Occurrence of heart diseases complicate 1 to 4% of pregnancy in healthy women. Normal physiologic changes that occur during pregnancy can exacerbate symptoms of underlying cardiac disease even in previously asymptomatic patients. Therefore existing heart disease in females particularly (mitral/aortic) valvular stenosis congenital or acquired abnormality with pulmonary hypertension or Rt to Lt shunts, CCF due to any cause & coronary heart disease should be counseled in advanced about risk of pregnancy.

The physiologic changes that occur during pregnancy are maternal blood volume rises progressively till end of 6th & 7th month. Stroke volume also increases at the same time due to volume change & an increase in EF. These changes occur due to reduced peripheral resistance because of vasodilatation & low resistance shunting through placenta. Heart rate rises in third trimester to further increase the cardiac output by 30 to 50%. Systolic blood pressure rise slightly but diastolic BP fall significantly. In supine position gravid uterus compress IVC & can lead to reduced venous return & lower cardiac output. The blood volume increases to 40 to 50% during normal pregnancy is disproportionate to red blood cell mass causing fall in hemoglobin concentration & anemia.

Due to high cardiac output the cardiac symptoms & signs are altered & mimic the signs & symptoms of heart disease. Normal pregnancy is associated with fatigue, dyspnea, reduced exercise capacity, peripheral edema & jugular venous distension. Presence of third heart sound, pulmonary flow murmurs, ECG changes include rate related decrease in PR & QT intervals, Lt axis deviation inferior lead Q waves due horizontal position of heart & nonspecific ST-T changes. 2DEcho shows increase in chamber size, functional valvular regurgitation & occasionally small pericardial effusion. During labor & delivery the hemodynamic changes can be profound. Each uterine contraction displaces 300 to 500 ml blood in systemic circulation further increases cardiac output during labor & delivery which is above the base line. Blood loss during delivery can contribute to hemodynamic stress. During postpartum state the hemodynamic changes are dramatic. IVC compression relieved, venous return increased, increase in cardiac output & brisk diuresis occur. The hemodynamic changes return to normal in 2-4 wks (vaginal delivery) or 4 to 6 wks (cesarean section) to the pre-pregnant state.

Women with underlying heart disease should be fully aware of impact of maternal & foetal risks of pregnancy and potential long term maternal & foetal morbidity & mortality. Wherever the risk of HD is very high, the pregnancy in these women should not be advised.

**SPECIFIC CONGENITAL OR ACQUIRED CARDIAC LESIONS CAN BE CLASSIFIED AS LOW, INTERMEDIATE OR HIGH RISK DURING PREGNANCY.**

**Low Risk Cardiac lesions:-**
Congenital – ASD, VSD, PDA & mild PS & repaired acyanotic congenital heart disease without any residual cardiac dysfunction. 
Acquired – Asymptomatic AS with low mean gradient <50mm Hg & normal LV functions, AR, MR & MVP with normal LV functions & MS with mean gr. <5mmHg, MVA >1.5cm² & without pulmonary hypertension.

**Intermediate Risk:-**
Large Lt to Rt shunt, coarctation of the aorta Marfan syndrome with a normal aortic root, moderate to severe MS, mild to moderate AS & severe PS.

**High Risk Conditions:-**
Eisenmenger’s syndrome, severe PH, complex cyanotic heart disease (TOF, TGA) Ebstein’s anomaly & Tricuspid atresia, Marfan syndrome with AV & Aortic root involvement, symptomatic severe AS, double Aortic or mitral valve disease with moderate to severe LV dysfunction. History of prior peripartum cardiomyopathy & pregnancy with NYHA III or IV symptoms of valvular, CCF or cardiomyopathy.

Young women with uncomplicated congenital heart lesion with small shunt usually tolerate pregnancy well & once these shunts are repaired the risk during pregnancy is minimal. But development of pulmonary hypertension with Lt to Rt shunt increases risk of complications during pregnancy.

**Mitral & Aortic regurgitation:-**
Chronic MR due to rheumatic, myxomatous degeneration tolerated well during pregnancy but new onset AF & Hypertension, ruptured
of chorade tendineae precipitate hemodynamic deterioration.

Aortic regurgitation usually due to rheumatic, congenital, infective endocarditic or connective tissue disorder. Women with such lesion should undergo operative repair before pregnancy or conception. Congestive cardiac failure from MR or AR treated with Diagoxin, diuretic & vasodilator (hydralazine) therapy.

**Mitral & Aortic Stenosis:**

Mitral stenosis in women of childbearing age is most often rheumatic etiology. Moderate to severe MS causes severe hemodynamic changes in IIIrd trimester, during labor & delivery. Pulmonary edema is due to physiologic increase in blood volume, rise in heart rate which leads to elevation of Lt arterial pressure. Development of AF in the pregnant pts with MS may result in rapid decompensation. Digoxin & β blocker can be used safely to reduced blood volume & raised LA pressure. If AF not controlled, electrocardioversion can be performed safely. Anticoagulation therapy is essential to avoid stroke & fetal complications.

The women requiring operative repair, Percutaneous volvotomy during pregnancy is usually deferred to II & IIIrd trimester. Epidural anesthesia is usually better tolerated.

In India Aortic stenosis in child bearing age is due to rheumatic or congenital bicuspid valve. Severe AS (AVA <1 cm² & mean gr>50mmHg) with symptoms of dyspnea, angina, syncope which become apparent in II or IIIrd trimester, need surgical repair.

High risk condition described (in ‘c’) above are associated with increased maternal & fetal mortality. If risk is very high consideration of medical termination of pregnancy should assessed an individual basis.

**Other Risk Factors:**

It has been seen that birth rate for older women (25 to 44 yrs age) have increased. In these age group, prevalence of traditional cardiovascular risk factors such as diabetes, hypertension, smoking, hyperlipidemia & thrombophelia are associated with increased risk of spontaneous abortion, premature birth, acute arterial & venous thrombosis during pregnancy. These factors also predict future development of coronary artery disease, chronic hypertension, stroke & peripheral arterial disease in mother. The cumulative incidence of type II diabetes appears to increase markedly in the first 5yrs after pregnancy.

**ACQUIRED CARDIOVASCULAR DISORDER DURING PREGNANCY:**

**Maternal placental syndrome:**

These are group of disorder have been associated with high maternal risk of premature CVS diseases. Maternal placental syndrome (MPS) defined as “presence of preeclampsia, gestational hypertension, placental abruption or placental infarction during pregnancy”. MPS occurred in 7% of 1.03 million women, who were free from CVS disease before pregnancy. Traditional cardiovascular risk factors are more in women with MPS. So women with MPS are twice as likely to experience a hospital admission for revascularization procedure for coronary, cerebrovascular or peripheral vascular disease.

**Peripartum Cardiomyopathy:**

Peripartum cardiomyopathy (PPCM) is defined as “development of idiopathic Lt ventricular systolic dysfunction in the interval between the last month of pregnancy up to the first 5 postpartum months in women without preexisting cardiac dysfunction.”

The incidence of PPCM in USA is 1 in 3000 to 4000 live births. The exact etiology is unknown but, viral myocarditis, autoimmune phenomena & specific genetic mutation that formation of prolactin have been proposed as possible cause. The clinical symptoms rarely develop before 36 wks of gestation, but some studies shown symptoms at 17th weeks of gestation.

The treatment for PPCM should be started during pregnancy & continue postpartum. Digoxin, diuretics & hydralazine may be used safely during pregnancy & breast feeding. Beta blocker may improve LV function in pts with cardiomyopathy. ACE inhibitors can be initiated in postpartum period. Anticoagulation therapy for selected pts with severe Lt ventricular dilation & dysfunction. Some pts with PPCM may require mechanical assist device or cardiac transplantation. More than 50% women with PPCM recover completely with 6 month of delivery. If LVEF <30% at diagnosis there is persistence of LV dysfunction or shows clinical deterioration, such women should avoid subsequent pregnancy.

**Coronary Artery Disease:**

Acute MI during pregnancy is rare (1:35000). Associated risk factors chr: hypertension, women age >33 years, diabetes & history of preeclampsia can give rise to acute MI during pregnancy, during IIIrd trimester. Coronary spasm, Coroary thrombosis & coronary dissection occur more commonly than classic atherosclerotic lesion. Maternal mortality is high in antepartum & intrapartum period. But over past decade improvement in diagnosis & therapy mortality & morbidity improved.

Management of Acute MI in pregnant women must be modified. Thrombolytic therapy increases risk of maternal hemorrhage (8%) but can be used in certain situation. Low dose aspirin & nitrates are considered safe. Beta blocker are generally safe. Short term heparin is also safe, hydralazine, nitrate, clopidogrel & GPIIIb/IIa receptor inhibitors have been used safely. Percutaneous coronary intervention, balloon angioplasty & stenting has been successfully performed in pregnant pts with AMI with lead shielding to protect foetus.

**Arrhythmias in Pregnancy:**

Atrial & ventricular premature complexes are common during pregnancy & not require therapy. But SVT require β blocker or digoxin to control ventricular rate. Adenosine & direct current cardioversion are safe during pregnancy. Prepregnancy tachyarrhythmias have recurrence during pregnancy & fatal outcome. Direct current cardioversion can be safely carried out.
Pregnancy & Heart Disease


Anticoagulents:

Several cardiovascular condition require anticoagulation therapy.

- mechanical valves, prior history of DVT/DVT.
- Prothrombotic conditions & thromboembolism.
- Antiphospholipid antibody syndrome & AF.

The most common three agents used in heart disease and pregnancy are the unfractionated heparin, low molecular weight heparin & warfarin are considered for use during pregnancy. The seventh American college of chest physician consensus conference on Antithrombotic therapy has recommended three potential strategies for anticoagulation during pregnancy.

1. In women with venous thromboembolism LMWH is choice of therapy.
2. In women with mechanical valve, use of heparin to prevent valve thrombosis.
3. Warfarin therapy is safe during breast feeding.

Warfarin freely crosses placental barrier & can harm fetus. Incidence of warfarin embryopathy (foetal bone & cartilage abnormality) is 4% to 10%, risk is more when given during 6 to 12 wks of gestation, II & III trimester CNS abnormality is more. The risk of warfarin embryopathy may be low when dose < 5 mg/day.

UFH doesn't cross placenta & is considered safer for fetus. But maternal osteopenia, hemorrhage, thrombocytopenia or thrombosis & (HITT) is common. For mechanical valve dose of UFH is 17500 to 20,000 unit twice daily but aPPT should be 2 to 3 times of control level. Lower doses required in prevention of various thromboembolism. UFH can be reversed with protamine sulfate.

LMWH produce more predictable anticoagulation response than UFH, & less likely to cause HITT. LMWH can be administered subcutaneously & dose to achieve an anti-factor Xa level of 1.0 to 1.2 unit/mL 4 to 6 hrs after injection. There is data to support that LMWH in deep vein thrombosis in pregnant women but safety & efficacy of LMWH in pregnant women with mechanical valve prosthesis are limited.

Anticoagulation in the pregnant patient can be difficult because of the risk profile associated with each drug regimen. In planned pregnancies, a careful discussion about the risks and benefits of warfarin, UFH, and LMWH will help the patient and physician involved to choose an anticoagulation strategy. Unplanned pregnancies are often diagnosed partway through the first trimester. It is advisable to stop warfarin when the pregnancy is discovered and to use UFH or LMWH, at least until after the 12 th week. Dosing regimens for warfarin, UFH, and LMWH may differ during any stage of pregnancy.

### Medication guideline during pregnancy

<table>
<thead>
<tr>
<th>Drug</th>
<th>Use</th>
<th>Potential side Effects</th>
<th>Safe During Pregnancy</th>
<th>Safe During Breast Feeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>Arrhythmias</td>
<td>None reported</td>
<td>Yes</td>
<td>No data</td>
</tr>
<tr>
<td>Beta blockers</td>
<td>Hypertension</td>
<td>Fetal brady-cardia, low birth weight, hypoglycemia, respiratory depression prolonged labor</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Hypertension</td>
<td>Low birth weight prematurity Reduced uteroplacental Perfusion depression</td>
<td>Unclear</td>
<td>Yes</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Arrhythmia, CHF</td>
<td>Low birth weight prematurity Reduced uteroplacental Perfusion depression, Neonatal CNS hemorrhage, Limited data</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Arrhythmia, anesthesia</td>
<td>Low birth weight prematurity Reduced uteroplacental Perfusion depression, Neonatal CNS hemorrhage, Limited data</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Low Molecular Weight Heparin</td>
<td>Mechanical valve hypercoagulable state, DVT, AF, Eisenmenger's syndrome</td>
<td>Hemorrhage, limited data</td>
<td>Limited data</td>
<td>Limited data</td>
</tr>
<tr>
<td>Nitrates</td>
<td>Hypertension</td>
<td>Foetal distress with Maternal hypotension</td>
<td>Yes</td>
<td>No data</td>
</tr>
<tr>
<td>Procainamide</td>
<td>Arrhythmia</td>
<td>None reported</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Unfractionated heparin</td>
<td>Mechanical valve hypercoagulable state, DVT, Eisenmenger's syndrome</td>
<td>Maternal osteoporosis hemorrhage thrombo-cytopenia, thrombosis,</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Warfarin</td>
<td>Mechanical valve, Hypercoagulable state, DVT, AF, Eisenmenger's syndrome</td>
<td>Warfarin embryopathy, fetal mortality, CNS abnormalities, hemorrhage</td>
<td>Yes, after week 12 of gestation</td>
<td>Yes</td>
</tr>
</tbody>
</table>

AF, arterial fibrillation; CHF, congestive heart failure; CNS, central nervous system; DVT, deep vein thrombosis; HCM, hypertrophic cardiomyopathy; MI, myocardial infarction.

AF, arterial fibrillation; CHF, congestive heart failure; CNS, central nervous system; DVT, deep vein thrombosis; HCM, hypertrophic cardiomyopathy; MI, myocardial infarction.
vary by diagnosis; detailed dosing guidelines have been published.

**Infective endocarditis – Prophylaxis.**

There is no universal agreement but many authorities recommend antibiotic prophylaxis during labor for pts at risk of infective endocarditis, specially in forceps delivery or episiotomy is performed. Ampicilin 2mg IV + gentamycin 1.5mg/kg IV/IM followed by amoxicillin 1.5 gm orally 6 hourly is recommended therapy. Recent guideline from ACC/AHA suggest that prophylaxis is unnecessary.

**SUMMARY:-**

- Heart disease during pregnancy encompasses a wide spectrum of disorder.
- Blood volume & cardiac output rises during normal pregnancy & reaches to peak the late 2nd trimester.
- Preexisting cardiac diseases should be evaluated with respect to the risk they impart during pregnancy.
- High risk lesion severe pulmonary hypertension, Eisenmenger’s syndrome cardiomyopathy with NYHA class III or IV symptoms, cyanotic congenital heart disease, Marfan syndrome with abnormal aorta & uncorrected severe valvular diseases pregnancy should be avoided.
- Hypertension & cardiac failure during pregnancy drugs that are contraindicated should be aware.
- Anticoagulation therapy during pregnancy should be carefully selected to avoid maternal & fetal side effects.
- Prophylaxis for IE should be given during labor & delivery.

**REFERENCES:-**

2. Cardiac risk in pregnant women with rheumatic mitral stenosis. Am J Cardiol. 91:2003;1382-1385