

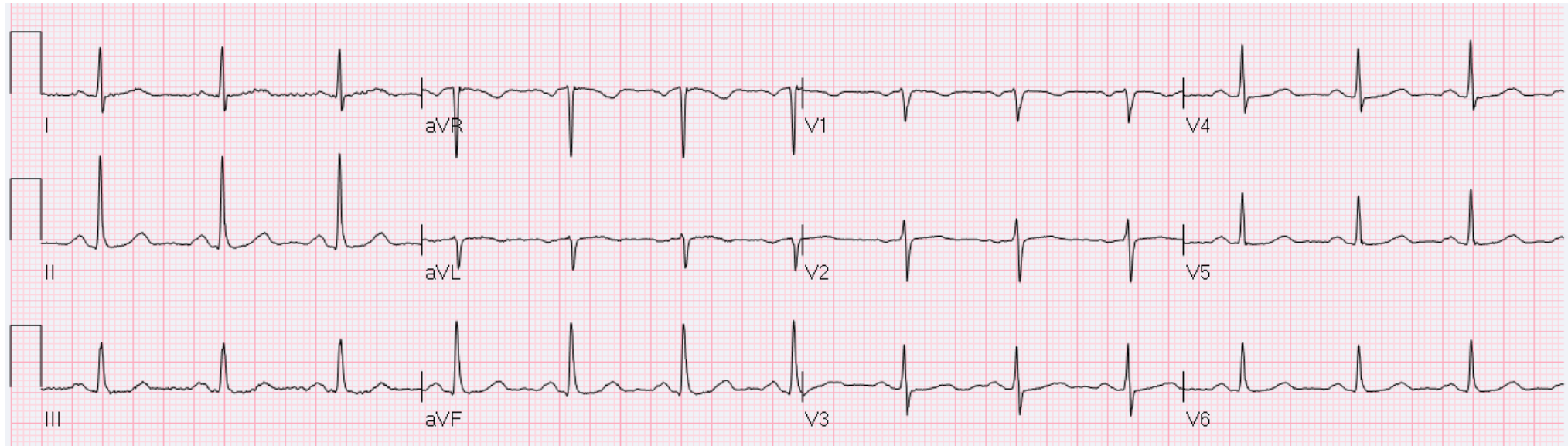
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EKG Interpretation

Jason Ryan, MD, MPH



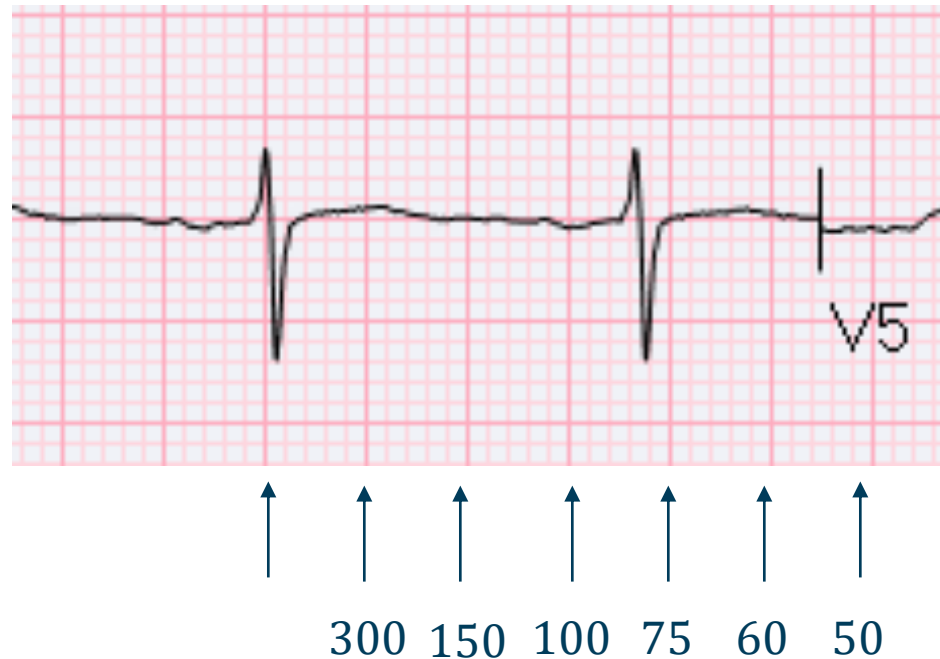
EKG



Determining Heart Rate

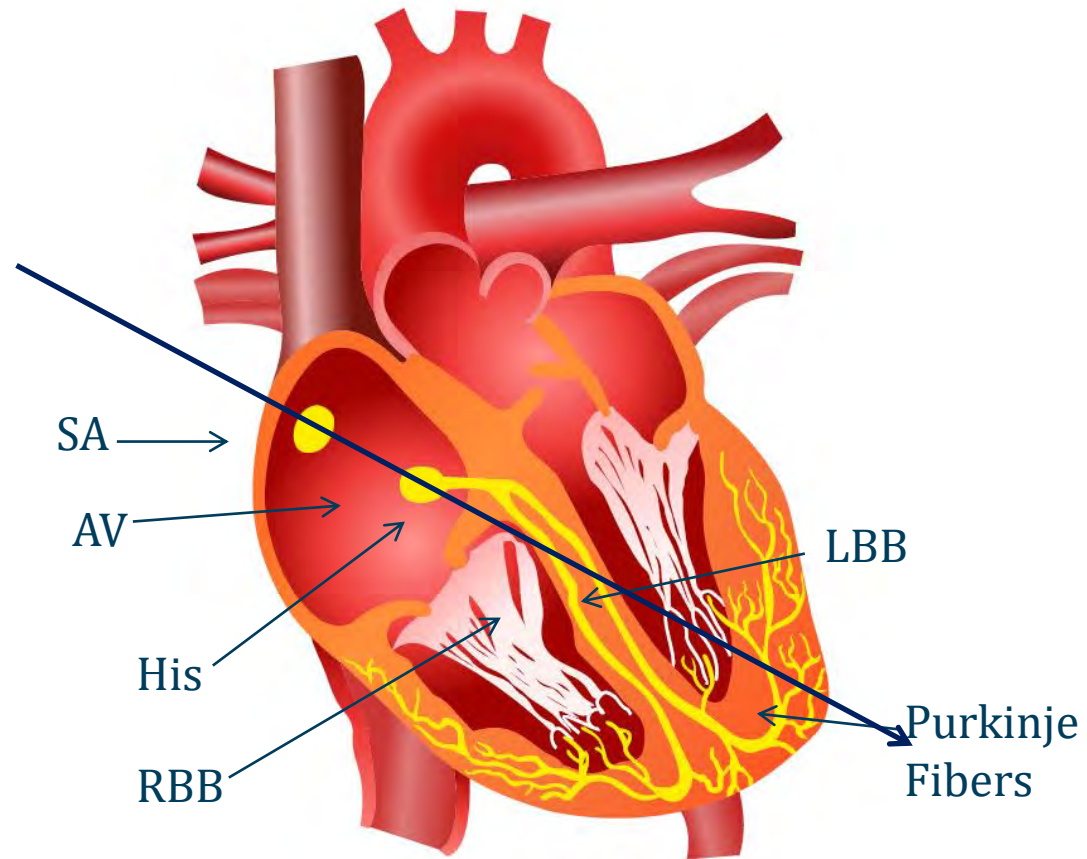
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- 3 – 5 big boxes between QRS complexes



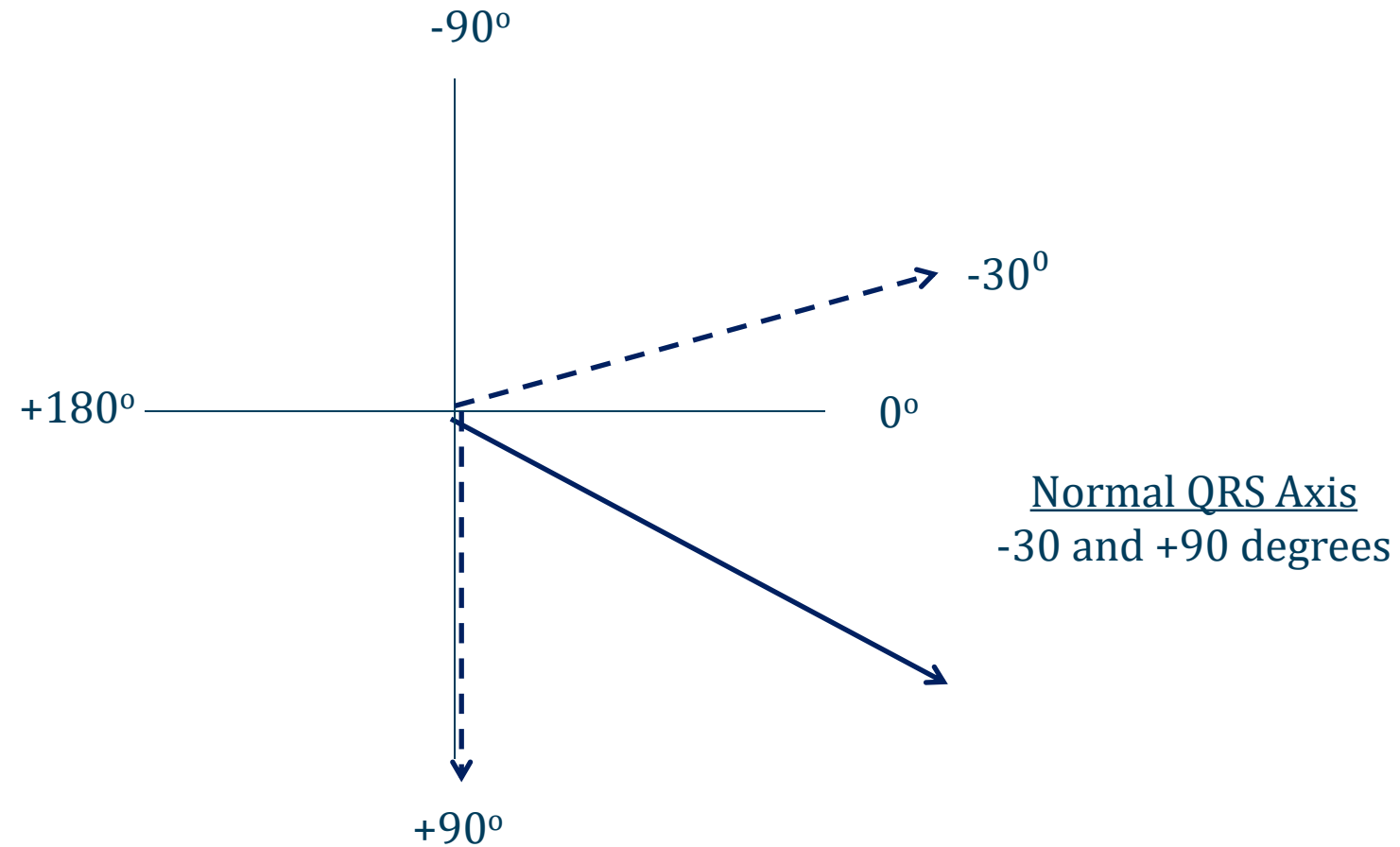
QRS Axis

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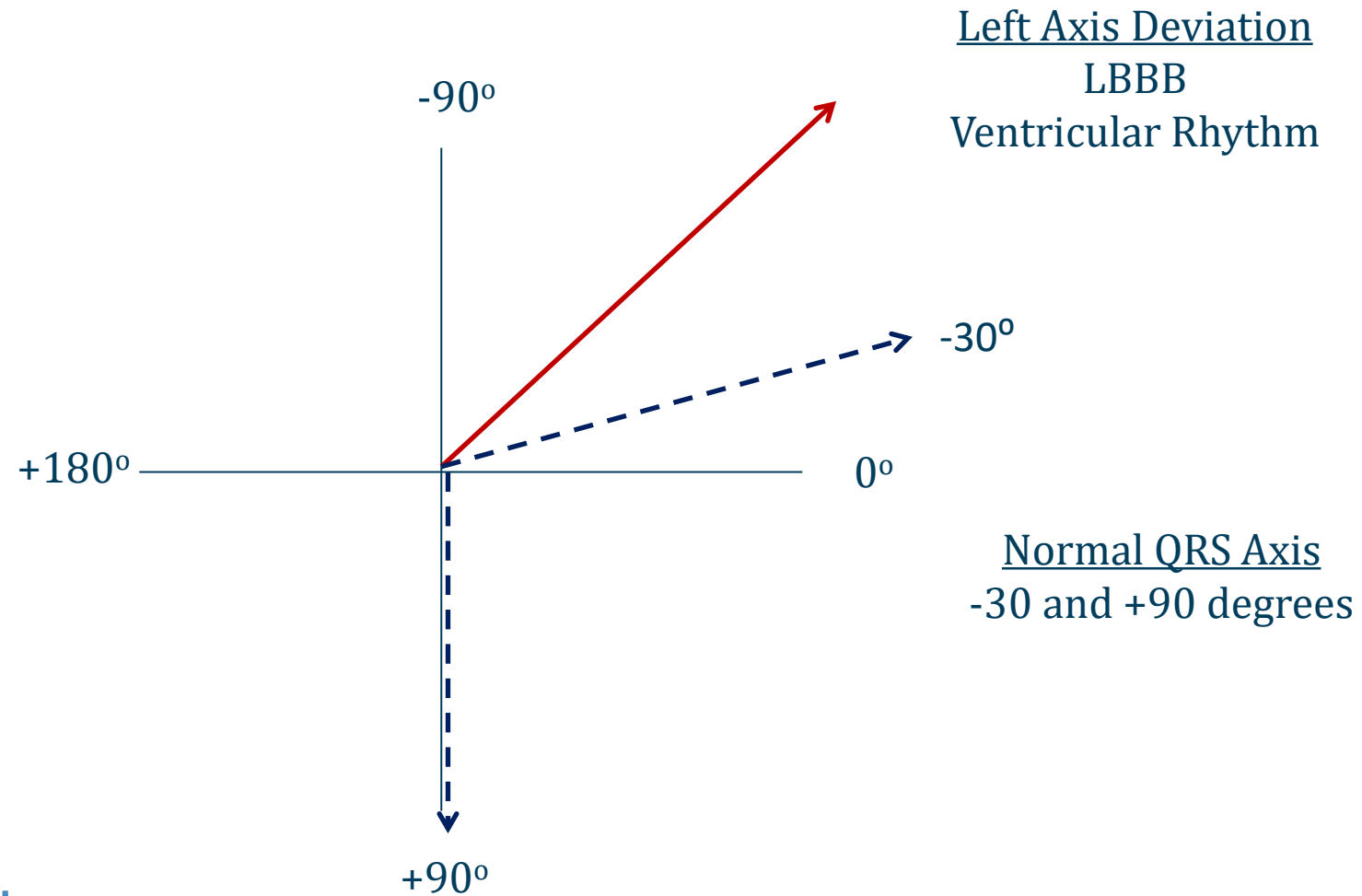
QRS Axis

www.afratafreeh.com



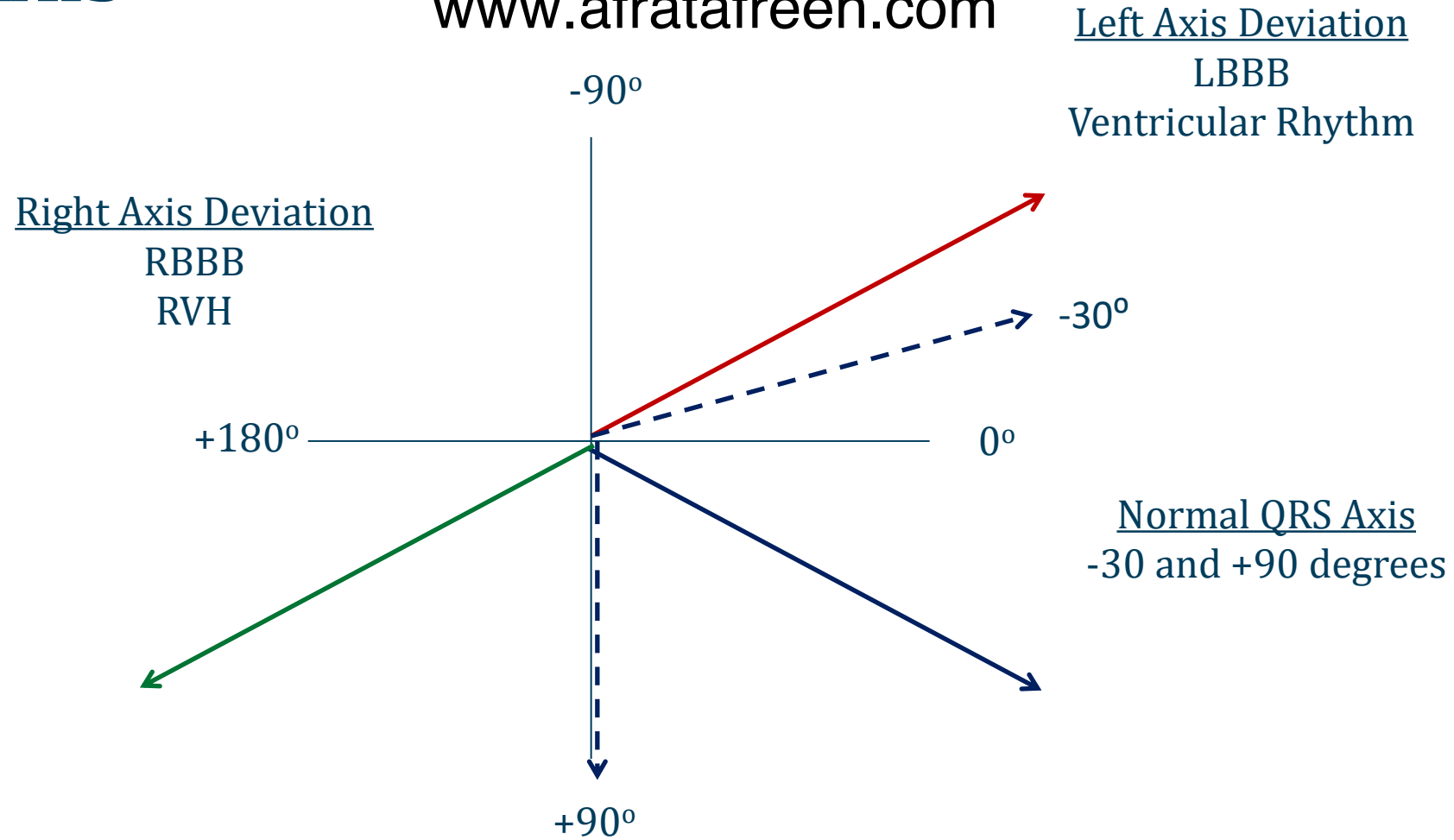
QRS Axis

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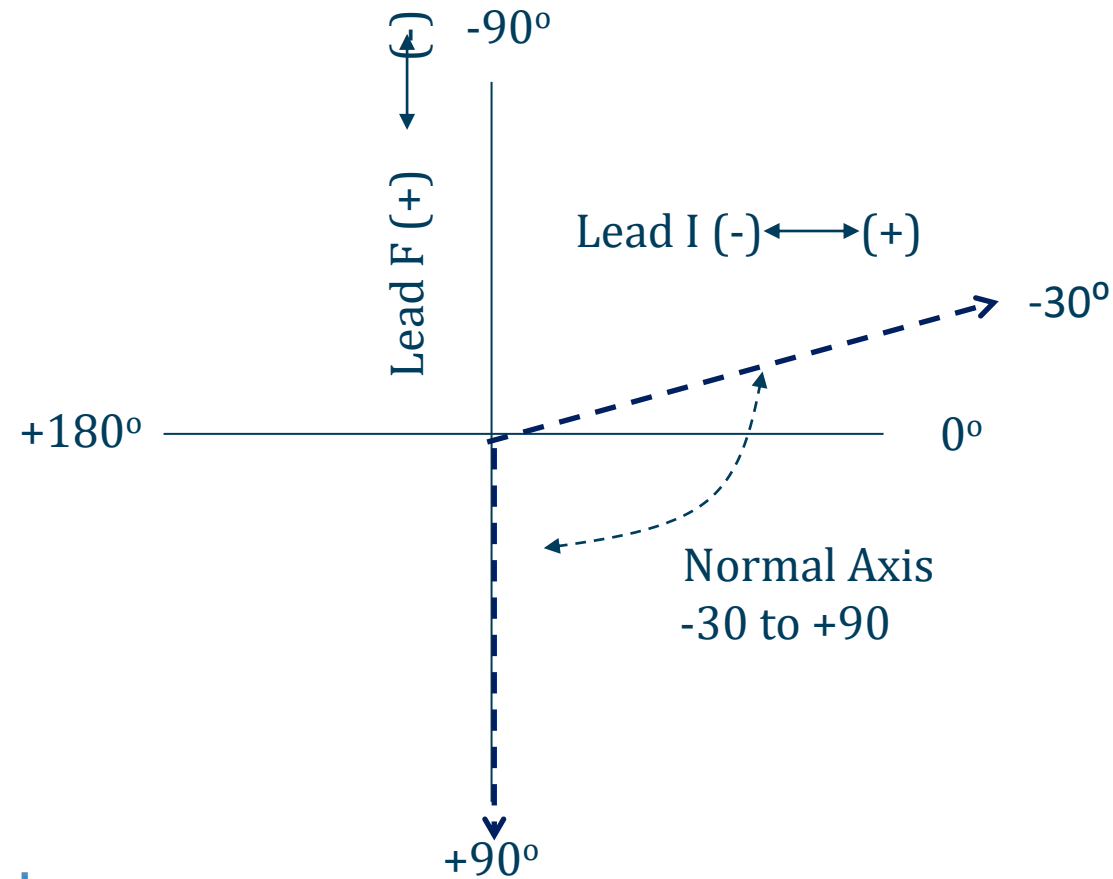
QRS Axis

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Determining Axis

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Axis Quick Method

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- First, glance at aVr
- It should be negative
- If upright: limb lead reversal



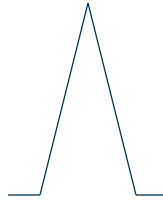
Normal

Axis Quick Method

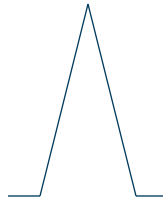
www.afratafreeh.com

- If leads I and II are both positive, axis is normal

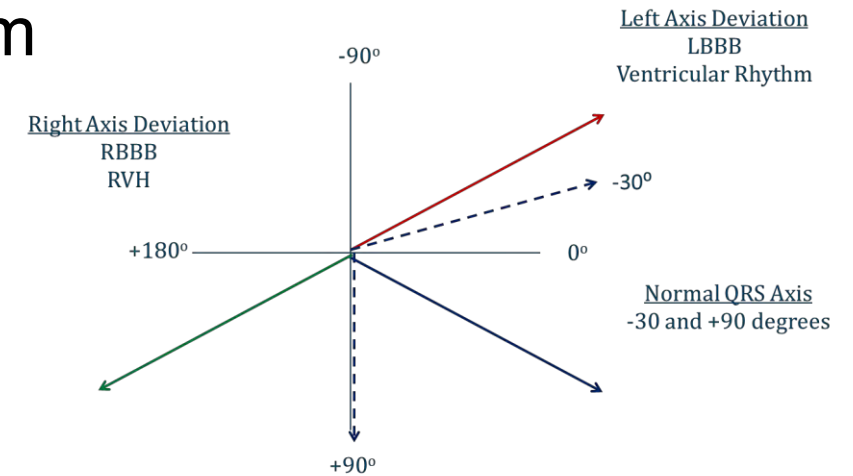
Lead I



Lead II



Axis 0 to 90°

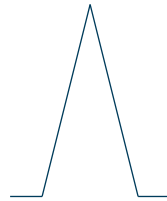


Axis Quick Method

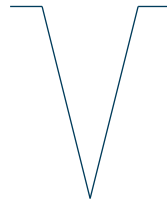
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- For left axis deviation:
 - All you need is lead II

Lead I

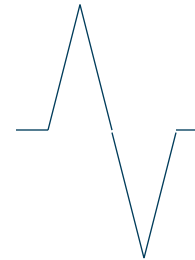


Lead II

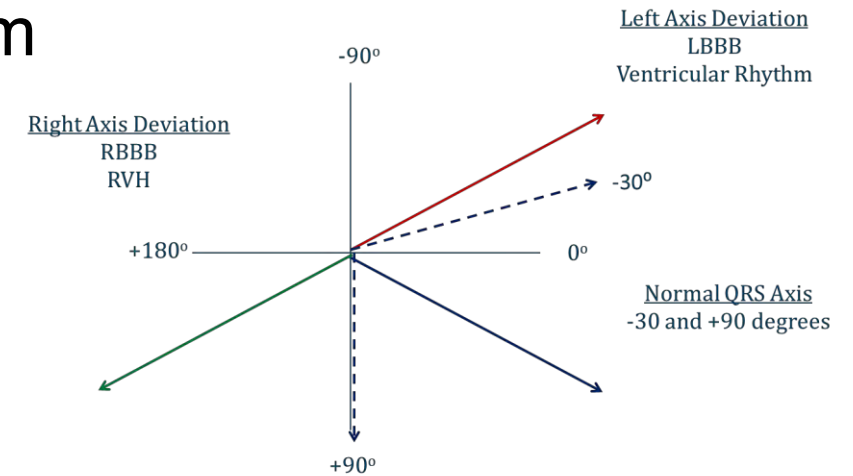


Axis -30 to -90°

Lead II



Axis 0 to -30°
Physiologic
Left Axis

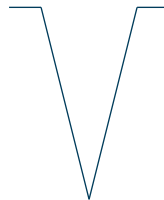


Axis Quick Method

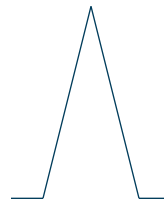
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- For right axis deviation:
 - All you need is lead I
 - Negative = RAD

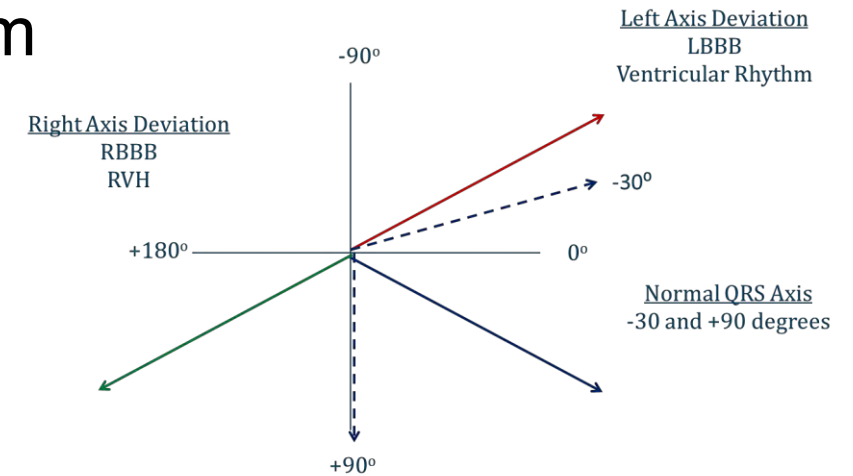
Lead I



Lead II



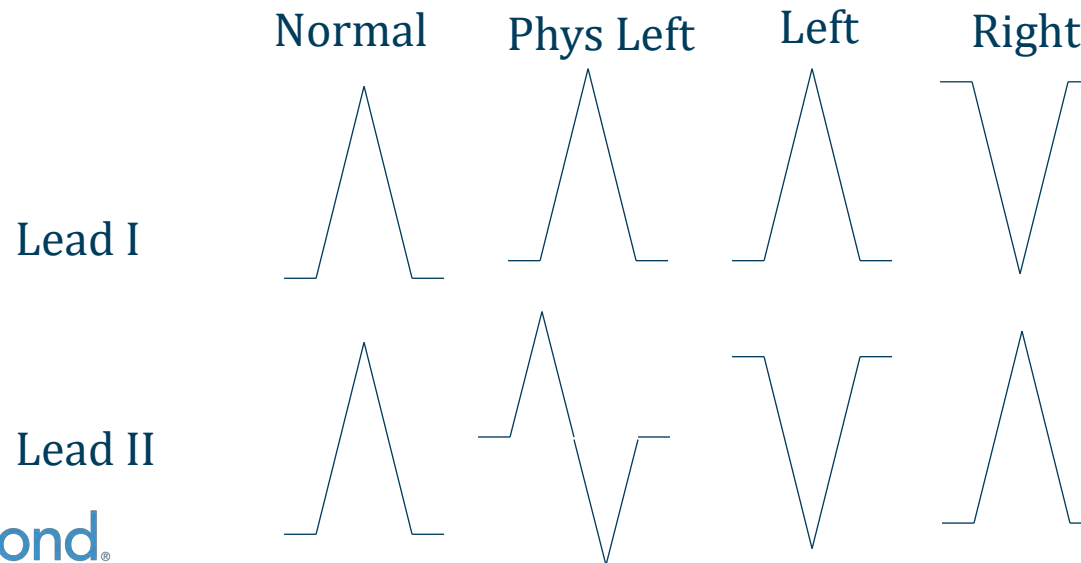
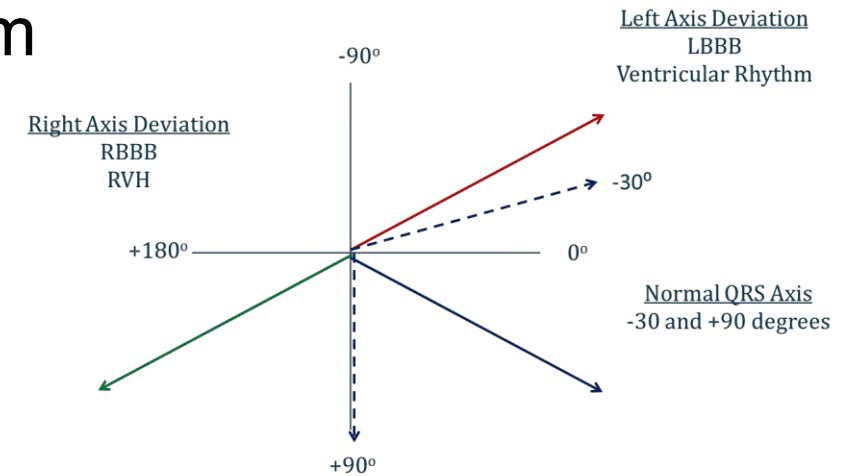
Axis 90 to 180°



Axis Quick Method

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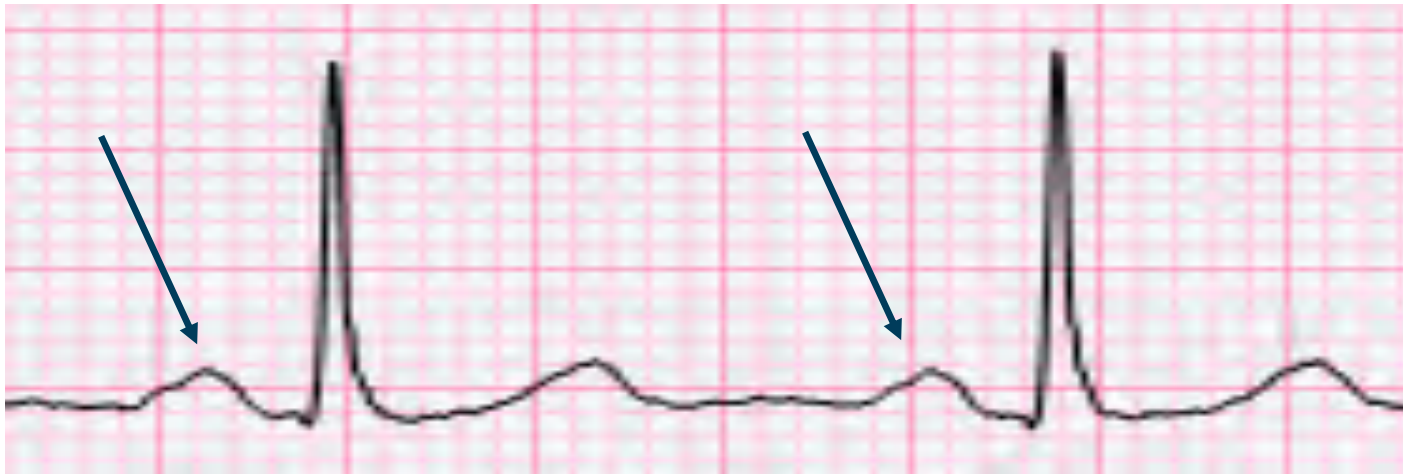
- Look at aVr: Make sure its negative
- Look at I, II: If both positive, axis is normal
- If II is negative: LAD
- If I is negative: RAD



Step 1: Find the p waves

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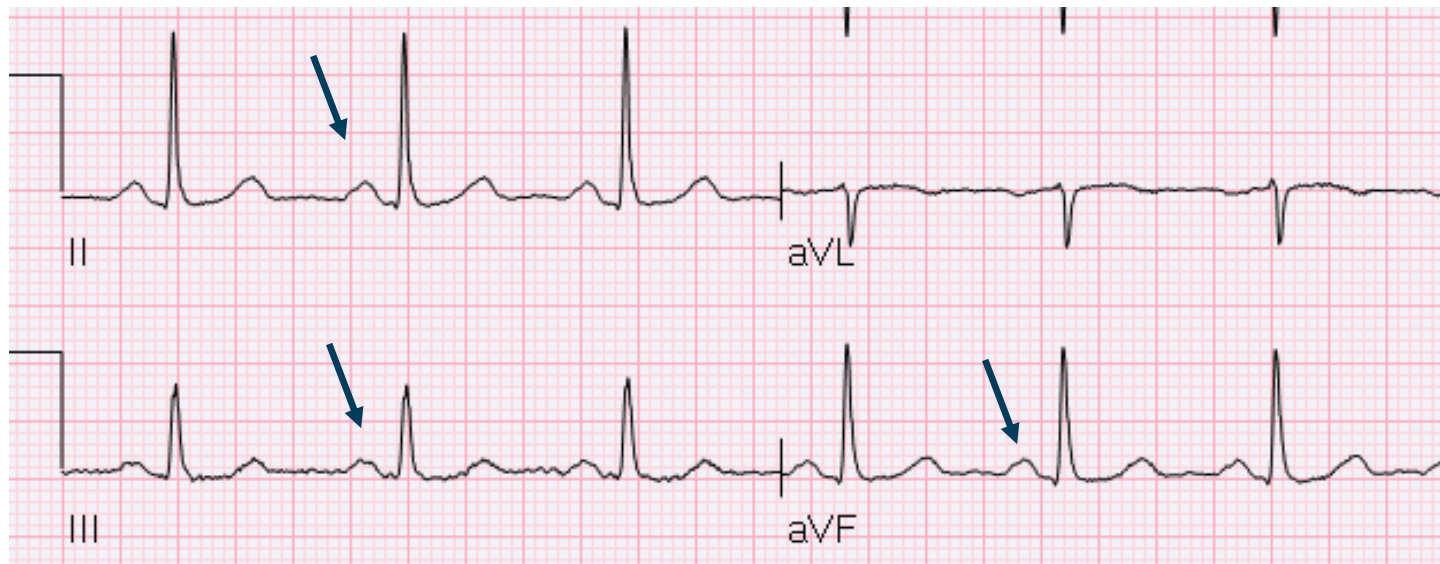
- Are p waves present?



Sinus p waves

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- Originate in sinus node
- Upright in leads II, III, F



Step 2: Regular or Irregular

www.afratafreeh.com

- Distance between QRS complexes (R-R intervals)

Regular



Irregular



Steps 1 & 2

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- P waves present, regular rhythm
 - **Sinus rhythm**
 - Rare: atrial tachycardia, atrial rhythm
- No p waves, irregular rhythm
 - **Atrial fibrillation – irregularly irregular**
 - Atrial flutter with variable block

Sinus Rhythm



Atrial Fibrillation



Steps 1 & 2

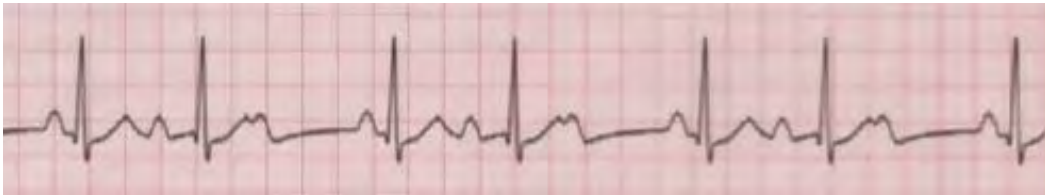
www.afratafreeh.com

- P waves present, irregular rhythm
 - Sinus rhythm with PACs
 - Multifocal atrial tachycardia
 - Sinus with AV block

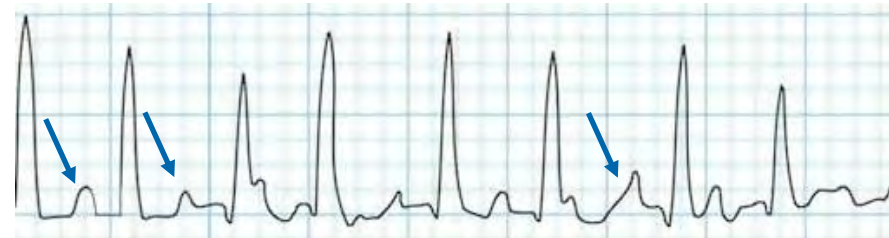
Sinus rhythm with PAC



Mobitz I AV Block (Wenckebach)



Mutifocal Atrial Tachycardia

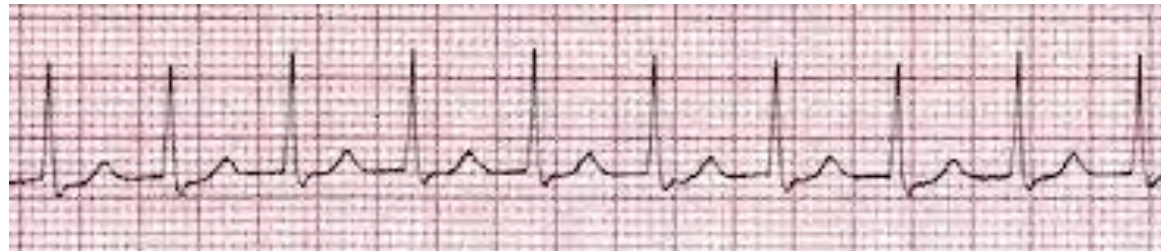


Steps 1 & 2

www.afratafreeh.com

- No p waves, regular rhythm
 - Hidden p waves: retrograde
 - Supraventricular tachycardias (SVTs)
 - Ventricular tachycardia

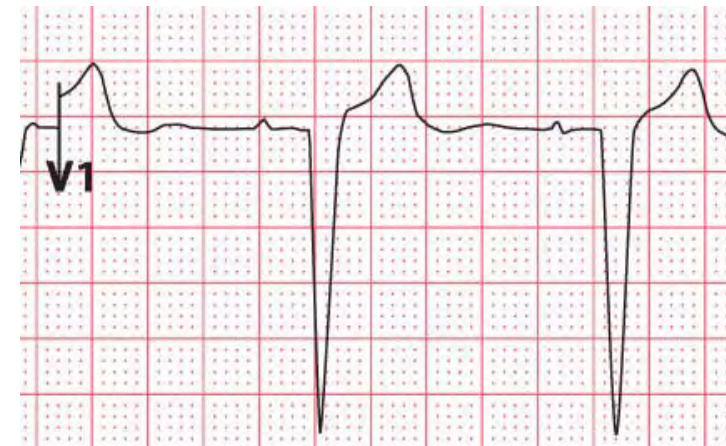
AV Nodal Reentrant Tachycardia (AVNRT)



Step 3: Wide or narrow

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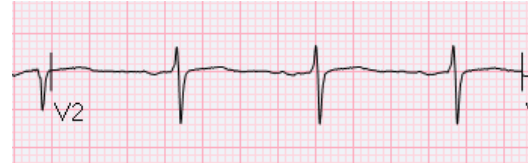
- Narrow QRS (< 120 ms; 3 small boxes)
 - His-Purkinje system works
 - No bundle branch blocks present
- Wide QRS
 - Most likely a **bundle branch block**
 - Ventricular rhythm (i.e. tachycardia)



QRS Interval

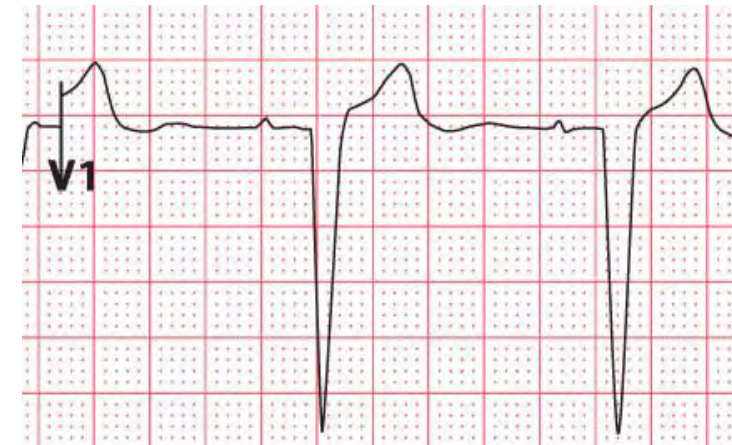
www.afratafreeh.com

Normal QRS



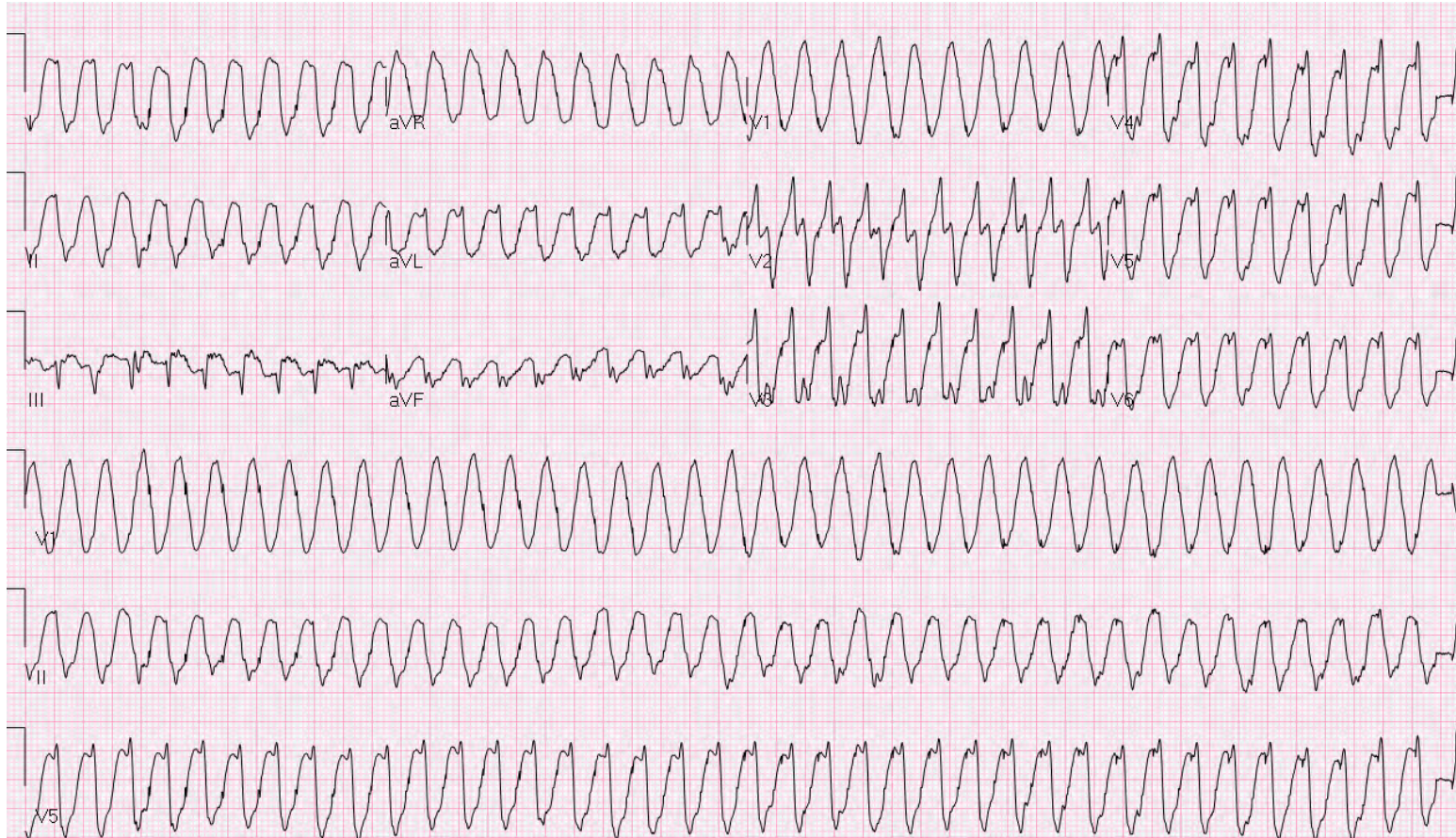
Right Bundle
Branch Block

Left Bundle
Branch Block



Ventricular Tachycardia

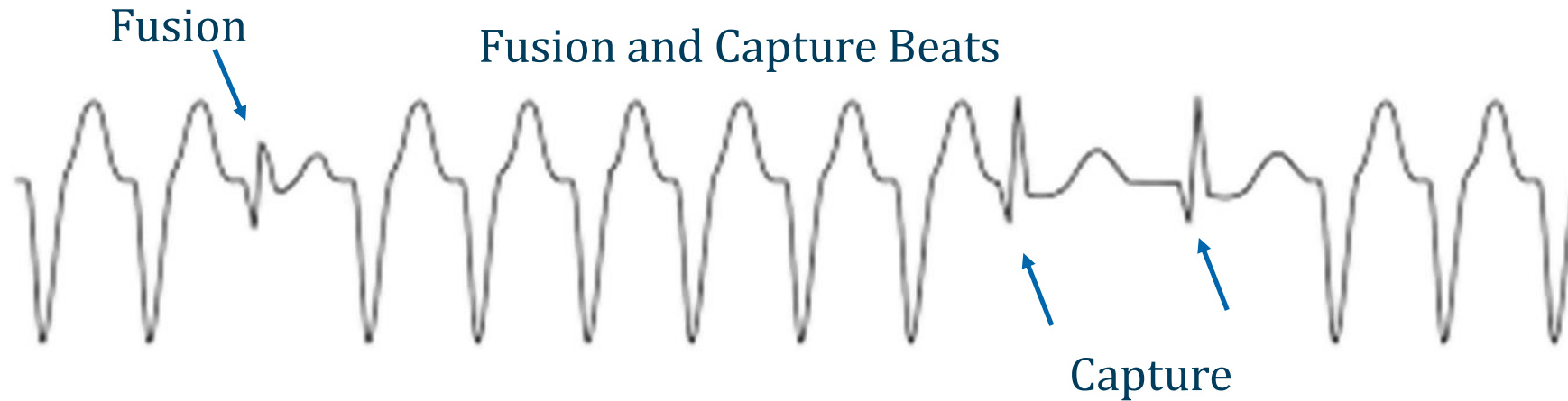
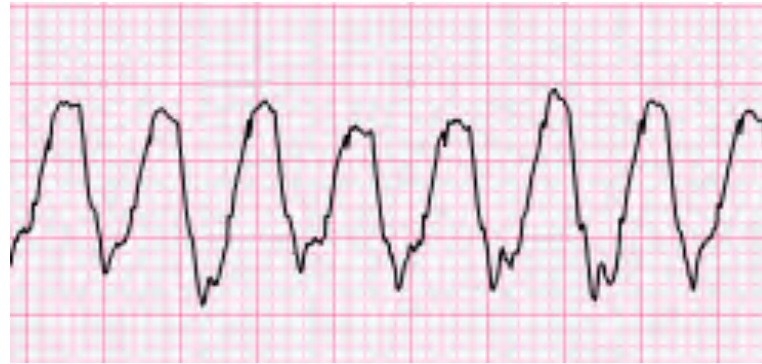
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Ventricular Tachycardia

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AV Dissociation



Step 4: Check the intervals

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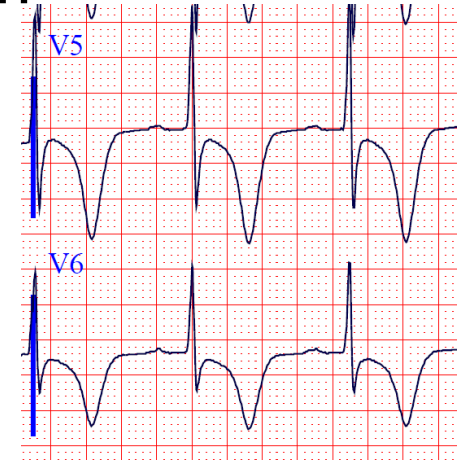
- PR (normal < 210 ms; ~5 small boxes; ~1 big box)
 - Prolonged in AV block, drugs
- QT (normal < 1/2 R-R interval)
 - Prolonged with ↓ Ca
 - Shortened with ↑ Ca
 - Prolonged by antiarrhythmic drugs, other drugs
 - Can lead to torsades de pointes



Step 5: ST segments

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- T wave abnormalities
 - Inverted: ischemia
 - Peaked: Early ischemia, hyperkalemia ($\uparrow K$)
 - Flat/U waves: hypokalemia ($\downarrow K$)
- ST depression
 - Subendocardial ischemia
 - Common in UA/NSTEMI
- ST elevation
 - Transmural ischemia
 - STEMI



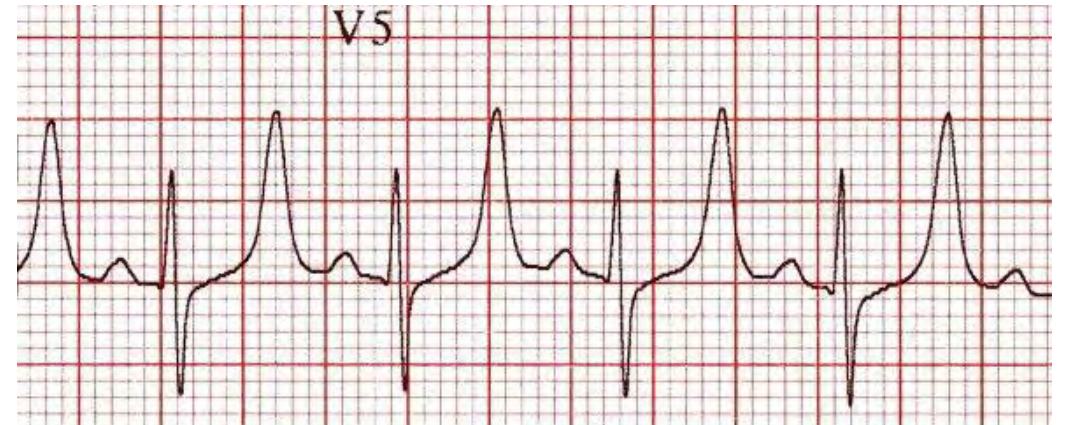
Peaked T waves www.afratafreeh.com

- Hyperkalemia
- Early ischemia: “hyperacute”
 - Precedes ST elevation

Normal T waves



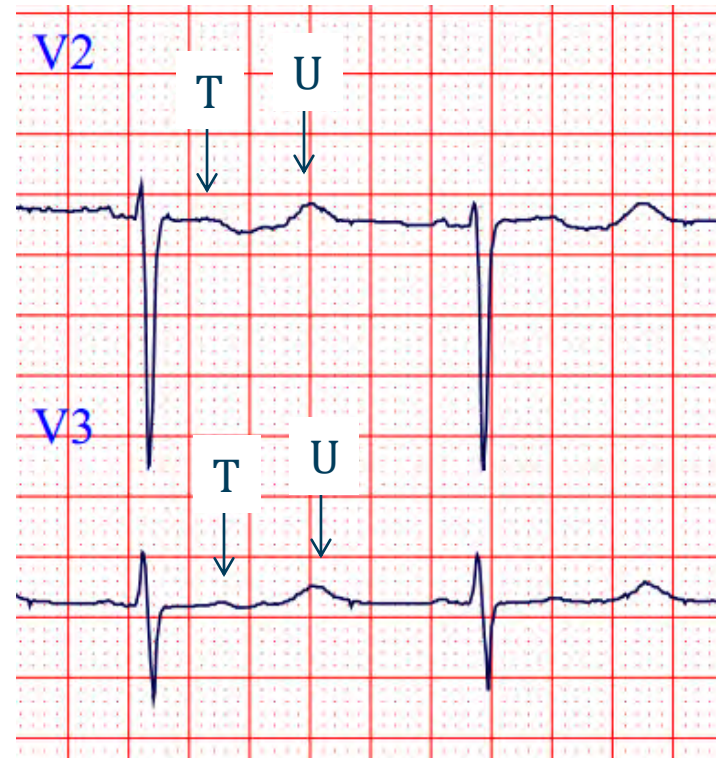
Peaked T waves



U waves

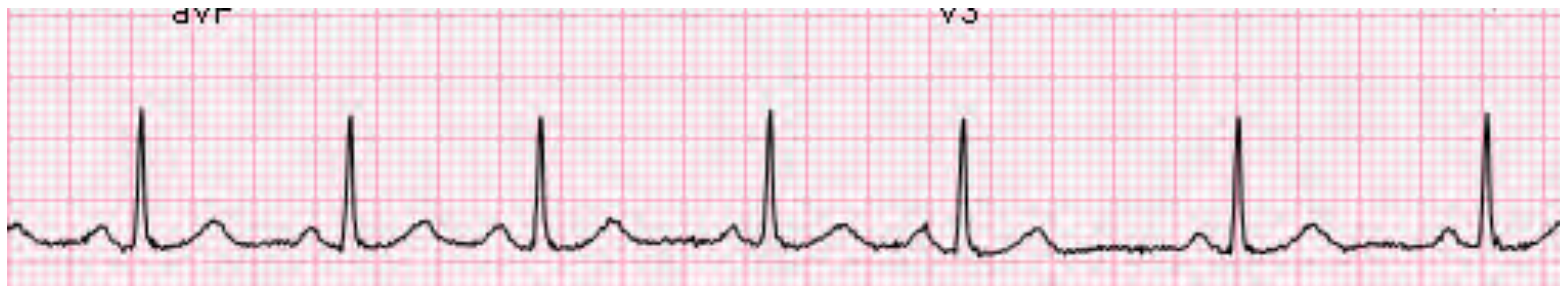
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- Can be normal
- Also seen in *hypokalemia*



PAC and PVC

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Coronary Artery Disease

Jason Ryan, MD, MPH



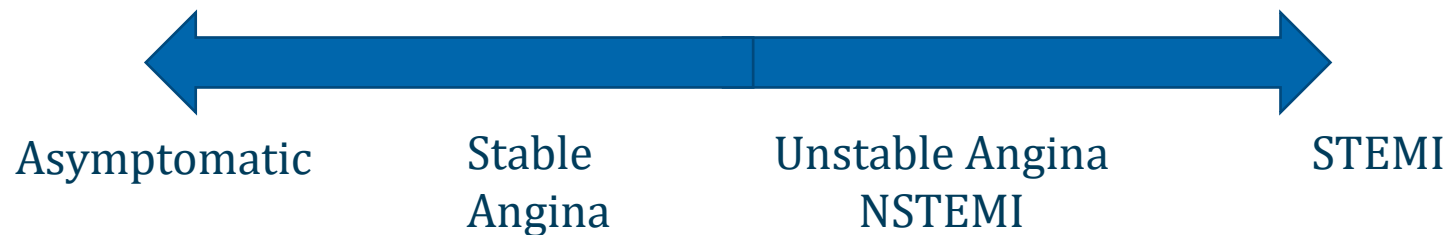
Coronary Artery Disease

www.afratafreeh.com

- Narrowing of coronary artery
- Caused by atherosclerosis
- Asymptomatic until ~75% artery lumen occluded
- **Chest pain (angina)**
- May also cause dyspnea, other symptoms



Freestocks.org



Major Risk Factors

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CAD Equivalents

Diabetes

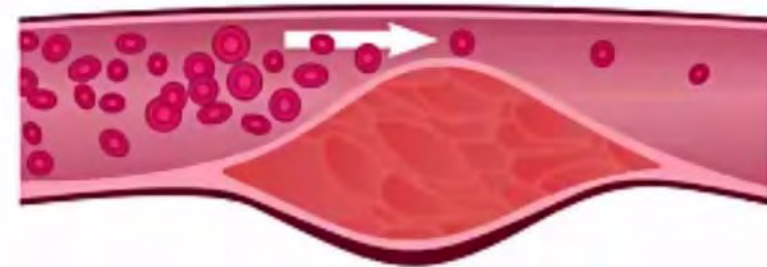
CKD

- Diabetes
- Chronic kidney disease
- Hypertension
- Hyperlipidemia (LDL)
- Age (M > 45, F > 55)
- Family History of premature CAD (1° relative, M < 55, F < 65)
- Smoking (quitting smoking → significantly ↓ risk)
- Obesity, sedentary lifestyle

Stable Angina

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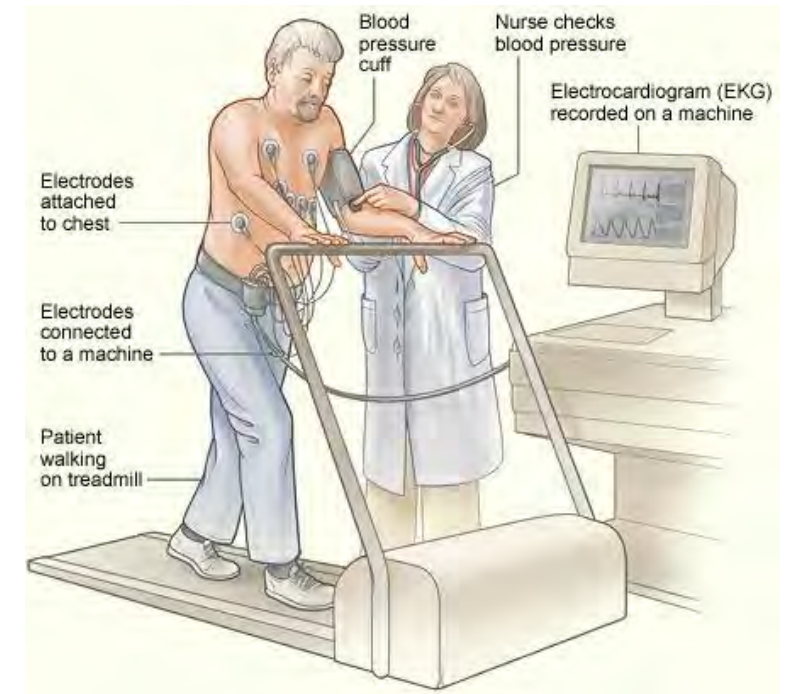
- Plaque occluding ~75% or more of coronary artery
- Causes “typical” chest pain
 - Pressure-like chest pain
 - Occur with exertion
 - Relieved by rest or nitroglycerine
- EKG at rest: normal
- Symptoms at rest: absent
- Diagnosis: symptoms or stress testing
 - Stress testing key when diagnosis uncertain



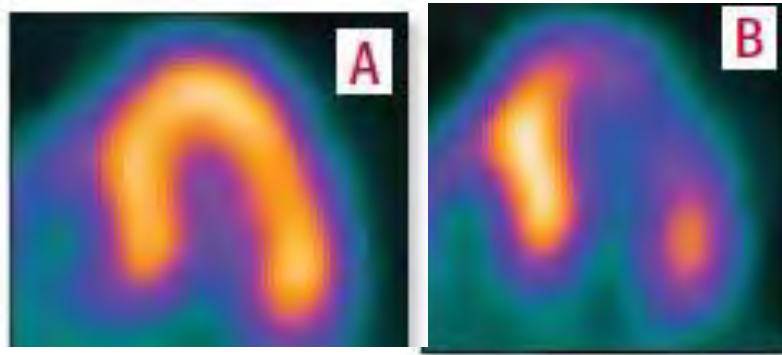
Stress Testing

www.afratafreeh.com

- Patient must be asymptomatic
- Goal: provoke ischemia and detect ischemia
- Provocation: exercise always preferred
- Detection:
 - EKG changes (ST-depressions)
 - Nuclear imaging (usually Technetium)
 - Echocardiography



Wikipedia/Public Domain



Pharmacologic Stress Testing

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- Pharmacologic nuclear (induce **coronary steal**)
 - Regadenoson
 - Dipyrimadole
 - Adenosine
 - Persantine
 - Contraindication: reactive airway disease/wheezing
- Dobutamine stress echocardiography
 - Risk of arrhythmias

Stress Testing

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- Identifies ischemia due to **“flow-limiting” stenosis** (usually >75%)
- Non-flow-limiting lesions not detected
- Acute MI may occur despite negative stress test

Angiography

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Stable Angina

Management

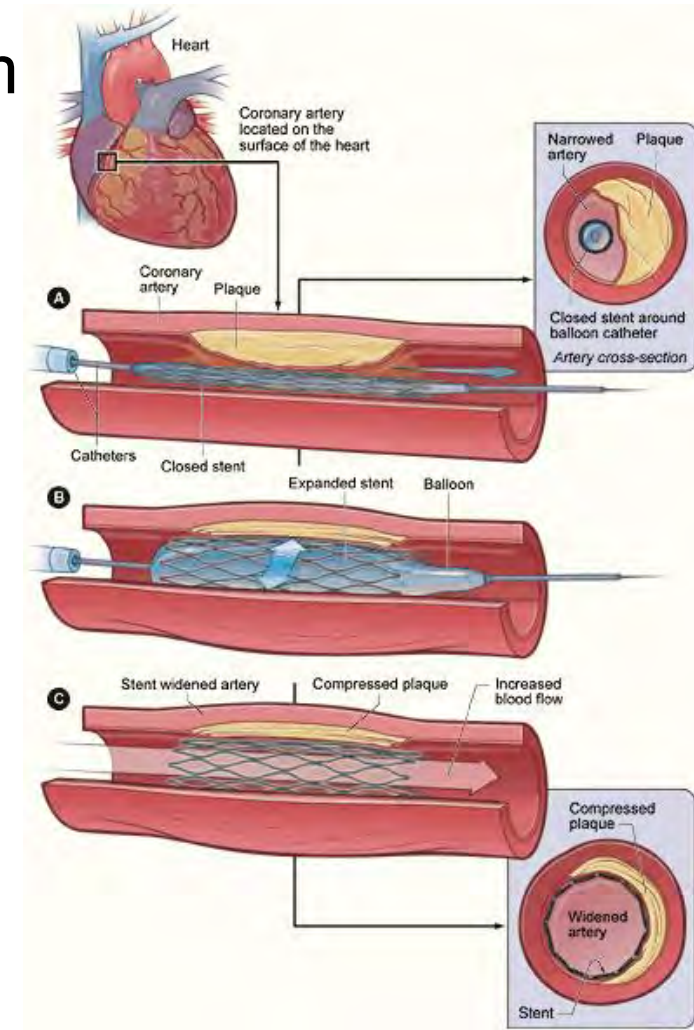
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- **Preventative therapy** (↓ risk of mortality and myocardial infarction)
 - Aspirin
 - Statin
- Anti-angina therapy (goal: ↓ O₂ demand)
 - Beta-blockers or calcium-channel blockers (↓ heart rate/contractility)
 - Nitroglycerine (↓ preload)
- Coronary stent implantation
- Coronary artery bypass grafting (CABG) surgery
- COURAGE Trial: medical therapy has similar outcomes to stent implantation

Coronary Stents

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- Percutaneous Coronary Intervention (PCI)
- About 600,000 stents/year implanted US

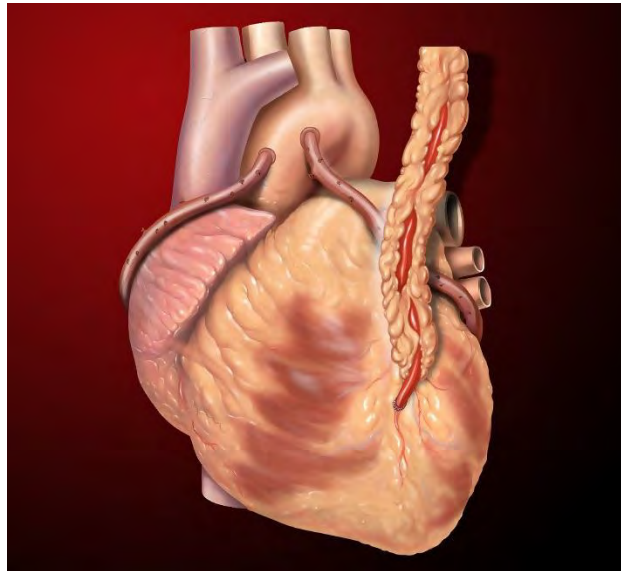


CABG

Coronary Artery Bypass Grafting

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- “Bypass Surgery”
- Left Internal Mammary Artery (LIMA) Graft
- Saphenous (leg) Vein Grafts
- Radial (arm) Artery Grafts



Patrick J. Lynch/Wikipedia

Stable Angina: Typical Case

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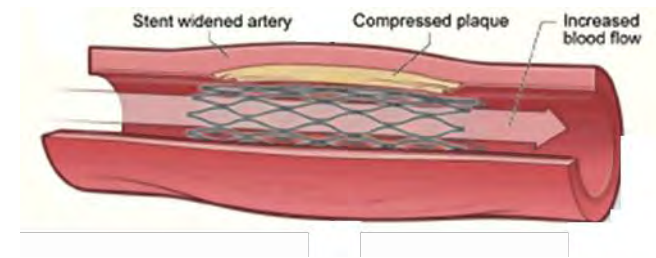
- 65-year-old man with chest pain while walking
- Relieved with rest
- Presents to ED:
 - EKG normal
 - Biomarkers normal
- Stress test
 - Walks on treadmill → chest pain, EKG changes
- Cardiac catheterization performed
- 90% LAD artery blockage
- Stent placed → angina resolved

Stent Complications

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Restenosis

- Slow, steady growth of scar tissue over stent
- “Neo-intimal hyperplasia”
- Re-occlusion of vessel
- Rarely life-threatening
- Slow, steady return of angina
- Most stents coated “drug-eluting stents”
 - Metal stent covered with polymer
 - Polymer impregnated with drug to prevent tissue growth
 - Sirolimus

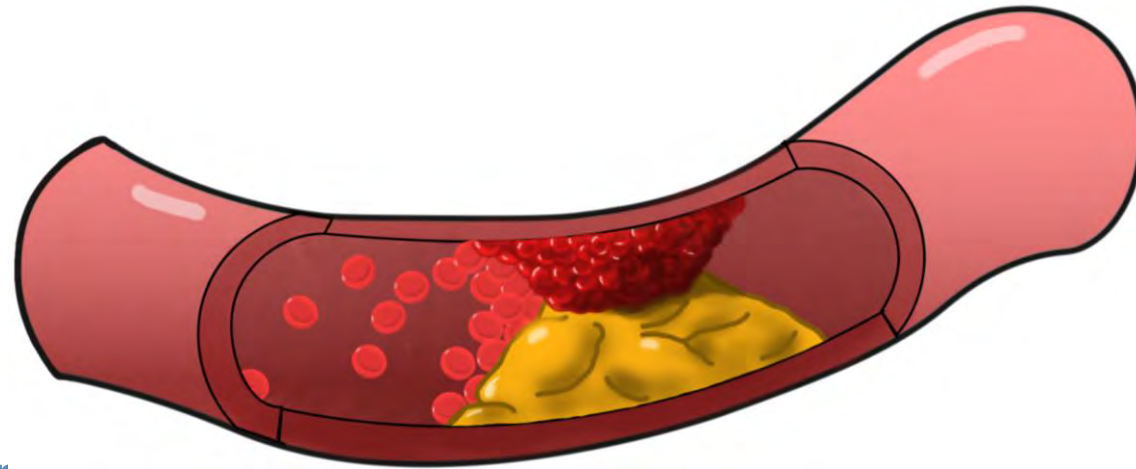


Stent Complications

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Thrombosis

- Acute closure of stent
- Same as STEMI: life-threatening event
- Dual anti-platelet therapy for prevention
- Associated with **missed medication doses**



Stent Thrombosis Prevention

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- “Dual antiplatelet therapy”
- Typically one year of:
 - Aspirin
 - Clopidogrel, prasugrel or ticagrelor (P2Y₁₂ receptor antagonists)
- After one year, stent metal no longer exposed to blood
 - Scar tissue and endothelial growth
 - Risk of thrombosis is lower (but not zero)
 - Most patients take aspirin only

Variant (Prinzmetal) Angina

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- Episodic vasoconstriction of coronary vessels
- Episodes usually **at rest**
- Midnight to **early morning**
- Sometimes symptoms improve with exertion
- Associated with smoking
- Diagnosis: usually based on history

Variant (Prinzmetal) Angina

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Treatment

- Quit smoking
- **Calcium channel blockers, nitrates**
 - Dihydropyridine CCB: amlodipine
 - Vasodilators
 - Dilate coronary arteries, oppose spasm
- Avoid propranolol
 - Nonselective beta blocker
 - Can cause unopposed alpha stimulation
 - Symptoms may worsen

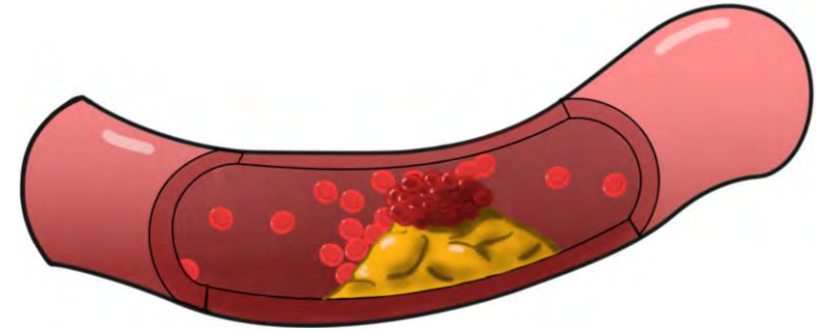


Pixabay/Public Domain

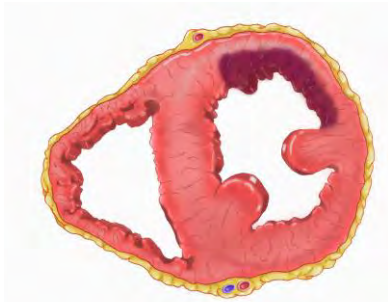
Acute Coronary Syndromes

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- Plaque rupture → thrombus formation
- Subtotal occlusion
 - Unstable angina
 - Non-ST elevation myocardial infarction
- Total occlusion (100%)
 - ST-elevation myocardial infarction (STEMI)



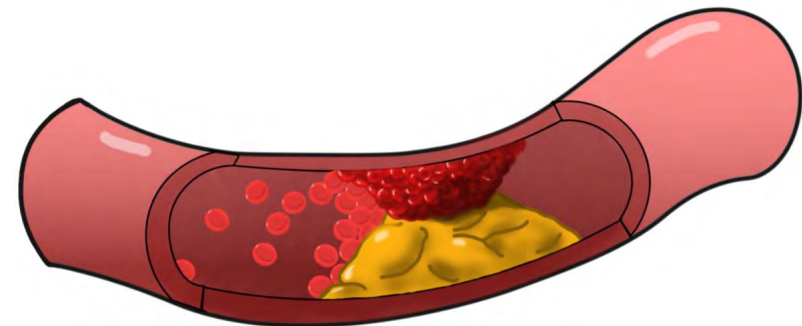
Subtotal Occlusion



Subendocardial Ischemia



Transmural Ischemia



Total Occlusion

Unstable Angina and NSTEMI

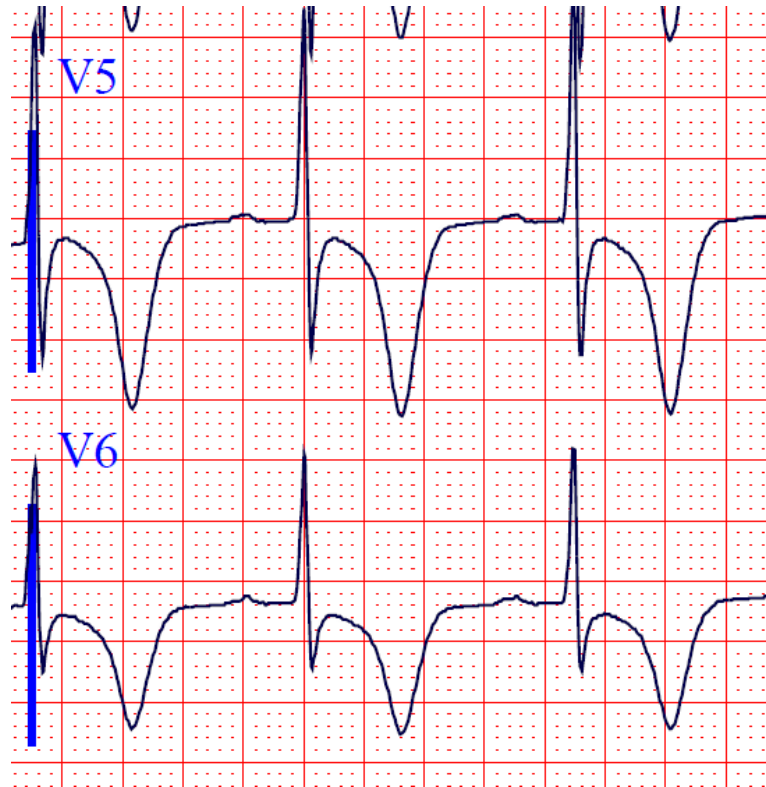
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- Unstable angina
 - Ischemic symptoms occurring with increasing frequency or at rest
 - Negative biomarkers
- NSTEMI
 - Positive biomarkers
- Ischemic EKG changes (other than ST-elevation) may occur in both

ECG Changes

- ST depressions
- T-wave inversions

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UA/NSTEMI

Cardiac Biomarkers

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- Biomarkers spill into blood with cardiac injury
- Most common marker used: **Troponin I or T**
 - Increase 2-4 hours after MI
 - Stay elevated for weeks
- **CK-MB also used**
 - Increase 4-6 hours after MI
 - Normalize within 2-3 days
 - Very good for re-infarction

Treatment of UA/NSTEMI

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- Thrombotic and ischemic syndrome (like STEMI)
- No “ticking clock” (unlike STEMI)
 - Subtotal occlusion
 - Some blood flow to distal myocardium
 - No emergency angioplasty
 - No benefit to thrombolysis
- Aspirin
- Beta-blocker
- Heparin
- Angioplasty (non-emergent)

Typical UA/NSTEMI Course

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- Presents to ER with chest pain
- Biomarkers elevated
- Absence of ST-segment elevations ← Obtain EKG in all chest pain patients
- Medical Therapy
 - Aspirin ← Give aspirin to chest pain patients with possible NSTEMI
 - Metoprolol
 - Heparin drip
- Admitted to cardiac floor
- Hospital day 2 → angiography
- 90% blockage of LAD → Stent

UA/NSTEMI Treatment

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Long-term therapy

- Goal: ↓ **mortality and recurrent infarction**
 - Aspirin
 - Statin
- Beta-blocker for prevention of recurrent disease
 - Strong indication in STEMI
 - Weak indication NSTEMI/UA
 - Used for prevention in NSTEMI only

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STEMI

Jason Ryan, MD, MPH



STEMI

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ST-Elevation Myocardial Infarction

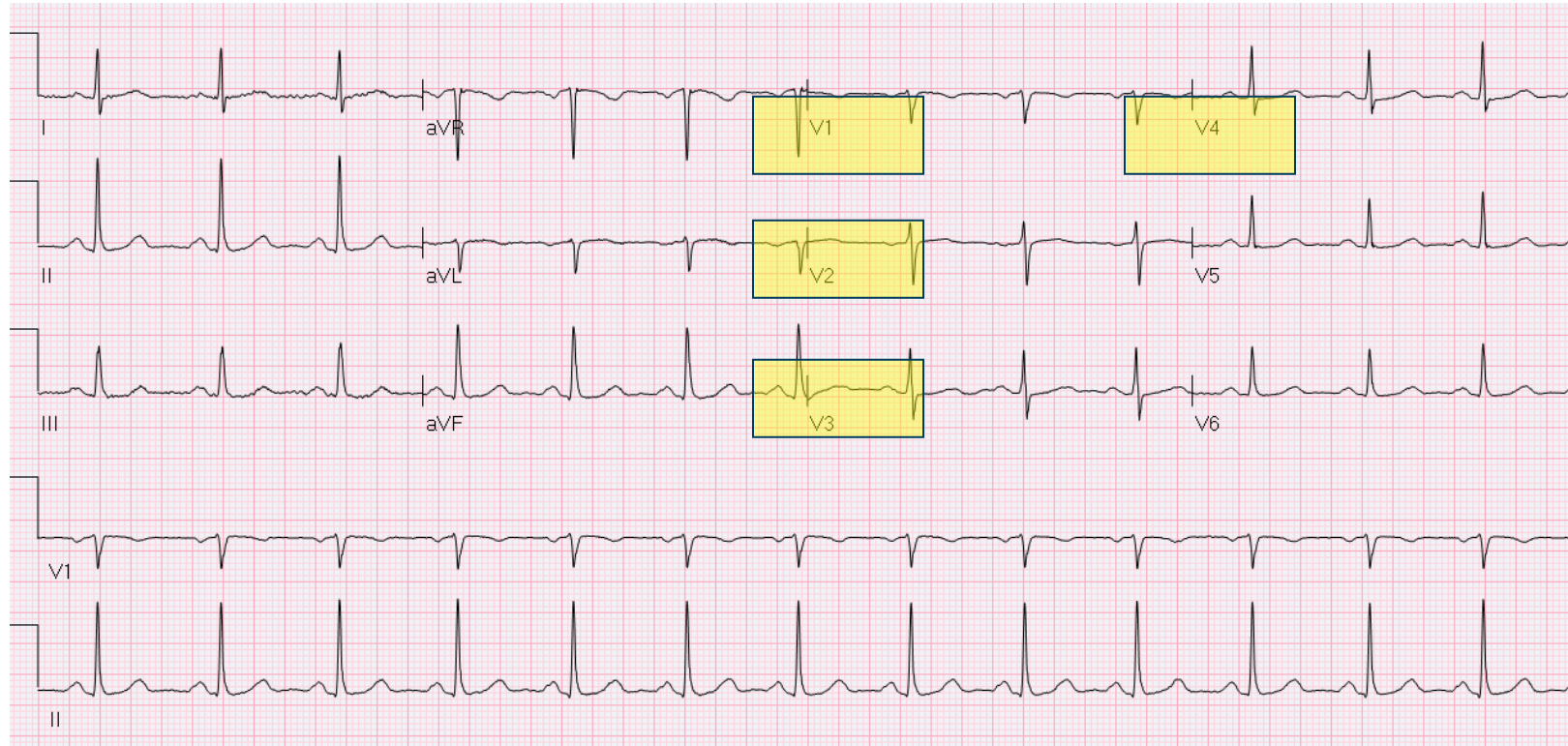
- Angina at rest with ST-segment elevation
- Biomarkers elevated after 4-6 hours



Leads go together

ST Elevations - Anterior

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Leads go together

ST Elevations - Anterior

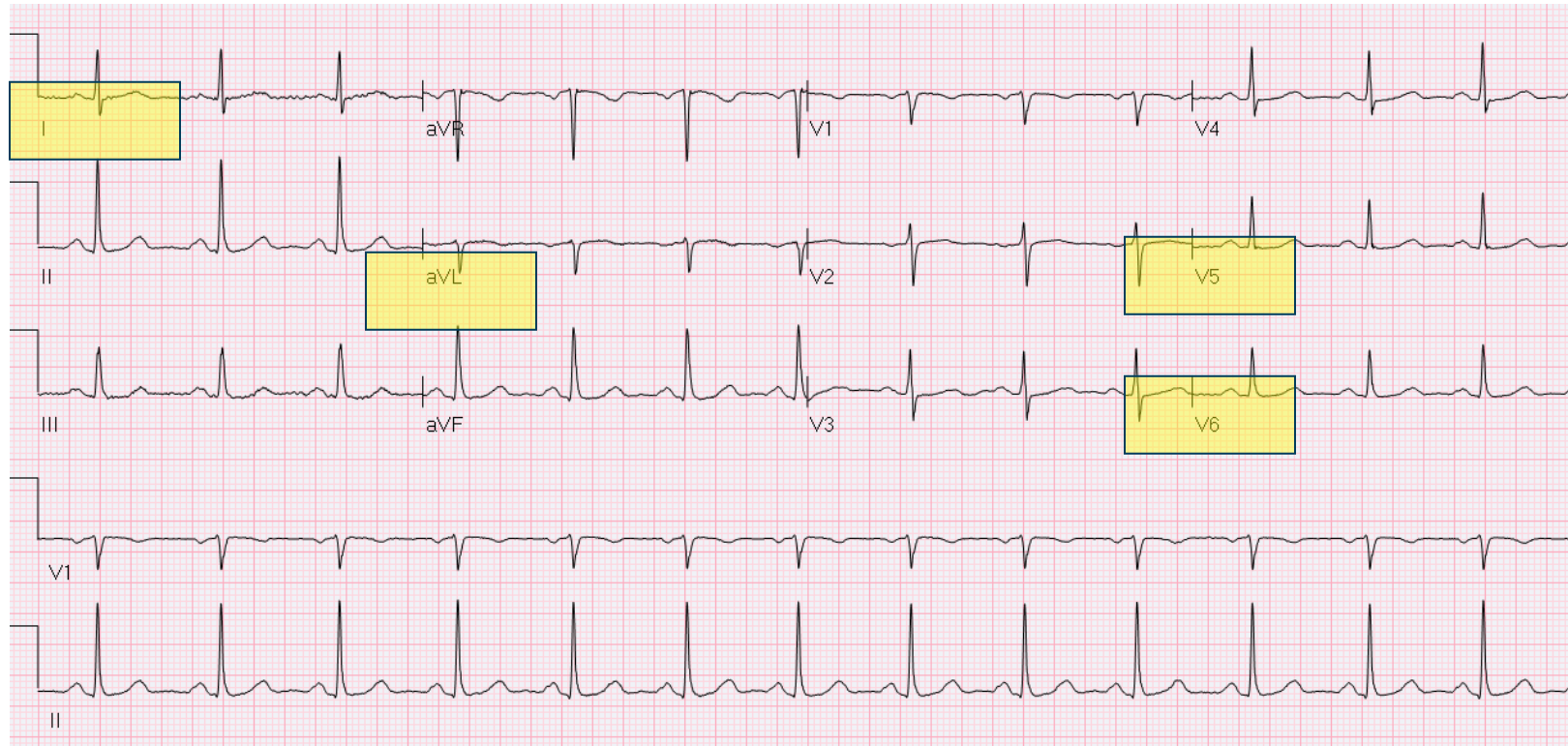
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Leads go together

ST Elevations - Lateral

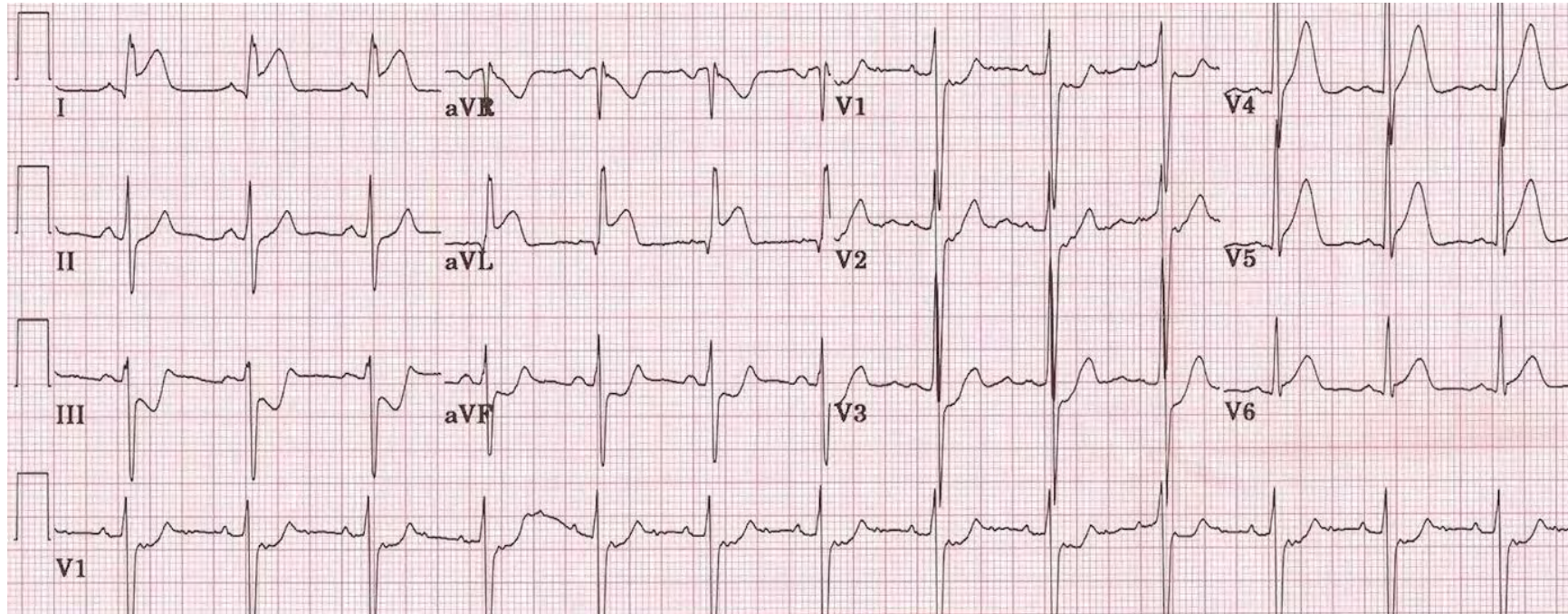
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Leads go together

ST Elevations - Lateral

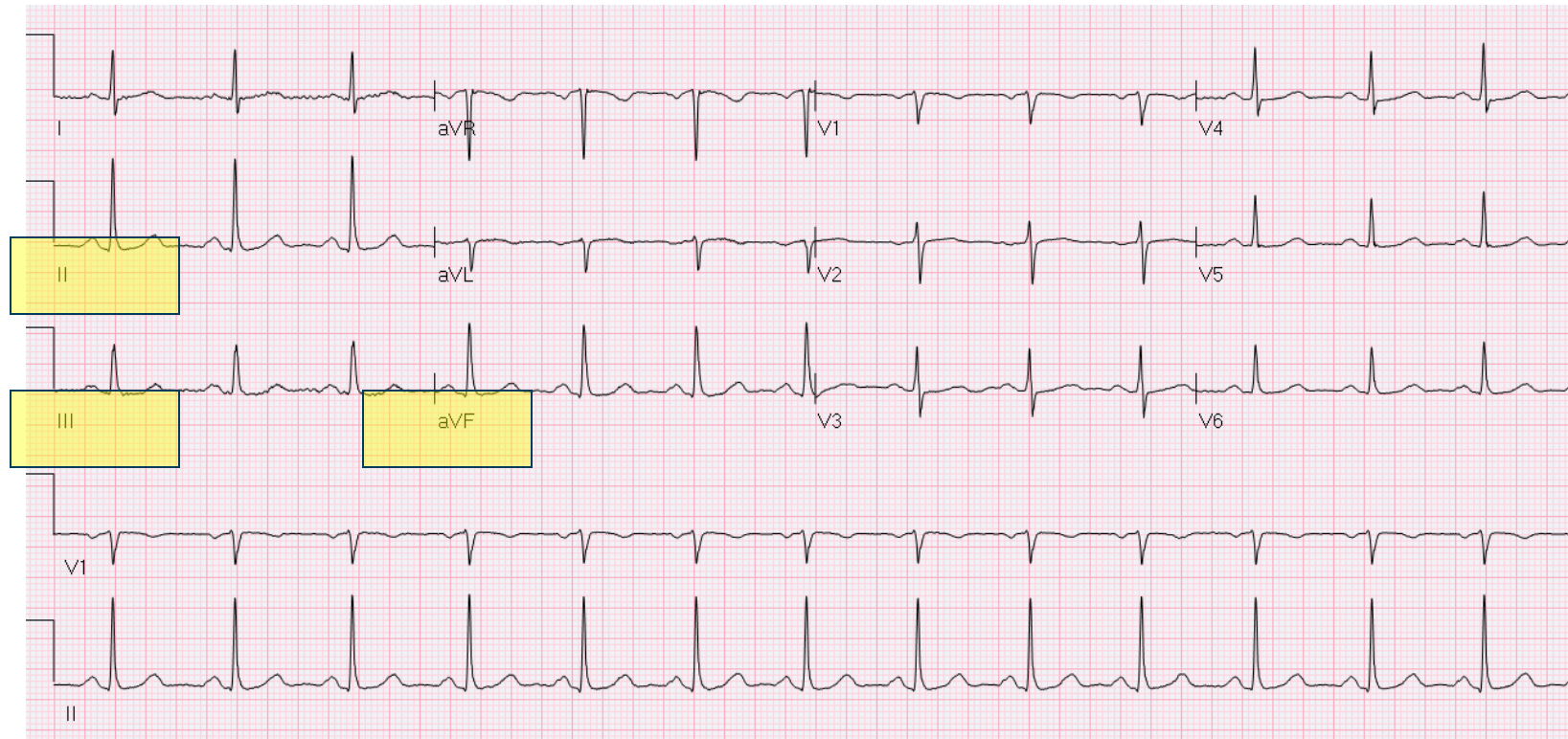
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Leads go together

ST Elevations - Inferior

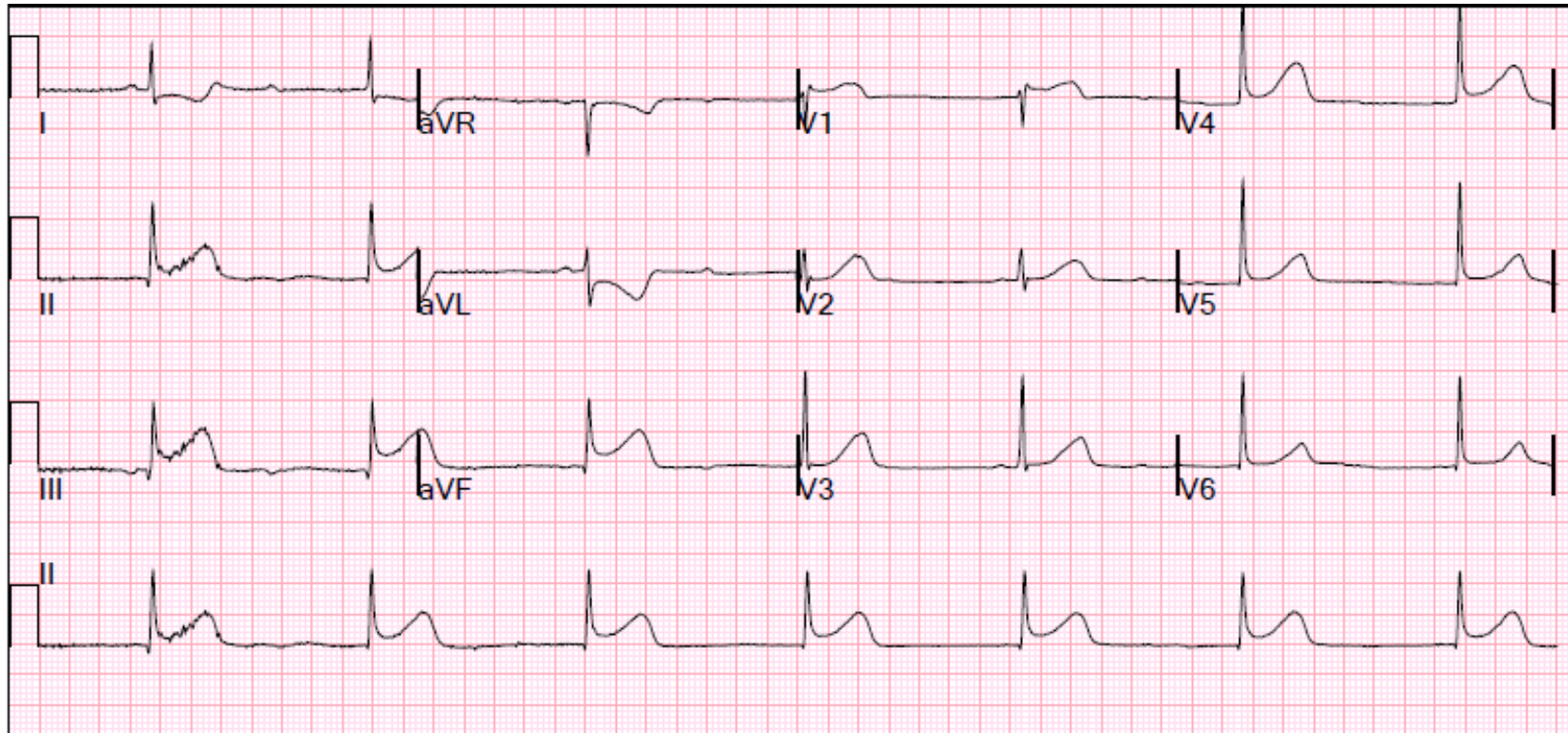
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Leads go together

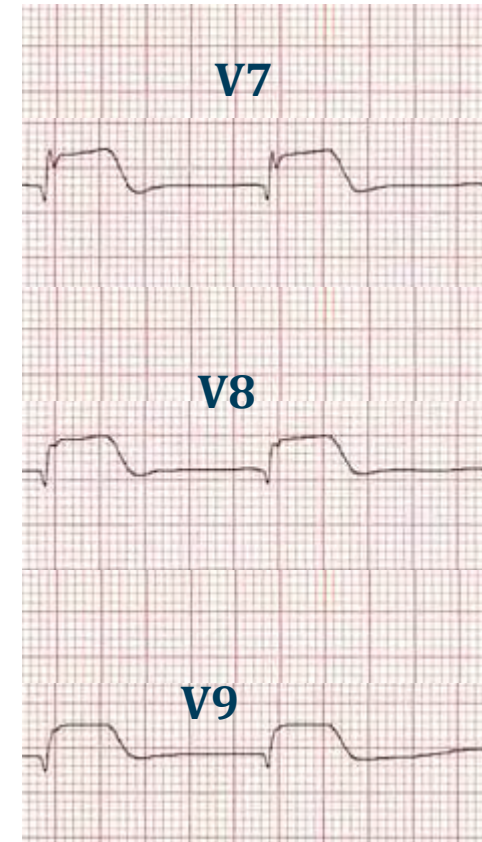
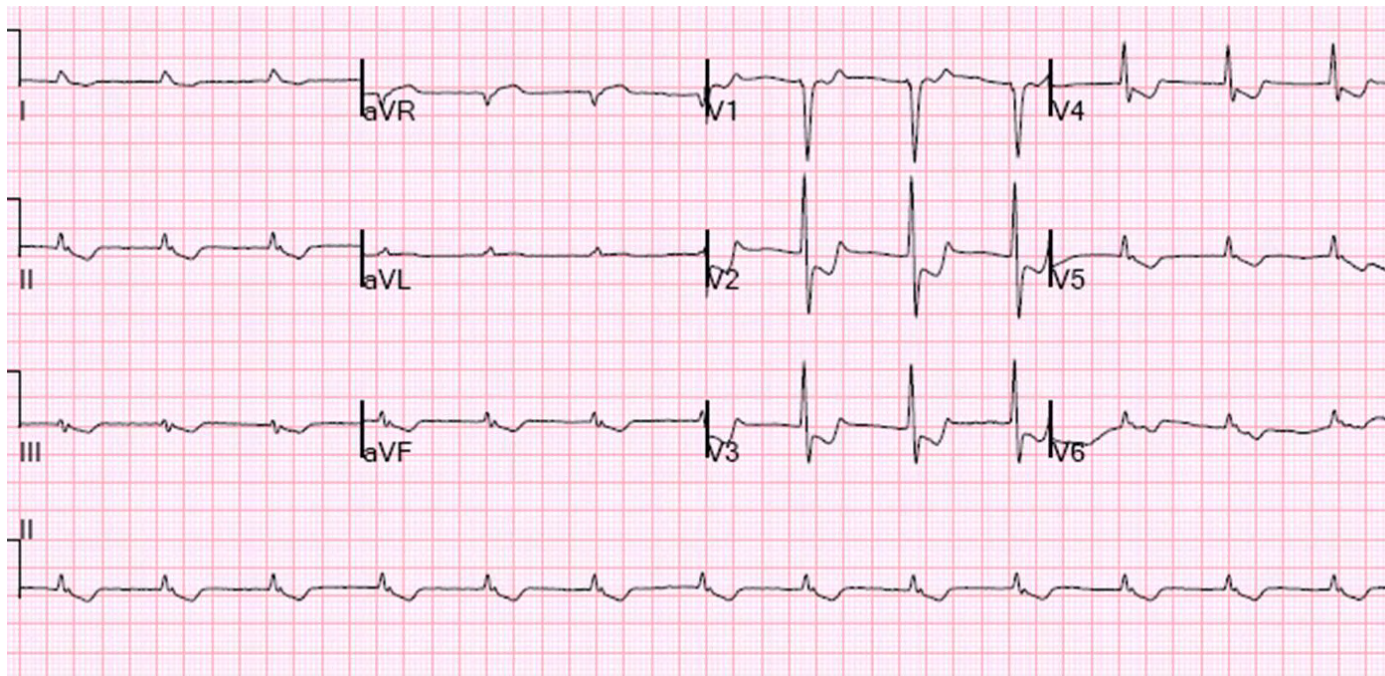
ST Elevations - Inferior

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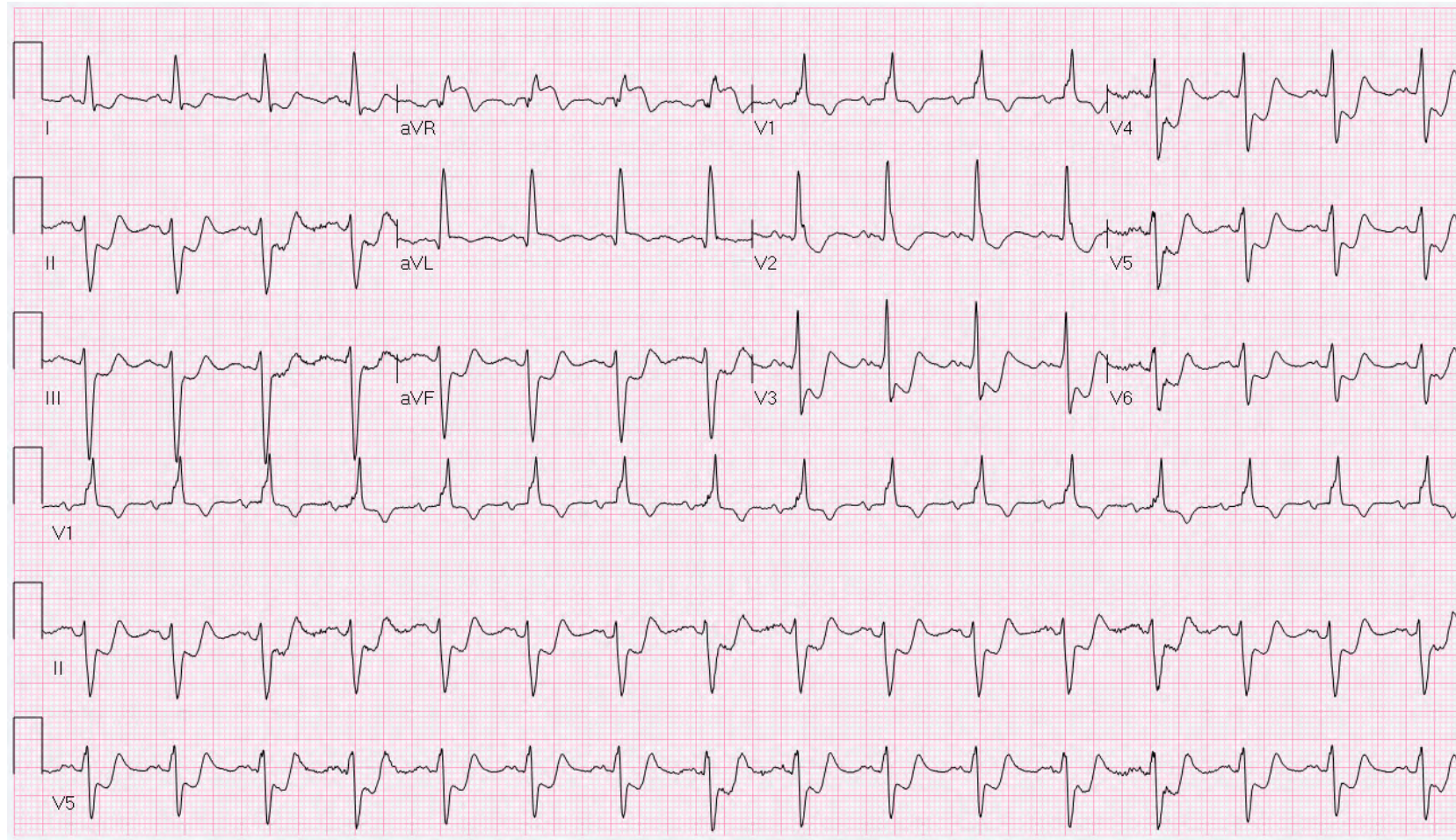
Posterior STEMI www.afratafreeh.com

- Anterior ST depressions with standard leads
- ST-elevation in **posterior leads (V7-V9)**



Left Main Occlusion

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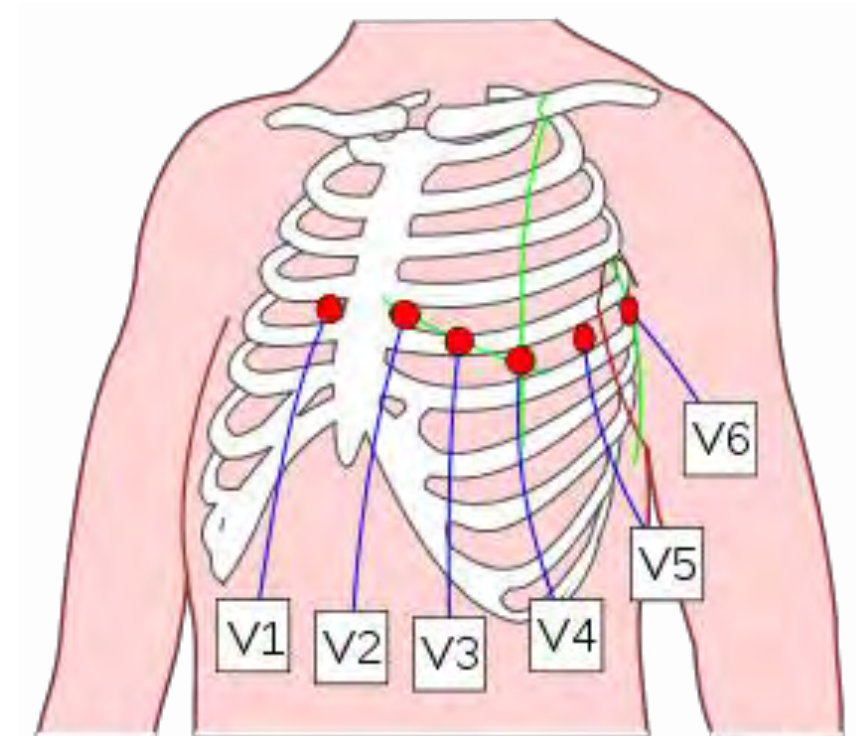


Special Complications

Inferior MI

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- Right ventricular infarction
 - Occurs in inferior STEMI (II, III, aVF)
 - Loss of right ventricular contractility
 - **Elevated jugular venous pressure**
 - Decreased preload to left ventricle → **hypotension**
 - Diagnosis: right-sided chest leads
 - Avoid nitroglycerine
 - Treatment: IV fluids (↑ preload)

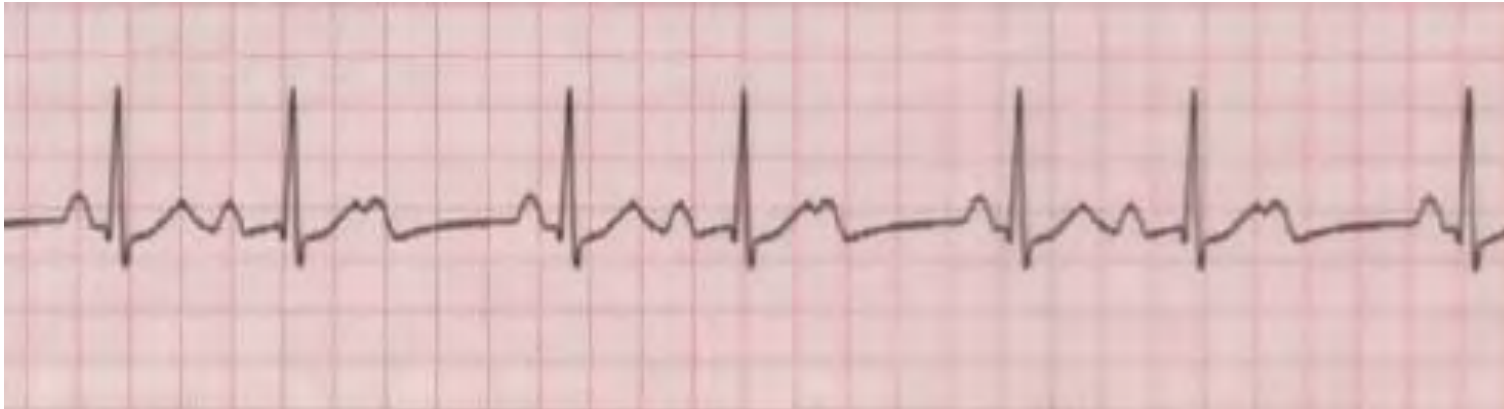


Special Complications

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Inferior MI

- Sinus bradycardia and heart block
 - Inferior wall ischemia
 - RCA: SA node 60%, AV node 90%



Special Complications

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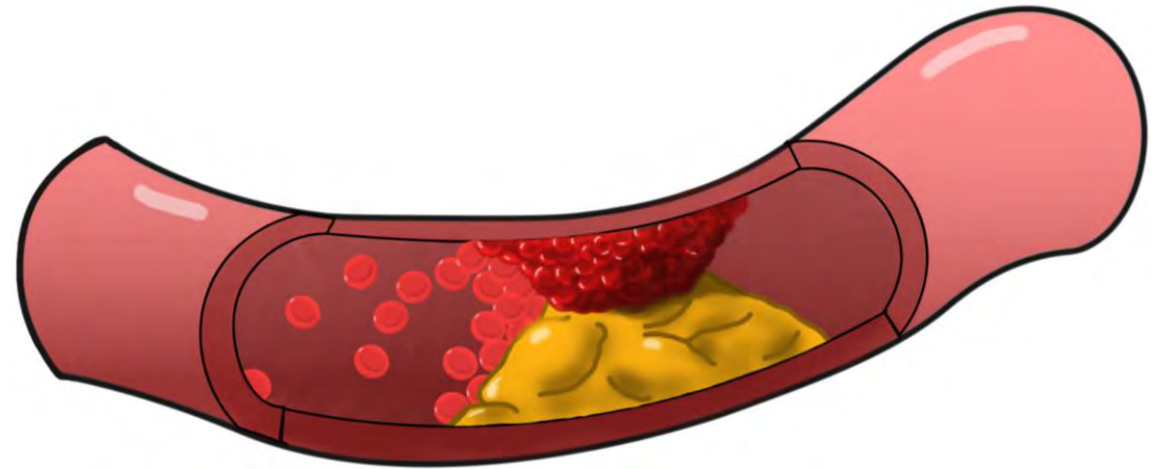
Anterior MI

- **Cardiogenic shock**
 - Usually occurs with large anterior STEMIs
- Hypotension
- Tachycardia
- Pulmonary edema
- Respiratory distress

Treatment of STEMI

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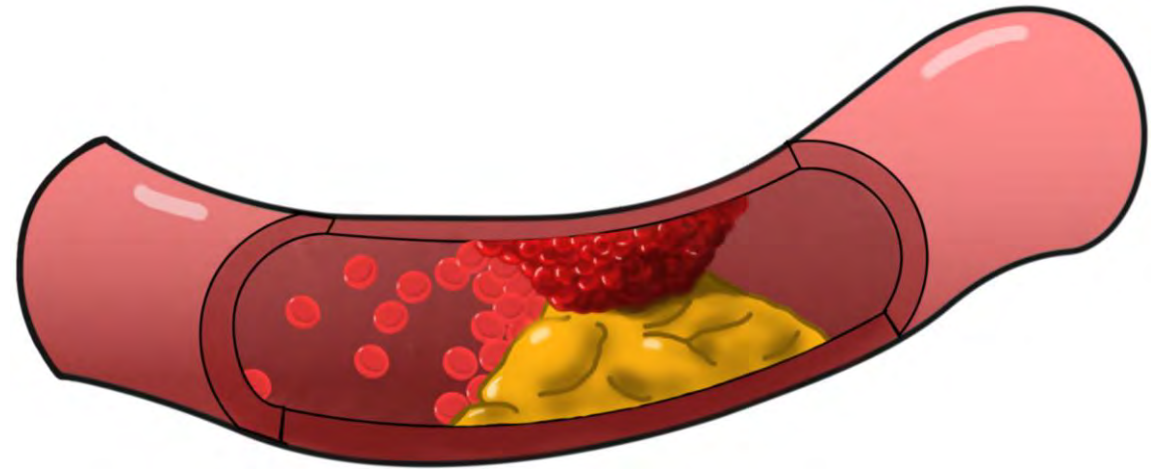
- “Time is muscle”
- Coronary artery occluded by thrombus
- Longer occlusion → more muscle dies
 - More likely the patient may die
 - More heart failure symptoms
 - More future hospitalization for heart disease
- **Medical emergency**



Treatment of STEMI

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- Main objective is to open the artery
 - “Revascularization”
- Option 1: Emergency angioplasty
 - Mechanical opening of artery
 - Stent placement
 - Should be done < 90min
- Option 2: Thrombolysis
 - Alteplase, TPA
 - Should be done < 30min
- “Door to balloon” or “door to needle”



Treatment of STEMI

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- Time matters
 - Medical therapy is supportive
 - Given while working to open artery
- This is a **thrombotic** problem
 - Aspirin to inhibit platelet aggregation
 - Heparin to inhibit clot formation
- This is also an **ischemic** problem
 - Beta-blockers to reduce O2 demand
 - Nitrates to reduce O2 demand

Cautions

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- Beta-blockers
 - Inferior MI
 - **Bradycardia and AV block** can develop
- Nitrates
 - Occlusion of RCA can cause **RV infarct**
 - RV infarction → ↓ preload
 - Nitrates ↓ preload → **hypotension**

Other STEMI Treatments

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- Clopidogrel
 - ADP receptor blocker
 - Inhibits platelets
- Eptifibatide
 - IIB/IIIA receptor blocker
 - Inhibits platelets
- Bivalirudin
 - Direct thrombin inhibitor
 - Inhibits clot formation

Typical STEMI Course

www.afratafreeh.com

- Arrival in ER with chest pain 5:42 pm
- EKG done 5:50 pm
 - STEMI identified
- Cardiac cath lab activated for emergent angioplasty
- Meds given in ER
 - Aspirin
 - Metoprolol
 - Nitro drip
 - Heparin bolus
 - Transport to cath lab 6:15 pm
- Artery opened with balloon 6:42 pm
 - DTB time 60 minutes (ideal < 90min)

Typical STEMI Course

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- Arrival in ER with chest pain 5:42 pm
- EKG done 5:54 pm
 - STEMI identified
- Meds given in ER
 - Aspirin
 - Metoprolol
 - Nitro drip
 - Heparin bolus
- tPA given based on weight 6:07 pm
 - IV push
 - Door to needle time 25 min (ideal < 30)

Complications of Ischemia

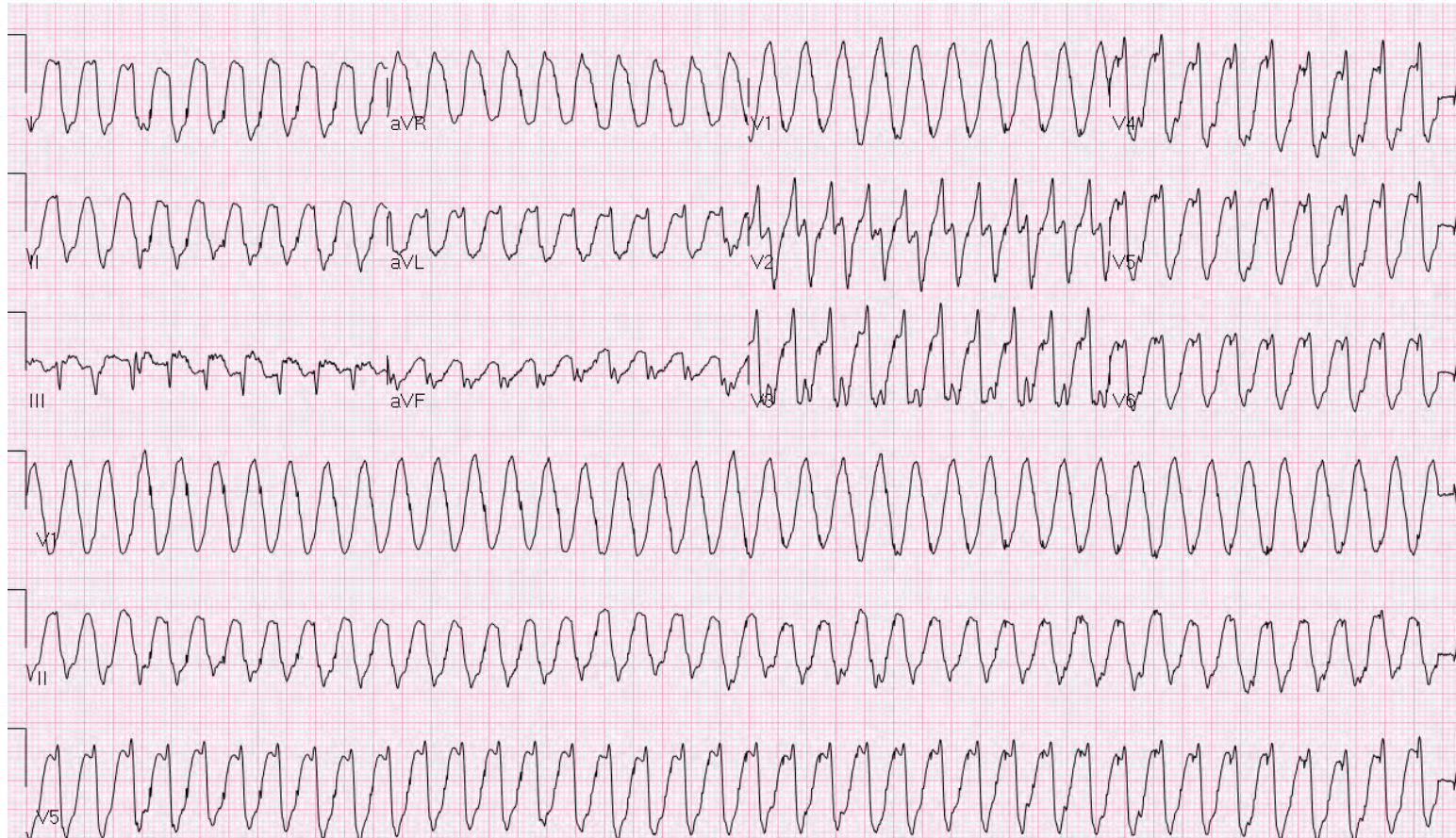
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- First 4 days
 - Arrhythmia
- 5 – 10 days
 - Free wall rupture
 - Tamponade
 - Papillary muscle rupture
 - VSD (septal rupture)
- Weeks later
 - Dressler's syndrome
 - Aneurysm
 - LV Thrombus/CVA

Cause of Death

0 – 4 days after MI

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Sudden Cardiac Death

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- Most common cause among older patients: **myocardial infarction**
- Ventricular tachycardia → ventricular fibrillation → cardiac arrest



Cause of Death

5-10 days after MI

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Best Test:

Transthoracic Echocardiogram

- **Free wall rupture**
 - Usually fatal – sudden death
 - May lead to tamponade
- **Papillary muscle rupture**
 - Acute mitral regurgitation (holosystolic murmur)
 - Heart failure, respiratory distress
 - More common inferior MIs
- **Septal rupture – VSD**
 - Loud, holosystolic murmur (thrill)
 - Hypotension, right heart failure (↑ JVP, edema)



Ventricular Aneurysm

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Weeks to months after MI

- More common **anterior infarction**
- Risk of thrombus → stroke, peripheral embolism
- Causes persistent ST elevations

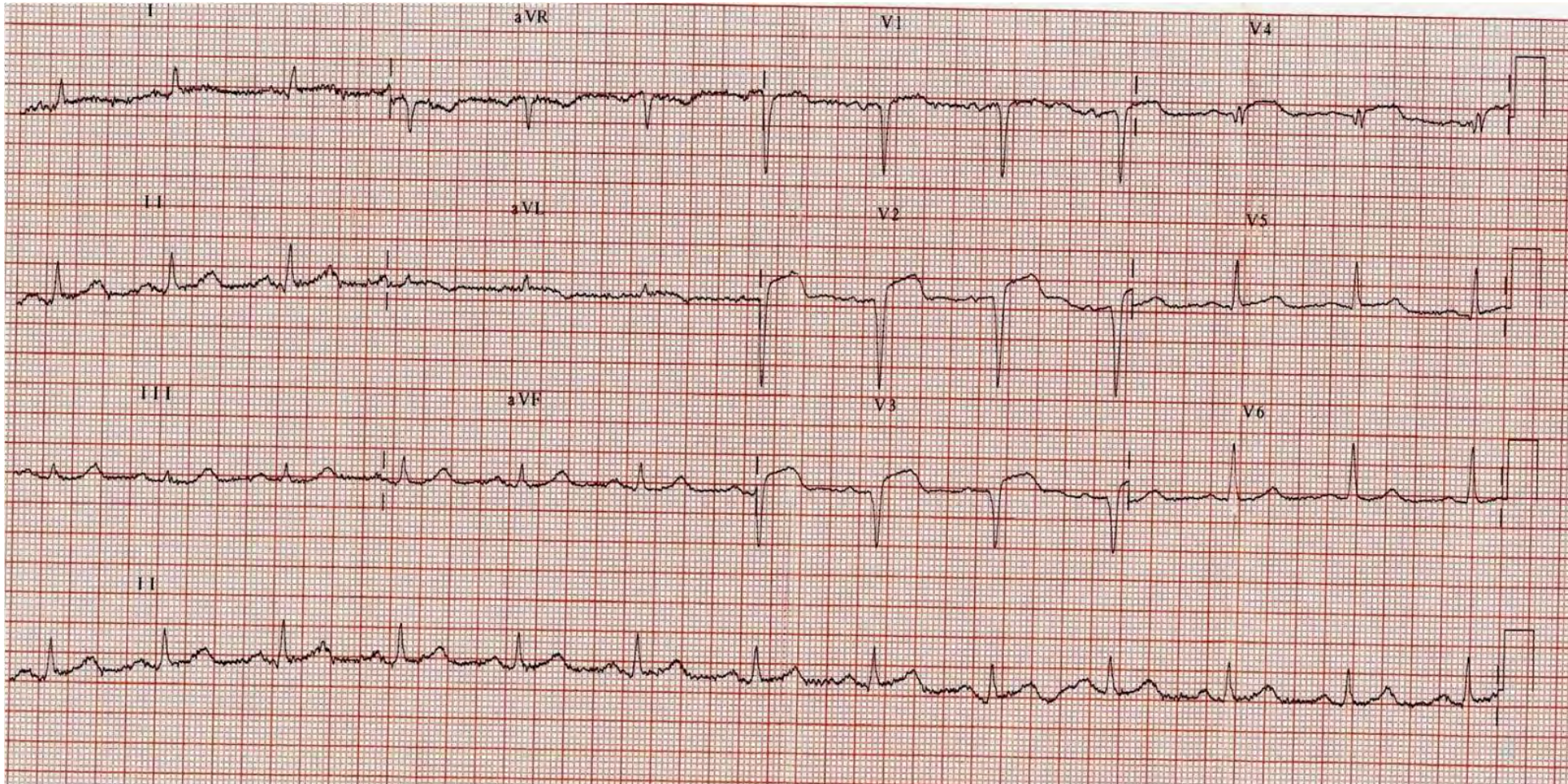


Patrick J. Lynch, medical illustrator/Wikipedia

Ventricular Aneurysm

www.afratafreeh.com

Weeks to months after MI



Ventricular Pseudoaneurysm

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1 to 2 weeks after MI

- **Rupture contained by pericardium/scar tissue**
- Not a true aneurysm
 - No endocardium or myocardium
- May rupture
- Presents as chest pain or dyspnea
- Often seen in the **inferior wall**
- Occurs earlier (< 2 weeks) than true aneurysm

Dressler's Syndrome

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Weeks to months after MI

- Form of pericarditis
 - Chest pain
 - Friction rub
- Immune-mediated (details not known)
- Diagnosis: clinical
- Treatment: NSAIDs or steroids

Fibrinous Pericarditis

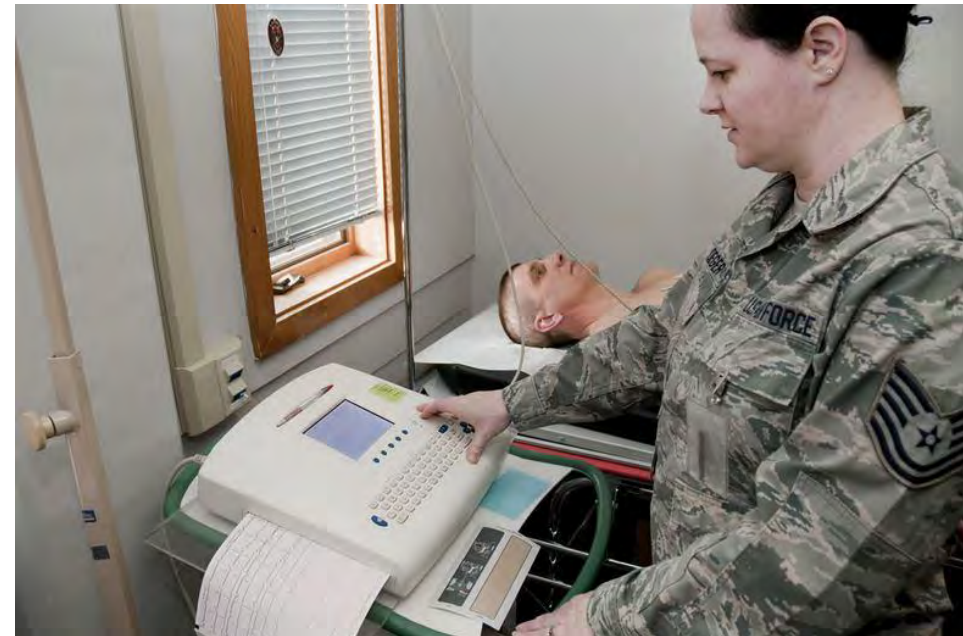
www.afratafreeh.com

- Occurs *days* after MI
 - Sometimes called “post-MI” pericarditis
 - Not autoimmune
 - Extension of myocardial inflammation
- Dressler’s occurs *weeks* after MI
 - Sometimes called “post cardiac injury” pericarditis
- Rarely life-threatening
- Diagnosis: clinical

Evaluation of Chest Pain

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- Must consider: STEMI, aortic dissection and pulmonary embolism
- Best first test: **EKG** to evaluate for STEMI
- Additional testing considerations:
 - CT scan (PE or dissection)
 - Cardiac biomarkers (NSTEMI)
 - D-dimer



Public Domain

Heart Failure I

Jason Ryan, MD, MPH

* aortic regurgite and stenosis → most common cause of them depends on age

calcification ← $>60y$ ←

in stenosis ← tricuspid aortic valve ← Congenital ← $<60y$ ←

in regurgite ← bicuspid aortic valve ←

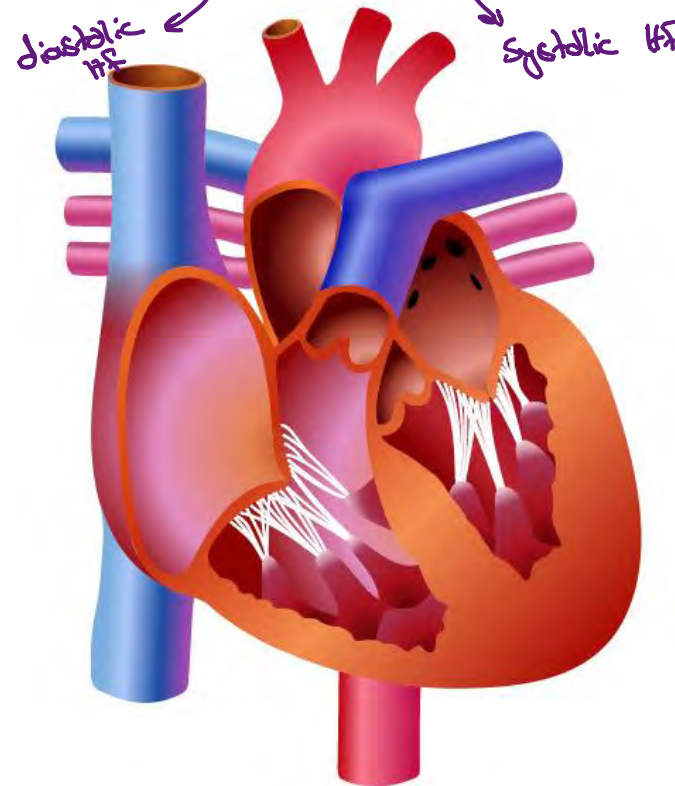
Heart Failure

Definition

- Clinical syndrome
- Impaired ability of left ventricle to fill or eject blood

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→ ^{lung} Lt side HF → Symptoms: dyspnea, orthopnea, PND, night cough
→ Rt side HF → Symptoms: up → ↑ JVP
down → not neg liver, ascites, cardiac cirrhosis, edema "bilateral"



* Congestive heart failure
↳ Rt + Lt HF

Heart Failure

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Types

Heart failure with reduced ejection fraction

م < 45

ischemia 50% "previous MI"
valvular heart disease
cardiomyopathy

• HFrEF

• Systolic heart failure

مشكلة في طرد الدم الى خارج

• Problem ejecting blood from left ventricle

• Usually caused by a dilated cardiomyopathy

Heart failure with preserved ejection fraction

• HFpEF

• Diastolic heart failure or diastolic dysfunction

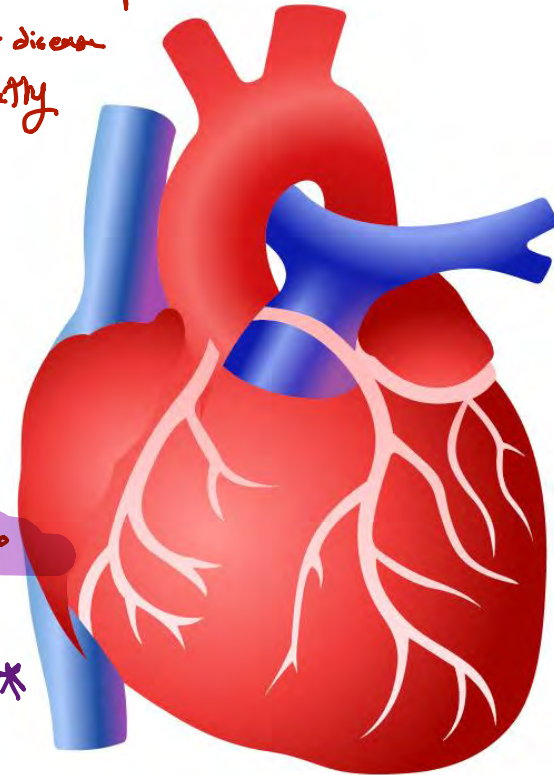
• Problem filling left ventricle with blood

↳ loss of lusitropy = loss of compliance

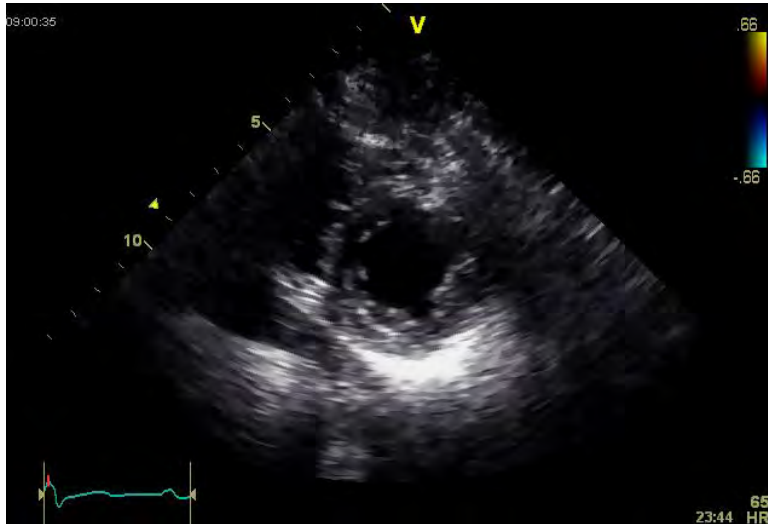
مشكلة في استقبال الدم

normal ejection fraction 50-65%

* في احواله HF لكنه normal ejection fraction
بتصغير في الكفاءة نتيجة
long standing HTN, diabetes

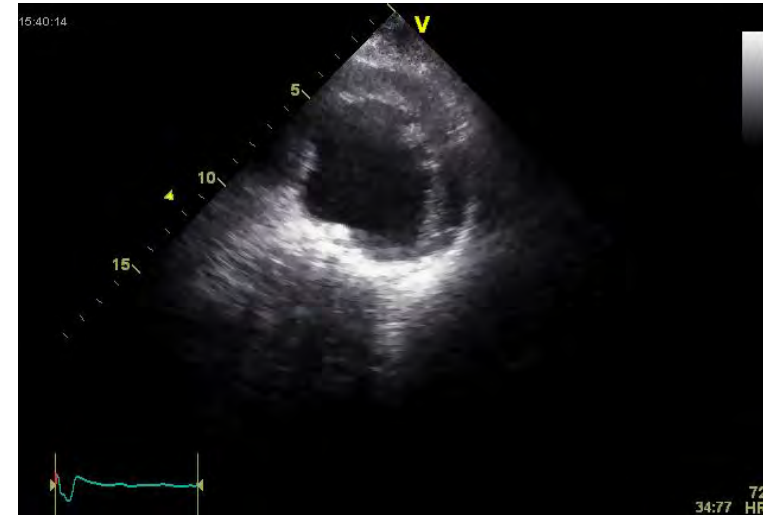


Diastolic Heart Failure



EF is normal (55-65%)

Systolic Heart Failure



Ejection fraction is reduced

Clinical **بشرح**

Volume Status

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* Euvolemic

* Hypervolemic

- Volume overloaded
- Wet

* Hypovolemic

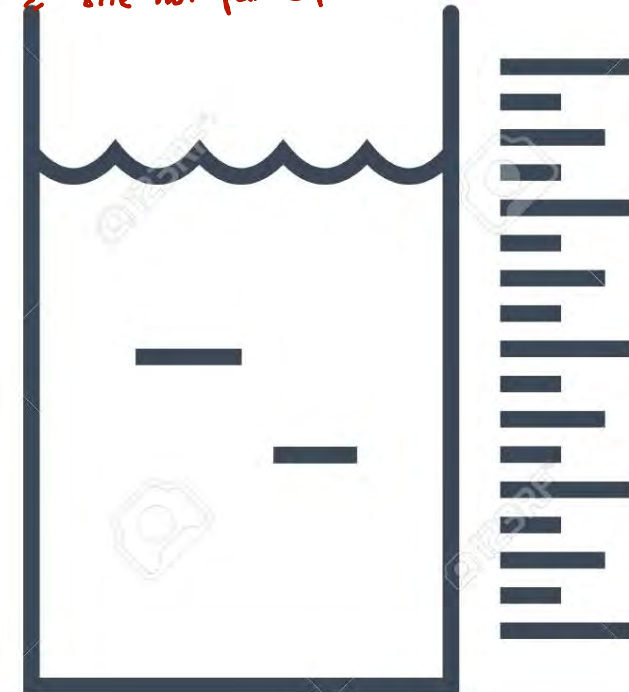
- Volume depleted
- Dry
- Most heart failure symptoms from **volume overload**

→ 3rd space loss
→ LL edema
→ crackles in lungs
→ ↑ JVP
→ ascites and pleural effusion

normal
20cc
50cc
100cc
X-ray, Clinical signs
↓
~ 500cc
~ 3000cc
تقریر

1st space → fluid in cells
2nd " → " = interstitial
3rd " → " = site not participate in circulation

ascites
sepsis
burn
pancreatitis
cavities
Pleural effusion
pericardial
peritoneal



X-ray → anteroposterior
→ lateral

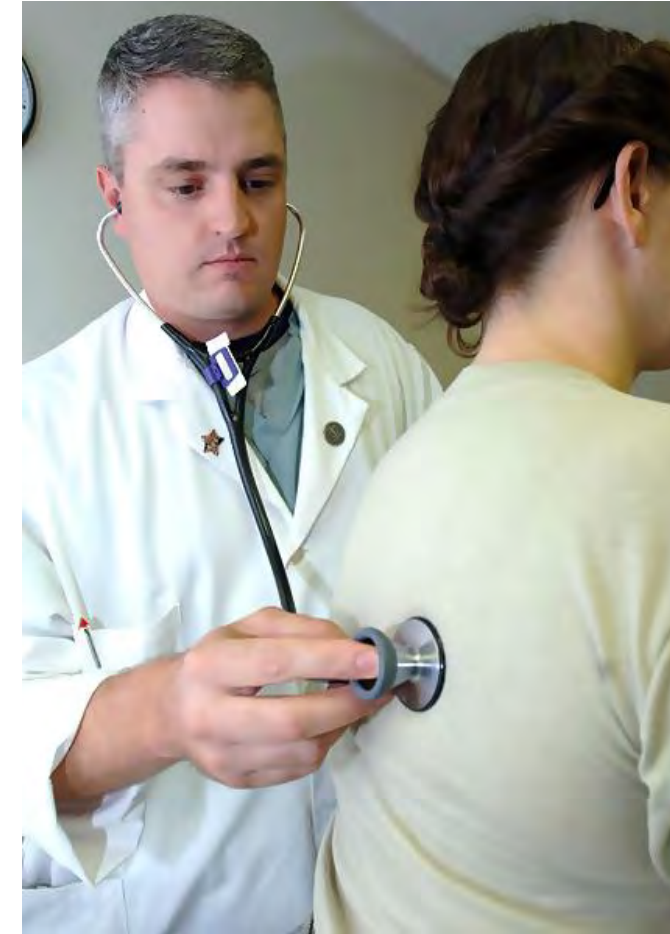
$$JVP = \underset{\substack{\rightarrow 5 \\ \text{Rt atrial pressure}}}{\text{Rt atrial pressure}} + \underset{\substack{\rightarrow 1 \\ \text{Central veins pressure}}}{\text{Central veins pressure}} = 6-8$$

Volume Status

www.afratafreeh.com

- Assessed by physical exam
 - Rales, edema, jugular venous pressure
- Unrelated to ejection fraction**
 - LVEF of 20% can be dry → severe systolic HF
 - LVEF of 70% can be wet → has edema "HFrEF"

hyperolemia wet patient over loaded



Wikipedia/Public Domain

Volume Overload Symptoms

Normal

clinic

- Orthopnea → when lying flat
- Paroxysmal nocturnal dyspnea → يعني مع النوم خفوفه
- Classic finding is rales خرخرة
 - Fluid filled alveoli "pop" open on inspiration
- Chest X-ray shows congestion
 - ↳ pulmonary edema



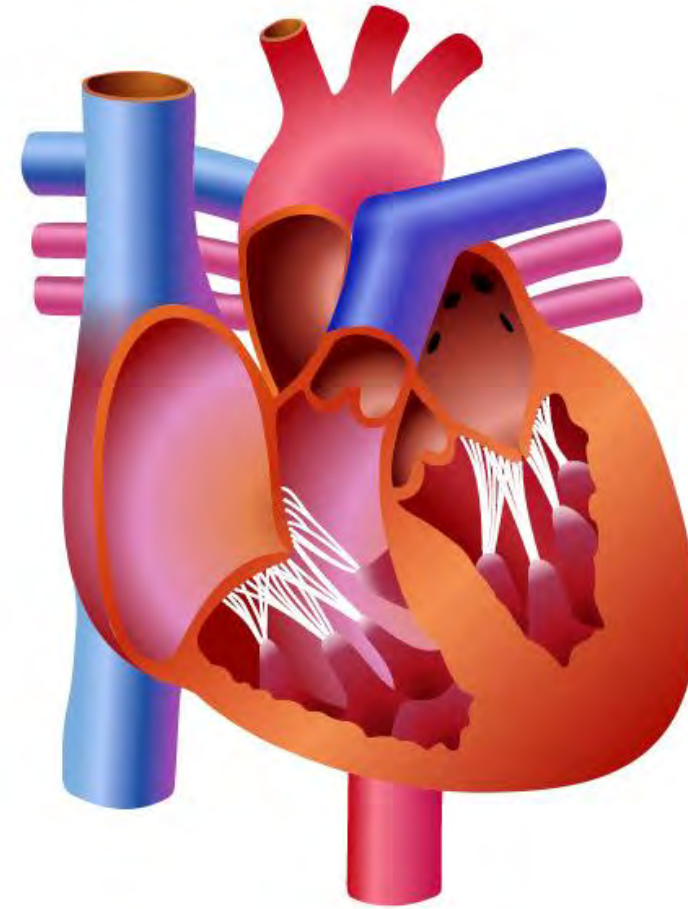
- ① Bat wing appearance
- ② pleural effusion $\left\{ \begin{array}{l} \rightarrow \text{transudative} \rightarrow \text{MC cause HF} \\ \rightarrow \text{exudative} \rightarrow \text{MC cause infection} \end{array} \right.$
- ③ Kerley B line $\rightarrow$ peribronchovascular thickening
- ④ Pulmonic cephalization
- ⑤ Cardiomegaly

Increased Pressures

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- * Increased left atrial pressure
- * Pulmonary capillary wedge pressure
- * Increased pulmonary artery pressure $\uparrow$ μ PAP
- * Increased right atrial pressure

μ PAP = 93 mmHg = $80 + \frac{1}{3} (120 - 80)$
 = $D + \frac{1}{3} \text{ pulse pressure [systolic-diastolic]}$
 μ PAP $> 25 \text{ mmHg}$ at rest or $> 30 \text{ mmHg}$ at exercise
 is class



NYHA Classification

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Class	Symptoms
I	Asymptomatic
II	<u>Symptoms with moderate exertion</u>
III	<u>Symptoms with activates of daily living</u>
IV	<u>Symptoms at rest</u>

has history of heart disease

دوبو، راح عالمام

① S3 Gallop

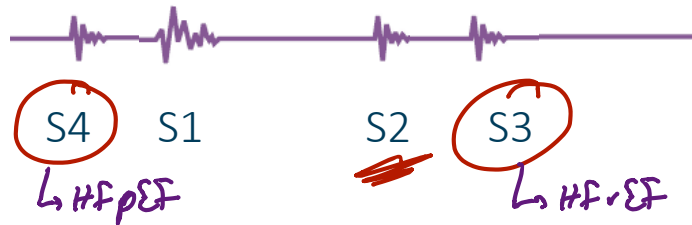
Signs/Symptoms

- Classic finding associated with **increased LA pressure**
- Indicates volume overload state
- Normal finding in children pregnant women

murmurs {
→ Diastolic → always pathologic ⇒ S₄
→ Systolic → pathologic
→ physiologic

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⇒ Systolic HF



Heart Failure

Signs/Symptoms

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a part of precordial examination

② Elevated **jugular venous pressure** (normal 6-8cmH₂O)

- Look for height of double bounce

• **Hepatojugular reflux** →

JVP المرتفع كما أنه 45° يضغط على liver يزيـد JVP

→ normal increase JVP [3]

في كثير من صيغ الـ HF
Volume overload

→ between 2 sternocleidomastoid m.

→ above 1-3 of sternum → أكثر من صيغ حاد
المرتبعة صلي، راجلي

→ 6-8 mm Hg

→ CVP = 5 + JVP → ICU في كثير من الأحيان

سؤال جراحة
القلب

What is the best method to detect volume status
to patients in ICU?

Urine output → anuric
oliguria, normal

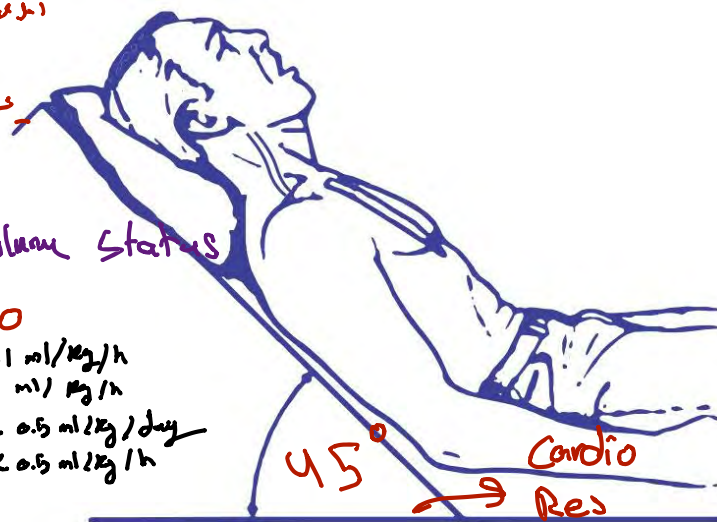
CVP in cardiac patients

* Normal UO

adult: 0.5-1 ml/kg/h

child: 1-2 ml/kg/h

- anuria < 0.5 ml/kg/day
- oliguria < 0.5 ml/kg/h



45°

Cardio
Res

15° → GI

Jugular

Carotid

between 2 sternocleidomastoid m.

seen not palpable

has 2 waves A, V

palpable

has 1 wave

Heart Failure

www.afratafreeh.com

Signs/Symptoms

③ Lower extremity pitting edema

- Increased capillary hydrostatic pressure
- Fluid leak from capillaries → tissues
- Gravity pulls fluid to lower extremities

سبب تورم في الساقين
Causes of edema → unilateral → local cause as DVT, Cellulitis, lymphedema
bilateral → Pitting → ↑ hydrostatic → HF R+ side
non pitting → ↓ oncotic → liver cirrhosis, nephrotic \$, malabsorption, malnutrition
↓ albumin



James Heilman, MD/Wikipedia

EKG and Echocardiogram

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- **EKG** *ECG*

* Exclude ischemia as precipitating cause

Q waves suggest prior infarction *old ische*
Q wave > 1/4 R wave → infarct Q wave < 1/4 R wave

* Evaluate for arrhythmia (atrial fibrillation)

- **Transthoracic echocardiogram** *→ ejection fraction and volumes ←*

- Used to distinguish **HFpEF** from **HFrEF** *→ ↓ Ejection*
- Critical to determine therapy

* اذا شنتف يني عني infarction old لكه اذا ما شنتف مکه يني عني inf old

Brain Natriuretic Peptide

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BNP

هرمونات
هرمونات

hormones

- ANP (atrial natriuretic peptide) released by atrial myocytes
- **BNP (brain natriuretic peptide)** released by ventricles → If high in HF patient is a poor prognosis
- Serum levels rise with volume/pressure overload → in HF
- Both counter effects of RAAS system
- BNP sometimes used for diagnosis in dyspnea
 - **Highly sensitive** for diagnosis of heart failure
 - High levels seen in most cases of heart failure but also many other disorders
 - Low levels strongly suggest other causes of dyspnea

HF with hyponatremia } الى بخونتي
HF with high BNP } في سريه
مستقل
MRCB
مستقل استانه
* HF patient - , which of the following is the worst prognostic factor of this patient?
اجاب → Hyponatremia

بغالب مريض هذه dyspnea مشكاه في clinical
بدى test لblood يفرسي اذا Cardiac & res ?
الاجاب → BNP
sensitive
↑
normal → HF
Cardiac → HF
res → pneumonia, COPD
normal في سريه

AUA → SLT
D-dimer → PE

RAAS

ANP/BNP

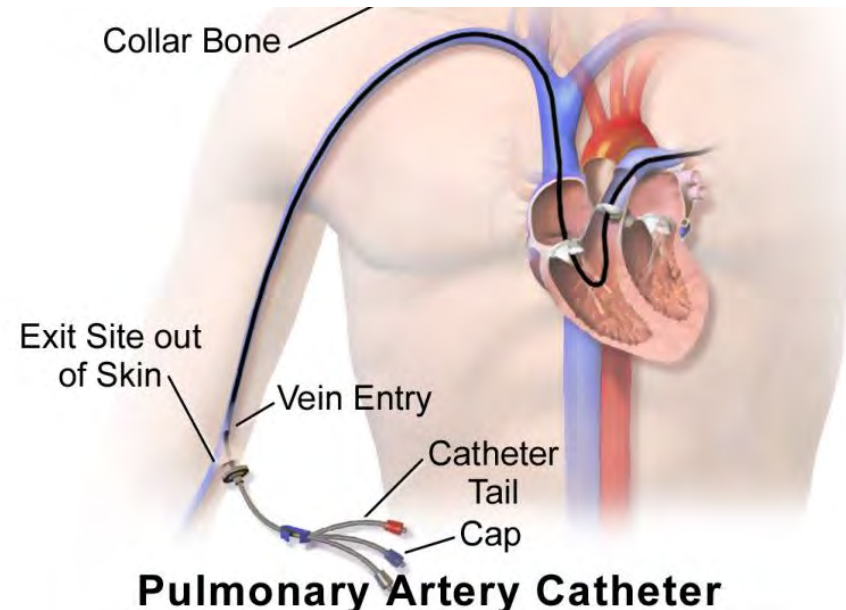
Right heart catheterization

Pulmonary Artery Catheterization

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Handwritten note: *Handwritten text in Arabic script, possibly indicating a source or context.*

- Increased PCWP = left heart congestion/failure
 - PCWP = pulmonary capillary wedge pressure
- Increased RA, RVEDP = right heart congestion/failure
 - ↓ RA atrial pressure ↓ RA ventricle in diastolic pressure



Pulmonary Artery Catheter

BruceBlaus

Heart Failure

www.afratafreeh.com

Diagnosis

- History
- Physical examination
- Chest X-ray
- Transthoracic echocardiogram
- EKG
- BNP level
- Right heart catheterization

Cor Pulmonale

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- Isolated **right heart failure**
 - Hypertrophy
 - Dilation
 - Decreased contractility
- Caused by long standing pulmonary hypertension
 - Lung disease (e.g., COPD)
 - Primary pulmonary hypertension



Cor Pulmonale

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- Dyspnea with **right heart failure**
 - Elevated jugular venous pressure
 - Lower extremity edema
 - Hepatomegaly
 - Absence of rales
- Treatment:
 - Usually aimed at underlying disease (COPD, etc.)



High Output Heart Failure

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- Exact mechanism unclear
 - Decreased LV filling time
- Defining characteristic: **HIGH cardiac output**
 - Heart failure symptoms in absence of low output
 - ↑JVP, pulmonary edema
- Heart in overdrive
 - Severe anemia
 - Thyroid disease
 - Thiamine (B1) vitamin deficiency (beriberi)
 - A-V fistulas



John Liu/Flickr

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Heart Failure II

Jason Ryan, MD, MPH



Heart Failure

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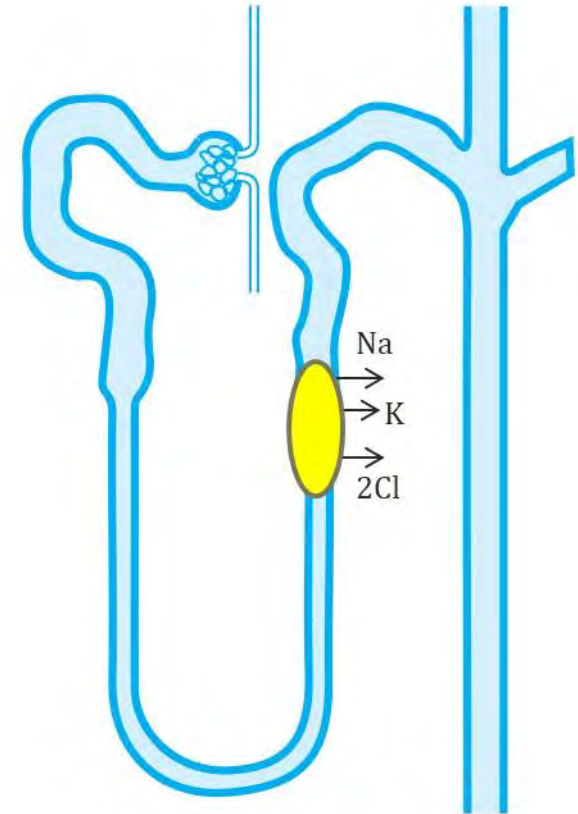
Treatment

- Acute heart failure
 - Volume overloaded
 - Goal: **improve symptoms** of dyspnea, pulmonary congestion
- Chronic heart failure
 - Euvolemic
 - Goal: **prevention** of hospitalizations, mortality

Acute Heart Failure

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- Goal: **reduce left atrial pressure and remove fluid**
 - Improves pulmonary edema
- Most commonly used drug class: **loop diuretics**
 - Furosemide, torsemide
 - Increased urine output
 - Reduce intracardiac pressures
 - Improve pulmonary edema and lower extremity edema
 - Main adverse effect: **hypokalemia**
 - Other adverse effects: acute renal failure, hypotension
- Also cause some venous dilation: ↓ left atrial pressure



Acute Heart Failure

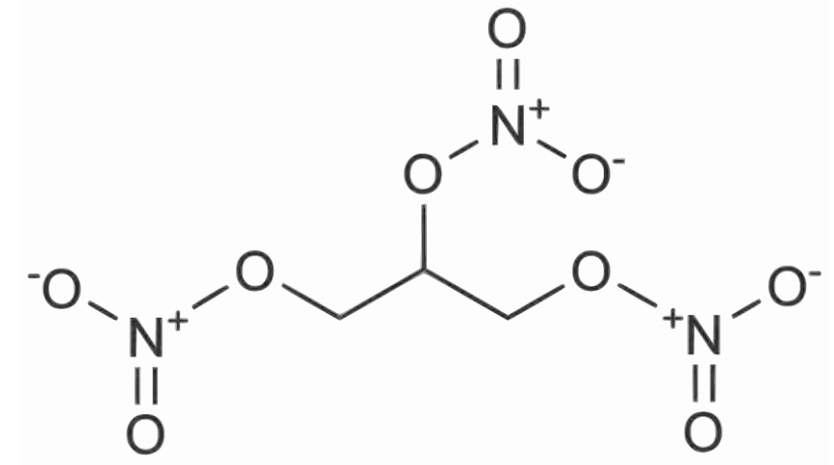
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- **Nitroglycerine**

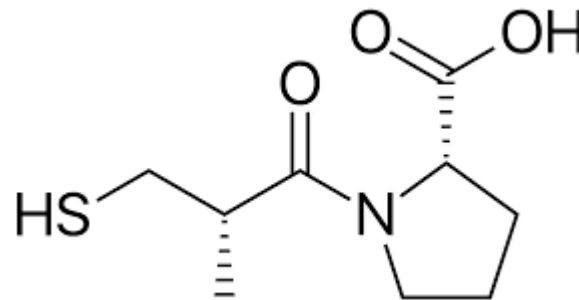
- Venous dilation → pool volume in venous system
- Reduces left atrial pressure
- Improves dyspnea

- **Afterload reducers**

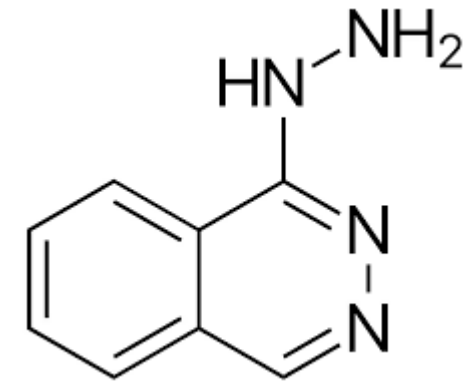
- Hydralazine, ACE-inhibitors
- Increased cardiac output → improve renal perfusion → diuresis



Nitroglycerine



Captopril



Hydralazine

Chronic Heart Failure

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- **HFpEF**

- No specific therapies
- Control blood pressure, blood sugar
- Chronic diuretics often used (furosemide)

- **HFrEF**

- Many specific therapies shown to reduce hospitalizations and mortality
- Treatments aimed at prevention of **cardiomyopathy progression**

Chronic Systolic Heart Failure

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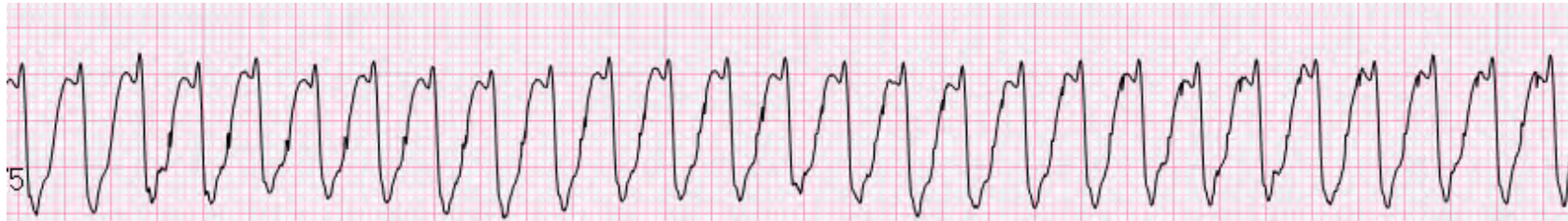
- Goal: reduce **hospitalizations and mortality**
- **Guideline directed medical treatment (GDMT)**
 - **Beta-blockers** (metoprolol, carvedilol, bisoprolol)
 - **ACE-inhibitors** (captopril, lisinopril)
 - **Aldosterone antagonists** (spironolactone, eplerenone)
 - **Neprilysin inhibitors** (sacubitril → increases ANP)
 - **Ivabradine** (inhibits SA node → ↓ heart rate)
 - **Nitrates/hydralazine** (combination therapy)
- Most drugs disrupt chronic activation of SNS and RAAS
- Prevent cardiac remodeling (progression of LV dysfunction)

ICD

Implantable Cardiac Defibrillator

www.afratafreeh.com

- Annual risk SCD > 20% some studies
- Most due to ventricular tachycardia



ICD

Implantable Cardiac Defibrillator

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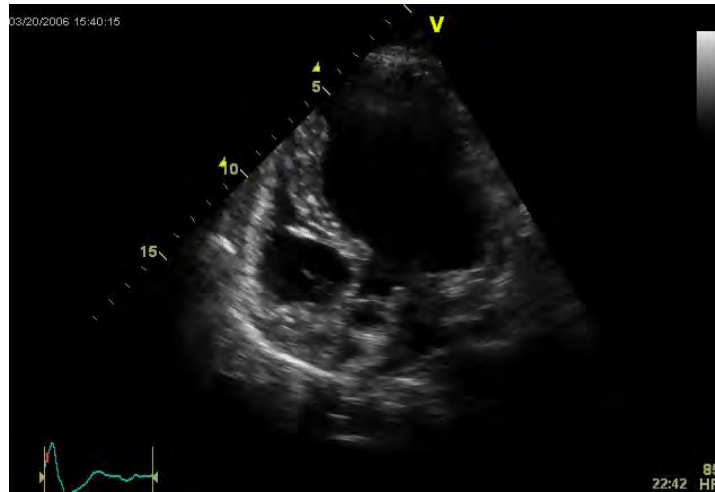
- Improve **mortality** in appropriate patients
- Indications:
 - Aborted sudden cardiac death
 - LVEF < 35%



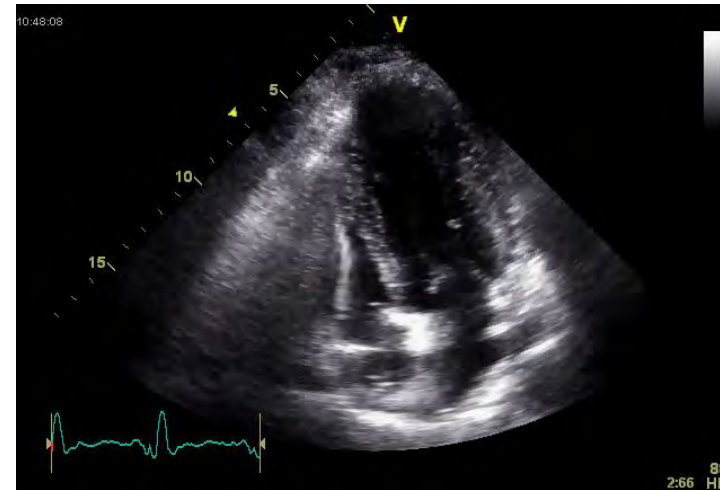
Biventricular Pacemakers

www.afratafreeh.com
Cardiac Resynchronization Therapy (CRT)

Out of Synch



After Pacemaker

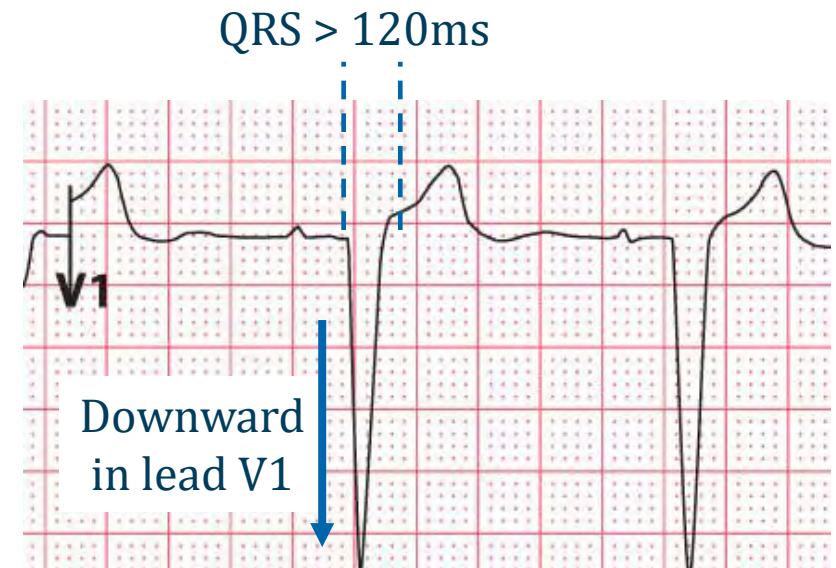
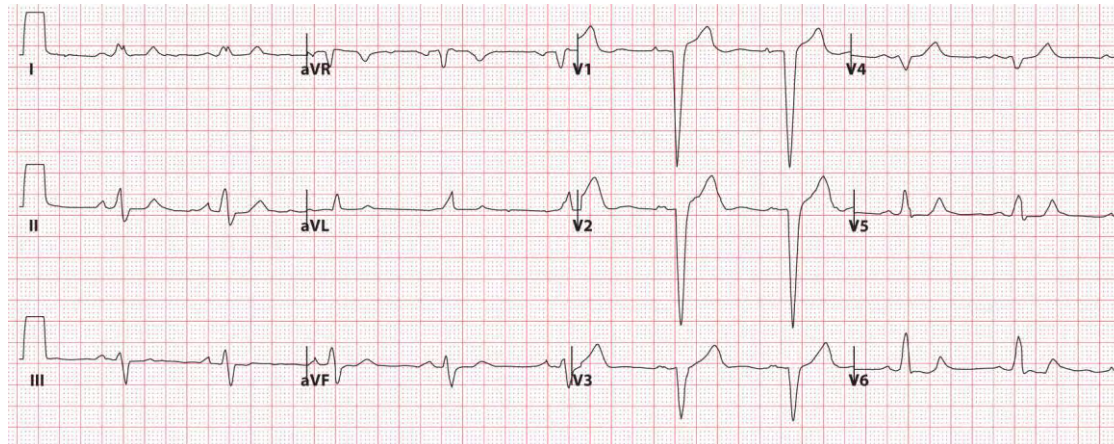


Biventricular Pacemakers

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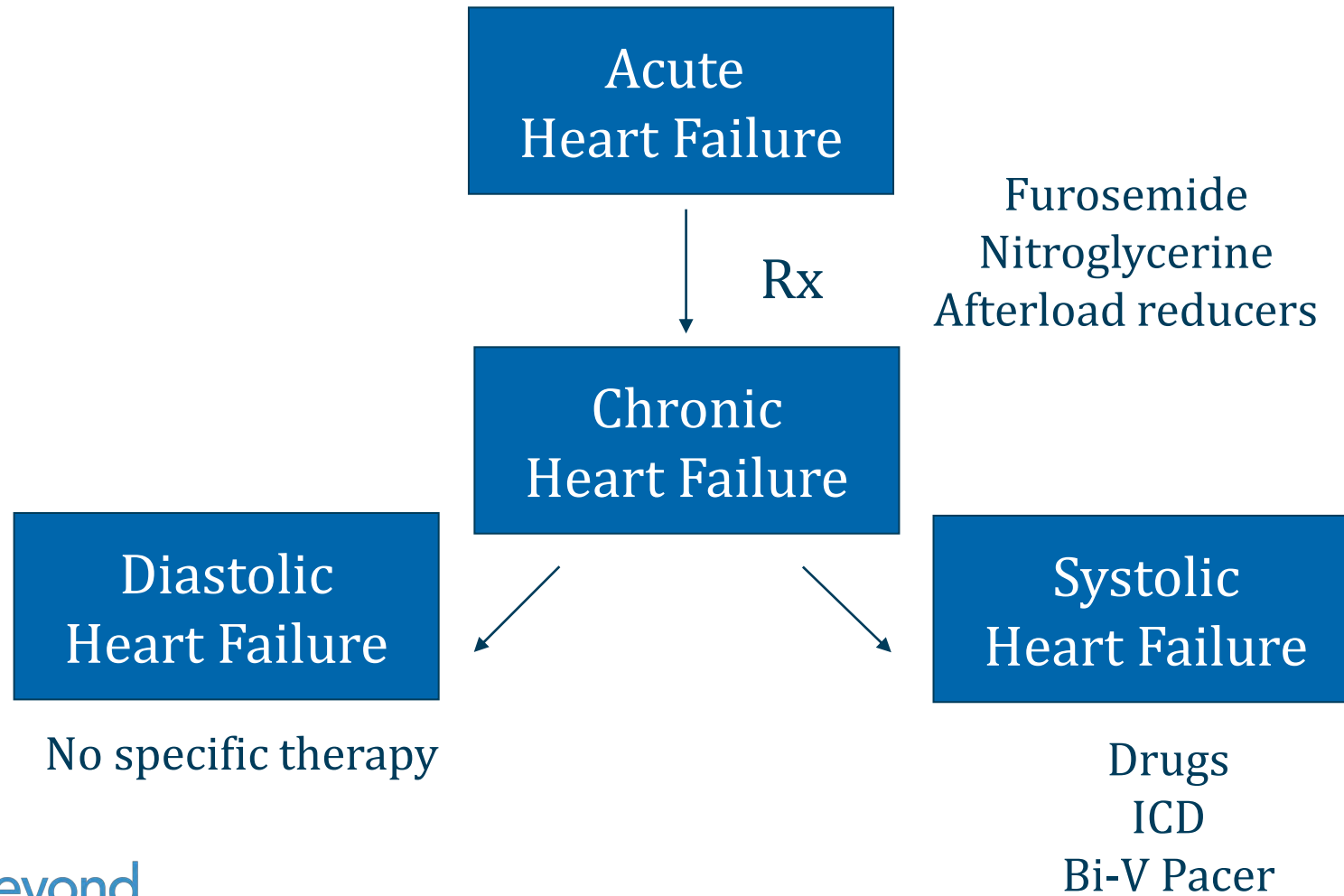
Cardiac Resynchronization Therapy (CRT)

- Improve **mortality** in appropriate patients
- Indications:
 - Left bundle branch block (wide QRS; $> 120\text{ms}$)
 - LVEF $< 35\%$



Heart Failure Treatment Pathway

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Advanced Heart Failure

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- Severe form of HFrEF
- Severely reduced cardiac output
 - Fatigue
 - Hypotension
 - Cool extremities
 - Confusion
 - Decreased appetite (cardiac cachexia)

Advanced Heart Failure

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Treatments

- **Inotropes (associated with ↑ mortality)**
 - Dobutamine
 - Milrinone
 - Digoxin (oral)
- Left ventricular assist devices (LVADs)
- Heart transplantation

Acute Exacerbations

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Causes

- #1: Dietary indiscretion
 - High salt intake
- #2: Poor medication compliance



Acute Exacerbations

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Causes

- Infection/trauma/surgery
 - Activation of sympathetic nervous system
- Ischemia (rare)
 - Decreased cardiac output
- Arrhythmias (A fib)
- NSAIDs
 - Inhibit cyclooxygenase (COX) → ↓ prostaglandins
 - Prostaglandins maintain renal perfusion
 - Result: Less renal perfusion → salt/water retention

Typical Acute Heart Failure Course

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- ER presentation:
 - Dyspnea, edema, sleeping in chair
- Admitted to hospital
 - Nitro drip to relieve dyspnea
 - IV Furosemide to remove fluid
- Hospital Day 2
 - Weight down 4 kg, feels better
 - Nitro drip stopped
 - Changed to oral furosemide
- Hospital Day 3: Discharge

More Complex Heart Failure Course

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- ER presentation:
 - Dyspnea, edema, sleeping in chair
 - Known LVEF 10%
- Admitted to hospital
 - Nitro drip to relieve dyspnea
 - IV Furosemide to remove fluid
- Hospital Day 2
 - Poor urine output, Cool extremities, Cr rises 1.1 → 1.4
 - Dobutamine drip started

More Complex Heart Failure Course

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- Hospital Day 3-5
 - Good urine output
 - Weight loss 4 kg
 - Breathing improves
- Hospital Day 6
 - Dobutamine stopped
 - Furosemide drip stopped
- Hospital Day 7
 - Oral furosemide given
- Hospital Day 8: Discharge

Noninvasive Ventilation

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- Positive pressure ventilation
- Administered through mask not ETT
- Used in acute respiratory failure
- Often effective for pulmonary edema
- Often avoids intubation
- Patient must be awake, alert, cooperative
- Classic case
 - HF patient in respiratory distress
 - Tachypnea, hypoxemia
 - NIV → improved respiratory status

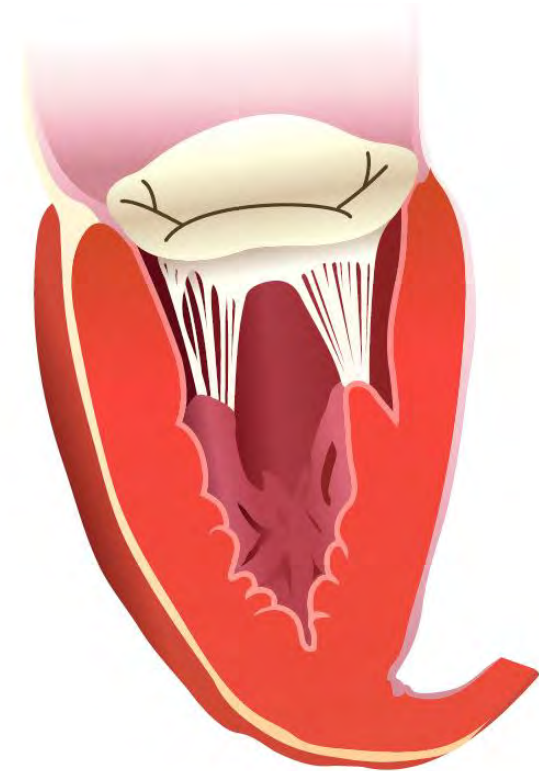


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Functional Mitral Regurgitation

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- Caused by left ventricular dilatation
- Mitral valve leaflets pulled apart
- Can cause holosystolic murmur at cardiac apex
- May resolve with diuresis



Hyponatremia

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- Occurs in severe heart failure
 - RAAS activation → ADH release → water retention
 - Poor renal perfusion → decreased water excretion
- **Poor prognostic indicator**

1 H			
3 Li	4 Be		
11 Na	12 Mg		
19 K	20 Ca	21 Sc	22 Ti
37 Rb	38 Sr	39 Y	40 Zr

Venous Stasis Edema

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- Common in the elderly
- Caused by poor venous drainage
- Unrelated to heart
 - No dyspnea
 - Normal lung exam
 - Normal jugular venous pressure
- Treatments:
 - Leg elevation
 - Compression stockings
 - Diuretics rarely work
- May lead to skin ulcers/infection



Drugs to Avoid in Heart Failure

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- Metformin
 - May cause lactic acidosis
- Thiazolidinediones (glitazones)
 - Pioglitazone, rosiglitazone
 - Cause fluid retention
- Calcium channel blockers
 - Negative inotropes
- NSAIDs
 - Cause fluid retention



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Cardiomyopathy

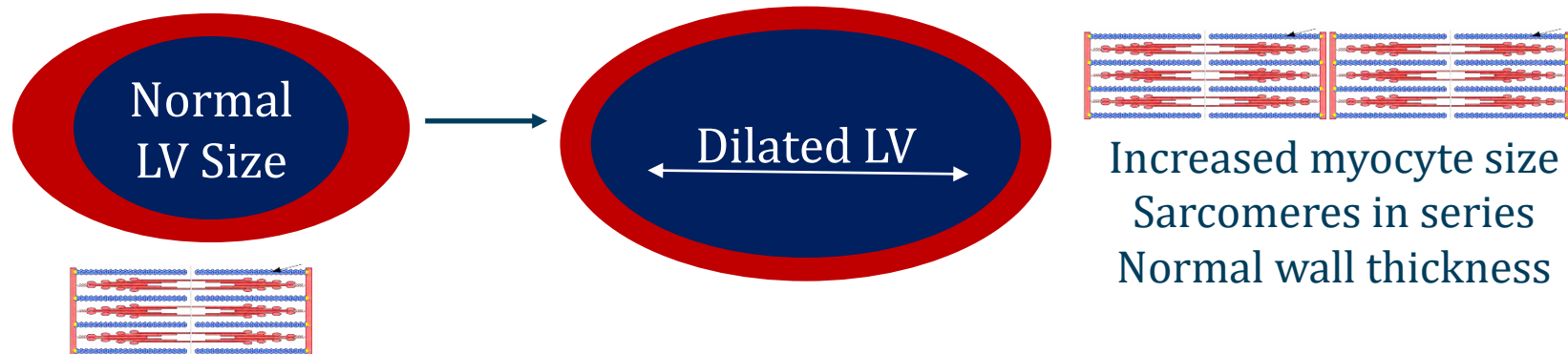
Jason Ryan, MD, MPH



Dilated Cardiomyopathy

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- Systolic heart failure with LV cavity dilation
- **“Eccentric” hypertrophy**
 - Volume overload (chronic retention of fluid in cavity)
 - Longer myocytes
 - Sarcomeres added in series

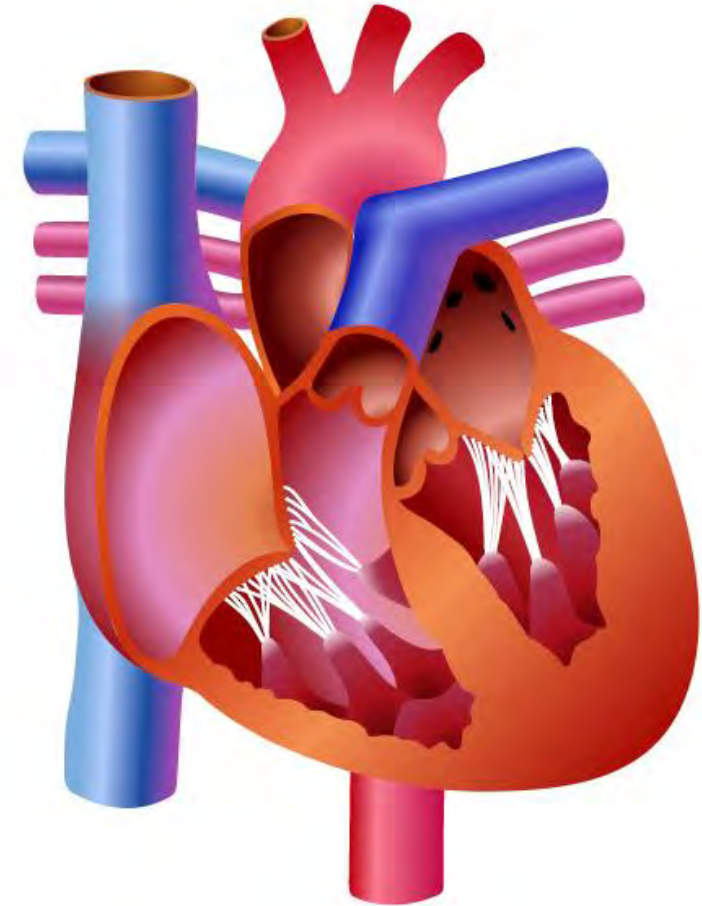


Dilated Cardiomyopathy

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Symptoms

- **Depend on volume status**
- Volume overload → acute systolic heart failure
 - Dyspnea
 - Pitting edema
- Severely reduced LVEF
 - Fatigue
 - Cachexia
 - Confusion



Dilated Cardiomyopathy

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- Diagnosis: **Transthoracic echocardiography**
- Treatment: **Guideline directed medical treatment (GDMT)**
 - Beta-blockers
 - ACE inhibitors
 - Aldosterone antagonists
 - Neprilysin inhibitors
- Defibrillators
- Bi-ventricular pacemakers



Ejection fraction is reduced

Dilated Cardiomyopathy

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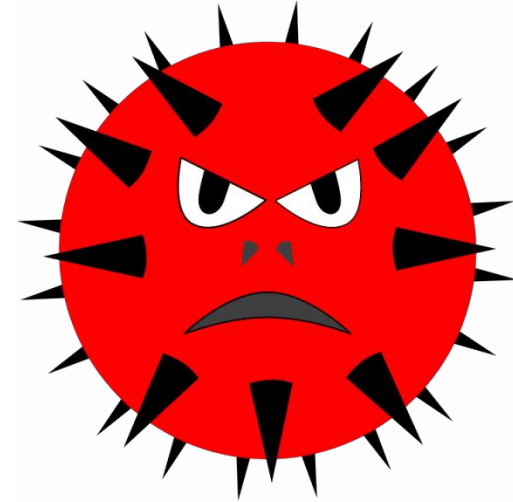
- Most common cause: **myocardial infarction**
 - Ischemic cardiomyopathy
 - Myocytes replaced by scar tissue
 - Focal hypokinesis (e.g., anterior wall)
- Many causes of **“non-ischemic” cardiomyopathy**
 - About 50% idiopathic
 - Many other causes
 - Global hypokinesis

Nonischemic Cardiomyopathy

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Viral

- May follow upper respiratory infection
- Many associated viruses
 - **Coxsackie**
 - Influenza, adenovirus, others
- No specific therapy for virus



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Nonischemic Cardiomyopathy

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Peripartum

- Late in pregnancy or early post-pregnancy
- Exact cause unknown (likely multifactorial)
- Women often advised to avoid future pregnancy



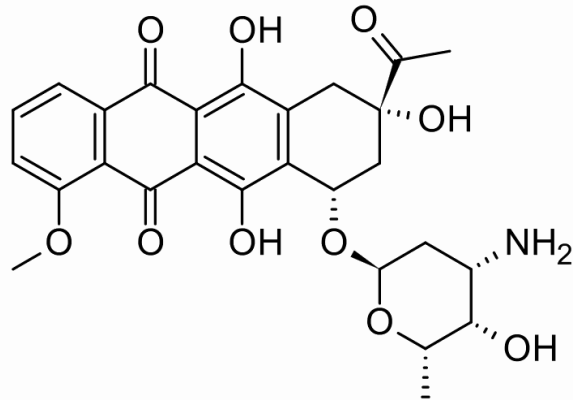
Øyvind Holmstad/Wikipedia

Nonischemic Cardiomyopathy

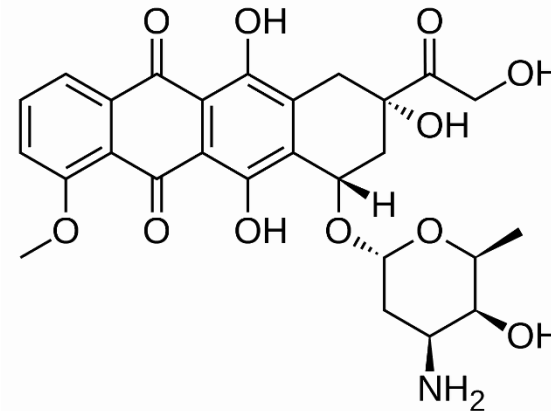
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Chemotherapy

- Usually after treatment with anthracyclines
 - Antitumor antibiotics
 - Doxorubicin and daunorubicin



Daunorubicin



Doxorubicin
(Adriamycin)

Nonischemic Cardiomyopathy

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Familial

- Mutations
 - Often sarcomere proteins
 - Beta-myosin heavy chain
 - Alpha-myosin heavy chain
 - Troponin
- Many autosomal dominant
- X-linked, autosomal recessive also described



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Nonischemic Cardiomyopathy

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Tachycardia-mediated

- Constant, rapid heart rate for weeks/months
- Leads to depression of LV systolic function
- **Reversible** with slower heart rate



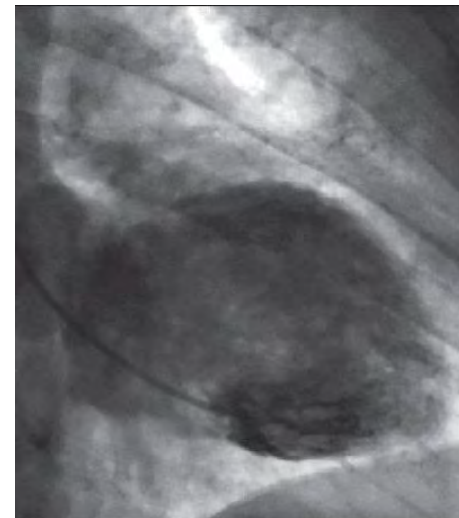
Nonischemic Cardiomyopathy

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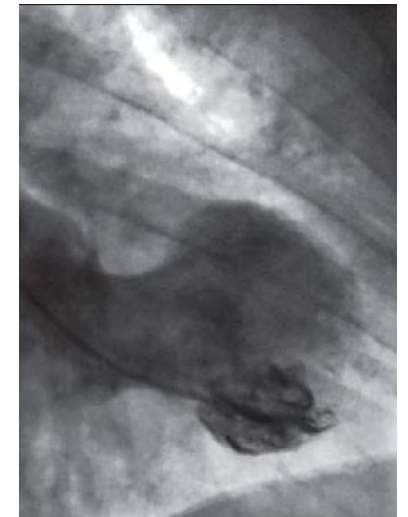
Takotsubo/Apical ballooning

- **Stress-induced** cardiomyopathy
- Occurs after severe emotional distress and **high catecholamines**
- Markedly reduced LVEF
- Increase CK, MB, Troponin; EKG changes
- Looks like anterior MI (but no coronary disease)
- Usually recovers 4-6 weeks

Diastole



Systole



Alcohol

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- Chronic consumption can cause cardiomyopathy
- Believed to be due to toxic metabolites
- Can recover with cessation of alcohol



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Restrictive Cardiomyopathy

www.afratafreeh.com

- Something “infiltrates” the myocardium
 - Amyloid protein (Amyloidosis)
- Heart cannot relax and fill
- **SEVERE diastolic dysfunction**
 - Presents as diastolic heart failure
 - Treatment: diuretics
 - Also treat underlying cause



MarkBuckawicki/Wikipedia

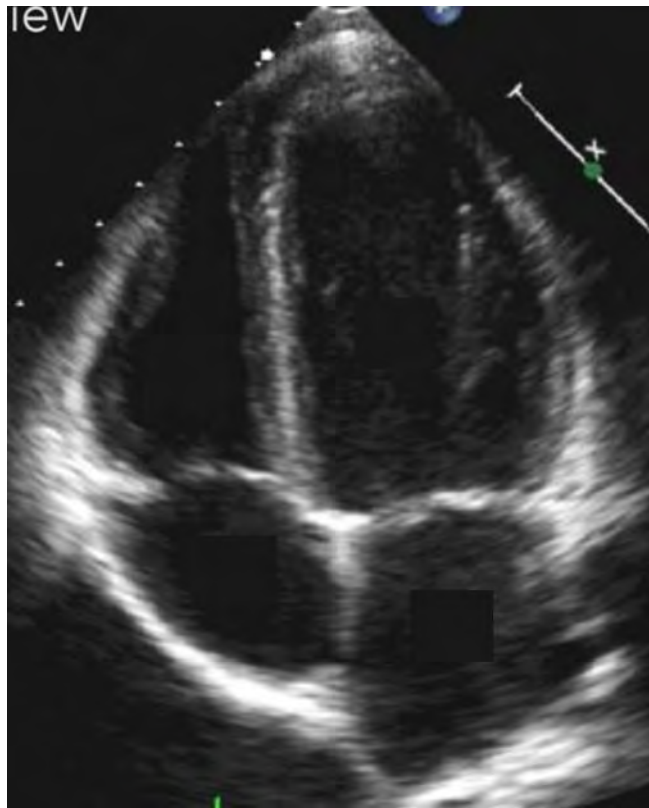
Restrictive Cardiomyopathy

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- **LVEF = normal**
- Restricted filling = $\uparrow$ atrial pressure
- **Dilated left and right atria**
- Classic imaging findings:
 - Normal left ventricular function/size
 - Bi-atrial enlargement

Restrictive Cardiomyopathy

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Normal



Restrictive Cardiomyopathy

Restrictive Cardiomyopathy

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Clinical Features

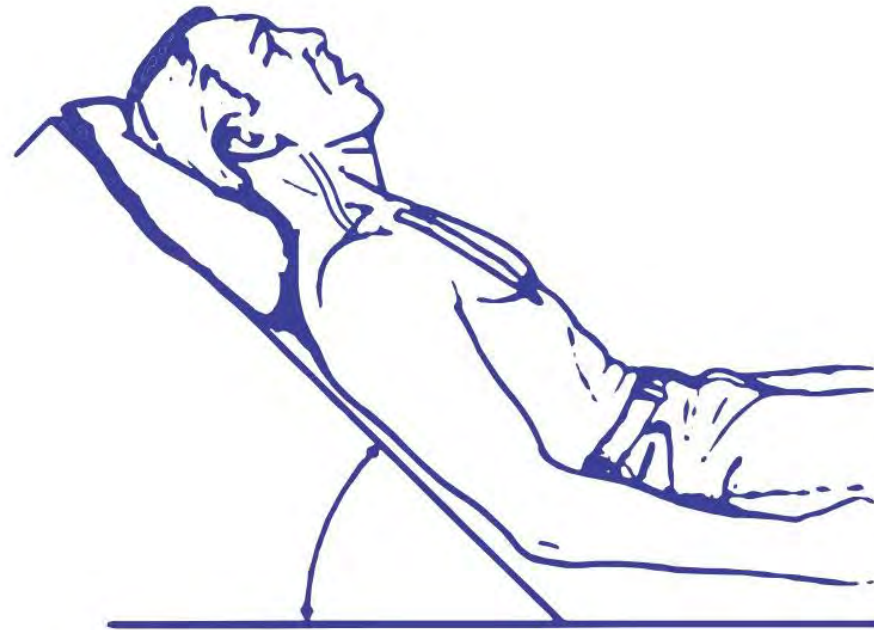
- Dyspnea
- Prominent **right heart failure**
 - Markedly elevated jugular venous pressure
 - Lower extremity edema
 - **Liver congestion**
 - May lead to cirrhosis

Restrictive Cardiomyopathy

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Classic signs

- **Kussmaul's sign**
 - Inspiration causes rise in JVP

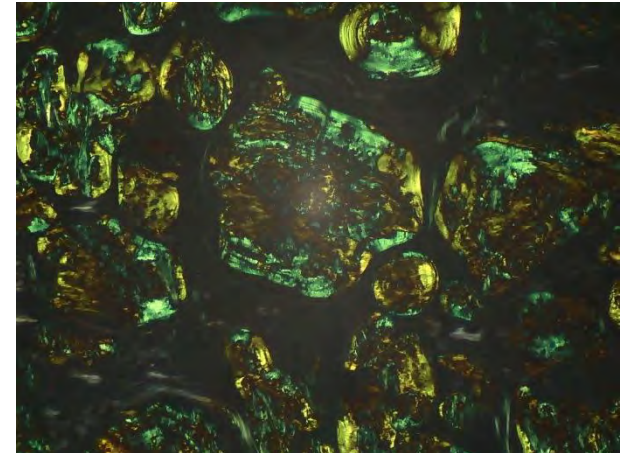


Restrictive Cardiomyopathy

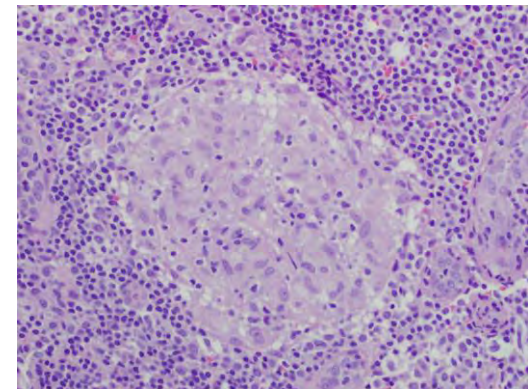
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Major Causes

- Amyloidosis
- Sarcoidosis
- Fabry disease (lysosomal storage disease)
- Hemochromatosis (rare)
- Post-radiation
- Loeffler's syndrome (hypereosinophilic syndrome)
- Endocardial fibroelastosis (babies)



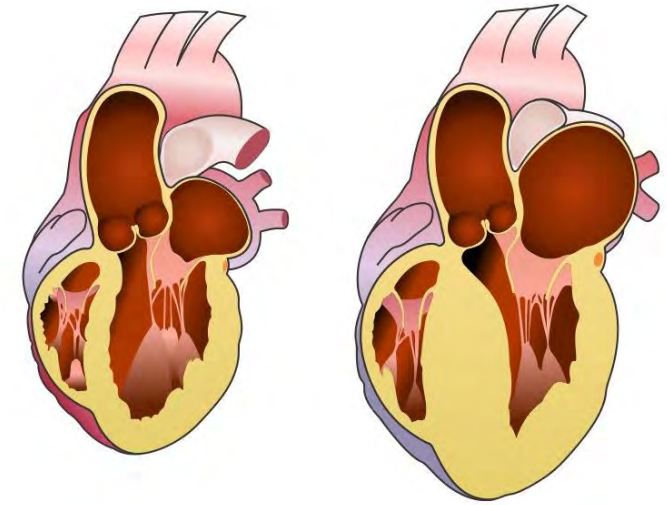
Ed Uthman, MD



Hypertrophic Cardiomyopathy

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- Cardiomyopathy of **myocyte hypertrophy**
 - Concentric hypertrophy
- Pathologic hypertrophy
 - Not due to stress
 - Absence of HTN or athlete's heart
- Diagnosis:
 - Echocardiography
 - Genetic testing
- LVEF normal with increased wall thickness

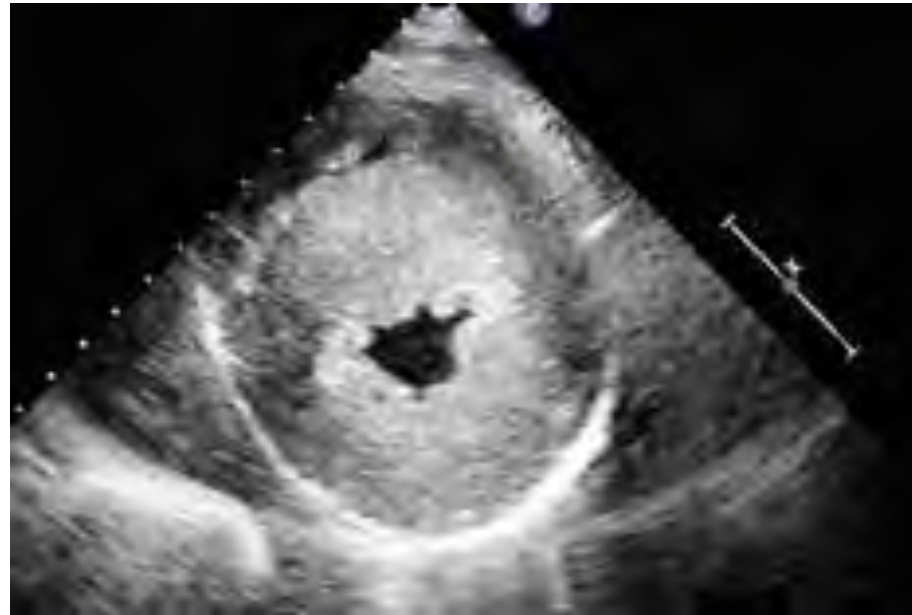


Hypertrophic Cardiomyopathy

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Normal



Hypertrophic Cardiomyopathy

Hypertrophic Cardiomyopathy

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- Genetic disorder caused by gene mutations
- About 50% cases familial (50% sporadic)
- **Autosomal dominant**
- Variable expression
 - Significant variation in severity of symptoms
 - Many variations in location/severity of hypertrophy



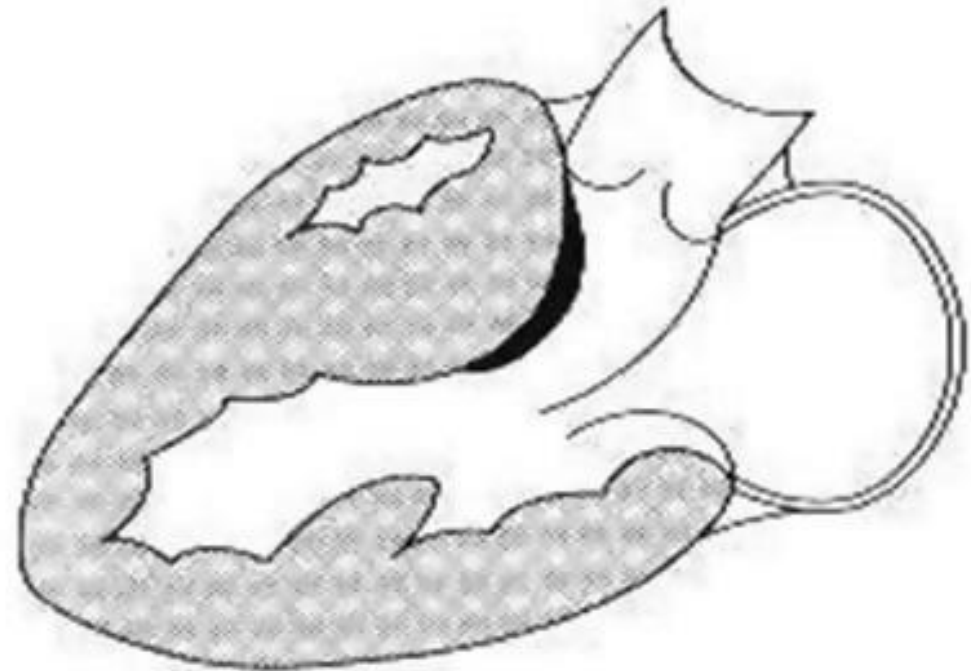
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Hypertrophic Cardiomyopathy

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Clinical Features

- Many patients asymptomatic
- Many symptoms from **LVOT obstruction**
 - Left ventricular outflow tract

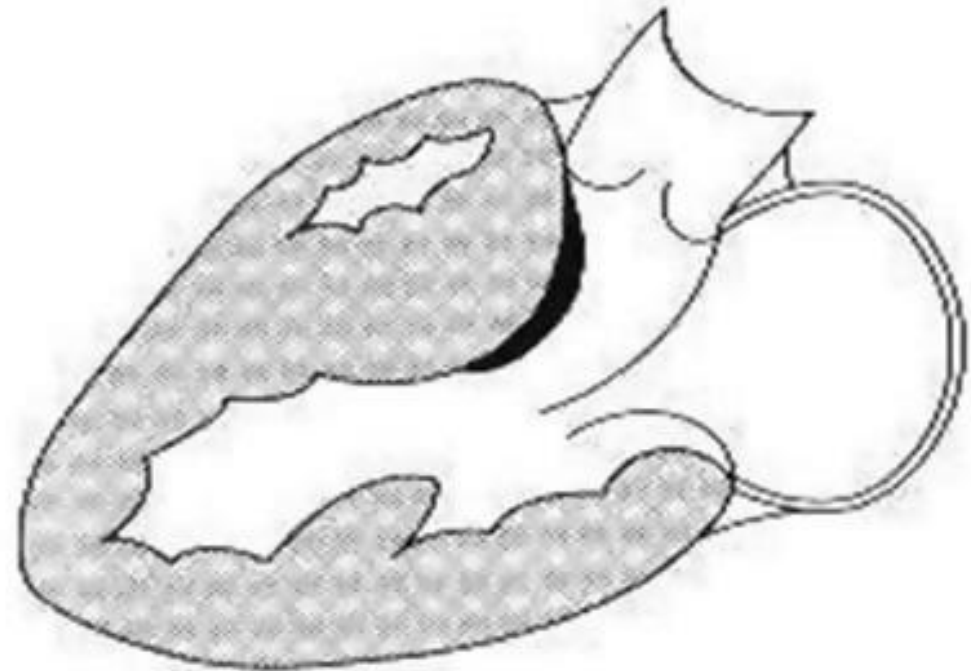


Hypertrophic Cardiomyopathy

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Clinical Features

- **Heart failure**
 - Diastolic dysfunction
 - LVOT obstruction
- **Chest pain (angina)**
 - Increased O₂ demand

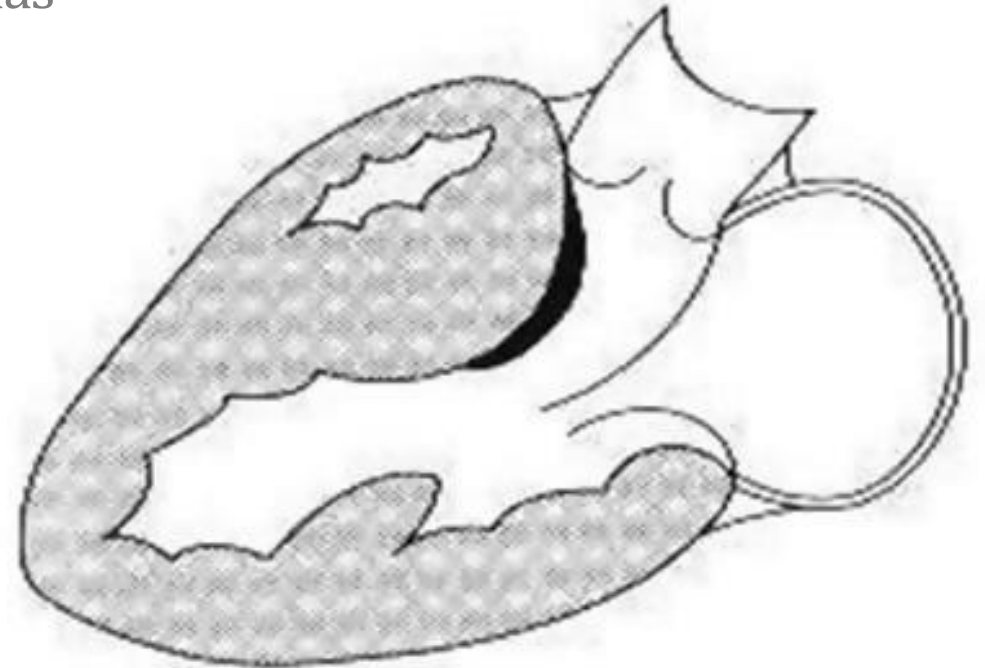


Hypertrophic Cardiomyopathy

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Clinical Features

- **Sudden cardiac death**
 - Abnormal myocytes → ventricular arrhythmias
 - Most common cause SCD in young patients
- **Syncope**
 - Arrhythmias may lead to syncope
 - LVOT obstruction
- **Mitral regurgitation**
 - Systolic anterior motion (SAM)

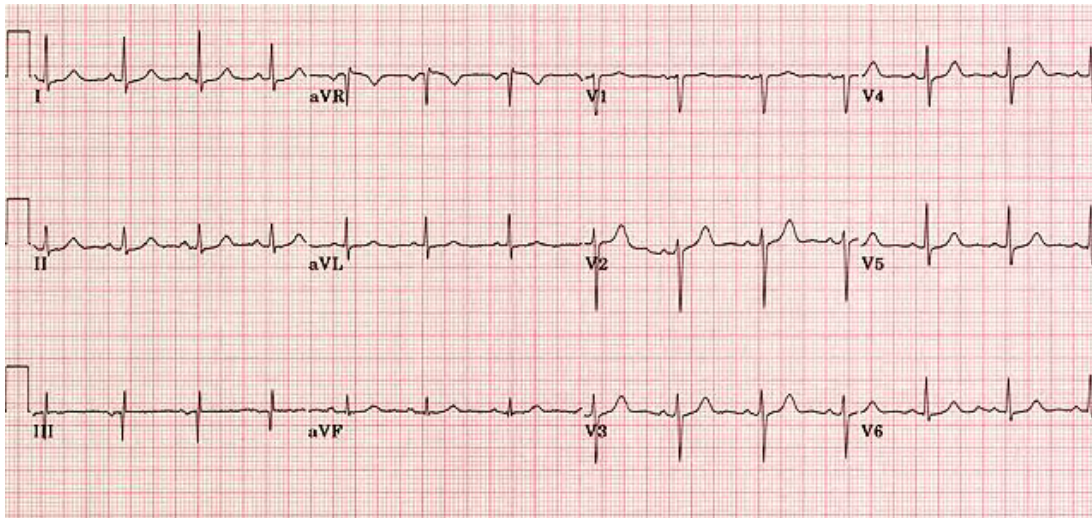


Hypertrophic Cardiomyopathy

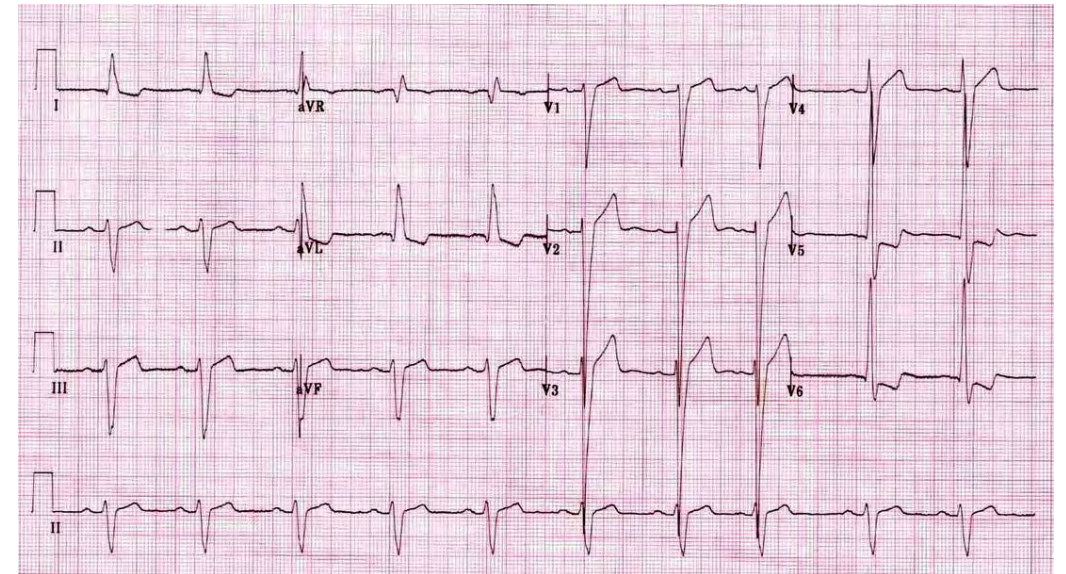
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Clinical Features

- EKG: Left ventricular hypertrophy (LVH)



Normal



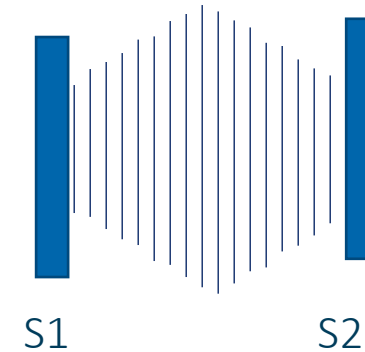
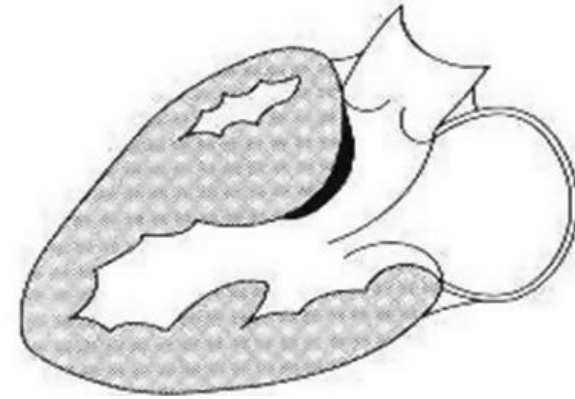
Hypertrophic Cardiomyopathy

Hypertrophic Cardiomyopathy

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Clinical Features

- **Systolic ejection murmur**
- Usually left lower sternal border
- Caused by outflow tract obstruction
- Sounds just like AS unless you do maneuvers
- Other associated abnormal heart sounds
 - S4
 - Holosystolic murmur of MR
 - Paradoxical split S2

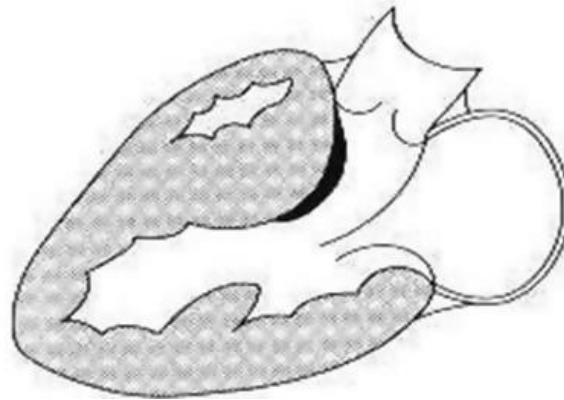


Hypertrophic Cardiomyopathy

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Maneuvers

- For any HCM maneuver, think about size of LV
- $\uparrow$ LV size $\rightarrow$ $\downarrow$ murmur
- $\downarrow$ LV size $\rightarrow$ $\uparrow$ murmur

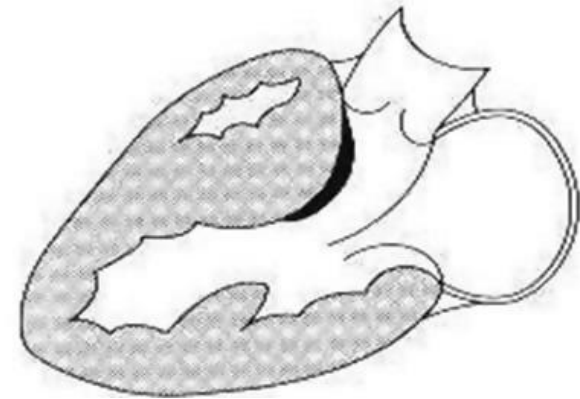


Hypertrophic Cardiomyopathy

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Maneuvers

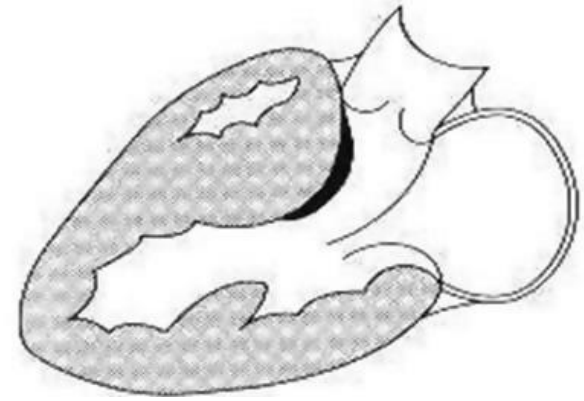
- Valsalva
 - Patient bears down as if having a bowel movement
 - Or blows out against closed glottis
 - Increase thoracic pressure → compression of veins → ↓ VR
 - Less VR → Less preload → Smaller LV cavity
 - Obstructing septum moves further into the outflow tract
 - Murmur **INCREASES** in intensity



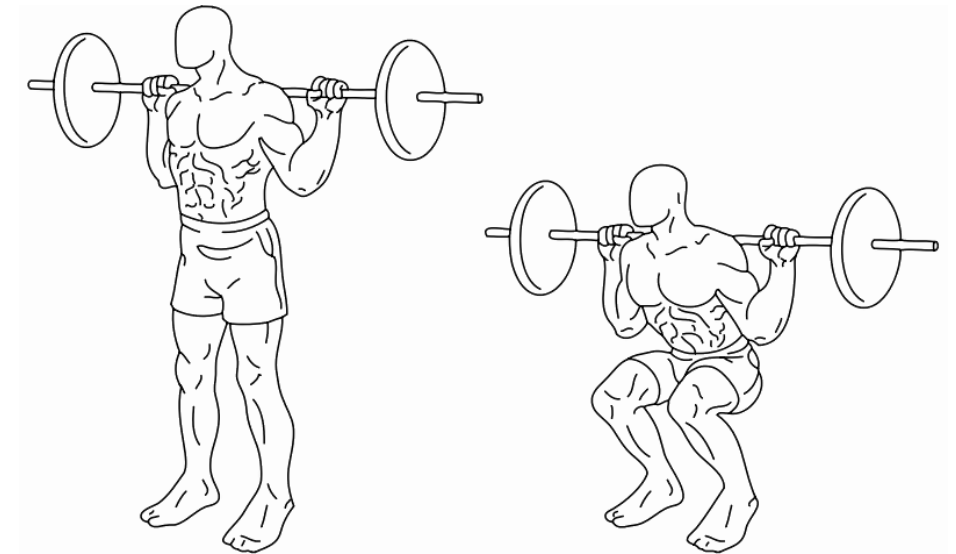
Hypertrophic Cardiomyopathy

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Maneuvers



- Squatting
 - Forces blood volume stored in legs to return to heart
 - Preload rises → size of LV increases → less obstruction
 - Murmur **DECREASES** in intensity
- Raising the legs
 - Increases venous return
 - Murmur **DECREASES** in intensity
- Standing
 - Opposite mechanism of leg raise
 - Murmur **INCREASES** in intensity



Wikipedia

Aortic Stenosis

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- Both HCM and AS cause a systolic ejection murmur
- Opposite effects of maneuvers in aortic stenosis
 - Less preload → less flow → quieter AS murmur

Hypertrophic Cardiomyopathy

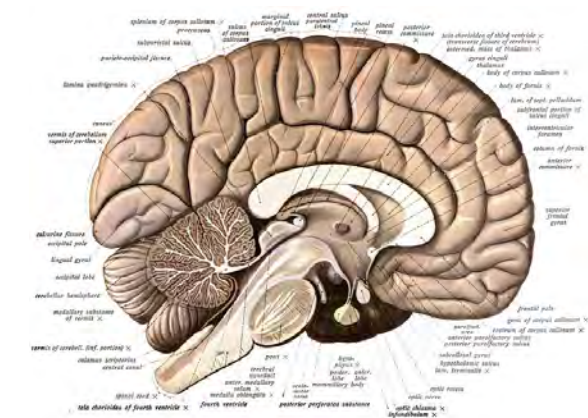
Associations

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- **Maternal diabetes**
 - Infants: transient hypertrophic cardiomyopathy
 - Usually thickening of interventricular septum
 - Resolves by a few months of age
- **Friedreich Ataxia**
 - Autosomal recessive CNS disease
 - Cause of death



Øyvind Holmstad/Wikipedia



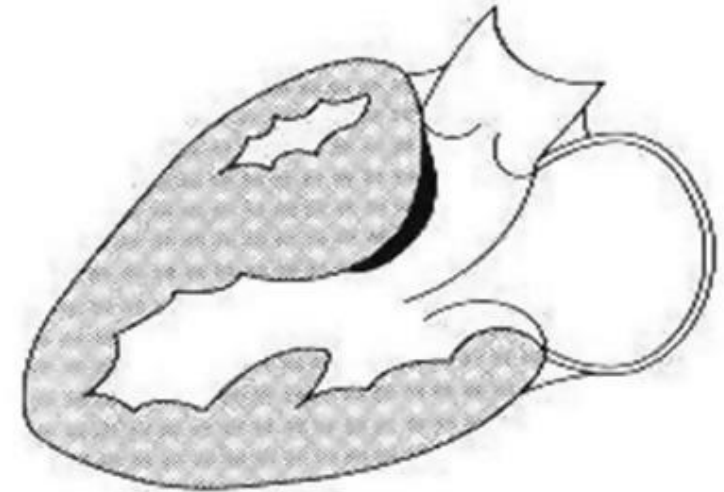
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Hypertrophic Cardiomyopathy

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Treatment

- **Beta-blockers and calcium channel blockers**
 - ↓ contractility
 - ↓ outflow gradient
- **Surgery**
 - Myomectomy
 - Alcohol septal ablation
 - Eliminates outflow obstruction
- Cautious diuretics for pulmonary edema
 - ↓ preload → hypotension
- Implantable cardiac defibrillators (ICDs)

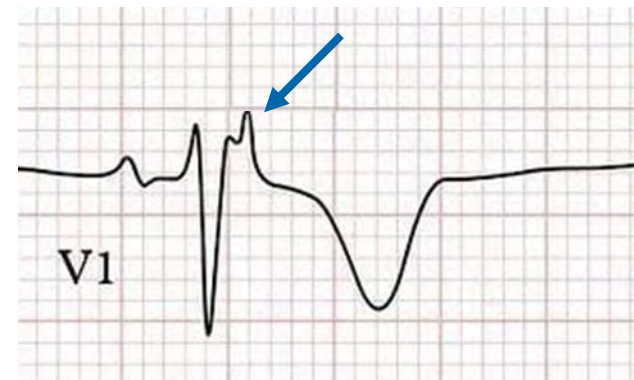
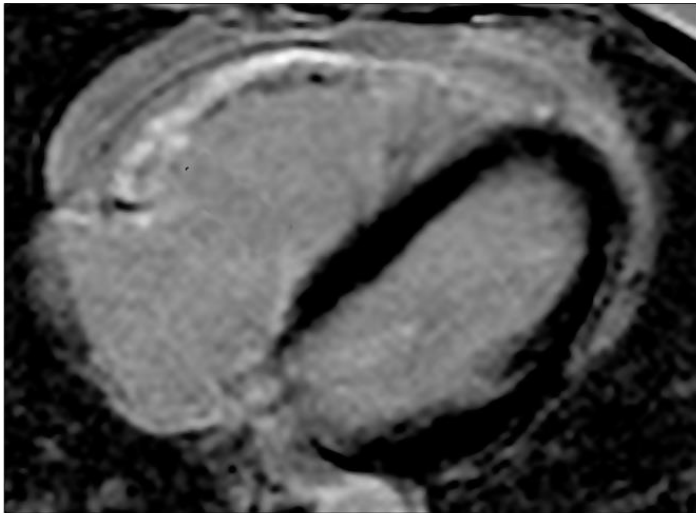


ARVD

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Arrhythmogenic right ventricular dysplasia

- Genetic disorder
- Cardiomyopathy of right ventricle → arrhythmias
- Associated with **sudden cardiac death** in young adults
- EKG: epsilon wave
- Diagnosis: **cardiac MRI**



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Pericardial Disease

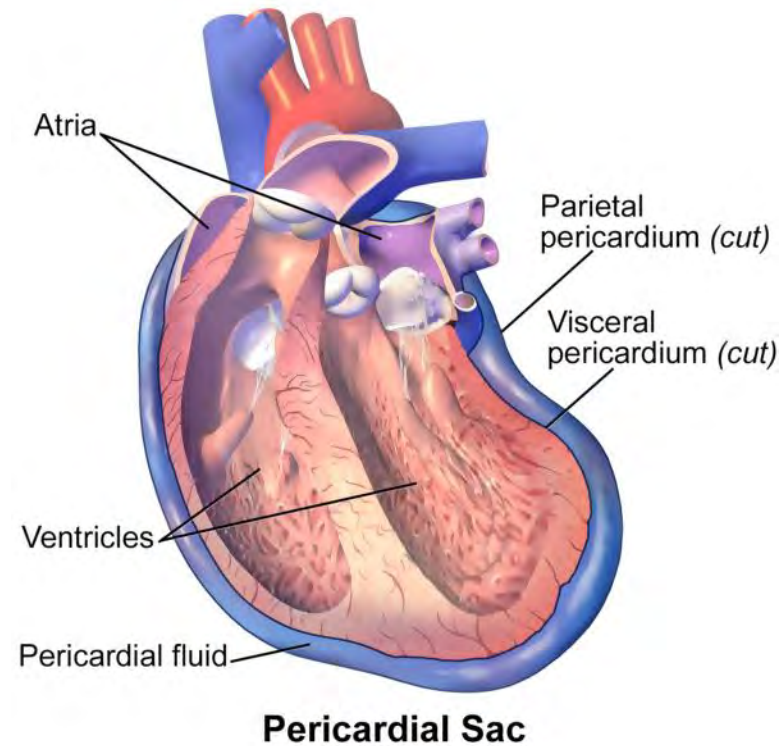
Jason Ryan, MD, MPH



Pericardial Diseases

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- Pericarditis
- Effusion/Tamponade
- Constrictive pericarditis



Blausen Medical Communications, Inc.

Acute Pericarditis

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- Most common pericardial disorder
- Inflammation of the pericardium
- May recur after treatment

Pericarditis

Clinical Features

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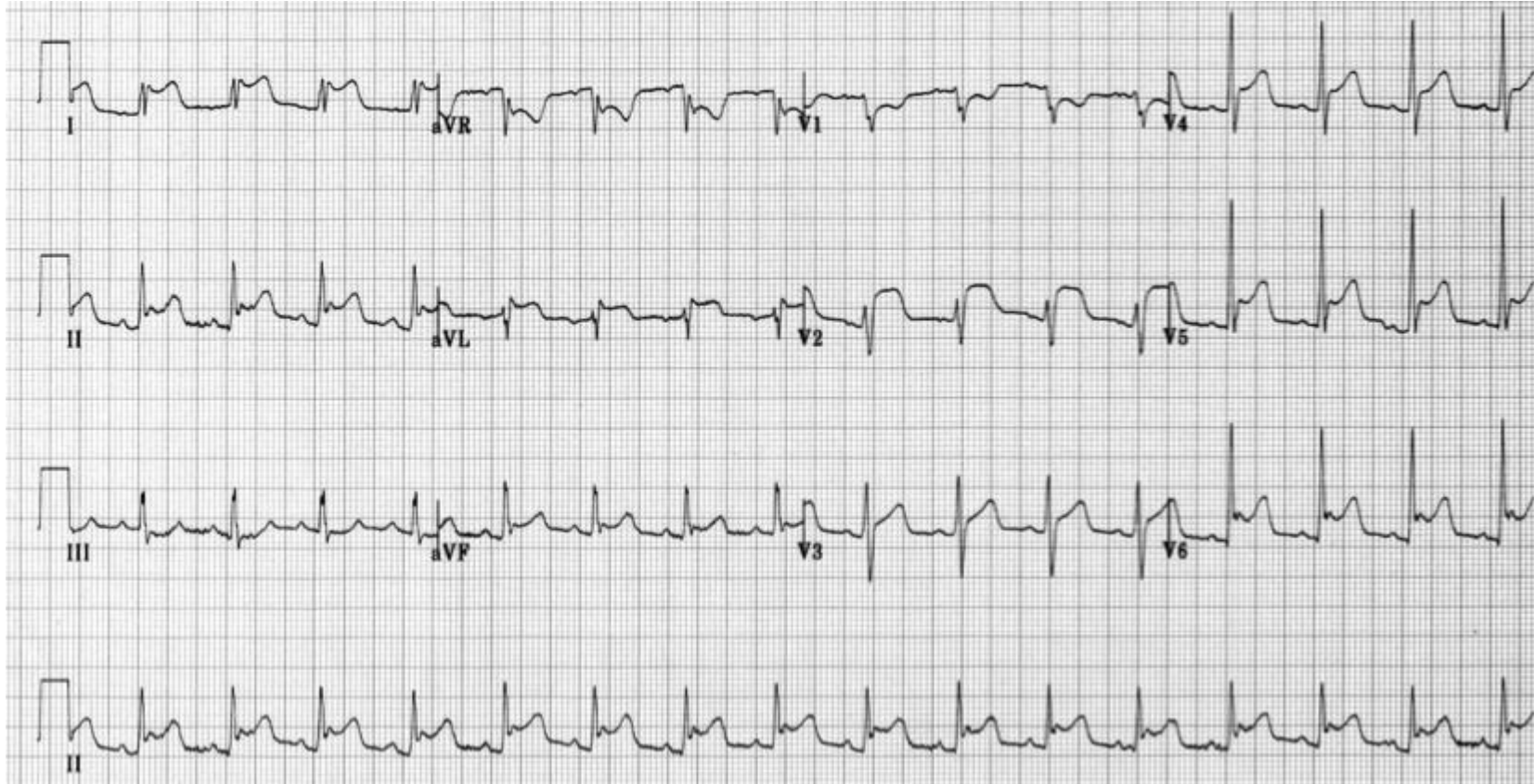
- **Chest pain**
 - Sharp
 - Worse with deep breath (pleuritic)
 - Worse lying flat (supine)
 - Better sitting up/leaning forward
- Fever
- Leukocytosis
- Elevated ESR
- About 50% have a **pericardial effusion**



Pericarditis

EKG Findings

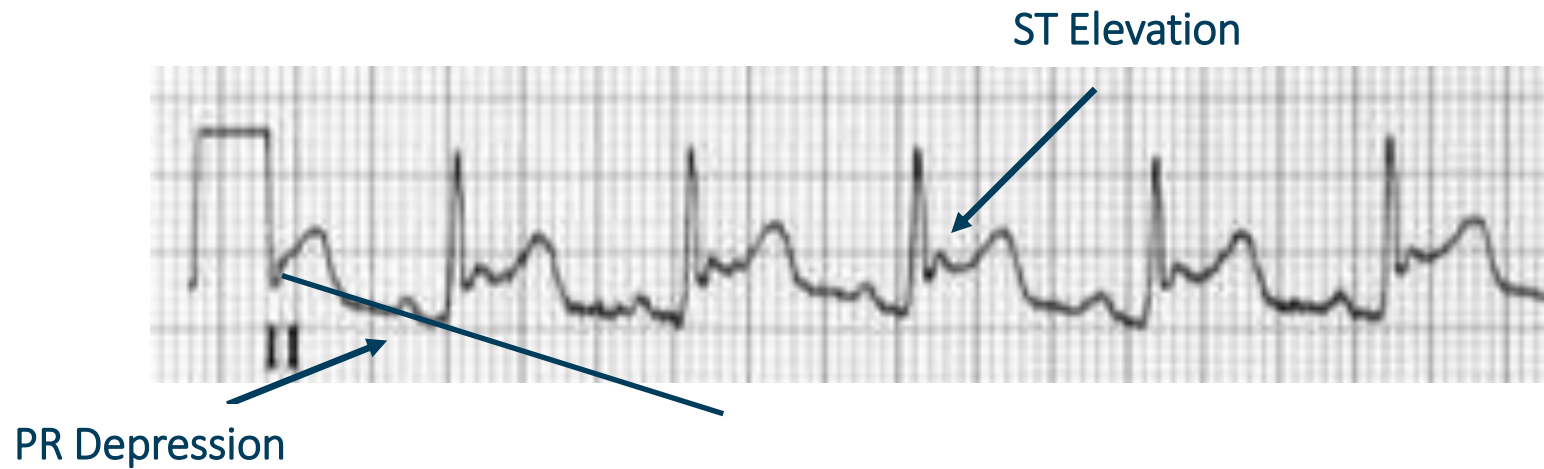
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Pericarditis

EKG Findings

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Pericarditis
Diffuse ST elevation
PR depression

Pericarditis

Physical Exam

- Pericardial friction rub
- **Scratchy** sound
- Systole and diastole

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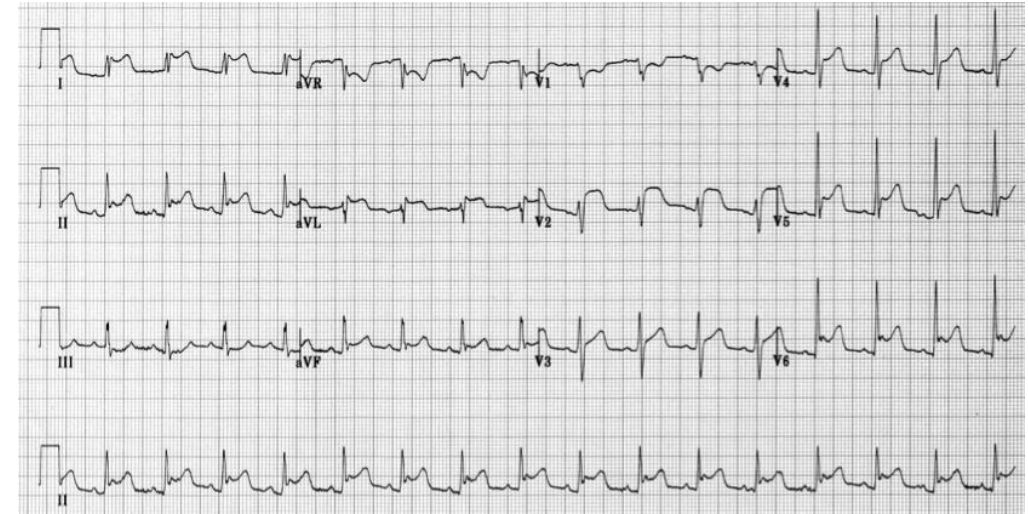


Pericarditis

Diagnosis

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- Clinical diagnosis based on **two of four criteria**:
 - Typical pericarditis chest pain
 - Pericardial friction rub
 - Pericarditis ECG changes
 - Pericardial effusion
- All patients: **transthoracic echocardiogram**
 - Evaluate for effusion

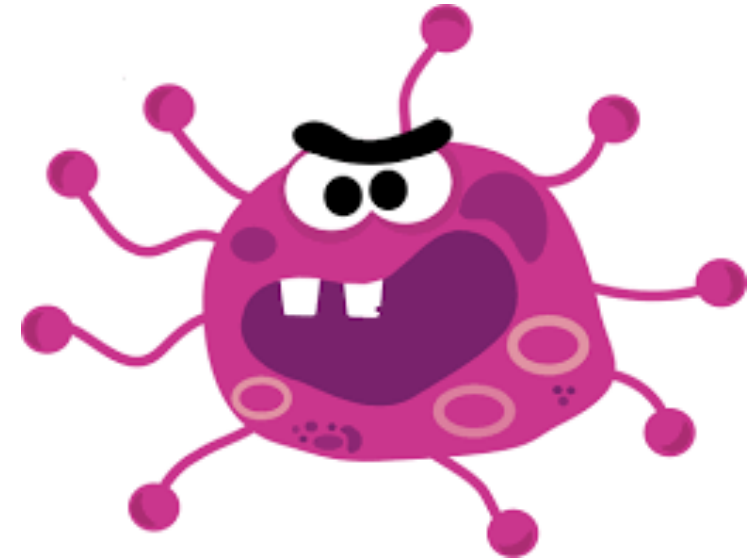


Pericarditis

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Etiology

- Usually idiopathic
- Viral
 - Classic cause is Coxsackievirus
 - Often follows viral **upper respiratory infection (URI)**
- Bacterial
 - Spread of pneumonia
 - Complication of surgery
 - Tuberculosis
- Fungal



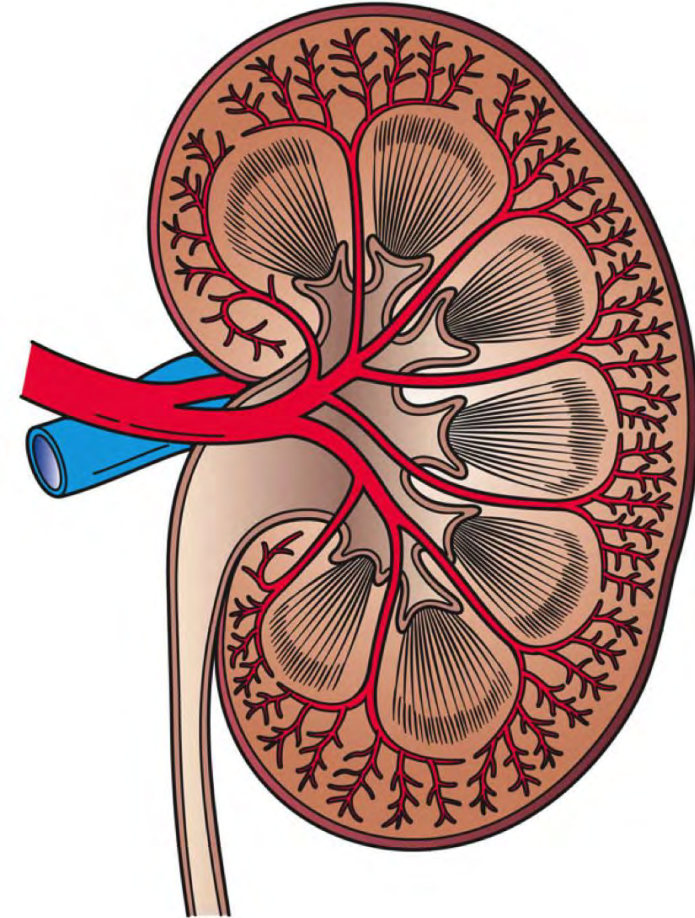
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Pericarditis

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Etiology

- Uremic (renal failure)
 - Treatment: hemodialysis (not drugs!)
- Post-myocardial infarction
 - Fibrinous (days after MI)
 - Dressler's syndrome (weeks after MI)
- Autoimmune disease (RA, Lupus)



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Pericarditis

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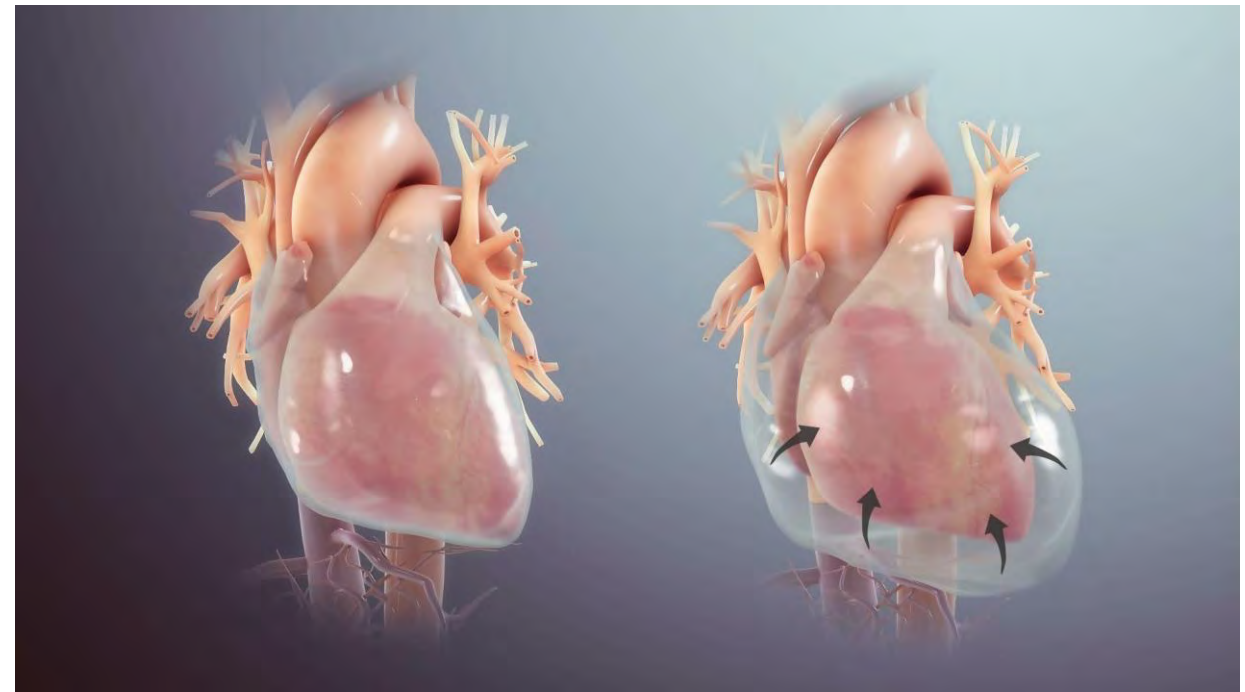
Treatment

- NSAIDs
- Glucocorticoids
- Colchicine
 - Inhibits WBCs via complex mechanism
 - Useful in gout and familial Mediterranean fever
 - Added to NSAIDs to lower risk of recurrence
- **NSAIDs plus colchicine** used in most cases

Pericardial Effusion

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- **Fluid in the pericardial space**
- Often asymptomatic
- May cause pericardial tamponade

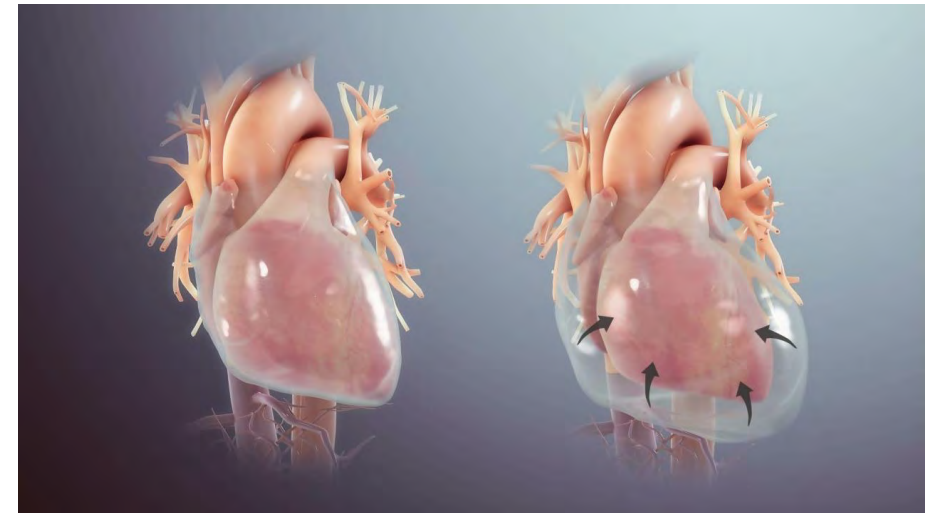


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Pericardial Tamponade

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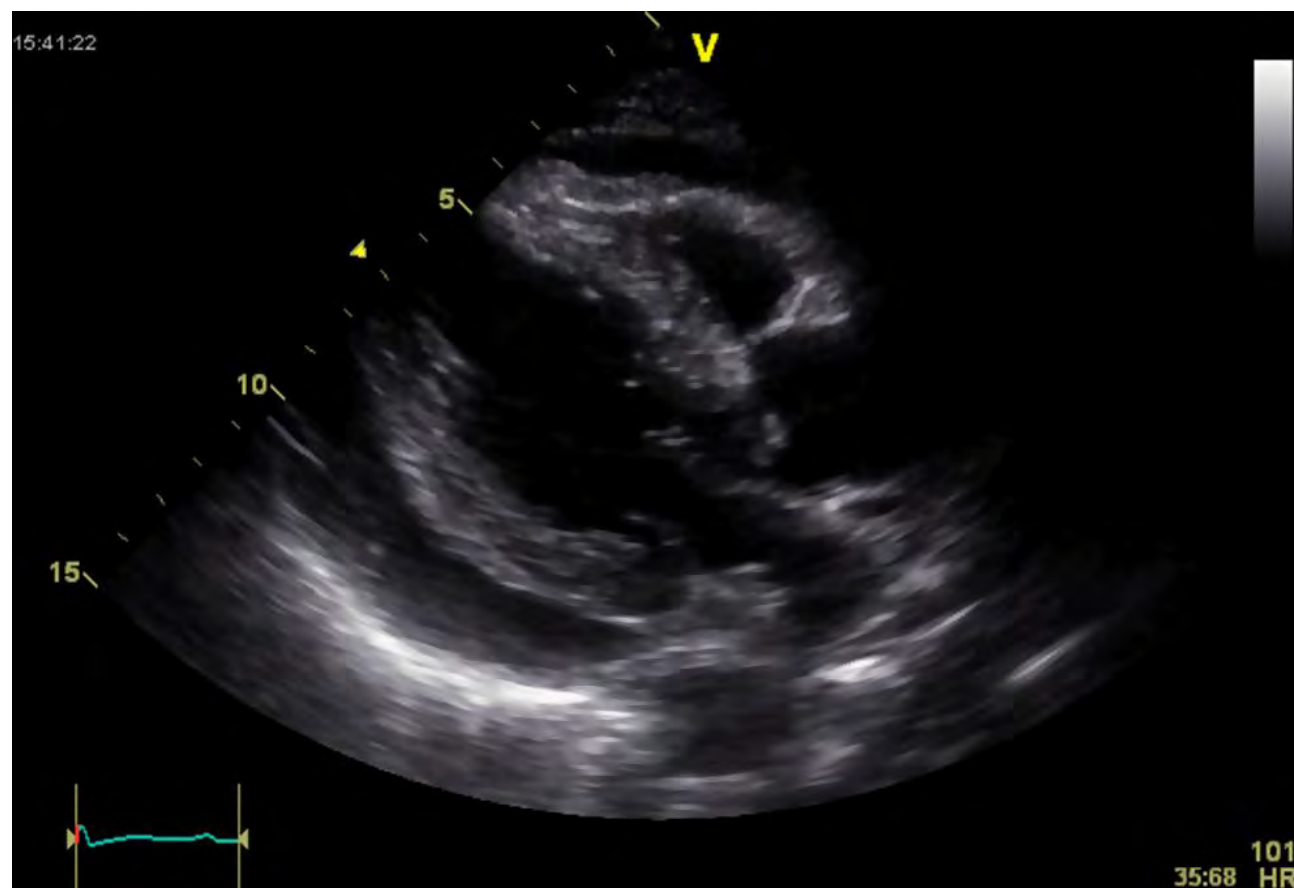
- Caused by **pericardial effusions**
- High pericardial pressure → ↓ filling of ventricles in diastole
- Amount of fluid required for tamponade varies
 - Acute accumulation (bleeding): small amount of fluid
 - Chronic accumulation (cancer): large amount of fluid



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Pericardial Effusion

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Pericardial Effusion

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Pericardial Tamponade

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Clinical features

- Distant heart sounds
- Dyspnea with absence of pulmonary edema
- Elevated jugular venous pressure

Pericardial Tamponade

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Clinical features

- Beck's Triad
 - Distant heart sounds
 - Elevated JVP
 - Hypotension
- Seen in rapidly-developing traumatic effusions
- **Low cardiac output**
 - Decreased filling of right and left ventricles
 - Decreased preload
- Slower effusions: Pericardium stretches/dilates

Pulsus Paradoxus

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- Classic finding in tamponade
- Systolic BP always falls slightly on inspiration
- Exaggerated fall (> 10 mmHg) = pulsus paradoxus
- Severe fall = pulse disappears
- Also seen in **asthma and COPD**
 - Inspiration normally $\downarrow$ left sided flow
 - Exaggerated in lung disease



Public Domain

Pericardial Tamponade

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EKG

- Seen in large pericardial effusions
- Usually tamponade is present
- Sinus tachycardia
- Low voltage – EKG sees less electricity due to effusion

Electrical Alternans



Tamponade

Diagnosis and Treatment

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- Diagnosis:
 - Pulsus paradoxus (clinical evidence of tamponade)
 - Transthoracic echocardiography
- Treatment:
 - Intravenous fluids (do NOT give diuretics)
 - Pericardiocentesis
 - Surgical pericardial window



Tamponade

Right heart catheterization

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- Equalization of pressures
- Occurs when cardiac chambers cannot relax
- Seen in tamponade and pericardial constriction

Parameter	Normal	Tamponade/ Constriction
RA mean	5	20
RV Pressure	20/5	44/20
PCWP Pressure	10	20
LVEDP	10	20

Effusions/Tamponade

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Causes

- **Pericarditis**
- **Cancer metastases** to pericardium
- Trauma

Constrictive Pericarditis

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Constrictive Pericarditis

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- Fibrous, calcified scar in pericardium
- Loss of elasticity: stiff, thickened, sticky
- Can result from many pericardial disease processes
 - Pericarditis
 - Radiation to chest
 - Heart surgery

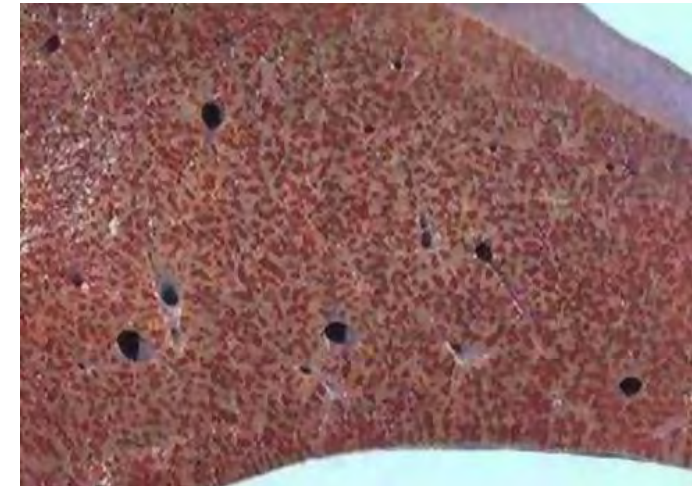
Constrictive Pericarditis

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Clinical Features

- Major pathologic process: ventricles cannot fill normally
- Filling abruptly stops
- Dyspnea (low cardiac output)
- Prominent **right heart failure**
 - Markedly-elevated jugular venous pressure
 - Lower extremity edema
 - Liver congestion
 - May lead to cirrhosis (“nutmeg liver”)

Nutmeg Liver (dark spots)



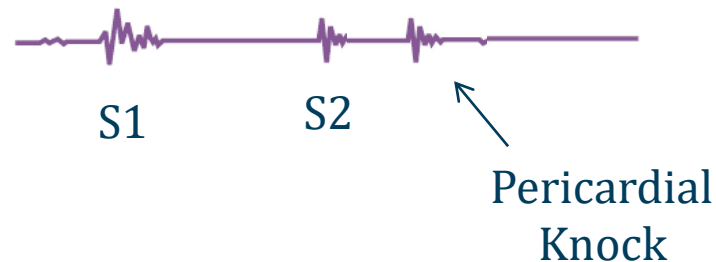
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Constrictive Pericarditis

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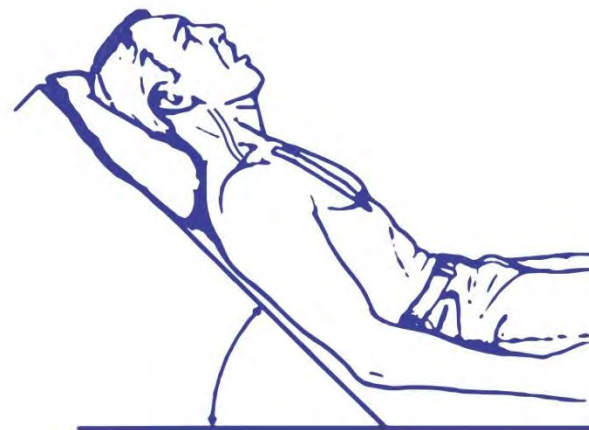
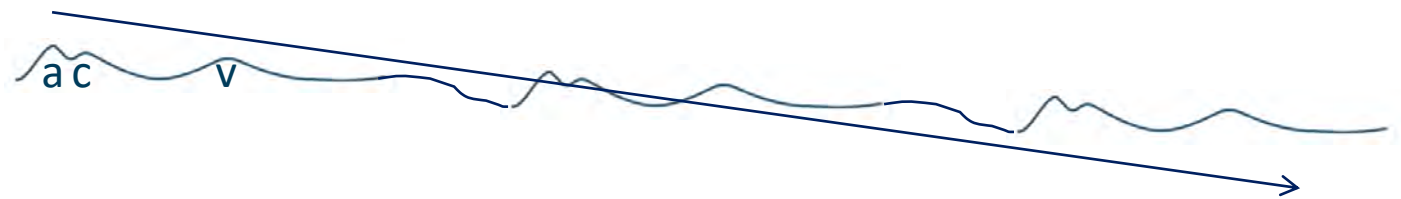
Other Features

- Pulsus paradoxus uncommon (~20%)
- High RA, RVEDP, PCWP pressures
- Equalization of pressures
- **Pericardial knock**



Kussmaul's Sign www.afratafreeh.com

- Inspiration $\rightarrow$ $\uparrow$ VR $\rightarrow$ slight fall in mean JVP
- **Kussmaul's sign** = **$\uparrow$ JVP with inspiration**
 - Ventricle cannot accept $\uparrow$ VR
- Constrictive pericarditis
- Restrictive cardiomyopathy
- RV myocardial infarction



Pulsus and Kussmaul's

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- Pulsus paradoxus: classic sign of tamponade
 - **Pulsus in tamPonade**
- Kussmaul's sign: classic sign of constriction
 - Also seen in restrictive heart disease
 - **Kussmaul's in Konstriction/Restriction**

	Tamponade	Constriction	Restrictive
Pulsus	Yes	No	No
Kussmaul's	No	Yes	Yes

Constrictive Pericarditis

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Diagnosis and Treatment

- Diagnosis:
 - Best first test: **chest X-ray**
 - Cardiac MRI or CT
 - Right heart catheterization
- Echocardiography:
 - Not very useful
 - Poor pericardial visualization
 - Shows normal LVEF
 - Enlarged atria
- Treatment: pericardiectomy



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Valvular Heart Disease

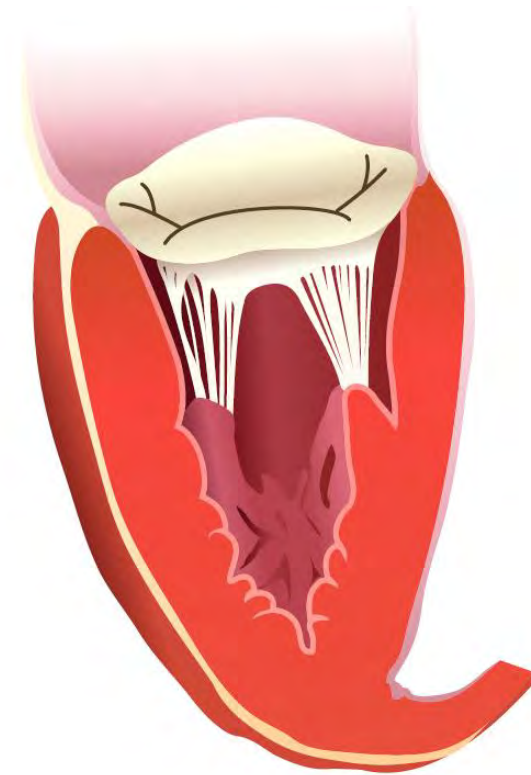
Jason Ryan, MD, MPH



Valvular Heart Disease

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- **Stenosis**
 - Stiffening/thickening of valve leaflets
 - Obstruction to forward blood flow
- **Regurgitation**
 - Malcoaptation of valve leaflets
 - Leakage of blood flow backwards across valve



Regurgitant Lesions

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- **Acute and chronic forms**
- Acute regurgitation (often from endocarditis)
 - May cause shock
- Chronic regurgitation
 - No shock
 - Leads to chronic heart failure

Valve Disorders

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Diagnosis and treatment

- Best initial test: **transthoracic echocardiography**
 - EKG not helpful but may show signs of chamber enlargement
- Only severe valvular lesions treated
- Mostly **surgical diseases**
 - Surgical repair
 - Valve replacement
 - Bioprosthetic (pig or cow)
 - Mechanical (requires life-long anticoagulation)
 - Valvuloplasty (mitral and aortic stenosis)



Valve Disorders

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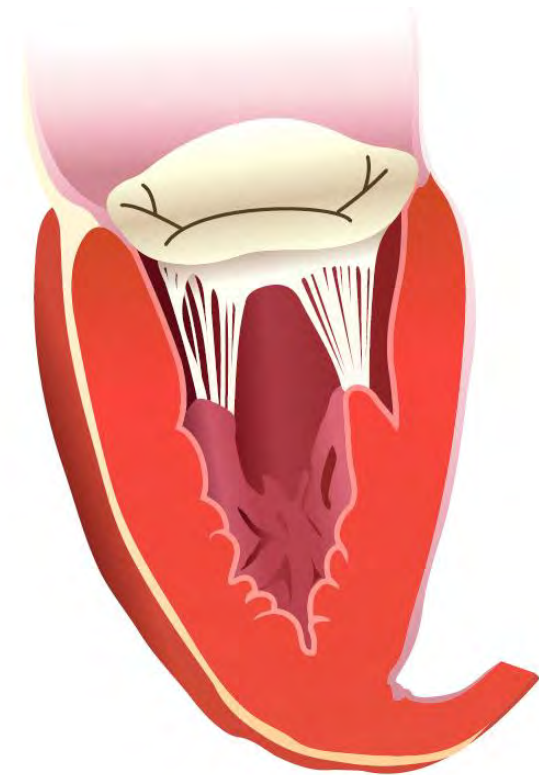
Medical treatment

- Medical therapy used rarely in patients with heart failure
 - Loop diuretics
- Afterload reduction
 - Vasodilators
 - In theory could improve forward flow in regurgitant valve disease (AR, MR)
 - Clinical trials have shown little benefit of drugs

Rheumatic Heart Disease

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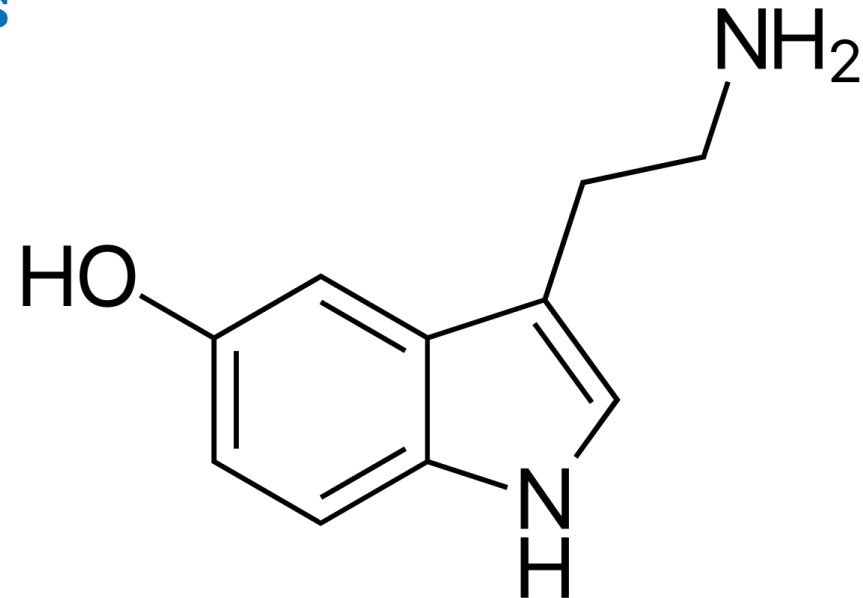
- Damage to heart valves by rheumatic fever
- Often presents years after acute rheumatic fever
- Many patients do not recall acute symptoms
- **Mitral valve** most commonly involved
- Common in **developing countries**
 - Limited access to medical care for pharyngitis
 - Often seen in **immigrants** to US



Carcinoid Heart Disease

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- Caused by carcinoid tumors of intestines
- Secrete **serotonin**
- Fibrous deposits **tricuspid/pulmonic valves**
- Leads to stenosis and regurgitation
- Serotonin inactivated by lungs
- Left-sided lesions rare



Serotonin

Aortic Stenosis

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Pathophysiology

- Fibrotic/calcified aortic valve
- **Increased afterload**



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Aortic Stenosis

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Hemodynamics

- **LV systolic pressure >> aortic systolic pressure**
 - LVSP = 160 mmHg (normal = 120)
 - SBP = 120 mmHg (normal = 120)
 - Gradient = 40 mmHg



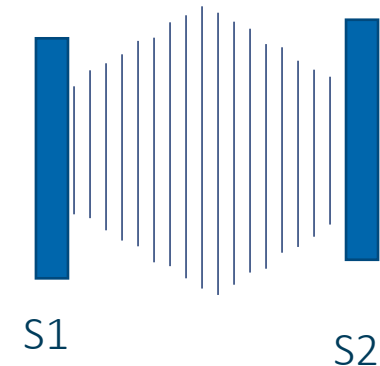
Aortic Stenosis

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Clinical features

- Physical Exam
 - **Systolic crescendo-decrescendo murmur at 2nd right intercostal space**
 - Often radiates to carotids
- Severe disease findings
 - Soft, single S2 (restricted leaflet motion)
 - Pulsus parvus et tardus
- Symptoms
 - Angina
 - Syncope
 - Left heart failure (worst prognosis)

Crescendo-Decrescendo
Murmur

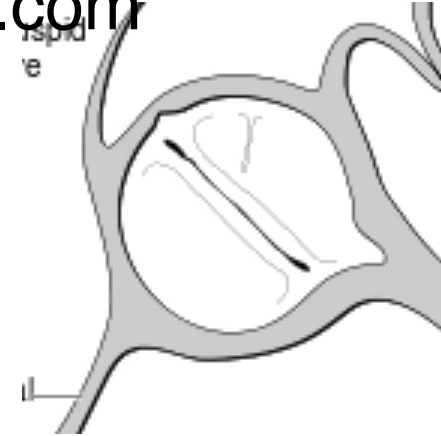


Aortic Stenosis

Causes

- **Senile aortic stenosis**
 - “Wear and tear”
 - Collagen breakdown
 - Calcium deposition
- **Bicuspid aortic valve**
- Rarely rheumatic heart disease

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Patrick J. Lynch, medical illustrator



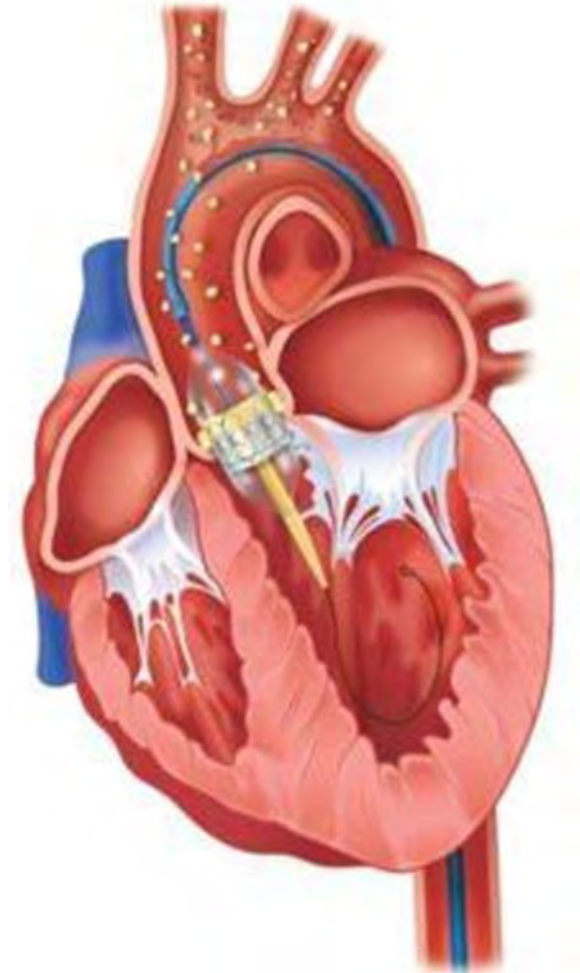
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Aortic Stenosis

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Special treatments

- **Transcatheter aortic valve replacement (TAVR)**
 - No chest incision
 - Faster recovery



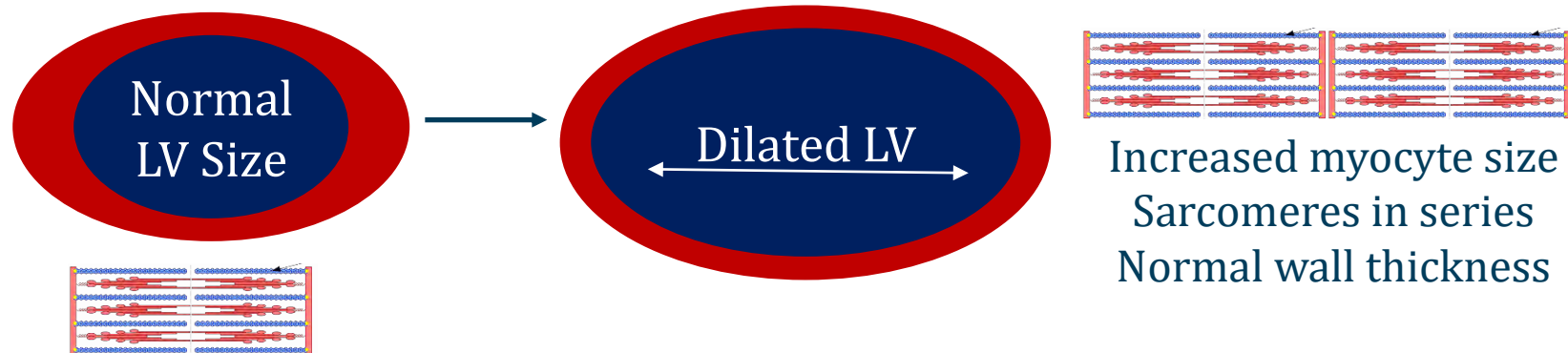
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Aortic Regurgitation

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Pathophysiology

- Inadequate closure of leaflets → blood leaks across aortic valve in diastole
- **Increased preload, stroke volume**
- Eccentric hypertrophy eventually leading to heart failure

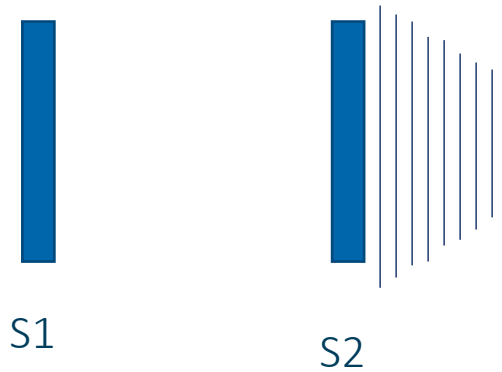


Aortic Regurgitation

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Clinical features

- Blowing, decrescendo diastolic murmur at lower left sternal border



Aortic Regurgitation

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Clinical features

- Leaking blood back into LV causes **low diastolic BP**
 - 120/80 (normal) → 120/40
 - Low diastolic pressure
- **Wide pulse pressure**
 - High cardiac output with low diastolic pressure
- Wide pulse pressure symptoms
 - “Water hammer” pulses
 - Head bobbing
 - Many, many others (mostly historical)

Aortic Regurgitation

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Causes

- **Dilated aortic root** → leaflets pull apart
 - Often from HTN or other aortic aneurysm (Marfan)
 - Rarely from tertiary syphilis (aortitis)
- **Bicuspid aortic valve**
 - Turner syndrome
 - Coarctation of the aorta
- Endocarditis
- Rheumatic heart disease
 - Almost always with mitral disease

Mitral Stenosis

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Pathophysiology

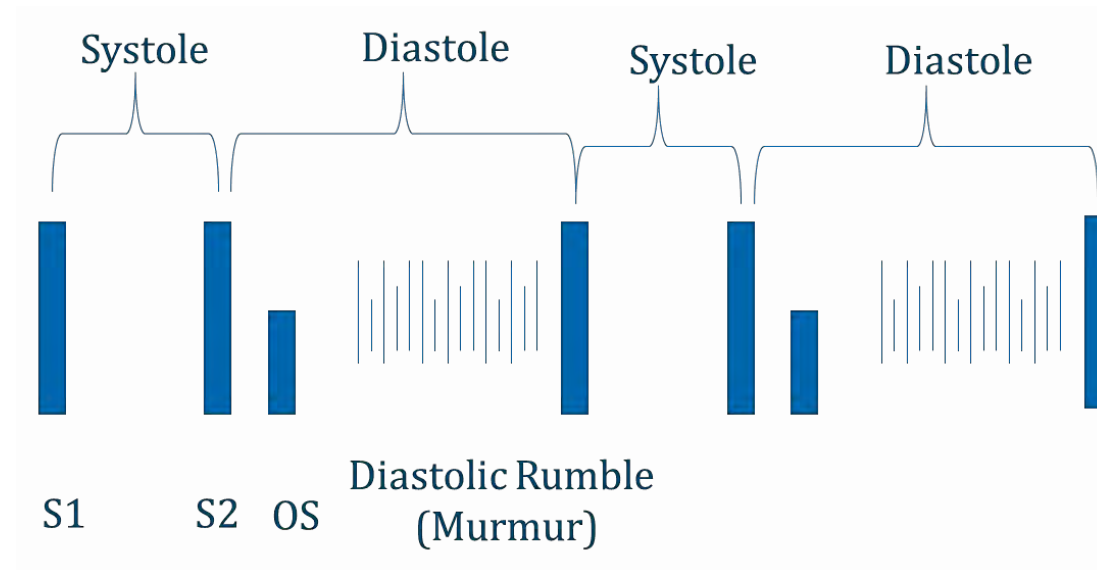
- Most cases caused by **rheumatic heart disease**
- Stiff mitral valve
- **LA pressure >> LV diastolic pressure**
 - Left atrial pressure 20 mmHg (normal = 10)
 - LVEDP 5 mmHg (normal = 10)
 - Gradient = 15 mmHg
- Left atrium may become massively dilated
 - Commonly causes atrial fibrillation

Mitral Stenosis

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Clinical features

- Most common symptom: **dyspnea**
 - ↑ LA pressure → pulmonary congestion
- Murmur: diastolic rumble with opening snap

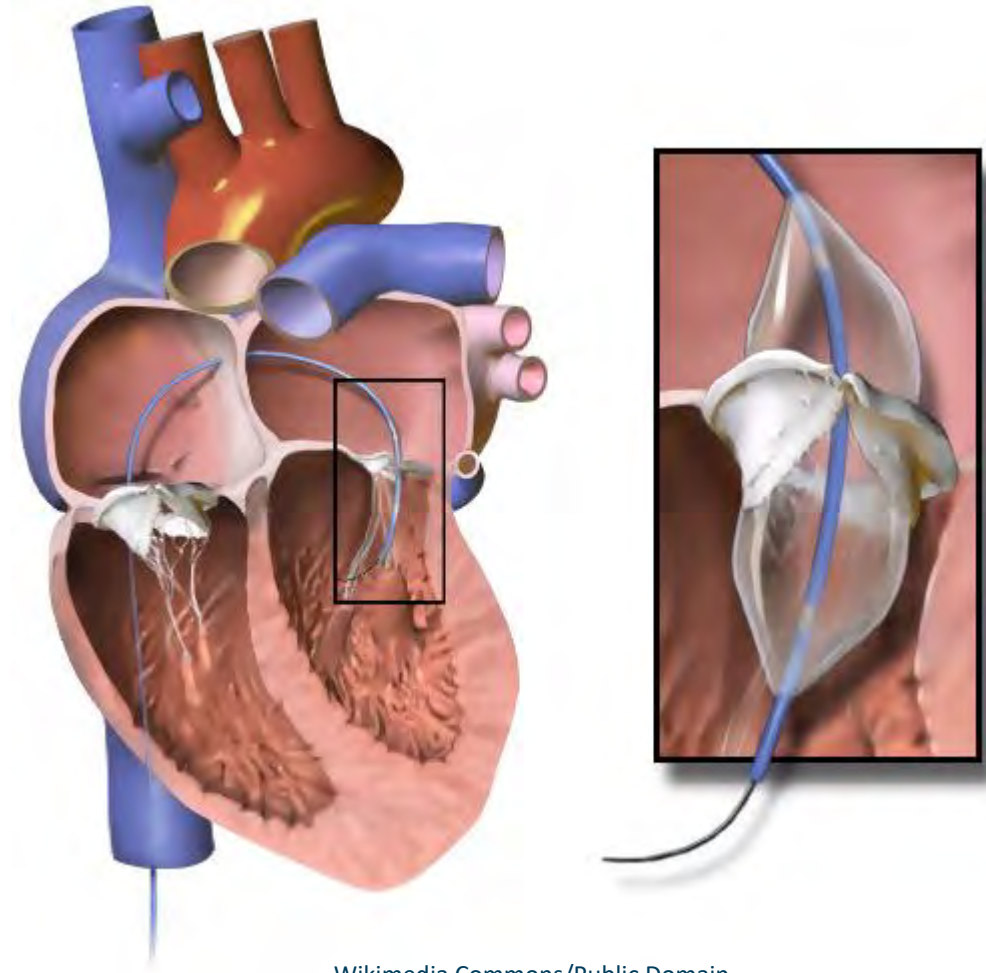


Mitral Stenosis

Special treatments

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- **Balloon valvuloplasty**
 - Used in rheumatic mitral stenosis
 - Breaks up fibrous tissue
 - Valve must have little/no calcium deposition



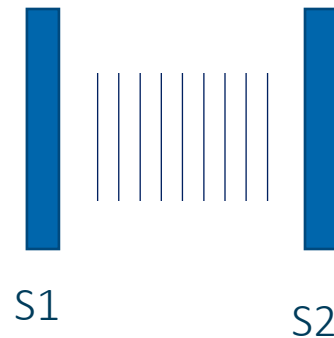
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Mitral Regurgitation

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Clinical Features

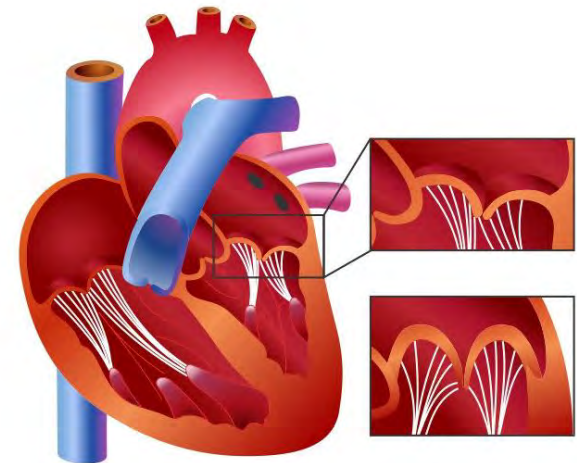
- Holosystolic murmur at apex
- Radiates to the axilla



Mitral Valve Prolapse

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- Primary mitral regurgitation
 - Also called degenerative or myxomatous
 - Billowing of mitral valve leaflets above annulus
- Causes a **systolic click**
 - Don't confuse with opening snap of mitral stenosis
- Systolic murmur from MR
 - Increases with standing or Valsalva
 - Decreases with squatting

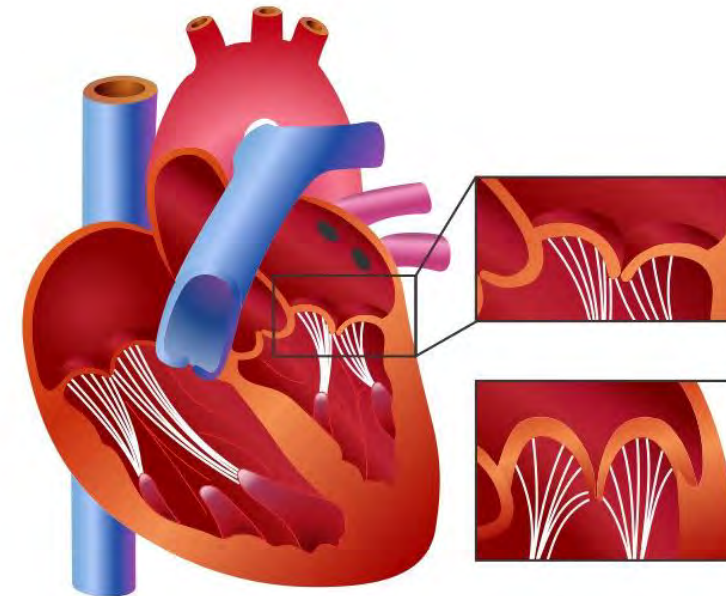


Mitral Regurgitation

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Secondary causes

- Ischemia → damage to papillary muscle
- Left ventricular dilation
 - Dilated cardiomyopathy
 - Leaflets pulled apart
 - **“Functional” MR**
- Hypertrophic cardiomyopathy
 - Systolic anterior motion (SAM)



Mitral Regurgitation

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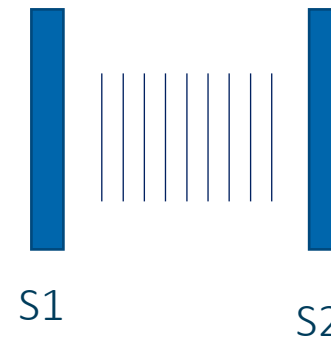
Secondary causes

- Endocarditis
- Rheumatic heart disease
- Congenital
 - Cleft mitral valve
 - Endocardial cushion defect
 - Down syndrome

Tricuspid Regurgitation

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- Holosystolic murmur at left sternal border
- Small amount of TR normal (“physiologic TR”)
- Pathologic causes
 - Functional TR from **RV enlargement**
 - **Endocarditis - classically IV drug users**
 - Carcinoid
 - Ebstein’s anomaly



Tricuspid Stenosis

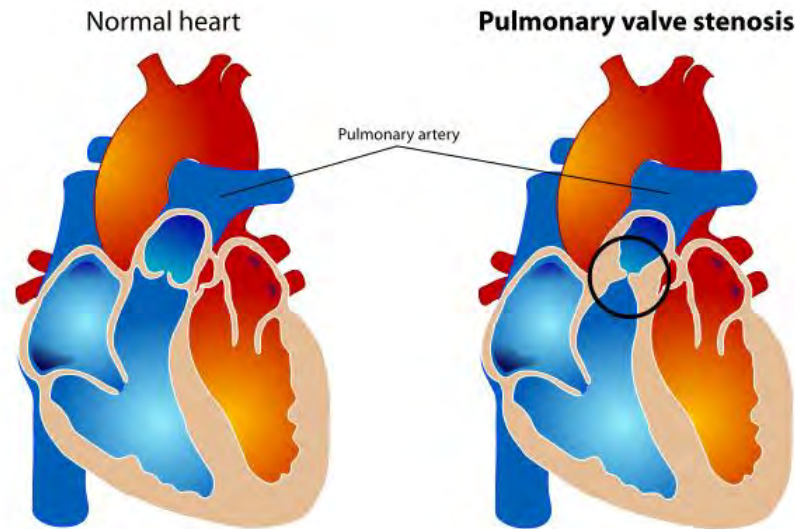
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- Very rare valve disorder
- **Diastolic murmur at left lower sternal border**
- Caused by rheumatic heart disease (with mitral disease)
 - Tricuspid regurgitation more common
- Carcinoid heart disease

Pulmonic Stenosis

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- **Congenital defect** in children
 - Fused commissures with thickened leaflets
- Carcinoid heart disease
- Systolic crescendo-decrescendo murmur at left upper sternal border

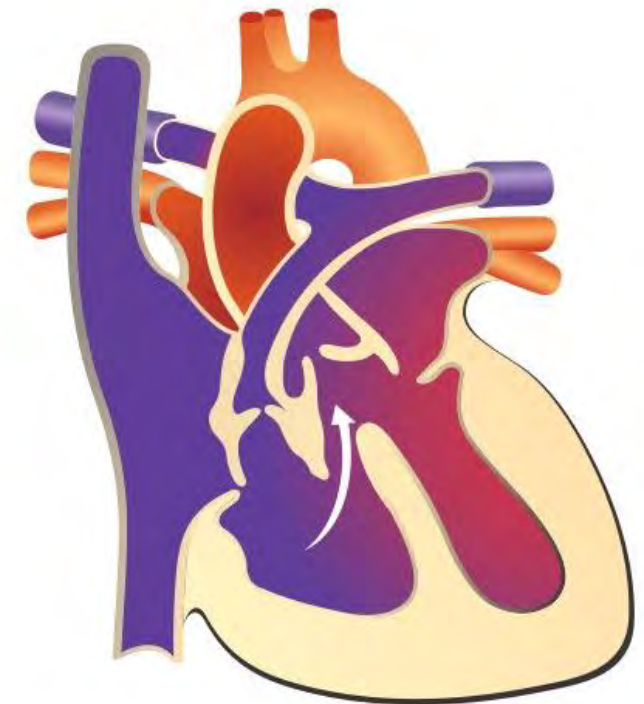


Pulmonic Regurgitation

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- Most common cause: repaired **Tetralogy of Fallot**
 - Repair of RVOT obstruction damages valve
- Endocarditis (rare)
- Rheumatic heart disease (rare)
- Diastolic decrescendo murmur at left upper sternal border

Tetralogy of Fallot

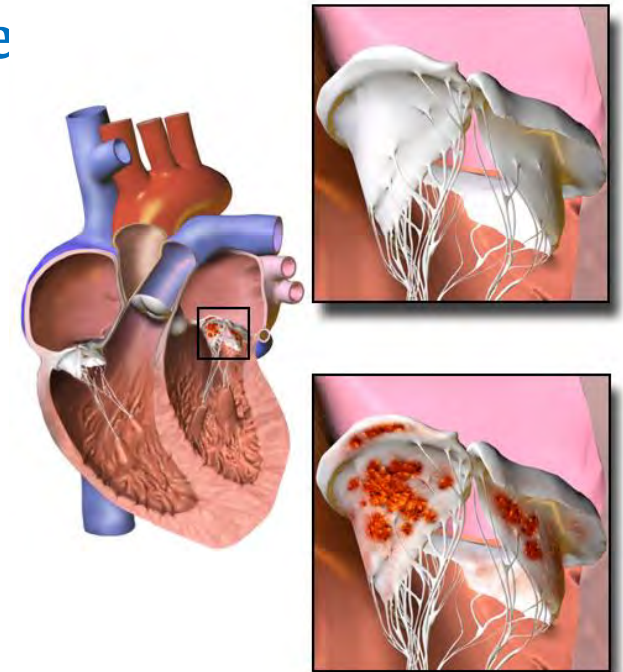


Endocarditis Prophylaxis

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- Antibiotics prior to dental work or surgical procedures
- Sometimes indicated in patients with valve disease
- Indicated in patients with **prior endocarditis**
- Indicated only after **valve replacement with prosthetic valve**
- NOT indicated for patients with unrepaired valve disease

Endocarditis



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Heart Murmurs

Jason Ryan, MD, MPH



Heart Murmurs

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- Cardiac sound heard with stethoscope
- Caused by **turbulent blood flow**
- May be normal or pathologic



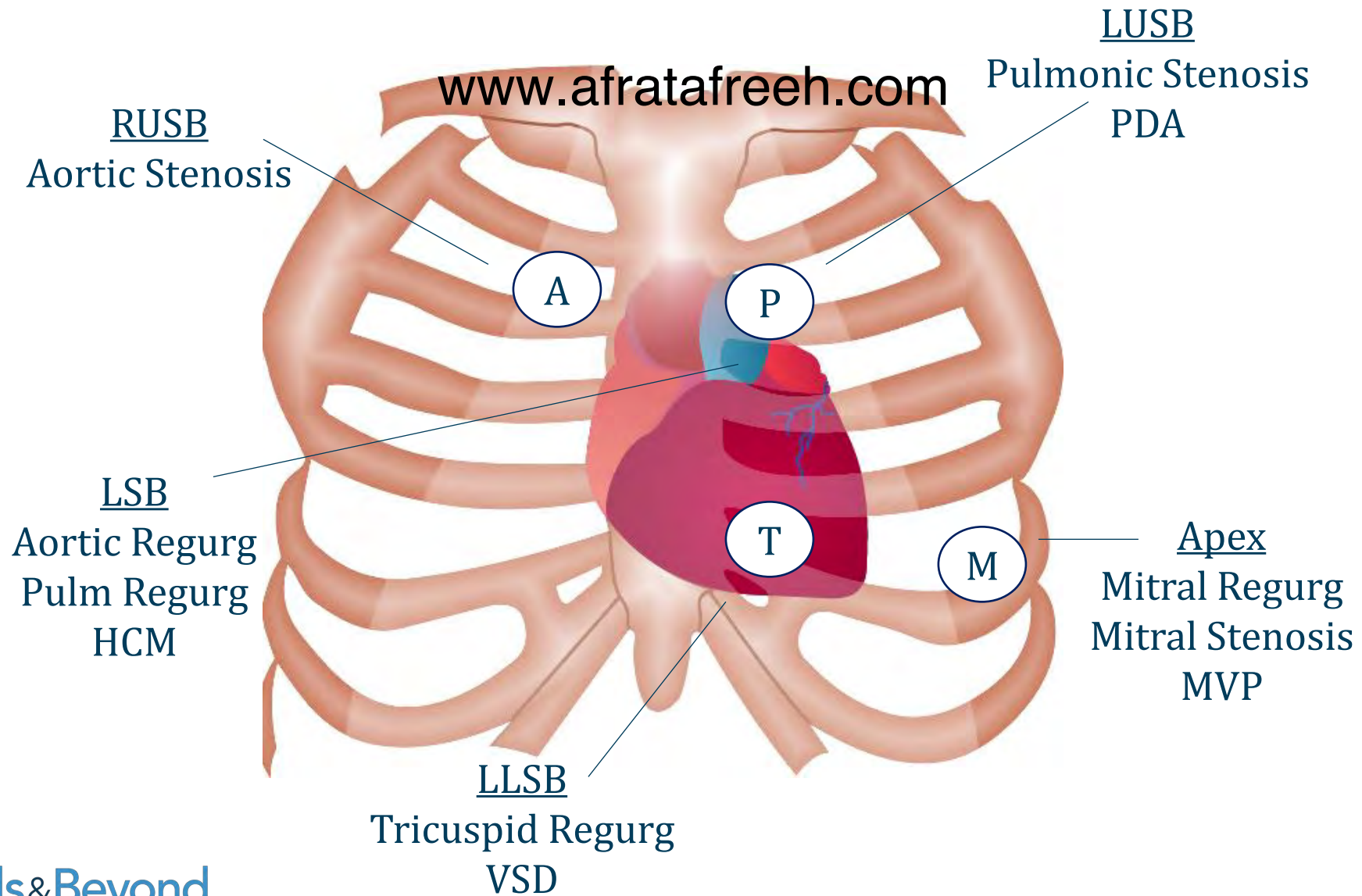
Murmurs

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Grading

- I - barely audible on listening carefully
- II - faint but easily audible
- III - loud and easily audible, no thrill
- IV - loud murmur with a thrill
- V - heard with scope barely touching chest
- VI - audible with scope not touching the chest

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Innocent/Functional Murmurs

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- Caused by normal flow of blood
- Common in children
- Also young, thin patients
- Generally soft murmurs
- No signs/symptoms of heart disease
- Still's murmur
- Pulmonic flow murmur
- Venous hum

Systolic Murmurs

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- Occur when heart contracts/squeezes
- Between S1-S2
- Flow murmur (benign)
- Aortic stenosis
- Mitral regurgitation
- Pulmonic stenosis
- Tricuspid regurgitation
- Hypertrophic cardiomyopathy
- Ventricular septal defect (VSD)

Diastolic Murmurs

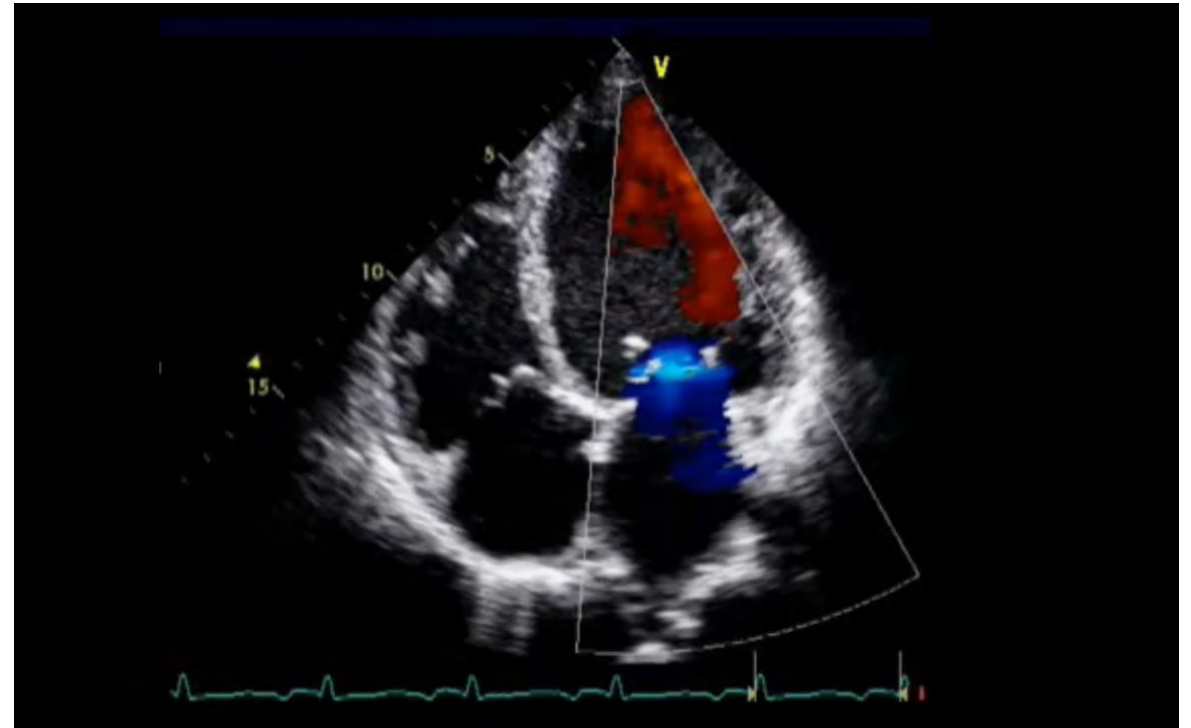
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- Occur when heart relaxes/fills
- Between S2-S1
- Aortic regurgitation
- Mitral stenosis
- Pulmonic regurgitation
- Tricuspid stenosis

Murmur Evaluation

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- Test of choice: **transthoracic echocardiography**
 - Visualize valves
 - Measure flow velocities

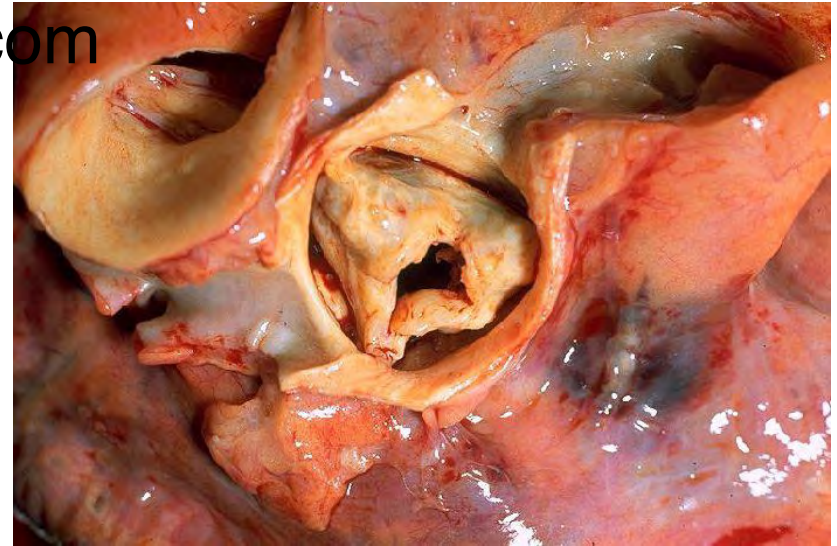
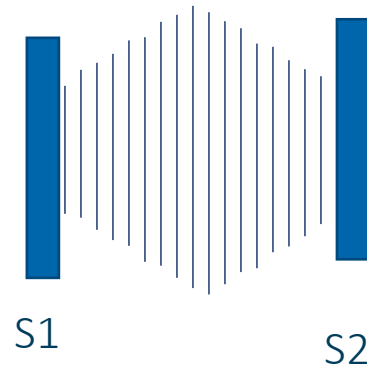


Aortic Stenosis

Murmur

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- Systolic crescendo-decrescendo murmur
- Also called an “ejection murmur”



Aortic Stenosis

Severe Disease Findings

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- **Late-peaking murmur**
 - Slow flow across stenotic valve
- **Soft/quiet S2**
 - Stiff valve can't slam shut
- **Pulsus parvus et tardus**
 - Weak and small carotid pulses
 - Delayed carotid upstroke

HCM

Hypertrophic Cardiomyopathy

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- Same murmur as aortic stenosis
- Differentiated by maneuvers
- **Valsalva**
 - Decreases venous return/preload
 - Increases HCM murmur
 - Decreases AS murmur



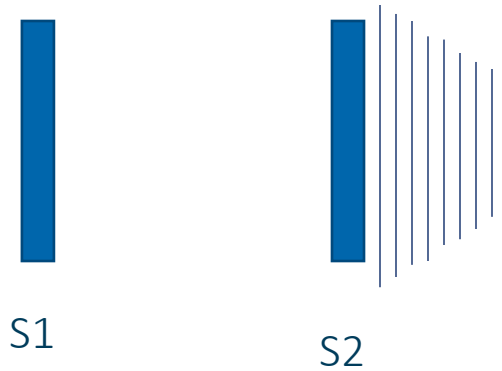
HCM

Aortic Regurgitation

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Aortic insufficiency, aortic incompetence

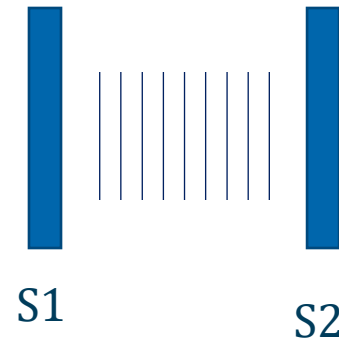
- Decrescendo, **blowing** diastolic murmur



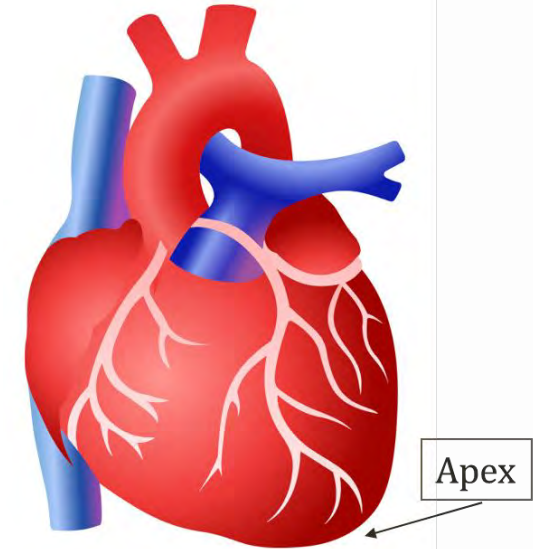
Mitral Regurgitation

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- **Holosystolic murmur heard best at the apex**
 - 5th intercostal space, mid-clavicular line



Holosystolic
(Pansystolic)

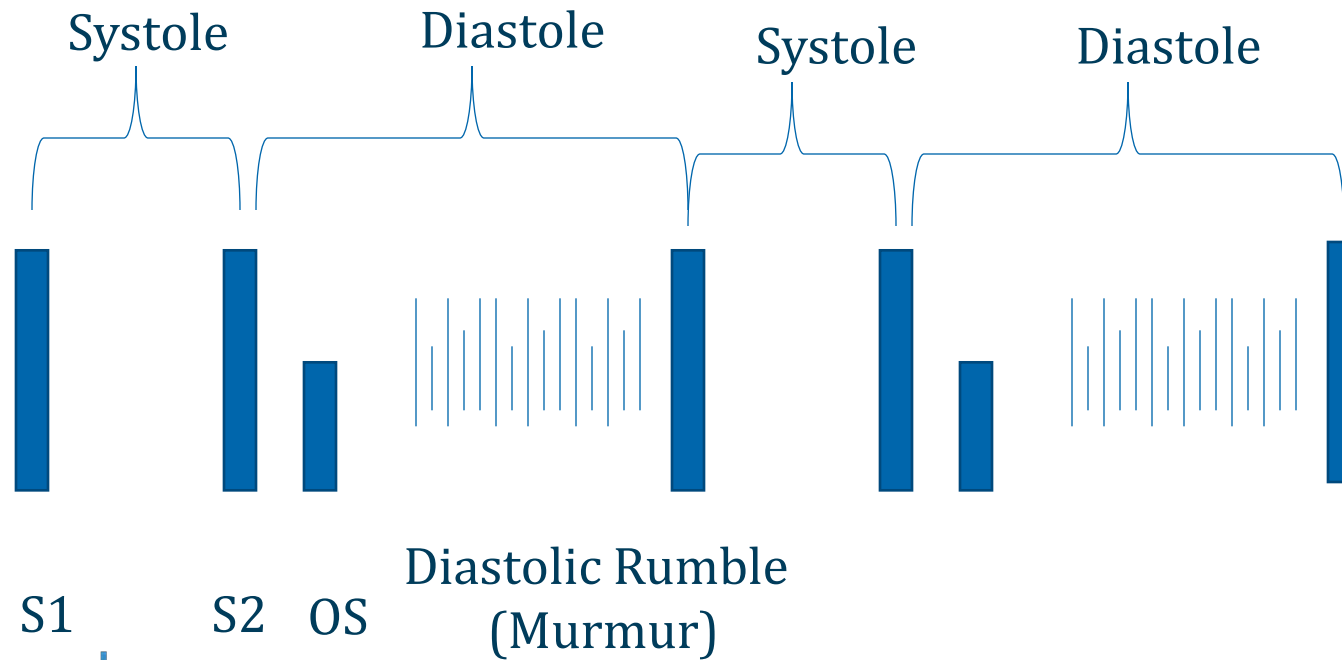


Mitral Stenosis

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- **Diastolic rumbling murmur** preceded by **opening snap**
- Loud S1



Mitral Stenosis

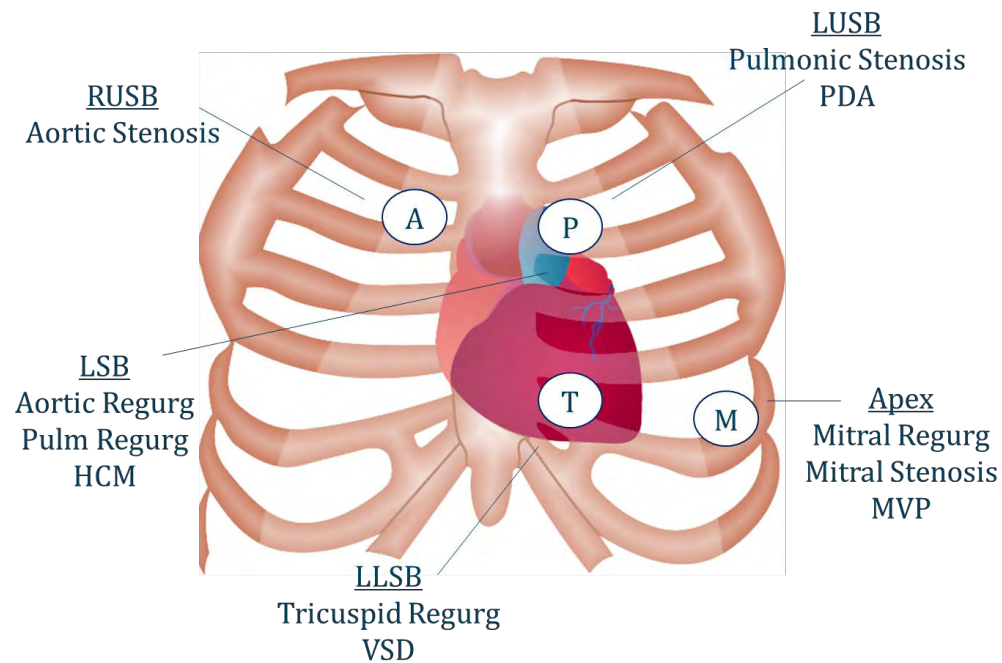
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- No left sided S3, S4 in mitral stenosis
- **Time to opening snap** associated with severity
 - **High left atrial pressure** in severe disease
 - Higher left atrial pressure → ↓ time to opening snap
 - Short time to opening snap seen in severe disease

Tricuspid/Pulmonic Disease

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- Valve lesions sound like left-sided counterparts
- Heard in different locations



Carvallo's Sign

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- Tricuspid regurgitation gets louder during inspiration
- Mitral regurgitation gets softer during inspiration
- Inspiration draws blood volume to lungs
- Louder right sided murmurs
- Softer left-sided murmurs
- Exhalation does the opposite
- **right-sided** murmurs increase with Inspiration
- **Left-sided** murmurs increase with Exhalation

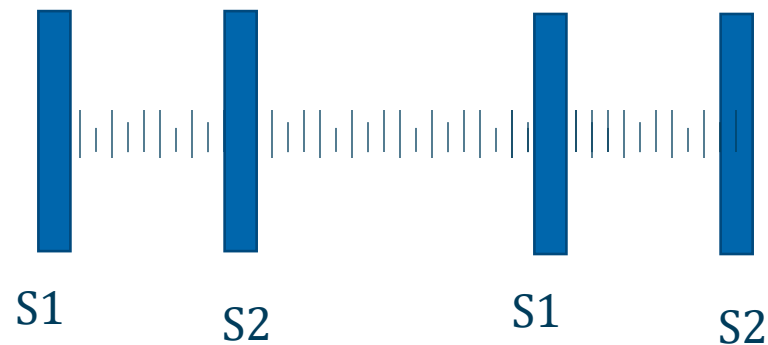
PDA

Patent Ductus Arteriosus

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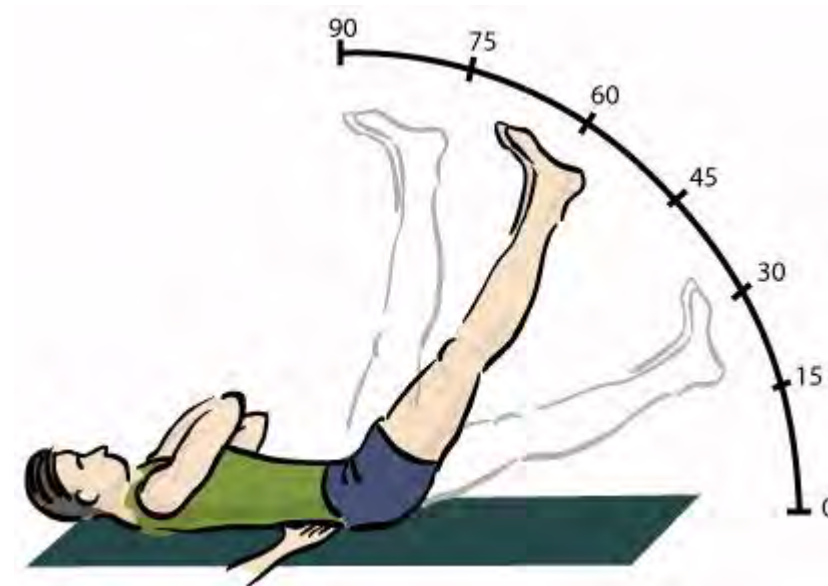
- Continuous, “machine-like” murmur



Maneuvers

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- Performed at bedside with patient
- May increase or decrease murmur
- Used to make diagnosis



Davidjr74/Wikipedia

Maneuvers

Preload/Venous Return

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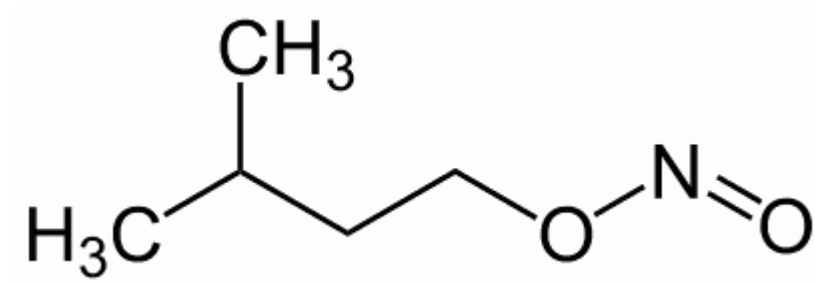
- Increase preload/venous return
 - **Leg raise** – blood falls back toward heart
 - **Squatting** – blood in legs forced back toward heart
- Decrease preload/venous return
 - **Valsalva** – $\uparrow$ intra-thoracic pressure $\rightarrow$ vein compression $\rightarrow \downarrow$ VR
 - **Standing** – Blood falls toward feet, away from heart
- Most murmurs INCREASE with more preload except:
 - HCM
 - MVP

Maneuvers

Afterload

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- Increase Afterload
 - **Hand grip** - clench fist
- Decrease Afterload
 - **Amyl Nitrate** - vasodilator



Amyl Nitrate

Maneuvers

Afterload

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- Backward Disorders
 - AR, MR, VSD
 - Louder with more afterload
- Forward Disorders
 - MS, AS
 - Softer with more afterload
- MVP, HCM
 - Softer
 - Increased LV cavity size

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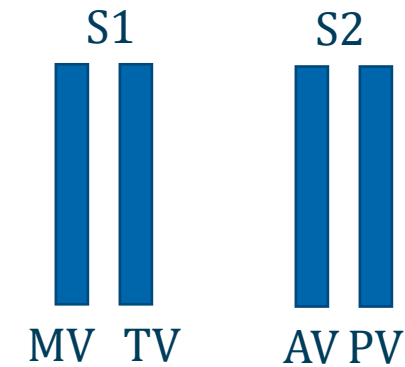
Heart Sounds

Jason Ryan, MD, MPH



S1 and S2

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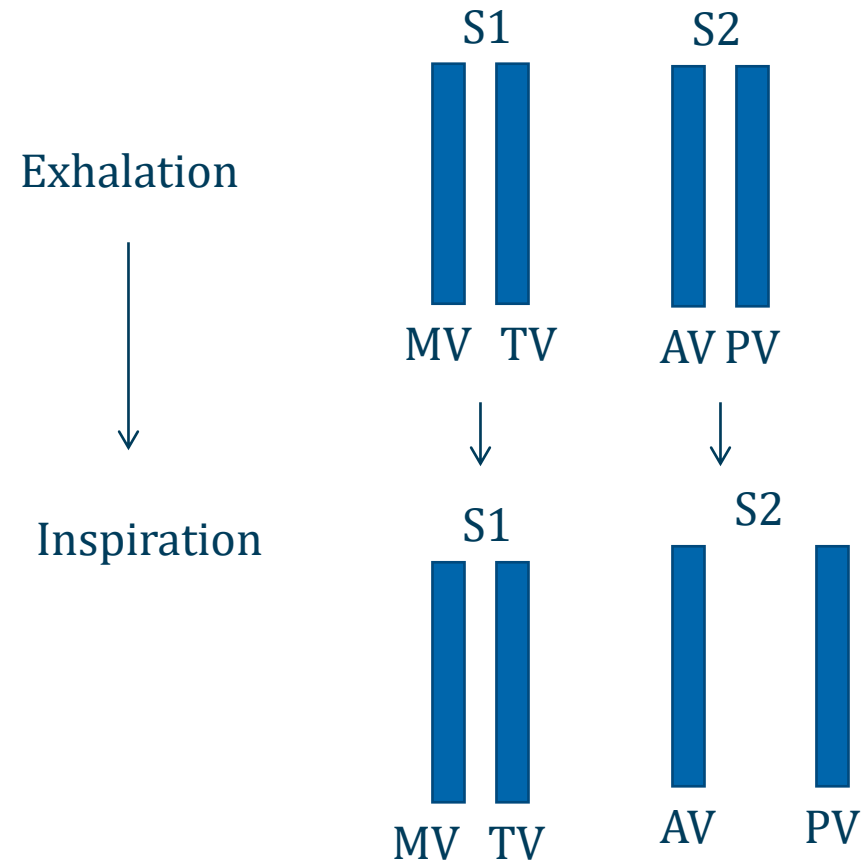


- **Normal** heart sounds
- Each has **two components**
 - One from left-sided valves (aortic, mitral)
 - One from right-sided valves (tricuspid, pulmonic)
- S1 usually “single”
 - Two components close together
 - Cannot distinguish separate sounds
- S2 can be “split”
 - Two components far enough apart to be audible



Physiologic S2 splitting

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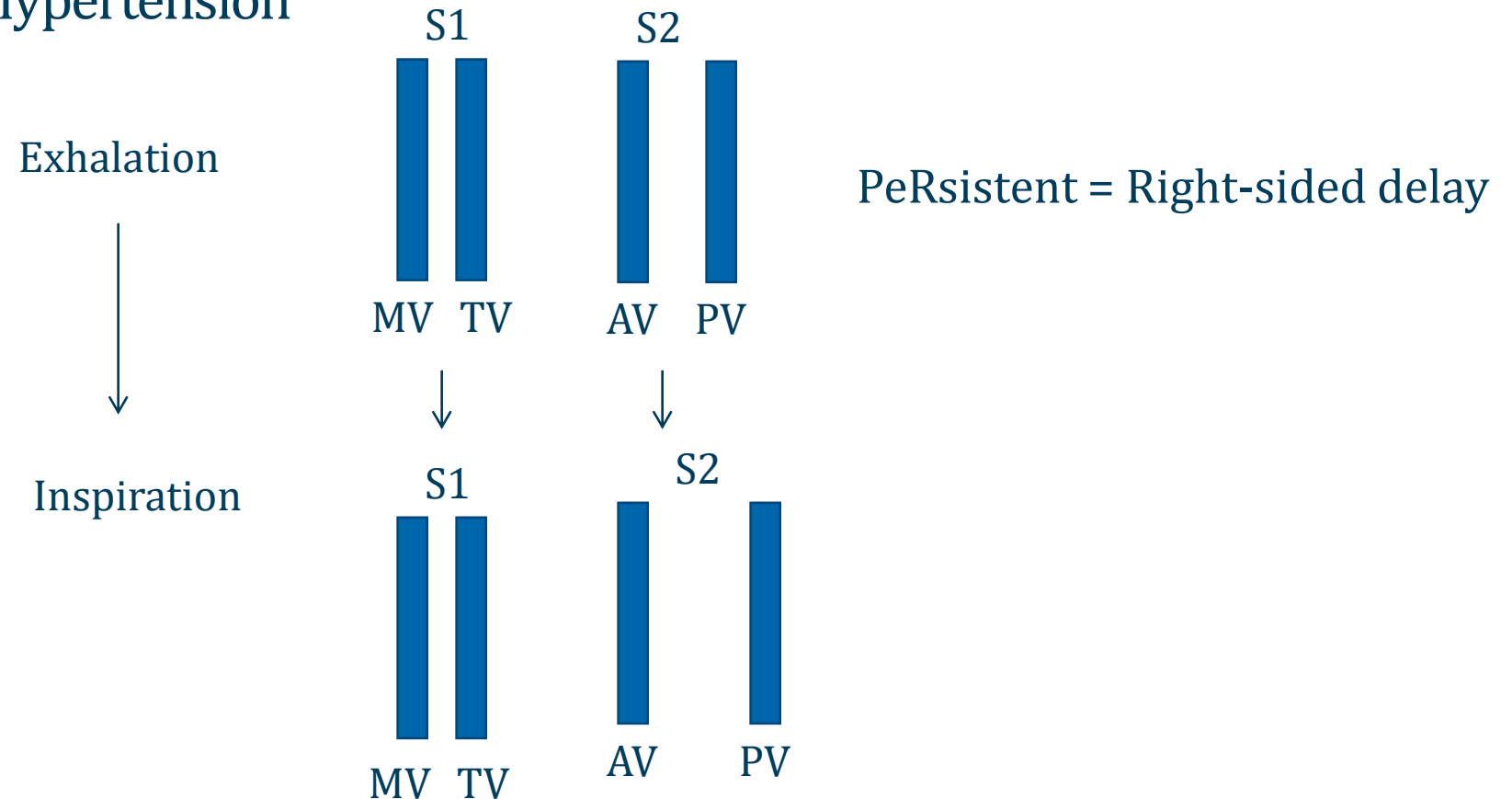


Increased venous return delays P2 by 40-60 ms
Single to split with inspiration

Persistent S2 splitting

RBBB or Pulmonary Hypertension

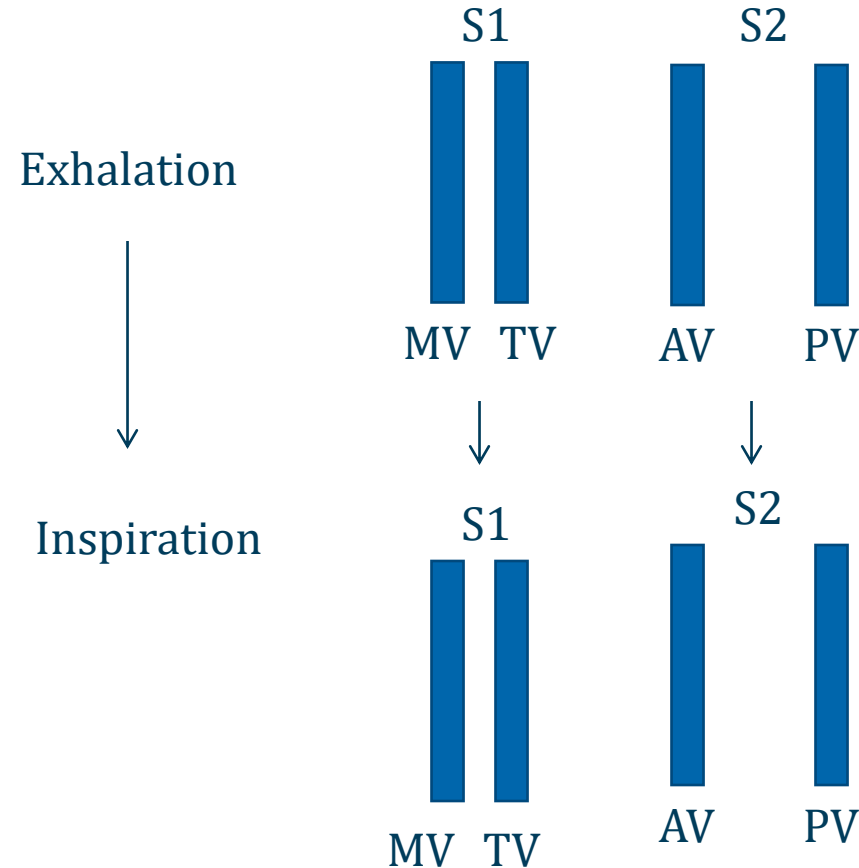
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Fixed S2 splitting

Atrial septal defect

www.afratafreeh.com



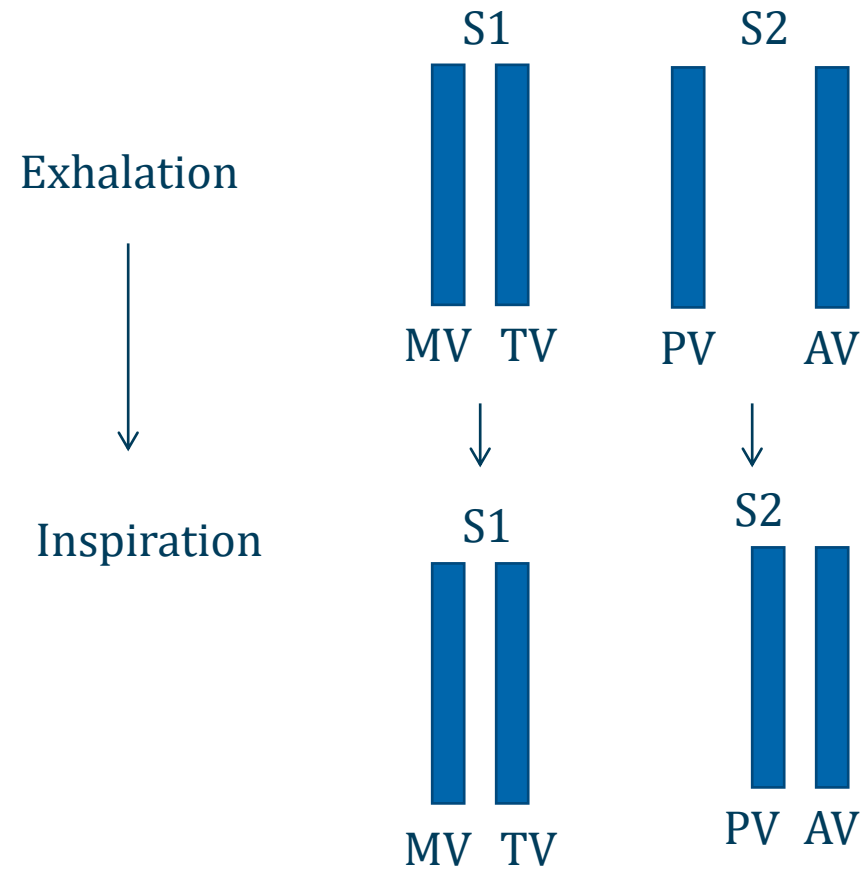
Atrial Septal Defect
Fixed split S2
Systolic Ejection Murmur LSB



Paradoxical S2 splitting

Delayed closure of aortic valve

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Paradoxical Splitting

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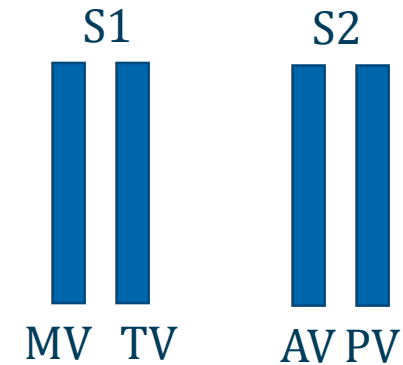
- Electrical causes → delayed LV activation
 - LBBB
 - RV pacing
- Mechanical causes → delayed LV outflow
 - LV systolic failure
 - Aortic stenosis
 - Hypertrophic cardiomyopathy

Paradoxical L = Left-sided delay

Loud P2

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- Loud pulmonic component of S2
- **Pulmonary hypertension**
- Forceful closure of pulmonary valve
- Normally P2 not heard at apex
 - If you hear it here, it's "loud"



S3 and S4

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- Pathologic/abnormal heart sounds
- Occur in **diastole** during filling of left ventricle
- **Low-pitched** sounds heard best with bell
- S3: Early filling sound
- S4: Late filling sound



Tama988/Wikipedia

S3

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- Commonly seen in **acute heart failure**
 - High LA pressure → rapid early filling of LV → S3
 - Associated with ↑ LAP & ↑ LVEDP
 - Very specific sign of high left atrial pressure
- **May be heard in normal hearts**
 - Young patients (< 30), pregnant women
 - Vigorous LV relaxation

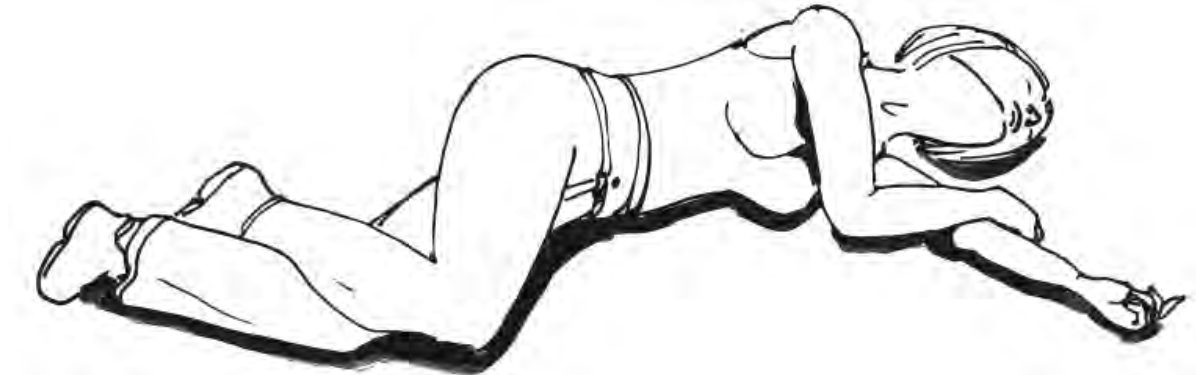


Wikipedia/Public Domain

S3

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- Louder in **left lateral decubitus position**
- Loudest at **apex**



S4

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- Heard in patients with **stiff left ventricle**
 - Long-standing hypertension
 - Hypertrophic cardiomyopathy
 - Diastolic heart failure
- Rapid late filling of LV due to atrial kick
- Not heard in **atrial fibrillation**



Systolic Clicks

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Ejection Click

Early in systole
BEFORE carotid pulse
Bicuspid Aortic Valve
Pulmonic stenosis



Non-Ejection Click

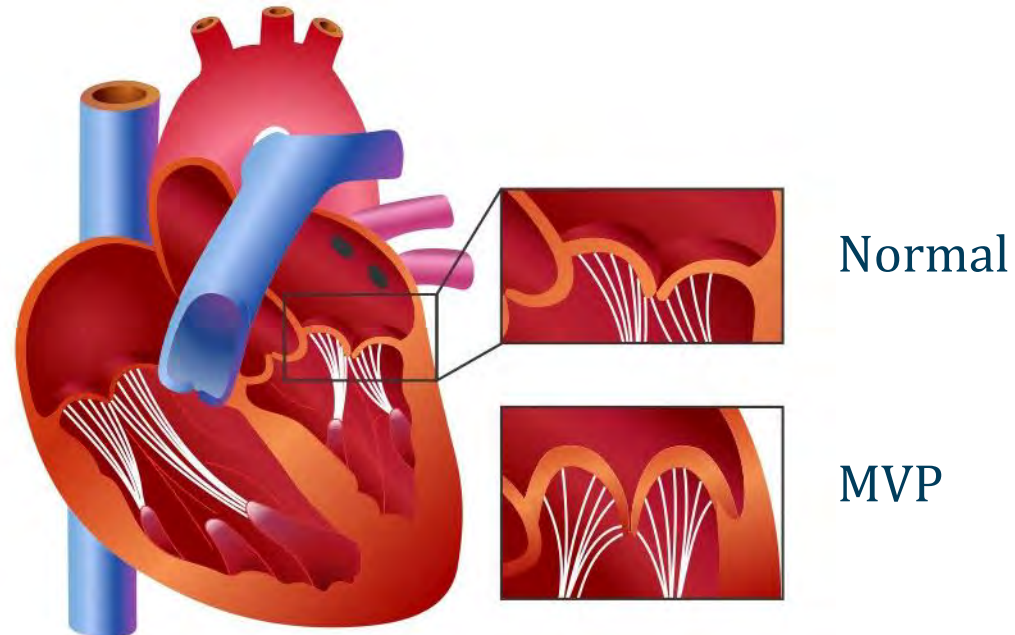
Late in systole
AFTER carotid pulse
Mitral Valve Prolapse

Mitral Valve Prolapse

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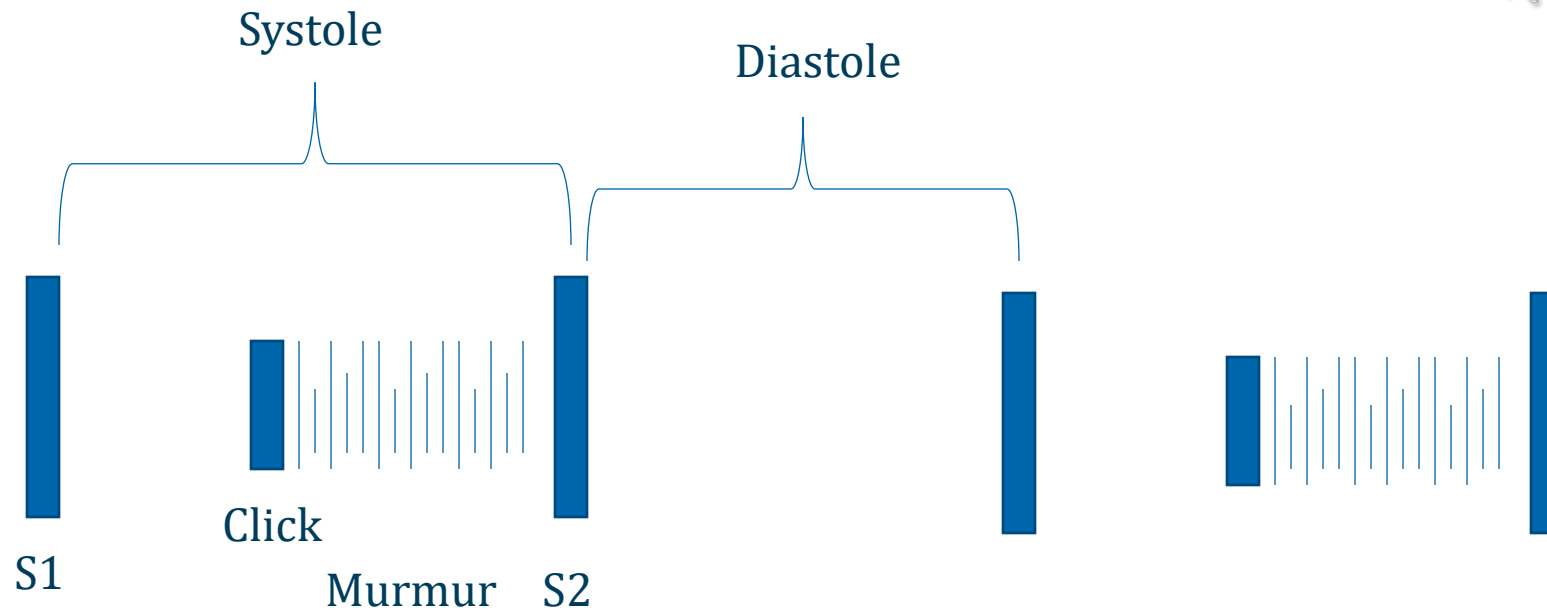
Classic Patients
Young women
Marfan syndrome

- Billowing of mitral valve leaflets above annulus
- Common cause of mitral regurgitation
- Causes a systolic click
 - Don't confuse with opening snap of mitral stenosis (diastole)



Mitral Valve Prolapse

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Murmur intensity increases with Valsalva

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Bradycardia

Jason Ryan, MD, MPH



Bradycardia

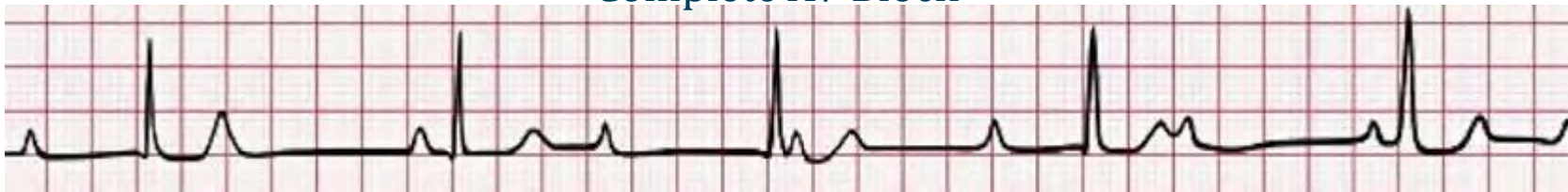
www.afratafreeh.com

- Pulse < 60/min
- Sinus bradycardia: slow SA node depolarization
- AV Block: blocked conduction through AV node

Sinus Bradycardia



Complete AV Block



Bradycardia

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Symptoms

- Often asymptomatic
- Symptoms with severe/persistent forms only
- Fatigue
- Exercise intolerance
- Dizziness
- Syncope

Sinus Bradycardia

www.afratafreeh.com

- Sinus rate $< 60/\text{min}$
- Often an incidental finding
- Drugs: beta-blockers, calcium channel blockers
- Well-trained athletes

Sinus Bradycardia



Sinus Bradycardia

www.afratafreeh.com

- Usually no treatment required
- Rare, severe cases treated with:
 - Atropine (muscarinic antagonist)
 - Dopamine or epinephrine (beta-1 agonists)
 - Pacemaker implantation

Sinus Bradycardia



Sinus Node Dysfunction

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Sick Sinus Syndrome

- Bradycardia due to abnormal SA node function
- Usually due to age-related changes
- Slow or absent SA node function after atrial fibrillation conversion
 - “Conversion pause”
- Treatment: **pacemaker implantation**

Atrial Fibrillation Conversion Pause



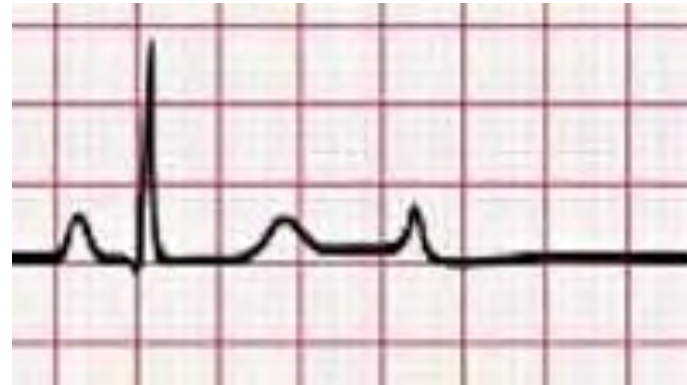
AV Block

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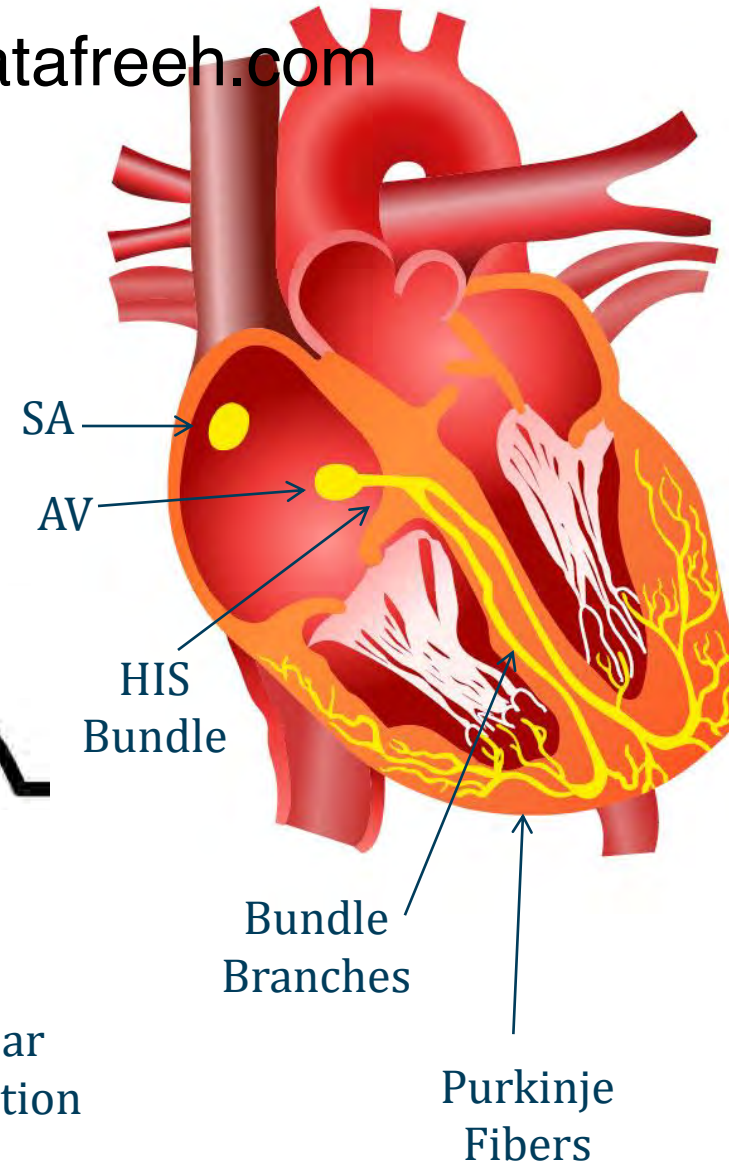
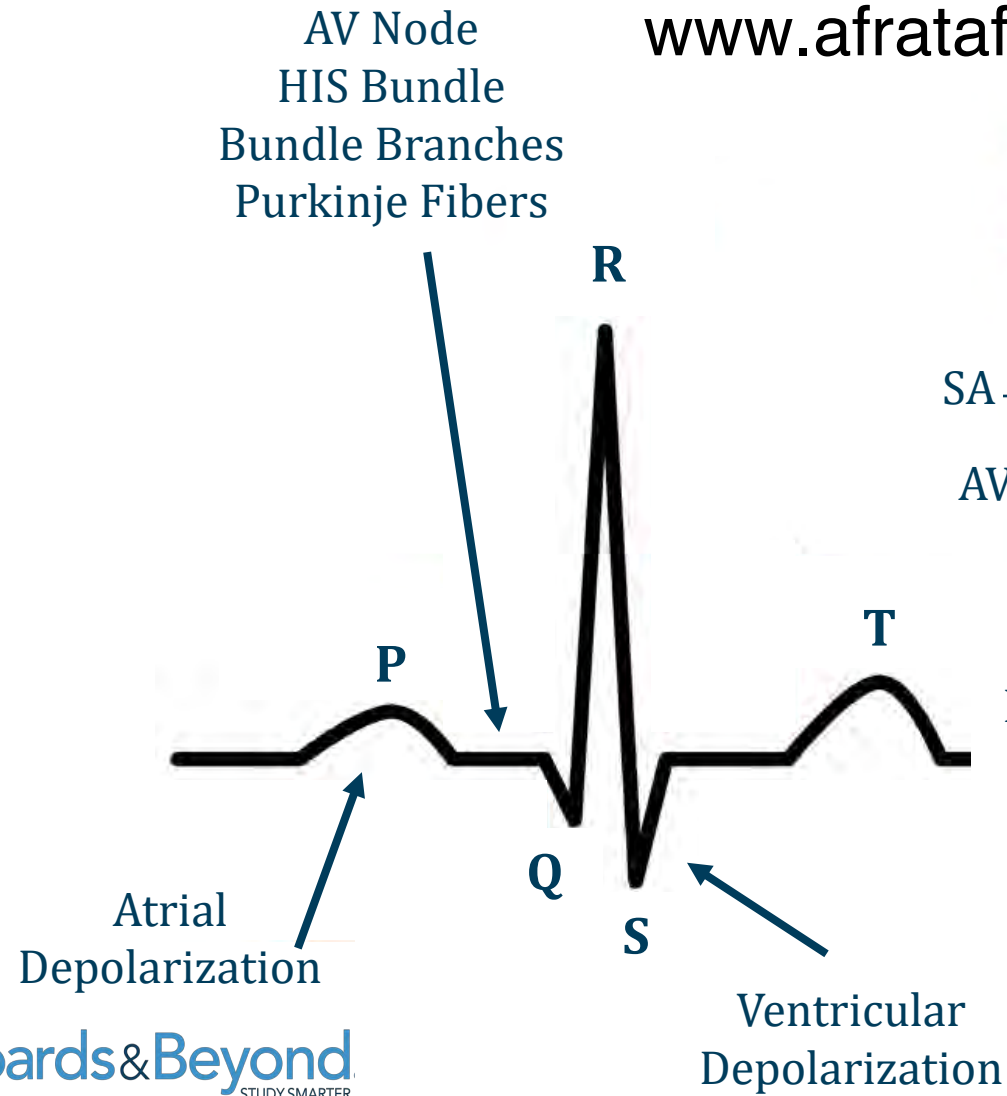
- Slowed or blocked conduction atria → ventricles
- Can cause prolonged PR interval
- Can cause non-conducted p wave



Prolonged PR Interval



Non-conducted P wave



AV Block

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Symptoms

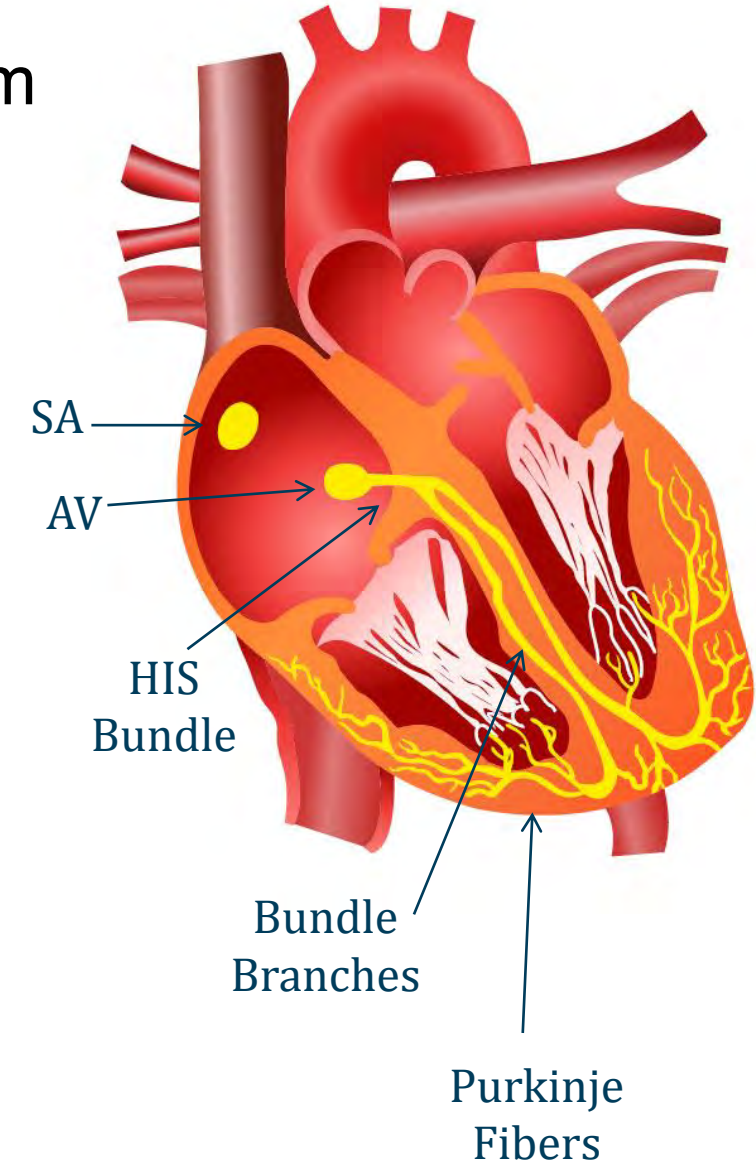
- Often incidentally noted on EKG
 - Especially milder forms with few/no non-conducted p waves
- Can cause bradycardia symptoms
 - Occurs when many or all p waves not conducted
 - Fatigue, dizziness, syncope
 - Symptomatic AV block often treated with a pacemaker

AV Blocks

Anatomy

www.afratafreeeh.com

- Caused by disease in AV conduction system
 - AV node → HIS → Bundle Branches → Purkinje fibers
- Divided into two causes
 - **AV node disease**
 - **HIS-Purkinje disease**

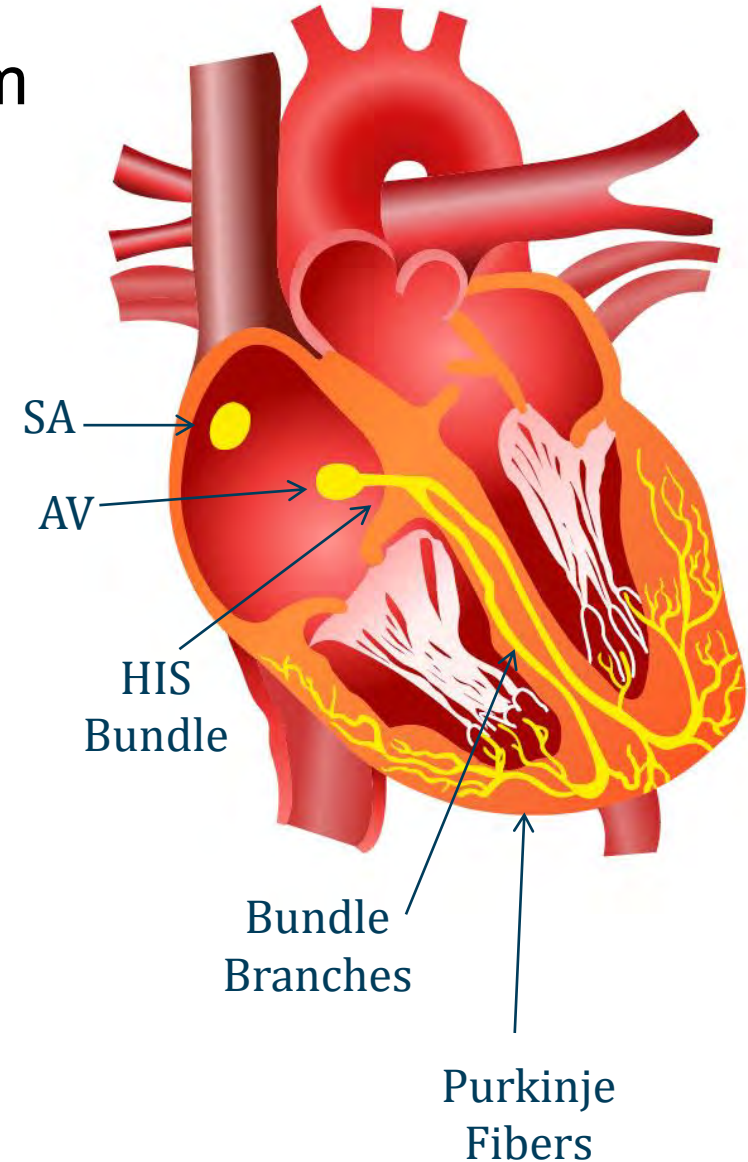


AV Blocks

Anatomy

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- **AV node disease**
 - Usually less dangerous
 - Conduction improves with exertion (sympathetic activity)
- **HIS-Purkinje disease**
 - More dangerous
 - Usually does not improve with exertion
 - Often progresses to complete heart block
 - Often requires a pacemaker



AV Blocks

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Four Types

- Type 1
 - **Prolongation of PR interval only**
 - All p waves conducted
- Type II
 - **Some p waves conducted**
 - **Some p waves NOT conducted**
 - Two sub-types: Mobitz I and Mobitz II
- Type III
 - **No impulse conduction** from atria to ventricles

1st degree AV Block

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Prolonged PR (normal < 200-210 ms)

Block usually in AV Node

Beta-blockers

Calcium channel blockers

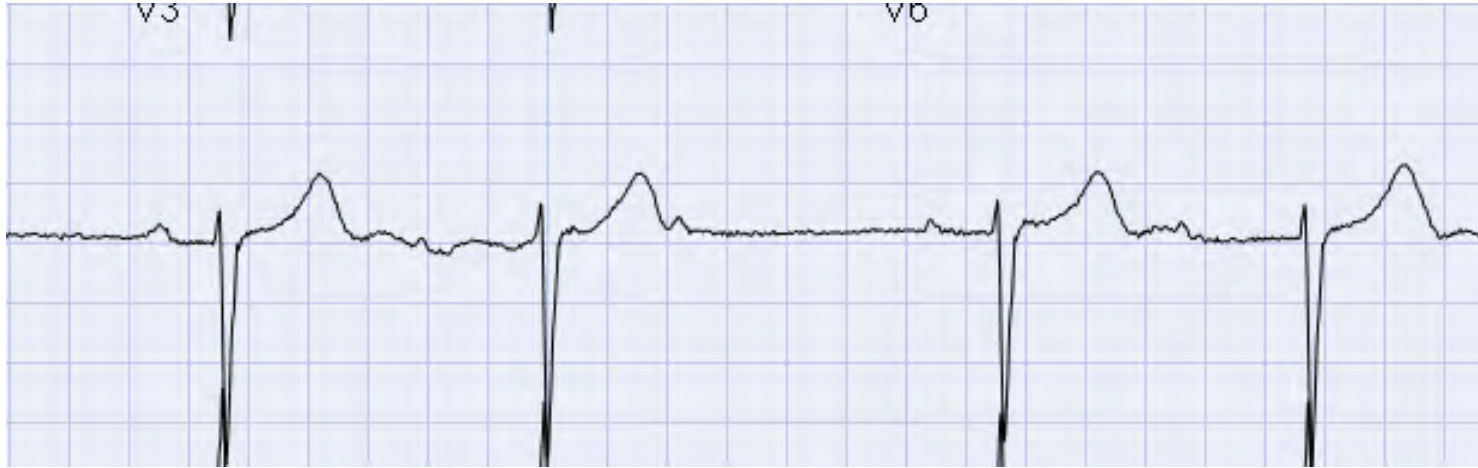
Well-trained athletes

Treatment: Usually none

2nd degree AVB

Mobitz I/Wenckebach

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Block usually in AV Node

Progressive PR prolongation

Grouped Beating

RR intervals NOT regular

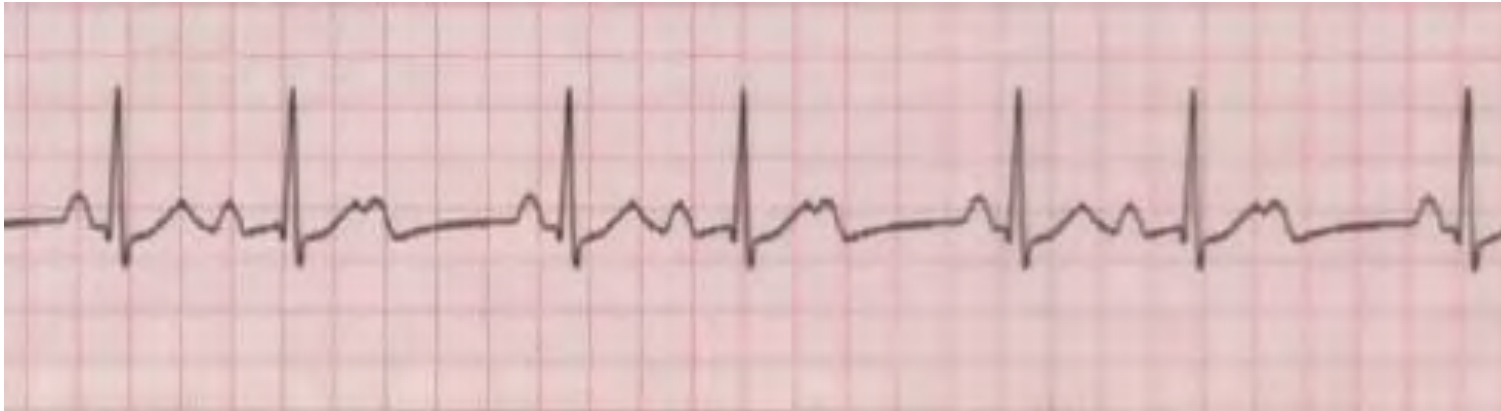
Similar causes as 1st degree AV block

Treatment: Usually none

2nd degree AVB

Mobitz I/Wenckebach

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Block usually in AV Node

Progressive PR prolongation

Grouped Beating

RR intervals NOT regular

Similar causes as 1st degree AV block

Treatment: Usually none

2nd degree AVB

Mobitz II

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Block usually in the HIS-Purkinje System

Often seen with bundle branch block

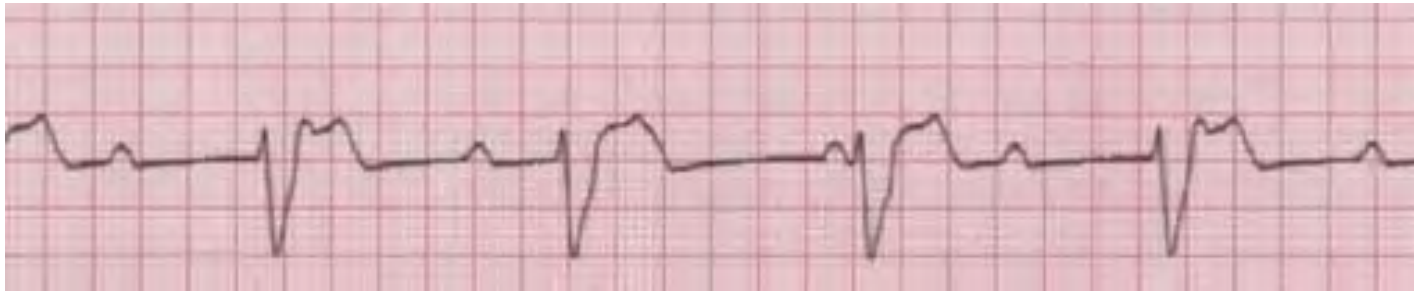
Usually symptomatic

Dizziness, syncope

Treatment: Pacemaker

3rd degree AVB

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Block usually in the HIS-Purkinje System

Regular RR intervals excludes Wenckebach

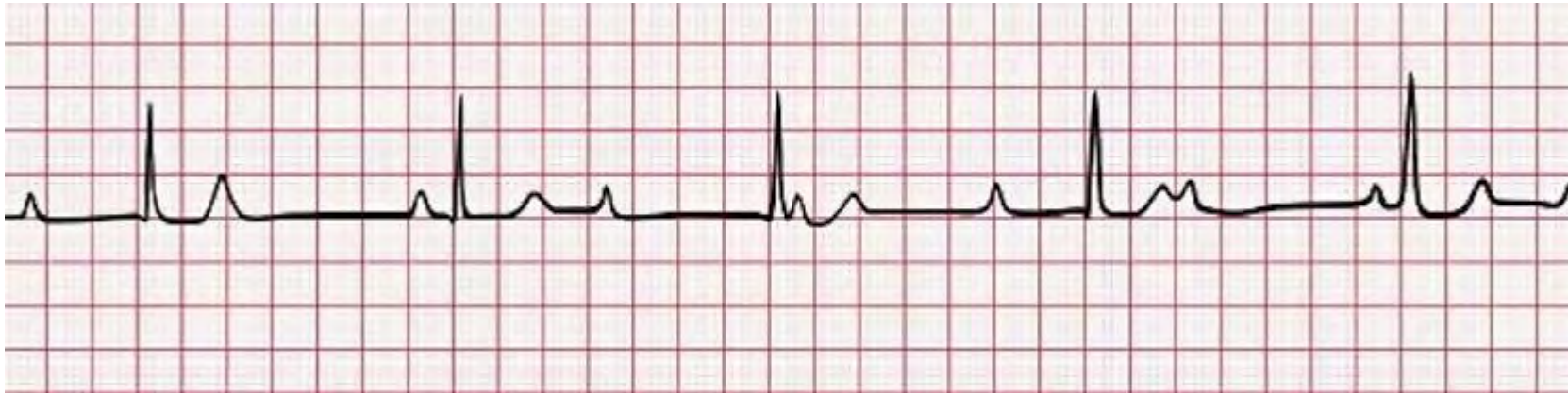
Usually symptomatic

Dizziness, syncope

Treatment: Pacemaker

3rd degree AVB

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Block usually in the HIS-Purkinje System

Regular RR intervals excludes Wenckebach

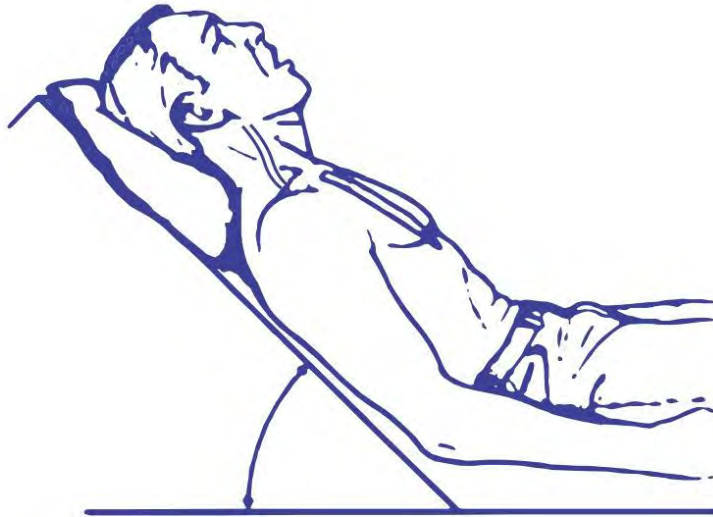
Usually symptomatic

Dizziness, syncope

Treatment: Pacemaker

Cannon a waves www.afratafreeh.com

- See in complete heart block (3rd degree)
- Caused by atrial contraction with closed tricuspid valve
- Visible as large venous pulsations



Lyme Disease

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- Spirochete infection with **Borrelia burgdorferi**
- Stage 2: Lyme carditis
- Varying degrees of AV block
 - 1st, 2nd, 3rd
- AV block improves with antibiotics



Image courtesy of Wikipedia/Public Domain

Causes of AV Block

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- Drugs
 - Beta-blockers, calcium channel blockers
 - Digoxin
- Athletes
 - At rest: sinus bradycardia plus slow AV node conduction
- **Fibrosis and sclerosis of conduction system**

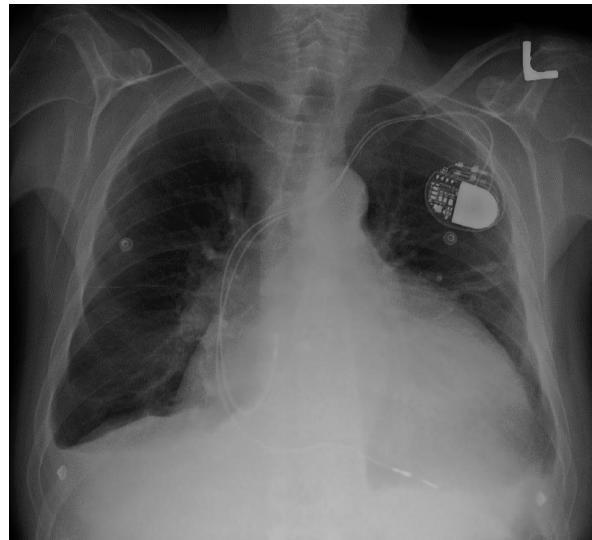


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Pacemaker

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- Treatment for sinus node dysfunction
- Also “high-grade” AV block
 - Usually Mobitz II or 3rd degree
- Often in patients with symptoms (syncope, dizziness)



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Atrial Fibrillation and Flutter

Jason Ryan, MD, MPH



Atrial Fibrillation

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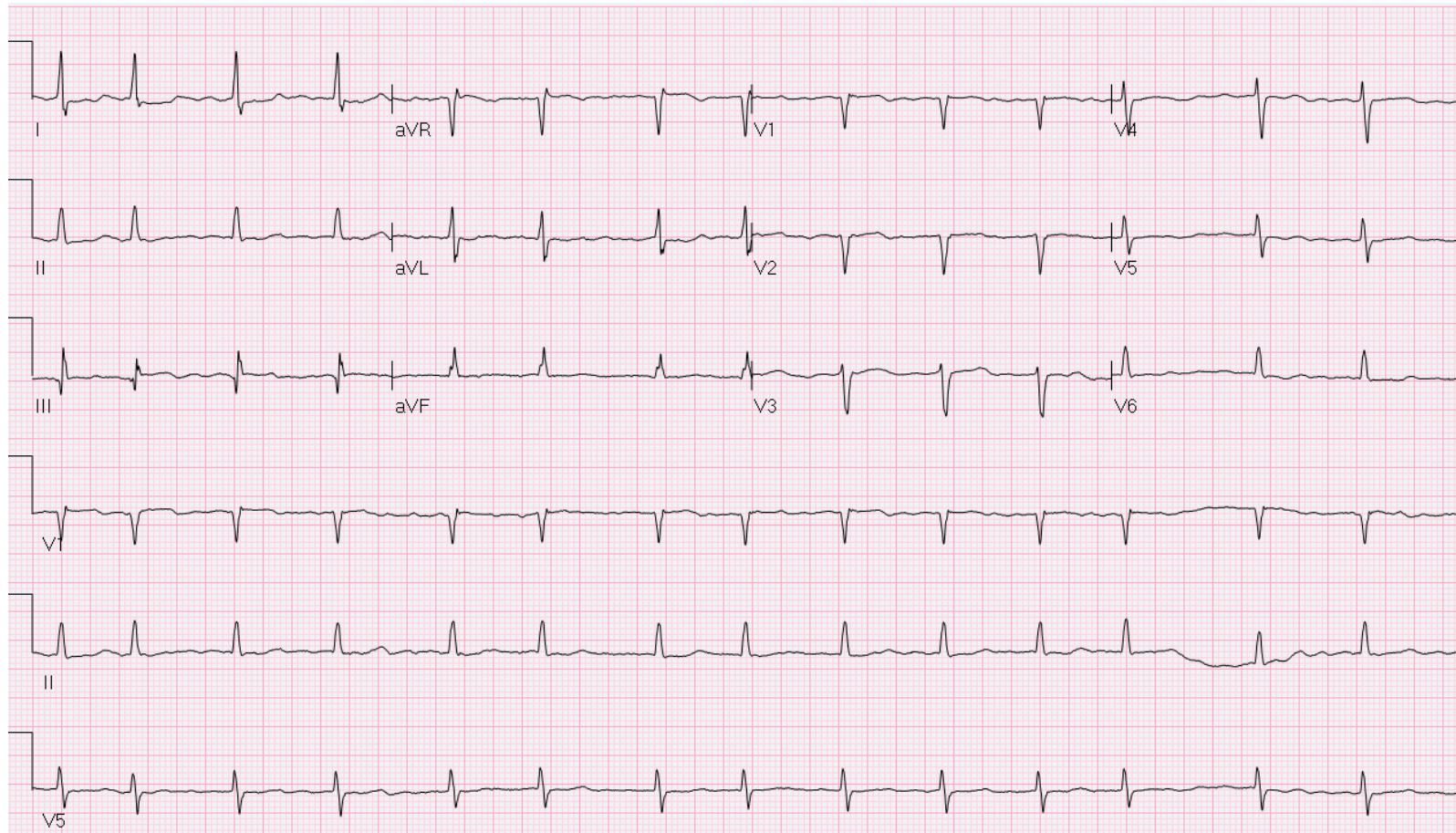
- Cardiac arrhythmia
- Results in an **irregularly, irregular** pulse
- Can cause palpitations, fatigue, dyspnea
- Diagnosis: EKG



J. Heuser/Wikipedia

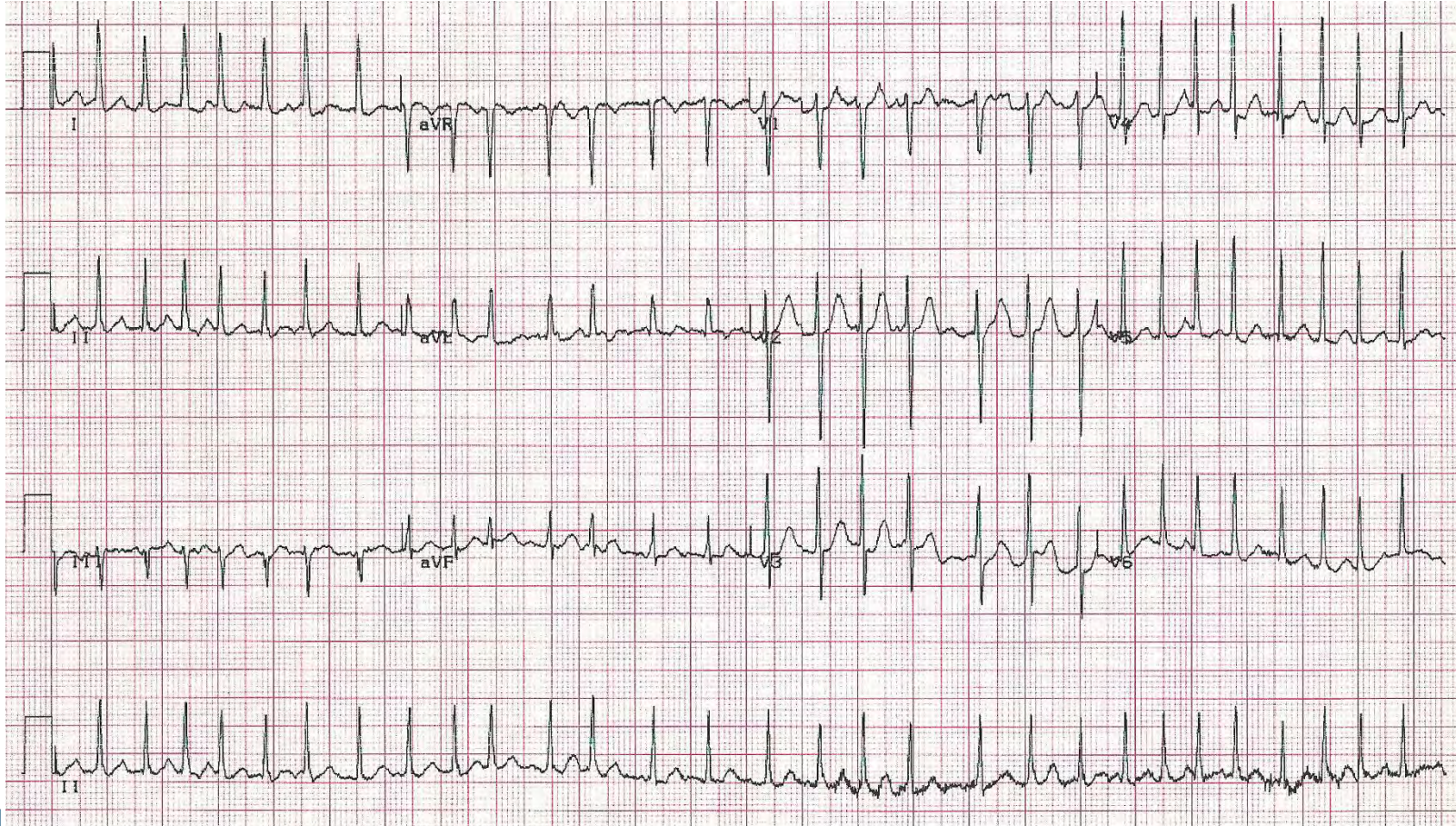
Atrial Fibrillation

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Atrial Fibrillation

www.afratafreeh.com



Atrial Fibrillation

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Terminology

- **Paroxysmal**
 - Comes and goes; spontaneous conversion to sinus rhythm
- **Persistent**
 - Lasts days/weeks; often requires cardioversion
- **Permanent**

Atrial Fibrillation

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Symptoms

- Wide spectrum of symptoms



Asymptomatic

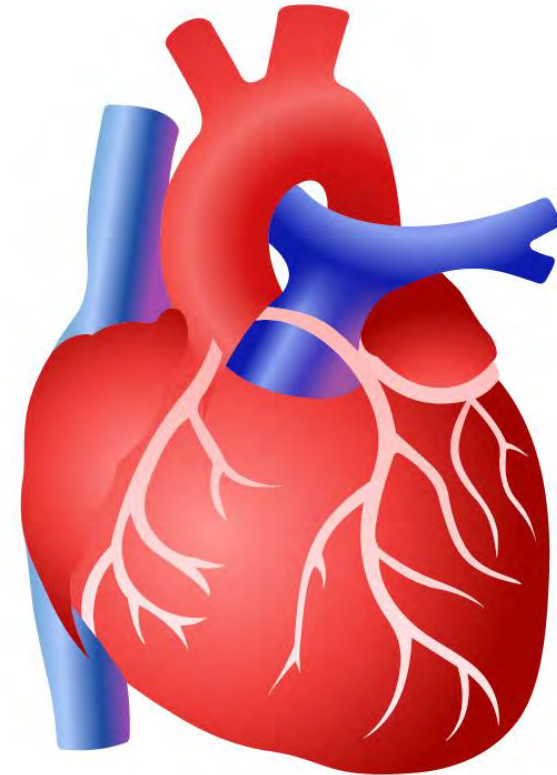
Heart Rate < 100 bpm

Palpitations, Dyspnea, Fatigue

Heart Rate > 100 bpm

Cardiomyopathy www.afratafreeh.com

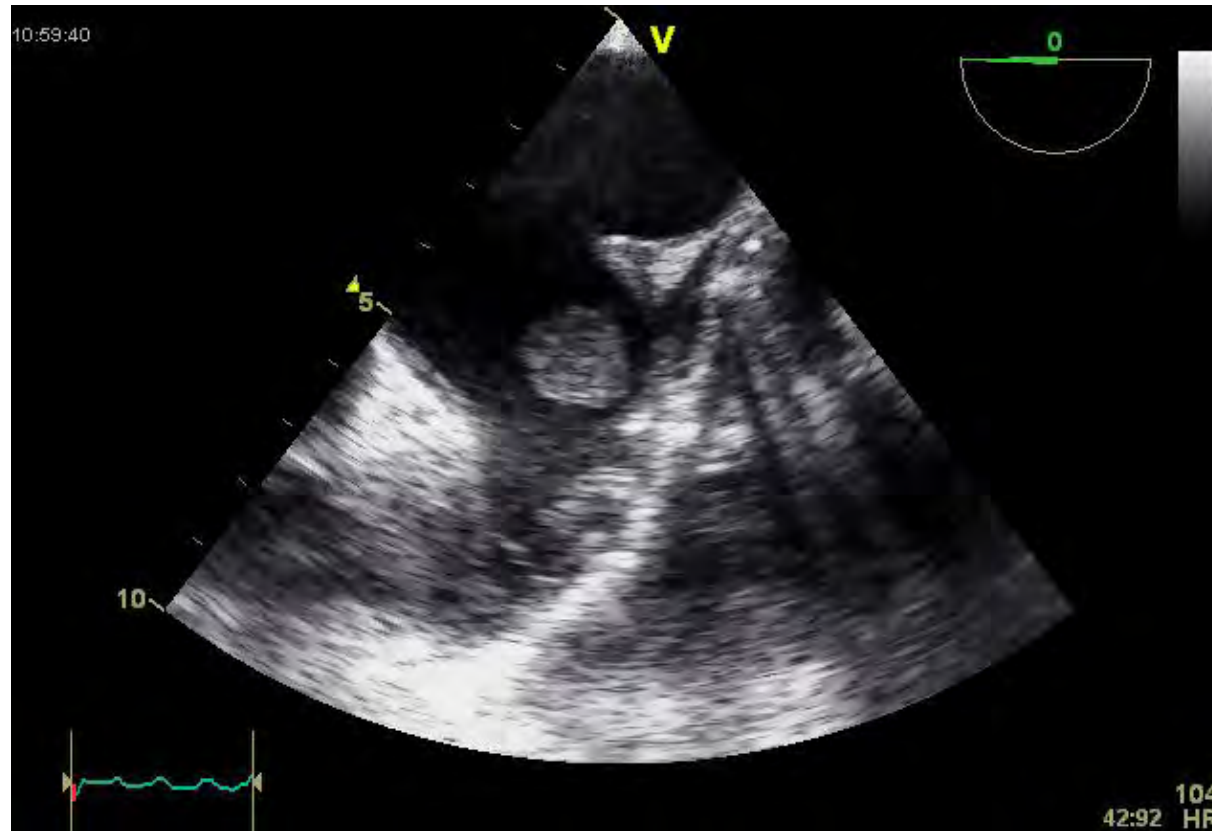
- Caused by untreated, **rapid** atrial fibrillation
- “Tachycardia-induced cardiomyopathy”
- ↓ LVEF
- Systolic heart failure



Atrial Fibrillation

Thrombus in Left Atrial Appendage

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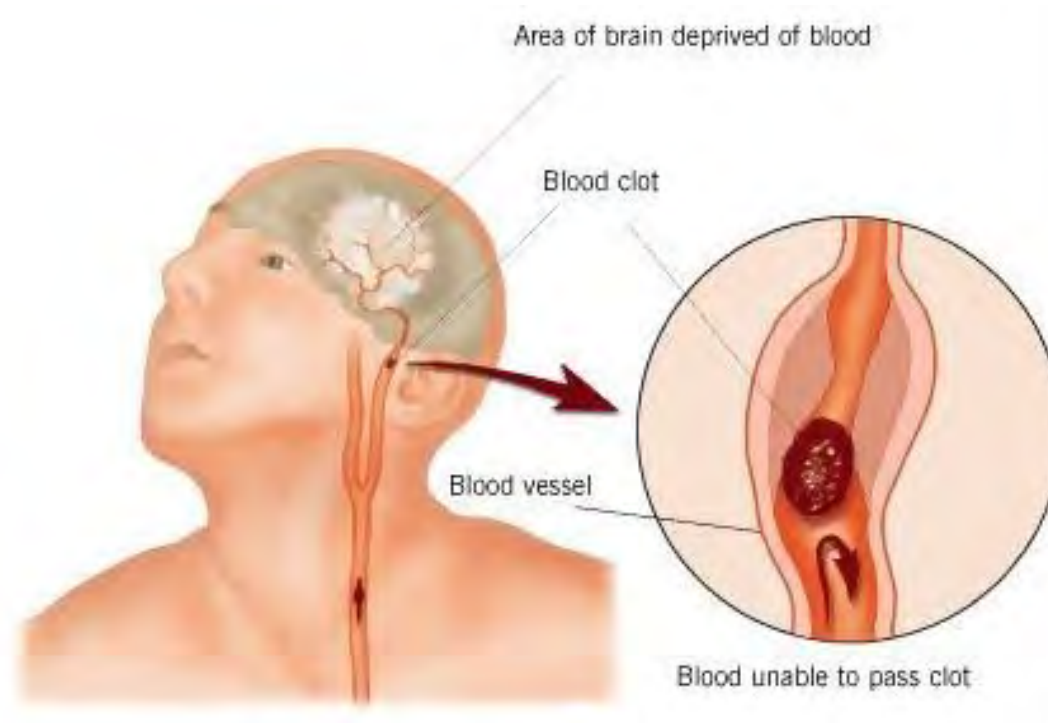


Atrial Fibrillation

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Cardiac Embolism

- Brain (stroke)
- Gut (mesenteric ischemia)
- Spleen



Atrial Fibrillation

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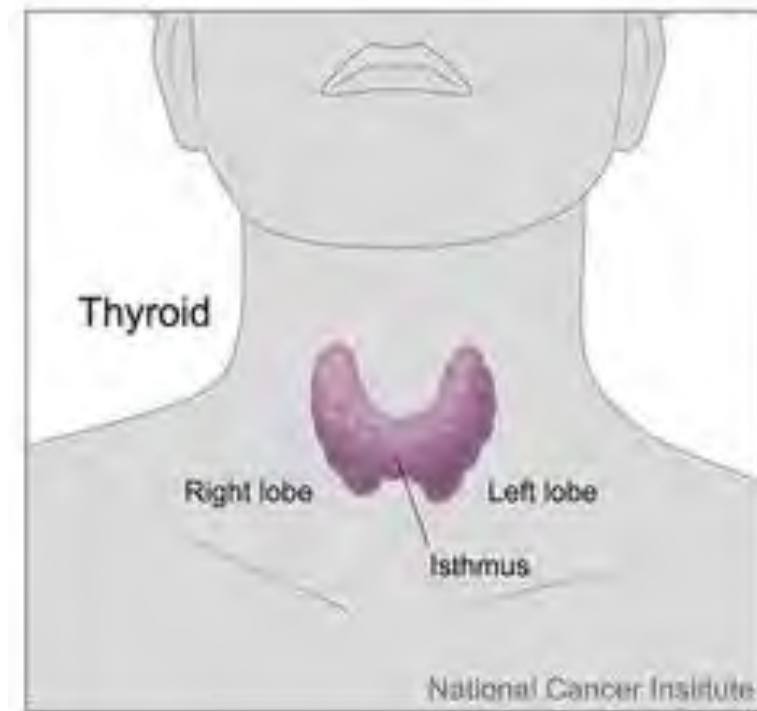
Risk Factors

- **Age**
 - ~10% of patients > 80
 - < 1% of patients < 55
- More common in women
- Most common associated disorders: **HTN, CAD**
- Anything that dilates the atria → atrial fibrillation
 - Heart failure
 - Valvular disease
- Key diagnostic test: **Echocardiogram**

Hyperthyroidism

www.afratafreeh.com

- Commonly leads to atrial fibrillation
- Reversible with therapy for thyroid disease
- Atrial fibrillation therapies less effective
- Key diagnostic test: **TSH**



Atrial Fibrillation

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Triggers

- Often no trigger identified
- Binge drinking (“holiday heart”)
- Increased catecholamines
 - Infection
 - Surgery
 - Pain



Public Domain

Atrial Fibrillation

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Treatment

- **Rate control**
 - Control of heart rate
 - Ideally < 110 bpm
- **Rhythm control**
 - Restoration of sinus rhythm
- **Anticoagulation**

Rate Control

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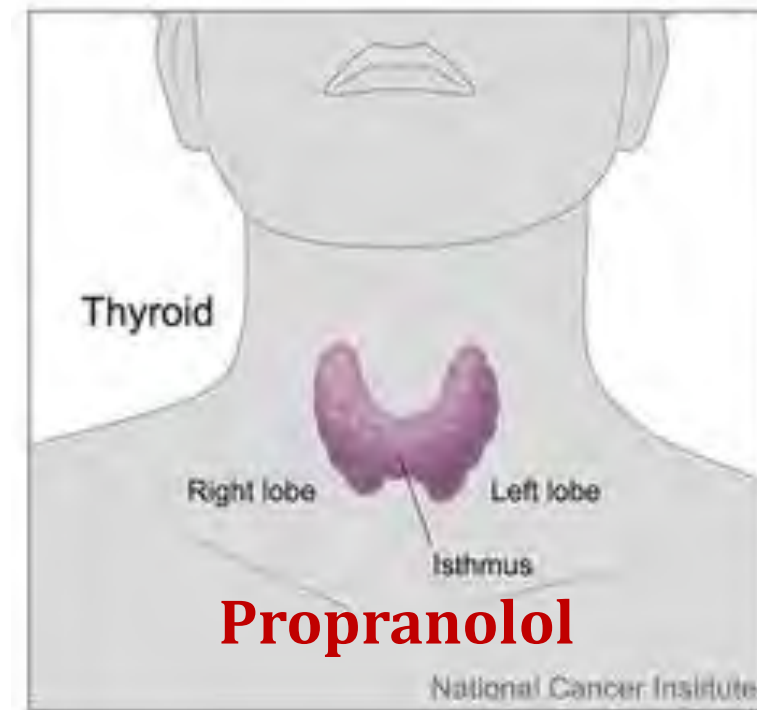


Beta Blockers
Calcium Channel Blockers
Digoxin

Rate Control

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- **Slow AV node conduction**
- Beta-blockers
 - Usually β_1 -selective agents
 - Metoprolol, Atenolol
 - Hyperthyroid patients: propranolol
- Calcium channel blockers
 - Verapamil, Diltiazem
- Digoxin
 - Increases parasympathetic tone to heart



Rhythm Control www.afratafreeh.com

- Goal: restore sinus rhythm



Cardioversion

Cardioversion

www.afratafreeh.com

- **Electrical**
 - Deliver “synchronized” shock at time of QRS
 - Administer anesthesia
 - Deliver electrical shock to chest
 - All myocytes depolarize
 - Usually sinus node first to repolarize/depolarize



Cardioversion

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- **Chemical**
 - Administration of antiarrhythmic medication
 - Often Ibutilide (class III antiarrhythmic)
 - Less commonly used due to drug toxicity

Cardioversion

www.afratafreeh.com

- **Spontaneous**
 - Often occurs after hours/days



Cardioversion

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Risk of Stroke

- Chemical/electrical cardioversion may cause stroke
- 48 hours required for thrombus formation
- Symptoms < 48 hours: cardioversion safe
- Symptoms > 48 hours (or unsure)
 - Anti-coagulation 3 weeks → cardioversion
 - Transesophageal echocardiogram to exclude thrombus
- Exception: Hypotension/shock
 - Emergent cardioversion performed

Rhythm Control www.afratafreeh.com

- **Antiarrhythmic medications**
- Administered before/after cardioversion
- Class I drugs
 - Flecainide, propafenone
- Class III drugs
 - Amiodarone, sotalol, dofetilide
- AFFIRM trial: no mortality difference between rate and rhythm control

Stroke Prevention

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- Warfarin
 - Requires regular INR monitoring
 - Goal INR usually 2-3
- Rivaroxaban, Apixaban
 - Factor Xa inhibitors
- Dabigatran
 - Direct thrombin inhibitor
- Aspirin
 - Less effective
 - Only used if risk of stroke is very low
 - Less risk of bleeding

Anticoagulation www.afratafreeh.com

- Anticoagulation MUST be administered
- Does not matter whether atrial fibrillation persists, or sinus rhythm restored
- Studies show similar stroke risk for rate control versus rhythm control

Stroke Risk

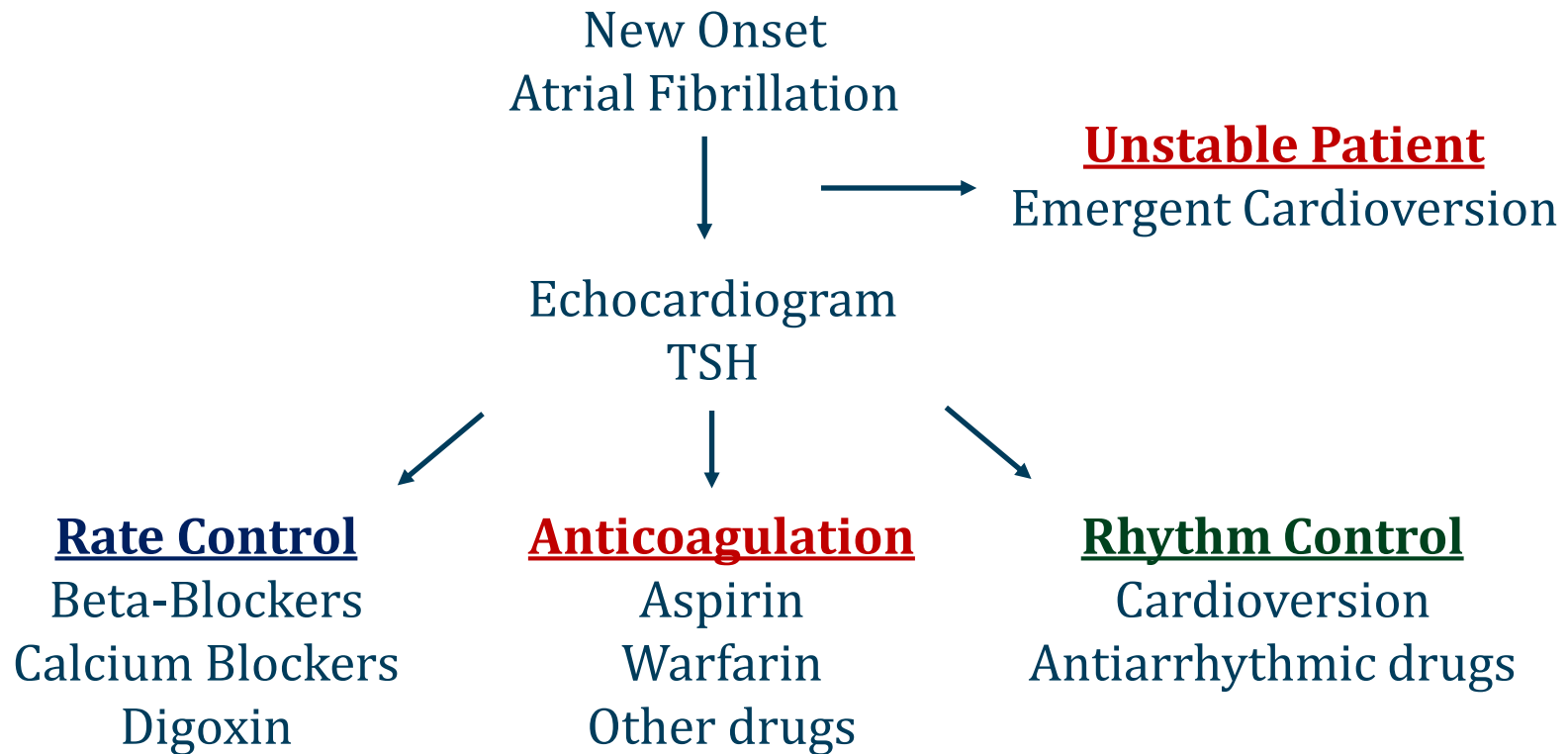
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- **CHADS VASC Score**
 - CHF (1 point)
 - HTN (1 point)
 - Diabetes (1 point)
 - Stroke (2 points)
 - Female (1 point)
 - Age 65-75 (1 point)
 - Age > 75yrs (2 points)
 - Vascular disease (1 point)
- Score ≥ 2 = Warfarin or other anticoagulant
- Score 0 -1 = Aspirin or no therapy

Atrial Fibrillation

Summary

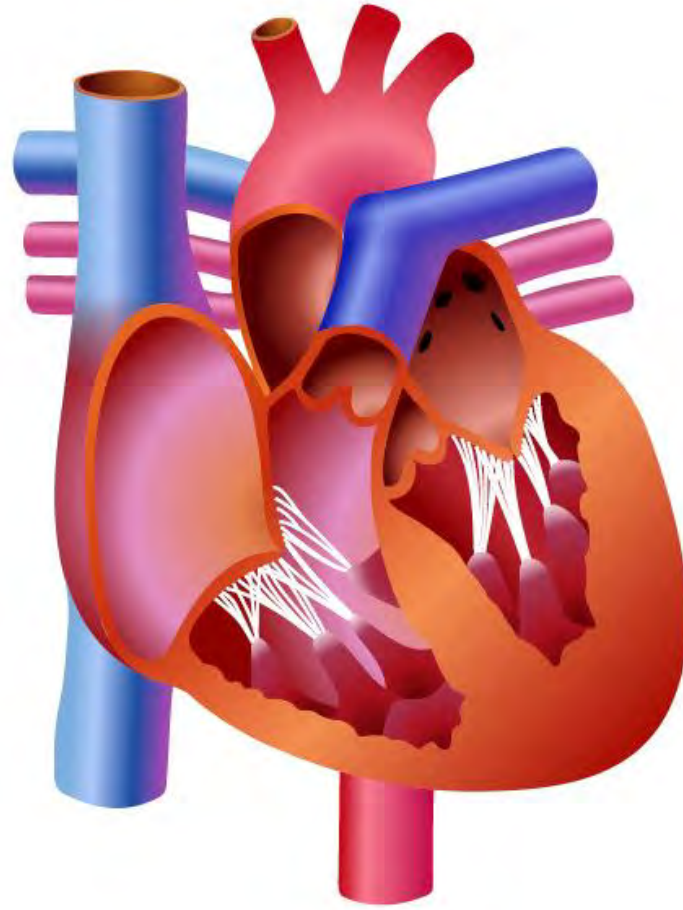
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Pulmonary Vein Isolation

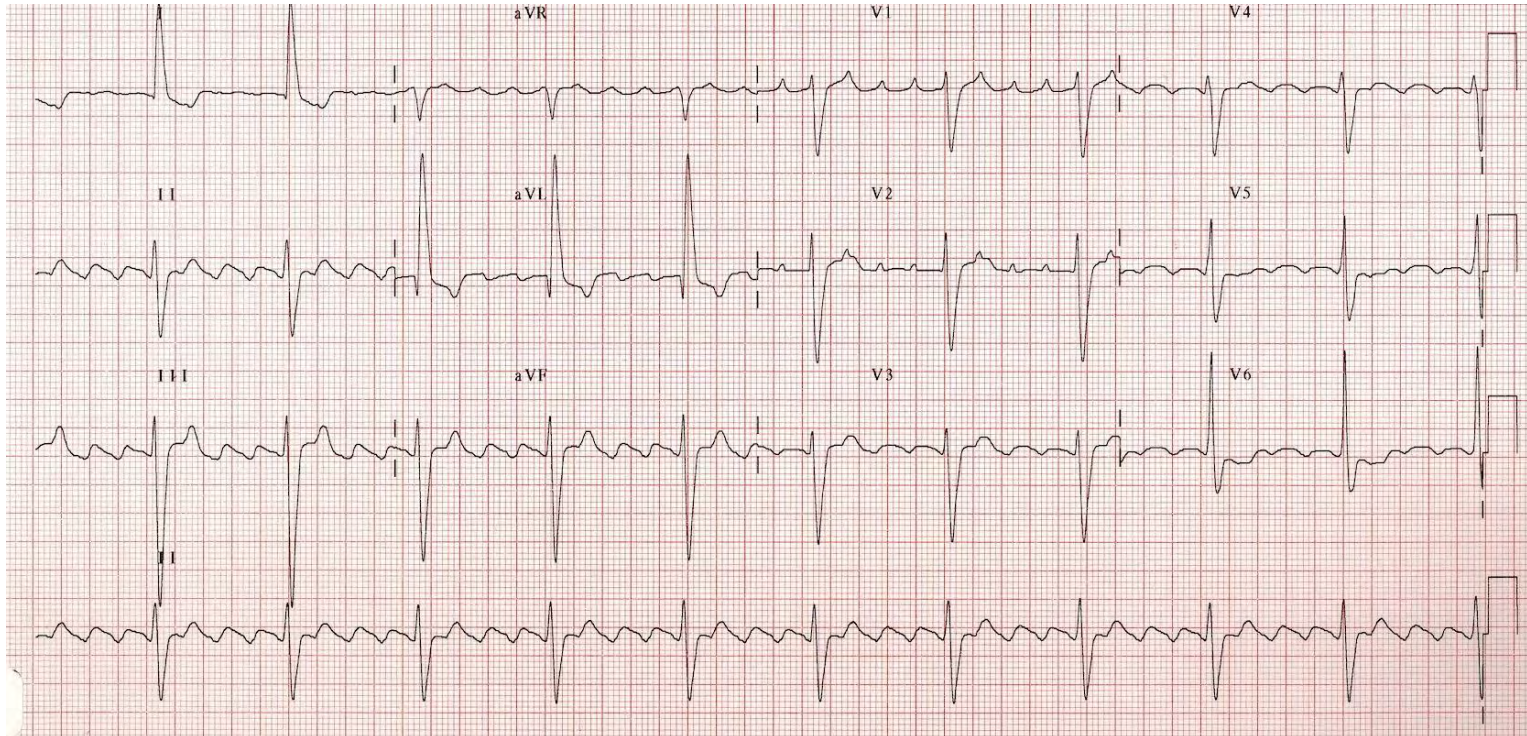
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Surgical Therapy for Atrial Fibrillation



Atrial Flutter

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Atrial Flutter

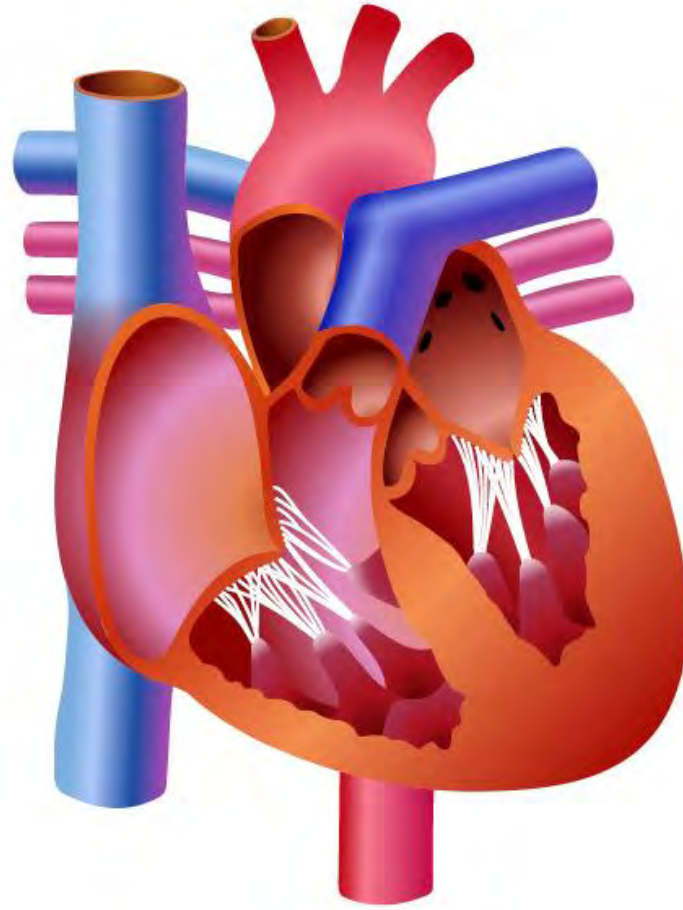
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Symptoms

- Symptoms
 - Generally the same as atrial fibrillation
 - May be asymptomatic
 - Palpitations, dyspnea, fatigue
- Treatment
 - Generally the same as atrial fibrillation
 - Rate or rhythm control
 - Rate-slowing drugs
 - Cardioversion
 - Anticoagulation based on stroke risk

Atrial Flutter Ablation

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ACLS and Tachycardias

Jason Ryan, MD, MPH



Cardiac Arrest

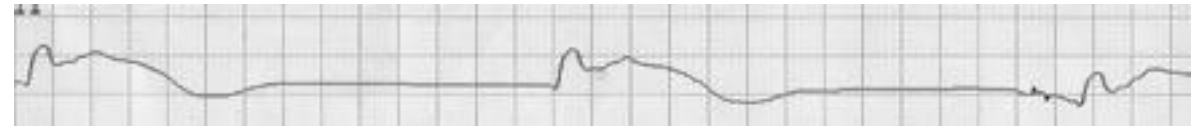
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- Sudden cessation of cardiac activity with hemodynamic collapse
- Usually associated with one of four underlying cardiac rhythms

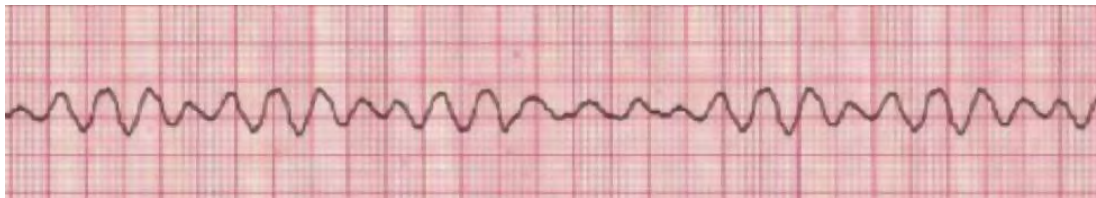
Ventricular Tachycardia



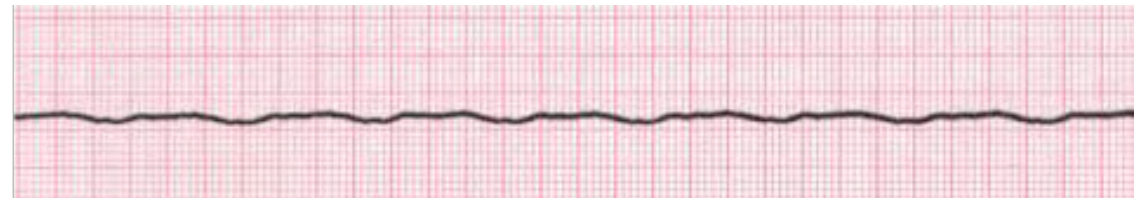
Pulseless Electrical Activity



Ventricular Fibrillation



Asystole



ACLS

Advanced cardiac life support

www.afratafreeh.com

- Clinical algorithm for treatment of life-threatening cardiac emergencies
- Applied to unresponsive patients



Public Domain

ACLS

Advanced cardiac life support

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- Unresponsive, pulseless patient → call for help
- Start chest compressions (CPR)
- Apply oxygen
- Attach monitor and defibrillator



Wikipedia common

ACLS

Advanced cardiac life support

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- Is the rhythm shockable?
- YES if ventricular tachycardia or fibrillation
- NO if PEA or asystole

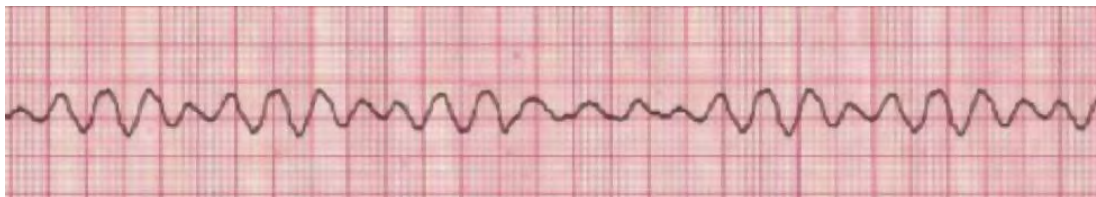
Ventricular Tachycardia



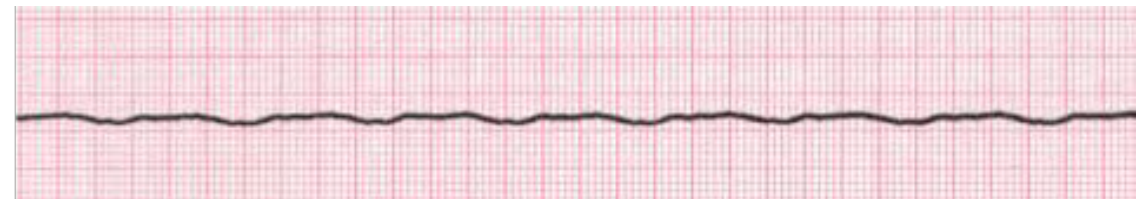
Pulseless Electrical Activity



Ventricular Fibrillation



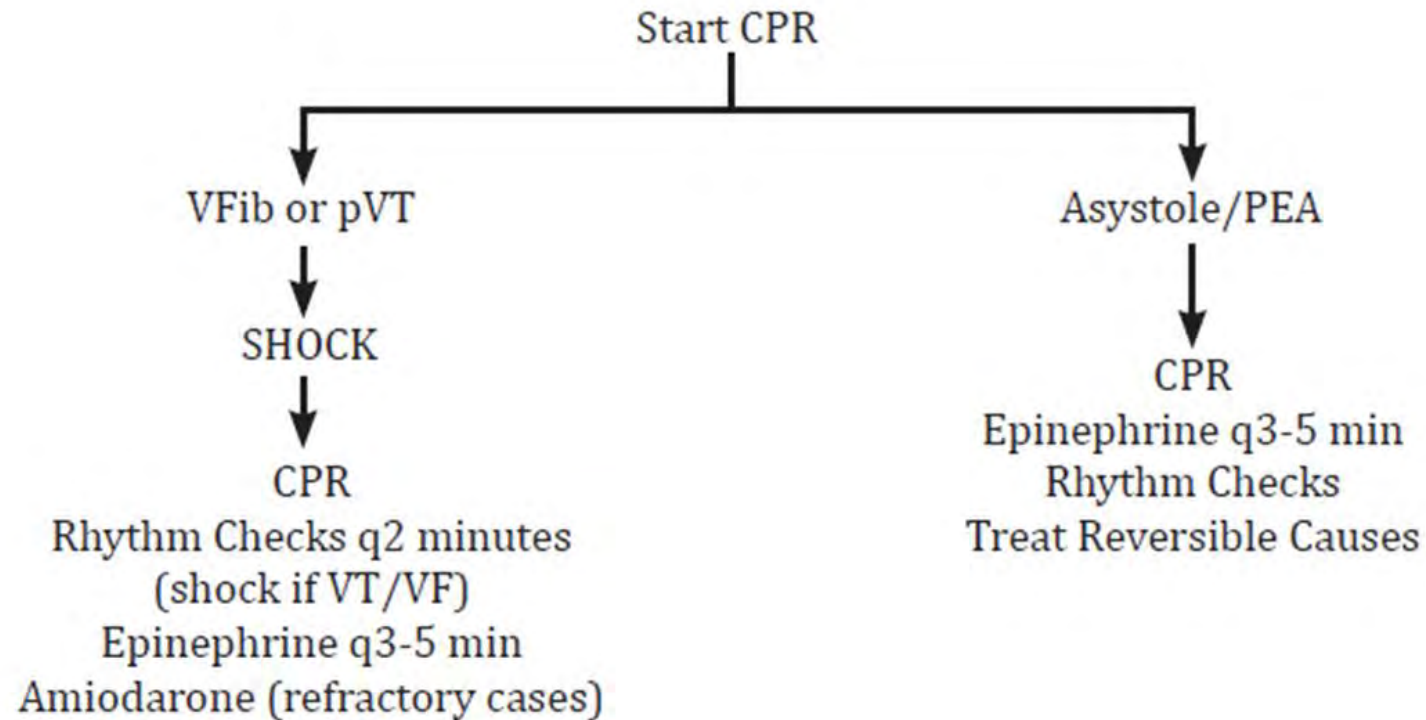
Asystole



ACLS

Advanced cardiac life support

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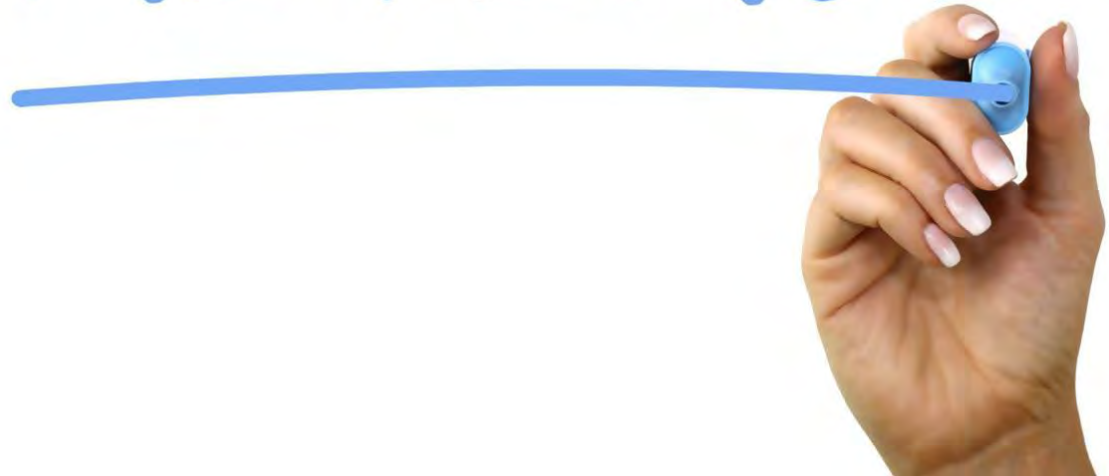


Reversible Causes

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- Hypovolemia
- Hypoxia
- Acidosis
- Hypo/hyperkalemia
- Hypothermia
- Myocardial infarction
- Tension pneumothorax
- Cardiac tamponade
- Pulmonary embolism

REVERSE



Stable Ventricular Tachycardia

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- Pulse intact
- Patient remains awake
- No evidence of hemodynamic compromise (hypotension, chest pain)
- **Intravenous amiodarone**
 - Alternatives: lidocaine or procainamide
- Do not perform cardioversion while patient is conscious

Ventricular Tachycardia

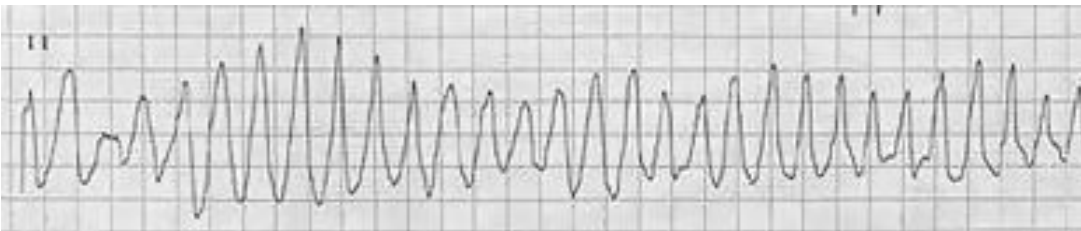


Polymorphic Ventricular Tachycardia

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- Subtype of ventricular tachycardia
- Continuously varying QRS complex morphology
- Caused by multiple foci of QRS complexes within the ventricle
- Unstable rhythm that often leads to cardiac arrest
- Almost always caused by **myocardial ischemia**

Polymorphic Ventricular Tachycardia



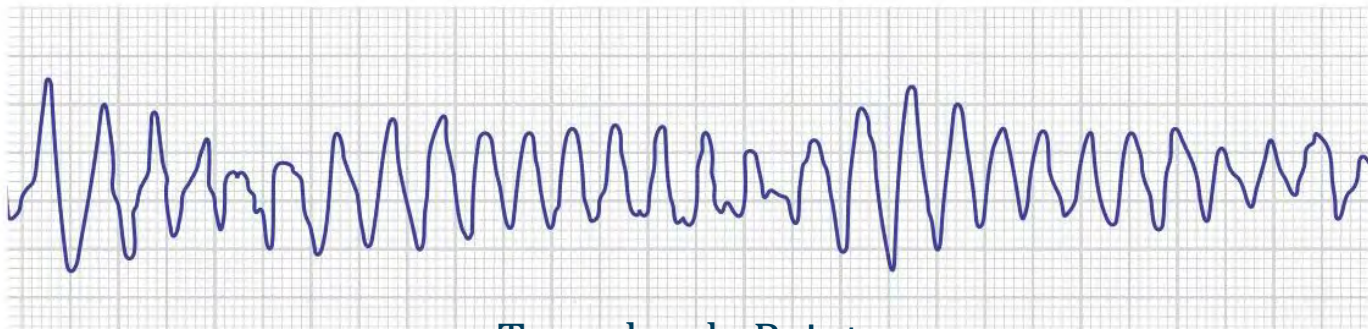
Monomorphic Ventricular Tachycardia



Torsades de Pointes

www.afratafreeh.com

- Polymorphic ventricular tachycardia that occurs with **prolonged QT segment**
- Specific subtype of polymorphic ventricular tachycardia
- Urgent defibrillation indicated in hemodynamically unstable patients
- For patients with recurrent episodes: **IV magnesium sulfate**



Torsades de Pointes



Prolonged Qt Interval

Out-of-Hospital Cardiac Arrest

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- **Time to resuscitation:** strongest predictor of survival
 - Short time: good survival
 - Long time: poor prognosis
- Other poor prognostic signs
 - Initial PEA or asystole
 - Prolonged CPR > 5 minutes
 - Advanced age



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Tachycardias

Unstable Patients

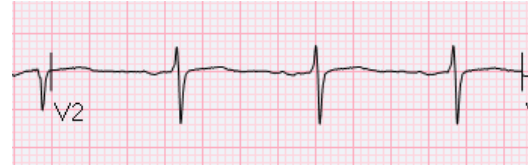
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- Hemodynamic instability: hypotension, chest pain or respiratory distress
- Often associated with tachycardia
- Tachycardia may be cause or consequence of hemodynamic instability
- Key distinction: **wide versus narrow QRS complex**

QRS Interval

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Normal QRS



Ventricular Tachycardia



Right Bundle Branch Block



Left Bundle Branch Block



Wide Complex Tachycardias

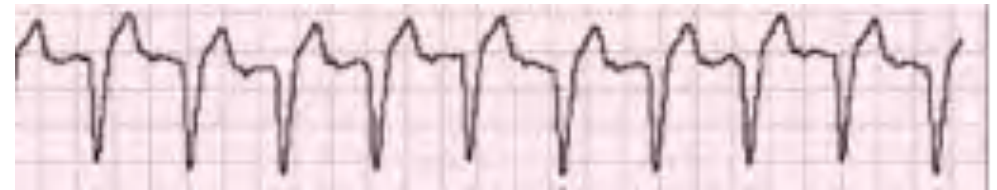
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- Rapid heart rate with wide QRS complex (> 120 ms)
- Differentiation based on ECG
 - Ventricular tachycardia
 - SVT with aberrancy (LBBB, RBBB)
- **Sudden onset/unstable WCT: shock**

Ventricular Tachycardia



Sinus Tachycardia with LBBB



Narrow Complex Tachycardias

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- Rapid heart rate with narrow/normal QRS complex ($<120\text{ms}$)
- Electrical rhythm originating above the ventricle
 - Sinus node, atrial, AV node
 - “Supraventricular tachycardias” or SVTs
- Many causes
 - Sinus tachycardia
 - Atrial fibrillation/flutter
 - Atrial tachycardia
 - AVNRT
- Treatment based on underlying cause
- **Sudden onset/unstable SVT: shock**

Narrow Complex Tachycardia



Electrical Cardioversion

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- Unsynchronized
 - High energy shock delivered immediately
 - Used for ventricular fibrillation and pulseless ventricular tachycardia
- Synchronized
 - Low energy shock synchronized to occur during QRS complex
 - Avoids shock during T wave which can cause ventricular fibrillation
 - Used for SVT (atrial fibrillation, flutter)
- **Unstable SVT: *synchronized* cardioversion**

Rapid Atrial Fibrillation



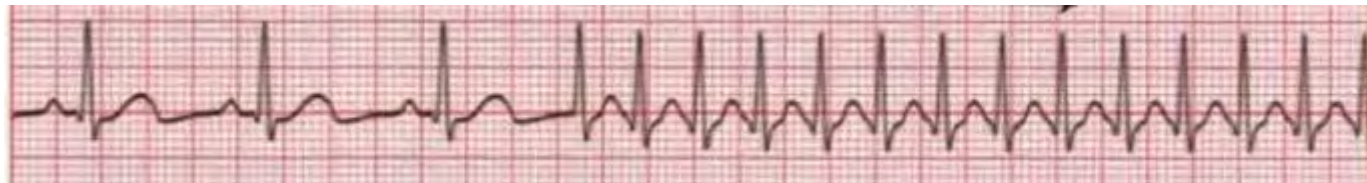
PSVT

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Paroxysmal Supraventricular Tachycardia

- Subtype of SVT
- **Regular**, supraventricular rhythm with **abrupt onset**
- Often causes **palpitations**
- May cause chest discomfort
- Rarely syncope or hypotension

PSVT



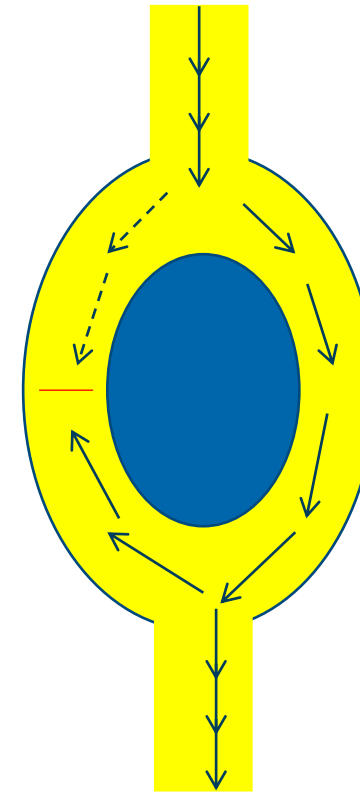
AVNRT

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Atrioventricular nodal reentrant tachycardia

- **Most common cause of PSVT**
- More common in young women
- Mean age onset: 32-years-old
- Requires **dual AV nodal pathways**

**Slow
Conduction**



**Fast
Conduction**

HIS

Retrograde P Waves

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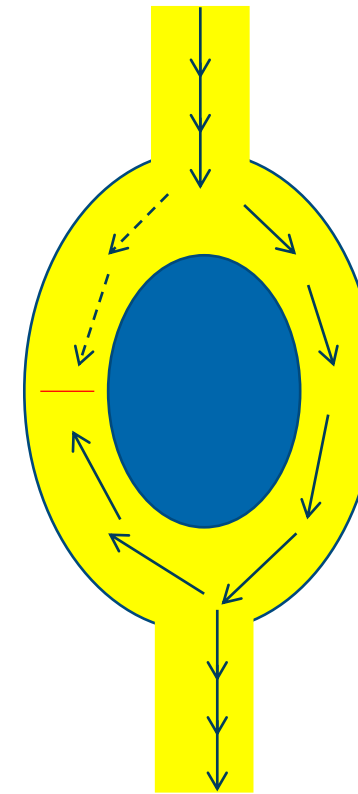
AVNRT

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- Recurrent episodes of palpitations
- Episodes usually spontaneously resolve
- **↓ conduction in AV node breaks arrhythmia**
 - Will halt conduction in slow pathway
- Carotid massage
- Vagal maneuvers
- Adenosine

**Slow
Conduction**

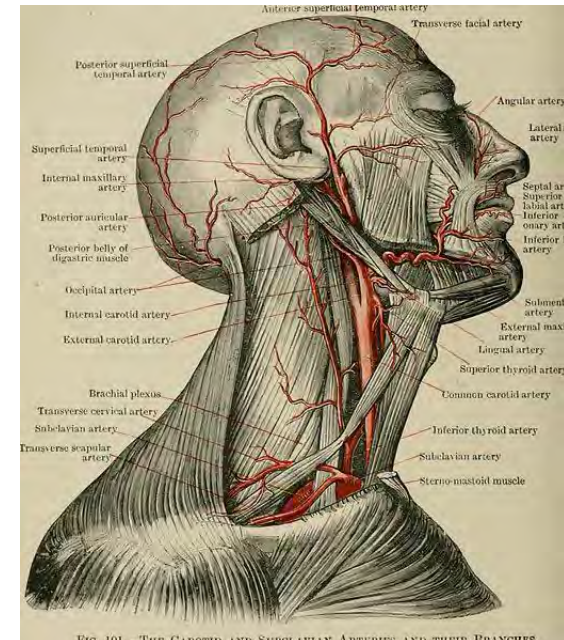
**Fast
Conduction**



HIS

Carotid Massage www.afratafreeh.com

- Examiner presses on neck near carotid sinus
- **Stretch of baroreceptors**
- CNS response as if **high blood pressure**
- Increased vagal tone
- ↓ AV node conduction



Vagal Maneuvers

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- **Valsalva**
 - Patient bears down as if moving bowels
 - Increased thoracic pressure
 - Aortic pressure rises → ↓ heart rate and AV conduction
- Breath holding
- Coughing
- Deep respirations
- Gagging
- Swallowing

AVNRT

Chronic Treatment

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- Many patients need no therapy
- Beta-blockers, Verapamil/Diltiazem
 - Slow conduction in slow pathway
- Surgical ablation of slow pathway

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Hyperlipidemia

Jason Ryan, MD, MPH



Lipid Measurements

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- Total Cholesterol
- LDL
- HDL
- Triglycerides

LDL Cholesterol www.afratafreeh.com

- **“Bad” cholesterol**
- Associated with CV risk
- < 100 mg/dl very good
- > 200 mg/dl high
- Evidence that treating high levels reduces risk

HDL Cholesterol

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- **“Good” cholesterol**
- Inversely associated with risk
- < 45mg/dl low
- Little evidence that raising low levels reduces risk

Triglycerides

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- Normal TG level < 150mg/dl
- Levels > 1000 can cause **pancreatitis**
- Elevated TG levels modestly associated with CAD
- Little evidence that lowering high levels reduces risk

Hyperlipidemia www.afratafreeh.com

- Elevated total cholesterol, LDL or triglycerides
- Risk factor for **coronary disease and stroke**
- Modifiable – often related to lifestyle factors
 - Sedentary lifestyle
 - Saturated and trans-fatty acid foods
 - Lack of fiber

Secondary Hyperlipidemia

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Selected Causes of Hyperlipidemia
Alcohol
Pregnancy
Beta-blockers
HCTZ
Thyroid disease
Nephrotic syndrome

Hyperlipidemia

Treatment

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- **Lifestyle modification** recommended for all patients
 - Healthy diet, weight loss, quit smoking
- Major clinical decision is related to **statin therapy**



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Hyperlipidemia

Treatment

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- **Moderate-intensity statins**

- Many choices
- Atorvastatin 10 to 20 mg/day
- Rosuvastatin 5 to 10 mg/day
- Simvastatin 20 to 40 mg/day

- **High-intensity statins**

- Atorvastatin 40 to 80 mg/day
- Rosuvastatin 20 to 40 mg/day



Wikimedia Commons

Hyperlipidemia

Treatment

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Indication	Statin
CAD, Stroke or PAD	High-intensity
LDL > 190 mg/dL	High-intensity
Diabetics > 40 years old	Moderate- or High-Intensity
ASCVD Risk > 7.5% over 10 years	Moderate- or High-Intensity

Hyperlipidemia

Treatment

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Age (years)	40-79
Gender	☒ Male ☐ Female
Race	☐ African American ☒ Other
Total cholesterol (mg/dL)	130-320
HDL cholesterol (mg/dL)	20-100
Systolic blood pressure (mmHg)	90-200
Diastolic blood pressure (mmHg)	30-140

Treated for high blood pressure	☒ No ☐ Yes
Diabetes	☒ No ☐ Yes
Smoker	☒ No ☐ Yes
Calculate	

2.2%

10-year risk of heart disease or stroke

Hyperlipidemia

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Treatment

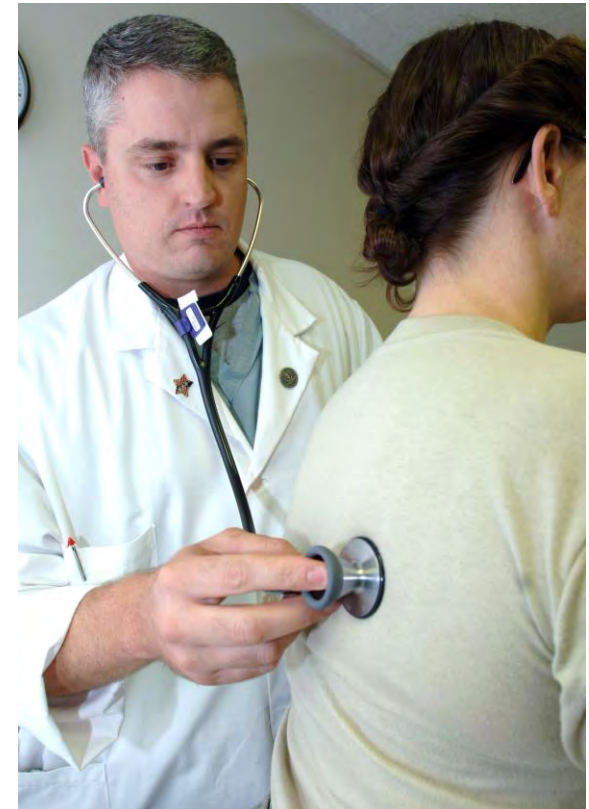
- Treatment goal usually < 100 mg/dL
- For patients with known vascular disease goal often < 70 mg/dL



Signs of Hyperlipidemia

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- Most patients have **no signs/symptoms**
- Physical findings occur in patients with severe $\uparrow$ lipids
- Usually familial syndrome



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Signs of Hyperlipidemia

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- Xanthomas
 - Plaques of lipid-laden cells
 - Appear as skin bumps or on eyelids
- Tendinous Xanthoma
 - Lipid deposits in tendons
 - Common in Achilles
- Corneal arcus
 - Lipid deposit in cornea
 - Ring around iris



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Min.neel/Wikipedia



Klaus D. Peter, Gummersbach, Germany

Familial Dyslipidemias

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Type	Name	Mxn	Clinical
I (AR)	Hyperchylomicronemia	LPL Deficiency	Trigs up → Pancreatitis
IIa (AD)	Hypercholesterolemia	LDL-R Def.	Elevated cholesterol (LDL)
III (AR)	Dysbetalipoproteinemia	APO-E Def.	Atherosclerosis
IV (AD)	Hypertriglyceridemia	VLDL Overpdx	Trigs up → Pancreatitis

Type I Dyslipidemia

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Hyperchylomicronemia

- **↑↑↑ triglycerides (> 1000; milky plasma appearance)**
- LPL deficiency or dysfunction
- **Recurrent pancreatitis**
- Enlarged liver, xanthomas
- Treatment: **very low-fat diet**
 - Reports of normal lifespan
 - No apparent ↑risk atherosclerosis

Type II Dyslipidemia

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Familial Hypercholesterolemia

- Autosomal dominant
- Few or zero LDL receptors
- **Very high LDL (> 300 heterozygote; > 700 homozygote)**
- Tendon xanthomas, corneal arcus
- **Severe atherosclerosis (can have MI in 20s)**

Type III Dyslipidemia

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Familial Dysbetalipoproteinemia

- Mutations of **apolipoprotein E gene**
- Poorly lipid particle cleared by liver
 - Accumulation of chylomicron remnants and VLDL
 - Collectively know as β -lipoproteins
- Elevated total cholesterol and triglycerides
- Usually mild (TC > 300 mg/dl)
- Xanthomas
- **Premature coronary disease**

Type IV Dyslipidemia

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Hypertriglyceridemia

- Autosomal dominant
- VLDL overproduction or impaired catabolism
- **↑TG (200-500)**
- ↑VLDL
- Associated with diabetes type II
- Often diagnosed on routine screening bloodwork
- Increased coronary risk/premature coronary disease

Lipid-Lowering Therapy

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- Statins
- Niacin
- Fibrates
- Absorption blockers
- Bile acid resins
- PCSK9 Inhibitors
- Omega-3 fatty acids

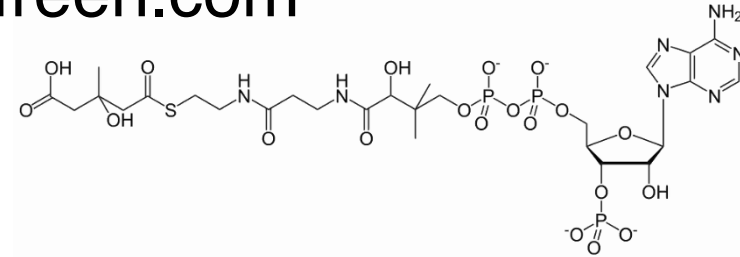
Statins

Lovastatin, Atorvastatin, Simvastatin

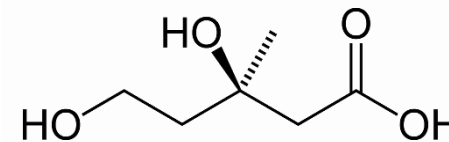
- HMG-CoA reductase inhibitors
- ↓cholesterol synthesis in liver
- ↑LDL receptors in liver
- **Major effect: ↓ LDL decrease**
 - Some ↓TG, ↑HDL
- **Excellent outcomes data (↓MI/Death)**
- Adverse effects:
 - Hepatotoxicity (↑ AST/ALT)
 - Muscle problems

3-hydroxy-3-methylglutaryl-coenzyme A
HMG-CoA

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**HMG-CoA
Reductase**



Mevalonate

Statin Muscle Problems

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- Myalgias
 - Weakness, soreness
 - Normal CK levels
- Myositis
 - Like myalgias, increased CK
- Rhabdomyolysis
 - Weakness, muscle pain, dark urine
 - CKs in 1000s
 - Acute renal failure → death
 - ↑ risk with some drugs (gemfibrozil, P450 inhibitors)



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P450

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- Most statins metabolized by liver P450 system
 - Examples: atorvastatin, simvastatin and lovastatin
 - Exceptions: ravastatin and rosuvastatin
- Potential interactions with other drugs
- P450 inhibitors increase **↑ risk LFTs/myalgias**
 - Cyclosporine
 - Macrolide antibiotics
 - Azole antifungal agents
 - HIV protease inhibitors
 - Grapefruit juice



Citrus_paradisi/Wikipedia

Niacin

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- Complex, incompletely understood mechanism
- Overall effect: LDL ↓↓ **HDL ↑↑**
 - ↓ HDL breakdown
 - Often used when HDL is low

Supplement Facts		
Serving Size: 8 fl oz (240 ml)		
Serving Per Container: About 2		
Amount Per Serving		%DV*
Calories	120	
Total Carb	29g	10%
Sugars	28g	†
Riboflavin	1.7mg	100%
Niacin	20mg	100%
Vitamin B6	2mg	100%
Vitamin B12	6mcg	100%
Sodium	120mg	5%
Protein	1g	
Ginseng Extract	200mg	†
Energy Blend	3000mg	†
Taurine, L-Carnitine, Caffeine, Guarana, Inositol, Glucuronolactone, Maltodextrin		
Percent Daily Values (DV) are based on a 2,000 calorie diet. †Daily Value not Established		

Niacin

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- Major side effects is **flushing**
 - Stimulates release of prostaglandins in skin
 - **Face** turns red, warm
 - Can blunt with **aspirin** (inhibits prostaglandin) prior to Niacin
 - Fades with time



Niacin

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- Hyperglycemia
 - Insulin resistance (mechanism incompletely understood)
 - Avoid in diabetes
- Hyperuricemia

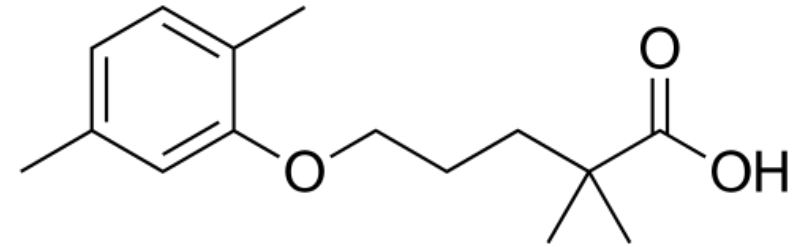


Fibrates

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Gemfibrozil, clofibrate, bezafibrate, fenofibrate

- Activate **PPAR-a**
 - Modifies gene transcription
 - ↑ activity lipoprotein lipase
 - ↑ liver fatty acid oxidation
- Major overall effect → TG breakdown
- Used for patients with very **high triglycerides**



Gemfibrozil

Fibrates

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Gemfibrozil, clofibrate, bezafibrate, fenofibrate

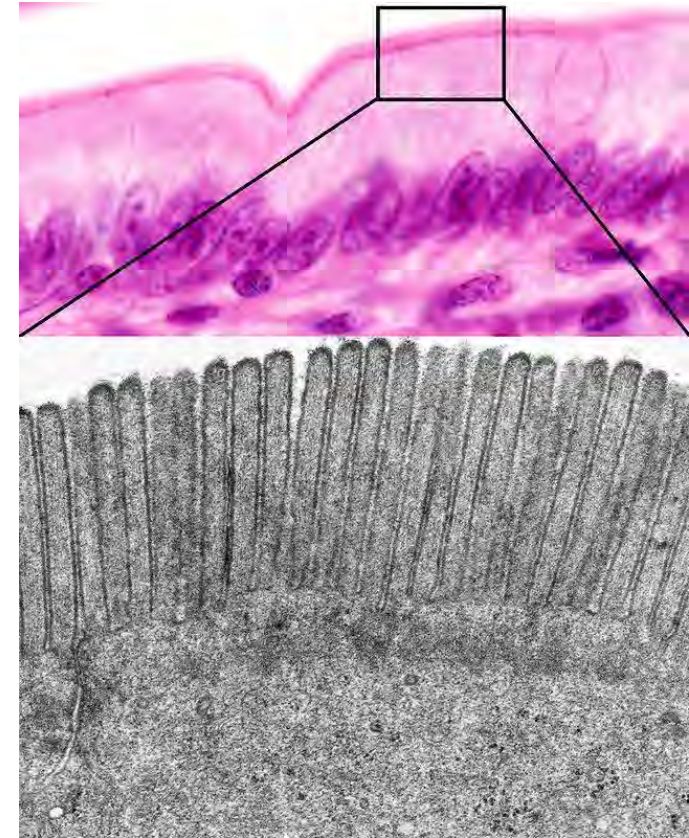
- **Myositis**
 - Rhabdomyolysis associated with gemfibrozil
 - Caution when used with statins
- ↑ LFTs
- Cholesterol gallstones

Absorption blockers

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Ezetimibe

- Blocks cholesterol absorption
- Works at **intestinal brush border**
- Blocks dietary **cholesterol** absorption
 - Highly selective for cholesterol
 - Does not affect fat-soluble vitamins, triglycerides



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Absorption blockers

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Ezetimibe

- Result: ↑ LDL receptors on liver
- Modest reduction LDL
- Some ↓ TG, ↑ HDL
- Weak data on hard outcomes (MI, death)
- ↑ LFTs
- Diarrhea

Bile Acid Resins

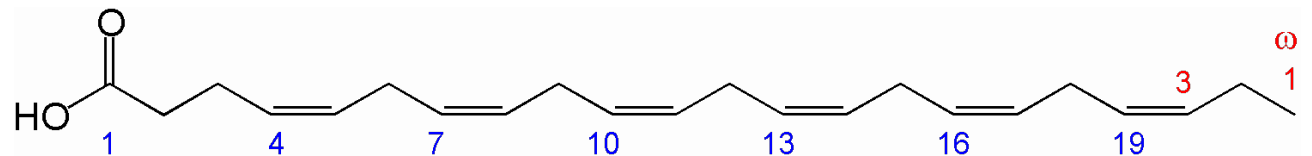
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Cholestyramine, colestipol, colesevelam

- Old drugs; rarely used
- Prevent intestinal reabsorption bile
 - Cholesterol → bile → GI tract → reabsorption
- Resins lead to more bile excretion in stool
- Liver converts cholesterol → bile to makeup losses
- Modest lowering LDL
- Miserable for patients: **bloating, bad taste**
- Can't absorb certain fat-soluble vitamins
- Cholesterol gallstones

Omega-3 Fatty Acids

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- Found in fish oil
- Consumption associated with ↓ CV events
- **Reduce VLDL production**
- **Lowers triglycerides** (~25 to 30%)
- Modest ↑ HDL
- Commercial supplements available (Lovaza)
- GI side effects: nausea, diarrhea, “fishy” taste



docosahexaenoic acid (DHA)

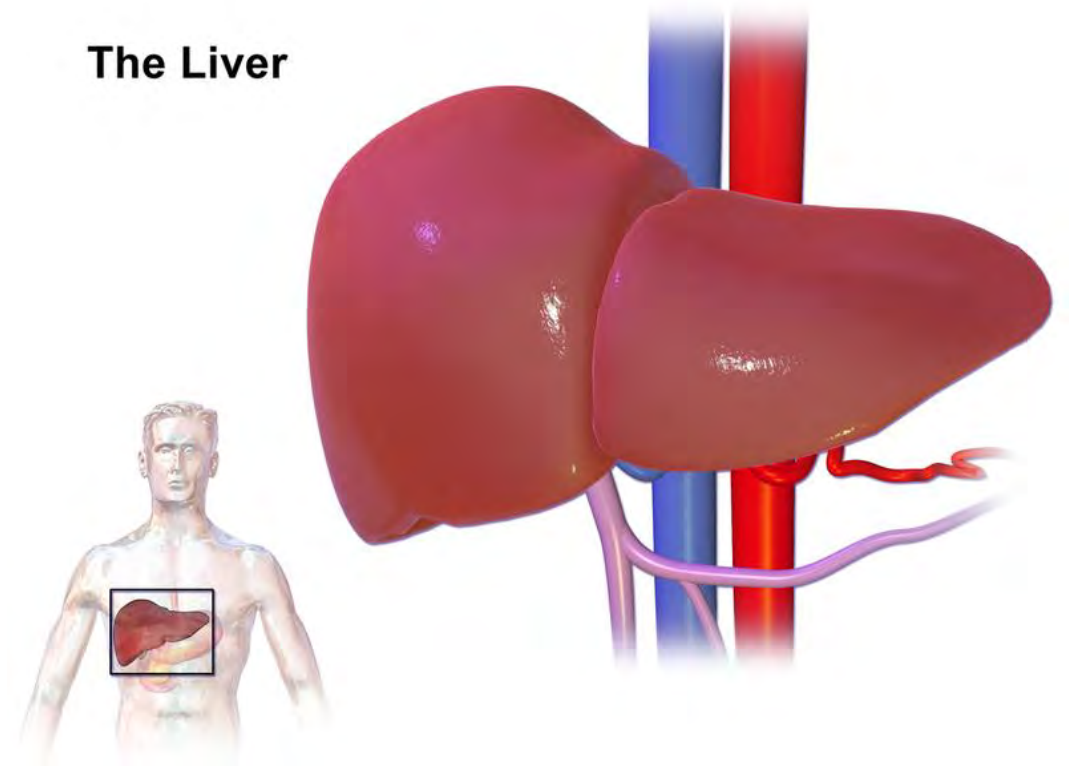
PCSK9 Inhibitors

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Alirocumab, Evolocumab

- FDA approval in 2015
- PCSK9 → **degradation of LDL receptors**
- Alirocumab/Evolocumab: Antibodies
- Inactivate PCSK9
 - ↓ LDL-receptor degradation
 - ↑ LDL receptors on hepatocytes
 - ↓ LDL cholesterol in plasma

The Liver



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PCSK9 Inhibitors

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Alirocumab, Evolocumab

- Given by subcutaneous injection
- Results in significant LDL reductions (> 60%)
- Major adverse effect is injection site skin reaction
- Used when hyperlipidemia persists despite statins
- Downside: **cost**



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Hypertension

Jason Ryan, MD, MPH



Hypertension

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- Diagnosis of hypertension requires more than one measurement

Stage	SBP		DBP
Normal	<120	and	<80
Prehypertension	120-129	and	<80
Stage I hypertension	130-139	or	80-89
Stage II hypertension	>140	or	>90
Hypertensive Crisis	>180	or	>120

Etiology

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- Most (90%) is primary (“essential”) HTN
 - Cause not clear
- Remainder (10%) secondary
 - Most associated with chronic kidney disease
 - Hyperaldosteronism
 - Renal artery stenosis
 - Pheochromocytoma
- White coat hypertension
 - Elevated BP in clinic only



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Hypertension

Risk Factors

- Family history
- African-American race
- High salt intake
- Alcohol
- Obesity
- Physical inactivity

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Hypertension

Associations

- Stroke
- Heart disease
 - MI
 - Heart failure
- Renal failure
- Aortic aneurysm
- Aortic dissection

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New Onset Hypertension

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- Screen for **complications and comorbid conditions**
 - Hemoglobin A1c or fasting glucose
 - Lipid panel
 - EKG
 - Serum creatinine
 - Urinalysis (protein)

Hypertension

Treatment

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- Lifestyle modification recommended for **all patients**
- Weight loss – most effective intervention
- **DASH diet** – 2nd most effective intervention
 - Dietary Approaches to Stop Hypertension
 - Vegetables, fruits
 - Whole grains
 - Poultry, fish
 - Low in sugar and red meats



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Hypertension

Treatment

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- Exercise
- Sodium restriction
- Alcohol limitation



"Mike" Michael L. Baird/Wikipedia

Hypertension

Drug Therapy

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- Recommended initial choices:
 - Thiazide diuretics
 - ACE inhibitors/ARBs
 - Dihydropyridine calcium channel blockers



Pixabay

Hypertension

Compelling indications

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Indication	Drug
Prior myocardial infarction	Beta-blocker
Diabetes	ACEi or ARB
Osteoporosis	Thiazide diuretic
Proteinuria	ACEi or ARB

Hypertensive Urgency

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- Severe hypertension without end-organ damage
- No agreed upon BP value
- Usually $> 180/120$



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Hypertensive Emergency

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Hypertensive Crisis

- Also no definite value
- BP usually $> 180/120$
- Often a patient with longstanding HTN who stops meds
- Neurologic impairment
 - Retinal hemorrhages, encephalopathy
- Renal impairment
 - Acute renal failure
 - Hematuria, proteinuria
- Cardiac ischemia

Malignant Hypertension

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- Sometimes used to refer to hypertensive emergency with **papilledema**



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Hypertensive Emergency

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Treatment

- Gradual reduction in blood pressure
 - ↓ MAP no more than 25 to 30% in first two hours
 - Rapid reduction → ischemia
 - Goal usually < 160/100 mmHg
- Drug choices (all intravenous)
 - Hydralazine
 - Esmolol
 - Nitroprusside
 - Labetalol
 - Clevidipine or nicardipine

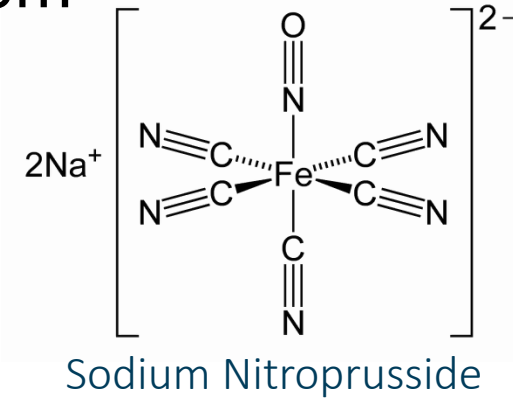


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Nitroprusside

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- Short-acting drug
 - Smooth muscle relaxation
 - Venous and arteriolar vasodilation
 - ↓ afterload (arteriolar dilation)
 - ↓ preload (venous dilation)
- **Cyanide toxicity** with prolonged use
 - Multiple cyanide groups per molecule
 - Inhibits electron transport
 - Toxic levels with prolonged infusions
 - Lactic acidosis



Ambulatory Blood Pressure Monitor

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- Used when clinic BP may not represent average BP
- White coat hypertension: elevated BP only in clinic
- Masked hypertension: normal BP in clinic only



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Peripheral Vascular Disease

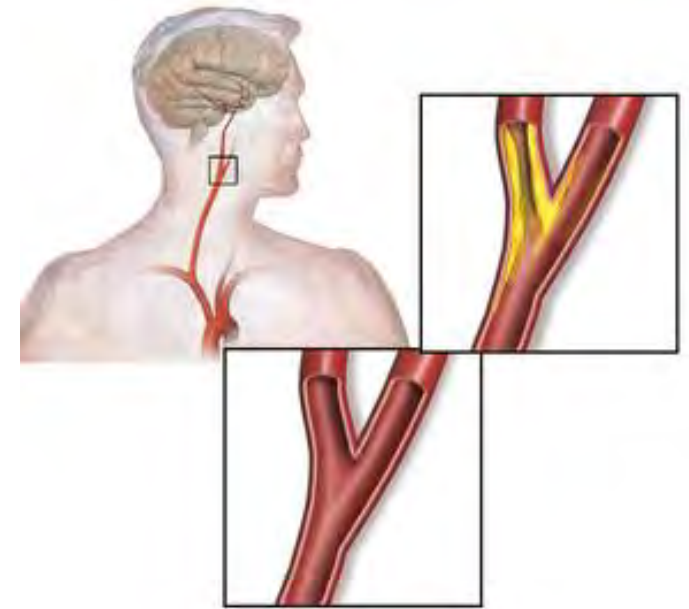
Jason Ryan, MD, MPH



Carotid Artery Disease

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- Atherosclerotic narrowing of the carotid artery
- May ulcerate → thrombus → embolization
- Occurs at carotid bifurcation
- Usually asymptomatic
- “Symptomatic” if embolization within past 6 months



Carotid Artery Disease

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Clinical Features

- Stroke
 - Usually MCA territory
 - Homonymous hemianopsia, hemiparesis, and hemisensory loss
 - Aphasia (left-sided)
 - Spatial neglect (right-sided)
- Amaurosis fugax
 - Transient monocular blindness caused by embolus to ophthalmic artery (branch of ICA)
- Carotid bruit

Hollenhorst plaques

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- Cholesterol embolization to retinal artery
- May also occur with aortic plaque embolization



Larry Halperin, MD/Retinal Image Bank

Carotid Artery Disease

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Diagnosis

- Carotid duplex ultrasound
- Magnetic resonance angiography
- Computed tomographic angiography
- Angiography

MRA



Carotid Artery Disease

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Treatment

- Carotid endarterectomy (CEA)
- Carotid artery stenting (CAS)
- Numerous clinical trials comparing CEA to CAS in various groups

Carotid Artery Disease

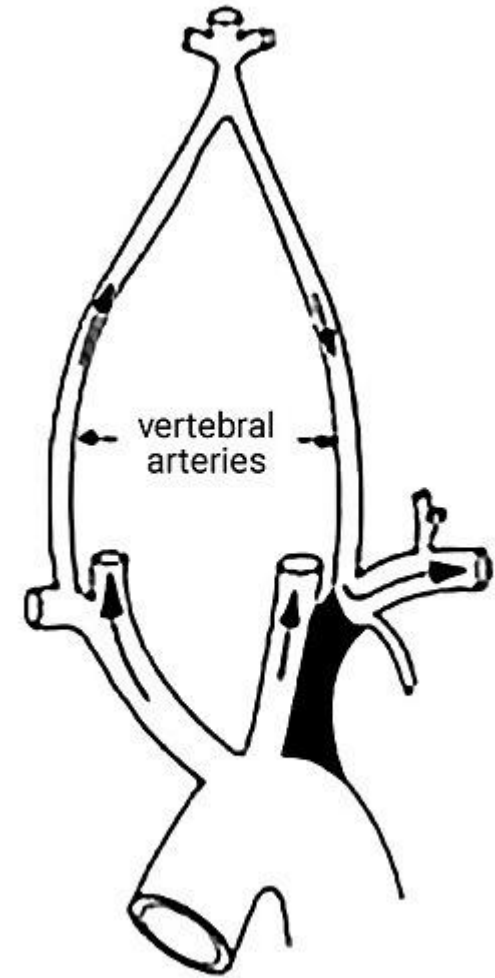
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Management

- All patients: **statin, aspirin**, BP control, lifestyle counseling
- Asymptomatic patients
 - $\geq 80\%$ stenosis: carotid endarterectomy or stenting
 - $< 80\%$ stenosis: medical management
- Symptomatic patients
 - 100% stenosis: medical management
 - 70-99%: carotid endarterectomy or stenting
 - Men 50-69%: carotid endarterectomy or stenting
 - All others: medical management

Subclavian Steal www.afratafreeh.com

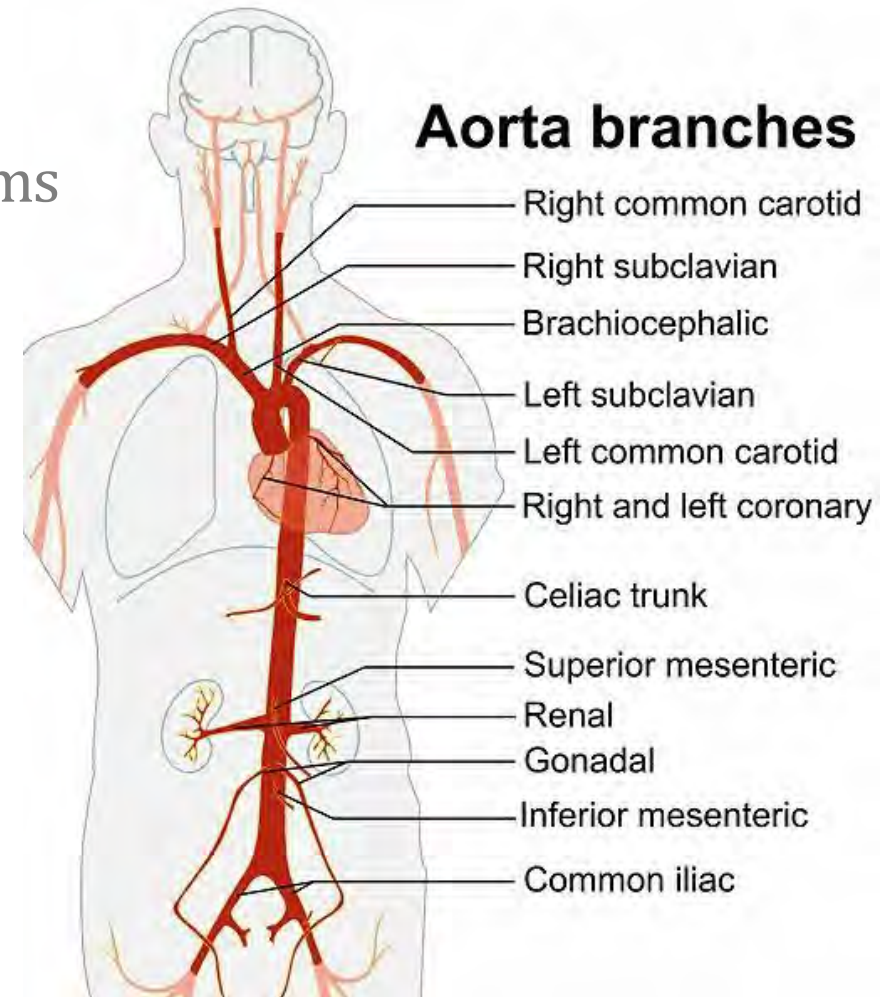
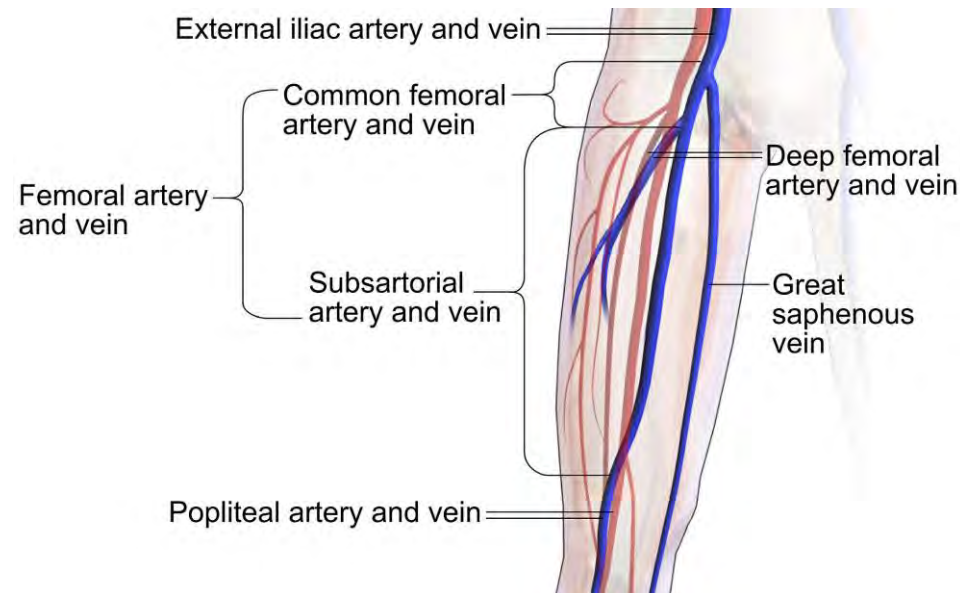
- Flow reversal in vertebral artery
- Caused by **stenosis of subclavian artery**
- Blood pressure discrepancy (>15 mmHg SBP)
- **Exercise-induced arm ischemia**
 - Pain, fatigue, numbness
- Rarely dizziness or syncope
- Diagnosis: clinical plus subclavian ultrasound
- Treatment: bypass surgery or stenting



Peripheral Artery Disease

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- Atherosclerotic disease of **lower extremity**
- Most commonly aortoiliac or femoral systems

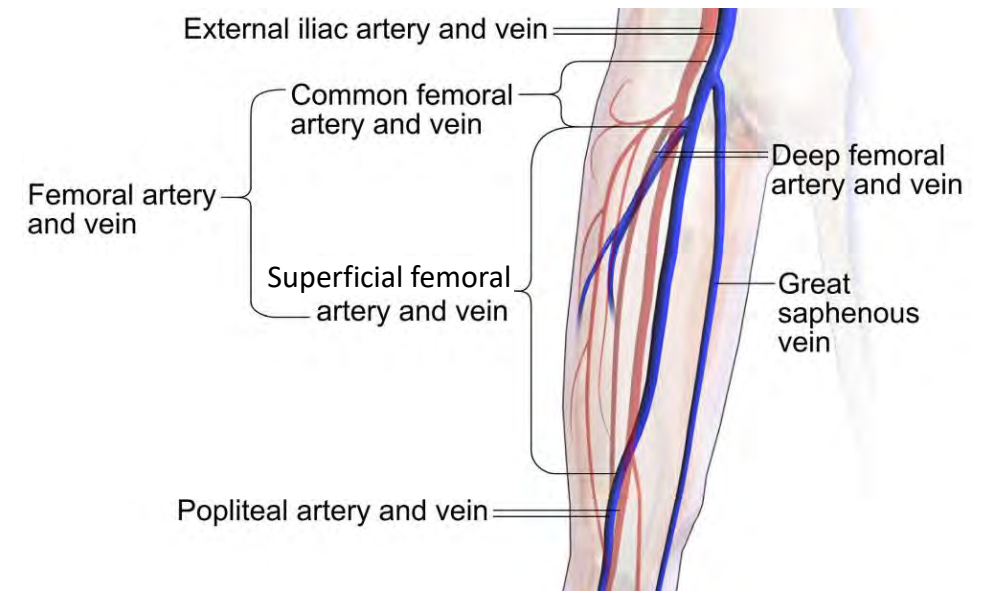


Peripheral Artery Disease

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Clinical Features

- **Claudication**
 - Pain induced by walking, relieved with rest
 - Quads, calves, and gluteal muscles most commonly involved
- Aortoiliac disease: buttock and hip claudication
- Common femoral: thigh or calf claudication
- Superficial femoral or popliteal: calf pain



Peripheral Artery Disease

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Clinical Features

- Hairless legs/feet
- Shiny skin
- Ulcers
- Buerger's Sign: elevation turns foot pale, dangling turns bright red

Peripheral Artery Disease

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Clinical Features

- Rutherford Symptom Scale

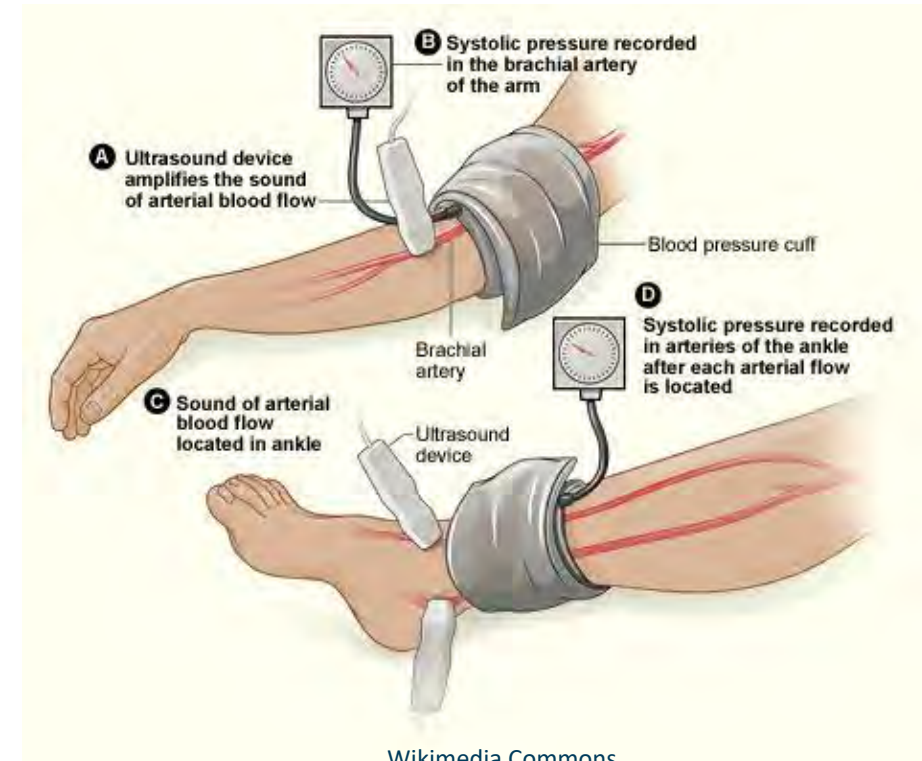
Category	Symptoms
0	Asymptomatic
1	Mild claudication
2	Moderate claudication
3	Severe Claudication
4	Rest pain
5	Minor tissue loss
6	Major tissue loss

Peripheral Artery Disease

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Diagnosis

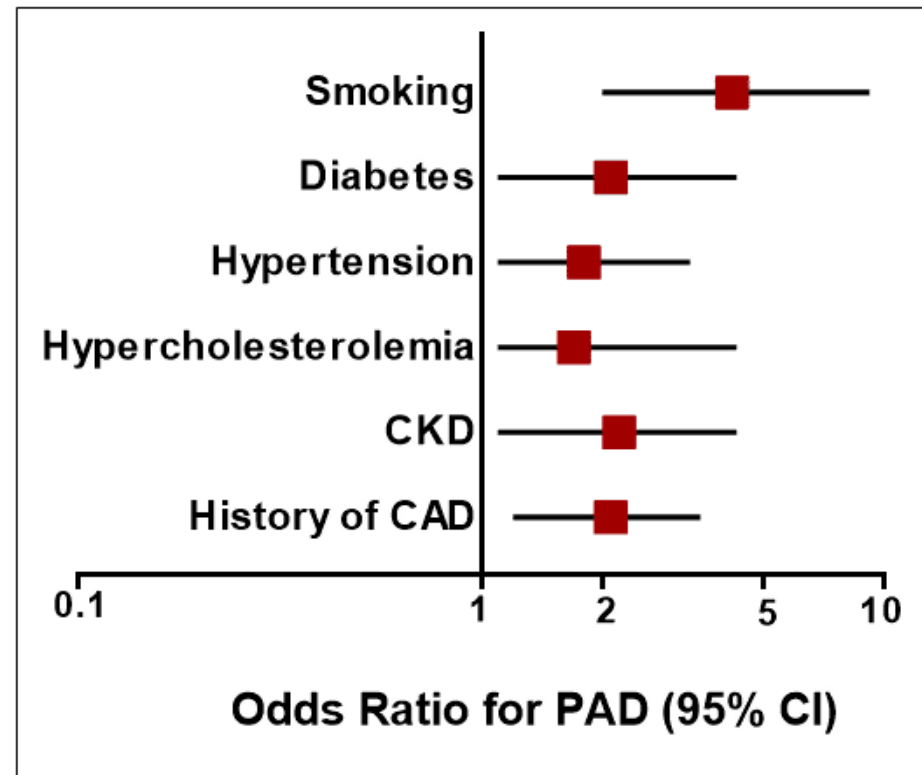
- Ankle-brachial index
 - Normal: 1.0
 - Abnormal ≤ 0.9
 - At rest and with exercise
- Imaging
 - Ultrasound
 - CT/MRI



Peripheral Artery Disease

Risk Factors

www.afratafreeh.com



Selvin E, Erlinger TP. Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999-2000. *Circulation*. 2004 Aug 10;110(6):738-43.

Peripheral Artery Disease

www.afratafreeh.com

Treatment

- Highest risk among PAD patients: **cardiovascular disease** (stroke, MI)
 - Aspirin or clopidogrel
 - Statin
 - Smoking cessation
 - Treatment of diabetes and hypertension

Peripheral Artery Disease

www.afratafreeh.com

Treatment

- Initial treatment for claudication: **exercise program**
- Cilostazol: phosphodiesterase inhibitor
 - Some evidence it decreases claudication symptoms
- Surgical revascularization or stenting

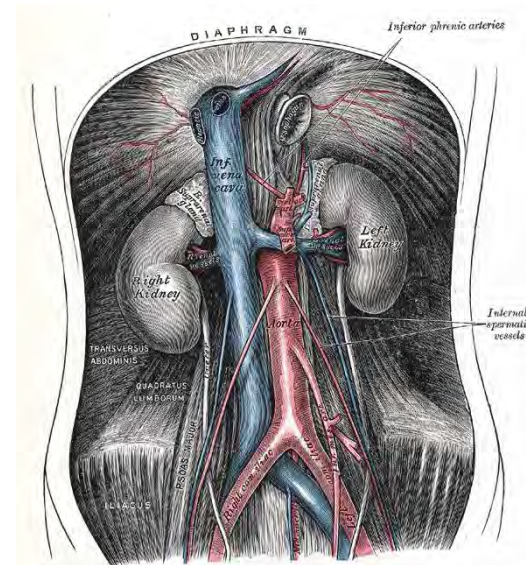


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Leriche Syndrome

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- Narrowing of aorta above iliac bifurcation
- Bilateral thigh/hip claudication
- Impotence
- Absent or diminished femoral pulses

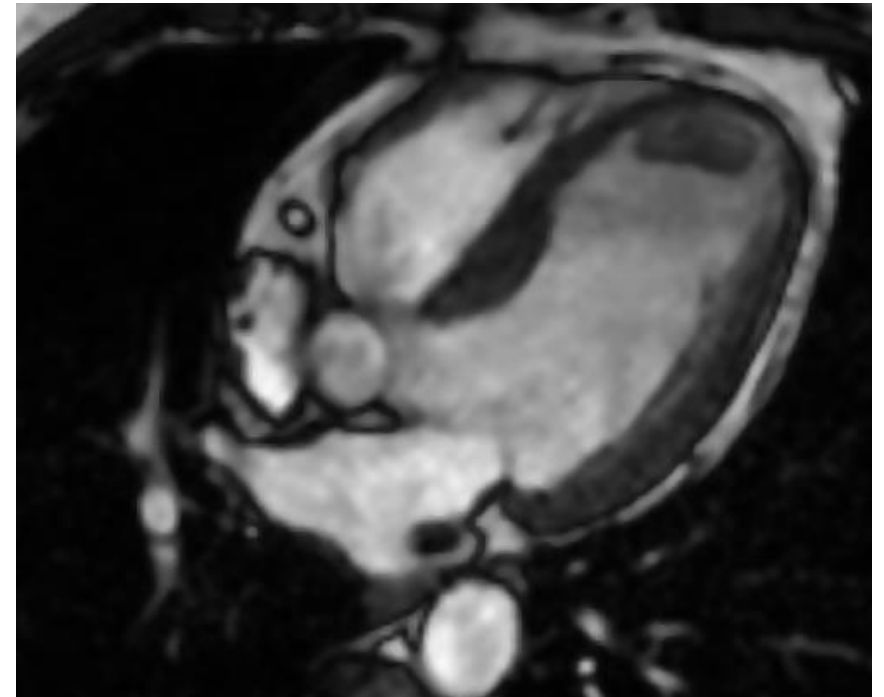


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Acute Limb Ischemia

www.afratafreeh.com

- Sudden decrease in limb perfusion
- High risk of amputation
- **Thrombosis**
 - At site of atherosclerotic plaque
 - At aneurysm
 - Hypercoagulable state
- **Cardiac embolism**
 - Left atrial appendage in atrial fibrillation
 - Left ventricle in patients with anterior infarction



Acute Limb Ischemia

www.afratafreeh.com

Clinical Features

- **P**ain
- **P**allor: Pale or mottled (livedo reticularis)
- **P**ulselessness
- **P**oikilothermia (cool skin)
- **P**aresthesia: numbness, tingling
- **P**aralysis

Mottled Skin

Blotchy, red, marbled



Public Domain

Acute Limb Ischemia

www.afratafreeh.com

Clinical Features

- Claudication or known PAD prior to acute limb ischemia
 - Suggests thrombosis
 - Can have slower onset of symptoms: hours to days
- No prior PAD
 - Suggests embolism
 - No time for recruitment of collateral blood vessels
 - Severe signs of limb ischemia develop quickly
 - Rapid onset of symptoms: minutes

Acute Limb Ischemia

www.afratafreeh.com

Diagnosis and treatment

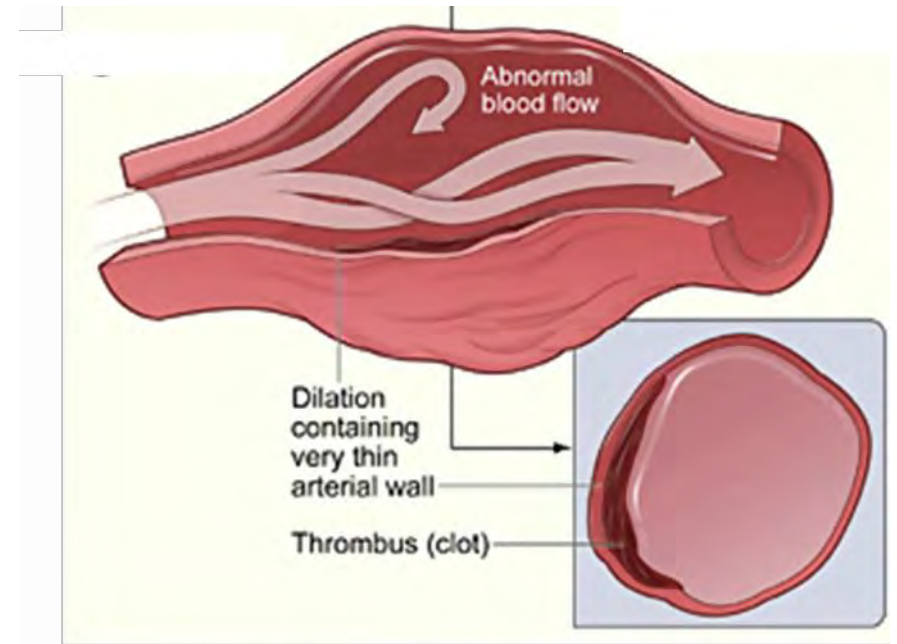
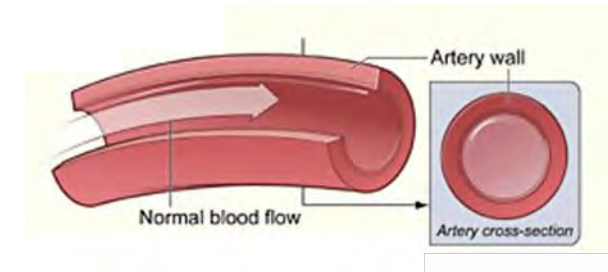
- Diagnosis: CT angiogram
- Treatment:
 - IV fluids
 - Leg in dependent position
 - Heparin
 - Thrombolysis
 - Surgical revascularization
 - Amputation

Class	Definition	Intervention
Viable (I)	- Mild pain, but intact capillary refill, peripheral pulses	None
Marginally threatened (IIa)	- Moderate pain, diminished pulses, possible sensory deficit	Urgent revascularization
Immediately threatened (IIb)	- Severe pain, sensory/motor deficits, no pulses	Emergent revascularization
Irreversible ischemia (III)	- Complete paralysis/no sensation - Signs of dead tissue	Amputation

Aneurysms

www.afratafreeeh.com

- Focal dilation more than 50% above vessel diameter
- May develop thrombosis → limb ischemia
- Popliteal artery: most common
- Femoral artery: 2nd most common
- Diagnosis by ultrasound, CT, or MRI



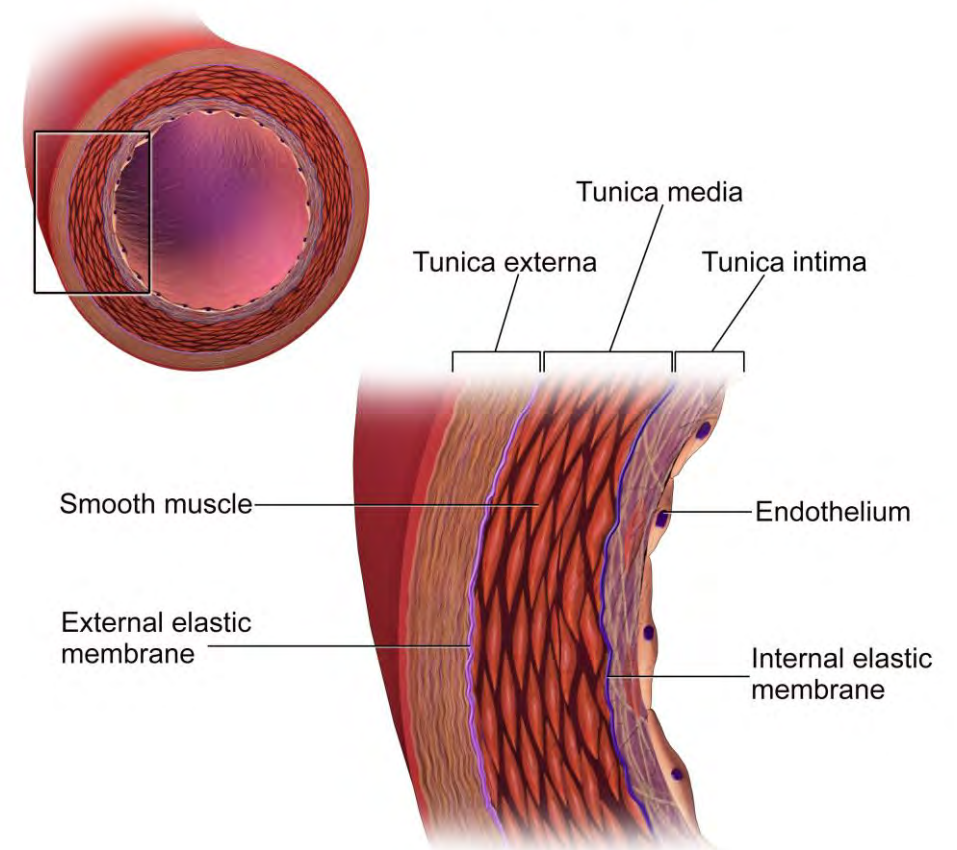
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Pseudoaneurysms

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- Hole through intima/media
- Blood fills space between layers
- Usually traumatic
- Common in **femoral artery**
 - Vessel damage from catheterization

The Structure of an Artery Wall

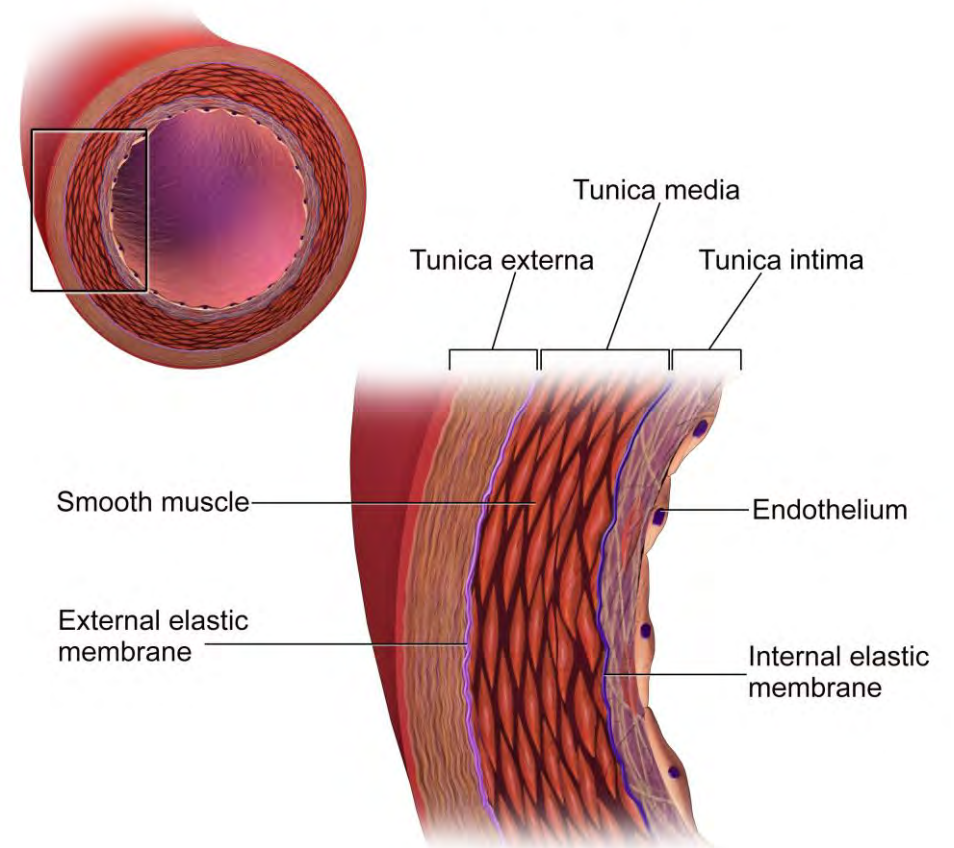


Pseudoaneurysms

www.afratafreeh.com

- Diagnosis: ultrasound
- Treatments:
 - Surgery
 - Stenting
 - Ultrasound guided thrombin injection

The Structure of an Artery Wall



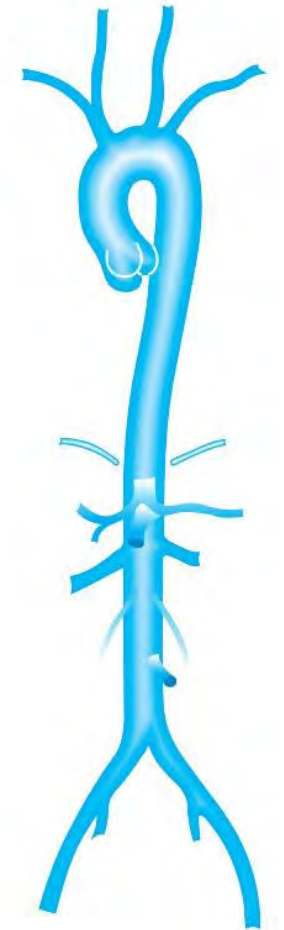
Atheroembolism

www.afratafreeh.com

- Cholesterol embolization from aortic plaque
- Commonly occurs after cardiac catheterization or vascular surgery
- Classic cause of livedo reticularis (mottled skin)
- May cause **eosinophilia**
- Treatment: supportive



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Aortic Disease

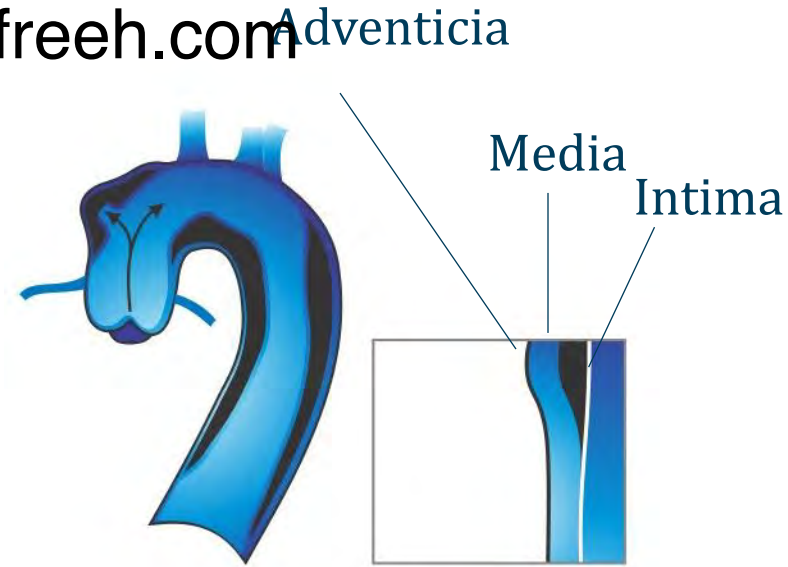
Jason Ryan, MD, MPH



Aortic Dissection

www.afratafreeh.com

- Three layers to aorta
 - Intima
 - Media
 - Adventicia
- Dissection → **tear in intima**
- Blood “dissects” intima and media



Types

www.afratafreeh.com

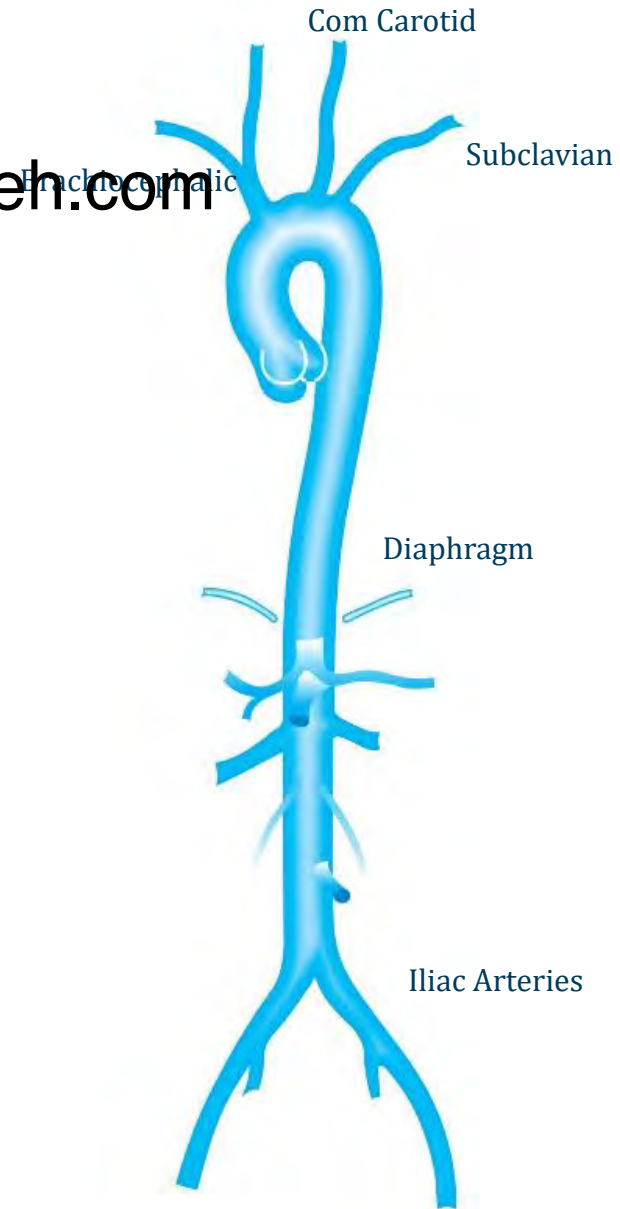
- Type A
 - Involves ascending aorta and/or arch
 - Treated **surgically**
- Type B
 - Descending aorta
 - Can be treated **medically**
 - Control hypertension/symptoms
 - Surgical mortality high



Propagation

- Blood enters dissection plane
- Spreads proximal, distal
- Can disrupt flow to vessels

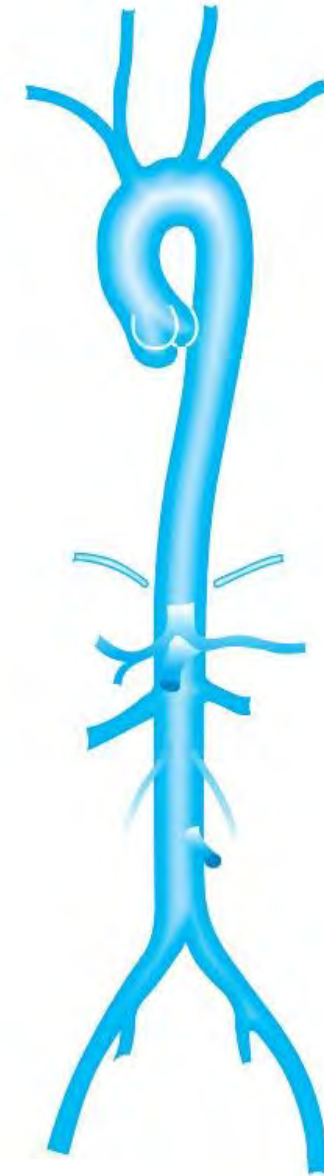
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Propagation

www.afratafreeh.com

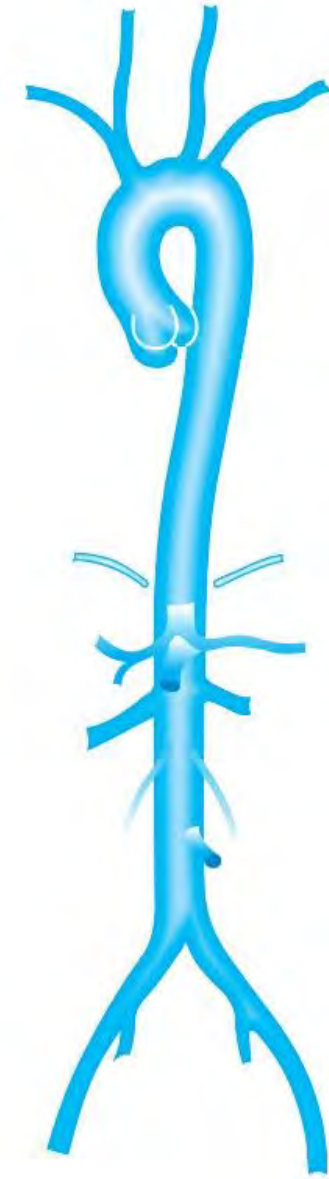
- Propagation to aortic root
 - Aortic regurgitation
 - **Pericardial effusion/tamponade**
 - Myocardial ischemia (obstruction RCA origin)
- Propagation to aortic arch
 - Stroke (carotids)
 - Horner's syndrome
 - Vocal cord paralysis



Propagation

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- Propagation to distal aorta (type B)
 - Limb ischemia
 - Mesenteric ischemia
 - Renal failure



Symptoms and Findings

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- **“Tearing” chest pain radiating to back**
- Asymmetric blood pressure
 - >20 mmHg difference between right and left

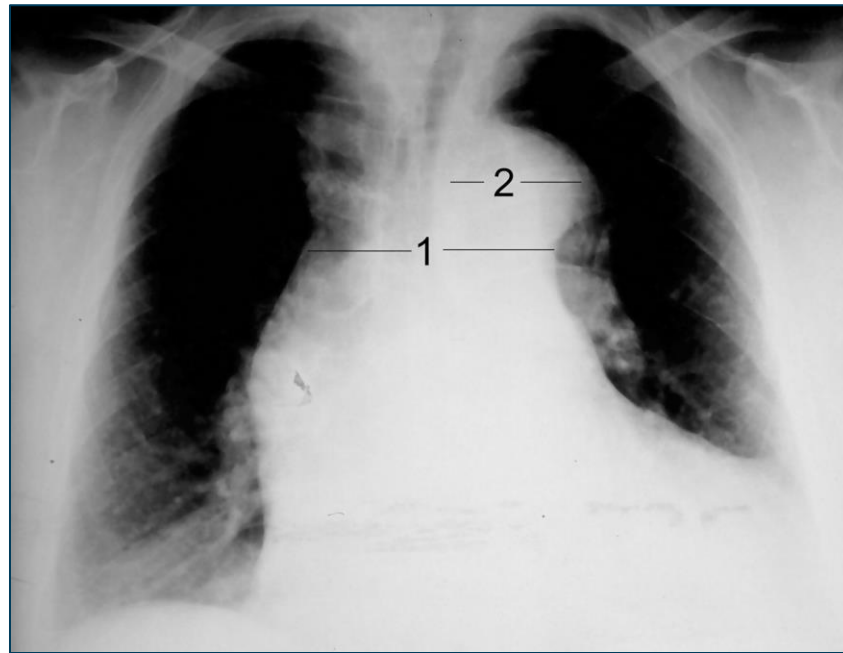


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Other findings

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- **Widened mediastinum** on chest x-ray



Risk Factors

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Aortic Dissection

- Aortic damage
 - **HTN - #1 risk factor**
 - Atherosclerosis
 - Thoracic aneurysm
- Abnormal collagen
 - Marfan Syndrome
 - Ehlers-Danlos
- Others
 - Bicuspid aortic valve
 - Turner Syndrome (bicuspid, coarctation)
 - Tertiary syphilis: Aortitis

Diagnosis

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- Suggested by history, exam, chest x-ray
- Definitive diagnosis
 - **CT scan**
 - MRI
 - Transesophageal echocardiogram (TEE)



Dr. James Heilman/Wikipedia

Treatment

www.afratafreeh.com

- Type A: **emergent surgery**
- Type B: medical therapy
 - **Blood pressure control**
 - Beta-blockers: IV esmolol or labetalol
 - Nitroprusside

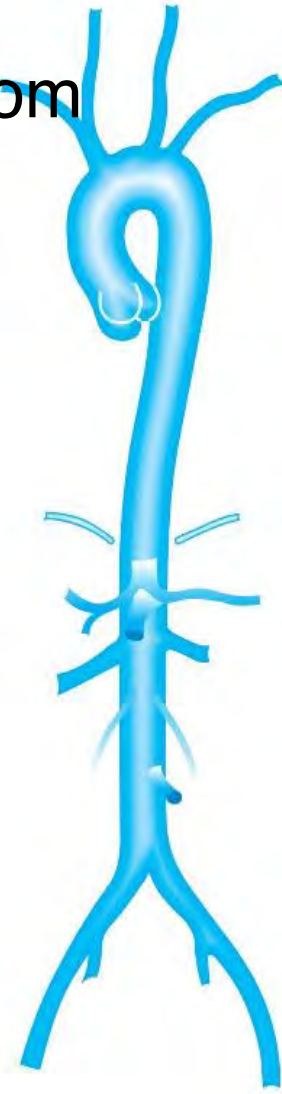


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Aortic Aneurysms

www.afratafreeh.com

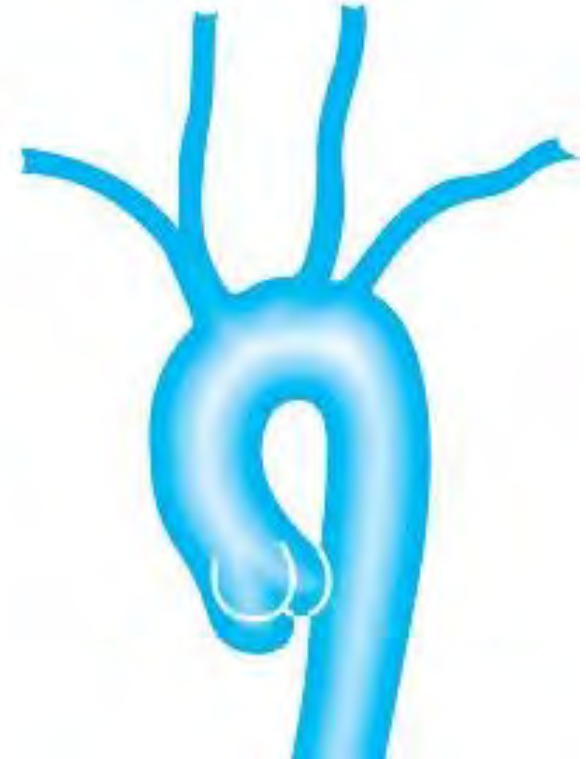
- Dilation/bulge of aorta
- More than 1.5x normal
- Involves all 3 layers
- Thoracic (TAAs)
- Abdominal (AAAs)



Thoracic Aortic Aneurysms

www.afrataalreem.com

- Important risk factor for dissection
- Usually occur in proximal/ascending aorta
- Major risk factor: **hypertension**
 - Also hyperlipidemia, smoking
- Often seen in association with another disorder
 - Marfan, Turner, bicuspid aortic valve, tertiary syphilis



Thoracic Aortic Aneurysms

www.afrataalreem.com

Diagnosis and treatment

- Most are asymptomatic
- Can cause aortic regurgitation
- Diagnosis:
 - Echocardiography
 - CT
 - MRI
- For most patients, surgery if size > 5.5 cm



Abdominal Aortic Aneurysms

www.afratafreeh.com

- More common than thoracic aneurysms
- Associated with **atherosclerosis**
- Most common site: **infrarenal aorta**
 - Most affected by atherosclerosis



Abdominal Aortic Aneurysms

www.afratafreen.com

Risk Factors

- **Smoking:** strongest association with AAA
- Males: 10x more common
- Age
 - Rare before 55
 - As high as 5% in men > 65
- Hypertension
- Hyperlipidemia



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Abdominal Aortic Aneurysms

www.afratafreeh.com

- Most are asymptomatic
- Some detected on physical exam
- **Pulsatile mass** from xiphoid to umbilicus
- Natural history is enlargement → rupture
- Followed with **ultrasound** or CT scan
- Surgery if > 5.0 cm



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USPSTF Guidelines

www.afratafreeh.com

United States Preventative Services Task Force

- **One-time ultrasound** screening recommended
- Men
- Aged 65-75 years old
- Any smoking history (current or former)

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Cardiovascular Pharmacology I

Jason Ryan, MD, MPH

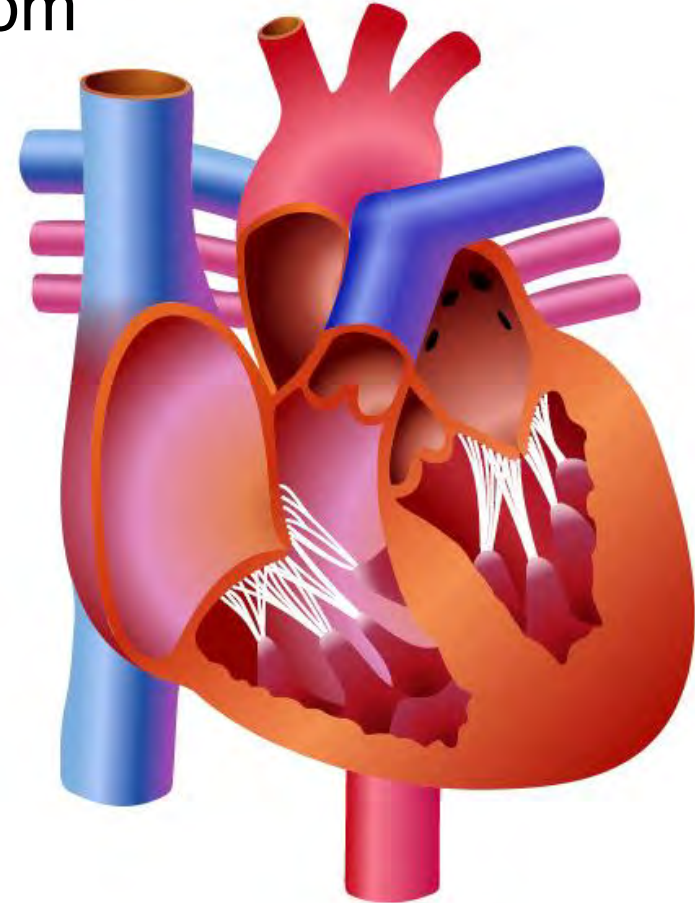


Beta-blockers

β 1-selective antagonists

www.afratafreeh.com

- Atenolol, Metoprolol, Esmolol, Bisoprolol
- Used for **hypertension**
 - Blockade $\rightarrow$ $\downarrow$ CO $\rightarrow$ $\downarrow$ BP
- Metoprolol: **systolic heart failure**
 - Blocks sympathetic stimulation of heart
 - Reduces mortality

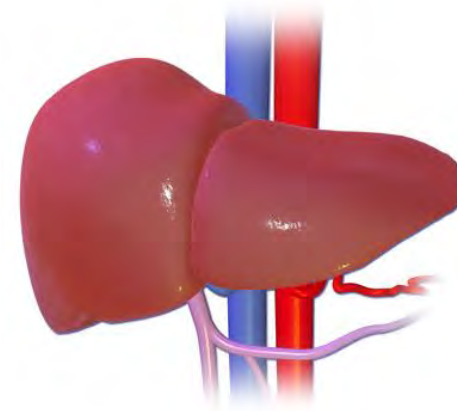


Beta-blockers

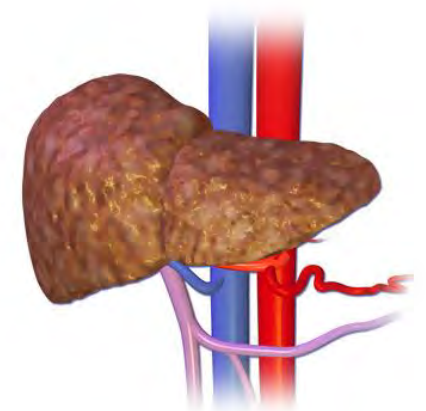
$\beta_1\beta_2$ (nonselective) antagonists

www.afratafreeh.com

- Propranolol, Timolol, Nadolol
- Nadolol, Propranolol: Used in **portal hypertension**
 - Beta 1 blockade: $\downarrow$ CO, $\downarrow$ ECV $\rightarrow$ $\downarrow$ BP
 - Beta 2 blockade: $\downarrow$ portal blood flow
- Timolol: Used in **glaucoma**
 - Beta 1 and Beta 2 $\rightarrow$ aqueous humor production
- Propranolol: **hyperthyroidism**
 - Blocks T4 $\rightarrow$ T3 conversion



Normal Liver



Liver Cirrhosis

Beta-blockers

$\beta_1\beta_2\alpha_1$

www.afratafreeh.com

- Labetalol: hypertensive emergency
 - Rapid reduction in blood pressure
- Carvedilol: systolic heart failure
 - Blocks sympathetic stimulation of heart
 - Reduces mortality



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Beta-blockers

Side effects

www.afratafreeh.com

- Fatigue, erectile dysfunction, depression
 - More common with older beta-blockers (propranolol)
- Hyperlipidemia
 - Mild increase in triglycerides
 - Mild decrease in HDL
 - Effect varies with different beta-blockers



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Beta-blockers

Side effects

www.afratafreeh.com

- Caution in **diabetes**
- Blockade of epinephrine effects
 - Epinephrine raises glucose levels
 - Blockade → hypoglycemia
- Blockade of hypoglycemia symptoms
 - ↓ glucose → sweating/tachycardia
 - Symptoms “masked” by beta-blockers



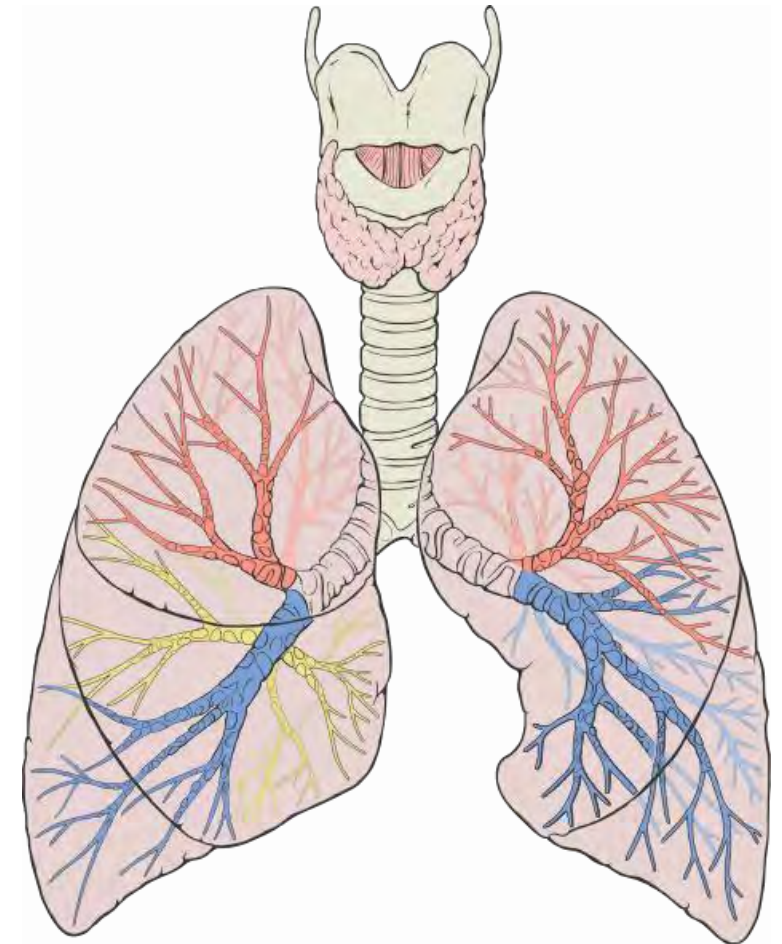
Victor/Flickr

Beta-blockers

Side effects

www.afratafreeh.com

- Caution in **asthma/COPD**
 - B2-receptors: bronchodilators
 - B2-blockade may cause a flare
 - B1-blockers (“cardioselective”) often used
- Decompensated **heart failure**
 - B1-blockers lower cardiac output → worsening of symptoms
 - Commonly used in compensated heart failure
 - Mortality benefit



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Beta-blockers

Overdose

www.afratafreeh.com

- Depression of myocardial contractility → shock
- Bradycardia/AV block
- Treatment: **glucagon**

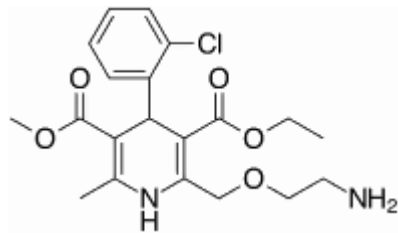


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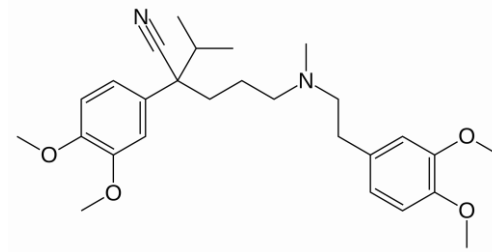
Calcium Channel Blockers

www.afratafreeh.com

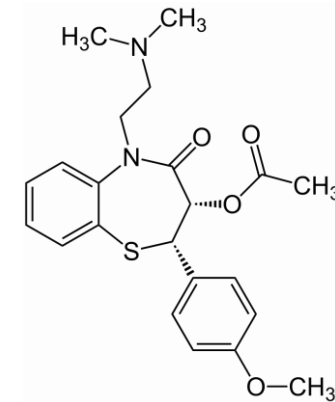
- Three major classes of calcium antagonists
 - dihydropyridines (amlodipine)
 - phenylalkylamines (verapamil)
 - benzothiazepines (diltiazem)
- **Vasodilators** and **negative chronotropes/inotropes**



Amlodipine



Verapamil

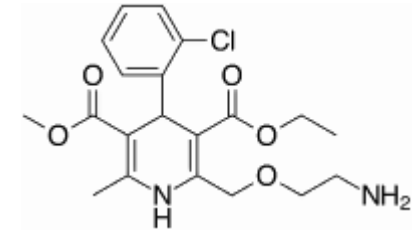


Diltiazem

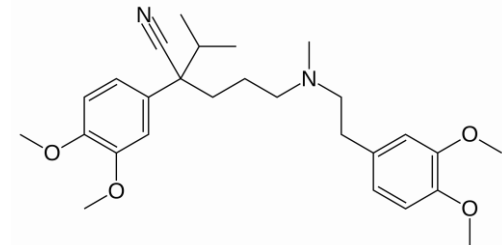
Calcium Channel Blockers

www.afratafreeh.com

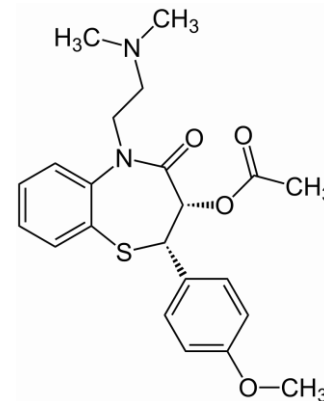
- Dihydropyridines (nifedipine) → vasodilators
 - Main effect: ↓ TPR/BP
- Non-dihydropyridines (verapamil, diltiazem)
 - Similar to β 1-blockers
 - Main effects: ↓HR; ↓ contractility
- Vascular smooth muscle effects
 - Nifedipine>Diltiazem>Verapamil
- Heart rate/contractility effects
 - Verapamil>Diltiazem>Nifedipine



Amlodipine



Verapamil



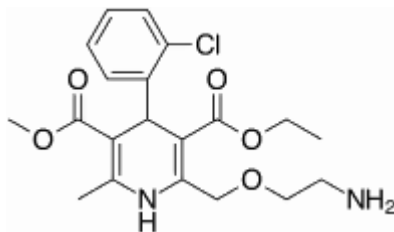
Diltiazem

Calcium Channel Blockers

www.afratafreeh.com

Dihydropyridines (amlodipine)

- Used for **hypertension**
- Flushing, headache, hypotension
 - Peripheral vasodilation
- Key side effect: **edema**
 - Pre-capillary arteriolar vasodilation
 - Flood capillaries with fluid → edema



Amlodipine



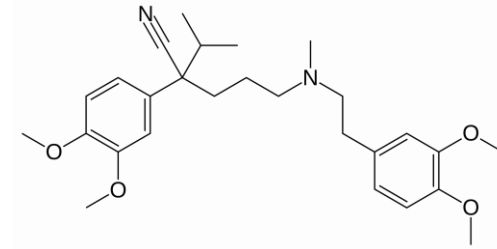
James Heilman, MD/Wikipedia

Calcium Channel Blockers

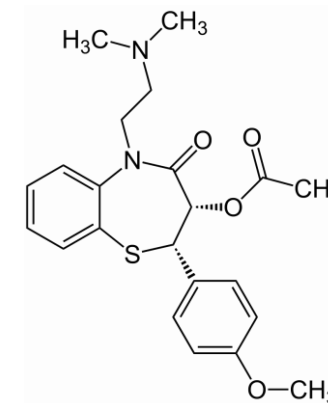
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Verapamil, diltiazem

- Used for **hypertension**
- Also used in heart disease
 - Arrhythmias (atrial fibrillation)
 - Stable angina (lower oxygen demand)
- Potential side effect: **negative inotropes**
 - Contraindicated in heart failure



Verapamil



Diltiazem

Calcium Channel Blockers

www.afratafreeh.com

Other Side Effects

- Constipation
 - Most commonly with **verapamil**
- Hyperprolactinemia
 - Seen with **verapamil**
 - Blocks calcium channels CNS → ↓ dopamine release
 - Galactorrhea
 - Leads to hypogonadism
 - Men: ↓ libido, impotence
 - Pre-menopausal women: irregular menses



Elya/Wikipedia

Calcium Channel Blockers

www.afratafreeh.com

Other Side Effects

- **Gingival hyperplasia**
 - Seen in all types CCB
 - Also with phenytoin, cyclosporine



Lesion/Wikipedia

Vaughan Williams Classification

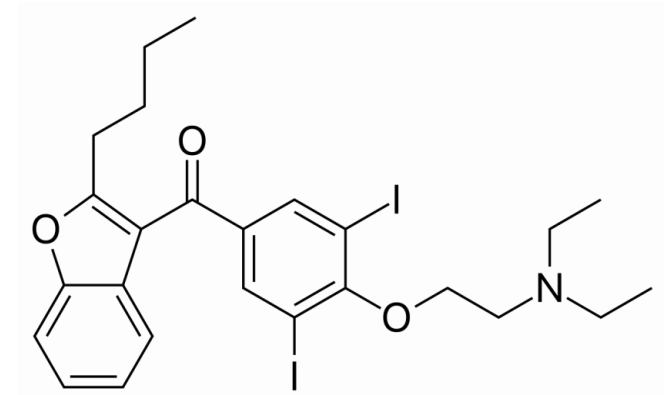
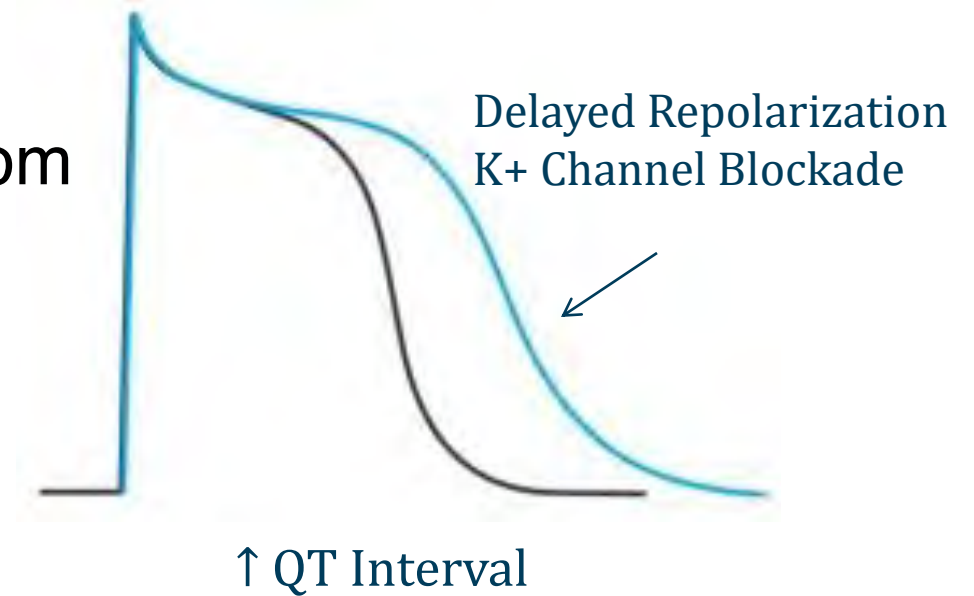
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<u>Class I</u>	<u>Class II</u>
Quinidine } Procainamide } Ia Lidocaine } Mexiletine } Ib Flecainide } Propafenone } Ic	Beta-blockers
<u>Class III</u>	<u>Class IV</u>
Amiodarone Sotalol Dofetilide Ibutilide	Ca-channel Blockers (Verapamil/Diltiazem)

Amiodarone

www.afratafreeh.com

- Class III drug
 - K channel blocker
 - Prolongs Qt interval
- Also has class I, II, and IV effects
 - Class II, IV: Slow HR
- Highly effective drug
- Suppresses atrial fibrillation
- Suppresses ventricular tachycardia

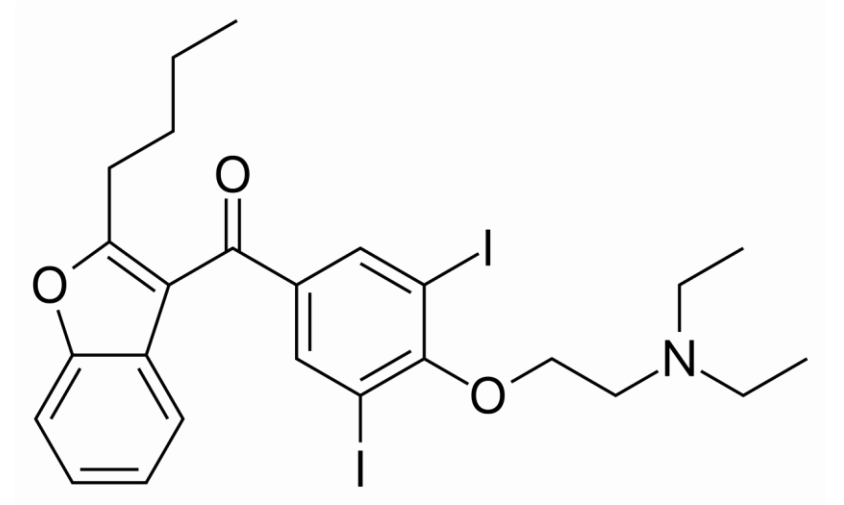


Amiodarone

Amiodarone

www.afratafreeh.com

- Highly **lipid soluble**
- Accumulates in liver, lungs, skin, other tissues
- Half-life **about 58 days**
- Once steady state reached, very long washout
- Safe in renal disease (biliary excretion)



Amiodarone

Amiodarone

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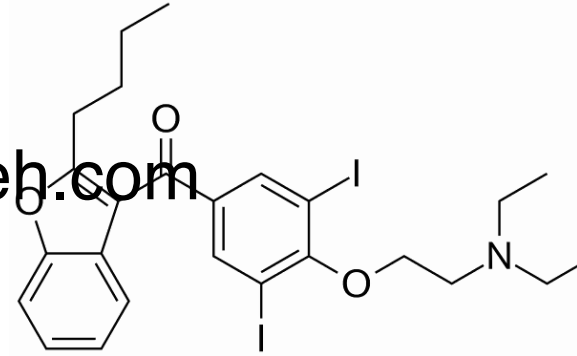
- Many potential side effects related to accumulation
- Less likely at lower dosages
- Risk accumulates over time
- Young patients on indefinite therapy at greatest risk
- Often used in older patients

Amiodarone

Side Effects

- **Hyper and hypothyroidism**
 - Contains iodine
- **Increased LFTs**
 - Usually asymptomatic and mild
 - Drug stopped if elevation is marked
- **Skin sensitivity to sun**
 - Patients easily sunburn

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Amiodarone

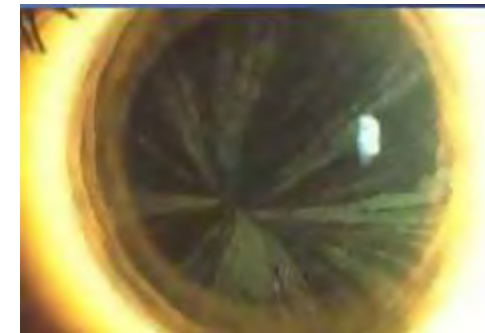
Side Effects

www.afratafreeh.com

- **Blue-gray discoloration**
 - Less common skin reaction
 - “Blue man syndrome”
 - Most prominent on face
- **Corneal deposits**
 - Secretion of amiodarone by lacrimal glands
 - Accumulation on corneal surface
 - Appearance of “cat whiskers” on cornea
 - Does not usually cause vision problems
 - See in many patients on chronic therapy



Abby Crawford/Youtube/Public Domain



Amiodarone

Side Effects

www.afratafreeh.com

- **Pulmonary fibrosis**
- Most common cause of death from amiodarone
- Foamy macrophages seen in air spaces
- Filled with amiodarone and phospholipids
- “Honeycombing” pattern on chest x-ray



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Amiodarone

Side Effects

www.afratafreeh.com

- When starting amiodarone
 - Chest X-ray
 - Pulmonary function tests (PFTs)
 - Thyroid function tests (TFTs)
 - Liver function tests (LFTs)

Sotalol and Dofetilide

www.afratafreeh.com

- Commonly used in patients with **atrial fibrillation**
- Typical case
 - Recurrent episodes symptomatic atrial fibrillation
 - Sotalol/Dofetilide started
 - Cardioversion to restore sinus rhythm
 - Sinus rhythm persists on therapy
- Other antiarrhythmics also used in this manner
 - Amiodarone
 - Propafenone
 - Flecainide

Ibutilide

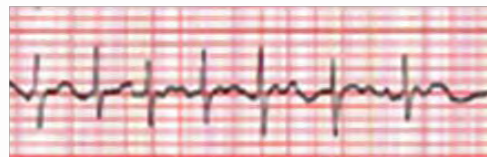
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- Intravenous drug
- Half life of 2 to 12 hours
- Used for “chemical cardioversion”



Electrical Cardioversion

Requires sedation



Ibutilide



Chemical Cardioversion

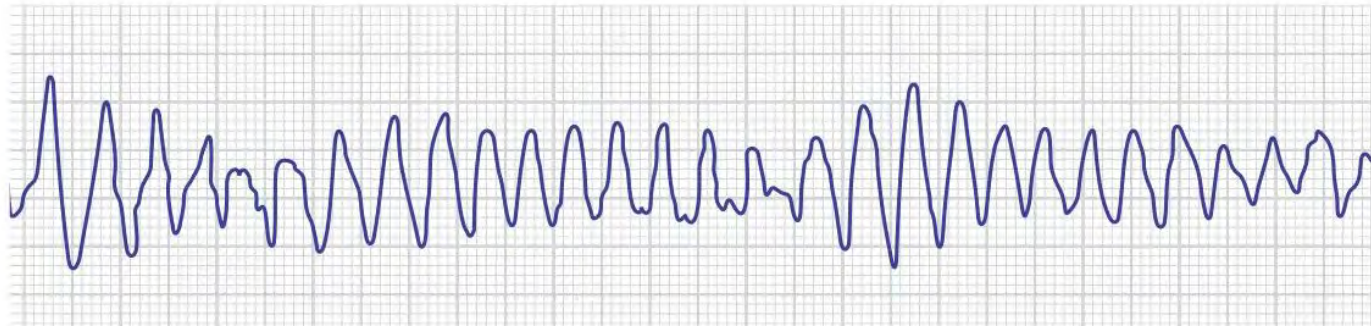
No sedation
May cause Torsade



Class III Antiarrhythmic Drugs

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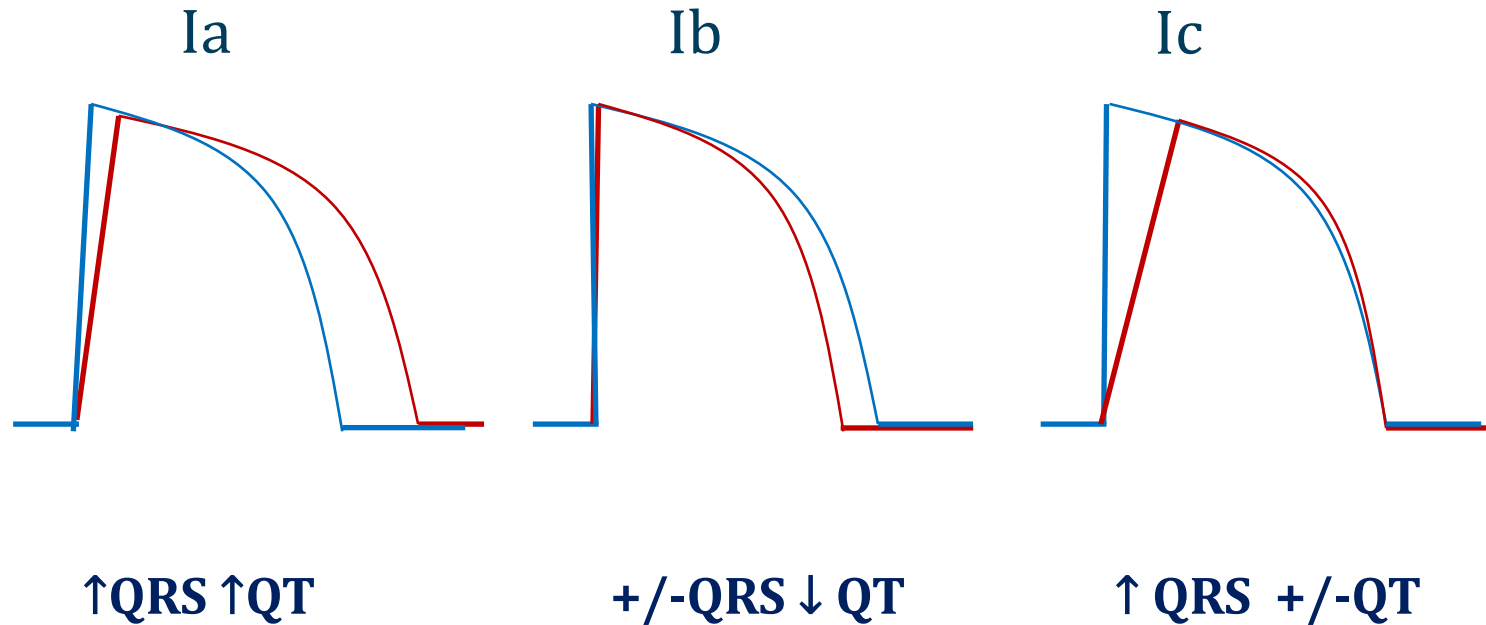
- Amiodarone, sotalol, dofetilide, ibutilide
- Major adverse effect: prolongation of Qt interval
- Increased risk of **torsades de pointes**



Class I drugs

Effects on Resting Action Potential

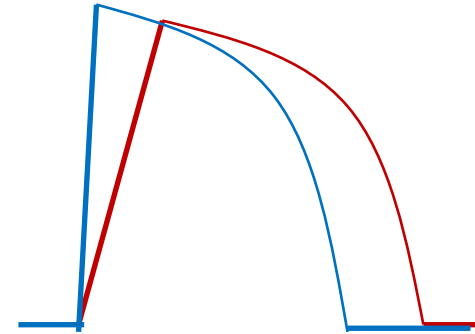
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Class Ia Drugs

Quinidine, procainamide

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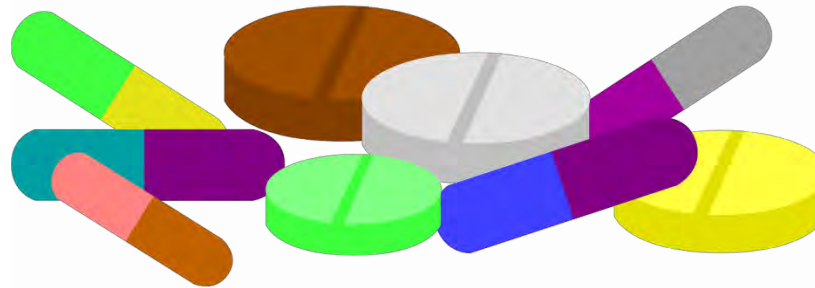


- **Prolong QRS**
- **Can also prolong Qt** ($\downarrow K^+$ outflow)
- Quinidine
 - Oral drug
 - Can decrease recurrence rate of atrial fibrillation
 - Associated with increased mortality
- Procainamide
 - Intravenous drug
 - Slows conduction in accessory pathways (WPW)
 - Used in arrhythmias associated with bypass tracts

Procainamide

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- Associated with **drug-induced lupus**
 - Classic drugs: **INH, hydralazine, procainamide**
- Often rash, arthritis, anemia
- Antinuclear antibody (ANA) can be positive
- Key features: **anti-histone antibodies**
- Resolves on stopping the drug

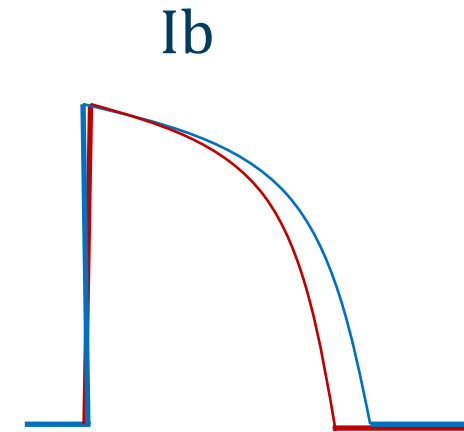


Class Ib Drugs

Lidocaine, Mexiletine

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- Na channel blockers
- Most Na channel binding in **depolarized state**
- Ischemia → more depolarized myocytes
- Effective drugs in ischemic arrhythmias
- More effective at fast heart rates
- Less time to unbind before Na channels open again
- Main use: ischemic ventricular tachycardia
 - Fast heart rates
 - Depolarized Na channels



+/-QRS ↓ QT

Class Ib Drugs

Lidocaine, Mexiletine

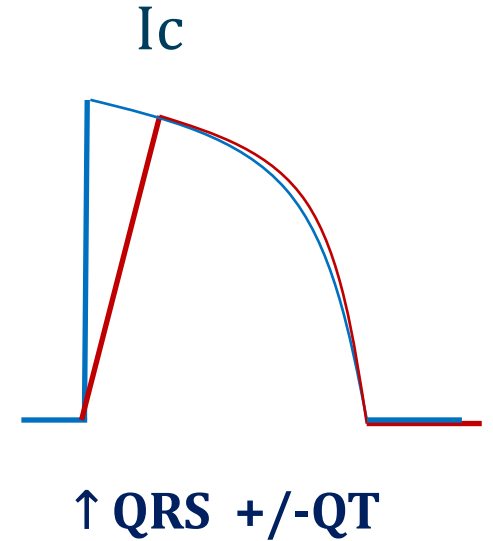
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- Lidocaine also a local anesthetic
 - Na channel nerve block
- May cause **CNS stimulation**
 - Tremor, agitation
 - Tremor in patient on Mexiletine = toxicity
- Cardiovascular side effects
 - From excessive block of Na channels
 - Bradycardia, heart block, hypotension

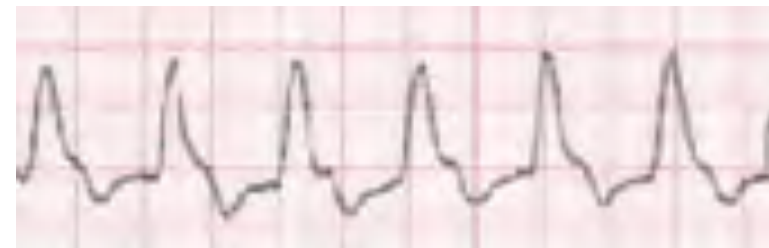
Class Ic Antiarrhythmic Drugs

www.afratafreeh.com

- Flecainide, propafenone
- Used for suppression of atrial fibrillation
- Only used in patients with “structurally normal hearts”
 - Normal LV function
 - No significant valvular disease
- Major adverse effect: **prolongation of QRS interval with exercise**



Baseline



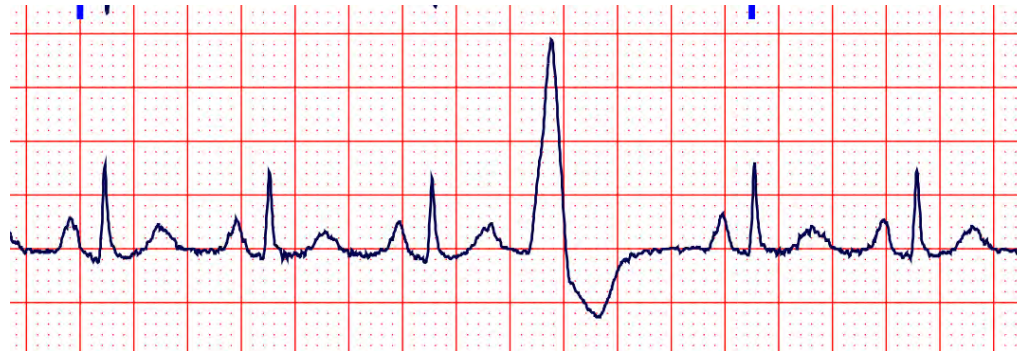
Exercise

Antiarrhythmic Drugs

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- Class I and class III agents associated with **increased mortality**
- Only used when absolutely necessary
 - Recurrent ventricular tachycardia
 - Highly symptomatic atrial fibrillation
- Not used for PACs or PVCs

Premature Ventricular Contraction



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Cardiovascular Pharmacology II

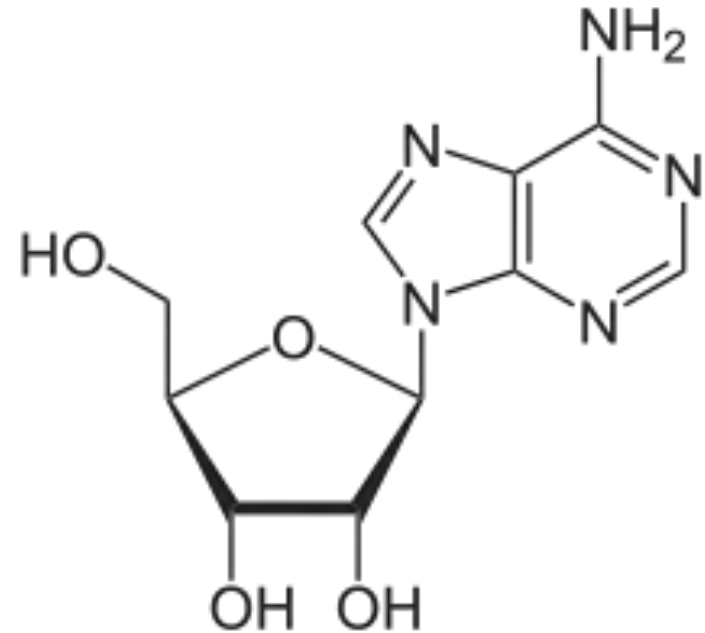
Jason Ryan, MD, MPH



Adenosine

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- AV nodal cells:
 - **Activates K⁺ channels**
 - Drives K⁺ out of cells
 - Hyperpolarizes cells: Takes longer to depolarize
- Result: **Slowing** of conduction through AV node



Adenosine

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- Short half life – given IV
- Used to treat supraventricular tachycardias
- Most common SVT: **AV node reentrant tachycardia (AVNRT)**
- Slow and fast circuits in AV node → arrhythmia
- Adenosine slows AV node conduction → arrhythmia with terminate

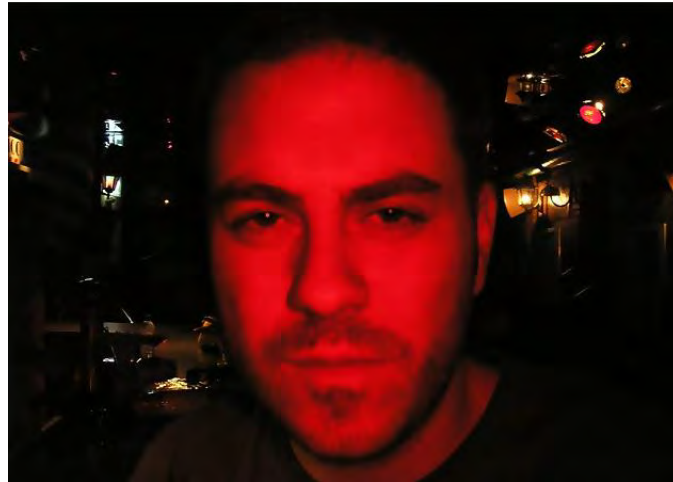
SVT Break with Adenosine



Adenosine

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- Also a vasodilator
- Causes skin flushing, hypotension
- Some patients also develop dyspnea, chest pain
- Effects quickly resolve
- Must warn patients before administration for SVT

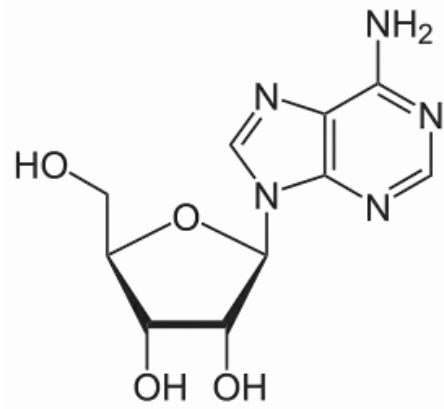


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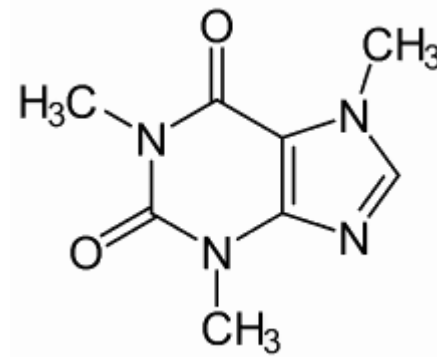
Adenosine

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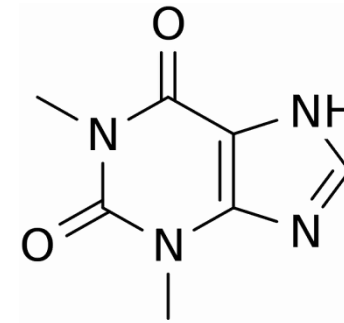
- Effects blocked by **theophylline** and **caffeine**
- Block adenosine receptors



Adenosine



Caffeine



Theophylline

Atropine

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- Muscarinic receptor antagonist
 - Parasympathetic block → ↑ HR and AV conduction
- Used in bradycardia → **↑ heart rate**
- Also speeds conduction through AV node
- Useful for bradycardia from AV block



Before Atropine



After Atropine

Atropine

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- Many **side effects** related to muscarinic block
- Toxicity:
 - Dry mouth
 - Constipation
 - **Urinary retention**
 - Confusion (elderly)

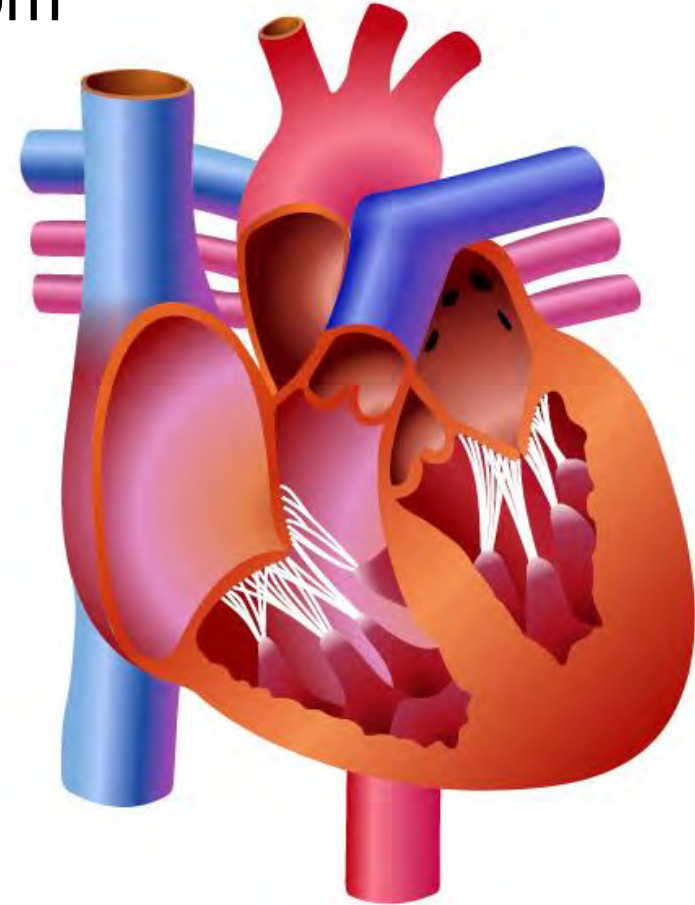


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Digoxin

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- Two cardiac effects
- **#1: Increases contractility**
 - Used in systolic heart failure with ↓ LVEF
- **#2: Slows AV node conduction**
 - Used in atrial fibrillation to slow ventricular rate

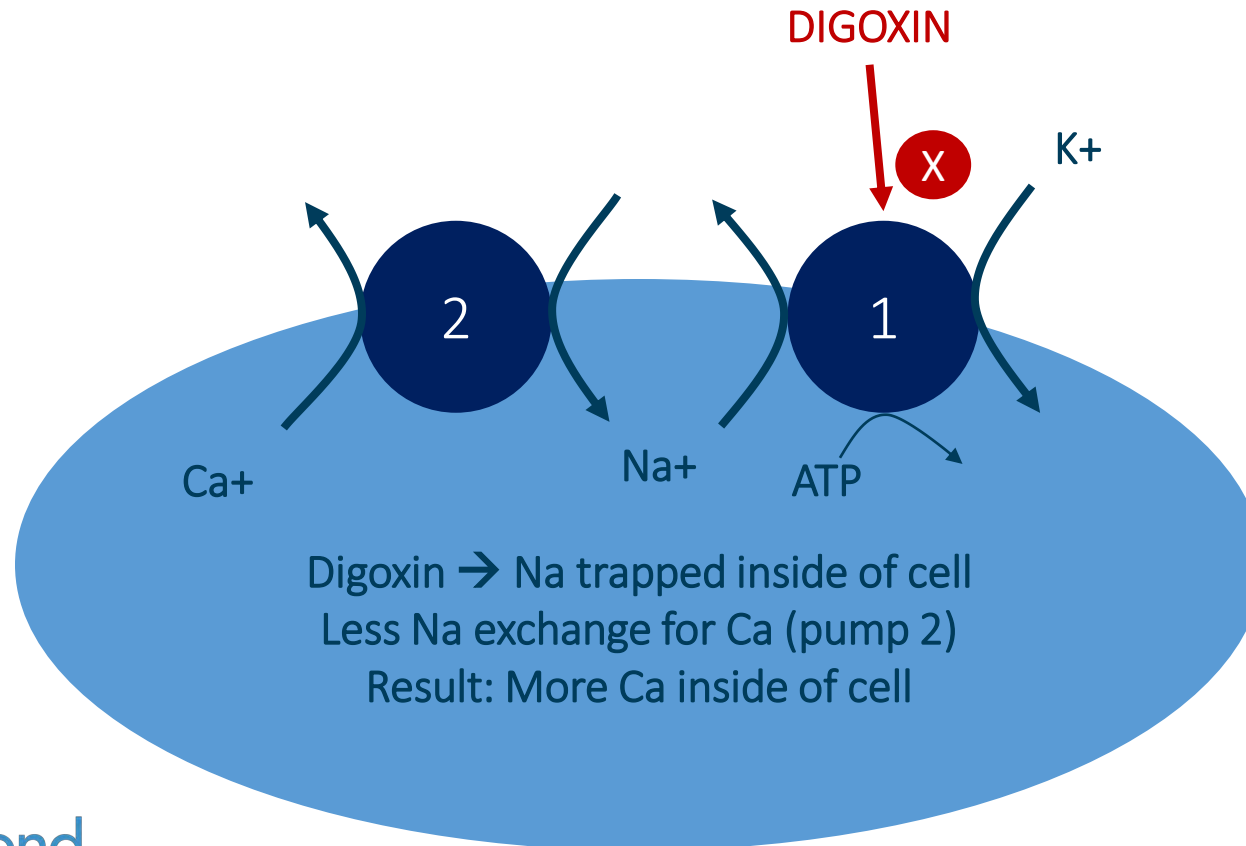


Digoxin

Increased Contractility

- Inhibits Na-K-ATPase

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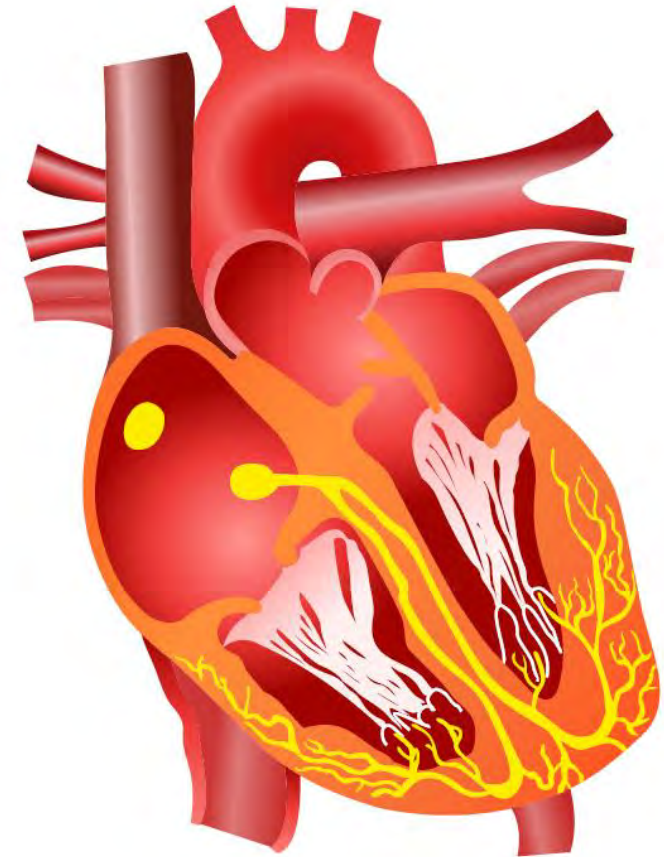


Digoxin

AV Nodal Slowing

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- Suppresses **AV node conduction**
 - Increased vagal (parasympathetic) tone
 - Separate effect from blockade of Na-K-ATPase
- Can be used to ↓ heart rate in rapid atrial fibrillation
 - Continued atrial fibrillation
 - Fewer impulses to ventricle → slower heart rate
- Effects similar to BB and CCB in AV node



Digoxin Toxicity www.afratafreeh.com

- **Renal clearance**
 - Risk of toxicity in patients with chronic kidney disease
- **Hypokalemia** promotes toxicity
 - Caused by many diuretics, especially loop diuretics
 - Digoxin patient on furosemide → toxicity
- Levels often need to be monitored

Digoxin Toxicity www.afratafreeh.com

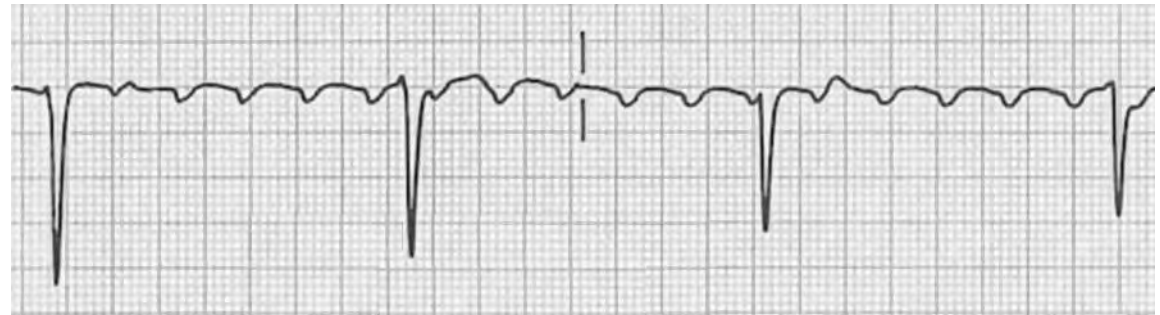
- Gastrointestinal
 - Anorexia, nausea, vomiting, abdominal pain
- Hyperkalemia
- Neurologic
 - Lethargy, fatigue
 - Delirium, confusion, disorientation
 - Weakness
- Visual changes
 - Alterations in color vision, scotomas, blindness
- Cardiac arrhythmias

Digoxin Toxicity www.afratafreeh.com

Cardiac Toxicity

- More Na inside of cell
- ↑ resting potential atrial/ventricular cells
- Increased automaticity
- **Extra beats:** PACs, PVCs
- Evidence of **AV node block**

Atrial Tachycardia with AV block



Digoxin Toxicity

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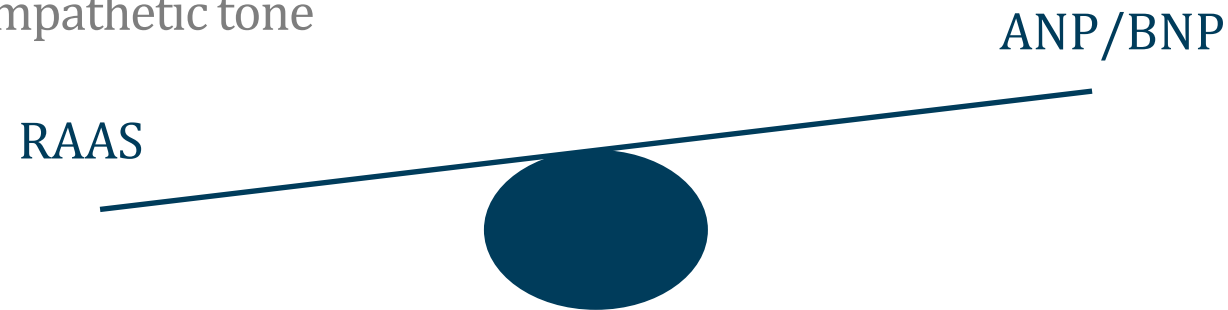
Treatment

- **Digibind**
 - Antibody binds digoxin
- Correct **hyperkalemia**

Sacubitril

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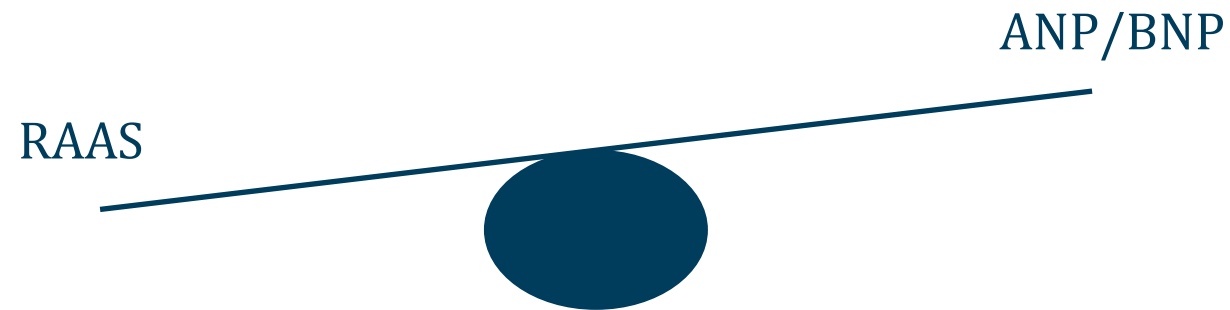
- Neprilysin inhibitor
- Neprilysin: Degrades atrial/brain natriuretic peptide
- Inhibition → **↑ANP/BNP**
 - Antagonists to RAAS system
 - Vasodilatation
 - Natriuresis (sodium excretion)
 - Diuresis (water excretion)
 - Reduced sympathetic tone



Sacubitril

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- Entresto: oral combination sacubitril/valsartan
 - Valsartan: angiotensin receptor blocker (ARB)
- ↓ **mortality**
- ↓ **hospitalizations**



Sacubitril

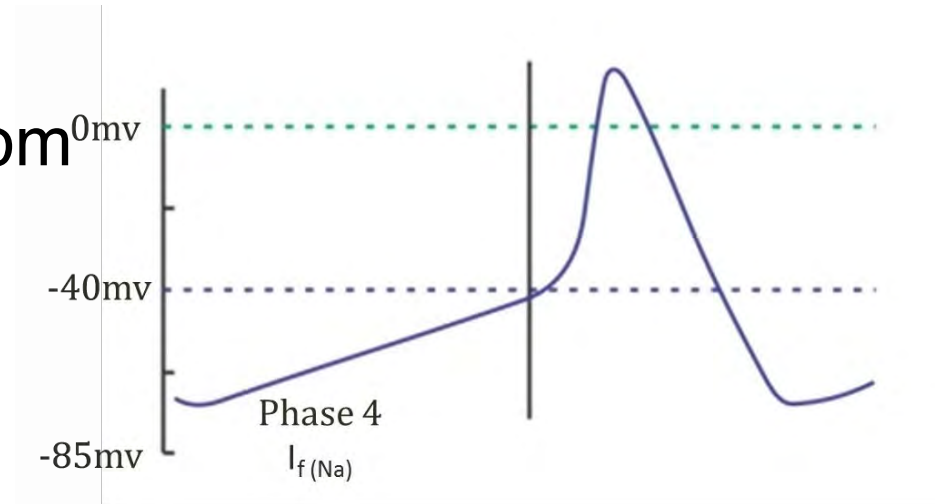
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- Studied in combination with valsartan
- Many side effects similar to ARBs
- Hypotension
- Hyperkalemia
- Angioedema
 - Tissue swelling
 - Rare, feared adverse effect
 - Caused by elevated bradykinin levels
 - Neprilysin degrades bradykinin
 - **Cannot be given together with ACE inhibitors**

Ivabradine

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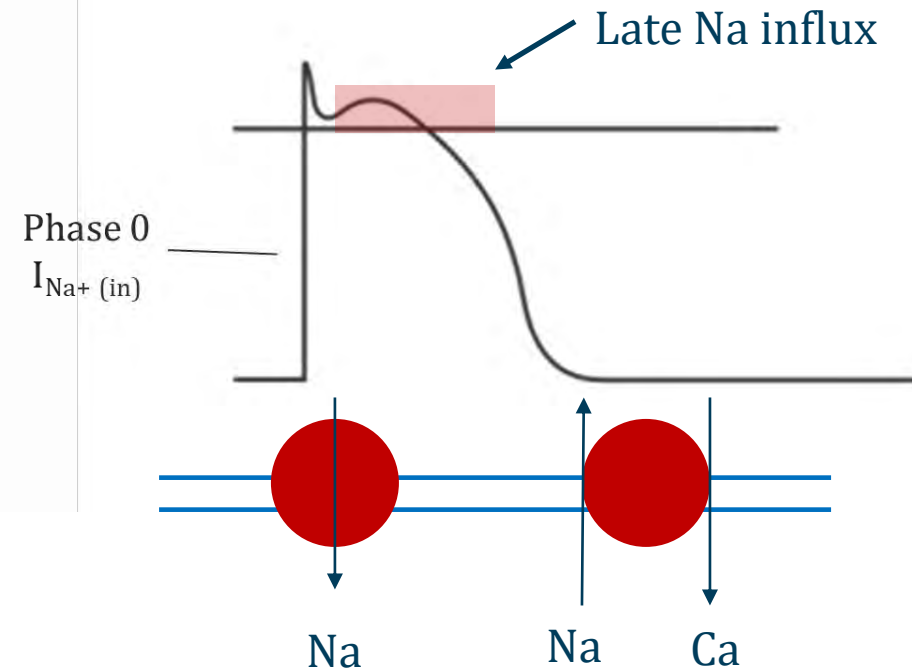
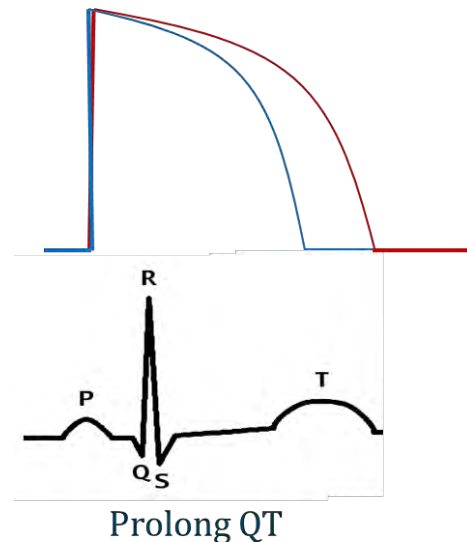
- Selective sinus node inhibitor
- Elevated HR → worse prognosis
- Slows heart rate without ↓ contractility
- Inhibits SA pacemaker **“funny current” (I_f)**
- Used in patients on max-dose beta-blocker with ↑HR
- Limited evidence of ↓ mortality and hospitalizations
- About 15% of patients experience **phosphenes**
 - Visual disturbance
 - Rings or spots of bright light



Ranolazine

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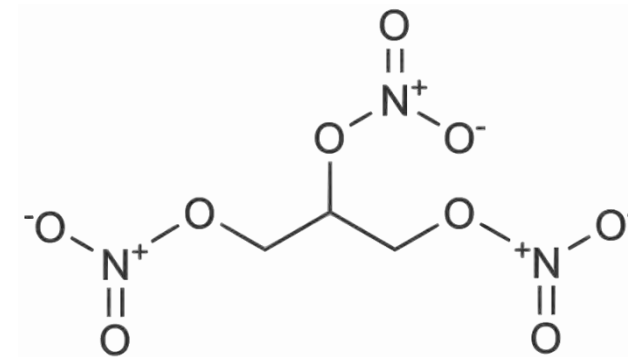
- Used for refractory angina
- Inhibits **late sodium current**
- Reduces **calcium overload** → high wall tension
- Reduces **wall tension** and O₂ demand
- QT prolongation



Nitrates

www.afratafreeh.com

- Used to treat stable angina
- Converted to nitric oxide → vasodilation
- Predominant mechanism is **venous dilation**
 - Bigger veins hold more blood
 - Takes blood away from left ventricle
 - **Lowers preload**
- Also arterial vasodilation (art << veins)
 - Increase coronary perfusion
 - Some peripheral vasodilation



Nitroglycerine

Nitrates

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- ↓ preload → ↓ cardiac output
- **Sympathetic nervous system activation**
- Increased heart rate/contractility
 - Increases O₂ demand
 - Opposite of what we want to do for angina
- **Co-administer beta-blocker or Ca channel blocker**
 - Blunts “reflex” effect

Nitrates

Adverse Effects

- Headache (meningeal vasodilation)
- Flushing
- Hypotension

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Nitrates and Erectile Dysfunction

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- ED common among men with coronary artery disease
- Sildenafil **contraindicated** in men taking nitrates
 - Life-threatening hypotension may occur



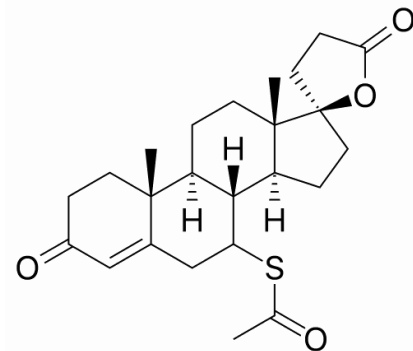
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Spirolactone, Eplerenone

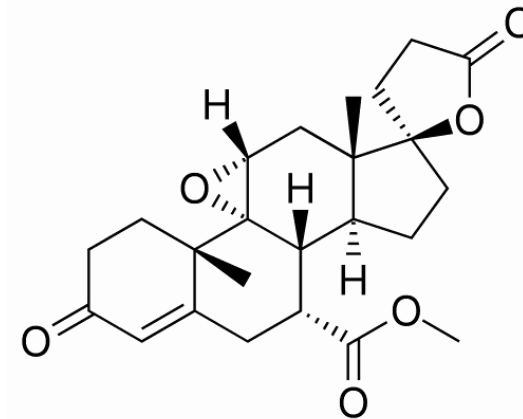
Potassium-sparing diuretics

- Diuretics: $\uparrow$ Na/H₂O excretion
- “Spare” potassium
 - Unlike other diuretics, do not increase K⁺ excretion
- **HYPERkalemia is side effect**
- Reduced mortality
- Reduced hospitalization rate

Check BUN/Cr and K⁺ before starting drug
Monitor BUN/Cr and K⁺ after starting drug



Spirolactone



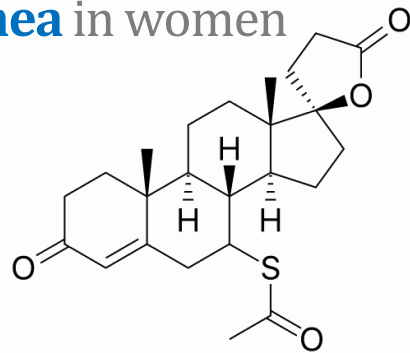
Eplerenone

Spironolactone, Eplerenone

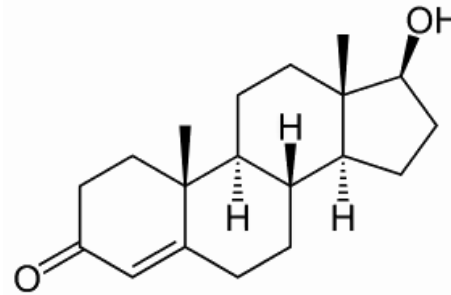
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Potassium-sparing diuretics

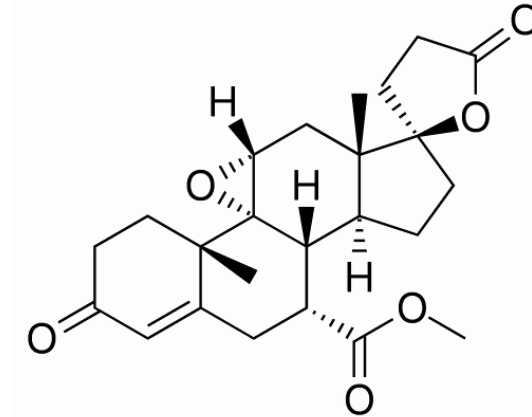
- Similar structure to testosterone
 - Blocks testosterone effects
 - **Gynecomastia** in men
 - Eplerenone: No gynecomastia
- Derivative of progesterone
 - Activates progesterone receptors
 - **Amenorrhea** in women



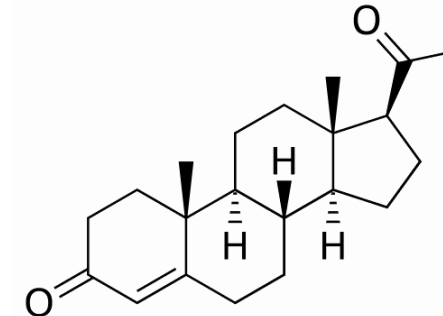
Spironolactone



Testosterone



Eplerenone



Progesterone

ACE Inhibitors and ARBs

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- **ACE Inhibitors**
 - Captopril, Enalapril, Lisinopril, Ramipril
 - Block conversion AI → AII
- **Angiotensin Receptor Blockers (ARBs)**
 - Candesartan, Irbesartan, Valsartan
 - Directly block AII receptor
- Both classes: **↓ mortality and hospitalizations**
- Side effects
 - Hyperkalemia (↓aldosterone)
 - Renal failure (↓GFR)

ACE Inhibitors

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Unique Side Effects

- Due to increased **bradykinin**
- Dry Cough
 - Occurs in ~10% of patients
- Angioedema
 - Swelling of face, tongue
 - Can be life-threatening

