

## Cardiac Disease in Pregnancy

The following section is entitled “**Cardiac Disease in Pregnancy**”. This section deals with some of the basic concepts important in the diagnosis, management and investigation of cardiac disease during pregnancy. The section begins with a *learner handout* with space for the learner to make their own notes. The *learner handout* is followed by the *teaching script* for the educator. The section then concludes with several cases for discussion and a brief bibliography for this topic.

# CARDIAC DISEASE IN PREGNANCY

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### Physiologic Changes of Pregnancy

- Blood volume and cardiac output rise in pregnancy to a peak that is 150% of normal by 24-28 weeks gestation.
- Systemic vascular resistance drops significantly during pregnancy.
- The gravid uterus can dramatically affect venous return to the heart (preload) in some positions.

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### Physiologic Changes of Pregnancy

- Cardiac disease can be unmasked or worsen in pregnancy because of the increased cardiac demands of the gravid state.
- Particular periods of high risk for cardiac decompensation are:
  - when blood volume peaks at the end of the second trimester
  - during the work of labor
  - with fluid shifts that occur postpartum.

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## CARDIAC DISEASE IN PREGNANCY

### Palpitations

- Pregnant women commonly experience palpitations after exertion or when supine. Most often they will have sinus tachycardia or ectopic beats.
- Increased baseline heart rate, contractility, and catecholamine levels, and shift of the heart closer to the anterior chest wall can explain some of the symptoms.

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### Palpitations

- Increased body awareness and exposure to health care providers may contribute to the increased reporting of palpitations in pregnancy as well.
- Fast regular heart racing that runs for greater than several minutes and is associated with lightheadedness is more likely to be due to a significant tachyarrhythmia and always warrants a workup.

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### Arrhythmias in Pregnancy

- Pregnancy may increase the frequency of SVT in women with a history of SVT prior to pregnancy.
- SVT can be safely treated with adenosine in pregnancy.
- DC cardioversion can be safely carried out during pregnancy if the patient is unstable.

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# CARDIAC DISEASE IN PREGNANCY

## Structural Cardiac Disease

The course of structural heart disease in pregnancy is best predicted by the NYHA classification for cardiac function.

NYHA Classification	Prognosis for Pregnancy
class I	good
class II	good
class III	moderate; may need hemodynamic monitoring and special anesthetic management
class IV	poor; will need peripartum hemodynamic monitoring and special anesthetic management

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## Structural Cardiac Disease

- Patients with stenotic valves tend to have increased symptoms and more potential for morbidity during pregnancy.
- Incompetent valves tend to have an improvement in their symptoms during pregnancy.

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## Structural Cardiac Disease

- Severe pulmonary hypertension greater than 80mm Hg and Eisenmenger's syndrome carry an extremely high risk of maternal mortality in pregnancy.

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## CARDIAC DISEASE IN PREGNANCY

### **Congenital Heart Disease**

- **Women who have undergone repair seem to tolerate pregnancy very well.**
- **Risk of maternal and/or fetal complications is higher with:**
  - **NYHA Class III or IV**
  - **Maternal cyanosis or erythrocytosis**
  - **Stenotic lesions**
  - **Presence of a right to left shunt**

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### **Structural Cardiac Disease**

- **SBE prophylaxis is not officially recommended for normal spontaneous vaginal delivery or cesarean sections.**

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### **Ischemic Heart Disease**

- **Although uncommon in pregnancy, ischemic heart disease can manifest itself in pregnancy, especially in those women with Type 1 diabetes for over 10 years.**
- **Stress echocardiograms are probably the best stress test in pregnancy although EST, Thallium scans, Dobutamine Echo testing and coronary angiograms have all been done safely in pregnancy.**
- **CPK-MB can be elevated after a routine cesarean section.**

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## CARDIAC DISEASE IN PREGNANCY

### Peripartum Cardiomyopathy

- Peripartum cardiomyopathy is a cardiomyopathy that occurs in the third trimester or in the months following delivery and presents with congestive heart failure.
- The etiology is poorly understood.
- Treatment must include anticoagulation because of the high risk of thromboembolism.
- Over one-third of patients have complete recovery.
- A risk of recurrence exists in subsequent pregnancies.

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### Cardiac Resuscitation

- CPR can be performed on a pregnant woman
  - have someone pull the woman's uterus to the left side to decrease IVC compression and thereby improve venous return
  - DC cardioversion can be done safely in pregnancy but fetal monitoring devices must be removed first.
- If after 5 minutes of CPR no response has occurred, an emergent C/section may help improve maternal outcome.

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# CARDIAC DISEASE IN PREGNANCY

## *Teaching Script*

### **Physiologic Changes in Pregnancy**

Three main changes in cardiac physiology occur during pregnancy. First, blood volume and cardiac output rise to a peak at 24 to 28 weeks gestation that is 150% of normal. After that point in pregnancy, blood volume and cardiac output remain stable until delivery. By the second week postpartum, non pregnant cardiac physiology has been re-established. Second, systemic vascular resistance decreases in pregnancy by 25%. This can improve the status of some cardiac lesions such as aortic insufficiency which may benefit from afterload reduction. Third, the gravid uterus can compress the inferior vena cava (IVC) when the patient is in the supine position and thereby dramatically decrease venous return to the heart. This can precipitate hypotension especially in the presence of a volume dependent lesion like aortic stenosis or very poor left ventricular function.

### **Periods of Particular Cardiac Risk in Pregnancy**

Because of the increased cardiac demands intrinsic to the gravid state, cardiac disease can be unmasked or worsen in pregnancy. A particular period of high risk for cardiac decompensation is at 24 to 28 weeks gestation (the end of the second trimester) when blood volume and cardiac output peak. Twenty five percent of mitral stenosis in women in this country presents for the first time in pregnancy and presentation often occurs around this point in gestation.

A second period of high risk for cardiac decompensation during pregnancy is the work of labor. Every time the uterus contracts, about one to two units of blood is auto-transfused from

the utero-placental circulation into the systemic circulation. When the contraction stops the “auto-transfusion” returns to the uteroplacental circulation. A compromised heart may not be able to compensate for such rapid volume shifts.

A third high risk period is the postpartum. Cardiac stress in this period is due to a series of fluid shifts that include the involution of the uterus (with loss of the low resistance circulatory unit of the placenta), the normal (or abnormal) postpartum blood loss, and the increase in preload that occurs because of release of the gravid uterus’ compression of the IVC. Particularly in patients with Eisenmenger’s syndrome and severe pulmonary hypertension, the postpartum period may be associated with a substantial morbidity and mortality.

### **Specific Cardiac Complaints and Lesions**

#### **Palpitations and Dysrhythmias**

Palpitations are commonly experienced by pregnant women and may be due to the shift of the heart closer to the anterior chest wall, the faster resting heart rate, and increased contractility seen during pregnancy. Ectopy (especially premature atrial and ventricular contractions) may be found in symptomatic patients and is thought to be caused by increased atrial and ventricular stretch from the increased blood volume. Also, increased body awareness among many pregnant women coupled with increased exposure to health care providers may contribute to increased reporting of palpitations during pregnancy. Complaints of palpitations that can be characterized as fast, regular heart rates that run for greater than several minutes and are associated with lightheadedness, presyncope or syncope are most likely to be due a significant tachyarrhythmia and always warrant investigation. Investigation lesser complaints such as infrequent, brief “extra beats” rarely yields significant underlying medical conditions. However, an increased index of suspicion of tachyarrhythmias is warranted during pregnancy because of the potential for pregnancy to unmask tachyarrhythmias or exacerbate an underlying supraventricular tachycardia (SVT).

In the absence of cardiac disease, new onset of SVT during pregnancy is rare. Acute supraventricular tachycardia can be safely treated with adenosine in pregnancy. Use of digoxin, beta-adrenergic blocking agents, and verapamil is also acceptable. While case reports of fetal cardiovascular collapse following the use of verapamil exist, it occurred in association with maternal hypotension. If the patient is hemodynamically unstable, cardioversion can be safely carried out in pregnancy.

### **Assessing the Risk of Pregnancy for Rheumatic and Congenital Cardiac Disease**

The New York Heart Association (NYHA) Functional Classification for congestive heart failure is very useful for assessing the risk of pregnancy associated with structural cardiac disease. Class I and Class II patients generally tolerate pregnancy very well. Class III patients are at moderate risk with a pregnancy and often need hemodynamic monitoring during labor and delivery. Class IV patients do very poorly with pregnancy and almost certainly will need hemodynamic monitoring and a multi-disciplinary team should be assembled for the care of such patients throughout their pregnancy. Cardiologists, high-risk obstetricians and anesthesiologists should all be a part of such a team. The patient should also be made aware of the potential for mortality and morbidity prior to becoming pregnant.

In addition to the NYHA classification, some general comments about particular lesions can be made. Stenotic valves tend to have increased symptoms in pregnancy because of the difficulty in moving the increased blood volume across the stenotic valve. Also, stenotic valves may be volume dependent and therefore lying on the back (which leads to compression of the IVC by the uterus) should be avoided in these women. Patients with incompetent or regurgitant valves tend to have improvement in their symptoms during pregnancy. The decrease in systemic vascular resistance associated with pregnancy favors forward flow across these valves, much in the same way that ACE inhibitors improve hemodynamic function in these patients.

Pregnancy for patients with severe (greater than 80 mmHg systolic) pulmonary hypertension and/or Eisenmenger's syndrome (a "reversed" or right to left intracardiac shunt)

carries an extremely high risk of maternal mortality. Deaths in these patients often occur during the postpartum fluid shift that often occur in the 72 hours after a delivery.

### **SBE Prophylaxis**

The American Heart Association does not officially recommend subacute bacterial endocarditis (SBE) prophylaxis for routine vaginal deliveries or routine cesarean sections. However, it is reasonable to offer prophylaxis to patients with prosthetic valves in the form of ampicillin and gentamycin given intravenously.

### **Ischemic Heart Disease**

Ischemic heart disease, although uncommon in pregnancy, can manifest itself during pregnancy especially in those women who have had diabetes for over 20 years. However, the majority of acute ischemic heart disease in pregnancy is not related to atherosclerosis. About 50% of ischemic heart disease in pregnancy is related to idiopathic dissections of the coronary arteries. Other reported causes of ischemic heart disease in pregnancy are cocaine, antiphospholipid antibody associated arterial thrombosis and arterial vasospasm.

There are reported cases of coronary angiograms, angioplasty, bypass surgery, and intra-aortic balloon pumps all being used successfully during pregnancy. Our favored test for ischemic heart disease in pregnancy is a stress echocardiogram because of its high specificity in young women and the avoidance of the need for any radiation exposure. However, exercise stress testing, stress thallium and dobutamine echocardiography can also be done safely in pregnancy. Clinicians should be aware that CK-MB has been shown to rise above normal levels after routine cesarean section. The explanation is that CK-MB in this setting is being released from the uterus or placenta.

### **Peripartum Cardiomyopathy**

Peripartum cardiomyopathy can occur at any time in the third trimester and up to 6 months following delivery. It presents with congestive heart failure. The etiology is poorly

understood. Treatment must include anticoagulation because of the very high risk of cardiovascular thromboembolic disease. About one third of patients with this diagnosis will have a complete recovery, one third will have chronic cardiac insufficiency and one third will have progressive cardiac failure ending in either cardiac transplant or death. A risk of recurrence of peripartum cardiomyopathy in subsequent pregnancies is thought to exist, but the exact risk is difficult to quantify.

### **Cardiac Resuscitation in Pregnancy**

Cardio-Pulmonary Resuscitation (CPR) can be performed successfully on a pregnant woman. To improve efficacy of chest compression someone should pull the uterus to the patient's left side because this will improve venous return by decreasing compression of the IVC by the gravid uterus. The usual Advanced Cardiac Life support (ACLS) protocols can be employed for the pregnant woman. DC cardioversion can be done safely in pregnancy, but all fetal monitoring devices should be removed because there is a theoretical risk of the scalp electrode arc-ing and electrocuting the baby. If after five minutes of CPR no response has occurred, an emergency C-section may actually help improve the maternal outcome. It is believed that the mother will do better if the baby is delivered because delivery relieves the pressure of the gravid uterus on the IVC, removes the low resistance placental vascular bed, and thereby further improves the effectiveness of chest compressions.

## CARDIAC DISEASE IN PREGNANCY

### *Case Discussion*

#### Case #1

A 22 year old woman that you have followed in your practice for the past four years comes into your office with her husband for an appointment. She is clearly very distraught.

Her only medical problem has been mild mitral stenosis secondary to rheumatic fever that she had as a child in the Dominican Republic. She has always been asymptomatic, (New York Heart Association Classification Class I). The cardiologist has been very happy with her functional status. Echocardiogram has confirmed the clinical diagnosis of mild mitral stenosis that has remained unchanged over the past several years. Her EKG has always been normal and her cardiac exam is only remarkable for a loud S1 and a Grade 2/6 diastolic rumble heard at the apex.

Two weeks ago, she went to her annual visit to her cardiologist where she was told that a pregnancy could have disastrous consequences for her so she needed a foolproof method of contraception. The patient and her husband had been planning to have a large family and they were devastated to hear the cardiologist say that she should not get pregnant.

***What do you think of the cardiologist's statements?***

***What role does the primary care physician have in preventing this sort of crisis?***

***What are particular parameters to look at in determining the individual patient's prognosis for a pregnancy?***

***What are the complications of mitral stenosis in pregnancy?***

***What is the basis for these complications?***

***What are the highest risk periods for complications of mitral stenosis in pregnancy?  
Is there an increased risk of a cardiac anomaly in a baby whose mother has mitral valve disease?***

***What other medical professionals are available to assist you in counseling of this patient?***

The patient is greatly relieved by the information you have given her. She has a consultation with the Maternal-Fetal Medicine specialist who confirms the information you have given her. She and her husband decide to proceed with having a family now, while her mitral valve disease is asymptomatic. She comes to see you four months later, delighted to tell you that she has missed her period, and a home pregnancy test is positive. After confirming the pregnancy, you send her to an obstetrician you trust and whom you have familiarized with the case. You decide in collaboration with the obstetrician that you will see this patient each month during her pregnancy and to both you and your patient's delight, she has no problems for the first 20 weeks.

You continue to follow her along and she continues to do very well until you see her at 24 weeks. She tells you that she has a "cold" that she thinks is turning into bronchitis. You ask her her symptoms and she states that she feels short of breath, has a nonproductive cough, and just doesn't feel herself. She denies fevers, chills, myalgias, sputum, sore throat, sore ears, but she does report some mild sinus fullness. You do a complete exam on her and you can find nothing except for her previous findings. A diastolic rumble is still audible and perhaps now Grade 3/6.

***Why would her murmur be louder?***

***What are the possible diagnoses to explain her cough and shortness of breath?***

You ask her about paroxysmal nocturnal dyspnea or orthopnea and she reports that she is having orthopnea and the last two nights has taken to sleeping in the Lazy Boy® rocker in her living room.

You send her for a chest x-ray which you review in your office, and it is normal. You do notice that when she gets back to the office, she appears to be more dyspneic. She now states that she has to stop midway before she can go up one flight of stairs. She thought this was normal for pregnancy.

***What are important causes of dyspnea at this point of the pregnancy?***

***What are the risks of the radiation from a chest x-ray in pregnancy?***

You decide to send her to the emergency room where she will be seen by both her obstetrician and her cardiologist. As you finish up your office, you get a phone call from the cardiologist who states that he hears crackles at both lower bases in the patient's lungs and feels there is jugular venous distention. You come to the emergency room and confirm his findings. The obstetrician has done fetal monitoring and feels the fetus is in good shape. A pulse oximeter shows an O<sub>2</sub> saturation of 95% on room air and a blood gas shows a PaCO<sub>2</sub> of 30, a PO<sub>2</sub> of 90 with a normal pH and bicarb.

*What do you think of this blood gas?*

After discussion with the patient, a chest x-ray is repeated which now shows cephalization of vessels, small pleural effusion, and some Kerley B lines. The heart appears enlarged.

*Why are the x-ray and clinical findings different now, despite the fact that the patient does not appear to have deteriorated?*

*What are the possible causes of congestive heart failure at this point in pregnancy?*

The patient is admitted to the hospital. She has a repeat echocardiogram that confirms that there has been on deterioration of valve status. She is given a small dose of Lasix and after a good diuresis, her symptoms revolve.

*Is there a role for ace inhibitors here?*

*What vasodilator can be used safely in pregnancy?*

*Was it alright to have given this patient Lasix?*

*Is there a role for beta blockers?*

She is sent home on three times a week small dose of Lasix 20 mg po, which keeps her out of failure. However, at 32 weeks, she comes into the hospital with sustained palpitations and a presyncopal feeling. In the emergency room, her pulse is noticed to be 150 bpm. It is felt to be irregularly regularly. Her blood pressure is 90/60, although it usually runs around 100/70 at this gestation. An EKG is obtained and you see atrial fibrillation with a rapid ventricular response of 150.

*Why has this patient developed atrial fibrillation at this point?*

*Could this occur in the absence of mitral stenosis?*

*What medications can be used to control this patient's rate acutely?*

*Can DC cardioversion be performed during pregnancy?*

*What would you do if this patient had a cardiac arrest?*

*How does pregnancy effect this patient's risk of cerebral vascular accident with atrial fibrillation?*

It is clear from her history that the onset of her symptoms was very sudden and after only 20 minutes of the symptoms, she called the ambulance to be brought in. The patient is loaded with IV Digoxin to try and slow down her rate. After receiving 0.5 mg of IV Digoxin, her rate is less than 100 and she spontaneously converts back to normal sinus rhythm.

***If you are going to keep her on Digoxin, what sort of special dosing might be required in pregnancy?***

***How would you anticoagulate this patient if you felt it were indicated?***

The patient is maintained on Digoxin without anticoagulation for the rest of her pregnancy and no further episodes of sustained palpitations/atrial fib are picked up. At 39 weeks gestation, she goes into spontaneous labor. After initial progression of labor, her cervical dilation starts to slow down and the obstetricians decide that they would like to augment her labor with Pitocin. However they call you to double check that they can give Pitocin to a woman with cardiac disease.

***What do you tell them?***

***What are the important aspects of analgesia/anesthesia for the cardiac patient in labor?***

***How is cardiac work affected by active labor?***

***Should she have SBE prophylaxis?***

After three hours of Pitocin the patient enters the second stage of labor and the baby begins to descend through the birth canal.

**What should this woman do about pushing?**

**Can a baby be delivered without the woman pushing?**

**Would a cesarean section be safer because it is more “controlled”?**

The woman spontaneously delivers a 7 lb. 4 oz. baby girl. Her estimated blood loss is 300 cc and she, her husband, and you, are ecstatic.

***Is this woman “out of the woods” yet?***

***What is the risk in the postpartum period?***

***In general, how long does the risk period last?***

***Why is estimated blood loss particularly important in cardiac patients?***

The woman flies through the postpartum period without any complications and is discharged home at 72 hours post delivery. Follow up is arranged for a week from now with you in the office.

***What will you do with the Lasix and Digoxin?***

***What will you tell this woman about her risk in subsequent pregnancies?***

## CARDIAC DISEASE IN PREGNANCY

### *Case Discussion*

#### **Case #2**

R.J. is a 32-year-old G<sub>2</sub>P<sub>1</sub> at 34 weeks gestation who has had an uneventful pregnancy thus far. Her first pregnancy was a term delivery by NSVD of a 7 lb 2 oz baby girl two years ago, who is in good health. She is referred to you by her obstetrician for the complaint of palpitations. She describes herself as generally well, although she is wrapping up the end of her school year where she is a special education teacher and is having to meet with families and make plans for students for next year. This involves extensive evaluation and she does feel that she is under her most stress at this time of the year. She describes herself as having brief fluttering in her chest both during the day and at night. The longest one has lasted for about one minute. They are not associated by chest pain, just a funny feeling in her chest. She denies dizziness, orthopnea, PND, or ankle swelling. She states that she does not recall having this feeling during her prior pregnancy. She is currently on no medications with the exception of her prenatal vitamins. She is not taking over-the-counter decongestants and has not changed her caffeine intake this entire pregnancy which is one cup of caffeinated coffee in the morning.

On examination, she is afebrile, her pulse is 95 and regular, her respirations are 14, her BP is 100/60. Examination of the heart, lungs, and extremities are entirely normal with no evidence of edema and a soft 1:6 flow murmur that sounds normal to you for pregnancy. You order a few screening lab tests, reassure her and tell her that it is very unlikely that she has any cardiac disease.

***What lab tests should you order?***

***Are there any further historical points or diagnostic testing you would like at this time?***

The results of a CBC, electrolytes, thyroid function tests, calcium, and magnesium return and are entirely normal, so you decide to forgo any usual Zebra hunt. You do ask her to please call you with any increase or change in her symptoms or any associated dizziness, shortness of breath, chest pain, or any further complaints. She returns home, goes to work, and you get a call from her obstetrician's office two days later that she has been brought into the ER by rescue complaining of dizziness and shortness of breath. You arrive at the obstetric emergency room

where the patient is resting comfortably, sitting up, and to your surprise, is not on a cardiac monitor. You hook her up to a cardiac monitor, order a 12-lead EKG, ask someone to start an IV and note that on the monitor, she is having frequent PVCs including occasional runs of a 3 to 4 beats v-tach. She states that this is similar to the feeling she has been having all morning, although it has slowed down somewhat and she feels less dizzy. Her blood pressure is 90/50 supine and she does feel somewhat short of breath when you lay her flat to examine her. The only change in her exam from your previous exam is that her resting HR is now 110 to 120. Her lungs are still clear and there is no lower extremity edema. Her respiratory rate has increased to 24. You do a 12-lead EKG which shows a sign of tachycardia at a rate of 115 with occasional PVCs. A pulse ox is 95%.a You ask that she be on continuous pulse ox and stay on an EKG monitor where you have also requested that someone watch the monitor and ask that she get a chest x-ray and blood gases and repeat some blood work.

***What do you expect the results of the current laboratory testing and chest x-ray to be?***

***What possibilities are there to explain her current complaints?***

CBC including platelet count, urinalysis, BUN, creatinine, AST, electrolytes are all normal. A uric acid has not yet returned. Her ABGs show a pH of 7.45, PO<sub>2</sub>, of 92, a CO<sub>2</sub> of 26. Chest x-ray shows changes consistent with congestive heart failure. The cardiac size is at the upper limit of normal. You review your physical exam of the patient and do not hear any evidence of valvular heart disease.

***What is the differential diagnosis at this time?***

***What interventions should we do?***

***What further diagnostic procedures may you want?***

The echocardiogram shows diffuse hypokinesis of the heart with an ejection fraction of 40%.

***Now, what is the most likely diagnosis?***

***Now how would you suggest this patient be treated?***

***What special considerations are there for labor and delivery of this patient?***

***When is she “out of the woods?”***

***What is her prognosis for future pregnancies?***

# CARDIAC DISEASE IN PREGNANCY

## *Case Discussion*

### Case #3

A 32 year-old woman at 20 weeks gestation in her first pregnancy was sent to see you by her obstetrician because she is experiencing frequent palpitations. She describes the palpitations as an awareness of extra beats that can occur frequently and last 1 or 2 minutes. They are not associated with any presyncope, chest pain, or shortness of breath. She feels otherwise well. She has never had similar symptoms before in her life. Her only cardiac risk factor is a family history of coronary artery disease in her father at age 48. She denies ingestion of alcohol or caffeinated beverages during this pregnancy. She denies the use of drugs.

On examination her blood pressure is 90/50, respiratory rate is 12. Pulse is 102. Her temperature is not elevated. Her thyroid is normal in size. Her chest is clear. Auscultation of the heart reveals a grade 3/6 injection systolic murmur heard at the left upper sternal border. She has 1+ nontender symmetrical lower limb edema.

### Key Points to Review

1. The main risk of chronic hypertension to a pregnancy is its' association with preeclampsia which occurs in up to 20% of chronic hypertensives. There is no way to predict which chronic hypertensive will develop preeclampsia nor is there any way of preventing the development of preeclampsia. In particular, good blood pressure control during pregnancy does not decrease the risk of preeclampsia.
2. Chronic hypertensive women can either come off their blood pressure medication during pregnancy (as long as their blood pressure stays less than 160/1200) or remain on a medication for which there is good data to support its use in pregnancy.
3. First line antihypertensives acceptable for use in pregnancy are: Labetalol (Trandate® or Normodyne® 100-1200 mg po BID) or methyl dopa (Aldomet® 250-1500 mg po BID)

Second line agents include: Pindolol (Visken 5-30 mg. BID) Atenolol (Tenormin® 50-400 mg DAILY).

***The ACE inhibitors and angiotensin II antagonists are contraindicated in pregnancy even though they are not teratogens.***

4. The symptoms of preeclampsia are a migraine type headache, migraine type visual phenomena, and epigastric pain. Edema is no longer felt to be a reliable manifestation of preeclampsia.
5. Physical signs of preeclampsia are: 1) worsening hypertension, 2) retinal vasospasm, 3) epigastric tenderness, and 4) clonus.
6. “Preeclampsia labs” are a CBC, AST, uric acid, creatinine and uranalysis for protein.
7. Severe early preeclampsia needs investigation for underlying thrombotic tendencies or underlying renal disease.

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## CARDIAC DISEASE IN PREGNANCY

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*For this case series, those with a history of surgical repair had a similar live-birth rate as those without surgical repair. However maternal cyanosis and poor functional status were associated with fetal growth restriction and a lower live-birth rate, regardless of surgical history.*

