

Cardiovascular Assessment During Pregnancy

6

Yumi Shiina

Abstract

Appropriate risk stratification for pregnancy in patients with cardiovascular disease is important, and pregnant patients at intermediate and high risk require special care. Physiologic adaptations to the pregnant state occur with peak effects of these changes generally seen late in the second trimester or early in the third trimester. Heart failure and arrhythmia are common major cardiovascular complications, particularly in the second and third trimester and after birth. Even healthy pregnant patients often complain of edema or breathlessness during these periods, so objective cardiovascular assessments are required, particularly for women with heart disease. Imaging investigations in the antenatal and perinatal periods should be performed with minimal risks to the mother and developing fetus. Therefore, noninvasive techniques such as echocardiogram and magnetic resonance imaging that do not use ionizing radiation are preferable. Antenatal and perinatal management by a multidisciplinary team experienced in cardiovascular disease is recommended for women with underlying congenital heart disease and other severe cardiovascular conditions.

Keywords

Echocardiography \cdot Magnetic resonance imaging \cdot Congenital heart disease Multidisciplinary team

6.1 Care Team and Members During Pregnancy

6.1.1 Where Should Cardiac Assessment and Monitoring Take Place?

Ideally, all pregnant women with heart disease should be referred to a specialist high-risk antenatal service and should have at least one special cardiology review with clinical assessment and transthoracic echocardiogram. The specialist should clarify the anatomical, physiological, and electrical features of the condition and also define the functional status, derive a global risk score for adverse maternal cardiac events in pregnancy, and consider lesion-specific risks. Pregnant patients with cardiac problems can be classified by lesion complexity and graded from simple to highly complex. Table 6.1 shows the modified WHO risk classification of pregnancy with cardiac problems [1], and Table 6.2 focuses on the special team and care, which should be arranged according to level, as follows:

- Level 1—exclusive care with a specialist cardiology review and local obstetric care, with defined lines of communication between specialists [2, 3]
- Level 2—shared care with a regular specialist cardiology review and local obstetric care, with defined lines of communication between specialists
- Level 3—local care with a local cardiology review and local obstetric care, with clearly defined lines of communication between all specialists

Table 6.2 shows an ideal framework for hierarchical care among general physicians, nonspecialist cardiologists, and congenital heart disease (CHD) specialist units.

6.1.2 Sharing Information Among Team Members

A report should be generated that includes diagnosis, a heart diagram, a clinical summary, the anticipated hemodynamic impact of pregnancy, and recent investigations. In the last stages of pregnancy, the report should also provide guidance on delivery planning (monitoring requirements, drugs to be available on the labor ward, requirements for endocarditis prophylaxis, and peri-/postpartum care requirements). There is flexibility in this structure of antenatal clinic provision, in that the care pathway can change and be reassessed if unexpected complications occur or if any of the care providers do not feel comfortable to continue care for any reason. There must also be clear policies outlining the indications for re-referring to the specialist unit. In addition to outpatient review, cases should be discussed in a joint forum (multidisciplinary meeting) once a month. Cardiologists, obstetricians, perinatologists, anesthetists, neonatologists, and others, as part of a multidisciplinary team, should develop a written management plan for labor, delivery, and postnatal

Table 6.1 Modified WHO classification

WHO 1 no risk Uncomplicated, small or mild pulmonary stenosis,	WHO 2 if otherwise well and uncomplicated small-risk complications Unoperated atrial septal defect	WHO 2–3 depending on individual Mild left ventricular impairment	WHO 3 significant risk of complications Mechanical valve	WHO 4 contraindication for pregnancy Pulmonary arterial hypertension of any cause
ventricular septal defect, patent ductus arteriosus Mitral valve	Repaired	Hypertrophic	Systemic right	Severe systemic
prolapse with no more than trivial mitral regurgitation	tetralogy of Fallot	cardiomyopathy	ventricle (e.g., congenitally corrected transposition, simple transposition post-Mustard or Senning repair)	ventricular dysfunction
Successfully repaired simple lesions ostium secundum artrial septal defect, ventricular septal defect, patent ductus arteriosus, total anomalous pulmonary venous drainage	Most arrhythmias	Native or tissue valvular heart disease not considered WHO 4	Post Fontan operation	(NYHA FC III–IV or EF <30%)
Isolated ventricular extrasystoles and atrial ectopic beats		Marfan syndrome without aortic dilatation	Cyanotic heart disease	Previous peripartum cardiomyopathy with any residual impairment of left ventricular function
			Other complex congenital heart disease	Severe left heart obstruction
				Marfan syndrome with aortic dilated >40 mm

WHO world health organization, NYHA FC New York Heart Association functional class, EF ejection fraction

Table 6.2 Care levels for pregnant women with heart disease

Level 1	 Repairs of conduits
Exclusive care in a specialist unit with a	• Fontan
multidisciplinary team	Marfan syndrome
	Ebstein anomaly
	Pulmonary atresia
	Eisenmenger syndrome
	 Repaired complete transposition of the
	great arteries (atrial/arterial switch)
	Congenitally corrected transposition of the
	great arteries
	 Pulmonary hypertension
	Cyanotic congenital heart disease
	Native coarctation of the aorta
	Aortic stenosis
	 Tetralogy of Fallot with pulmonary
	regurgitation (moderate)
	Ventricular septal defect/aortic regurgitation
	Mechanical valves
	 Hypertrophic cardiomyopathy
Level 2	Repaired coarctation of the aorta
Shared care between a specialist cardiologist	Atrioventricular septal defect
and a local obstetric team	Aortic stenosis
	 Pulmonary stenosis/pulmonary
	regurgitation (mild)
	 Tetralogy of Fallot with minimal residua
	 Ventricular septal defect/aortic regurgitation
Level 3	Repaired patent ductus arteriosus
Shared care between general adult cardiology	Mild pulmonary stenosis
Shares care between general addit cardiology	
unit and a local obstetric team	Small ventricular septal defect

care. This care conference is also attended by labor ward midwives, as well as by the core personnel of the high-risk team. The focus is on delivery planning and staff education. The explanation of cardiovascular anatomy and pathophysiology is required in a language that can be understood by competent colleagues not highly trained in cardiology; this is especially relevant for women with CHD, where a schematic diagram of the cardiac anatomy is often helpful for the entire health-care team. The essential factors for successful functioning of a high-risk antenatal care team are good communication and teamwork. "One-stop shopping" (care in the same hospital) can reduce the number of medical visits for pregnant women and enhance immediate communication among doctors in different departments.

6.2 Reliable Risk Scores in Pregnancy with Cardiac Problems

Some studies have suggested that the modified WHO classification (Table 6.1) is more appropriate for all pregnant patients than the CARPREG (Table 6.3) [4] and ZAHARA (Table 6.4) scores [5]. The modified WHO classification is the

Table 6.3 CARPREG risk score

	Point
Prior cardiac event (heart failure, transient ischemic attack, stroke, arrhythmia)	1
NYHA FC III/IV or cyanosis (SPO2 <90%)	1
Left heart obstruction (MVA <2 cm ² , AVA <1.5 cm ² , peak LVOTO >30 mmHg on echocardiogram)	1
Reduced systemic ventricular systolic function (EF <40%)	1

0 point 5%

NYHA FC New York Heart Association functional class, MVA mitral valve area, AVA aortic valve area, LVOTO left ventricular outflow tract obstruction, EF ejection fraction

Table 6.4 ZAHARA risk score

	Point
Prior arrhythmia	1.5
NYHA FC III/NYHA FC IV	0.75
Left heart obstruction (AVA <1.0 cm ² , peak LVOTO >50 mmHg)	2.5
Mechanical valve prosthesis	4.25
Systemic atrioventricular valve regurgitation (moderate/severe)	0.75
Pulmonary atrioventricular valve regurgitation (moderate/severe)	0.75
Cardiac medication before pregnancy	1.50
Cyanotic heart disease (corrected and uncorrected)	1.0

NYHA FC New York Heart Association functional class, AVA aortic valve area, LVOTO left ventricular outflow tract obstruction

only method that takes into account both specific heart lesions (Marfan syndrome, bicuspid aortic valve, tetralogy of Fallot, aortic coarctation, Fontan circulation, or systemic ventricular dysfunction) and general status. This classification and the CARPREG score can be applied to congenital and acquired heart conditions; however, the ZAHARA score is exclusively for women with CHD. The CARPREG score can predict complications in intermediate-risk patients but not in low- and high-risk groups, whereas the ZAHARA score can predict complications in low- and intermediate-risk groups. However, the risk in patients with pulmonary hypertension is underrepresented by these two scores. The better prediction performance of the WHO classification may be attributable to the inclusion of important cardiac lesions or clinical conditions such as systemic right ventricle and pulmonary hypertension, which are relatively uncommon. The disadvantage of the WHO classification is that expert knowledge is sometimes required, especially when choosing the gray zone between WHO II and WHO III. Left ventricular (LV) dysfunction also requires careful interpretation. The CARPREG score includes patients at high risk if LV systolic function is <40%, whereas the WHO classification considers pregnancy to be contraindicated if LVEF is <30% [1]. Overall, a combination of the WHO classification and other risk scores may provide the best assessment of cardiovascular risks in pregnancy [6, 7].

¹ point 27%

>1 point 75%

6.3 Cardiac Assessment During Pregnancy

6.3.1 Physiologic Changes and Adaptations

Physiologic adaptations to the pregnant state generally reach peak effects late in the second trimester or early in the third trimester. In particular, a decrease in total peripheral vascular resistance to 40–70% of prepregnancy levels, augmentation of blood volume by 30–50% compared with baseline, an increase in mean heart rate by 10–20 bpm, and ultimately a 30–50% increase in cardiac output can occur [8]. Physiologic anemia results from an increase in plasma volume that exceeds the increase in red blood cell mass. A thorough physical examination is mandatory with consideration of the physiological changes that occur during pregnancy.

6.3.2 Important Items in an Initial Assessment

- Preexisting maternal cardiovascular disease should be addressed before conception, including hypertension, obesity, atherosclerotic disease, heart failure, arrhythmias, thromboembolic events, and CHD (including residual lesions).
- The risk of adverse maternal cardiac, obstetrical, or neonatal events should be evaluated using a combination of general and lesion-specific maternal cardiac risk factors, as well as classic obstetric risk factors.
- If prior to conception, to consider whether a cardiac intervention might improve the eventual outcome of pregnancy may be indicated in the near future independent of pregnancy.
- To ensure that the nature of the condition is well understood by the woman herself.
- To share the risk assessment with the woman and clarify her understanding and tolerance of the identified risks; there is often ambiguity in such assessments, and this should be acknowledged.
- To explore relevant issues of maternal health and life expectancy that might have an impact on the ability to raise and nurture a child.
- To assess the likelihood of recurrence of heart disease in the offspring. When indicated, to consider genetic consultation, genetic testing, and transabdominal fetal echocardiography.
- To be ready to deal with rare occasions when pregnancy has such a high risk that
 advice against continuation must be offered. Mental support may be required for
 these women.

6.4 Methods of Cardiac Assessment and Monitoring During Pregnancy

6.4.1 Objective Assessment of Functional Status

Maternal functional class is predictive of a poor outcome during pregnancy and women in NYHA functional class III and class IV, regardless of causes, should be

strongly discouraged from becoming pregnant. Functional status should be assessed objectively and carefully because some women are accustomed to situations as chronic heart failure and low cardiac output or cyanosis and often state that they have no symptoms in their daily lives. Cardiopulmonary tests before pregnancy should be performed if needed. Cardiovascular morbidity is lower, and the live birth rate is higher in mothers in NYHA functional class I compared with others in pregnancies with CHD, and in the CARPREG study, maternal prepregnancy NYHA class >II was an independent predictor of adverse maternal cardiac events during pregnancy [4].

6.4.2 Cardiac Medications in Pregnancy

Cardiac medication should be carefully assessed before conception. The decision to continue medications during pregnancy requires weighing the benefits of use against known or possible risks, which may depend on how many weeks pregnant the patient is.

- Choice of drugs: teratogenic and fetopathic effects of cardiac drugs need to be
 considered and alternate therapies found when necessary. The risks and benefits
 of modification of drug therapy have to be addressed in terms of the health and
 safety of the mother and the fetus; the needs of the two do not always coincide.
 Such issues arise with many drugs but are particularly problematic for warfarin,
 angiotensin-converting enzyme (ACE) inhibitors, and angiotensin II receptor
 blockers, amiodarone, and some other antiarrhythmics.
- Anticoagulation: for each patient, the optimal form of therapy must be carefully
 considered. There is a need for particularly precise control, and dosing requirements change as pregnancy progresses. Drug dosing and frequency may need
 adjustment in pregnancy because of changes in the volume of distribution of
 many drugs, increases in glomerular filtration rate enhancing drug excretion, and
 changes in hepatic metabolism affecting drug processing by the liver.

6.4.3 Family History

Many disorders can be identified by taking a careful personal and family history, including cardiomyopathies, Marfan syndrome, CHD, juvenile sudden death, long QT syndrome, catecholaminergic ventricular tachycardia (VT), and Brugada syndrome. It is important to ask specifically about possible sudden death in the family.

6.4.4 Physical Assessments

Normal findings in a healthy pregnant woman include a mild increase in resting heart rate, widened pulse pressure, peripheral edema, and slight elevation of venous pressure.

Heart murmur

In the later stages of pregnancy, there is physiological fixed splitting of the second heart sound (S2). Systolic murmurs are common, secondary to increased cardiac output. However, diastolic murmurs are unusual and require further evaluation. When a new pathological murmur is heard, an echocardiogram is indicated.

• Blood pressure and oxygen saturation

It is crucial to measure blood pressure in the left lateral recumbent position using a standardized method and to look for proteinuria, especially with a history or family history of hypertension or preeclampsia. Oximetry should be performed in patients with CHD, particularly cyanotic patients with intracardiac shunts. Cyanosis poses a significant risk to the fetus, with a live birth unlikely or miscarriage (12%) if oxygen saturation is <85% [9]. A patient with bidirectional shunt may become cyanotic in the second or third trimester, secondary to the increased blood volume.

6.4.5 Electrocardiogram (ECG) (Fig. 6.1)

Most pregnant patients have a normal ECG. The ECG changes as a result of the upward shift of the diaphragm caused by the growing uterus. The heart is rotated toward the left, and there is a 15–20 left axis deviation. In the third trimester, Q waves in lead III and aVF and inverted T waves in leads III, V1, and V2 are seen (Fig. 6.1). ECG changes can be related to a gradual change in the position of the heart and may mimic LV hypertrophy and other structural heart diseases. Holter monitoring (24 h tape) should be performed in patients with previous paroxysmal or persistent documented arrhythmia (VT, atrial fibrillation, or atrial flutter) or those with symptoms of palpitations. In a review of 87 pregnancies in 73 women, 44% with normal sinus rhythm at baseline but a history of tachyarrhythmia before pregnancy had a recurrence of tachyarrhythmia [10]. Bradycardia is poorly tolerated in women with underlying cardiac disease, limiting the ability to meet the cardiac output demands of pregnancy, because cardiac output depends on the product of stroke volume and heart rate.

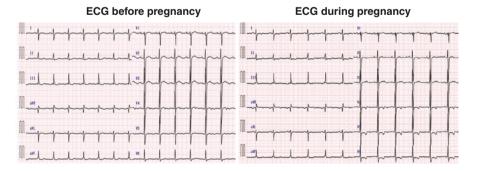


Fig. 6.1 ECG before and during pregnancy

6.4.6 **Imaging**

- *Chest X-ray* (Fig. 6.2) Chest X-ray should be performed as required and without concern about the effect of radiation.
- *Echocardiogram* (Table 6.5)

An echocardiogram is a noninvasive technique that is very useful for cardiac assessment during pregnancy. Ionizing radiation is not required, allowing simple and repeatable investigations. In a normal pregnancy, a significant increase in cardiac output, LV end-diastolic volume, and LV wall thickness are observed. Depending on the underlying congenital defect, pregnancy can be associated with persistent structural cardiac remodeling, deterioration in valvular dysfunction, and worsening ventricular function. One longitudinal study of cardiac function during pregnancy found permanent reduction in systolic and diastolic LV function, but this issue remains controversial [11].

The CARPREG risk score is mainly based on women with congenital and valvular heart disease. Significant predictors for an adverse maternal outcome include

Chest X-ray before pregnancy



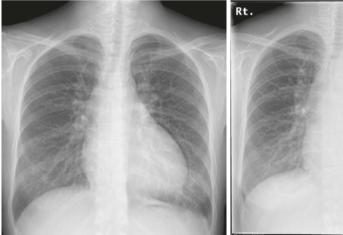




Fig. 6.2 Chest X-ray before and during pregnancy

Table 6.5 Optimal (minimal) timing of echocardiogram

Before pregnancy (baseline)

The peak timing of the cardiac output, late in the second trimester or early in the third trimester

36 weeks pregnancy

4-6 weeks after delivery

In Marfan syndrome, measurements of the aortic root diameter every 4-6 week until 6 months postpartum

left heart obstruction (mitral valve area <2 cm², aortic valve area <1.5 cm², peak left ventricular outflow tract gradient >30 mmHg by echocardiogram) and reduced systemic ventricular systolic function (ejection fraction <40%). In patients with aortic dilatation, an echocardiogram should be recorded at 6- to 8-week intervals in pregnancy until 6 months postnatally. The subpulmonary ventricle may be vulnerable to the consequences of physiologic volume overload during pregnancy. RV longitudinal systolic function and tricuspid annular plane systolic excursion (TAPSE) are related to impaired uteroplacental circulation and offspring complications [12].

A small study in healthy women showed that the subpulmonary ventricular volume increases far more than the systemic ventricle during pregnancy. This physiological dilatation may be aggravated in women with CHD due to abnormal myoarchitecture, damage from prior surgery, and preexistent volume or pressure overload, leading to persistent subpulmonary ventricle dilatation. For specific congenital lesions including systemic right ventricles and tetralogy of Fallot, pregnancy can be associated with persistent deterioration in cardiac function [13]. Women with cardiovascular complications during pregnancy are at risk for persistent dilatation of the RV; however, the details are unclear, and the relationship between subpulmonary ventricular dilatation and pulmonary valve insufficiency is particularly controversial [14]. Such women are also at higher risk for developing cardiovascular complications and dilatation of the subpulmonary ventricle at 1 year postnatally [15].

• MRI during pregnancy

MRI is increasingly used to evaluate pregnant patients with complex heart disease if an echocardiogram is inconclusive, for example, for a dilated ascending aorta. MRI is considered to be safe from 12 to 18 weeks' gestation [16]. In later gestation, left lateral decubitus positioning may be necessary to prevent caval compression. Clinically, cine MRI and whole heart MRI (MR angiogram) without contrast medium provide enough information in pregnant patients with complex cardiac structure. Pregnancy is a contraindication for gadolinium-containing contrast media to avoid exposing the fetus to drugs and an unknown teratogenic risk. There is no clear evidence that gadolinium agents cause chromosomal damage or are teratogenic for the fetus or neonate after injection in pregnant or breast-feeding mothers, but the potential persistence of gadolinium in amniotic fluid is a concern. Therefore, extra caution during the first trimester may be appropriate.

• CT during pregnancy: radiation exposure (Tables 6.6 and 6.7)
Common cardiovascular complications may occur in 1% of all pregnant women. In such a situation, selection of imaging requires thoughtful planning. The use of radiation, radiopharmaceuticals, and contrast agents should be minimized. Pulmonary and cardiac CT angiography cause minimal fetal radiation exposure and ventilation-perfusion scintigraphy involves relatively low fetal irradiation. Cardiac catheterization, coronary angiography, and electrophysiologic procedures, including complex interventions, also cause relatively low fetal exposure. The effects of radiation on the fetus depend on the dose and the gestational age at which exposure occurs. If possible, procedures should be delayed until at least the end of the period of major organogenesis (12 weeks after menses). There is no evidence of an increased fetal risk of congenital malformations, intellectual disability, growth restriction,

Adult effective	Pediatric effective	
dose estimate range	dose estimate range	
(mSv)	(mSv)	Example examinations
0	0	Ultrasound, MRI
<0.1	<0.03	Chest radiography, hand radiography
0.1-1	0.03-0.3	Pelvis radiography, mammography
>1-10	>0.3-3	Abdomen CT, nuclear medicine bone scan
>10-30	>3-10	Abdomen CT with and without contrast administration, whole-body PET
>30–100	>10–30	CT angiography chest, abdomen and pelvis contrast administration; transjugular intrahepatic portosystemic shunt placement

Table 6.6 American College of Radiology relative radiation levels

Table 6.7 Radiological protection suspected in utero induced deterministic radiation effects

Menstrual or gestational age	Conception age	Radiation dose <50 mGy	Radiation dose 50–100 mGy	Radiation dose >100 mGy
0–2 week		None	None	None
3rd and 4th week	1st to 2nd week	None	Probably none	Possible spontaneous abortion
5th to 10th week	3rd to 8th week	None	Potential effects are scientifically uncertain and probably too subtle to be clinically detectable	Possible malformations increasing in likelihood as dose increases
11th to 17th week	9th to 15th week	None	Potential effects are scientifically uncertain and probably too subtle to be clinically detectable	Increased risk of deficits in intelligence quotient or mental retardation that increase in frequency and severity with increasing dose
18th to 27th week	16th to 25th week	None	None	Intelligence quotient deficits not detectable at diagnostic doses
>27 week	>25 week	None	None	None applicable to diagnostic medicine

or pregnancy loss at radiation doses to the pregnant woman of 50 mGy [17–21]. There may be a small increase in risk (1:2000 vs. 1:3000) of childhood cancer [17–21]. Some evidence suggests that risk of malformations is increased at high doses, whereas the risk at doses of 50 to 100 mGy is less clear. In the first 14 days after fertilization, intact survival without fetal abnormality or death is the most likely an outcome of radiation exposure of 50 mGy. After the first 14 days, radiation of 50 mGy may be associated with an increased risk of congenital malformations, growth restriction, and intellectual disability. Most medical procedures do not expose the fetus to such high levels of radiation (Table 6.7). For most diagnostic medical

procedures involving doses to the fetus of up to 1 mGy, the associated risks of child-hood cancer are very low. Shielding of the maternal abdomen (fetus) is required, but is not completely effective because of radiation scattering.

6.4.7 Laboratory

BNP and NT-pro-BNP

Increased B-type natriuretic peptide (BNP) levels are found during pregnancy in many women with heart disease. BNP <100 pg/mL has a negative predictive value of 100% for identifying events during pregnancy [22]. Therefore, evaluating serial BNP levels during pregnancy could be helpful to exclude suspected adverse cardiac events. In the ZAHARA II study, the negative predictive value of NT-pro-BNP <128 μ g/mL at 20 weeks of gestation was 96.9% [23]. Increased NT-pro-BNP at 20 weeks of gestation is an independent predictor of cardiovascular events in women with CHD. Women with cardiovascular events in pregnancy have significantly higher NT-pro-BNP at 1 year postnatally compared to women without cardiovascular events in pregnancy, and NT-pro-BNP <128 μ g/mL at 20 weeks of gestation has a negative predictive value of 98.7% for occurrence of cardiovascular events 1 year postnatally [15].

· CK and troponin

For the diagnosis of acute coronary syndrome (ACS), creatine kinase (CK), CKMB, and troponin may be useful, but careful interpretation is required. During labor, elevated CK occurs due to uterine contractions, and these levels normalize during the second day after labor. Troponin T or troponin I is not elevated in normal pregnancy but can be elevated in patients with preeclampsia or a hypertensive crisis [24].

• *D-dimer and ultrasonography in deep venous thrombosis (DVT)* (Table 6.8)

Table 6.8	Radiation	exposure	of	various	imaging	examinations	performed	for	pulmonary
embolism									

Examination	Effective whole-body dose (mSv)	Fetal dose (mGy)	Effective dose per breast (mGy)
Posteroanterior or lateral chest radiography	0.06-0.25	0.01	
Low-dose perfusion scintigraphy	0.6–1.0	0.1-0.37	0.11–0.3
Ventilation-perfusion scintigraphy	1.2–6.8	0.1-0.8	0.22-0.28
Low-dose pulmonary CT angiography	2–20	0.01-0.66	10–70
Pulmonary digital subtraction angiography	2.7		
Pulmonary digital subtraction angiography	3.2–30.1	0.5	
Evaluation of background radiation	2.5	1.1–2.5	

D-dimer levels increase physiologically with each trimester. In one study, the mean preconception of D-dimer concentration of 0.43 mg/L increased in the first, second, and third trimesters to 0.58, 0.83, and 1.16, respectively, indicating a 39% increase in D-dimer for each trimester compared with the previous one [25]. Thus, a positive D-dimer test based on the conventional cutoff level is not necessarily indicative of DVT, and new cutoff levels are needed based on further objective testing.

Compression ultrasound is the diagnostic imaging procedure of choice for suspected DVT in pregnancy. This method has high sensitivity and specificity for proximal DVT but less for distal DVT and DVT in the vasculature of the pelvis. Serial compression ultrasound evaluations in pregnancy give a high negative predictive value of 99.5% [25]. If a proximal DVT is detected, treatment should be continued. Ideally, women with suspected DVT in pregnancy should be assessed for pretest probability and then undergo a D-dimer test and compression ultrasonography. In a case with a high pretest probability, a positive D-dimer test, and normal initial compression, magnetic resonance venography may be used to exclude isolated pelvic DVT.

6.5 Postnatal Follow-Up

We provide a postnatal cardiac review at the same time as the obstetric postnatal check, typically at 4–6 weeks after birth. Little is known about the long-term effects of pregnancy on maternal cardiac status, but there is the evidence in normal women that pregnancy-related physiological changes resolve by 6 months after delivery.

References

- Thorne S, MacGregor A, Nelson-Piercy C (2006) Risks of contraception and pregnancy in heart disease. Heart 92(10):1520–1525
- 2. Deanfield J, Thaulow E, Warnes C, Webb G, Kolbel F, Hoffman A, Sorenson K, Kaemmer H, Thilen U, Bink-Boelkens M, Iserin L, Daliento L, Silove E, Redington A, Vouhe P, Priori S, Alonso MA, Blanc JJ, Budaj A, Cowie M, Deckers J, Fernandez Burgos E, Lekakis J, Lindahl B, Mazzotta G, Morais J, Oto A, Smiseth O, Trappe HJ, Klein W, Blömstrom-Lundqvist C, de Backer G, Hradec J, Mazzotta G, Parkhomenko A, Presbitero P, Torbicki A, Task Force on the Management of Grown Up Congenital Heart Disease, European Society of Cardiology; ESC Committee for Practice Guidelines (2003) Management of grown up congenital heart disease. Eur Heart J 24(11):1035–1084
- Connelly MS, Webb GD, Somerville J, Warnes CA, Perloff JK, Liberthson RR, Puga FJ, Collins-Nakai RL, Williams WG, Mercier LA, Huckell VF, Finley JP, McKay R (1998) Can J Cardiol 14(3):395–452
- Siu SC, Sermer M, Harrison DA, Grigoriadis E, Liu G, Sorensen S, Smallhorn JF, Farine D, Amankwah KS, Spears JC, Colman JM (1997) Risk and predictors for pregnancy-related complications in women with heart disease. Circulation 96(9):2789–2794
- Drenthen W, Boersma E, Balci A, Moons P, Roos-Hesselink JW, Mulder BJ, Vliegen HW, van Dijk AP, Voors AA, Yap SC, van Veldhuisen DJ, Pieper PG, ZAHARA Investigators (2010) Predictors of pregnancy complications in women with congenital heart disease. Eur Heart J 31(17):2124–2132

 CW L, Shih JC, Chen SY, Chiu HH, Wang JK, Chen CA, Chiu SN, Lin MT, Lee CN, Wu MH (2015) Comparison of 3 risk estimation methods for predicting cardiac outcomes in pregnant women with congenital heart disease. Circ J 79(7):1609–1617

- 7. Balci A, Sollie-Szarynska KM, van der Bijl AG, Ruys TP, Mulder BJ, Roos-Hesselink JW, van Dijk AP, Wajon EM, Vliegen HW, Drenthen W, Hillege HL, Aarnoudse JG, van Veldhuisen DJ, Pieper PG, ZAHARA-II Investigators (2014) Prospective validation and assessment of cardiovascular and offspring risk models for pregnant women with congenital heart disease. Heart 100(17):1373–1381
- 8. Hebson C, Saraf A, Book WM (2016) Risk assessment and Management of the Mother with cardiovascular disease. Clin Perinatol 43:1):1–1)22
- Presbitero P, Somerville J, Stone S, Aruta E, Spiegelhalter D, Rabajoli F (1994) Pregnancy in cyanotic congenital heart disease. Outcome of mother and fetus. Circulation 89(6):2673

 –2676
- Silversides CK, Harris L, Haberer K, Sermer M, Colman JM, Siu SC (2006) Recurrence rates of arrhythmias during pregnancy in women with previous tachyarrhythmia and impact on fetal and neonatal outcomes. Am J Cardiol 97(8):1206–1212
- 11. Kampman MA, Valente MA, van Melle JP, Balci A, Roos-Hesselink JW, Mulder BJ, van Dijk AP, Oudijk MA, Jongbloed MR, van Veldhuisen DJ, Pieper PG, ZAHARA II Investigators (2016) Cardiac adaption during pregnancy in women with congenital heart disease and healthy women. Heart 102(16):1302–1308
- 12. Pieper PG, Balci A, Aarnoudse JG, Kampman MA, Sollie KM, Groen H, Mulder BJ, Oudijk MA, Roos-Hesselink JW, Cornette J, van Dijk AP, Spaanderman ME, Drenthen W, van Veldhuisen DJ, ZAHARA II Investigators (2013) Uteroplacental blood flow, cardiac function, and pregnancy outcome in women with congenital heart disease. Circulation 128(23):2478–2487
- 13. Uebing A, Arvanitis P, Li W, Diller GP, Babu-Narayan SV, Okonko D, Koltsida E, Papadopoulos M, Johnson MR, Lupton MG, Yentis SM, Steer PJ, Gatzoulis MA (2010) Effect of pregnancy on clinical status and ventricular function in women with heart disease. Int J Cardiol 139(1):50–59
- Egidy Assenza G, Cassater D, Landzberg M, Geva T, Schreier J, Graham D, Volpe M, Barker N, Economy K, Valente AM (2013) The effects of pregnancy on right ventricular remodeling in women with repaired tetralogy of Fallot. Int J Cardiol 168(3):1847–1852
- 15. Kampman MA, Balci A, Groen H, van Dijk AP, Roos-Hesselink JW, van Melle JP, Sollie-Szarynska KM, Wajon EM, Mulder BJ, van Veldhuisen DJ, Pieper PG, ZAHARA II Investigators (2015) Cardiac function and cardiac events 1-year postpartum in women with congenital heart disease. Am Heart J 169(2):298–304
- 16. Bulas D, Egloff A (2013) Benefits and risks of MRI in pregnancy. Semin Perinatol 37(5):301–304
- ACOG Committee on Obstetric Practice (2004) ACOG Committee opinion number 299, September 2004 (replaces no. 158, September 1995): guidelines for diagnostic imaging during pregnancy. Obstet Gynecol 104:647–651
- Chintapalli KN, Montgomery RS, Hatab M, Katabathina VS, Guiy K (2012) Radiation dose management: part 1, minimizing radiation dose in CT-guided procedures. AJR Am J Roentgenol 198(4):W347–W351
- Goldberg-Stein SA, Liu B, Hahn PF, Lee SI (2012) Radiation dose management: part 2, estimating fetal radiation risk from CT during pregnancy. AJR Am J Roentgenol 198(4):W352–W356
- Wang PI, Chong ST, Kielar AZ, Kelly AM, Knoepp UD, Mazza MB, Goodsitt MM (2012) Review. Imaging of pregnant and lactating patients: part 1, evidence-based review and recommendations. Am J Roentgenol 198:778–784
- Wang PI, Chong ST, Kielar AZ, Kelly AM, Knoepp UD, Mazza MB, Goodsitt MM (2012) Review. Imaging of pregnant and lactating patients: part 2, evidence-based review and recommendations. Am J Roentgenol 198:785–792
- Tanous D, Siu SC, Mason J, Greutmann M, Wald RM, Parker JD, Sermer M, Colman JM, Silversides CK (2010) B-type natriuretic peptide in pregnant women with heart disease. J Am Coll Cardiol 56(15):1247–1253

- 23. Kampman MA, Balci A, van Veldhuisen DJ, van Dijk AP, Roos-Hesselink JW, Sollie-Szarynska KM, Ludwig-Ruitenberg M, van Melle JP, Mulder BJ, Pieper PG, ZAHARA II Investigators (2014) N-terminal pro-B-type natriuretic peptide predicts cardiovascular complications in pregnant women with congenital heart disease. Eur Heart J 35(11):708–715
- Bozkurt M, Yumru AE, Sahin L, Salman S (2015) Troponin I and D-dimer levels in preeclampsia and eclampsia: prospective study. Clin Exp Obstet Gynecol 42(1):26–31
- Simcox LE, Ormesher L, Tower C, Greer IA (2015) Pulmonary thromboembolism in pregnancy: diagnosis and management. Breathe (Sheff) 11(4):282–289