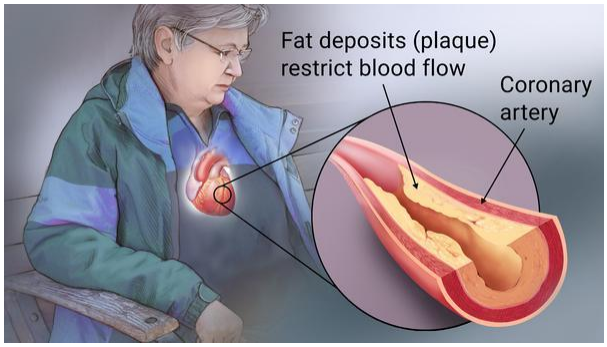


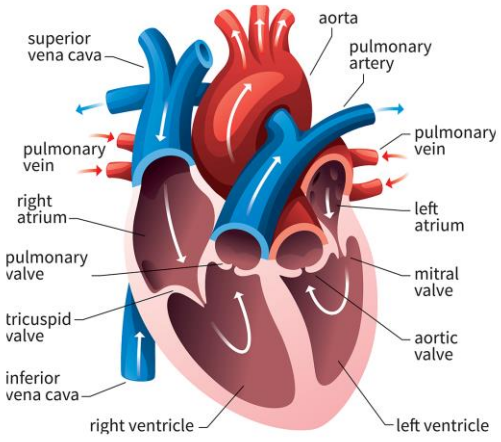
Chapter 16

Ischemic Heart Disease and Conduction Disorders

1

Heart

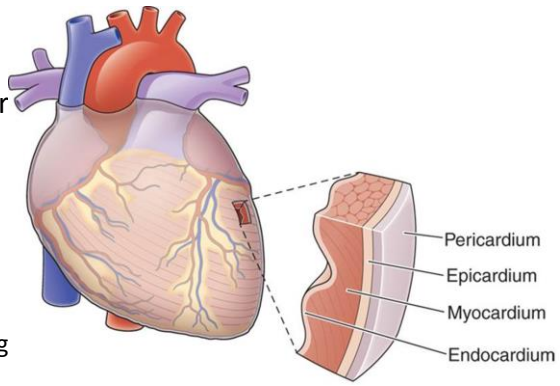
- Size of fist
  - Located in mediastinum
  - Base: upper part
  - Apex: pointed end
- Acute coronary syndrome (ACS)
  - Due to ischemia
- Myocardial infarction (MI)
  - Prolonged ischemia leading to cell death



2

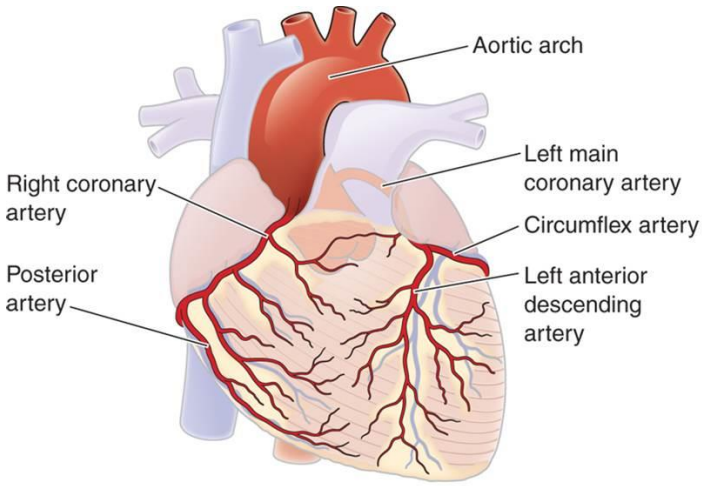
Heart Structure

- Tissue layers
  - Epicardium: outer
  - Myocardium: muscle
  - Endocardium: lines interior
- Coronary circulation
  - Right coronary artery
  - Left coronary artery
    - LAD: left anterior descending
  - Exercise
    - Increases coronary collateral vessels



3

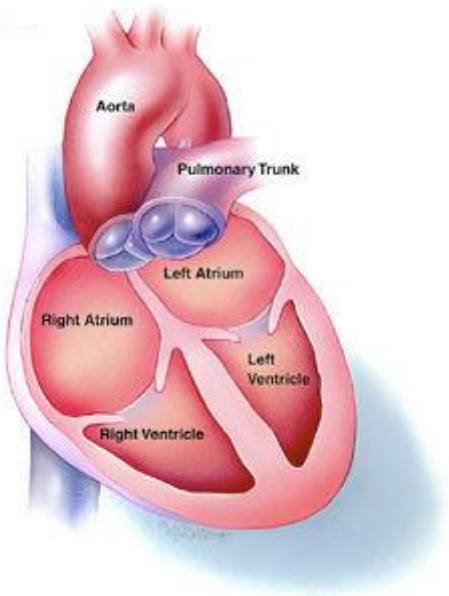
Coronary Arteries



4

Heart Overview

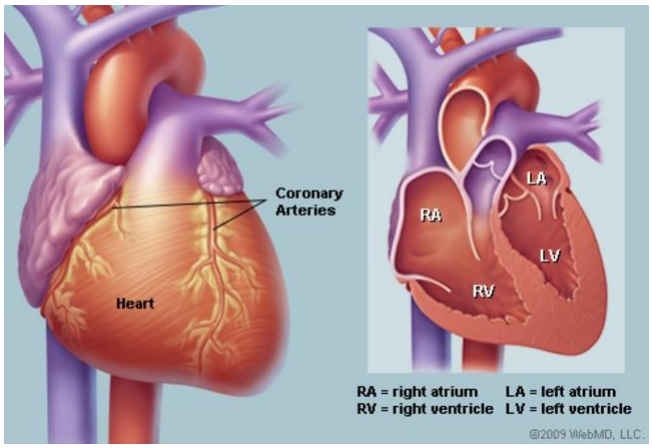
- 4 chambers
  - Right atrium
    - Receives blood from superior an inferior vena cava and coronary sinus
  - Right ventricle
    - Pumps blood to lungs
  - Left atrium
    - Receives blood from pulmonary veins
  - Left ventricle
    - Pumps blood to body



5

Circulation Overview

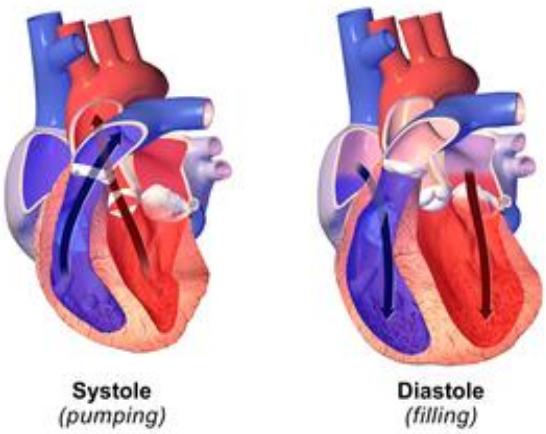
- Two circuits
  - Pulmonary
    - Low pressure system, RV to LA
    - Picks up oxygen at lungs
  - Systemic
    - High pressure system, LV to RA
    - Delivers oxygen to body tissues



6

Circulation Overview (continued)

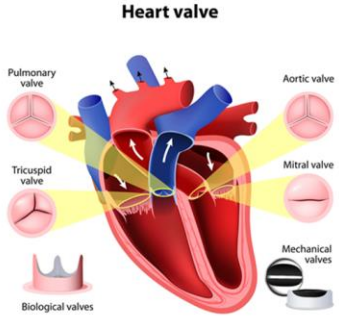
- Systolic
  - Contraction
  - SBP: systolic blood pressure
- Diastolic
  - Relaxation
  - DBP: diastolic blood pressure

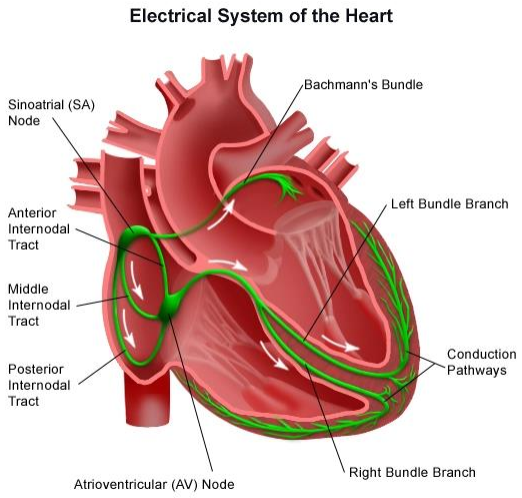


7

Conduction System Overview

- Sinoatrial node (SA node)
  - Pacemaker
- AV node
- Bundle of His
- Bundle branches
- Purkinje fibers





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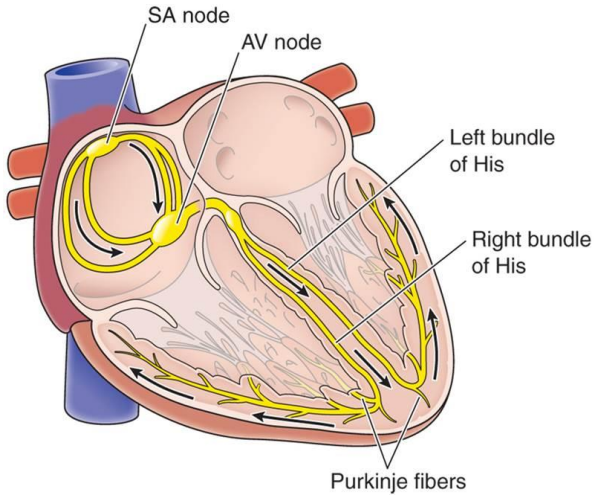
Conduction System



9

The "PQRST"

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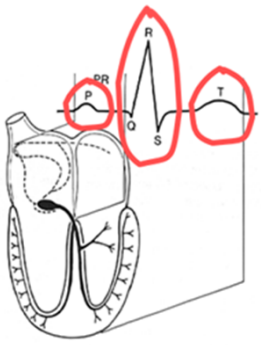


- P wave - Atrial depolarization
- QRS - Ventricular depolarization
- T wave - Ventricular repolarization

10

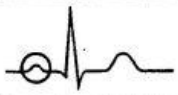
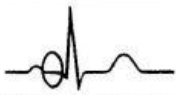
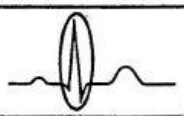
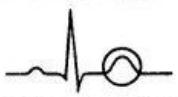
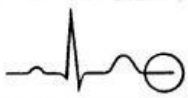
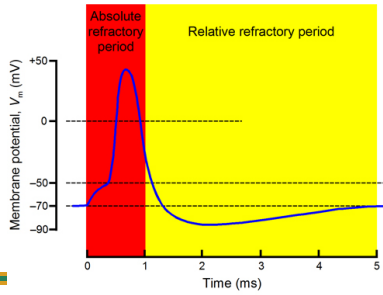
Electrical Activity	Graphic Depiction	Associated Pattern
Atrial Depolarization		P Wave
Delay at AV Node		PR Segment
Ventricular Depolarization		QRS Complex
Ventricular Repolarization		T Wave
No electrical activity		Isoelectric Line

Figure 2-5. Electrocardiogram wave patterns produced by electrical activity in the heart.

11

Refractory Period

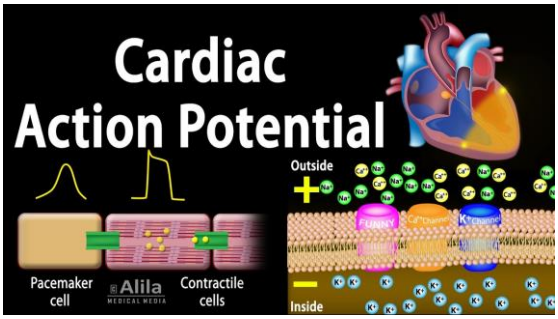
- The membrane of cardiac muscle has 3 types of **ion channels (channels of Na+, K+, Ca+)** that allow the voltage (electrical) changes during the phases of the "**action potential**".
- The movements of the ion channels (transportation of electrical signals) make the heart to contract and relax. This is called "**action potential**".
- **Absolute refractory period** = when no stimuli can generate another; i.e., cells can not be re-stimulated; cells cannot depolarize again. ➔ this means that another contraction cannot occur during this time
  - Phases 0, 1, 2, part of 3
- This "Absolute refractory period"
  - Enables heart to relax
  - Enables heart to fill
  - Protects against fatal arrhythmias



12

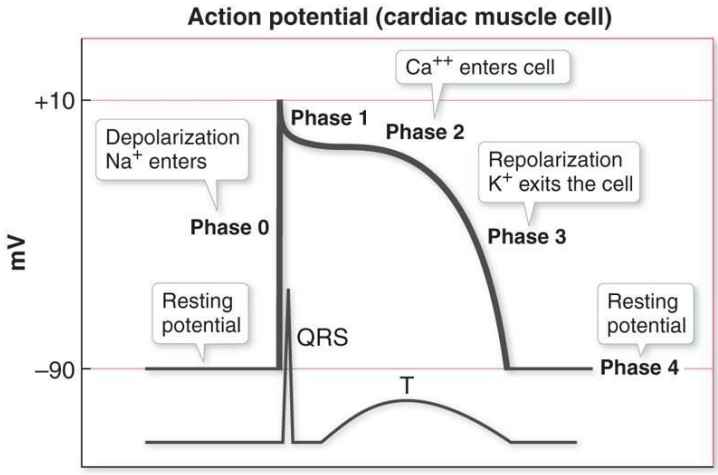
Cardiac Muscle Action Potential

- 5 phase action potential with 3 types of channels:
  - Phase 0: fast sodium channels open and increase potential from -90 mV to +20 mV
  - Phase 1: peak, abrupt closure fast sodium channels
  - Phase 2: plateau, slow sodium-calcium channels
  - Phase 3: repolarization, K+ channels open
  - Phase 4: resting potential, Na+/K+ pump



13

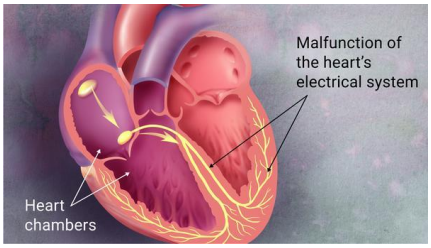
Cardiac Cell Action Potential



14

Dysrhythmias (Arrhythmias)

- Disruption in electrical signal
  - Supraventricular
    - SA node, atria, AV node
  - Ventricular
    - Bundle of His, bundles branches, Purkinje fibers, and ventricle muscle
  - Tachyarrhythmias and bradyarrhythmias
- Block
  - Most commonly at AV node
- Ectopic pacemaker
  - Outside normal conduction pathway
    - *Example:* PVC (premature ventricular contraction)



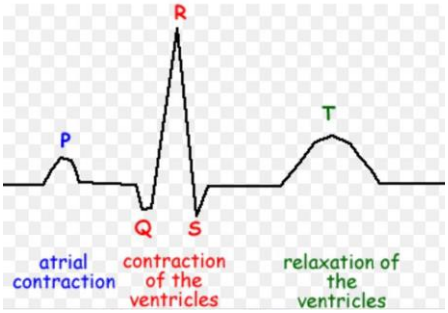
15

Electrocardiogram

Electrical activity of the heart

- X-axis: time
- Y-axis: millivolts

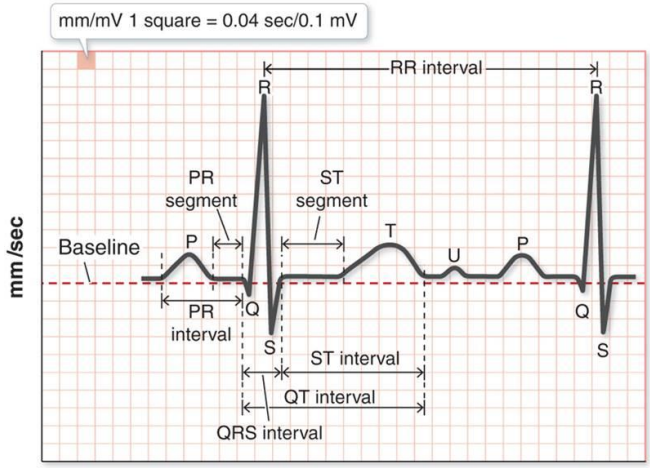
- Standard waveforms
  - P wave: atrial depolarization
  - QRS complex: ventricular depolarization
  - T wave: ventricular repolarization



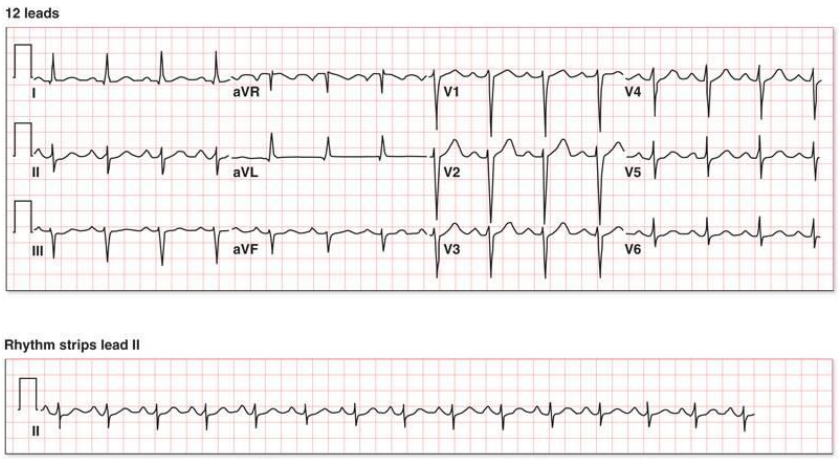
- 12-Lead ECG ➔ Provides 12 different views of electrical activity of the heart

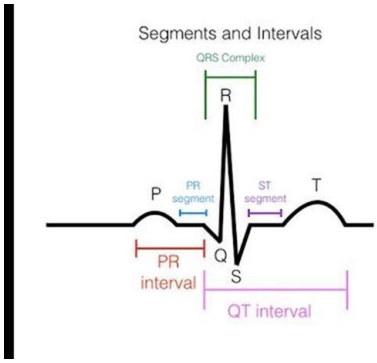
16

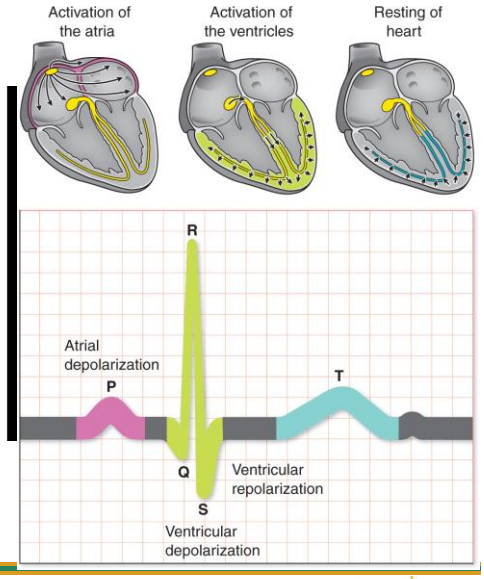
Electrocardiogram (continued)



12-Lead Electrocardiogram (ECG)



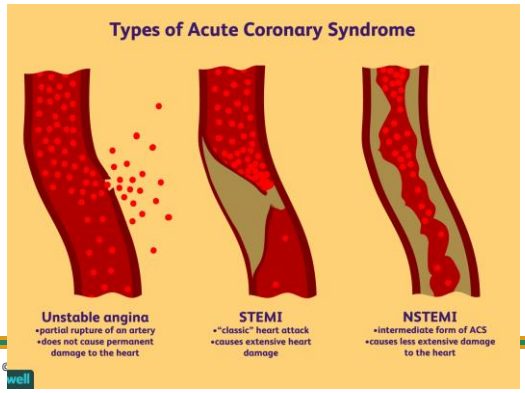


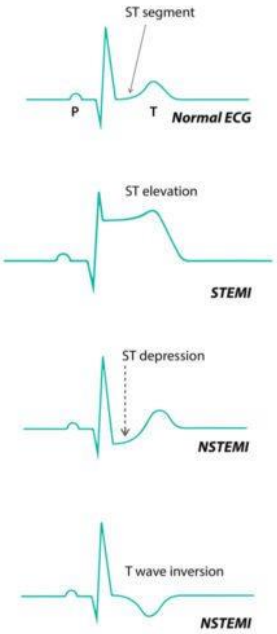


19

Acute Coronary Syndrome (ACS)

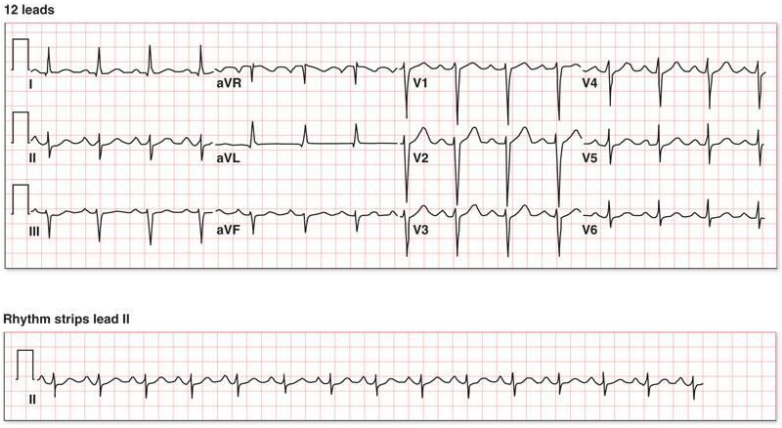
- Two types
  - **Unstable angina**
  - **Myocardial infarction (MI)**
    - STEMI: ST segment elevation MI
    - NSTEMI: Non-ST segment elevation MI





20

STEMI

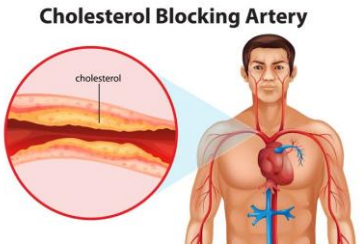


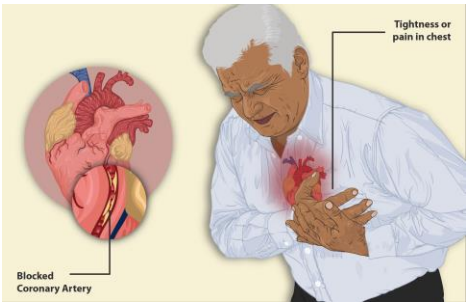
Angina

- Angina pectoris
  - Symptom: Chest pain

Two types:

- Stable
  - Consistent, chronic pain
  - Treatment: Self-medication with nitroglycerin
- Unstable
  - New episode or increased severity
  - New regions of heart experiencing ischemia
  - Medical emergency

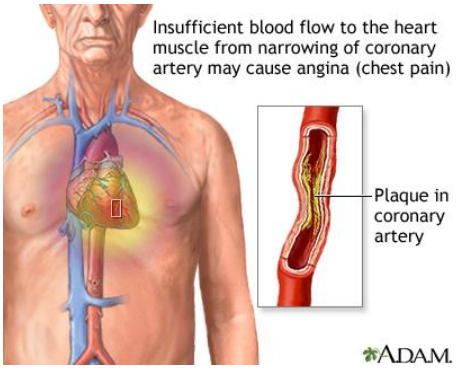




Anginal Equivalents

Signs of MI that differ from classic angina:

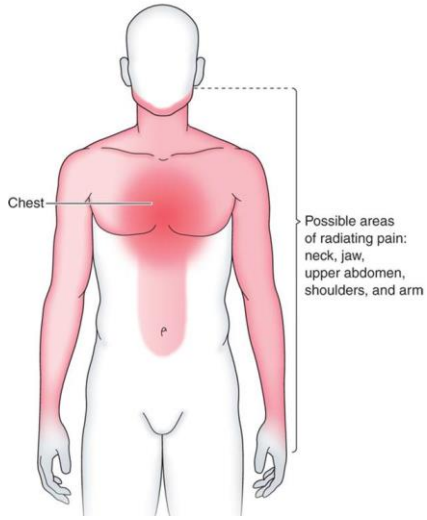
- Angina may present more commonly in women
- Symptoms: Dyspnea, feeling faint, epigastric pain, dizziness, extreme fatigue
- As signs differ from classic angina, MIs may be missed in women
- Studies show women treated less aggressively for possible MI than men



Angina Pectoris

- Myocardial ischemia: causes
  - Blood clot (coronary thrombosis)
  - Atherosclerotic plaque
  - Coronary artery vasospasm (Prinzmetal's or variant angina)
- Ischemia causes cellular hypoxia → myocardial cells undergo anaerobic metabolism = 2 ATP) + lactic acid accumulation → resulting in pain

Angina Pectoris (continued)



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Angina and Coronary Artery Disease (CAD)

- Risk factors
  - Cigarette smoking, diabetes mellitus, hypercholesterolemia, metabolic syndrome
- Signs and symptoms
  - Chest pain brought on by exertion; crushing sensation on left side of chest; pain radiating to shoulder, jaw, and down the arm
  - Levine's sign: clenched fist over sternum
- Pain does not change with alterations in position, respiration, or cough

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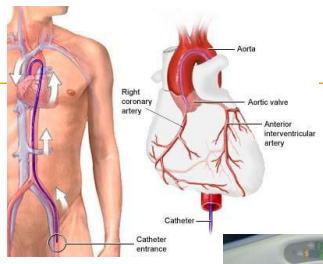
Angina and CAD Diagnosis

- Blood lipids
- Blood pressure
- ECG: ST elevation
  - Not confirmatory test of MI
- hs-CRP (increased risk of CAD)
- Cardiac enzymes
- Cardiac troponin (cTn)
  - Confirmatory test of MI
- Chest x-ray
- Calcium computed tomography
- Cardiac angiogram
- Cardiac catheterization

Diagnosis



Intravascular coronary angiography

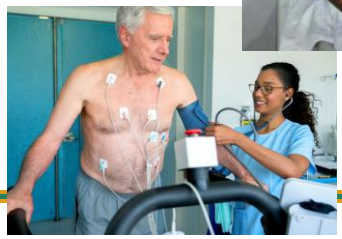
- Gold standard
- Risk of rupturing atherosclerotic plaques

Non-invasive coronary computed tomographic angiography (CCTA)

- Decreased risk of plaque rupture

Graded exercise stress test

- Echocardiogram or scintigraphy (thallium injection)
- Heart under stress, determine areas of injury



Treatment

- Overall
  - Relieve symptoms, reduced CAD progression, prevent MI
  - Education and modification of lifestyle risk factors
- Therapeutic interventions
  - Oxygen (if saturation is less than 95%)
  - Nitrates: vasodilators
  - Aspirin: decrease platelet adherence
  - Beta blockers: decrease myocardial oxygen demand
  - Calcium antagonists: arteriole dilation
  - ACE inhibitors: decrease BP and resistance

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Surgical and Endovascular Options

- Percutaneous transluminal coronary angioplasty (PCTA)
  - Uses catheter with balloon on tip
  - Angioplasty
    - Inflated balloon pushes plaque against arterial wall
  - Atherectomy
    - Catheter with blade reduces plaque
  - Stent
    - Maintains open vessel
    - Drug-eluting stents

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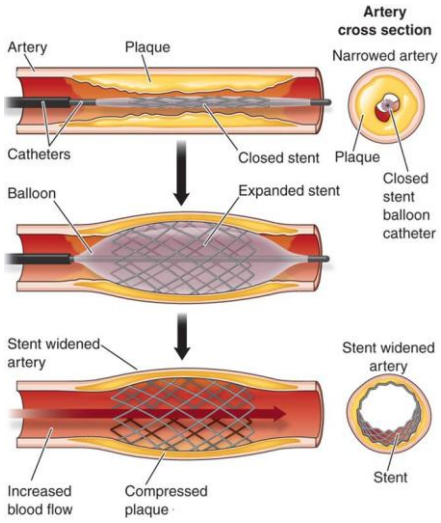
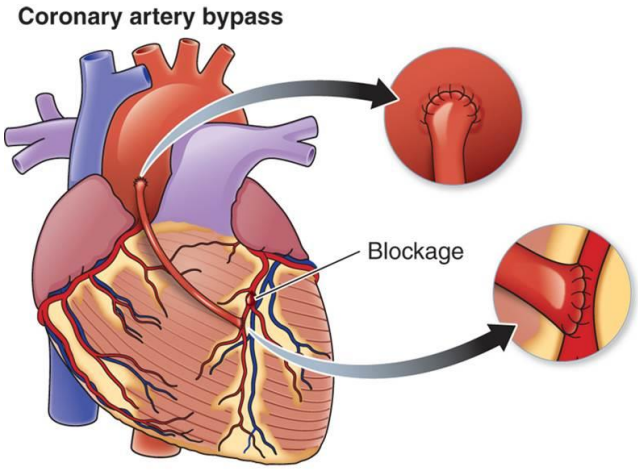
Percutaneous Transluminal Angioplasty (PCTA)



Surgical and Endovascular Options (continued)

- Coronary artery bypass graft (CABG)
  - Creates new routes around occlusions
  - Saphenous vein of leg often used
  - Triple, quadruple, or quintuple bypasses may be performed
  - Traditionally
    - Cardiopulmonary heart pump used
  - Currently
    - "Off pump" procedures

Coronary Artery Bypass Graft (CABG)



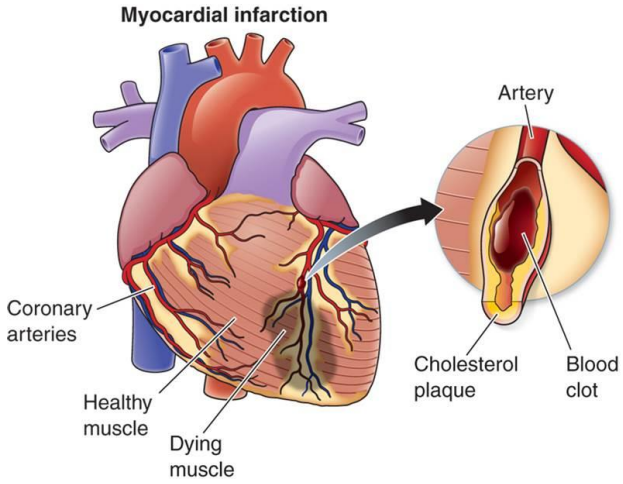
Myocardial Infarction (MI)

Prolonged ischemia leading to irreversible damage

- Blood flow obstruction
- Myocardial oxygen demand exceeds supply

- ~1.5 million MIs in U.S. per year
  - ~600,000 deaths
    - Delayed request for medical help plays a role
    - With prompt treatment, MI survival in U.S. is ~90%

Myocardial Infarction (MI) (continued)



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Classification of MI

Classification system still under discussion

- Type 1
  - Spontaneous due to plaque or thrombus
- Type 2
  - Lack of oxygen to meet demands
- Type 3
  - MI resulting in death with no biomarkers
- Type 4
  - MI due to stent insertion
- Type 5
  - MI due to CABG

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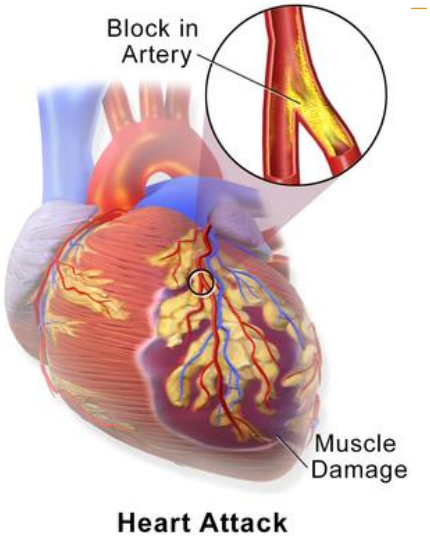
MI Damage

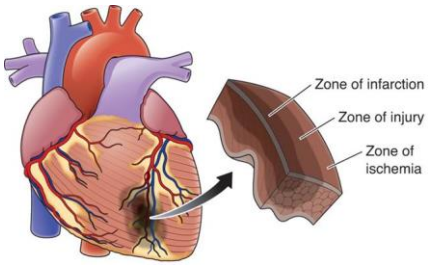
Depends on:

- Location
- Length of time
  - Greater than 30 minutes of ischemia causes irreversible damage
- Available collateral circulation



MI Damage (continued)

- Area of infarct surrounded by area of injury
  - Some cells in area of injury will recover
- Necrotic cells
  - Altered electrical activity (ECG changes)
  - Release cellular contents (cardiac markers such as troponin)



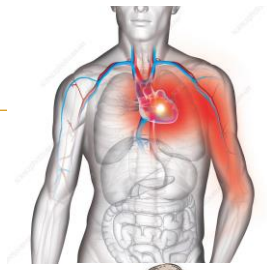
Reperfusion Injury

Ischemic myocardial tissue can be damaged by normalized levels of oxygen and nutrients

- Known as "*myocardial stunning*"
- → Reperfusion injury may be due to oxidized free radicals generated by WBCs
- Arrhythmias and reduced contractility may occur

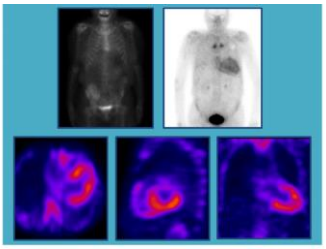
MI Signs and Symptoms

- Crushing chest pain
- Levine's sign
- Pain radiating to shoulder, jaw, down left arm
- Sweating, nausea, anxiety
- Anginal equivalents



MI Diagnosis

- ECG: cannot confirm MI
  - STEMI: infarction through cell wall
  - NSTEMI: infarction not through cell wall
- Blood test (cardiac markers)
  - CPK-MB: rise within 4 hours of MI, subside over 3–4 days
  - cTnI: rise 4–8 hours after chest pain onset, baseline within 5 to 9 days Elevated serum concentration of **cardiac troponin I** (cTnI) is a highly sensitive and specific marker of myocardial damage.
- Echocardiogram
- Radionuclide myocardial perfusion



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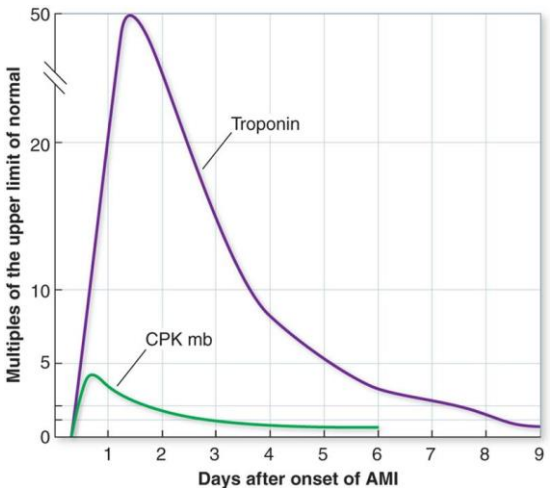
Cardiac Biomarkers



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MI Treatment

- Antiplatelet (aspirin), oxygen, nitrates, morphine
- Percutaneous coronary intervention (PCI)
- Coronary artery bypass graft (CABG)
- Thrombolytic agent: tPA (tissue plasminogen activator)
  - Concern for uncontrolled bleeding
- Anticoagulants

MI Treatment (continued)

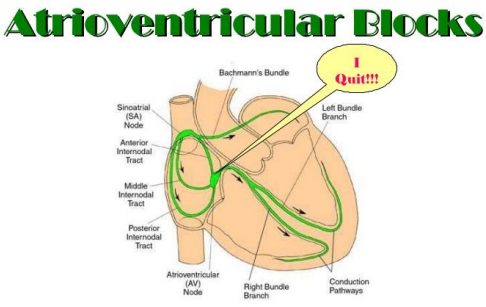
- Anti-platelet medications
  - Aspirin and P2Y12 receptor inhibitors
- Beta adrenergic blockers
  - Decrease HR, myocardial oxygen demand
- Calcium channel antagonists
  - Decrease coronary arteriole constriction

MI Complications

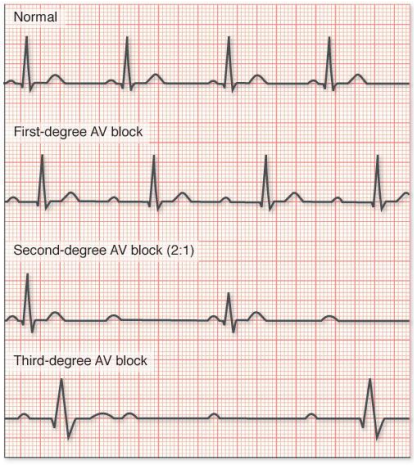
- Post-myocardial infarction dysrhythmias
- Risk greatest after onset of MI symptoms
- Reentry
  - Ischemic areas do not conduct impulses as expected
  - Impulses may “re-activate” already depolarized areas
  - Excitable activation sequence occurs, disrupting normal rhythm
  - Heart destabilized electrically

Atrioventricular Block

- Anterior and inferior wall MI
- SA atrial impulse fails to be conducted to the ventricles
- Prolonged PR interval

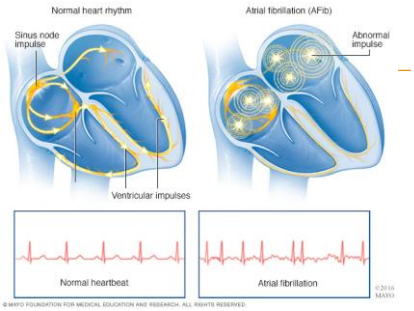


Delayed electrical impulses that originate from the SA node.



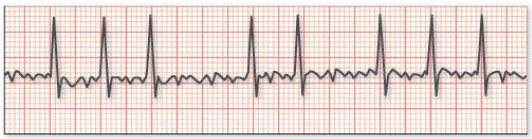
AV block rhythm disturbances

Atrial Fibrillation



Absence of coordinated, rhythmic atrial contractions

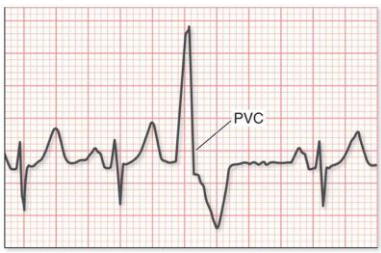
- Multiple irregular fibrillatory P waves
- May or may not stimulate a rapid ventricular response
- Increase the risk of clot formation and stroke



Atrial fibrillation

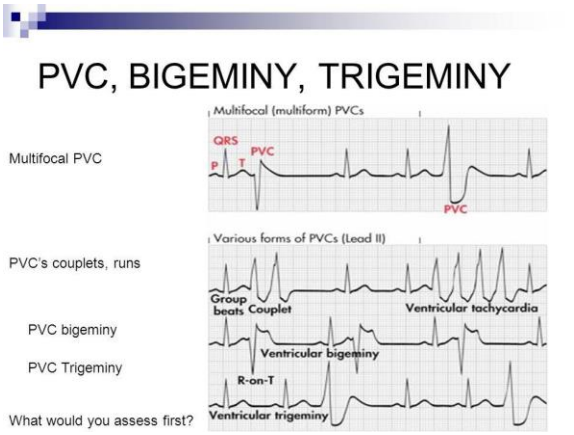
Premature Ventricular Contraction

- Sporadic PVCs most common arrhythmia post-MI
- Ventricle beats independently
- Widened QRS complex



Premature Ventricular Contraction (continued)

- May occur singularly or in patterns of bigeminy, trigeminy, quadrigeminy
- Sporadic, infrequent PVCs do not require treatment
- 2 sequential PVCs (couplets) may produce a dangerous ventricular rhythm



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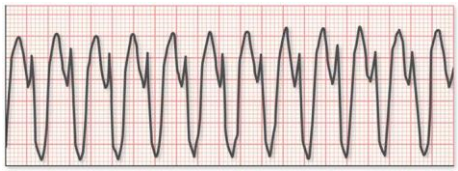
Ventricular Tachycardia

- Series of widened QRS waves without P or T waves
- Rapid, ineffective ventricular contractions
- Rate greater than 100 bpm
- Does not allow for effective pumping
- Dyspnea, palpitations, and lightheadedness



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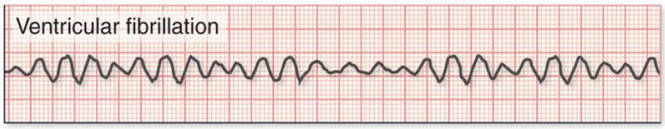
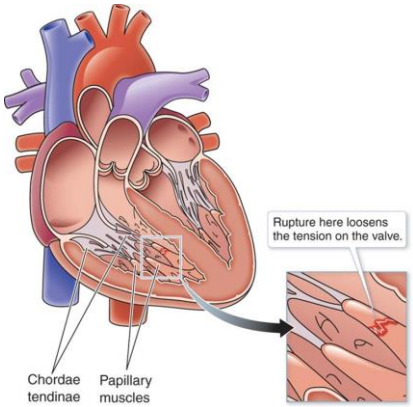
Ventricular Fibrillation

- May be precipitated by PVC falling on the T wave, interrupting refractory period
- Rhythm degenerates
- Fibrillation = quivering
- Contraction is uncoordinated, with no effective pumping
- Loss of consciousness, death may occur



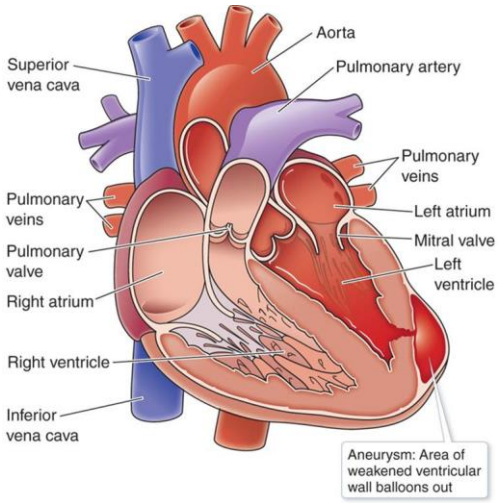
MI Complications (continued_1)

- Papillary muscle rupture
  - If in LV, mitral valve affected
    - Mitral insufficiency (regurgitant murmur)
    - Backup of blood into left atrium and pulmonary circulation
    - Dyspnea, crackles may develop
- Thromboembolism
  - Poorly contracting heart chambers cause stagnant blood, increasing risk for clot formation



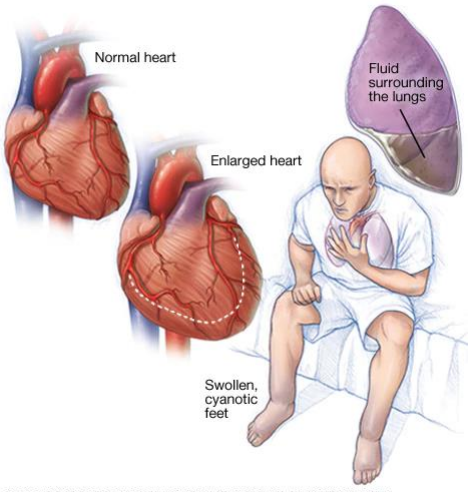
MI Complications (continued_2)

- Ventricular aneurysm and rupture
  - Weakened, bulging area of heart wall
  - May rupture, surgical intervention needed
- Pericarditis
  - Occurs 2–3 days post-MI
  - Pericardial friction rub, stabbing pain with inspiration
  - May occur as component of Dressler's syndrome
    - Hypersensitivity reaction to tissue necrosis in MI



MI Complications (continued_3)

- Heart failure
  - Ability to generate sufficient pressure is compromised
  - LV
    - Hypotension; fluid backs up in pulmonary circulation
  - RV
    - Fluid backs up in systemic circulation leading to jugular vein distention and peripheral edema



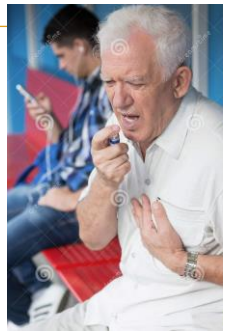
Prinzmetal's Angina

- variant or vasospastic angina
- Angina while at rest due to coronary artery vasospasm (i.e., unrelated to activity level)
- Unclear etiology: lack of nitric oxide?
  - Endothelial dysfunction due to plaques may play a role
- Not always associated with typical CAD risks

Prinzmetal's Angina (continued)

- Diagnosis usually requires 24-hour cardiac monitoring, coronary angiography, and serial cardiac enzymes
- Nitrates and calcium channel blockers may be used for treatment
- Acute MI is a possible complication of this form of angina

Chronic Coronary Artery Disease (Stable Angina)



Symptom:

- Recurrent chest pain
- Stable, consistent pain presentations

Pathophysiology: Transient myocardial ischemia

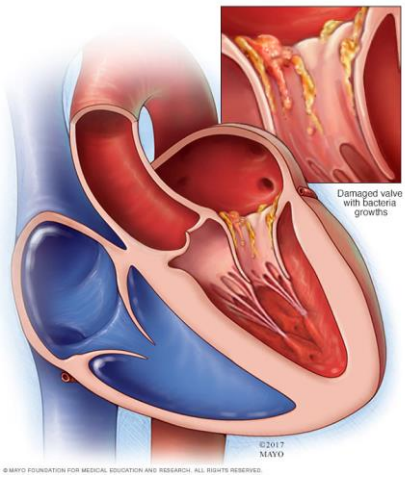
- At least one coronary artery 70% occluded

- Treatment: Self-medicate with nitrates
  - Essentially, **nitrates** dilate – that is, widen or relax – the arteries and the veins not only in the heart but also elsewhere in the body. By dilating the blood vessels of the heart, **nitrates** can reduce the stress on the heart by improving blood flow to the heart muscle. This will relieve **angina** symptoms.

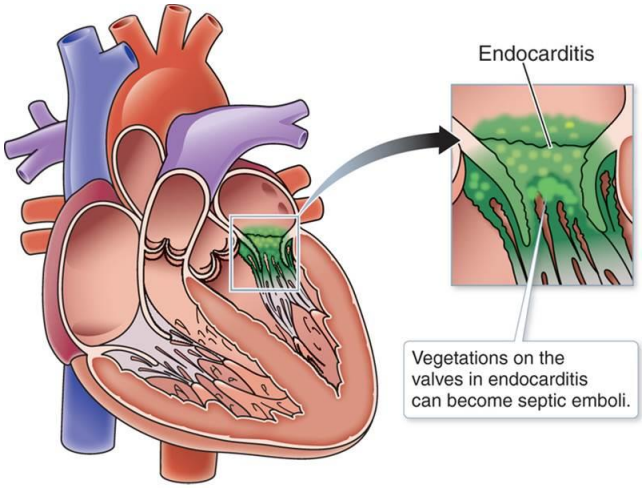
(Notes: Nitrates should not be prescribed for those taking phosphodiesterase-5 inhibitors [sildenafil] as may cause severe hypotension.)

Infectious Endocarditis (IE)

- Normally bacterial (*Staphylococcus aureus*)
- Damages valves
- Risk factors
  - Prosthetic valves, pacemakers, intravascular devices, IV drug usage
  - Pneumonia, pyelonephritis, dental procedures can be a source of microorganisms
- Vegetation develops on the valve
  - Vegetation fragments can break off and enter bloodstream (septic emboli)



Infectious Endocarditis (IE) (continued_1)



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Infectious Endocarditis (IE) (continued_2)

- Different potential causes account for different categories of endocarditis
  - *Examples:* prosthetic valve endocarditis, IVDA endocarditis, etc.
- Different organisms associated with the different causes
- Native valve endocarditis
  - Group A beta hemolytic *Streptococcus* (GABHS)

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Infectious Endocarditis (IE) (continued_3)

- Types: Acute or subacute infection
- S/S: New or worsening heart murmur is important sign
- Diagnosis based on labs and echocardiographic findings (Duke Criteria)
  - 3 blood cultures drawn 12 hours apart is preferred method
- Treatment
  - IV antibiotics for 6 weeks or longer

Myocarditis

inflammatory cardiomyopathy

- Mild disorder to lethal
- Viruses common cause (influenza, herpesvirus, hepatitis)
- Autoimmune diseases; toxin exposure
- Symptoms may include fever, myalgia, arthralgia, palpitations
- Pericardial friction rub and S3 gallop may be present

Myocarditis (continued)

- Diagnosis involves CBC, ESR, CRP
- cTnI may be elevated indicating myocardial damage
- Viral antibody titers should be measured
- Treatment is focused on reducing myocardial workload and treating underlying etiology
  - Activity restricted for 6 months or more
  - Treatments to support heart
    - ACE inhibitors, inotropic agents may be necessary

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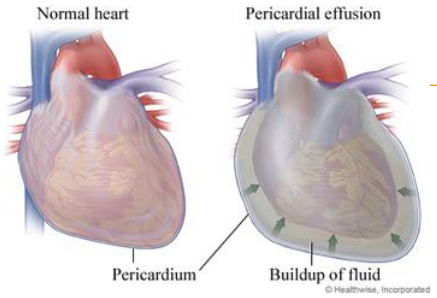
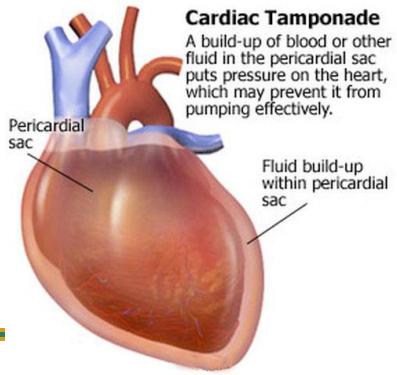
Pericarditis

Inflammation of pericardium and epicardium

- Pericardial effusion
  - Fluid surrounding the heart
- Cardiac tamponade
  - Fluid accumulation begins to compress the heart
- Acute pericarditis
  - Presents in manner similar to MI
  - Viral and bacterial infections may cause
  - May occur post-MI
- Dressler's syndrome
  - Specific form of pericarditis after MI





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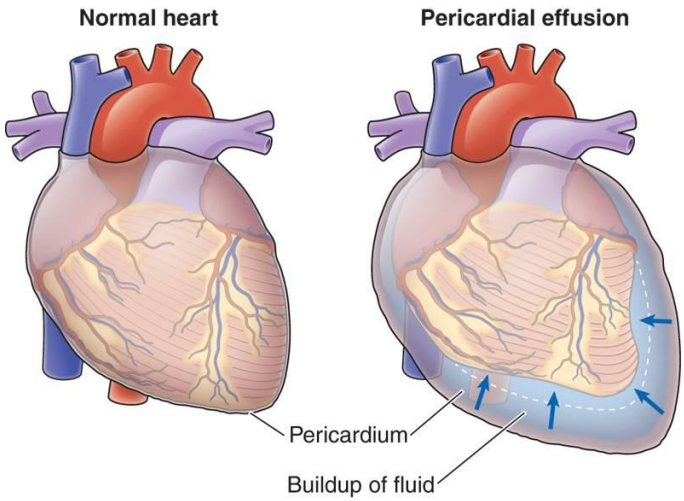
Pericardial Effusion



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Pericarditis (continued)

- Presents with chest pain, fever, pericardial friction rub
- Pain worsens with deep breathing
  - *Beck triad*: hypotension, jugular vein distention, muffled heart sound
  - Pulsus paradoxus: decrease in SBP by 10 mm Hg or more with inspiration
- Diagnosis
  - Echocardiogram, cardiac MRI
- Treatment depends on etiology

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