

# Cardiovascular Disease in Pregnancy

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## Abstract

Heart disease is a major cause of mortality and morbidity in pregnant women. It is reported in 0.2 to 4% of all pregnancies. The incidence of heart disease in pregnancy has increased in the last two decades due to better surgical treatment for congenital heart disease, due to which more number of women are surviving to reach adulthood and opting to conceive and continue pregnancy. Pregnancy is associated with major physiological adaptations in to ensure adequate blood supply to uterus and to ensure blood supply to growing fetus. The net result is increase in cardiac output, plasma volume, heart rate and decrease in systemic vascular resistance. Risk assessment in pregnancy is based on CARPREG scoring system. Patient should be informed about the increased chance of miscarriage, prematurity and fetal growth restriction. If pregnant female is on warfarin it needs to be switched to heparin at pregnancy detection and again at 36 weeks. In general, vaginal delivery is preferred and delivery should occur in tertiary centres with readily available team of cardiologist, anesthetist, senior obstetrician and neonatologist.

**Keywords:** Heart disease, Pregnancy, blood supply, prematurity.

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## INTRODUCTION

Heart disease remains a major cause of mortality and morbidity in pregnant and postpartum women. Cardiac disease is reported in 0.2 to 4 % of all pregnancies [1]. It is the second most common cause of maternal death in USA (17.8%) [2]. It is also the leading cause of indirect maternal death during or up to six weeks after pregnancy in UK [3]. The incidence of heart disease in pregnancy has increased in the last two decades mostly due to better surgical treatments for congenital heart disease, due to which more number of women are surviving to reach adulthood and opting to conceive and continue pregnancy [4]. Risk factors for cardiovascular disease such as diabetes, hypertension, obesity, smoking are also rising in women of reproductive age, thus increasing the prevalence of heart disease in pregnancy. Heart disease in pregnancy is categorized into congenital defects, valvular disease, coronary artery disease and cardiomyopathies.

### Physiological Changes in Pregnancy

Pregnancy is associated with a major change in cardiovascular system to ensure adequate blood supply

to the growing fetus. In first trimester there is upregulation of RAAS (Renin angiotensin aldosterone system) that increases sodium and water retention in the body [5]. Despite activation of RAAS, there is decrease in vascular responsiveness to angiotensin II [6]. There is increase in plasma volume by 40% in pregnancy that starts by 8 weeks and reaches maximum by 24 weeks gestation. Net result is increase in cardiac output, plasma volume, heart rate and stroke volume whereas there is decrease in systemic peripheral resistance (Table-1).

**Table-1: Physiological changes in pregnancy**

Physiological parameter	Change
Heart Rate	Increased 15-20 bpm
Cardiac output	Increased 30-50%
Blood pressure	Decreased by 10 mm Hg
Peripheral vascular resistance	Decreased
Blood volume	Increased by 30-50%
Red cell mass	Increased by 20-30%

The hemodynamic changes begin as early as six weeks antenatally. There are several danger periods in which patient is vulnerable for cardiac decompensation mainly end of first trimester, between 28-32 weeks (when blood volume is maximum) and the peripartum period [5]. Heart failure is also more common around 28-32 weeks and maximum risk is in peripartum period due to sudden gush of blood from uterus into maternal circulation thus increasing the preload [7].

### Preconception Counseling and Risk Assessment

This includes identification of women at risk of further cardiac deterioration and pre pregnancy counseling to optimize maternal-fetal outcome. Women should be counseled about the hemodynamic changes that occur in pregnancy, the danger periods during pregnancy that can further worsen the cardiac condition and the warning signs of cardiac deterioration. Teratogenic drugs need to be stopped and if possible, should be replaced with drugs that do not cross placenta or are less embryotoxic. Parents should be informed about the increased risk of miscarriage, prematurity, fetal growth restriction and low birth weight babies and long term implications of these complications.

In the present era of assisted reproduction, females with heart disease should be counseled about risk of multiple pregnancy and additional hemodynamic stress that it poses to pregnant female [8].

Women with genetic conditions should know the likelihood of recurrence of the disease in fetus, that ranges from 2 to 3% in simple valvular disease and upto 50% in autosomal dominant conditions like Marfans or Noonans syndrome [9].

Risk assessment in pregnant women with heart disease should include a complete history, physical examination, ECG and echocardiography. Cardiopulmonary exercise testing can be done prenatally to estimate the likelihood of complications [1]. Several risk assessment scores are used, most popular being CARPREG [10] which is summarized in Table-2. WHO risk assessment is summarized in Table-3. Recently a new risk index CRAPREG II has been introduced in an attempt to accurately predict cardiovascular risk in pregnant women [11]. After risk assessment patient should be clearly informed whether she will require delivery in a specialist unit with more frequent antenatal visits or if she can be managed by the routine antenatal team.

**Table-2: Risk assessment in pregnant women with heart disease using the CARPREG scoring system**

<b>CARPREG scoring (1 point for each factor)</b>
Prior cardiac event (arrhythmia, stroke, heart failure)
NYHA Class > II or cyanosis
Left heart obstruction (mitral valve area <2 cm <sup>2</sup> , aortic valve area <1.5 cm <sup>2</sup> , peak LV outflow tract gradient >30 mm by echocardiography)
Systemic ventricular dysfunction (ejection fraction <40%)

<b>Total points</b>	<b>Risk of cardiac complication (%)</b>
0	5
1	27
>1	75

**Table-3: Modified WHO classification for risk assessment of cardiac conditions in pregnancy**

<b>Category</b>	<b>Risk</b>
I	No detectable increased risk of maternal mortality and no/mild increase in morbidity.
II	Small increased risk of maternal mortality or moderate increase in morbidity
III	Significantly increased risk of maternal mortality or severe morbidity. Expert counselling required. If pregnancy is decided upon, intensive specialist cardiac and obstetric monitoring needed throughout pregnancy, childbirth, and the puerperium.
IV	Extremely high risk of maternal mortality or severe morbidity; pregnancy contraindicated. If pregnancy occurs termination should be discussed.

Conditions in which pregnancy risk is WHO I:

- Uncomplicated, small or mild pulmonary stenosis, patent ductus arteriosus, mitral valve prolapse
- Successfully repaired simple lesions (atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage)

- Atrial or ventricular ectopic beats

Conditions in which pregnancy risk is WHO II (if otherwise well and uncomplicated)

- Unoperated atrial or ventricular septal defect
- Repaired tetralogy of Fallot
- Most arrhythmias

Conditions in which pregnancy risk is WHO II–III (depending on individual)

- Mild left ventricular impairment
- Hypertrophic cardiomyopathy
- Native or tissue valvular heart disease not considered WHO I or IV
- Aorta <45 mm in aortic disease associated with bicuspid aortic valve
- Repaired coarctation

Conditions in which pregnancy risk is WHO III

- Mechanical valve
- Systemic right ventricle
- Fontan circulation • Cyanotic heart disease (unrepaired)
- Other complex congenital heart disease
- Aortic dilatation 40–45 mm in Marfan syndrome
- Aortic dilatation 45–50 mm in aortic disease associated with bicuspid aortic valve

Conditions in which pregnancy risk is WHO IV (pregnancy contraindicated)

- Pulmonary arterial hypertension of any cause
- Severe systemic ventricular dysfunction (LVEF<30%, NYHA III/IV)
- Aortic dilatation >50 mm in aortic disease associated with bicuspid aortic valve
- Native severe coarctation
- Marfan syndrome with aortic dilatation >45 mm
- Severe mitral stenosis, severe symptomatic aortic stenosis
- Previous peripartum cardiomyopathy with any residual impairment of left ventricular function

### Management of Specific Cardiac Conditions in Pregnancy

1. Congenital heart disease: an increasing number of women are presenting with childhood operated ASD, VSD or other septal defects who are opting for motherhood due to near normal cardiac function status [12]. If cardiac function as assessed by echocardiography are normal, such patients can be followed like routine antenatal patients without the need for IE prophylaxis or need for anticoagulants which is often required in patients with valvular heart disease.

2. Valvular heart disease: most common valvular heart disease in pregnancy is rheumatic valvular disease contributing around 80% of cardiac problems in developing countries. Most common valvular heart disease is mitral stenosis. Patients with severe mitral stenosis (valve area < 1.5 cm<sup>2</sup>) and severe Aortic stenosis (valve area < 1 cm<sup>2</sup>) are at a significant risk for cardiac decompensation during pregnancy [13]. In such patients percutaneous balloon valvuloplasty is indicated [14]. Pharmacological treatment consists of beta blockers, vasodilators, diuretics and rest.

Antibiotic prophylaxis with penicillin or third generation cephalosporins like cefazolin or ceftriaxone is recommended in patients with stenotic lesion or with history of prosthetic valve. Antibiotic prophylaxis is often given to such patients to prevent bacterial endocarditis.

Anticoagulants are not required in well compensated patient or in patients with bioprosthetic valves. Patients with prosthetic valves / mechanical valves are at increased risk of thromboembolism and therefore require anticoagulation [15]. Warfarin is the best anticoagulant, preferred in non-pregnant state due to its high efficacy, but is teratogenic. Risk of embryopathy is maximum at dose more than 5 mg/day and during the time of organogenesis i.e. 6-9 weeks. To avoid warfarin embryopathy, coumarin is stopped at 6 weeks of gestation or when cardiac activity appears and is switched over with either unfractionated heparin (usually 18 mg/kg body weight) or low molecular weight heparin (at dose of 1 mg/kg twice daily). From 12 weeks onwards warfarin is restarted and stopped again at 36 weeks when switchover is usually done with unfractionated heparin or low molecular weight heparin because they do not cross the placenta and hence are safe for fetus [16].

1. Cardiomyopathy: Peripartum cardiomyopathy is the most common cardiomyopathy found in pregnancy. It is diagnosed when there is acute left ventricular impairment occurring between the last 4 weeks of pregnancy and 5 months postpartum with unknown etiology. ECHO finding of such patient shows Ejection fraction < 45% with fractional shortening of <30% and end diastolic dimension of >2.7cm<sup>2</sup>. Based on recent studies, bromocriptine, a prolactin blocker has been used with variable results. Despite pharmacological advancements mortality still remains high and only 50% of recover fully at 6 months with recurrence of 25% in subsequent pregnancy [17].
2. Coronary artery diseases lead to myocardial infarction and heart failure which is rare in

pregnancy. BNP (Brain natriuretic peptide) has a good negative predictive value for diagnosis of heart failure [18]. Heart failure should be managed with rest, a combination of hydralazine and nitrates to reduce cardiac after load as ACE inhibitors are contraindicated in pregnancy.

## ANTENATAL CARE

Women with WHO class 3 and 4 should be managed in a cardiac specialized unit with close follow up. During each antenatal visits following parameters should be assessed and recorded -

1. Blood pressure and heart rate
2. Heart rhythm
3. Auscultate for additional heart sounds or murmurs or basal crepitations
4. Assess fetal growth

Patients with infection, anemia, sepsis need to be admitted and monitored under supervision of expert cardiologist as these factors can cause cardiac decompensation. Common symptoms of pregnancy such as breathlessness and fatigue can mimic cardiac symptoms. Abnormal signs and symptoms that point towards the possibility of cardiac deterioration include extreme breathlessness, marked edema, fourth heart sound, diastolic murmur, raised jugular venous pressure and persistent tachycardia [19]. Women with heart disease are most likely to encounter ectopic beats, arrhythmia in late second or third trimester.

Electrocardiogram (ECG) is a useful diagnostic tool helpful throughout pregnancy. However subtle changes like left axis deviation, inverted T waves and inferior Q waves due to diaphragmatic elevation are common in third trimester and are physiological. After ECG other most common investigation is echocardiography and is safe throughout pregnancy. Assessment of ventricular function using ejection fraction, tissue Doppler and M-mode measurements is done. Cardiac magnetic resonance imaging is rarely used. Gadolinium may be used as a contrast agent only if it significantly increases diagnostic accuracy and is expected to improve maternal and fetal outcome [20].

Fetal echocardiography is done between 18-22 weeks and is indicated in women with congenital heart disease such as Marfan syndrome as there is a high risk of inheritance. Antenatally fetal growth scans are to be followed with clinical correlation as there are high chances of fetal growth restriction. Fetal growth restriction is more prominent if there is history of intake of beta blockers.

## DELIVERY

Ideally one should wait for spontaneous onset of labor. Delivery of women with cardiovascular disorders should occur in tertiary centres with readily

available team of expert obstetrician, cardiologist, critical care anesthetist and neonatologist. Combination of regional anesthesia for pain relief, passive second stage with assisted delivery with forceps/ventouse allow a "pain free, push free labor" which is safer for mother. In general, vaginal delivery is preferred. Contrary to obstetrician perception C-section is associated with more profound hemodynamic changes, more blood loss, increased risk of postpartum infection and venous thromboembolism [21]. It should be considered only in cases of severe stenotic lesion-MS or AS, heart failure, severe pulmonary hypertension (eisenmengers, aortic aneurysm > 4.5 cm, aortic dissection).

During intrapartum period oxytocin should be given in concentrated form avoiding fluid overload. Ergometrine should be avoided in third stage of labor as it has pronounced vasoconstrictive effect. Misoprostol may be used for postpartum hemorrhage.

After delivery cardiac patient needs monitoring in ICU for at least 24 hours due to major shift of fluid from extravascular to intravascular compartment.

Postnatally females should be counseled regarding contraception. Vasectomy in male partner should be encouraged, in female partners the options include: progesterone intrauterine devices, DMPA (depot medroxy progesterone acetate) injection, or progesterone only pills. Barrier methods can be used as adjunct. Oral contraceptive pills are to be avoided due to risk of thromboembolism associated with COC pills.

## REFERENCES

1. Endorsed by the European Society of Gynecology (ESG), the Association for European Paediatric Cardiology (AEPC), and the German Society for Gender Medicine (DGesGM), Authors/Task Force Members, Regitz-Zagrosek, V., Blomstrom Lundqvist, C., Borghi, C., Cifkova, R., ... & Gorennek, B. (2011). ESC Guidelines on the management of cardiovascular diseases during pregnancy: the Task Force on the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology (ESC). *European heart journal*, 32(24), 3147-3197.
2. Kuriya, A., Piedimonte, S., Spence, A. R., Czuzoj-Shulman, N., Kezouh, A., & Abenhaim, H. A. (2016). Incidence and causes of maternal mortality in the USA. *Journal of Obstetrics and Gynaecology Research*, 42(6), 661-668.
3. Knight, M., Tuffnell, D., Kenyon, S., Shakespeare, J., Gray, R., & Kurinczuk, J. J. (2015). Saving lives, improving mothers' care—surveillance of maternal deaths in the UK 2012–2014 and lessons learned to inform maternity care from the UK and Ireland confidential enquiries into maternal deaths and morbidity 2009–2014. Oxford: National

- Perinatal Epidemiology Unit, University of Oxford.
4. Elkayam, U., Goland, S., Pieper, P. G., & Silversides, C. K. (2016). High-risk cardiac disease in pregnancy: part I. *Journal of the American College of Cardiology*, 68(4), 396-410.
5. Pritchard, J. A. (1965). Changes in the blood volume during pregnancy and delivery. *Anesthesiology: The Journal of the American Society of Anesthesiologists*, 26(4), 393-399.
6. Gant, N. F., Worley, R. J., Everett, R. B., & MacDonald, P. C. (1980). Control of vascular responsiveness during human pregnancy. *Kidney international*, 18(2), 253-258.
7. Hunter, S., & Robson, S. C. (1992). Adaptation of the maternal heart in pregnancy. *British heart journal*, 68(6), 540-543.
8. Qin, J., Wang, H., Sheng, X., Liang, D., Tan, H., & Xia, J. (2015). Pregnancy-related complications and adverse pregnancy outcomes in multiple pregnancies resulting from assisted reproductive technology: a meta-analysis of cohort studies. *Fertility and sterility*, 103(6), 1492-1508.
9. Drenthen, W., Pieper, P. G., Roos-Hesselink, J. W., van Lottum, W. A., Voors, A. A., Mulder, B. J., ... & Ebels, T. (2007). Outcome of pregnancy in women with congenital heart disease: a literature review. *Journal of the American College of Cardiology*, 49(24), 2303-2311.
10. Siu, S. C., Sermer, M., Colman, J. M., Alvarez, A. N., Mercier, L. A., Morton, B. C., ... & Taylor, D. A. (2001). Prospective multicenter study of pregnancy outcomes in women with heart disease. *Circulation*, 104(5), 515-521.
11. Silversides, C. K., Grewal, J., Mason, J., Sermer, M., Kiess, M., Rychel, V., ... & Siu, S. C. (2018). Pregnancy outcomes in women with heart disease: the CARPREG II study. *Journal of the American College of Cardiology*, 71(21), 2419-2430.
12. Yadav, V., Sharma, J. B., Mishra, S., Kriplani, A., Bhatla, N., Kachhawa, G., ... & Kriplani, I. (2018). Maternal and fetal outcome in operated vs non-operated cases of congenital heart disease cases in pregnancy. *Indian heart journal*, 70(1), 82-86.
13. Sharma, J. B., Yadav, V., Mishra, S., Kriplani, A., Bhatla, N., Kachhawa, G., ... & Toshayan, V. (2018). Comparative study on maternal and fetal outcome in pregnant women with rheumatic heart disease and severe mitral stenosis undergoing percutaneous balloon mitral valvotomy before or during pregnancy. *Indian heart journal*, 70(5), 685-689.
14. Vinayakumar, D., Vinod, G. V., Madhavan, S., & Krishnan, M. N. (2016). Maternal and fetal outcomes in pregnant women undergoing balloon mitral valvotomy for rheumatic mitral stenosis. *Indian heart journal*, 68(6), 780-782.
15. Malhotra, M., Sharma, J. B., Arora, P., Batra, S., Sharma, S., & Arora, R. (2003). Mitral valve surgery and maternal and fetal outcome in valvular heart disease. *International Journal of Gynecology & Obstetrics*, 81(2), 151-156.
16. Deans, C. L., Uebing, A., & Stear P. (2006). Progress in Obstetrics and Gynecology Elsevier; Edinburgh, 17:164-182. (Cardiac Disease in Pregnancy. InStuddJ, TanSL, ChervenakFA).
17. McKenna, W. J., Maron, B. J., & Thiene, G. (2017). Classification, epidemiology, and global burden of cardiomyopathies. *Circulation research*, 121(7), 722-730.
18. Tanous, D., Siu, S. C., Mason, J., Greutmann, M., Wald, R. M., Parker, J. D., ... & Silversides, C. K. (2010). B-type natriuretic peptide in pregnant women with heart disease. *Journal of the American College of Cardiology*, 56(15), 1247-1253.
19. Metcalfe, J., & Ueland, K. (1974). Maternal cardiovascular adjustments to pregnancy. *Progress in cardiovascular diseases*, 16(4), 363-374.
20. Committee on Obstetric Practice. (2017). Committee Opinion No. 723: Guidelines for Diagnostic Imaging During Pregnancy and Lactation. *Obstetrics and gynecology*, 130(4), e210-e216.
21. Ruys, T. P., Cornette, J., & Roos-Hesselink, J. W. (2013). Pregnancy and delivery in cardiac disease. *Journal of cardiology*, 61(2), 107-112.