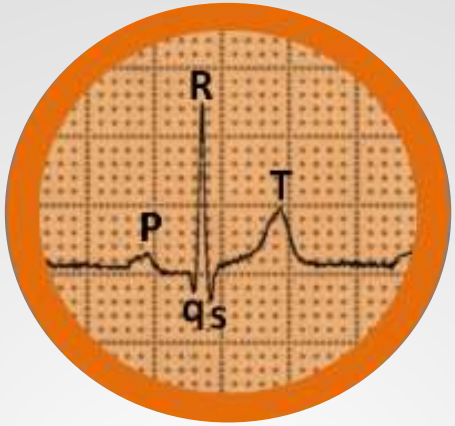

Life-Threatening ECGs in OPD

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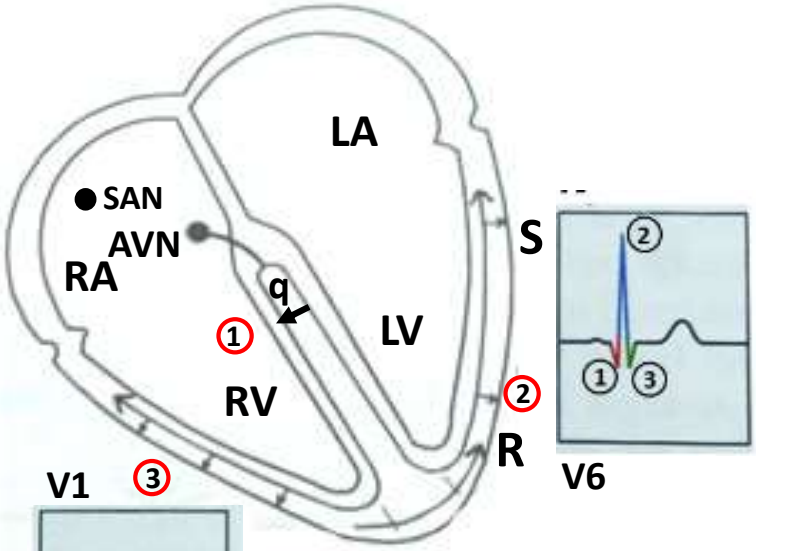
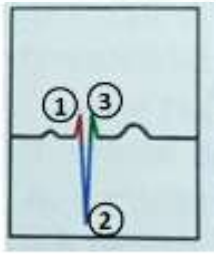
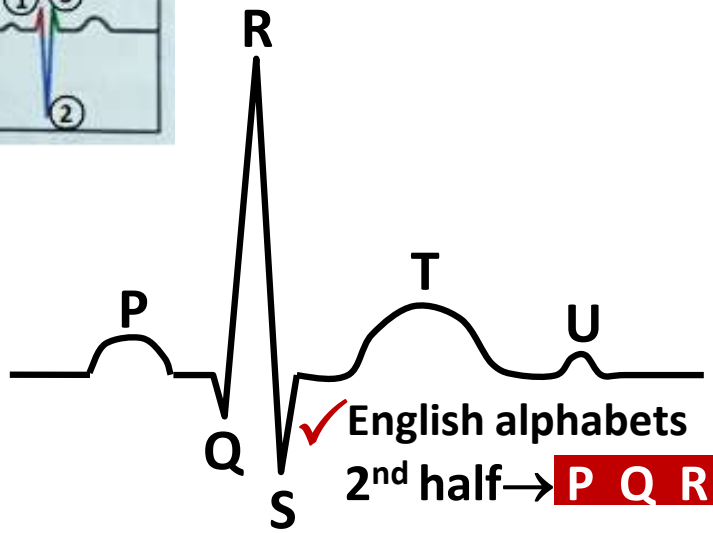
Past National President CCDSI (2020-21)
Immediate Past Dean ICCMD (2022-2023)
Awarded Prestigious Master Teacher Award from CCDSI in 2022
National Veteran Academic Excellence Award (IMA , 2023)
(100 Articles and 16 e-Books on different aspects of ECG to his credit)

Basics : Normal ECG

- Positive wave towards the flow of current
- Negative wave away from the flow of current



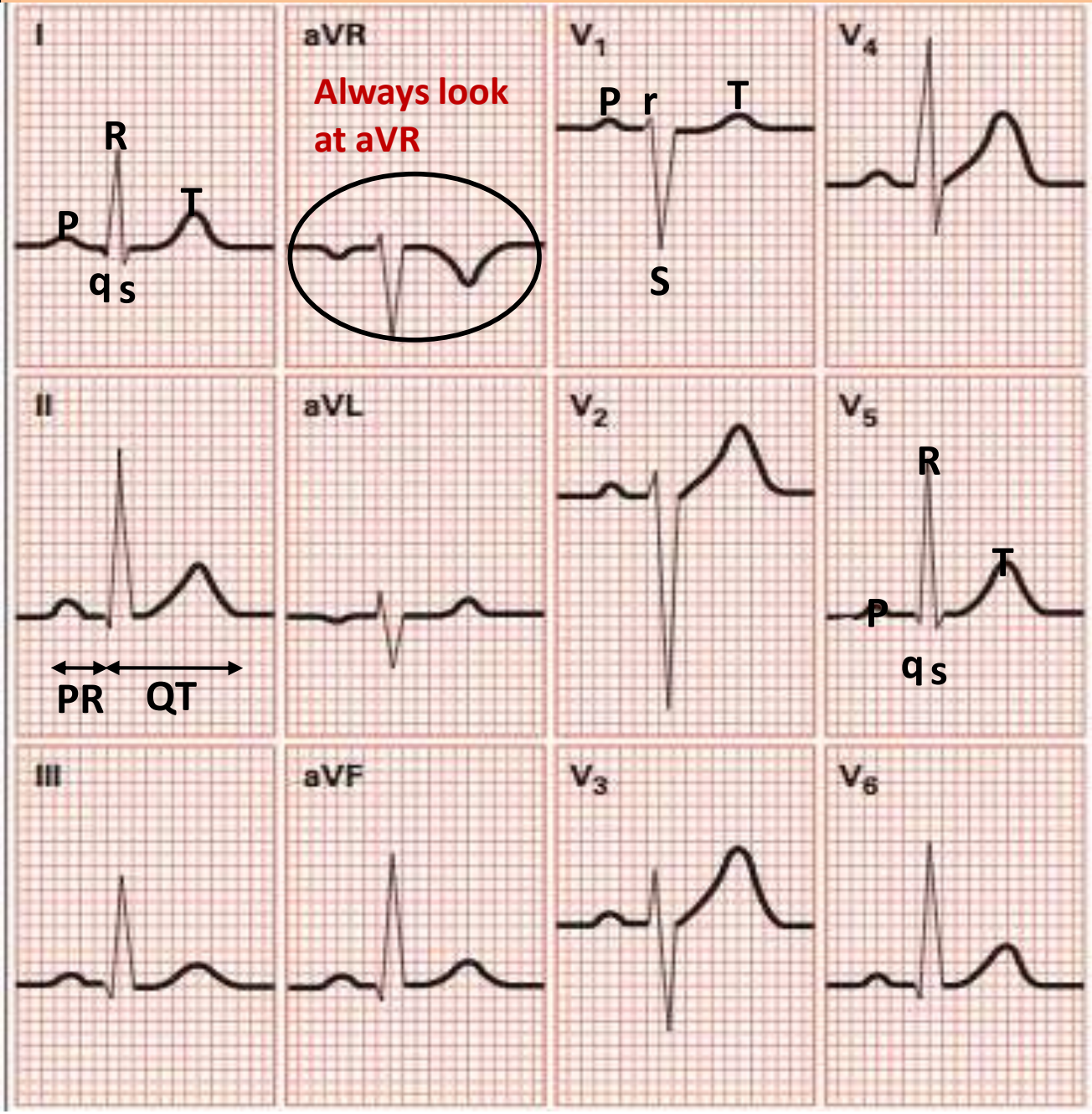




English alphabets

2nd half → P Q R S T U

Normal 12-ECG



Life-threatening ECGs at a glance

PR interval

- LGL syndrome
- WPW pattern
- Mahaim pathway

QRS

- HCM
- LVH as independent risk factor
- Fragmented QRS

ST-T

- Hyperacute T-wave – STEMI
- Occlusion MI : dWp , Wellens , Posterior STEMI , Aslanger
- Angina pectoris , Non-STEMI

✓QT↕

- Electrolytes – K , Ca
- Drugs
- Congenital – Short/long QT

Misc.

- Brugada syndrome
- Pul. Embolism, Peri. Effusion , Vent. Anneurysm , Spiked helmet sign , DCMP

Misc.

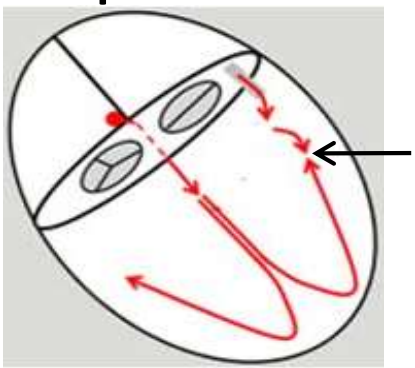
- Digitalis, Osborn wave
- Masquerading BBB
- Ventricular tachycardia

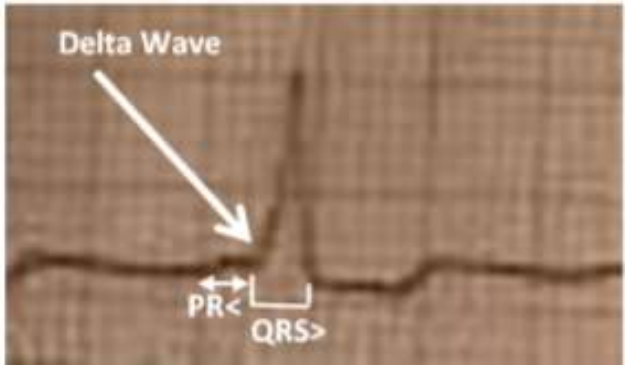
Pre-excitation syndrome

❑ LGL syndrome

Shortened PR interval with normal QRS

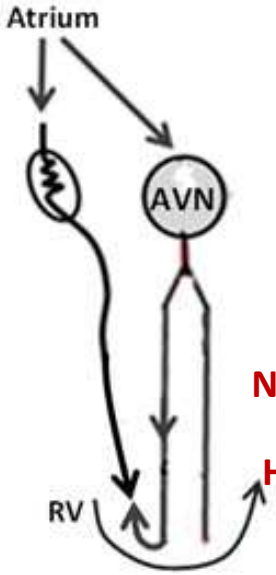
❑ WPW pattern



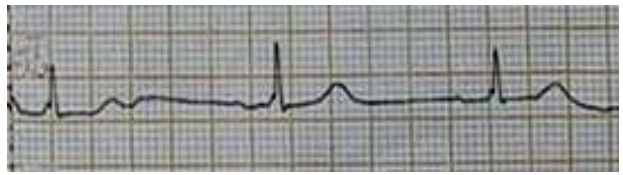


Shortened PR interval with **immediate delta wave** superimposed on QRS

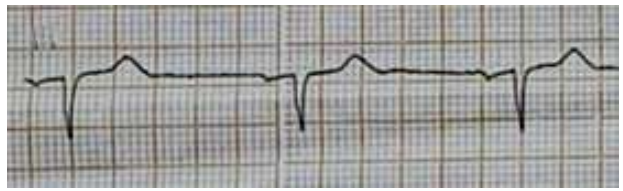
❑ Mahaim pathway



Normal PR interval
+
Half-hearted LBBB



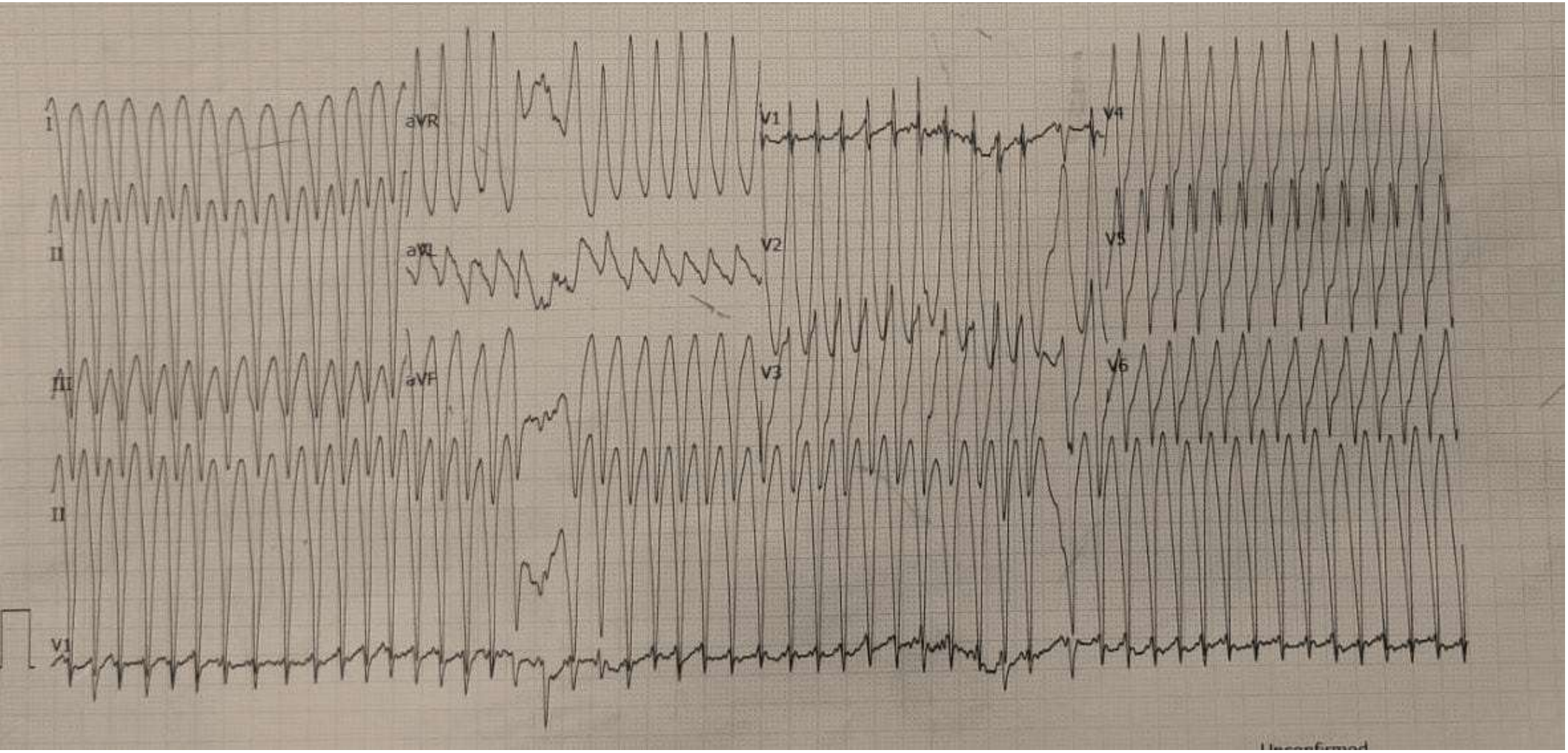
I , aVL, V5 V6 - No Septal W



rS pattern in lead III

<u>During sinus rhythm :</u>
Minimal ventricular pre-excitation
(Half hearted LBBB) =
No septal wave in I, aVL , V5 or V6 or the presence of rS pattern in lead III , delayed transitional shift

Pre-excitation atrial fibrillation

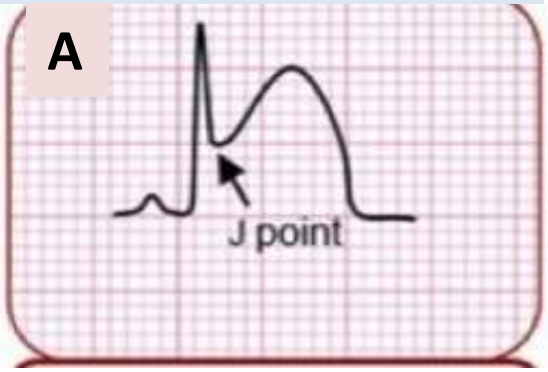


Escape of flooded atrial fibrillation to the opened gate of accessory pathway

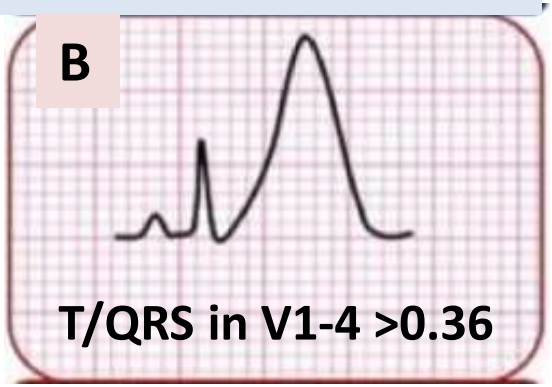
: the chances of atrial fibrillation becomes more with multi-accessory pathways

Life-threatening occlusion MI

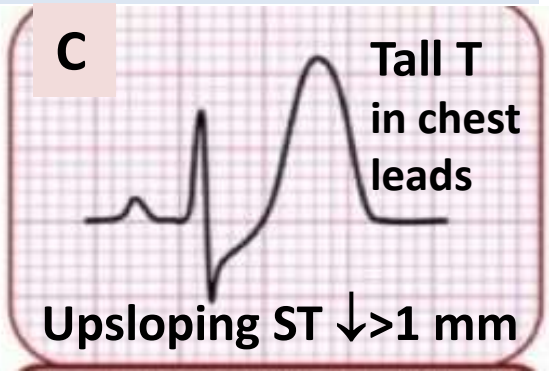
Conventional STEMI



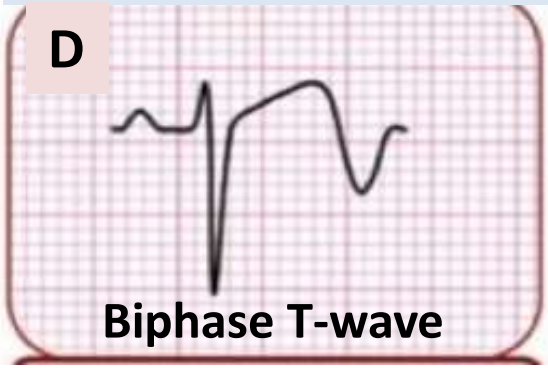
Hyperacute T-wave



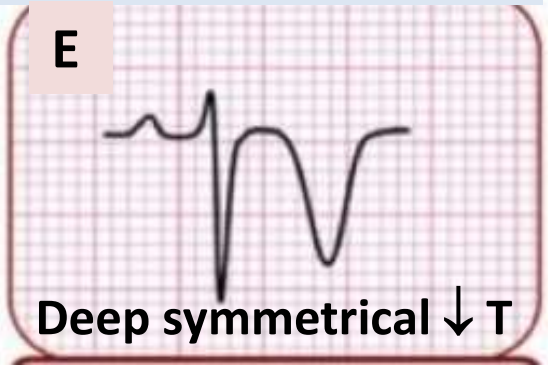
De Winter Syndrome



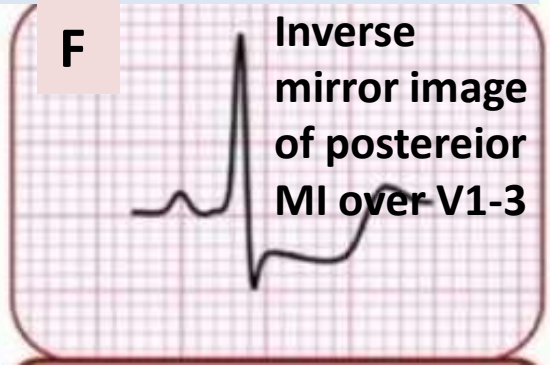
Wellens Sign A



Wellens Sign B

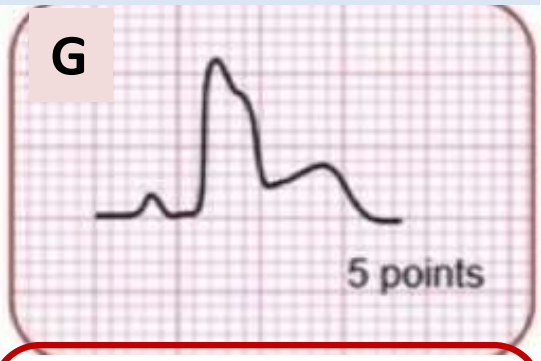


Posterior STEMI



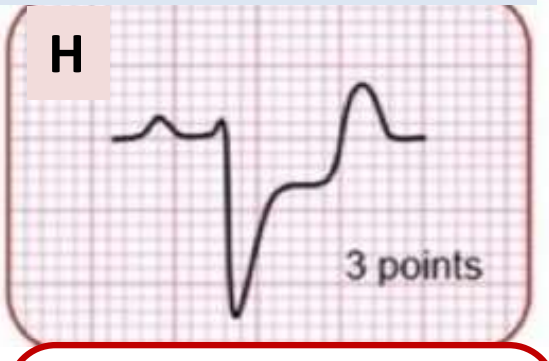
Occlusion MI (contd.)

Sgarbossa criterion 1



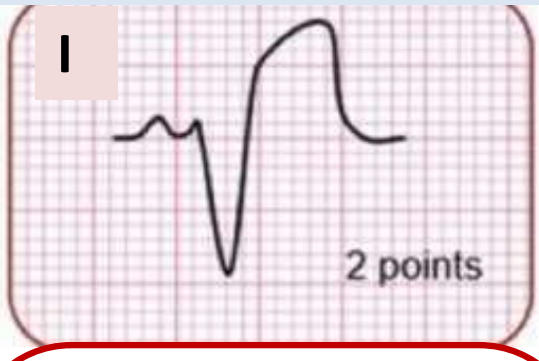
ST elevation > 0.1 mV concordant to the QRS in any of the leads I, aVL , V4 to V5

Sgarbossa criteria 2



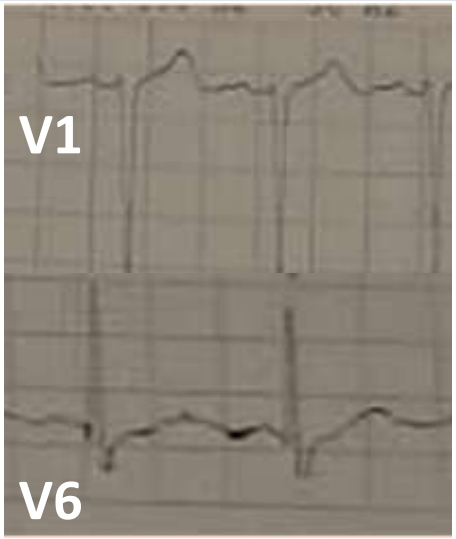
ST depression ≥ 0.1 mV concordant to the QRS in any of the leads V1 to V3

Sgarbossa criteria 3

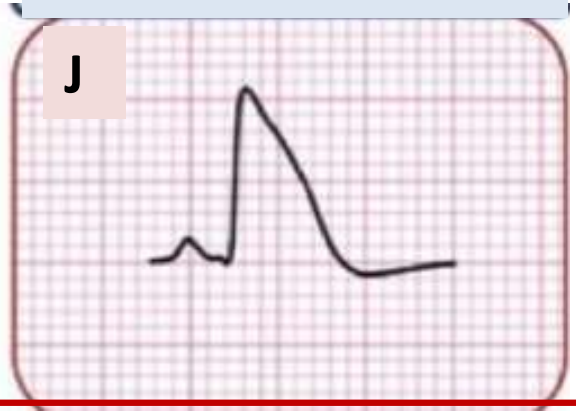


ST elevation with amplitude >25% of the depth of the preceding S-wave with discordant QRS complex (leads V1 to V3)
Smith-Modified Sgarbossa criteria is sufficient to diagnose OMI in the presence of LBBB

Hyperacute T-wave T V1> TV6

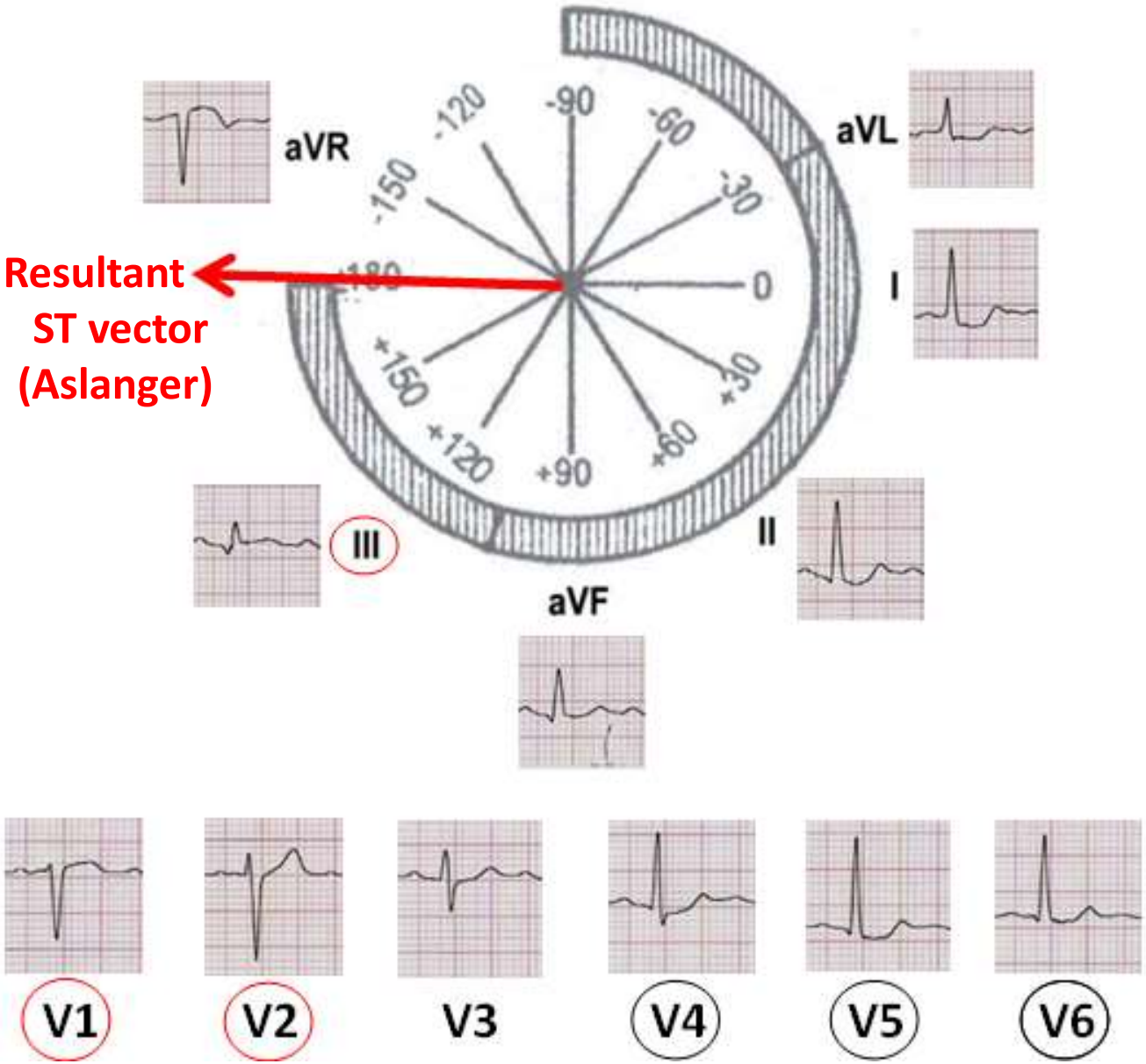


"Shark fin"



Co-merging of elevated convex ST segment with T-wave simulating "Shark fin"
Acute OMI with grave prognosis

Aslanger pattern on ECG

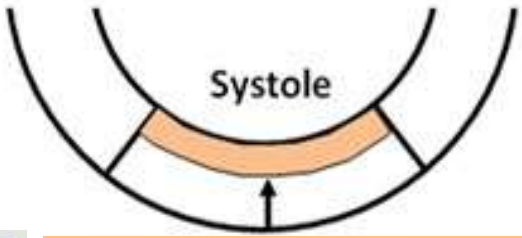


- ST↑ only in lead III as evidence of inferior MI
- ST ↑ in V1>V2
- **Concomitant ST↓in any of V4-V6, with a positive/terminally positive T-wave**

Resultant ST vector (Aslanger)

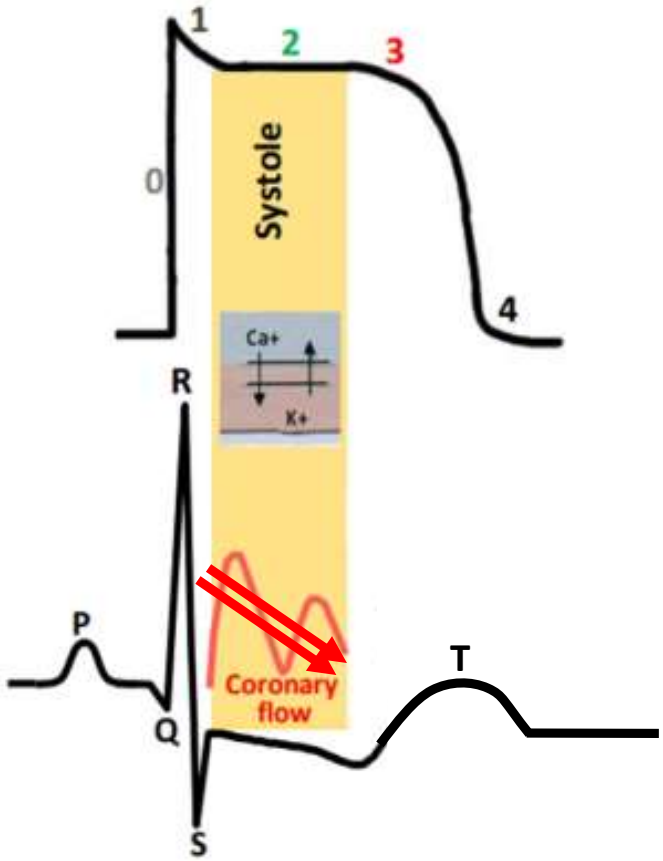
There are two ST vector forces – one from inferior occlusive MI and the other from subendocardial ischemia. The resultant average ST vector is directed rightwards , as indicated by **red arrow** , causing ST elevation in leads III and aVR.

ST↓ in myocardial ischemia



Subendocardial Injury

- The flow of current is away from the exploring electrode (Potential difference)
- ST segment depression

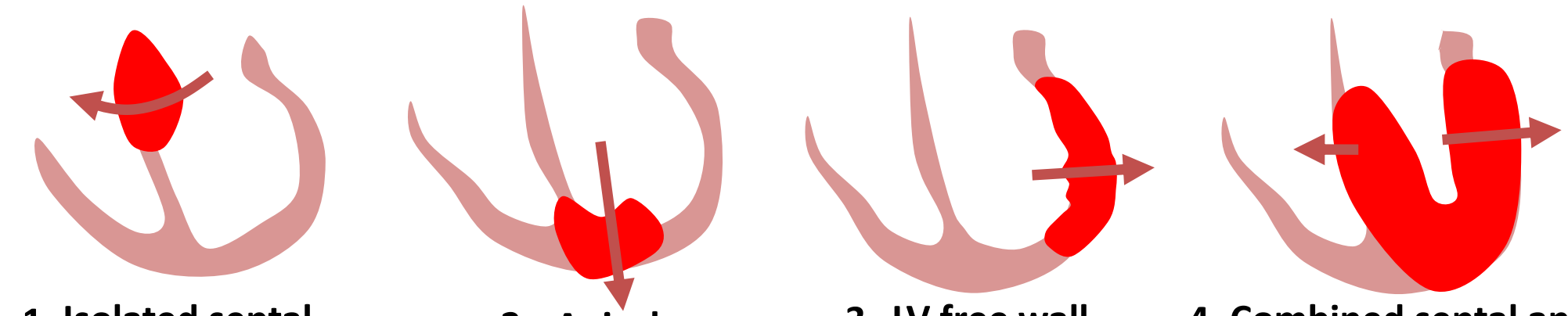


Downsloping ST segment depression

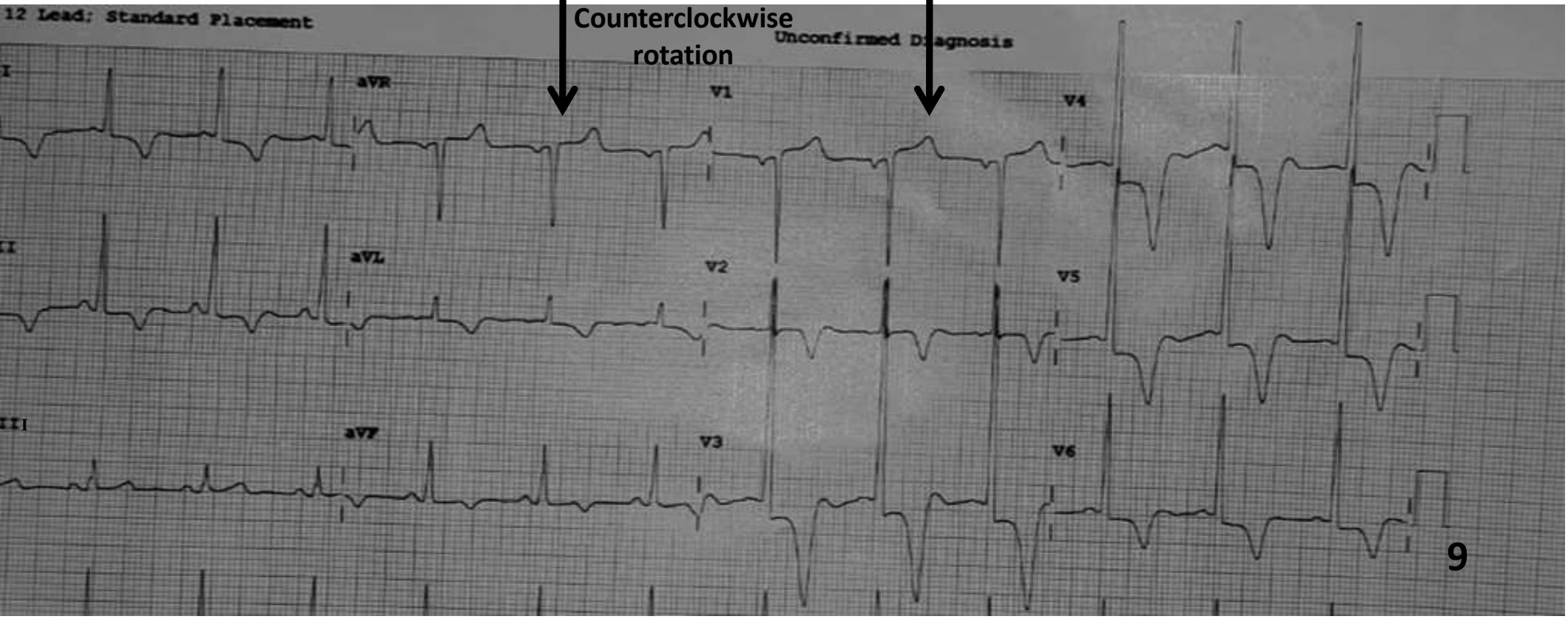
Horizontal or downsloping ST segment depression

- ≥ 0.5 mm ≥ 2 in two contiguous leads myocardial ischemia
- ST depression ≥ 1mm in two contiguous leads a worse prognosis.
- ST depression ≥ 2 mm in ≥ 3 leads possibility of Non-ST elevation myocardial infarction (NSTEMI) ± T inversions or flat T

HCM (Hypertrophic element + secondary ST depression with dragger T inversion , ↓PR interval ± other associations)



1. Isolated septal hypertrophy
2. Apical hypertrophy
3. LV free wall hypertrophy
4. Combined septal and Free LV wall hypertrophy



Counterclockwise rotation

QT interval

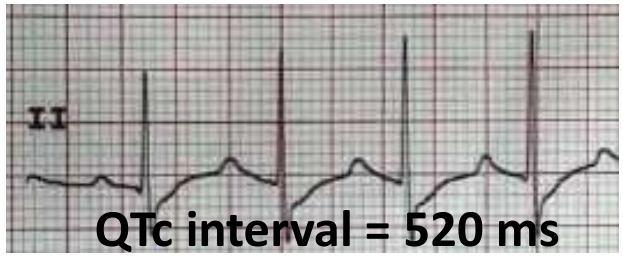
Normal range 0.35- 0.45 sec in males 0.36-0.46 in females

- Prolonged QTc in hypokalemia , hypocalcaemia , myocardial disease , congenital long QT syndrome

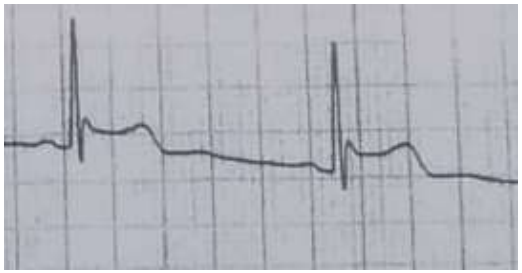
  Shortened QT interval : In Hyperkalemia , Hypercalcaemia , Vagotonia , Digoxin toxicity , congential short QT syndrome

- Illustration by ECGs

Prolonged QT by rule of thumb



Hypokalemia (A young male with Quadriparesis and respiratory arrest)

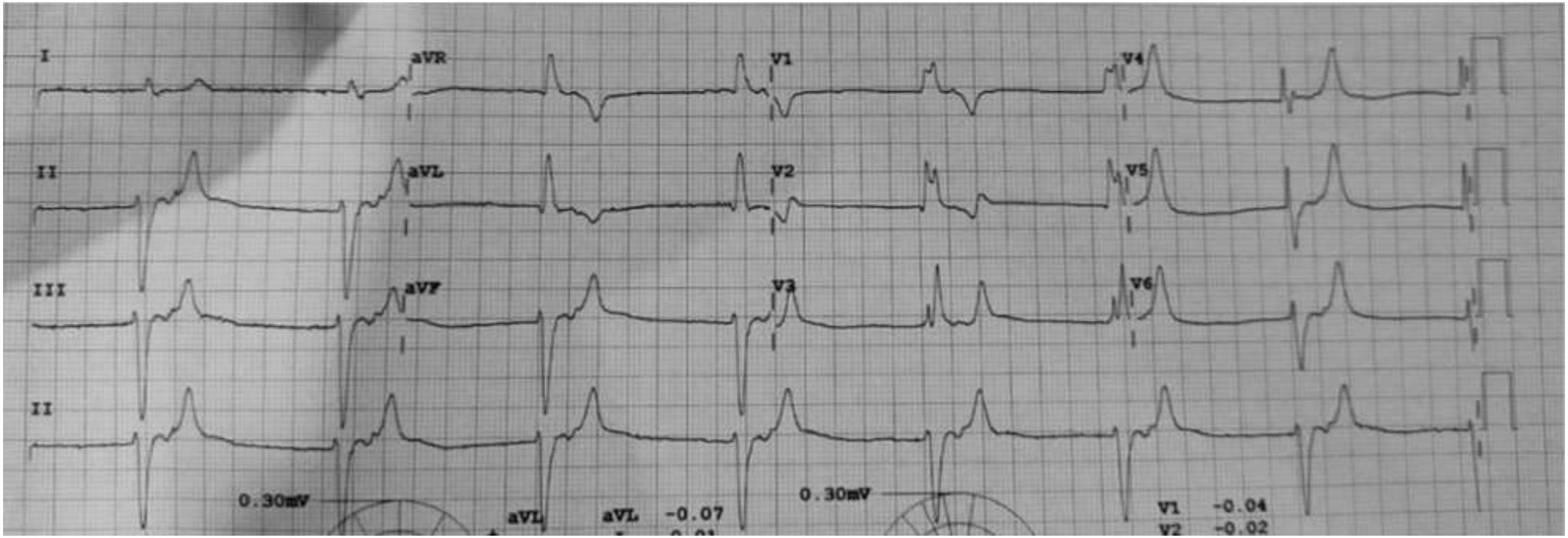


Hypercalcaemia : with 340 ms with virtual absence of ST segment and a widening of T-wave

Congenital short QT syndrome

- QT and QTc intervals respectively : 0.32 and 0.32 ms both are equal and unchanged.
- Tall / peaked T , best seen over precordial leads V1-V4.

A diabetic male aged 45years with Serum creatinine 2.5 mg and K 6.5 mEq/L .

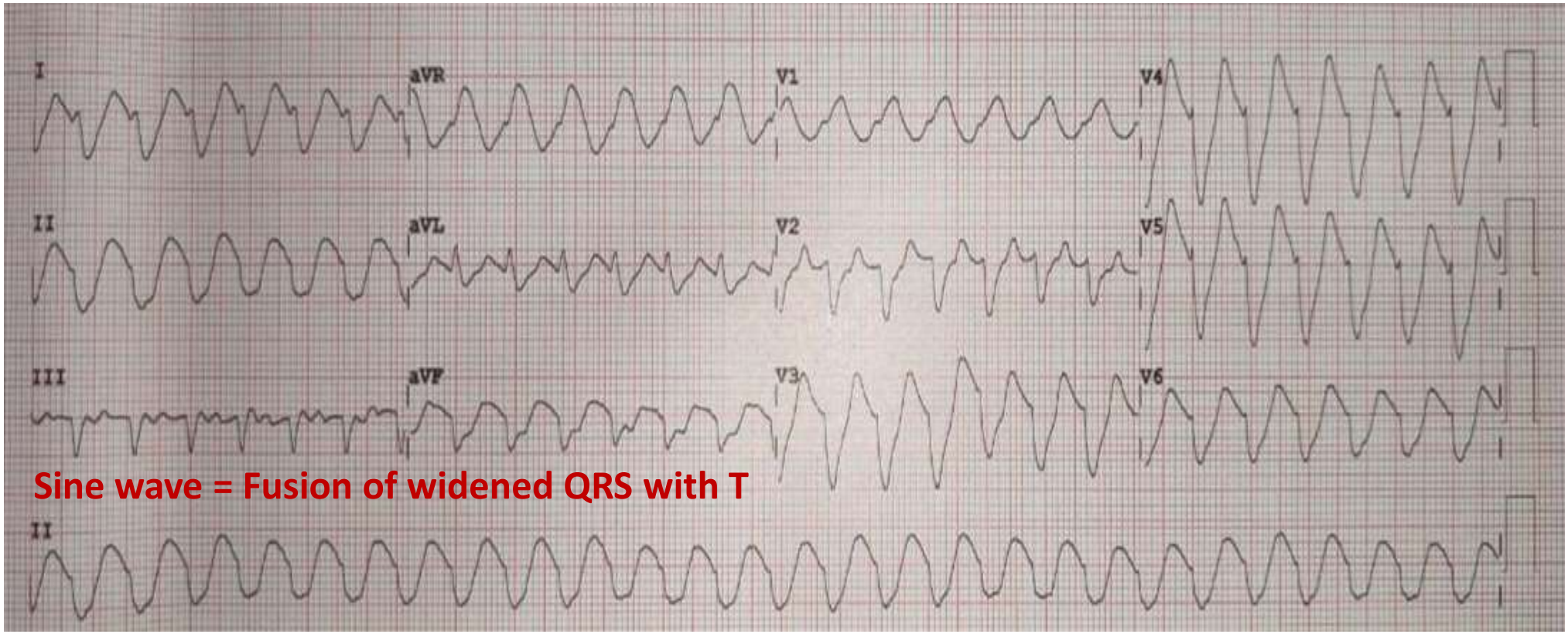


ECG findings :

- **Junctional rhythm with retro negative P in inferior leads with heart rate 44 bpm**
- **RBBB pattern (see V1)**
- **Peaked tented T seen over V3 to V6 and over leads I , II , III and aVF**

Sine Wave in hyperkalemia

Middle aged Diabetic female presenting with weakness , giddiness , nausea and vomiting



Findings on ECG :

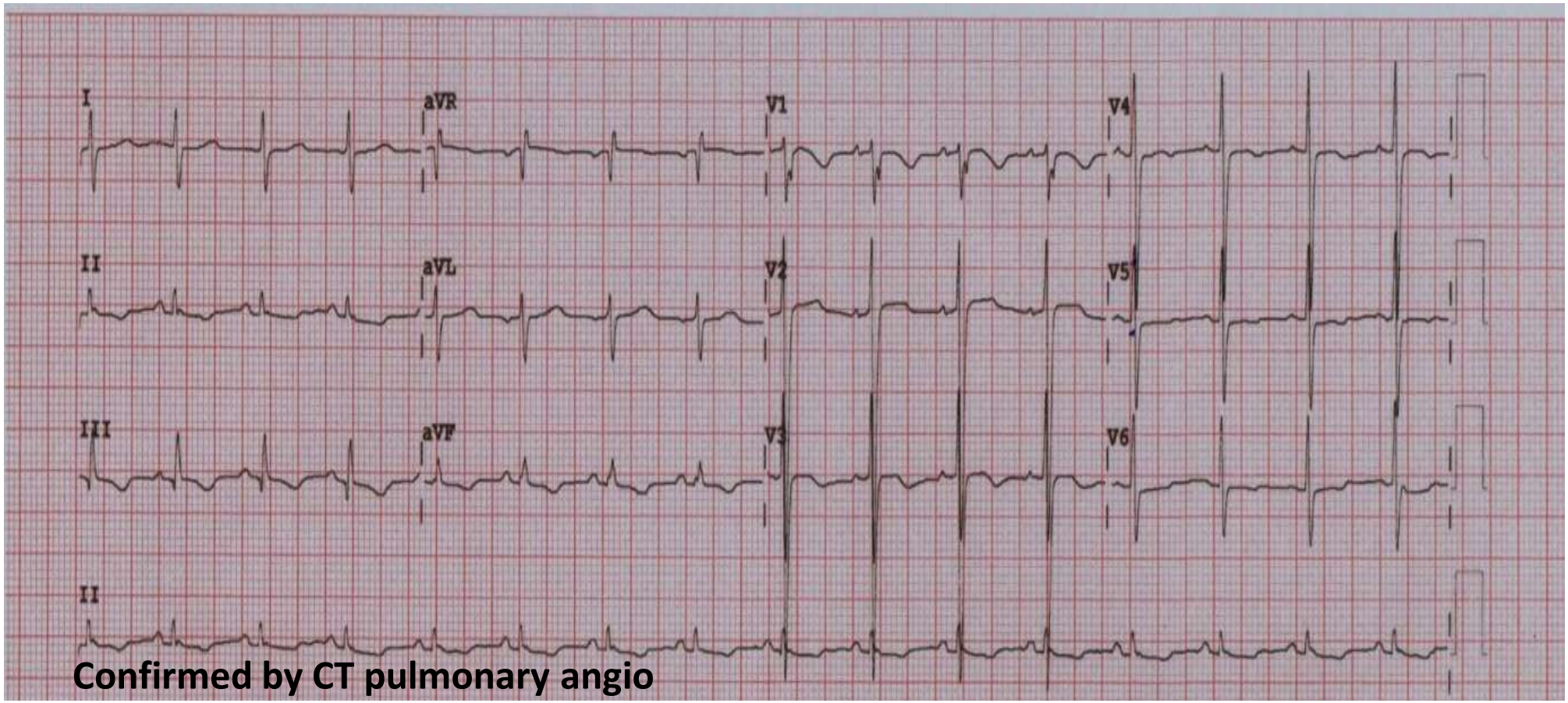
- **Ventricular rate 186 bpm**
- **Rhythm lead II shows a run of up and down oscillations , almost with equal pacing and amplitude --- sine wave**
- **Lead V1 is showing much widening of the QRS complex almost equivalent to 0.28 sec simulating with right bundle branch block pattern.**

Miscellaneous ECGs

Acute Pulmonary Embolism

Acute dilatation of RV with Strain either fully or partially in combination

A 42 years male suddenly presented with breathlessness

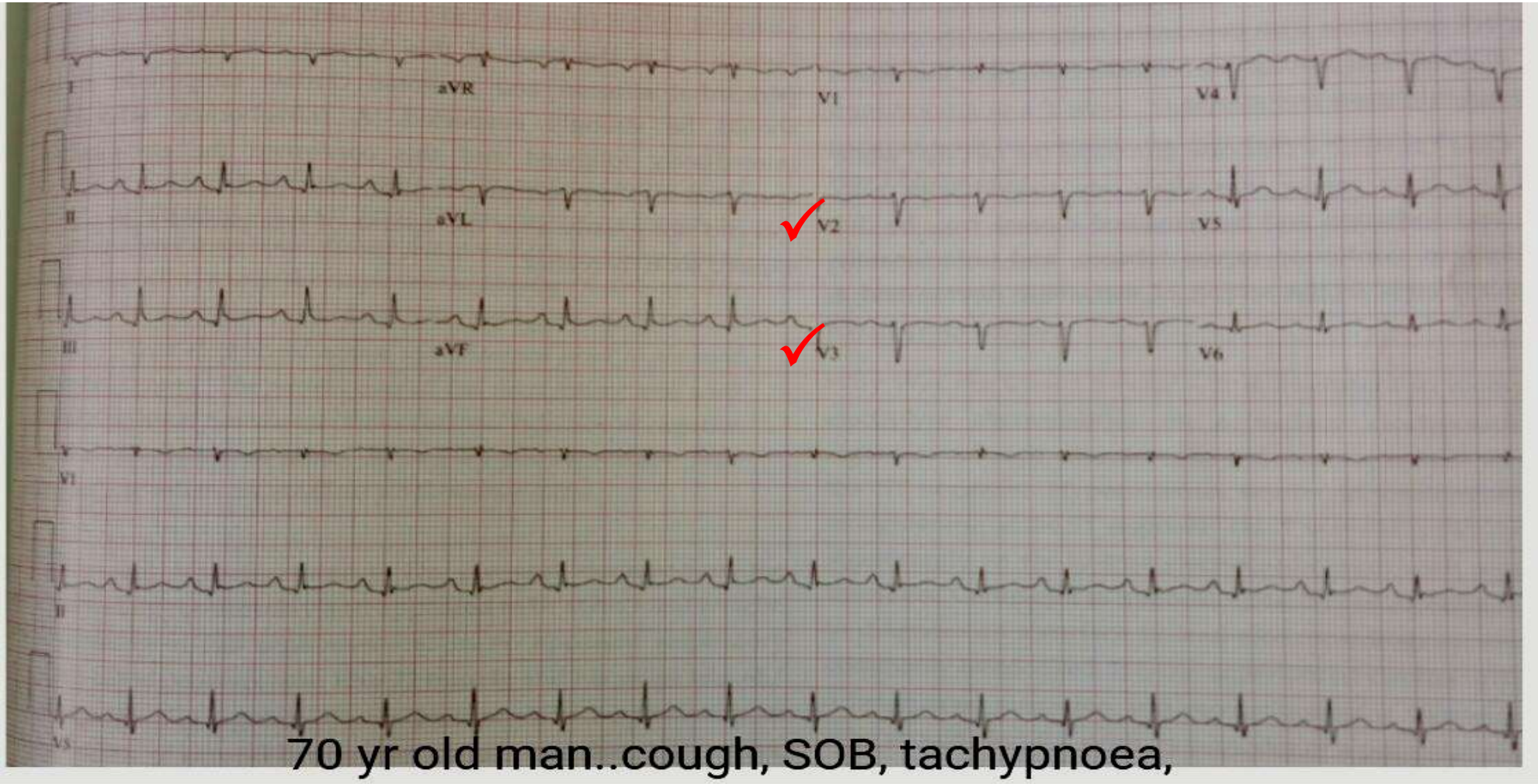


Confirmed by CT pulmonary angio

- **HR =100 bpm**
- **$S_I Q_{III} T_{III}$ Pattern**
- **T ↓ over V1 -3 , also over inferior leads (II, III, aVF). Mild ST elevation in V1-3 (? RV epicardial current of injury due to stretching)**
- **Clockwise rotation**

Pericardial effusion

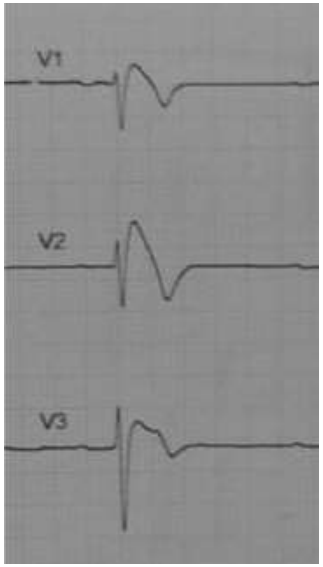
Attenuating effect of QRS complexes due to the surrounding pericardial fluid



- **Triad of Sinus tachycardia (here Heart Rate = 111 bpm) , Electrical alternans , most obvious over precoardial leads V2-3 and low voltage over both the plane.**
- **Cardiac echo showed the presence of Pericardial effusion.**

Some intersting ECGs

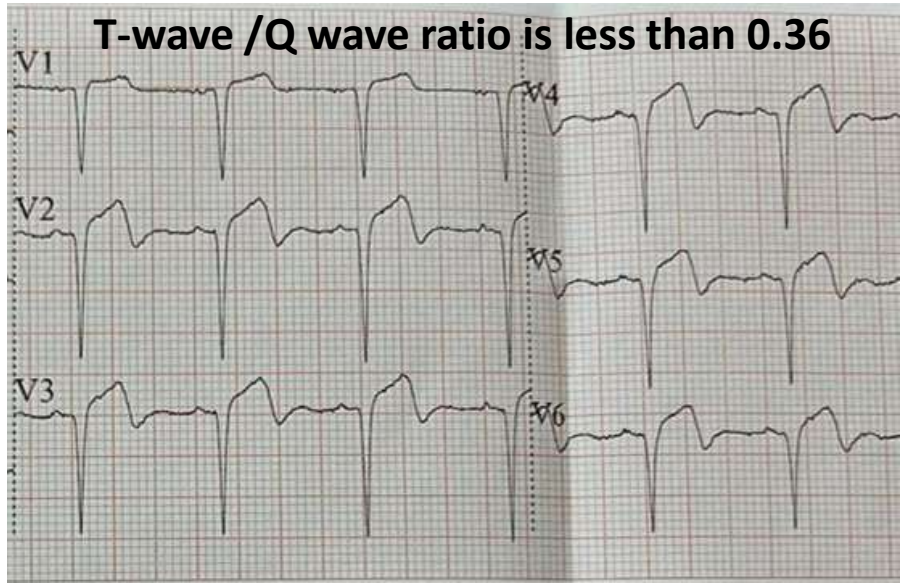
Brugada syndrome – Type I



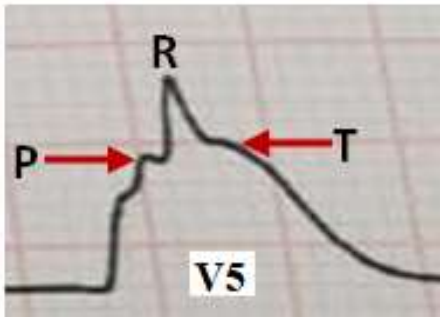
Coved-ST segment elevation > 2mm > 1 of V1-V3 followed by a negative T-wave

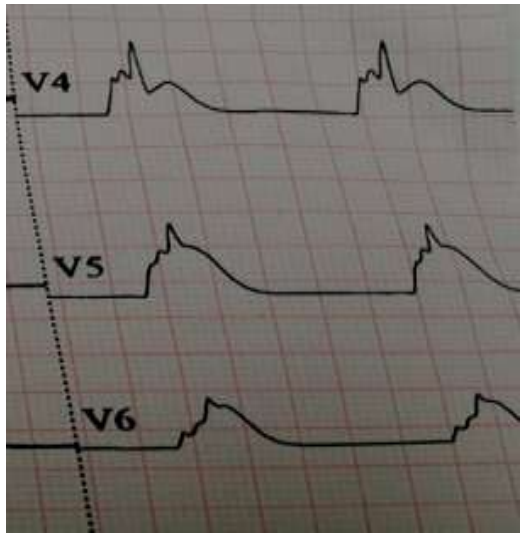
Ventricular anneurysm

T-wave /Q wave ratio is less than 0.36



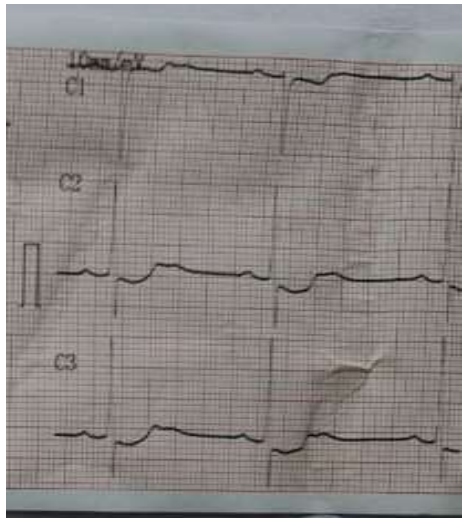
Spiked helmet sign





Digitalis

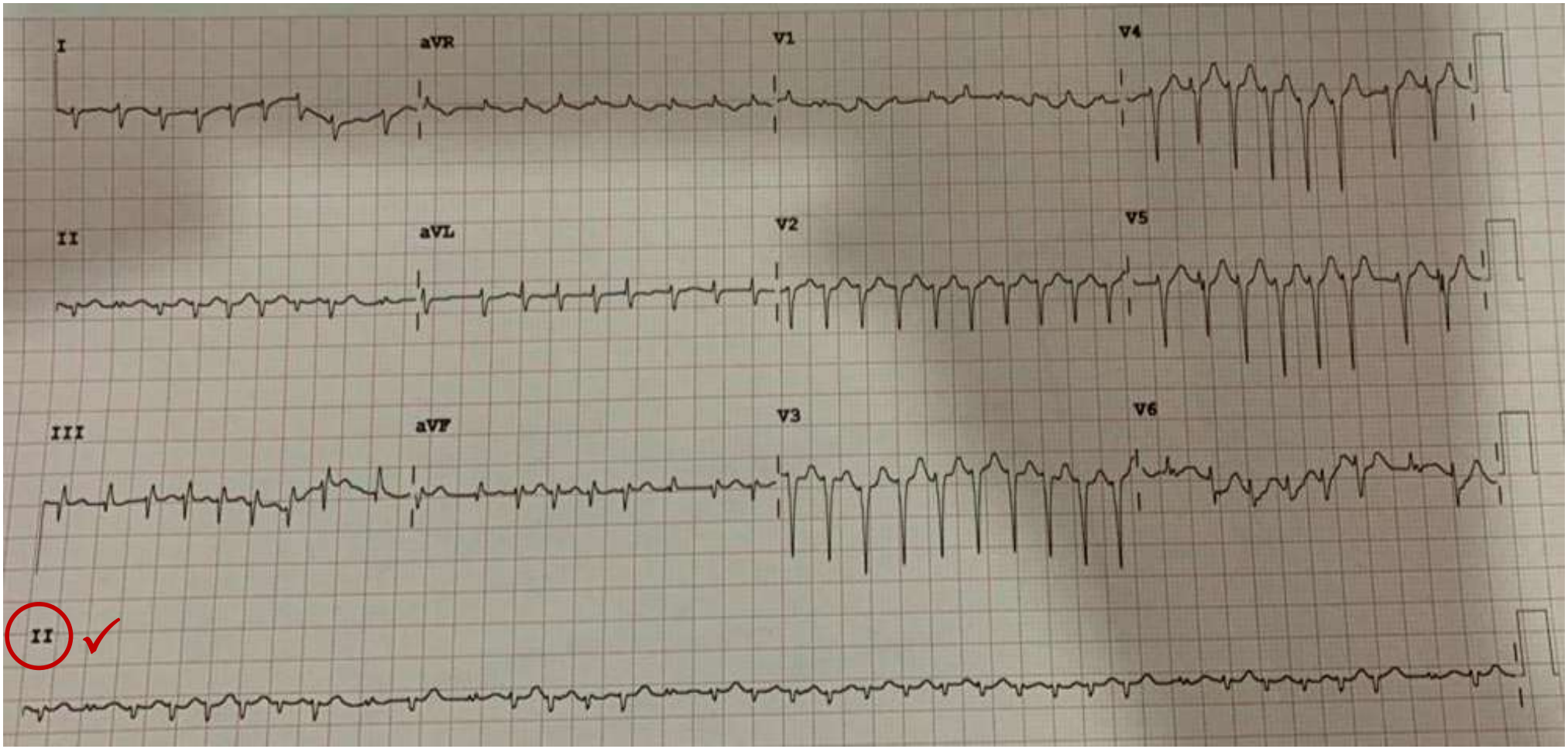
- Sinus bradycardia
- PR interval = 0.22 sec
- Mirror image of correction mark with the rising of the T-wave above the baseline
- Shortening of the QT interval = 0.34 sec



(Digitalis toxicity Note that T wave does not go beyond the baseline)

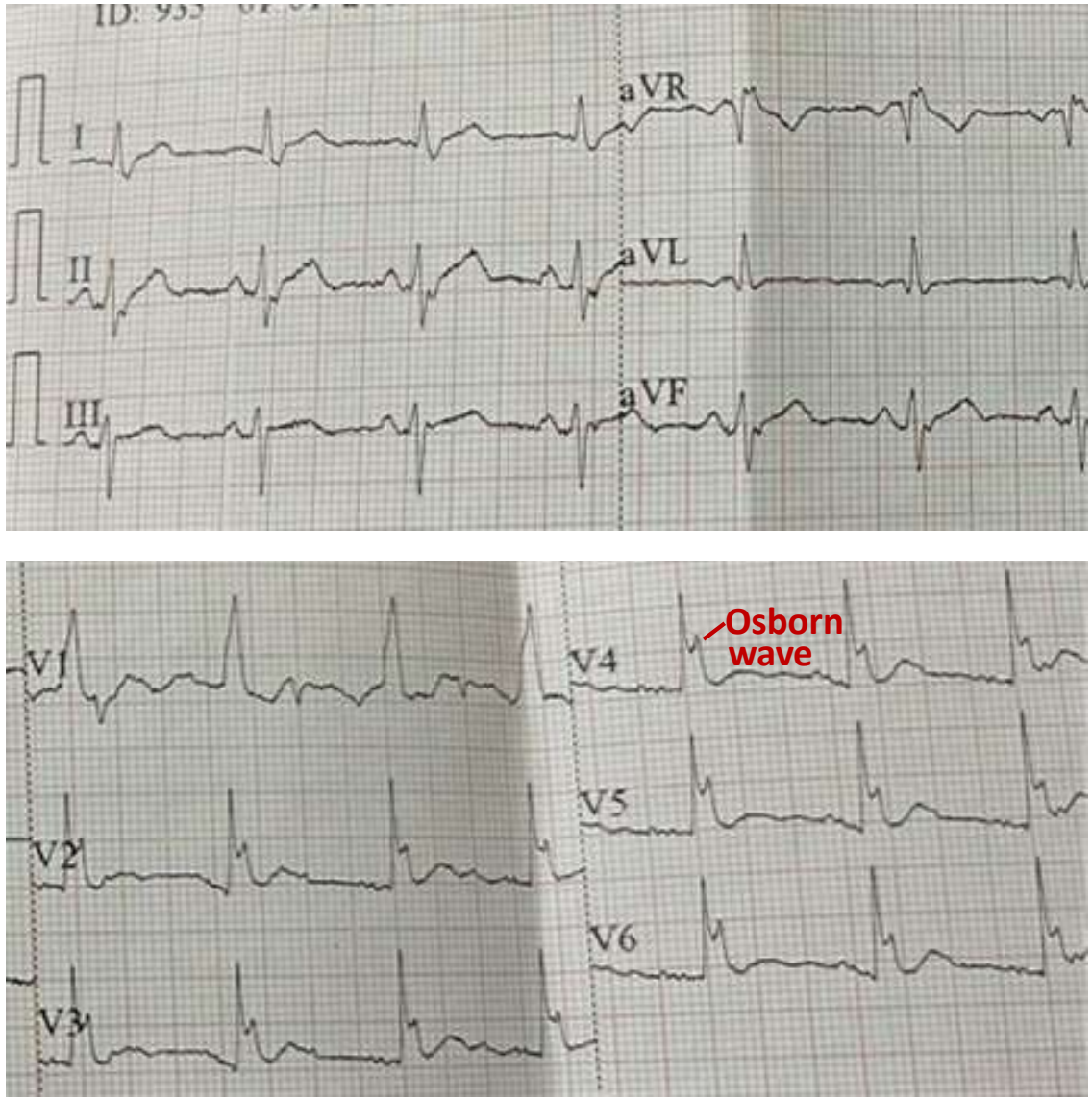
DCMP

Middle aged lady presenting with breathlessness (Globular cardiomegaly on radiology)



- **Low voltage QRS in limb leads**
- **Poor R-wave progression in chest leads.**
- **Atrial fibrillation with fast ventricular rate (250 bpm) (please see the rhythm lead II : rhythm is irregularly irregular)**
- **North-west axis + 170^0 (anatomical distortion of His bundle and its branches)**

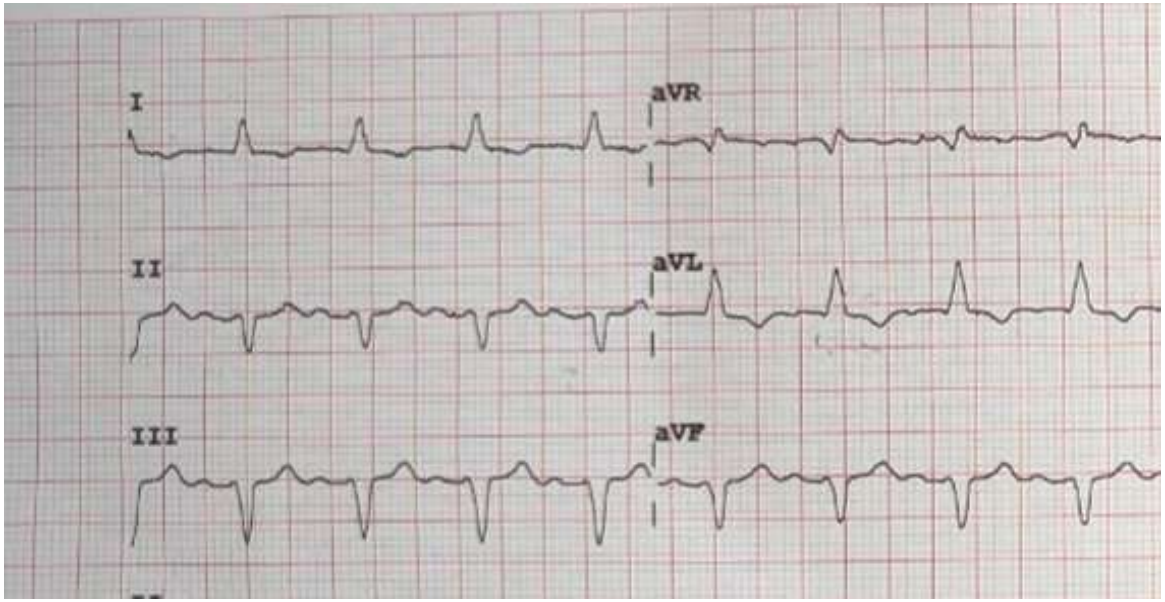
Osborn wave



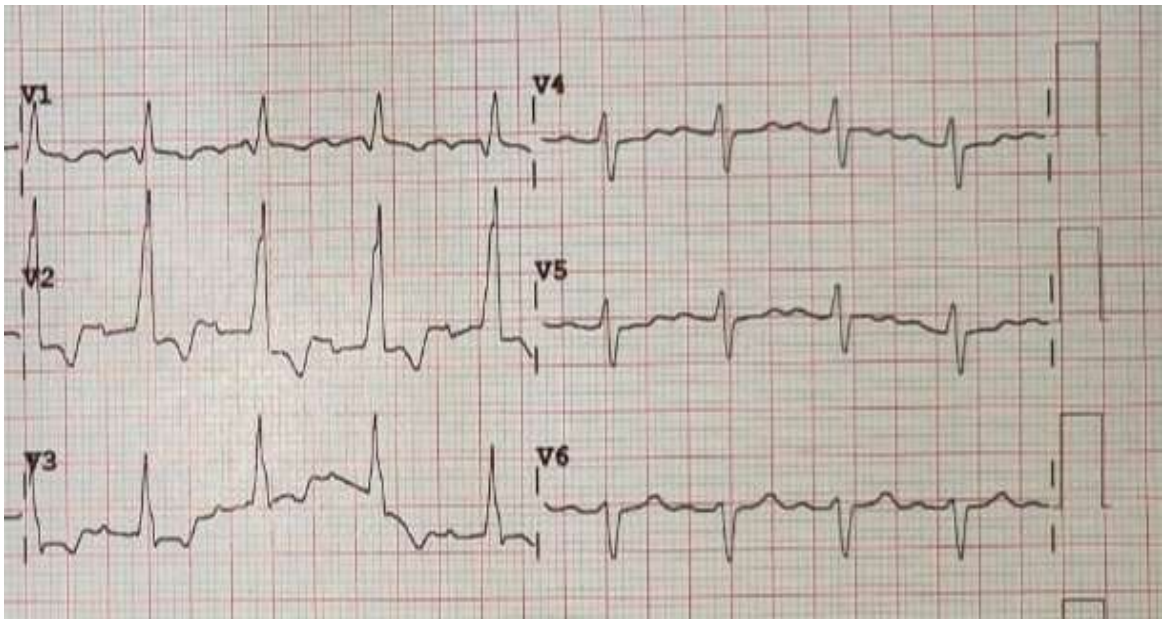
Osborn waves in the setting of underlying RBBB (seen in hypothermia , hypercalcaemia and other miscellaneous causes as well)

Although controversial , a notable propensity to herald ventricular fibrillation so never be ignored

Masquerading BBB ⟶ Higher risk to CHB



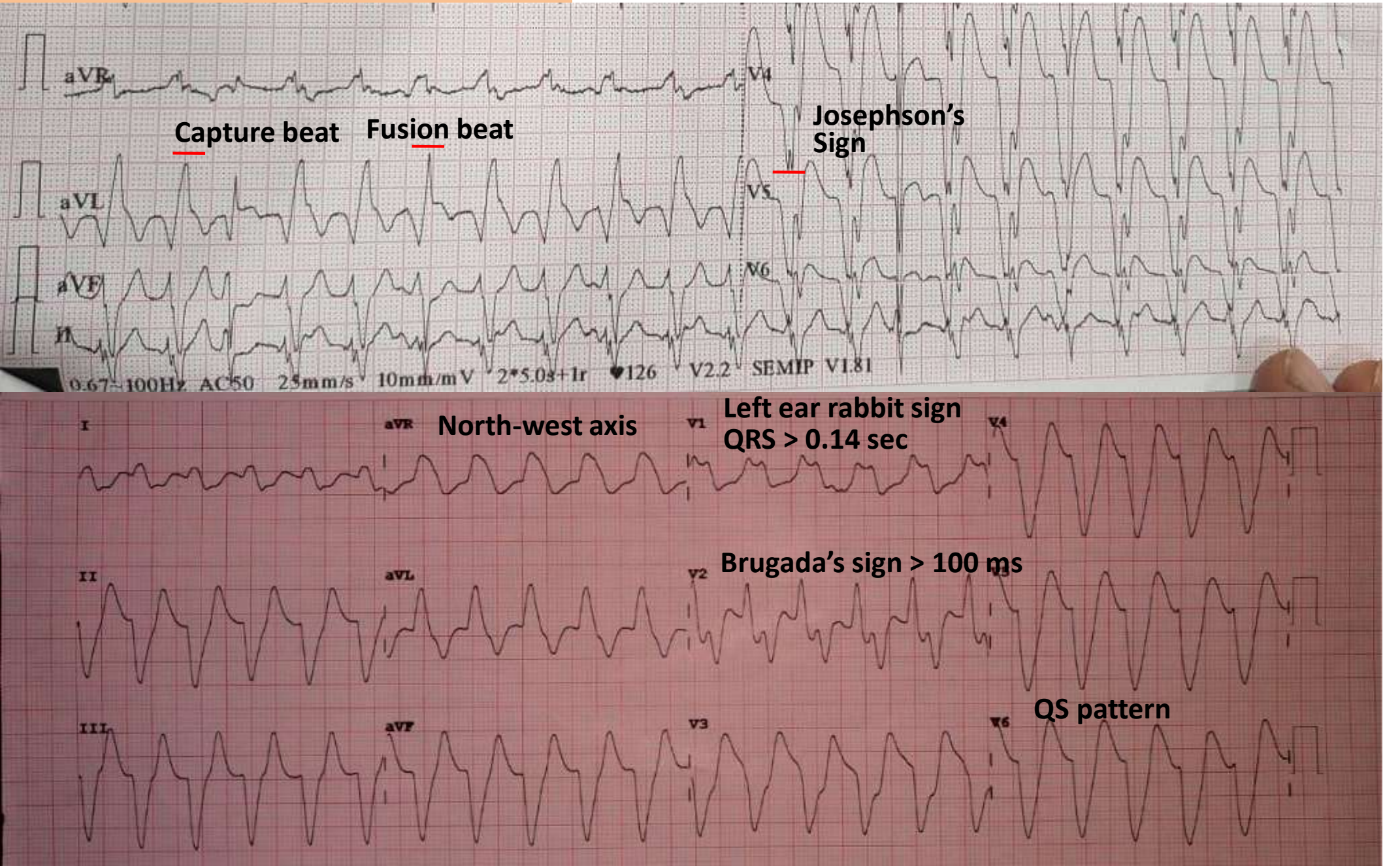
Limb leads resembles LBBB pattern in limb leads with left axis deviation



Precordial leads shows RBBB pattern

Ventricular tachycardia - VT

AV dissociation with faster ventricular rate (ventricular origin)



NB : +Ve or –Ve concordance throughout the chest lead , i.e. with leads V1-6 so entirely positive (R) or entirely negative (QRS complexes) , with no RS complexes seen

Concluding remarks

- The 12-lead ECG continues to be a very remarkable tool for identification of life-threatening ECGs in OPD

- It is likely that these ECG models , discussed so far to provide very useful information as predictors of increased SCD but will rather be utilized more to predict risk in even in combination with other risk stratification factors


Thanks