

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

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

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: cardiovascular disease, gestational hypertension, HELLP syndrome, hypertension, peripartum cardiomyopathy, preeclampsia, pregnancy, pregnancy complications, prenatal care

Cardiovascular diseases (CVDs) constitute a leading cause of maternal and fetal mortality in pregnant women.¹ 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.² 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.³⁻⁵ To limit possible adverse maternal and fetal outcomes, timely recognition and management are essential.⁶

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.⁷ 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—was responsible for between one-quarter and two-thirds of deaths associated with pregnancy-specific CVDs.^{8,9} 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.¹⁰⁻¹² One recent review published in this journal focused on the nursing approach to managing preeclampsia.¹³ 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 to learn more. To that end, we searched CINAHL, PubMed, and Google Scholar, using the terms *gestational hypertension*, *preeclampsia*, and

peripartum cardiomyopathy, 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 obstetric and cardiovascular societies.

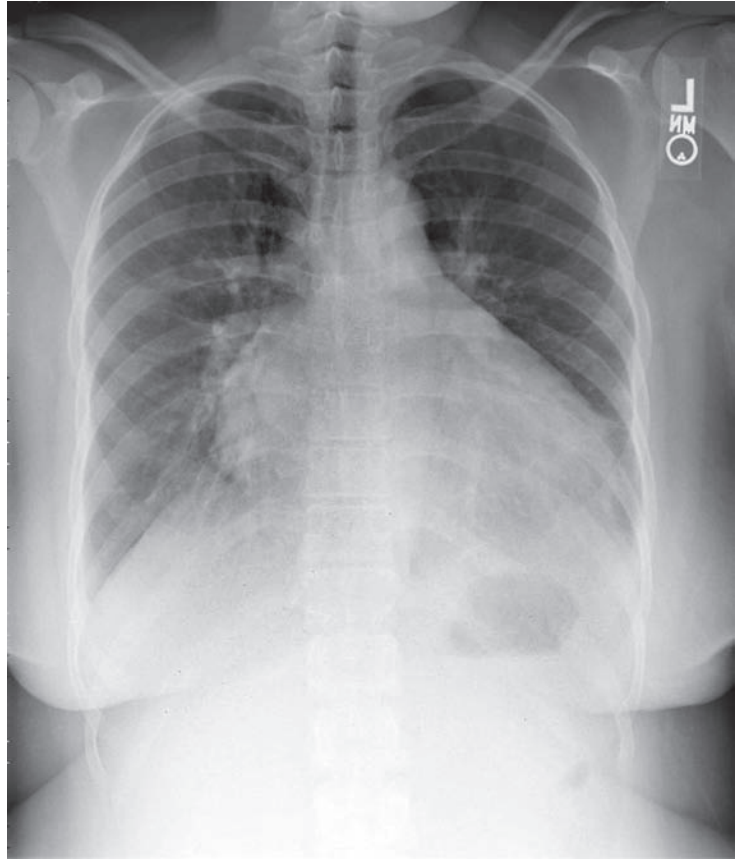
This review provides an overview of three pregnancy-specific CVDs—gestational hypertension, preeclampsia, and peripartum cardiomyopathy—and synthesizes the relevant information regarding the role of nurses in their prevention, assessment, and management.

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 pathophysiological 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.¹⁴ 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.¹⁴ One study found that in singleton pregnancies, cardiac outputs increased by as much as 45% above nonpregnant levels.¹⁵ 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.¹⁶ At rest, maternal heart rate and blood pressure should remain in the normal range: heart rate less than 100 beats per minute, 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.^{14, 17} In adult women, the left ventricular ejection fraction (the percentage of blood exiting the left ventricle with each contraction) ranges from 54% to 74%, and should not go below the lower value even in pregnancy.¹⁸ 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.¹⁷ Thus edema is relatively common in pregnant women.



An X-ray shows evidence of peripartum cardiomyopathy: the enlarged heart of a woman who presented to the ED five months postpartum. Copyright © 2007 Ibebuogu UN, et al.; licensee BioMed Central Ltd.

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.¹ 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).¹⁹ 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.^{19, 20} Although most women with gestational hypertension will not suffer any complications,⁶ the condition has been associated with higher risk of developing diabetes and kidney disease in later life.²¹ Gestational hypertension severity is a predictor of worse outcomes. A blood pressure higher than 160/110 mmHg is considered a criterion of increased severity.⁶

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.²² 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^{20,23} or when the protein concentration of a 24-hour urine excretion sample equals or exceeds 300 mg.^{19,23} 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.²⁴ Although the cause of preeclampsia remains unclear, most theories cite a combination of immunologic factors and oxidative stress, leading to placental dysfunction.⁵ The latter leads to the release of certain antiangiogenic factors into the maternal blood flow, which cause endothelial damage and abnormal vascular remodeling.²⁵

For pregnant women, preeclampsia significantly increases the risk of cardiopulmonary failure and cerebrovascular accident later in life.²⁶ It's also associated with a cluster of symptoms known as HELLP syndrome (characterized by hemolysis, elevated liver enzymes, and low platelet count).⁵ One review found that in 70% to 80% of cases of preeclampsia, HELLP syndrome was also present.²⁷

Outcomes are worse in cases of early-onset or severe preeclampsia. For example, a Norwegian study found that risk of stillbirth overall was about 0.5% among women with preeclampsia but was substantially higher among women with early-onset preeclampsia.²⁸

Peripartum cardiomyopathy has been diagnosed in up to 37% of women with gestational hypertension or preeclampsia.³ 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.^{29,30} Diagnosis is by exclusion: a left ventricle ejection fraction of less than 45% has to be present on echocardiography, with this finding unexplained by another underlying heart disease.³¹

Multiple factors appear to be associated with peripartum cardiomyopathy, and its evolution varies among individuals.³² 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.^{33,34} Many women with peripartum cardiomyopathy regain cardiac function: one study found that at one year after delivery, 60% showed full recovery and 31% showed partial recovery.³⁵ Maternal and fetal outcomes are generally positive during future pregnancies.³⁶ That said, about one-third of women who have had peripartum cardiomyopathy experience relapse in subsequent pregnancies.³⁷ At two years postpartum, maternal mortality ranges from 0% to 9%, with higher rates seen in women of African descent.³⁸ 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.^{39,40} For details about this system, visit www.heart.org/en/health-topics/heart-failure/what-is-heart-failure/classes-of-heart-failure.

RISK FACTORS AND CLINICAL ASSESSMENT

Informing women considering pregnancy of their risk of pregnancy-specific CVDs will help them to make an informed decision. Women at higher risk include those who are older than 30 years,⁴¹ are overweight or obese,^{42,43} have a preexisting metabolic or cardiovascular condition such as diabetes or hypertension,^{42,43} had a previous pregnancy complicated by a pregnancy-specific CVD, or have a family history of pregnancy-specific CVDs.⁴⁴ Furthermore, having a lower educational level is associated with higher risk of gestational hypertension and preeclampsia,¹ and being of African descent is associated with higher risk of peripartum cardiomyopathy.⁴⁵

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 Gynecologists⁴⁶), engaging regularly in low-to-moderate-intensity physical activity, and refraining from drinking alcohol and smoking.^{47,48} The adoption of these behaviors can help to improve metabolic outcomes and prevent other pregnancy disorders, such as gestational diabetes.⁴⁹ That said, such behavioral adoptions have not been specifically associated with reduced risks for pregnancy-specific CVDs.⁵⁰ Research to develop interventions aimed at preventing pregnancy-specific CVDs is ongoing.⁵¹

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

Table 1. Key Diagnostic Criteria for Pregnancy-Specific Cardiovascular Diseases

Gestational Hypertension ^{19,20}	Preeclampsia ^{19,20,27}	HELLP Syndrome ^{4,27,52}	Peripartum Cardiomyopathy ³¹
SBP \geq 140 mmHg or DBP \geq 90 mmHg, as measured at two points in time at least four hours apart	SBP \geq 140 mmHg or DBP \geq 90 mmHg, as measured at two points in time at least four hours apart AND Protein-to-creatinine ratio of two urine samples exceeds 0.3 mg/dL AND/OR Protein concentration of a 24-hour urine excretion sample \geq 300 mg	Hemolysis (serum haptoglobin \leq 25 mg/dL) AND Elevated liver enzymes (serum LDH $>$ 600 IU/L OR total bilirubin $>$ 1.2 mg/dL) AND Low platelet count ($<$ 100,000 cells/ μ L)	LVEF $<$ 45%, not explained by another cardiac disease

DBP = diastolic blood pressure; HELLP = hemolysis, elevated liver enzymes, low platelet count; LDH = lactate dehydrogenase; LVEF = left ventricular ejection fraction; SBP = systolic blood pressure.

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.^{4, 19, 20, 27, 31, 52}

Gestational hypertension. Elevated blood pressure will often be the only visible sign at clinical assessment, with no further symptoms.⁵³ Women with severe hypertension—160/110 mmHg or greater—should be admitted to a hospital for further assessment and proper management until blood pressure falls below that threshold.⁶

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.⁶ 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 an oxygen saturation of less than 97%. Early signs of kidney disorders are detectable by laboratory testing and include decreased glomerular filtration rate and increased urinary albumin excretion rate.⁵⁴ Screening for proteinuria can be done at each visit using urine dip-

sticks.⁵⁵ Nonspecific symptoms of kidney disorders include fatigue, nausea, dyspnea, peripheral pitting edema, and oliguria.^{55,56} Symptoms of liver disorders include nausea, epigastric pain at the upper right quadrant, and shoulder pain.⁵ A complete blood count and kidney and liver function tests are useful in assessing systemic organ dysfunction.⁶

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.”⁵⁷ 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.⁵⁸ Delays in diagnosis can exacerbate such feelings. Early recognition is essential to ease the woman’s emotional pain as well as lower the risk of 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.⁵⁹ Volume overload may also lead to peripheral pitting edema.³² Enlargement of

the atria and ventricles may lead to development of ectopic foci and thus cardiac arrhythmia.⁶⁰ At clinical assessment, cardiac auscultation may reveal new-onset murmurs, indicating a mitral or tricuspid regurgitation, and elevated jugular venous pressure.³² Depending on the degree to which peripartum cardiomyopathy has progressed, different symptoms of heart failure at varying levels of severity may be present.⁶¹ The patient may either have a normal heart rate or be tachycardic, and arterial hypertension or hypotension may also be found.⁶¹ One review found that, at the time of diagnosis, about 75% of pregnant women with peripartum cardiomyopathy had heart failure symptoms corresponding to class III or IV of the NYHA Functional Classification system.⁴⁵ 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).³⁹

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.⁵⁵ Performing a fetal ultrasound can permit identification of an abnormal fetal heart rate (less than 120 or more than 160 beats per minute), 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.⁶² 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.⁵⁵

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, to determine the safest and most appropriate approach. In collaboration with other interdisciplinary team members, nurses should educate their pregnant patients on 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,^{20, 62} 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.⁶² 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 medications frequently used in managing pregnancy-specific CVDs and their specific use during pregnancy and breastfeeding, see Table 2.^{19, 23, 48, 55, 63–67}

Table 2. Drugs Used in Managing Pregnancy-Specific Cardiovascular Diseases: Recommendations for Use During Pregnancy and Breastfeeding

Drug Class	Use During Pregnancy	Use When Breastfeeding
Angiotensin-converting enzyme (ACE) inhibitors (such as benazepril, fosinopril)	Not recommended. ⁶³	Generally acceptable for use. ⁵⁵ But certain ACE inhibitors (such as captopril, enalapril) are preferred. ⁶⁴
Angiotensin receptor blockers (such as losartan, valsartan)	Not recommended. ⁶³	Not recommended, as profound hypotension in the infant may result. ⁴⁸
β-blockers (such as labetalol, metoprolol)	Generally acceptable for use. ⁶³	Generally acceptable for use. ⁵⁵
Calcium channel blockers (such as amlodipine, nifedipine)	Generally acceptable for use. ⁶⁵	Generally acceptable for use. ⁵⁵
Centrally acting antiadrenergics (such as clonidine, methyldopa)	Generally acceptable for use. ¹⁹	Methyldopa is generally acceptable for use. ^{19, 23} Clonidine is not recommended, as there are potential adverse effects for the infant. ⁶⁶
Diuretics (such as furosemide, hydrochlorothiazide)	Use cautiously to avoid compromising fetal perfusion. ⁶³	Generally acceptable for use. ⁶⁷ May decrease milk production.

Gestational hypertension. Antepartum phase.

First-line pharmacotherapy typically involves the administration of methyldopa, a centrally acting antiadrenergic, or labetalol (Trandate), a dual α -blocker and nonselective β -blocker.⁶⁸ 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.^{23,62} Prolonged or severe hypertension can lead to central nervous system injury.⁶²

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.¹⁹ Nurses can suggest home self-monitoring with an automated blood pressure device and explain its use.⁶⁹ For best results, patients should refrain from exercising at least 30 minutes before taking a reading and should sit up straight with legs uncrossed and 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 the 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 of thromboembolism.⁷⁰

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.⁷¹ Vaginal birth is preferred when the woman is stable and there are no obstetric indications for a cesarean section.⁶³ Advantages of vaginal birth include less blood loss, better hemodynamic stability, lack of surgery-related stress and anxiety, and fewer pulmonary complications.⁷²

Postpartum phase. During this phase, it's recommended that maternal blood pressure be assessed at least once at three to 10 days postpartum.⁷³ As for breastfeeding and drugs commonly used to treat either gestational hypertension or preeclampsia, there are usually no contraindications.⁵⁵ 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.⁷⁴

Preeclampsia and HELLP syndrome. Management of preeclampsia and HELLP syndrome includes all the recommendations described above for gestational hypertension, as well as these below.

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).^{20,75}

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.⁶²

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, β -blockers, hydralazine, nitrates, and heparin.⁷⁶ Managing volume status is essential.⁴¹ As such, salt and fluid intake restriction are necessary to prevent volume overload.⁶³ Light physical activity may still be encouraged in women with peripartum cardiomyopathy.^{77,78}

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.⁷⁹ 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.⁷⁹

Intrapartum phase. Early delivery is not indicated as long as the maternal-fetal condition is stable.^{30,71,80} In cases of maternal instability requiring the use of inotropes or mechanical support, fetal delivery by planned cesarean section 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.⁸¹ 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.⁸² Although 500 mL of crystalloid fluids is a typical dose,⁸³ the potential benefits of this practice must be weighed against the risks of volume

overload, hypoperfusion, and pulmonary edema.⁸⁴ A vasopressor can also be added as needed.⁸²

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.⁸⁵⁻⁸⁷ 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.^{32,71}

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.^{32, 88, 89} In such cases, nurses should offer counseling on contraceptive methods to reduce the odds of unplanned pregnancy.³² Progesterone-only forms of contraception are recommended, and emergency contraception should be considered if need be.⁹⁰ Combined hormonal contraceptives are contraindicated.

Fetal considerations. Pregnancy-specific CVDs can contribute to intrauterine or neonatal mortality,⁵⁵ especially in low- and middle-income countries.⁹¹ Pregnancy-specific CVDs also increase the risks of undesirable outcomes such as preterm birth, low birth weight, and low Apgar scores.^{71, 92} The severity of the maternal condition does not directly predict fetal and neonatal outcomes.⁶² Lower gestational age and abnormal results from more than one type of fetal monitoring may be better indicators of fetal morbidity and mortality risks.^{28, 55}

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.^{19, 55} All deliveries before 34 weeks of gestation should occur only in a clinical setting with the necessary maternal and neonatal intensive care resources.⁶²

Parents of infants born prematurely or with complications are likely to experience emotional shock, self-blame, sadness, and fear.⁹³ 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. ▼

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REFERENCES

1. Umehara M, Kobashi G. Epidemiology of hypertensive disorders in pregnancy: prevalence, risk factors, predictors and prognosis. *Hypertens Res* 2017;40(3):213-20.
2. Adam K. Pregnancy in women with cardiovascular diseases. *Methodist Debaque Cardiovasc J* 2017;13(4):209-15.
3. Bello N, et al. The relationship between pre-eclampsia and peripartum cardiomyopathy: a systematic review and meta-analysis. *J Am Coll Cardiol* 2013;62(18):1715-23.
4. Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. *Obstet Gynecol* 2003;102(1):181-92.
5. Verklan MT, Walden M, editors. *Core curriculum for neonatal intensive care nursing*. 5th ed. St. Louis: Elsevier Saunders; 2015.
6. American College of Obstetricians and Gynecologists. ACOG committee opinion No. 767: emergent therapy for acute-onset, severe hypertension during pregnancy and the postpartum period. *Obstet Gynecol* 2019;133(2):e174-e180.
7. World Health Organization (WHO) UNFPA, the United Nations Children's Fund (UNICEF), the International Confederation of Midwives (ICM), the International Council of Nurses (ICN), the International Federation of Gynecology and Obstetrics (FIGO) and the International Pediatric Association (IPA). *Definition of skilled health personnel providing care during childbirth: the 2018 joint statement by WHO, UNFPA, UNICEF, ICM, ICN, FIGO and IPA*. Geneva, Switzerland; 2018. WHO/RHR/18.14.
8. Hameed AB, et al. Pregnancy-related cardiovascular deaths in California: beyond peripartum cardiomyopathy. *Am J Obstet Gynecol* 2015;213(3):379.e1-e10.
9. Hernandez LE, et al. Pregnancy-related deaths, Florida, 1999-2012: opportunities to improve maternal outcomes. *Matern Child Health J* 2018;22(2):204-15.
10. Anthony J, et al. Hypertensive disorders of pregnancy: what the physician needs to know. *Cardiovasc J Afr* 2016;27(2):104-10.
11. Cairns AE, et al. Postpartum management of hypertensive disorders of pregnancy: a systematic review. *BMJ Open* 2017;7(11):e018696.
12. Gillon TE, et al. Hypertensive disorders of pregnancy: a systematic review of international clinical practice guidelines. *PLoS One* 2014;9(12):e113715.
13. Anderson CM, Schmella MJ. Preeclampsia: current approaches to nursing management. *Am J Nurs* 2017;117(11):30-8.
14. Melchiorre K, et al. Maternal cardiovascular function in normal pregnancy: evidence of maladaptation to chronic volume overload. *Hypertension* 2016;67(4):754-62.
15. Hunter S, Robson SC. Adaptation of the maternal heart in pregnancy. *Br Heart J* 1992;68(6):540-3.
16. Sanghavi M, Rutherford JD. Cardiovascular physiology of pregnancy. *Circulation* 2014;130(12):1003-8.
17. Cheung KL, Lafayette RA. Renal physiology of pregnancy. *Adv Chronic Kidney Dis* 2013;20(3):209-14.

18. Lang RM, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr* 2015;28(1):1-39, 39.e1-e14.
19. Butalia S, et al. Hypertension Canada's 2018 guidelines for the management of hypertension in pregnancy. *Can J Cardiol* 2018;34(5):526-31.
20. Pfaff NF. The new hypertensive guidelines for pregnancy: what every nurse should know. *J Perinat Neonatal Nurs* 2014;28(2):91-3.
21. Kintiraki E, et al. Pregnancy-induced hypertension. *Hormones (Athens)* 2015;14(2):211-23.
22. Melamed N, et al. Gestational hypertension and preeclampsia: are they the same disease? *J Obstet Gynaecol Can* 2014;36(7):642-7.
23. American College of Obstetricians and Gynecologists, Task Force on Hypertension in Pregnancy. 2013. Hypertension in pregnancy. Washington, DC.
24. Ghulmiyyah L, Sibai B. Maternal mortality from preeclampsia/eclampsia. *Semin Perinatol* 2012;36(1):56-9.
25. Townsend NS, Drummond SB. Preeclampsia: pathophysiology and implications for care. *J Perinat Neonatal Nurs* 2011;25(3):245-52.
26. Preeclampsia Foundation. *Heart disease and stroke*. 2019. <https://www.preeclampsia.org/health-information/heart-disease-stroke>.
27. Abildgaard U, Heimdal K. Pathogenesis of the syndrome of hemolysis, elevated liver enzymes, and low platelet count (HELLP): a review. *Eur J Obstet Gynecol Reprod Biol* 2013;166(2):117-23.
28. Harmon QE, et al. Risk of fetal death with preeclampsia. *Obstet Gynecol* 2015;125(3):628-35.
29. European Society of Gynecology (ESG), Association for European Paediatric Cardiology (AEPIC), German Society for Gender Medicine (DGesGM), et al. ESC Guidelines on the management of cardiovascular diseases during pregnancy: the Task Force on the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology (ESC). *Eur Heart J* 2011;32(24):3147-97.
30. Sliwa K, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on Peripartum Cardiomyopathy. *Eur J Heart Fail* 2010;12(8):767-78.
31. Hibbard JU, et al. A modified definition for peripartum cardiomyopathy and prognosis based on echocardiography. *Obstet Gynecol* 1999;94(2):311-6.
32. Sharma P, Kumar B. Peripartum cardiomyopathy: an obstetric review. *Int J Reprod Contracept Obstet Gynecol* 2017;6(2):371-8.
33. Hilfiger-Kleiner D, et al. Peripartum cardiomyopathy: current management and future perspectives. *Eur Heart J* 2015;36(18):1090-7.
34. Ware JS, et al. Shared genetic predisposition in peripartum and dilated cardiomyopathies. *N Engl J Med* 2016;374(3):233-41.
35. Abou Moulig V, et al. Five-year follow-up in patients with peripartum cardiomyopathy (PPCM) shows high and stable recovery rate and longterm use of cardiovascular medication. *Eur J Heart Fail* 2018 (Suppl S1):396.
36. Codsí E, et al. Subsequent pregnancy outcomes in patients with peripartum cardiomyopathy. *Obstet Gynecol* 2018;131(2):322-7.
37. Elkayam U. Risk of subsequent pregnancy in women with a history of peripartum cardiomyopathy. *J Am Coll Cardiol* 2014;64(15):1629-36.
38. Sliwa K, et al. Long-term prognosis, subsequent pregnancy, contraception and overall management of peripartum cardiomyopathy: practical guidance paper from the Heart Failure Association of the European Society of Cardiology Study Group on Peripartum Cardiomyopathy. *Eur J Heart Fail* 2018;20(6):951-62.
39. American Heart Association (AHA). *Classes of heart failure*. 2017. <https://www.heart.org/en/health-topics/heart-failure/what-is-heart-failure/classes-of-heart-failure>.
40. Sliwa K, et al. Peripartum cardiomyopathy: inflammatory markers as predictors of outcome in 100 prospectively studied patients. *Eur Heart J* 2006;27(4):441-6.
41. Arany Z. Understanding peripartum cardiomyopathy. *Annu Rev Med* 2018;69:165-76.
42. Patel PA, et al. A contemporary review of peripartum cardiomyopathy. *Clin Med (Lond)* 2017;17(4):316-21.
43. Shen M, et al. Comparison of risk factors and outcomes of gestational hypertension and pre-eclampsia. *PLoS One* 2017;12(4):e0175914.
44. Sliwa K, et al. Clinical characteristics of patients from the worldwide registry on peripartum cardiomyopathy (PPCM): EURObservational Research Programme in conjunction with the Heart Failure Association of the European Society of Cardiology Study Group on PPCM. *Eur J Heart Fail* 2017;19(9):1131-41.
45. Asad ZUA, et al. Peripartum cardiomyopathy: a systematic review of the literature. *Clin Cardiol* 2018;41(5):693-7.
46. American College of Obstetricians and Gynecologists. *Nutrition during pregnancy*. Washington, DC; 2018 Feb. FAQ001. FAQs; <https://www.acog.org/-/media/For-Patients/faq001.pdf>.
47. Davenport MH, et al. Prenatal exercise for the prevention of gestational diabetes mellitus and hypertensive disorders of pregnancy: a systematic review and meta-analysis. *Br J Sports Med* 2018;52(21):1367-75.
48. Gupta D, Wenger NK. Peripartum cardiomyopathy: status 2018. *Clin Cardiol* 2018;41(2):217-9.
49. Gilbert L, et al. How diet, physical activity and psychosocial well-being interact in women with gestational diabetes mellitus: an integrative review. *BMC Pregnancy Childbirth* 2019;19(1):60.
50. Syngelaki A, et al. Diet and exercise for preeclampsia prevention in overweight and obese pregnant women: systematic review and meta-analysis. *J Matern Fetal Neonatal Med* 2018;1-7.
51. Ohkuchi A, et al. Prediction and prevention of hypertensive disorders of pregnancy. *Hypertens Res* 2017;40(1):5-14.
52. Marchand A, et al. The predictive value of serum haptoglobin in hemolytic disease. *JAMA* 1980;243(19):1909-11.
53. Surányi A, et al. Evaluation of placental vascularization by three-dimensional ultrasound examination in second and third trimester of pregnancies complicated by chronic hypertension, gestational hypertension or pre-eclampsia. *Pregnancy Hypertens* 2017;8:51-9.
54. Wouters OJ, et al. Early chronic kidney disease: diagnosis, management and models of care. *Nat Rev Nephrol* 2015;11(8):491-502.
55. Magee LA, et al. Diagnosis, evaluation, and management of the hypertensive disorders of pregnancy: executive summary. *J Obstet Gynaecol Can* 2014;36(5):416-41.
56. Moore PK, et al. Management of acute kidney injury: core curriculum 2018. *Am J Kidney Dis* 2018;72(1):136-48.
57. Sibai BM. Diagnosis, controversies, and management of the syndrome of hemolysis, elevated liver enzymes, and low platelet count. *Obstet Gynecol* 2004;103(5 pt 1):981-91.
58. Patel H, et al. Symptoms in women with peripartum cardiomyopathy: a mixed method study. *Midwifery* 2016;32:14-20.
59. Moiola M, et al. Peripartum cardiomyopathy. *Arch Gynecol Obstet* 2010;281(2):183-8.
60. Andrade J, et al. The clinical profile and pathophysiology of atrial fibrillation: relationships among clinical features, epidemiology, and mechanisms. *Circ Res* 2014;114(9):1453-68.
61. Elkayam U. Clinical characteristics of peripartum cardiomyopathy in the United States: diagnosis, prognosis, and management. *J Am Coll Cardiol* 2011;58(7):659-70.

62. Kirkpatrick SJ, et al., editors. *Guidelines for perinatal care*. 8th ed. Elk Grove Village, IL; Washington, DC: American Academy of Pediatrics and the American College of Obstetricians and Gynecologists; 2017.
63. Bouabdallaoui N, et al. Current knowledge and recent development on management of peripartum cardiomyopathy. *Eur Heart J Acute Cardiovasc Care* 2017;6(4):359-66.
64. Bauersachs J, et al. Current management of patients with severe acute peripartum cardiomyopathy: practical guidance from the Heart Failure Association of the European Society of Cardiology Study Group on peripartum cardiomyopathy. *Eur J Heart Fail* 2016;18(9):1096-105.
65. Alabdulrazzaq F, Koren G. Fetal safety of calcium channel blockers. *Can Fam Physician* 2012;58(7):746-7.
66. National Library of Medicine. Drugs and Lactation Database (LactMed) [Internet]. In: *Clonidine*. Bethesda, MD; 2018. <https://www.ncbi.nlm.nih.gov/books/NBK501628>.
67. Schlembach D, et al. Treating hypertension in pregnancy. *Curr Hypertens Rep* 2015;17(8):63.
68. Al Khaja KA, et al. Drug treatment of hypertension in pregnancy: a critical review of adult guideline recommendations. *J Hypertens* 2014;32(3):454-63.
69. Uhlig K, et al. Self-measured blood pressure monitoring in the management of hypertension: a systematic review and meta-analysis. *Ann Intern Med* 2013;159(3):185-94.
70. Spiro L, Scemons D. Management of chronic and gestational hypertension of pregnancy: a guide for primary care nurse practitioners. *Open Nurs J* 2018;12:180-3.
71. Ersbøll AS, et al. Peripartum cardiomyopathy: a systematic literature review. *Acta Obstet Gynecol Scand* 2016;95(11):1205-19.
72. Ray P, et al. Recognition and management of maternal cardiac disease in pregnancy. *Br J Anaesth* 2004;93(3):428-39.
73. American College of Obstetrics and Gynecologists. ACOG committee opinion No. 736: optimizing postpartum care. *Obstet Gynecol* 2018;131(5):e140-e150.
74. Folker-Maglaya C, et al. Implementing a breastfeeding toolkit for nursing education. *J Perinat Neonatal Nurs* 2018;32(2):153-63.
75. Witcher PM, et al. Multisystem effects of hypertensive disorders of pregnancy: a comprehensive review. *J Perinat Neonatal Nurs* 2015;29(3):229-39.
76. Jackson AM, et al. Peripartum cardiomyopathy: diagnosis and management. *Heart* 2018;104(9):779-86.
77. American College of Obstetricians and Gynecologists. ACOG committee opinion No. 650: physical activity and exercise during pregnancy and the postpartum period. *Obstet Gynecol* 2015;126(6):e135-e142.
78. Jain R, et al. Peripartum cardiomyopathy: a review. *J Evol Med Dent Sci* 2016;5(43):2714-7.
79. Loyaga-Rendon RY, et al. Outcomes of patients with peripartum cardiomyopathy who received mechanical circulatory support. Data from the Interagency Registry for Mechanically Assisted Circulatory Support. *Circ Heart Fail* 2014;7(2):300-9.
80. Regitz-Zagrosek V, et al. 2018 ESC guidelines for the management of cardiovascular diseases during pregnancy. *Eur Heart J* 2018;39(34):3165-241.
81. Langesæter E, Dyer RA. Maternal haemodynamic changes during spinal anaesthesia for caesarean section. *Curr Opin Anaesthesiol* 2011;24(3):242-8.
82. Arendt KW. Anesthesia for labor and delivery in high-risk heart disease: general considerations. *UpToDate* 2019. <https://www.uptodate.com/contents/anesthesia-for-labor-and-delivery-in-high-risk-heart-disease-general-considerations>.
83. Lindstrom H, et al. How midwives manage rapid pre-loading of fluid in women prior to low dose epidurals: a retrospective chart review. *J Adv Nurs* 2018;74(11):2588-95.
84. Caughey AB, et al. Guidelines for intraoperative care in cesarean delivery: enhanced recovery after surgery society recommendations (part 2). *Am J Obstet Gynecol* 2018;219(6):533-44.
85. Hilfiker-Kleiner D, et al. Bromocriptine for the treatment of peripartum cardiomyopathy: a multicentre randomized study. *Eur Heart J* 2017;38(35):2671-9.
86. Johnson-Coyle L, et al. Peripartum cardiomyopathy: review and practice guidelines. *Am J Crit Care* 2012;21(2):89-98.
87. Safirstein JG, et al. Predictors of left ventricular recovery in a cohort of peripartum cardiomyopathy patients recruited via the internet. *Int J Cardiol* 2012;154(1):27-31.
88. Bozkurt B, et al. Current diagnostic and treatment strategies for specific dilated cardiomyopathies: a scientific statement from the American Heart Association. *Circulation* 2016;134(23):e579-e646.
89. Hess RF, Weinland JA. The life-changing impact of peripartum cardiomyopathy: an analysis of online postings. *MCN Am J Matern Child Nurs* 2012;37(4):241-6.
90. Curtis KM, et al. U.S. medical eligibility criteria for contraceptive use, 2016. *MMWR Morb Mortal Wkly Rep* 2016;65(3):1-104.
91. Saleem S, et al. A prospective study of maternal, fetal and neonatal deaths in low- and middle-income countries. *Bull World Health Organ* 2014;92(8):605-12.
92. Magee LA, et al. The CHIPS randomized controlled trial (control of hypertension in pregnancy study): is severe hypertension just an elevated blood pressure? *Hypertension* 2016;68(5):1153-9.
93. Yang YY, et al. Perceptions of parents with preterm infants hospitalized in Singaporean neonatal intensive care unit. *J Perinat Neonatal Nurs* 2017;31(3):263-73.

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