

ECG

Interpretation



Outlines:

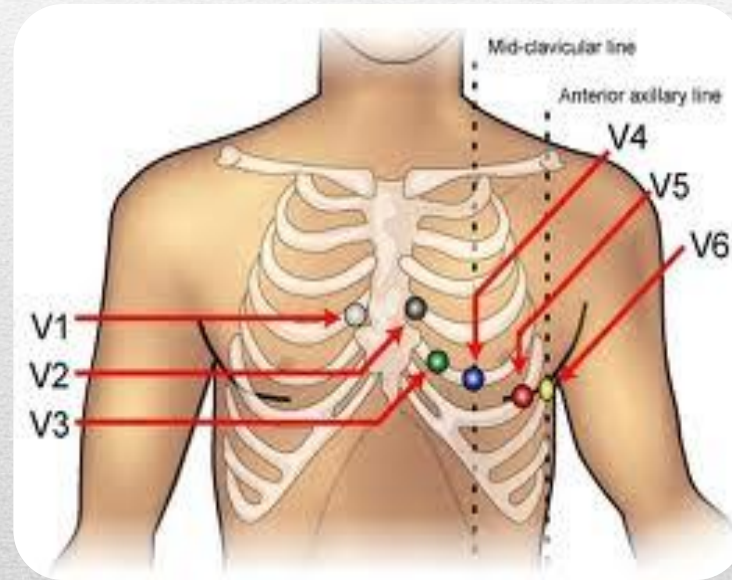
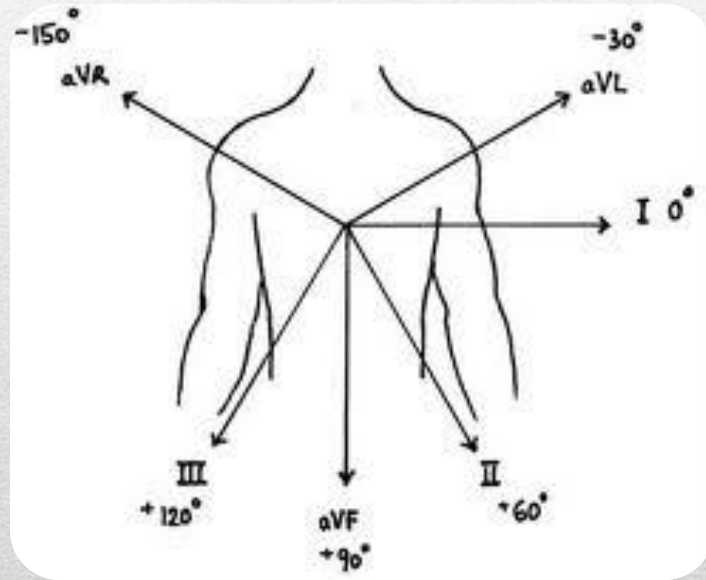
- ECG definition.
 - Leads presentation.
 - Electrical conduction.
 - How to read an ECG paper.
 - Cardiac dysrhythmias.
-

Part 1

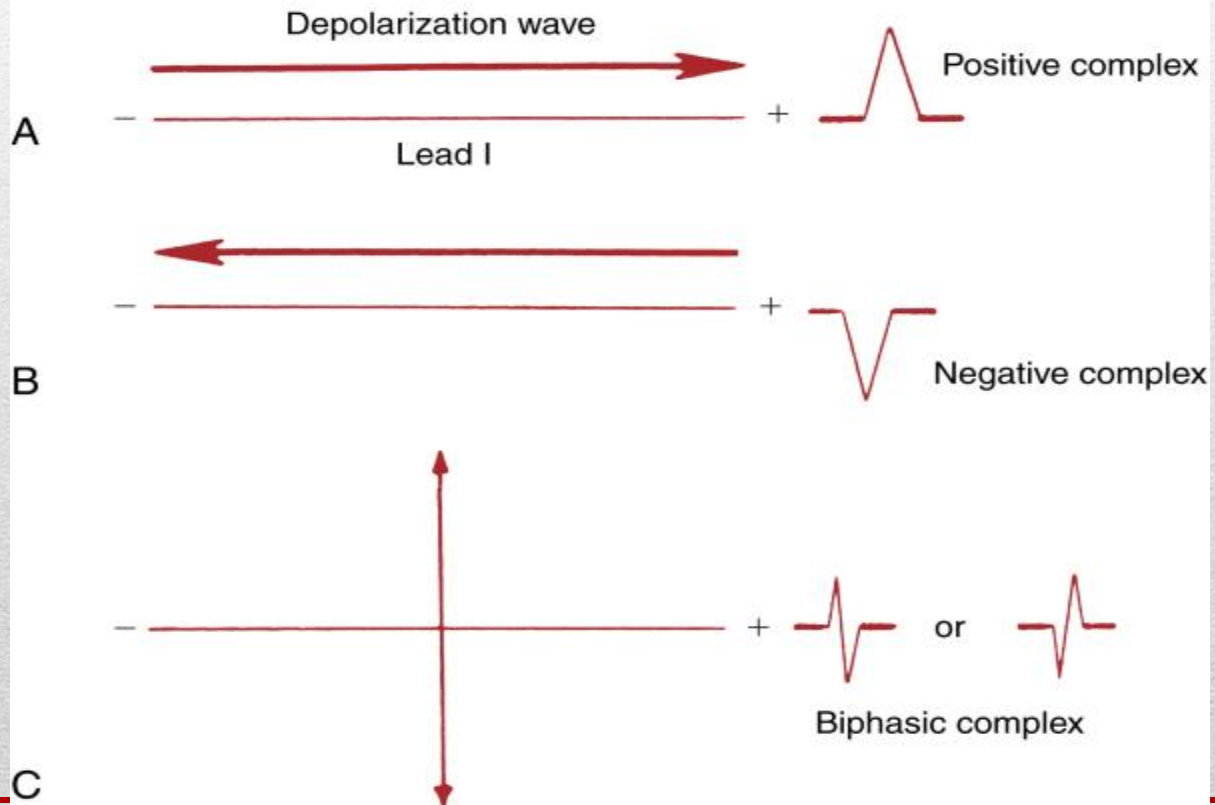
- **ECG definition.**
- **Leads presentation.**
- **Electrical conduction.**
- **How to read an ECG paper.**



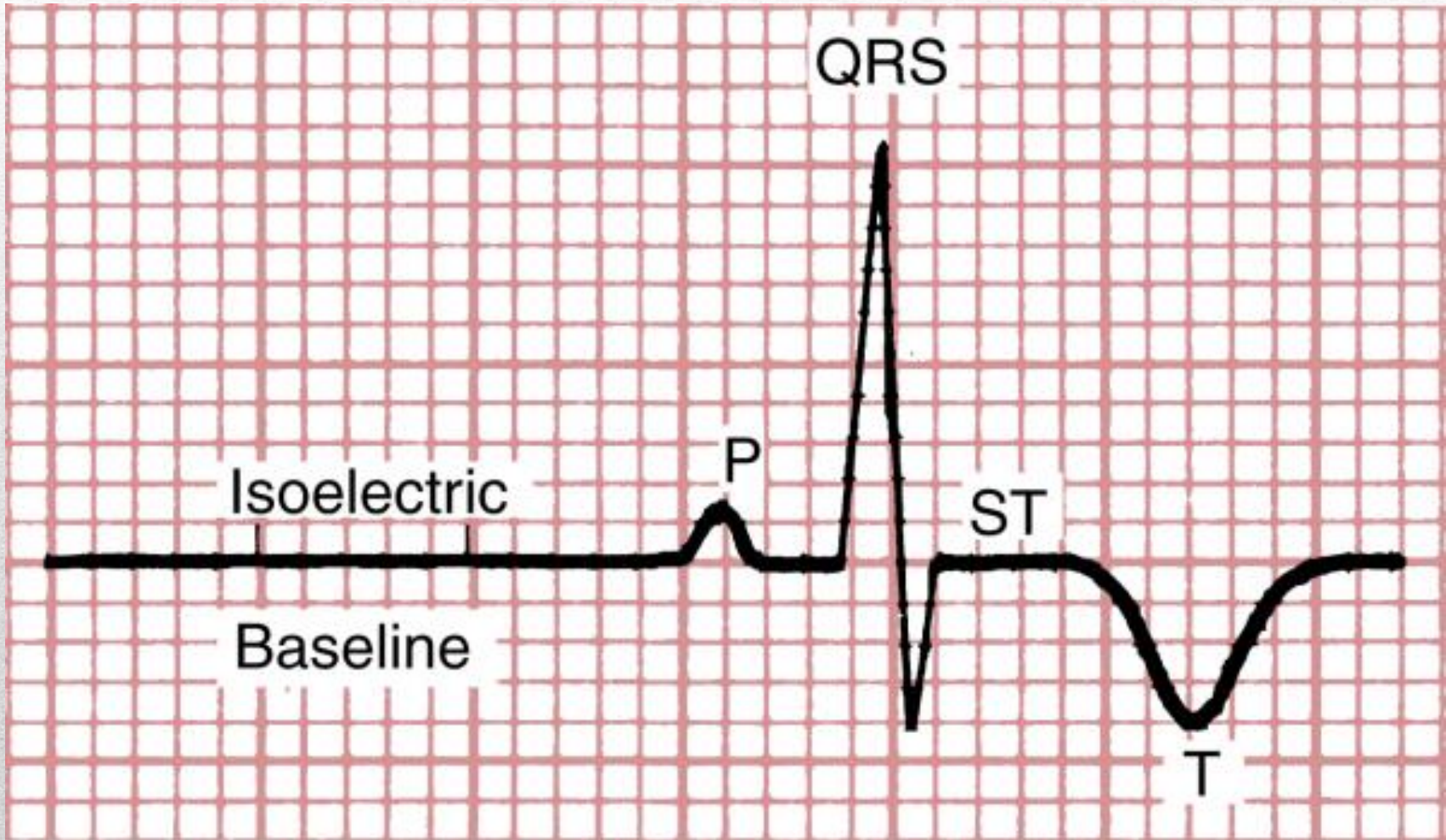
Leads Presentation



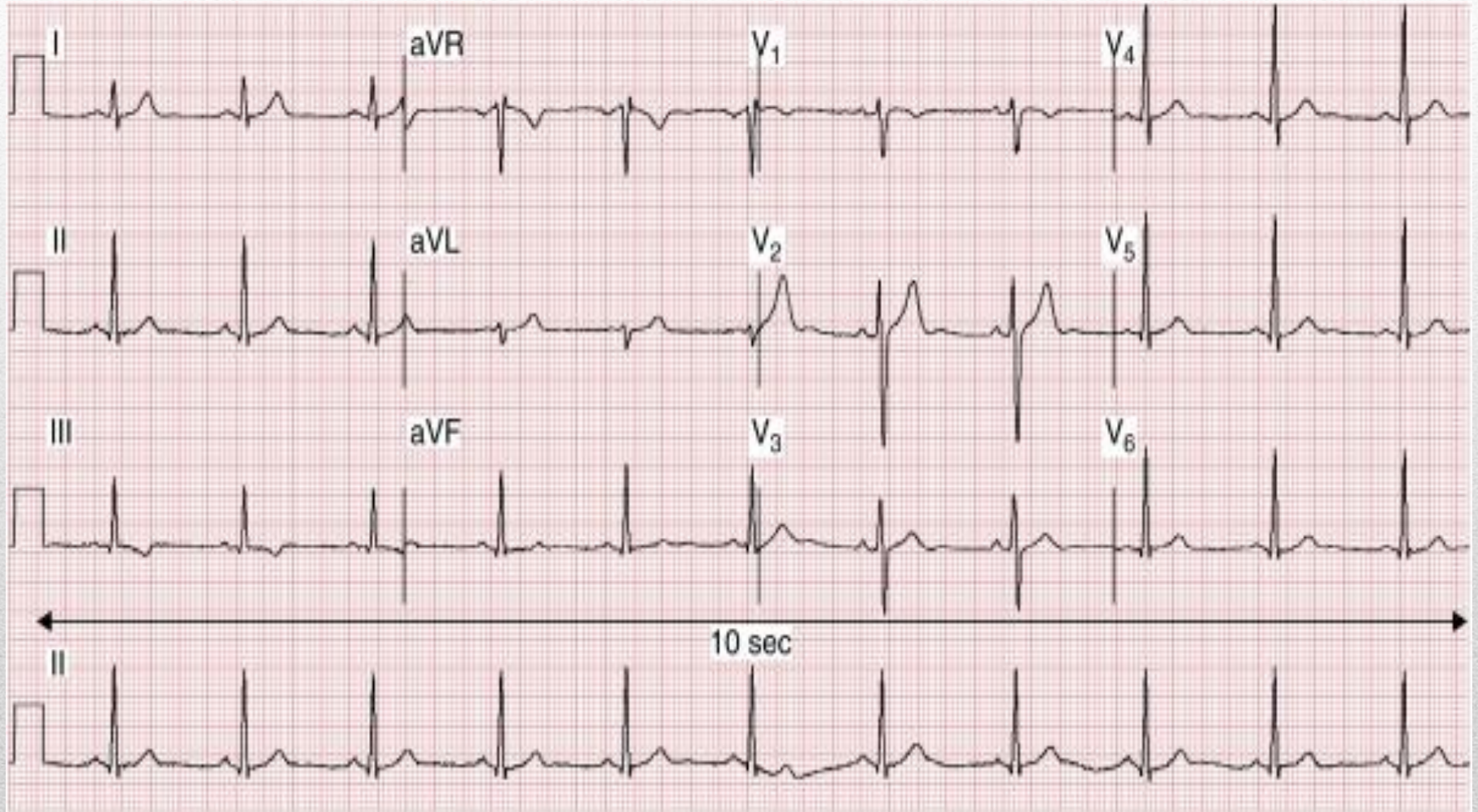
Three Basic Laws of Electrocardiography



Waves



12 Leads EKG

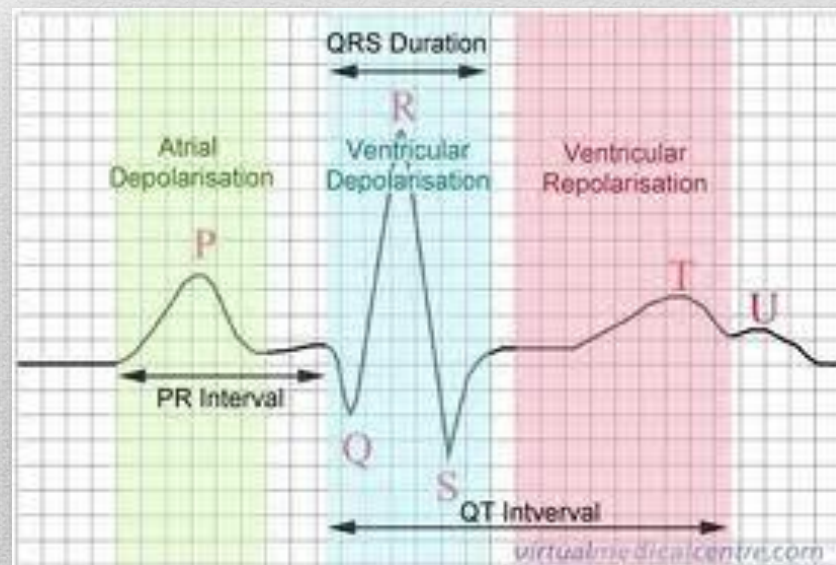


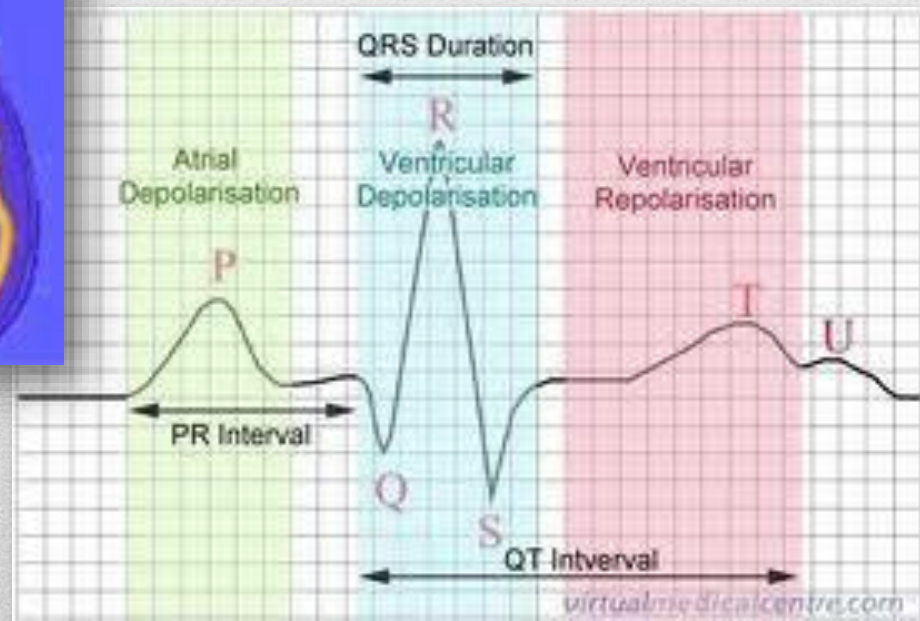
QRS Complex

It represents ventricular depolarization.

Normal QRS duration

≤ 3 ss (≤ 120 seconds)





Electrical Conduction

Waves



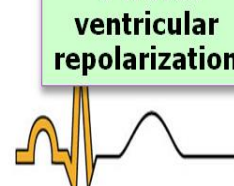
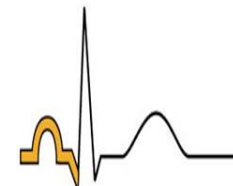
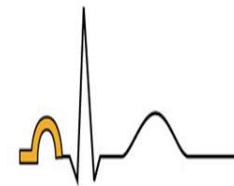
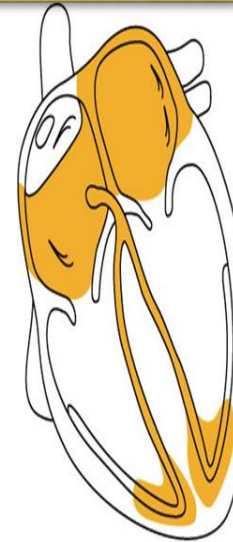
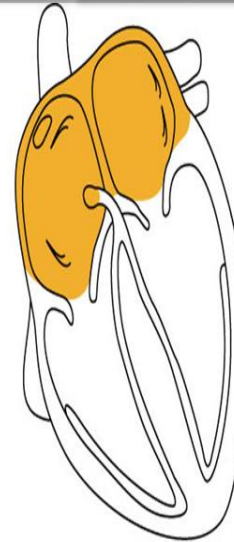
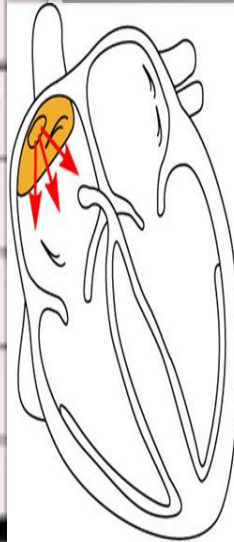
Sequence of cardiac excitation

SA node generates impulse; atrial excitation begins

Impulse delayed at AV node

Impulse passes to heart apex; ventricular excitation begins

Ventricular excitation complete



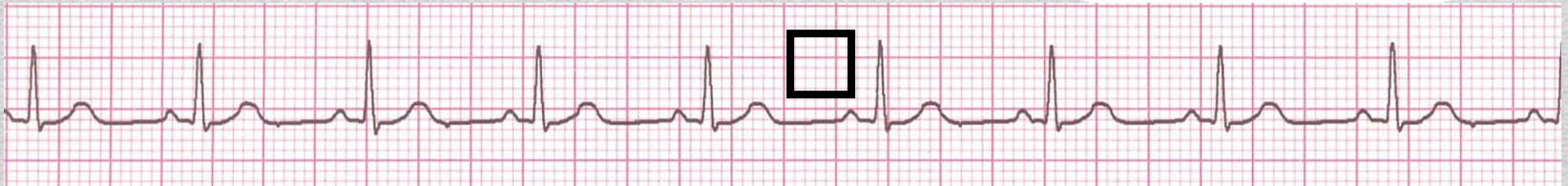
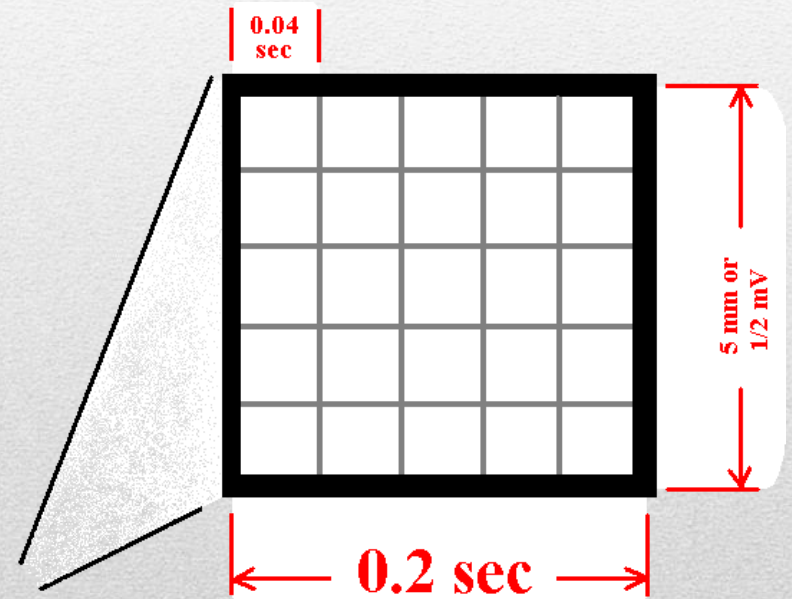
P wave: Atrial depolarization

QRS complex: ventricular depolarization

T wave: ventricular repolarization

The ECG Paper

- Horizontally
 - One small box - 0.04 s
 - One large box - 0.20 s
- Vertically
 - One large box - 0.5 mV



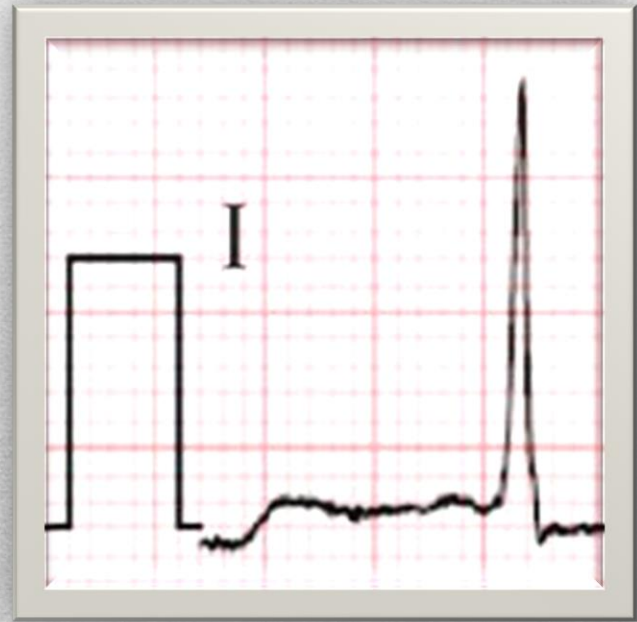
How to interpret ECG

- Caliber.
- Rhythm.
- Rate.
- Axis.
- Waves.



Caliber

It should be ≤ 10 mm otherwise you have to repeat the ECG.



Normal Sinus Rhythm



Rhythm

- **LEDA II:** Positive P wave preceding each QRS.
- **V1:** Biphasic P wave.

Lead II

V1



Rate

- **Normal sinus rate:** (60-100).
 - **Tachycardia:** >100 .
 - **Bradycardia:** <60 .
-

How to Calculate Rate

- $300/\text{No. of large squares R-R.}$
- $1500/\text{No. of small squares R-R.}$
- $\text{No. of (R) waves in the strip} \times 6.$
- $300 \ggg 150 \ggg 75 \ggg 60 \ggg 55$



Rate

Start if possible on a beat whose QRS (usually R wave) is on the border of a large square

Count Large squares
(0.2 seconds each)

300 100 60 40

150 75 50



This tracing example shows a rate of 100 bpm

Rate determination for irregular rhythm

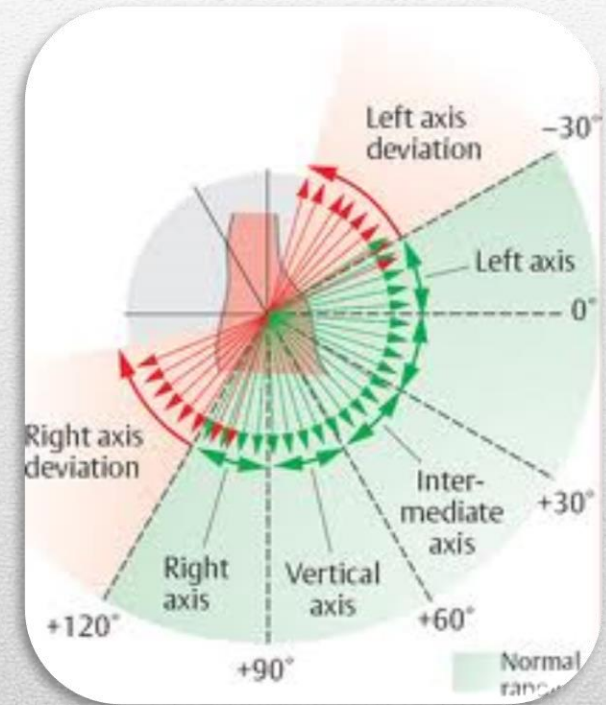


For irregular rhythm (such as atrial fibrillation), the method shown on the last slide may be inaccurate. Use this alternate method.

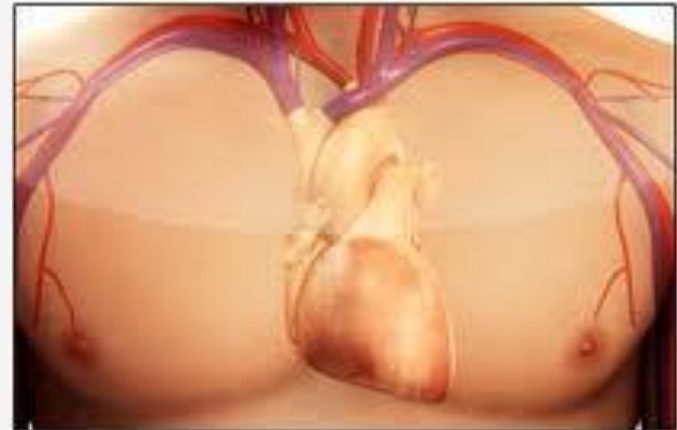
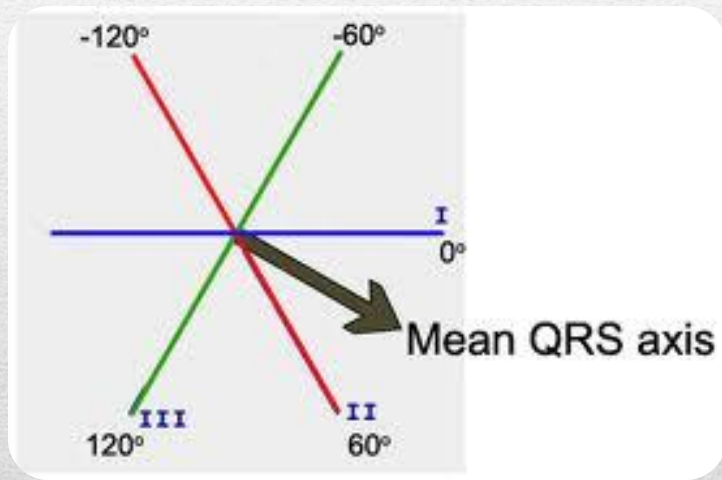
Start as before by finding a QRS that lands on the border of a large square (*). Then count 30 large squares ($= 0.2 \times 30 = 6$ seconds). Add up all beats (QRSs) that land within the interval (not counting that first beat (*)) and multiple by 10. This equals the number of beats per minute.

Axis

- Normal axis.
- Right axis deviation.
- Left axis deviation.
- Extreme axis deviation.



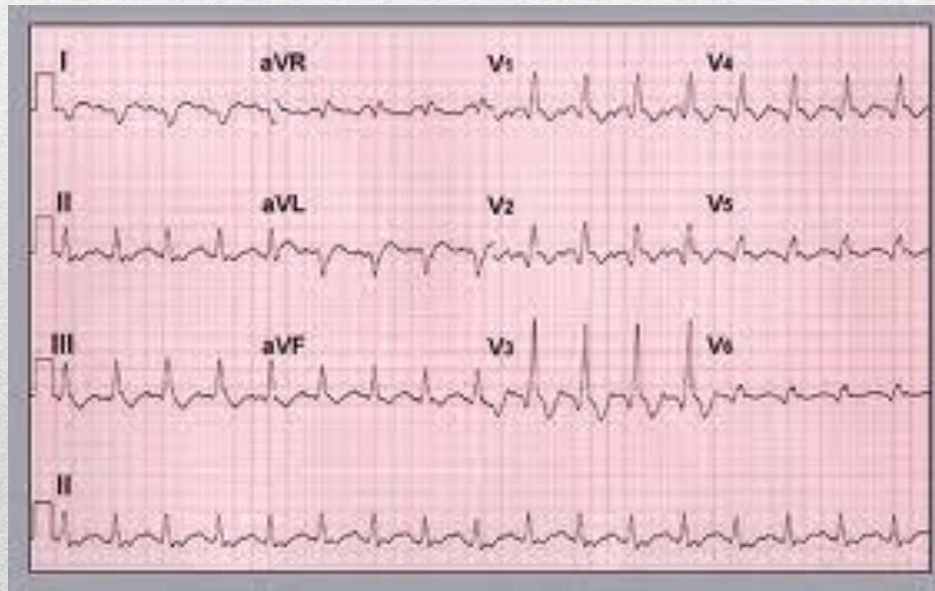
Normal Heart Axis



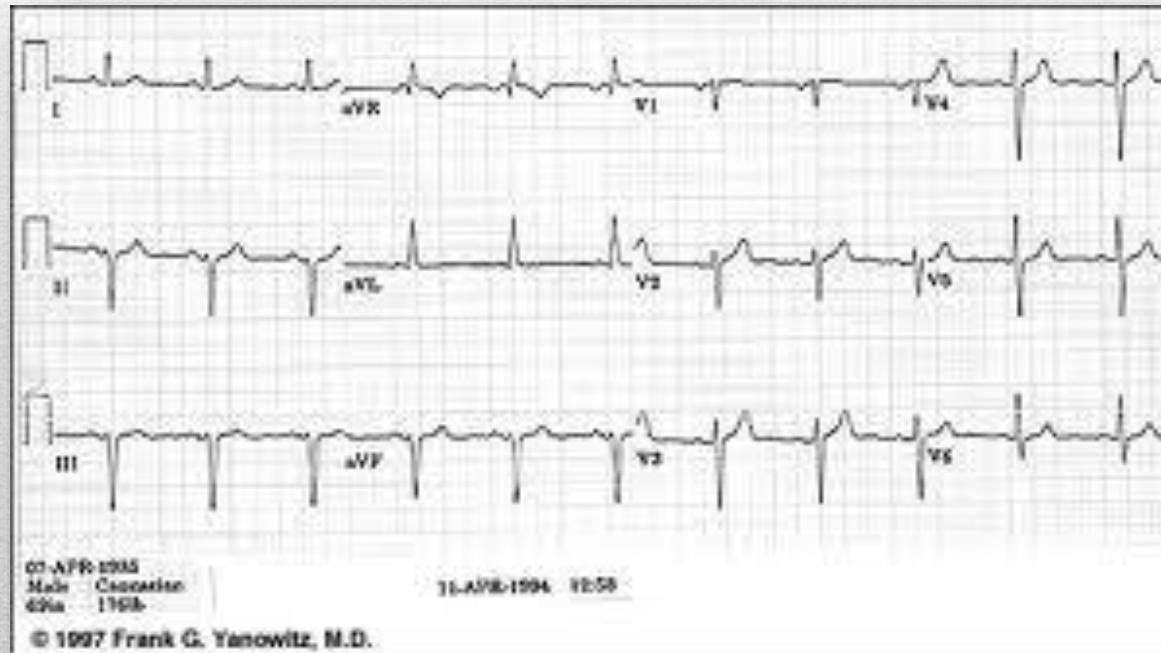


Normal Heart Axis Cont..

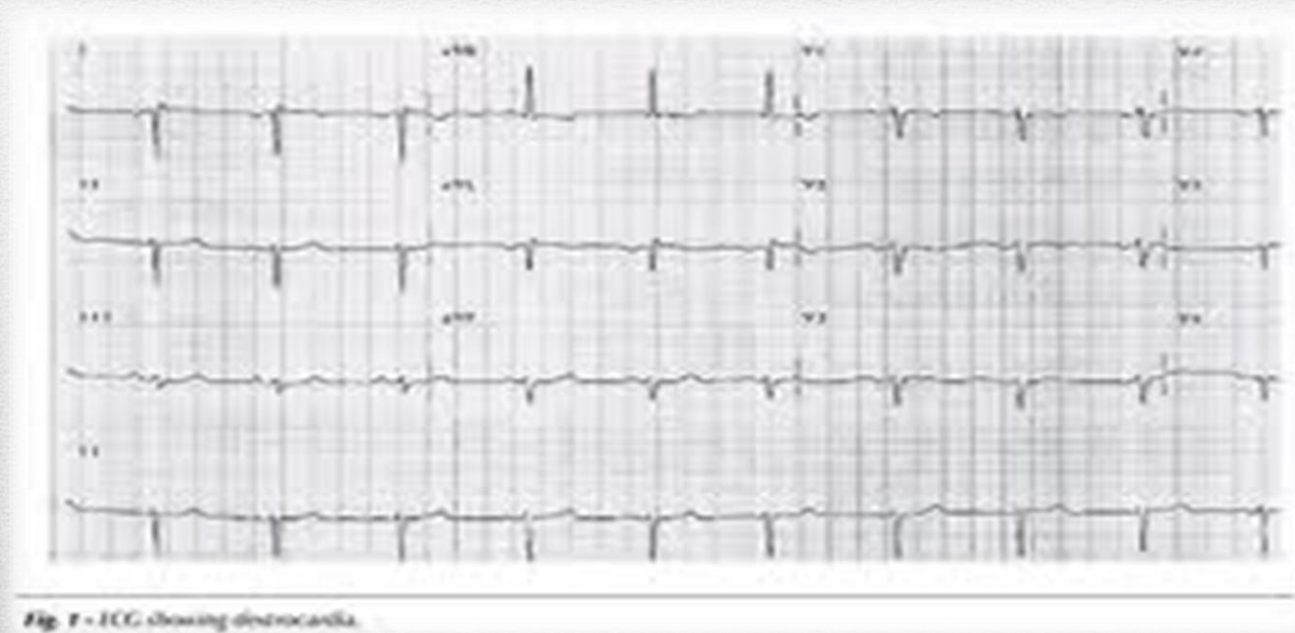
Right Axis Deviation



Left Axis Deviation

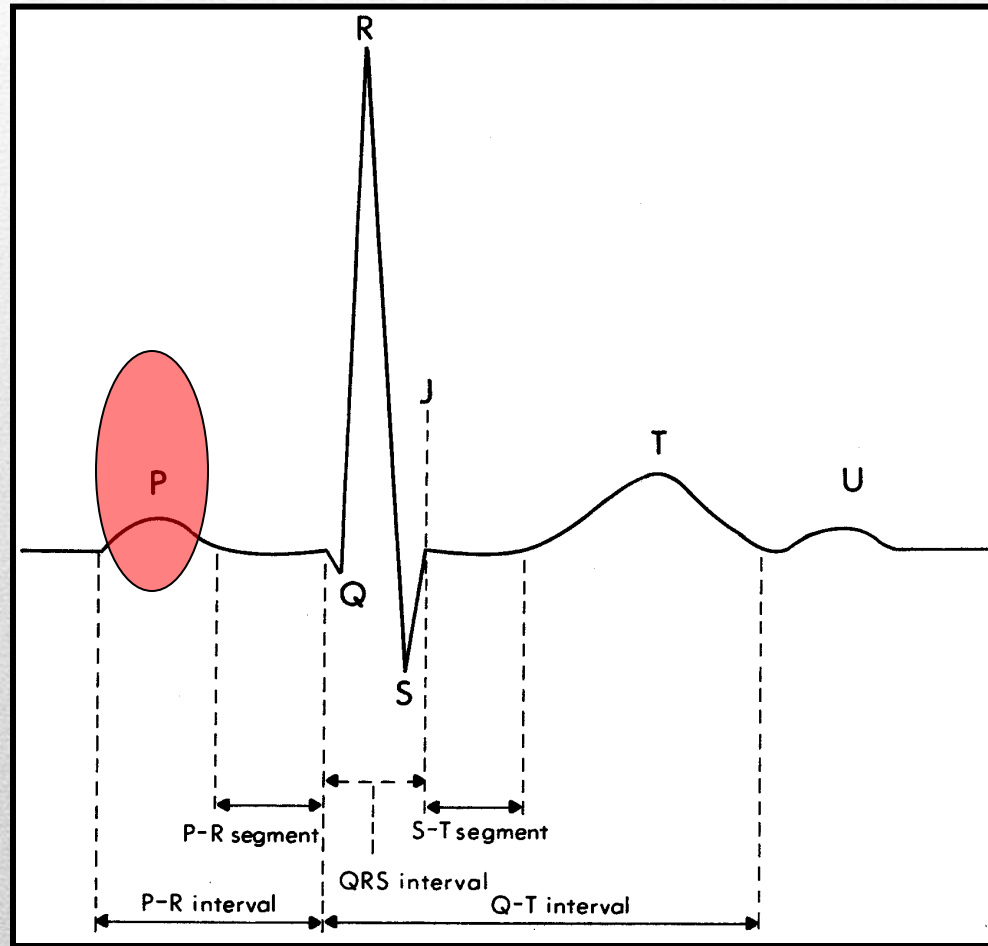


Extreme Axis Deviation



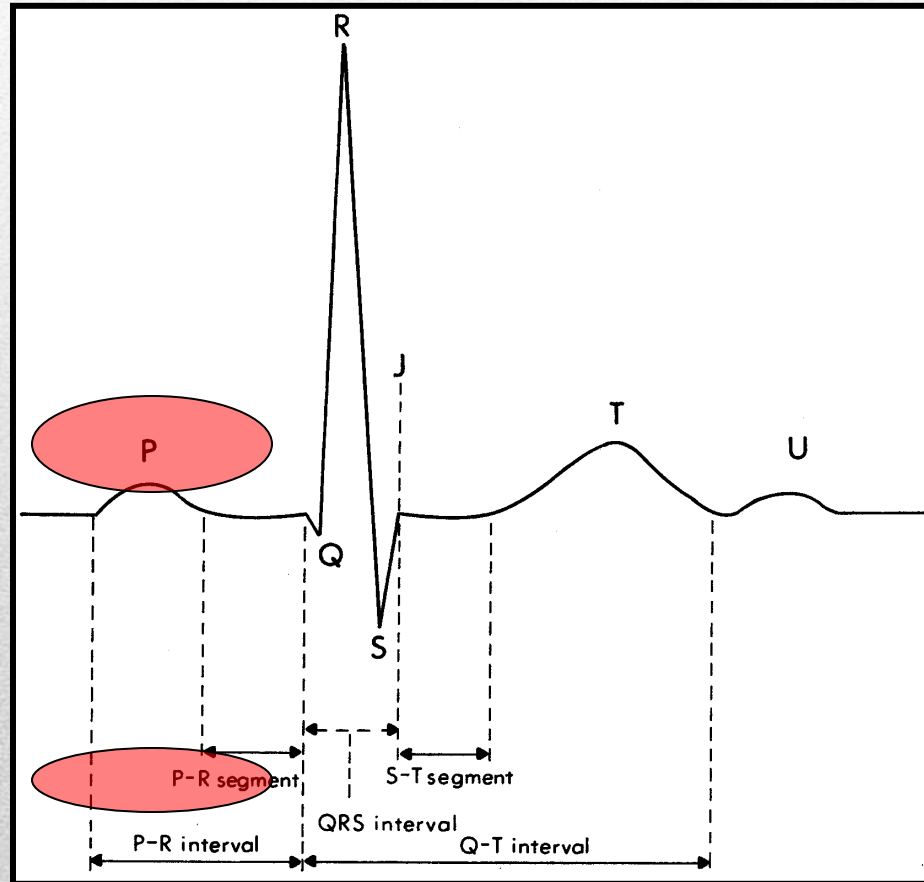
The P Wave

- Normally from sinus node
- Upright in I, II, aVF, V4-V6
- Monophasic (except V1)
- Normal ranges:
 - < 0.12 sec wide
 - < 2.5 mm tall

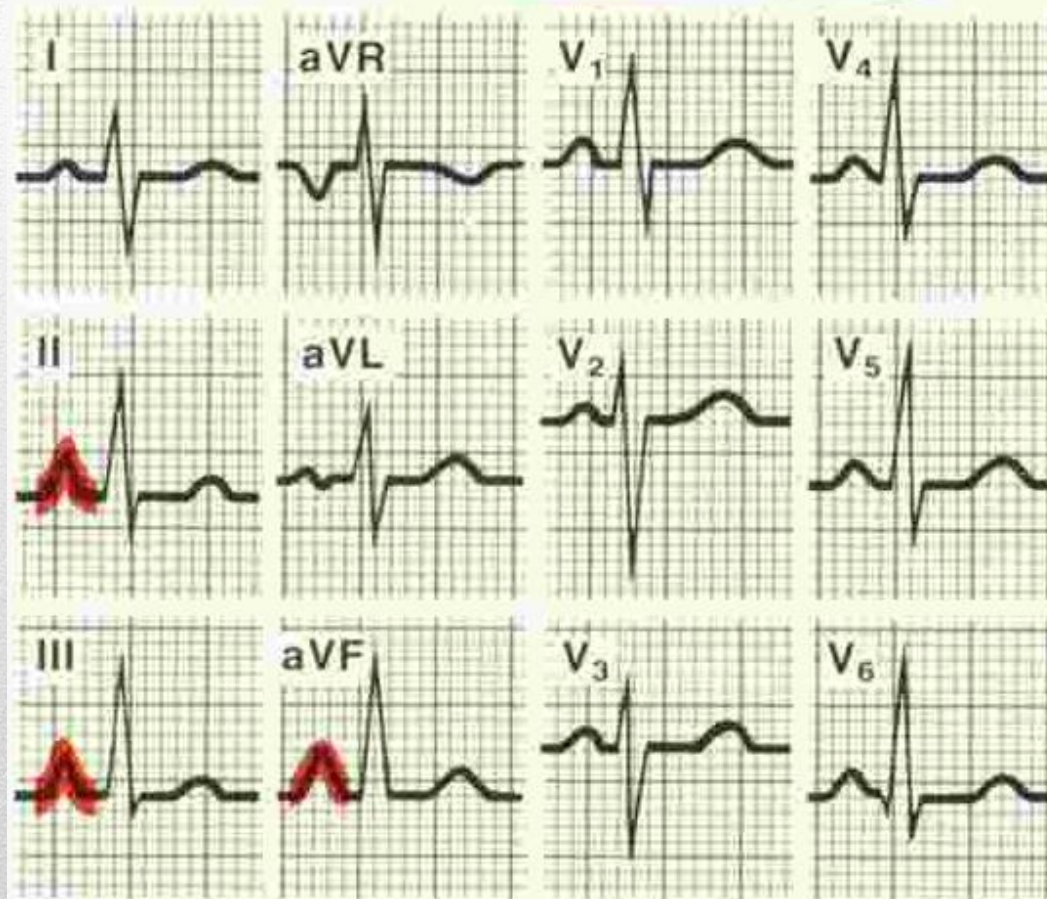


The PR Interval

- Measure from beginning of P wave to onset of QRS. Usually measure in Lead II
- Measure the longest PR interval in the limb leads
- Normal range 0.12-0.20 seconds
- < 0.12 = Accelerated conduction
- > 0.2 = Heart block

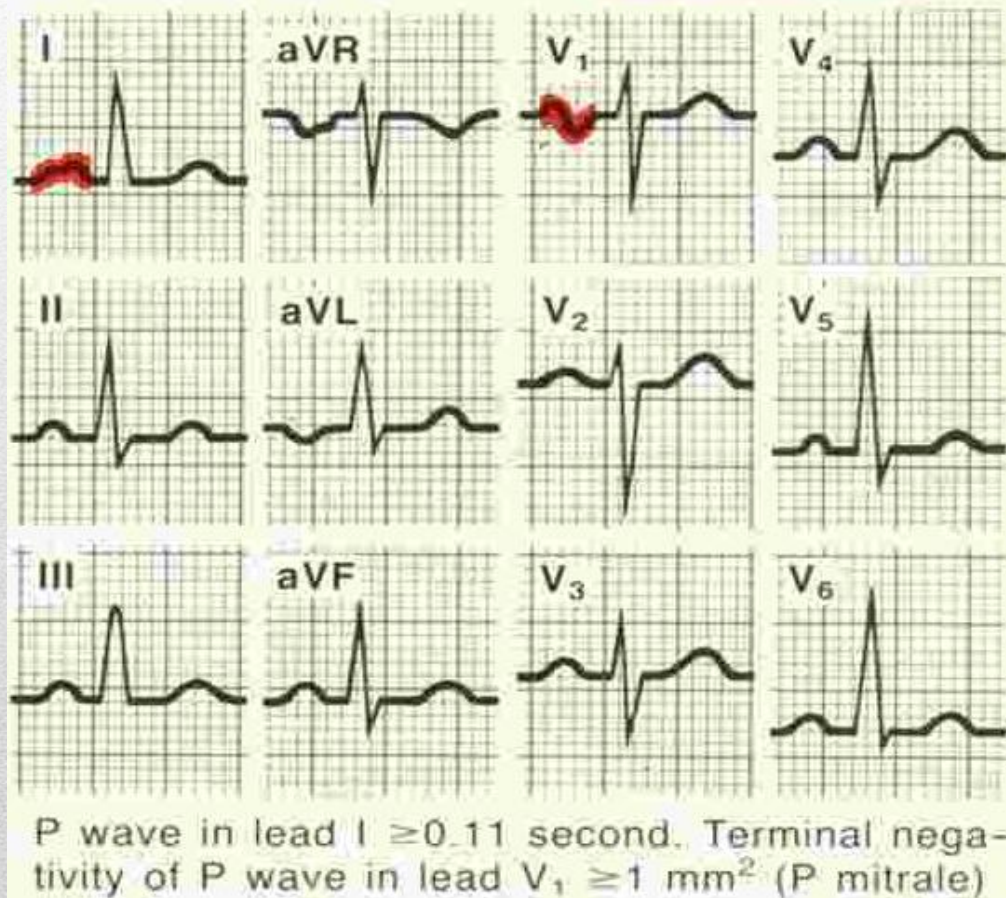


Right Atrial Enlargement

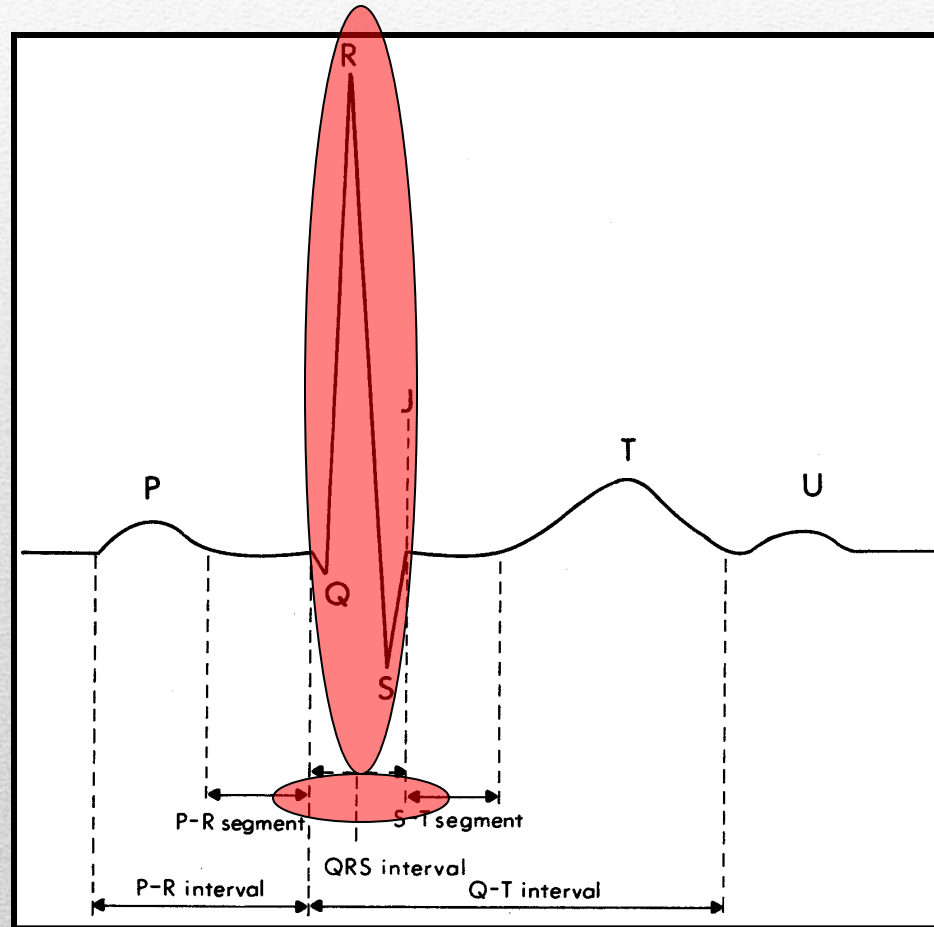


Tall P waves in leads II, III and aVF ≥ 2.5 mm
(P pulmonale)

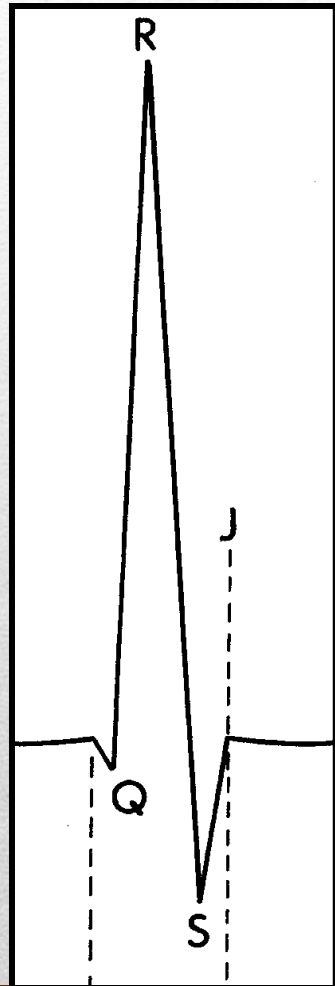
Left Atrial Enlargement



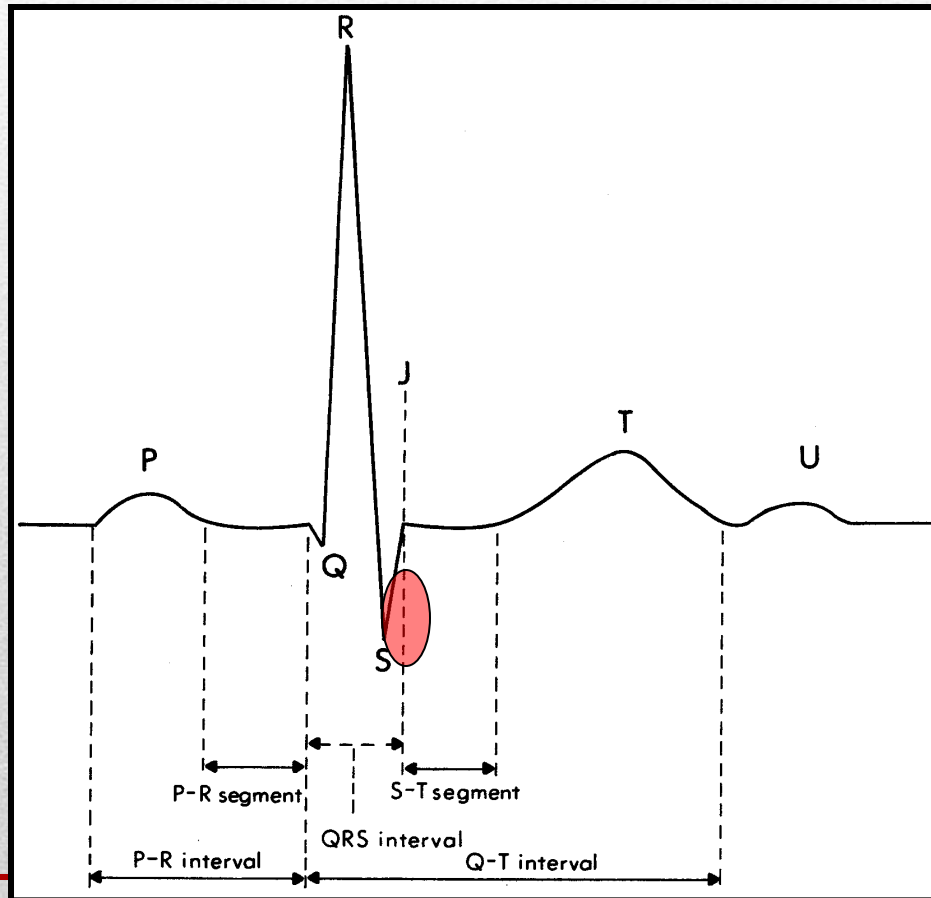
The QRS Complex



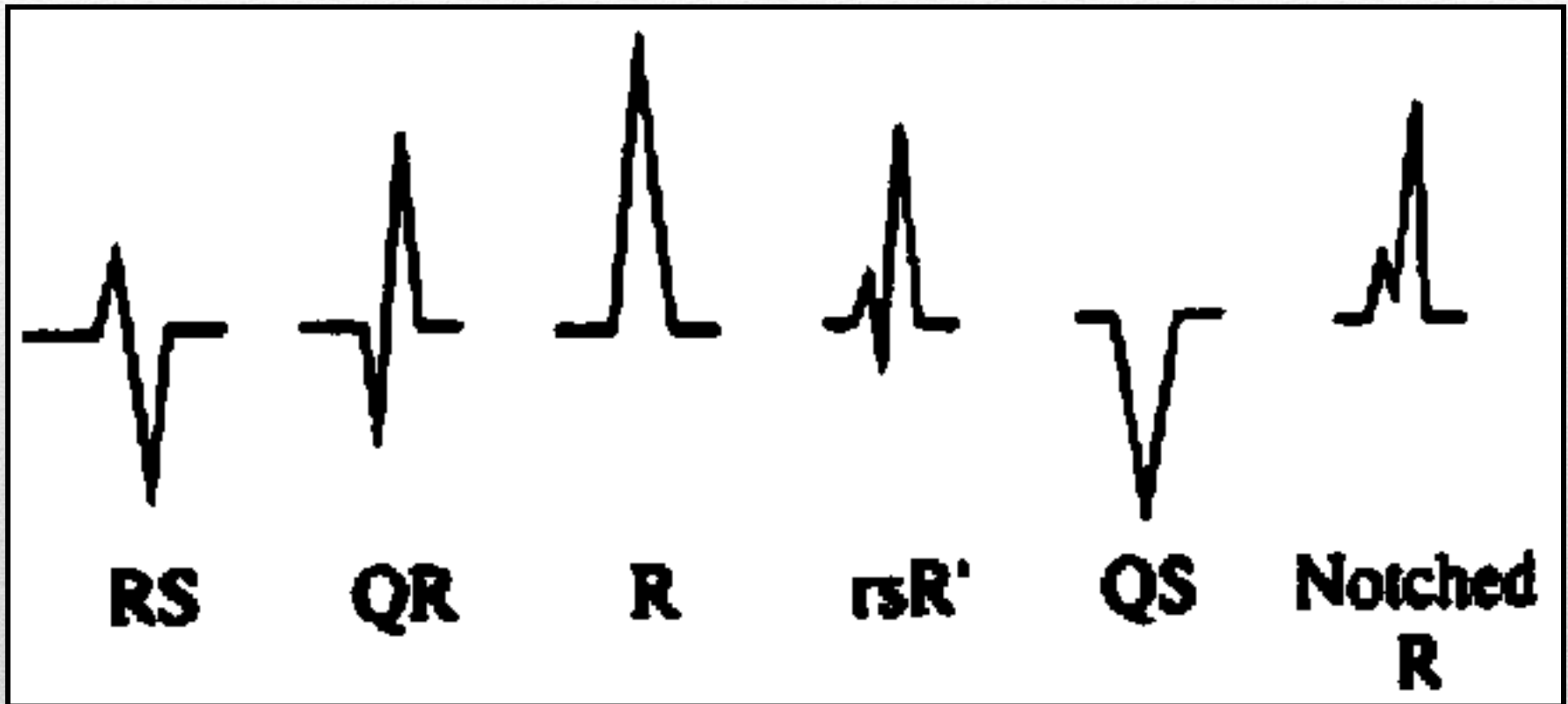
The Q Wave



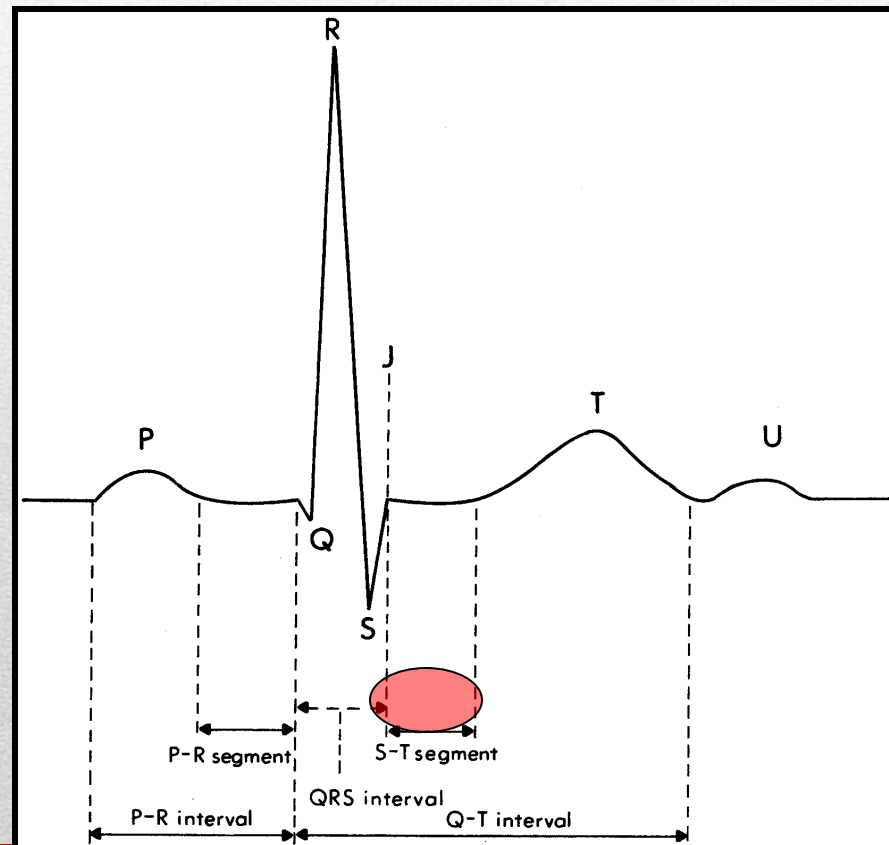
The J - Point



QRS Waveforms

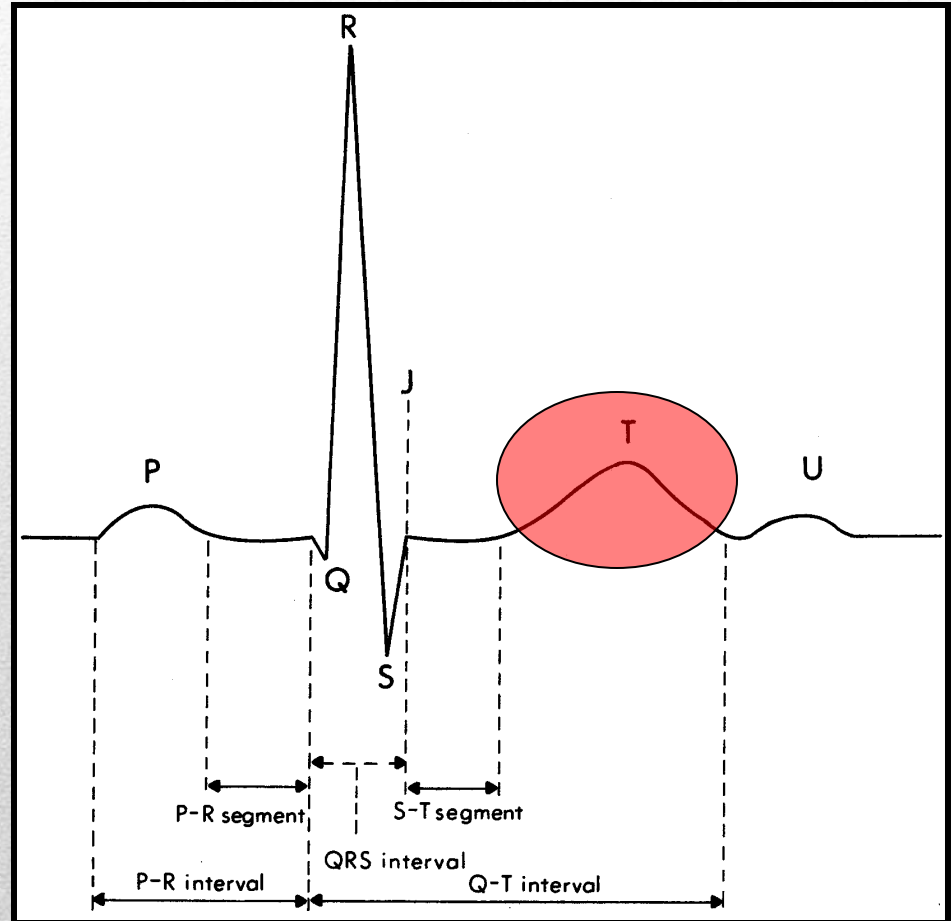


The ST Segment

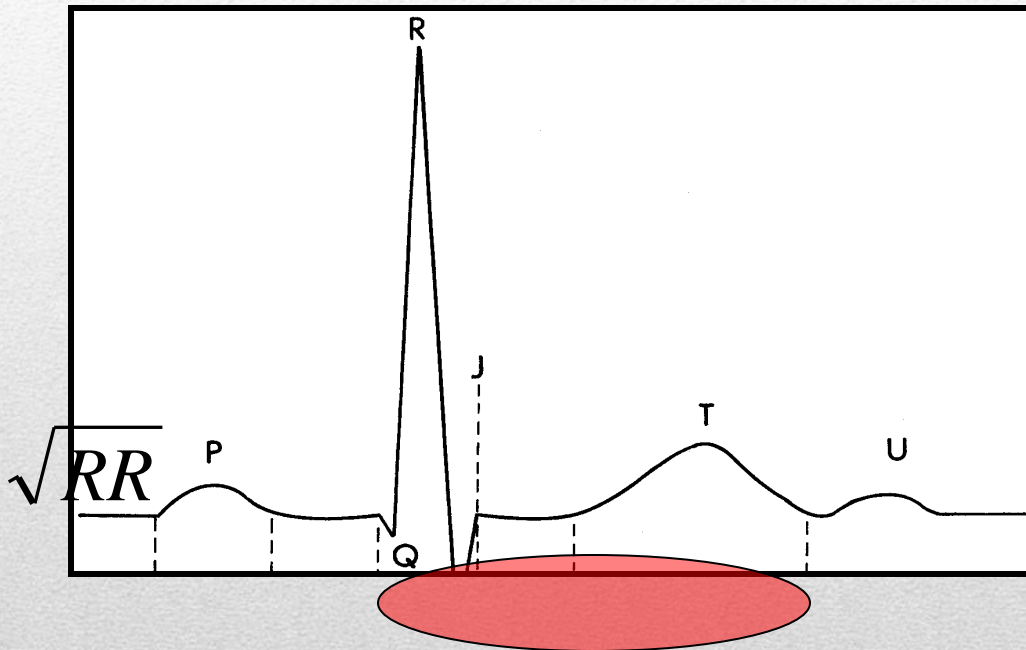


The T Wave

- T waves may be normally inverted in aVR (almost always), III (frequently), and V1 (sometimes).
- T waves are “tall” if their height is:
 - > 50% QRS height
 - > 5mm in limb lead
 - > 10 mm in precordial lead

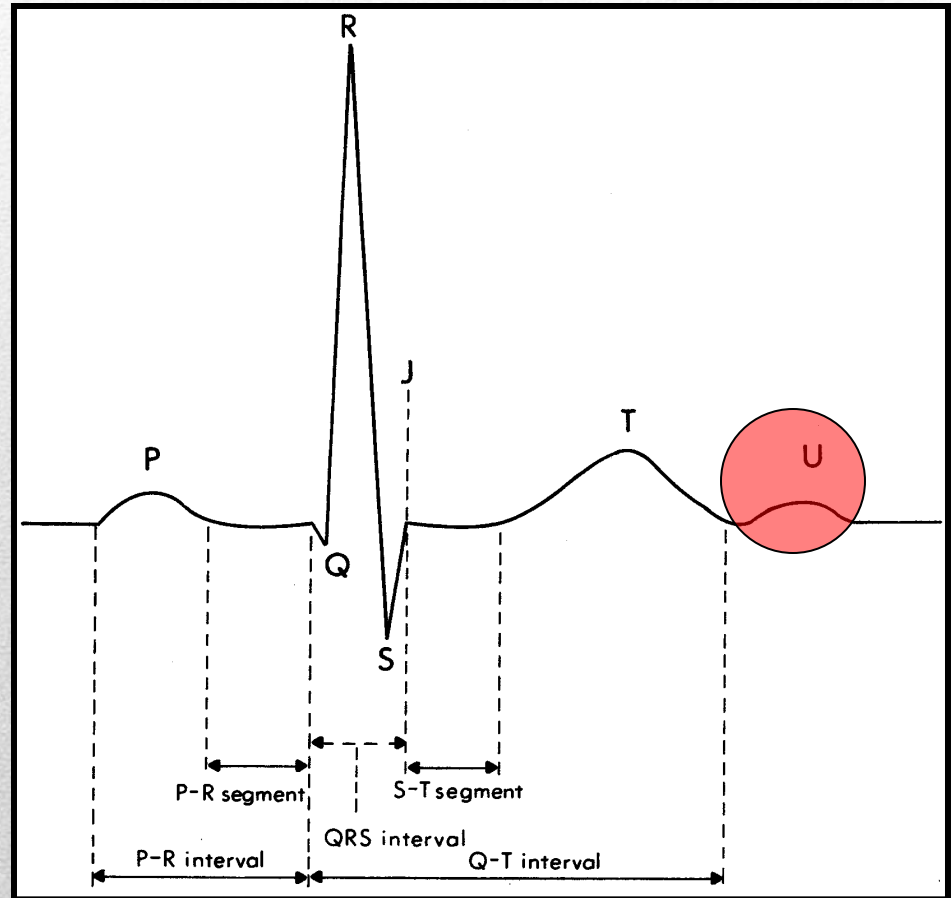


The QT Interval



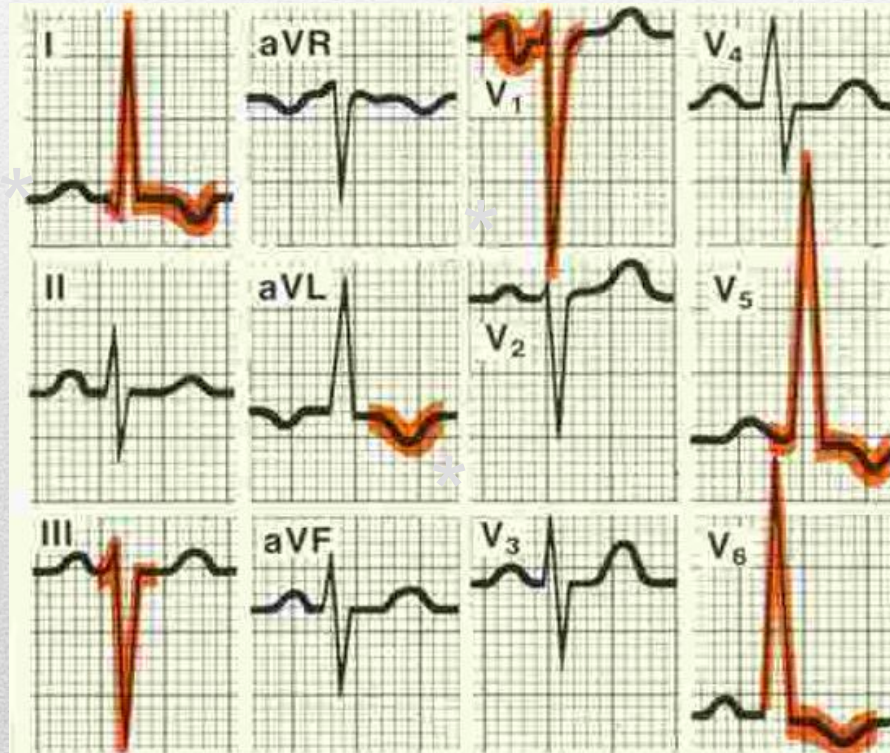
The U Wave

- Causes:
 - Normal
 - Bradycardia
 - CAD
 - Hypertension
 - Hypokalemia
 - Hypercalcemia

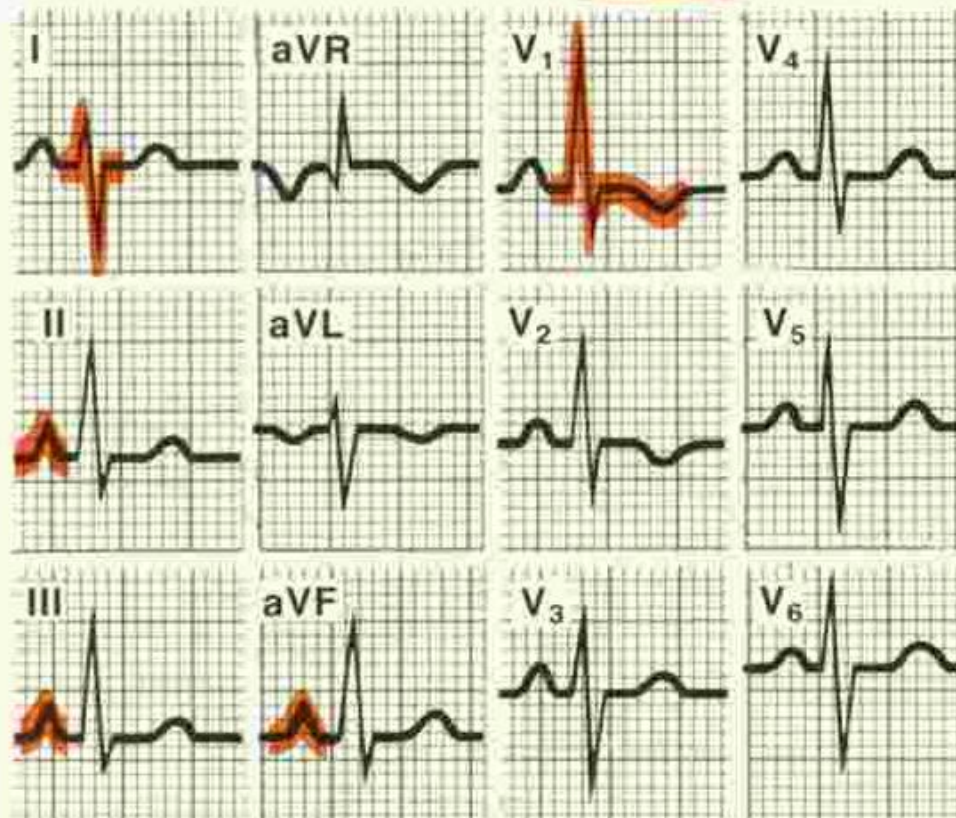


Left Ventricular Hypertrophy

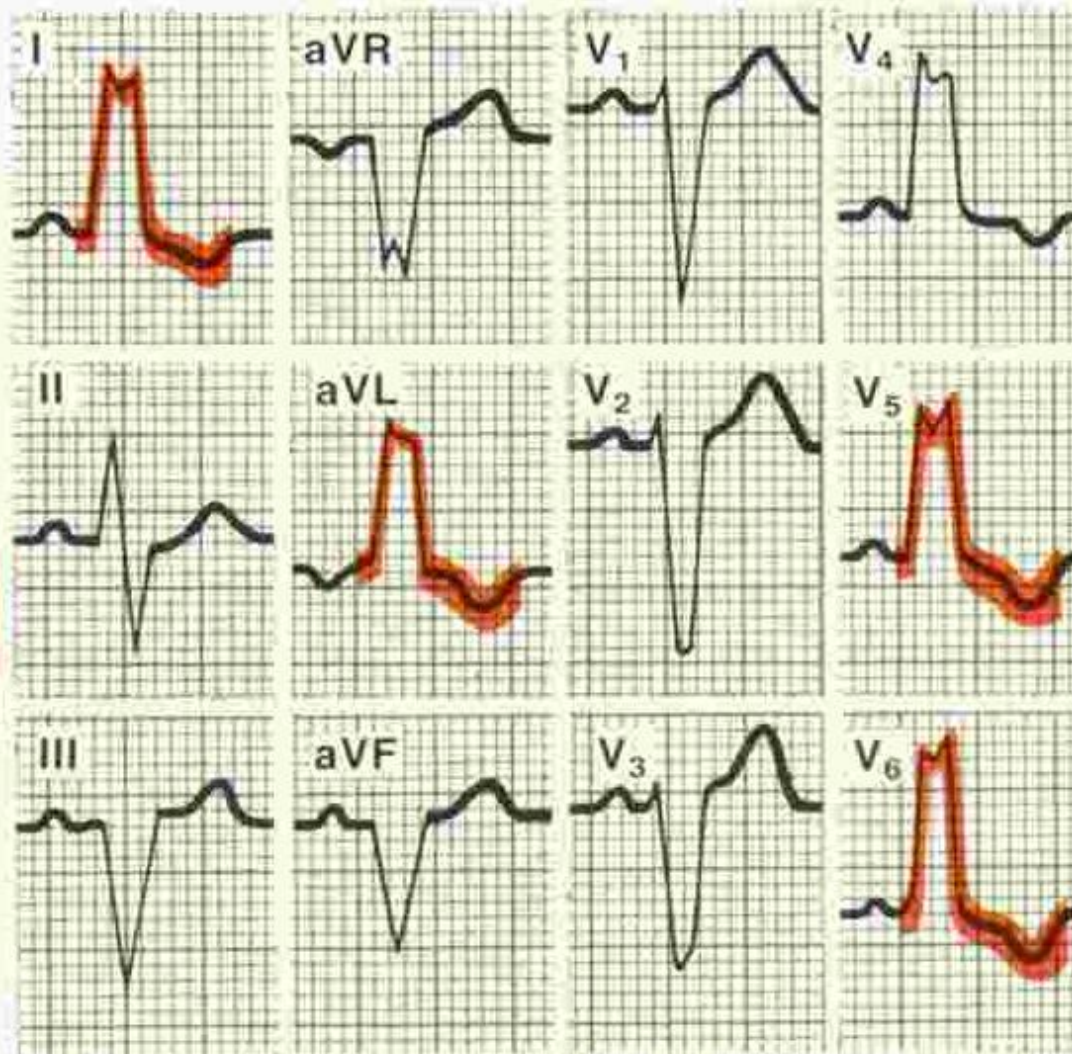
*



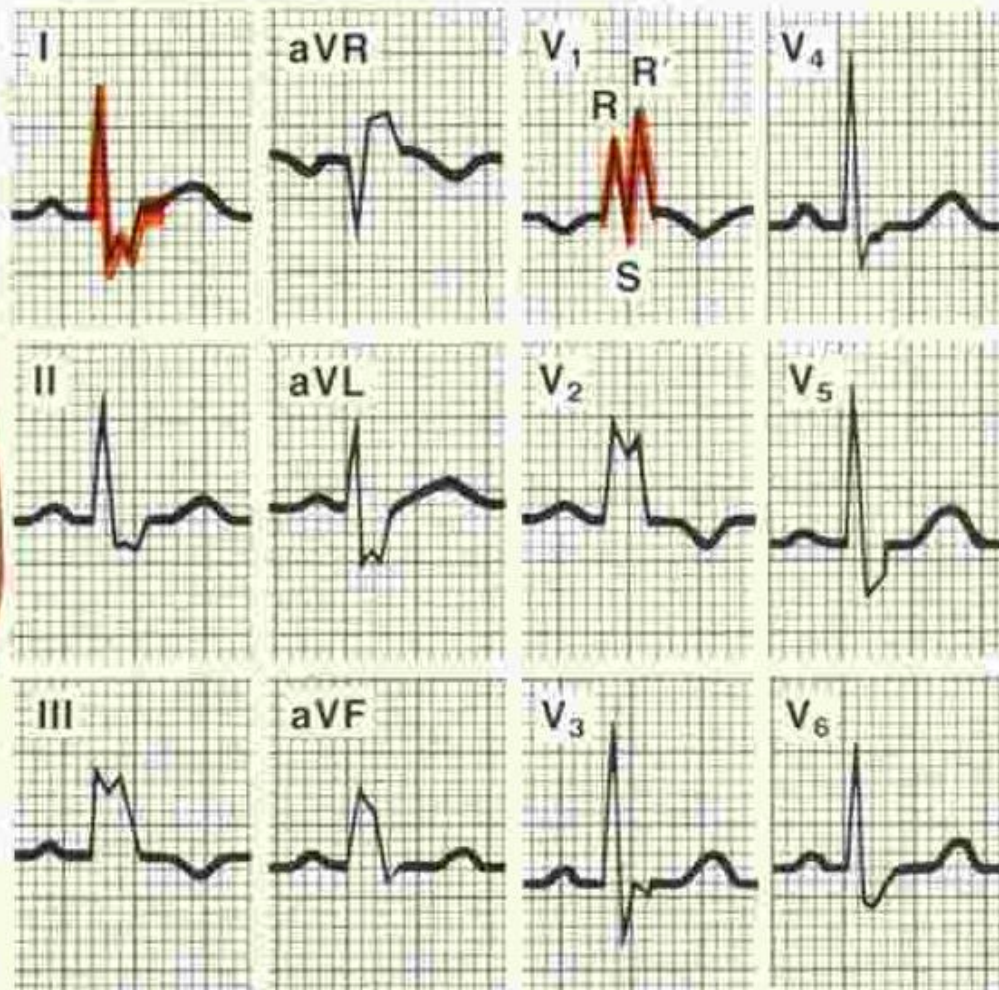
Right Ventricular Hypertrophy



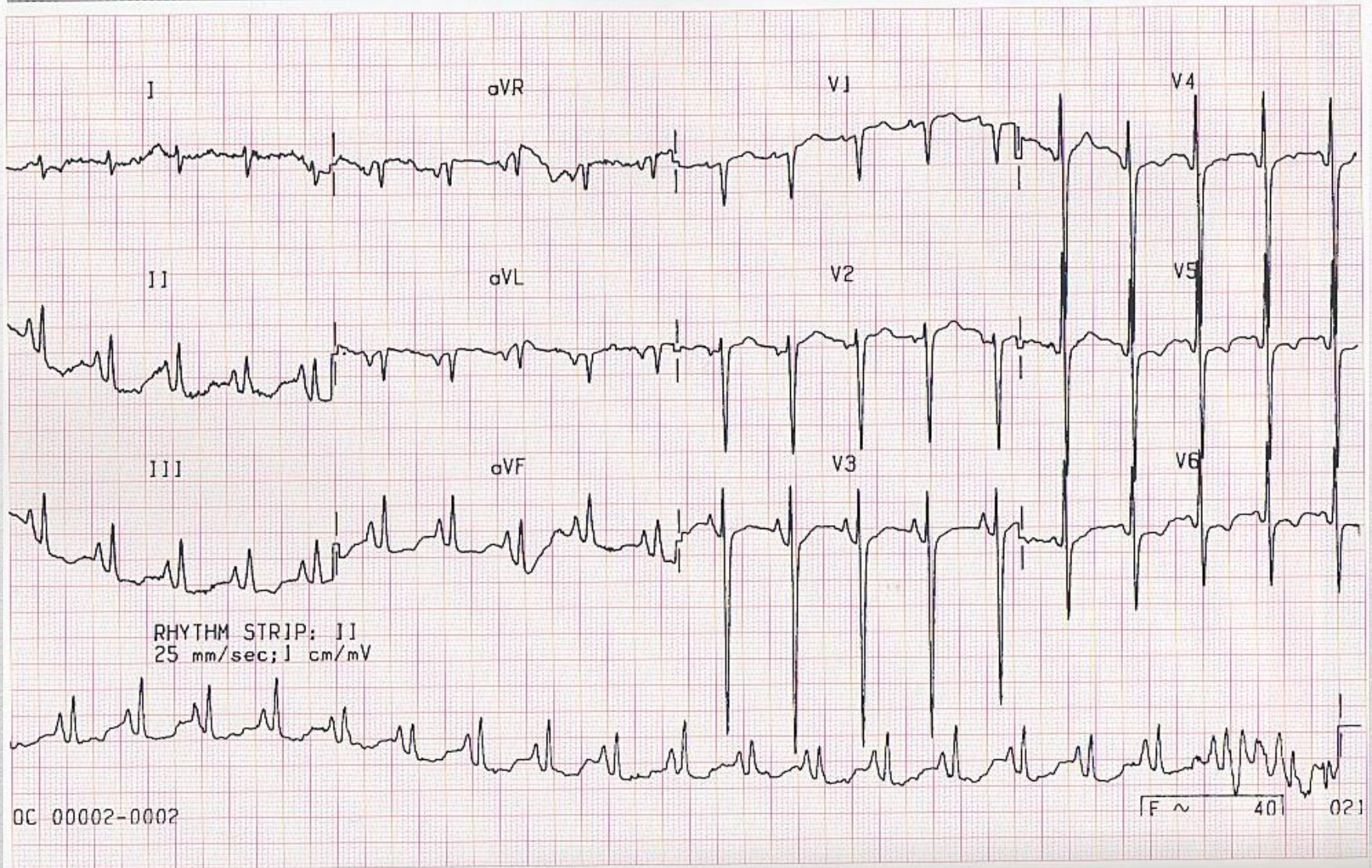
LBBB

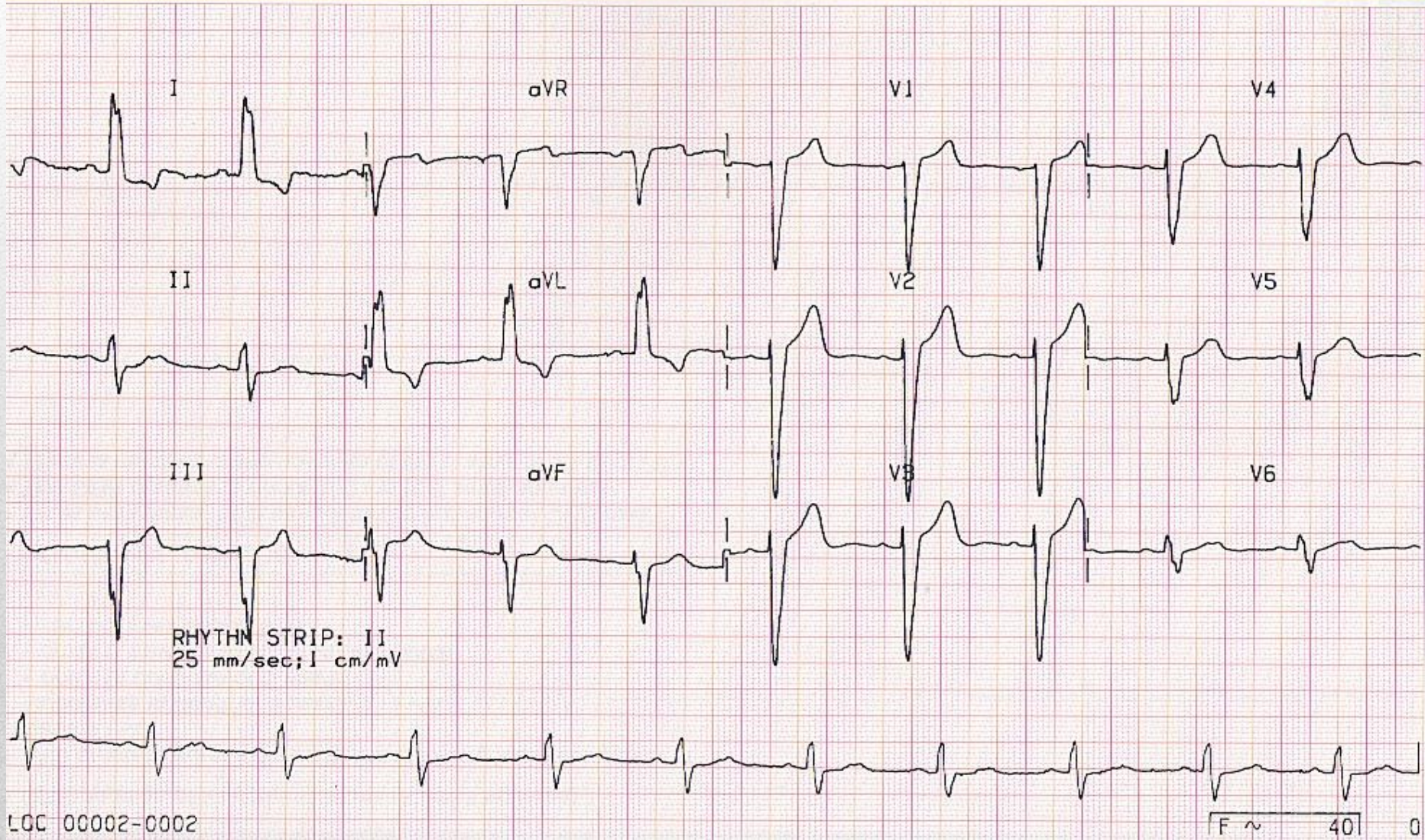


RBBB



Realty check







Heart Block

1st°

- Constant PR prolongation without drop beat.

2nd°

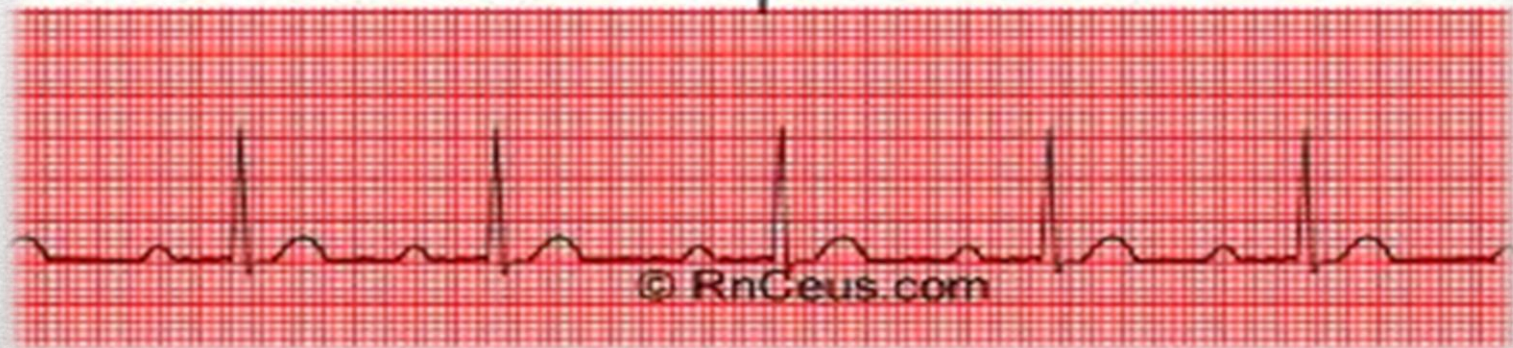
- **Mobitz1:** Progressive PR prolongation + drop beat.
- **Mobitz2:** Constant PR prolongation + drop beat.

3rd°

- Complete dissociation between P and QRS.
-

First Degree Heart Block

FIRST DEGREE A-V HEART BLOCK

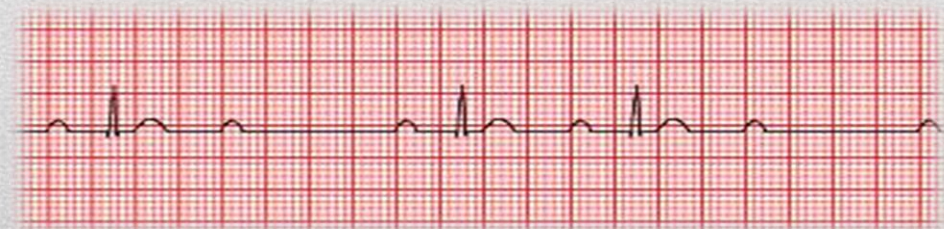


Second Degree Heart Block

- **Mobitz (I):**



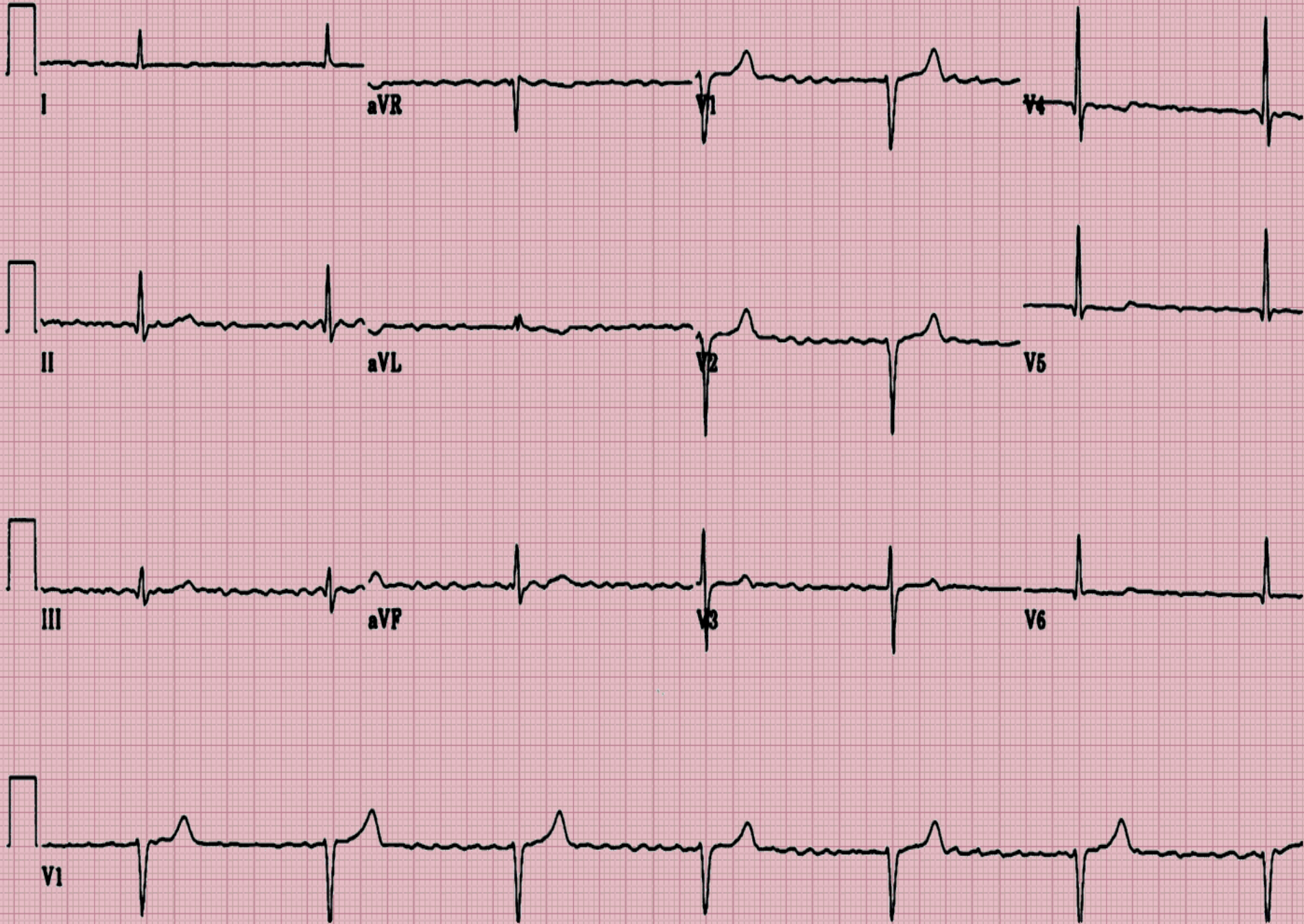
- **Mobitz (II):**



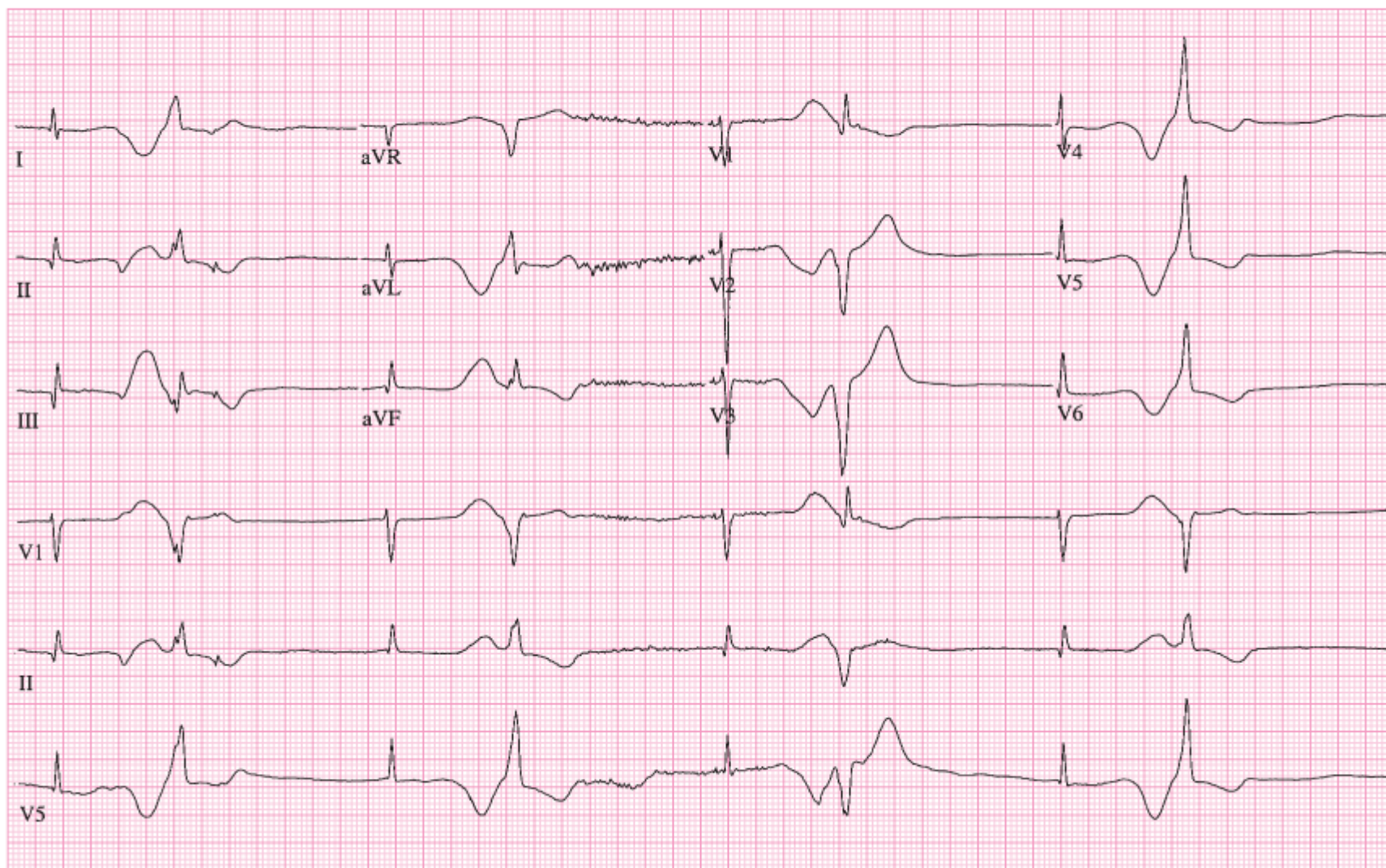
Third Degree Heart Block



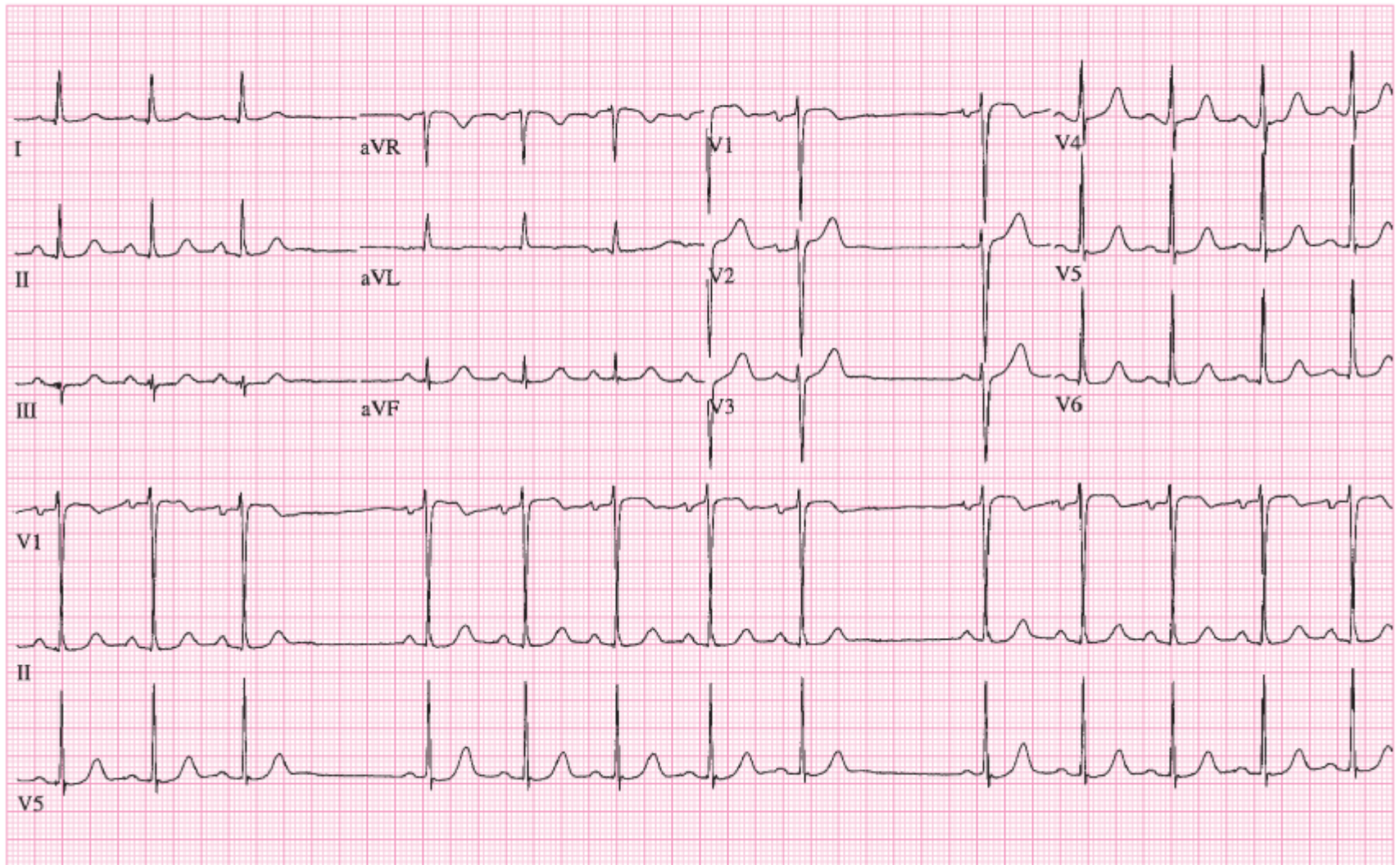
60 yrs old man with dilated CHF presented with nausea&vomating



ECG 20. 62-year-old male with history of treated atrial fibrillation and recent weakness:



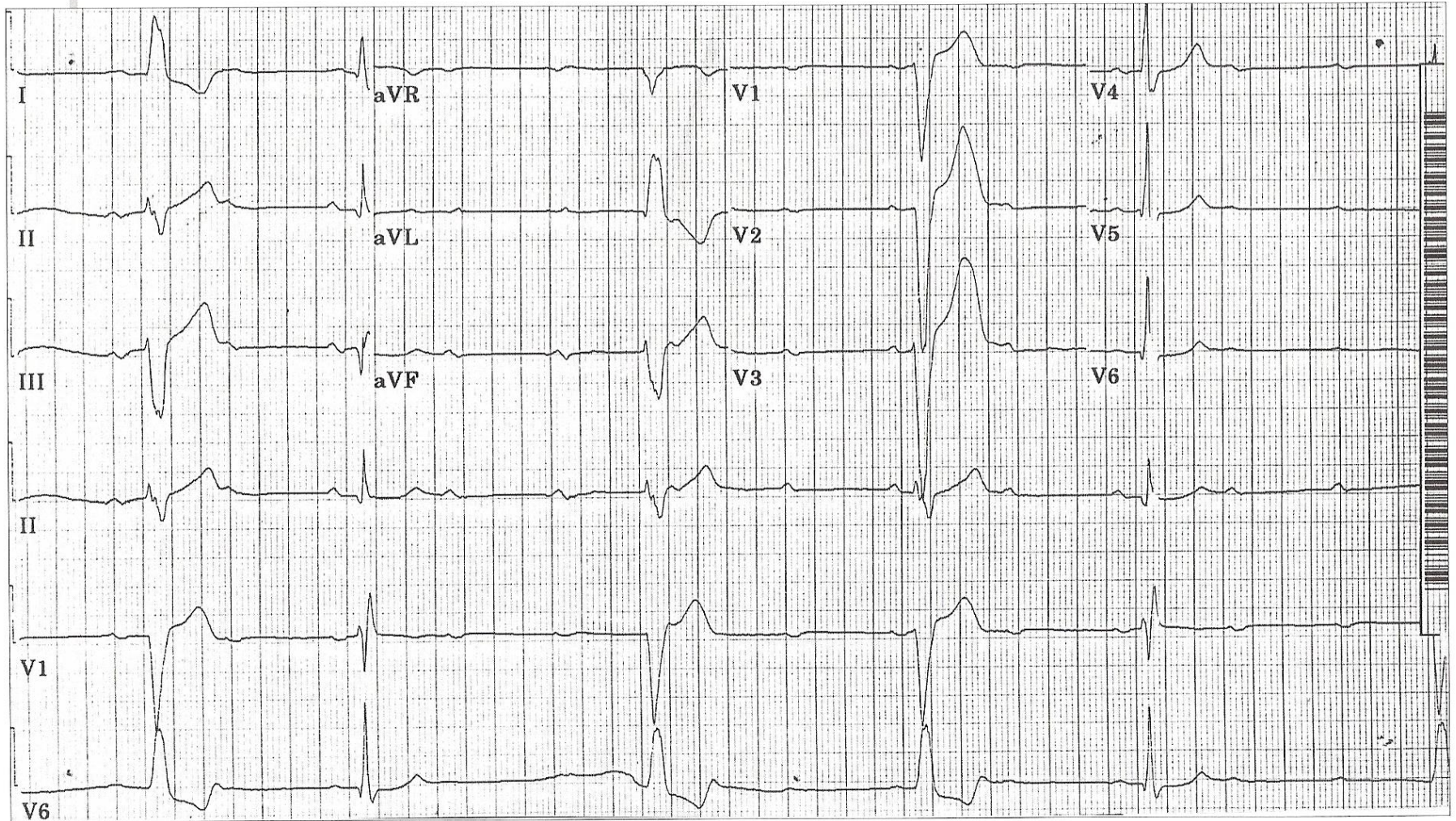
ECG 38. 87-year-old female with dizziness:



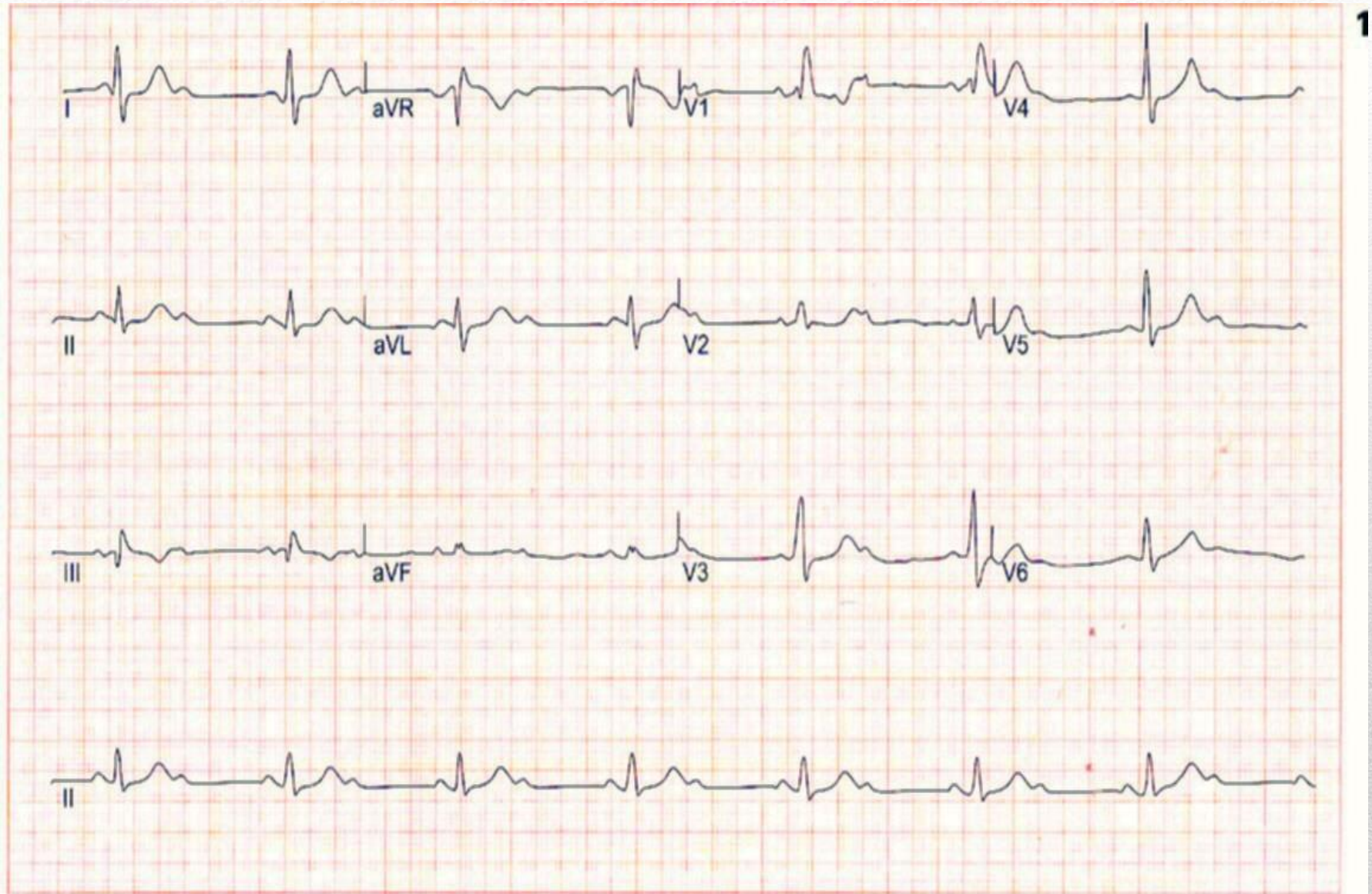
Realty check



ECG V-24

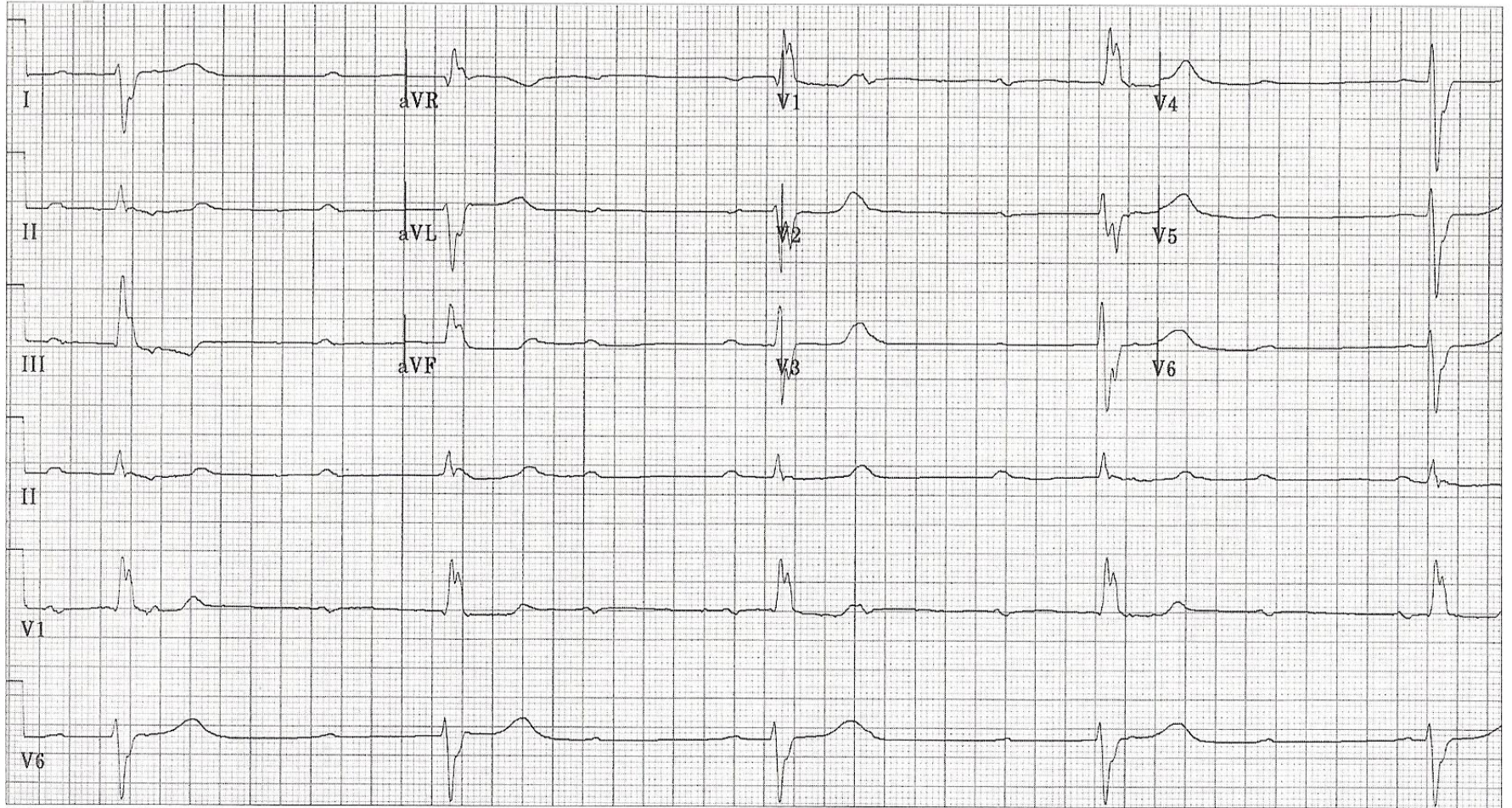


Realty check

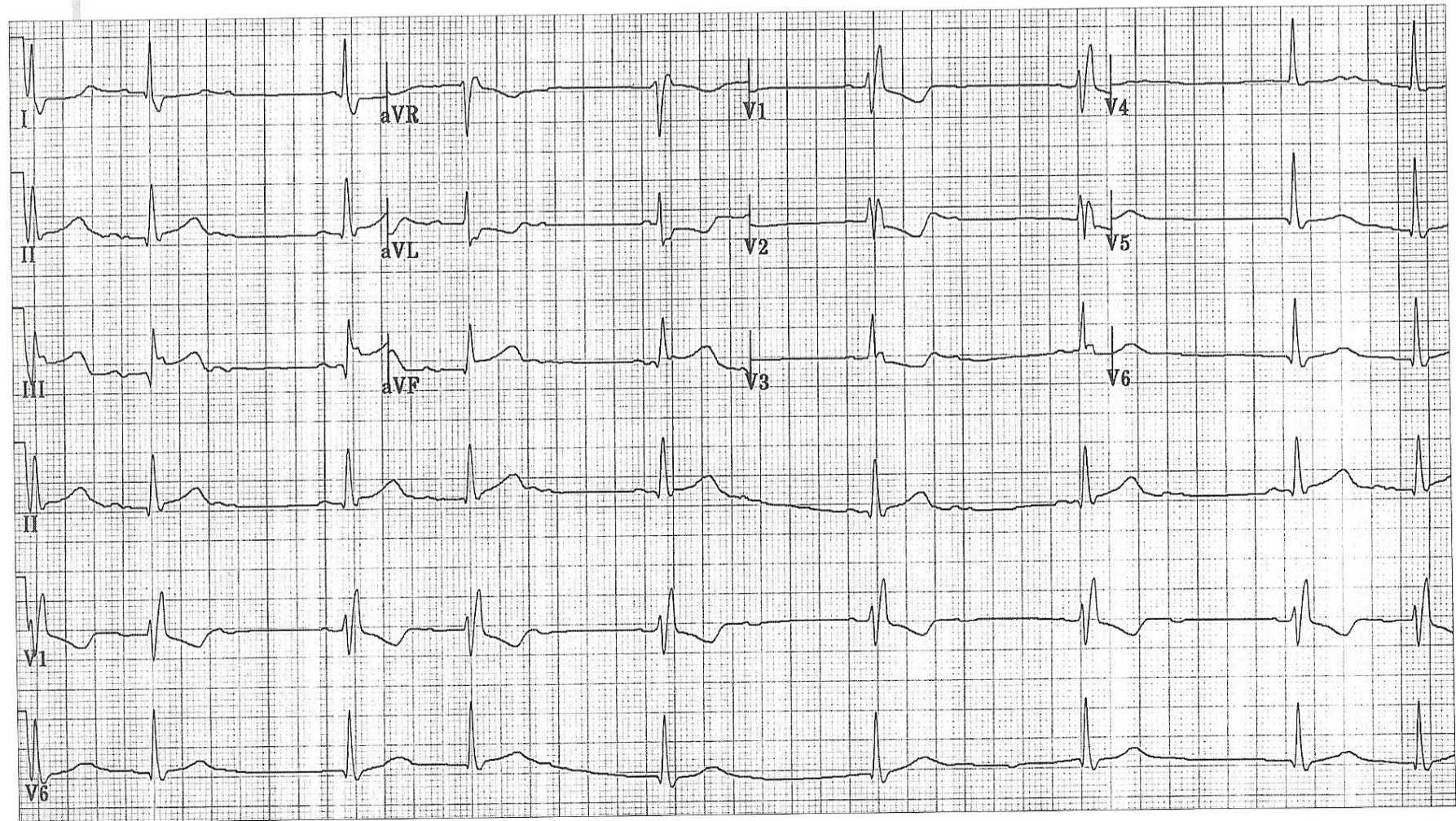


Realty check

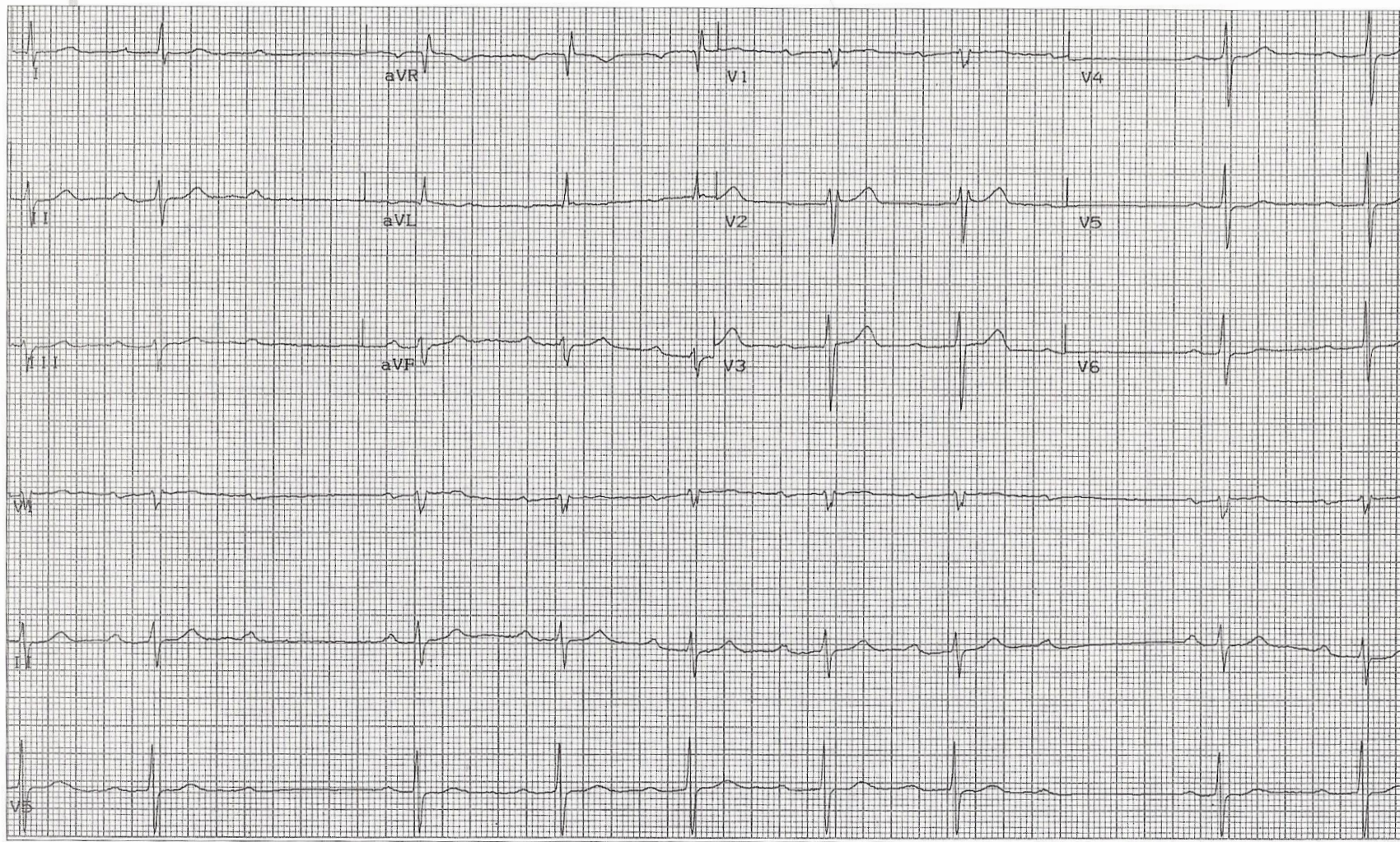
ECG V-1



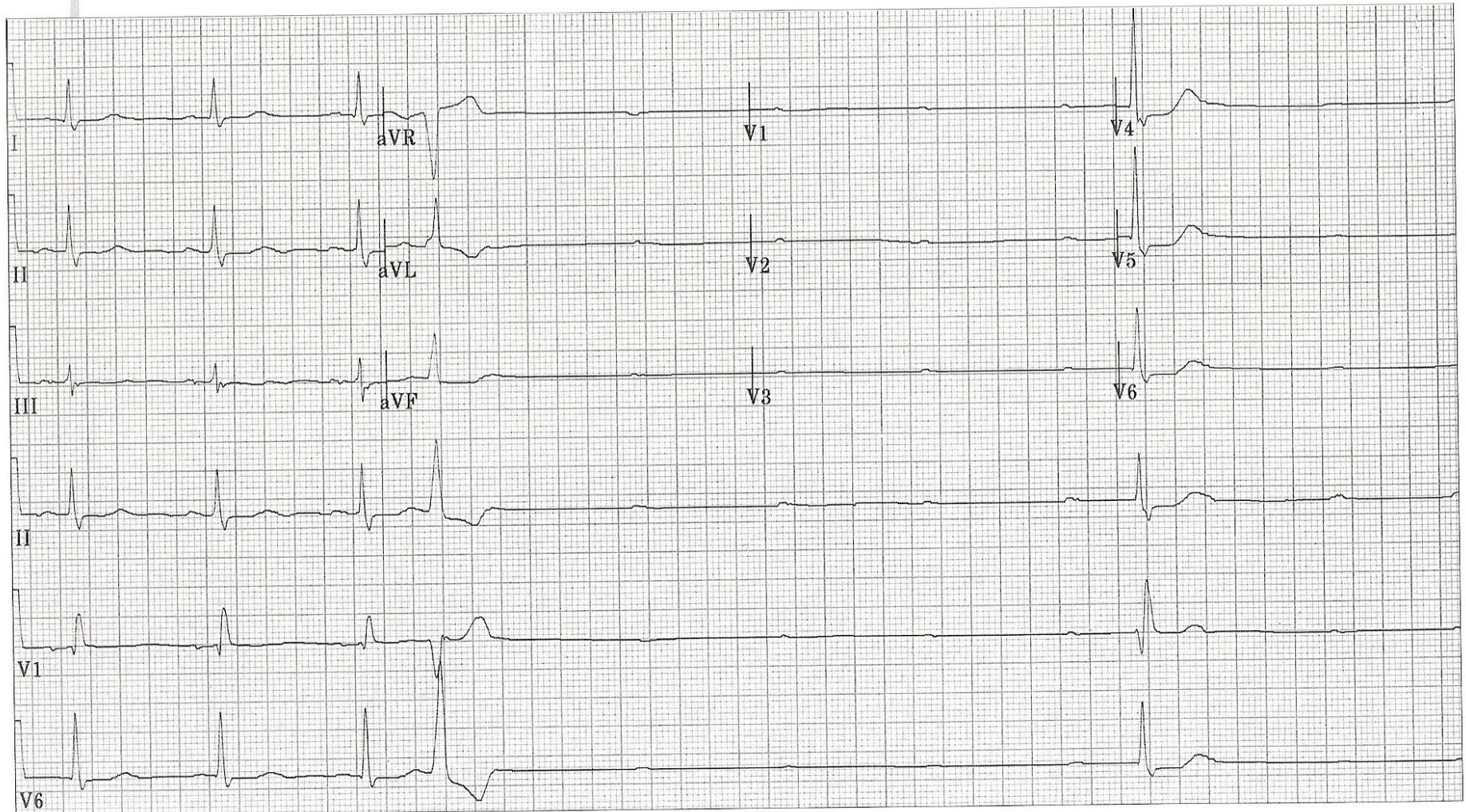
ECG V-6



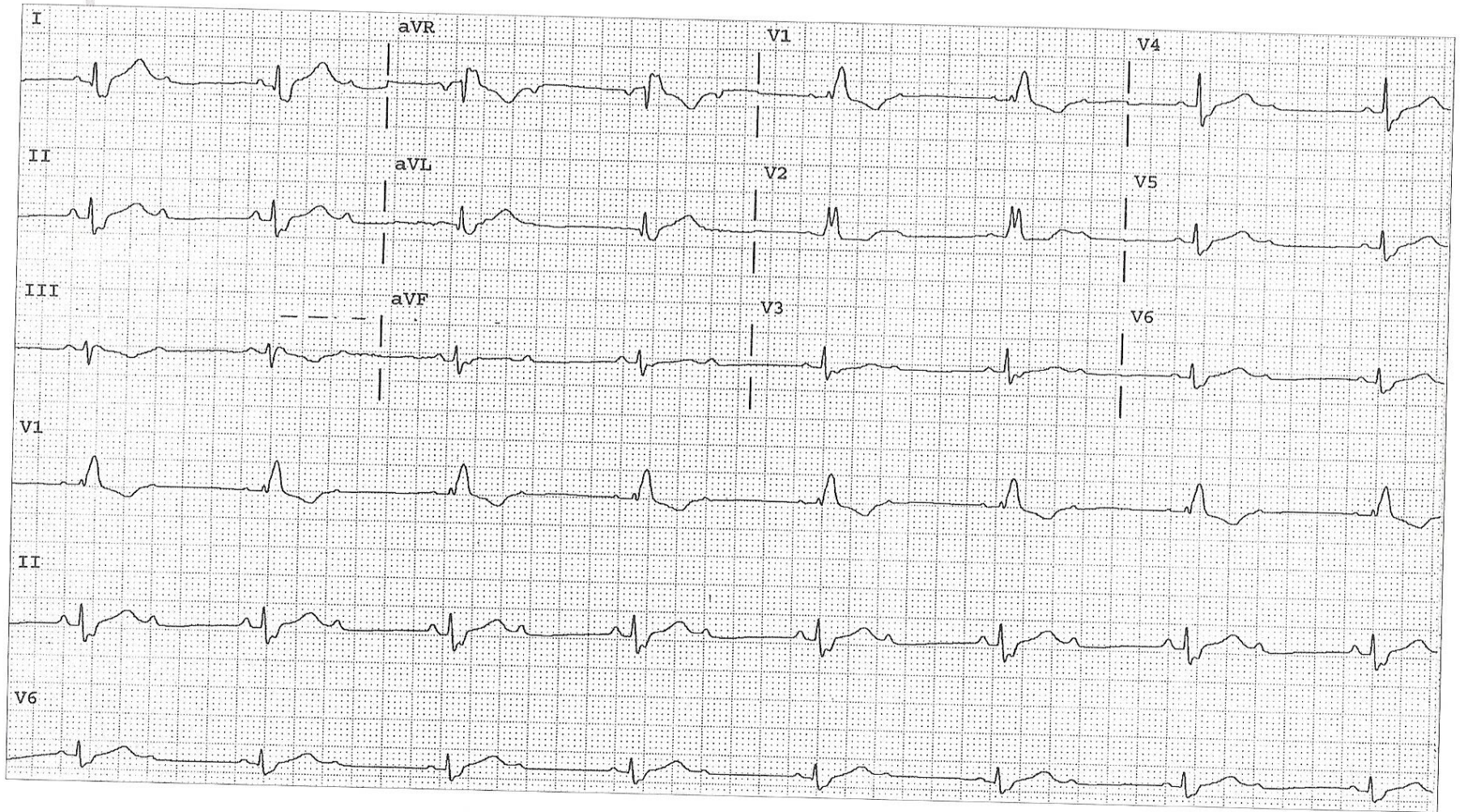
ECG V-15



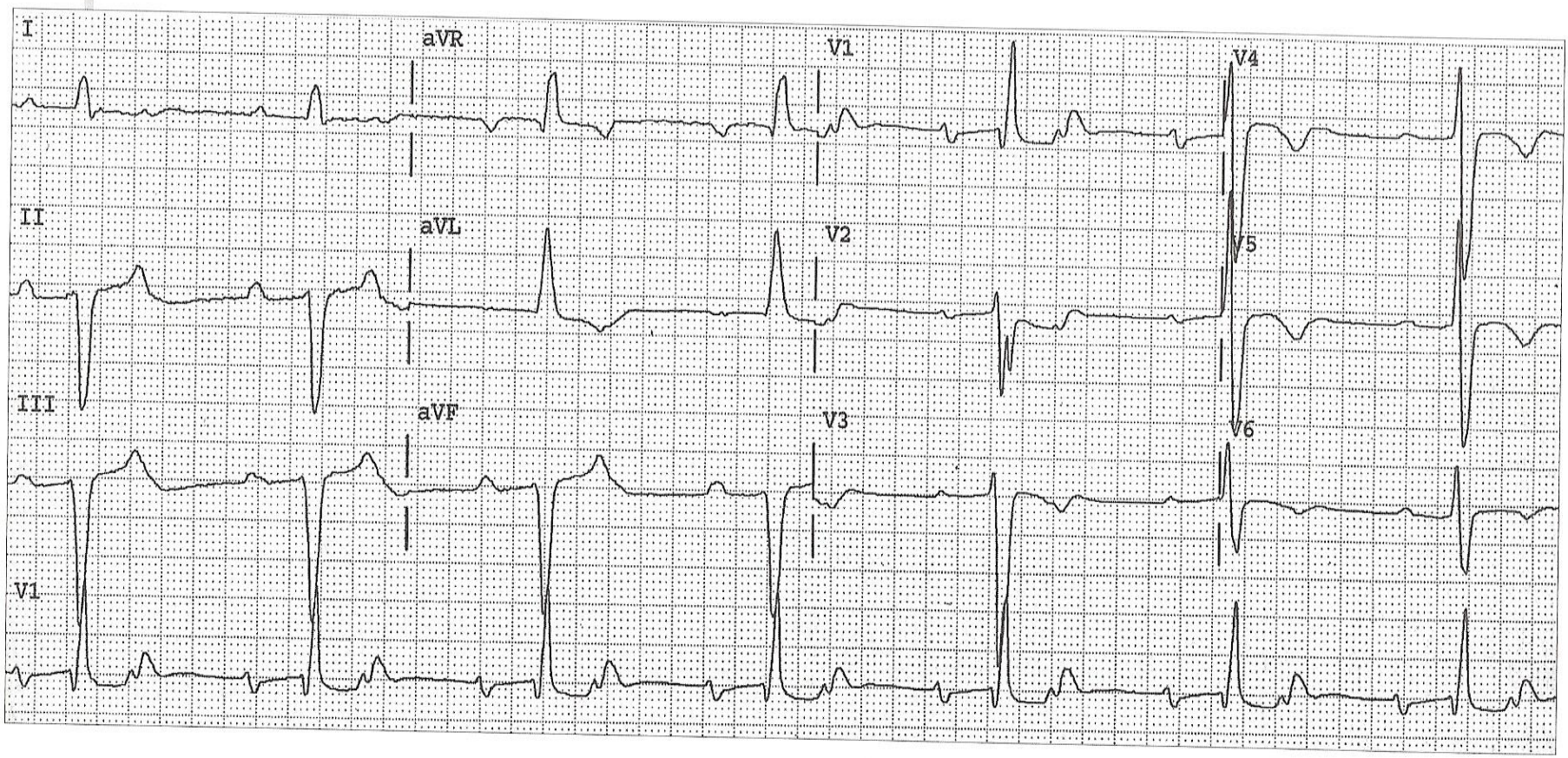
ECG V-22



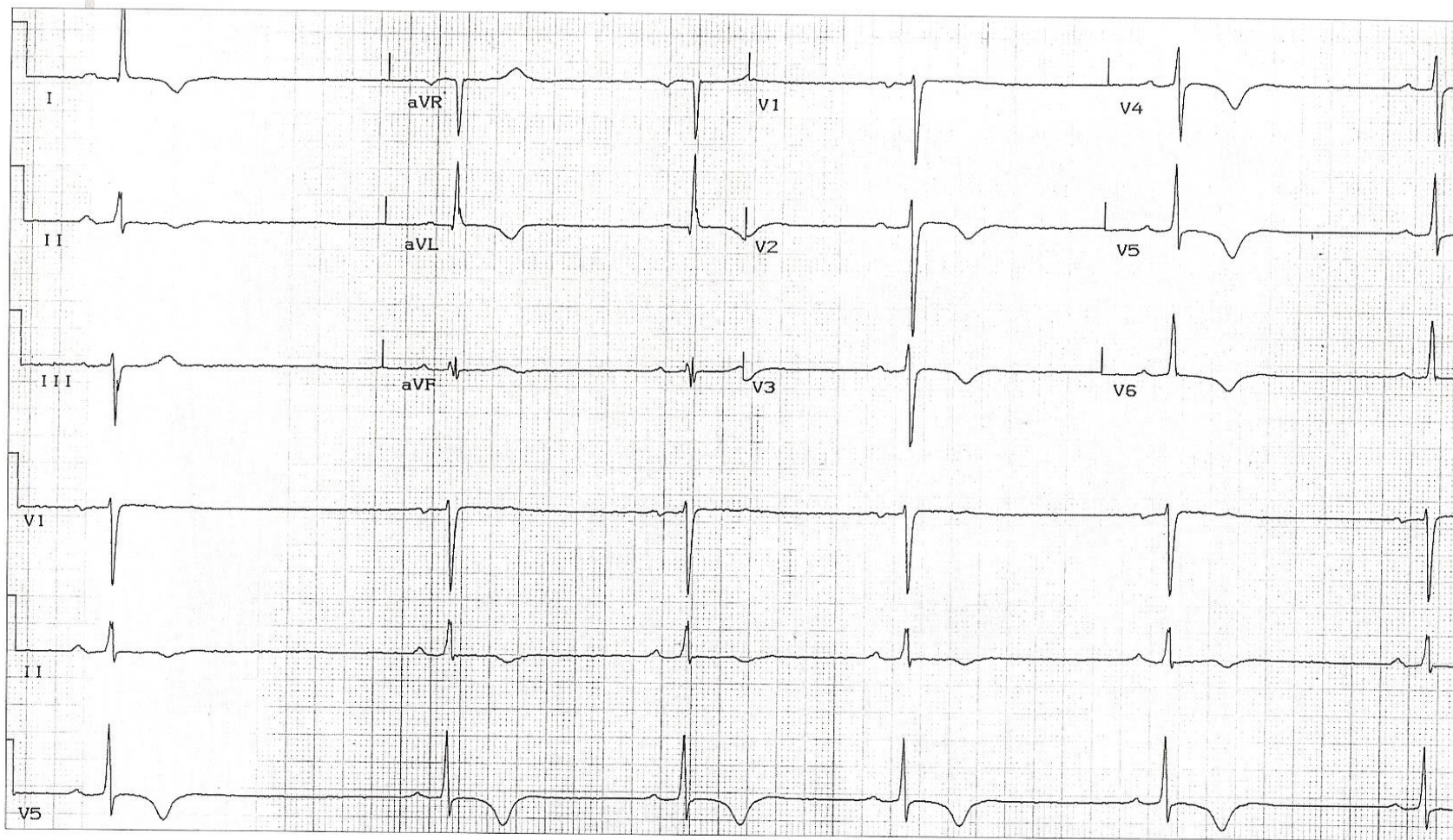
ECG V-26



ECG V-27

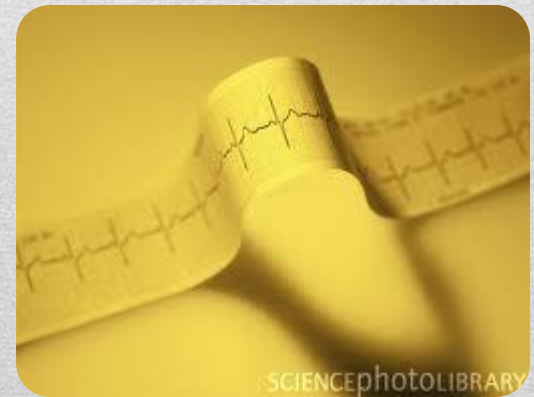


ECG VI-1



Part 2

Dysrhythmia



SCIENCEPHOTOLIBRARY

Outline

- Definition
 - Mechanisms of tachyarrhythmia's
 - Differential diagnosis
 - Common examples
 - Approach to diagnosis
-

What are NCT?

- Fast cardiac rhythms (>100 bpm)
 - QRS duration ≤ 120 ms (less than 2.5 small squares).
 - Originate from the atria or AV junction
 - If aberrant conduction is present ,the QRS will be long even though the arrhythmia originates above the AV junction.
-

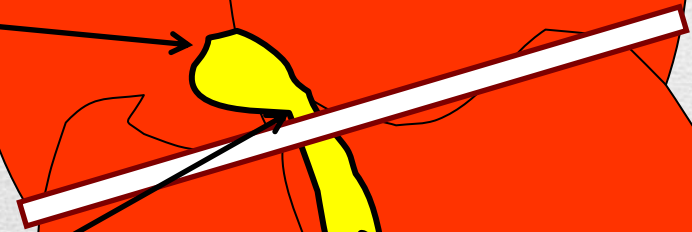


SA



SVT

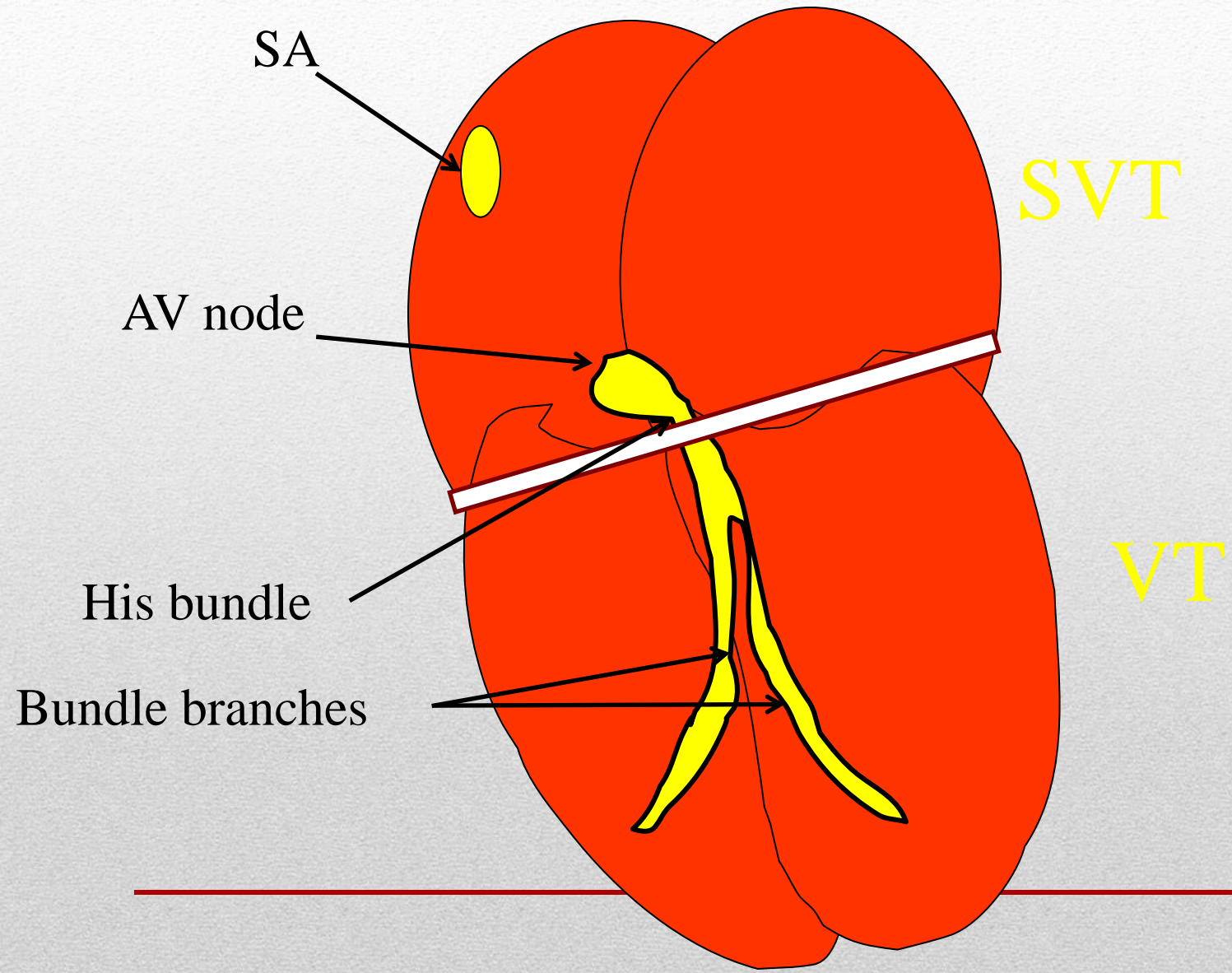
AV node



His bundle

VT

Bundle branches





MECHANISMS OF TACHYARRHYTHMIA'S

Mechanism

1) Accelerated automaticity

Automatic impulse generation from unusual site or overtakes sinus node.

2) Triggered activity

Secondary depolarization during or after repolarization

3) Reentry

Automatic foci

SA

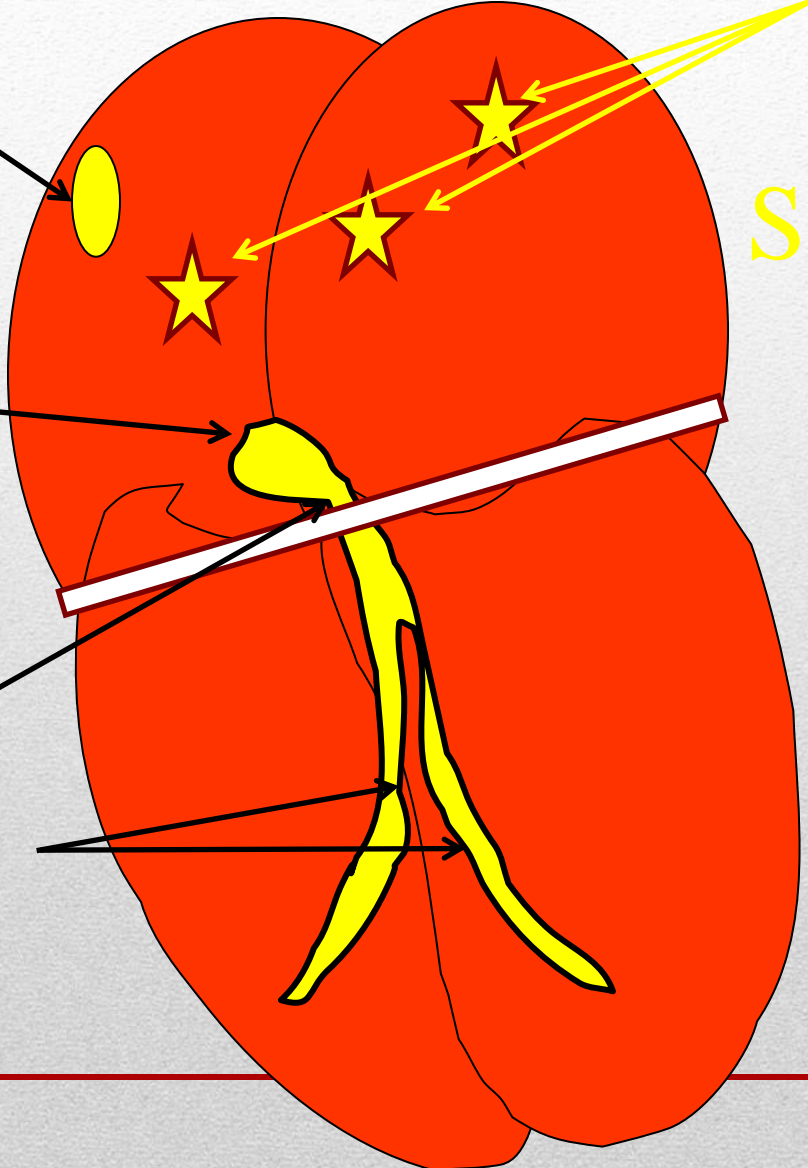
SVT

VT

AV node

His bundle

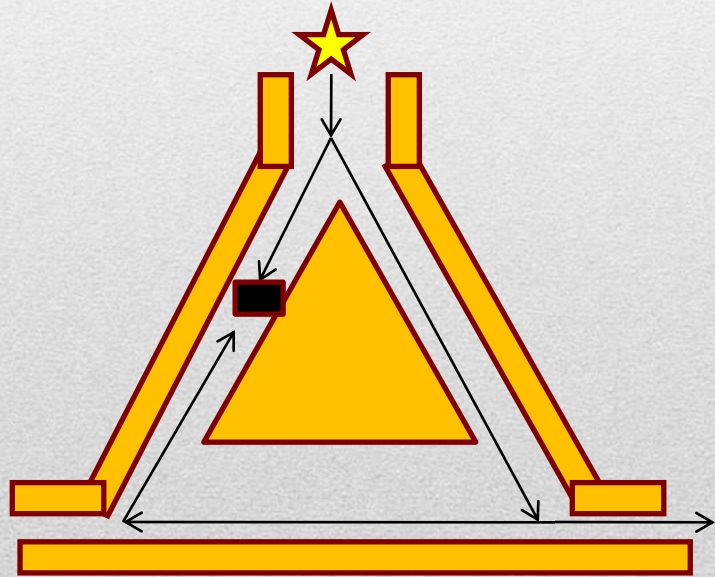
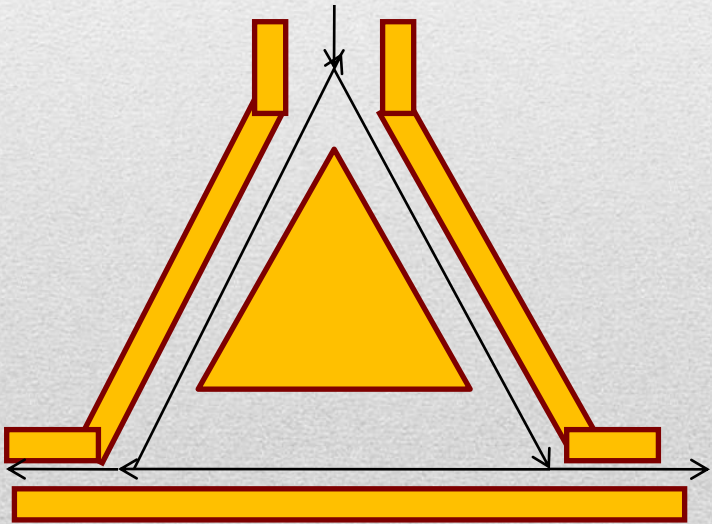
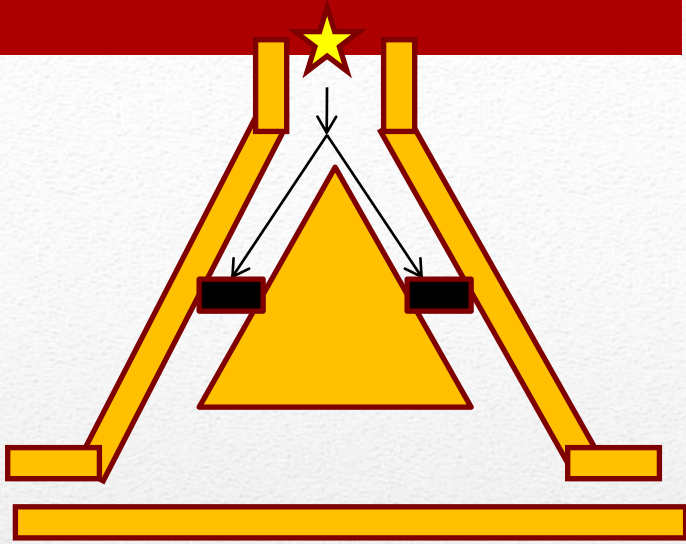
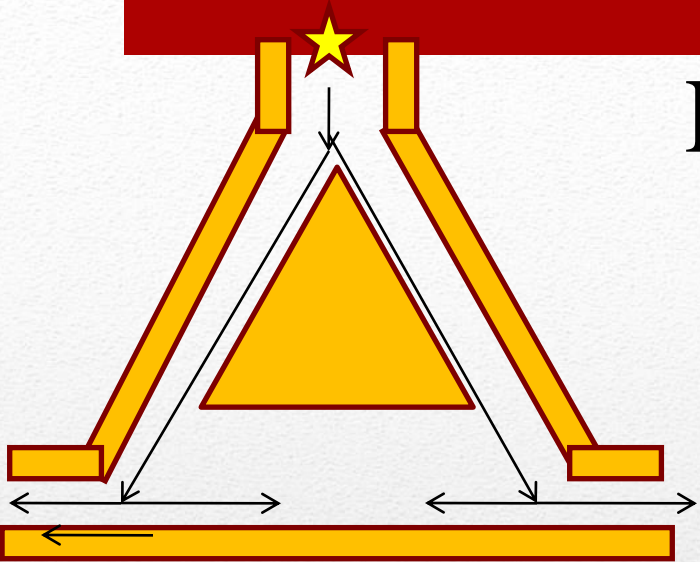
Bundle branches



Reentry

- Most common mechanism (around 90%)
 - 3 prerequisites to develop reentry:
 1. Available circuit
 2. Difference in the refractory periods of the 2 limbs in the circuit
 3. Slow enough conduction somewhere in the circuit
-

Reentry



reentry

Mechanisms...why know them?

- Each mechanism may have unique features on the surface ECG.
Example: - *Automaticity arrhythmias start & stop gradually.*
 - *Reentry starts & stops abruptly*
 - Impacts management
-

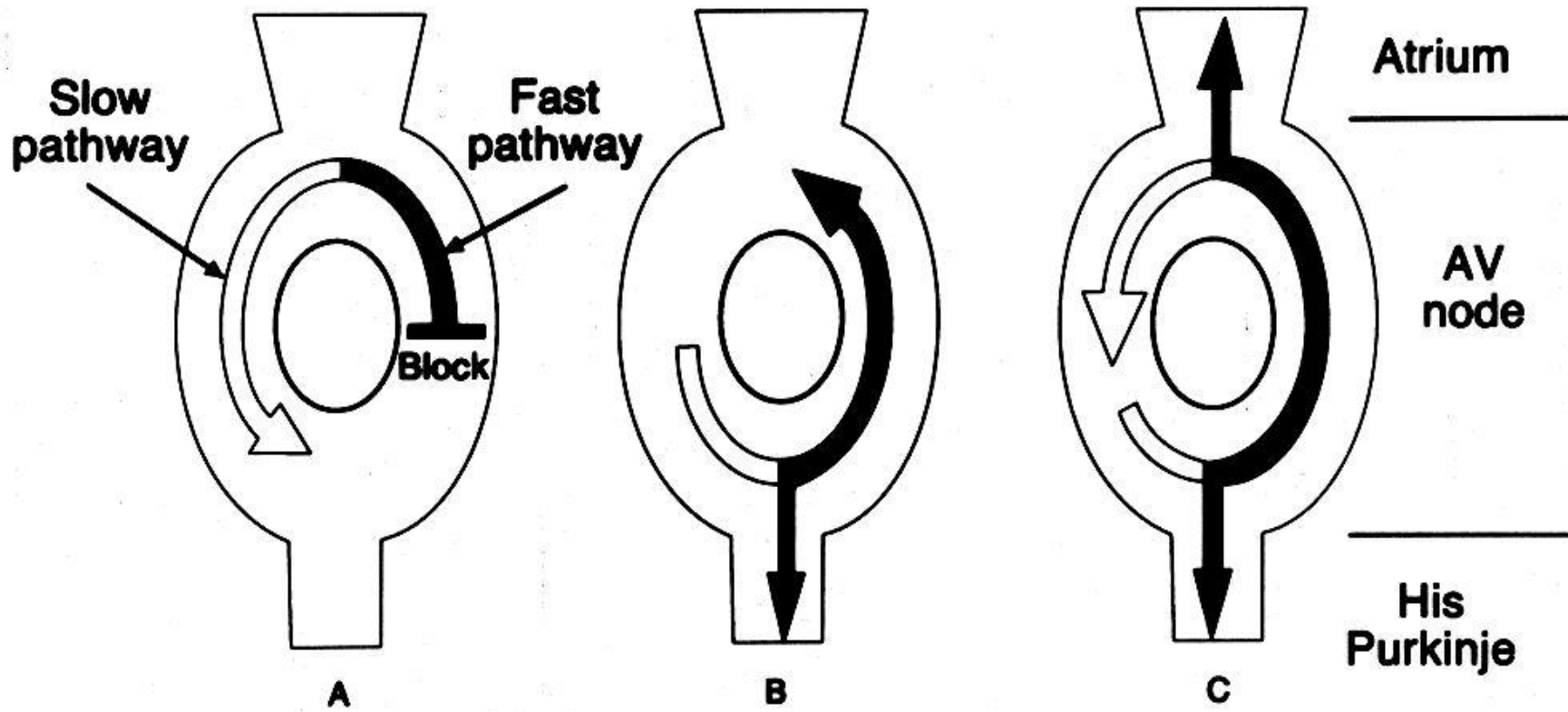
DIFFERENTIAL DIAGNOSIS

- Sinus tachycardia
 - SA nodal reentry tachycardia
 - Atrial tachycardia /PAT/MAT
 - Atrial flutter
 - Atrial fibrillation
 - AVNRT (AV nodal reentrant tachycardia)
 - AVRT (AV reentrant tachycardia)
 - Junctional tachycardia
-

AV NODAL REENTRANT TACHYCARDIA (AVNRT)

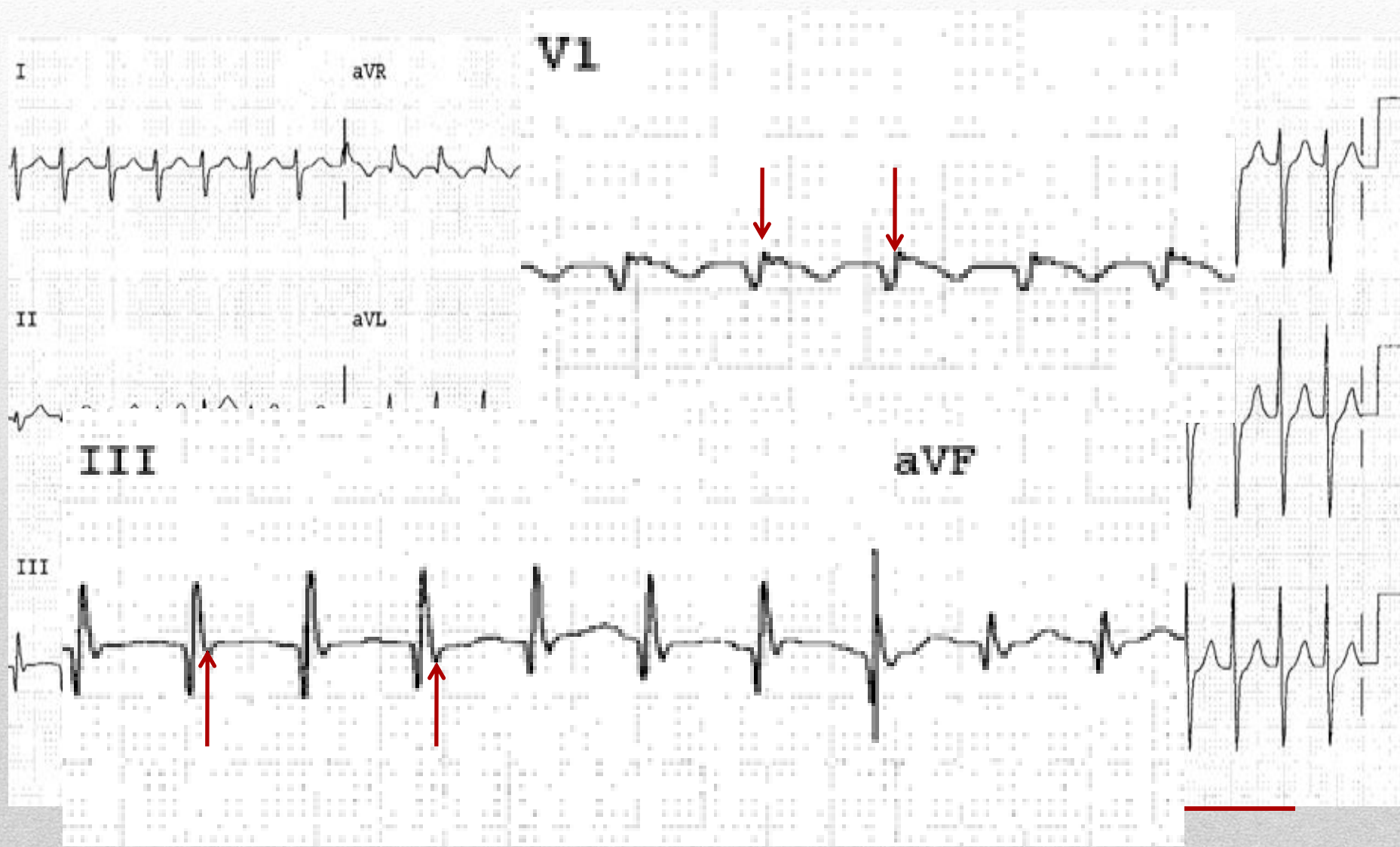
AVNRT

- Most common SVT (AF/Aflutter excluded).
 - Initiated by a PAC
 - Reentry at the level of the AV node
 - P waves are seen either immediately following the QRS or buried within it.
 - Ventricular rate can reach 280 bpm
-



AVNRT

- Slow-fast verity is the most common, but the reverse could happen i.e. fast-slow
 - Slow-fast is mostly in adults
 - Triggered by a PAC
 - On ECG, a retrograde P wave (short RP) manifest as :
 - R' in V1
 - Pseudo S in inferior leads (II,III, AVF)
 - 2/3 of cases no P wave seen
-



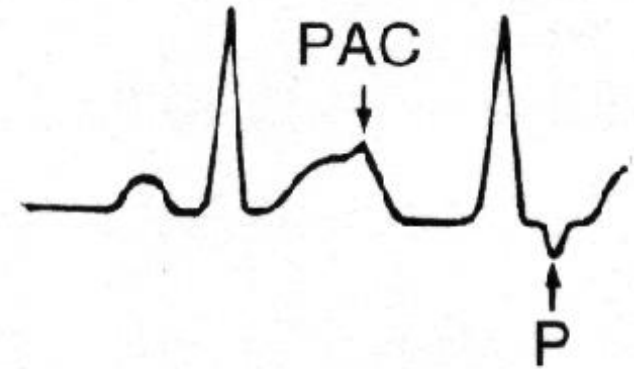
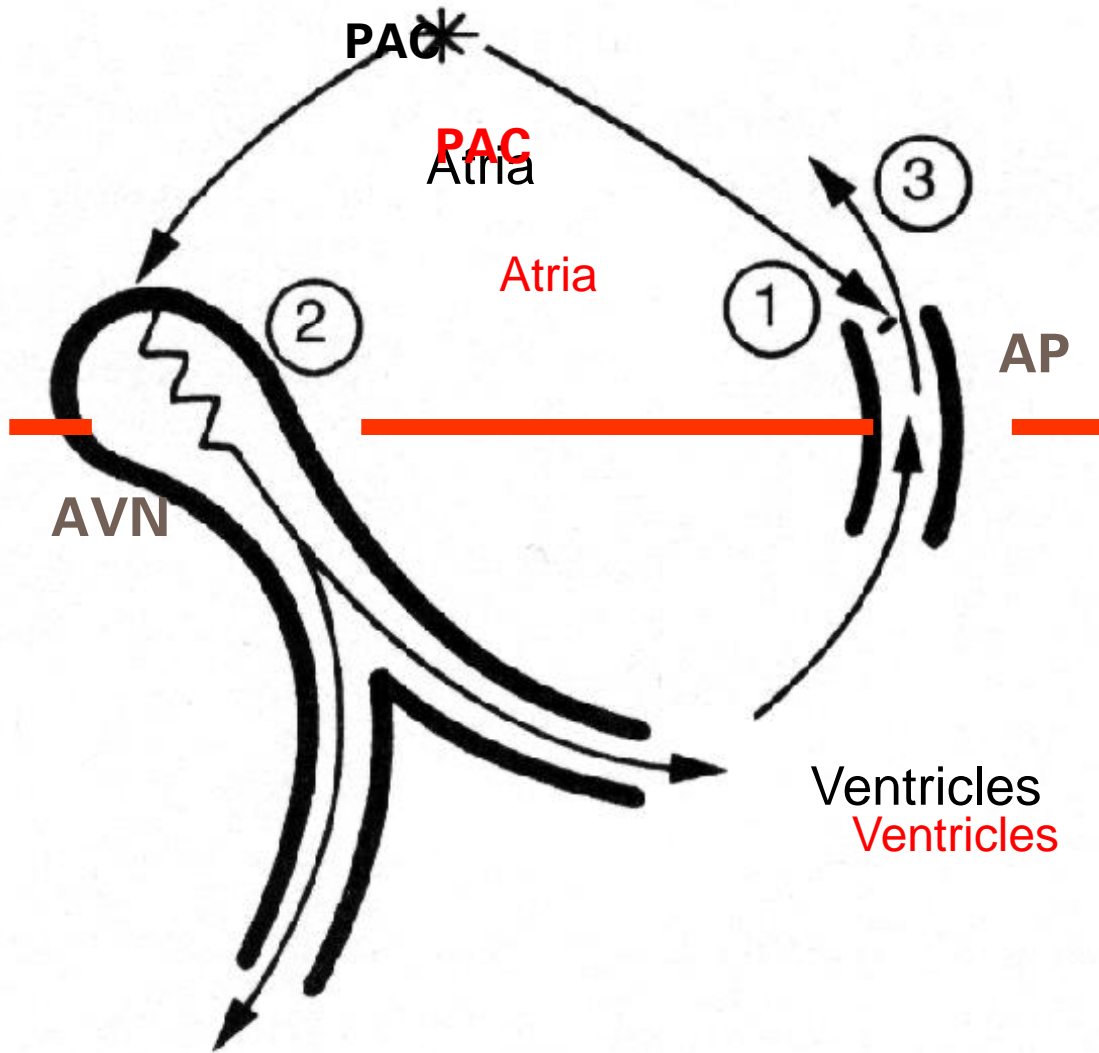
- 2nd most common SVT
- Involves reentry between the atrium and ventricle with use of the AV node as the antegrade, slow pathway and an accessory AV connection as the retrograde, fast pathway.
- Also known as “Orthodromic reciprocating tachycardia”

AVRT

- **Orthdromic tachycardia**= conduction occurs in an antegrade fashion down the AV node.
- **Antidromic tachycardia**= conduction occurs in a retrograde fashion up the AV node.

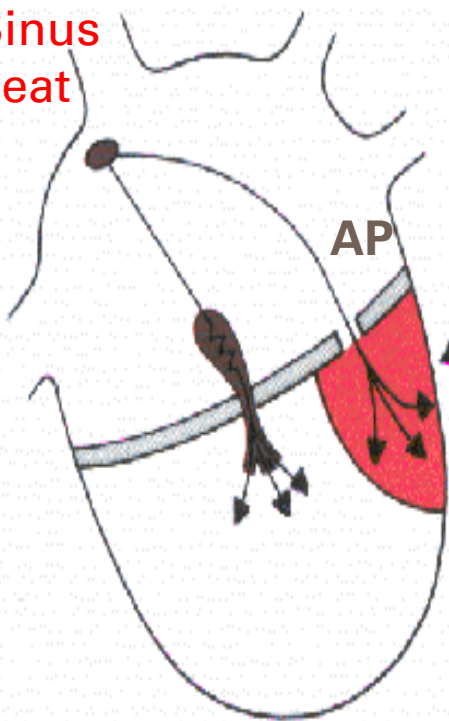
Terminology in use

Initiation of Orthodromic AV Reentrant Tachycardia



PAC = premature atrial complex (beat)

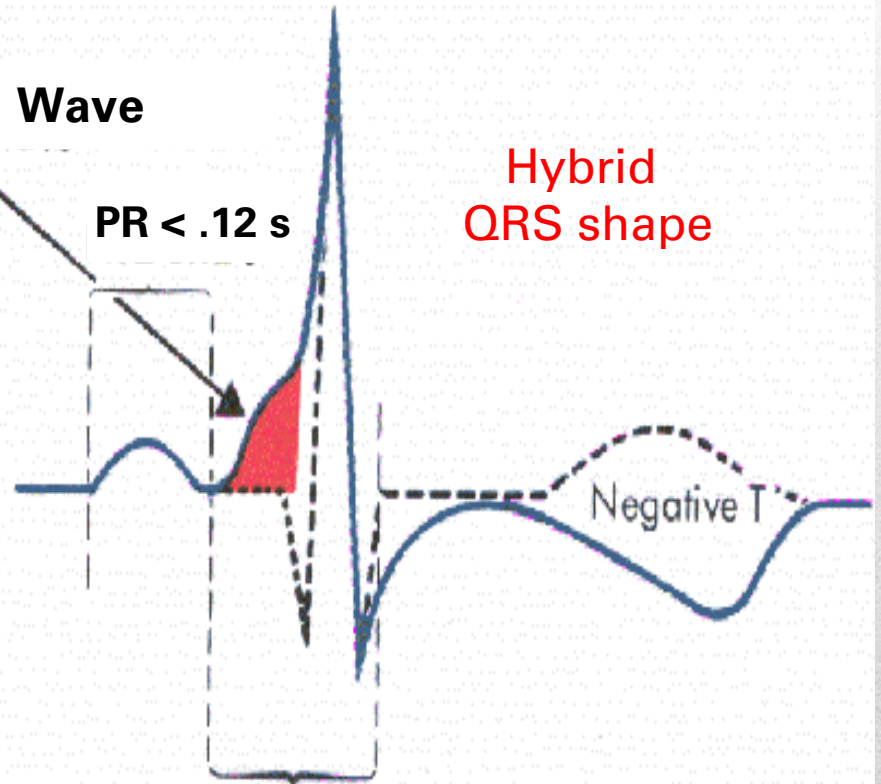
Sinus
beat



Fusion activation
of the ventricles

"Delta" Wave

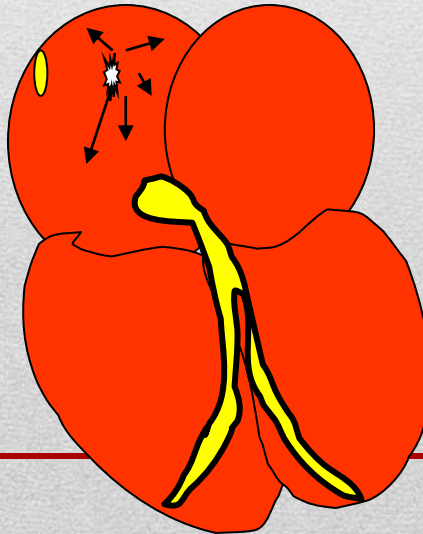
PR < .12 s



QRS ≥ .12 s

Atrial tachycardia

- Most common long RP tachycardia
- Rate 150-250 bpm
- An AV nodal independent tachycardia
- Can be associated with variable block



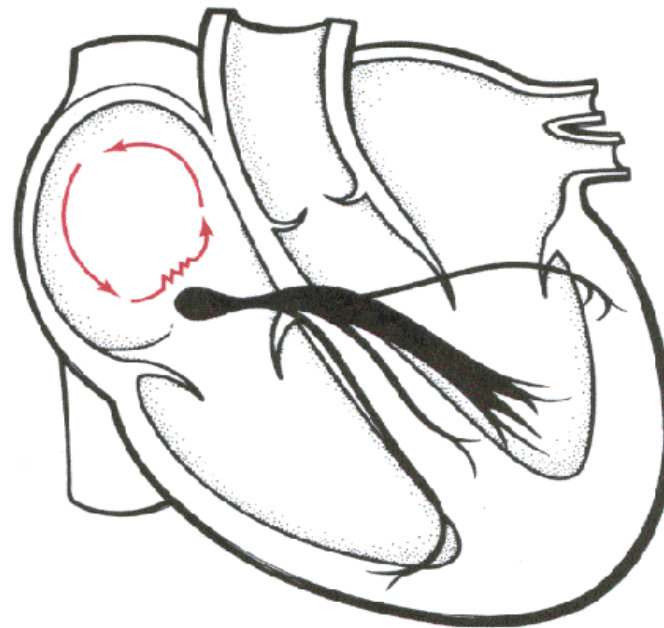


**Differs from
AV nodal or
AV reentrant
SVT**

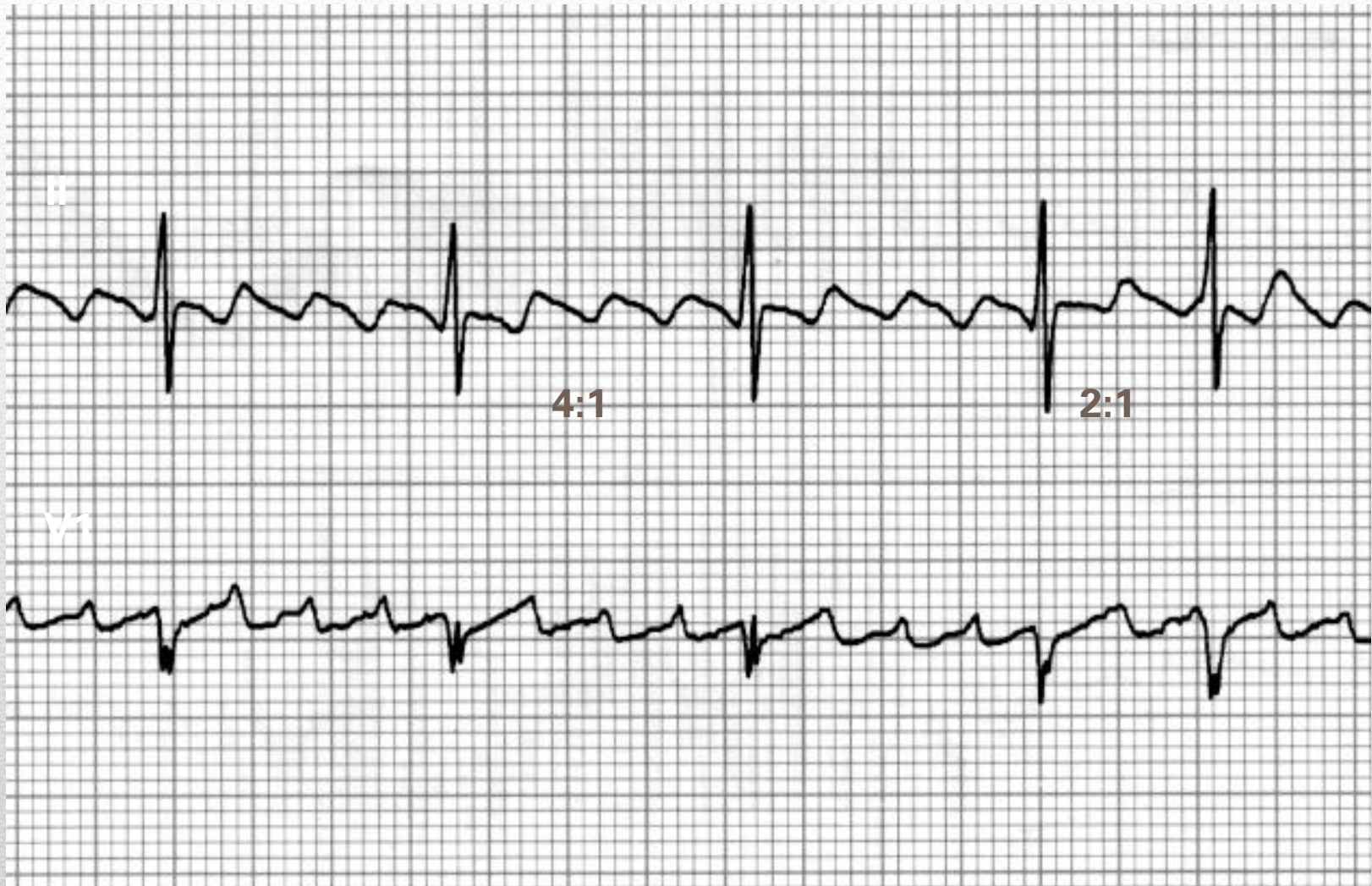
- **RP** intervals can be variable
- **RP** often $>$ PR
- (Example slower than more common rate
mof 150-250 beats per min)

- Reentrant rhythm of the ri
- Typical atrial rate is 300b
- P waves have a characteri

Atrial

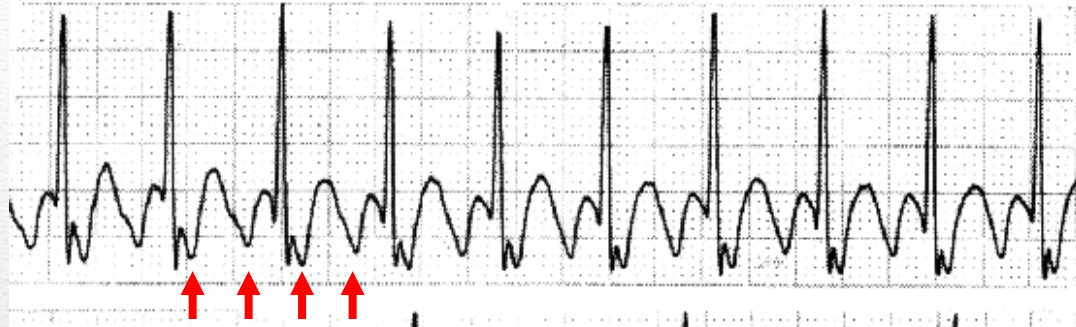


Atrial Flutter

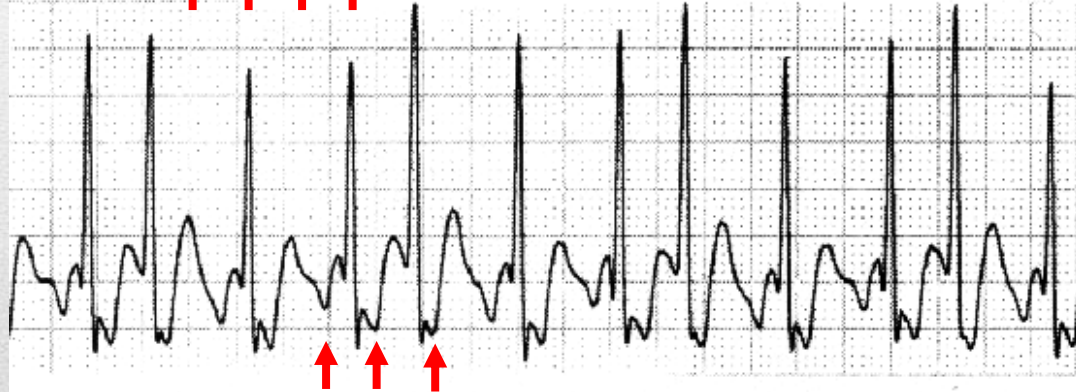


Atrial Flutter

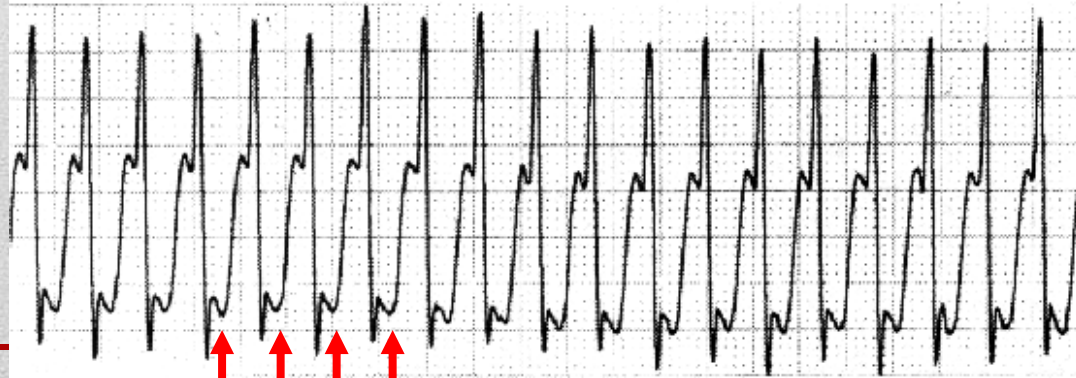
**2:1
Conduction**



**2:1 & 3:2
Conduction**



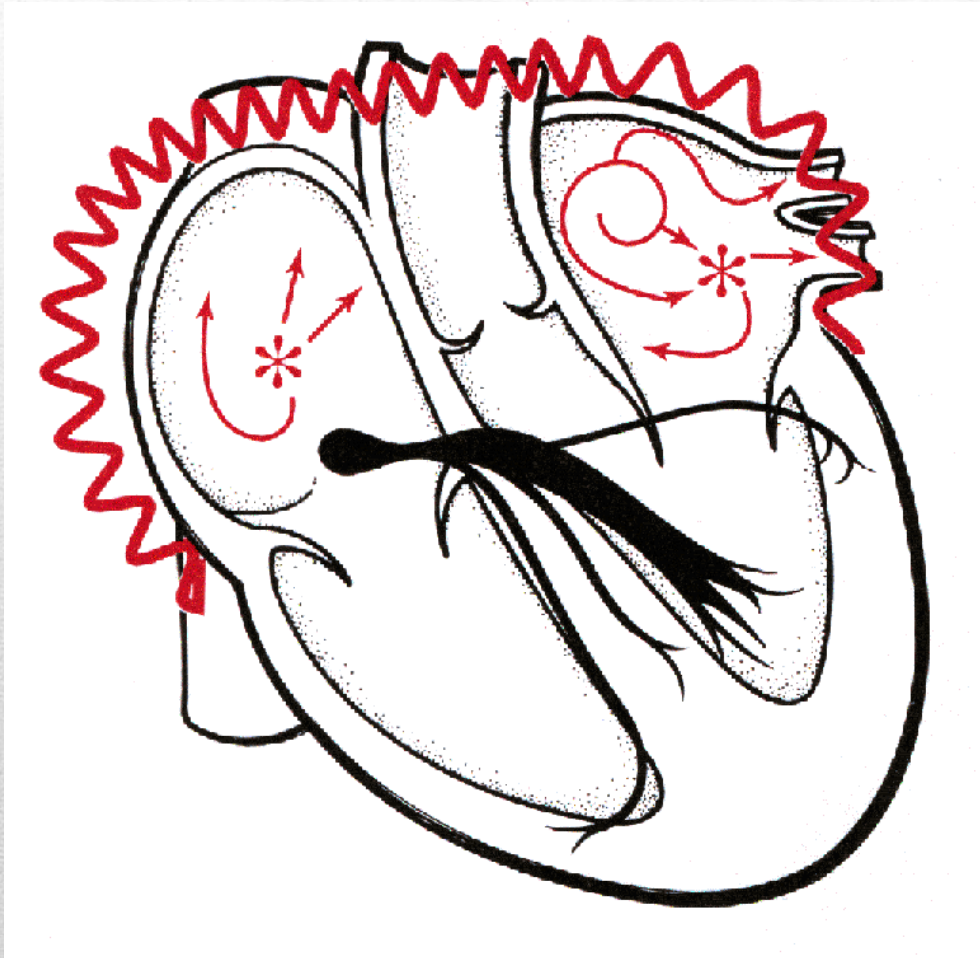
**1:1
Conduction**



**V. rate
140-160
beats/mi
n**

Atrial Fibrillation

*Focal firing
or
multiple
wavelets*



**Chaotic, rapid
atrial rate at
400-600
beats per min**

Atrial Fibrillation: Characteristic “Irregularly Irregular” Ventricular Response



- Rapid, undulating baseline (best seen in V1)
- Most impulses block in AV node → *Erratic* conduction

HOW TO MAKE THE DIAGNOSIS?

- Is it a tachycardia?
 - Is it narrow or wide complex?
 - What is the shape/axis of the P wave?
 - What is the relationship of the P wave to the QRS?
-

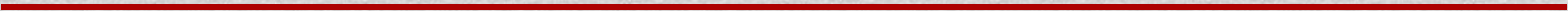
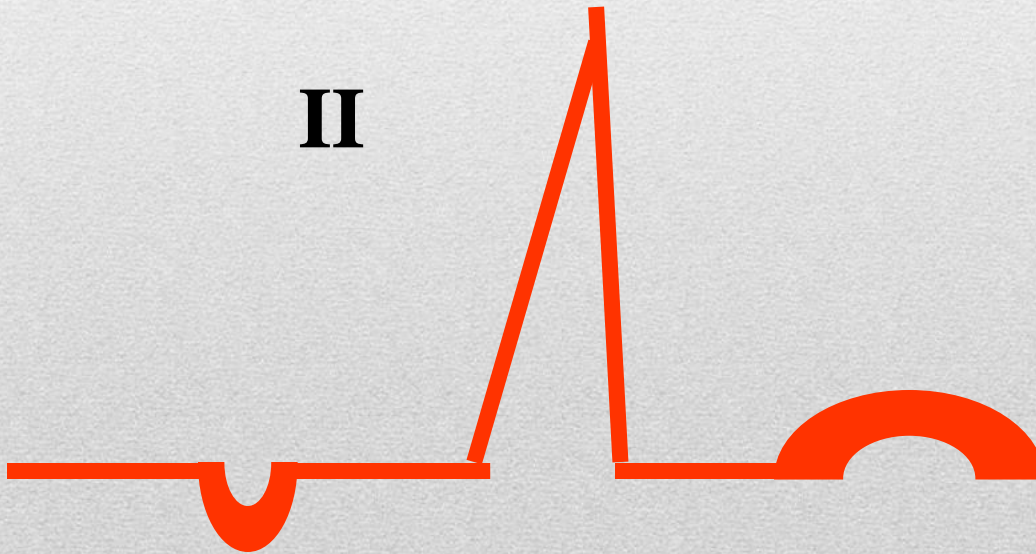
- Morphology of the p wave can aid in identifying the origin
- Look at leads II and V1 , if upright in II, and biphasic in V1, then it's likely from the SN or near to it.
- If negative in II, and upright in V1 then it likely is from elsewhere (LA)

Shape/axis of the P wave

V1



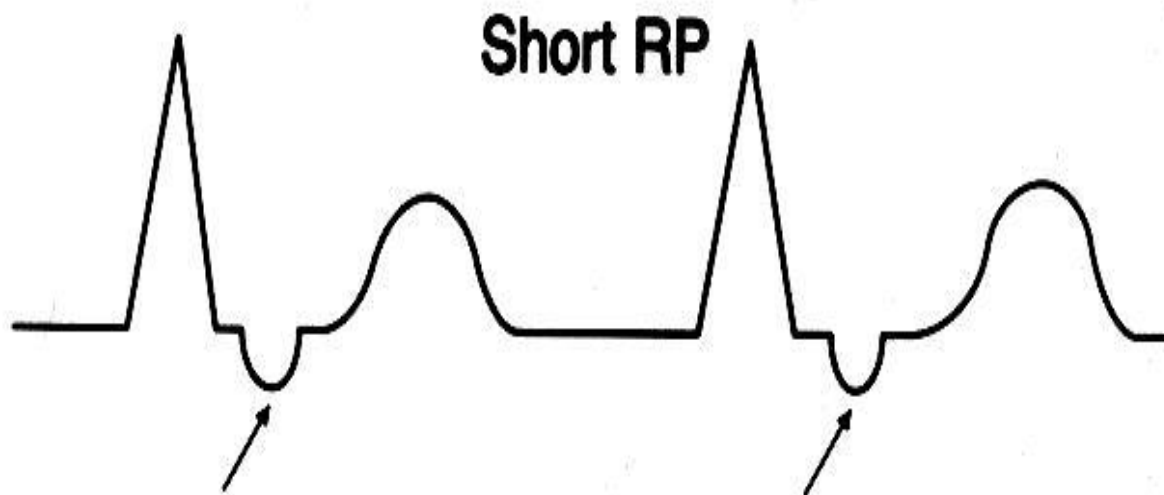
II



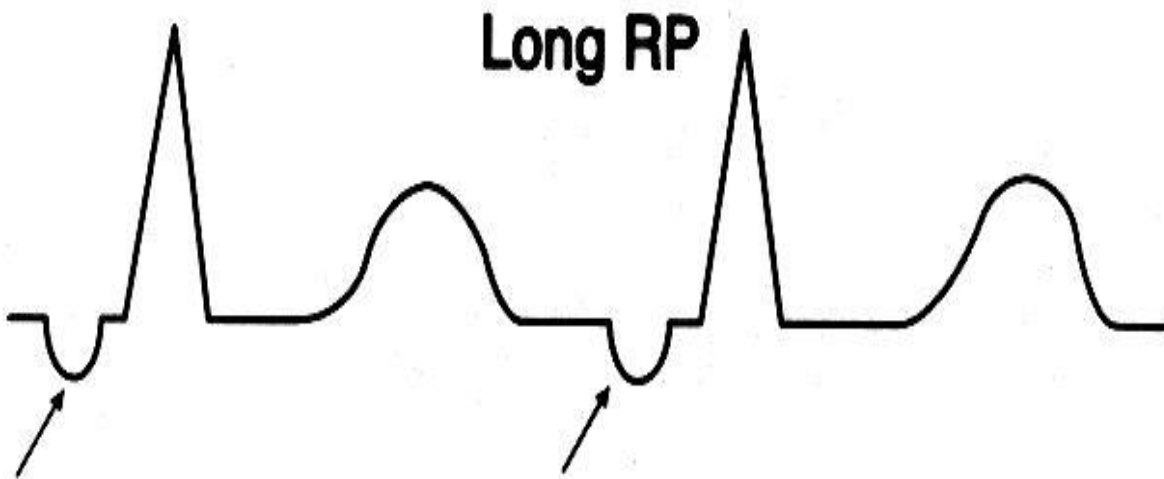
3 situations could be described:

1. P just precedes the QRS (**Long RP**)
2. P occurs simultaneously or immediately follows the QRS (**Short RP**)
3. No relation between P and QRS (AV dissociation)

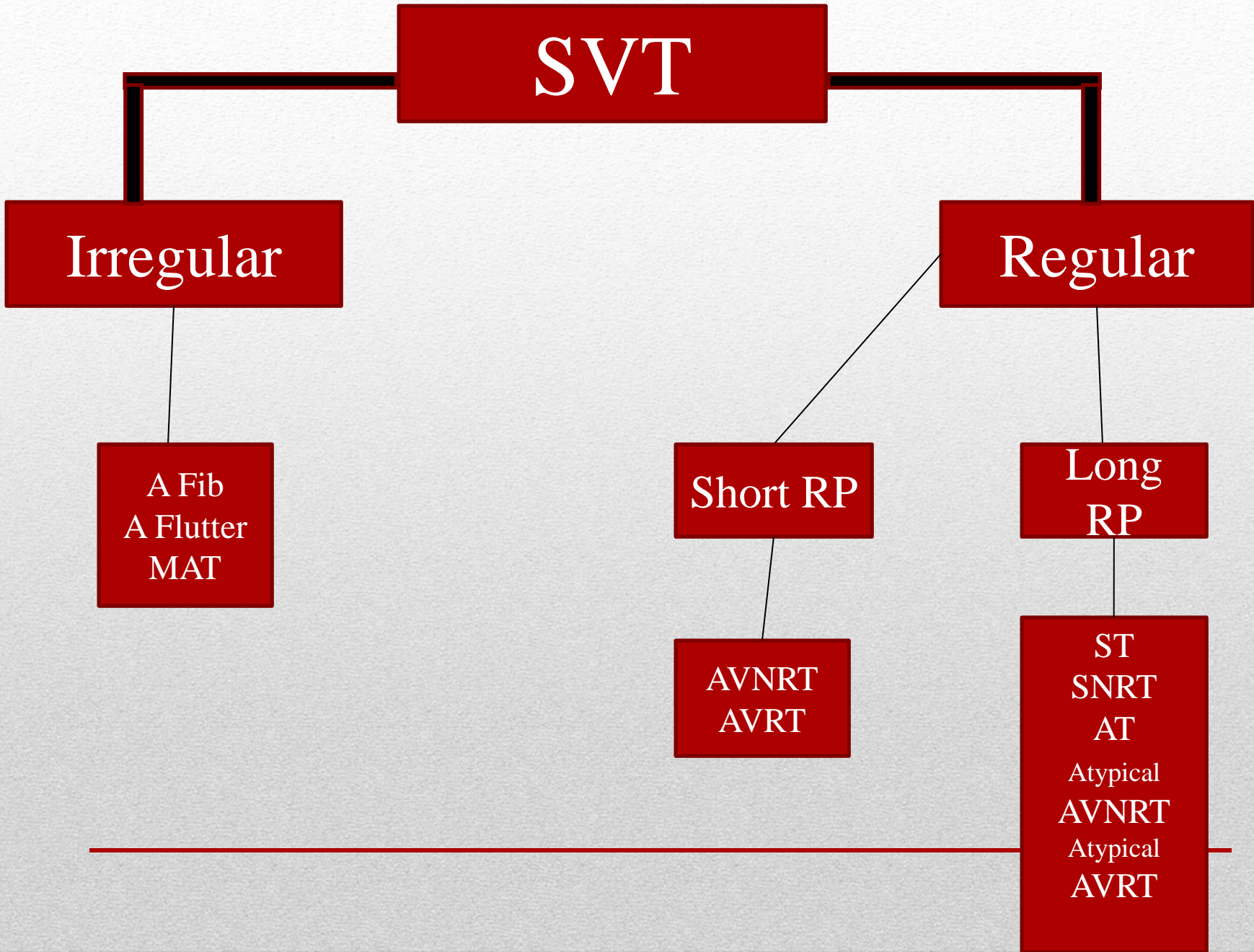
Relationship to QRS



$$RP < \frac{1}{2} RR$$



$$RP > \frac{1}{2} RR$$



BROAD THERAPEUTIC APPROACH TO CLASSIFY (NCT)

- AV nodal dependant NCT
(AVNRT, AVRT)
- AV nodal independent NCT
(AT, Flutter, ST)

Classification

- Vagotonic Maneuvers
 - Carotid sinus massage
 - Valsalva maneuver (bearing down)
 - Facial ice pack (“diving reflex;” for kids)
- Adenosine (6-12 mg I.V.)
- If SVT “breaks,” a reentrant mechanism involving the AV node is likely
- If atrial rate unchanged, but ventricular rate slows (#P’s > #QRS’s), SVT is atrial in origin

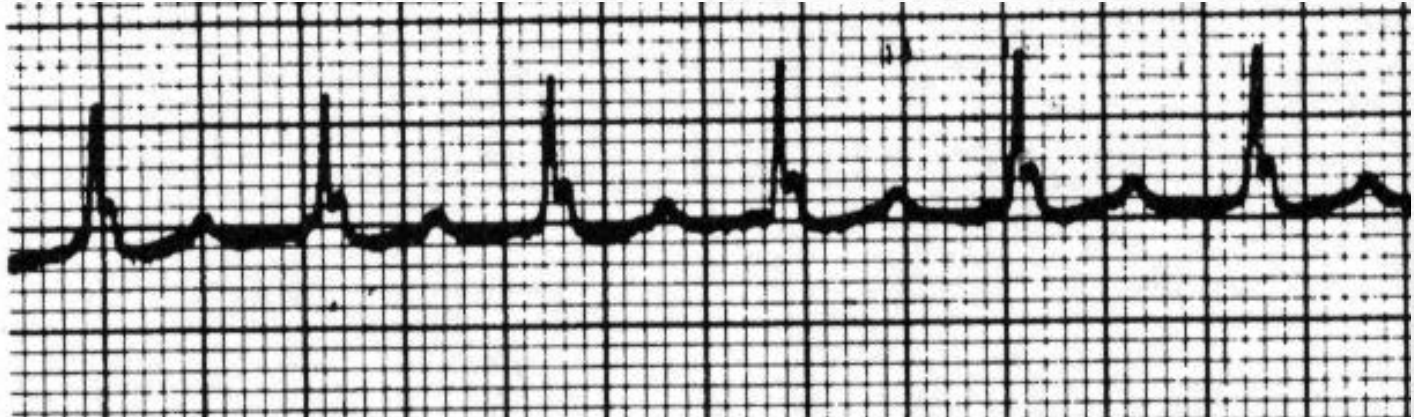
Methods of increasing AV node stimulation

- SVT termination
 - AV nodal reentrant tachycardia
 - AV reentrant tachycardia
 - No SVT termination (despite maximal attempts)
 - Sinus tachycardia
 - Atrial flutter or fibrillation
 - *Most* atrial tachycardias (a minority are “adenosine-sensitive”)
-

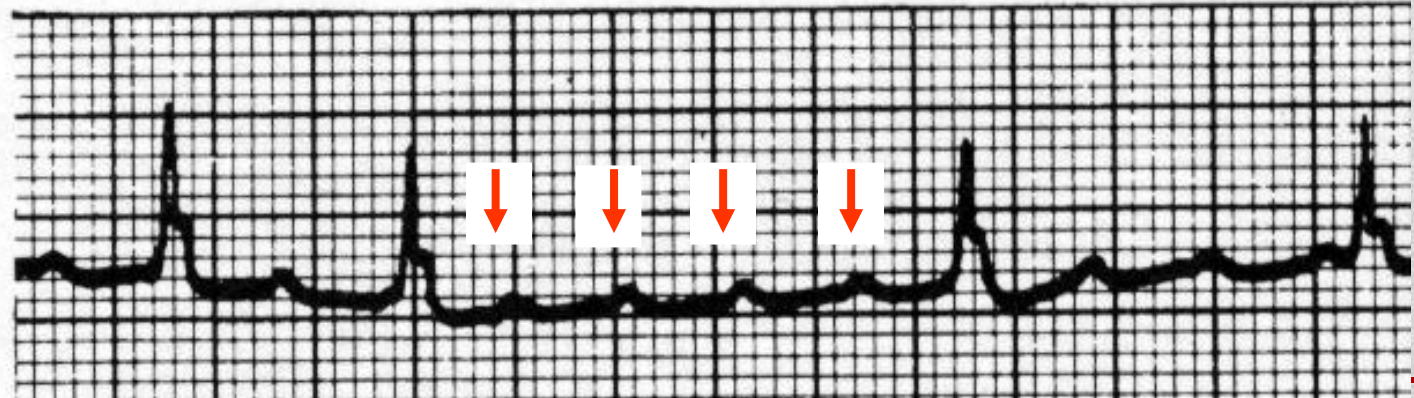
AVNRT / AVRT

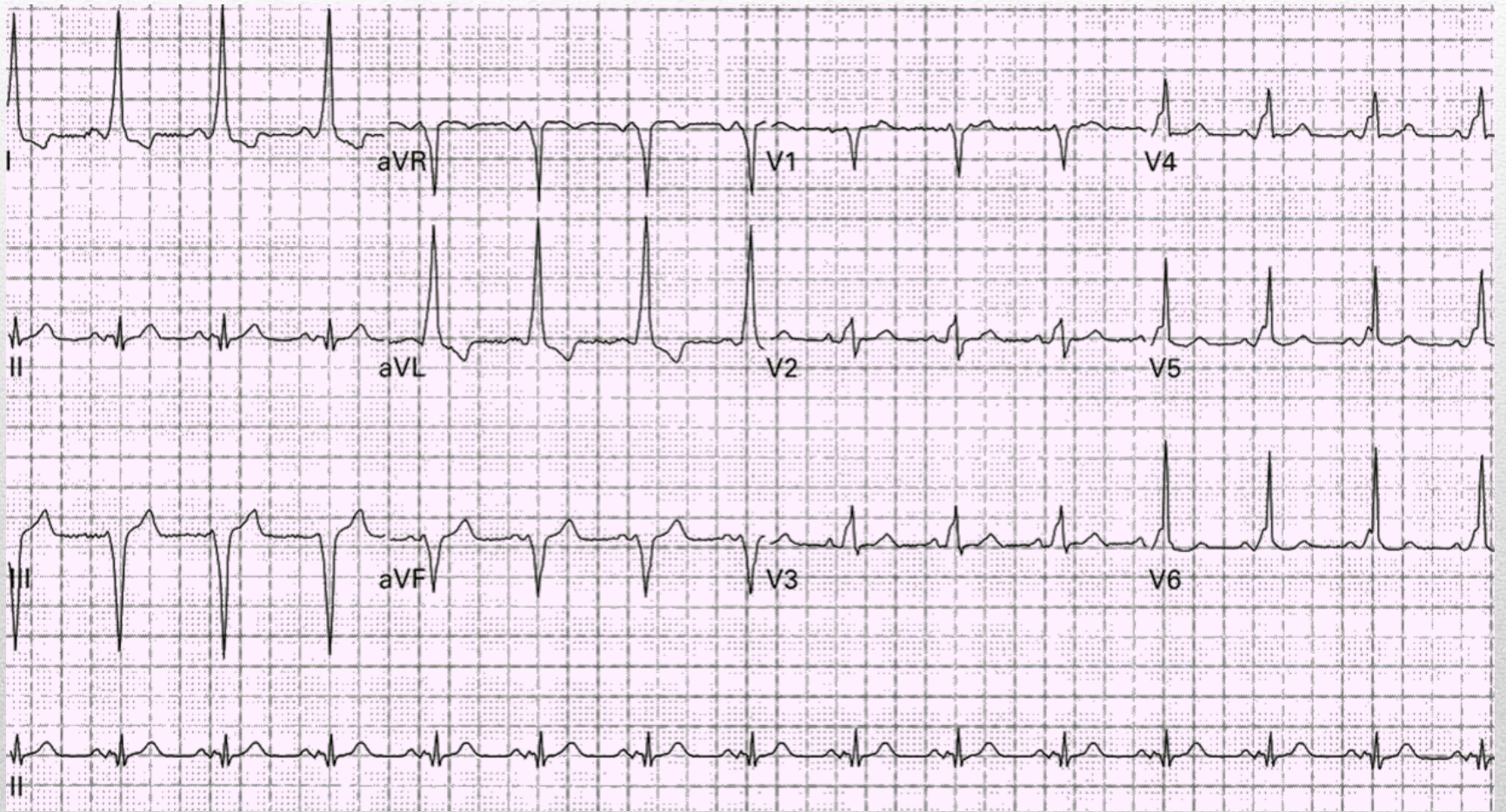


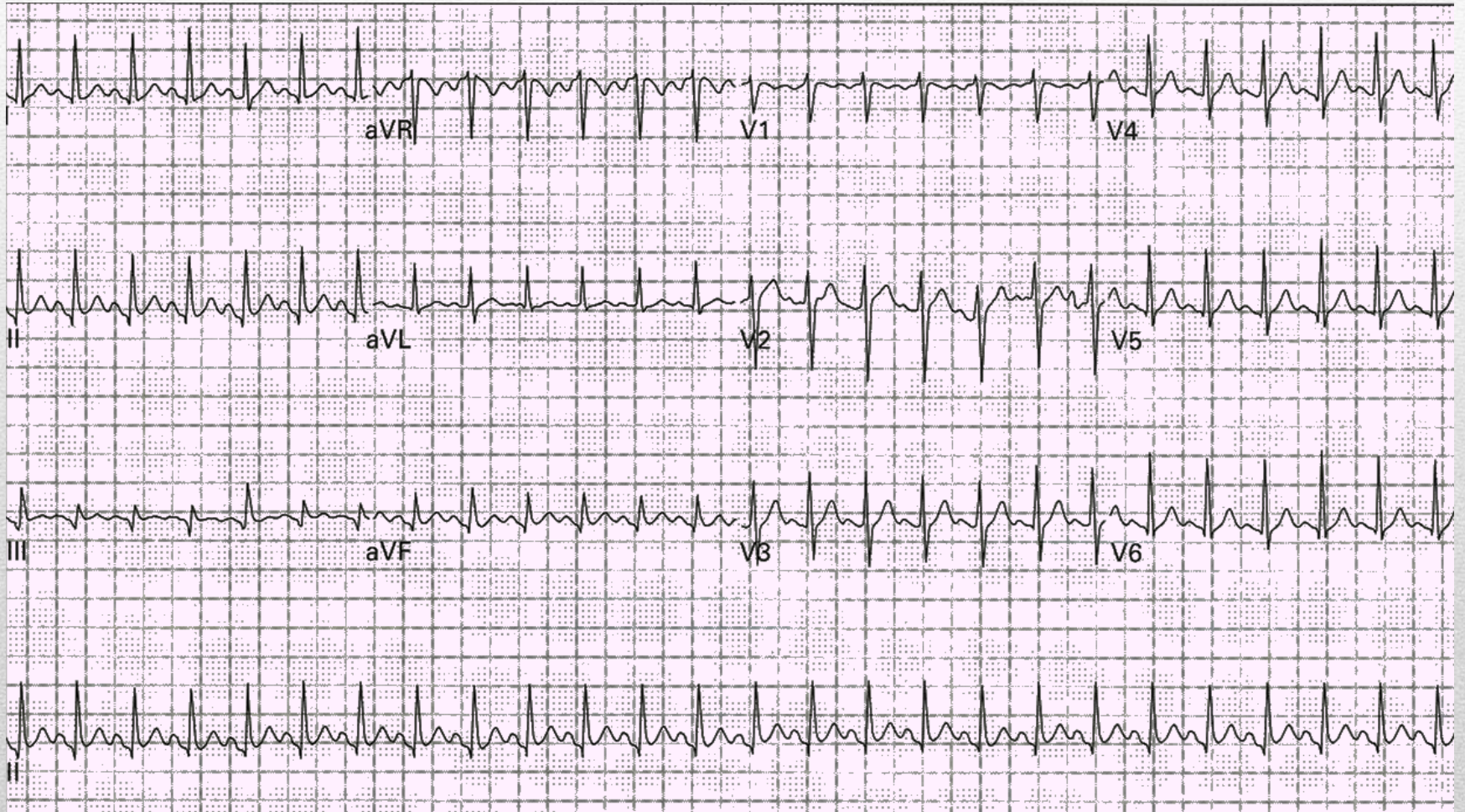
A. Flutter

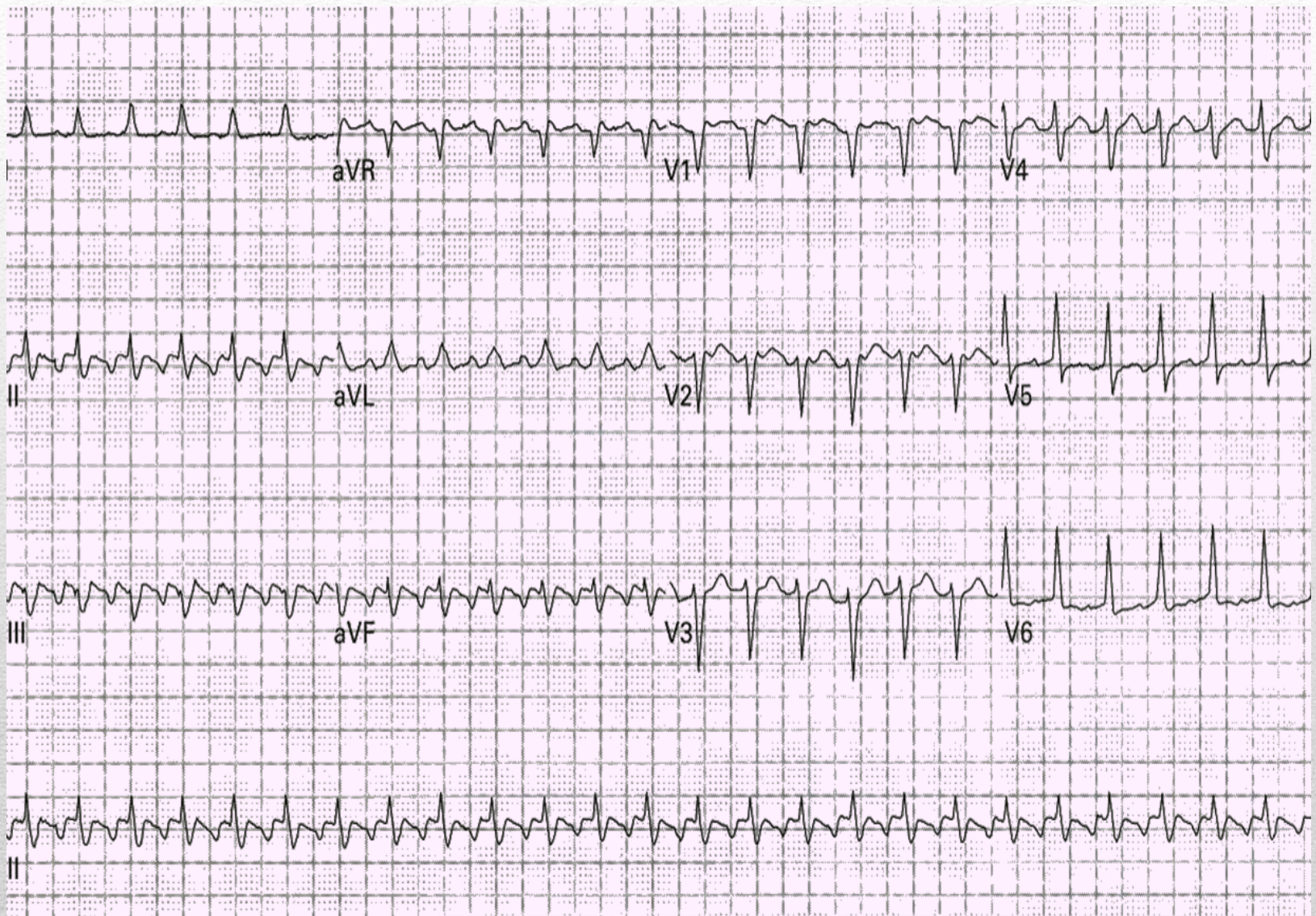


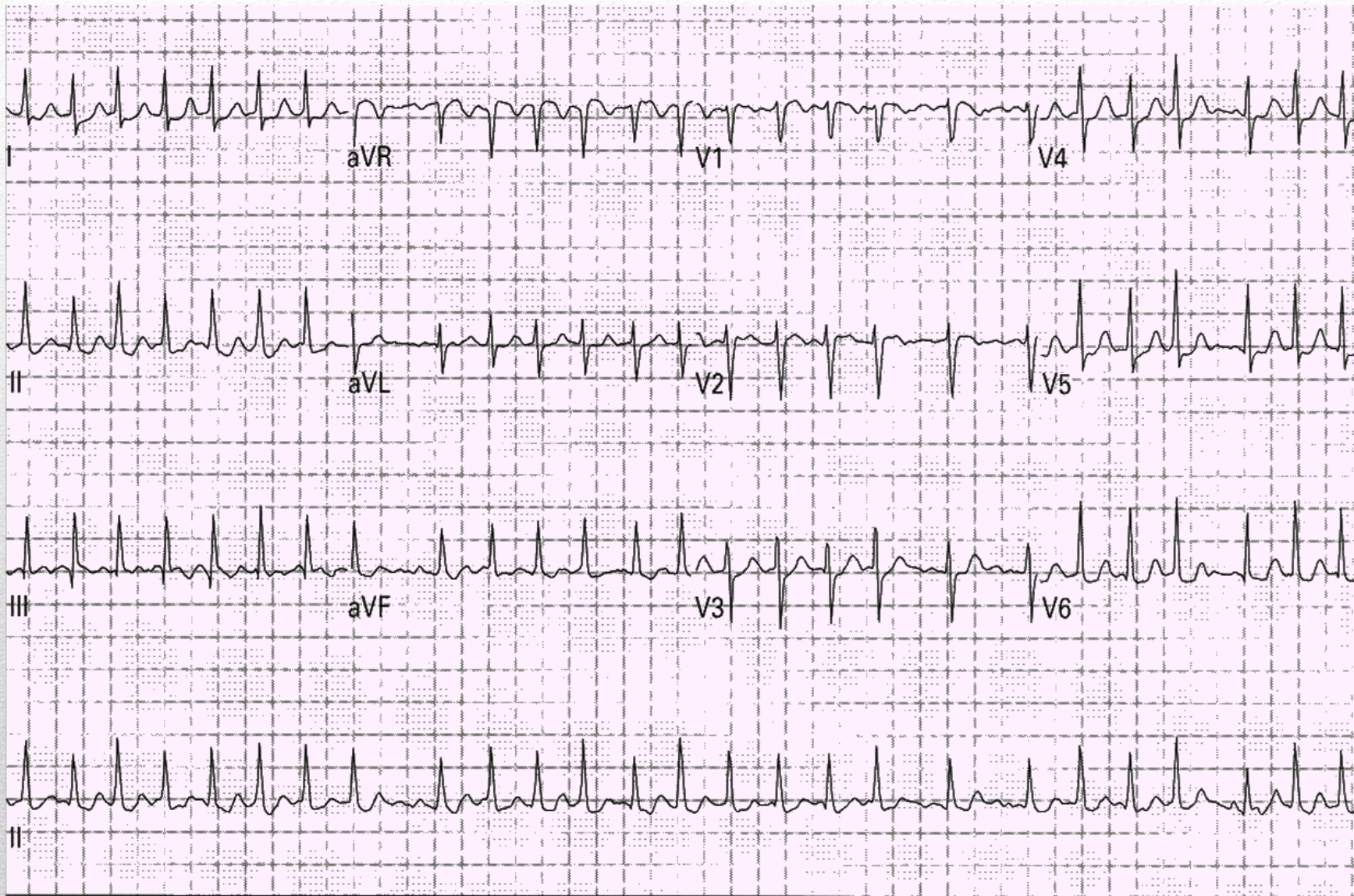
Carotid Sinus Massage









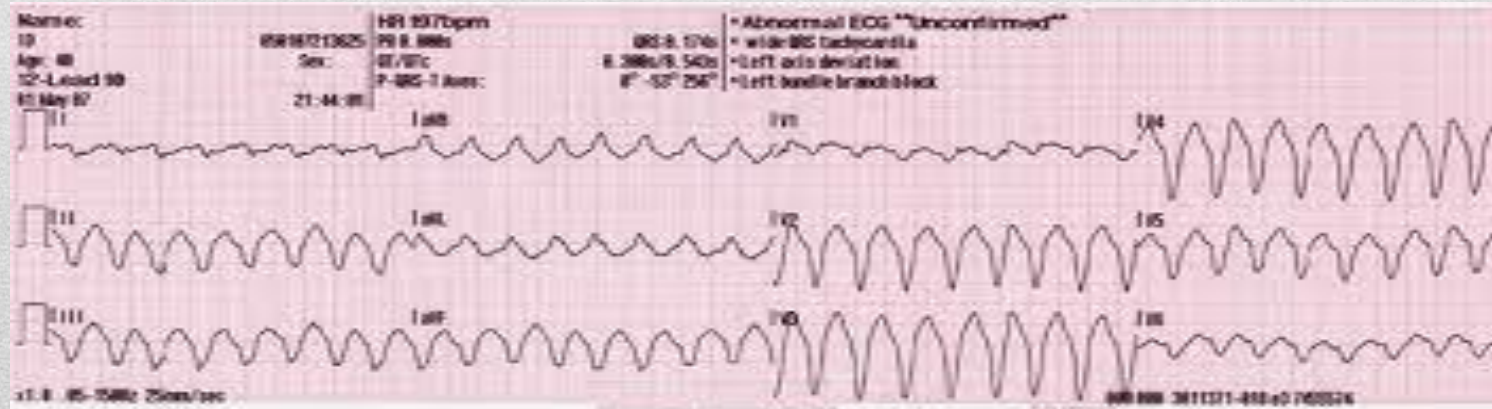




FunnyChill.com

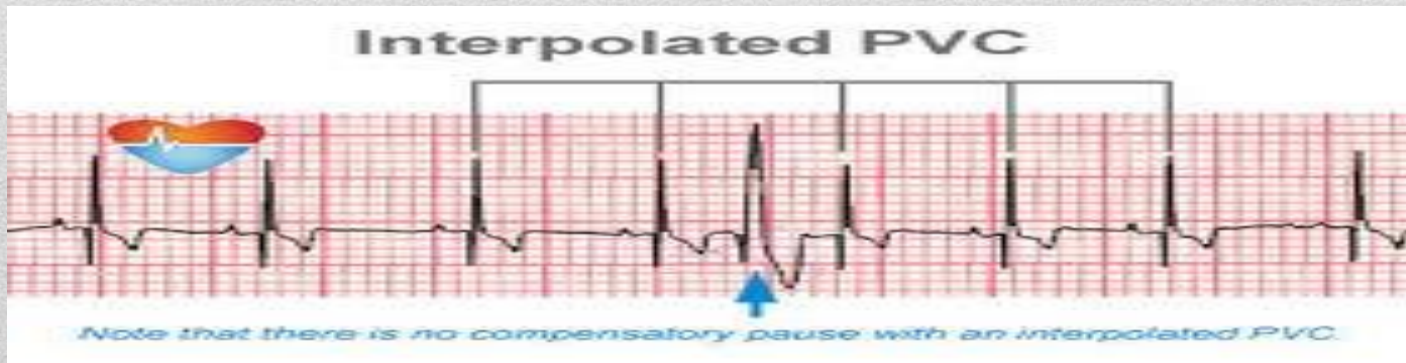
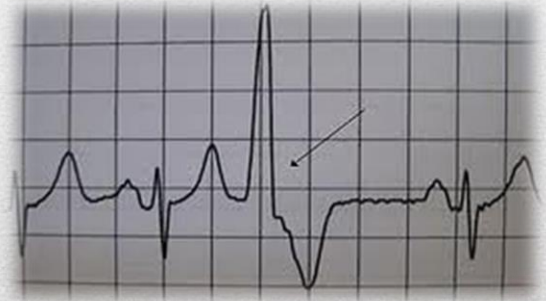
Ventricular Tachycardia

- Impulse is generated by the ventricle below bundle of His.
- **ECG findings:**
 - 1) **Wide QRS complex with absent P wave.**
 - 2) **HR > 100 BPM (REGULAR)**



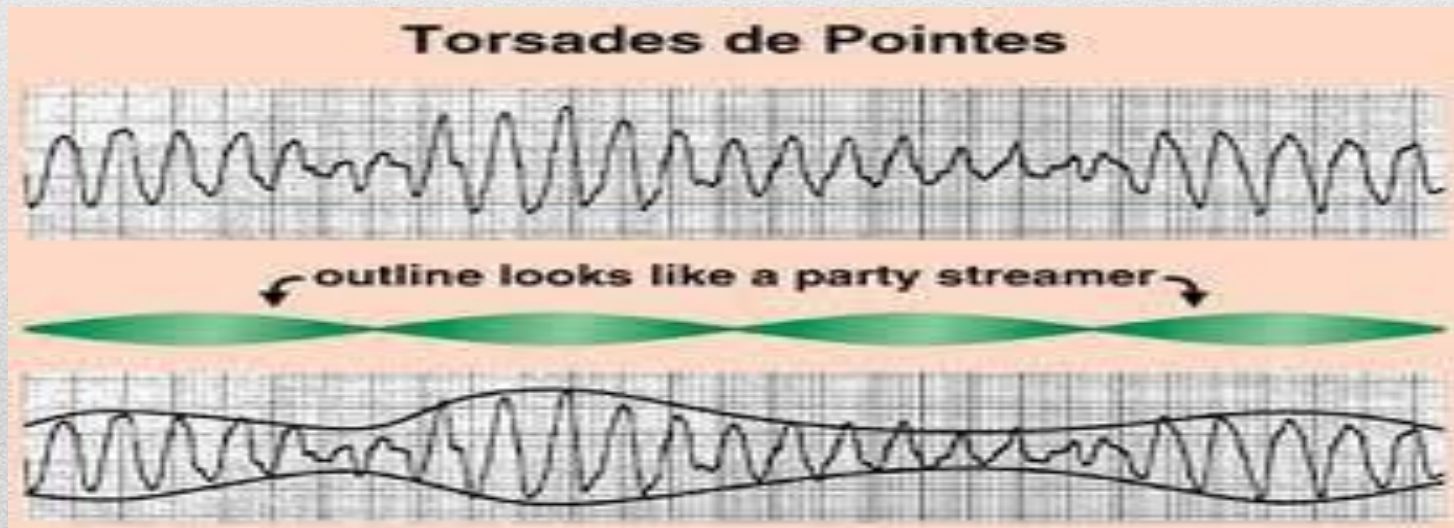
Premature Ventricular Contractions

- The ventricles fire an early impulse which causes the heart to beat earlier causing irregularity in the heart rhythm.



Torsade De point

- **ECG findings:**
 - 1) Polymorphic wide complex (ventricular) tachycardia.
 - 2) Looks like if QRSs are twisted around the isoelectric line.



Ventricular Fibrillation

- ECG findings:
Rapid, erratic electrical impulses.

