

# High Yield Cardio for Step 1/2

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# As I am sure you are well aware...

- They love making you think during these tests
  - You may know the murmur but they want to know if you can take it a few steps further
  - Seeing an EKG or having to listen to a murmur can make you stress out because it throws you off
    - Our goal is to help you not get thrown off by these cardio questions
- Challenge yourself to think of ways to integrate the information
  - Could easily ask about genetics or embryology of murmur
  - Also a lot of high yield infections that mess with your heart-rubella, GAS

# Murmur Questions

- Text descriptions
  - Megri's method will save you valuable time
- Audio questions
  - Check for radiation of the murmur
  - Clinical correlation to vignette-old lady with syncope with a murmur that radiates to the carotids will be AS 99% of the time

# Murmurs

- If it is a text question-go to the description of the murmur and start eliminating (Megri's Method)
  - Systolic
    - ASS (Aortic stenosis, pulmonic stenosis, tricuspid regurg, mitral regurg)
  - Diastolic
    - Aortic regurg, pulmonic regurg, tricuspid stenosis, mitral stenosis
  - Holosystolic
    - VSD, tricuspid and mitral regurg
- If you get in trouble-try to think about the murmur relative to the cardiac cycle
  - Murmurs-think of as feed forward issues or backflow issues
    - Feed forward-from valves that should be open but are not good at opening because they are stenosed
    - Backflow-valves that should be closed that are allowing backflow
    - Systole-open A/P, closed M/T
    - Diastole-open M/T, closed A/P

# Systolic vs. Diastolic Murmurs

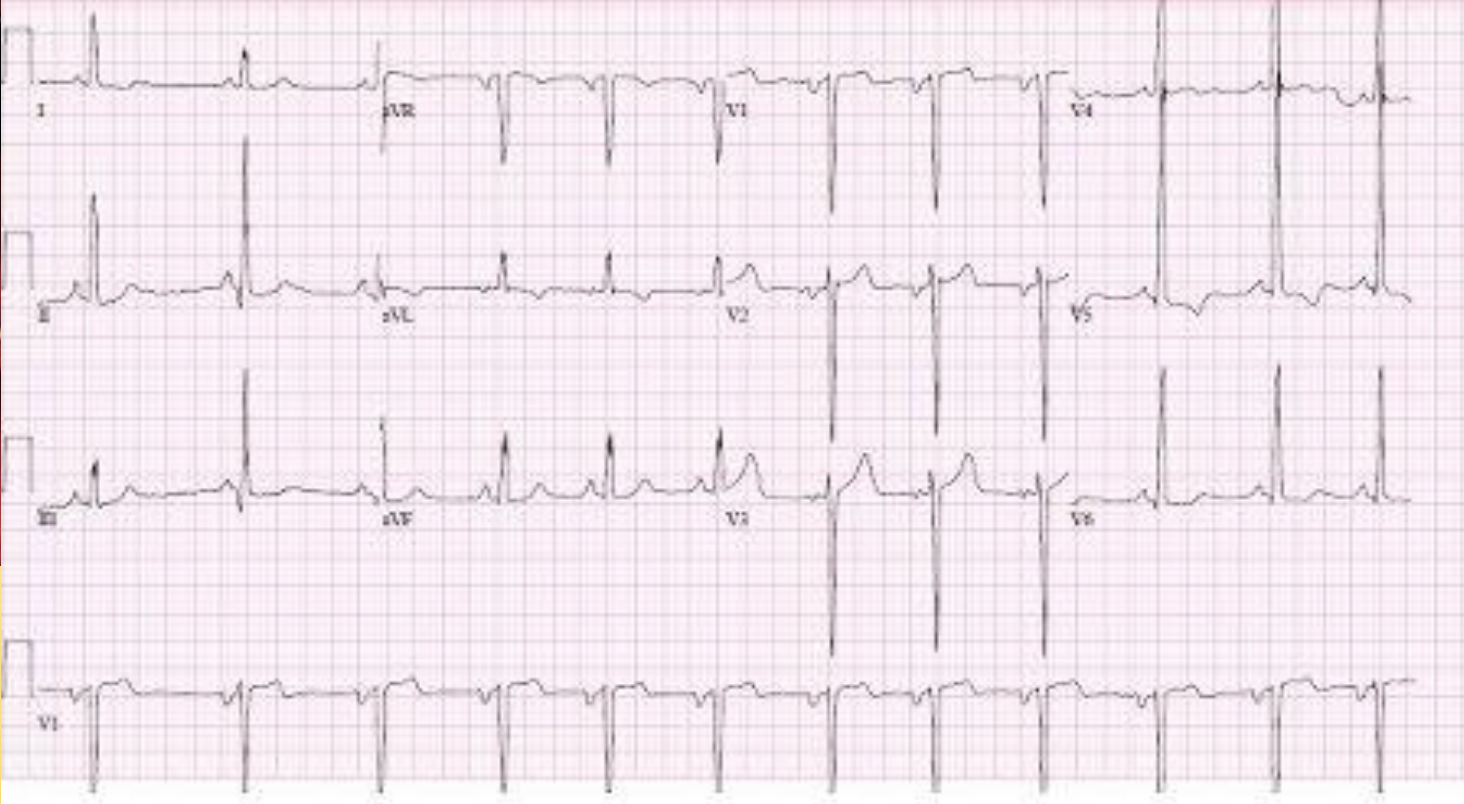
- In real life, being able to feel a radial pulse makes it easier to distinguish between systolic and diastolic murmurs
  - If you are unsure while you are seeing a patient in the clinic, feel for a pulse and the bounding corresponds to systole
- Unfortunately, USMLE has to use weird animations and sound clips
- Try tapping out the lub-dub and then using that to determine if it is systolic (between the lub and dub) or diastolic (between the dub and lub)
  - Just knowing that information can help you narrow it down
- Also, listening for the harsher sound can help you determine the murmur
  - Close your eyes and listen for whether it is sloshy then distinct or distinct then sloshy

# Aortic Stenosis-crescendo decrescendo systolic murmur, loudest at base, radiates to carotids

- Radiates to carotids
- Older patient w/ **syncope**, angina, dyspnea
- Pathology-calcification, could be secondary to rheumatic heart (MR to AS)
- Bicuspid aortic valve calcifies earlier
  - High association with Turner
- Just for kicks and giggles-LVH 2/2 aortic stenosis
  - Septal leads will show crazy amplitude of QRS



# Aortic Stenosis



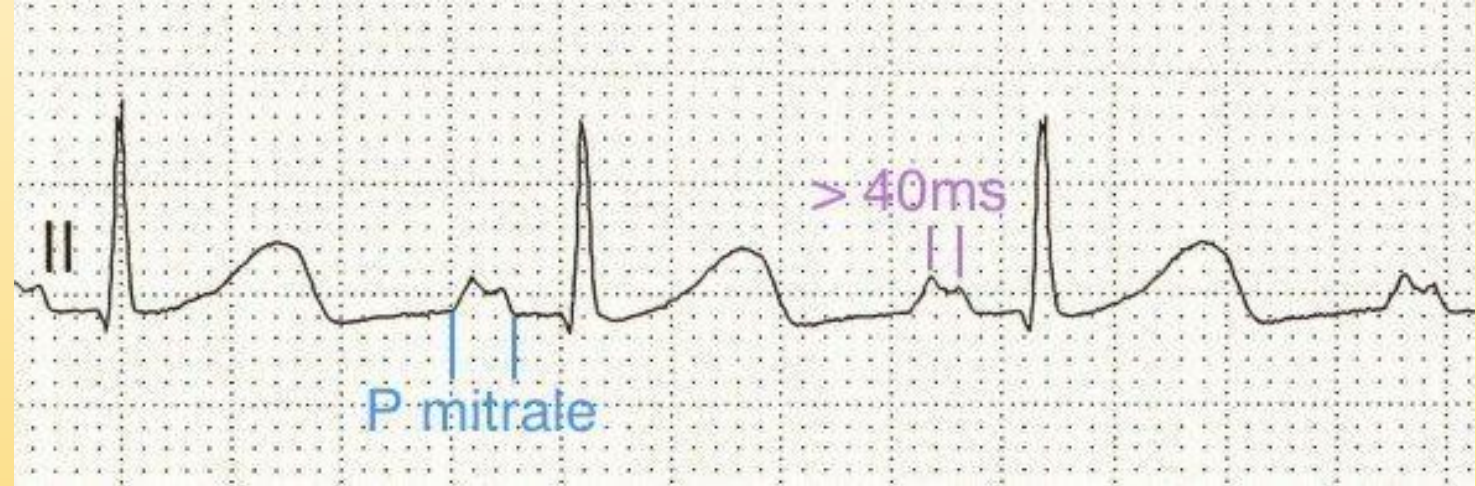
# Mitral Regurg

- Description-holosystolic helps narrow it down
- Mitral radiates to axilla
- Can be from infection-early or late manifestation of rheumatic fever (classically earlier manifestation of RF is MR)
- Can occur in the setting of IE/MI-chordae rupture leading to regurg or papillary muscle (chordae more susceptible to vegetations and papillary muscle rupture 2/2 ischemia post MI)
- Because of the backflow you will get enlargement of the LA and LV
- Just for kicks and giggles-P wave abnormalities in EKG (looks like m)
  - Can also have splayed carina on x-ray because LA is closest to carina
  - LAE can also cause hoarseness (L recurrent laryngeal nerve) and dysphagia (esophagus compression)



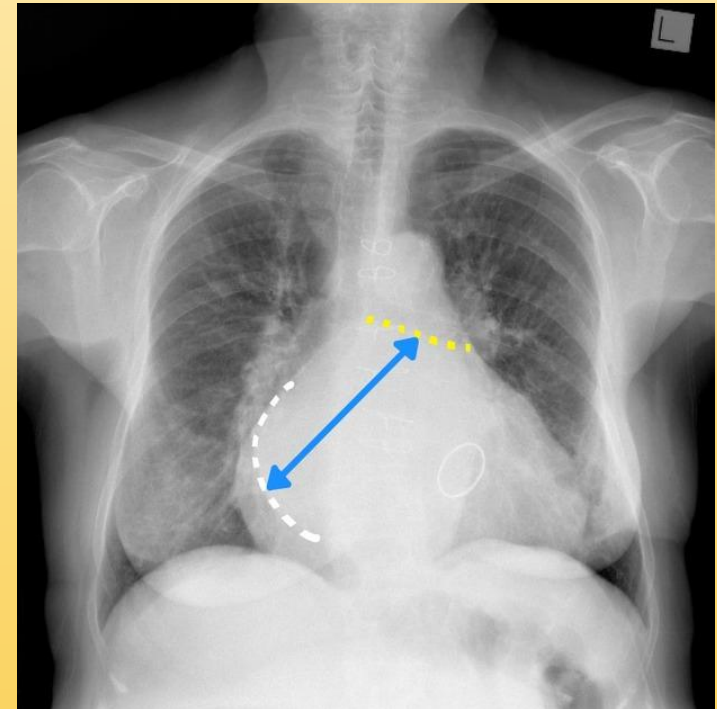


# Mitral Regurg



# Mitral Regurg

- More specific sign of MR on CXR is the “double density” sign-I just learned this so this is really just for kicks and giggles
- Normal right border on CXR is from RA



# Tricuspid Regurg

- Essentially the same as MR
  - Less classical causes/associations
  - Different location
  - Does not radiate to the axilla
  - RVH and RAE
  - Louder with inspiration (like all R sided murmurs)



# VSD

- Holosystolic (like MR/TR)
- Embryologically-most commonly occurs at the membranous portion
  - From growth of endocardial cushion



# MVP

- Most frequently described as murmur in a click in a young female
- Myxomatous degeneration
- If symptomatic with heart palpitations-can treat with BB
- Late systolic with a click
- Infective endocarditis-degeneration predisposes to IE (most commonly associated with Strep Viridans because it frequently attacks damaged valves)

**Mitral  
Valve  
Prolapse**  
Heart Sound



# Aortic Regurg

- Early diastolic blowing murmur
- Head bobbing
- Bounding pulse felt in fingertips
- Widened pulse pressures
- Seen from endocarditis, aortic root dilation (syphilis, connective tissue disorders), bicuspid aortic valve



# Mitral Stenosis

- Opening snap
- Radiates to axilla
- Seen in RF (RF murmurs are in alphabetical order MR early, MS late)



# Patent Ductus Arteriosus

- In utero connection between pulmonary arteries and aorta is advantageous because your lungs aren't useful
- Continuous machine
- Seen in congenital rubella
- When would the ductus be advantageous?
  - Cyanotic heart lesions-give PGE to keep it open
- Indomethacin can close the ductus-disadvantage to use in the world of obstetrics to slow down labor





# But then they add movements

- Once again, they are trying to make you uncomfortable
- Only 2 murmurs do the opposite of the other
  - General rule-increasing preload should increase a murmur, increasing afterload should decrease a murmur
  - The two exceptions (MVP, HOCM) are going to be seen in young patients
- Aortic stenosis and HOCM sound pretty freaking similar
  - Aortic stenosis-older, calcified valve or bicuspid valve (most commonly associated with Turners), murmur will decrease with squat and increase with leg raise
  - HOCM-younger male athlete, easy way for them to tie in genetics or histo/physio (murmur will increase with valsalva and decrease with leg raise)
  - MVP is also systolic like AS and HOCM-distinguish with click

# Maneuvers

	Venous Return / Preload		Afterload		Drugs	
	Increase	Decrease	Increase	Decrease	Diuretic	ACEIs
	(Leg raise / Squat)		(Handgrip)	(Amyl Nitrate)		
MS, AS	↑	↓	↓(AS)	↑(AS)	Yes, but better	
			Negligible Effect in (MS)		AS (Replace)	×
					MS(Ballon)	
MR, AR	↑	↓	↑	↓	✓	✓
VSD	↑	↓	↑	↓	✓	✓
HOCM	↓	↑	↓	↑	×	×
MVP	↓	↑	↓	↑	×	×

# TLDR Murmurs

- Description-ASS and work from there
- Listen for radiation-all of the murmurs below are systolic and will sound differently but if you start panicking with the sound quality-moving your stethoscope could save you
  - Radiation to carotid-AS
  - Radiation to axilla-mitral valve (MR)
- Maneuvers-only HOCCM and MVP differ in the general rule that preload increases a murmur and afterload decreases a murmur
  - Increase preload by squatting or leg raise/decrease afterload hand grip
  - Decrease preload by standing up/valsalva

# But what if they throw in an EKG-I failed the cardio exam and still don't get them

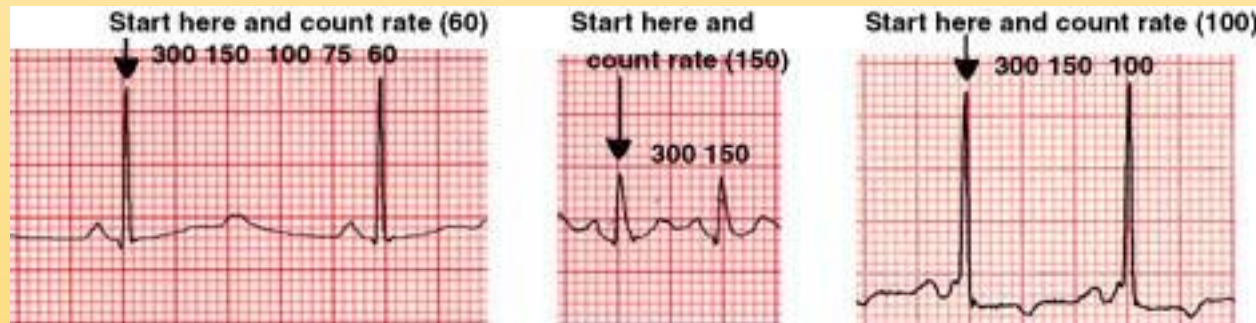
- Remember-EKGs are measuring vectors based on particular lead placement
  - Heart should depolarize from R to L and top to bottom
    - Natural conduction system is the most efficient conduction system
      - If you have the current depolarizing but in a direction that the system isn't made for-timing of a particular segment is going to be off
        - Wide QRS-WPW, LBBB, RBBB
  - Net current towards something gives an upward deflection, away gives a downward deflection

# EKG

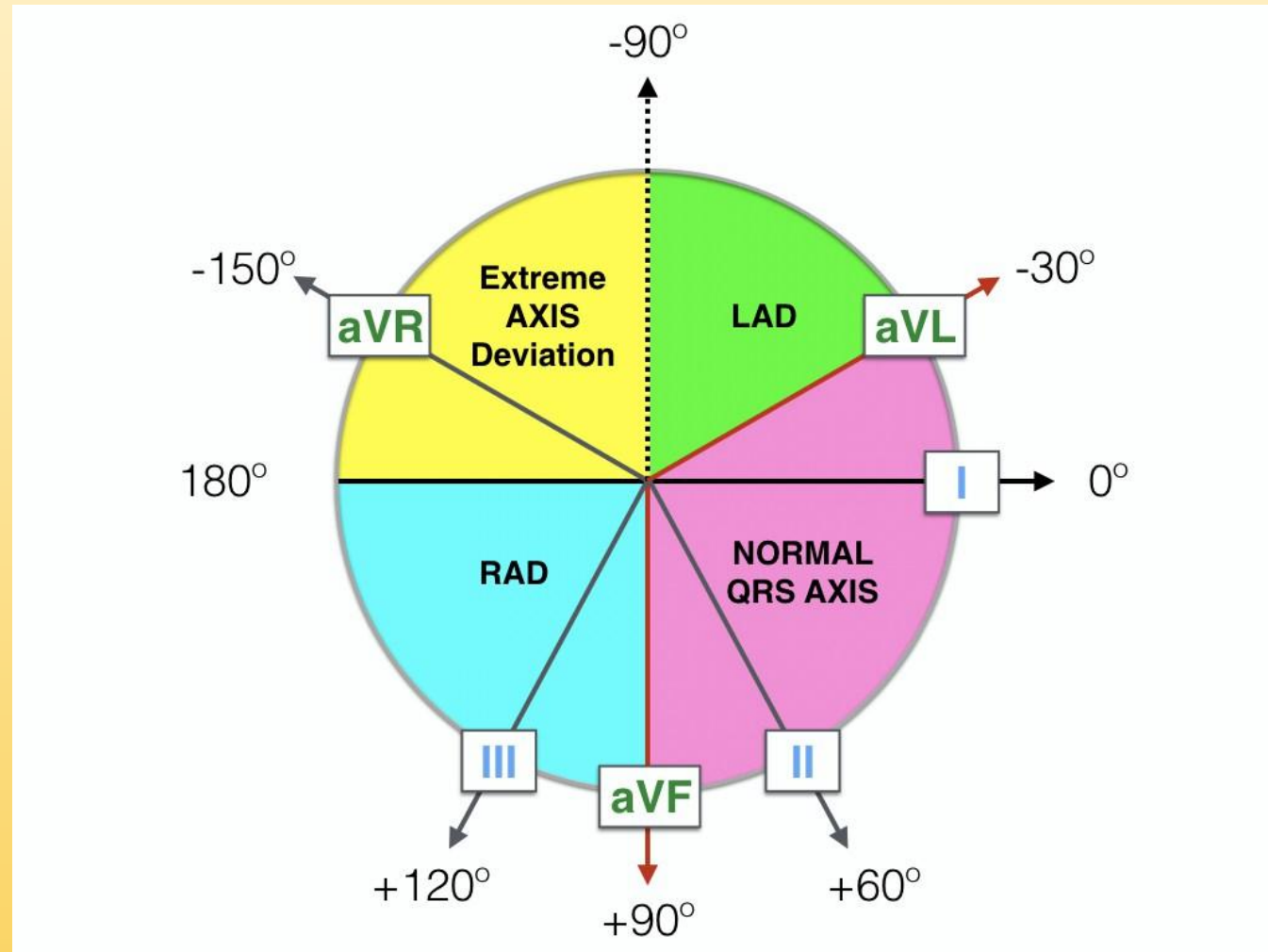
- Hard to know the basics because they seem so simple until you are put on the spot
- Try to understand rather than just memorize and recognize a pattern
- Dr. Petrany does a great job teaching a systematic way of going through EKGs-rate, rhythm, axis, PR, QRS, ST, QT
  - Admittedly lower yield for Step 1 but high yield for shelf exams and Step 2
  - Regardless of which field you go into-you WILL order an EKG on your patient at some point-so know how to read them

# Back to Basics-lower yield for Step 1

- Rate 60-100, count off method (300, 150, 100, 75, 60)
  - If they give you a 12 lead with a rhythm strip-x6

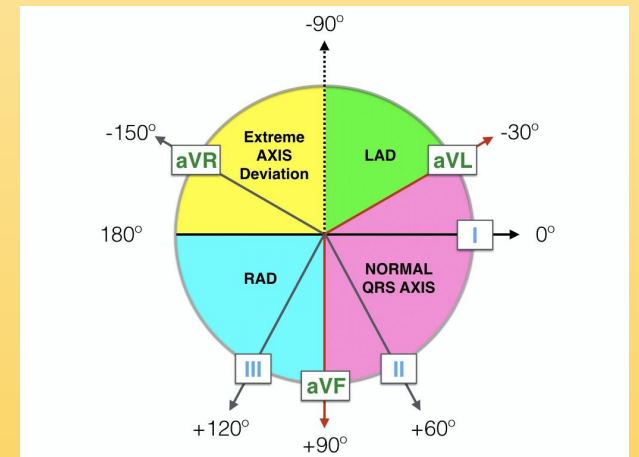


# Back to Basics-Axis



# Back to Basics

- Axis-normal from  $-30$  to  $90$ 
  - Remember that positive currents mean that the net depolarization is going towards that lead
  - Lead I lines up with  $0$  and aVF lines up with  $90$ 
    - If they are both positive you are between  $0$  and  $90$
    - If aVF is positive and I is negative you have RAD
    - If I is positive and aVF is negative, you are between  $0$  and  $-90$ 
      - To determine if LAD or normal, look at II
      - If II is positive-NAD
      - If II is negative-LAD





# PR Interval

- Atrial depolarization to ventricular depolarization
  - Tells you if the conduction from SA to AV node is working
- Shortened ( $<.2$ ) in WPW because the Bundle of Kent depolarizes before the conduction from SA to AV occurs
- Prolonged ( $>.2$ ) in AV Blocks

# QRS

- Back to the rule that normal conduction is the fastest-if the QRS is prolonged that means that the normal conduction pathway is not working
- Recall that the heart depolarizes SA->AV->Bundle of His (RBB and LBB, LBB further splits into anterior and posterior fascicles->Purkinje)
- If the heart is using the normal conduction pathway, the QRS (ventricular depolarization) should be less than .12s
- If the heart cannot use that conduction pathway because there is a block or current is going the wrong direction, it will take >.12s
  - RBBB, LBBB, Anterior hemi-block, posterior hemi-block, pacemaker, WPW

# ST

- ST segment will change as a reflection of ischemia and infarction
  - Can only be called if there are reciprocal changes
    - ST depression-ischemia
    - ST elevation-infarction
  - Occurs in group of leads
    - Inferior leads-II, III, aVF, give fluids (not nitroglycerin) because problem is preload and vasodilation decreases preload
    - Septal-V1-V2
    - Lateral-I, aVL
  - Diffuse STE seen in setting of pericarditis
    - NSAIDS and colchicine

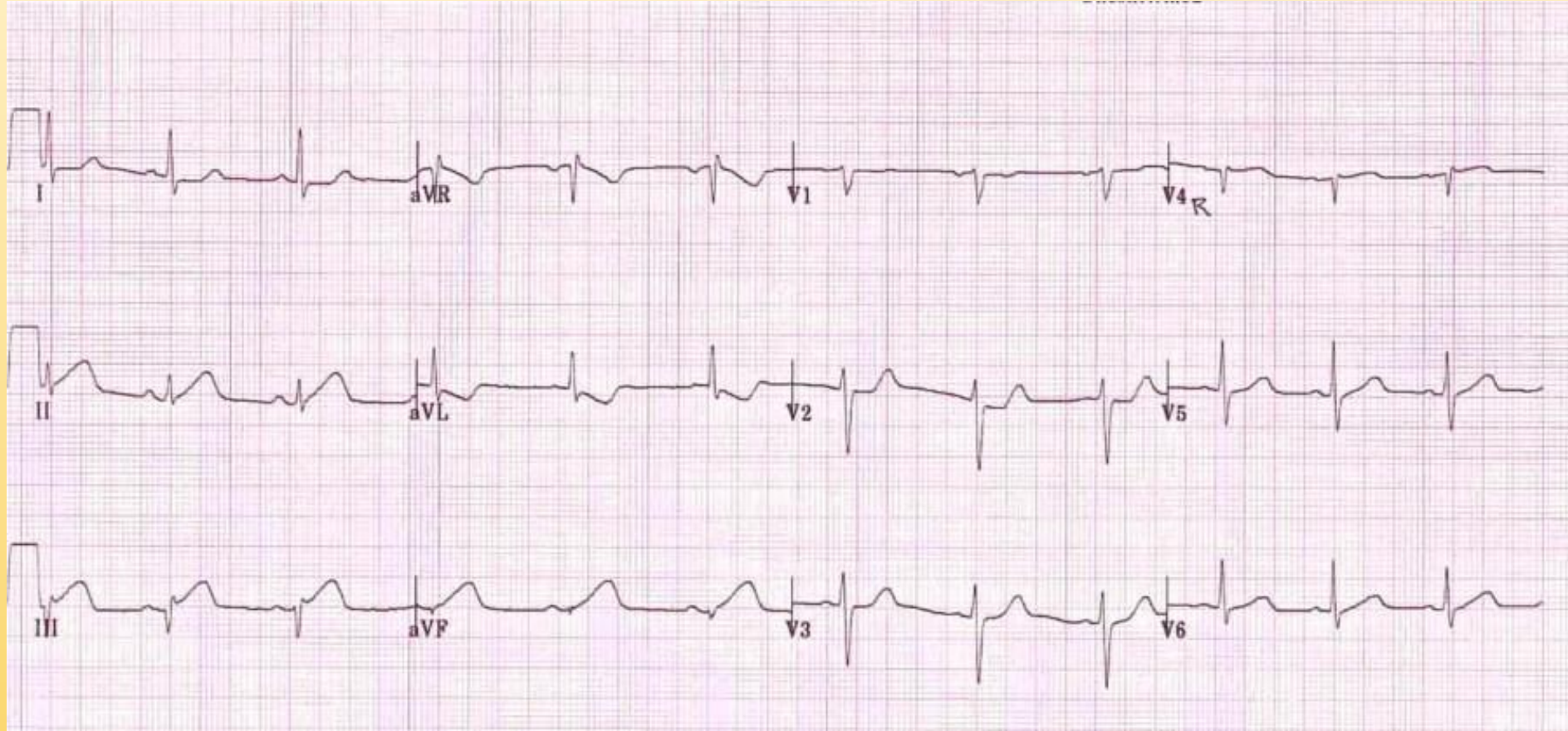
# ST

- Important to know groupings because they signify which artery is occluded
  - Nodes supplied by RCA
    - Can get HB from inferior MI
  - Dominance becomes important
    - MC PDA from RCA (R dominant)
    - PDA from LCX (L dominant)

## Lead Summary

I Lateral Circumflex Artery	aVR	V1 Septal Left Anterior Descending Artery	V4 Anterior Right Coronary Artery
II Inferior Right Coronary Artery	aVL Lateral Circumflex Artery	V2 Septal Left Anterior Descending Artery	V5 Lateral Circumflex Artery
III Inferior Right Coronary Artery	AVF Inferior Right Coronary Artery	V3 Anterior Right Coronary Artery	V6 Lateral Circumflex Artery

# ST

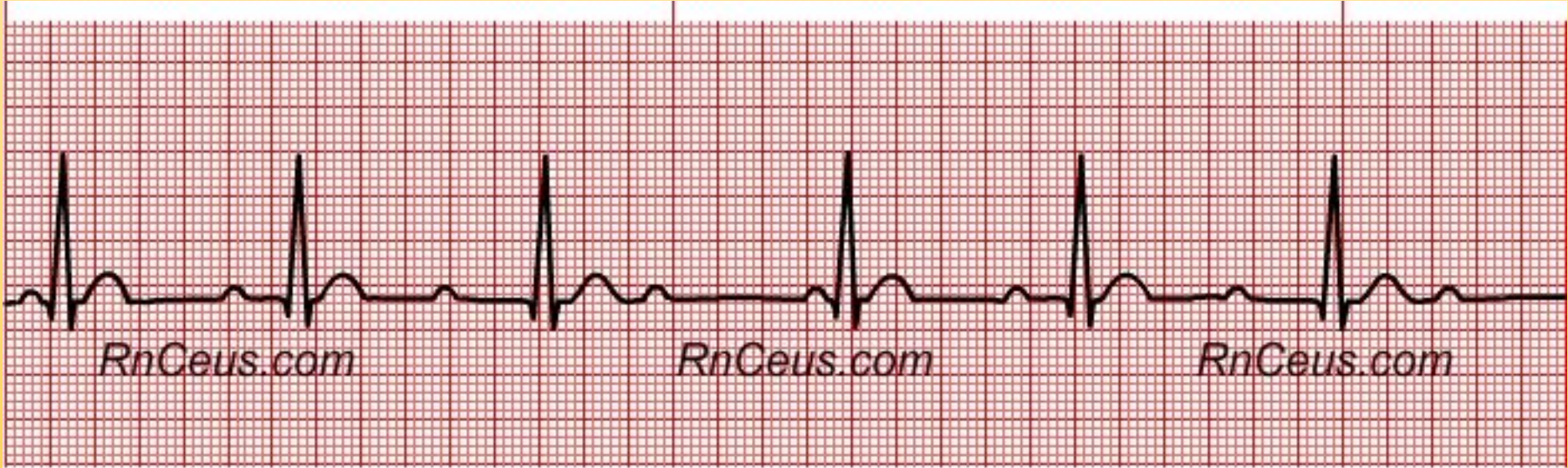


# AV Blocks

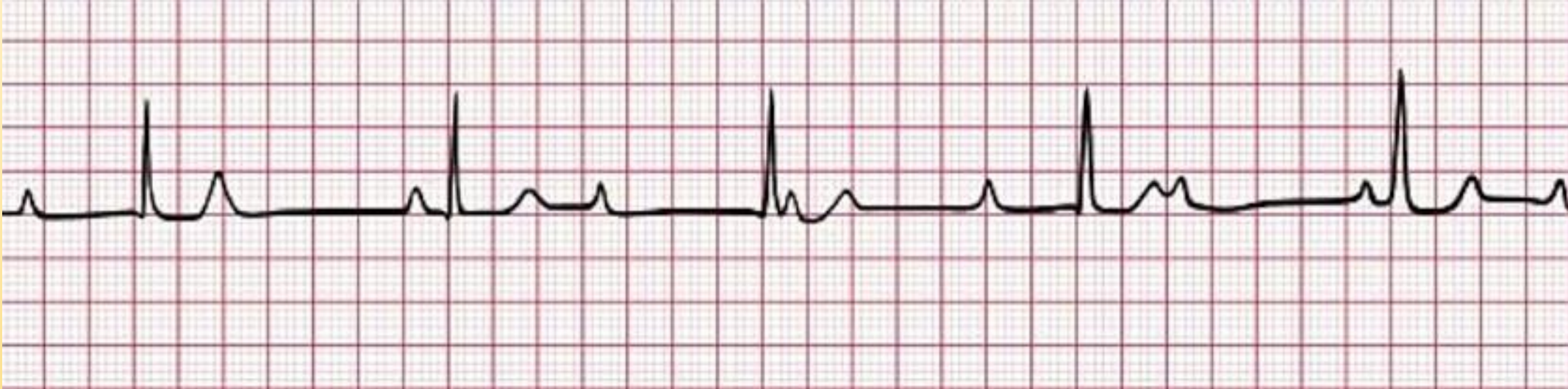
- Heart Block

- 1<sup>st</sup> Degree is no big deal, just a long PR interval
- 2<sup>nd</sup> Degree type 1-PR lengthens then drops
- 2<sup>nd</sup> Degree type 2-drops a beat without lengthening
- 3<sup>rd</sup> Degree-no association with P and QRS
  - Highest yield associations are with 3HB
    - Lyme disease, baby from mother with Lupus AC
  - Just for kicks and giggles-PE finding of 3HB is cannon a-waves
    - No association between cardiac cycle so you are shooting blood against a closed valve at times

# AV Blocks



# AV Blocks





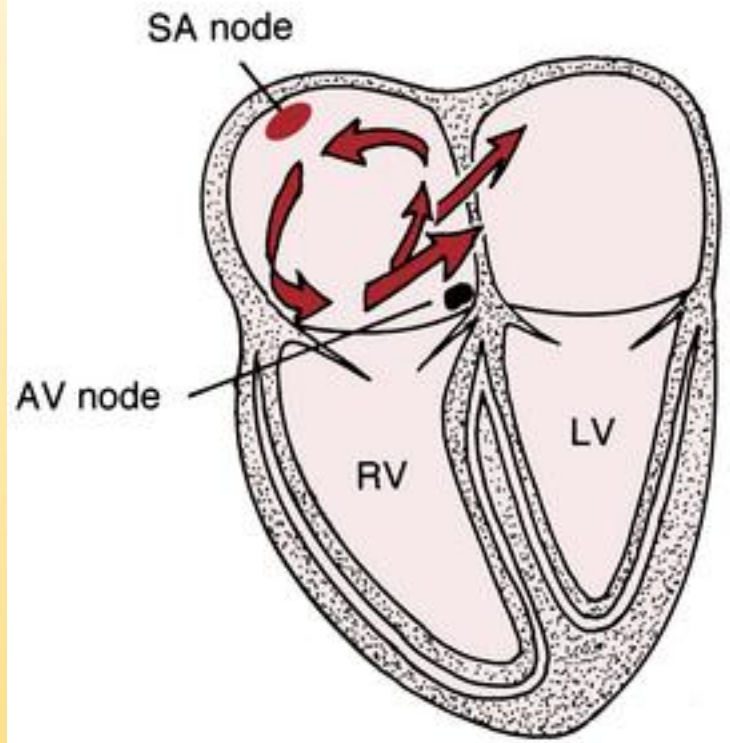
# Atrial Fibrillation

- Irregularly irregular
  - No p waves
  - Irregular RR intervals
- Risk factors-at the basic level is from AE
  - AE from HTN or CAD
  - Risk for stroke
    - Thrombus formation in the LA appendage
    - Rate or rhythm control and anti-coagulation
    - Cardioversion-TEE to ensure no thrombus in LA appendage
- MCC of new onset Afib is hyperthyroidism-reflexively check TSH/T4

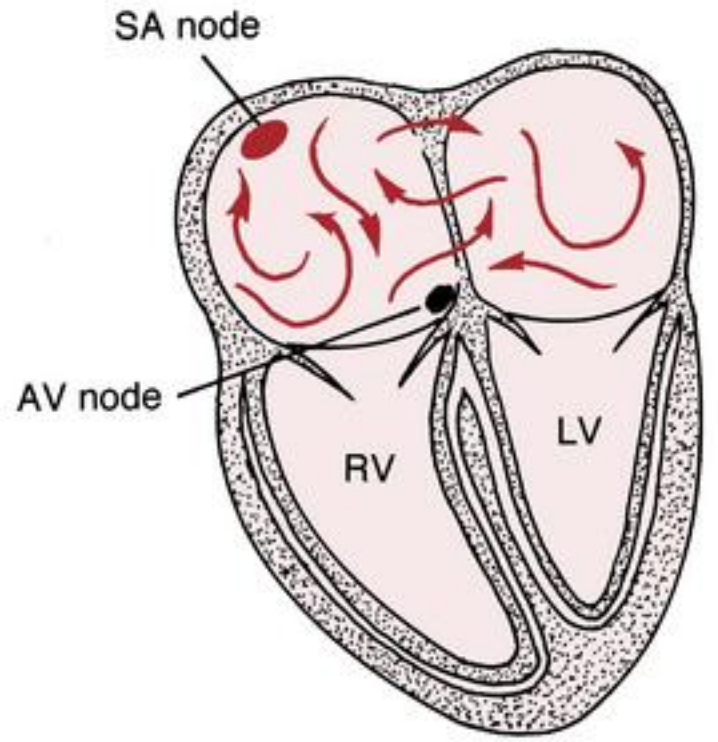
# Atrial flutter

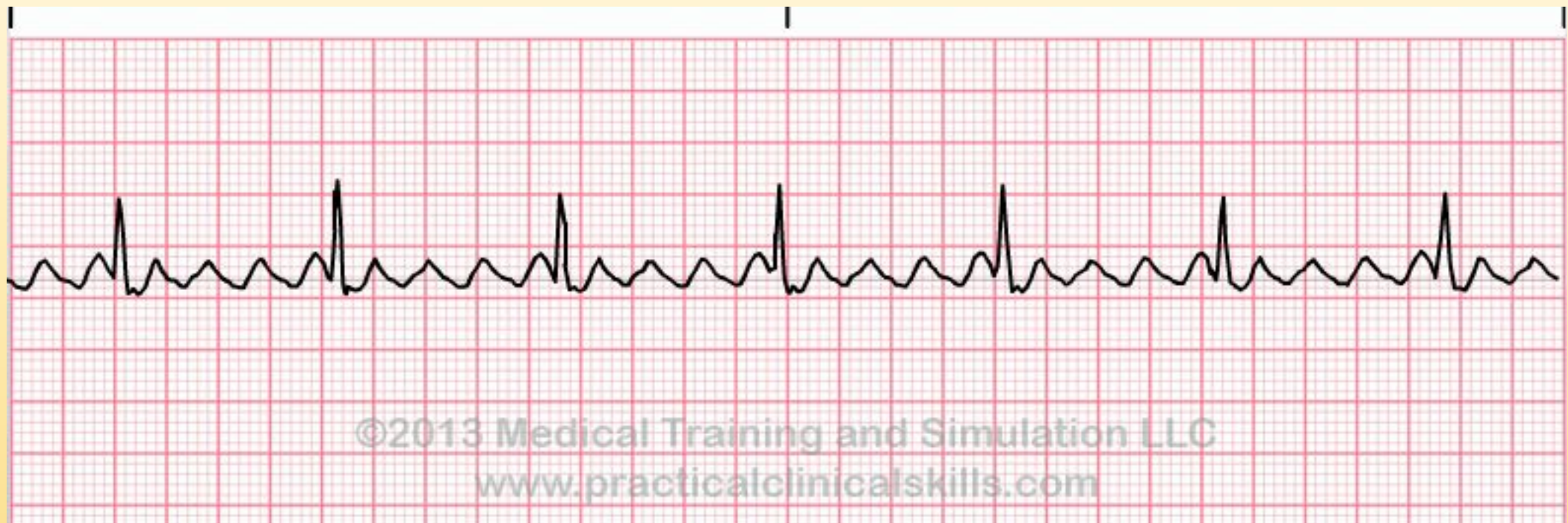
- Consistent electrical circuit
- Sawtooth appearance
  - For kicks and giggles-direction of the sawtooth pattern dictates if it can be ablated (RA>LA, medical management of LA ectopic circuit)
- Consistent RR interval
- Treat with OAC, rate or rhythm control

### ATRIAL FLUTTER



### ATRIAL FIBRILLATION





# Ventricular fibrillation

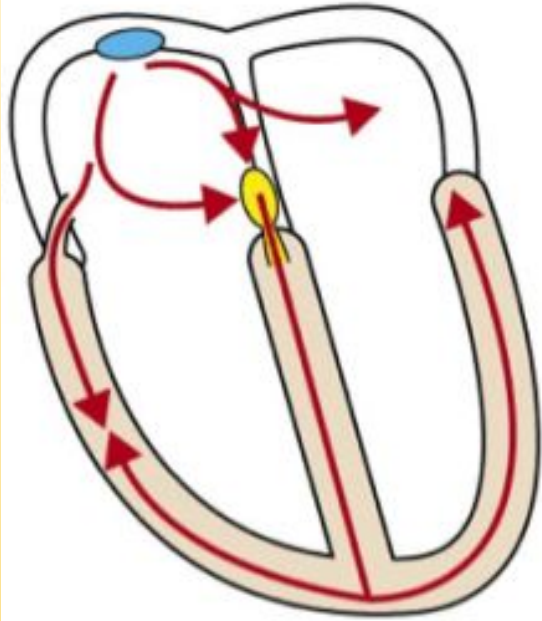
- Not using the right conduction pathway
- Wide QRS
- CPR and defibrillation
- This will kill ya



# WPW

- Short PR and prolonged QRS
- Accessory pathway in Bundle of Kent
- Treat with procainamide (IA antiarrhythmic)
  - Possible drug side effect-lupus

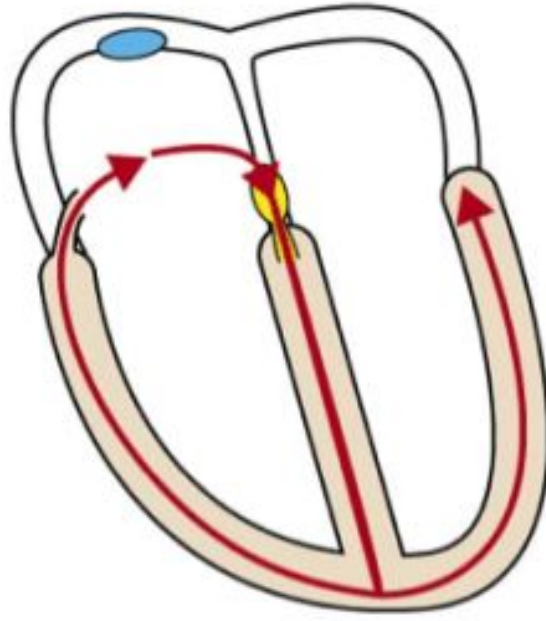
### Pre-excitation



- Short PR interval
- In this case the PR segment cannot be seen.

### Orthodromic AVRT

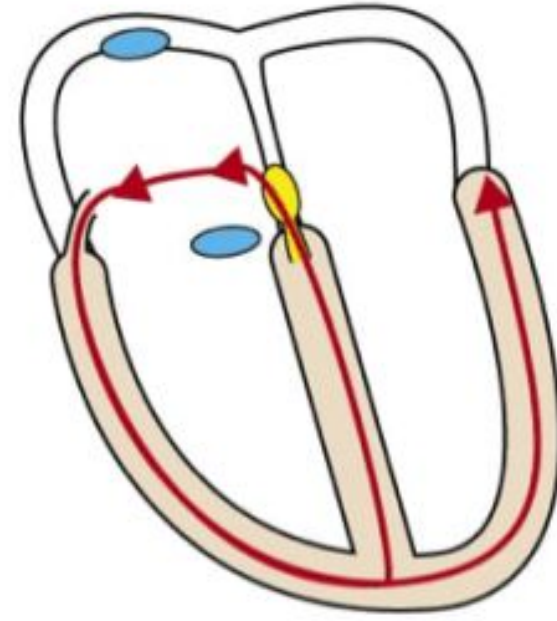
Antegrade conduction through atrioventricular node



- Normal QRS duration
- No delta wave
- Retrograde P-wave after QRS

### Antidromic AVRT

Retrograde conduction through atrioventricular node

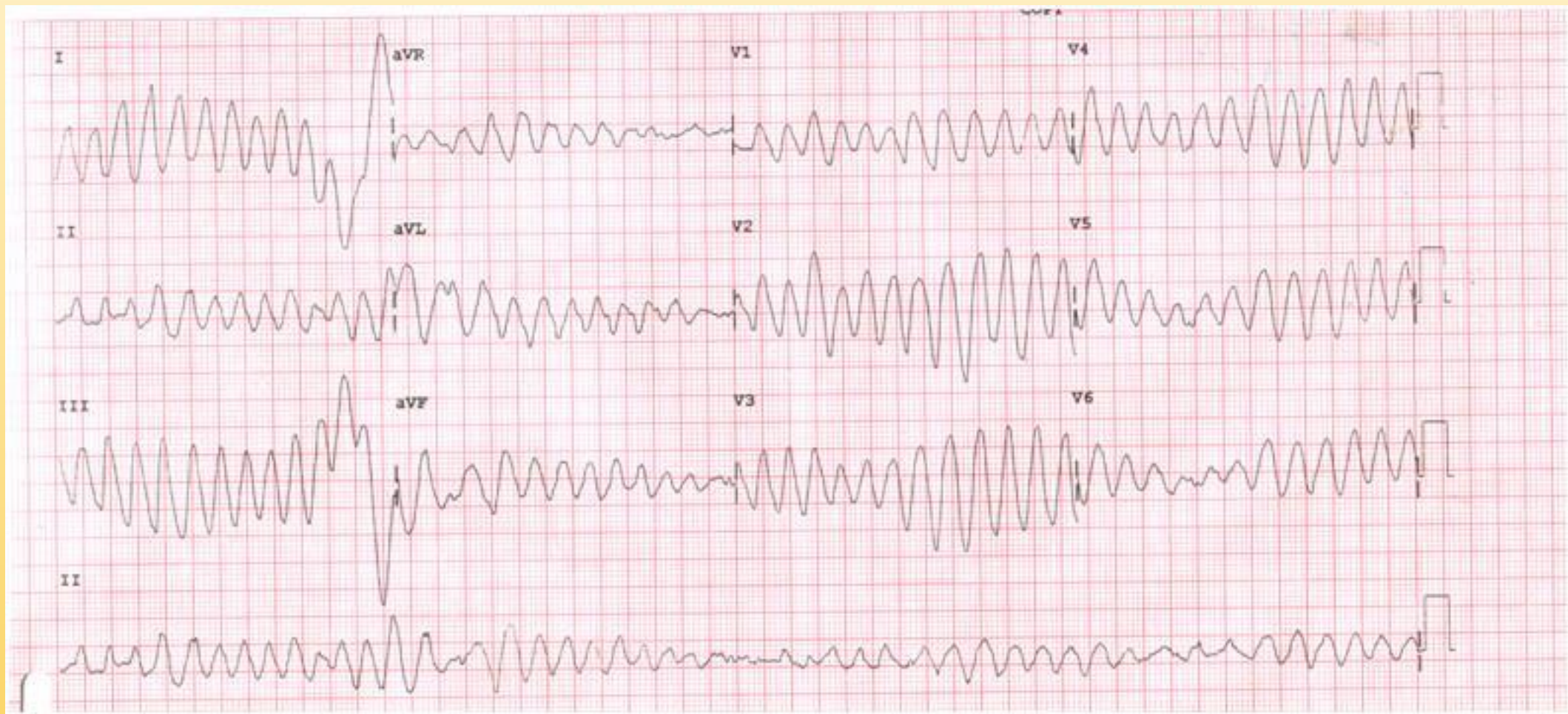


- Wide QRS complex with delta wave
- P-wave rarely seen
- If P-wave visible, it is retrograde and occurs just before the QRS

# Torsades

- Form of VTach that can quickly deteriorate into VFib
- Treatment-magnesium sulfate
- Can be caused by lots of drugs
  - Ondansetron
  - TCA-give sodium bicarb in TCA poisoning
  - Anti-arrhythmics
  - Antipsychotics
  - Antibiotics-macrolides, FQN



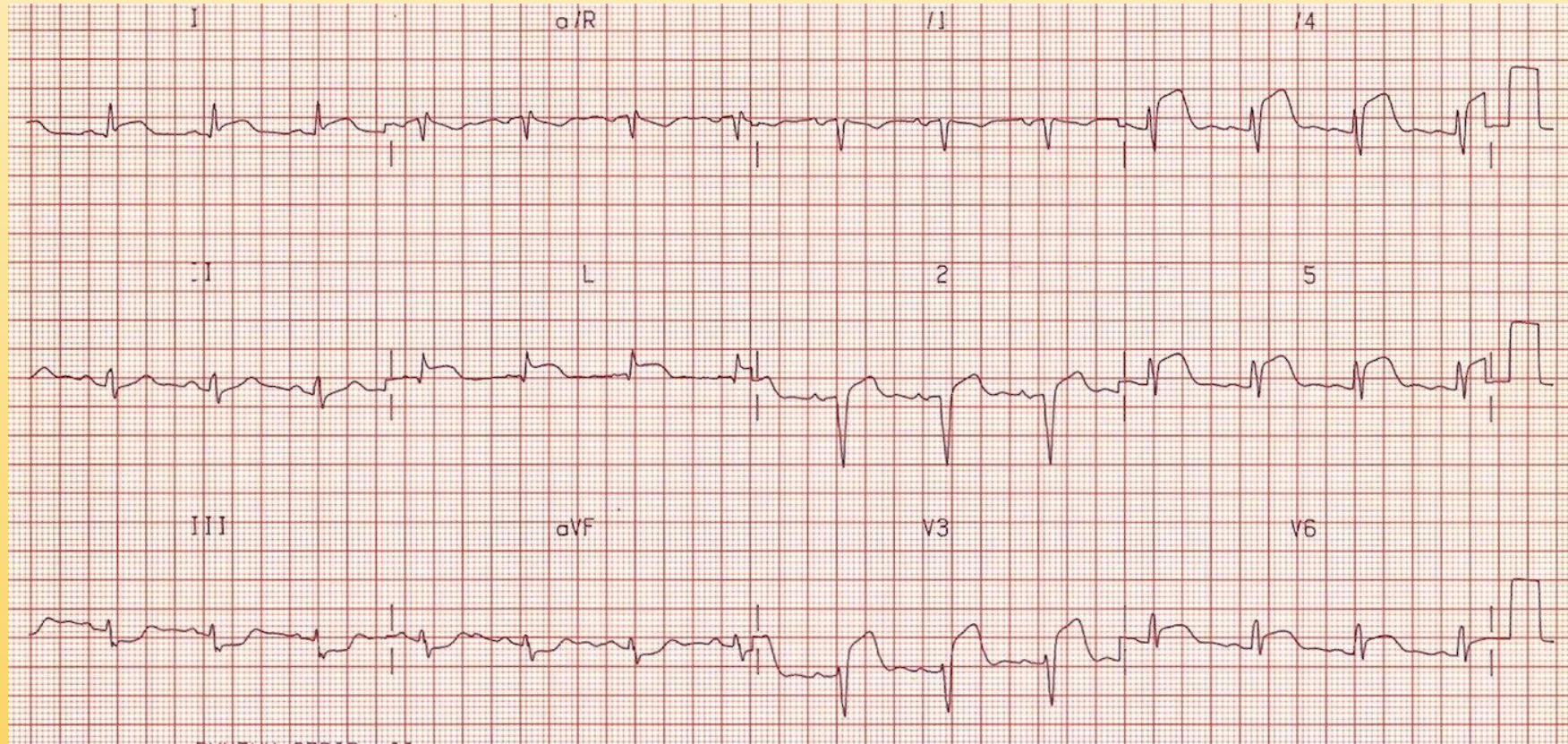


# TLDR EKG

- Normal conduction is the most efficient
- ST elevation transmural, must be in group with reciprocal changes to call as STEMI
- A fib disorganized, no p waves
- A flutter organized into “sawtooth” pattern
- Torsades rotating vfib (wide QRS), high yield drug causes, give Mg
- WPW delta wave and long QRS (give procainamide)
- AV Blocks-First degree long (no treatment), Second degree Type 1 lengthening and drop, Second degree Type 2 drop, Third degree no relationship (Lupus AC, Lyme Dz)

# Putting it all together

- A patient comes in with this EKG



# Looks like a STEMI

- Which leads/which artery?
  - Septal leads-LAD
- Well, a couple days later you listen to him and you hear a new holosystolic murmur
  - Can only be MR, TR, and VSD

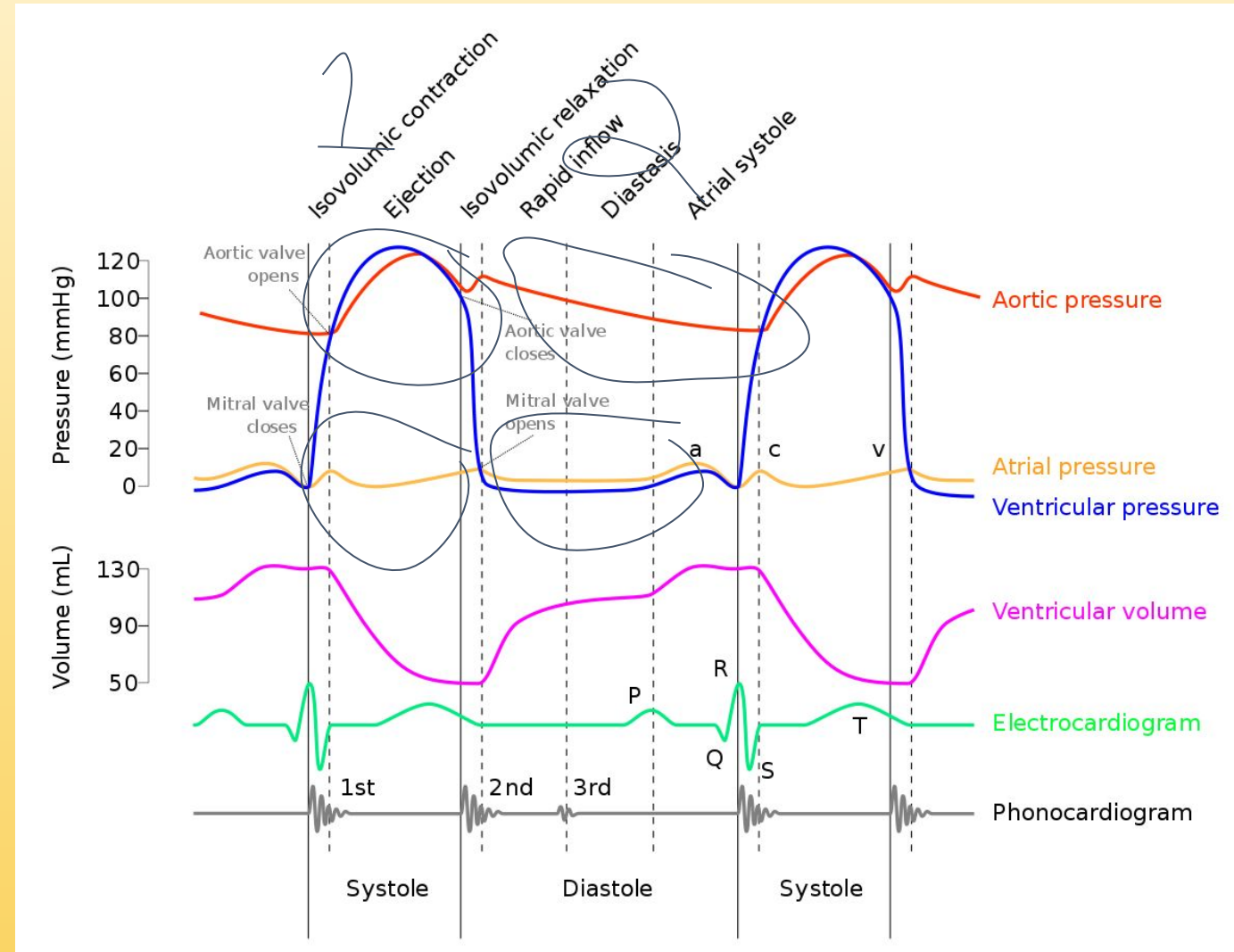
# What if they say that there is a step-up in O<sub>2</sub> concentration?

- New VSD from loss of interventricular septum

# Wigger's Diagrams

## Normal

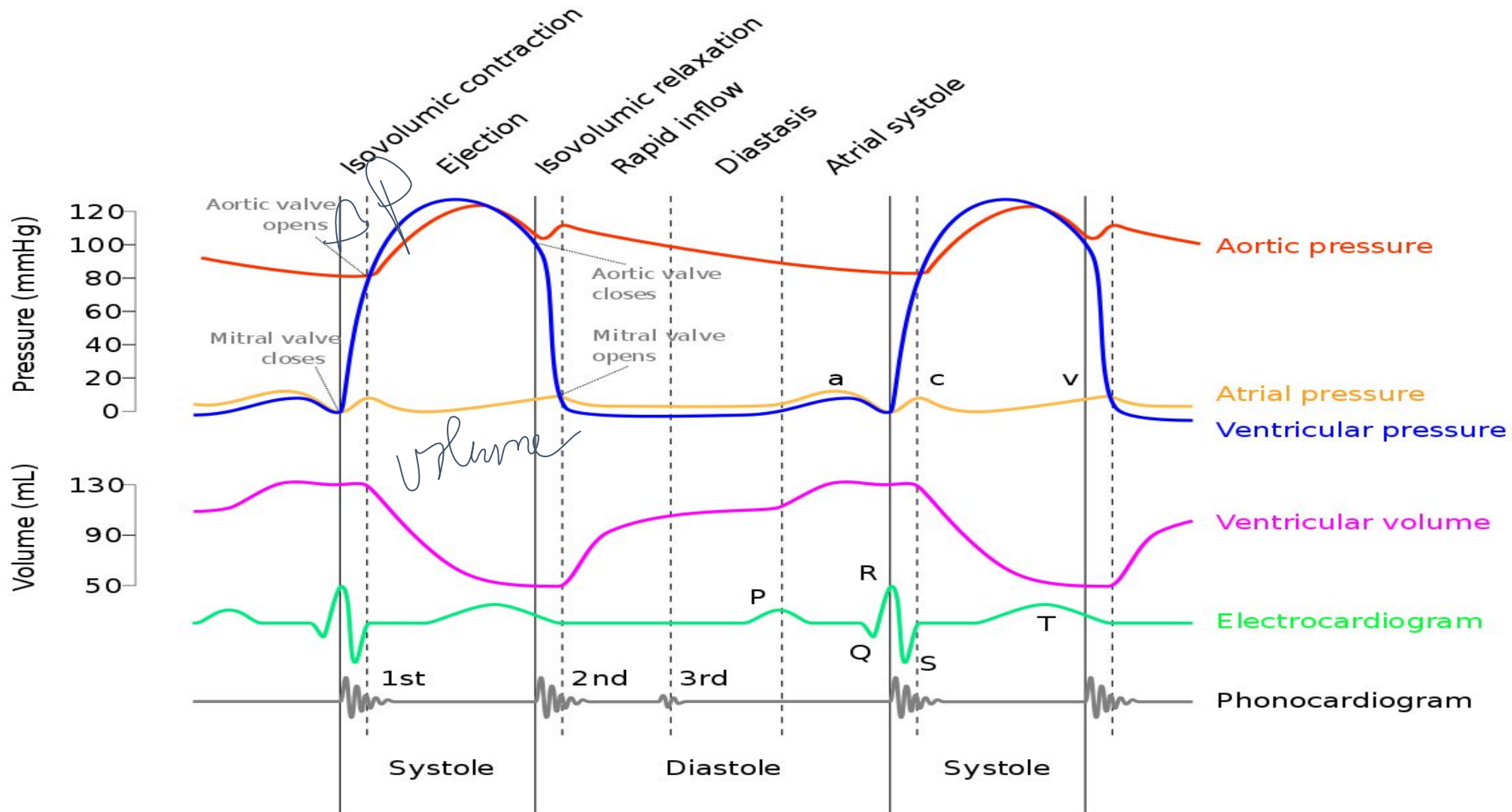
- Ventricular pressure is the portion that moves the most
- Opening of valves depends on pressure changes



# Systole (1 on the diagram)

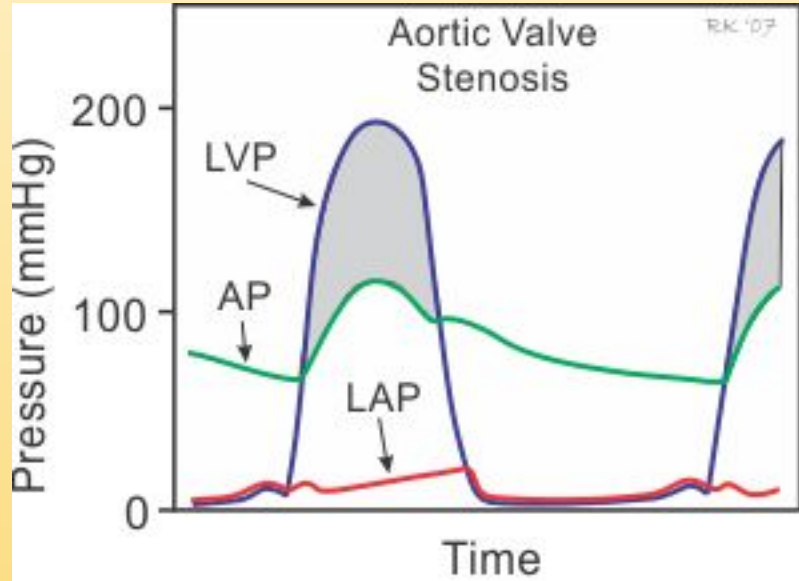
- Think of these graphs in two ways
  - Pressure differences that are too great
    - Occurs with stenotic valves
  - Filling too quickly
    - Occurs with regurgitation
- In systolic portion-pressure from AS/PS, filling from MR/TR

# Systole (1 on the diagram)

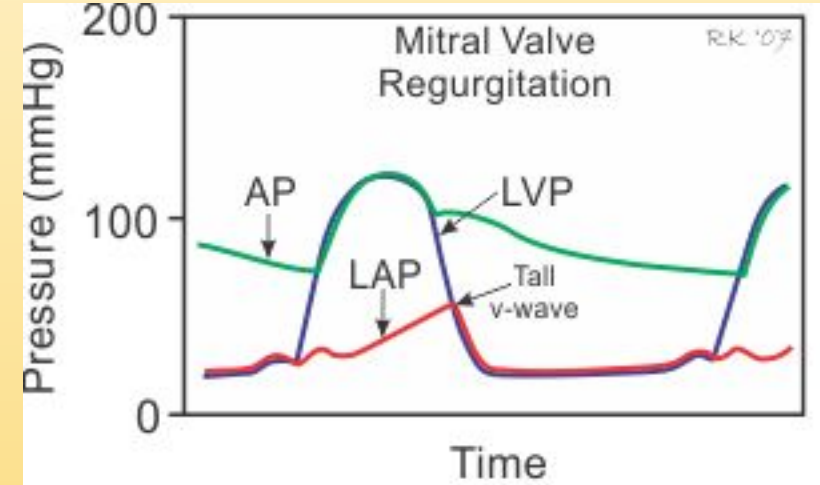




# Systole

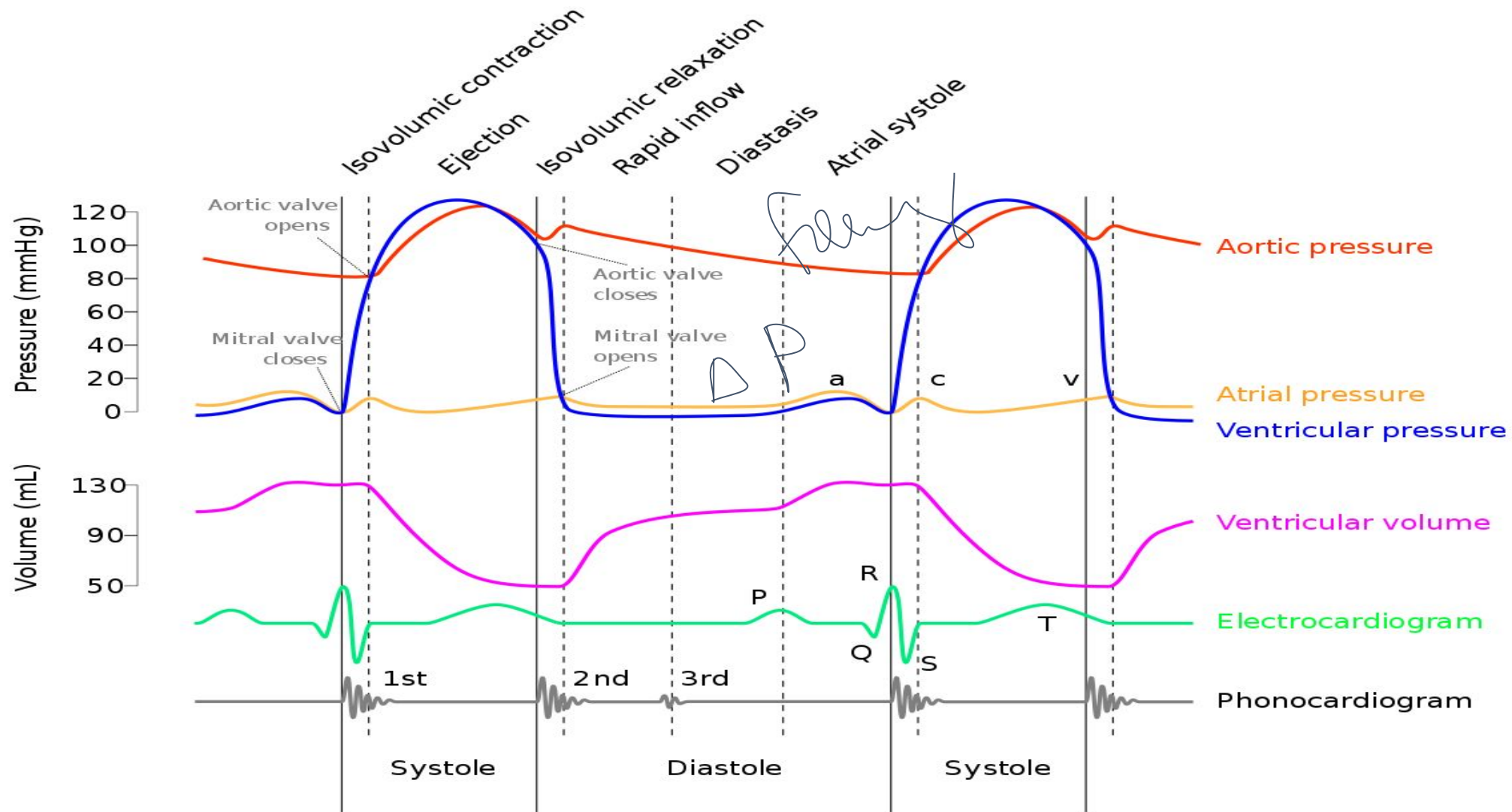


During ventricular ejection, LVP exceeds AP (gray area, pressure gradient generated by stenosis).  
*Abbreviations:* LAP, left atrial pressure; LVP, left ventricular pressure; AP, aortic pressure.

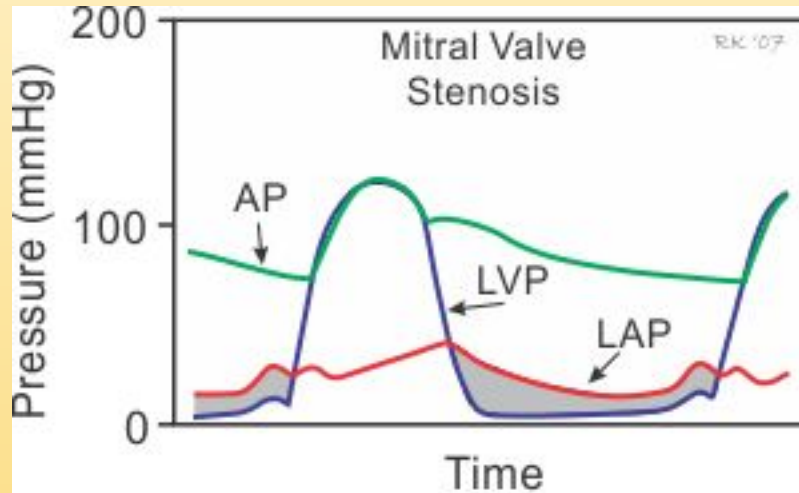


During ventricular contraction (systole), the left ventricle ejects blood back into the left atrium as well as into the aorta, thereby increasing LAP, particularly the v-wave. *Abbreviations:* LAP, left atrial pressure; LVP, left ventricular pressure; AP, aortic pressure.

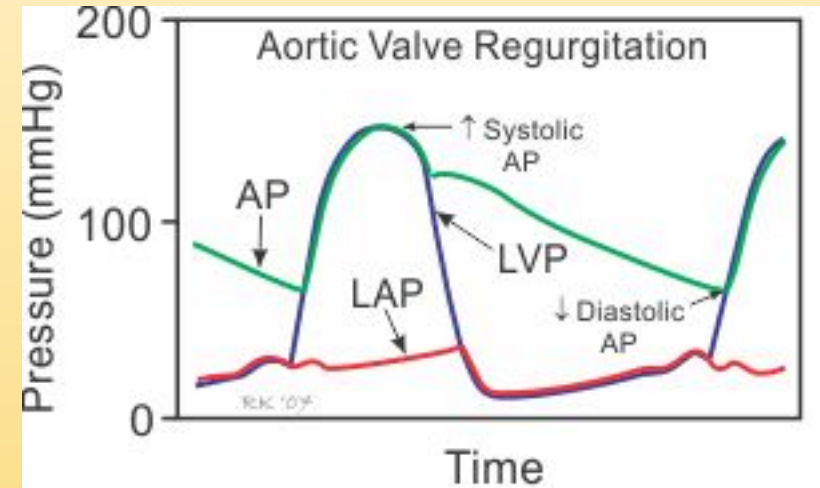
# Diastole (2 on the diagram)



# Diastole



During ventricular filling (diastole), LAP exceeds LVP (gray area, pressure gradient generated by stenosis). *Abbreviations:* LAP, left atrial pressure; LVP, left ventricular pressure; AP, aortic pressure.



During ventricular relaxation, blood flows backwards from aorta into the ventricle. Aortic systolic pressure increases, aortic diastolic pressure decreases, and pulse pressure increases; LAP increase. *Abbreviations:* LAP, left atrial pressure; LVP, left ventricular pressure; AP, aortic pressure.

# TLDR Wigger's Diagram

- Pressure differences greater than expected from stenotic valves
  - Systolic-AS
  - Diastolic-MS
- Filling too quickly from regurgitation
  - Systolic-MR
  - Diastolic-AR

# Good Luck!

Questions or comments:

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