HYPERTENSIVE DISORDERS OF PREGNANCY

Hypertension during pregnancy has been classified by the American College of Obstetricians and Gynecologists into four distinct categories: (a) preeclampsia and eclampsia, (b) chronic hypertension (hypertension that was present before pregnancy), (c) chronic hypertension with superimposed preeclampsia or eclampsia, and (d) gestational hypertension (1). Most chronic hypertensive pregnant patients have essential hypertension, which has no appreciable effect during pregnancy unless end-organ damage is present. Chronic hypertension is seen in a critical care unit typically only when a patient has a hypertensive urgency/emergency unrelated to pregnancy, or if the patient has a secondary cause of hypertension that represents a short-term risk to maternal health. Similarly, latent or transient hypertension is also relatively benign, occurring in the last trimester or the immediate postpartum period, with a return of normotension by the first 3 weeks after delivery. It is preeclampsia or eclampsia—whether occurring de novo or superimposed on pre-existing hypertension—that is most likely to require critical care support, and therefore will be the focus of this section.

Preeclampsia and Eclampsia

Preeclampsia is a multisystem disorder unique to human pregnancies. Its pathophysiology is not well understood, and its cause is unknown. It is associated with an increased risk of fetal loss, intrauterine growth restriction, and preterm birth, and remains a leading cause of maternal death worldwide. Eclampsia refers to preeclampsia that is complicated by seizures, but it is our present understanding that the underlying condition is the same (2).

Although much of the care of the preeclamptic patient will fall into the domain of the obstetrician, familiarity with the manifestations and management of preeclampsia is important for any critical care physician. Primary obstetric disorders—of which, preeclampsia and its complications constitute the majority—account for 50% to 80% of ICU admissions during pregnancy and the puerperium in all parts of the world (3).

Risk Factors for Preeclampsia

Five percent to 8% of all pregnancies are complicated by preeclampsia, typically occurring in the final weeks prior to the due date and very rare prior to 20 weeks of gestation. The risk factors for preeclampsia are listed in Table 78.1. The diverse nature of the risk factors suggests that preeclampsia may be a common end point for a variety of processes related to placental dysfunction (4,5).

Etiology and Pathophysiology

Preeclampsia is believed to be an abnormal vascular response to the formation of the placenta. It is associated with endothelial cell dysfunction, activation of the coagulation system, enhanced platelet aggregation, and increased systemic vascular resistance. The maternal effects of these changes are manifest in the cardiovascular system, kidneys, lungs, and brain. Pathologic examination of affected maternal organs reveals areas of edema, endothelial swelling, microinfarctions, and microhemorrhages. The cardiovascular features of preeclampsia include decreased plasma volume—despite an increase in total-body water and salt retention—and colloid osmotic pressure, largely due to a drop in serum albumin (6). Generalized arteriolar vasospasm accounts for the hypertension in preeclampsia, which is often very labile.

Our understanding of the etiology of preeclampsia is evolving. The condition is felt to begin early in pregnancy, long before it becomes clinically apparent as the maternal syndrome. Three distinct, sequential phases occur in its evolution (4,7,8). The first phase is incomplete invasion of the trophoblast into the endometrium, perhaps due to a maladaptive immune response in the mother, followed by inadequate “placentation”—formation of the placenta—which leads to the second phase in which decreases in the levels of angiogenic growth factors and increased placental debris is found in the maternal circulation. This stage of the development of preeclampsia is not associated with any clinical symptoms or signs, however, decreases in placental growth factor (PlGF) and elevations of soluble FMS-like tyrosine kinase 1 (sFlt-1) and endoglin can be detected (9–11); these changes then incite a maternal inflammatory response. The third phase is the response of the maternal endothelium and cardiovascular system to these stressors, which is modulated by the woman’s own level of metabolic and cardiovascular health and then leads to the clinical presentation of the maternal preeclamptic syndrome. Although this response is manifested predominantly as hypertension and proteinuria, less common manifestations of cardiac, pulmonary, hematologic, neurologic, and hepatic complications can lead to admission in the intensive care unit.

Clinical Features

Clinically preeclampsia can have a highly variable presentation. It can manifest as a fetal syndrome (abnormal fetal oxygenation, reduced amniotic fluid, and fetal growth restriction), a maternal syndrome (proteinuria and hypertension with or without other multisystem abnormalities), or a combination of both. In most patients, both the fetal and maternal syndrome will be apparent, however one or the other will often predominate in an individual case. This chapter will focus on the maternal manifestations.
Preeclampsia is defined by the maternal manifestations of hypertension and proteinuria occurring in the second half of pregnancy. The presentation and diagnostic features of preeclampsia are reviewed in Tables 78.2 and 78.3 (12–17). Although hypertension above 140/90 mmHg and proteinuria over 300 mg/24 hr are required for the diagnosis of preeclampsia, some cases may present initially without these features, or may present—as in the case of postpartum eclampsia—after some of these features have already resolved.

Preeclampsia is defined to be severe by the presence of one or more of the following (1):
- Hypertension: systolic above 160 mmHg or diastolic above 110 mmHg on two occasions at least 4 hours apart while the patient is on bed rest
- Thrombocytopenia (platelet count <100,000 cells/µL)
- Impaired liver function (elevated blood levels of liver transaminases to twice the normal concentration)
- Severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by alternative diagnoses
- New development of renal insufficiency (elevated serum creatinine >1.1 mg/dL, or doubling of serum creatinine in the absence of other renal disease)
- Pulmonary edema
- New-onset cerebral or visual disturbances

Eclampsia results when seizures occur that are not related to other underlying disorders. These features describe a group of patients with an increased risk of fetal and maternal morbidity for whom delivery should be strongly considered. Preeclamptic patients who lack any of the features of severe preeclampsia may have to be observed without moving toward delivery if the fetus is significantly premature and the mother remains under close observation; however, such patients are rarely seen in intensive care settings.

Life-threatening maternal complications of preeclampsia such as severe hypertension, seizure, cerebral hemorrhage, pulmonary edema, disseminated intravascular coagulation (DIC), acute renal failure (ARF), and hepatic failure and/or

### TABLE 78.1 Risk Factors for Preeclampsia

<table>
<thead>
<tr>
<th>Feature</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetal</td>
<td>Multiple gestations</td>
</tr>
<tr>
<td></td>
<td>Molar pregnancies (can cause preeclampsia at &lt;20 wk gestation)</td>
</tr>
<tr>
<td></td>
<td>Fetal hydrops</td>
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<tr>
<td></td>
<td>Triploidy</td>
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<tr>
<td>Maternal</td>
<td>First pregnancy</td>
</tr>
<tr>
<td></td>
<td>New partners</td>
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<td></td>
<td>Age younger than 18 or older than 35 yr</td>
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<tr>
<td></td>
<td>Prior history of preeclampsia</td>
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<tr>
<td></td>
<td>Family history of preeclampsia</td>
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<tr>
<td></td>
<td>Gestational diabetes</td>
</tr>
<tr>
<td></td>
<td>Obesity</td>
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<tr>
<td></td>
<td>Thrombophilias</td>
</tr>
<tr>
<td></td>
<td>Antiphospholipid antibody syndrome</td>
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<tr>
<td></td>
<td>Systemic lupus erythematosus</td>
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<td></td>
<td>Renal disease</td>
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</tbody>
</table>

### TABLE 78.2 Clinical Features of Preeclampsia

<table>
<thead>
<tr>
<th>Feature</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>Headache</td>
</tr>
<tr>
<td></td>
<td>The headache that characterizes preeclampsia is typically frontal in location, throbbing in character, persistent, and not responsive to mild analgesia.</td>
</tr>
<tr>
<td></td>
<td>Visual phenomena</td>
</tr>
<tr>
<td></td>
<td>The visual disturbances that characterize preeclampsia are presumed to be due to cerebral vasospasm and are typically scintillations or scotomas. Longer-lasting visual field deficits and rarely transient blindness can result from edema, posterior reversible encephalopathic syndrome, and even infarction in the occipital region of the brain.</td>
</tr>
<tr>
<td></td>
<td>Epigastric pain</td>
</tr>
<tr>
<td></td>
<td>The epigastric or right upper quadrant discomfort that occurs in preeclampsia can be marked, and may be out of proportion to the degree of liver enzyme abnormalities. It is believed to be caused by edema in the liver that stretches the hepatic capsule. In rare cases, it may be caused by hepatic infarction or rupture.</td>
</tr>
<tr>
<td></td>
<td>Edema</td>
</tr>
<tr>
<td></td>
<td>Edema is present in more than 30% of normal pregnancies, and is thus not a reliable sign of preeclampsia. Rapid weight gain (more than 1 lb per week in the third trimester) or edema in the hands or facial area (nondependent edema) is best viewed as a sign that should lead the clinician to evaluate the patient for other, more specific, evidence of preeclampsia.</td>
</tr>
<tr>
<td>Signs</td>
<td>Hypertension &gt;140/90 mmHg</td>
</tr>
<tr>
<td></td>
<td>Hypertension in preeclampsia is due to vasospasm and can be very labile. Ideally, blood pressure should be measured in the sitting position with a manual cuff, with the brachial artery at the level of the heart. There is a literature suggesting that some automated blood pressure cuffs may be less reliable in preeclampsia and that either a manual cuff or arterial line should be used to verify blood pressure in preeclamptic patients with severe hypertension (13).</td>
</tr>
<tr>
<td></td>
<td>Epigastric or right upper quadrant tenderness</td>
</tr>
<tr>
<td></td>
<td>Abdominal pain in preeclampsia is attributed to hepatic capsular stretching from edema. The degree of tenderness is often out of proportion to the degree of elevation of liver function tests. Epigastric tenderness is suggestive of severe preeclampsia, and is associated with an increased risk of both maternal and fetal adverse outcomes.</td>
</tr>
<tr>
<td></td>
<td>Hyperreflexia</td>
</tr>
<tr>
<td></td>
<td>Clonus is an important sign of preeclampsia but should be distinguished from the very brisk reflexes commonly seen in normal pregnancies.</td>
</tr>
<tr>
<td></td>
<td>Retinal artery vasospasm on fundoscopy</td>
</tr>
<tr>
<td></td>
<td>Retinal vasospasm, retinal edema (in the form of soft exudates), hemorrhage, and exudative retinal detachment are uncommon findings in preeclampsia. Papilledema is rare.</td>
</tr>
</tbody>
</table>
TABLE 78.3 Laboratory Features of Preeclampsia

<table>
<thead>
<tr>
<th>Feature</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete blood count with elevated hemoglobin and/or thrombocytopenia</td>
<td>The “elevation of hemoglobin” seen with preeclampsia (which may manifest as a hemoglobin of 12 g/dL at 37 wk, when it would be expected to be closer to 10 g/dL because of the physiologic dilutional anemia that is seen in pregnancy) is due to hemoconcentration. Much less commonly, hemoglobin may fall with preeclampsia due to a microangiopathic hemolytic anemia. Platelet consumption in preeclampsia can cause an increased mean platelet volume and thrombocytopenia, and is an important manifestation of severe disease (14). In severe cases of preeclampsia or HELLP (a subset of preeclampsia), schistocytes (fragmented red cells) may be seen on peripheral smear and can lead to a mild drop in hemoglobin. Risk of hemolysis is rare, however, and should lead to the consideration of HUS or TTP.</td>
</tr>
<tr>
<td>Elevated serum creatinine</td>
<td>Typically serum creatinine is &lt;0.8 mg/dL (70 μmol/L) in pregnancy and values greater than this are considered abnormal. Renal function impairment is caused by decreased renal blood flow and glomerular filtration rate secondary to swelling of intracapillary glomerular cells, fibrin deposition along the basement membranes, and afferent arteriolar spasm.</td>
</tr>
<tr>
<td>Elevated serum uric acid</td>
<td>Typically serum uric acid is &lt;5.0 mg/dL (280 μmol/L) in pregnancy. Uric acid is the most sensitive test for identifying preeclampsia but it is still only elevated in approximately 80% of cases of preeclampsia. Uric acid rises in this setting due to impaired excretion of uric acid in the renal tubules that is caused by preeclampsia-related changes in the renal microcirculation (14). Although an important sign of preeclampsia, the elevated uric acid level is distinct from an elevated creatinine. AST, or decreased platelet count in that the uric acid level is not generally believed to have any direct clinical consequences and should not be used as a marker of disease severity.</td>
</tr>
<tr>
<td>Elevated liver enzymes</td>
<td>Mild elevations of AST, typically &lt;100 U/L, suggest hepatic involvement. Greater levels may be due to severe preeclampsia, HELLP syndrome, hepatic infarction, hepatic rupture, or superimposed acute fatty liver of pregnancy.</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>Proteinuria is an essential diagnostic feature of preeclampsia. Urine dipsticks are routinely used to screen for proteinuria in asymptomatic patients. However, dipsticks lack the needed sensitivity and specificity to make them a reliable test for proteinuria in patients in whom the diagnosis of preeclampsia is suspected because of the presence of other features of this disease. When preeclampsia is suspected, a 24-hour urine test for proteinuria with creatinine and creatinine clearance should be obtained. Proteinuria is present if there is more than 300 mg of protein excreted over 24 hours. Total urinary creatinine should be measured to assess the adequacy of urine collection. The creatinine clearance can be used in conjunction with the serum creatinine as a measure of renal function. The use of a random spot urinary protein-to-creatinine ratio to diagnose proteinuria in pregnancy has had many advocates, but it remains unclear at this time whether this test can replace the 24-hour urine in pregnant patients with suspected preeclampsia (15,16).</td>
</tr>
<tr>
<td>DIC screen</td>
<td>Severe preeclampsia can rarely cause DIC, but it is almost always seen in association with thrombocytopenia. Checking INR, PT, and fibrinogen degradation products is usually only done if the patient with preeclampsia has thrombocytopenia or is undergoing an invasive procedure.</td>
</tr>
</tbody>
</table>

HUS: hemolytic uremic syndrome; TTP: thrombotic thrombocytopenic purpura; AST: aspartate aminotransferase; DIC: disseminated intravascular coagulation; INR: international normalized ratio; PT: partial thromboplastin time.

rupture, occur in a minority of cases of preeclampsia. However, these conditions are most likely to require intensive care and are therefore reviewed here in more detail.

Severe Hypertension

A single blood pressure threshold that would absolutely necessitate treatment in the setting of preeclampsia is not established. Expert opinion favors urgent treatment of blood pressures greater than 180 mmHg (systolic) and 110 mmHg (diastolic) and, in the setting of obvious hypertensive end-organ damage (retinal hemorrhage, papilledema, pulmonary edema, severe headache, or renal failure), the blood pressure should be kept under 160/100 mmHg; beyond this consensus, opinions vary considerably (18,19).

Although no evidence suggests that treating blood pressures between 160/100 and 180/110 mmHg in the setting of preeclampsia improves maternal or fetal outcomes, many experts believe that the risks for seizure, placental abruption, stroke, and cerebral hemorrhage are decreased by bringing blood pressures down into the normal or mildly hypertensive range (20). Because preeclampsia is felt to be a dynamic vasospastic disorder with associated target-organ ischemia, some experts suggest letting blood pressures run in a moderately severe range to avoid worsening ischemia in areas of regional vasospasm. In the absence of direct evidence of end-target-organ damage from severe hypertension, it is our practice to treat all blood pressures over 160/105 mmHg. However, although we treat these blood pressures urgently, we are careful to avoid any severe, sudden decreases in maternal blood pressure that may adversely affect uteroplacental and cerebral perfusion.

If urgent blood pressure reduction is required, intravenous labetalol or intravenous hydralazine can be used. Increasing evidence indicates that labetalol may be the better choice of the two; it is our preferred agent, although both agents are still acceptable (20). Hydralazine has been associated with an increased risk of an emergency cesarean in women who receive it while still pregnant and with lower Apgar scores in the infants of mothers who have been given this agent prior to delivery. Short-acting oral nifedipine is also used at some
centers as an alternative to labetalol or hydralazine for the acute treatment of severe hypertension. Although its use in medical patients is now discouraged, its use for control of blood pressure in young pregnant or postpartum women without coronary artery disease remains an acceptable practice. Previous concerns about a drug interaction between magnesium and calcium channel blockers appear to be ill-founded (21). Diuretics should not be used in this setting unless pulmonary edema is present because, despite the edema that is so common in preeclamptic patients, most hemodynamic studies of preeclamptic women suggest that they are actually intravascularly volume depleted.

Once the patient has delivered, any antihypertensive agent can be used for blood pressure control. At that point, nitroprusside and nitroglycerin are excellent choices because of their very short half-lives.

Seizures

Seizures are the most well-known severe manifestation of preeclampsia. The risk of an eclamptic seizure in a patient with untreated preeclampsia is estimated to be about 1 in 200. Because of early identification of preeclampsia and the widespread use of magnesium prophylaxis, the incidence of eclampsia in the United States ranges from 1 in 1,000 to 1 in 20,000 deliveries; when it does occur, eclampsia is associated with a maternal mortality rate of 5% and a perinatal mortality rate between 13% and 30%.

Eclamptic seizures are typically of the grand mal variety, with clonic-tonic muscular activity followed by a postictal period. However, focal, jacksonian-type and absence seizures have been described. Most eclamptic seizures occur in the setting of established preeclampsia with hypertension and proteinuria. Classically, they are preceded by evidence of neuromuscular irritability such as tremulousness, agitation, nausea, vomiting, and/or clonus. However, some patients will present with seizure as their first manifestation of preeclampsia, usually occurring in the absence of hypertension or proteinuria.

The onset of eclamptic convulsions can be antepartum (38% to 53%), intrapartum (18% to 36%), or postpartum (11% to 44%). Postpartum eclamptic seizures generally occur in the first 48 hours after delivery, but it is not unusual to see them occur anytime in the first week after delivery; eclamptic seizures have been reported as late as 23 days postpartum.

The underlying pathophysiology of the eclamptic seizure is unclear. They cannot be attributed simply to severe hypertension, because eclampsia can be seen in patients with only mild elevations in blood pressure. Electroencephalograms may show epileptiform abnormalities, but usually show only a nonspecific diffuse slowing that may persist for weeks after delivery. Computed tomography (CT) and magnetic resonance imaging (MRI) of the eclamptic patient can be normal, or may show findings ranging from diffuse edema to focal areas of hemorrhage or infarction. Symmetrical white matter edema in the posterior cerebral hemispheres, particularly the parieto-occipital regions is characteristic for reversible posterior leukoencephalopathy syndrome (RPLS). Some suggest that RPLS could be considered an indicator of eclampsia, even when the other features of eclampsia (proteinuria, hypertension) are not present. MRI is more sensitive in detecting abnormalities in eclamptic patients, but both CT and MRI of the brain can be normal, particularly if done in the first 24 hours after the seizure. When radiologic changes are present, some—but not all—of these changes usually resolve with time (22).

Management of Eclamptic Seizures

Even when delivery is impending, a preeclamptic woman should still receive an anticonvulsant to prevent eclamptic seizures; magnesium sulfate is the medication of choice for this purpose (23). It halves the risk of eclampsia in patients with preeclampsia and lowers the risk of recurrent seizures and maternal death in women with eclampsia. It is superior to phenytoin and benzodiazepines in preventing further seizures. Magnesium is typically given as an intravenous bolus of 4 to 6 g, followed by a continuous intravenous infusion of 1 to 4 g/hr. Either monitoring plasma concentrations (which should run between 4 and 7 mmol/L), or observing the patient closely for symptoms and signs of toxicity (hypotension, hypotonia, muscular weakness, and respiratory depression) are reasonable options. Carefully monitoring for toxicity is important, particularly in patients with worsening renal function. Severe respiratory depression in a patient on magnesium should be treated with intravenous calcium. The only role of magnesium in preeclampsia is that of an anticonvulsant. Despite the possibility of a transient decrease in blood pressure with its initial administration, magnesium has no significant sustained effect on blood pressure. Its mechanism of action remains unclear, but it does not seem to have any intrinsic anticonvulsant effect, and may actually prevent seizures through its action as a cerebral vasodilator.

If the woman does have an acute eclamptic seizure, intravenous benzodiazepine is indicated to acutely stop the seizure, and magnesium should then be initiated if this has not already occurred. If an eclamptic convolution occurs while a patient is receiving magnesium, most clinicians will add phenytoin to the regimen. Continued seizures should warrant the involvement of neurology and consideration of the use of other antiepileptic drugs. Anticonvulsant therapy can generally be stopped once postpartum diuresis has begun and the manifestations of preeclampsia have started to improve.

Neuroimaging with CT or MRI is recommended for most patients with eclamptic seizures to rule out an intracerebral hemorrhage. The timing of these neuroimaging tests should be determined by the level of clinical suspicion for this diagnosis, and should not substantially delay delivery.

Cerebrovascular Accidents

Cerebrovascular accidents are three to seven times more common in pregnancy. Preeclampsia accounts for over a third of the strokes that do occur during pregnancy, and at least half of the deaths from preeclampsia in the developed world are due to stroke. Most of the strokes in patients with preeclampsia will be related to intracerebral hemorrhage, but can also occur due to vaso spas tic ischemia (24). Preeclampsia-related stroke is often, but not always, associated with severe hypertension and/or eclamptic convulsions. Sudden onset or worsening of a headache, a change in mental status, or any focal neurologic complaint occurring in the context of preeclampsia should lead to consideration of this diagnosis and urgent neuroimaging.

Pulmonary Edema

Pulmonary edema occurs in about 3% of cases of preeclampsia, and can cause significant maternal morbidity (25–27). It occurs as a result of the interplay of preeclampsia-related pulmonary endothelial damage and the low plasma oncotic pressure.
pressure seen in all pregnancies; excessive intravenous fluid is also, typically, a contributing factor. It is often seen in the postpartum period after a patient has received a substantial amount of intravenous fluid in labor (or with cesarean delivery) and when mobilization of fluid from the involuting uterus begins. Pulmonary edema in this setting is often amenable to gentle diuresis but may be severe enough to warrant mechanical ventilation.

Echocardiographic studies demonstrate that transient systolic or diastolic ventricular dysfunction is present in up to one-third of preeclampsia cases associated with pulmonary edema. This preeclampsia-related myocardial dysfunction is believed to be a manifestation of vasospastic coronary ischemia, and usually resolves rapidly with resolution of the preeclampsia. We consider this to be a distinct entity from peripartum cardiomyopathy (PPCM) and do not believe there is a substantial recurrence risk of cardiac disease for these patients in a subsequent pregnancy.

**Prevention and Treatment of Pulmonary Edema**

It is important to avoid excessive fluid administration to patients with preeclampsia because of their propensity for pulmonary edema. Ideally, one individual should be designated to approve and monitor all fluid administration in these patients. Regular auscultation of the lungs and use of transcutaneous pulse oximetry in patients with severe preeclampsia will help identify cases of pulmonary edema as they evolve. This careful observation should be continued in the postpartum period because pulmonary edema often occurs as late as 2 to 3 days after delivery. Acute treatment of pulmonary edema should involve supplemental oxygen, low-dose furosemide, and, if needed, morphine (27, 28). Blood pressure control may help treat pulmonary edema by decreasing afterload. An echocardiogram should be obtained to look for an underlying cardiac contribution. Intubation and mechanical ventilation may become necessary if the above measures do not improve the patient's oxygenation.

**Disseminated Intravascular Coagulation**

Disseminated intravascular coagulation (DIC) can occur as a late and severe complication of preeclampsia or eclampsia (29). Because most patients with preeclampsia-related DIC have low platelet counts or elevated transaminase levels, DIC screening in the absence of these abnormalities is generally not necessary (30). However, a DIC screen should be ordered in all preeclamptic patients with rising liver enzymes, dropping platelet counts, and/or any abnormal bleeding. This is particularly important if there is a possibility of an operative delivery.

**Acute Renal Failure**

Preeclampsia is often associated with a mild degree of renal impairment manifesting as a slightly elevated creatinine or a decreased urine output. This is due to a combination of intravascular volume depletion, renovascular vasospasm, and a preeclampsia-related glomerular lesion known as glomerular endotheliolysis. This mild renal impairment usually resolves rapidly after delivery.

Acute renal failure in preeclampsia is not common. If it does occur, acute tubular necrosis (ATN) and partial or total cortical necrosis are the most likely underlying lesions, and are thought to be caused by preeclampsia-related, vasospasm-induced renal ischemia. A history of transient hypotension is also typically present in these cases. The differential diagnosis includes ATN from sepsis or hemorrhage, or renal failure from causes unassociated with pregnancy such as hemolytic uremic syndrome, medication effects, or acute glomerulonephritis.

Most renal failure in the setting of preeclampsia is rapidly reversible, but if significant hypotension has occurred (as may happen with placental abruption or DIC-related hemorrhage), ATN or renal cortical necrosis may result and necessitate dialysis. In persons with sustained oliguria in the setting of preeclampsia, fluid challenges should be given cautiously because of the risk of pulmonary edema. Poor outcomes in preeclampsia are far more commonly related to pulmonary edema than they are to decreased urine output. Diuretics to improve urine output should be avoided in the absence of pulmonary edema because of the intravascular volume depletion present in most patients with preeclampsia.

If the patient is unresponsive to small fluid boluses, the use of central venous pressure (CVP) monitoring may be a helpful, if not completely reliable, guide. The role of the pulmonary artery catheter in this context is unproven, and should only be used by nurses and physicians who are trained and experienced in its use. Increasing data from randomized control trials have shown that pulmonary artery catheters are of less benefit than previously believed in nonpregnant patients, and there is little reason to believe this tool has a uniquely beneficial role in the pregnant population.

Sustained oliguria in preeclampsia is unusual, and therefore significant and rapid peripartum renal deterioration should also lead to consideration of differential diagnoses that include the hemolytic uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP), and renal cortical necrosis. It is therefore advisable to perform careful microscopic examination of urinary sediment and a peripheral smear in all pregnant or postpartum patients with oliguria (31).

**HELLP Syndrome**

A distinct clustering of the manifestations of preeclampsia is the HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet counts). This constellation of findings represents a particularly severe form of preeclampsia with significant risk for maternal illness and fetal injury or death (32, 33). HELLP occurs in up to 20% of cases of severe preeclampsia. The hemolysis is microangiopathic, and therefore schistocytes (fragmented erythrocytes) are seen on peripheral smears of the blood. Lactate dehydrogenase levels are usually increased and liver enzyme elevation may be two- to threefold. The thrombocytopenia can be precipitous and severe. High-dose dexamethasone is often given to treat patients with HELLP, but it is not clear that this intervention has clinically significant effects on outcomes, and the treatment remains supportive care coupled with delivery (34, 35).

**Hepatic Rupture, Infarction, or Hemorrhage**

Epiptigeric or right upper quadrant pain and elevation of hepatic enzymes due to preeclampsia are common. When these factors are present, it suggests severe disease and preeclampsia-related hepatic edema and ischemia. It generally is associated with no more than a two- to fourfold increase in aspartate aminotransferase (AST) or alanine aminotransferase (ALT). When pain is severe and/or hepatic enzymes rise above this level, preeclampsia-related hepatic infarction, hemorrhage, and rupture should be considered and investigated with
CHAPTER 78  Cardiac Disease and Hypertensive Disorders in Pregnancy

Diabetes Insipidus

Diabetes insipidus is a rare complication of preeclampsia with significant hepatic involvement. It can also be seen with AFLP. It has been hypothesized that the acute liver dysfunction in these patients reduces the degradation of vasopressinase (an enzyme which itself degrades vasopressin), and results in a state of relative vasopressin deficiency (37). The course of the condition follows that of the underlying disorder and can be treated with additional vasopressin until it resolves.

The Role of Arterial Lines, Central Venous Pressure Monitors, and Pulmonary Artery Catheters in Preeclamptic Patients

Most severe preeclamptic patients have normal or hyperdynamic left ventricular function with normal pulmonary artery pressure. Thus, a CVP monitor usually is adequate to assess volume status and left ventricular function. However, severely preeclamptic patients may develop cardiac failure, progressive and marked oliguria, or pulmonary edema. In such cases, some authors suggest that a pulmonary artery (PA) catheter may be helpful for proper diagnosis and treatment, because right and left ventricular pressures may not correlate (38,39). Given that evidence has evolved that the routine use of pulmonary artery catheters may not be as beneficial in the care of nonobstetric patients as once believed, the rather limited literature about their use in obstetric populations cannot help but be questioned (40–42). No clear consensus exists as to their role in the management of preeclampsia (43). We rarely employ them in any obstetric patients, as the risks—especially on labor and delivery units where the personnel have less experience in their placement and interpretation—seem to outweigh the evidence justifying their use. When questions arise as to whether cardiac dysfunction is contributing to a preeclamptic patient’s pulmonary edema and/or renal failure, we obtain an urgent bedside echocardiogram to guide our care and, in the absence of a significant cardiac cause, manage these patients clinically.

An intra-arterial catheter monitor may be indicated for protracted severe hypertension during therapy with potent antihypertensive agents or when there is a significant disparity between automated and manual cuff measurements of blood pressure.

Cardiac Disease during pregnancy has an incidence rate of 0.4% to 4%, and is associated with a maternal mortality of 0.4% to 6%, depending on the cardiac lesion being discussed (44). Indeed, it is now the leading cause of maternal mortality in North America (45). Although rheumatic heart disease is far less of a concern in the West than it was several decades ago, it remains a problem, along with peripartum cardiomyopathy, pulmonary hypertension, adult congenital heart disease, and myocardial ischemia. These conditions will be the focus of this section. Although many of the patients with cardiac disease who end up under the care of a critical care physician will have cardiac disease that was identified prior to pregnancy, a significant portion of patients will also have their cardiac disease present for the first time during pregnancy. The physiologic changes of pregnancy may exacerbate, and thereby unmask, previously undiagnosed cardiac disease, and pregnancy can predispose patients to the onset of certain cardiac diseases such as PPCM or ischemic heart disease. Some of the physiologic changes associated with pregnancy are reviewed below and are summarized in Table 78.4.

Physiologic Changes

Maternal blood volume gradually increases during pregnancy to 150% of nonpregnant levels (46). The increase in plasma volume (45% to 55%) is greater than the increase in red blood cell volume (20% to 30%), resulting in a relative anemia of pregnancy. This increase in blood volume is associated with an increase in cardiac output (CO), which begins early in gestation and peaks at levels 30% to 40% over nonpregnant values between 20 and 30 weeks (46); the increase then plateaus until term. CO in a twin pregnancy is 15% higher than that of a singleton pregnancy (47).

The increase in CO with gestation is dependent on heart rate and stroke volume. Heart rate gradually increases throughout pregnancy, starting as early as 4 weeks of gestation, with a 10% to 15% increase by term. Stroke volume, in contrast, peaks during the second trimester, with a 20% to 40% increase over the nonpregnant state.

During labor, CO rises another 15% to 45% above prelabor values with an additional increase of 10% to 25% during uterine contractions. The increase in CO in labor during contractions versus that seen between contractions is greater late in the first stage (34%) versus early in the first stage (16%) (48).

Oxygen consumption increases 20% during pregnancy, and may increase as much as 40% to 100% during active labor. In

Cardiac output

<table>
<thead>
<tr>
<th>Pregnancy</th>
<th>Labor and Delivery</th>
<th>Postpartum</th>
</tr>
</thead>
<tbody>
<tr>
<td>+30–50</td>
<td>+50–65*</td>
<td>+60–80</td>
</tr>
<tr>
<td>+10–15</td>
<td>+10–30*</td>
<td>−15–10</td>
</tr>
<tr>
<td>+20–30</td>
<td>+40–70</td>
<td>+60–80</td>
</tr>
<tr>
<td>+20–80</td>
<td></td>
<td>+0–10</td>
</tr>
<tr>
<td>+44–55</td>
<td></td>
<td>+0–30</td>
</tr>
<tr>
<td>+20–30</td>
<td></td>
<td>−10</td>
</tr>
<tr>
<td>+20</td>
<td>+40–100*</td>
<td>−10–15</td>
</tr>
<tr>
<td>−10–25</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Percentage change from nonpregnant state.

**TABLE 78.4 Hemodynamic Changes in Pregnancy**
the immediate postpartum period, CO increases 30% to 40% over the labor period or 60% to 80% over the nonpregnant state, with the increased blood volume shifting to the central circulation from the contracted uterus, as well as alleviation of aortocaval compression and a slight decrease in total peripheral resistance.

CO and other hemodynamic parameters are thought to return to their baseline prepregnant state by 6 weeks after delivery. However, CO may remain elevated for up to 12 weeks postpartum.

Systemic arterial pressure decreases by 10 to 15 mmHg over the first two trimesters and then gradually returns to baseline by term. Systemic vascular resistance decreases 10% to 20% during pregnancy. Moreover, systemic vascular resistance may remain decreased for at least 12 weeks postpartum.

Venous pressure in the lower extremities increases and peaks near term as the gravid uterus compresses the inferior vena cava—especially when the patient is supine—while CVP remains unchanged. Total-body water increases by about 2 kg throughout pregnancy.

Invasive PA catheterization in low-risk, near-term pregnant patients (36 to 38 weeks) reveals a significant decrease in pulmonary vascular resistance, colloid oncotic pressure (COP), and COP–pulmonary artery occlusion pressure (PAOP) gradient, with no change in PAOP or left ventricular stroke work index (50).

With a significant increase in oxygen consumption, especially during labor, along with a decrease in functional residual capacity, the importance of adequate preoxygenation (denitrogenation) before rapid sequence induction of anesthesia cannot be overemphasized. Morbidity and mortality statistics from England and Wales reveal that anesthetic-related maternal mortality is predominantly caused by the inability to intubate the trachea or by pulmonary aspiration during general anesthesia (51). Thus, an awake orotracheal intubation should be considered when airway patency is suspect. The most experienced person available should typically be the individual who intubates pregnant women on a regular basis.

Despite an average 200- to 500-mL blood loss for routine, uncomplicated vaginal deliveries and an 800- to 1,000-mL blood loss for cesarean section deliveries, blood transfusions are seldom necessary because of the increased blood volume and the autotransfusion of approximately 500 mL of blood from the contracted uterus in the postpartum period. Although this increase in blood volume protects against blood loss at delivery, pulmonary congestion and cardiac failure can result in patients with underlying cardiac dysfunction.

Pregnant women have a predisposition to pulmonary edema. Physiologic changes in pregnancy that favor the development of pulmonary edema include an increase in intravascular volume, decreased blood viscosity (“physiologic anemia of pregnancy”), decreased COP, and fluid shifts, especially in the immediate postpartum period.

Patients with minimal cardiac reserve may tolerate early pregnancy, and subsequently decompensate from increasing blood volume and the need for an increased CO in the late second trimester and early third trimester. Patients with moderate cardiac reserve may tolerate pregnancy well until labor and delivery or the puerperium. Thus, cardiac patients should continue to be closely monitored in the postpartum period because cardiac decompensation most frequently occurs during this time; the prepregnant baseline state may not be reached for as long as 12 weeks after delivery.

The enlarging uterus in the third trimester predisposes to aortocaval compression and decreased CO in supine patients. Inferior vena cava compression occurs in up to 90% of near-term parturients in the supine position. However, only about 10% to 15% of patients manifest the supine hypotensive syndrome because of shunting of venous blood away from the caval system to the azygos system by the intervertebral plexus of veins. Patients most susceptible to supine hypotension are those with polyhydramnios and multiple gestation. However, in most patients in the lateral position, CO is maintained. Turning from the supine to the lateral decubitus position increases CO from 8% at 20 to 24 weeks to as much as 30% near term (52). Therefore, to avoid aortocaval compression, measures such as uterine displacement by maternal position (lateral decubitus), bed position (left lateral tilt), or uterine displacement devices are imperative, especially in the last trimester. Moreover, maternal hypotension and placental hypoperfusion from aortocaval compression can be compounded by regional anesthesia that interferes with compensatory sympathetic nervous system mechanisms (53).

As a consequence of these cardiovascular changes, normal symptoms of pregnancy can include fatigue, dyspnea, decreased exercise capacity, and lightheadedness. Cardiac signs that may be seen in normal pregnancies include distended neck veins, peripheral edema, loud first heart sound, loud third heart sound, systolic ejection murmurs, and continuous murmurs (cervical venous hums and mammary souffle). Fourth heart sounds and diastolic murmurs occur rarely in normal pregnancy and should be considered pathologic unless proven otherwise. These changes are reviewed in Table 78.5. Therefore, the normal signs and symptoms of pregnancy may simulate pathologic disease states, thereby rendering the diagnosis of heart disease difficult.

### Table 78.5 Normal Cardiac Symptoms and Signs in Pregnancy

<table>
<thead>
<tr>
<th>SYMPTOMS</th>
<th>SIGNS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue</td>
<td>General</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>Distended neck veins</td>
</tr>
<tr>
<td>Decreased exercise tolerance</td>
<td>Peripheral edema</td>
</tr>
<tr>
<td>Lightheadedness</td>
<td>Hyperventilation</td>
</tr>
<tr>
<td>Syncope</td>
<td>Heart</td>
</tr>
<tr>
<td></td>
<td>Loud S1; increased split S1</td>
</tr>
<tr>
<td></td>
<td>Loud S2</td>
</tr>
<tr>
<td></td>
<td>Systolic ejection murmur</td>
</tr>
<tr>
<td></td>
<td>Continuous murmurs (venous hums, mammary souffle)</td>
</tr>
<tr>
<td></td>
<td>Chest radiograph</td>
</tr>
<tr>
<td></td>
<td>Increased pulmonary vasculature</td>
</tr>
<tr>
<td></td>
<td>Horizontal position of heart</td>
</tr>
<tr>
<td></td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td></td>
<td>Left axis deviation</td>
</tr>
<tr>
<td></td>
<td>Nonspecific ST–T–wave changes</td>
</tr>
<tr>
<td></td>
<td>Mild sinus tachycardia</td>
</tr>
</tbody>
</table>

**SIGNS**

- General
- Distended neck veins
- Peripheral edema
- Hyperventilation
- Heart
- Loud S1; increased split S1
- Loud S2
- Systolic ejection murmur
- Continuous murmurs (venous hums, mammary souffle)
- Chest radiograph
- Increased pulmonary vasculature
- Horizontal position of heart
- Electrocardiogram
- Left axis deviation
- Nonspecific ST–T–wave changes
- Mild sinus tachycardia
Normal chest radiographic findings demonstrate increased lung markings (prominent pulmonary vasculature partly due to both increased blood volume and increased breast shadow). Electrocardiographic (ECG) changes may include a left QRS axis deviation and nonspecific ST-segment and T-wave changes.

**Who Is Most at Risk and When Is That Risk Greatest?**

Table 78.6 classifies the risk of various cardiac lesions in pregnancy. When we speak about “risk” for these patients, we refer to congestive heart failure, arrhythmias, stroke, and death. Overall, about 13% of cardiac patients will suffer one of these outcomes in pregnancy. The presence of pulmonary hypertension is always associated with an increased risk, and this risk is commensurate to its degree of severity. Other factors associated with an increased risk of cardiac complications in pregnancy include the following (54):

- **New York Heart Association (NYHA) functional class.** This is perhaps the most important predictor of pregnancy outcome. Patients with NYHA class I and II cardiac disease generally have a good prognosis during pregnancy. Patients with NYHA class III and IV are more likely to experience complications and may require special management around the time of delivery.
- **Left-sided obstructive cardiac lesions.** Patients with lesions such as aortic stenosis may have difficulty accommodating the increased blood volume and CO seen in pregnancy, and become increasingly symptomatic. Interestingly, patients with regurgitant valvular lesions may have less difficulty in pregnancy, as CO in these cases may benefit from the decrease in systemic vascular resistance seen in pregnancy.
- **Cyanosis**
- **Left ventricular systolic dysfunction**
- **Prior cardiac events or previous dysrhythmia**

Although pregnant women with cardiac disease may experience complications at any point during pregnancy, there are three periods of particular risk:

1. **At the end of the second trimester, when CO has increased to its peak**
2. **At the time of labor and delivery, when cardiac work may be increased dramatically by both pain and the autotransfusion of blood from the placenta and uterus with each contraction**
3. **In the first 72 hours following delivery, when the uterine involution and resolution of pregnancy-related edema leads to mobilization of large amounts of fluid**

### General Management of Cardiac Patients During Pregnancy

Management of patients with cardiac disease in pregnancy should, in general, include good preconception counseling to assess and inform the patient of the risks associated with a pregnancy. Although no woman should be told that she “should never get pregnant,” a clear discussion of the risk is essential. With cases such as severe pulmonary hypertension or Eisenmenger syndrome, the patient should be strongly cautioned against pursuing a pregnancy. Women with congenital heart disease need also be informed that they are at increased risk of giving birth to a child with congenital heart disease. If a woman with cardiac disease decides to pursue a pregnancy after a clear discussion of risk, the cardiologist should ensure that her cardiac status is clearly delineated and optimized. Ideally, any necessary investigations or interventions should be carried out prior to conception. Once a woman is pregnant, regular visits with a medical specialist and an obstetrician trained in the care of high-risk pregnancies to watch for evidence of heart failure and arrhythmias are essential. Consultation with an obstetric anesthesiologist prior to delivery is also prudent.

As stated earlier, most cardiac medications can be used in pregnancy when indicated. Table 78.7 lists many common cardiac medications, and classifies them as to which drugs we know the most about regarding their safe use during pregnancy and which drugs we know the least. However, it should be emphasized that among the more commonly used cardiac

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**TABLE 78.6 Peripartum Risk of Various Cardiac Lesions**

<table>
<thead>
<tr>
<th>Risk Category</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower-risk lesion</td>
<td>Mitral valve prolapse</td>
</tr>
<tr>
<td></td>
<td>Atrial septal defect</td>
</tr>
<tr>
<td></td>
<td>Ventricular septal defect with normal</td>
</tr>
<tr>
<td></td>
<td>pulmonary pressures</td>
</tr>
<tr>
<td></td>
<td>Trace to mild valvular regurgitation</td>
</tr>
<tr>
<td></td>
<td>NYHA class I</td>
</tr>
<tr>
<td></td>
<td>History of SVT with recent good control</td>
</tr>
<tr>
<td></td>
<td>Presence of an implanted pace maker</td>
</tr>
<tr>
<td>Intermediate-risk</td>
<td>Stable ischemic heart disease</td>
</tr>
<tr>
<td>lesion</td>
<td>Mild to moderate pulmonary hypertension</td>
</tr>
<tr>
<td></td>
<td>Moderate to severe valvular insufficiency</td>
</tr>
<tr>
<td>High-risk lesion</td>
<td>NYHA class II</td>
</tr>
<tr>
<td></td>
<td>Cardiomyopathy with ejection fraction</td>
</tr>
<tr>
<td></td>
<td>30–50%</td>
</tr>
<tr>
<td></td>
<td>Poorly controlled SVT</td>
</tr>
<tr>
<td></td>
<td>Unstable ischemic heart disease</td>
</tr>
<tr>
<td></td>
<td>Moderate to severe left ventricular</td>
</tr>
<tr>
<td></td>
<td>obstruction (e.g., aortic: &lt;1.5 cm²) or</td>
</tr>
<tr>
<td></td>
<td>mitral-valvular stenosis: &lt;2 cm², peak</td>
</tr>
<tr>
<td></td>
<td>gradient LV outflow tract: &gt;30 mmHg)</td>
</tr>
<tr>
<td></td>
<td>NYHA class III</td>
</tr>
<tr>
<td></td>
<td>Cardiomyopathy with ejection fraction &lt;30%</td>
</tr>
<tr>
<td></td>
<td>Dilated aortic root, Marfan syndrome,</td>
</tr>
<tr>
<td></td>
<td>Ehlers-Danlos syndrome</td>
</tr>
<tr>
<td></td>
<td>Moderate pulmonary hypertension</td>
</tr>
<tr>
<td></td>
<td>History of ventricular tachycardia with or</td>
</tr>
<tr>
<td></td>
<td>without AICD</td>
</tr>
<tr>
<td></td>
<td>Mechanical prosthetic heart valve</td>
</tr>
<tr>
<td></td>
<td>History of TA or CVA</td>
</tr>
<tr>
<td>Highest-risk lesion</td>
<td>Pulmonary hypertension &gt;80 mmHg</td>
</tr>
<tr>
<td></td>
<td>Eisenmenger syndrome</td>
</tr>
<tr>
<td></td>
<td>NYHA class IV</td>
</tr>
<tr>
<td></td>
<td>Cyanosis</td>
</tr>
</tbody>
</table>

NYHA, New York Heart Association; SVT, supraventricular tachycardia; AICD, automated implantable cardioverter-defibrillator; TA, transient ischemic attack; CVA, cerebrovascular accident.

Items above can be used to calculate a risk index with 1 point being assigned for each and 0, 1, and >1 points being associated with a risk of some cardiac event during the entire pregnancy of 5%, 27%, and 75%, respectively.

medications, only angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and warfarin are known or strongly suspected to be human teratogens. Amiodarone has had mixed data with respect to its safety in pregnancy, with some reports of congenital hypothyroidism, goiter, prematurity, hypotonia, and bradycardia (55,56). Although use in an acute setting is appropriate, it is not a first-line agent for maintenance therapy in pregnancy. Angiotensin-converting enzymes and angiotensin receptor blockers both have been associated with fetal anomalies, fetal loss, oligohydramnios, cranial ossification abnormalities, and neonatal renal failure. Although their use in the first trimester was once supported, recent evidence suggests they should not be used at any time in gestation (57). Warfarin is associated with a high risk of miscarriage and anomalies of the eyes, hands, neck, and central nervous system (58). Again, the guiding principle of managing critical illness in pregnancy should be that, because fetal wellbeing depends on maternal well-being, medications that are of benefit to maternal health should also be considered to be in the fetus’ best interest. Useful sources for reviewing the available safety data for medications during pregnancy and with breastfeeding are found in references (59–65).

### Table 78.7 Commonly Used Cardiac Medications and Their Safety in Pregnancy

<table>
<thead>
<tr>
<th>Indication in Pregnancy</th>
<th>Use Generally Justifiable for This Indication in Pregnancy</th>
<th>Use Justifiable in Special Circumstances for This Indication in Pregnancy</th>
<th>Use Almost Never Justifiable for This Indication in Pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dysrhythmia</strong></td>
<td>Digoxin</td>
<td>Amiodarone</td>
<td>Disopyramide, mexiletine, and flecainide (less is known about these agents in pregnancy but there is no evidence at this point of human teratogenesis; they should generally be considered second-line agents in pregnancy)</td>
</tr>
<tr>
<td></td>
<td>β-Blockers (all probably safe but most avoid propranolol and atenolol, which may cause intrauterine growth restriction)</td>
<td>Calcium channel blockers, especially verapamil and diltiazem (less is known about amlodipine)</td>
<td>Calcium channel blockers, especially verapamil and diltiazem (less is known about amlodipine)</td>
</tr>
<tr>
<td></td>
<td>Adenosine</td>
<td>Calcium channel blockers, especially verapamil and diltiazem (less is known about amlodipine)</td>
<td>Calcium channel blockers, especially verapamil and diltiazem (less is known about amlodipine)</td>
</tr>
<tr>
<td></td>
<td>Quinidine</td>
<td>Calcium channel blockers, especially verapamil and diltiazem (less is known about amlodipine)</td>
<td>Calcium channel blockers, especially verapamil and diltiazem (less is known about amlodipine)</td>
</tr>
<tr>
<td></td>
<td>Procainamide</td>
<td>Calcium channel blockers, especially verapamil and diltiazem (less is known about amlodipine)</td>
<td>Calcium channel blockers, especially verapamil and diltiazem (less is known about amlodipine)</td>
</tr>
<tr>
<td></td>
<td>Lidocaine</td>
<td>Calcium channel blockers, especially verapamil and diltiazem (less is known about amlodipine)</td>
<td>Calcium channel blockers, especially verapamil and diltiazem (less is known about amlodipine)</td>
</tr>
<tr>
<td><strong>Ischemia</strong></td>
<td>Nitrates</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
</tr>
<tr>
<td></td>
<td>Low-dose (&lt;100 mg) ASA</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
</tr>
<tr>
<td></td>
<td>β-Blockers</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
</tr>
<tr>
<td></td>
<td>Heparin (unfractionated or low molecular weight)</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
</tr>
<tr>
<td></td>
<td>Tissue plasminogen activator</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
</tr>
<tr>
<td></td>
<td>Streptokinase</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
<td>HMG-CoA reductase inhibitors (&quot;statins&quot;) have concerning animal pregnancy data, but very limited reported human experiences thus far have been encouraging; should only be used in pregnancy when short-term benefits are clear</td>
</tr>
<tr>
<td><strong>Heart failure</strong></td>
<td>Furosemide</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
</tr>
<tr>
<td></td>
<td>Digoxin</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
</tr>
<tr>
<td></td>
<td>Hydralazine</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
</tr>
<tr>
<td></td>
<td>β-Blockers</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
</tr>
<tr>
<td></td>
<td>Dopamine</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
</tr>
<tr>
<td></td>
<td>Dobutamine</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
</tr>
<tr>
<td><strong>Hypertension</strong></td>
<td>Labetalol</td>
<td>ACE inhibitors</td>
<td>ACE inhibitors</td>
</tr>
<tr>
<td></td>
<td>β-Blockers</td>
<td>ACE inhibitors</td>
<td>ACE inhibitors</td>
</tr>
<tr>
<td></td>
<td>Nifedipine</td>
<td>ACE inhibitors</td>
<td>ACE inhibitors</td>
</tr>
<tr>
<td></td>
<td>Hydralazine</td>
<td>ACE inhibitors</td>
<td>ACE inhibitors</td>
</tr>
<tr>
<td></td>
<td>Methyldopa</td>
<td>ACE inhibitors</td>
<td>ACE inhibitors</td>
</tr>
<tr>
<td></td>
<td>Thiazide diuretics (in this category for the treatment of hypertension because of effects of blood volume in pregnancy)</td>
<td>Thiazide diuretics (in this category for the treatment of hypertension because of effects of blood volume in pregnancy)</td>
<td>Thiazide diuretics (in this category for the treatment of hypertension because of effects of blood volume in pregnancy)</td>
</tr>
<tr>
<td></td>
<td>Clonidine, prazosin, verapamil, diltiazem, and amlodipine (in this category for the treatment of hypertension because of limited data on safety and the availability of many good alternatives with more data)</td>
<td>Thiazide diuretics (in this category for the treatment of hypertension because of effects of blood volume in pregnancy)</td>
<td>Thiazide diuretics (in this category for the treatment of hypertension because of effects of blood volume in pregnancy)</td>
</tr>
<tr>
<td></td>
<td>Nitroprusside (fetal cyanide toxicity possible at high doses)</td>
<td>Thiazide diuretics (in this category for the treatment of hypertension because of effects of blood volume in pregnancy)</td>
<td>Thiazide diuretics (in this category for the treatment of hypertension because of effects of blood volume in pregnancy)</td>
</tr>
</tbody>
</table>

ASA, acetylsalicylic acid; ACE, angiotensin-converting enzyme.
CHAPTER 78  Cardiac Disease and Hypertensive Disorders in Pregnancy 915
demed acceptable during pregnancy. Contrast agents appear to be well tolerated by the fetus. MRI has not been associated with any ill effects in human pregnancies. Because fetal well-being is dependent on maternal well-being, more harm will generally be caused to a mother and her fetus by withholding necessary investigations than by obtaining them.

Women with congenital heart disease should undergo a detailed fetal ultrasound in the early second trimester to allow early diagnosis of congenital heart disease in the fetus. This will allow informed decision-making by the mother, and will prepare the neonatology team should a problem be present.

Labor and delivery and the first 72 hours postpartum warrant special consideration with respect to assembling the appropriate team and determining what monitoring will be needed. For most cardiac patients, a multidisciplinary patient care conference should be assembled well in advance of the anticipated time of delivery and a written care plan developed for the peripartum management of the patient. This team should generally include representation from critical care, nursing, anesthesia, obstetrics, and cardiology. The plans that are developed should be explicit and detailed and recognize that the labor and delivery room is a place where cardiac care is not commonly provided. Even the best-trained obstetricians and obstetric nurses will lack the volume of experience in the management of cardiac cases that is common among cardiac and critical care providers. It is our conviction that joint nursing of such patients by obstetric and cardiology-trained nurses during labor and delivery, followed by postpartum care in a cardiac or critical care unit, seems the ideal approach when possible. Table 78.8 offers a comprehensive checklist of parameters to be considered and addressed in a multidisciplinary patient care conference dedicated to developing a labor and delivery plan for a cardiac patient.

The mode of delivery should not generally be determined by medical concerns. The need for cesarean deliveries is generally dictated by obstetric concerns, and vaginal deliveries should generally be viewed as the safest and best option for cardiac patients. The choice between spontaneous labor and elective induction of labor should be made both on the likelihood of successful induction and the availability of medical expertise and resources should a cardiac patient go spontaneously into labor during the off hours and weekends.

Most patients should be kept in neutral fluid balance over the course of their delivery period, and careful monitoring of both input and output will be essential. Early and good anesthe sia is important to decrease the cardiac work of delivery, and most patients should receive regional anesthesia in a manner that will minimize the need for the fluid boluses typically given to decrease the hypotension associated with establishing regional anesthesia. It is also important to consider that certain lesions, such as an aortic stenosis, may be highly volume dependent and require this additional fluid support.

Intra-arterial lines are advisable for cardiac lesions for which moment-to-moment monitoring of blood pressure might be desirable, such as severe aortic stenosis. The role of the pulmonary artery catheter in the laboring patient remains unclear and, in the absence of clear benefit, it is this author’s opinion that their use during delivery should be limited to the most severe cardiac cases, if it is used at all.

Bacterial endocarditis prophylaxis is no longer recommended by the American Heart Association for vaginal or cesarean deliveries because the bacteremia associated with delivery is unlikely to cause endocarditis (67). If done at all, endocarditis prophylaxis should be reserved for patients with prosthetic heart valves, a prior history of subacute bacterial endocarditis, complex cyanotic congenital heart disease, or surgically constructed systemic pulmonary shunts or conduits, and an agent active against enterococci such as penicillin, ampicillin, or vancomycin should be utilized.

It is critical that all team members recognize that the cardiac patient remains at risk for at least 72 hours postpartum, so despite the sense of completion that comes with a successful delivery, caregivers need to remain vigilant for early signs of deterioration in the days following the birth.

Specific Lesions

Mitral Stenosis. Rheumatic heart disease remains a potential form of heart disease in pregnancy despite its declining incidence in the developed world. Mitral stenosis (MS) accounts for approximately 90% of the rheumatic valvular lesions in pregnancy. It often presents for the first time in pregnancy; complications include atrial fibrillation, pulmonary edema, and thromboembolic stroke. The normal physiologic cardiac changes in pregnancy are poorly tolerated in MS. Most patients will experience some worsening of symptoms during pregnancy, and the risk continues during labor and delivery as the increased blood volume after delivery of the placenta may worsen pulmonary edema (68). Complication rates were found in one study to be 38% in moderately severe MS and 67% in severe cases (69). Avoidance of tachycardia (which decreases time available for ventricular filling), increased PA pressure, decreased systemic vascular resistance, and increased central blood volume are essential to patient management. For this reason, many patients will benefit from β-blockade to improve left ventricular filling time during pregnancy (70). Echocardiograms should be done once every trimester and with any change in status in these patients. Careful attention should be focused on pulmonary pressures (although echocardiography may provide a less reliable estimate of pulmonary pressures in pregnancy). Pulmonary edema should be treated with diuretics and β-blockade. If severe symptoms persist despite optimal medical management, percutaneous mitral balloon valvuloplasty, commissurotomy, or even valve replacement may be warranted; all have been successfully performed in pregnancy (71–73). Open procedures may be associated with a higher risk of miscarriage, fetal loss, and preterm labor, and thus balloon valvuloplasty may be preferable at centers experienced with this procedure. Although surgery can be performed at any point in the pregnancy, the risk to the fetus is lowest in the second trimester.

If atrial fibrillation occurs, it should be treated promptly to decrease tachycardia and the associated risk of a low CO state or degeneration into more malignant dysrhythmias. Rate control, full anticoagulation with heparin, and consideration of either medical or electrical cardioversion remain the core management principles in pregnant women with atrial fibrillation as they are for nonpregnant women.

For labor and delivery (vaginal or cesarean), excellent pain control is important and is best achieved with early establishment of regional anesthesia. Control of pain will limit the undesirable effects of labor on heart rate and blood pressure (which are tachycardia and increased systemic vascular resistance). A conservatively dosed lumbar epidural anesthetic with special attention to fluid status, left uterine displacement, and careful use of α-adrenergic agents to treat hypotension is often helpful. These patients are dependent on high left ventricular (text continues on page 14)
### Table 78.8 Cardiac Patient Delivery Plan Checklist

**Prior to Hospitalization**

<table>
<thead>
<tr>
<th>Question</th>
<th>Consider for all levels of risk.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Is additional testing needed to assess risk or guide peripartum therapy?</td>
<td></td>
</tr>
<tr>
<td>- Baseline ECG done in third trimester</td>
<td></td>
</tr>
<tr>
<td>- Echocardiogram at any time in the past for lowest-risk lesions, in this pregnancy for moderate-risk lesions, and in the third trimester for high- and highest-risk lesions</td>
<td></td>
</tr>
<tr>
<td>- Stress testing (exercise echo or dobutamine echo in past year for patients with known or suspected ischemic heart disease or more recently if they are symptomatic)</td>
<td></td>
</tr>
<tr>
<td>- EP testing for life-threatening dysrhythmia (investigation often deferred until postpartum but can be done in pregnancy if warranted)</td>
<td></td>
</tr>
<tr>
<td>Has the patient’s cardiac status been optimized?</td>
<td></td>
</tr>
<tr>
<td>- Is medical therapy optimized and have appropriate dose adjustments been made for the changes of pharmacokinetics in pregnancy?</td>
<td></td>
</tr>
<tr>
<td>- Are there interventions that would be done if the woman was not pregnant that should be done while she is pregnant to optimize patient’s status for delivery (e.g., diagnostic or therapeutic cardiac catheterization [angioplasty/stent], valvuloplasty, valve replacement, diagnostic or therapeutic EP studies, AICD or pacemaker placement or adjustment)?</td>
<td></td>
</tr>
<tr>
<td>- Multidisciplinary team meeting needed and arranged (generally should have occurred by 34 wk). Team should include:</td>
<td>Consider having meeting of RN/MFM/Anesthesia for moderate-risk patients and all of the listed providers for high- and highest-risk patients.</td>
</tr>
<tr>
<td>- Nursing (LDR and postpartum care RN +/- ICU/CCU nursing)</td>
<td></td>
</tr>
<tr>
<td>- Maternal fetal medicine</td>
<td></td>
</tr>
<tr>
<td>- Anesthesia (ideally obstetric anesthesia; also consider cardiac anesthesia for high- and highest-risk cases)</td>
<td></td>
</tr>
<tr>
<td>- Cardiology</td>
<td></td>
</tr>
<tr>
<td>- ICU/CCU doctor</td>
<td></td>
</tr>
<tr>
<td>Written delivery plan should be generated and distributed and made available to all relevant parties including nursing (should include who to call and how to do so when the patient comes in)</td>
<td>Consider for all levels of risk.</td>
</tr>
<tr>
<td>Case-specific nursing education should occur in advance of delivery.</td>
<td>Consider for all levels of risk. For lowest-risk lesions it may be adequate to have a standardized nursing care plan or the written delivery plan.</td>
</tr>
</tbody>
</table>

**Intrapartum**

<table>
<thead>
<tr>
<th>Question</th>
<th>Decision to be made on the basis of obstetric factors and the need to ensure availability of necessary members of the care team. Planned delivery may be advisable for high- and highest-risk patients. Decision to be made on the basis of local facilities and expertise. In general, care during delivery is best provided in LDR and afterward in medical setting.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Determine mode and timing of delivery:</td>
<td></td>
</tr>
<tr>
<td>- Planned induction at what gestation/cervical status</td>
<td></td>
</tr>
<tr>
<td>- Planned cesarean delivery at what gestation</td>
<td></td>
</tr>
<tr>
<td>- Spontaneous delivery</td>
<td></td>
</tr>
<tr>
<td>Delivery location:</td>
<td></td>
</tr>
<tr>
<td>- Standard LDR</td>
<td></td>
</tr>
<tr>
<td>- Specialized LDR</td>
<td></td>
</tr>
<tr>
<td>- Obstetric ICU</td>
<td></td>
</tr>
<tr>
<td>- MICU</td>
<td></td>
</tr>
<tr>
<td>- CCU</td>
<td></td>
</tr>
<tr>
<td>Delivery personnel who should be notified of admission (make sure needed parties available on day of any planned delivery)</td>
<td>Consider having both LDR nurse and critical care nurse for high- and highest-risk patients.</td>
</tr>
<tr>
<td>Medical Attendants:</td>
<td></td>
</tr>
<tr>
<td>- Obstetrician</td>
<td></td>
</tr>
<tr>
<td>- Cardiologist</td>
<td></td>
</tr>
<tr>
<td>- Anesthesia (ideally obstetric anesthesia; also consider cardiac anesthesia for high- and highest-risk cases)</td>
<td></td>
</tr>
<tr>
<td>- Intensivist</td>
<td></td>
</tr>
<tr>
<td>Nursing (Consider need for team approach of ICU/CCU/RR/ER nurse with LDR nurse. Define necessary nurse-to-patient ratio):</td>
<td>Consider required response time of ACLS trained personnel if nursing team caring for patient is not ACLS certified/experienced.</td>
</tr>
<tr>
<td>- LDR nurse</td>
<td></td>
</tr>
<tr>
<td>- LDR nurse with ACLS training</td>
<td></td>
</tr>
<tr>
<td>- LDR nurse with ACLS and special critical care training</td>
<td></td>
</tr>
<tr>
<td>- Critical care nurse (ICU/CCU/RR/ER) nurse</td>
<td></td>
</tr>
<tr>
<td>EDUCATION</td>
<td></td>
</tr>
<tr>
<td>- Verify written care plan is available to all team members</td>
<td>Consider summary in-service on day of delivery for high- and highest-risk patients and any patient for whom medications may be required urgently that are not routinely used on obstetric floors.</td>
</tr>
<tr>
<td>- Is a “recap” in-service for care team advisable on day of delivery?</td>
<td></td>
</tr>
</tbody>
</table>
CHAPTER 78  Cardiac Disease and Hypertensive Disorders in Pregnancy

TABLE 78.8 Cardiac Patient Delivery Plan Checklist (Continued)

**MONITORING**
- Cardiac monitor options (choose one)
  - Not necessary
  - To be in room but does not need to be on if patient asymptomatic
  - To be on patient at all times but not continuously observed
  - To be on patient at all times and should be continually observed by ACLS-trained individual
  - To be on patient at all times and should be observed at all times by critical care nurse/MD/PA/NP

**Pulse oximeter (choose one)**
- Not necessary
- Readily available but use only with symptoms
- In room and check hourly
- In room and on continuously

**Fetal monitoring**
- Obtain explicit plan from obstetric team including who will read the fetal monitoring strips and the plan of action should they be concerning.

**Defibrillator**
- On the unit with ready access to defibrillator pads
- Defibrillator and defibrillator pads in the room
- Defibrillator pads on patient but machine not hooked up
- Patient to be monitored using defibrillator with defibrillator pads

**IV access**
- No IV necessary
- Single peripheral IV lines needed
- Two peripheral IV lines needed
- Central line
- Central line with CVP
- Central line with pulmonary artery catheter

**FLUID BALANCE**
- All patients need strict ins and outs measured throughout hospitalization.
- Most cardiac patients we will want to keep in a neutral fluid balance during hospitalization.

<table>
<thead>
<tr>
<th>Fluid to be run:</th>
<th>Rate:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial line</td>
<td>No arterial line needed</td>
</tr>
<tr>
<td>Arterial line warranted</td>
<td></td>
</tr>
</tbody>
</table>

**MEDICATIONS**
- Need for SBE prophylaxis (SBE prophylaxis for high-risk lesions only and even then not absolutely necessary)
- Special issues related to interactions with commonly used obstetric medications
- Possibly necessary cardiac medications not routinely used on obstetric units
  - Need for RN/MD education regarding these medications
  - Need for written instructions with respect to preparation and administration of this medication
  - Need for medication to be at bedside
  - Pharmacy notified in advance of request (especially if free-standing obstetrics hospital)

**Anesthetic concerns**
- Special issues related to anesthesia
- Special issues with respect to cautery for cesarean delivery

**Thromboprophylaxis**
- Intermittent compression stockings
- Heparin 5,000 units SQ q12h
- Heparin 5,000 units SQ q8h
- Enoxaparin 40 units SQ daily
- Enoxaparin 30 units SQ q12h
- Full anticoagulation necessary in peripartum period (please see peripartum anticoagulation protocol)

Most cardiac patients, aside from the highest-risk patients or those with a history of life-threatening hemodynamically unstable arrhythmias, will not need continuous monitoring by ACLS-trained personnel. Low-risk lesions may warrant one of the first two approaches. Moderate- and high-risk lesions may warrant only option 3.

Pulse oximeter may provide evidence of CHF but should always be interpreted in view of strength of pulse signal. Option 2 is probably adequate for most cardiac patients aside from those with cyanotic heart disease or those in CHF, who probably warrant option 4.

Obtain explicit plan from obstetric team including who will read the fetal monitoring strips and the plan of action should they be concerning.

Option 1 is generally adequate. Consider other options in highest-risk patients.

Option 2 is enough for most patients. Consider central line in highest-risk lesions.

Make sure to add in all fluids given with medications and for regional anesthesia.

Arterial line advisable when hemodynamics make moment-to-moment monitoring of blood pressure useful (e.g., aortic stenosis)

May be given for prosthetic heart valves, prior SBE, complex cyanotic congenital heart disease, surgically constructed systemic pulmonary shunts or conduits but not necessary for the rest

- Standard dosing: Ampicillin 2 g IV plus gentamicin 1.5 mg/kg within 30 min of delivery; ampicillin 1 g IV 6 h after delivery
- Penicillin allergy: Vancomycin 1 g IV over 1–2 h plus gentamicin 1.5 mg/kg IV within 30 min of delivery

Anesthesia will determine preferred modality of anesthesia timing and precautions in technique. Implanted defibrillators may need to be turned off prior to surgery because of interference from cautery.

Options 1 and 2 compatible with epidural anesthesia. Options 3–6 should only be done after the epidural catheter is removed. Consider option 1 or 2 antepartum and option 2, 3, 4, or 5 for most patients postpartum while in hospital.
## TABLE 78.8 Cardiac Patient Delivery Plan Checklist (Continued)

<table>
<thead>
<tr>
<th>POSTPARTUM</th>
<th>Low-risk patients probably only warrant the usual period of observation given all patients. Moderate-risk patients warrant 6 h. High-risk patients warrant between 6 and 48 h and highest-risk patients 72–96 h.</th>
</tr>
</thead>
</table>
| How long postpartum will patient require special observation? | • Usual period of postpartum observation  
• 6 h  
• 12 h  
• 24 h  
• 48 h  
• 72 h  
• 96 h  
Location of special postpartum observation | • Room on regular postpartum floor  
• Room on high-risk antenatal floor  
• Standard LDR/postop CS area  
• Specialized LDR/postop CS area  
• Obstetric ICU  
• MICU  
• CCU  
• Other  
Location of special postpartum observation | Option 1, 2, or 3 for low-risk; 2, 3, or 4 for moderate-risk; and 4, 5, 6, or 7 for high- and highest-risk patients  
| MONITORING | Cardiac monitor options (choose one)  
• Not necessary  
• To be in room but does not need to be on if patient asymptomatic  
• To be on patient at all times but not continuously observed  
• To be on patient at all times and should be continually observed by ACLS-trained individual  
• To be on patient at all times and should be observed at all times by critical care nurse/MD/PA/RNP  
Postpartum monitoring/interventions recommended and for how long | No special monitoring or interventions for low-risk patients; 1 for 24 h for moderate-risk patients; and 1 or 2 for 48–72 h for high- and highest-risk patients  
| All patients need strict ins and outs measured throughout hospitalization.  
Most cardiac patients we will want to keep in a neutral fluid balance during hospitalization. | Make sure to add in all fluids given with medications and for regional anesthesia.  
| | Fluid to be run:  
Rate:  
Pulse oximeter in room and checked how often  
• Not necessary  
• In room but use only with symptoms  
• In room and check hourly  
• In room and on continuously  
| Availability of ACLS trained physician/PA/RNP: | Consider required response time of ACLS-trained personnel if not present  
• Special availability not necessary  
| Medical attendants: |  
| Obstetrician  
Cardiologist  
Anesthesia (ideally obstetric anesthesia; also consider cardiac anesthesia for high- and highest-risk cases)  
General internist  
ICU team  
CCU team  
Medical ICU vs. LDR with cardiac nursing (consider need for team approach and necessary nurse-to-patient ratio)  
LDR nurse  
LDR nurse with ACLS training  
LDR nurse with ACLS and special critical care training  
Critical care (ICU/CCU/RR/ER) nurse  
Defibrillator  
• On the floor  
• In the room  
Pads on patient  
Patient to be monitored with defibrillator pads on  
Special issues related to interactions with commonly used obstetric medications  
| | Option 1 is generally adequate. Consider other options in highest-risk patients.  
| -Peripheral IV  
-Central line  
-CVP  
-Arterial line  
Pulmonary artery catheter  
Pulmonary artery catheter  
Pulse oximeter in room and checked how often |  
• Not necessary  
• In room but use only with symptoms  
• In room and check hourly  
• In room and on continuously  
| Pulse oximeter in room and checked how often | Pulse oximeter may provide evidence of CHF but should always be interpreted in view of strength of pulse signal. Option 2 probably adequate for most cardiac patients aside from those with cyanotic heart disease and those in CHF, who probably warrant option 4.  
| Availability of ACLS trained physician/PA/RNP: |  
• Special availability not necessary  
| Medical attendants: |  
| Obstetrician  
Cardiologist  
Anesthesia (ideally obstetric anesthesia; also consider cardiac anesthesia for high- and highest-risk cases)  
General internist  
ICU team  
CCU team  
Medical ICU vs. LDR with cardiac nursing (consider need for team approach and necessary nurse-to-patient ratio)  
LDR nurse  
LDR nurse with ACLS training  
LDR nurse with ACLS and special critical care training  
Critical care (ICU/CCU/RR/ER) nurse  
Defibrillator  
• On the floor  
• In the room  
Pads on patient  
Patient to be monitored with defibrillator pads on  
Special issues related to interactions with commonly used obstetric medications  
| | Option 1 is generally adequate. Consider other options in highest-risk patients.  
| -Peripheral IV  
-Central line  
-CVP  
-Arterial line  
Pulmonary artery catheter  
Pulse oximeter in room and checked how often |  
• Not necessary  
• In room but use only with symptoms  
• In room and check hourly  
• In room and on continuously  
|
after the first trimester, it is important to be aware that both surgical repair, or valve replacement. Ideally, such procedures are best done in the middle of the pregnancy but, if necessary, can be done at any time. When severe disease is identified, it will likely be those patients with severe obstructive lesions or very poor ejection fractions. If the pulmonary artery catheter is used for patients with MS, it will be important to remember that the PAOP may overestimate left ventricular end-diastolic pressure.

Aortic Stenosis

Aortic stenosis is a valvular lesion rarely seen during pregnancy, and can be of rheumatic or congenital origin. Although bicuspid aortic valves are common, they are unlikely to be associated with significant stenosis in the childbearing years. They may be associated with an increased risk of both coarctation and dissection. Although mild to moderate aortic stenosis is generally well tolerated in pregnancy, severe stenosis (defined as <1.0 cm²) carries a significant fetal and maternal risk. The rate of complication varies from 10% to 31%, depending on the severity of the lesion (76–78). Ideally, symptomatic aortic stenosis should be repaired prior to pregnancy. If the patient is classified as NYHA functional class III or IV while pregnant, consideration should be given to percutaneous valvuloplasty, surgical repair, or valve replacement. Ideally, such procedures are best done in the middle of the pregnancy but, if necessary, can be done at any time. When severe disease is identified after the first trimester, it is important to be aware that both labor and delivery and a late termination are associated with significant risks. Due to the fixed outflow obstruction, these patients will not tolerate sudden drops in volume or preload, and their peripartum period should be managed in such a way as to minimize the risk of such events and ensure the ability to respond rapidly if and when they do occur. Arterial lines are strongly advised, and the use of pulmonary artery catheters, while not proven, may be of benefit.

In the past, with severe stenotic lesions of the aorta, regional anesthesia has been avoided because of the resulting local anesthetic–induced sympathetic block, which can lead to bradycardia and decreased venous return. However, good results have been obtained in patients with severe aortic stenosis managed during labor with a carefully titrated epidural anesthetic (78,79).

Mitrail and Aortic Insufficiency

Mitrail insufficiency is the second most common valvular lesion seen in pregnancy, and is typically due to rheumatic heart disease (75). Aortic insufficiency is less common, and may be due to rheumatic, infectious, or rheumatologic conditions. These lesions, when found in isolation, tend to do well in pregnancy unless there is associated ventricular decompensation. Treatments when symptomatic may include diuretics, β-blockers, or vasodilators, but angiotensin receptor blockers should not be used despite the benefits of afterload reduction. Increases in systemic vascular resistance, decreased heart rate, atrial dysrhythmias, and myocardial depressants may be poorly tolerated. Perhaps the most important peripartum issue for these patients is early regional anesthesia to prevent pain-associated increases in systemic vascular resistance.

**TABLE 78.8 Cardiac Patient Delivery Plan Checklist (Continued)**

<table>
<thead>
<tr>
<th>Thromboprophylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>START___________</td>
</tr>
<tr>
<td>DURATION__________</td>
</tr>
<tr>
<td>• Intermittent compression stockings</td>
</tr>
<tr>
<td>• Heparin, 5,000 units SQ q2h</td>
</tr>
<tr>
<td>• Heparin, 5,000 units SQ q8h</td>
</tr>
<tr>
<td>• Enoxaparin, 40 units SQ daily</td>
</tr>
<tr>
<td>• Enoxaparin, 30 units SQ q12h</td>
</tr>
<tr>
<td>• Full anticoagulation will be needed postpartum; See peripartum anticoagulation protocol</td>
</tr>
<tr>
<td>• Possibly necessary cardiac medications not routinely used on obstetric units</td>
</tr>
<tr>
<td>• Need for RN/MD education regarding these medications</td>
</tr>
<tr>
<td>• Need for written instructions with respect to preparation and administration of this medication</td>
</tr>
<tr>
<td>• Need for medication to be at bedside</td>
</tr>
<tr>
<td>• Pharmacy notified in advance of request (especially if free-standing obstetric hospital)</td>
</tr>
</tbody>
</table>

**DISCHARGE PLANNING**

Will there be any adjustments to medication necessary postpartum (e.g., resumption/replacing of medications stopped/started because of pregnancy OR dosing adjustments necessary in postpartum period because of increases made during pregnancy)?

Who will follow the patient after discharge and when will patient need to be seen (letter or phone call should be sent/made to receiving MD):

- Cardiology
- Primary care doctor
- Obestrics

**ECG, electrocardiogram; EP, electrophysiologic; AICD, automatic implantable cardioverter-defibrillator; LDR, labor and delivery room; RN, registered nurse; ICU, intensive care unit; CCU, critical care unit; MFM, maternal–fetal medicine; MICU, medical intensive care unit; RR, recovery room; ER, emergency room; ACLS, Advanced Cardiac Life Support; MD, doctor; PA, physician assistant; RN, registered nurse practitioner; CHF, congestive heart failure; CVP, central venous pressure; SBE, subacute bacterial endocarditis; CS, cesarean section.**
**Congenital Heart Disease**

Approximately 25% of heart disease in pregnancy is congenital. It can be categorized as left-to-right shunt, right-to-left shunt, and aortic lesions.

**Left-to-Right Shunt**

The most common congenital heart lesions are atrial septal defects (ASDs) and ventricular septal defects (VSDs), which are usually well tolerated in pregnancy. The risk of cardiac complications is greatest in patients with large defects. Congestive heart failure (due to increased blood volume in pregnancy leading to cardiac decompensation), atrial dysrhythmias, shunt reversal (occurring due to sudden systemic hypotension), and thromboembolic disease are all possible complications seen with ASD and VSD in pregnancy. Ideally, hemodynamically significant septal defects should be repaired prior to pregnancy. However, when symptomatic septal defects present in pregnancy, the principles of management include (a) acetyl salicylic acid (ASA) 81 mg daily to prevent thromboembolism, (b) use of diuretics and digoxin to treat heart failure, (c) avoidance of hypotension with epidural administration or postpartum blood loss, and (d) rapid rate control with any arrhythmia.

**Right-to-Left Shunt and Pulmonary Hypertension**

The high-risk congenital disorders in pregnancy include right-to-left shunts, as seen in Eisenmenger syndrome (any congenital heart lesion with a bidirectional or right-to-left shunt at the atrial, ventricular, or aortic level), and any other lesions associated with significant pulmonary hypertension. Patients with uncorrected cyanotic heart disease have increased spontaneous abortion rates, pulmonary embolization, congestive heart failure, and incidence for congenital heart defects in the fetus. A high hematocrit (≥65%) is not only an indication of the severity of the cardiac disease, but also in itself has a poorer prognosis secondary to complications from hyperviscosity (decreased CO, organ hypoperfusion, and thrombosis).

During pregnancy, right-to-left shunting is increased because of decreased systemic vascular resistance, resulting in decreased pulmonary artery perfusion and hypoxia. A review on maternal and fetal outcome in patients with Eisenmenger syndrome reveals maternal mortality rates of 25% to 52% and fatal loss as high as 44% (80–82). Because of the grim prognosis for these pregnancies, these women should be strongly warned about the dangers of pursuing a pregnancy and, if they do become pregnant, should be offered the opportunity for an early termination. If they continue with the pregnancy, they may warrant hospitalization from 20 weeks onward. Oxygen should be administered for dyspnea, and prophylactic heparin should be considered throughout pregnancy and for 6 weeks postpartum. The mode of delivery should be determined on the basis of obstetric indications. Pulmonary artery catheterization can carry additional risks in patients with significant pulmonary hypertension, and should probably be avoided in these patients. Active efforts should be made to avoid sudden decreases in systemic vascular resistance, blood volume, and venous return. Increased pulmonary vascular resistance promotes right-to-left shunting; therefore, hypercapnia and hypoxia are to be avoided. How best to provide peripartum anesthesia to these patients is not clear, and discussion of this matter is beyond the scope of this chapter. What is clear is that if regional anesthesia is used, care must be taken to prevent precipitous drops in venous return. Patients with pulmonary hypertension and/or Eisenmenger syndrome should be observed for 72 hours postpartum in a cardiac setting, as many of the maternal deaths associated with these conditions will occur during this period.

**Aortic Disease**

Coarctation of the aorta and aortic manifestations of Marfan syndrome pose significant problems in pregnancy (83–85). The physiologic changes during pregnancy, including increased blood volume and increased blood pressure during labor and delivery, may promote aortic dissection in either of these conditions. Patients with coarctation of the aorta may also suffer from worsening hypertension or congestive heart failure in pregnancy.

Marfan syndrome is often associated with aortic dilation, aortic valve regurgitation, and mitral valve disease. Aortic dissection occurs in about 10% of patients with Marfan syndrome who undergo a pregnancy, and is most likely to occur if the aortic root measures beyond 4.5 cm in diameter (86,87). Ideally, women with this severity of aortic root dilation should have their aorta repaired prior to pregnancy. However, if they have not, serial echocardiography during pregnancy to watch for worsening dilation should be performed. If the root is increasing in size, aortic repair should be considered. The activity of patients with significant aortic dilation in pregnancy should be limited, and they should be placed on β-blockers to decrease shear stresses upon the vessel wall (88,89). Although we generally teach that the indications for cesarean delivery are obstetric and not medical, it is common practice to deliver women with aortic roots dilated beyond 4.0 cm by cesarean to avoid additional stress on the aorta associated with the pain and pushing of a vaginal delivery. However, it is worth noting that the majority of aortic dissections in these patients occur prior to the onset of labor.

Aortic coarctation in pregnancy is associated with an increased risk of worsening hypertension and, less commonly, congestive heart failure or preeclampsia (90). It is much less likely to be associated with aortic dissection than Marfan syndrome, but dissection can and does occur. Blood pressure should be kept less than 160/100 mmHg in these patients but not brought below 120/70 mmHg, as there may be a significant gradient between blood pressure measurement in the arm and the estimated blood pressure of the placenta circulation that is distal to the aortic narrowing. β-Blockers are the preferred antihypertensives for these patients. Patients with coarctation can undergo a vaginal delivery but should have a limited second stage (i.e., prolonged pushing should be avoided by the use of vacuum extractor or forceps).

**Tetralogy of Fallot**

Tetralogy of Fallot is the most common cyanotic congenital heart disease. It consists of a VSD, an overriding aorta, infundibular pulmonary stenosis, and secondary right ventricular hypertrophy. Patients with uncorrected tetralogy have significant complications in pregnancy including biventricular failure, dysrhythmias, stroke, and risk of shunt reversal with worsening cyanosis. Preconception surgical repair should be undertaken if at all possible. If these patients do proceed with a pregnancy unrepaired, they should be managed in a manner similar to patients with Eisenmenger syndrome.
Patients with a surgically corrected tetralogy of Fallot who enter a pregnancy with a good functional status generally tolerate pregnancy well. The main risks are right-sided heart failure and dysrhythmias. Their volume status should be monitored throughout pregnancy and complaints of palpitations or syncope investigated with a Holter or an event monitor. Delivery should include cardiac monitoring (91–94).

**Other Repaired Congenital Heart Conditions**

An increasing number of women with congenital heart problems that were repaired in childhood are reaching adulthood and undergoing pregnancy. In general, these patients’ course in pregnancy is readily predictable by the parameters outlined earlier in this chapter. The majority will have a good pregnancy outcome for both themselves and their offspring if they enter the pregnancy with a good functional status (75,95).

**Peripartum Cardiomyopathy**

The National Heart, Lung, and Blood Institute (NHLBI) defines PPCM as the new onset of systolic dysfunction occurring in the absence of other plausible causes anytime between the final month of pregnancy up to 5 months postpartum. The incidence is between 1 in 2,000 to 1 in 15,000 pregnancies, and may be increasing (96–98). Multiparity, twin gestation, maternal age greater than 30 years, presence of preclampsia/eclampsia and black race are all known to be risk factors, but causality is not yet proven. Precise mechanisms that lead to PPCM remain poorly defined. Many etiologic processes have been suggested (99) including abnormal immune response to pregnancy, maladaptive response to the hemodynamic changes of pregnancy, prolonged tocolysis, increased concentration of inflammatory cytokines, angiogenic imbalance mediated via prolactin and its fragments (100), and possible genetic mechanisms, though a specific genetic mutation causally associated with PPCM is yet to be identified. Generally, patients with PPCM have a greater recovery of LV function and a better prognosis than patients with other forms of dilated cardiomyopathy. Most women with PPCM will have complete or at least partial recovery within 6 months of onset while women with depressed LV function beyond 6 months of diagnosis have worse clinical outcomes and higher 5-year mortality rates (101). Mortality can be due to end-stage heart failure, arrhythmia, or thromboembolism.

Pathologic findings include four-chamber enlargement with normal coronary arteries and valves. Light microscopic findings include myocardial hypertrophy and fibrosis with scattered mononuclear infiltrates. Clinical signs include symptoms of ventricular failure with possible associated dysrhythmias and/or pulmonary emboli. Treatment includes bed rest, sodium restriction, diuresis, and preload/afterload reduction with a calcium channel blocker and hydralazine while pregnant and an angiotensin-converting enzyme inhibitor postpartum. Because pregnancy is associated with increased risk of thrombosis, patients with an ejection fraction less than 35% should be considered for anticoagulation with low–molecular-weight heparin (LMWH) while pregnant and warfarin postpartum. Antidysrhythmics should be used in a manner similar to what would be done for any patient with an idiopathic cardiomyopathy. Although the exact risk remains unclear, there is evidence that PPCM may recur or worsen with subsequent pregnancies (102).

**Hypertrophic Cardiomyopathy**

During pregnancy, the course of hypertrophic cardiomyopathy is variable because while the normal increase of blood volume is beneficial, the decrease in systemic vascular resistance and the increase in heart rate may be detrimental. Several large case series have highlighted the risks for these patients during pregnancy (103–106). The risks are inherently greater for those women who are symptomatic before pregnancy or in those with severe left ventricular outflow tract obstruction. Complications are not common, but include congestive heart failure, chest pain, supraventricular tachycardias (SVTs), ventricular tachycardia, and sudden death. Complications can occur at any point in the pregnancy or during labor as a result of stress, pain, and increased circulating catecholamines. Moreover, the immediate postpartum period can increase risk due to blood loss and decrease in systemic vascular resistance. Atrial fibrillation and SVTs are a common feature of this cardiac anomaly; thus, cardiolytic β-blockers and verapamil are usually administered to these patients. Tocolytics, sympathomimetic agents, and digoxin should be avoided in these patients, as they may increase the risk of dysrhythmia. The peripartum period should include cardiac monitoring and use of forceps or vacuum extractor so that the mother has to do little or no pushing. If regional anesthesia is employed, it should be done incrementally and with agents that minimize the risk of a sudden drop in preload.

**Ischemic Heart Disease in Pregnancy**

Although myocardial infarction in pregnancy is uncommon, with an incidence estimated at between 1 in 10,000 and 1 in 35,700, it does appear to be increasing. Risk factors include advancing age, preeclampsia, multiparity, chronic hypertension, obesity, and diabetes. Myocardial infarctions associated with pregnancy can occur at any time during gestation, with one report finding that 38% occurred antepartum, 21% intrapartum, and 41% in the first 6 weeks postpartum. Maternal mortality rate ranges from 7% to 35%, with a disproportionate number of deaths occurring among the antenatal cases (107–109). A large portion of pregnancy-associated myocardial infarctions are not due to atherosclerotic heart disease but instead due to coronary artery in situ thrombus formation, dissection, or spasm.

Diagnosis of ischemic heart disease in pregnancy does require considering it as part of the differential diagnosis, even in the absence of traditional risk factors. Clinicians should also be aware that creatine phosphokinase (CPK) and creatine kinase-MB (CK-MB) can be mildly elevated following a cesarean delivery and that troponin is a more specific marker of cardiac disease in the peripartum period. All forms of stress testing can be safely carried out in pregnancy, including nuclear imaging, although many centers prefer exercise echocardiography for this population. Diagnostic coronary angiography can and should be performed on pregnant women for the same indications as for nonpregnant patients.

Treatment of coronary artery disease remains largely unchanged in pregnancy. None of the medications commonly used to treat ischemic heart disease have been shown to cause adverse effects in the fetus. There is broad experience with low-dose aspirin, nitrates, β-blockers, and heparins in pregnancy. The paucity of data regarding the use of clopidogrel and the platelet glycoprotein IIb/IIIa inhibitors should limit their use in pregnancy to clinical scenarios with proven benefits. Statins
Antidysrhythmic agents for which we have the most pregnancy
were previously considered to be teratogenic but a recent sys-
tematic review suggests that the doses which are commonly
prescribed do not in fact increase any adverse fetal outcomes
(110). Coronary angiography, angioplasty and stenting, and
thrombolysis have been and can be carried out safely through-
out pregnancy (111–114).

The management of laboring patients with ischemic heart
disease should be the same for other cardiac patients as dis-
cussed in the section above on general principles of manage-
ment of cardiac disease at the time of delivery, and has strong
parallels with the management of the cardiac patient undergoing
general surgery.

**Cardiac Dysrhythmias in Pregnancy**

Dysrhythmias during gestation, and especially labor and
delivery, appear to be more common than in the nonpregnant
population (115). Hormonal changes, stress, and anxiety are
contributing factors; however, most dysrhythmias are not seri-
ous unless they are associated with organic heart disease.

**Atrial Fibrillation**

Atrial fibrillation occurring in pregnancy is usually associated
with underlying disease such as MS, peripartum cardiomyo-
pathy, hypertensive heart disease, thyroid disease, or ASDs.
Patients with acute atrial fibrillation and significant hemody-
namic changes—such as hypotension or loss of consciousness—
require direct current cardioversion. Cardioversion appears to
have no adverse effects on the fetus. Most patients, however,
will require only medical management with rate-controlling or
rhythm-restoring antidysrhythmics. β-Adrenergic blockers such
as metoprolol, calcium channel blockers such as diltiazem or
verapamil, and agents such as procainamide or digoxin can all be
used safely during pregnancy. Amiodarone would not be consid-
ered a first-line agent for hemodynamically stable atrial fibrilla-
tion because of its possible effects on the fetal thyroid, but its use
in pregnancy is not absolutely contraindicated and the risks of
potential use must be weighed against the potential benefits and
seriousness of the indication. Anticoagulation for atrial fibrilla-
tion in pregnancy has the same indications as in nonpregnant
patients, but the agent that must be used is heparin—usually in
the form of subcutaneous LMWH—because warfarin is associ-
ated with adverse fetal effects throughout gestation.

**Supraventricular Tachycardia**

SVTs during pregnancy can occur with or without organic
heart disease. Four percent of women with SVT report that
their condition was first identified in pregnancy, and up to 22%
state that pregnancy exacerbated their condition (116). In the
absence of underlying cardiac disease, these tachycardias are
not usually associated with increased morbidity. However, in
patients with underlying structural cardiac disease or cardio-
myopathy, SVT can lead to heart failure and death. Treatment
protocols for SVT remain unchanged in pregnancy and include
carotid sinus massage, adenosine, calcium channel blockers,
β-blockers, and direct current cardioversion (117,118).

**Ventricular Dysrhythmias**

Ventricular dysrhythmia during pregnancy may be associ-
ated with cocaine use, peripartum or any other form of
cardiomyopathy, ischemic heart disease, and digitalis toxicity.
Antidysrhythmic agents for which we have the most pregnancy
data are lidocaine, β-blockers, and procainamide. Amiodarone
is associated with an increased risk of fetal thyroid disease
and, although its use in pregnancy is permissible, it should
not be considered a first-line agent. Implantable defibrillators
can and should be used when indicated in pregnancy, although
they will need to be turned off during surgical procedures that
require the use of cautery.

**Bradycardia**

Bradysyndrhythmias during pregnancy are rare and may result
from infection such as Lyme disease (which can cause heart
blocks of varying degrees), hypothyroidism, myocarditis,
drug-induced, or congenital or acquired heart blocks. Perman-
ent pacemakers are indicated for hemodynamically signifi-
cant bradycardia. Patients with pre-existing pacemakers may
need to have their baseline rate increased during pregnancy to
mimic the normal physiologic changes of pregnancy.

**Antidysrhythmic Drugs**

Table 78.7 classifies the commonly used antidysrhythmic
agents on the basis of what is known about their safety in
pregnancy. Although there are obviously agents that we know
more about than others, it is important to reemphasize here
that both mother and fetus benefit from the use of the best
agent to control cardiac symptoms in pregnancy, and treat-
ment should never be withheld from a pregnant woman based
on theoretic fears of fetal harm.

**Cardiac Surgery During Pregnancy**

As in other semielective nonobstetric surgery during pregnancy,
if nonurgent cardiac surgery is necessary, it should ideally take
place during the second trimester. Deferring when possible
until after the first trimester avoids the period of organogen-
esis and the risk of miscarriage. Third-trimester surgery carries
the risk of precipitating preterm labor. However, surgery that is
important to a patient’s short-term well-being and survival
should be done at any point in gestation as required. Coronary
tree bypass grafts, valvuloplasties, valvular replacements,
and aortic root replacements have all been done in pregnancy
with good outcomes for mother and baby. When medical man-
agement can ameliorate the disease process, surgery may be
postponed until the patient has recovered at least 4 to 6 weeks
postpartum; however, such decisions should be based on
the best plan of action for the mother’s safety rather than a
cultural discomfort related to performing surgery in pregnancy.

Special intraoperative considerations in pregnant patients
include fetal monitoring during and after surgery, maintenance
of high flow and systemic mean arterial pressure (during car-
diopulmonary bypass), and uterine displacement devices if
the patient is in the supine position for a median sternotomy.
Although the pregnant patient has fared well with open heart
procedures, fetal mortality rate can be high. Generally, better
results are seen in closed heart procedures. Postoperatively,
fetal monitoring should be continued and maternal analgesia
maintained to avoid precipitating labor from accelerated post-
operative pain.

**Pregnancy After Prosthetic Valve Surgery**

Patients with mechanical heart valve prostheses pose a sig-
ificantly increased risk for thromboembolic events during
Cardiac Transplant Patients

With the increasing number and survival of heart transplant recipients, increasing numbers of women who have undergone this procedure have become pregnant (122,123). The pregnancy experience with solid tissue transplant patients in general has found a 25% risk of maternal complications (with over half of these complications being hypertension), a 29% risk of miscarriage, and a 41% risk of prematurity (123). The best data specific to cardiac transplantation describe 32 US pregnancies in women who had undergone cardiac transplantation, and found a 44% rate of hypertension, a 22% risk of rejection, and a 13% risk of worsening renal function. Neonatal complications were similar to the data described above for all solid tissue transplants (124). In light of these data, women who have undergone cardiac transplantation are warned of the possible risks of a pregnancy, and are encouraged to wait 2 years after transplantation before becoming pregnant to avoid both fetal and maternal bleeding associated with delivery. Other experts would use LMWH but carry out frequent testing of the peak and trough heparin levels—anti-Xa levels—to ensure that the patient is adequately anticoagulated.

Cardiopulmonary Resuscitation

Pregnancy poses some unique problems during cardiopulmonary resuscitation (CPR). In the third trimester and particularly near term, the gravid uterus impairs venous return. Thus, during CPR, the uterus should be displaced (i.e., left uterine tilt). Moreover, if defibrillation is required, the left breast needs to be displaced because of marked enlargement during pregnancy. The unlikely but theoretical possibility that there may be electrical arcing between a defibrillator and any fetal monitoring devices means that fetal monitoring devices should be removed prior to defibrillation. Otherwise, the Advanced Cardiac Life Support (ACLS) protocols, including medications and the use of the defibrillator, should be followed as done in a nonpregnant patient. Some experts would suggest that the use of amiodarone should be deferred in cardiac resuscitation until alternative appropriate agents have failed. However, in the context of a cardiac arrest, the authors would support the use of any recommended ACLS medication, as the one-time use of any of these agents is very unlikely to be of any harm, and may be of great benefit to both mother and fetus.

Data about the risk and benefits of an emergency cesarean delivery in the context of maternal resuscitation are very limited. The present-day view is that if the fetus has reached a point in the pregnancy where survival after delivery is possible (typically more than 24 weeks of gestation), emergency cesarean should be considered a part of the resuscitative efforts. Evacuation of the gravid uterus, with the concomitant release of pressure on the inferior vena cava and removal of the low-resistance circulatory unit that is the placenta, may improve the efficacy of chest compressions and improve the outcome for both mother and baby. Present recommendations are for consideration of cesarean delivery in pregnant women greater than 24 of weeks gestation who have had a cardiac arrest and failed to respond to 4 minutes of aggressive and appropriate resuscitative efforts (125). Consensus statements have been published by both the American Heart Association (126) and Society of Obstetric Anesthesia and Perinatology (127) on this topic.

Key Points

- Preeclampsia complicates 5% to 8% of all pregnancies, can occur at any time in the second half of pregnancy and the postpartum period, and is related to placental insufficiency.
- The following are all life-threatening maternal complications of preeclampsia: severe hypertension, eclamptic seizures, cerebral hemorrhage, pulmonary edema, disseminated intravascular coagulation, acute renal failure, hepatic failure and/or rupture, and diabetes insipidus.
- A particularly severe form of preeclampsia is the HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet counts).
- Magnesium sulfate is the drug of choice to prevent eclamptic seizures, but careful monitoring for toxicity (hypotension, muscular weakness, respiratory depression) is important particularly in patients with worsening renal dysfunction.
- Cardiac disease during pregnancy has an incidence rate of 0.4% to 4%, and is now the leading cause of maternal mortality in the developed world.
- Factors associated with an increased risk of cardiac complications in pregnancy are: premorbid New York Heart Association (NYHA) functional class III or IV status, cyanosis, presence of pulmonary hypertension, left-sided obstructive cardiac lesions, left ventricular systolic dysfunction, prior cardiac events and/or previous arrhythmias.
- Three periods of particular risks are: end of second trimester, time of labor and delivery, immediate postpartum period (first 72 hours).
Among the more commonly used cardiac medications, only angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and warfarin are absolutely contraindicated in pregnancy. The radiation exposure associated with all plain film radiographs, CT scans, nuclear scans, and angiography are all below what is deemed acceptable during pregnancy. None of the contrast agents used have been associated with any fetal complications.

For most cardiac patients, it is recommended that a multidisciplinary patient care conference be assembled well in advance of the anticipated time of delivery. The need for cesarean deliveries is generally dictated by obstetric concerns, and vaginal deliveries should generally be viewed as the safest and best options for cardiac patients.

For either mode of delivery, early establishment of regional anesthesia is recommended to reduce the undesired effects of labor on heart rate (tachycardia) and blood pressure (increased systemic vascular resistance). PPCI is defined as the new onset of systolic dysfunction occurring in the absence of other plausible causes anytime between the final month of pregnancy up to 5 months postpartum.

Emergency cesarean delivery should be strongly considered in pregnant women beyond 24 weeks of gestation who have had a cardiac arrest and failed to respond to 4 minutes of aggressive and appropriate resuscitative efforts.
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