Cardiology

Medicine

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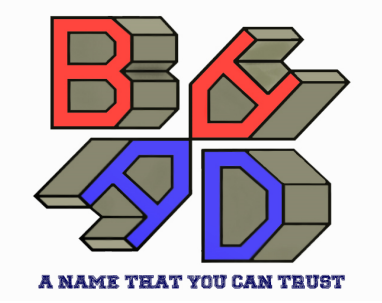


Price = 1.5 JDs

Ischemic heart disease

- Ischemic heart disease (IHD) is one of the major causes of death worldwide. Moreover, it is considered one of the major diseases of the heart.
- Classification of heart diseases:
    - IHD
    - Heart failure (HF)
    - Cardiomyopathies
    - Arrhythmias
    - Infective heart diseases
- The heart is a muscle that weighs 300-400 grams. The heart needs to function continuously; this is why it needs a good blood supply and a good amount of energy.
- The blood supply of the heart comes from the coronaries. There are three major coronary arteries; two on the left, one on the right:
    - Left main:
        - Arises from the left coronary sinus
        - Divides into:
            - Circumflex: supplies the lateral wall, and sometimes the inferior wall. Injury is reflected on the ECG with changes on lead I, aVL, lead V5-V6
            - Left anterior descending (LAD): LAD is the major supply of the left ventricle. Any injury in the LAD is reflected as ECG changes on the precordial leads (mainly V1-V4)
            - If the disease involves the ostium of the LAD or the left main, the changes will affect all the leads from V1-V6 (extensive antero-lateral changes)
    - Right coronary artery:
        - Arises from the right coronary sinuses
        - Supplies the right ventricle
        - changes in this area are reflected on lead II, III, and aVF.

- A collateral circulation normally exists between these arteries; however, it is nonfunctional. To make these collaterals functional, you need ischemia. The most potent stimulus for collateralization is transient ischemia. This is why people with a history of angina have open collateral circulation.
- The flow of the blood to the heart is about 70-80 mL/min/100gm. This flow can increase up to eight times with exercise.
- The heart muscles are characterized by high oxygen extraction (75%). This means that the oxygen extraction is fixed even with high amounts of exertion. If the myocardium

needs more oxygen during exercise, this need is met by increasing blood flow to the coronaries. To have this increase in blood flow, you need a healthy patent coronary artery. If the coronaries are diseased (stenosed), this need will not be met and the heart will suffer from shortage of oxygen supply. In other words, the muscle will suffer from ischemia.

- 80% of the blood supply to the coronaries comes during diastole. Other organs are supplied during systole. The heart's blood supply decreases during diastole because, during the systolic phase, the pressure is high which causes less blood to flow through the coronaries (increased jet flow through the left ventricle through the aorta).
- If the patient develops tachycardia, the diastolic phase is shortened; thus, decreasing the flow of the blood to the heart. Therefore, patients with IHD should avoid tachycardia.
- Ischemic heart disease develops when there is a mismatch between supply and demand of oxygen.
- Factors affecting oxygen demand:
    - Heart rate (the most important)
    - Myocardial contractility
        - Beta blockers work through decreasing heart rate and contractility (negative inotropic and chronotropic effects).
    - Left ventricular wall tension (more tension more oxygen requirement)
    - Muscle mass: (patients with the following diseases have a higher oxygen demand. Therefore, they are liable to demand-supply mismatch)
        - LV hypertrophy
        - HOCM
        - HTN
        - Aortic Stenosis
- Factors affection blood supply:
    - Patency of the coronary arteries: a clinically significant stenosis is between 50-70%
    - Hemoglobin levels" Anemic patients are at higher risk of angina. Anemia can cause angina without the presence of coronary artery disease
    - Fixed cardiac output states
    - Decreased oxygen saturation
        - Hypoxia
        - Shock
    - Coronary artery disease)
- One common cause of IHD is CAD; and the most common cause of CAD (95% of the cases) is atherosclerosis. The rest of the cases are caused by a variety of disorders including, but not limited to: vasculites, autoimmune diseases, amyloidosis, or coronary artery anomalies (coronary arteries arising from the pulmonary trunk instead of the aortic root).

- Risk factors for atherosclerosis:
    - Modifiable
        - HTN
        - Smoking
        - Hyperlipedemia (high LDL, low HDL, high triglycerides)
        - Diet
        - Sedentary lifestyle
        - DM
        - hyperurecemia
    - Non-modifiable
        - Family history:
            - Male side less than 45 years of age
            - Female side less than 55 years of age
            - previous stroke
            - Male gender
            - Age
            - Post-menopause female
- Atherosclerosis is a disease that affects all arteries. If a territory is affected, there is a higher chance that other territories will be affected, as well.
- 95% of our patients have at least one risk factor.
- Risk factors coalesce in an exponential fashion
    - HTN: 3-fold increase
    - Hyperlipedemia: 4-fold increase
    - Both of the aforementioned conditions: 9-fold increase
- Endothelial cell dysfunction represents the first step towards developing atherosclerosis. This happens in cases of HTN, DM, and hyperlipedemia. The endothelium has many functions in our body (anticoagulation, and vasodilator release). LDL goes through the endothelium to the subentimal layer where it is engulfed by macrophages forming foamy cells. Accumulation of foamy cells leads to the formation of plaques.
- Atherosclerosis usually starts early in childhood, and it is affected by both environmental and genetic factors.
- Types of atheromatous plaques:
    - Stable
        - Strong, not liable for rupture
        - Strong fibrous capsule
        - High number of smooth muscle cells
        - Few inflammatory cells
        - Small lipid core
    - Unstable

        - Liable for rupture, erosion and fissuring. This leads to the formation of a thrombus which leads to ACS
        - Thin fibrous capsule
        - Abundant inflammatory cells
        - Few smooth muscle cells
        - Large lipid core.
- The mainstay of treatment for unstable plaques is to convert them to stable plaques. For this, we use statins (all patients with IHD are on statins).The goal of statin therapy is to keep LDL as low as 70. Moreover, these patients should be on antiplatelets such as aspirin.
- Stages of plaque development:
    - At first the plaque is confined to the intimal and subentimal layers. After a while, the wall cannot withhold the pressure, and the plaques start to move towards the lumen of the artery. Symptoms usually develop when the lumen's diameter is decreased to 50%. Angina develops when stenosis reaches 70%.
    - Stages of atheroma:
        - Foamy cell
        - Fatty cell
        - Extracellular fatty streaks
        - Lipid core
        - Plaque formation (stable and unstable)
- Usually, it takes patients many years to develop the symptoms. When the patient reaches the symptomatic stages, it is too late. This is why managing the disease should be started as early as possible.
- Manifestations of atherosclerosis depend on the site of the lesion:
    - Coronaries (ACS)
    - Cerebral circulation (TIA's and CVA's)
    - Peripheral arteries (PVD)
    - Renal arteries (renal artery stenosis)
    - Mesenteric arteries (mesenteric ischemia)
- The symptoms can range from asymptomatic to sudden death.
- Other manifestations of IHD:
    - HF
    - Angina
        - Stable (More on this later in the lecture)
        - Unstable (more on this in the next lecture)
        - Variant:
            - Coronary artery spasm at rest
            - Troponin negative
            - Females > males

        - Patients usually have signs of other vasospastic diseases (Raynaud's, migraine)
        - During the attack patients might suffer from arrhythmias which might lead to sudden death.
        - Treatment is by CCB's and nitrates. These patients should avoid beta blockers as these leave alpha receptors unopposed causing more vasospasm.
        - Good prognosis
    - Arrhythmias
    - Acute coronary syndrome
- Unstable angina and MI happen when the plaque is unstable. The difference between these two is subtotal or total occlusion of the artery respectively.
- In chronic IHD, the plaque is stable.

- **<u>Stable angina:</u>**

    - The most common presentation of IHD, and it results from an imbalance between supply and demand.
    - It is characterized by retrosternal chest pain or left precordial pain (less common sites are: left shoulder, left arm, left forearm, neck, throat, epigastrium)
    - It is usually precipitated by exertion or anything that increases the myocardial oxygen demand or heart rate such as walking.
    - Relieved by rest.
    - Diagnosis of stable angina is made through history.
    - If the angina is more than 8 weeks old, it is stable (less than that, it is termed unstable)
    - It is a heavy, squeezing, crushing, or burning pain.
    - The pain usually lasts 5-10 minutes, and is relieved by nitrates.
    - Sometimes, patients might complain of nausea and sweating; however these symptoms are less common (more common with MI)
    - Grades of angina:
        - Grade 1: No symptoms (no limitation on daily activities)
        - Grade 2: Symptoms with ordinary activity (slight limitation)
        - Grade 3: symptoms with minimal exertion (sever limitation)
        - Grade 4: pain at rest (severe limitation)
    - Diagnosis:
        - On examination, there is no specific sign for angina; however, you should look for signs of risk factors despite normal findings. You need to look for

HTN and signs of atherosclerosis in other arteries (pulses, and bruits such as mesenteric bruit).
        - You should ask about signs of atherosclerosis in lower limbs, such as intermittent claudication.
        - Symptoms of TIA;s and strokes.
        - Signs of peripheral artery disease
    - Some patients might have grade 3 angina; however, when catheterized those patients might have normal catheterization results. This can be explained by valvular heart disease, HOCM, or any other non-atherosclerotic cause of IHD.
    - Differential diagnosis:
        - Respiratory diseases:
            - Pleurisy
            - pulmonary HTN
            - pneumonia
        - GERD
        - Cardiac syndrome X:
            - Typical angina with a positive exercise test
            - Normal coronary arteries.
            - The defect is usually at the level of the arterioles which have a decreased diffusion capacity.
            - Affects females more than males
            - Happens in young patients.
    - Usually patients come when they are pain free. However, if they come when in pain, you might find S4.
    - ECG is normal.
        - A normal ECG does not rule out any form of IHD.
        - You might find slight ST depression
        - Stress ECG (increase the heart rate and increase demand inducing certain ischemic changes) causes changes in the ECG e.g. (ST segment depression or chest pain)
    - Stress echocardiogram shows less contractility in the affected region of the heart
    - CT angiograms to look for area of stenosis
        - Angiograms are performed to locate the region of the lesion and to evaluate the extent of the disease.
    - Lipid profile
    - Glucose check
    - CBC (to rule out anemia)
    - Stress test modalities:
        - Exercise testing
        - Chemical exercise e.g. dobutamine

- Coronary artery ultrasound
- Treatment and management:
    - Goal: to improve prognosis and decrease mortality
    - Statins
    - Aspirin
    - ACE-I
    - Revascularization (if needed)
    - Correction of risk factors.
    - Treat angina symptoms:
        - Increase supply
        - Decrease demand
    - GTN, when needed (symptomatic) sublingual.
        - If symptoms occur more than three times every week, patients should be started on a sustained (long acting) form of GTN (Oral sustained, or patch)
        - If symptoms occur, the patient can take up to 3 GTN tablets 5 minutes apart.
        - If symptoms do not subside after the third tablet, the patient must present to the hospital because it is most likely to be an infarction.
        - Beta blockers
        - CCB's
- Prognosis depends on the amount of stenosis, site of stenosis, and number of vessels affected.

Acute coronary syndrome

- Acute coronary syndrome is divided into:
    - Myocardial infarction
        - ST segment elevation (Q wave) (STEMI)
        - Non-ST segment elevation (non-Q wave) (NSTEMI)
    - Unstable angina
- In STEMI, the artery is totally occluded, while in NSTEMI and unstable angina the artery is subtotally occluded.
- The difference between unstable angina and NSTEMI is the presence of cardiac markers in NSTEMI
- The aforementioned conditions are characterized by the same pathophysiological process, which is plaque rupture (non-intact plaque)
- An important element in the pathogenesis of ACS is platelet function. When we have a plaque rupture, the subendothelial layer is exposed to the circulation. The platelets will interact with the exposed subendothelial surface via the von Willebrand factor (a ligand found on the subendothelial layer). Then, platelet adhesion will happen. More platelets will adhere via platelet adhesion mediators including thromboxane A2, thrombin, and ADP. Platelets coalesce together to form a platelet plug (fibrinogen). After that, a clot will be formed (coagulation).
- Pathophysiology in steps:
    - Plaque rupture
    - Thrombosis, which starts by primary hemostasis
    - Platelet adhesion, activation, and aggregation
    - Formation of platelet plug (fibrinogen rich, white thrombus)
    - Activation of secondary hemostasis, which will lead to the formation of the red thrombus (fibrin rich thrombus)
- Here, we have a dynamic interaction between the platelets and the subendothelial layers which leads to this cascade of events which will occlude the arteries and this is manifested as symptoms (NSTEMI, STEMI, unstable angina)
- Acute MI is the most common clinical cause of death.
- **Symptoms:**
    - Chest pain at rest, usually in the early hours of the morning. This is due to high thrombotic activity along with low thrombolytic activity.
    - The pain is usually persistent and lasts for more than 30 minutes
    - Retrosternal pain, left precordial pain
        - Can be referred to the same areas as angina.
        - Epigastric pain is common especially in inferior wall MI.
        - Severe jaw pain
        - Severe intrascapular pain
        - Severe shoulder pain

  - Associated with nausea vomiting and sweating more than angina
  - 15% of patients can present with painless MI. Painless MI's are more common in diabetic and elderly patients
  - Some patients present with unexplained hypotension as the only sign for MI
- **Examination:**
  - There is no specific sign on examination.
  - The patient looks anxious, in pain, or irritated
  - Vital signs depend on the infarction time:
    - Blood pressure might be elevated due to pain (increased catecholamines); however, it can be low
  - Shock
  - Pulse: tachycardia (usually)
    - Sometimes, patients present with bradychardia due to complete heart block (more common with inferior MI)
  - Temperature: usually normal
    - Some patients might suffer from a low grade fever 24 hours after the infarction
  - **Auscultation:**
    - S4 due to ventricular stiffness.
    - S3 is a bad sign indicating impending systolic heart failure which carries a bad prognosis
    - Look for murmurs. Murmurs usually presents later on, and usually come as a complication after the MI due to muscle rupture or VSD
    - Look for friction rub to exclude pericarditis. Pericarditis might produce similar symptomatology. Moreover, pericarditis is a common complication of an MI
- ECG is used to differentiate between STEMI and NSTEMI
  - Hyperacute T wave is one of the earliest signs on ECG. These last for a few minutes.
  - ST segment elevation
  - Formation of Q waves
    - Q waves can be seen as an early or a late manifestation.
  - NSTEMI is usually accompanied with T wave inversion and ST segment depression
- A normal ECG does not rule out an MI. 10-15% patients with MI present with normal ECG
- **Cardiac markers:**
  - Troponin
    - Troponin I and T
    - Troponin I and T are usually elevated 4-6 hours after an MI.

        - Peak in 24 hours
        - Back to normal in 10-14 days
    - CK-MB (two main isoforms, 1 and 2)
        - CK-MB2 : CK-MB1 ratio more than 1.5 is highly suggestive of MI
        - Rises after 4-6 hours
        - Peak in 24 hours
        - Back to normal in 72 hours.
    - Although highly sensitive, troponin can be elevated in other causes:
        - Tachycardia
        - Pulmonary embolism
        - Renal failure
        - Post PCI
        - Open heart surgery
        - Heart failure
        - Strenuous exercise
        - Amyloidosis
        - Critically ill patients
        - Sepsis
    - We do not need to perform tests to detect for both markers, unless we want to determine the age of the infarct.
- ECG changes will give you an idea of the site of the infarction:
    - ST segment elevation is considered significant only if present in 2 or more adjacent leads (this applies to most ECG abnormalities)
    - ST elevation is significant when the elevation is more than 1 mm in limb leads and 2 mm in precordial leads
    - ST elevation in anterior and lateral leads: the artery most probably involved is LAD or left main.
    - ST elevation in Lead II, III, aVF: inferior infarct (right coronary artery)
    - ST elevation in Lead I, aVL, V5, and V6: lateral wall infarct (left circumflex)
    - ST elevation in V2-V4: anteroseptal infarct
    - Prominent R wave and ST segment depression in leads V1-V2: posterior wall infarct
- **<u>Other investigations:</u>**
    - CBC: mild lymphocytosis
    - Elevated glucose in non-diabetic patients (bad prognostic factor)
    - High ESR
    - Lipid profile (4-6 weeks after MI)
    - Echocardiogram to look for changes (hypokinesia or dyskinesia)

- **Management:**
    - Revascularization: (definitive measure)
        - The sooner the better.
        - This is to decrease the amount of myocardial necrosis and preserve the function of the muscle.
    - Management should be ideally started at home (calling an ambulance and giving aspirin in case of suspected MI)
    - The patient should be given aspirin as soon as they arrive to the hospital.
    - GTN will not benefit the patient.
    - The patient should be transferred to the nearest hospital with proper equipment
    - The most common cause of death in MI patient is arrest due to ventricular fibrillation
    - In the emergency room,
        - Quick history
        - Quick physical exam
        - ECG immediately
        - Take vitals
        - Start with MONAH (Morphine, Oxygen, Nitrogen, Aspirin, and Heparin (unless one of them is contraindicated)
            - Morphine is given 25 mg IV preceded by antiemetics.
            - If the patient has bradychardia, avoid morphine. Give pethidine instead
        - Give other antiplatelet agents such as clapidogrel
    - Prepare patient for PCI as soon as possible
        - If you are in a periphery hospital where PCI is not readily available, administer thrombolytics and schedule for PCI as soon as possible
        - The patient should be taken from the ER to the catheterization lab within 10 minutes of presentation
        - The aim of treatment is to restore normal flow. This can be achieved with a success rate of 95% in PCI. With thrombolytics this percentage drops to 55-60%. With thrombolytics, you probably can open the artery. But, the flow remains abnormal. If the artery was opened, but the flow was still sluggish, the prognosis is as bad as an occluded artery.
        - **Contraindications for thrombolytics:**
            - Cardiogenic shock post MI
            - Post CABG MI
        - **Indications of thrombolytics:**
            - ST segment elevation
            - New onset bundle branch block

- Thrombolytics are divided into several generations. The best are 3rd generation thrombolytics such as tenecteplase. Tenecteplase can be given as an IV bolus. Older generations need to be given as infusions.
- With thrombolytics, there is a high risk for an anaphylactic reaction
- Do not give thrombolytics in patients with active bleeding, CVA, CPR, ST depression
- One of the worst complications of thrombolytics is the creation of emboli:
- MI's can be complicated with formation of thromboemboli which can be lodged anywhere in the body. Due to stagnation of blood in the left ventricle, a clot might form. There is a high risk that this clot can cause a CVA.
- **Management:**
    - The patient stays in the CCU for 24-48 hours post MI for observation.
    - On discharge, the patient is kept on lifelong aspirin. Moreover, the patient should be kept on clapidogrel for at least a year. Statins lifelong. ARB's, beta blockers if there is no contraindication. The patient should revisit in 4 weeks or whenever complications arise.
- **Complications of MI:**
    - Electrical:
        - Arrhythmias (benign or malignant):
            - sinus tachycardia (benign)
            - Persistent sinus tachycardia (malignant). It can lead to heart failure, shock or hypotension
            - PVC (benign)
            - Ventricular tachycardia (malignant)
            - Atrial fibrillation (malignant)
        - Atrial and AV blocks…
    - Mechanical:
        - Heart failure:
            - It means that the patient lost a significant amount of muscle (more than 20%)
            - If the patients lose 40% of the muscle, they would develop a cardiogenic shock
        - Myocardial rupture
        - Papillary muscle rupture
        - Early pericarditis (1-3 days after MI)
        - Late pericarditis (weeks to months later, usually called Dressler's syndrome)
        - Aneurysm
- **Differential diagnosis:**
    - Aortic dissection:
        - Severe tearing pain

        - Mimics pain of MI, especially if it involves the ascending aorta (type A dissection).
        - The difference between MI pain and dissection pain is that the peak of the dissection pain is right from the start. While in MI, the pain usually progresses
        - Dissection type B: usually the pain is in the back (interscapular)
    - Massive pulmonary embolism
    - Acute pericarditis
    - Pneumonia
- High mortality rate in MI up to 20%
- The commonest cause of death outside the hospital is ventricular fibrillation
- The commonest cause of death inside the hospital is cardiogenic shock
- **Prognosis:**
    - TIMI score to assess risk of mortality in MI:
        - DM history, hypertension or history of chest pain. (1 point)
        - Systolic blood pressure < 100 mmHg (3 points)
        - Heart rate greater than 100 BPM (2 points)
        - Killip class II-IV (2 points)
        - Body weight less than 150 lbs (I point)
        - ≥75 years old (3 point)
        - 65-74 years old (2 points)
        - Less than 65 years old (0)
    - TIMI risk score : 0 points (0.8%); 1 point (1.6%); 2 points (2.2%); 3 points (4.4%); 4 points (7.3%); 5 points (12%); 6 points (16 %); 7 points (23%); 8 points (27%); 9-14 points (36.0%).

- **Unstable angina:**
    - Any new onset angina is unstable angina.
    - Angina at rest or minimal exertion is unstable angina
    - Crescendo angina: (worsening angina) angina increasing in frequency, duration, severity, or a change in the pattern of chest pain.
    - Any angina after an MI (in a period of two weeks is considered unstable angina).
    - It is usually caused by a plaque rupture (causing subtotal occlusion of the artery.)
    - Normal ECG
    - Normal cardiac enzymes
    - **Classification:**
        - Primary (no secondary causes, absence of extracardiac causes)
        - Secondary (secondary causes presence , presence of extracardiac conditions that increase the risk of angina)

- Patients with primary angina are considered low risk patients. While patients with secondary angina, are considered high risk patients.
- Low risk patients are treated as patients with chronic stable angina
- High risk patients are treated like NSTEMI patients
- Signs of increased risk:
    - If the pain lasts for more than 20 minutes
    - Pain has an accelerated pattern
    - Pain changes in character (from on exertion to at rest)
    - The patient has signs of heart failure during the attack (lower limb edema for example)
    - S3
    - Murmurs
    - Hypotension
    - Transient ST segment changes (dynamic changes; ST depression is more dangerous than ST elevation during an episode of unstable angina)
    - Diabetes
- TIMI classification (more objective classification of high and low risk patients)
    - Age 65 or older (1 point)
    - At least three risk factors for CAD (1 point)
    - Prior coronary stenosis of 50% or more (1 point)
    - ST-segment changes on ECG (more than 0.5 mm) (1 point)
    - Use of aspirin prior 7 days (1 point)
    - At least 2 anginal events in prior 24 hours (1 point)
- If the patient scores more than 4, the patient is at high risk.
- Management of high risk patients:
    - CCU admission, treat as MI
    - **NO** thrombolytics
    - Aspirin
    - Clapidogrel
    - Anticoagulants
    - Statins
    - Consider a treatment strategy:
        - Invasive (better, usually done within 24 hours of presentation)
        - Conservative

Hypertension

- Blood pressure is the product of cardiac output (CO) and systemic vascular resistance (SVR). Cardiac output is the product of stroke volume (SV) and heart rate (HR). each of these factors is further controlled by other factors, mainly the sympathetic and parasympathetic nervous systems.
    - Systemic vascular resistance → vasoconstriction and vasodilation under control of nervous system
    - Stroke volume is regulated by venous tone which controls venous return.
    - Stroke volume is further controlled by certain hormones such as the RAAS system (rennin angiotensin system), aldosterone, and antidiuretic hormone (ADH).
- Hypertension: is one of the most common chronic diseases that we have to deal with on daily basis. It is defined as systolic blood pressure of 140 or above, and/or diastolic blood pressure of 90 or above.
- Females usually have lower blood pressure than males. It is quite common to find a female with blood pressure lower than 120/80.
- Classification of HTN:
    - Pre-hypertension:
        - Systolic120-139
        - Diastolic 80-89
        - These patients require lifestyle modifications
    - Stage 1 HTN
        - Systolic: 140-159
        - Diastolic: 90-99
    - Stage 2
        - Systolic: above160
        - Diastolic: above100
- Isolated systolic HTN is common in people after the age of 50. This happens due to arterial calcification
- Starting at 115/75, each increment of 20/10 increases your cardiovascular risk by double
- Classification according to etiology:
    - Primary:
        - is of unknown etiology
        - Most of the cases are primary (90-95% of all HTN cases)
    - Secondary HTN
        - The etiology is known (5-10% of all HTN cases)
        - If we treat the cause, we treat the HTN

- There are many theories for causes of primary HTN. The most accepted is sympathetic overactivity and increased peripheral vascular resistance.
- Common associations with HTN:
    - Male above 55, Female above 65
    - Alcoholic
    - Diabetic
    - Smoker
    - Excess dietary sodium intake
    - Family history
    - Obesity
    - Ethnicity (African Americans)
    - Sedentary lifestyle
    - Socioeconomic status
    - Stress
- HTN clinical manifestations:
    - Usually asymptomatic; named the silent killer.
    - Unless you have end organ damage, symptoms are usually absent
    - Some patients do feel a headache, and those are lucky
- HTN is a risk factor for atherosclerosis because it causes endothelial cell damage. This might lead to plaque formation; and this increases the risk for developing an MI or PAD.
- The end organs mostly affected are:
    - Brain
    - Heart (CAD, LV hypertrophy, HF)
    - Kidneys
    - Eyes (retinopathy)
- Diagnosis:
    - **White coat syndrome**: patients have falsely elevated reading at the clinic due to stress. Home or ambulatory readings are preferred for these patients.
    - Blood pressure should be taken from both arms while the patient is rested. This procedure is repeated twice to confirm the diagnosis.
- Treatment goals:
    - Patients with DM, CKD, or below the age of 60 → keep BP below 140/90
    - If the patient is above 60, the target is 150/90 unless DM or CKD are present.
    - Proper treatment decreases risk of the following
        - HF up to 50%
        - MI up to 25%
        - Stroke up to 40%
    - Lifestyle changes:
        - Weight reduction
        - Change in diet

        - Limitation of alcohol intake
        - Decrease stress
        - Increase exercise
        - Tobacco cessation
        - Sodium restriction
        - Calorie restriction in overweight
        - Drug treatment: they decrease systemic vascular resistance along with blood volume
- JNC8 recommendations for blood pressure control:
    - In the general population, pharmacological treatment of HTN starts if the systolic BP is above 150 or diastolic BP is above 90 → if the patient is over 60 years of age.
    - In the general population, pharmacological treatment of HTN starts if the systolic BP is above 140 or diastolic BP is above 90 → if the patient is less than 60 years of age.
    - If the patient suffers from DM or CKD, BP should be below 140/90
    - In the general non-black population, including those with diabetes, initial therapy should include one of 4 classes:
        - ACE-I
        - ARB's
        - CCB's
        - Thiazide
    - In the general black population, including those with diabetes, initial treatment should be started with:
        - Thiazide diuretics
        - CCB's
    - Patients who suffer from CKD should have an initial or add-on therapy with ACE-I/ARB's
    - The objective of blood pressure treatment is to attain ideal blood pressure no matter what medications you use
    - If the goal blood pressure is not reached within one month of initiation of treatment, increase the dose or add a second drug
    - If blood pressure cannot be adjusted with two drugs, add a third drug from the aforementioned 4 classes.
    - If goal blood pressure cannot be reached with three drugs, or if you cannot use any drugs due to contraindications, use drugs from other classes e.g. Centrally acting, alpha blockers, or beta blockers.

- **Secondary HTN**
    - Testing for secondary HTN can be expensive and should only be done when you suspect a secondary cause:
        - New onset HTN if the patient is less than 30, or above 50
        - If HTN is refractory to treatment (requiring more than three antihypertensive drugs)
        - If physical examination, imaging, or lab investigations reveal:
            - Hypokalemia
            - Epigastric bruits
            - Differential blood pressure between the arms and legs
            - Episodic HTN
            - Flushing
    - **Pathophysiology:**
        - Blood pressure is cardiac output times systemic vascular resistance
        - Cardiac output is heart rate times stroke volume
        - Stroke volume is equal to cardiac contractility multiplied by venous return
        - Venous return is controlled by venous tone and blood volume
        - Each of these factors is further regulated by other mechanisms
    - **Causes of secondary HTN:**
        - Intrinstic renal disease (MOST COMMON):
            - Abnormal sodium and water balance
            - Increased sodium and water retention
            - Depletion of antagonists of vasodepressors (prostaglandins)
            - Decreased effect of vasodilators
            - If the cause of renal disease is treatable, like GN, we treat it.
            - If the patient has proteinurea due to diabetes, give ACE-I or ARB's to decrease proteinurea
        - Renovascular disease (1-3%): When the renal artery is narrowed, the kidney's perfusion decreases. When that happens, the juxtaglomerular cells sense that this lack of perfusion, and release rennin. Rennin release activates RAAS causing excessive aldosterone production. Aldosterone is responsible for the sustained high blood pressure. Bilateral artery stenosis or the presence of a solitary kidney leads to rapid volume expansion. In normal functioning kidneys, the working kidney can compensate by excreting the excess volume. Renal artery stenosis is characterized by sudden onset HTN or accelerated malignant HTN. On examination, you hear an epigastric bruit. If you place the patient on ACE-I, in case of bilateral artery stenosis, an acute kidney injury will ensue. This happens due to the decrease in GFR which is caused by the effect of ACE-I on the

efferent arterioles. Testing for renal artery stenosis can be done through duplex ultrasound, captopril renography, or MRI. Treatment is with ACE-I or ARB's (if the stenosis is unilateral). Kidney function tests should be performed three weeks after the initiation of treatment. Stenosis is treated only when symptoms arise. The following are causes of renovascular disease:

- Atherosclerosis:
  - Affects males more than females
  - After the age of 50
  - Association with HTN and diabetes
  - Proximal part of the artery (ostium)
  - Treated with ballooning or stenting. Stenting cures a third, improves a third, and leaves the disease unchanged in a third.
  - High risk for developing ESRD
- Fibromuscular dysplasia:
  - young females (15-40)
  - Disease in the media of the artery
  - The artery looks beaded
  - Involves distal renal artery (can involve mid-artery)
  - Complete occlusion of the artery is rare
  - Treated with angioplasty; success rate is 80-100%. Restenosis happens in 10%. Cure of HTN in 60%.
- Aortic dissection
- Renal artery dissection
- Takayasu's arteritis
- Thromboembolic events
- Post-radiation
- Post-transplant

▪ Hyperaldosteronism (Cohn's syndrome):

- 0.5-2%
- Adenoma or bilateral hyperplasia of the adrenals
- Patients might be asymptomatic
- Hypokalemia is present (some patients complain of muscle cramps)
- Metabolic alkalosis
- Retinopathy in sever HTN
- Sodium retention
- Diagnosed by the ration between Plasma Aldosterone and Plasma Renin activity. A ratio between 20-45 is abnormal and indicates a

disease. Plasma aldosterone more than 15 and plasma rennin activity less than 1 indicate a problem.
    - The confirmatory test is an infusion of 2.5 liters of normal saline over a period of two hours followed by taking a plasma sample. A serum aldosterone of less than 8.5 after the infusion, rules out the disease.
    - After diagnosis, we need to find a cause. A CT is performed; if the cause is an adenoma, we remove the glands. If the cause is hyperplasia, medical treatment is advised.
    - It is important to treat these patients with aldosterone antagonists (spironlactone), as they restore potassium to its normal levels.
    - Blood pressure response to spironlactone is an indicator of the surgical outcome.
- Obstructive Sleep Apnea
    - Patients are usually obese and short
    - OSA is linked to multiple cardiovascular issues including atrial fibrillation, heart failure, and resistant HTN.
    - Published reports state that 33% of patients with essential HTN have OSA, while 50% of patients with OSA have HTN.
    - Prospective studies show a strong a link between apnea/hypopnea index and HTN independent of other risk factors for HTN.
    - OSA is characterized by daytime sleepiness, loss of concentration, snoring, morning headaches, episodes of apnea during sleep.
    - Treatment is with weight loss, CPAP, and, sometimes, surgical solutions might be considered.
- Pheochromocytoma:
    - 0.11%
    - Chromaffin tumore cells that secrete excess catecholamines
    - Young female patients
    - Present with intermittent HTN, palpitations, sweating, and anxiety.
    - Episodes might be precipitated by tyramine rich foods (cheese, beer, and wine), pain, trauma, or certain drugs (TCA, opiates, clonidine)
    - Diagnosis with plasma free metanephrines, 24 hours hour urine metanephrine, or 24 hour PMA.
    - Treated with surgical removal of the tumor. Complications of surgery include renal artery ligation, post-operative hypoglycemia, hemorrhage, or volume loss. Mortality rate is less than 2%

        - Anesthesia: avoid benzodiazepines and barbiturates as they might trigger catecholamine release.
        - 5-year survival rate is 95% with 10% recurrence rate.
        - Best drugs to be used pre-opertatively are alpha blockers as beta blockers might cause a hypertensive emergency in the OR.
- Glucocorticoid excess (Cushing's syndrome or disease):
    - 0.1-0.6%
    - The most common cause is iatrogenic due to excessive use of steroids.
    - Characterized with: sudden weight gain, abdominal striae, moon face, trunkal obesity, DM, proximal muscle wasting, hirsutism, and skin atrophy
    - Diagnosed with 24 hour urine free cortisone, midnight saliva sample, or dexamethasone suppression test
    - Treatment depends on the cause. Can be treated with transsphenoidal resection of the pituitary if the disease involves the pituitary. Adrenalectomy, or tumor lung resection…
- Coarctation of the aorta
    - Narrowing of the aorta past the subclavian artery.
    - Blood pressure in the upper limbs is normal
    - Low blood pressure in the lower limbs
    - Radiofemoral delay
    - Decreased or absent femoral pulses
    - Treatment is either surgical or with stenting
    - Associated with bicuspid aortic valve
    - If uncorrected, 70% of the patients will develop heart failure by the age of 40.
- Hyperthyroidism:
    - 33% of the patients will develop HTN
    - Other obvious signs of thyrotoxicosis are usually present.
    - Treated with radioactive iodine ablation or thyroidectomy
- Hypothyroidism
    - HTN develops due to increased peripheral vascular resistance and decreased cardiac output
    - Mostly leads to diastolic hypertension

Cardiomyopathies

- Cardiomyopathies are a group of disorders involving the myocytes.
- The WHO classification of cardiomyopathies is based on the anatomic and physiological findings:
    - Dialated cardiomyopathy: enlarged heart, systolic dysfunction
    - Hypertrophic cardiomyopathies: thickened heart, diastolic dysfunction
    - Restrictive cardiomyopathy: diastolic dysfunction
    - Arryhtmogenic right ventricular dysplasia (ARVD): fibro-fatty replacement of the myocytes in the right ventricle
    - Unclassified: left ventricular non-compaction cardiomyopathy
- **Dilated cardiomyopathy:**
    - Ventricle is dilated
    - Can be caused by:
        - IHD
            - The main cause of dilation is an infarction
            - The infarction leads to necrosis, which if left untreated, will leave dead muscle tissue.
            - Muscle remodeling causes morphological changes in the left ventricle (globular instead of an elliptical heart)
            - The remodeling is a pathological process that leads to a change in the shape, thus a change in the normal function of the heart
            - The orientation of the papillary muscles (anterolateral and postermedial) is changes leading to mitral regurgitation, which is a common finding with a dilated left ventricle
            - Impairment of contractility and loss of systolic function without heart failure.
                - Heart failure is a volume overload problem (signs of overload always present). You can have heart failure with preserved systolic function.
                - Some patients have a low EF without heart failure; these patients can be euvolemic.
                - Systolic dysfunction is related to heart failure, but not in 1:1 fashion.
        - Valvular heart disease
        - Postpartum cardiomyopathy
            - 1 month before delivery or 5 months after delivery
            - Inflammatory cytokines are involved in the pathology

        - More in African Americans
        - More than 30
        - Smokers
        - Multiple pregnancies
        - Good prognosis with treatment
        - Not advised to have a baby again if EF is less than 50
        - EF less than 30 is an absolute contraindication
        - Complete recovery is a relative contraindication. But the patient has to understand the risks.
    - Toxins like alcohol
        - Chronic use of alcohol
        - Reversible with abstinence
    - **Broken heart syndrome (Stress induced, Takotsubo)**
        - **Female**
        - **More common in 50's**
        - **Apical ballooning**
    - Viruses like coxsackie B virus and echovirus:
        - Post viral
        - Due to a self limited infection
        - Young patients
        - Do not respond to immunosuppressive or steroid therapy
        - Mechanism of failure: cell death and fibrosis
    - idiopathic dilated cardiomyopathy: incidence is 5-8/100,000
        - Here, the incidence might be higher, because many patients with idiopathic cardiomyopathy have mild to no symptoms.
        - Familial idiopathic about 30% of all idiopathic cases. The inheritance pattern is variable. Changes in myosin and actin encoding genes.
        - The mechanism of failure is through failure of energy production and contractile force generation.
- Dilated cardiomyopathy occurs more in males than in females.
- More common in African Americans
- Low ejection fraction is associated with high risk of sudden cardiac death due to arrhythmias (ventricular tachycardia and ventricular fibrillation)
- Prognosis (in descending order):
    - Postpartum
    - Idiopathic next
    - IHD
    - Infiltrative hemochromatosis

        - Doxorobucin induced (a chemotherapeutic agent that leads to myocardial cell death)
        - HIV
    - Patients with HF, regardless of the etiology, have shared findings of:
        - overload
        - Tachycardia
        - Edema
        - Hypotension
        - Cold extremeties
        - Pulsus alternans
        - Venous congestion
        - Laterally displaced PMI
        - S3
        - Mitral reurge murmur
        - Increased JVP
        - Splenomegaly
        - Ascites
        - On X-ray, you will see an enlarged heart with pulmonary edema
        - Elevated BNP
    - Echocardiogram will show a dilated left ventricle
    - during catheterization look for hemodynamics and coronary etiologies If you need to, do an endomyocardial biopsy
    - With low EF, survival decreases due to different arrhythmias
    - These patients have different frequencies of PVCs, which form a nidus for ventricular tachycardias. With normal EF, PVC's are insignificant; with low EF, more than 10 PVC/ hour is a bad prognostic sign

- **<u>Hypertrophic cardiomyopathy:</u>**
    - Needs to be differentiated from an athletic heart:
        - The septum is not thicker than 1.5 cm
        - End diastolic dimensions do not get beyond 4.5 cm
        - Left atrium is less than 4
        - Hypertrophy is always concentric and regresses after three months of stopping the athletic activity.
        - These criteria are reassuring. If in doubt, ask the patient to stop athletic activity and reassess in three months.
    - Types:
        - Familial: AD with variable inheritance
        - Sporadic: result of spontaneous mutations

- HOCM is a widely hetergenous disease
- It is the most common cause of death in people less than 35 years of age
- The degree of LVH is directly correlated with the incidence of death
- Young patients with extreme hypertrophy might have very few symptoms
- Symptoms:
    - Angina
    - HF
    - Syncope
    - Sudden death (might be the only presentation)
- LVH is not due to pressure overload; it is genetic and has nothing to do the pressure overload state
- They have supernormal systolic function with an impairment in the diastolic function. Impaired diastolic relaxation and increased diastolic pressures
- Prevalence as high as 1 in 500 in the general population
- Genetic forms:
    - Beta myosin heavy chain
    - Cardiac troponin T and this form
    - Myosin protein binding C
    - Alpha tropomyosin
    - The remainder are rare mutations
- Eccentric hypertrophy involving the septum called ASH (Asymmetric septal hypertrophy). The septum is at least 1.4 times the size of the posterior wall
- The hypertrophy can be:
    - Subaortic
    - Septal
    - Midventricular
    - Apical
    - Diffuse
- The myocyte hypertrophy leads to ventricular arrhythmias
- The syncope usually happens due to an arrhythmic focus
- The angina and dyspnea have to do with the diastolic dysfunction that happens due to the hypertrophy
- Presence of SAM (systolic anterior motion):
    - Pulling of the mitral valve towards the ventricular outflow tract causing obstruction
    - Ejection systolic murmur… caused by the movement of the mitral valve towards the septum blocking the outflow
    - Valsalva and standing increase these murmurs because they decrease the preload decreasing the volume of the ventricle. This increases the chances for obstruction as the ventricle gets smaller with a decreased preload.

        - With large volumes, there will be a separation between the anterior mitral valve leaflet and the septum (murmur is less audible)
    - Symptoms increase in summer due to increase in insensible volume losses
    - Diagnostic criteria are:
        - ECG
        - Echocardiogram: dagger shaped envelope
        - Cathetarization if needed: reveals a separation in the gradient following a PVC called Brockenbrough-Braunwald phenomenon.
    - They need an ICD
    - Increased symptoms and thus need for ICD:
        - Cardiac arrest
        - Sustained ventricular tachycardia
        - Multiple familial sudden deaths
        - Recurrent syncope
        - Adverse genotypes
        - Multiple repetitive non-sustained ventricular tachycardias
        - Massive LVH (30 mm septum)
    - Risk of death increases with increasing septal thickness
    - Symptoms are a result of diastolic function due to a gradient across left ventricular hypertrophy
    - Treated with beta blockers and non dihydropyridine CCB's
        - Negative inotropic and chronotropic effects.
        - Digoxin is avoided
    - If patients do not respond to medical therapy, surgical myomectomy or alcohol septal ablation are recommended.
        - Alcohol is injected through the artery that feeds the septum causing fibrosis which improves cardiac function
    - Mitral regurgitation is a common association due to SAM.
    - Mitral regurgitation usually improves with the procedure. If it doesn't, mitral surgery is recommended
    - Apical hypertrophy is called the Japanese variant. It has a better prognosis. Characterized by T wave inversion in Lead III and V6

- **<u>Restrictive cardiomyopahty:</u>**
    - Problem in filling (diastolic problem)
    - Ventricle is stiff and rigid while there is normal systolic function
    - Intraventricular pressure increases with small amounts of volume
    - Infiltration of the myocytes with abnormal substances leads to fibrosis and scarring

- On echo, the atria are dilated while the ventricle's size is normal
- Progressive worsening of the diastolic function
- Amyloid deposit is the pro-example of restrictive cardiomyopathy
- Granular sparkling on echo
- Increased thickness of the papillary muscles, interatrial septa, valve leaflets and left ventricular wall thickness
- Valvular regurgitation
- Thrombi in the atrial appendages
- Minimal pericardial effusion
- Dilation of the atria
- Microscopic findings: polarized light microscopy yellow birefringence due to amyloid tissue. Congo red stain
- Seen mostly with patients with ESRD and multiple myeloma
- AL and AA type
- Causes:
    - Senile
    - Familial
    - chronic inflammatory
    - Multiple myeloma
- Diagnosis:
    - Biopsy from the gingiva or rectum
- Treatment is very difficult. Poor response and poor prognosis. Alkylating agents can be used
- Sudden death is one of the most common manifestations
- Conduction disturbances and heart failure in non fatal conditions
- Cardiac dysfunction is often severe and progressive

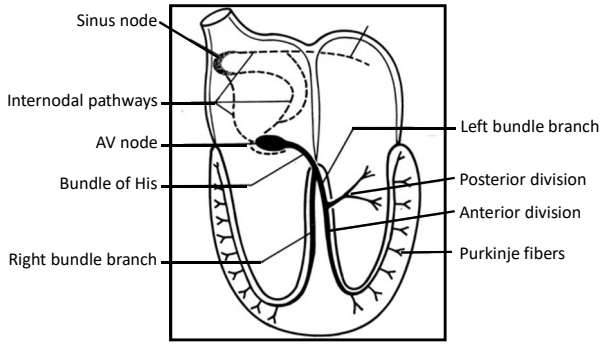
Non-compaction and eosinophilic (required but not explained)

CVS Slides

Bradycardia

Omar Obeidat, MD, FACC

Cardiac Conduction System 1

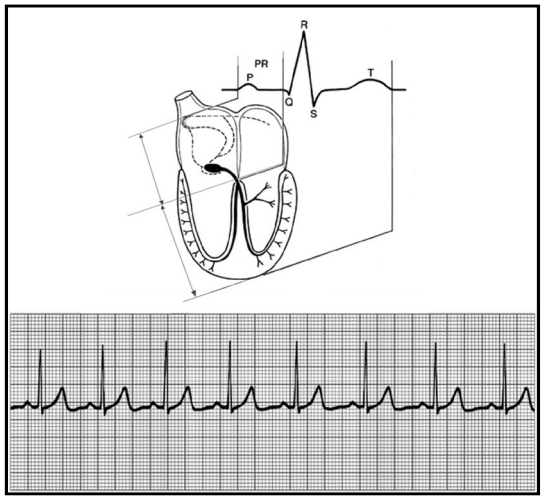


Rates of Intrinsic Cardiac Pacemakers

- Primary pacemaker
  - Sinus node (60-100 bpm)
- Escape pacemakers
  - AV junction (40-60 bpm)
  - Ventricular (<40 bpm)

Normal sinus rhythm

- Rhythm of the healthy human heart.
- Rate: 60 and 100 b/m
- P wave upright in leads I, **II** and aVL, and a negative P wave in lead aVR.
- The duration generally around 80 milliseconds, and the amplitude is <0.25 millivolts.



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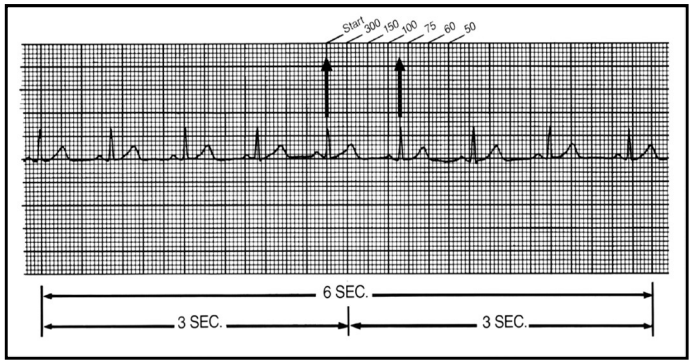
Determining the Rate



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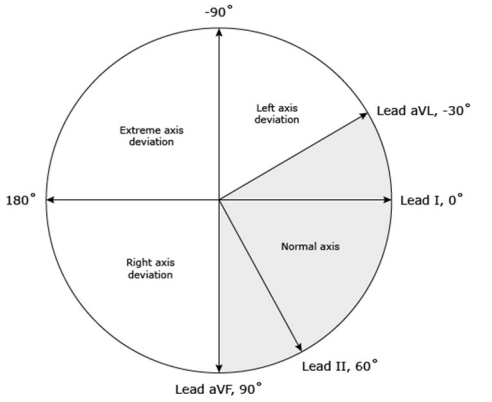
Calculation of frontal plane axis



If the QRS complex is positive in leads I and II, it falls between -30 and 90° and is normal, as indicated by the yellow area. If the QRS complex is negative in I and positive in aVF, there is right axis deviation. If the QRS complex is positive in I and negative in II, there is left axis deviation. If the QRS complex is negative in I and aVF, there is extreme axis deviation.

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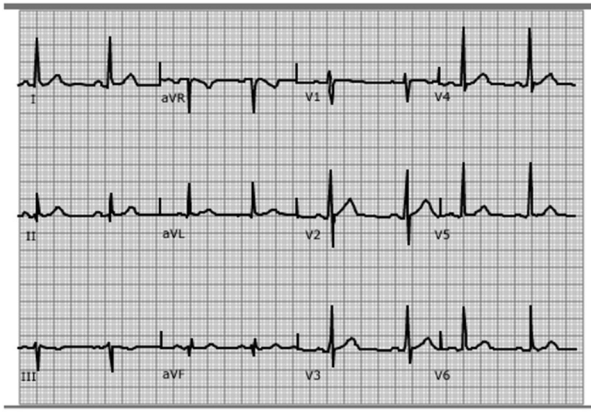
Normal ECG



Normal sinus rhythm at a rate of 71 beats/min, a P wave axis of 45°, and a PR interval of 0.15 sec.

Courtesy of Morton Arnsdorf, MD.

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Bradycardia

- Bradycardia < 60 beats per minute
- Sinus bradycardia sinus rhythm <60 beats per minute

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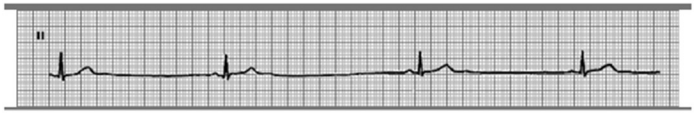
Single lead electrocardiogram (ECG) showing sinus bradycardia



Marked sinus bradycardia at a rate of 25 to 30 beats/min. The normal P waves (upright in lead II) and PR interval are consistent with a sinus mechanism with normal atrioventricular (AV) conduction.

Courtesy of Ary Goldberger, MD.

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Bradycardia: ETIOLOGY

- Well-conditioned athletes: increased vagal tone
- **Sick sinus syndrome**
- **Medications**
- Acute myocardial infarction
- Obstructive sleep apnea
- Exaggerated vagal activity: carotid sinus stimulation, vomiting, coughing, and Valsalva maneuver
- Increased intracranial pressure and other CNS conditions
- Infectious causes: Lyme disease, Chagas disease, etc
- Other: **hypothyroidism**, anorexia nervosa, hypothermia, severe prolonged hypoxia and long QT syndrome

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Bradycardia medications

- Parasympathomimetic agents (eg, acetylcholine, carbachol, acetylcholinesterase inhibitors)
- Sympatholytic drugs (eg, **beta blockers**, methyldopa, **clonidine**)
- Opioids and sedatives
- Cimetidine
- **Digitalis**
- Non-dihydropyridine **CCB(diltiazem and verapamil**)
- Ivabradine
- **Amiodarone** and other antiarrhythmic drugs
- Lithium
- Chemotherapeutic agents (eg, thalidomide, lenalidomide, paclitaxel)
- Organophosphate compounds

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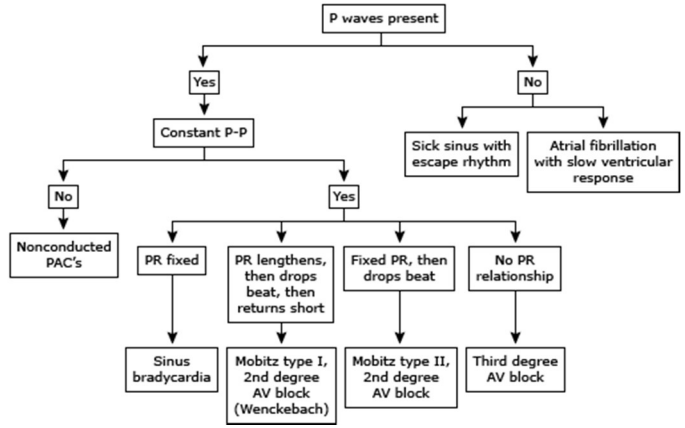
Approach to bradycardia



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Analyzing Rhythm Strips

- Key questions
  - Are QRS complexes present?
  - Are P waves present?
  - How is the P wave related to the QRS complex?

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Relationship of P Waves and QRS Complexes

- Every P wave is followed by a QRS complex with a normal P–R interval
- Every P wave is followed by a QRS complex but the P–R interval is prolonged
- Some P waves are ***not*** followed by a QRS complex; more P waves than QRS complexes

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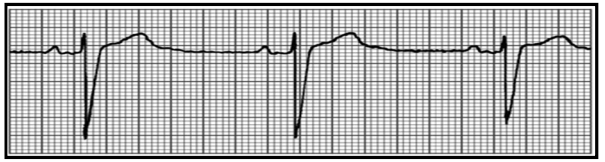
What Is This Rhythm?

Sinus Bradycardia



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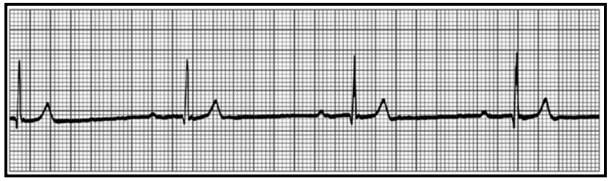
What Is This Rhythm?

First Degree AV Block



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FIRST DEGREE A-V HEART BLOCK

- **Rate:** variable
- **P wave:** normal
- **QRS:** normal
- **Conduction:** impulse originates in the SA node but has **prolonged conduction in the AV junction**;
- **P-R interval is > 0.20** seconds.
- **Rhythm:** regular
- This is the most common conduction disturbance. It occurs in both **healthy and diseased hearts.**
- First degree AV block can be due to:
  - inferior MI,
  - digitalis toxicity
  - hyperkalemia
  - increased vagal tone
  - acute rheumatic fever
  - myocarditis.
- Interventions include treating the underlying cause and observing for progression to a more advanced AV block.

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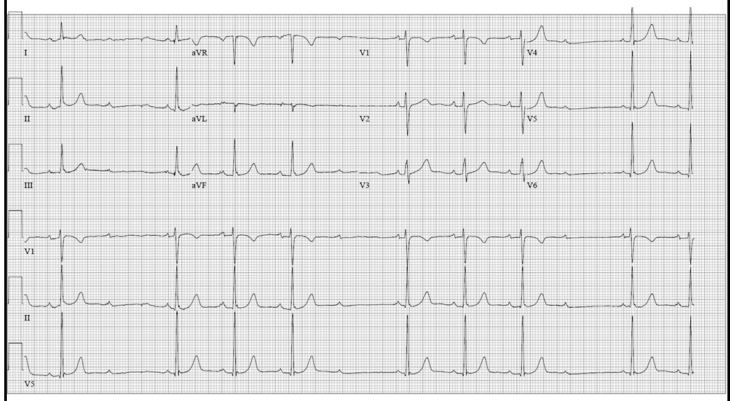
Type I, 2nd degree AV block (Wenckebach)



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SECOND DEGREE A-V BLOCK MOBITZ TYPE I (WENCKEBACK)

- **Rate:** variable
- **P wave:** normal morphology with constant P-P interval
- **QRS:** normal
- **Conduction:** the **P-R interval is progressively longer until one P wave is blocked**; the cycle begins again following the blocked P wave.
- **Rhythm:** irregular
- Second degree AV block type I occurs **in the AV node above the Bundle of His.**
- It is often transient and may be due to acute inferior MI or digitalis toxicity.
- Treatment is usually not indicated as this rhythm usually produces no symptoms.

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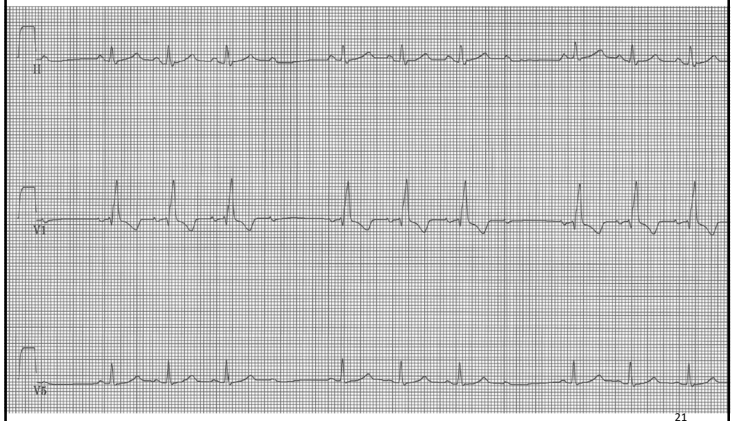
Type 2, 2nd degree AV block



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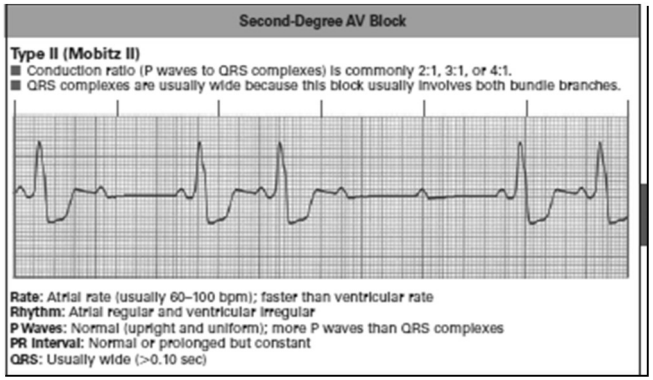
Second Degree AV Block Type II



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SECOND DEGREE A-V BLOCK MOBITZ TYPE II

- **Rate:** variable
- **P wave:** normal with constant P-P intervals
- **QRS:** usually widened because this is usually associated with a bundle branch block.
- **Conduction:** **P-R interval may be normal or prolonged, but it is constant until one P wave is not conducted to the ventricles.**
- **Rhythm:** usually regular when AV conduction ratios are constant
- This block **usually occurs below the Bundle of His** and may progress into a higher degree block.
- It can occur after an acute anterior MI due to damage in the bifurcation or the bundle branches.
- It is more serious than the type I block.
- Treatment is usually artificial pacing.

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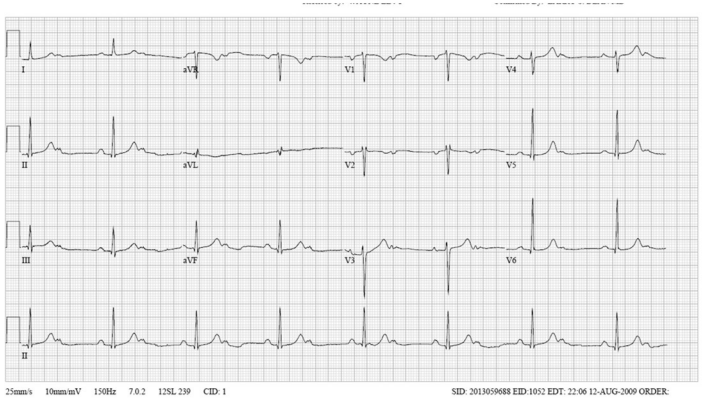
2:1 AV block



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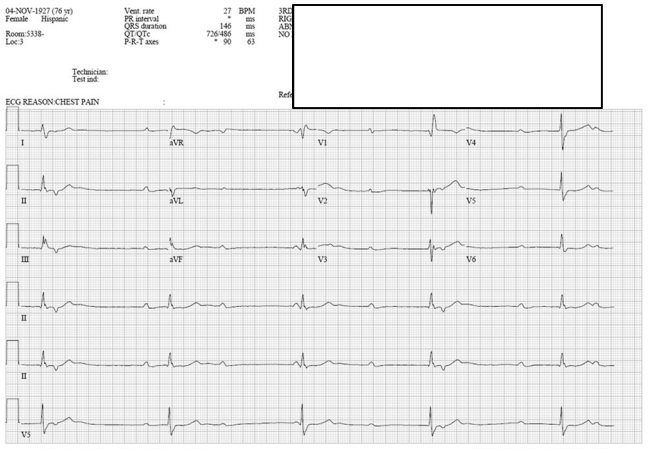
Complete heart block



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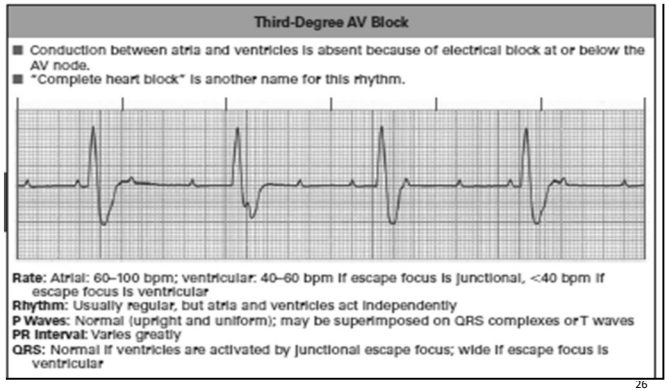
Third Degree AV Block



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THIRD DEGREE (COMPLETE) AV block

- **Rate:** atrial rate is usually normal; ventricular rate is usually less than 70/bpm. The atrial rate is always faster than the ventricular rate.
- **P wave:** normal with constant P-P intervals, but not "married" to the QRS complexes.
- **QRS:** may be normal or widened depending on where the escape pacemaker is located in the conduction system
- **Conduction: atrial and ventricular activities are unrelated** due to the complete blocking of the atrial impulses to the ventricles.
- **Rhythm:** irregular
- Complete block of the atrial impulses occurs at the A-V junction, common bundle or bilateral bundle branches.
- **Another pacemaker distal to the block takes over in order to activate the ventricles** or ventricular standstill will occur.
- May be caused by:
  - digitalis toxicity
  - acute infection
  - MI and
  - degeneration of the conductive tissue.
- Treatment modalities include:
  - external pacing and atropine for acute, symptomatic episodes and
  - permanent pacing for chronic complete heart block.

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Bradycardia Management

- Usually, HR <40bpm, to be symptomatic
- Is the patient symptomatic?
  - Mental status changes, hypotension, angina, shock, heart failure
- Acute or chronic
- Are they sleeping? Do they have sleep apnea?
- Not everyone with bradycardia, even complete heart block, needs acute treatment if stable

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Management

- Atropine 0.5mg Q3-5min, max 3mg
  - Avoid if cardiac transplant (may worsen block)
- Dopamine infusion
- Epinephrine infusion
- Isoproterenol infusion
- Glucagon if beta-blocker overdose
- Transvenous pacing (Sedate, call cardiology)

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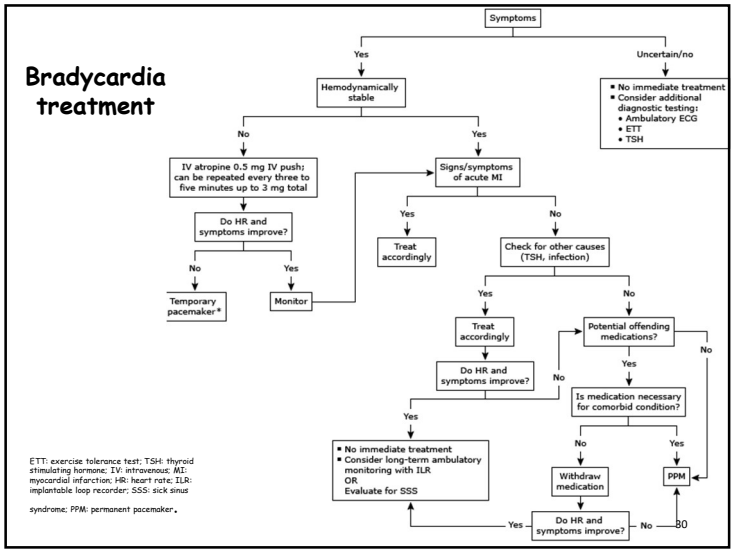


Code: Bradycardia Algorithm (1 of 2)

Bradycardia

Heart Rate less than 60 bpm and inadequate for clinical condition

- Maintain patent airway; assist breathing as needed
- Give oxygen
- Monitor ECG (identify rhythm), blood pressure, oximetry
- Establish IV access

Serious signs or symptoms of poor perfusion caused by the bradycardia?

(eg. acute altered mental status, ongoing chest pain, hypotension or other signs of shock)

31

Code: Bradycardia Algorithm (2 of 2)

Adequate Perfusion

Poor Perfusion

Observe/Monitor

Reminders

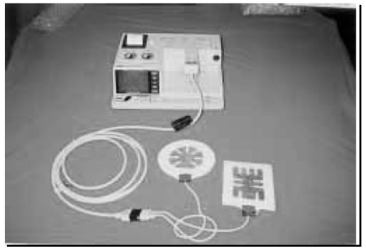
- *If pulseless arrest develops* go to Pulseless Arrest Algorithm
- Search for and treat possible contributing factors:
  - Hypovolemia
  - Hypoxia
  - Hydrogen ion (acidosis)
  - Hypo/Hyper-kalemia
  - Hypoglycemia
  - Hypothermia
  - Toxins
  - Tamponade, cardiac
  - Tension pneumothorax
  - Thrombosis (coronary or pulmonary)
  - Trauma (hypovolemia, inc ICP)

- *Prepare for transcutaneous pacing;* use without delay for high degree block (type II second -degree block or third-degree AV block)
- *Consider Atropine* 0.5 mg/IV while awaiting pacer. May repeat to a total dose of 3 mg. If ineffective, begin pacing.
- *Consider Epinephrine (*2 to 10 µg/min) or *dopamine (*2 to 10 µg/kg/min infusion while awaiting pacer or if pacing ineffective.

- Prepare for transvenous pacing
- Treat contributing causes
- Consider expert consultation

32

Transcutaneous Pacing



33

Sick Sinus Syndrome (SSS)

- Chronic sinoatrial (SA) node dysfunction: bradycardia, sinus pauses, sinus arrest
- In the presence or absence of atrioventricular (AV) nodal conduction disturbances
- May also manifest as **chronotropic incompetence** with inappropriate heart rate responses to physiological demands during activity.
- SSS can also be accompanied by atrial tachyarrhythmias as part of the **tachycardia-bradycardia syndrome.**

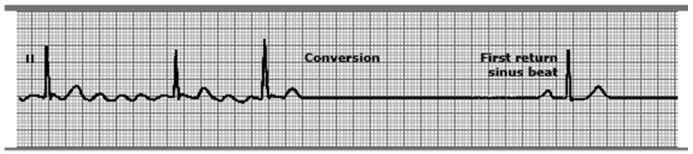
34

SSS clinical presentation

- Asymptomatic
- Symptomatic: fatigue, lightheadedness, palpitations, **presyncope, syncope**, dyspnea on exertion, or chest discomfort.
- Symptoms are frequently intermittent with gradual progression in frequency and severity,

35

Single lead electrocardiogram (ECG) showing sick sinus syndrome and atrial fibrillation



Rhythm strip showing sick sinus syndrome. The initial part (left) of the tracing reveals coarse atrial fibrillation with irregular ventricular response in the absence of drugs that slow AV nodal conduction. The atrial fibrillation terminates and is followed by a sinus beat with a prolonged sinus node recovery time of nearly four seconds.

Courtesy of Ary Goldberger, MD.

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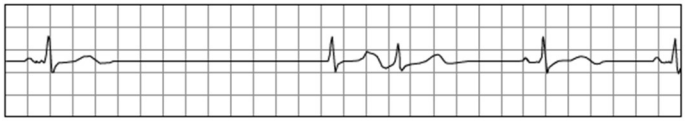
Single-lead electrocardiogram (ECG) showing manifestations of sick sinus syndrome (SSS) with sinus arrest



Example of sick sinus syndrome (SSS). In this example, sinus arrest is seen with a junctional escape beat, a premature atrial complex, and eventual resumption of sinus activity.

Courtesy of Alan Cheng, MD, FACC, FAHA, FHRS.

37

SSS evaluation

- Comprehensive history and physical examination
- Resting 12-lead ECG, review of prior records and ECG tracings, May need Holter or event moniter
- exercise stress testing: excluding myocardial ischemia, chronotropic incompetence
- medication use eg, beta blockers, calcium channel blockers, digoxin, antiarrhythmics
- R/o myocardial ischemia, hypothyroidism

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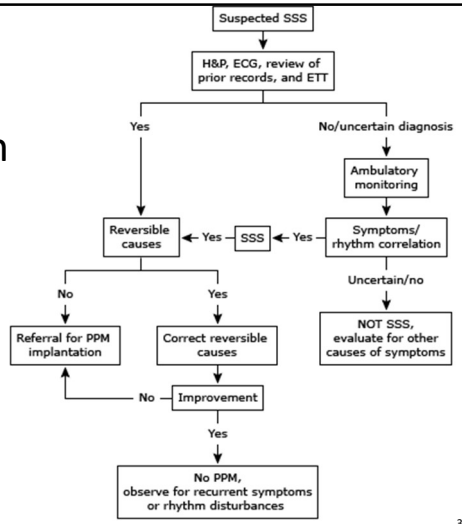
SSS Algorithm



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Questions

Thank you

40

Overview of Heart Failure
Fourth year medical students

Tareq Yousef Goussous, M.D., FACC
Interventional Cardiologis

41

- A 72-year-old-male patient with a pmhs of HTN presents with dyspnea on exertion and orthopnea for the past 5 months that got worse in the last week.
- On P/E: BP 150/80 mm Hg, HR 110 bpm, S3 & S4, 4/6 SEM in the 2 RICS, bilateral crackles & bilateral LLE.
- CXR: pulmonary vascular congestion.

42

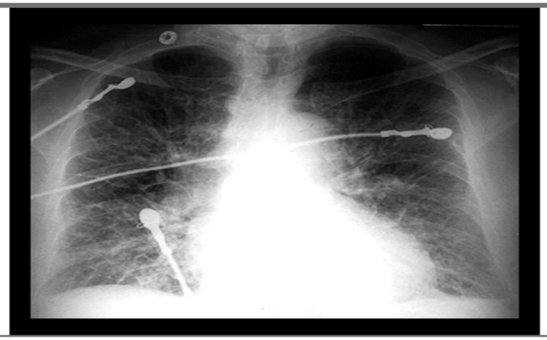
- A 28-year-old-male patient presents with dyspnea on exertion and fatigue of several months duration.
- P/E: BP 140/40 mm Hg, HR 90 bpm & 3/6 diastolic murmur at the left lower ICS.

43

- A 60-year-old-male patient who sustained an acute MI 2 weeks ago presented with acute dyspnea, orthopnea and PNDs of 1 day duration.
- On P/E: BP 100/60 mm Hg, HR 110 bpm, S3 and bilateral crackles.

44

Pulmonary edema



This plain frontal chest radiograph of a 55-year-old male with known coronary artery disease demonstrates characteristic radiographic features of heart failure with interstitial pulmonary edema, bilateral perihilar alveolar edema producing a characteristic butterfly pattern and bilateral pleural effusions.
Photo courtesy of Jonathan Kruskal, MD.

45

- A 65-year-old-female patient presented with acute dyspnea on exertion, feeling of suffocation and frothy sputum.
- P/E: BP 190/100 mm Hg, HR 115 bpm, & bilateral crackles all over the chest.

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Introduction

- Heart failure (HF) is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.
- It is characterized by specific symptoms, such as dyspnea and fatigue, and signs, such as fluid retention.
- There are many ways to assess cardiac function.

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- However, there is no diagnostic test for HF, since it is largely a clinical diagnosis that is based upon a careful history and physical examination.

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- Systolic heart failure.
- Diastolic heart failure, or heart failure with preserved ejection fraction. (relaxation & filling).

49

- Systolic dysfunction: ischemia/MI, DCMP, chronic AI/MR,
- Diastolic dysfunction: HCMP, AS, HTN, RCMP, ischemia.
- High output HF: A-V fistula, Paget's, sepsis, Beriberi, anemia, thyrotoxicosis.
- Pericardial diseases: (usually right-sided HF): tamponade and constriction.

50

- Classification of HF severity:
- The classification system that is most commonly used to quantify the degree of functional limitation imposed by HF is one first developed by the New York Heart Association (NYHA).
- This system assigns patients to one of four functional classes, depending on the degree of effort needed to elicit symptoms:

51

- Class I — symptoms of HF only at activity levels that would limit normal individuals.
- Class II — symptoms of HF with ordinary exertion.
- Class III — symptoms of HF with less than ordinary exertion.
- Class IV — symptoms of HF at rest.

52

- Stages in the development of HF:
- There are several stages in the evolution of HF, as outlined by the American College of Cardiology/American Heart Association (ACC/AHA) guidelines:

Stage A: High risk for HF, without structural heart disease or symptoms.

Stage B: Heart disease with asymptomatic left ventricular dysfunction.

Stage C: Prior or current symptoms of HF.

Stage D: Refractory end stage HF.

53

- **Etiology:**
- There are two basic pathophysiologic mechanisms that cause reduced cardiac output and HF: systolic dysfunction and diastolic dysfunction.
- Systolic and diastolic dysfunction each may be due to a variety of etiologies.
- Effective management is often dependent upon establishing the correct etiologic.

54

- Systolic dysfunction:
- The most common causes of systolic dysfunction are:
- Coronary (ischemic) heart disease.
- Idiopathic dilated cardiomyopathy (DCM).
- Hypertension.
- Valvular disease.

55

- Diastolic dysfunction:
- Diastolic dysfunction can be induced by many of the same conditions that lead to systolic dysfunction.
- The most common causes are:
- Hypertension.
- Ischemic heart disease.
- Hypertrophic obstructive cardiomyopathy.
- Restrictive cardiomyopathy.

56

- **Clinical assessment:**
- The approach to the patient with HF or cardiomyopathy includes the history and physical examination, and diagnostic tests to establish the diagnosis, assess acuity, severity and etiology.

57

- History:
- Symptoms of HF include those due to excess fluid accumulation (dyspnea, edema, hepatic congestion, and ascites) and those due to a reduction in cardiac output (fatigue, weakness) that is most pronounced with exertion.

58

- The history and other findings may be helpful in identifying the etiology of HF. As examples:
- Classic exertional angina usually indicates ischemic heart disease.
- Acute HF after an antecedent flu-like illness suggests viral myocarditis.
- Long-standing hypertension or alcohol use suggests hypertensive or alcoholic cardiomyopathy.

59

- Primary valvular dysfunction should be considered in a patient with a history of murmurs.
- A diagnosis of amyloidosis should be strongly considered in patients who have a family history of unexplained cardiomyopathy or amyloidosis, low voltage on EKG, left ventricular hypertrophy (especially without hypertension),and a history of heavy proteinuria.

60

- HF may be provoked or worsened by drugs, including antiarrhythmic agents such as disopyramide and flecainide; calcium channel blockers, particularly verapamil; beta blockers; and nonsteroidal antiinflammatory drugs (NSAIDs).

61

- Acute pulmonary edema occurring during, or shortly after, infusion of blood products suggests transfusional volume overload.

62

- Physical examination:
- There are three major manifestations of volume overload in patients with HF:
- pulmonary congestion.
- peripheral edema.
- elevated jugular venous pressure.

63

- Pulmonary congestion is more prominent in acute or subacute disease.
- Peripheral edema is manifested by swelling of the legs (which is more prominent when the patient is upright), ascites, hepatomegaly, and splenomegaly.
- Hepatojugular reflux.

64

- Manual compression of the right upper quadrant to increase venous return may elevate jugular venous pressure above the transient 1 to 3 cm elevations seen in normal individuals. This sign is known as the hepatojugular reflux.

65

- Elevated jugular venous pressure is usually present if peripheral edema is due to HF, since it is the high intracapillary pressure that is responsible for fluid movement into the interstitium.
- With the patient sitting at 45º jugular venous pressure can be estimated from the height above the left atrium of venous pulsations in the internal jugular vein.

66

- Pulsus alternans — Pulsus alternans, if present, is virtually pathognomonic of severe left ventricular failure.
- This phenomenon is characterized by evenly spaced alternating strong and weak peripheral pulses.
- It is best appreciated by applying light pressure on the peripheral arterial pulse

67

- Precordial palpation — Ventricular chamber size can be estimated by precordial palpation.
- An apical impulse that is laterally displaced past the midclavicular line is usually indicative of left ventricular enlargement.

68

- Heart sounds — An S3 gallop is associated with left atrial pressures exceeding 20 mmHg, increased left ventricular end-diastolic pressures (>15 mmHg) and elevated serum brain natriuretic peptide concentrations.

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Modified Framingham clinical criteria for the diagnosis diagnosis of heart failure

Major
Paroxysmal nocturnal dyspnea
Orthopnea
Elevated jugular venous pressure
Pulmonary rales
Third heart sound
Cardiomegaly on chest x-ray
Pulmonary edema on chest x-ray
Weight loss ≥4.5 kg in five days in response to treatment of presumed heart failure
Minor
Bilateral leg edema
Nocturnal cough
Dyspnea on ordinary exertion
Hepatomegaly
Pleural effusion
Tachycardia (heart rate ≥120 beats/min)
Weight loss ≥4.5 kg in five days
Diagnosis
The diagnosis of heart failure requires that **2 major or 1 major and 2 minor criteria** cannot be attributed to another medical condition.

From Senni, M, Tribouilloy, CM, Rodeheffer, RJ, et al, Circulation 1998; 98:2282; adapted from McKee, PA, Castelli, WP, McNamara, PM, Kannel, WB. N Engl J Med 1971; 85:1441.

70

UpToDate

- **Initial tests:**
- Electrocardiogram:
- Potentially diagnostic findings on ECG include the following:
- Evidence of ischemic heart disease including evidence of prior or acute myocardial infarction or ischemia.

71

- Left ventricular hypertrophy due to hypertension.
- Low limb lead voltage on the surface ECG with a pseudo-infarction pattern (loss of precordial R wave progression in leads V1-V6) can suggest an infiltrative process such as amyloidosis.

72

- Heart block, that may be complete, and various types of intraventricular conduction defects are observed in patients with cardiac sarcoidosis.
- The presence of a persistent tachycardia such as atrial fibrillation with a rapid ventricular response may result from or lead to HF, since this arrhythmia can cause cardiomyopathy (tachycardia-mediated cardiomyopathy).

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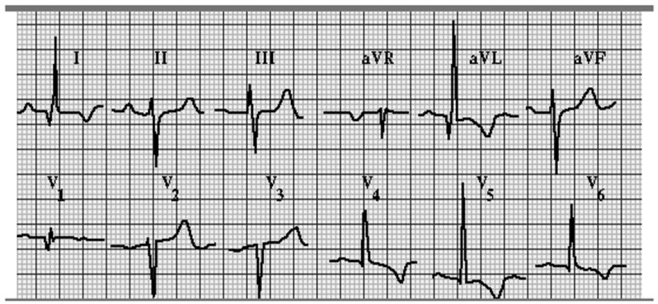
Left ventricular hypertrophy



This electrocardiogram demonstrates several features of left ventricular hypertrophy: the QRS complex is slightly widened due to an intraventricular conduction delay; there is left axis deviation; there is ST depression and inverted T waves noted in several leads; several voltage criteria are met, including an R wave in aVL which is greater than 18 mm (in this case 20 mm).

UpToDate

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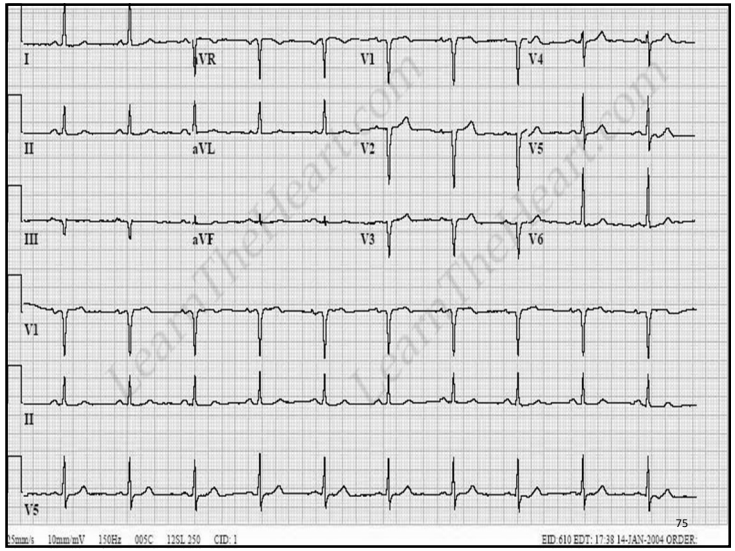

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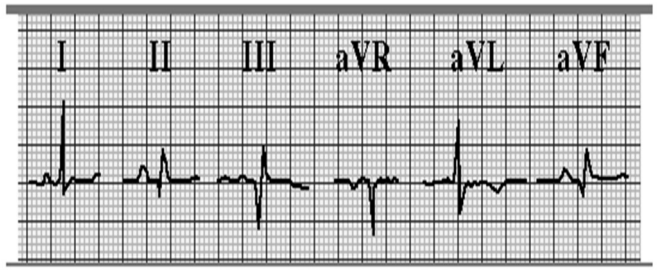
Chronic inferior wall myocardial infarction



A chronic inferior wall infarct is characterized by the presence of initial Q waves which are deep and broad in the inferior leads 2, 3, aVF.

UpToDate

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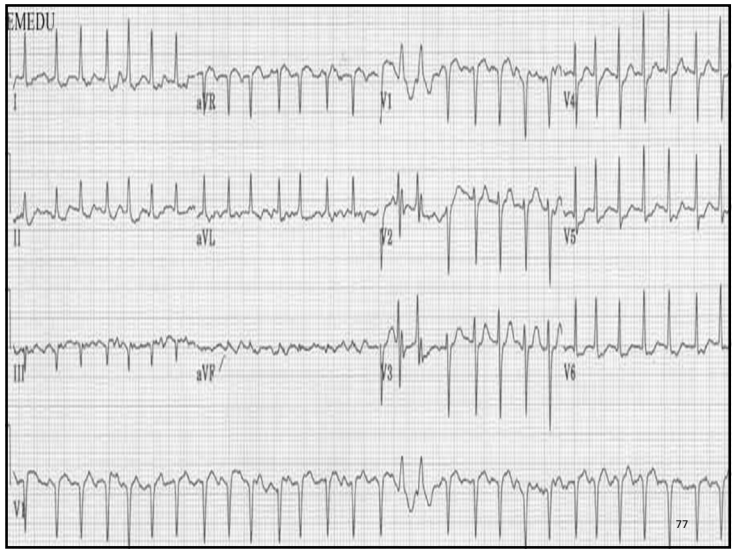
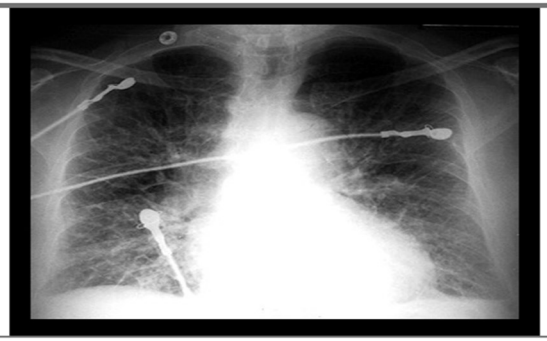


77

- Chest x-ray — A chest x-ray is generally indicated to evaluate pulmonary edema, cardiopulmonary structural abnormalities and other potential causes of dyspnea.

78

Pulmonary edema



This plain frontal chest radiograph of a 55-year-old male with known coronary artery disease demonstrates characteristic radiographic features of heart failure with interstitial pulmonary edema, bilateral perihilar alveolar edema producing a characteristic butterfly pattern and bilateral pleural effusions.
Photo courtesy of Jonathan Kruskal, MD.

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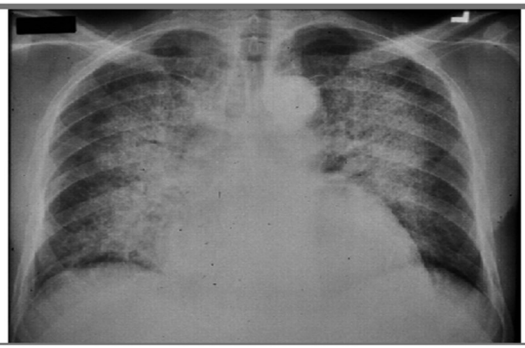
Cardiogenic pulmonary edema



Pulmonary edema in a "butterfly distribution" due to left ventricular failure. Chest radiograph shows large perihilar opacities in patient with enlarged cardiac silhouette.
Courtesy of Paul Stark, MD.

80

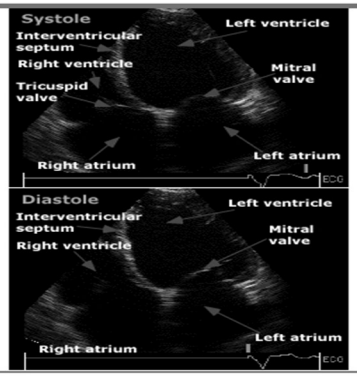
- Initial blood tests — Recommended initial blood tests for patients with symptoms and signs of HF include:
- CBC.
- Serum electrolytes,
- Creatinine & urea.
- LFT.
- FBS.

81

- **Echocardiography:**
- Echocardiography should be performed in all patients with new onset HF and can provide important information about ventricular size and function.
- For example, patients with idiopathic dilated cardiomyopathy typically have both left and right ventricular enlargement (four chamber dilatation) with decreased left systolic ventricular function.

82

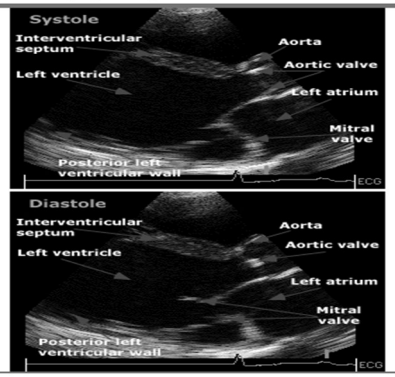
Dilated cardiomyopathy



Four chamber view from a 2-D echocardiogram shows a very dilated left ventricle which is diffusely hypokinetic. There is also enlargement of the right ventricle and right and left atria.
Courtesy of Thomas Binder, MD. University of Vienna.

83

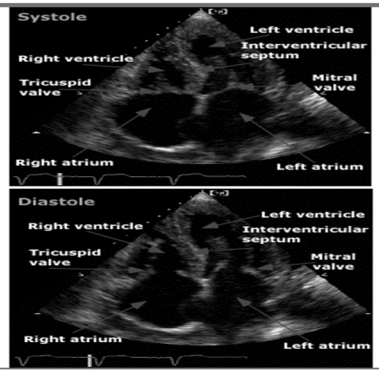
Dilated cardiomyopathy



Parasternal long axis view from a 2-D echocardiogram shows a markedly dilated and hypokinetic left ventricle. The left atrium is also enlarged and there is reduced excursion of the mitral and aortic valves suggesting a reduced stroke volume.
Courtesy of Thomas Binder, MD. University of Vienna.

84

Cardiac amyloidosis

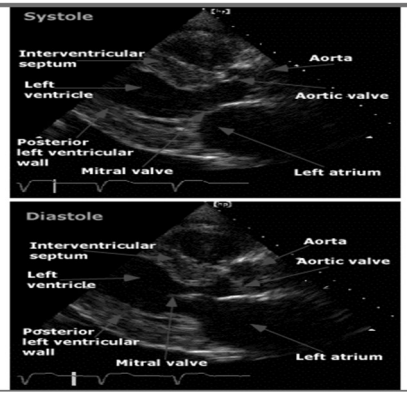


Four chamber view from a 2-D echocardiogram shows concentric hypertrophy of the right and left ventricular myocardium which has a "sparkling" appearance. The mitral and tricuspid valves are thickened and the right and left atria are dilated.

Courtesy of Thomas Binder, MD. University of Vienna.

85

Cardiac amyloidosis



Long axis view from a 2-D echocardiogram showing concentric left ventricular hypertrophy, thickened mitral and aortic valve leaflets and left atrial dilatation.

Courtesy of Thomas Binder, MD. University of Vienna.

86

Treatment

- Chronic compensated heart failure.
- Acute decompensated heart failure.

87

Factors potentially contributing to worsening heart failure

Cardiovascular factors
Superimposed ischema or infarction
Uncontrolled hypertension
Unrecognized primary valvular disease
Worsening secondary mitral regurgitation
New onset or uncontrolled atrial fibrillation
Excessive tachycardia
Pulmonary embolism
Systemic factors
Inappropriate medications
Superimposed infection
Anemia
Uncontrolled diabetes
Thyroid dysfunction
Electrolyte disorders
Pregnancy
Patient-related factors
Medication noncompliance
Dietary indiscretion
Alcohol consumption
Substance abuse

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- General principles:
- The management of HF begins with an accurate assessment of the etiology and severity of the disease.
- This is followed by a therapeutic regimen aimed at the following factors: Correction of systemic factors (eg, thyroid dysfunction, infection, uncontrolled diabetes).
- Lifestyle modification:

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- Cessation of smoking.
- Restriction of alcohol consumption.
- Salt restriction to approximately 2 to 3 g (or less) of sodium per day to minimize fluid accumulation.
- Weight reduction in obese subjects with goal of being within 10 percent of ideal body weight.
- Daily weight monitoring to detect fluid accumulation before it becomes symptomatic.

90

- Review of drugs that may contribute to HF (eg, nonsteroidal antiinflammatory drugs, antiarrhythmic drugs, calcium channel blockers, thiazolidinediones).
- Pneumococcal vaccination and annual influenza vaccination.
- Treatment of the cause of the heart disease.
- Pharmacologic therapy directed at relieving symptoms, slowing the progression of the HF, and improving patient survival.

91

- Pharmacologic therapy:
- The goals of pharmacologic therapy are to improve symptoms, slow or reverse deterioration in myocardial function, and reduce mortality.
- Additional pharmacologic therapy is directed at the prevention of arrhythmias and embolic events and the treatment of anemia and other possible exacerbating factors.

92

- A number of drugs are recommended in HF for symptom relief and improvement in outcome:
- Improvement in symptoms can be achieved by digoxin, diuretics, beta blockers, angiotensin converting enzyme (ACE) inhibitors, and angiotensin II receptor blockers (ARBs).
- Prolongation of patient survival has been documented with ACE inhibitors, beta blockers, ARBs, hydralazine/nitrates, and aldosterone antagonists.

93

- Order of therapy — the following sequence of drugs is recommended in the typical patient, with allowance for variations depending upon clinical response:
- Loop diuretics are introduced first for fluid control in patients in overt HF. The goal is relief of signs or symptoms of volume overload, such as dyspnea and peripheral edema.

94

- ACE inhibitors, or if not tolerated, angiotensin II receptor blockers (ARBs) are typically initiated during or after the optimization of diuretic therapy. These drugs are usually started at low doses and then titrated to goals based upon trial data.
- Beta blockers are initiated after the patient is stable on ACE inhibitors, again beginning at low doses with titration to trial goals as tolerated.

95

- The following drugs should be given to selected patients in the absence of a contraindication:
- The addition of an aldosterone antagonist (spironolactone or, if not tolerated, eplerenone) to improve survival in patients who can be monitored for preserved renal function and a normal plasma potassium concentration and have NYHA functional class II HF and a LVEF ≤30% ; or NYHA functional class III to IV HF and an LVEF <35%.

96

- Angiotensin II receptor blockers (ARBs) as an alternative to ACE inhibitors in patients who cannot tolerate these drugs.
- The addition of the combination of hydralazine and a nitrate for patients (particularly blacks) with a reduced LVEF who have persistent symptoms despite therapy with an ACE inhibitor and beta blocker.

97

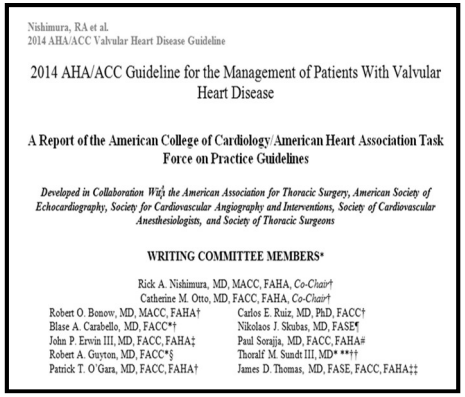
- Digoxin to reduce hospitalization for HF or for patients with concomitant atrial fibrillation, for rate control.

98

Valvular Heart Disease

Omar Obeidat, MD, FACC

99


Nishimura, RA et al.
2014 AHA/ACC Valvular Heart Disease Guideline

2014 AHA/ACC Guideline for the Management of Patients With Valvular Heart Disease

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

Developed in Collaboration With the American Association for Thoracic Surgery, American Society of Echocardiography, Society for Cardiovascular Angiography and Interventions, Society of Cardiovascular Anesthesiologists, and Society of Thoracic Surgeons

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http://content.onlinejacc.org/article.aspx?doi=10.1016/j.jacc.2014.02.536

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ACC/AHA Valve Disease Guidelines
Stage A - D

At risk for disease	Stage A
Progressive disease	Stage B
Severe disease (asymptomatic)	Stage C
Severe disease (symptomatic)	Stage D

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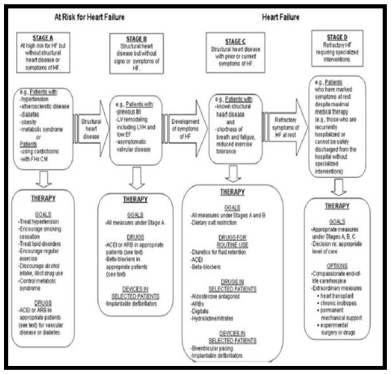
ACC/AHA Heart Failure Guidelines
Stage A - D



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ACC/AHA Valve Disease Guidelines
Stage A - D

At risk for disease	
Progressive disease	
Severe disease (asymptomatic)	C1: Compensated LV
Severe disease (symptomatic)	C2: Decompensated LV

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Valvular Heart Disease

1. Aortic Stenosis
2. Aortic Regurgitation
3. Mitral Stenosis
4. Mitral Regurgitation
5. Tricuspid Stenosis
6. Tricuspid regurgitation
7. Pulmonary valve regurgitation
8. Pulmonary valve Stenosis

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Aortic Stenosis

- Etiology/Classification
- Pathophysiology
- Clinical Manifestations
- Evaluation
- Management

105

Obstruction to left ventricular outflow

- Valvular
  - Calcific/Degenerative
  - Congenital: Biscuspid, rarely unicuspid/quadricuspid
  - Rheumatic
- Supravalvular
  - Uncommon: Williams syndrome
- Subvalvular
  - Uncommon: Fibromuscular membrane or ridge present in the LVOT
- Hypertrophic Cardiomyopathy

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Valvular Aortic Stenosis

- Calcific/Degenerative
  - Most common
  - Normal trileaflet valve develops “age-related” calcification/thickening
  - Presents later in life (age 70-85)
  - Same risk factors for atherosclerosis: HTN, smoking, dyslipidemia
- Rheumatic AS:
  - Almost always occurs concurrently with mitral valve disease

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Aortic stenosis:

- Bicuspid valve
  - Present in 1-2% of the population
  - More prevalent in men (70-80% of cases)
  - Usually presents at a younger age (45-65)
  - 50% associated with dialted root
  - Associated with coarctation of the aodrta
  - Up to 30% is familial

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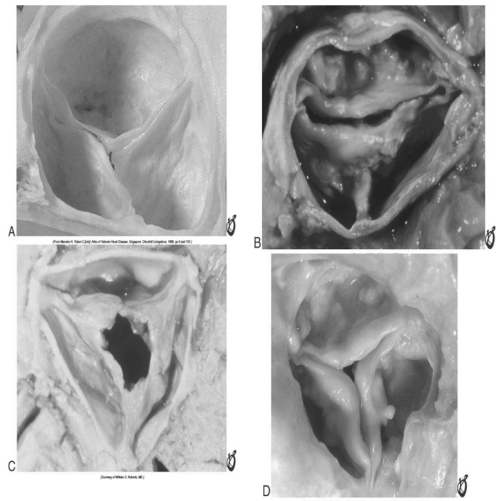

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Normal

- Normal anatomy: three thin cusps with unrestricted systolic opening
- Normal area 3-4 cm²
- Normal opening produces 2cm leaflet separation
- Normal velocity: approx 1 m/s

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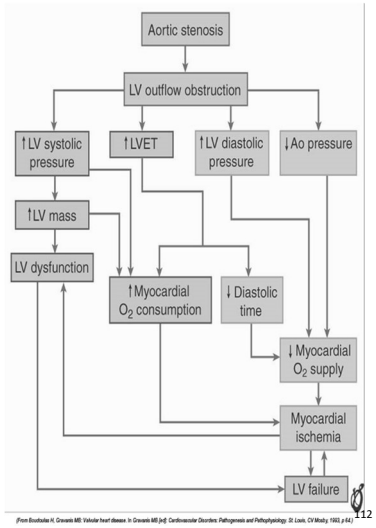
Pathophysiology

- Aortic sclerosis produces thickening of the valves, but no obstruction to outflow
- When stenosis develops, the functional area of the valve decreases and causes a measurable obstruction of outflow
- Concentric LVH develops with normal chamber size
- Diastolic dysfunction due to increased myocardial cell mass and interstitial fibrosis
- Atrial contraction plays an important role in filling of the left ventricle in AS
- Loss of appropriately timed atrial contraction could cause clinical deterioration

111

Pathophysiology

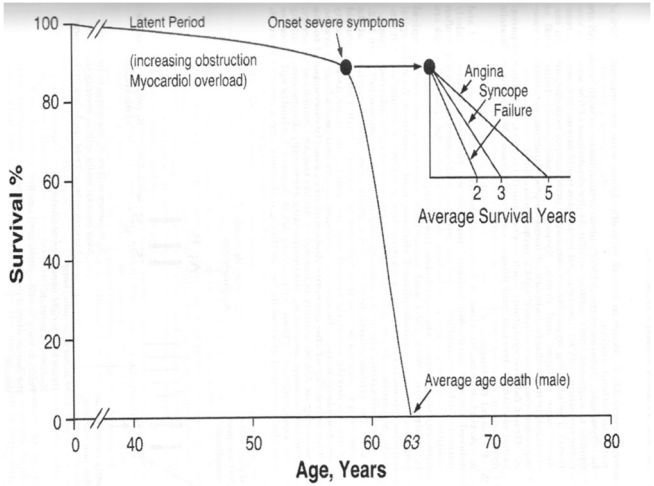


112

Symptoms

- Asymptomatic (long latency period)
- Decrease in exercise tolerance
- Fatigue
- **Angina: demand ischemia, 50% has CAD**
- **Syncope: drop in BP, can be to arrhythmias(less likely)**
- **Dyspnea/Heart Failure: diastolic dysfunction, later systolic dysfunction**

113



114

Signs (Physical Exam)

- Loud, late-peaking systolic murmur
- Radiation to the carotids
- Paradoxically split S2
- Absent A2: severe AS
- Delayed and Diminished carotid upstroke (**Pulsus parvus et tardus**)
- Low output states may affect intensity of the murmur and carotid upstroke
- Gallaverdin phenomenon
- Associated increase in GI bleeding from angiodysplasia/von Wilbrand

115

Diagnostic studies:

ECG:

- Left ventricular hypertrophy (85 percent of patients)
- Left atrial abnormality (80 percent)

CXR:

- Normal LV size or cardiomegaly
- Calcified aortic valve
- Dilatation of the ascending aorta

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Echocardiography

- Qualitative
  - 2D imaging of valve anatomy (number of leaflets)
  - Calcification/thickening, valve opening
  - Left ventricular function
  - Chamber size, wall thickness
- Quantitative
  - Peak aortic valve velocity
  - Mean and maximum pressure gradients
  - Aortic valve area (continuity equation)
  - Ratio of outflow tract to aortic jet velocity

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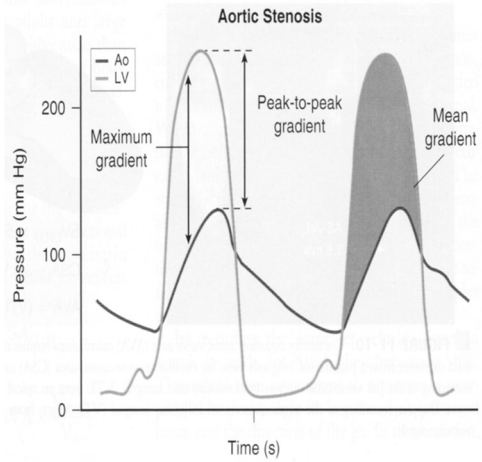
Cardiac catheterization: assess gradient and coronaries



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Severity of Aortic Stenosis

Indicator	Aortic Stenosis		
	Mild	Moderate	Severe
Jet velocity (m per second)	Less than 3.0	3.0–4.0	Greater than 4.0
Mean gradient (mm Hg)*	Less than 25	25–40	Greater than 40
Valve area (cm^2)	Greater than 1.5	1.0–1.5	Less than 1.0
Valve area index (cm^2 per m^2)			Less than 0.6

119

Classification of AS severity

Severity	Mean gradient	AVA
Mild	< 25	>1.5
Moderate	25-40	1.0-1.5
Severe	>40	<1.0

ACC/ AHA guidelines

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Natural History of Progression

- Aortic jet velocity: 0.3 m per second per year
- Increase in mean gradient pressure of 7mmHg per year
- Decrease in valve area of 0.1cm² per year

(JACC Vol 48 (3): e1-148.)

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Management

- Presence or absence of symptoms
- Severity of stenosis
- Status of the left ventricle
- Presence of comorbidities

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Aortic Valve Replacement

Class I

1. AVR is indicated for symptomatic patients with severe AS.[a] *(Level of Evidence: B)*
2. AVR is indicated for patients with severe AS[a] undergoing coronary artery bypass graft surgery (CABG). *(Level of Evidence: C)*
3. AVR is indicated for patients with severe AS[a] undergoing surgery on the aorta or other heart valves. *(Level of Evidence: C)*
4. AVR is recommended for patients with severe AS[a] and LV systolic dysfunction (ejection fraction less than 0.50). *(Level of Evidence: C)*

Class IIa

AVR is reasonable for patients with moderate AS[a] undergoing CABG or surgery on the aorta or other heart valves (see Section 3.7 on combined multiple valve disease and Section 10.4 on AVR in patients undergoing CABG). *(Level of Evidence: B)*

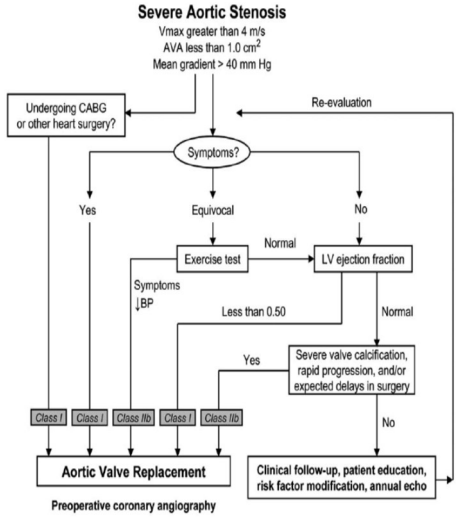
Class IIb

1. AVR may be considered for asymptomatic patients with severe AS[a] and abnormal response to exercise (e.g., development of symptoms or asymptomatic hypotension). *(Level of Evidence: C)*
2. AVR may be considered for adults with severe asymptomatic AS[a] if there is a high likelihood of rapid progression (age, calcification, and CAD) or if surgery might be delayed at the time of symptom onset. *(Level of Evidence: C)*
3. AVR may be considered in patients undergoing CABG who have mild AS[a] when there is evidence, such as moderate to severe valve calcification, that progression may be rapid. *(Level of Evidence: C)*
4. AVR may be considered for asymptomatic patients with extremely severe AS (aortic valve area less than 0.6 cm^2, mean gradient greater than 60 mm Hg, and jet velocity greater than 5.0 m per second) when the patient's expected operative mortality is 1.0% or less. *(Level of Evidence: C)*

Class III

AVR is not useful for the prevention of sudden death in asymptomatic patients with AS who have none of the findings listed under the class IIa/IIb recommendations. *(Level of Evidence: B)*

123



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Low Output Low Gradient severe AS

- LV dysfunction (<40%), Low cardiac output
- Low trans-aortic mean gradient (< 30 Hg)
- Aortic stenosis (valve area < 1.0 cm2)

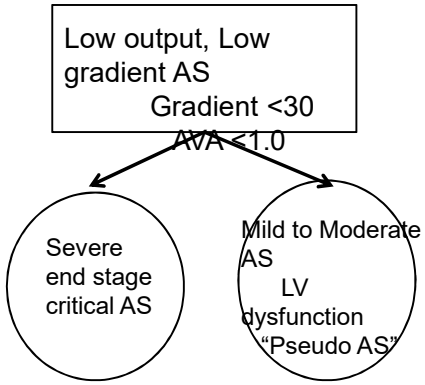
☆

☆

125

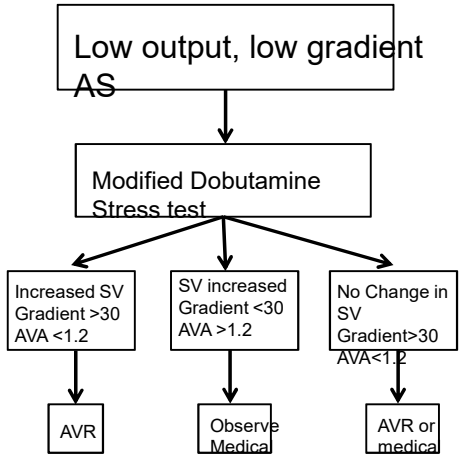
Severity	Mean gradient	AVA
Mild	< 25	>1.5
Modera te	25-40	1.0-1.5
Severe	>40	<1.0

126



The main challenges are to differentiate these two states and then to determine whether the left ventricle is likely to recover after aortic valve surgery.

127



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Aortic Regurgitation

- Epidemiology
- Etiology
- Pathophysiology
- Clinical Presentation / Symptoms
- Natural History
- Diagnosis
- Treatment

129

Epidemiology

- Prevalence
  - Framingham Offspring Study
    - 13% in men; 8.5% in women
    - Advanced age and male gender associated with AR
    - Singh,et al. *American Journal of Cardiology*.1999:83:897-902
  - Strong Heart Study (Native American Population)
    - 10% prevalence
    - Advanced age and aortic root diameter associated with AR
    - Lebowitz, et al. *JACC.* 2000;36:461-7.
  - In both studies the majority of cases were mild

130

Etiology

- Valvular (primary) vs. Aortic Root(secondary)
- Acute AR vs Chronic AR

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Acute AR

- Usually from Aortic dissection or infective endocarditis or Sinus rupture
- LV size is normal and not able to handle large regurgitant volume
- Rapid rise in LV filling pressure(short murmur, pulse pressure may not be wide, premature closure of the MV)
- This leads to acute pulmonary edema
- Diagnosis: echocardiography
- Treatment: urgent surgery

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Chronic AR:Etiology

- Valvular
  - Calcified AV with AS+AR in the elderly
  - Infective endocarditis
  - Congenital bicuspid
    - More commonly stenosis
    - Incomplete closure/prolapse can lead to isolated regurgitation
  - Rheumatic fever
    - Cusps become infiltrated with fibrous tissue and retract preventing cusp apposition during diastole
  - Less Common
    - Congenital: quadricuspid
    - Inflammatory conditions: SLE, RA, Whipple's Disease, Takayasu
    - Fenfluramine-phentermine

133

Etiology

- Aortic Root Disease
  - Age related (degenerative) aortic dilation
    - HTN longstanding
    - Atherosclerosis
  - Marfans
  - Aortic dilation related to bicuspid valve
  - Osteogenesis imperfecta
  - Syphilitic aortitis
  - Bechets, Psoriatic arthritis, GCA

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Pathophysiology

- Regurgitant flow produces increase in LVEDV
  - Thereby increasing LV diameter and raising wall tension (via Lapace's law)
  - Wall stress proportional to (Intraventricular pressure x radius)/wall thickness
- LV responds by compensatory eccentric hypertrophy of myocytes
- LV hypertrophy x increased LVEDV= increased stroke volume

135

Symptoms

- Exertional dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Angina pectoris
  - Late in clinical course
- PVCs
- Complaints may be present for many years before symptoms of overt LV dysfunction manifest

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- **Corrigan's pulse**
  - Rapid upstroke and collapse of the carotid artery pulse
- **de Mussett's sign**
  - Head nodding in time with heart beat
- **Hill sign**
  - Increase in manually measured blood pressure of the lower extremity compared to the upper extremity
- **Rosenbach sign**
  - Pulsatile liver
- **Mueller sign**
  - Pulsatile uvula
- **Becker sign**
  - Accentuated retinal artery pulsation
- **Gerhard sign**
  - Pulsatile spleen

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Severity

Table 4 Qualitative and quantitative parameters useful in grading aortic regurgitation severity

	Mild	Moderate	Severe
Structural parameters			
LA size	Normal*	Normal or dilated	Usually dilated**
Aortic leaflets	Normal or abnormal	Normal or abnormal	Abnormal/flail, or wide coaptation defect
Doppler parameters			
Jet width in LVOT –Color Flow[§]	Small in central jets	Intermediate	Large in central jets; variable in eccentric jets
Jet density–CW	Incomplete or faint	Dense	Dense
Jet deceleration rate –CW (PHT, ms)[¶]	Slow > 500	Medium 500-200	Steep < 200
Diastolic flow reversal in descending aorta –PW	Brief, early diastolic reversal	Intermediate	Prominent holodiastolic reversal
Quantitative parameters*			
VC width, cm[§]	< 0.3	0.3-0.60	> 0.6
Jet width/LVOT width, %[§]	< 25	25-45 46-64	≥ 65
Jet CSA/LVOT CSA, %[§]	< 5	5-20 21-59	≥ 60
R Vol, ml/beat	< 30	30-44 45-59	≥ 60
RF, %	< 30	30-39 40-49	≥ 50
EROA, cm^2	< 0.10	0.10-0.19 0.20-0.29	≥ 0.30

J Am Soc Echocardiogr 2003;16:777-802

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AR: treatment

- Treat HTN with dihydroperidine CCB, or ACEi/ARB
- Afterload reducers remains controversial if normotensive

Treat Marfan syndrome or dilated root with BB and/or ARB

- AVR surgery for symptomatic severe AR or LV dysfunction
- AVR surgery for LV dysfunction: EF<50% or LVESD >50mm
- Aortic root surgery if >55mm
  >50mm in bicuspid AV
  >45-50mm in Marfan

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Recommendations	COR	LOE	References
AVR is indicated for symptomatic patients with severe AR regardless of LV systolic function (stage D)	I	B	(33, 92, 93)
AVR is indicated for asymptomatic patients with chronic severe AR and LV systolic dysfunction (LVEF <50%) (stage C2)	I	B	(92, 94-96)
AVR is indicated for patients with severe AR (stage C or D) while undergoing cardiac surgery for other indications	I	C	N/A
AVR is reasonable for asymptomatic patients with severe AR with normal LV systolic function (LVEF ≥50%) but with severe LV dilation (LVESD >50 mm, stage C2)	IIa	B	(97-99)
AVR is reasonable in patients with moderate AR (stage B) who are undergoing other cardiac surgery	IIa	C	N/A
AVR may be considered for asymptomatic patients with severe AR and normal LV systolic function (LVEF ≥50%, stage C1) but with progressive severe LV dilation (LVEDD >65 mm) if surgical risk is low*	IIb	C	N/A

*Particularly in the setting of progressive LV enlargement.

AR indicates aortic regurgitation; AVR, aortic valve replacement; COR, Class of Recommendation; LOE, Level of Evidence; LV, left ventricular; LVEDD, left ventricular end-diastolic dimension; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension; and N/A, not applicable.

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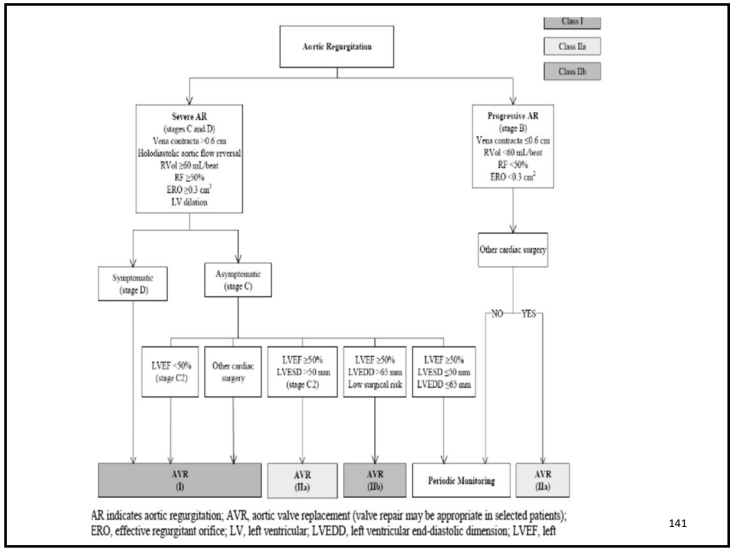

AR indicates aortic regurgitation; AVR, aortic valve replacement (valve repair may be appropriate in selected patients); ERO, effective regurgitant orifice; LV, left ventricular; LVEDD, left ventricular end-diastolic dimension; LVEF, left

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Mitral Regurgitation

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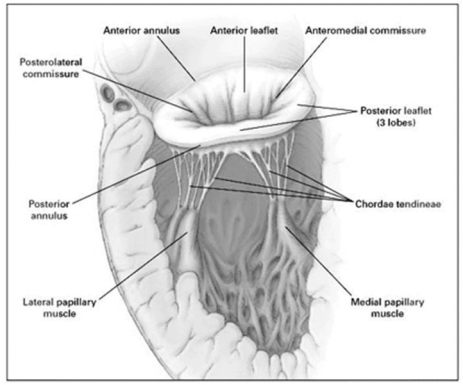
Mitral Valve



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Mitral Regurgitation (MR)

- Failure of mitral valve to prevent movement of blood from left ventricle to left atrium during ventricular systole.
- Can be classified into **Acute MR or chronic MR**
- Also, Can be classified into **Primary MR or secondary MR**

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Acute sever MR

- Can be due to flail leaflet, papillary muscle rapture, perforation
- Poorly tolerated due to:
  - **poor forward LV flow**, lack of LV adaptation
  - LA size is normal and pressure will rise abruptly

  (**short murmur**) and lead to **acute pulmonary edema**
- Usually requires urgent surgery

145

Chronic MR: pathogenesis

- In chronic MR this will lead to **dilated LV/LA** with

  increase in preload and

  decrease afterload
- Initially ejection fraction will increase but overtime it will drop if unrepaired.

146

MR: etiology

1. Degenerative (primary)
   - Primary lesions of the **mitral leaflets**
     - MVP, degenerative
     - Rheumatic heart disease
   - primary lesions of the **subvalvular apparatus**
     - **Posterior papillary muscle dysfunction** more common (IWMI or PWMI)
2. Functional(secondary)
   - annular or left ventricular dilation

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MR: etiolog

Common Causes of Chronic Mitral Regurgitation and Their Mechanisms

Etiology	Affected Valve Level(s)
Chronic Coronary artery disease Ischemic MR	LV, papillary muscles, annulus
Myxomatous degeneration Mitral valve prolapse Barlow's valve	Leaflets, chordae, annulus
Nonischemic dilated cardiomyopathy	LV, papillary muscles, annulus
Rheumatic heart disease	Leaflets, chordae
Healed infective endocarditis	Leaflets, chordae
Hypertrophic obstructive cardiomyopathy	AML, papillary muscles, LV
Mitral annular calcification	Annulus, leaflets
Congenital	Anterior leaflet
Connective tissue diseases Rheumatoid arthritis Systemic lupus erythematosus Antiphospholipid antibody syndrome	Leaflets, annulus
Radiation	Leaflets, chordae
Drug Ergotamines Methysergide Pergolide (fenfluramine, dexfenfluramine)	Leaflets, chordae

AML=anterior mitral leaflet; LV=left ventricular; MR=mitral regurgitation.

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Chronic MR: symptoms and signs

- Exertional dyspnea
- Progressive fatigue over many years
- Overtime: LV dysfunction and CHF/pulmonary edema/dyspnea
- Increased risk of atrial fibrillation, pulmonary HTN
- Exam: **pansystolic murmur at the apex radiating to axilla or base**
- Exam: Hyperdynamic apex,S3
- Exam**: MVP MR murmur radiate opposite to the affected leaflet**

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MR: Echocardiography

- Qualitative
  - 2D imaging of valve anatomy: MVP, flail leaflet, rheumatic, vegetation
  - Annular Calcification, annular dilatation, subvalvular apparatus
  - Left ventricular function and size: critical in timing surgery
  - LA/RA size, RV size and unction, PA pressure estimation
  - TEE: better imaging and quantification: critical if repair is planned
- Quantitative
  - Assess severity: quantify regurgitant volume/fraction and orifice

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Grade	RVol, mL	RF, %	ERO, mm^2
1	<30	<30	<20
2	30-44	30-39	20-29
3	45-59	40-49	30-39
4	≥60	≥50	≥40

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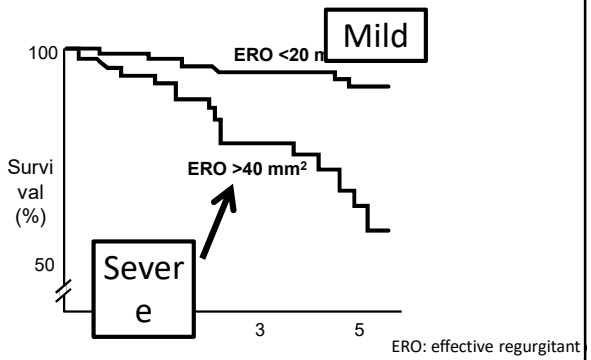
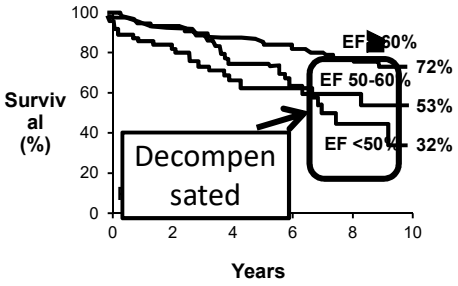
Mitral regurgitation



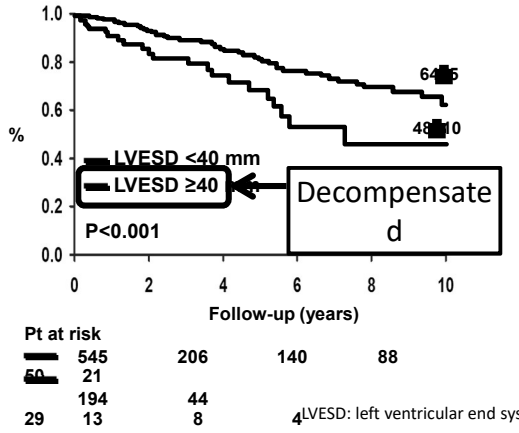
Enriquez-Sarano, M. et al. N Engl J Med 2005;352:875-883

Mitral Regurgitation
Preop EF vs Postop Survival



Enriquez-Sarano, M. et al.

Mitral Regurgitation
Preop LVESD vs Postop Survival



LVESD: left ventricular end systolic diame

MR treatment

- For chronic degenerative MR, no pharmacological agents have been shown to slow progression toward surgical intervention.
- For patients with CHF and MR we start them on the standard HF treatments:
  - Afterload reducers: ACEi/ARBs
  - BB
  - diuretics for fluid overload

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MR: surgery

- Repair is preferred if feasible over replacement(MVR) due to
  - preserve LV function
  - lower rates of complications than prosthetic valves
  - lower operative mortality rate
- No surgery is indicated(in general) for MR due to annular dilatation(secondary MR)
- Surgery indicated for:

1. Severe symptomatic MR
2. Severe asymptomatic MR with EF<60% or LVESD >4cm
3. MVP: severe asymptomatic MR if early repair can be performed

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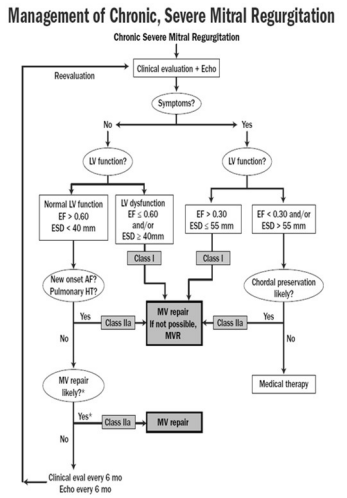
Chronic severe MR management



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MR treatment

Recommendations	COR	LOE	References
MV surgery is recommended for symptomatic patients with chronic severe primary MR (stage D) and LVEF >30%	I	B	(156, 179)
MV surgery is recommended for asymptomatic patients with chronic severe primary MR and LV dysfunction (LVEF 30%–60% and/or LVESD ≥40 mm, stage C2)	I	B	(150-153, 180-182)
MV repair is recommended in preference to MVR when surgical treatment is indicated for patients with chronic severe primary MR limited to the posterior leaflet	I	B	(155, 183-198)
MV repair is recommended in preference to MVR when surgical treatment is indicated for patients with chronic severe primary MR involving the anterior leaflet or both leaflets when a successful and durable repair can be accomplished	I	B	(195-197, 199-203)
Concomitant MV repair or replacement is indicated in patients with chronic severe primary MR undergoing cardiac surgery for other indications	I	B	(204)

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MVP

- Mid-systolic click
- Late systolic murmur or sometimes pansystolic(depends on degree MR)
- Murmur increases with standing and valsalva
- Often associated with marfanoid features(check aortic root), more in females
- Present with chest pain, palpitation(use BB) or heart murmur
- Confirm diagnosis with echocardiography
- May lead to significant MR
- Surgery for severe asymptomatic MR if early repair can be performed
- No need for endocarditis prophylaxis

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MITRAL STENOSIS

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Epidemiology of Mitral Stenosis

- Usually caused by rheumatic fever
  - Rare in developed countries today
  - Other causes: anorectic drugs, carcinoid syndrome, congenital, mitral annular calcification
- More common in women than men (2-3:1)
- Rheumatic fever:
  - Group A beta-hemolytic streptococcus has M-protein, also found in myocardium
  - Leads to autoimmune response causing inflammation of the endocardium, myocardium, and pericardium
  - Chronic inflammation and scarring of endocardium, leading to commisural/chordae fusion and retraction, calcification
  - May involve all cardiac valves; mitral valve most common (40% can have isolated MS)

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MS: symptoms

- Long latency period
- Exertional dyspnea
- Orthopnea
- PND
- Worse symptoms with atrial fibrillation or pregnancy
- Usually LV size and function are preserved !

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Natural history of MS

- Defined from studies in 1950's and 1960's
- Continuous, progressive, lifelong disease
- Developed countries
  - Latent period 20-40 years after rheumatic fever
  - 10 years for symptoms to become disabling
- Asymptomatic: >80% at 10 years
- Limiting symptoms: 0-15% at 10 years
  - Pulmonary hypertension: mean survival < 3 years
- Death caused by:
  - Pulmonary and systemic congestion: 60-70%
  - Systemic embolism: 20-30%
  - Pulmonary embolism: 10%
  - Infection 1-5%

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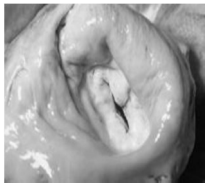
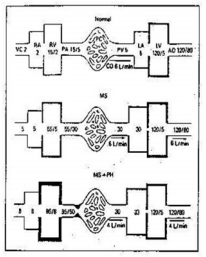
Pathophysiology of Mitral Stenosis

- LA and LV pressure gradient
  - Leads to elevated LA pressures → LA enlargement and pulmonary congestion/edema
  - Restricted flow → decreased cardiac output
- Pulmonary hypertension
  - Pulmonary arteriolar vasoconstriction, medial hypertrophy, intimal hyperplasia
  - Protective mechanism from pulmonary edema
- RV generates force to drive blood across MV and pulmonary bed, causing RV pressure overload
  - Eventually dilates and fails





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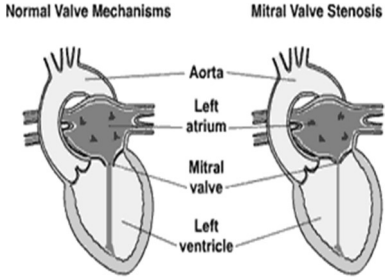
Pathophysiology of MS



- Symptoms generally do not occur until MVA <2.5 cm^2
- MVA > 1.5cm^2 usually does not cause rest symptoms (precipitated by exercise, pregnancy, atrial fibrillation, etc.)

165

Mitral Stenosis: Physical Exam

- First heart sound (S1) is accentuated and snapping
- Opening snap (OS) after aortic valve closure
- Low pitch diastolic rumble at the apex
- Pre-systolic accentuation (esp. if in sinus rhythm)

166

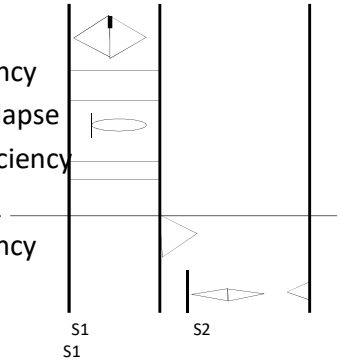
Common Murmurs and Timing

<u>Systolic Murmurs</u>

- Aortic stenosis
- Mitral insufficiency
- Mitral valve prolapse
- Tricuspid insufficiency

<u>Diastolic Murmurs</u>

- Aortic insufficiency
- Mitral stenosis



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Auscultation- Timing of A2 to OS Interval

- Width of A2-OS inversely correlates with severity
- The more severe the MS the higher the LAP the earlirthe LV pressure falls below LAP and the MV opens

Say	*Timing seconds*	*Severity of MS*	*Other HS's*
Prrr	< 0.06	Severe	
Pada	.07-.08	Mod-severe	
Pata	.08-.09	Mod	
Papa	> 0.10	Mild	PK 0.1-0.110
Tu-huh	≥ .12		A2-S3 0.12-0.18

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2D characteristics of MS

- Thickened and calcified leaflets and subvalvular apparatus
- “Hockey-stick” appearance of AML in diastole
- Immobility of PML
- “Fish-mouth” orifice in short-axis view
- Increased LA size
- Mitral valve area calculation by pressure ½ time, continuity equation
- Mitral valve gradient

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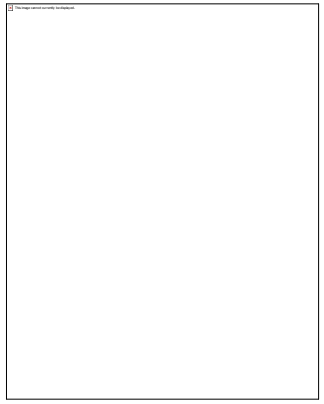
MS: Hockey-stick



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MS: Fish-mouth

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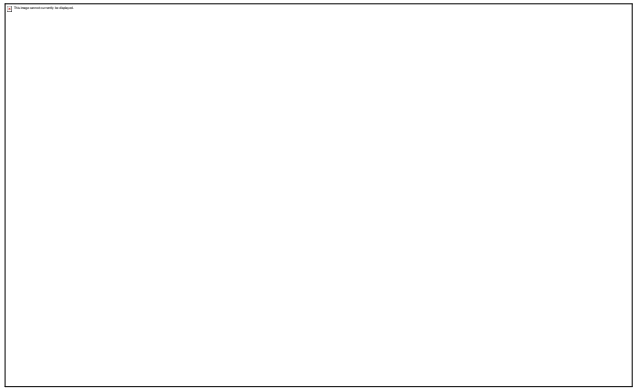
Stages of MS:



172

MS: management

- Medical therapy: limited
  - Diuretics for mild symptoms
  - Atrial fibrillation – often presents in RVR, impairing LV filling (abrupt LA hypertension and decreased CO)
    - Rate controlling agents (b-blockers, CCB, Digoxin!)
    - Anticoagulation with warfarin even if paroxysmal
    - annual Embolic CVA risk 7-15%
    - New agents are not approved
  - Warfarin also for LA thrombus or history emboli event
  - BB or CCB for tachycardia !?
- Serial testing – change in symptoms/exam findings
  - Annual echocardiogram for severe MS
- Definitive therapy with mechanical relief of obstruction by surgery or balloon valvuloplasty

173

MS: surgical indications

- Percutaneous mitral balloon commissurotomy(PMBC) is recommended: for symptomatic severe MS (MVA<1.5cm2) and favorable morphology and no LA thrombus and no significant MR
- Mitral valve surgery is indicated for severe MS with NYHA III-IV who are not candidate for PMBC
- Mitral valve surgery is indicated for severe MS if pt undergoing cardiac surgery for other cause.

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MS

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Tricuspid Stenosis

- Causes: Rheumatic mostly, Carcinoid syndrome, post repair or replacement
- More common in females
- Frequently with tricuspid regurgitation
- Symptoms: right HF: ascites/hepatomegaly/LLE
- Exam: prominent a wave in JVP, diastolic rumble in left parasternal
- Diagnosis: echocardiography: severe TS if mean gradient >5mmHg
- Treatment: right HF medications, refractory cases needs surgery with usually tissue TV replacement

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Tricuspid regurgitation

- Mostly due to RV dilatation, rarely due to primary valve problem
- RV dilation due to left heart failure(most common), pulmonary HTN, others such as RV dysplasia, sarcoidosis
- Primary valve problem: Ebstein anomaly, TV prolapse, carcinoid, endocarditis
- Ebstein anomaly: displacement of the septal leaflet into the RV
- Symptoms: RV failure
- Exam: pansystolic mumur, Right sided S3
- DX: echocardiography
- Treatment: HF treatment, underlying cause, rarely surgery is needed

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Pulmonary Regurgitation

- Causes: divided into high pressure and low pressure causes
- High pressure due to pulmonary HTN(most common)
- Low pressure due to: dilated annulus, bicuspid PV, dysplastic PV, carcinoid disease, s/p surgical repair in tetralogy of Fallot
- Mostly well tolerated and asymptomatic
- Treat the underlying cause especially in the high pressure PR
- Tetralogy of Fallot: RV enlargement/QRS>180ms with severe PR will need PV replacement

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Pulmonic valve stenosis

- Stenosis of the pulmonary valve is a relatively common congenital defect, occurring in approximately 10 percent of children with congenital heart disease.
- A trileaflet valve with varying degrees of fibrous thickening and fusion of the commissures.
- A small proportion (Noonan syndrome) have a markedly dysplastic valve associated with severe stenosis.
- Pulmonary arterial stenosis often occurs in association with other cardiac or noncardiac congenital defects. These include **tetralogy of Fallot** and the **congenital rubella syndrome** as well as rarer disorders such as **Williams syndrome, Noonan syndrome**
- Symptoms of pulmonic stenosis with severe obstruction include exertional dyspnea, fatigue, syncope, and chest pain.
- The systolic ejection murmur of pulmonic stenosis becomes later peaking and louder as the stenosis worsens, assuming preserved right ventricular function.
- Balloon valvotomy is preferred unless dysplastic valve(surgery), for severe PS: peak gradient >60mmHg in asymptomatic or >50mmHg in symptomatic

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Antibiotic prophylaxis

- Antimicrobial prophylaxis for patients with:
  - the highest risk medical conditions
  - undergoing procedures likely to result in bacteremia

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Highest risk conditions

1. Prosthetic heart valves, including bioprosthetic and homograft valves
2. Prior history of IE
3. Unrepaired cyanotic congenital heart disease, including palliative shunts and conduits
4. Completely repaired congenital heart defects with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first six months after the procedure
5. Repaired congenital heart disease with residual defects at the site or adjacent to the site of the prosthetic patch or prosthetic device
6. Valve regurgitation due to a structurally abnormal valve in a transplanted heart

181

Highest risk procedures

1. Dental procedures that involve manipulation of either gingival tissue or the periapical region of teeth or perforation of the oral mucosa; this includes routine dental cleaning.
2. Procedures of the respiratory tract that involve incision or biopsy of the respiratory mucosa
3. Gastrointestinal (GI) or genitourinary (GU) procedures in patients with ongoing GI or GU tract infection
4. Procedures on infected skin, skin structure, or musculoskeletal tissue
5. Surgery to place prosthetic heart valves or prosthetic intravascular or intracardiac materials

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Notes:

- Antimicrobial prophylaxis is warranted for tonsillectomy, adenoidectomy, or bronchoscopy with biopsy
- Antimicrobial prophylaxis is not warranted for any GI or GU procedure.
- Antimicrobial prophylaxis is not warranted for vaginal or cesarean delivery.
- Patients with skin or musculoskeletal infections undergoing procedures should receive antimicrobial therapy with activity against staphylococci and beta-hemolytic streptococci.

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Infective Endocarditis Prophylaxis

Description	Drug	Adult Dose
Standard general prophylaxis	Amoxicillin	2 g PO
Unable to take oral medications	Ampicillin	2 g IV/IM
Allergic to penicillin	Clindamycin	600 mg PO
	Cephalexin or other 1st/2nd generation oral cephalosporin in equivalent does	2 g PO
	Azithromycin or clarithromycin	500 mg PO
Allergic to penicillins & unable to take oral medications	Clindamycin	600 mg IV
	Cefazolin or ceftriaxone	1 g IV/IM

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Prosthetic valves

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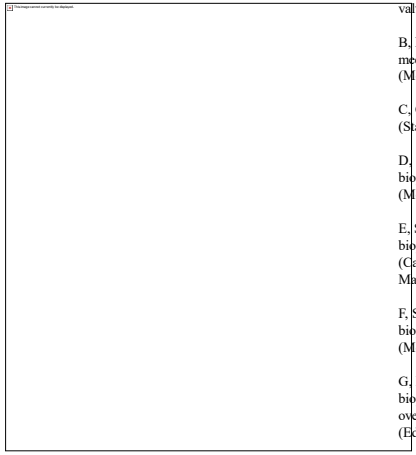

A, Bileaflet mechanical valve (St Jude)

B, Monoleaflet mechanical valve (Medtronic Hall)

C, Caged ball valve (Starr-Edwards)

D, Stented porcine bioprosthesis (Medtronic Mosaic)

E, Stented pericardial bioprosthesis (Carpentier-Edwards Magna)

F, Stentless porcine bioprosthesis (Medtronic Freestyle)

G, Percutaneous bioprosthesis expanded over a balloon (Edwards Sapien)

186

Prosthetic Heart Valves

- Antithrombotic therapy with warfarin and aspirin is recommended in patients with mechanical prosthetic heart valves to prevent valve thrombosis and thromboembolic events.
- Goal INR vary with the type of valve, the site of valve replacement (the risk is greater with mitral than aortic valves), and the presence or absence of thrombotic risk factors.
- Baseline echocardiogram is recommended at first postoperative follow-up visit six weeks to three months after prosthetic valve implantation.
- Antibiotic prophylaxis is recommended
- Regular moderate exercise to maintain cardiovascular fitness is encouraged in patients with prosthetic valves.
- Recommendations for competitive sports vary depending upon valve type and need for anticoagulation. All patients treated with anticoagulation should not participate in sports associated with risk of bodily contact or danger of trauma.
- Pregnancy in women with prosthetic heart valves is complicated by the need for antithrombotic therapy!?!

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Bradycardia

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First degree atrioventricular (AV) nodal block

- Prolonged PR interval (>0.20 seconds)
- Occurs when there is a prolongation or delay in impulse conduction through the AV node (most common) or His-Purkinje system

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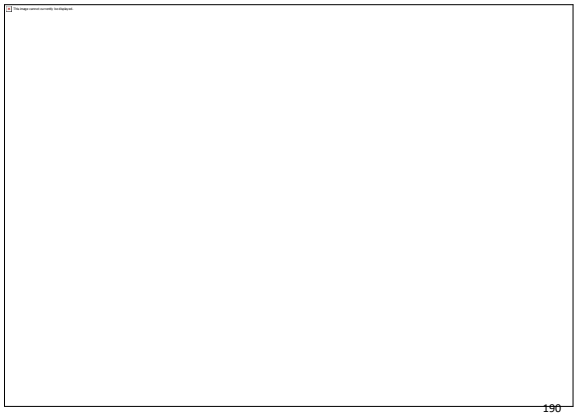

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Second degree AV block

- Defined as an occasional long RR interval resulting from an occasional non-conducted P wave, may either be **Mobitz type I or type II:**
- **Mobitz type I (Wenkebach)** second degree AV block is usually a result of progressive slowing of AV conduction through the AV node with subsequent failure to conduct one atrial impulse from the atria to the ventricles
  - This is manifest as progressive PR prolongation before the dropped beat.
  - There is only one non-conducted P wave.
- **Mobitz type II** second degree AV block
  - underlying disease of the His-Purkinje conduction system
  - episodic and unpredictable failure of the node to conduct the impulse (or more than one impulse)
  - The PR interval does not change prior to or after the dropped beats.
  - There may be more than one non-conducted P wave.
- **2:1 AV block** is identified by the fact that every other P wave is non-conducted. This may be either **Mobitz I or Mobitz II.**

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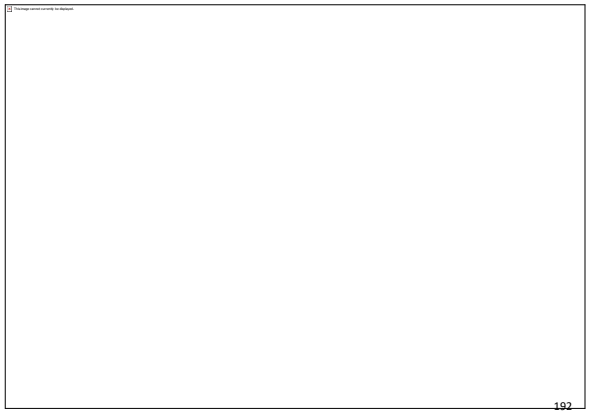

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Third degree or complete AV block

- complete failure of the AV node to conduct any impulses from the atria to the ventricles, resulting in an escape rhythm.
- There is AV dissociation (ie, variable PR intervals) and the atrial rate is faster than the ventricular rate.
- The escape rhythm may be either junctional or ventricular and this is based on the QRS complex morphology and not the rate of the escape rhythm.

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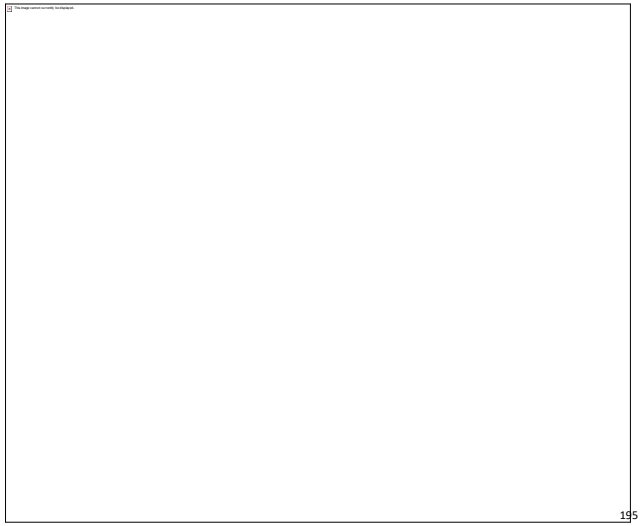


195

Thank you
Questions

196

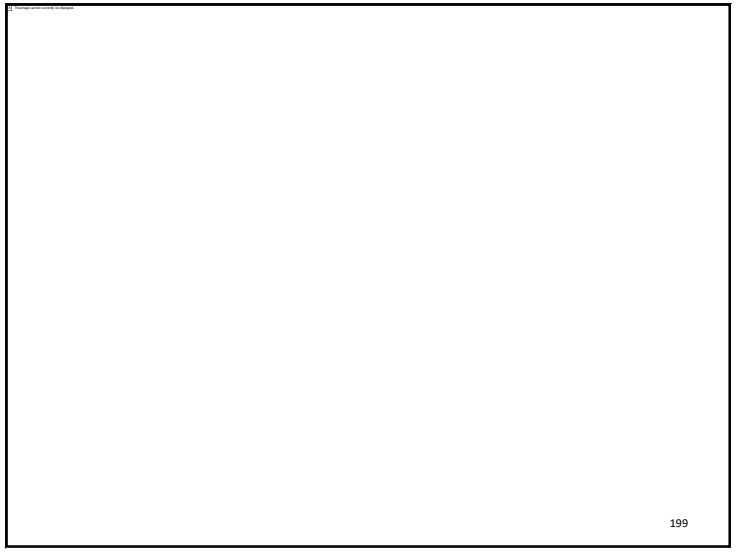
Pericarditis
Fourth year medical students

Tareq Yousef Goussous, M.D., FACC
Interventional Cardiologist

197

- A 32-year-old-male patient presented to your office for an ECG as ordered by his insurance company.

198



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- A 46-year-old-male patient with a pmhs of hypertension and smoking presented to the ED with chest pain.

200

201

- A 25-year old male pt. presented with worsening dyspnea to the ED. His heart rate was 140 bpm and BP 80/40 mm Hg.

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- **Introduction:**
- The pericardium is a fibroelastic sac made up of visceral and parietal layers separated by a space, the pericardial cavity.
- In healthy individuals, the pericardial cavity contains 15-50 mL of an ultrafiltrate of plasma.

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- Acute pericarditis refers to inflammation of the pericardial sac.
- The term myopericarditis, or perimyocarditis, is used for cases of acute pericarditis that also demonstrate myocardial inflammation.

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- Most cases of acute pericarditis are considered of possible or confirmed viral origin, although the exact etiology of most cases remains undetermined following a traditional diagnostic approach.
- Acute pericarditis is a common disorder in several clinical settings, where it may be the first manifestation of an underlying systemic disease or may represent an isolated process.

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- **Clinical presentation:**
- Acute pericarditis can present in a variety of ways, depending on the underlying etiology.
- Patients with an infectious etiology may present with signs and symptoms of systemic infection such as fever and leukocytosis.
- Viral etiologies in particular may be preceded by “flu-like” respiratory or gastrointestinal symptoms.

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- Patients with a known autoimmune disorder or malignancy may present with signs or symptoms specific to their underlying disorder.
- The major clinical manifestations of acute pericarditis include:

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- Chest pain — typically sharp and pleuritic, improved by sitting up and leaning forward.
- Pericardial friction rub — a superficial scratchy or squeaking sound best heard with the diaphragm of the stethoscope over the left sternal border.
- Electrocardiogram (ECG) changes — new widespread ST elevation or PR depression
- Pericardial effusion.

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- **I-Chest pain:**
- The vast majority of patients with acute pericarditis present with chest pain (>95% of cases).
- Chest pain that results from acute pericarditis is typically fairly sudden in onset and occurs over the anterior chest.
- Unlike pain from myocardial ischemia, chest pain due to pericarditis is most often sharp and pleuritic in nature, ***with exacerbation by inspiration or coughing.***

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- One of the most distinct features is the **tendency for a decrease in intensity when the patient sits up and leans forward.**
- This position (seated, leaning forward) tends to reduce pressure on the parietal pericardium, particularly with inspiration, and may also allow for splinting of the diaphragm.

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- **II-Pericardial friction rub:**
- The presence of a pericardial friction rub on physical examination is highly specific for acute pericarditis.
- Pericardial friction rubs, which occur during the maximal movement of the heart within its pericardial sac, are said to be generated by friction between the two inflamed layers of the pericardium.

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- Pericardial rubs have a superficial scratchy or squeaking quality that is best heard with the **diaphragm** of the stethoscope.
- They may be localized or widespread, but are usually loudest over the left sternal border.

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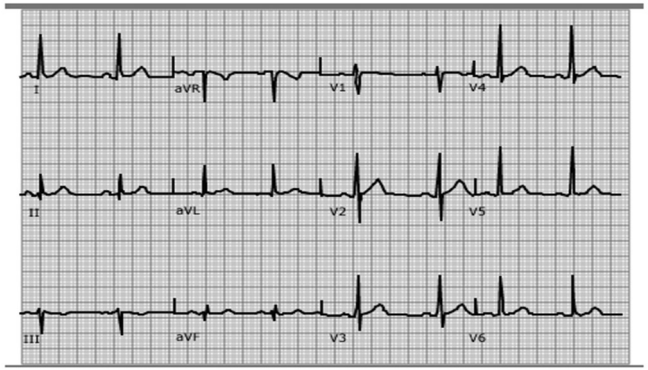
Normal ECG



Normal sinus rhythm at a rate of 71 beats/min, a P wave axis of 45°, and a PR interval of 0.15 sec.
Courtesy of Morton Arnsdorf, MD.

216

- **III-Electrocardiogram:**
- Changes in the electrocardiogram (ECG) in patients with acute pericarditis signify inflammation of the epicardium, since the parietal pericardium itself is electrically inert.

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- The typical progression of ECG changes in patients with acute pericarditis is described below:
- Stage 1, seen in the **first hours to days,** is characterized by **diffuse ST elevation** (typically concave up) with **reciprocal ST depression in leads aVR and V1.**
- There is also an atrial current of injury, reflected by **elevation of the PR segment in lead aVR and depression of the PR segment in other leads.**

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- Stage 2, typically seen in the first week, is characterized by normalization of the ST and PR segments.
- Stage 3, is characterized by the development of diffuse T wave inversions, generally **after** the ST segments have become isoelectric.
- Stage 4, is represented by normalization of the ECG or indefinite persistence of T wave inversions ("chronic" pericarditis).

222

- Echocardiography is often normal in patients with the clinical syndrome of acute pericarditis unless there is an associated pericardial effusion.
- Chest radiography is typically normal in patients with acute pericarditis. Although patients with a substantial pericardial effusion may exhibit an enlarged cardiac silhouette with clear lung fields .

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- Acute pericarditis may be associated with increases in serum biomarkers of myocardial injury such as cardiac troponin or MB fraction of creatine kinase.
- Since pericarditis is an inflammatory disease, laboratory signs of inflammation are common in patients with acute pericarditis.
- These include elevations in the white blood cell count, erythrocyte sedimentation rate, and serum C-reactive protein concentration.

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- **Treatment:**
- In cases of pericarditis due to an identifiable cause (eg, bacterial infection or malignancy), management is focused upon the underlying disorder and, if necessary, drainage of an associated pericardial effusion.

230

- For most patients with acute idiopathic or viral pericarditis, combination therapy with colchicine plus NSAIDs rather than NSAIDs alone.
- This is based upon a reduced rate of recurrent pericarditis and a low incidence of side effects with colchicine.

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- In patients with acute pericarditis following an MI, aspirin plus colchicine rather than another NSAID plus colchicine
- This is principally due to the possibility that other NSAIDs may interfere with healing and scar formation.
- Although the evidence of potential harm from glucocorticoids and NSAIDs other than aspirin is modest, there is no evidence that these medications improve outcomes.

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- F***or these reasons glucocorticoids and NSAIDs other than aspirin should generally be <u>AVOIDED</u> in patients with acute pericarditis following an acute MI.***

233

- Among patients with acute pericarditis, initial management with systemic glucocorticoid therapy should be restricted to patients with pericarditis due to connective tissue disease, autoreactive (immune-mediated) pericarditis, uremic pericarditis not responding to dialysis, and to patients who have contraindications to NSAID therapy.
- Glucocorticoid therapy is also used for patients with idiopathic or viral pericarditis that is refractory to combination therapy with NSAIDs and colchicine.

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Tareq Yousef Goussous, M.D., FACC
Interventional Cardiologist

Bacterial Endocarditis

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- A 35-year old female patient with PMHx of atrial septal defect (ASD) called you to ask you if she needs antibiotics before her root canal next week?
- Ventricular septal defect (VSD).
- Bioprosthetic (tissue) valve.
- Patent ductus arteriosus (PDA).

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Outline

- Definition.
- Predisposing factors.
- Clinical manifestations.
- Physical exam.
- Diagnosis & Rx.
- Prophylaxis.

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Definition

- Infection of endothelium of heart (including but not limited to the valves).
- Acute Bacterial Endocarditis (ABE):

  Infection of normal valves with virulent organism (eg, *S.aureus*, group A or other β-hemolytic strep, *Strep pneumo*)
- Subacute (SBE):

  Indolent infection with less virulent organism (eg, S.viridans); often abnormal valves.

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Predisposing conditions

- Abnormal valve:

  High risk: prior endocarditis, rheumatic valvular disease, AoV disease (including bicuspid), complex cyanotic lesions, prosthesis (annual risk 0.3-1%)

  Medium risk: Mitral valve disease (including MVP with MR or leaflet thickening), Hypertrophic cardiomyopathy (HCMP)

- Abnormal risk of bacteremia : IDU, indwelling venous catheters, poor dentition, hemodialysis, DM, intracardiac devices (eg, pacemakers, ICD)

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Table 1 – Modified Duke Criteria

Major	Minor
•**Sustained bacteremia** by an organism known to cause endocarditis (or 1 BCx or +ve serology for *Coxiella*) •**Endocardial involvement** document by either +ve **echocardiogram** (vegetation, abscess, prosthetic dehiscence) *Or* **new valvular regurgitation**	• Predisposing condition • Fever • **Vascular phenomena:** septic arterial or pulmonary emboli, mycotic aneurysms, ICH, Janeway lesions • **Immune phenomena:** +ve RF, GN, Osler's nodes, Roth spots • +ve **BCx** not meeting major criteria
Definitive (ie, highly probable): 2 major or 1 major+ 3 minor or 5 minor criteria **Possible: 1 major + 1 minor or 3 minor criteria**	

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Table 2 – Microbiology of Endocarditis

	Native valve endocarditis (NVE)		**Prosthetic valve endocarditis .(PVE)**	
Etiology	**Non-IVDA**	**IVDA**	**Early**(≤60 d post)	**Late** (>60 d post)
S. viridans et al.	**36%**	**13%**	<5%	**20%**
Enterococcus	**11%**	5%	8%	13%
S. aureus	**28%**	**68%**	**36%**	**20%**
S. epidermidis	**9%**	<5%	**17%**	**20%**
GNR	<5%	**<5%**	**6%**	<5%
Other	<5%	<5%	10%	10%
Fungal	1%	1%	9%	3%
Culture -ve	11%	<5%	17%	12%

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Clinical manifestations

- Persistent bacteremia**:** fever (80-90%), chills, night sweats, anorexia, weight loss, fatigue.
- Valvular or perivalvular infection: CHF, conduction abnormalities
- Septic emboli: systemic emboli (eg, to periphery, CNS, kidneys, spleen or joints), stroke, PE (if right-sided), mycotic aneurysm, MI (coronary artery embolism).
- Immune complex phenomena**:** arthritis, glomerulonephritis, +ve RF, ↑ ESR.
- SBE : can p/w fatigue , nonspecific sx in patients w/o risk factors ; need high index of suspicion.

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Physical exam

- HEENT : Roth spots (retinal hemorrhage + pale center), petechiae (conjunctiva, palate)
- Cardiac: murmur (85%), new valve regurgitation (40-85%) ± thrill (fenestrated valve or ruptured chordae), muffled sounds (PV). Frequent exams for murmurs , s/s CHF.
- Abdomen: tender splenomegaly; musculoskeletal: arthritis, vertebral tenderness

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Physical exam - Continued

- Extremeties (typically seen in SBE, not ABE)
  - **Janeway lesions** (septic emboli → nontender, hemorrhagic macules on palms or soles).
  - **Osler's nodes** (immune complexes→ tender nodules on pads of digits).
- Neuro: change in MS or focal deficits.
- Devices: erythyma, tenderness or drainage at catheter site

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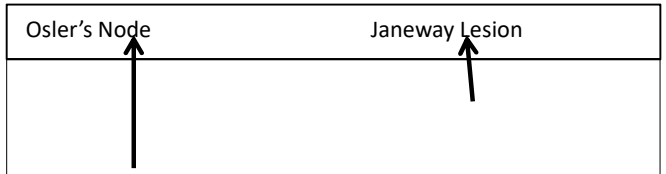

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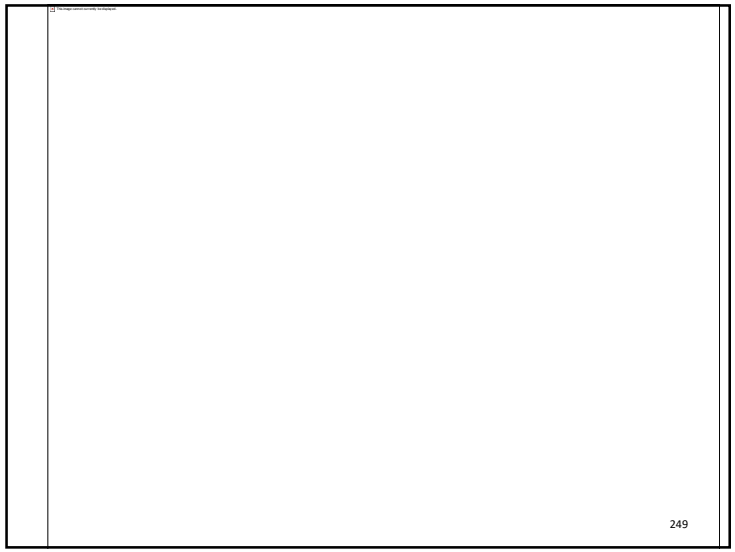
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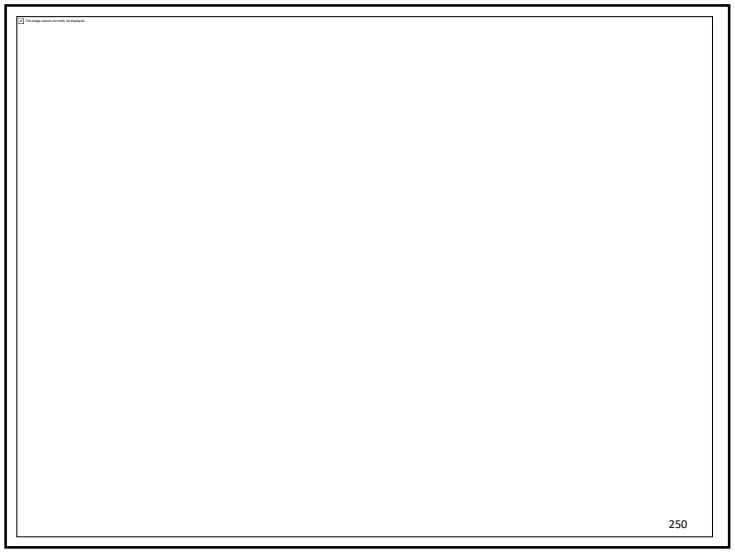
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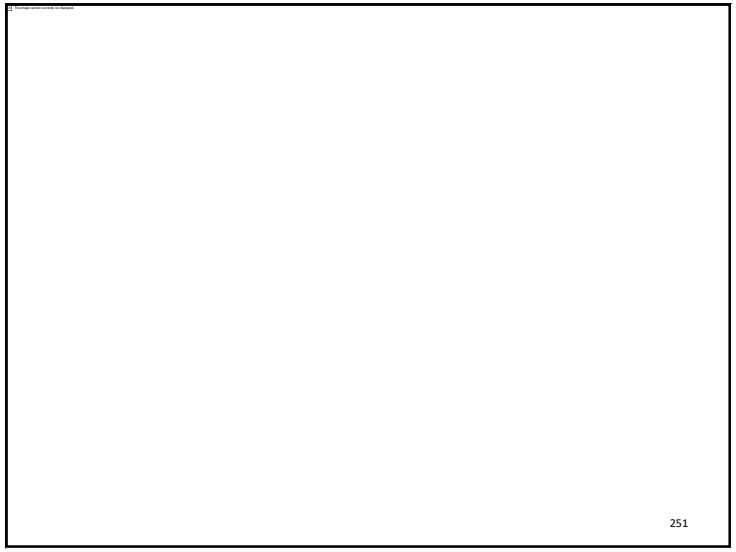
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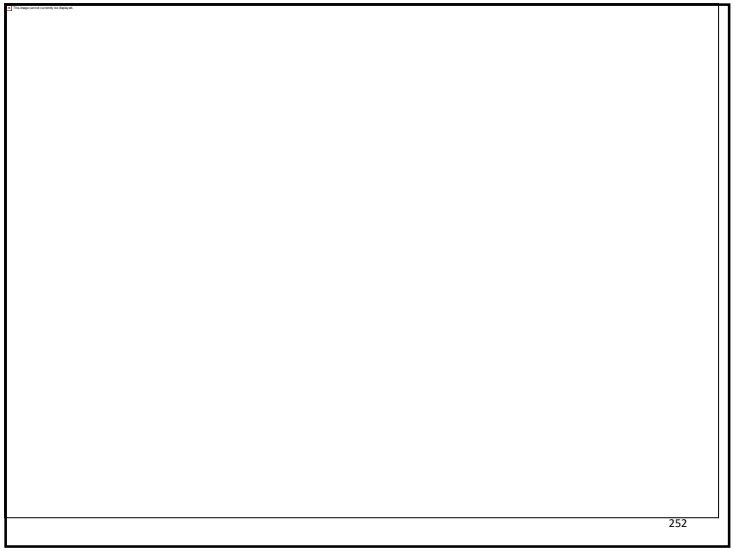
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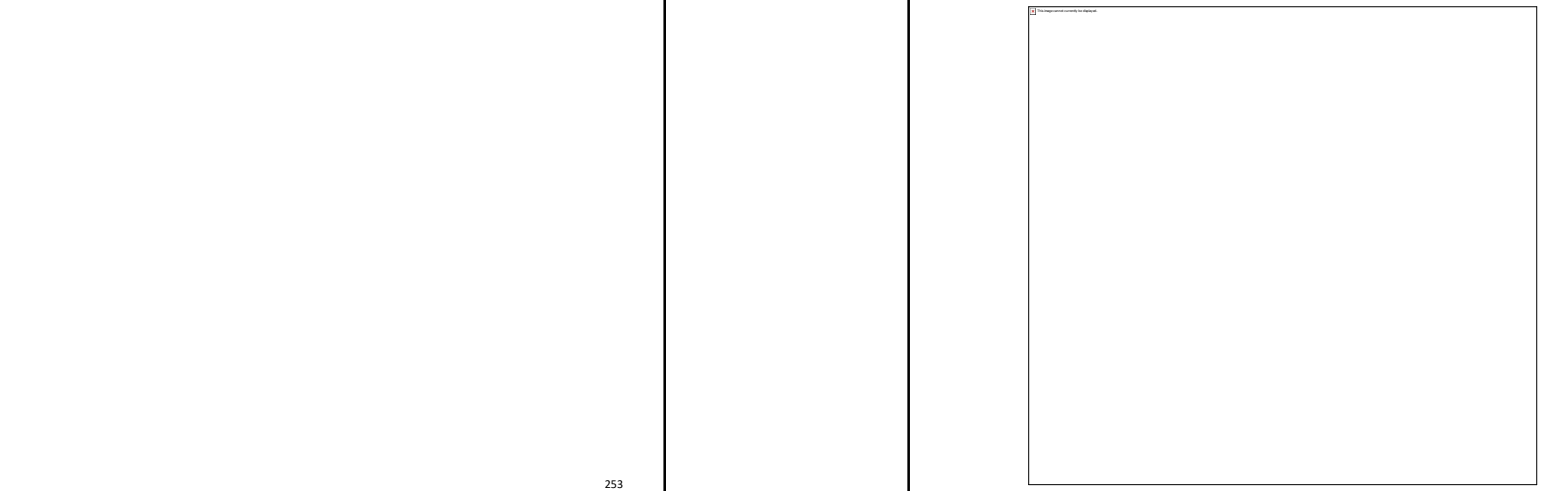

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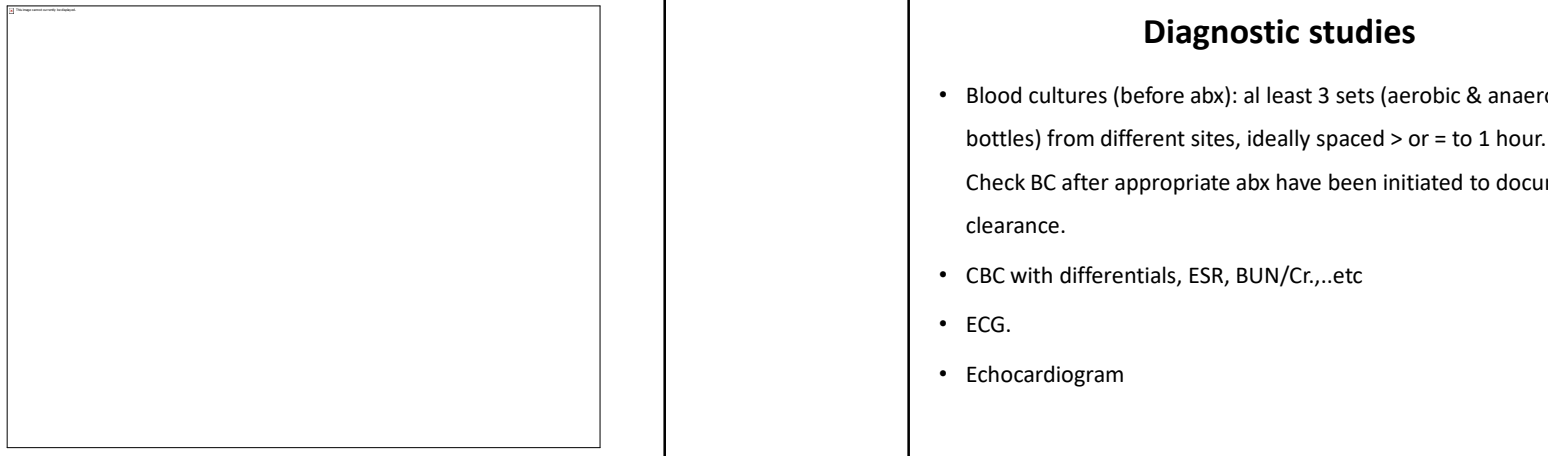

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Diagnostic studies

- Blood cultures (before abx): al least 3 sets (aerobic & anaerobic bottles) from different sites, ideally spaced > or = to 1 hour. Check BC after appropriate abx have been initiated to document clearance.
- CBC with differentials, ESR, BUN/Cr.,..etc
- ECG.
- Echocardiogram

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Treatment

- Obtain culture data first.
- Suggested empiric therapy:
  - Native valve ABE: vancomycin
  - Native valve SBE: ceftriaxone +/- gentamycin
  - PVE:
    - Early (<60 days): vanco + gent + cefepime
    - Intermediate (60-365 days): vanco + gent.
    - Late (>1y): vanco + CTX + gent.)

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Indications for surgery

1) Severe vavular dysfunction→ refractory CHF.
2) Uncontrolled infection.
3) Organism.
4) Systemic embolism.
5) PVE

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Table 3 – Endocarditis prophylaxis

Cardiac conditions*	**Prosthetic valve; previous NVE; congenital heart disease**(CHD) including unrepaired or incompletely repaired cyanotic CHD (palliative shunts or conduits), 1st 6 mo after completely repaired CHD using prosthetic material; **cardiac transplant recipients w/ valvulopathy** (prophylaxis no longer rec. in acquired valvular dysfxn, bicuspid AoV, MVP with leaflet thickening or regurgitation, HCMP)
Procedures*	**Dental:** manipulation of gingival tissue or periapical region of teeth or perforation of oral mucosa (eg, extraction, periodontal procedures, implants, root canal, cleanings) **Respiratory:** incision or biopsy of respiratory mucosa (prophylaxis no longer rec. for GI or GU procedures)
Regimens	Oral : amoxicillin 2 g 30-60 min before Unable to take PO: amp 2 g IM/IV or cefazolin or Cftx 1 gm IM/IV PCN-allergic : clinda 600 mg PO/IM/IV

* Pts should meet both indications (high risk condition and high risk procedure) to qualify for prophylaxis

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THE END

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