

Management of heart diseases in pregnancy

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cardiovascular changes associated with normal pregnancy

- **First trimester** (conception to 13+6 weeks of gestation) – Maternal systemic vasodilation begins at approximately 5 weeks of gestation.
 - Systemic vascular resistance (SVR) progressively drops by approximately 35 to 40 percent and nadirs in the **mid-second trimester** while cardiac output begins to rise.
- **Second trimester** (14 to 27+6 weeks of gestation) – The reduction in SVR that began in the first trimester ends in a **plateau in the middle of the second trimester**. Cardiac output continues to rise, but in a nonlinear fashion.

cardiovascular changes associated with normal pregnancy

- **Third trimester** (28 weeks of gestation to delivery) – Cardiac output peaks in the early third trimester.
- Heart rate, which rises throughout gestation, peaks in the **late third trimester** at an average of 16 beats per minute (bpm; 24 percent) above nonpregnant values.
- **Supine positioning** reduces cardiac output and stroke volume and **increases heart rate** due to compression of the aorta and vena cava from the enlarging uterus.

- **Placing the woman in the left lateral decubitus** position shifts the uterus off the aorta and vena cava which in turn increases blood flow to the heart and results in increased cardiac output and stroke volume.
- **Blood pressure (BP)** returns to prepregnancy levels during the third trimester.

cardiovascular changes associated with normal pregnancy

- **Intrapartum** – Cardiac output increases by 15 percent above prelabor levels in early labor and 25 percent during the active phase.
- During pushing in the second stage, cardiac output rises by 50 percent .
- Position changes from supine to lateral decubitus during labor increases cardiac output.
- **Postpartum** – Following birth, heart rate and BP returns to nonpregnant values and remains unchanged throughout the postpartum period

cardiovascular changes associated with normal pregnancy

- **Heart rate** — During normal pregnancy, the resting heart rate begins to rise in **the first trimester**, with an average increase of **10 to 30 bpm** (71 ± 10 bpm) having been reported.
- Heart rate then decreased slightly **at 40 weeks** to a median of 89 bpm (3 to 97 centiles: 65 to 114 bpm).
- Thus, the upper limit of the resting heart rate is typically **not greater than 115 bpm**, and those **exceeding 115 bpm** warrant evaluation

cardiovascular changes associated with normal pregnancy

Cardiac output

The cardiac output rises **30 to 50 percent** (1.8 L/min) above baseline during normal pregnancy.

Approximately **one-half** of the increase in cardiac output occurs by **eight weeks** of gestation

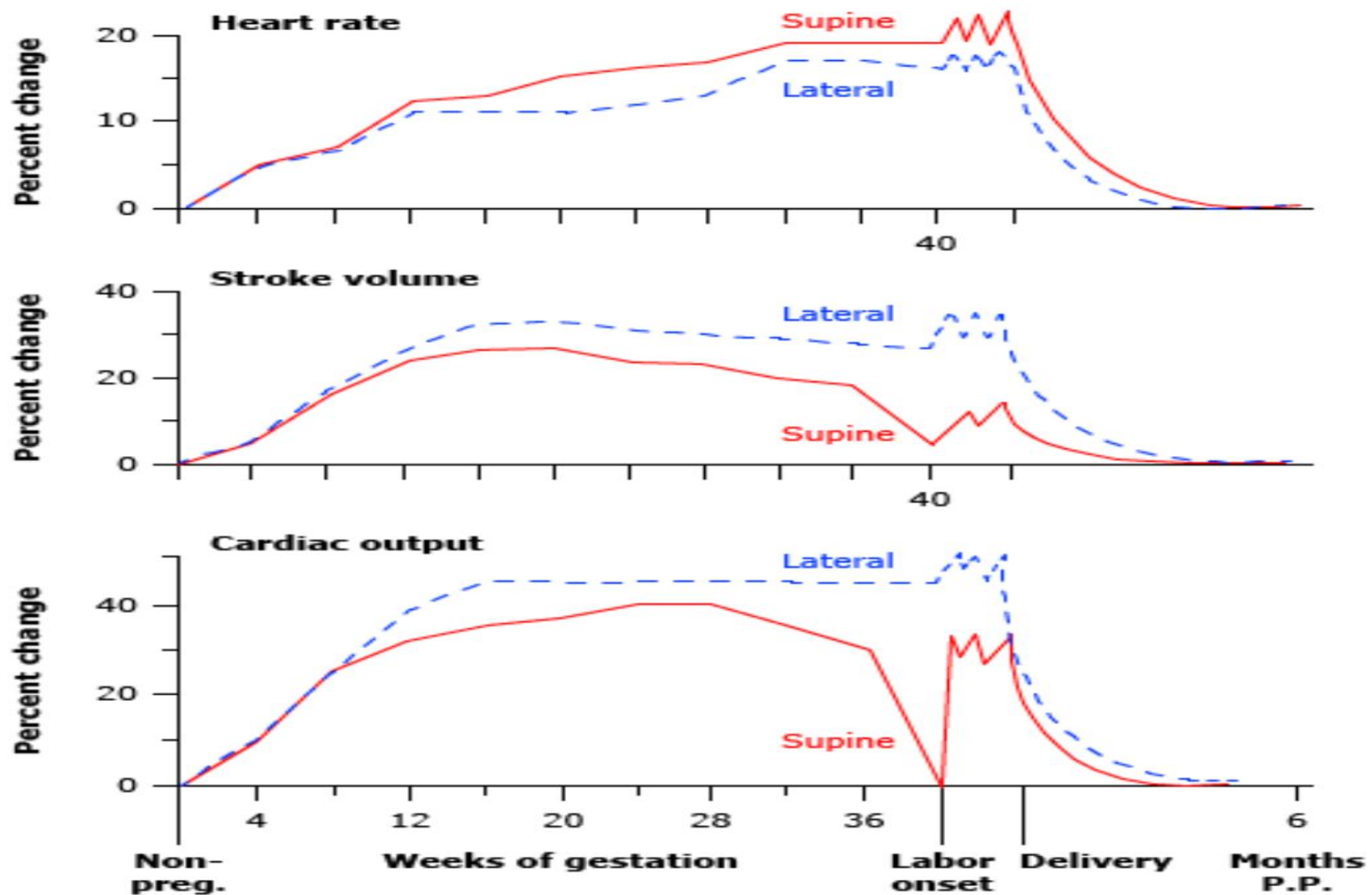
cardiovascular changes associated with normal pregnancy

Expansion of the plasma volume and an increase in red blood cell mass begin as early as the fourth week of pregnancy, peak at 28 to 34 weeks of gestation, and then plateau. Plasma volume expansion exceeds the increase in red cell volume, leading to "physiologic anemia."

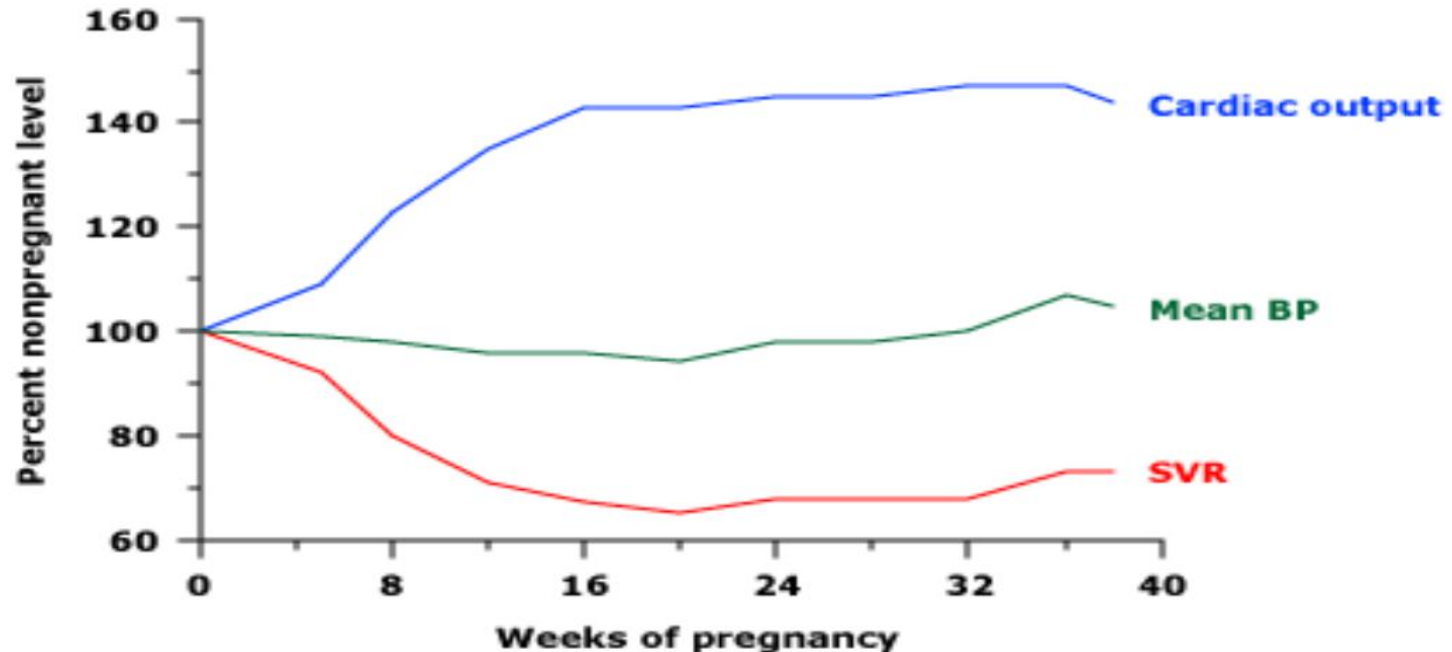
The major hemodynamic changes induced by pregnancy include an increase in cardiac output and reductions in systemic vascular resistance and systemic blood pressure.

Cardiac output peaks a few minutes after delivery, before gradually returning to prepregnancy levels.

Systemic hemodynamics during normal pregnancy



Hemodynamic changes in normal pregnancy



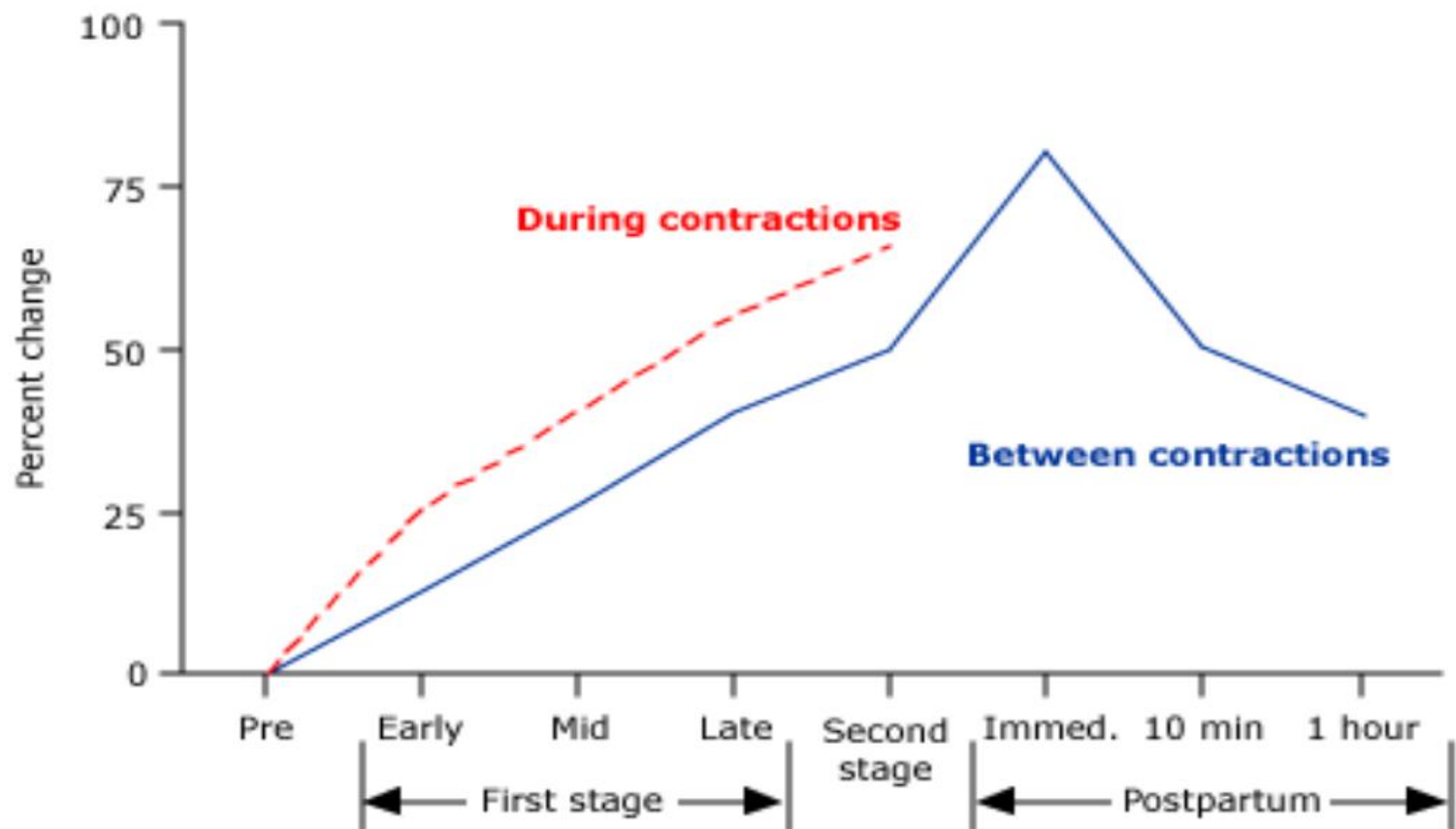
Normal pregnancy is characterized by an increase in cardiac output, a reduction in systemic vascular resistance, and minimal change in mean blood pressure. These changes are associated with a 10 to 15 beat/minute increase in heart rate.

Maternal cardiovascular changes during pregnancy by trimester

First trimester	Second trimester	Third trimester	Early postpartum
Decrease: <ul style="list-style-type: none"> SVR 	Decrease: <ul style="list-style-type: none"> SVR 	Decrease: <ul style="list-style-type: none"> SVR (with a late rise) LV longitudinal strain 	Decrease: <ul style="list-style-type: none"> Heart rate LVEDD LV mass Cardiac output
Increase: <ul style="list-style-type: none"> Heart rate LVEDD LV mass Cardiac output 	Increase: <ul style="list-style-type: none"> Heart rate LVEDD LV mass Cardiac output 	Increase: <ul style="list-style-type: none"> Heart rate LVEDD LV mass (with a late drop) Cardiac output (with a late drop) 	Increase: <ul style="list-style-type: none"> SVR LV longitudinal strain
No change: <ul style="list-style-type: none"> LV longitudinal strain 	No change: <ul style="list-style-type: none"> LV longitudinal strain 		

SVR: systemic vascular resistance; LV: left ventricular; LVEDD: left ventricular end diastolic diameter.

Cardiac output during normal labor, delivery, and postpartum



Maternal and Fetal Risks

- Serious maternal cardiac disease complicating pregnancy is relatively uncommon; however, it can have a significant adverse effect on maternal and fetal outcomes despite modern cardiac care.
- The overall incidence of serious heart disease complicating pregnancy is approximately 1%.

- Despite the potential for significant maternal morbidity, most patients with cardiac disease can expect a satisfactory outcome with careful antenatal, intrapartum, and postpartum management.

Maternal and Fetal Risks

- Serious complications during pregnancy and the postpartum period such as congestive heart failure, arrhythmias, and stroke are seen in 12% to 20% of patients.
- Mortality in some conditions can be as high as 30%. The rate of complications is related to several factors, including maternal functional status, myocardial dysfunction, significant aortic or mitral valve stenosis, and history of arrhythmias or a cardiac event.

Maternal and Fetal Risks

Fetal complications in pregnancies associated with maternal cardiac disease commonly include **growth restriction and preterm delivery**.

Despite the risk of low birth weight, **overall perinatal mortality is not significantly greater than** that in the general population.

When a pregnant patient has **congenital heart disease**, the fetus is at increased risk for this disease.

The increase in incidence ranges **from 0% to 18%**, depending on the specific lesion.

The risk of a cardiac lesion in the fetus is also increased when other **first-degree family** members have a congenital heart lesion.

Risk of Maternal and Fetal Morbidity Associated with Pregnancy

Mitral valve prolapse without severe regurgitation
Atrial and ventricular septal defect previously repaired or without pulmonary hypertension

Low risk

Corrected congenital heart disease without residual cardiac dysfunction

Patent ductus arteriosus

Pulmonary stenosis

Mild mitral or aortic valvular disease (stenosis or regurgitation) with normal left ventricular function:
New York Heart Association class I or II

Moderate Risk



Marfan's syndrome with normal aorta

History of peripartum cardiomyopathy
with no residual ventricular dysfunction

Previous myocardial infarction

High Risk

Any condition with New York Heart Association class III or IV

Moderate to severe systemic ventricular dysfunction

Pulmonary hypertension from any cause

Tetralogy of Fallot; uncorrected or with residual disease

Coarctation of the aorta

Mitral stenosis with atrial fibrillation

Severe aortic stenosis

Mechanical valve requiring anticoagulation

Marfan's syndrome with aortic involvement

History of peripartum cardiomyopathy with residual ventricular dysfunction

Management Options: *Prepregnancy*

Ideally in patients with significant heart disease, pregnancy is a **planned** event. This assumes regular and reliable use of an effective contraceptive method.

Before discontinuation of contraception, preconception evaluation and **counseling** should take place.

Maternal disease status should be determined. An **echocardiogram** can be used not only to define the cardiac anatomy but also to describe ventricular function and estimate intracardiac pressure gradients.

Magnetic resonance imaging can also be useful, for example, in visualizing the descending aorta in cases of **coarctation**.

Nuclear medicine scans, although helpful in the nonpregnant state, should be avoided during pregnancy.

Prepregnancy

A **careful history** is obtained to identify previous cardiac complications, including **arrhythmias**.

The patient's functional status should also be established.

The New York Heart Association (NYHA) functional classification system is commonly used.

Ninety percent or more of patients are categorized as having **class I or II disease**.

Outcomes are favorable in these two groups, but deterioration may occur. The reported frequency of adverse cardiac events **varies from 3% to 69% depending on patient risk factors**.

Although few patients have class III or IV disease, historically, **nearly 85% of maternal deaths occur in these groups**.

New York Heart Association Cardiac Functional Classification

Class I : No limitations of physical activity; ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.

Class II : Slight limitation of physical activity; ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain

Class III : Marked limitation of physical activity; less than ordinary activity causes fatigue, palpitation, dyspnea, or anginal pain.

Class IV : Inability to perform any physical activity without discomfort; symptoms of cardiac insufficiency or anginal syndrome may be present, even at rest; any physical activity increases discomfort.

Prenatal

Few patients are seen for pre-pregnancy evaluation. Therefore, most evaluation and counseling will be initiated **at the first prenatal visit.**

Cardiac surgery, although not contraindicated, is usually not required during pregnancy. If possible, it is best delayed **until postpartum.**

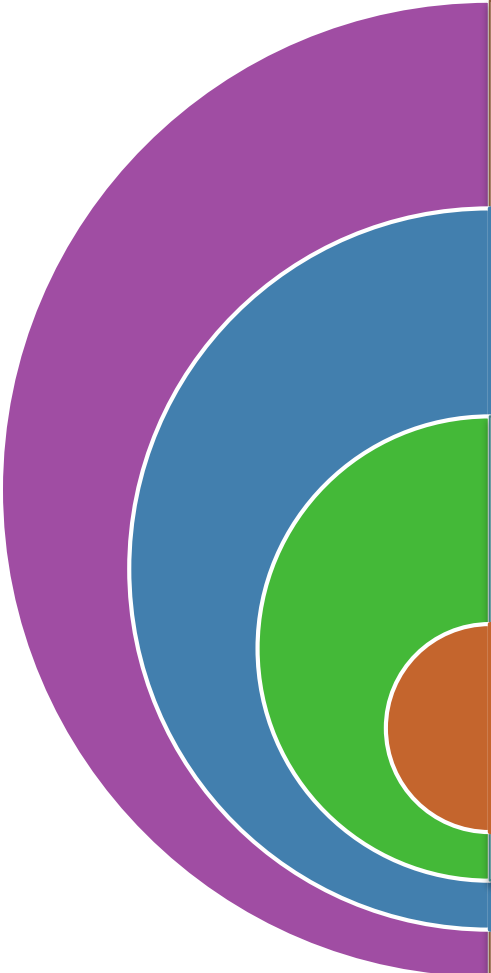
When the maternal mortality rate is excessive, as in Eisenmenger's syndrome, **termination of the pregnancy** should be discussed.

Prenatal

The **five predictive** indicators were NYHA:
classification greater than grade II
cyanosis
left ventricular obstruction
cardiac dysfunction
previous arrhythmia
and previous cardiac complication

Left ventricular outflow obstruction was defined as aortic valve stenosis with a valve area **less than 1.5 cm**, **mitral stenosis with a valve area of less than 2.0 cm**, or a peak left ventricular outflow tract gradient **greater than 30 mm Hg**

Prenatal



Myocardial dysfunction was defined as an ejection fraction less than **40%**, restrictive or **hypertrophic cardiomyopathy**, or **complex congenital heart disease**.

A *significant history of arrhythmia* was defined as symptomatic **bradyarrhythmia** or **tachyarrhythmia** requiring therapy.

If no predictive factors were present at the beginning of pregnancy, fewer than **5% of patients** have a **cardiac complication**.

When **one factor was present**, **30%** of patients have a complication, and when **two or more predictors** were present, nearly **70%** of patients have a complication. This information is useful in counseling patients

Prenatal

Medical management of the patient's cardiac condition should be optimized.

During maternal drug therapy, potential **fetal effects** must be considered.

Coexisting conditions that may aggravate preexisting heart disease, such as **anemia, arrhythmias, and hypertension,** should be appropriately treated and controlled.

Ideally, necessary **cardiac surgery** is carried out before conception.

Prenatal

During prenatal care, the patient should be routinely questioned and examined for signs or symptoms of cardiac failure.

Vital signs and weight gain should be closely monitored.

When there is an increased risk of intrauterine growth restriction (IUGR), serial ultrasound examinations **every 2 to 4 weeks** in the third trimester allow assessment of interval fetal growth.

Prenatal

Antenatal testing may begin **at 32 to 34 weeks** unless earlier surveillance is indicated because of compromised maternal or fetal status.

Anesthesiology consultation should be obtained prior to delivery.

Future **fertility desires and contraceptive** plans should be addressed in the antepartum period.

Topics should include a discussion of **sterilization**, depending on future fertility desires, the maternal risk due to pregnancy, and the long-term prognosis.

Prenatal

The most common significant intercurrent events during the antepartum period are **febrile episodes**.

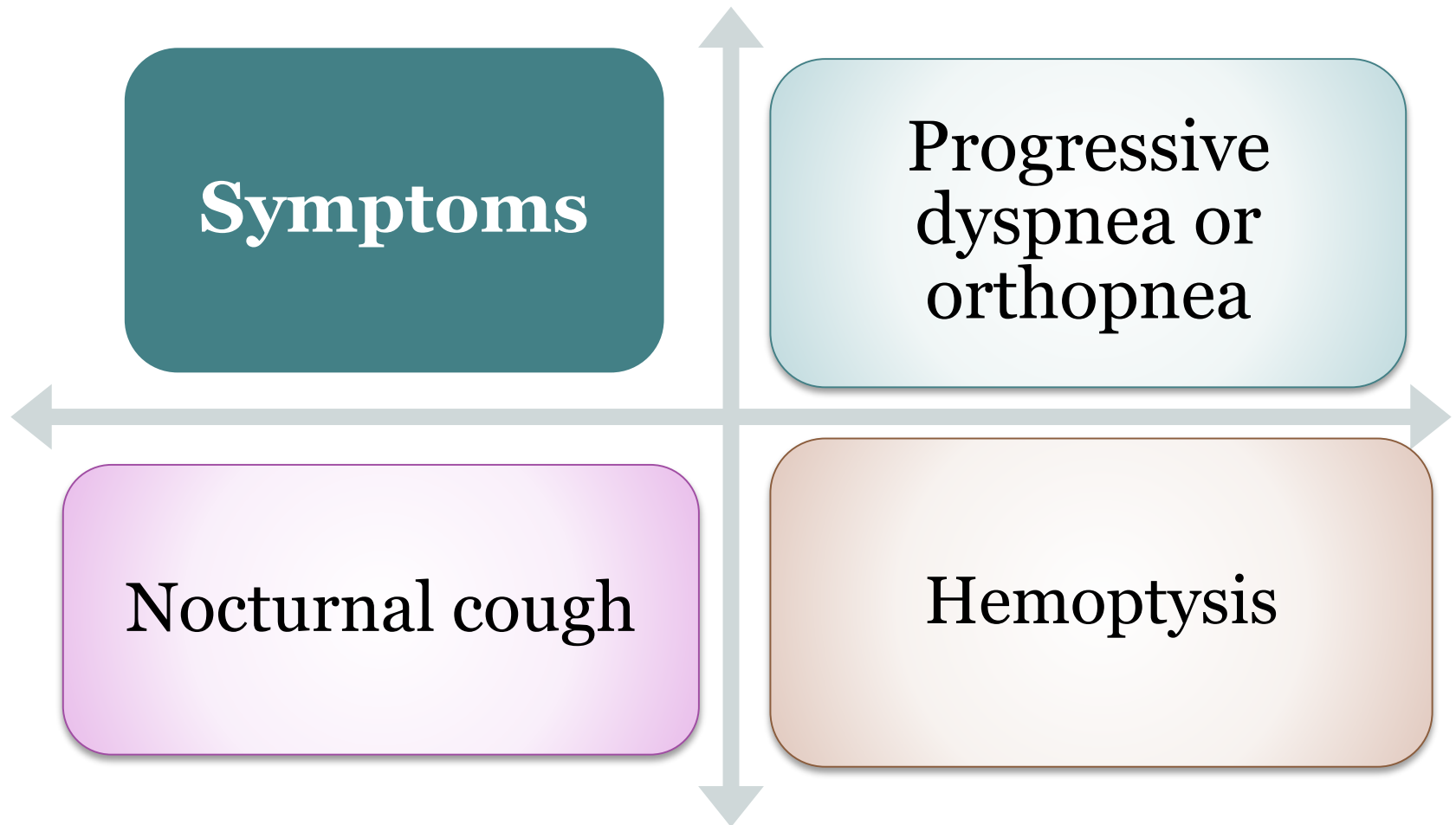
Screening for **bacteriuria and vaccination** against influenza and pneumococcus are appropriate.

Patients should be instructed to report symptoms of upper respiratory infection, particularly fever.

Many women with heart disease (adolescents, recent immigrants, and those living in poverty) are also at **risk for iron deficiency**.

Prophylaxis against anemia with iron and folate supplementation may decrease cardiac work.

Clinical Indicators of Heart Disease During Pregnancy

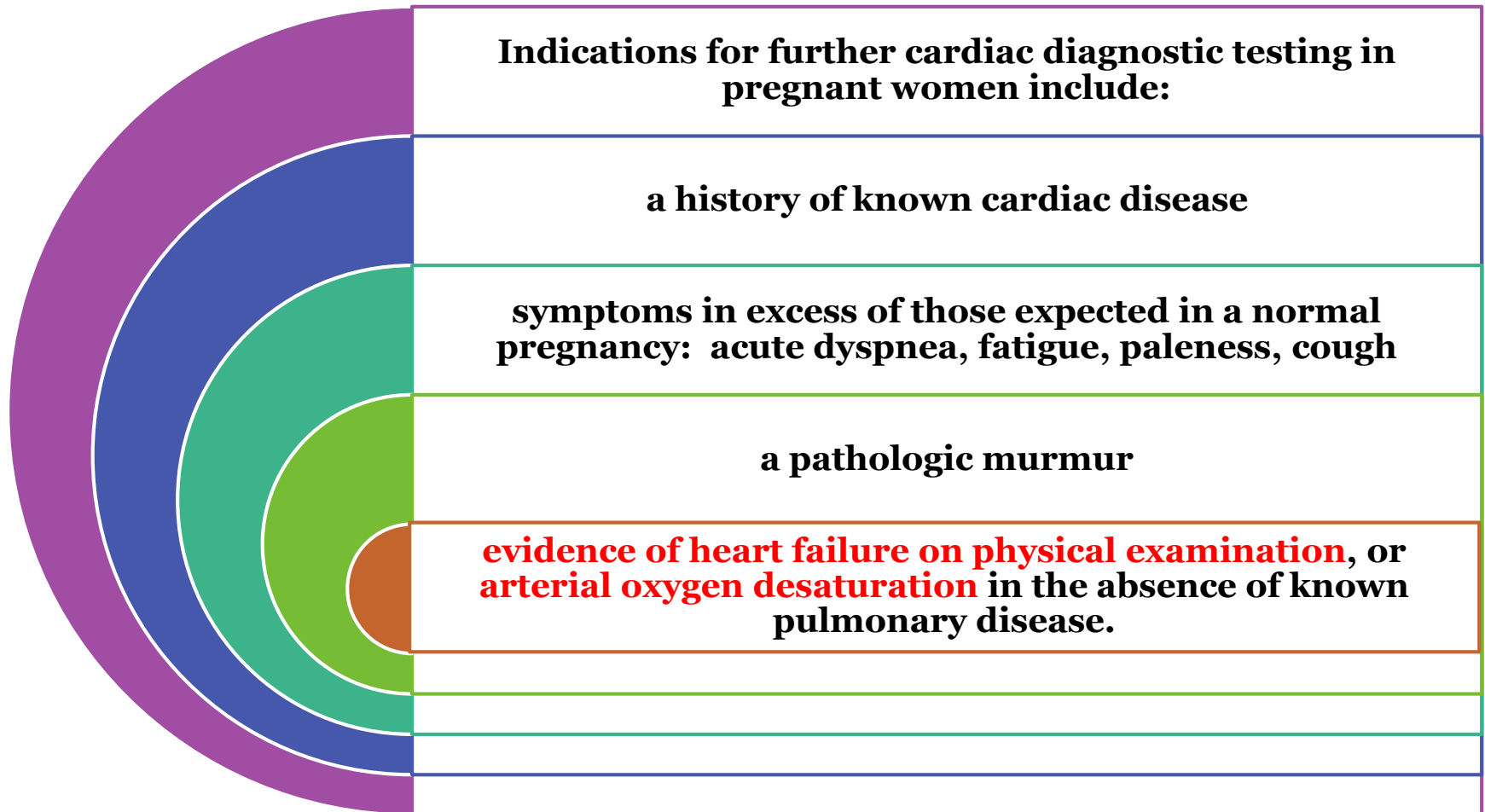


Clinical Indicators of Heart Disease During Pregnancy

➤ Clinical Findings

- Cyanosis
- Clubbing of fingers
- Persistent neck vein distention
- Systolic murmur grade 3/6 or greater
- Diastolic murmur
- Cardiomegaly
- Persistent tachycardia and/or arrhythmia
- Persistent split second sound
- Fourth heart sound
- Criteria for pulmonary hypertension

Diagnosis and Evaluation of Heart Disease



Modified World Health Organization (WHO) classification

class I—uncomplicated, mild pulmonary stenosis

class II— unoperated ASD or VSD and repaired tetralogy of Fallot

class III—mechanical valves, systemic right ventricle, Fontan circulation, unrepaired cyanotic heart disease, other complex congenital heart disease, Marfan syndrome with an aorta 40 to 45 mm in width, and bicuspid aortic valve with aorta 45 to 50 mm

class IV—pulmonary hypertension/Eisenmenger syndrome, systemic EF less than 30%, systemic dysfunction NYHA class III–IV, severe mitral stenosis, severe symptomatic aortic stenosis, Marfan syndrome with aorta greater than 45 mm, bicuspid valve with aorta greater than 50 mm, and native severe coarctation(containdiacation)

	mWHO I	mWHO II	mWHO II–III	mWHO III	mWHO IV
Diagnosis (if otherwise well and uncomplicated)	<p>Small or mild</p> <ul style="list-style-type: none"> – pulmonary stenosis – patent ductus arteriosus – mitral valve prolapse <p>Successfully repaired simple lesions (atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage)</p> <p>Atrial or ventricular ectopic beats, isolated</p>	<p>Unoperated atrial or ventricular septal defect</p> <p>Repaired tetralogy of Fallot</p> <p>Most arrhythmias (supraventricular arrhythmias)</p> <p>Turner syndrome without aortic dilatation</p>	<p>Mild left ventricular impairment (EF >45%)</p> <p>Hypertrophic cardiomyopathy</p> <p>Native or tissue valve disease not considered WHO I or IV (mild mitral stenosis, moderate aortic stenosis)</p> <p>Marfan or other HTAD syndrome without aortic dilatation</p> <p>Aorta <45 mm in bicuspid aortic valve pathology</p> <p>Repaired coarctation</p> <p>Atrioventricular septal defect</p>	<p>Moderate left ventricular impairment (EF 30–45%)</p> <p>Previous peripartum cardiomyopathy without any residual left ventricular impairment</p> <p>Mechanical valve</p> <p>Systemic right ventricle with good or mildly decreased ventricular function</p> <p>Fontan circulation.</p> <p>If otherwise the patient is well and the cardiac condition uncomplicated</p> <p>Unrepaired cyanotic heart disease</p> <p>Other complex heart disease</p> <p>Moderate mitral stenosis</p> <p>Severe asymptomatic aortic stenosis</p> <p>Moderate aortic dilatation (40–45 mm in Marfan syndrome or other HTAD; 45–50 mm in bicuspid aortic valve, Turner syndrome ASI 20–25 mm/m², tetralogy of Fallot <50 mm)</p> <p>Ventricular tachycardia</p>	<p>Pulmonary arterial hypertension</p> <p>Severe systemic ventricular dysfunction (EF <30% or NYHA class III–IV)</p> <p>Previous peripartum cardiomyopathy with any residual left ventricular impairment</p> <p>Severe mitral stenosis</p> <p>Severe symptomatic aortic stenosis</p> <p>Systemic right ventricle with moderate or severely decreased ventricular function</p> <p>Severe aortic dilatation (>45 mm in Marfan syndrome or other HTAD, >50 mm in bicuspid aortic valve, Turner syndrome ASI >25 mm/m², tetralogy of Fallot >50 mm)</p> <p>Vascular Ehlers–Danlos</p> <p>Severe (re)coarctation</p> <p>Fontan with any complication</p>

Risk	No detectable increased risk of maternal mortality and no/mild increased risk in morbidity	Small increased risk of maternal mortality or moderate increase in morbidity	Intermediate increased risk of maternal mortality or moderate to severe increase in morbidity	Significantly increased risk of maternal mortality or severe morbidity	Extremely high risk of maternal mortality or severe morbidity
Maternal cardiac event rate	2.5–5%	5.7–10.5%	10–19%	19–27%	40–100%
Counselling	Yes	Yes	Yes	Yes: expert counselling required	Yes: pregnancy contraindicated: if pregnancy occurs, termination should be discussed
Care during pregnancy	Local hospital	Local hospital	Referral hospital	Expert centre for pregnancy and cardiac disease	Expert centre for pregnancy and cardiac disease
Minimal follow-up visits during pregnancy	Once or twice	Once per trimester	Bimonthly	Monthly or bimonthly	Monthly
Location of delivery	Local hospital	Local hospital	Referral hospital	Expert centre for pregnancy and cardiac disease	Expert centre for pregnancy and cardiac disease

Type	Cause	Pathophysiology	Pregnancy
Mitral stenosis	Rheumatic valvulitis	LA dilation and passive pulmonary hypertension Atrial fibrillation	Heart failure from fluid overload, tachycardia
Mitral insufficiency	Rheumatic valvulitis Mitral valve prolapse LV dilation	LV dilation and eccentric hypertrophy	Ventricular function improves with afterload decrease
Aortic stenosis	Congenital bicuspid valve	LV concentric hypertrophy, decreased cardiac output	Moderate stenosis is tolerated; severe is life-threatening with decreased preload, e.g., obstetrical hemorrhage or regional analgesia
Aortic insufficiency	Rheumatic valvulitis Connective tissue disease Congenital	LV hypertrophy and dilation	Ventricular function improves with afterload decrease
Pulmonary stenosis	Rheumatic valvulitis Congenital	Severe stenosis associated with RA and RV enlargement	Mild stenosis usually well tolerated; severe stenosis associated with right heart failure and atrial arrhythmias

Timing and mode of delivery: risk for mother and child

A **delivery plan** should be made with details of induction, management of labor, delivery, and postpartum surveillance.

Specific expertise and collaborative management by a **pregnancy heart team** in specialist centers is mandatory for all moderate- and high-risk patients.

Women with cardiac disease have an increased risk of **obstetric complications**, including **premature labor**, **pre-eclampsia**, and **postpartum hemorrhage**.

Timing of delivery

Induction of labor should be considered **at 40 weeks** of gestation in all women with cardiac disease.




Timing of induction will depend on **cardiac status**, obstetric evaluation including cervical assessment, fetal well-being, and fetal lung maturity.



Physiologically, the ideal labor for a woman with heart disease is **short and pain free.**

Labor induction

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Both **misoprostol** [25 microg, prostaglandin **E1** (PGE1)] or Dinoprostone [1–3 mg or slow-release formulation of 10 mg (PGE2)] can be used safely to induce labor.

Reassuringly, in women without heart disease, high-dose (**600 microg**) **misoprostol** has no effect on cardiac parameters, although there remains a theoretical risk of **coronary vasospasm and arrhythmias**.

Labor induction

Dinoprostone may cause profound **hypotension**, but only when injected blindly into the myometrium, and this route of administration should be avoided.

Artificial **rupture of membranes** and **infusion of oxytocin** can be used safely in women with heart disease.

Mechanical methods such as a cervical ripening balloon might be preferable in patients where a drop in systemic vascular resistance would be detrimental.

Vaginal or caesarean delivery

elective caesarean section carries no maternal benefit and results in earlier delivery and lower birth weight.

Vaginal delivery is associated with less blood loss and lower risk of infection, venous thrombosis, and embolism, and should be advised for most women.

Caesarean section should be considered for obstetric indications and for patients presenting in labor **on oral anticoagulants (OACs), with aggressive aortic pathology, sever MS, and in acute intractable HF, dilated aortic root >4cm, recent myocardial infarction**

Caesarean section is advised in **severe forms of PH** (including Eisenmenger's syndrome).

Delivery in anticoagulated women (not including mechanical valve)

For women with a planned caesarean section, therapeutic low molecular weight heparin (LMWH) dosing can be simply omitted for 24 h prior to surgery.

In high-risk women, therapeutic UFH can be restarted at 6 h post-delivery.

In women at moderate or low-risk, a single prophylactic dose of LMWH-for example, in the case of enoxaparin, 20 mg if weight is <50 kg, 40 mg if 50–90 kg, and for women with a raised body mass index (BMI) 0.5 mg/kg-can be given at 6 h post-delivery, before restarting therapeutic LMWH 12 h later.

Delivery in anticoagulated women (not including mechanical valve)

If vaginal delivery is planned, moderate- and high-risk patients can be converted to an **infusion of UFH with regular checks of aPTT to optimize control**, and the infusion **stopped at least 4–6 hours prior** to insertion of regional anesthesia or anticipated delivery.

For women at low-risk, therapeutic LMWH can be omitted **for 24 h** prior to anticipated delivery.

STANDARD CARDIAC CARE FOR LABOR AND DELIVERY

1. Accurate diagnosis

2. Mode of delivery based on obstetric indications

3. Maintenance of hemodynamic stability

Invasive hemodynamic monitoring when required

4. Avoidance of pain and hemodynamic responses

Epidural analgesia with narcotic/low-dose local technique

5. Prophylactic antibiotics when at risk for endocarditis as per American Heart Association and American College of Obstetricians and Gynecologists guidelines

STANDARD CARDIAC CARE FOR LABOR AND DELIVERY

6. Low or outlet forceps or vacuum delivery if goal is to avoid pushing

7. Avoidance of maternal blood loss

Proactive management of the third stage

Early but appropriate fluid replacement

8. Close observation during postpartum period

Early volume management postpartum balancing hypovolemia (anemia) and hypervolemia (redistribution of volume) risks

Often careful but aggressive diuresis

Urgent delivery on therapeutic anticoagulation

Delivery in a patient taking therapeutic anticoagulation carries a high risk of **maternal hemorrhage**.

For UFH, **protamine sulfate should be given**, the exact dose depending on the mode of administration and time since the last dose of UFH .

In the case of LMWH, protamine sulfate should be given; however, not only may anti factor Xa activity remain prolonged and bleeding tendency persist, but the half-life of LMWH is longer and absorption after subcutaneous injection is prolonged, **such that repeated doses or an infusion of protamine sulfate may be required**.

If the patient is on OACs, caesarean section is preferred to reduce the risk of fetal **intracranial hemorrhage**.

Urgent delivery on therapeutic anticoagulation

1mg of protamine sulfate will usually neutralize at least 100 international units heparin ..

1-1.5 mg per 100 USP units of heparin; not to exceed 50 mg(<30 min)

For example, if 30-120 minutes have elapsed since heparin was injected intravenously, 0.5-0.75mg protamine sulfate per 100 units of heparin is recommended.

If two hours or more have elapsed, 0.25-0.375mg per 100 units of heparin should be administered.

Urgent delivery on therapeutic anticoagulation

Patients should be carefully monitored using either the activated **partial thromboplastin time** or the **activated clotting time**, carried out **5-15 minutes after protamine sulfate administration.**

Further doses may be needed because protamine is cleared from the blood more rapidly than heparin.

Urgent delivery on therapeutic anticoagulation

Neutralization of low molecular weight (LMW) heparins:

Enoxaparin :

- 1 mg per mg enoxaparin (if enoxaparin overdose given within 8 hr); if >8 hr of overdose or bleeding continues after 4 hr after first dose, give 0.5 mg protamine per mg enoxaparin.
- Intermittent injections or continuous infusion of protamine sulfate have been recommended for the neutralization of LMW heparin following subcutaneous administration, as there may be continuing absorption from the subcutaneous depot.

Urgent delivery on oral therapeutic anticoagulation

Reversal of anticoagulation is better with four-factor prothrombin complex concentrate, best given as an individualized dose dependent on **maternal weight, initial international normalized ratio (INR), and target INR** than fresh frozen plasma (**12–15 mL/kg**), and should be given prior to caesarean delivery to achieve an **INR < 1.5** .

Vitamin K (**5–10 mg iv**) may also be given, but may take up to 8–12 h to reverse the INR and has a persistent effect making reanticoagulation more difficult.

Urgent delivery on therapeutic anticoagulation

The fetus may remain anticoagulated for **8–10 days after discontinuation** of maternal OACs, and may need to be given fresh frozen plasma as well as vitamin K.

Woman with mechanical valve and HIGH dose VKA
(warfarin >5 mg/day or phenprocoumon >3 mg/day or acenocoumarol >2 mg/day)
who contemplates pregnancy: Pre-pregnancy counselling - Continue VKA antagonist until pregnant

PREGNANT

1st
trim.

Continue VKA, monitor INR
at least 2-weekly (IIb)

OR

In-hospital change
to i.v. UFH aPTT $\geq 2\times$ control
(IIa)^a

OR

In-hospital change to
LMWH 2-daily, close
monitoring (IIa)^{a,b}

2nd/3rd
trim.

Continue VKA, monitor INR
at least 2-weekly (IIa)

In-hospital change from
LMWH/UFH to VKA (IIa).
When on target INR, monitor
INR at least 2-weekly

Continue LMWH 2-daily
close monitoring (IIb)^b

36
weeks

In-hospital change to i.v. UFH aPTT $\geq 2\times$ control (I);
or in-hospital change to LMWH 2-daily or continue LMWH, close monitoring^b (I)

36 hrs before
planned
delivery

i.v. UFH (aPTT $\geq 2\times$ control) (I)

Delivery

stop UFH 4-6 hours before delivery and restart 4-6 hours after delivery if no bleeding

Woman with mechanical valve and LOW dose VKA
(warfarin <5 mg/day or phenprocoumon <3 mg/day or acenocoumarol <2 mg/day)
who contemplates pregnancy: Pre-pregnancy counselling - Continue VKA antagonist until pregnant

PREGNANT

1st
trim.

Continue VKA, monitor INR
at least 2-weekly (IIa)

OR

In-hospital change to LMWH
2-daily, close monitoring
(IIb)^{a,b}

OR

In-hospital change to i.v. UFH
aPTT $\geq 2\times$ control) (IIb)^a

2nd/3rd
trim.

Continue VKA, monitor INR
at least 2-weekly (I)

In-hospital change from
LMWH to VKA (I).
When on target INR, monitor
INR at least 2-weekly

In-hospital change from UFH
to VKA (I).
When on target INR monitor
INR at least 2-weekly

36
weeks

In-hospital (change to) i.v. UFH aPTT $\geq 2\times$ control) (I)
or in-hospital change to LMWH 2-daily, close monitoring^b (I)

36 hrs before
planned
delivery

i.v. UFH (aPTT $\geq 2\times$ control) (I)

Delivery

stop UFH 4-6 hours before delivery and restart 4-6 hours after delivery if no bleeding

Hemodynamic monitoring during delivery

Maternal BP and heart rate should be monitored in all patients with cardiac disease. In women with more severe heart disease, an **arterial line** provides more accurate data.

Pulse oximetry and continuous ECG monitoring are advised to detect early signs of decompensation, and to identify those in whom delivery should be expedited.

In some high-risk patients (PH), **right atrial pressure monitoring** may be considered

Anesthesia/analgesia

Epidural analgesia reduces labor pain and can be used to provide anesthesia for caesarean section if necessary.

However, it can **cause systemic hypotension** (10%) and must be carefully titrated, especially in patients with **obstructive valve lesions or** diminished ventricular function who may benefit from invasive BP monitoring.

All IV fluids need to be used carefully.

Labor

Mobilization may facilitate fetal head descent and a **lateral decubitus position** can attenuate the hemodynamic impact of cava compression by the gravid uterus.

Assisted delivery with **forceps** or a **ventouse** may be used to further reduce maternal effort, as indicated by the underlying cardiac lesion.

Continuous electronic fetal heart rate monitoring is recommended.

Post-partum care

A **slow IV infusion of oxytocin** reduces the risk of post-partum hemorrhage and has a minimal impact on cardiovascular parameters.

PGE₁ analogues [sulprostone (100–500 microg/h) and misoprostol (200–1000 microg)] can be used to treat postpartum hemorrhage; however, **ergometrine and prostaglandin F analogues** should be avoided.

Meticulous leg care, elastic support stockings, and early ambulation are important to reduce the risk of thrombo-embolism.

Post-partum care

post-partum period is associated with significant hemodynamic changes and fluid shifts, particularly in the first 24–48 h after delivery, which may precipitate HF. Hemodynamic monitoring should therefore be continued for at least 24–48 h in those at risk.

Breastfeeding

Lactation is associated with a **low-risk of bacteremia** secondary to **mastitis** and should be encouraged in patients with heart disease whenever possible.

Most drugs used in patients enter the milk and could thus contraindicate breastfeeding.

Methods of contraception

The risk of using a particular type of contraception needs to be balanced against the risk of pregnancy, estimated using the modified WHO classification which assesses the risk with each method for a given medical condition.

Advice is best provided by cardiologists with appropriate training or obstetricians, and should be given from the time of menarche since an **unplanned pregnancy** has to be avoided.

Hormonal contraception can have important non-contraceptive benefits, including the control of menstruation, prevention of anemia, reduction of dysmenorrhea, and of hyperandrogenism.


Methods of contraception

Ethinylestradiol-containing contraceptives have the greatest risk of **thrombosis** and are not advised in women with **high-risk of thrombo-embolic disease**; they also increase BP and are contraindicated in pre-existing hypertension.

Progestin-only contraceptives are an alternative, since they have little (implant or depot injection) or no (levonorgestrel-loaded intrauterine device or oral desogestrel) effect on coagulation factors, BP, and lipid levels.

Oral desogestrel inhibits ovulation, which could be an advantage for patients with polycystic ovary syndrome, endometriosis, or dysfunctional uterine bleeding.

Methods of contraception



Levonorgestrel-based long-acting reversible contraception implants or intrauterine devices are the safest and most effective contraceptives.

However, intrauterine device insertion may cause a **vasovagal** response; consequently, this should be performed in a **hospital setting**, particularly for **Fontan and Eisenmenger's syndrome patients**.

The levonorgestrel-releasing intrauterine device reduces periods, causing amenorrhea in **<_60% of women**, in contrast to copper intrauterine devices, which cause heavier periods.

The newer, smaller levonorgestrel-based intrauterine devices are easier to insert, reducing the **risk of pain and therefore vasovagal response**.

Methods of contraception

Barrier methods are unreliable but reduce the risk of pelvic inflammatory disease.

A good approach is the combination of **barrier methods and long-acting reversible contraception** (levonorgestrel-based long-acting reversible contraception, progestin-releasing implant, or progestin-releasing intrauterine devices).

For emergency contraception, a **copper intrauterine device** is most effective and additionally provides ongoing contraception.

Alternatively, a single dose of **1.5 mg levonorgestrel is effective if** taken within 72 h after unprotected sex (1.1% failure rate), with no evidence of increased rates of thrombosis.

The progesterone receptor modulator ulipristal acetate (UPA) has been shown to be more effective than levonorgestrel.

UPA is not associated with an increased risk of thrombosis.

Sterilization

Sterilization by tubal ligation is not unreasonable if pregnancy is contraindicated or the family is complete.

Laparoscopy is not without risks in patients with PAH, cyanosis, and a Fontan circulation, and the risks are probably lower with the hysteroscopic method performed under regional anesthesia.

Vasectomy is an option.

Methods of termination of pregnancy

Pregnancy termination should be discussed if there is a high-risk of maternal morbidity or mortality, and/or of fetal abnormality.

Both medical and surgical methods are effective with similar rates of major complications, but the greater need for unanticipated operative evacuation (2.1 vs. 0.6%) favors the surgical approach in this group of women.

Medical terminations can be considered **up to 9 weeks** of gestation using a reduced misoprostol dose of 100 microg.(avoid saline infusion)

Antibiotics are given to reduce the risk of endometritis and these should be modified to provide endocarditis prophylaxis.

Endocarditis Prophylaxis

The American College of Obstetricians and Gynecologists does not recommend endocarditis prophylaxis for either **vaginal or cesarean delivery** **in the absence of pelvic infection** except with the lesions cited above.

Women at highest risk for endocarditis are those with cyanotic cardiac disease, prosthetic valves, or both.

When indicated, and for women not already receiving **intrapartum antimicrobial therapy** for another indication that would also provide coverage against endocarditis

These are administered as close to **30 to 60 minutes** before the anticipated delivery time as is feasible.

Endocarditis Prophylaxis

American College of Obstetricians
and Gynecologist :



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graph TD; A[American College of Obstetricians and Gynecologist :] --> B[Standard (IV): ampicillin 2 g or cefazolin or ceftriaxone 1 g]; B --> C[Penicillin-allergic (IV): cefazolin or ceftriaxone 1 g or clindamycin 600 mg]; C --> D[Oral: amoxicillin 2 g];
```

Standard (IV): ampicillin 2 g *or*
cefazolin or ceftriaxone 1 g

Penicillin-allergic (IV): cefazolin or
ceftriaxone 1 g *or* clindamycin 600 mg

Oral: amoxicillin 2 g

Endocarditis Prophylaxis

American Heart Association/European Society of Cardiology

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graph TD; A[American Heart Association/European Society of Cardiology] --> B[Standard: amoxicillin 2 g PO or ampicillin 2 g IV or IM]; B --> C[Penicillin-allergic: clarithromycin or azithromycin 500 mg PO; cephalexin 2 g PO;]; C --> D[clindamycin 600 mg PO, IV, or IM; or cefazolin or ceftriaxone 1 g IV or IM];
```

Standard: amoxicillin 2 g PO **or** ampicillin 2 g IV or IM

Penicillin-allergic: clarithromycin or azithromycin 500 mg PO; cephalexin 2 g PO;

clindamycin 600 mg PO, IV, or IM; **or** cefazolin or ceftriaxone 1 g IV or IM

Educate the mother

- Limit daily activities if needed and according to the heart disease class.
- Patients in class one or two need **10 hours of sleep** a night and **30 minutes of rest after meals**.
- Patients in class Four usually need to **rest in bed most of the day**.
- **Avoid hot and humid** environments as it may worsen heart failure.
- Prescribe cardiovascular drugs regularly and correctly.
- stop **smoking** if used. Drug addicts, especially cocaine, are more likely to have heart attacks.
- **Prevent constipation** and straining during defecation by eating sufficient amounts of fluids and foods containing fiber. Laxatives are also useful if needed.
- Patients taking warfarin Talk to your doctor about the type of fiber in your diet.
- **Avoid contact** with people with **respiratory infections**.
- To help better blood circulation to be in the correct position of the **body(left lateral decubitus)**
- **Avoid gaining too much weight**.

THANK YOU FOR YOUR ATTENTION

