

HEART DISEASE IN PREGNANCY 1

Assessment and Management of Cardiac Disease in Pregnancy

Gregory A.L. Davies, MD, FRCSC, FACOG,¹ William N.P. Herbert, MD, FACOG²

¹Professor and Chair, Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynaecology, Queen's University, Kingston ON

²William Norman Thornton Professor and Chair, Department of Obstetrics and Gynecology, University of Virginia, Charlottesville VA, USA

Abstract

Approximately 1% of pregnancies are affected by congenital or acquired cardiac disease. The obstetric care provider requires an understanding of the expected cardiorespiratory adaptations to pregnancy in order to anticipate when and how the cardiac patient may decompensate. Although the majority of women with cardiac disease in pregnancy can expect a positive outcome, women should be evaluated for predictors of poor perinatal outcome to aid in determining the appropriate location for and surveillance in labour. Women affected with congenital heart disease require counselling about the risk of recurrence in their offspring. The discussion of contraceptive needs for the woman with cardiac disease is critical in the appropriate planning of her family.

Résumé

Environ 1 % des grossesses sont affectées par une cardiopathie congénitale ou acquise. Le fournisseur de soins obstétricaux se doit de comprendre les adaptations cardiorespiratoires à la grossesse auxquelles il est en droit de s'attendre, afin d'anticiper le moment où une décompensation affectera la patiente cardiaque et la façon dont cette décompensation s'effectuera. Bien que la plupart des femmes présentant une cardiopathie pendant la grossesse puissent s'attendre à une issue positive, elles devraient néanmoins faire l'objet d'une évaluation visant les prédicteurs d'une issue périnatale indésirable, afin d'aider à déterminer l'endroit où devrait idéalement se dérouler le travail et les mesures de surveillance à déployer dans le cadre de ce dernier. Les femmes qui présentent une cardiopathie congénitale nécessitent des services de counseling au sujet du risque de récurrence chez leur progéniture. Il s'avère crucial d'aborder la question de la contraception avec les patientes présentant une cardiopathie, et ce, afin de leur permettre de procéder adéquatement à leur planification familiale.

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INTRODUCTION

Approximately 1% of pregnancies are complicated by cardiac disease,¹ and the management of these cases can challenge the entire team providing care to the mother and fetus. This is the first in a series of five articles reviewing in detail the assessment and management of specific cardiac disorders in pregnancy.

This first article will review the cardiorespiratory changes that normally accompany gestation, bacterial endocarditis prophylaxis, preconception counselling, intrapartum and postpartum care and contraception. The subsequent articles will discuss congenital heart disease in pregnancy; acquired heart disease in pregnancy; ischemic heart disease and cardiomyopathy in pregnancy; and prosthetic heart valves and arrhythmias in pregnancy.

CARDIORESPIRATORY CHANGES ASSOCIATED WITH PREGNANCY

Significant cardiorespiratory adaptations occur during pregnancy. For the normal woman these changes are usually of no consequence. For women with cardiac disease, however, these physiologic alterations of pregnancy can provoke increased symptoms, morbidity, and even mortality. The major physiologic changes associated with pregnancy are listed in Table 1.

Heart rate, stroke volume, cardiac output, and blood pressure are significantly dependent on maternal body position, especially after the 28th week of gestation, when, in the supine position, the gravid uterus can partially obstruct the vena cava.² This diminution in flow can in turn cause a

significant fall in preload and, subsequently, stroke volume. The result is a picture of presyncope.

A rise in cardiac output is associated with an increased blood flow to the organs crucial in pregnancy, especially to the uterus. By the 10th week of gestation, uterine blood flow is 50 mL/minute; by term, it has increased to 500 mL/minute.^{3,4} Blood flow to the kidneys is also increased by 30%, resulting in an increase in glomerular filtration rate of 50%.^{5,6} An increase in blood volume begins in the first trimester and reaches its peak at 32 weeks' gestation. The mean increase for a singleton pregnancy is 1570 mL.⁷ The discordant increase in plasma volume versus red blood cell mass leads to a decline in the hematocrit.⁸

Central hemodynamic changes identified in pregnant volunteers during the third trimester can be found in Table 2. In normal pregnancy there is a significant fall in the colloid oncotic pressure–pulmonary wedge pressure gradient. This increases the propensity for pulmonary edema in situations of decreased pulmonary capillary permeability or, more importantly for the woman with cardiac disease, increased cardiac preload.⁹ This is particularly true in the immediate postpartum period in women whose cardiac lesion is sensitive to a sudden increase in preload.

Left and right ventricular dimensions in both systole and diastole are unchanged in pregnancy; however, left and right atrial dimensions are significantly larger at 17 ± 4 (mean \pm standard deviation) and 14 ± 3 cm², respectively.¹⁰ Mitral and tricuspid ring diameters are also significantly increased at 24 ± 0.5 and 2.7 ± 3.2 cm, respectively. This is associated with a significantly increased maximal velocity of atrial contribution to mitral inflow in the pregnant population of 57 ± 10 cm/second.¹⁰ These changes associated with the third trimester of pregnancy may represent the cardiac adaptation to a significantly increased preload.

Pregnancy is also associated with a significant increase in respiratory tidal volume, leading to an increase in minute ventilation,¹¹ but the respiratory rate remains unchanged. The increase in tidal volume is offset by a proportionate decrease in functional residual capacity.¹² The increase in minute ventilation results in a reduction in pCO₂ to 30 mm Hg. This in turn leads to a compensatory, though less than equal, increase in renal excretion of bicarbonate. Ultimately this is expressed by the slight respiratory alkalosis seen in pregnancy.

Intrapartum Hemodynamics

The dramatic changes in cardiovascular physiology associated with pregnancy are magnified in labour. Each uterine contraction is associated with an expulsion of 300 to 500 mL of blood from the uterus into the general circulation, adding to preload.^{13,14} This is accompanied by an

Table 1. Major cardiorespiratory changes associated with pregnancy

Pregnancy Parameter	Alteration	Reference
Increased		
Heart rate	+12 bpm	(10)
Stroke volume	+27%	(9)
Cardiac output	4.6–6.0 L/min	(2,9)
Blood volume	+25–50%	(7,28)
Plasma volume	+45–50%	(7,28)
Red cell mass	+20%	(7,28)
Glomerular filtration rate	+50%	(5)
O ₂ consumption	+21%	(11)
Minute ventilation	+48%	(11)
Tidal volume	+40%	(11,12)
Unchanged		
Respiratory rate	Unchanged	(31)
Decreased		
SVR (dyne•sec/cm ⁵)	1200–1250	(9,32)
Blood pressure*		(14)
Hematocrit	33–38%	(8)
Hemoglobin	11–12 g/dL	(8)
FRC	–18%	(12)
pCO ₂	< 30 mm Hg	(14)

* Decrease to 28 weeks then increase to normal at term.

SVR: Systemic vascular resistance; FRC: functional residual capacity; pCO₂: carbon dioxide pressure.

increase in stroke volume and, subsequently, cardiac output.

Overall, the cardiac output in active labour is increased by approximately 2.5 L/minute into the range of 7 to 8 L/minute.^{15,16} Cardiac output and stroke volume are highest when the patient is recumbent in the left lateral position, as there is less obstruction of the vena cava by the gravid uterus.² Blood pressure and central venous pressure are elevated in association with uterine contractions.

Postpartum Hemodynamics

Immediately after delivery there is a significant increase in cardiac output. This increase is probably a result of the dramatically reduced vena caval obstruction by the uterus and the autotransfusion from the contracted uterus. In a group of parturient women who had epidural anaesthesia, cardiac output was reported to be approximately 40% above baseline values at 15 minutes after vaginal delivery and 25% at 30 minutes postpartum. A comparison group who received general anaesthetic also had elevations in cardiac output of

Table 2. Central hemodynamic changes associated with pregnancy

	Nonpregnant	Pregnant
Cardiac output (L/min)	4.3 ± 0.9	6.2 ± 1
Heart rate (beats/min)	71 ± 10	83 ± 10
SVR (dyn•sec/cm ⁵)	1530 ± 520	1210 ± 266
PVR (dyn•sec/cm ⁵)	119 ± 47	78 ± 22
Colloid oncotic pressure (mm Hg)	20.8 ± 1	18 ± 1.5
COP–PCWP (mm Hg)	14.5 ± 2.5	10.5 ± 2.7
Mean arterial pressure (mm Hg)	86.4 ± 7.5	90.3 ± 5.8
Pulmonary capillary wedge pressure (mm Hg)	6.3 ± 2.1	7.5 ± 1.8
Central venous pressure (mm Hg)	3.7 ± 2.6	3.6 ± 2.5
Left ventricular stroke work index (g·m·m ⁻²)	41 ± 8	48 ± 6

SVR: Systemic vascular resistance; PVR: pulmonary vascular resistance; COP–PCWP: colloid oncotic pressure–pulmonary capillary wedge pressure.

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approximately 30% and 15% above baseline at 15 and 30 minutes postpartum, respectively.¹⁷ This elevation of cardiac output persists for 48 hours or so, and is secondary to a persistently elevated stroke volume resulting from increased venous return.

Maternal heart rate actually falls by approximately 10 beats per minute during this time, despite a mean blood loss of approximately 500 mL associated with vaginal delivery and 1000 mL associated with Caesarean section.^{15,18} By two weeks postpartum, cardiac output has reduced by 33%.¹⁹ A postpartum diuresis peaks by the second to fifth postpartum day and lasts for several weeks.

PHYSICAL EXAMINATION IN THE PREGNANT PATIENT

The normal physiologic adaptations to pregnancy may simulate cardiac disease. Displacement of the heart by the gravid uterus results in a more diffuse apical impulse and a palpable systolic pulsation along the left sternal border.² Occasionally the second heart sound is palpable just to the left of the sternal border at the level of the second intercostal space.²

Auscultation of the heart in a healthy pregnant woman often reveals the first heart sound to be increased in intensity and widely split. In the third trimester the splitting of the second heart sound widens less than normal with inspiration.²⁰ Cutforth and MacDonald,²¹ in their series of 50 normal primigravidas found that 84% of gravid patients developed a loud third heart sound and 92% developed an ejection systolic murmur usually heard along the left sternal border. These are usually I to II out of VI in intensity.¹ Diastolic murmurs can occasionally be heard. The most

common is a tricuspid valve inflow murmur or a Graham–Steell pulmonary regurgitation murmur associated with physiologic dilatation of the pulmonary artery. Both of these resolve after delivery.¹ A continuous murmur, the systolic mammary souffle, can be heard over the breasts late in pregnancy or in the postpartum period in breastfeeding mothers.²⁰ A venous hum may also be heard over the suprasternal or upper sternal areas.²⁰

Murmurs associated with mitral and aortic regurgitation and the midsystolic click and murmur of the prolapsing posterior leaflet of the mitral valve often decrease during pregnancy. This is thought to be secondary to the decrease in systemic vascular resistance.^{22,23}

Prominent neck veins or inspiratory wheezes may normally be identified.¹ Pedal edema is a very common finding in normal pregnancy, with 50% to 80% of women affected.²⁰ In most cases this is of a benign nature, and treatment with diuretics is reserved for the most debilitating cases.

NONINVASIVE CARDIAC INVESTIGATIONS

The electrocardiogram in normal pregnancy may show mild left-axis deviation in the third trimester, as well as non-specific ST–T wave changes.²⁰ On echocardiographic assessment, the heart appears mildly volume overloaded and hyperkinetic during the second half of gestation.²⁰ Echocardiographic techniques for estimating cardiac output and stroke volume in pregnant women have been verified with comparison to thermodilution measurements.²⁴

Table 3. Suggested offspring risk for congenital heart defects, given one affected parent

Defect	Mother affected (%)	Father affected (%)
Aortic stenosis	13–18	3
Atrial septal defect	4–4.5	1.5
Atrioventricular canal	14	1
Coarctation of the aorta	4	2
Patent ductus arteriosus	3.5–4	2.5
Pulmonic stenosis	4–6.5	2
Tetralogy of Fallot	2.5	1.5
Ventricular septal defect	6–10	2

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BACTERIAL ENDOCARDITIS PROPHYLAXIS

Prophylaxis against bacterial endocarditis in the pregnant patient with structural cardiac disease, either congenital or acquired, is not currently recommended.²⁵ The risk of bacteremia at the time of vaginal delivery or Caesarean section is low. The American Heart Association guidelines state that antibiotic prophylaxis is not required at vaginal delivery or Caesarean section except in some high risk patients.²⁵

RISK OF CONGENITAL HEART DISEASE IN OFFSPRING

For women with congenital cardiac disease, a discussion of the increased risk of congenital heart disease in their offspring is an important component of prenatal counselling. The incidence of heart disease is increased in the offspring of patients with almost all forms of congenital heart disease (Table 3). The risk is generally higher if the mother, rather than the father, is affected.²⁶ Fetal echocardiography at 18 to 21 weeks' gestation is recommended for a pregnant patient with a congenital heart defect.

GENERAL CARDIAC MANAGEMENT ISSUES FOR THE PREGNANT PATIENT

Preconception Counselling

Preconception counselling is a vital component of the care of women with cardiac disease who are considering pregnancy. A team approach involving obstetricians and cardiologists ensures that the patient has been appropriately investigated and is in optimal condition for and well informed about the planned pregnancy. For patients with congenital heart disease, genetic counselling, either before or early in pregnancy, is recommended to identify the risk for their offspring.

Prenatal Care

Once pregnant, the patient with cardiac disease should be seen early in the first trimester by her obstetrician to document gestational age, as this may play an important role in decisions regarding timing of delivery. Assessment by a cardiologist early in pregnancy is also indicated. Thereafter, patients are seen for prenatal visits every two weeks, or more frequently if necessary. A fetal echocardiogram is recommended at 18 to 21 weeks' gestation for patients with congenital heart disease. If a fetal cardiac anomaly is identified, it should be remembered that this carries a 4% to 5% risk of an associated chromosomal abnormality. Amniocentesis is recommended in this setting.²⁷

Anemia is a common problem in pregnancy and should be avoided in the patient with cardiac disease through the judicious use of iron, prenatal vitamins, and dietary counselling. Discussions of work schedules and levels of activity should be continuous. Consultation with an anaesthesiologist experienced in obstetrical care is recommended, usually in the third trimester. For those at risk, vaccination for influenza is not contraindicated in pregnancy.

Predictors of Poor Maternal and Neonatal Outcomes

Classification of the severity of heart disease can aid in the prediction of maternal and neonatal outcomes and in the counselling of prospective parents.^{28,29} In a large prospective Canadian cohort of pregnant women with heart disease, those at greatest risk of a cardiac event in pregnancy had more than one of the following predictors: a prior cardiac event or arrhythmia, New York Heart Association (NYHA) functional class > II or cyanosis, left heart obstruction, or systemic ventricular dysfunction. The estimated risk of a cardiac event in pregnancy with 0, 1, and > 1 of these predictors was 5%, 27% and 75%, respectively.²⁸ In the same population, the risk of fetal or neonatal death was

doubled from the baseline of 2% to 4% if any of the following five predictors was present: NYHA class > II or cyanosis at the baseline prenatal visit, maternal left heart obstruction, smoking during pregnancy, multiple gestations, and use of anticoagulants throughout pregnancy.²⁸

Labour and Delivery

In most circumstances, patients may await spontaneous labour and can be counselled that the rate of Caesarean section is not increased because of heart disease alone.²⁸ Induction of labour should be reserved for the usual obstetric indications. Once a woman is in labour, she and the fetus should be carefully monitored; especially close attention must be paid to fluid management, because patients can be sensitive to both hypovolemia and hypervolemia. Electrocardiographic monitoring is suggested for those with a history of, or propensity for, cardiac ischemia. Invasive monitoring is not required in patients who have remained relatively asymptomatic throughout pregnancy. However, invasive hemodynamic monitoring may be helpful for patients with severe valvular disease or evidence of pulmonary hypertension.³⁰

Postpartum Care

The postpartum period may be the most crucial time for some patients with cardiac disease. Close monitoring should be maintained while the cardiac output remains elevated; that is, for at least 48 hours.³⁰ Unless otherwise indicated, most patients are reassessed at four to six weeks postpartum, by which time the woman's hemodynamic status has returned to the nonpregnant state.

Contraception

Contraception is an important part of the complete care of the female patient with cardiac disease. This is especially true in circumstances in which pregnancy is contraindicated or pregnancy timing is critical to maximizing maternal consequences. Sterilization of the male partner obviously carries the least risk for the woman with cardiac disease in monogamous couples who have completed their family. Barrier methods, when used consistently and properly, are usually effective, with a failure rate of 2% to 18%.^{31,32} Oral contraceptives can be used in patients with cardiac disease with several exceptions: patients with right to left shunts should avoid oral contraceptives, and those patients whose cardiac disease is associated with hypertension should also choose an alternative method of contraception.^{31,32} Progestin-only oral contraceptives or depot medroxyprogesterone acetate may be used by these women, as the thromboembolic risk of oral contraceptives is thought to be due to the estrogen component.³¹ The progestin-releasing intrauterine device (IUD) can be an excellent choice for

r cardiac patients, because it has a typical failure rate of < 1%.³³ In a monogamous couple the risk of endocarditis associated with use of an IUD is rare, occurring less than once per million woman-years.³³ Prophylaxis with oral amoxicillin (2 g taken one hour prior to insertion or removal) is recommended.³³ Women with cardiac disease who are not in a monogamous relationship would be best to choose an alternative form of contraception.

CONCLUSION

The difficult issues in a pregnancy complicated by cardiac disease are best managed through a team approach. Although most patients begin pregnancy with mild to moderate symptoms associated with their cardiac disease, these can change dramatically as gestation progresses. Patients with severe symptoms need close attention and may require medical and occasionally surgical treatment during pregnancy. Labour, delivery, and the immediate postpartum period are associated with significant hemodynamic challenges, and patients should be monitored throughout. Except for the minority of patients with severe disease requiring medical or surgical therapy before pregnancy, and for those with lesions where pregnancy is contraindicated, pregnancy usually has a successful outcome.

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