investigating 32 obstetric patients requiring admission to ICU showed that preeclampsia was the most common reason (22%) for admission to the ICU (2).

It has been postulated that failure of cardiac adaptations, such as increased plasma volume and reduced systemic vascular resistance due to the failure of the second wave of trophoblastic invasion into the spiral arteries of the uterus, is the main pathophysiology of preeclampsia. Severe preeclampsia may rapidly deteriorate the cardiopulmonary status of the mother and fetus. Regardless of the duration of gestation, the recommended treatment is prompt delivery of the fetus. For gestations <34 wks, prolongation of pregnancy with close monitoring may be indicated to improve neonatal survival...
and reduce short-term and long-term neonatal morbidity.

Preeclampsia with acute pulmonary edema is a common reason for ICU admission. Preeclampsia was the admitting diagnosis in 18–28% of pregnant patients presenting with pulmonary edema (33,34). Pulmonary edema has been noted in approximately 2.9% of patients with severe preeclampsia (35). Preeclamptic patients frequently have multiple causes of pulmonary edema including increased capillary permeability due to endothelial cell injury, hypoalbuminemia, and afterload-induced left ventricular dysfunction. Benedetti et al. (36) noted that the development of pulmonary edema in patients with preeclampsia seemed to be multifactorial according to hemodynamic monitoring. In this study, ten patients with severe preeclampsia were evaluated hemodynamically with a pulmonary artery catheter. Two of ten patients had cardiogenic pulmonary edema, three patients had pulmonary capillary leak, and five had a reduced colloid oncolytic pressure/ pulmonary artery occlusion pressure gradient, which caused or contributed to the development of pulmonary edema.

The most frequently reported indications for pulmonary artery catheter placement in severe preeclampsia are for renal failure and pulmonary edema. The complication rate in these patients is <5%. The incidence of complications may be somewhat lower in pregnant patients due to their young age and the absence of underlying comorbid conditions (4). Pulmonary edema has been associated with severe preeclampsia in approximately 3% of cases (37). Patients with preeclampsia associated with pulmonary edema were found to have significant maternal (11%) and perinatal (9–23%) mortality rates (38,39). Gilbert et al. (23) showed that patients with severe preeclampsia complicated with pulmonary edema had elevated right and left filling pressures. Their hemodynamic measurements influenced by therapy are listed in Table 4. Diuretic use increased from 87% to 100% after PAC was placed in these patients.

Gilbert et al. (23) found that renal failure was the most common indication for pulmonary artery catheterization. Clark et al. (40) reported on nine patients who had persistent oliguria complicated with severe preeclampsia. In this study, the authors described three hemodynamic subsets: a) women with a low pulmonary artery occlusion pressure and high cardiac output and systemic vascular resistance; b) women with a normal or increased pulmonary artery occlusion pressure and cardiac output and normal systemic vascular resistance; and c) women with an increased pulmonary artery occlusion pressure and systemic vascular resistance and decreased cardiac output. These findings may underscore the need for invasive hemodynamic monitoring in patients with renal failure associated with severe preeclampsia if conservative management fails. However, most patients with severe preeclampsia in the absence of pulmonary edema and renal failure can be managed without invasive monitoring. No supporting data exist demonstrating an outcome benefit using this intervention, and, therefore, no evidence-based recommendations can be made concerning the use of the PAC in critically ill pregnant patients.

### Primary Pulmonary Hypertension

The incidence of primary pulmonary hypertension is one to two per million (41). Women are affected four to five times more often than men. Secondary pulmonary hypertension can develop as a complication of cardiac and pulmonary disease or as a complication of drugs such as cocaine or appetite suppressants. The maternal mortality rate associated with pregnancy and pulmonary hypertension ranges from 30% to 50% (42,43).

Pulmonary artery catheterization provides an early warning of rising pulmonary artery pressures as well as deterioration in right ventricular function. Its use has not been associated with improved survival (44). Because of the poor prognosis of both primary and secondary pulmonary hypertension in obstetrics, some clinicians have advocated the use of pulmonary artery catheterization during deliveries and in the immediate postpartum period (45). In a case study of a 26-wk pregnant patient with primary pulmonary hypertension, an epoprostenol infusion was titrated using the pulmonary artery pressure and cardiac output measurements obtained with a PAC, and the patient had a favorable outcome (45). The author concluded that early recognition and treatment with a pulmonary vasodilator in conjunction with hemodynamic monitoring and anticoagulation therapy for primary pulmonary hypertension may reduce the likelihood of complications and mortality associated with pulmonary hypertension and pregnancy.

### Mitral Stenosis

Mitral stenosis is the most common rheumatic valvular lesion encountered in pregnancy. Complications of mitral stenosis include pulmonary edema, right ventricular failure, and atrial arrhythmias with risk of embolization. Several pathophysiologic effects can exacerbate cardiac function in the setting of mitral stenosis. The expanded maternal blood volume can increase the risk of pulmonary congestion and edema, and the physiologic tachycardia of pregnancy decreases left ventricular filling time, which leads to elevated left atrial pressures that cause pulmonary edema and decreased forward flow causing hypotension, fatigue, and syncope. Because of the relatively fixed cardiac output associated with mitral stenosis, this patient is at risk for the development of acute pulmonary edema. During labor, adequate pain control with epidural anesthesia prevents...
pregnant patient, with optimally at least a management should be similar to the non-matched nonpregnant women (54). Management of myocardial infarction than age-partum women have a six-fold higher rate myocardial ischemia and infarction. Post-partum in the number of women with peripartum decades, there has been a slight increase in the systemic vascular resistance because of the increased blood volume and decreased systemic vascular resistance occurring in pregnancy may cause right-to-left shunting, decreased pulmonary perfusion, and marked hypoxemia. This is aggravated by decreased filling pressures due to hemorrhage or anesthesia and can result in marked hypotension, hypoxemia, and sudden death (57). For these patients, maintaining high filling pressure may be valuable during labor (56). On the contrary, left-to-right shunts are well-tolerated in pregnancy. Left-to-right shunts eventually can lead to pulmonary hypertension and reversal of the shunt with cyanosis. Hypertrophic obstructive cardiomyopathy may first manifest during pregnancy with a decrease in the systemic vascular resistance and tachycardia that reduce left ventricular filling, thus aggravating the outflow obstruction. Management with careful monitoring and the avoidance of hypotension, Val-salva maneuvers, and possibly β-blockade generally produce a good outcome (58). Uncontrolled tetralogy of Fallot may cause similar hemodynamic effect with right-to-left shunting. Marfan’s syndrome may occasionally be responsible for life-threatening aortic dissection in pregnancy, especially associated with aortic root diameters >4.5 cm (59). Pregnancy increases the risk of arterial rupture, and aortic dissection, intracerebral hemorrhage, and splenic artery rupture may occur (60). Hypertension and aortic root diameter should be tightly monitored (Table 6) (61).

### Aortic Stenosis

Severe aortic stenosis is far less common in the pregnant women. The greatest risk is imposed by the limited cardiac output, which may affect the coronary artery perfusion. These patients do not tolerate the significant fluid shifts that are seen during delivery and in the immediate postpartum period. The overall mortality rate is as high as 17%, and pulmonary artery catheterization may be useful to maintain an adequately high occlusion pressure without precipitating pulmonary edema (53).

### Aortic and Mitral Regurgitation

In contrast, aortic and mitral insufficiencies are generally well tolerated during pregnancy. The hemodynamic changes of pregnancy are beneficial to a patient who has aortic or mitral regurgitation because of the increased blood volume and decreased systemic vascular resistance that promotes forward flow across the regurgitant valve (51).

### Ischemic Heart Disease

Although coronary artery disease is uncommon in women of childbearing age, myocardial infarction may occur because of increased myocardial oxygen demands of pregnancy and the hypercoagulable state. With the increase in maternal age seen during the past few decades, there has been a slight increase in the number of women with peripartum myocardial ischemia and infarction. Postpartum women have a six-fold higher rate of myocardial infarction than age-matched nonpregnant women (54). Management should be similar to the non-pregnant patient, with optimally at least a 2-wk period of convalescence before delivery (55).

### Congenital Heart Disease

Congenital cardiac abnormalities such as uncomplicated atrial septal defect, ventricular septal defect, and patent ductus arteriosus are usually well tolerated in pregnancy. However, the development of pulmonary hypertension associated with these conditions markedly increases the risk, with maternal mortality reported at 30–50% and fetal loss of up to 75% (53, 56, 57). The decreased systemic vascular resistance occurring in pregnancy may cause right-to-left shunting, decreased pulmonary perfusion, and marked hypoxemia. This is aggravated by decreased filling pressures due to hemorrhage or anesthesia and can result in marked hypotension, hypoxemia, and sudden death (57). For these patients, maintaining high filling pressure may be valuable during labor (56). On the contrary, left-to-right shunts are well-tolerated in pregnancy. Left-to-right

<table>
<thead>
<tr>
<th>Condition</th>
<th>Maternal Mortality Rate, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>All mitral stenosis</td>
<td>0–1.6</td>
</tr>
<tr>
<td>NYHA class III or IV</td>
<td>5.0–7.0</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>0–2.0</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>30–50</td>
</tr>
<tr>
<td>Mechanical heart valve</td>
<td>1.0–4.0</td>
</tr>
<tr>
<td>Coarctation of the aorta</td>
<td>0–2.0</td>
</tr>
<tr>
<td>Marfan’s syndrome</td>
<td>0–1.1</td>
</tr>
<tr>
<td>All Marfan’s patients</td>
<td>50</td>
</tr>
<tr>
<td>Patients with risk factors</td>
<td>36</td>
</tr>
<tr>
<td>Eisenmenger’s syndrome</td>
<td>1.0</td>
</tr>
<tr>
<td>Cyanotic congenital heart disease</td>
<td>18–50</td>
</tr>
<tr>
<td>Peripartum cardiomyopathy</td>
<td>19</td>
</tr>
<tr>
<td>In current pregnancy</td>
<td>50</td>
</tr>
<tr>
<td>In previous pregnancy with persistent LV dysfunction</td>
<td></td>
</tr>
</tbody>
</table>

NYHA, New York Heart Association; LV, left ventricular. Reproduced from Ref. 51; Copyright © 2004, with permission from Elsevier.

<table>
<thead>
<tr>
<th>Table 6. Valvular heart lesions that are associated with high maternal risk or fetal risk during pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Severe aortic stenosis with or without symptoms</td>
</tr>
<tr>
<td>2. Aortic regurgitation with NYHA class III–IV symptoms</td>
</tr>
<tr>
<td>3. Mitral stenosis with NYHA class II–IV symptoms</td>
</tr>
<tr>
<td>4. Mitral regurgitation with NYHA class III–IV symptoms</td>
</tr>
<tr>
<td>5. Aortic or mitral valve diseases that result in severe pulmonary hypertension (pulmonary pressure &gt;75% of systemic pressure)</td>
</tr>
<tr>
<td>6. Aortic or mitral valve disease with severe left ventricular dysfunction (ejection fraction &lt;40%)</td>
</tr>
<tr>
<td>7. Mechanical prosthetic valves that require anticoagulation</td>
</tr>
<tr>
<td>8. Aortic regurgitation in Marfan’s syndrome</td>
</tr>
</tbody>
</table>

NYHA, New York Heart Association. Reproduced from Ref. 61; Copyright © 1998, with permission from The American College of Cardiology Foundation and American Heart Association, Inc.
REFERENCES

53. Lupton M, Ong-Tin N, Ayida G, et al:
Cardiac disease in pregnancy. *Curr Opin Obstet Gynecol* 2002; 14:137–143


