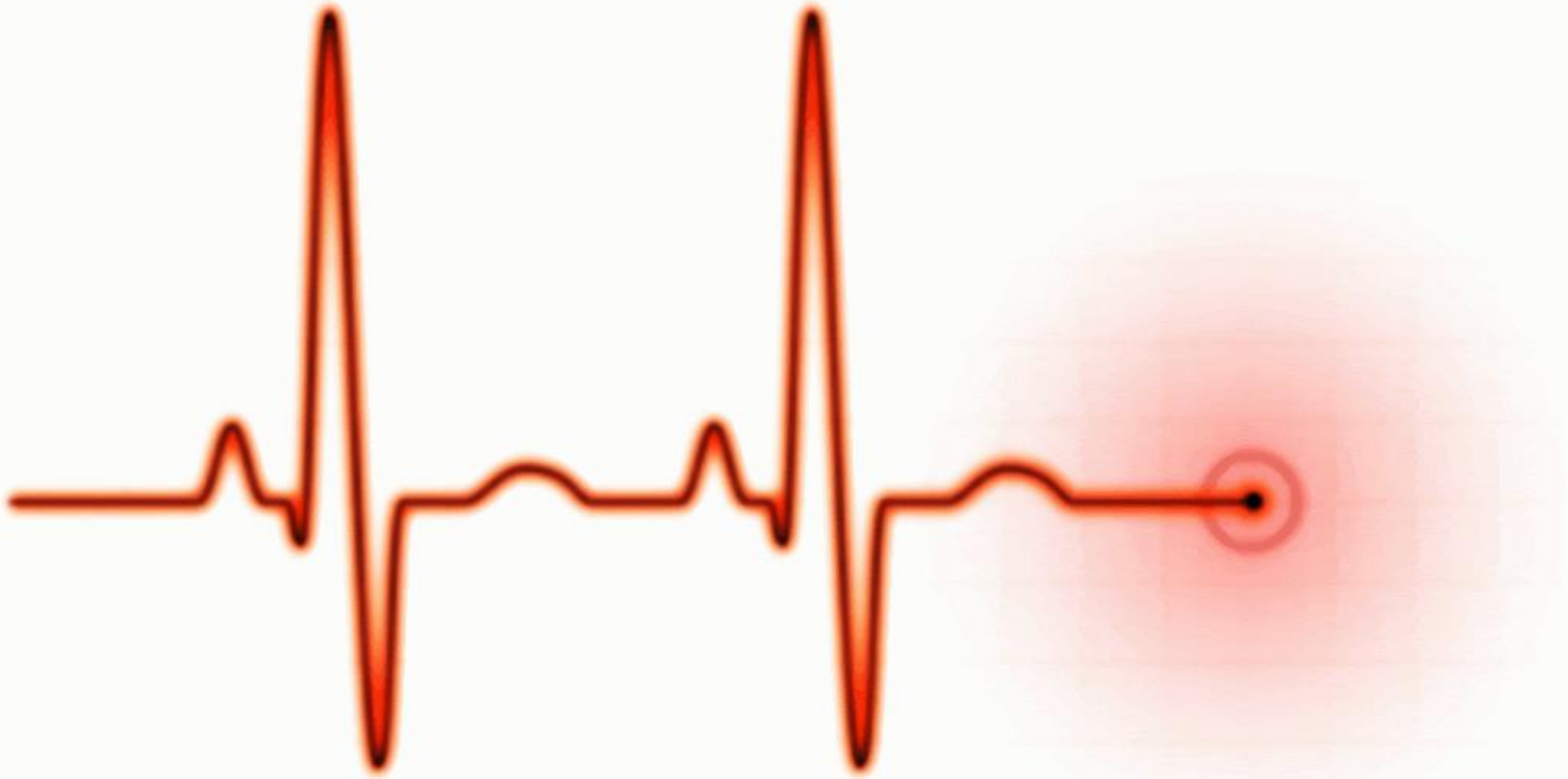
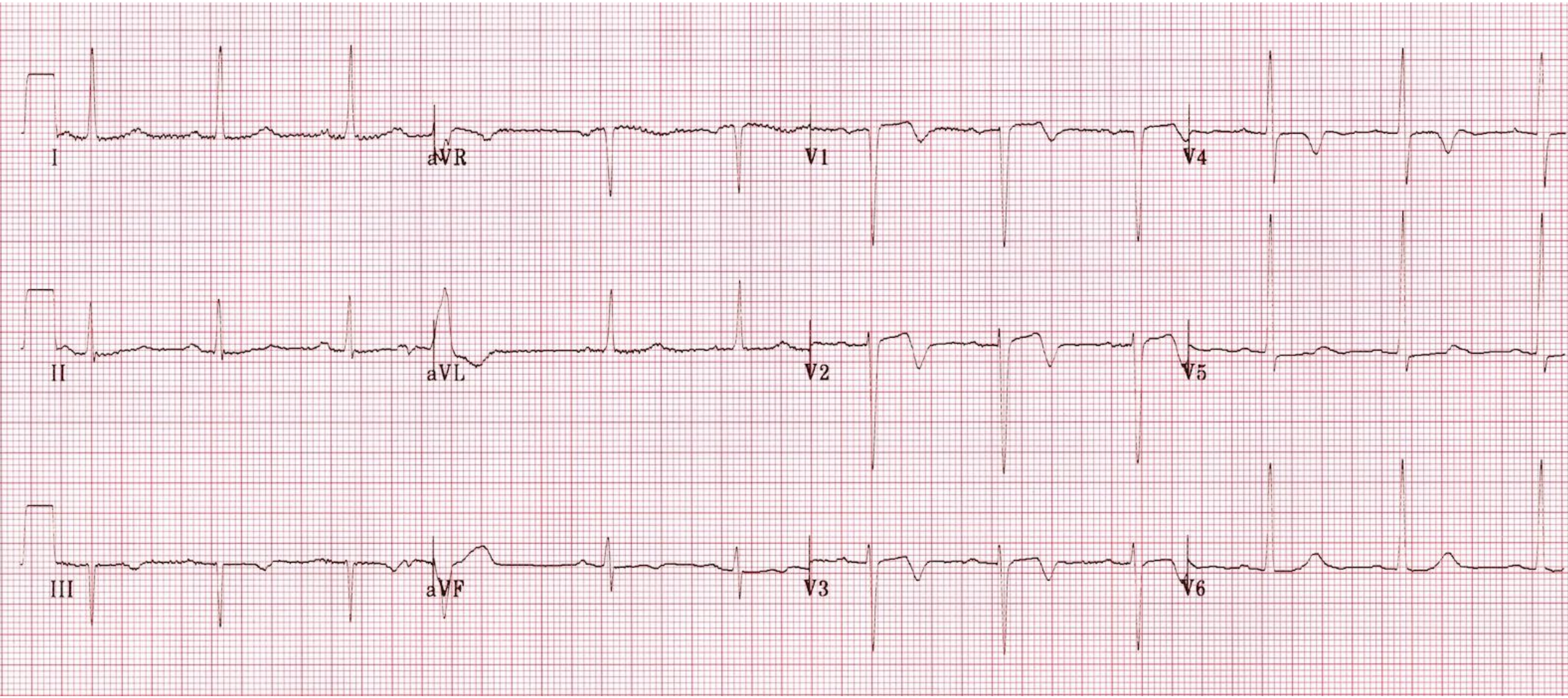


ECG Revision

Emergency Medicine Fellowship Program

Acknowledgement to Dr C Banks & LITFL

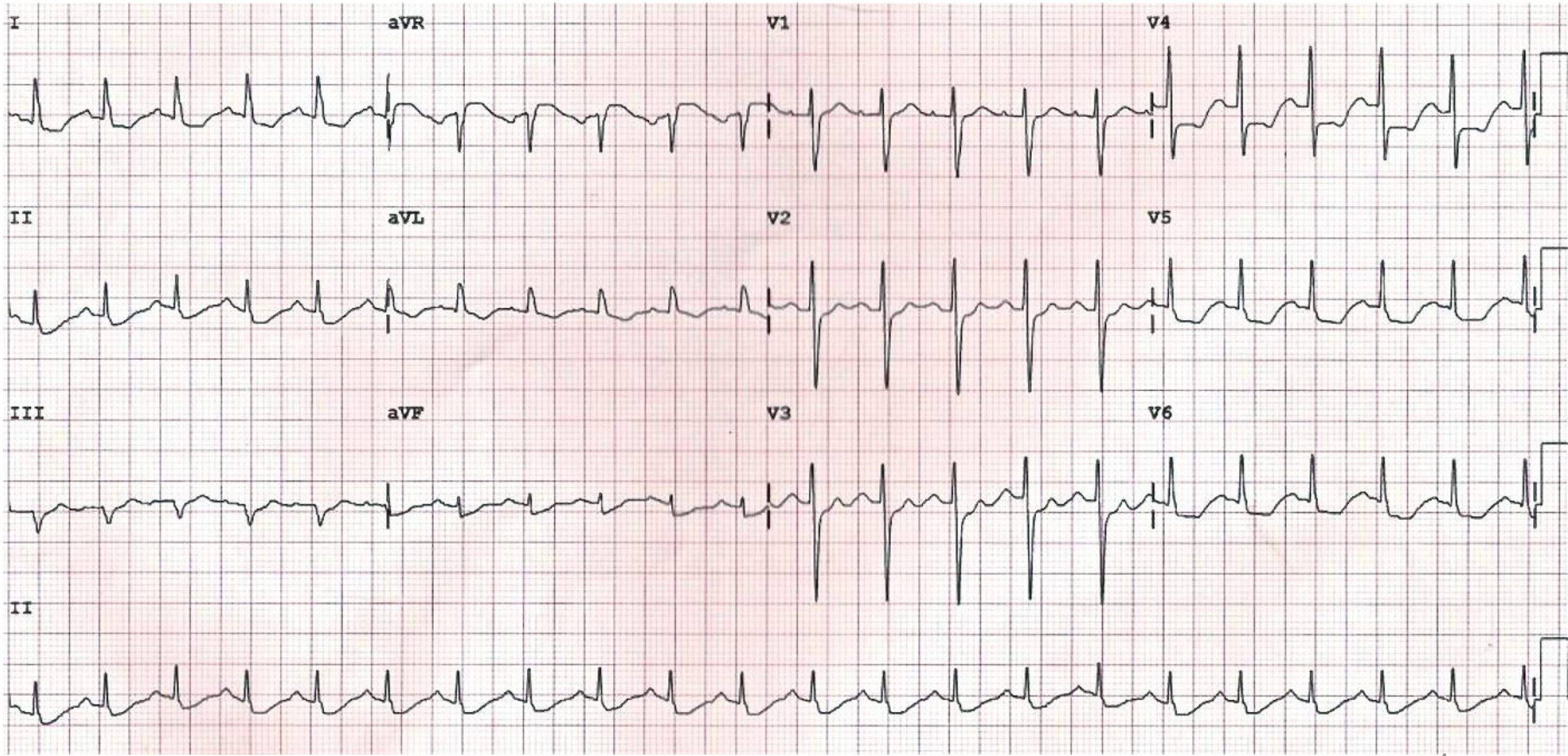

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Wellen's Syndrome

- de Zwaan C, Bär FW, Wellens HJ. Am Heart J. 1982 Apr;103(4 Pt 2):730-6.
  - 12 of 16 patients with UAP and Wellen's treated conservatively developed extensive anterior MI

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

- Types
  - Type A Biphasic 25%
  - Type B Inverted 75%


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

STE in aVR

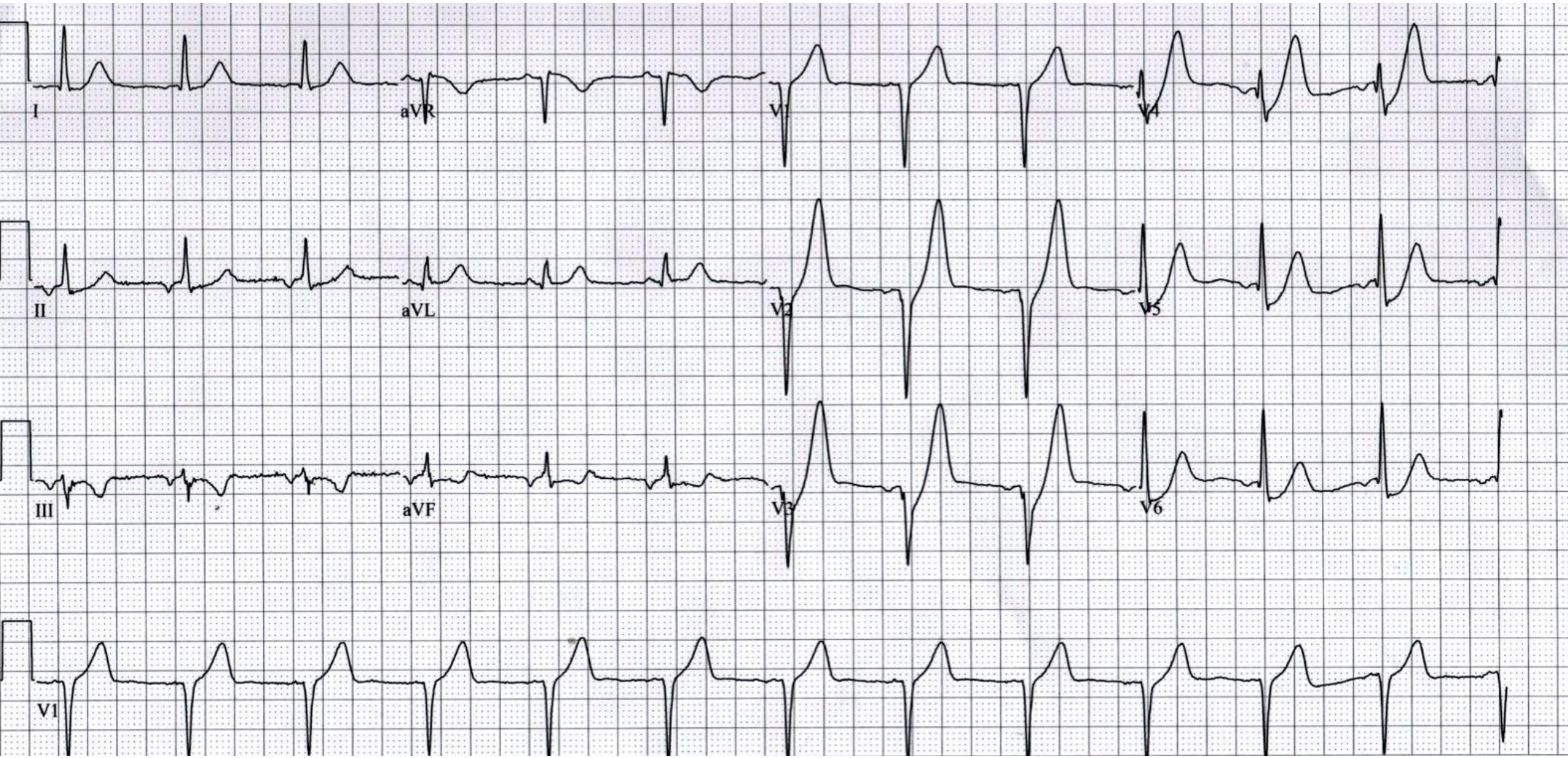
- Signifies
  - LM occlusion
  - Prox LAD occlusion
  - Severe TVD
  - Diffuse sub-endocardial ischaemia

An Early and Simple Predictor of Severe Left Main and/or Three-Vessel Disease in Patients With Non–ST-Segment Elevation Acute Coronary Syndrome

Masami Kosuge, MD*, Toshiaki Ebina, MD, Kiyoshi Hibi, MD, Satoshi Morita, PhD, Mitsuaki Endo, MD, Nobuhiki Maejima, MD, Noriaki Iwahashi, MD, Kozo Okada, MD, Toshiyuki Ishikawa, MD, Satoshi Umemura, MD, and Kazuo Kimura, MD

Clopidogrel should be initiated as soon as possible in patients with non–ST-segment elevation acute coronary syndrome (NSTE-ACS) except those who urgently require coronary artery bypass grafting (CABG). The present study assessed the ability to predict severe left main coronary artery and/or 3-vessel disease (LM/3VD) that would most likely require urgent CABG based on only clinical factors on admission in 572 patients with NSTE-ACS undergoing coronary angiography. Severe LM/3VD was defined as ≥75% stenosis of LM and/or 3VD with ≥90% stenosis in ≥2 proximal lesions of the left anterior descending coronary artery and other major epicardial arteries. Patients were divided into the 3 groups according to angiographic findings: no LM/3VD (n = 460), LM/3VD but not severe LM/3VD (n = 57), and severe LM/3VD (n = 55). Severe LM/3VD was associated with a higher rate of urgent CABG compared to no LM/3VD and LM/3VD but not severe LM/3VD (46%, 2%, and 2%, p <0.001). On multivariate analysis, degree of ST-segment elevation in lead aVR was the strongest predictor of severe LM/3VD (odds ratio 29.1, p <0.001), followed by positive troponin T level (odds ratio 1.27, p = 0.044). ST-segment elevation ≥1.0 mm in lead aVR best identified severe LM/3VD with 80% sensitivity, 93% specificity, 56% positive predictive value, and 98% negative predictive value. In conclusion, ST-segment elevation ≥1.0 mm in lead aVR on admission electrocardiogram is highly suggestive of severe LM/3VD in patients with NSTE-ACS. Selected patients with this finding might benefit from promptly undergoing angiography, withholding clopidogrel to allow early CABG. (Am J Cardiol 2011;107: 495–500)

	Sensitivity	Specificity	PPV	NPV	Predictive Accuracy
ST-segment elevation in lead aVR					
≥0.5 mm	91%	79%†	32%†	99%	80%†
≥1.0 mm	80%	93%	56%	98%	92%
≥1.5 mm	27%†	98%†	58%	93%†	91%
Positive troponin T	60%*	69%†	17%†	94%†	68%†


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

de Winter T Waves

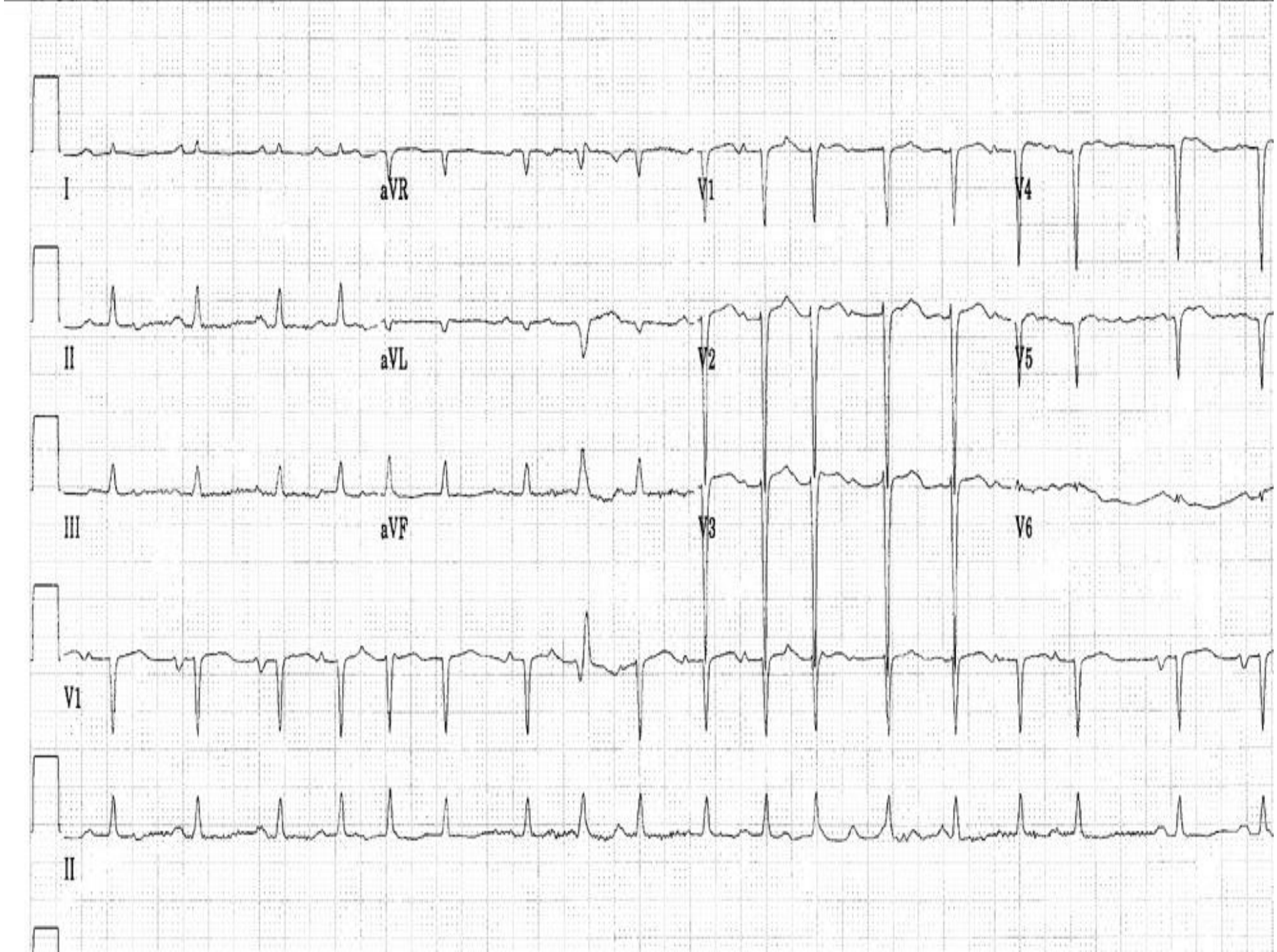
- De Winter RJ, Verouden NJ, Wellens HJ, et al. N Engl J Med 2008;359:2071–3
  - anterior STEMI equivalent
  - ST depression and peaked T waves in the precordial leads
  - ~2% of acute LAD occlusions

Persistent precordial ''hyperacute'' T-waves signify proximal left anterior descending artery occlusion

N J Verouden,[1] K T Koch,[1] R J Peters,[1] J P Henriques,[1] J Baan,[1] R J van der Schaaf,[1] M M Vis,[1] J G Tijssen,[1] J J Piek,[1] H J Wellens,[2] A A Wilde,[1] R J de Winter[1]

Heart 2009;**95**:1701–1706. doi:10.1136/hrt.2009.174557

Conclusions: In patients presenting with chest pain, ST-segment depression at the J-point with upsloping ST-segments and tall, symmetrical T-waves in the precordial leads of the 12-lead ECG signifies proximal LAD artery occlusion. It is important for cardiologists and emergency care physicians to recognise this distinct ECG pattern, so they can triage such patients for immediate reperfusion therapy.


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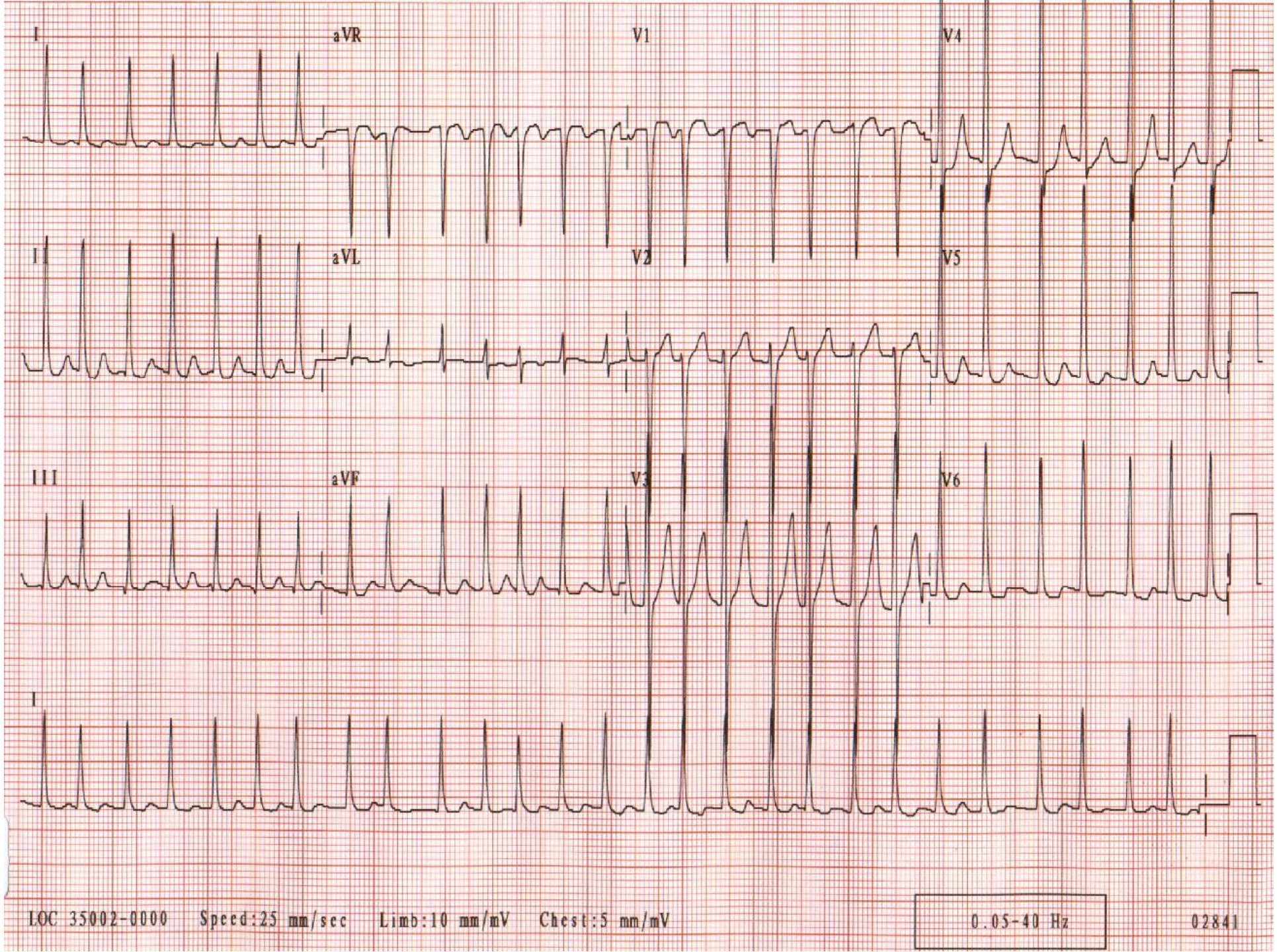
II
aVL
V2
V5
III
aVF
V3
V6
V1
II


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
I
LOC 35002-0000 Speed:25 mm/sec Limb:10 mm/mV Chest:5 mm/mV
0.05-40 Hz
02841

Atrial fibrillation

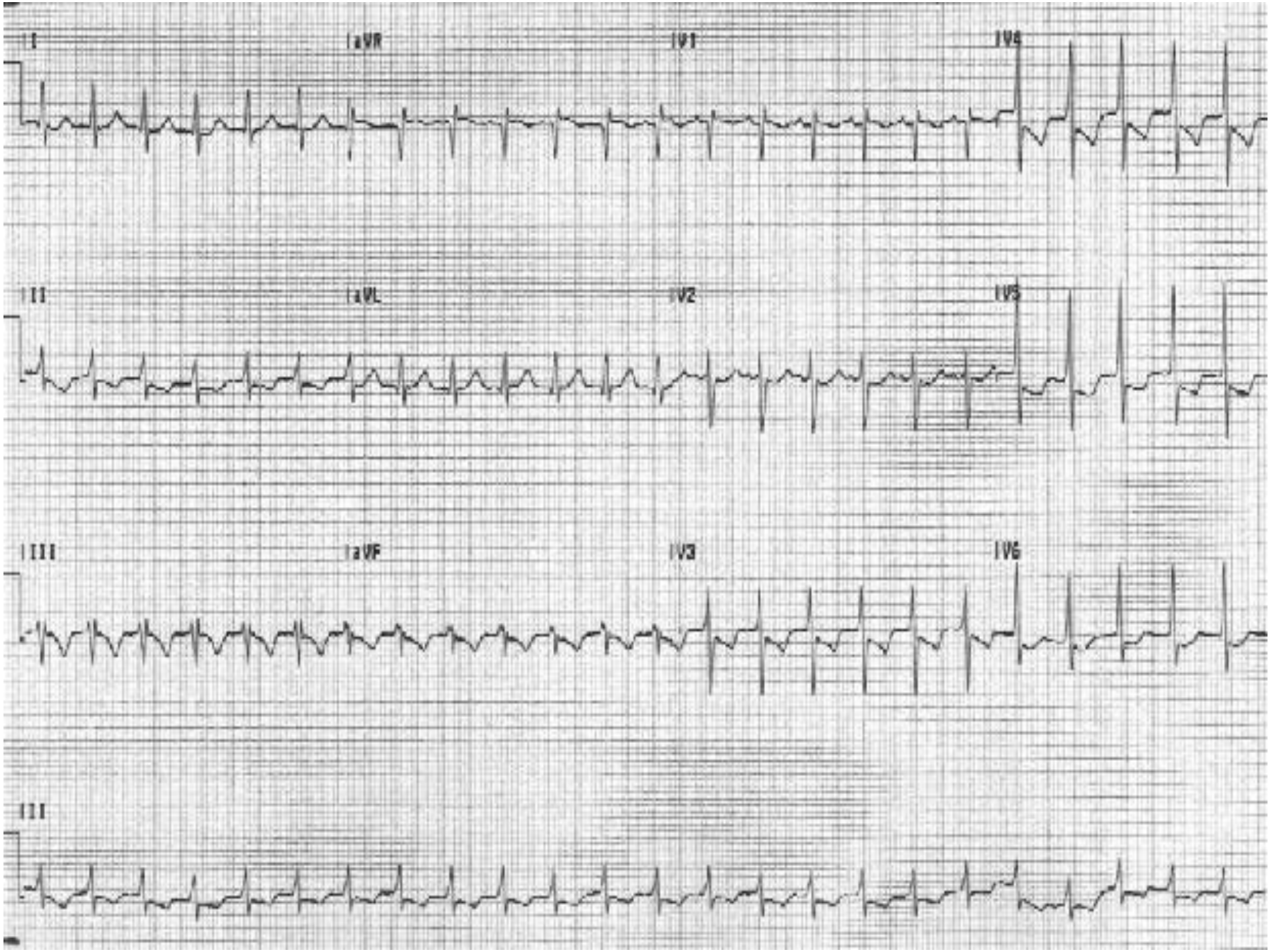
- 3 Ps
  - Paroxysmal
  - Persistent
  - Permanent (chronic)

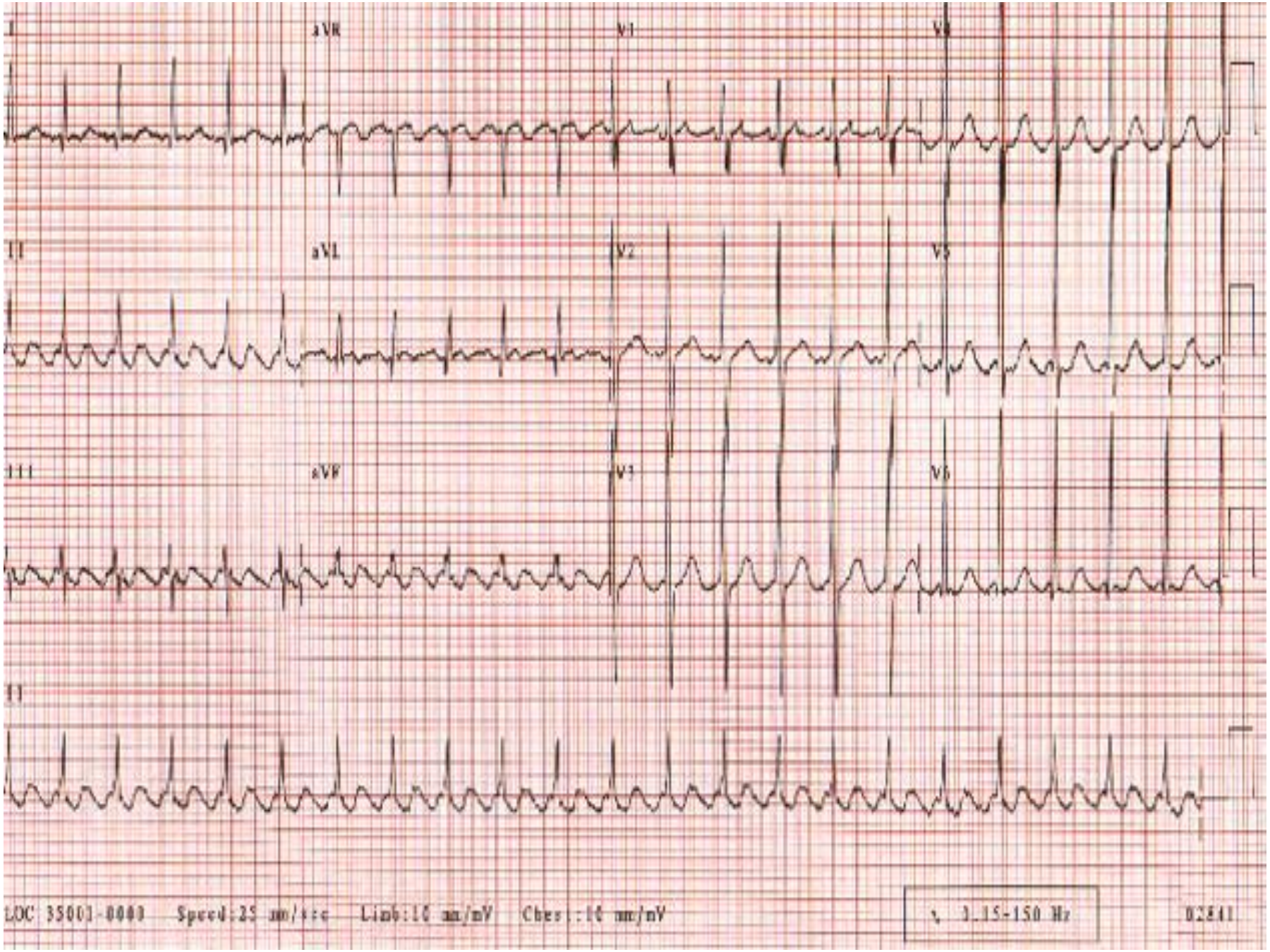
- Rhythm control vs. rate control
  - AFFIRM trial NEJM 2002

- Anticoagulation
  - Everyone unless contraindicated
  - Atrial stunning

Atrial fibrillation

- Rhythm
  - Revert only if
    - < 48 hrs
      - or negative TOE
      - or anticoagulated
  - Amiodarone
  - DCR
    - 100J synchronised
  - Flecainide
- Rate
  - Digoxin
  - B blockers
    - Atenolol
    - Metoprolol
  - Ca channel blockers
    - Verapamil
    - Diltiazem

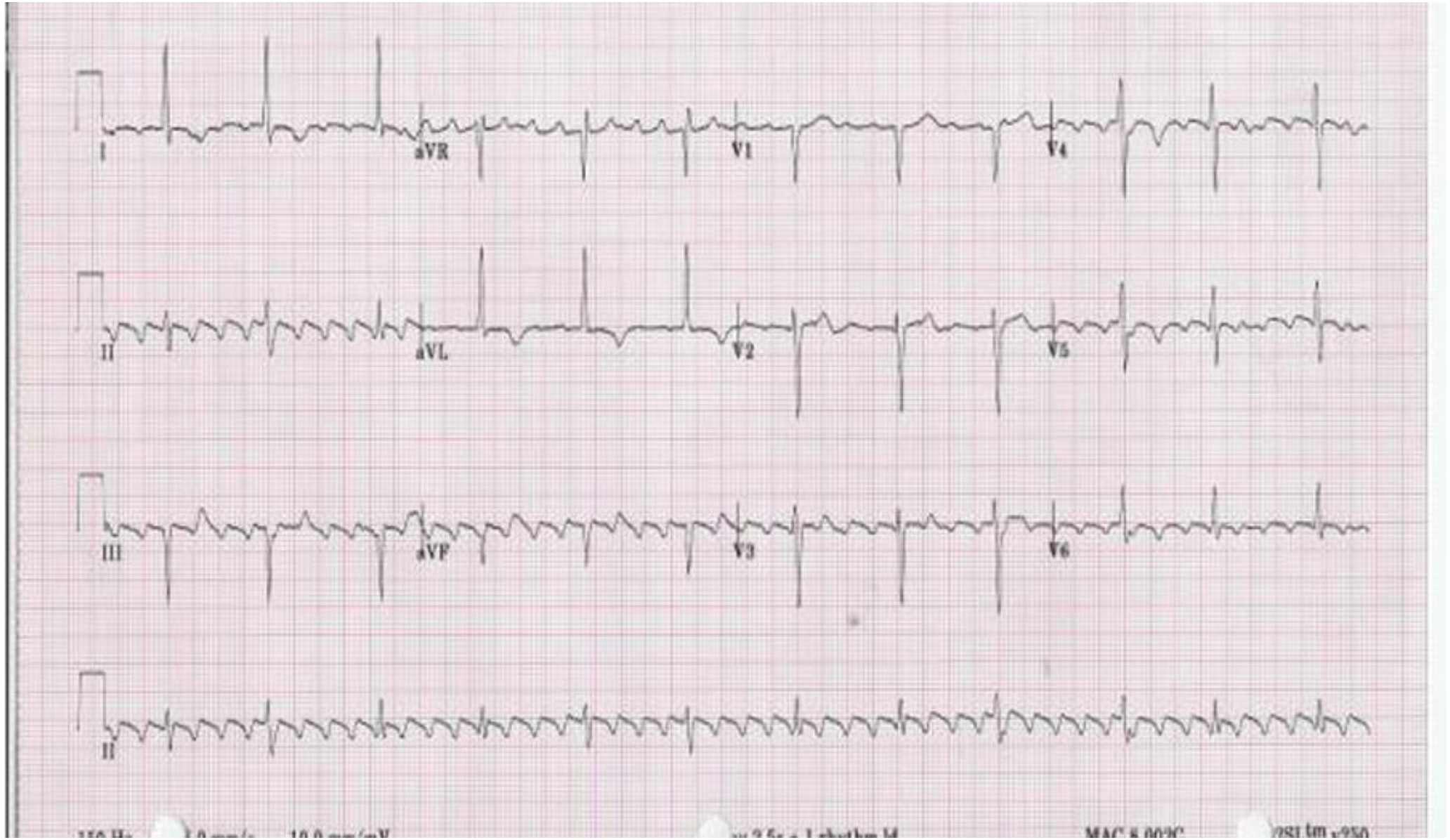

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
III


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
LOC 35001-0003 Speed:25 mm/sec Limb:10 mm/mV Ches::10 mm/mV
0.15-150 Hz
02841

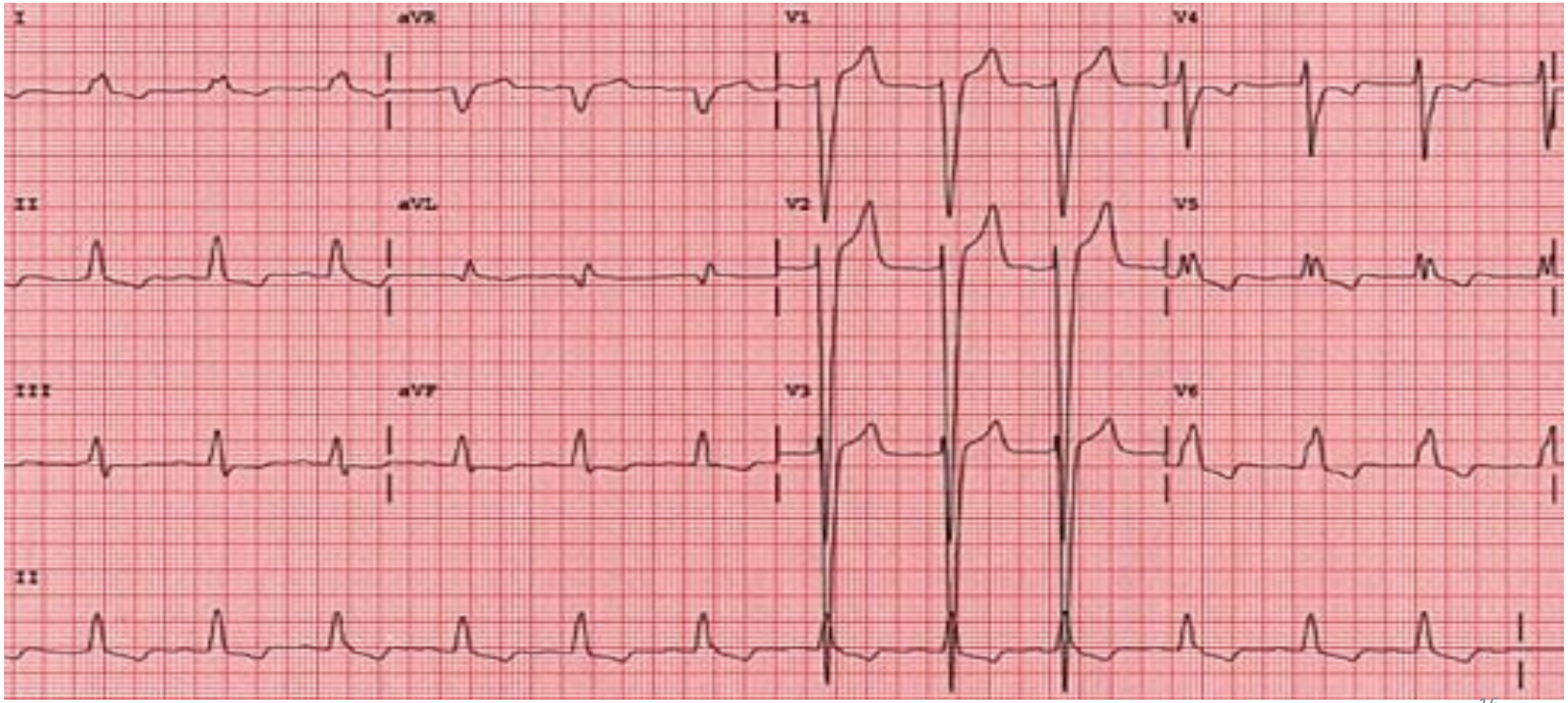
Atrial flutter

- Anticoagulate as per atrial fibrillation
- More drug resistant
- More DCR sensitive
  - Use 50J synchronised

Another Example

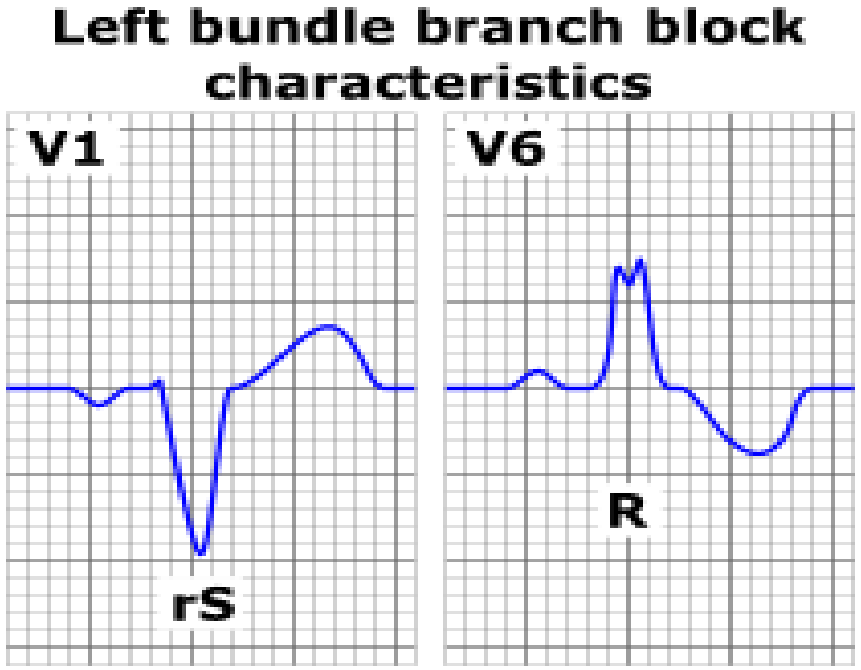


LBBB



LBBB

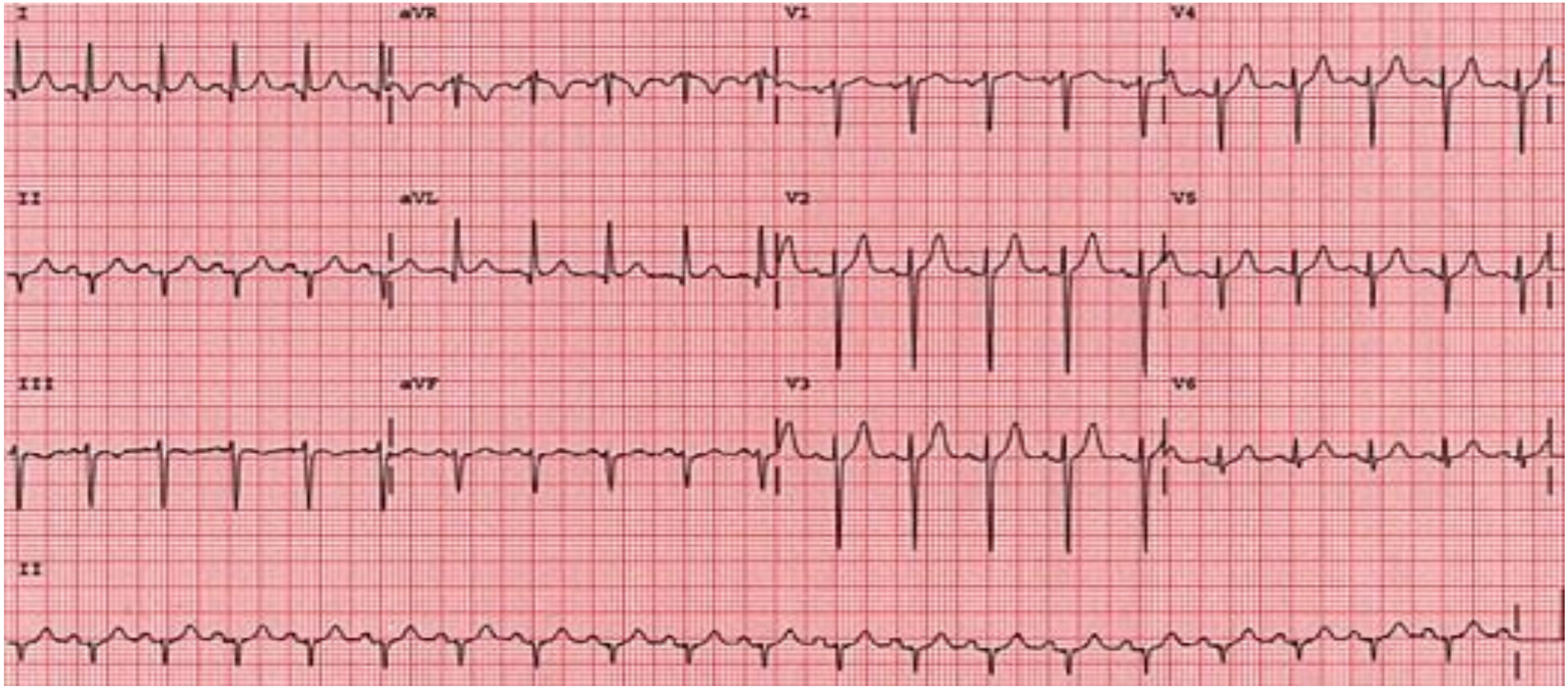
- Always pathological
- Bifascicular block
- Heart depolarises from right to left
  - ie right ventricle first
- Can have LAD if severe
- Discordant ST segments and T waves
  - Appropriate discordance
  - V1-6
- Paced rhythm produces similar picture



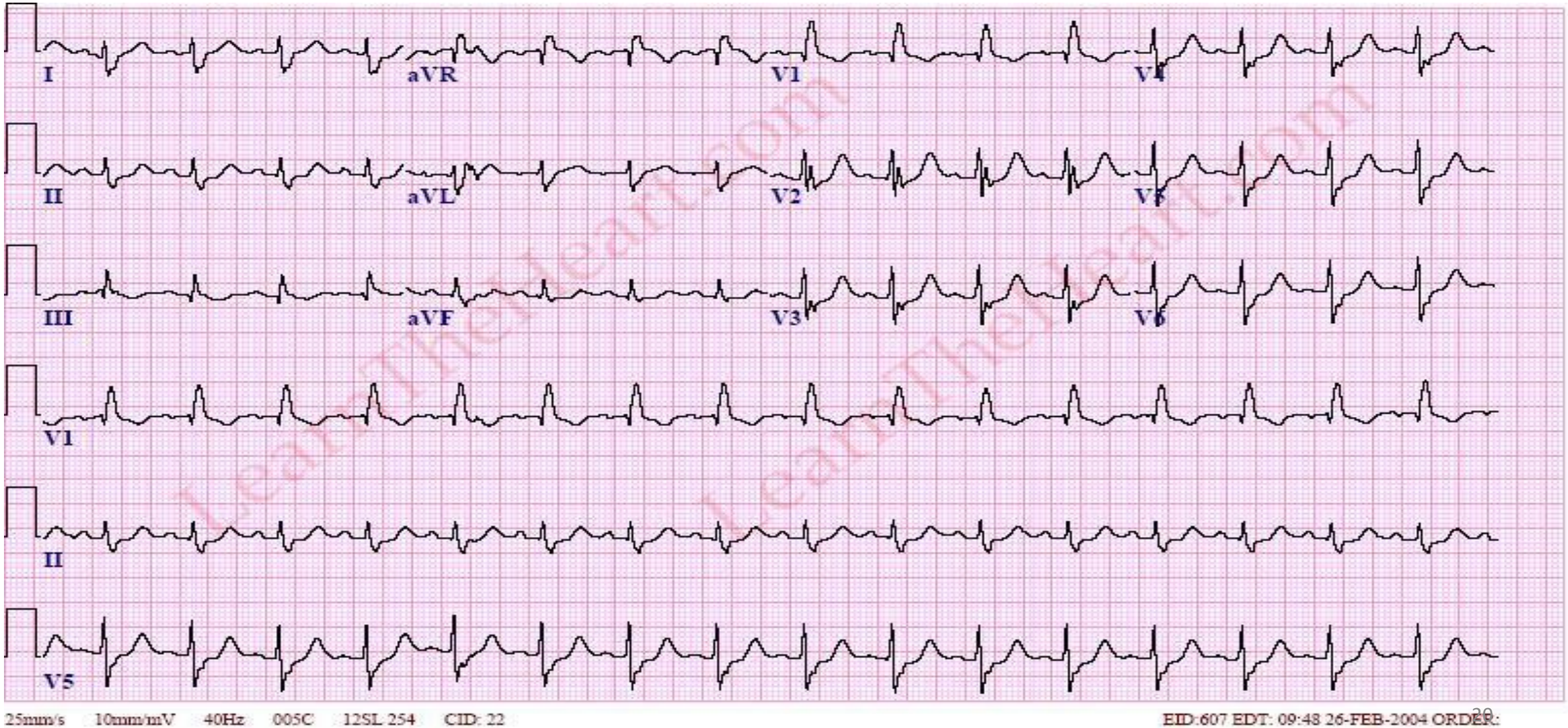
Bundle Branch Blocks

- Conduction along right or left bundle is blocked or delayed
  - ie degrees of severity
- Must be a supraventricular rhythm
- QRS => 0.12ms
- If < 0.12 but abnormal pattern
  - Non specific intraventricular conduction delay
  - Incomplete BBB

LAHB

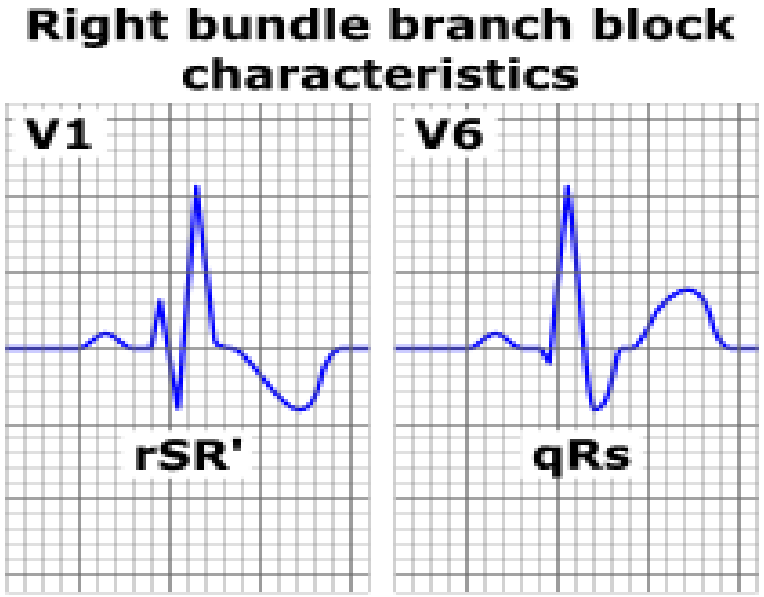


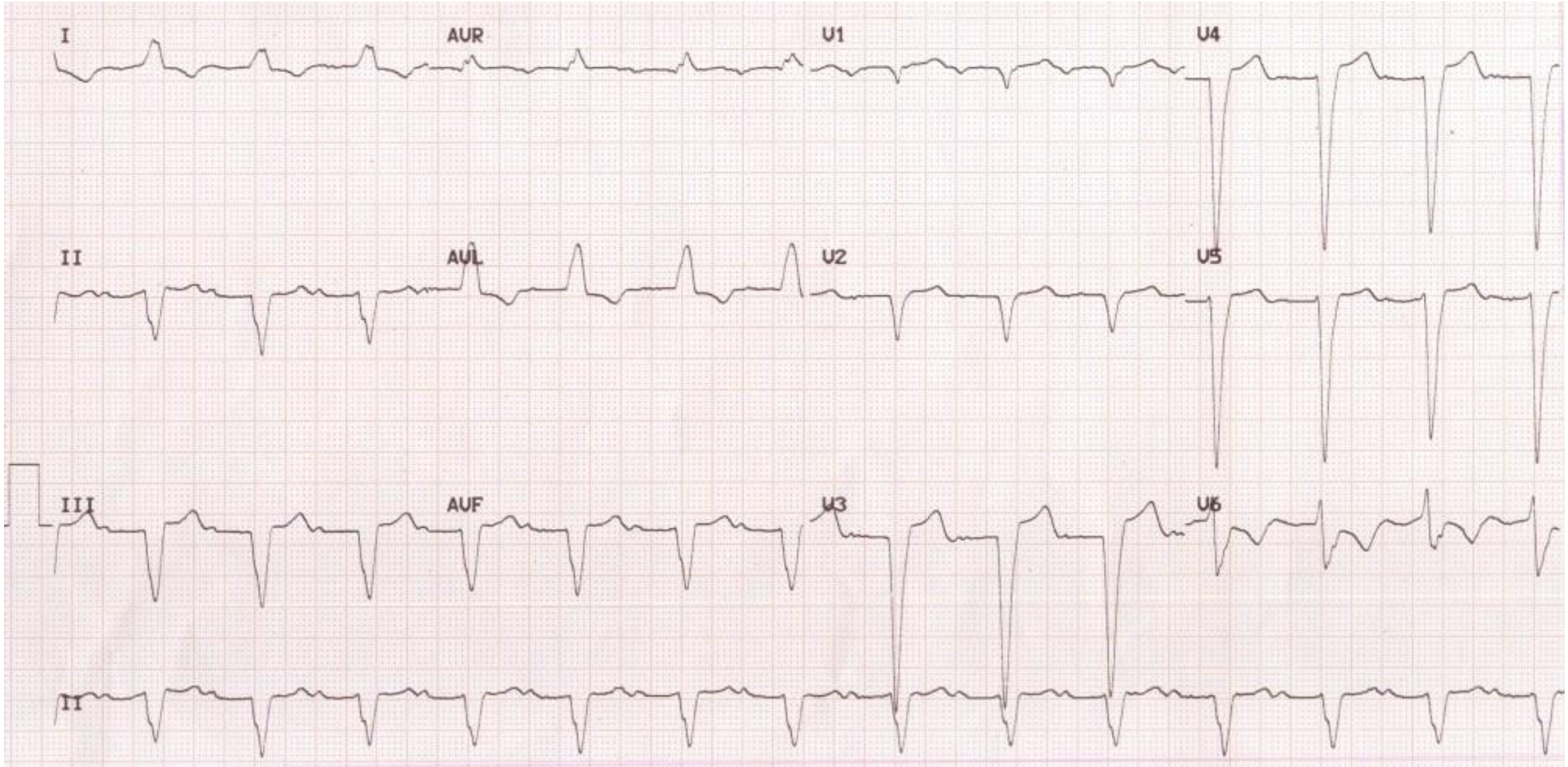
RBBB + LPHB


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 254 CID: 22
EID:607 EDT: 09:48 26-FEB-2004 ORDER:

RBBB

- Can be normal
- Heart depolarises from left to right
- Discordant ST segments and T waves
  - Appropriate discordance
  - V1-3 only
- No axis change

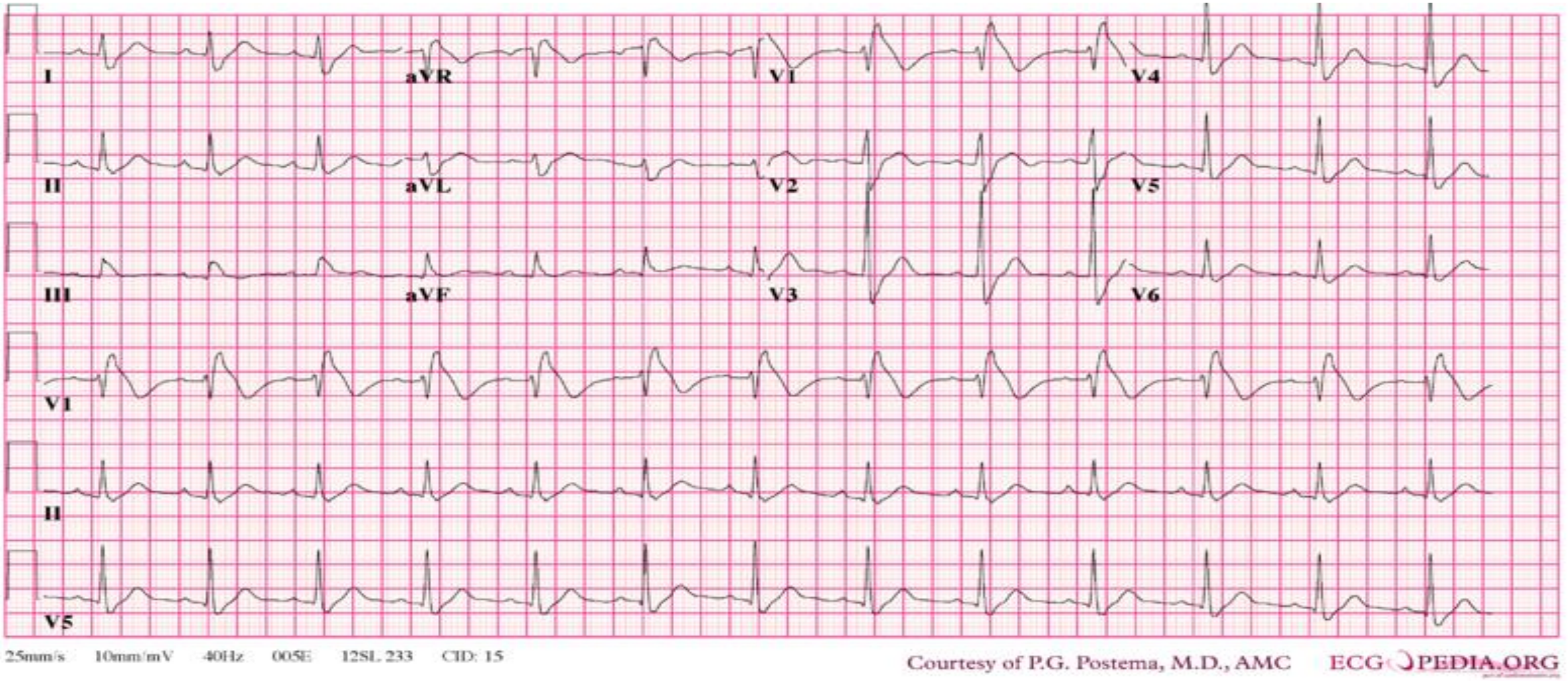



I
AUR
U1
U4
II
AUL
U2
U5
III
AUF
U3
U6
II

Trifascicular Block

- Complete
  - Bifascicular block + 3rd degree AV block
- Incomplete
  - Bifascicular block + 1st degree AV block (most common)
  - Bifascicular block + 2nd degree AV block
  - RBBB + alternating LAFB / LPFB
- Significance
  - Can deteriorate into CHB
  - Symptomatic
    - Pacemaker (or consideration)
  - Asymptomatic
    - Nil

Seizure [First episode]

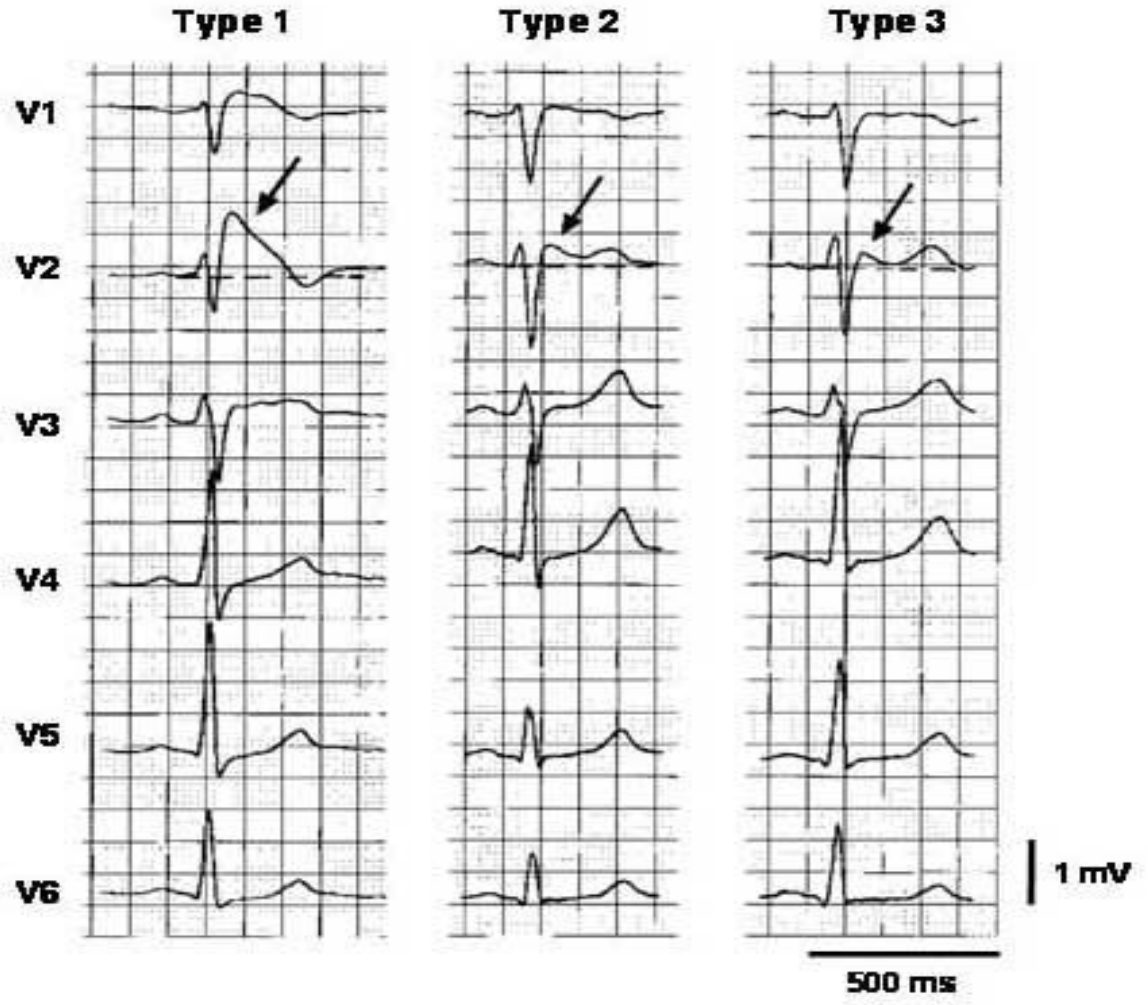


Brugada Syndrome

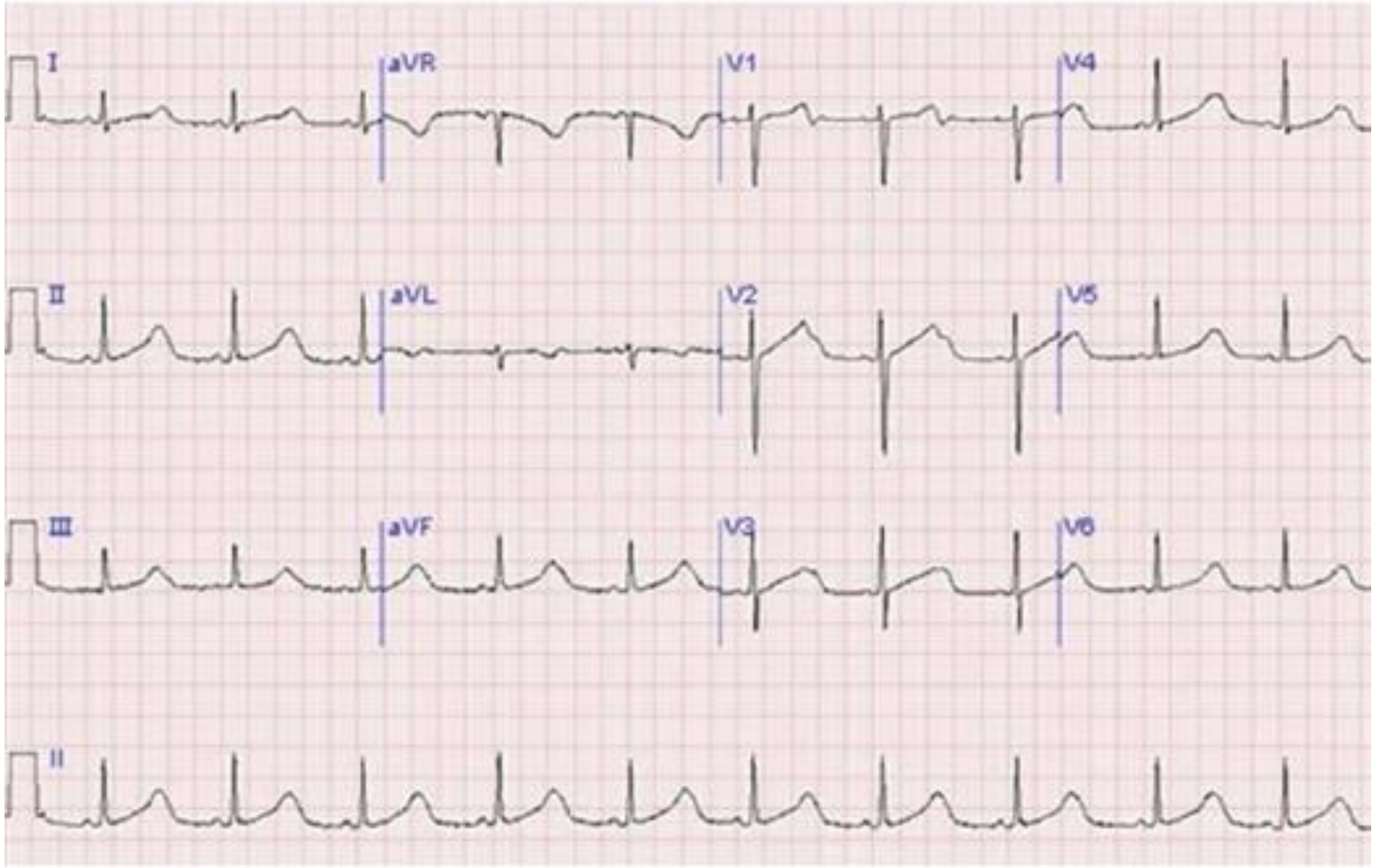
- Genetic abnormality that leads to polymorphic VT or VF
- Na channelopathy first described in 1992
  - 20-30% linked to SCN5A gene on chromosome 3
  - > 100 mutations of this gene have been described
  - Variable penetrance
  - Autosomal dominant
- Combination of (aborted) sudden death, syncope with Brugada pattern ECG
  - rSR in V1-3 (RBBB)
  - Downsloping ST elevation
- Treatment is an implantable cardioversion device (ICD)

Types of Brugada Syndrome

- Type 1
  - Diagnostic
- Type 2 & 3
  - Require unmasking
  - Na channel blockers
  - Type 2
    - Saddleback
    - Japan



Syncope



Long QT

- QT prolonged at 450ms
  - ? Longer in women
- Causes TdP
- Multiple formulae

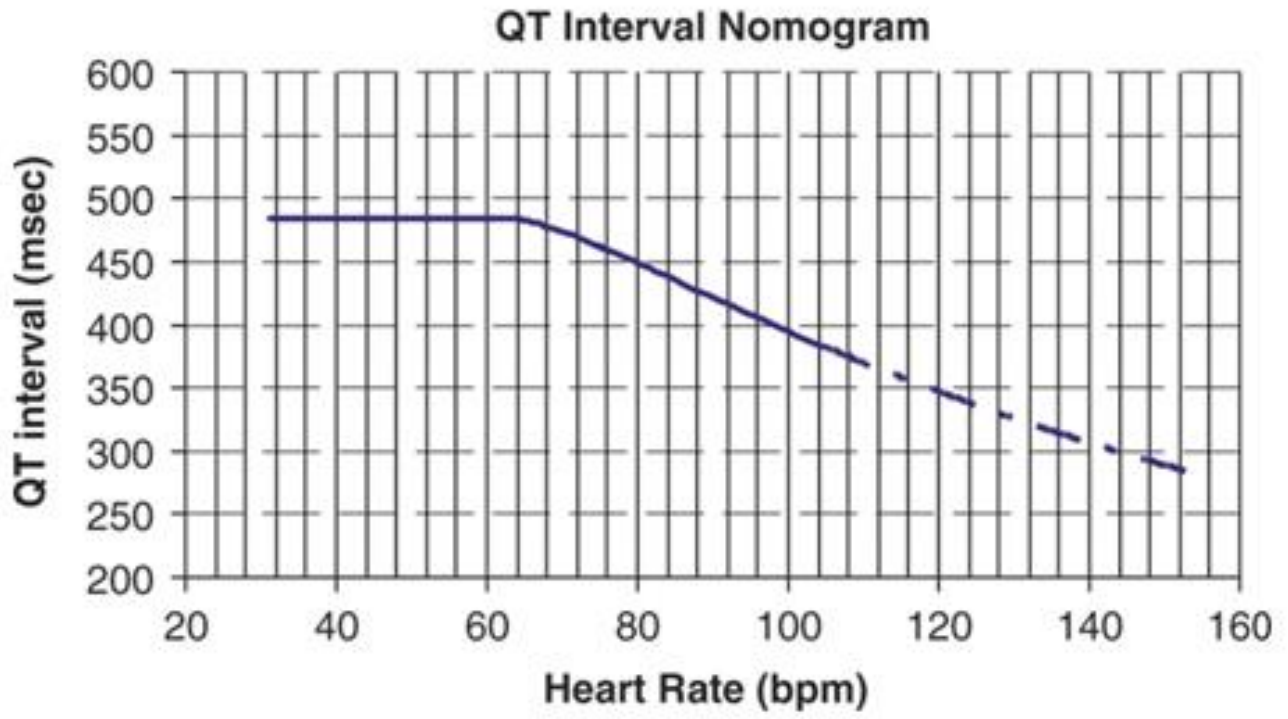
- Causes
  - Congenital
    - Romano Ward
    - Jervell Lange Nielsen
  - Acquired
    - Hypokalaemia
    - Hypomagnesaemia
    - Hypocalcaemia
    - Hypothermia
    - Myocardial ischemia
    - Post-cardiac arrest
    - Raised intracranial pressure
    - DRUGS

Formulae

- Bazett
  - $QTc = QT / (RR)^{1/2}$
- Fridericia
  - $QTc = QT / (RR)^{1/3}$
- Framingham
  - $QTc = QT + 0.154(1-RR)$
- Hodges
  - $QTc = QT + 1.75(HR-60)$
- Nomogram

- Bazett
  - What ECG machine uses
  - Worst of all
- Nomogram best

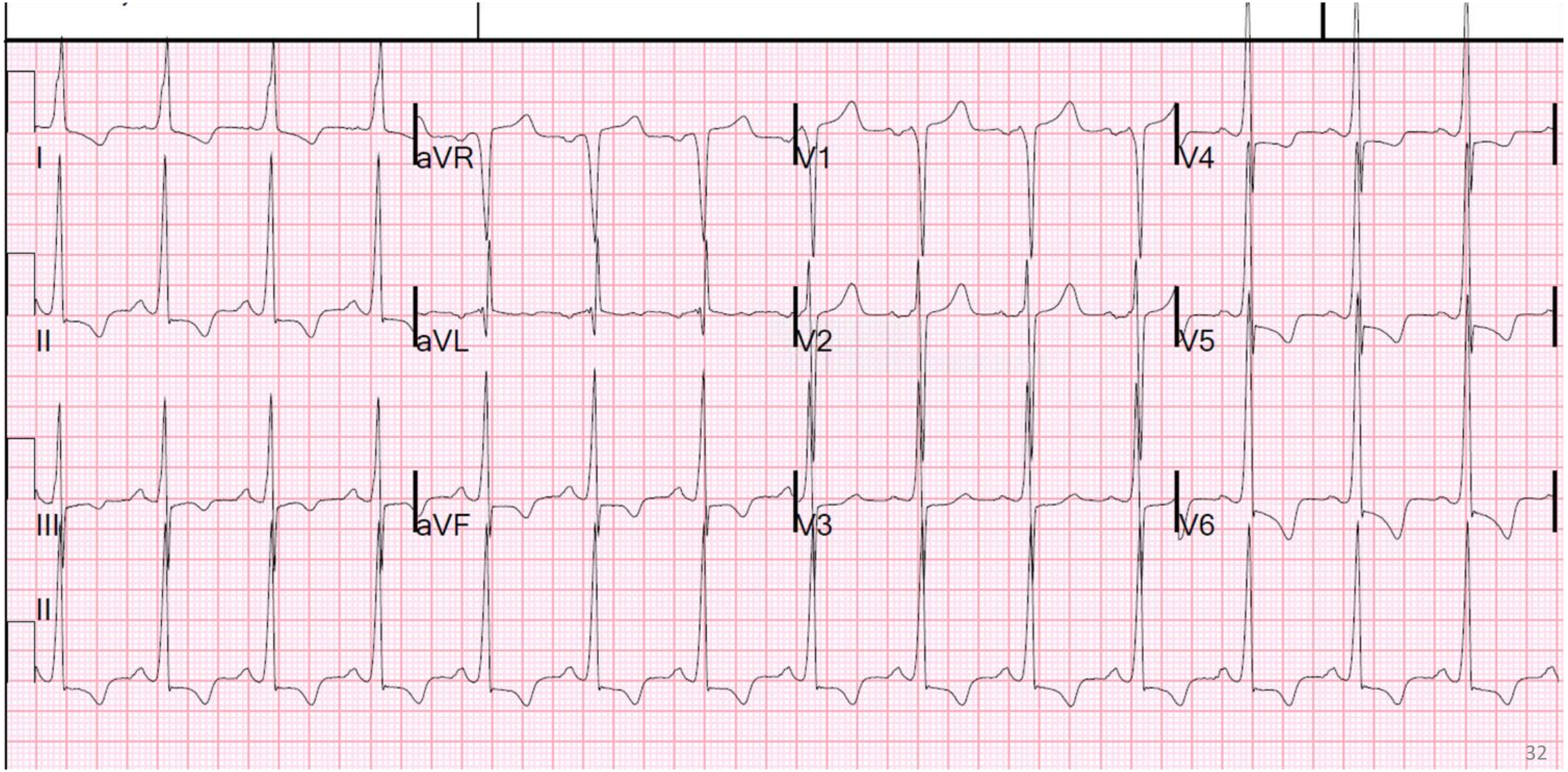
QT Interval Nomogram for determining 'at risk' QT–HR pairs from a single 12-lead ECG (modified from Figure 1 of reference 7).



Treatment TDP

- If unstable
  - DCR
- Mg
- Overdrive pacing
  - Isoprenaline
  - Pacing
    - External
    - Internal
- Treat cause

Chest Pain, now resolved



LVH - Criteria

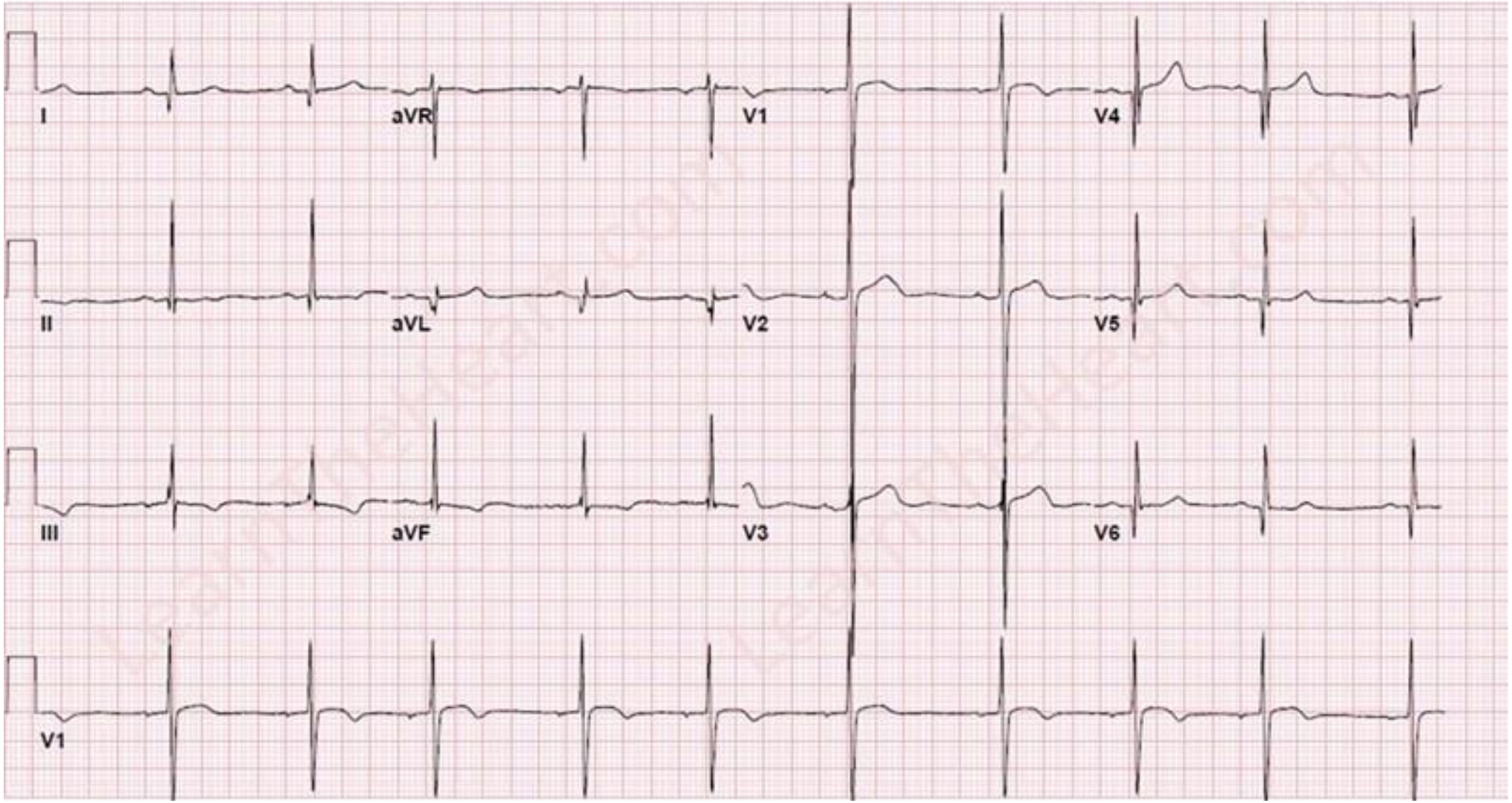
- Limb
  - R wave in lead I + S wave in lead III > 25 mm
  - R wave in aVL > 11 mm
  - R wave in aVF > 20 mm
  - S wave in aVR > 14 mm

- Praecordial
  - R wave in V4, V5 or V6 > 26 mm
  - R wave in V5 or V6 plus S wave in V1 > 35 mm
  - Largest R wave plus largest S wave in precordial leads > 45 mm

- Also get
  - Left atrial enlargement.
  - Left axis deviation.
  - ST elevation V1-3
  - Prominent U waves

- Causes
  - Hypertension
  - Aortic stenosis
  - Aortic regurgitation
  - Mitral regurgitation
  - Coarctation of the aorta
  - Hypertrophic cardiomyopathy

Syncope

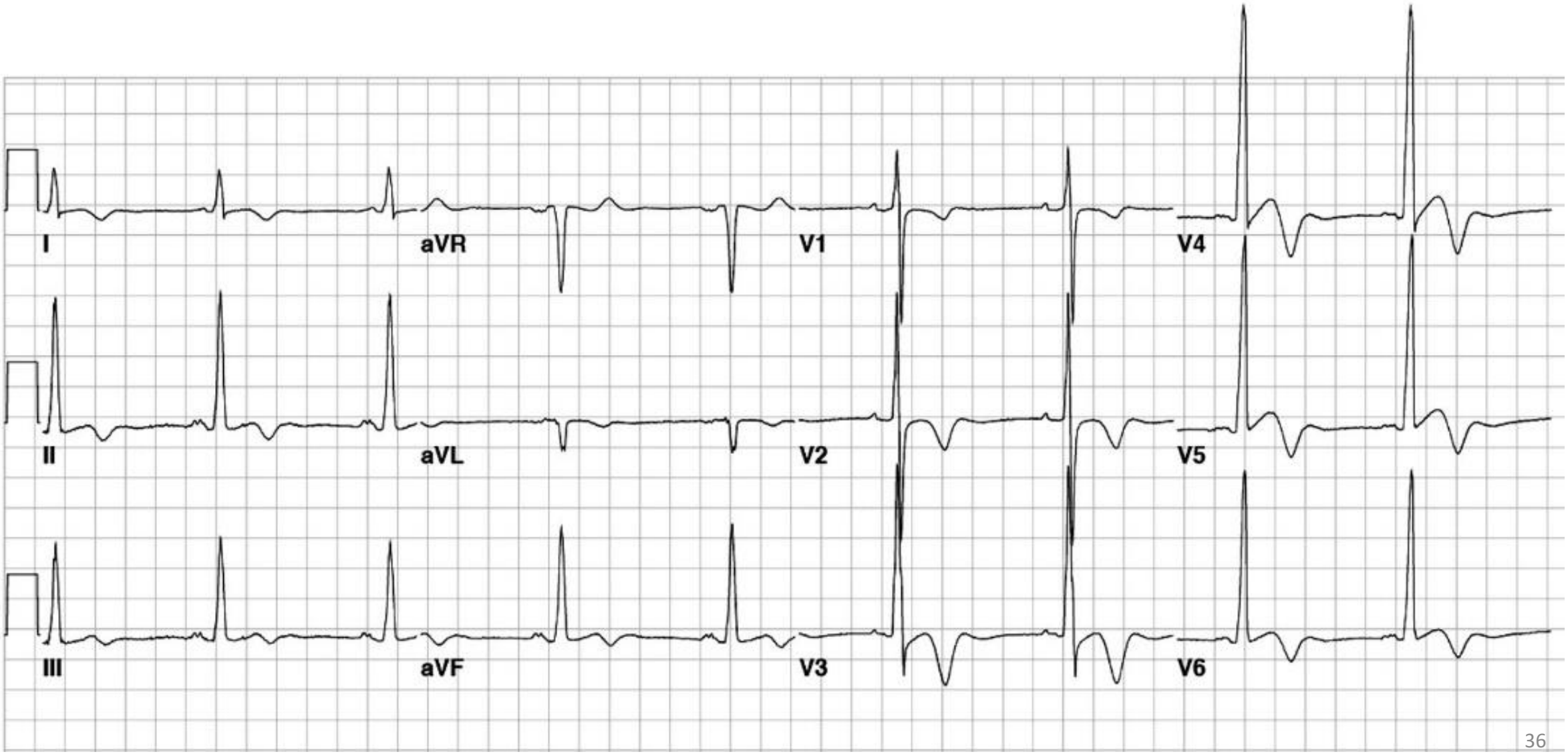


HCM

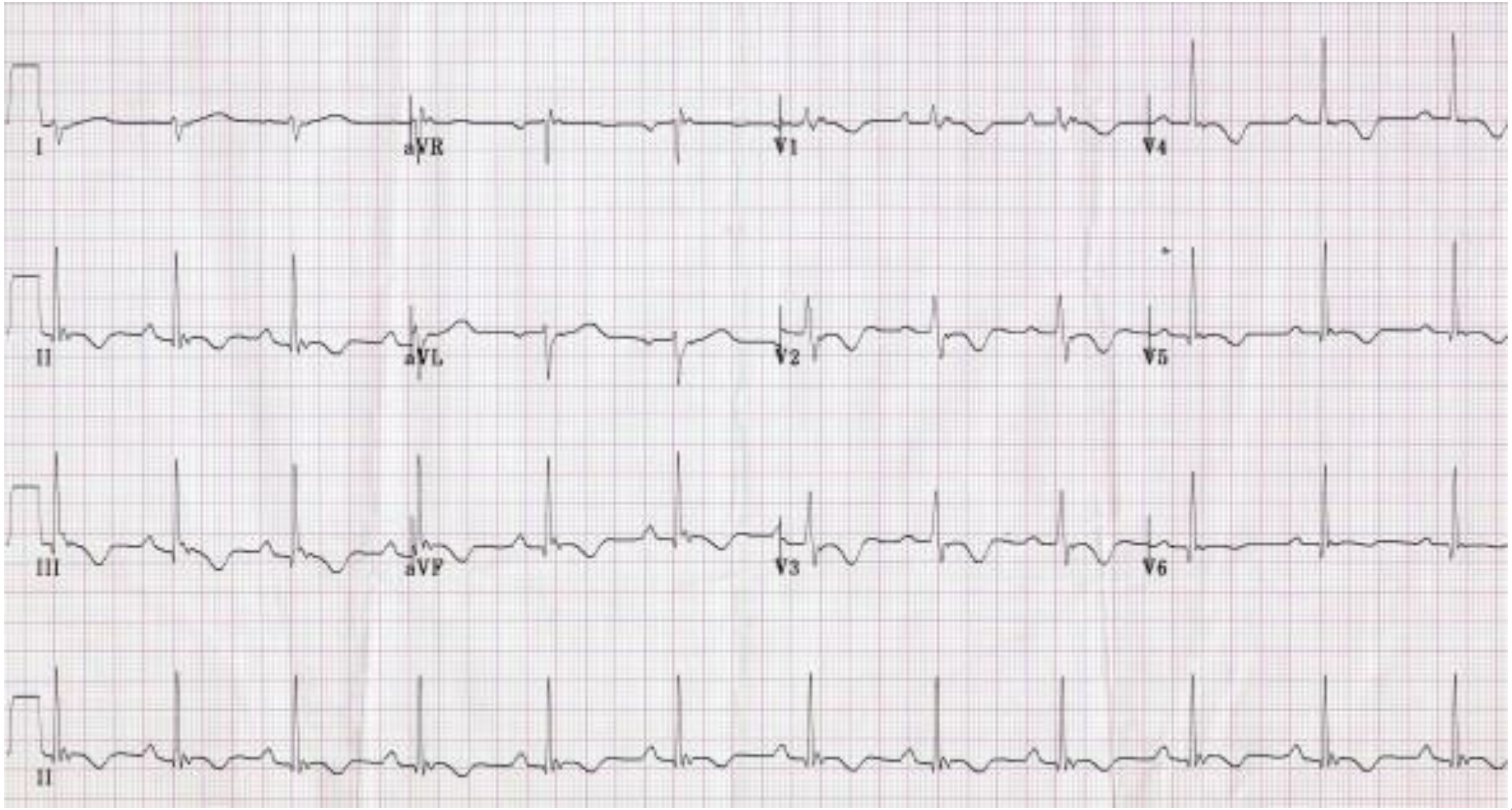
- Clinical findings
  - Number one cause of sudden cardiac death in young athletes
  - Inheritance is primarily autosomal dominant
  - Left ventricular hypertrophy (LVH), occurring in the absence of any inciting stimulus such as hypertension or aortic stenosis
  - ASH > Concentric LVH > Apical LVH
  - ASH associated with LVOT obstruction
    - Minority of cases
  - Arrythmogenic

- ECG findings
  - Left atrial enlargement
  - Left ventricular hypertrophy with associated ST segment / T-wave abnormalities
  - Deep, narrow (“dagger-like”) Q waves in the lateral > inferior leads
  - Giant precordial T-wave inversions in apical HCM
  - Signs of WPW (short PR, delta wave).
  - Dysrhythmias: atrial fibrillation, supraventricular tachycardias, PACs, PVCs, VT

Apical HCM (Yamaguchi)



Syncope

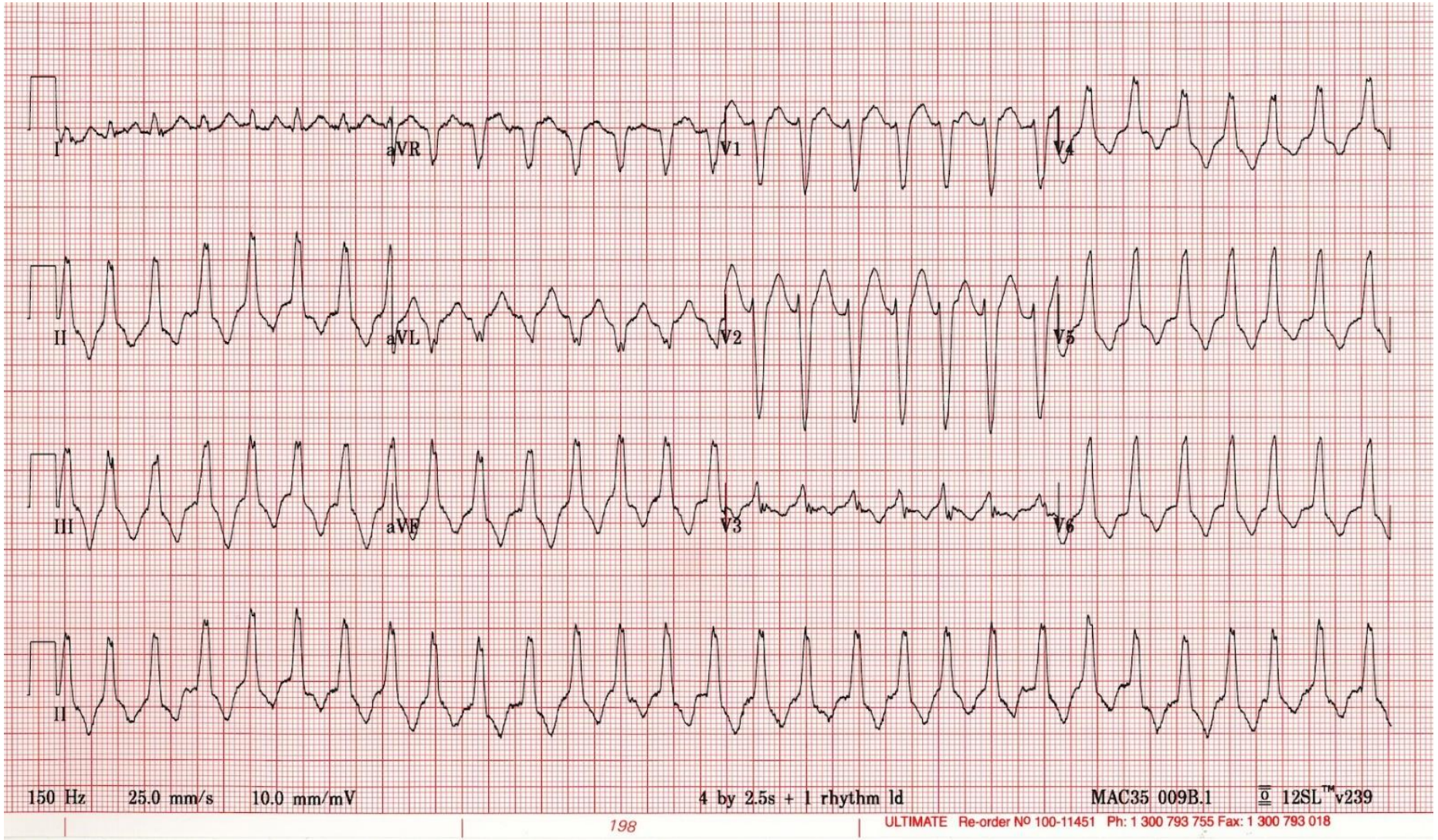


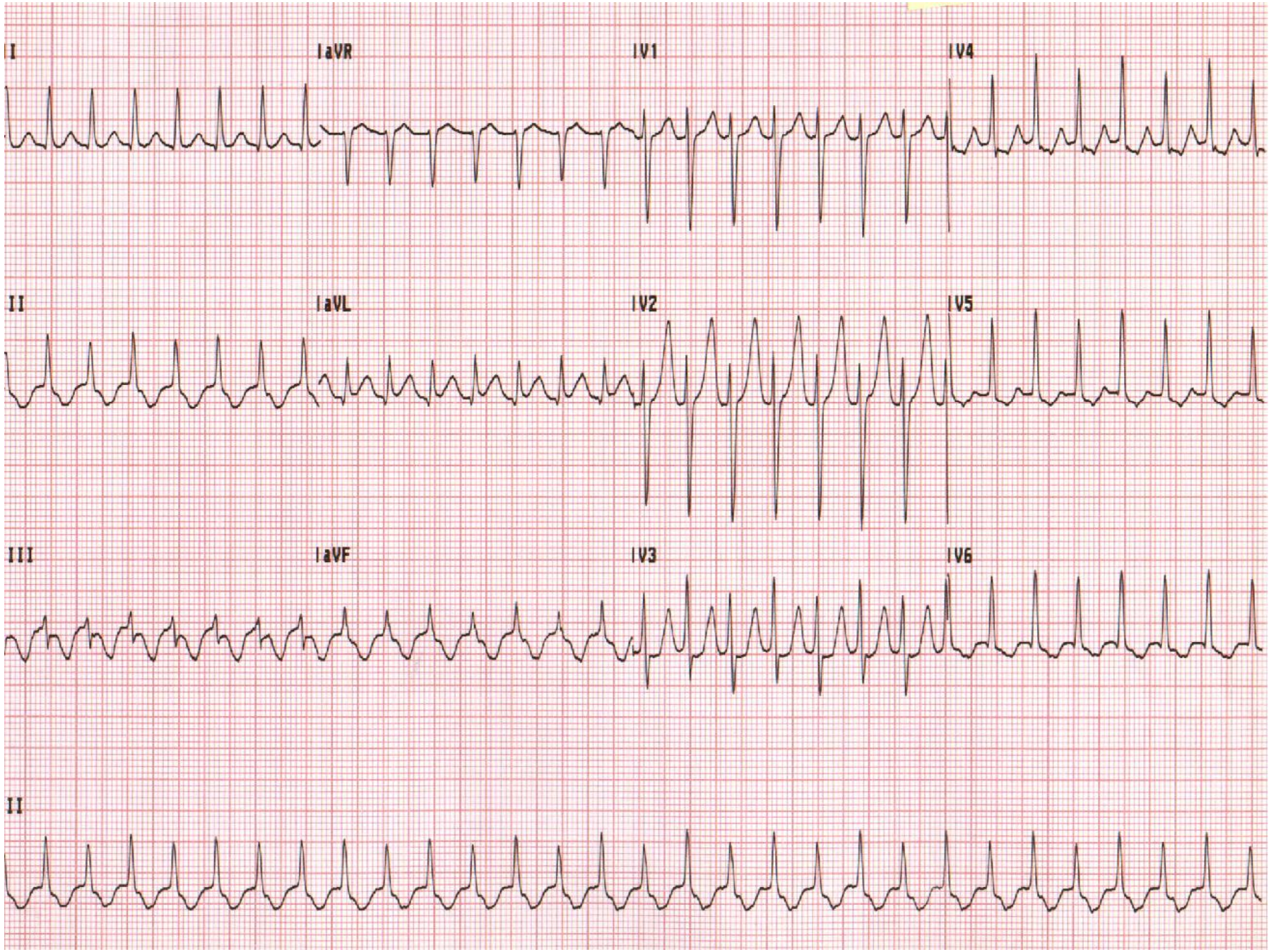
ARVD [arrhythmogenic right ventricular dysplasia]

- Clinical
  - Fibro-fatty replacement of the right ventricular myocardium.
  - second most common cause of sudden cardiac death in young people
  - Typically inherited as autosomal dominant
  - Greek Italian

- ECG
  - Epsilon wave (most specific finding, seen in 30% of patients)
  - T wave inversions in V1-3 (85% of patients)
  - Prolonged S-wave upstroke of 55ms in V1-3 (95% of patients)
  - Localised QRS widening of 110ms in V1-3
  - Paroxysmal episodes of ventricular tachycardia with a LBBB morphology

RV VT


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
150 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 1 rhythm ld
MAC35 009B.1
12SL™v239
198
ULTIMATE Re-order Nº 100-11451 Ph: 1 300 793 755 Fax: 1 300 793 018


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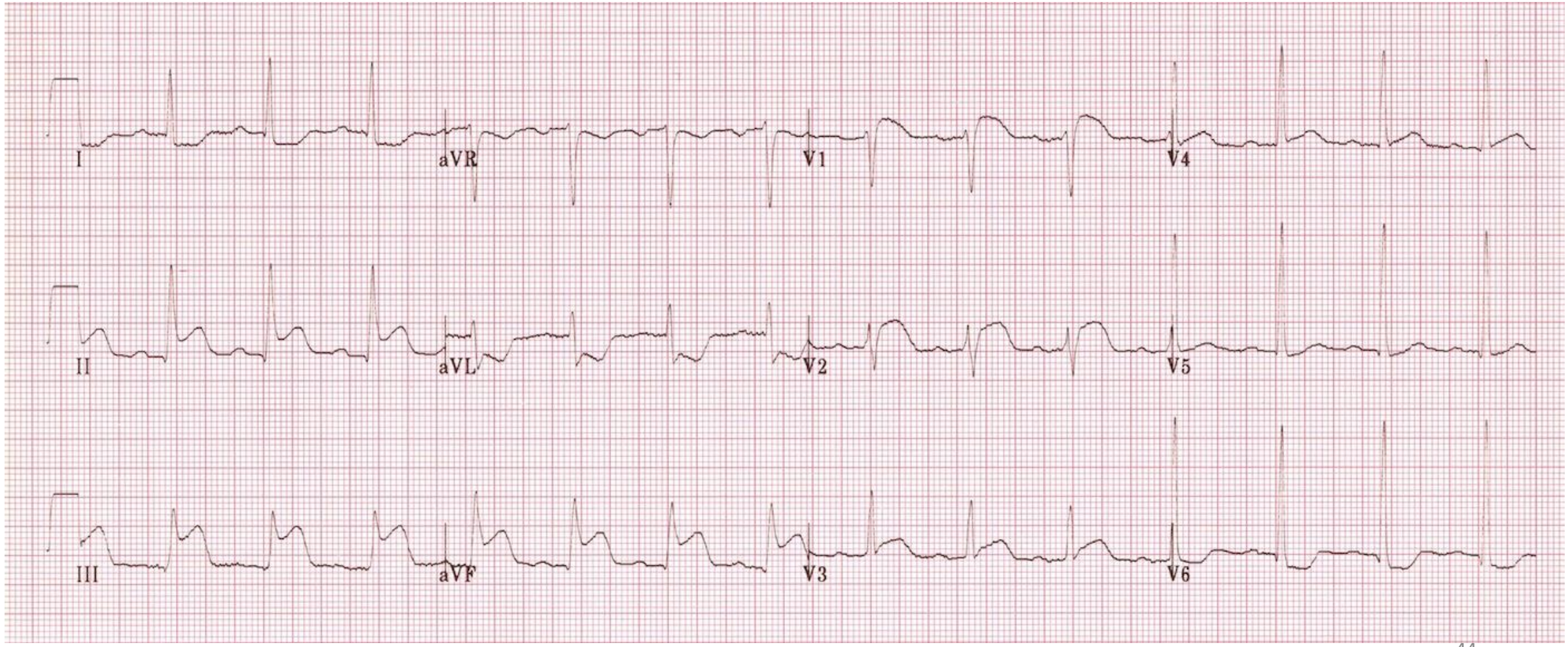
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

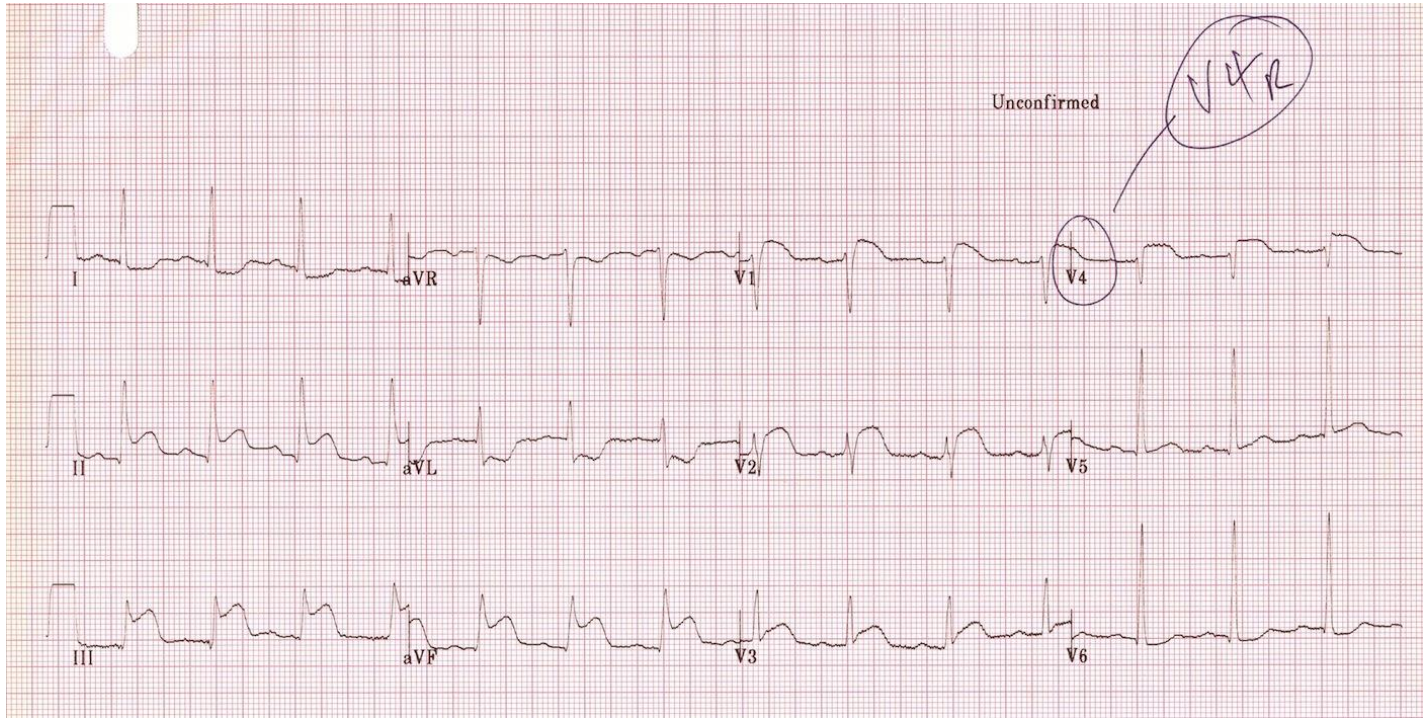
Paroxysmal SVT

- Most common cause is AVNRT

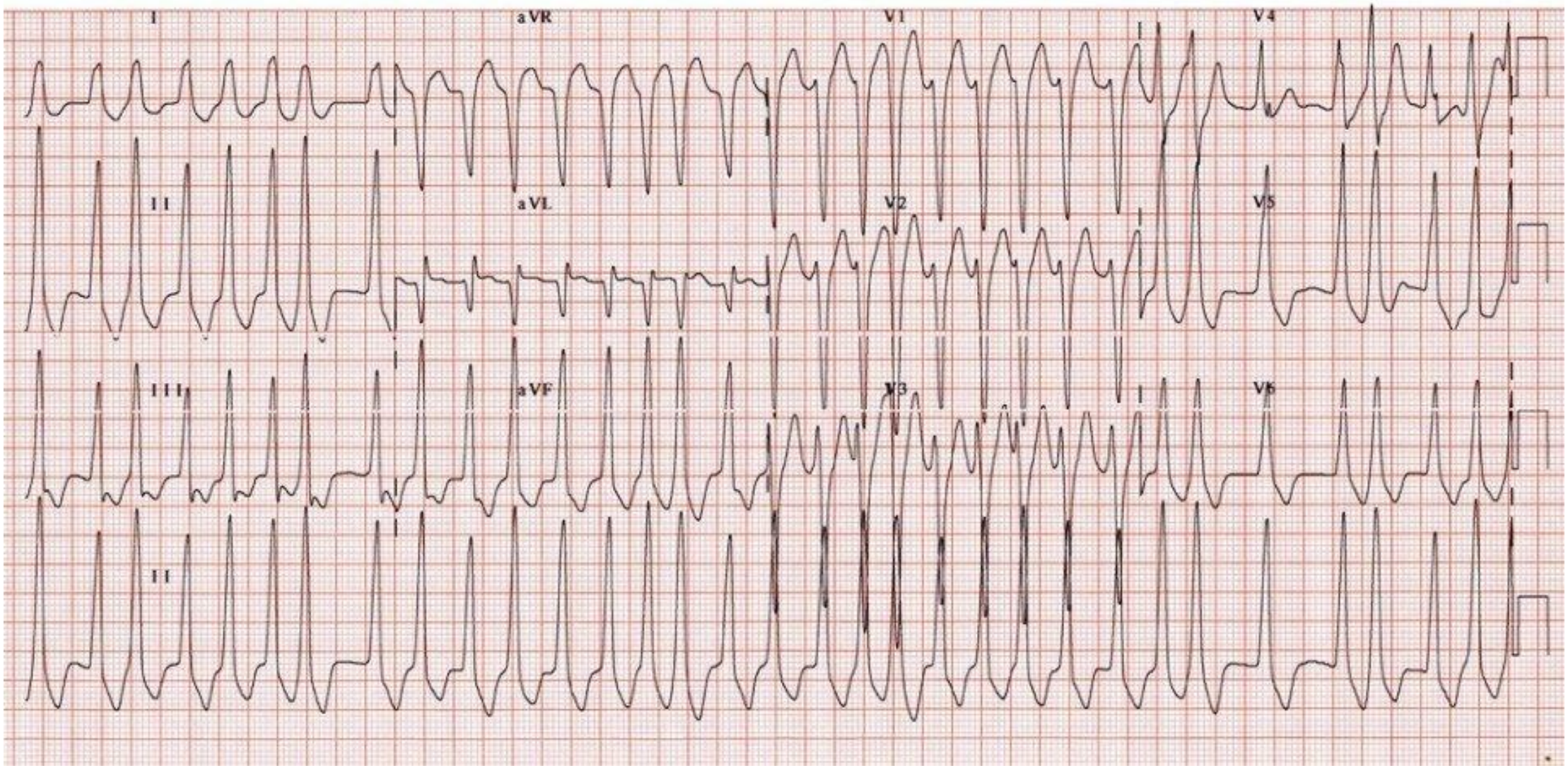
- Rapid
- Narrow complex
- Treatment
  - Vagal manoeuvres [Modified Valsalva – REVERT]
  - Adenosine
  - Verapamil
  - DCR
    - 50J synchronised
    - 100J synchronised
  - Overdrive pacing

Middle-aged patient presenting with chest pain and diaphoresis.
BP dropped to 80/50 following sublingual nitrates.





- **General:**
- Sinus rhythm, rate 84bpm.
- Normal axis.
- Borderline 1st degree AV block (PR 220ms).
- **Signs of** inferior STEMI**:**
- STE in inferior leads II, III, aVF.
- Reciprocal STD in lateral leads I, aVL, V6.
- **Signs of associated** right ventricular infarction**:**
- STE in III > II.
- STE in V1-2.
- This patient also had STE in V4R, confirming the diagnosis of RV infarction


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

Main Abnormalities:

- Irregularly irregular broad complex tachycardia.
- Extremely rapid ventricular rates — up to 300 bpm in places (RR intervals as short as 200ms or 1 large square).
- Beat-to-beat variability in the QRS morphology, with subtle variation in QRS width.

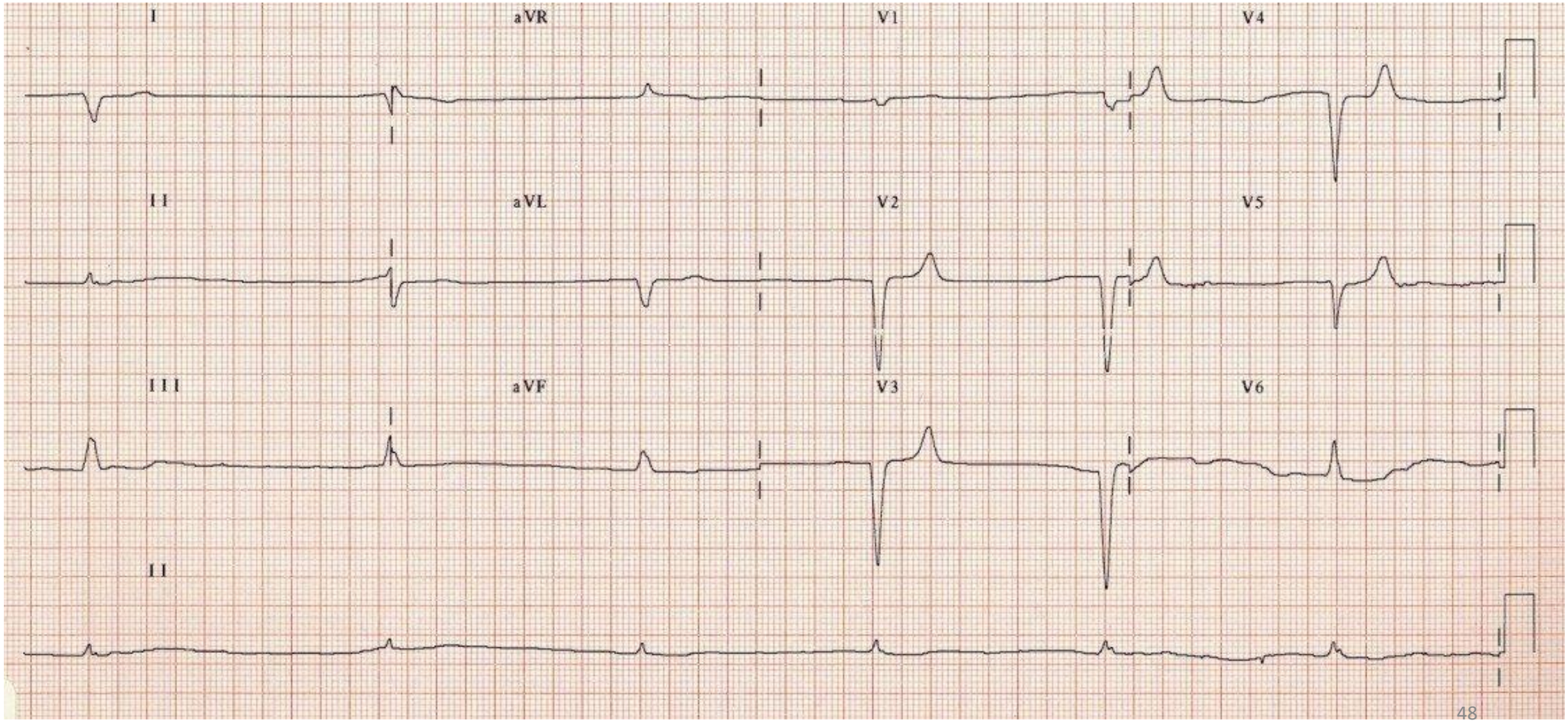
Explanation of ECG Findings:

- Irregularly irregular rhythm is consistent with atrial fibrillation.
- There is a left bundle branch block morphology to the QRS complexes.
- However, the ventricular rate is far too rapid for this to be simply AF with LBBB.
- The rates of 250-300 bpm and the variability in QRS complex morphology indicate the existence of an accessory pathway between the atria and ventricles.

Diagnosis:

- These findings indicate atrial fibrillation in the context of Wolff-Parkinson-White syndrome.

Middle-aged diabetic patient presenting with shortness of breath. Clinical evidence of pulmonary oedema. Describe the ECG



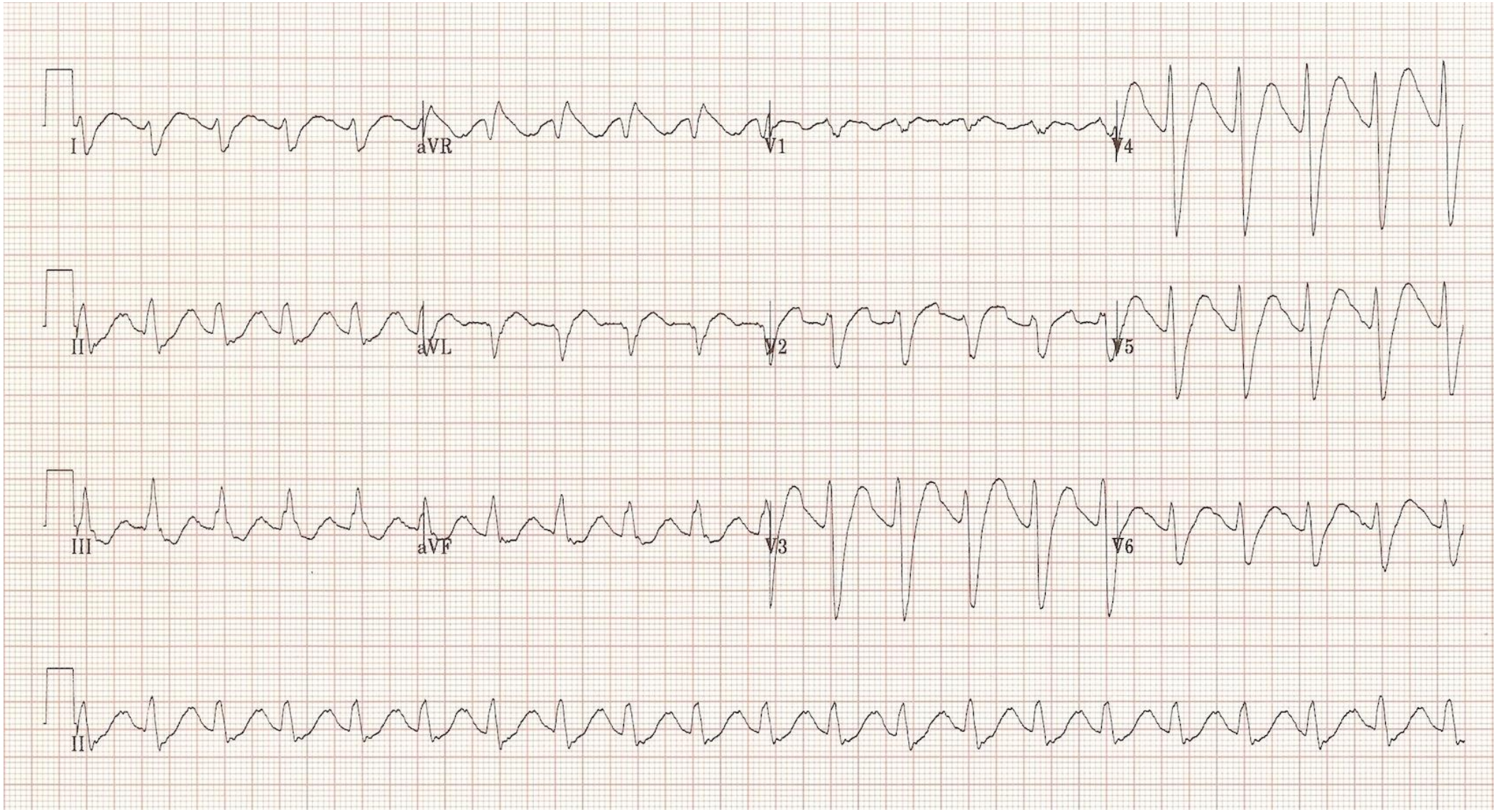
Main Abnormal Findings

- Severe bradycardia of 36 bpm.
- Rhythm is difficult to ascertain — appears irregular (?slow AF) although there are some small-voltage P waves seen in V1-2.
- Broad QRS complexes with an atypical LBBB morphology.
- Subtle symmetrical peaking ("tenting") of the T waves in V2-5.

Diagnosis

- The combination of bradycardia, flattening and loss of P waves, QRS broadening and T wave abnormalities is highly suspicious for severe hyperkalaemia. This patient had a potassium of 8.0 in the context of anuric renal failure.

20 year old male presenting with seizures. BP 80/50. Describe the ECG.



Main Abnormalities

- Broad complex tachycardia, rate ~ 130 bpm.
- The rhythm is likely sinus tachycardia with a 1st degree AV block — note the "camel hump" appearance to the T waves indicating a hidden P wave.
- Interventricular conduction delay (QRS duration > 100ms, not typical LBBB / RBBB morphology)
- Right axis deviation.
- Secondary R' wave in aVR > 3 mm.

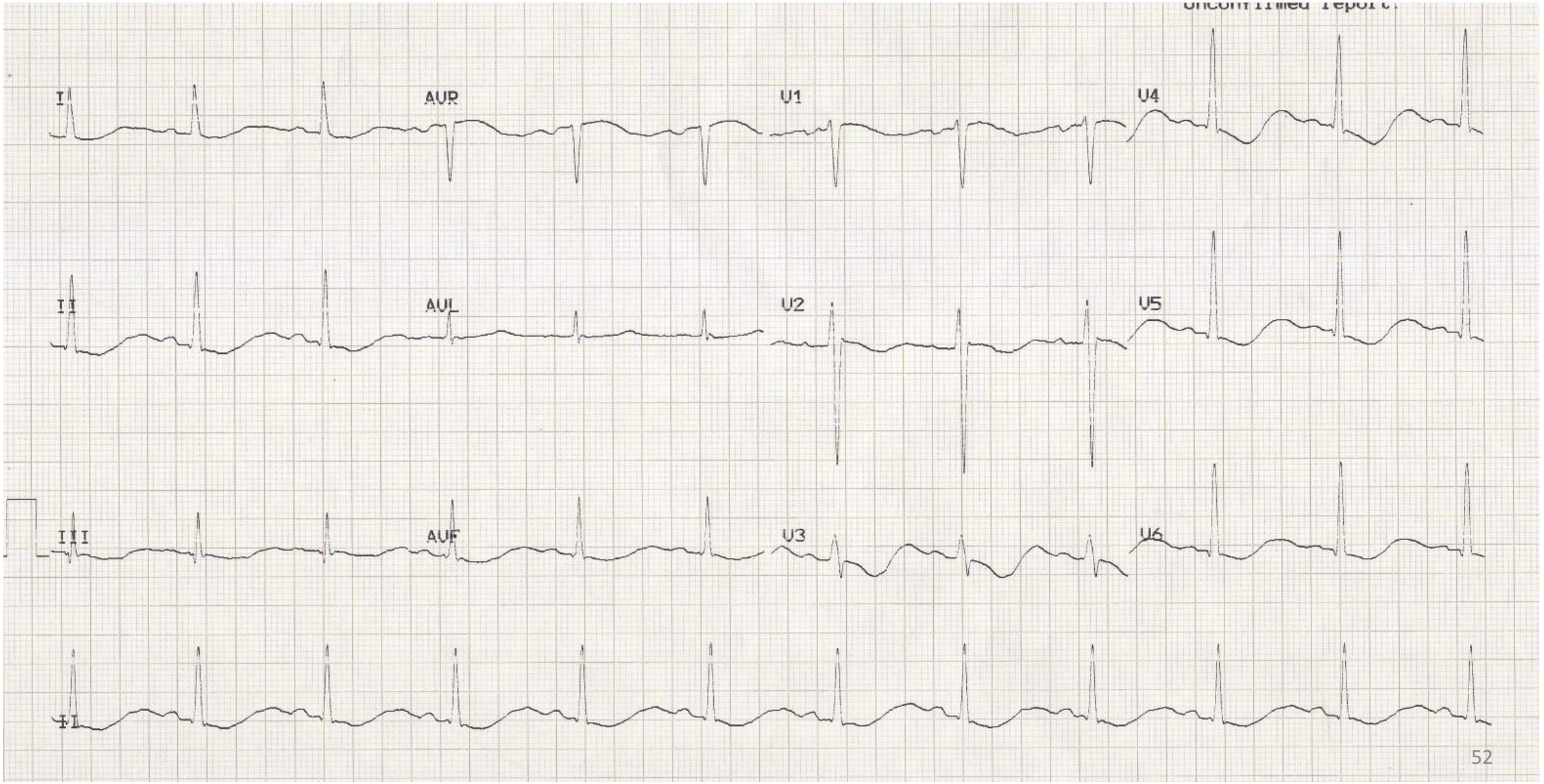
Diagnosis

- In the context of a patient presenting with seizures and hypotension, the combination of…
- QRS broadening > 100 ms
- R' wave in aVR > 3 mm
- … is highly suggestive of poisoning with a sodium-channel blocking agent — e.g. tricyclic antidepressant.
- The sinus tachycardia may be due to the anticholinergic effects of the TCA.

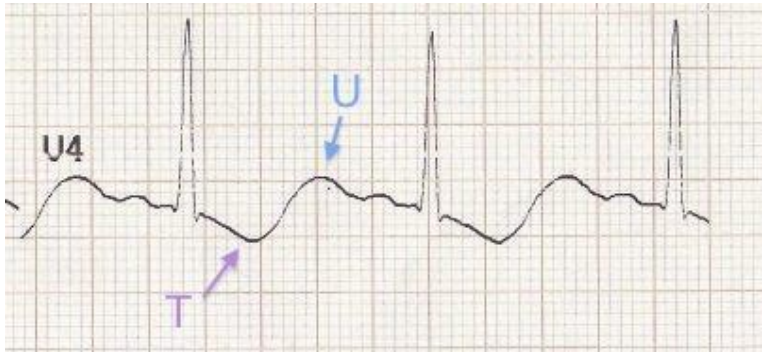
Clinical Pearls

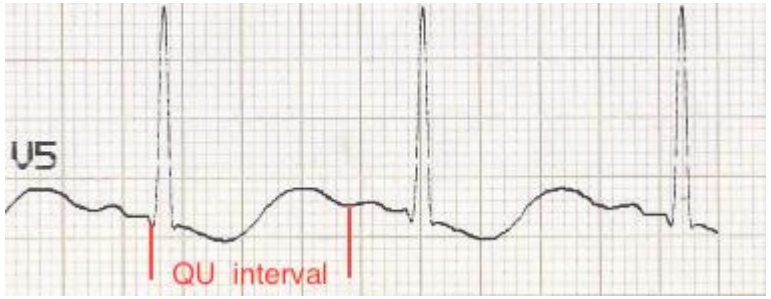
- In the context of sodium channel blockade:
- A QRS duration > 100 ms is predictive of seizures.
- A QRS duration > 160 ms is predictive of cardiotoxicity.
- This patient is already manifesting life-threatening toxicity and needs aggressive resuscitation, including:
- Serum alkalinisation with NaHCO3 to reverse pH-dependent toxicity.
- Intubation and hyperventilation aiming for alkaline arterial pH (e.g. 7.45 to 7.55).
- Seizure management with benzodiazepines.
- BP management with fluid boluses +/- pressors.

30-year old patient presenting with generalised weakness. Describe and interpret the ECG.



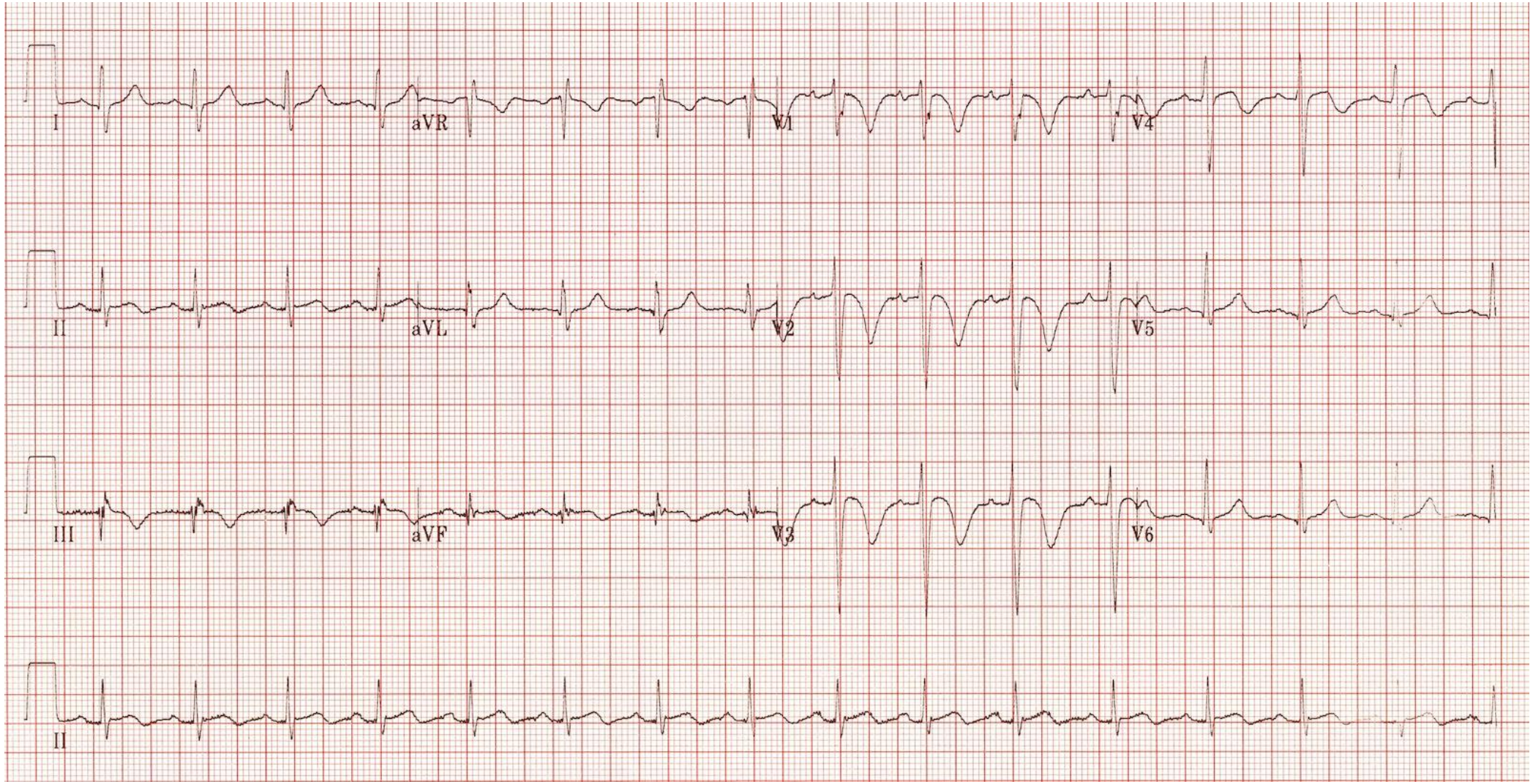
Main Abnormalities
•The ECG shows widespread ST segment abnormalities.
•There is a biphasic appearance to the ST segments and T waves, with initial negative deflection (= ST segment depression / T wave inversion) followed by a terminal positive deflection (= U wave).
•All these waves merge into each other and it is difficult to tell where one wave ends and the other begins.
•There is gross prolongation of the QU interval (= time from onset of QRS complex to end of T/U wave).





Diagnosis
The combination of…
•Widespread ST depression / T wave inversion
•Prominent U waves
•Long QU interval (> 500 ms)
…. is highly suggestive of severe hypokalaemia.
This patient had a serum K of 1.7 mmol/L in the context of decompensated Conn's syndrome (primary aldosteronism).

70-year old patient presenting with chest pain, dyspnoea and dizziness. BP 90/50. SaO2 83% RA. Describe the ECG.



Main Abnormalities

•Sinus tachycardia ~ 100 bpm.
•Anterior T wave abnormalities: inverted in V1-3, biphasic in V4.
•Inferior T wave abnormalities: biphasic in III, aVF.
•Subtle ST elevation in III and aVF.

Significance of ECG Findings

This pattern of T wave inversions in the right precordial leads V1-4 plus the inferior leads (especially the rightward-facing lead III) is referred to as the right ventricular strain pattern. It is a marker of right ventricular hypertrophy or dilatation.

Diagnosis

In a patient presenting with acute shortness of breath, the combination of…

•Sinus tachycardia
•RV strain pattern in V1-4 (+/- lead III)

… is highly suggestive of acute cor pulmonale due to massive pulmonary embolism.

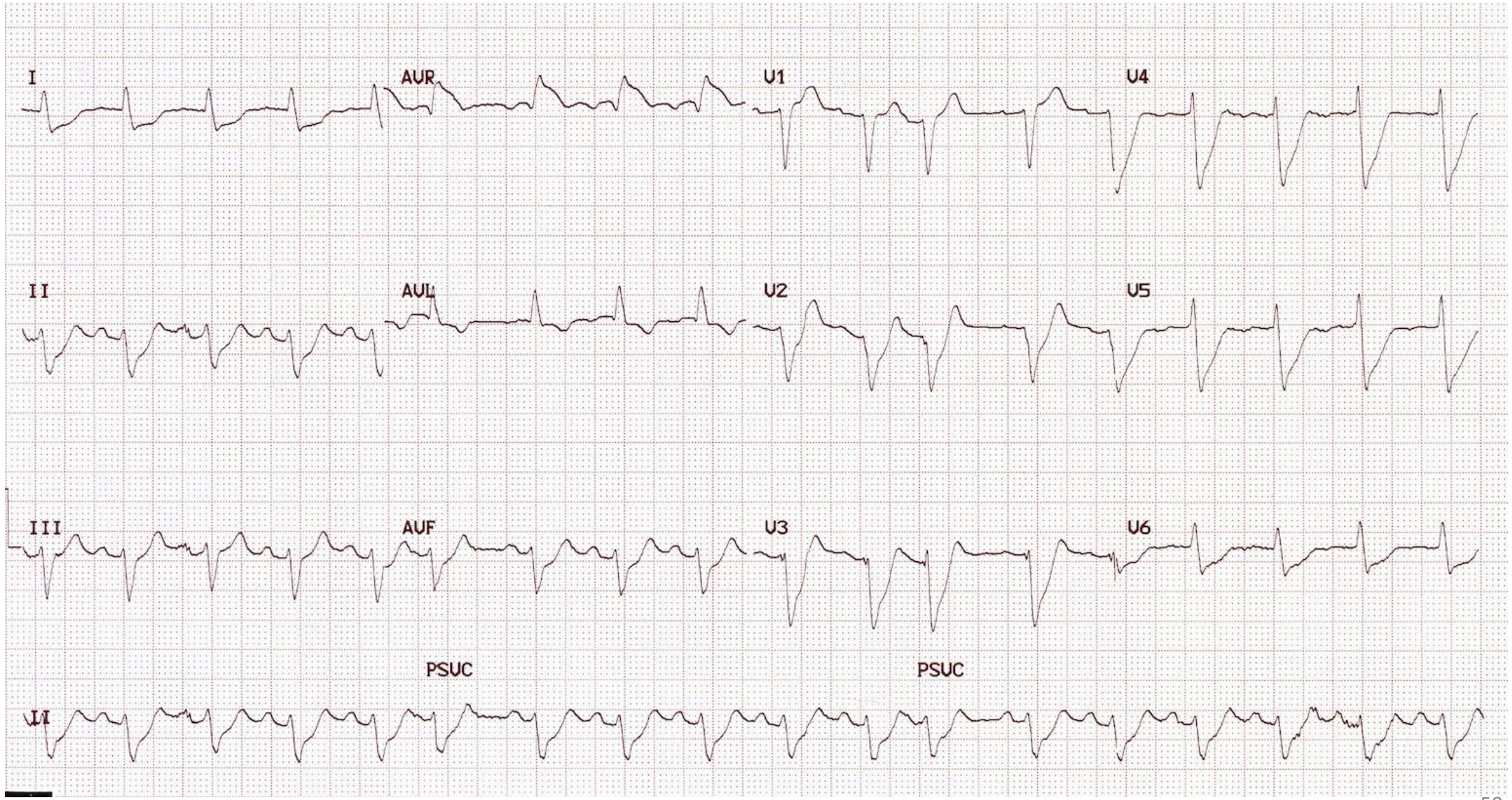
However, these ECG changes are not specific to PE and may be seen in other conditions associated with pulmonary hypertension and RV enlargement including:

•Chronic lung disease (COPD, lung fibrosis) with chronic cor pulmonale
•Right ventricular hypertropy — e.g. due to congenital causes, valvular heart disease
•Arrhythmogenic right ventricular cardiomyopathy

Clinical Pearls

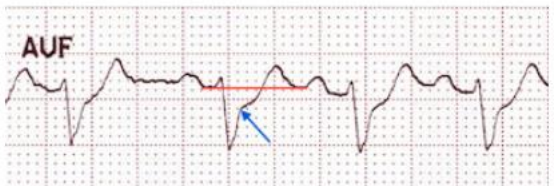
- Other ECG findings associated with pulmonary embolism include:
- New right axis deviation
- New right bundle branch block
- New dominant R wave in V1
- Non-specific ST segment changes
- The oft-quoted S_I Q_{III} T_{III} pattern (deep S wave in lead I, Q wave in III, inverted T wave in III) is neither sensitive nor specific for PE and is infrequently seen (20% of cases).
- Similarly, sinus tachycardia is not as ubiquitous in PE as people seem to think (< 50% of cases), and certainly should not be relied up to exclude PE.

70-year old patient presenting with severe chest pain, diaphoresis and syncope. BP 65/40.

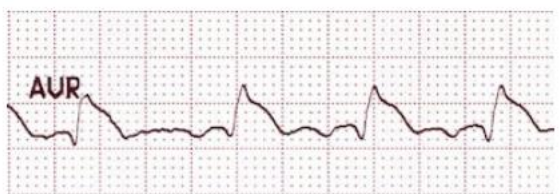


Main Abnormalities

- Widespread ST depression affecting multiple precordial (V2-6) and limb leads (esp. I, II, avF).
- To some extent this is masked by an indistinct J point, upsloping (rather than horizontal) ST depression and some baseline wander of the ECG.
- There is~3 mm ST elevation in aVR.



ST depression in aVF relative to the T-P baseline. The blue arrow denotes the approximate position of the J point.



ST elevation in aVR

Diagnosis

In the context of ischaemic chest pain and cardiogenic shock, the combination of…

- Widespread ST depression
- ST elevation in aVR > 1 mm
- ST elevation in aVR > V1

… is extremely concerning for left main coronary artery occlusion.

However, this pattern is not entirely specific for LMCA occlusion. It may be seen whenever there is diffuse severe subendocardial ischaemia, e.g.

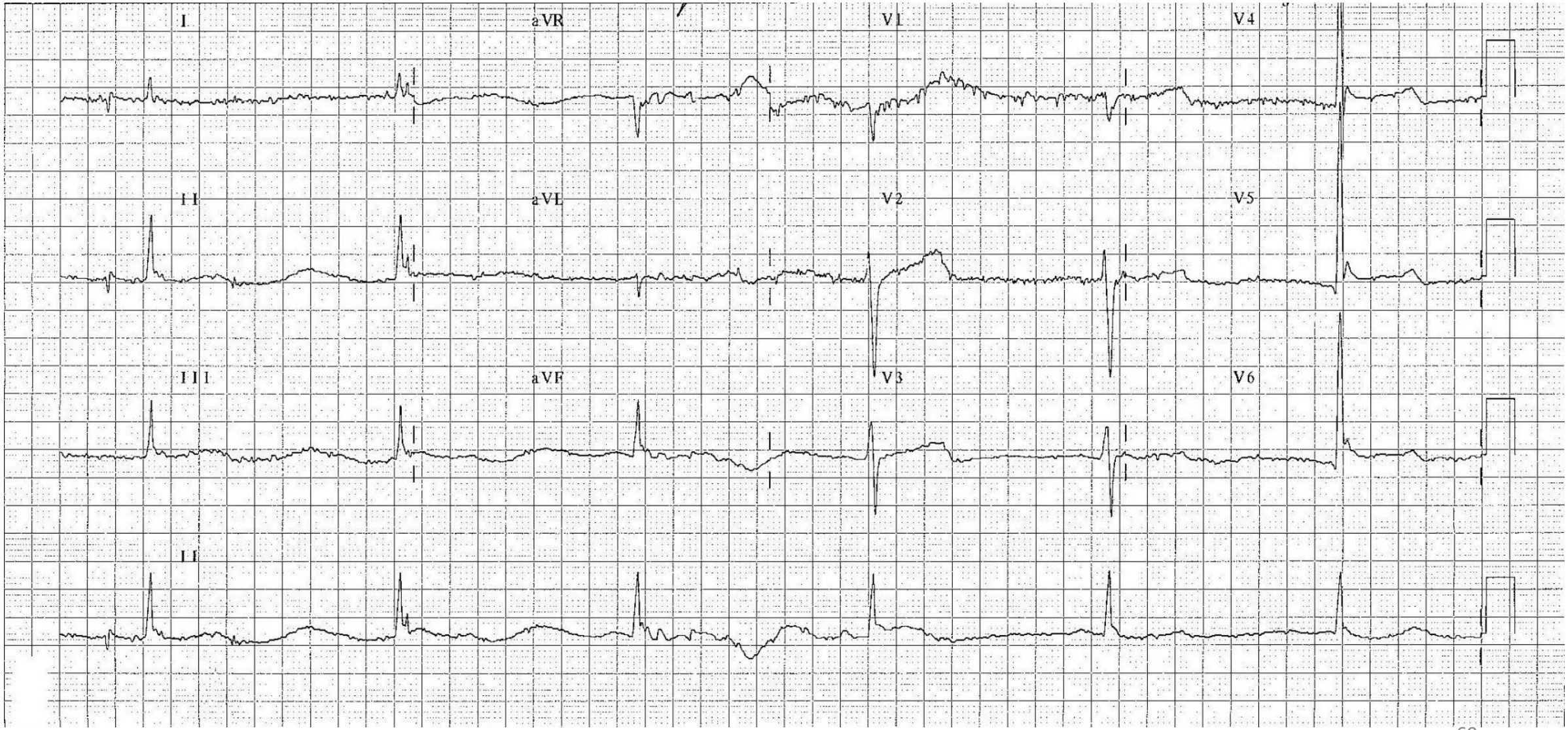
- Severe triple vessel disease
- Severe anaemia or hypoxaemia
- Following resuscitation from cardiac arrest

This patient developed progressive cardiogenic shock complicated by runs of ventricular tachycardia. He was taken for immediate angiography where he was found to have a complete ostial occlusion of his left main coronary artery.

Pitfalls

A similar ECG pattern of diffuse ST depression with ST elevation in aVR may also be seen with supraventricular tachycardias (AVNRT / atrial flutter). This **rate-related change** is usually benign and resolves with resolution of the SVT.

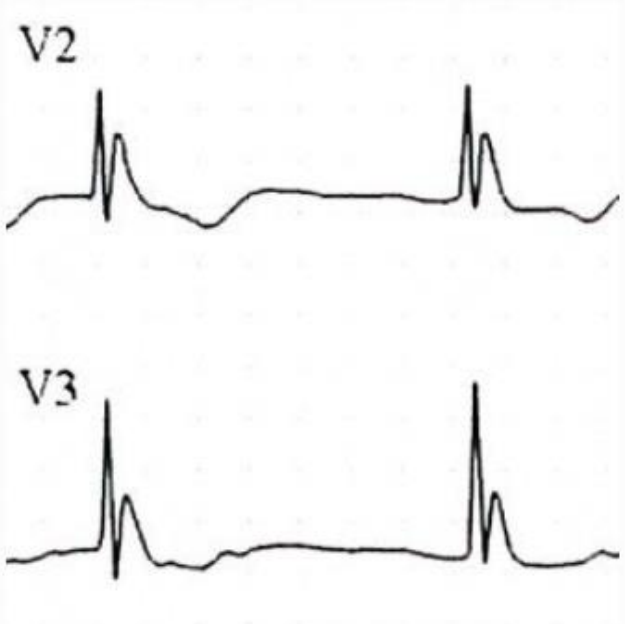
90-year old patient found on the floor at home. Describe what his ECG shows.



This ECG demonstrates all the classic features of hypothermia:

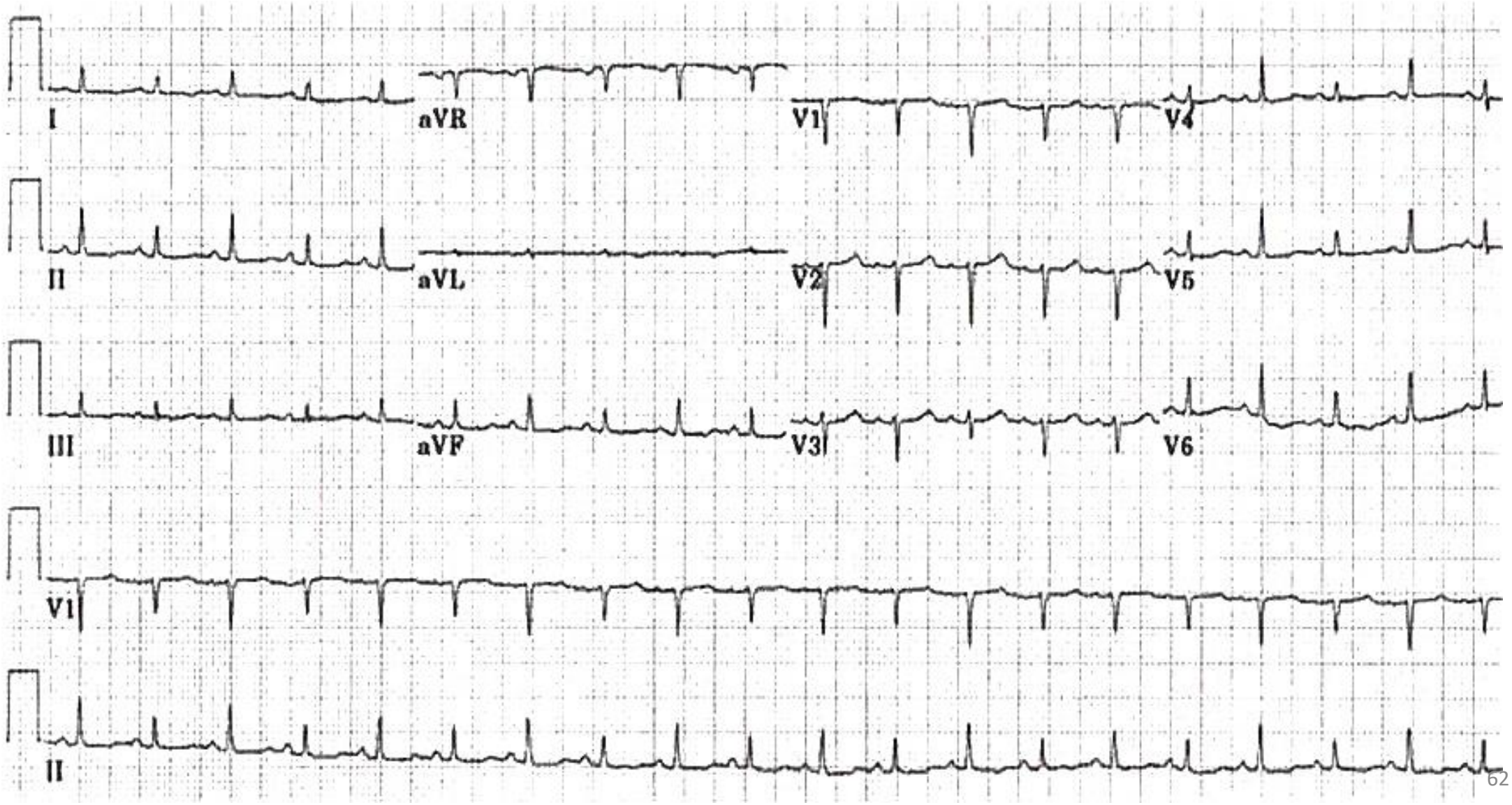
- Bradycardia
- Osborne waves (J waves) = notching at the J point seen in V4-6
- Long QT interval (~ 600 ms)
- Shivering artefact

The rhythm is probably sinus bradycardia — mapping out the RR intervals reveals a regular rhythm despite the obliteration of the baseline by the shiver artefact.



Typical Osborne waves (J waves)

Middle aged female presenting with dyspnoea. Previous mastectomy for breast carcinoma. What does the ECG show?



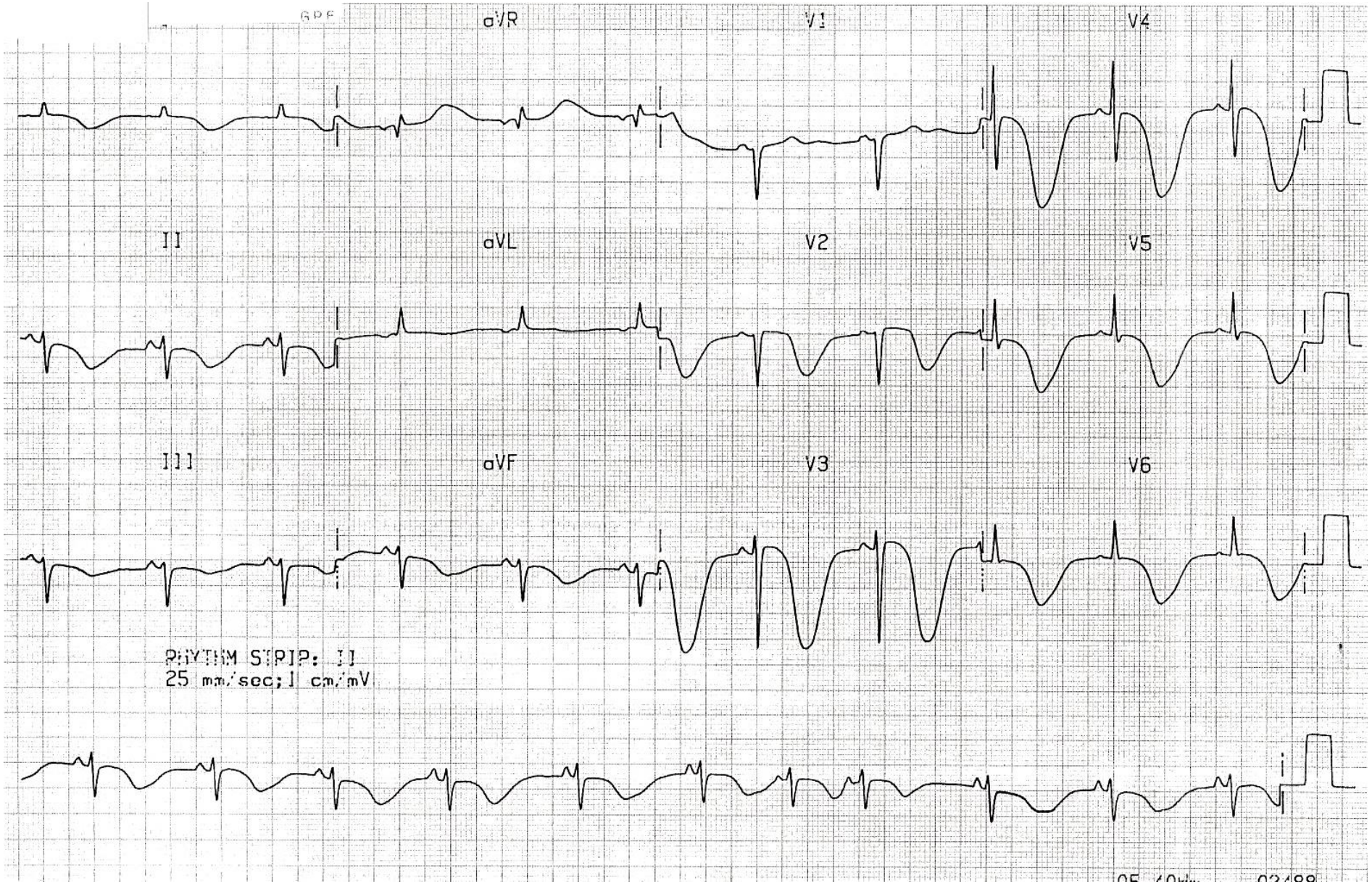
Main Abnormalities

- Sinus tachycardia
- Low QRS voltages — Multiple limb lead QRS complexes < 5 mm in amplitude.
- Electrical alternans — There is a beat-to-beat variation in the QRS complex height. Taller complexes alternate with shorter ones.

The triad of tachycardia, low QRS voltages and electrical alternans is extremely suspicious for massive pericardial effusion.

Given the clinical history, I would be concerned about the presence of a malignant pericardial effusion causing tamponade. The diagnosis can be rapidly confirmed on bedside echo (watch these videos from The Ultrasound Podcast to learn how: Part 1, Part 2). There may also be clinical evidence of pulsus paradoxus.

Young male found collapsed at home, apparently intoxicated. What does the ECG show?



Main Abnormalities

- Giant T-wave inversions in multiple leads, most prominent in V2-6
- Marked QT prolongation > 600 ms

Diagnosis

This ECG pattern is characteristic of raised intracranial pressure and is classically seen in the context of **massive intracranial haemorrhage**, particularly:

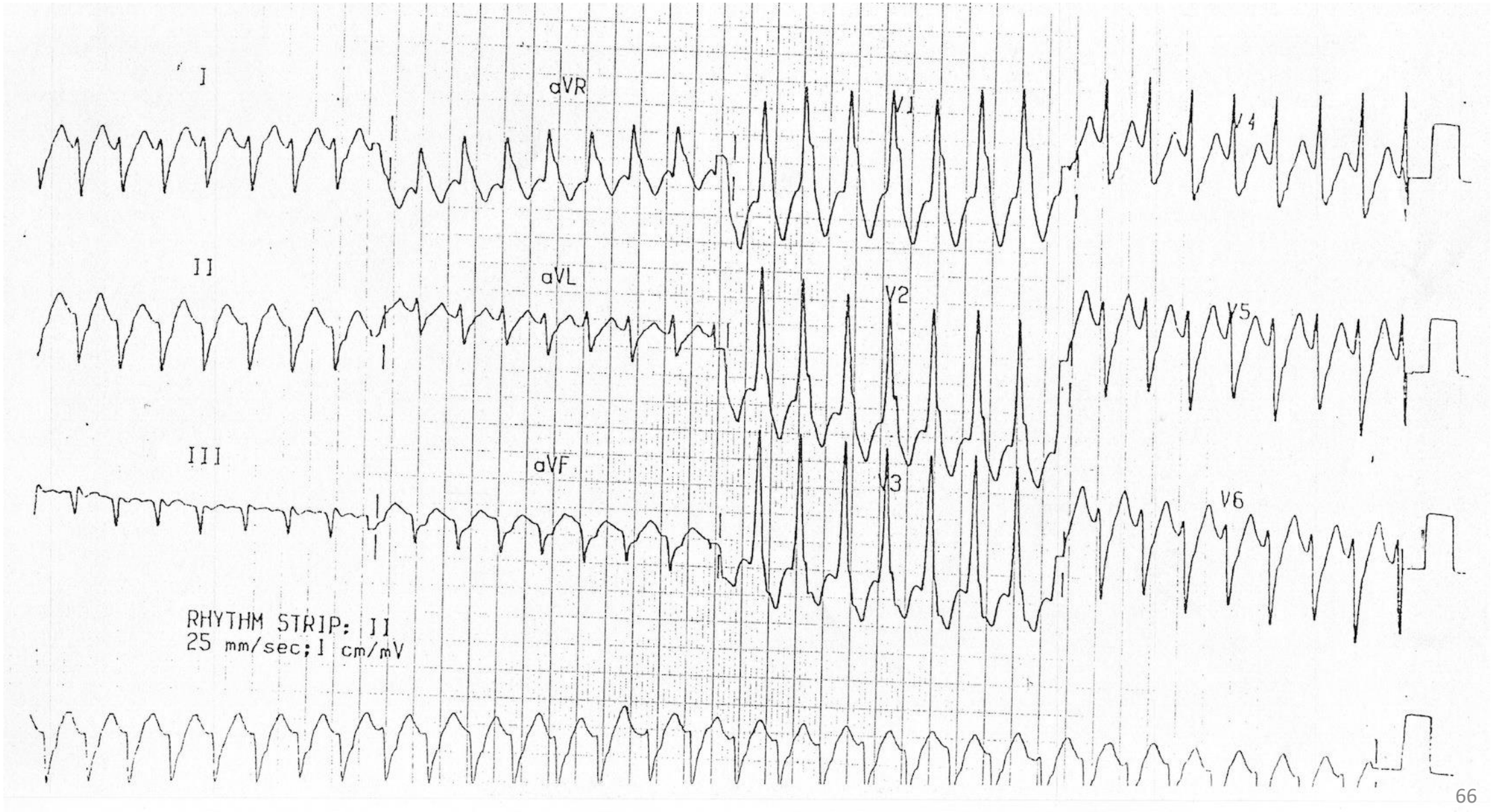
- Aneurysmal subarachnoid haemorrhage
- Haemorrhagic stroke

Similar ECG patterns have also been reported in patients with raised ICP due to:

- Large-territory ischaemic stroke causing cerebral oedema (e.g. MCA occlusion)
- Traumatic brain injury

The differential diagnosis for widespread T-wave inversions and QT prolongation includes myocardial ischaemia (e.g. Wellen's syndrome) and electrolyte abnormalities (e.g. hypokalemia). However, neither condition would cause the gigantic "cerebral T waves" seen here.

Middle-aged patient presenting with palpitations and dizziness. What does the ECG show?



This ECG shows a regular broad complex tachycardia with an RSR' pattern in V1.

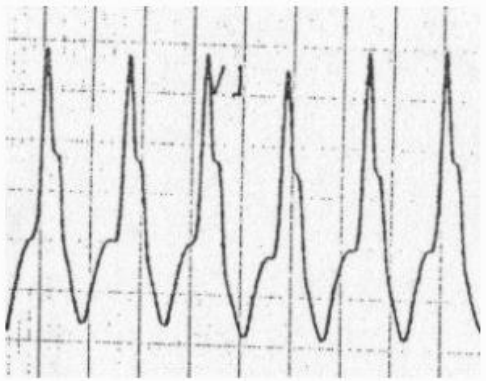
The differential diagnosis could include:

- Ventricular tachycardia.
- SVT with aberrant conduction — either due to RBBB or WPW.

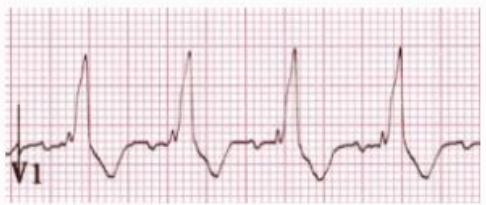
On closer inspection, the ECG demonstrates some classic features of **ventricular tachycardia**:

- Northwest axis — QRS is positive in aVR, negative in I and aVF.
- The **taller left rabbit ear** sign — There is an atypical RBBB pattern in V1, where the left "rabbit ear" is taller than the right.
- Negative QRS complex (R/S ratio < 1) in V6.

These findings indicate VT rather than SVT with aberrancy.



Taller left rabbit ear = VT



Taller right rabbit ear = RBBB

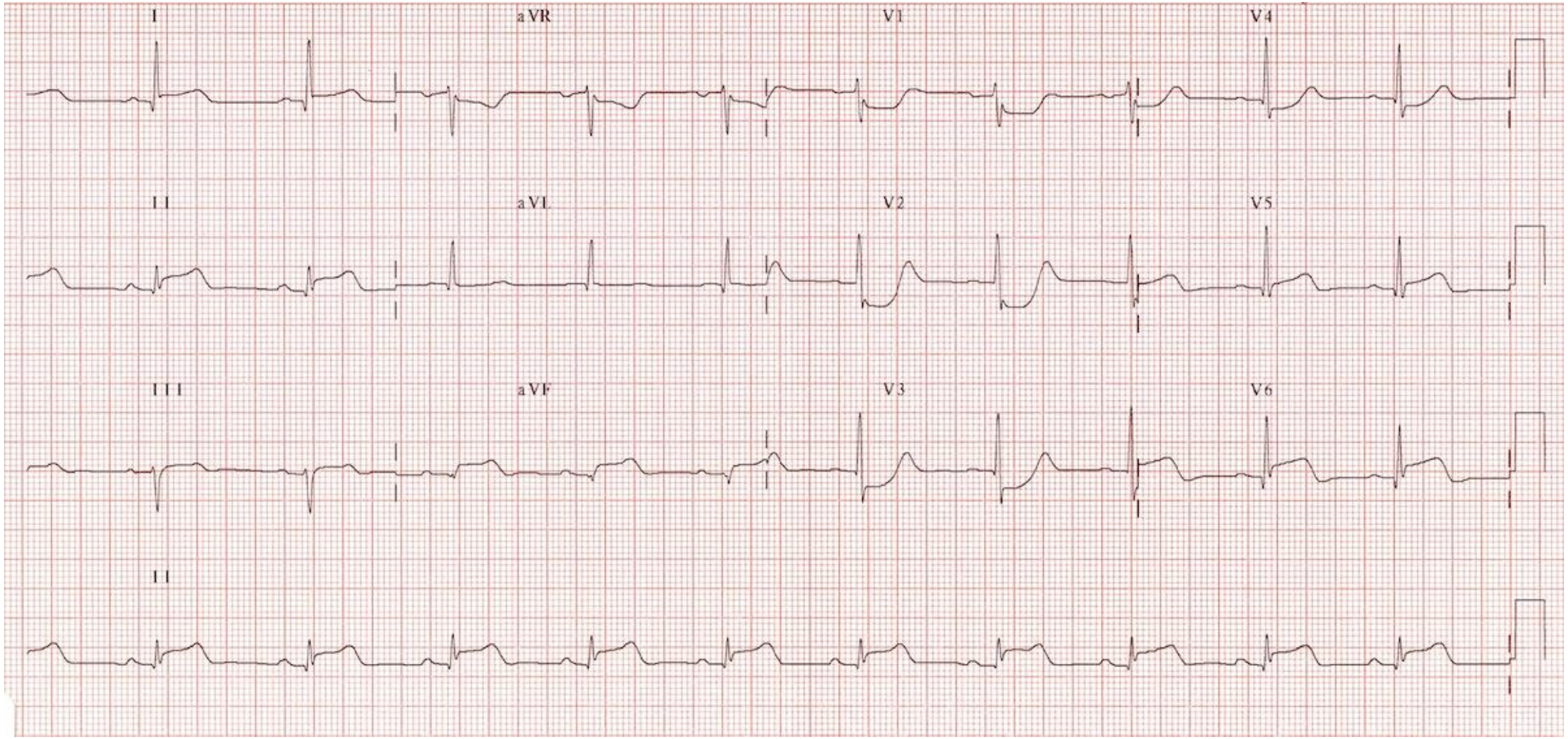
Clinical Pearls

Other factors that increase the likelihood of VT in patients presenting with regular broad complex tachycardia include:

- Age > 35 (positive predictive value of 85%).
- Structural heart disease — e.g. IHD, CCF, cardiomyopathy.
- Family history of sudden cardiac death or arrhythmogenic conditions such as HOCM, Brugada syndrome or ARVC that are associated with episodes of VT.

In any patient with a broad complex rhythm, also consider the possibility of toxic / metabolic conditions such as hyperkalaemia or sodium-channel blockade.

Middle aged patient presenting with central chest pain. What does the ECG show?



Evidence of inferolateral STEMI

- ST elevation in the inferior leads (II, III, aVF)
- ST elevation in the lateral leads (I, V5, V6)

Evidence of posterior STEMI

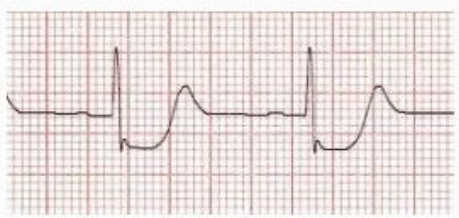
- Horizontal ST depression in V1-4 (maximal in V2-3)
- Dominant R wave in V2 (R/S ratio > 1)
- Upright T wave in V2

This pattern of infero-postero-lateral STEMI is most likely caused by occlusion of a dominant left circumflex artery.

Tips for spotting posterior infarction

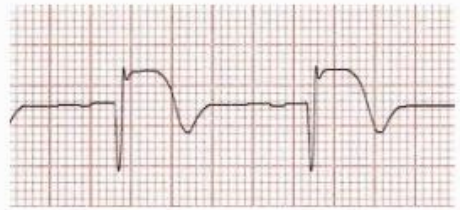
Look specifically at lead V2 for the combination of

- Horizontal ST depression.
- Tall, broad R wave (>30ms wide, R/S ratio > 1) — this is a Q-wave equivalent.
- Upright T wave — particularly the terminal portion of the T wave.



Typical appearance of posterior infarction in V2

One common trick is to turn the ECG over, hold it up to the light and look through it from behind. This inverts lead V2, which then takes on the appearance of a classic STEMI.

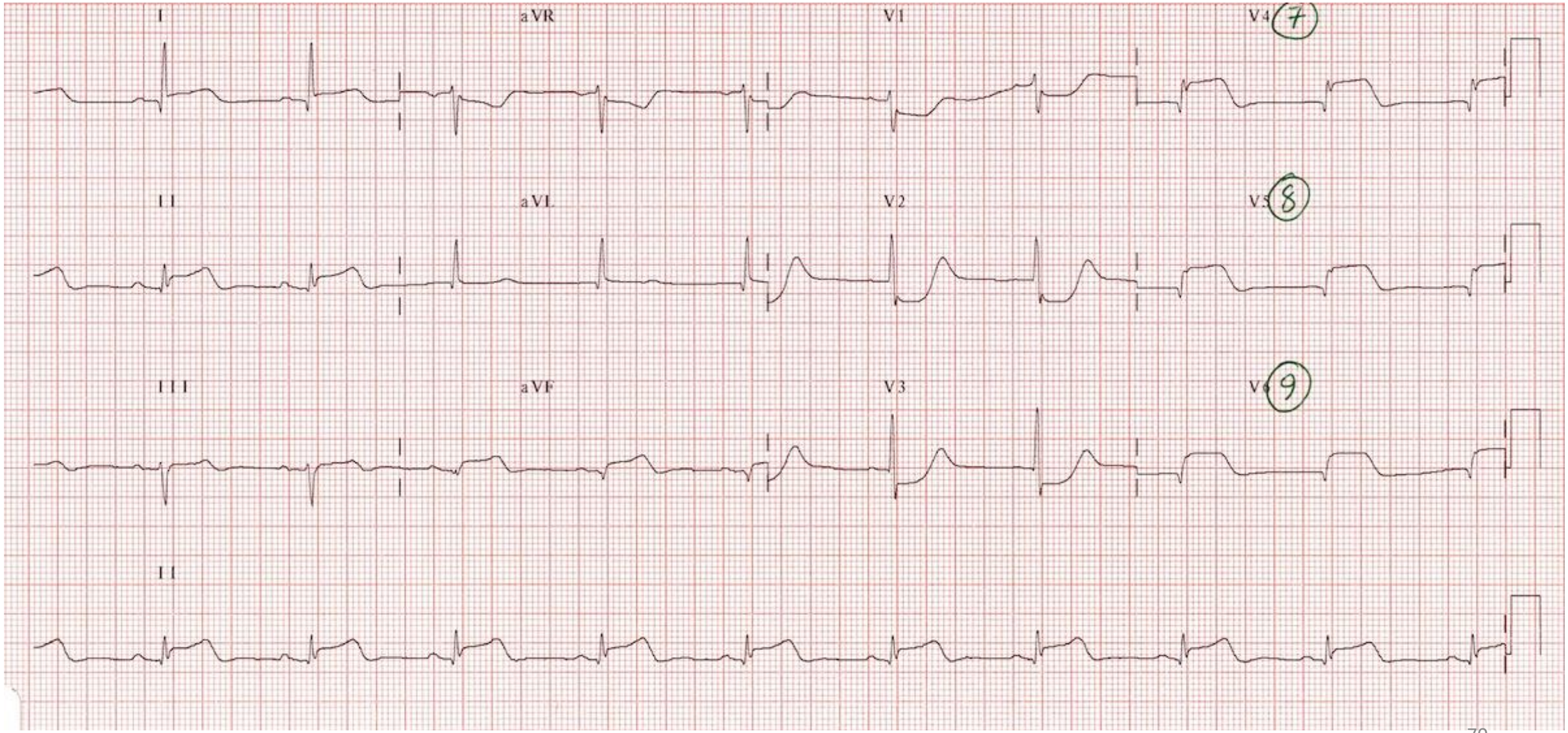


V2 inverted — the complexes now resemble a typical STEMI

Look for evidence of posterior involvement in any patient with an inferior or lateral STEMI.

Sometimes it can be difficult to determine whether ST depression in V2-3 is due to posterior STEMI or simply subendocardial ischaemia affecting the anteroseptal wall. The diagnosis can be confirmed by recording posterior leads V7-9.

Middle aged patient presenting with central chest pain. Posterior leads V7-9. What does the ECG show?



Posterior leads confirm the presence of posterior wall infarction by demonstrating typical STEMI morphology:

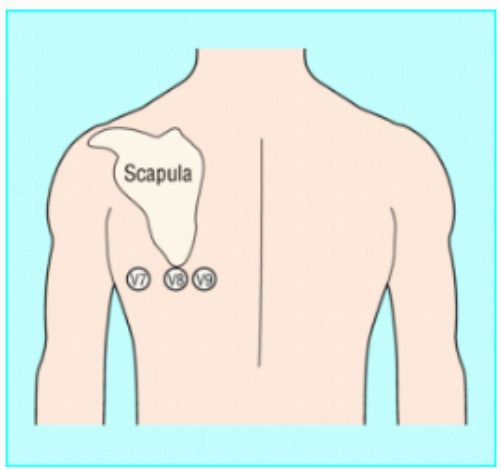
- ST elevation in V7-9
- Q waves in V7-9
- Inversion of the terminal portion of the T wave ("U wave inversion") in V7-9

How To Record Posterior Leads

Simply move the V4-6 electrodes around to the back in the same horizontal plane as V6. Annotate the ECG accordingly.

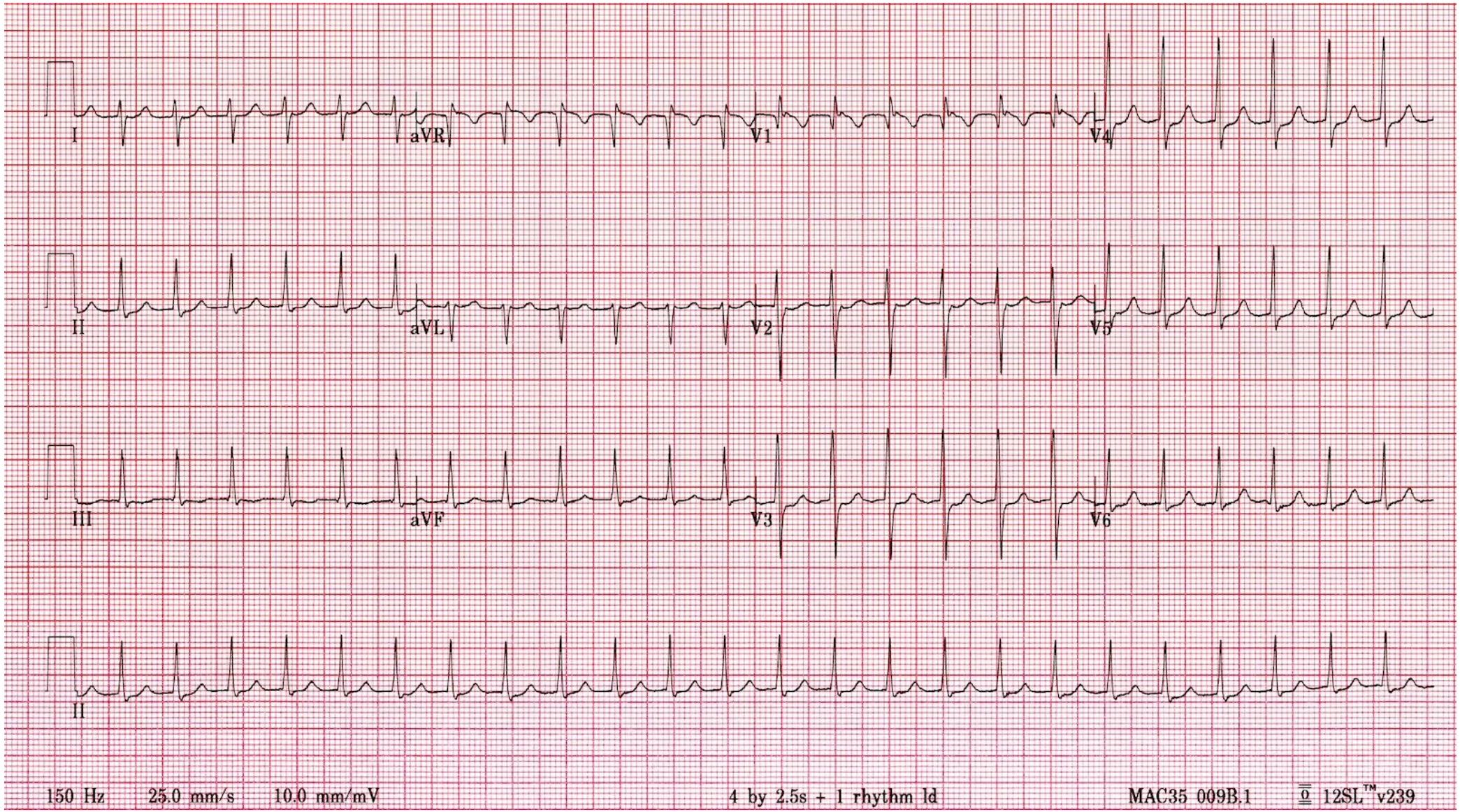
Approximate positions for V7-9 are:

- V7 – posterior axillary line
- V8 – tip of scapula
- V9 – left paraspinal region

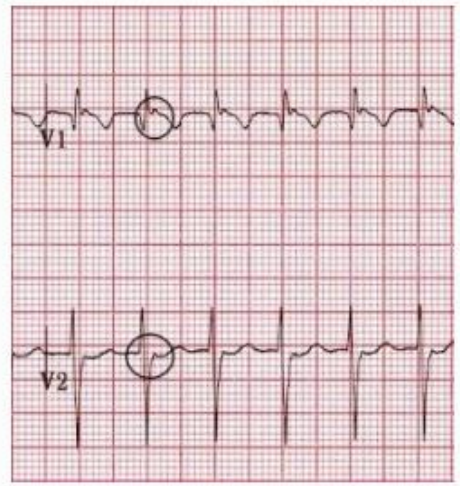


Reproduced from Morris and Brady, 2002.
Click image for link to original article.

20-year old patient with sudden onset of palpitations. What does the ECG show?



- Narrow complex tachycardia at ~ 150 bpm.
- Right axis deviation = just rightward of +90 degrees.
- Pseudo-R' waves in V1-2 = retrograde P waves superimposed on the terminal QRS causing peaking of the J-point.
- No clear sinus P waves or flutter waves seen.



Pseudo R' waves

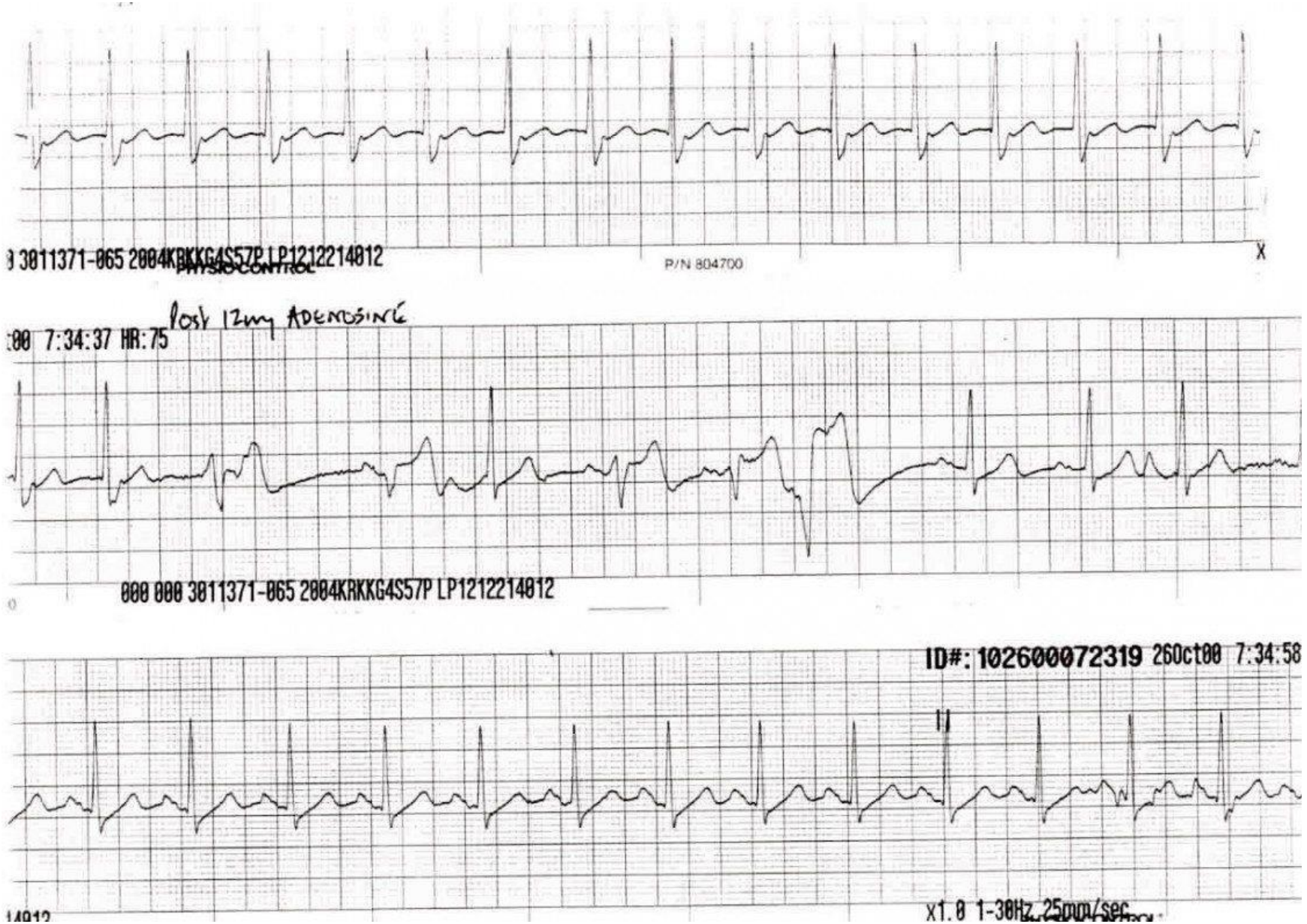
Differential Diagnosis

When you see a regular narrow complex tachycardia at 150 bpm, you should think of four main diagnoses:

- Atrial flutter with 2:1 block (*especially* in elderly, IHD, CCF)
- AV-nodal reentry tachycardia ("SVT")
- Orthodromic AV reentry tachycardia in WPW
- Sinus tachycardia — should see P waves but may be hidden in the T waves (e.g. with concurrent 1st degree AV block). There should also be some HR variability compared to the other 3 rhythms.

The patient's young age and presence of retrograde P waves (pseudo R' waves) suggest a paroxysmal reentry tachycardia involving the AV node — either AVNRT ("SVT") or orthodromic AVRT.

20-year old patient with sudden onset of palpitations. What does the rhythm strip demonstrate?



Top rhythm strip

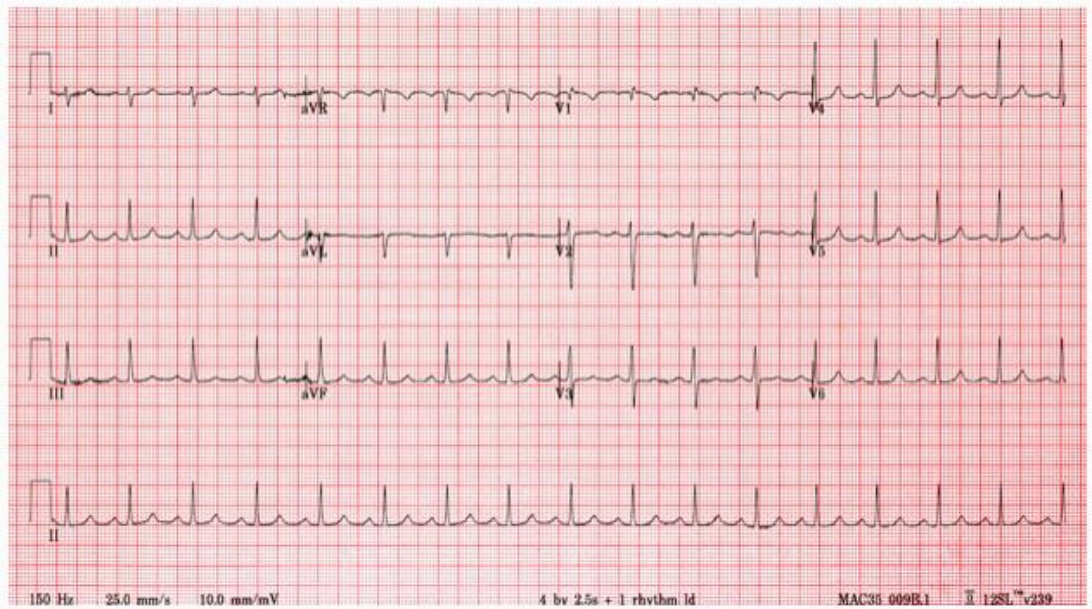
- Regular narrow complex tachycardia.
- Pseudo-R' waves (retrograde P waves) are seen deforming the J point.

Middle rhythm strip

- 12mg adenosine given
- A salvo of broad and bizarre-looking complexes interrupts the rhythm (this is a common phenomenon during chemical cardioversion with adenosine)

Bottom rhythm strip

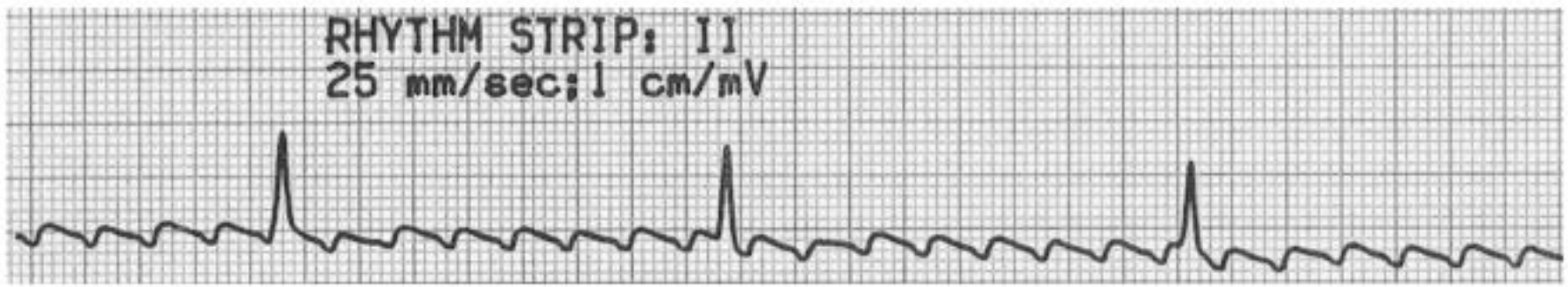
- The patient has reverted to sinus rhythm.
- The pseudo-R' waves have now disappeared.
- There are no obvious delta waves of WPW, but this should be confirmed on a 12-lead ECG.



The 12-lead ECG confirms reversion to sinus rhythm and does not demonstrate any features of WPW. The diagnosis is AV-nodal reentry tachycardia (AVNRT).

75-year old patient with narrow complex tachycardia at 150 bpm.

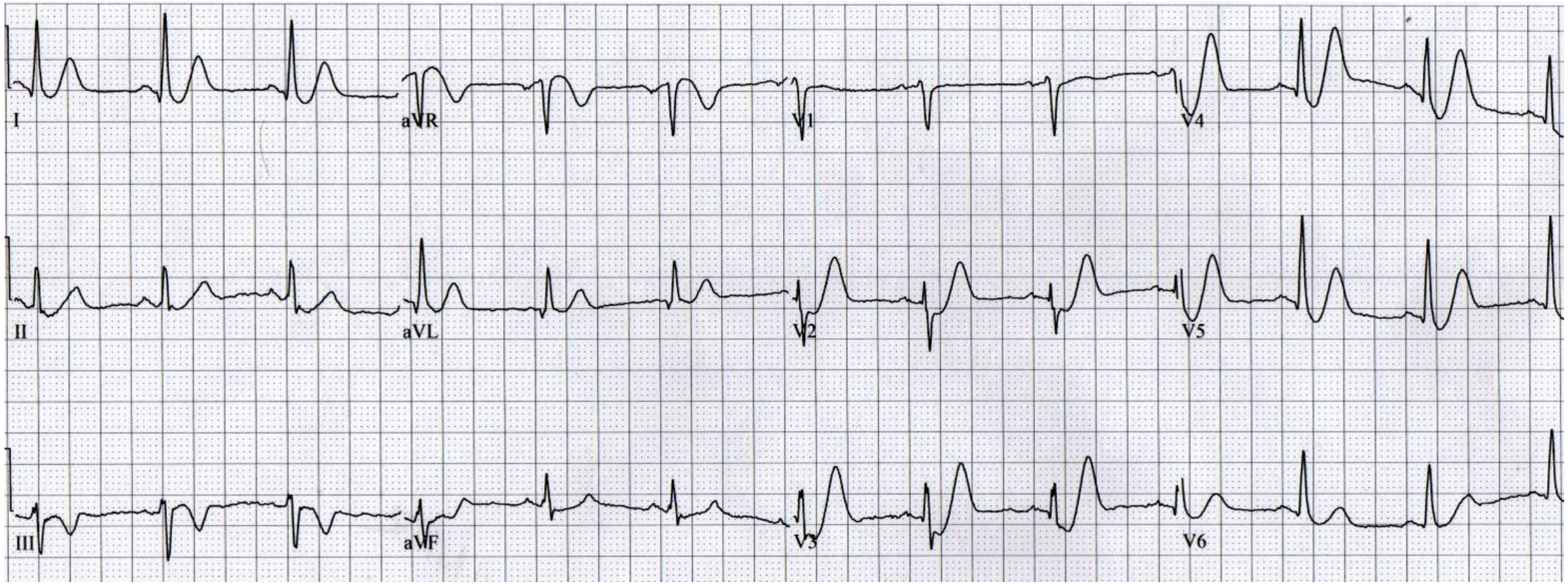
Rhythm strip during administration of IV adenosine. What is happening here?



In comparison to the previous example, this patient does not cardiovert to sinus rhythm following an adenosine bolus.

Instead, the degree of AV block is transiently increased, revealed underlying flutter waves and confirming the diagnosis of atrial flutter with a 2:1 block.

Middle aged patient presenting with chest pain and diaphoresis. What does the ECG show?

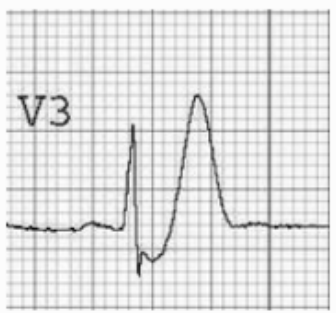


Main Abnormalities

- ST depression in V2-5, which slopes upwards and joins the ascending limb of the T wave.
- Prominent, “rocket-shaped” T waves in the precordial leads V2-5.
- Subtle ST elevation in aVR.

Diagnosis

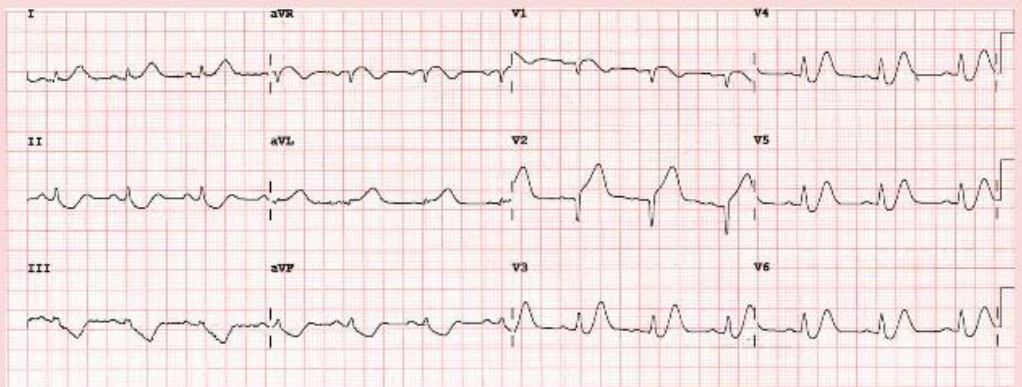
- This combination of ST depression with rocket-shaped T waves in the precordial leads V1-6 is referred to as the De Winter ECG pattern or “De Winter’s T waves”.
- It is becoming increasingly recognised as an anterior STEMI equivalent (~2% of LAD occlusions).
- Some authors are now recommending that this ECG pattern be treated identically to anterior STEMI, with urgent PCI or thrombolysis.



Typical De Winter’s T Wave

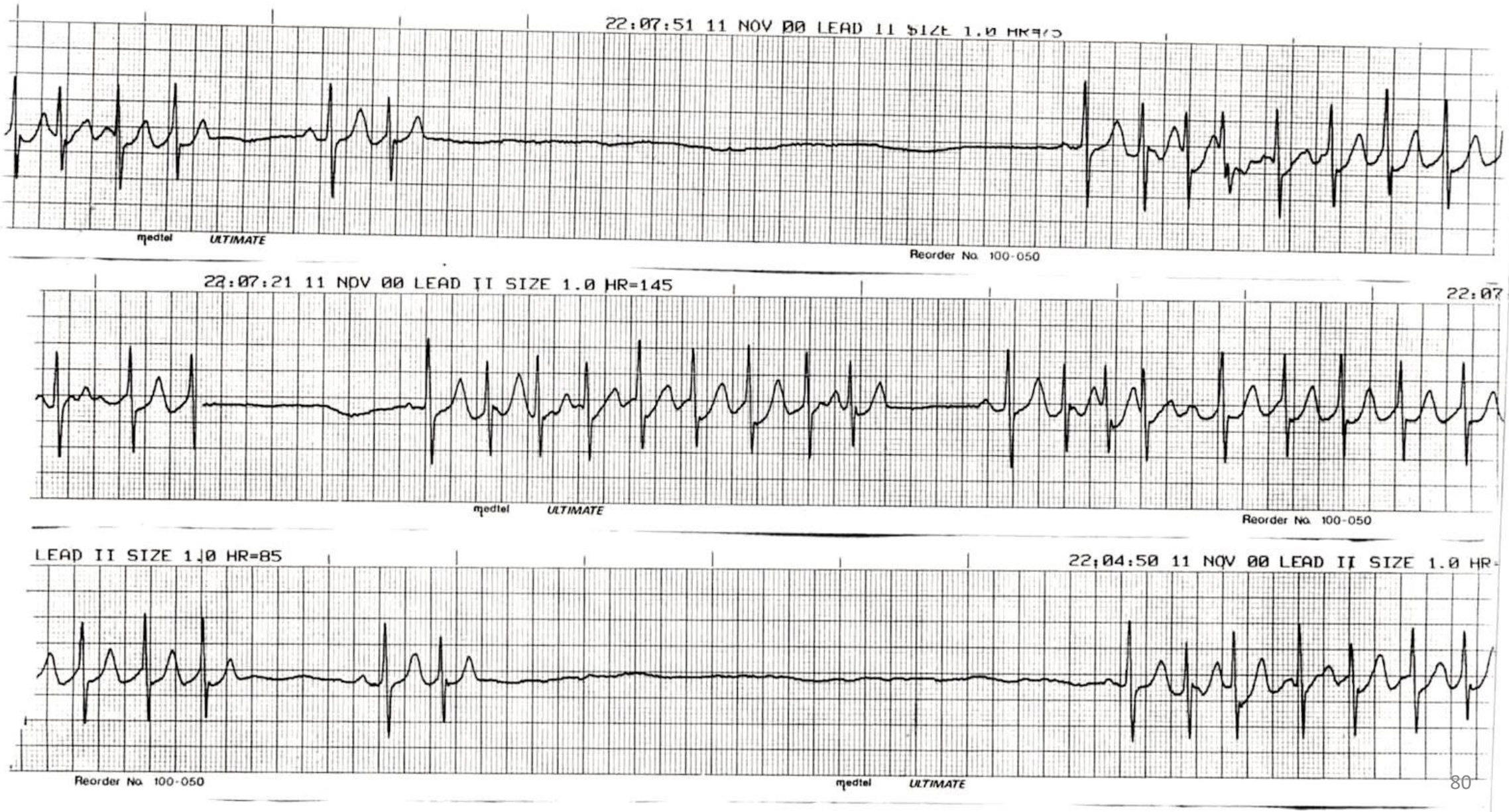
Clinical Pearls

The De Winter ECG pattern may evolve into (or even evolve from) a more classic STEMI pattern.



ECG courtesy of Jennifer Davidson

80-year old patient with palpitations and syncope. What does the ECG show?



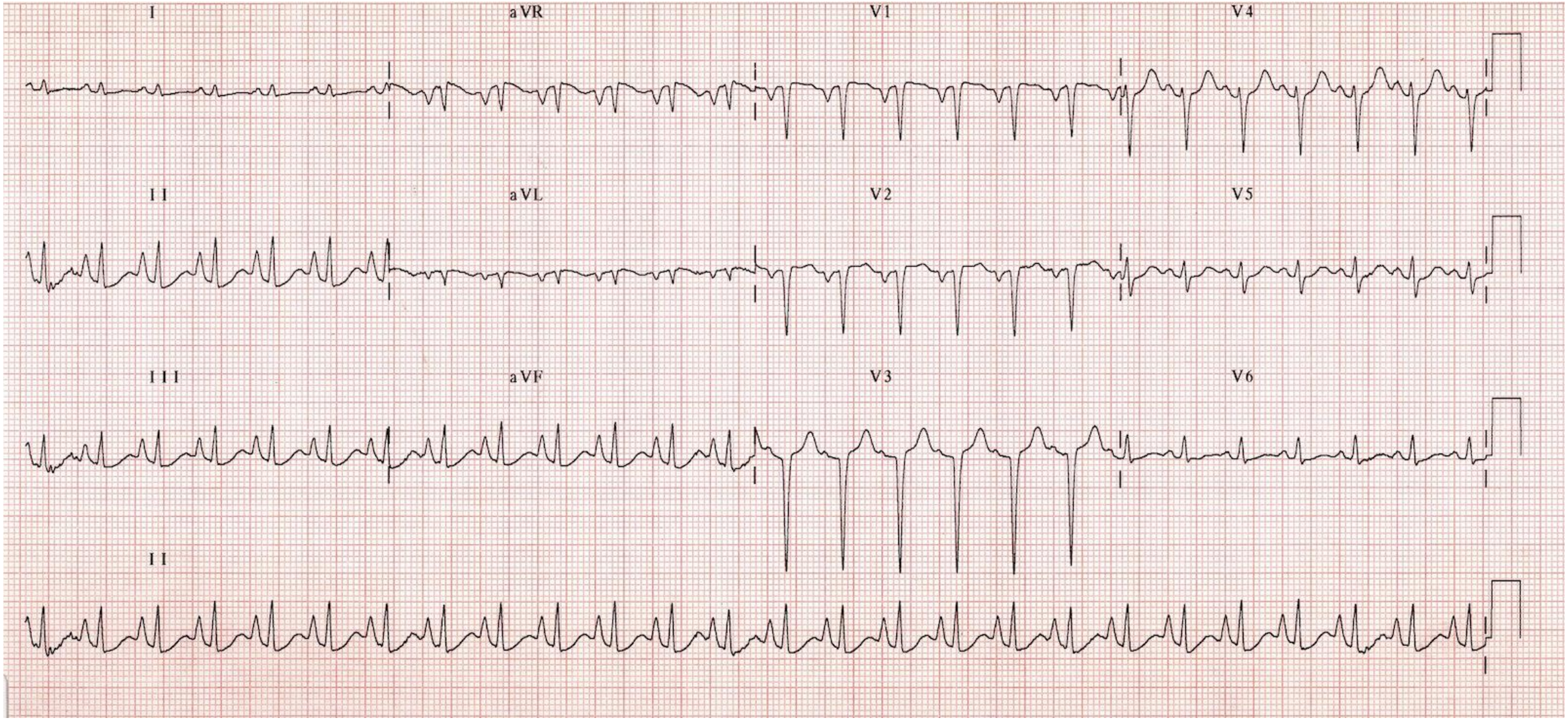
Main Abnormalities

- Runs of tachycardia are interspersed with long sinus pauses (up to 6 seconds).
- The sinus rate is extremely slow, varying from 40 bpm down to around 10 bpm in places.
- Sinus beats are followed by paroxysms of junctional tachycardia at around 140 bpm.

Diagnosis

- This is a good example of sick sinus syndrome leading to the tachycardia-bradycardia syndrome.
- The flurries of junctional tachycardia are a compensatory phenomenon attempting to maintain cardiac output in the face of critically low sinus node rates.
- The syncope likely occurred due to a long sinus pause with temporary loss of cardiac output. This patient needs a pacemaker!

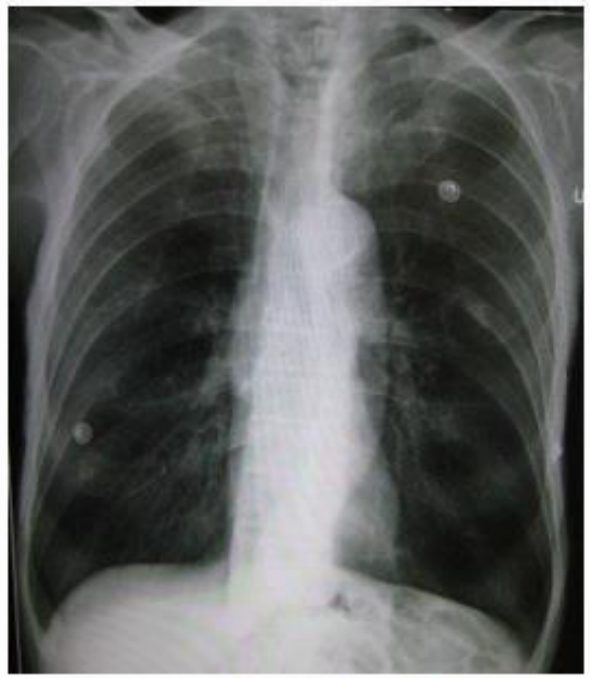
75-year old smoker presenting with acute dyspnoea and productive cough. Describe the ECG.



This ECG demonstrates many of the features of chronic pulmonary disease:

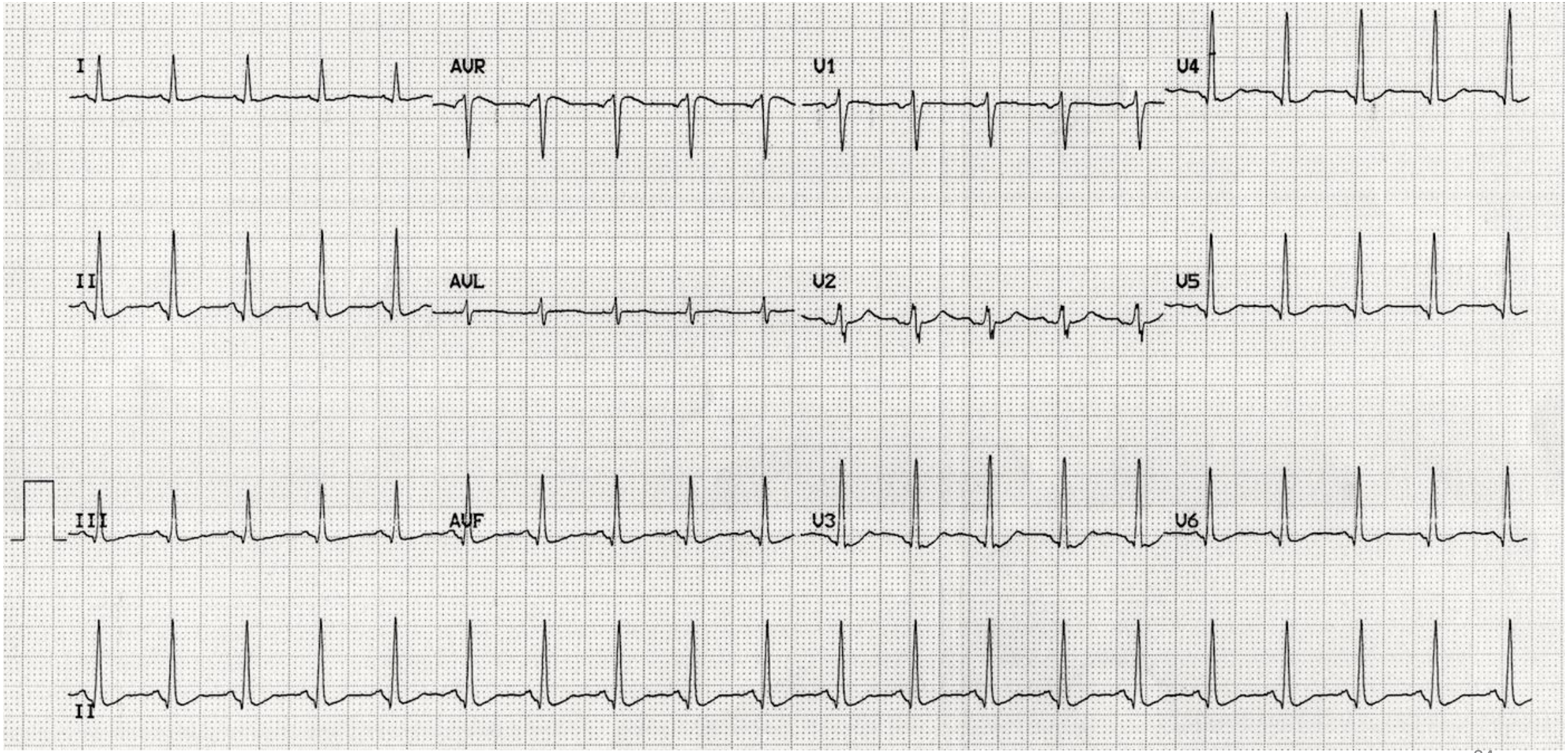
- Rightward QRS axis (+90 degrees).
- Peaked P waves in the inferior leads > 2.5 mm (P pulmonale).
- Rightward P-wave axis (inverted in aVL).
- "Clockwise rotation" of the heart with a delayed R/S transition point (transitional lead = V5).
- Absent R waves in the right precordial leads (SV1-SV2-SV3 pattern).
- Low voltages in the left-sided leads (I, aVL, V5-6).

Tachycardia may be due to dyspnoea, hypoxia or beta-agonist treatment. This ECG pattern is a common finding in patients with COPD. The vertical axis (+90 degrees) is due to hyperinflation of the lungs causing vertical orientation of the heart.



Vertical heart orientation in COPD

Post-intubation ECG of a young adult presenting with coma following a 6g quetiapine overdose. Describe the ECG.



Main Abnormalities This ECG displays the characteristic electrocardiographic features of quetiapine toxicity:

- Sinus tachycardia due to anticholinergic effects.
- Prolonged QT interval (QT interval > half the RR interval; QTc = 560ms).

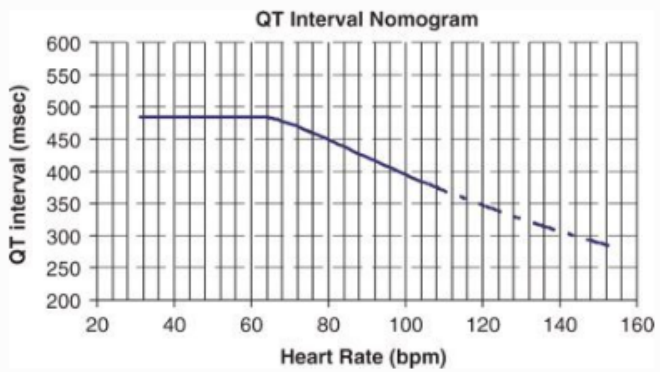
A similar pattern would be seen with other atypical antipsychotic agents such as olanzapine or clozapine.

Significance of QT prolongation

- QT prolongation is a common source of concern in patients with antipsychotic toxicity, because of the theoretical risk of Torsades de Pointes.
- A QTc interval > 500 ms is commonly cited as a marker of increased risk of TdP.
- However, tachycardia (which is almost ubiquitous in significant poisoning with quetiapine, olanzapine or clozapine) is actually protective against TdP.
- For this reason, TDP rarely occurs with quetiapine toxicity.

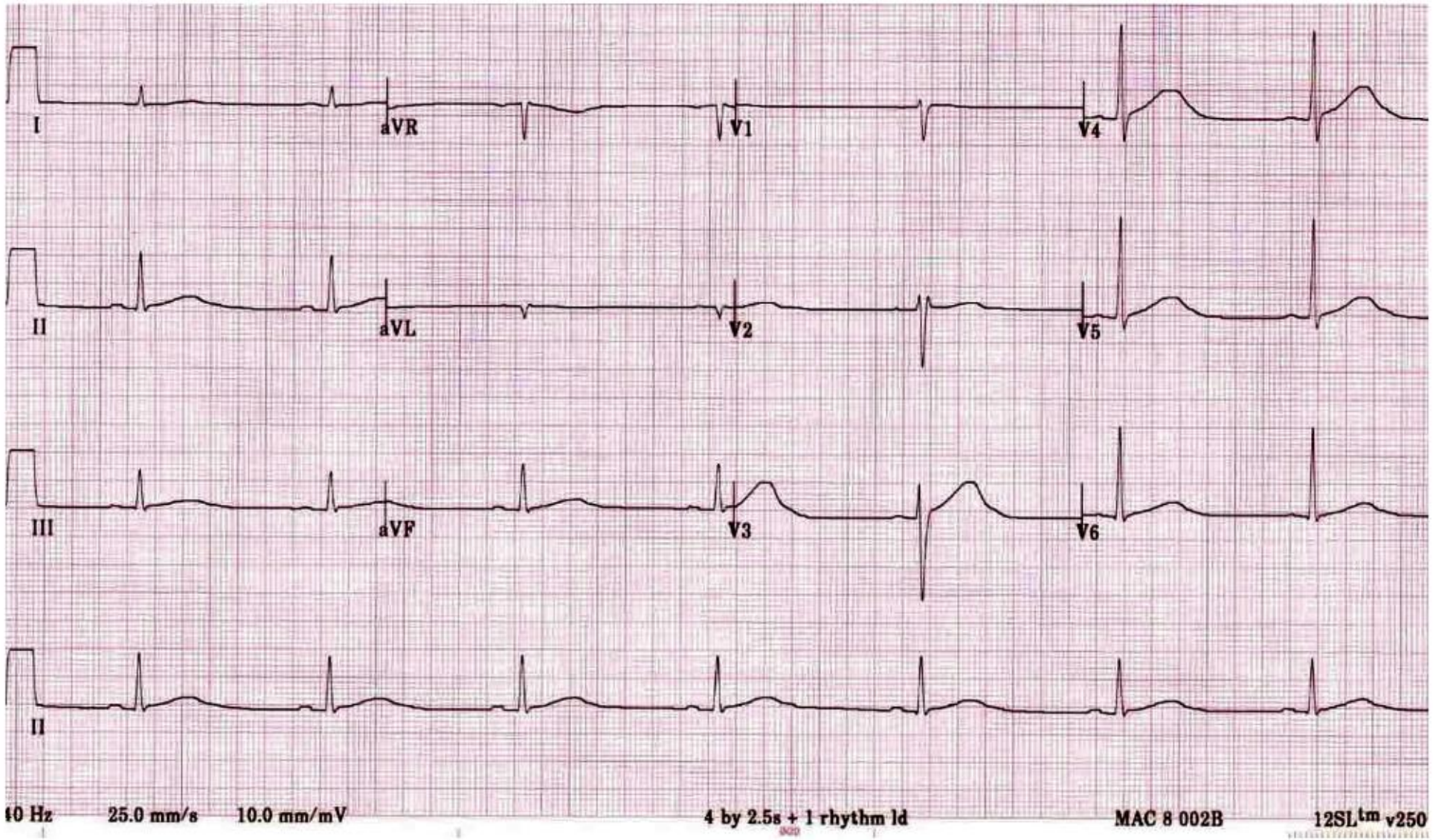
QT interval nomogram

- Many Australian toxicologists use the QT interval nomogram to assess risk of TdP.
- The *absolute* QT interval is measured manually in multiple leads and the median QT interval plotted on the nomogram (read how to do this here).
- Plots *above* the line indicate significant QT prolongation and consequent risk of TdP.



By my measurements, our patient has an absolute QT of ~320 ms with HR 120 so plots below the line — i.e. *not* at significant risk of TdP.

Elderly patient with accidental overdose of sotalol. Describe the ECG.



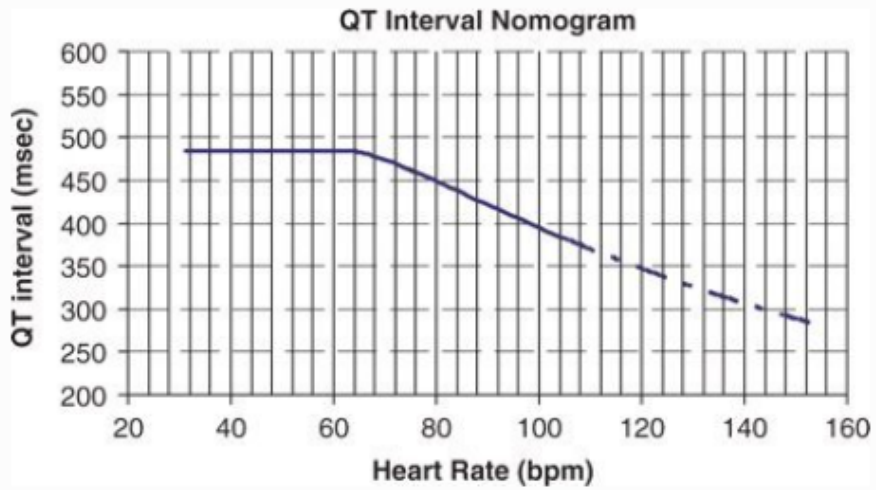
Main Abnormalities This ECG demonstrates the key features of sotalol toxicity:

- Sinus bradycardia (42 bpm)
- Very long QT interval (~600 ms).

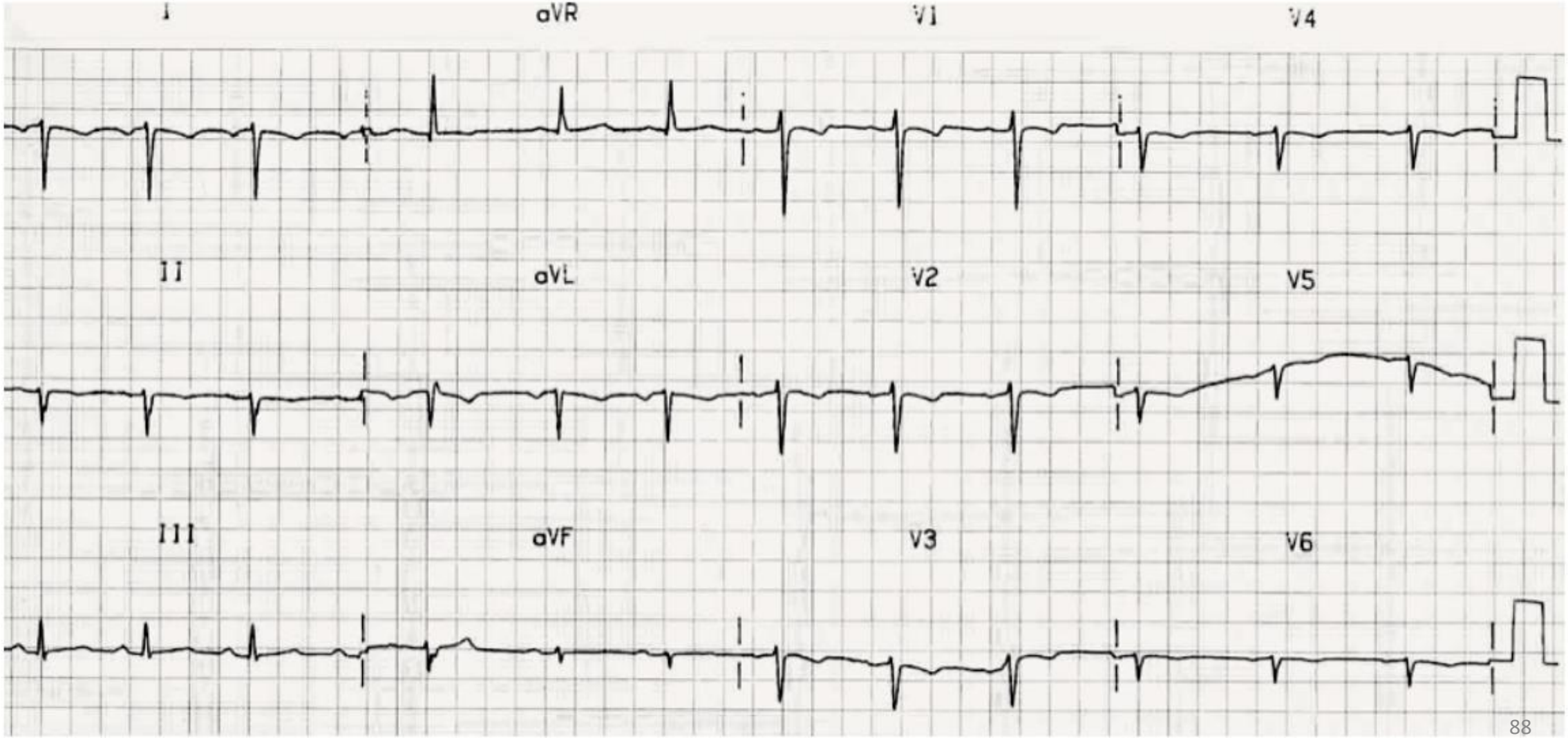
Sotalol is a beta blocker with additional class III effects (potassium channel blockade), so it causes both bradycardia and QT prolongation in overdose.

Risk of Torsades

- In comparison to the previous case, this patient is at significant risk of TdP.
- The combination of bradycardia and significant QT prolongation means that this patient plots well above the "at risk" line on the QT nomogram.
- Prophylaxis of TdP in this case would include correction of QT-dependent electrolytes (K, Mg, Ca) to the high-normal range and positive chronotropy (e.g. with isoprenaline) to move the patient below the at-risk line.



Asymptomatic adult patient. Routine ECG. Describe the ECG.



Main Abnormalities

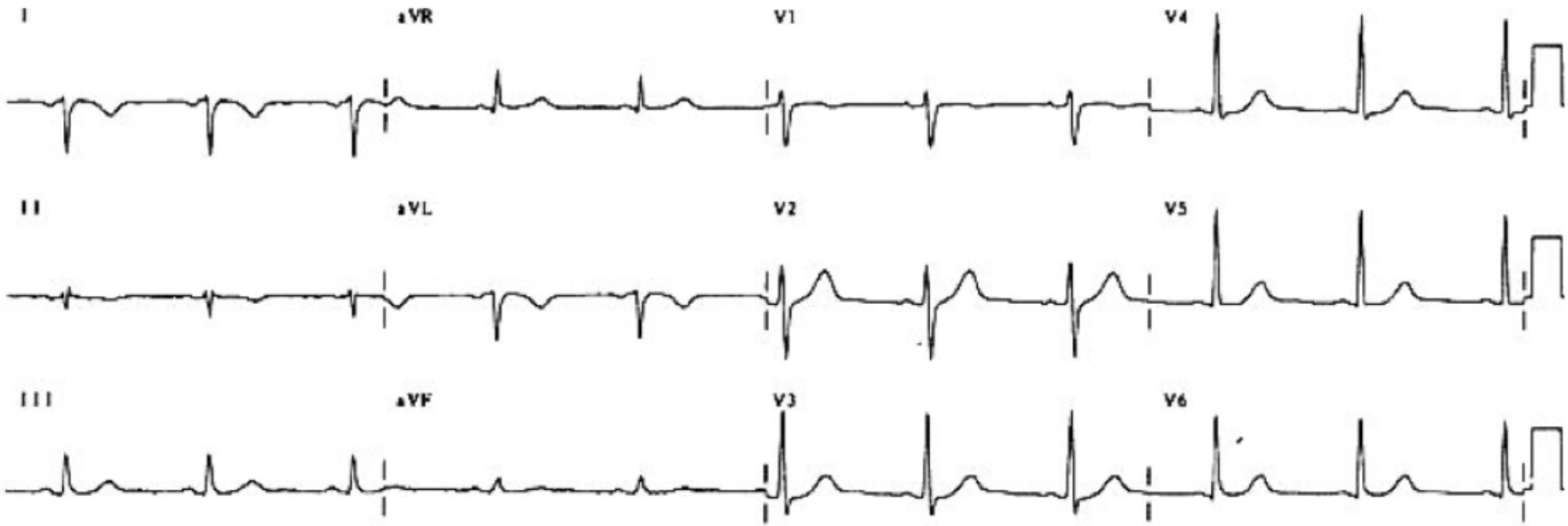
This ECG demonstrates the typical features of dextrocardia:

- Marked right axis deviation (+180 degrees).
- Lead aVR: Positive QRS complex (upright P and T waves).
- Lead I: inversion of all complexes, aka ‘global negativity’ (inverted P-QRS-T).
- Absent R-wave progression in the chest leads (dominant S waves throughout).

Differential Diagnosis

Accidental reversal of the left and right arm electrodes may produce a similar picture to dextrocardia in the limb leads, but with normal appearances in the precordial leads.

Asymptomatic adult patient. Routine ECG. Describe the ECG.



Main Abnormalities This ECG is a classic example of limb lead misplacement with a left arm / right arm lead reversal:

- Positive P-QRS-T in lead aVR.
- Inverted complexes in leads I and aVL.
- Normal complexes in the precordial leads rules out dextrocardia (compare this to the previous ECG).

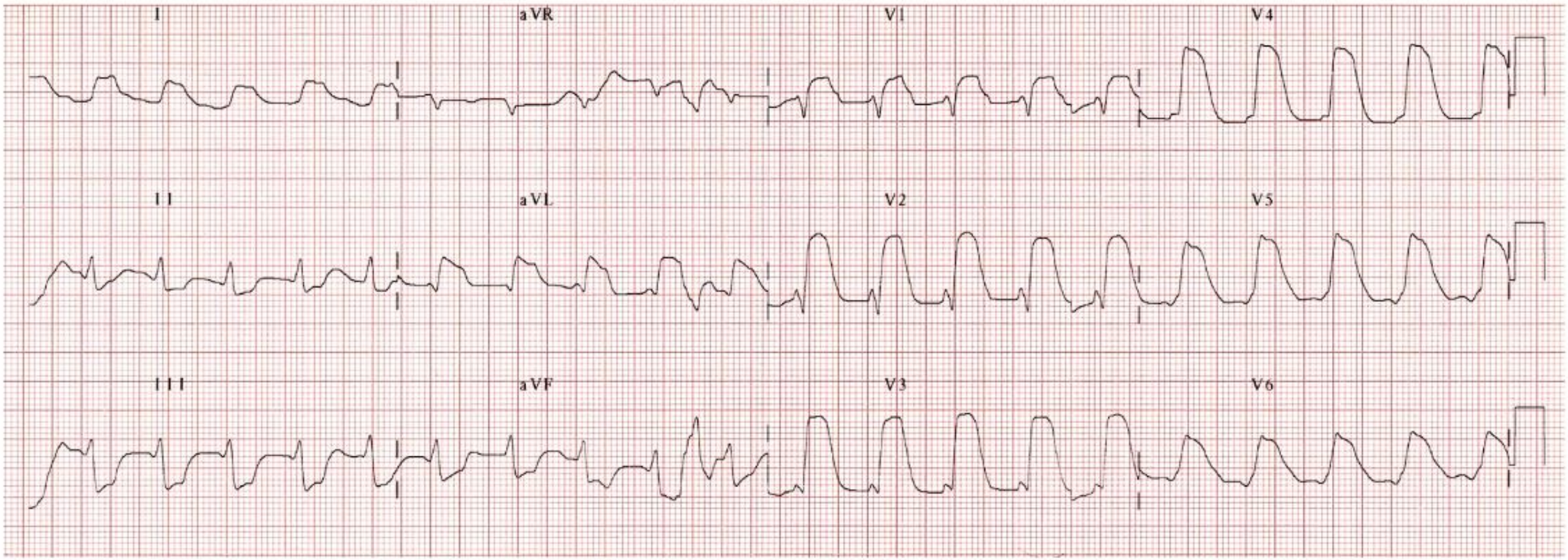
The most obvious abnormality on this ECG is the positive QRS complex in aVR. This is unusual and should always prompt a search for additional evidence of limb lead misplacement.

Effects of LA-RA lead reversal

Switching the LA and RA electrodes produces the following effects:

- Lead I becomes inverted.
- Leads II and III switch places.
- Leads aVL and aVR switch places (hence aVR becomes positive, aVL negative).
- Lead aVF remains unchanged

70-year old patient presenting with chest pain and diaphoresis. Describe the ECG.

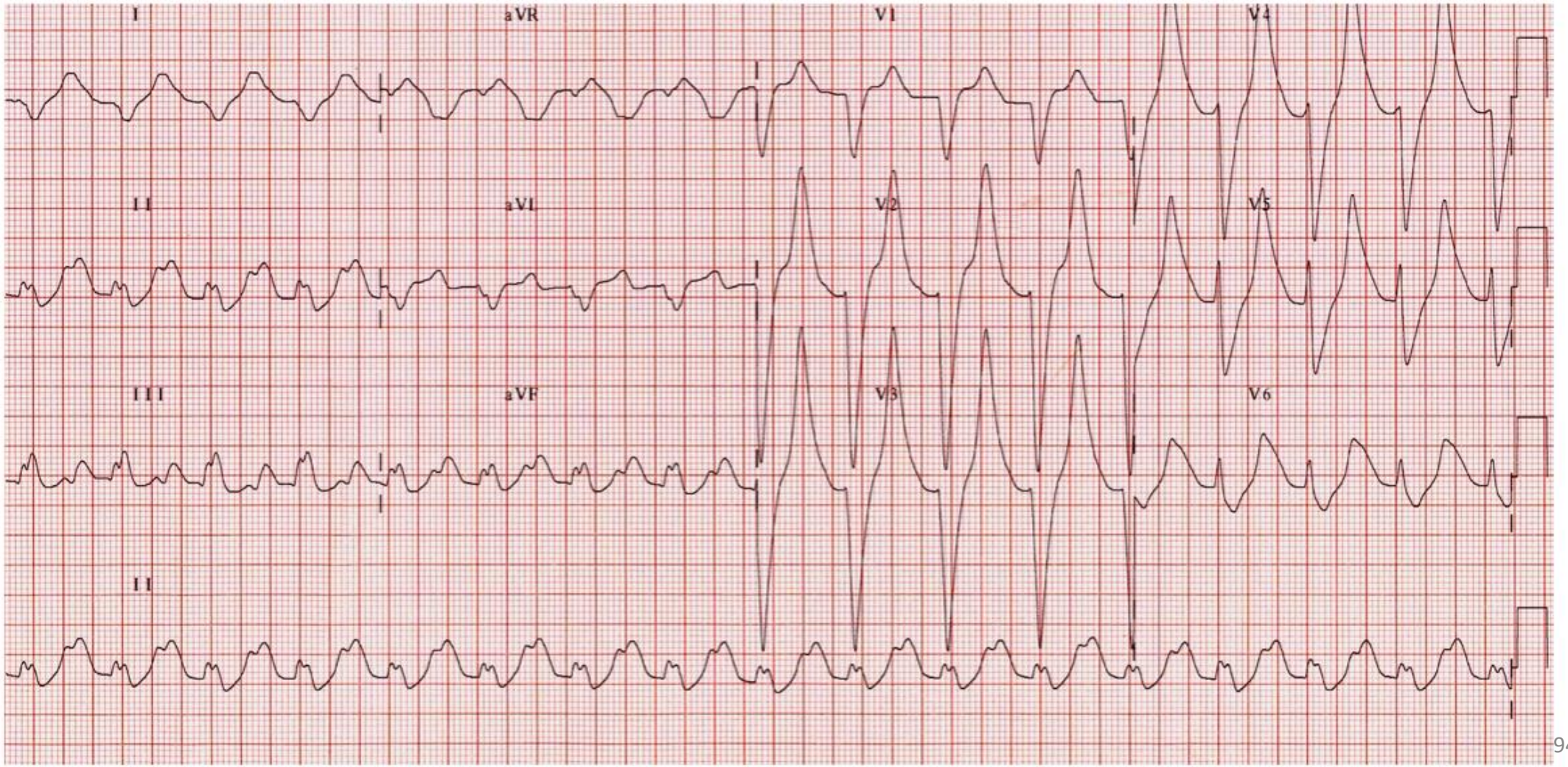


This is a very worrying ECG demonstrating massive anterolateral STEMI with “tombstone” morphology:

- Gross ST elevation in V1-6, I and aVL.
- Early Q wave formation in aVL.
- Reciprocal ST depression in inferior leads II, III and aVF.

This ECG pattern is seen in proximal LAD occlusion and indicates a large territory infarction with a poor LV ejection fraction and high likelihood of cardiogenic shock and death.

Elderly patient feeling generally unwell. PMHx of T2DM, hypertension, IHD, CCF, osteoarthritis. Describe the ECG.



Main Abnormalities

- Bizarre appearing complexes.
- Marked T wave peaking in V2-6.
- Gross QRS prolongation (~200 ms).
- Some leads (I, aVR) are starting to take on a sine wave appearance.

Diagnosis

The combination of…

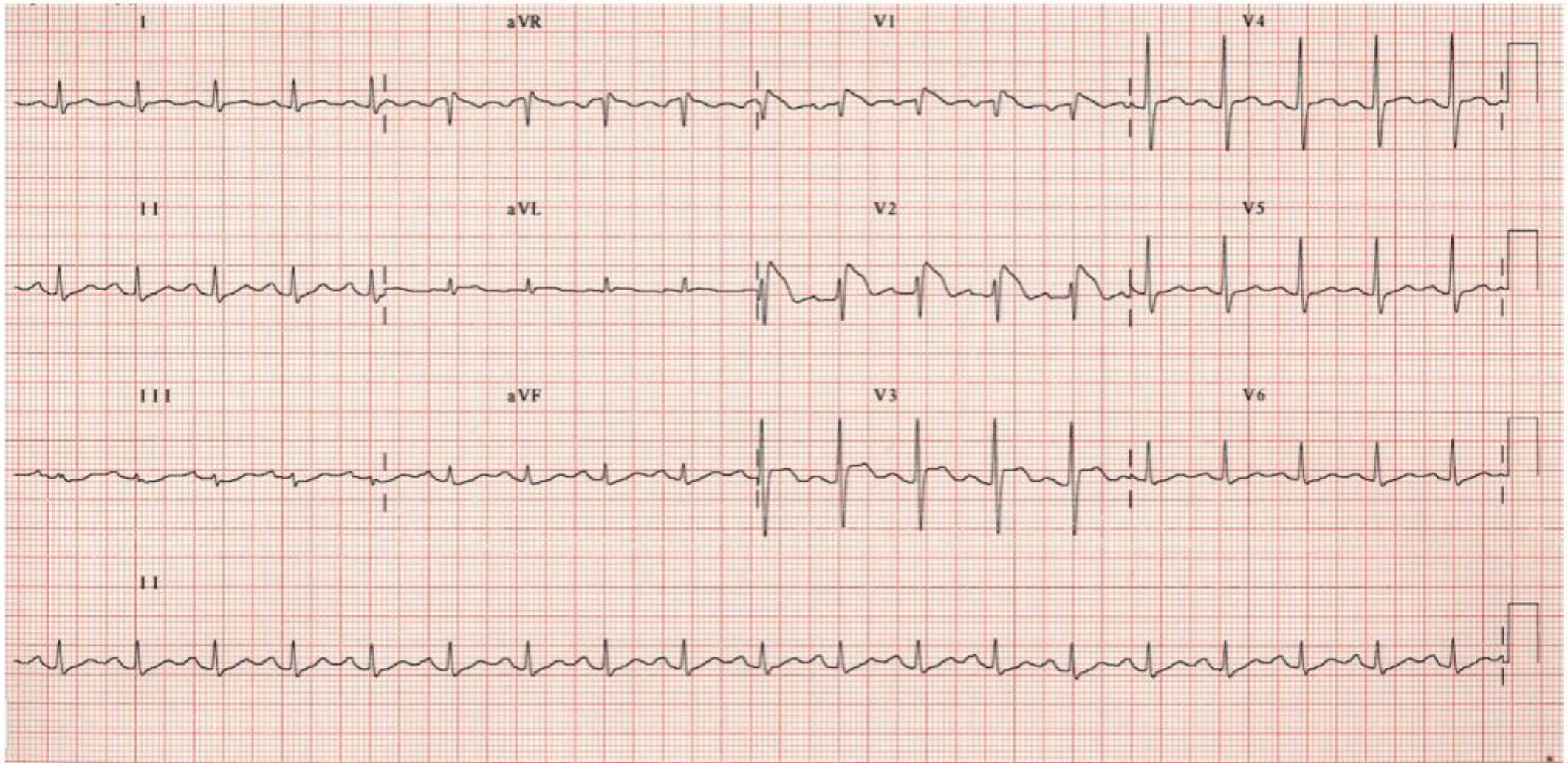
- Bizarre complexes
- QRS prolongation
- Peaked T waves
- Sine wave appearance

… are all strongly suggestive of severe hyperkalemia.

This patient had a serum K of 9.2 mmol/L!

In this elderly patient with multiple medical problems, causes could include renal failure (e.g. due to diuretics, NSAIDs, intercurrent illness) or treatment with ACE-inhibitors, spironalactone or K-supplements.

30-year old Thai male presenting with syncope. Describe the ECG.



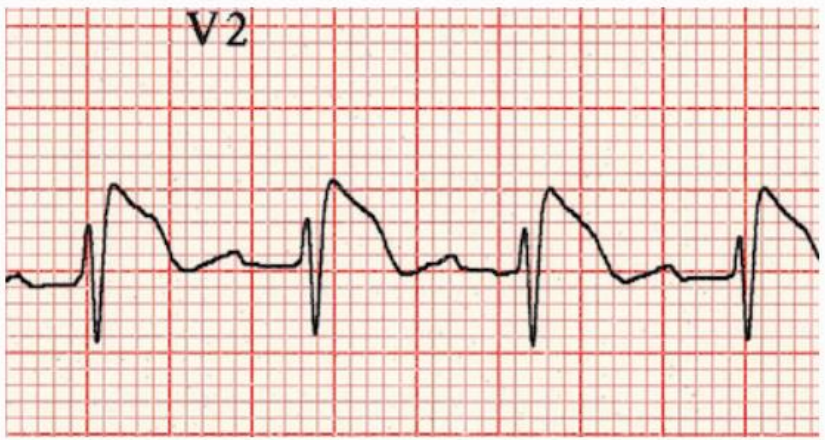
ECG Findings The patient is in sinus rhythm with no evidence of dysrhythmia or AV block.

The QT interval is normal and there is no evidence of WPW syndrome, HOCM or ARVC.

There is a characteristic pattern of abnormalities in V1-2:

- RSR' pattern / partial RBBB.
- ST elevation with a "coved" morphology.
- Inversion of the terminal portion of the T wave.

In a patient presenting with syncope, this ECG is diagnostic of the Brugada syndrome.



Classic appearance of Brugada syndrome in V2

Syncope ECG Checklist

When faced with a patient presenting with syncope, I systematically assess the ECG for the following abnormalities (click each item for details):

Too Fast?

- Any abnormal tachycardia — e.g. ventricular tachycardia, TdP.

Too Slow?

- AV block — e.g. Mobitz II or complete heart block.
- Sinus bradycardia or sinus pauses.

Pump Failure

- Evidence of myocardial ischaemia.
- Evidence of pulmonary embolism.

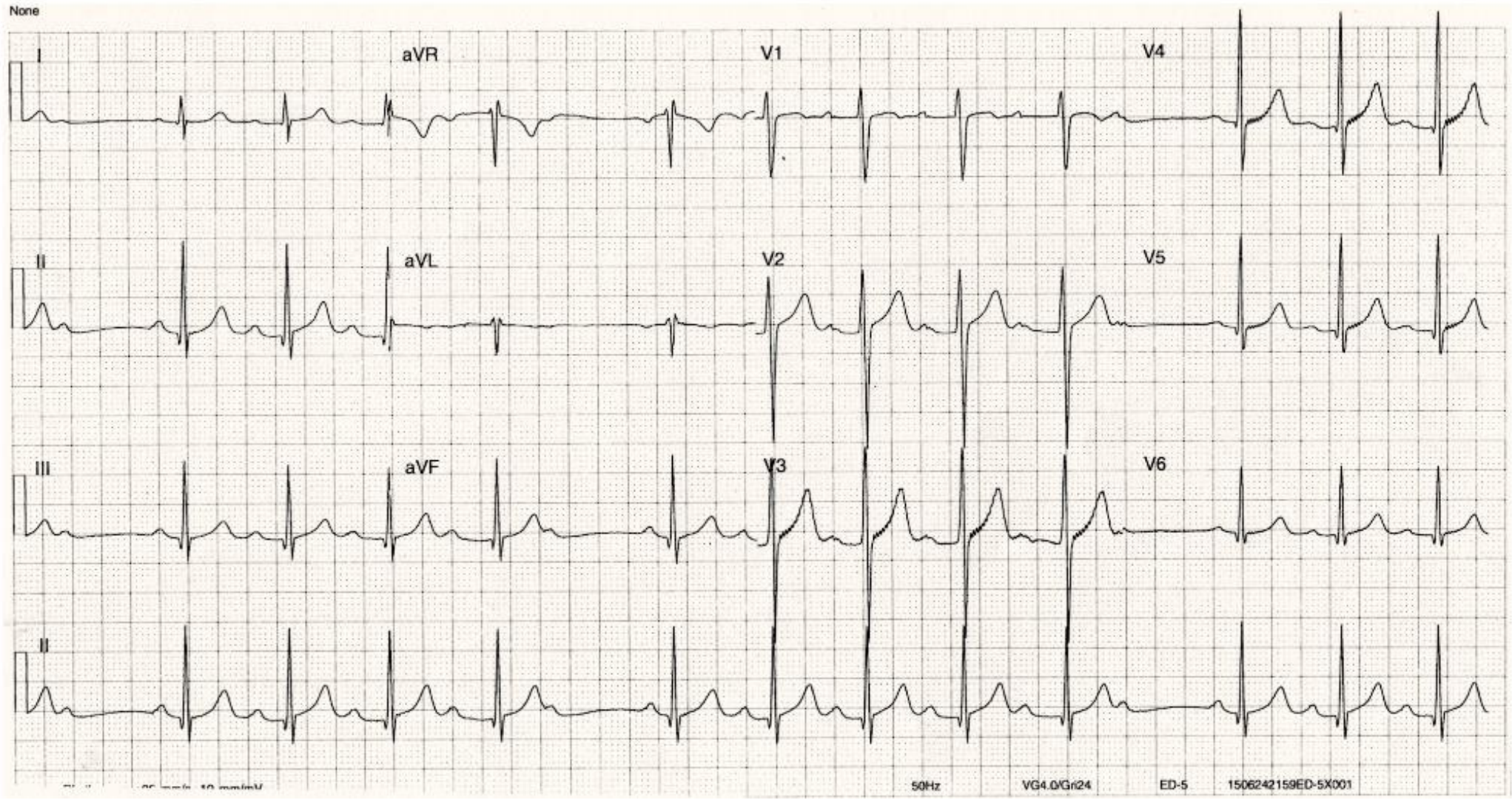
Syncope Syndromes

- WPW — delta wave, short PR.
- Long QT syndrome? — QT > 500 ms.
- Short QT syndrome? — QT < 320 ms.
- Brugada syndrome? — RSR' with STE in V1-3.
- HOCM — voltage criteria for LVH, precordial TWI, "dagger" Q waves.
- ARVC — signs of RVH, Epsilon waves.

Electrical Problems

- Electrolyte abnormality — e.g. hypokalaemia or hyperkalaemia.
- Evidence of pacemaker failure.

Asymptomatic 40-year old patient. Describe the ECG.



Main Findings

- Irregular narrow-complex rhythm (overall rate = 72 bpm).
- Normal sinus P waves are seen (upright in lead II), indicating a sinus origin of the rhythm.
- QRS complexes cluster in groups, separated by non-conducted P waves.
- The PR interval progressively prolongs within each group.
- The PR prolongation can be appreciated by comparing the first and last PR interval of each group.

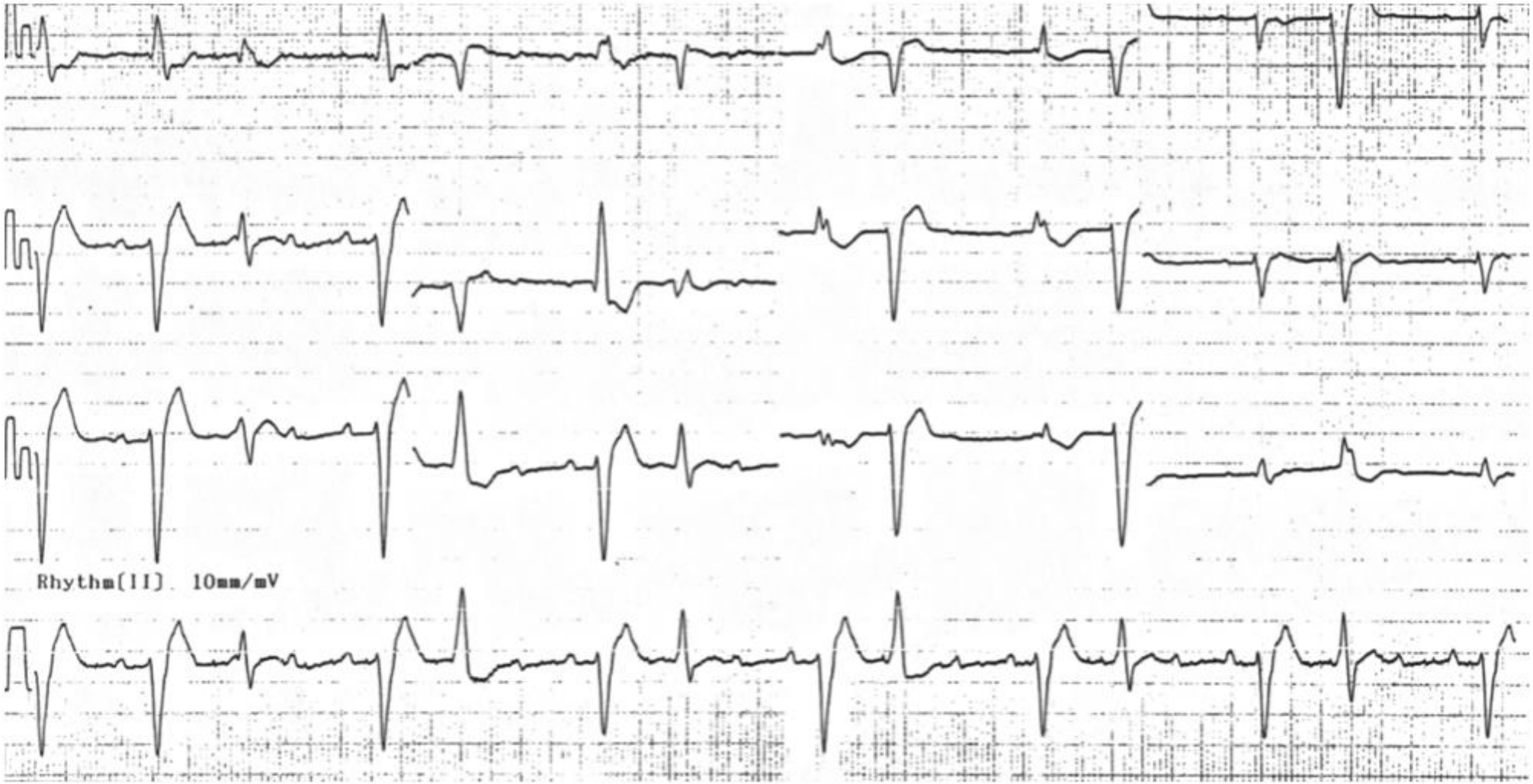
Diagnosis

- This is the typical appearance of 2nd degree AV block with Mobitz I conduction (Wenchebach phenomenon).

Clinical Significance

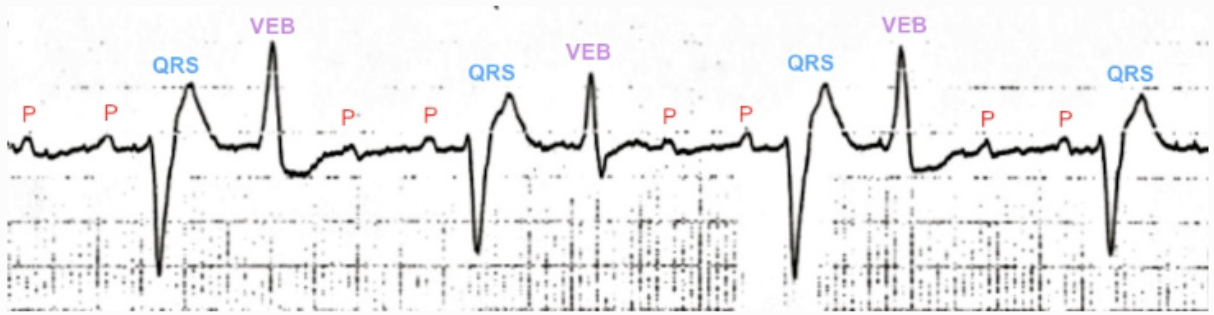
In comparison to patients with Mobitz II, who typically require a pacemaker for prophylaxis of complete heart block and ventricular standstill, patients with Mobitz I do not necessarily need any intervention. This is provided that they are asymptomatic with a normal BP, and that reversible causes such as drug toxicity (beta-blockers, digoxin), hyperkalaemia and myocardial ischaemia have been excluded. The risk of progression to haemodynamically unstable AV block in these patients is very low.

85-year old patient presenting with nausea, vomiting and visual disturbance. Looks clinically dehydrated. Describe the ECG.



Main Abnormalities

- Atrial tachycardia, with regular P waves visible at ~ 160 bpm (many of the P waves are hidden within T waves and VEBs).
- Evidence of high-grade AV block — there is a 4:1 conduction ratio between P waves and QRS complexes, with a QRS rate of ~ 40 bpm.
- Frequent ventricular ectopic beats occurring in a pattern of ventricular bigeminy.
- **Alternating LBBB and RBBB morphology**, with the conducted QRS complexes demonstrating RBBB morphology (RSR' in V1) and the VEBs demonstrating LBBB morphology (dominant S wave in V1).



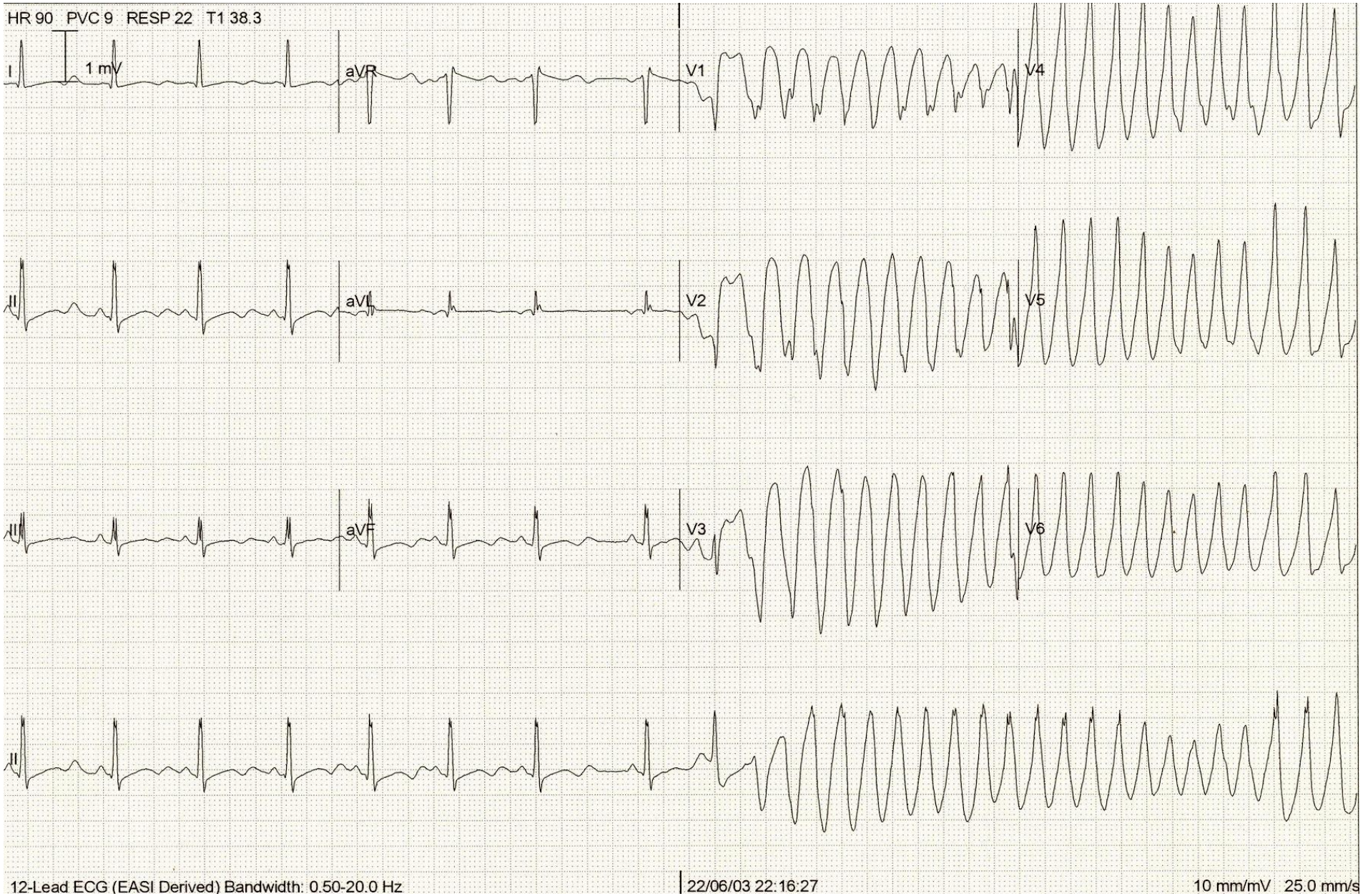
Diagnosis

The combination of…

- Atrial tachycardia
- Frequent ventricular ectopic beats
- High-grade AV block

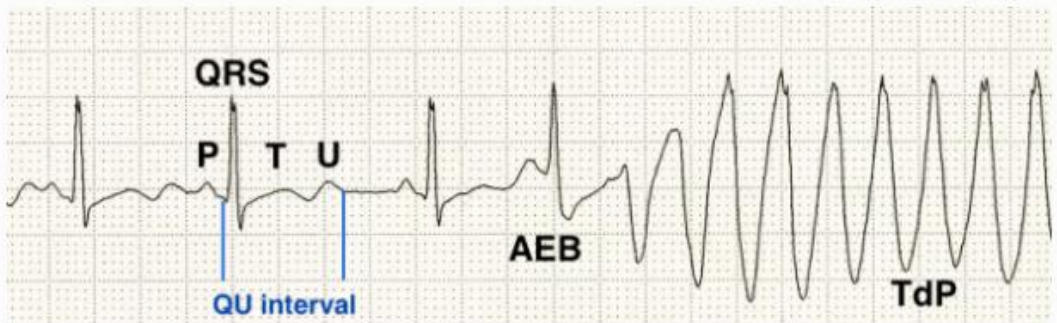
… is almost pathognomonic of severe digoxin toxicity.

Young adult patient presenting with syncope. History of eating disorder. Describe the ECG.



Main Abnormalities

- The first half of the ECG shows sinus rhythm with prominent U waves and a long QU interval (520ms).
- An atrial ectopic beat kicks off a run of Torsades de Pointes by landing on the T/U wave during the vulnerable phase of repolarisation and causing "R on T" (or "R on U") phenomenon.



Diagnosis

The combination of…

- Atrial ectopy
- Prominent U waves
- Long QU interval
- Torsades de Pointes

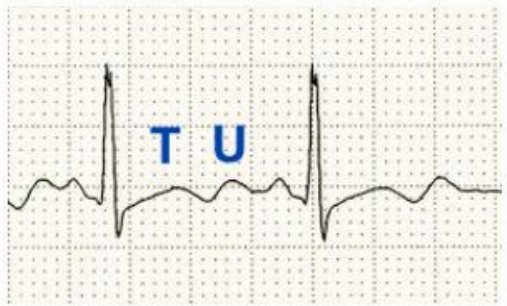
… is strongly suggestive of severe hypokalaemia. This patient had a K of 1.9 mmol/L.

Hypokalaemia occurs in eating disorders via multiple mechanisms including:

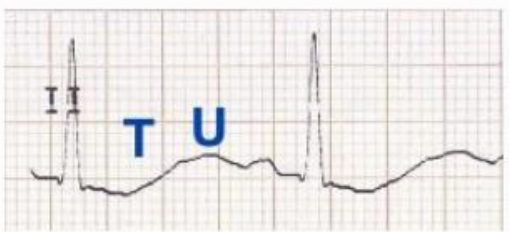
- Loss of K+ in bodily secretions — vomiting, purging with laxatives or diuretics.
- Reduced oral intake.
- Metabolic alkalosis from vomiting causing intracellular shifts of K+.
- Hypovolaemia causing secondary aldosteronism with renal loss of K+.

U wave morphology

The appearance of U waves in hypokalemia may vary. This example demonstrates discrete U waves that are clearly distinguishable from the T wave. Compare this with Quiz ECG 006, where the whole ST-T-U complex is fused together to produce a "rollercoaster" appearance, with the U wave appearing as a positive deflection that emerges from a negative ST segment and T wave.

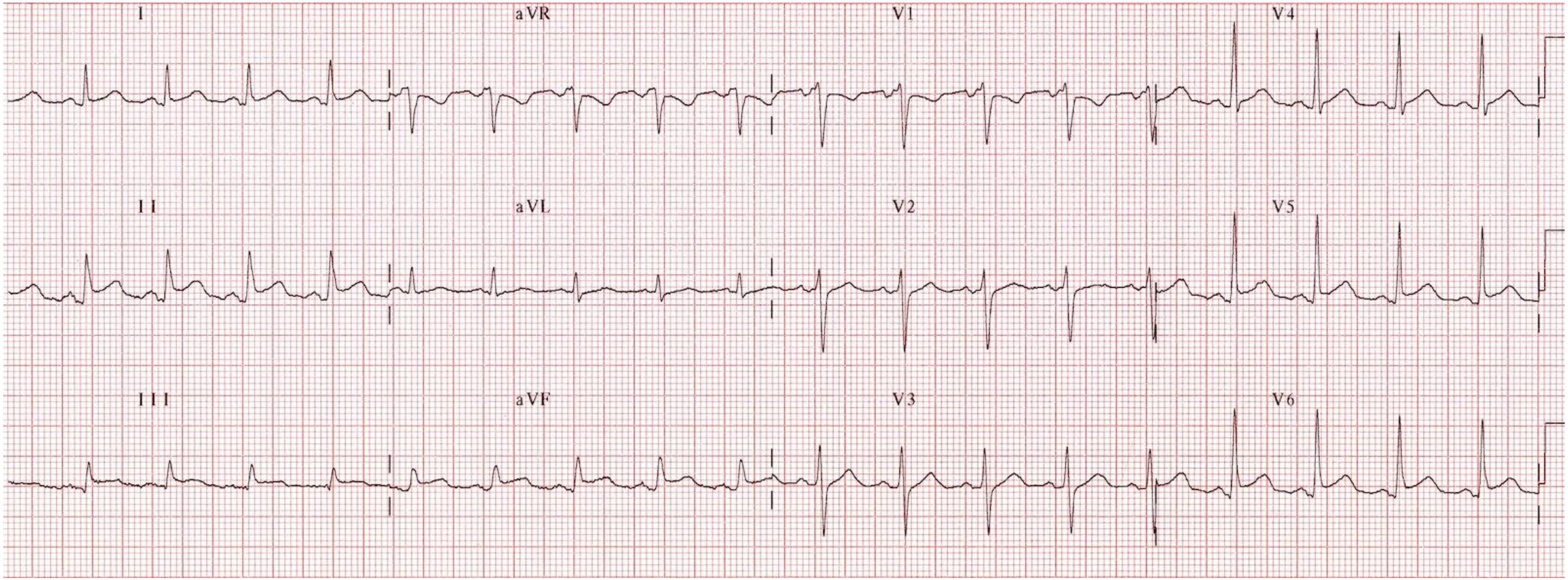


Discrete U waves



"Rollercoaster" U waves

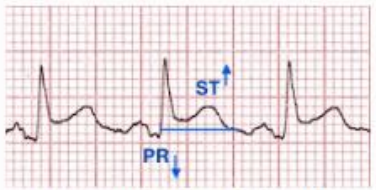
Young adult patient with pleuritic chest pain. Describe the ECG.



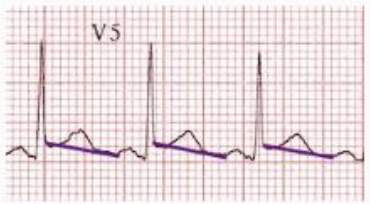
Main Abnormalities

This ECG demonstrates many of the classic features of acute pericarditis:

- Widespread concave ST elevation with PR depression — most notable in I, II, III, aVF, V5-6.
- PR elevation in the inverted leads aVR and V1.
- Downward sloping of the TP segment = "Spodick's sign".
- No reciprocal changes of STEMI.
- ST segment / T wave ratio > 0.25 (favours pericarditis over BER)



ST elevation and PR depression



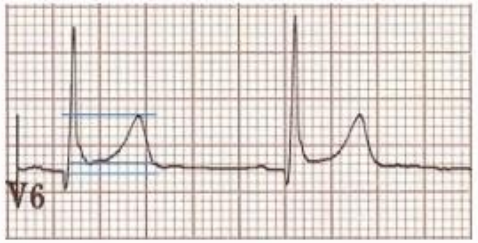
Spodick's sign

Pericarditis versus Benign Early Repolarisation

Pericarditis can be difficult to differentiate from BER as both conditions are associated with concave STE. One useful trick to distinguish between these two entities is to look at the **ST segment / T wave ratio**.

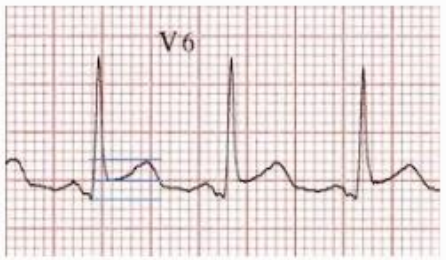
- The amplitude of the STE (from PR segment to J point) is measured in V6 and compared to the T wave amplitude.
- A ratio of > 0.25 suggests pericarditis
- A ratio of < 0.25 suggests BER

Example 1: Benign Early Repolarisation



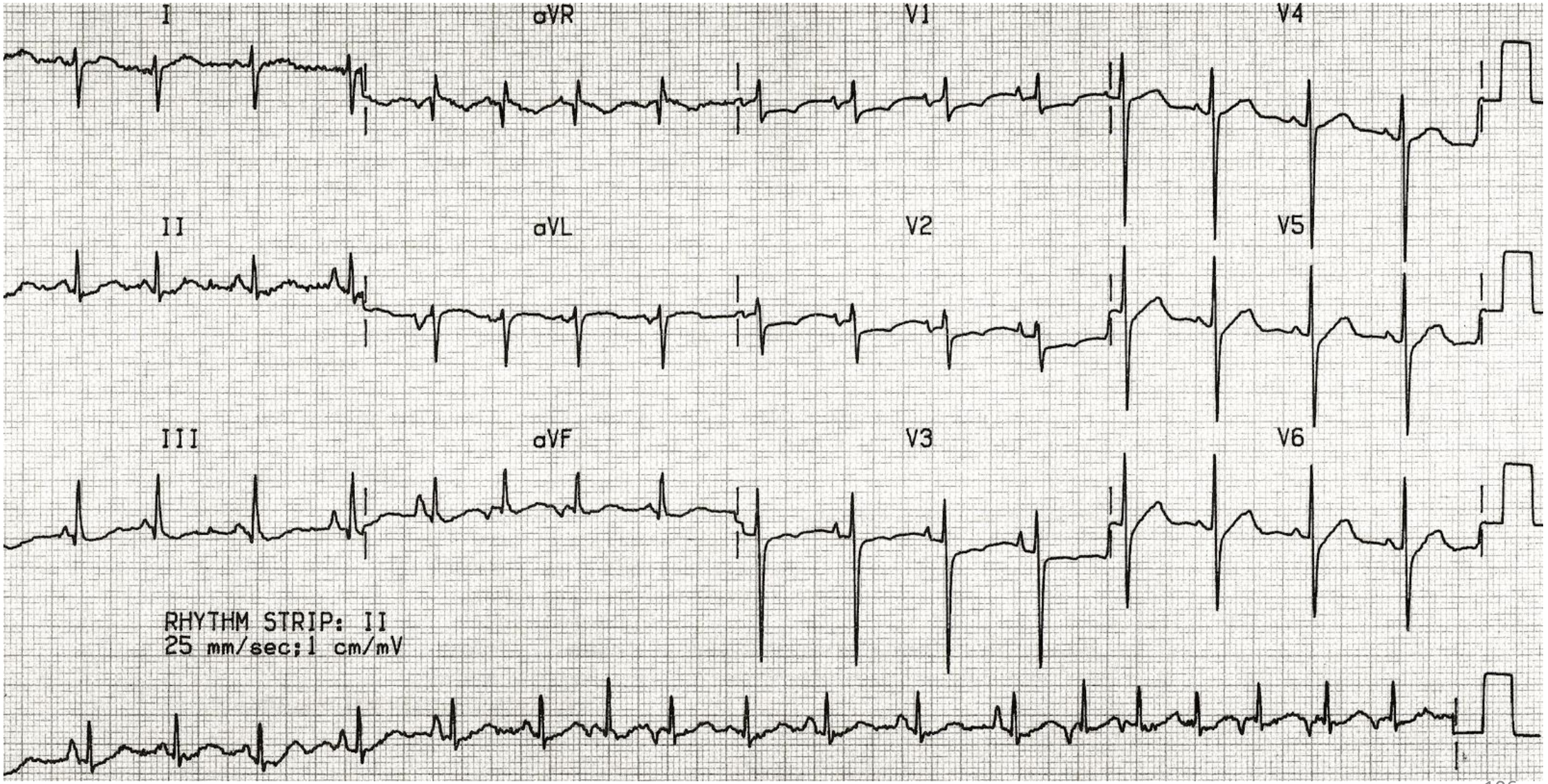
- ST segment height = 1 mm
- T wave height = 6 mm
- ST / T wave ratio = 0.16
- The ST / T wave ratio < 0.25 is consistent with BER.

Example 2: Pericarditis



- ST segment height = 2 mm
- T wave height = 4 mm
- ST / T wave ratio = 0.5
- The ST / T wave ratio > 0.25 is consistent with pericarditis.

75-year old patient presenting with acute dyspnoea, productive cough and wheeze. Describe the ECG.



Main Abnormalities

- Irregularly irregular narrow-complex tachycardia at ~ 110 bpm.
- At least 3 different P wave morphologies seen in the lead II rhythm strip, indicating multiple foci of activity within the atria.
- No flutter or fibrillatory waves — rules out AF or flutter with variable block.
- Evidence of right ventricular hypertrophy — RAD, dominant R wave in V1, deep S wave in V6.

Diagnosis

The combination of…

- Irregular narrow-complex tachycardia (> 100 bpm)
- Multifocal atrial activity (3 or more distinct P wave morphologies)
- No evidence of flutter / AF

… is diagnostic of multifocal atrial tachycardia (MAT).

ECG changes of right ventricular hypertrophy may represent *cor pulmonale* due to COPD.

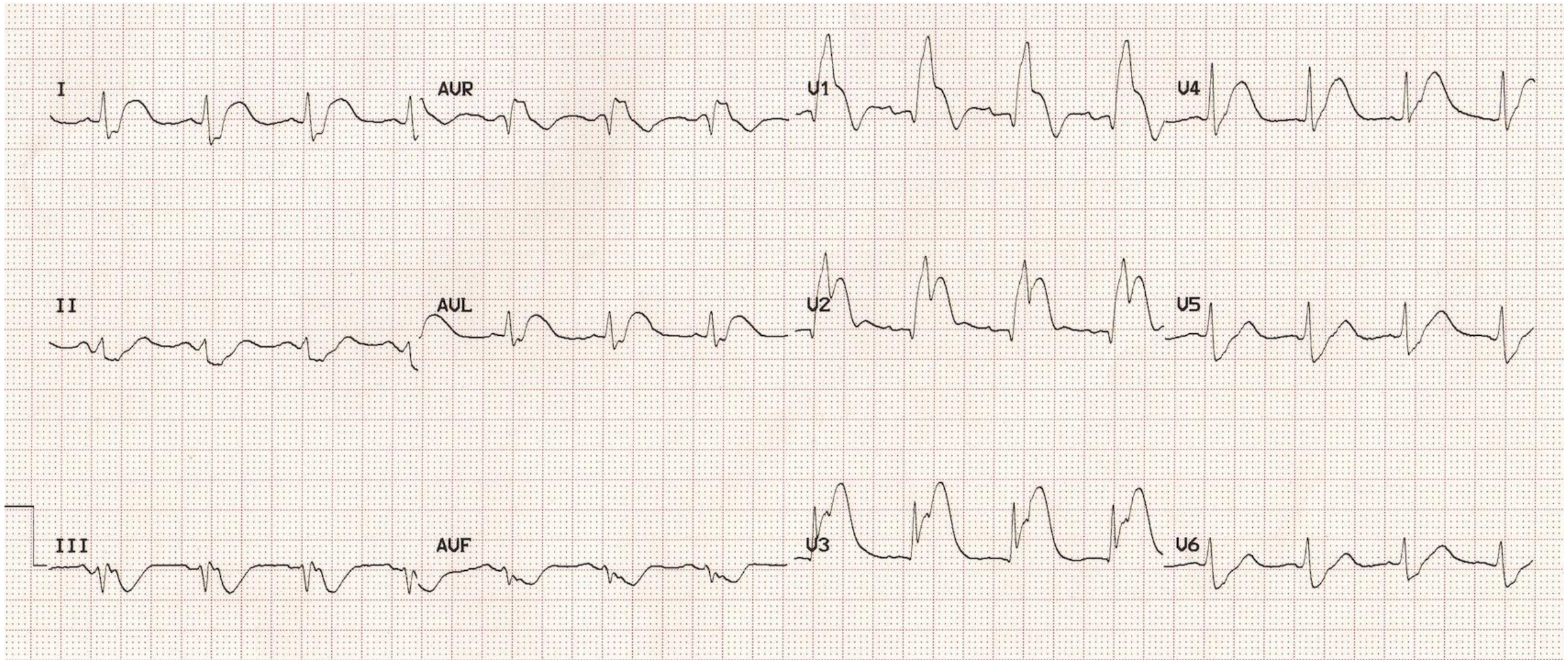
Clinical Significance

MAT typically occurs in patients with severe COPD, as the final common pathway of multiple arrhythmogenic mechanisms:

- Right atrial dilatation (from *cor pulmonale).*
- Increased sympathetic drive — due to hypoxia / hypercarbia.
- Bronchodilators — beta-agonist, theophylline.
- Electrolyte abnormalities — hypokalaemia and hypomagnesaemia (e.g. secondary to diuretics / beta-agonists).

MAT typically resolves with treatment of the underlying COPD exacerbation and correction of any electrolyte abnormalities, although it may evolve into AF or flutter.

Middle-aged patient presenting with chest pain. Describe the ECG.



Main Abnormalities The ECG changes are partially masked by the presence of a right bundle branch block, but there is clear evidence of anteroseptal STEMI:

- Gross ST elevation in V1-3 (~ 5mm in V2).
- Convex ST elevation in I and aVL.
- Reciprocal ST depression and T wave inversion in the inferior leads (II, III, aVF).

Predicting the Site of LAD Occlusion

This ECG demonstrates some markers of a very proximal LAD occlusion, involving the two most proximal branches of the LAD — the first septal branch (S1) and the first diagonal branch (D1).

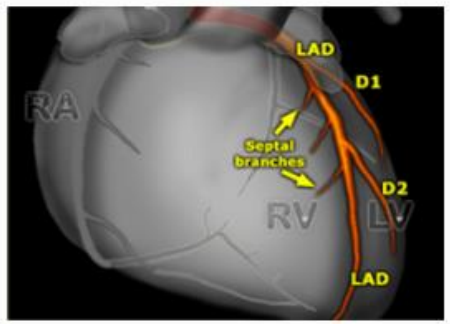


***Diagram courtesy of** The Radiology Assistant*

Signs of occlusion proximal to S1

Signs of basal septal involvement:

- New RBBB — occurs due to septal infarction
- ST elevation in V1 > 2.5 mm
- ST elevation in aVR
- ST depression in V5

Signs of occlusion proximal to D1

Signs of high lateral involvement:

- ST elevation in aVL
- Inferior reciprocal ST depression > 1 mm

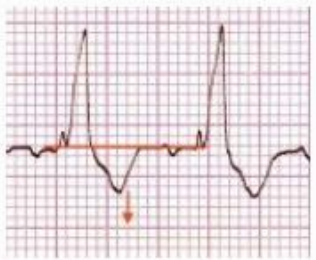
This patient arrived by ambulance following an out-of-hospital VF arrest and was taken straight to the cath lab where he was found to have a complete ostial occlusion of his LAD.

A Common Pitfall

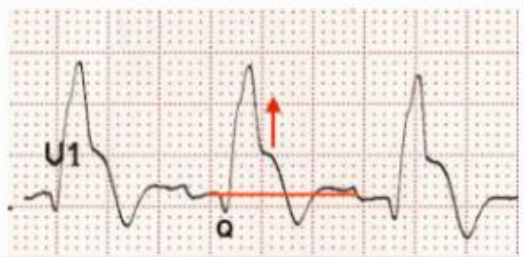
This STEMI pattern is occasionally missed, when clinicians erroneously attribute the ST segment changes in V1-3 to RBBB alone.

However, the two patterns are quite different:

- Typical RBBB will have ST depression and TWI in V1-3.
- Superimposed septal STEMI will lead to ST elevation, Q wave formation, loss of the initial R wave and inversion of only the terminal portion of the T wave.

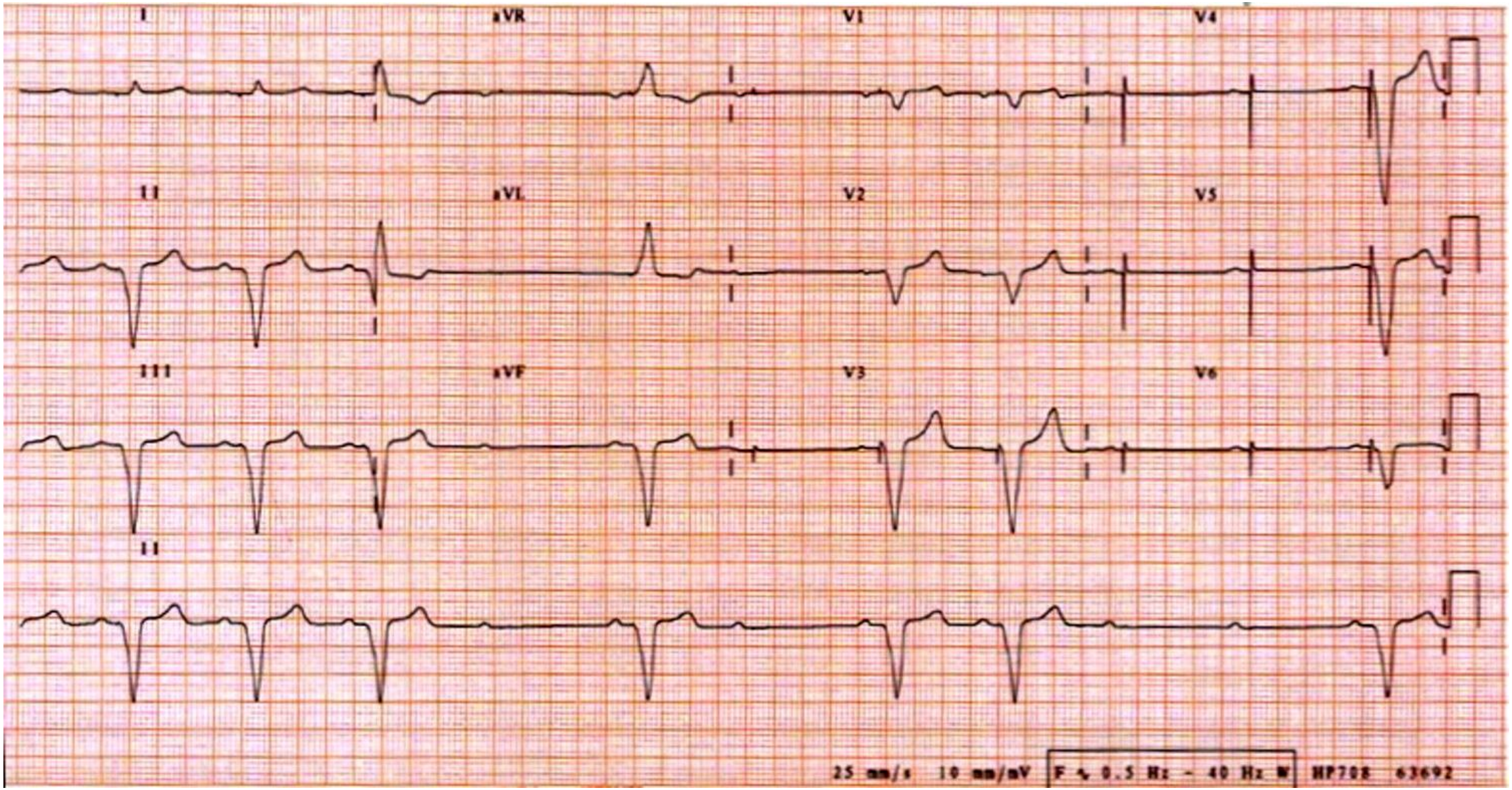


Typical RBBB



RBBB + STEMI

80-year old patient presenting with syncope. Describe the ECG.



On first glance this ECG could easily be mistaken for an example of Mobitz II AV block — there are intermittent non-conducted P waves with a constant PR interval. However, regular pacing spikes can be seen following the P waves in leads V3-6.

This is an example of pacemaker malfunction, with intermittent **failure to capture**:

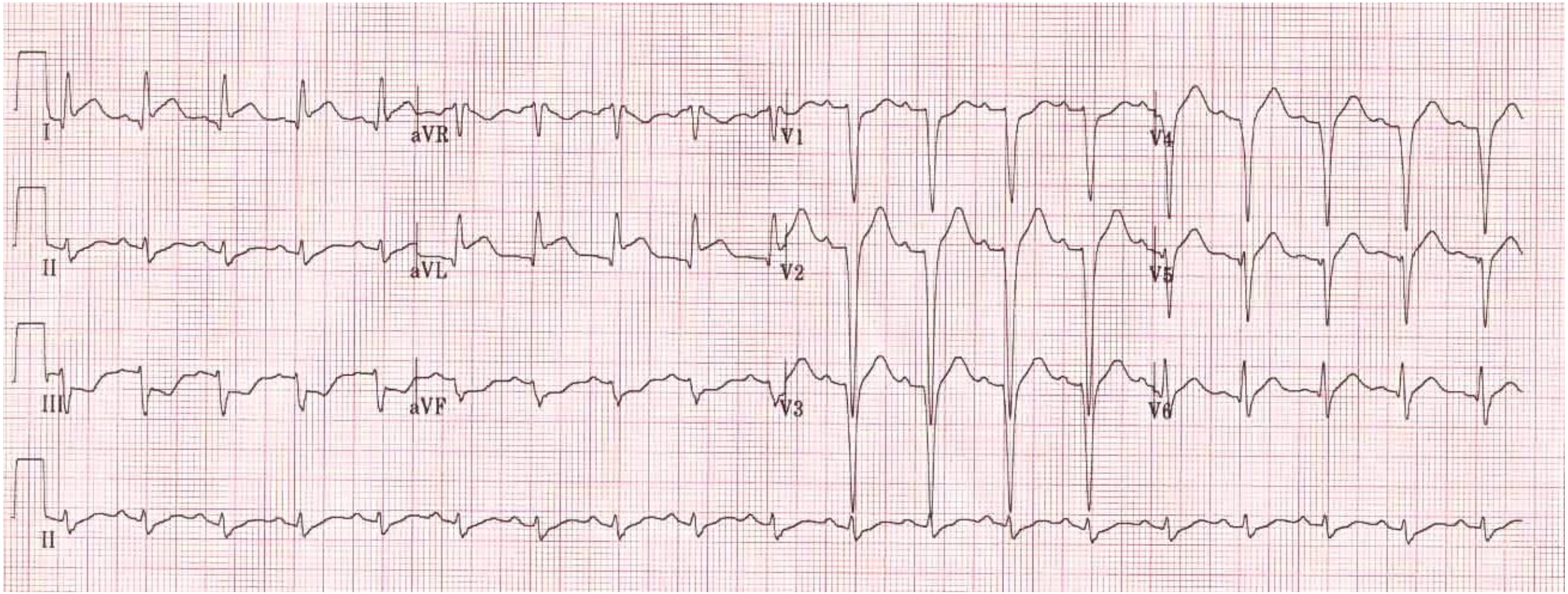
- Regular P waves are seen at ~65 bpm.
- Each P wave is following by a pacing spike (best seen in V3-6, subtle pacing spikes also present in I, aVR, V1). This indicates that atrial sensing is intact. *NB. Pacing spikes will typically not be seen in all 12 leads.*
- Some of the pacing spikes are followed by typical ventricular-paced complexes. The LBBB morphology indicates that the pacing lead is in the right ventricle — the heart depolarises from right to left in the same way as LBBB. Also note the negative concordance in V1-6 (all QRS complexes are negative). This is often quoted as a feature of ventricular tachycardia, but simply indicates that the rhythm is arising from the anterior surface of the right ventricle — the heart is depolarising *away* from the V1-6 electrodes.
- Several of the P waves / pacing spikes are not followed by QRS complexes, producing a ventricular rate of ~ 40 bpm.
- Quite worryingly, there does not seem to be any native ventricular activity kicking in when the heart rate drops. The second half of the rhythm strip shows two sequential non-conducted P waves with no evidence of any escape rhythm. This suggests the presence of underlying complete heart block with inadequate escape mechanisms and significant risk of ventricular standstill.

Failure to Capture

The problem here is failure of the pacemaker to "capture" (depolarise) the ventricular myocardium. Causes of this include:

- Pacemaker lead fracture or migration (e.g. due to Twiddler's syndrome).
- Refractory myocardium — due to electrolyte abnormality (esp. hyperkalaemia) or myocardial ischaemia.

Middle-aged patient presenting with chest pain. Describe the ECG.



Main Abnormalities This ECG is a good example of high lateral STEMI:

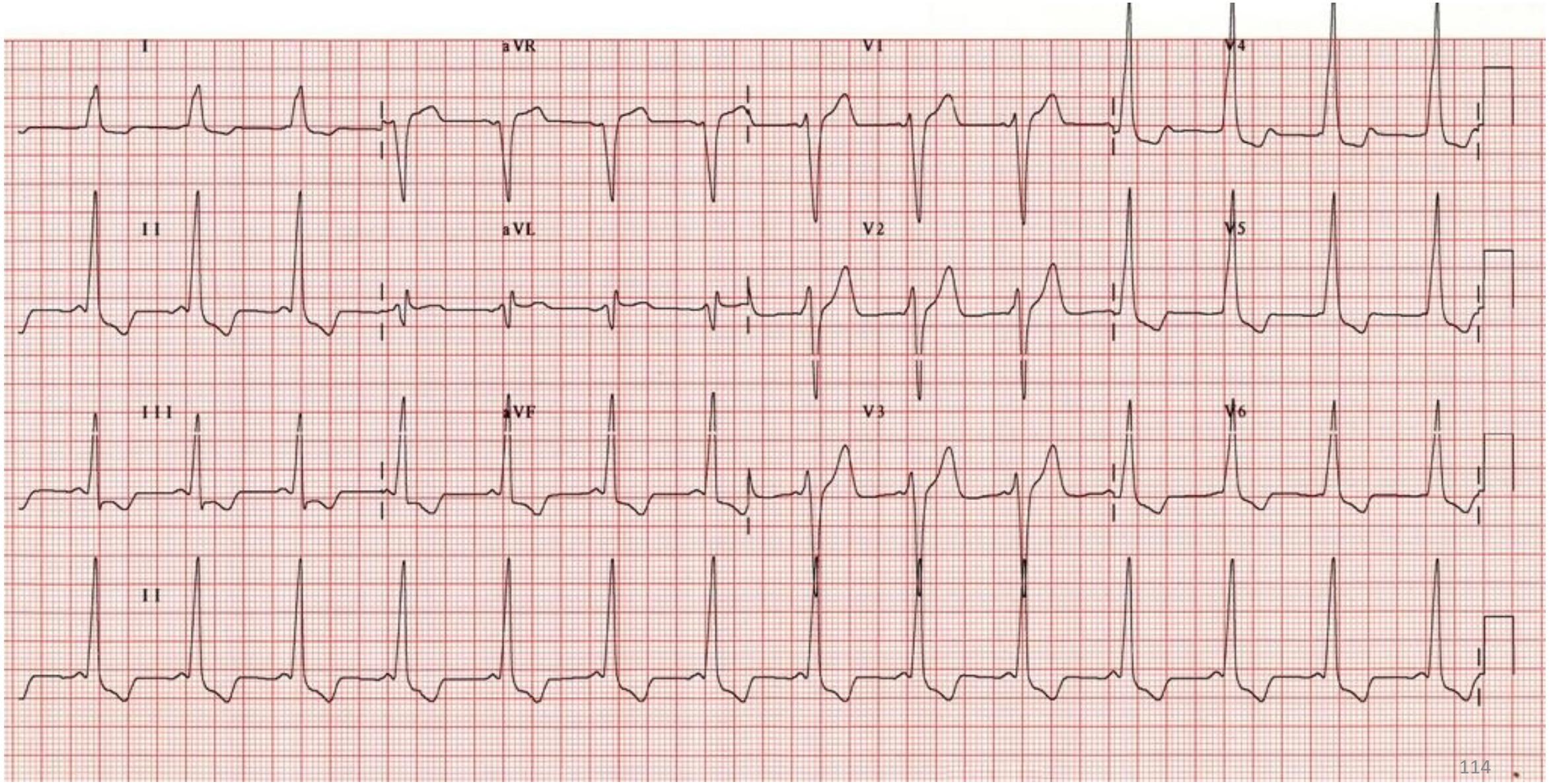
- ST elevation is confined primarily to the high lateral leads I and aVL.
- There is reciprocal ST depression in the inferior leads II, III and aVF.
- The deep Q waves and poor R wave progression in V1-4 suggest prior anteroseptal infarction or dilated cardiomyopathy.

High lateral STEMI is classically associated with occlusion of the **first diagonal branch (D1) of the LAD**, but may also occur with occlusion of the obtuse marginal branch (OM) of the circumflex artery, or the ramus intermedius.

Clinical Pearls

High lateral STEMI may be extremely subtle. Sometimes the only clue is the presence of new inferior ST depression. Such localised ST depression should always be considered to be reciprocal change rather than “inferior ischaemia” as *subendocardial ischaemia does not localise*.

15-year old patient presenting with rapid palpitations and dizziness. Symptoms resolved en route to hospital. Describe the ECG.



Main Abnormalities

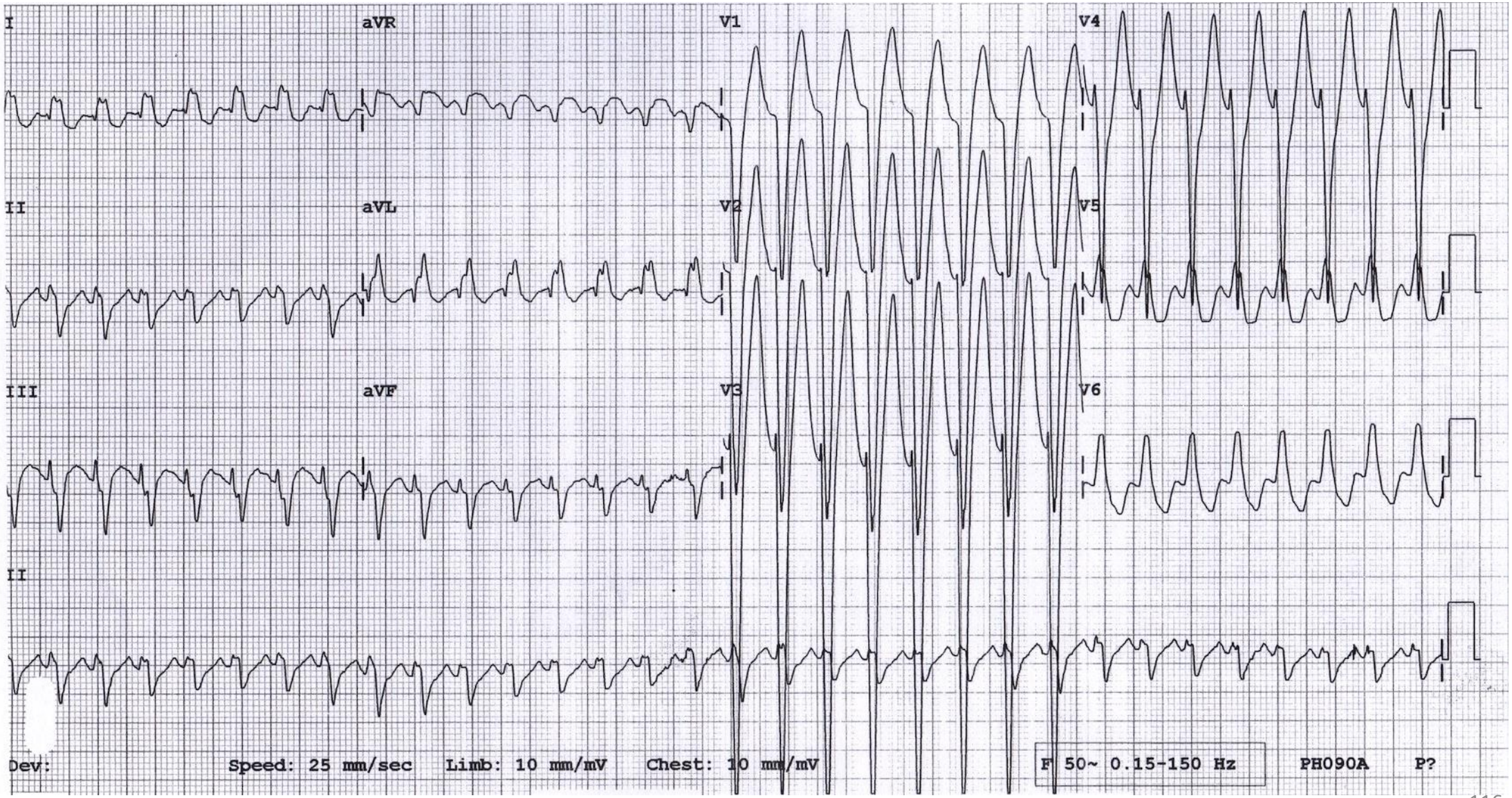
This ECG is diagnostic of the Wolff-Parkinson-White (WPW) syndrome:

- Sinus rhythm with a very short PR interval (< 120ms).
- Broad QRS complexes.
- Delta waves = slurred upstroke to the QRS.

Other Features:

- Dominant S wave in V1 — this "type B" pattern indicates a right-sided accessory pathway.
- Tall R waves and inverted T waves mimic the appearance of LVH — this is an electrical phenomenon due to WPW and not a sign of ventricular hypertrophy.
- ST segments and T waves show typical "discordant" changes — they point in in the opposite direction to the QRS complex, similar to LBBB.

15-year old patient presenting with rapid palpitations and dizziness. Symptoms recur in ED. Describe the ECG.



Main Abnormalities

- Very rapid, regular broad-complex tachycardia (~ 200 bpm).
- LBBB morphology (dominant S wave in V1).
- No clear atrial activity — no flutter waves or fibrillatory waves.
- No obvious diagnostic features for VT.

Differential Diagnosis

In a patient presenting with a regular broad-complex tachycardia and no evidence of atrial activity, the main diagnostic considerations are:

- Ventricular tachycardia.
- SVT with aberrant conduction due to bundle brach block.
- SVT with aberrant conduction due to WPW.

Although diagnostic criteria exist to aid in differentiation of these rhythms, none of them have 100% sensitivity or specificity — leading many authors to recommend treating as VT if uncertain.

However, clinical context is everything…

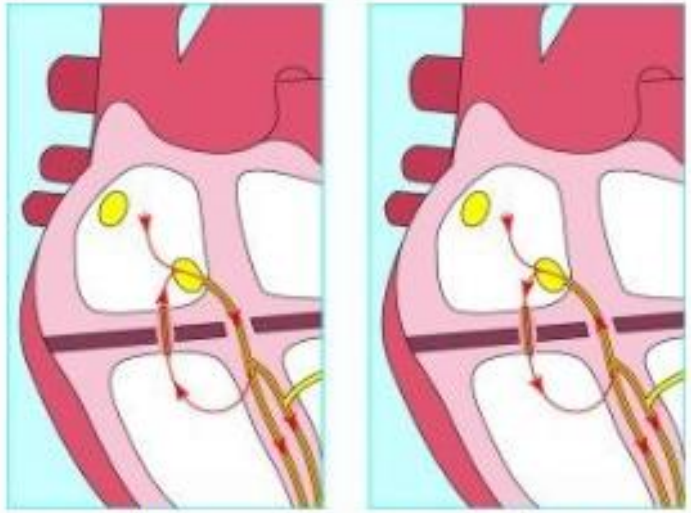
This patient has two strong indicators of SVT with aberrancy:

- Young age — the vast majority of BCTs in children are SVT with aberrancy.
- Evidence of WPW on previous ECGs.

Diagnosis

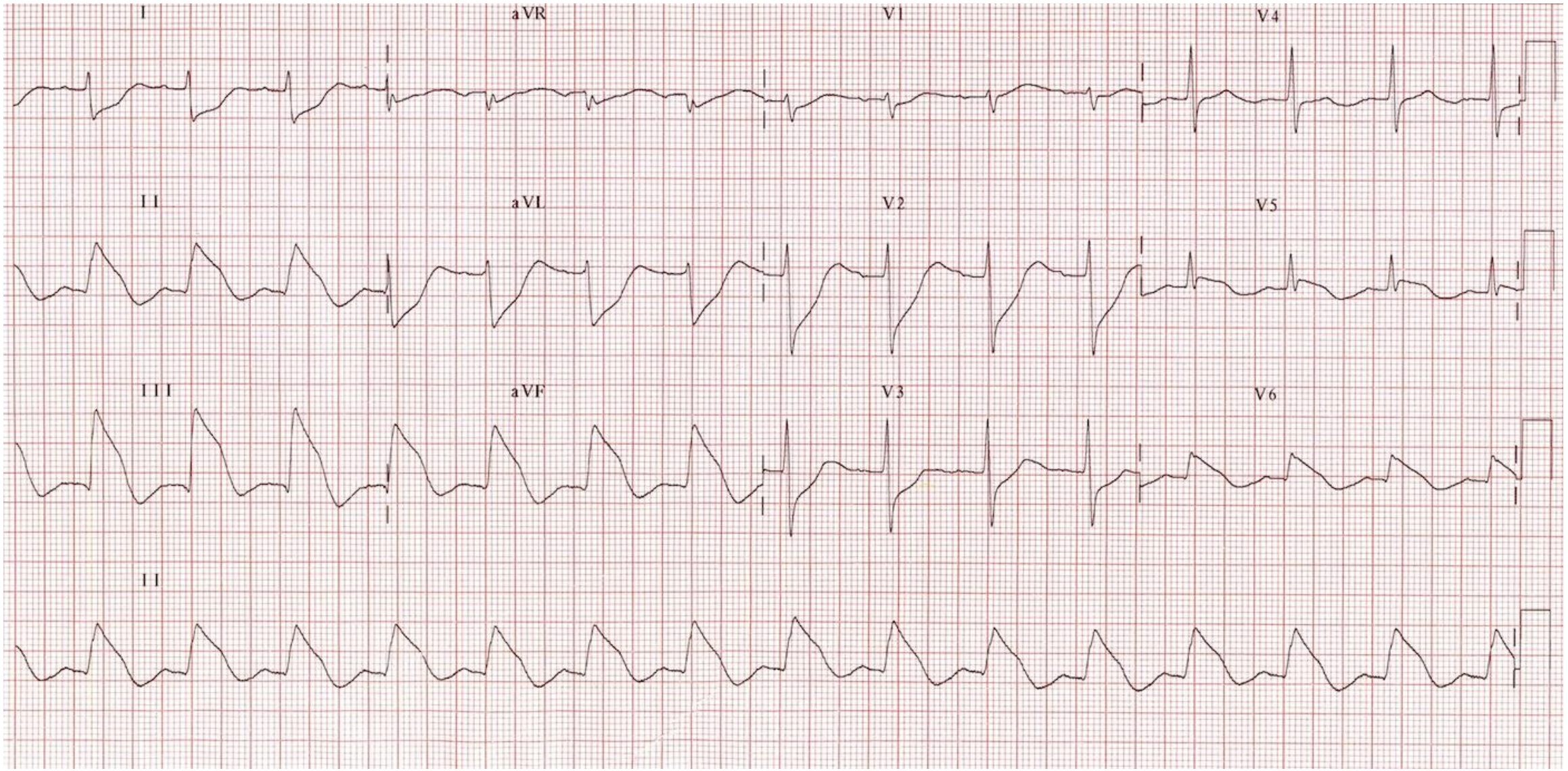
This is an example of antidromic AV reentry tachycardia — a reentrant SVT seen in WPW where the impulse travels from atria to ventricles via the accessory pathway, recycling backwards through the AV node (hence “antidromic”). Activation of the ventricles via the accessory pathway produces a broad complex that may be indistinguishable from VT. This is in comparison to orthodromic SVT, where the impulse travels forwards through the AV node producing a normal-looking, narrow QRS.

This patient reverted back to sinus rhythm with vagal maneouvres. The WPW pattern was once again visible on his sinus rhythm ECG.



AVRT with orthodromic (left) and antidromic (right) AV nodal conduction

Elderly patient presenting with chest pain. BP 80/50. Describe the ECG.



This ECG shows extensive infero-postero-lateral STEMI with “tombstone” morphology:

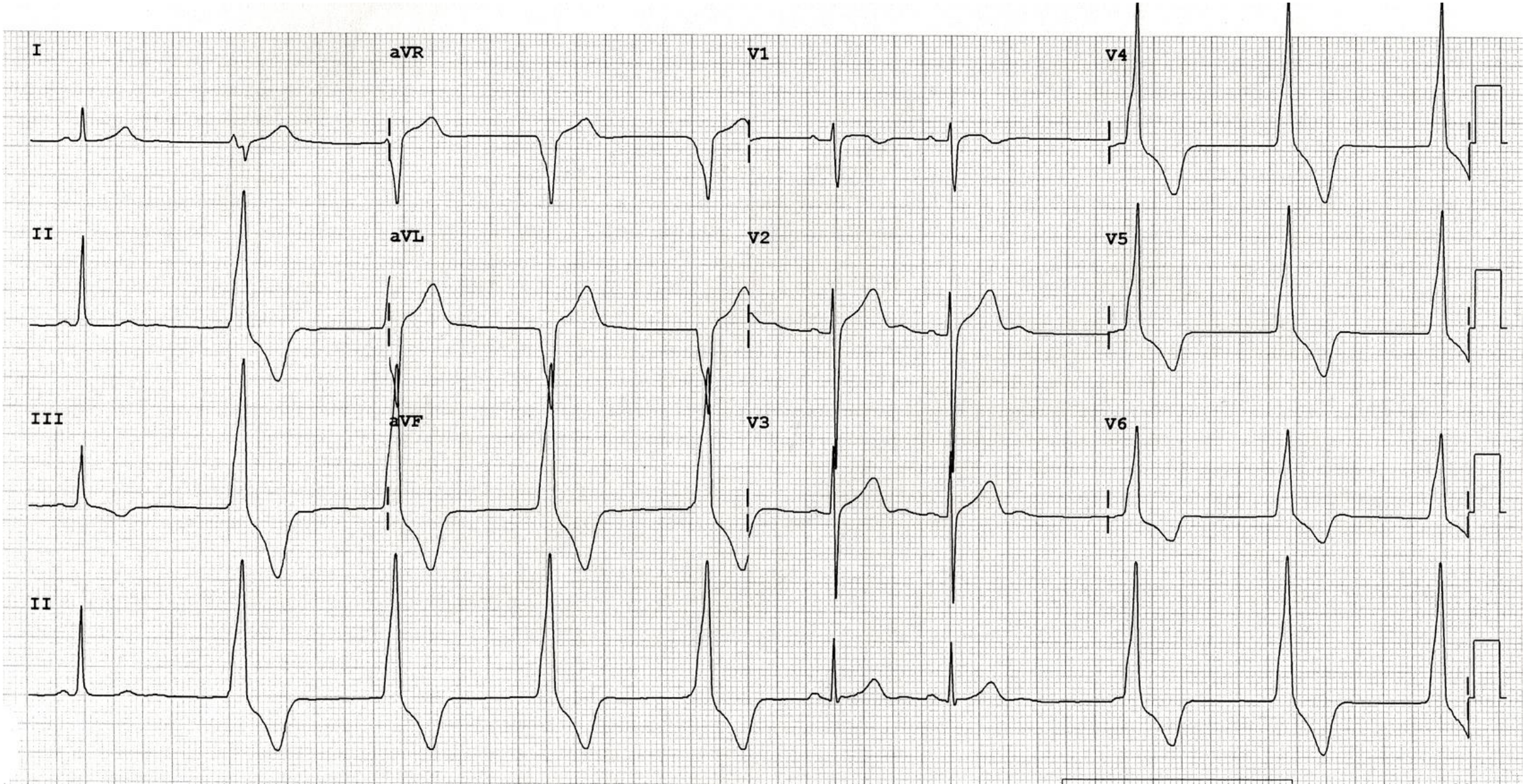
- Gross ST elevation in II, III, aVF consistent with inferior infarction.
- Reciprocal ST depression seen in I, aVL.
- ST elevation in V5-6 indicating lateral wall involvement.
- ST depression in V2 is suggestive of associated posterior wall infarction — the morphology is the exact inverse of the ST elevation in the inferior leads.

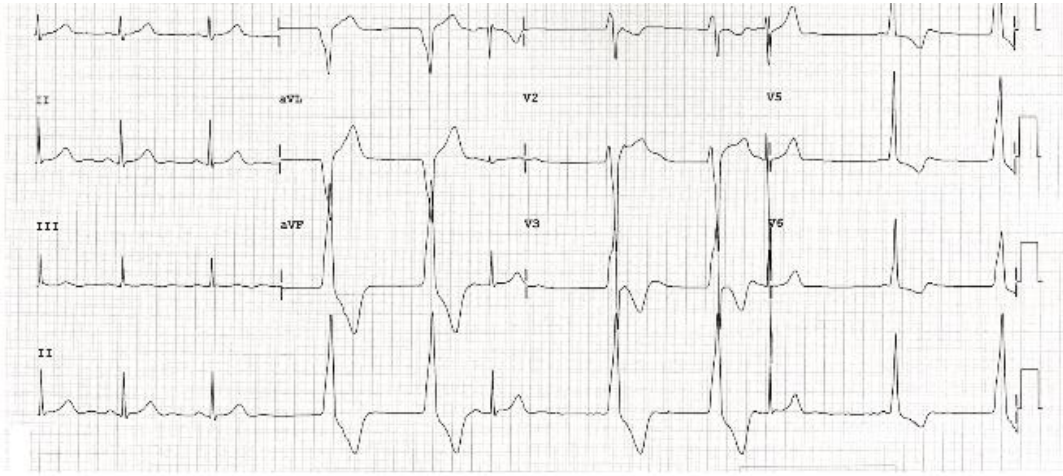
There are some additional features suggest of right ventricular infarction:

- STE in lead III > lead II.
- Deep ST depression in V2 with an isoelectric ST segment in V1.

This is a huge infarct with a likely poor prognosis. Hypotension may be due to nitrate therapy causing exaggerated preload reduction in the context of RV infarction, or may simply reflect the large infarct size with development of early cardiogenic shock.

36-year old old athlete, asymptomatic. Describe the ECG.





Reveal Answer

Main Abnormalities

There are two competing rhythms at similar rates (= isorhythmic AV dissociation):

- Accelerated idioventricular rhythm (AIVR) — broad-complex ventricular rhythm at around 60 bpm.
- Sinus bradycardia with sinus arrhythmia — the sinus rate varies from 70 bpm down to < 50 bpm.

The QRS morphology varies depending on which focus is capturing the ventricles at any given moment.

Other Findings

Specific ECG findings that confirm the presence of simultaneous sinus and ventricular rhythms are:

- Capture beats — sinus beats that intermittently wrest control of the rhythm producing narrow complexes.
- Fusion beats (seen in the second ECG) — these are intermediate width complexes that occur when sinus and ventricular beats coincide.

- Capture beats — sinus beats that intermittently wrest control of the rhythm producing narrow complexes.
- Fusion beats (seen in the second ECG) — these are intermediate width complexes that occur when sinus and ventricular beats coincide.

NB. Fusion and capture beats are often discussed in the context of VT. They are not specific to VT, but rather can be seen with any ventricular rhythm, including paced rhythms and AIVR.

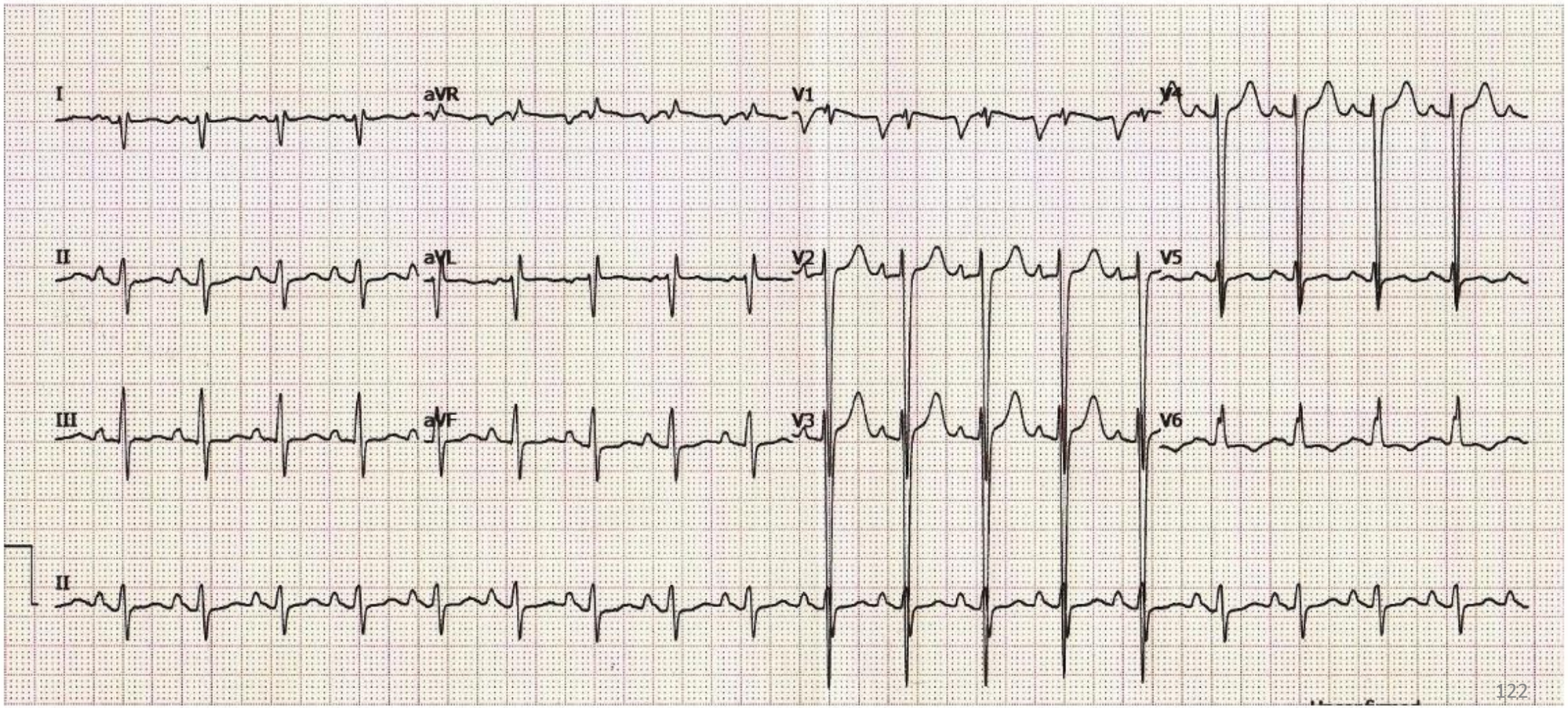
Explanation

- Competing sinus and ventricular pacemakers are present. There is underlying sinus arrhythmia, with sinus capture occurring when the sinus rate exceeds the idioventricular rate.
- This patient was a fit athlete with presumably high resting vagal tone that suppressed his sinus node output and allowed an ectopic ventricular pacemaker to emerge.

Definitions

- **Accelerated idioventricular rhythm**
  An ectopic ventricular rhythm consisting of three or more ventricular complexes occurring at a rate of 50-110 bpm. The rate differentiates AIVR from ventricular escape rhythms (rate < 50 bpm) and VT (> 110bpm).
- **Isorhythmic AV dissociation**
  AV dissociation with sinus and ventricular complexes occurring at similar rates, unlike 3rddegree heart block where the atrial rate is usually faster than the ventricular rate.
  Isorhythmic AV dissociation is usually due to functional block at the AV node from retrogradely conducted ventricular impulses (“interference-dissociation”), which leaves the AV node refractory to the anterograde sinus impulses.

70-year old patient presenting with acute pulmonary oedema. Describe the ECG

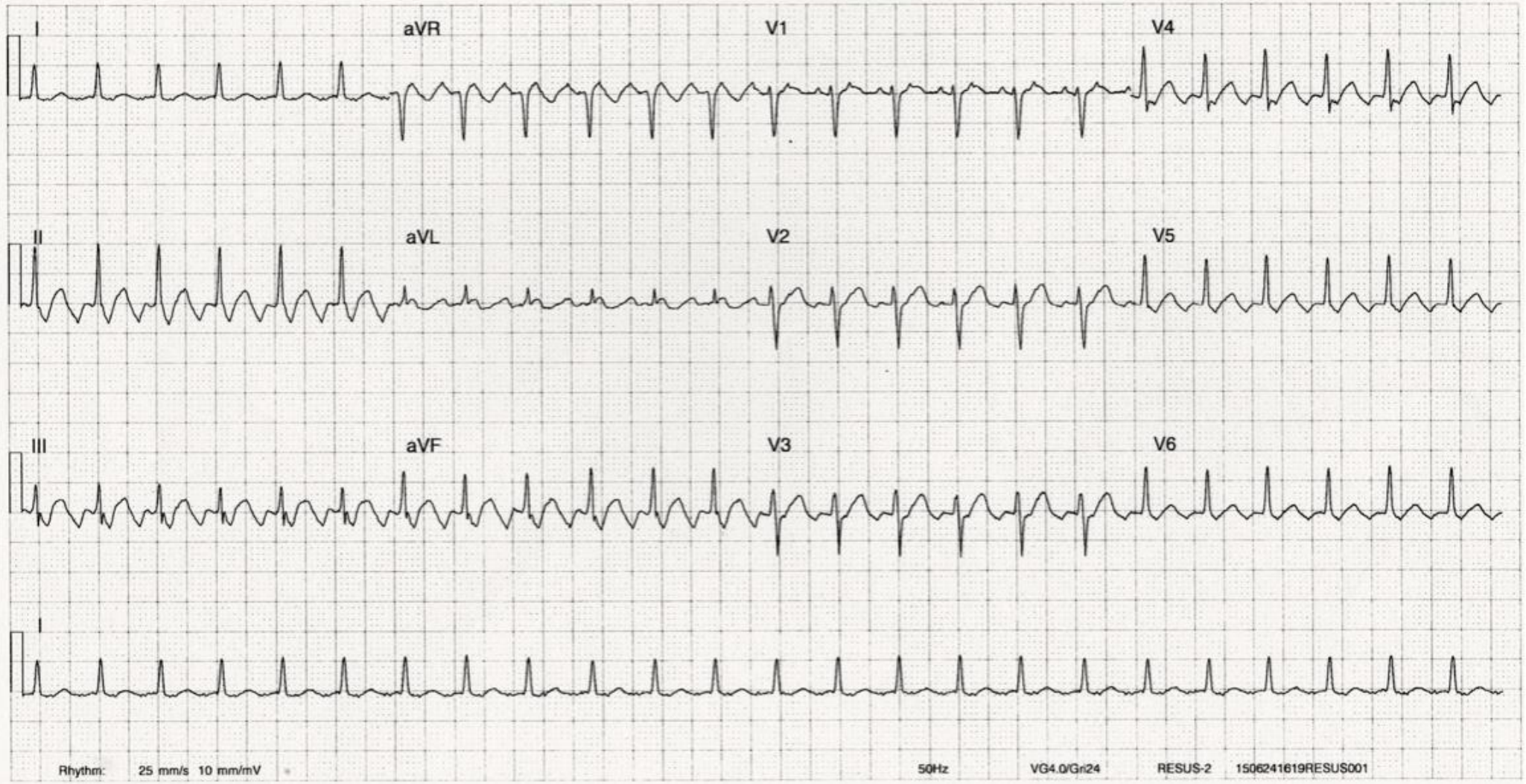


This is an ECG example of dilated cardiomyopathy demonstrating signs of enlargement of all four cardiac chambers:

- There is marked LVH with very deep S waves in V2-4.
- Right axis deviation suggests associated RV enlargement (= biventricular enlargement).
- Evidence of left atrial enlargement (deep, wide terminal portion of the P wave in V1).
- Peaked P waves in lead II suggestive of right atrial enlargement (~ 2.5mm in height).

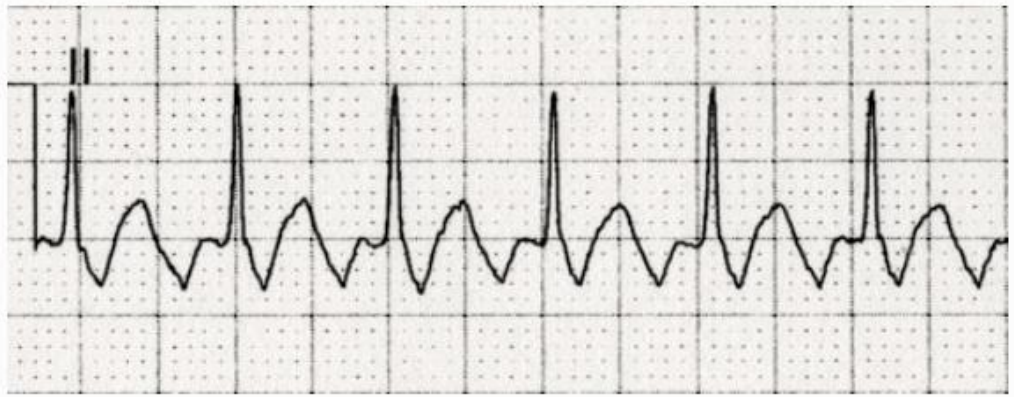
This patient had four-chamber dilatation on echocardiography with severe congestive cardiac failure (awaiting cardiac transplantation).

75-year old patient presenting with palpitations. Describe the ECG

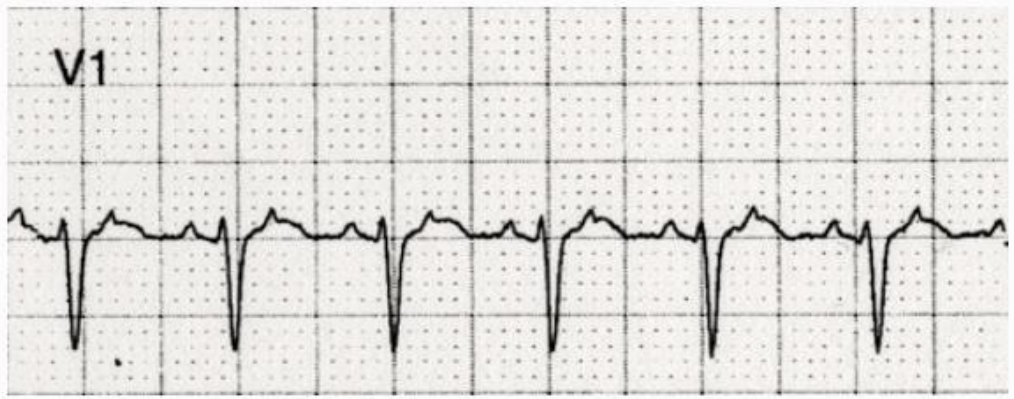


This is a typical example of atrial flutter with 2:1 AV block

- Narrow complex tachycardia at 150 bpm.
- Sawtooth flutter waves are seen in the inferior leads II, III, aVF.
- Upright flutter waves in V1 appear either as pseudo-P waves or as notches in the T wave.
- There is a clear 2:1 relationship between the flutter waves (300 bpm) and QRS complexes (150 bpm).



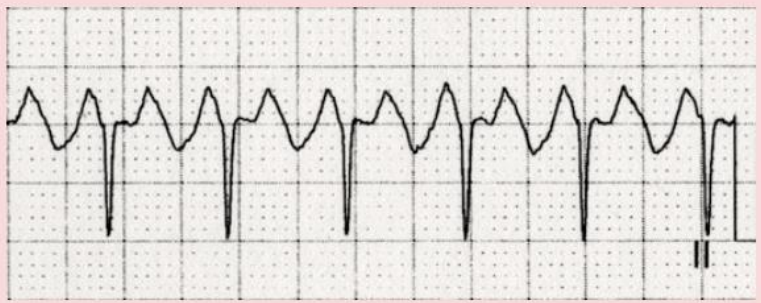
Inverted flutter waves in lead II.



Upright flutter waves in V1.

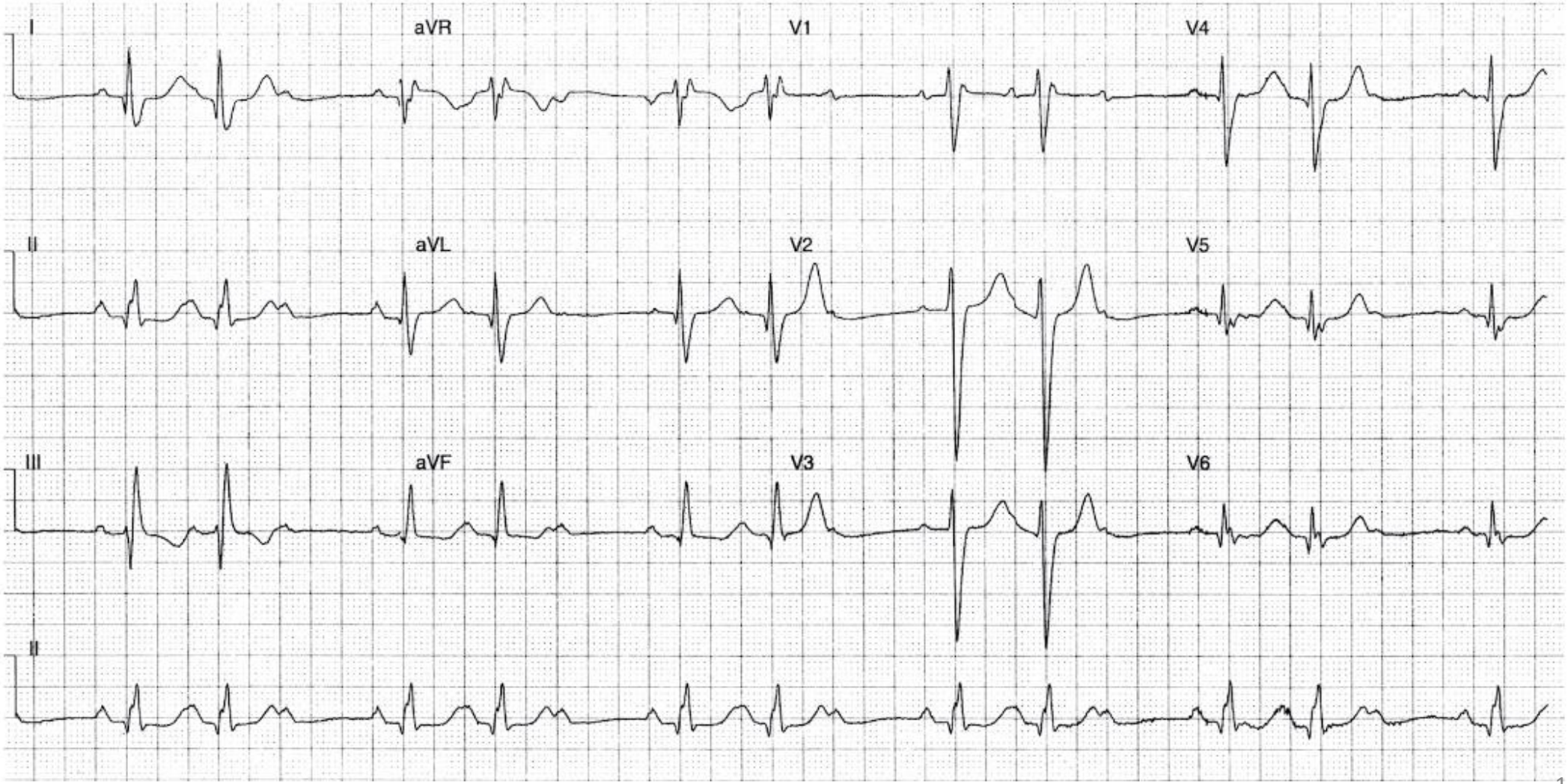
Tips for Spotting Atrial Flutter

- Suspect flutter with 2:1 block in any patient with a regular NCT at 150 bpm.
- Scrutinise leads II and V1 for flutter waves.
- Flutter waves are typically sawtooth in lead II and resemble P waves in V1.
- Try turning the ECG upside down — this can make the flutter waves in lead II easier to see.



Inverting the ECG makes flutter waves in lead II easier to see

Middle-aged patient presenting with syncope. Describe the ECG

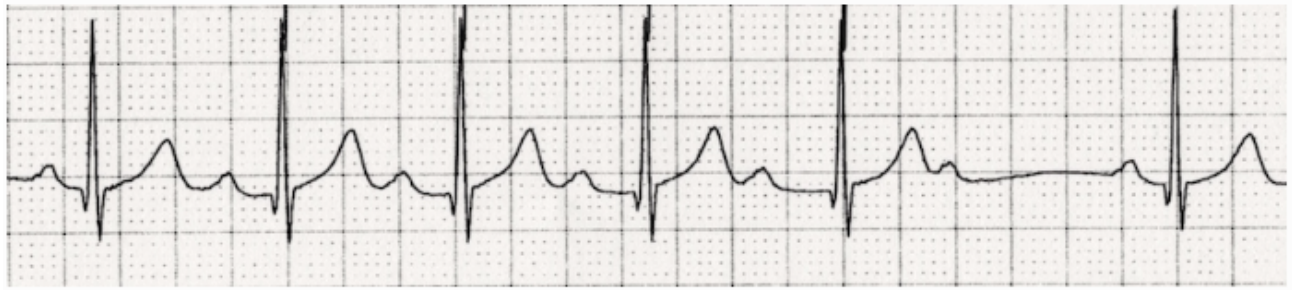


Main Abnormalities

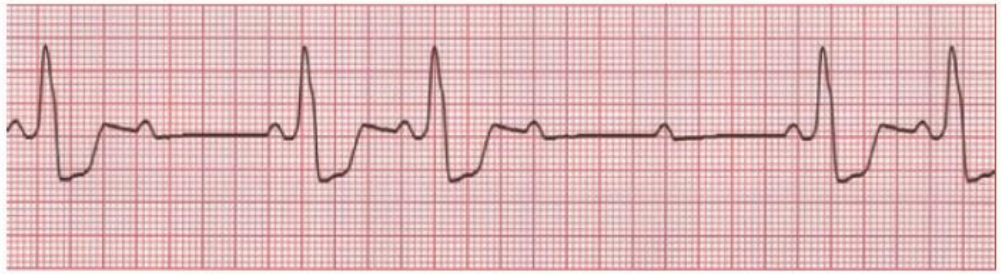
Sinus rhythm with evidence of 2nd degree AV block

- The QRS complexes cluster in groups separated by non-conducted P waves.
- There is a 3:2 relationship between the P waves and QRS complexes.
- With fixed ratio blocks like this, it can sometimes be difficult to distinguish between Mobitz I and Mobitz II conduction, as there are not always enough successive PR intervals to judge whether progressive PR prolongation is occurring.
- The PR interval seems relatively constant between the first and second beats of each group, suggesting Mobitz II conduction.
- However, the clustering of QRS complexes into repeating groups with P:QRS ratios of 3:2, 4:3, 5:4, etc. is a characteristic feature of Mobitz I.
- There is an atypical RBBB — typical RSR' pattern in V1 with slurred S wave in lead I, but atypical morphology elsewhere.

Pragmatically, I wouldn't get too focussed on trying to work out the rhythm from a single tracing. I would just record multiple ECGs aiming to capture any longer P:QRS cycles to assess whether successive PR prolongation is present (Mobitz I) or absent (Mobitz II).



Progressive PR prolongation with Mobitz I



Constant PR interval with Mobitz II

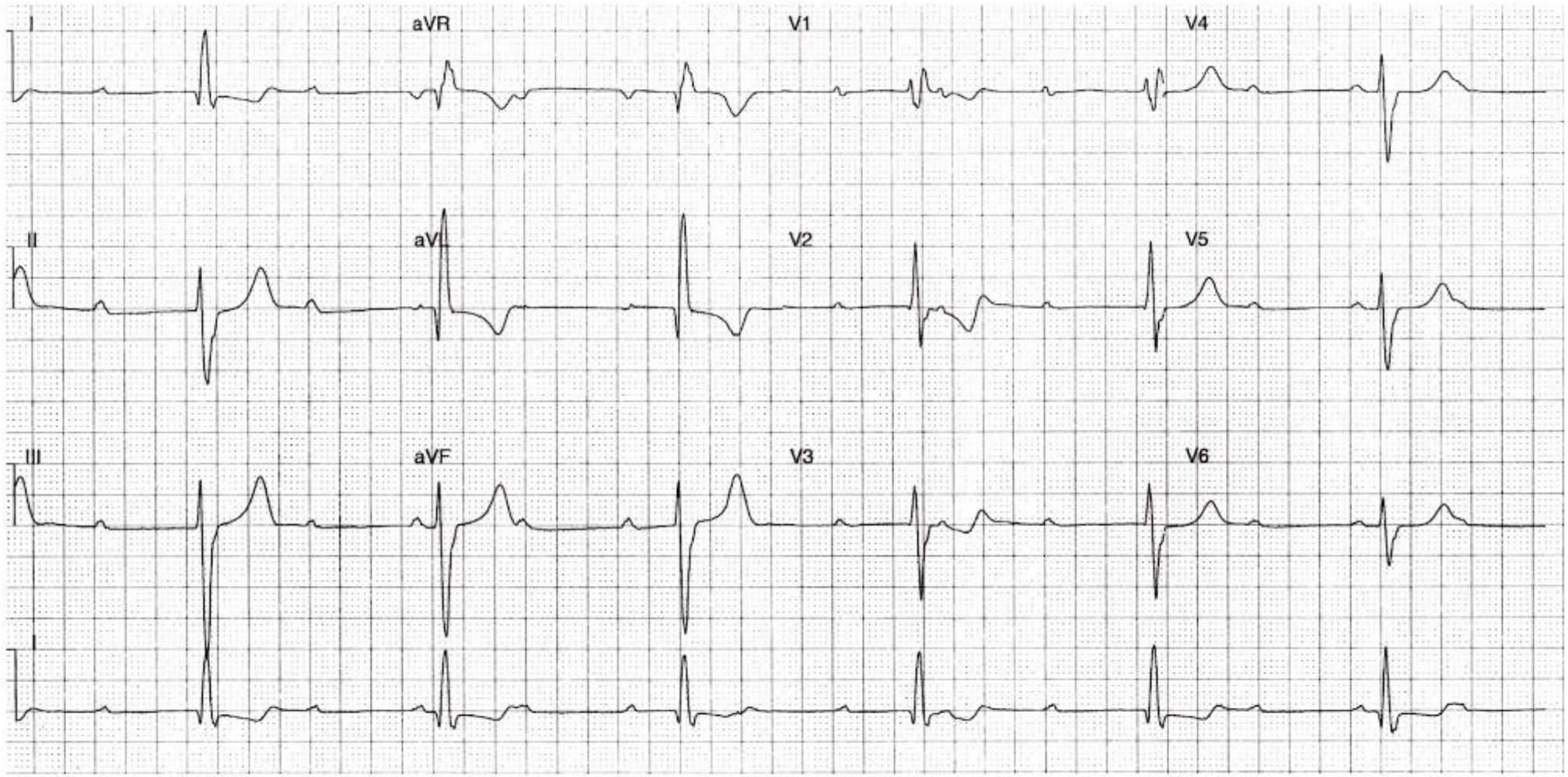
Another clue to aid differentiation is QRS width:

- Mobitz I is typically due to block at the level of the AV node — hence QRS complexes are usually (but not always) narrow.
- Mobitz II typically occurs on a background of widespread conducting system disease (LBBB or bifascicular block). The block is typically at the level of the fascicles — hence QRS complexes are usually (but not always) broad.

Clinical Pearls

- There is some prognostic value in differentiating between Mobitz I and II — Patients with Mobitz II have a much higher rate of complete heart block and are more likely to require a permanent pacemaker.
- However, in the acute situation it is more important to *look at the patient* rather than to fixate on the ECG.
- Treatment decisions such as whether to commence temporary pacing will be largely determined by the patient's clinical stability.

Middle-aged patient presenting with syncope. Becomes hypotensive in ED (BP 80/50). Describe the ECG



There is sinus rhythm with complete heart block:

- Normal P waves (upright in II, inverted in aVR) are present at a rate of ~ 85 bpm.
- There is no relationship between the P waves and QRS complexes — the PR intervals vary randomly.
- A ventricular escape rhythm is present at ~ 36 bpm.

The broad QRS complexes, RBBB morphology and left axis deviation (resembling trifascicular block) indicate a ventricular escape rhythm arising in the left posterior fascicle. Note how the QRS axis and morphology have changed significantly from the previous ECG.

This patient had complete heart block due to cardiac sarcoidosis.

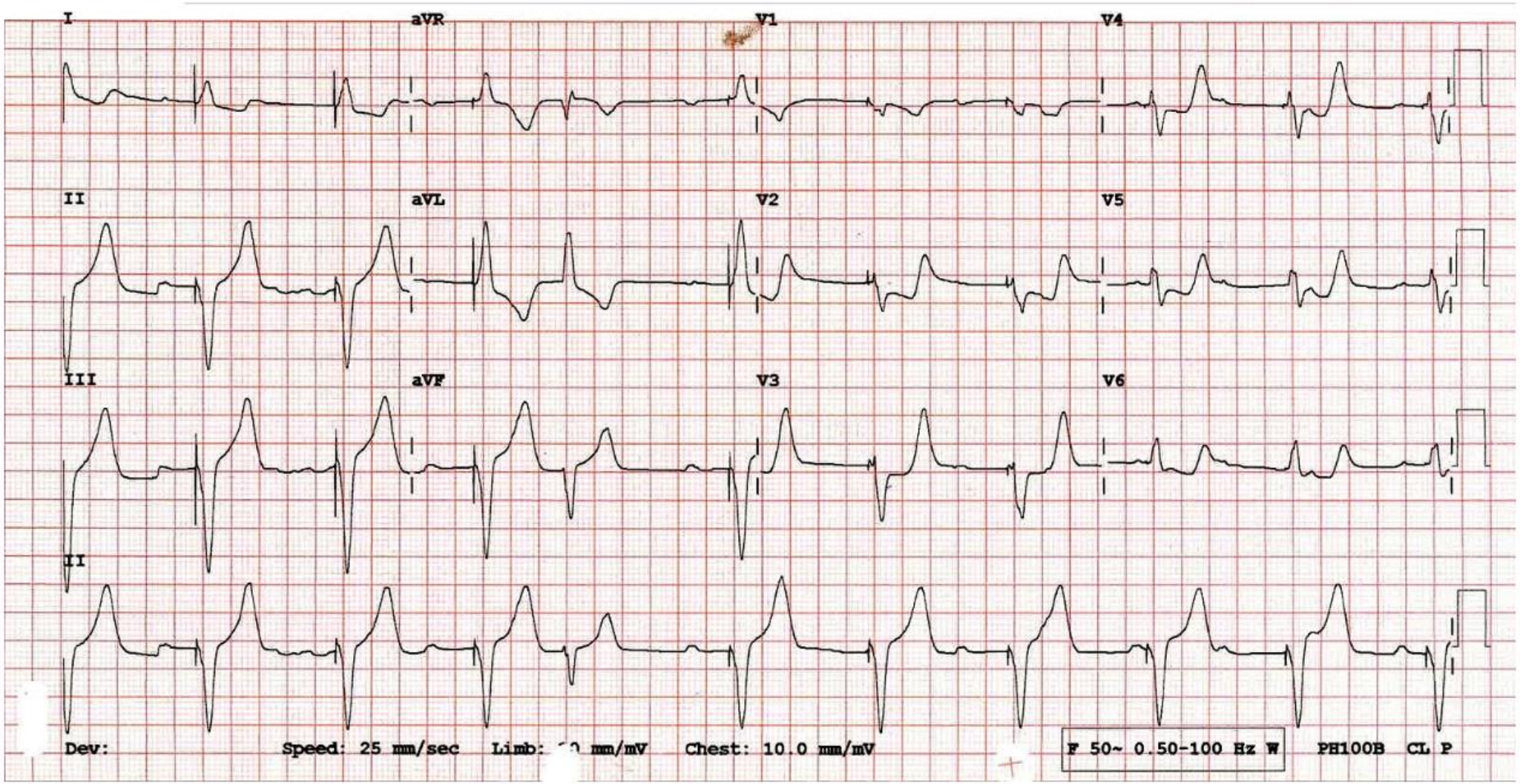
Clinical Pearls

- Sarcoidosis should always be considered as a differential diagnosis in younger patients presenting with complete heart block, particularly if other manifestations of sarcoidosis are present such as bilateral hilar lymphadenopathy or cutaneous lesions (erythema nodosum, lupus pernio).
- One of the most common reversible causes of complete heart block is severe hyperkalaemia — always get an urgent K+ (e.g. run a VBG) on patients presenting with CHB. You look a bit silly inserting an unnecessary pacing wire when you could have corrected the problem with some calcium gluconate!

Causes of Complete Heart Block

- AV nodal blocking drugs (e.g.calcium-channel blockers, beta-blockers, digoxin)
- Severe hyperkalaemia.
- Inferior myocardial infarction — due to increased vagal tone.
- Anterior myocardial infarction — due to septal necrosis.
- Idiopathic fibrosis of the conducting system (Lenegre's or Lev's disease).
- Cardiac surgery (especially surgery occurring close to the septum, e.g. mitral valve repair)
- Infiltrative myocardial disease (amyloidosis, haemochromatosis, sarcoidosis).
- Inflammatory conditions (rheumatic fever, myocarditis, Lyme disease).
- Autoimmune (SLE, systemic sclerosis).

64-year old female presenting with severe chest pain and diaphoresis. Describe the ECG



This ECG shows a ventricular paced rhythm with positive Sgarbossa criteria:

- There is **concordant ST depression** in V2-5. This violates the expected pattern of discordance for a V-paced rhythm and is a marker of superimposed **myocardial infarction**.
- The morphology in V2-5 is reminiscent of posterior STEMI, with horizontal ST depression and prominent upright T waves.
- Multiple non-conducted P waves are seen, indicating the presence of underlying high-grade AV block (probably the indication for pacemaker insertion). However, the fusion complex (beat #5 on rhythm strip) suggests that P waves are occasionally transmitted, arguing against complete heart block.

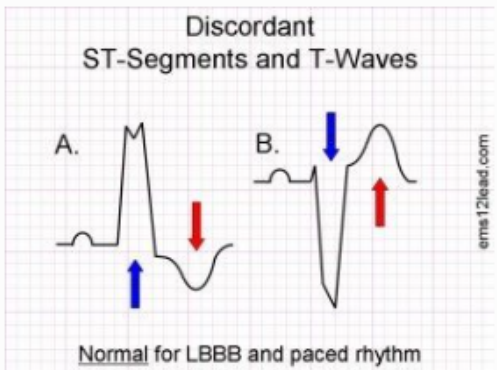
This patient did indeed have an isolated posterior infarction, due to complete occlusion of a posterolateral branch of the RCA. She was successfully treated with PCI.

Sgarbossa Criteria

These criteria allow for detection of myocardial infarction in patients with LBBB and V-paced rhythms (previously thought to be "impossible").

Normal Pattern in LBBB / VPR

- The expected finding in patients with uncomplicated LBBB / V-paced rhythm is **discordance** — i.e. the ST segments and T waves point in the opposite direction to the QRS complex.



Superimposed myocardial infarction is suspected if there is either:

- Loss of the usual pattern of discordance — i.e. **concordant ST changes.**
- **Excessive discordant ST elevation** — i.e. out of proportion to what would be expected for LBBB / paced rhythm.

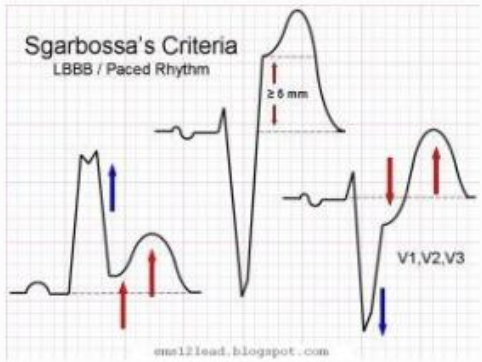


Image reproduced from ECGMedicalTraining.com with permission.

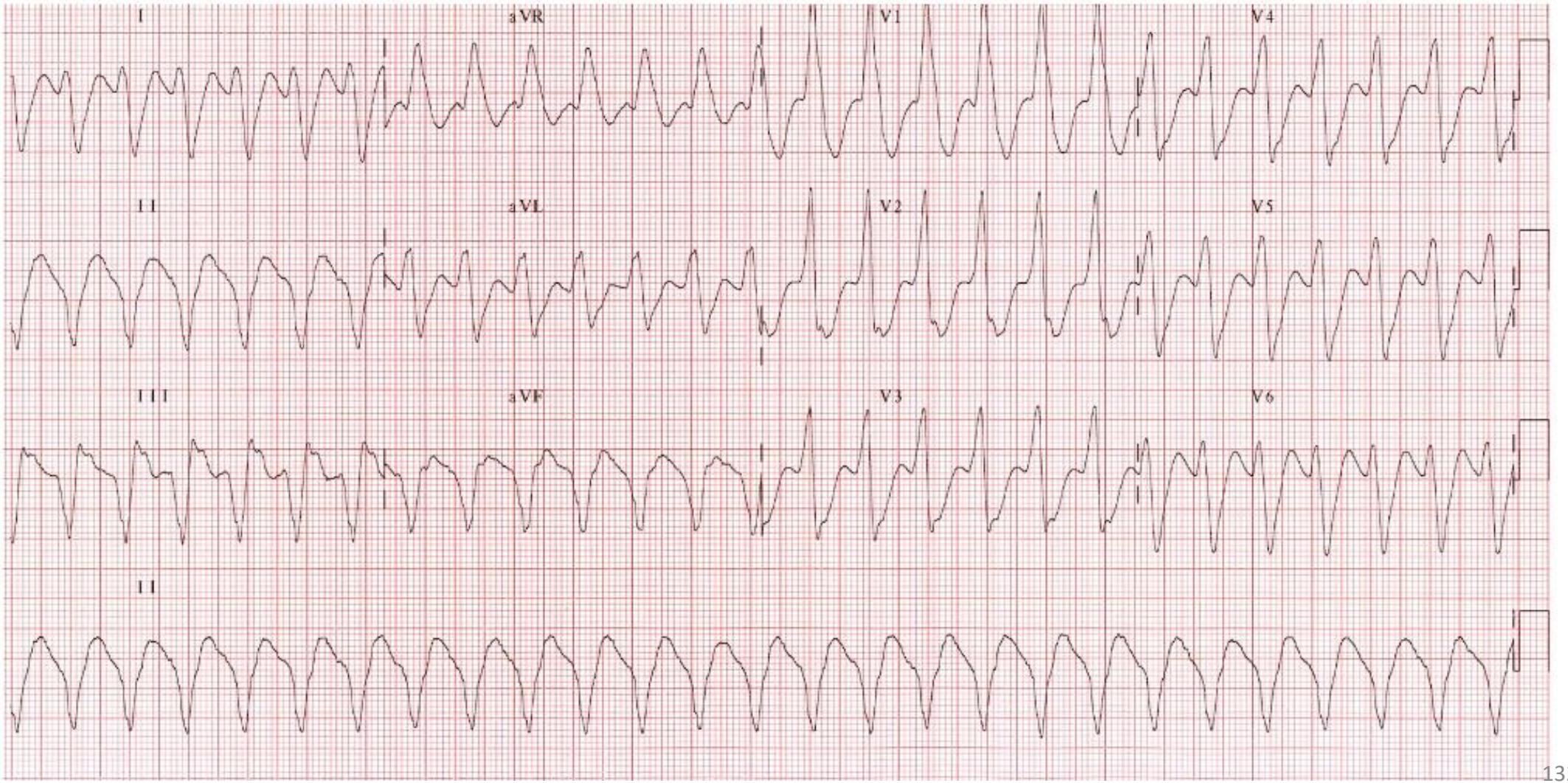
Sgarbossa Criteria

Diagnosis of MI in LBBB / VPR requires at least one of the following criteria to be present:

- Concordant ST depression > 1 mm in V1-3.
- Concordant ST elevation > 1 mm in any lead.
- Excessively discordant ST elevation in any lead >5 mm (original Sgarbossa criteria) or >25% of the corresponding S-wave depth (modified Sgarbossa criteria = more specific).

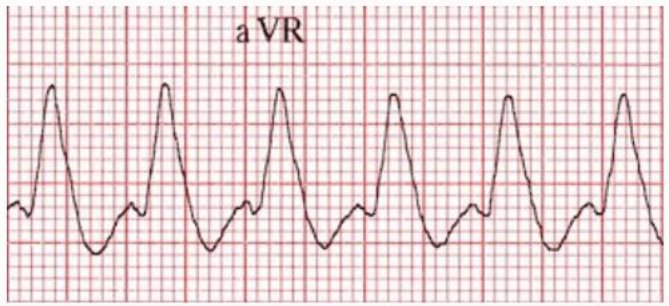
Changes only have to be present in a single lead to be diagnostic of MI.

Middle-aged patient presenting with palpitations. Describe the ECG

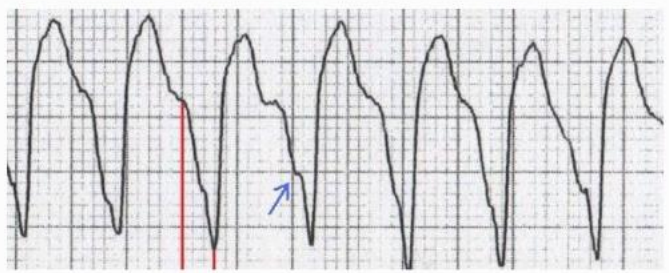


This ECG contains multiple diagnostic features for ventricular tachycardia:

- Regular broad complex tachycardia at ~150 bpm.
- Very broad QRS complexes (~200 ms).
- Northwest axis (-120 degrees) with positive QRS in aVR.
- Brugada's sign – Time from onset of the QRS complex to nadir of the S-wave > 100ms.
- Josephson's sign – Notching near the nadir of the S wave.



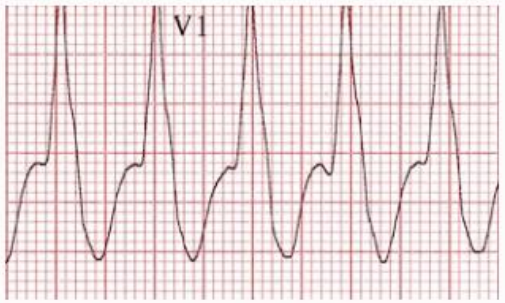
Northwest Axis



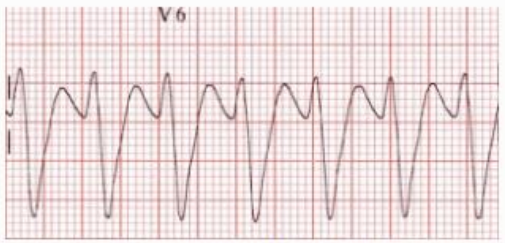
Brugada's sign (red callipers) and Josephson's sign (blue arrow)

Also note the presence of morphology criteria favouring VT over RBBB:

- Tall monophasic R wave in V1.
- Dominant S wave in V6.

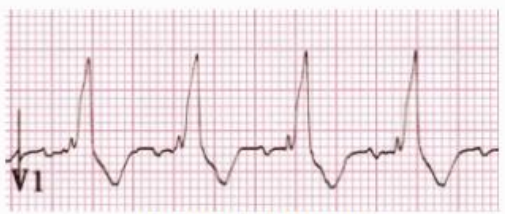


Monophasic R wave in V1 with VT

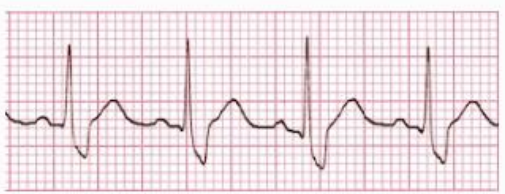


Dominant S wave in V6 with VT

This pattern in V1 and V6 is very different from the expected morphology in RBBB.

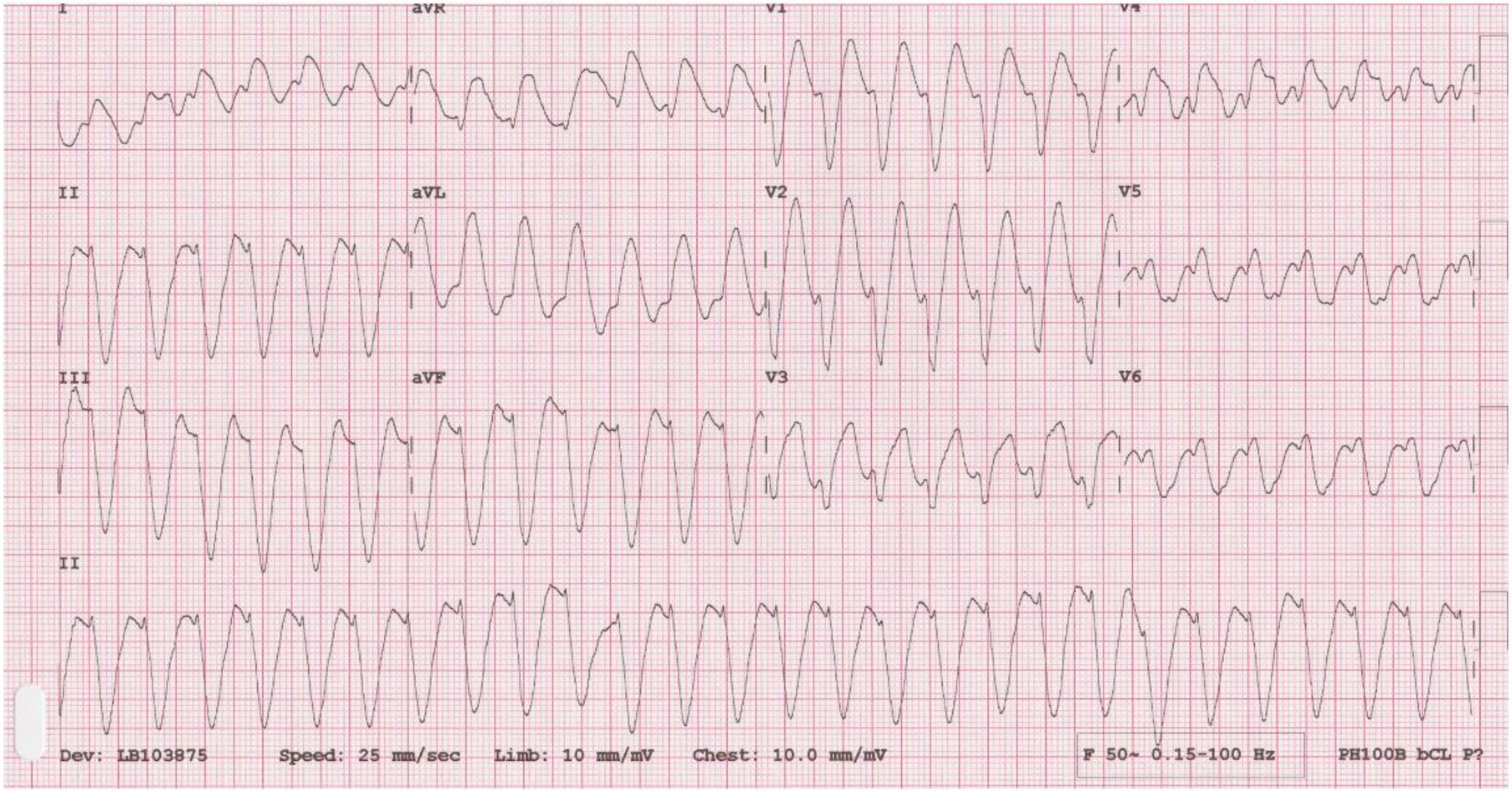


RSR' pattern in V1 with RBBB



Dominant R wave with wide slurred S wave in V6

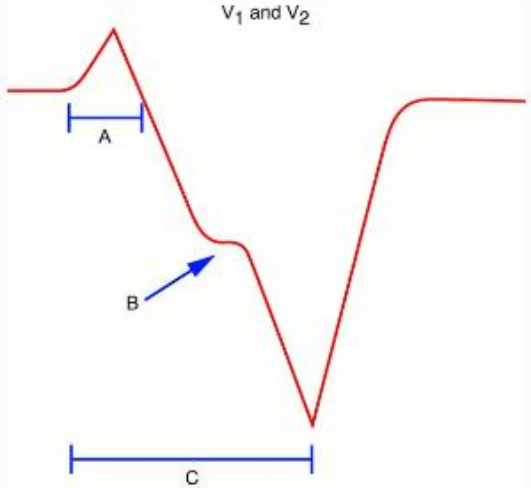
Middle-aged patient presenting with palpitations. Describe the ECG



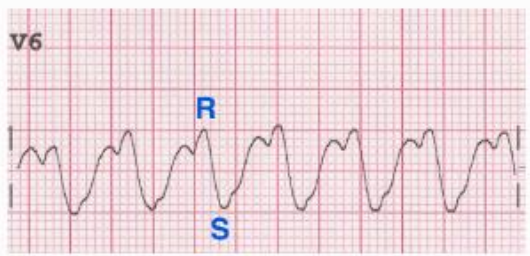
This is another example of ventricular tachycardia, this time with a LBBB morphology (compare this with ECG 046).

- Regular broad complex tachycardia at ~ 160 bpm.
- Features of VT in V1: Initial R wave > 30 ms wide, RS interval > 70 ms (= Brugada sign).
- Features of VT in V6: Dominant S wave in V6, absence of typical LBBB morphology.
- Abnormal axis with positive aVR, although does not quite meet criteria for northwest axis.

NB. Note that a positive Brugada sign only requires an RS interval of >70 ms when LBBB morphology is present, compared to >100 ms when RBBB morphology is present.



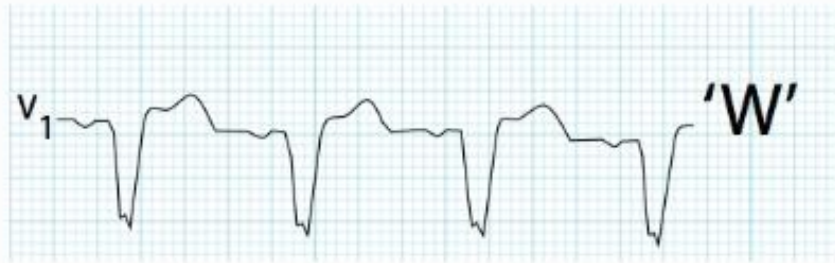
VT – appearance in V1. Reproduced from Wellens (2001).



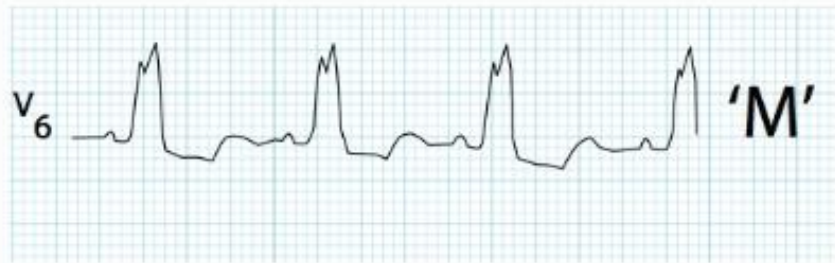
VT – appearance in V6.

Again, these features are very different to the expected pattern in LBBB, which has:

- Dominant S wave in V1, but with an R/S interval < 70 ms and minimal initial R wave.
- Dominant R wave in V6 (often slurred).



LBBB – appearance in V1.



LBBB – appearance in V6.

Looking for VT in RBBB morphology

[NB. RBBB morphology = QRS > 120ms with dominant R wave in V1]

Suspect VT in any patient with a regular broad complex tachycardia (especially if > 160 ms wide).

Look at aVR

- Positive QRS complex?
- Leads I and aVF negative?
- If yes to both -> northwest axis is present -> probable VT.

Look at V1

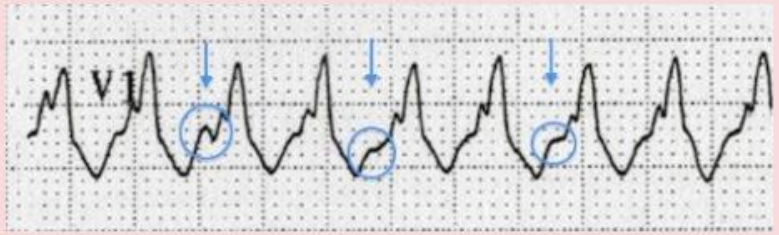
- Monophasic R wave or taller left rabbit ear? -> probable VT.
- RSR' pattern with taller right rabbit ear? -> possible SVT with RBBB.

Look at V6

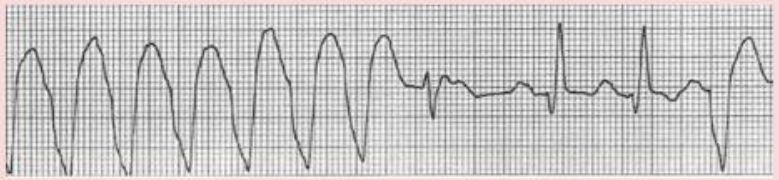
- Dominant S wave (R/S ratio < 1)? -> probable VT.
- Dominant R wave with wide slurred S wave -> possible SVT with RBBB.

If still uncertain…scrutinise the ECG for:

- Brugada's and Josephson's signs (see above).
- AV dissociation — P waves randomly deforming the QRS complexes and T waves.
- Fusion and capture beats.



AV dissociation: superimposed P waves at a different rate to the QRS complexes



The first of the narrower complexes is a fusion beat, the next two are capture beats.

Tips for Spotting VT when LBBB morphology present

[NB. LBBB morphology = QRS > 120ms with dominant S wave in V1]

Suspect VT in any patient with a regular broad complex tachycardia (esp if > 160 ms wide).

Look at aVR

- Positive QRS complex?
- Leads I and aVF negative?
- If yes to both -> northwest axis is present -> probable VT.

Look at V1

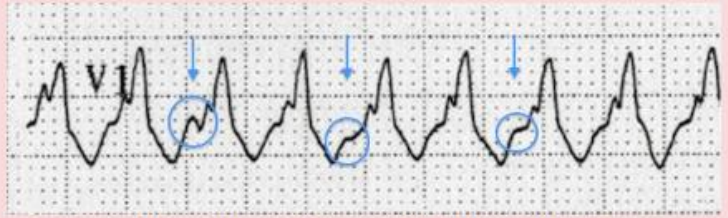
- Initial R wave > 30 ms? -> probable VT.
- Notching of the S wave (Josephson's sign)? -> probable VT.
- RS interval > 70 ms (Brugada's sign)? -> probable VT.
- None of the above -> possible SVT with LBBB.

Look at V6

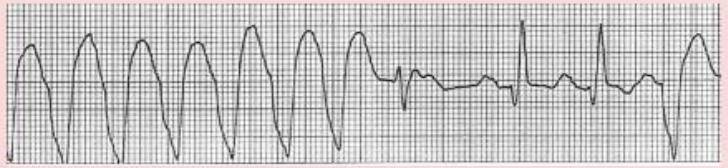
- Dominant S wave (R/S ratio < 1)? -> probable VT.
- Dominant R wave (R/S ratio > 1)?-> possible SVT with LBBB.

If still uncertain, scrutinise the ECG for:

- AV dissociation — P waves randomly deforming the QRS complexes and T waves.
- Fusion and capture beats.

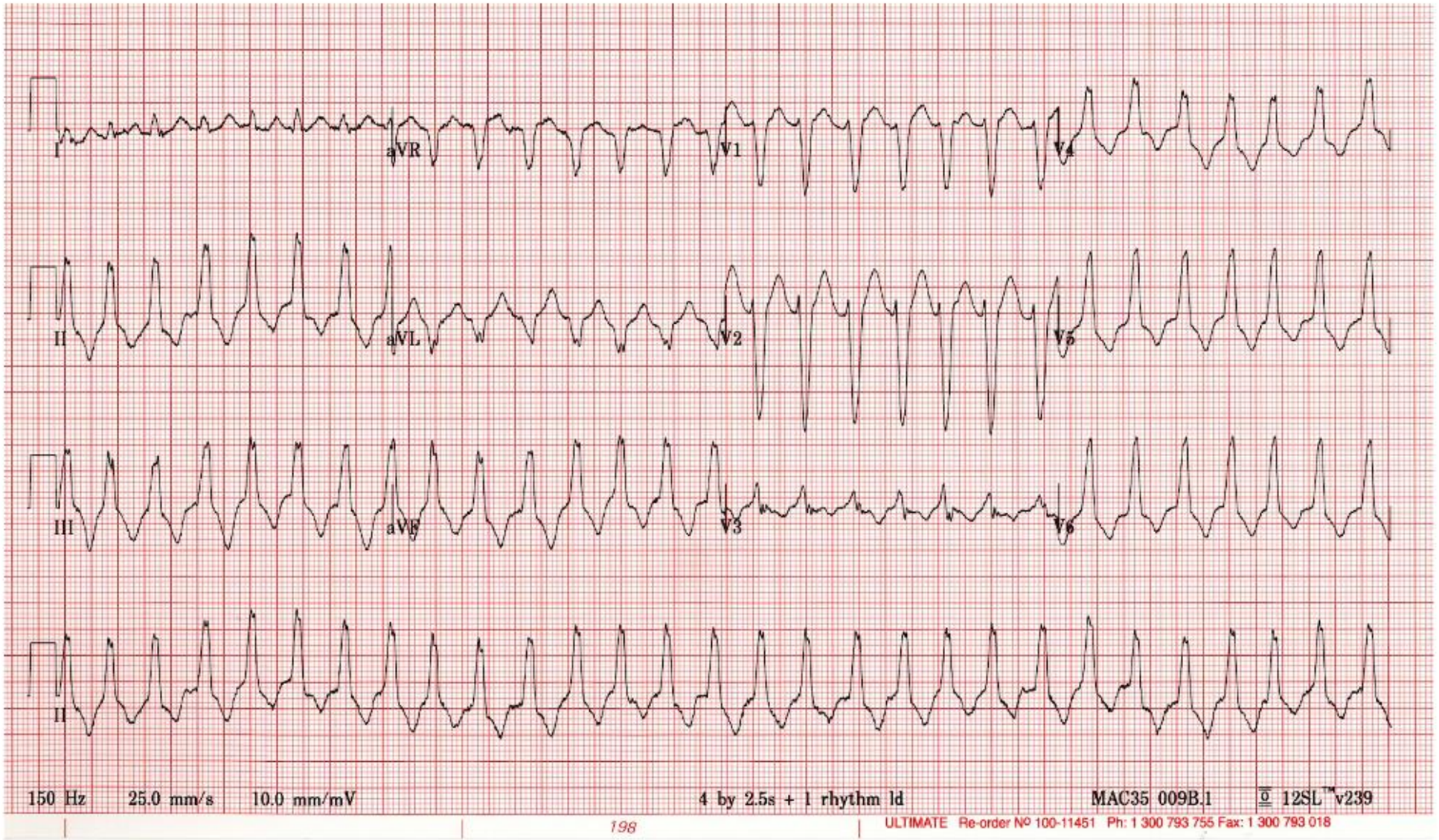


AV dissociation: superimposed P waves at a different rate to the QRS complexes



The first of the narrower complexes is a fusion beat, the next two are capture beats.

30-year old female presenting with sudden onset of palpitations. Normally well. Describe the ECG.



On first glance this would appear to be SVT with LBBB as there is:

- Regular broad-complex tachycardia.
- No atrial activity seen.
- Typical LBBB morphology in aVR, V1 and V6.
- No obvious diagnostic features for VT — compare this with ECG 047.

However, there is ***one feature*** here that is unusual for LBBB, can you spot it?

Reveal Answer

- There is an **inferior axis (+90 degrees)**, which is atypical for LBBB.
- LBBB normally has a leftward axis.

This combination of…

- Broad complex tachycardia with typical LBBB morphology.
- Inferior axis (+90 degrees).

… is suggestive of a specific type of VT known as right ventricular outflow-tract tachycardia (RVOT).

RVOT is a relatively common form of right ventricular VT, occurring in two main groups:

- Patients with structurally normal hearts (= 70% of idiopathic VT).
- Patients with arrhythmogenic right ventricular cardiomyopathy.

It may be very difficult to differentiate RVOT from SVT with LBBB.

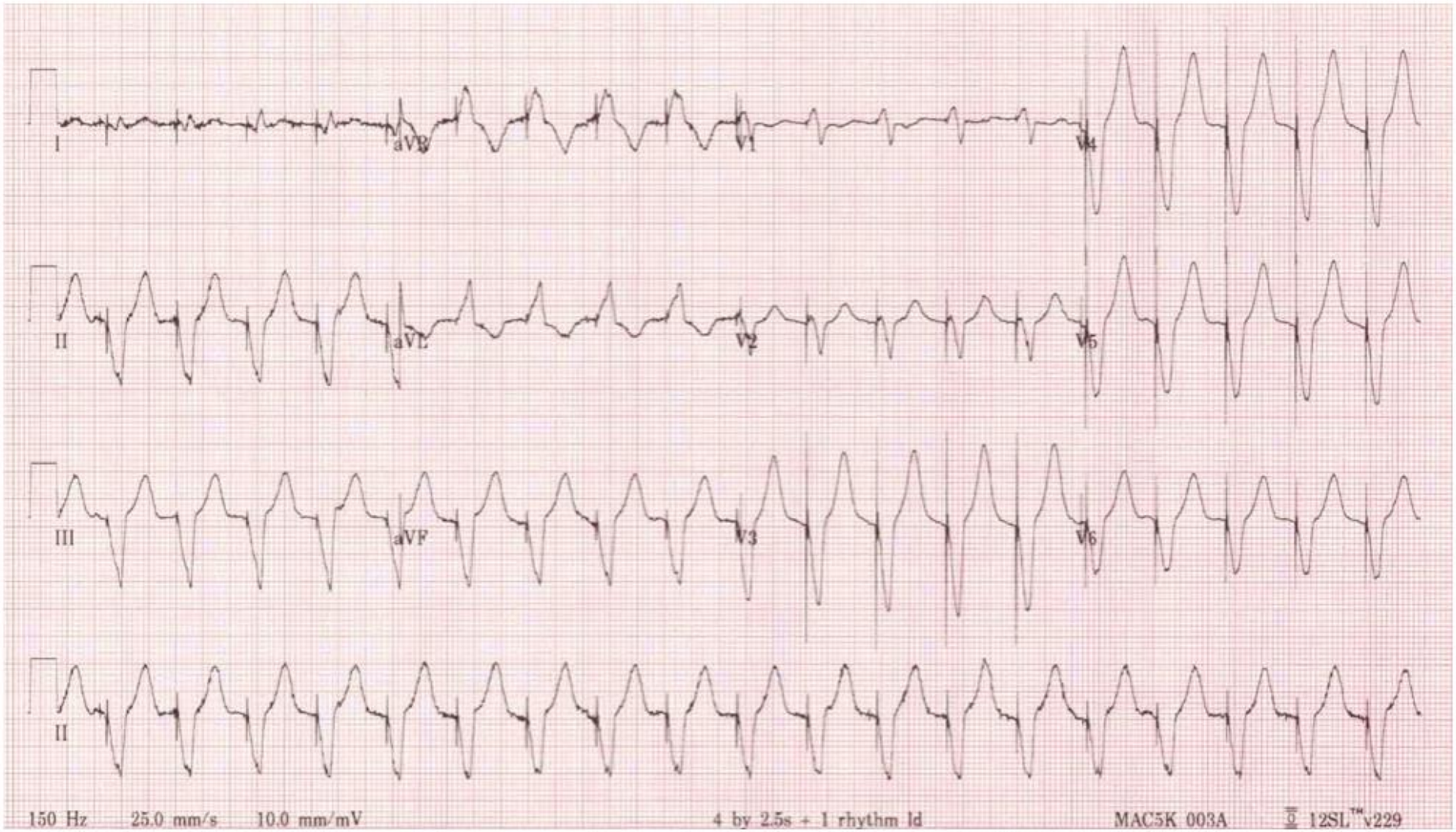
[NB. Left bundle branch block *morphology* simply indicates that the heart is depolarising from *right to left*. Hence, similar QRS patterns are seen with LBBB, RVOT and RV-pacing]

Tips for Spotting RVOT

- Suspect RVOT when you see LBBB morphology + inferior axis.
- Record a long rhythm strip looking for fusion and capture beats.

I have diagnosed this only a couple of times in the past. Each time I had to stand by the monitor with my finger on the “print” button waiting for a fusion or capture beat to appear before anyone would believe me!

Elderly male presenting with dizziness. Describe the ECG



Main Abnormalities

- Broad complex tachycardia at ~ 120 bpm.
- Pacing spikes precede each QRS complex.
- LBBB morphology (dominant S wave in V1-2) indicates a pacing electrode in the *right* ventricle.
- *Negative concordance* is seen in V1-6 (all precordial leads show negative complexes). This is an often-cited feature of VT, but also occurs with paced rhythms. It simply indicates that ventricular depolarisation is spreading from anterior to posterior (away from V1-6), e.g. due to a pacemaker electrode stimulating the anterior wall of the RV.

These features are consistent with a pacemaker malfunction resulting in a rapid ventricular-paced rhythm.

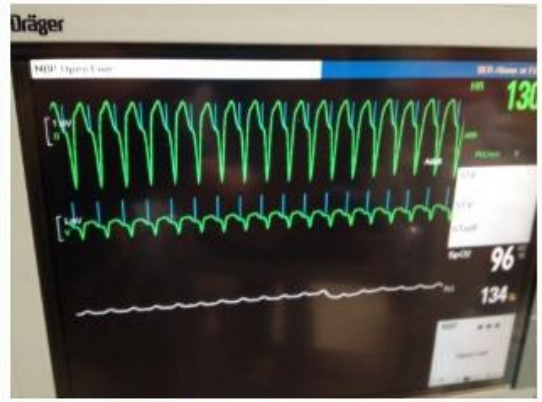
Differential Diagnosis

The differential diagnosis of this rhythm includes:

- Pacemaker-mediated tachycardia
- Sensor-induced tachycardia
- Atrial tachycardia (e.g. sinus, AF) driving the pacemaker to its maximum rate — may be appropriate response to exercise, shock, sepsis, etc.

Pacemaker Mediated Tachycardia (PMT)

This is a re-entrant rhythm involving the pacemaker circuit. It behaves very much like the atrioventricular re-entry tachycardia (AVRT) seen with WPW syndrome, except that in this case the "accessory pathway" is formed by the pacemaker circuit. PMT is triggered when ventricular impulses pass retrogradely through the AV node and depolarise the atria. This retrograde P wave is sensed by the pacemaker, which then immediately paces the ventricles. This is followed by another retrograde P wave that maintains the circus movement. This rhythm can be terminated by activating magnet mode (which switches off sensing), or by reprogramming the pacemaker box (e.g. increasing the refractory period to "block out" the retrograde P waves).



Pacemaker Mediated Tachycardia

Activation of magnet mode terminates PMT and switches to AV sequential pacing (no sensing).

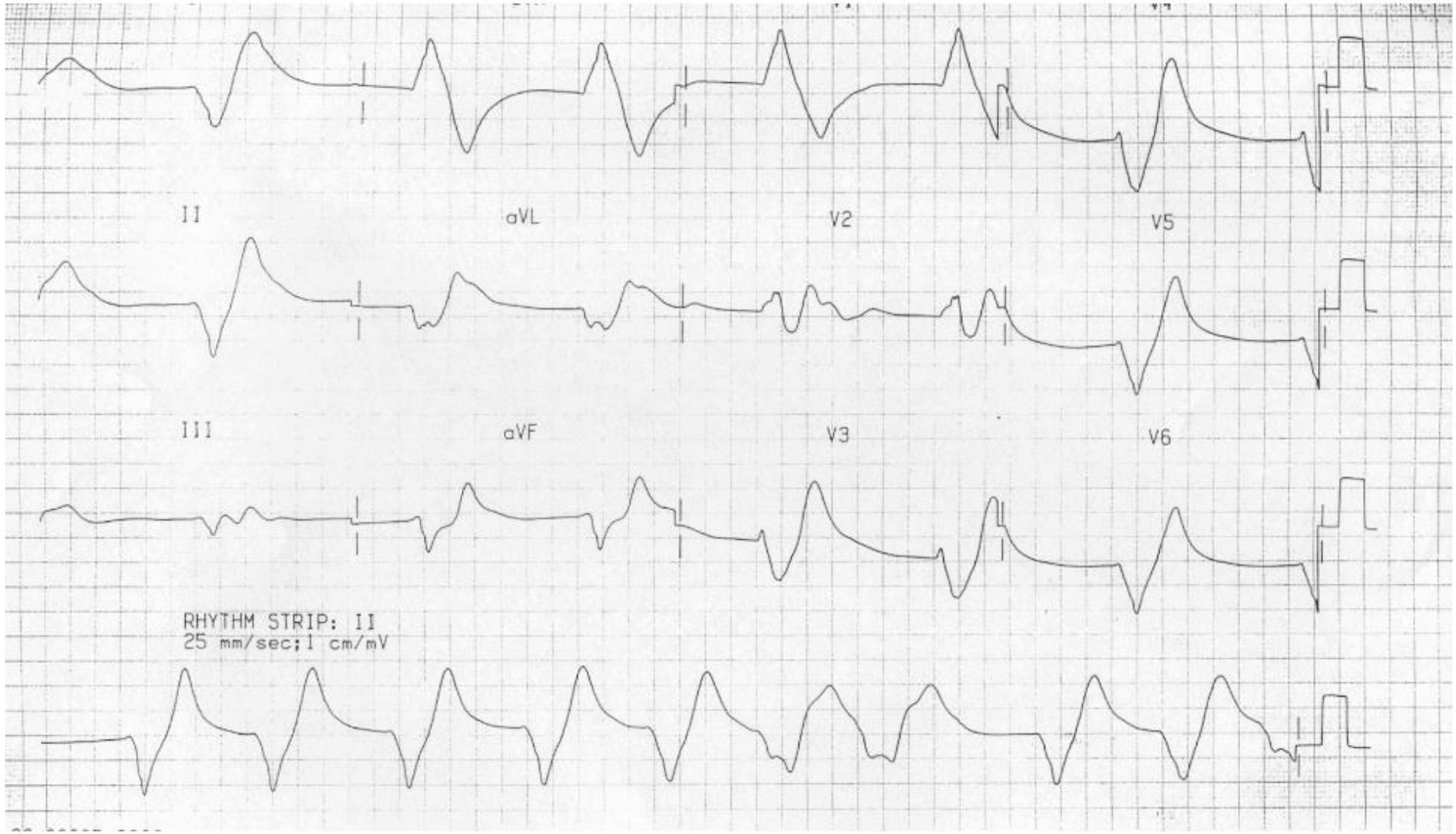
Sensor Induced Tachycardia (SIT)

- Modern pacemakers are programmed to allow increased heart rates in response to physiological stimuli such as exercise, tachypnoea, hypercapnia or acidaemia.
- Sensors may "misfire" in the presence of distracting stimuli such as vibrations, loud noises, fever, limb movement, hyperventilation or electrocautery (e.g. during surgery).
- This misfiring leads to pacing at an inappropriately fast rate.
- The ventricular rate cannot exceed the pacemaker's upper rate limit.
- Similar to PMT, these may also terminate with application of a magnet, or with removal of the inciting stimulus.

Tips for Dealing with Rapid Paced Rhythms

- If the patient is attached to critical care monitoring, ensure that you **switch off respiratory impedence monitoring mode** (where the monitor senses respiratory rate via the ECG electrodes). This gives off an electrical signal that is known to interfere with pacemakers. If this doesn't work, try changing monitors to one that does not use respiratory impedance technology.
- **Apply a magnet** to the pacemaker box. This activates magnet mode, which resets the pacemaker to provide temporary asynchronous pacing (AOO, VOO or DOO).
- Arrange for an **urgent pacemaker check**. A pacemaker technician may be able to adjust the settings to terminate the dysrhythmia and prevent its recurrence.
- If a pacemaker technician is not readily available, then a **trial of AV nodal blocking drugs** (e.g. beta blockers, verapamil) may be successful in terminating PMT.

Elderly patient presenting with collapse. Clinically dehydrated. Describe the ECG.



This is a classic **sine wave** ECG of critical hyperkalaemia

- Bradycardia (~ 55 bpm).
- Bizarre-looking QRS complexes.
- Gross QRS prolongation (> 300 ms).
- Massively peaked T waves.

This patient had a K+ of 9.9 mmol/L!

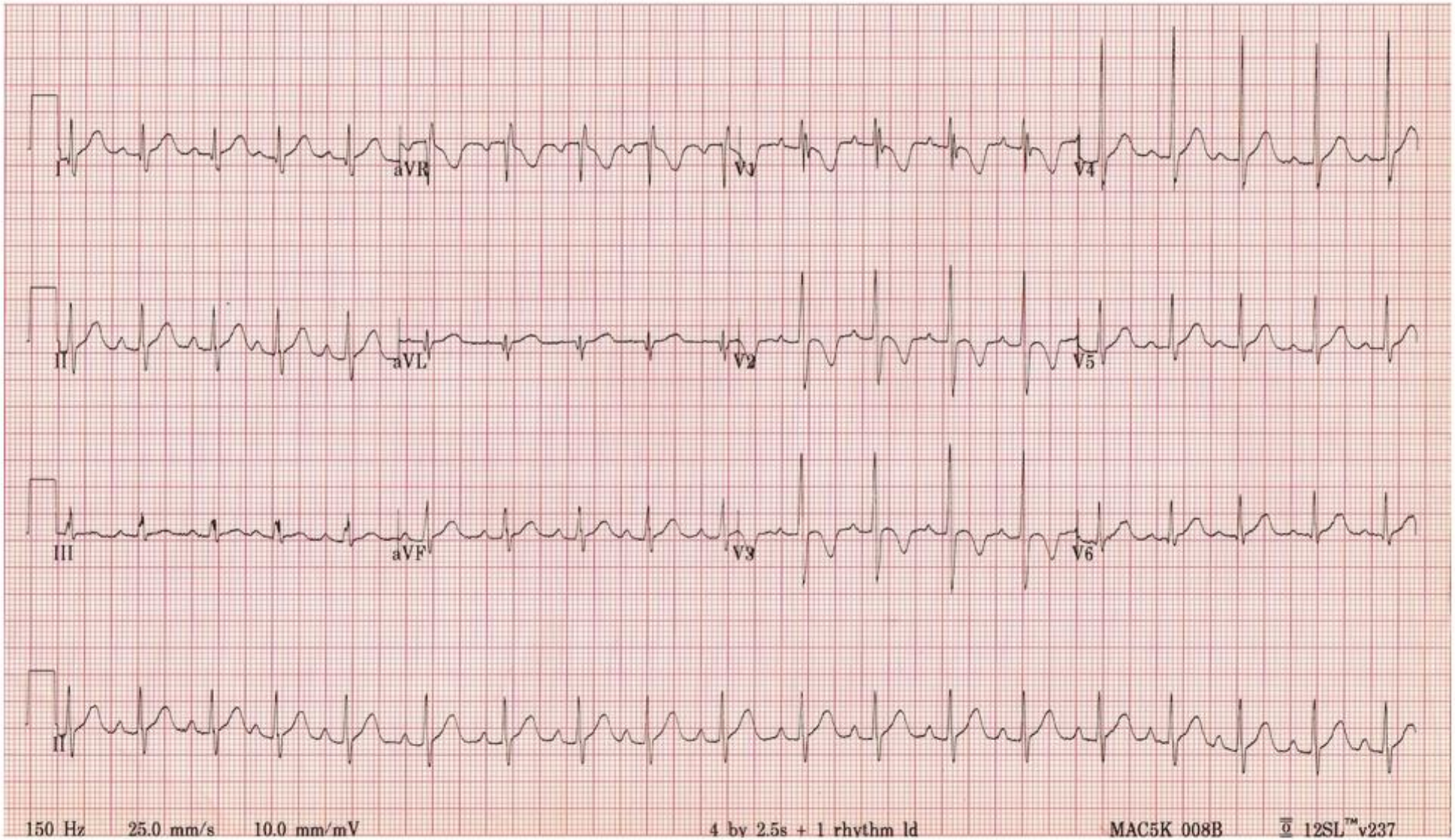
Clinical Pearls

Any time you see an ECG that is…

- **Blocked** (AV block, bundle branch block)
- **Bradycardic** (sinus brady, slow AF, junctional bradycardia)
- **Bizarre** in appearance

… consider hyperkalaemia and check the K+ urgently!

2-year old boy presenting with febrile seizure. Interpret the ECG.



This is a normal paediatric ECG, displaying multiple age-appropriate features:

- Heart rate of 110 bpm (normal for age).
- Dominant R waves in V1-3.
- Partial RBBB (RSR' pattern in V1).
- Juvenile T-wave pattern (T wave inversion in V1-3).

Any of the following findings may be normal on the paediatric ECG:

Heart rate and rhythm

- Heart rate >100 beats/min.
- Marked sinus arrhythmia.

Right-sided predominance

- Rightward QRS axis > +90°
- Dominant R wave in V1
- RSR' pattern in V1
- T wave inversions in V1-3 ("juvenile T-wave pattern")

P waves

- Slightly peaked P waves (< 3mm in height is normal if ≤ 6 months)

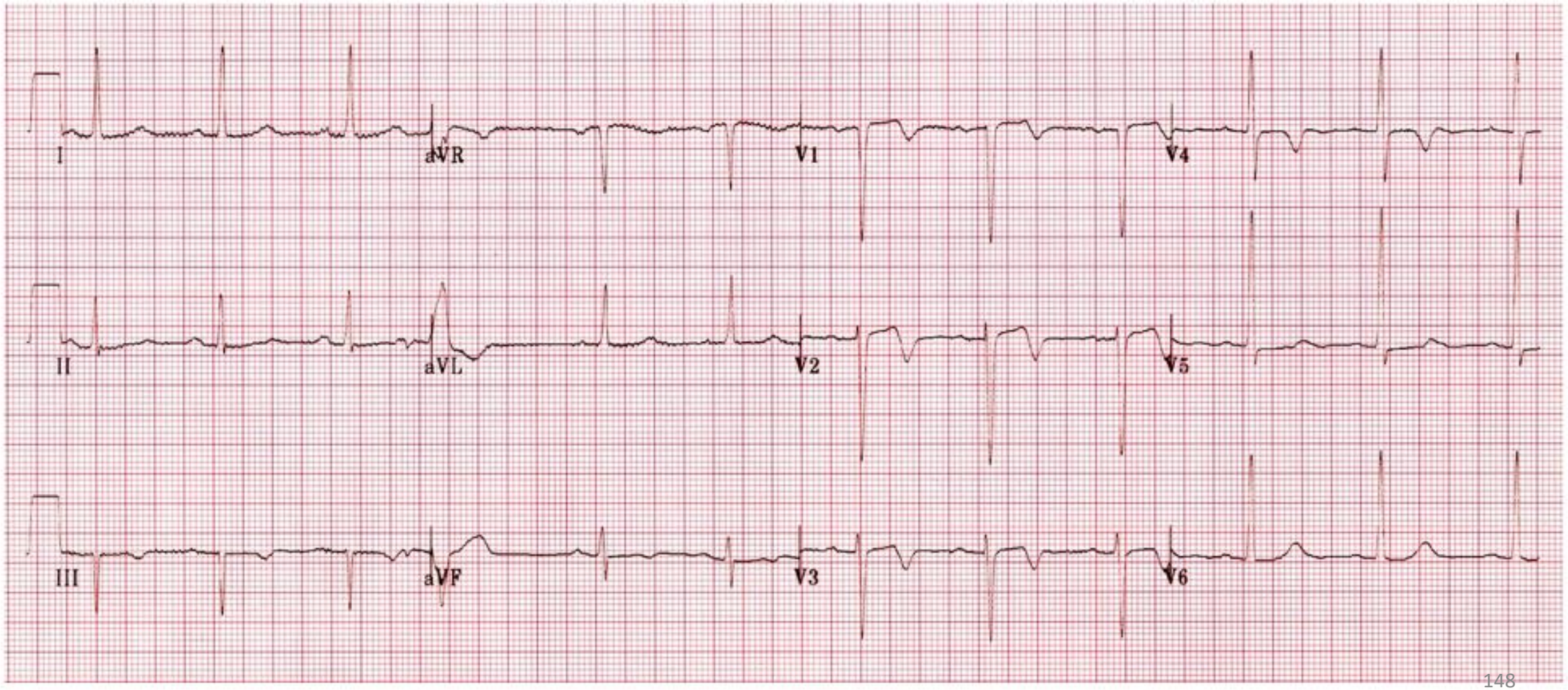
Intervals

- Short PR interval (< 120ms) and QRS duration (<80ms)
- Slightly long QTc (≤ 490ms in infants ≤ 6 months)

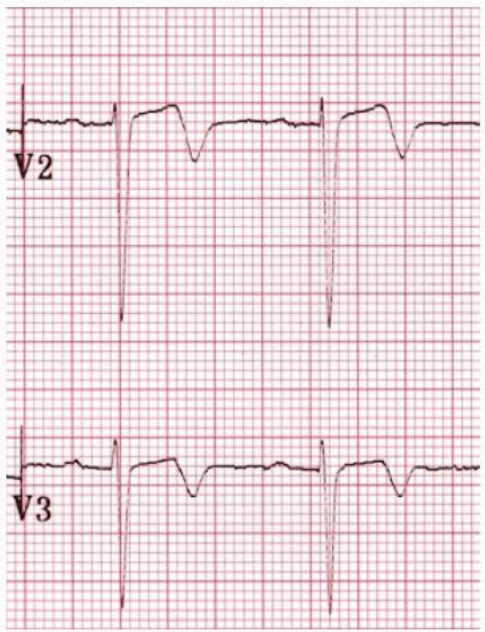
Pseudo-infarction patterns

- Q waves in the inferior and left precordial leads.

Middle-aged patient presenting with central chest pain. Now asymptomatic. Interpret the ECG.



- This is classic ECG of Wellens' syndrome, demonstrating characteristic biphasic T waves in V1-3.
- This ECG pattern is highly predictive of a significant occlusive lesion of the LAD.
- The biphasic T waves are a marker of reperfusion and may occur after an aborted anterior STEMI.
- Despite often being pain free and having normal cardiac enzymes at presentation, these patients are at risk of sudden LAD re-occlusion leading to massive anterior STEMI and are best managed with early angiography and PCI / CABG.



Biphasic T waves in Wellens' syndrome

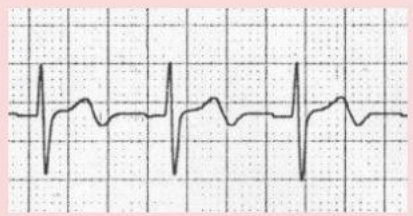
Clinical Pearls

Biphasic T waves may be seen with both Wellens' syndrome and hypokalaemia.

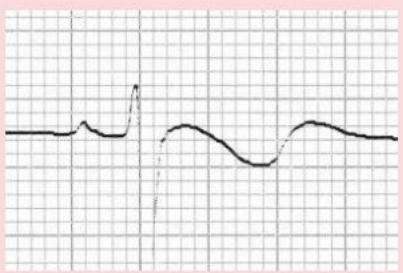
The main differentiating factor (apart from the clinical picture) is the direction of the T waves:

- Wellens' biphasic T waves go UP then down.
- Hypokalaemic T waves go DOWN then up.

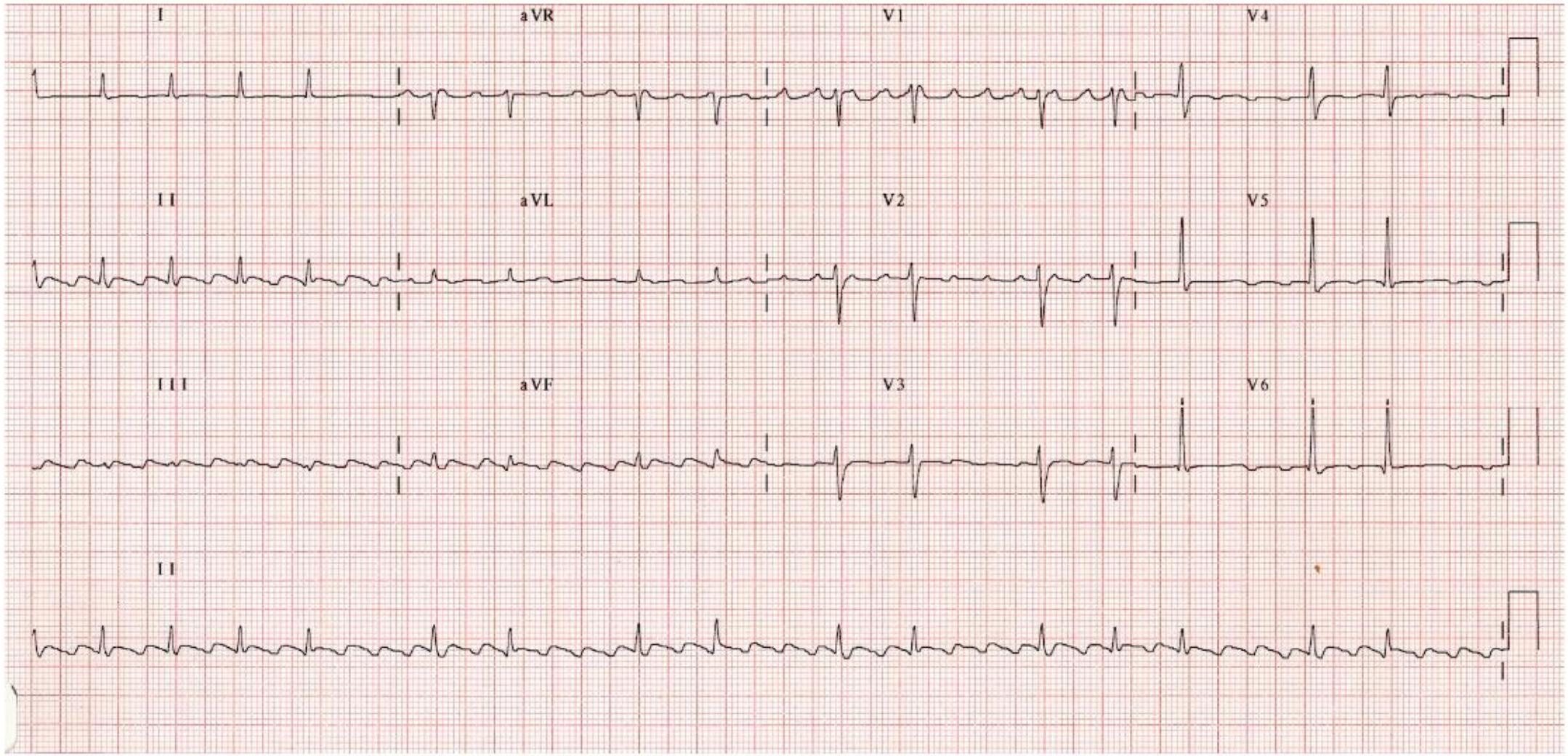
Wellens' Syndrome



Hypokalaemia



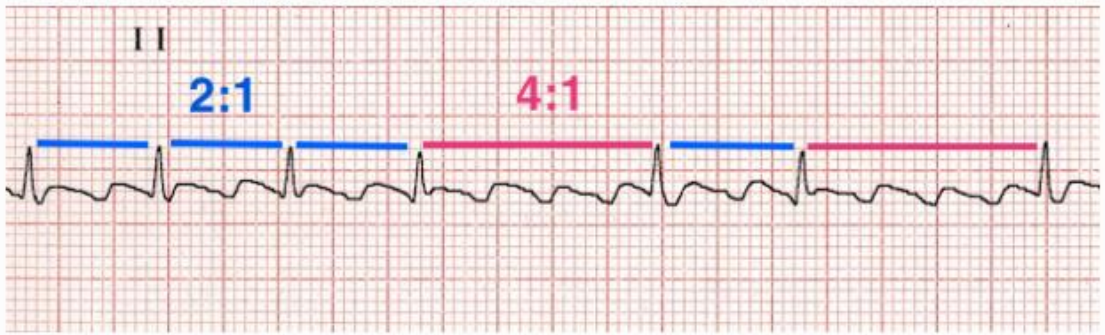
Elderly patient presenting with nausea and palpitations. Interpret the ECG.



This rhythm is frequently misidentified as atrial fibrillation. However, note in this case:

- Well-defined sawtooth waves in II, III, aVF with regular rate (~300bpm) and consistent morphology.
- Positive flutter waves ("pseudo-P waves") in V1-2.
- The rhythm is *not* irregularly irregular. There are repeating patterns of identical R-R intervals that crop up throughout the rhythm strip, corresponding to AV conduction ratios of either 2:1 or 4:1.

This is atrial flutter with a variable block.

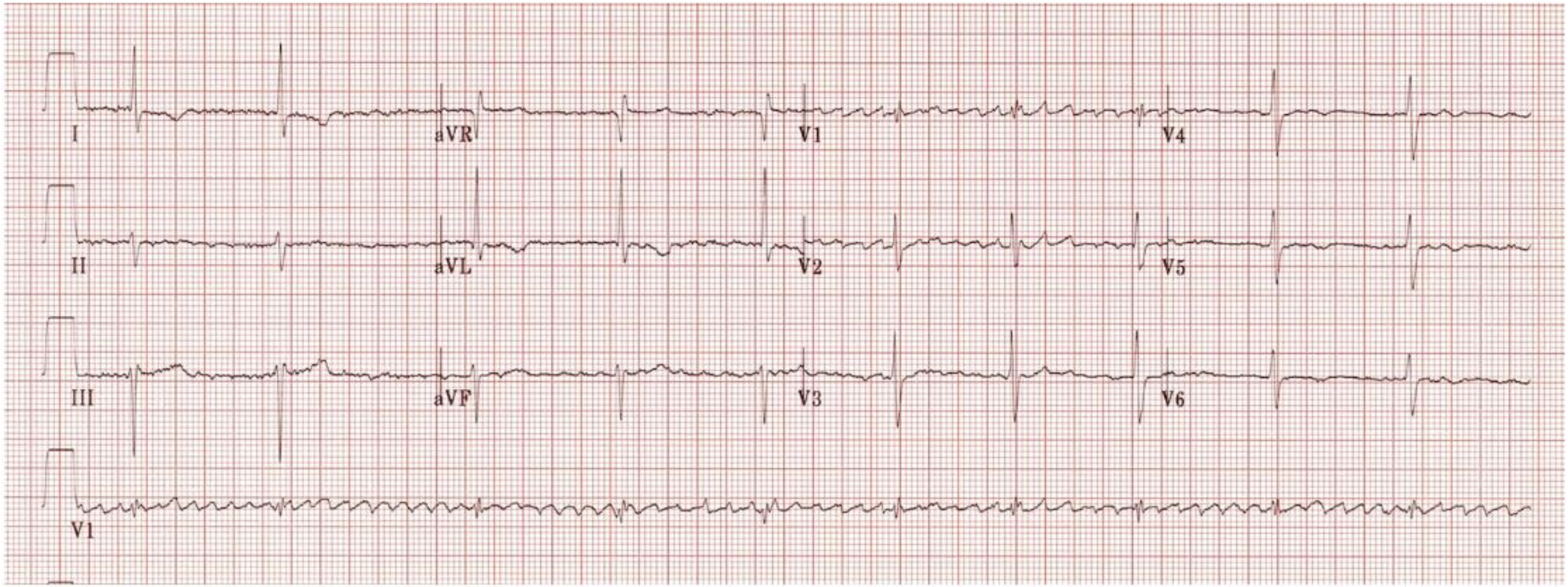


Recurring RR intervals in flutter with variable block

Clinical Pearls

- Misidentifying this rhythm as AF is not normally a big problem as both conditions are managed similarly — i.e. rate control, anticoagulation.
- However, flutter will cardiovert with lower energy than AF — e.g. 50 j compared with 100-200 j.

Elderly patient presenting with nausea and visual disturbance. Interpret the ECG.



This is a tricky ECG!

There is evidence of atrial fibrillation, as evidenced by the irregular baseline with fibrillatory waves most prominent in V1-2.

NB. Fibrillatory waves are characteristically seen in V1-2 (which overlie the atria), as opposed to tremor artefact which may be in seen in multiple leads without a predominance for V1-2.

However, the ventricular rhythm is regular. How can this be? AF is irregular by definition…

This is an example of "regularised AF" due to digoxin toxicity:

- The underlying rhythm is AF, which is being treated with digoxin.
- There is complete heart block, prevent atrial impulses from reaching the ventricles.
- There is an accelerated junctional rhythm maintaining cardiac output.

If this all seems like too much of a coincidence, then consider the pathophysiology of digoxin toxicity…

Mechanisms of Digoxin Toxicity

Digoxin toxicity produces a wide variety of dysrhythmias, due to:

- **Increased automaticity** of atrial, junctional and ventricular tissues — via actions at the Na/K and Na/Ca exchangers causing increased intracellular calcium and therefore increased spontaneous depolarisation of cardiac pacemaker cells.
- **Decreased AV conduction** — via increased vagal tone at the AV node.

Digoxin toxicity therefore usually produces some combination of:

- **Increased atrial automaticity** — especially atrial tachycardia, but also atrial ectopics, AF, flutter.
- **Increased junctional automaticity** — especially accelerated junctional rhythms.
- **Increased ventricular automaticity** — frequent VEBs and bigeminy, polymorphic VT.
- **AV blocks** — including 1st, 2nd and 3rd degree AV block.

Characteristic ECG patterns include:

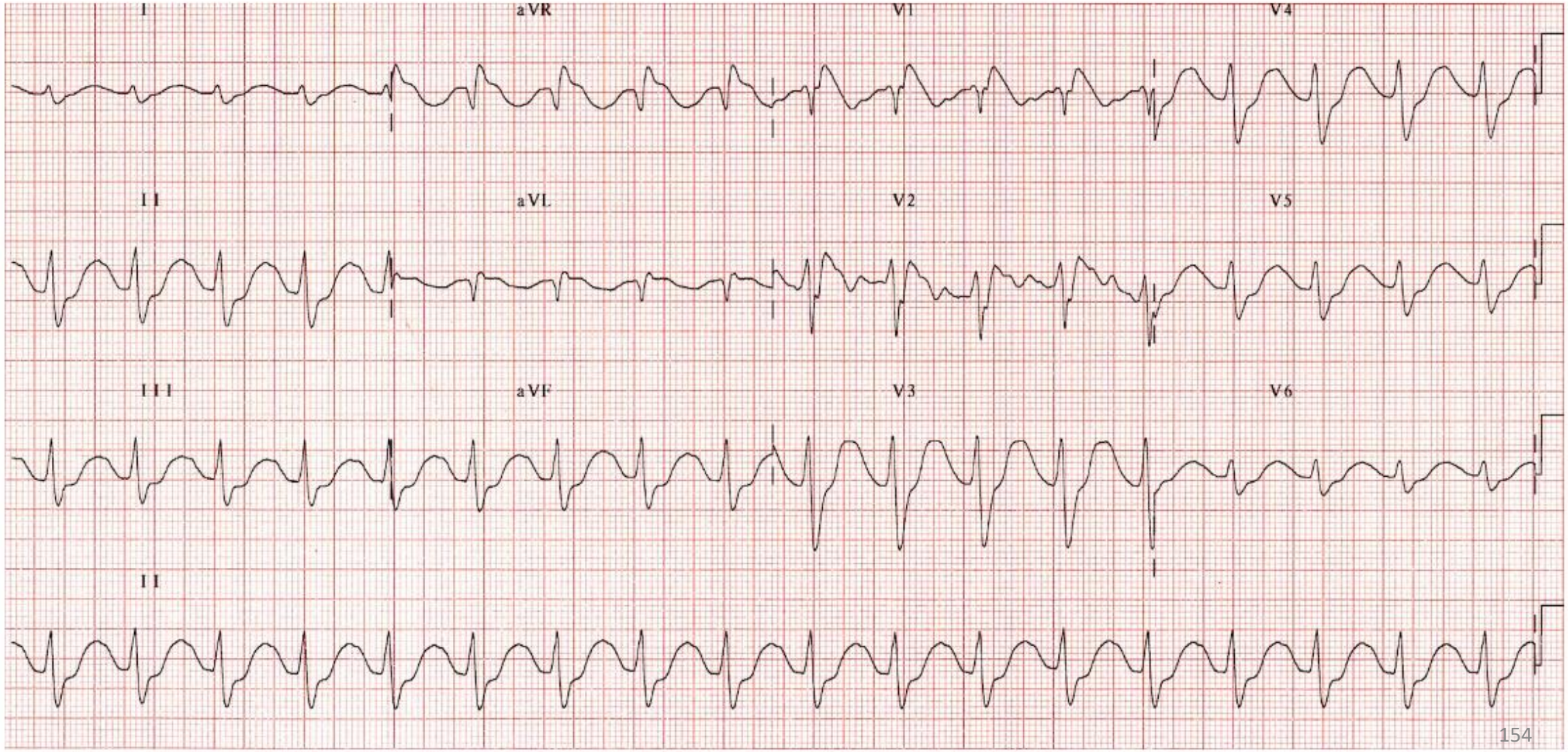
- Atrial tachycardia with high-grade AV block (= the classic dig-toxic rhythm).
- Regularised AF = AF with complete heart block + accelerated junctional escape rhythm, producing a paradoxically regular rhythm.
- Bidirectional VT = polymorphic VT with QRS complexes that alternate between left- and right-axis-deviation, or between LBBB and RBBB morphology.

NB. Digoxin toxicity should not be confused with digoxin effect (= "sagging" ST depression and T-wave inversion in patients on therapeutic doses of digoxin; not predictive of toxicity).

Clinical Pearls

- Check for tremor artefact before you start diagnosing regularised AF!
- If the ECG pattern appears genuine and the clinical picture is compatible with digoxin toxicity (GI upset, xanthopsia, current digoxin treatment), then check an urgent digoxin level.

Middle-aged patient presenting with drowsiness. BP 85/50. Pupils dilated. Interpret the ECG.



Main Abnormalities

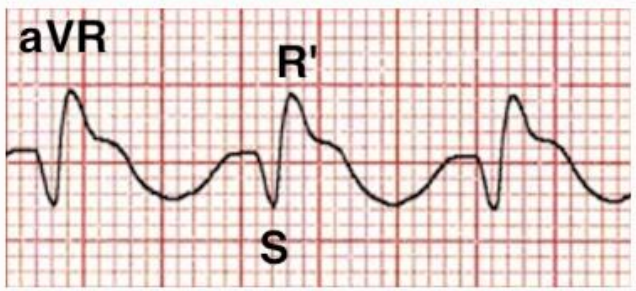
This is a classic ECG of tricyclic antidepressant toxicity, demonstrating multiple characteristic abnormalities.

Signs of sodium-channel blockade

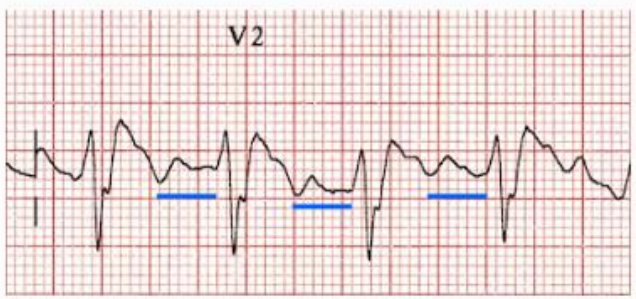
- Broad QRS complexes (120 ms, or 3 small squares).
- Positive R' wave in lead aVR > 3 mm.
- Prolonged PR interval (240 ms).
- Long QT interval (> 1/2 the RR interval).
- Brugada-like pattern in V1.

Signs of anticholinergic toxidrome

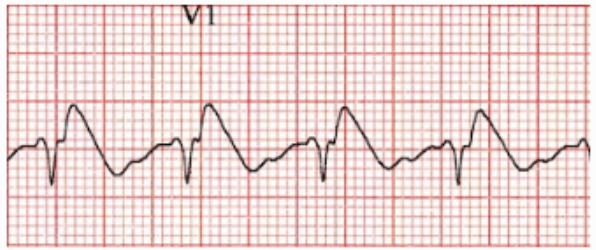
- Sinus tachycardia (~ 110 bpm), with P waves embedded in each T wave.



Positive R' wave in aVR > 3mm



P waves visible in V2 with long PR interval



Pseudo-Brugada pattern in V1

This patient had taken a life-threatening overdose of dosulepin (a TCA).

How to Spot Sodium-Channel Blockade

- QRS prolongation (> 100ms or 2.5 small squares), typically measured in lead II.
- A terminal or secondary R wave (R' wave) in aVR > 3 mm.
- An R'/S ratio in aVR > 0.7.

Prognostic Value of the ECG

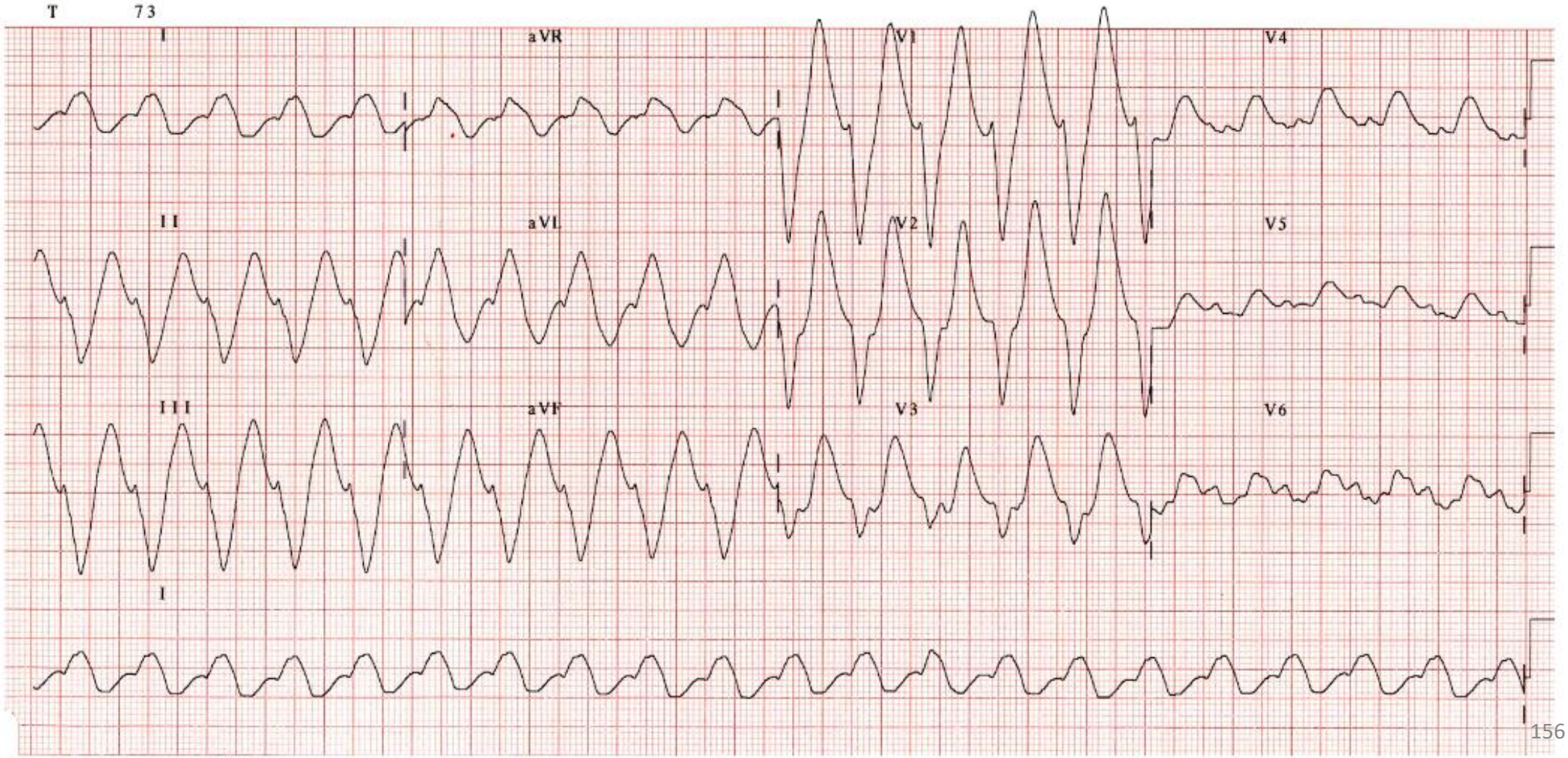
In patients with TCA overdose, the degree of QRS prolongation correlates with the degree of clinical toxicity:

- QRS width > 100 ms is predictive of seizures.
- QRS width > 160 ms is predictive of cardiotoxicity (e.g. broad-complex dysrhythmias, hypotension).

Clinical Pearls

- The combination of PR prolongation and sinus tachycardia with TCA overdose often makes the P waves difficult to see, and may lead the rhythm to be incorrectly identified as VT. This patient needs bicarbonate and hyperventilation, not electricity and amiodarone!
- The clinical significance of a TCA-induced Brugada ECG pattern remains controversial — i.e. is it purely a manifestation of severe sodium-channel blockade, or does it represent "unmasking" of underlying Brugada syndrome? These issues are discussed here.

Middle-aged patient presenting with drowsiness. Brief seizure in ED. BP unrecordable. Interpret the ECG.

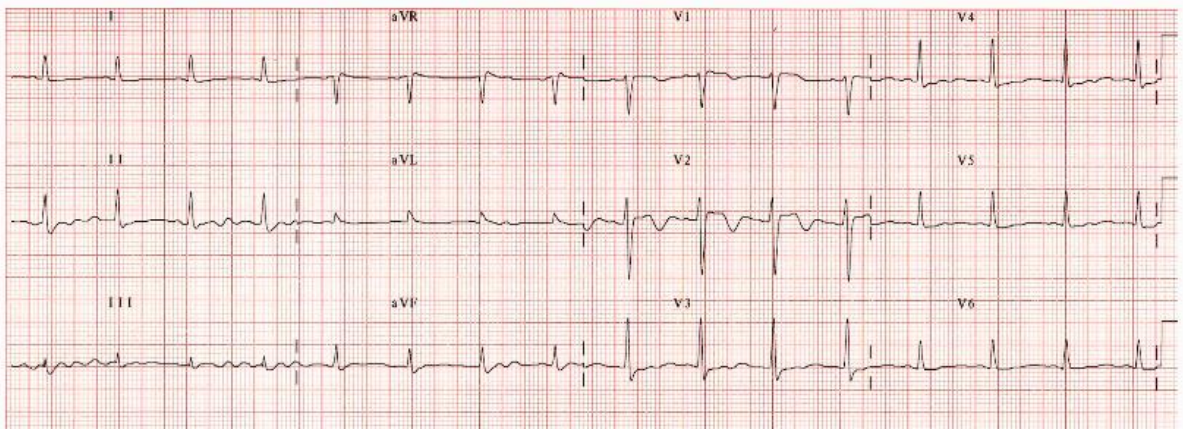


This is a repeat ECG of the previous patient with massive TCA overdose, taken a short while later.

- There is now evidence of advanced sodium-channel blockade, with grossly prolonged QRS and QT intervals and further evolution of the R' wave in aVR.
- The ECG is beginning to take on bizarre morphology and a sine wave appearance reminiscent of severe hyperkalaemia.
- In some leads (II, III, aVF), the QRS morphology resembles ventricular tachycardia.

These features are all due to sodium-channel blockade, and resolved following aggressive treatment with IV bicarbonate, intubation and hyperventilation.

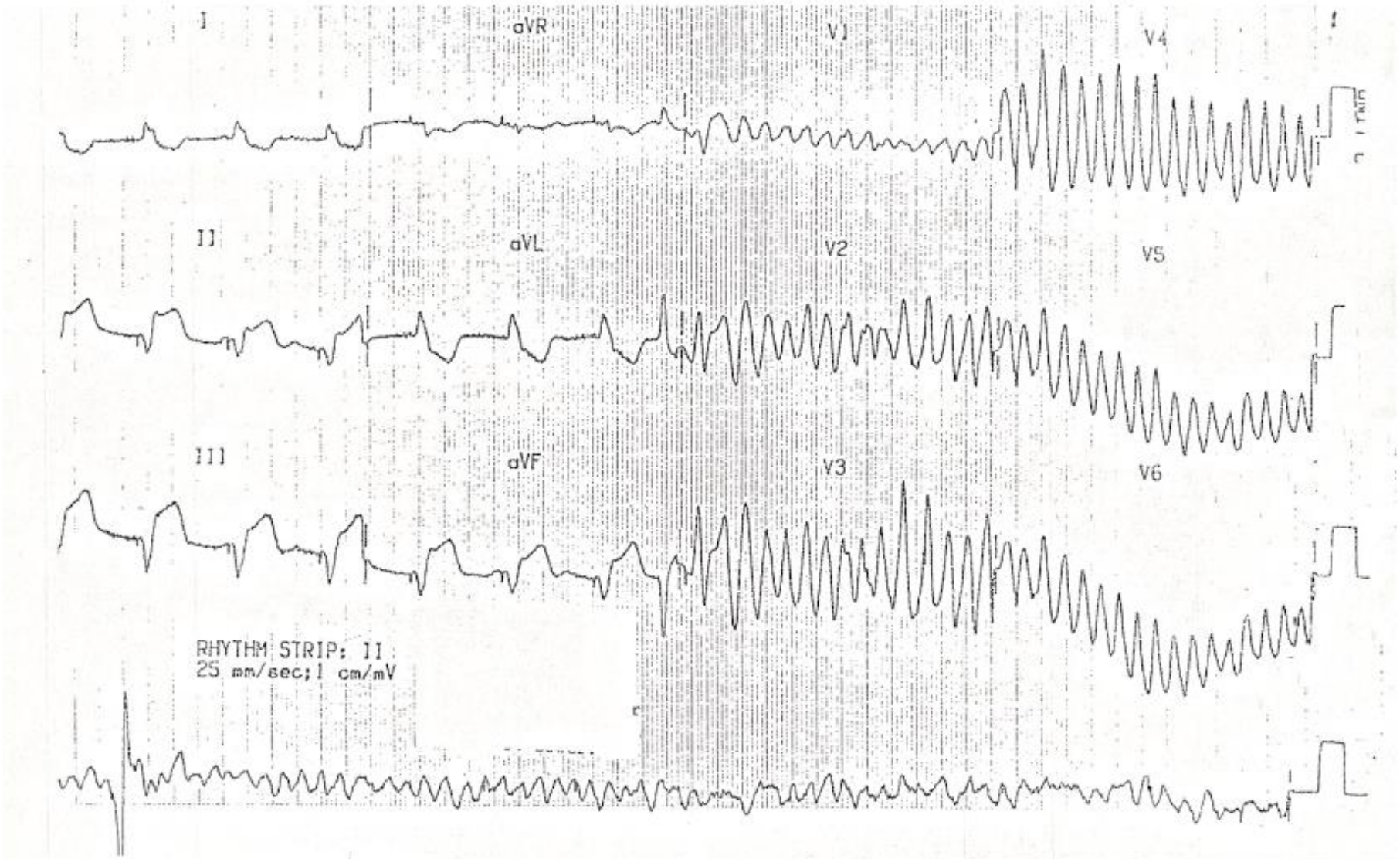
Show post-treatment ECG



Clinical Pearls

- Advanced sodium-channel blockade can resemble either VT or hyperkalaemia.
- Standard VT treatments such as DC cardioversion and amiodarone are likely to be ineffective and potentially harmful if the broad complex rhythm is due to sodium-channel blockade. Consider the clinical context and look for clues of TCA toxicity (e.g. anticholinergic toxidrome).
- In arrested / peri-arrest patients with a broad or bizarre-looking ECG, consider empirical treatment for both hyperkalaemia (with calcium) and sodium-channel blockade (with bicarbonate and hyperventilation).

Middle-aged patient presenting with chest pain and diaphoresis. Becomes unresponsive during recording of ECG. Interpret the ECG.



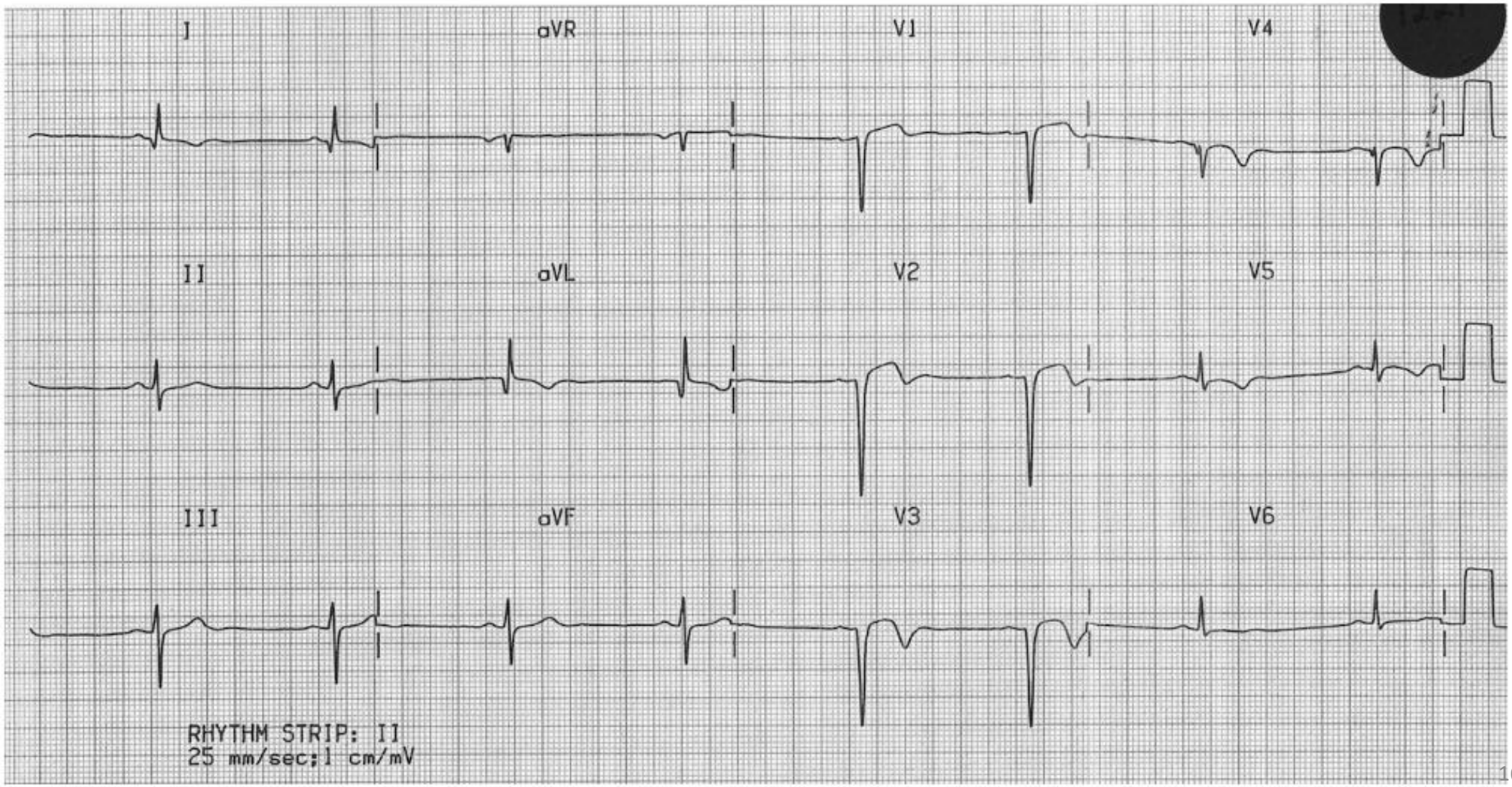
Main Abnormalities This is a fascinating ECG with multiple abnormalities:

- The first half of the tracing shows a ventricular paced rhythm with positive Sgarbossa criteria indicating superimposed inferior STEMI.
- There is excessively discordant ST elevation (> 25% of Q/S wave depth) in II, III and aVF with reciprocal change in I and aVL.
- A ventricular ectopic (beat #8) occurs at a vulnerable time, resulting in a run of ventricular flutter (very rapid VT at rates > 200-300 bpm).
- This rapidly degenerates to ventricular fibrillation (seen in the rhythm strip, which is recorded *after* the other 12 leads).
- The artefact at the start of the rhythm strip may represent a precordial thump!

Clinical Pearls

- While most rhythm strips are recorded simultaneously with the 12-lead ECG, some older machines may record the rhythm strip *after* the other 12 leads.
- Consider this as a possibility if your rhythm strip doesn't seem to "line up" with the rest of the ECG.

Elderly patient presenting with chest pain. Interpret the ECG.



Main Abnormalities

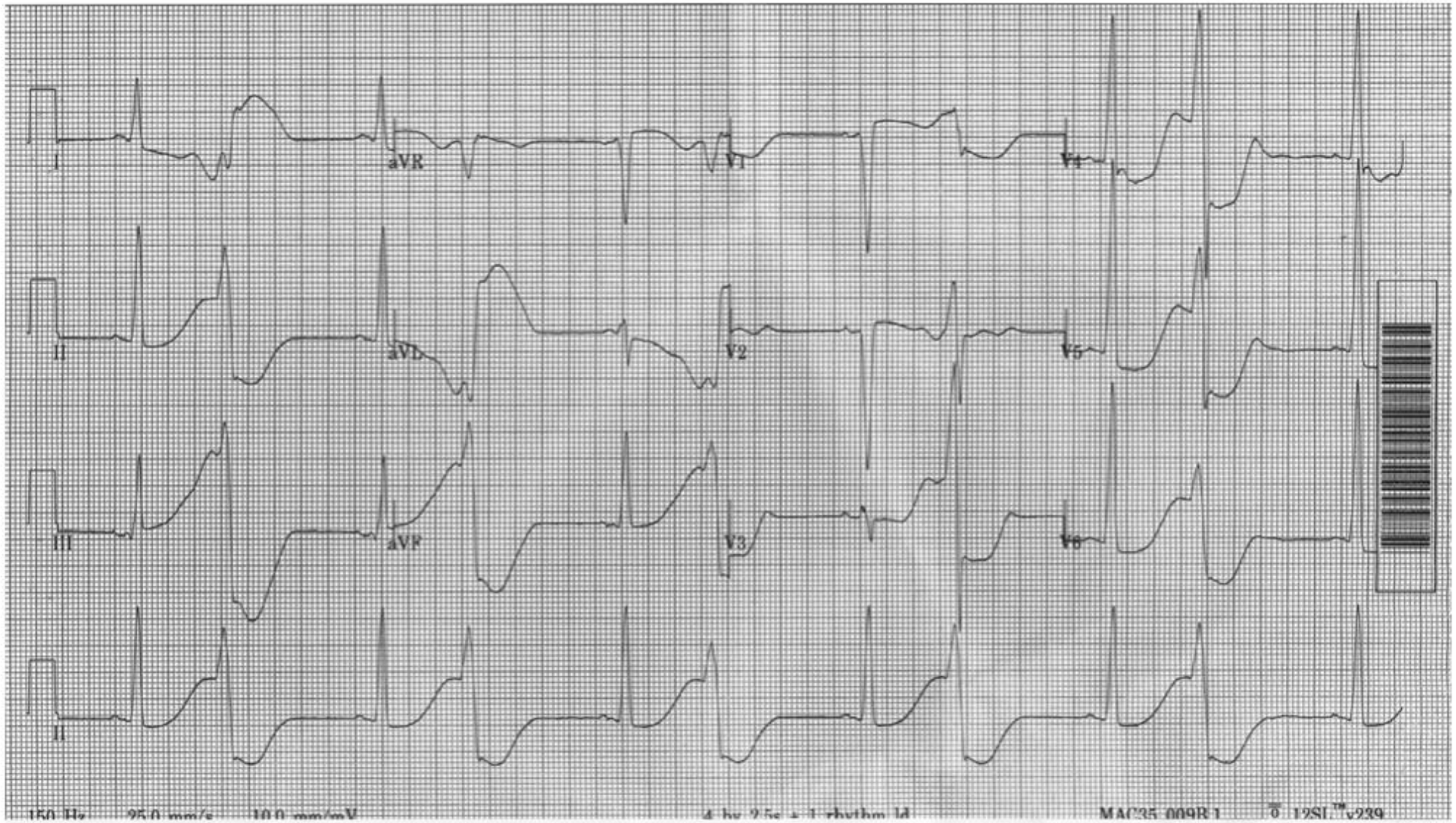
This ECG shows changes consistent with an old anterolateral infarction — the so-called left ventricular aneurysm pattern.

- ST elevation in V1-3 associated with deep Q waves (= LV aneurysm morphology).
- Pathological Q waves also seen in I, aVL and V4.
- Poor R wave progression (= R wave height < 3 mm in V3).
- Biphasic / inverted T waves in V1-5, I and aVL.

Clinical Pearls

- The LV aneurysm pattern refers to the combination of residual ST elevation, deep Q waves and inverted or biphasic T waves seen in patients following an acute myocardial infarction. This ECG pattern is associated with transmural scarring and paradoxical movement of the LV on wall on echocardiography.
- Around 60% of patients with anterior STEMI develop some degree of chronic ST elevation on their ECG, which can cause diagnostic confusion.
- If these patients present with chest pain, the safest approach is to take serial ECGs looking for signs of evolving STEMI such as evolving ST elevation or pseudo-normalisation of T waves.

57-year old man with ROSC following VF arrest. Interpret the ECG.



Main Abnormalities

- Sinus rhythm with frequent ventricular ectopics in a pattern of ventricular bigeminy.
- Grossly prolonged QT interval (> 600 ms).
- "R on T" phenomenon is present, with each VEB falling on the end of the T wave — this ECG pattern is very high risk for deterioration to torsades de pointes and ventricular fibrillation.
- Relatively short PR interval and possible delta waves (leads I, II, V6) are suggestive — but not diagnostic — of WPW syndrome.
- Voltage criteria for left ventricular hypertrophy are present in multiple leads.

This patient had suffered a cardiac arrest in the context of severe hypertrophic cardiomyopathy and long QT syndrome.

(NB. ~1/3 of patients with HOCM will have some evidence of WPW on their ECG).

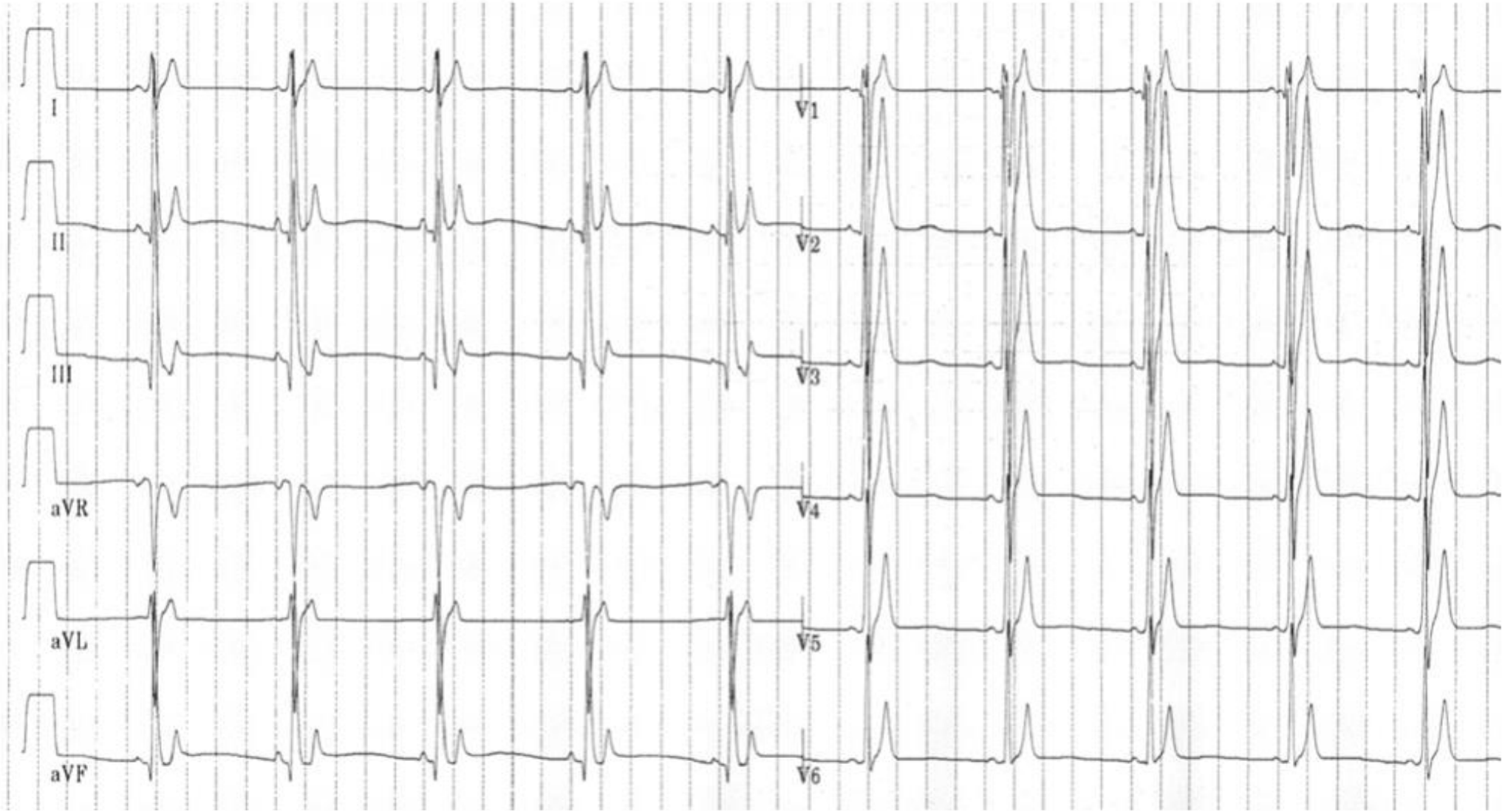
Can you guess what happened next?

+ Reveal Answer

- The patient had a further TdP cardiac arrest!
- This was treated with IV magnesium and potassium, with restoration of sinus rhythm.

This interesting case is discussed in more detail here.

16-year old male presenting with syncope. Describe and interpret his ECG.



Main Abnormalities

- Markedly peaked T waves in V2-6.
- Extremely short QT interval (~240 ms).

In a patient presenting with syncope, this ECG pattern is very suspicious for the short QT syndrome.

Short QT Syndrome

- This is a recently-described arrhythmogenic condition associated with paroxysmal atrial and ventricular fibrillation.
- The hallmark is a significantly shortened QT interval (at least < 360 ms, often < 330 ms) with associated T-wave peaking.
- SQTS is a genetically-inherited cardiac channelopathy on the same spectrum as other familial arrhythmogenic diseases such as Long QT Syndrome (LQTS), Brugada Syndrome and Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT).

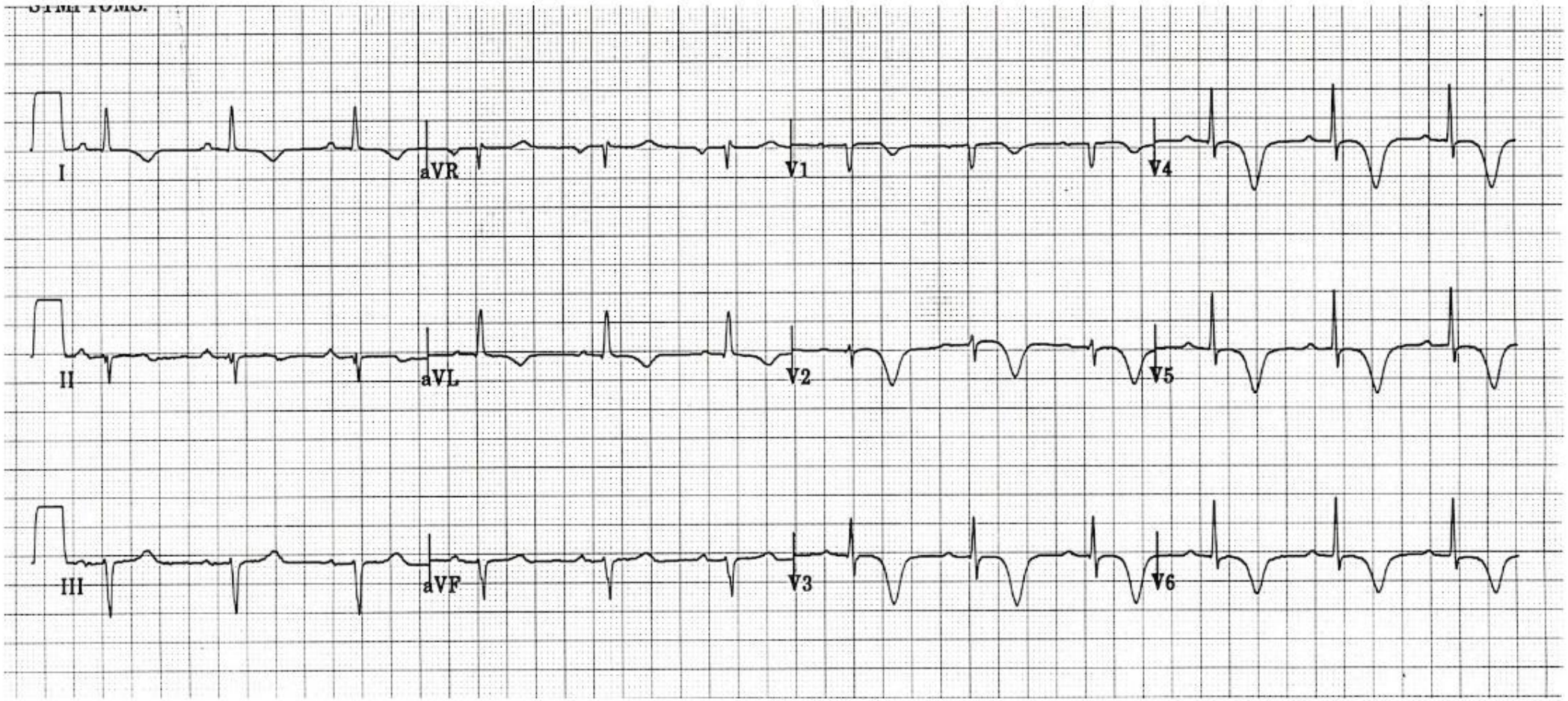
When to Suspect SQTS

- Any patient with a QT interval < 330 ms.
- QT interval < 360 ms and convincing symptoms (syncope, cardiac arrest) or family history.

Differential Diagnosis

- Peaked T waves and short QT interval may be seen with severe hyperkalaemia.
- A shortened QT interval may also be seen with severe hypercalcaemia.

Middle-aged patient presenting with an episode of chest pain. Currently asymptomatic. Describe and interpret his ECG



Reveal Answer

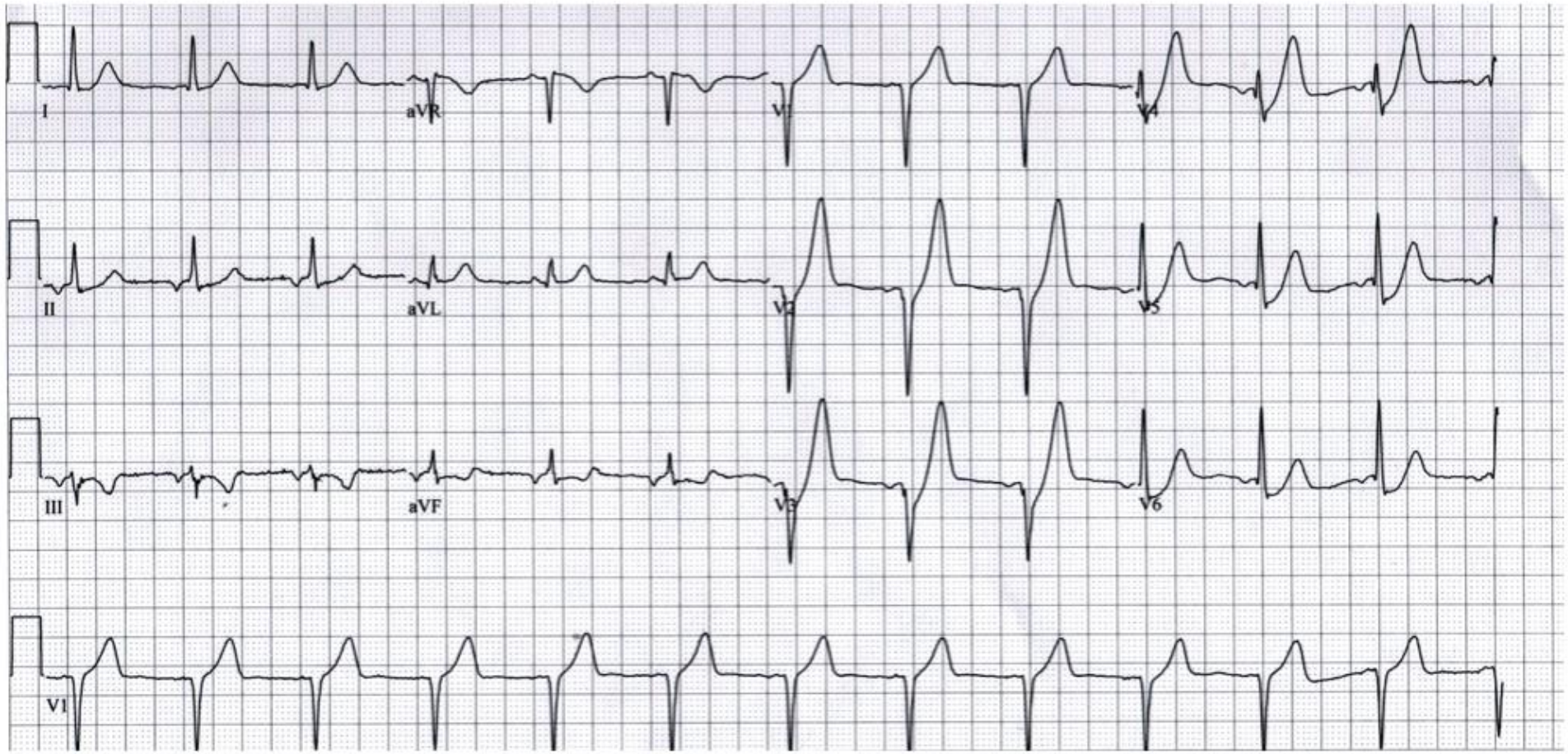
- This pattern of deeply inverted T waves in the anterolateral leads V2-6, I and aVL is characteristic of Wellens syndrome.
- This ECG pattern is highly predictive of a significant occlusive lesion of the LAD.
- The inverted T waves are a marker of reperfusion and may occur after an aborted anterior STEMI.
- Despite often being pain free and having normal cardiac enzymes at presentation, these patients are at risk of sudden LAD re-occlusion leading to massive anterior STEMI and are best managed with early angiography and PCI / CABG.

Differential Diagnosis

A similar pattern of deep anterolateral T-wave inversions may also be seen with:

- Apical hypertrophic cardiomyopathy – suspect if associated LVH
- Raised intracranial pressure – patient will be comatose (see Quiz ECG #12)

Middle aged patient presenting with chest pain and diaphoresis. Describe and interpret his ECG



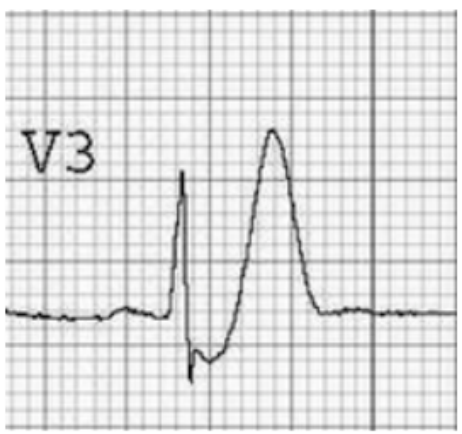
ECG courtesy of Mat Goebel

Main Abnormalities

- ST depression in V2-6, which slopes upwards and joins the ascending limb of the T wave.
- Prominent, “rocket-shaped” T waves in the precordial leads V2-5.
- Subtle ST elevation in aVR.

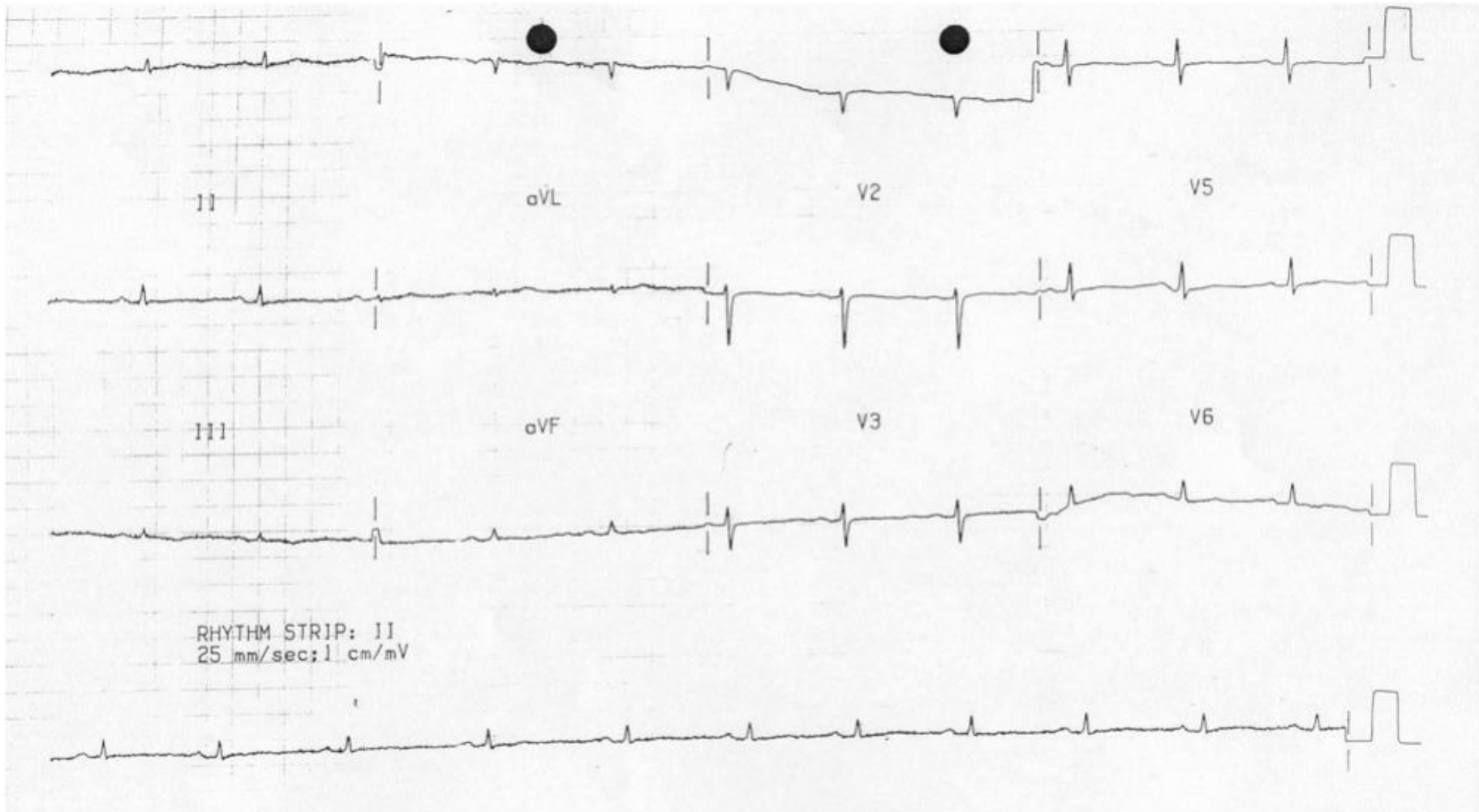
Diagnosis

- This combination of ST depression with rocket-shaped T waves in the precordial leads V1-6 is referred to as the De Winter ECG pattern or “De Winter’s T waves” (also see ECG #19)
- It is becoming increasingly recognised as an anterior STEMI equivalent (~2% of LAD occlusions).
- Some authors are now recommending that this ECG pattern be treated identically to anterior STEMI, with urgent PCI or thrombolysis.



Typical De Winter’s T Wave

Elderly patient presenting with reduced level of consciousness, hypothermia and hypotension refractory to inotropes. Describe and interpret his ECG



The most striking abnormality on this ECG is extremely low QRS voltage, in this case due to severe myxoedema.

Definition

The QRS is said to be low voltage when:

> - The amplitudes of all the QRS complexes in the limb leads are < 5 mm; or
> - The amplitudes of all the QRS complexes in the precordial leads are < 10 mm

Mechanisms

Low voltage is produced by…

> - The “damping” effect of increased layers of fluid, fat or air between the heart and the recording electrode.
> - Loss of viable myocardium.
> - Diffuse infiltration or myxoedematous involvement of the heart.

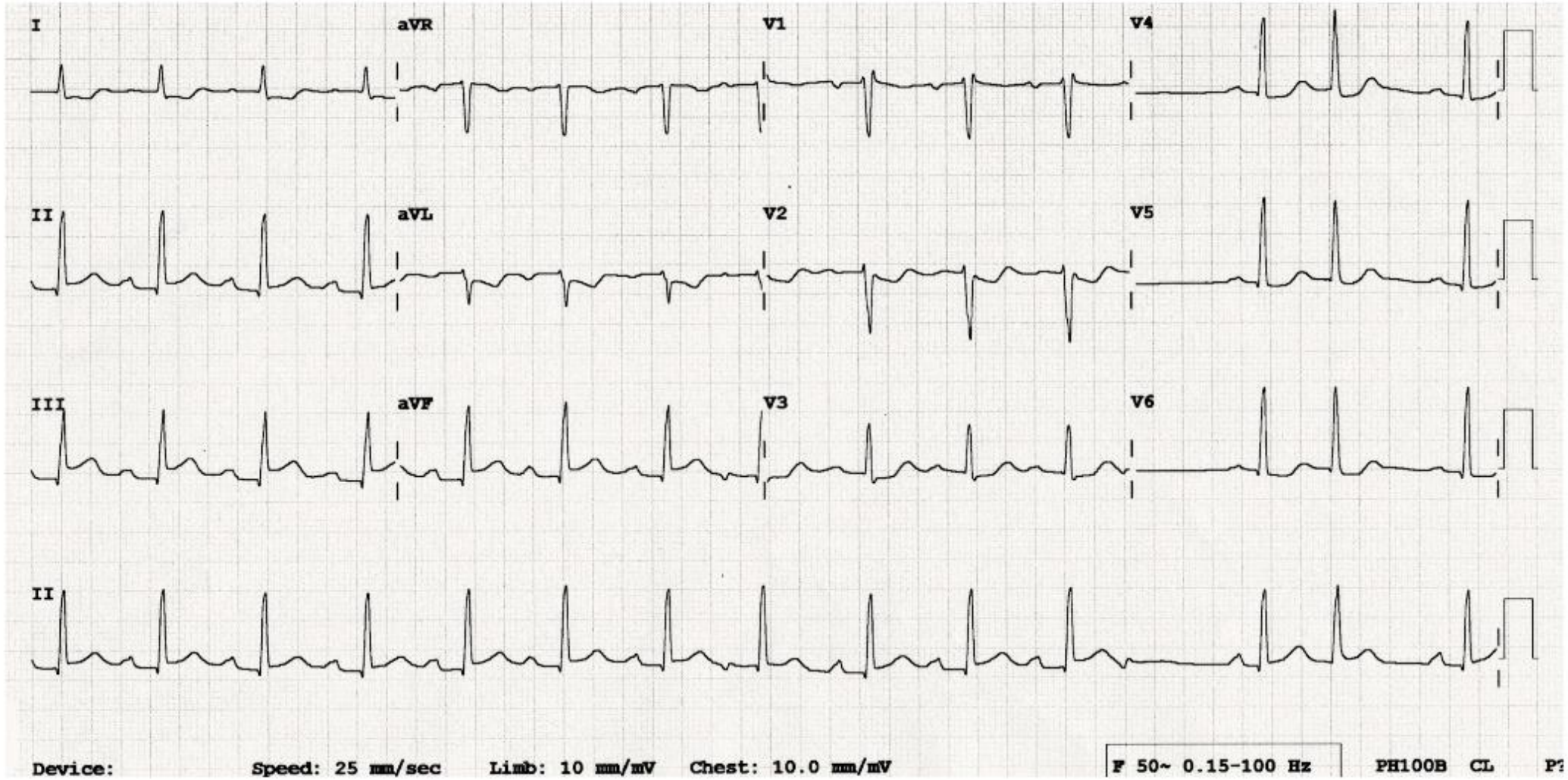
Specific causes of low voltage include:

> **Fluid**
>
> - Pericardial effusion
> - Pleural effusion
>
> **Fat**
>
> - Obesity
>
> **Air**
>
> - Emphysema
> - Pneumothorax
>
> **Infiltrative / Connective Tissue Disorders**
>
> - Myxoedema
> - Infiltrative myocardial diseases — i.e. restrictive cardiomyopathy due to amyloidosis, sarcoidosis, haemochromatosis
> - Constrictive pericarditis
> - Scleroderma
>
> **Loss of viable myocardium**
>
> - Previous massive MI
> - End-stage dilated cardiomyopathy

The most important cause is **massive pericardial effusion**, which produces a triad of:

> - Low voltage
> - Tachycardia
> - Electrical alternans

Chest pain and diaphoresis. BP 80/50. Describe and interpret his ECG



Key Abnormalities

- There is ST elevation in the inferior leads II, III and aVF.
- The concave morphology might lead you to suspect pericarditis — however, there is reciprocal change in the high lateral leads I and aVL, confirming the diagnosis of inferior STEMI.

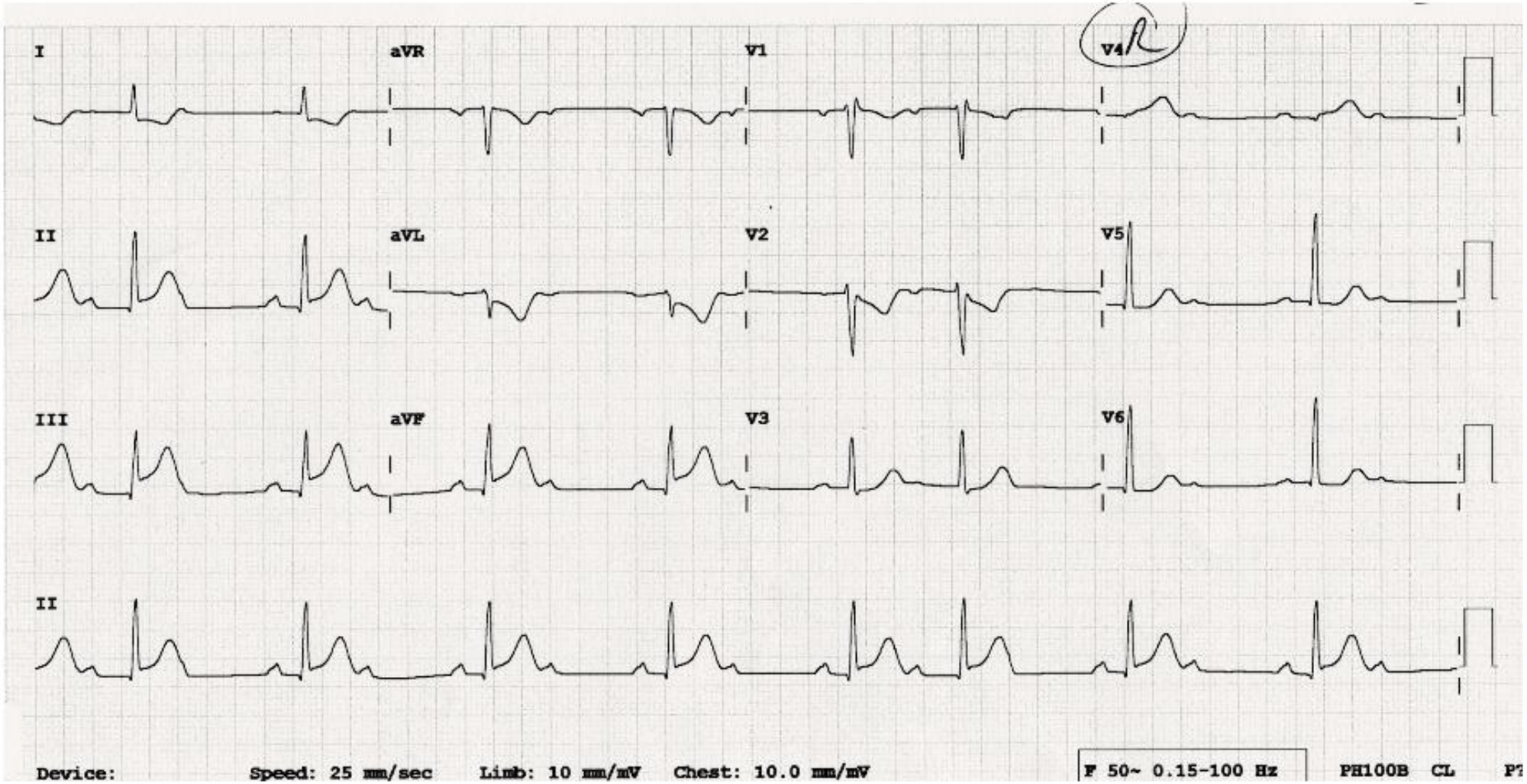
There are additional features suggestive of right ventricular infarction:

- ST elevation in III > II
- Isoelectric ST segment in V1 with ST depression in V2

Other Abnormalities

- There is a break in the rhythm towards the end of the rhythm strip, with what appears to be a non-conducted P wave, suggesting the development of 2nd degree AV block — e.g. a slowly-evolving Wenckbach cycle.
- The 13th QRS complex appears to be a supraventricular ectopic beat (PAC or PJC).

Chest pain and diaphoresis. BP 80/50. Describe and interpret his ECG



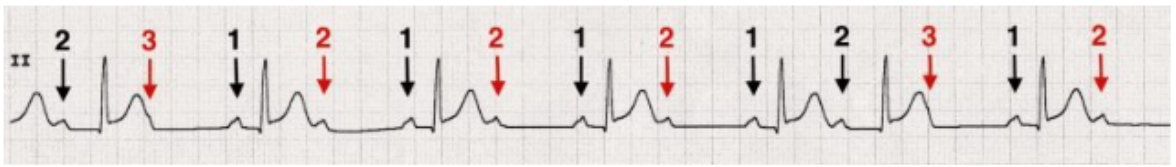
Reveal Answer

This is a repeat ECG of the previous patient, demonstrating:

- Inferior STEMI — STE in II, III, aVF with reciprocal change in I and aVL.
- Evidence suggesting RV infarction — STE in III > II.
- Evidence confirming RV infarction — STE and hyperacute T wave in V4R.
- Evolving second degree AV block with alternating 2:1 block and 3:2 Wenckebach cycles.

This ECG pattern is diagnostic of a right coronary artery occlusion.

Rhythm Strip Explanation



- Arrows indicate the position of P waves.
- Black arrrows indicate conducted P waves.
- Red arrows indicate *non-conducted* P waves — some of these are concealed within the preceding T wave, causing a small bump at the back of the T wave.
- Complexes cluster together in groups with either 2:1 conduction or as 3:2 Wenckeback cycles, with prolongation of the PR interval prior to the non-conducted P wave.
- The number above each P wave denotes its position in the sequence.

Bradycardia and AV Block in Inferior STEMI

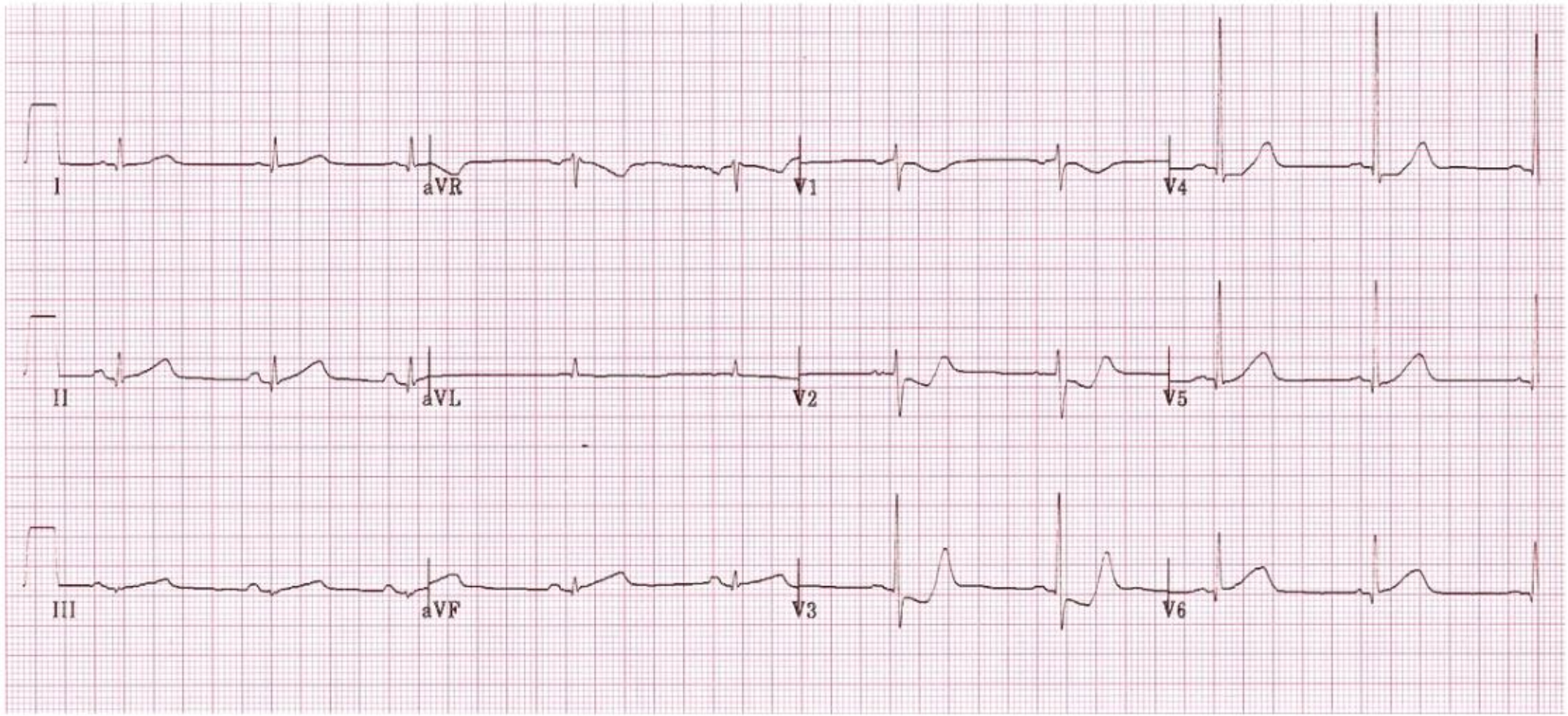
Up to 20% of patients with inferior STEMI will develop either second- or third degree heart block.

There are two presumed mechanisms for this:

> - **Ischaemia of the AV node** due to impaired blood flow via the AV nodal artery. This artery arises from the RCA 80% of the time, hence its involvement in inferior STEMI due to RCA occlusion.
> - **Bezold-Jarisch reflex** = increased vagal tone secondary to ischaemia.

- The conduction block may develop either as a step-wise progression from 1st degree heart block via Wenckebach to complete heart block (in 50% of cases) or as abrupt onset of second or third-degree heart block (in the remaining 50%).
- Patients may also manifest signs of sinus node dysfunction, such as sinus bradycardia, sinus pauses, sinoatrial exit block and sinus arrest. Similarly to AV node dysfunction, this may result from increased vagal tone or ischaemia of the SA node (the SA nodal artery is supplied by the RCA in 60% of people).
- Bradyarrhythmias and AV block in the context of inferior STEMI are usually transient (lasting hours to days), respond well to atropine and do not require permanent pacing.

Elderly patient presenting with chest pain and diaphoresis. Describe and interpret his ECG



Reveal Answer

This ECG demonstrates an infero-posterior STEMI, as manifest by:

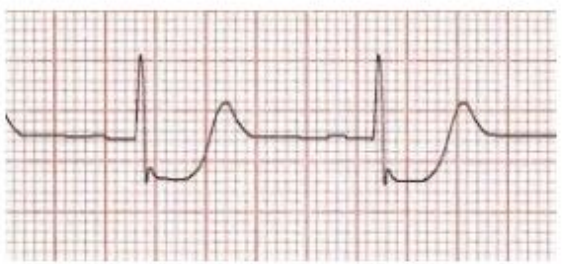
- Subtle inferior STE and hyperacute T waves (T waves > QRS complexes)
- ST depression and terminal T-wave positivity in V2-3 = posterior wall involvement

See Quiz ECG 14 for another example of posterior infarction.

Tips for spotting posterior infarction

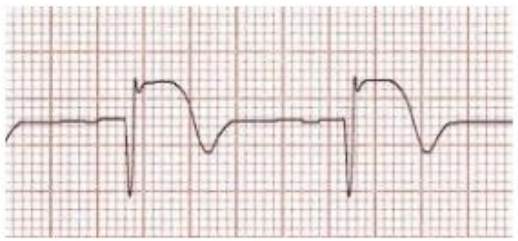
Look specifically at leads V2-3 for the combination of:

- Horizontal ST depression.
- Tall, broad R wave (>30ms wide, R/S ratio > 1) — this is a Q-wave equivalent.
- Upright T wave — particularly the terminal portion of the T wave.



Typical appearance of posterior infarction in V2-3

One common trick is to turn the ECG over, hold it up to the light and look through it from behind. This invert lead V2, which then takes on the appearance of a classic STEMI.



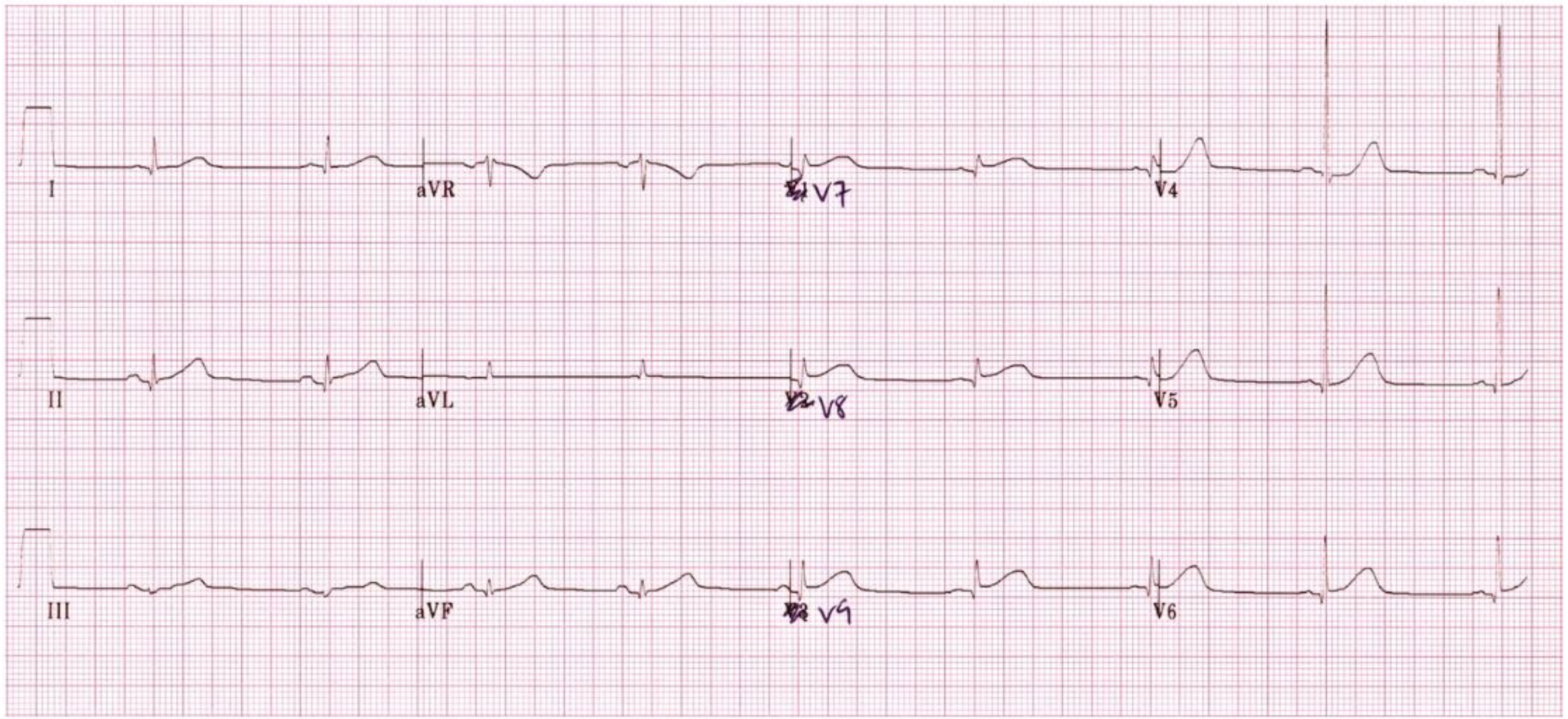
Inverted ECG — the complexes now resemble a typical STEMI

In this ECG example, V3 is the most characteristically abnormal lead.

Look for evidence of posterior involvement in any patient with an inferior or lateral STEMI.

Sometimes it can be difficult to determine whether ST depression in V2-3 is due to posterior STEMI or simply subendocardial ischaemia affecting the anteroseptal wall. The diagnosis can be confirmed by recording posterior leads V7-9.

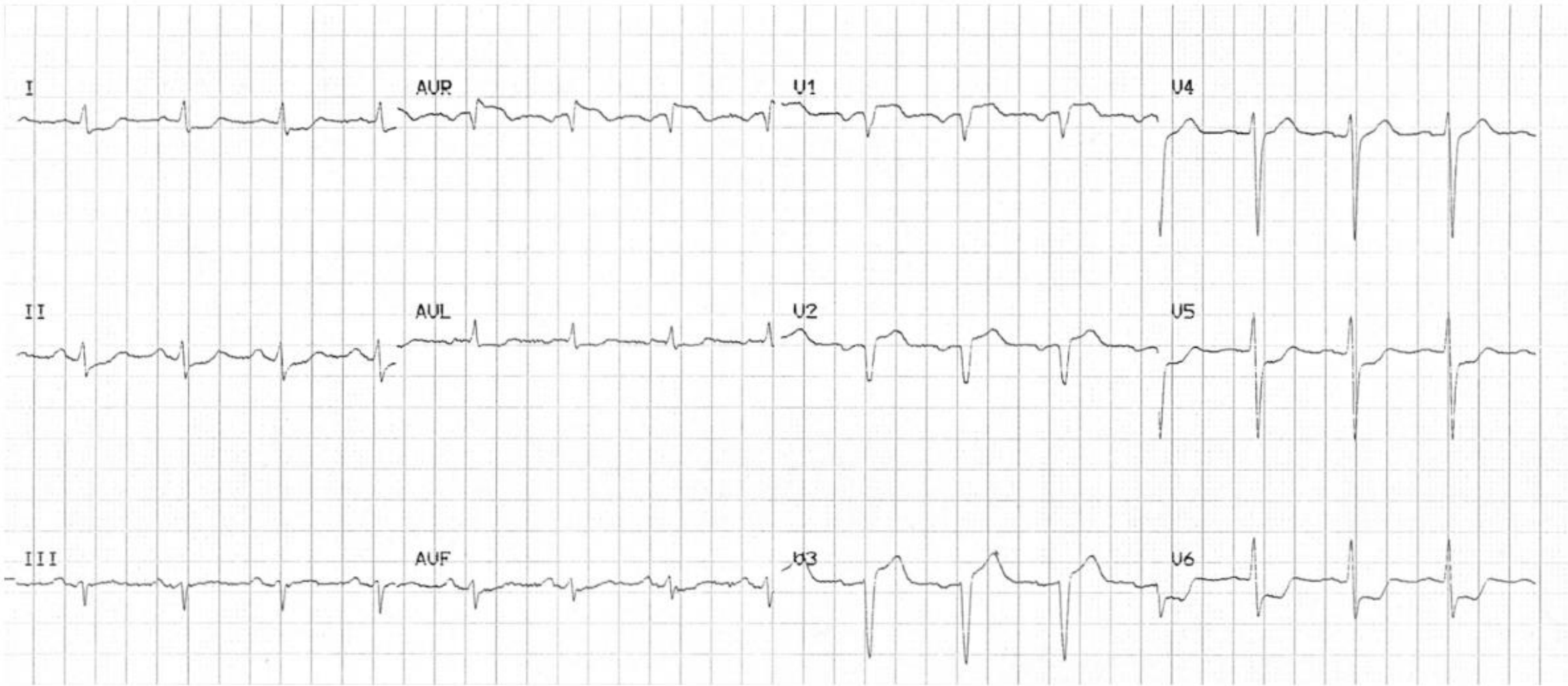
Elderly patient with chest pain and diaphoresis. Describe and interpret his ECG



Reveal Answer

- This is the same patient as ECG 066.
- Posterior leads V7-9 show subtle ST elevation with early Q-wave formation, confirming the presence of posterior infarction.

Severe chest pain and hypotension (70/40) in an elderly man. Describe and interpret his ECG



Reveal Answer

This ECG shows:

- ST elevation in aVR.
- ST depression in multiple leads (V5-6, I, II, aVL, aVF).
- Evidence of anteroseptal STEMI – ST elevation with Q wave formation in V1-3.

In the context of ischaemic chest pain and cardiogenic shock, the combination of…

- Widespread ST depression
- ST elevation in aVR > 1 mm
- ST elevation in V1-3

… is concerning for proximal LAD occlusion (compare this to the LMCA pattern seen in Quiz ECG 008).

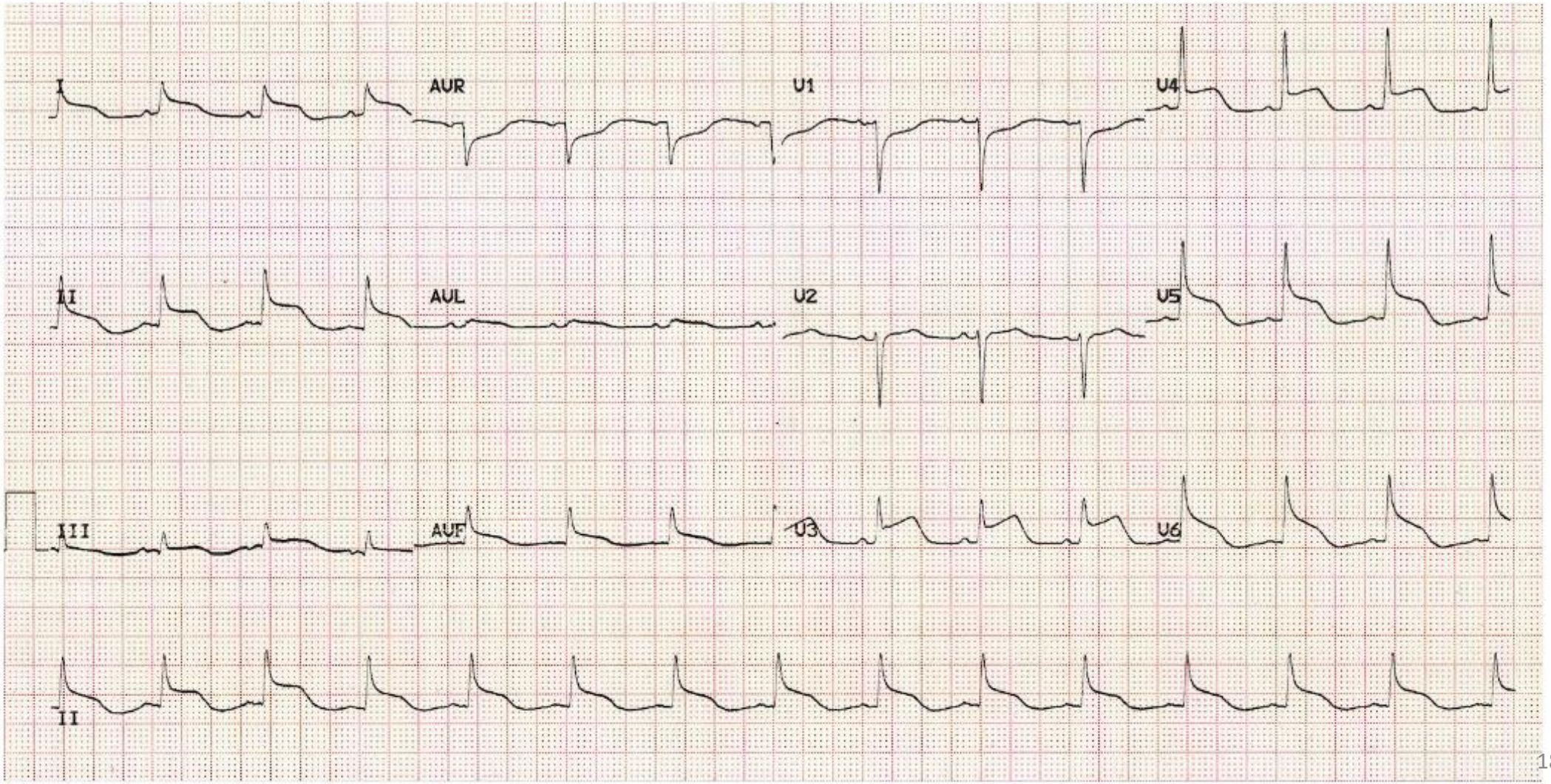
While this pattern of diffuse ST depression with STE in aVR if often referenced as a marker of LMCA / proximal LAD occlusion, may be seen whenever there is diffuse severe subendocardial ischaemia, e.g.

- Severe triple vessel disease
- Severe anaemia or hypoxaemia
- Following resuscitation from cardiac arrest

Outcome

*This patient actually had **severe multi-vessel disease**. Angiography demonstrated a chronic total occlusion of his circumflex artery, with critical stenoses of his proximal LAD, RCA and ramus intermedius. Surprisingly, in this case the culprit vessel was thought to be the RCA, which had been collateralising his chronically occluded circumflex. He went on to receive a CABG x 4.*

18-year old female with severe traumatic brain injury, ICP 40mmHg. Fluctuating BPs. Describe and interpret her ECG



Reveal Answer

- The ECG shows diffuse ST elevation.
- The morphology is atypical, there is no clear anatomical predominance for a vascular territory and no obvious reciprocal changes (except the inverted leads V1 and aVR).
- This is an example of a pseudo-infarction pattern due to raised intracranial pressure.
- Compare this with Quiz ECG 012 (raised ICP causing giant T wave inversions).

ECG changes with elevated ICP

Raised ICP is associated with certain characteristic ECG changes:

> - Widespread giant T-wave inversions ("cerebral T waves").
> - QT prolongation.
> - Bradycardia *(the Cushing reflex – indicates imminent brainstem herniation).*

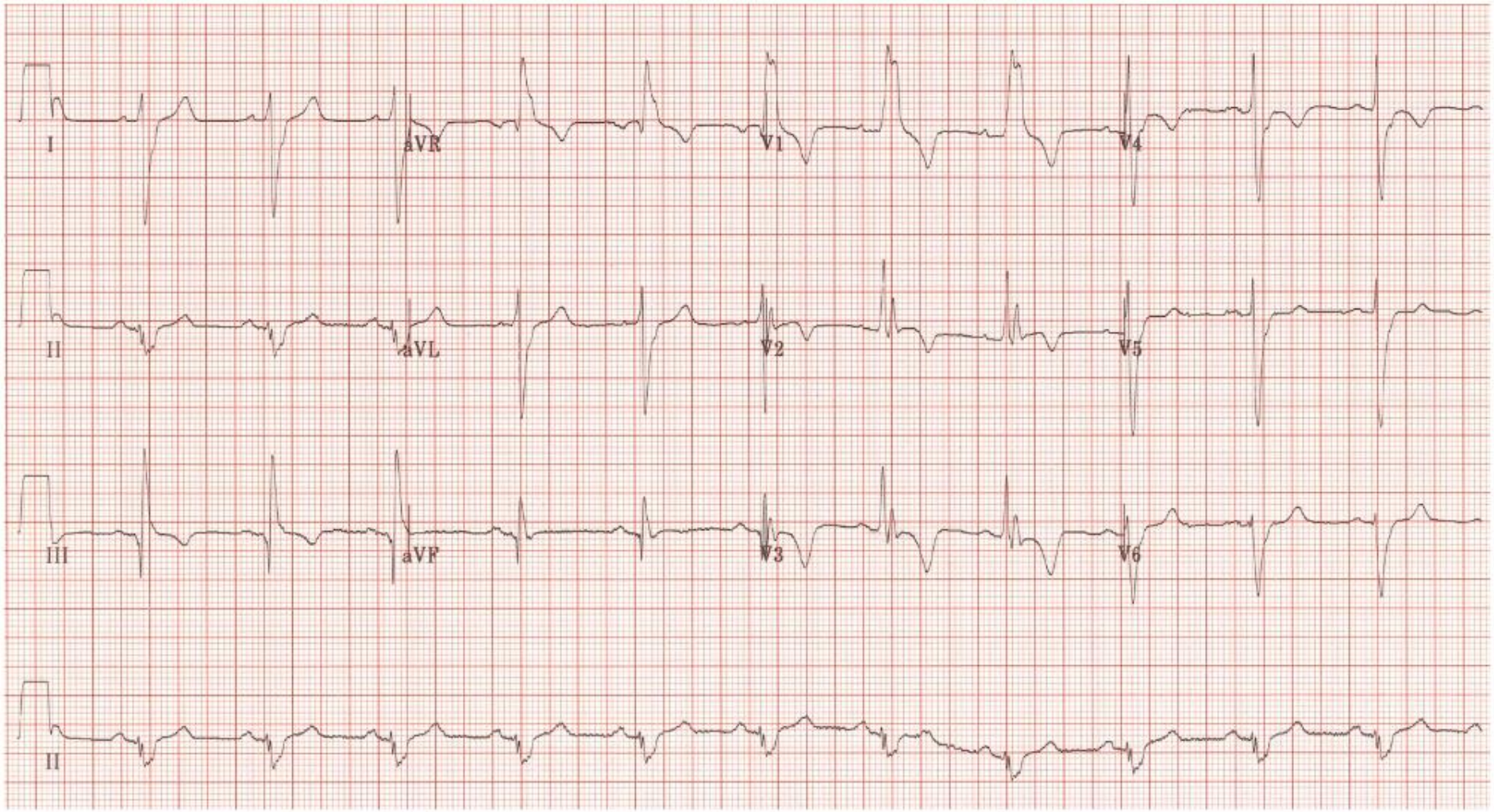
Other possible ECG changes that may be seen:

> - ST segment elevation / depression — *this may mimic myocardial ischaemia or pericarditis.*
> - Increased U wave amplitude.
> - Other rhythm disturbances: sinus tachycardia, junctional rhythms, premature ventricular contractions, atrial fibrillation.

In some cases, these ECG abnormalities may be associated with echocardiographic evidence of regional ventricular wall motion abnormality — so-called neurogenic stunned myocardium or neurogenic stress cardiomyopathy. The presumed mechanism is massive release of catecholamines, similar to Takutsubo syndrome.

This patient developed labile blood pressures and transient wall motion abnormalities plus these ECG changes during a sustained spike in her ICP. The ECG changes and wall motion abnormalities improved once her ICPs came under control.

Elderly patient presenting with sudden onset of chest pain and shortness of breath. Hypoxic (SaO2 82% RA) and hypotensive (80/50). Describe and interpret his ECG



- Right axis deviation
- Dominant R wave in V1
- Right bundle branch block
- Right ventricular strain pattern — T wave inversions in V1-4, lead III
- S_I Q_{III} T_{III} pattern
- Clockwise rotation of the heart, with a persistent S wave in V6

Given the clinical history, the most likely scenario is acute right heart strain due to massive pulmonary embolism.

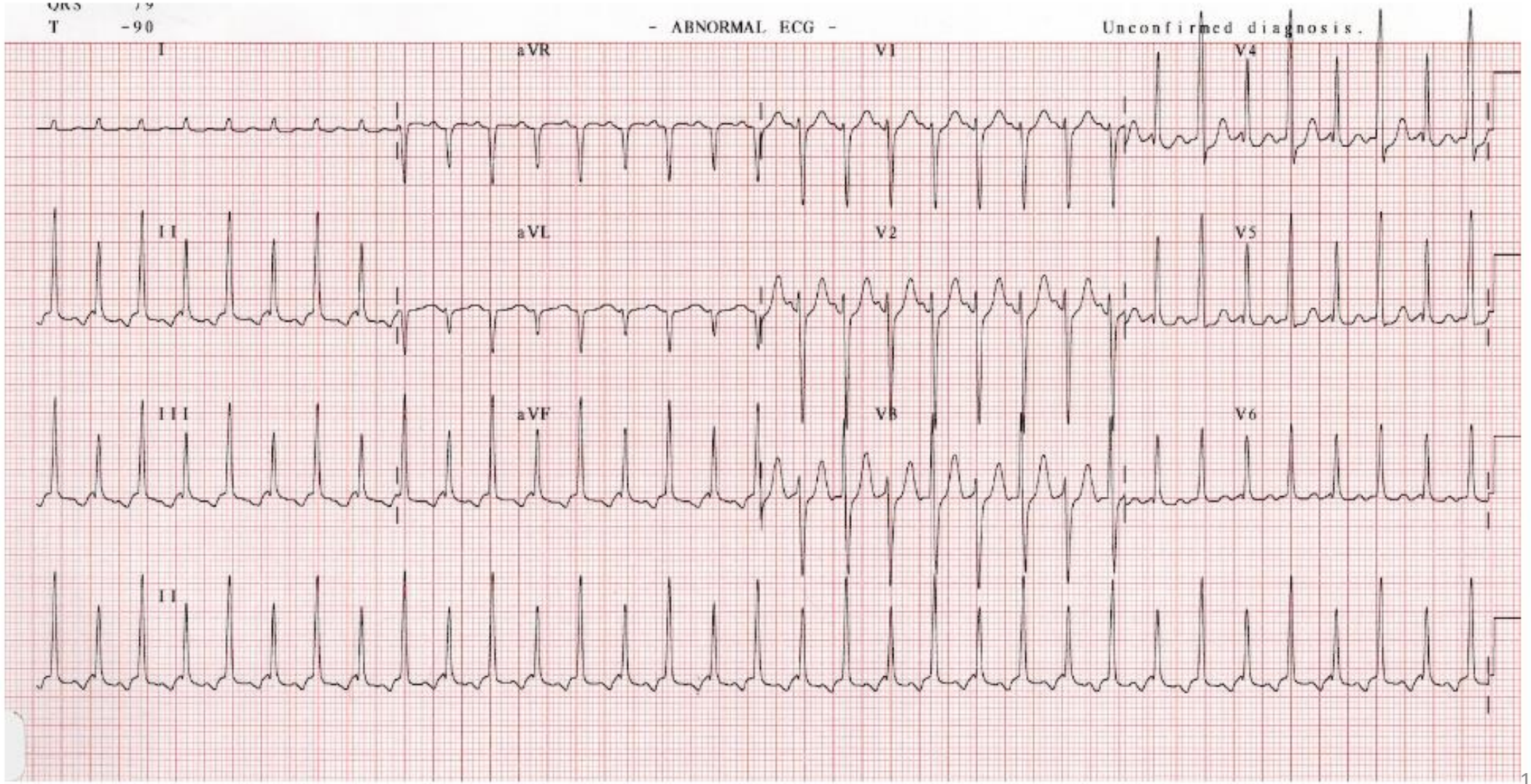
A similar ECG pattern may also be seen with chronic cor pulmonale, pulmonary hypertension or RV hypertrophy (e.g. due to congenital heart disease).

ECG changes in pulmonary embolism include:

- Sinus tachycardia **–** the most common abnormality; seen in 44% of patients.
- Complete or incomplete RBBB **–** associated with increased mortality; seen in 18% of patients.
- Right ventricular strain pattern **–** T wave inversions in the right precordial leads (V1-4) ± the inferior leads (II, III, aVF). This pattern is seen in up to 34% of patients and is associated with high pulmonary artery pressures.
- Right axis deviation – seen in 16% of patients. Extreme right axis deviation may occur, with axis between zero and -90 degrees, giving the appearance of left axis deviation ("pseudo left axis").
- Dominant R wave in V1 – a manifestation of acute right ventricular dilatation.
- Right atrial enlargement (P pulmonale) **–** peaked P wave in lead II > 2.5 mm in height. Seen in 9% of patients.
- **S_I Q_{III} T_{III} pattern** – deep S wave in lead I, Q wave in III, inverted T wave in III. This "classic" finding is neither sensitive nor specific for pulmonary embolism; found in only 20% of patients with PE.
- Clockwise rotation **–** shift of the R/S transition point towards V6 with a persistent S wave in V6 ("pulmonary disease pattern"), implying rotation of the heart due to right ventricular dilatation.
- **Atrial tachyarrhythmias –** AF, flutter, atrial tachycardia. Seen in 8% of patients.
- **Non-specific ST segment and T wave changes**, including ST elevation and depression. Reported in up to 50% of patients with PE.

Simultaneous T wave inversions in the inferior (II, III, aVF) and right precordial leads (V1-4) is the most specific finding in favour of PE.

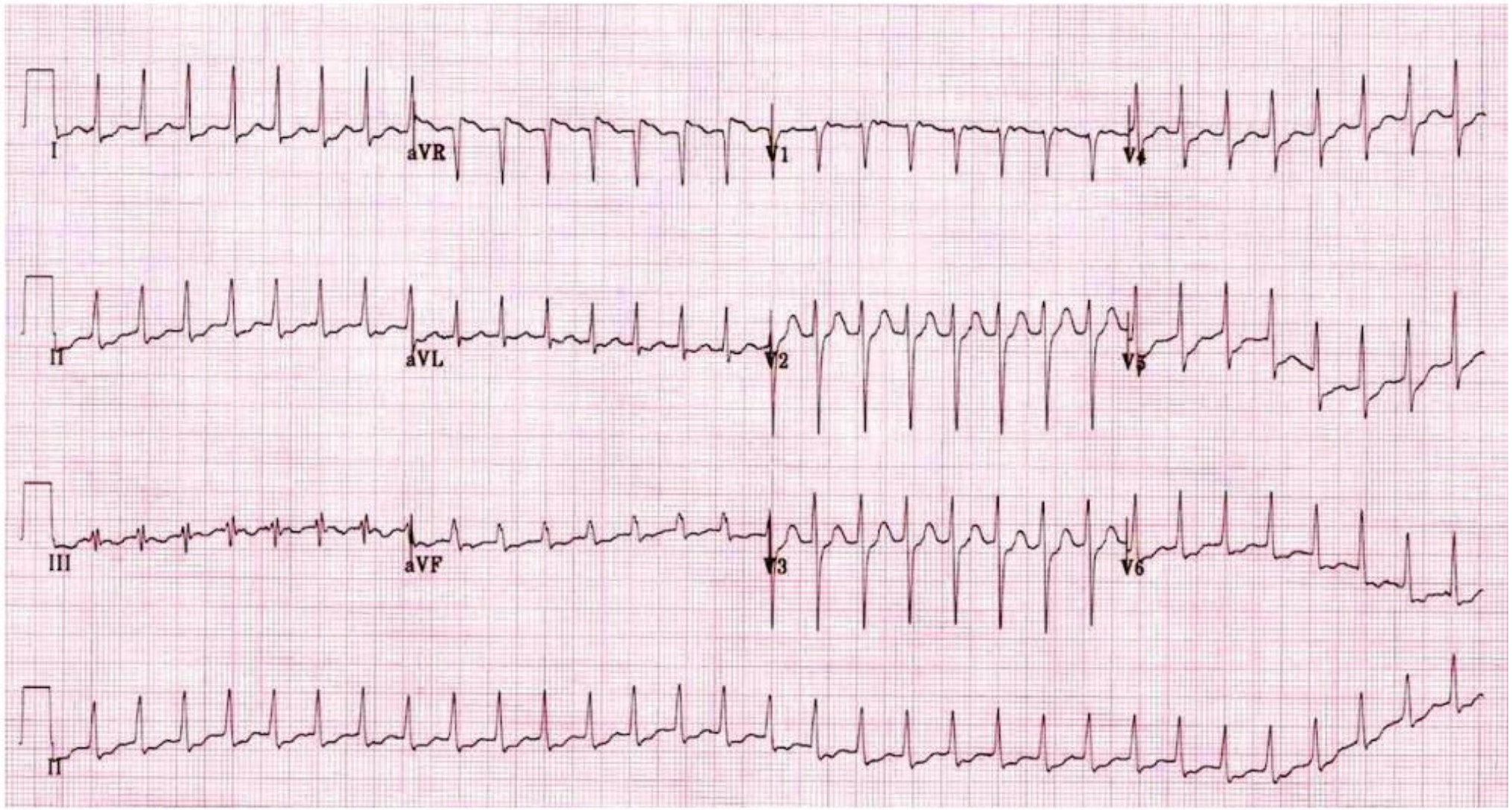
Palpitations. Describe and interpret this ECG



Reveal Answer

QRS alternans – due to AVNRT (i.e. electrical phenomenon), not pericardial effusion as normal voltages.

Palpitations, chest heaviness

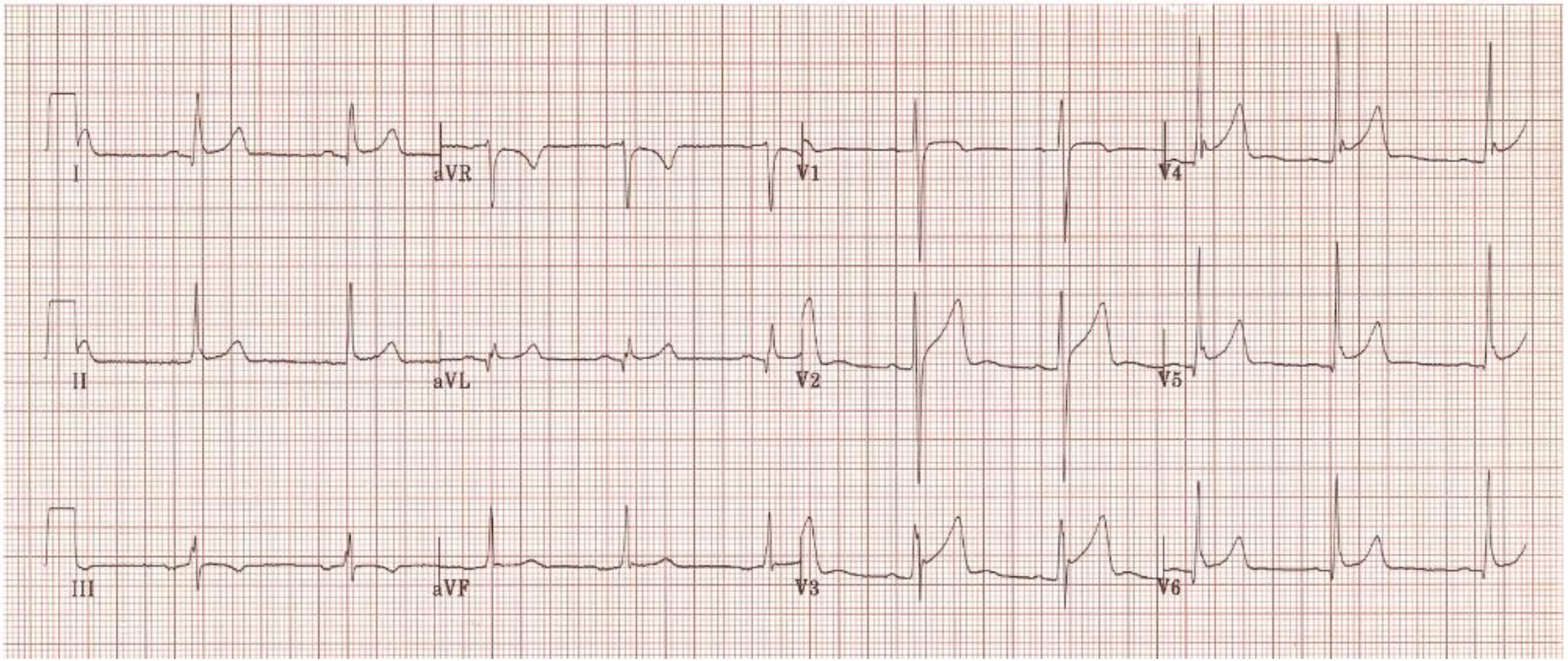


Reveal Answer

Rate-related ST change with SVT = not necessarily ischaemic.

Mimics appearance of LMCA occlusion.

Pleuritic chest pain after weight lifting. Describe and interpret this ECG



Reveal Answer

Benign early repolarisation.