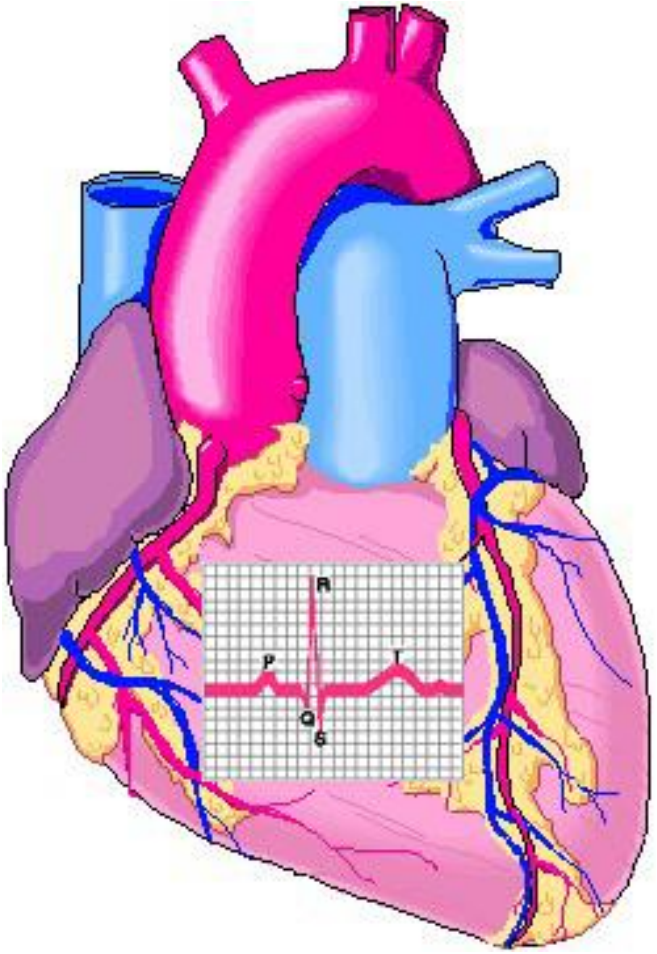
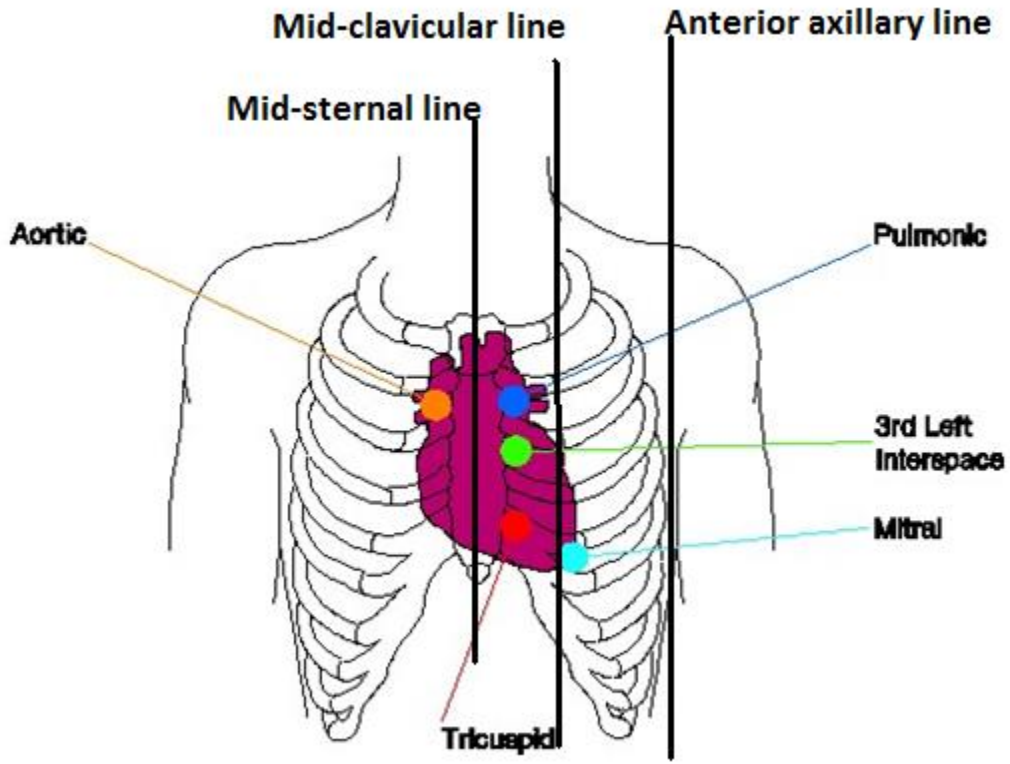
Cardiology Essentials



Physical Location in Thoracic Cavity



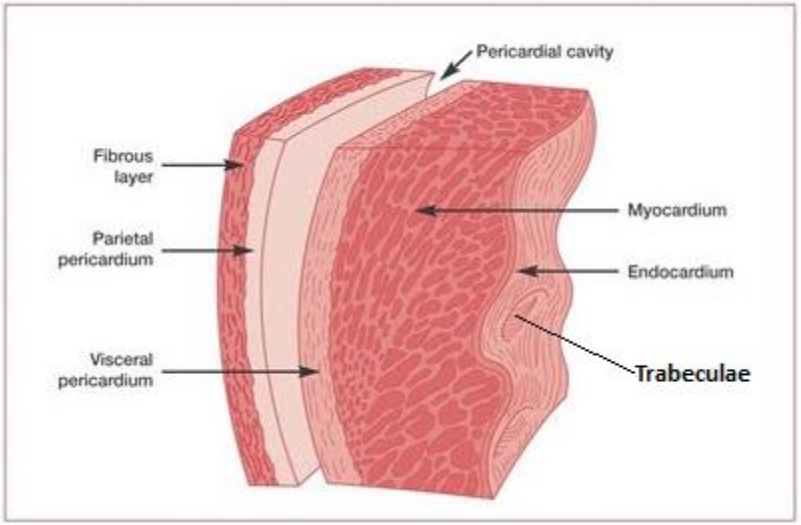
Aortic valve – 2d rib/intercostal space, right sternal border

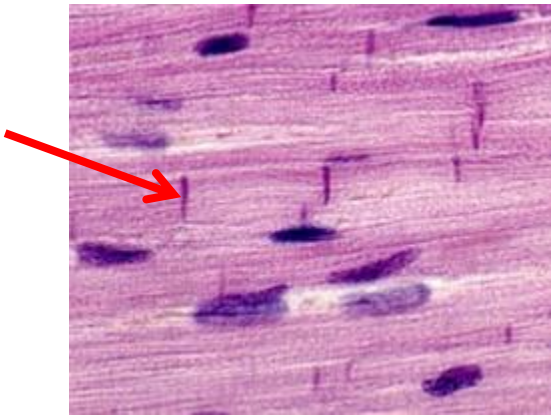
Pulmonic valve – 2d intercostal space left sternal border

Tricuspid valve – 5th rib, left sternal border

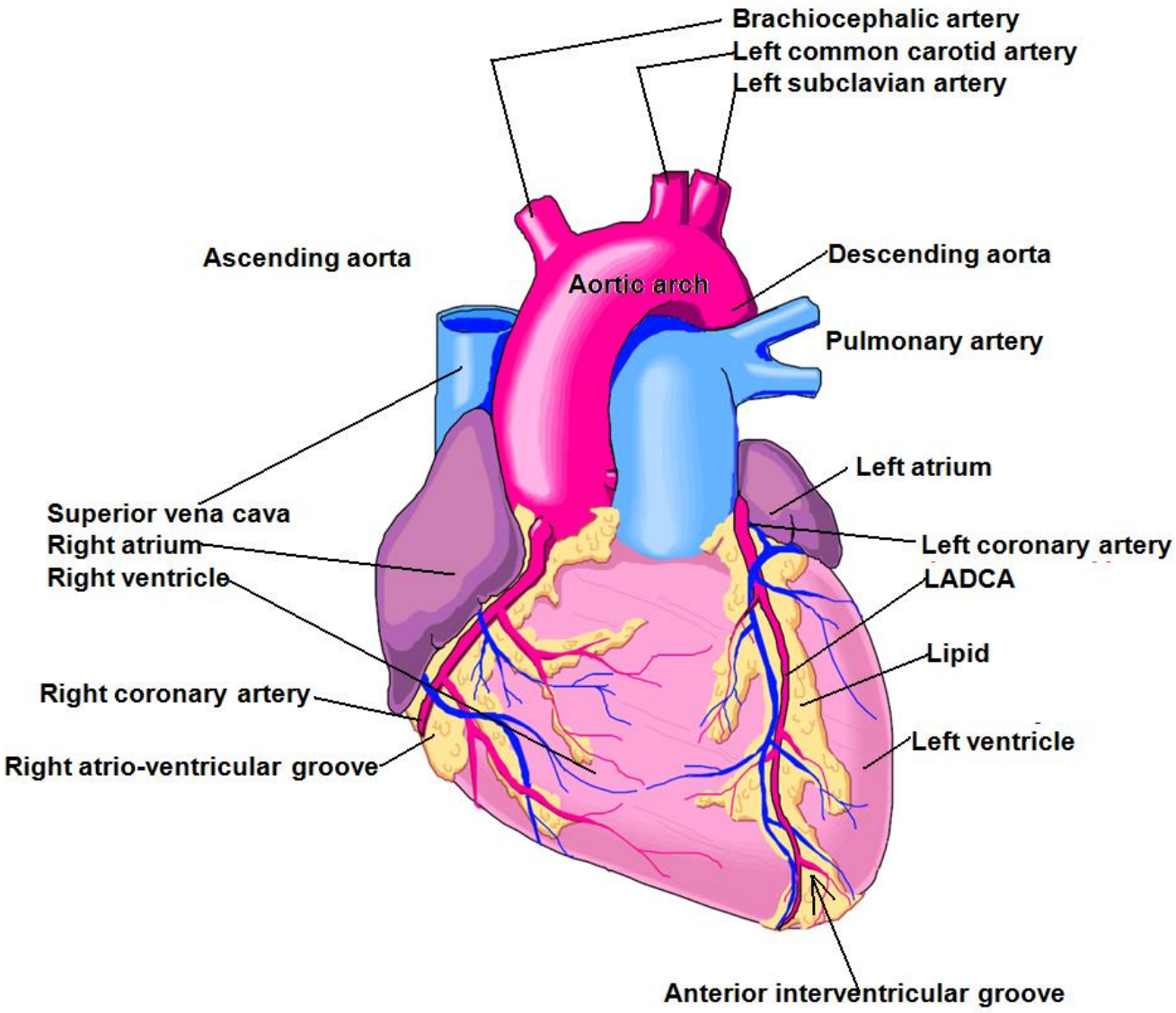
Mitral valve & PMI – 5th intercostal space – left mid-clavicular line

Structural Organization of the Myocardium

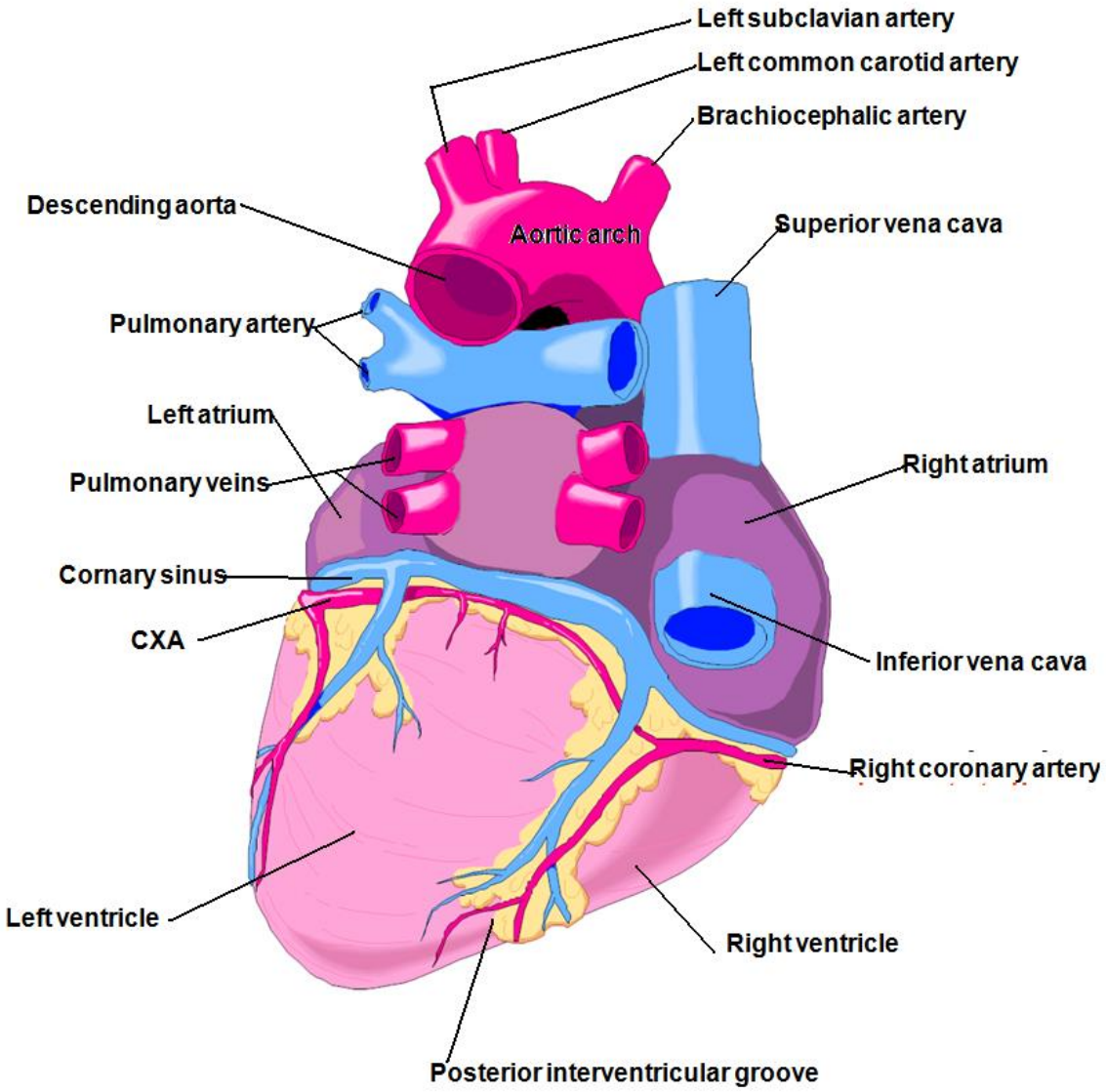




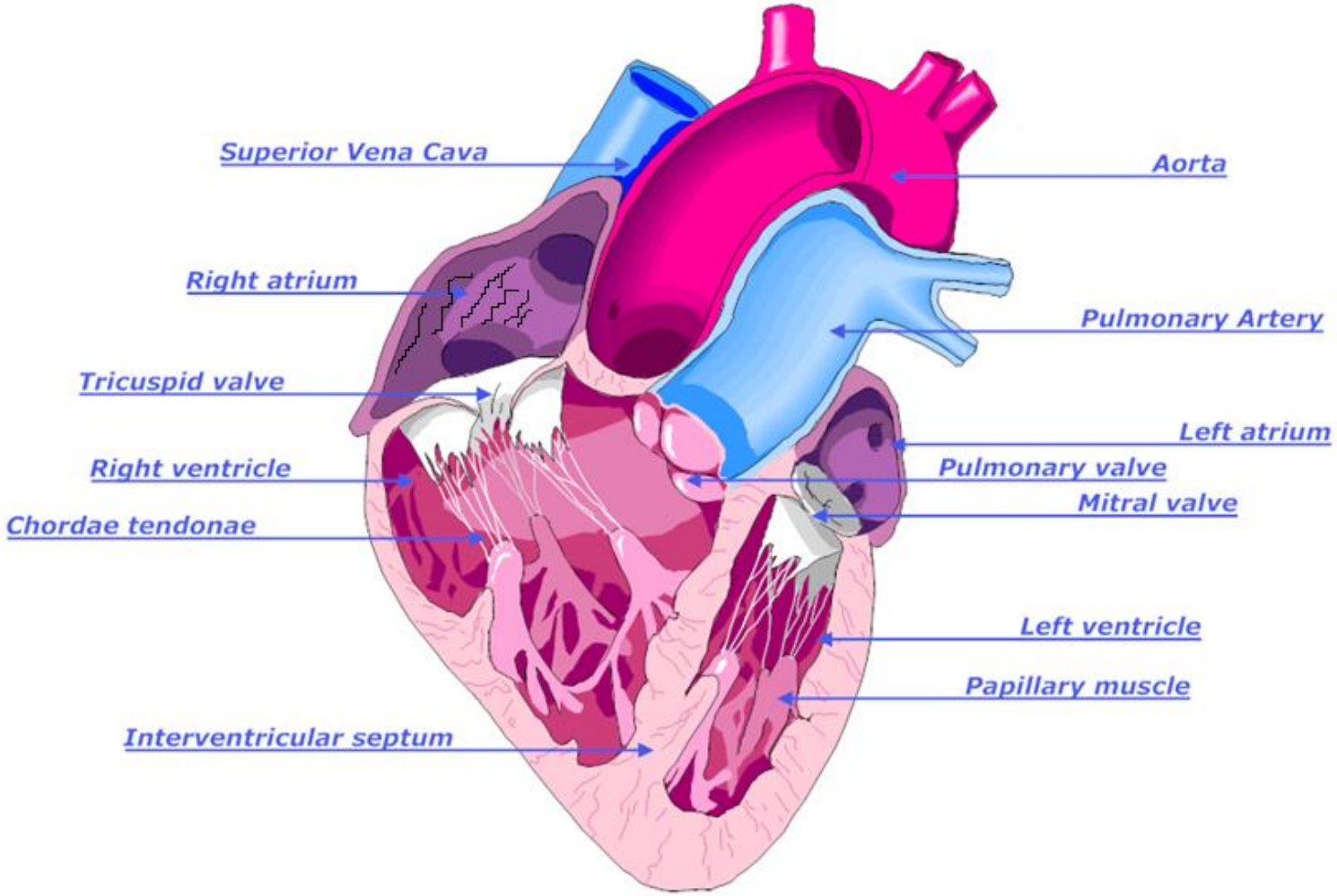
Gross Anatomy – Anterior View



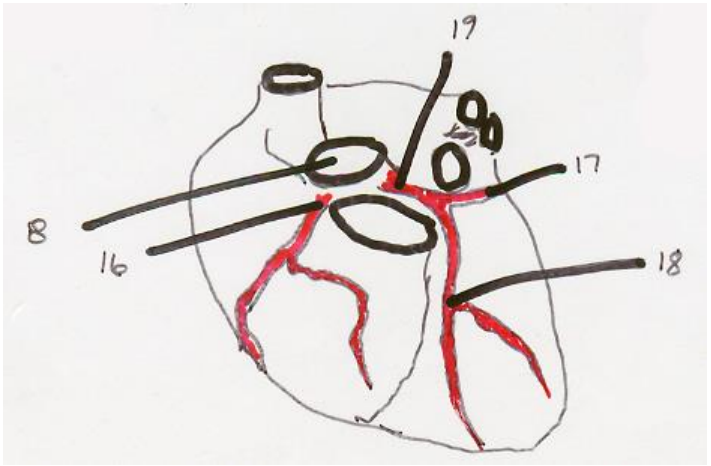
Gross Anatomy – Posterior View

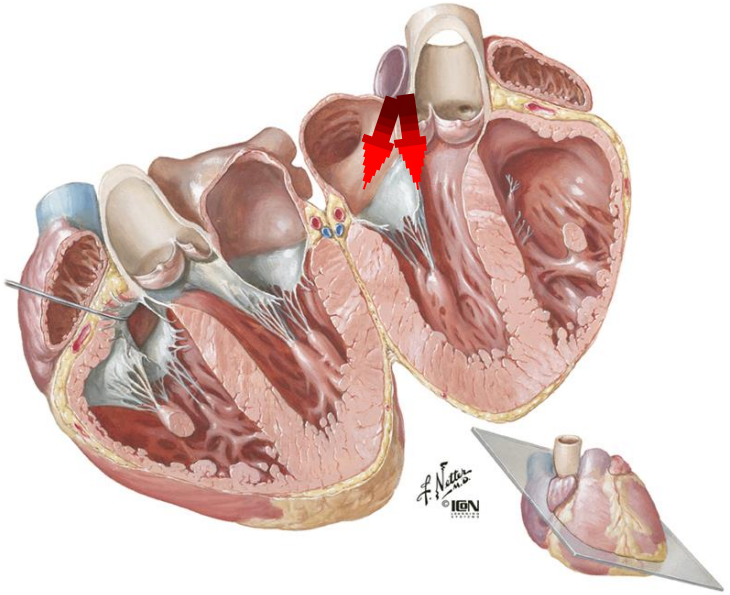


Parts of the Internal Heart

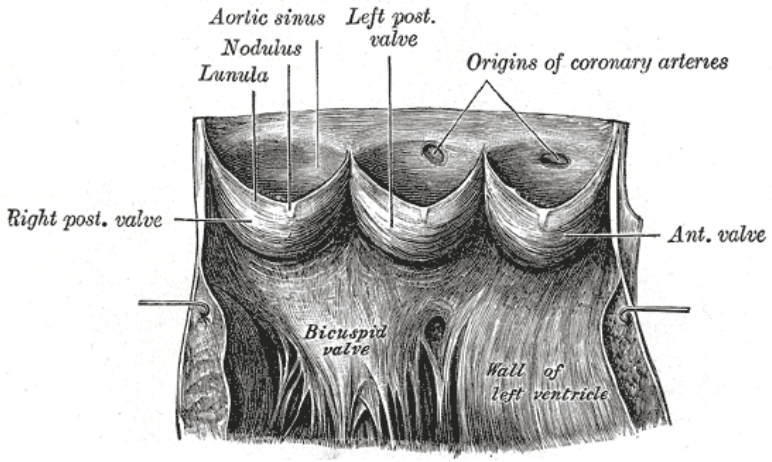


FOCUS: Coronary Arteries' Origins

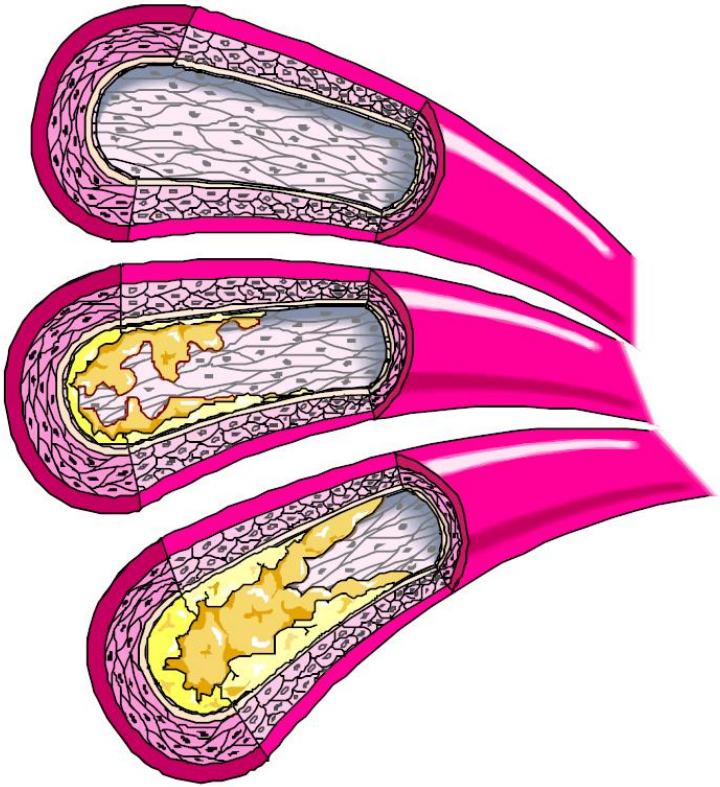


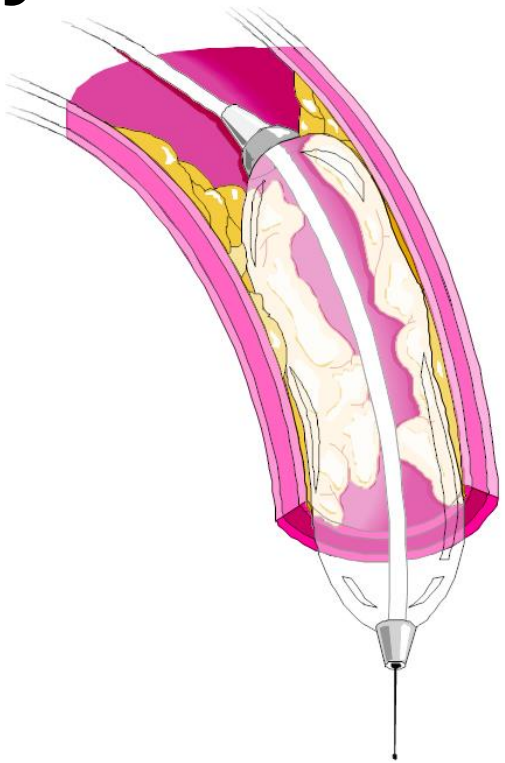


16. RCA
17. CXA
18. LADCA
19. LCA
8. Ascending aorta



Plaque Formation and Balloon Angioplasty

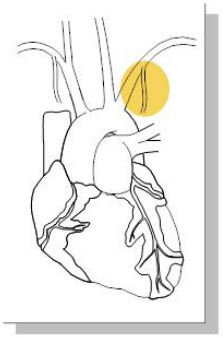


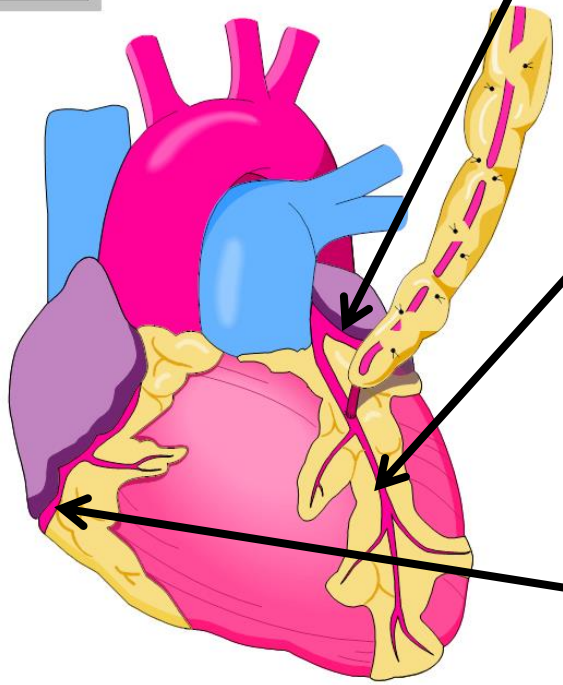


Link for angioplasty with stent placement

Link to angioplasty

Coronary Artery By-pass Graft -- CABG





CXA: blood to LA, lateral wall LV, Inferior/diaphragmatic area of LV

LAD: blood to anterior and apex of LV, anterior 2/3 of septum

RCA: blood to SA node, RA and RV, inferior/diaphragmatic LV

Heart Valves and Heart Sounds

AV Valves and S_1

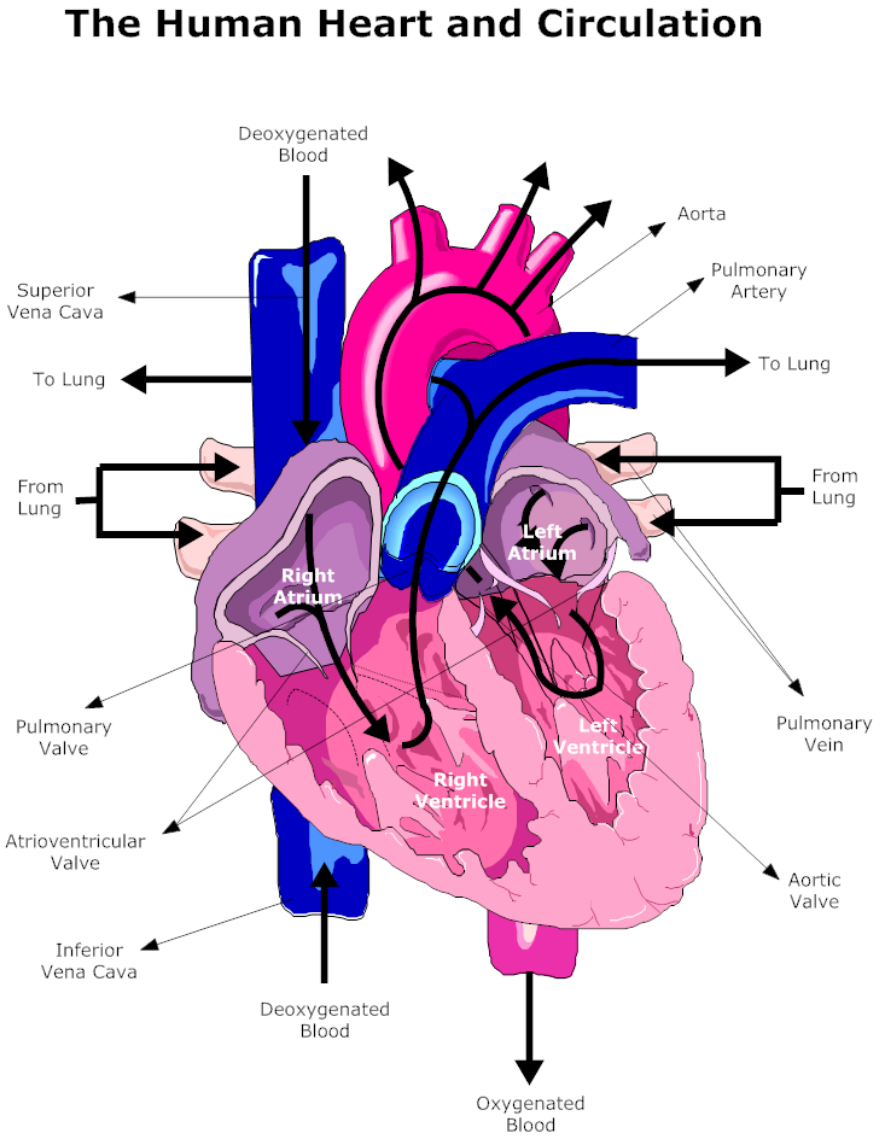
- Ventricles contract and eject blood into pulmonary trunk and aorta
- Blood ALSO sent towards AV valves
- Blood HITS/SLAPS valves; valves press into atria; before valves press all the way into the atria, papillary muscles contract, pull on chordae tendinae which holds valves taut
- Blood leaves ventricles through vessels NOT through AV valves

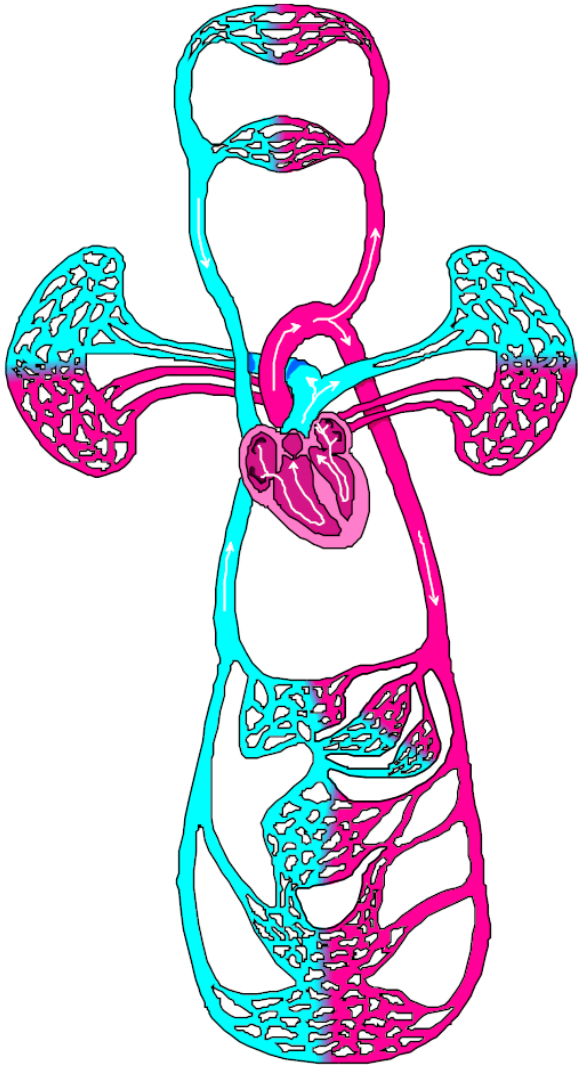
Heart Valves and Heart Sounds

SLV Valves and S_2

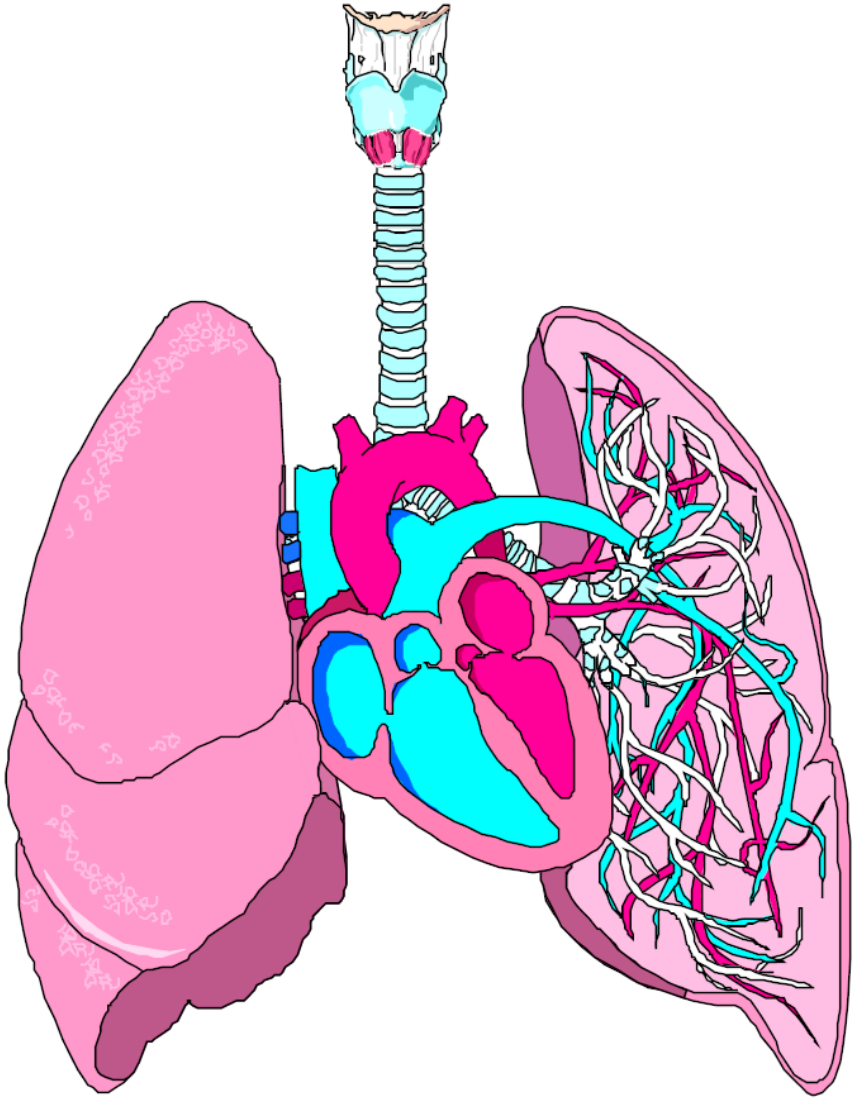
- Ventricles relax and blood falls back through pulmonary trunk and aorta
- Blood slaps back into cusps of SLV valves, filling cusps, closing valves
- Blood goes to lungs and body through vessels NOT through SLV valves back to ventricles
- Concurrently, blood fills the coronary arteries, TOO – RCA and LCA origins are superior to aortic valve

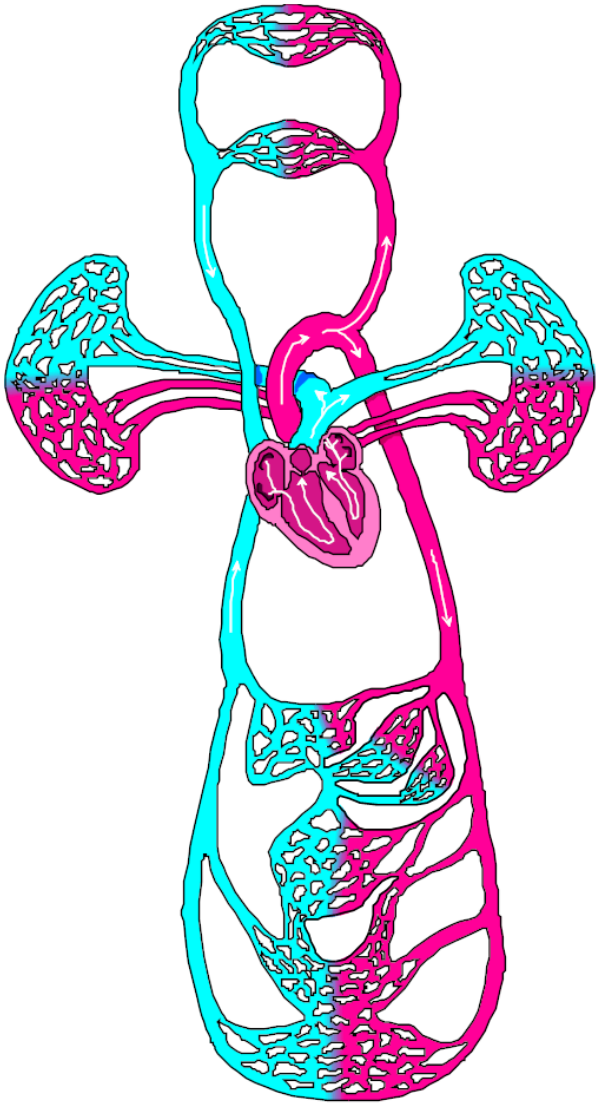
Blood Flow Review





Blood Flow Review -- 2





Heart Murmur

- A “whooshing”, blowing sound heard as blood flows through incompetent valves, i.e., valves that are not closed tightly for one reason or another
- May not necessarily drive heart or valvular disease

Myocardial Contraction -- 1

Isometric Contraction

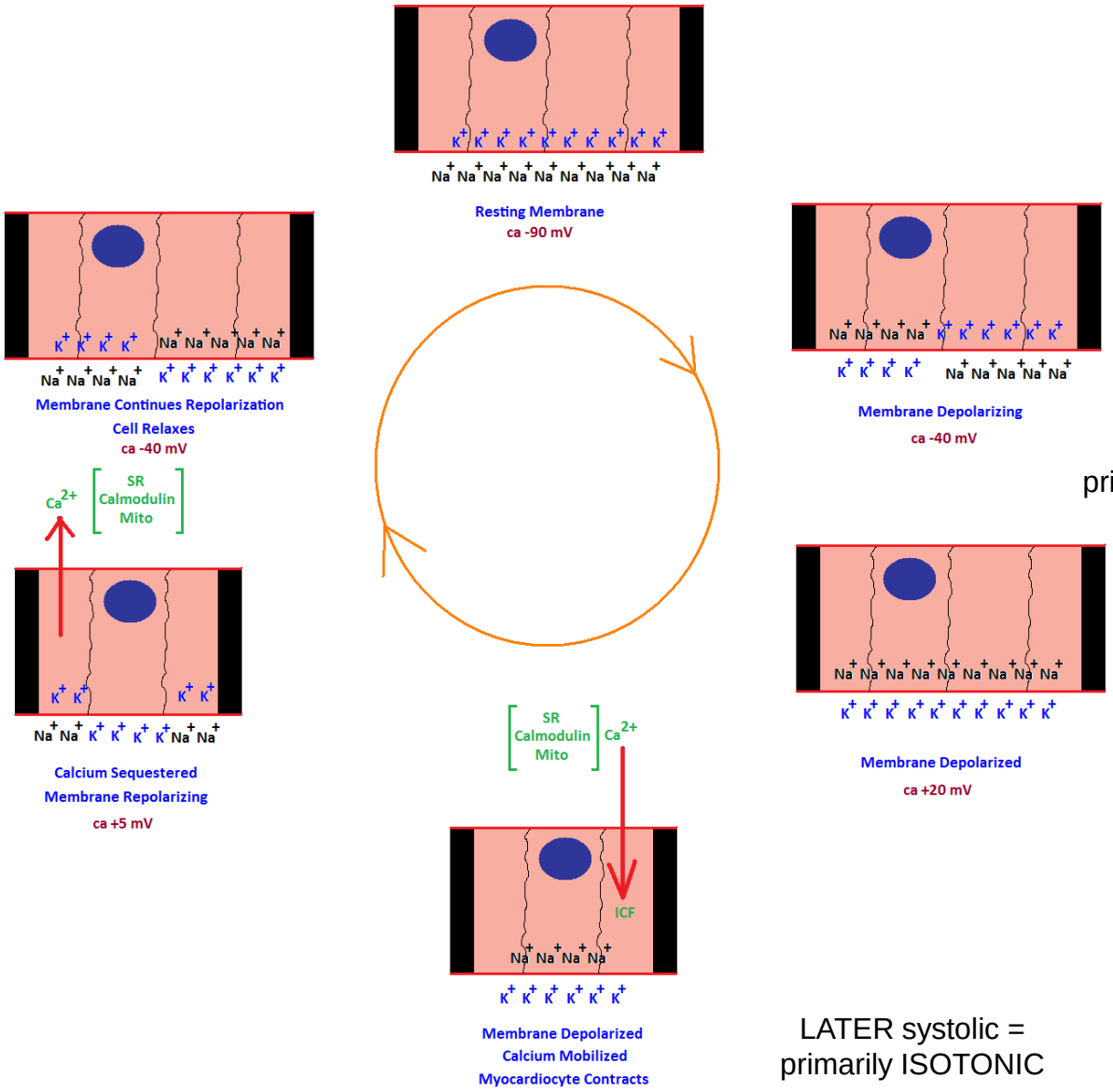
- A muscle is held at a fixed length and increasing tension; no movement occurs, e.g., pushing against a building wall
- E.g., in walking: keeps limb stiff as it touches the ground
- Holding barbed wire fence up for buddies to dive through
- aka STATIC exercise, e.g., sustained hand grip
- Impedes blood flow mechanically: requires increased cardiac output due to increased HR, with a >> increase in BP
- aka Strength/Power Building Exercise

Myocardial Contraction -- 2

Isotonic Contraction

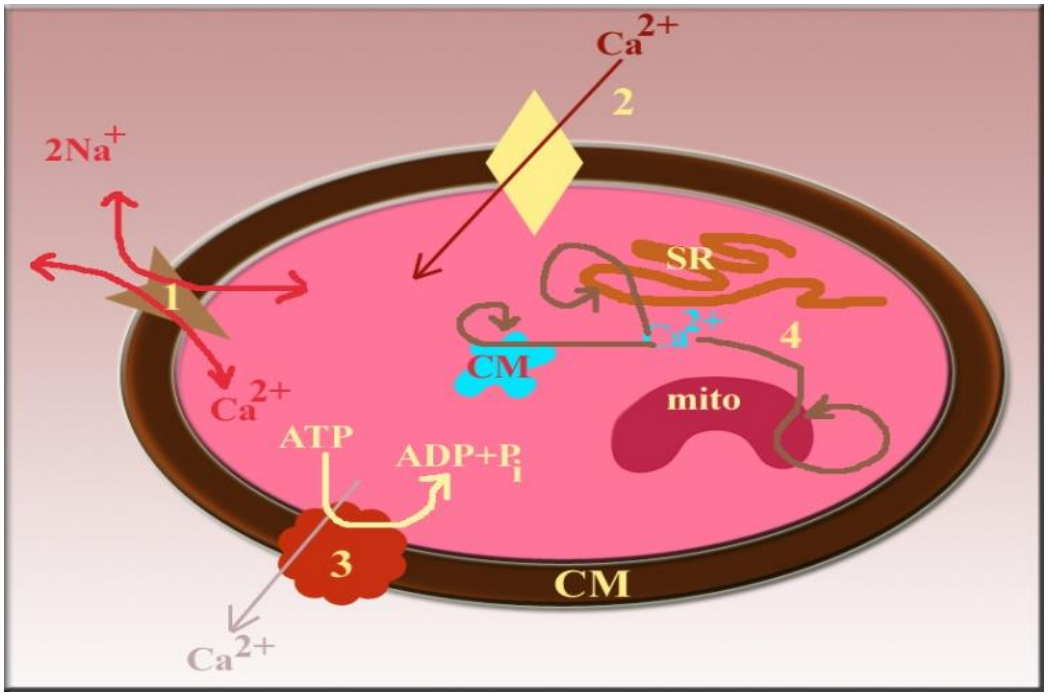
- Shortened stimulated muscle with no increase in tension
- Movement occurs
- E.g., in walking: causes leg to bend and lift upward
- Lifting an object with movement

Electrical Events During Myocardial Contraction/Relaxation



EARLY systole =
primarily ISOMETRIC

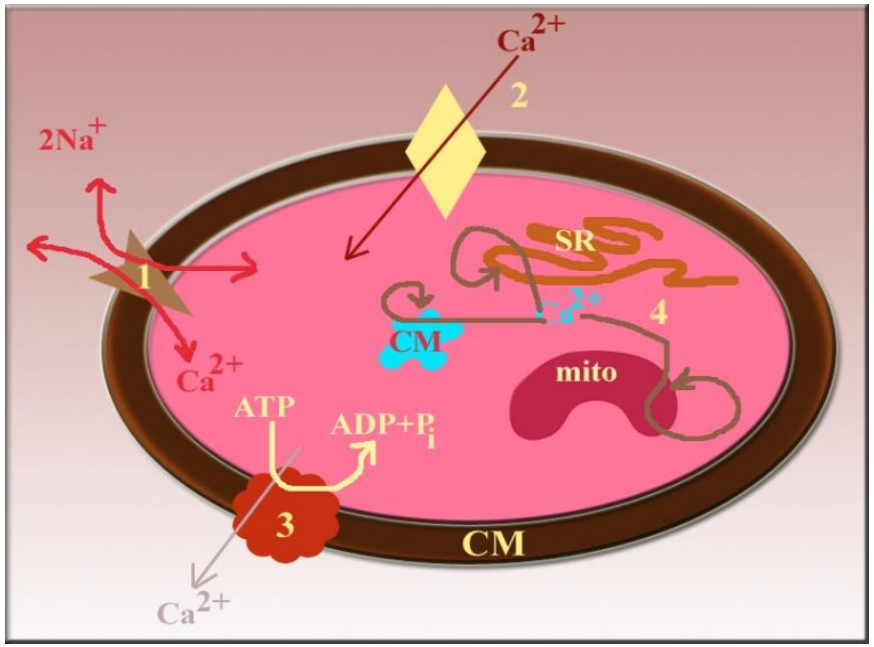
LATER systolic =
primarily ISOTONIC



Sodium:Calcium Exchange: 1 (See “1” in the graphic; “CM” = cell membrane; “mito” = mitochondrion; “CM” = calmodulin; “SR” = sarcoplasmic reticulum) -- Requires two sodium ions to go the opposite direction for every calcium ion that goes into or out of the cell, respectively.

Calcium:ATP’ase Efflux: 3 -- an energy driven mechanism (at the expense of ATP) that removes calcium ions from our cells.

Calcium Sequestration: 4 -- an intracellular mechanism by which our cells sequester calcium ions by "tying" them up in the mitochondrion of the cell, the sarcoplasmic reticulum or by calmodulin.



•Receptor Mediated Calcium Ion Influx Mechanism: 2 -- The calcium ion channel we <u>can</u> control is the receptor-mediated calcium ion influx mechanism.

•We can turn this channel off using calcium ion channel blockers such as verapamil (Calan or Isoptin) which effects both smooth and cardiac muscle, diltiazem (Cardizem) which effects both smooth and cardiac muscle or nifedipine (Procardia) which effects smooth muscle.

•We can turn this channel on, as well, with drugs like nitrendipine (lowers blood pressure), nimodipine (Nimotop; causes cerebrovascular dilation) or amlodipine (Norvasc; lowers blood pressure).

Grey thick bars are the intercalated disks;

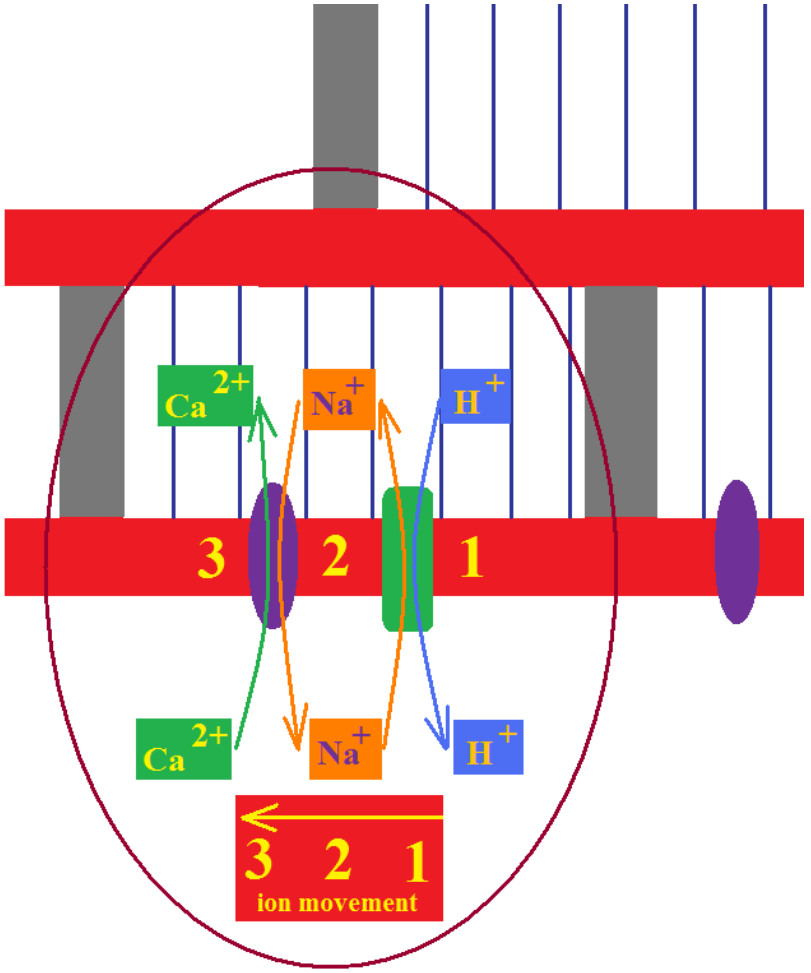
thin blue bars are the striations found in myocardiocytes;

red thick bars represent the cell membrane;

purple oval a Na^+ -- Ca^{2+} exchange transport protein;

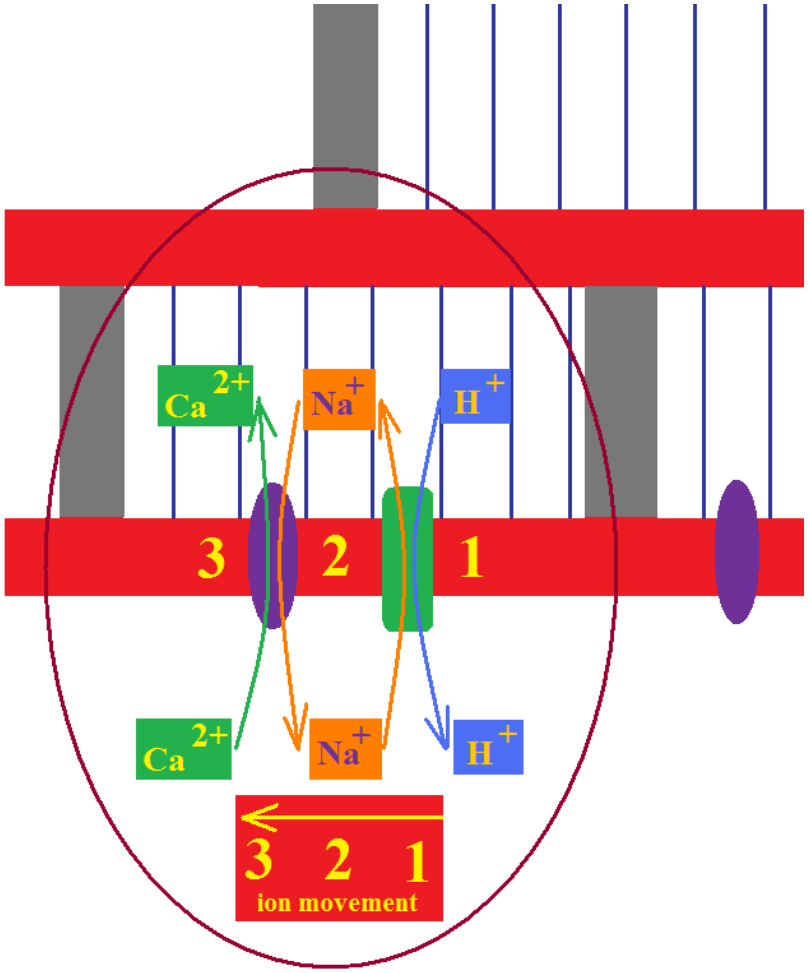
green rectangle a H^+ -- Na^+ transport protein;

numbers in yellow indicate the direction of ionic transport

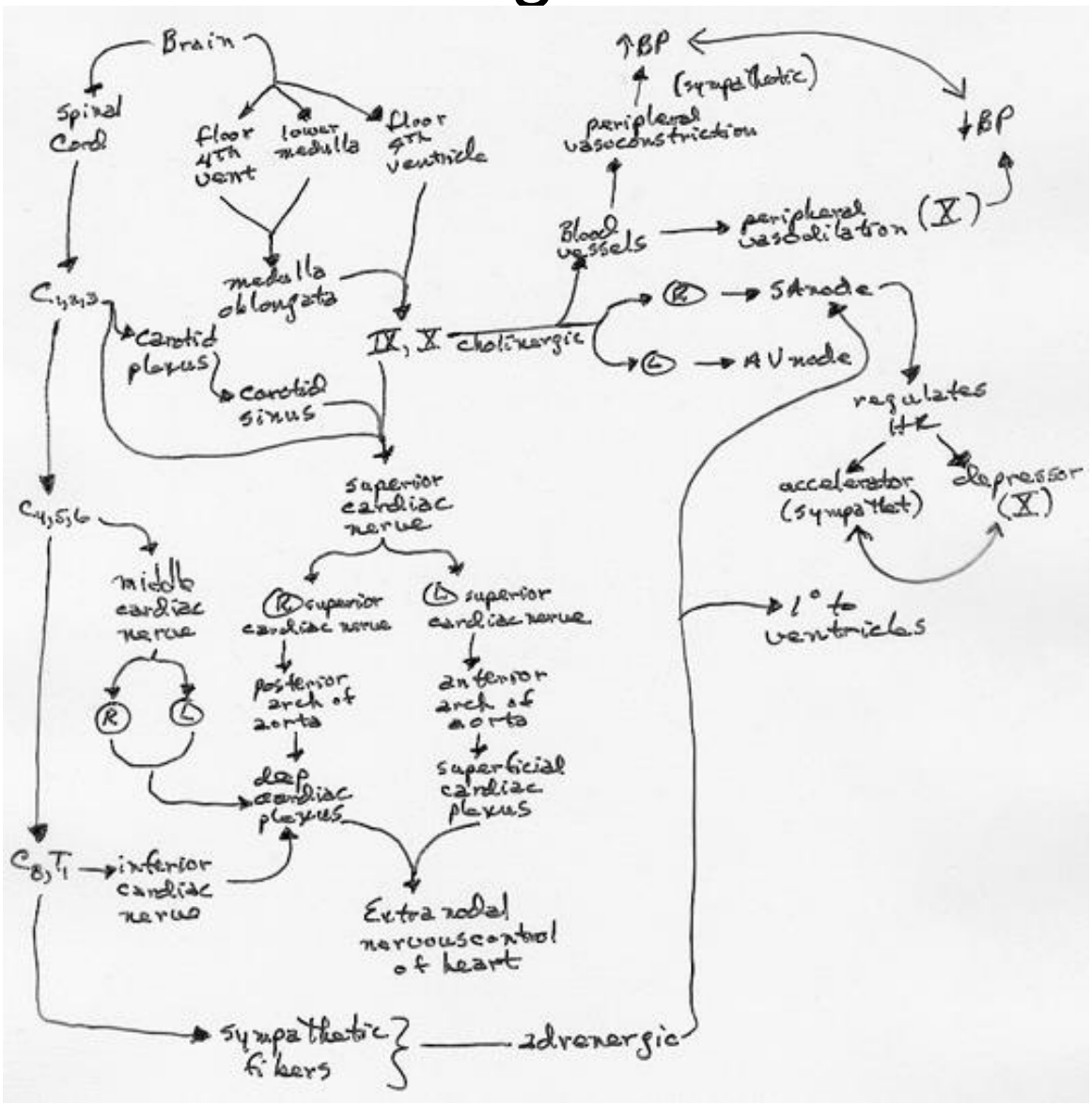


One of the biggest concerns clinicians have regarding heart health during a myocardial infarction (MI; heart attack) is that the [H^+] may increase due to a build-up (and dissociation) of lactate and/or fatty acids, which may contribute to a metabolic acidosis in the heart muscle, which will thus kill more and more heart muscle.

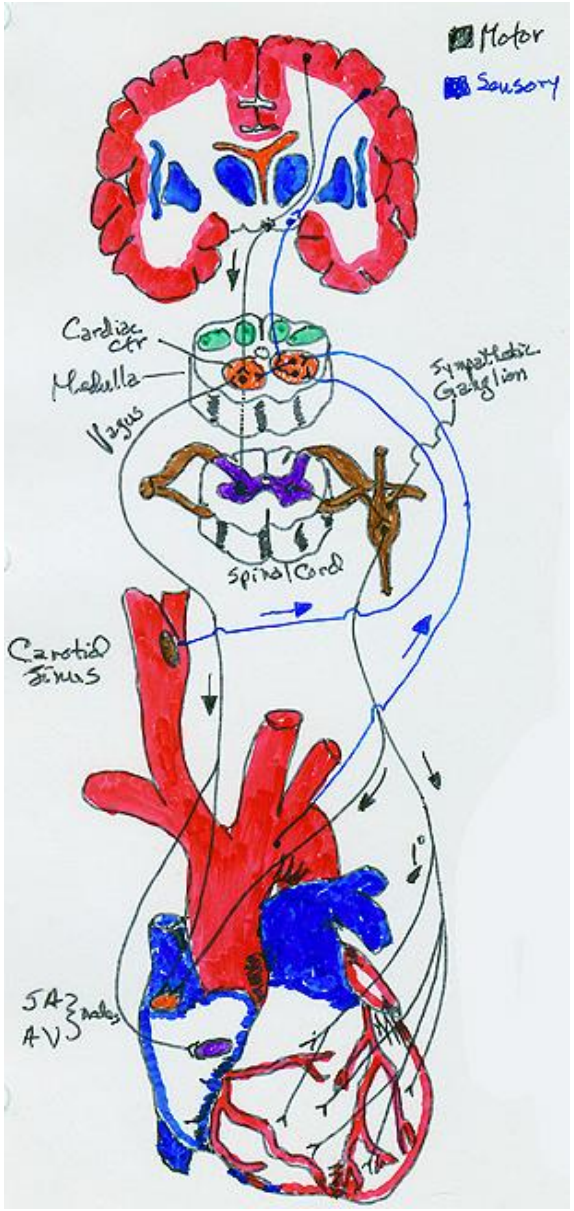
This process is rendered even more critical in that as the H^+ are exchanged OUT of the cardiac cells to compensate for the intracellular metabolic acidosis, Na^+ and Ca^{2+} exchange occurs leading to excessively high levels of Ca^{2+} in the cells which may progress to further cell, and, hence, organ, death.



Cardiac "Circuit Diagram" -- Homeostasis



Cardiac Circuitry



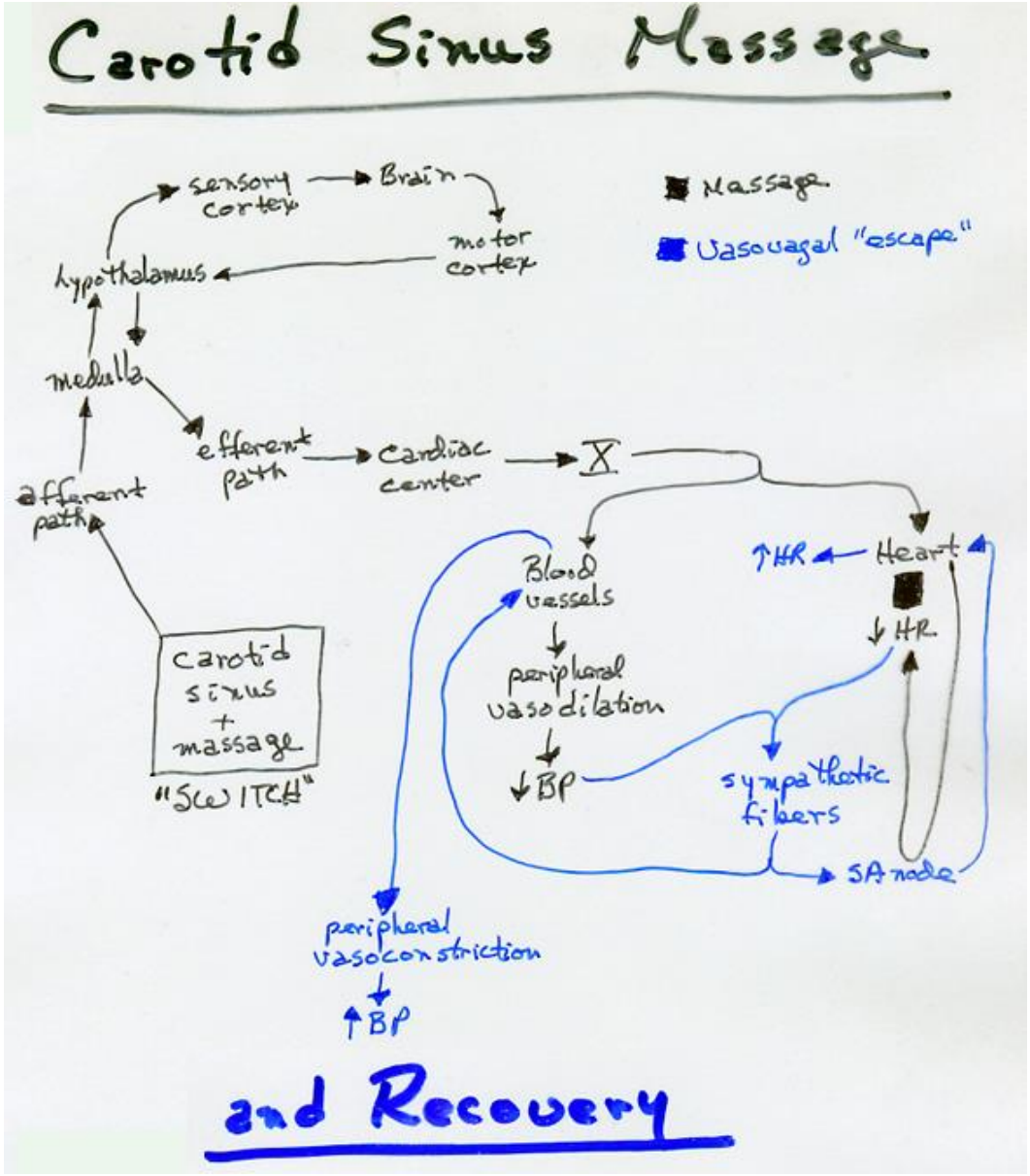
Carotid Sinus Massage

1. 5-10 seconds
2. Unilaterally
3. Patient must be supine
4. When no bruit is present!!! (bruit: murmurs heard best over carotid bifurcation; not of cardiac origin; caused by partial obstruction of the carotid)
5. Use an EKG and obtain BP
6. Pt must have no hx of TIA ($\rightarrow$ >$\uparrow$'d risk of CVA)

Causes Vasovagal Response

1. Vasodepressor response (BP reduced by $\geq$50 mm Hg)
2. Cardioinhibitory response ($\downarrow$ HR by $\geq$ 3 second sinus pause)

Carotid Massage Mechanism



- Carotid Sinus Massage not used much, any more – if at all.
- Periodically, one will run across its use in the literature or online
- Adenosine used now

Carotid Sinus Syncope

- Syncope ≡ is temporary loss of consciousness and posture, described as "fainting" or "passing out." It's usually related to temporary insufficient blood flow to the brain.
- Another way to define it is that of the room spinning around you.
- Of Cardiac origin

- Vertigo ≡ a sensation of spinning [around the room or wherever you may be].
- Of Neurological origin

Vasovagal Response

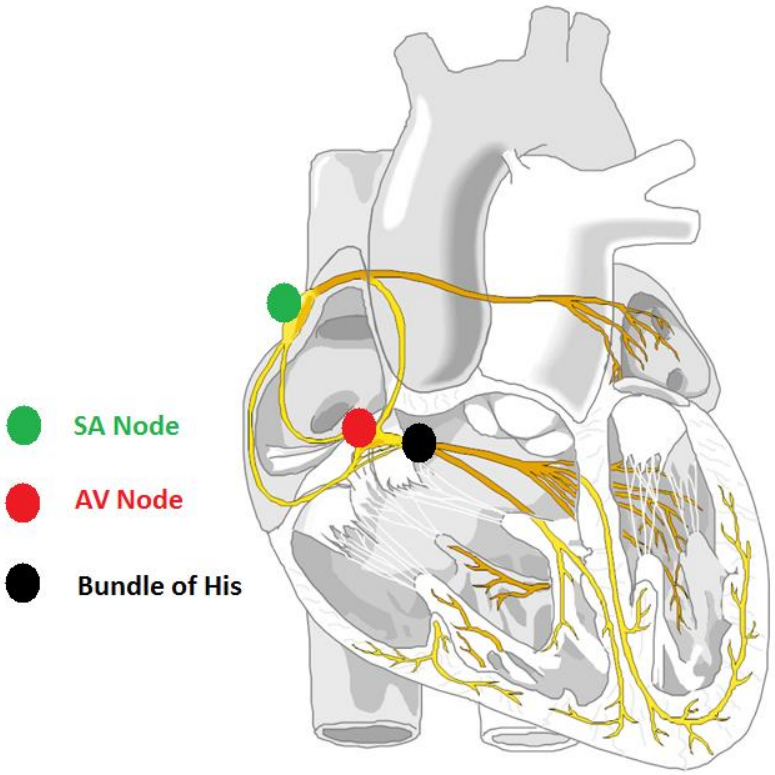
- A vasovagal episode or vasovagal response or vasovagal attack (also called neurocardiogenic syncope) is mediated by the vagus nerve. When it leads to syncope or "fainting", it is called vasovagal syncope, which is the most common type of fainting.

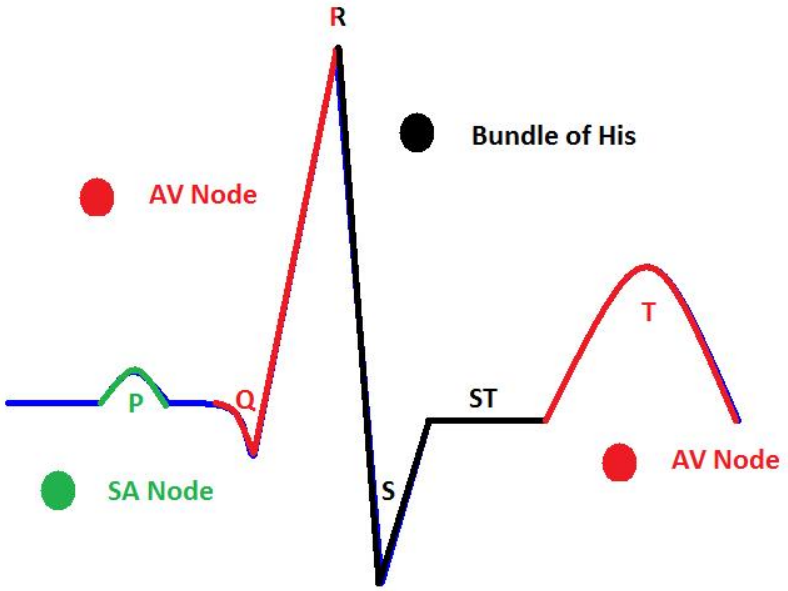
- Prior to losing consciousness, the individual frequently experiences a prodrome of symptoms such as lightheadedness, nausea, diaphoresis, tinnitus, uncomfortable feeling in the heart, weakness and visual disturbances such as lights seeming too bright, fuzzy or tunnel vision.
- These last for at least a few seconds before consciousness is lost (if it is lost), which typically happens when the person is sitting up or standing. When sufferers pass out, they fall down (unless this is impeded); and when in this position, effective blood flow to the brain is immediately restored, allowing the person to wake up.
- Tabor's describes this as the "feeling of impending death" caused by expansion of the aorta, drawing blood from the head and upper body.

Carotid Sinus Syncope

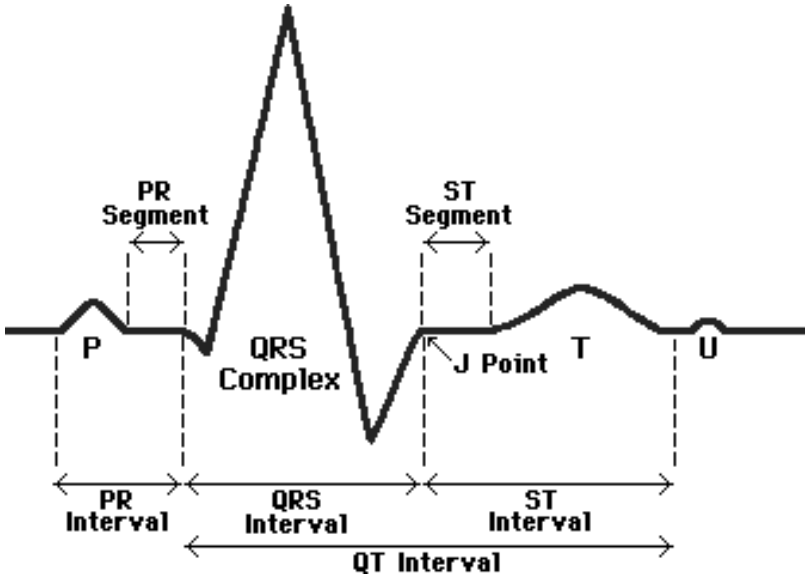
- If chronic and due to cardioinhibitory response (head turned, tight shirt collar), is “fixable” with permanent pacing
- Other tx ≡ surgical removal, by stripping, of nerves from the carotid artery above and below the bifurcation

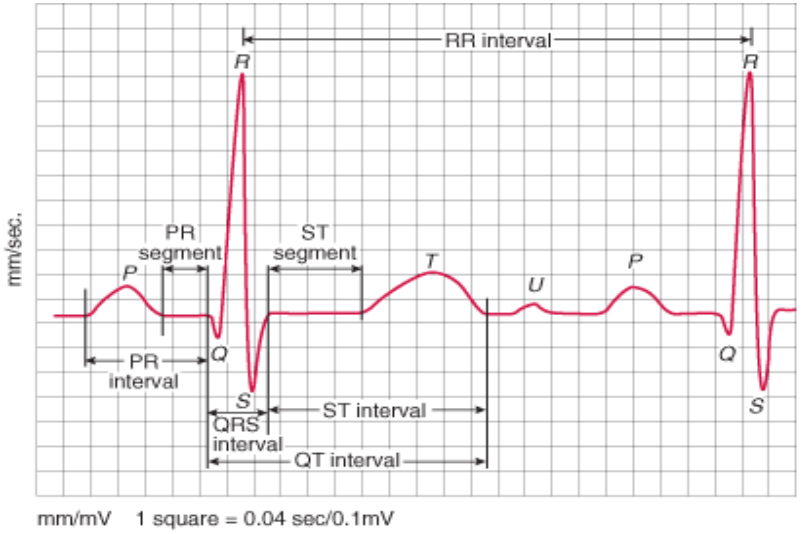
Electrical System of the Heart -- 1





Overview of Normal Cardiac Cycle EKG Complex





- P wave = atrial contraction
- Q-R = AV node
- R-S = Bundle of His
  - QRS = ventricular depolarization
- ST = artifact
- T wave = ventricular repolarization
- U waves are thought to represent repolarization of the papillary muscles or Purkinje fibers; normally seen in younger, athletic individuals.

Long QT Syndrome

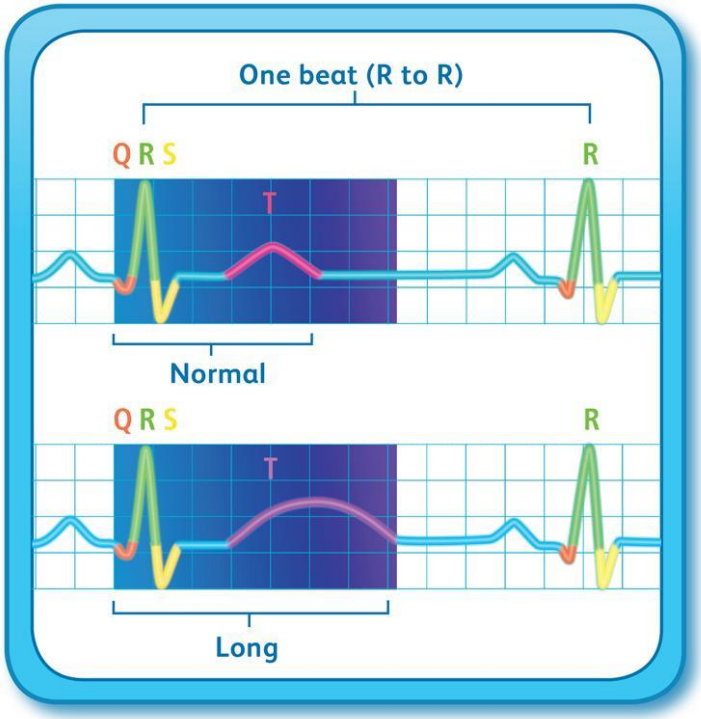


Illustration showing prolonged QT interval on an electrocardiogram (ECG)

http://www.genedx.com/wp-content/uploads/2010/12/LQT.jpg

The electrical activity that occurs between the Q and T waves is called the QT interval. This interval shows electrical activity in the heart's lower chambers, the ventricles.

The term "long QT" refers to an abnormal pattern seen on an EKG (electrocardiogram).

The timing of the heart's electrical activity is complex, and the body carefully controls it. Normally the QT interval is about a third of each heartbeat cycle. However, in people who have LQTS, the QT interval lasts longer than normal.

A long QT interval can upset the careful timing of the heartbeat and trigger dangerous heart rhythms.

Long QT Syndrome, Cont'd

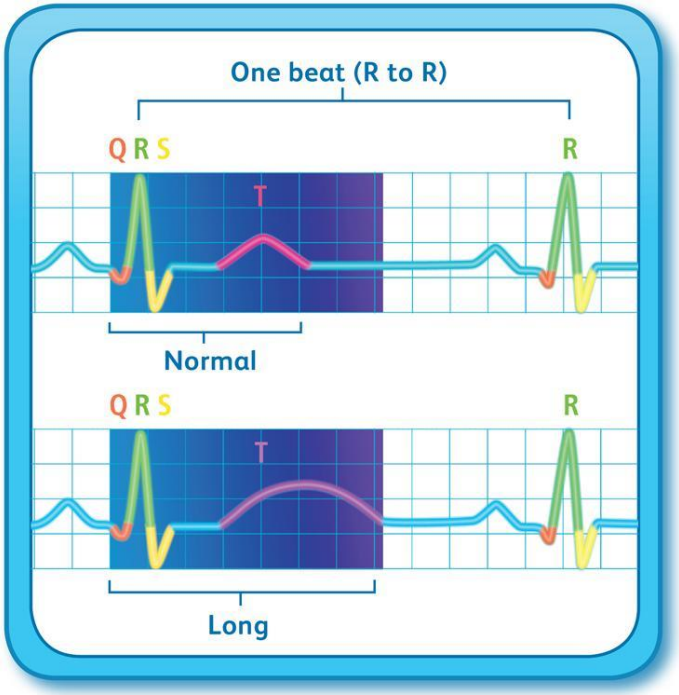


Illustration showing prolonged QT interval on an electrocardiogram (ECG)

http://www.genedx.com/wp-content/uploads/2010/12/LQT.jpg

On the surface of each heart muscle cell are ion channels.

Ion channels open and close to let electrically charged sodium, calcium, and potassium atoms (ions) flow into and out of each cell.

This generates the heart's electrical activity.

In people who have LQTS, the ion channels may not work well, or there may be too few of them. This may disrupt electrical activity in the heart's ventricles and cause dangerous arrhythmias.

Long QT Syndrome, Cont'd

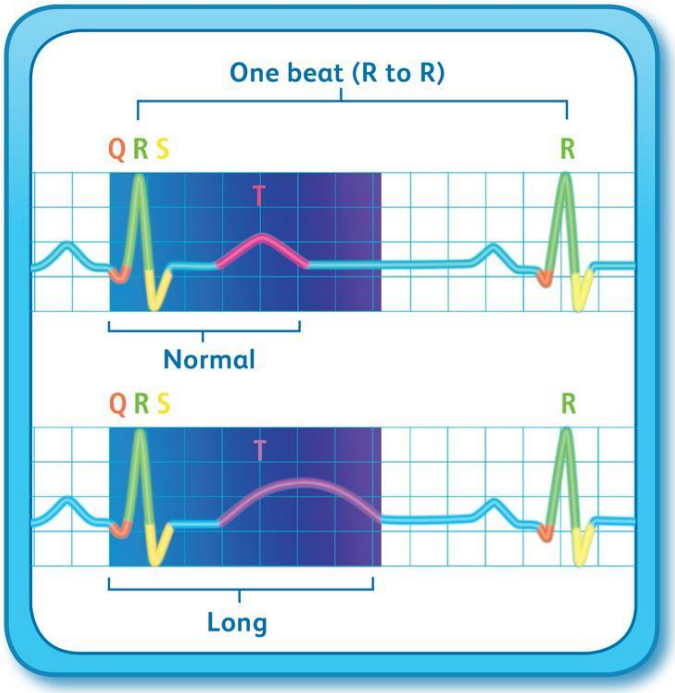


Illustration showing prolonged QT interval on an electrocardiogram (ECG)

http://www.genedx.com/wp-content/uploads/2010/12/LQT.jpg

- LQTS often is inherited, which means you're born with the condition and have it your whole life. There are seven known types of inherited LQTS. The most common ones are LQTS 1, 2, and 3.
- In LQTS 1, emotional stress or exercise (especially swimming) can trigger arrhythmias.
- In LQTS 2, extreme emotions, such as surprise, can trigger arrhythmias.
- In LQTS 3, a slow heart rate during sleep can trigger arrhythmias.
- You also can acquire LQTS: you develop it during your lifetime. Some medicines and conditions can cause acquired LQTS.

http://www.nhlbi.nih.gov/health/health-topics/topics/qt/

Long QT Syndrome, Cont'd

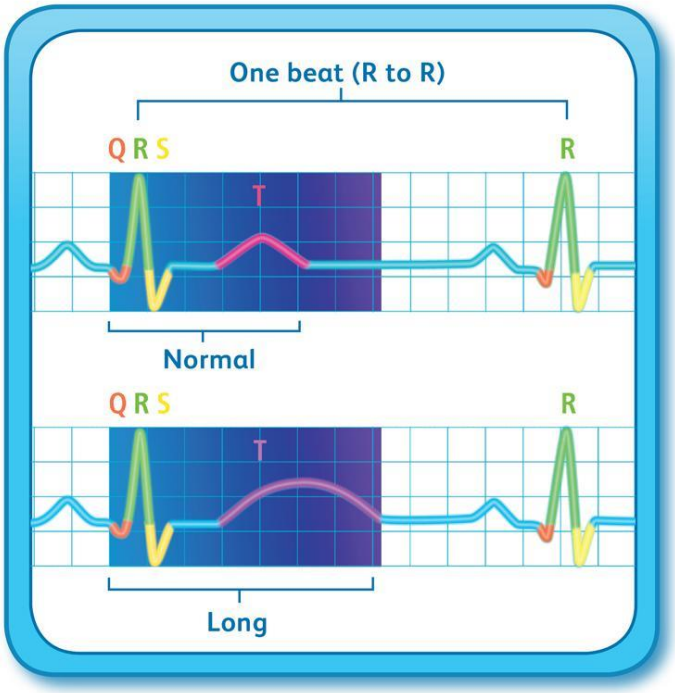


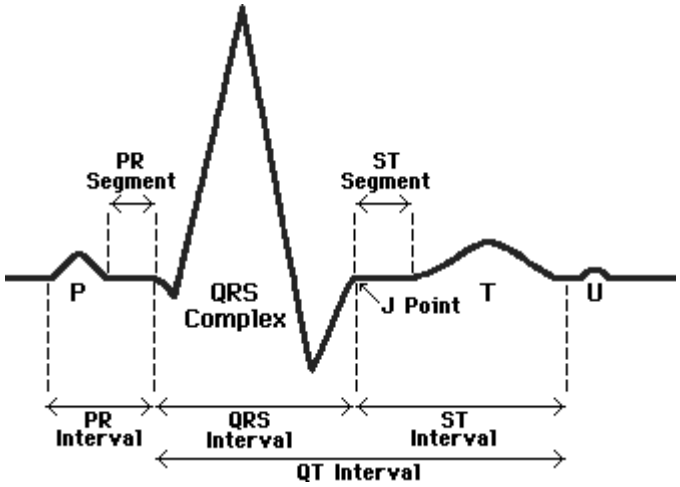
Illustration showing prolonged QT interval on an electrocardiogram (ECG)

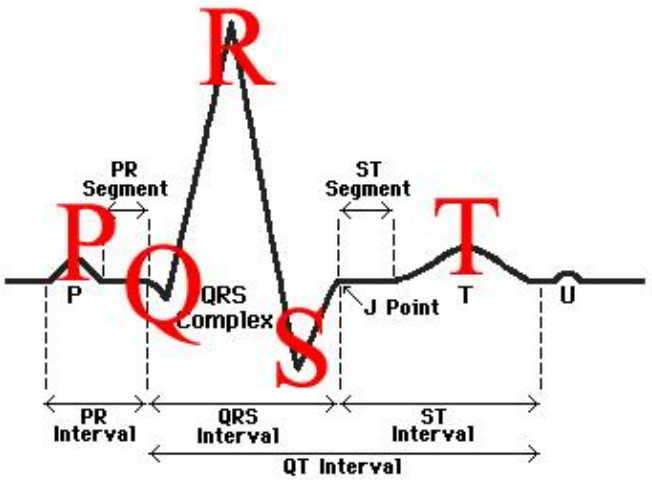
http://www.genedx.com/wp-content/uploads/2010/12/LQT.jpg

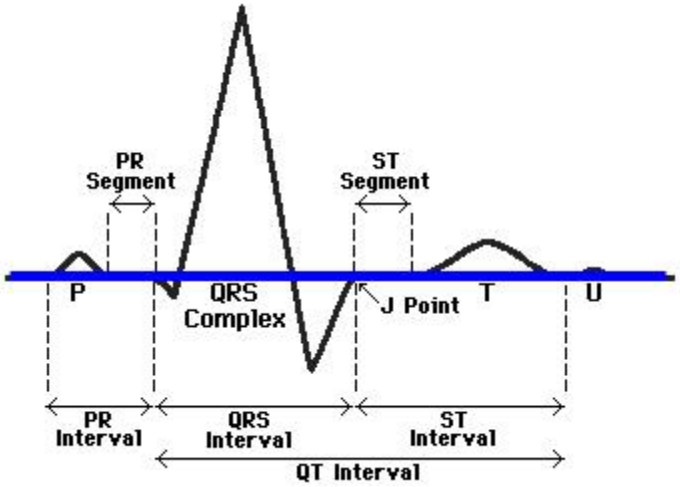
- Lifestyle changes and medicines can help people who have LQTS prevent complications and live longer.
- Some of these lifestyle changes and treatments include:
  - Avoiding strenuous physical activity or startling noises.
  - Adding more potassium to your diet (as your physician advises).
  - Taking heart medicines called beta blockers. These medicines help prevent sudden cardiac arrest.
  - Having an implanted medical device, such as a pacemaker or implantable cardioverter defibrillator. These devices help control abnormal heart rhythms.

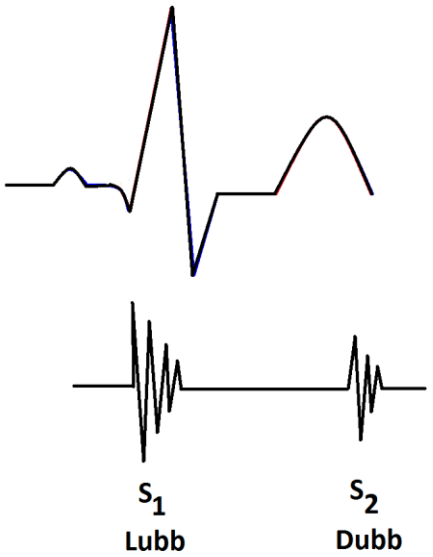
http://www.nhlbi.nih.gov/health/health-topics/topics/qt/

Isoelectric Line & Correlation of EKG with Heart Sounds

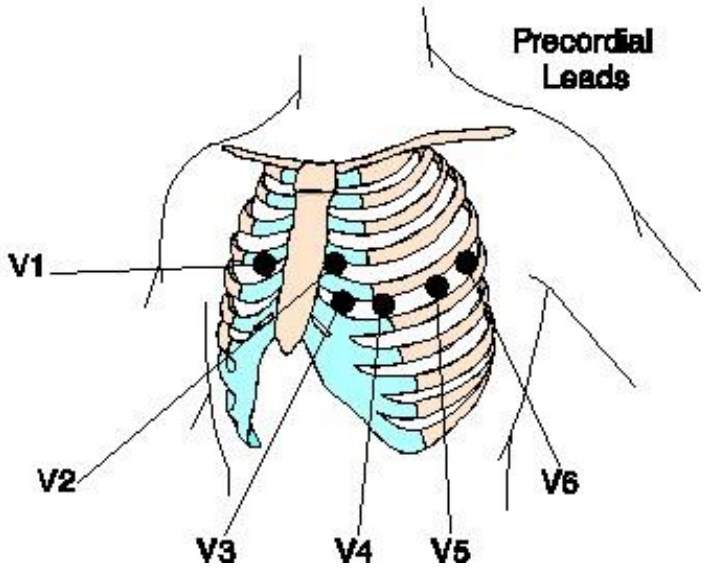


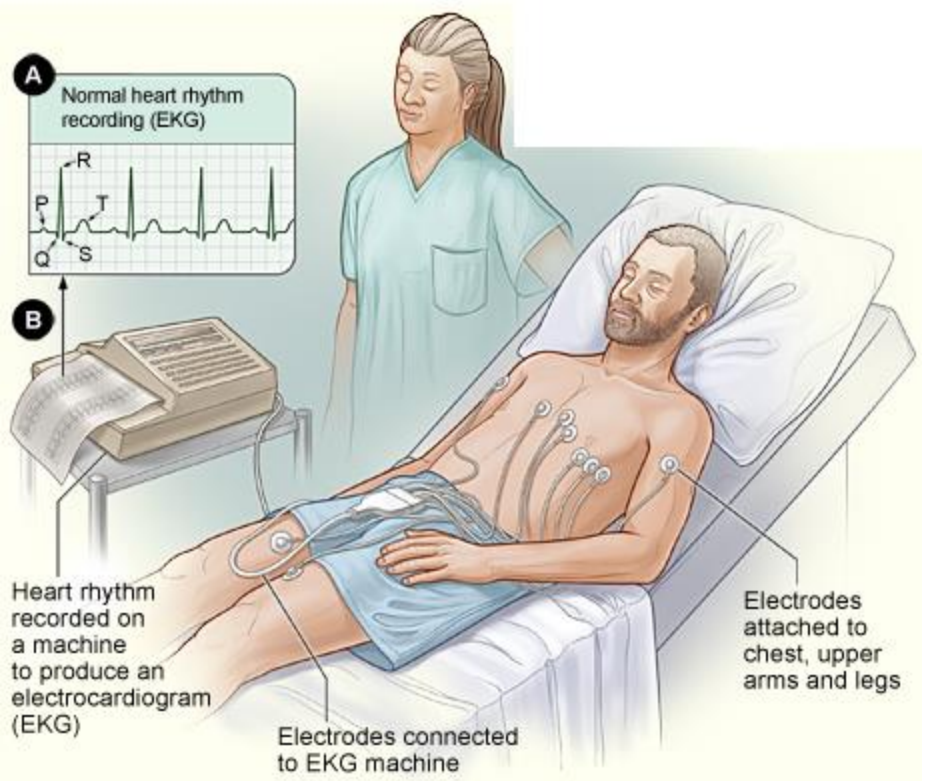






EKG Electrodes -- Organization





http://www.nhlbi.nih.gov/health/health-topics/topics/qt/

EKG Electrodes -- Organization

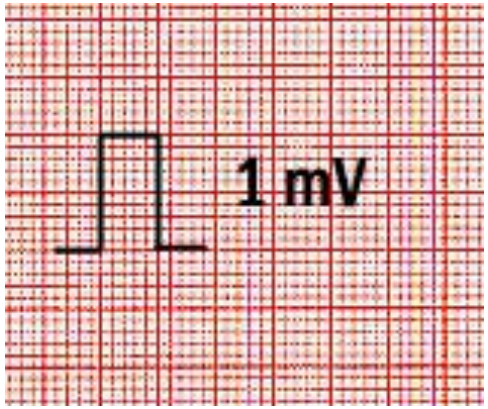
- **2 electrodes on each calf – medial gastroc, fleshy, away from bone**
- **2 electrodes on each forearm – fleshy, away from bone**
- **6 electrodes on chest (for 12 lead EKG):**
- **V_1 – 4th intercostal space, right sternal border**
- **V_2 – 4th intercostal space, left sternal border**
- **V_3 – half way between V_2 and V_4**
- **V_4 – 5th intercostal space, mid-clavicular line (MCL)**
- **V_5 – 5th intercostal space, anterior axillary line**
- **V_6 – 5th intercostal space, left mid-axillary line**
- **In women with large breasts, move the breasts for electrode placement as necessary**
- **Men: ALWAYS take a female employee (e.g., RN, CNA, LPN) with you when doing an EKG on a female patient**
- **10 electrodes give 12 leads**

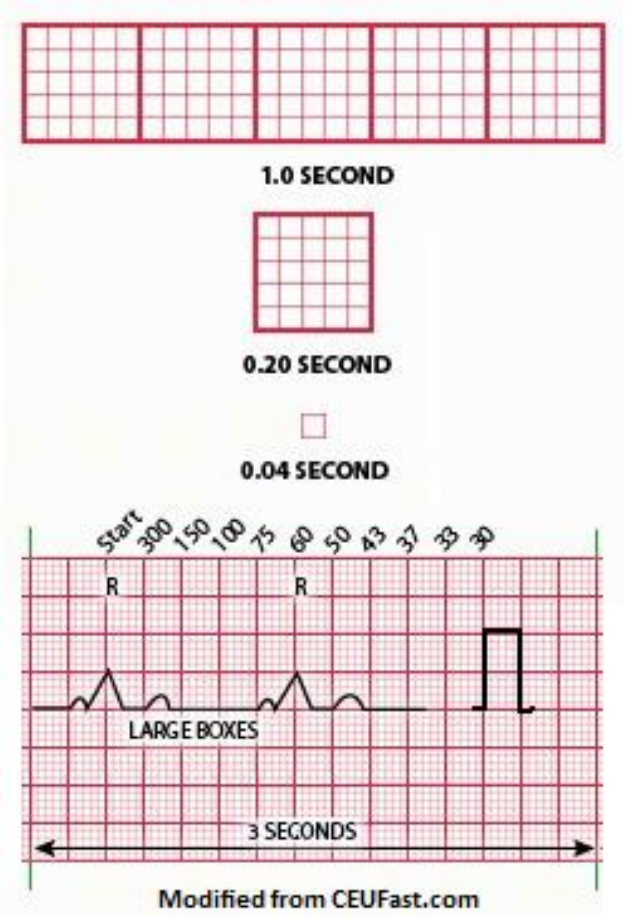
Electrocardiographic Primer

- Representative Arrhythmia Strips
- Infarcts
- Axis Determination
- Bundle Branch Blocks
- Atrial and Ventricular Hypertrophies

- This will NOT turn you into a cardiologist over night – it WILL prepare you for your future career path comfortably.

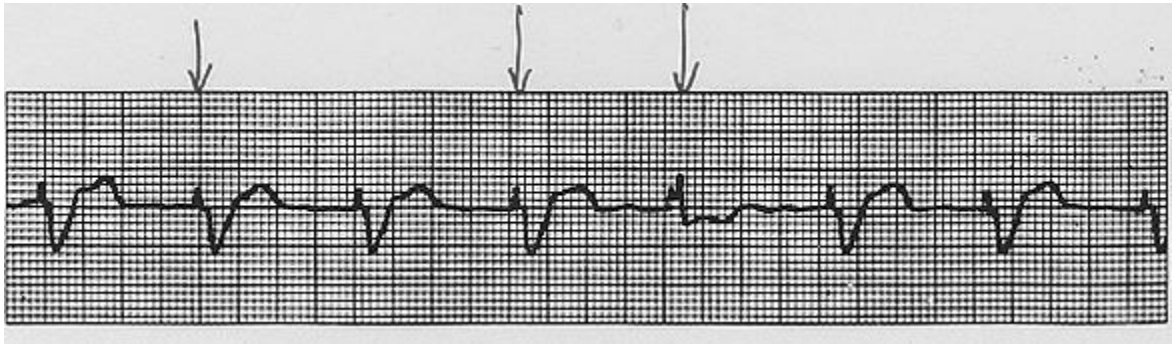
EKG Standard and Heart Rate (Rule of 300)





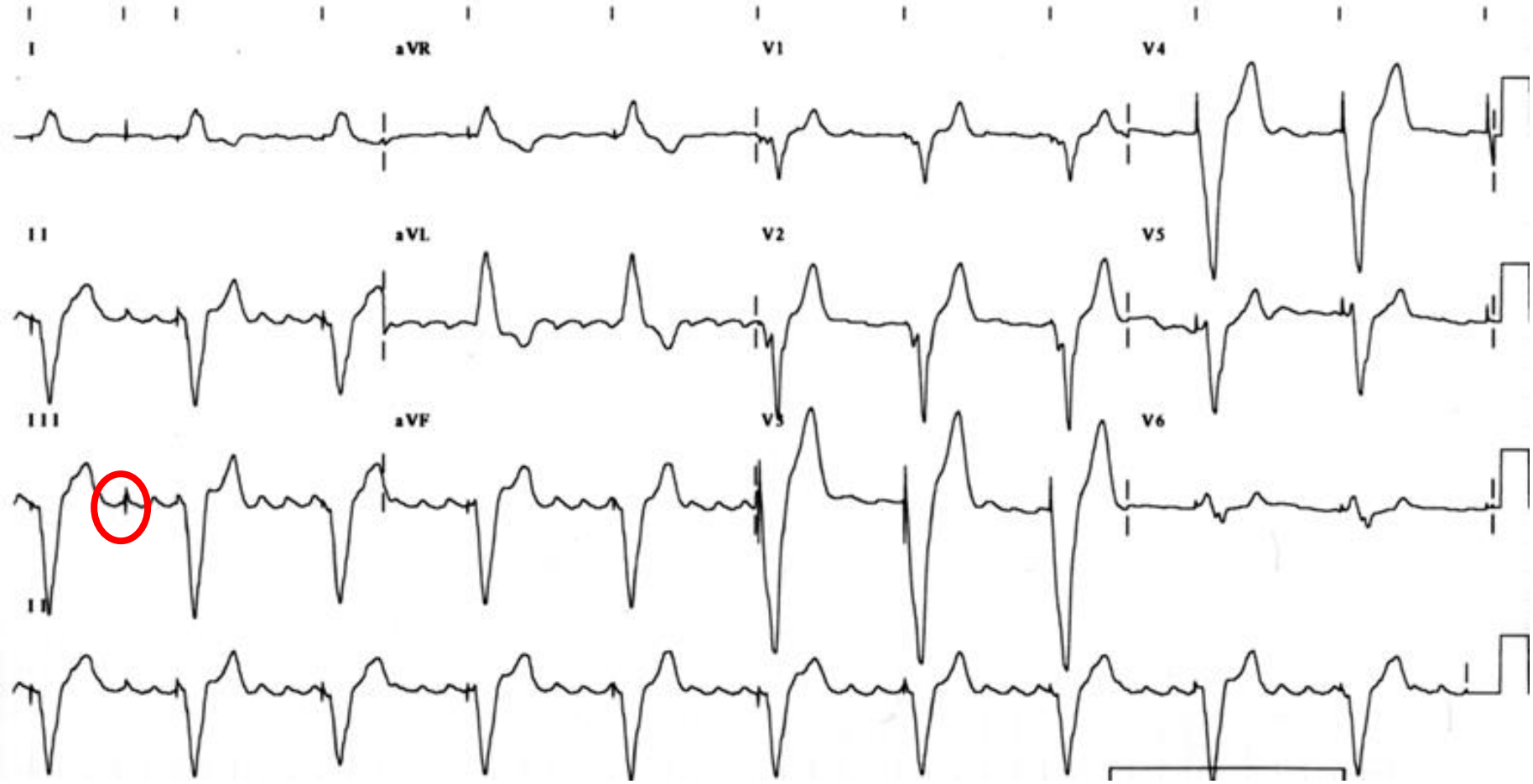
- Standard on an EKG = 2 big squares = 1 mV

Pacemaker artifacts -- 1

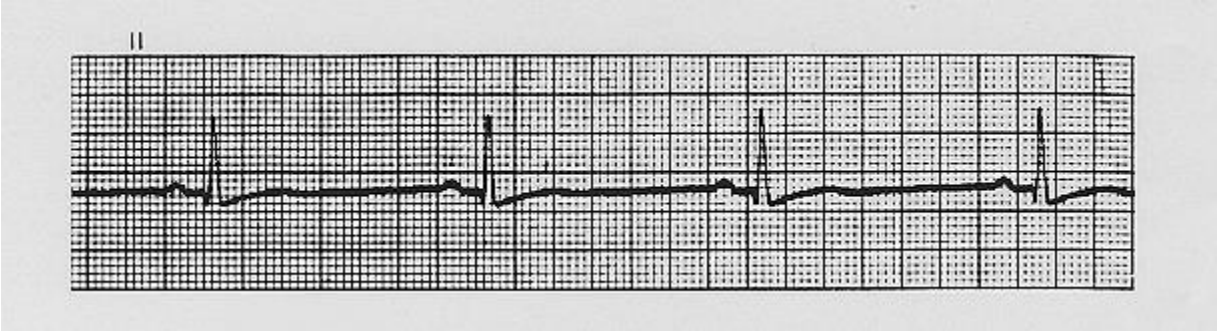


- Pacers “click” up, down or both
- Fifth complex is the patient’s own QRS Complex

Pacemaker artifacts -- 2

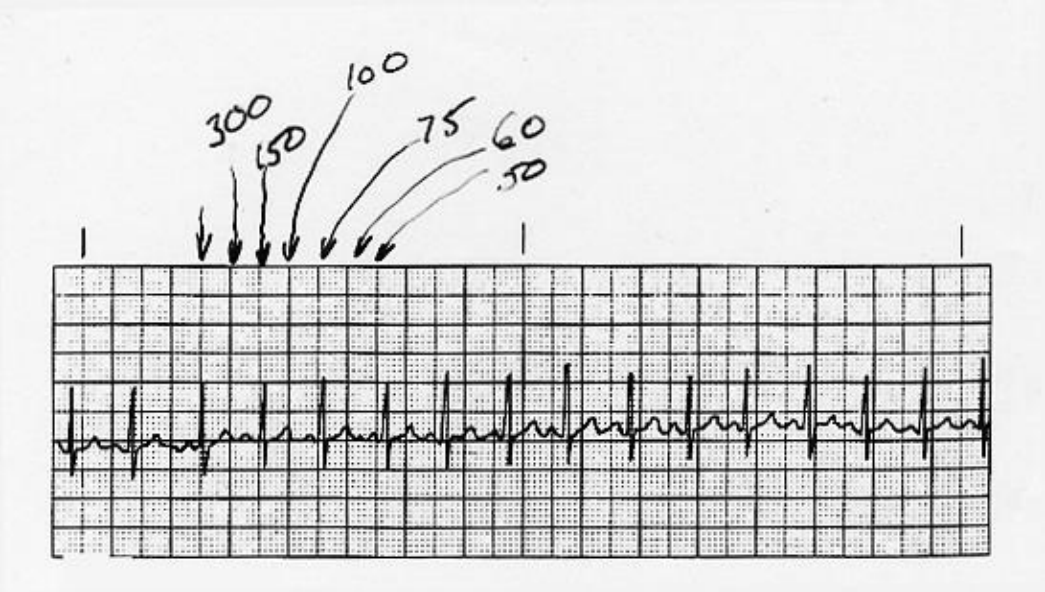


Normal Sinus Rhythm (NSR); Bradycardia



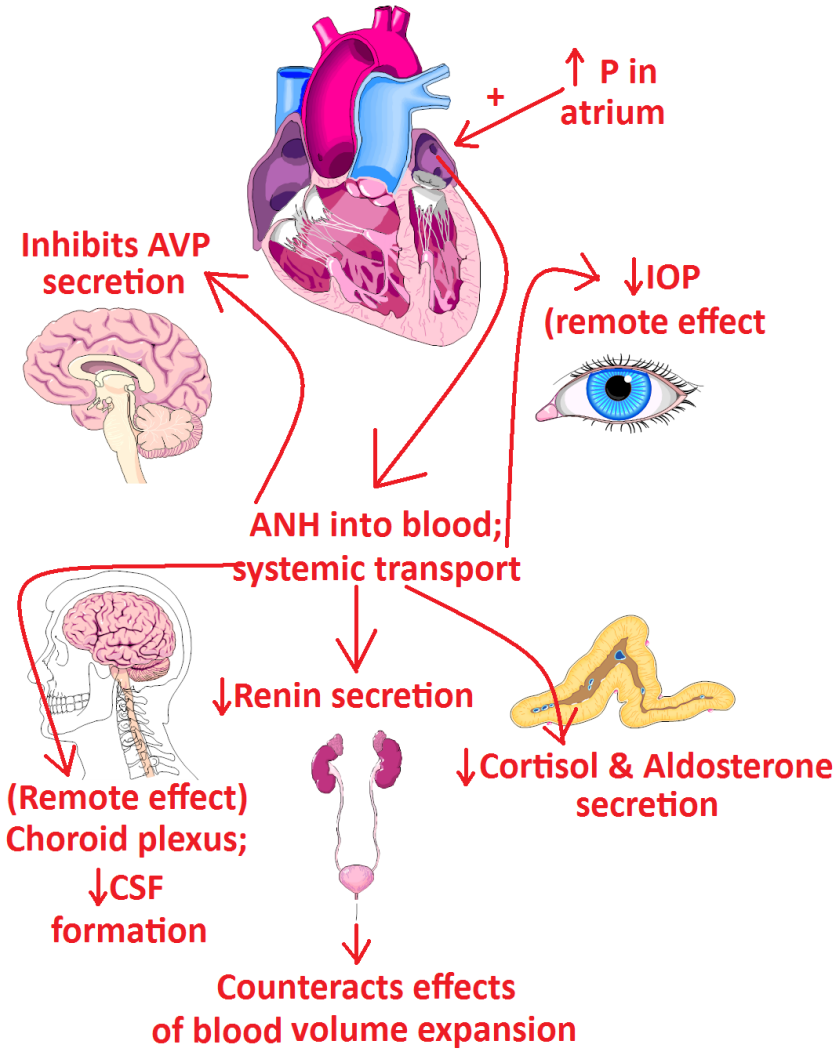
- Has P wave – has to be of sinus origin
- R waves far apart – slow rate

NSR -- Tachycardia

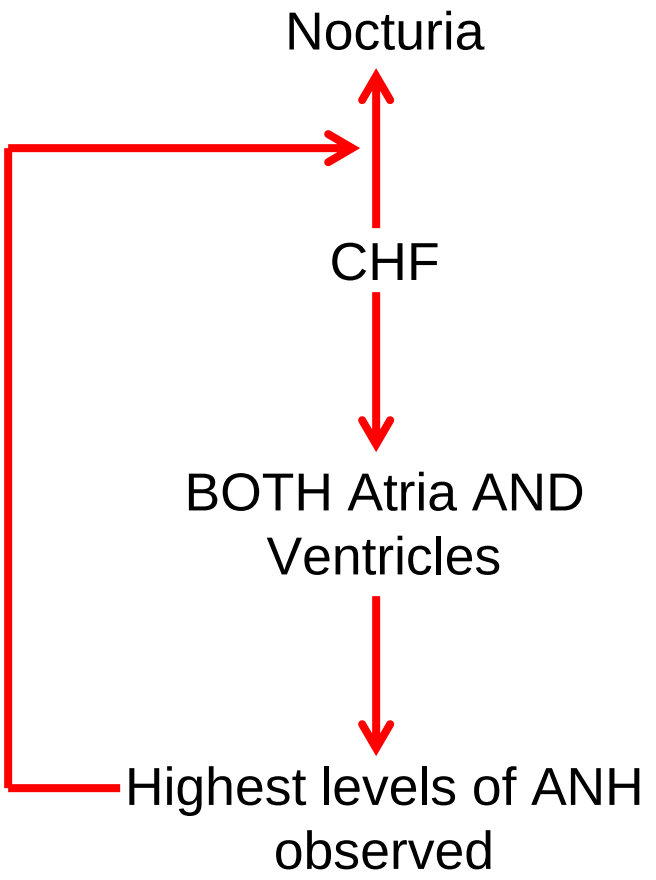


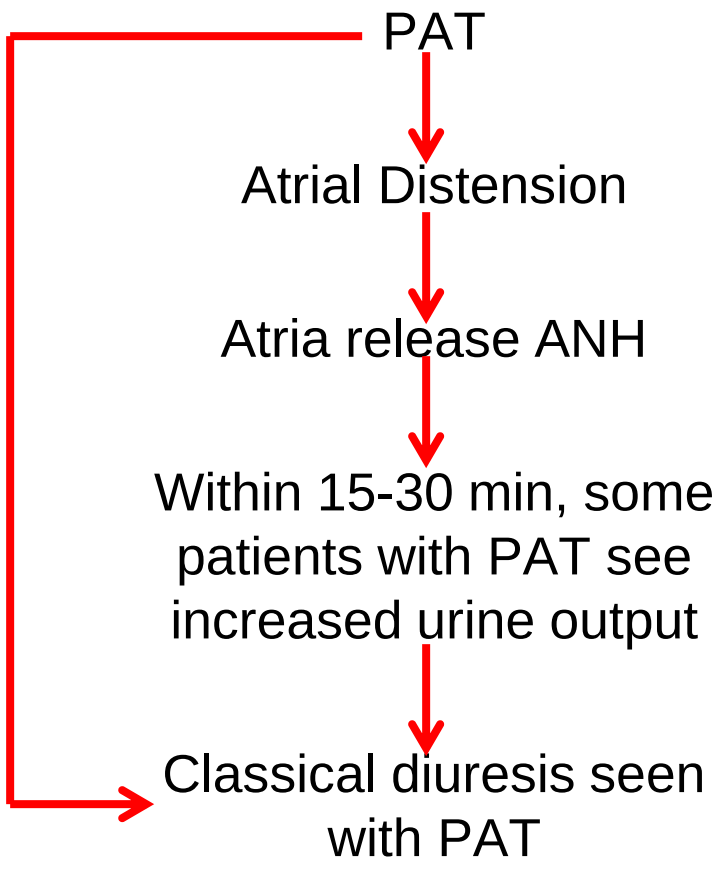
- Rule of 300
- R waves close together – fast rate
- Has P wave – has to be sinus

Atrial Natriuretic Hormone – ANF, ANP, ANH -- atriopeptin



Nocturia, [C]HF and ANH





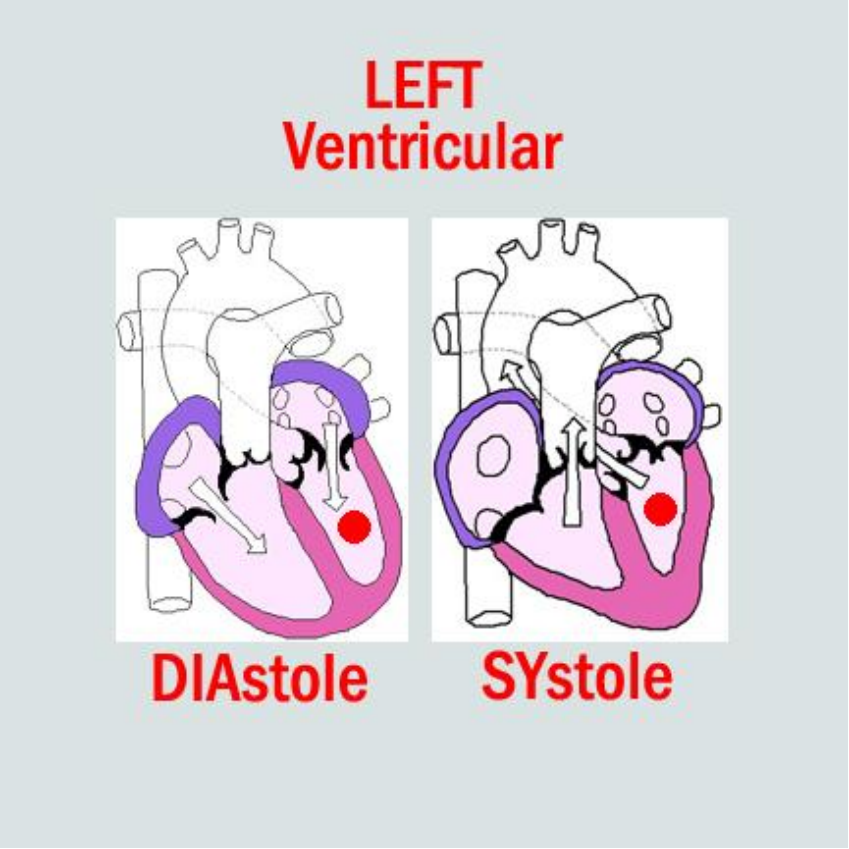
Nocturia ≡ due to increased left atrial pressure and is ≡ an abnormally large amount of urine at night – NOT increased frequency in urination!!!!

OCCASIONALLY, ANH is increased in hypertension BUT not enough for a direct relationship between ANP and hypertension.

Ventricular Failures

- Right ventricular failure
- Edema of extremities
- Left ventricular failure
- Pulmonary edema

Ejection Fraction



- = % of blood in the LEFT ventricle that is ejected per beat
- This method used to be invasive
- Normal in 2 planes is 67±9%
- Performed with 2-D echocardiography and computers
- Computer calculates ratios of areas and “spits out” the ejection fraction
- End-diastole vs end-systole

Ejection Fraction

- Calculation

$$Ejection\ \ Fraction = EF$$

$$EF = \frac{(End\ -diastolic\ \ volume) - (End\ -systolic\ \ volume)}{End\ -diastolic\ \ volume} * 100$$

$$EF = \frac{Stroke\ volume}{End\ -diastolic\ \ volume} * 100$$

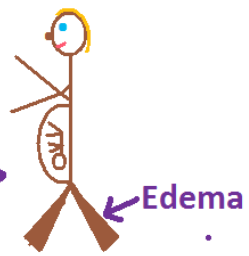
Ejection Fraction

- EF is small in heart failure
- EF may be large in well-conditioned people
- May be normal if both volumes decrease proportionately – diastolic dysfunction causing impaired filling of LV

ANP and Pre-eclampsia

- Pre-eclampsia = hypertension, edema and proteinuria in the last trimester – approximately 5% of all pregnancies result in pre-eclampsia
- [ANH] is elevated even though the plasma volume is increased in normal pregnancies
- ANH doesn't lead to hypertension, BUT is for homeostatic regulation, i.e., opens the kidneys up to excrete the overload of water and sodium ions in urine to aid in the recovery from cardiac and renal failure

Pre-eclampsia occurs in about 5% of all pregnancies



Edema

[Pre-]Eclamptic Pregnancy

Normal Pregnancy

↑Plasma Volume

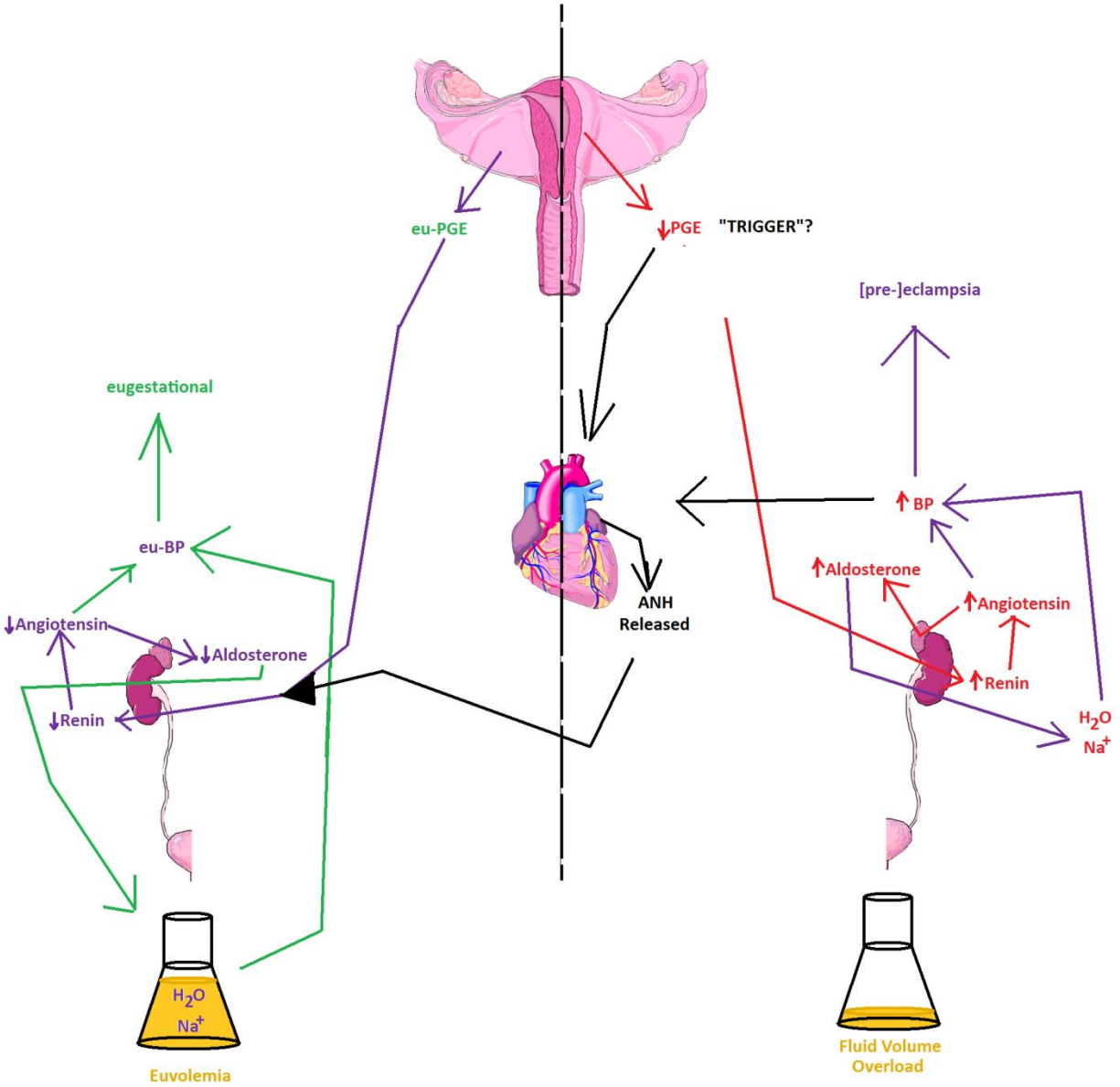
↑BP in Pregnancy

[↑ANH]

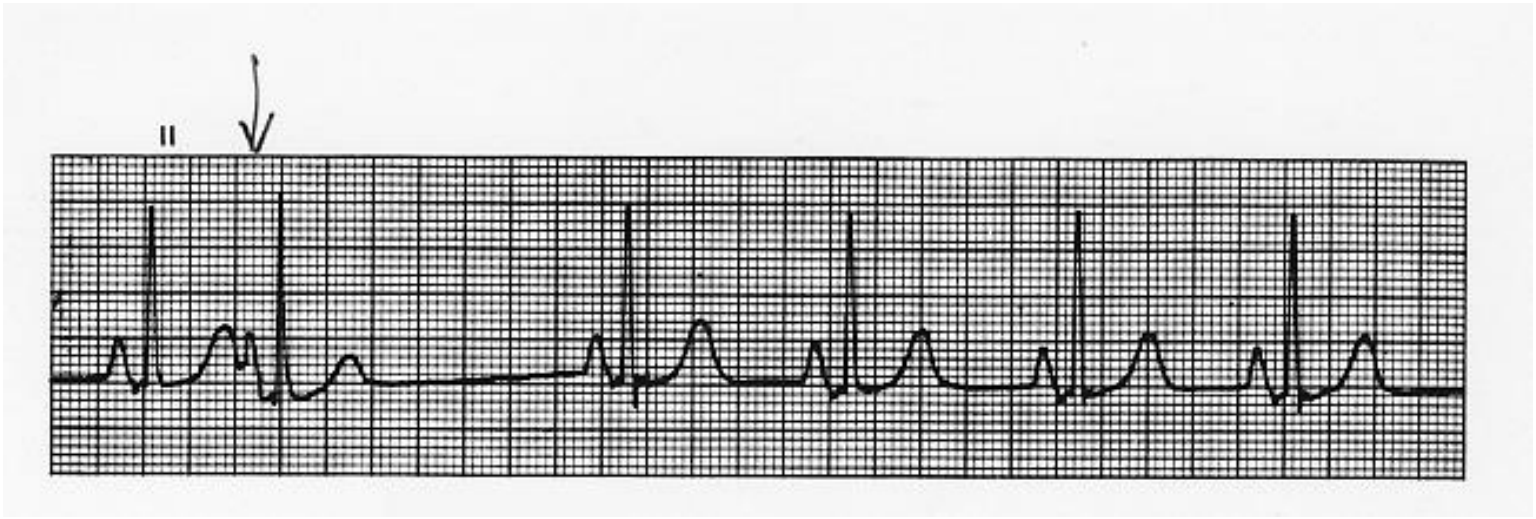
Homeostatic regulation:
opens kidney for recovery from cardiac/renal failure in pregnancy;
excrete excess H_2O and Na^+

↑BP
Proteinuria
Edema
Last Trimester

ANP and Pre-eclampsia

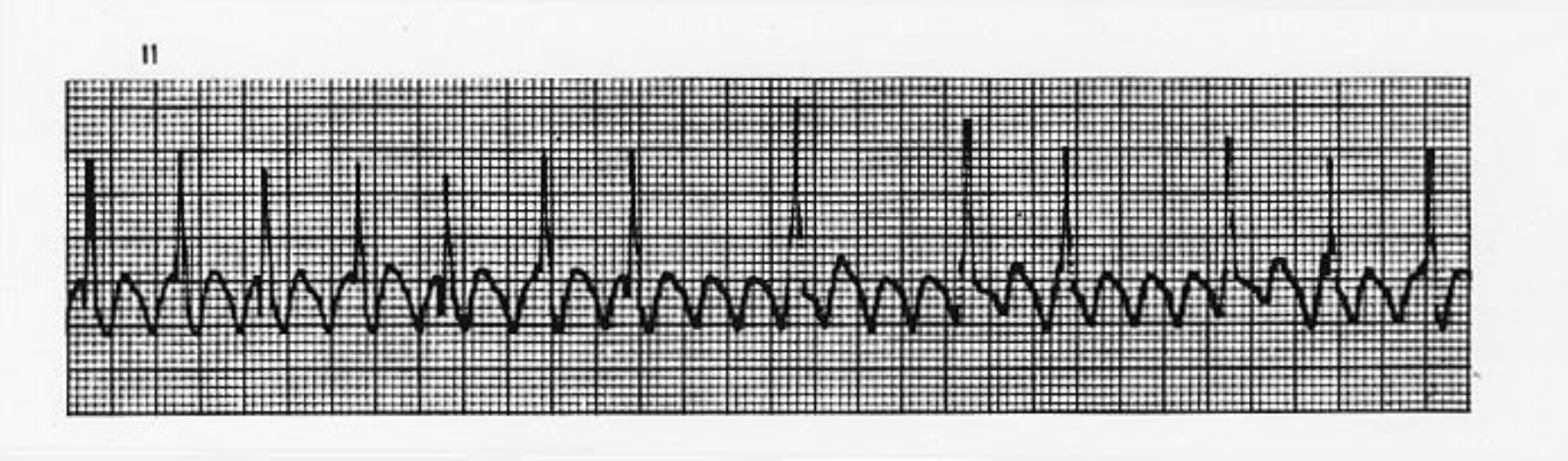


Pre-Mature Atrial Contraction



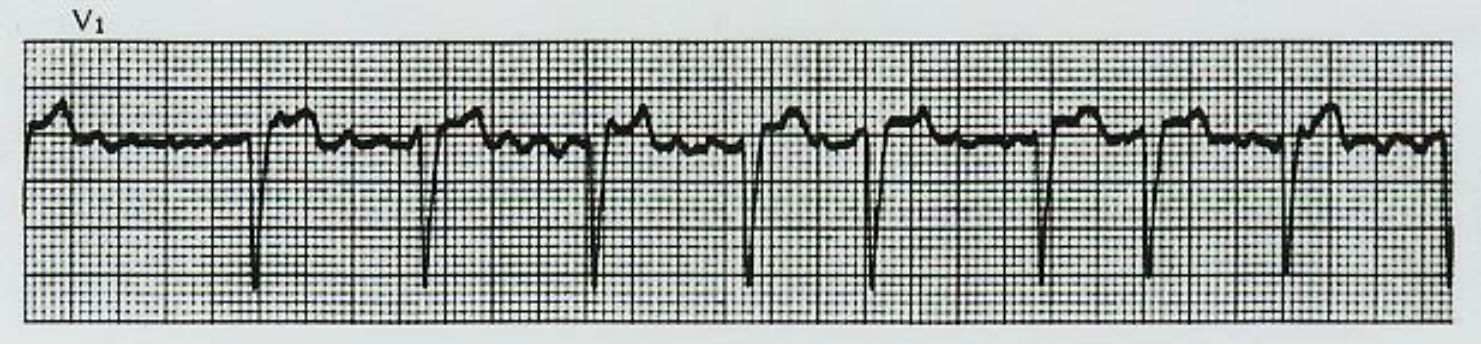
- Note premature P wave
- Since is P, has to be atrial

Atrial Flutter (with Secondarily Increased Ventricular Rate)



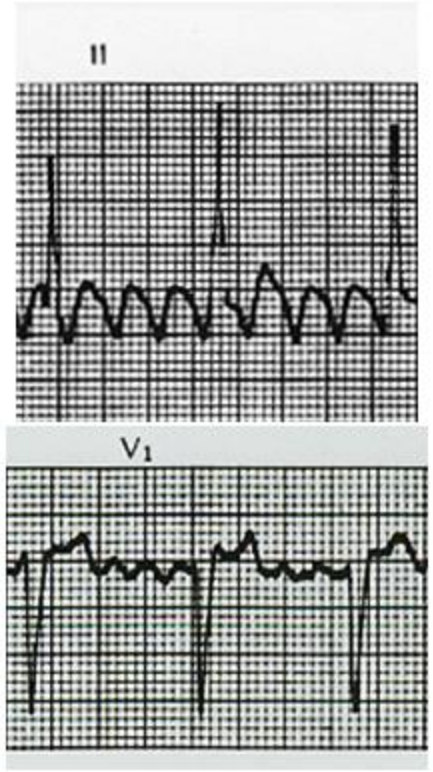
- Atria running at 300-350 bpm
- Ventricular rate about 150 bpm
- Note saw-tooth or shark-tooth pattern

Atrial Fibrillation with Secondarily Increased Ventricular Rate (Irregular)

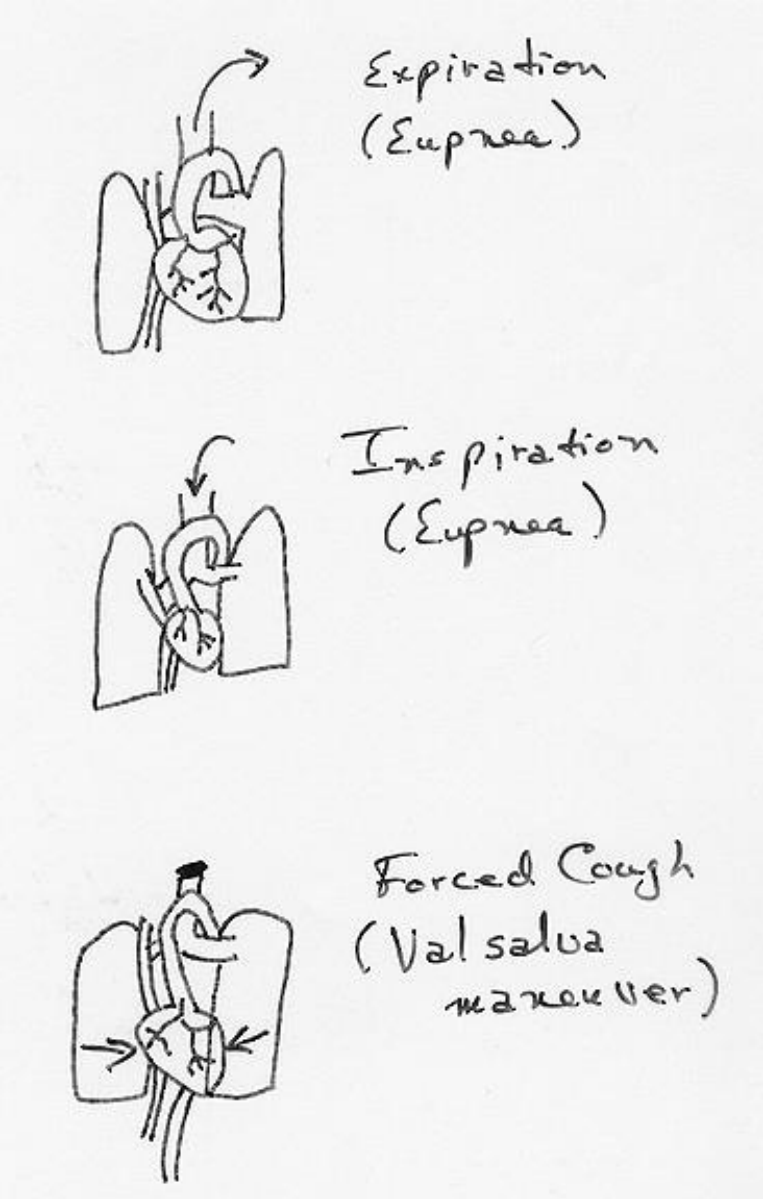


- Note “fineness” of “tremor”
- Note T waves, too

Compare and Contrast Atrial Flutter (top) with Atrial Fibrillation (bottom)

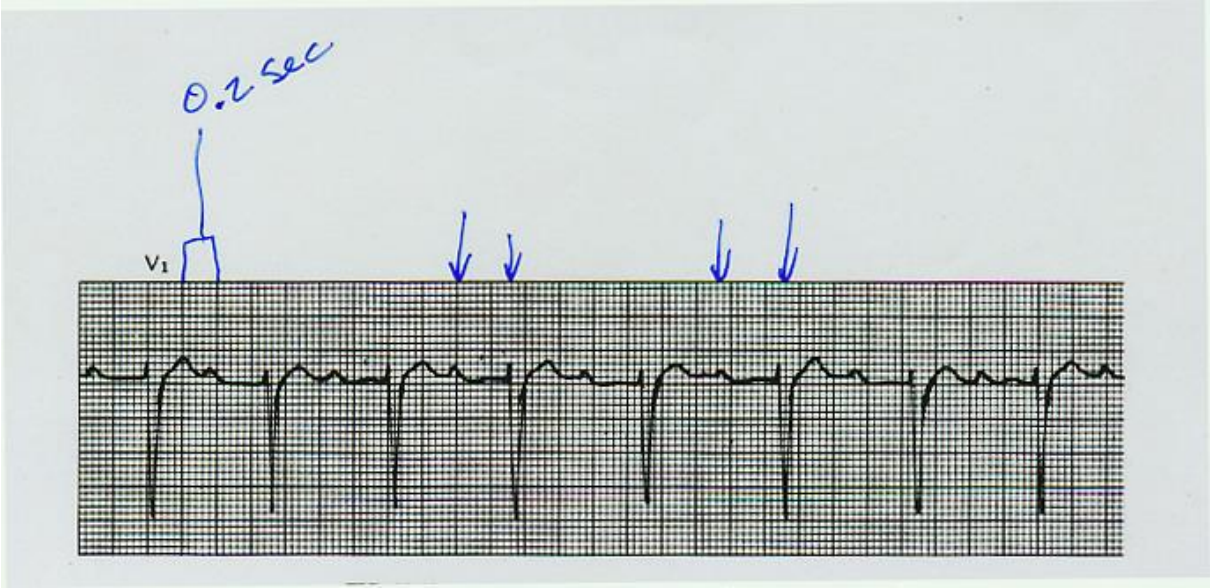


Valsalva Maneuver



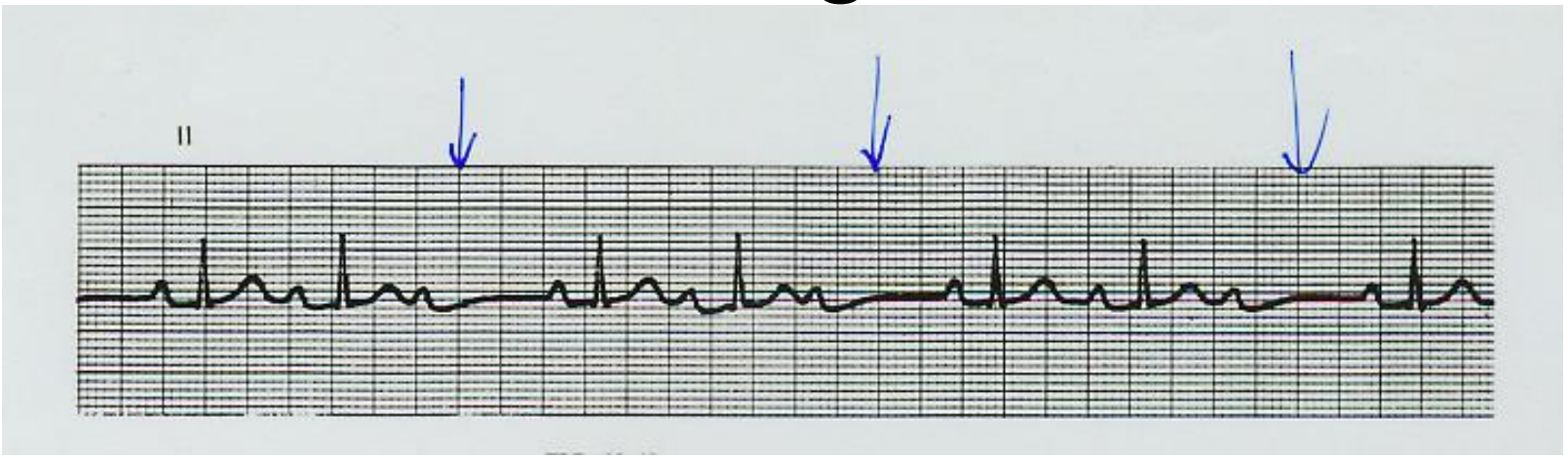
- Is worth maybe 40 Watts on a really good day
- Can be used to “knock” the heart back into a normal rhythm

First Degree Heart Block

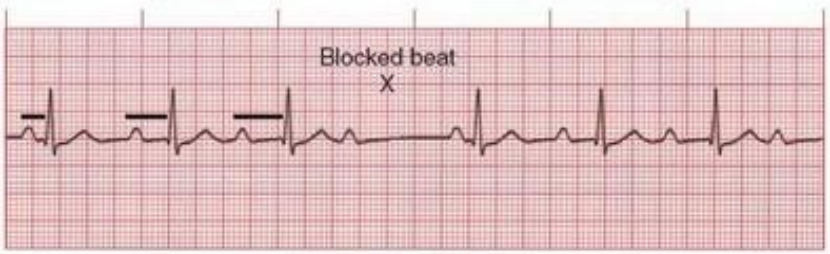


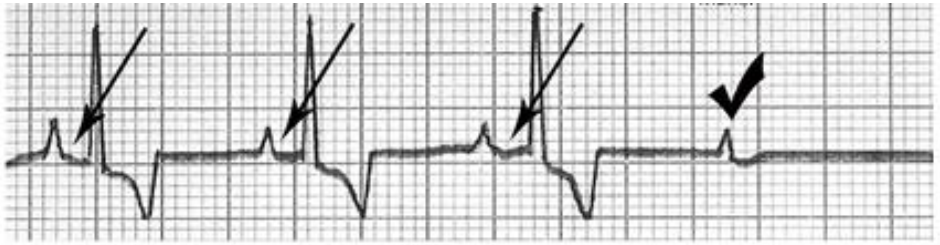
- Increased P to R interval – see arrows
- Time is greater than 0.20 seconds
- Each large square = 0.20 seconds (200 msec)

Second Degree Block

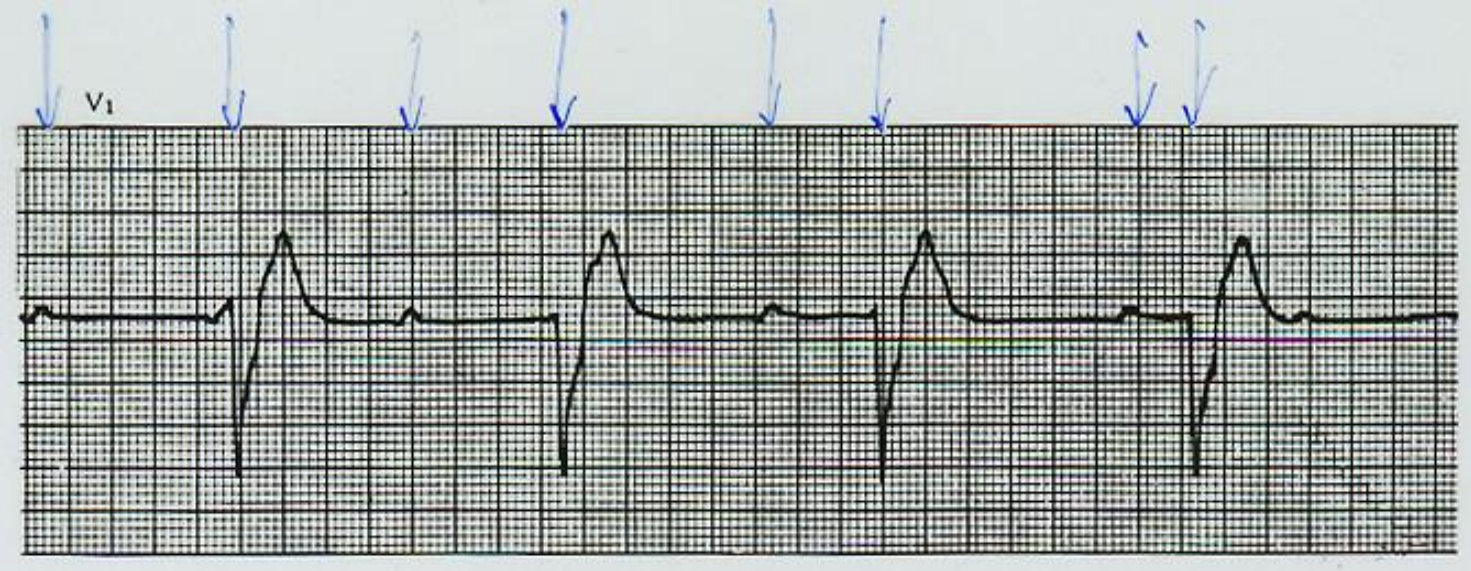


- Prolonged conduction
- Beats “dropped” on occasion
- See arrows
- Below LEFT: Mobitz I or Wenckebach – P-R Interval lengthens and beat drops
- Below RIGHT: Mobitz II or Hay – P-R Interval remains constant and beat drops



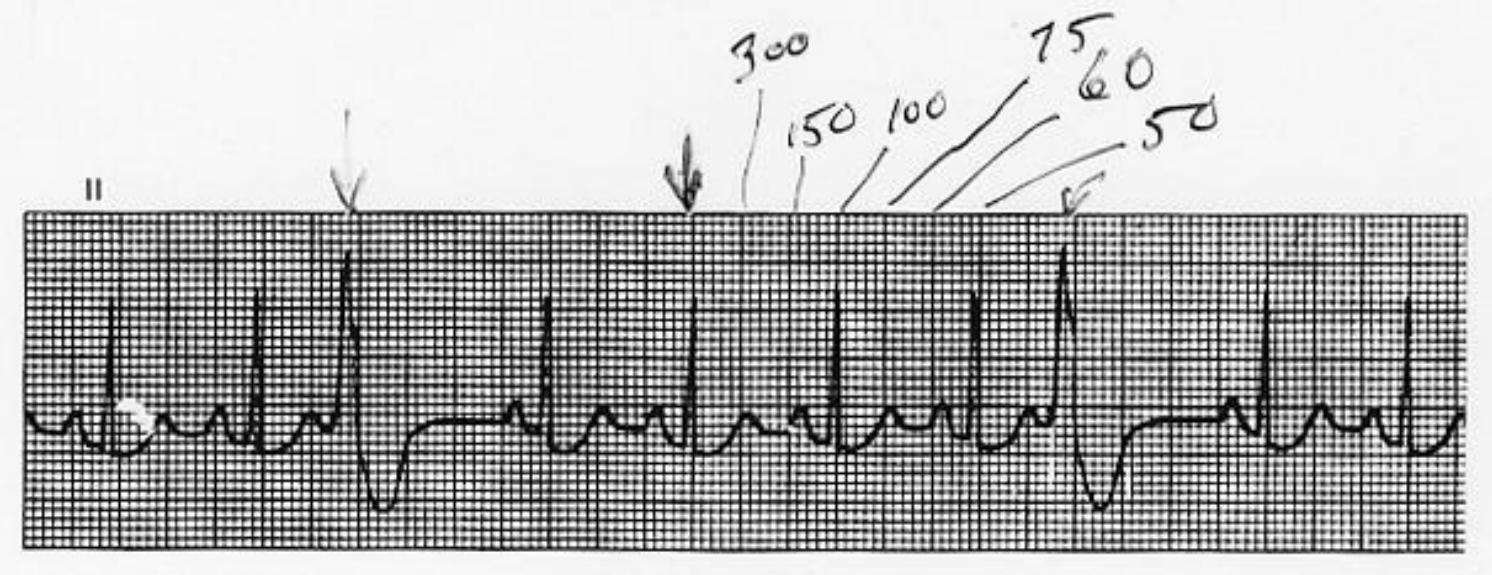


Third Degree (Complete) Heart Block



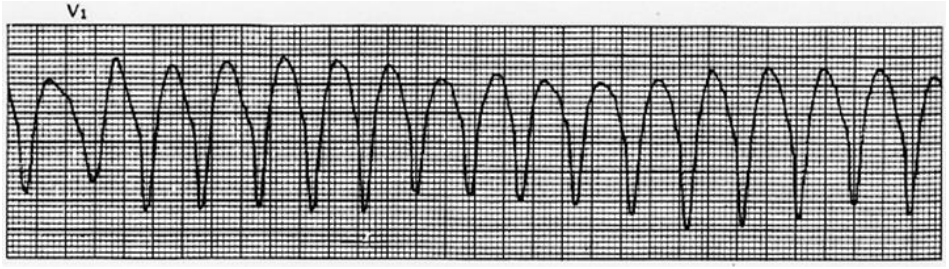
- NO SA node stimulus to AV node
- See arrows
- Ventricular Rate approximately 30 bpm
- Ventricular contraction due to ventricular pacing

Premature Ventricular Contractions (PVC's)

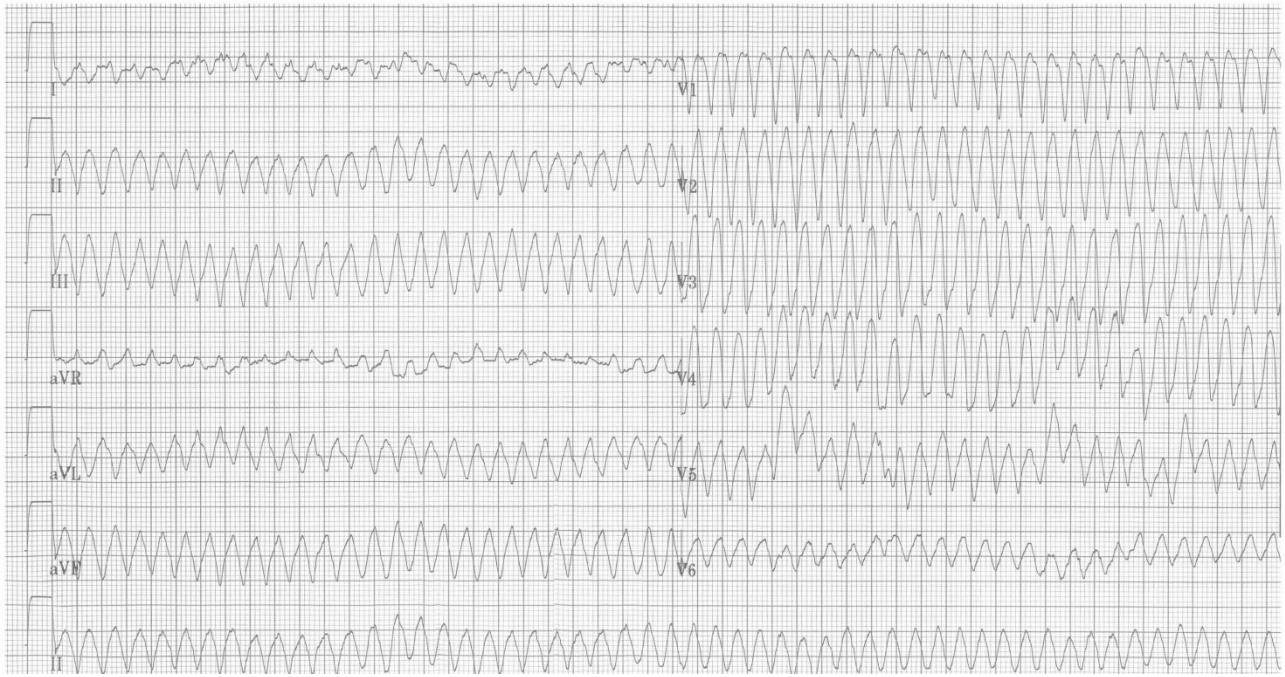


- Dangerous when "off" the T wave
- Dangerous when are 2 or more in a row
- Dangerous when are 6 or more per minute (not everyone agrees on this)
- PVC's every other beat is called bigeminy

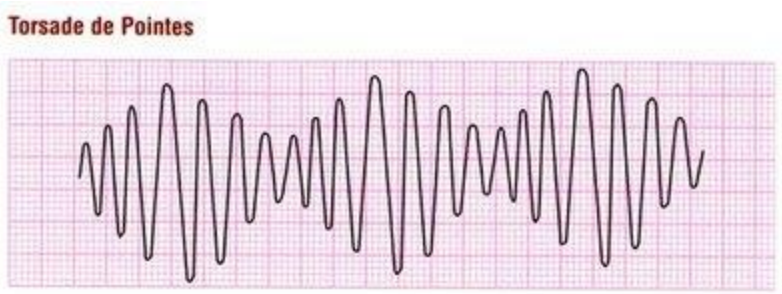
Ventricular Tachycardia – aka flutter



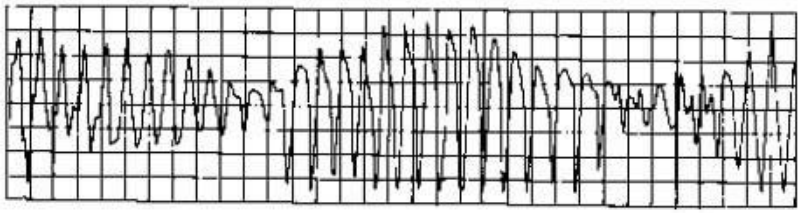
- Precursor to ventricular fibrillation; Rate above is between 150-250 bpm



Torsades de Pointes



http://mstcparamedic.pbworks.com/w/page/21902876/Torsades%20de%20Pointes



Torsades de pointes ventricular tachycardia

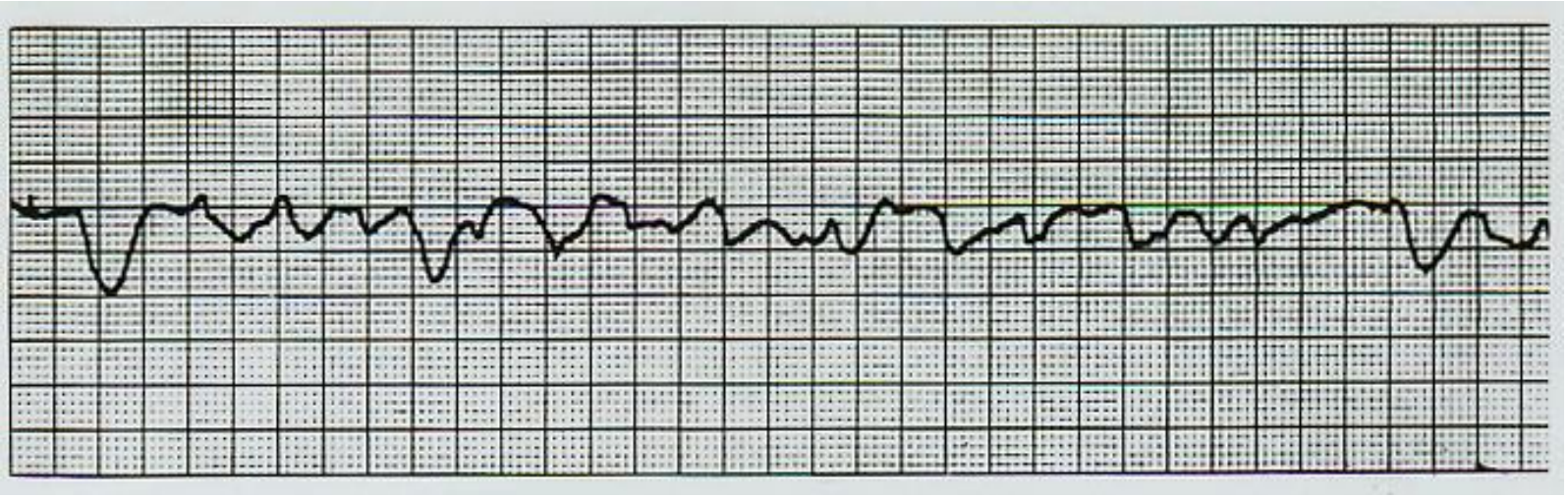
http://patient.info/doctor/torsades-de-pointes

- **Torsades de pointes** is a specific form of polymorphic VT in patients with a long QT interval. It is characterized by rapid, irregular QRS complexes, which appear to be twisting around the ECG baseline. This arrhythmia may cease spontaneously or degenerate into ventricular fibrillation.

http://www.merckmanuals.com/professional/cardiovascular-disorders/arrhythmias-and-conduction-disorders/long-qt-syndrome-and-torsades-de-pointes-ventricular-tachycardia

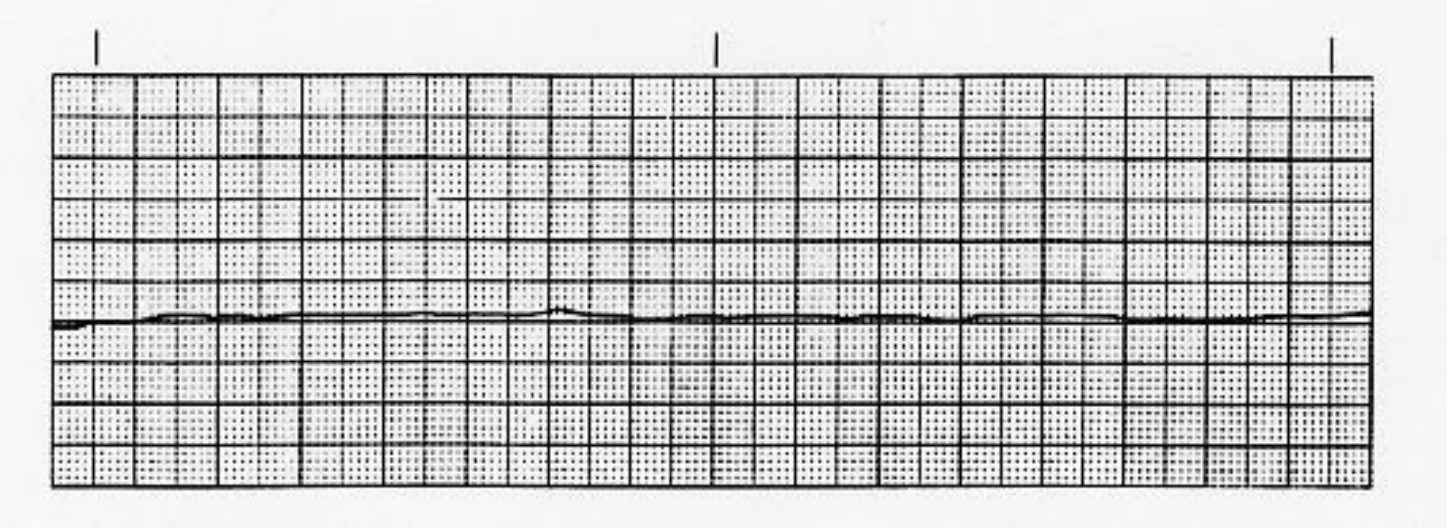
It causes significant hemodynamic compromise and often death. Diagnosis is by ECG. Treatment is with IV magnesium, measures to shorten the QT interval, and DC defibrillation when ventricular fibrillation is precipitated.

Ventricular Fibrillation



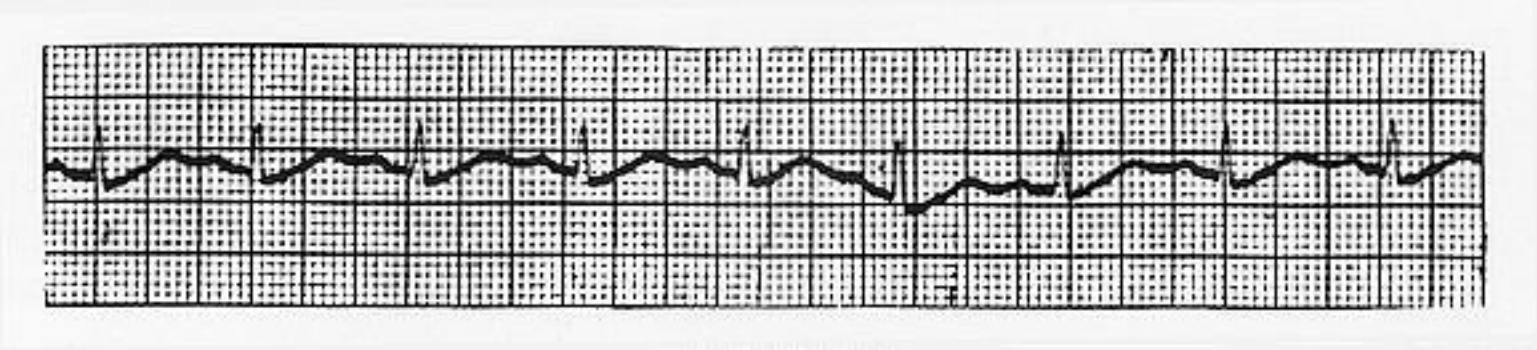
- Inneffective, disorganized ventricular beating
- Quivering
- INCOMPATIBLE with life

Asystole



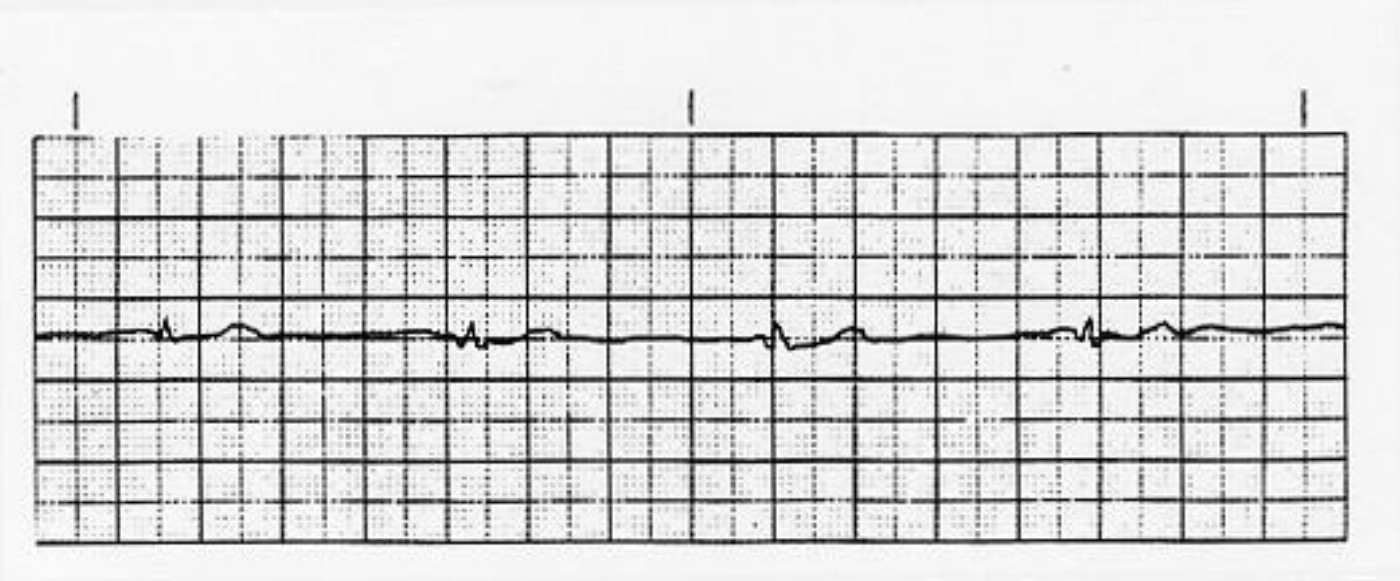
- Cardiac arrest
- Or electrodes have fallen off or been taken off

Electromechanical Dissociation



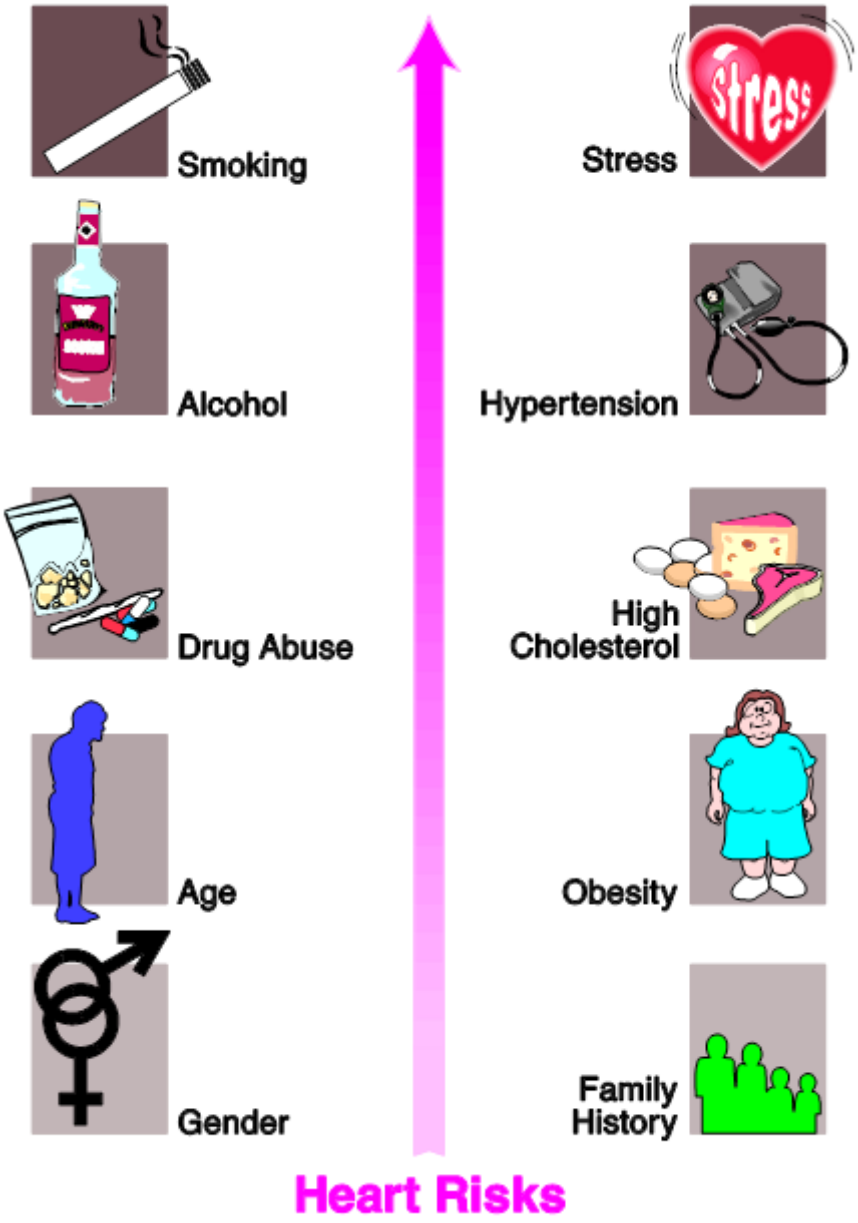
- EMD is old name
- Now called Pulseless Electrical Activity (PEA)
- Electrical system works – myocardium doesn't
- Monitor looks normal – patient has no pulse
- Worthwhile to walk around and actively look at patients periodically
- Resuscitative pharmacology includes:
  - Epinephrine,
  - Vasopressin, and
  - Atropine (If the underlying rhythm is bradycardia (ie, heart rate < 60 bpm) associated with hypotension,).
  - Sodium bicarbonate may be administered only in patients with severe, systemic acidosis, hyperkalemia, or a tricyclic antidepressant overdose. Routine administration is discouraged because it worsens intracellular and intracerebral acidosis and does not appear to alter the mortality rate.

Slow Idioventricular Rhythm



- No pulse
- Beats are of ventricular origin
- Note lack of P waves

Cardiac Disease Risk Factors



Treadmill Testing (Bruce Protocol) for Potential Heart Disease

- Based on “MET’s”
- A “MET” = 3.5 – 4 mL O_2 consumed/kg BW/minute at rest
- Has STOPPING CRITERIA:
  - Angina worse than usual
  - Mental Confusion
  - Dropping Systolic BP
  - Arrhythmias
  - Target Heart Rate Reached for Age
  - Nausea, ashen pallor, cold skin
  - Fatigue
  - NO increase in HR with an increasing work load
  - Patient’s request – or demand, in some cases

Bruce Protocol

Stage	Speed (mph)	% Gradient	Minutes	MET's
I	1.7	10	3	5
II	2.5	12	3	7
III	3.4	14	3	9-12
IV	4.2	16	3	12-14
V	5.0	18	3	14-16±
VI	5.5	20	3	16

Energy Cost in MET's

MET	Example	MET	Example
1	Eating, resting, writing, knitting	6	Shovel snow, saw wood, walk 5 mph level
2	Driving (more or less), walking 2.2 mph	8	Level skiing at 4 mph, walking 5-6 mph level, cycling 13 mph
3	**Self care (wash and dress self)**	10	Fast downhill skiing, walking 5 mph uphill
4	Weeding, ballroom dancing, golf, walking level at 4 mph		

Myocardial Infarction

- Lay term = “heart attack”; death of heart muscle from oxygen deprivation
- Symptoms and Signs vary between the genders
  - **Women's major symptoms prior to their heart attack included:**
    - Unusual fatigue - 70%
      Shortness of breath - 42%
      Anxiety - 35%
      Sleep disturbance - 48%
      Indigestion - 39%
      Frequency varies by researcher/study!
  - **Major symptoms during the heart attack include:**
    - Shortness of breath - 58%
      Unusual fatigue - 43%
      Dizziness - 39%
      Weakness - 55%
      Cold sweat - 39%
      Frequency varies by researcher/study
  - **Women's symptoms are not as predictable as men's**
  - **Women have more unrecognized heart attacks than men and are more likely to be, "mistakenly diagnosed and discharged from emergency departments”.**
    McSweeney, JC et al. "Women's Early Warning Symptoms of Acute Myocardial Infarction," *Circulation*, 2003 Nov 25;108(21):2619-23.
  - **Many physicians <u>still</u> don't recognize that women's symptoms differ from men’s symptoms!**
  - **Men’s symptoms, which some women experience:**
    - Pressure, fullness or a squeezing pain in the center of the chest, which may spread to the neck, shoulder or jaw;
    - Chest discomfort with lightheadedness, fainting, sweating, nausea or shortness of breath;
    - Pain due to “shorting out” of nerves across Vagus (X) and middle cervical nerves
- Besides using EKG’s to diagnose MI’s, clinicians can use lab tests, too, including external cardiology consults

MI – Lab Tests -- Classical

- CK – used to be CPK – Creatine Phosphate Kinase

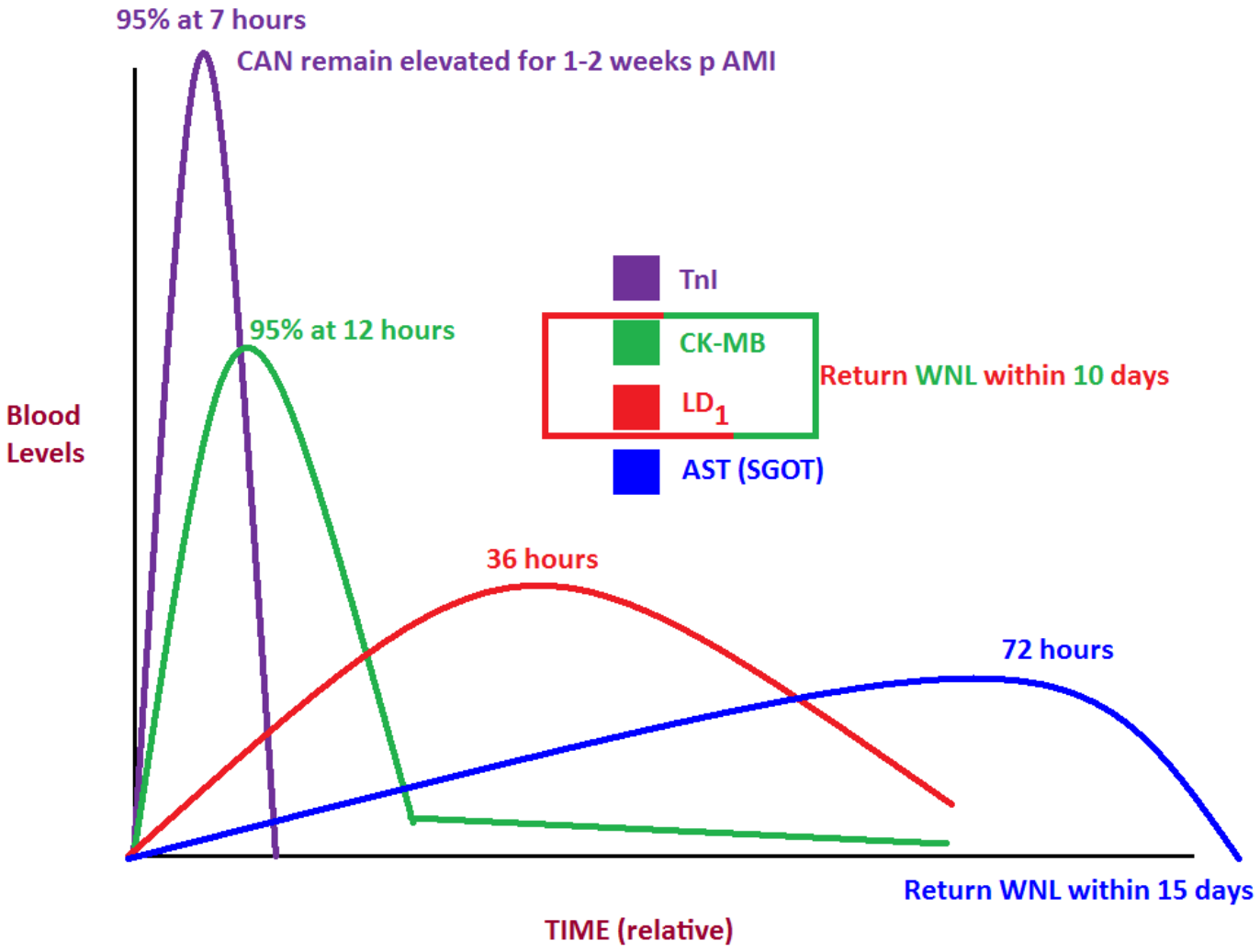
CK-BB – Brain and Lung
CK-MM – Skeletal Muscle
CK-MB – Cardiac Muscle

- CK – regardless of fraction – is used to catalyze the phosphorylation of creatine (C) to creatine phosphate (CP).
- CP then is used to phosphorylate ADP to ATP – see the significance?

MI – Lab Tests -- Classical

Serum LDH Levels in Disease States						
Fx	Source	MI	Hepatitis	PE	Tumor	Shock
LD_1	RBC, heart, kidney	↑	0	0	0	↑
LD_2	Heart	↓	0	0	↑	↑
LD_3	Lung	0	0	↑	↑	↑
LD_4	?	0	0	0	↑	↑
LD_5	Liver	0	↑	0	0	↑

MI – Lab Tests – Classical, too



MI – Lab Tests – Current Additions

- Troponin I and T are structural components of cardiac muscle. They are released into the bloodstream with myocardial injury. They are highly specific for myocardial injury--more so than CK-MB--and help to exclude elevations of CK with skeletal muscle trauma. Troponins will begin to increase following MI within 3 to 12 hours, about the same time frame as CK-MB. However, the rate of rise for early infarction may not be as dramatic as for CK-MB.
- Troponins will remain elevated longer than CK--up to 5 to 9 days for troponin I and up to 2 weeks for troponin T. This makes troponins a superior marker for diagnosing myocardial infarction in the recent past--better than lactate dehydrogenase (LDH). However, this continued elevation has the disadvantage of making it more difficult to diagnose reinfarction or extension of infarction in a patient who has already suffered an initial MI.
- Troponin T lacks some specificity because elevations can appear with skeletal myopathies and with renal failure.

http://www.hallym.or.kr/~kdcp/chemistry/MI-Diagnisis.htm

MI – Lab Tests – Current Additions

- Myoglobin is a protein found in skeletal and cardiac muscle which binds oxygen. It is a very sensitive indicator of muscle injury. The rise in myoglobin can help to determine the size of an infarction. A negative myoglobin can help to rule out myocardial infarction. It is elevated even before CK-MB. However, it is not specific for cardiac muscle, and can be elevated with any form of injury to skeletal muscle.

http://www.hallym.or.kr/~kdcp/chemistry/MI-Diagnisis.htm

Low Level Stress Test – post-MI

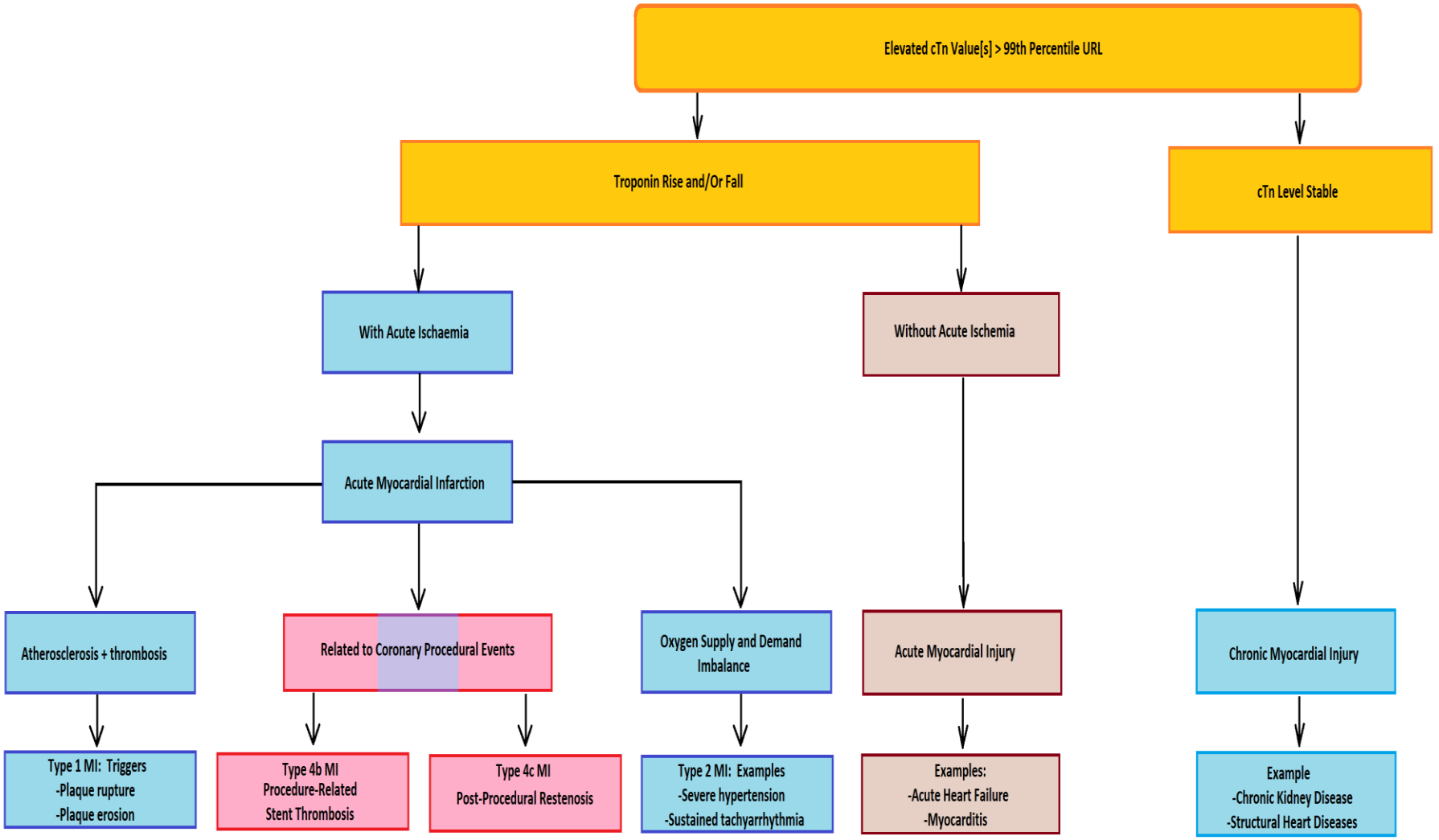
Determines if you get to go home – need a minimum of 3 MET's to go home			
Speed	% Gradient	Minutes	MET's
1.2	0	3	2.14
1.2	3	3	2.34
1.2	6	3	2.74
1.7	6	3	3.30

4th Universal Definition of MI: Nutshell Version Spring 2020

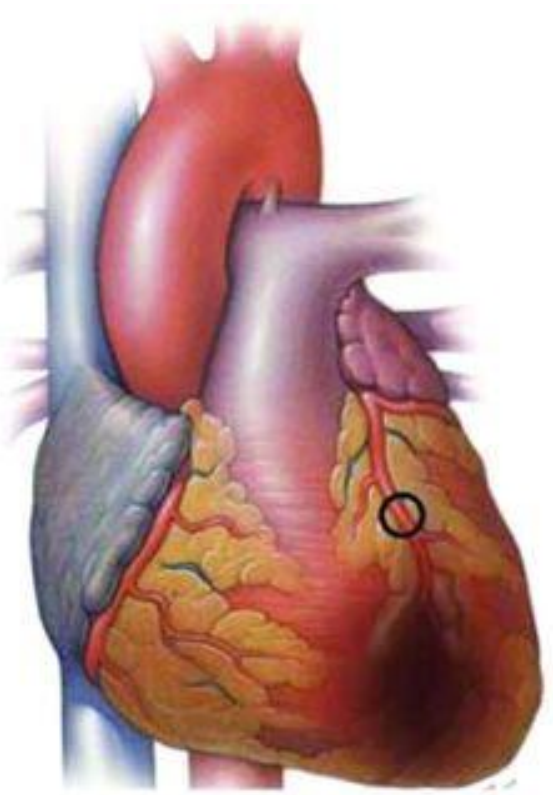
Source 1: Accessed 6-8 Jan 2020, https://www.acc.org/latest-in-cardiology/articles/2018/11/16/09/06/fourth-universal-definition-of-mi

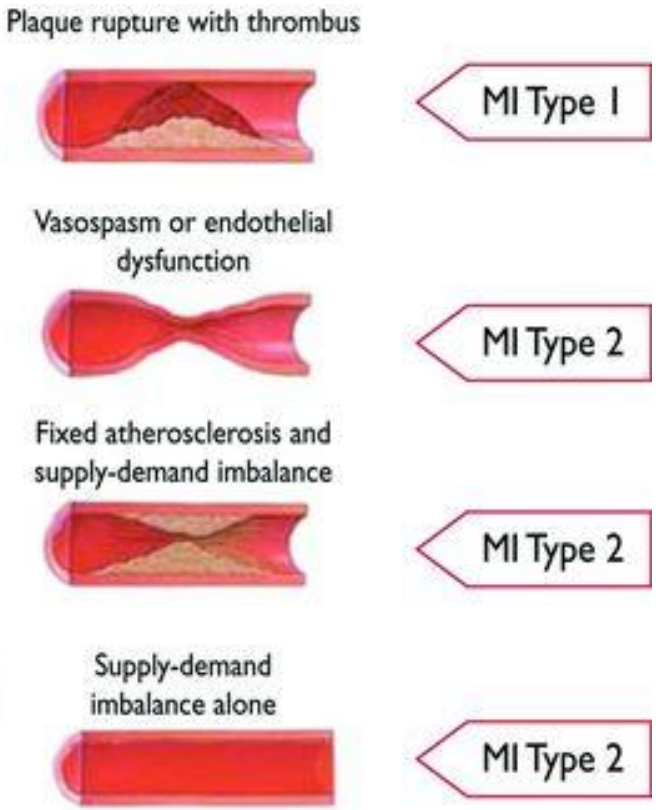
Source 2: Accessed 6-8 Jan 2020, https://ahajournals.org/doi/10.1161/cir.0b013e31826e1058

4th Universal Definition of MI: Nutshell Version



Types 1 and 2 MI's Illustrated





Type 1 MI: Triggers
-Plaque rupture
-Plaque erosion

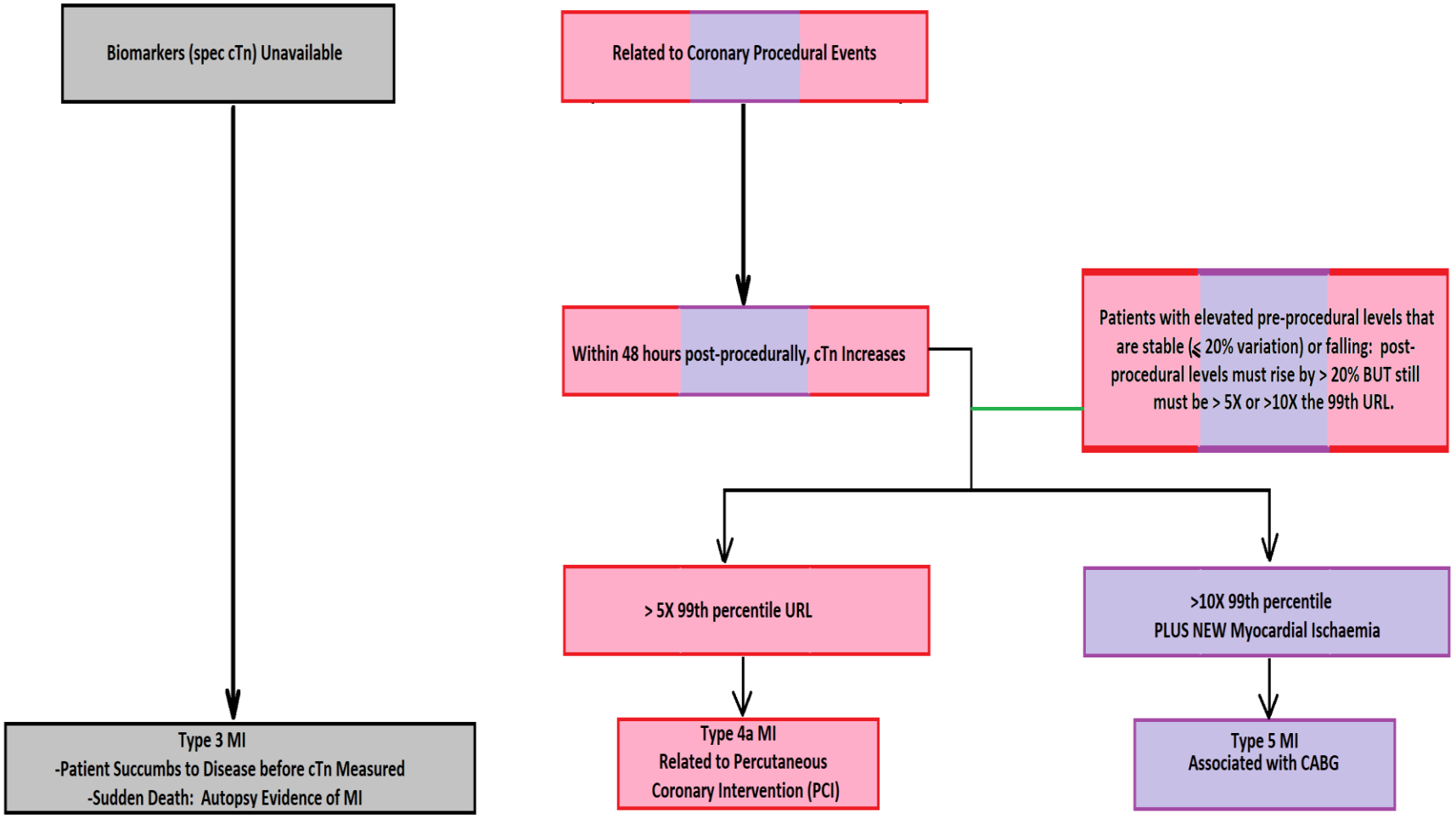
Type 2 MI: Examples
-Severe hypertension
-Sustained tachyarrhythmia

Oxygen Supply and Demand Imbalance

Kristian Thygesen. Circulation. Third Universal Definition of Myocardial Infarction, Volume: 126, Issue: 16, Pages: 2020-2035, DOI: (10.1161/CIR.0b013e31826e1058)

© 2012 American Heart Association, Inc.

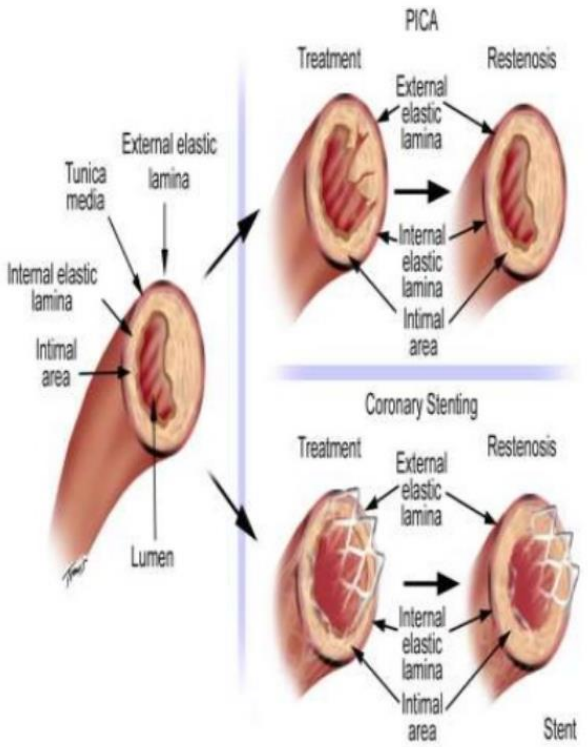
4th Universal Definition of MI: Nutshell Version



Percutaneous Coronary Interventions

Percutaneous Coronary Interventions

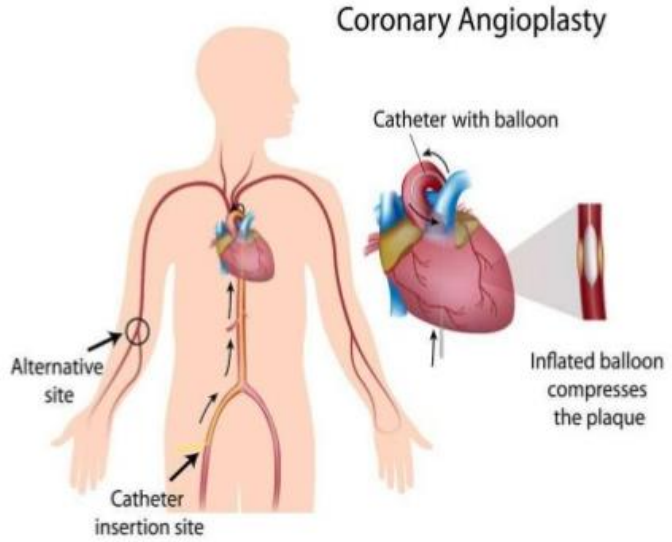
- Percutaneous coronary interventions (PCI) include percutaneous transluminal coronary angioplasty (PTCA) with or without stent insertion
- First performed in human by Andreas Gruentzig in 1971
- Most commonly performed interventional procedure
- PTCA and stent placement within 90 min of onset of pain is the optimal treatment of transmural ST-segment–elevation myocardial infarction (STEMI).
- Elective PCI may be appropriate for post-MI patients who have recurrent or inducible angina before hospital discharge and for patients who have angina and remain symptomatic despite medical treatment.



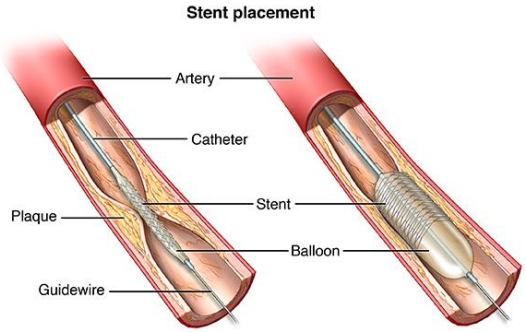
Michael J. Shea. Percutaneous Coronary Interventions. MSDManual. Feb. 2016

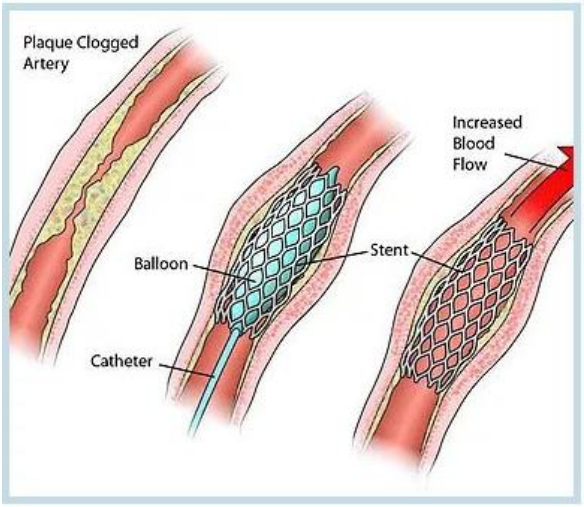
https://www.slideshare.net/WaseemAkramSiddiqui/basics-of-pci ... Accessed 8 Jan 2020, 0547 hours PST

PCI – Non-Surgical – Still Invasive



https://www.slideshare.net/LadiAnudeep/percutaneous-coronary-intervention-99173322 ... Accessed 8 Jan 2020, 0539 hours PST

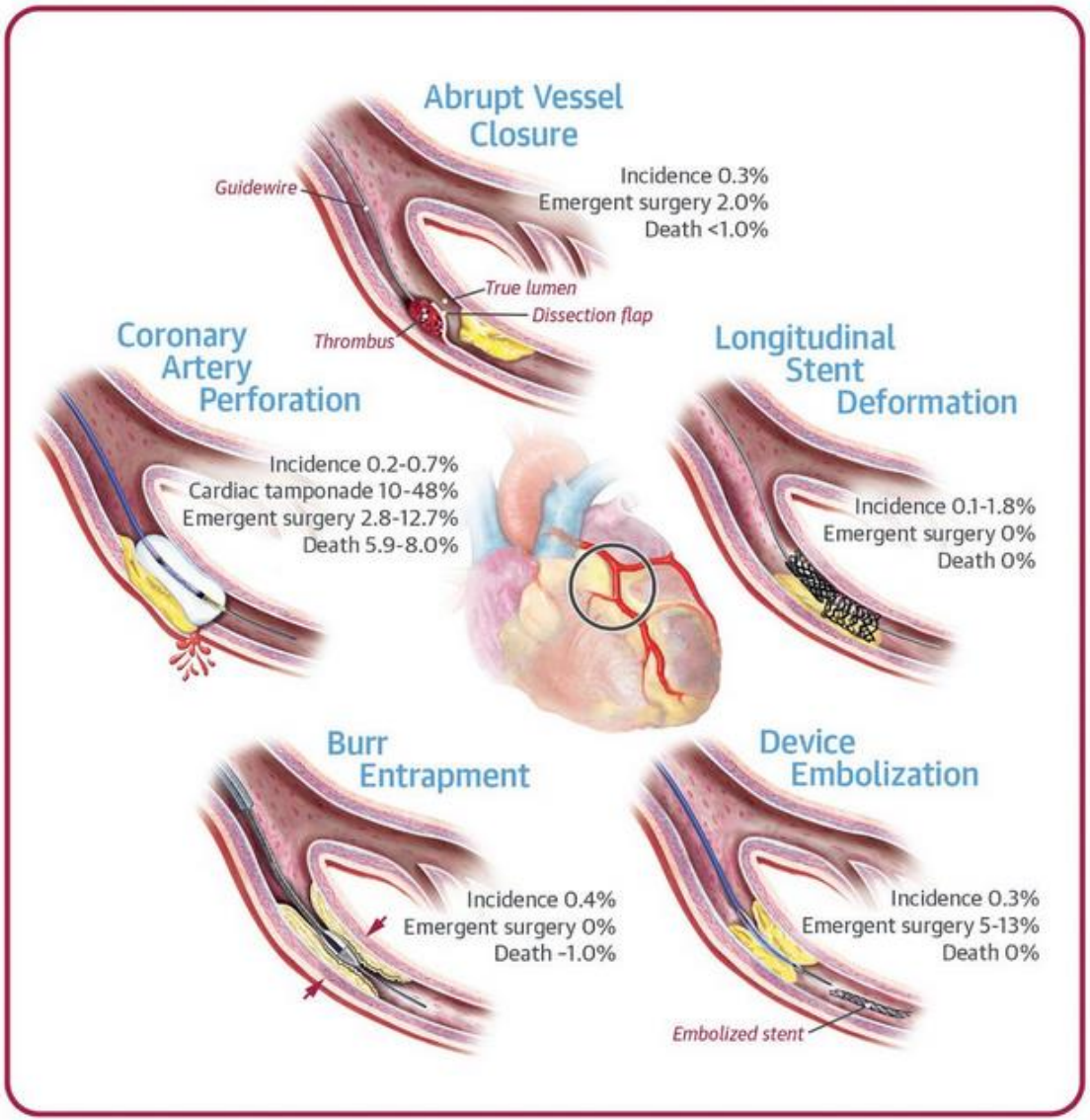




https://www.totallyvein.com/stent-placement/ accessed 8 Jan 2020, 0530 hours PST – Angioplasty + Stent Placement = PCI

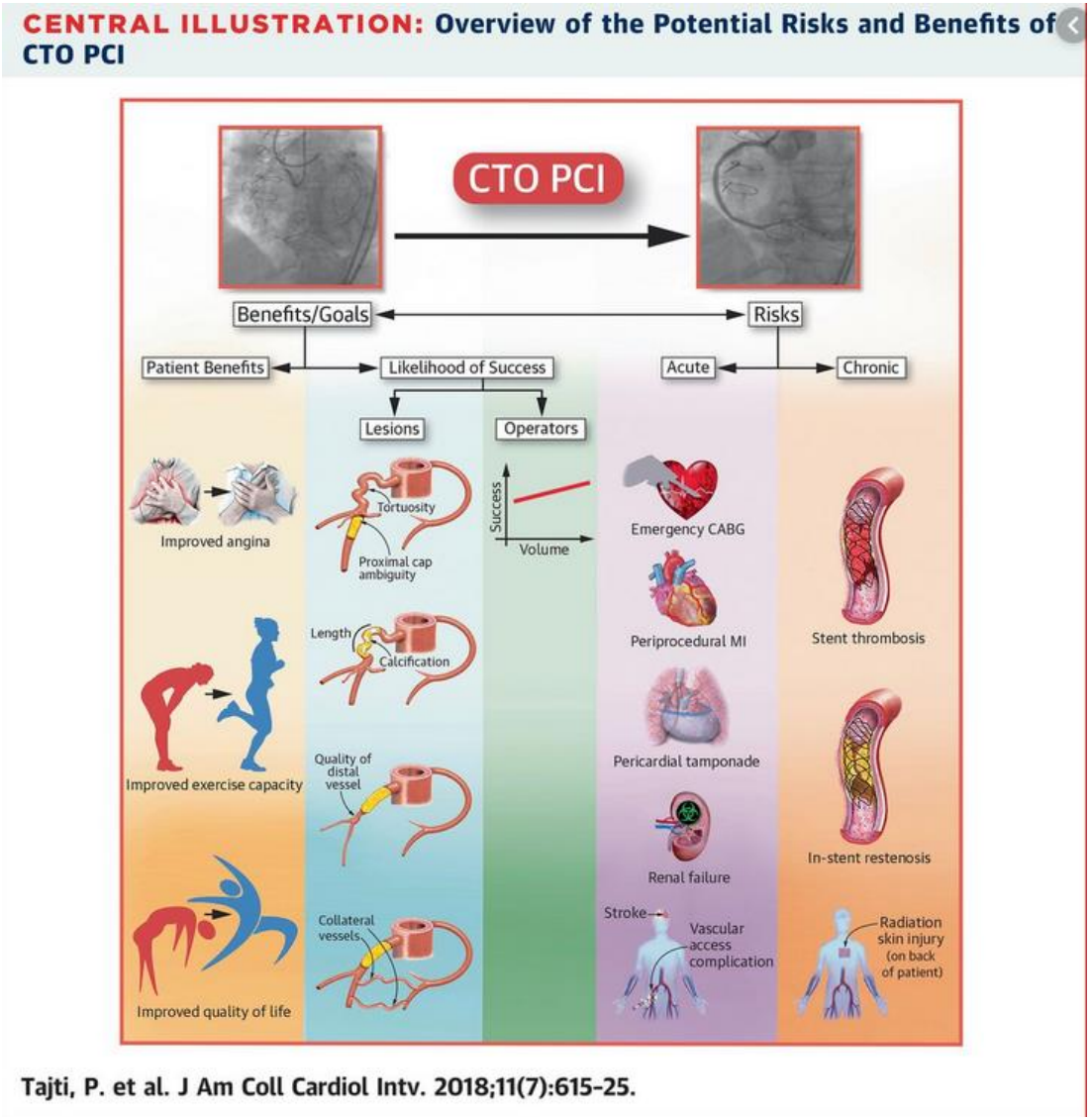
https://teleme.co/doctors/profile/zainal-hamid ... Accessed 8 Jan 2020, 0533 hours PST

CENTRAL ILLUSTRATION: Epidemiology of Mechanical Complications During Percutaneous Coronary Intervention



Giannini, F. et al. J Am Coll Cardiol Intv. 2018;11(18):1797-810.

Chronic Total Occlusion PCI



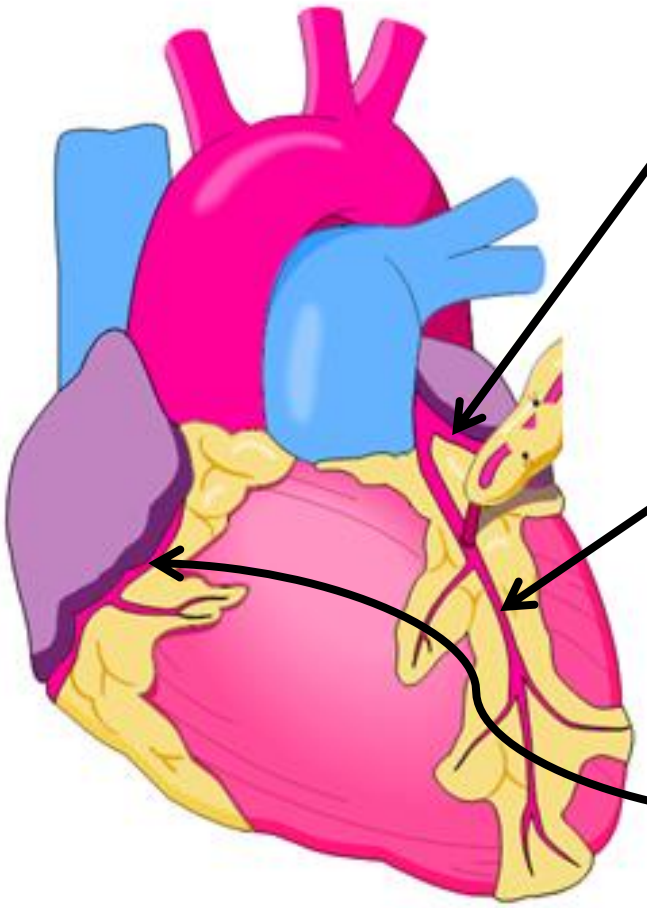
http://interventions.onlinejacc.org/content/11/7/615 ... Accessed 8 Jan 2020, 0552

Chronic Total Occlusion PCI

- **Radiation skin injury**
- CTO PCIs are often long procedures with high patient (and operator) radiation dose **(55)**. High radiation dose may lead to acute dermatitis of the exposed area that can progress to chronic skin ulcer and even require surgical intervention. In a study of 2,124 patients undergoing 2,579 PCIs (including 238 CTO PCIs), a chronic skin ulcer developed in 0.34% (9 patients, 5 of which were CTO PCIs with skin lesion onset after 1 to 3 months of interventions) requiring surgical intervention in 8 of them **(56)**. Most operators currently recommend stopping the procedure after reaching 7- or 8-Gy air kerma dose. It is also recommended to monitor the patient for radiation skin injury if >4- or 5-Gy air kerma dose is administered. With use of newer x-ray equipment, low cine and fluoroscopy frame rate, and meticulous attention to technique, radiation dose can be significantly reduced **(57)**. Additionally, the use of disposable sterile radiation shields during CTO PCI can reduce operator radiation dose to levels similar to those of non-CTO PCIs **(58)**.

http://interventions.onlinejacc.org/content/11/7/615 ... Volume 11, Issue 7, April 2018; Accessed 8 Jan 2020, 0555 hours PST

Coronary Artery By-pass Graft – Blood Supplies



CXA: blood to LA, lateral wall LV, Inferior/diaphragmatic area of LV

LAD: blood to anterior and apex of LV, anterior 2/3 of septum

RCA: blood to SA node, RA and RV, inferior/diaphragmatic LV

Arrhythmias Associated with MI's -- 1

Left Coronary Artery Compromisation

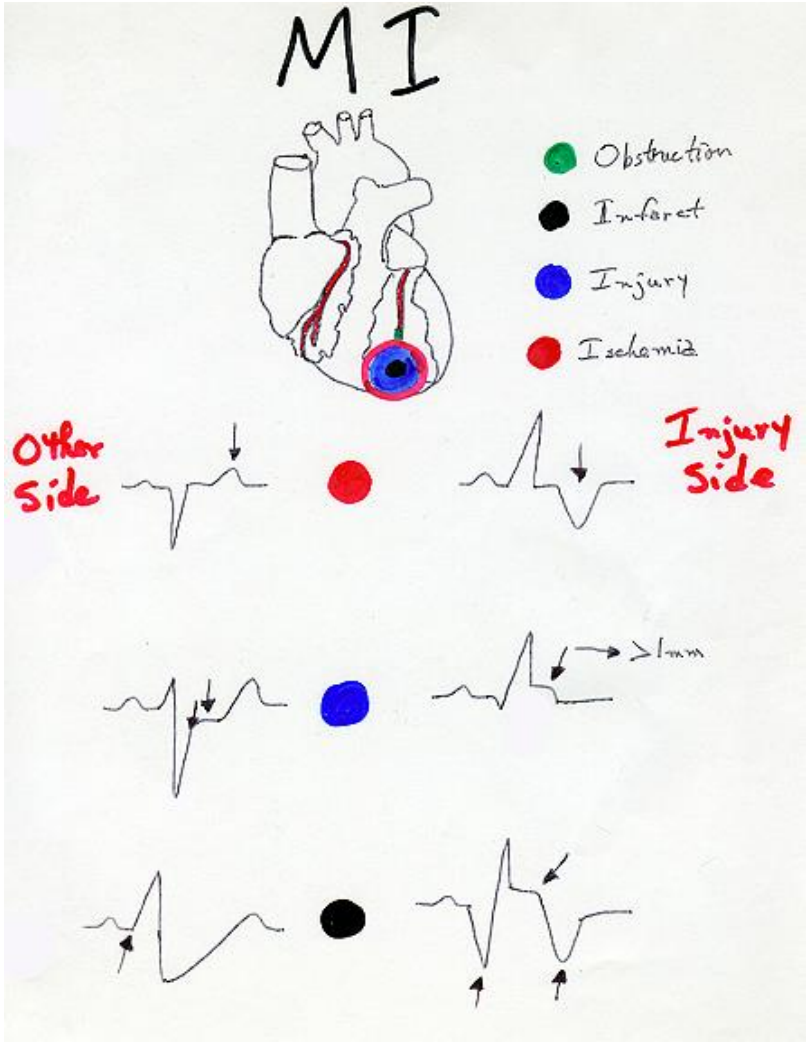
- Reduced Blood Flow (BF) to Anterior Muscle of LV
- Causes Anterior MI
- Leads to Secondary Compromisation
- E.g., Increased Heart Size, Increased Heart Rate
- TACHYarrhythmias: Sinus and Atrial

Arrhythmias Associated with MI's -- 2

Right Coronary Artery Compromisation

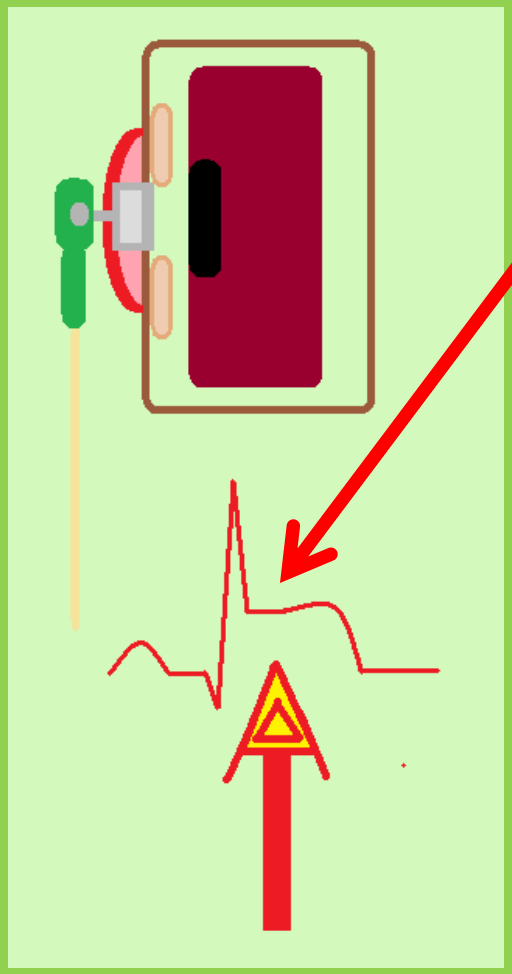
- Reduced BF to Upper Conduction System and to Inferior Region of LV
- Causes Inferior MI
- Due to Reduced BF, Nodes and Fibers Slow Down
- This Causes BRADYarrhythmias: Sinus, Nodal (Junctional) or Varying Degree of Heart Block
  - Junctional: an abnormal heart rhythm resulting from impulses coming from a locus of tissue in the area of the AV node, the "junction" between atria and ventricles.

Myocardial Infarction -- Anatomy



- "Ischemia" goes along with symmetrical T-wave inversion (or elevation),
- "Injury" refers to abnormal ST-segment changes and
- "Necrosis" goes along with abnormal Q waves.

ST Elevation v Depression

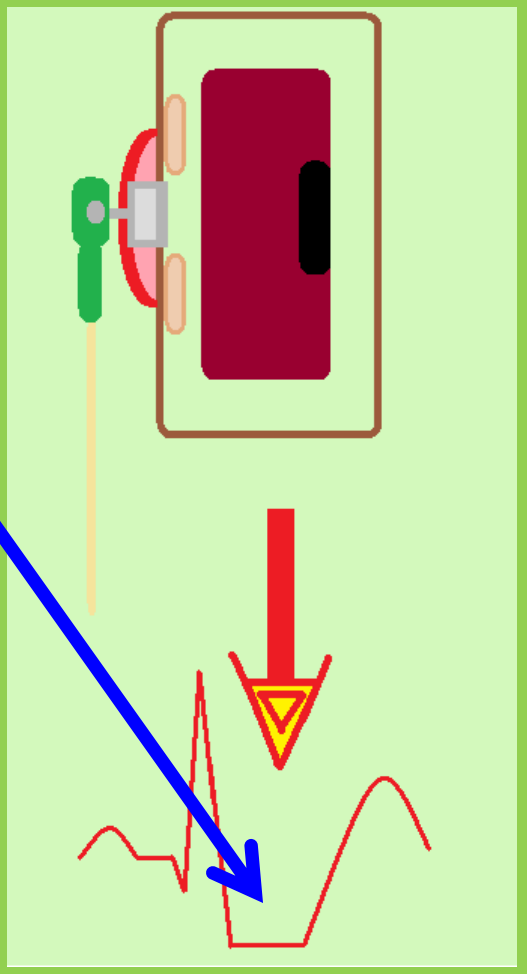


Per “current-of-injury” theory:

ST-segment **elevation** occurs when the injured muscle is located between normal muscle and the corresponding electrode.

ST-segment **depression** occurs when normal muscle is located between the injured tissue and the corresponding electrode.

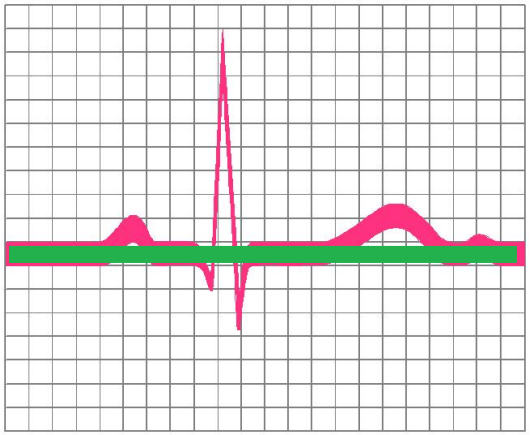
a flow of current to *(systolic current of injury)* or from *(diastolic current of injury)* the injured region of an ischemic heart, due to regional alteration in transmembrane potential

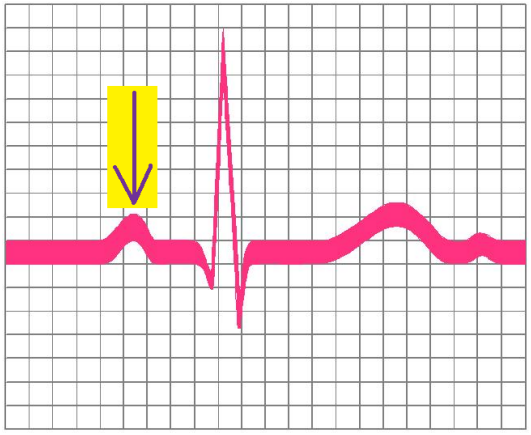


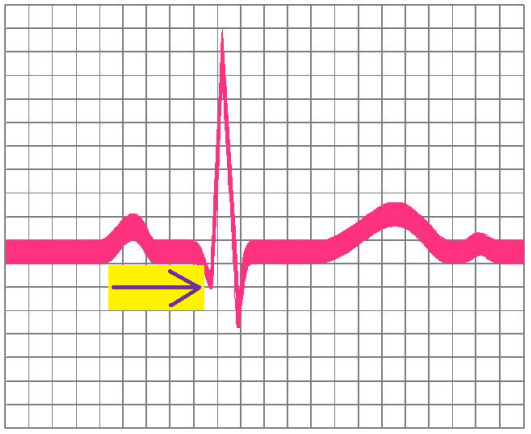
Electrode cap **Electrode wire** **Electrode**

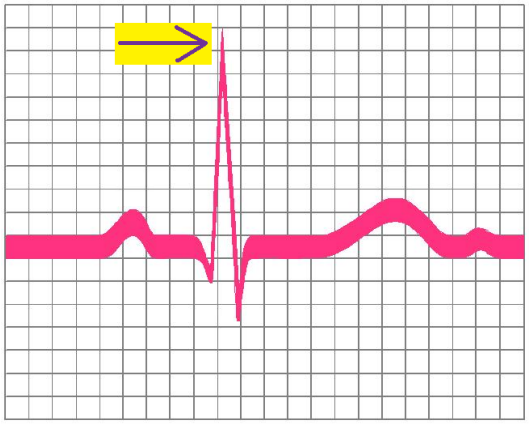
Gel Button **Chest Wall** **Ribs** **Injured Muscle**

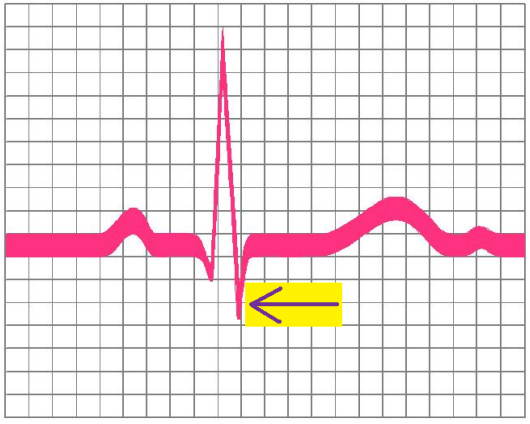
Healthy Heart Muscle

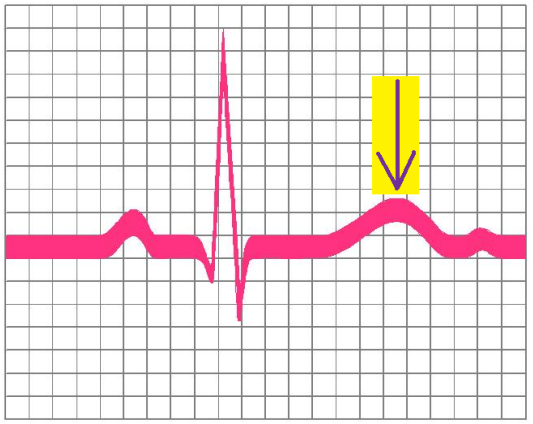


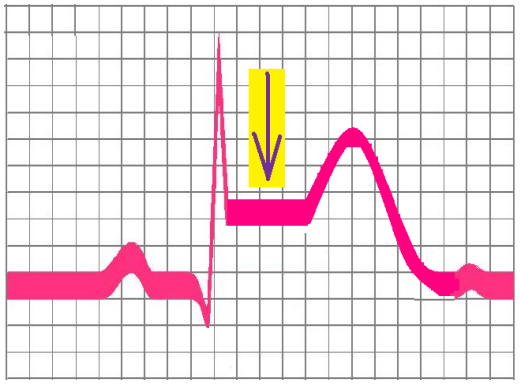








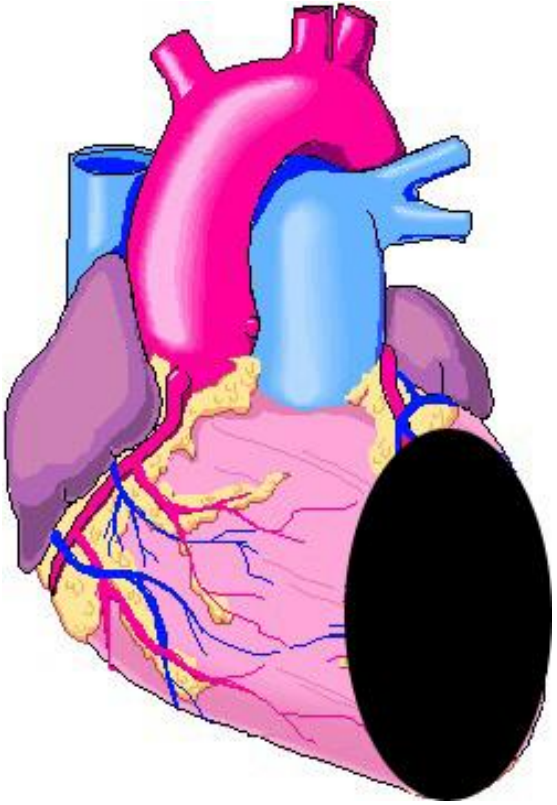


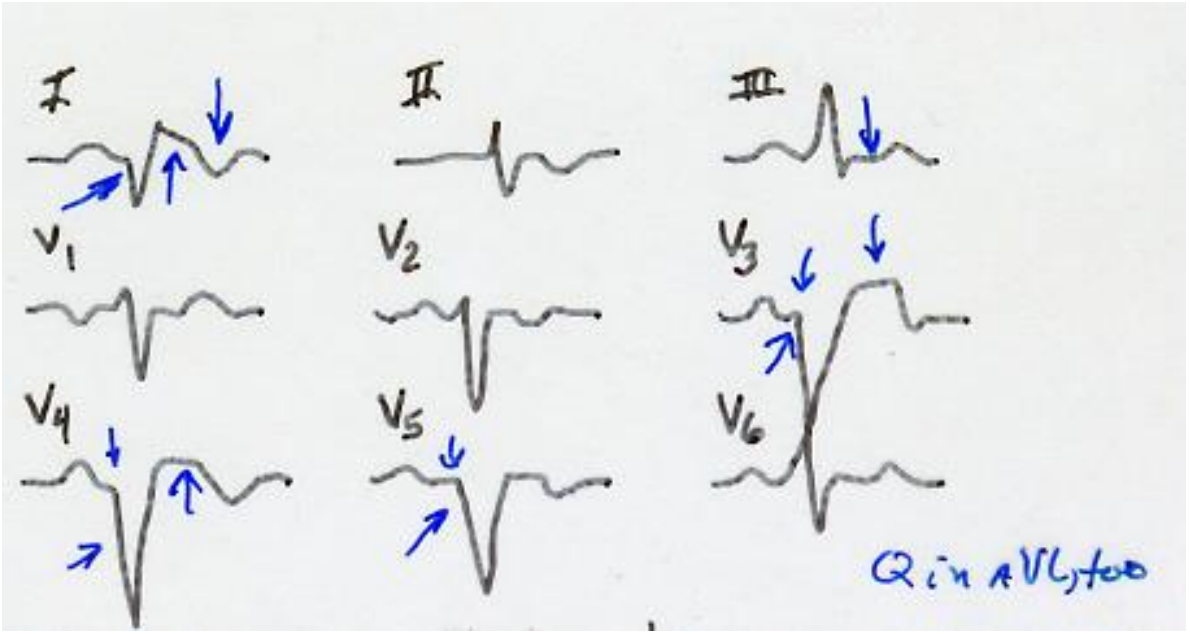


Highlights of Elementary MI Diagnosis

Anterior MI	Lateral MI	Inferior MI – aka Diaphragmatic	Posterior MI
LAD Blockage	CXA Blockage	R or L CA Blockage	RCA Blockage
Q in V_1, V_2, V_3, V_4	Q in I and aVL	Q in II, III, aVF	R in V_1, V_2 – use mirror if = Q = PMI V_1 and V_2 ST depression held to mirror and looks elevated = PMI

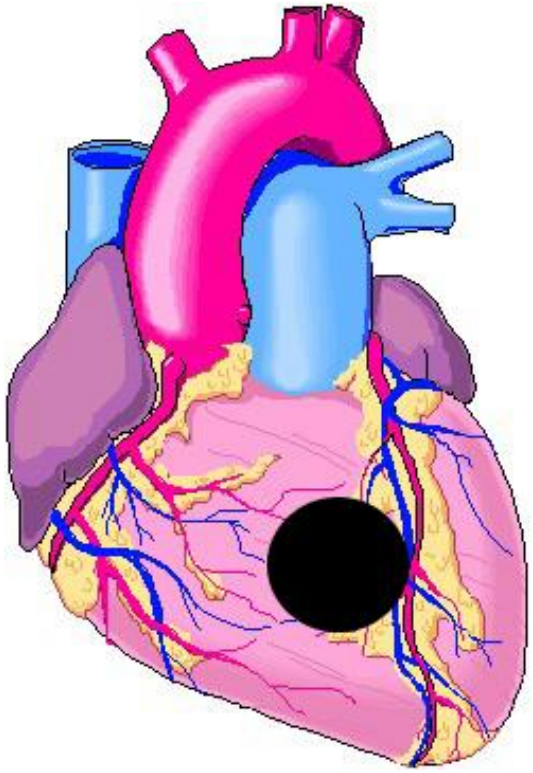
Anterior Infarcts -- Anterolateral

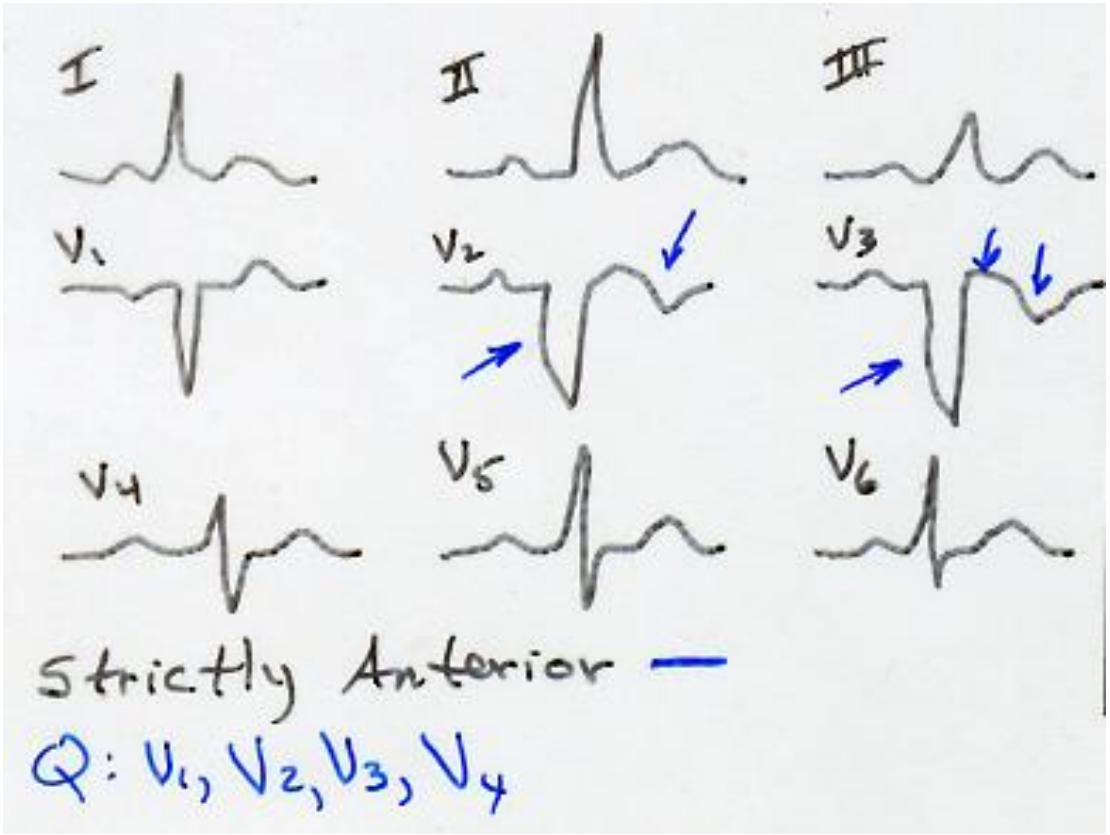




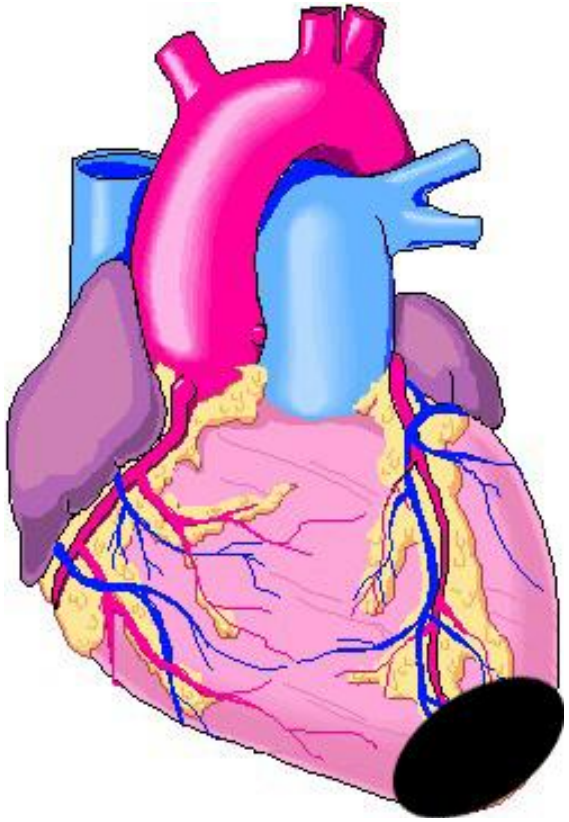
- Significant Q wave is between a quarter to a third the height of the QRS complex
- or more than 1 mm wide (1 little square on EKG paper)

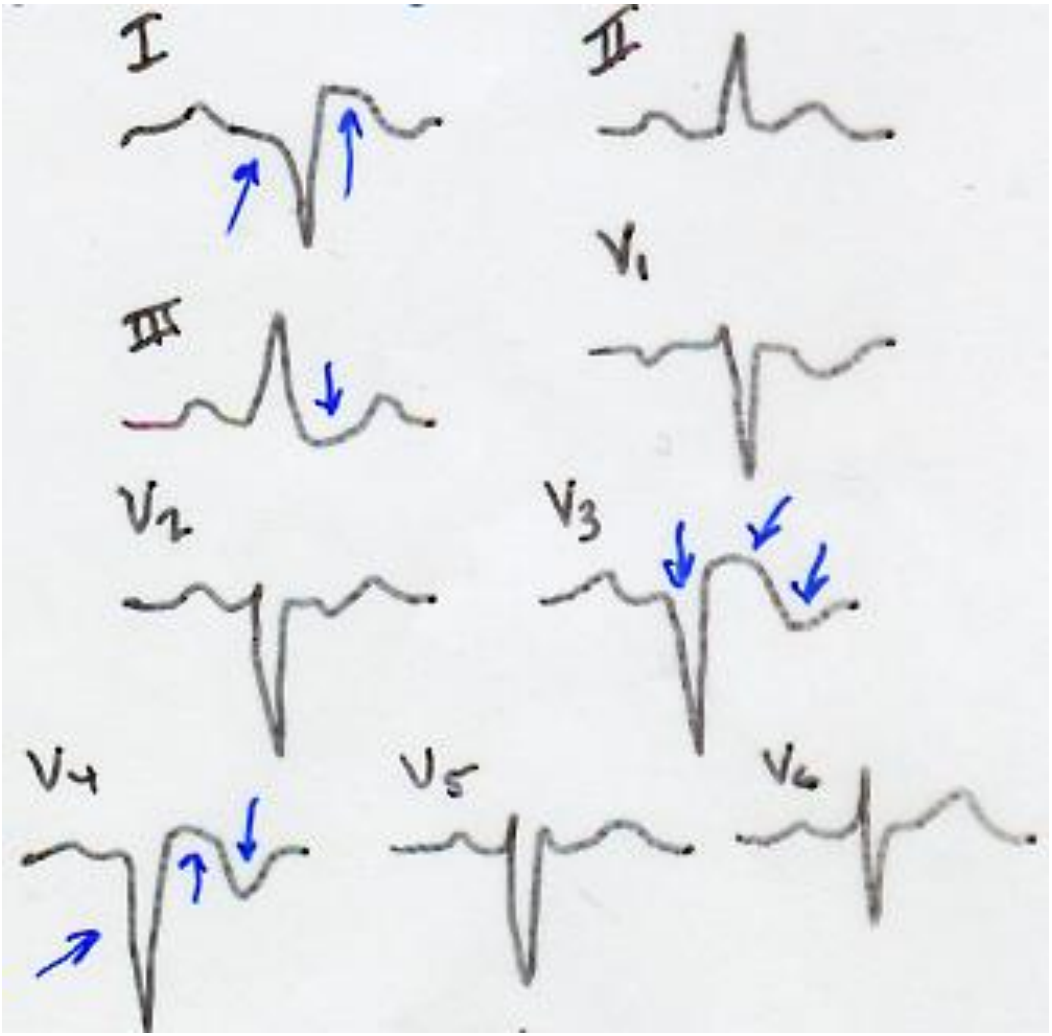
Strictly Anterior Infarct



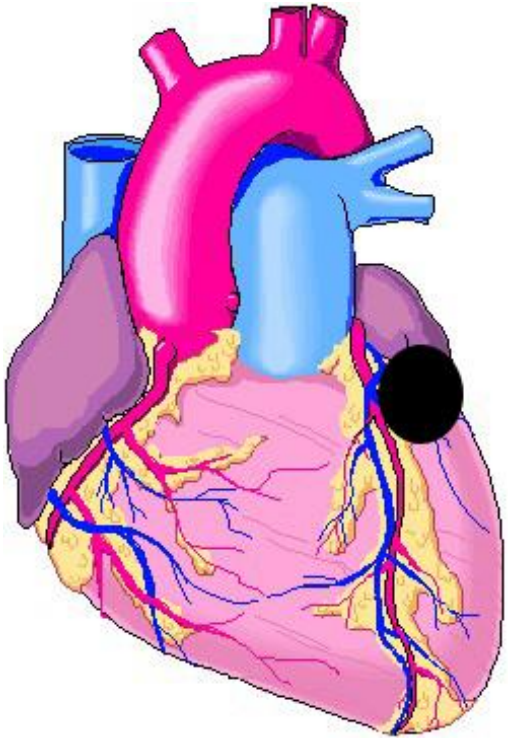


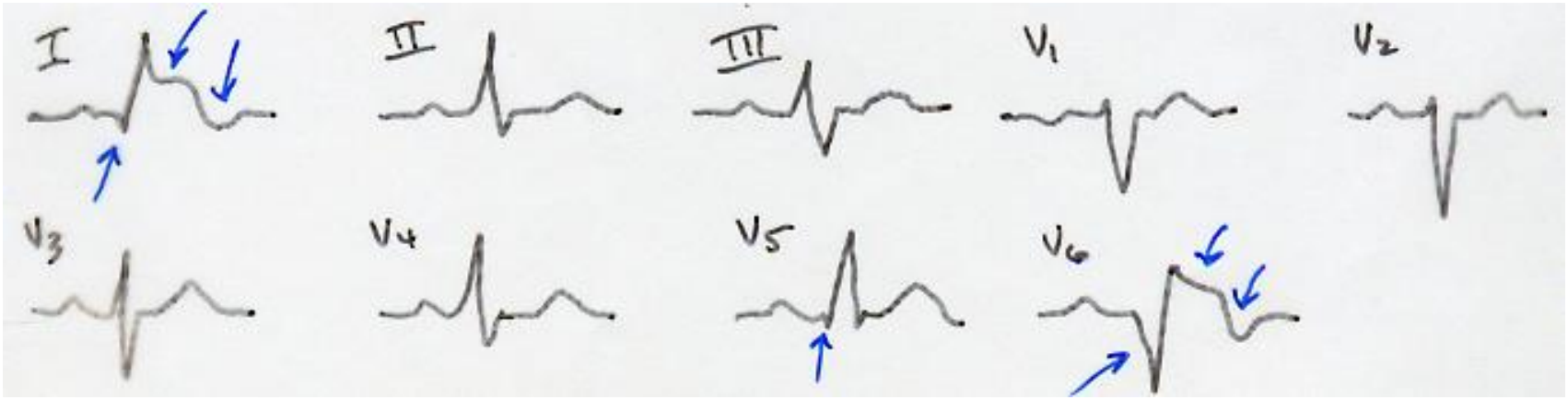
Apical Infarct



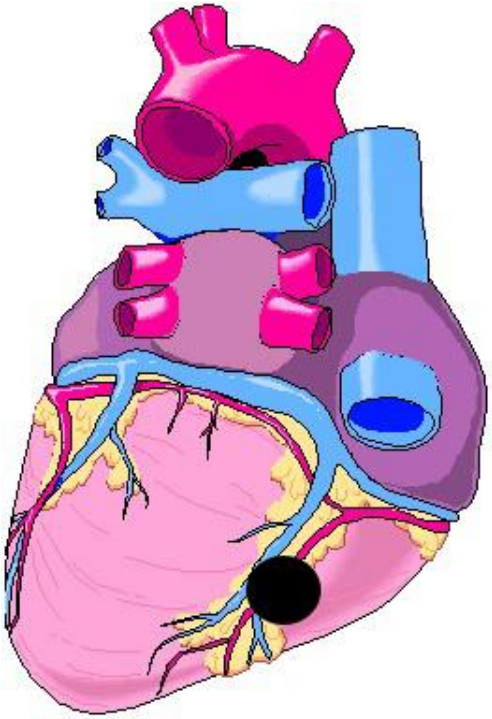


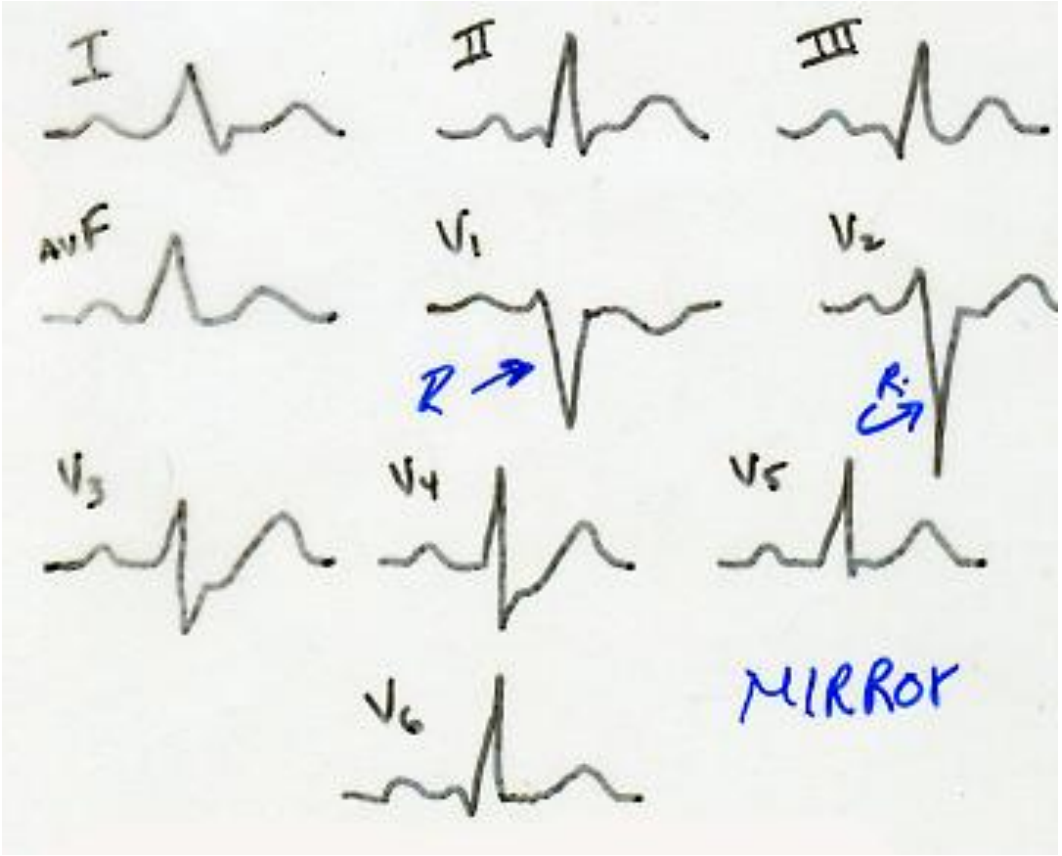
Antero-Basal Infarct





Posterior Wall Infarcts – Strictly Posterior

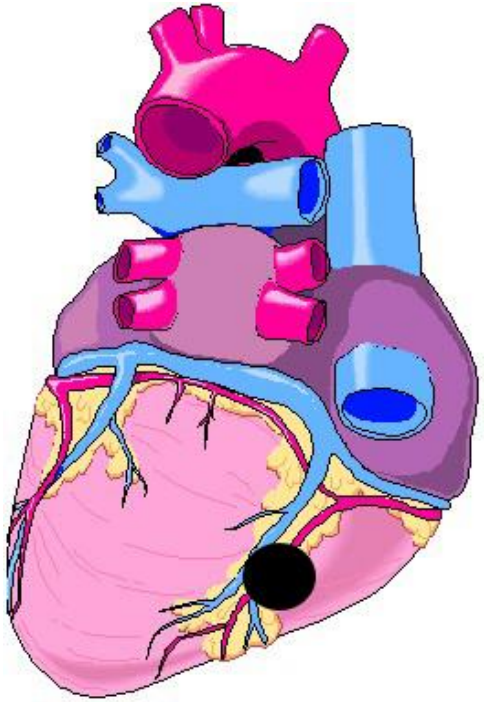


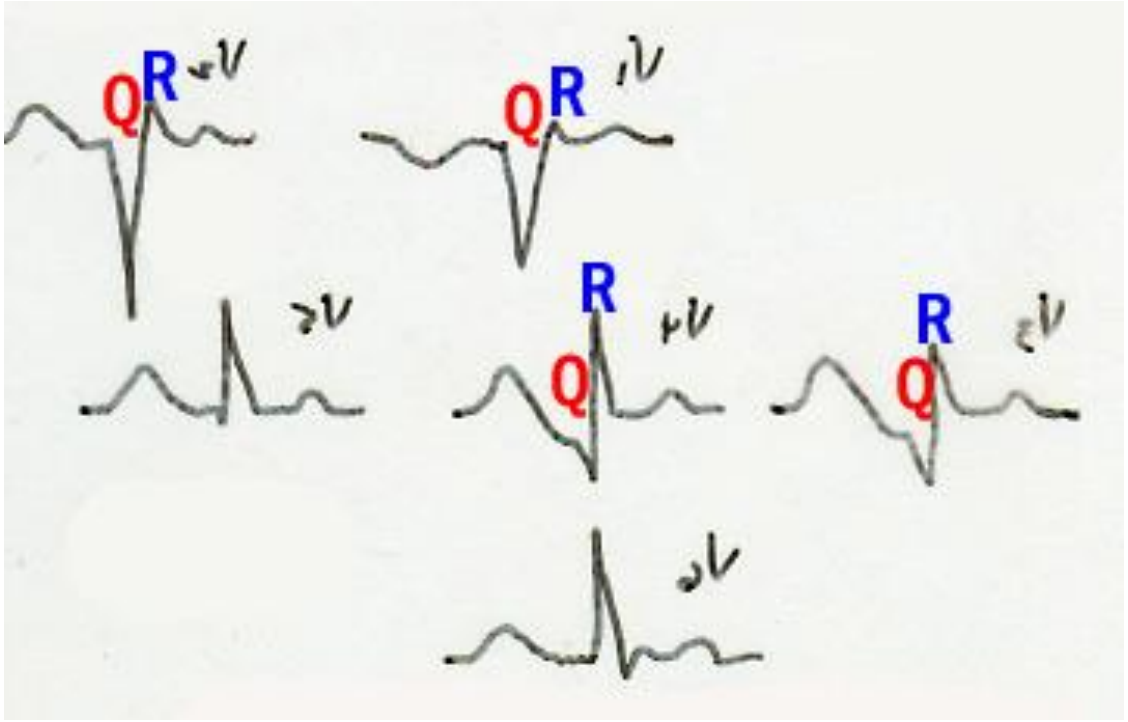


Posterior MI Diagnostic Trick

- Posterior infarcts are tricky to dx
- A mirror helps: flip upside down and backwards to look at precordial leads
- S waves in normal presentation look like R waves in mirror
- R waves in normal presentation look like Q waves in mirror

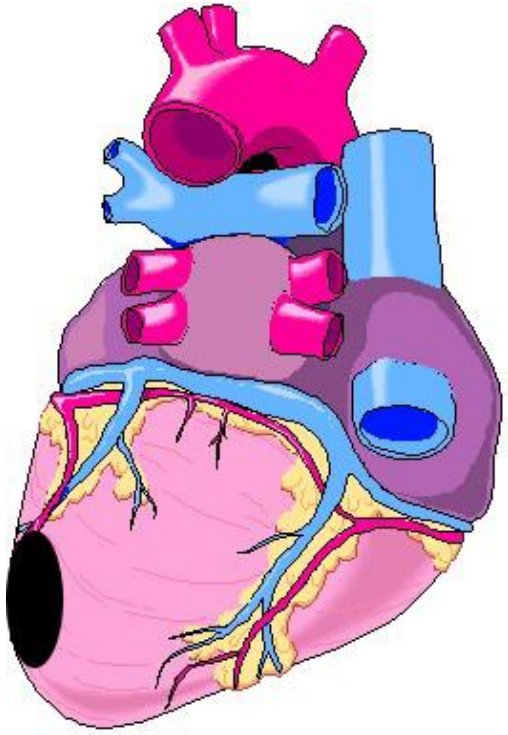
Posterior Wall Infarcts – Strictly Posterior

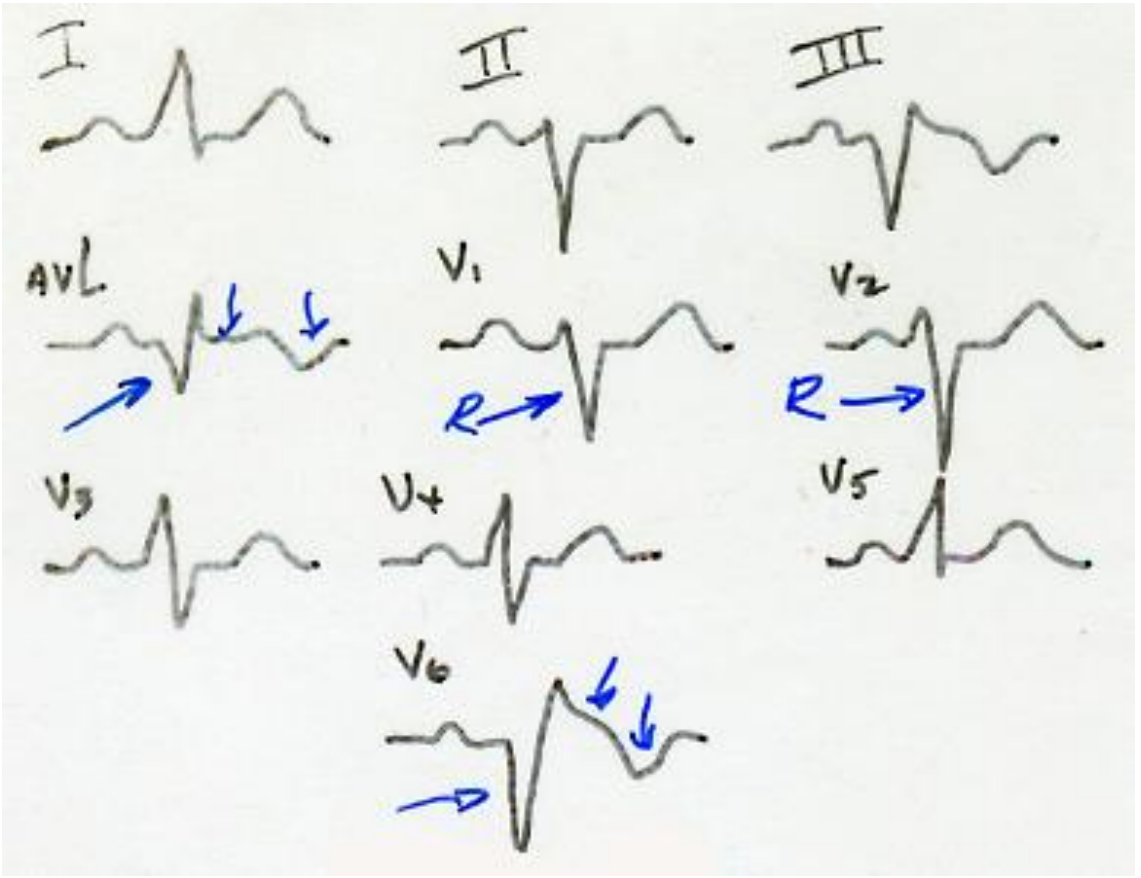




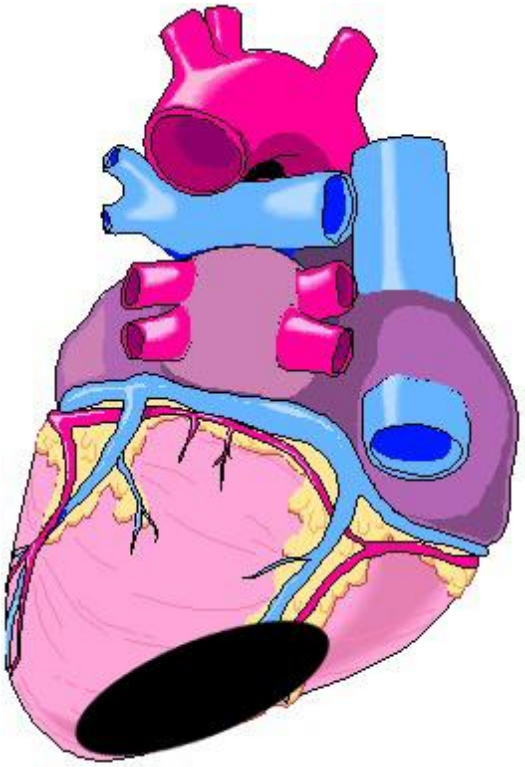
- Flipped and mirrored

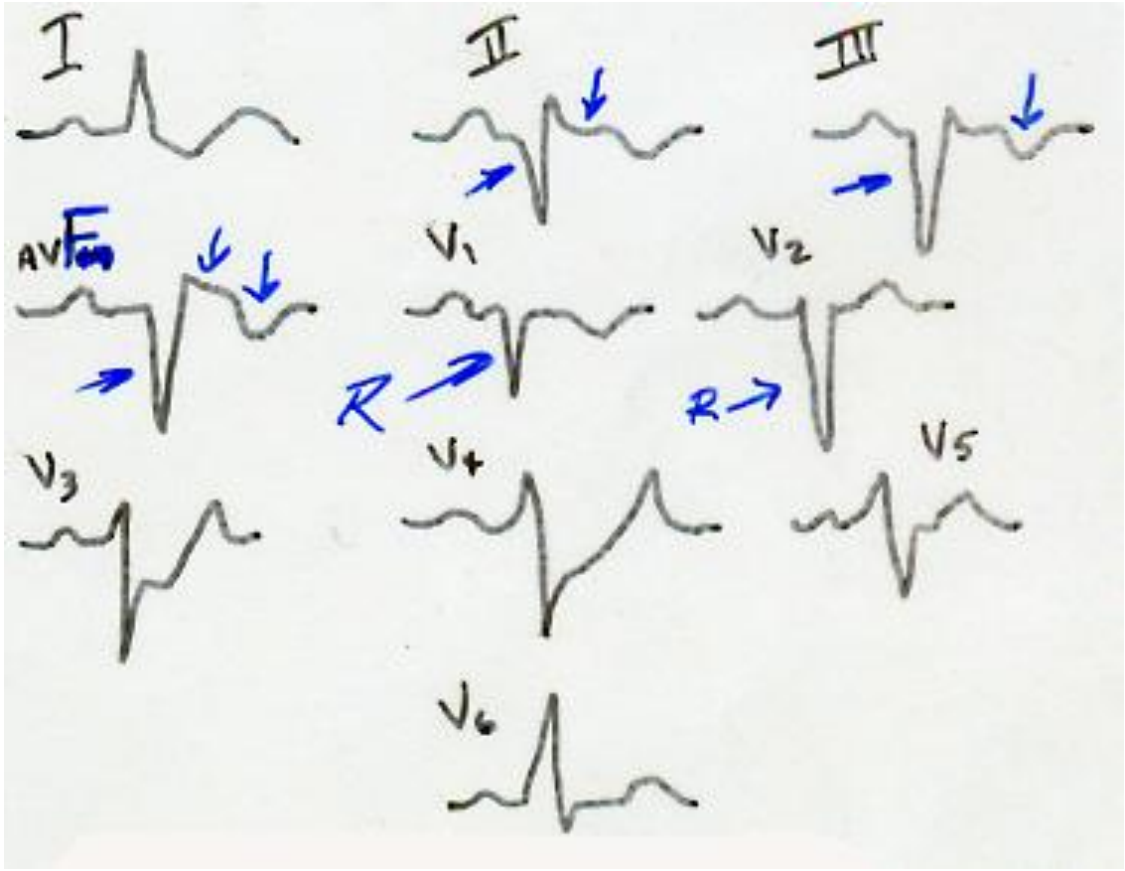
Postero-Lateral Infarct



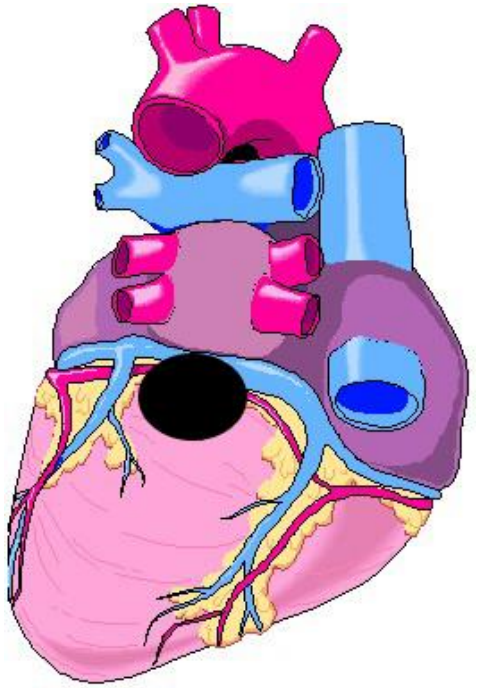


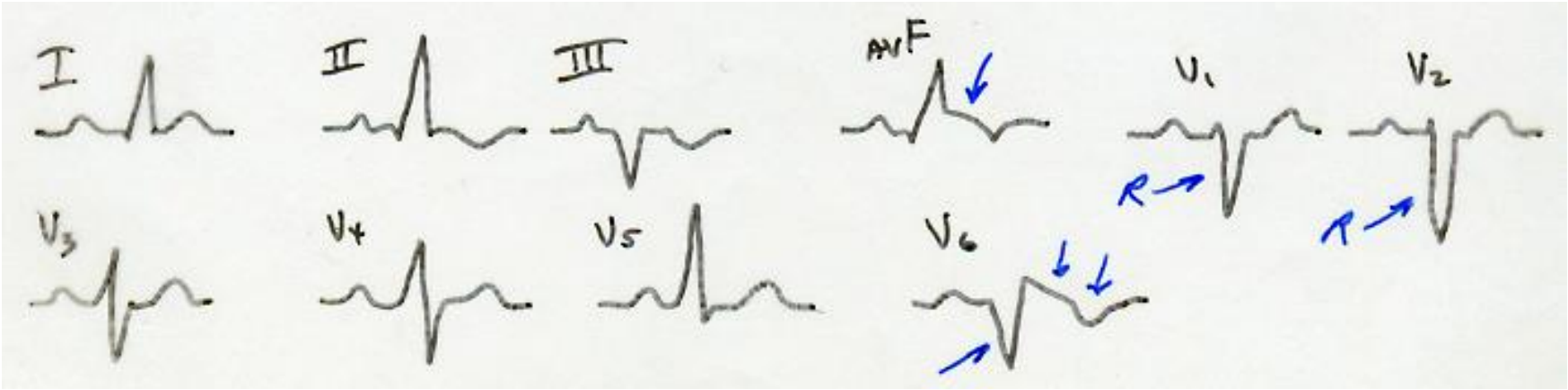
Postero-Inferior Infarct



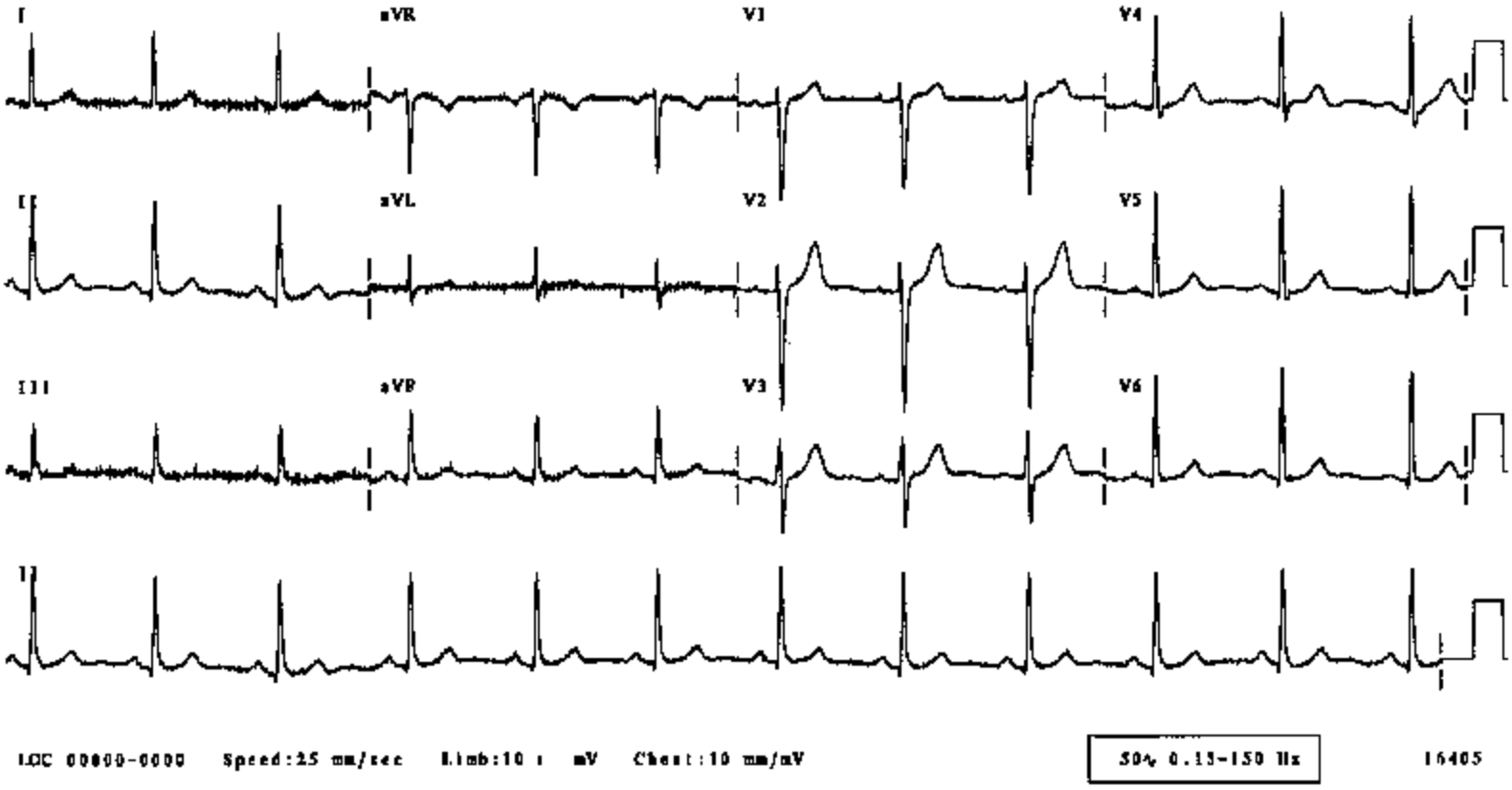


Postero-Basal Infarct

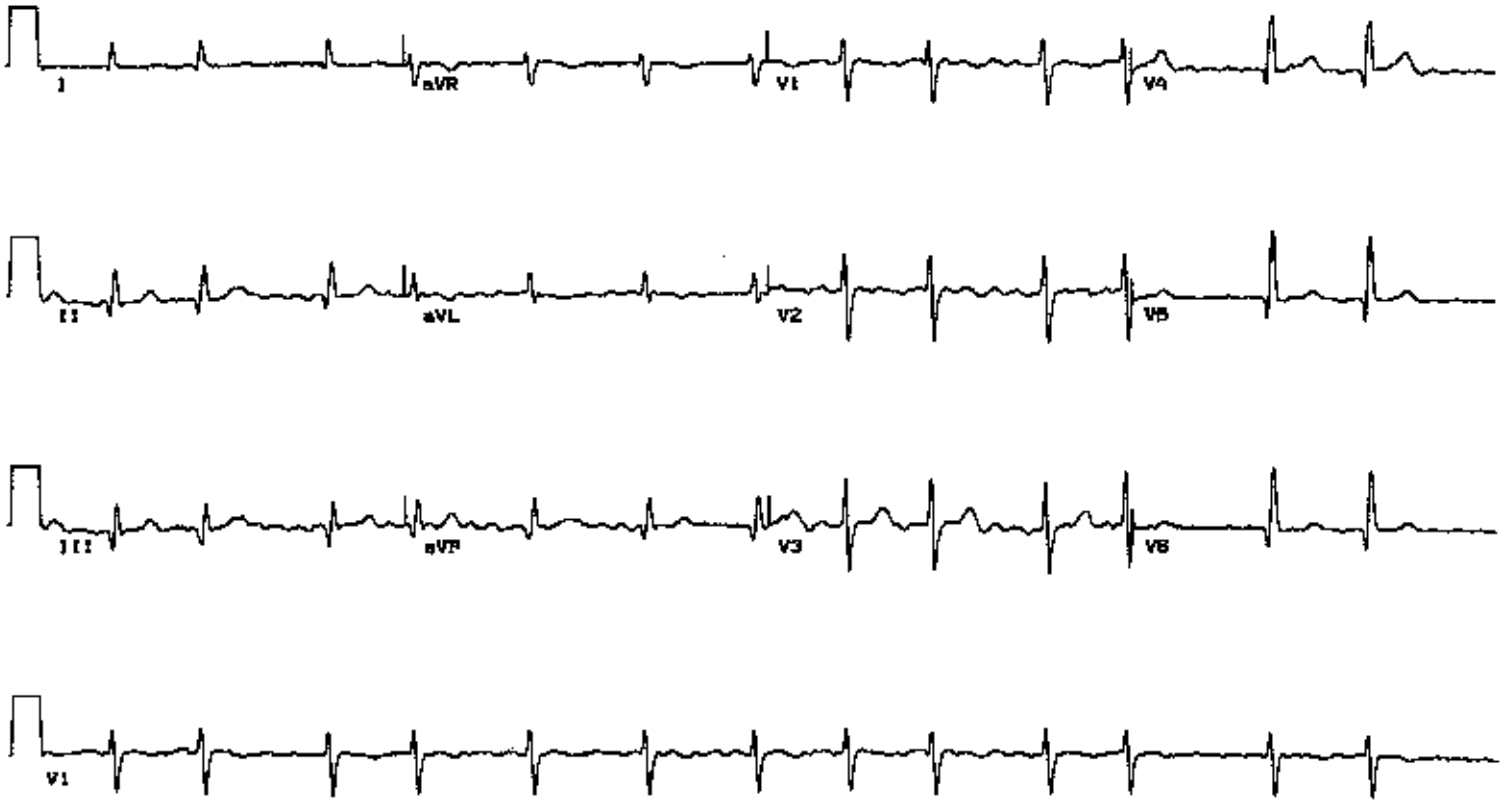




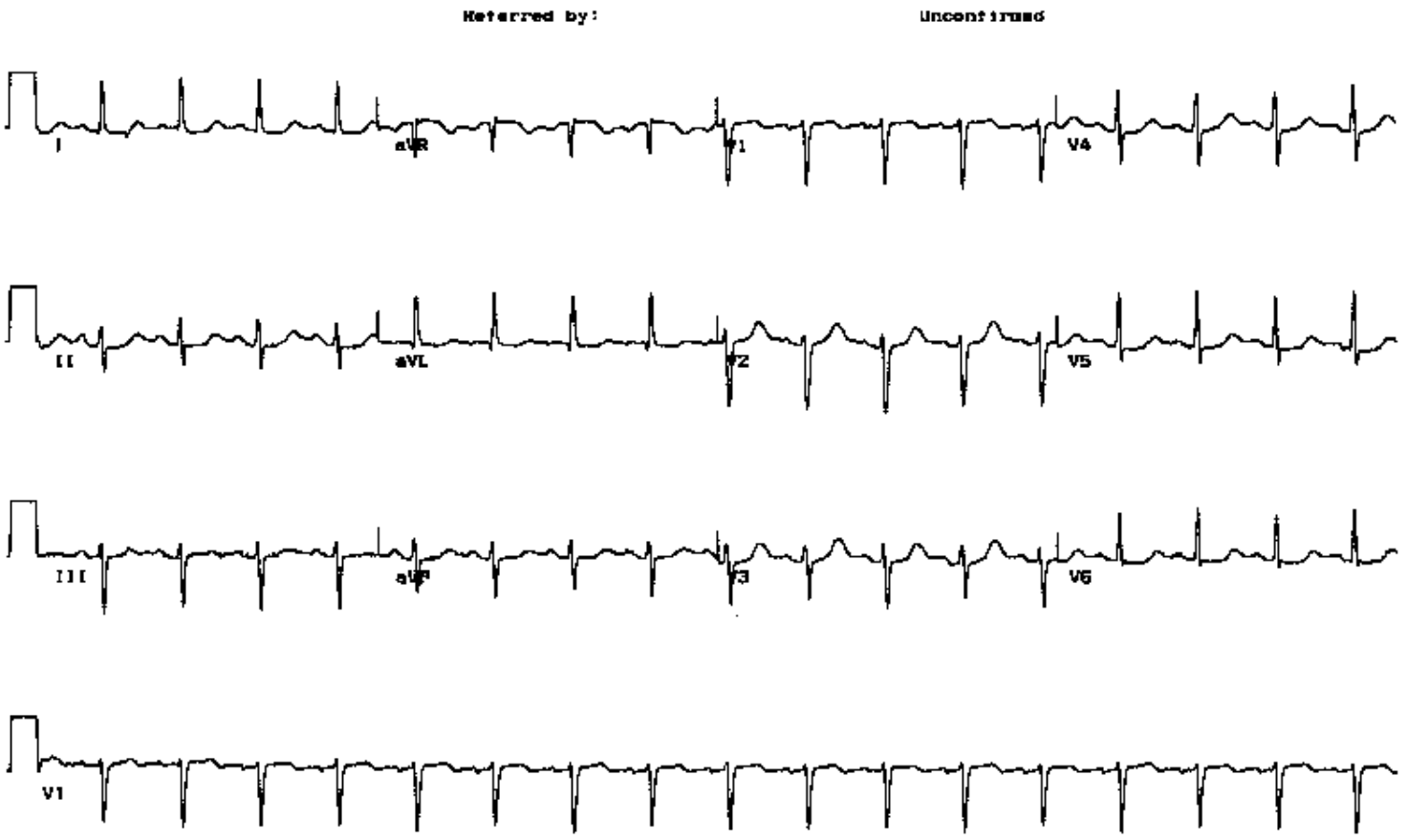
Normal 12-Lead EKG



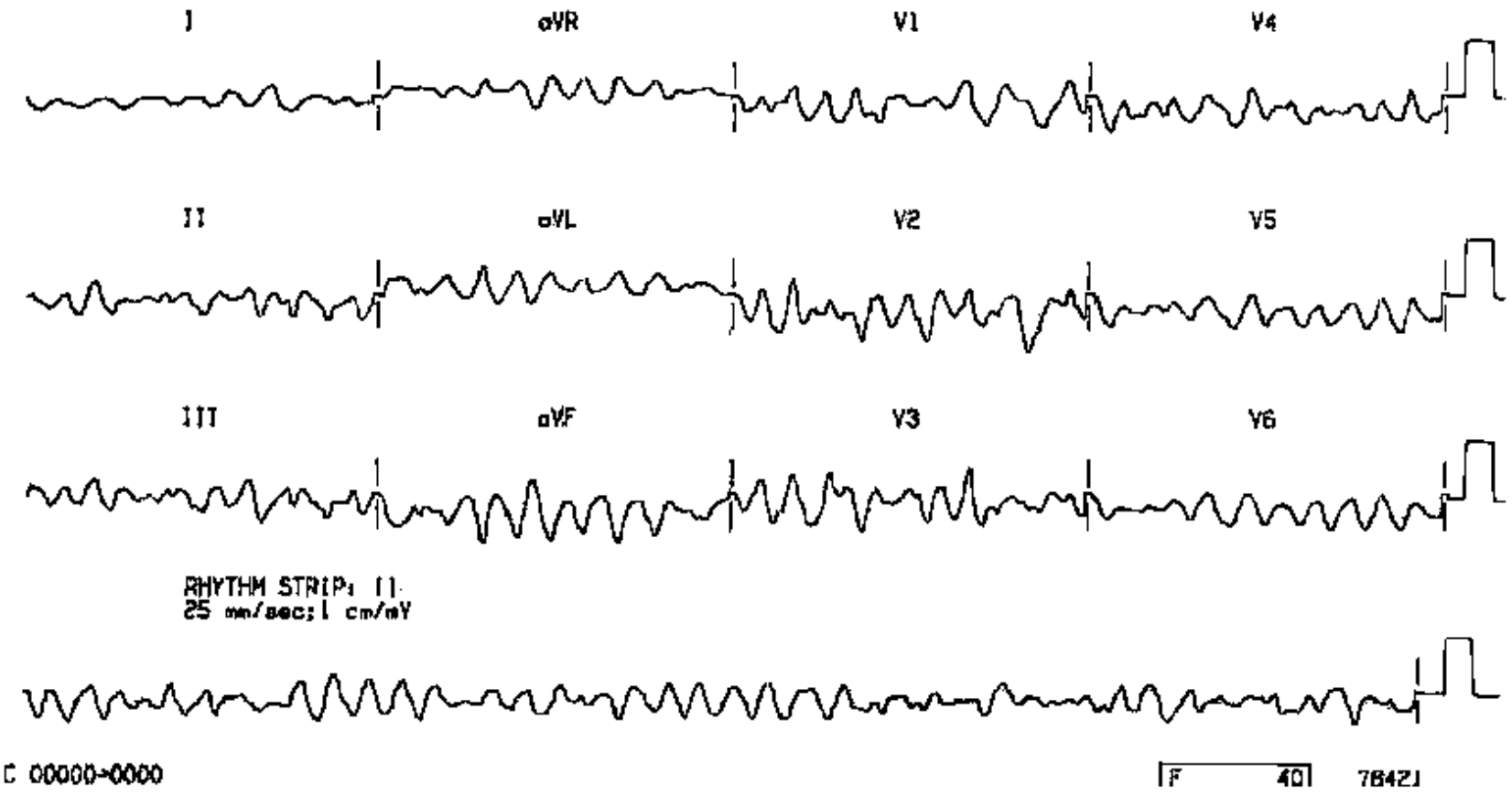
Old MI – Inferior Wall



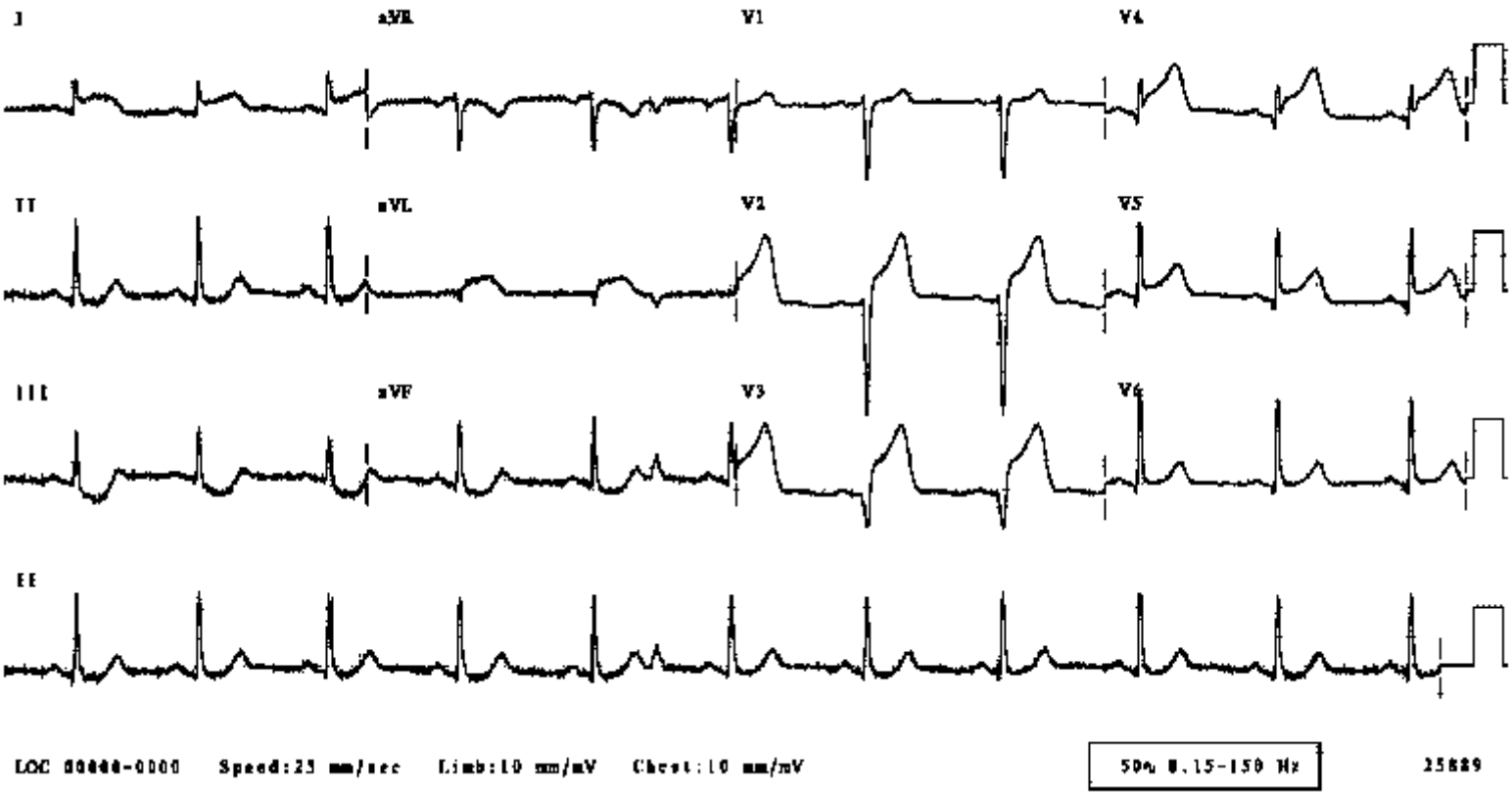
Sinus Tachycardia



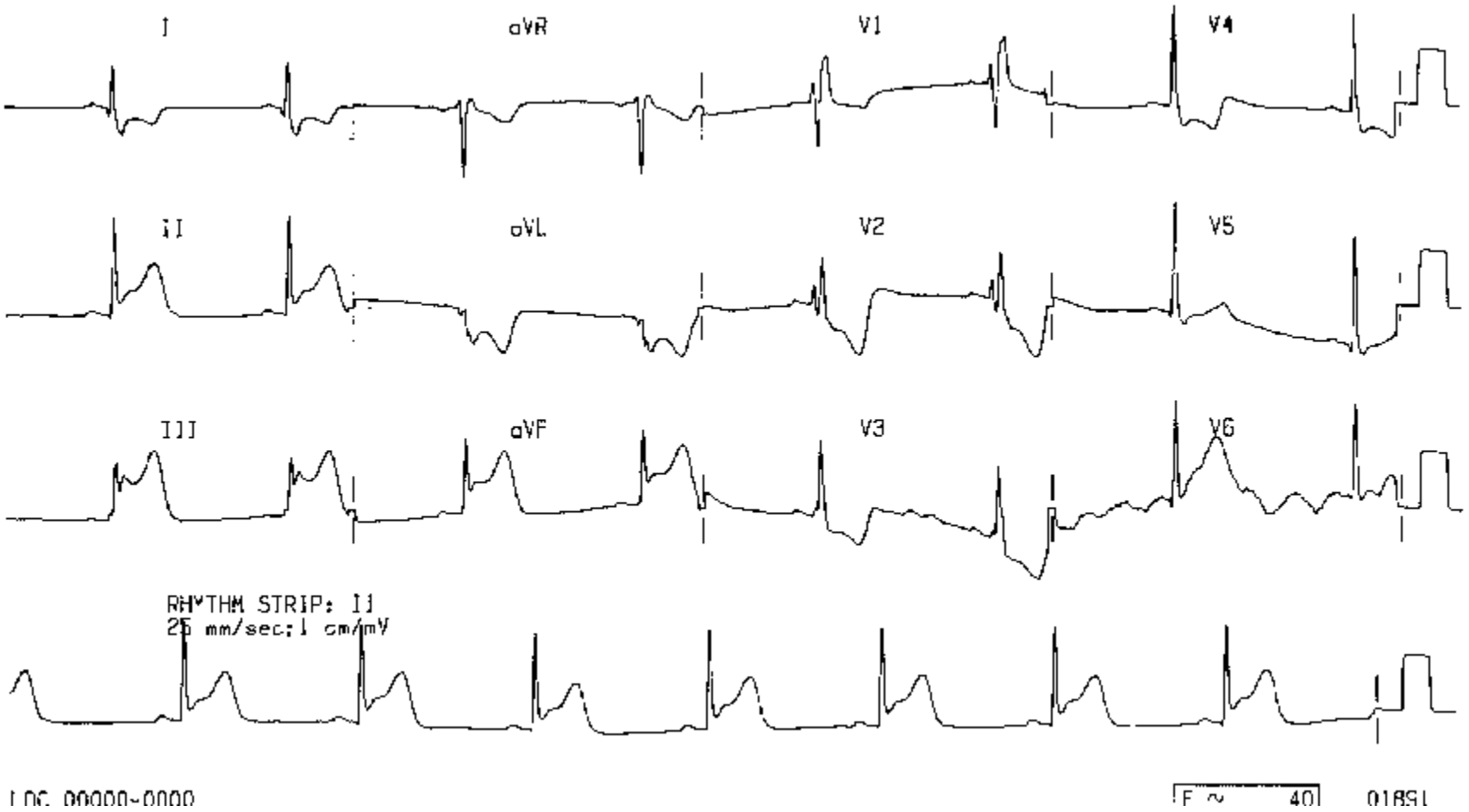
V Fib


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV

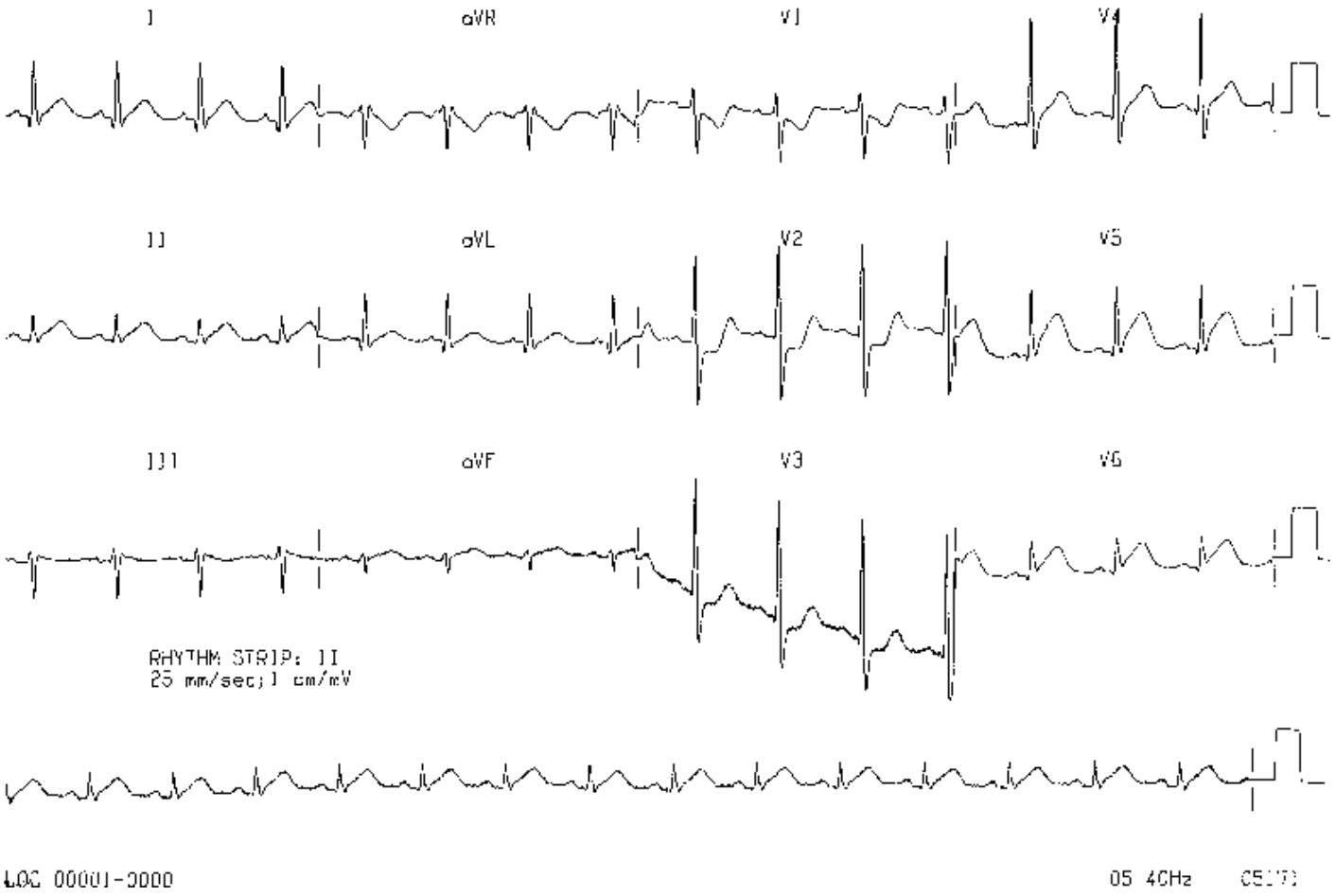
Anterolateral Infarct



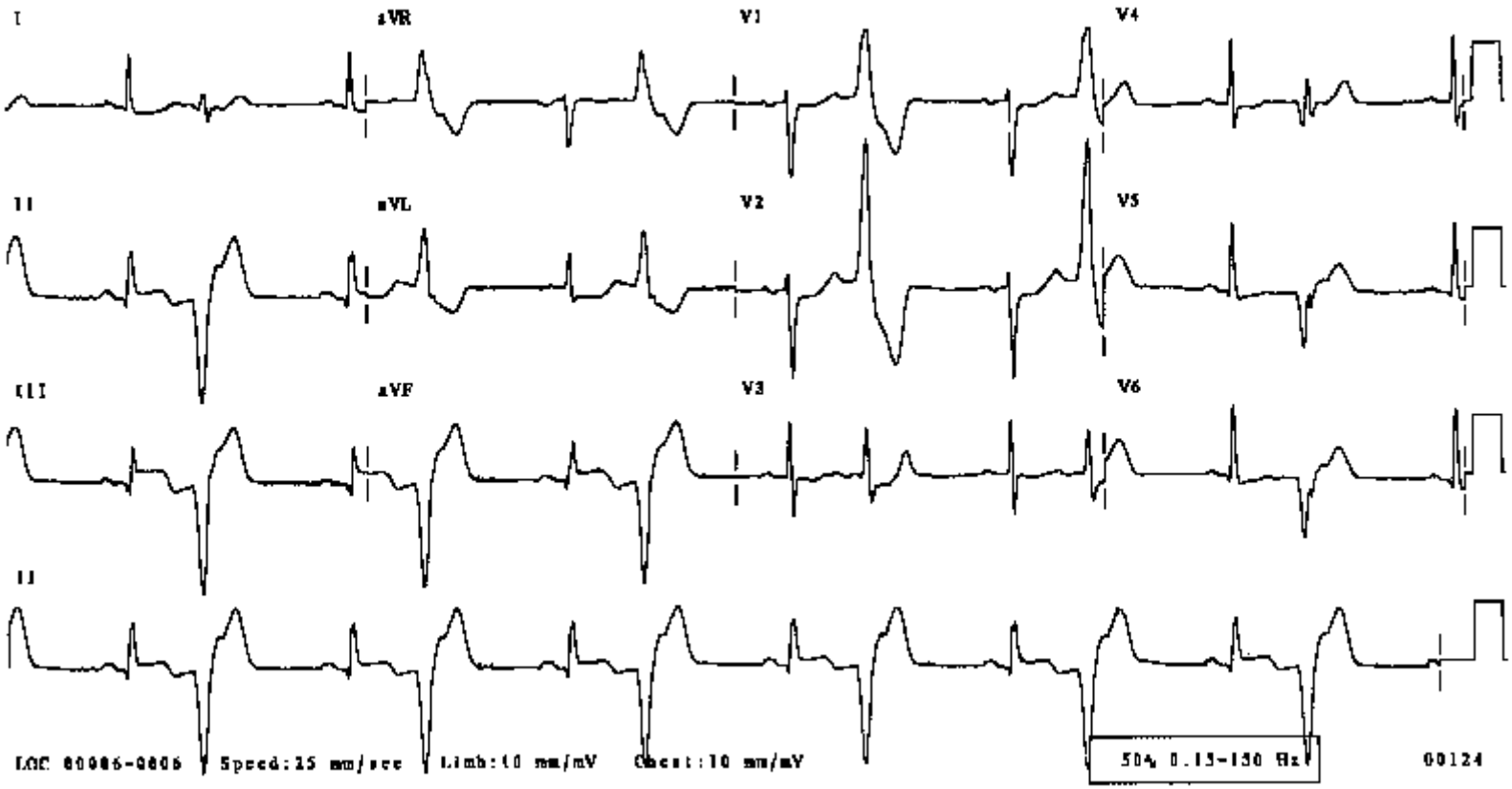
Inferior Wall Infarct



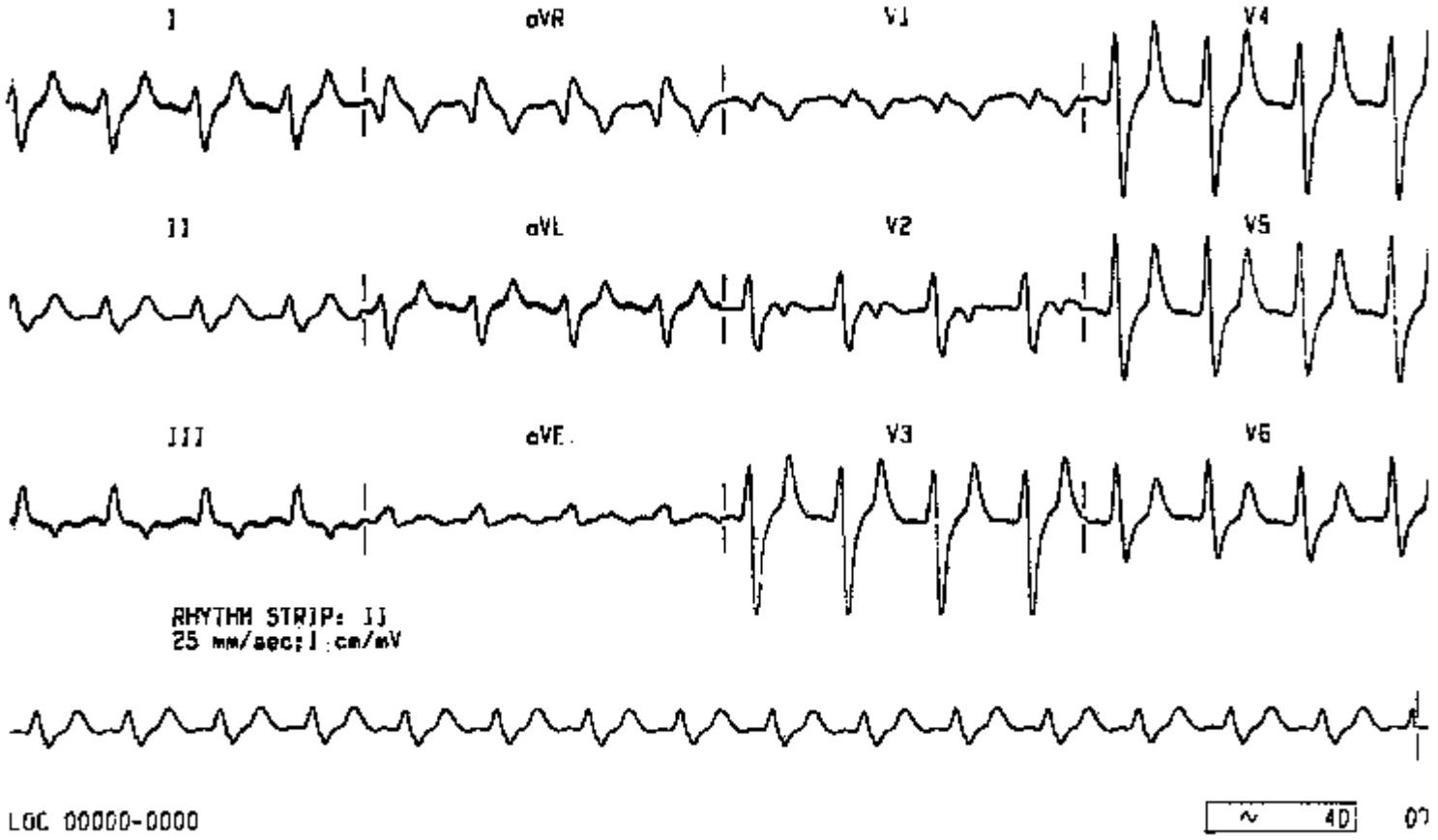
Postero-Lateral Infarct



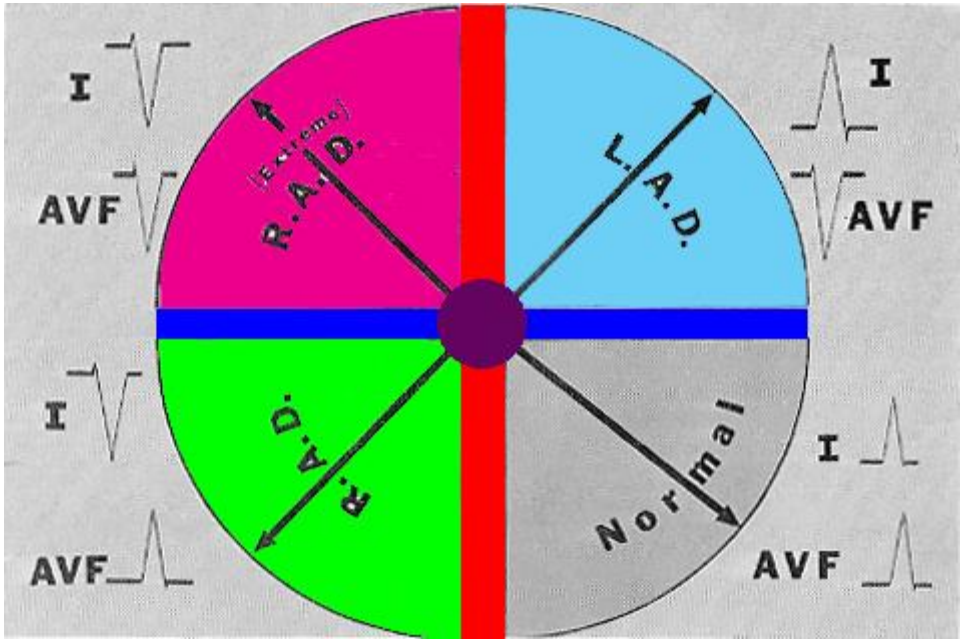
Unifocal PVC's -- Bigeminy



Hyperkalemia



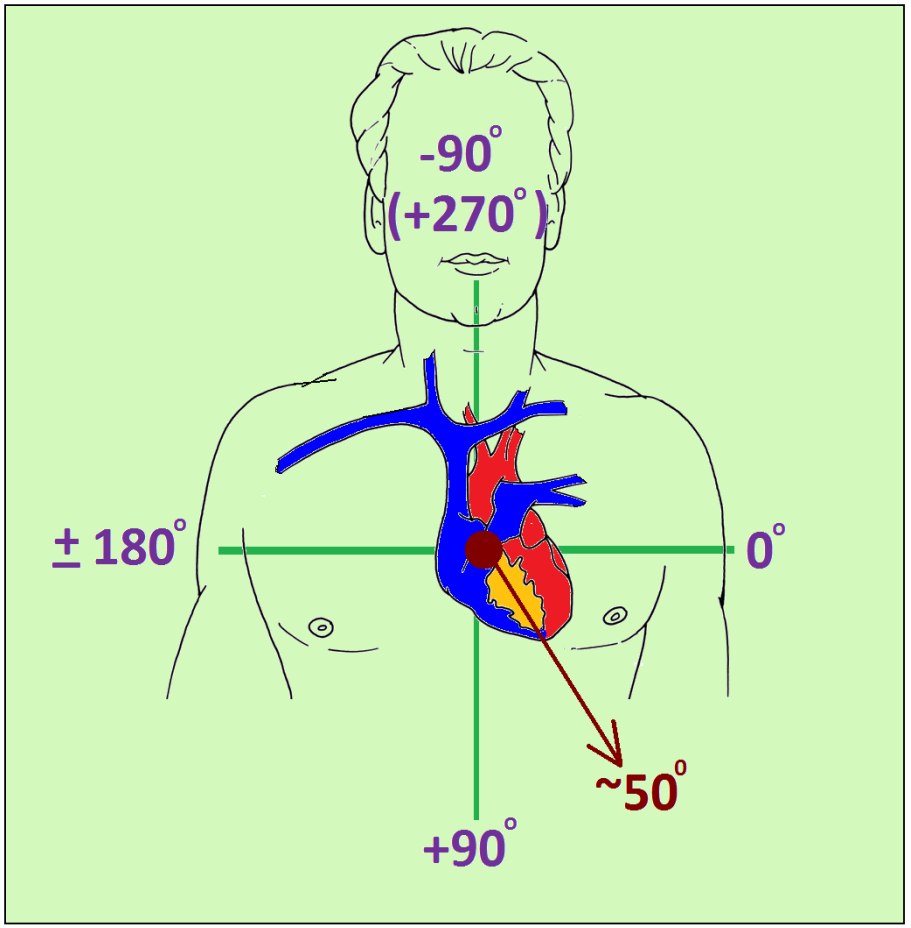
Axis Deviation



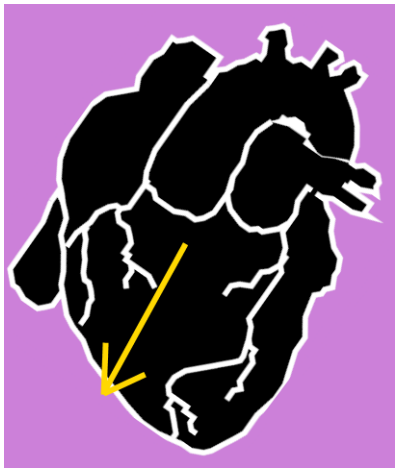
Axis Deviations

Normal axis

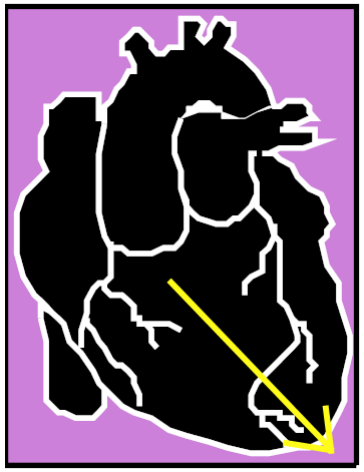
- Back, down and to the left
- Due to greatest force from the left ventricle
- Left ventricle ejects blood to whole body
- Septum REpolarizes from RIGHT to LEFT – Septum **DEpolarizes** from **LEFT** to **RIGHT** (left BB discharges first) ADJUST your NOTES!
- Average vector originates from AV node (to the left, at right)
- Graphically, vector (right) – is between 0° and +90°

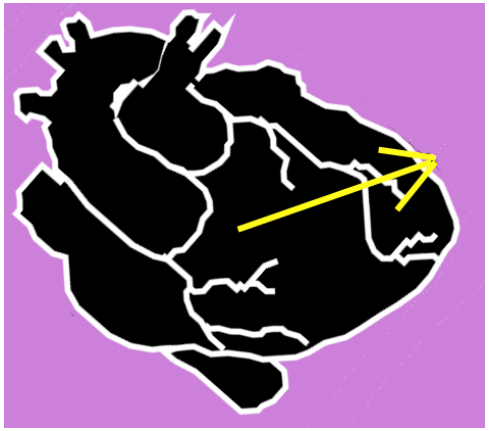


If heart changes position in the chest, vector FOLLOWS the direction of the heart



Right Axis Deviation





Left Axis Deviation

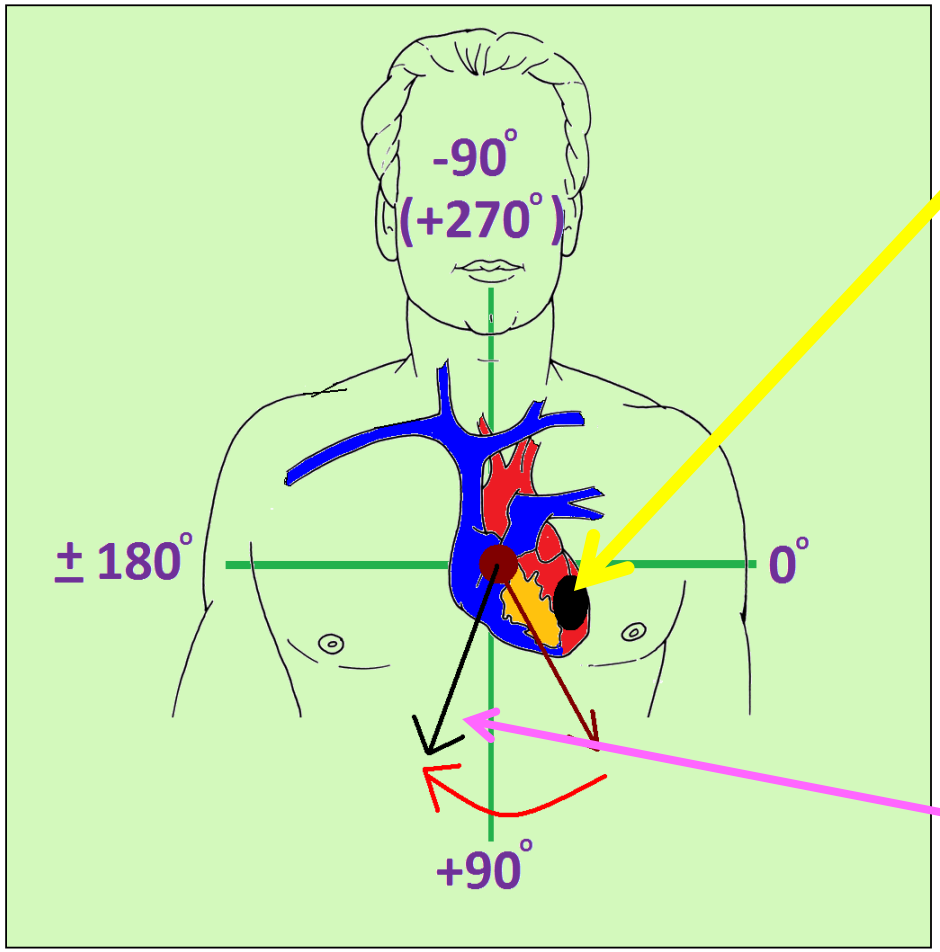
Causes

- May be normal (dextrocardia)
- Smokers
- Cor pulmonale
- COPD
- RVH
- RBBB

Causes

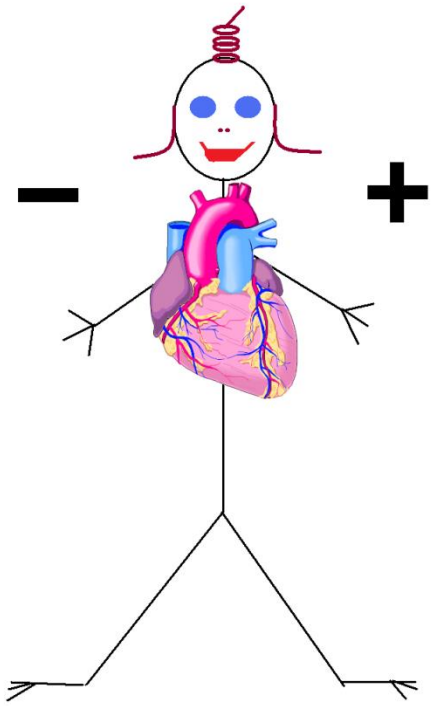
- May be normal
- Obesity
- Pregnancy
- COPD
- LVH
- LBBB
- Abdominal tumor
- Ascites

Deviation of Axis Due to Infarction

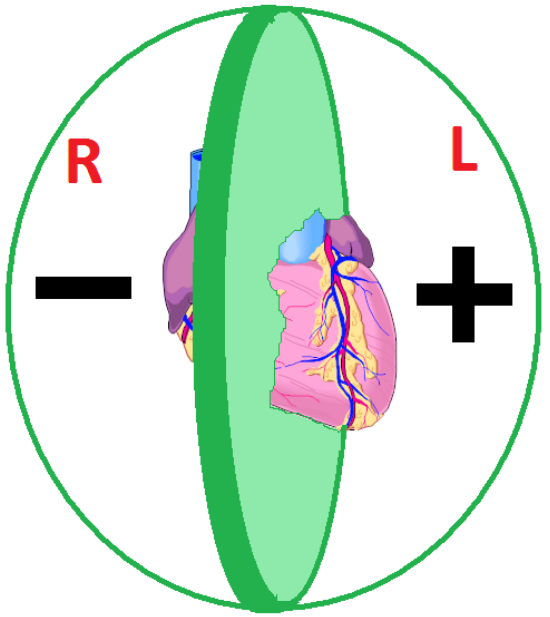


- With MI, a part of the heart dies
- Dead myocardium loses its ability to conduct (scar tissue)
- Dead myocardium leaks K^+ proportional to area of death
- K^+ causes lingering Q wave of previous MI
- Vector shifts AWAY from site of infarction

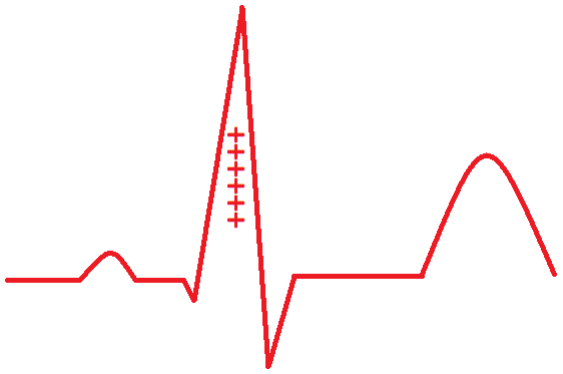
Approximating the Axis – Use Lead I



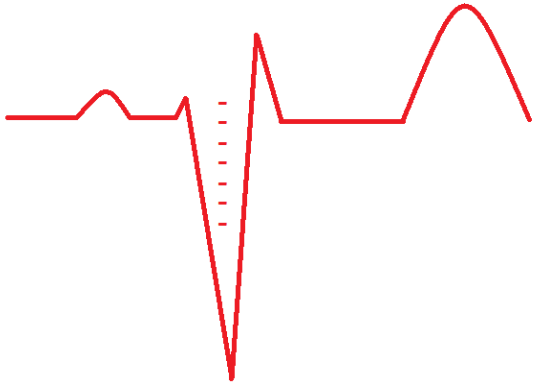
Lead I: L (+); R (-)



“split into” R (-) and L (+) hemispheres

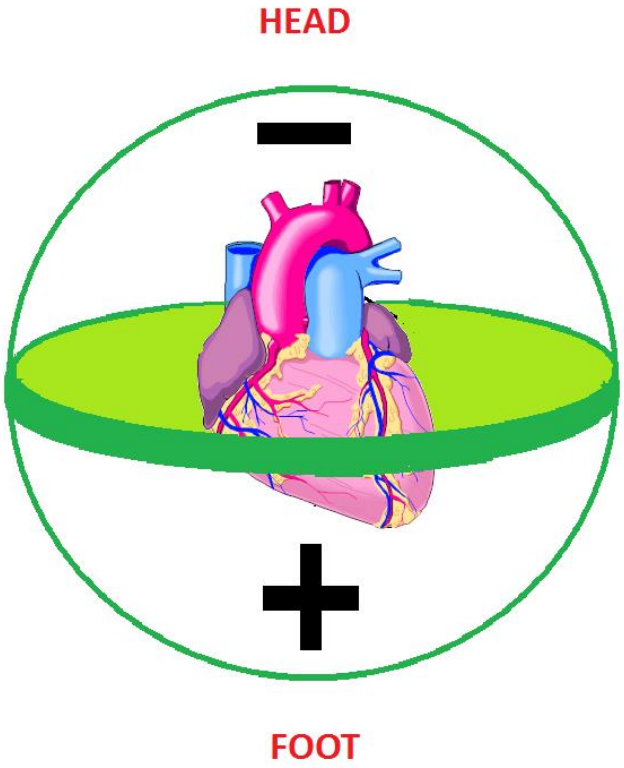


- If QRS in I is primarily upward (positive)
- Vector points LEFT
- Does NOT equal LAD
- 0 - +90 is normal
- and 0 – -90 is LAD
- NOT enough info, yet, to dx LAD



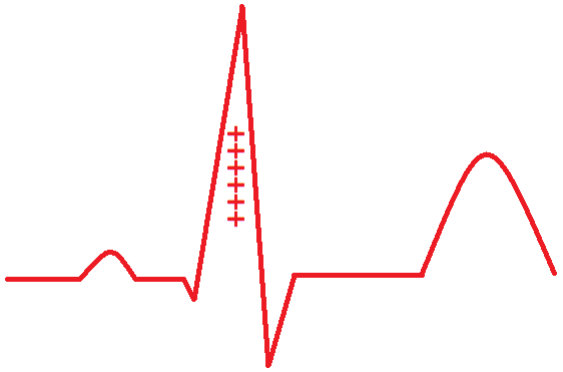
- If QRS in I is primarily downward (negative)
- Vector points RIGHT
- This IS equal to RAD

Lead I does NOT give us enough information – NEED aVF (F = FOOT!)

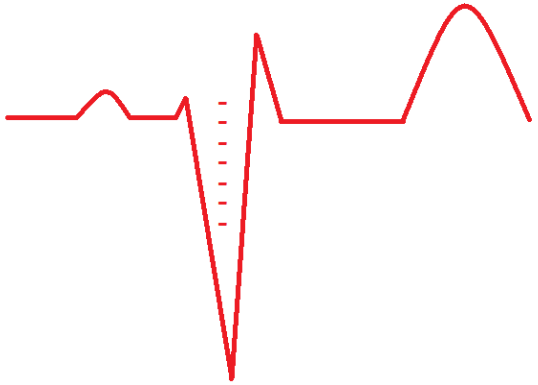


- Draw sphere around body, again
- Divide in to TOP and BOTTOM hemispheres
- F = FOOT!!!! Remember!!!!, i.e., BOTTOM of heart

Look for QRS in aVF for

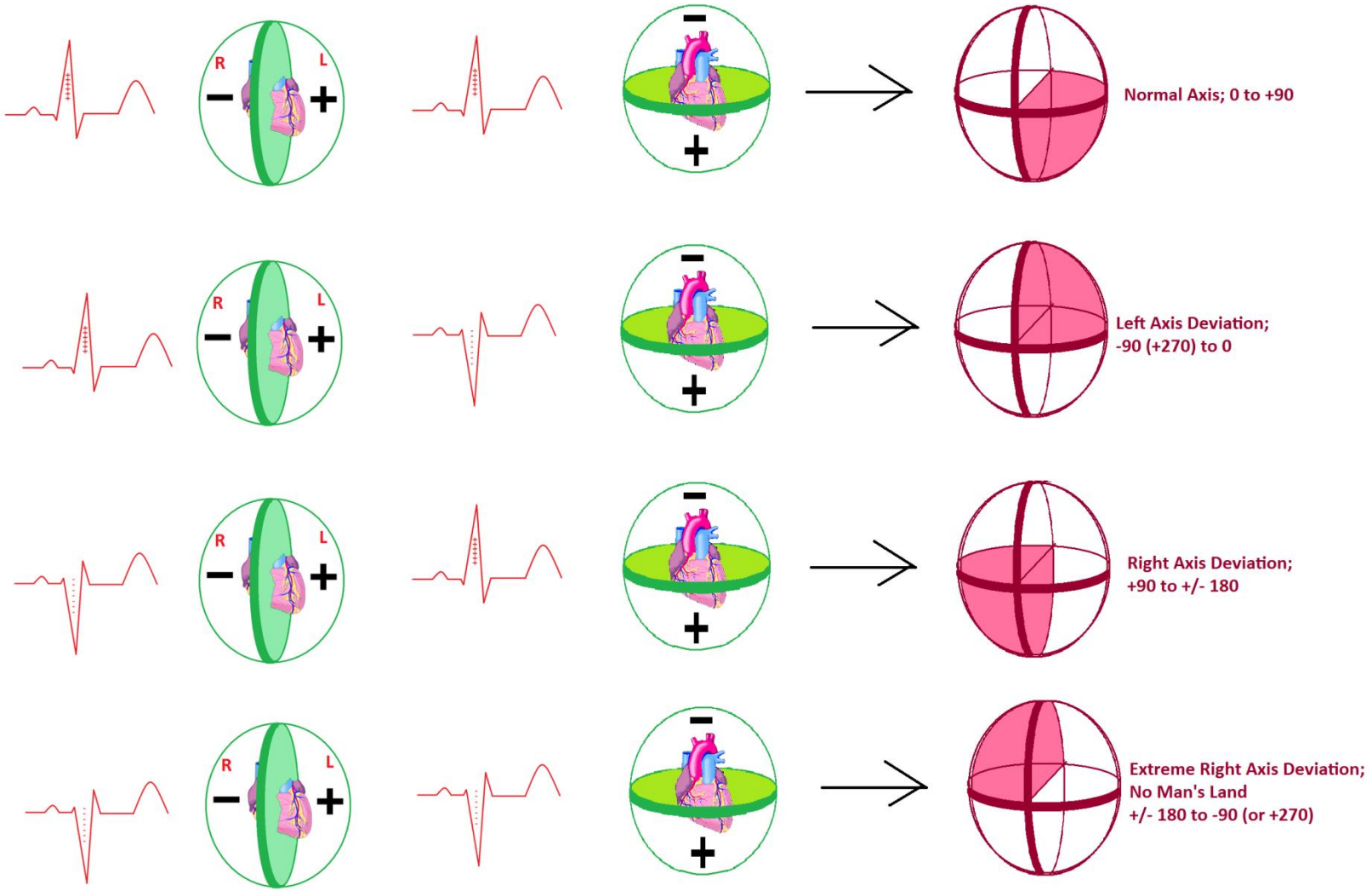


- Positive QRS
- Vector is DOWN and points into bottom of sphere
- Bottom of sphere is positive



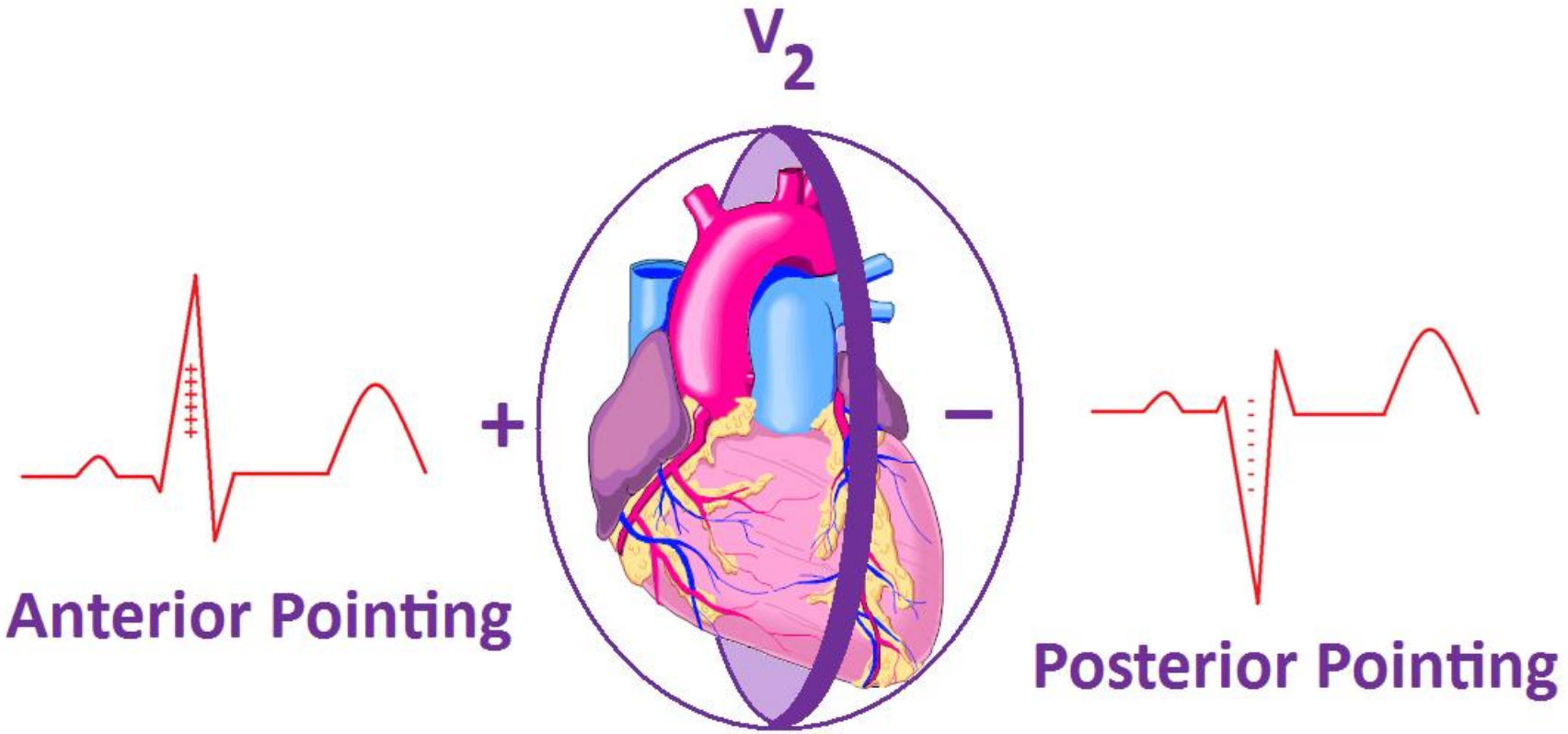
- Negative QRS
- Vector is UP and points into top of sphere
- Top of sphere is negative

2-Dimensional Axis Determination



Can We Add the Third Dimension – Depth?? YES!!

Examine QRS in V_2 – front/back sphere



2-D Axis Deviations

- Give crude approximation of deviation
- Could use a table – online – not in lecture only introduction to concept
- Table also uses I, II, III, R, L, F to determine deviation
- Have to determine which is the most **isoelectric** lead, though

Isoelectric Leads

- Isoelectric = equal voltage
- Means that the QRS complex is equally up and down compared to the isoelectric line
- Isoelectric line = the flat part of the EKG tracing
- When QRS is mostly upward, = primarily positively charged
- When QRS is mostly downward, = primarily negatively charged

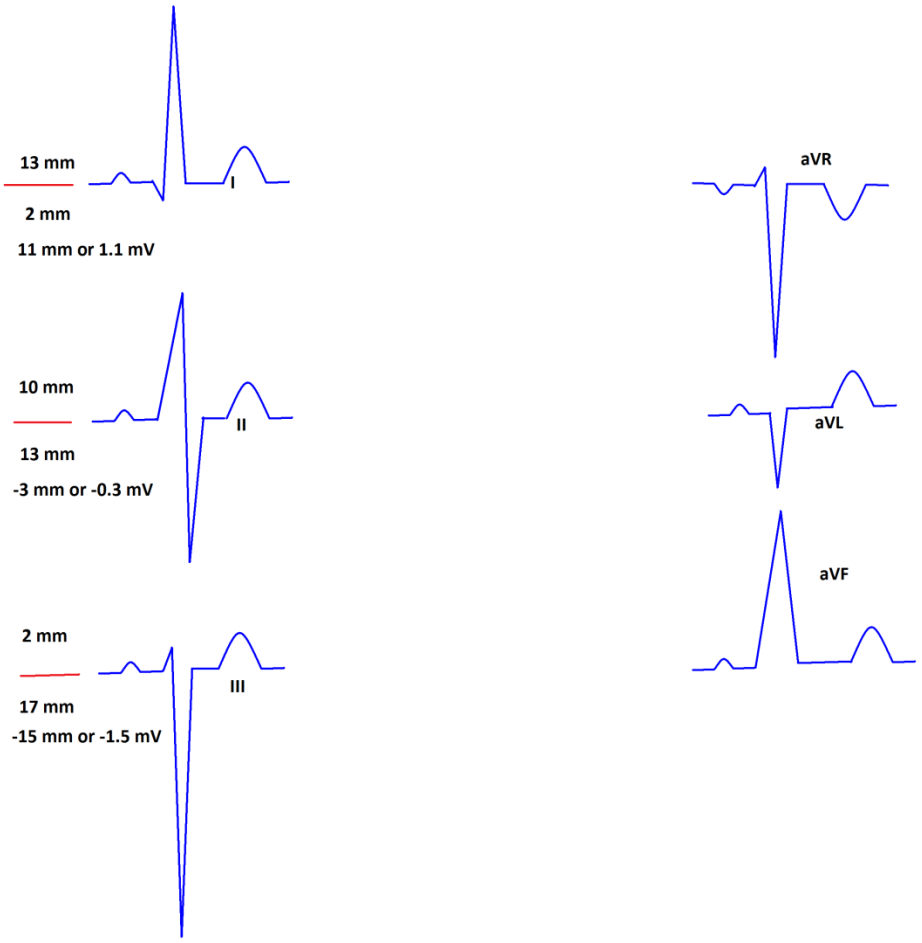
Isoelectric Leads

- If an R wave is +0.5 mV (upward)
- If an S wave is -0.5 mV downward
- Sum is zero – perfectly isoelectric
- Rarely happens in real-life
- In practice, one searches for the QRS that is most isoelectric
- Can also measure in mm instead of mV
- This method requires a table of values – online, not in lecture ☺ ← just did the concept

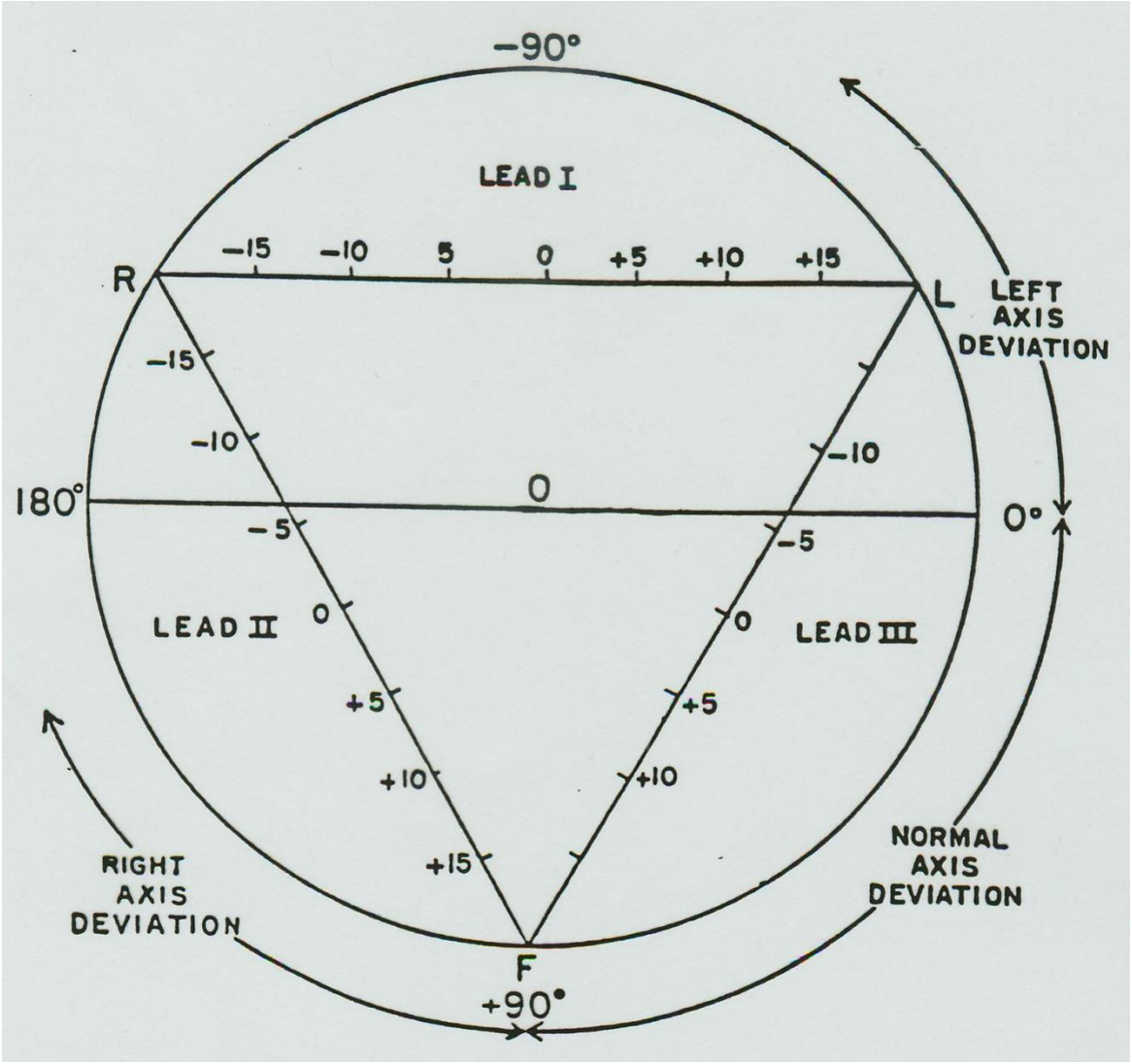
Another Method: Using a Nomogram

- Measure height of positive part of QRS complex (R wave)
- Measure height of negative part of QRS complex (either Q wave OR S wave)
- Both measures are absolute values: |x|
- Subtract the negative height from the positive height and record value
- Graph each vector on the nomogram

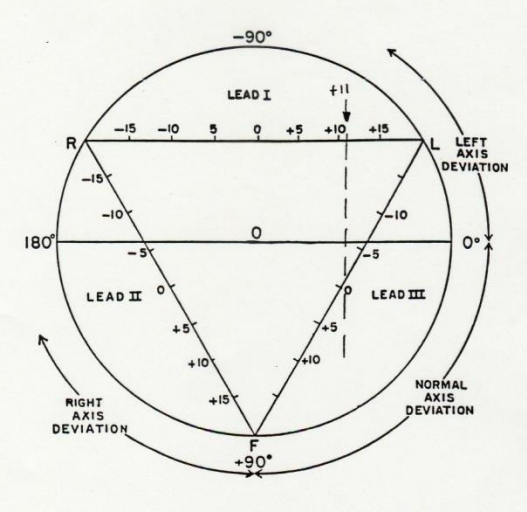
Nomogram – Need I and III data, minimally – II can be used for more fine tuning

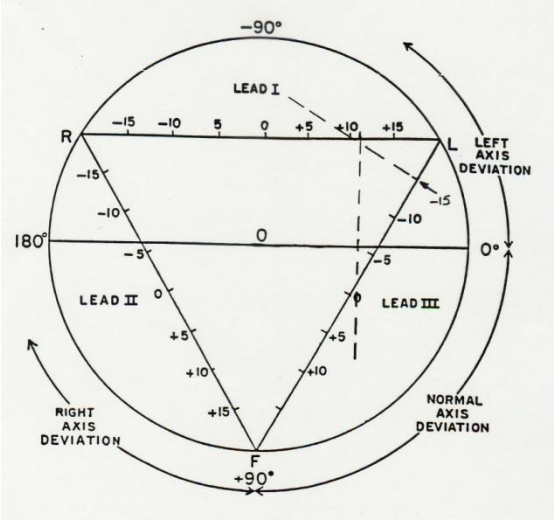


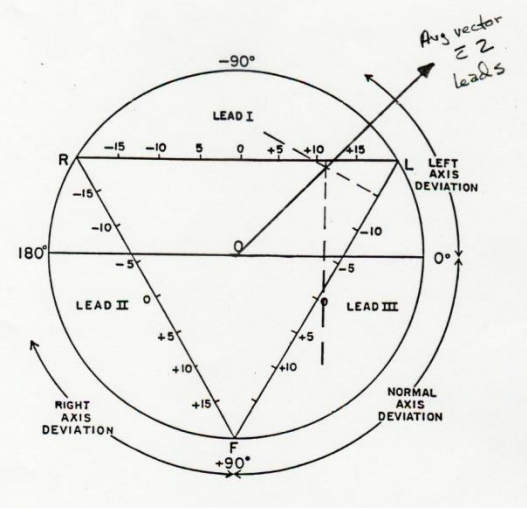
Triangulating Method -- Introduction

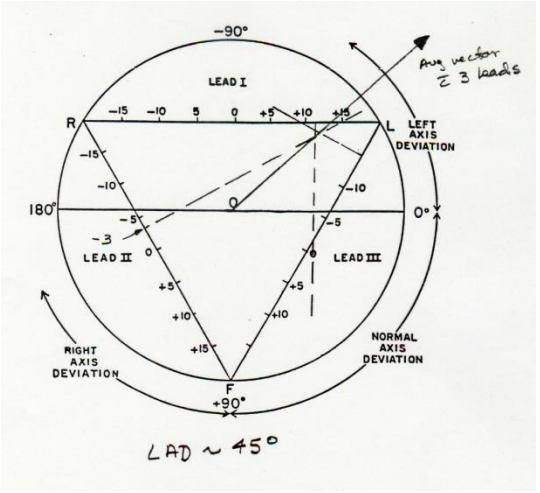


Triangulating Method -- Application

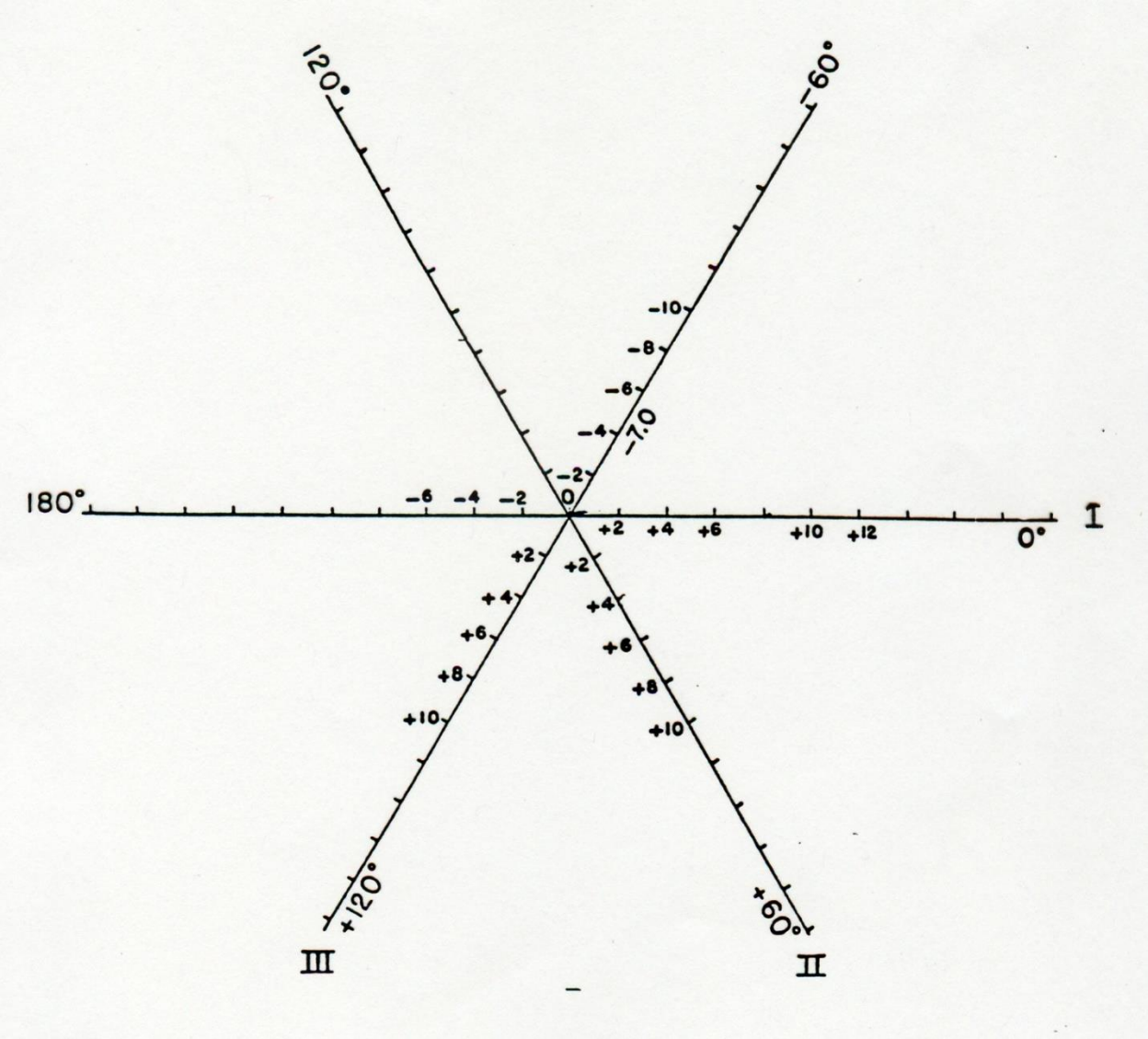




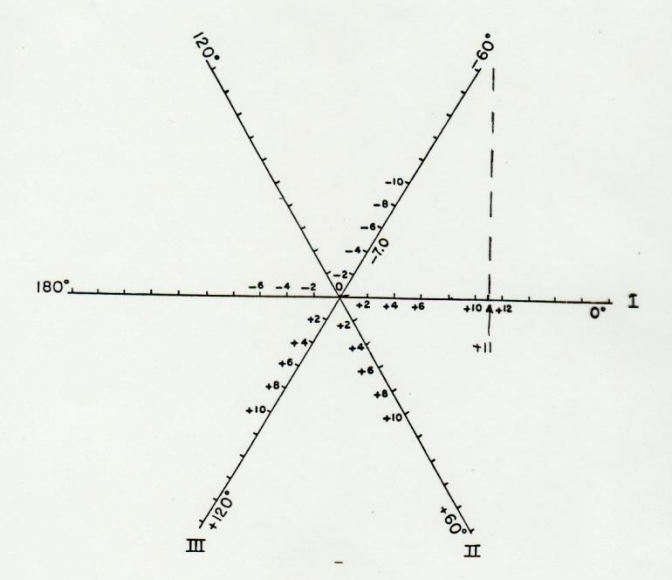


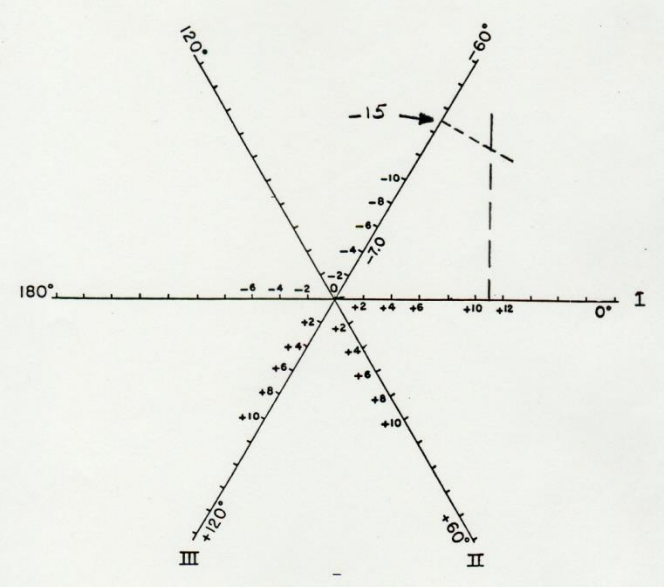


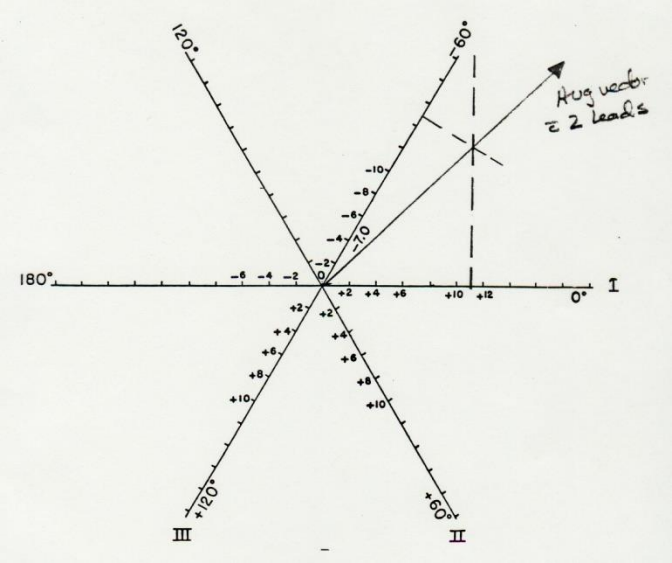
Triaxial Method -- Introduction

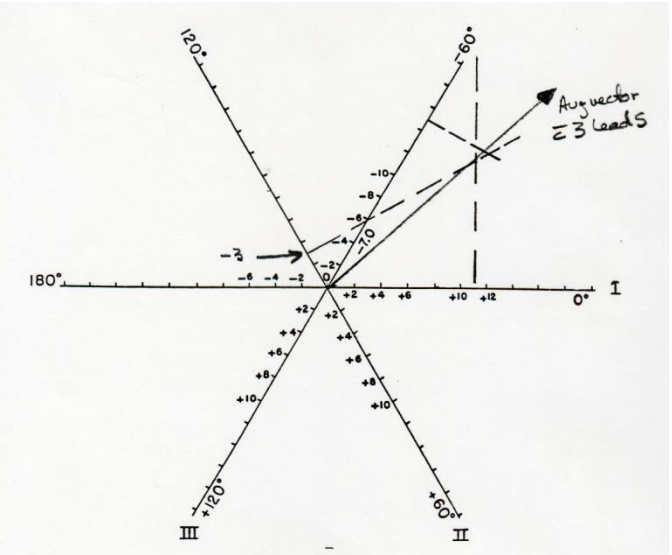


Triaxial Method -- Application

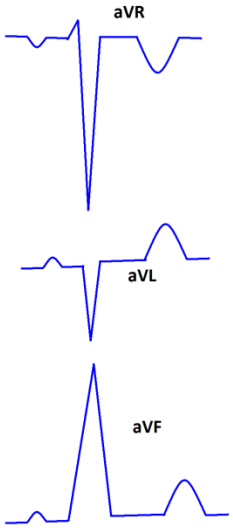






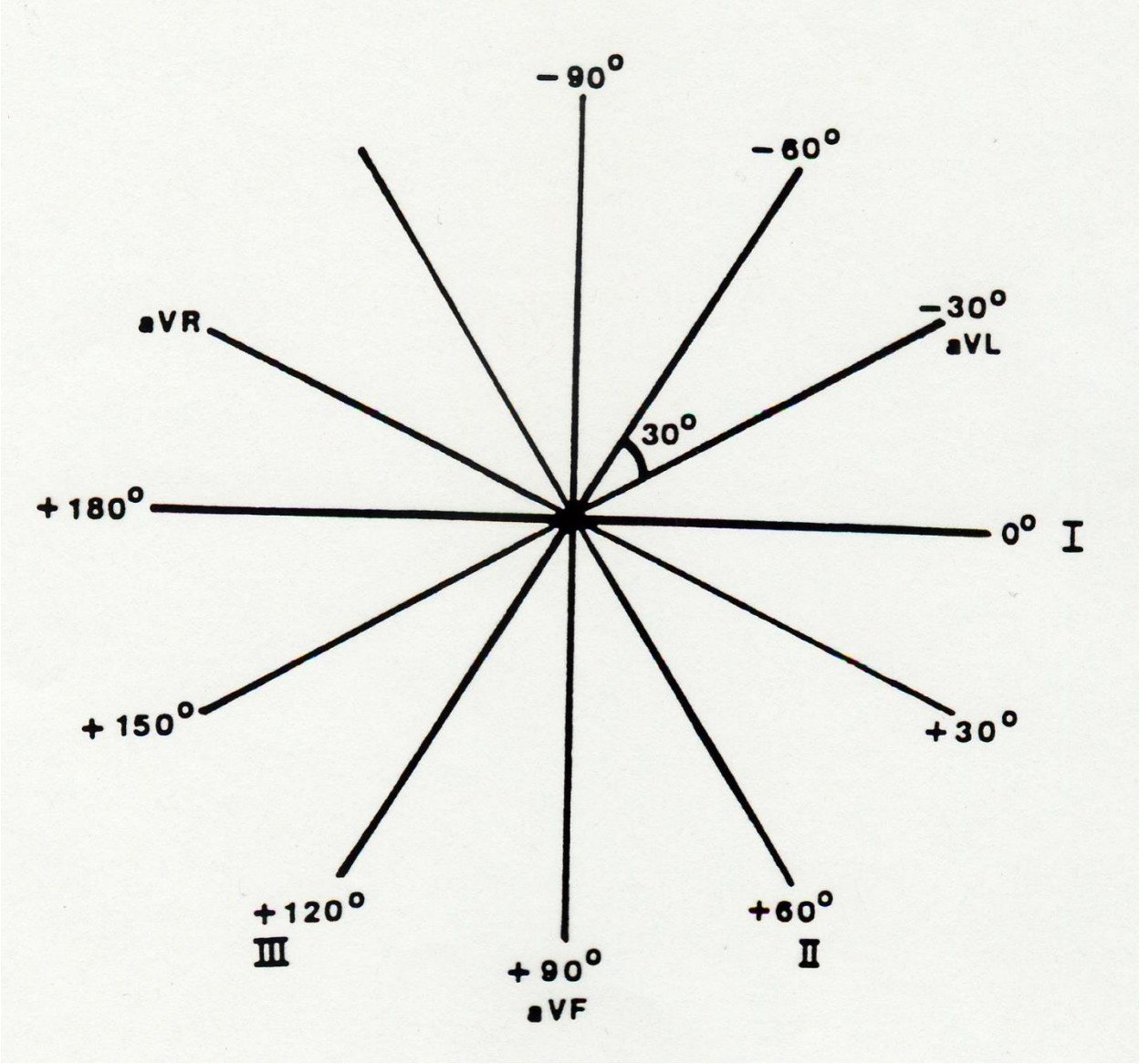


Hexaxial Method

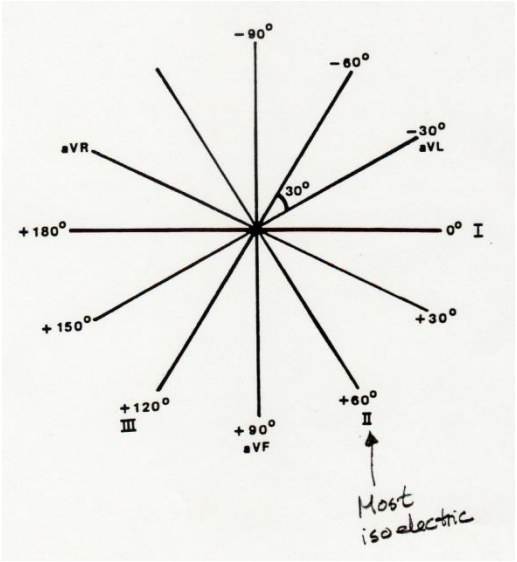


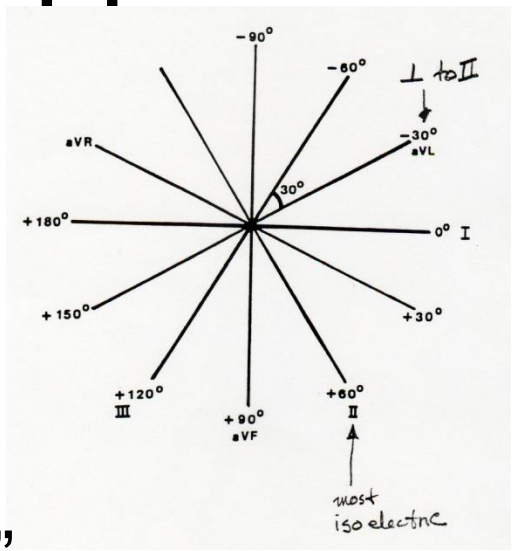
- Requires R, L and F for this
- Application of tables without using tables
- Tables on line – not in lecture

Hexaxial Method -- Introduction

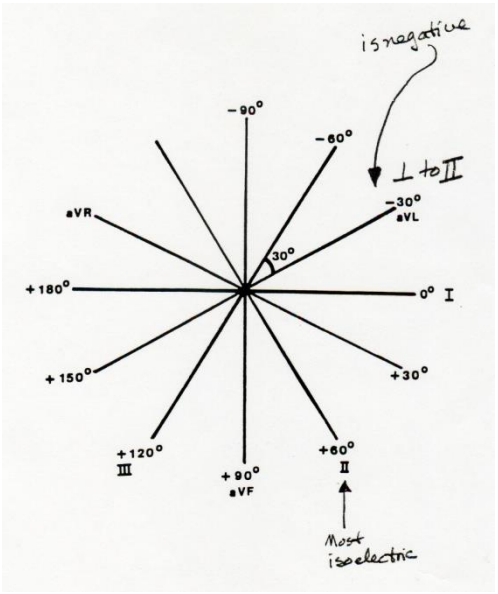


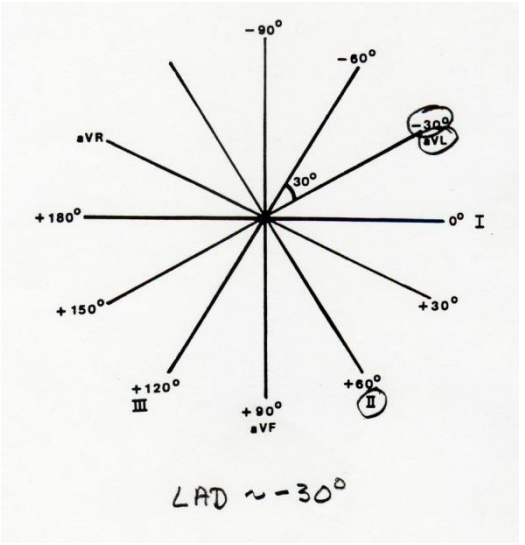
Hexaxial Method -- Application



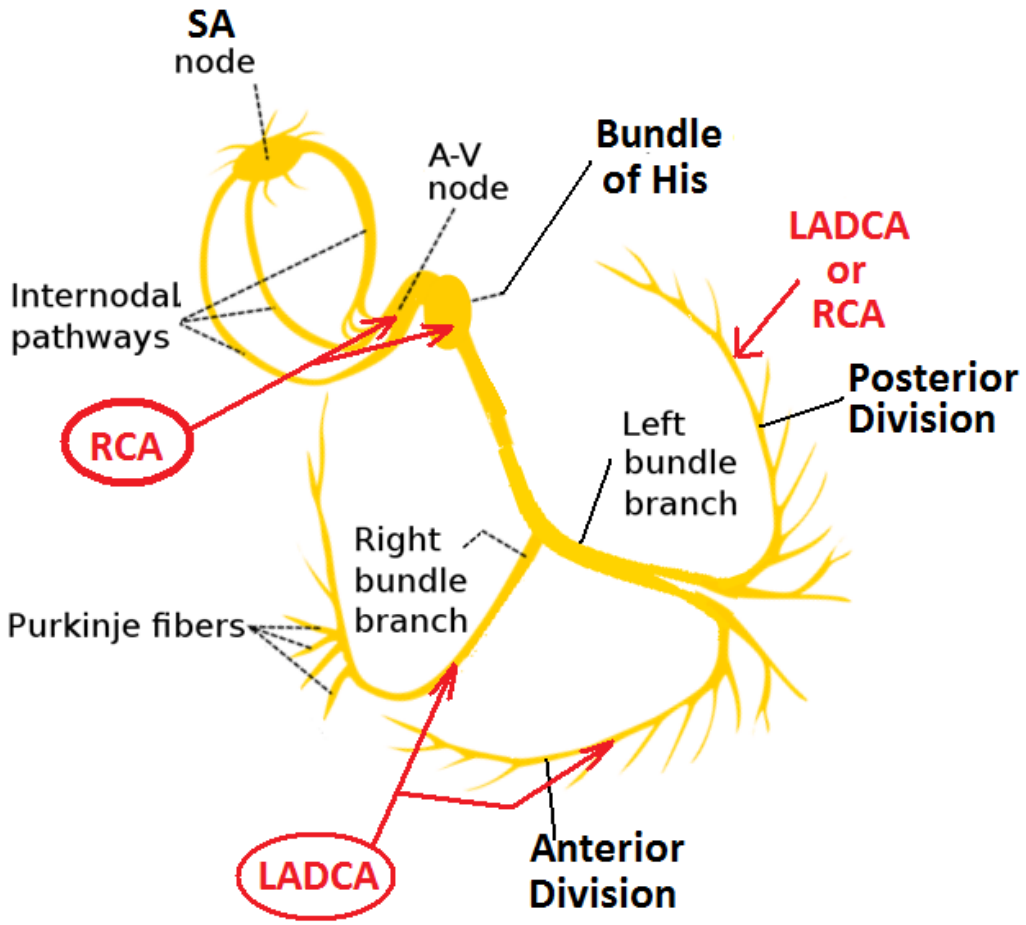


Isoelectric “stuff”





Bundle Branch Blocks -- BBB



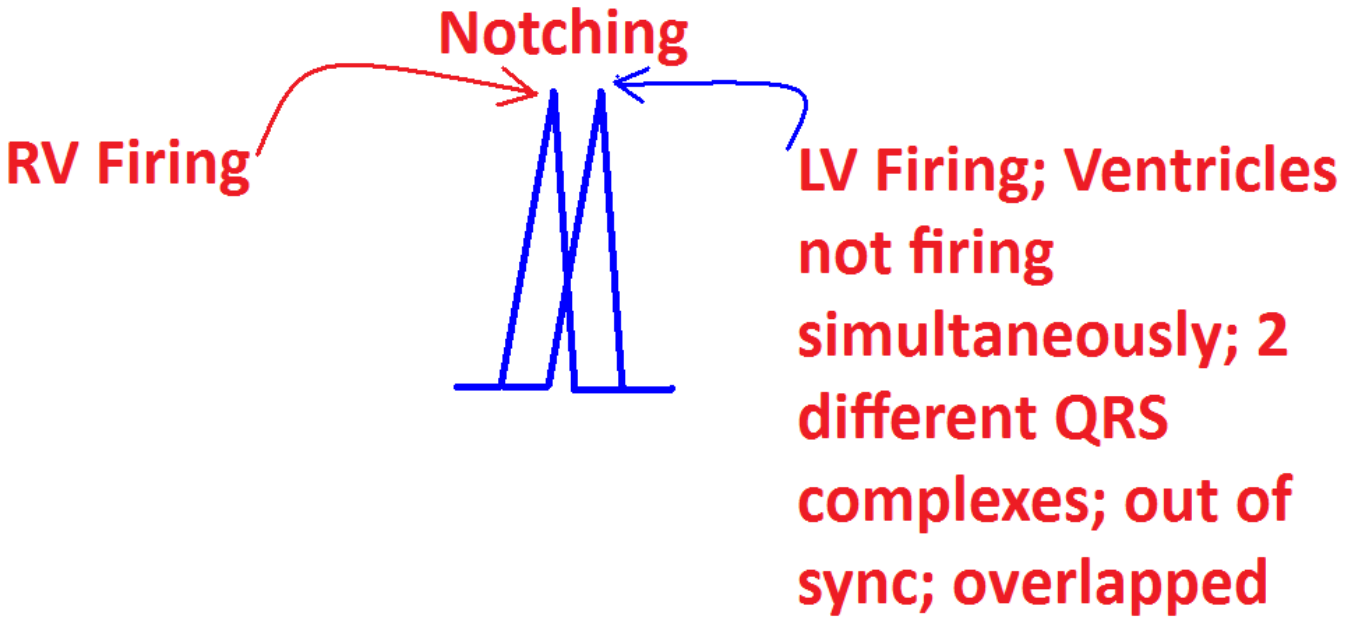
- BBB = delayed or obstructed conduction of signals in one of the branches beyond the Bundle of His
- A block to either BB = delayed electrical impulse to that ventricle
- Posterior Division of LBB receives blood flow from either the LADCA or the RCA – this dual blood supply seems to protect against blockage

Causes of BBB

Left BBB	
MI	Conduction system fibrosis
Valvular disease	Tumors
Rheumatic disease	Cardiomyopathy
Syphilis	(C)HF
Trauma	Hypertension
CHD	

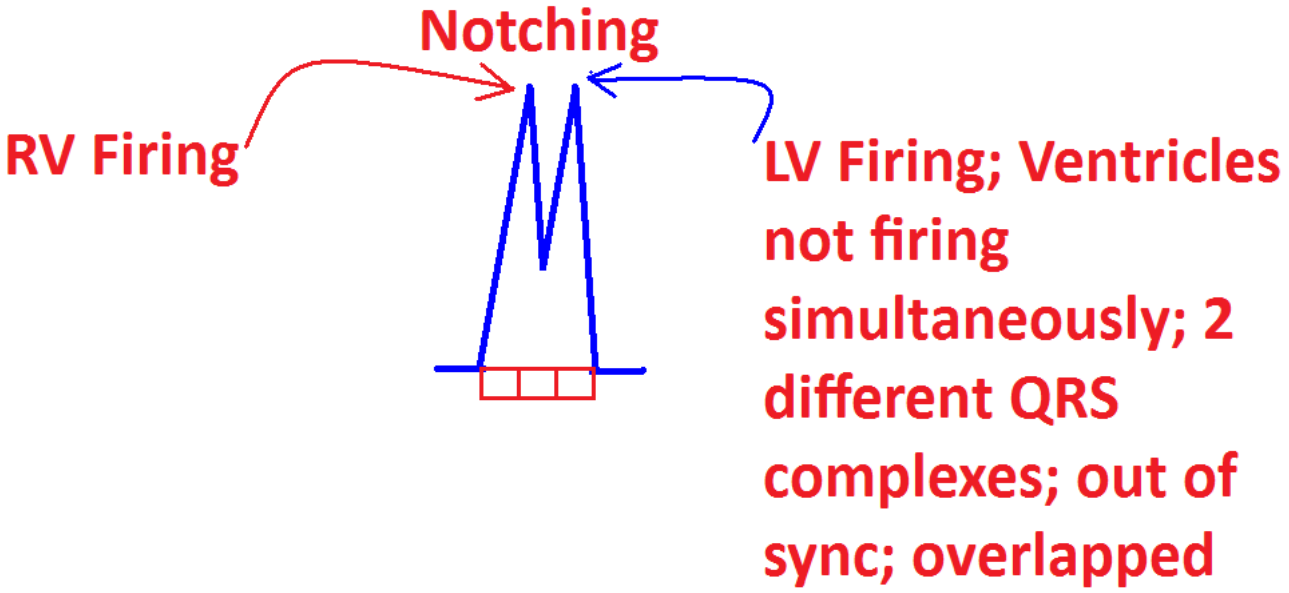
Right BBB	
CHD	RV strain following pulmonary stenosis or pulmonary hypertension

EKG changes in BBB



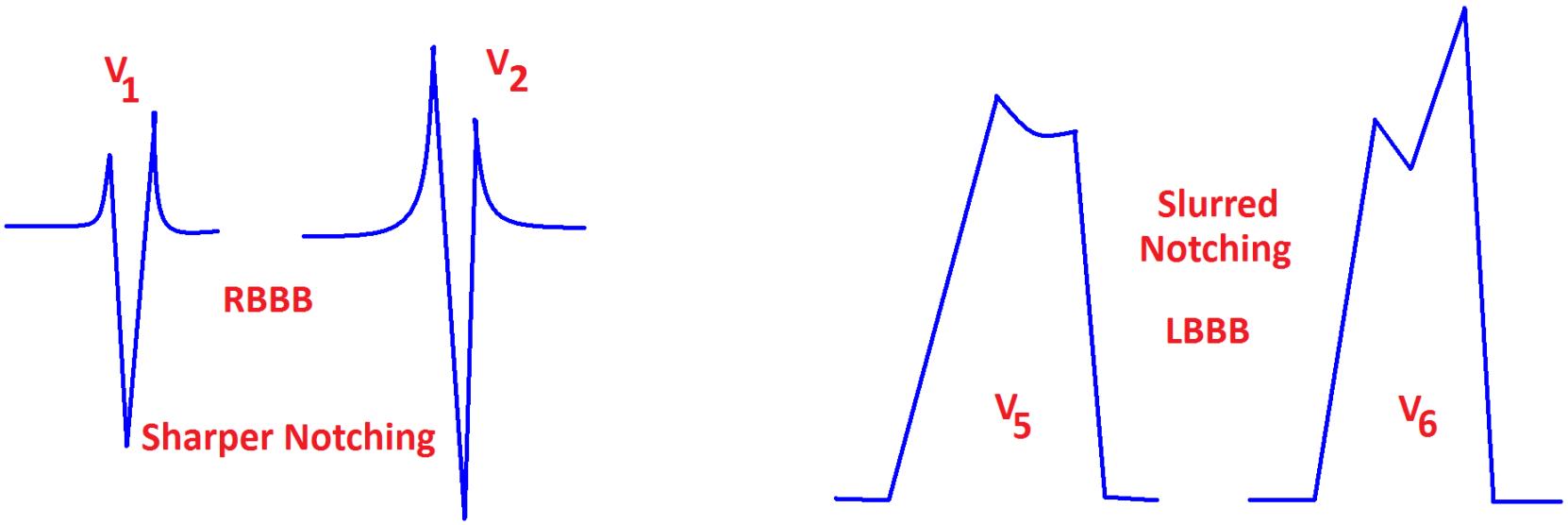
- This example = LBBB
- WHY??? – look at image above

When the 2 Complexes Fuse



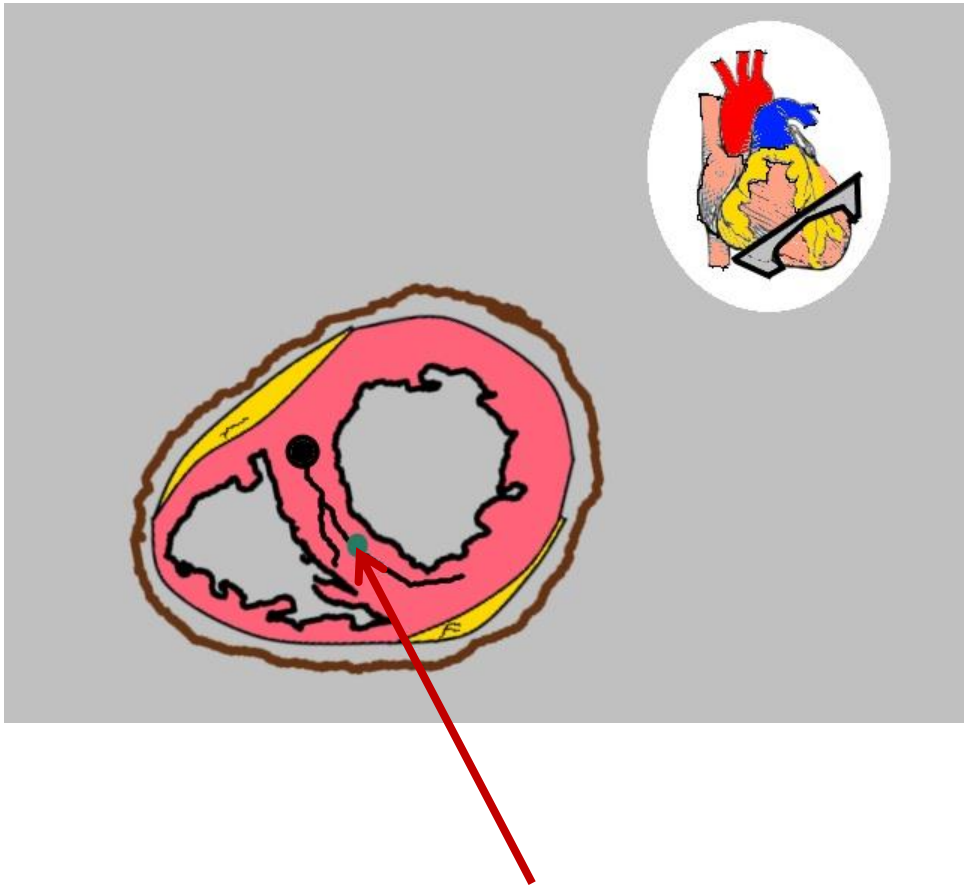
- 3 or more little squares wide (0.12 sec) is diagnostic for BBB, i.e., widening of the QS interval

How is LBBB Determined vs RBBB?



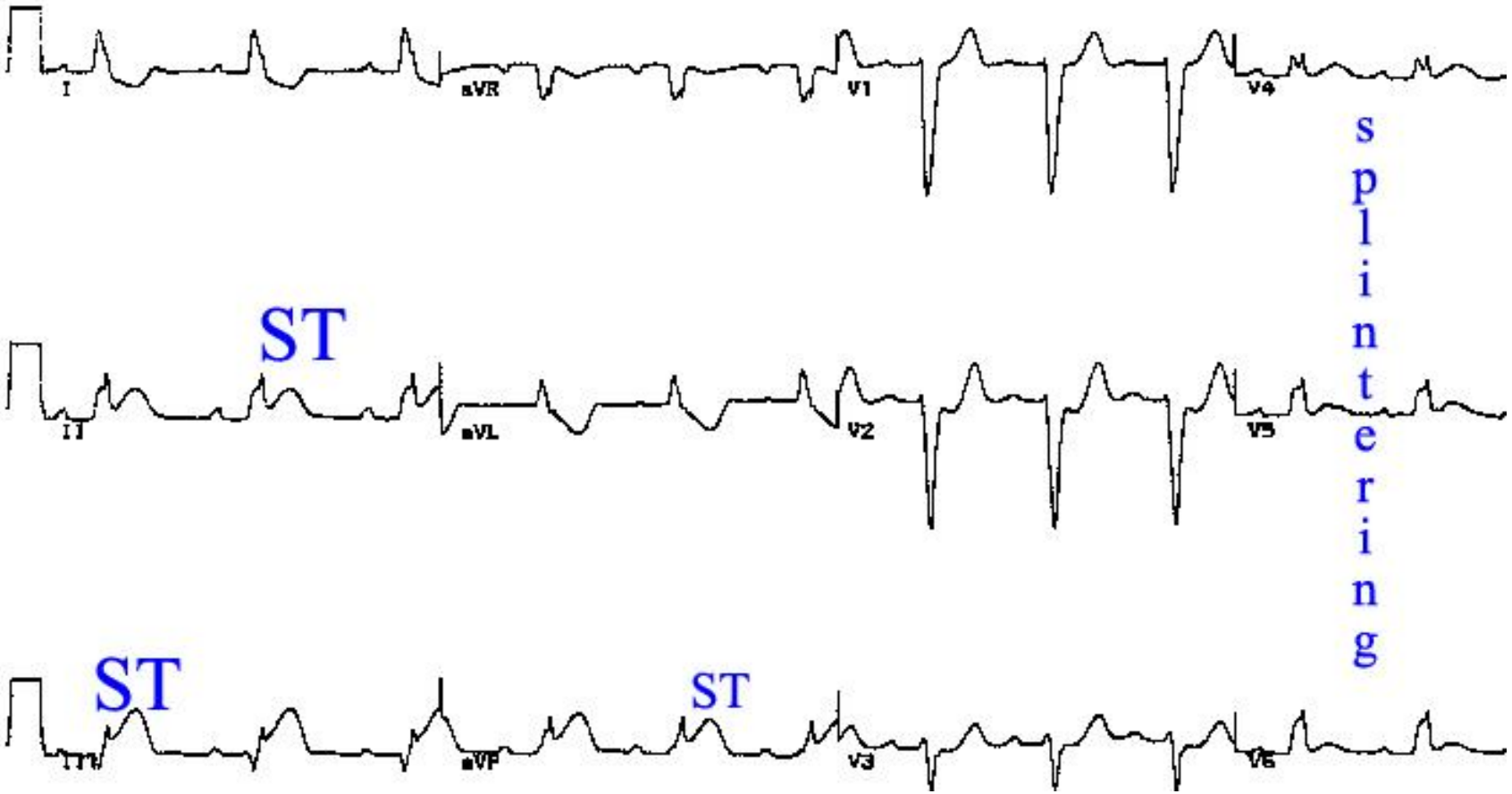
- Look at precordial leads
  - V_1 and V_2 for RBBB
  - V_5 and V_6 for LBBB
- Right and Left sides of the heart
- NOTE: with LBBB, you can not accurately dx MI by EKG (RIGHT ventricle hides the Q waves, making them undetectable)

LBBB: How Does Left Ventricle "Fire"?

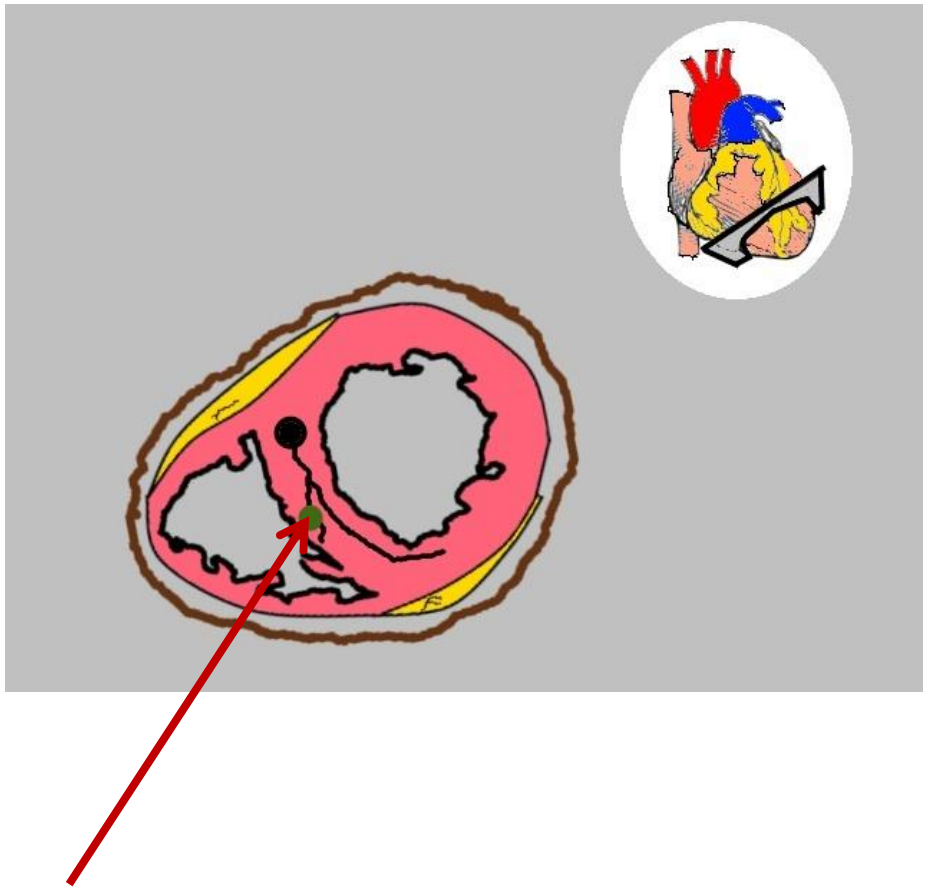


1. Signal directly to RV
2. Signal indirectly to LV via LBB "arborization" fibers below the block – takes approx 0.06-0.07 sec
3. RV fires
4. LV fires after RV fires
5. Beginning septal activation is from R to L and there are no Q waves in L precordial (chest) leads or I and L many times
6. MAY be Q waves in II, III and aVF, MIMICKING inferior MI
7. BIGGEE: with LBBB, can not accurately dx MI by EKG – no Q waves – hidden by R ventricle
8. Slurred notching in 5th and 6th precordial leads

Left Bundle Branch Block – Inferior Wall MI? … YES! … NO! … HUH?!

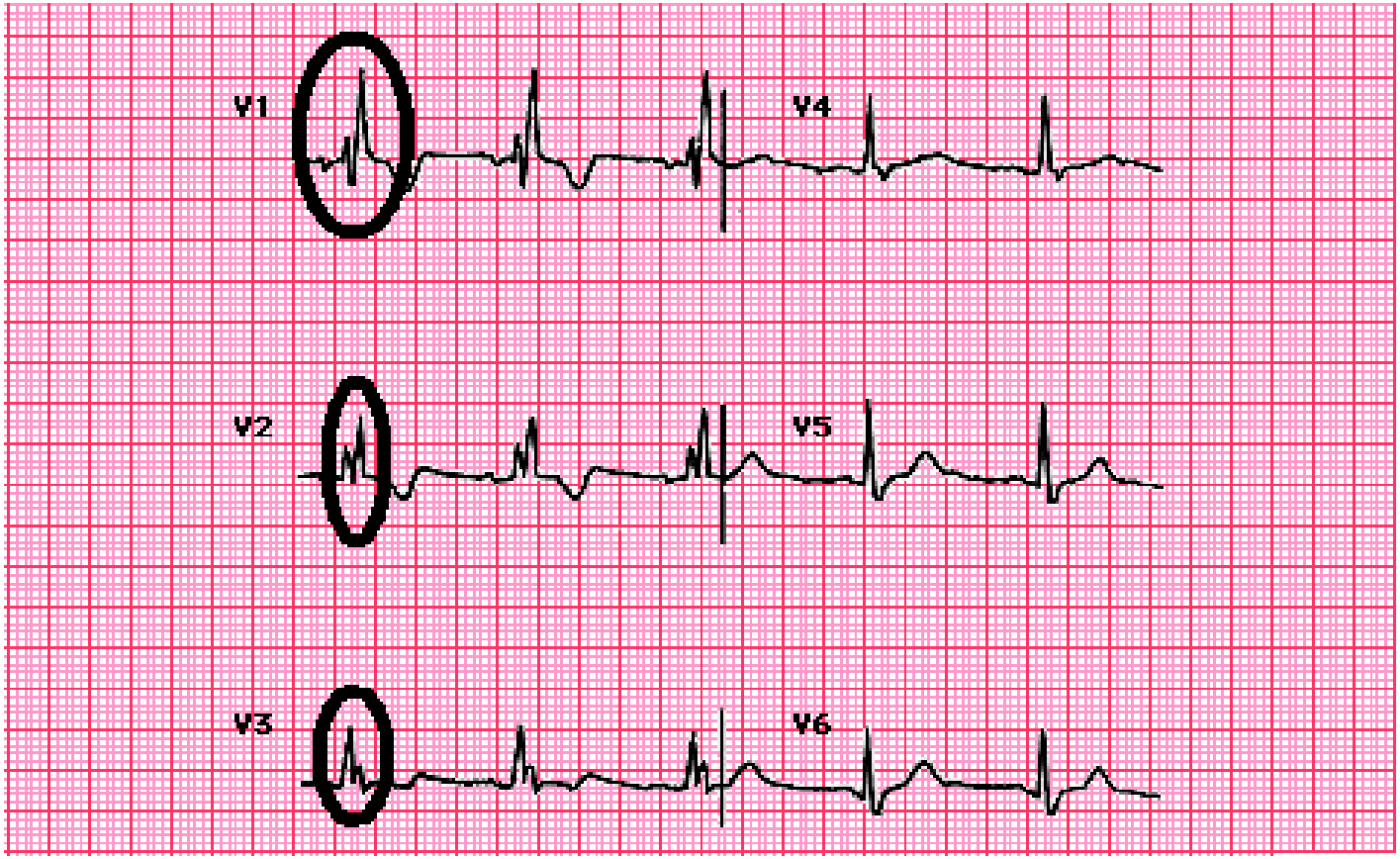


RBBB: How Does Right Ventricle "Fire"?



1. Signal directly to LV
2. Signal indirectly to RV via RBB "arborization" fibers below block – approx. 0.06-0.07 seconds
3. LV fires
4. RV fires after LV
5. Q waves are present; inverted T waves in V_1 that become more upright by V_6
6. Sharp notched R waves (R and R') in V_1 and V_2

RBBB



Common RBBB Electrocardiogram showing characteristic changes in the precordial leads in common RBBB. Ary Goldberger, MD.

With BBB One

CAN NOT

- Determine average vector
- Determine ventricular hypertrophy

CAN

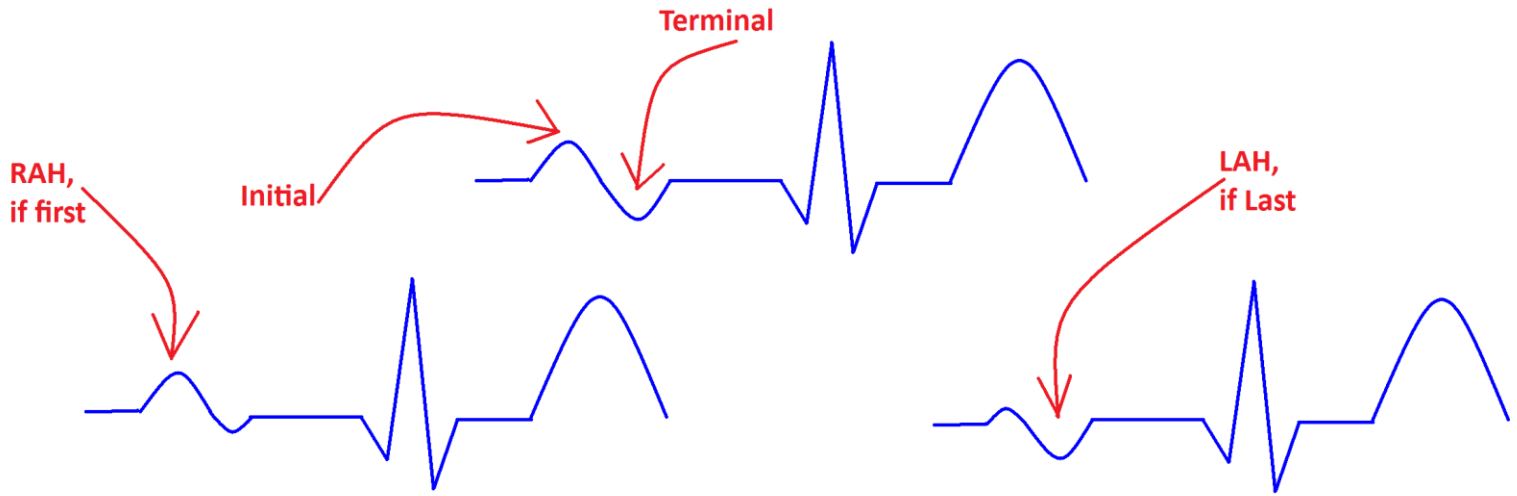
- Determine ATRIAL hypertrophy

Atrial and Ventricular Hypertrophies – without BBB!

- Right Ventricular Hypertrophy
- Large R wave in V_1 with small S
- NORMALLY: S > R in V_1
- R gets smaller in precordial leads
- RAD is an indicator, too

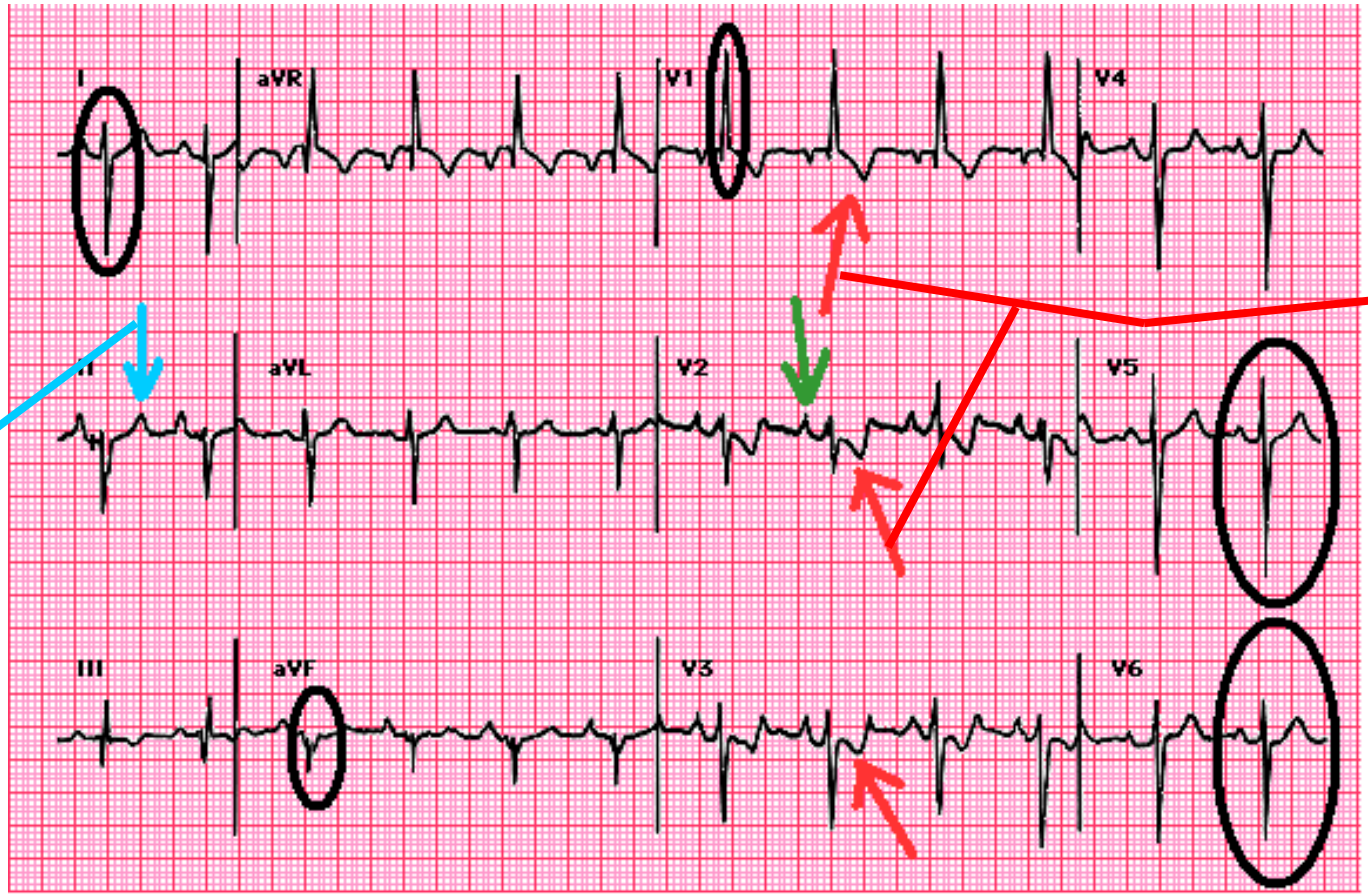
- Left Ventricular Hypertrophy
- DEEP S in V_1 with increasing R by V_6 (large S in V_1 and large R in V_5
- LAD is an indication, too
- If sum of mm S in V_1 plus mm R in V_5 is greater than 35 mm, patient has LVH

Atrial Hypertrophy Determination



- Examine P wave in V_1 – over atria – with hypertrophy = diphasic P waves
- Also: confirmatory = RAH with P wave > 2.5 mm tall in II or > 1.5 mm tall in V1
- Also: confirmatory = LAH with P wave notched ("m" shaped) in II; P wave wider than 3 mm (3 little squares) in II; aka p mitrale: left atrial enlargement is often caused by left atrial dilatation or hypertrophy due to stenosis of the mitral valve, hence the term *P mitrale*.

RVH -- Ignore Green Arrow



Right ventricular hypertrophy Right ventricular hypertrophy due, in this case, to primary pulmonary hypertension. The characteristic features include marked right axis deviation (+210° which is equal to −150°), tall R wave in V1 (as part of a qR complex), delayed precordial transition zone with prominent S waves in leads V5 and V6, inverted T waves and ST depression in V1 to V3 consistent with right ventricular "strain", and peaked P waves in lead II consistent with concomitant right atrial enlargement. Courtesy of Ary Goldberger, MD.