CONTEMPORARY REVIEW

Maternal Heart Failure

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ABSTRACT: Heart failure (HF) remains the most common major cardiovascular complication arising in pregnancy and the postpartum period. Mothers who develop HF have been shown to experience an increased risk of death as well as a variety of adverse cardiac and obstetric outcomes. Recent studies have demonstrated that the risk to neonates is significant, with increased risks in perinatal morbidity and mortality, low Apgar scores, and prolonged neonatal intensive care unit stays. Information on the causal factors of HF can be used to predict risk and understand timing of onset, mortality, and morbidity. A variety of modifiable, nonmodifiable, and obstetric risk factors as well as comorbidities are known to increase a patient's likelihood of developing HF, and there are additional elements that are known to portend a poorer prognosis beyond the HF diagnosis. Multidisciplinary cardio-obstetric teams are becoming more prominent, and their existence will both benefit patients through direct care and increased awareness and educate clinicians and trainees on this patient population. Detection, access to care, insurance barriers to extended postpartum follow-up, and timely patient counseling are all areas where care for these women can be improved. Further data on maternal and fetal outcomes are necessary, with the formation of State Maternal Perinatal Quality Collaboratives paving the way for such advances.

Key Words: adverse neonatal outcomes ■ cardiomyopathy ■ heart failure ■ hypertensive disorders ■ pregnancy ■ pulmonary hypertension

Heart disease remains the leading cause of maternal morbidity and mortality in the United States and other developed countries[1] and has been on the rise over recent decades.[2] According to the Centers for Disease Control and Prevention's recent data from 2011 to 2016, cardiomyopathies and other cardiac conditions were responsible for 26.7% of pregnancy-related deaths, accounting for the greatest percentage of any cause.[3] The rise in maternal mortality is in part related to a rise in the prevalence of women with cardiac disease delivering babies, which has increased by ≈24% and complications in such pregnancies by 18% in the past decade.[4] The increase in prevalence of high-risk cardiac conditions in pregnancy from 2003 to 2012, such as cardiomyopathies, which account for a rise of 18%, as well as pulmonary hypertension (PH) is notable.[4] Both of these conditions are considered modified World Health Organization (mWHO) class III or IV pregnancy risk classifications, that is, pregnancy either lends a significantly elevated risk of maternal morbidity and mortality or is contraindicated altogether.

Heart failure (HF) has been identified as the underlying cause of over 9% of in-hospital deaths among pregnancy-related hospitalizations, with the rate of HF diagnoses in pregnancy increasing from 2001 to 2011, an issue of growing concern.[5] Notably, in expectant mothers with preexisting heart disease of any kind, whether from cardiomyopathies or congenital heart disease, HF remains the most common major cardiovascular complication arising in pregnancy complicating 11% of such pregnancies.[5–8] The number of high-risk pregnancies has grown dramatically from 0.7% in 2007 to 2010 to 10.9% in 2015 to 2018, with more women with preexisting medical complications, including heart disease, choosing motherhood.[8] As major cardiac complications associated with such deliveries increased by 18% during this time frame, we are urged to be continuously cognizant of this understudied issue.[4] Notably, cardiomyopathies represent

Nonstandard Abbreviations and Acronyms

ACHD	adult congenital heart disease
CO	cardiac output
MACE	major adverse cardiovascular events
mWHO	modified World Health Organization
PH	pulmonary hypertension
PPCM	peripartum cardiomyopathy

the largest group of women who present with HF, where the risk of cardiovascular adverse events approaches 50%.[9]

Maternal health can affect the well-being of both mothers and their children, 2 young populations whose health can massively alter their life's trajectory. Mothers who develop HF have been shown to experience an increased risk of death and a variety of adverse cardiac and obstetric outcomes. Additionally, recent findings have already begun to show that the risk to neonates is significant, including an increase in perinatal mortality, preterm birth, low Apgar scores, and prolonged neonatal intensive care unit stays.[10] Proper preconception counseling, provider education, and population-specific guidelines create opportunities to minimize the potential for negative outcomes in these populations. The majority of HF diagnoses are made in the postpartum period, with a considerable number of cases occurring as late as 5 to 12 months after delivery.[5,11–13] Among cardiovascular pregnancy-related deaths, 70% occurred within the first 6 weeks after delivery, and the chance of death occurring beyond 6 weeks postpartum was higher for women succumbing to cardiovascular than to noncardiovascular deaths.[14] This emphasizes the importance of access to follow-up care and to insurance coverage for mothers beyond 6 to 12 weeks that may or may not be available through most public options. As many women lose health coverage at this point, they are left vulnerable. A variety of social, demographic, and medical factors contribute to a woman's risk of developing HF in pregnancy and therefore we can use our understanding of these elements to provide better care to this deserving group of patients.

MATERNAL CARDIOVASCULAR PHYSIOLOGY

During pregnancy, anatomic and physiologic hemodynamic changes within the maternal cardiovascular system are orchestrated to support the health and safety of the mother and her fetus. Increases in plasma volume (leading to physiologic anemia) and maternal heart rate occur, and arterial blood pressure and systemic vascular resistance decrease. Cardiac output (CO) increases, beginning in early pregnancy beause of an increase in stroke volume, and is maintained further by way of tachycardia (Figure 1).[15–19] Twin pregnancies exhibit an additional 15% higher CO throughout pregnancy.[16] Increased left ventricular dimensions, aortocaval compression, and hypercoagulability also play a role in the dynamic alterations observed.[20]

As labor begins, additional factors amplify the divergence from nonpregnant cardiovascular physiology; uterine contractions force a large volume of blood away from the uterus into general circulation, raising preload and CO in turn, and pain and anxiety increase sympathetic tone, raising blood pressure, heart rate, and CO as well.[21,22] Within an hour of delivery, heart rate and CO return to prelabor levels,[23] although still elevated above baseline nonpregnant levels. Stroke volume, heart rate, and cardiac output decrease dramatically over the first 2 weeks postpartum. Within the first 6 weeks postpartum, there is a persistent shift in the balance of the autonomic nervous system and endothelial reactions as they aim to reapproximate the non-pregnant state.[16,17,24,25] Atrial natriuretic peptide and B-type natriuretic peptide (BNP) levels further increase postpartum, mediating diuresis after delivery.[18,19] The physiologic stress that these continuous adaptions create can both reveal previously undiagnosed cardiovascular disease in some and lead to development of new cardiac conditions in others, ultimately putting mothers at an increased risk of developing HF throughout their pregnancy course.

CAUSE AND TIMING OF HEART FAILURE

HF is the most common complication of cardiac disease in pregnancy, with prevalence reported from 13% in a large worldwide registry of women with preexisting cardiac disease[6] to ≈33% in women with preexisting cardiomyopathy.[9] HF prevalence among postpartum and antepartum hospitalizations have increased from 2001 to 2011[5] and the proportion of women diagnosed with peripartum cardiomyopathy has risen in the past decade.[26,27] Despite the poor prognosis associated with HF and pregnancy, the literature appears incomplete on the subject.

Unsurprisingly, HF is the most common complication among women with preexisting heart disease regardless of the cause, whether related to cardiomyopathy (new or preexisting), PH, or valvular, ischemic, or congenital heart disease (Figure 2). The largest group of women who present with HF are related to cardiomyopathy, where the risk of adverse cardiovascular
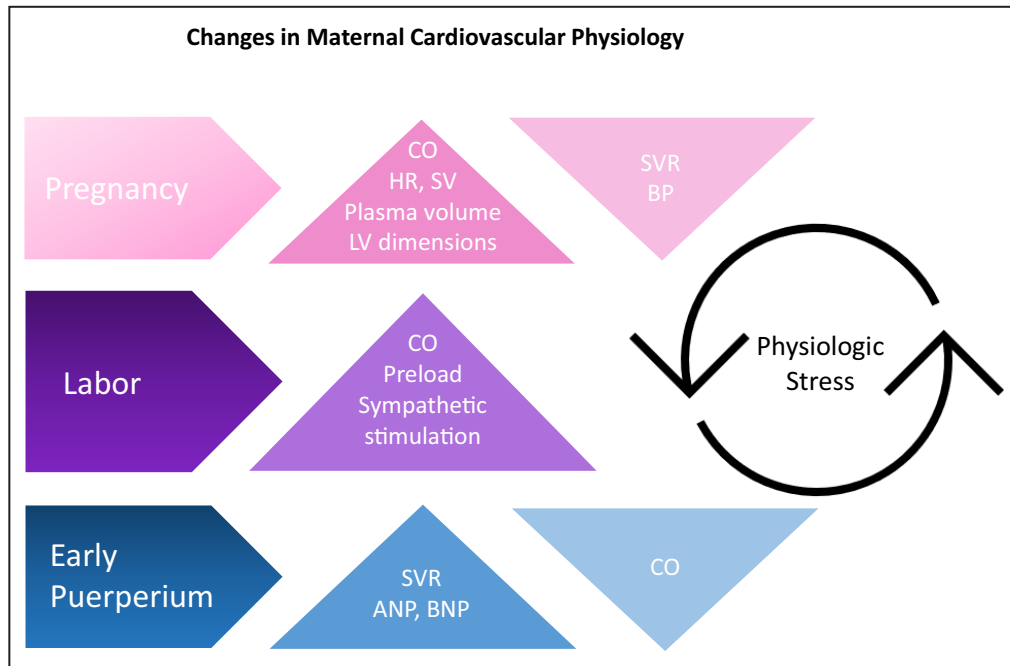
Figure 1.   Changes in maternal cardiovascular physiology.[15–19]
Upward arrows display physiologic increases and downward arrows display physiologic decreases.
ANP indicates atrial natriuretic peptide; BNP, B-type natriuretic peptide; BP, blood pressure; CO, cardiac output; HR, heart rate; SV, stroke volume; and SVR, systemic vascular resistance.

events during pregnancy approaches 50%.[9] Although peripartum cardiomyopathy (PPCM) may cause maternal HF more commonly,[9,10] mothers with known heart disease before pregnancy collectively make up the largest proportion of mothers with HF.[28,29] Moreover, women with HF more commonly have left-sided than right-sided (68% versus 45%) etiologies,[6] including left-sided valve disease or any form of cardiomyopathy (dilated, peripartum or hypertrophic).[9] Mothers with PH, right ventricular dysfunction, adult congenital heart disease (ACHD), or combinations thereof are also at increased risk.[30] Congenital heart disease in pregnancy management has been reviewed recently and review of this topic here is beyond the scope of this paper.[31]

Information regarding causal factors provides us with an opportunity to stratify risk and account for trends in HF incidence, morbidity, and mortality (Table 1).[6,9,10,28,29] Major adverse cardiovascular event (MACE) rates are higher in those with cardiomyopathy and PH and lower in those with ACHD.[28,29] PH is associated with the highest overall risk of complications, particularly when combined with cardiomyopathy or valvular disease.[8,30] In mothers developing HF who had preexisting cardiomyopathy, nonischemic forms followed by hypertrophic cardiomyopathy were more common than ischemic cardiomyopathy.[10] Further research is required regarding those with ischemic heart disease, but recent findings suggest that despite being a subset of older patients, outcomes for these mothers have been better in comparison to other groups.[8] Among pregnant women with valvular heart disease, over half had rheumatic heart disease, and 23% of mothers with mitral regurgitation, 31.8% with moderate mitral stenosis, and 48.1% with severe mitral stenosis developed HF.[6,8,32]
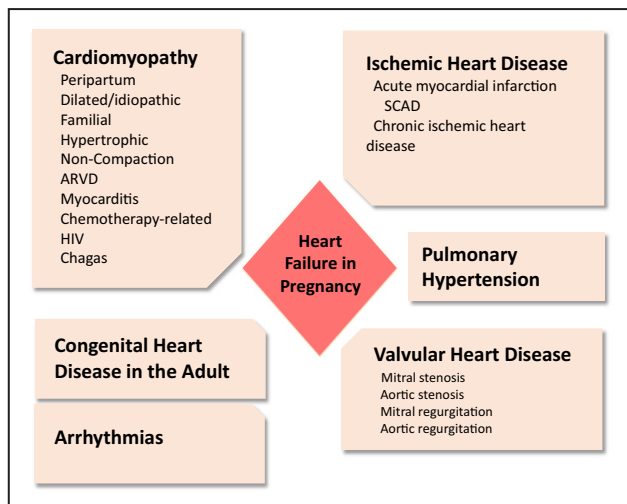

Figure 2. Major categories of potential etiologies of maternal heart failure.
Delineated are the major categorical etiologies that can result in mothers developing heart failure in pregnancy or the puerperium. ARVD indicates arrhythmogenic right ventricular dysplasia; and SCAD, spontaneous coronary artery dissection.

Table 1. Cause of Heart Failure

Reference	Data Set Used	Study Population	Major Etiological Findings
Ruys et al 2014[6]	The ROPAC (Registry on Pregnancy and Cardiac Disease)	Mothers with structural or ischemic heart disease	• 13% of mothers in the registry developed HF during pregnancy or after delivery. • More women with HF demonstrated to have a left-sided lesion, and fewer had a right-sided lesion or shunt lesion.
Lima et al 2015[9]	National Inpatient Sample obtained through the Healthcare Cost and Utilization Project	Mothers with CM (including PPCM)	• Of mothers with CM, 2.5% had hypertrophic CM, 50% had PPCM, and 47.5% other CM. • Presence of CM was independently predictive of MACE during delivery hospitalization. • Patients with PPCM had highest likelihood of MACE among CM subtypes, although dilated CM and hypertrophic CM followed.
Owens et al 2018[29]	Statewide Planning and Research Cooperative System from the New York State Department of Health	Mothers with heart disease	• 40% had VHD, 35% had ACHD, 17% had CM, and 8% had PH. • MACE occurred most frequently among those with CM or PH, least frequently among those with ACHD. • Obstetric complications more frequent in those with heart disease than those without.
Ng et al 2018[10]	Retrospective cohort from the Kaiser Permanente Southern California healthcare system	Mothers who develop HF in pregnancy	• Of mothers who developed HF, 68.2% had PPCM, 6.9% had nonischemic CM, 9.6% had ACHD, 3.5% had VHD, and 1% had ischemic CM.
Lima et al 2019[28]	Nationwide Readmission Database obtained through the Healthcare Cost and Utilization Project	Mothers with heart disease	• 46% had ACHD, 25% had VHD, 23% had CM, and 6% had PH. • Highest rates of MACE and obstetric complications occurred in mothers with CM or PH, lowest rate of MACE among mothers with ACHD. • Among CM subtypes, PPCM had the highest rates of index hospital MACE, obstetric complications, 42-d readmission, and postpartum MACE. • Preexisting heart disease was associated with over 4 times increased risk of postpartum MACE.

ACHD indicates adult congenital heart disease; CM, cardiomyopathy; HF, heart failure; MACE, major adverse cardiovascular events; PH, pulmonary hypertension; PPCM, peripartum cardiomyopathy; and VHD, valvular heart disease.

Though mothers with ACHD saw the best outcomes of those with known heart disease, findings were worse for those whose lesions remained uncorrected or who had shunt lesions present.[33]

Timing of HF is equally as important. Overall, 60% of HF cases occur in the postpartum period, followed by 27% at delivery, and 13% during pregnancy.[5] Timing of HF in mothers with known heart disease exhibits a bimodal distribution, with peaks at both 23 to 30 weeks and postpartum.[6] Patients with underlying valvular heart disease develop HF throughout pregnancy, whereas those with cardiomyopathy primarily experienced onset in the weeks before and following delivery.[6] Women with shunt lesions experienced an earlier onset of HF at ≈25 weeks gestation.[6]

PPCM Timing

Although PPCM can occur at any time in pregnancy, it most commonly occurs in the last trimester or within 6 weeks postpartum.[10,34] However, PPCM has also been described as late as 5 to 12-months after delivery.[12] Late onset was associated with a 2- to 3-fold increased risk of maternal mortality and MACE.[12,13]

RISK FACTORS

Multiple studies have established that demographic risk factors for the development of maternal HF include Black race, older age, tobacco use, alcohol use, drug use, and insurance under either Medicare or Medicaid[5,10,11] (Figure 3).[5,6,8,10,11] HF in pregnancy is more than twice as common in Black women compared with White women and is least common among Hispanic women.[10] Moreover, patients may have multiple cardiac diagnoses and/or medical or obstetric issues placing them at an additive risk. As patients are not siloed, a mother may have cardiomyopathy and coexisting PH or mitral stenosis and PH, placing her at an additional increased cardiac risk. Medical and obstetric risk factors play an important role in outcome. Risk factors for developing HF, among other MACE, that are uniquely relevant to women with known heart disease include prepregnancy HF, ejection fraction <40%, prepregnancy New York Heart Association class >II or mWHO class IV, cardiomyopathy, and PH.[6,8,35] The interplay between a variety of modifiable and nonmodifiable risk factors serves an additive effect, as exemplified in risk indexes such as CARPREG II (Cardiac Disease in Pregnancy Study) relating to maternal
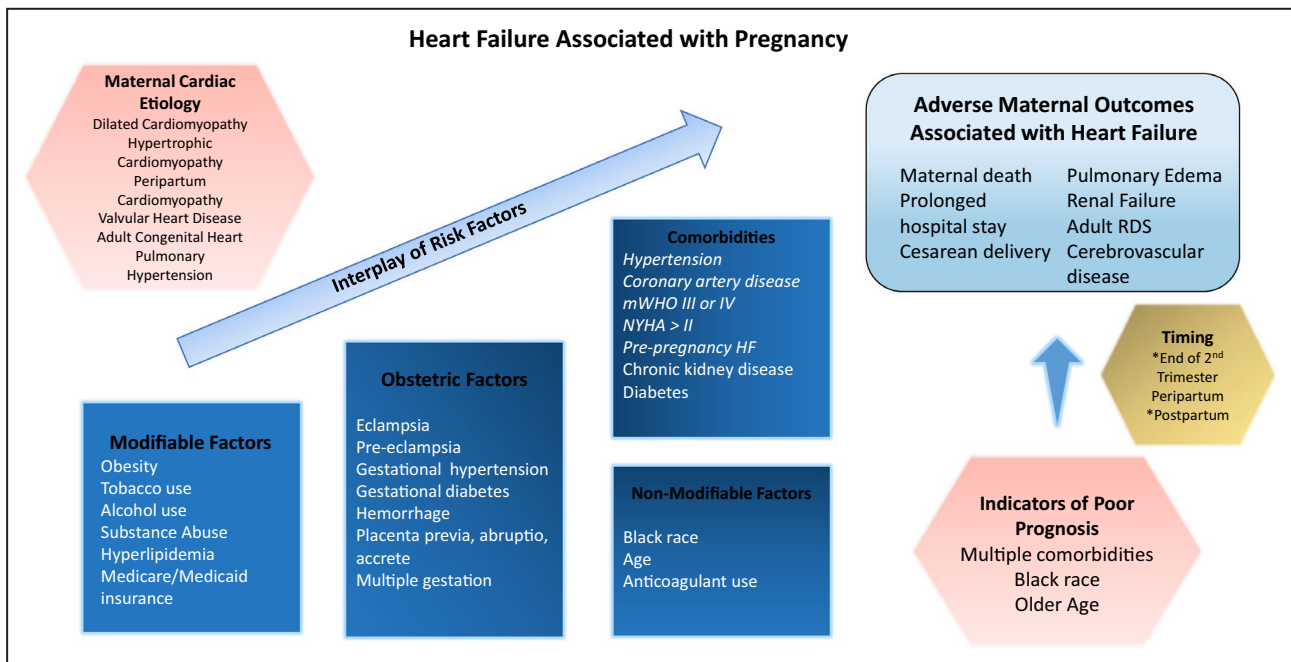
Figure 3. Heart failure associated with pregnancy.[5,6,8,10,11]
A variety of modifiable factors, nonmodifiable factors, obstetric factors, and comorbidities (cardiac comorbidities italicized) can contribute to a summative increase in risk of heart failure for pregnant women. Ultimately heart failure can result in adverse outcomes with a greater likelihood of such outcomes occurring among those with multiple comorbidities, Black race, and increased age. HF indicates heart failure; mWHO, modified World Health Organization; NYHA, New York Heart Association; and RDS, respiratory distress syndrome.

cardiac complications as a whole.[35] Notably, mWHO risk classification has been shown to be less effective in emerging/developing countries than advanced countries through data collected from the ROPAC (Registry on Pregnancy and Cardiac Disease).[36]

Obstetric risk factors for HF include multiple gestation, gestational or chronic hypertension, preeclampsia/eclampsia, postpartum hemorrhage, placenta accreta/abruptio/previa, and gestational diabetes mellitus.[5,10] Additionally, hyperlipidemia, preexisting diabetes mellitus, obesity, chronic kidney disease, and anticoagulant use increase the risk for developing HF.[5,10,11]

Hypertensive disorders of pregnancy deserve special mention, consisting of a spectrum of disorders from exacerbation of preexisting hypertension, hypertension in pregnancy, preeclampsia, eclampsia, and postpartum hypertension. In women with preexisting heart disease, the added strain of preeclampsia precipitated HF in as many as 30% of patients,[6] a finding that has been reproduced in several populations[9,29] and is noted as a cause of postpartum readmission.[28] However, a distinct syndrome of acute HF with preserved ejection fraction (ejection fraction ≥50%) exists in pregnancy and postpartum and is common, mainly related to hypertensive syndromes of pregnancy and acute diastolic HF related to excessive afterload.[37] Common echocardiographic findings are evidence of abnormal diastolic function (pseudonormal or grade II),

left-sided chamber remodeling including increased left ventricular mass, elevated pulmonary artery pressure, and evidence of abnormal left- and right-sided global longitudinal strain.[38]

Risk Factors in PPCM

Several studies have shown that the majority of patients with PPCM are Black, and incidence is highest among Black women and lowest among Hispanic women, as is true for maternal HF overall.[34,39] The association between PPCM and preeclampsia is well established[37,40,41] accounting for approximately one third of women, whereas the other two-thirds occurred in other forms of heart disease.

ADVERSE MATERNAL OUTCOMES

Mothers with a diagnosis of HF are more than 7 times more likely to die.[10] In ROPAC, maternal mortality was higher in patients with HF (4.8%) compared with those without (0.5%).[6] Much of the data on maternal outcomes offer a truncated understanding because of the data set used or the period of time studied. Some studies focus only on in-hospital deaths where admission-related or inpatient complications are accounted for, thereby failing to identify patients managed in an outpatient setting and deaths that occur outside the

hospital late after pregnancy that are not attributed to being pregnancy related. Mothers with a HF diagnosis at any point in the pregnancy continuum are more likely to experience adverse outcomes, having a significantly increased risk of developing pulmonary edema, renal failure, cerebrovascular disease, and adult respiratory distress syndrome, as well as requiring mechanical ventilation, delivering by cesarean section, and having a prolonged hospital stay.[5,11] Although mortality is uncommon among mothers with HF, it is between 4 and 35 times higher than that of healthy women delivering, a massive disparity.[5,10,11] Certain factors are associated with poorer outcomes among mothers that develop HF; those who succumbed as a result of HF complications were more likely to be Black, older, and have multiple comorbidities.[5,11]

Adverse Maternal Outcomes in PPCM

Despite the high likelihood of experiencing MACE, mothers who develop PPCM specifically also have a high likelihood (72%) of achieving complete recovery with standard HF treatment.[42] Left ventricular ejection fraction (LVEF) <30% at presentation portends a smaller chance of recovery; Black women diagnosed with PPCM were found to have worse left ventricle dysfunction at presentation and at 1 year postpartum, demonstrating the interplay between prognostic factors.[42,43] Interestingly, one National Inpatient Sample study showed that though prevalence of PPCM was low among Asian women, those who did have such a diagnosis had higher in-hospital mortality rates compared with other racial/ethnic groups.[34] Internationally, in a large meta-analysis, the risk of death in women with PPCM was higher in developing countries (14%) than in advanced countries (4%), and significantly higher for mothers of African descent.[44] Unfortunately, promising improvements in LVEF do not necessarily reflect the entirety of the patient experience, as more than half of women in a survey study reported never returning to their emotional baseline before their PPCM diagnosis and over a quarter discontinued their jobs because of their diagnosis.[45] These findings reflect the current understanding that though mortality is a rare occurrence, the toll of maternal cardiovascular morbidity is formidable.[46]

ADVERSE PERINATAL OUTCOMES

Assessing the true impact that maternal HF has on the babies born to affected mothers is a difficult task, as there is a paucity of data on fetal and neonatal outcomes. This may, in part, be related to a lack of a centralized, national US database on mothers, detailed delivery information, outpatient information and child outcome data. Recent studies have shown that

experiencing HF during pregnancy, particularly because of known cardiac diseases, puts fetuses at an increased risk for perinatal mortality and morbidity (Table 2).[10,11,29,47–51] Neonates of affected mothers have lower birth weight, are more likely to be small for gestational age, have lower Apgar scores, and are more likely to be born premature than neonates of healthy controls.[10,11] Of neonates born to mothers with known heart disease who develop HF, nearly one quarter had birth weights under 2500 g, a significant concern as low birth weight is attributed as the second most frequent cause of infant death.[1,6] Neonatal mortality is ≈1% for offspring of affected mothers, as opposed to 0.4% in the unaffected population.[10] In a study of delivery admissions in New York State over a 15-year period with linked fetal and neonatal data, pregnant women with multiple forms of cardiac disease including cardiomyopathy, valvular heart disease, ACHD, and PH experienced an increased risk of adverse fetal/neonatal events. Women with cardiomyopathy and PH and their offspring had the worst overall outcomes.[29] This study demonstrated that maternal MACE, as well as obstetric complications, were both associated with higher risk of neonatal adverse clinical events.

An overwhelming majority of the data in the arena of fetal or neonatal outcomes in women with heart disease are centered on the outcomes of women with ACHD[49,52–54] and infrequently on those with acquired heart disease such as cardiomyopathy, valvular heart disease, or PH, although these forms of heart disease are collectively more common. Table 2 summarizes key findings of studies on various subgroups of mothers with heart disease, focusing on outcomes for their neonates and the risk factors identified for such outcomes. Smoking during pregnancy, multiple gestation pregnancies, cardiomyopathy, and hypertension are among the more consistently demonstrated risk factors for neonatal adverse clinical events (Figure 4).[10,11,29,47,49,50]

SYMPTOMS AND DETECTION OF HEART FAILURE

Many normal symptoms in pregnancy may mimic HF Signs such as peripheral edema, palmar erythema, systolic murmur, and a third heart sound, further complicate detection of cardiac decompensation. Signs that warrant further investigation for cardiac disease are cyanosis, clubbing, resting tachycardia, any type of arrhythmia, hypertension or hypotension, hypoxia, tachypnea, elevated jugular venous pressure, diastolic murmur, murmur radiating beyond the left sternal edge, murmur associated with a thrill, marked peripheral edema, or evidence of pulmonary edema; symptoms such as shortness of breath and orthopnea also

Table 2. Neonatal Outcomes in Mothers With Heart Disease and/or Heart Failure

Authors	Study Type	Study Population	Mortality	Major Findings/Morbidity
Siu et al 2001 (Cardiac Disease in Pregnancy Study)[47]	Prospective	Neonates born to mothers with preexisting HD	2% of fetuses/neonates	• Neonatal events occurred in one fifth of pregnancies, with the most common events being premature birth and SGA. • 5 predictors of neonatal events identified: New York Heart Association class >II or cyanosis during baseline prenatal visit, maternal left heart obstruction, smoking during pregnancy, multiple gestation, and the use of anticoagulation throughout the pregnancy.
Siu et al 2002[48]	Prospective	Neonates born to mothers with preexisting HD	3% in affected population, vs 0.7% in controls	• Neonatal complications were more than 2 times as frequent in those born to mothers with HD vs controls. • Statistically significant increase in likelihood of premature births and infant respiratory distress in neonates born to mothers with HD vs controls.
Drenthen et al 2010 (Zwangerschap bij Aangeboren Hartafwijkingen)[49]	Retrospective	Neonates born to mothers with adult congenital heart disease	4% in affected population	• Premature birth (12%) and SGA (14%) were the most common neonatal complications. • Smoking during pregnancy and twin gestations found to be associated with increased risk of neonatal events.
Koutrolou-Sotiropoulou et al 2015[50]	Retrospective	Neonates born to mothers with preexisting HD (cardiomyopathy vs other)	5% in offspring of mothers with cardiomyopathy, 0% in those with other HD	• Both cardiomyopathy and hypertension were found to be independent predictors of infant respiratory distress syndrome, as well as fetal adverse clinical events. • Maternal cardiomyopathy placed neonates at higher risk for premature delivery, being underweight, infant respiratory distress, and lower 1 and 5 min Apgar scores.
Lawley et al 2015[51]	Meta-analysis	Neonates born to mothers with prosthetic heart valves	Perinatal mortality of 5%	• The pooled event rates were 25.9 per 100 for preterm birth and 14.5 per 100 for SGA.
Barasa et al 2017[11]	Retrospective	Neonates born to mothers with HF in late pregnancy and postpartum	Not applicable	• Neonates of affected mothers were shorter, had lower birth weights, and were more than 6 times as likely to be SGA as their control counterparts.
Ng et al 2018[10]	Retrospective	Neonates born to mothers who developed HF	1% in affected population	• Neonatal deaths higher among neonates born to mothers than developed HF. • Infant births weights lower, more likely to be SGA, Apgar scores lower.
Owens et al 2018[29]	Retrospective	Neonates born to mothers with preexisting HD	In-hospital deaths were 0.7% in affected population (vs 0.2% in controls)	• Neonatal adverse clinical events was more common among neonates born to women with HD than women without HD. • Maternal cardiomyopathy and pulmonary hypertension portended the greatest risk both to mothers and their babies.

HD indicates heart disease; HF, heart failure; and SGA, small for gestational age.

indicate a need for further consideration.[55] Pregnant women with HF commonly displayed dyspnea, cough, bilateral pitting pedal edema, elevated jugular venous pressure, third heart sound, and tender hepatomegaly.[56] Most patients with HF presented with bibasal crepitations.[56] To help clinicians identify cardiac disease in pregnancy the California Maternal Quality Care Collaborative and California Department of Public Health created a California Quality Improvement Toolkit on cardiovascular screening and assessment.[57] This framework gives providers clear guidance on what signs and symptoms are cause for concern and how to proceed with diagnostic testing and further management. Application of the toolkit evidenced that 88% of pregnancy-related cardiovascular mortalities could have been identified before death with the use of this screening tool.[57] It is now recommended that all women should be assessed for cardiovascular disease in the antepartum and postpartum periods using this toolkit algorithm.[58] Family history and other underlying medical conditions can also help guide medical judgment.[58]
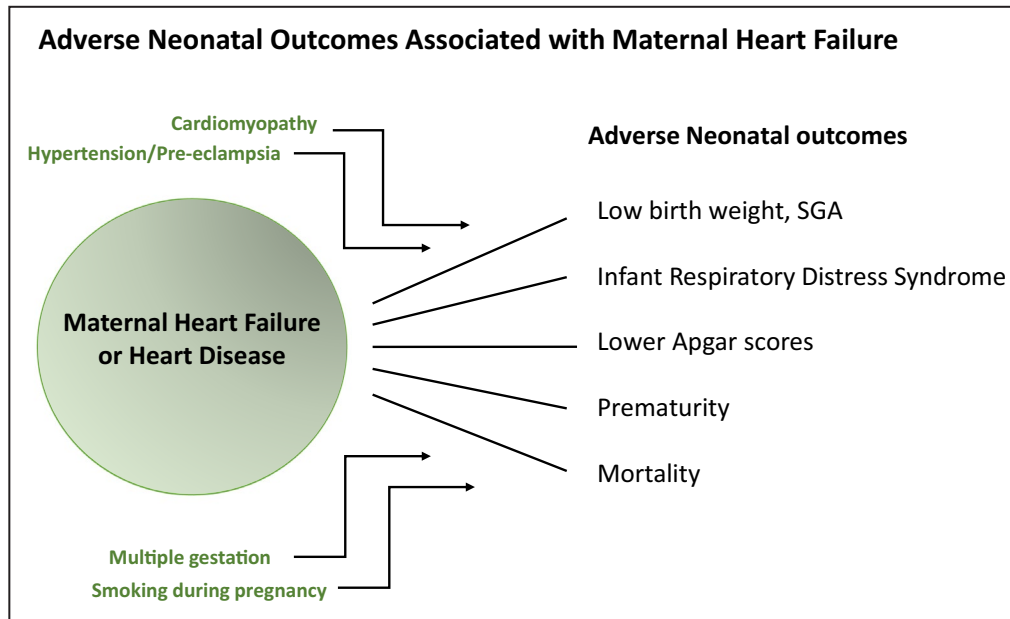
Figure 4. Adverse neonatal outcomes associated with maternal heart failure.[10,11,29,47,49,50]
Adverse neonatal outcomes are listed in black on the right, and independent risk factors for such outcomes in the setting of maternal heart failure or heart disease are listed in dark green. SGA indicates small for gestational age.

A pure clinical evaluation of pregnant patients with suspected HF often does not suffice in steering physicians to a diagnosis of HF; ECG, BNP, and echocardiogram, with the possible addition of chest X-ray in the setting of pulmonary complaints, are needed.[55,57] The utility of BNP in diagnosing HF in pregnant populations has been elucidated. Median BNP levels in normal pregnancies rest at 19 to 20 pg/mL which is increased from nonpregnancy levels (median 10 pg/mL) but still lower than levels correlating to congestive HF in the general population.[59,60] For mothers with preeclampsia this level is elevated even further.[60] Mean peak BNP levels are higher in pregnant women with heart disease than in those without known heart disease, and studies suggest that by using certain cut-offs and serial BNP levels, clinicians can better detect adverse cardiac events during pregnancy.[61] Similarly, this method has shown promise in diagnosing superimposed cardiac processes in women with known preeclampsia related HF.[62] A BNP level of ≤100 pg/mL has a 100% negative predictive value for identifying cardiac events during pregnancy.[61] A threshold of 111 pg/mL as an "elevated" BNP level to predict HF in pregnant or postpartum women exhibited 95% sensitivity and a negative likelihood ratio of 0.1, but this study was conducted only in women with no known cardiac disease proceeding through a singleton pregnancy.[62] Echocardiography serves the same utility in pregnant women as in the general population and can detect structural or physiologic changes to the heart, which may be because of either expected pregnancy-related alterations or cardiovascular disease.

PREVENTING HEART FAILURE

When compared with the women of 10 other industrialized countries, US women were found to have the greatest chronic disease burden (20% considering diabetes mellitus, obesity, heart disease, arthritis, asthma, and hypertension), while having the highest rates of skipping health care (visits, medication) because pf cost, being least likely to have a regular primary care doctor, and being the least satisfied with what care they do receive.[63] Comorbidities, fragmented care, and poor doctor–patient relationships all work against the aims of reducing risks and improving outcomes for these mothers. To combat this, advancement in physician identification and management of pregnant patients that require cardiovascular care is an area that recent literature has brought into focus. A substantial portion (up to 73%) of cardiac events in pregnant women have been shown to be preventable,[7] and 92% of cardiovascular mortalities in pregnancy were classified as having at least some chance at prevention.[64] Delayed cardiovascular diagnosis/treatment initiation as well as failure to refer patients to higher level care are the 2 provider-related factors most often implicated in maternal adverse cardiac outcomes and are more commonly found in the context of cardiac-related deaths, compared

with those that were noncardiac related.[64,65] Among pregnant women with heart disease, 74% of serious cardiac events were attributed to such errors in management determined by providers.[7] This is where cardio-obstetric teams, broader training, and the use of toolkits can be useful.

THE INTERDISCIPLINARY CARDIO-OBSTETRICS TEAM: MATERNAL CARE REIMAGINED

In order to provide moderate and high-risk mothers with effective care aimed at preventing HF and other associated adverse maternal and fetal outcomes, a distinct multidisciplinary approach coordinated among experts from cardiology, obstetrics, maternal fetal medicine, obstetric anesthesiology, neonatology, nursing departments, pharmacists, and social workers must be in place; this forms the cardio-obstetrics team (Figure 5).[66–68] This should occur before pregnancy, at the precontemplative stage or within routine cardiology or gynecologic visits, to prepare patients for the considerations that are in their future. Discussing pregnancy within the context of routine visits years ahead of an actual pregnancy would help set the stage for pregnancy contemplation and allow

for family planning. Directing care for at-risk patients should be done at centers with a such a team and experience in high-risk deliveries.[58] Ideally, a combination of clinics, cardiologist and obstetrician/maternal fetal medicine integrated appointments, as well as periodic interdisciplinary case conferences would establish foundational elements in both patient care as well as research and education in the field.[69] This cardio-obstetrics team should be used at multiple points, starting with prenatal counseling through labor and delivery planning as well as close postpartum follow-up and long-term longitudinal care. Key prenatal visit elements include assessment of symptoms, medication review, preconception health evaluation, maternal cardiovascular risk screening with the mWHO scoring system (Table 3),[70] and patient education that would include advising against pregnancy for those women falling under mWHO class IV.[57,67] Realistically, many women at high risk will present already pregnant, and therefore the team should find ways to coordinate care and moderate risks moving forward.[67] If a woman requires more advanced specialty care, transfer to a center in which a HF specialist, PH specialist, interventional cardiologist, cardiac surgeon, adult congenital specialist, or electrophysiology specialist can be available depending on the needs of the patient. For women experiencing
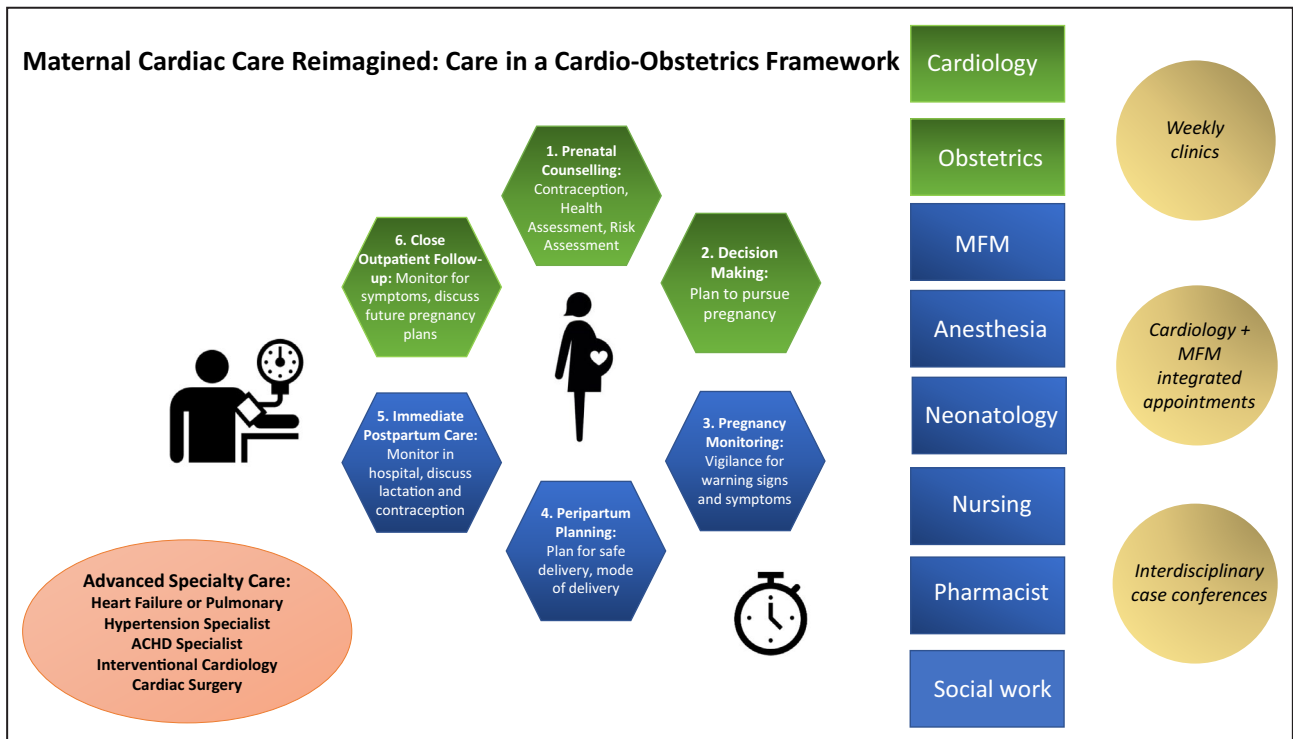
Figure 5.   Maternal cardiac care reimagined: care in a cardio-obstetrics framework.
Elements of a functional cardio-obstetrics program are listed in yellow circles. These will bolster the interdisciplinary team in the next column. Green corresponds to the core cardio-obstetrics team, and blue indicates additional experts that will lend their services at select times. The hexagons depict goals of care throughout the pregnancy course. ACHD indicates adult congenital heart disease; and MFM, maternal fetal medicine.

Table 3. Modified World Health Organization Classification of Maternal Cardiovascular Risk[70]

Class 1	Class 2	Class 3	Class 4
Uncomplicated, small or mild: pulmonary stenosis, patent ductus arteriosus, mitral valve prolapse Successfully repaired simple lesions (atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage) Atrial or ventricular ectopic beats, isolated	Otherwise well or uncomplicated: Unoperated atrial or ventricular septal defect Repaired Tetralogy of Fallot Most arrhythmias	Mechanical valve Systemic right ventricle Fontan circulation Unrepaired cyanotic heart disease Other complex congenital heart disease Marfan syndrome with aorta 40–45 mm Bicuspid aortic valve with aorta 45–50 mm	Pulmonary arterial hypertension Eisenmenger syndrome Systemic ventricular ejection fraction <30% or systemic ventricular dysfunction with New York Heart Association class III–IV Severe mitral stenosis Severe symptomatic aortic stenosis Marfan syndrome with aorta >45 mm Bicuspid aortic valve with aorta >50 mm Native severe coarctation
	Class 2–3 (depending on individual) Mild left ventricular impairment Hypertrophic cardiomyopathy Native or tissue valvular heart disease not considered World Health Organization I or IV Marfan syndrome without aortic dilation Aorta <45 mm in association with bicuspid aortic valve disease Repaired coarctation		

HF, as well as those with significant cardiomyopathy, PH, arrhythmia, significant valve disease, aortopathy, or recent myocardial infarction, postpartum care will require that they continue with monitoring until stability is achieved.[67] Consideration of breast feeding and contraceptive care planning should begin before delivery and should take into account the mother's preferences as well as medical factors.[57]

In addition to a dedicated cardio-obstetrics team, expanding physician knowledge of cardio-obstetrics is key and may be achieved by conscious curricular changes to cardiology fellowship programs aimed at fostering more frequent opportunities to collaborate with obstetrician-gynecologists during training. Finally, without patient education, this system would be fragmented. One study found that one third of patients with reported PPCM diagnoses felt inadequately counseled, with only one quarter feeling satisfied with the counseling they received.[45] Another study showed that half of women with ACHD who should have been advised to avoid pregnancy could not recall having been told so.[71] Women should be aware of their risk, understand warning signs indicating the need for prompt care, be counseled on the option of choosing vaginal delivery, and if diagnosed with HF should understand the implications this may have for future management.[57] As more formal cardio-obstetric teams become established, we hope to see that gaps in both management and awareness be minimized.

THERAPEUTICS AND PRINCIPLES OF MANAGEMENT

When a pregnant or postpartum woman presents in acute HF, initial goals should be to stabilize the patient,

confirm the diagnosis, assess the severity of HF, assess fetal status and viability, and contact members of an interdisciplinary cardio-obstetrics care team.[67,68,72] Any reversible factors that may have contributed to the patient's presentation, such as anemia, thyroid dysfunction, pulmonary embolism, preeclampsia/eclampsia, or infection, should be addressed. The last initial step is to assess fetal viability as well as stability. Steroids can be considered if there is concern for poor fetal lung maturity.[72]

Treatment will vary depending on whether a woman is pregnant or postpartum, as once mothers have delivered there is no longer a concern about fetal stability/maturity or teratogenicity of medications and more standard HF therapies can be initiated (Figure 6).[67,68,73,74] An extensive review of the medication use in pregnancy is beyond the scope of this article but has been reviewed recently.[75] In patients with stable HF, medical treatment approach parallels that of nonpregnant patients; however, there is a need to avoid teratogenic drugs (angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, angiotensin receptor neprilysin inhibitors, mineralocorticoid receptor antagonists, atenolol, direct factor Xa inhibitors).[68,73,76] Mainstays of treatment include hydralazine, nitrates, and β-blockers.[68] Diuretics should be used in those patients with signs or symptoms of pulmonary edema; however, caution must be exercised as they have the potential to reduce placental perfusion.[68,77] In patients who have already delivered, treatment with standard HF therapy should be the highest priority, focusing on angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, angiotensin receptor neprilysin inhibitors, β-blockers, and aldosterone antagonists, as they are known to provide a mortality and treatment benefit to HF patients.[68,72] For patients with cardiogenic shock or severe HF, transfer to a tertiary center

where mechanical support can be provided should be pursued immediately.[68] Treatment in these patients must aim to optimize preload (consider diuretics) and oxygenation, use inotropes/vasopressors, consider adding PPCM-specific therapies, and if the patient is pregnant plan for urgent cesarean section.[68]

If the mother's condition is complicated by the presence of hypertensive disease as well, the best pharmacologic options include intravenous labetalol or hydralazine or oral nifedipine for severe hypertension (persistent blood pressure of ≥160/110 mm Hg), and in cases of less severe hypertension labetalol, nifedipine, and methyldopa are first-line choices. Intravenous nitroglycerin is preferred for patients who present with evidence of pulmonary edema.[67,78]

Patients with newly diagnosed PPCM or other dilated cardiomyopathies have a favorable response rate to medical HF management; therefore, implantation of implantable cardioverter-defibrillators should be deferred. A more appropriate option is a wearable cardioverter-defibrillator for the first 3 to 6 months after diagnosis in those with severe left ventricular impairment (ejection fraction <35%).[72,79]

Delivery Considerations

In women with severe HF or persistent hemodynamic instability despite treatment, urgent delivery by cesarean section should be considered irrespective of gestational duration.[68,72] For women with stable HF, vaginal delivery with epidural anesthesia is the preferred route.[68] If vaginal delivery is pursued, instrumentation is often used to shorten the second stage of labor. Invasive hemodynamic monitoring by way of intra-arterial line may be used to monitor stability in labor and delivery. Epidural is the preferred anesthetic technique.[80] Postpartum cardiorespiratory monitoring is often required in these patients in an intensive care unit or stepdown setting.[80]

PPCM-Specific Considerations

Bromocriptine (dopamine agonist) is being used in the treatment of PPCM as an intervention aimed to improve LVEF. In a 2017 trial of patients with PPCM and LVEF ≤35%, participants were randomized to receive standard HF treatment with either 1 or 8 weeks of bromocriptine treatment. At 6 months postinitiation, left ventricular function was assessed; no significant differences in outcomes were observed between the 2 groups, thus suggesting that a 1-week addition of bromocriptine treatment is sufficient. More important, a higher overall full recovery rate was reflected in this study of patients with PPCM than in others, pointing to a benefit in recovery rates in the use of bromocriptine therapy. Two major limitations of this study are that only 1 patient included in the study was of Black race, which

does not reflect the general patient pool with PPCM, and that there is no nontreatment control group.[81] As there are no strong data available on bromocriptine's use in the United States, it therefore has been stated that using bromocriptine in PPCM still experimental.[82] Anticoagulation with low molecular weight heparin or unfractionated heparin would be necessary to include alongside bromocriptine therapy.[68,72] Patients with PPCM who have had a cardiac transplant have shorter graft survival times and mortality rates after transplantation.[83] Pregnancy in these posttransplant patients is generally not advised.[84]
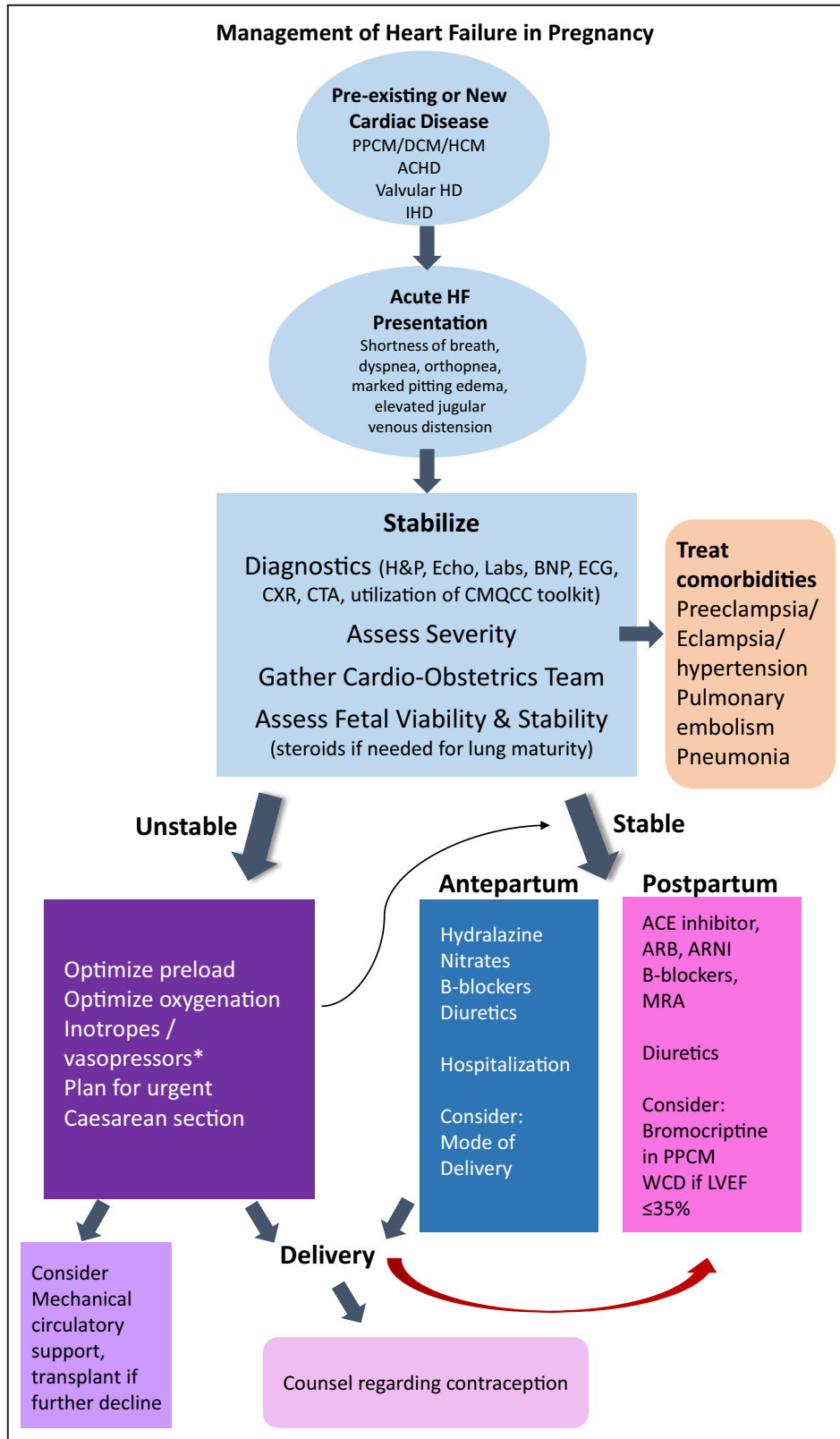
Lactation and Nursing

In 2015, the Food and Drug Administration transitioned from using the previous A through X risk categories to the new Pregnancy and Lactation Labeling Rule.[85] The updated system provides detailed descriptive information on the data and safety of each drug/drug class and eliminates clearly delineated categories of safety. Providers should be aware of this change in labeling when assessing safety of medications during nursing and pregnancy. Mothers with severe HF with reduced ejection fraction (New York Heart Association III or IV) may be advised not to breastfeed.[68] In the clinical setting, many times mothers are too unwell to even attempt it. Though further research must be done, a recent study showed no difference in morbidity between mothers with PPCM who did and did not breastfeed.[86] Currently, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, diuretics, β-blockers, mineralocorticoid-receptor antagonists, and sacubitril/valsartan should be avoided in breastfeeding.

Mechanical Circulatory Support

One study showed temporary percutaneous mechanical circulatory support with Impella heart pump to be a successful bridge to either left ventricular recovery or implantation of durable left ventricular assist devices in women with PPCM-related cardiogenic shock who experienced an initial mean LVEF of 15%.[87] Extracorporeal membrane oxygenation has been shown in small studies to be an effective rescue therapy in pregnant and postpartum mothers.[88,89] Cardiac transplantation is only pursued when mechanical circulatory support is not feasible or desired, or for patients who fail to recover 6 to 12 months after onset.[68]

Discharge Recommendations

Before discharge the following should be addressed with the patient: initiation of goal-directed medical

Management of Heart Failure in Pregnancy

Pre-existing or New Cardiac Disease
PPCM/DCM/HCM
ACHD
Valvular HD
IHD

Acute HF Presentation
Shortness of breath, dyspnea, orthopnea, marked pitting edema, elevated jugular venous distension

Stabilize

Diagnostics (H&P, Echo, Labs, BNP, ECG, CXR, CTA, utilization of CMQCC toolkit)

Assess Severity

Gather Cardio-Obstetrics Team

Assess Fetal Viability & Stability
(steroids if needed for lung maturity)

Treat comorbidities
Preeclampsia/ Eclampsia/ hypertension
Pulmonary embolism
Pneumonia

Unstable

Stable

Antepartum

Postpartum

Optimize preload
Optimize oxygenation
Inotropes / vasopressors*
Plan for urgent Caesarean section

Hydralazine
Nitrates
B-blockers
Diuretics

Hospitalization

Consider: Mode of Delivery

ACE inhibitor, ARB, ARNI
B-blockers, MRA

Diuretics

Consider: Bromocriptine in PPCM
WCD if LVEF ≤35%

Consider Mechanical circulatory support, transplant if further decline

Delivery

Counsel regarding contraception

therapy; identification of causes of HF, barriers to care, and limitations to support; assessment of volume status and blood pressure (adjust therapy accordingly); optimization of chronic oral HF therapy; assessment of renal function and electrolytes; discussion of management of comorbid conditions;

Figure 6. Management of heart failure in pregnancy.[67,68,73,74]

Overall scheme for approaching a pregnant or postpartum patient in acute heart failure. Importantly, management will be determined by pregnancy status and severity of disease. ACE indicates angiotensin-converting enzyme; ACHD, adult congenital heart disease; ARB, angiotensin II receptor blocker; ARNI, angiotensin receptor neprilysin inhibitors; BNP, B-type natriuretic peptide; CM, cardiomyopathy; CMQCC, California Maternal Quality Care Collaborative; CTA, computed tomography angiography; CXR, chest X-ray; DCM, dilated cardiomyopathy; H&P, history and physical exam; HCM, hypertrophic cardiomyopathy; HD, heart disease; HF, heart failure; IHD, ischemic heart disease; LVEF, left ventricle ejection fraction; MRA, mineralocorticoid receptor antagonists; PPCM, peripartum cardiomyopathy; and WCD, wearable cardioverter-defibrillator.

provision of HF education about self-care and methods of adherence; and agreement on a follow-up visit within 1 to 2 weeks.[90] Additionally, discussion of contraception is key for these mothers, as many of them are not appropriately counseled regarding the risks of HF recurrence in subsequent pregnancies.

POSTPARTUM CARE: OPPORTUNITY TO CHANGE THE TRAJECTORY OF DISEASE

Access to care especially during the postpartum period, also called the "fourth trimester," is a clear opportunity for improving care of these vulnerable patients. The early postpartum period is particularly concerning as most HF cases are diagnosed postpartum, the majority of cardiovascular mortalities among mothers occur within the first 6 weeks after delivery, and most venous thromboembolisms occur within 1 week of delivery. Yet, common practice is for mothers to have 1 follow-up visit at 6 weeks postpartum.[5,14,65,91] The American College of Obstetricians and Gynecologists has stated that women determined to be at high risk should undergo cardiovascular evaluation at 3 months postpartum by a cardio-obstetric team, and therefore payment models should develop provisions to supply healthcare coverage to accommodate this.[58] The highest level of continuous coverage from preconception through postpartum by race/ethnicity was found to be 75% among White women, which still leaves ≈1 in 4 women uninsured at some point during their pregnancy.[92] Lack of continuous perinatal insurance disproportionately affected indigenous, Hispanic, and Black non-Hispanic women. Black women are at increased risk for development of HF and subsequent adverse outcomes and this added obstacle of insurance that nearly half of pregnant Black women face puts this population of mothers in a vulnerable position.[92] One in 3 US women have skipped care because of costs, indicating that financial accessibility compounds this issue.[63] Approximately 2 in 5 deliveries are insured by Medicaid, which covers only through the first 60 days after delivery.[93] Efforts are being made to extend this coverage from 60 days to

12 months postpartum at both the state and federal levels.

Discussing contraception plans and lactation with mothers before delivery is another avenue whereby improvements in care can be made. Decision-making around breastfeeding requires individualization in the context of HF; medications should be reviewed for safety and lactating patients should be educated on the current understanding regarding risks. European Society of Cardiology guidelines suggest that lactation should be encouraged in patients with heart disease whenever possible but should be stopped in cases of HF with reduced ejection fraction.[68] As labeling for medications used in pregnancy and lactation has improved after a Food and Drug Administration change in classification in 2014, the ease with which clinicians will be able to address breastfeeding in these patients may be increased.[85] Contraceptive strategies need to be tailored to specific cause of HF.[94] One in 4 patients with previous PPCM are sexually active and not using any form of contraception; the same proportion of women did not recall discussing contraceptive strategies with their provider.[95]

PROGNOSIS

The prognosis of HF in pregnancy varies, ranging from full recovery seen in many patients with PPCM, to lethal outcomes. Though there are risk factors for adverse outcomes, there are relatively few prognostic factors applicable to these patients. Interrogating prognostic indicators as well as likelihood of partial and complete recovery in mothers with HF overall is imperative to advancing management decisions and providing patients with adequate education and information about their condition.

Prognosis in PPCM

Certain prognostic indicators have been identified in relation to PPCM exclusively, such as initial LVEF <30% predicting a lower likelihood of recovery.[42] A recent study investigated the utility of prognostic nutritional index in predicting outcomes for women with PPCM, finding that poor nutritional status was associated with adverse outcomes.[96] This has not yet been studied in other populations.

FUTURE DIRECTIONS AND RESEARCH

The majority of research on HF in pregnancy has focused either on mothers with known preexisting heart conditions or on PPCM specifically, leaving data regarding the overall population of new mothers lacking. Additionally, a better understanding of neonatal outcomes is warranted. The research collaborative, HOPE (Heart Outcomes in Pregnancy: Expectations for Mom and Baby) Registry, aims to gather data on both mothers affected by HF and other cardiac conditions and their babies.[97] This multi-institutional prospective registry would provide insights into delivery approaches, management plans, and contraception counseling practices for this patient population and provide a better understanding of all aspects related to maternal and neonatal outcomes through to 5 years postpartum. Other opportunities for progress lie in State Perinatal Quality Collaboratives, which are networks of multidisciplinary teams, striving to improve measurable outcomes for maternal and neonatal health by examining evidence-based clinical practices and processes using quality improvement principles.[98] Partnership with maternal mortality review committees provides these collaboratives with data and other metrics on temporal changes as well. Successful examples include the toolkits created by the California Maternal Quality Care Collaborative, with improved outcomes in that state of California.[98] Finally, with the presence of an increasing number of cardio-obstetrics teams, continued research into this area, and broader provider experience with maternal cardiac disease, there is promise for improvements in the care and outcomes of these patients.

ARTICLE INFORMATION

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Disclosures

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