Gestational Hypertension, Preeclampsia, and Peripartum Cardiomyopathy: A Clinical Review

An evidence-based guide to these major pregnancy-specific cardiovascular diseases.

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ABSTRACT

Gestational hypertension, preeclampsia, and peripartum cardiomyopathy are among the most common and often severe pregnancy-specific cardiovascular diseases (CVDs) and causes of complications in pregnancy. This clinical review provides nurses with an overview of pregnancy-specific CVDs, outlines their pathophysiology, and discusses risk factors and assessment. It describes management interventions according to timing: the antepartum, intrapartum, and postpartum phases are each addressed.

Keywords: breastfeeding; fetus; gestational hypertension; hypertension; preeclampsia; pregnancy complications, cardiovascular; peripartum cardiomyopathy; pregnant women; prenatal care

This is the accepted version of a published article in the American Journal of Nursing. The reference to the published article is:

Cardiovascular diseases (CVDs) constitute a leading cause of maternal and fetal mortality in pregnant women. (Umesawa et Kobashi, 2017) A large subset of these diseases is nonspecific to pregnancy (for example, ischemic and congenital heart disease, cardiac valvulopathies, and chronic hypertension) and proper management should ideally start before conception. (Adam, 2017) A smaller subset is composed of pregnancy-specific CVDs that appear only during the peripartum period. Gestational hypertension, preeclampsia, and peripartum cardiomyopathy are among the most common of these, as well as causes of complications during pregnancy. (Bello et al., 2013; Sibai, 2003; Verklan et Walden, 2015) To limit possible adverse maternal and fetal outcomes, timely recognition and management are essential. (American College of Obstetrics and Gynecologists, 2019)

In a 2018 joint statement, the World Health Organization and several other organizations asserted that all women should have access during pregnancy to a competent health care professional able to identify and manage related complications. (World Health Organization (WHO), 2018) Yet recent population studies found that inadequate peripartum follow-up—such as failure to evaluate new symptoms, reevaluate existing symptoms, or respond to changes without delay—were responsible for between one-quarter and two-thirds of deaths associated with pregnancy-specific CVDs. (Hameed et al., 2015; Hernandez et al., 2018) Nurses clearly have a vital role to play in efforts aimed at the prevention, assessment, and management of pregnancy-specific CVDs.

Previous reviews targeting pregnancy-specific CVDs have been conducted mainly in the medical field, focusing on underlying risk factors and pharmacotherapy. (Anthony et al., 2016; Cairns et al., 2017; Gillon et al., 2014) One recent review published in this journal focused on the nursing approach to managing preeclampsia. (Anderson et Schmella, 2017) But to our knowledge, no review has specifically examined the role of nurses regarding pregnancy-specific CVDs in relation to maternal and fetal health.

We decided to conduct a clinical review of the literature in order to learn more. To that end, we searched the Cumulative Index of Nursing and Allied Health Literature (CINAHL), PubMed, and Google Scholar with the terms “gestational hypertension,” “preeclampsia,” and “peripartum cardiomyopathy” in order to find systematic reviews and primary research articles.
pertinent to our aim. We also searched for the latest clinical practice guidelines from the major national obstetrical and cardiovascular societies.

This review provides an overview of three pregnancy-specific CVDs—gestational hypertension, preeclampsia, and peripartum cardiomyopathy—and synthesizes the relevant information regarding the role of nurses in their prevention, assessment, and management.

NORMAL CARDIOVASCULAR CHANGES DURING PREGNANCY
An understanding of the normal physiological changes that occur in the maternal cardiovascular system during pregnancy allows for better comprehension of the pathophysiologic changes that occur with pregnancy-specific CVDs. This will also help nurses to distinguish between normal changes and those that may indicate a pregnancy-specific CVD.

Cardiovascular changes during pregnancy serve to ensure proper fetal blood flow. During a pregnancy, the heart will gradually be geometrically and mechanically remodeled to accommodate an increase in circulatory volume load. (Melchiorre et al., 2016) Such remodeling includes, for example, an increase in the volume and mass of the atria and ventricles. As maternal body surface area increases, cardiac output will increase significantly throughout the pregnancy. (Melchiorre et al., 2016) One study found that in singleton pregnancies, cardiac outputs increased by as much as 45% above nonpregnant levels. (Hunter et Robson, 1992) The same study also found that in twin pregnancies, the average cardiac output was 15% higher than it was in singleton pregnancies.

Although such changes are normal, tolerance for physical exertion is generally lower in pregnant than in nonpregnant women, and pregnant women may experience shortness of breath and fatigue while performing even light physical activities. (Sanghavi et Rutherford, 2014) At rest, maternal heart rate and blood pressure should remain in normal range: heart rate less than 100 beats per minute (bpm), blood pressure less than 140/90 mmHg. (It’s worth noting that blood pressure often tends to drop slightly during pregnancy, most notably during the first trimester. (Cheung et Lafayette, 2013; Melchiorre et al., 2016)) In adult women, the left ventricular ejection fraction (the percentage of blood leaving the left ventricle at the end of each contraction) ranges from 54% to 74%, and should not go below the lower value even in pregnancy. (Lang et al.,
From the first to the third trimester, activation of the renin-angiotensin-aldosterone system causes increased retention of salt and water, which leads to a rise in blood volume, venous return, and cardiac preload. (Cheung et Lafayette, 2013) Thus edema is relatively common in pregnant women.

PREVALENCE AND PATHOPHYSIOLOGY OF COMMON PREGNANCY-SPECIFIC CVDs

Gestational hypertension is one of the most common problems in pregnant women, with prevalence ranging from 1.8% to 4.4% worldwide. (Umesawa et Kobashi, 2017) Gestational hypertension is diagnosed after 20 weeks of gestation in women with an average blood pressure of 140/90 mmHg or higher, without any of the supplementary features of preeclampsia (described below). (Butalia et al., 2018) For diagnostic purposes, blood pressure should be measured in a clinical setting twice with at least four hours between measurements, using the arm with the highest values. (Butalia et al., 2018; Pfaff, 2014) Although most women with gestational hypertension will not suffer any complications, (American College of Obstetrics and Gynecologists, 2019) the condition has been associated with higher risk for developing diabetes and kidney disease in later life. (Kintiraki et al., 2015) Gestational hypertension severity is a predictor of worse outcomes. A blood pressure higher than 160/110 mmHg is considered a criterion of increased severity. (American College of Obstetrics and Gynecologists, 2019)

Preeclampsia and HELLP syndrome. Preeclampsia is another common problem, occurring in 5% to 8% of pregnant women overall and in 17% to 46% of those with gestational hypertension. (Melamed et al., 2014) Preeclampsia can be diagnosed after 20 weeks of gestation when there is new-onset hypertension with either proteinuria or indications of target organ dysfunction, including pulmonary edema; cerebrovascular disturbances (including visual disturbances like flashing lights, blurred vision); or signs of kidney failure (such as low urine output, electrolyte imbalance). Proteinuria is diagnosed when the protein-to-creatinine ratio of two urine samples taken at least six hours apart exceeds 0.3 mg/dL (American College of Obstetricians and Gynecologists, 2013; Pfaff, 2014) or when the protein concentration of a 24-hour urine excretion sample equals or exceeds 300 mg (American College of Obstetricians and Gynecologists, 2015).
If not managed, preeclampsia can progress to eclampsia, as defined by the onset of seizures. Eclampsia can be lethal; mortality rates are estimated at up to 1.8% in developed countries and up to 15% in developing countries. (Ghulmiyyah et Sibai, 2012) Although the cause of preeclampsia remains unclear, most theories cite a combination of immunologic factors and oxidative stress, leading to placental dysfunction. (Verklan et Walden, 2015) The latter leads to the release of certain antiangiogenic factors to the maternal blood flow, which cause endothelial damage and abnormal vascular remodeling. (Townsend et Drummond, 2011)

For pregnant women, preeclampsia significantly increases the risk of cardiopulmonary failure and cerebrovascular accident later in life. (Preeclampsia Foundation, 2019) It’s also associated with a cluster of symptoms known as HELLP syndrome (the acronym stands for hemolysis, elevated liver enzymes, and low platelet count). (Verklan et Walden, 2015) One review found that in 70% to 80% of cases of preeclampsia, HELLP syndrome was also present. (Abildgaard et Heimdal, 2013)

Outcomes are worse in cases of early-onset or severe preeclampsia. For example, a Norwegian study found that among women with preeclampsia, the risk of stillbirth overall was about 0.5% but was substantially higher with early-onset preeclampsia. (Harmon et al., 2015)

**Peripartum cardiomyopathy** has been diagnosed in up to 37% of women with gestational hypertension or preeclampsia. (Bello et al., 2013) But the links between these disorders have yet to be clarified. Peripartum cardiomyopathy was once thought to be a silent underlying dilated cardiomyopathy (a condition in which the left ventricle is stretched), but it’s now recognized as a distinct idiopathic cardiomyopathy that can manifest between the last month of pregnancy through the fifth month postpartum. (European Society of Gynecology (ESG) et al., 2011; Sliwa et al., 2010) Diagnosis is by exclusion: a left ventricle ejection fraction (the percentage of blood exiting with each contraction) of less than 45% has to be present on echocardiography, with this finding unexplained by another underlying heart disease. (Hibbard et al., 1999)

Multiple factors appear to be associated with peripartum cardiomyopathy, and its evolution varies among individuals. (Sharma et Kumar, 2017) The presence of certain genetic variants, excessive oxidative stress, fetal microchimerism (migration of a few fetal cells to the mother’s myocardium, prompting an autoimmune response), and the abnormal metabolism of prolactin (a
hormone involved in breast milk production) are all suggested factors in its development. (Hilfiker-Kleiner et al., 2015; Ware et al., 2016) Many women with peripartum cardiomyopathy regain cardiac function: one study found that at one year post-delivery, 60% showed full recovery and 31% showed partial recovery. (Abou Moulig et al., 2018) Maternal and fetal outcomes are generally positive during future pregnancies. (Codsi et al., 2018) That said, about one-third of women who have had peripartum cardiomyopathy experience relapse in subsequent pregnancies. (Elkayam, 2014) At two years postpartum, maternal mortality ranges from 0% to 9%, with higher rates seen in women of African descent. (Sliwa et al., 2018) Outcomes are generally better when the level of maternal heart failure at time of diagnosis is classified as class I or II (little or no impact on physical activity) rather than class III or IV (marked or severe impact) per the New York Heart Association (NYHA) Functional Classification system. (American Heart Association (AHA), 2017; Sliwa et al., 2006) For details about this system, visit http://bit.ly/30Gy1cp.

**RISK FACTORS AND CLINICAL ASSESSMENT**

Informing women considering pregnancy of the risk for pregnancy-specific CVDs will help them to make an informed decision. Women at higher risk include those who are older than age 30 years, (Arany, 2018) are overweight or obese, (Patel et al., 2017; Shen et al., 2017) have a preexisting metabolic or cardiovascular condition such as diabetes or hypertension, (Patel et al., 2017; Shen et al., 2017) had a previous pregnancy complicated by a pregnancy-specific CVD, or have a family history of pregnancy-specific CVDs (Sliwa et al., 2017). Furthermore, having a lower educational level is associated with higher risk for gestational hypertension and preeclampsia, (Umesawa et Kobashi, 2017) and being of African descent is associated with higher risk for peripartum cardiomyopathy. (Asad et al., 2018)

Lifestyle behaviors that promote healthy pregnancy include eating a well-balanced diet (for example, by following the dietary recommendations of the American College of Obstetricians and Gynecologists (American College of Obstetricians and Gynecologists, 2018)), engaging regularly in low- to moderate-intensity physical activity, and refraining from drinking alcohol and smoking. (Davenport et al., 2018; Gupta et Wenger, 2018) The adoption of these behaviors can help to improve metabolic outcomes and prevent other pregnancy disorders, such as gestational
diabetes. (Gilbert et al., 2019) That said, such behavioral adoptions have not been specifically associated with reduced risks for pregnancy-specific CVDs. (Syngelaki et al., 2018) Research to develop interventions aimed at preventing pregnancy-specific CVDs is ongoing. (Ohkuchi et al., 2017)

Recognizing pregnancy-specific CVDs can often be a complex endeavor for nurses delivering peripartum care, as some of the clinical manifestations, such as dyspnea, edema, and excessive fatigue, can be confused with signs and symptoms of normal pregnancy. Noticing the onset of new signs and symptoms or the progression of existing ones are both vital to timely recognition of pregnancy-specific CVDs. For a synopsis of key diagnostic criteria for the pregnancy-specific CVDs discussed in this article, see Table 1 (Abildgaard et Heimdal, 2013; Butalia et al., 2018; Hibbard et al., 1999; Pfaff, 2014; Sibai, 2003).

**Gestational hypertension.** Elevated blood pressure will often be the only visible sign at clinical assessment, with no further symptoms. (Surányi et al., 2017) Women with severe hypertension (160/110 mmHg or greater) should be admitted to a hospital for further assessment and proper management until the blood pressure falls below that threshold. (American College of Obstetrics and Gynecologists, 2019)

**Preeclampsia and HELLP syndrome.** Pregnant women with elevated blood pressure should also be assessed for preeclampsia, which has a different course and prognosis, and to determine whether hypertension is severe, which affects management and outcomes. (American College of Obstetrics and Gynecologists, 2019) As noted earlier, the presence of proteinuria or systemic organ dysfunction (or both) are defining features of preeclampsia. Women with elevated blood pressure should be evaluated for signs and symptoms of nervous system disorders such as hyperreflexia, clonus, tremor, headaches, paresthesia, and visual disturbances, as well as cardiovascular signs and symptoms such as oxygen saturation under 97%. Early signs of kidney disorders are detectable by laboratory testing and include decreased glomerular filtration rate and increased urinary albumin excretion rate. (Wouters et al., 2015) Screening for proteinuria can be done at each visit using urine dipsticks. (Magee et al., 2014) Nonspecific symptoms of kidney disorders include fatigue, nausea, dyspnea, peripheral pitting edema, and oliguria. (Magee et al., 2014; Moore et al., 2018) Symptoms of liver disorders include nausea, epigastric pain at the upper
right quadrant, and shoulder pain. (Verklan et Walden, 2015) A complete blood count and kidney and liver function tests are useful in assessing systemic organ dysfunctions. (American College of Obstetrics and Gynecologists, 2019)

Patients with HELLP syndrome often present with nonspecific symptoms that overlap with those found in preeclampsia. The cluster of symptoms seen in HELLP syndrome—hemolysis, elevated liver enzymes, and low platelet count (thrombocytopenia)—will also be present. Hemolysis is considered the “hallmark of the triad.” (Sibai, 2004) Symptoms of thrombocytopenia include ecchymoses, hematuria, and bleeding from areas rich in vessels (for example, epistaxis).

Peripartum cardiomyopathy often manifests not only with physical symptoms such as dyspnea, edema, and excessive fatigue, but also with emotional symptoms such as anxiety, panic, and helplessness. (Patel et al., 2016) Delays in diagnosis can exacerbate such feelings. Early recognition is essential to ease the woman’s emotional pain as well as lower the risk for further complications.

Signs and symptoms of heart failure consistent with volume overload and systemic hypoperfusion can be found in women with peripartum cardiomyopathy. Volume overload in the lungs can result in dyspnea during ordinary daily activities, orthopnea, persistent nocturnal dry cough, and paroxysmal nocturnal dyspnea. (Moioli et al., 2010) Volume overload may also lead to peripheral pitting edema. (Sharma et Kumar, 2017) Enlargement of the atria and ventricles may lead to development of ectopic foci and thus cardiac arrhythmia. (Andrade et al., 2014) At clinical assessment, cardiac auscultation may reveal new-onset murmurs, indicating a mitral or tricuspid regurgitation, and elevated jugular venous pressure. (Sharma et Kumar, 2017) Depending on the degree to which peripartum cardiomyopathy has progressed, different symptoms of heart failure at varying levels of severity may be present. (Elkayam, 2011) The patient may either have a normal heart rate or be tachycardic; and arterial hyper- or hypotension may also be found. (Elkayam, 2011) One review found that, at the time of diagnosis, about 75% of pregnant women with peripartum cardiomyopathy had heart failure symptoms corresponding to classes III or IV of the NYHA Functional Classification system. (Asad et al., 2018) That is, symptoms such as dyspnea, fatigue, and palpitations either markedly impeded daily activities (class III) or severely impeded daily activities, with discomfort even at rest (class IV). (American Heart Association (AHA), 2017)
Fetal assessment. If the initial maternal assessment for pregnancy-specific CVDs is negative, but there are ongoing medical concerns about fetal health, fetal monitoring is recommended. (Magee et al., 2014) Performing a fetal ultrasound can permit identification of an abnormal fetal heart rate (under 120 bpm or over 160 bpm), oligohydramnios (insufficient amniotic fluid), and intrauterine growth restriction (delayed fetal growth). If evidence of fetal stress is found, antenatal testing is suggested using umbilical artery Doppler velocimetry. (Kirkpatrick et al., 2017) This test examines the direction and impedance of umbilical arterial blood flow. Pregnancy-specific CVDs have been linked to placental abnormalities (such as inflammation, infarct, thrombosis), which can increase placental vascular resistance and impair blood perfusion. The absence or reversal of end-diastolic flow in the umbilical arteries can be a further indication of fetal stress. (Magee et al., 2014)

MANAGEMENT
Primary care providers should discuss with their pregnant patients the risks and benefits of pharmacotherapy for pregnancy-specific CVDs, as well as the potential impact of untreated illness, in order to determine the safest and most appropriate approach. In collaboration with other interdisciplinary team members, nurses should educate their pregnant patients about any medications that are then prescribed. Moreover, aerobic exercise is “absolutely contraindicated” in pregnant women with pregnancy-induced hypertension, preeclampsia, HELLP, and hemodynamically significant heart disease. (Kirkpatrick et al., 2017; Pfaff, 2014) and nurses should counsel patients accordingly. The timing and mode of delivery should be based on the severity of hypertension and the stability of the maternal–fetal condition. (Kirkpatrick et al., 2017) As in any pregnancy, decisions about delivery should be made collaboratively by the pregnant woman, her family members, and the health care team.

Here we address clinical management for each pregnancy-specific CVD, with consideration for when to initiate: during the antepartum, the intrapartum, or, if applicable, the postpartum phase. For a synthesis of indications for frequently used medications in these patients during pregnancy and breastfeeding, see Table 2 (Alabdulrazzaq et Koren, 2012; American College of Obstetricians and Gynecologists, 2013; Bauersachs et al., 2016; Bouabdallaoui et al., 2017;
Butalia et al., 2018; Gupta et Wenger, 2018; Magee et al., 2014; National Library of Medicine, 2018; Schlembach et al., 2015).

**Gestational hypertension. Antepartum phase.** First-line pharmacotherapy typically involves the administration of methyldopa (Aldomet), a centrally acting antiadrenergic, or labetalol (Trandate), a dual alpha- and nonselective beta-blocker.(Al Khaja et al., 2014) Both lower blood pressure mainly through vasodilation. Although antihypertensive medication can be initiated if the blood pressure is over 150/100 mmHg, for pregnant women this isn’t usually recommended unless the blood pressure is consistently over 160/110 mmHg.(American College of Obstetricians and Gynecologists, 2013; Kirkpatrick et al., 2017) Prolonged or severe hypertension can lead to central nervous system injury.(Kirkpatrick et al., 2017)

Once pharmacotherapy begins, maternal blood pressure must be closely monitored to evaluate treatment effectiveness and to avoid hypoperfusion; a diastolic blood pressure of 85 mmHg should be the target.(Butalia et al., 2018) Nurses can suggest home self-monitoring with an automated blood pressure device and explain its use.(Uhlig et al., 2013) For best results, patients should refrain from exercising at least 30 minutes before taking a reading and should sit up straight with the legs uncrossed and the feet flat; the upper arm should be unclothed and held at heart level. Blood pressure should be measured at the same time daily. The correct cuff size matters—it should be 1.5 times arm circumference—and nurses or pharmacists can help women to determine the right size. Partial bed rest may be recommended for women with mild hypertension (between 140/90 and 159/109 mmHg). Strict bed rest is not advised because of the increased risk for thromboembolism.(Spiro et Scemons, 2018)

**Intrapartum phase.** During this phase, positioning the woman in a left lateral or sitting position may help lower cardiovascular stress by avoiding aortocaval compression and reduced venous return.(Ersbøll et al., 2016) Vaginal birth is preferred when the woman is stable and there are no obstetric indications for a cesarean.(Bouabdallaoui et al., 2017) Advantages of vaginal birth include less blood loss, better hemodynamic stability, lack of surgery-related stress and anxiety, and fewer pulmonary complications.(Ray et al., 2004)

**Postpartum phase.** During this phase, it’s recommended that maternal blood pressure be assessed at least once at three days to 10 days postpartum.(American College of Obstetrics and
Gynecologists, 2018) As for breastfeeding and drugs commonly used to treat either gestational hypertension or preeclampsia, there are usually no contraindications. (Magee et al., 2014) As with any new parents, nurses should provide standard information and recommendations regarding the benefits of breastfeeding, proper positioning and latching of the infant on the nipple, and common problems and ways to address them. (Folker-Maglaya et al., 2018)

**Preeclampsia and HELLP syndrome.** Management for preeclampsia and HELLP syndrome includes all the recommendations described above for gestational hypertension, as well as the following.

*Antepartum phase.* During this phase, management is focused on preventing the onset of seizures. In some cases, magnesium sulfate might be administered intravenously or intramuscularly to help prevent seizures. After such administration, it’s important to monitor for signs of magnesium toxicity, which include bradycardia, bradypnea, oliguria, and altered states of consciousness (such as confusion, anxiety). (Pfaff, 2014; Witcher et al., 2015)

*Intrapartum phase.* In women with mild preeclampsia without signs of clinical instability or indicators for preterm delivery, full-term delivery may be considered. In women with severe preeclampsia or with signs of maternal or fetal instability, delivery is recommended as soon as the maternal condition is stabilized. (Kirkpatrick et al., 2017)

**Peripartum cardiomyopathy.** *Antepartum phase.* To our knowledge, there have been no clinical trials specifically evaluating the management of heart failure in peripartum cardiomyopathy. Thus, during the antepartum phase, standard management of heart failure is warranted. This can include the cautious administration of diuretics, beta blockers, hydralazine, nitrates, and heparin. (Jackson et al., 2018) Managing volume status is essential. (Arany, 2018) As such, salt and fluid intake restriction are necessary to prevent volume overload. (Bouabdallaoui et al., 2017) Light physical activity may still be encouraged in women with peripartum cardiomyopathy. (American College of Obstetrics and Gynecologists, 2015; Jain et al., 2016)

Women with a severely impaired ejection fraction (below 25%) despite treatment or who are in cardiogenic shock and receiving IV positive inotropes (such as dobutamine) may require mechanical support. (Loyaga-Rendon et al., 2014) Left ventricular assist devices (a surgically implanted pump that assists the heart in pumping blood) are one such type of support. In women
with peripartum cardiomyopathy, these devices are often installed to support those who are waiting for a heart transplant.(Loyaga-Rendon et al., 2014)

**Intrapartum phase.** Early delivery is not indicated as long as the maternal–fetal condition is stable.(Ersbøll et al., 2016; Regitz-Zagrosek et al., 2018; Sliwa et al., 2010) In cases of maternal instability requiring the use of inotropes or mechanical support, fetal delivery by planned cesarean may reduce the hemodynamic stress. In cases of less severe maternal instability, regional anesthesia and assisted vaginal delivery are preferred. Pain control during delivery is essential. Regional anesthesia (such as epidural analgesia or continuous spinal anesthesia) can improve cardiac loading and stabilize cardiac output by reducing preload and afterload.(Langesæter et Dyer, 2011) Anesthesia also reduces anxiety and lowers sympathetic nervous system stress, which further benefits cardiovascular function.

Administration of IV fluids in this population requires close monitoring to avoid overhydration and rapid preloading. With epidural anesthesia, some women may require IV fluids before the anesthesia is delivered.(Arendt, 2019) Although crystalloid fluids 500 cc is a typical dose,(Lindstrom et al., 2018) the potential benefits of this practice must be weighed against the risks for volume overload, hypoperfusion, and pulmonary edema.(Caughey et al., 2018) A vasopressor agent can also be added as needed.(Arendt, 2019)

**Postpartum phase.** There is a lack of consensus as to whether women with peripartum cardiomyopathy can safely breastfeed. Some experts have advised against it, theorizing that prolactin production could potentially exacerbate the condition.(Hilfiker-Kleiner et al., 2017; Johnson-Coyle et al., 2012; Safirstein et al., 2012) But recent literature reviews report that, unless there are pharmacologic contraindications, women with peripartum cardiomyopathy who are clinically stable should not be discouraged from breastfeeding.(Ersbøll et al., 2016; Sharma et Kumar, 2017)

With regard to future pregnancies, the left ventricular ejection fraction should be checked before attempting to conceive. Experts agree that women with an incompletely recovered ejection fraction should refrain from becoming pregnant again.(Bozkurt et al., 2016; Hess et Weinland, 2012; Sharma et Kumar, 2017) In such cases, nurses should offer counseling on contraceptive methods to reduce the odds of unplanned pregnancy.(Sharma et Kumar, 2017) Progesterone-only
forms of contraception are recommended, and emergency contraception should be considered if need be. (Curtis et al., 2016) Combined hormonal contraceptives are contraindicated.

**Fetal considerations.** Pregnancy-specific CVDs can contribute to intrauterine or neonatal mortality, (Magee et al., 2014) especially in low- and middle-income countries. (Saleem et al., 2014) Pregnancy-specific CVDs also increase the risks of undesirable outcomes such as preterm birth, low birth weight, and low APGAR scores (the acronym stands for appearance, pulse, grimace, activity, respiration). (Ersbøll et al., 2016; Magee et al., 2016) The severity of the maternal condition does not directly predict fetal and neonatal outcomes. (Kirkpatrick et al., 2017) Lower gestational age and abnormal results from more than one type of fetal monitoring may be better indicators of fetal morbidity and mortality risks. (Harmon et al., 2015; Magee et al., 2014)

The delivery care plan should be made in collaboration with the parents and should address topics such as fetal prematurity, neonatal intensive care, and maternal postpartum posttraumatic stress disorder. (Butalia et al., 2018; Magee et al., 2014) All deliveries before 34 weeks of gestation should occur only in a clinical setting with the necessary maternal and neonatal intensive care resources. (Kirkpatrick et al., 2017)

Parents of infants born prematurely or with complications are likely to experience emotional shock, self-blame, sadness, and fear. (Yang et al., 2017) For such parents, being able to participate in the care of and to interact with their baby; focusing on positive aspects and improvements; receiving information about their child’s health and specific needs, as well as available resources; and receiving emotional support from nurses and other health care professionals are all vital to helping them cope.
Table 1. Synthesis of Diagnosis Criteria for Pregnancy-Specific Cardiovascular Diseases

<table>
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<tr>
<th></th>
<th>Gestational Hypertension (Butalia et al., 2018; Pfaff, 2014)</th>
<th>Preeclampsia (Abildgaard et Heimdal, 2013; Butalia et al., 2018; Pfaff, 2014)</th>
<th>HELLP Syndrome (Abildgaard et Heimdal, 2013; Marchand et al., 1980; Sibai, 2003)</th>
<th>Peripartum Cardiomyopathy (Hibbard et al., 1999)</th>
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<tr>
<td>Diagnostic criteria</td>
<td>SBP ≥ 140 mmHg or DBP ≥ 90 mmHg, as measured at two points in</td>
<td>SBP ≥ 140 mmHg or DBP ≥ 90 mmHg when measured at two points in</td>
<td>Hemolysis (serum haptoglobin ≤ 25 mg/dL) AND</td>
<td>LVEF &lt; 45%, not explained by another cardiac</td>
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<td>time at least four hours apart</td>
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<td>The protein-to-creatinine ratio of two urine samples exceeds 0.3</td>
<td>The protein concentration of a 24-hour urine excretion sample ≥ 300</td>
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<td>Low platelet count (&lt; 100,000 cells/µl)</td>
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Note: DBP, diastolic blood pressure; HELLP, hemolysis, elevated liver enzymes, low platelet count; LDH, lactate dehydrogenase; LVEF, left ventricular ejection fraction; SBP, systolic blood pressure
Table 2. Indications for Drugs Often Used In Managing Pregnancy-specific Cardiovascular Diseases

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Indications during Pregnancy</th>
<th>Indications regarding Breastfeeding</th>
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<tr>
<td><strong>Angiotensin-converting enzyme inhibitors (ACEIs)</strong> (such as benazepril, fosinopril)</td>
<td>Not recommended. (<a href="#">Bouabdallaoui et al., 2017</a>)</td>
<td>Generally acceptable for use. (<a href="#">Magee et al., 2014</a>) But certain ACEIs (such as captopril, enalapril) are preferred. (<a href="#">Bauersachs et al., 2016</a>)</td>
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<td><strong>Angiotensin receptor blockers</strong> (such as losartan, valsartan)</td>
<td>Not recommended. (<a href="#">Bouabdallaoui et al., 2017</a>)</td>
<td>Not recommended, as profound hypotension in the infant may result. (<a href="#">Gupta et Wenger, 2018</a>)</td>
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<td><strong>Beta-blockers</strong> (such as labetalol, metoprolol, propranolol)</td>
<td>Generally acceptable for use. (<a href="#">Bouabdallaoui et al., 2017</a>)</td>
<td>Generally acceptable for use. (<a href="#">Magee et al., 2014</a>)</td>
</tr>
<tr>
<td><strong>Calcium channel blockers</strong> (such as amlodipine, nifedipine)</td>
<td>Generally acceptable for use. (<a href="#">Alabdulrazzaq et al., 2018</a>)</td>
<td>Generally acceptable for use. (<a href="#">Magee et al., 2014</a>)</td>
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<tr>
<td><strong>Centrally-acting antiadrenergics</strong> (such as methyldopa, clonidine)</td>
<td>Generally acceptable for use. (<a href="#">Butalia et al., 2018</a>)</td>
<td>Methyldopa is generally acceptable for use. (<a href="#">American College of Obstetricians and Gynecologists, 2013; Butalia et al., 2018</a>) Clonidine is not recommended, as there are potential adverse effects for the infant. (<a href="#">National Library of Medicine, 2018</a>)</td>
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<td><strong>Diuretics</strong> (such as furosemide, hydrochlorothiazide)</td>
<td>Use cautiously in order to avoid compromising fetal perfusion. (<a href="#">Bouabdallaoui et al., 2017</a>)</td>
<td>Generally acceptable for use. May decrease milk production. (<a href="#">Schlembach et al., 2015</a>)</td>
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REFERENCES


