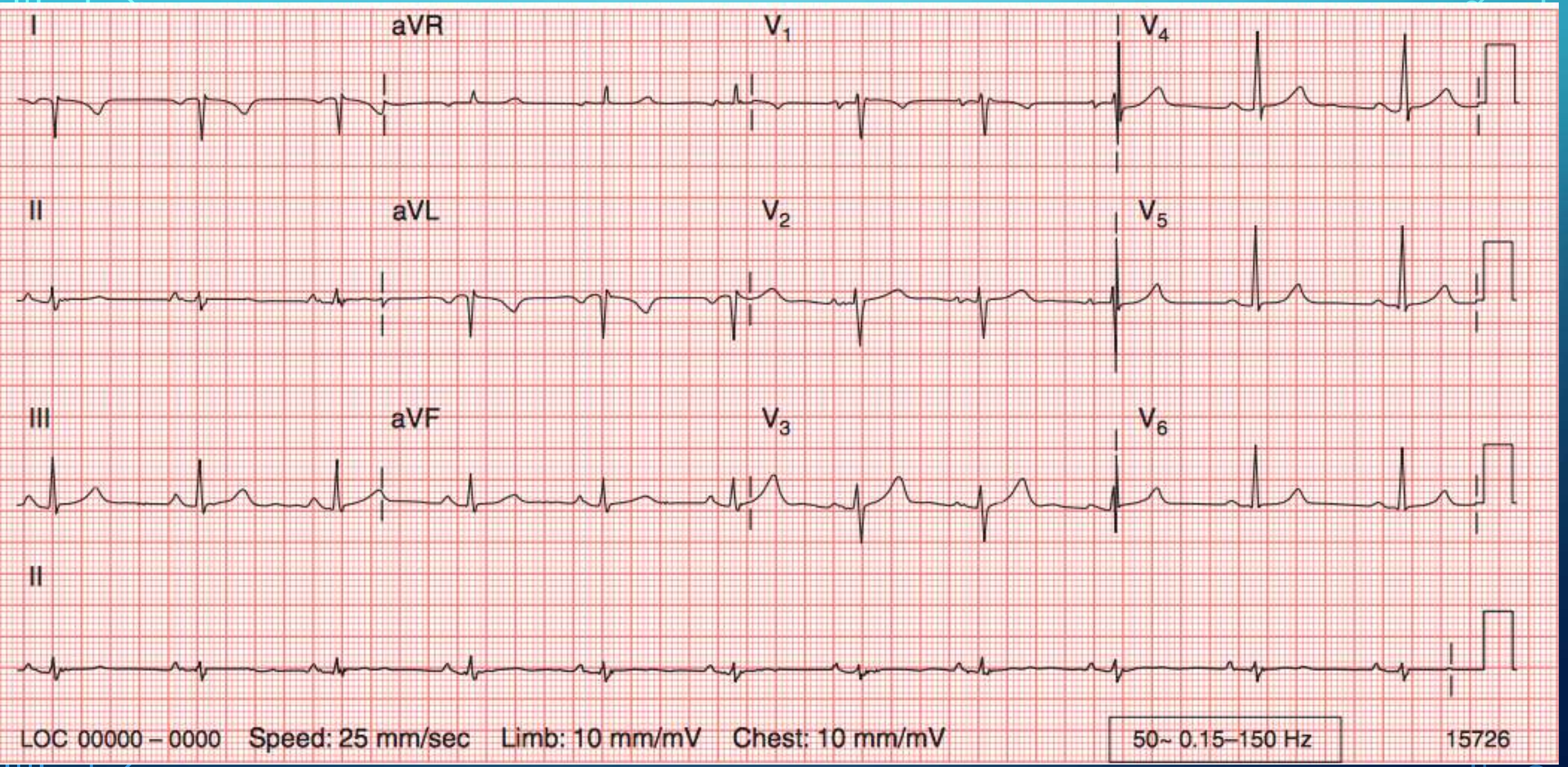
DR.ELHAM FARAHANI

CARDIOLOGIST


I
aVR
V1
V4
II
aVL
V2
V5
III
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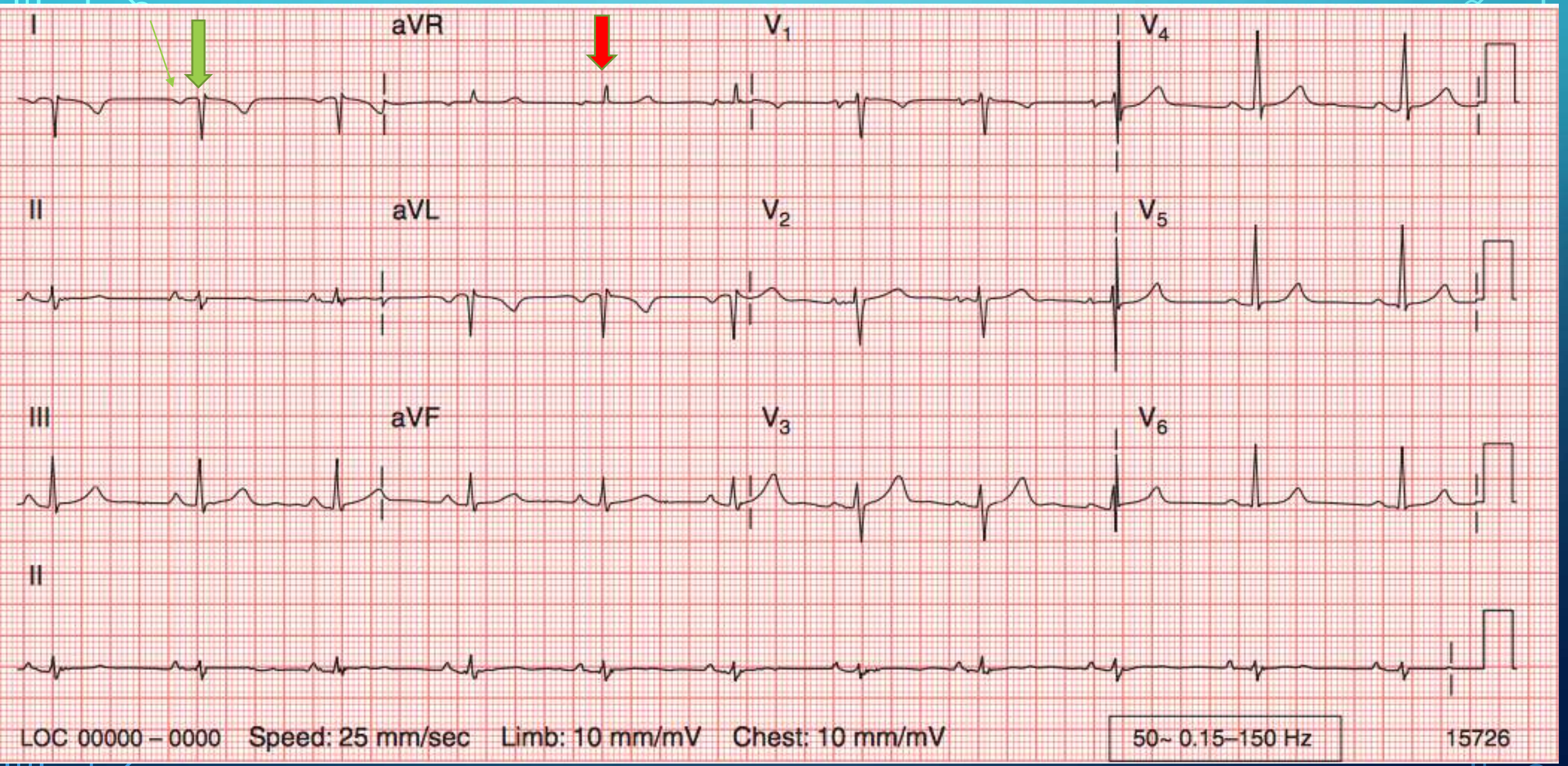
V3
V6
II
LOC 00000 – 0000 Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10 mm/mV
50~ 0.15–150 Hz
15726


I
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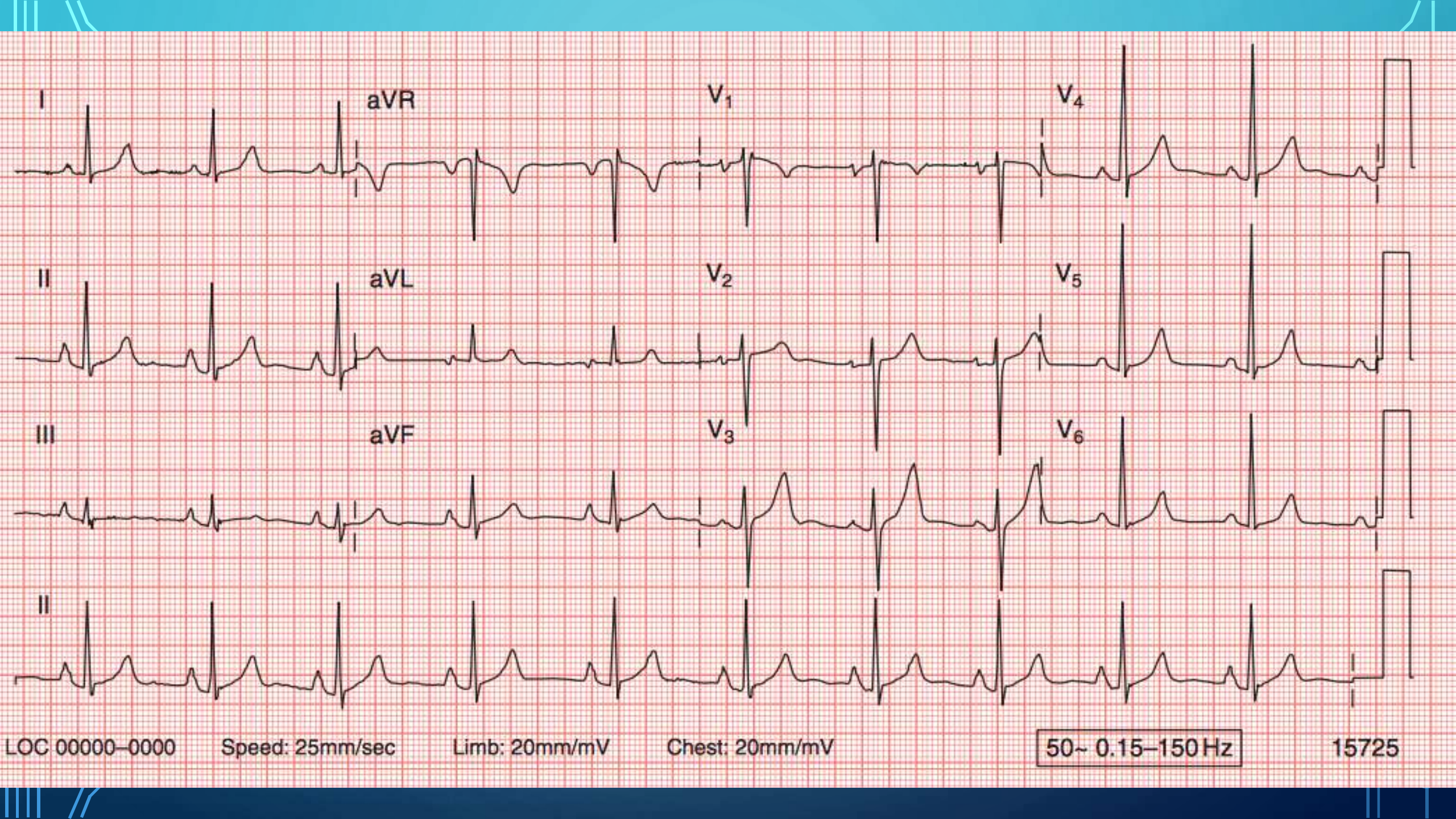
V5
III
aVF
V3
V6
II
LOC 00000 – 0000 Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10 mm/mV
50~ 0.15–150 Hz
15726

MISPLACEMENT OF THE RIGHT AND LEFT ARM ELECTRODES.

- There is extreme right axis deviation (152°), and a positive QRS complex in lead aVR.
- ● The QRS complexes in leads I and aVL are negative, and there is P wave and T wave inversion in these leads too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LOC 00000–0000
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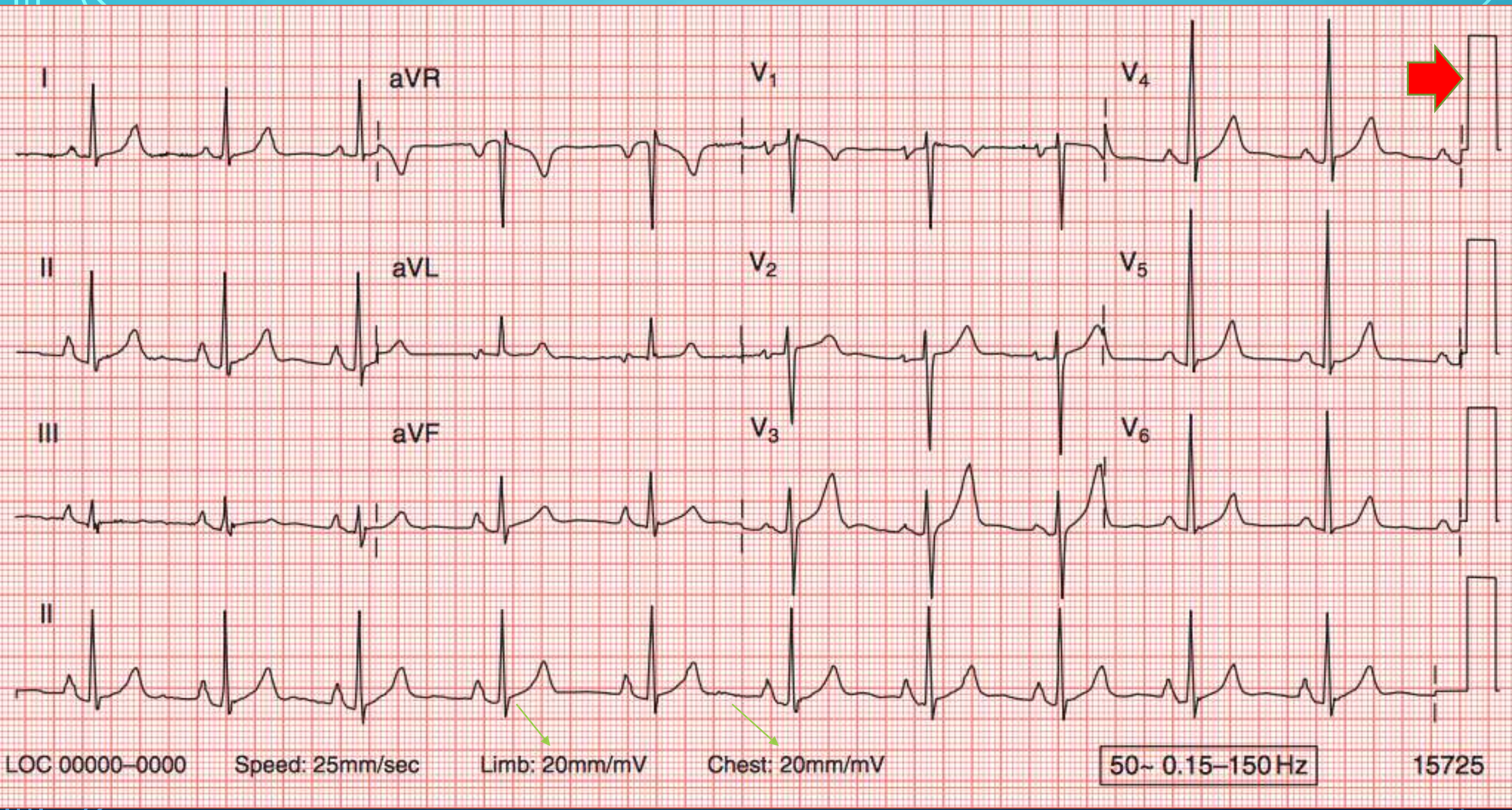
Limb: 20mm/mV
Chest: 20mm/mV
50~ 0.15–150 Hz
15725


I
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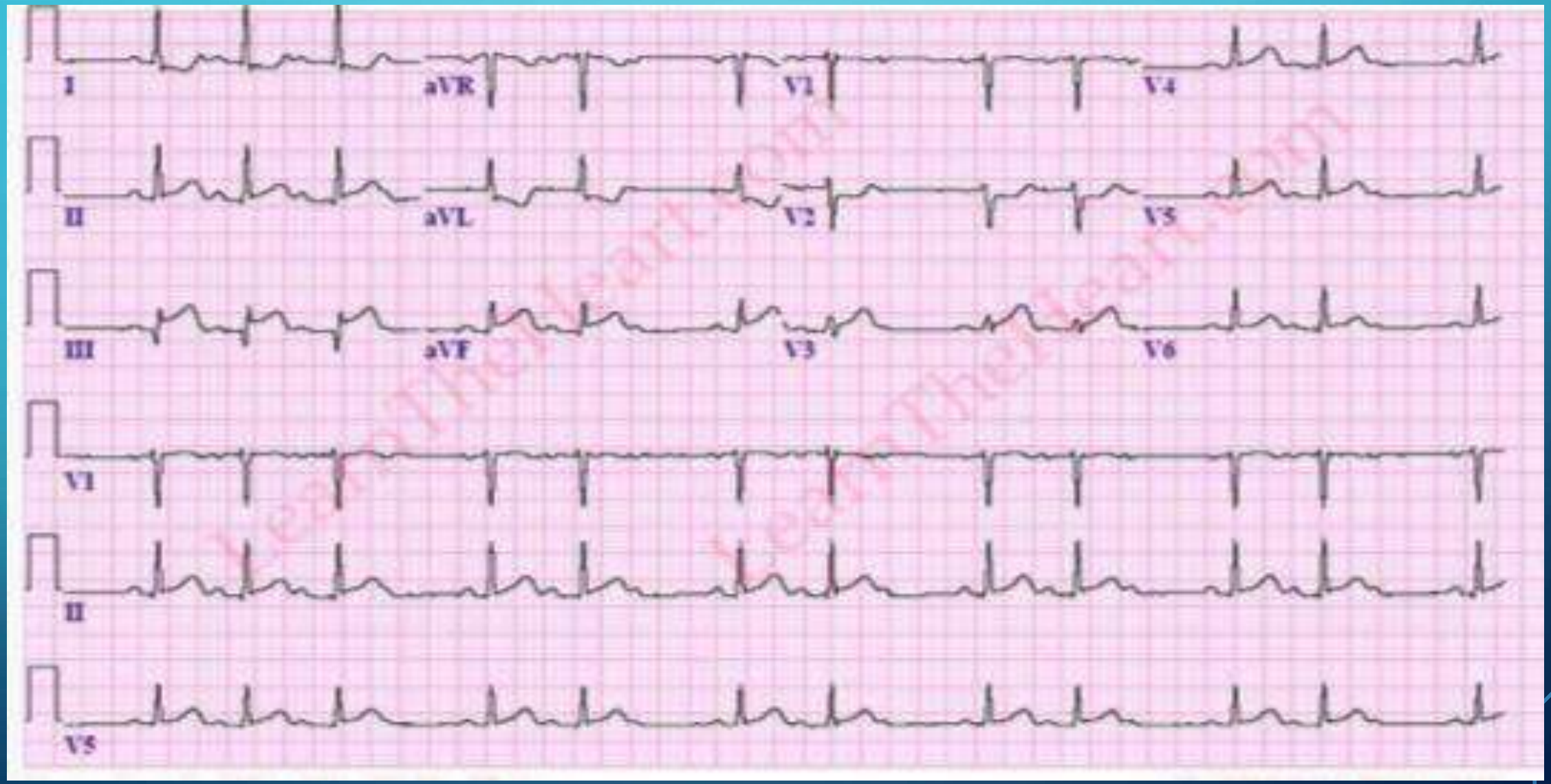
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
LOC 00000–0000
Speed: 25mm/sec
Limb: 20mm/mV
Chest: 20mm/mV
50~ 0.15–150 Hz
15725

- The voltage calibration setting is 20 mm/mV, double the ‘standard’ setting
- **normal.**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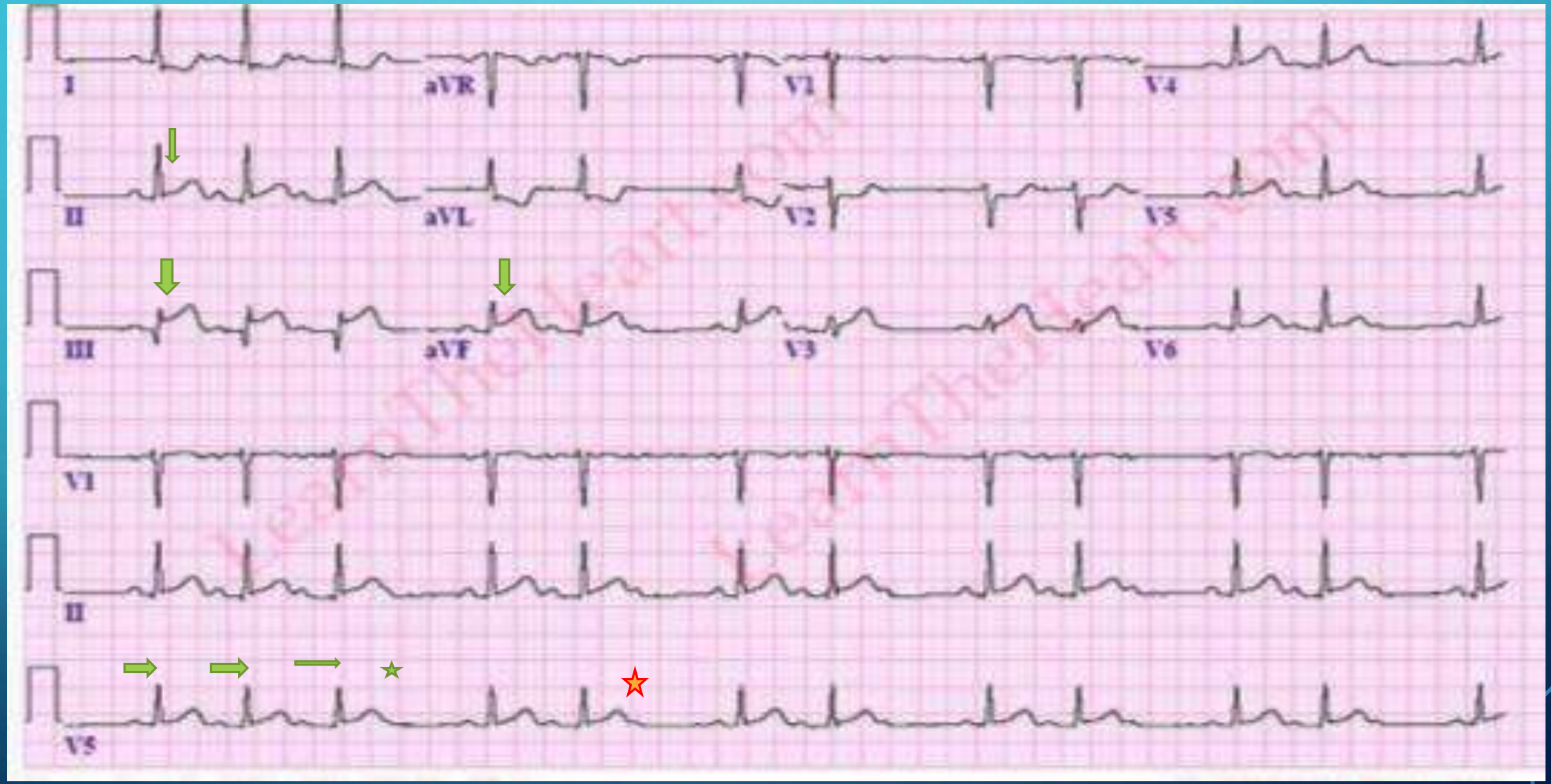
I
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V1
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V5
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V1
II
V5

THE ECG FINDINGS INCLUDE:

- 1. Sinus rhythm with 2nd degree type I AV block (Wenkebach)
- 2. Inferior ST segment elevation MI (leads II, III, and aVF) with reciprocal ST depression (leads I and aVL)

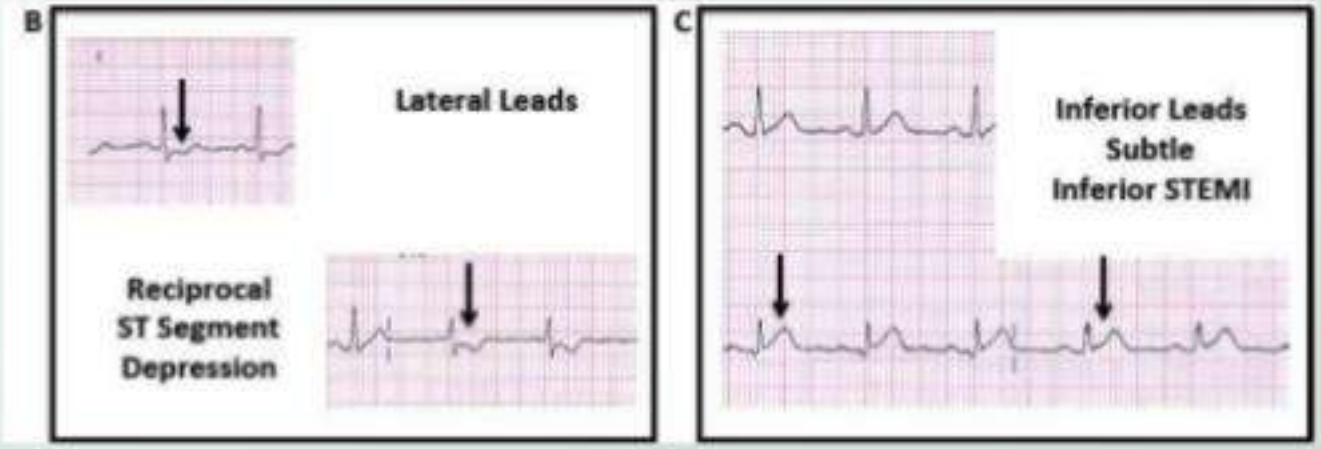
STEMI criteria

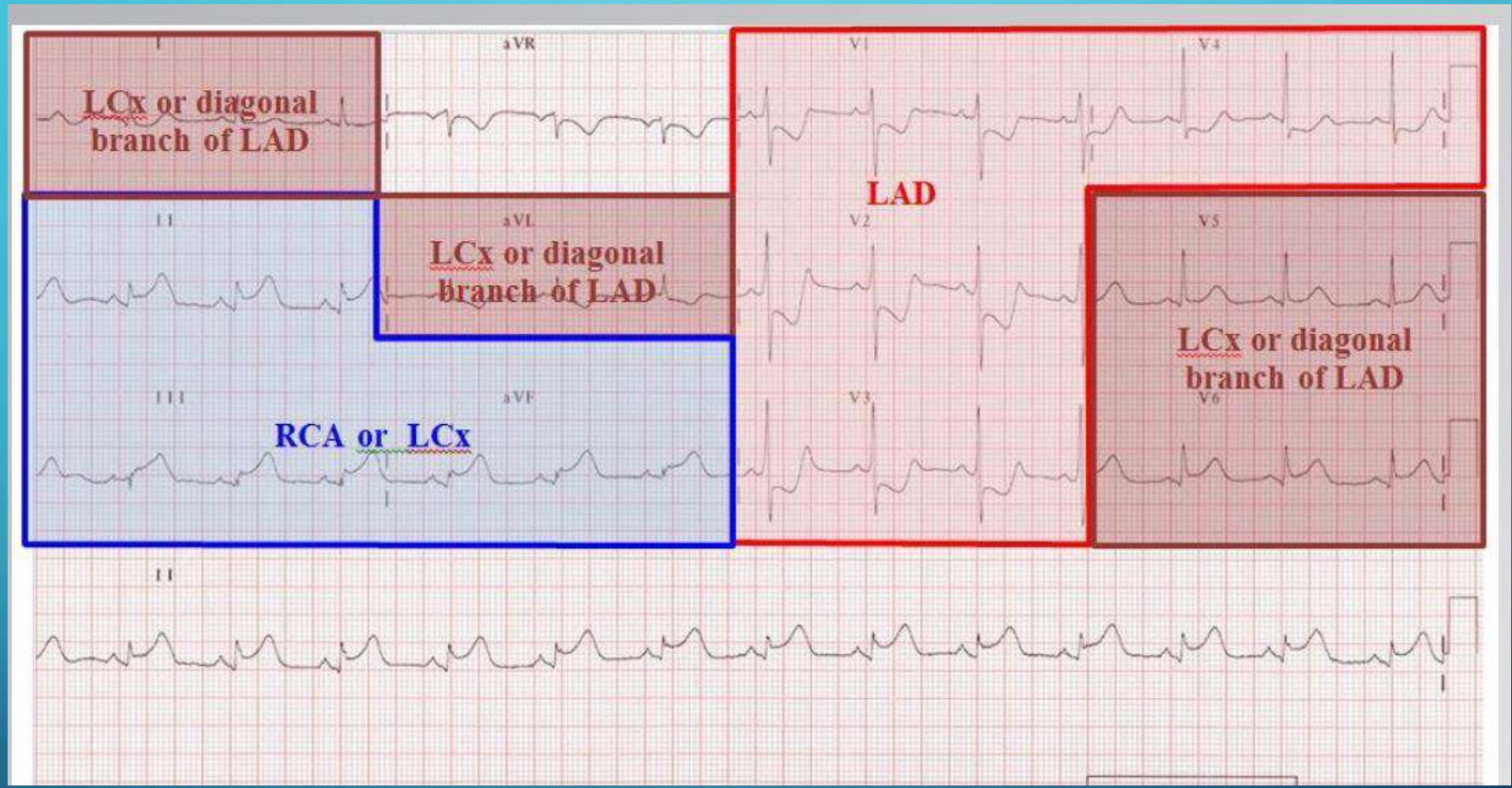
Clip slide

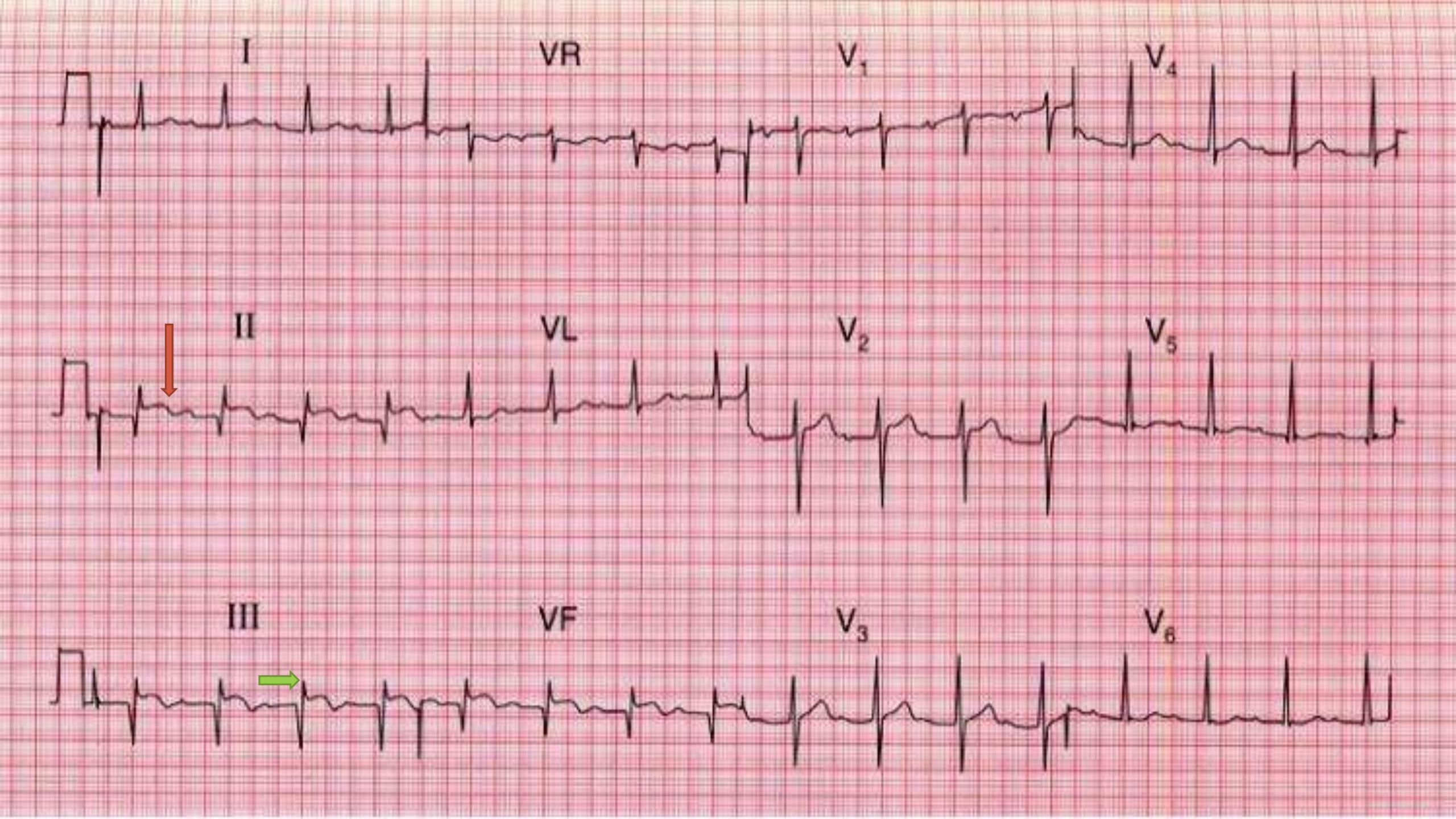
- ≥1 mm (0.1 mV) of ST segment elevation in the limb leads
- ≥ 2 mm elevation in the precordial leads and present in anatomically contiguous leads

Examples on reciprocal changes :

Type of MI	Reciprocal changes (ST depression)
Inferior MI	In lead 1 & aVL
Lateral MI	In lead2, lead3 & aVF




I
aVR
V1
V4
LCx or diagonal branch of LAD
LAD
II
aVL
V2
V5
LCx or diagonal branch of LAD
LCx or diagonal branch of LAD
III
aVF
V3
V6
RCA or LCx
II


I
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II
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III
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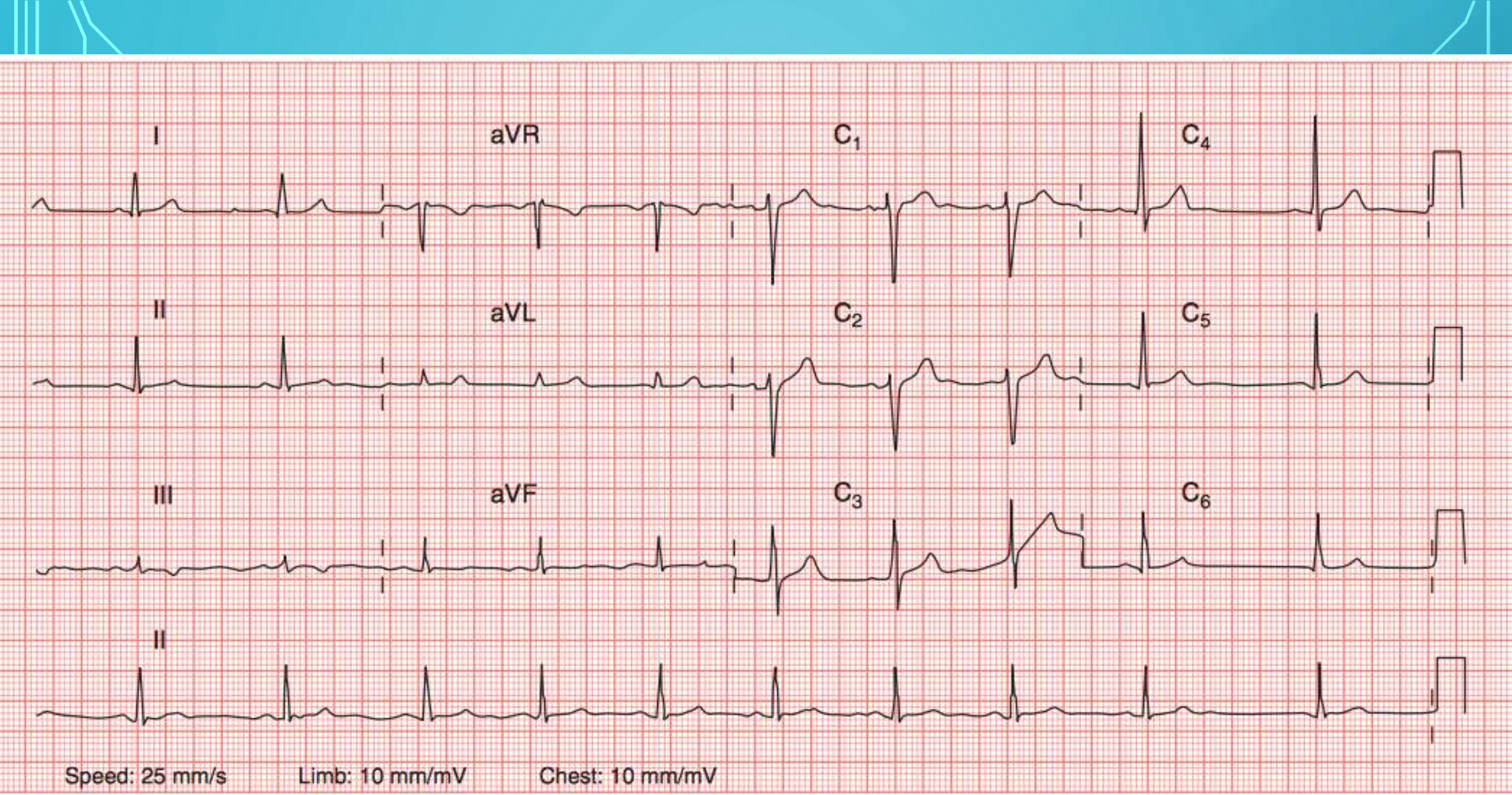
I
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C4
II
aVL
C2
C5
III
aVF
C3
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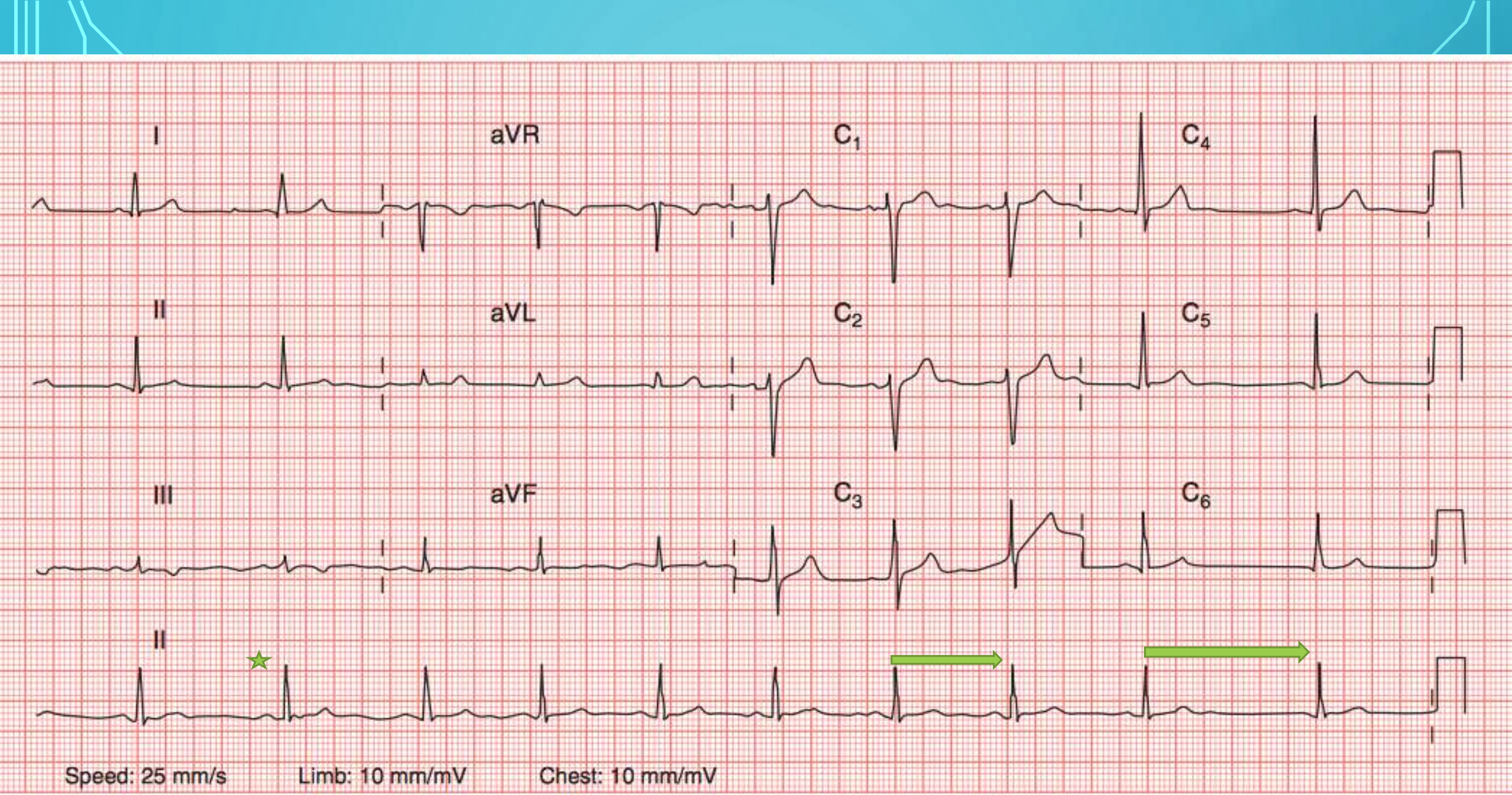
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV


I
aVR
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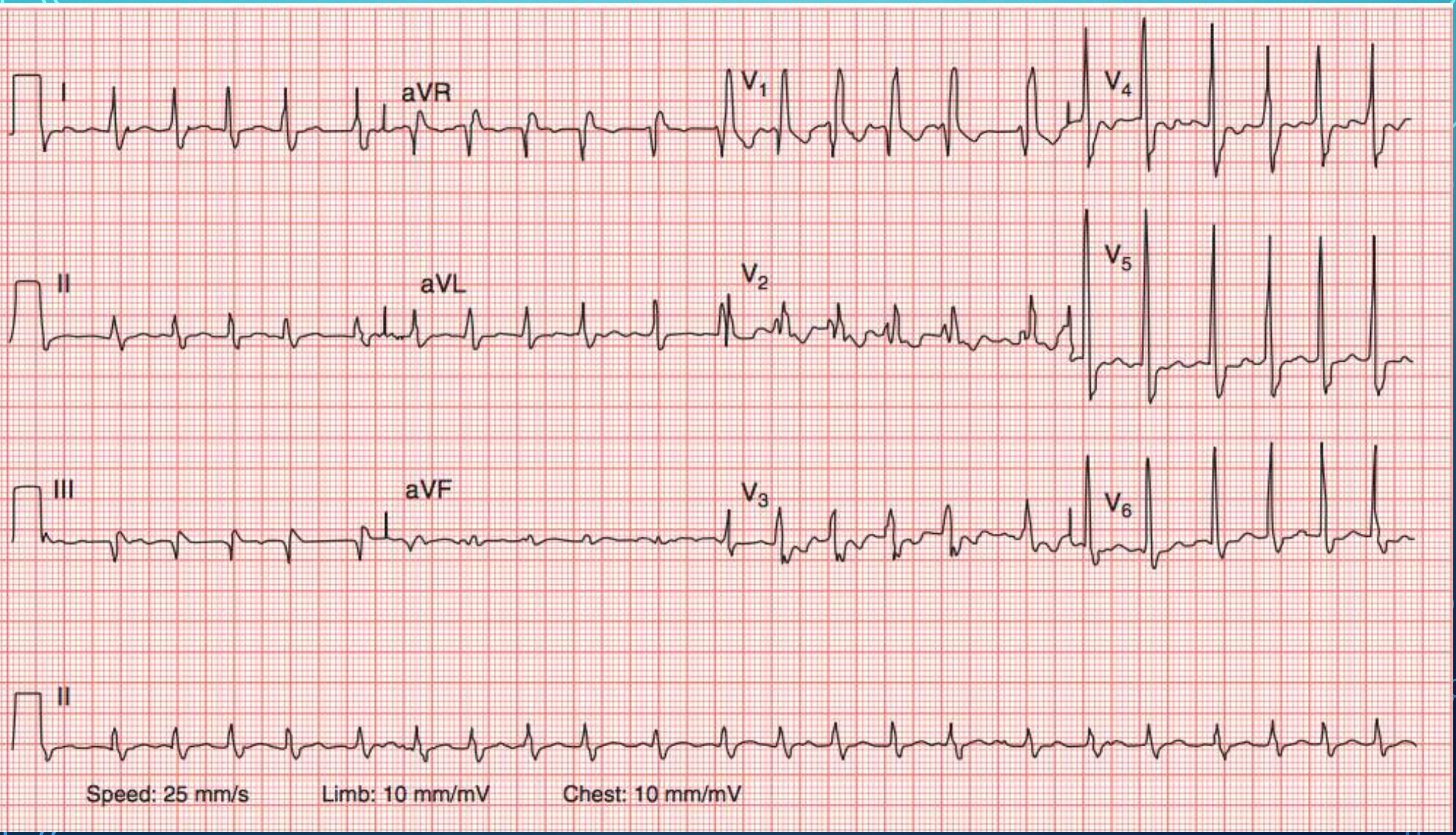
C4
II
aVL
C2
C5
III
aVF
C3
C6
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

- 1 Every P wave is followed by a normal QRS complex, but the heart rate varies. Observation of the patient confirms that this coincides with respiration, with the heart rate increasing on inspiration and decreasing on expiration. This is sinus arrhythmia.
- Max CL-Min CL>120msec
- Max CL-Min CL/Min CL>10%
- NL P wave ,PR>120msec
- PP interval dec with inspiration

- Physiologic,dec with age and autonomic dysfunction(DM)
- Non respiratory:dig toxicity:RF for SCD


I
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V4
II
aVL
V2
V5
III
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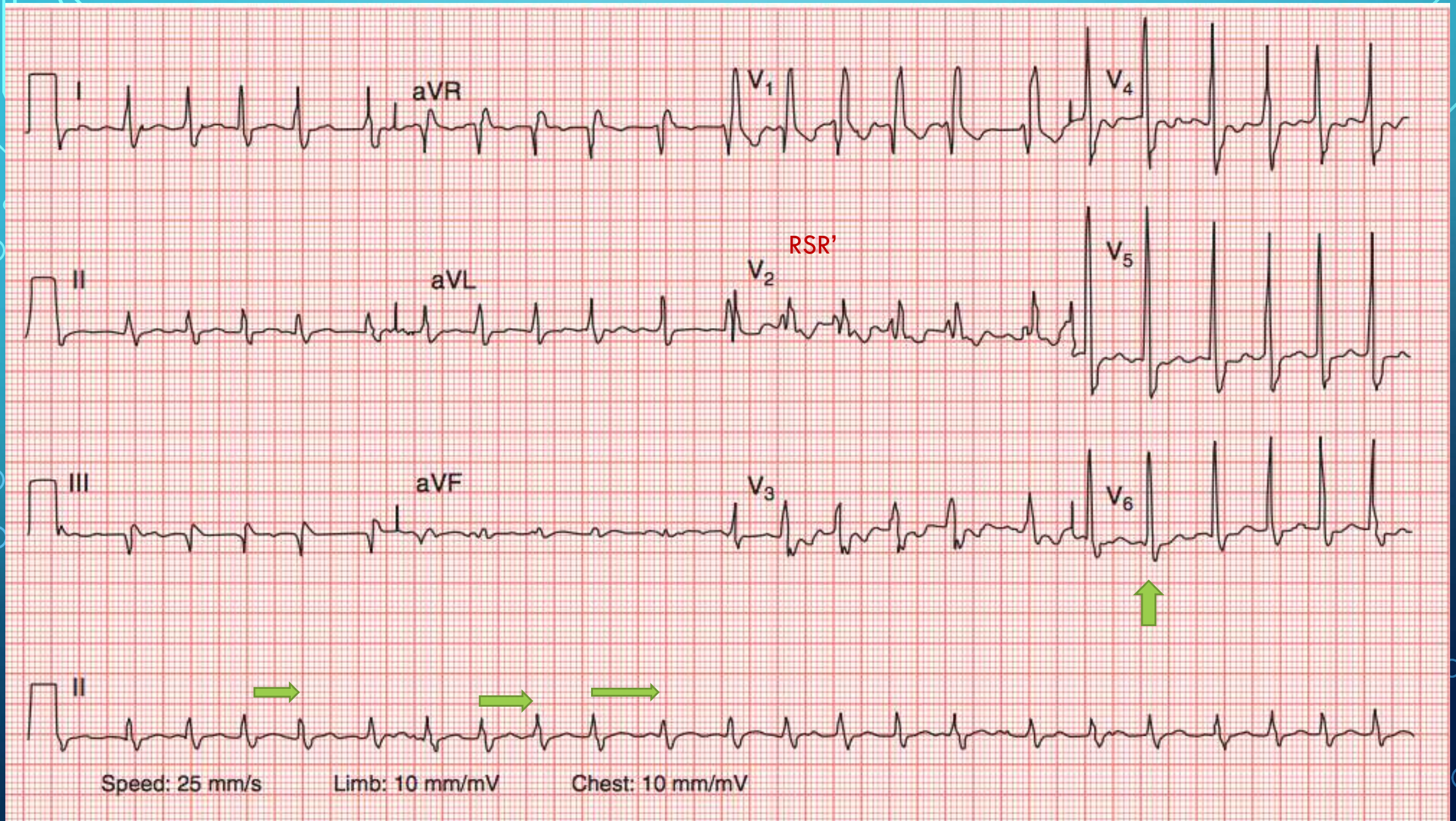
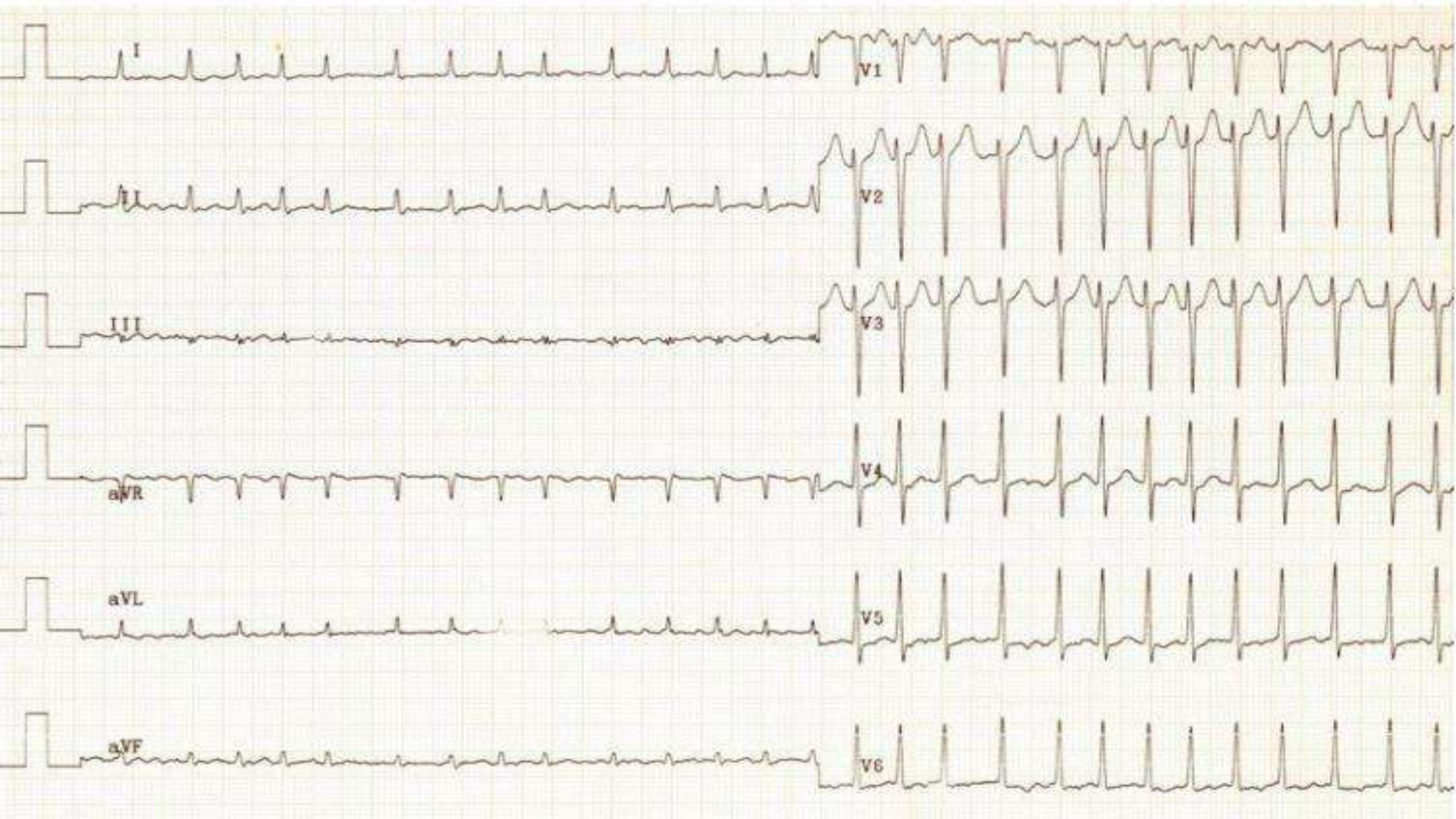
V3
V6
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV


I
aVR
V1
V4
II
aVL
V2
RSR'
V5
III
aVF
V3
V6
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

- The irregularly irregular rhythm with no discernible P waves means that this is atrial fibrillation (with a fast ventricular response). There is also right bundle branch block.


I
II
III
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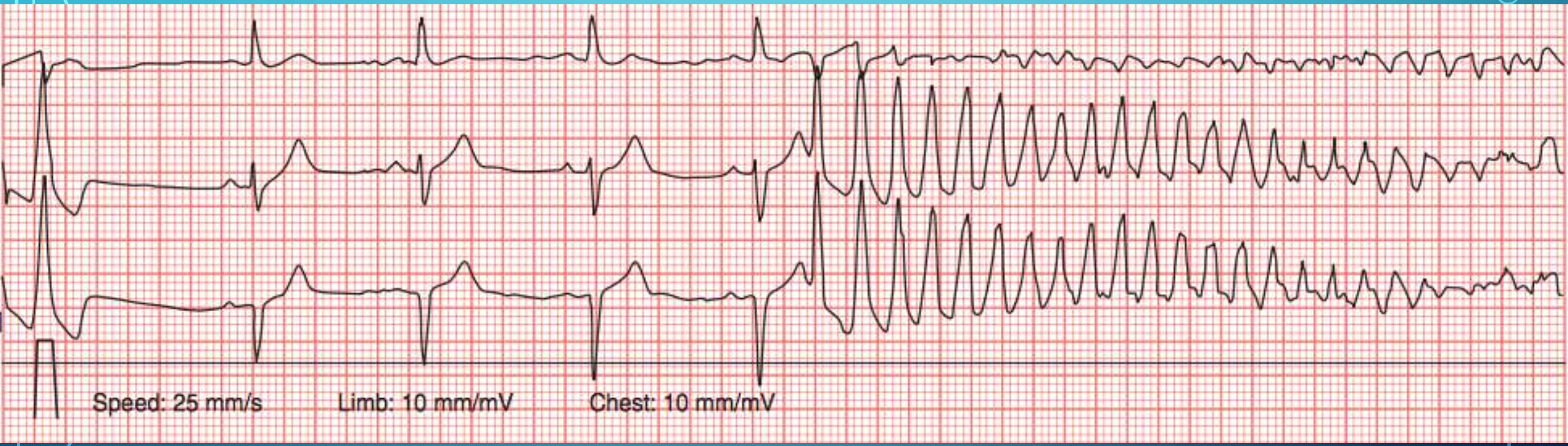
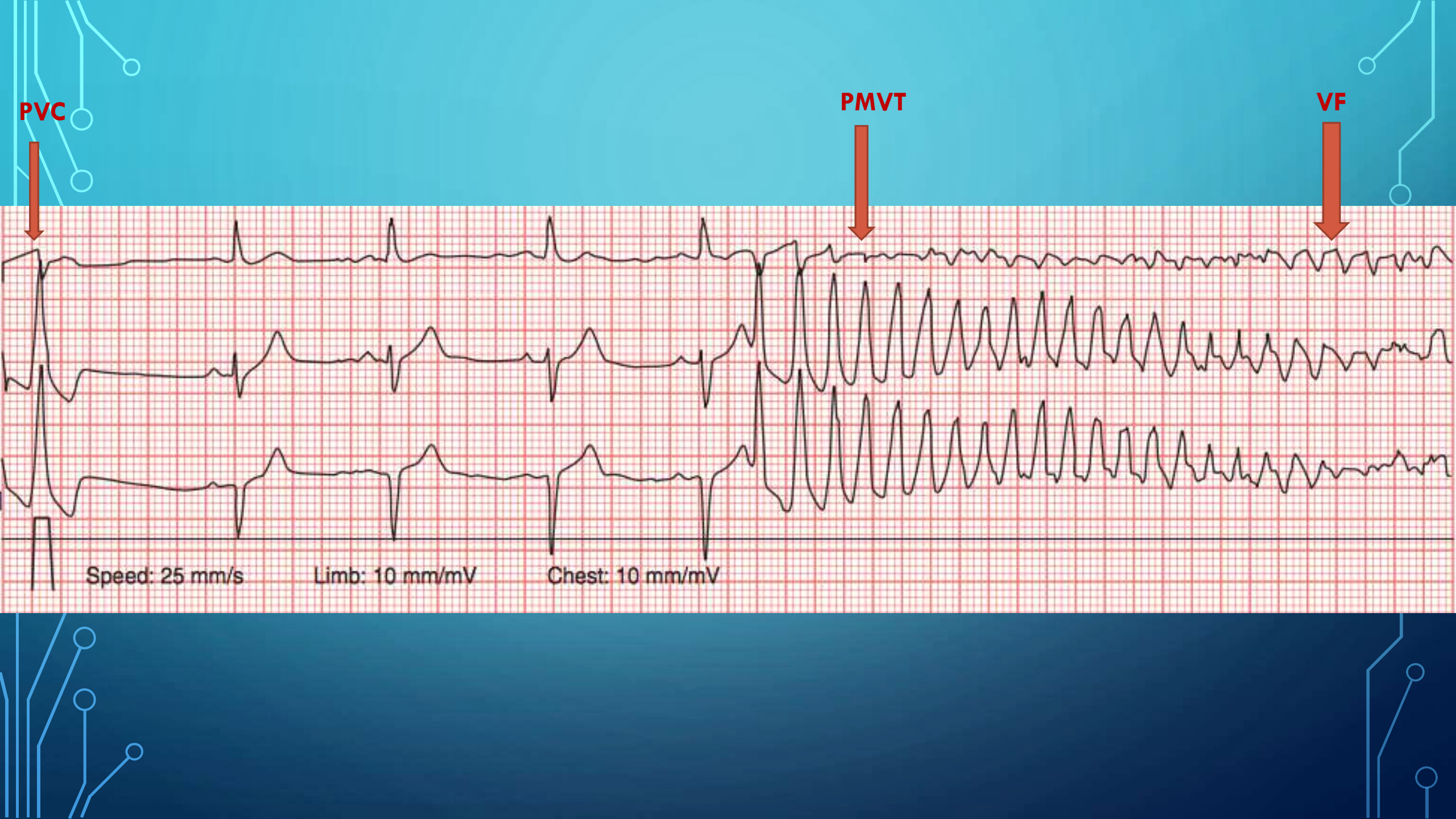
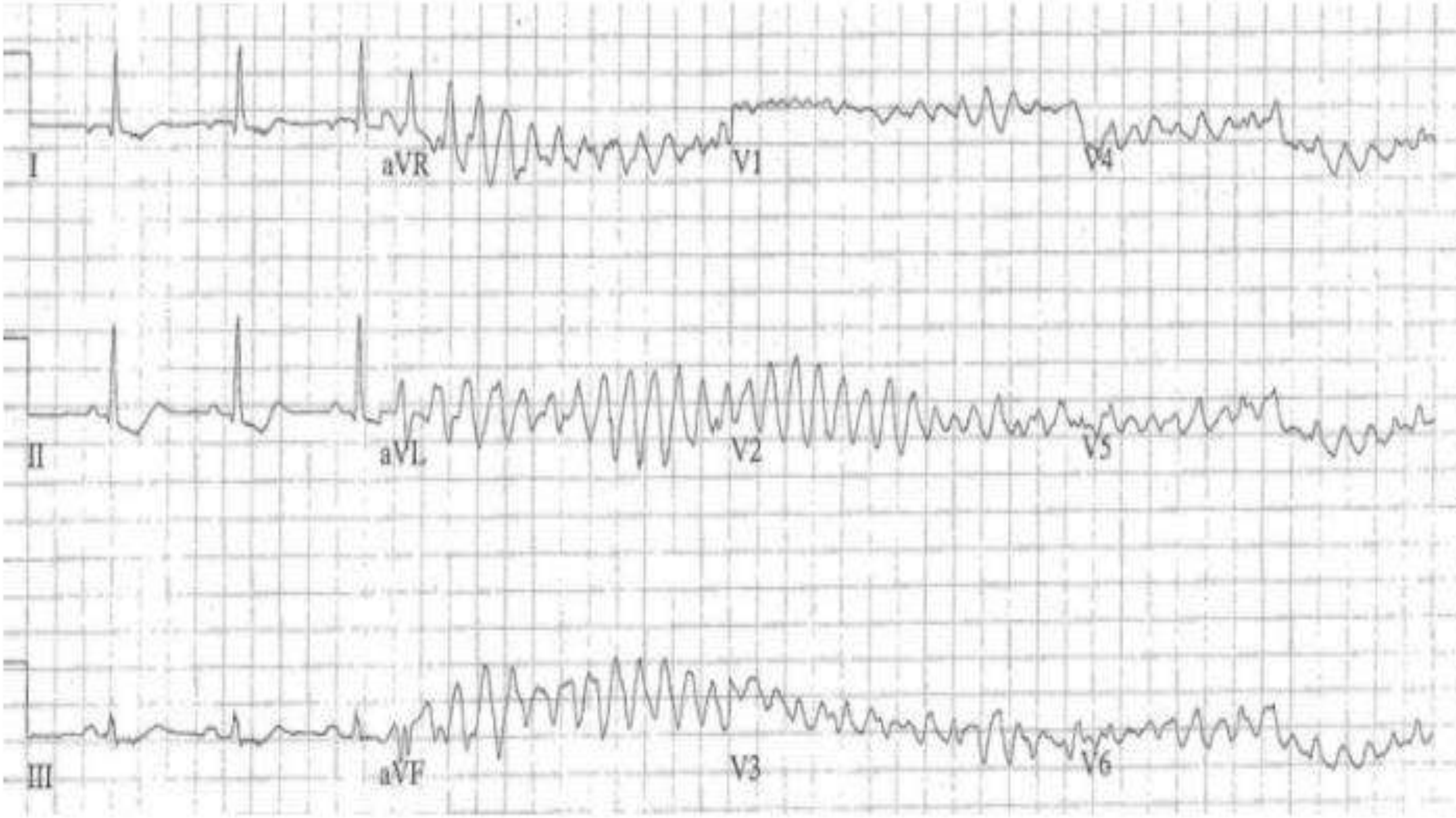
aVL
aVF
V1
V2
V3
V4
V5
V6


Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV


PVC
PMVT
VF
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

- Sinus rhythm with ventricular ectopics, followed by ventricular tachycardia (VT) which rapidly degenerates into ventricular fibrillation (VF)

- The ventricular tachycardia is triggered by a ventricular ectopic beat occurring during the T wave of the fourth sinus beat (R on T ectopic).


I
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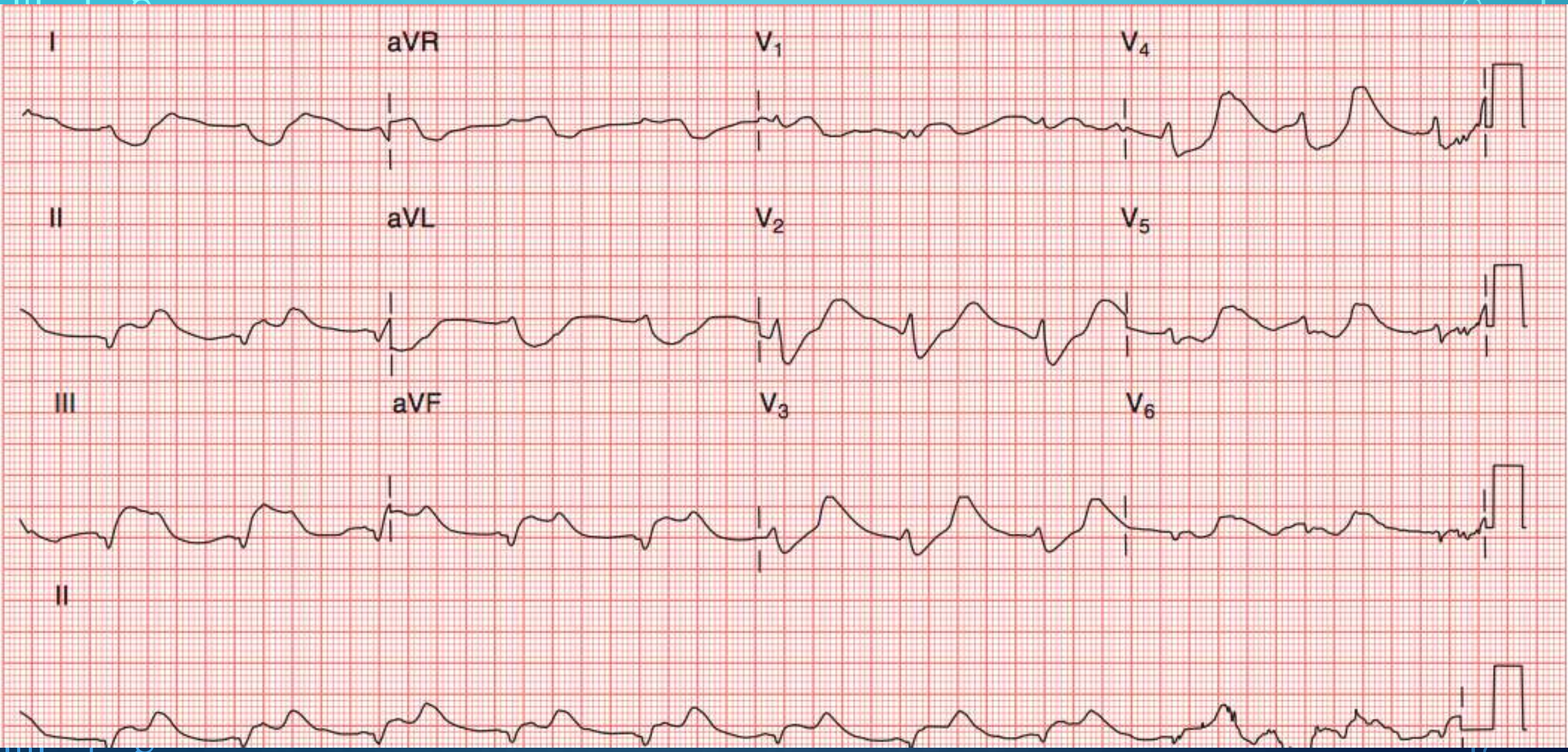
V4
II
aVL
V2
V5
III
aVF
V3
V6


I
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aVL
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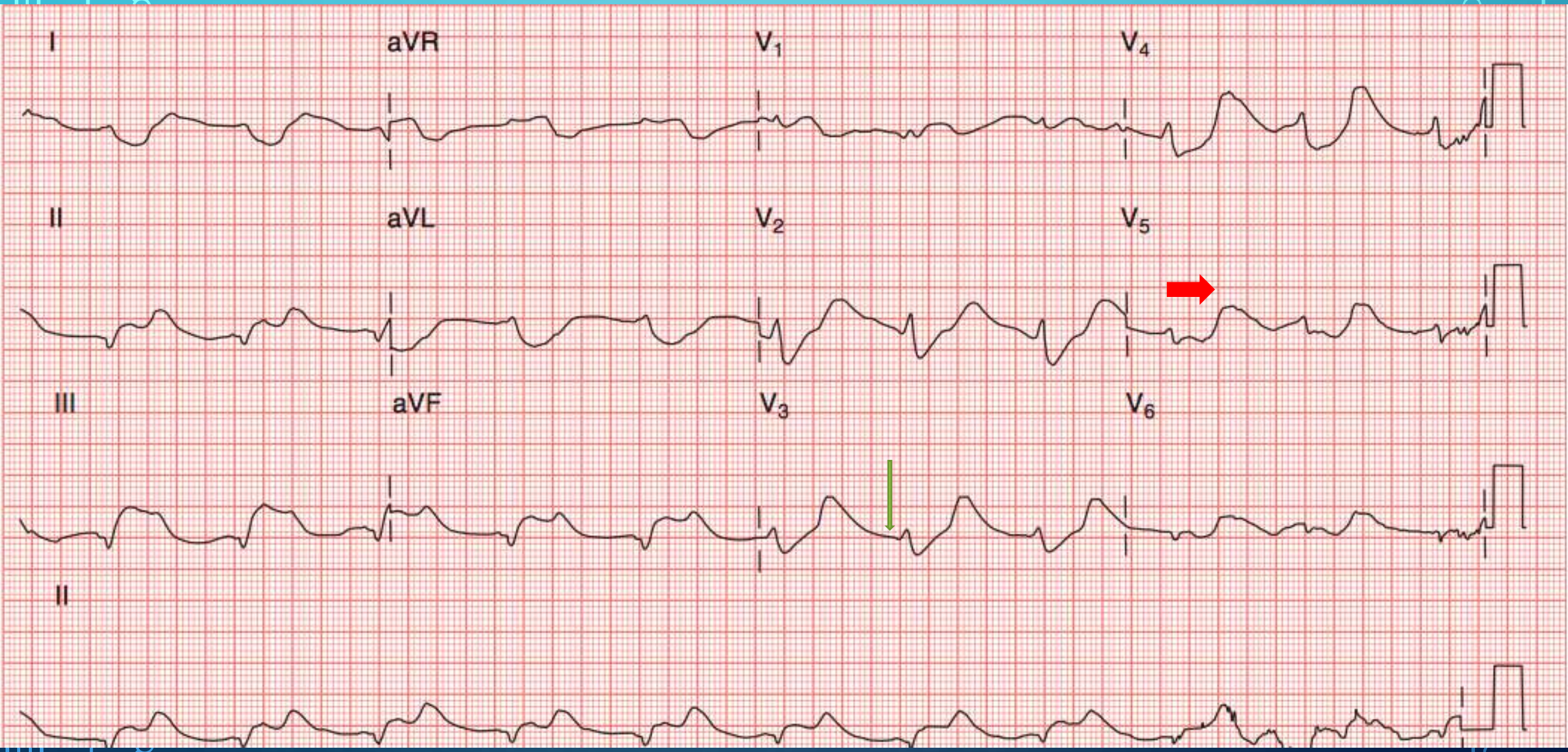
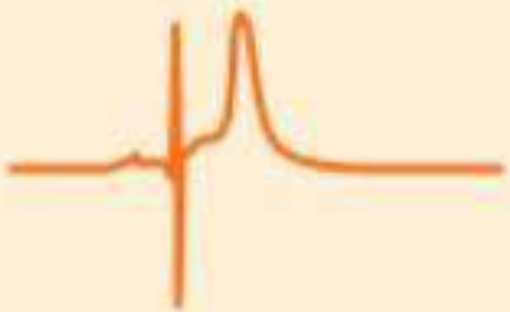
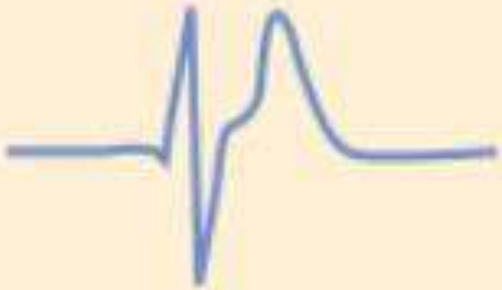
V5
III
aVF
V3
V6
II


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

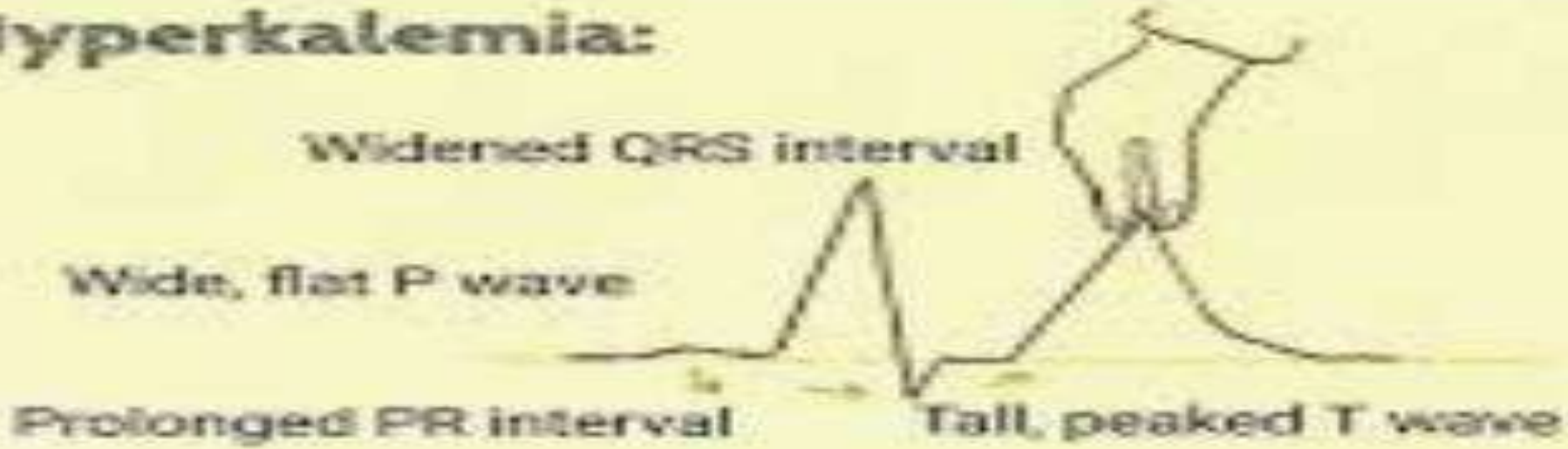
- junctional rhythm

  This ECG shows absent P waves and broad, bizarre QRS complexes. With increasing potassium levels, the P waves become smaller in size before disappearing altogether. Patients can also develop sinoatrial and atrioventricular block

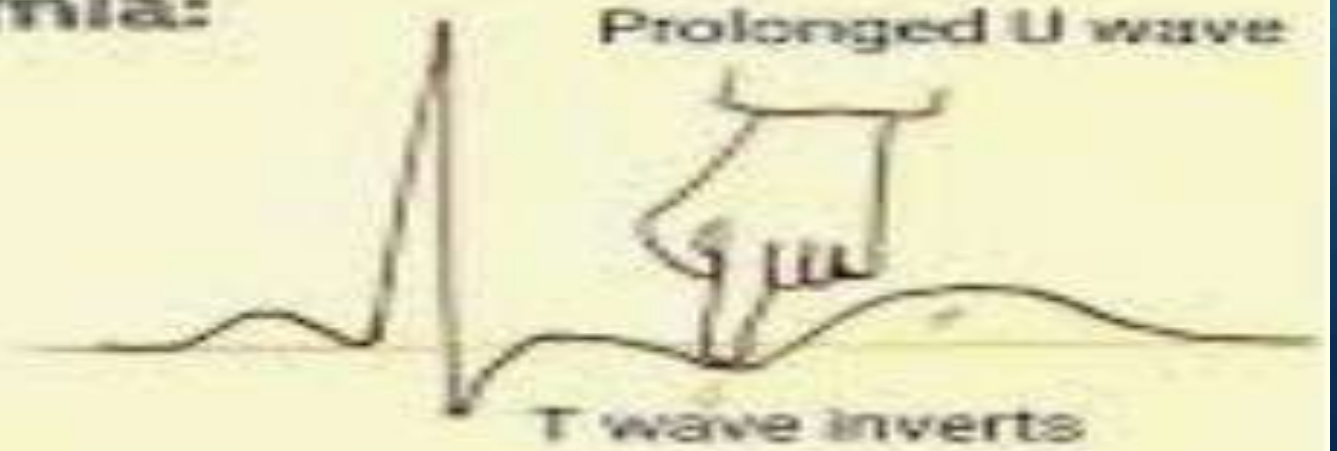
Serum potassium	Typical ECG appearance	Possible ECG abnormalities
Mild (5.5–6.5 mEq/L)		Peaked T waves Prolonged PR segment
Moderate (6.5–8.0 mEq/L)		Loss of P wave Prolonged QRS complex ST-segment elevation Ectopic beats and escape rhythms
Severe (>8.0 mEq/L)		Progressive widening of QRS complex Sine wave Ventricular fibrillation Asystole Axis deviations Bundle branch blocks Fascicular blocks

EpoMedicine

Hyperkalemia:



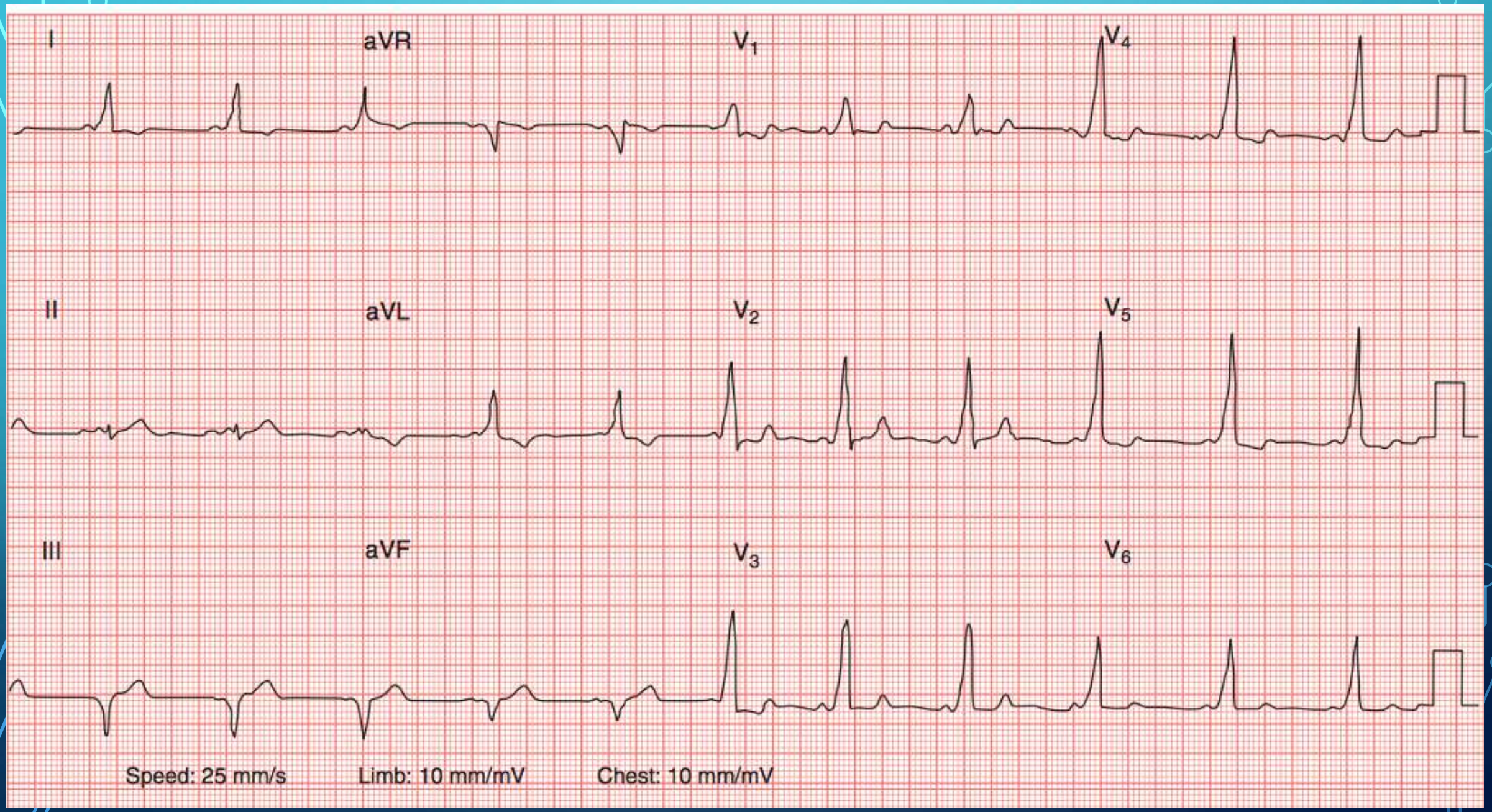
Hypokalemia:



" Pull and Push effects"

- ● early ECG changes include tall ‘tented’ T waves, shortening of the QT interval and ST segment depression
  ● at higher potassium levels, the QRS complexes become broad and there is lengthening of the PR interval (with flattening or even loss of the P wave) ● sinoatrial and atrioventricular block can develop ● at very high potassium levels, the QRS complexes become increasingly bizarre and merge with the T waves to resemble a sine wave
- ● Hyperkalaemia needs urgent treatment if it is causing ECG abnormalities or the plasma potassium level is above 6.5 mmol/L.
- ● arrhythmias (including ventricular fibrillation and
- asystole) can occur at any point

Potassium level (mmol/L)	Mechanism	ECG changes
5.5 – 6.5	Repolarisation abnormalities	Peaked T waves
6.5 – 7.0	Progressive atrial paralysis	P wave widening/flattening PR prolongation P waves eventually disappear
7.0 – 9.0	Conduction abnormalities	**Bradyarrhythmias**: Sinus bradycardia; high-grade AV block with slow junctional and ventricular escape rhythms; slow AF Conduction blocks (bundle branch block, fascicular blocks) Prolonged QRS interval with bizarre QRS morphology
> 9.0	All of above	Development of sine wave appearance (pre-terminal rhythm) Asystole Ventricular fibrillation PEA with bizarre, wide complex rhythm


I
aVR
V1
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aVL
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III
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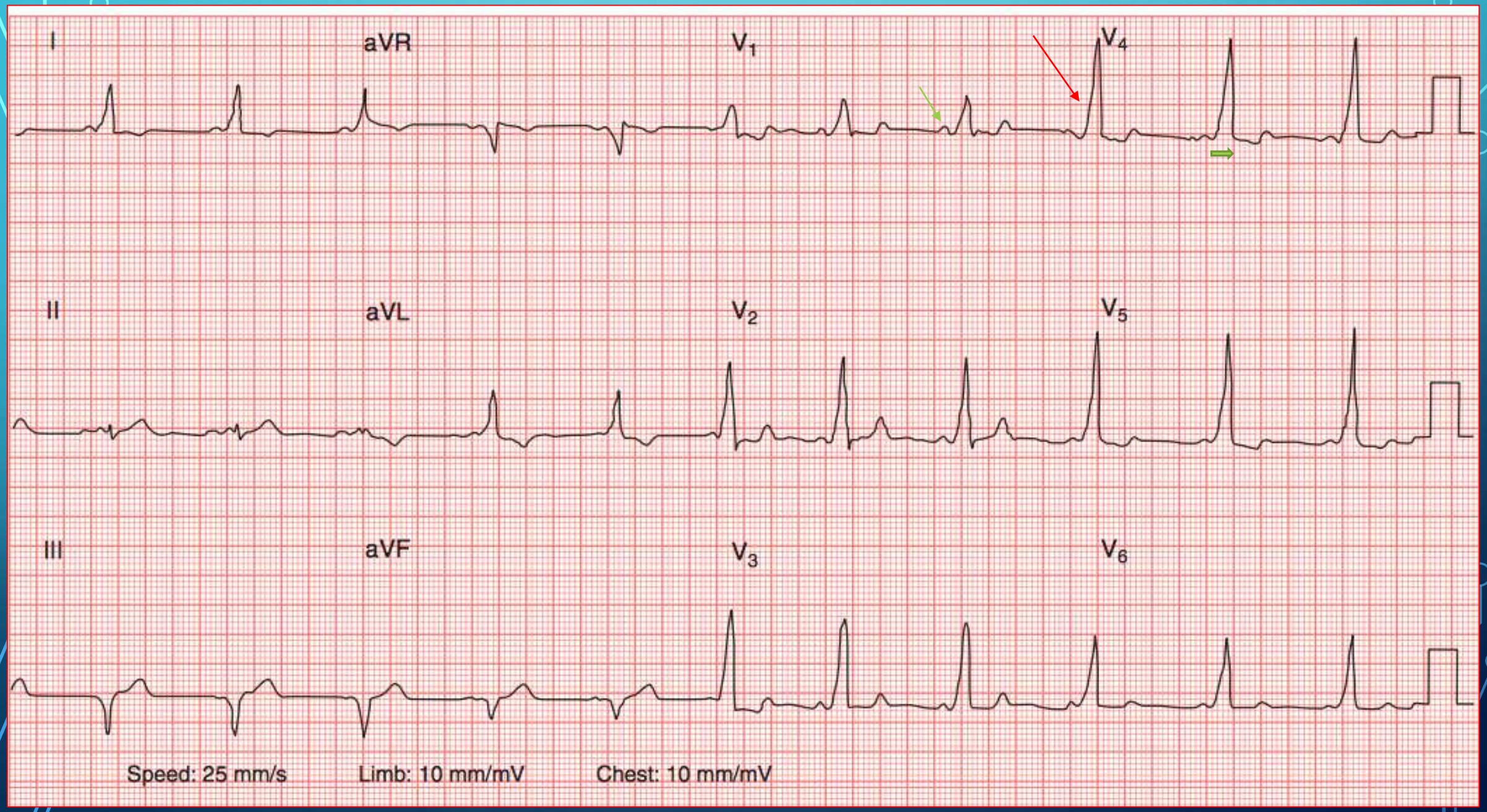
V3
V6
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV


I
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III
aVF
V3
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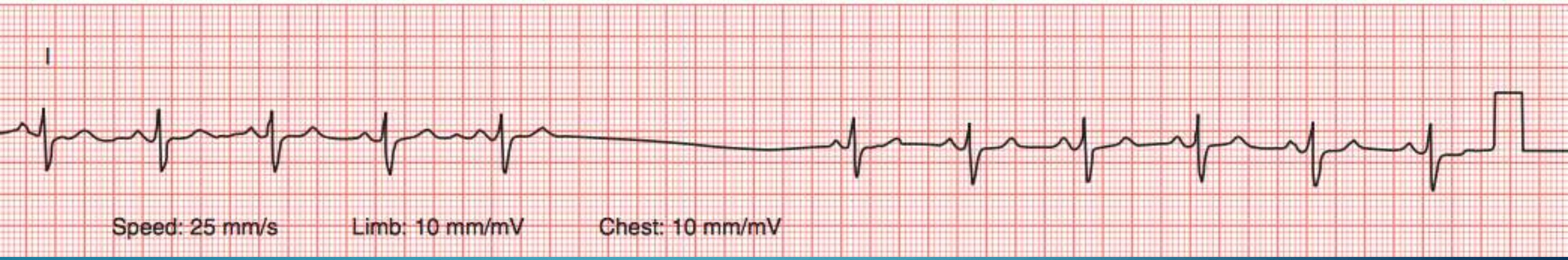
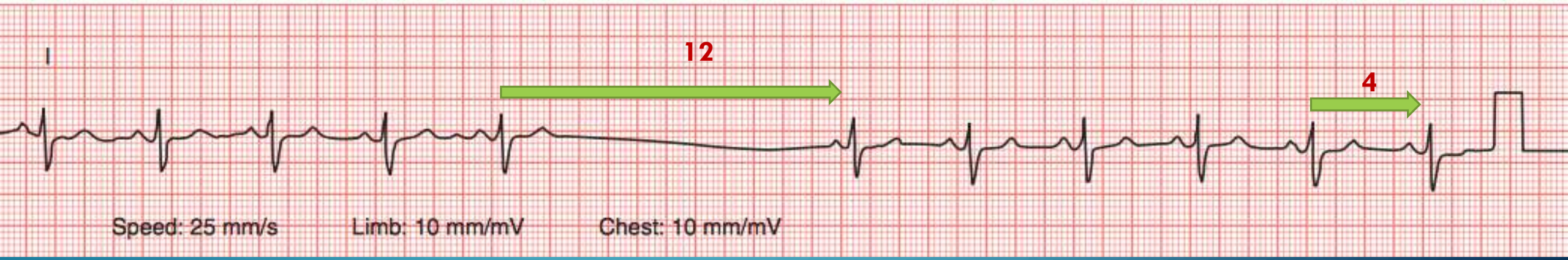
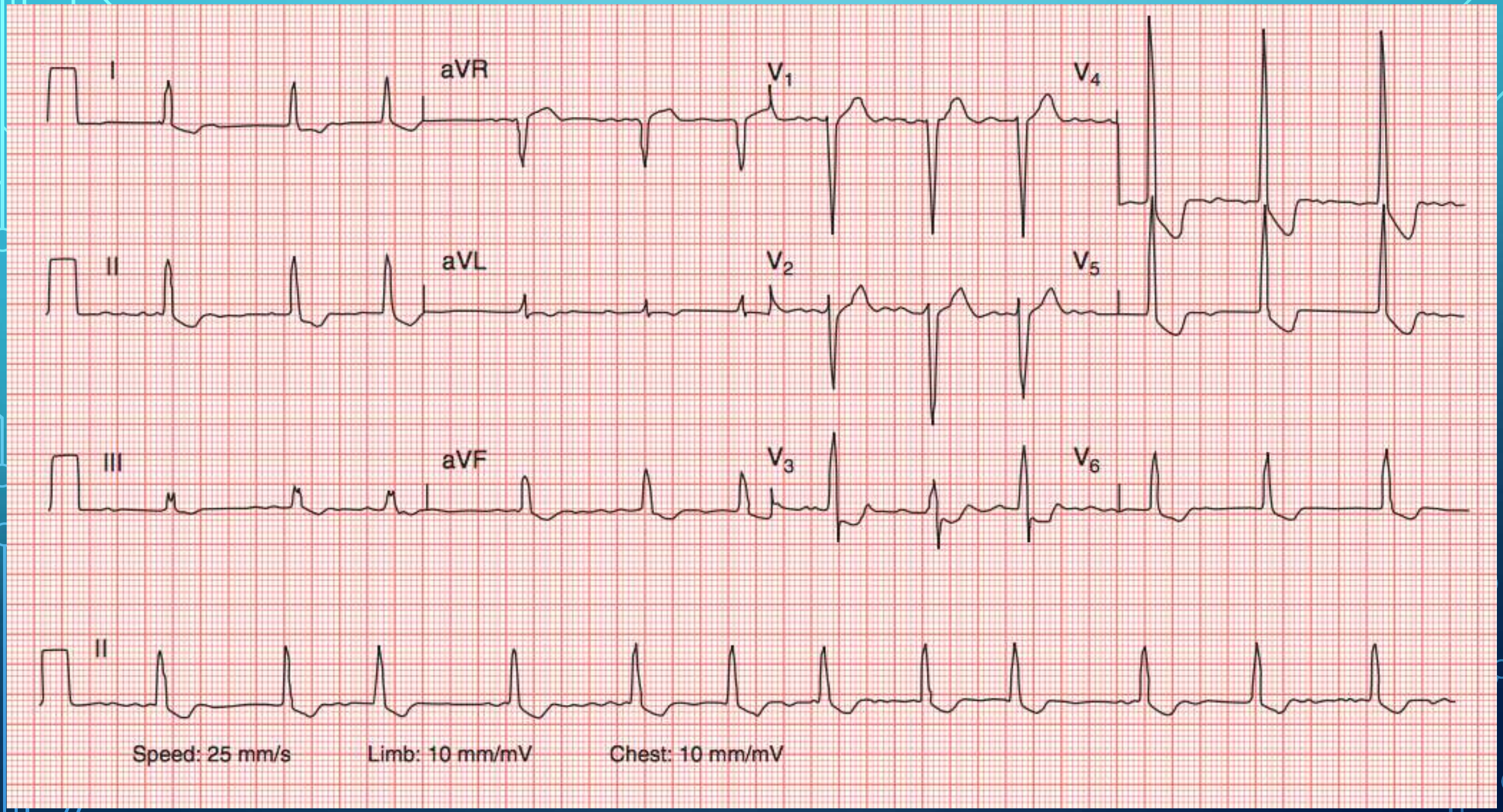
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

- A P wave precedes every QRS complex so the rhythm is sinus rhythm. However, the PR interval is short, and there is slurring of the initial part of the QRS complex producing a delta wave, clearly visible in leads I, aVL and V1–V6. This is Wolff–Parkinson–White (WPW) syndrome


I
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV


I
12
4
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

- The underlying rhythm is normal sinus rhythm but then a P wave fails to appear; the next P wave appears after a pause of 2.4 s. The R-R interval is 0.8 s, so the P wave has arrived ‘on schedule’, three complete cycle lengths after the last P wave. This is sinoatrial node exit block


I
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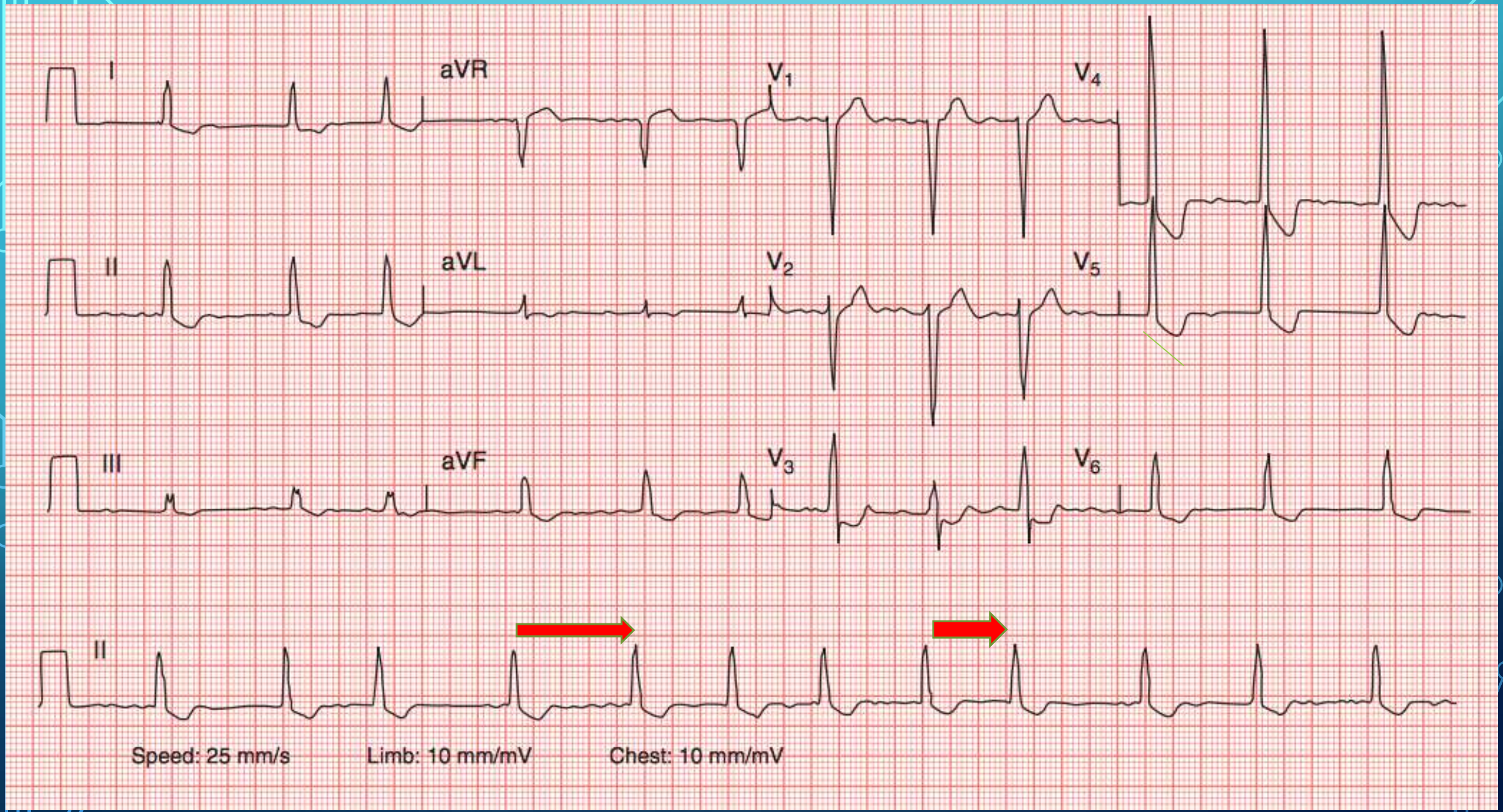
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV


I
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III
aVF
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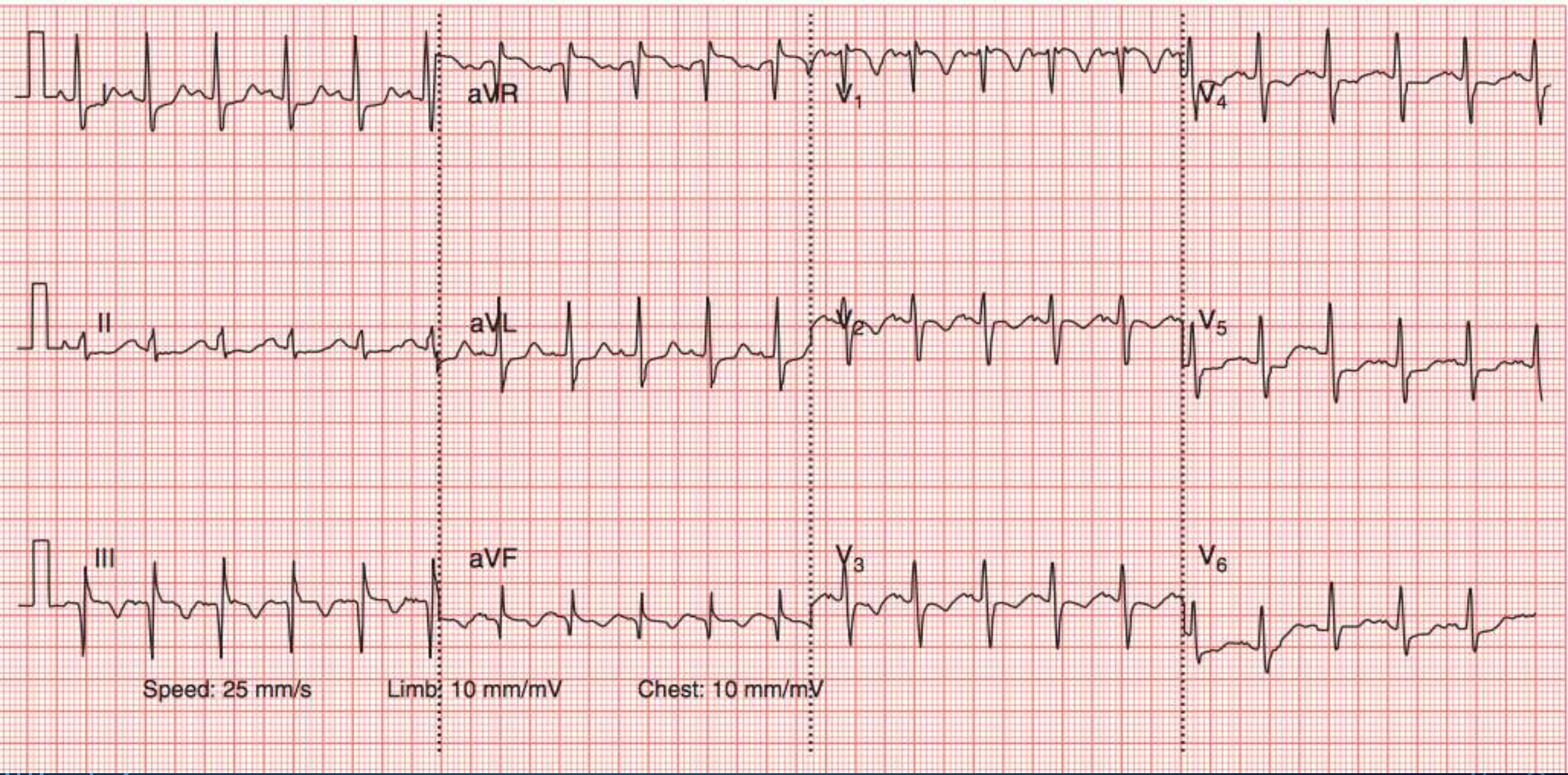
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

- The rhythm is irregularly irregular with no discernible P waves (atrial fibrillation). The QRS complexes are normal but the ST segments are downward-sloping with a ‘reverse tick’ morphology: this is typical (although not diagnostic) of digitalis (digoxin) effect.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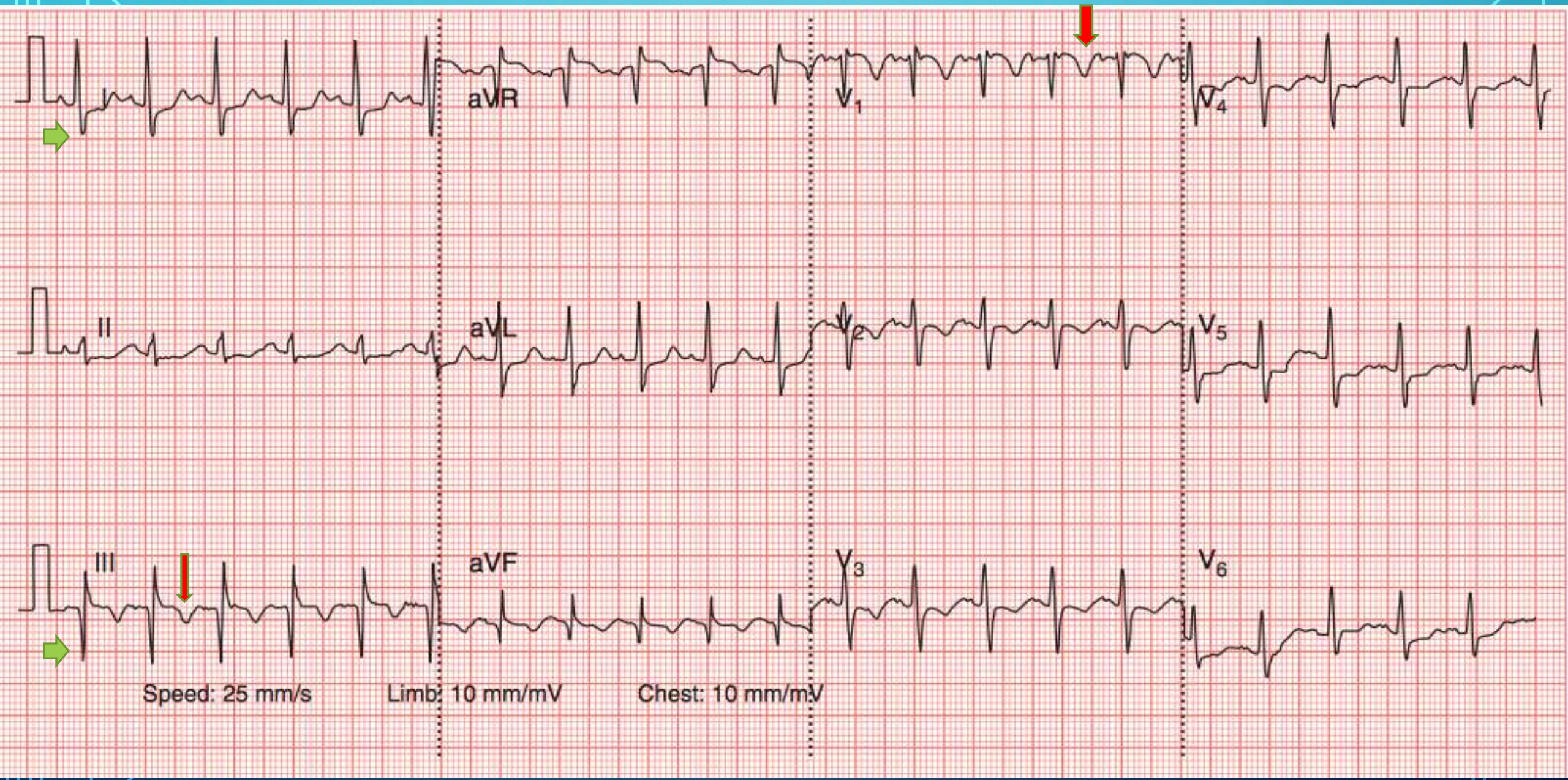
aVF
V3
V6
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV


aVR
V1
V4
II
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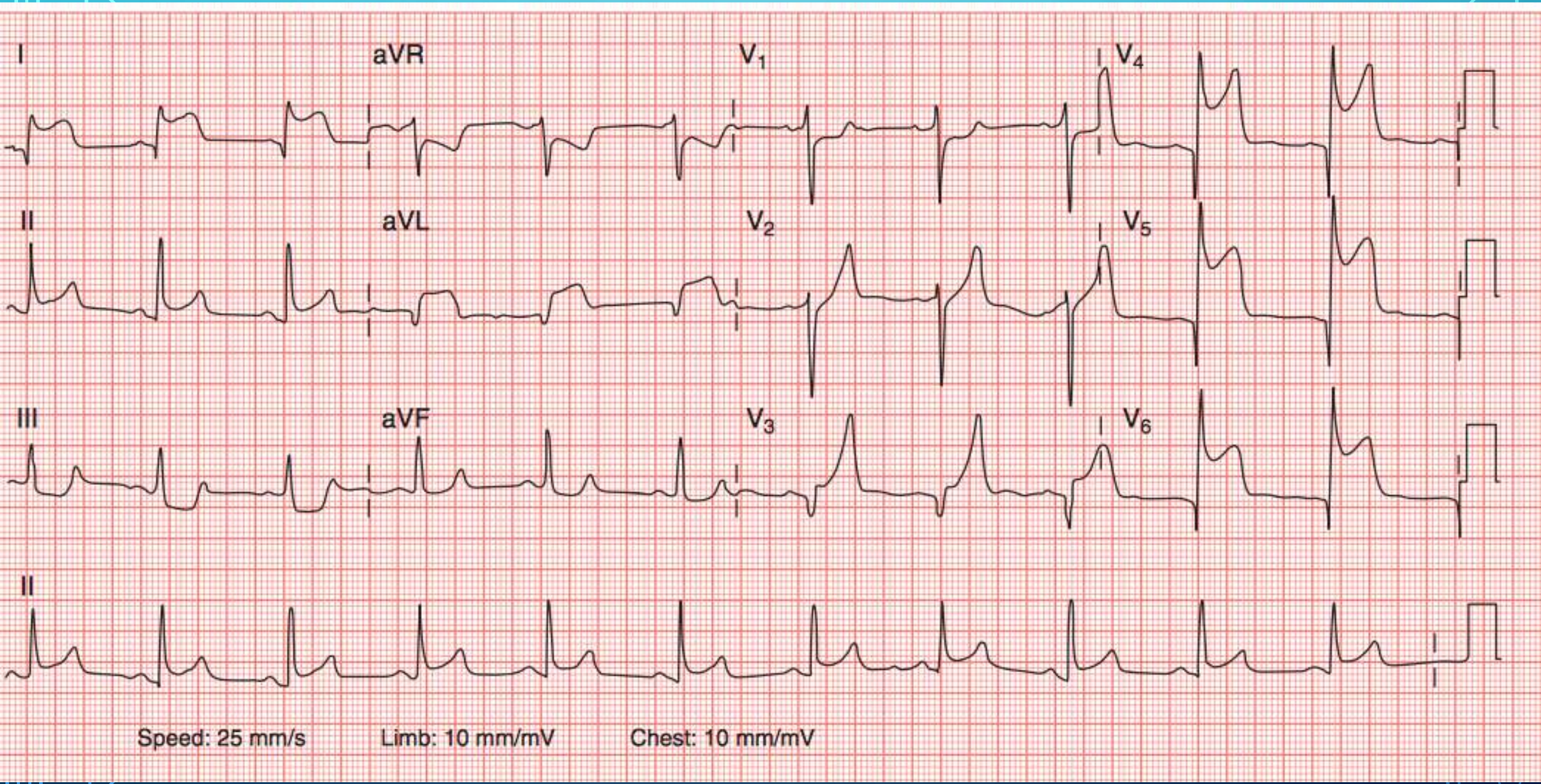
V5
III
aVF
V3
V6
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

- sinus tachycardia
  - an S wave in lead I, and a Q wave and an inverted T wave in lead III (S1Q3T3)
  - anterior T wave inversion.
- Acute pulmonary embolism


I
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Speed: 25 mm/s
Limb: 10 mm/mV
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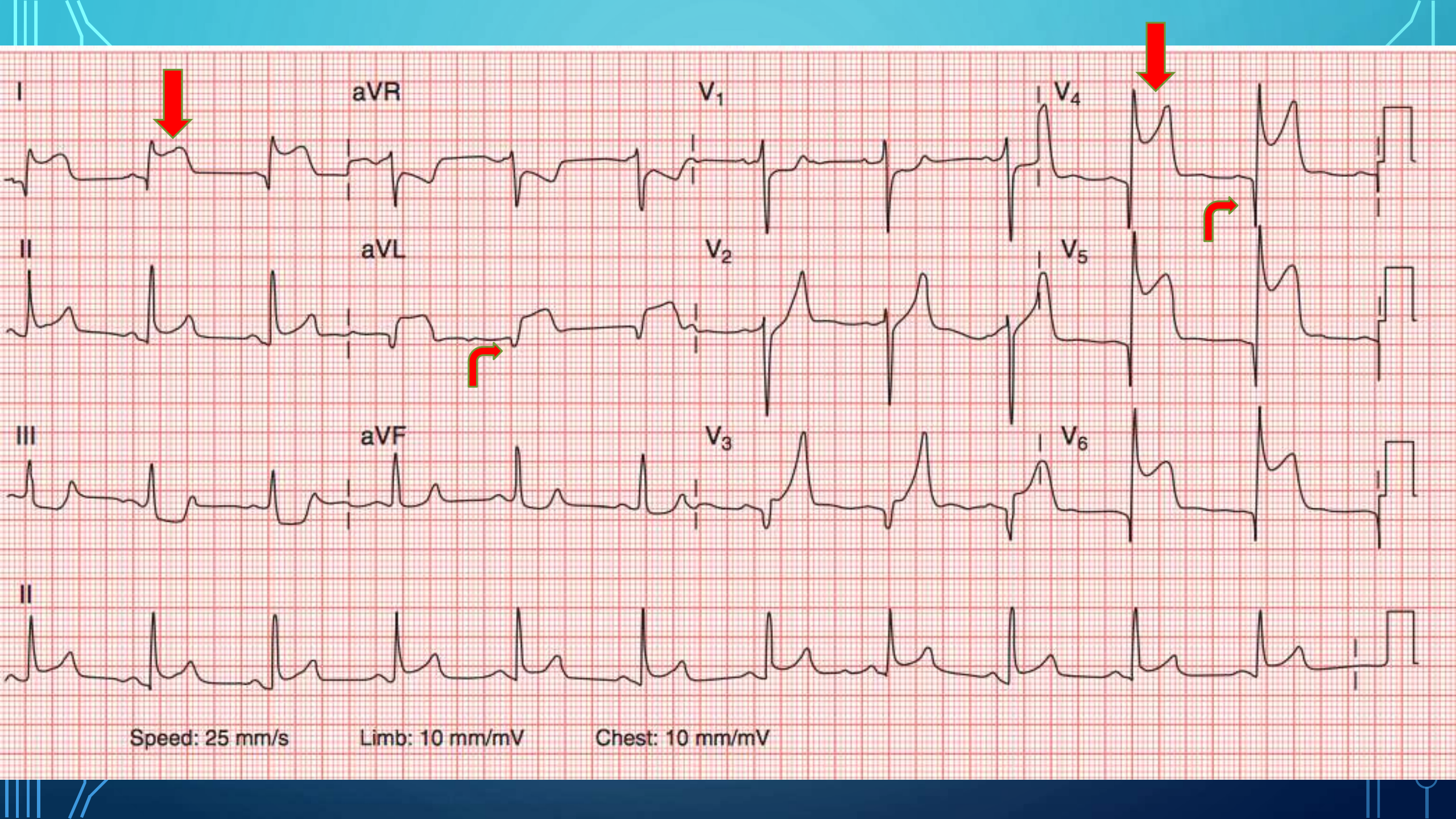
I
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V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

- There is ST segment elevation in limb leads I and aVL and chest leads V3–V6. This is an acute anterolateral ST elevation myocardial infarction (STEMI).

ST SEGMENT

ST-ELEVATION MI (STEMI)

0 HOUR

Pronounced T Wave initially
ST elevation (convex type)

1-24H

Depressed R Wave, and Pronounced T Wave. Pathological Q waves may appear within hours or may take greater than 24 hr.- indicating full-thickness MI. Q wave is pathological if it is wider than 40 ms or deeper than a third of the height of the entire QRS complex

Day 1-2

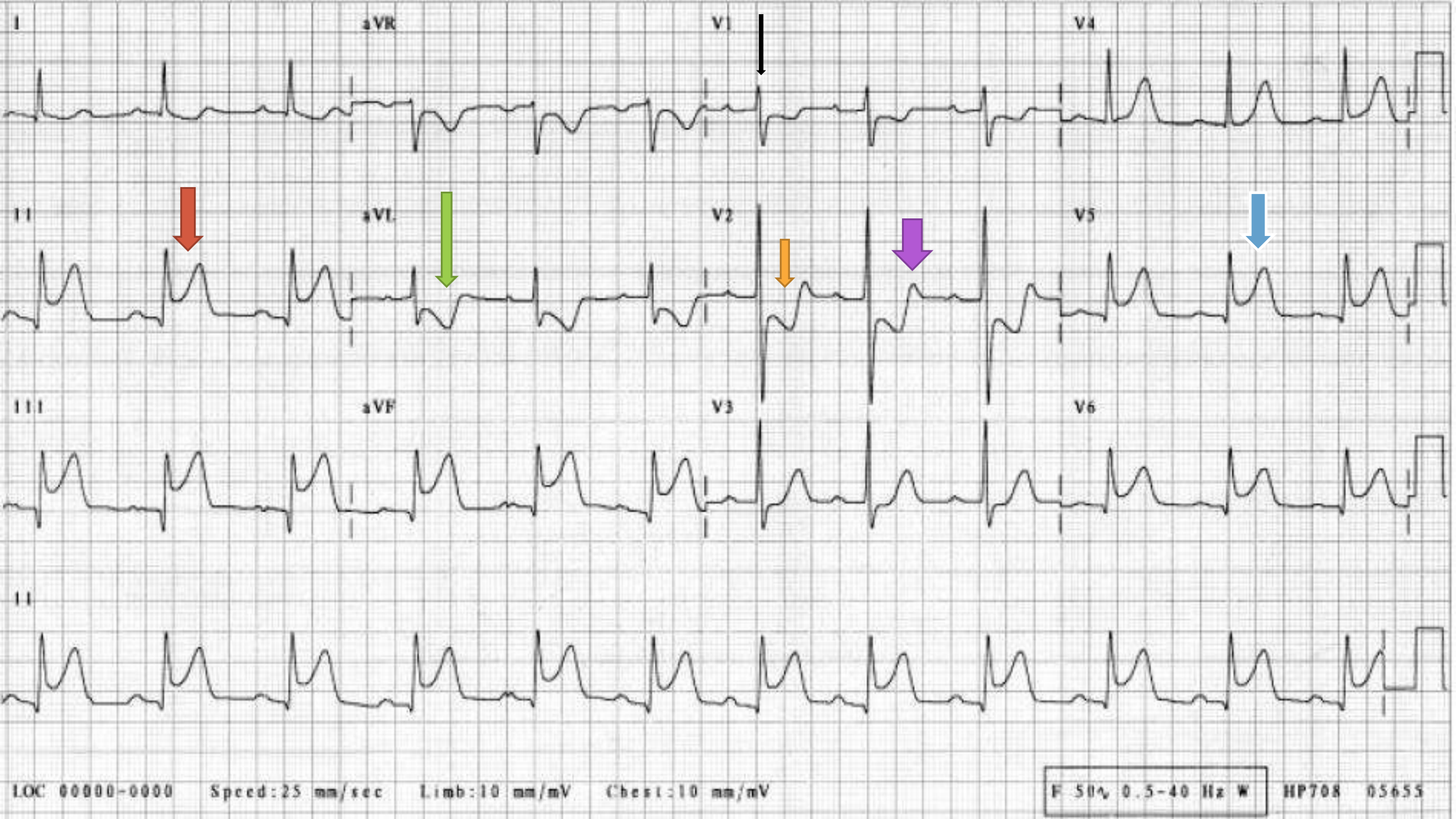
Exaggeration of T Wave continues for 24h.

Days later

T Wave inverts as the ST elevation begins to resolve. Persistent ST elevation is rare except in the presence of a ventricular aneurysm.

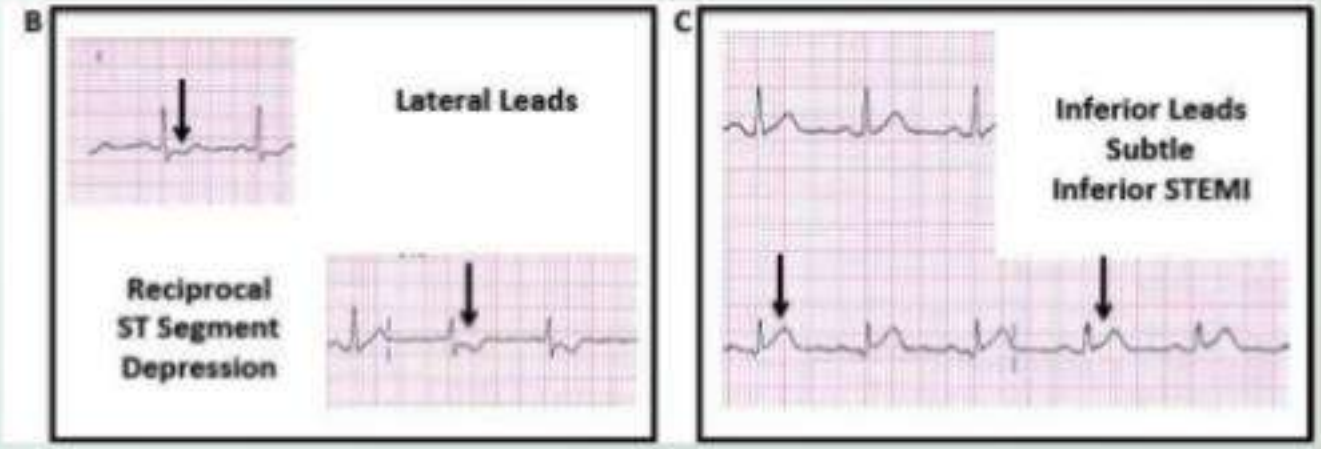
Weeks later

ECG returns to normal T wave, but retains pronounced Q wave. An old infarct may look like this


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
LOC 00000-0000
Speed:25 mm/sec
Limb:10 mm/mV
Chest:10 mm/mV
F 50∿ 0.5-40 Hz W
HP708
05655

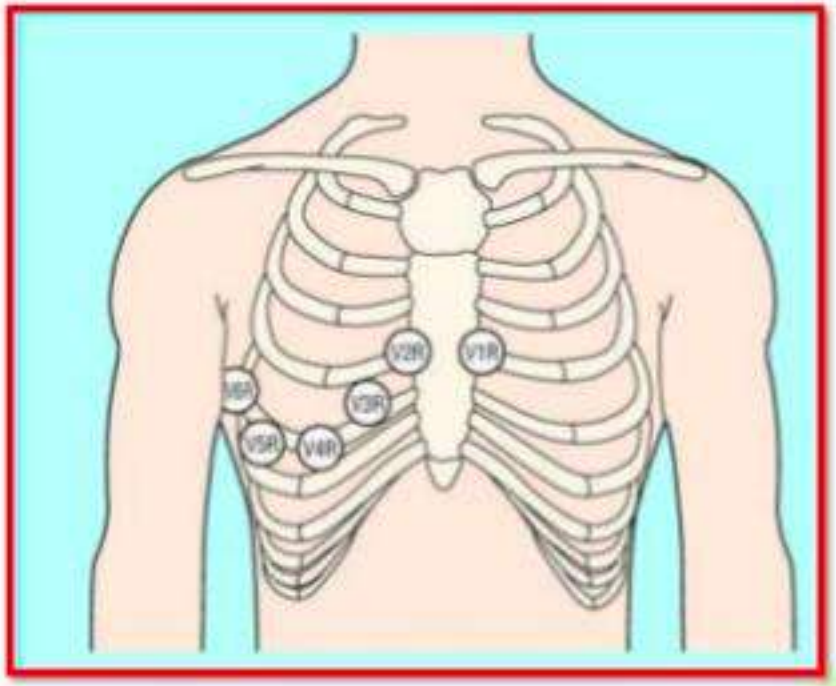
Examples on reciprocal changes :

Type of MI	Reciprocal changes (ST depression)
Inferior MI	In lead 1 & aVL
Lateral MI	In lead2, lead3 & aVF

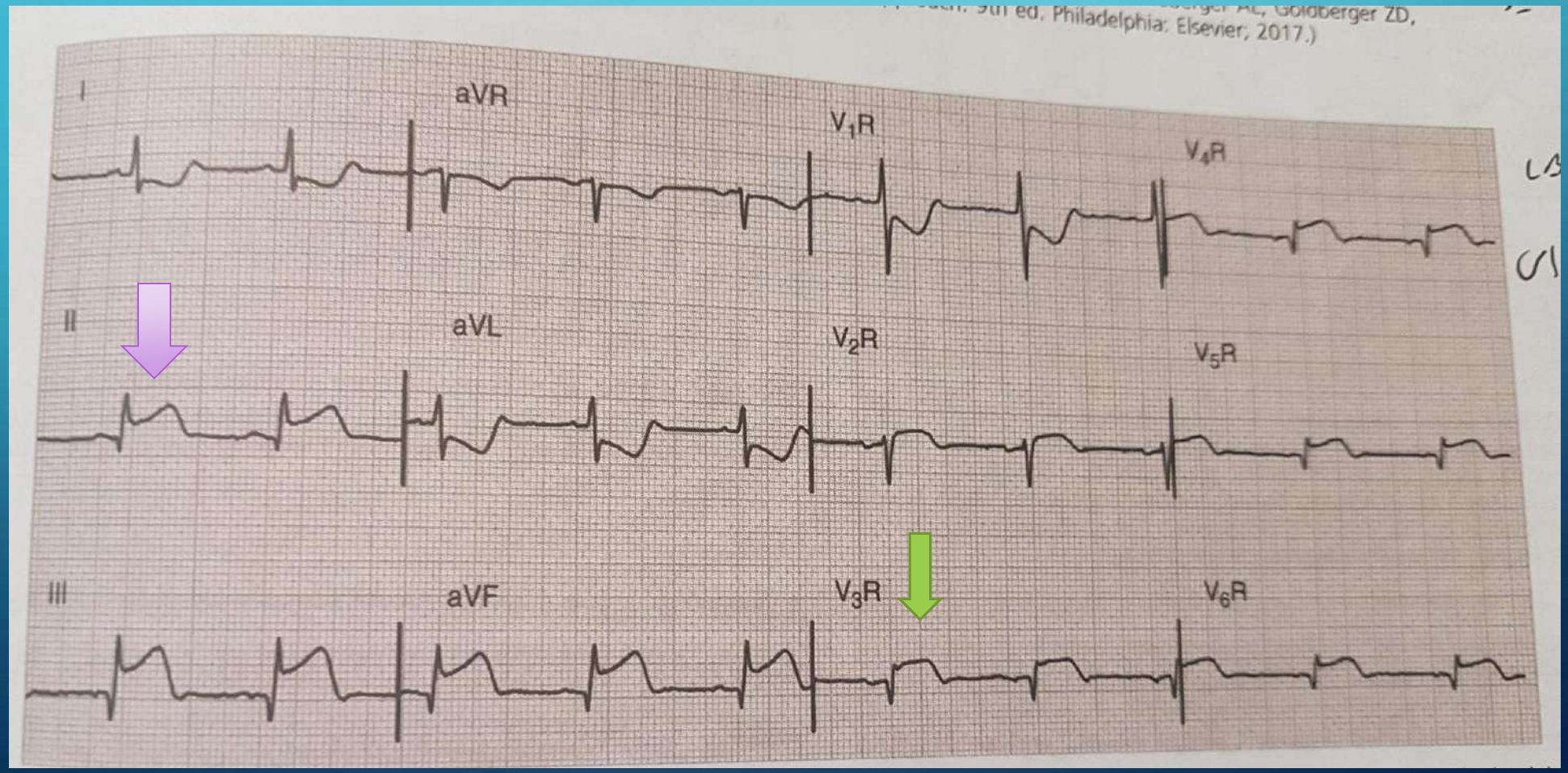


Right-Sided Leads

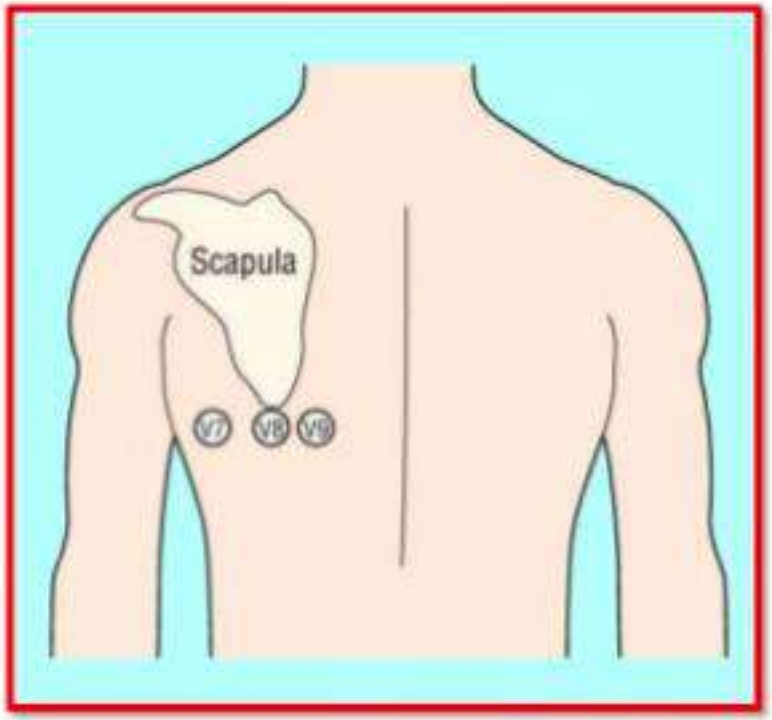
- *Right ventricular infarction is confirmed by the presence of ST elevation in the right-sided leads (V3R-V6R).*
- *ST elevation in V4R has a sensitivity of 88%, specificity of 78% and diagnostic accuracy of 83% in the diagnosis of RV MI.*
- *ST elevation in the right-sided leads is a transient phenomenon, lasting less than 10 hours in 50% of patients with RV infarction.*



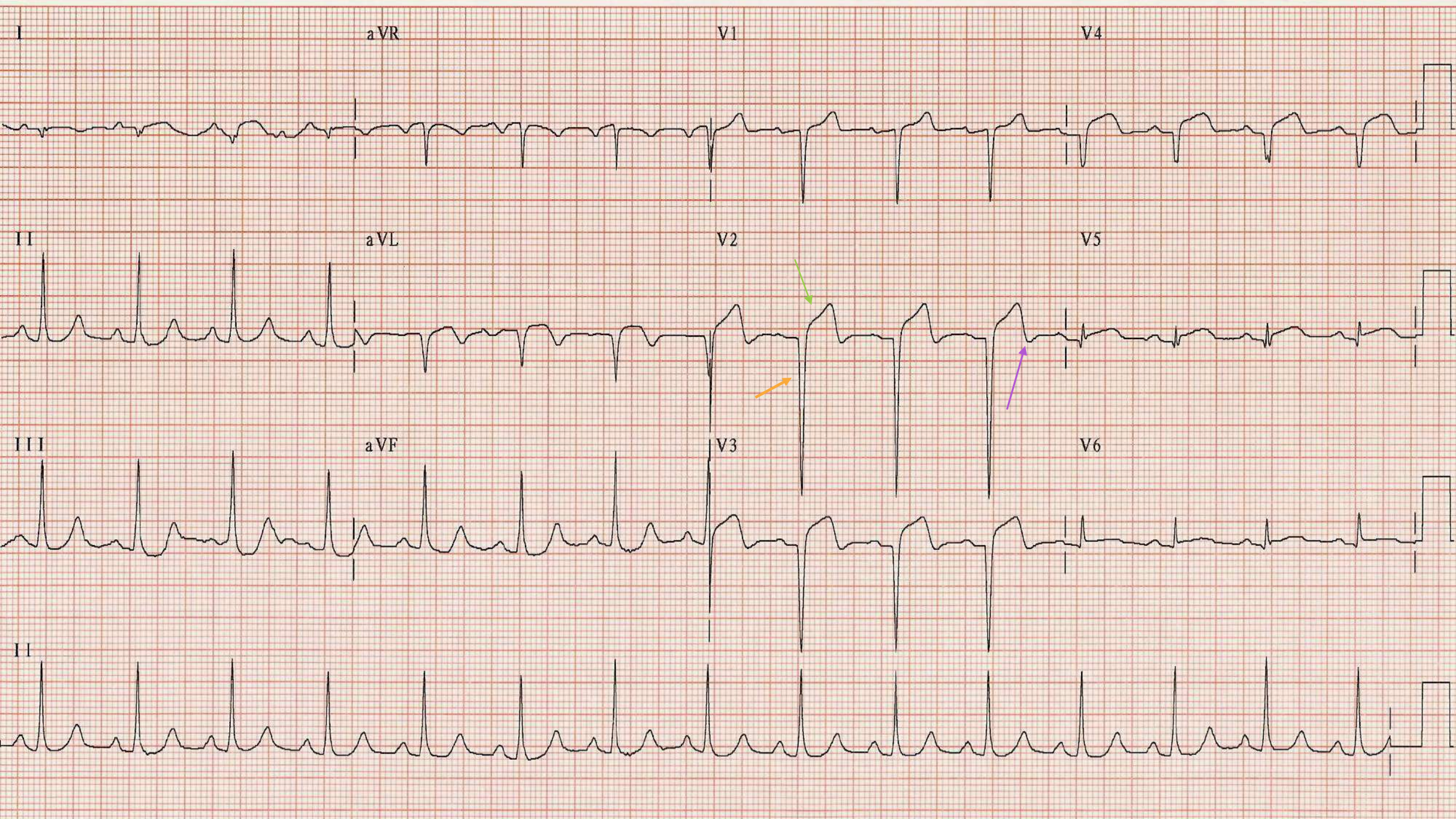
RV INF MI

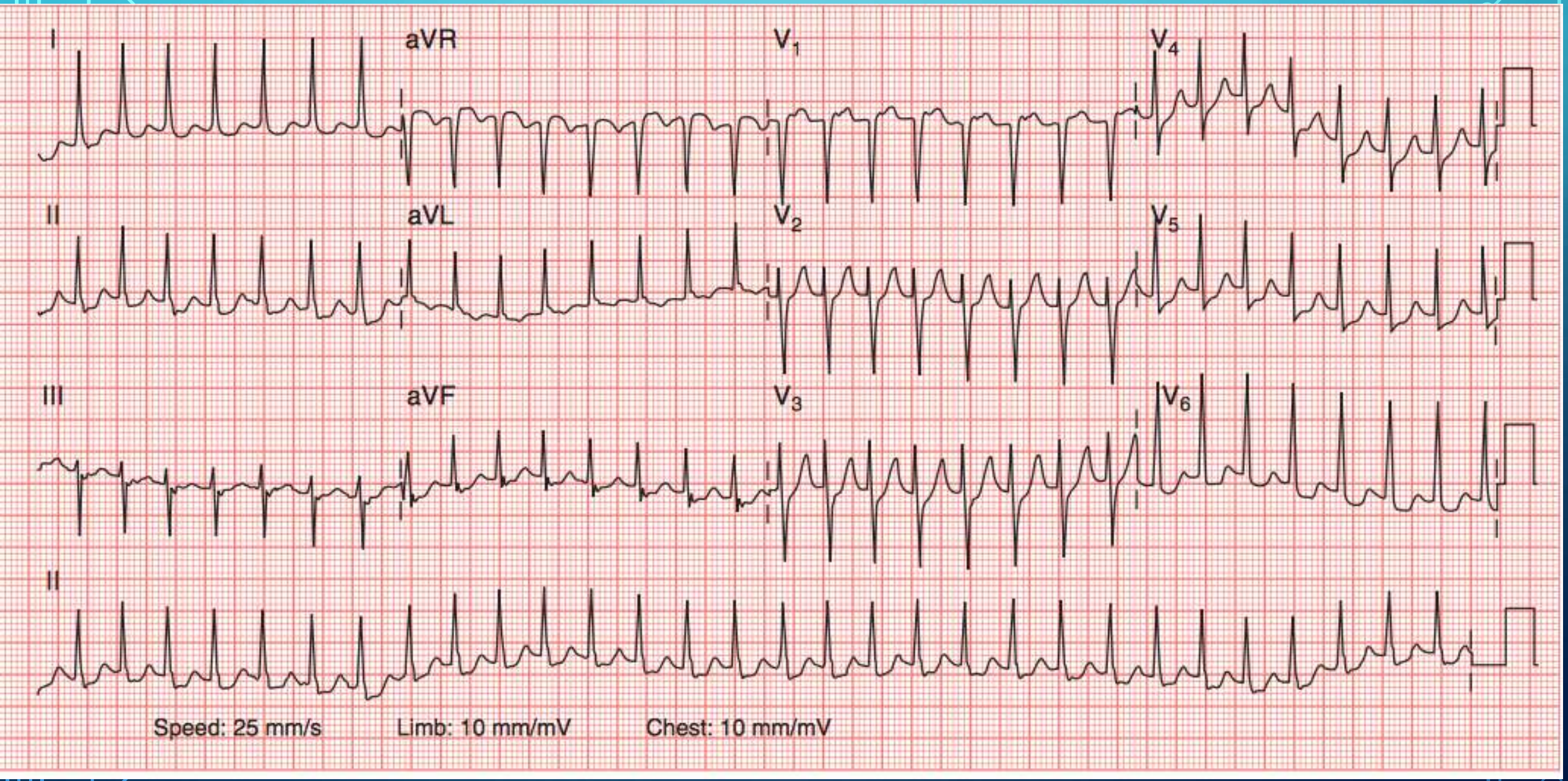

ed. Philadelphia: Elsevier; 2017.)
I
aVR
V1R
V4R
II
aVL
V2R
V5R
III
aVF
V3R
V6R

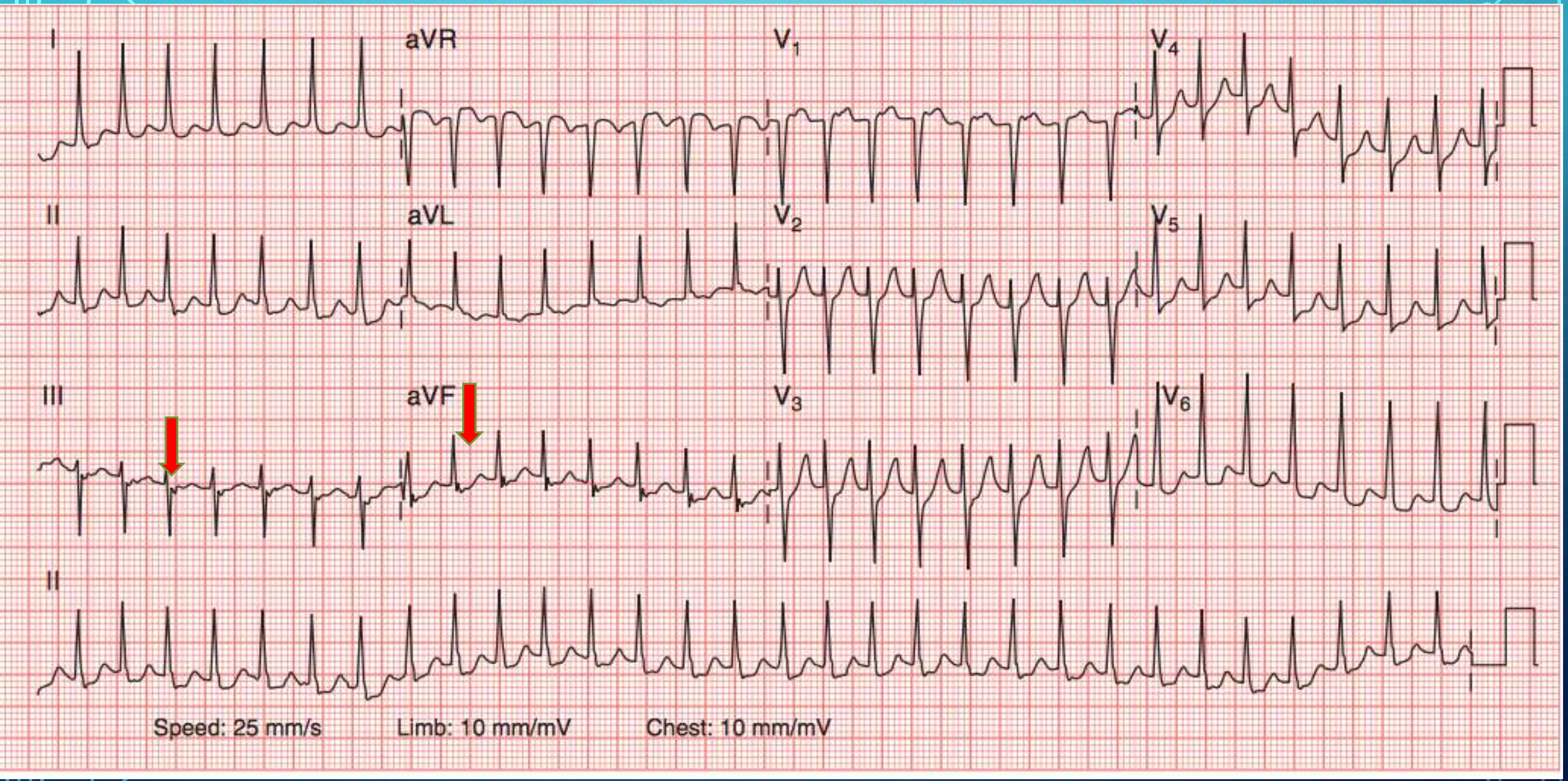
- **Posterior Leads**
- Leads V7-9 are placed on the posterior chest wall in the following positions.
- V7 – Left posterior axillary line, in the same horizontal plane as V6.
- V8 – Tip of the left scapula, in the same horizontal plane as V6.
- V9 – Left paraspinal region, in the same horizontal plane as V6.



The degree of ST elevation seen in V7-9 is typically modest – note that only 0.5 mm of ST elevation is required to make the diagnosis of posterior MI


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


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

AVNRT

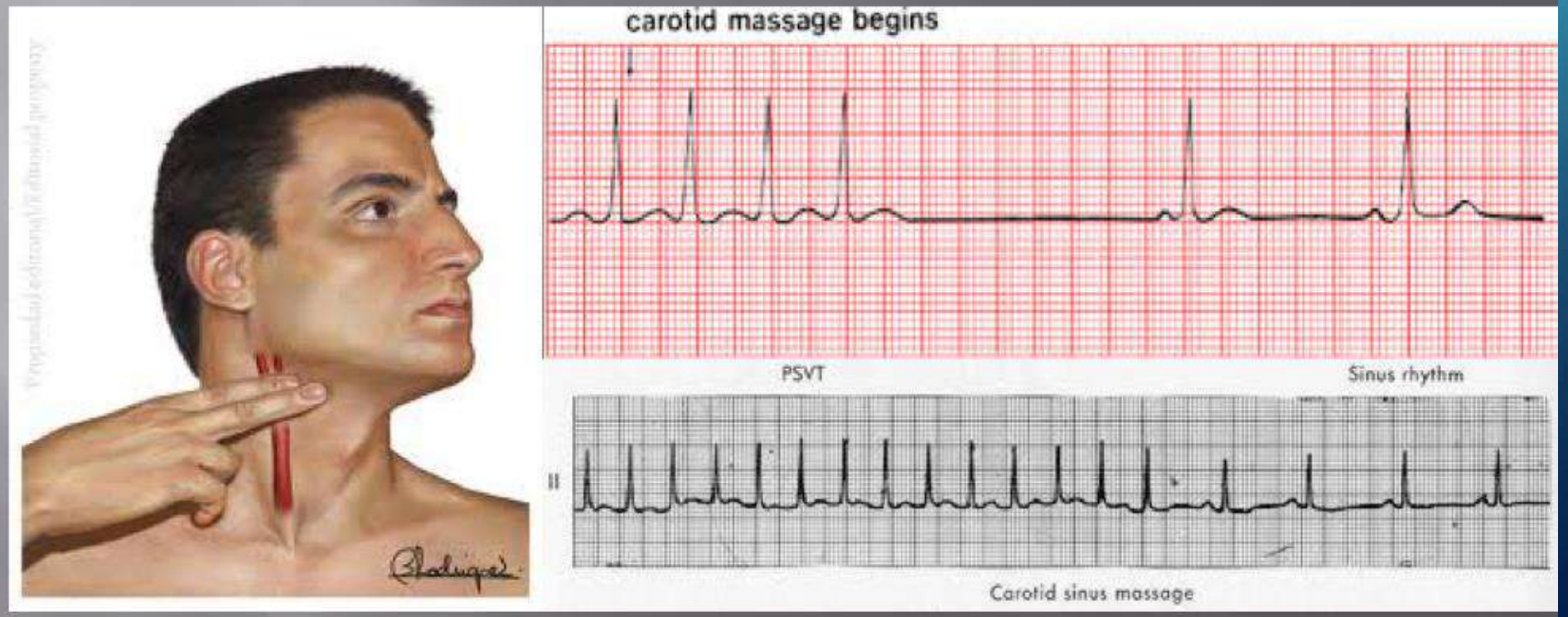
Rate	180 bpm
Rhythm	Atrioventricular nodal re-entry tachycardia
QRS axis	Normal (+22°)
P waves	Visible as a small negative deflection at the end of the QRS complex in the inferior leads
PR interval	Not applicable
QRS duration	Normal (60 ms)
T waves	Normal
QTc interval	Normal (450 ms)

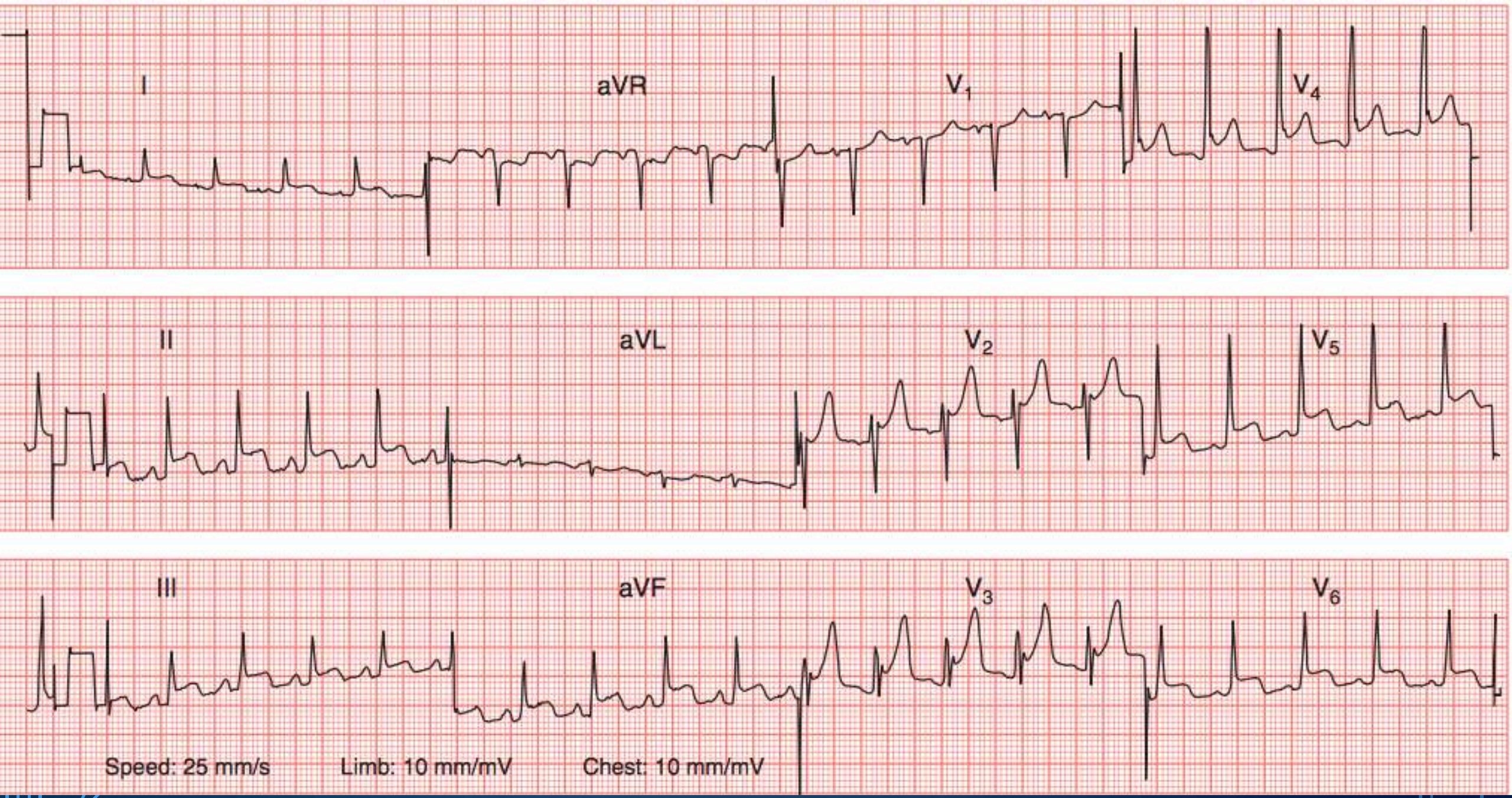
AVNRT

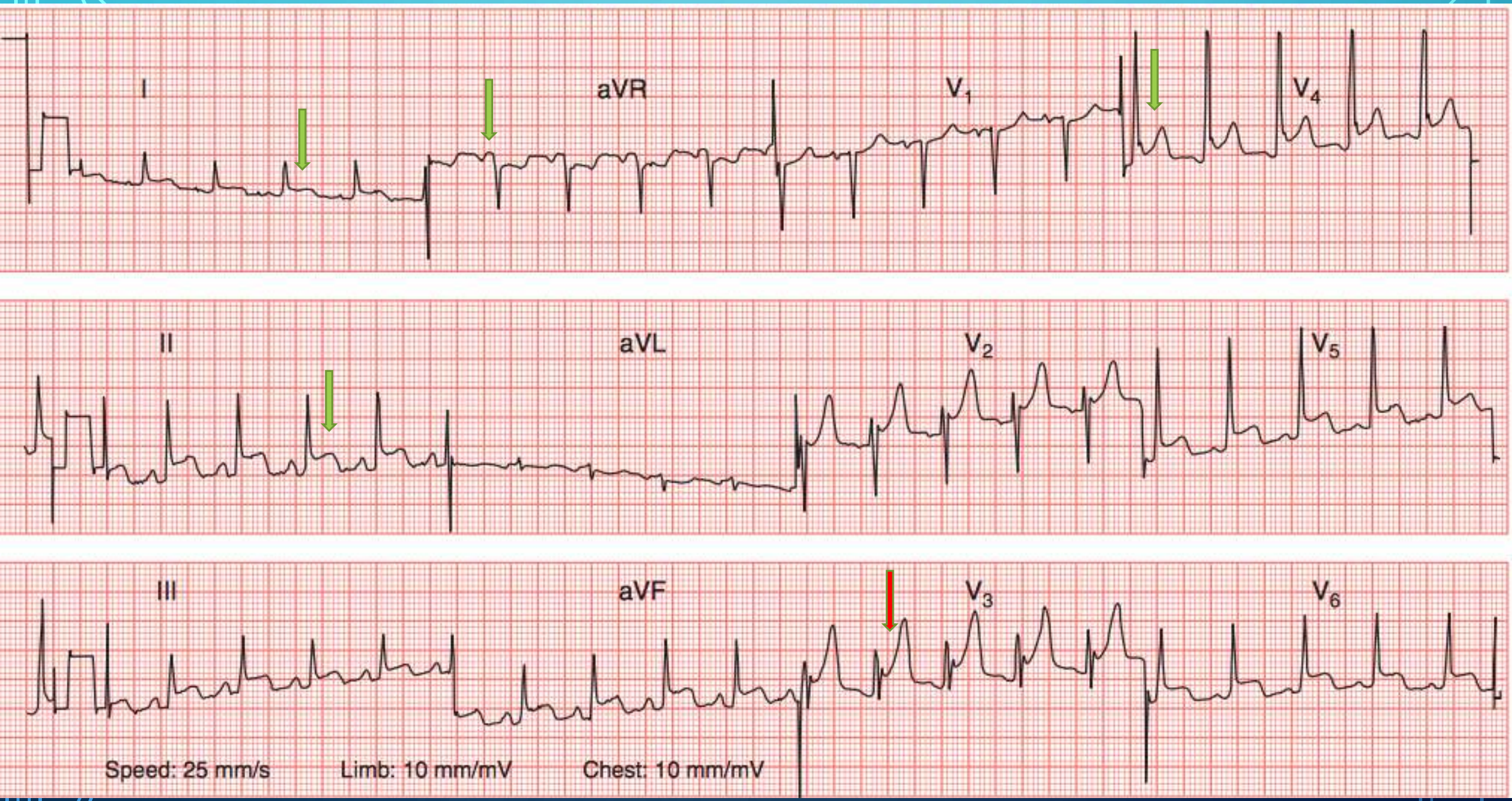
- HR:150-250
- شروع وختم ناگهانی
- Pseudo s inf- pseudo r v1
- اگرموج p دیده شود بسمت بالا وباریک
- شروعpac باPR طولانی
- Short RP<50%RR
- 10-50J Cardioversion

تحریک رفلکس واگ با ماساژ یا فشار روی سینوس کاروتید یا باعث کندکردن موقت یا قطع ناگهانی تاکیکاردی حمله ای میشود

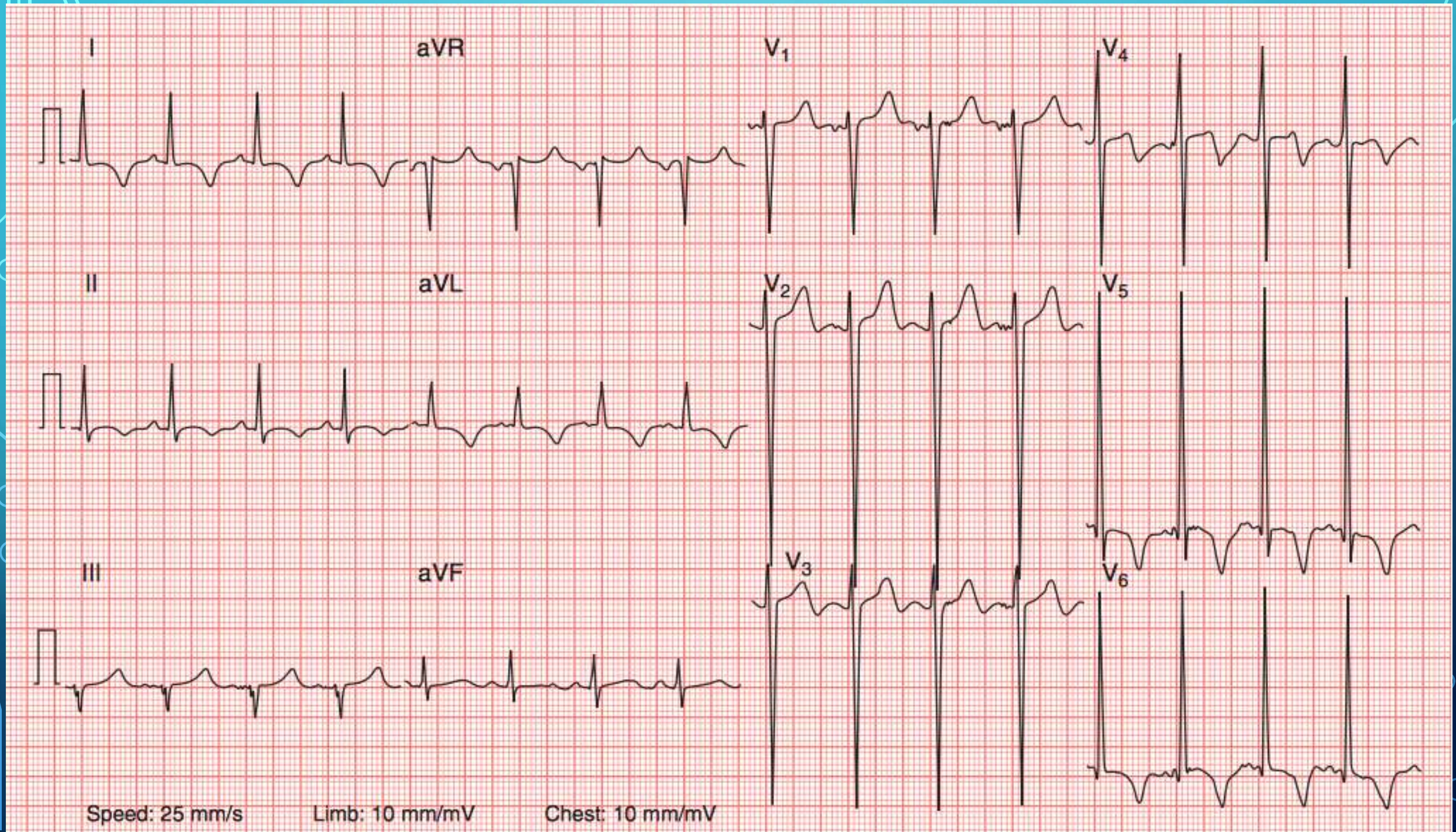
Carotid Massage

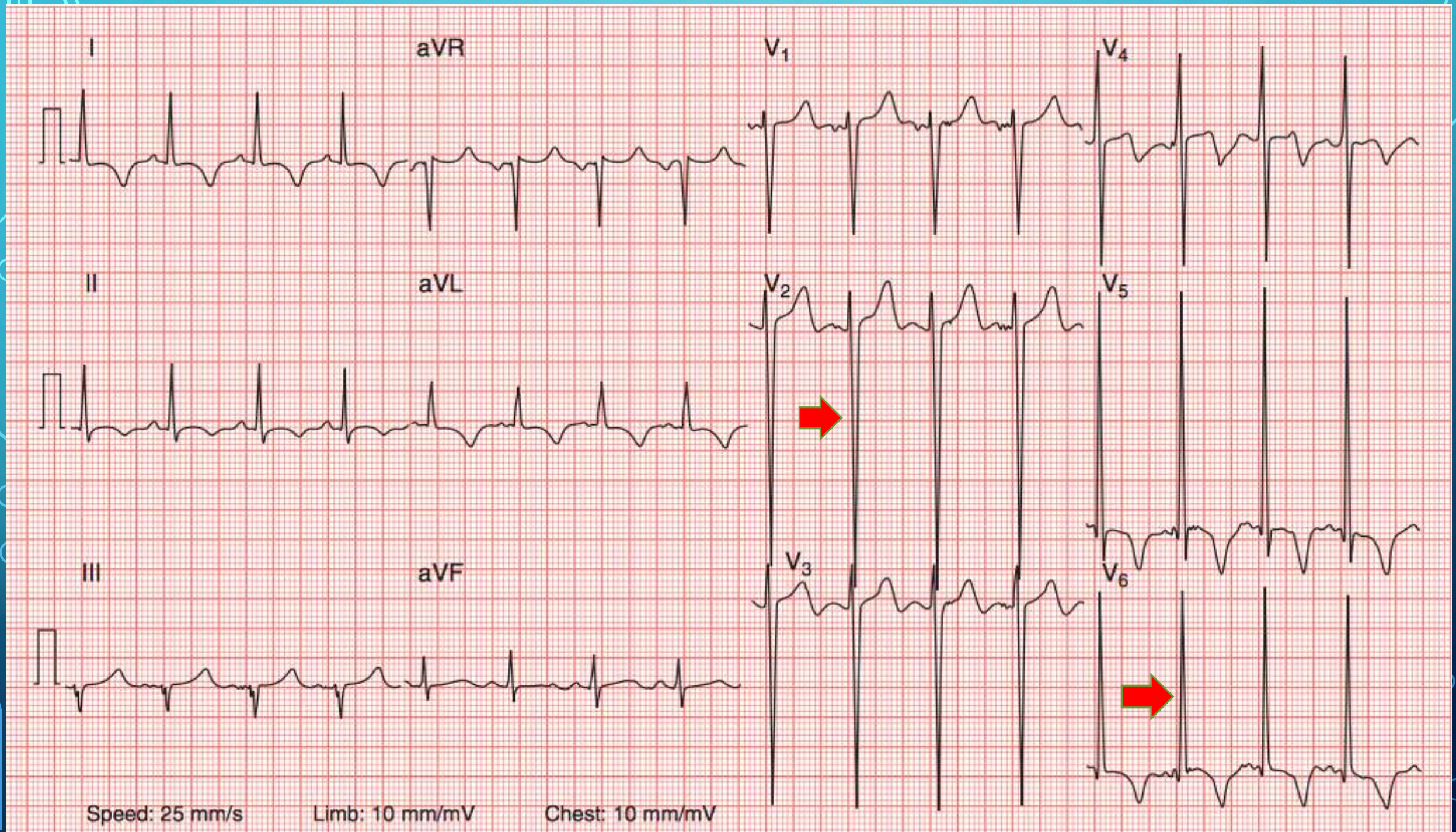



I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

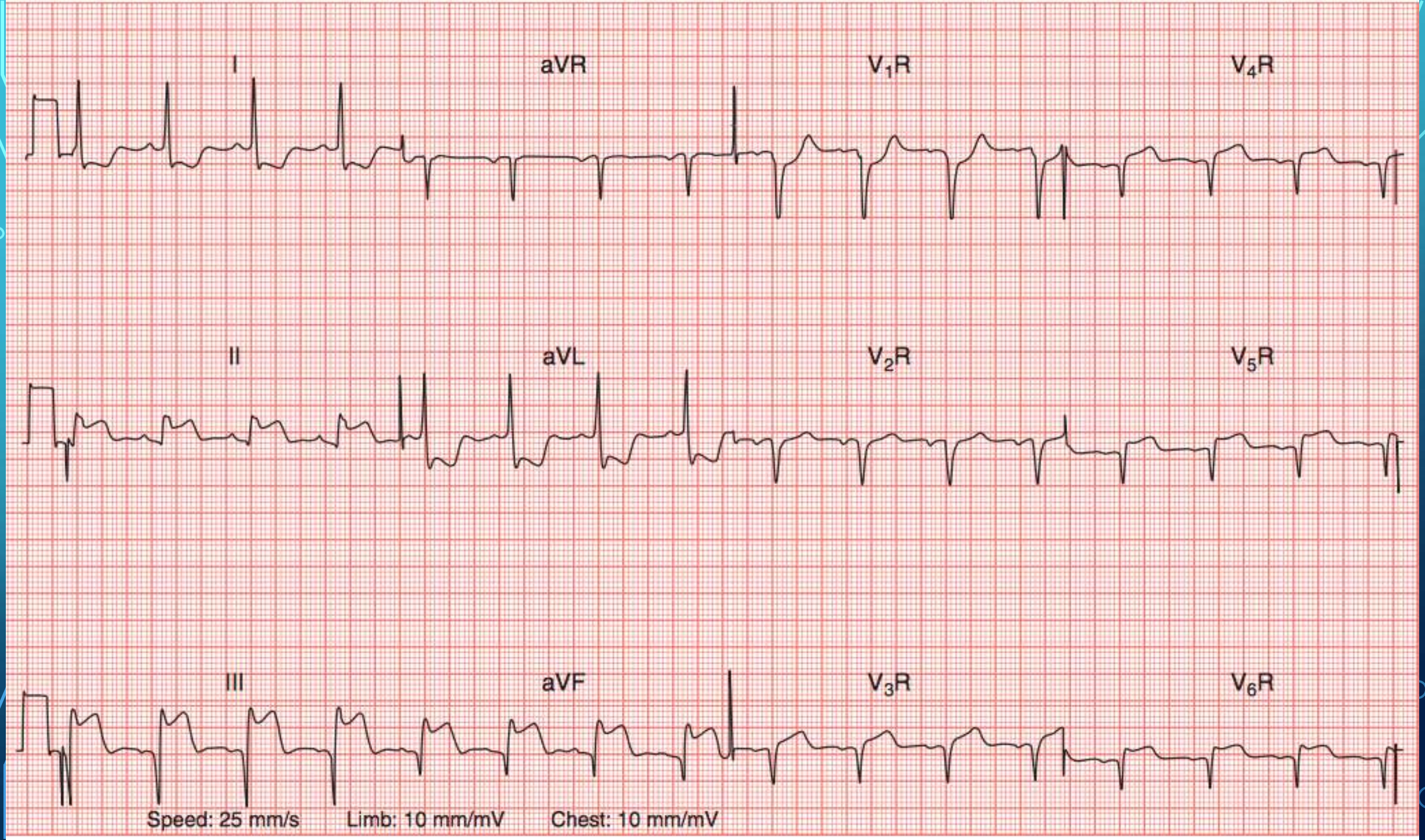

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

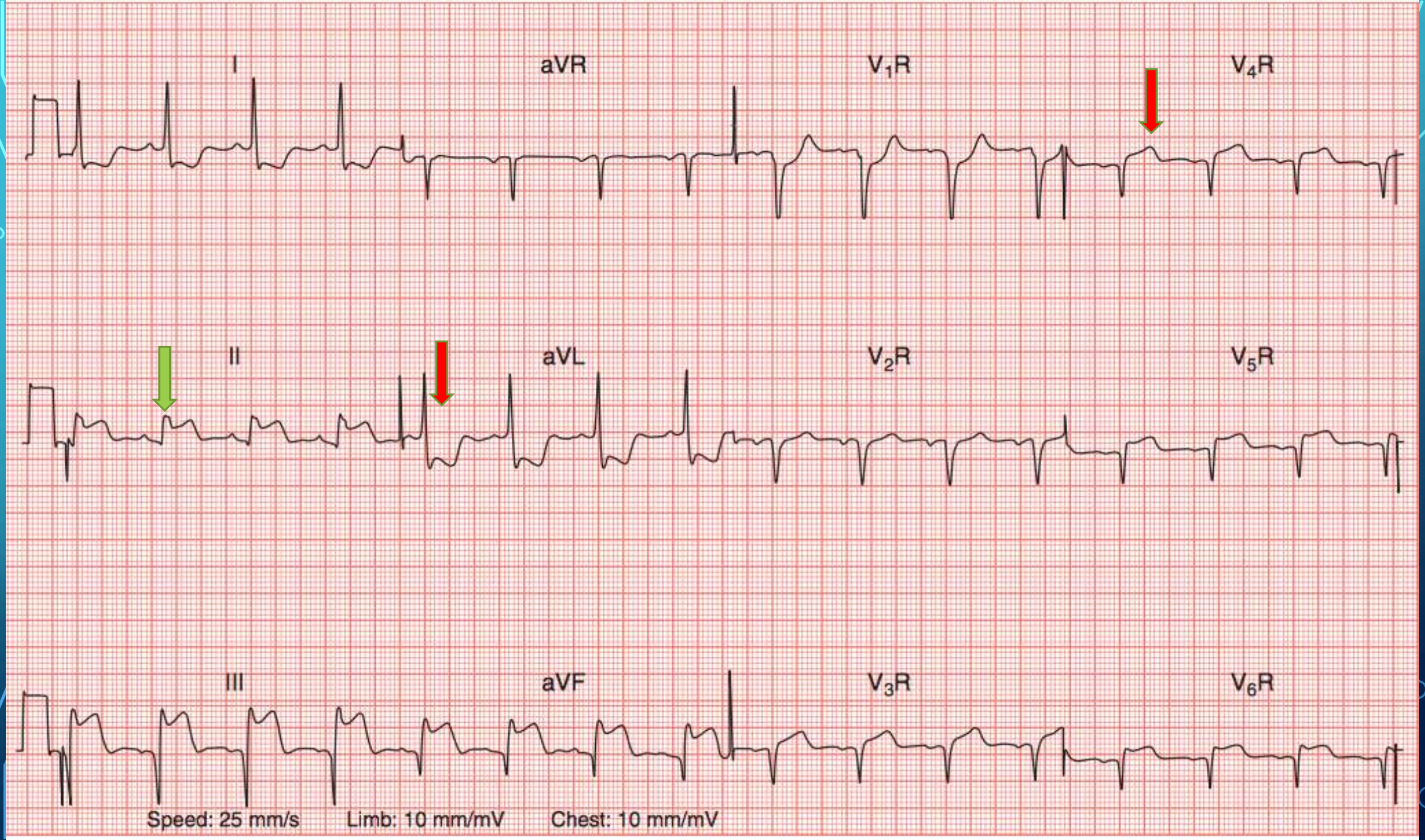
- This ECG shows widespread ST segment elevation (concave upward or ‘saddle-shaped’) in leads I, II, III, aVF and V2–V6, with reciprocal ST segment depression in lead aVR. In the clinical context, these findings are consistent with a diagnosis of pericarditis.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

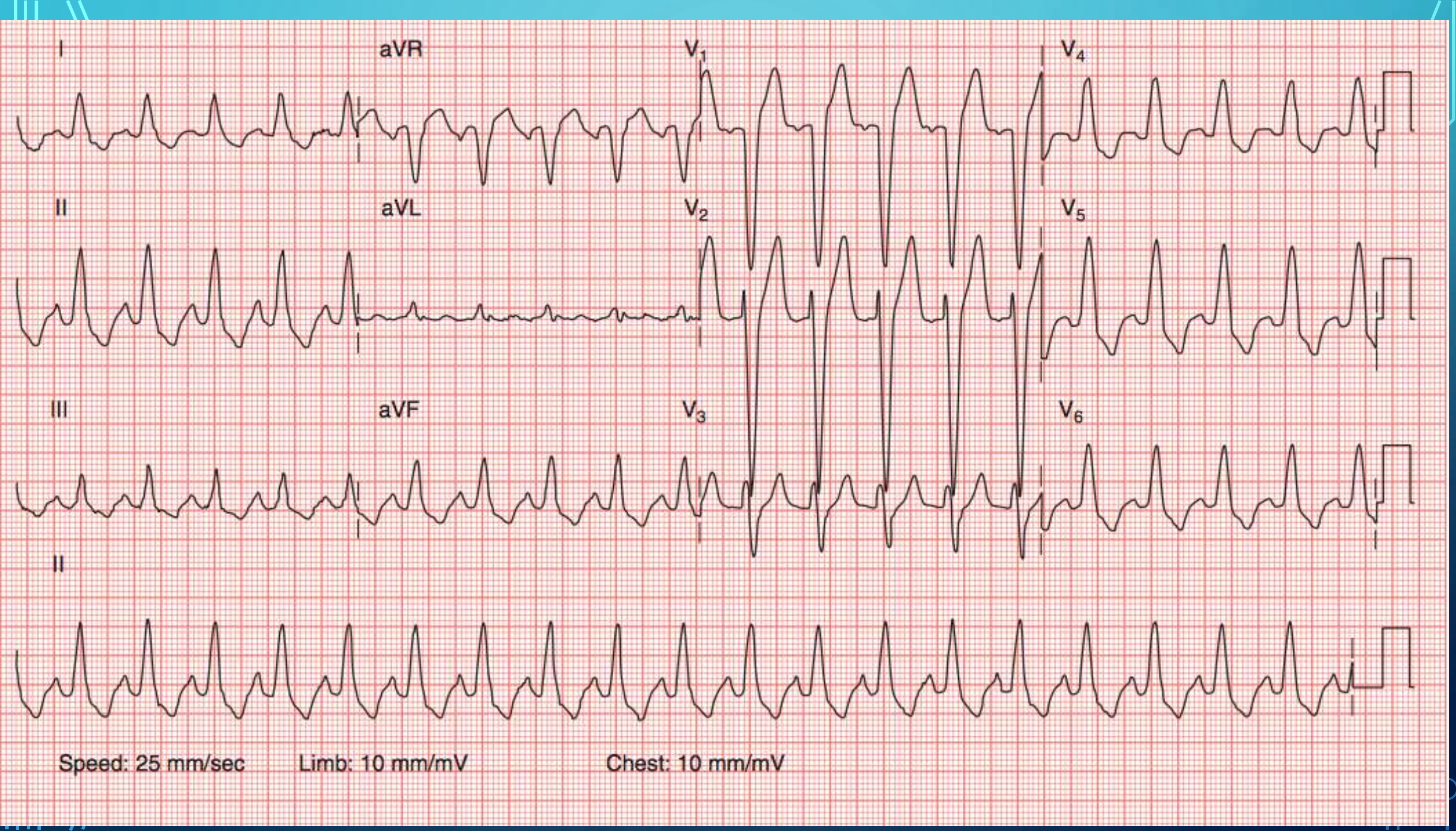

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

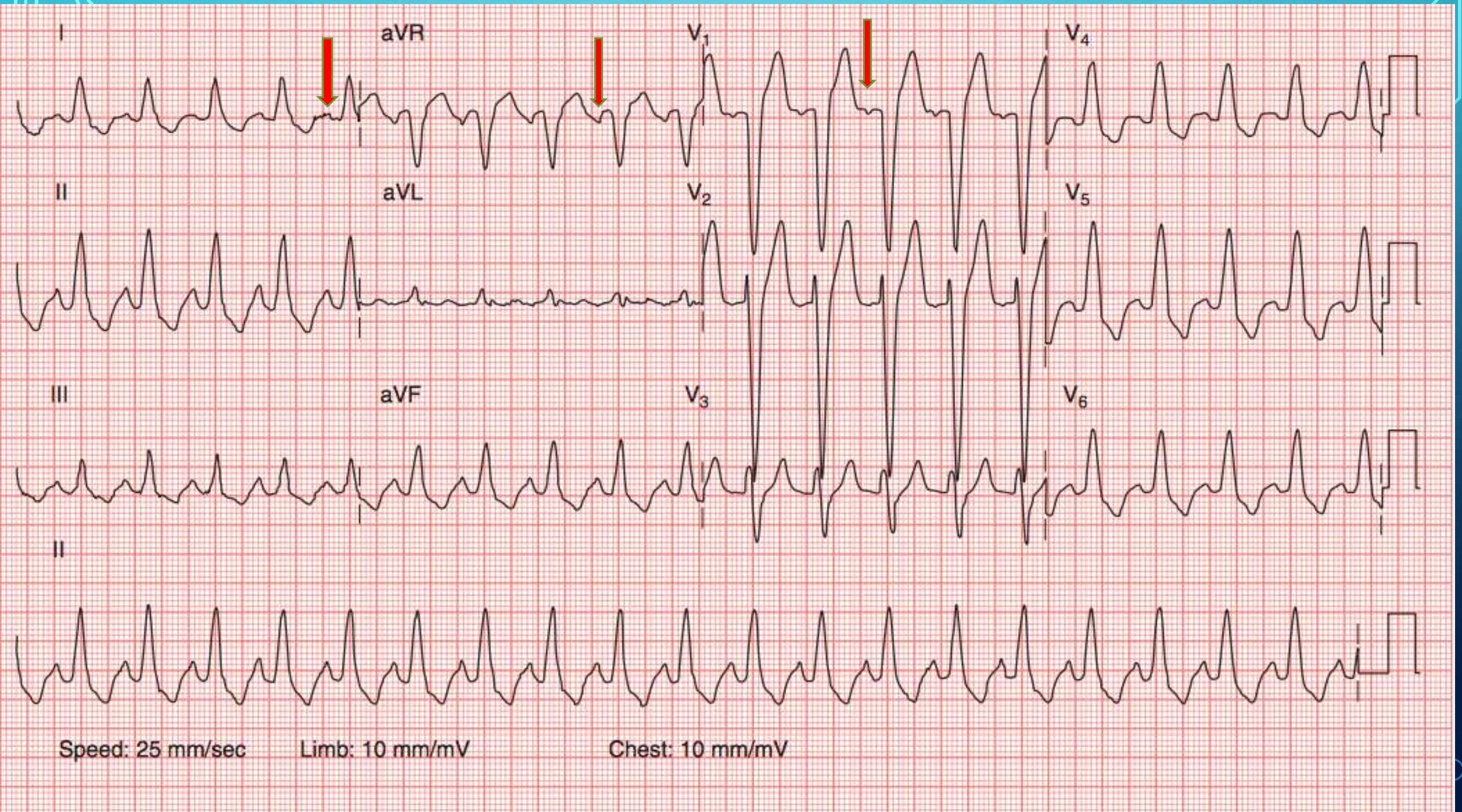
- This ECG shows very deep S waves (up to 48 mm) in leads V2–V3 and very tall R waves (up to 44 mm) in leads V5–V6, together with inverted T waves in leads I, aVL, V4–V6 (and also in lead II). These appearances are indicative of left ventricular hypertrophy with ‘strain’
- S V1+R V5>3.5mV,R aVL>1.1mV


I
aVR
V1R
V4R
II
aVL
V2R
V5R
III
aVF
V3R
V6R
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

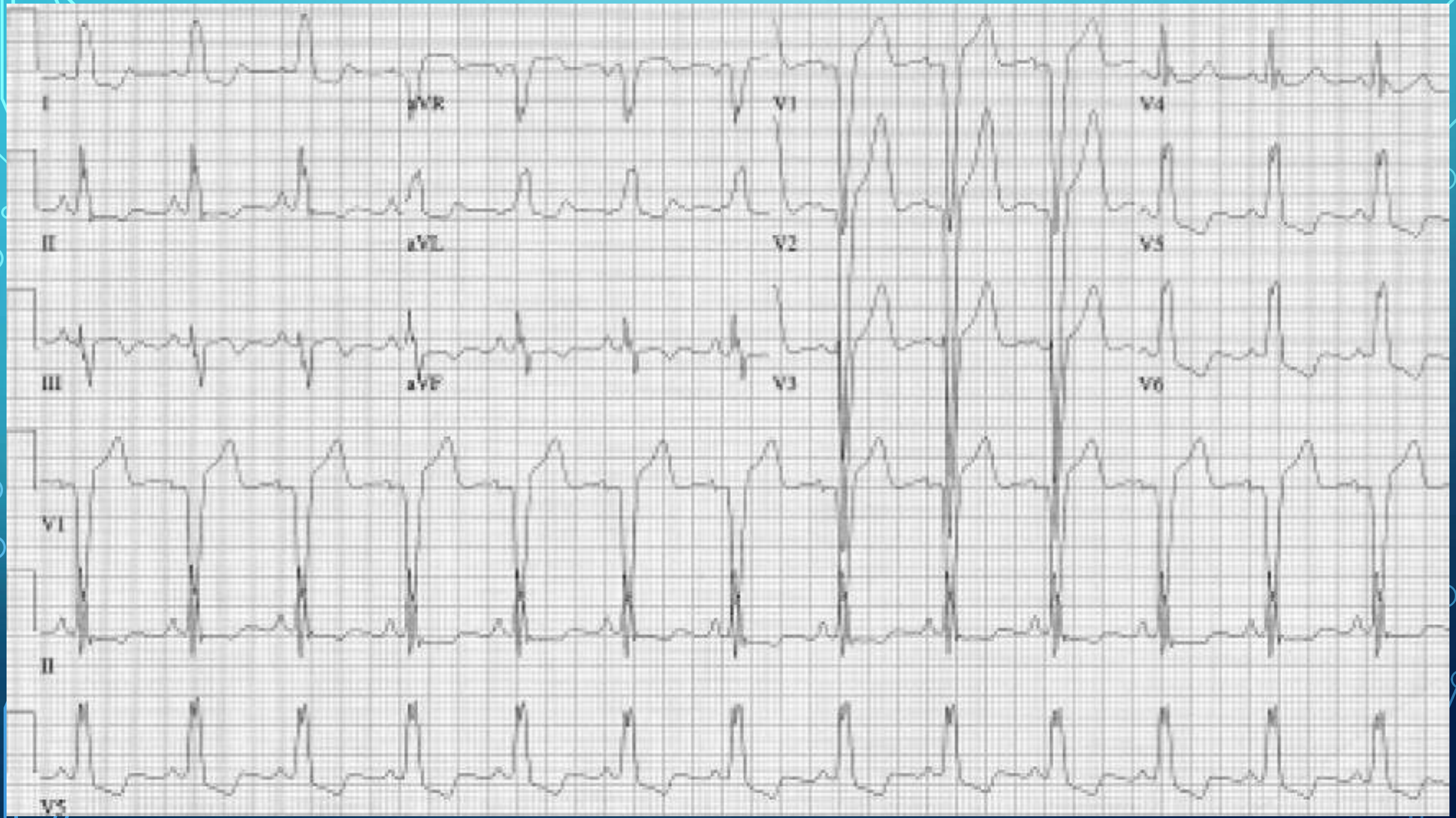

I
aVR
V1R
V4R
II
aVL
V2R
V5R
III
aVF
V3R
V6R
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

- The ECG shows an acute inferior STEMI (ST segment elevation in leads II, III, aVF) with reciprocal ST segment depression laterally (leads I and aVL). There is ST segment elevation in leads V3R–V6R. The presence of ST segment elevation in lead V4R is indicative of right ventricular involvemen


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10 mm/mV

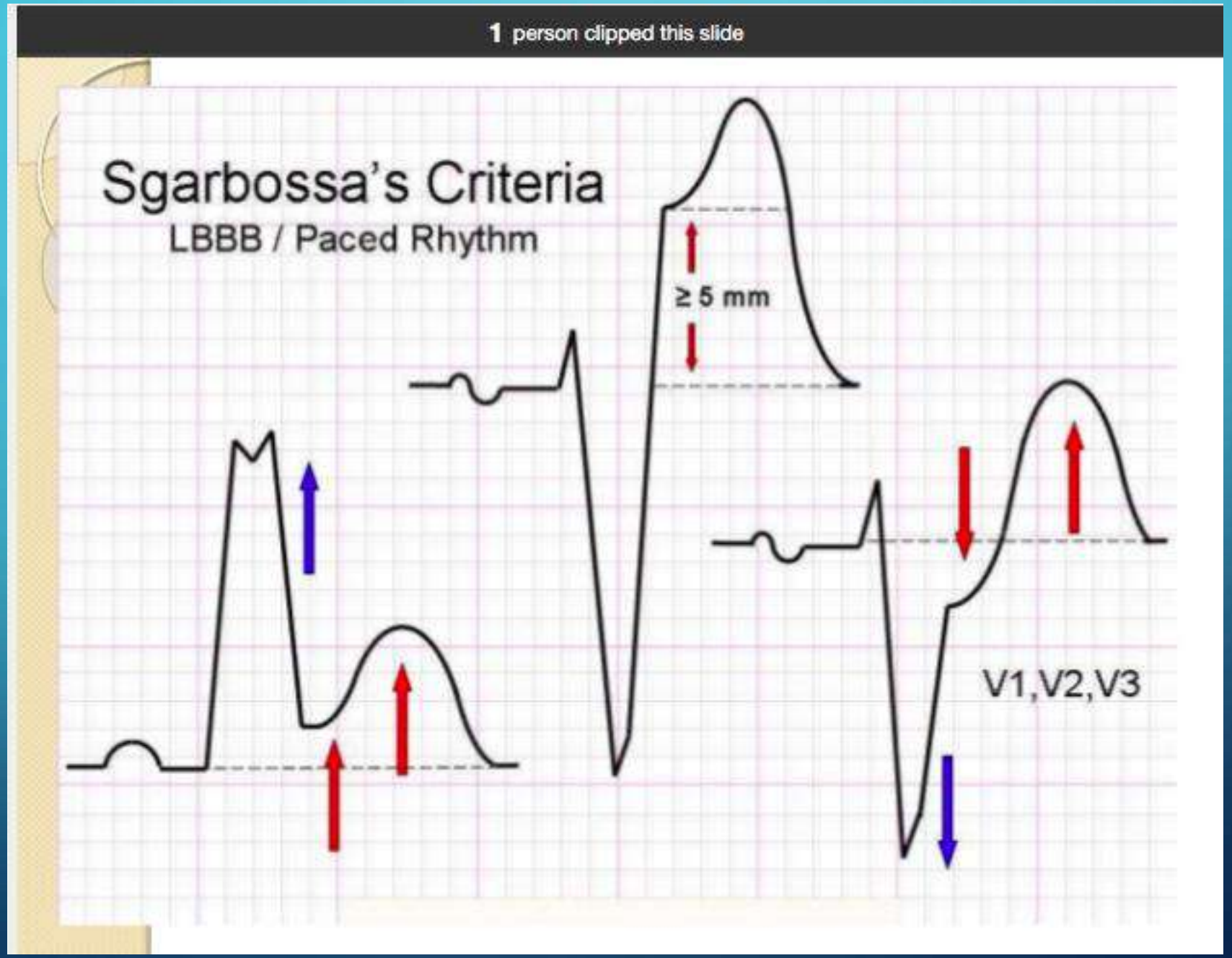

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10 mm/mV

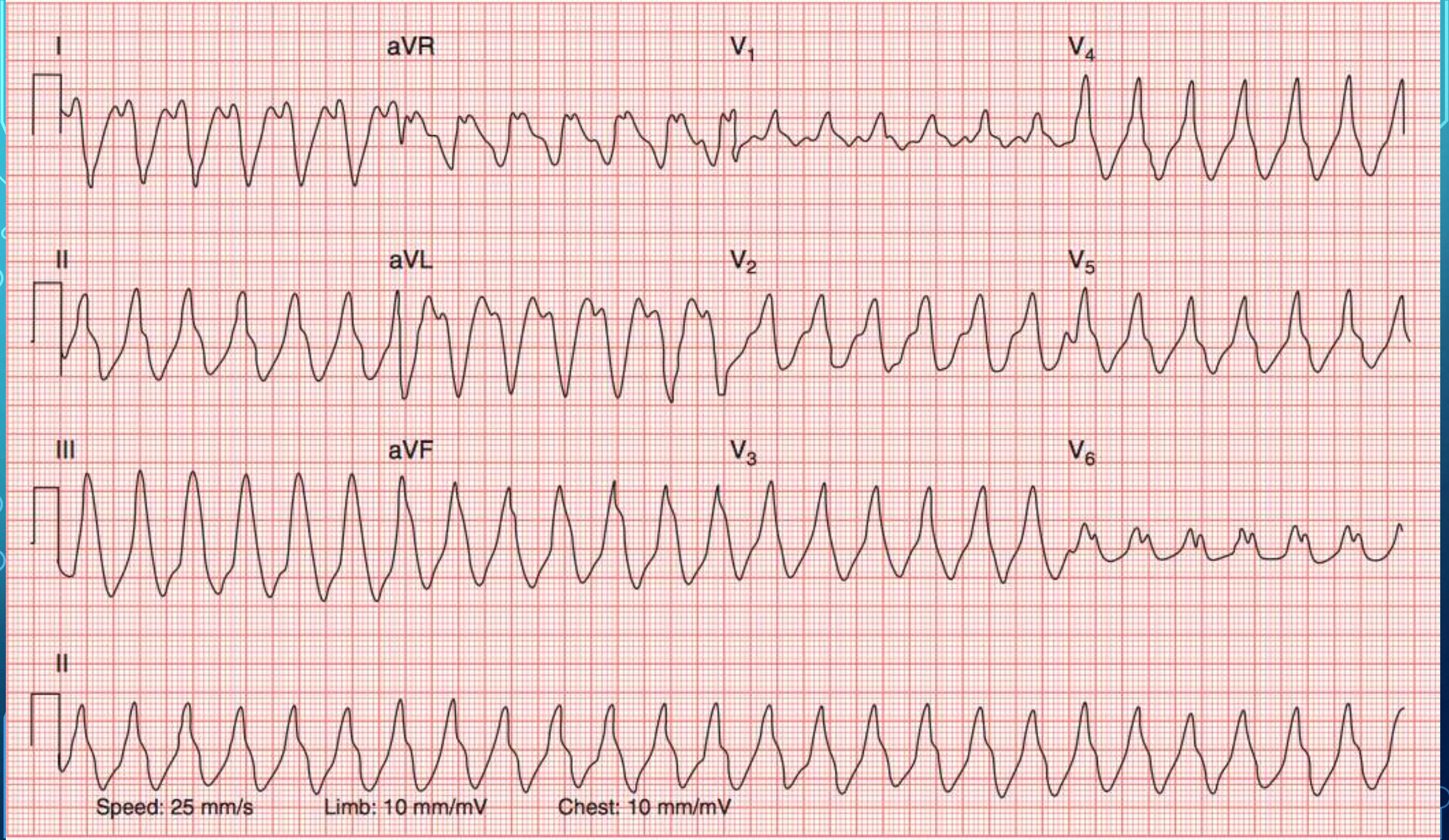
- This ECG shows a tachycardia (heart rate 120bpm) with broad QRS complexes (QRS duration 130 ms). The QRS complexes have a left bundle branch block (LBBB) morphology. On careful inspection, P waves can be seen before the QRS complexes – the P waves are most easily seen in lead V1. This broad-complex tachycardia is therefore sinus tachycardia with aberrant conduction (LBBB).

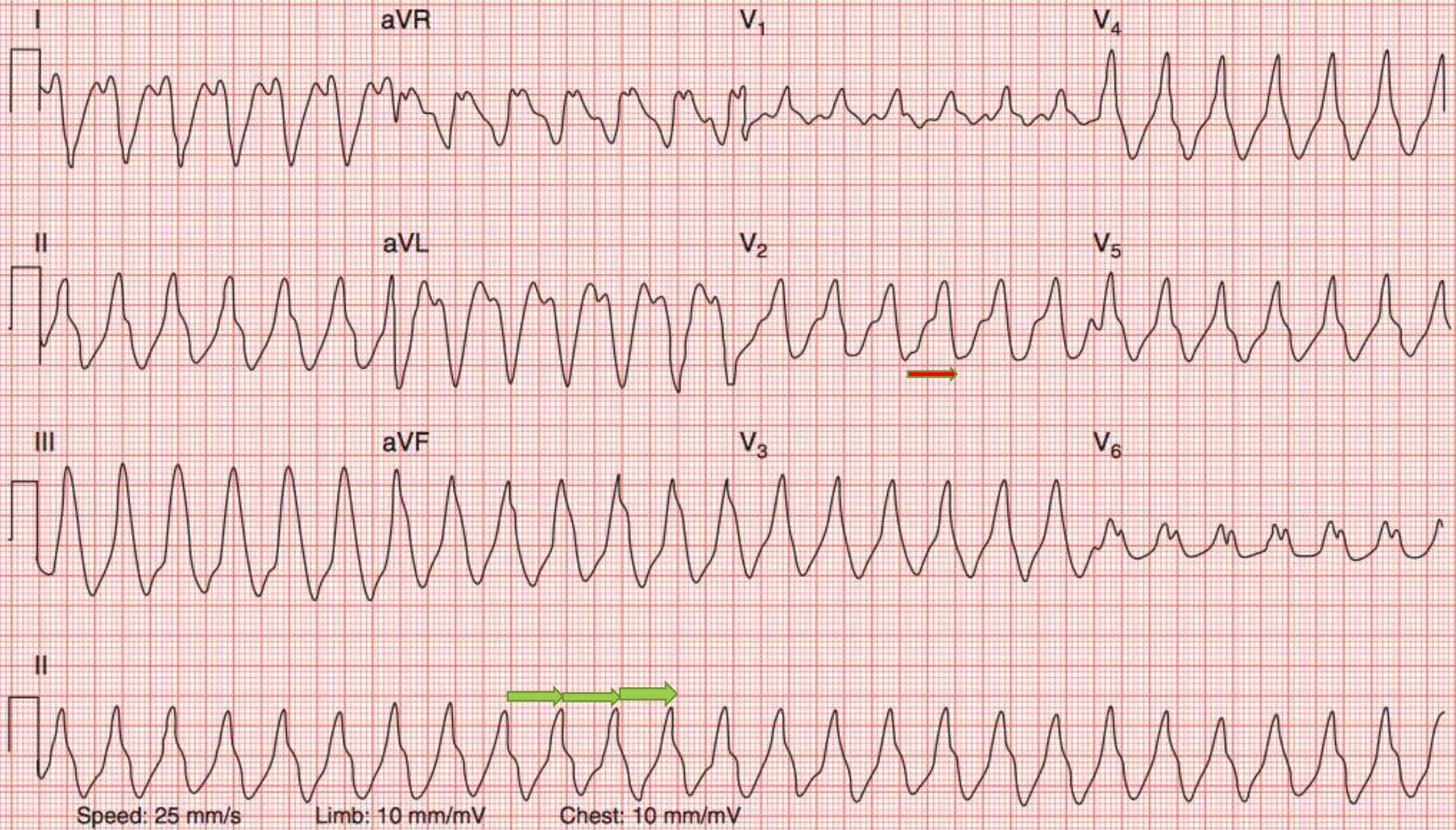

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

MI WITH LBBB

New onset of LBBB suggests acute MI.
In patients with documented LBBB earlier,it is difficult to diagnose AWMI due to masking effect of LBBB on QRST changes.
CRITERIA USED FOR ACUTE AWMI WITH PRIOR LBBB IS SGARBOSSA CRITERIA
1.ST elevation in atleast one lead of >1mm concordant to positive QRS complex[5]
2.ST depression of >1mm in V1 to V3[3]
3.Discordant ST elevation >5mm in atleast one leads with prominant negative QRS[2]
A total of >= 3 points suggests


1 person clipped this slide
Sgarbossa's Criteria
LBBB / Paced Rhythm
≥ 5 mm
V1,V2,V3


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

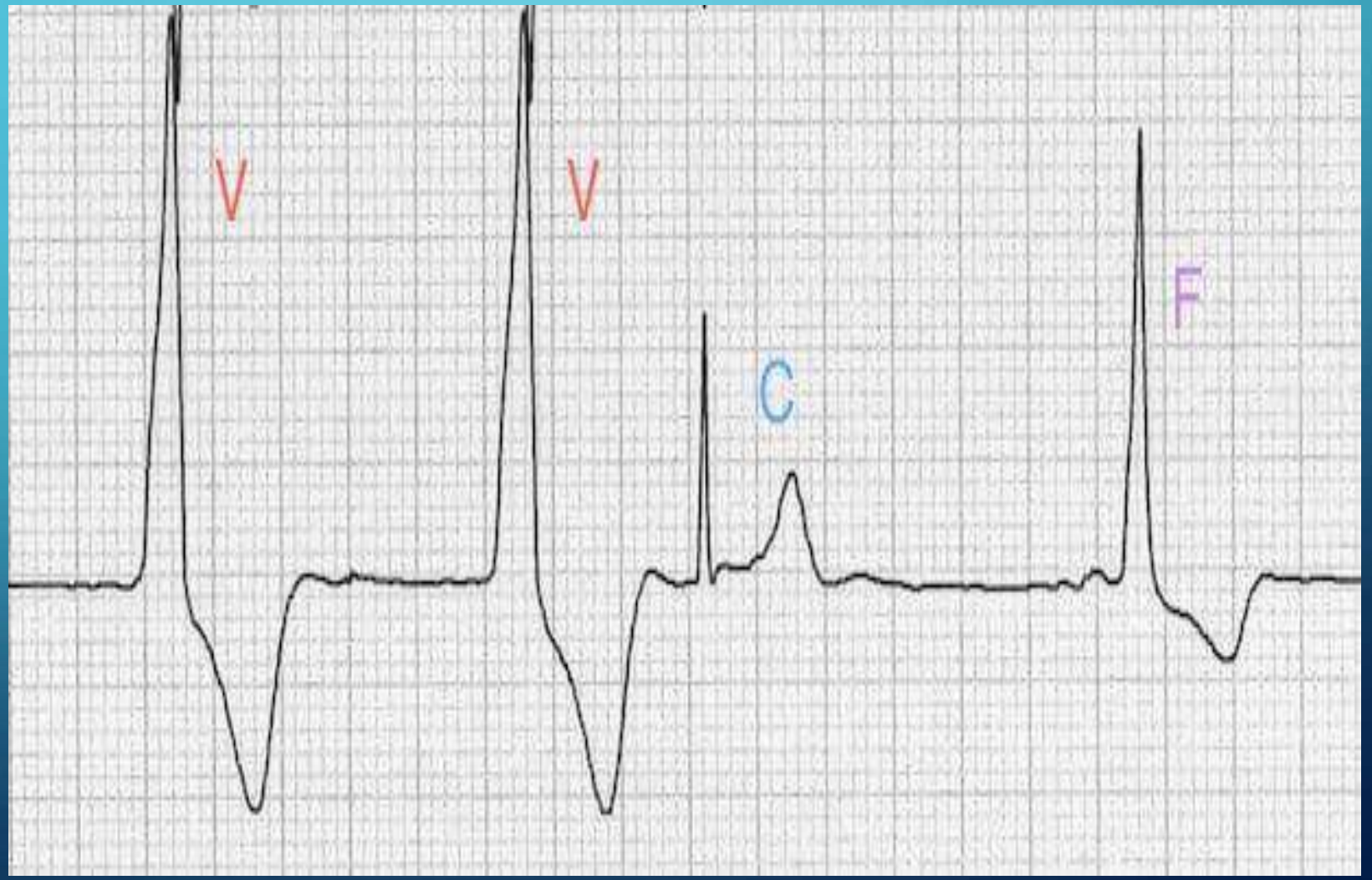

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

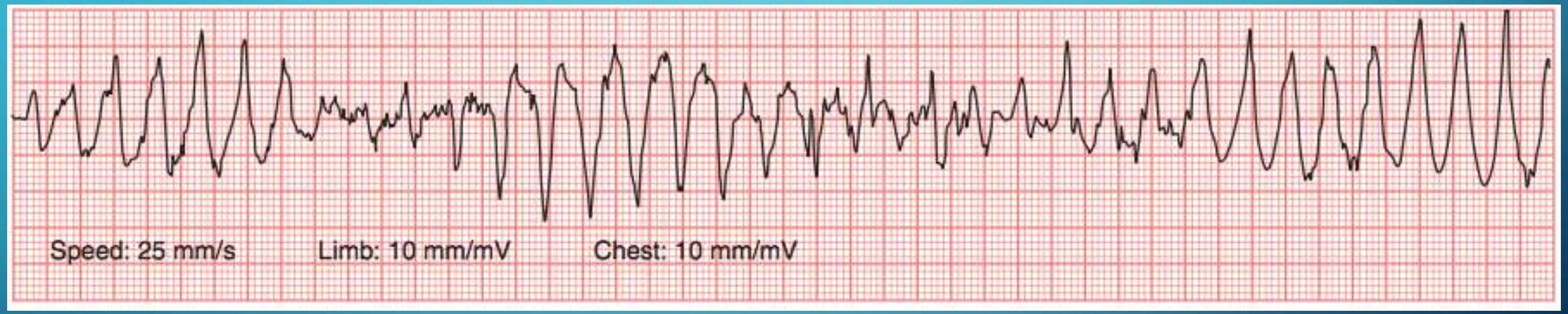
- Wide QRS tachycardia
- There is positive concordance of the anterior chest leads (the QRS complexes in the anterior leads are all positive). This is ventricular tachycardia (VT).

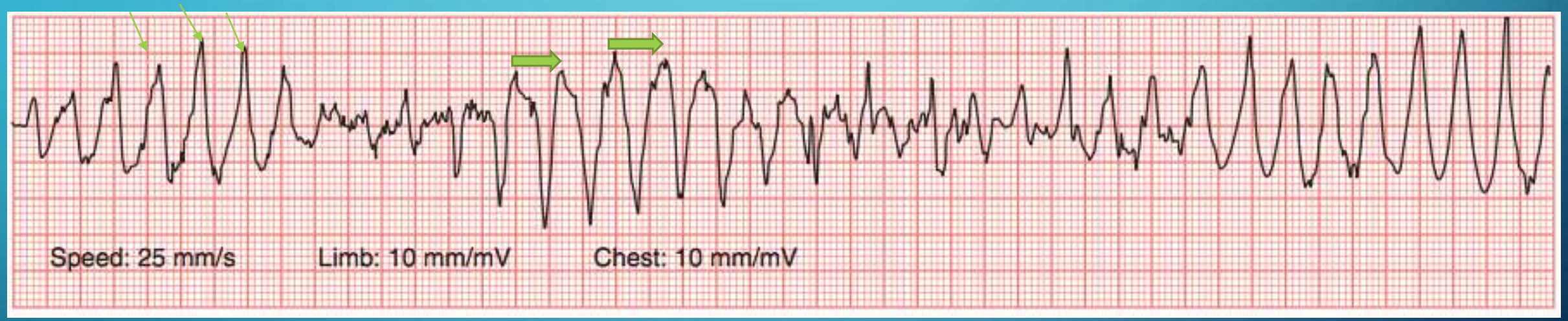
VT

- AV dissociation
- Fusion beat:like VT but narrower
- Capture beat:reverse VT
- QRS>140
- R>R’ V1,QS V6
- Concordance
- R+ AVR

Capture beat:reverse VT
FUSION:LIKE VT

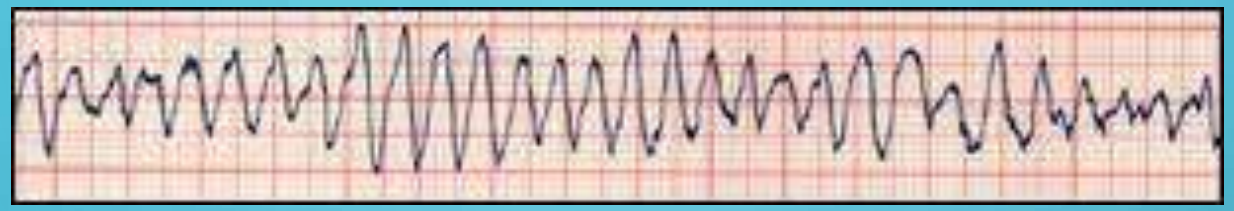



Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

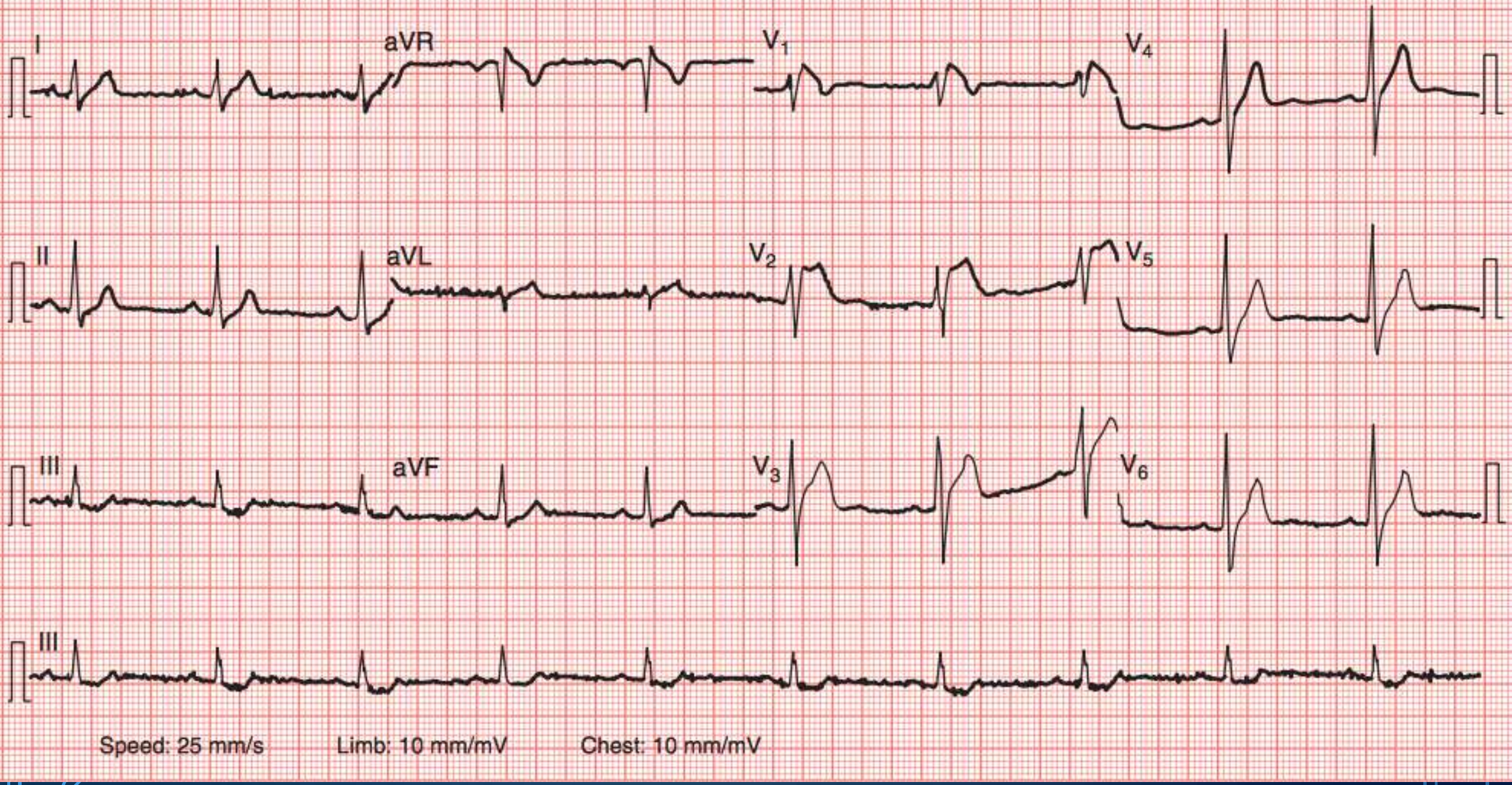

Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

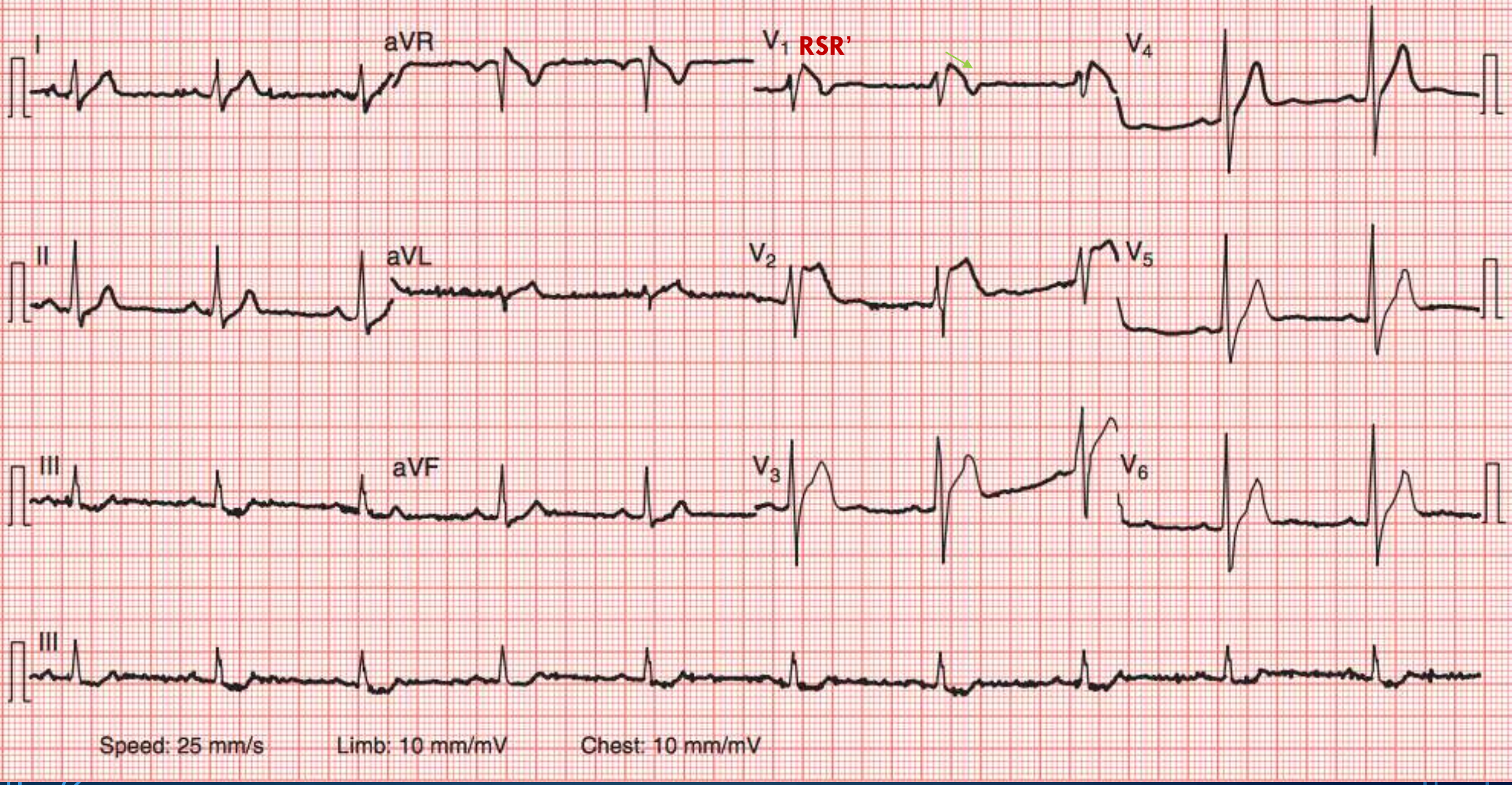
- Polymorphic VT has a number of recognized causes (see Commentary) which prolong the QT interval and predispose to polymorphic VT. In this patient's case the likely aetiology is the patient's electrolyte abnormalities (hypokalaemia and hypomagnesaemia).

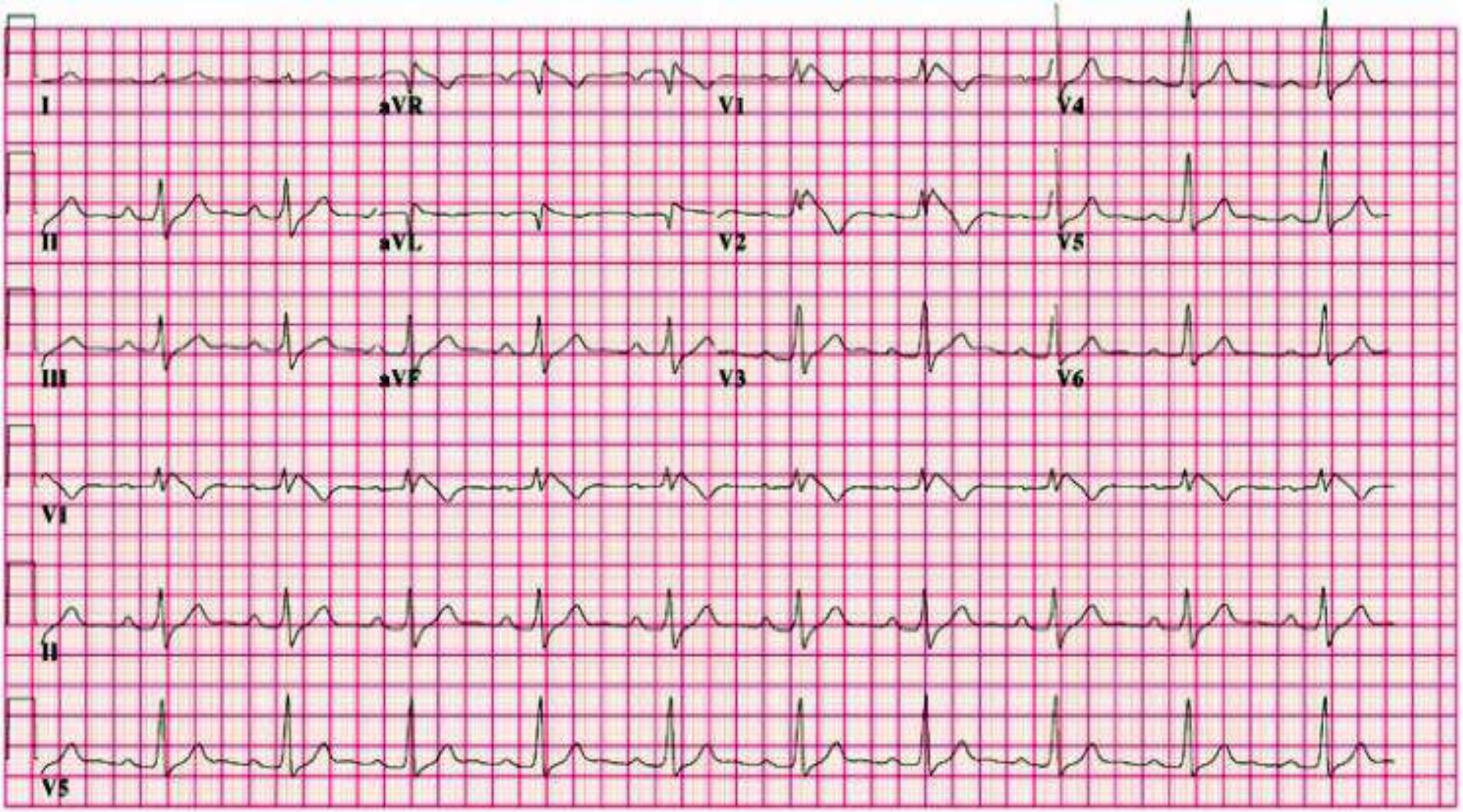
TORSADES DE POINTES



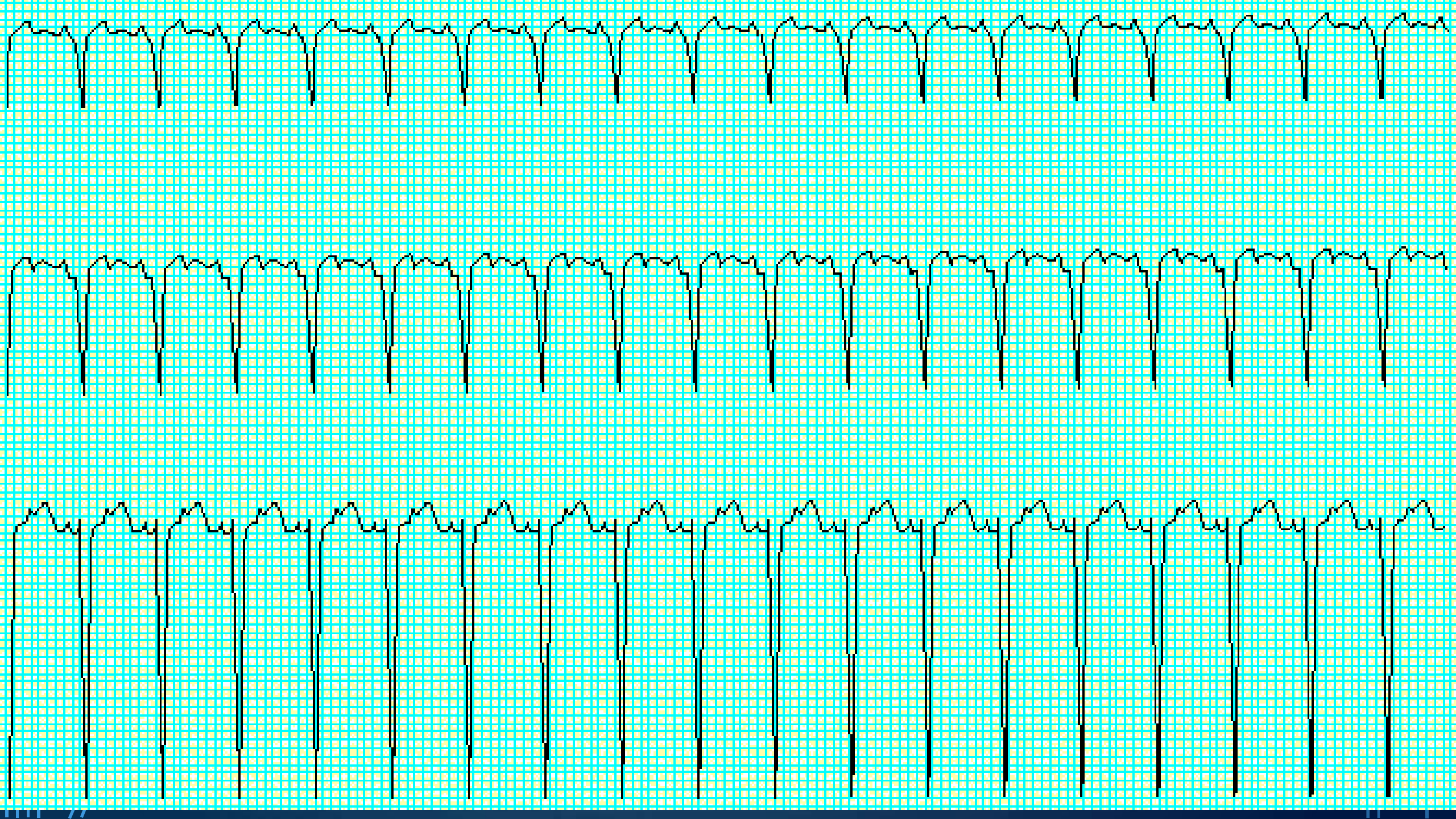
- "Twisting of the points" is usually caused by medication (quinidine, disopyramide, sotalol, TCA), hypokalemia or bradycardia especially after MI
- HR=200-250
- Has prolonged QT interval
- Acute: Remove offending medication. Shorten the QT interval with **magnesium, lidocaine,phenytion isoproterenol, or temporary overdrive pacing(1B)**
- amiodarone, beta-blockers

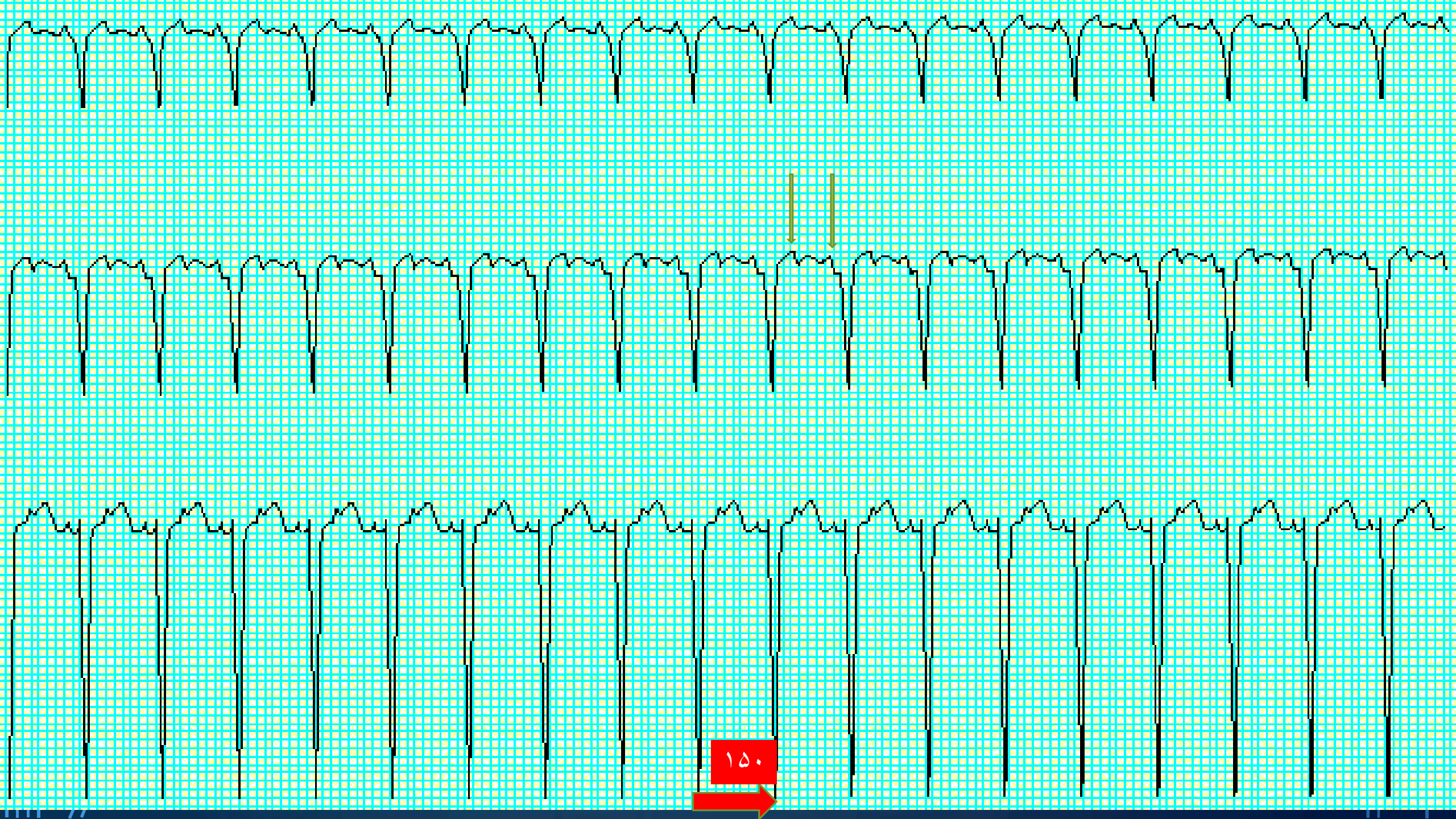

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
III
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV


I
aVR
V1
RSR'
V4
II
aVL
V2
V5
III
aVF
V3
V6
III
Speed: 25 mm/s
Limb: 10 mm/mV
Chest: 10 mm/mV

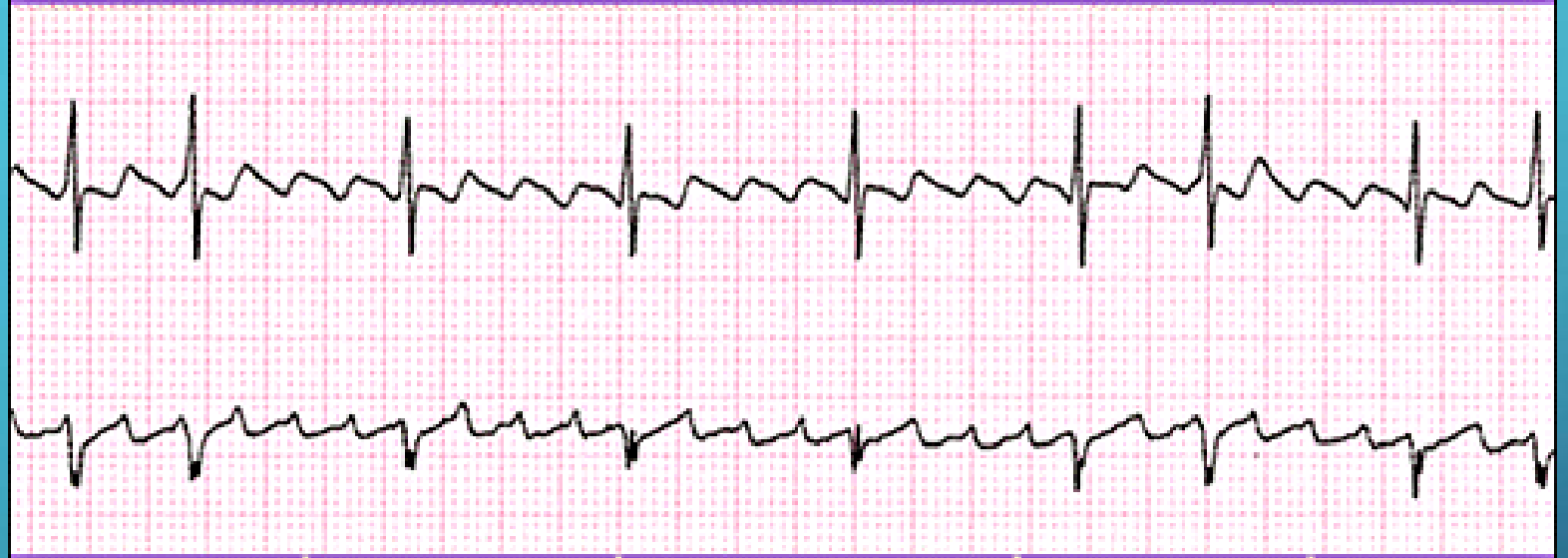

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
VI
II
V5

- There is ST segment elevation in the right chest leads (V1–V3) and a right bundle branch block (RBBB) morphology – this combination of ECG signs is suggestive of Brugada syndrome.
- 2 In patients with a structurally normal heart but with the ECG characteristics shown above, Brugada syndrome is associated with syncopal or sudden death episodes. Collapse may be due to fast, polymorphic ventricular tachycardia or ventricular fibrillation, usually occurring without warning.



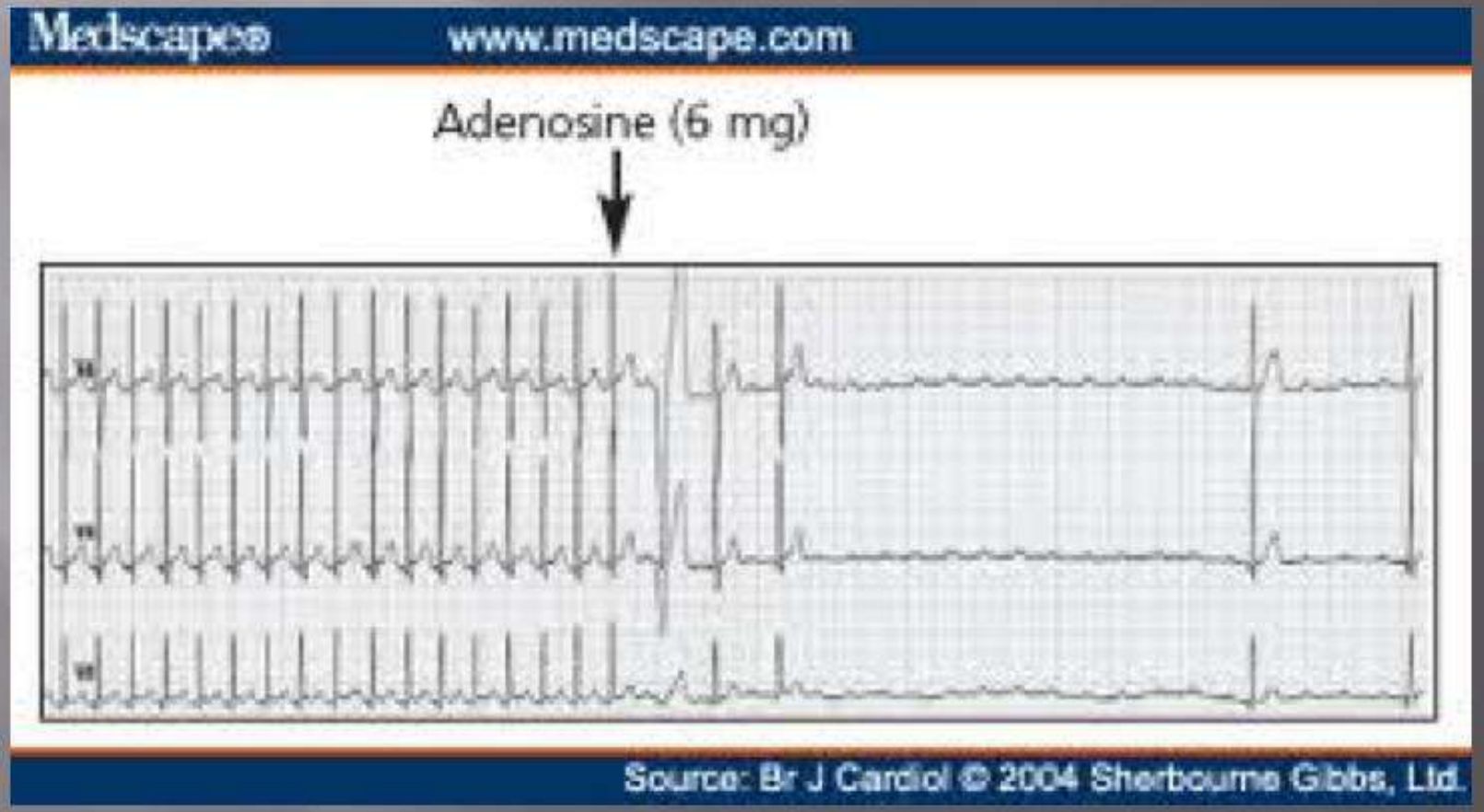

١٥٠

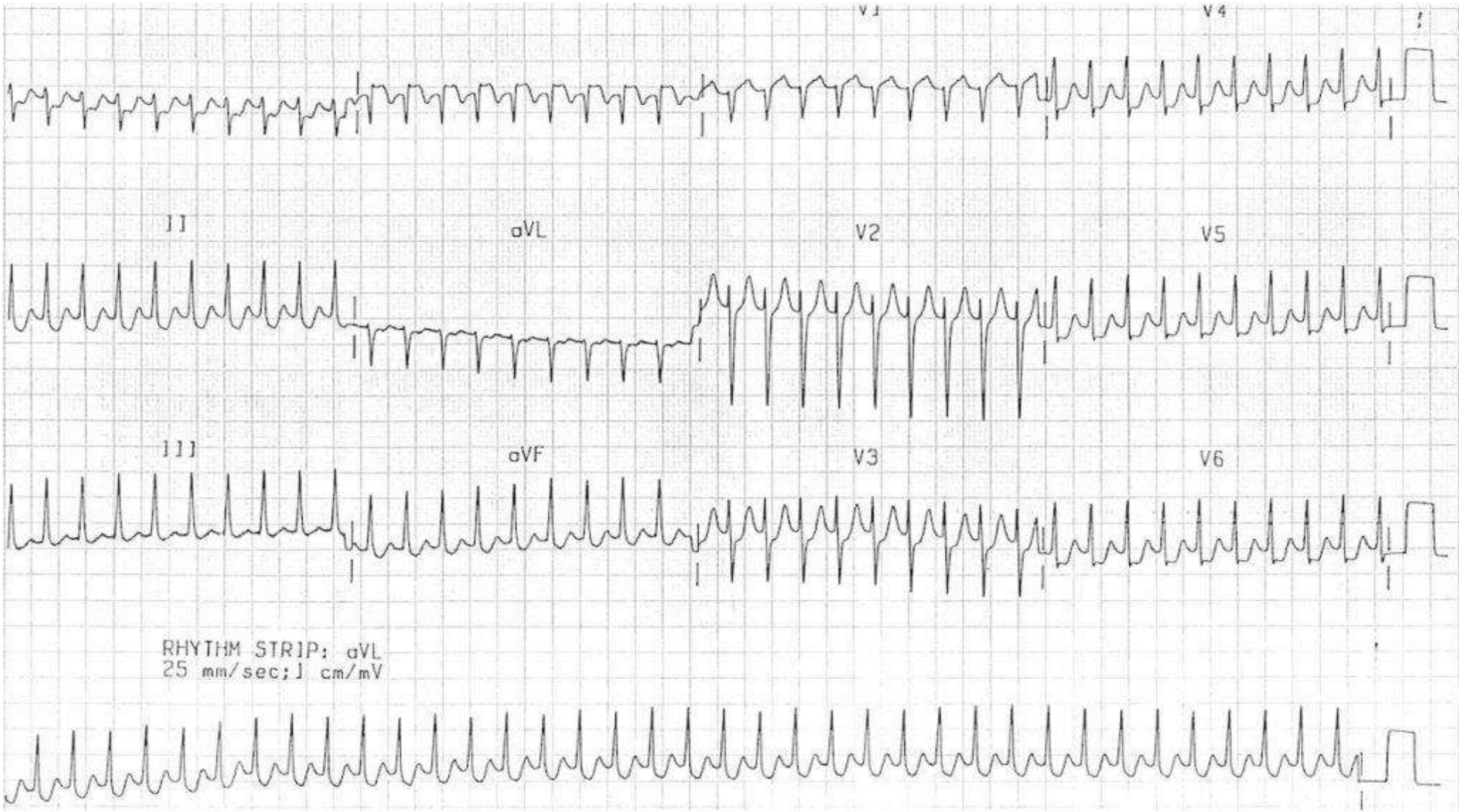
Atrial Flutter

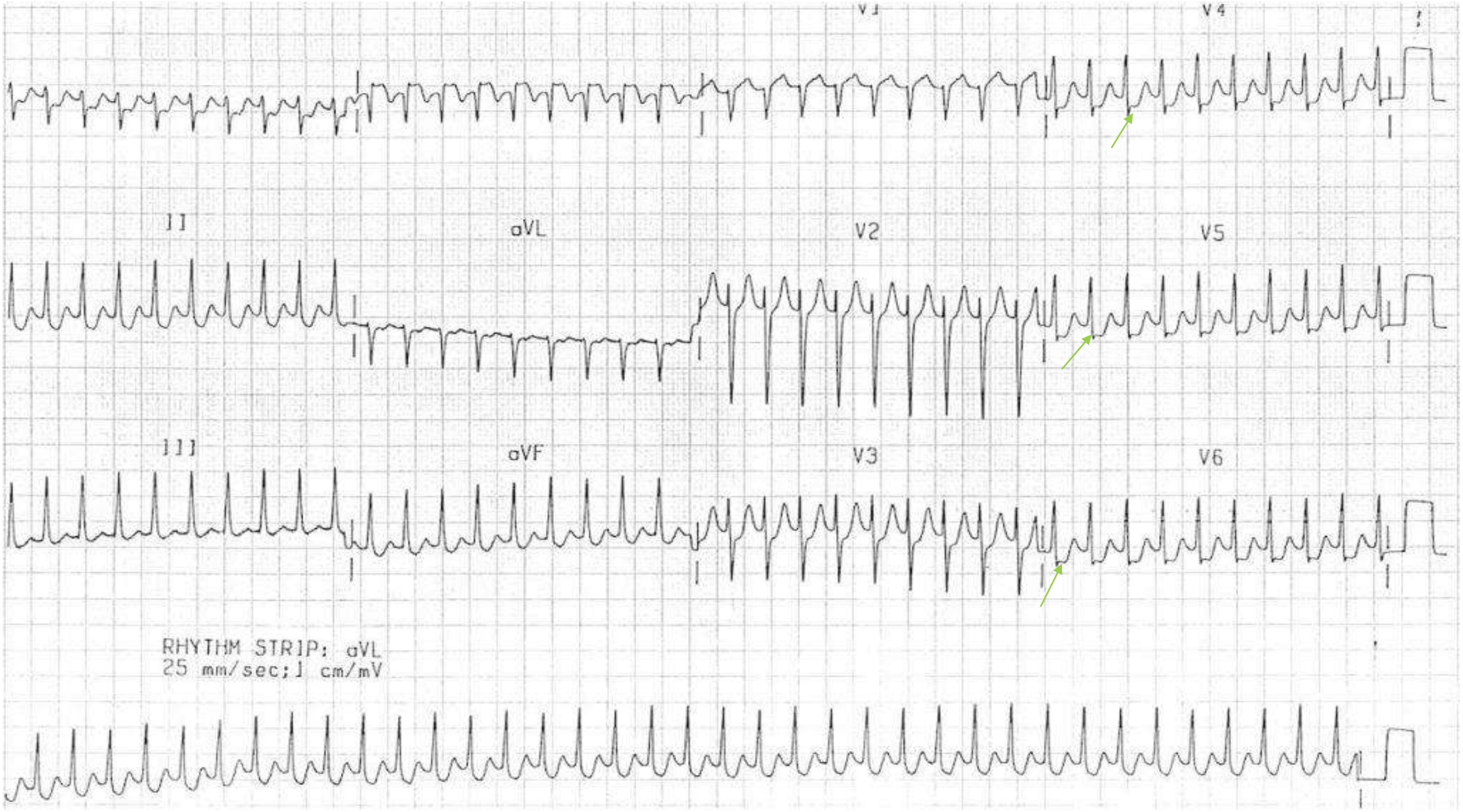


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12

Adenosin effect on AFL

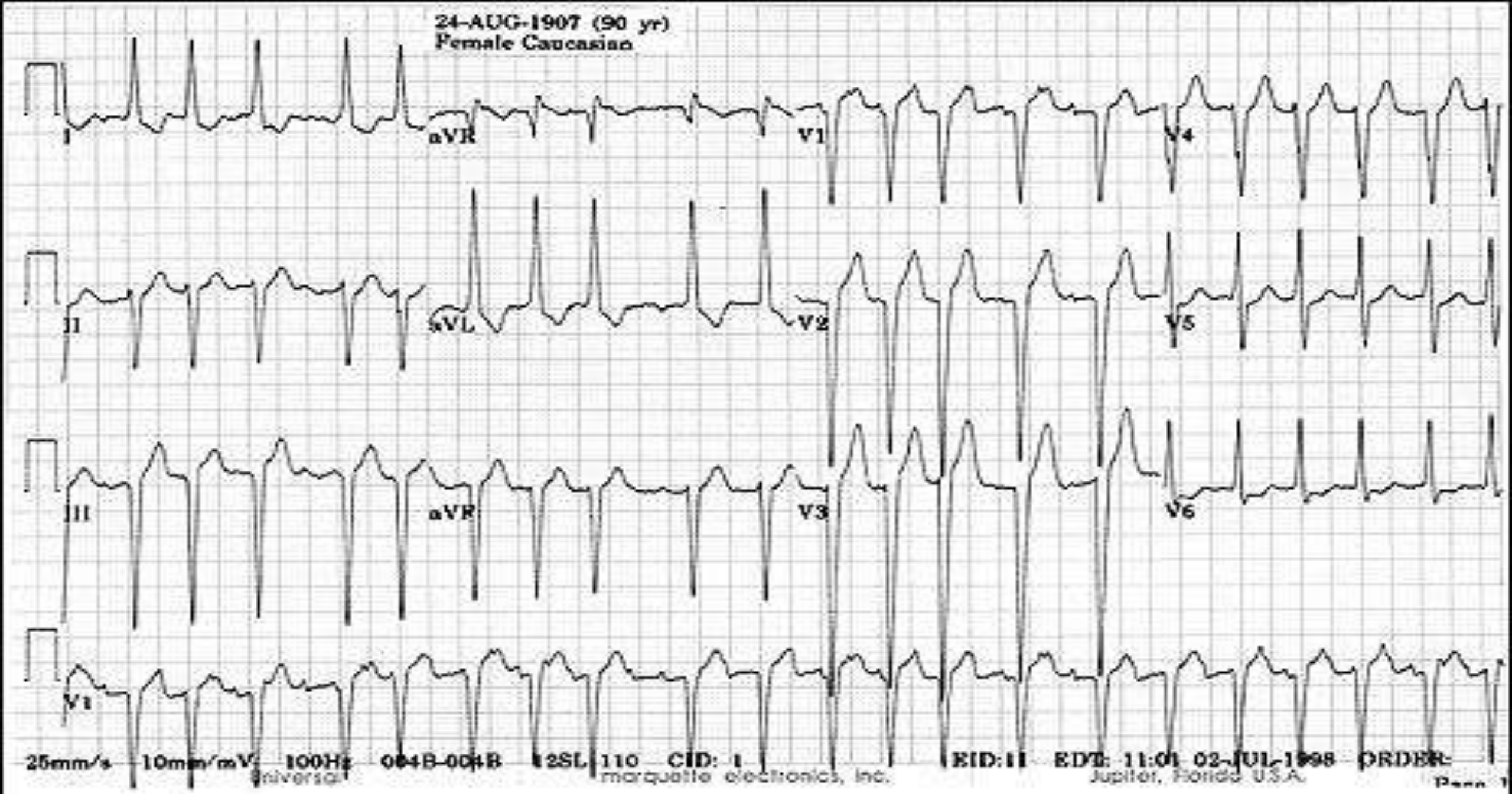


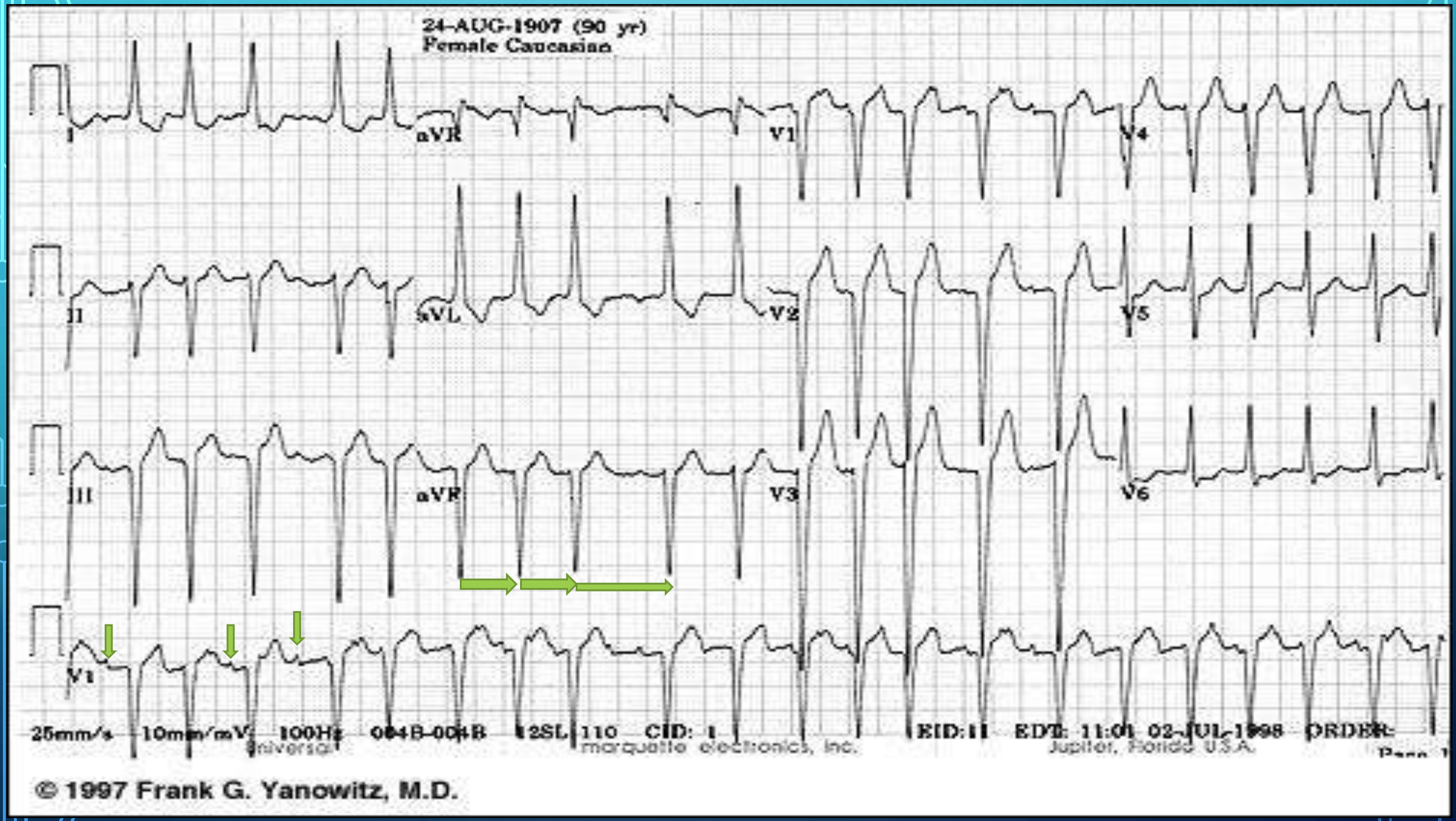

I
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: aVL
25 mm/sec; 1 cm/mV


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

AVRT

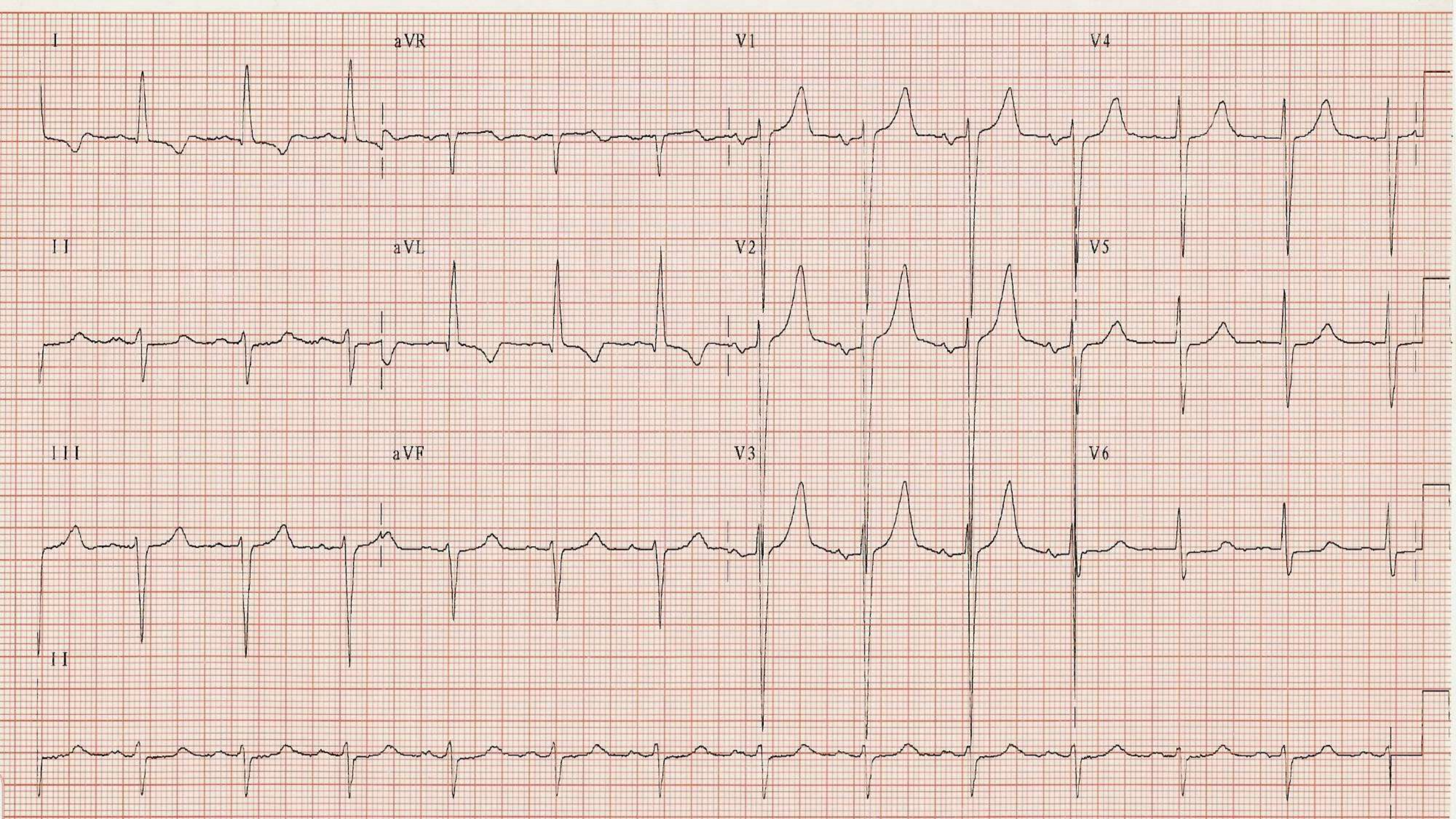
- شروع وختم ناگهانی
- ۱۵۰-۲۵۰
- No discernible P-waves
- Regular,faster than AVN،NARROW QRS
- ST depression during tachycardia
- Negative invert p before or<30ms after QRS excluded AP
- اگرموقعی که هیس دررفرکتوری است PVC موجa دهلیزی بدهد راه فرعی اثبات می شود
- VA<50% RR
- بطن ودهلیز اجزا اصلی مدار آریتمی وبلوک درهریک قطع آریتمی

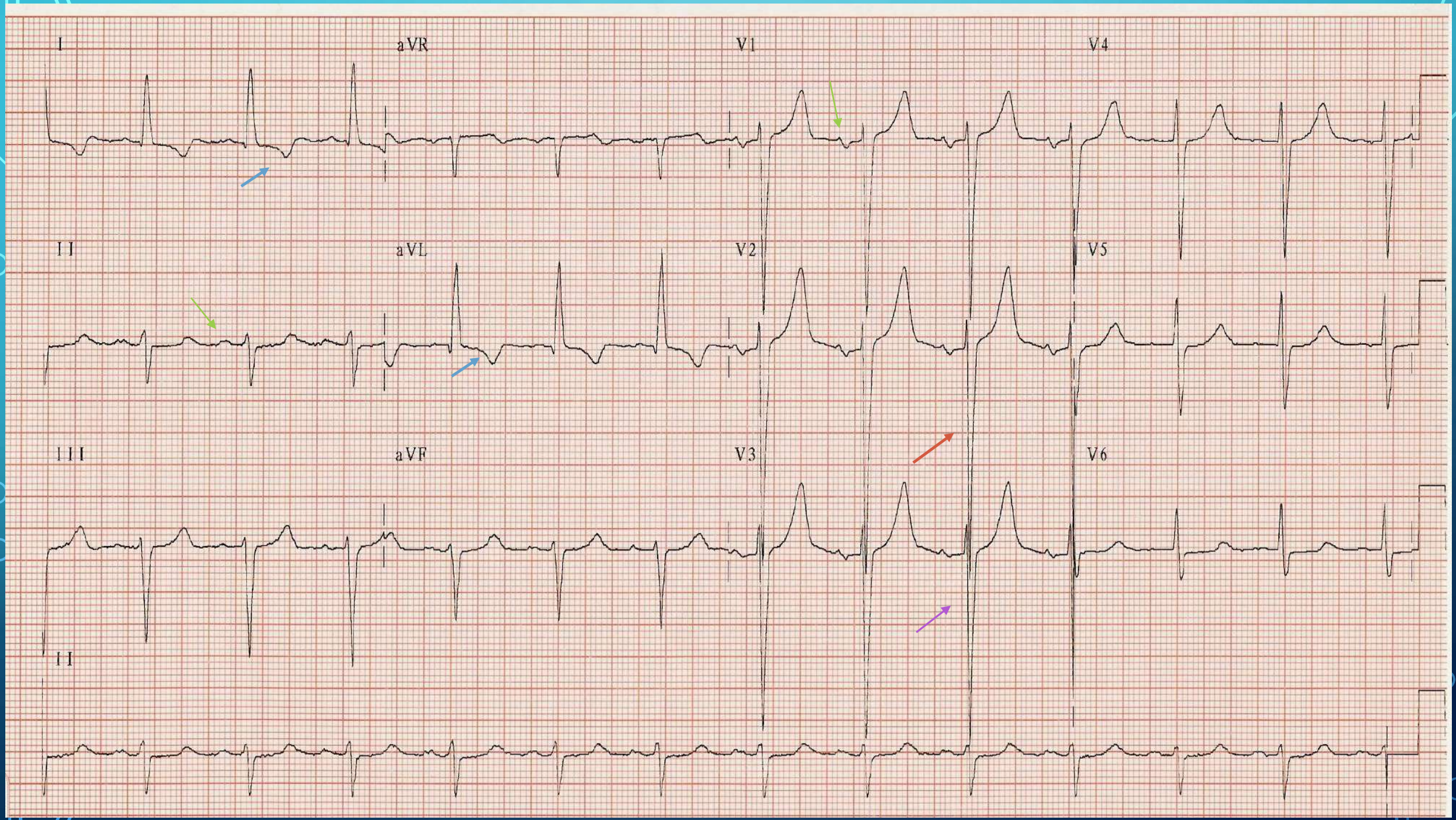


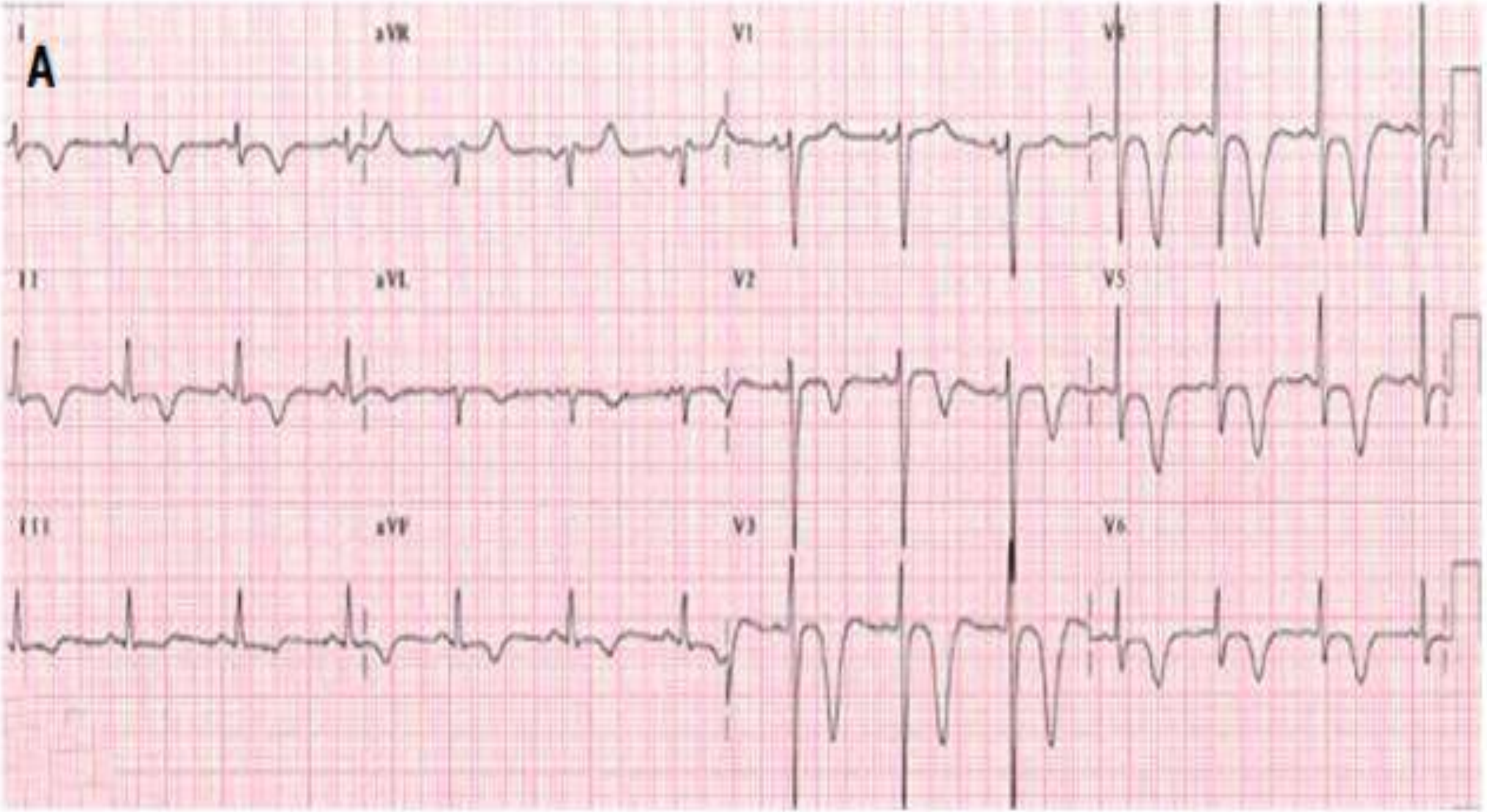


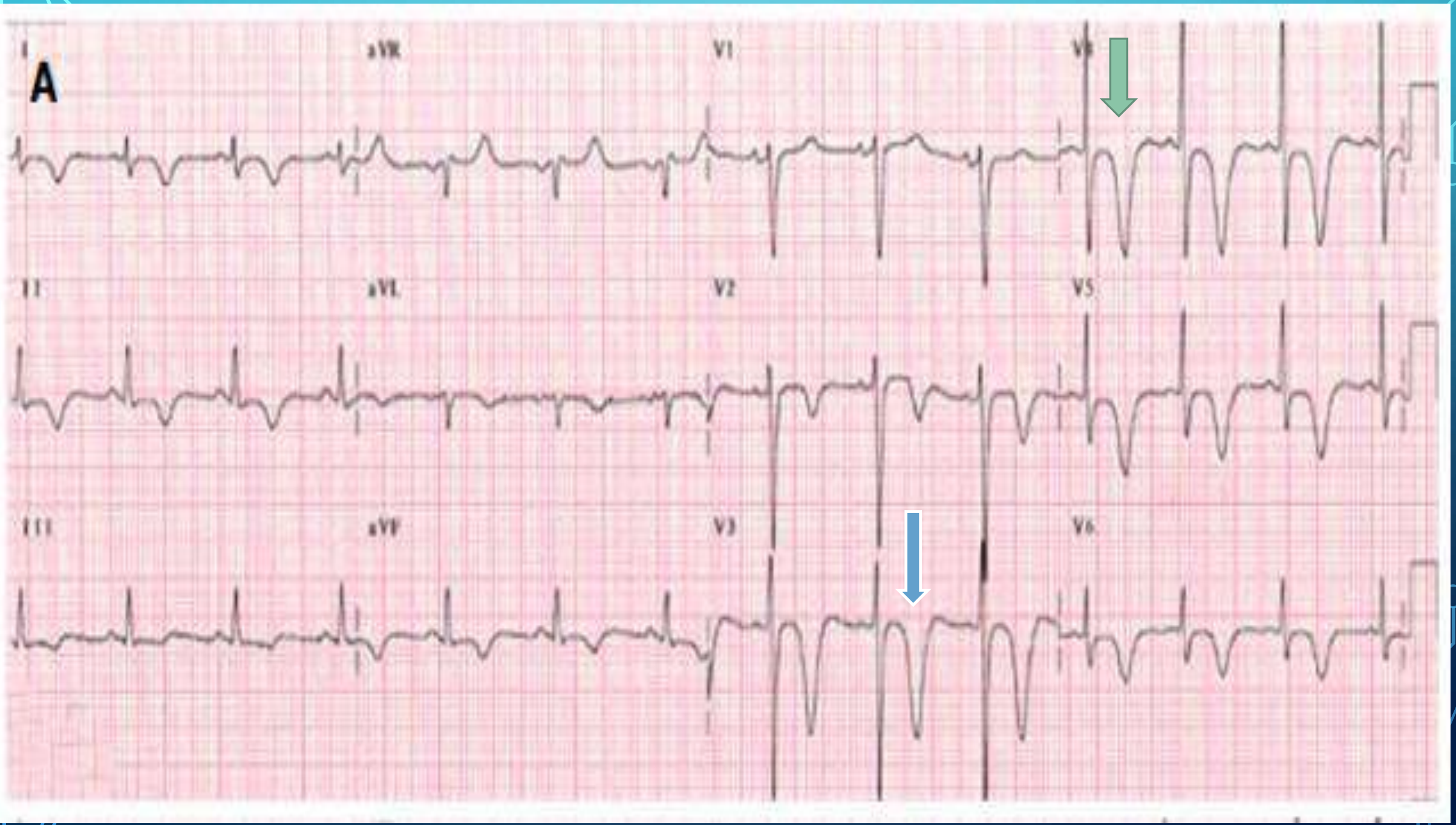
MAT

- Automatic atrial rhythm from various different foci
- HR:100-130
- Irregular PP-Irregular PR-different P wave (3 forms),isoelectric line
- Seen in hypoxia, COPD, atrial stretch and local metabolic imbalance. Old age-CHF-dig-theophylin
- Three or more types of p waves and a rate > 100
- treat with oxygen and slow channel blocker like verapamil or diltiazem. amiodaron-k-Mg-abl
- Shock is not useful


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

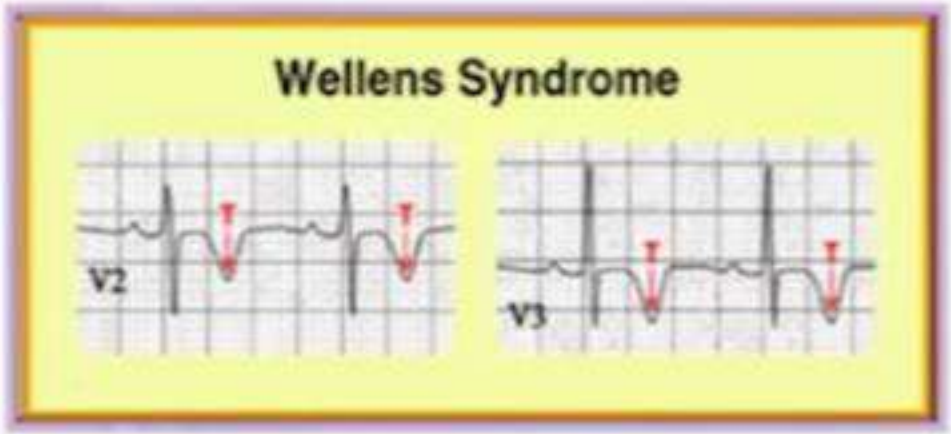

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II


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

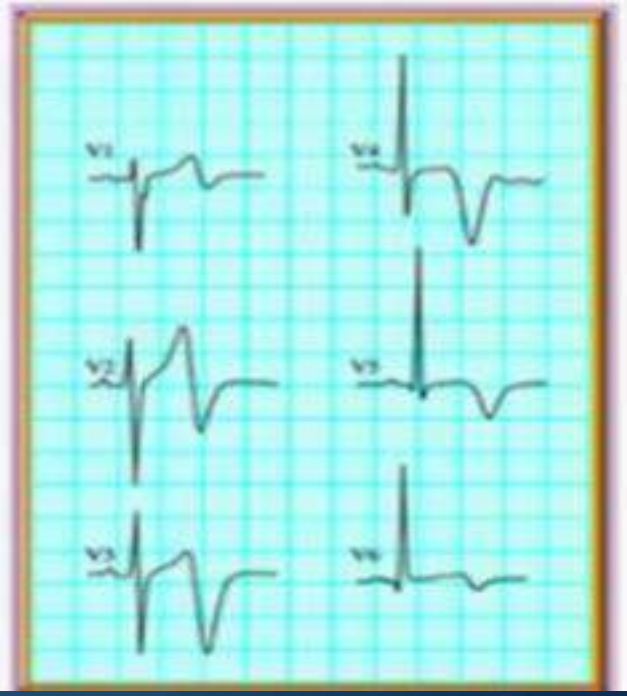

A
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Types of Wellens' Syndrome

Wellens' Type 1



Wellens' Type 2



Type 1 (A):

- Deep & symmetric T wave inversion in the mid-precordial leads.
- More common (75%).

Type 2 (B):

- Biphasic T wave in the mid-precordial leads.
- Less common (25%).

N.B.

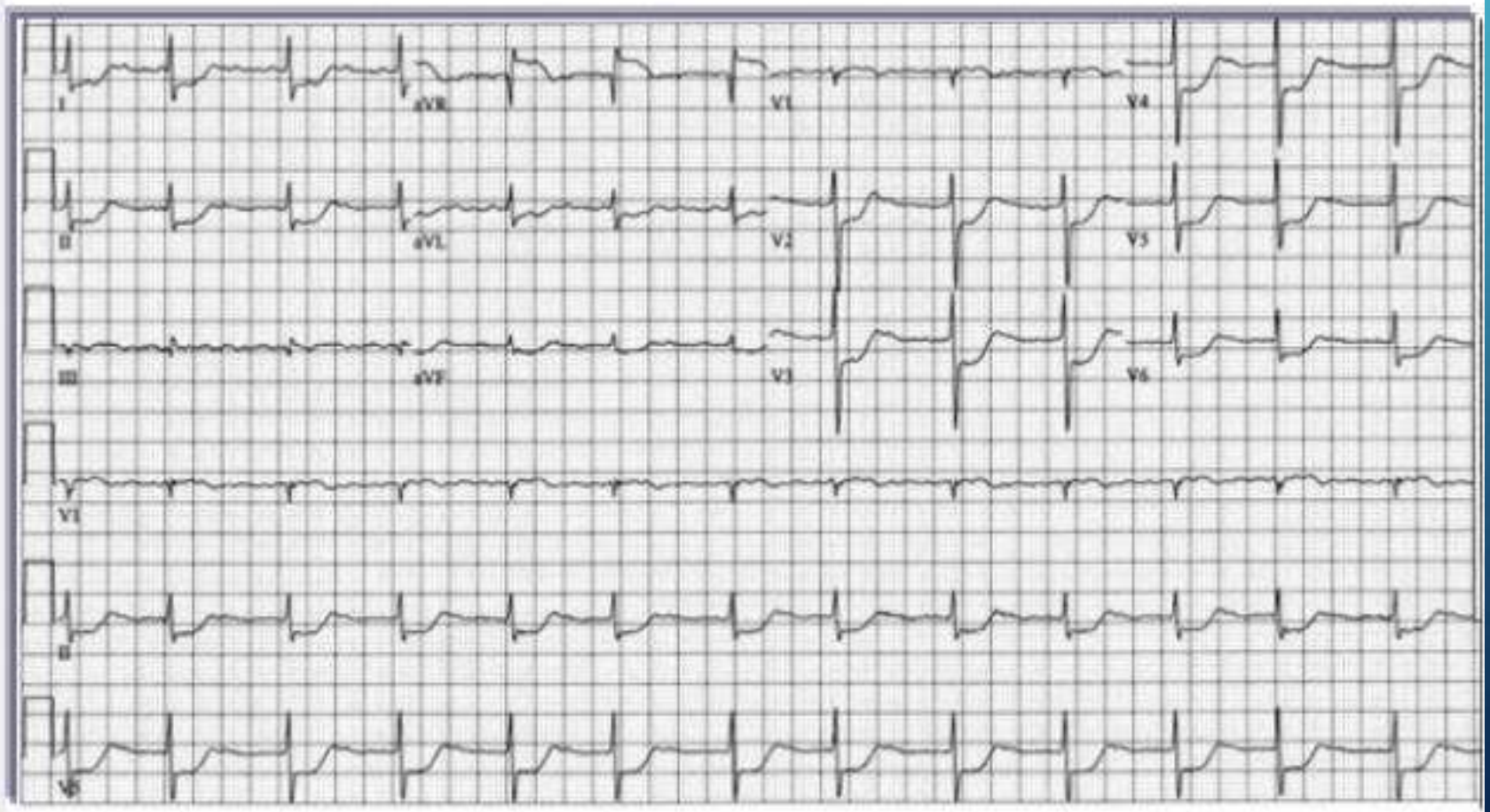
The T waves evolve over time from the symmetrical to the biphasic pattern.

Diagnostic Criteria

Rhinehart et al (2002)

1) Deeply-inverted or biphasic T waves in V2-3 (may extend to V1-6).
2) Isoelectric or minimally-elevated ST segment (<1mm).
3) No precordial Q waves.
4) Preserved precordial R wave progression.
5) Recent history of angina.
6) ECG pattern present in pain-free state.
7) Normal or slightly elevated serum cardiac markers.

"ST Elevation in aVR"



"Value of ST elevation in aVR" (2)(3)

In ST elevation in aVR + ST depression in multiple other leads, PLEASE consider:

1) LMCA occlusion, especially if:
   - ST elevation in aVR > V1.. (highly specific)
   - ST elevation in aVR & aVL..
2) Proximal LAD occlusion.
3) Triple vessel disease.

Why is it BAD?

1) ST elevation in aVR is directly proportionate to the mortality rate:
   - 0.5 mm → 10.8 %
   - 1 mm → 13.8 %
   - 1.5-2.5 mm → 22.2 %
   - > 3 mm → 50 %
2) Mortality is 70% without immediate PCI.
3) Medical treatment including thrombolysis does not improve the mortality!!!