

BCHM 4608
Common Cardiac Diseases and Diagnostics
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Cardiology-in application to BCHM4608

- Hypertension
- Coronary artery disease
- Chest pain evaluation
- Acute coronary syndrome
- Congestive heart failure
- Valvular heart disease
- Cardiac arrhythmias

Hypertension

Systolic HTN, most frequent in elderly after age 50 or 60s

Diastolic HTN spontaneously decrease with age

Systolic blood pressure and pulse pressure are important predictors of end organ damage

Pathophysiology

- In large arteries, aging, deposition of calcium salts, elastin and collagen that increase rigidity of vessel wall---AORTA
- Lessen pulsatile output from the heart
- Increase systolic BP while Diastolic BP and coronary perfusion decrease
- Increase end organ damage- Systolic HTN

Normal cut -
section of
artery



Tear in
artery
wall



Fatty material
is deposited
in vessel wall



Narrowed
artery
becomes
blocked by
a blood clot

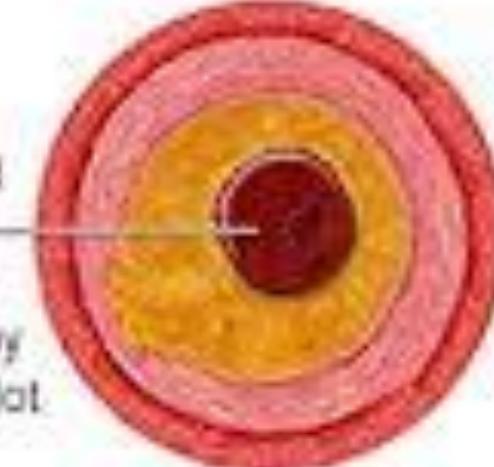
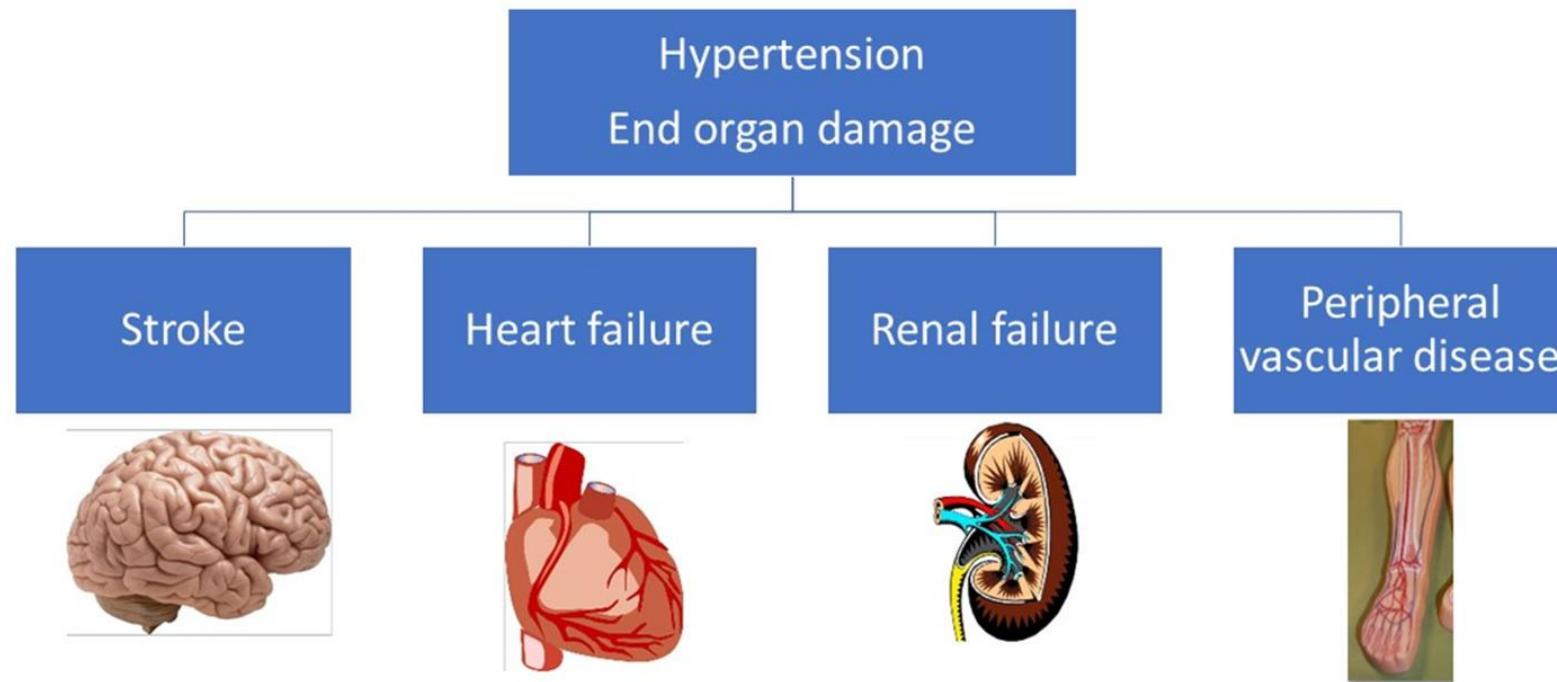


Image: Indian Association of Palliative Care



Pulse pressure

- Difference between systolic and diastolic blood pressure
- Depends on compliance of aorta and heart stroke volume
- Indicative of cvs diseases

2023 HTN guideline update

2017 ACC/AHA

- HTN define $>130/80$
- **Nl <120/80**
- Elevated 120-129/ <80
- Htn stage I 130-139/80-89
- Htn stage 2 $>140/90$

2023 ESH

- Define $>140/90$
- **Optimal <120/80**
- **Nl 120-129/80-84**
- High nl 130-139/85-89
- Grade I 140-159/90-99
- Grade 2 160-179/100-109
- Grade 3 $>180/110$

2023 HTN guideline update

2017 ACC/AHA

- BP targets for rx
- 18-64 <130/80
- 65-79 <130/80
- >80 <130/80
- For HTN and CKD
- Goal <130/80
- For HTN, CAD, DM, CVS
- Goal <130/80

2023 ESH

- BP targets for rx
<130/80
- <140/80
- 140-150/<80
- <140/90
- <130/80

Importance of accurate BP measurement

- Importance of standardized, accurate BP measurement and rec office-based measurement for dx
- ESH rec against use of cuffless devices
- Multiple office BP measurement and tracking at home or ABPM before dx htn.

Drug Therapy Initiation

ACC/AHA

- ▶ Established CVD
 $>130/80$
- ▶ CVS risk $>10\%$
- ▶ $>140/90$ regardless of CVS risk or CVD
- ▶ No age distinction
- ▶ Single pill combination
- ▶ Combination for stage 2
 $>20/10$ above target

EHS

- ▶ Established CVD $>130/80$
- ▶ $>140/90$ regardless of CVS risk or CVD
- ▶ >80 age, rx when SBP
 >160
- ▶ Single pill combination
- ▶ ACE/ARB with CCB or diuretics

Key points

- For HTN pts with CAD, DM, CVS <130/80
- For HTN pts with CKD <130/80 (AHA) vs <140/90 (ESH)

Drug therapy for Hypertension

- Focus on assessing patients” global risk for CVD
- Choose a medication or combination of medications that reduce to desire range

JNC on Prevention, detection, Evaluation, and Treatment of HTN

JNC 8

- Important distinction in drug therapy recommendation-
- Identification of patients with compelling indications versus patient w/o
- Compelling indications are comorbid conditions where specific classes of Anti-BP drugs are recommended to decrease M/M associated with the comorbid condition

Compelling indications

- Chronic kidney disease
- Diabetes
- Coronary disease
- History of Stroke
- History of MI
- Heart failure
- All these need specific class of drugs

Five classes of Hypertension drugs

- ▶ Thiazides
- ▶ ACE inhibitors
- ▶ ARBs/ Renin inhibitors
- ▶ Beta blockers
- ▶ Calcium channel blockers

Combination therapy

- JNC 8- initiate in patients whom single agents are unlikely to achieve needed BP targets (i.e. those with BP >20 above systolic or 10 > diastolic

Conclusion

- Goals of therapy for HTN need to focus on reducing overall CV risk
- Most patients will require 2 or more drugs to achieve goals
- JNC 8 recommend use of ACE as an option for all 6 of the compelling indications

Use of BP drug therapy...why so many?



Use of BP drug therapy



Use of BP combination chemotherapy



Recommendation

- Objective is to attain and maintain goal
- Try to reach goal in one month
- Increase or add second drug til goal reached
- Add third drug if goal not achieved
- Do not us ACE and ARB together

Other classes of drugs can be used if goal not reached (Grade E)

I cannot get my blood pressure down



Ambulatory Blood Pressure Monitoring

- Normal BP by ABPM day average <120/80 and night average <100/65
- Indicated for :
- Suspected white coat htn
- Masked hypertension (bp lower in office)
- Resistant htn
- Hypotension sx with bp med rx

Secondary causes of HTN

- When to suspect
- **Onset of young <20-30 or >60 with dbp**
- Resistant HTN despite 3 or more drugs, include diuretic
- Recurrence of HTN in a well controlled patient

Major causes of secondary HTN

- Kidney dz
- Renal artery stenosis
 - young age- fibromuscular dysplasia
 - old age- atherosclerosis
- Pheochromocytoma- tumors in adrenal medullar that release catecholamines periodically- HA, sweating palpitations, anxiety, weight loss, episodic or sustained hypertension

Other causes of secondary HTN

- Hyperaldosteronism- adenoma that produces aldosterone or bilateral hyperplasia
- Hypercortisolism-Acne, hirsutism, striae, moon face
- Coarctation of aorta- difference of rt and left arm BP, diminished femoral pulse
- OSA

Hypertensive Urgency

- SBP >180/110 without signs or sx of acute or impending organ damage.
- Often asx or mild HA
- Lower bp <25% first hr then <160/100 next 2 to 6 hrs
- Risk of rapid bp reduction, AKI, MI or Stroke

Hypertensive Emergency- DO NOT MISS !

- Sig BP elevation causing acute organ damage or dysfunction such as ischemic or hemorrhagic stroke, encephalopathy, AKI, MI, aortic dissection or HF
- No specific threshold syndrome defined
- BP goal reduced to <140 first hr or 120 if aortic dissection

Coronary artery disease

- In U.S. approx. 1 million die from CVS disease each year, 25 % are sudden death
- Leading cause of death among men and women

Pathophysiology of chronic CAD

- Imbalance between myocardial oxygen supply and demand
- Increase myocardial demand during exercise and stress is inadequately met because of **impaired coronary blood flow**
- Atherogenesis in epicardial vessels is the major role (refer to text regarding path)

- Chronic CAD 冠状动脉疾病 progress to Chest pain to Acute Coronary Syndrome 急性冠状动脉综合症

Acute coronary syndrome 急性冠状动脉综合症

- Risk of MI 心肌梗死
- Atherosclerotic plaque formation
- Atherosclerotic plaque covered by layer of connective tissue called fibrous cap that ruptures
- Thrombus formation

Image: Wikipedia

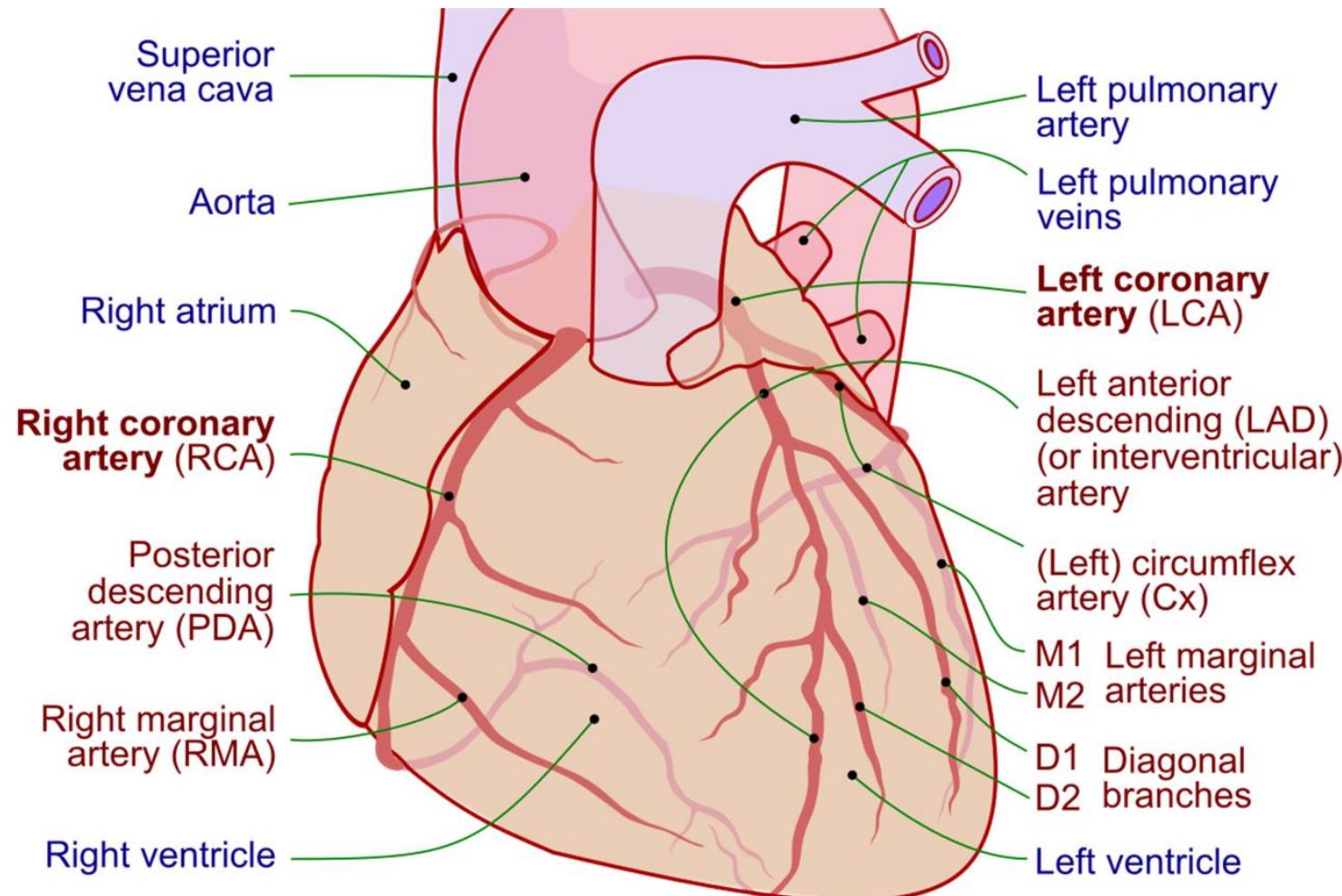
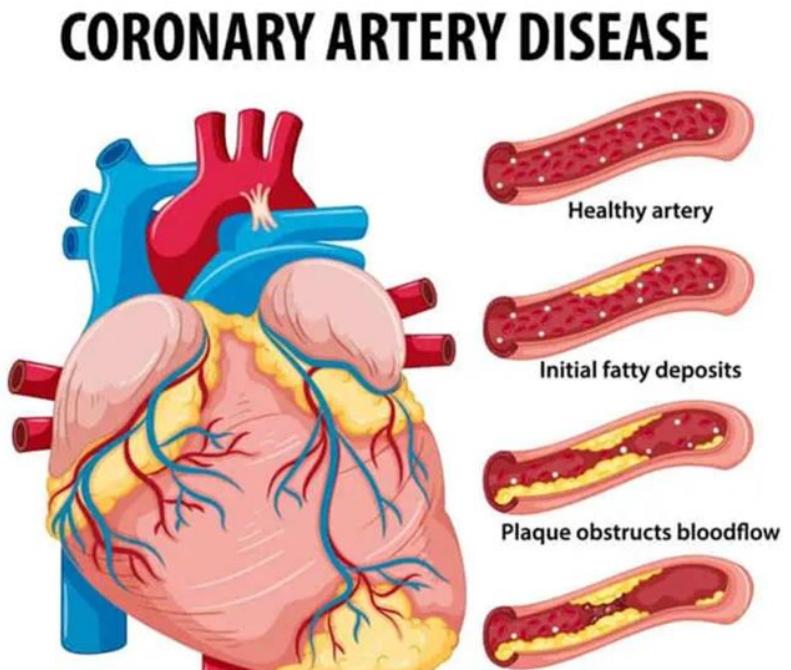
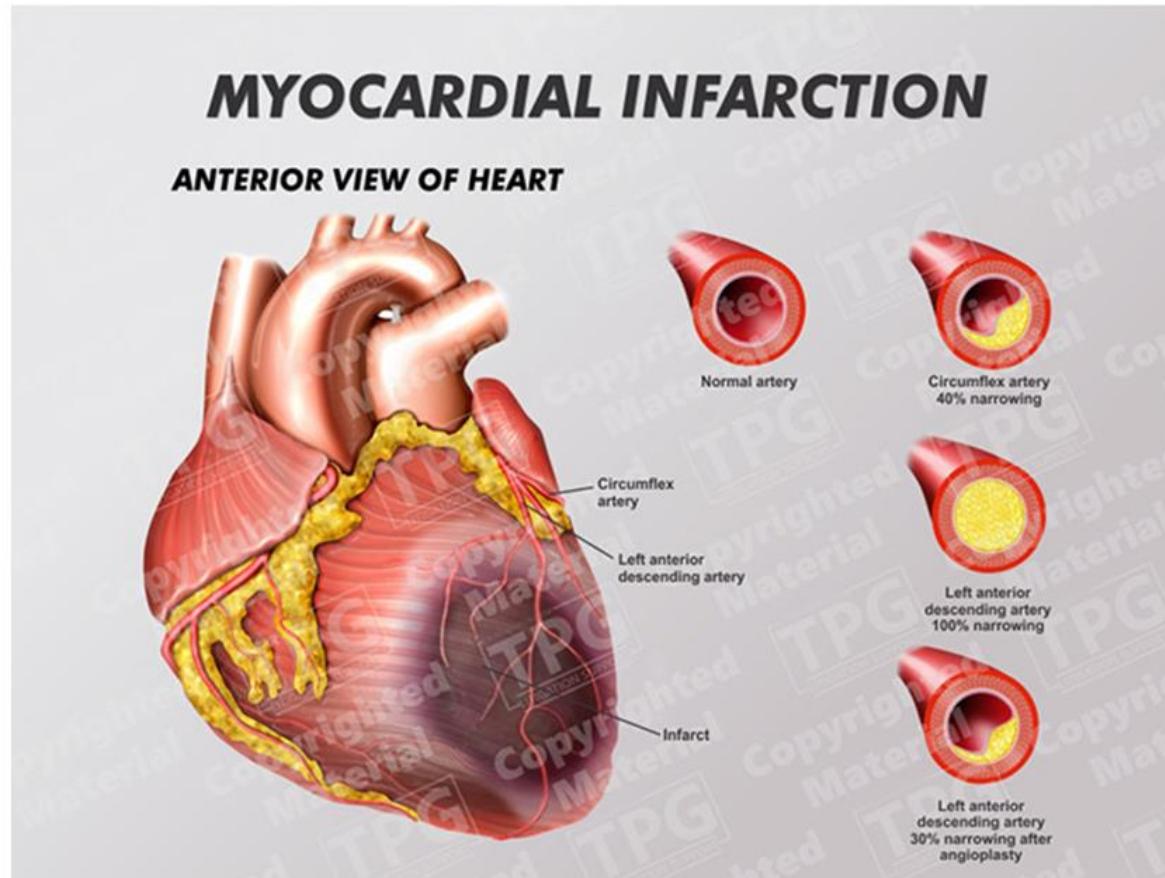


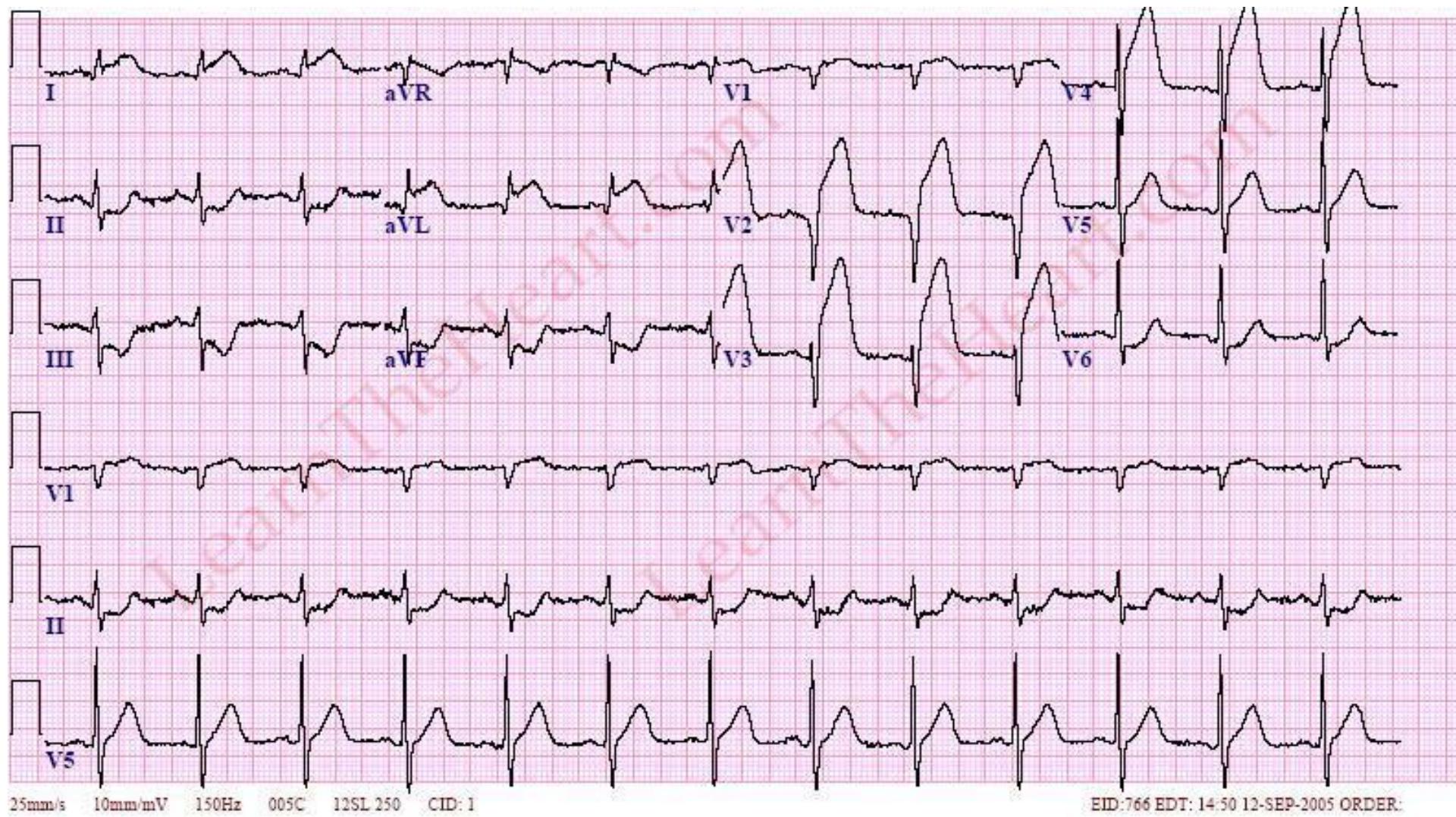
Image: continental hospitals.com

Atherosclerosis: endothelial injury >> lipid accumulation (LDL) >> inflammatory response>> foam cell formation >>>smooth muscle proliferation>> plaque growth and rupture



<https://presentationgroup.com/product/myocardial-infarct/>





DO NOT MISS & KNOW WHEN TO REFER!

Clinical presentation and assessment of chest pain

- 1. **Cardiac Risk factors** (potential future risks) and **clinical ASCVD** (actual presence of atherosclerotic CVS disease)
- 2. **Clinical symptoms** (Does the chest pain sounds real?)
- 3. **EKG**
- **KNOW T...H...E...S...E**

Cardiac risk factors

- ▶ Older age
 - ▶ Male
 - ▶ Hypercholesterolemia:
 - ▶ (High LDL, low HDL)
 - ▶ Smoker
 - ▶ Hypertension
 - ▶ DM
 - ▶ Family hx of CAD, 1st degree relative
 - ▶ obesity
 - ▶ postmenopausal
 - ▶ Lp(a), homocysteine,
High Triglycerides

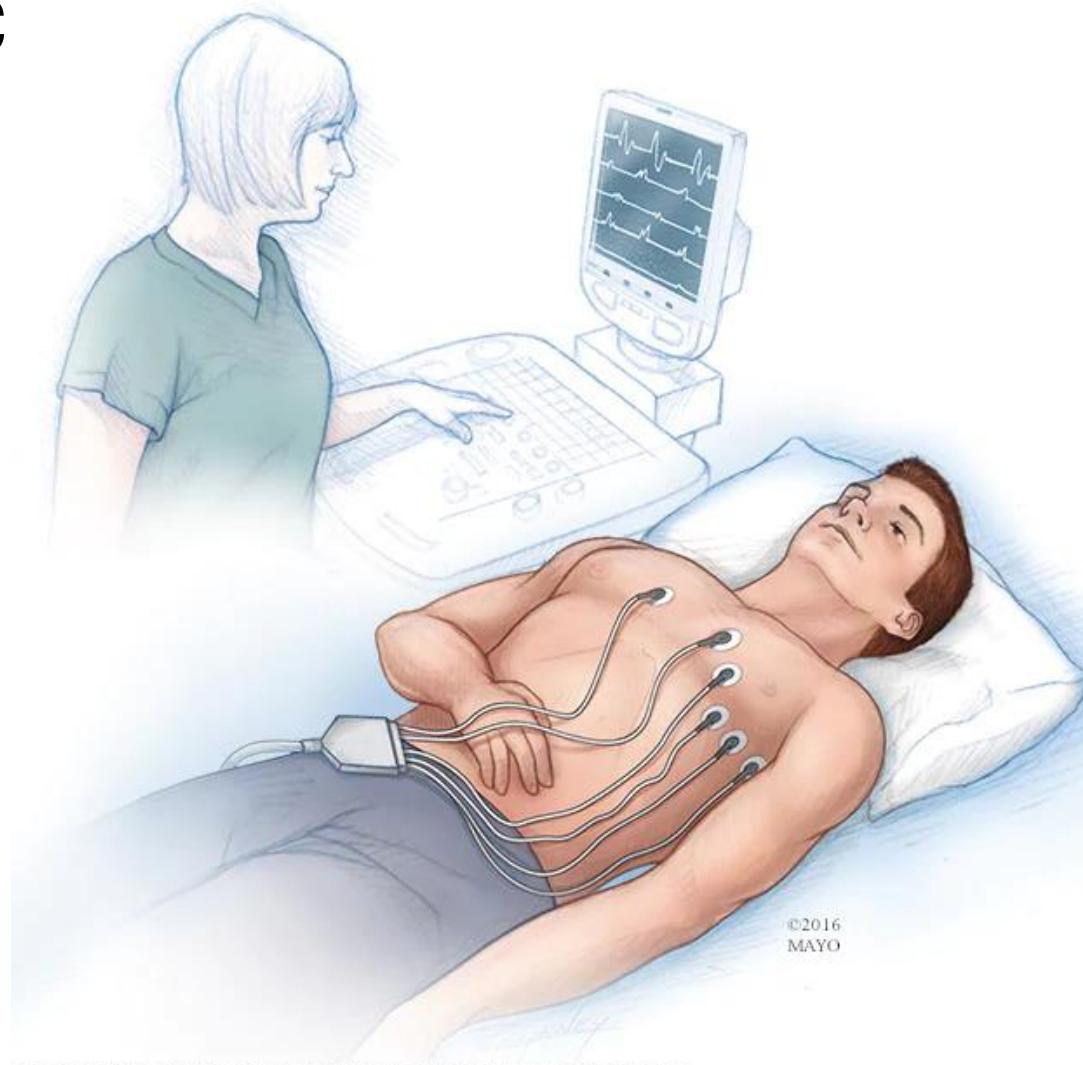
Clinical ASCVD (clinical atherosclerotic cardiovascular disease)

- Acute coronary syndromes history
- History of MI
- Stable or unstable angina
- Coronary or other arterial revascularization
- Stroke
- PAD
- TIA

Clinical symptoms (how is the chest pain sounds to you?) KNOW THESE !

- ▶ Chest pain, pressure
- ▶ location
- ▶ DOE, PND, Orthopnea
- ▶ Radiation
- ▶ Duration
- ▶ Characteristics of pain
- ▶ Aggravating and relieving factor
- ▶ diaphoresis

Mayo clinic

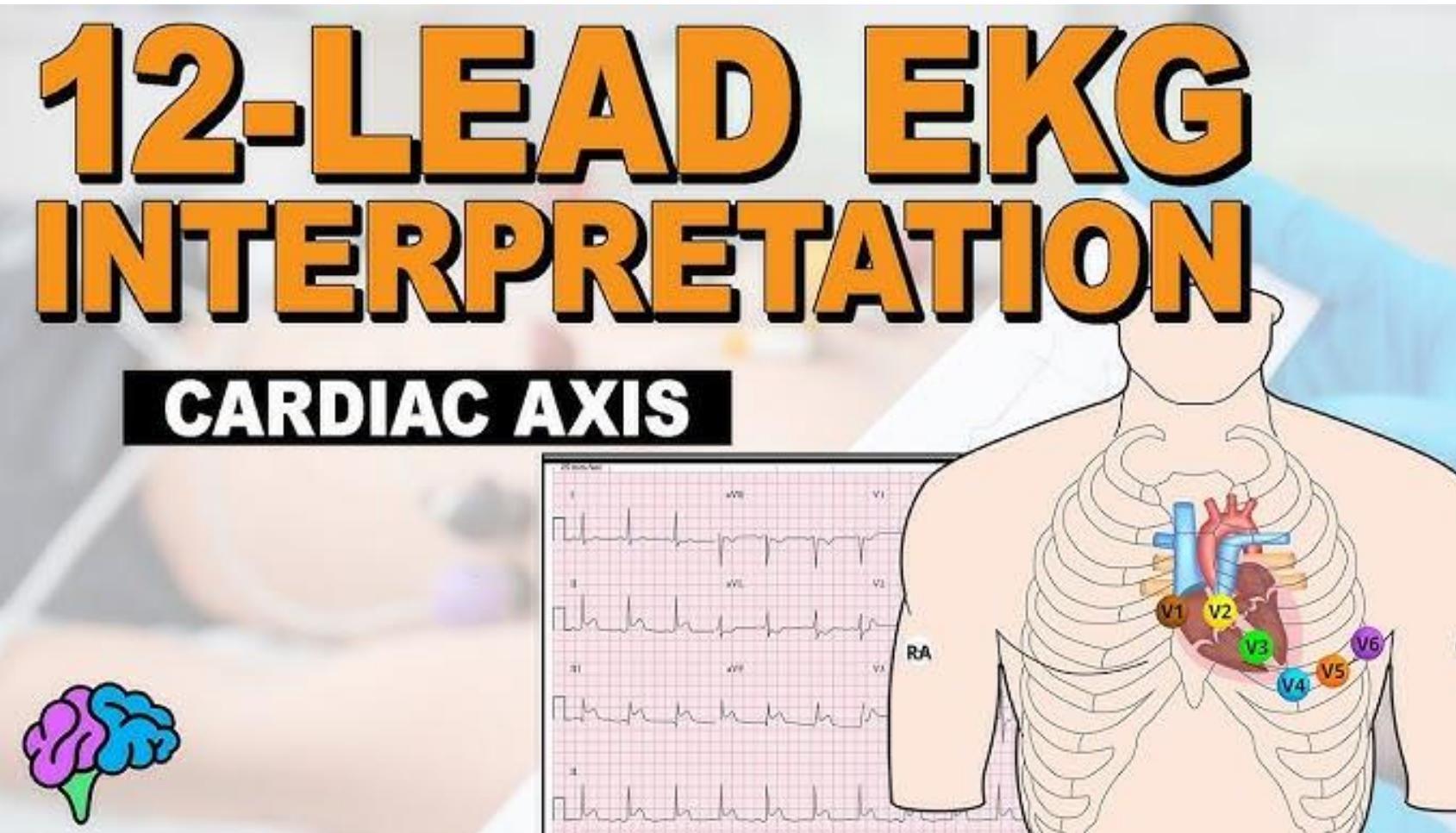


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12-LEAD EKG INTERPRETATION

CARDIAC AXIS



EKG

- What does EKG tell us?
- Q waves (possible MI)
- LVH, AE
- Arrhythmias
- Blocks
- A normal EKG does not rule out CAD

Whom to send for cardiac w/u

- Consider pretest probability based on all 3 criteria:
 - Refers to prevalence of CAD in the population being tested
 - Likelihood of a patient having CAD

Example #1

- 25 year old male presented with chest pain, sharp, seconds, no radiation, no SOB, Pain increased with body position, relieved with rubbing etc.
- Risk factor: none, father died from MI age 97
- EKG normal

Example #2

- 76 year old woman chest pain, substernal, pressure like, last one minute, relieved with rest, associate with SOB, radiate to back, chest pain upon gardening
- Risk factor: age, hypertension, LDL 170
- EKG T wave inversion V1, V2, V3

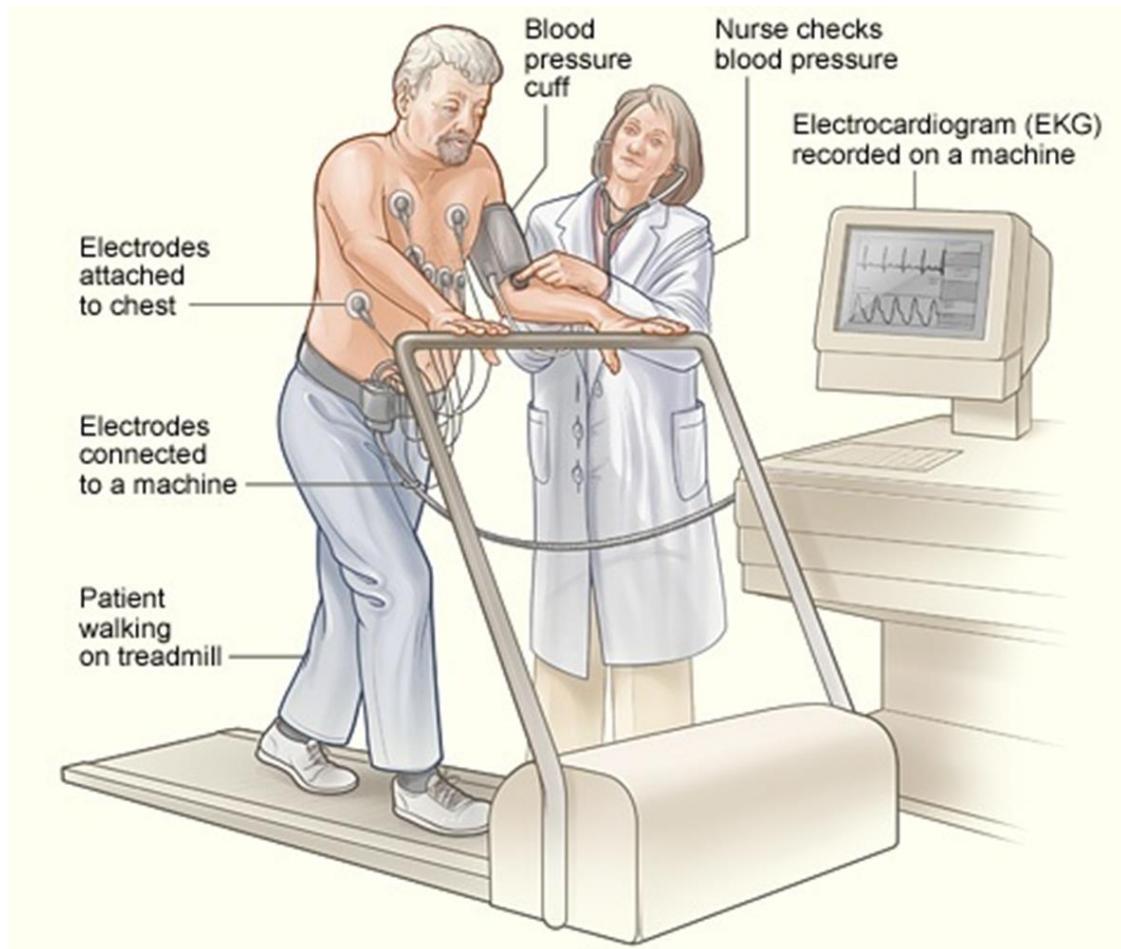
Example #3

- 52 year old male with chest pain sharp, 1-2 minutes upon exertion, no SOB, pain relieved with rest, feeling dizzy when chest pain comes, undergoing divorce, jog 40 minutes per day
- Risk factor, age, quit smoking 5 years ago, LDL 120, one brother died of MI age 50
- EKG normal

Stress tests

- Look for abnormal hemodynamic responses to exercise treadmill testing
- HR and BP major determinants of oxygen demand ($HR \times SBP$) at least work ≥ 25000 (rate-pressure product)
- Most effective used in pts with intermediate pretest probability of CAD
- Low pretest such as atypical sx, low risk \gg false positive test \gg unnecessary tests

Image: Magnolia Specialist Centre



Exercise testing for diagnosis and prognosis

- We are asking two questions:
- Does the patient have CAD
- Is he or she likely to die or suffer a CAD event soon

Prognostic variables-variables measured during treadmill that predict outcomes

- Exercise duration (longer the better), strongest prognostic indicator
- Exercise hypotension (failure of C.O. associated with CAD)
- Exercise hypertension (above 190-220/110) predict future hypertension
- Chronotropic incompetence (failure of HR to increase), predict all cause of CVS death
- Heart rate recovery (predict all cause of mortality and CVS events, sudden death)
- Ventricular ectopy (life threatening) esp sustained V tach or V fib

Other types of stress tests

- ▶ Stress Echocardiogram (if patient can run) treadmill + echo
- ▶ Pharmacologic
 - Dobutamine-beta agonist increase HR/contractility-Dobutamine stress echo
 - Adenosine, Dipyridamole are vasodilators produce hyperremia and flow disparity between obstructive vs nonobstructive myocardium
- ▶ Imaging-Thallium-201, potassium analog
 - Taken up by myocardial cells, hypoperfused myocardium decreased uptake, redistributes later

Image: <https://4hcm.org/stress-echo/>

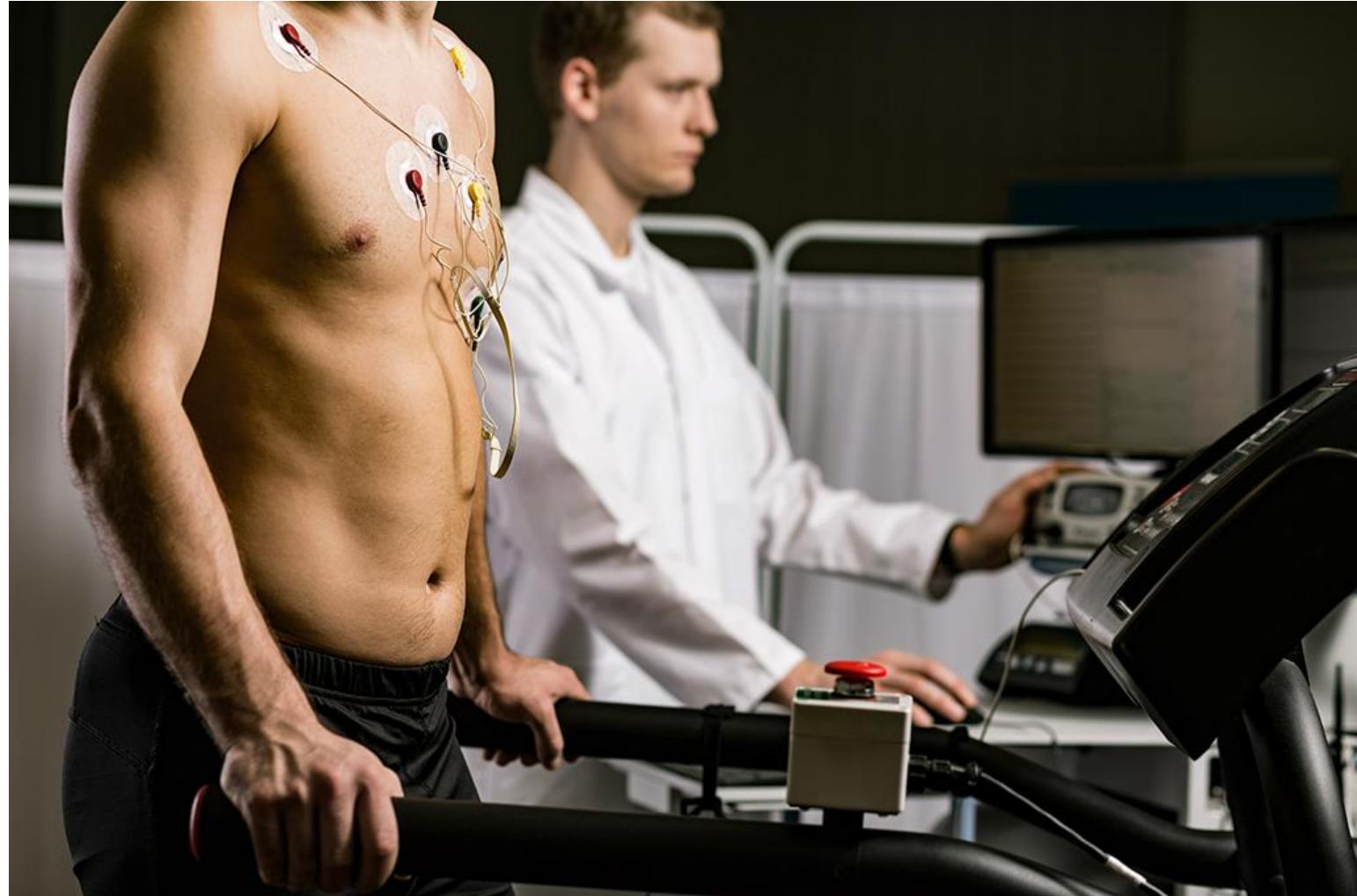
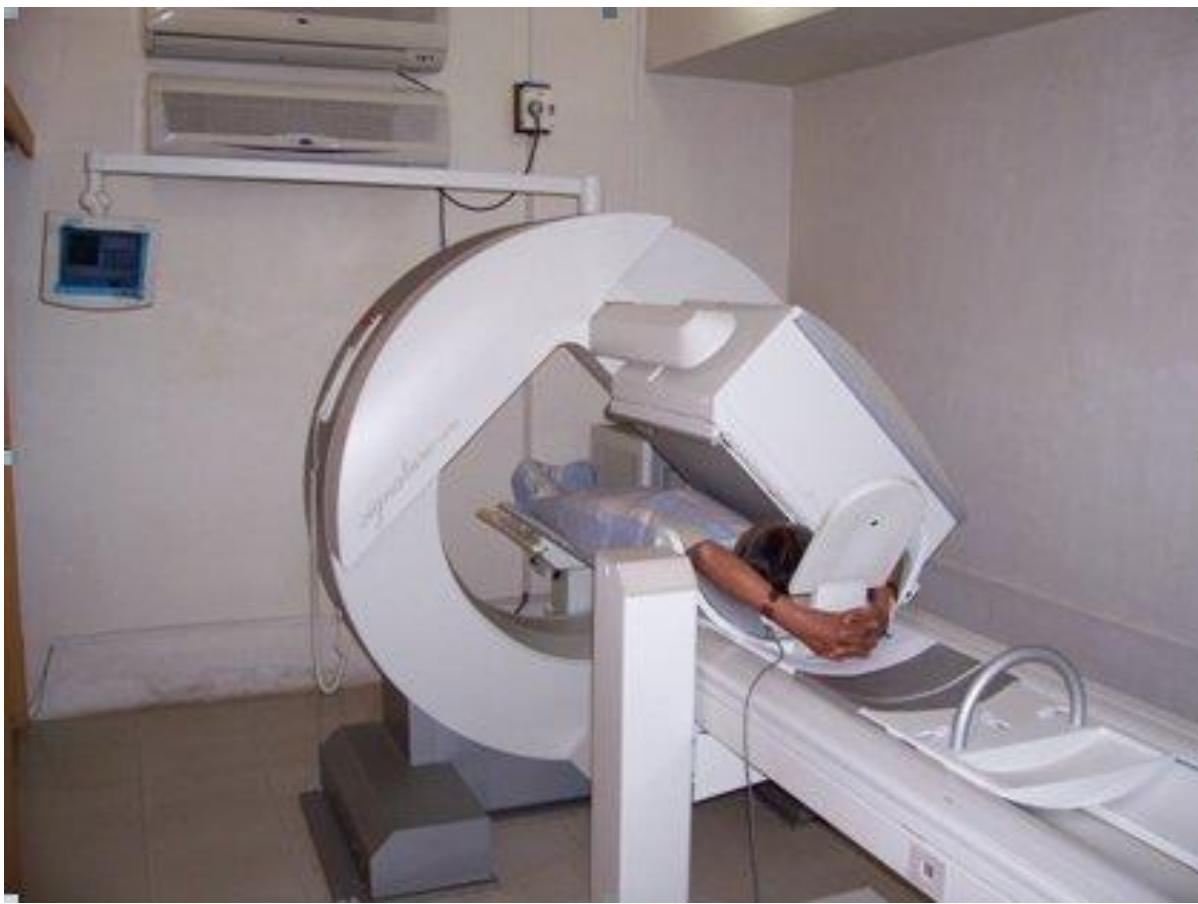
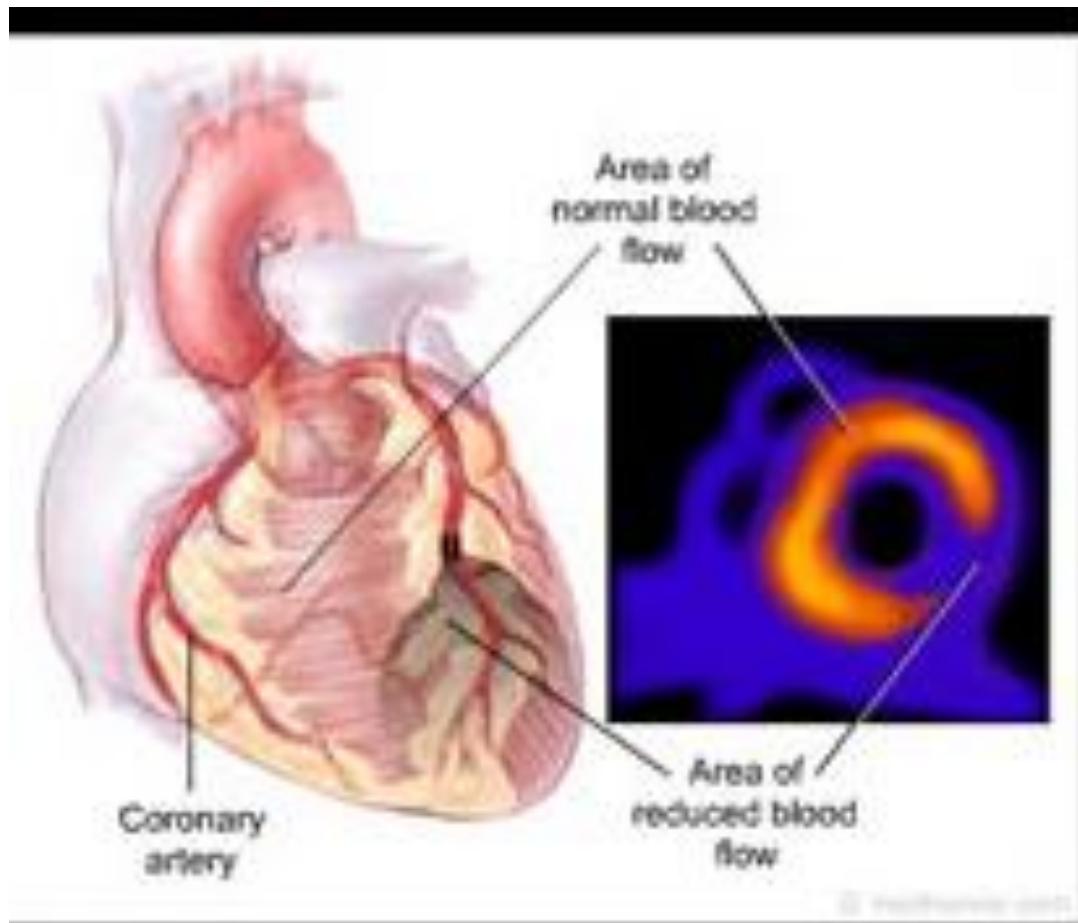
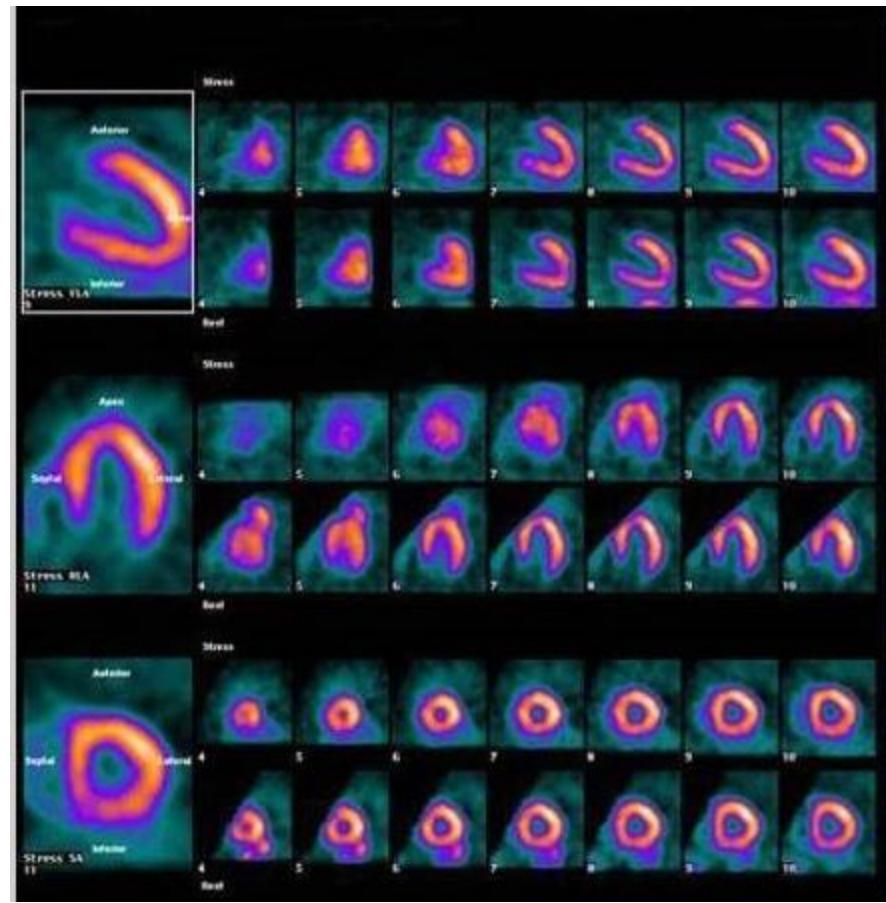


Image: Queensland cardiovascular group









Other Stress tests

- Stress echo vs pharmacological stress echo using Dobutamine (to achieve 85% predicted HR)
- Myocardial perfusion imaging and viability testing:
- MPI (nuclear stress)
- SPECT MPI using radiotracer to detect perfusion defects
- Cardiac PET/MRI

Key point

- Stress testing with imaging indicated in pts with inability to exercise, baseline ECG abn that limit interpretation of exercise ECG or indeterminate findings on exercise ECT

Other tests: visualization of Coronary Anatomy- CT Angiogram (CTA)

- **CTA** short of invasive angiography, visualization of coronary arteries and detect blockages, showing both calcified and noncalcified plaques
- Iodine based contrast dye injected into vein and CT scan imaging
- Effective option of dx CAD in pts with sx <65
- Suspects of acute aortic syndrome, coronary embolism, non STEMI, low TIMI risk score negative troponin

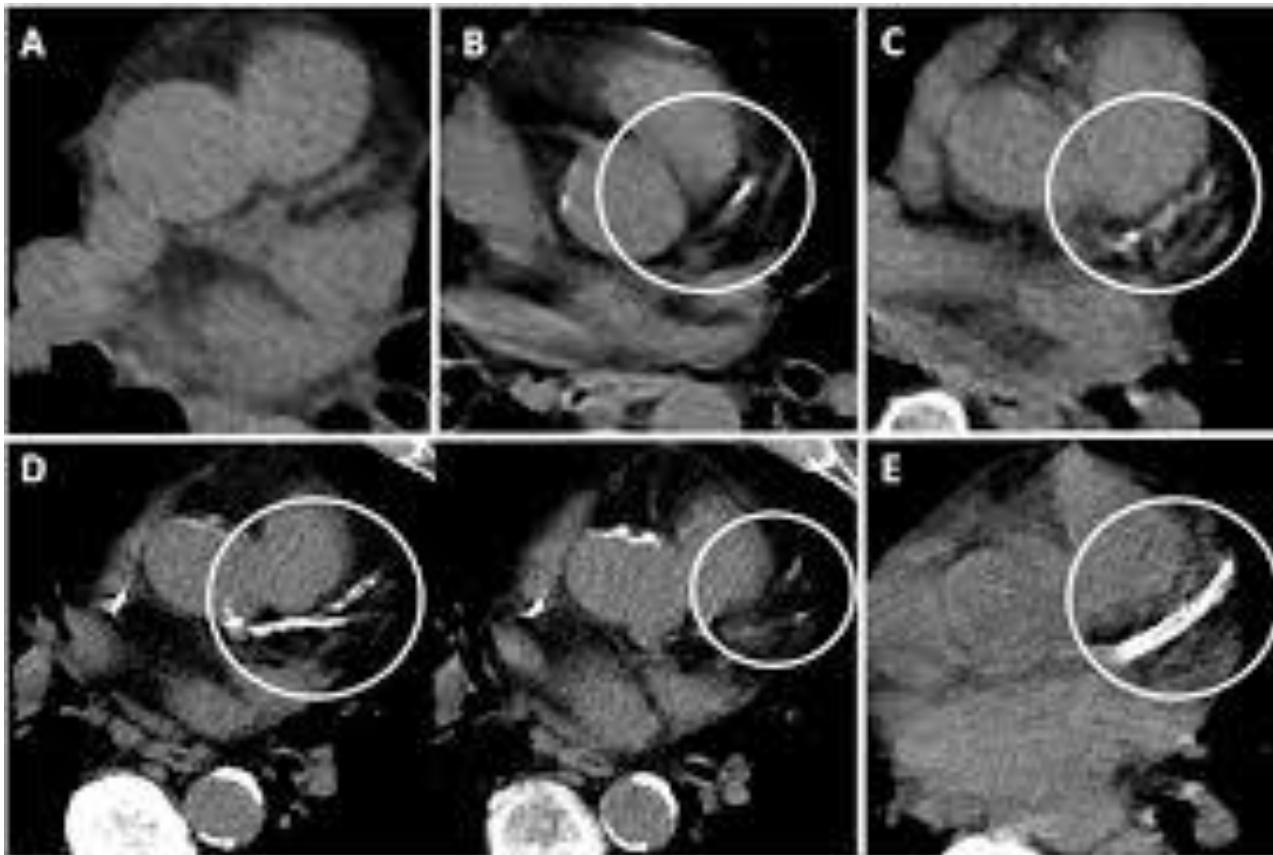
Other tests –Coronary Artery Calcium Scoring **CAC**

- Coronary artery calcium score
- Measures amount of calcium in the arteries
- Provides information on burden of dz
- NOT degree of obstruction
- Predict 10 years cvs risk



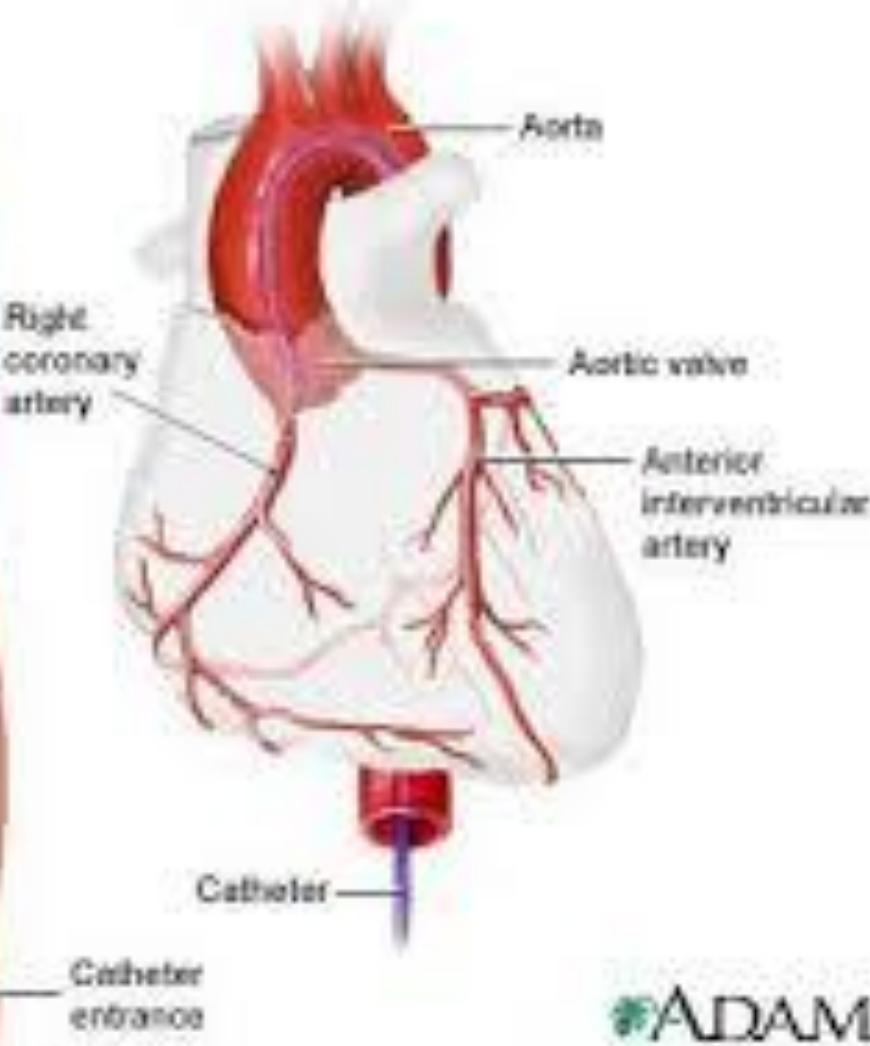
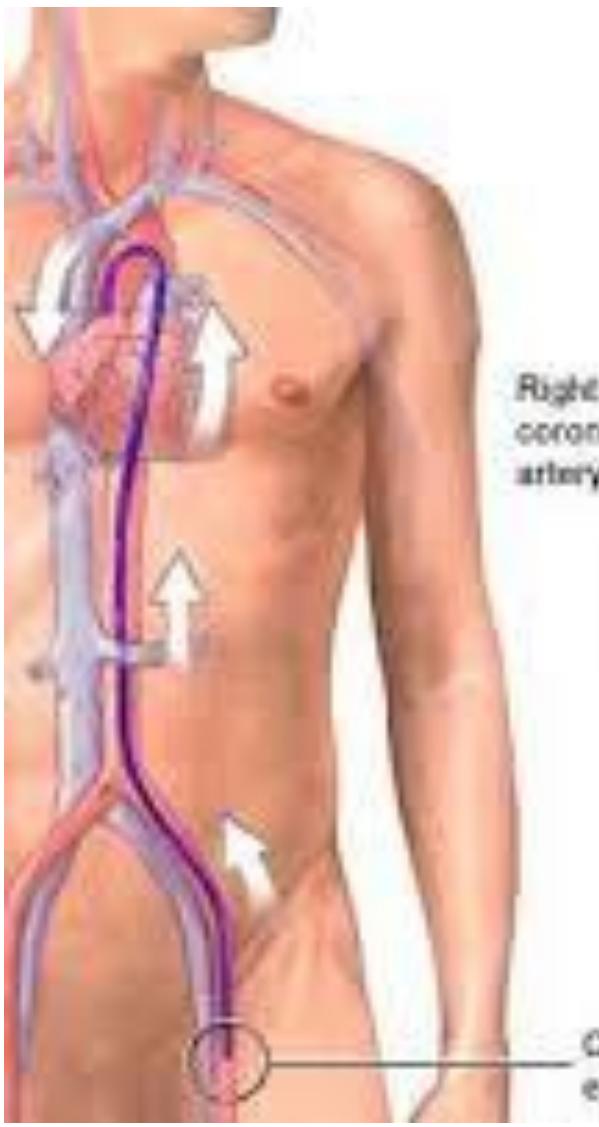
Coronary Artery Calcium Scoring

- 0 no dz
- 1-99 mild dz
- 100-400 mod dz
- >400 severe dz
- The absence of coronary artery calcification is associated with low risk of CVS events



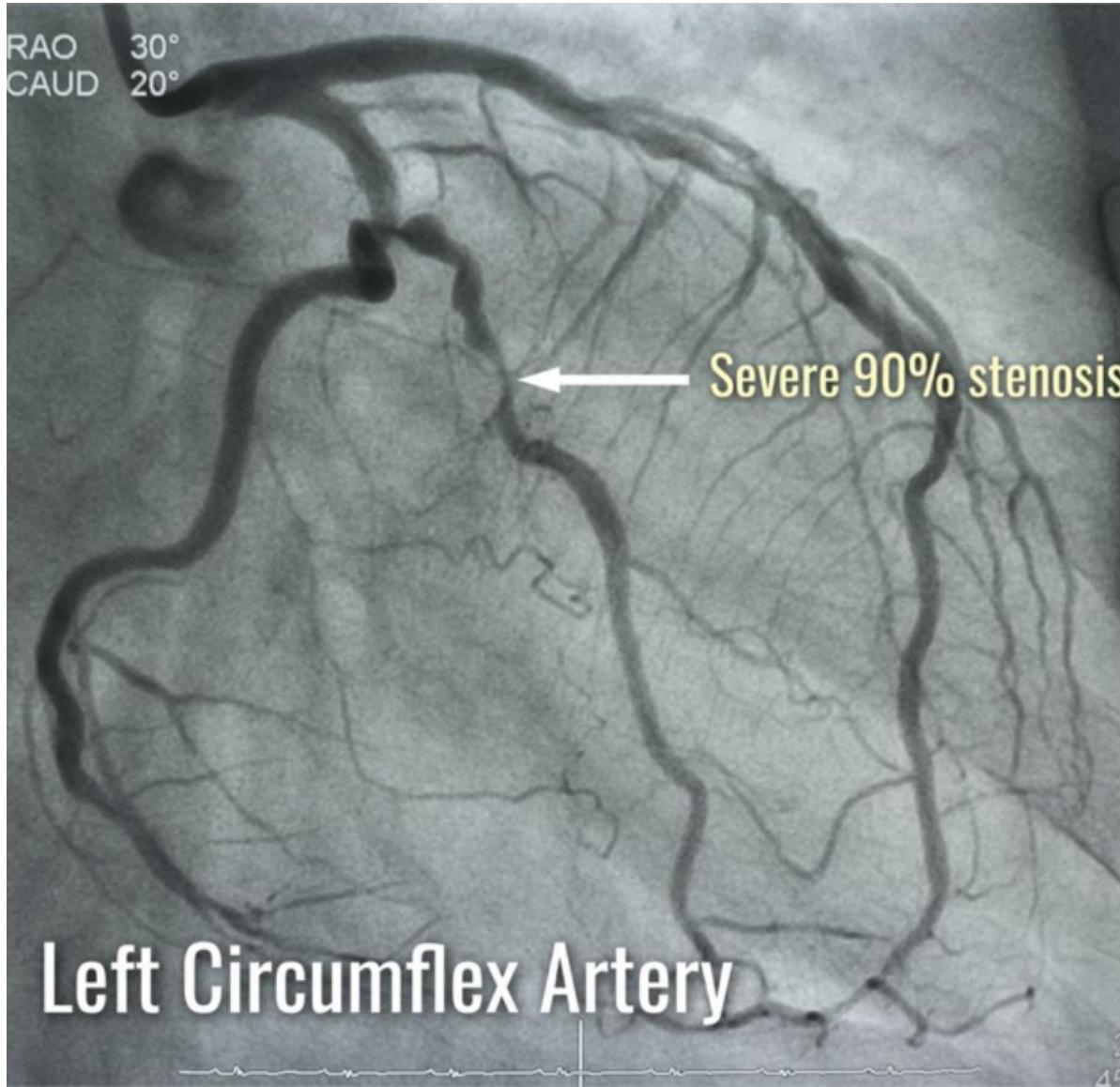
Visualization of coronary anatomy

- **Invasive coronary angiography**
- Provides 2 dimensional images of coronary vessels lumen
- Considered in highly sx pts with abn stress testing, selected pts with ACS and refractory sx to medical therapy



©ADAM

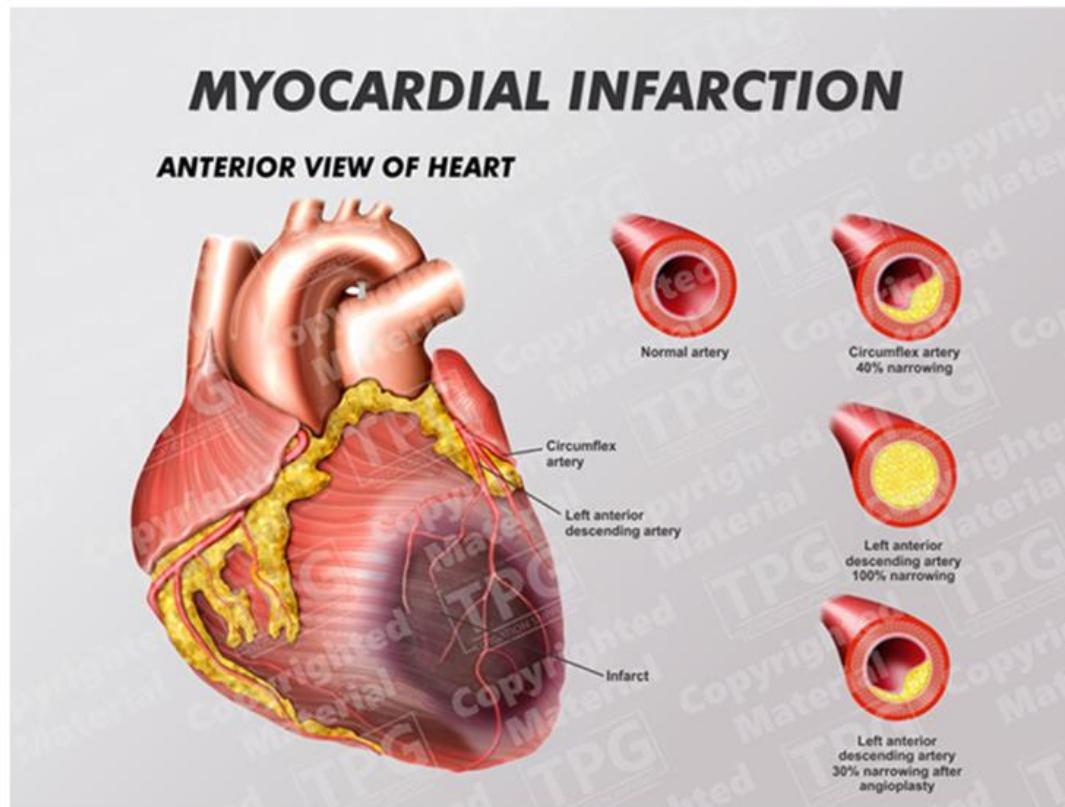
Image: Dr Ooi Yau Wei-sozo cardiology



TT or TE echocardiography

- TTE
- Imaging of structural heart abnormalities
- Evaluate for valvular, congenital heart dz, pericardial dz or ventricular dysfunction
- TEE
- Closer imaging

<https://presentationgroup.com/product/myocardial-infarct/>



Pathophysiology

- Rupture of a vulnerable plaque that leads to platelet activation and aggregation resulting in formation of intracoronary thrombus

Acute Coronary Syndromes 急性冠状动脉综合症

- Unstable angina 不稳定型心绞痛
- Non-ST segment elevation MI (NSTEMI) or NSTE-ACS 非ST段抬高型心肌梗死
- ST segment elevation MI (STEMI) ST段抬高型心肌梗死

Unstable angina

- Defined as either rest angina, new onset exertional angina in the preceding two months or acceleration of preexisting angina in the past two months

STEMI

- ST segment elevation on EKG and elevation of cardiac enzymes
- >80 % totally occluded
- Commonly single vessel
- Thrombolysis beneficial/ PCI performed

STEMI

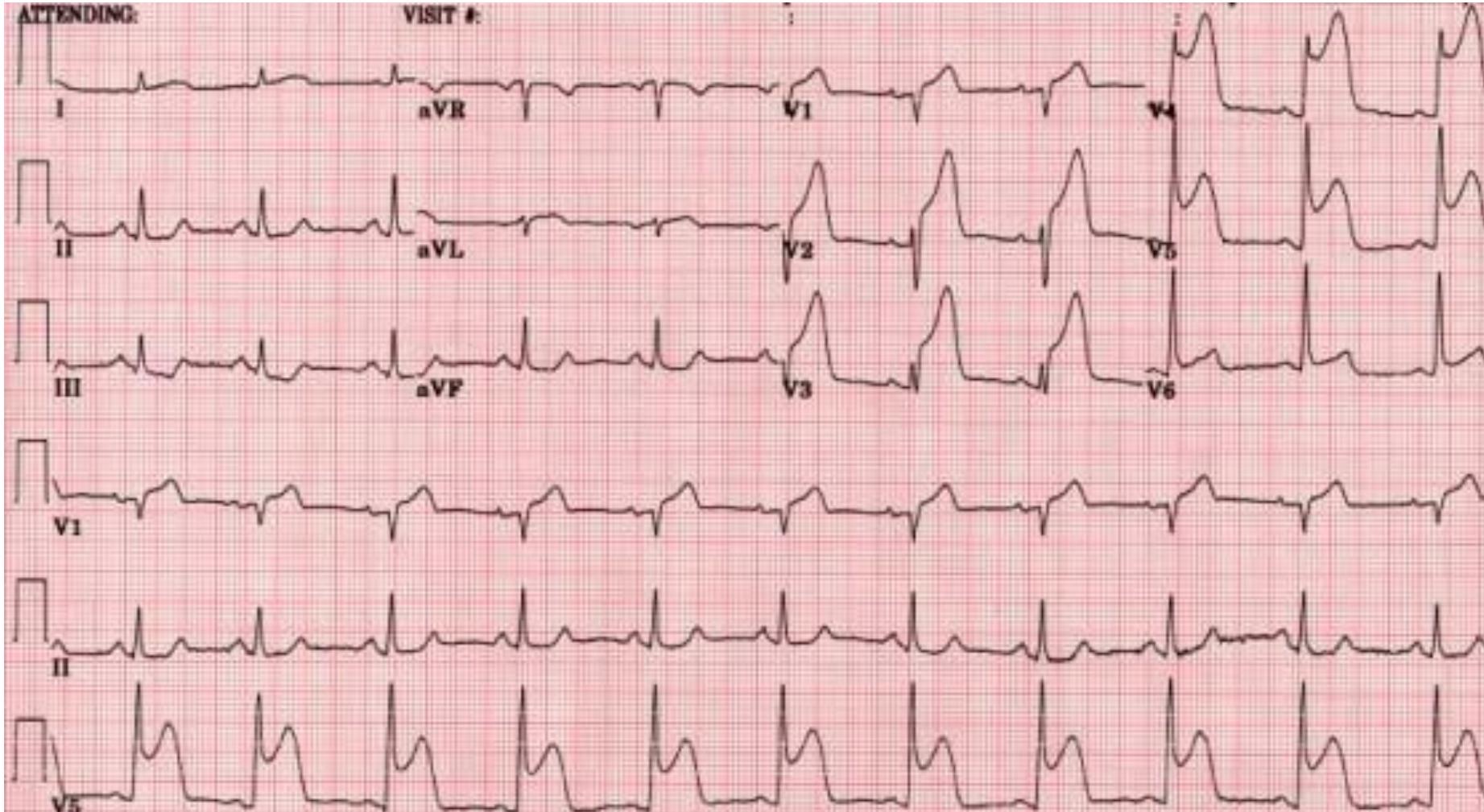
- ST seg elevation at least 1mm in 2 or more limb or chest leads,
- ST elevation in V2 & V3 must be at least 2mm and 1.5 mm in female
- Posterior MI ST depression >2mm in ant lead (V1 -V4) with tall R waves, often with ST elevation in inf or lateral leads
- NEW BBB considered as STEMI

STEMI other ddx presentation

- Acute aortic syndromes with dissection involves left or RCA to transmural myocardial ischemia
- Aortic dissection with differential bp both UE and widened mediastinum, piercing chest pain
- Acute pericarditis knucle sign in aVR

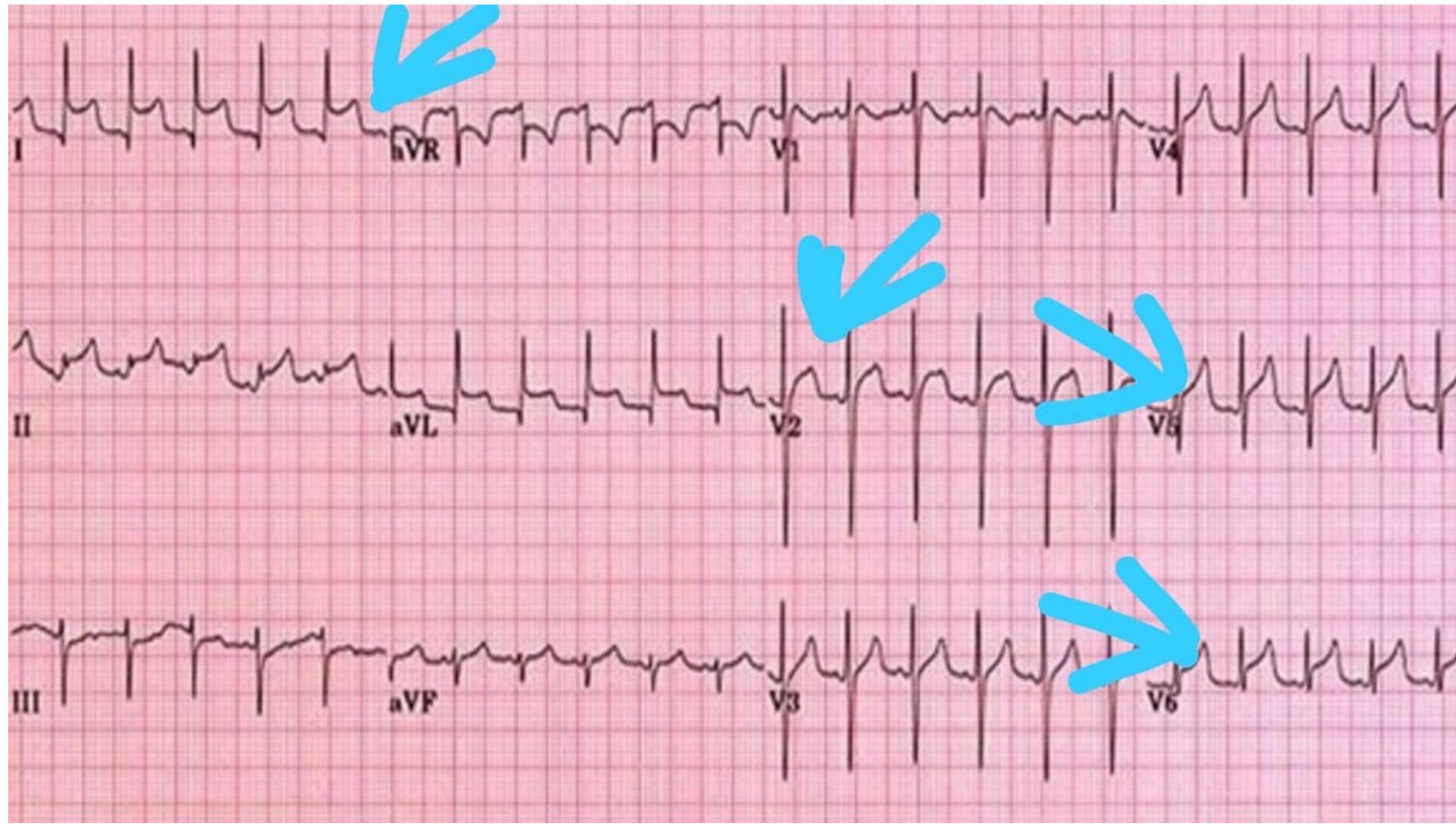
ATTENDING:

VISIT #:



25mm/s 10mm/mV 150Hz 005B 12SL 250 CID: 1

EID:6 EDT: 13:02 14-SEP-2000 ORDER:



Surgical interventions

- PCI- drug eluting stents preferred to bare metal stents to prevent restenosis, MI or acute stent thrombosis
- CABG- as long as internal mammary artery conduits are utilized
- Improved survival with LM or 3x CAD, improved 10 yr survival in pts with LV dysfunction

Treatment of ACS

- Medial management
- Reduce ischemia
 - Nitrates-symptoms relief
 - Beta-blockers-reduce ischemia, infarction size and decrease mortality
 - ACE inhibitors improve survival

Cont.

- Prevent thrombosis
- Aspirin
- Plavix (Clopidogrel) (P2Y12 inhibitors)
- Heparin
- Abciximab (Glycoprotein II-IIIA inhibitors)
- Thrombolysis

PCI (percutaneous coronary intervention)

- **PCI perform in STEMI** and Sx <12 hrs
- **Goal time med contact to PCI is <90 min**
- PCI preferred over thrombolysis for STEMI
- CABG >PCI reduction in cvs death and MI in pts with multi vessels dz
- PCI can be staged or nonstaged if hemodynamically stable for nonculprit lesions (remote area from infarction)

Thrombolytic therapy

- TT indicated for STEMI when sx onset <12 hrs and PCI not available within 120 min of first med contact
- Or sx 12 to 24 hrs and hemo unstable or sig myocardium at risk and PCI not avail.
- **Most effective within 3-6 hrs from sx onset**
- Thrombolytic agents: Alteplase, Reteplase, Tenecteplase
 >>Streptokinase

Key points

- PCI >>>TT for STEMI
- If PCI not available within 120 min from first med contact, pts should receive TT then to PCI center

Complications of STEMI

- A fib 20%
- VT, VF during post MI which warrant ICD
- Accelerated idioventricular rhythm after reperfusion usually benign
- AV blocks or complete HR
- Papillary muscle rupture
- Ventricular septal or wall rupture
- RV infarct
- Pericarditis

Complication of STEMI

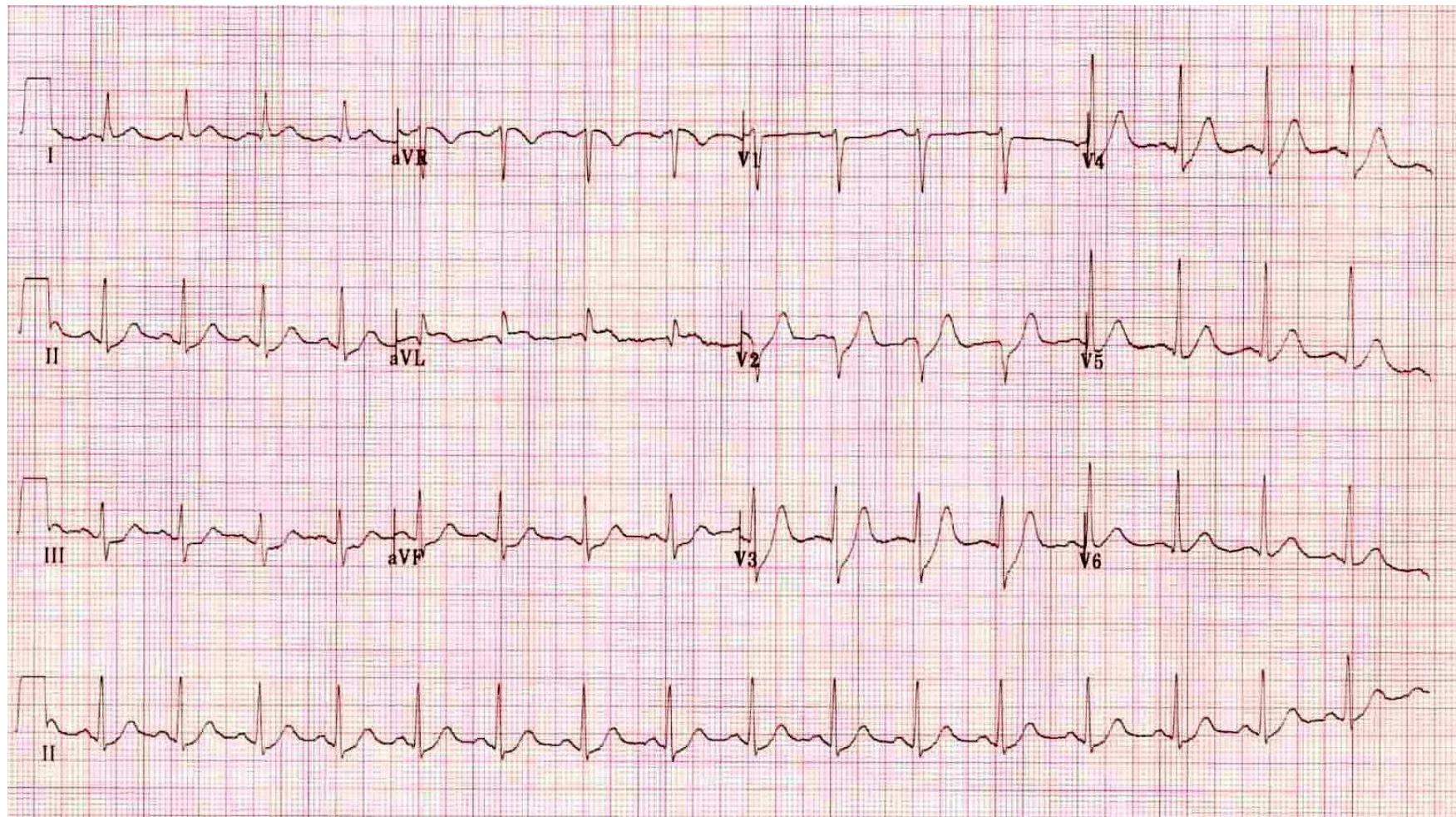
- Cardiogenic shock from large anterior MI
- Use of IABP or LV assist device or ECMO
- Left apical thrombus 10-20% in anterior STEMI
- LV aneurysm without associated thrombus usually not treated with anticoagulation unless other indications such as a fib
- Anticoagulation for min 3 months
- RV infarction, acquired VSD
- Acute MR 7 days after STEMI due to posteromedial papillary muscle damage from post descending artery during MI

NSTEMI

- Similar clinically to Unstable angina
- Distinguished by evidence of myocardial necrosis (positive cardiac enzymes)
- NO ST elevation in EKG
- ST depression
- Thrombolysis not recommended
- Usually, no PCI required immediately

EKG

- STEMI- at least 1 mm ST segment elevation in 2 contiguous leads or presence of a new LBBB
- NSTEMI- ST depression, inversions or normal



Differential diagnosis

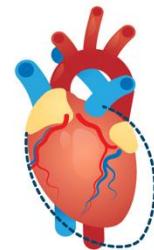
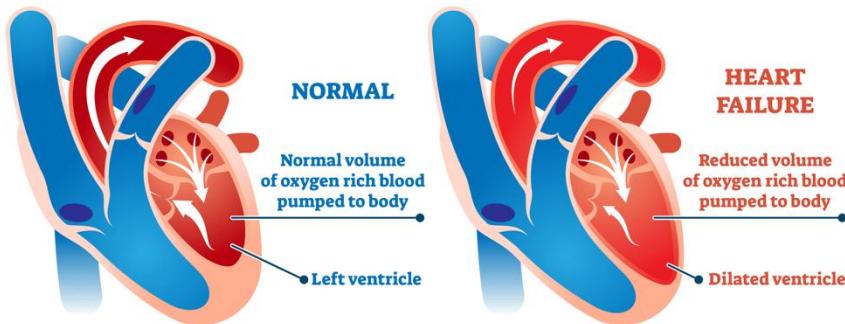
- Aortic dissection
- Acute pericarditis
- Pulmonary embolism

CONGESTIVE HEART FAILURE

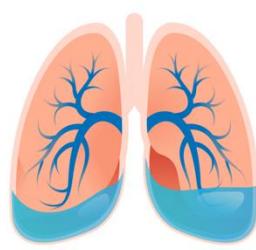
- Heart or circulation is unable to meet the metabolic demands of peripheral tissue at normal cardiac filling pressures
- Clinical signs and symptoms of congestion, fluid retension, effort intolerance, inadequate organ perfusion and arrhythmias

<https://www.respiratorytherapyzone.com/>

Congestive Heart Failure



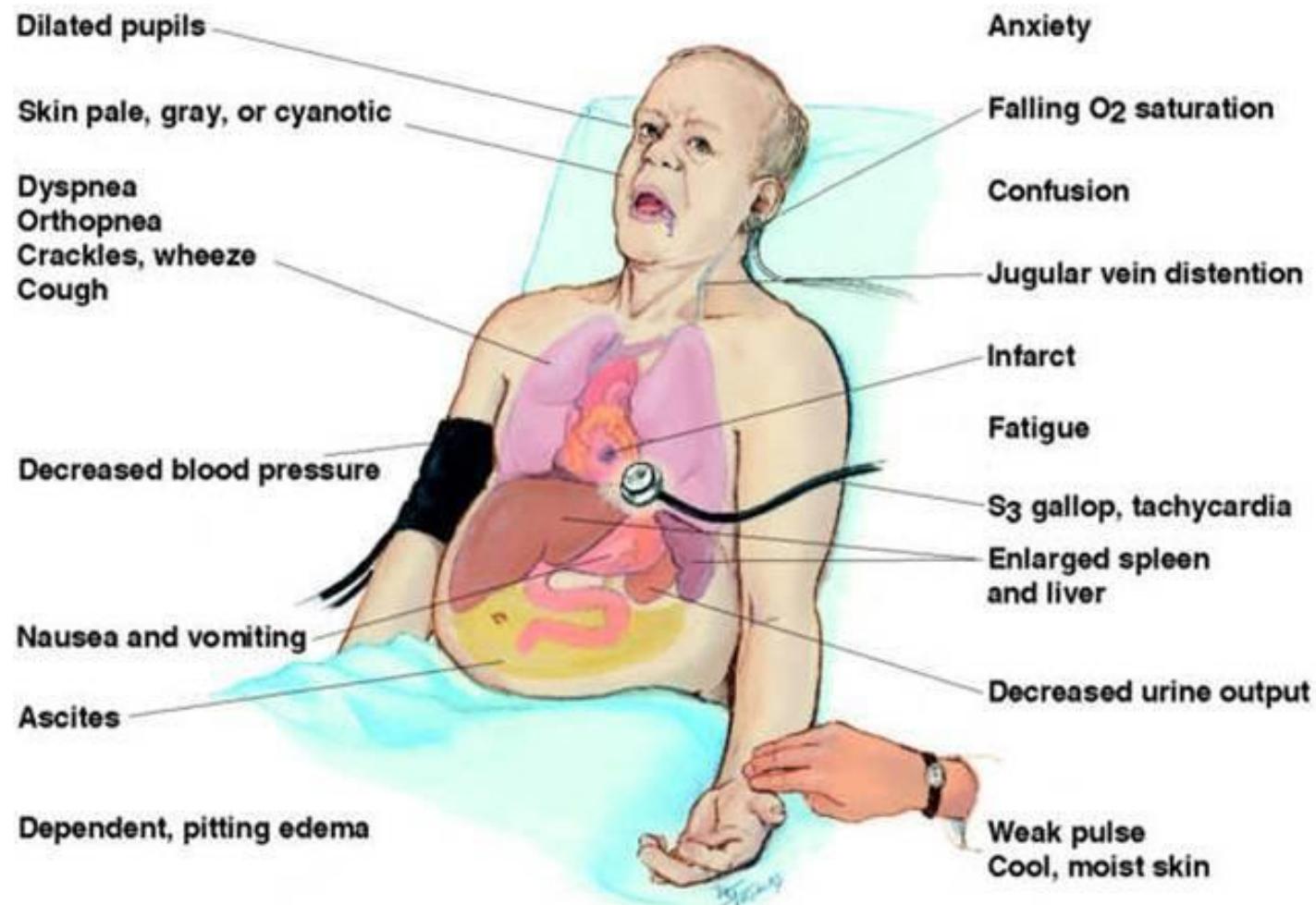
Enlarged heart
Chest congestion



Excess fluid around lungs
Shortness of breath



Swelling in legs and feet
Edema



Wikipedia



Medicove

Pitting Edema



Congestive Heart Failure

- Most common cause is CAD and MI causing systolic dysfunction
- Elderly, 50% may cause by diastolic dysfunction
- 24-28 % patients die within 1 year
- 45 to 59% die within 5 years

Systolic Heart Failure

- LV EF reduced= **HFrEF** defined <40%
- In HFrEF, RAAS system activated>>production of Angioensin II and aldosterone
- >>vasoconstriction, increase BP
- Aldosterone increase fluid retention, Na resorption.
- Sym system release E &NE , initially maintains CO and become maladaptive

Diastolic Heart Failure

- Stiffened LV with abnormal relaxation during diastole>> increase LV preload but with preserved EF
- **HFpEF** defined by LVEF >50% Vs
- **HFmEF** defined by LVEF 40-50% in 25% of all HF patients (most common cause is HTN)

Systolic dysfn

- Impaired ejection
- 70% of HF patients
- Causes: **CAD**
- Valvular HD
- MI
- Idio. CM
- Systemic Dz

Dyastolic dysfn

- impaired filling
- 30% of HF
- HTN
- Ischemia
- aging
- restrictive CM
- pericardial Dz

Leading cause of HF

- **CAD leading cause of HF >50%**
- Should be considered in all pts with newly dx CHF

Early and accurate detection of heart failure

- Clinical suspicion
- Congestion: edema, orthopnea, PND are late phenomena
- Many patients presents with unexplained fatigue, abdominal discomfort, wheezing, cough and insomnia
- Clinical suspicion in patient at risk
- Patients with leg edema

Imaging

- EKG
- Chest X-ray
- Echocardiogram

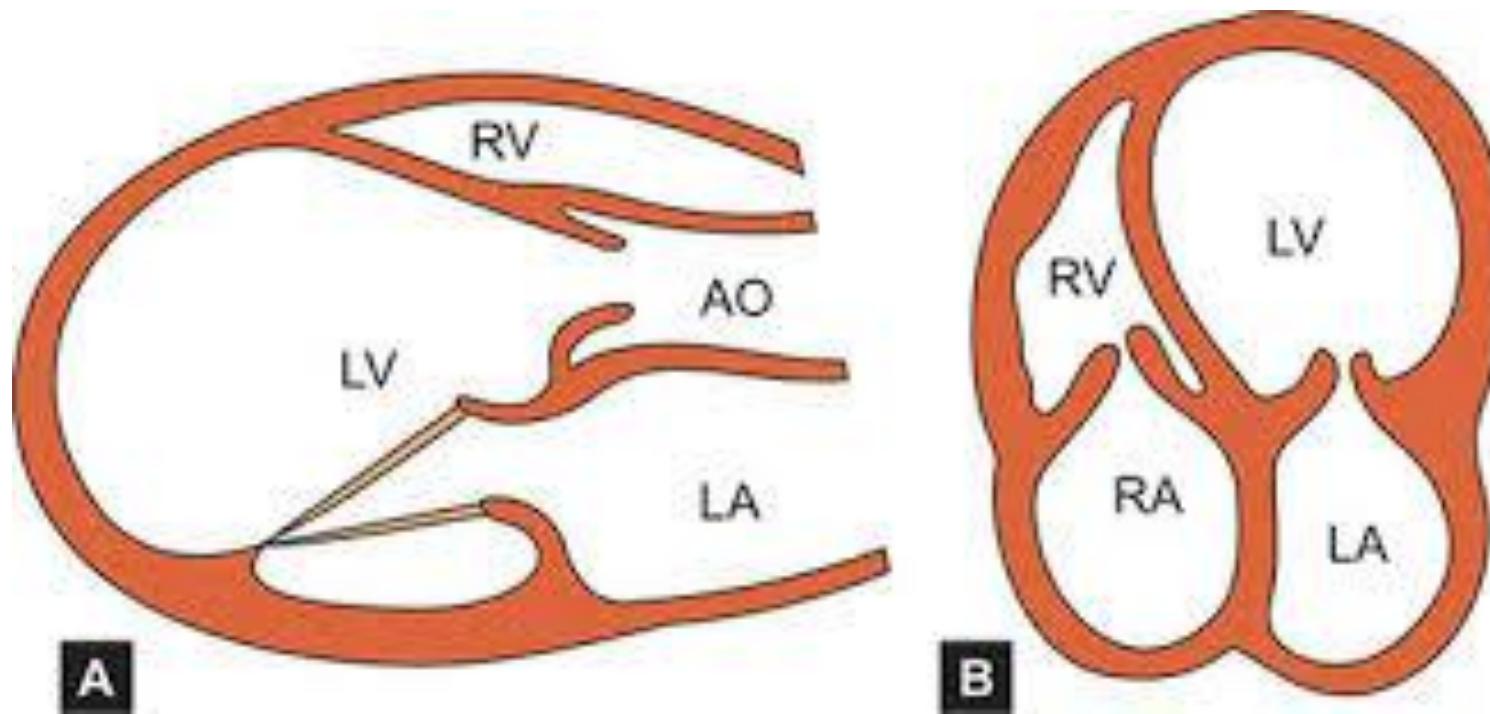
Imaging studies

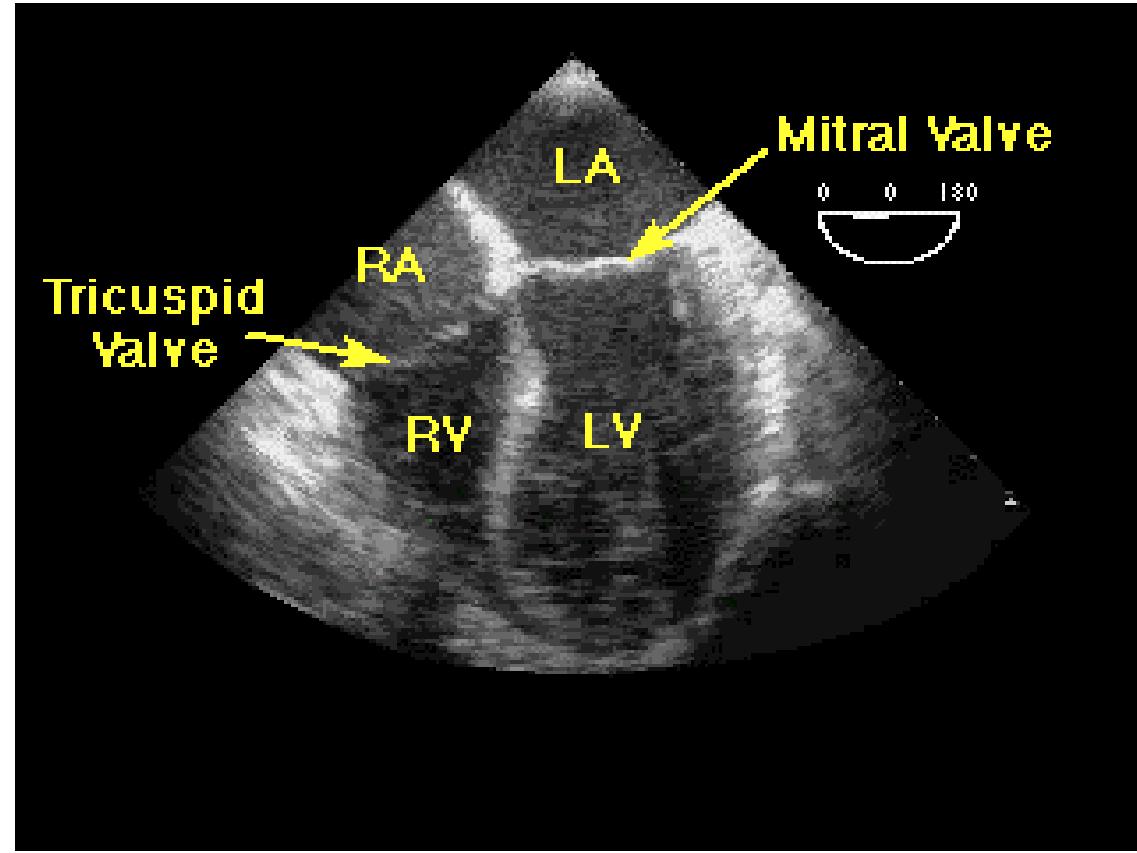
- Echocardiogram, assess cardiac structure and function in patients with evidence of heart failure
- Look for Ejection fraction, dilatation of chambers, stiffness of chambers, valvular abnormalities

https://www.teachingmedicine.com/tutorial/CHF/Fluid_fissures



Image: JaypeeDigital eBook Reader





Natriuretic peptides-biomarkers for CHF

- The white blood cell of HF
- B-type Natriuretic peptide (BNP) or NT pro BNP, a 32 amino acid peptide that is primarily secreted from the ventricular walls in response to myocyte stretch or damage
- BNP shorter ½ life Vs NT pro BNP longer ½ life (probably more sensitive detecting earlier forms of HF)
- Also increase in worsening kidney failure, sepsis, obesity use of ARNi which blocks breakdown of BNP and therefore elevates the BNP

BNP or NTproBNP

- BNP increases in HF (typically >400pg/ml)
- Low to normal in pt with pulm dz (<100)
- Sensitivity 95% in HF
- Not sensitive or specific to exclude HF if between 100-400
- NT pro BNP :
 - <age 75, normal <125 pg/ml
 - >age 75, normal <450 pg/ml
 - >900pg/ml, indicative of HF
 - Echo is primary modality to evaluate HF

Utilizing BNP biomarker for CHF

- Initial Diagnosis
- In hospital management and in acute decompensation
- Office monitoring during chronic phase to prevent rehospitalization

NYHA functional classification

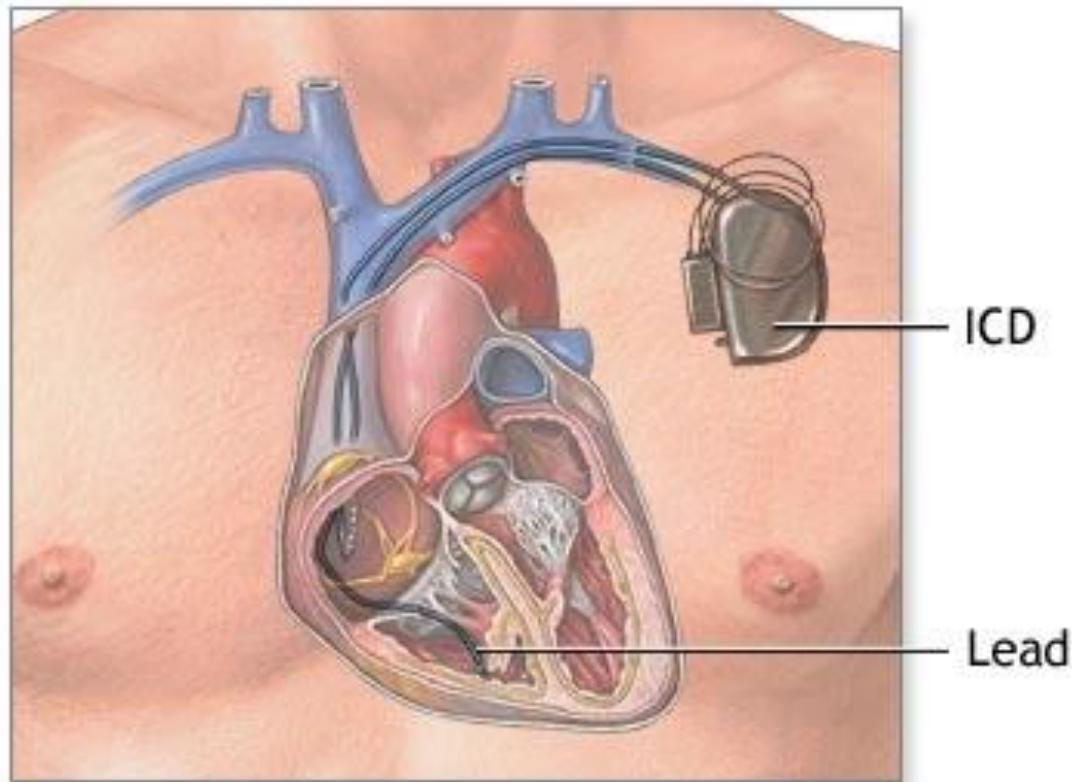
- I no limitation in activity
- II slight limitation
- III marked limitation
- IIIA Sx with less than ordinary activity
- IIIB Sx with minimal exertion
- IV Unable to carry on activity wo sx

Treatment of CHF

- Treatment is prevention of HF
- Tight control of BP, BS, and lipids
- Weight reduction, BMI < 30
- Physical activity, 20-30 min, 3-5 X/wk
- Smoking cessation
- Limit ETOH
- Limit Na intake
- Medications

Implantable cardioverter-defibrillator therapy

- ICD rec in pts receiving guideline directed therapy with LVEF <35% (II-III)
- Wearable cardioverter defibrillator no guidelines but option for bridge into ICD therapy



An implantable cardioverter-defibrillator (ICD)
detects a rapid heartbeat coming from the
bottom of the heart

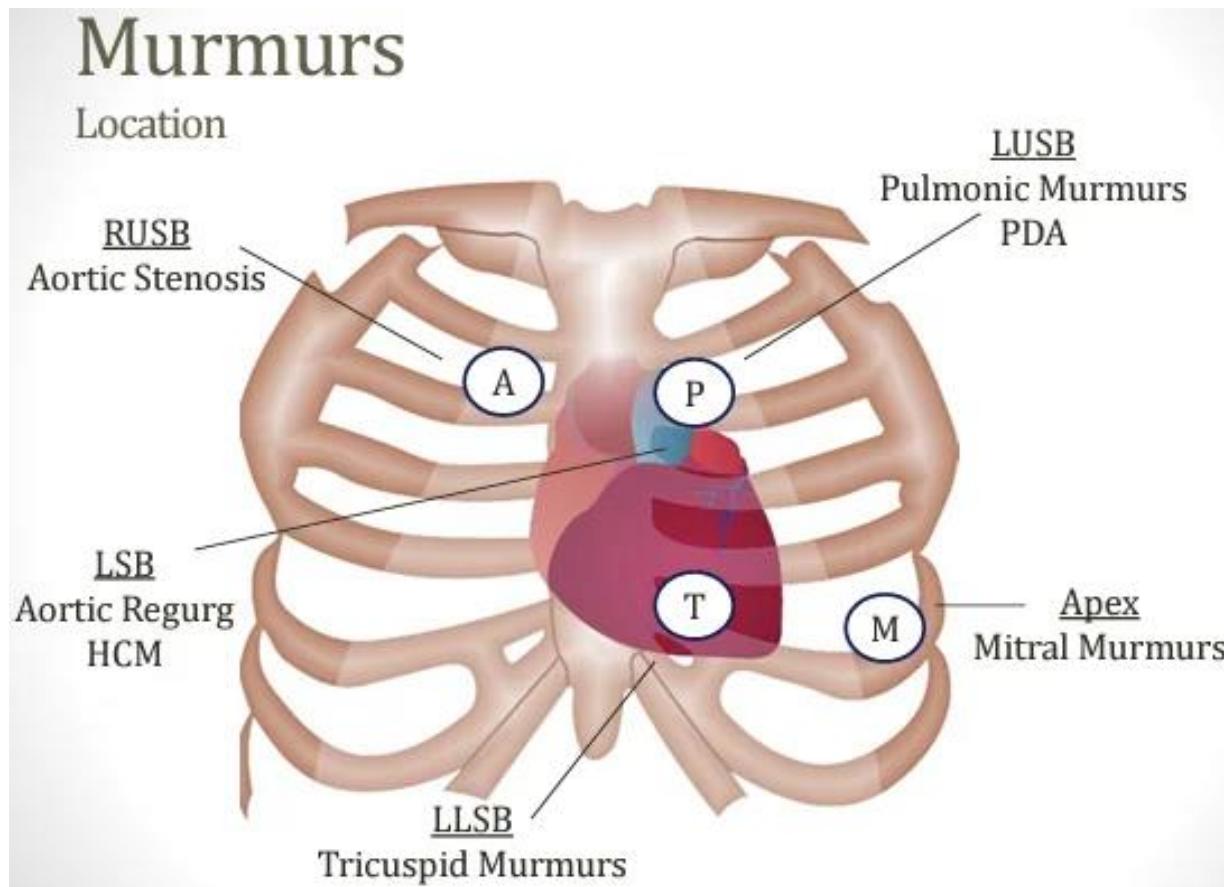
Stethoscope



Valvular heart disease

- Etiology is related to location of murmur
- Rheumatic fever was formerly the leading cause of mitral and aortic dz
- Now, are a result of degeneration, ischemia, calcification

Reddit



Heart murmurs for beginners 🔥

Part 1:Aortic & Mitral

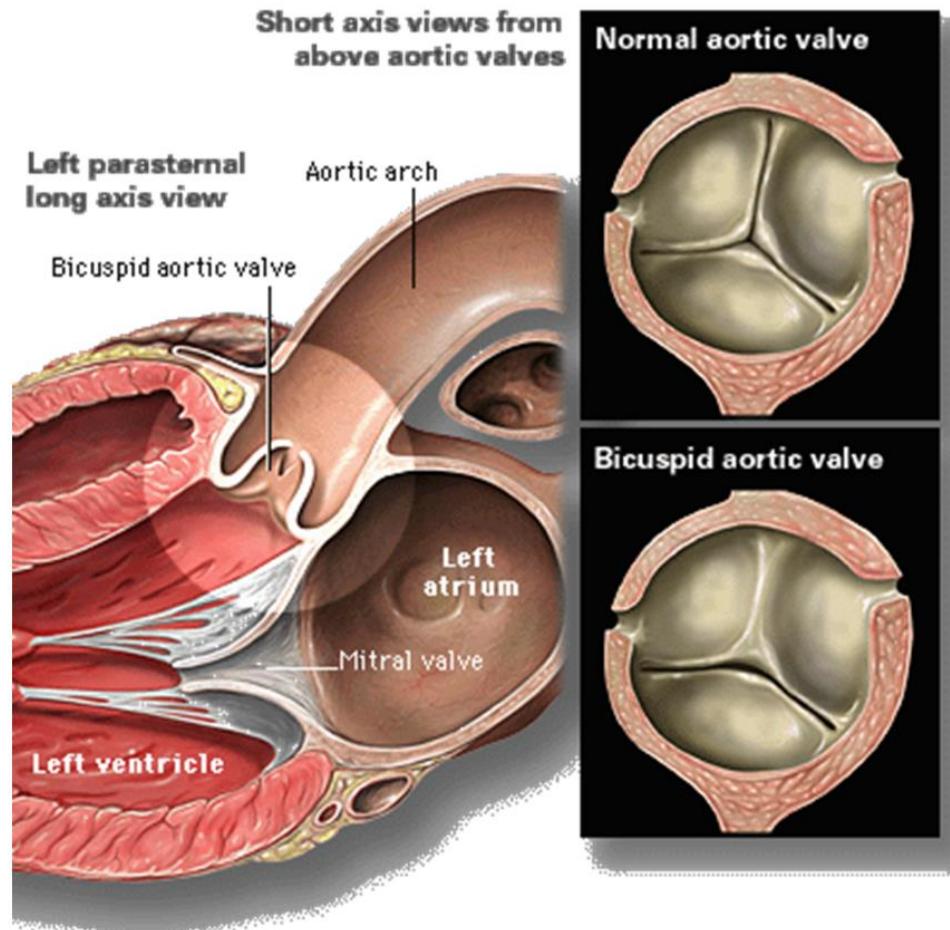
stenosis, Aortic & mitral

regurgitation.

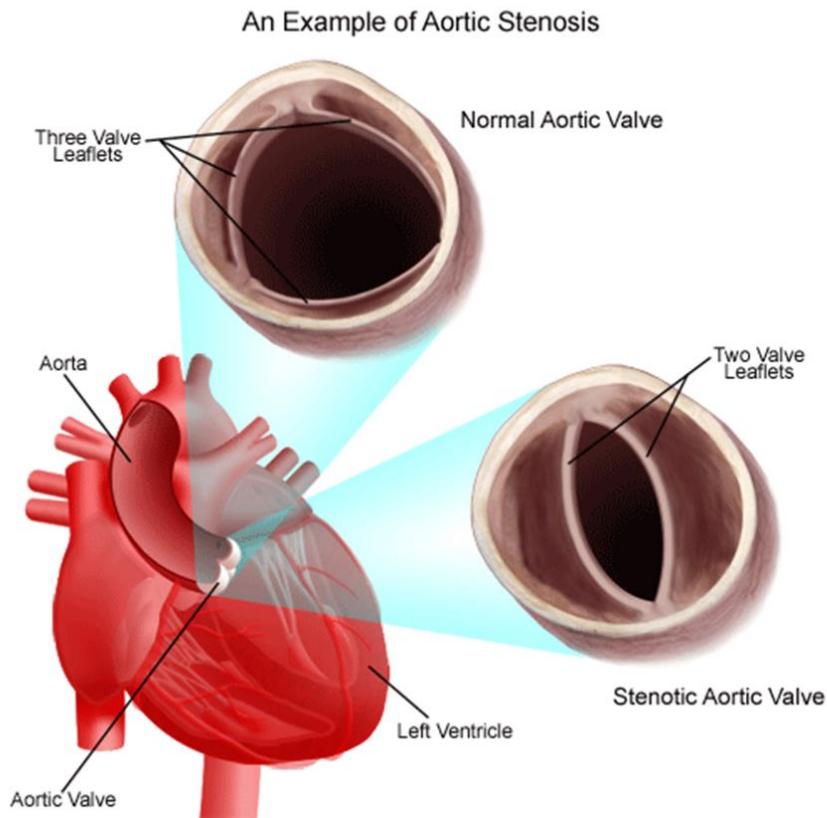
Aortic stenosis

- Degenerative-Stiffening, scarring and calcification of AV leaflets
- Rheumatic- commissures are fused
- Congenital- Unicuspid valve
- Bicuspid valve
 -

Healio-Bicuspid Aortic Valve



Cardiac Health- aortic stenosis



Clinical presentation

- ▶ Usually asymptomatic
- ▶ **Angina** (shortened diastolic perfusion time in combination of LV hypertrophy and relaxation)
- ▶ **Syncope/dizziness** (arrhythmia, decreased cerebral perfusion)
- ▶ **Heart failure** (harder for blood to push thru, heart enlarged)

Diagnostic tests of AS

- Echocardiogram:
- Severe AS mean gradient >40 mm and AVA <1 cm
- May underestimate AS severity
- Catheterization needed to measure CO and pressure gradient
- Surveillance q 6-12 mo in severe cases

Association of AS with CAD

- CAD present in 54% in AS
- New coronary events in 41% in nl patient
 - 62% in mild AS
 - 80% in mod AS
 - 93% in severe AS

Sym. PAD present in 42% AS and 25% W/O

Physical Exam

- Carotid pulses diminished and delayed upstroke (pulsus tardus) may be acc by delayed pulse amplitude pulses parvus (low CO)
- Diminished S2 (decreased mobility of Aortic valve)
- Systolic murmur radiate to carotids and apex
- Intensity of murmur not correlate with disease activity

Medical management

- Prophylactic antibiotics regardless of severity
- Patients with Sx and moderate/severe AS should undergo AVR promptly
- Diuretics should be used in caution to prevent decrease C.O. and hypotension
- Vasodilators should be avoided

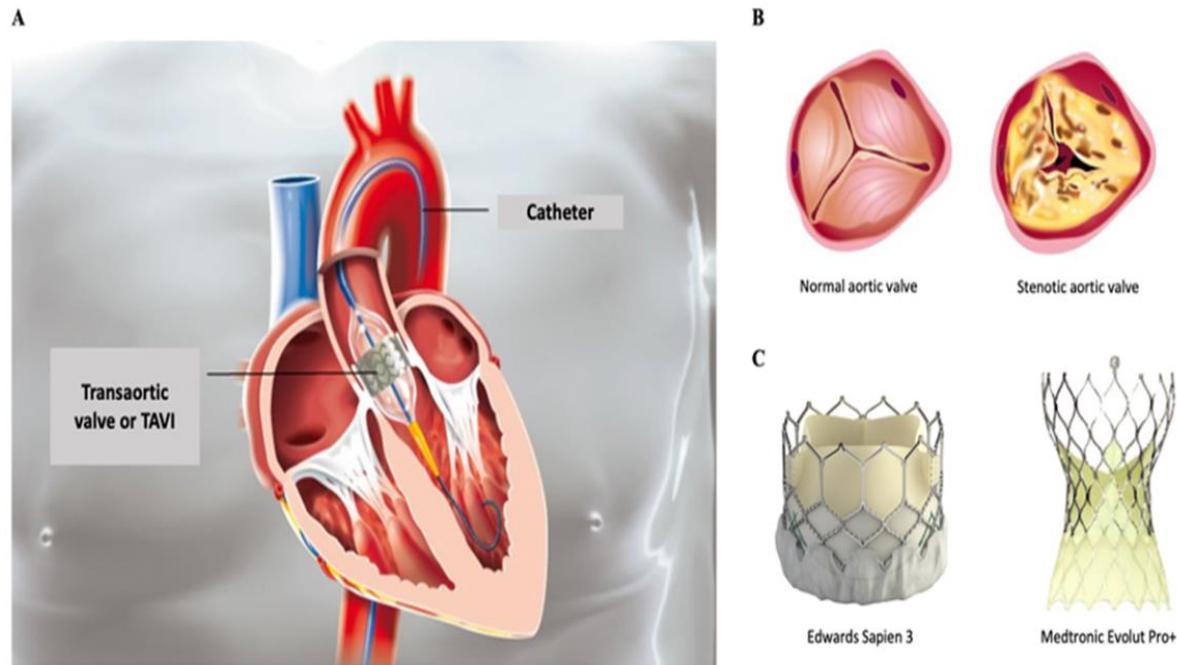
AV replacement indication

- Presence of sx (dyspnea, angina or HF)
- LV dysfunction (EF<50%) or asx pts with concomitant cardiac surgery
- Asx pts with very severe AS and low risk

AV replacement

- SAVR (Open cardiac surgical AV replacement) VS
- TAVI (Transcatheter AV Implantation)
- TAVI for pts >80 or younger with life expectancy <10 yrs or pts with high surgical risk
- Balloon valvuloplasty may be used for bridging
- Pts with trileaflet AS

CCIB centre de cardiologie interventionnelle bellaedonne



AORTIC REGURGITATION

Clinical presentation

- Ax. until 50s
- Heart failure

Physical exam

- Wide pulse pressure
- Diastolic decrescendo murmur
- LLSB, best with patient lean forward and holding breath at end expiration (valve pathology) vs
- RLSB (root pathology dilated aorta)
- Austin flint murmur: late diastolic rumble at apex (mimic Mitral stenosis)

Diagnostic test of AR

- TTE imaging followed by TEE if suboptimal
- CT angio in acute AR with dissection (surgical emergency)
- CMR or Cath
- Severe AR criteria: jet width occupies 65% of LV outflow tract or regurg vol >60ml or orifice >0.3cm

Management of AR

- Dihydropyridine CCB, ACE, ARB with chronic AR and HTN
- BB reasonable for LV dysfunction
- Counseling on warning symptoms
- Clinical and echo follow up
- Surgery with AV replacement in Sx or LVEF <55%
- NOT TAVI

Mitral stenosis

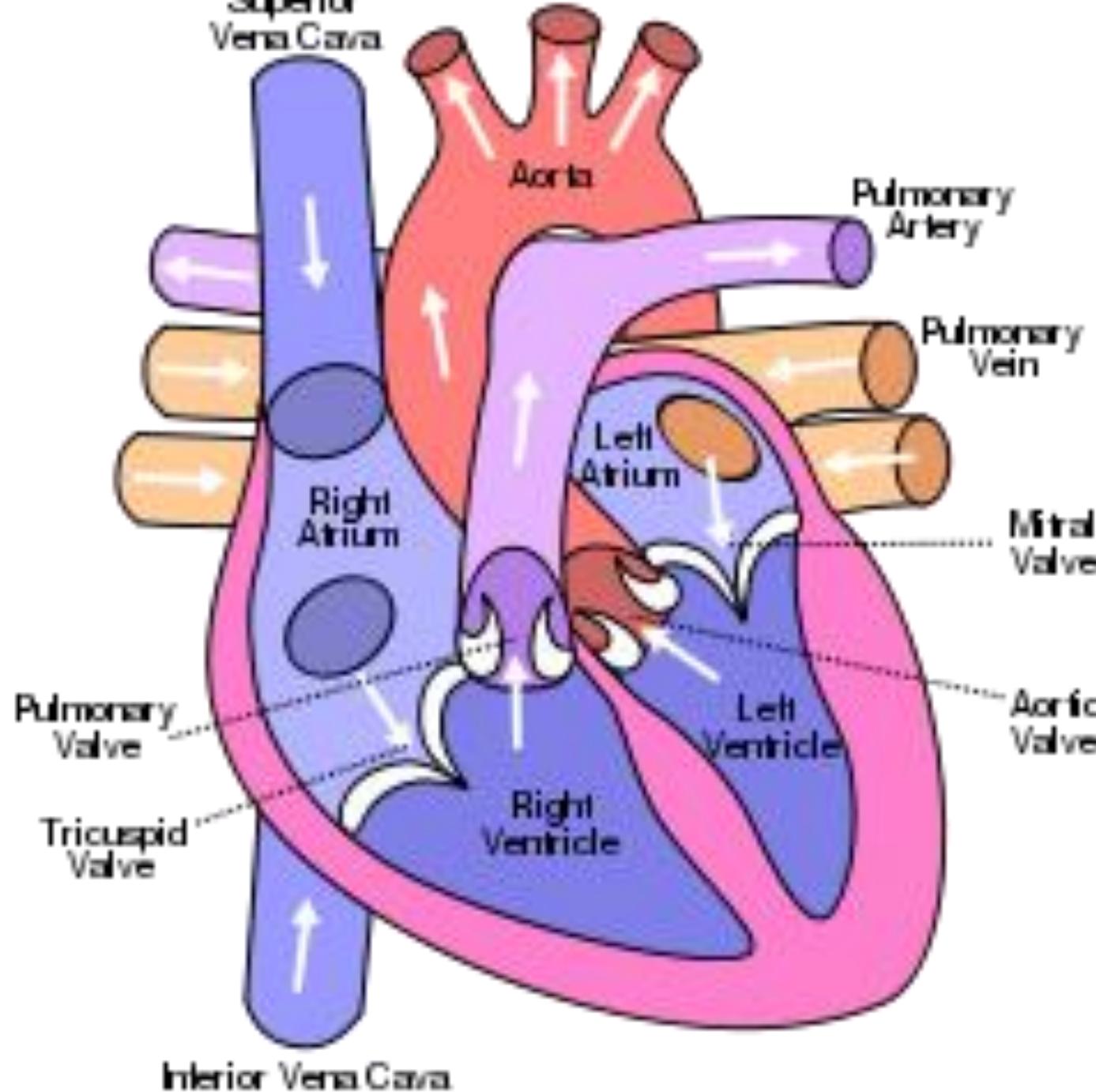
- Rheumatic heart disease is the most common cause, women >>men 4:1
- Thickening and calcification of the valve leaflets and fusion of the cusps, commissures and chordae

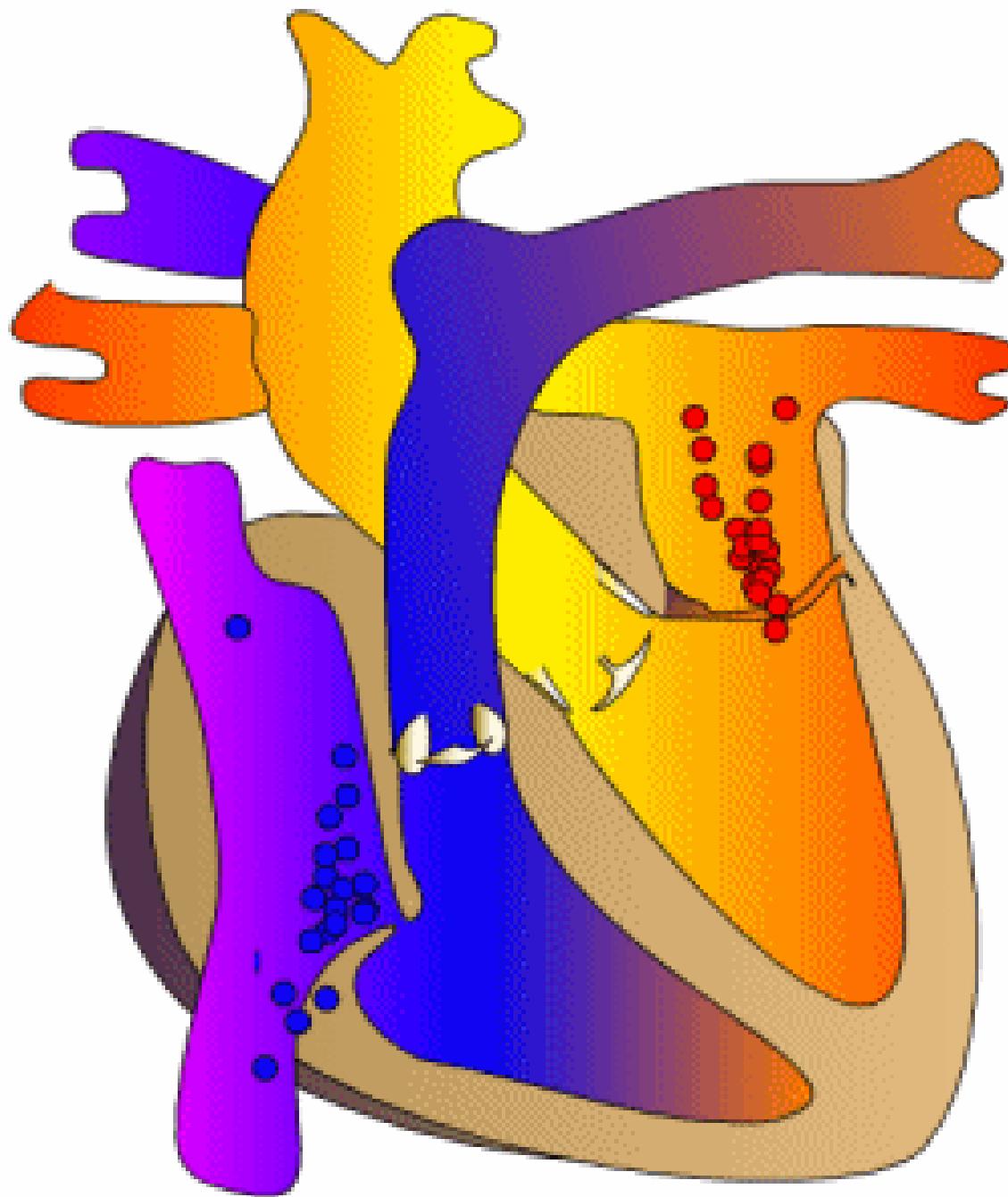
End results

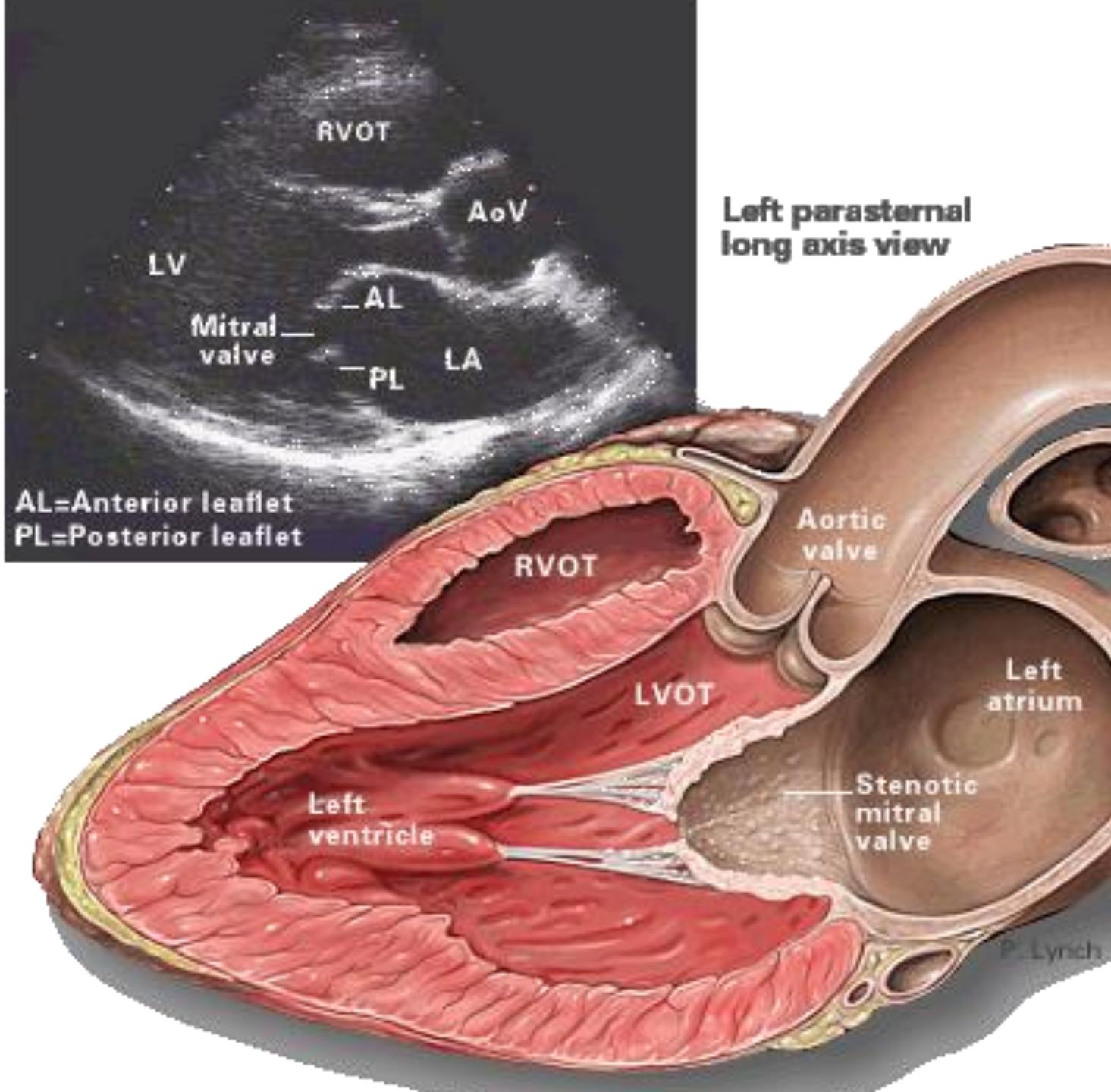
Atrial fibrillation

CHF

Right sided heart failure







Clinical presentation

- Predominance of women
- DOE
- Fatigue
- Pulmonary edema
- Right vent. HF
- Atrial fibrillation: pressure overload leads to marked structural and electrical remodeling of LA
- >CVA (50% pts have AF and risk of thromboembolism 25% if not anticoagulated)
- Hemoptysis

Physical exam

- Low pitched diastolic murmur
- Low pitched diastolic murmur
- Low pitched diastolic murmur
- Loud S1 and prom. P2
- Opening snap of MV

Acute and Chronic Mitral regurgitation

- Abnormalities of valve leaflets or annulus, chordae, papillary or LV free walls acute or chronic
- Papillary muscle dysfunction from MI
- Rheumatic heart disease
- Mitral valve prolapse
- Infective endocarditis
- LV dilatation and calcification

Acute MR

MR

- Endocarditis
- Papillary muscle ischemia or rupture from MI
- Chordae rupture
- Flail leaflet

Chronic

- Mitral valve proplase (2% US pop)
- Mysomatous or degenerative mv
- Xrt
- Rheumatic dz

Clinical presentation

- Longer time to present (> 20 years)
- Compensatory phase is Asymptomatic
- CHF, fatigue, dyspnea, orthopnea
- Irreversible LV dysfunction may have developed

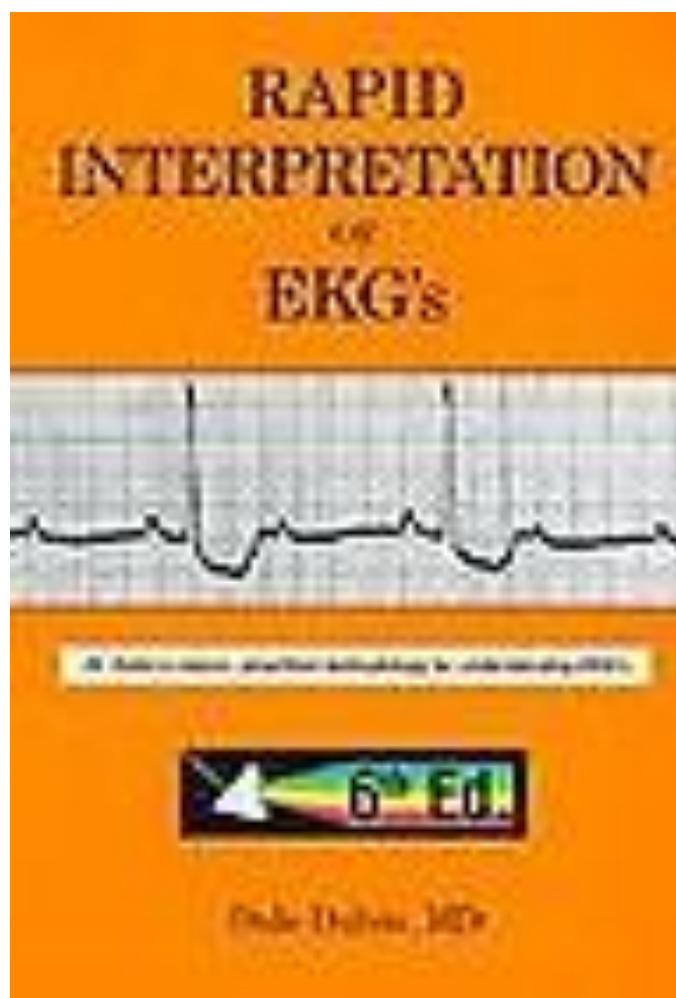
Physical exam

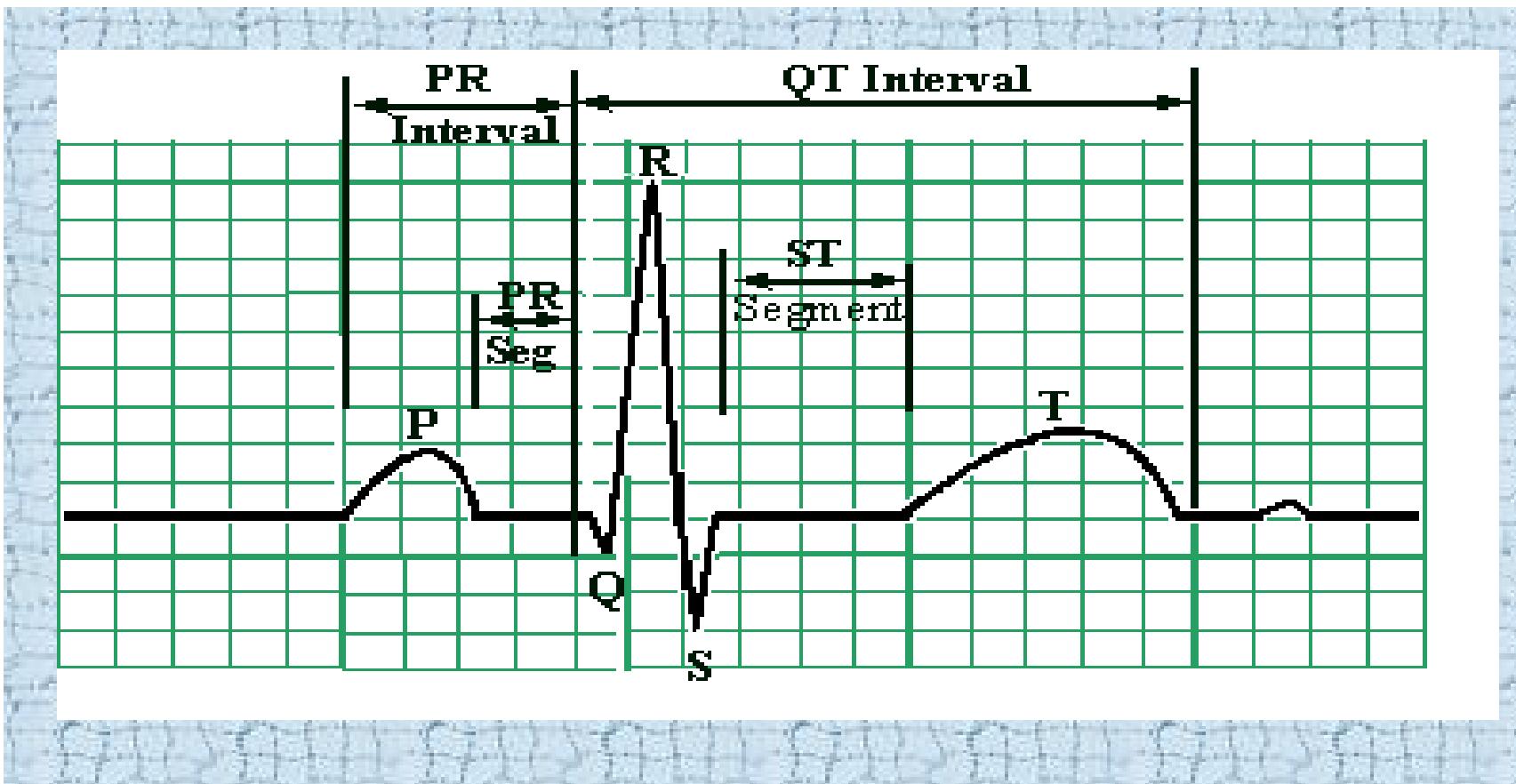
- Blowing Holosystolic murmur
- Constant at apex
- Radiation to axilla
- Pts with MVP one or more systolic clicks precede murmur, S3 if LV dysfunction

Cardiac arrhythmia

EKG

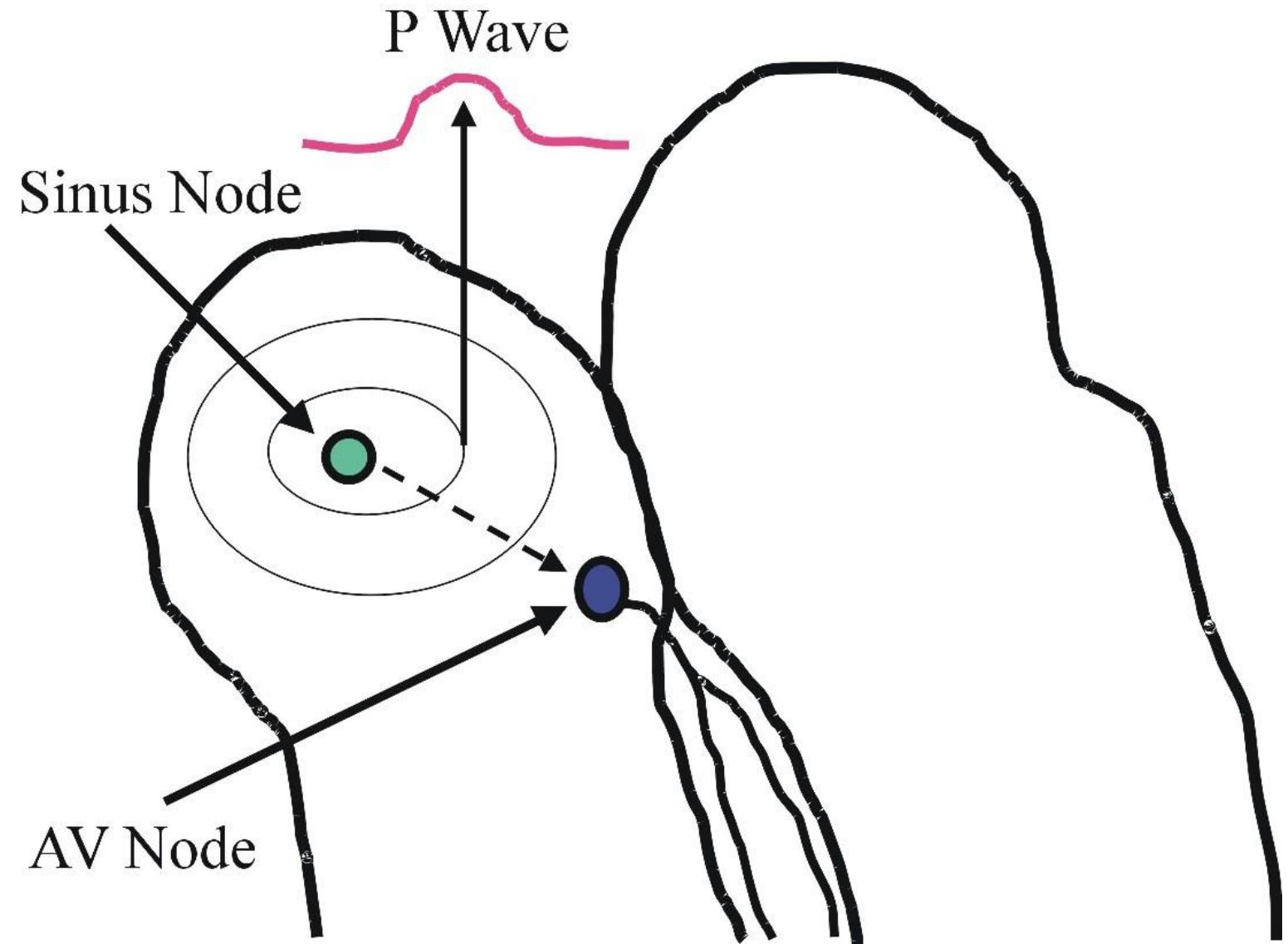
References for this EKG lecture

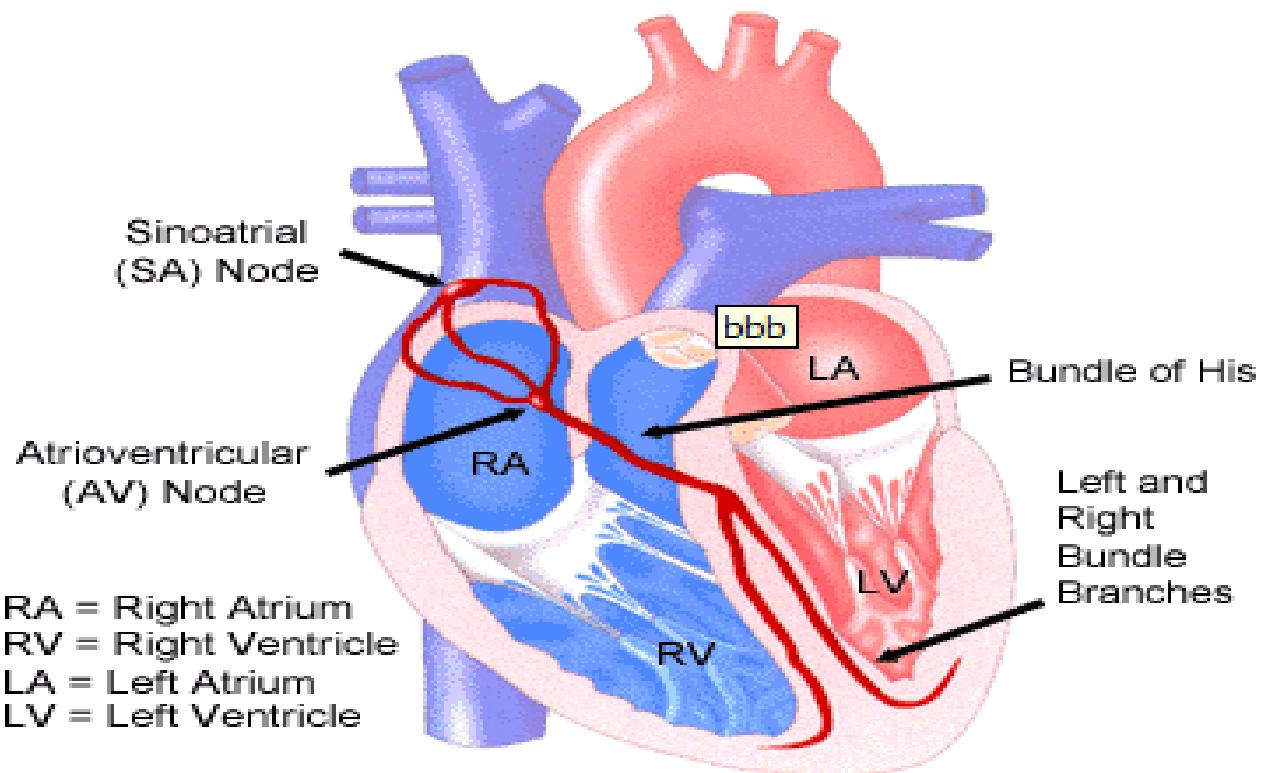


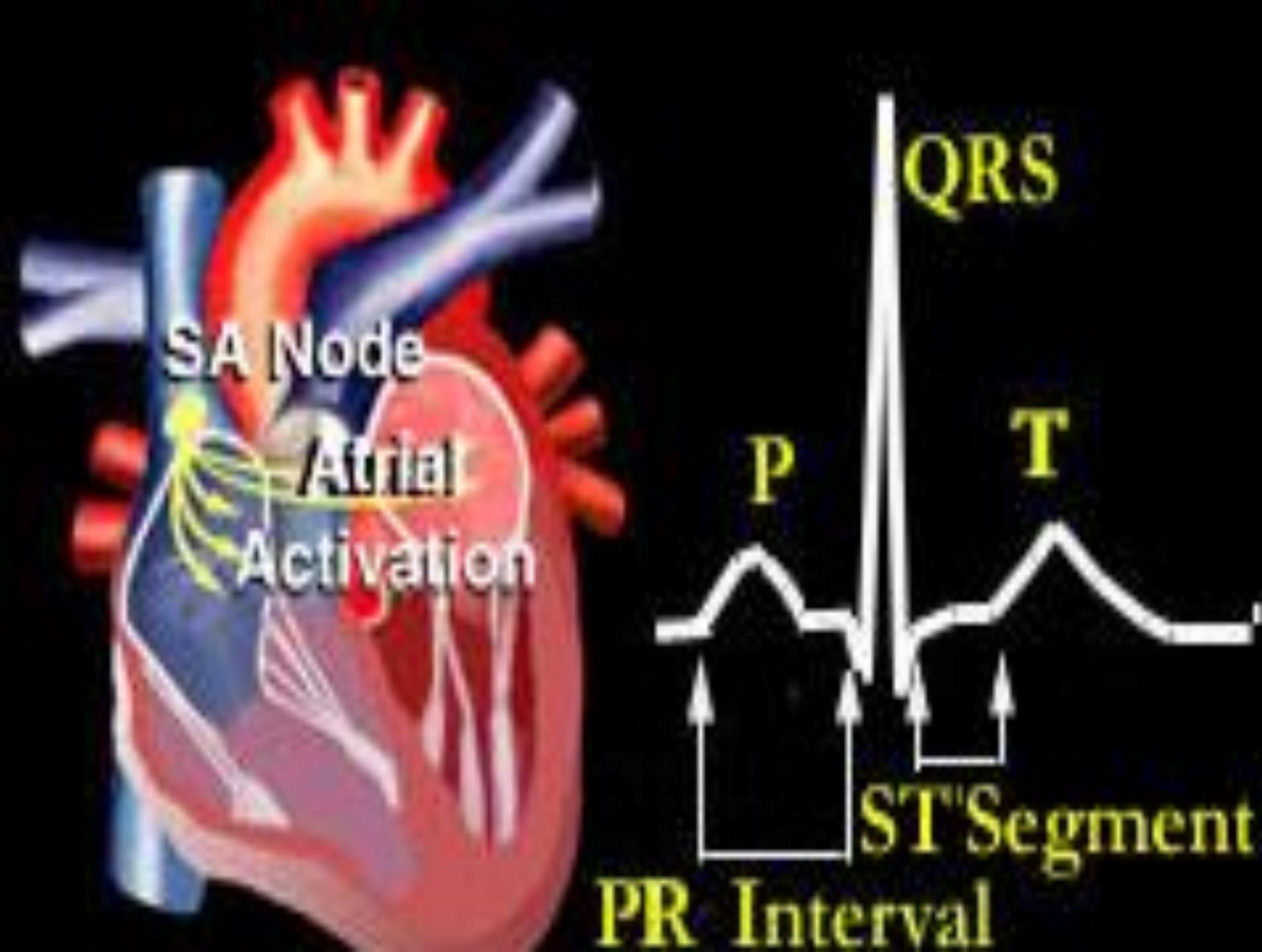


SA node (sinus node)

- SA node- located in upper posterior wall of right atrium
- Heart's dominant pacemaker
- Heart's main automaticity foci
- Depolarization from SA node producing an enlarging wave into atrium simulate a pebble dropped in a pool of water
- Atrial depolarization stim. Atrial contraction





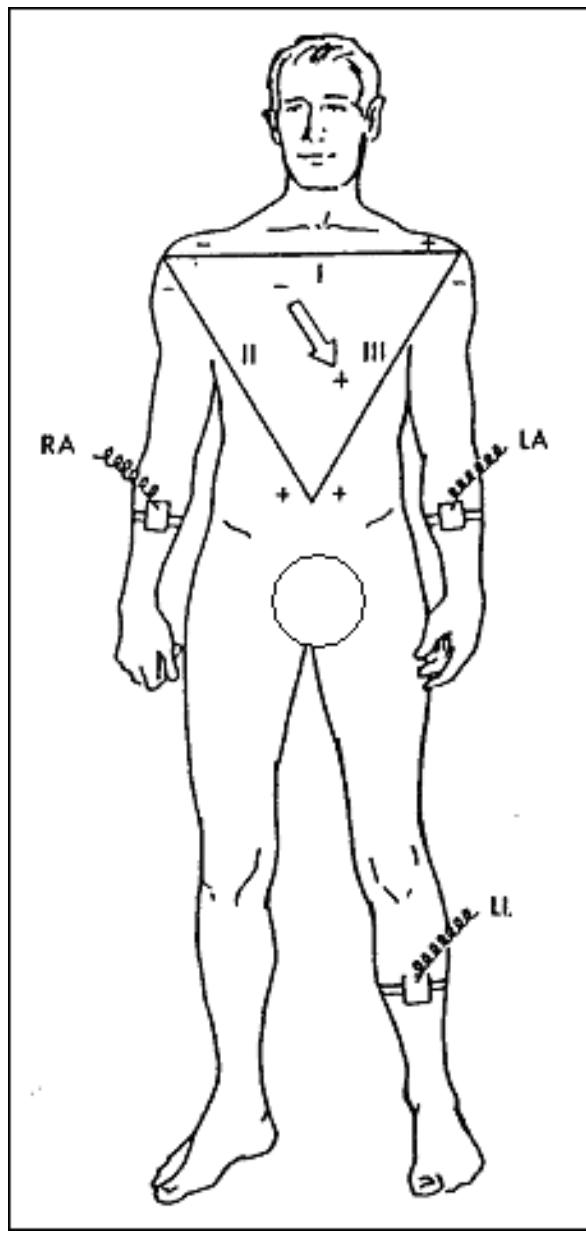


leads

- Limb leads
- I
- II
- III
- AVR
- AVL
- AVF
- Chest leads
- V1
- V2
- V3
- V4
- V5
- V6

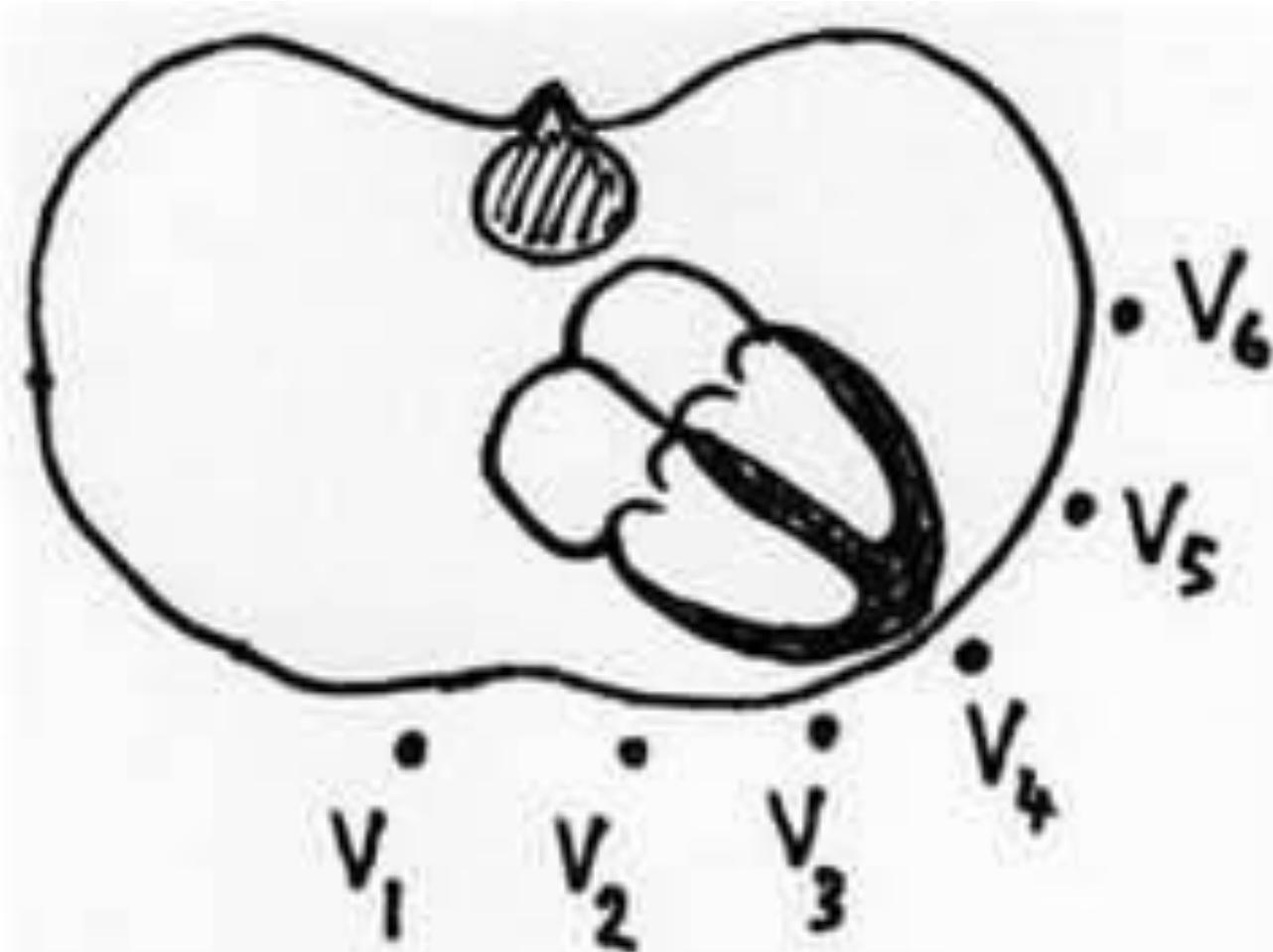
leads

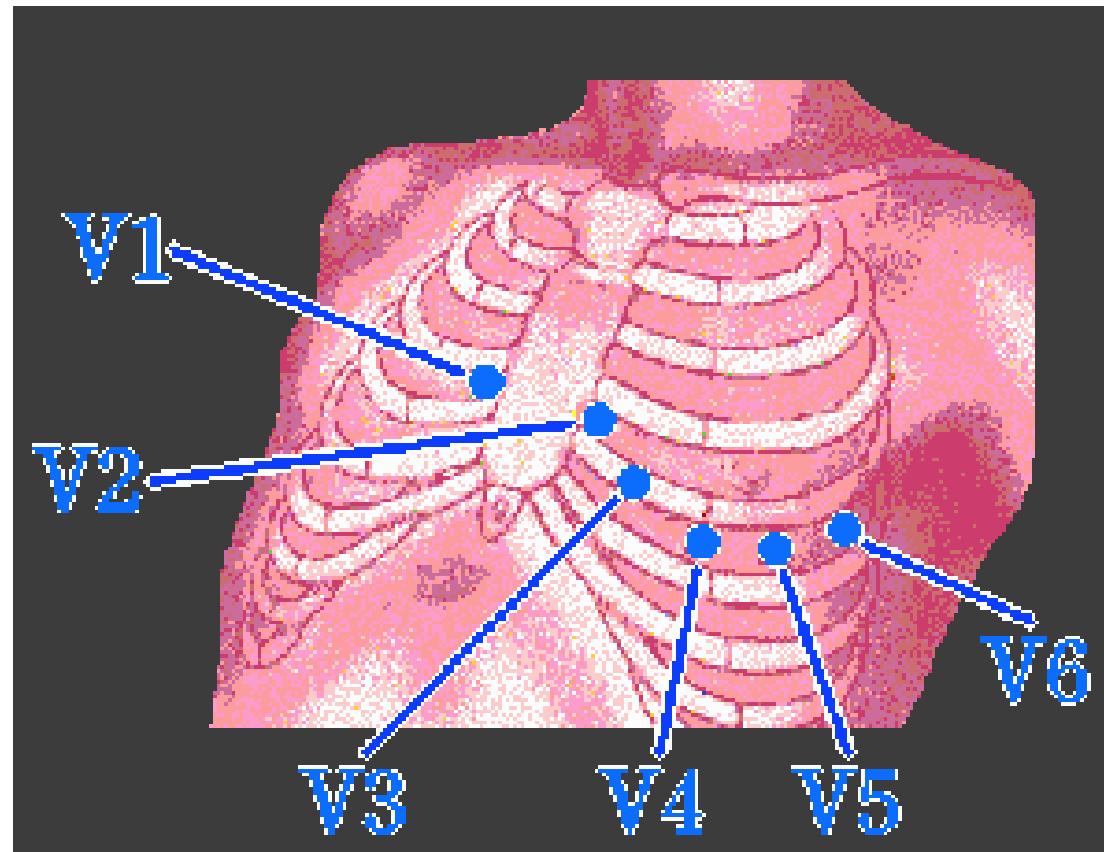
- Six limb leads record or view cardiac activity from different angles
- Six chest leads record six progressively different positions around the chest correspond to different parts of the heart



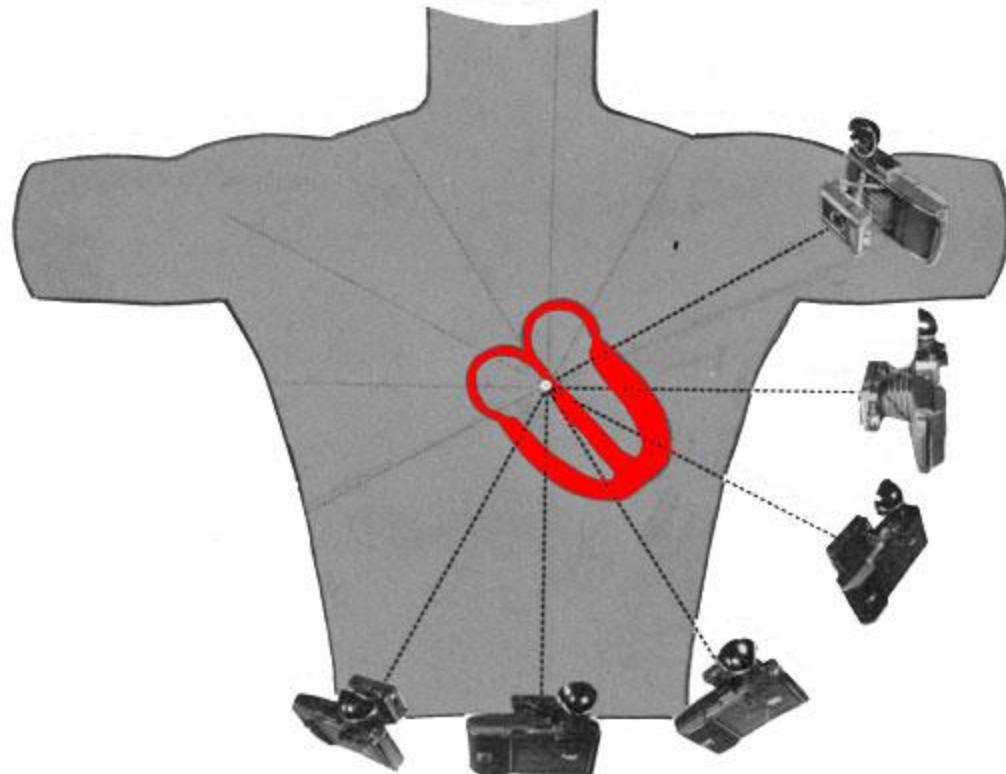
Chest leads record diff. position of heart

- V1, V2 are called the right chest leads
- V3, V4 are oriented over the interventricular septum
- V5, V6 are the left chest leads





5.5



DIAGRAM

Automaticity foci

- The ability to generate pacemaking stimuli is known as automaticity
- Automaticity foci: SA node
 - Atrium
 - AV node (prox,distal)
or junctional
 - Ventricular
 -

Interpretation of EKG

- Rate
- Rhythm
- Axis
- Hypertrophy
- Infarction

Rate/automaticity foci

- SA node
- Sinus Rhythm = 60-100/min
- Sinus Bradycardia = < 60/min
- Sinus Tachycardia = > 100/min

Rate/automaticity foci

- Atria 60-80/min
- AV junction 40-60/min
- Ventricles 20-40/min

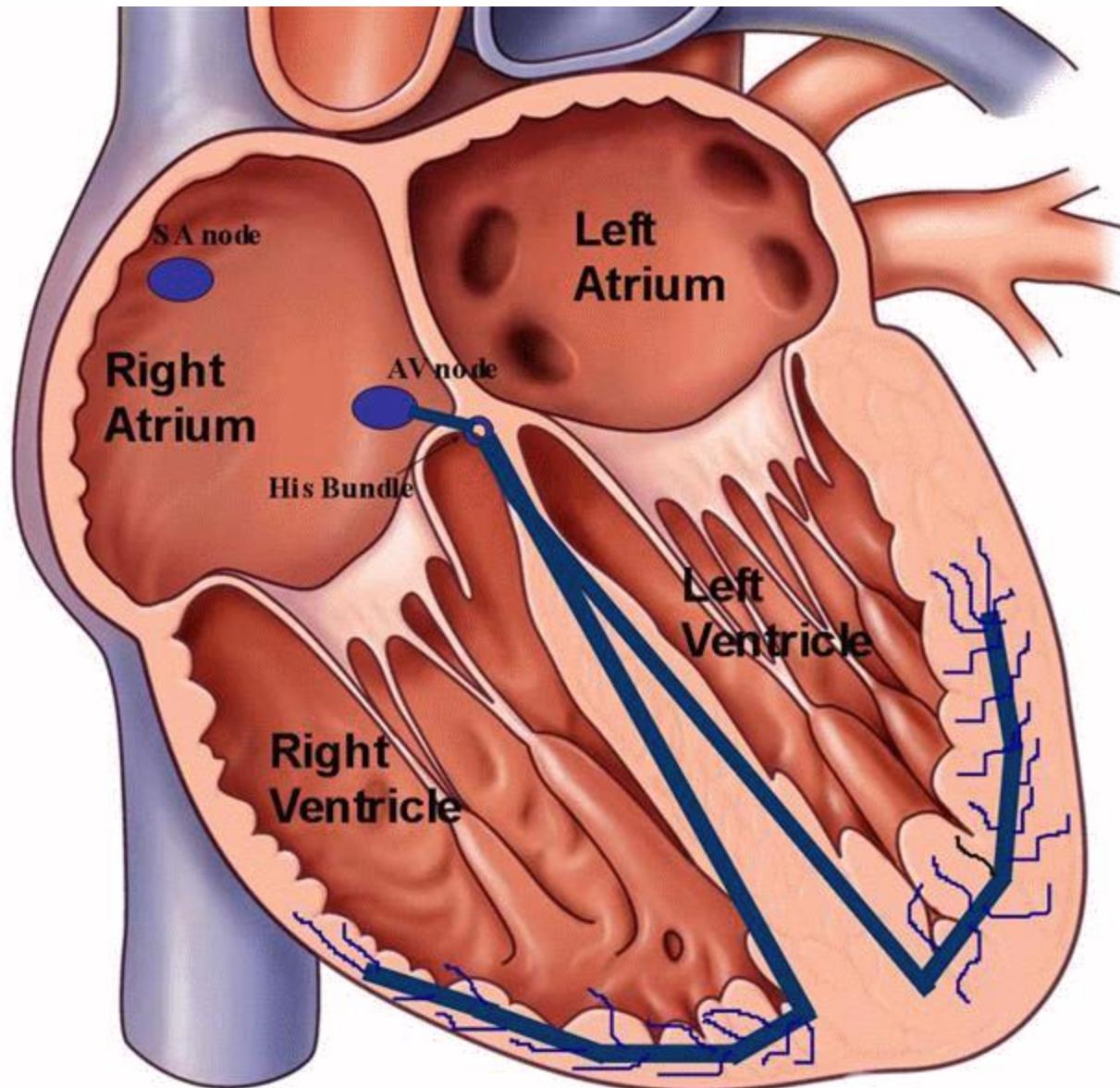
- SA node- located in upper posterior wall of the right atrium
- heart's dominant pacemaker- automaticity foci
- Heart has many automaticity foci

Overdrive suppressions

- SA node overdrive suppresses all lower foci since it has the higher inherent rate
- SA node >>
- Atrial >>
- AV junction >>
- Ventricular foci

Overdrive suppressions

- If SA node fails,
- Atria foci takes over as next pacemaker with its own inherent rate 60/min
- If Atria fails,
- AV junction (40-60/min), then Ventricular foci (20-40/min)
- e.g. Junctional rhythm in AV nodal block
- e.g. Ventricular rhythm in junction block



Calculate Rate

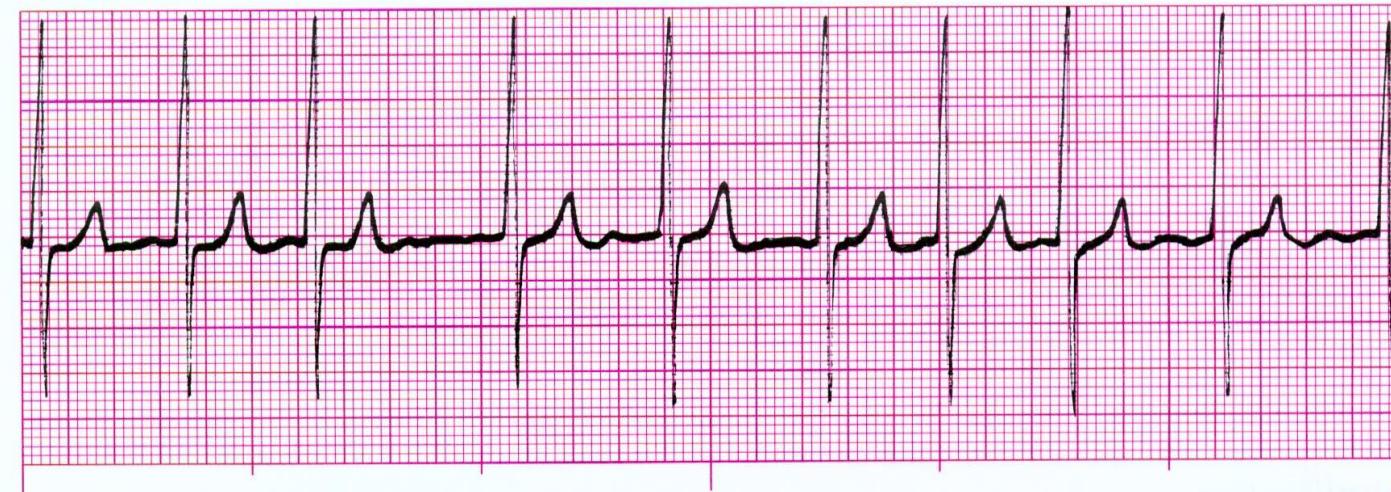
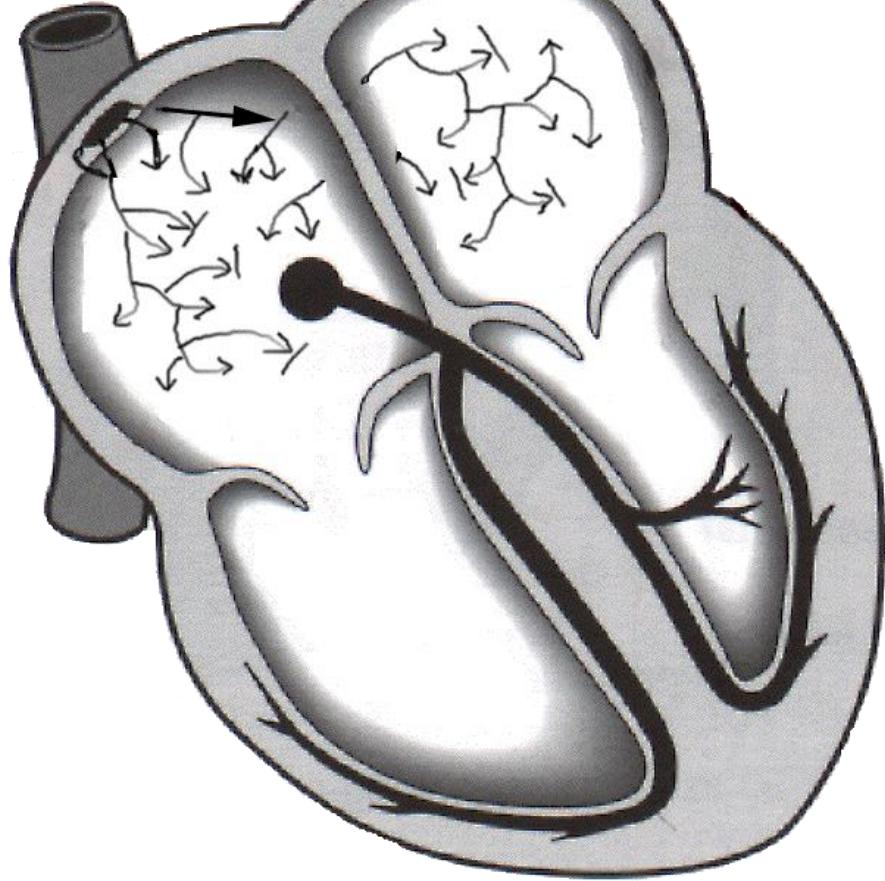
- 1st look at R wave on heavy black line
- 2nd look at the next R wave falls
- For every heavy black lines (5 small squares or 0.04×5 m sec):
- 300, 150, 100, 75, 60, 50

Irregular rhythms

- 1. Wandering pacemaker
 - 2. Multifocal Atrial Tachycardia
 - 3. Atrial Fibrillation
-
- All are caused by multiple automaticity foci activation

Atrial fibrillation

- Continuous rapid firing of multiple atrial automaticity foci
- No single impulses depolarize completely
- Only occasional impulse gets thru AV node and produce QRS
- No true P waves
- Irregular ventricular rate





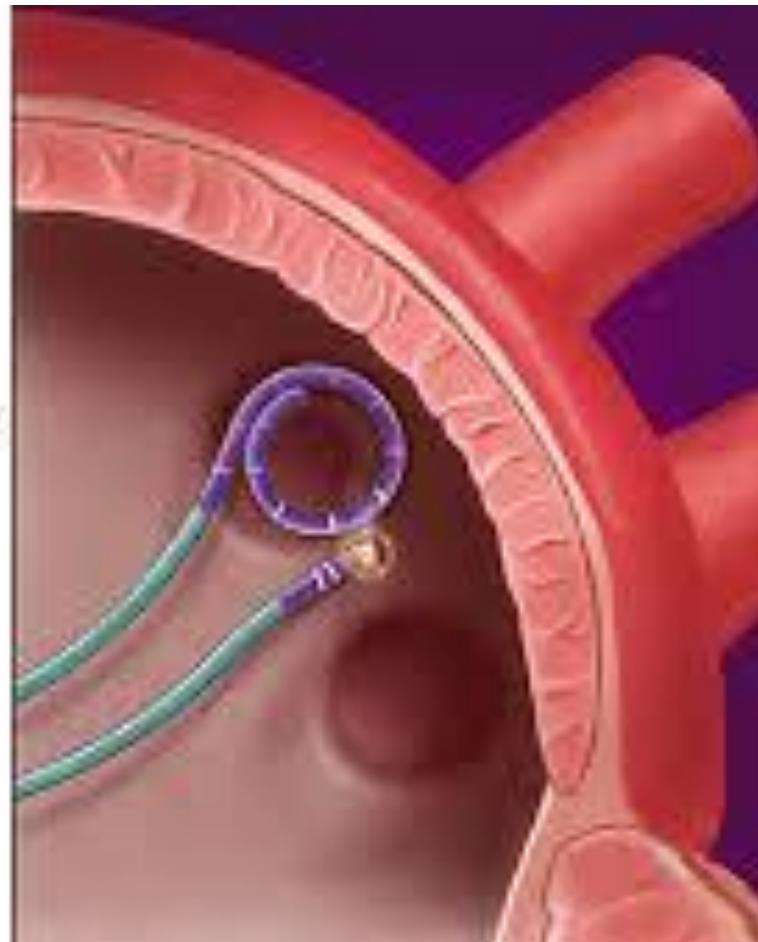
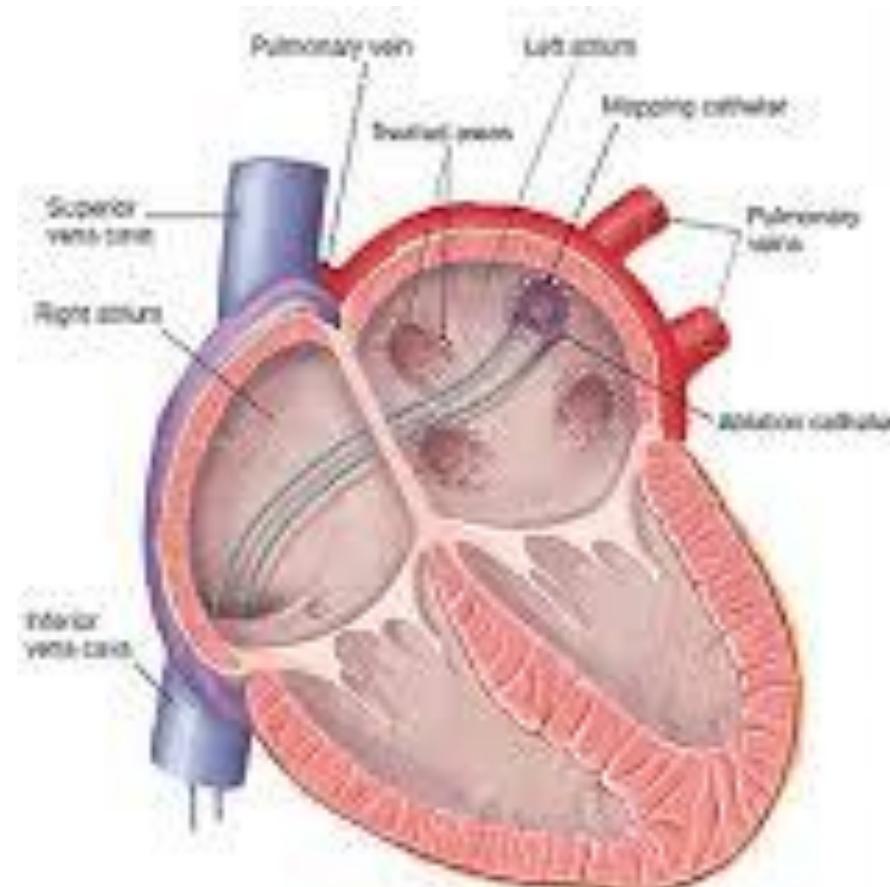
Causes of Atrial fibrillation

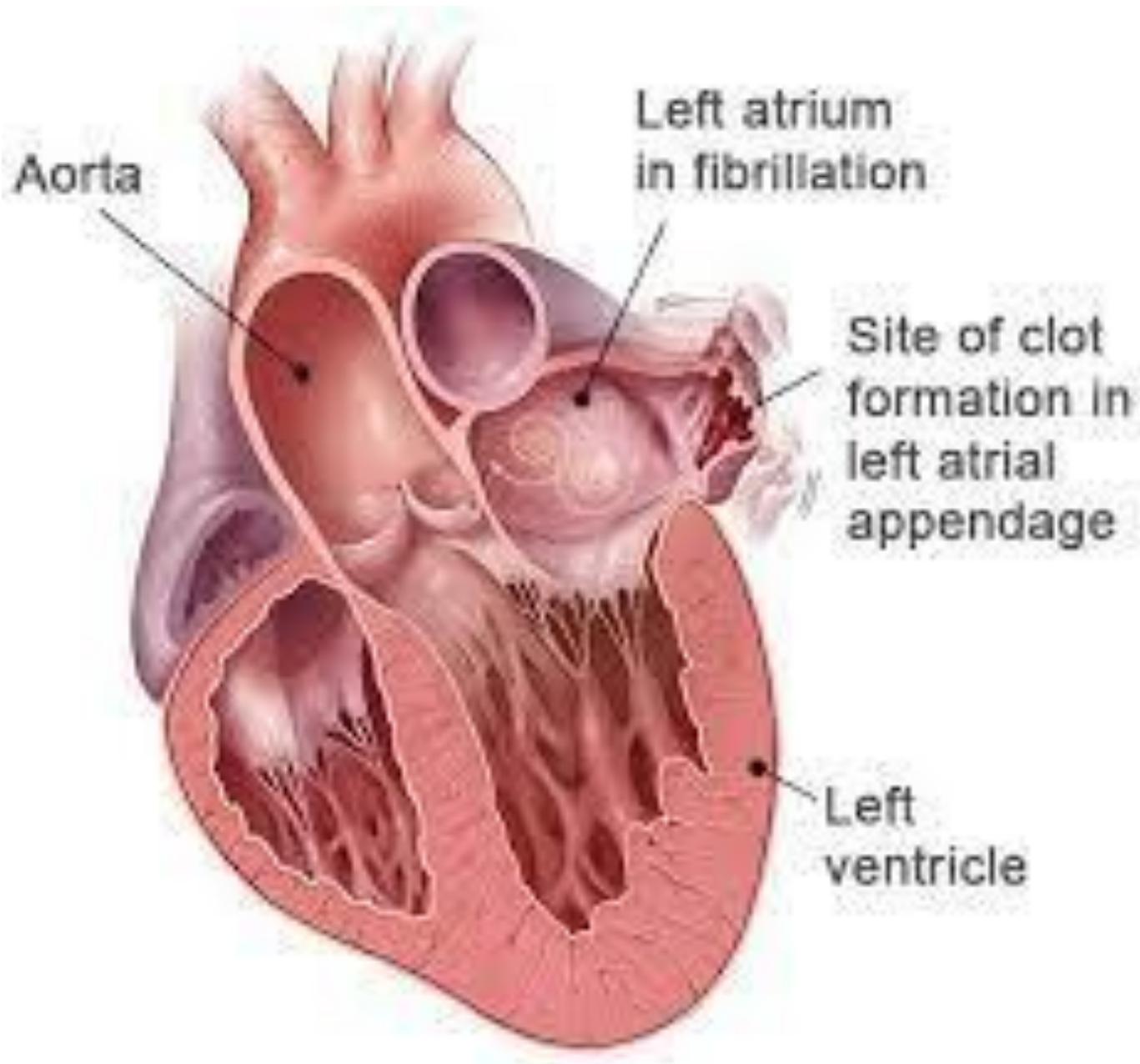
- High blood pressure
- Heart attacks
- Abnormal heart valves
- Heart defects you're born with (congenital)
- An overactive thyroid gland or other metabolic imbalance
- Exposure to stimulants, such as medications, caffeine or tobacco, or to alcohol
- Sick sinus syndrome — functioning of the heart's natural pacemaker
- Emphysema or other lung diseases
- Previous heart surgery
- Viral infections
- Stress due to pneumonia, surgery or other illnesses
- Sleep apnea

Atrial fibrillation management

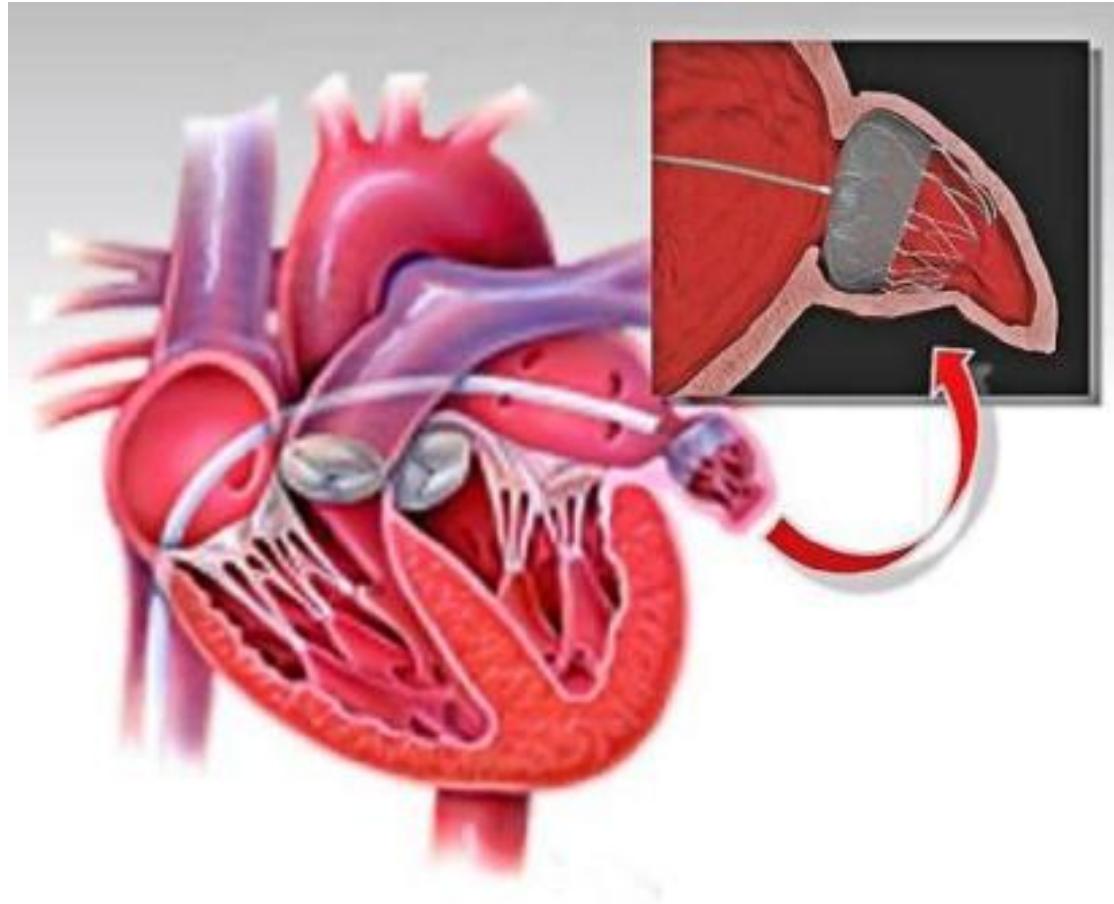
- Cardioversion
- Rate control
- Restoration of sinus rhythm
- Prevention of Stroke with anti-coagulation

Image:<https://ahvsc.com/procedures/cardiac-ablation-for-atrial-fibrillation/>





<https://www.intracare.co.nz/Procedures-and-treatments/Interventional-cardiology/Structural-heart/Left-atrial-appendage-closure-LAA>



CHADS2-VASc

CHADS2 score

Congestive heart failure

Hypertension

Age 75 older

Diabetes

History of **stroke** or Transient **ischemic** attack

Vascular disease (prior MI, PAD and Ao

Plaque

Aged 65-74

Sex: female

CHADS2-VASc

- Score =0 truly low risk AF pts
- No antithrombotic therapy
- Score of 1 or >, consider anticoagulants

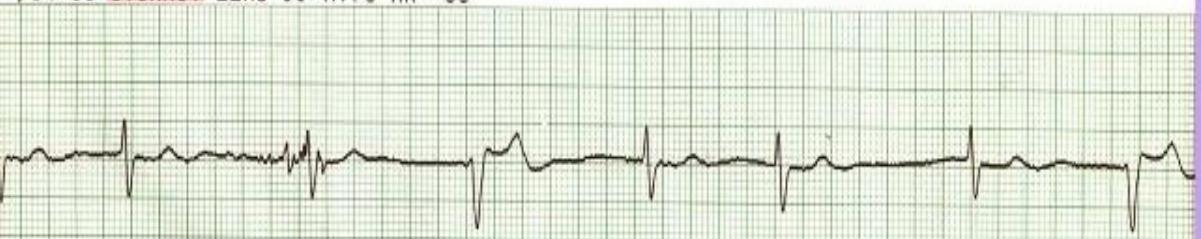
Premature ventricular contraction

- Ventricular focus become irritable from:
- Hypoxia
- Reduced cardiac output
- Ischemia, infarction
- Hypokalemia
- Most deadly V. tachycardias are due to coronary insufficiency or infarction
- Lesser degree, epinephrine like subs. Clinical significance unknown

Premature ventricular contraction/ PVCs

- PVCs = premature ventricular beats = hypoxia
- Characterized by great width and enormous amplitude (height and depth)
- When see PVCs, means a weak ventricular contraction (ventricles not completely filled)

►04:35 LEAD II X1.0 HR= 60



►04:36 LEAD II X1.0 HR= 68



►04:36 LEAD II X1.0 HR= 52



Lead II

This continuous tracing shows a very slow Atrial Fibrillation with Ventricular escape beats. The second and third strips show a regular rhythm.

Premature ventricular contractions

- Unifocal PVCs – originate from the same ventricular foci
- Multifocal PVCs – originate from multiple foci of the ventricle
(HELP..HELP)
- Six or more PVCs per minute is considered pathological til proven otherwise e.g. hypoxia, low K, epinephrine



Cardiology

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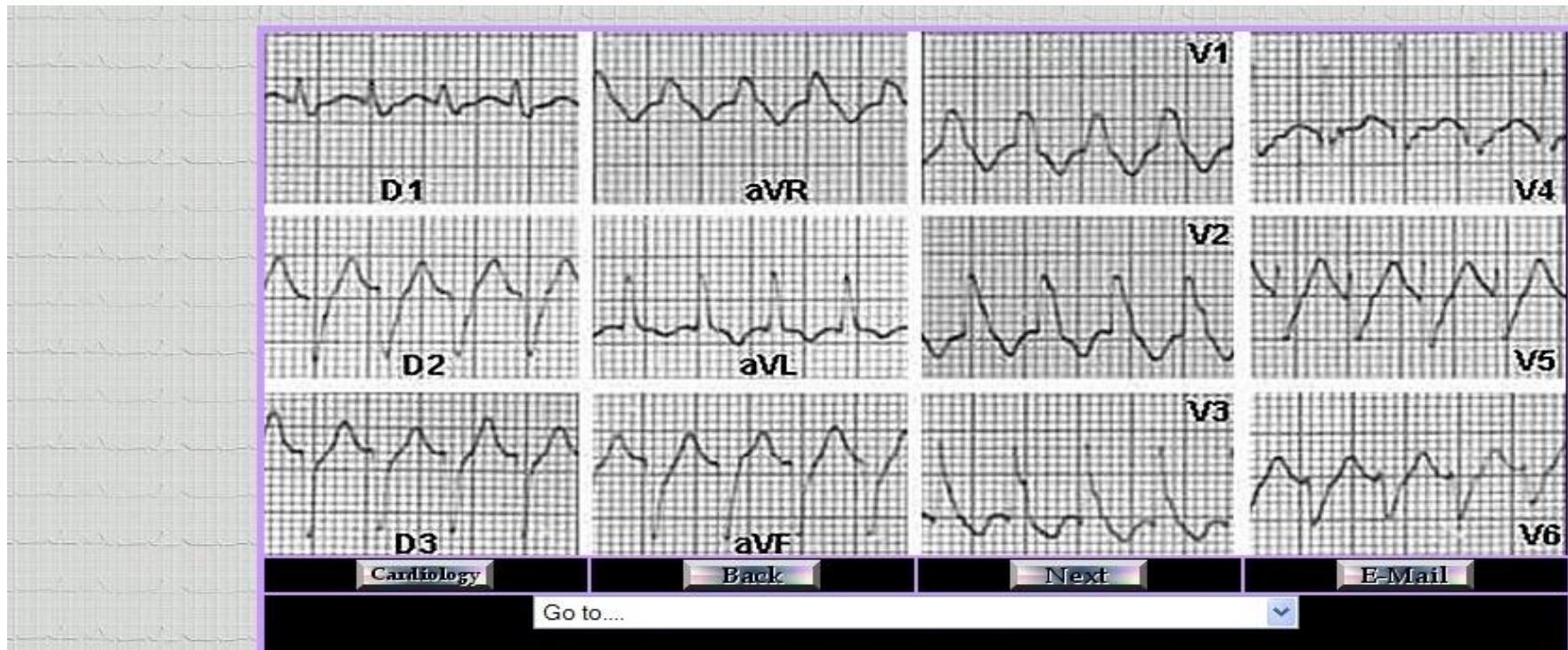
E-Mail

Go to....



Lead II

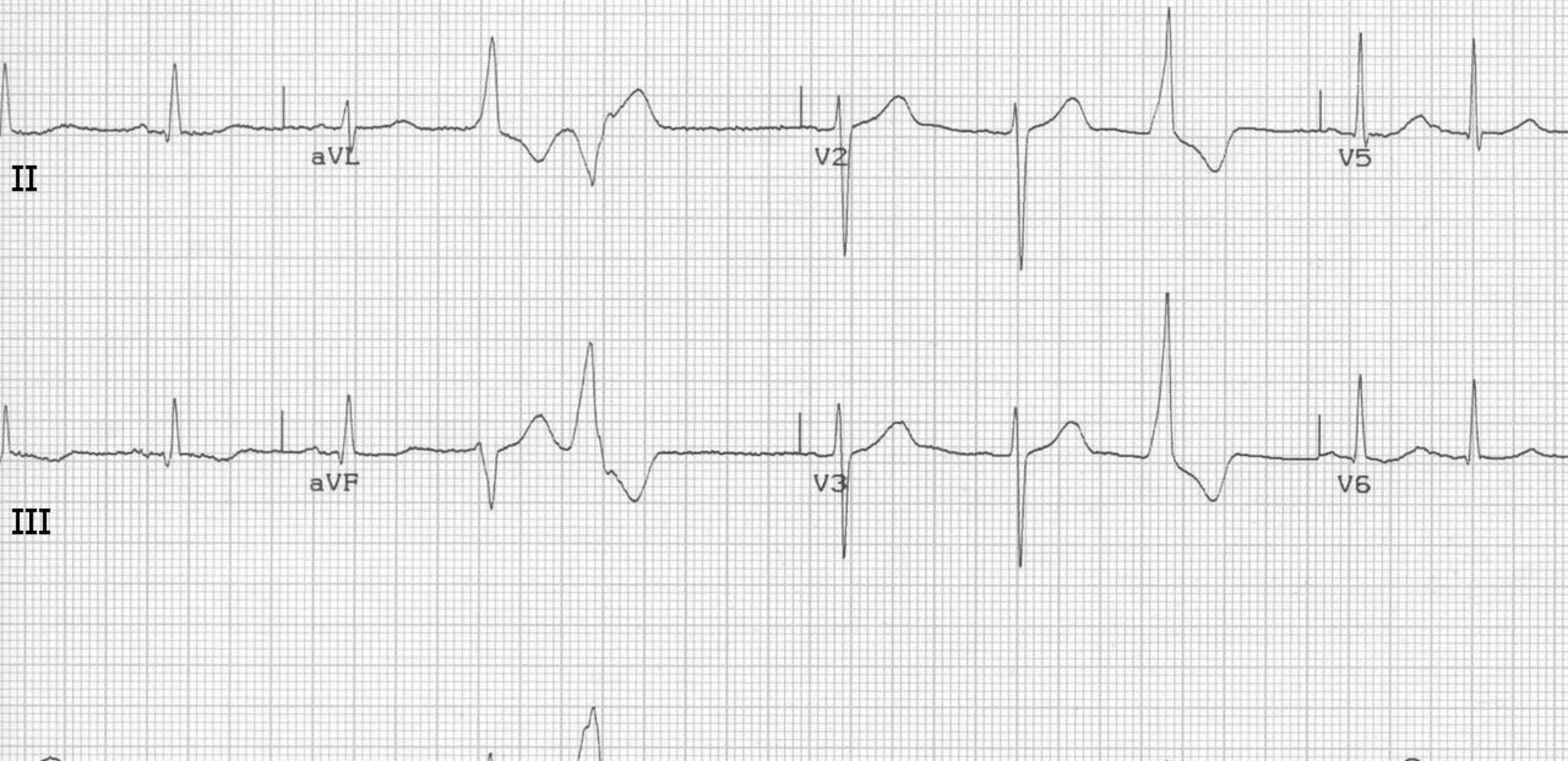
This is from an 80 YO Oriental female with a head injury from a fall. She presented with "Battle's Sign" and was unremarkable otherwise on physical exam.



Lead II

Ventricular Tachycardia.

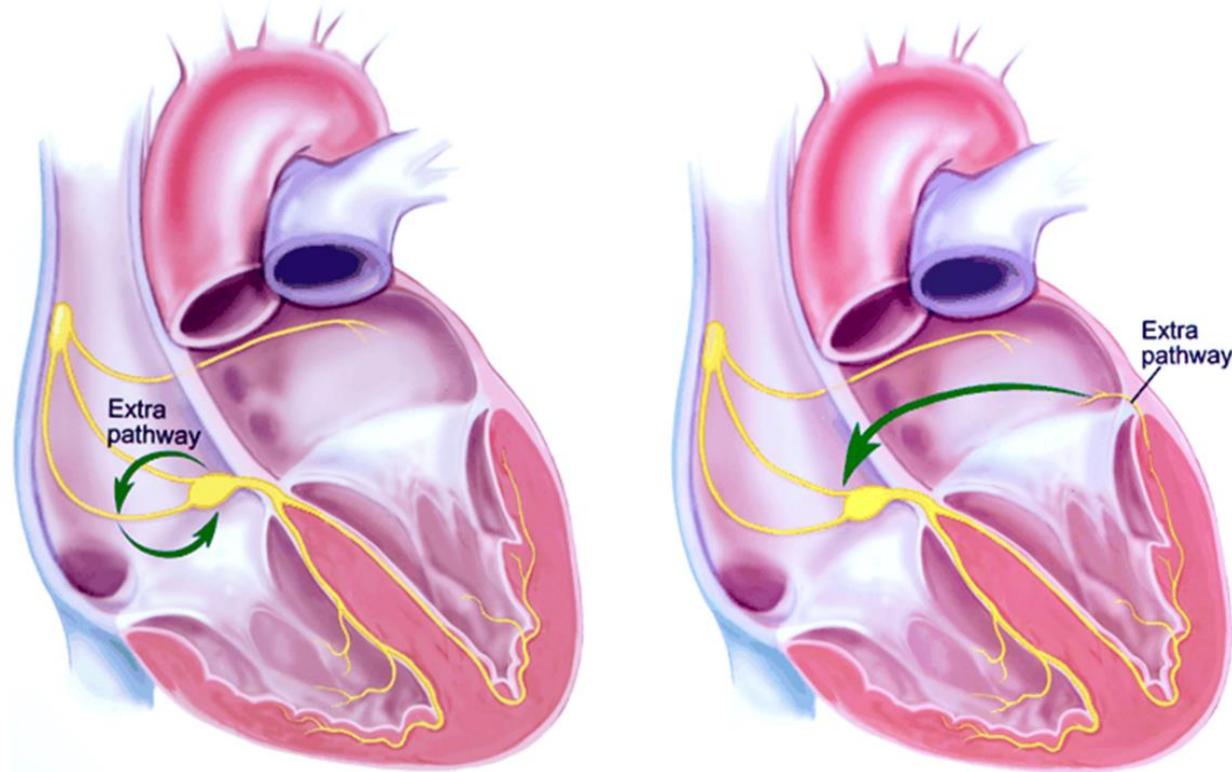
This tracing was contributed by a friend.



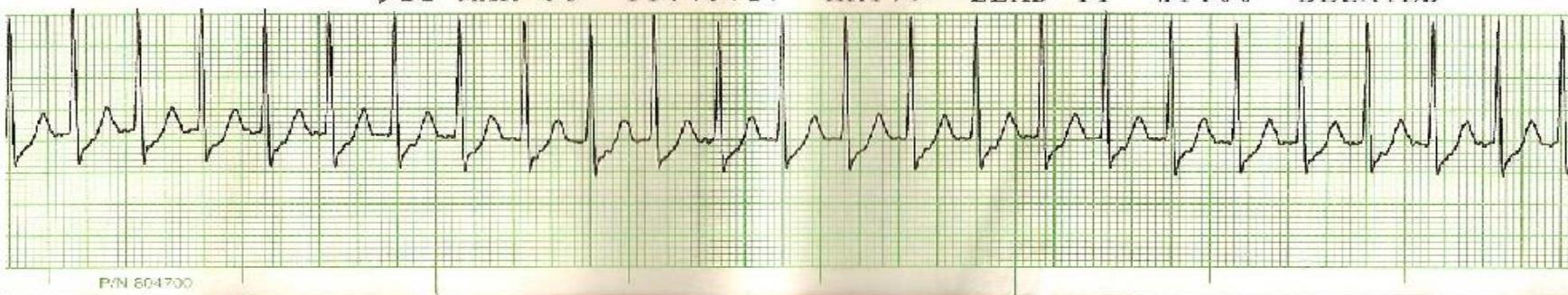
Supraventricular tachycardia

- SVTs
- All paroxysmal tachycardia which the all atrial or junctional foci with above the ventricles
- P wave runs into T waves make differentiation between two very difficult, therefore term both as SVTs – Rx is same

<https://www.ahajournals.org/doi/full/10.1161/01.cir.0000044341.43780.c7>



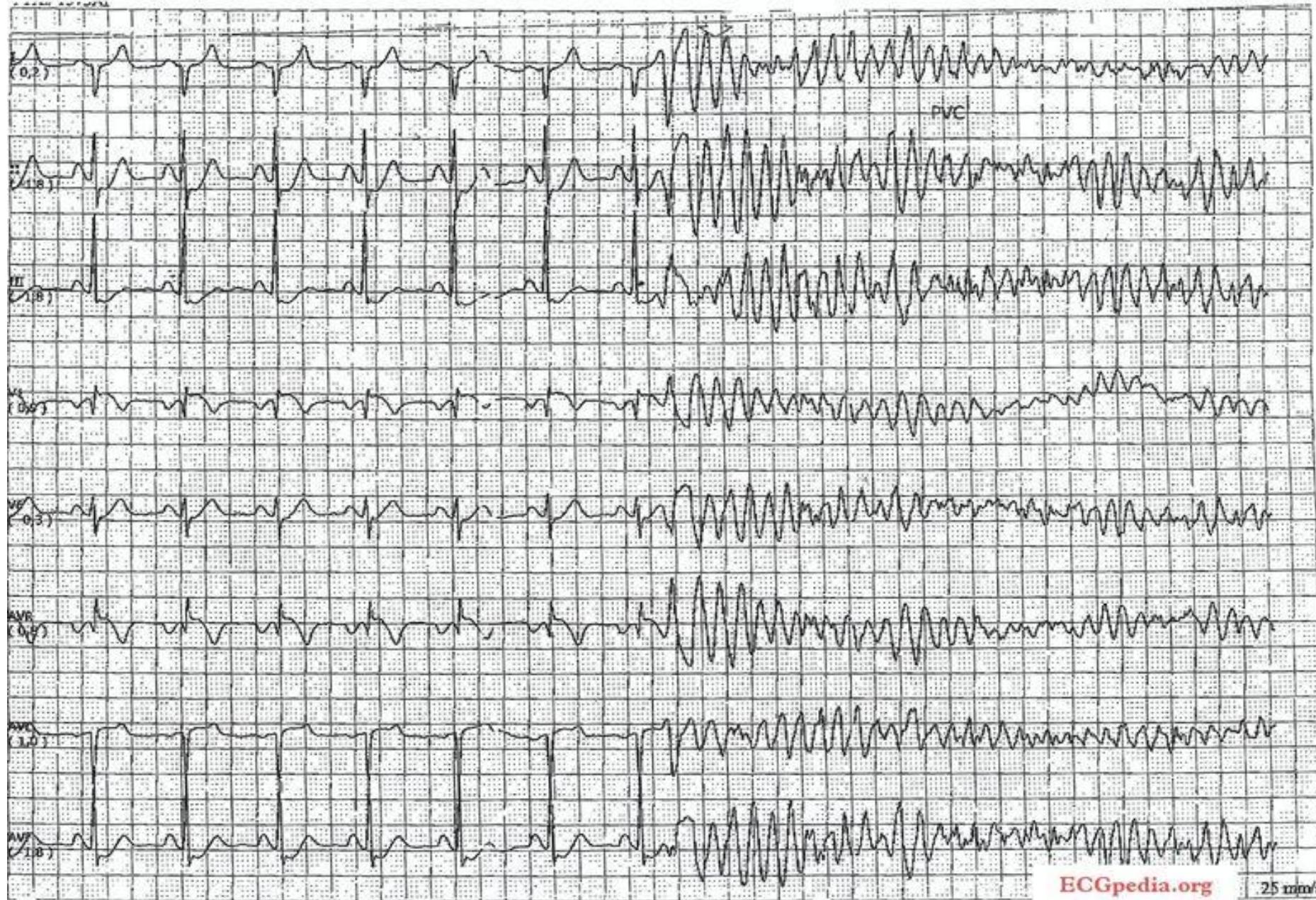
► 22 MAR 08 11:43:27 HR 179 LEAD 11 x 1.00 DELAYED



Ventricular fibrillation

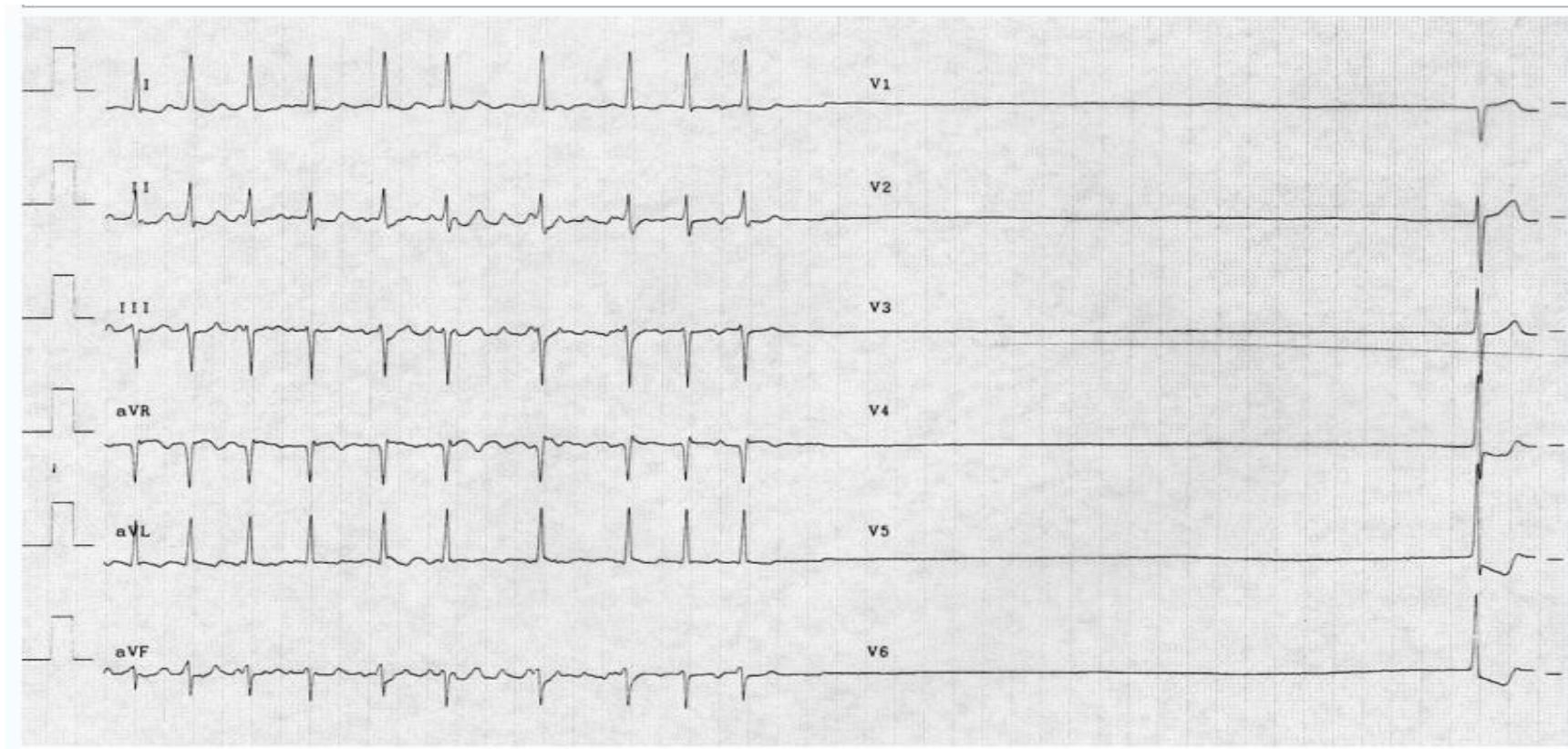
- Sometimes preceded by ventricular flutter
- Numerous irritable ventricular foci
- Erratic twitching
- Bag of worms
- Lack of identifiable waves





Heart blocks

- 1. Sinus block
- 2. AV block
- 3. Bundle branch block
- 4. Hemiblock



AV block

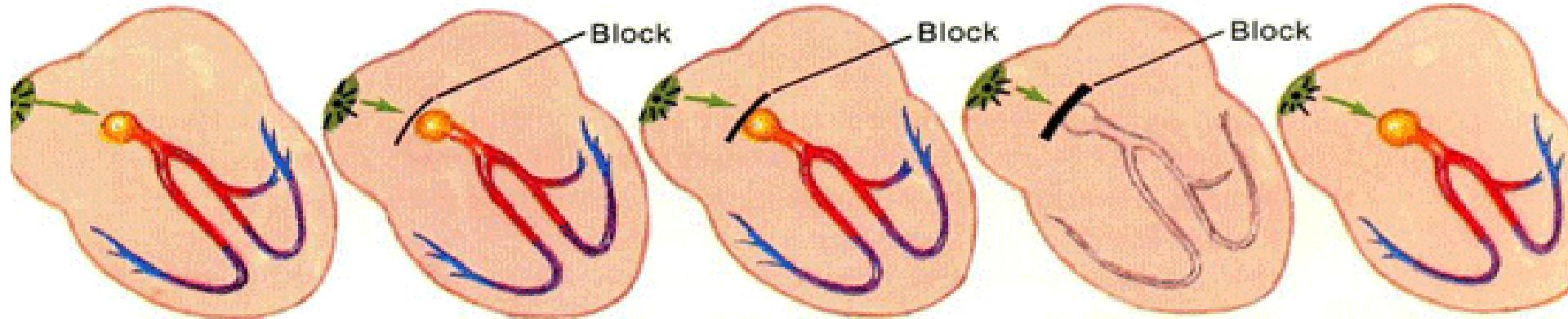
- 1 st degree AV block
- 2 nd degree AV block: Wenckebach
 - Mobitz II
- 3 rd degree AV block (complete heart block)

1 st degree AV block

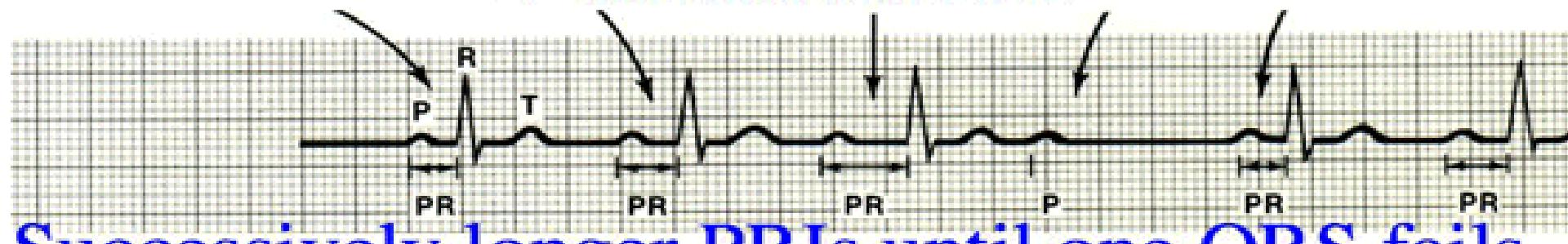
- Normal PR interval 0.12-0.20 sec
- Prolong PR interval of > 0.2 sec (one large square) = 1 st degree AV block
- No clinical significance

2 nd degree AV- Wenckebach

- Or called type I 2nd degree AV block
- PR interval becomes progressively longer from cycle to cycle until AV node no longer conduct a stimulus i.e. dropped QRS
- Block is at the AV node
- Parasympathetic excess or drugs
- Clinical significance unknown



P Waves look Similar!

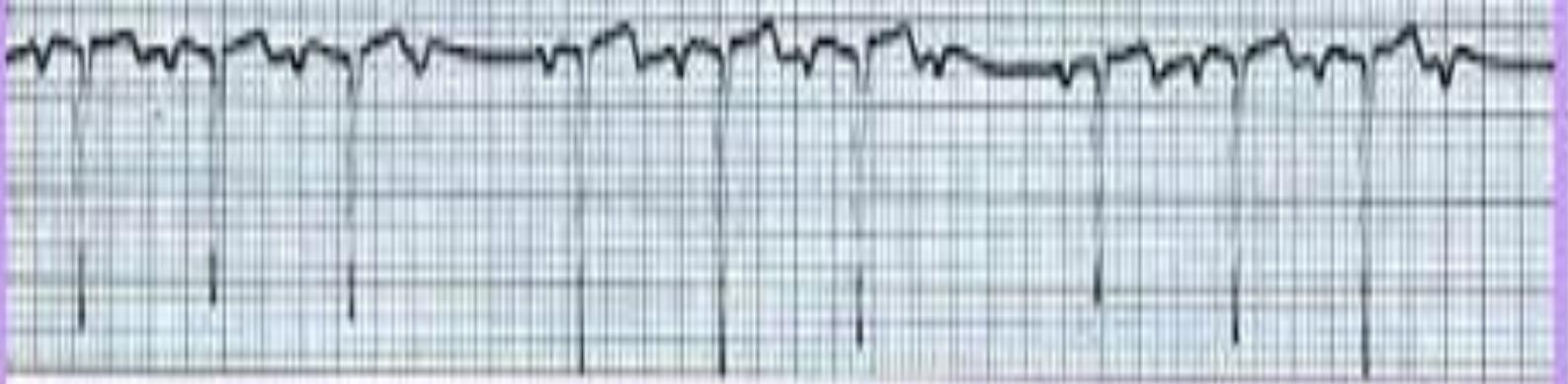


Successively longer PRIs until one QRS fails

Rhythm (ventricular) is often irregular

Atrial rhythm is ~ regular, QRS is normal



A continuous ECG tracing is visible at the top of the page, serving as a background.

Cardiology

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E-Mail

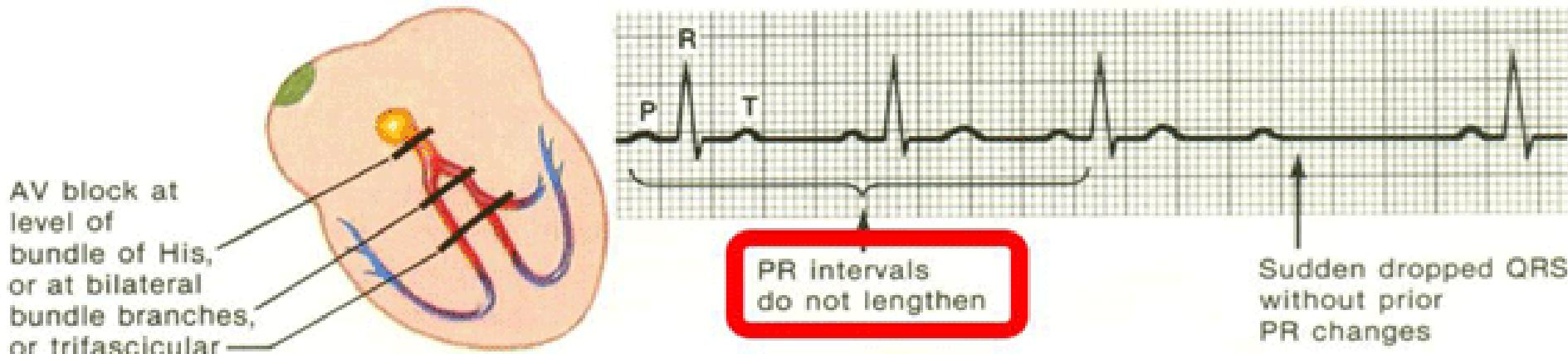
Go to....

2nd degree AV block-mobitz II

- PR interval is normal, AV block may cause every other QRS dropped causing 2:1 block or if every 3rd QRS dropped causing 3:1 AV block
- Originates below AV node in His bundle or branches
- Early warning sign of impending complete AV block, therefore warrants pacemaker

2° AV Block Mobitz II

Suddenly dropped QRS



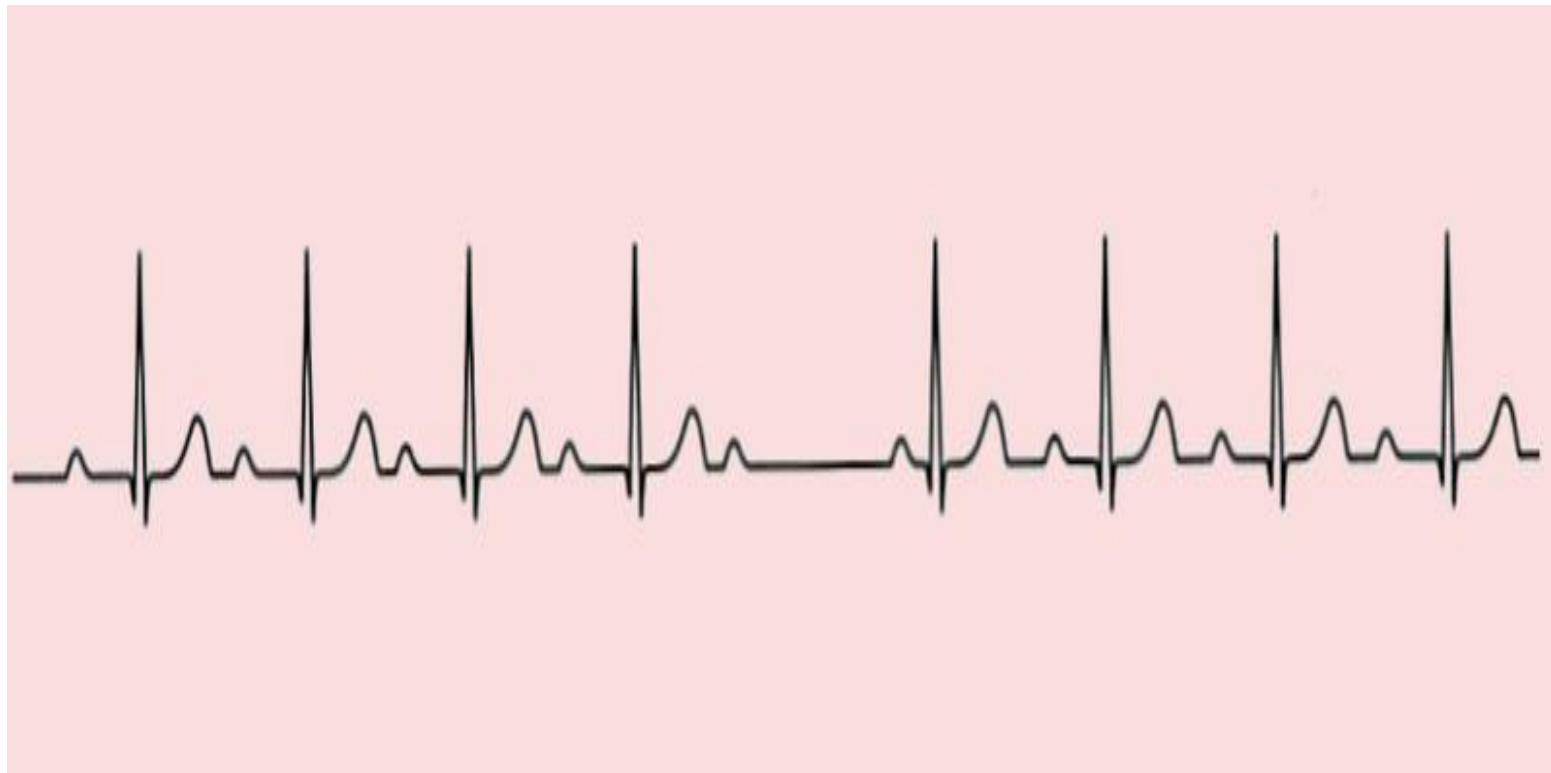
P waves are punctual and similar, **unlike a non-conducted PAC which is EARLY!**

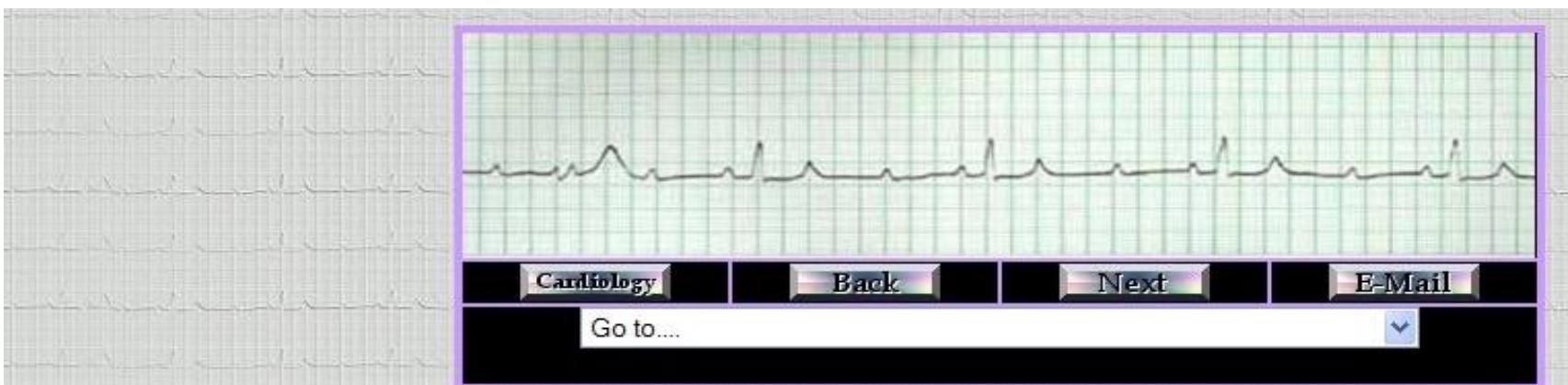
Ventricular rhythm = irregular, atrial rhythm is regular

PRI normal or prolonged

QRS: often abnormal

- WWW.UNM.EDU





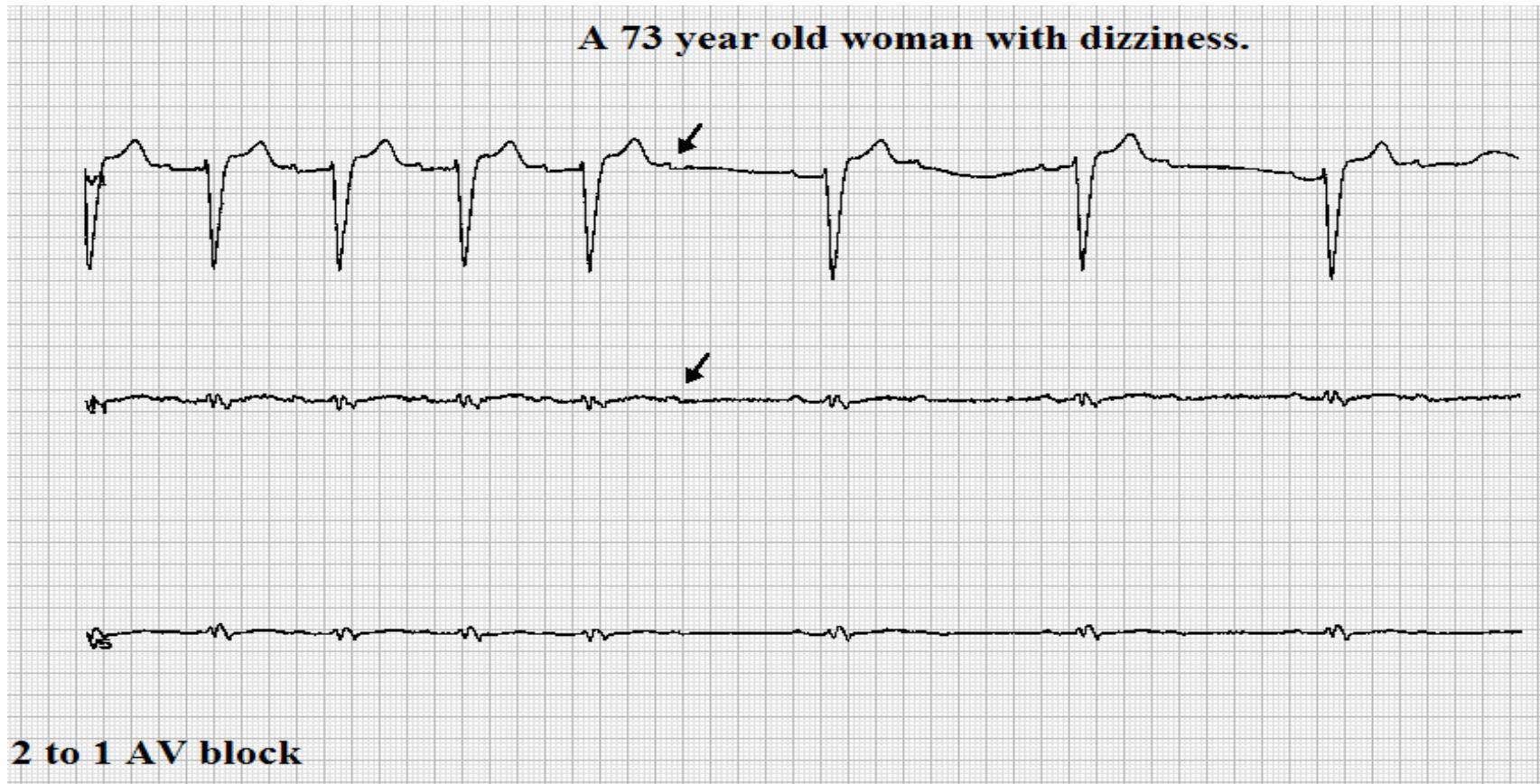
Lead II

Sinus Rhythm with Mobitz Type II AV Block.

Sinus Rate is 100/min. with a ventricular rate of 40/min. Note the second QRS complex.

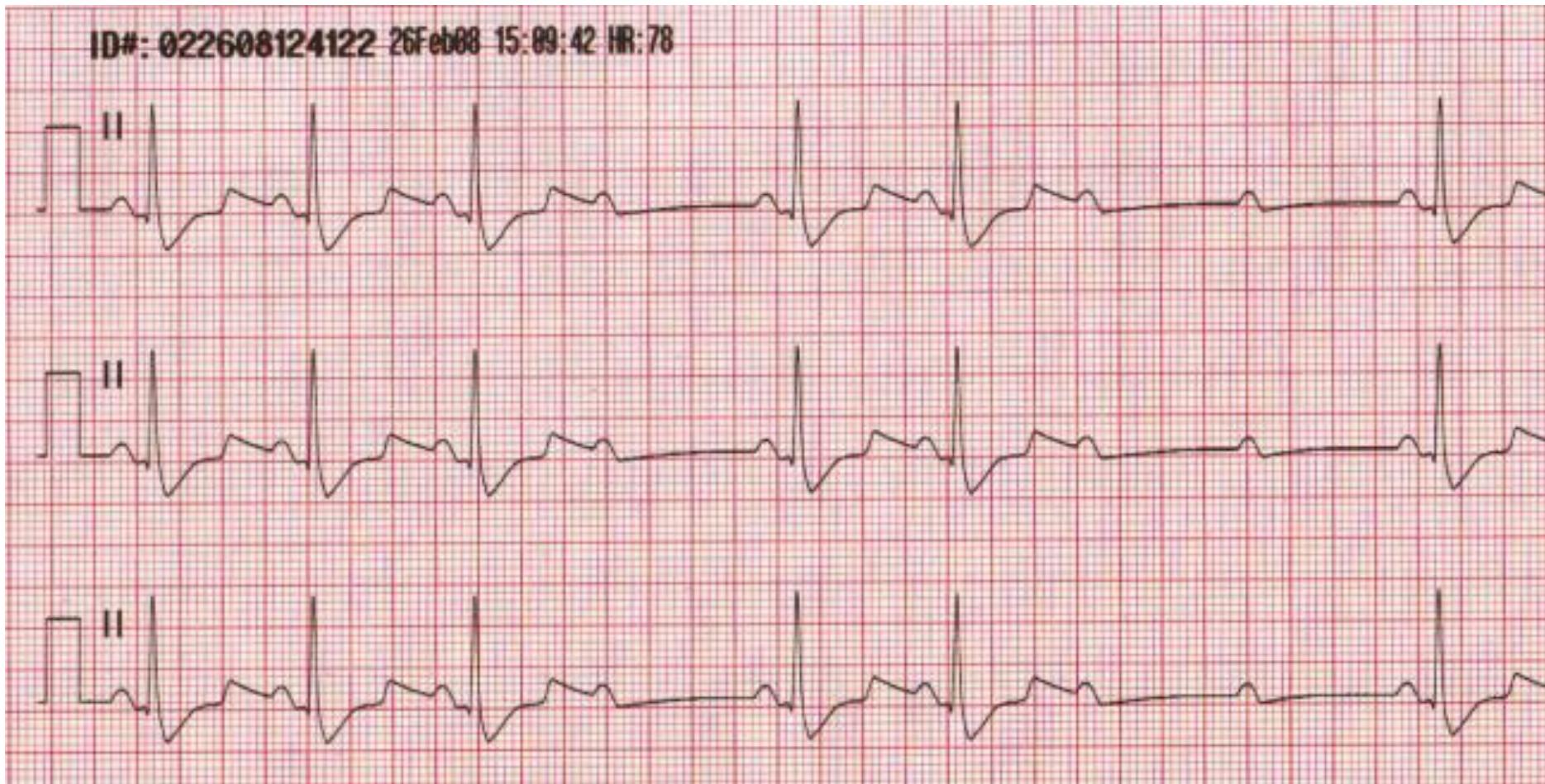
This tracing was contributed by a friend.

A 73 year old woman with dizziness.



2 to 1 AV block

ID#: 022608124122 26Feb08 15:09:42 HR:78



3 rd degree AV block (complete heart block)

- None of the atrial depolarizations conduct to ventricles. Atrial (P wave) and junctional or Ventricle (QRS) each pace at its own rate = AV dissociation
- If block above AV junction, junctional focus escapes to pace the ventricles, ventricular rate will be 40-60s = idio-junctional rhythm
- If block below AV junction, ventricular focus escapes to pace = AV dissociation
- Warrants pacemaker

The End