The Importance of Cardiovascular Risk Assessment and Pregnancy Heart Team in the Management of Cardiovascular Disease in Pregnancy

Garima Sharma, MD[a,*], Wendy Ying, MD[a], Candice K. Silversides, MD[b]

KEYWORDS

• Cardiovascular disease • Pregnancy heart team • Cardiovascular risk
• Adverse pregnancy outcomes

KEY POINTS

- Multidisciplinary team-based care is the cornerstone of management of cardiovascular disease in pregnancy.
- Understanding and anticipating how the physiologic changes of pregnancy may impact underlying cardiovascular status is important when addressing pregnancy risks in women with cardiac disease. Women with preexisting cardiac disease, prior adverse pregnancy outcomes, and traditional cardiovascular risk factors should undergo careful prepregnancy counseling and have antenatal surveillance once pregnant.
- Incorporating validated risk scores, lesion-specific information, imaging parameters, biomarkers, and patient-specific details can help identify women at highest risk, plan appropriate pregnancy care, and improve outcomes.
- Close postpartum follow-up for women with maternal placental syndromes, gestational diabetes, and other adverse pregnancy outcomes provides a unique opportunity to continue monitoring and implement guideline-directed strategies to reverse long-term cardiovascular complications.

INTRODUCTION

Cardiovascular disease (CVD) is the most common cause of pregnancy-related maternal mortality in the United States.[1] According to data from the Healthcare Cost and Utilization Project's National Inpatient Sample, hospital admissions in pregnant women with CVD increased by 25% from 2003 to 2012.[2] Additionally, the incidence of major adverse cardiac events and arrhythmias also increased. especially in women with pulmonary hypertension or cardiomyopathy.[2] Large cohort studies of pregnancies in women with heart disease report cardiac complication rates of 10% to 15%, of which 4% are serious or life threatening.[3,4] These numbers are likely to increase, because in the past two decades, deliveries among women with

[a] Division of Cardiology, Johns Hopkins Ciccarone Center for Prevention of Cardiovascular Disease, Johns Hopkins University School of Medicine and Hospital, 1800 Orleans Street, Zayed 7125s, Baltimore, MD 21287, USA;
[b] Division of Cardiology, University of Toronto Pregnancy and Heart Disease Research Program, Mount Sinai and Toronto General Hospitals, 700 University Avenue, Room 3-913, Toronto, Ontario M5G 1Z5, Canada
* Corresponding author.
E-mail address: gsharma8@jhmi.edu
Twitter: @GarimaVSharmaMD (G.S.); @WendyYingMD (W.Y.); @CandiceSilvers1 (C.K.S.)

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congenital heart disease (CHD) increased by 34.9%, a greater rise than the 21.3% increase observed in the general population, likely from advances in pediatric cardiac surgery and cardiac care.[5] This group of women are also more likely to have comorbidities, including pulmonary hypertension, congestive heart failure, coronary artery disease, arrhythmias, thromboembolic events, preeclampsia and placenta previa.[6] Importantly, among pregnancies complicated by serious cardiac events, almost 50% are preventable, highlighting the need for early recognition, risk stratification, and management of CVD in pregnancy.[7]

Adverse pregnancy outcomes (APO), such as maternal placental syndromes (preeclampsia, hypertensive disorders of pregnancy [HDP], preterm birth, and small for gestational age baby) and gestational diabetes (GDM), are increasingly recognized to be associated with future maternal cardiometabolic risk and CVD.[8–10] Although the pathogenetic mechanisms of how these conditions result in long-term cardiovascular damage are unclear, at least one explanation is that pregnancy may unmask preexisting elevated CVD risk.[8]

Although pregnancy in women with heart disease is associated with significant morbidity, most women have a safe pregnancy if they have appropriate prepregnancy evaluation and pregnancy care by an experienced pregnancy heart team (PHT). The understanding of pregnancy risk and how to care for women with CVD during pregnancy has evolved over the past two decades.[11,12] Prediction of maternal cardiac complications in women with heart disease is enhanced by integration of general, lesion-specific, and delivery of care variables.[4] A multidisciplinary approach to the care of patients improves outcomes and facilitates a consistent and clear message for the patient (and those caring for each patient). The PHT needs to address risk stratification and management of complications during pregnancy, and risks in the postpartum period.[13,14] In this review, we discuss (1) the multiorgan physiologic changes in pregnancy that can result in decompensation in women with preexisting CVD, (2) an approach to risk stratification in women with heart disease, and (3) the role of multidisciplinary team–based care (**Fig. 1**).

PHYSIOLOGIC CHANGES IN PREGNANCY

The pregnant mother undergoes significant anatomic and physiologic changes to nurture and accommodate the developing fetus. These changes begin after conception and affect every organ system in the body.[15] For most women experiencing an uncomplicated pregnancy, these changes resolve after pregnancy with minimal residual effects (**Table 1**). However, these changes can exacerbate underlying CVD in pregnancy and result in significant maternal morbidity and mortality.[16,17] One of the most important aspects of antenatal care is to assess the impact that these changes could have on existing CVD. This in turn can help guide antenatal follow-up, timing of interventions or medications when needed, and mitigate decompensation.

Changes in the Cardiovascular System

Hemodynamic changes in pregnancy are largely caused by an increase in the cardiac output (CO) by 30% to 50%, secondary to increased stroke volume (SV) and, to a lesser extent, heart rate (HR).[17,18] CO rises early in pregnancy and plateaus between the second and third trimester. The systemic vascular resistance (SVR) decreases until the second trimester and then starts to increase until term.[17] HR increases progressively throughout the pregnancy by 10 to 20 bpm, reaching a maximum in the third trimester. The overall change in HR represents a 20% to 25% increase over baseline.[19–21] Although plasma volume and SVR increase, pulmonary capillary wedge pressure and central venous pressure do not increase significantly.[22] Pulmonary vascular resistance, like SVR, decreases during pregnancy. Maternal hemodynamics during labor are influenced by active stage labor, pain, analgesia, and anesthesia and this can have a profound effect on the existing CVD, especially because of large volume shifts. CO increases by 30% during the active stage of labor. Uterine contractions lead to an autotransfusion of 300 to 500 mL of blood back into the circulation and the sympathetic response to pain and anxiety further elevate the HR and blood pressure. All of these changes can result in acute decompensation or heart failure in women with left-sided valvular obstructive lesions (aortic and mitral stenosis) or right ventricular dysfunction in setting of severe pulmonary hypertension. Following delivery, there is an immediate rise in CO caused by relief of the inferior vena cava obstruction and contraction of the uterus, which empties blood into the systemic circulation. CO starts to decline within about 1 hour of delivery.[18] During this early postpartum period, the transfer of fluid from the extravascular space increases venous return and SVR further especially consequential in those with CVD and therefore these patients are most at risk of pulmonary edema during the second stage of labor and the immediate postpartum period. Cardiac changes can take

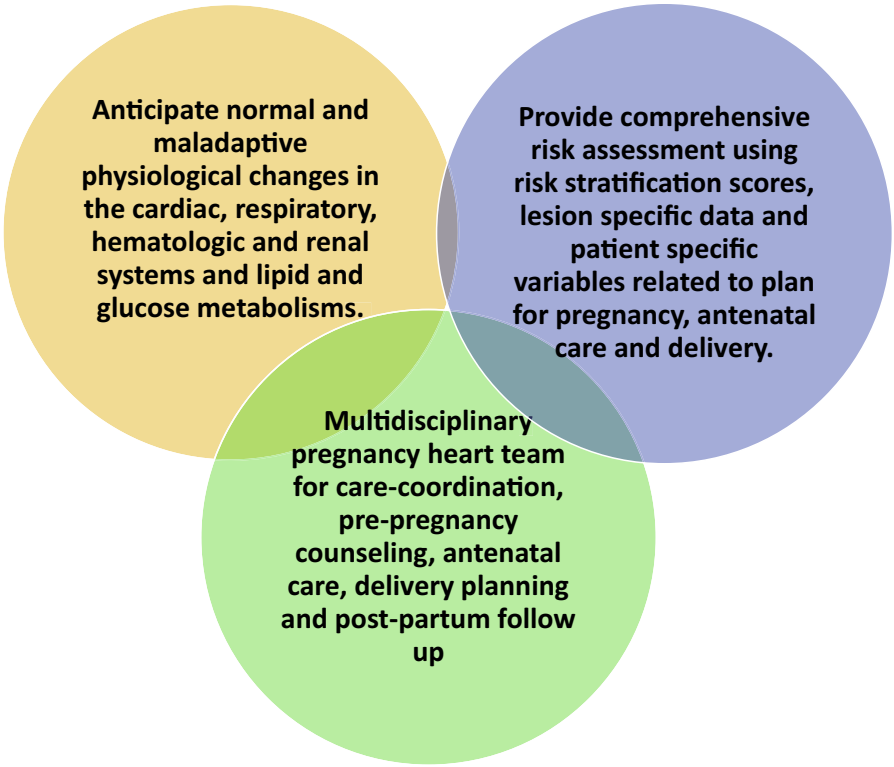

Fig. 1. Comprehensive approach to cardiovascular risk stratification in pregnancy.

6 months to return to normal (prepregnancy values). Some pathologic changes (eg, hypertension in preeclampsia) may take much longer.[23]

Changes in the Respiratory System

During a normal pregnancy, there is an increase in oxygen demand caused by a 15% increase in the metabolic rate and 20% increase in the oxygen consumption.[22,24] There is maternal hyperventilation caused by a 40% to 50% increase in minute ventilation, mostly because of an increase in tidal volume, rather than in the respiratory rate. This causes arterial P_{O_2} to increase and arterial P_{CO_2} to fall, with a compensatory fall in serum bicarbonate to 18 to 22 mmol/L resulting a mild fully compensated respiratory alkalosis (arterial pH, 7.44).[22] Diaphragmatic elevation in late pregnancy results in decreased functional residual capacity, but peak expiratory flow rate and forced expiratory volume in 1 second are unaffected by pregnancy. There is also a subjective feeling of breathlessness without hypoxia in pregnancy. This is most common in the third trimester but may start at any time during gestation, and may be worse at rest and improve with mild activity.[22] In patients with severe restricted lung disease, the ability to increase their ventilation is limited and their lung function and oxygen saturation needs to be monitored.[25] In patients with preeclampsia, there is an increase in minute ventilation because of increase in concentration of blood leptin (a ventilation-stimulating hormone) and decrease in vital capacity caused by pharyngeal edema, weight gain with higher adiposity around the neck, and overall decrease exercise tolerance, which can worsen the respiratory status.[26]

Changes in the Hematologic System

Plasma volume increases proportionally more than the red blood cell mass, resulting in a "physiologic anemia" from hemodilution, with hemoglobin levels as low as 11 g/dL considered physiologic.[18] There are significant increases in total blood volume, plasma volume, and red blood cell mass during pregnancy.[17,18,27] Normal pregnancy is accompanied by changes in the coagulation and fibrinolytic systems. These include increases in several clotting factors (I, II, VII, VIII, IX, and XII), a decrease in protein S levels, and inhibition of fibrinolysis.[28] As gestation progresses, there is also a significant fall in the activity of activated protein C, an important anticoagulant. Thus, pregnancy alters the balance within the coagulation system in favor of clotting, predisposing the

Table 1
Normal physiologic changes in pregnancy and implications in cardiovascular conditions

Organ System	Normal Physiologic Changes	Implications in Cardiovascular Conditions
Cardiovascular system	During pregnancy: ↑ Plasma flow (75%) ↑CO (30%–50%) ↓ SVR and PVR During labor: ↑ CO by 30% in active stage of labor Increased circulating blood volume (300–500 mL) caused by uterine contractions	Cardiac complications in women with lesions that cannot tolerate volume loading (cardiomyopathy), decreases in SVR (Eisenmenger syndrome with intracardiac shunts), or with fixed obstruction (aortic or mitral stenosis) Impaired hemodynamic adaptation ↑MAP, SVR and ↓CO in preeclampsia
Respiratory system	↑Metabolic rate and oxygen consumption Mild compensated respiratory alkalosis	Feeling of breathlessness during pregnancy ↑ Minute ventilation in preeclampsia Difficulty intubating in those who develop serious cardiac complications
Renal system	↑ Plasma flow (75%) ↑ GFR (40%–50%) ↑ Proteinuria	↓ GFR ↓ Uric acid clearance ↑ Proteinuria in preeclampsia Drug dosing may need to be adjusted based on GFR
Hematologic system	↑Plasma volume (50%) and red cell mass ↑ Coagulation factors ↓ Protein C Compression of the inferior vena cava	Physiologic anemia ↑ Risk of thromboembolism in women with prosthetic heart valves, atrial fibrillation, Fontan circulation ↓Lifespan of platelets in and hemolysis in severe preeclampsia (HELLP syndrome)
Lipid metabolism	↑Triglycerides, total cholesterol (50%) and in LDL (50%) and ↓HDL	↑Dyslipidemia with ↓ HDL ↑Free fatty acids in preeclampsia Existing maternal dyslipidemia is associated with adverse pregnancy outcomes
Glucose metabolism	↑Insulin resistance, mild diabetogenic state	Gestational diabetes

Abbreviations: ↓, decrease; ↑, increase; CO, cardiac output; GFR, glomerular filtration rate; HDL, high-density lipoprotein cholesterol; HELLP, hemolysis elevated liver enzymes and low platelets; LDL, low-density lipoprotein cholesterol; MAP, mean arterial pressures; PVR, pulmonary vascular resistance; SVR systemic vascular resistance.

pregnant and postpartum woman to venous thrombosis and other thromboembolic complications.[22] There is an increased risk for thromboembolism during pregnancy because of lower extremity venous stasis resulting from inferior vena caval compression by the gravid uterus and because of a hypercoagulable state caused by an increase in vitamin K–dependent clotting factors and a reduction in free protein S.[29] The issue of hypercoagulability is of particular relevance for women with mechanical heart valves, atrial fibrillation, Fontan circulation, or previous thromboembolic events.

Changes in the Renal System

In a normal pregnancy, there is substantial activation of the renin-angiotensin-aldosterone system, which occurs early in pregnancy, with increases in plasma volume starting at 6 to 8 weeks and rising progressively until 28 to 30 weeks.[30] The decrease in SVR and vasodilation that is seen in early pregnancy results in a 50% increase in renal plasma flow and glomerular filtration rates (GFR) by the end of the first trimester, resulting in decreases in serum creatinine, urea, and uric acid values.[31] Changes in GFR, and changes in the volume of distribution, are important to consider when dosing cardiac medications during pregnancy. In preeclampsia, there is maladaptation of the renin-angiotensin-aldosterone system caused by an increase in SVR and decrease in CO, resulting in decrease in GFR and decreased secretion of uric acid leading to hyperuricemia and exaggerated urinary excretion of proteins.[32]

Changes in the Lipid Metabolism

There is an increase in total serum cholesterol and triglyceride levels mainly as a result of increased synthesis by the liver and decreased lipoprotein lipase activity, resulting in decreased catabolism of adipose tissue. Maternal hypertriglyceridemia is a characteristic feature during pregnancy and corresponds to an accumulation of triglycerides not only in very-low-density lipoprotein but also in low- and high-density lipoprotein.[33] Low-density lipoprotein cholesterol levels also increase and reach 50% at term. High-density lipoprotein increase in the first half of pregnancy and fall in the third trimester but concentrations are 15% higher than nonpregnant levels. Increased triglyceride levels provide for the mother's energy needs and increase in low-density lipoprotein cholesterol is important for placental steroidogenesis. Dyslipidemia in pregnancy is associated with APOs with direct implications on perinatal outcomes.[34] It is prudent to screen women with existing lipid disorders, ideally before conception or at the initial obstetric visit. Abnormal lipids should be treated with diet, exercise, and weight management and bile acid sequestrants in severe cases. In general, statins and other cholesterol medications, such as ezetimibe, are not used during pregnancy and lactation.[35]

Changes in the Glucose Metabolism

During pregnancy, there is a mild diabetogenic state, characterized by increased insulin secretion and increased insulin sensitivity in early pregnancy, followed by progressive insulin resistance.[36] Physiologic pancreatic β-cell hyperplastic adaptation allows for shunting of glucose to the fetus while maintaining maternal nutrition. Maternal insulin resistance begins in the second trimester and peaks in the third trimester. Maladaptive changes in the maternal system that occur in GDM, such as lipotoxicity, inflammation and oxidative stress, and impairments in adipokine and placental signaling, are associated with impaired β-cell adaptation.[37] In the presence of preexisting maternal obesity or excessive gestational weight gain, insulin secretion becomes insufficient to overcome insulin resistance, resulting in hyperglycemia and glucose intolerance that is characteristic of GDM.[38] Preconception counseling, early antenatal risk assessment, and comprehensive health education strategies are helpful strategies to mitigate GDM.

COMPONENTS OF ANTENATAL RISK ASSESSMENT

Ideally, the maternal cardiovascular risk assessment should occur in the preconception phase, because this allows for informed and shared decision making about pregnancy. The prepregnancy encounter should include education about maternal and perinatal risks, optimization of maternal cardiac status, careful review of potentially teratogenic medications with switches to alternatives when necessary, and a discussion about what to expect during pregnancy. Potential risks for women with heart disease and their babies include: maternal and perinatal mortality; maternal cardiac complications, such as heart failure and arrhythmias; obstetric complications, such as postpartum bleeding, preterm birth, and growth restriction; transmission of CHD to offspring; and the potential negative impact of pregnancy on long-term maternal cardiac health.[16,39] Recognition of underlying comorbidities that can result in complications is also important so that early guideline-directed preventative strategies are implemented. For example, women with high-risk features for developing preeclampsia, such as advanced maternal age, prior history of preeclampsia, or diabetes, should be started on low-dose aspirin by 12 to 14 weeks.[40]

Maternal Cardiovascular Risk Assessment

For women with existing CVD including CHD, several risk prediction models have been developed to identify poor maternal cardiac outcomes. The three most commonly known are the CARPREG (Cardiac Disease in Pregnancy), the ZAHARA (Zwangerschap bij vrouwen met een Aangeboren HARtAfwijking-II), and the modified World Health Organization (WHO) risk models (**Table 2**).[3] The three risk models are described next. These risk scores are used as a starting point for risk estimation. Additional variables that are incorporated into risk assessment includes lesion-specific risks; cardiac imaging data (MRI, computed tomography); exercise test results; biomarkers levels, such as brain natriuretic peptide; and other patient-specific information including patient compliance and access to care.

Modified World Health Organization classification

Based on expert consensus, a British Working Group classified pregnancy risk in women with heart disease using modified WHO classification categories[41]: class I (conditions associated with no detectable increased risk of maternal mortality and no/mild increase in morbidity), class II (conditions with small increased risk of maternal mortality or moderate increase in morbidity), class III (conditions with significantly increased risk of maternal mortality or severe morbidity), and class IV (conditions with extremely high risk of maternal

Table 2
Comparison of cardiovascular risk assessment scores and classifications

Modified WHO Classification	ZAHARA Risk Score (Weighted Risk Score Based on Factors of Poor Predictive Outcomes)	CARPEG II Risk Predictors (Weighted Risk Score Based on Lesion, Imaging Parameters and Patient Factors)
Class I: No detectable increase in maternal mortality and no/mild increase in morbidity (uncomplicated and repaired ASD, VSD, PDA, and MVP, atrial and ventricular ectopic beats) Class II: Small increase in maternal mortality and moderate increase in morbidity (Unoperated ASD, VSD, TOF, and ventricular arrhythmias. Depending on the individual mild LV dysfunction, HCM, Marfan without aortic dilatation, repaired coarctation of aorta)	Mechanical valve prosthesis (4.25) Evidence of left-side obstruction (aortic valve peak gradient >50 mm Hg or AVA <1.0 cm^2 (2.50) History of arrhythmia (1.50) Use of cardiac medication prepregnancy (1.50) Repaired and unrepaired cyanotic heart disease (1.0) Moderate to severe atrioventricular valve dysfunction (possibly related to underlying LV dysfunction) (0.75) Baseline NYHA functional classification > II (0.75)	Prior cardiac events or arrhythmias (3) NYHA functional class III-IV or cyanosis (3) Mechanical valve (3) Systemic LV dysfunction (EF <55%) (2) High-risk valve disease (2) Pulmonary hypertension (RVSP >49 mm Hg (2) High-risk aortopathy (2) Coronary artery disease (2) No prior cardiac intervention (1) Late pregnancy assessment (1)
Class III: Significantly increased maternal mortality and morbidity. (Mechanical valve, systemic right ventricle, Fontan circulation, unrepaired cyanotic heart disease, aortic root dilatation in Marfan and bicuspid valve) Class IV: Extremely high-risk maternal mortality and morbidity (Cardiomyopathy with LVEF <30%, pulmonary hypertension, native severe coarctation, severe mitral and aortic stenosis)	Weighted risk score: Maternal cardiovascular complications risk: <0.5 points = 2.9%, 0.5–1.5 points = 7.5% 1.51–2.50 points = 17.5% 2.51–3.5 points = 43.1% >3.5 points = 70%	Weighted risk score: Maternal cardiac complications risk: 0–1 = 5% 2 = 10% 3 = 15% 4 = 22% >4 = 41%
Maternal cardiovascular complication risks: class I, 2.5%–5%; class II, 5.7%–10.5%; class II-III, 10%–19%; class III, 19%–27%; class IV, 40%–100%		

Abbreviations: ASD, atrial septal defect; AVA, aortic valve area; CARPREG, cardiac disease in pregnancy; HCM, hypertrophic cardiomyopathy; LVEF, left ventricular ejection fraction; PDA, patent ductus arteriosus; RVSP, right ventricular systolic pressure; TOF, teratology of Fallot; VSD, ventricular septal defect; ZAHARA, Zwangerschap bij vrouwen met een Aangeboren HARtAfwijking-II.

Adapted from Balci A, Sollie-Szarynska KM, Van Der Bijl AGL, et al. Prospective validation and assessment of cardiovascular and offspring risk models for pregnant women with congenital heart disease. Heart 2014;100(17):1375; with permission.

mortality or severe morbidity; pregnancy is contraindicated) (see **Table 2**). The 2018 American College of Cardiology/American Heart Association Guideline for the Management of Adults with Congenital Heart Disease[42] and the 2018 European Society of Cardiology Guidelines for the Management of Cardiovascular Diseases during Pregnancy[43] recommend the modified WHO classification to predict cardiovascular risk. One criticism of the WHO classification is that it provides a large risk margin for class IV lesions and does not take the patient's clinical characteristics into consideration; nevertheless, the designation of specific cardiac conditions that are considered contraindications for pregnancy is helpful for the clinician.

ZAHARA score

The ZAHARA risk score is a weighted scoring system to predict adverse maternal cardiac events in pregnant women with CHD.[44] The investigators identified eight predictors of poor outcomes: the presence of mechanical heart valve (4.25 points), severe left heart obstruction (mean aortic pressure gradient >50 mm Hg or aortic valve area <1.0 cm^2) (2.50 points), history of arrhythmias and cardiac medication use before pregnancy (1.50 points each), history of cyanotic heart disease (uncorrected or corrected) (1.00 points), moderate-to-severe pulmonary or systemic atrioventricular valve regurgitation, and symptomatic heart failure before pregnancy (New York Heart Association [NYHA] functional class ≥II) (0.75 points each). The score was divided into five categories of risk based on accrued points (see **Table 2**).[44]

CARPREG I and CARPREG II score

The original CARPREG risk score was a four-point risk score developed to predict maternal cardiac complications in women with heart disease. The four variables were equally weighted and included: (1) prior cardiac events, (2) poor NYHA functional class (NYHA class III or IV) or cyanosis, (3) impaired systemic ventricular function (ejection fraction <40%), and (4) left-sided obstruction (mitral valve area of <2 cm^2, aortic valve area of <1.5 cm^2, or peak left ventricular outflow gradient >30 mm Hg).[11] Higher risk scores were associated with higher risk of maternal cardiac complications.

The CARPREG II risk score, based on outcomes data from 1938 pregnancies in women with heart disease at two large Canadian hospitals, identified 10 risk predictors of maternal cardiac complications.[4] In contrast to the ZAHARA population, the CARPREG II study included a more diverse cardiac population that included women with CHD (63.7%), acquired heart disease (22.9%), cardiac arrhythmias (13.4%), mild left ventricular dysfunction (13.6%), and coronary artery disease (2%).[4] The CARPREG II risk score is a weighted risk score, which includes the original four CARPREG risk predictors, and six additional risk predictors. These include five general cardiac predictors (prior cardiac events or arrhythmias, poor functional class or cyanosis, high-risk valve disease/left ventricular outflow tract obstruction, systemic ventricular dysfunction, no prior cardiac interventions); four lesion-specific predictors (mechanical valves, high-risk aortopathies, pulmonary hypertension, coronary artery disease); and one predictor related to delivery of care (late pregnancy assessment) (see **Table 2**).[4] Women with a score greater than 4 had a risk of maternal cardiac complications of more than 40%, and even women in the lowest risk group (score of 0 or 1) had a 5% risk of cardiac complications.

Genetic Counseling

Genetic counseling is an important component of prenatal counseling in women with inherited CVD. For women with inherited genetic disease, a three-generation family history, including details on consanguinity, should be obtained.[45] Genetic evaluation should be made available to all women with CHD, particularly those who are syndromic or who have a family history of CHD; women with inherited arrhythmias, aortopathies, or cardiomyopathies; and women with known autosomal-dominant or recessive conditions.[45,46] The risk of inheriting CHD for the fetus from an affected mother is between 2% and 3% in simple valvular disease; 3% and 6% in complex CHD; and up to 50% in autosomal-dominant syndromes, such as Noonan or Marfan.[47,48]

Antenatal Surveillance

Based on the results of individualized risk assessment, the frequency of antenatal surveillance can be planned. Pregnancy surveillance should include a history and physical examinations, electrocardiograms and transthoracic echocardiograms, basic metabolic profile, complete blood count, and further testing as needed (ie, potential stress testing, cardiac MRI). However, close attention is essential, because some testing modalities involve radiation and therefore may be contraindicated during pregnancy. The goal is to optimize the patient's cardiovascular status. Careful considerations should be given to medications associated with embryopathy and they should be discontinued before pregnancy. For women on anticoagulation for prosthetic valves and other

conditions requiring anticoagulation, guideline-directed recommendations of changing to lower-molecular-weight heparin or continuing with warfarin if the recommended dose is less than 5 mg should be followed.[43]

Delivery Planning

Vaginal delivery remains the optimal method of delivery in most women with heart disease.[49] A Registry of Pregnancy and Cardiac disease (ROPAC) study of in 1262 women with cardiac disease showed that a planned caesarean delivery did not confer any maternal health advantages over planned vaginal delivery, but was associated with an adverse fetal outcome.[50] There is consensus that a planned vaginal birth for most women with CVD, including those with high-risk disease, is safe,[51] Cesarean delivery is generally reserved for obstetric indications, such as breech presentation, failure to progress, placenta previa, or some abnormal fetal HR patterns.[42] Cesarean delivery is necessary in women who have not stopped oral anticoagulation before delivery or in those with acute or chronic aortic dissection. Many experts recommend cesarean delivery in women with Marfan syndrome who have a dilated aorta (>45 mm) or those with intractable heart failure.[16,39,52] The risks of cesarean delivery include: general anesthesia and the risk of hemodynamic instability associated with intubation and the anesthetic agent; blood loss of at least twice that associated with vaginal delivery in some cases, especially in those patients on anticoagulation; and risk of postoperative wound infections. Other labor and delivery considerations include the location of delivery (intensive care unit vs labor and delivery), need for monitoring (oxygen saturation monitors, telemetry monitoring), mode of delivery (vaginal vs cesarean, Valsalva or second-assisted stage), timing of delivery, anesthetic approach (regional vs general), and postdelivery follow-up plans.

COMPONENTS OF POSTPARTUM RISK ASSESSMENT AND FOLLOW-UP

There is increasing focus on extending comprehensive care and close follow-up in the postpartum period. The weeks following birth are a critical period for a woman and her infant, setting the stage for long-term health and well-being. The American College of Obstetrics and Gynecology has released guidelines to optimize the health of women and infants in the postpartum phase and recommended that it should become an ongoing process, rather than a single encounter, with services and support tailored to each woman's individual needs.[53] Women are encouraged to have contact with their obstetrician-gynecologists or other obstetric care providers within the first 3 weeks postpartum.[53] On-going care for complications and postoperative issues should continue until these issues are resolved (**Fig. 2**).[53] Detailed screening for postpartum depression, hypertension, and chronic disease management should include a full assessment of physical, social, and psychological well-being, including the following domains: mood and emotional well-being; breastfeeding, contraception, and birth-control; sleep and fatigue; physical recovery from birth; exercise recommendations; chronic disease management; and health maintenance.[53] Women with chronic medical conditions, such as hypertensive disorders, obesity, diabetes, dyslipidemia, and ischemic or structural heart disease, should be counseled regarding the importance of timely follow-up with their cardiologists or primary care providers for ongoing coordination of care. For women with peripartum cardiomyopathy, aortopathies, and other complex cardiovascular conditions, immediate close follow-up with echocardiograms may be required.[43,52] The postpartum period offers an excellent time to discuss the possibility of future pregnancy and the likelihood of late cardiovascular risk in women with APOs.[13] This is especially important, because APOs are thought to unmask a woman's preexisting cardiovascular risk and act as a harbinger for long-term CVD.[32] Additionally, APOs may be the manifestation of the first diagnosis of a woman's CVD made in pregnancy, particularly chronic hypertension diagnosed as HDP or type 2 diabetes diagnosed as GDM. Unique considerations exist relating to the specific type of underlying CVD and APO. For example, in patients with preeclampsia who develop chronic hypertension, a detailed cardiovascular risk assessment in the postpartum time frame with atherosclerotic cardiovascular risk score and focused interventions on improving diet, lifestyle, exercise, and glucose control in those that have these risk factors can lead to improved health literacy and reduction of cardiovascular risk burden.[54] Several centers throughout the United States and Canada have reported on multidisciplinary maternal health clinics for women with APOs, such as HDP.[55,56] There are several care models incorporating in-person and telehealth visits for implementation of preventative measures, such as home blood pressure and weight monitoring, nutritional referral, exercise recommendations, and other cardiometabolic risk-reduction methods.[57,58] Women should also be counseled about safe contraceptive options.

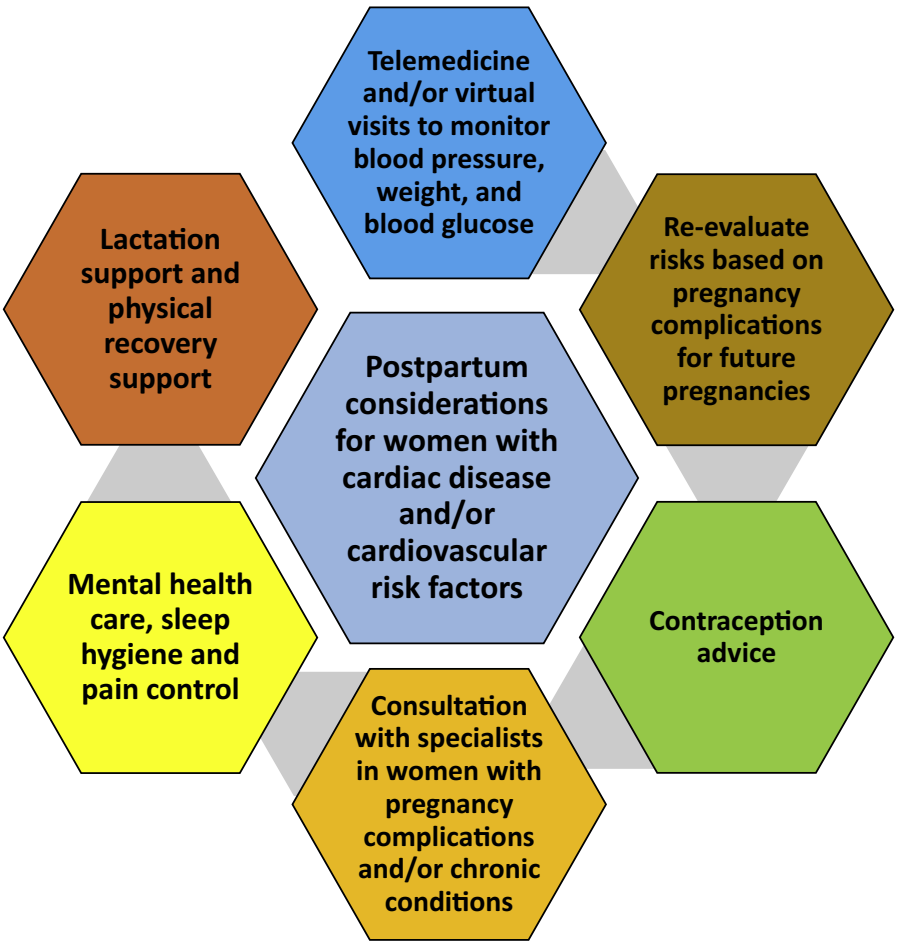

Fig. 2. Postpartum considerations in cardiovascular patients.

ROLE OF MULTIDISCIPLINARY PREGNANCY HEART TEAM

The rising prevalence of cardiovascular conditions complicating pregnancy and the contribution of advancing maternal age, chronic cardiovascular risk factors (eg, maternal obesity, chronic hypertension, sleep apnea, dyslipidemia, and diabetes), and known CVD has led to need for multidisciplinary care models to improve preconception counseling and screening for women.[13,59] More recently, this high-risk care coordination has been endorsed by the national scientific societies and there have been joint presidential statements urging comanagement.[60] The need for multidisciplinary care between cardiologists and obstetricians is supported by best practice statements from the American Heart Association and the American College of Obstetrics and Gynecology.[14] Drawing on this expert opinion and with growing evidence of heart centers for women, we advocate for the assembly of a similar multidisciplinary team for the management of women with CVD in pregnancy.[61]

Members of the Pregnancy Heart Team

Multidisciplinary teams include, at a minimum, a core group of providers from cardiology, obstetrics or maternal fetal medicine, anesthesia, and nursing. Providers should all have expertise in the management of CVD in pregnancy. Other medical specialists (ie, hematologists), geneticists, neonatologists, cardiac interventionalists or surgeons, pharmacists, and social workers can also serve as additional key members.[62] Optimal team-based care involves regular multidisciplinary meetings, during which time these key stakeholders discuss patient management, treatment of complications, and delivery plans (**Fig. 3**).[62]

Specific Goals of the Pregnancy Heart Team

There are several specific goals that a PHT can aim to achieve (see **Fig. 3**). First, the PHT can

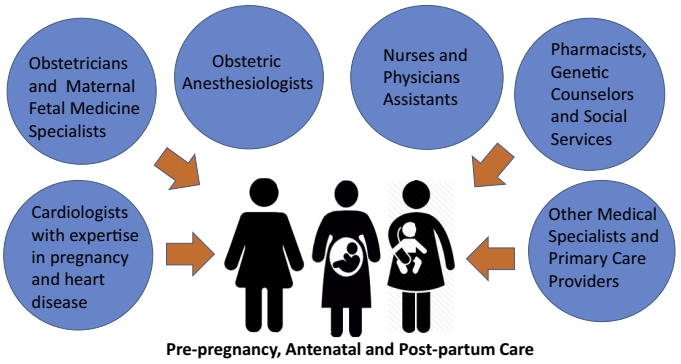

Fig. 3. Composition of the pregnancy heart team and specific goals. Specific goals of the pregnancy heart team: (1) improve the quality of care and establish safety protocols; (2) provide prepregnancy counseling, cardiovascular risk assessment, and pregnancy management with shared decision making; (3) develop detailed delivery plans; (4) organize multidisciplinary conferences for cross-specialty training and education; and (5) ensure postpartum cardiovascular risk assessment and close monitoring of those with chronic conditions.

create and implement standardized protocols aimed at improving quality and safety and reducing preventable causes of obstetric complications and maternal morbidity.[60,63] A list of such causes has been defined by the Alliance for Innovation on Maternal Health, a national maternal safety and quality improvement initiative, and includes maternal venous thromboembolism, obstetric hemorrhage, and HDP.[64] One potential benefit of the PHT is the opportunity for a standardized approach to pregnancy care. Checklists and protocols are known to reduce medical and surgical errors and offer similar benefits over the longitudinal care of a woman's pregnancy.[65–69] Protocols may include workflows to identify, educate, and manage women who are at high risk for APO and maternal morbidity and mortality.

Second, the PHT can risk stratify patients to identify women at high risk of developing cardiac complications, direct antenatal surveillance, and coordinate plans for a safe labor and delivery. Care may include referrals to other specialists and staff. Treatment plans should be clearly recorded into medical records with clear delineation as to the provider responsible for each action item. This provides patients with a uniform approach to counseling and ensures comfort among delivering obstetricians at a given institution when managing these high-risk women in labor. Additionally, the multidisciplinary PHT can help to formulate a birth plan. Given the unpredictable nature of labor, each patient needs a documented delivery plan that outlines mode of delivery, location of delivery, labor analgesia plan, and intrapartum monitoring needs that is accessible to all members of the clinical team. Postpartum follow-up for certain high-risk conditions could also be protocolized to ensure optimal postpartum care.

The logistics of developing a PHT may be a barrier in some institutions. Initiating such an effort requires substantial support from an organizational level from multiple departments. If feasible, establishing a shared clinic space between cardiology and obstetrics or maternal-fetal medicine can facilitate better communication between the two disciplines. It can improve care coordination and make it easier for patients to see providers from multiple specialties. Additionally, standardized methods for reviewing a patient's history and uniform documentation can help coalesce the different practice patterns of providers from different specialties and improve efficiency. Identification of quality metrics a priori to the implementation of a PHT is crucial and lays a foundation for quality improvement assessments.

Finally, the PHT can improve the training and education of residents and fellows. Trainees will be exposed to multidisciplinary clinics and patient conferences, which can provide a forum for discussions of diagnostic and treatment plans in this complex population. Although the primary purpose of these meetings is to optimize care for patients, these meetings also contribute to the continuing education and help to develop rapport among members of the team. Exposure to multidisciplinary care of the pregnant patient with heart is recommended by the Core Cardiovascular Training Statement 4 as part of the fundamental training for cardiology fellows.[70]

SUMMARY

For women with heart disease, pregnancy poses a hemodynamic stress. The increasing burden of CVD in young women of childbearing age has contributed to an increase in maternal morbidity and mortality in the United States. An understanding of the physiologic changes of pregnancy and how they impact women with heart disease is crucial when caring for this group of women. Optimizing the care of pregnant women with CVD

necessitates a multidisciplinary PHT. The PHT consists of specialists from cardiology, obstetrics and maternal fetal medicine, obstetric anesthesia, and nursing supports and provides standard approaches to antenatal, intrapartum, and postpartum care. The PHT also facilitates opportunities for cross-disciplinary education and to bridge the disciplines of cardiology and obstetrics and gynecology.

CLINICS CARE POINTS

- For women with existing CVD, risk prediction models, including the modified WHO, CARPREG II, and ZAHARA scores, should be used to estimate risk of maternal cardiac outcomes and formulate individualized plans for pregnancy, antenatal care, and delivery.
- Multidisciplinary care teams comprised of cardiology and obstetric team members should be utilized for management of patients with CVD in pregnancy.
- Standardized protocols aimed at specific preventable causes of obstetric complications and maternal morbidity can be important in improving quality and safety during pregnancy.
- Women who experience adverse pregnancy outcomes should undergo comprehensive postpartum follow up, with cardiovascular risk assessment focused on prevention of future atherosclerotic cardiovascular disease.

DISCLOSURE

Drs G. Sharma, W. Ying, and C.K. Silversides have no relevant disclosures.

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