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Society for Maternal-Fetal Medicine Consult Series #73: Diagnosis and management of right and left heart failure during pregnancy and postpartum

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Abstract

Heart failure is a major contributor to maternal morbidity and mortality. Pregnancy is a state of hemodynamic stress, and normal physiologic changes of pregnancy may mimic signs of heart failure. Prepregnancy counseling, multidisciplinary care, and referral to a center with expertise in managing pregnant patients with heart failure can help optimize outcomes. The option for abortion care should be available to all patients with heart failure, regardless of the severity of the disease, and is an essential component of individualized counseling. In this Consult, we provide guidance for managing patients with heart failure with reduced ejection fraction who are continuing pregnancy. The following are Society for Maternal-Fetal Medicine (SMFM) recommendations: (1) we recommend that all patients with right heart failure due to pulmonary arterial hypertension receive counseling about high rates of maternal morbidity and mortality; if pregnancy is pursued, the patient should be referred to a center with expertise in this condition to guide management during pregnancy and postpartum (GRADE 1C); (2) we recommend considering referral to a genetics provider with expertise in heritable cardiac disease for people with peripartum cardiomyopathy (PPCM), particularly when the index of suspicion is high and no other contributing factors are identified (GRADE 1C); (3) we recommend that other causes of heart failure be ruled out before making a diagnosis of PPCM (Best Practice); (4) for acute left ventricular heart failure during pregnancy, we recommend hydralazine or isosorbide dinitrate for afterload reduction and furosemide for diuresis. For acute left ventricular failure postpartum, we recommend afterload reduction with angiotensin-converting enzyme inhibitor (ACEi), angiotensin receptor blocker (ARB), or angiotensin receptor/neprilysin inhibitor (ARNi) unless contraindicated (e.g., renal failure) (GRADE 1B); (5) we recommend against inotropic blockade (i.e., beta-blockers) in the setting of acute decompensated left ventricular heart failure (GRADE 1B); (6) we recommend prophylactic anticoagulation administration in hospitalized pregnant patients with acute left ventricular heart failure (GRADE 1C); (7) in patients who are pursuing pregnancy or pregnant, we recommend discontinuing spironolactone, ACEi, ARB, and ARNi and continuing beta-blockers (metoprolol, carvedilol, bisoprolol) (GRADE 1C); (8) for pregnant patients with left ventricular failure and ejection fraction < 35%, we recommend pharmacologic thromboprophylaxis during pregnancy and for six weeks postpartum (GRADE 1C); (9) for patients with chronic left ventricular failure, we recommend starting or continuing guideline-directed medical therapy when medically able, in consultation with experts in cardiology (GRADE 1C); (10) we recommend fetal echocardiography when maternal heart failure is a result of an underlying congenital cardiac defect (GRADE 1C); (11) we recommend serial growth ultrasounds in pregnancies complicated by maternal heart failure (GRADE 1C); (12) we recommend continuous fetal heart rate monitoring during anesthesia administration, labor, and delivery for pregnant patients with heart failure (GRADE 1B); (13) in the case of maternal cardiovascular changes prompting inpatient assessment or treatment, we recommend continuous or intermittent fetal heart rate monitoring, taking into consideration the gestational age and any relevant maternal or fetal factors that may impact fetal viability or the maternal clinical status (GRADE 1C); (14) we recommend planned vaginal delivery at term in patients with heart failure in the absence of hemodynamic compromise or obstetric indications for cesarean (GRADE 1C); (15) we recommend the use of neuraxial anesthesia in most patients with heart failure to provide appropriate analgesia and to limit the effects of labor on cardiac parameters (GRADE 1C); (16) we recommend considering a limited or assisted second stage for some patients after input from cardiology about each individual patient's cardiac risk (GRADE 1C); (17) we recommend that postpartum patients with heart failure undergo routine counseling regarding infant feeding. We recommend reviewing all medications for compatibility with breastfeeding and using shared decision-making in the absence of robust data (GRADE 1B).

KEYWORDS
cardiac disease, counseling, echocardiography, heart failure, maternal monitoring, maternal morbidity, maternal mortality, peripartum cardiomyopathy, pulmonary hypertension

1 | INTRODUCTION

Heart failure is a complex clinical syndrome with signs and symptoms that result from any structural or functional impairment of ventricular filling or ejection of blood [1]. The right and left sides of the heart are interdependent yet independent; the "two hearts" model visualizes the right and left chambers as separate entities with the lungs between them [2]. This model helps illustrate the pathophysiology of heart failure and management strategies when one side of the heart is primarily compromised [2]. Diseases of the left heart invariably lead to changes in the right heart over time [3].

2 | CLINICAL QUESTIONS

2.1 | What is the maternal morbidity and mortality associated with heart failure?

It is difficult to calculate the current overall incidence of heart failure in pregnancy, given the varying etiologies of heart failure, limitations of existing data sources, geographic and temporal variation, and differing methodologies of previous analyses. Despite these limitations, heart disease is known to be a leading cause of pregnancy-related deaths in the United States [4–6], and the number of pregnant people with heart disease has increased [7, 8]. Concerning racial disparities persist in maternal mortality, and a disproportionate percentage of pregnancy-related deaths from cardiovascular conditions occur in non-Hispanic Black individuals [5, 9]. Pregnant and postpartum patients with heart failure are at increased risk for a variety of perinatal adverse outcomes, including maternal mortality [6, 10–12], and should be managed in or referred to an institution with experience caring for pregnant people with cardiac disease.

2.2 | What cardiac changes in pregnancy can potentiate heart failure?

Pregnancy is a state of hemodynamic stress. Physiologic changes of pregnancy increase perfusion conducive to the growth and development of the uteroplacental unit. There is a 30%–50% increase in blood volume and cardiac output by the third trimester, 75% of which has occurred by the end of the first trimester [13]. Heart rate increases steadily by 10–20 beats per minute (bpm) throughout pregnancy but seldom exceeds 100 bpm [14]. Moreover, heart rate varies across gestation with changes in maternal positioning [15, 16]. Systemic and pulmonary vascular resistance decreases due to the vasodilatory effects of progesterone, estrogen, relaxin, and prostaglandins. Vasodilation leads to decreases in systolic and diastolic blood pressure, though the effect on the latter is more pronounced [17]. These physiologic changes may lead to variations in some diagnostic tests commonly used for heart failure evaluation, such as B-type natriuretic peptide (BNP) levels and echocardiography; therefore, results should be interpreted with caution.

However, BNP remains a useful marker for diagnosing and managing heart failure. In normal pregnancy, BNP or its inactive amino-terminal fragment NT-pro-BNP may rise twofold compared to a non-pregnant state while remaining within the normal range [18, 19]. BNP levels are lower in individuals with obesity and may be elevated in those with preeclampsia, congenital heart disease, cardiomyopathy, sepsis, renal failure, pulmonary embolism, critical illness, and anemia [19, 20]. Echocardiographic changes in pregnancy include increased left and right ventricular dimension and volume, stroke volume, and cardiac output without a change in the left ventricular ejection fraction (LVEF) [21]. Table 1 summarizes specific measurements and findings on echocardiography beyond the overall function and ejection fraction (EF) [22–26].

2.3 | How is heart failure defined?

Heart failure is a clinical syndrome resulting from structural or functional abnormalities of the heart that compromise its ability to fill or eject blood normally. It may affect the left ventricle, the right ventricle, or both ventricles. Broadly, there are two types of heart failure: heart failure with reduced EF (HFrEF) and heart failure with preserved EF (HFpEF) [27]. Both types of heart failure (HFrEF and HFpEF) lead to a compromised ability of the heart to fill or eject blood, causing symptoms such as shortness of breath and fatigue [28].

HFrEF is defined by LVEF < 40% and constitutes the primary type of heart failure seen in pregnancy [29]. It includes peripartum cardiomyopathy (PPCM) and dilated cardiomyopathy (DCM). HFrEF is characterized by pressure overload, volume overload, and decreased contractility. In this setting, the adaptive response to maintain perfusion to vital organs leads to ventricular remodeling over time and the onset of shortness of breath and fatigue.

Although HFpEF constitutes a large proportion of heart failure in older adults, it is not frequently identified in pregnancy and is thus not discussed here [30].

2.4 | How should patients with a history of heart failure be counseled before pregnancy?

Prepregnancy counseling in patients with heart failure should involve assessing functional status using the New York Heart Association (NYHA) functional classification (Table 2) [31]; reviewing the etiology of heart failure and its impact on pregnancy; assessing for structural defects, prior cardiac events, and the presence or absence of arrhythmias; and evaluating the compatibility of heart failure medications with pregnancy. Cardiac risk stratification should be based on well-accepted models, such as the Cardiac Disease in Pregnancy Study risk prediction index (CARPREG II, Figure 1) and modified World Health Organization classification (mWHO, Figure 2) [32–34]. Patients with persistent left ventricular dysfunction (LVEF < 45%) after a diagnosis of PPCM in a prior pregnancy or those with EF < 30% [35–37] at the time of presentation with PPCM should be advised against pregnancy. The option for abortion care should be available to all patients with heart failure, regardless of the severity of the disease, and is an essential component of early pregnancy counseling [37].

The scores mentioned above (NYHA, CARPREG II, and mWHO) are not interchangeable; they can be used in an additive fashion to communicate the cardiac status of a pregnant patient to other care providers. NYHA is a scoring system used in non-pregnant and pregnant adults (Table 2), CARPREG II is a scoring system specific to pregnant individuals (Figure 1), and mWHO is a pregnancy-specific classification system (Figure 2). The total CARPREG II score determines the risk for an

TABLE 1 Relevant echocardiographic parameters, clinical implications, and reasonable next steps for management of abnormal results.

Parameter	Normal values in reproductive-aged females	Clinical implication	Next steps
RVSP	< 40 mmHg	The tricuspid regurgitant jet velocity is a non-invasive measure of pulmonary artery systolic pressure and quantifies pulmonary hypertension. Elevated levels can be indicative of pulmonary hypertension, fluid overload, or right ventricular dysfunction [22, 23].	Confirmation: 25% of RVSP measures are inaccurate. Consider other echocardiographic and clinical parameters, serial follow-up, and expert consultation. Right heart catheterization may be advised. If confirmed, classification into the five groups of pulmonary hypertension is recommended (see Table 4). The classification/group usually guides treatment [22, 23].
TAPSE	> 1.7 m/s	Assessment of right ventricular function. M-mode is used to measure the vertical movement of the lateral tricuspid valve annulus. Lower values can be indicative of right ventricular dysfunction and failure [22, 23].	Expert consultation: Right ventricular failure or depression can be very morbid and mortal in the context of pregnancy. Swift consultation is advised. Observe other right-sided echocardiographic findings, including right atrial volume and IVC diameter. Medical management could include diuresis, afterload reduction, and inotropy [22, 23].
Left atrial volume	22–52 mL [24]	Increased size indicates increased filling pressures and fluid overload and can place patients at risk for arrhythmia (i.e., atrial fibrillation).	Expert consultation: Increased left atrial volume can indicate heart failure or obstructive process and left-sided cardiac pathology (i.e., heart failure, mitral stenosis, aortic stenosis [24, 25], hypertrophic cardiomyopathy). Observe other echocardiographic findings. Assess mitral and aortic valves for stenosis or regurgitation, left ventricular failure, etc. Medical management can include diuresis, afterload reduction, beta-blockade for arrhythmia treatment.
Mitral septal E/e’ ratio	< 13	Increased levels are indicative of increased left ventricular filling pressure and increased pulmonary capillary wedge pressures and can be found in HFpEF [26].	Expert consultation: Increased mitral septal E/e’ values > 13 can be indicative of increased left ventricular filling pressures, fluid overload, and heart failure [26]. Observe other echocardiographic findings such as valvular function, left atrial volume, left ventricular systolic function, and left ventricular wall thickness. Medical management can include treatment of chronic medical conditions (i.e., hypertension), diuresis, and expanding differential diagnosis for chronic cardiac disease and hypertrophic cardiomyopathy.

Abbreviations: E, mitral inflow velocity of early diastolic filling; e’, tissue Doppler mitral annular velocity; HFpEF, heart failure with preserved ejection fraction; IVC, inferior vena cava; RVSP, right ventricular systolic pressure; TAPSE, transannular planar systolic excursion.

TABLE 2 New York Heart Association (NYHA) classification [31].

Class	Patient symptoms
I	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or shortness of breath.
II	Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity results in fatigue, palpitation, shortness of breath, or chest pain.
III	Marked limitation of physical activity. Comfortable at rest. Less ordinary activity causes fatigue, palpitation, shortness of breath, or chest pain.
IV	Symptoms of heart failure at rest. Any physical activity causes further discomfort.

PREDICTOR	POINTS
Prior cardiac events or arrhythmias	3
Baseline NYHA III-IV or cyanosis	3
Mechanical valve	3
Ventricular systolic dysfunction[a]	2
High risk left-sided valve disease/left ventricular outflow tract obstruction[b]	2
Pulmonary hypertension	2
Coronary artery disease[c]	2
High risk aortopathy[d]	2
No prior cardiac intervention[e]	1
Late pregnancy assessment[f]	1

FIGURE 1 CARPREG II risk predictors. Reprinted with permission from [33]. [a]Left ventricular ejection fraction <55%. [b]Aortic valve <1.5 cm^2, subaortic gradient >30 mmHg, mitral valve area <2 cm^2, moderate to severe mitral regurgitation. [c]Angiographically proven coronary obstruction or past myocardial infarction. [d]Marfan syndrome, bicuspid aortopathy with aortic dimension >45 mm, Loeys-Dietz syndrome, vascular Ehlers-Danlos syndrome, prior aortic dissection or pseudoaneurysm. [e]No cardiac repair of congenital lesions, valvular replacement or repair, percutaneous or operative treatment of arrhythmias. [f]First visit after 20 weeks of gestation. Abbreviation: NYHA, New York Heart Association functional class.

antepartum or postpartum cardiac event. In the primary study, the predicted risk for a primary cardiac event was 5% (0–1 points), 10% (2 points), 15% (3 points), 22% (4 points), and 41% (> 4 points) [33]. The mWHO integrates all known maternal cardiovascular risk factors and groups patients into risk categories ranging from I (shown in yellow, no detectable increased risk of maternal mortality and no/mild increased risk in morbidity) to IV (shown in red, extremely high risk of maternal mortality or severe morbidity). It provides considerations for follow-up care and delivery [34].

2.5 | What are the main causes of *acute* right ventricular failure during pregnancy?

Right ventricular failure is the impaired ability of the right ventricle to perfuse the lungs. The most common cause of right ventricular failure is left-sided heart failure. Under normal circumstances, the right ventricle pumps against low resistance, low pressure, and high compliance pulmonary vasculature that can accommodate a large volume of blood flow without an increase in pulmonary artery pressure [38, 39]. Right ventricular systolic dysfunction reduces forward flow to the pulmonary circulation, decreasing the left ventricular stroke volume and cardiac output. Subsequent neurohormonal activation promotes renal sodium and water retention, causing systemic venous hypertension resulting in hepatic congestion, ascites, and gut and lower extremity edema [40].

The right ventricle is sensitive to afterload. It is not capable of generating high systolic pressures under normal circumstances as it is accustomed to pumping into the low-resistance pulmonary circulation; therefore, any sudden increase in pulmonary artery pressure (e.g., from a pulmonary embolism) may lead to cardiogenic shock due to an inability to maintain forward flow [39]. The main causes of acute and chronic right ventricular failure are summarized in Table 3.

TABLE 3 Causes of acute and chronic right ventricular failure in pregnancy.

Acute right ventricular failure	Chronic right ventricular failure
Embolism • Pulmonary thromboembolism • Amniotic fluid embolism • Air or fat embolism Right ventricular infarction	Left heart failure Right-sided valve disease Cardiomyopathies involving the right ventricle Pulmonary hypertension Chronic thromboembolic disease Interstitial lung disease

2.6 | What are the main causes of *chronic* right ventricular failure?

Chronic heart failure may be asymptomatic in early pregnancy and become symptomatic with peaking of cardiac output as pregnancy progresses. Among the various causes of chronic right heart failure in pregnancy (Table 3), pulmonary hypertension deserves special attention. The Sixth World Symposium on Pulmonary Hypertension defined pulmonary hypertension as mean pulmonary artery pressure > 20 mmHg via right heart catheterization and classified it into five groups (Table 4) [41].

2.7 | How should right ventricular failure be diagnosed during pregnancy?

The symptoms of right ventricular failure may be nonspecific and include shortness of breath on exertion and fatigue [42]. Fluid retention, ascites, and hepatomegaly are more likely in advanced cases of right ventricular failure and may be challenging to discern in pregnancy. Echocardiogram, electrocardiogram, and BNP levels should be considered as the initial diagnostic tests [42]. Right ventricular function, size of the right atrium, collapsibility of

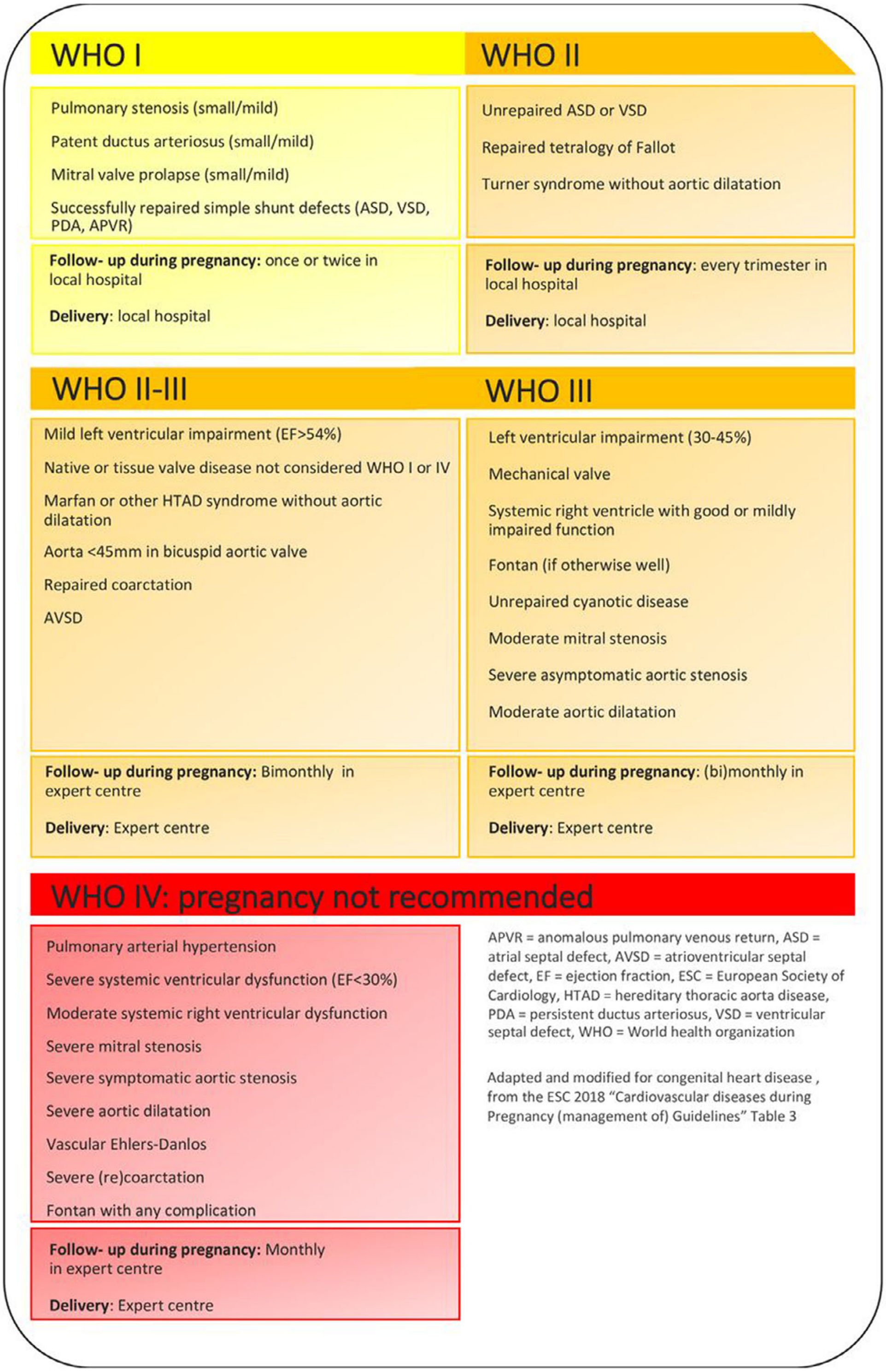

FIGURE 2 Modified World Health Organization classification of maternal cardiovascular risk. Reprinted with permission from [34].

the inferior vena cava, severity of tricuspid regurgitation, and estimation of the pulmonary artery pressure determine the severity of right ventricular dysfunction. Elevated central venous pressure (> 10 mmHg) and evidence of right heart dysfunction are hallmarks of right ventricular failure diagnosis on echocardiography. Right heart catheterization is considered the gold standard for diagnosis but is unlikely to be necessary in most cases. Patients with signs or symptoms concerning for right ventricular heart failure should have a careful physical examination, laboratory assessment, and echocardiography as part of the initial workup, with additional imaging as needed.

TABLE 4 Pulmonary hypertension groups and associated causes [41].

Group 1: Pulmonary arterial hypertension	Idiopathic, genetic, drug-induced, toxin, portal, connective tissue disorder, HIV, calcium channel related, PAH with overt features of venous/capillaries (PVOD/PCH) involvement, persistent pulmonary hypertension of the newborn syndrome
Group 2: Pulmonary hypertension due to left heart disease	Pulmonary hypertension due to left heart failure (reduced and preserved EF), valvular, congenital, or acquired heart disease
Group 3: Pulmonary hypertension due to lung disease and/or hypoxia	Obstructive lung disease, restrictive lung disease, other lung disease with mixed restrictive/obstructive, hypoxia without lung disease, and developmental lung disease
Group 4: Pulmonary hypertension due to pulmonary artery obstructions	Chronic thromboembolic emboli, other pulmonary artery obstructions
Group 5: Pulmonary hypertension with unclear and/or multifactorial mechanisms	Hematologic disorders, systematic and metabolic disorders, Others, complex congenital heart disease

Abbreviations: EF, ejection fraction; LVEF, left ventricular ejection fraction; PAH, pulmonary arterial hypertension; PCH, pulmonary capillary hemangiomatosis; PVOD, pulmonary veno-occlusive disease.
Adapted from [41]

2.8 | How should right ventricular failure be managed during pregnancy?

The varied etiologies of right ventricular failure necessitate individualized management strategies. In the case of preexisting right ventricular failure, prepregnancy pharmacotherapy should be continued with adjustment of medications as needed based on the pregnancy safety profile.

Pregnant patients with right heart failure due to pulmonary hypertension should be thoughtfully counseled about the high risk of maternal morbidity and mortality [32, 43, 44], particularly as abnormal right ventricular systolic function portends a poor prognosis [32, 43–45]. Generally, management involves optimizing tissue perfusion and oxygenation. Oxygen saturations should be maintained at ≥ 90% [46] (preferably ≥ 95%) as the pulmonary vasculature reacts to hypoxia with vasoconstriction, further decreasing perfusion and worsening the existing hypoxemia [47]. Intravascular volume must also be maintained to allow adequate right ventricular output. Most notably, these patients are at substantially increased risk for maternal death in the immediate postpartum period [43, 48–50]. The detailed management of pulmonary hypertension is beyond the scope of this review; all patients with a diagnosis of WHO group 1 pulmonary arterial hypertension should be referred to a center with expertise in pulmonary hypertension [51].

Right ventricular failure due to left heart failure or volume overload is largely managed with diuretic therapy. For patients with pulmonary embolism and right ventricular infarction, anticoagulation therapy and maintenance of intravascular volume are critical to maintain hemodynamic stability. Right ventricular failure due to arrhythmias and low cardiac output should be treated based on the underlying etiology. Patients with right ventricular failure and other indications (e.g., atrial fibrillation, thrombosis) may be candidates for anticoagulation [52]. **We recommend that all patients with right heart failure due to pulmonary arterial hypertension receive counseling about high rates of maternal morbidity and mortality; if pregnancy is pursued, the patient should be referred to a center with expertise in this condition to guide management during pregnancy and postpartum (GRADE 1C)**. Pregnant patients with right heart failure not due to pulmonary hypertension may be managed in conjunction with maternal-fetal medicine subspecialists and cardiologists.

2.9 | What are the main causes of *acute* left ventricular failure in pregnancy?

Common causes of left ventricular failure in pregnancy are outlined in the Box. PPCM is a form of DCM that occurs towards the end of pregnancy or in the months following delivery with no identifiable cause [53]. A 2014 study using the National Inpatient Sample noted that the incidence of pregnancies complicated by PPCM from 2004 to 2011 was 10.3 per 10,000 live births, increasing from 8.5 to 11.8 per 10,000 live births over the eight-year period [7]. This same study noted an overall rate of 13.5% for any major maternal adverse event (in-hospital mortality, cardiac arrest, heart transplant, mechanical circulatory support, acute pulmonary edema, thromboembolism, or implantable defibrillator/permanent pacemaker) in patients with PPCM. There was no temporal increase in this overall rate over the observed eight-year period; however, there was a slight increase in in-hospital mortality (07% to 1.8%), mechanical circulatory support (0.9% to 2.2%), and cardiogenic shock (1.0% to 4.0%) [7]. This study was not designed to address obstetric or neonatal outcomes.

BOX. Causes of left ventricular failure in pregnancy

Cardiomyopathy
- Peripartum cardiomyopathy
- Non-ischemic dilated cardiomyopathy
- Tachycardia-induced cardiomyopathy
- Stress (Takotsubo) cardiomyopathy
- Hypertrophic cardiomyopathy
- Left ventricular non-compaction

Progressive valve disease
- Moderate to severe aortic stenosis
- Moderate to severe mitral stenosis
- Severe aortic regurgitation
- Severe mitral regurgitation

Acute valve disease
- Prosthetic valve thrombosis

Arrhythmias
- Atrial fibrillation
- Atrial flutter
- High-frequency ventricular ectopy
- Ventricular tachycardia

Acute coronary syndrome
- Coronary artery dissection
- Thrombosis

Ion channel disorders

Myocarditis

Acute diastolic dysfunction
- Preeclampsia with severe range blood pressure

PPCM presents on echocardiography with left ventricular enlargement and dysfunction with EF < 45% [54, 55]. Although PPCM is a common cause of left ventricular failure in pregnancy, it is a diagnosis of exclusion, and other reasons for heart failure should be ruled out [37]. These include, but are not limited to, DCM, left ventricular noncompaction, chronic heart failure, arrhythmogenic causes of heart failure, and heart failure from acute coronary syndromes. Notably, up to 22% of individuals with PPCM have co-existing preeclampsia [56, 57]. Management principles for PPCM are the same as those for other etiologies of left ventricular failure.

Animal studies and limited human trials suggest that bromocriptine may benefit left ventricular recovery in PPCM, but this has not been confirmed in larger trials [58]. Bromocriptine is recommended as an addition to standard therapy for PPCM in Europe. Although it is not currently approved by the US Food and Drug Administration (FDA) for this indication in the United States, there is a multicenter study underway to evaluate the benefit of bromocriptine in PPCM [59].

Generally, pregnancy is considered contraindicated in patients with a history of PPCM with residual left ventricular dysfunction (EF < 45%) [60]. Left ventricular dysfunction should be evaluated in the context of (1) the overall health of the patient (i.e., NYHA functional class); (2) prior PPCM history (e.g., severely depressed EF, use of mechanical circulatory support); (3) medications needed to maintain current EF; and (4) the ability to access and receive comprehensive cardiac care [60]. Although PPCM is a diagnosis of exclusion, Ware et al. found that 15% of people diagnosed with PPCM had truncating variants (two-thirds of which were in *TTN*), a prevalence similar to that observed in a population with DCM, a lifelong condition [61]. In another study that included three families with cases of both PPCM and DCM, there were low rates of full recovery of left ventricular function following PPCM (10%). Among this population, 22% of families had pathogenic mutations in cardiomyopathy-related genes (e.g., *TTN*), and 33% had variants of unknown significance, often also in the *TTN* gene [62]. This suggests that a genetic etiology may be present for patients without left ventricular function recovery and/or with a family history of PPCM or DCM. **We recommend considering referral to a genetics provider with expertise in heritable cardiac disease for people with PPCM, particularly when the index of suspicion is high and no other contributing factors are identified (GRADE 1C). We recommend that other causes of heart failure be ruled out before making a diagnosis of PPCM (Best Practice)**.

2.10 | What are the main causes of *chronic* left ventricular failure in pregnancy?

Common causes of chronic left ventricular failure in pregnancy are outlined in the Box. Hypertrophic cardiomyopathy (HCM) is characterized by left ventricular hypertrophy in the absence of another etiology for cardiac hypertrophy [63]. Typically, there is asymmetric thickening of the myocardium ≥ 15 mm, which may lead to left ventricular outflow obstruction, diastolic dysfunction, ischemia, and mitral regurgitation. There is an increased risk of arrhythmias and sudden death [64]. Most patients with HCM tolerate pregnancy well [65] due to the associated volume expansion [66].

2.11 | How is left ventricular failure diagnosed during pregnancy?

Pregnant patients with new decompensated heart failure typically present with shortness of breath and cough, with or without chest pain, as these are some of the symptoms associated with low cardiac output and pulmonary edema [67]. Pregnant patients with preexisting cardiomyopathy may or may not have a diagnosis before pregnancy. Patients without a previous diagnosis typically decompensate during pregnancy, unmasking the underlying ventricular failure.

Evaluation begins with careful history and physical examination, followed by laboratory testing. Assessment should focus on signs of decompensation, such as weight gain, jugular venous distension, tachycardia, crackles, S3 or S4 heart sounds, murmurs, pedal edema, and functional capacity (i.e., evaluation of NYHA functional class) [68]. Initial diagnostic testing includes electrocardiography and echocardiography. However, cardiac magnetic resonance imaging (MRI) may be considered for patients with congenital heart disease and/or right ventricular dysfunction. For patients with unclear etiologies of heart failure disease, cardiac MRI may help delineate ischemic versus non-ischemic cardiomyopathies and describe myocardial perfusion. It also may help delineate diseases like left ventricular noncompaction in cases where heart failure etiology is not clear [69]. Pertinent laboratory tests include BNP [18], cardiac enzymes (troponins), electrolytes, renal function, and complete blood count.

2.12 | How should acute and chronic left ventricular failure be managed during pregnancy?

System-specific workflows likely exist at each institution to treat and manage cardiac disease in pregnancy. Some institutions have designated pregnancy heart teams, and others may have institutional experts [70, 71]. In the setting of acute congestive heart failure, both maternal-fetal medicine subspecialists and cardiologists should feel confident initiating the initial workup (electrocardiogram, echocardiography, imaging, cardiac biomarkers) and management, including afterload reduction and diuresis. Advanced heart failure specialists or institutional experts may be required for continuation of care, outpatient management, shock, nuanced cases, and cases where referral for surgical or procedural subspecialists is needed.

2.12.1 | Acute left ventricular heart failure

The management of acute left ventricular heart failure is deeply rooted in the etiology of the heart failure itself, and there are some key differences from chronic left ventricular heart failure. The mainstays of treatment for acute left ventricular heart failure are afterload reduction and achieving euvolemia (diuresis if indicated), as many patients are hypervolemic [1]. During pregnancy, afterload reduction is most suitably achieved with hydralazine or isosorbide dinitrate [72] given the restrictions on first-line agents [angiotensin-converting enzyme inhibitor (ACEi), angiotensin receptor blocker (ARB), angiotensin receptor/neprilysin inhibitor (ARNi) [1]] due to known or suspected fetal teratogenicity [73]. Although nifedipine and amlodipine are dihydropyridines and mainly cause vasodilation with minimal effects on chronotropy and inotropy, their use is controversial in the setting of acute heart failure and they are not recommended for heart failure management in pregnancy. For diuresis, loop diuretics such as furosemide can be used safely [74, 75]. In the postpartum period, afterload reduction can be achieved with the above-mentioned first-line agents. Loop diuretics can be continued postpartum, and diuresis can be augmented with spironolactone during this time. Diuresis should be employed until signs of volume overload abate and/or until euvolemia is achieved. In the acute setting, chronotropic and inotropic agents are not advised except for acute coronary syndrome because they can increase ischemia [1, 72]. **For acute left ventricular heart failure during pregnancy, we recommend hydralazine or isosorbide dinitrate for afterload reduction and furosemide for diuresis. For acute left ventricular heart failure postpartum, we recommend afterload reduction with ACEi, ARB, or ARNi unless contraindicated (e.g., renal failure) (GRADE 1B)**.

Identifying the etiology of acute left ventricular failure is as important as initiating treatment. Etiologies for acute left ventricular failure in pregnancy include PPCM, cardiomyopathy secondary to acute myocardial infarction, and heart failure in the setting of hypertensive emergency or increased afterload (i.e., preeclampsia). It is critical to consult with an advanced heart failure and/or cardiology specialist to help delineate a specific etiology of disease. Notably, beta-blockers are typically avoided in patients with acute decompensated heart failure, which may have implications for hypertension management in the setting of preeclampsia. In the inpatient setting, prophylactic anticoagulation, with heparin or low molecular weight heparin, is advised for patients with acute left ventricular failure (particularly for EF $< 30\%$) [1, 76–79]. Whether to continue anticoagulation on discharge is an individualized decision based on the patient's risk and shared decision-making. If outpatient prophylaxis is prescribed, the duration and dosage should be determined with input from the multidisciplinary care team. The data to guide the use of anticoagulants during pregnancy and postpartum in acute heart failure are limited [80]. In the postpartum period, such decisions may be individualized depending on the presence of risk factors for venous thromboembolism. **We recommend against inotropic blockade**

TABLE 5 Management of acute and chronic left ventricular failure [83].

	Acute left ventricular heart failure	Chronic left ventricular heart failure
Experts	Cardiologist, cardiac surgeon (MCS), intensivist, maternal-fetal medicine subspecialist, cardiac anesthesiologist, obstetric anesthesiologist	Maternal-fetal medicine subspecialist, cardiologist, heart failure specialist, obstetric anesthesiologist
Resources	Intensive care unit (sub-specialized in cardiac care preferred), MCS capabilities	Outpatient imaging capabilities
Medications	Afterload reduction: hydralazine, nitroprusside Diuresis: furosemide, bumetanide Inotropy: dobutamine, epinephrine	Afterload reduction: hydralazine, isosorbide dinitrate Beta-blockade: metoprolol, carvedilol, bisoprolol Diuresis: furosemide **All other GDMT agents (ACEi, ANRi, mineralocorticoid antagonists) are contraindicated during pregnancy**
Anticoagulation	Mechanical or pharmacological thromboprophylaxis	Consider if EF < 35%
Fetal monitoring	At least daily if the fetus is considered viable.	Individualized

Abbreviations: ACEi, angiotensin-converting enzyme inhibitors; ANRi, angiotensin receptor-neprilysin inhibitor; EF, ejection fraction; GDMT, guideline-directed medical therapy; MCS, mechanical circulatory support.

(i.e., beta-blockers) in the setting of acute decompensated left ventricular heart failure (GRADE 1B). We recommend prophylactic anticoagulation administration in hospitalized pregnant patients with acute left ventricular heart failure (GRADE 1C).

It is important to acknowledge that pregnant patients can experience HFpEF, previously called diastolic dysfunction [81, 82]. Although HFpEF is a major contributor to cardiac morbidity and mortality, there exists a large knowledge gap about its pathology and natural history in pregnancy, and it is thus beyond the scope of this document. If HFpEF is suspected, expert consultation should be considered.

2.12.2 | Acute decompensated left ventricular failure in pregnancy

Severe decompensated heart failure is a rare event in pregnancy and postpartum but requires swift multidisciplinary action when detected. It is characterized by an often rapid onset of fluid overload, contributing to the heart's inability to deliver oxygenated blood to meet the body's metabolic demands [83]. The cornerstones of therapy in decompensated heart failure are afterload reduction and diuresis, with inotropy and/or vasopressor as needed (Table 5). Pregnant patients with acute heart failure should have monitoring that includes continuous heart rate measurement, pulse oximetry, telemetry, frequent blood pressure assessment, and strict urine output measurements. Frequent cardiac imaging may also be needed. Ultimately, maternal monitoring depends on the severity of the heart failure and the interventions required, such as whether vasopressor and inotropic support are needed or whether the clinical scenario necessitates mechanical circulatory support. To accomplish the necessary medical interventions and monitoring, pregnant patients may require transfer to a higher level of care unit or center, particularly one with heart failure specialists, intensivists, maternal-fetal medicine subspecialists, and obstetric and cardiac anesthesiologists [71, 84].

Patients should be monitored for volume status and signs of pulmonary edema to ensure adequate oxygenation [67]. All patients should have daily weights to monitor their response to diuresis. Fluid intake and output are closely followed with daily or more frequent electrolyte measurements to guide replacement, particularly for potassium and magnesium, as aggressive diuresis may impact renal function and lower blood pressure. Cardiac telemetry monitoring, transthoracic echocardiography, blood pressure monitoring, hourly fluid status, pulse oximetry, and thromboprophylaxis should be initiated in all pregnant individuals with decompensated acute heart failure. Transfer to a center with additional expertise in caring for pregnant patients with heart disease is recommended.

2.12.3 | Chronic left ventricular failure in pregnancy

Thankfully, chronic or long-standing left ventricular failure is still a rarity in pregnancy. Patients with chronic left ventricular systolic failure should first be assessed for the risk of adverse outcomes during pregnancy; in those already pregnant, this discussion should involve the risks associated with continuing pregnancy. Avoidance of pregnancy is strongly recommended in patients with EF < 30%

[84]. If patients choose to pursue or continue pregnancy, a thorough medical history and review of medications should be performed. Although medical societies advocate for the use of guideline-directed medical therapy (GDMT) soon after discharge in left ventricular failure, the initiation of outpatient GDMT for heart failure remains low [85]. GDMT includes ACEi/ARB, beta-blockade, and mineralocorticoid receptor antagonist (i.e., spironolactone) [86]. Timely initiation of these medications reduces worsening heart failure within one year and decreases mortality [87], with 90% survival at one year in optimally treated patients [88]. For people with chronic heart failure who are pursuing pregnancy or are currently pregnant, contraindicated GDMT medications such as ACEi, ARB, ARNi, and spironolactone should be discontinued and replaced with alternatives, such as hydralazine. Beta-blockers should be continued, and the recommended agents in this patient population are metoprolol, carvedilol, and bisoprolol [1]. All three of these medications have adequate safety profiles, and it is not advised to switch to labetalol, despite long-term comfort using labetalol during pregnancy. Outpatient anticoagulation in chronic left ventricular failure is controversial and should be individualized. Outside of pregnancy, anticoagulation is not needed for chronic left ventricular failure alone; in the setting of EF < 35% in pregnancy and postpartum, however, prophylaxis is reasonable [1, 80]. In the inpatient setting, prophylactic anticoagulation, with heparin or low molecular weight heparin, should be considered for all pregnant individuals with heart failure. **In patients who are pursuing pregnancy or are pregnant, we recommend discontinuing spironolactone, ACEi, ARB, and ARNi and continuing beta-blockers (metoprolol, carvedilol, bisoprolol) (GRADE 1C)** [32, 89, 90]**. For pregnant patients with left ventricular failure and EF < 35%, we recommend pharmacologic thromboprophylaxis during pregnancy and for six weeks postpartum (GRADE 1C)** [76–79]**. For patients with chronic left ventricular failure, we recommend starting or continuing GDMT therapy when medically able, in consultation with experts in cardiology (GRADE 1C)**.

2.12.4 | Refractory heart failure

Pregnant or postpartum patients with heart failure refractory to standard treatment are candidates for intravenous inotropic therapy, left ventricular assist device (LVAD), extracorporeal membranous oxygenation, and cardiac transplantation. There are reports of LVAD use in pregnancy; however, complications include both thromboembolism and increased bleeding [91]. Cardiac transplantation is a last resort in patients who have exhausted all possible interventions. Any of these interventions will require transfer to a high level of care and, in most cases, will necessitate abortion care or delivery of the fetus, depending on the gestational age and fetal status. Preparations for a preterm delivery may be necessary, including administering antenatal corticosteroids [92–94] and consultation with neonatology as appropriate. Cases of refractory left ventricular heart failure during pregnancy should be managed at a center with expertise in caring for pregnant patients with heart disease and with the appropriate subspecialist support.

2.12.5 | Arrhythmia and heart failure

Sustained cardiac arrhythmias can cause heart failure. Arrhythmias can arise from underlying cardiovascular complications or independently co-exist in acute and chronic heart failure. The most commonly encountered arrhythmias are supraventricular tachycardia (SVT), including atrial fibrillation and atrioventricular nodal re-entry tachycardia (AVNRT), and ventricular tachycardia [95]. Treatment of SVT in heart failure involves nodal blocking agents (e.g., beta-blockers, calcium channel blockers) and diuresis, depending on the volume status, which is usually presumed to be overloaded in acute heart failure. Sodium channel blockers (e.g., procainamide, lidocaine) and potassium channel blockers (e.g., sotalol) can also be used under expert guidance. Electrical cardioversion should be done under the supervision of a heart specialist and/or critical care provider. Anticoagulation during medical and/or electrical cardioversion should be discussed with a multidisciplinary team, and bleeding risks should be assessed [95].

Ventricular arrhythmias, specifically ventricular tachycardia, can lead to sudden cardiac death in patients with heart failure. Ventricular arrhythmias occur at a much higher rate in the setting of reduced EF secondary to myocardial fibrosis, abnormal repolarization, subendocardial ischemia, and ventricular (and atrial) dilation. Anti-arrhythmogenic drugs such as amiodarone and lidocaine can be used in the acute setting with the addition of other blockade agents with expert advice. Electrical cardioversion has a larger role in ventricular arrhythmias, and implantable cardioverter-defibrillators (ICDs) have revolutionized care in the outpatient setting [95]. Medical and procedural care for SVT and ventricular arrhythmias in pregnant patients with heart failure is incredibly nuanced. In these situations, it is imperative to involve heart failure experts and/or electrophysiology specialists when available.

2.13 | What fetal considerations are relevant in pregnancies complicated by maternal heart failure?

Pregnant people with cardiac disease, including heart failure, are at increased risk for adverse perinatal outcomes, including small for gestational age (SGA) birth, lower Apgar scores, and prematurity [10–12]. A first-trimester ultrasound facilitates accurate pregnancy dating and identification of a multifetal gestation, if present [96]. Patients with heart failure are candidates for routine prenatal aneuploidy screening and diagnostic testing. In the case of maternal heart failure resulting from an underlying congenital defect, a fetal echocardiogram is indicated, given the increased risk for congenital heart disease in the fetus [32, 97, 98]. **We recommend fetal echocardiography when maternal heart failure is a result of an underlying congenital cardiac defect (GRADE 1C)** [99].

Several medications in GDMT cannot be used in pregnant or lactating people. ACEi, ARB, aldosterone antagonists, and sodium-glucose cotransporter inhibitors (SGLT2i) are contraindicated in pregnancy due to known or suspected fetal teratogenicity [100–102]. Beta-blockers (e.g., metoprolol, carvedilol, bisoprolol) are standard treatments employed in patients with heart failure and can safely be continued, although they may increase the risk for hypotension, hypoglycemia, bradycardia, and respiratory depression in neonates [32]. A large systematic review from 2014 (49 trials with 4723 participants) of pregnant individuals treated with antihypertensive agents (including beta-blockers) noted no difference in the rates of SGA births. Two additional randomized controlled trials investigating hypertension treatment in pregnancy (3395 pregnant individuals) that included labetalol use found no difference in the rates of SGA births with treatment [103, 104]. Notably, most well-designed studies have not included metoprolol or carvedilol, and some observational studies suggest a possible increase in the rates of SGA births [105, 106]. Given the increased risk for fetal growth restriction and SGA births among pregnant people with heart failure and the possible association with medications used, serial growth ultrasounds should be performed, and antepartum fetal surveillance should be instituted as indicated [107–109]. **We recommend serial growth ultrasounds in pregnancies complicated by maternal heart failure (GRADE 1C)** [110, 111].

Continuous fetal monitoring should be used for appropriate assessment of the fetus during the cardiac challenges of regional or general anesthesia administration, labor, and delivery. Aside from intrapartum management, continuous or intermittent fetal heart monitoring should be considered when changes in the maternal status prompt inpatient evaluation or treatment. This decision should consider gestational age and any relevant maternal or fetal factors that may impact fetal viability or the maternal clinical status. **We recommend continuous fetal heart rate monitoring during anesthesia administration, labor, and delivery for pregnant patients with heart failure (GRADE 1B)** [112–114]. **In the case of maternal cardiovascular changes prompting inpatient assessment or treatment, we recommend continuous or intermittent fetal heart rate monitoring, taking into consideration the gestational age and any relevant maternal or fetal factors that may impact fetal viability or maternal clinical status (GRADE 1C)** [110–114].

2.14 | How are pregnant patients with heart failure managed around the time of delivery?

Delivery planning considerations in patients with heart failure include timing and mode of delivery, laboratory testing, monitoring (e.g., pulse oximetry, telemetry, arterial line, central line), type of anesthesia, thromboprophylaxis, fluid management, endocarditis prophylaxis, and location of postpartum recovery. Planning starts with identifying the care team, including members from obstetrics, maternal-fetal medicine, cardiology, obstetric anesthesiology, cardiac anesthesiology (if needed), nursing, and other disciplines deemed appropriate.

Delivery timing should be based on cardiac stability and obstetric indications. There are no clinical studies on which to base delivery timing recommendations in this specific population. A term delivery should be pursued in an otherwise uncomplicated patient who is well compensated. In some cases, an early-term delivery may be appropriate based on the overall clinical considerations.

Vaginal delivery is generally preferred in patients with ventricular failure unless avoidance of labor and expeditious delivery is in the best interest of the patient due to hemodynamic compromise. Cesarean delivery is reserved for typical obstetric indications; compared to vaginal delivery, it is associated with increased likelihood of blood loss, general anesthesia, thromboembolism, infection, and bleeding complications, particularly for those on anticoagulation [115]. In some patients, labor may be well tolerated, particularly with the aid of neuraxial anesthesia, but there may be reasons to avoid Valsalva, or prolonged Valsalva. Although the Valsalva maneuver is associated with substantial hemodynamic alterations [116] (sudden rise and then fall in systolic blood pressure, decrease in venous return, heart rate fluctuations), many pregnant individuals with cardiac disease can tolerate Valsalva with or without operative vaginal delivery [117]. Instances in which to limit or avoid Valsalva during the second stage may include some cases of severe pulmonary hypertension, left ventricular outflow tract obstruction, compromised venous return, or substantially compromised myocardial contractility. In these cases, an assisted second stage with an operative delivery can be considered with insight from a heart failure specialist about each individual patient's cardiac risk.

In most cardiac patients, neuraxial analgesia and anesthesia are considered safe and may be desirable to limit fluctuations in cardiac output associated with catecholamine surges and to facilitate rapid obstetric interventions (such as cesarean delivery) if needed [118]. For patients at risk for arrhythmias in the peripartum period, telemetry is often utilized. An arterial line should be considered in patients who may benefit from continuous blood pressure and cardiac output monitoring during the peripartum period (e.g., critical aortic stenosis, previous PPCM with unrecovered function) [118]. Any obstetric patient is at risk for hemorrhage. No uterotonic is absolutely contraindicated for use in patients with heart failure but some carry additional cardiovascular risk. For example, ergot alkaloids (e.g., methergine) induce vasoconstriction and may cause increased afterload and cardiac ischemia [119]. Obstetric hemorrhage should be managed per unit protocols with consideration of the potential effect of each uterotonic based on individual patient factors. **We recommend planned vaginal delivery at term in patients with heart failure in the absence of hemodynamic compromise or obstetric indications for cesarean (GRADE 1C). We recommend the use of neuraxial anesthesia in most patients with heart failure to provide appropriate analgesia and to limit the effects of labor on cardiac parameters (GRADE 1C). We recommend considering a limited or assisted second stage for some patients after input from cardiology about each patient's cardiac risk (GRADE 1C)**. Pregnant patients at risk for requiring mechanical circulatory support in the peripartum period should be delivered at an appropriately resourced tertiary care center.

2.15 | How should patients with heart failure be managed in the immediate postpartum period?

Postpartum patients with heart failure require close monitoring due to substantial and rapid changes in the cardiovascular system during this time [120, 121]. The largest change in intravascular volume occurs during the second and third stages of labor and immediately postpartum, placing patients at risk for volume overload and arrhythmia exacerbation. Risk for compromise in the postpartum period largely depends on the etiology of heart failure, maternal status prior to delivery, and delivery events. Importantly, many maternal cardiovascular deaths and severe morbidity occur after discharge from the delivery hospitalization [122–124]. For this reason, efficient bridging of care is critical to aid in a safe maternal transition to cardiac care in the postpartum period. Postpartum patients can be transitioned to medications they could not take antenatally (i.e., ACEi, ARB, and mineralocorticoid receptor antagonists). Close maternal monitoring is needed in the immediate postpartum period with an individualized inpatient hospitalization plan for each patient; some patients may require monitoring for more than 48 hours after delivery [125]. Strong partnerships with the appropriate care teams can promote a safe transition to cardiac care, particularly after the immediate postpartum period.

2.16 | What are considerations for breastfeeding in postpartum people with heart failure?

Pregnant individuals with heart failure should receive routine counseling about infant feeding. Each patient's medication should be reviewed to confirm compatibility with breastfeeding (sometimes called chestfeeding). In the absence of robust data to guide counseling, shared decision-making should be pursued, taking into account the benefits of breastfeeding and possible risks [126, 127]. Notably, ACEi agents are regarded as safe for breastfeeding and should not be withheld in this setting because they represent an essential aspect of GDMT therapy [128–130]. Patients requiring therapeutic anticoagulation in the postpartum period should be made aware that warfarin is compatible with breastfeeding and poses no risk to the infant [131]. Direct oral anticoagulants (DOACs), however, are not recommended while breastfeeding due to insufficient safety data and the availability of effective alternatives. There may be theoretical concerns related to decreased breast milk production with diuretic use; however, the benefits of prompt initiation of GDMT outweigh these concerns. Currently, SGLT2i agents are not recommended for breastfeeding as animal studies have shown excretion into breastmilk [132]. **We recommend that postpartum patients with heart failure undergo routine counseling regarding infant feeding. We recommend reviewing all medications for compatibility with breastfeeding and using shared decision-making in the absence of robust data (GRADE 1B)**.

Summary of recommendations[a]

Number	Recommendation	GRADE
1	We recommend that all patients with right heart failure due to pulmonary arterial hypertension receive counseling about high rates of maternal morbidity and mortality; if pregnancy is pursued, the patient should be referred to a center with expertise in this condition to guide management during pregnancy and postpartum.	1C
2	We recommend considering referral to a genetics provider with expertise in heritable cardiac disease for people with PPCM, particularly when the index of suspicion is high and no other contributing factors are identified.	1C
3	We recommend that other causes of heart failure be ruled out before making a diagnosis of PPCM.	Best Practice
4	For acute left ventricular heart failure during pregnancy, we recommend hydralazine or isosorbide dinitrate for afterload reduction and furosemide for diuresis. For acute left ventricular heart failure postpartum, we recommend afterload reduction with ACEi, ARB, or ARNi unless contraindicated (e.g., renal failure).	1B
5	We recommend against inotropic blockade (i.e., beta-blockers) in the setting of acute decompensated left ventricular heart failure.	1B
6	We recommend prophylactic anticoagulation administration in hospitalized pregnant patients with acute left ventricular heart failure.	1C
7	In patients who are pursuing pregnancy or pregnant, we recommend discontinuing spironolactone, ACEi, ARB, and ARNi and continuing beta-blockers (metoprolol, carvedilol, bisoprolol).	1C
8	For pregnant patients with left ventricular failure and EF $< 35\%$, we recommend pharmacologic thromboprophylaxis during pregnancy and for six weeks postpartum.	1C
9	For patients with chronic left ventricular failure, we recommend starting or continuing GDMT therapy when medically able, in consultation with experts in cardiology.	1C
10	We recommend fetal echocardiography when maternal heart failure is a result of an underlying congenital cardiac defect.	1C
11	We recommend serial growth ultrasounds in pregnancies complicated by maternal heart failure.	1C
12	We recommend continuous fetal heart rate monitoring during anesthesia administration, labor, and delivery for pregnant patients with heart failure.	1B
13	In the case of maternal cardiovascular changes prompting inpatient assessment or treatment, we recommend continuous or intermittent fetal heart rate monitoring, taking into consideration the gestational age and any relevant maternal or fetal factors that may impact fetal viability or the maternal clinical status.	1C
14	We recommend planned vaginal delivery at term in patients with heart failure in the absence of hemodynamic compromise or obstetric indications for cesarean.	1C
15	We recommend the use of neuraxial anesthesia in most patients with heart failure to provide appropriate analgesia and to limit the effects of labor on cardiac parameters.	1C
16	We recommend considering a limited or assisted second stage for some patients after input from cardiology about each individual patient's cardiac risk.	1C
17	We recommend that postpartum patients with heart failure undergo routine counseling regarding infant feeding. We recommend reviewing all medications for compatibility with breastfeeding and using shared decision-making in the absence of robust data.	1B

[a]See "Supporting Information" for evidence summary table.

Society for Maternal-Fetal Medicine Grading of recommendations assessment, development, and evaluation (GRADE) system [133][a]

Grade of Recommendation	Clarity of Risk and Benefit	Quality of Supporting Evidence	Implications
1A. Strong recommendation, high-quality evidence	Benefits clearly outweigh risks and burdens, or vice versa.	Consistent evidence from well-performed, randomized controlled trials, or overwhelming evidence of some other form. Further research is unlikely to change confidence in the estimate of benefit and risk.	Strong recommendation that can apply to most patients in most circumstances without reservation. Clinicians should follow a strong recommendation unless a clear and compelling rationale for an alternative approach is present.
1B. Strong recommendation, moderate-quality evidence	Benefits clearly outweigh risks and burdens, or vice versa.	Evidence from randomized controlled trials with important limitations (inconsistent results, methodologic flaws, indirect or imprecise), or very strong evidence of some other research design. Further research (if performed) is likely to have an impact on confidence in the estimate of benefit and risk and may change the estimate.	Strong recommendation that applies to most patients. Clinicians should follow a strong recommendation unless a clear and compelling rationale for an alternative approach is present.
1C. Strong recommendation, low-quality evidence	Benefits appear to outweigh risks and burdens, or vice versa.	Evidence from observational studies, unsystematic clinical experience, or randomized controlled trials with serious flaws. Any estimate of effect is uncertain.	Strong recommendation that applies to most patients. Some of the evidence base supporting the recommendation is, however, of low quality.
2A. Weak recommendation, high-quality evidence	Benefits closely balanced with risks and burdens.	Consistent evidence from well-performed randomized controlled trials or overwhelming evidence of some other form. Further research is unlikely to change confidence in the estimate of benefit and risk.	Weak recommendation; best action may differ depending on circumstances or patients or societal values.
2B. Weak recommendation, moderate-quality evidence	Benefits closely balanced with risks and burdens; some uncertainty in the estimates of benefits, risks, and burdens.	Evidence from randomized controlled trials with important limitations (inconsistent results, methodologic flaws, indirect or imprecise), or very strong evidence of some other research design. Further research (if performed) is likely to influence confidence in the estimate of benefit and risk and may change the estimate.	Weak recommendation; alternative approaches likely to be better for some patients under some circumstances.
2C. Weak recommendation, low-quality evidence	Uncertainty in the estimates of benefits, risks, and burdens; benefits may be closely balanced with risks and burdens.	Evidence from observational studies, unsystematic clinical experience, or randomized controlled trials with serious flaws. Any estimate of effect is uncertain.	Very weak recommendation, other alternatives may be equally reasonable.
Best practice	Recommendation in which either (i) there is an enormous amount of indirect evidence that clearly justifies strong recommendation (direct evidence would be challenging, and inefficient use of time and resources, to bring together and carefully summarize) or (ii) recommendation to the contrary would be unethical.		

[a] Adapted from Guyatt et al [134].

Guidelines referenced

Organization	Title	Year of Publication
American College of Obstetricians and Gynecologists	ACOG Practice Bulletin No. 227: Fetal Growth Restriction: [107]	2021
American Heart Association, American College of Cardiology, Heart Failure Society of America	2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines [1]	2021
American Heart Association	Use of Medication for Cardiovascular Disease During Pregnancy: JACC State-of-the-Art Review [89]	2022
American Institute of Ultrasound in Medicine	AIUM practice guideline for the performance of fetal echocardiography [98]	2011
European Society of Cardiology	2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy [32]	2018
National Heart, Lung, and Blood Institute and Office of Rare Diseases	Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review [54]	2000

2.17 | Which contraceptive options may be used in patients with heart failure?

Individualized, patient-centered reproductive planning is essential for patients with heart failure because of the increased maternal and fetal morbidity and mortality in this population [10–12]. Potential adverse effects of contraceptive options, including fluid retention, hypertension, and thromboembolic risk, should be taken into consideration. If cesarean delivery is planned and future fertility is not desired, patients should be counseled about the option for concurrent permanent sterilization. The 2024 US Medical Eligibility Criteria for Contraceptive Use [135] lists the implant and progestin-only pill as category 1 (no restrictions for use) for patients with a history of PPCM who have NYHA class I or II functional status. Intrauterine devices (both copper and levonorgestrel) and depot-medroxyprogesterone acetate are listed as Category 2 (method generally can be used, although careful follow-up might be required). Combined hormonal contraceptives, however, are listed as Category 4 (unacceptable health risk if the method is used) in the first 6 months following PPCM and Category 3 (requires careful clinical judgment and access to clinical services) after 6 months. These recommendations are due to the fluid retention associated with combined hormonal contraceptives and the possible increased risk for arrhythmias. For patients with a history of PPCM who have NYHA class III or IV functional status, the implant, intrauterine devices, and progestin-only pills are Category 2, depot-medroxyprogesterone acetate is Category 3, and combined hormonal contraceptives are Category 4. Although recommendations are not listed for other etiologies of heart failure, it is reasonable to use these recommendations as guidance. For patients with heart failure who desire future fertility, comprehensive contraceptive counseling should take into consideration medical criteria and patient preferences. For patients with heart failure interested in permanent methods, surgical sterilization should be considered.

2.18 | What are the known health disparities in right and left ventricular failure during pregnancy?

Among pregnant and non-pregnant adults with heart failure, health disparities by patient race and socioeconomic status are well-documented [136–140]. Providers' practice differences attributed to implicit bias are also reported, affecting procedural and medical management (i.e., GDMT) [141]. Among pregnant patients, the data largely derive from cases of PPCM but demonstrate a similar pattern of worse clinical outcomes among those identifying as Black, with strong associations based on disadvantaged socioeconomic status [142–144]. Although disparities data are limited in the setting of pregnancy and heart failure, race and ethnicity likely affect diagnosis, treatment, and escalation of care. Providers and institutions should be vigilant and work towards reducing disparities and providing equitable care, for example, by standardly collecting accurate race, ethnicity, and language data in conjunction with other measures of social determinants of health [145].

3 | CONCLUSION

The management of pregnancies complicated by heart failure requires understanding the etiology and severity of the heart failure, as well as whether the heart failure is primarily right- or left-sided. In addition to maternal-fetal medicine and cardiology (sub)specialists, some particularly high-risk patients, such as those with heart failure due to pulmonary arterial hypertension or severely depressed systolic function, will likely require additional expertise specific to their conditions. Many of the medical cornerstones of heart failure management can be continued during pregnancy, with a few notable exceptions. Labor, delivery, and particularly postpartum represent times during which patients may be at high risk for decompensation, such that delivery at a center with experience in caring for these patients is recommended. Given the risk of complications even after discharge following delivery, a seamless transition to cardiac care is critical for these patients.

Although our understanding of heart failure in pregnancy continues to improve, research gaps remain. The management of PPCM, specifically whether bromocriptine is effective in the immediate time frame and whether GDMT is beneficial in the long term, is an active area of interest. Further knowledge gaps include the most effective imaging modalities to predict future cardiac health in those with heart failure and the impact of maternal heart failure on the cardiovascular health of offspring. Finally, the advance of cardiac genetics is likely to become an important component in the care of patients with heart failure, including in pregnancy.

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