Section 7

QT Abnormalities

Other Cardiac Conditions and EKG Abnormalities

Objectives

- At the conclusion of this presentation the participant will be able to
  - Outline a systematic approach to 12 lead ECG interpretation
  - Dysrhythmias
  - Demonstrate the process for determining axis
  - List criteria for LVH, RVH, RAE, LAE LBBB, RBBB, Bifasicular and trifasicular block, acute and chronic MI changes
  - **Define QTc significance and other EKG Abnormalities**

Causes of Regular, Wide Complex Tachycardia

- Ventricular Tachycardia
- SVT with preexisting BBB
- SVT with aberrant conduction

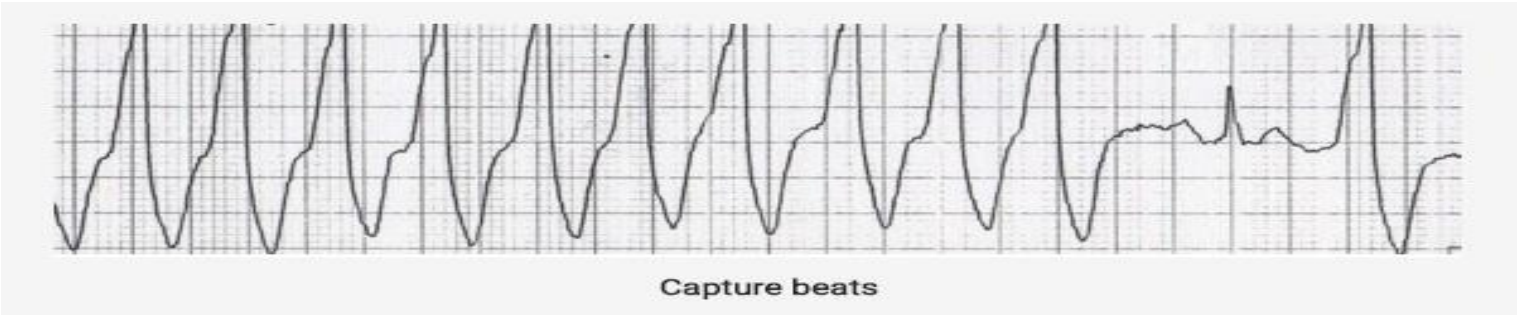
HIS DEBS

- H ypoxia
- I schemia
- S ympathomimetic disturbances
- D rugs
- E lectrolytes
- B rady
- S tretch

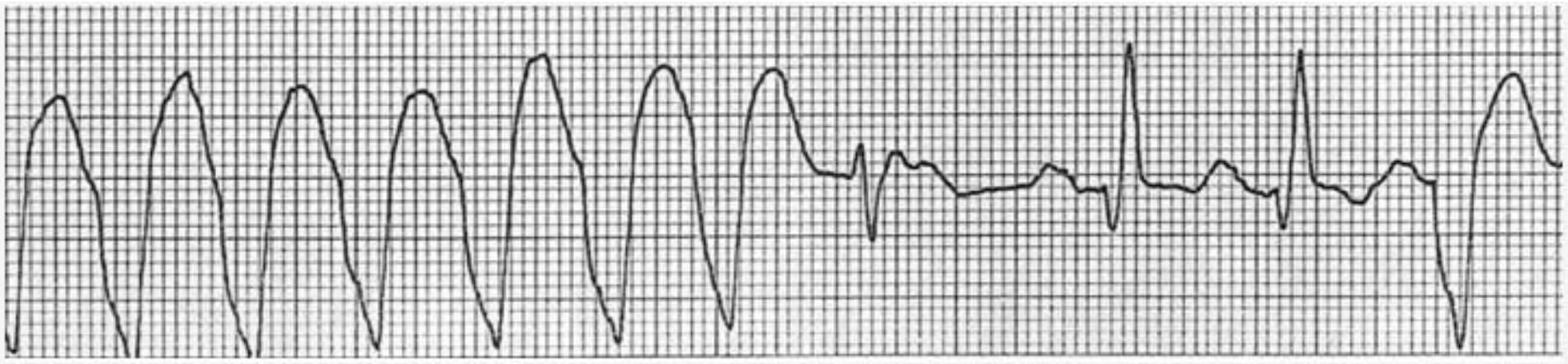
VT vs. SVT with aberrancy

- IT is more likely VT if:
  - Absence of typical RBBB or LBBB
  - Extreme axis deviation (northwest axis)
  - Very broad complexes (> 160 ms)
  - Capture beats
  - Fusion beats
  - Positive or negative concordance throughout chest leads
  - RSR’ complexes with a taller left rabbit ear. This is the most specific finding in favor of VT

Capture Beats



Capture beats



Fusion beats – the first of the narrower complexes is a fusion beat (the next two are capture beats)

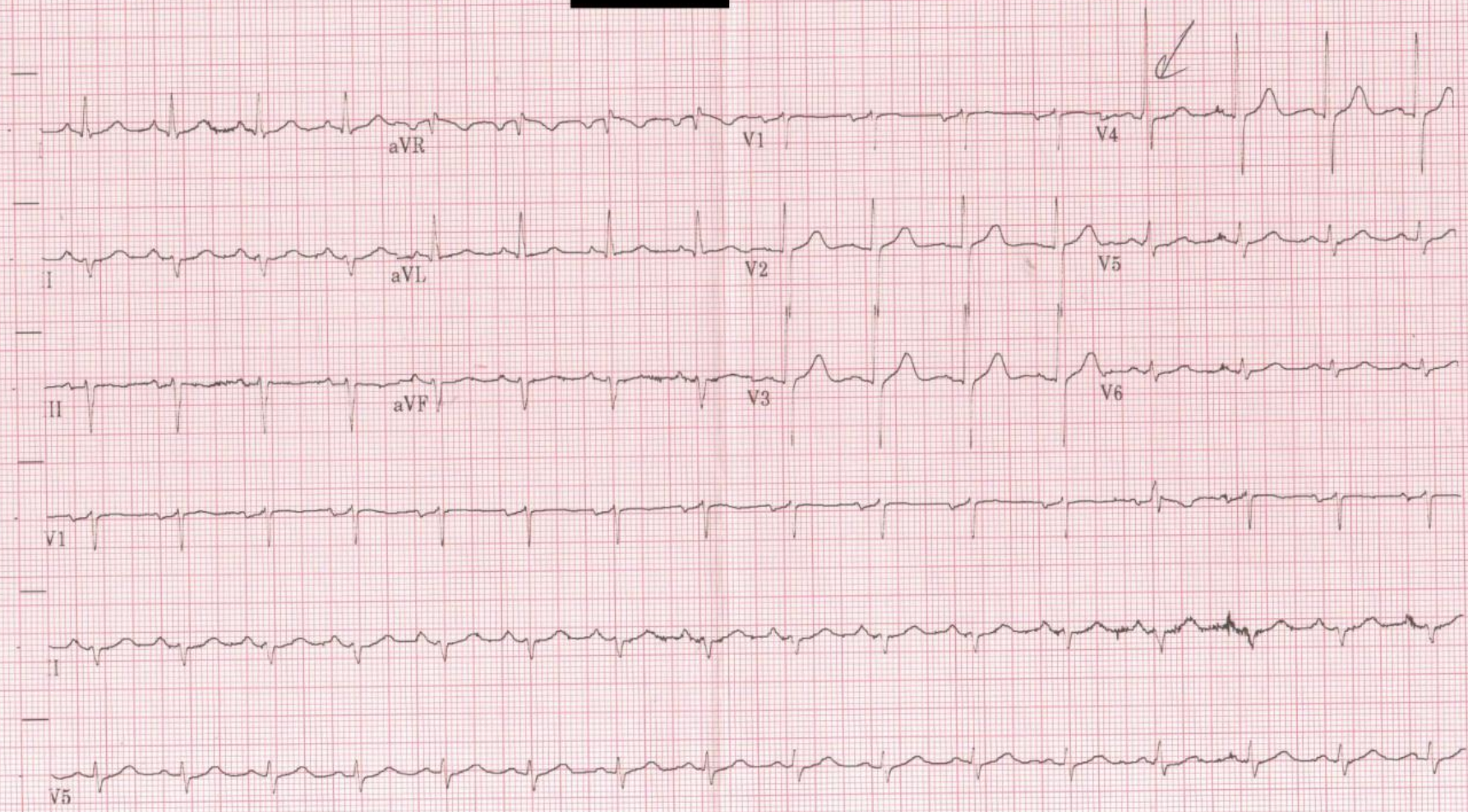
[redacted]

Vent. rate	95 bpm	Normal sinus rhythm
PR interval	148 ms	Left anterior fascicular block
QRS duration	88 ms	Abnormal ECG
QT/QTc	344/432 ms	
P-R-T axes	55 -46 25	

[redacted]

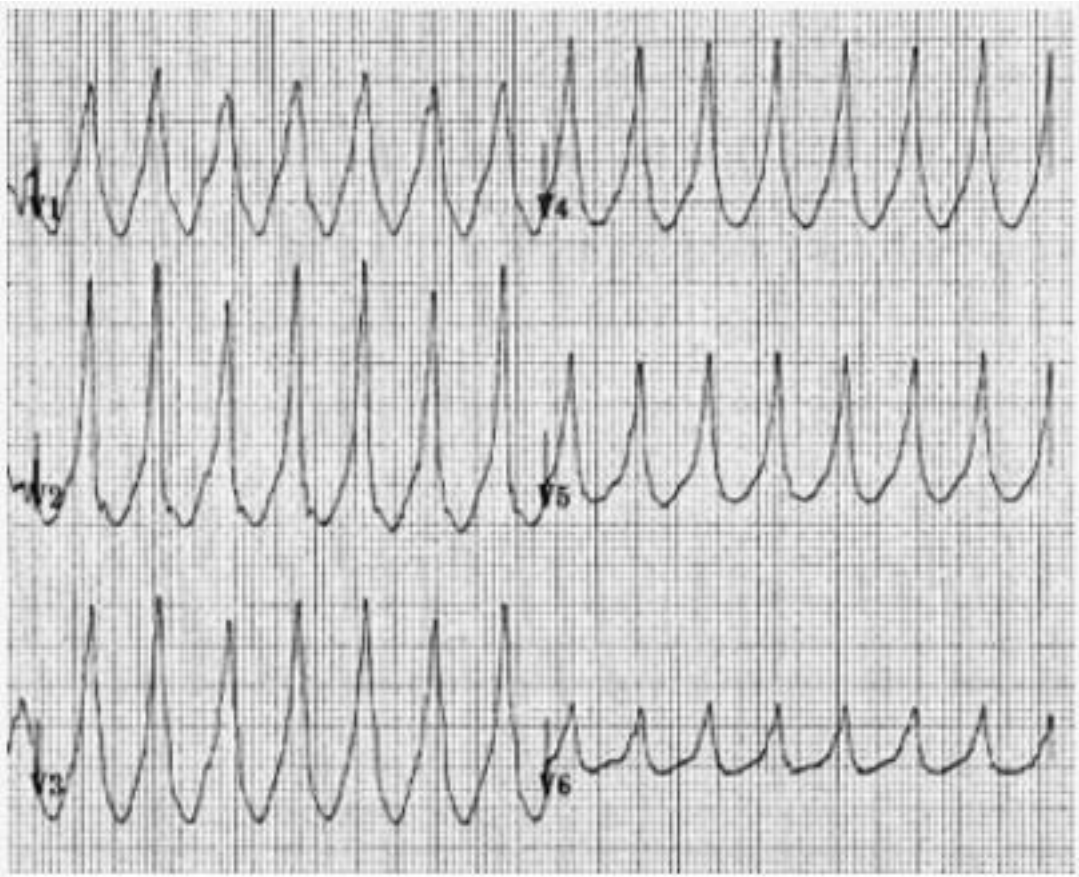
[redacted]

Unconfirmed Fusion

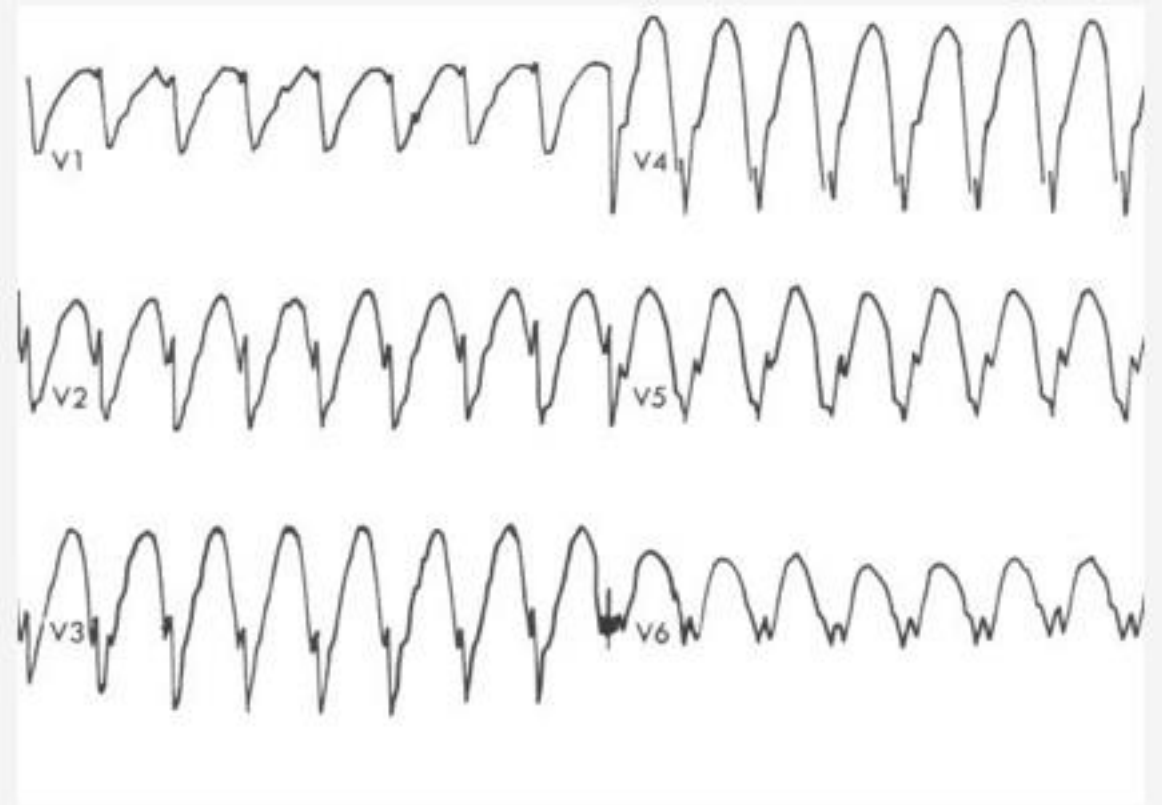


OAAPN 10/2023

150 Hz 25.0 mm/s 10.0 mm/mV 4 by 2.5s + 3 rhythm lds MAC5K 005A 12SL v231



Positive concordance in VT



Negative concordance in VT

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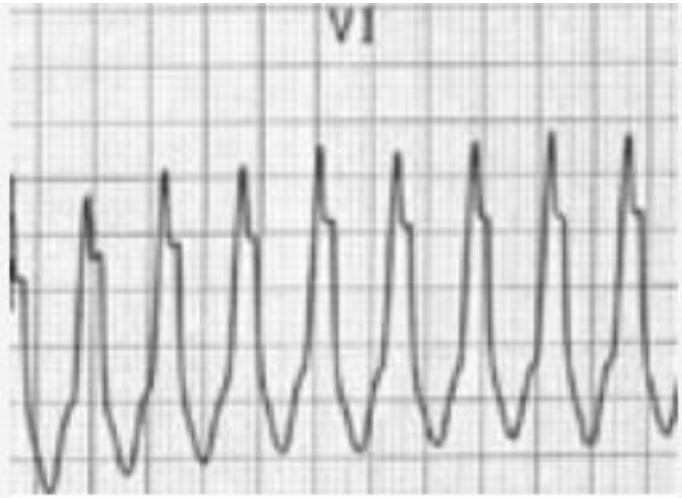
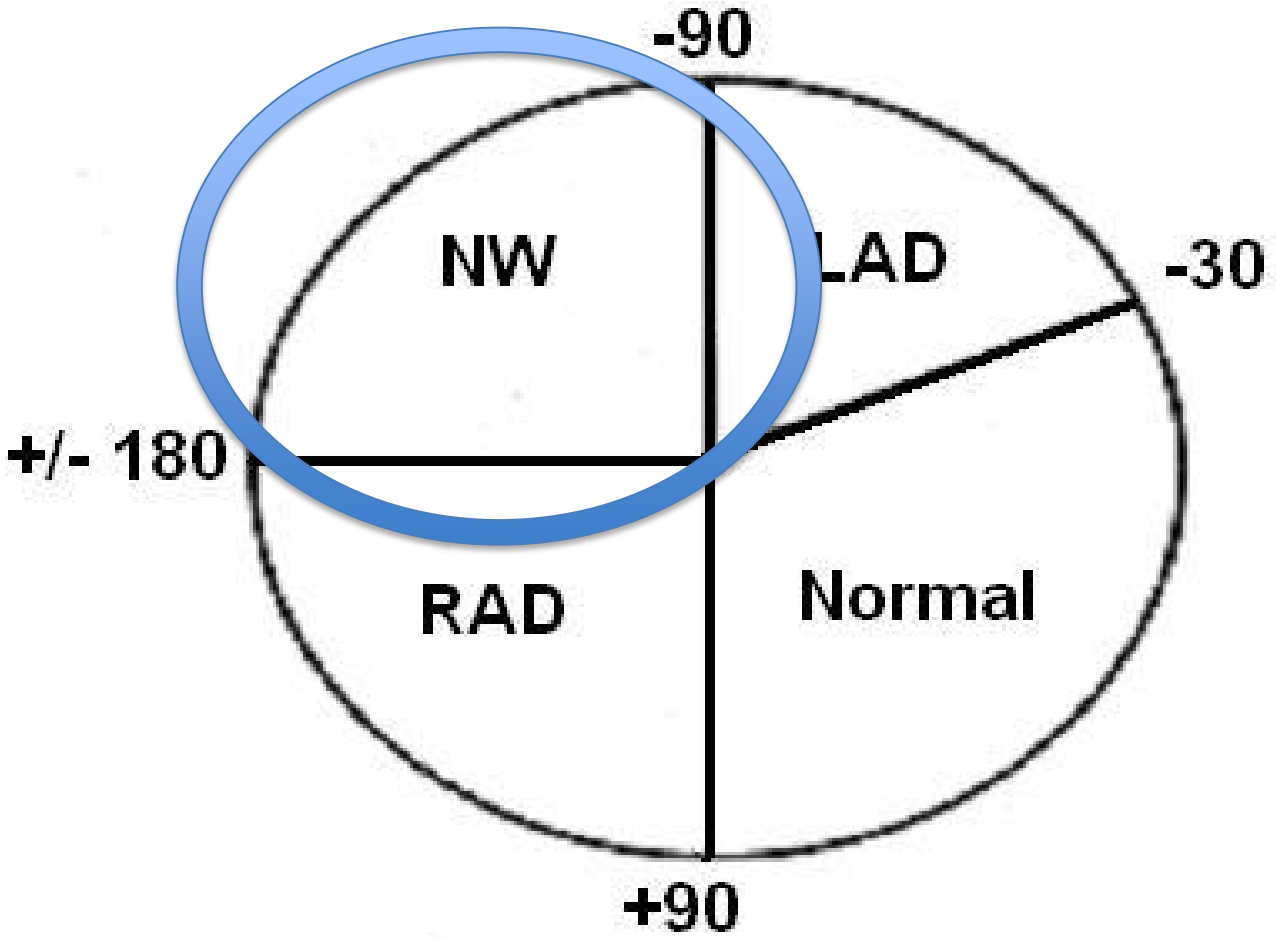


Taller left rabbit ear in VT

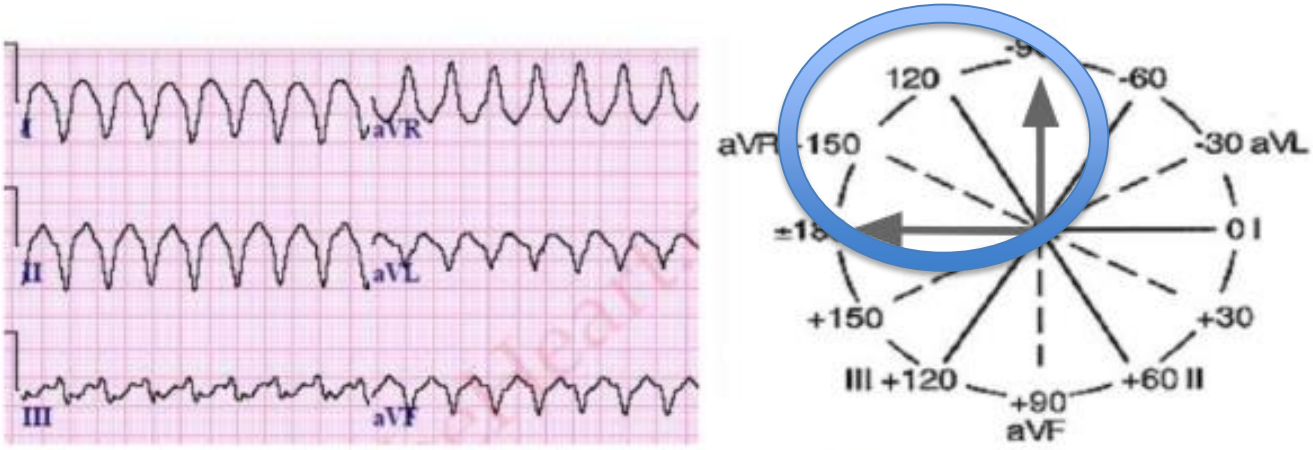
Northwest Axis



Indeterminate Axis or Northwest Axis

Indeterminate Axis

If the QRS is downward (negative) in lead I and downward (negative) in lead aVF, then the axis is indeterminate and sometimes referred to as "northwestern axis". This finding is uncommon and usually from ventricular rhythms, but can also be from paced rhythms, lead misplacement and certain congenital heart diseases.



Indeterminate Axis of the QRS Complex:
Negative in lead I and negative in lead aVF

SVT or AV nodal re-entry tachycardia (AVNRT)

- Classified based on site of origin (atria or AV node) or regularity (regular or irregular)

- QRS width not helpful and influenced by pre-existing BBB, Rate related aberrant conduction or accessary pathways

Classification of SVT by site of Origin

	Regular	Irregular
Atrial	ST Atrial Tach Atrial Flutter Inappropriate ST SN re-entrant tach	Atrial Fibrillation Atrial Flutter with variable block Multifocal atrial Tach
Atrioventricular	AV re-entry tach (AVRT) AV nodal re-entry Tach (AVNRT) Automatic Junctional tachycardia	

AVNRT

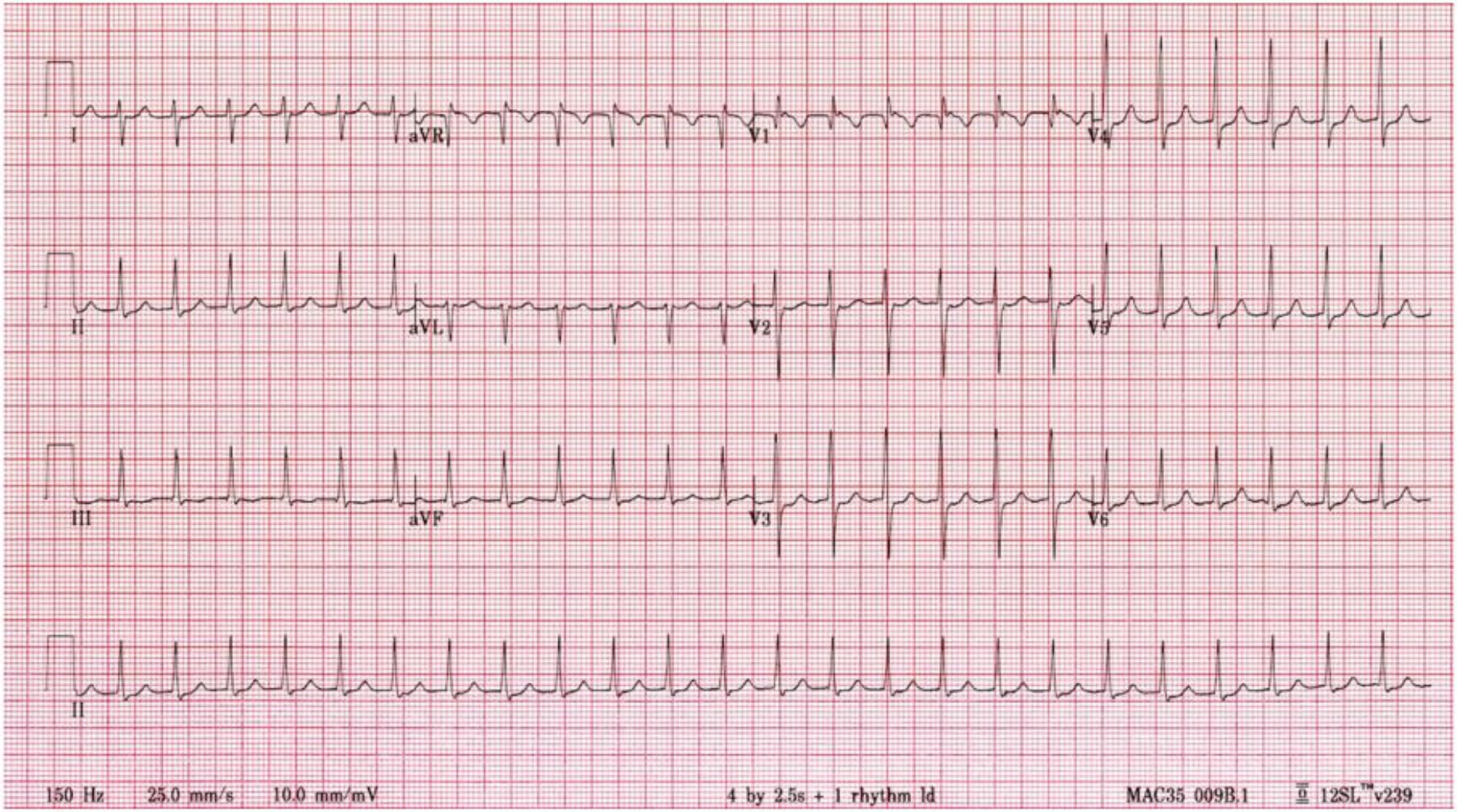
- Most common cause of palpitations in pts with structurally normal hearts
- Occurs spontaneously or upon provocation (caffeine, ETOH, Beta agonists, sympathomimetics (amphetamines)
- More common in women and may occur in young healthy patients
- Sudden onset of rapid, regular palpitations
- SOB
- Pts with CAD may c/o angina
- Tachy rate 140-220 bpm
- Generally well tolerated
- May cease spontaneously and abruptly

Typical ECG findings

- Regular tachy 140-280 bpm
- QRS complexes usually narrow (< 120 msec) unless pre-existing BBB
- ST-segment depression may be seen without CAD
- QRS alternans
- P waves if visible exhibit retrograde conduction with P-wave inversion in leads II, III, aVF
- P waves may be buried in the QRS

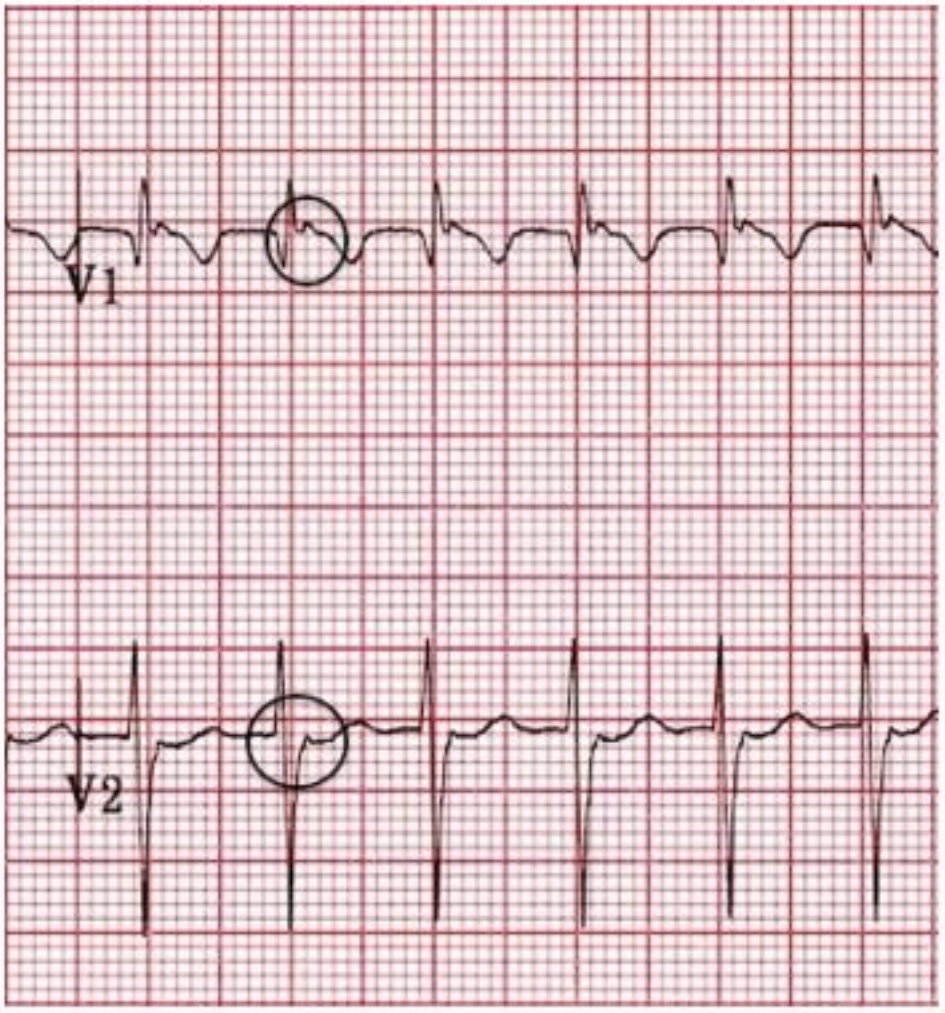
Slow –Fast AVNRT

Example 1a

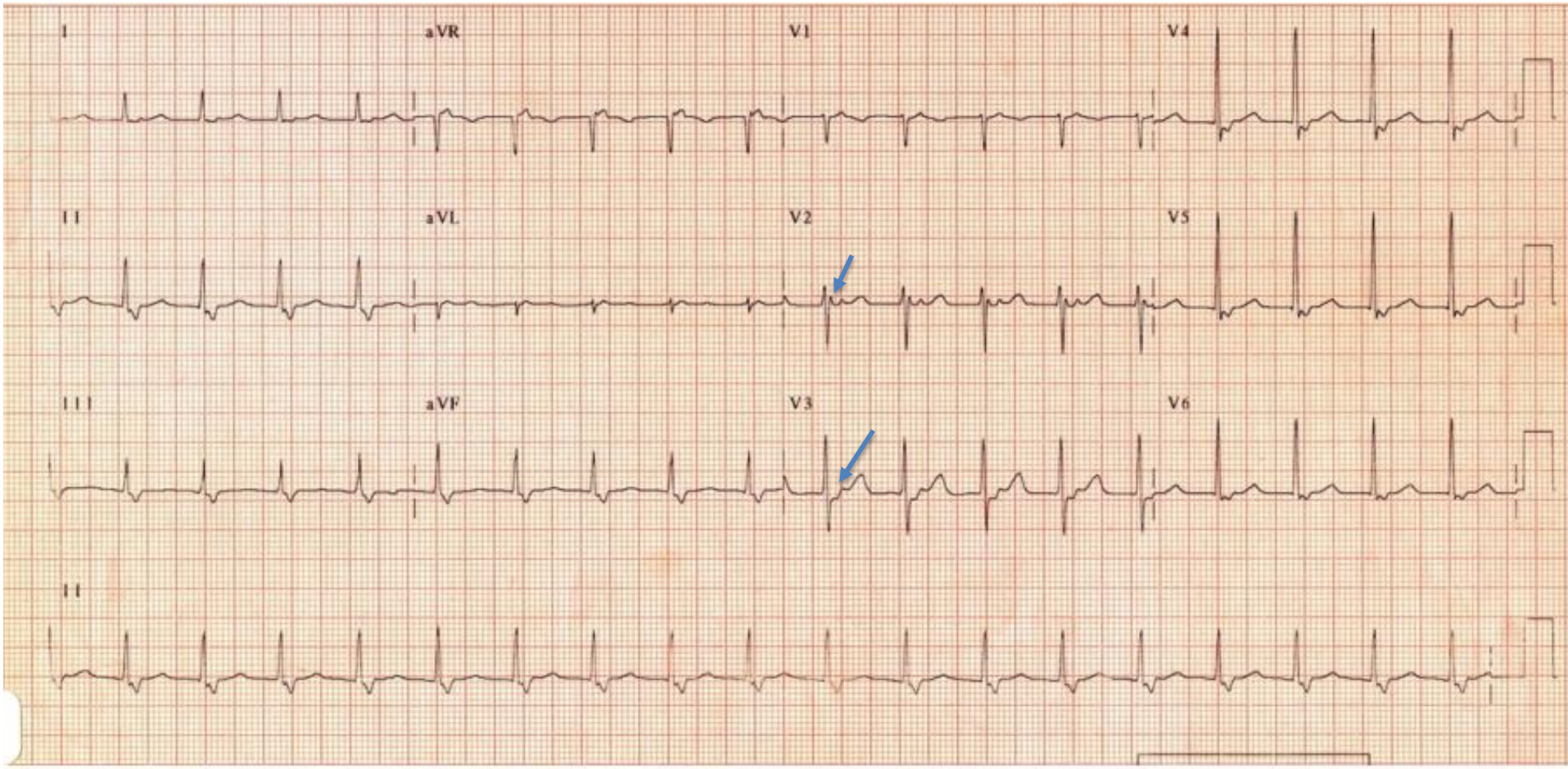


Typical AVNRT

- Narrow complex Tachycardia
- No visible P-waves
- There are pseudo R' waves in V1-2



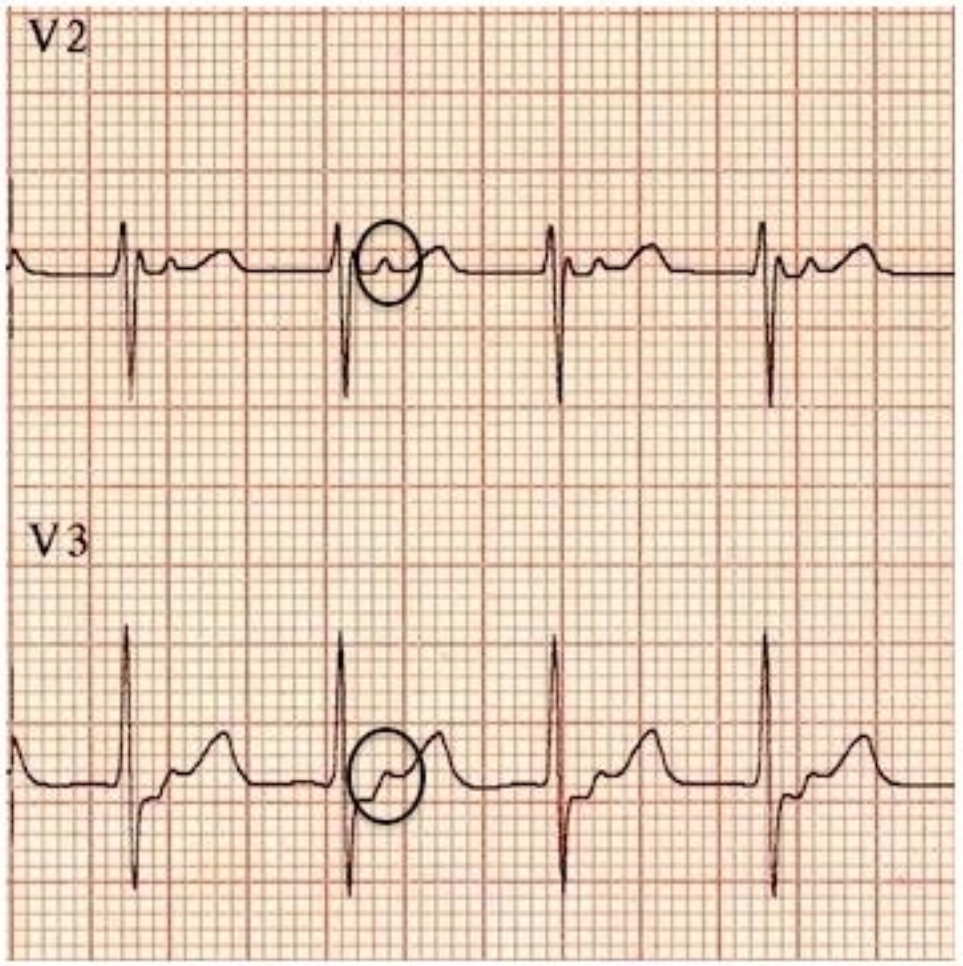
Pseudo R' waves in V1-2


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Fast-Slow AVNRT

Narrow complex Tachycardia

Retrograde P waves are visible after each QRS complex



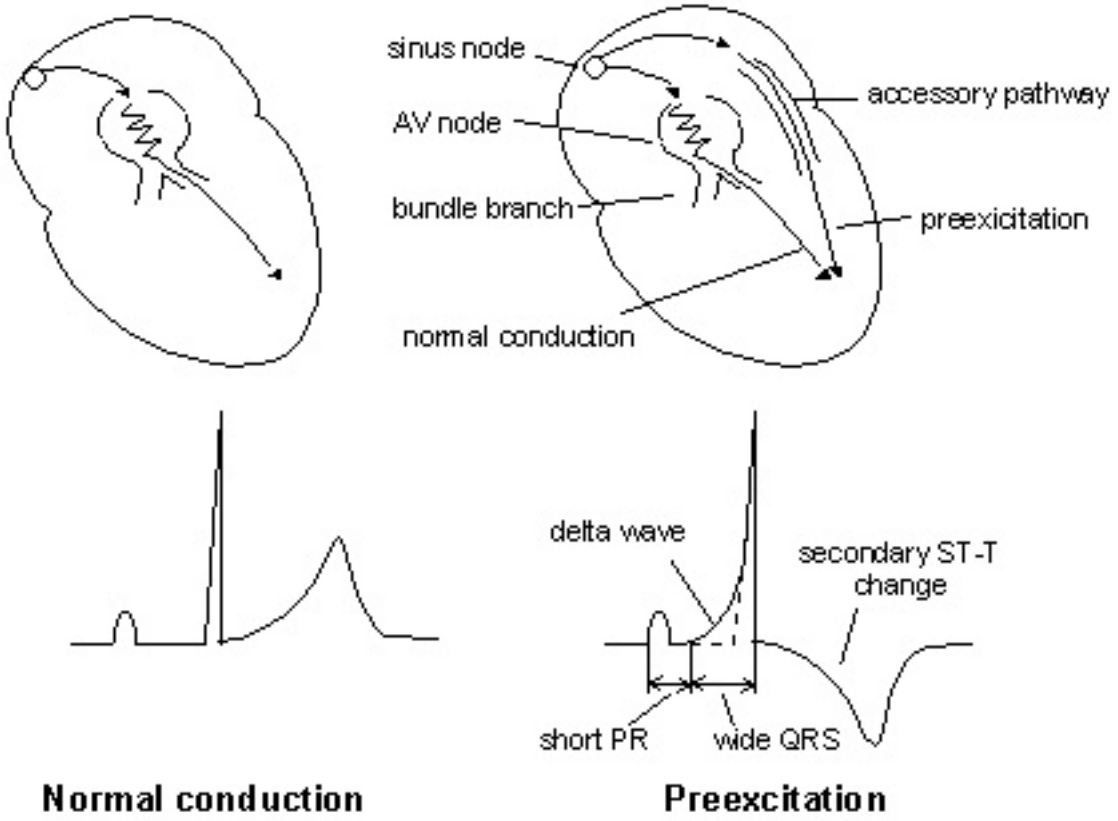
Retrograde P waves

Pre-Excitation & Accessory Pathways

- Activation of the ventricles due to impulse bypassing the AV node via an accessary pathway
- Abnormal conduction pathways
- Impulses conduct either antergrade towards the ventricle or retrograde, away or in both directions
- Majority conduct in both directions
- Reentry circuit involving accessary pathways termed Atrioventricular reentry tachycardias (AVRT)

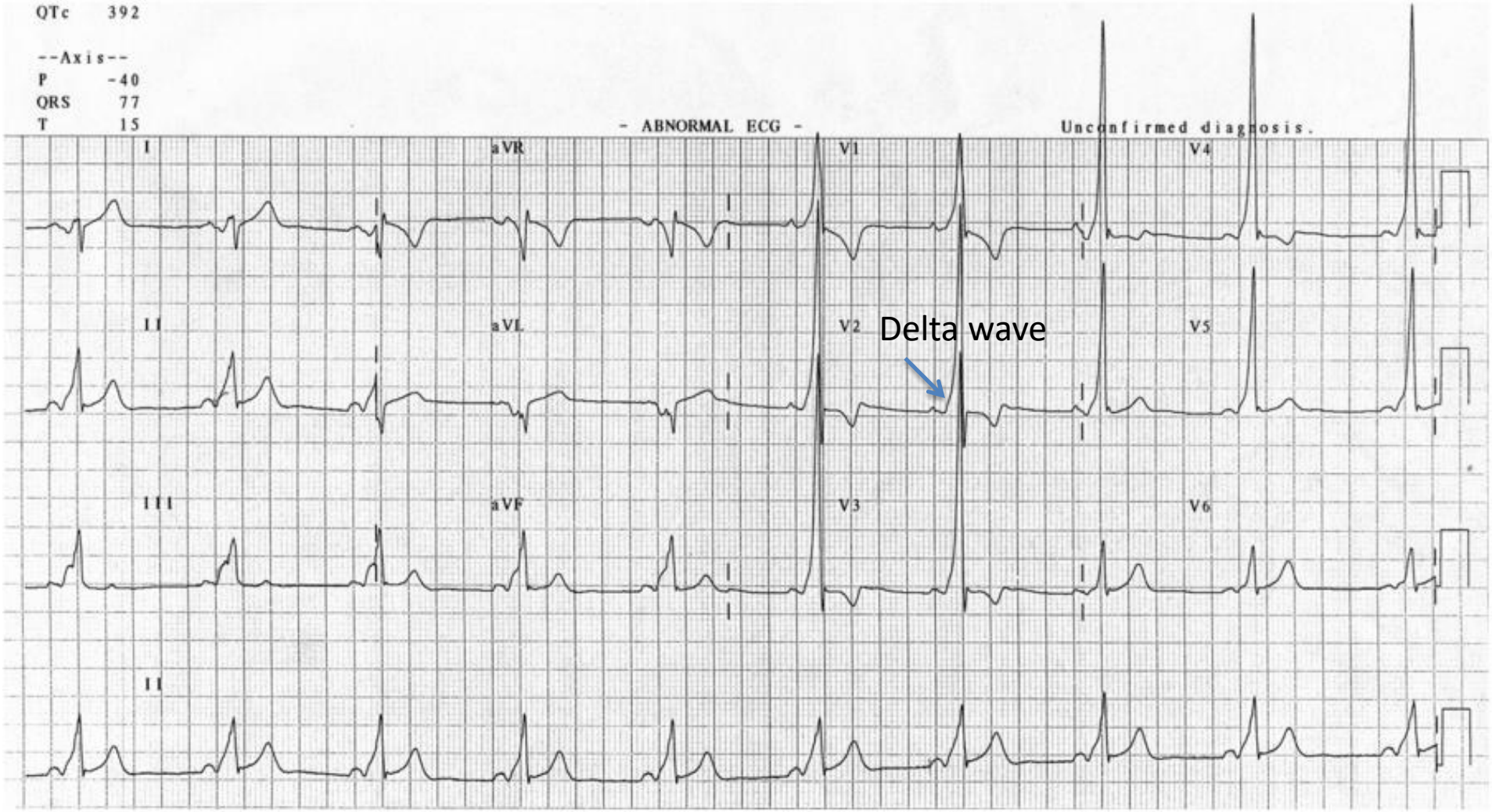
Wolf Parkinson White (WPW)

- PR interval < 120 ms
- Delta wave: slurring slow rise of initial portion of the QRS
- QRS prolongation > 110 ms
- ST segment and T wave discordant changes
- Pseudo-infraction pattern can be seen in up to 70% of patients (pseudo q waves, or prominent R wave in V1-V3 mimicking posterior infarction)


sinus node
AV node
bundle branch
normal conduction
accessory pathway
preexicitation
delta wave
secondary ST-T change
short PR
wide QRS
Normal conduction
Preexcitation

Other Pre-Excitation Syndromes

- Lown-Ganong-Levine (LGL) syndrome
  - Accessary pathway composed of *James Fibres*
  - ECG
    - PR interval < 120 ms
    - Normal QRS morphology
  - The term should not be used in the absence of paroxysmal tachycardia
  - Existence is disputed and may not exist



Sinus rhythm with a very short PR interval

Broad QRS with slurred upstroke (delta wave)

Dominant R wave V1

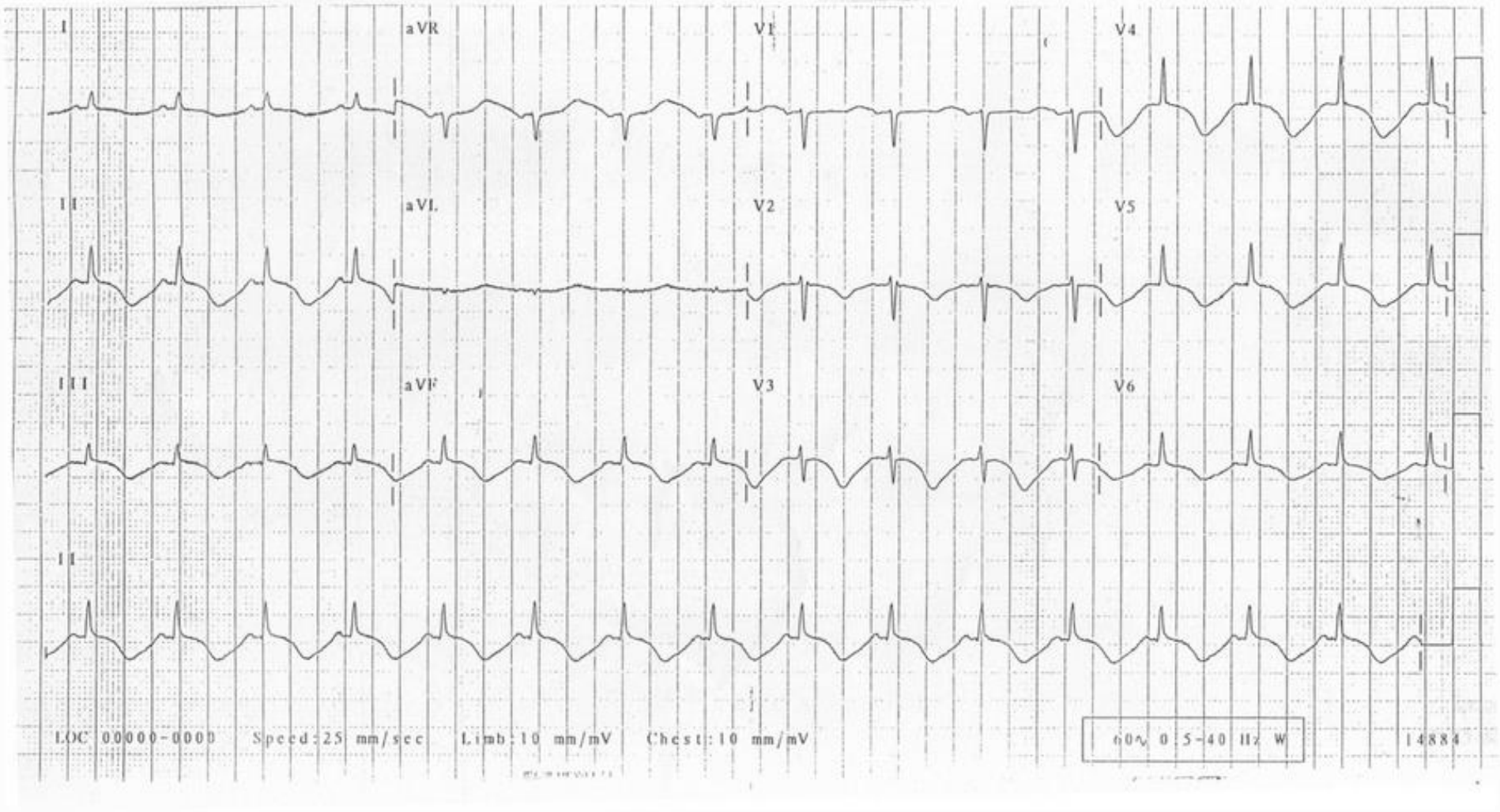
Tall R wave and inverted T wave in V1-3 mimicking RVH

Negative Delta wave in aVL (pseudo infarction pattern)

Common causes of QT Prolongation

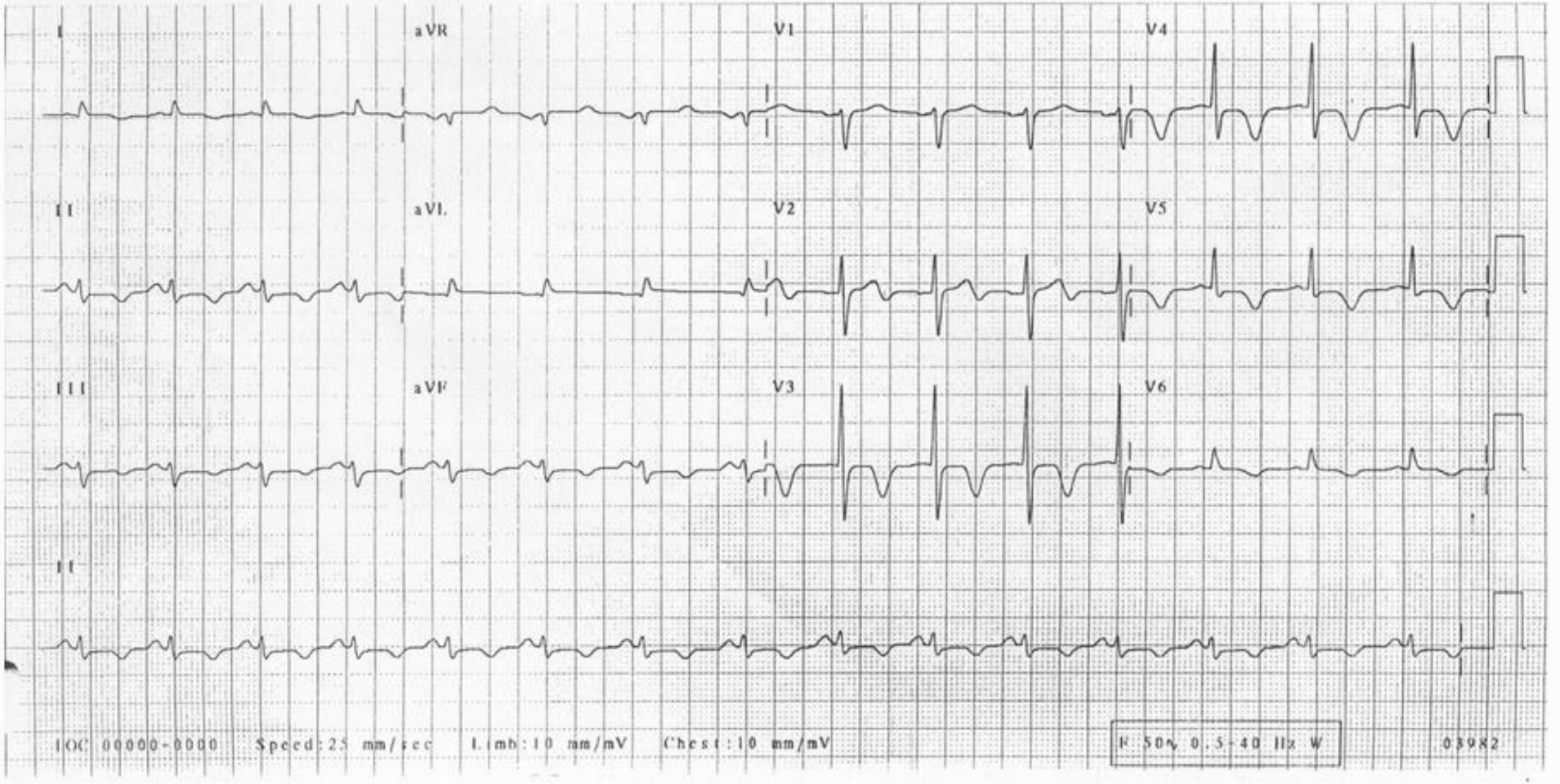
- Drugs
  - Type IA, III Antiarrythmic
  - Tricyclic antidepressants, Psychotropic meds (Haldol, methodone)
  - Phenothiazines
  - Macrolides
- Electrolyte disturbances
  - Hypokalelmia
  - Hypomagnesemia
  - Hypocalcaemia
- CNS disturbances
  - Stroke
  - ICB or Brainstem bleed
  - Coma

Example 2 – SAH



- Another example of cerebral T-waves with marked QT prolongation secondary to subarachnoid haemorrhage.

Example 3 – SAH



- Widespread T-wave inversions with slight ST depression secondary to subarachnoid haemorrhage.
- The QT interval is prolonged (greater than half the R-R interval).
- This ECG pattern could easily be mistaken for myocardial ischaemia as the T-wave morphology is very similar, although obviously the clinical picture would be very different (coma versus chest pain).

Common causes of ST Depression

- Ischemia
- Strain
- Digitalis effect
- Hypokalemia/hypomagnesemia
- Rate related changes
- Any combination of the above

Common causes of Tall R wave in V1

- WPW
- RBBB
- RVH
- Posterior MI
- Normal variant

Common causes of Nonspecific ST-T wave Abnormalities

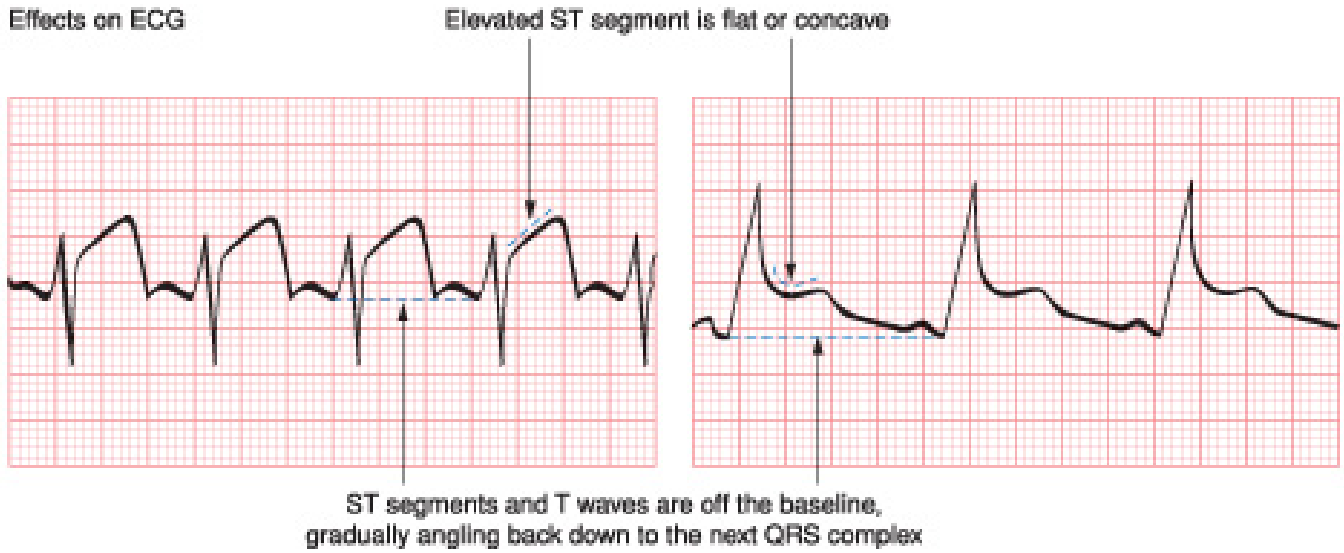
- Ischemia
- LVH
- Cardiomyopathy
- MVP
- Drug effect
- Lyte abnormalities
- CNS disorder
- Hyperventilation
- Severe medical illness
- Severe emotional stress
- Exercise
- Hypoxemia
- Acidosis
- Temp extremes
- Other causes

Other Cardiac Conditions

- Many conditions cause changes to the ECG
  - Electrolyte abnormality
  - Ischemia
  - Infarction
  - Inflammation
  - Medications

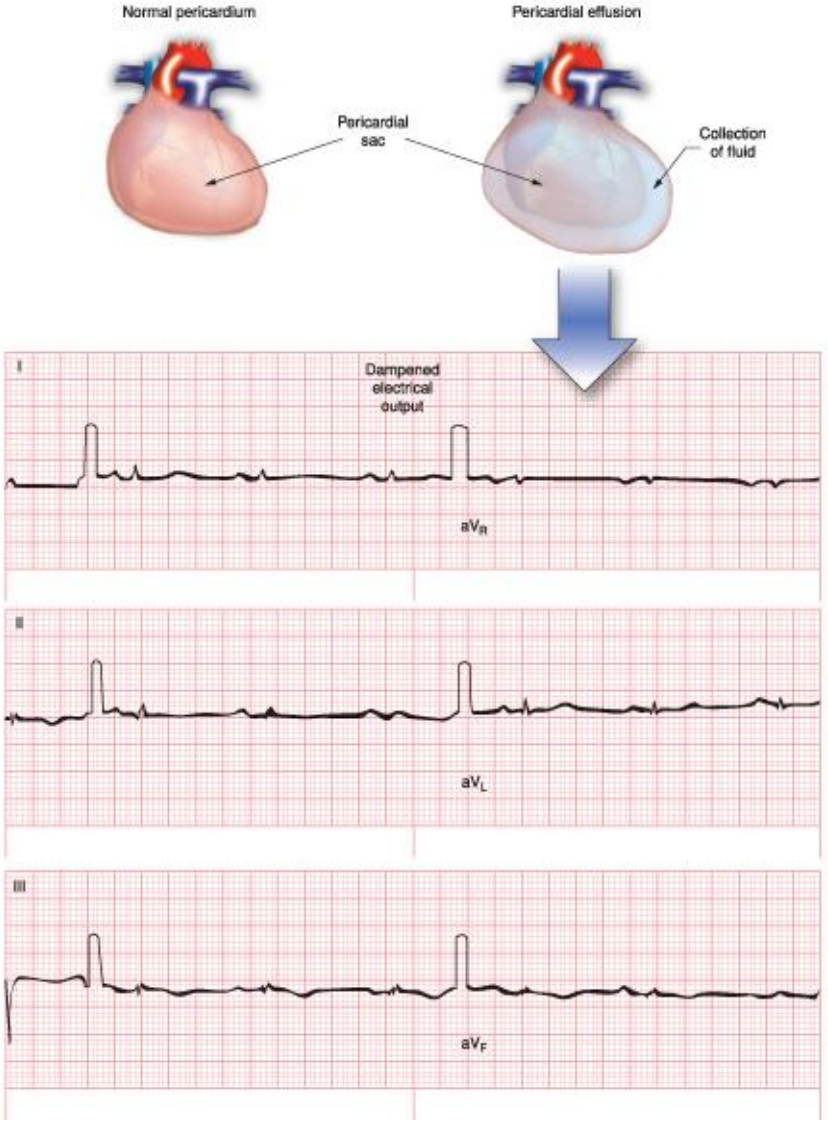
ECG Changes in Pericarditis

- T wave initially upright and elevated but then during recovery phase it inverts
- ST segment elevated and usually flat or concave



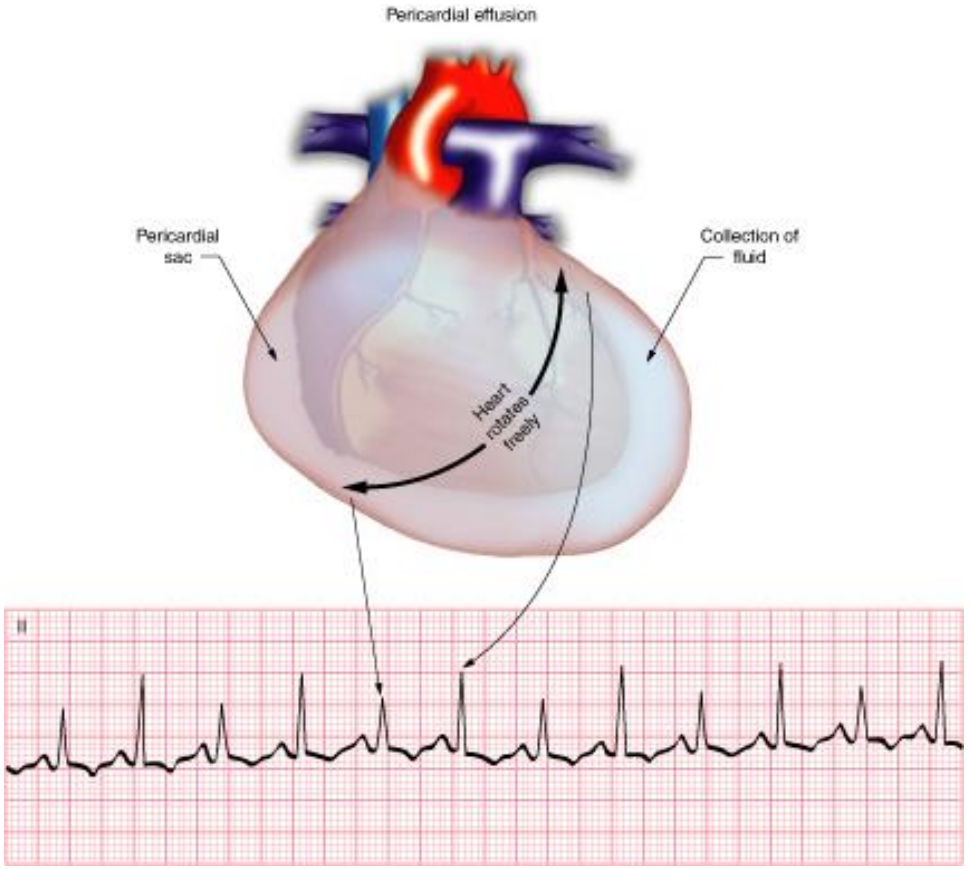
Pericardial Effusion

- Can occur with pericarditis
- Can cause low-voltage QRS complexes in all leads and electrical alternans



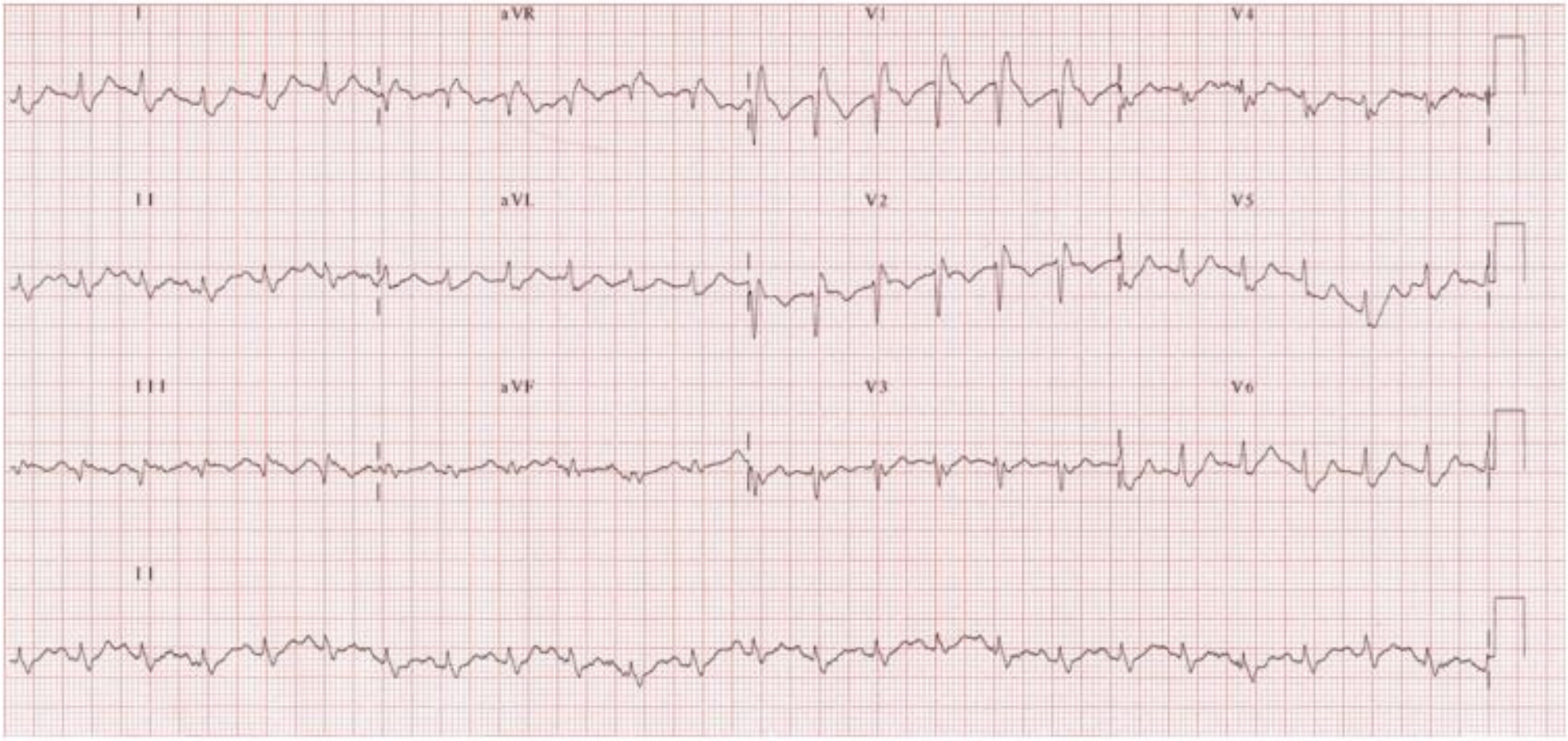
Electrical Alternans

- QRS complexes change in height with each successive beat



Pulmonary Embolism

- Acute blockage of one of the pulmonary arteries
- Leads to obstruction of blood flow to the lung segment supplied by the artery
- Produces large S wave in lead I, deep Q wave in lead III, inverted T wave in lead III
  - Called the *S1 Q3 T3* pattern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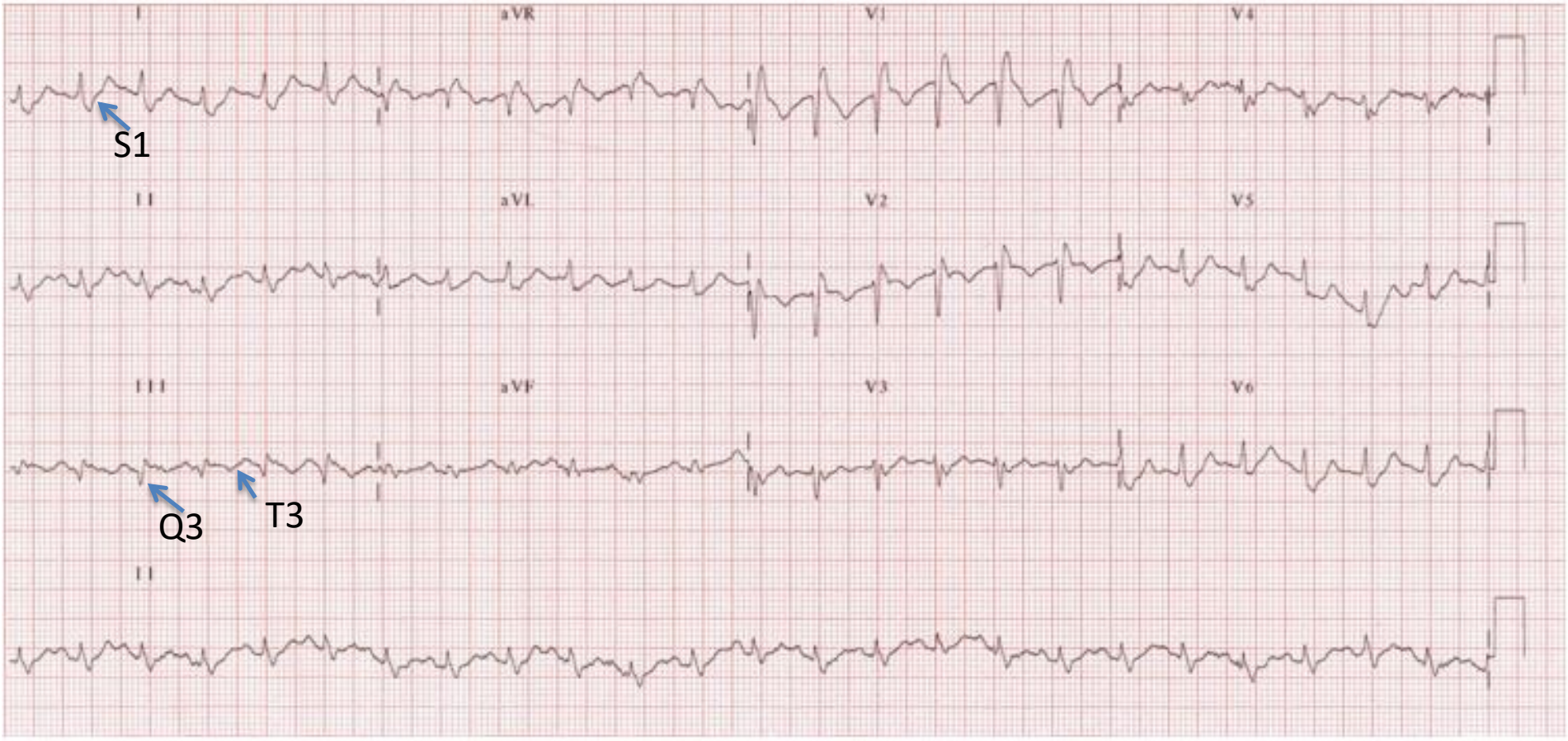
V3
V6
II

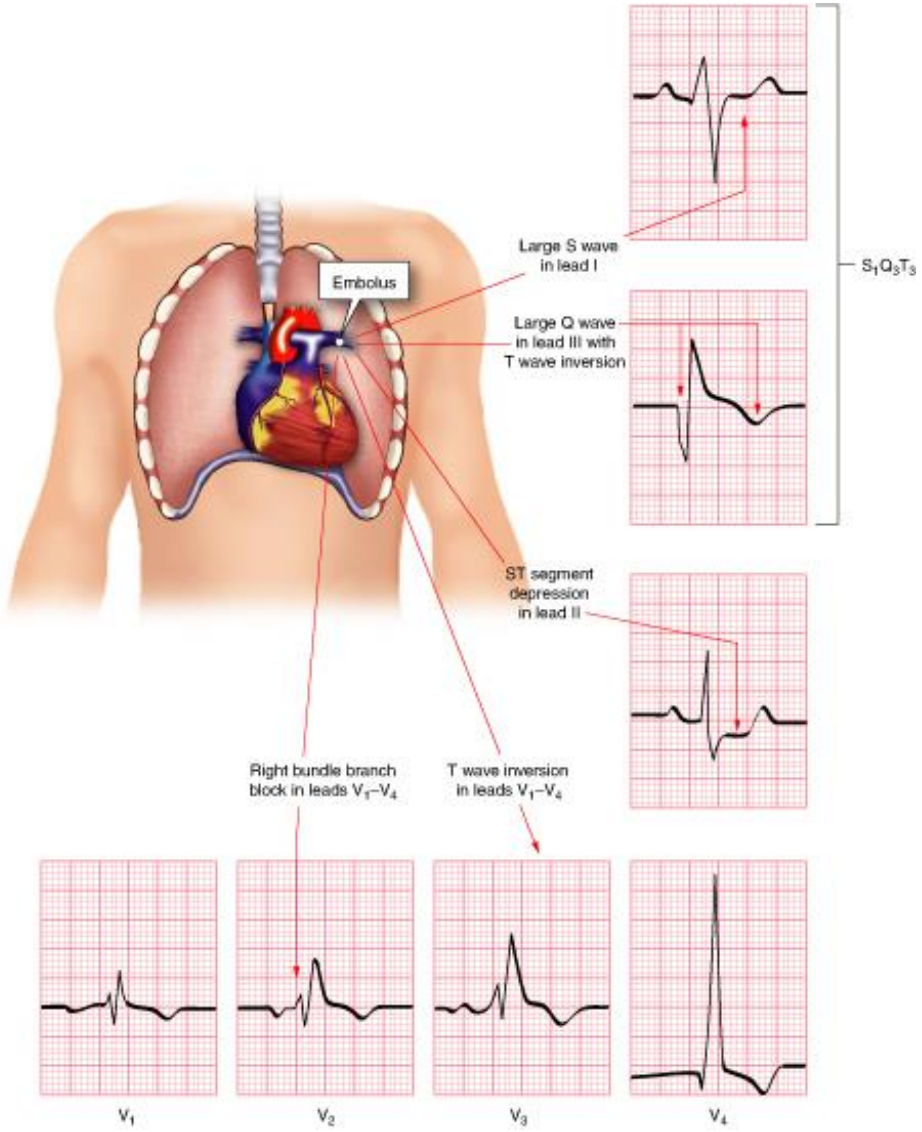


Sinus Tach

RBBB

T-wave inversions in right precordial leads (V1-3) as well Lead III

Pulmonary Embolism



Electrolyte Imbalances

- Increases or decreases in potassium and calcium serum levels can have a profound effect on the ECG

Hyperkalemia

- Key characteristics include:
  - T wave peaking
  - Flattened P waves
  - 1st-degree AV heart block
  - Widened QRS complexes
  - Deepened S waves
  - Merging of S and T waves

Definitions

- Hyperkalaemia is defined as a potassium level > 5.5 mEq/L
- Moderate hyperkalaemia is a serum potassium > 6.0 mEq/L
- Severe hyperkalaemia is a serum potassium > 7.0 mE/L

Effects Of Hyperkalaemia On The ECG

Serum potassium > 5.5 mEq/L is associated with **repolarization abnormalities**:

- Peaked T waves (usually the earliest sign of hyperkalaemia)

Serum potassium > 6.5 mEq/L is associated with **progressive paralysis of the atria**:

- P wave widens and flattens
- PR segment lengthens
- P waves eventually disappear

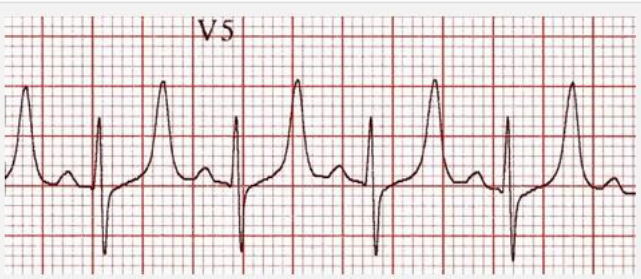
Serum potassium > 7.0 mEq/L is associated with **conduction abnormalities** and **bradycardia**:

- Prolonged QRS interval with bizarre QRS morphology
- High-grade AV block with slow junctional and ventricular escape rhythms
- Any kind of conduction block (bundle branch blocks, fascicular blocks)
- Sinus bradycardia or slow AF
- Development of a sine wave appearance (a pre-terminal rhythm)

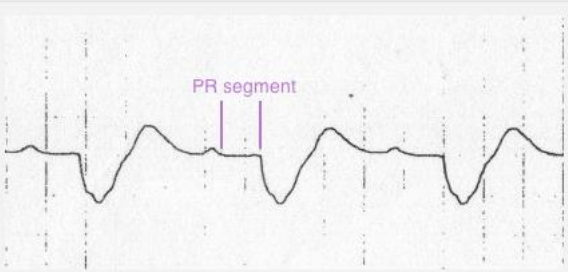
Serum potassium level of > 9.0 mEq/L causes **cardiac arrest** due to:

- Asystole
- Ventricular fibrillation
- PEA with bizarre, wide complex rhythm

Hyperkalemia

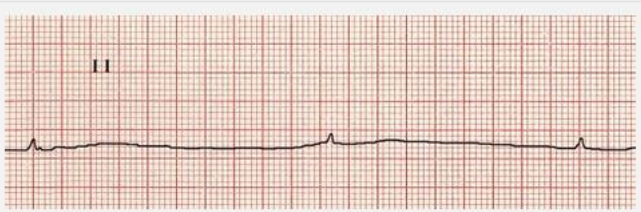


Peaked T waves

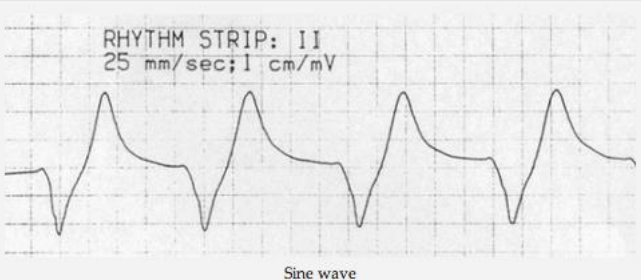


Prolonged PR segment

Loss of P waves



Bradycardia



Sine wave

Suspect Hyperkalemia

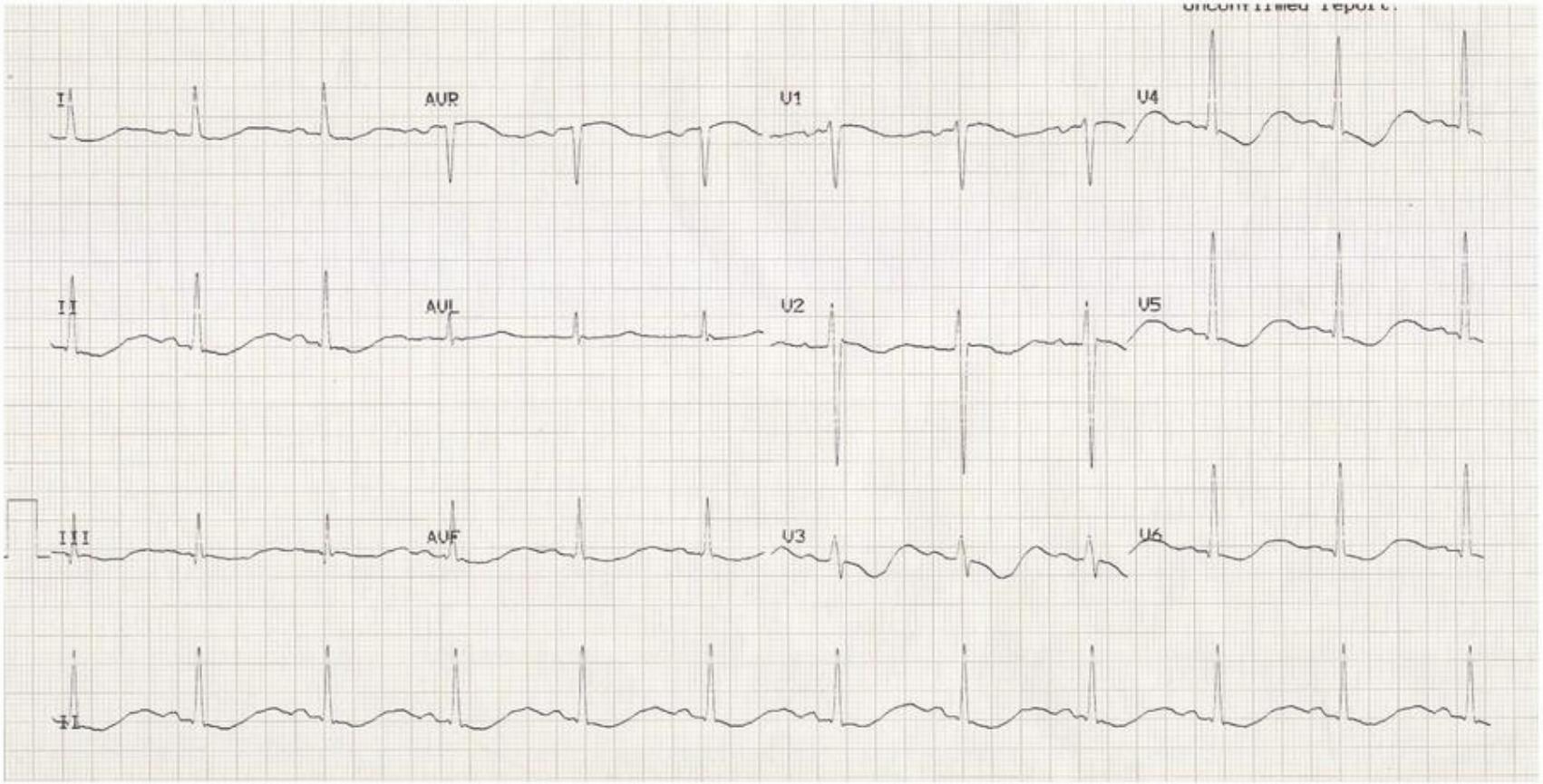
- New bradycardia

- New AV block especially with CKD or ESRD taking ACE-I or potassium sparing meds

Hypokalemia

- Key ECG characteristics include:
  - ST segment depression
  - Flattening of the T wave
  - Appearance of U waves

Example 1



Hypokalaemia:

- ST depression.
- T wave inversion.
- Prominent U waves.
- Long QU interval.

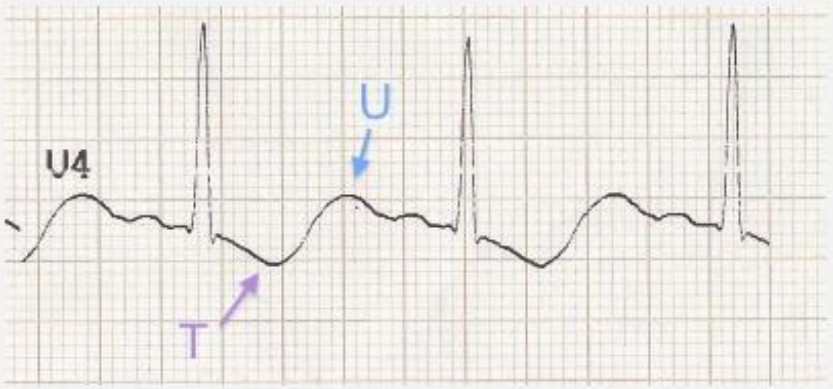
This patient had a serum K+ of 1.7

Handy Tips

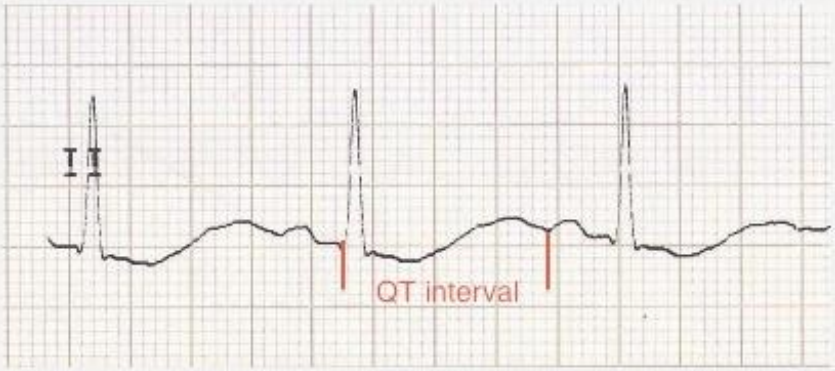
- Hypokalaemia is often associated with hypomagnesaemia, which increases the risk of malignant ventricular arrhythmias
- Check potassium and magnesium in any patient with an arrhythmia
- Top up the potassium to 4.0-4.5 mmol/l and the magnesium to > 1.0 mmol/l to stabilise the myocardium and protect against arrhythmias – this is standard practice in most CCUs and ICUs

With worsening hypokalaemia:

- Frequent supraventricular and ventricular ectopics
- Supraventricular tachyarrhythmias: AF, atrial flutter, atrial tachycardia
- Potential to develop life-threatening ventricular arrhythmias, e.g. VT, VF and Torsades de Pointes



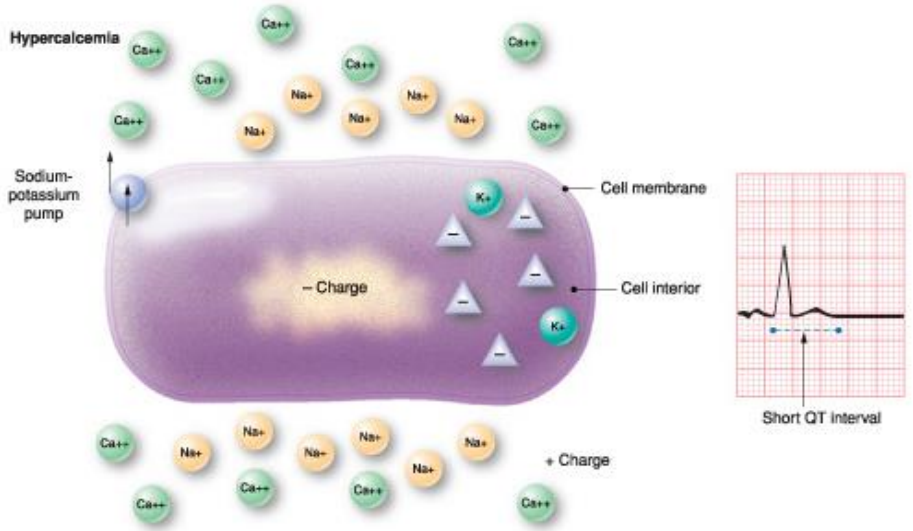
T wave inversion and prominent U waves in hypokalaemia

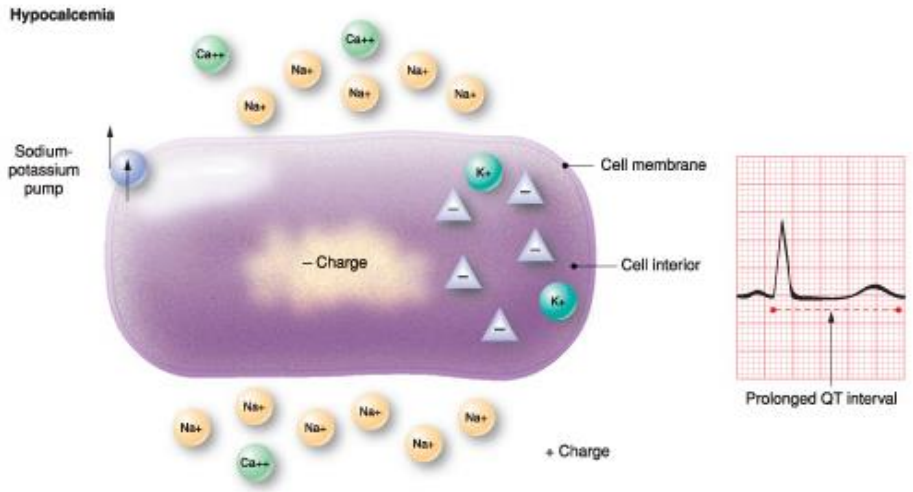


Apparent long QT interval with hypokalaemia (actually T-U fusion)

Hypocalcemia

- QT interval slightly prolonged





Definitions

- Normal serum corrected calcium = 2.2 – 2.6 mmol/L.
- Mild-moderate hypocalcaemia = 1.9 – 2.2 mmol/L.
- Severe hypocalcaemia = < 1.9 mmol/L.

Causes

- Hypoparathyroidism
- Vitamin D deficiency
- Acute pancreatitis
- Hyperphosphataemia
- Hypomagnesaemia
- Diuretics (frusemide)
- Pseudohypoparathyroidism
- Congenital disorders (e.g. DiGeorge syndrome)
- Critical illness (e.g. sepsis)
- Factitious (e.g. EDTA blood tube contamination)

Symptoms

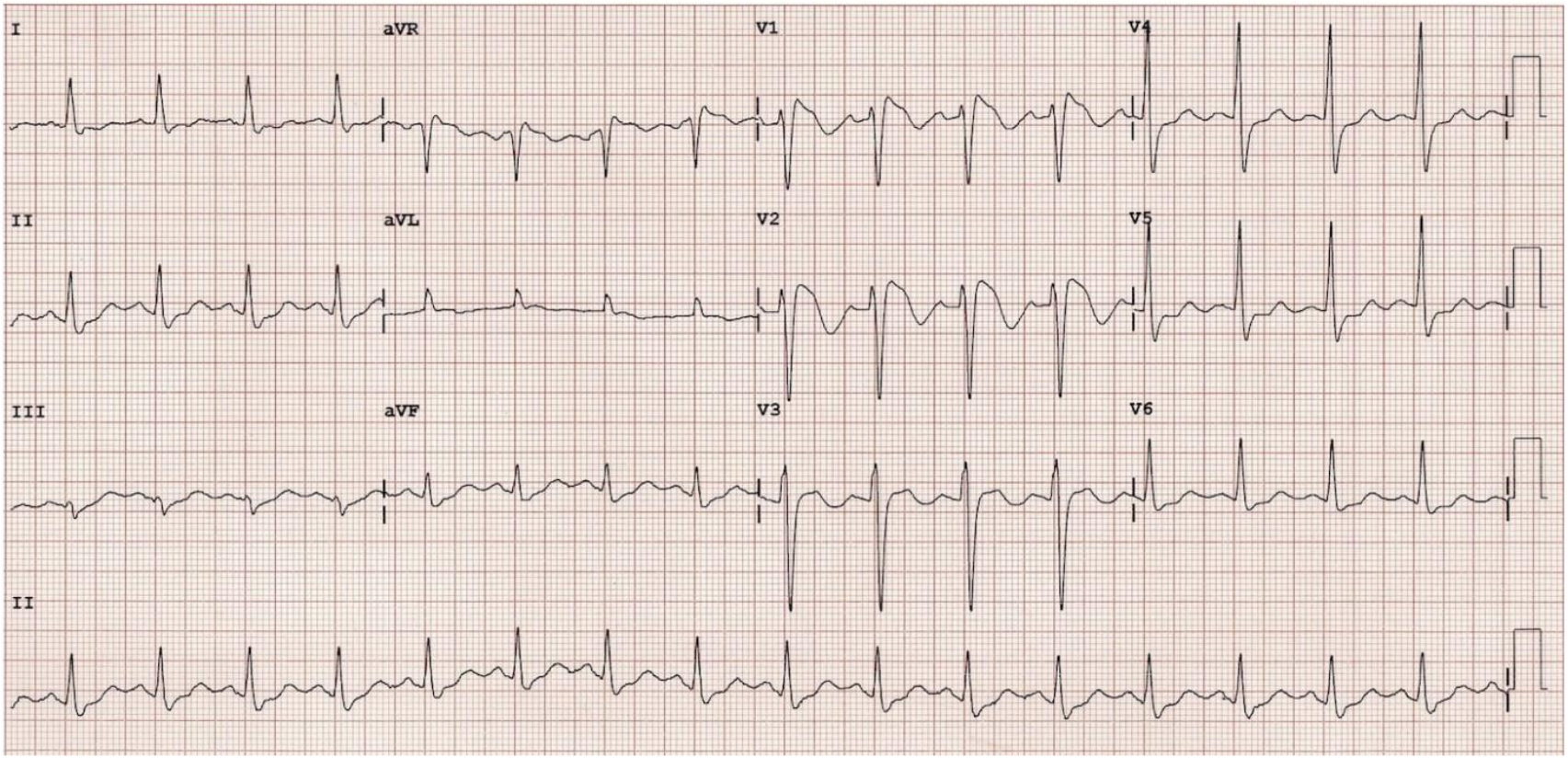
- Neuromuscular excitability
- Carpopedal spasm
- Tetany
- Chvostek's sign
- Trousseau's sign
- Seizures

ECG Changes

- Hypocalcaemia causes QTc prolongation primarily by prolonging the ST segment.
- The T wave is typically left unchanged.
- Dysrhythmias are uncommon, although atrial fibrillation has been reported.
- Torsades de pointes may occur, but is much less common than with hypokalaemia or hypomagnesaemia.

Hypocalcaemia

Hypocalcaemia causing a long QTc (510ms)


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Brugada Syndrome

- There is really only one type of Brugada syndrome
- Diagnosis depends on Characteristic ECG finding and clinical criteria
- Further risk stratification is controversial
- Definitive treatment is an ICD
- Brugada sign in isolation is of questionable significance

Etiology of Brugada

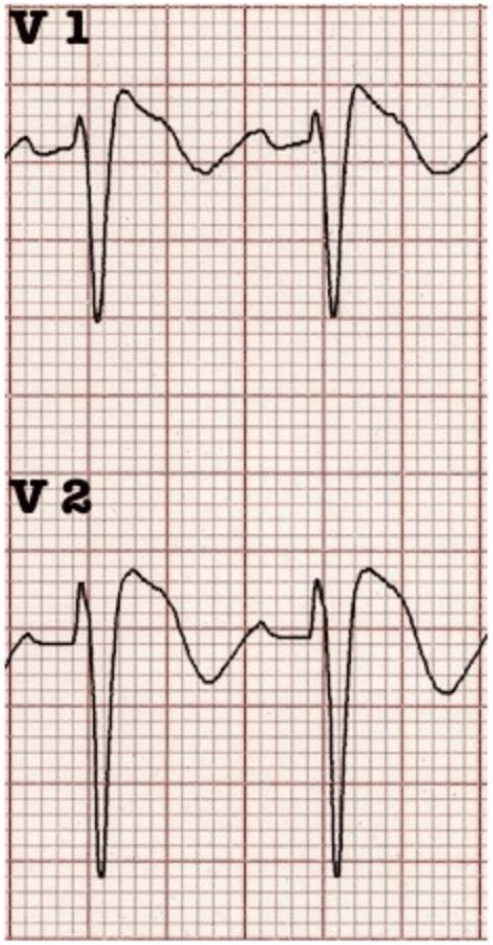
- Mutation in the cardiac sodium channel gene; often referred to as a sodium channelopathy
- Over 60 have been described with 50% being spontaneous mutations
- There is a familial clustering and autosomal dominant inheritance

Things that can unmask Brugada EKG changes

- Fever
- Ischemia
- Multiple drugs
  - Sodium channel blockers: flecainide, propafenone
  - CCB
  - Alpha agnoists
  - BB
  - Nitrates
  - Cholinergic stimulation
  - Cocaine
  - ETOH
  - Hypokalemia
  - Hypothermia
  - Post DCC

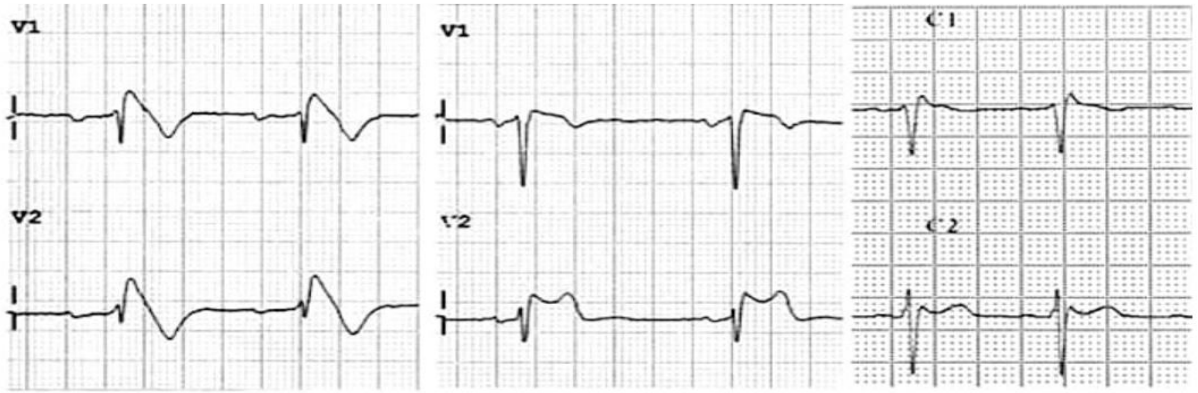
EKG Criteria for TYPE 1

- Coved ST Segment > 2mm in >1 of V1-V3 followed by a negative T wave
- Only EKG abnormality that is potentially diagnostic
- Referred to as a Brugada sign

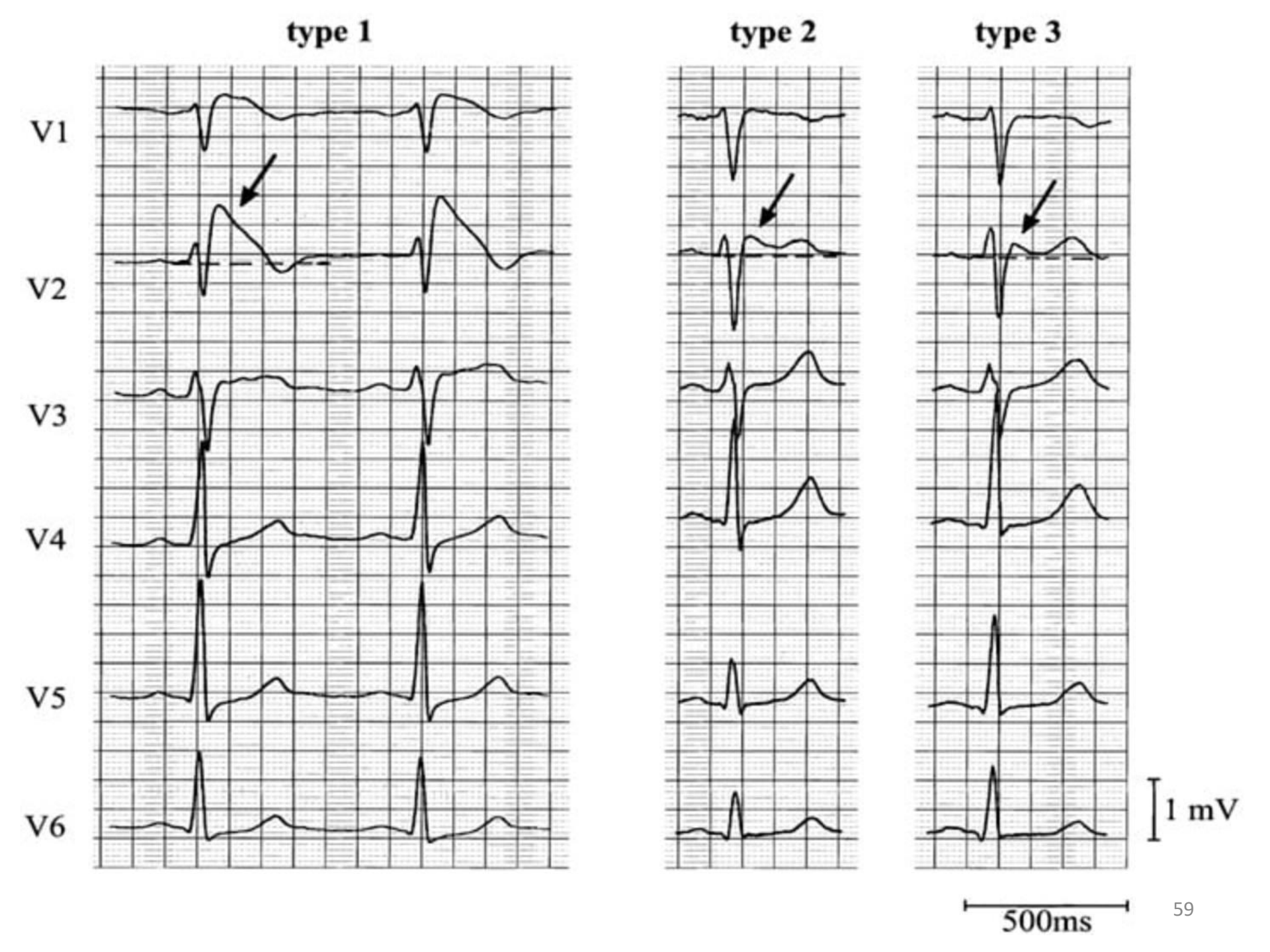


ECG associated clinical criteria

- Documented VF or Polymorphic VT
- Family history of SCD at < 45 yrs of age
- Inducibility of VT w programmed electrical stim
- Syncope
- Nocturnal agonal respiration



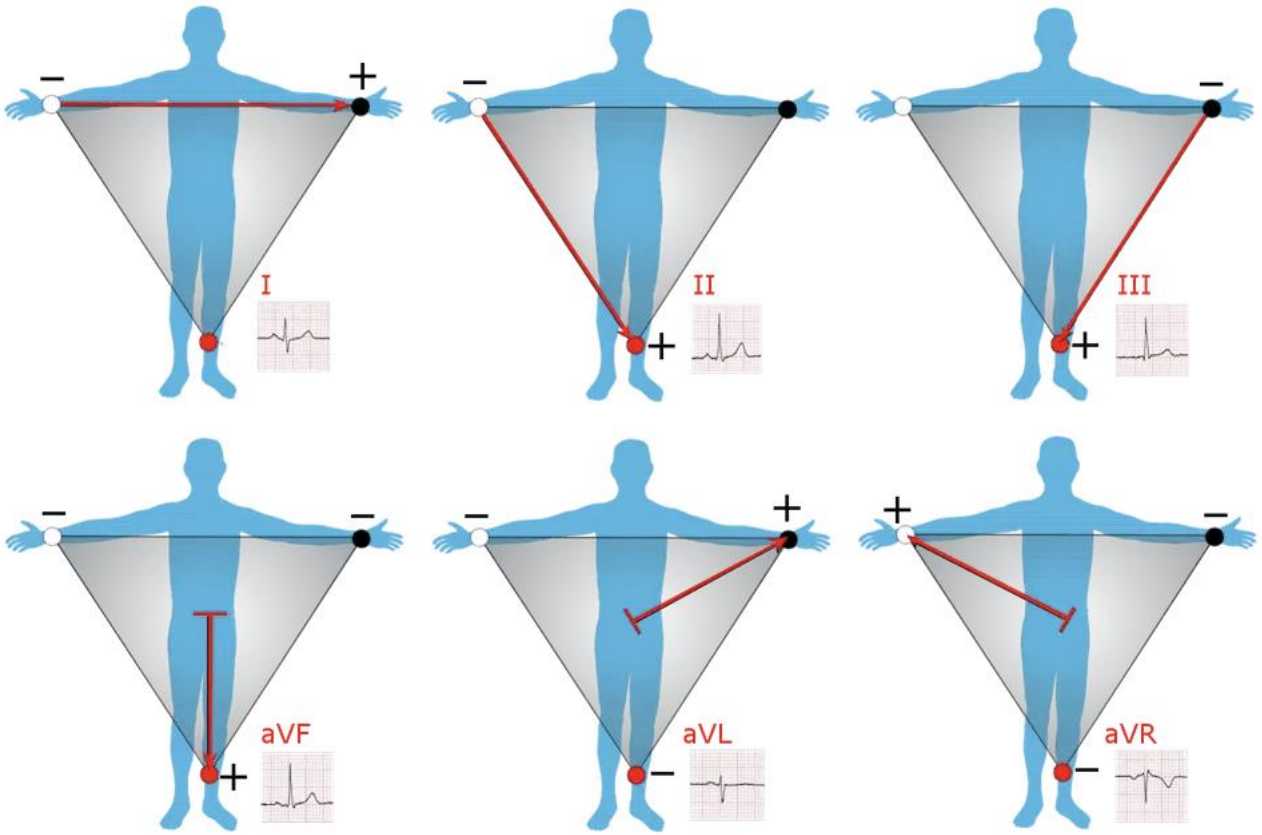
TYPE 1 ECG TYPE 2 ECG TYPE 3 ECG


type 1
type 2
type 3
V1
V2
V3
V4
V5
V6
1 mV
500ms

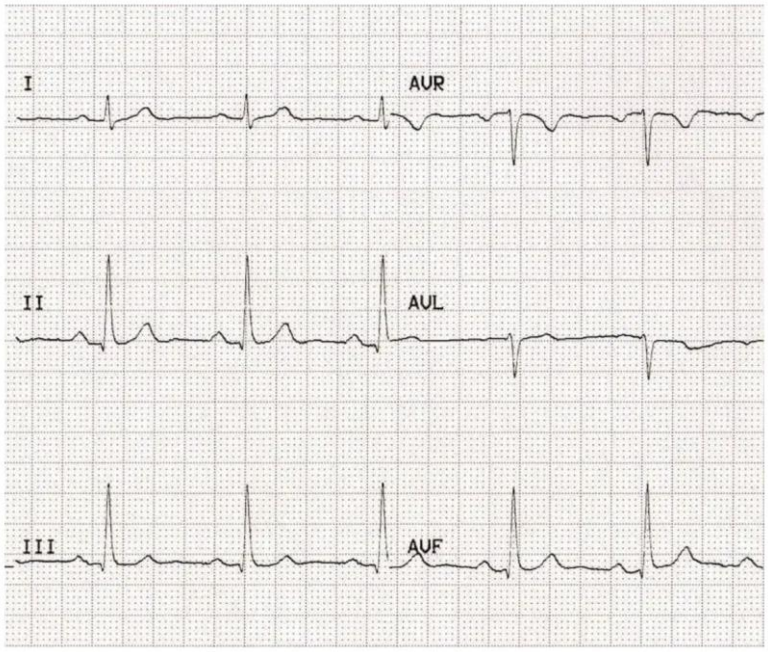
Limb reversal

- Various configurations can occur
- If you think the limbs are reversed, ask for a do over
- Know what a normal EKG looks like
- If possible, have an old EKG for comparison
- Does the pt look different or is complaining of cardiac/pulmonary issues?
- What are the VS?

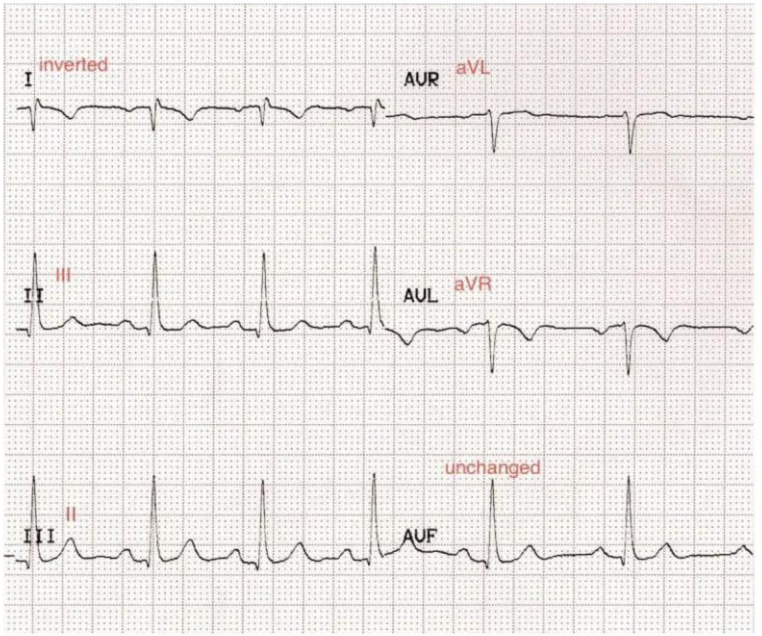
Normal Limb ECG Findings



LA/RA reversal

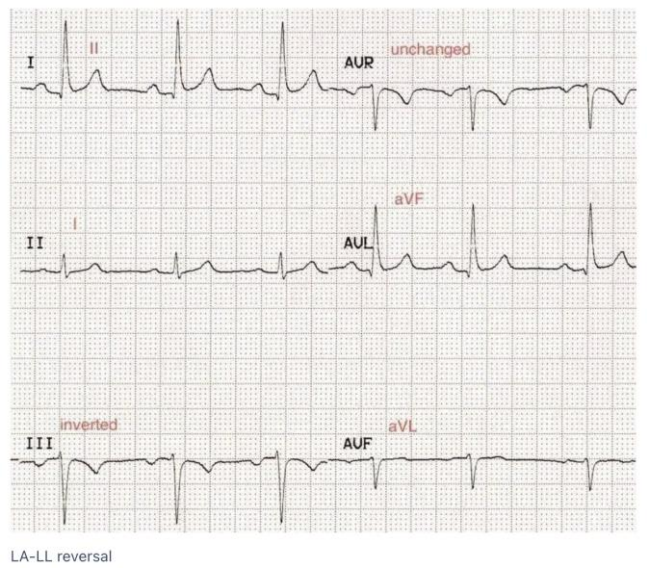


Baseline ECG

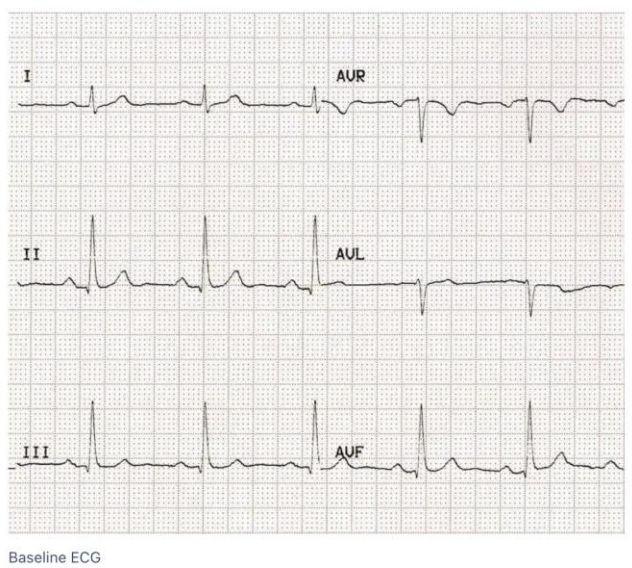


LA/RA reversal

LA/LL reversal

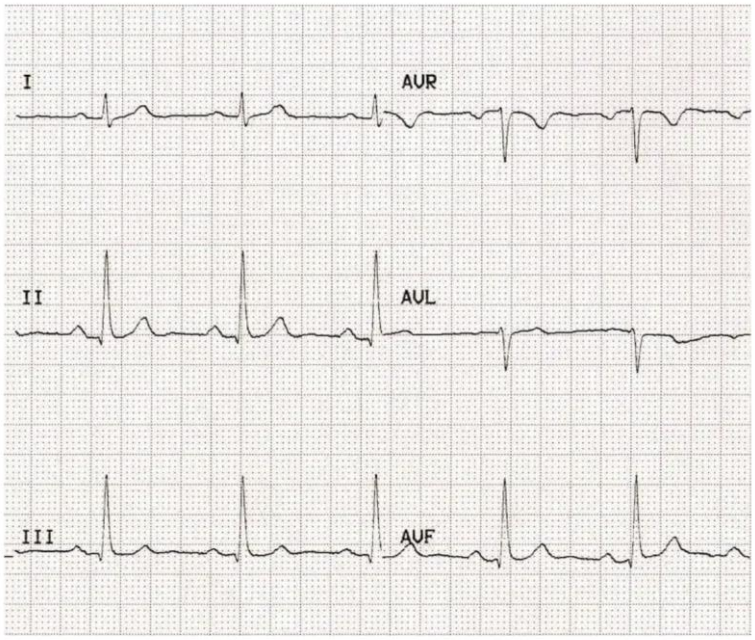


LA-LL reversal

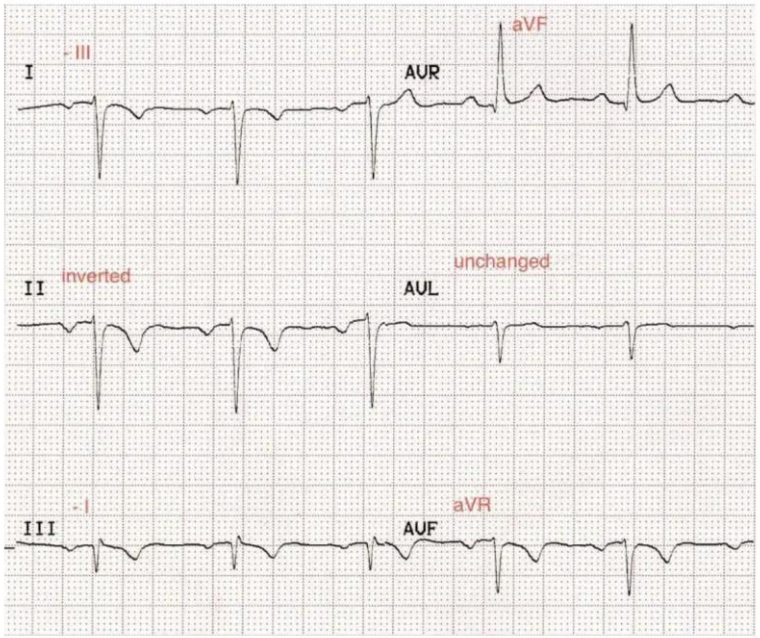


Baseline ECG

RA/LL Reversal

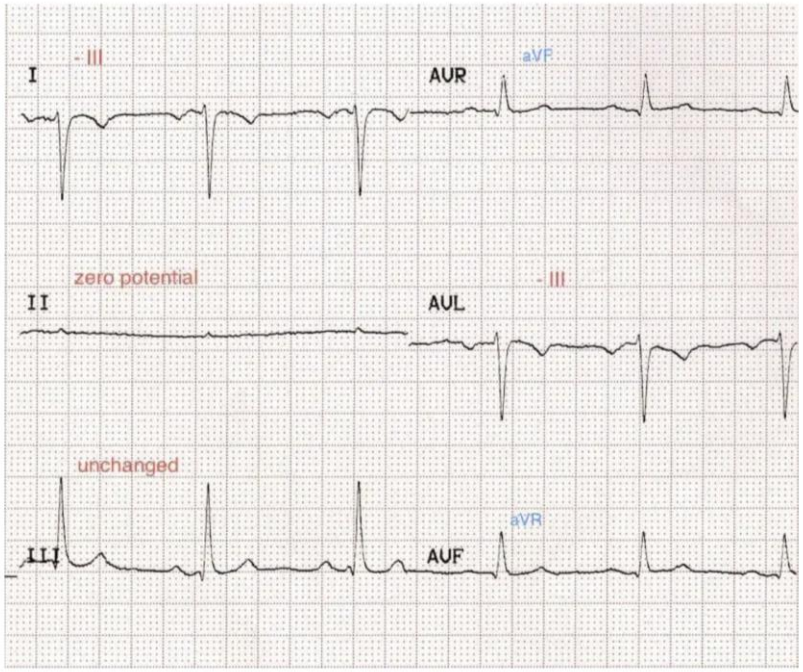


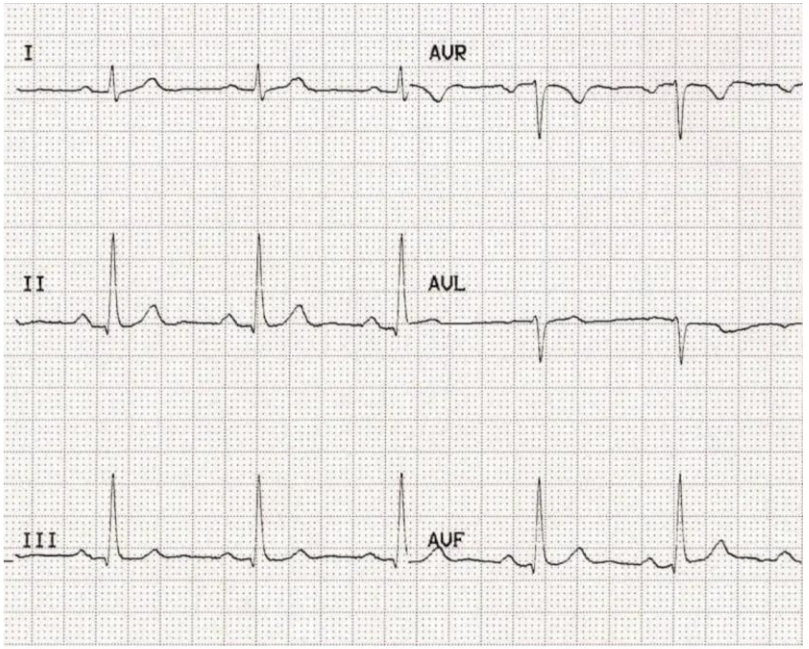
Baseline ECG



RA/LL reversal

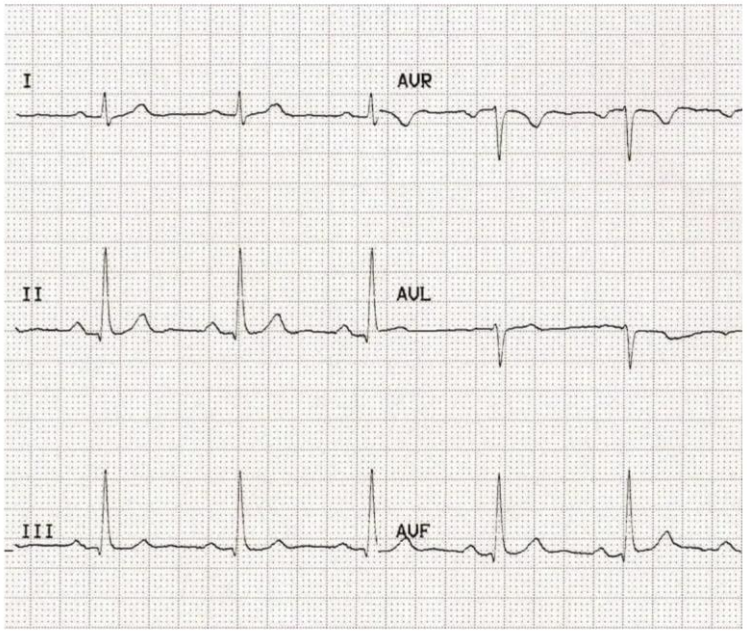
RA/RL reversal



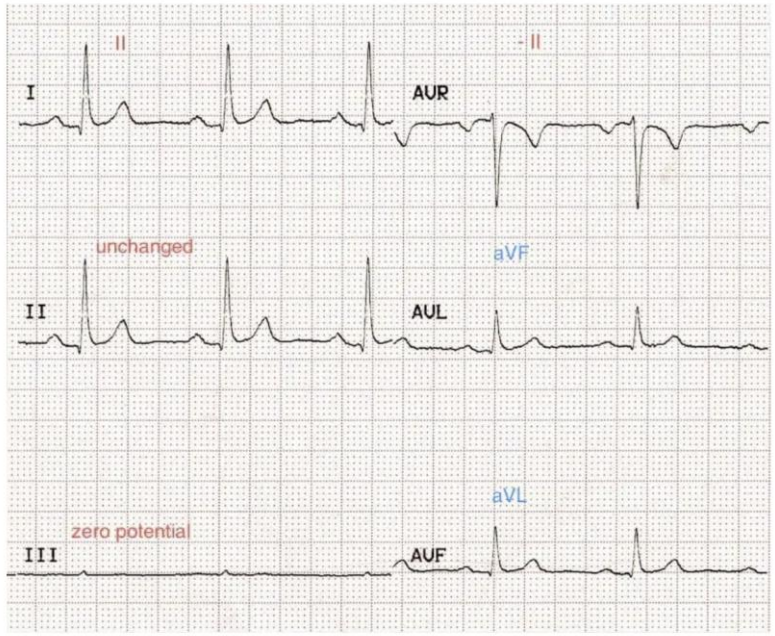


Baseline ECG

LA/RL reversal

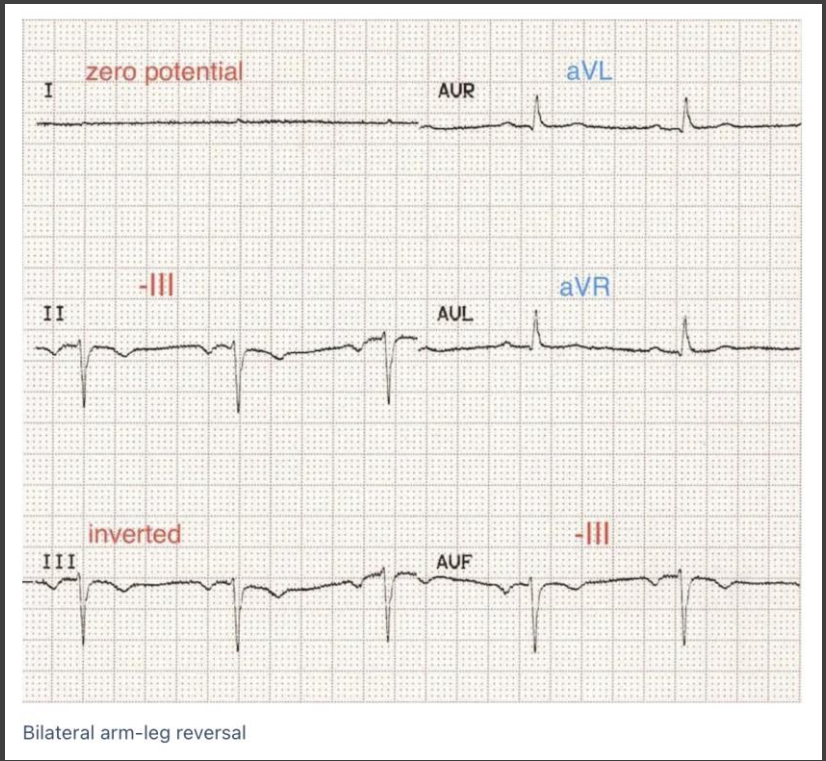


Baseline ECG

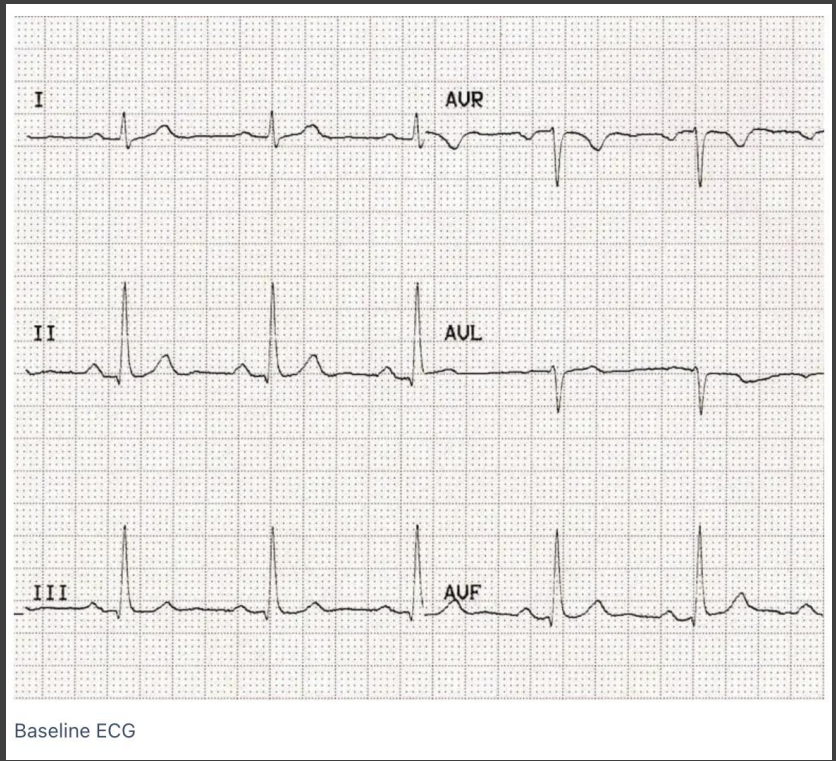


LA/RL(N) reversal

Bilateral Arm-Leg Reversal (LA-LL plus RA-RL)

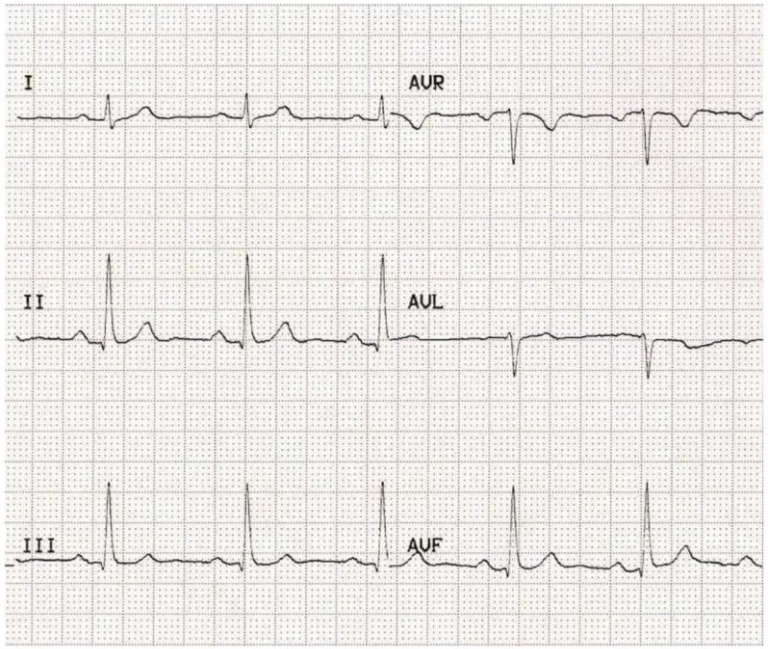


Bilateral arm-leg reversal

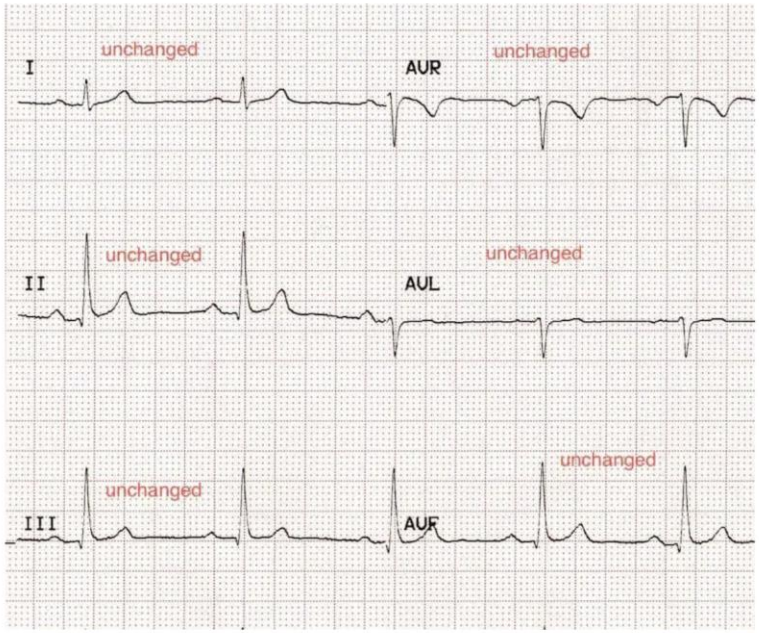


Baseline ECG

LL/RL reversal



Baseline ECG

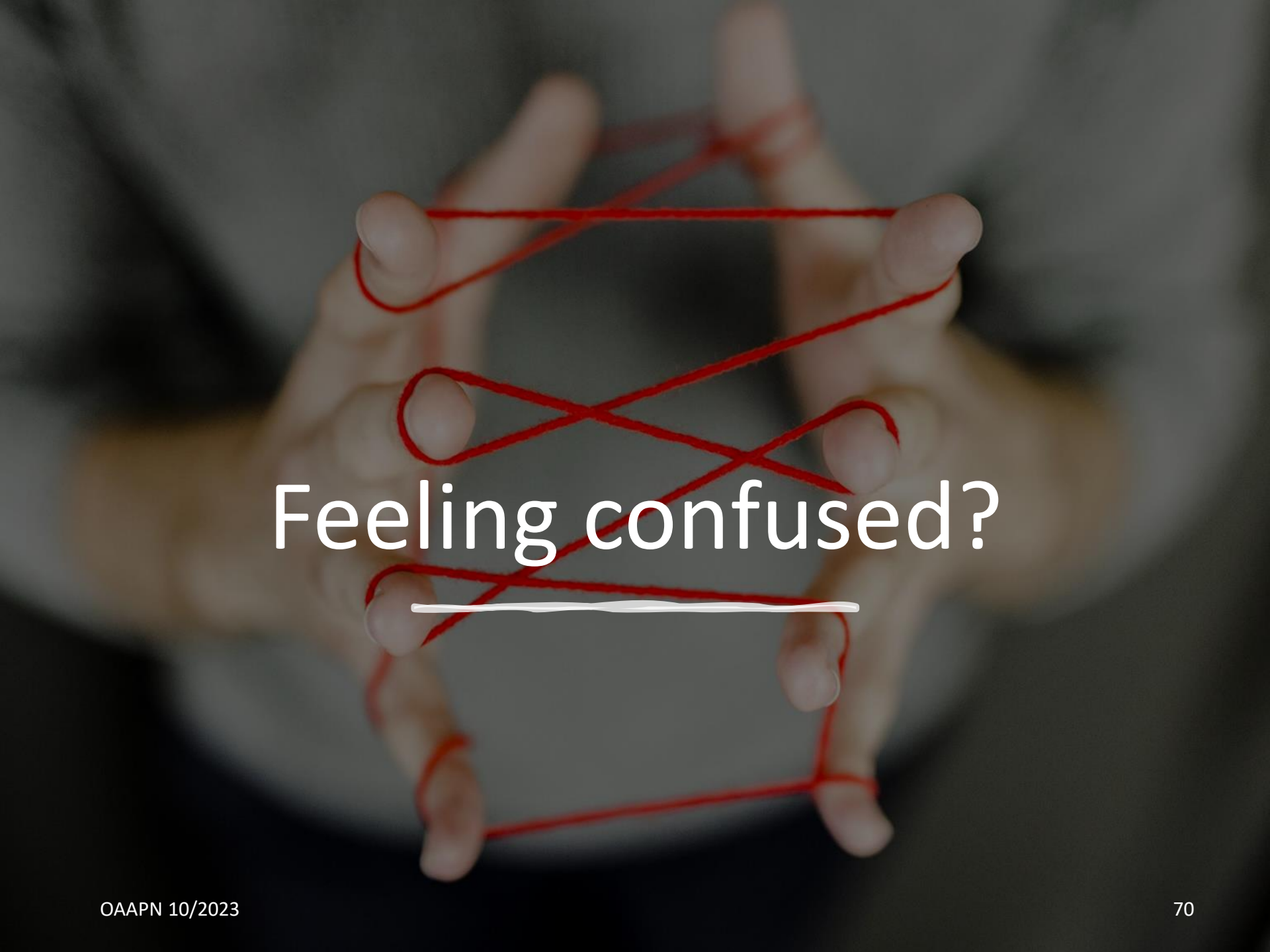


LL/RL(N) reversal

Summary
The KISS Principle

"True" lead Reversal	I	II	III	aVR	aVL	aVF	V1– V6
LA / RA	- I	III	II	aVL	aVR	aVF	No change
LA / LL	II	I	- III	aVR	aVF	aVL	No change
RA / LL	- III	- II	-I	aVF	aVL	aVR	No change
Clockwise	III	- I	- II	aVL	aVF	aVR	No change
Anti-Clockwise	- II	- III	I	aVF	aVR	aVL	No change

- **RA**—right arm; **LA**—left arm; **LL**—left leg;
- **Clockwise rotation**: RA→LA→LL→RA;
- **Anti-clockwise rotation**: RA→LL→LA→RA.
- The (-) sign signifies that the respective lead is inverted


Feeling confused?

Practice makes improvement

Systematic approach

Compare	Compare with old ECG
Look	Look at Rate
Look	Look at Rhythm
Look	Look at Axis
Look	Look at Hypertrophy
Look	Look at I's and others •Intervals, ischemia, injury, infarct

Get some good Reference material

- Only EKG Book you'll ever need, 8th ed. Malcom S. Thaler, ISBN-13: 978-1-4511-9394
- A Practical Guide to ECG Interpretation: Ken Grauer, ISBN 0-8016-2159-3
- https://litfl.com/ecg-library/
- **EKG / ECG Interpretation - Reading a 12 lead electrocardiogram**
- https://ecg.bidmc.harvard.edu/maven/mavenmain.asp
- http://www.clinicalskills.pitt.edu/electrocardiogram-interpretation/

The End

- Thank you for listening
- Please contact me with questions

- Louann Bailey at Lbaileycrnp@outlook.com
- Happy EKG reading!