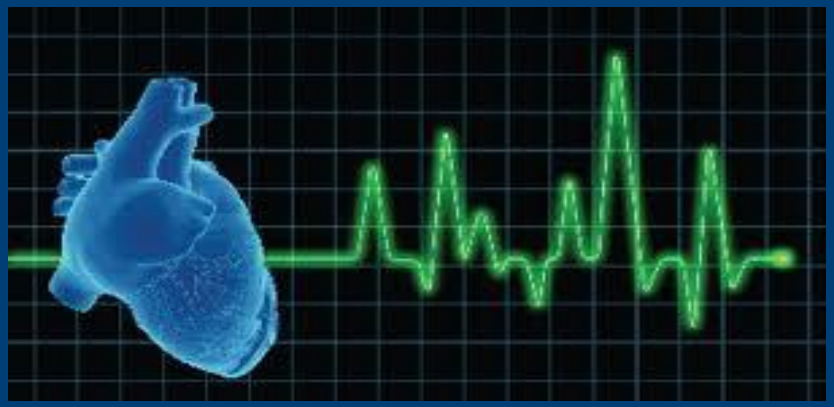
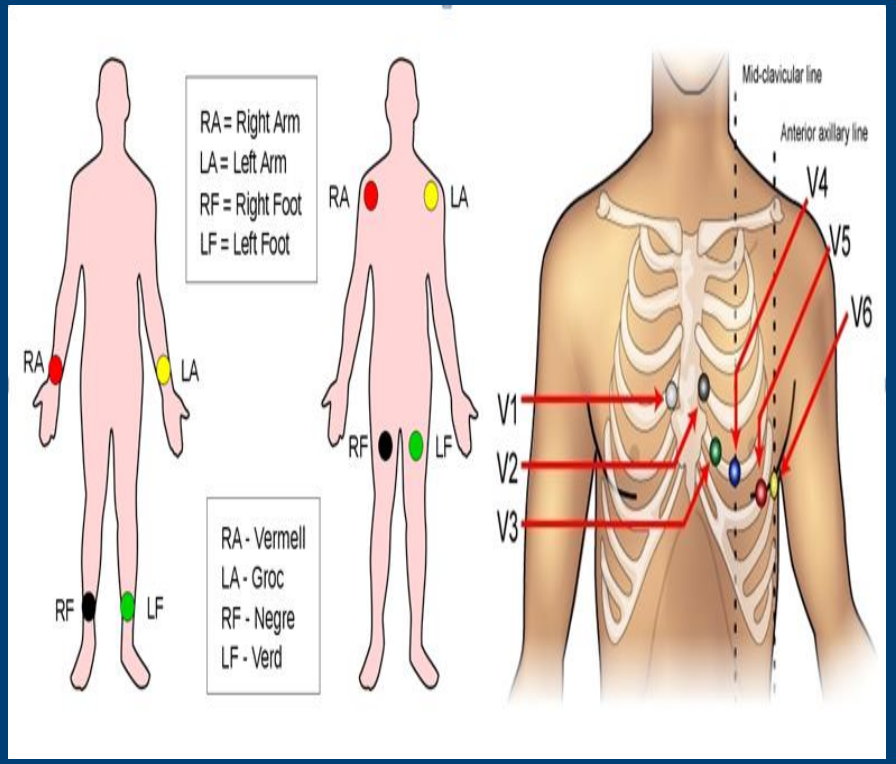
ECG



Dr.BSHARA AL-BEQAEEN
CONSULTANT INTERNAL MEDICINE
AL BASHEER HOSPITAL

Electrode placement in 12 lead ECG

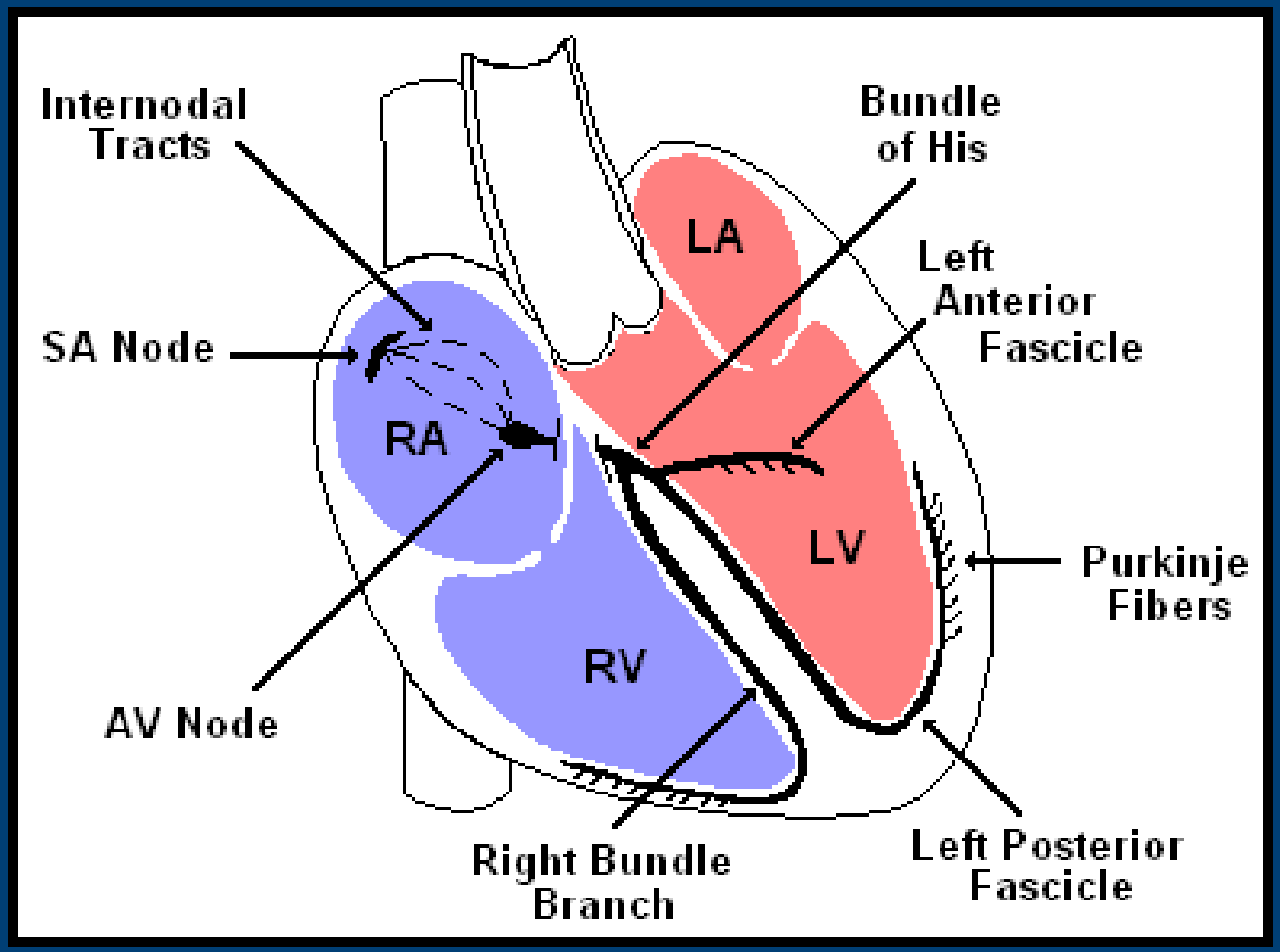
- 6 are chest electrodes
- Called V1-6 or C1-6
- 4 are limb electrodes
  - Right arm — Ride
  - Left arm — Your
  - Left leg — Green
  - Right leg — Bike



- Remember

The right leg electrode is a neutral or ""dummy""!

The His-Purkinje conduction system

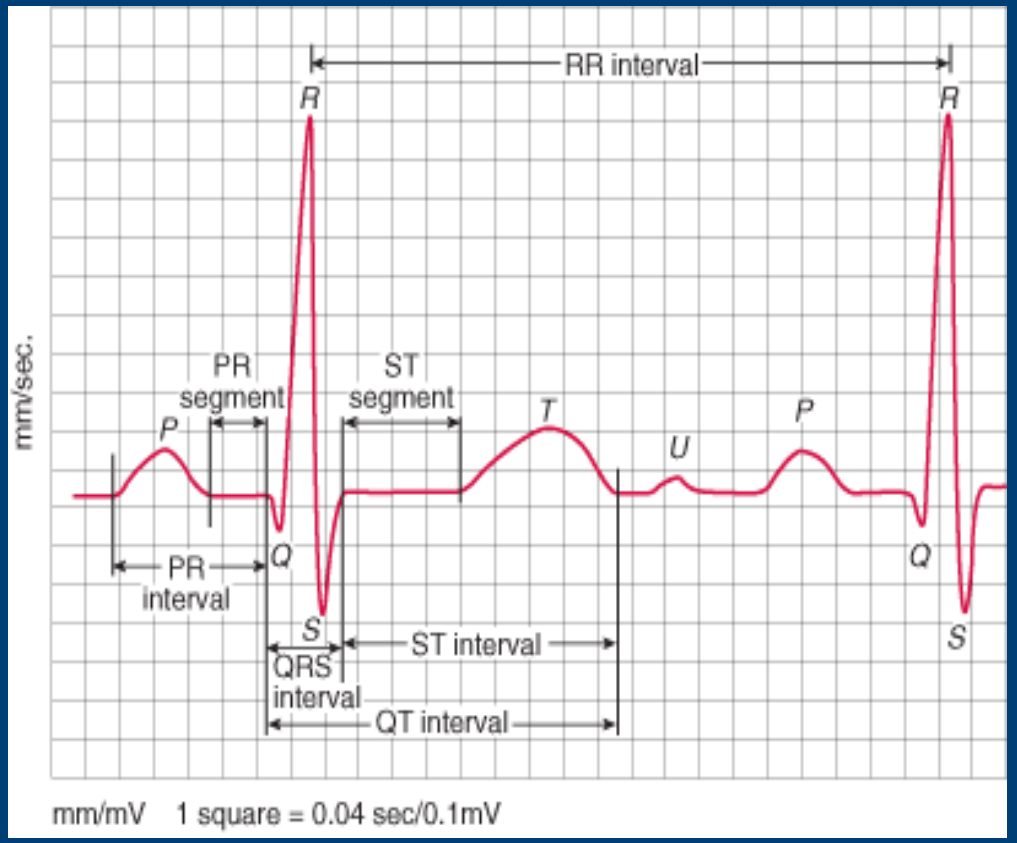


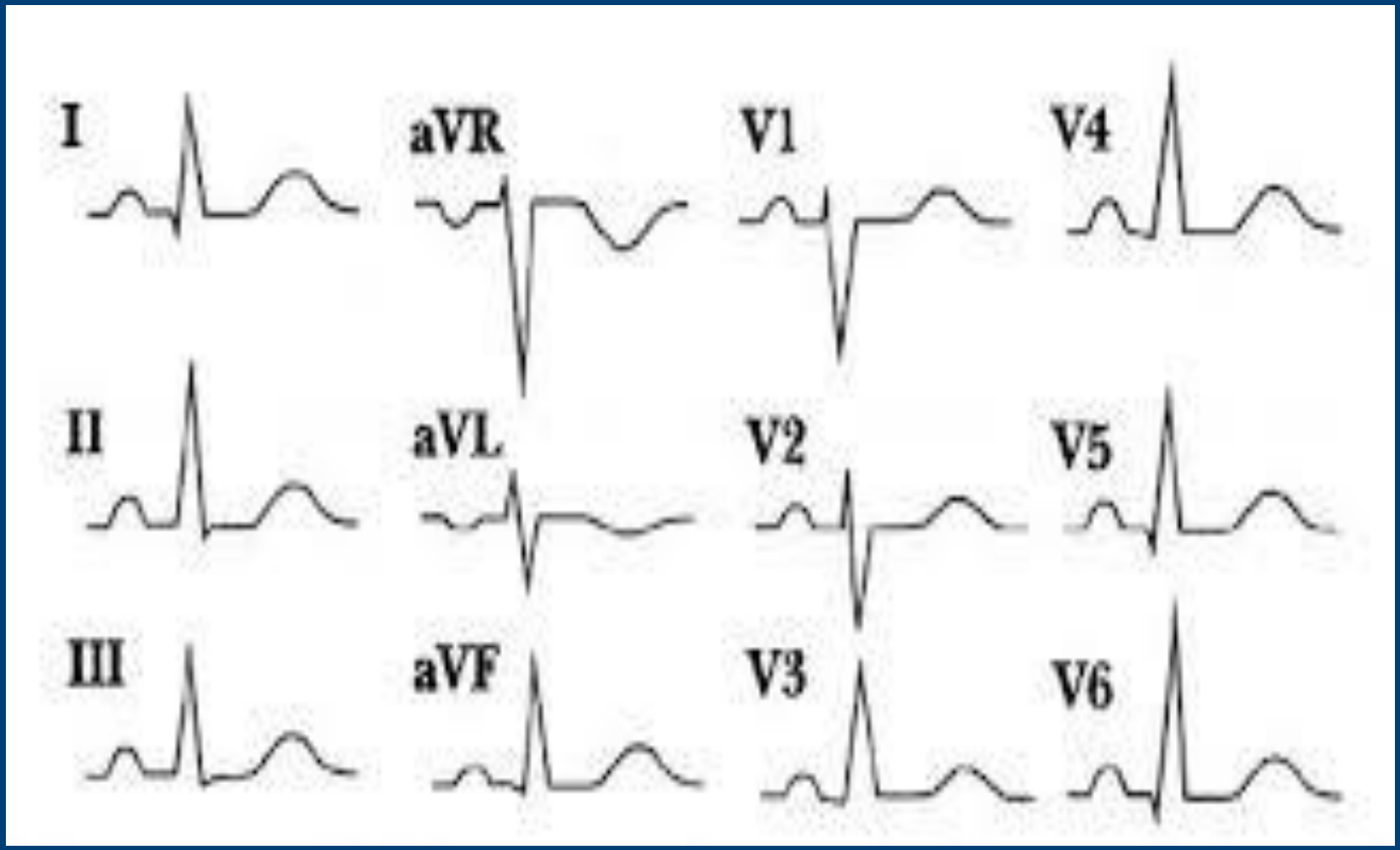
What do the components represent?

- P wave = atrial depolarisation
- QRS = ventricular depolarisation
- T = repolarisation of the ventricles

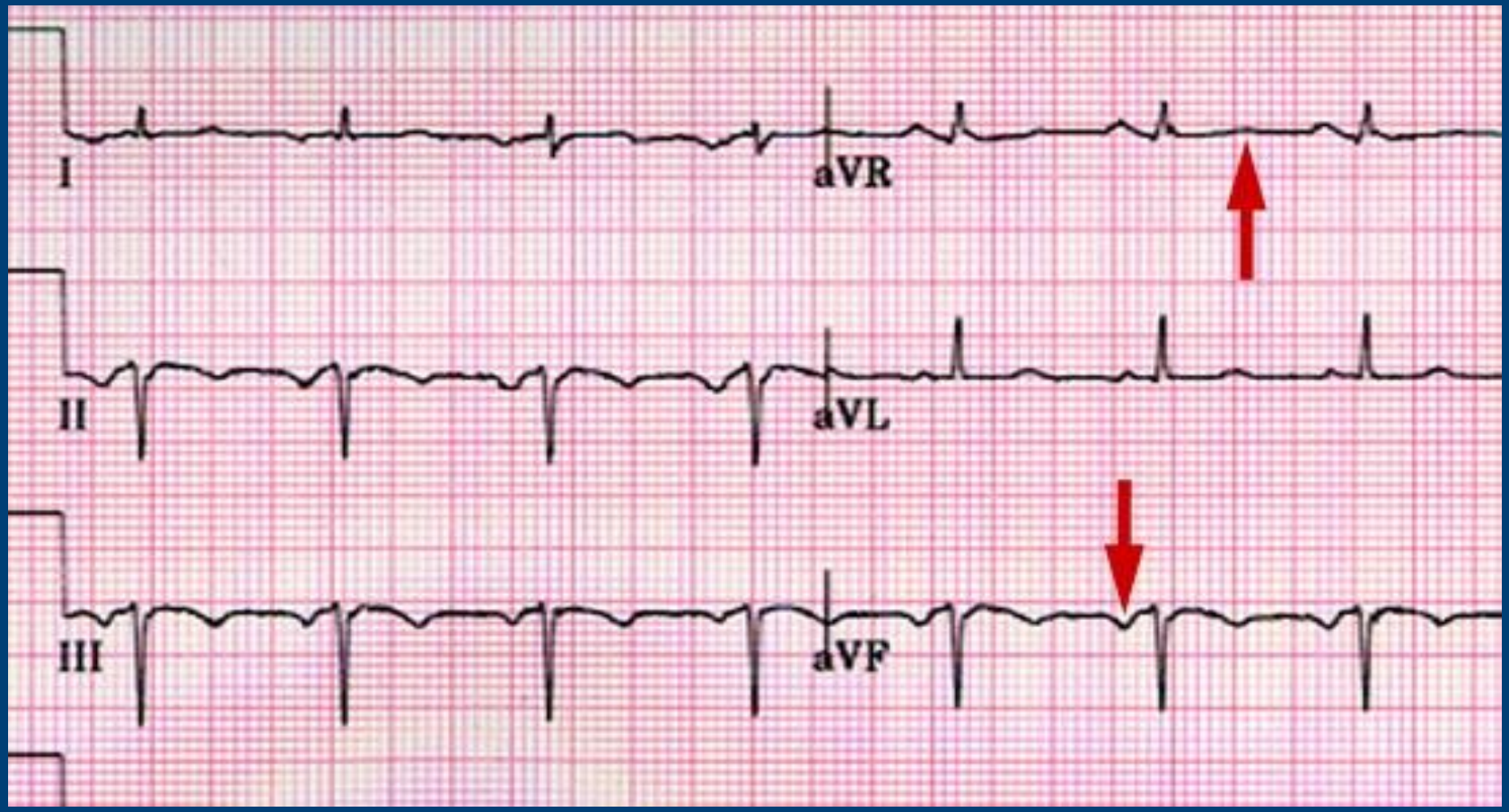
Normal ECG

- PR
  - 0.20 sec (less than one large box)
- QRS
  - 0.08 – 0.10 sec (1-2 small boxes)
- QT
  - 450 ms in men, 460 ms in women
  - Based on sex / heart rate
  - Half the R-R interval with normal HR




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

Wrong leads placement ECG



Start reading ECG with:

- *Check*
- Name
- DoB
- Time and date
- Rate
- Rhythm
- Axis

Normal waves in ECG

- A normal PR interval is between 0.12 and 0.2 seconds (3-5 small squares).
- A Q wave can be pathological if it is:
- Deeper than 2 small squares (0.2mV) and/or Wider than 1 small square (0.04s) and/or In a lead other than III or one of the leads that look at the heart from the left (I, II, aVL, , V_5 and V6) where small Qs (i.e. not meeting the criteria above) can be normal

CONT…

- The width of the QRS complex should be less than 0.12 seconds (3 small squares)
- If the QRS is wider than this, it suggests a ventricular conduction problem ,usually right or left bundle branch block (RBBB or LBBB)

- The ST segment should sit on the isoelectric line
- It is abnormal if there is planar (i.e. flat) elevation or depression of the ST segment

Characteristics of the P wave

- Positive in leads I and II
- Best seen in leads II and V1
- Commonly biphasic in lead V1
- < 3 small squares in duration
- < 2.5 small squares in amplitude

Abnormal P wave

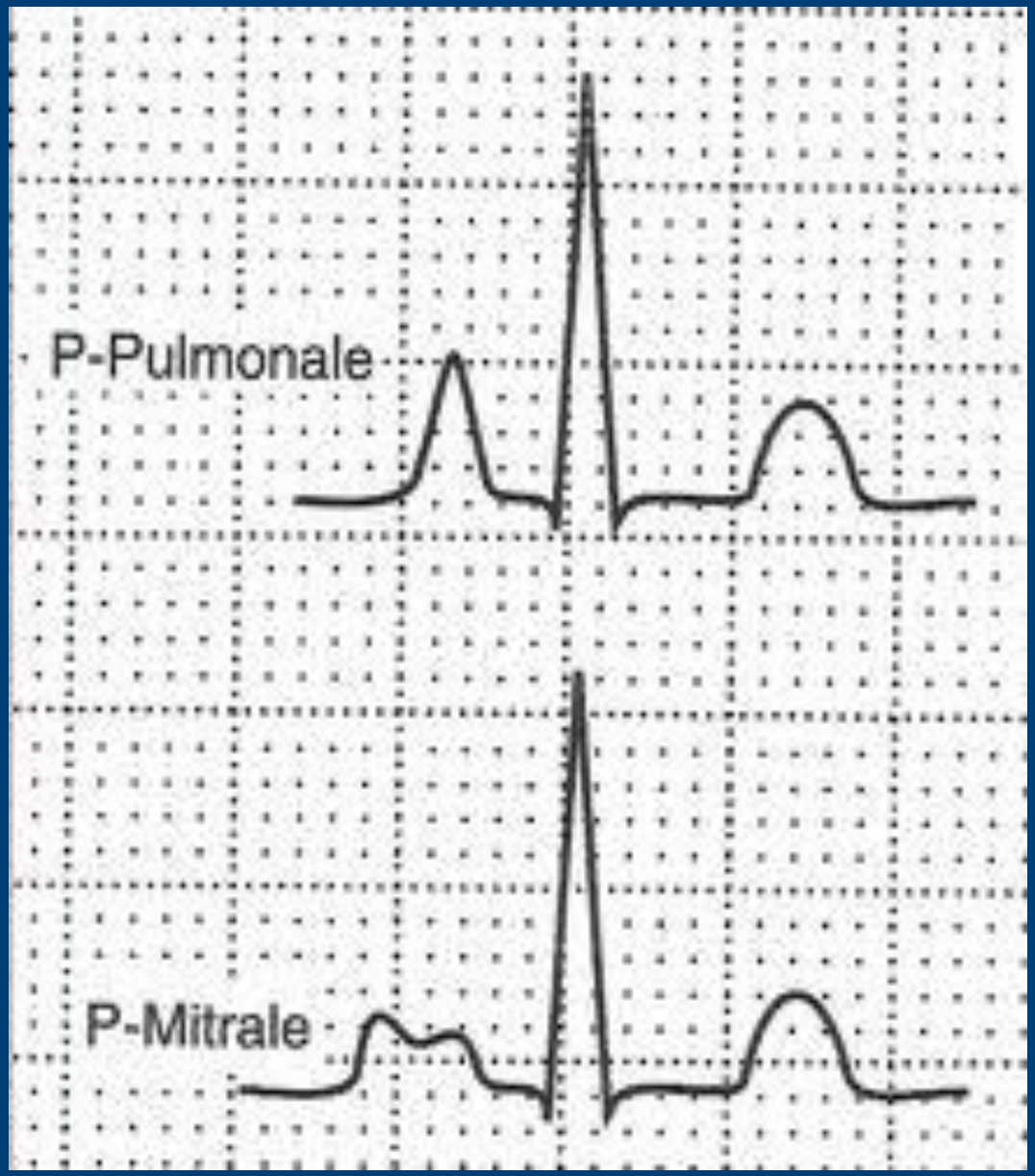
- A tall P wave (over 2.5mm) can be called P pulmonale
- Occurs due to R atrial hypertrophy

- *Causes include:*
  - pulmonary hypertension,
  - pulmonary stenosis
  - tricuspid stenosis

Abnormal P wave

- A P wave with a length >0.08 seconds (2 small squares) and a bifid shape is called P mitrale
- It is caused by left atrial hypertrophy and delayed left atrial depolarisation

- *Causes include:*
  - Mitral valve disease
  - LVH


P-Pulmonale
P-Mitrale

Prolonged QT

- **Drugs**
- ✓ Antiarrhythmic drugs: class Ia (disopyramide, procainamide, quinidine); class III (amiodarone, bretylium, sotalol)
- ✓ Antibacterials: erythromycin, fluoquinolones, trimethoprim
- ✓ Other drugs: terfenadine, cisapride, tricyclic antidepressants, haloperidol, lithium, phenothiazines, chloroquine, thioridazine

- **Electrolyte disturbances**
- ✓ Hypokalaemia
- ✓ Hypomagnesaemia
- **Congenital syndromes**
- ✓ Jervell and Lange-Nielsen syndrome
- ✓ Romano-Ward syndrome
- **Other causes**
- ✓ Ischaemic heart disease
- ✓ Myxoedema
- ✓ Bradycardia due to sick sinus syndrome or complete heart block
- ✓ Subarachnoid haemorrhage

Conditions associated with tall R wave in lead V1

- Right ventricular hypertrophy
- Posterior myocardial infarction
- Type A Wolff-Parkinson-White syndrome
- Right bundle branch block

✓ *A tall R wave in lead V1 is normal in children and young adults*

Suggested criteria for size of T wave

- 1/8 size of the R wave
- < 2/3 size of the R wave
- Height < 10 mm

Rate

- If the heart rate is regular
- Count the number of large squares between R waves
- ie. the RR interval in large squares
- Rate = 300/RR
- e.g. RR = 4 large squares

  300/4 = 75 beats per minute

- If the rhythm is irregular ,it may be better to estimate the rate using the rhythm strip at the bottom of the ECG (usually lead II)
- The rhythm strip is usually 25cm long (250mm
- i.e. 10 seconds) If you count the number of R waves on that strip and multiple by 6 you will get the rate

Cardinal features of sinus rhythm

- The P wave is upright in leads I and II
- Each P wave is usually followed by a QRS complex
- The heart rate is 60-99 beats/min

Left ventricular hypertrophy

Causes of LVH

- Hypertension (most common cause)
- Aortic stenosis
- Aortic regurgitation
- Mitral regurgitation
- Coarctation of the aorta
- Hypertrophic cardiomyopathy

Left ventricular hypertrophy

- **Criteria for Diagnosing LVH**

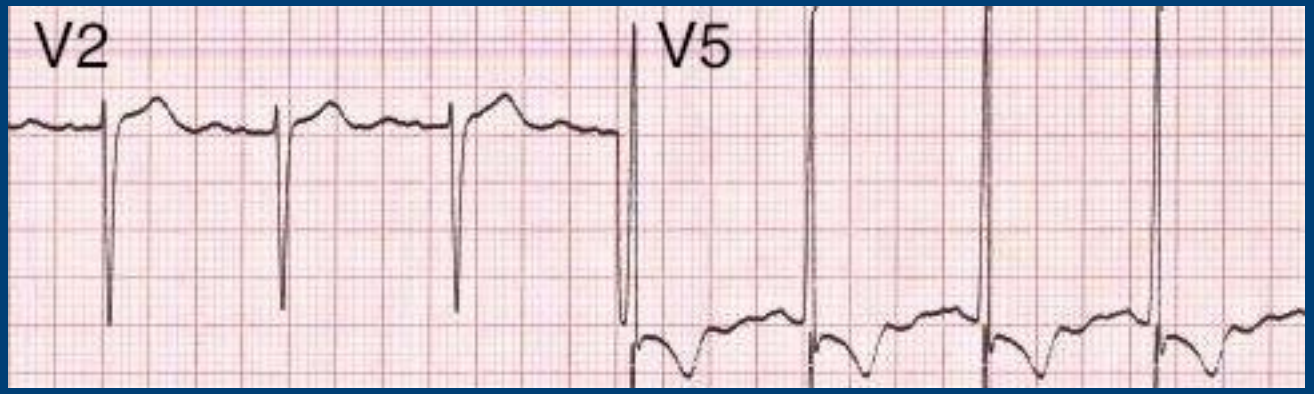
Voltage Criteria

- *Limb Leads*
- 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
- *Precordial Leads*
- R wave in V_4, V_5 or V_6 > 26 mm
- R wave in V_5 or V_6 plus S wave in V_1 > 35 mm
- Largest R wave plus largest S wave in precordial leads > 45 mm

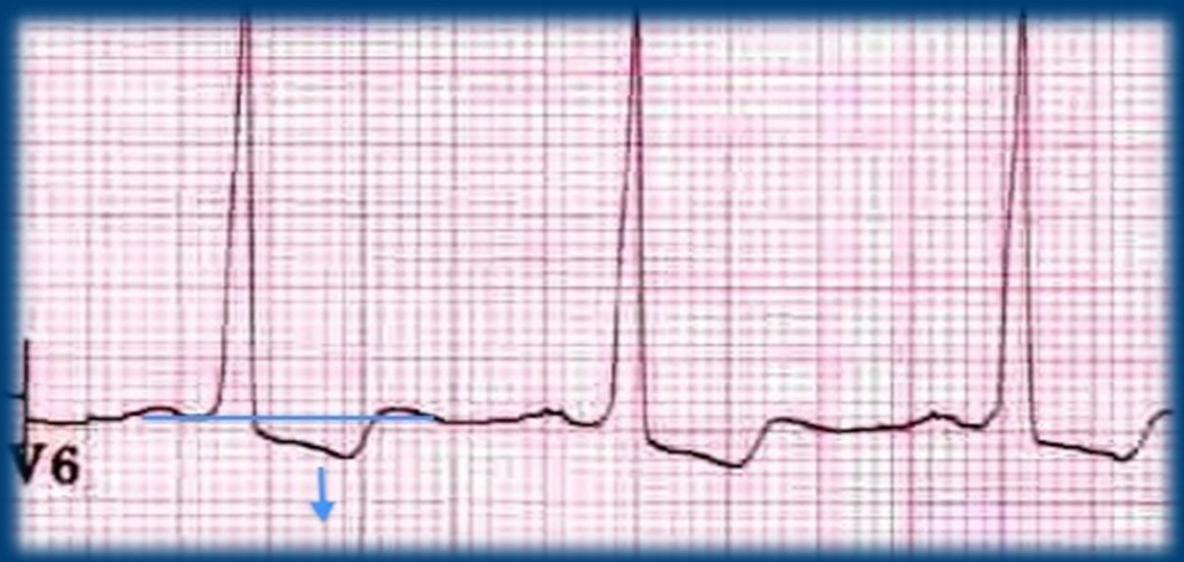
Non Voltage Criteria

- Increased R wave peak time > 50 ms in leads V_5 or V_6
- ST segment depression and T wave inversion in the left-sided leads: *AKA the left ventricular 'strain' pattern*

ECG for LVH 1

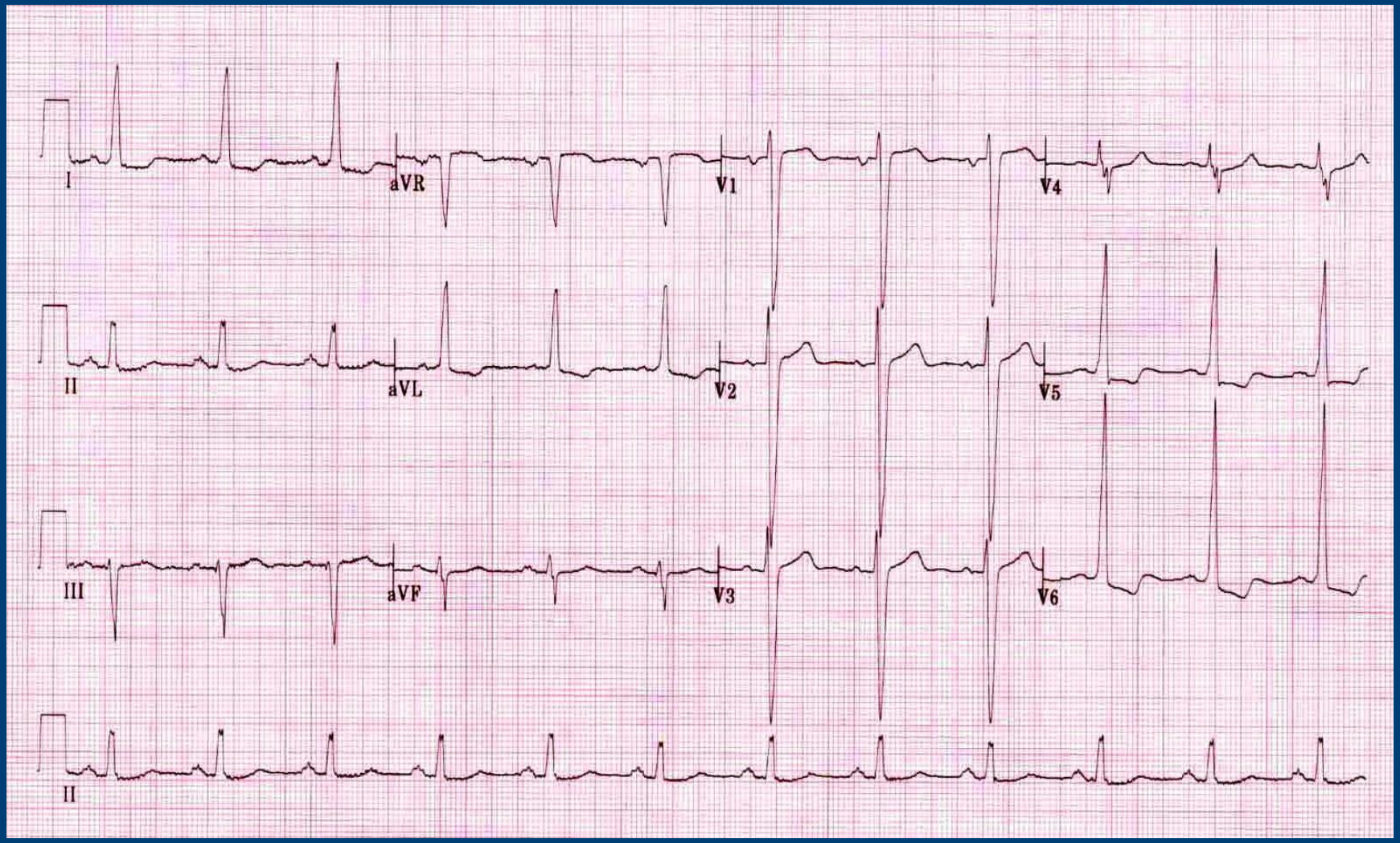


LVH by voltage criteria: S wave in V2 + R wave in V5 > 35 mm

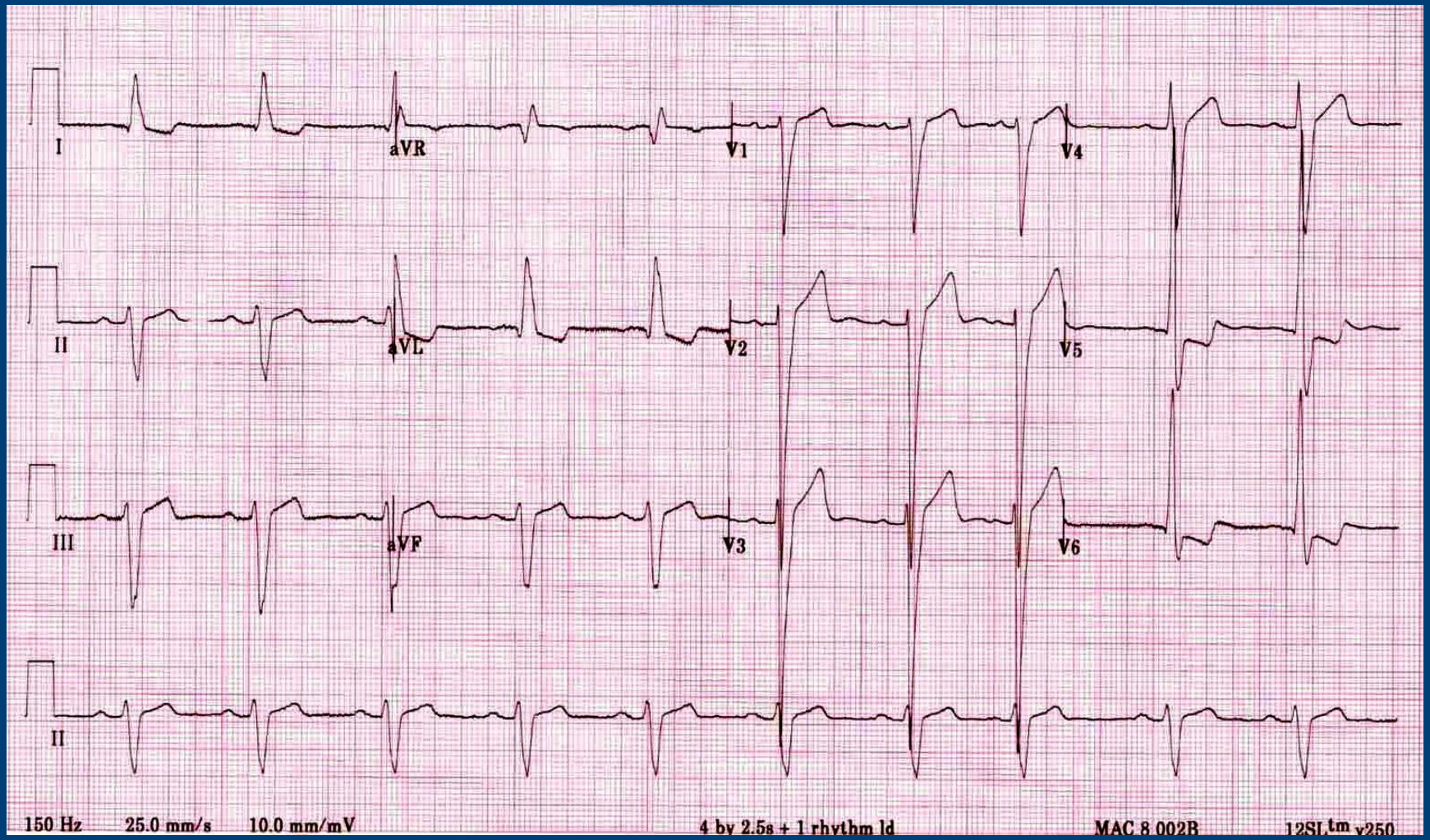


LV strain pattern: ST depression and T wave inversion in the lateral leads

ECG for LVH 2



ECG for LVH 3


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
MAC 8 002B
12SL tm v250

Right ventricular hypertrophy

Causes

- Pulmonary hypertension
- Mitral stenosis
- Pulmonary embolism
- Chronic lung disease (cor pulmonale)
- Congenital heart disease (e.g. Tetralogy of Fallot, pulmonary stenosis)
- Arrhythmogenic right ventricular cardiomyopathy

RVH

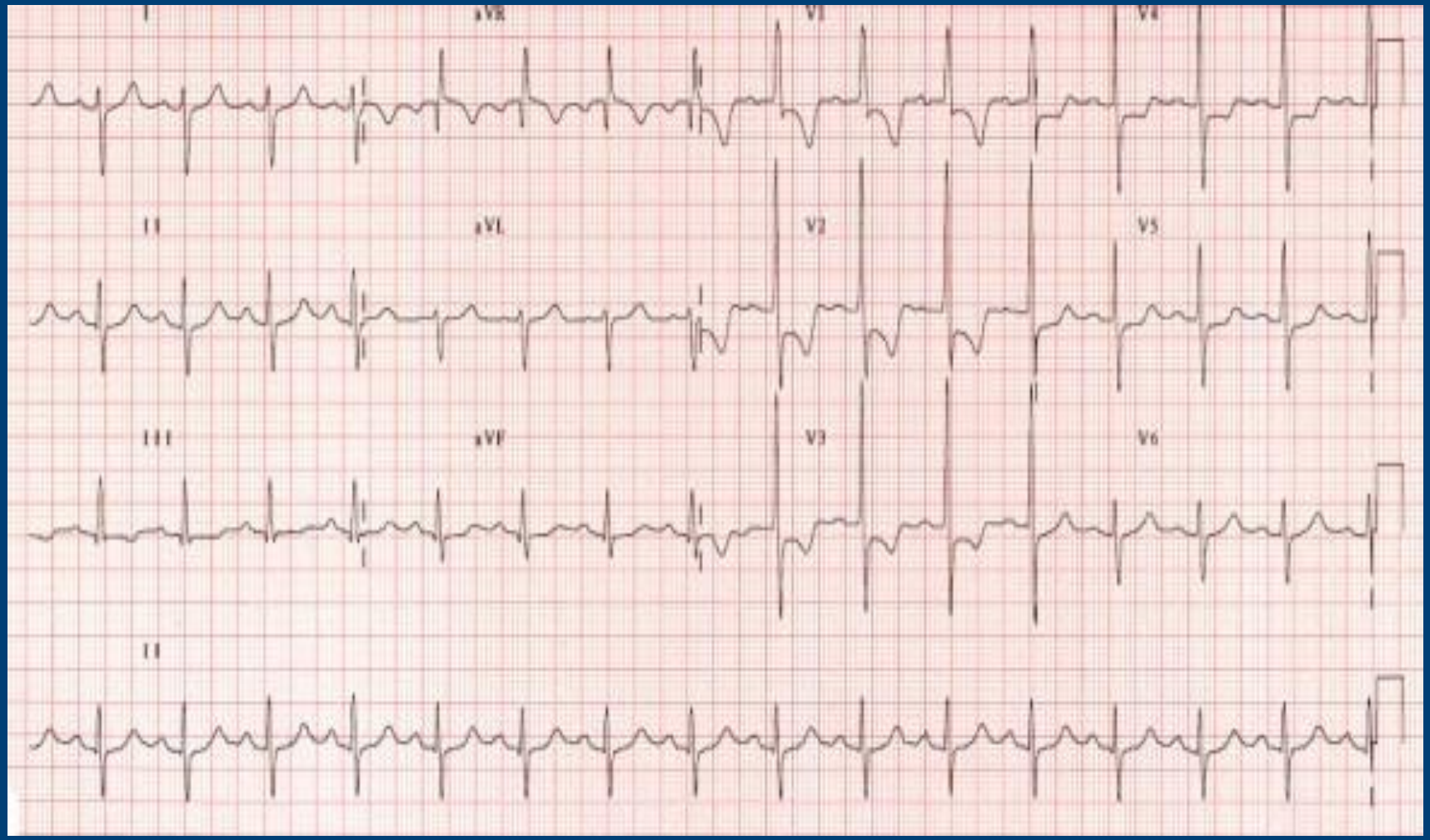
Diagnostic criteria

- Right axis deviation of +110° or more.
- Dominant R wave in V1 (> 7mm tall or R/S ratio > 1).
- Dominant S wave in V5 or V6 (> 7mm deep or R/S ratio < 1).
- *QRS duration < 120ms (i.e. changes not due to RBBB).*

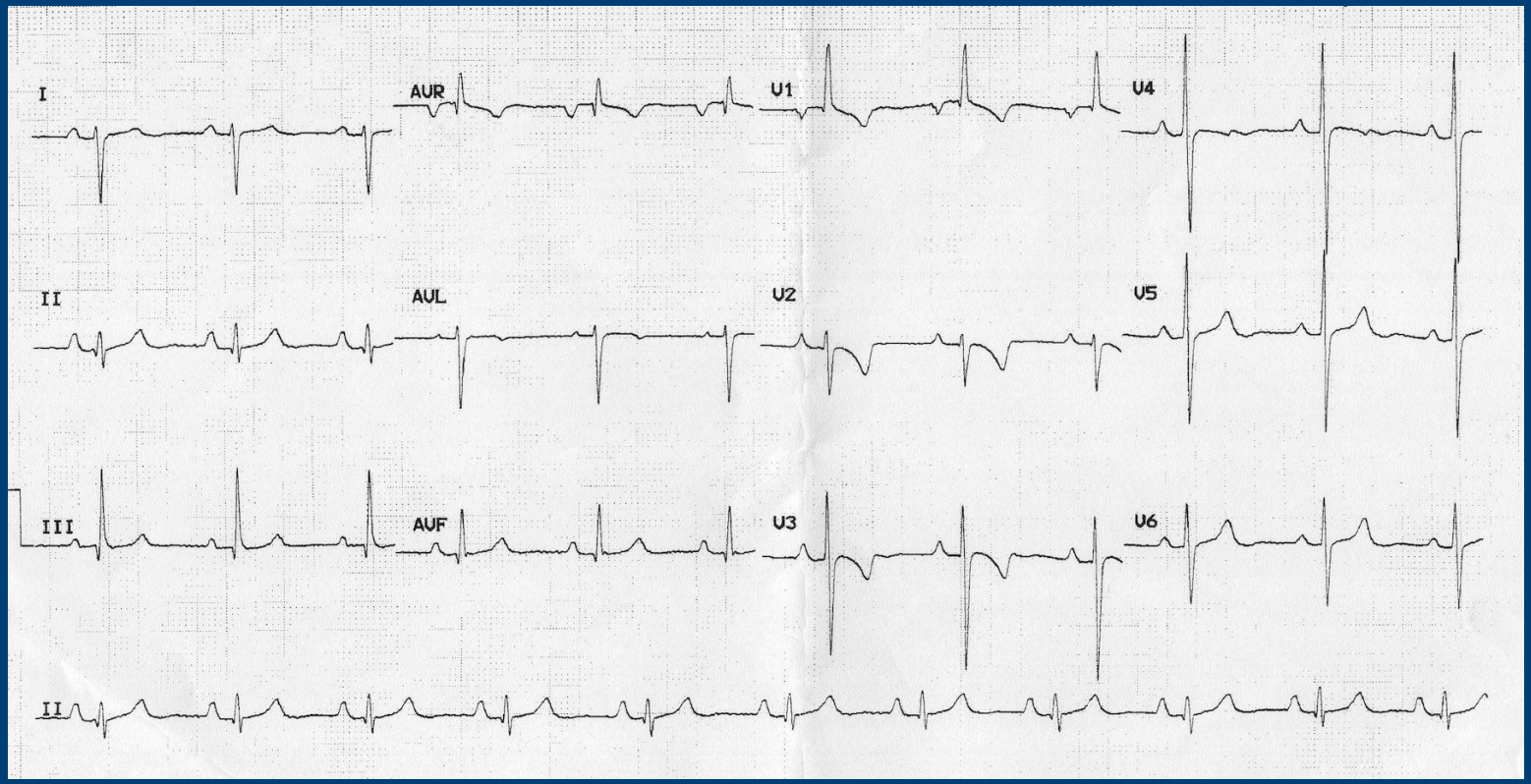
Supporting criteria

- Right atrial enlargement (P pulmonale).
- Right ventricular strain pattern = ST depression / T wave inversion in the right precordial (V1-4) and inferior (II, III, aVF) leads.
- S1 S2 S3 pattern = far right axis deviation with dominant S waves in leads I, II and III.
- Deep S waves in the lateral leads (I, aVL, V5-V6).

ECG for RVH



ECG for RVH



RVH in an adult with uncorrected Tetralogy of Fallot

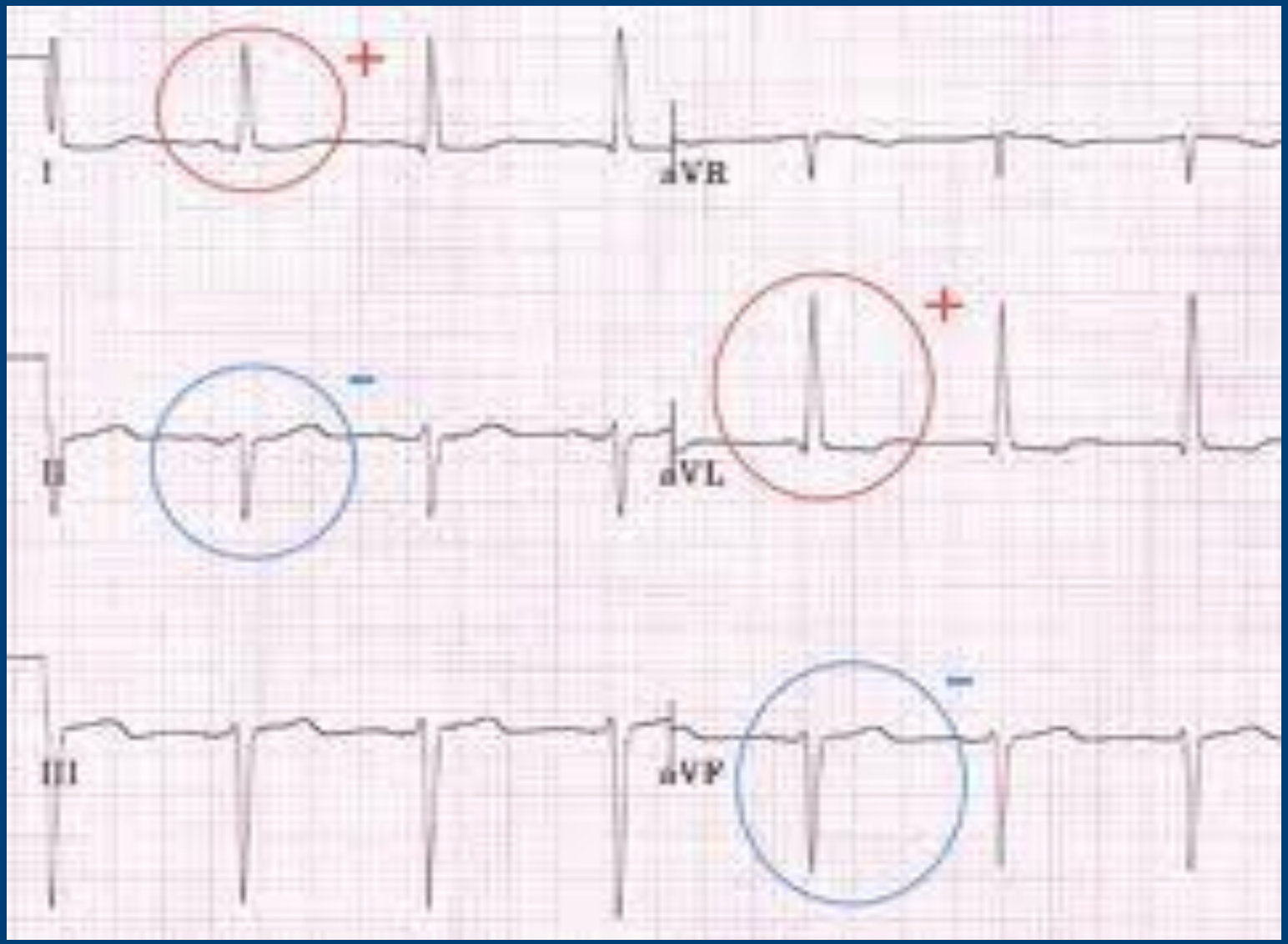
Anatomical relations of leads in a standard 12 lead ECG

- II, III, and aVF: inferior surface of the heart
- V1 to V4: anterior surface
- I, aVL, V5, and V6: lateral surface
- V1 and aVR: right atrium and cavity of left ventricle

Calculating the cardiac axis

	Normal axis	Right axis deviation	Left axis deviation
Lead I	Positive	Negative	Positive
Lead II	Positive	Positive OR negative	Negative
Lead III	Positive OR negative	Positive	Negative

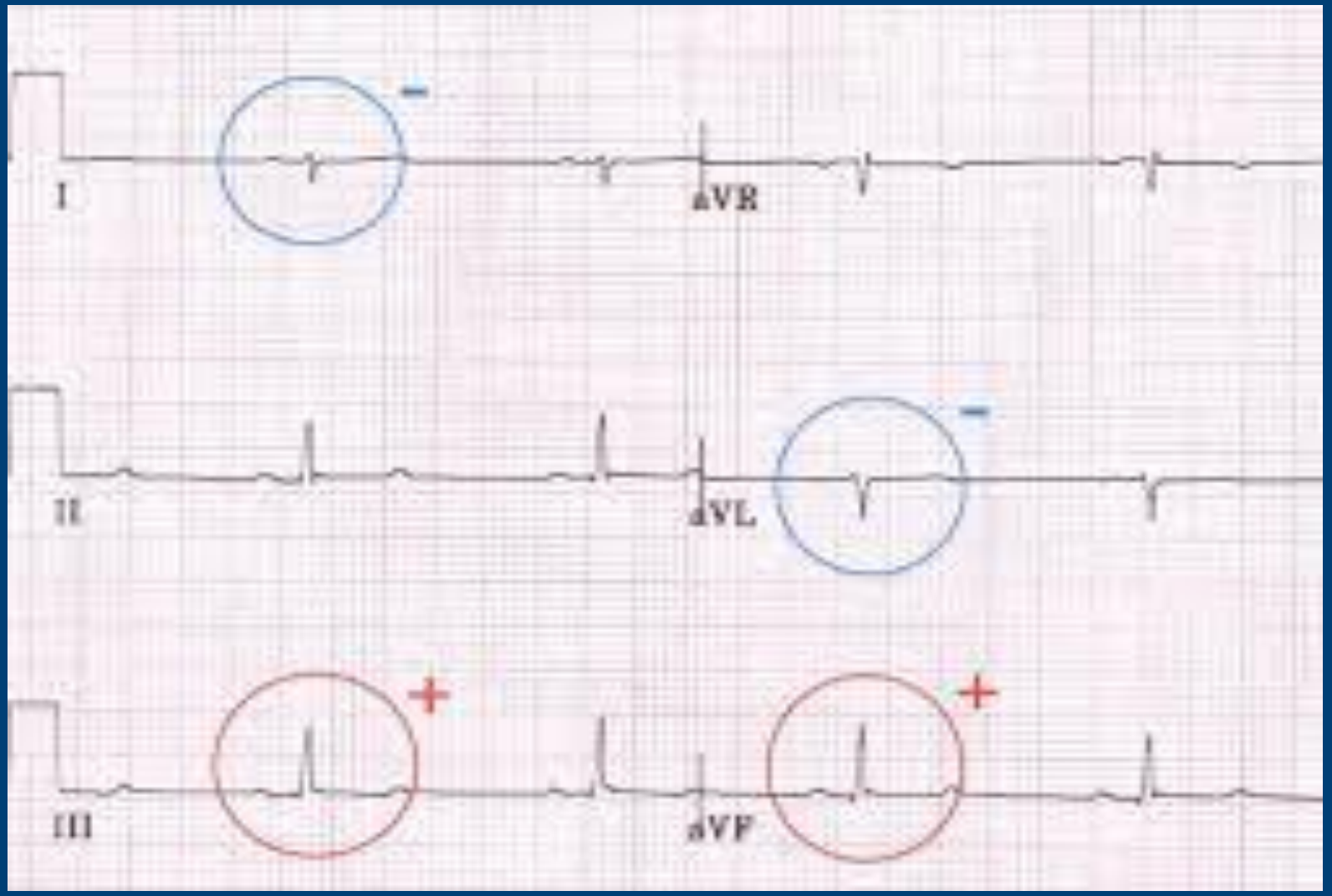
Left axis deviation ECG



Causes of left axis deviation

- Left bundle branch block(LBBB).
- Left anterior hemi-block(LAHB)
- Left ventricular hypertrophy(LVH)
- Primum atrial septal defect(ASD)
- Cardiomyopathies
- Tricusped atresia
- emphysema
- hyperkalaemia
- Wolff-Parkinson-White syndrome - right sided accessory pathway(type B)

Right axis deviation ECG



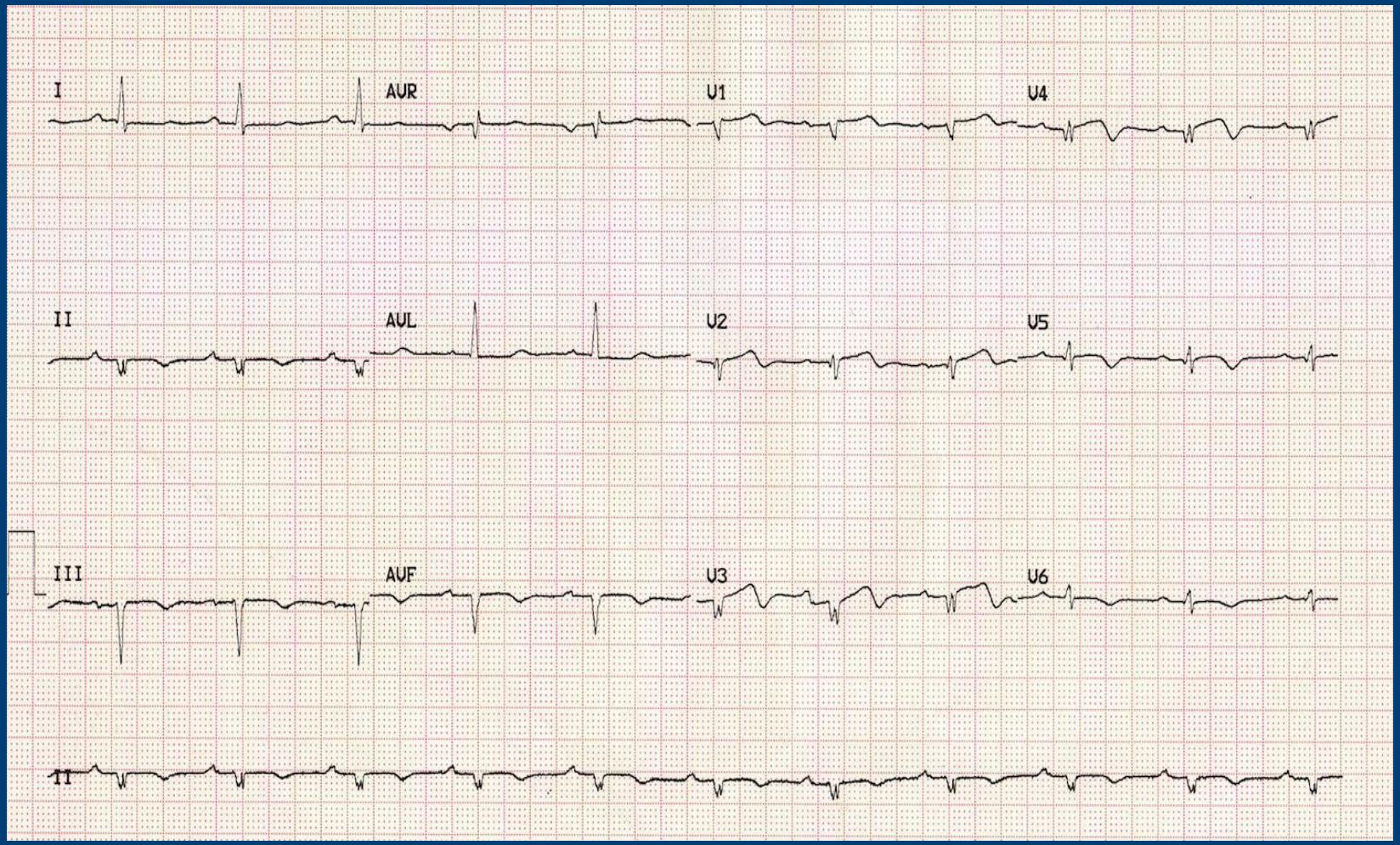
Causes of right axis deviation

- normal finding in children and tall thin adults
- Right bundle branch block(RBBB)
- Left posterior hemi-block
- Right ventricular hypertrophy(RVH), e.g. lung disease, pulmonary embolism, severe pulmonary stenosis, fallots.
- Large secondum ASD.
- Wolff-Parkinson-White syndrome - left sided accessory pathway (type A)
- ventricular septal defect(VSD)
- anterolateral myocardial infarction

Low voltage ECG

- Obesity
- COPD
- Pericardial effusion
- Severe hypothyroidism
- Subcutaneous emphysema
- Massive myocardial damage/infarction
- Infiltrative/restrictive diseases such as amyloid cardiomyopathy.

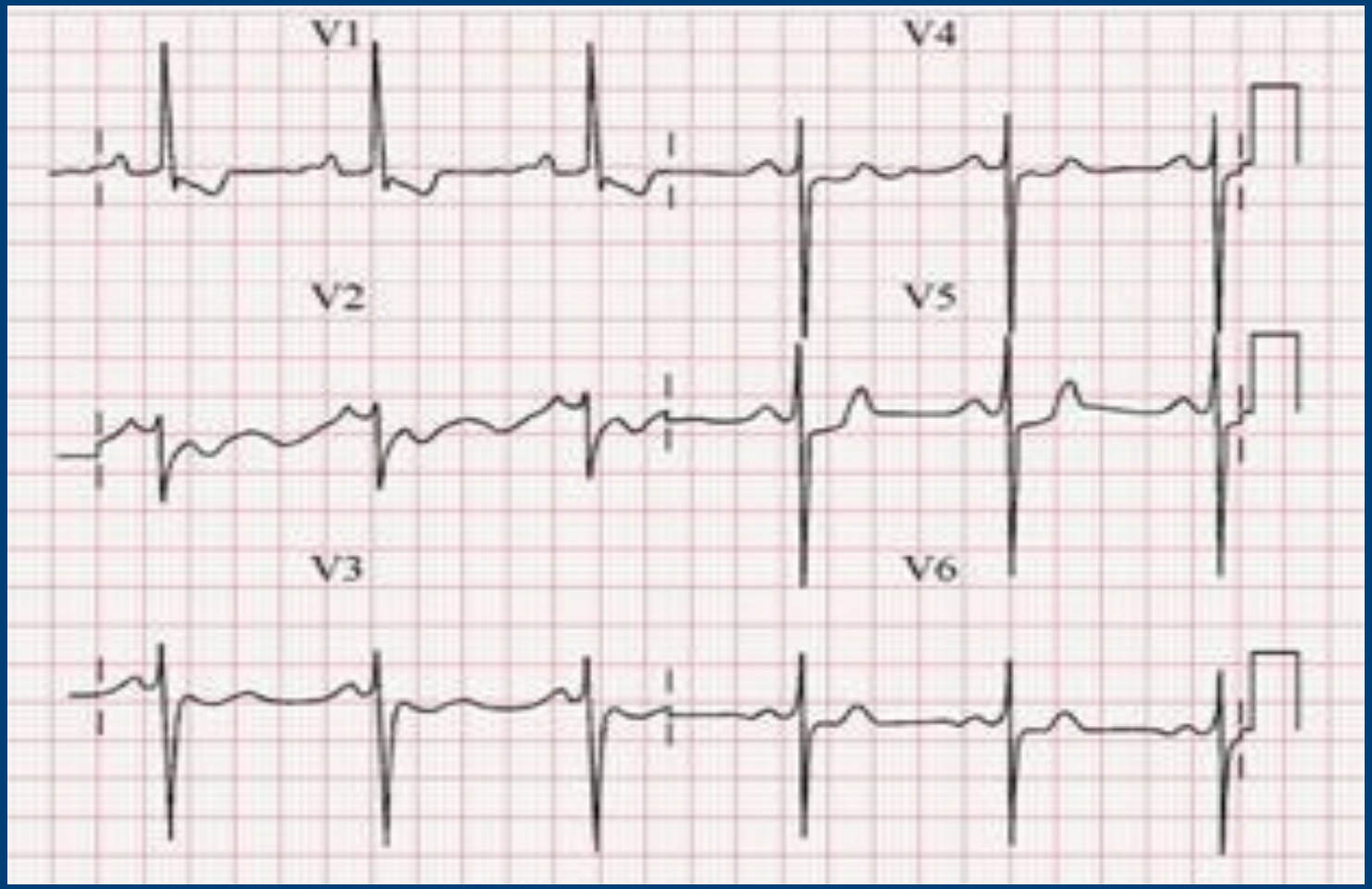
Low voltage ECG



Causes of tall R in V1

- True posterior infarct
- Right ventricular hypertrophy
- Ventricular septal hypertrophy (HOCM)
- Right bundle branch block
- Wolff-Parkinson-White syndrome (type A)
- Dextrocardia
- Pulmonary embolism

tall R in V1 ECG



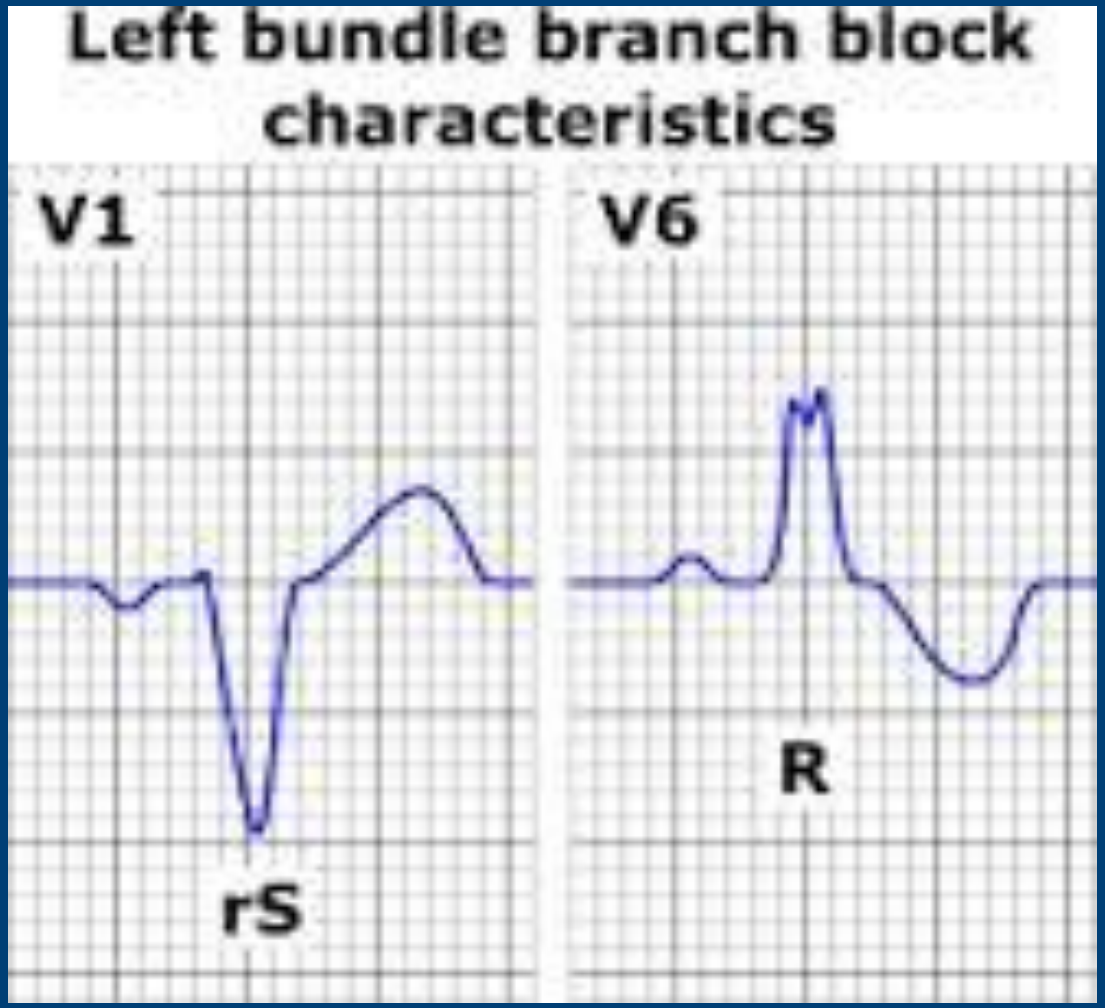
left bundle branch block (LBBB)

I. If left bundle branch block is present, the QRS complex may look like a "W" in V1 and/or an "M"shape in V6.

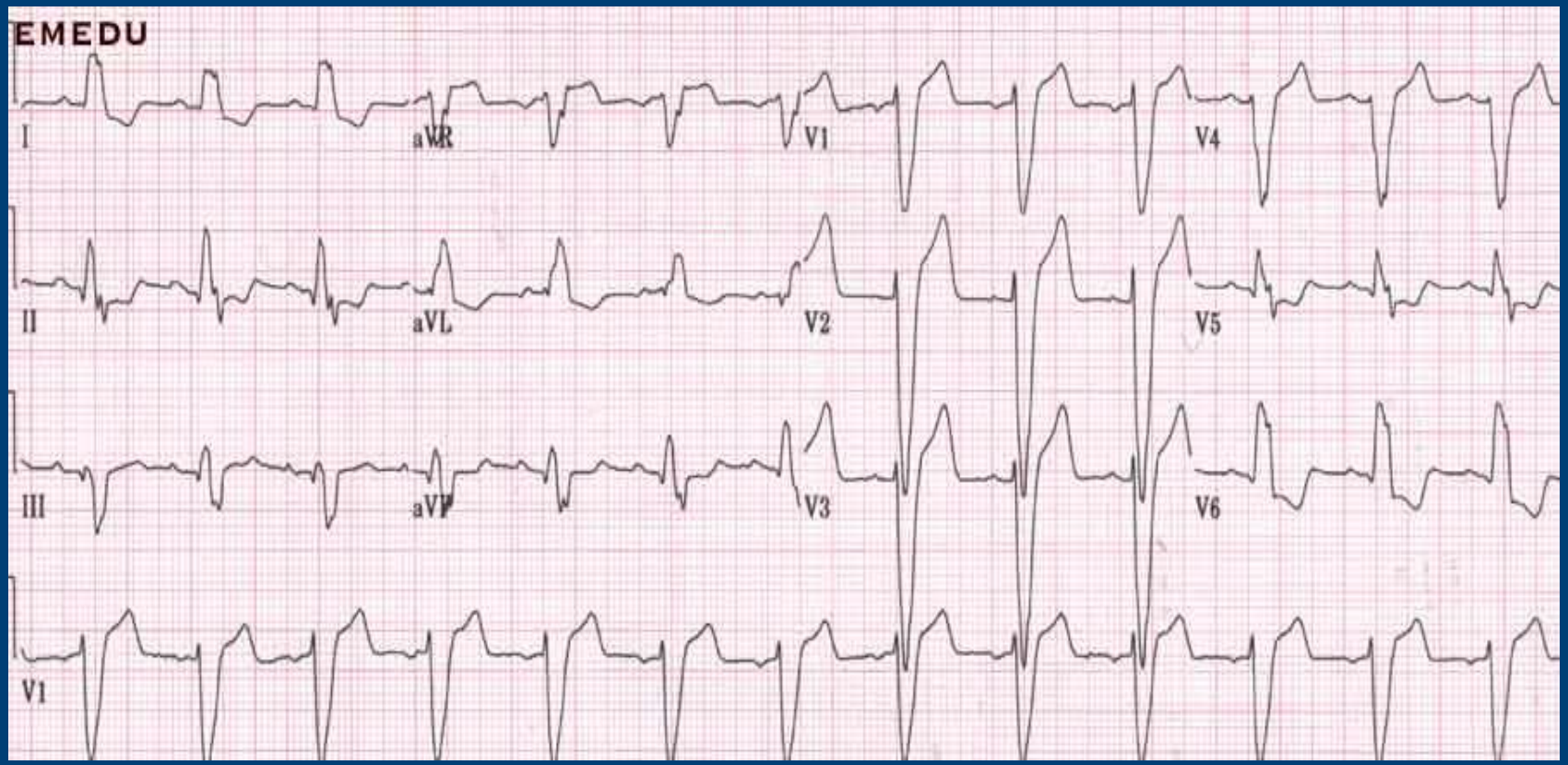
II. Wide QRS complex + ST depression in lateral leads (I, aVL, V5, V6)

- New onset LBBB with chest pain consider Myocardial infarction

left bundle branch block (LBBB) ECG



left bundle branch block (LBBB) ECG



Causes of left bundle branch block (LBBB)

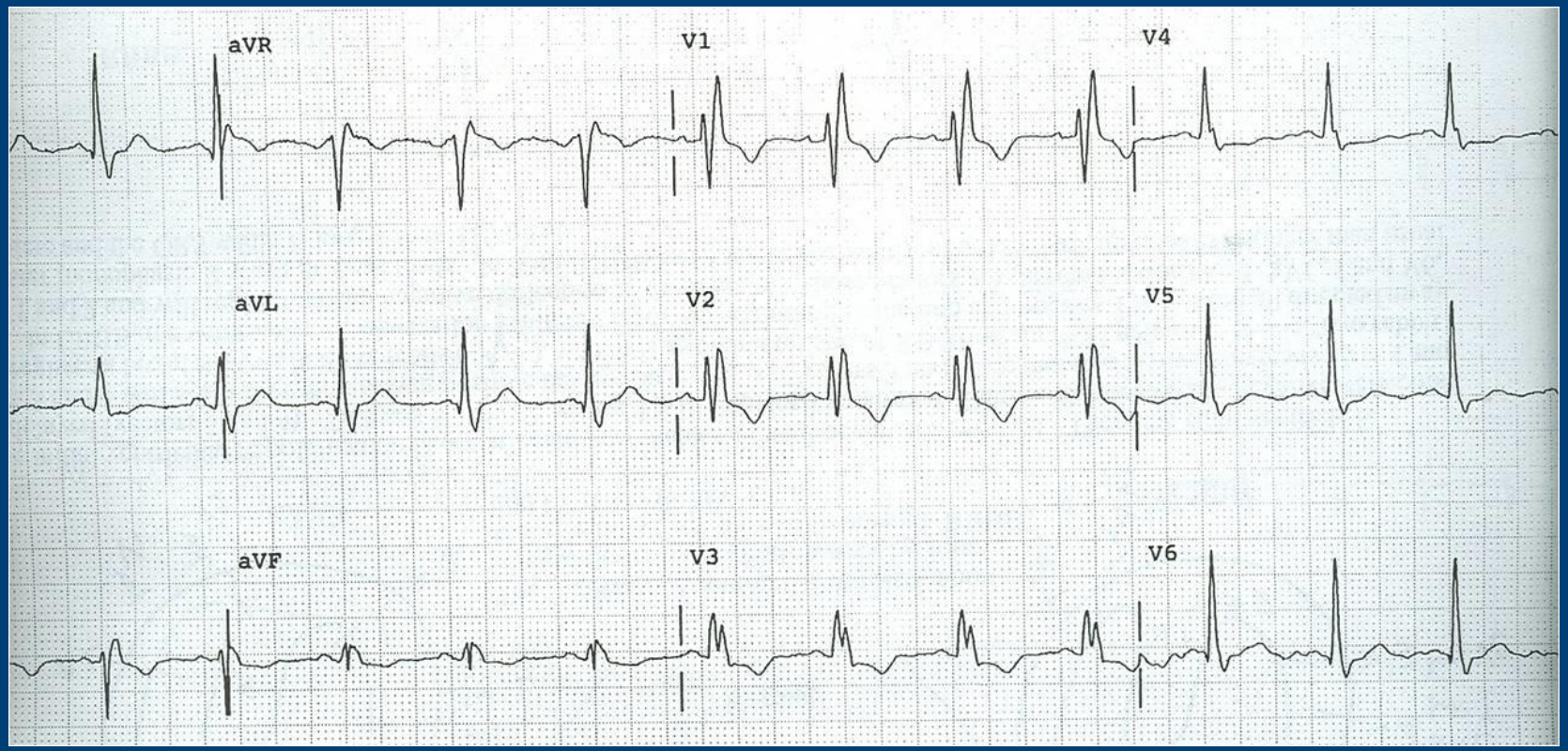
- Ischemic heart disease (recent or old MI)
- Left ventricular hypertrophy (LVH)
- Aortic valve disease
- Cardiomyopathy
- Myocarditis
- Post-valve replacment
- Bight ventricular pacemaker

Right bundle branch block (RBBB)

- It is also called RSR pattern
- If right bundle branch block is present, there may be an "M" in V1 and/or a "W" in V6.
- Can occur in healthy people with normal QRS width --partial RBBB

Right bundle branch block (RBBB)ECG

Right bundle branch block (RBBB)ECG

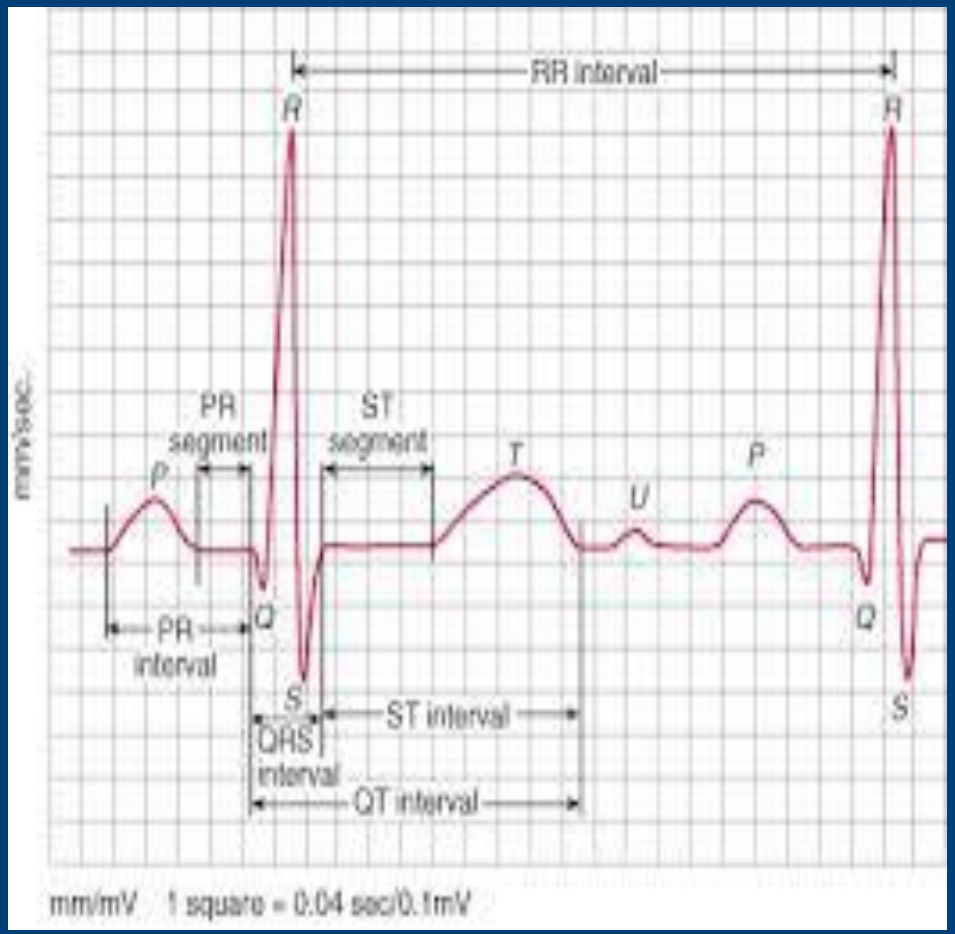


Causes of right bundle branch block (RBBB)

- Normal in young
- Right ventricular strain(e.g.plmonary embolus)
- Atrial septal defect (ASD)
- Ischemic heart disease
- Myocarditis
- Idiopathic

U wave

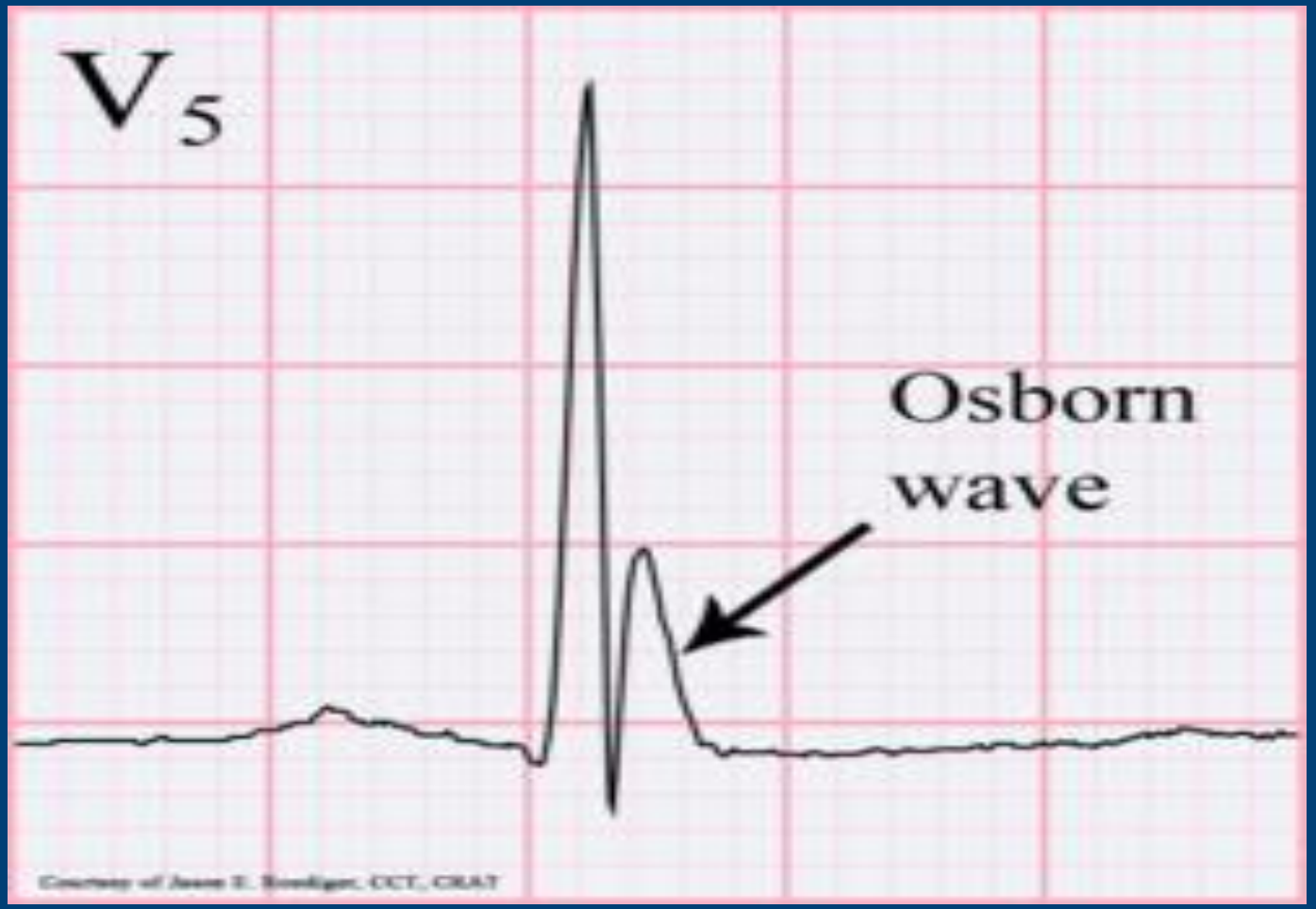
- The U wave is a small deflection that follows the T wave.
- Prominent U waves may be found in athletes and are associated with hypokalaemia and hypercalcaemia.



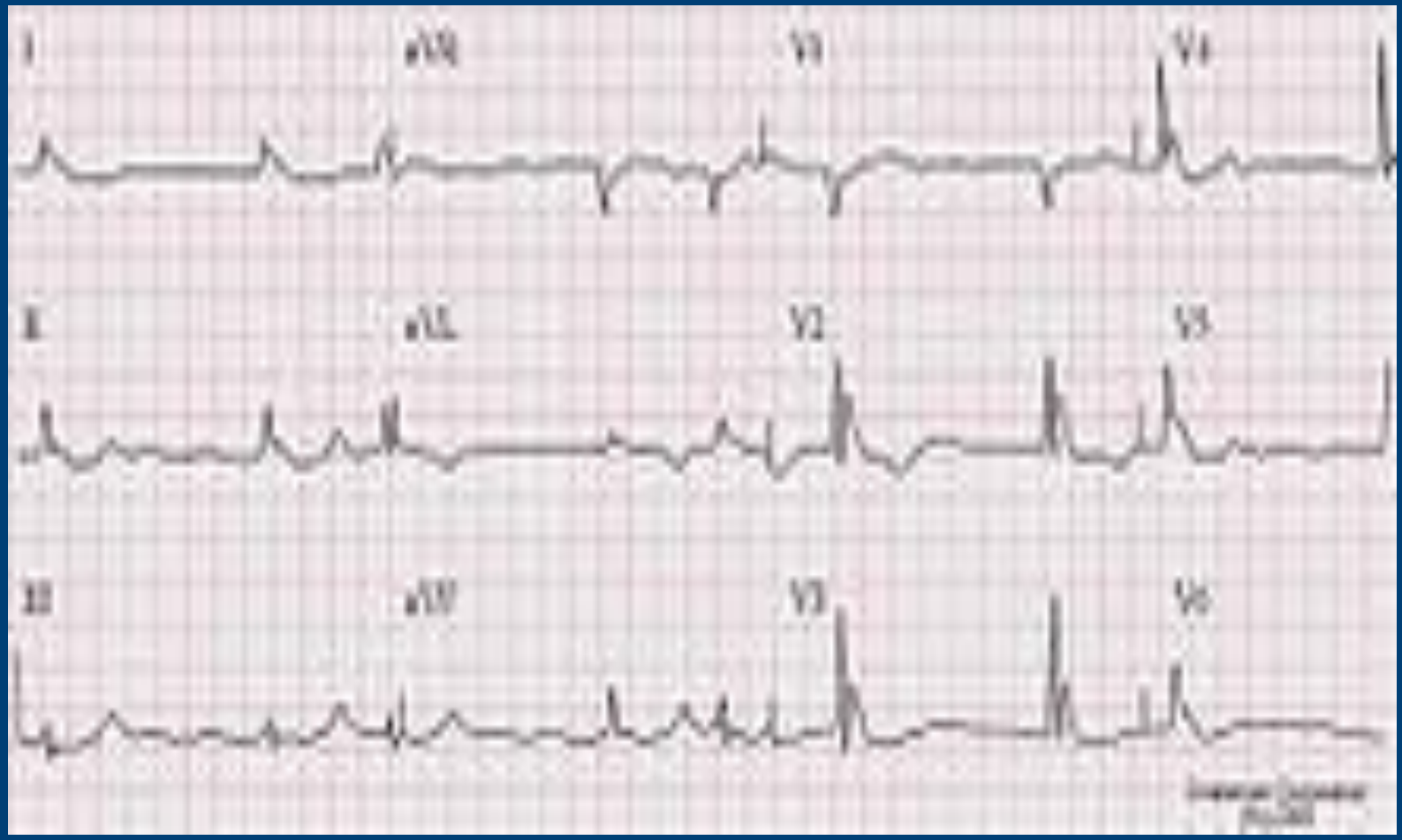
J WAVE

- positive deflections occurring at the junction between the QRS complex and the ST segment.
- observed in people suffering from hypothermia with a temperature of less than 32 C (90 F)
- though they may also occur in people with high blood levels of calcium (hypercalcemia), brain injury.

J wave ECG



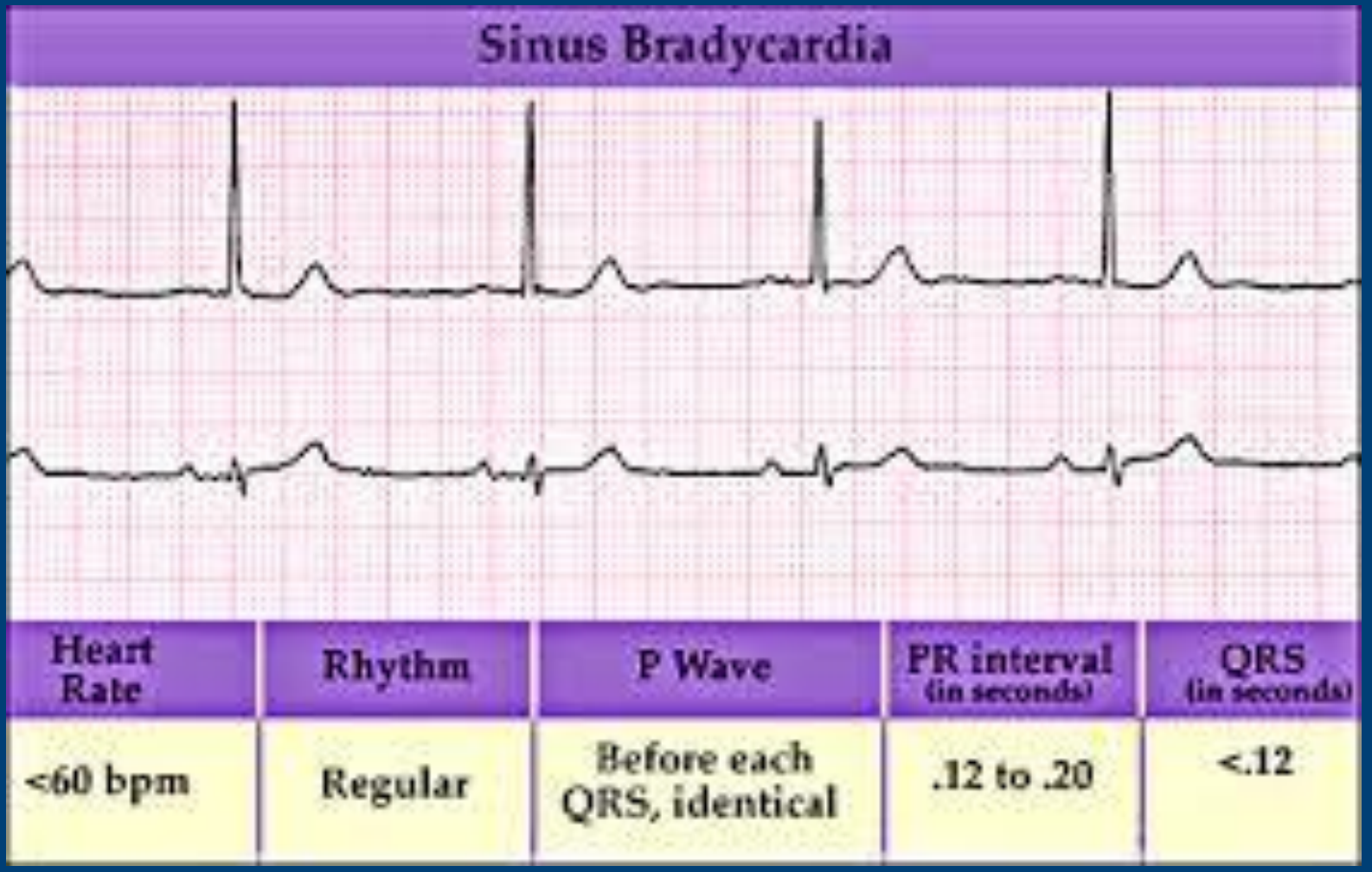
J wave ECG



Pathological causes of sinus bradycardia

- Acute myocardial infarction
- Drugs—for example, blockers, digoxin, amiodarone
- Obstructive jaundice
- Raised intracranial pressure
- Sick sinus syndrome
- Hypothermia
- Hypothyroidism

Sinus Bradycardia ECG



Atrioventricular conduction block

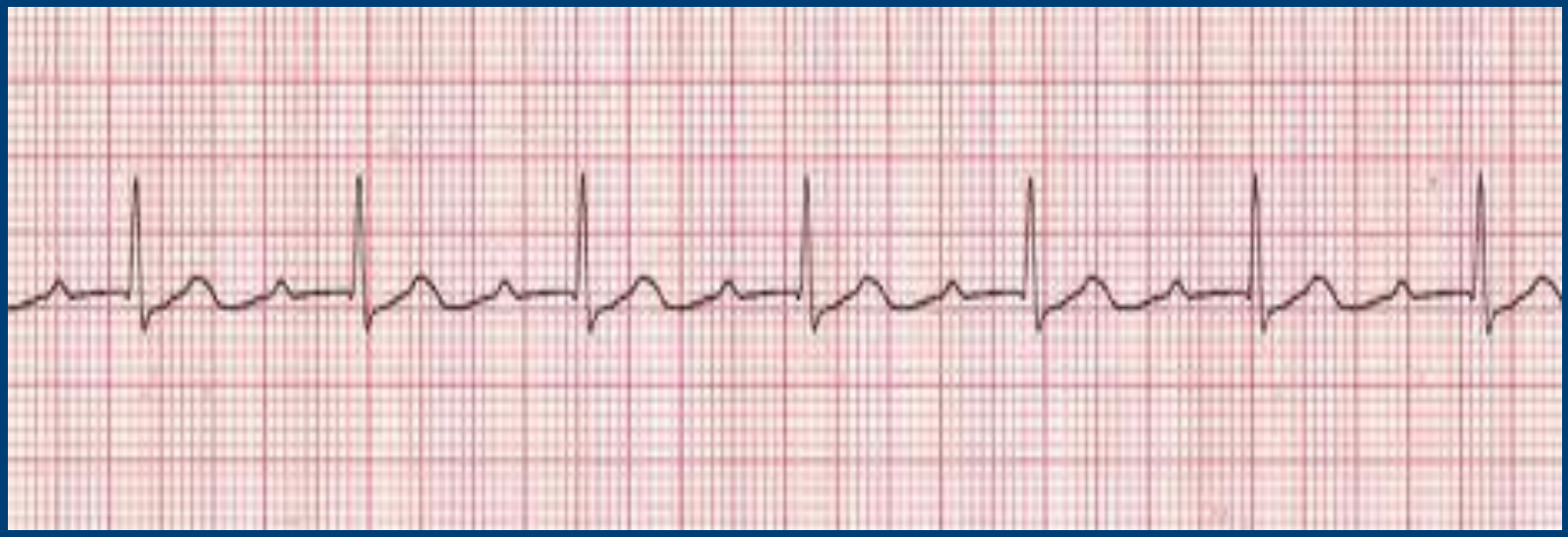
- ***Causes of atrioventricular conduction block***

- ✓ Myocardial ischaemia or infarction
- ✓ Degeneration of the His-Purkinje system
- ✓ Infection—for example, Lyme disease, diphtheria
- ✓ Immunological disorders—for example, systemic lupus erythematosus
- ✓ Surgery
- ✓ Congenital disorders

First degree block

- In first degree block there is a delay in conduction of the atrial impulse to the ventricles, usually at the level of the atrioventricular node.
- This results in prolongation of the PR interval to > 0.2 s.
- A QRS complex follows each P wave.
- the PR interval remains constant.

First degree block ECG



Second degree block

- In second degree block there is intermittent failure of conduction between the atria and ventricles.
- Some P waves are not followed by a QRS complex.
- There are two types of second degree block:

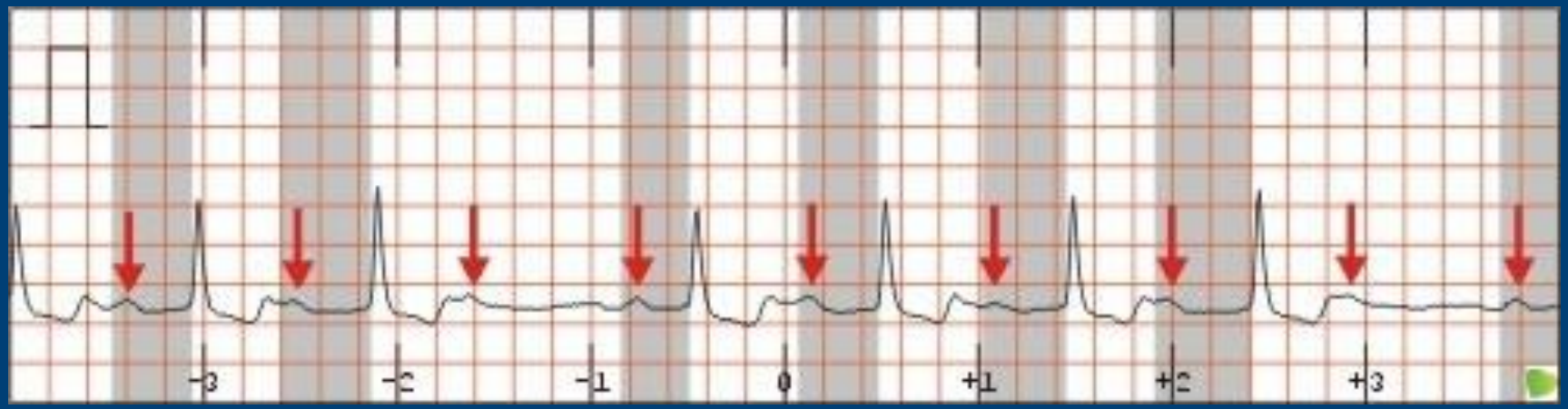
✓ Mobitz type I block(Wenckebach phenomenon)

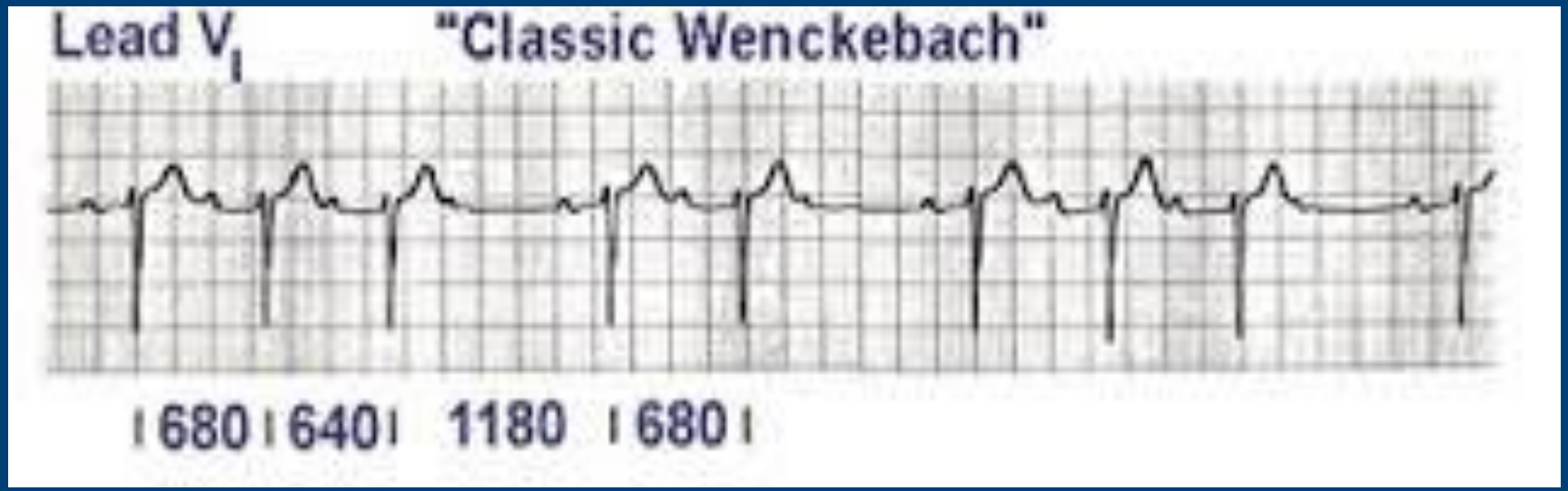
✓ Mobitz type II block

Mobitz type I block(Wenckebach phenomenon)

- The initial PR interval is normal but progressively lengthens with each successive beat until eventually atrioventricular transmission is blocked completely and the P wave is not followed by a QRS complex.
- The PR interval then returns to normal, and the cycle repeats.

Mobitz type I block ECG

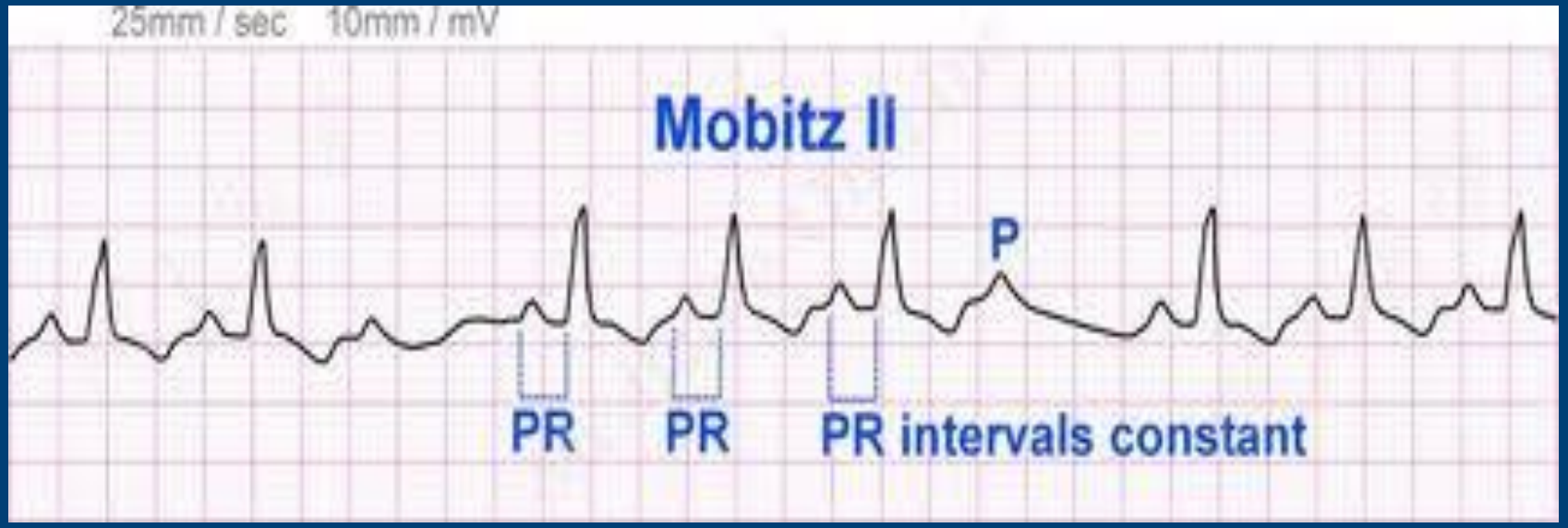




Mobitz type II block

- is less common but is more likely to produce symptoms.
- There is intermittent failure of conduction of P waves.
- The PR interval is constant, though it may be normal or prolonged.
- High degree atrioventricular block, which occurs when a QRS complex is seen only after every three, four, or more P waves,
- may progress to complete third degree atrioventricular block.

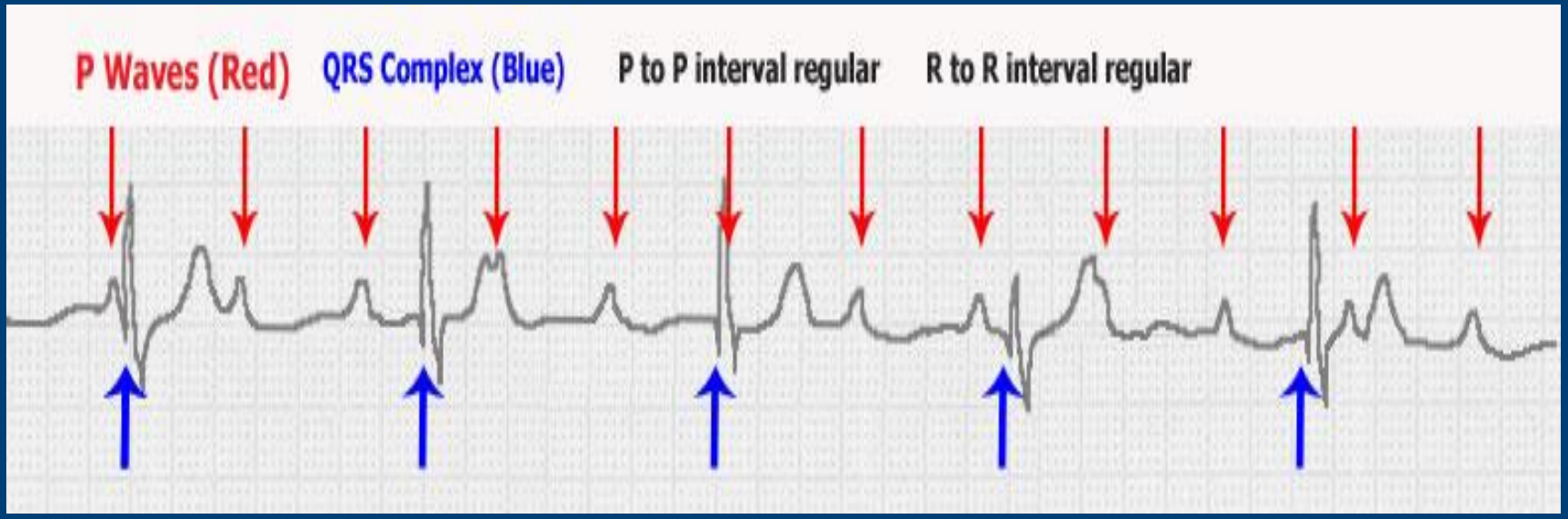
Mobitz type II ECG



Third degree block

- In third degree block there is complete failure of conduction between the atria and ventricles, with complete independence of atrial and ventricular contractions.
- The P waves bear no relation to the QRS complexes and usually proceed at a faster rate.

Third degree block ECG



Causes of sinus tachycardia

- *Physiological—Exertion, anxiety, pain*
- *Pathological—Fever, anaemia, hypovolaemia, hypoxia*
- *Endocrine—Thyrotoxicosis*
- *Pharmacological—Adrenaline as a result of phaeochromocytoma;* salbutamol; alcohol, caffeine

Differential Diagnosis of Tachycardia

Tachycardia	Wide Complex	Narrow Complex
Regular	ST w/ aberrancy SVT w/ aberrancy VT	ST SVT Atrial flutter
Irregular	A-fib w/ aberrancy A-fib w/ WPW VT	A-fib A-flutter w/ variable conduction MAT

Electrocardiographic characteristics of atrial arrhythmias

- **Sinus tachycardia**

✓ P waves have normal morphology
✓ Atrial rate 100-200 beats/min
✓ Regular ventricular rhythm
✓ Ventricular rate 100-200 beats/min
✓ One P wave precedes every QRS complex

- **Atrial tachycardia**

✓ Abnormal P wave morphology
✓ Atrial rate 100-250 beats/min
✓ Ventricular rhythm usually regular
✓ Variable ventricular rate

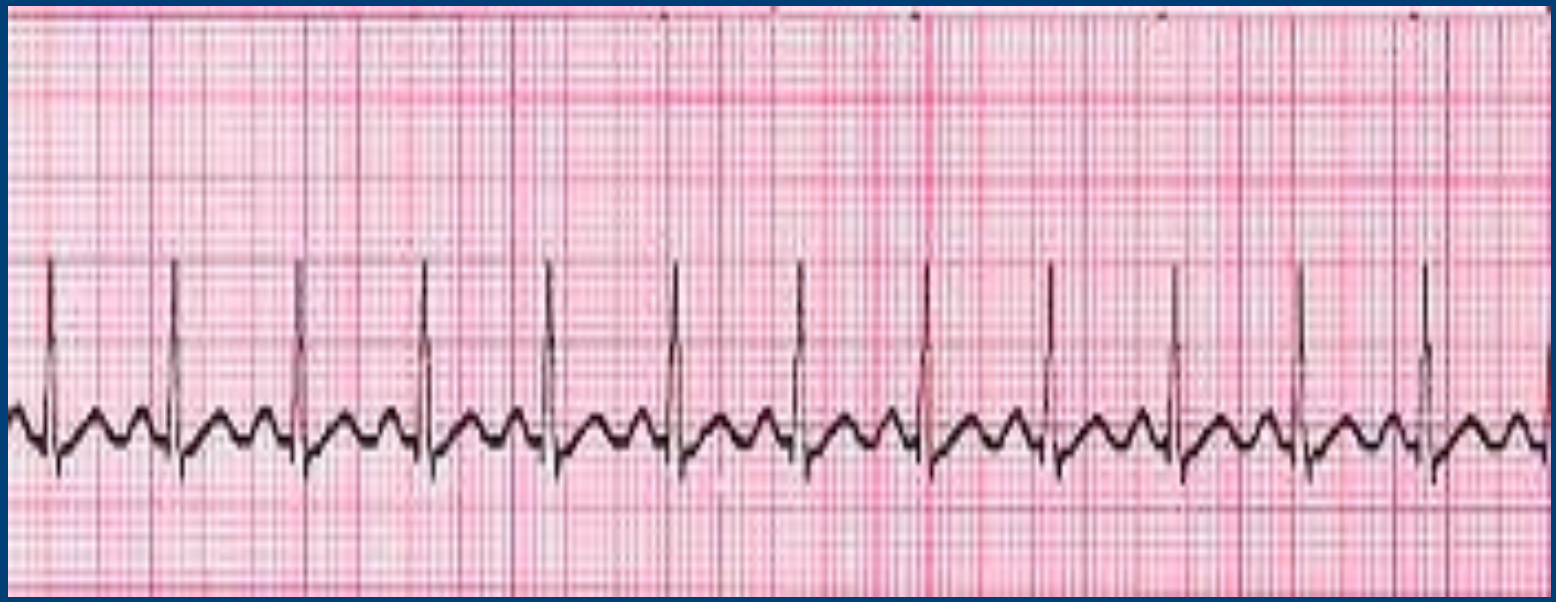
- **Atrial flutter**

✓ Undulating saw-toothed baseline F (flutter) waves
✓ Atrial rate 250-350 beats/min
✓ Regular ventricular rhythm
✓ Ventricular rate typically 150 beats/min (with 2:1 atrioventricular block)

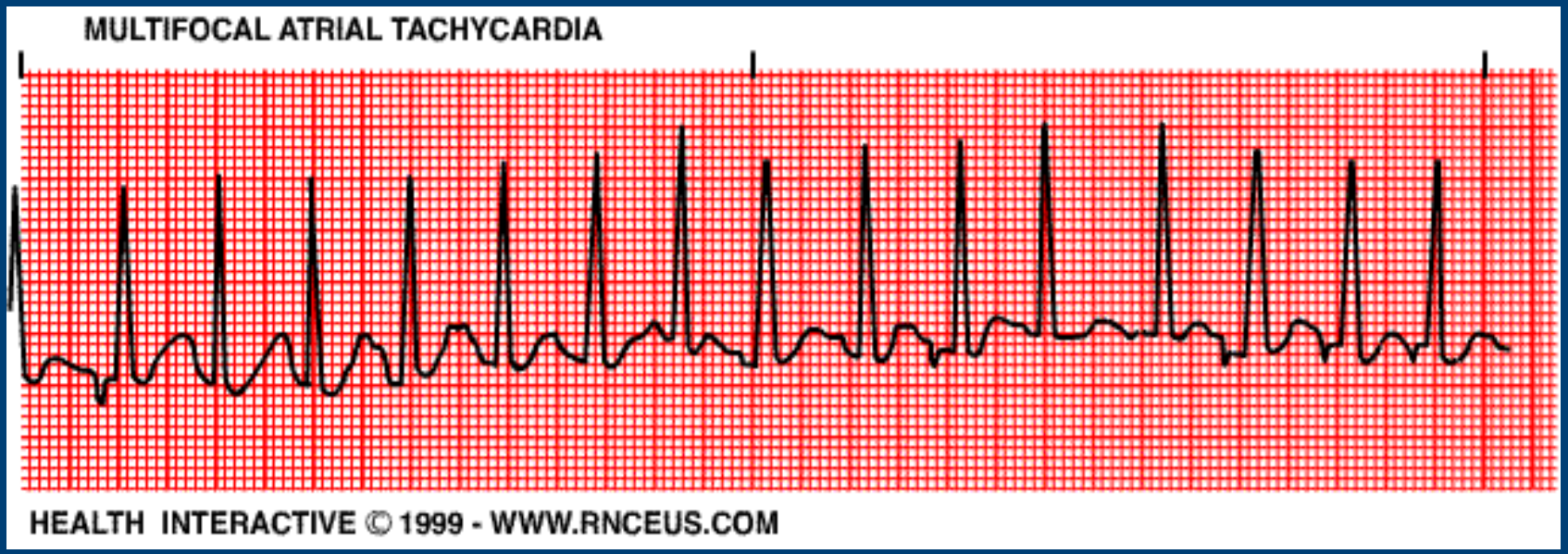
- **Atrial fibrillation**

✓ P waves absent; oscillating baseline f (fibrillation) waves
✓ Atrial rate 350-600 beats/min
✓ Irregular ventricular rhythm
✓ Ventricular rate 100-180 beats/min

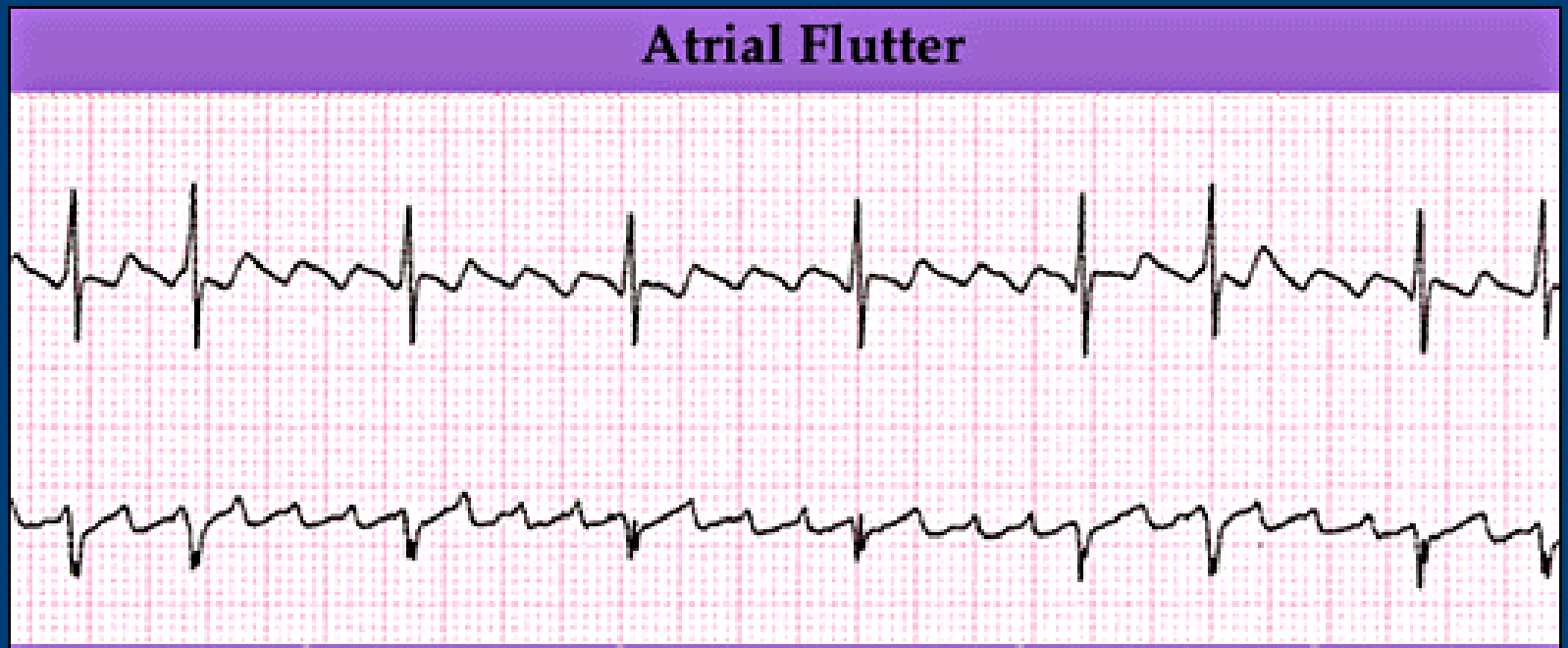
Sinus tachycardia ECG



Atrial tachycardia ECG

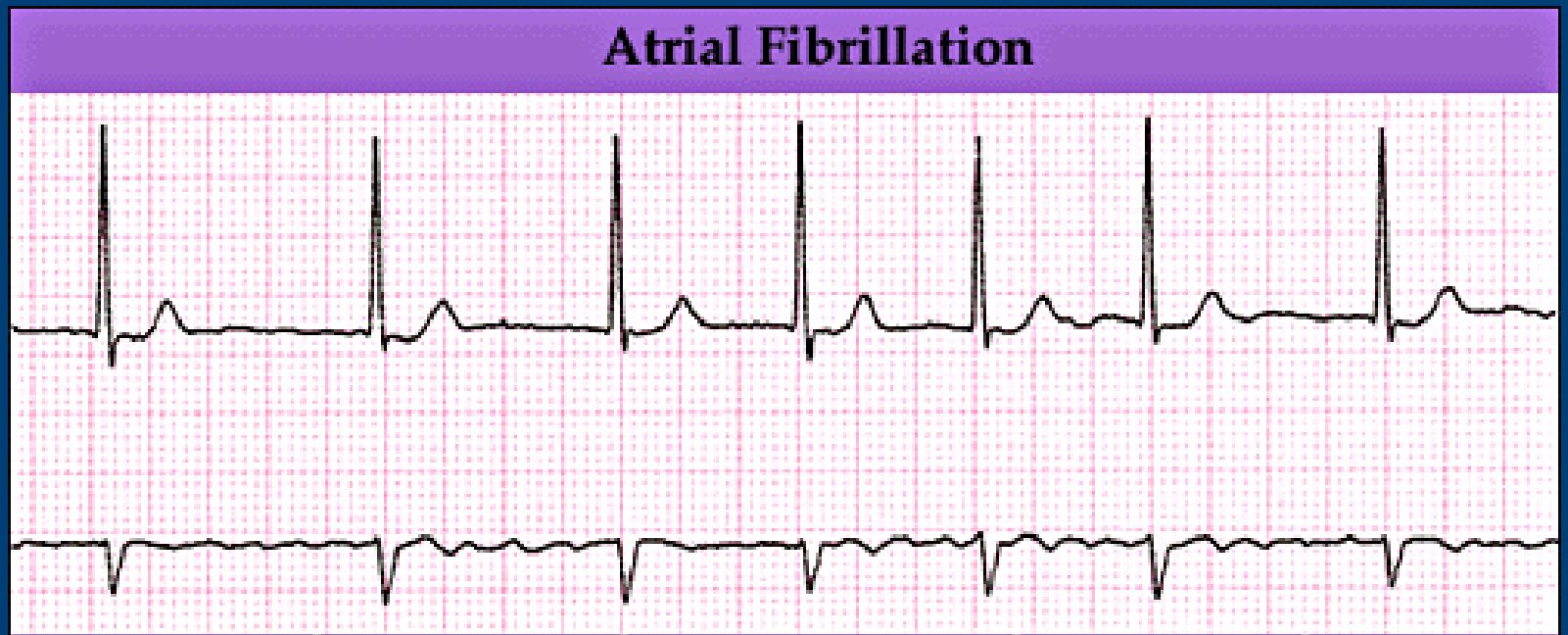


Atrial flutter ECG

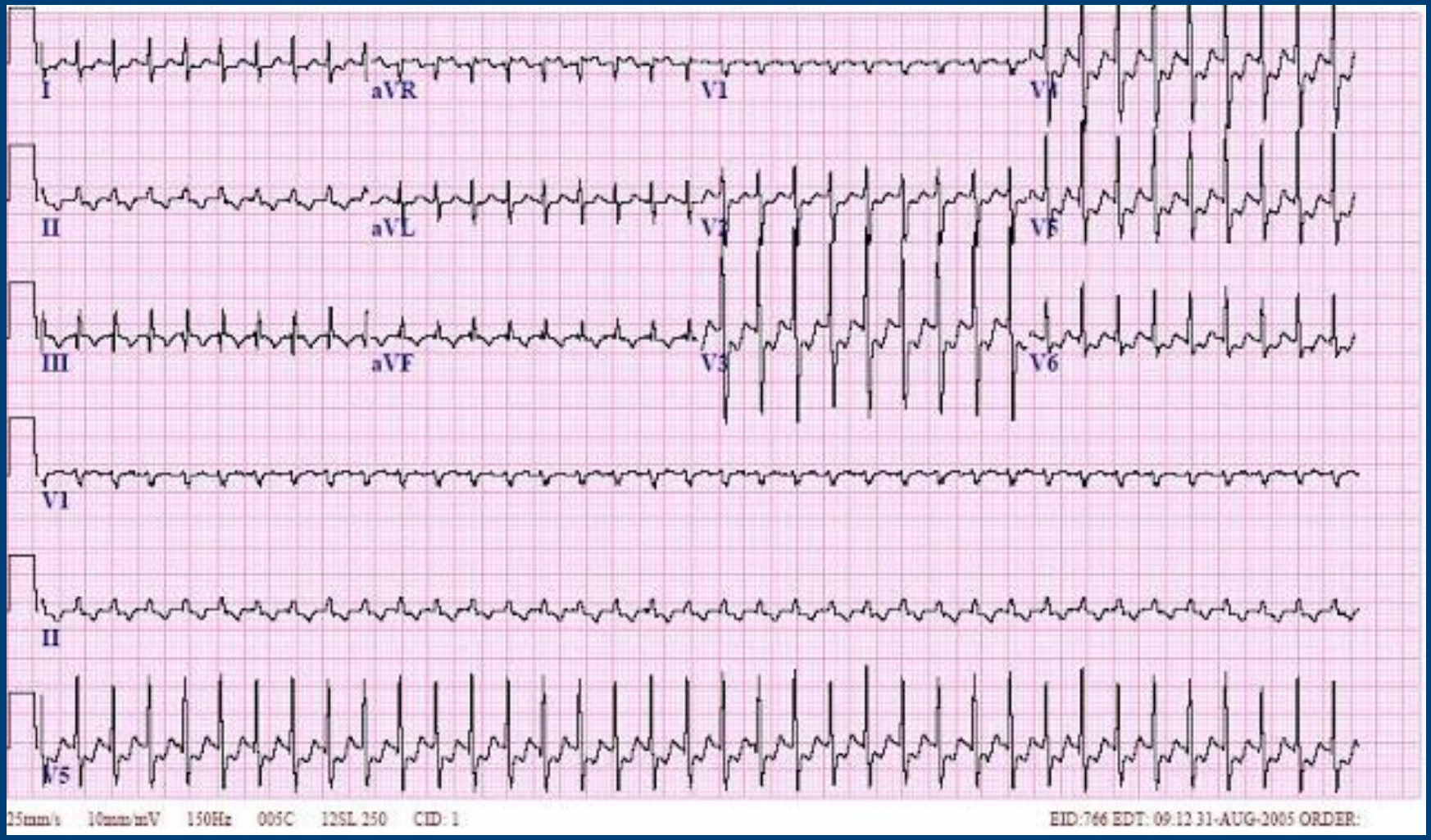


Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12

Atrial fibrillation ECG



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 350-650 bpm V: Slow to rapid	Irregular	Fibrillatory (fine to course)	N/A	<.12


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 150Hz 005C 12SL 250 CID: 1
EID:766 EDT: 09:12 31-AUG-2005 ORDER:

Causes of atrial fibrillation

- Ischaemic heart disease
- Hypertensive heart disease
- Rheumatic heart disease
- Thyrotoxicosis
- Alcohol misuse (acute or chronic)
- Cardiomyopathy (dilated or hypertrophic)
- Sick sinus syndrome
- Post-cardiac surgery
- Chronic pulmonary disease
- idiopathic (lone)

Wolff-Parkinson-White syndrome

- In this syndrome an accessory pathway (the bundle of Kent) connects the atria directly to the ventricles.
- It results from a failure of complete separation of the atria and ventricles during fetal development.
- Traditionally the Wolff-Parkinson-White syndrome has been classified into two types according to the electrocardiographic morphology of the precordial leads, type A and B.

Type A

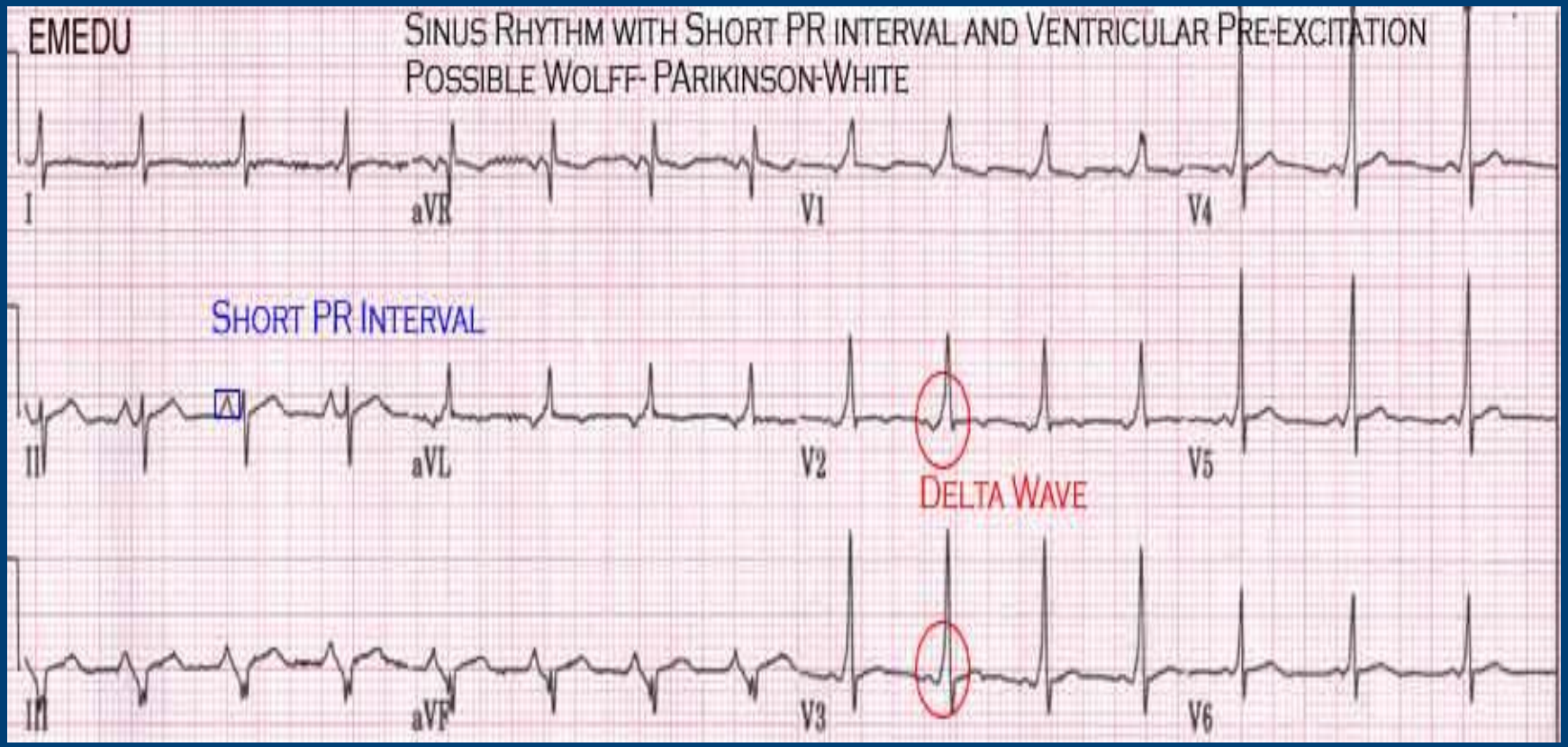
- The dominant R wave in lead V1 may be misinterpreted as right bundle branch block.
- ***Type A (dominant R wave in V1 lead) may be confused with:***

✓ Right bundle branch block

✓ Right ventricular hypertrophy

✓ Posterior myocardial infarction

Type A ECG


EMEDU
SINUS RHYTHM WITH SHORT PR INTERVAL AND VENTRICULAR PRE-EXCITATION
POSSIBLE WOLFF- PARIKINSON-WHITE
I
aVR
V1
V4
SHORT PR INTERVAL
II
aVL
V2
V5
DELTA WAVE
III
aVF
V3
V6

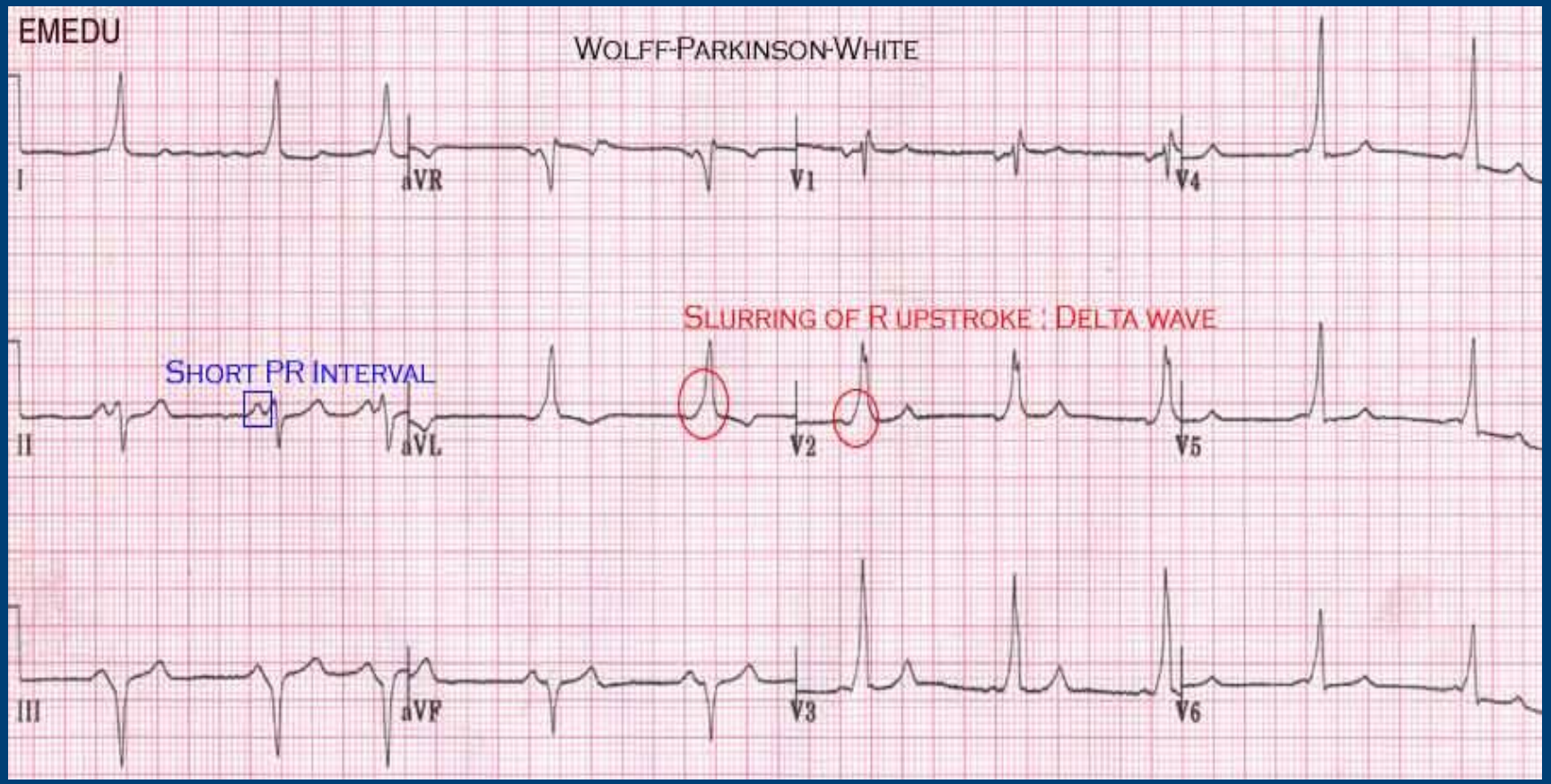
Type B

- In type B, the delta wave and QRS complex are predominantly negative in leads V1 and V2 and positive in the other precordial leads, resembling left bundle branch block.
- ***Type B (negative QRS complex in V1 lead) may be confused with:***

✓ Left bundle branch block

✓ Anterior myocardial infarction

Type B ECG


EMEDU
WOLFF-PARKINSON-WHITE
I
aVR
V1
V4
SLURRING OF R UPSTROKE : DELTA WAVE
SHORT PR INTERVAL
II
aVL
V2
V5
III
aVF
V3
V6

Varieties of broad complex tachycardia

- **Ventricular**

Regular

✓ Monomorphic ventricular tachycardia

Irregular

✓ Torsades de pointes tachycardia

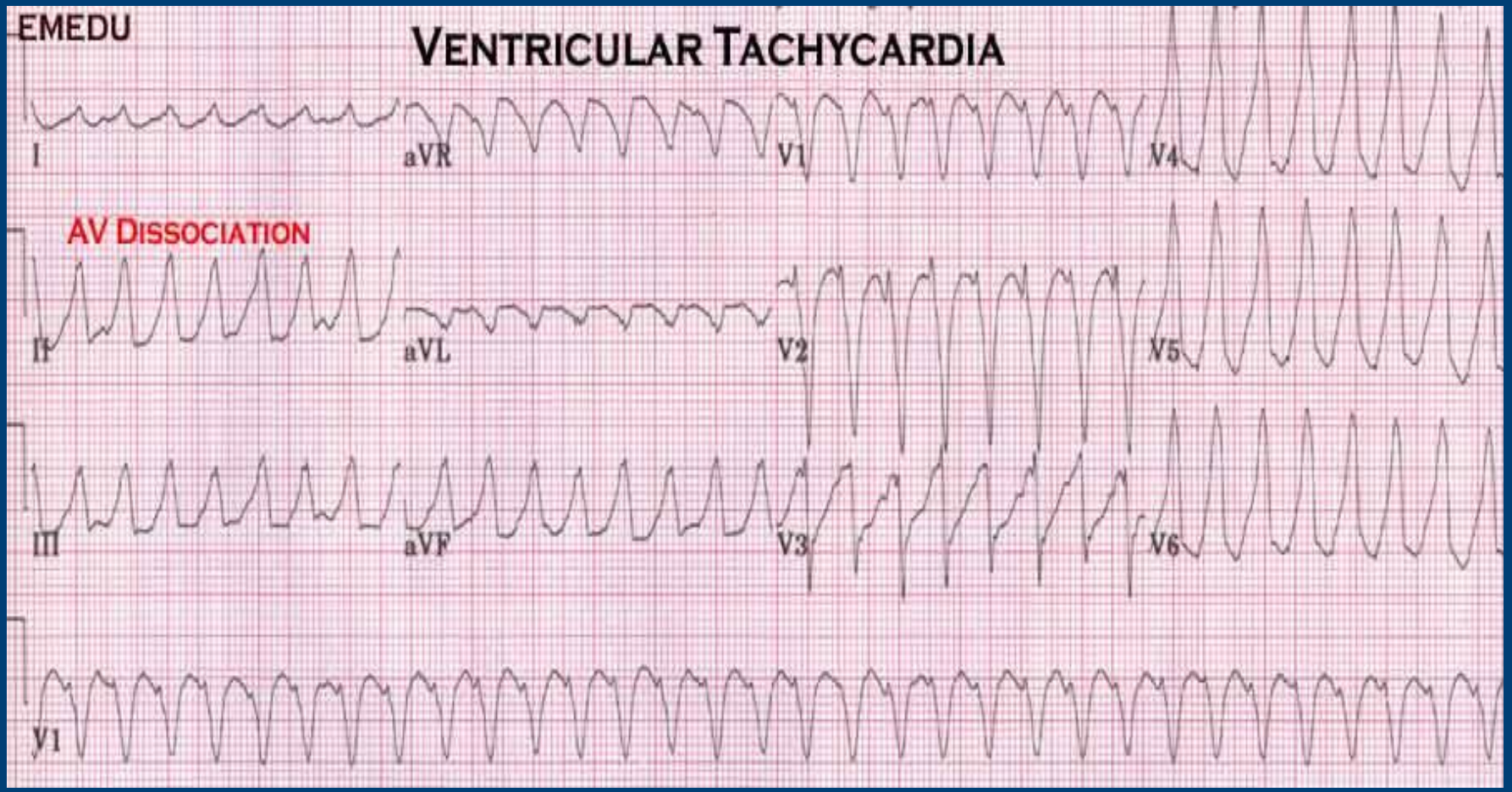
✓ Polymorphic ventricular tachycardia

- **Supraventricular**

VT

- QRS complexes are wide and irregular in shape
- Usually secondary to infarction
- As the rhythm originates in the ventricles, there is a
- broad QRS complex
- Hence it is one of the causes of a broad complex
- tachycardia
- Need to differentiate with
- Supraventricular tachycardia with aberrant conduction

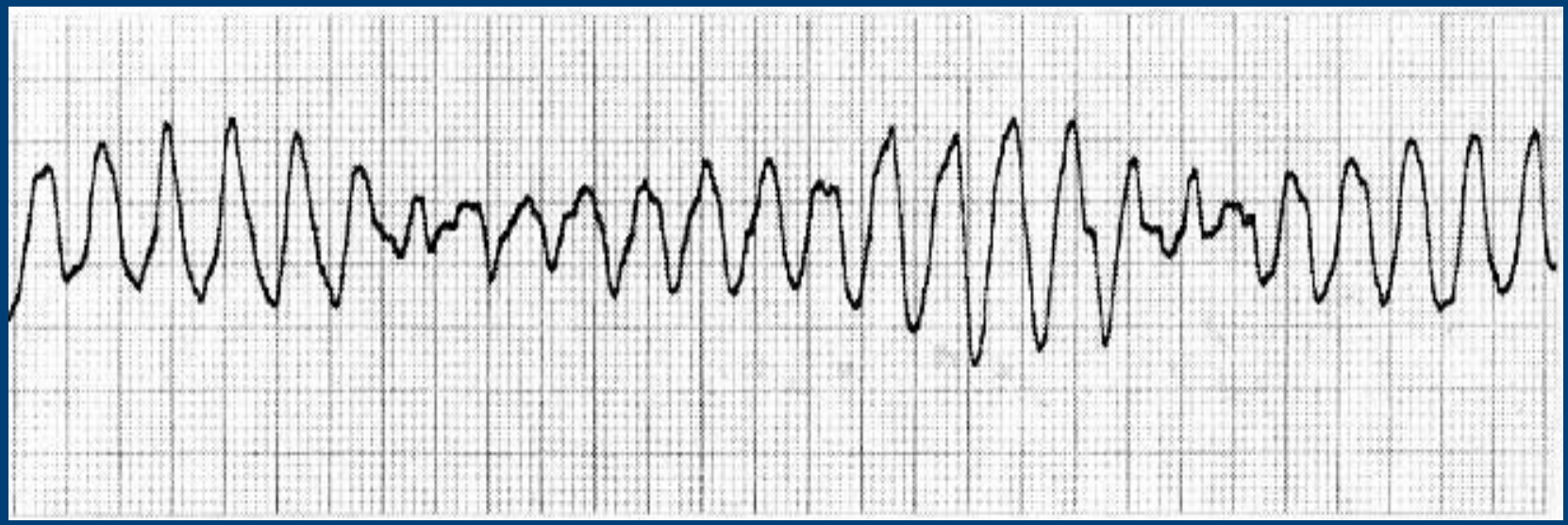
VT ECG


EMEDU
VENTRICULAR TACHYCARDIA
I
aVR
V1
V4
AV DISSOCIATION
II
aVL
V2
V5
III
aVF
V3
V6
V1

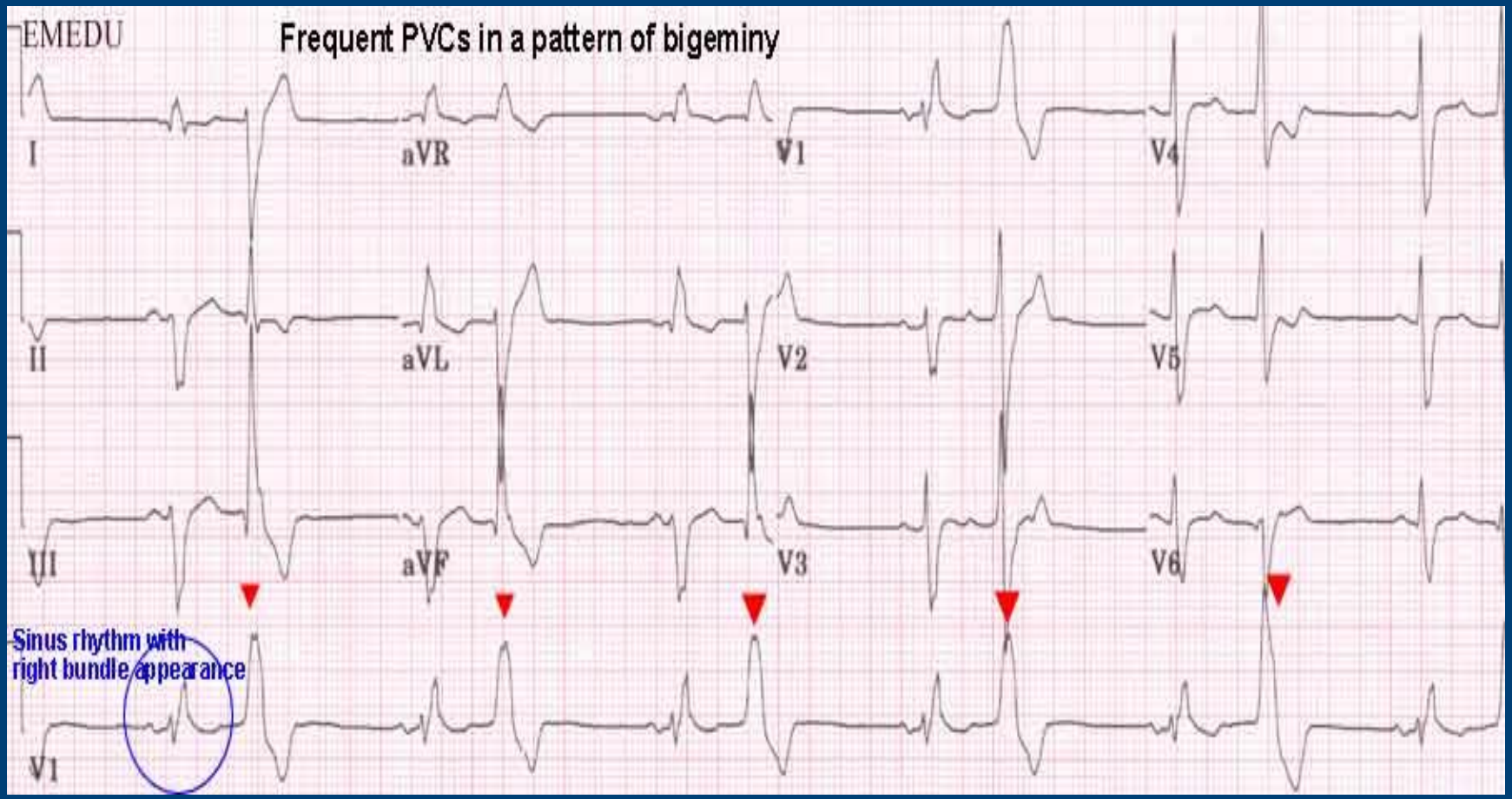
Torsades de pointes tachycardia

- is a type of polymorphic ventricular tachycardia
- In sinus rhythm the QT interval is prolonged and prominent U waves may be seen.
- Occasionally it may be prolonged or degenerate into ventricular fibrillation.
- It is associated with conditions that prolong the QT interval.
- Ability to recognise torsades de pointes is important because its management is different from the management of other ventricular tachycardias

torsades de pointes ECG



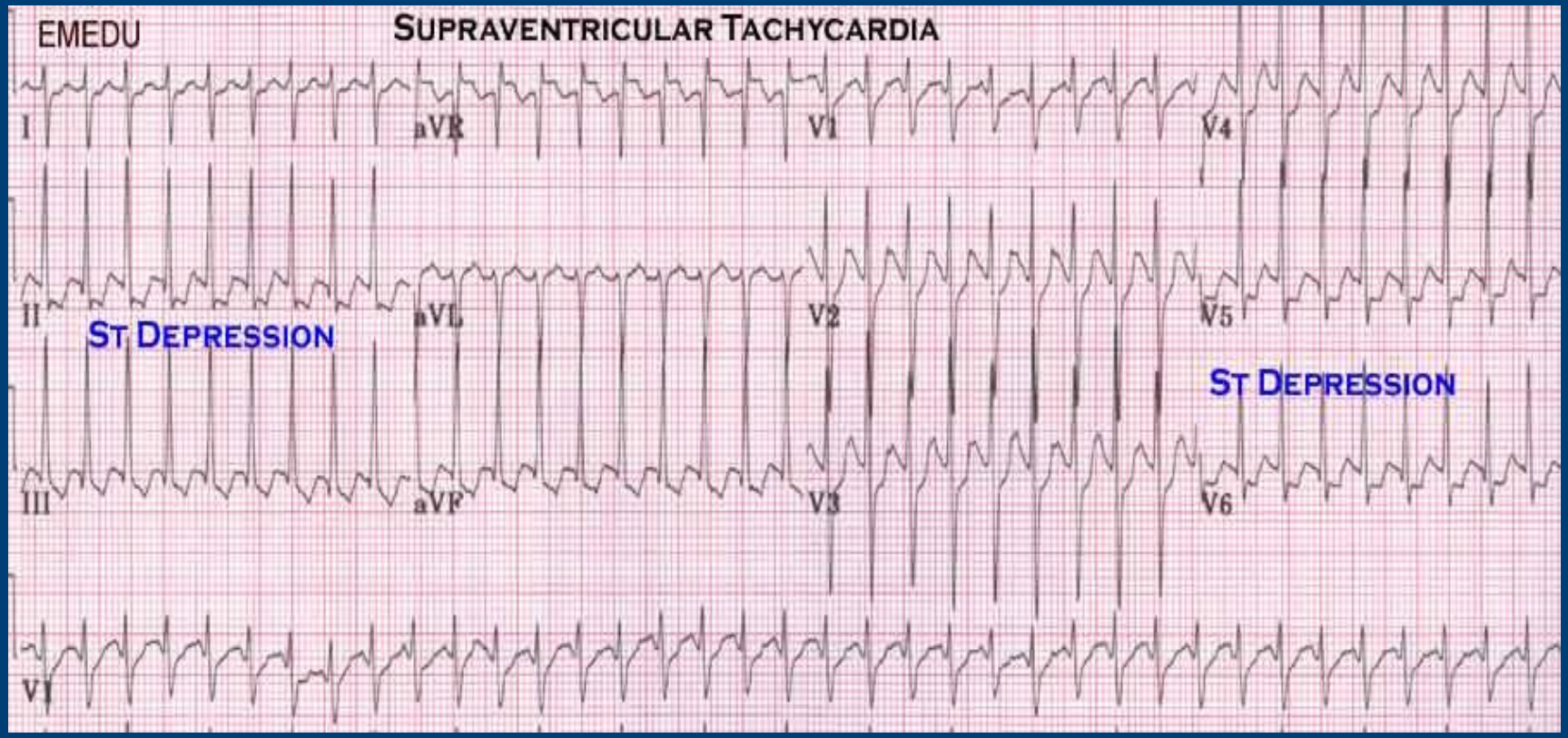
Bigeminy

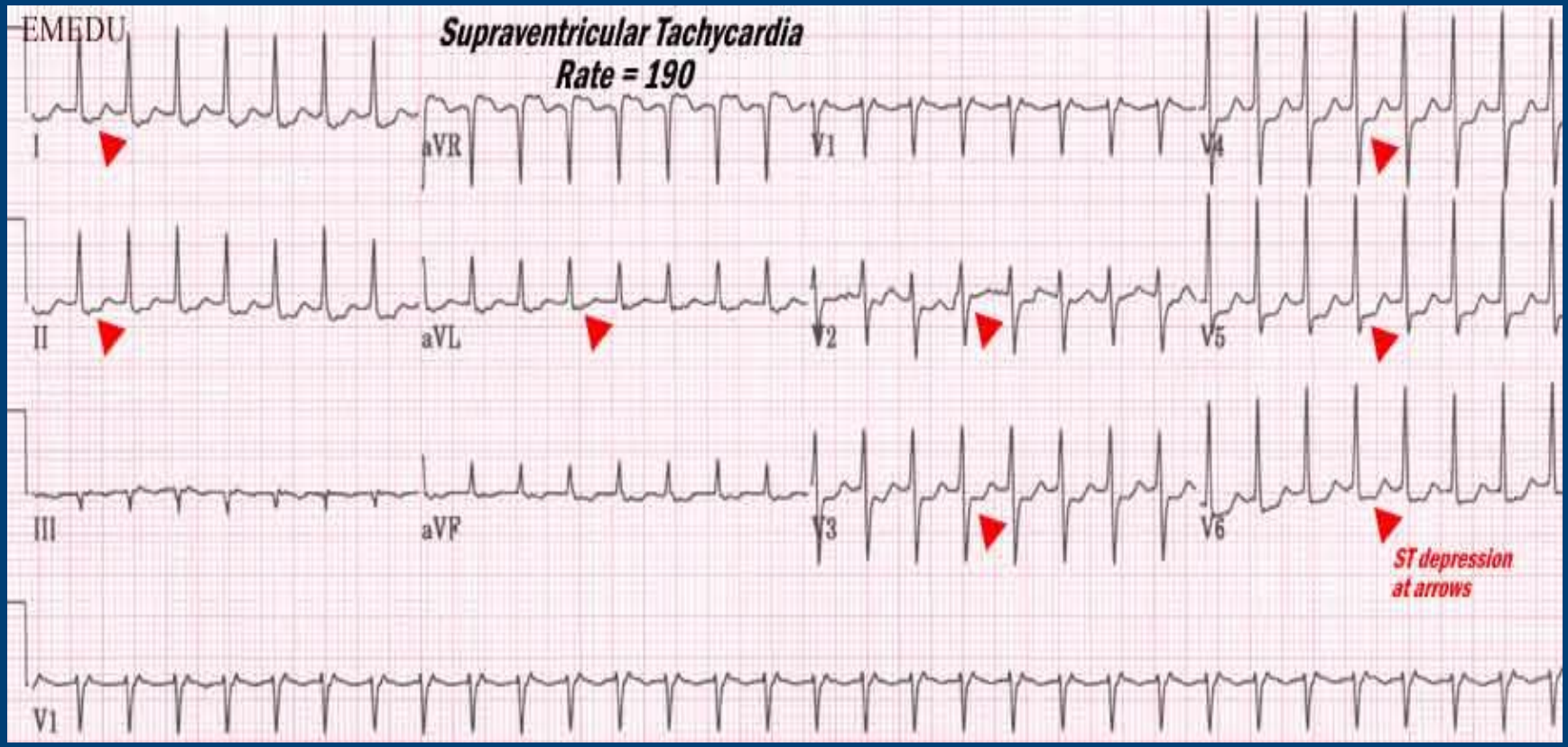

EMEDU
Frequent PVCs in a pattern of bigeminy
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Sinus rhythm with
right bundle appearance
V1

SVT

- Regular tachycardia ~140-280 bpm.
- QRS complexes usually narrow (< 120 ms) unless pre-existing bundle branch block, accessory pathway, or rate related aberrant conduction.
- ST-segment depression may be seen with or without underlying coronary artery disease.

SVT ECG


EMEDU
SUPRAVENTRICULAR TACHYCARDIA
I
aVR
V1
V4
II
aVL
V2
V5
ST DEPRESSION
ST DEPRESSION
III
aVF
V3
V6
VI


EMEDU
Supraventricular Tachycardia
Rate = 190
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
ST depression
at arrows
V1

Differentiation between ventricular tachycardia and supraventricular tachycardia with bundle branch block

- *If the tachycardia has a right bundle branch block morphology (a predominantly positive QRS complex in lead V1), a ventricular origin is suggested if there is:*

✓ QRS complex with duration > 0.14 s

✓ Axis deviation

✓ A QS wave or predominantly negative complex in lead V6

✓ Concordance throughout the chest leads, with all deflections positive

✓ A single (R) or biphasic (QR or RS) R wave in lead V1

✓ A triphasic R wave in lead V1, with the initial R wave taller than the secondary R wave and an S wave that passes through the isoelectric line

Cont…

- *If the tachycardia has a left bundle branch block morphology (a predominantly negative deflection in lead V1), a ventricular origin is suggested if there is:*

✓ Axis deviation

✓ QRS complexes with duration > 0.16 s

✓ A QS or predominantly negative deflection in lead V6

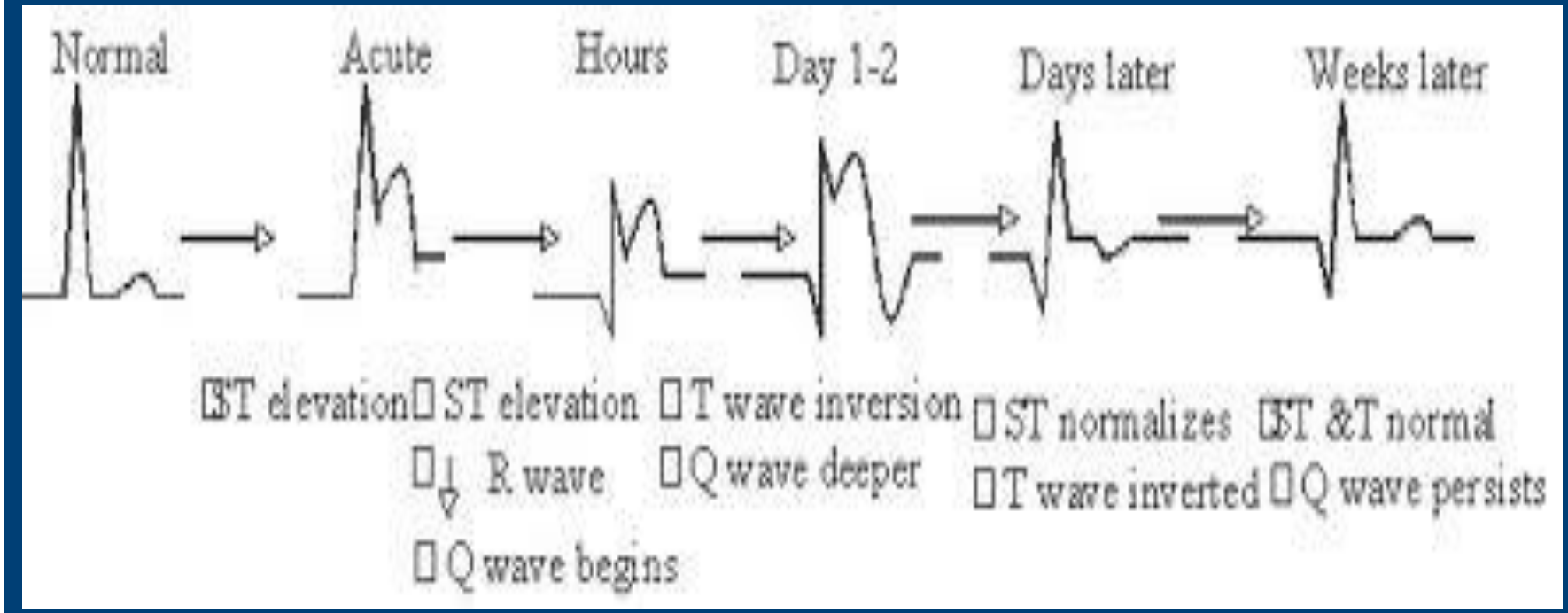
✓ Concordance throughout the chest leads, with all deflections negative

✓ An rS complex in lead V1

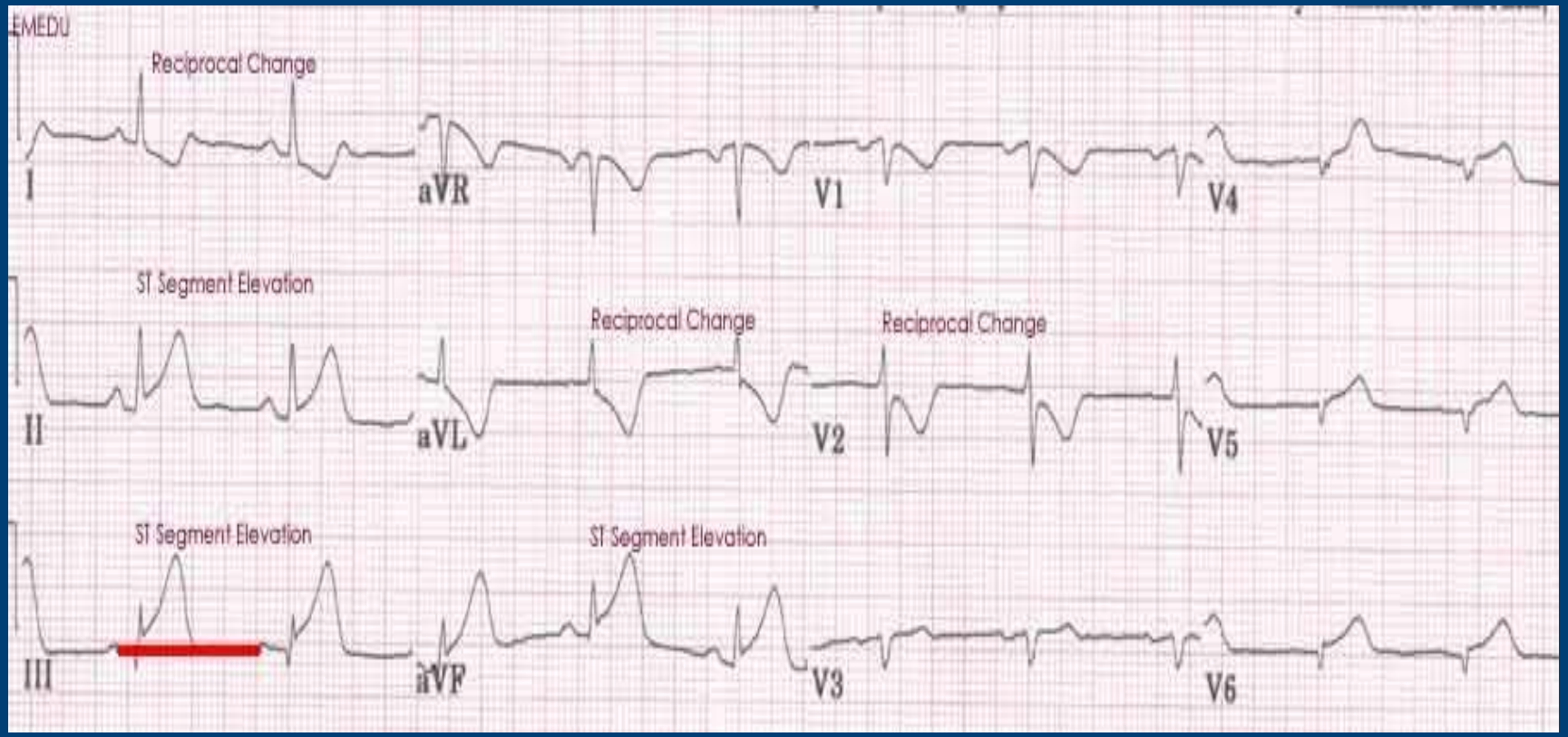
Causes of ST segment elevation

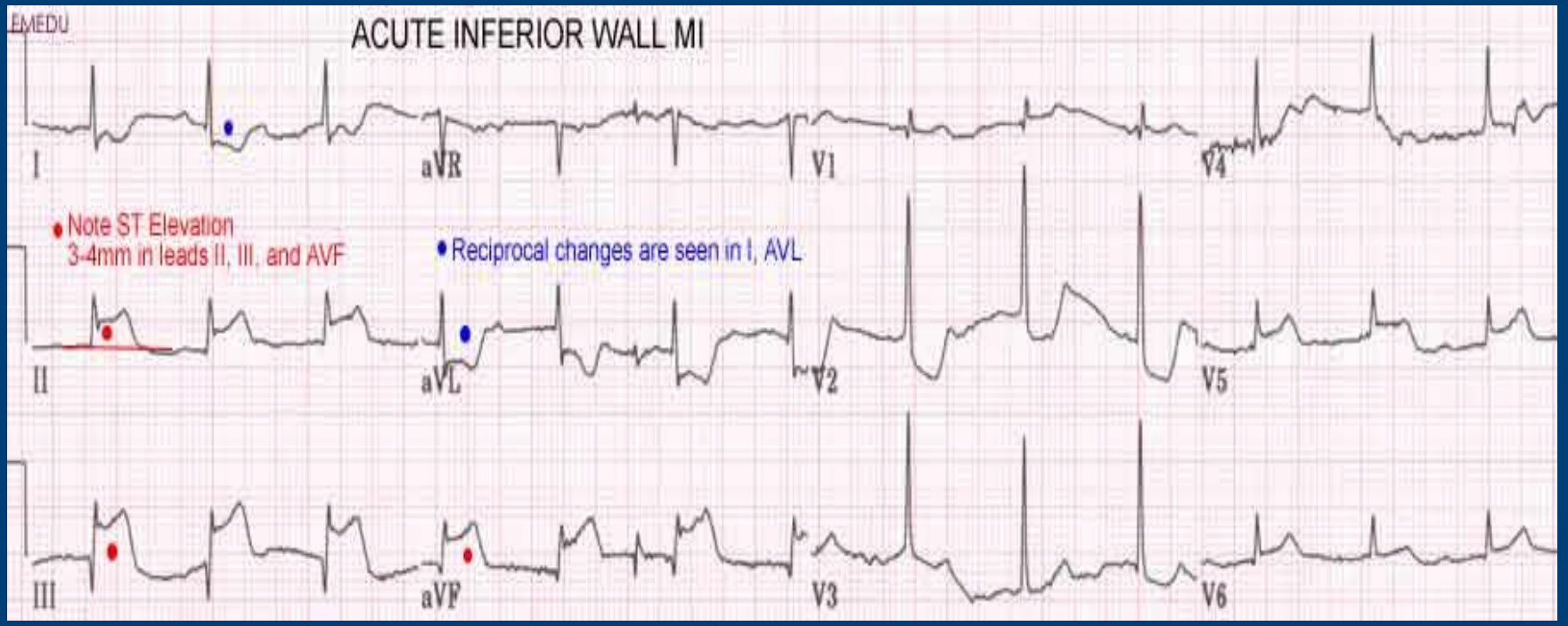
- Acute myocardial infarction
- Benign early repolarisation
- Left bundle branch block
- Left ventricular hypertrophy
- Ventricular aneurysm
- Coronary vasospasm/Printzmetal's angina
- Pericarditis
- Subarachnoid haemorrhage

Sequence of changes seen during evolution of myocardial infarction

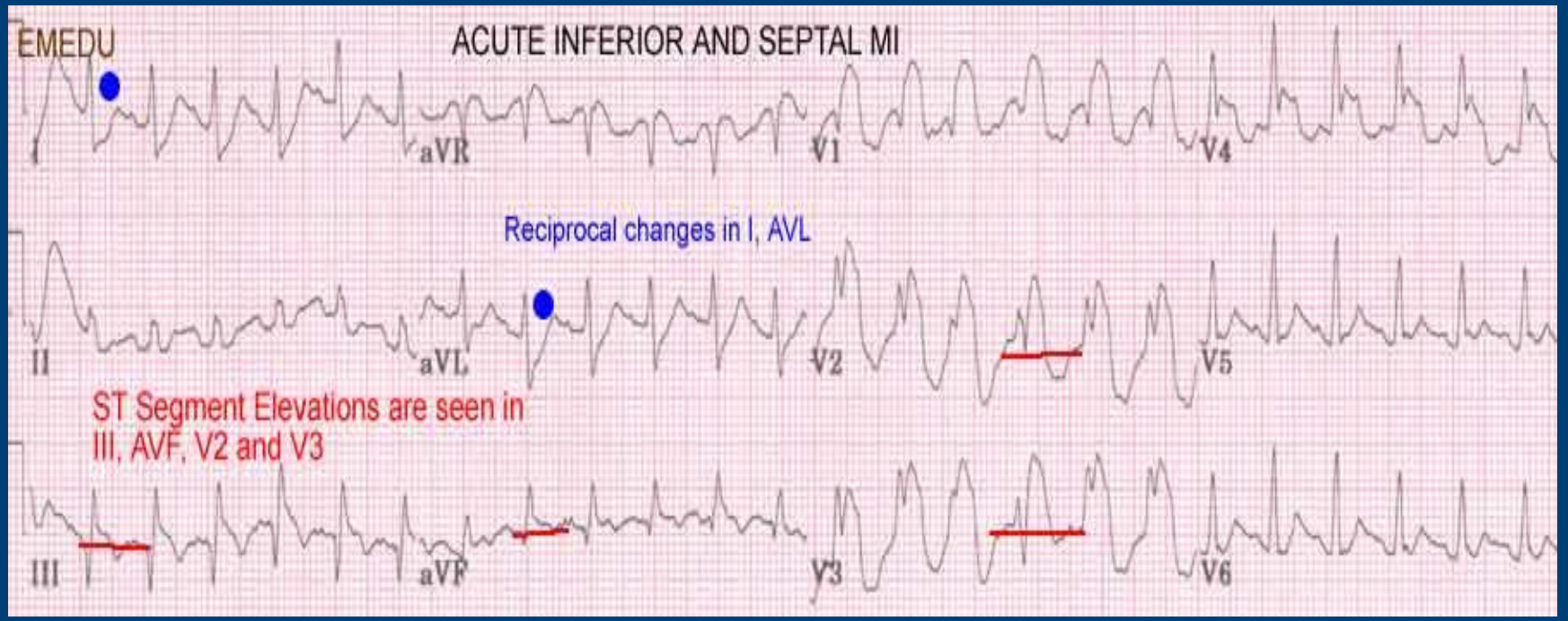


Inferior MI

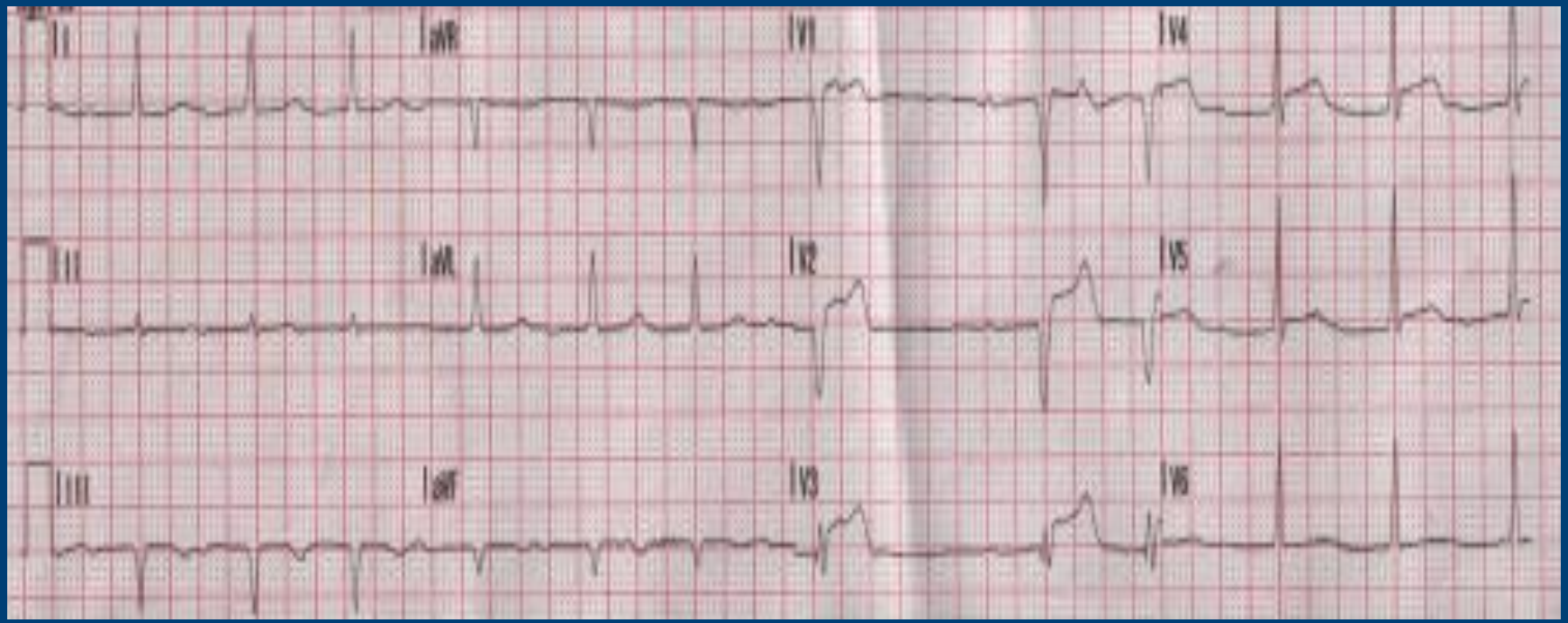

EMEDU
Reciprocal Change
I
aVR
V1
V4
ST Segment Elevation
Reciprocal Change
Reciprocal Change
II
aVL
V2
V5
ST Segment Elevation
ST Segment Elevation
III
aVF
V3
V6


EMEDU
ACUTE INFERIOR WALL MI
I
aVR
V1
V4
Note ST Elevation
3-4mm in leads II, III, and AVF
Reciprocal changes are seen in I, AVL
II
aVL
V2
V5
III
aVF
V3
V6

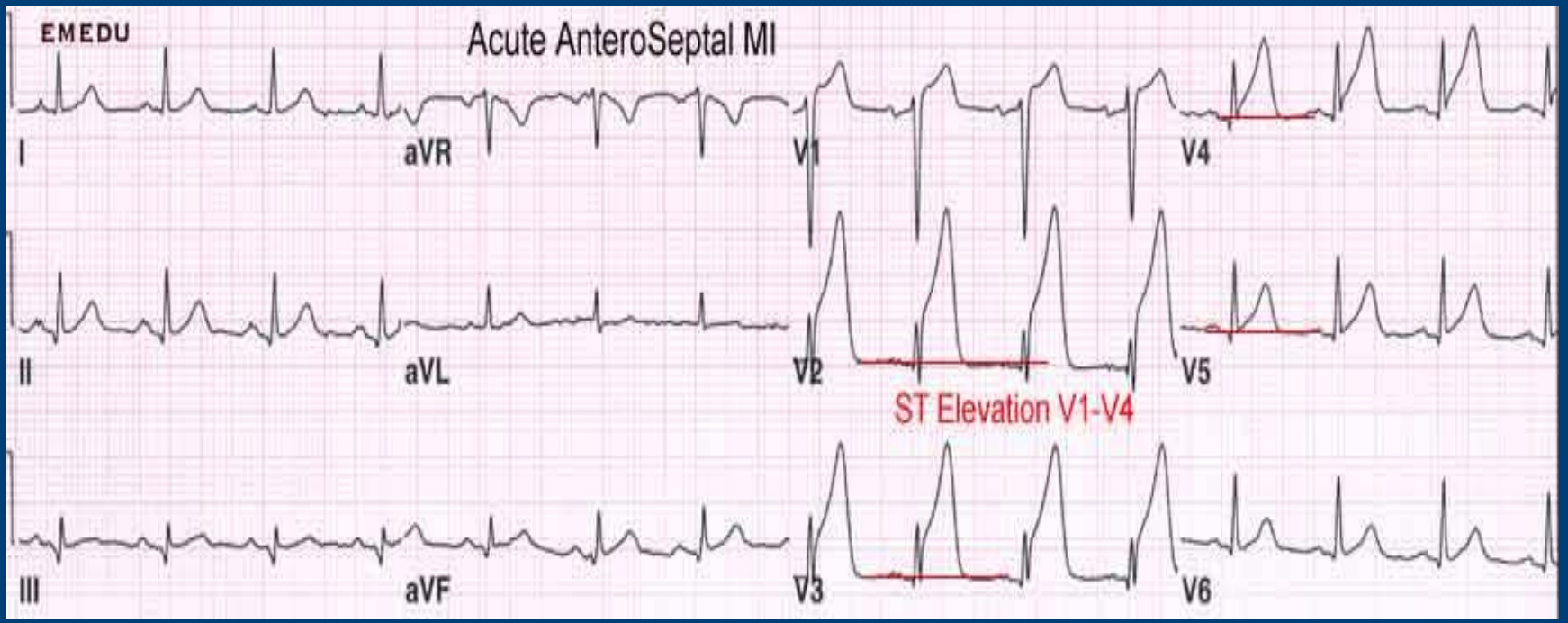
Inferio-septal MI



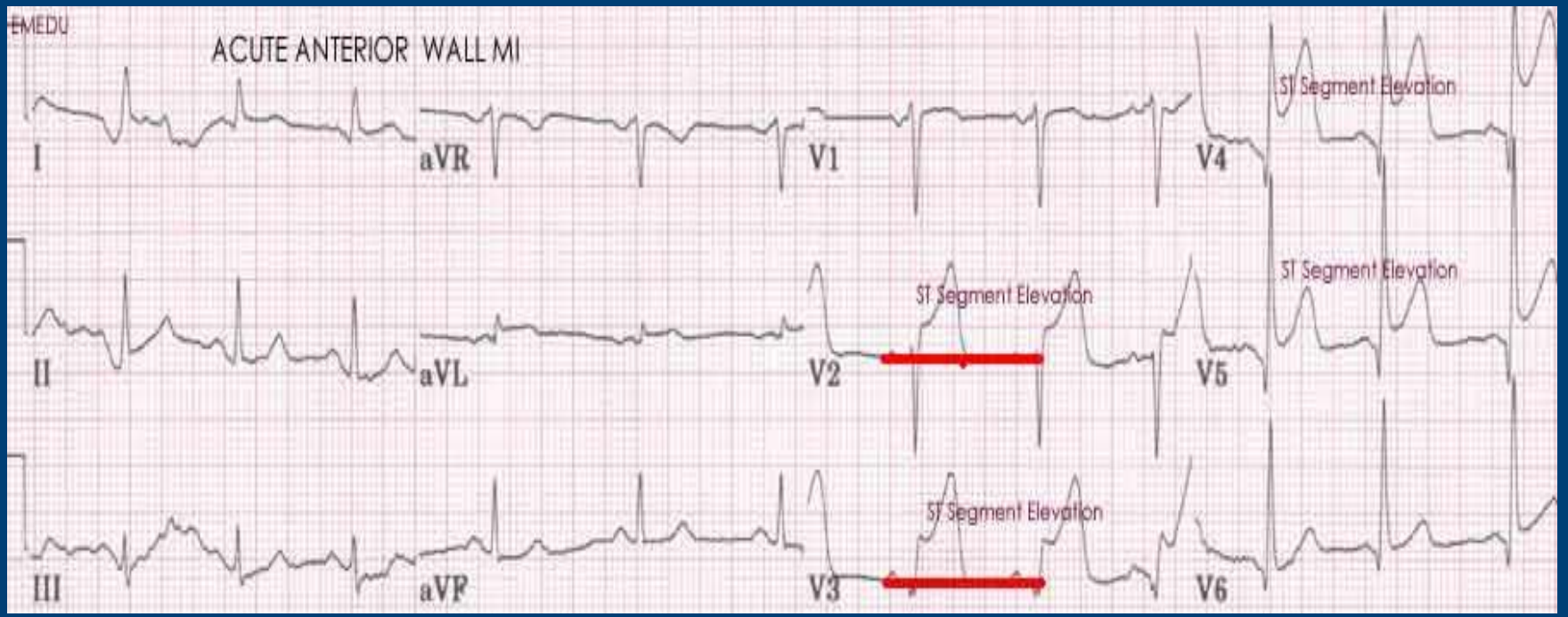
Anterior MI



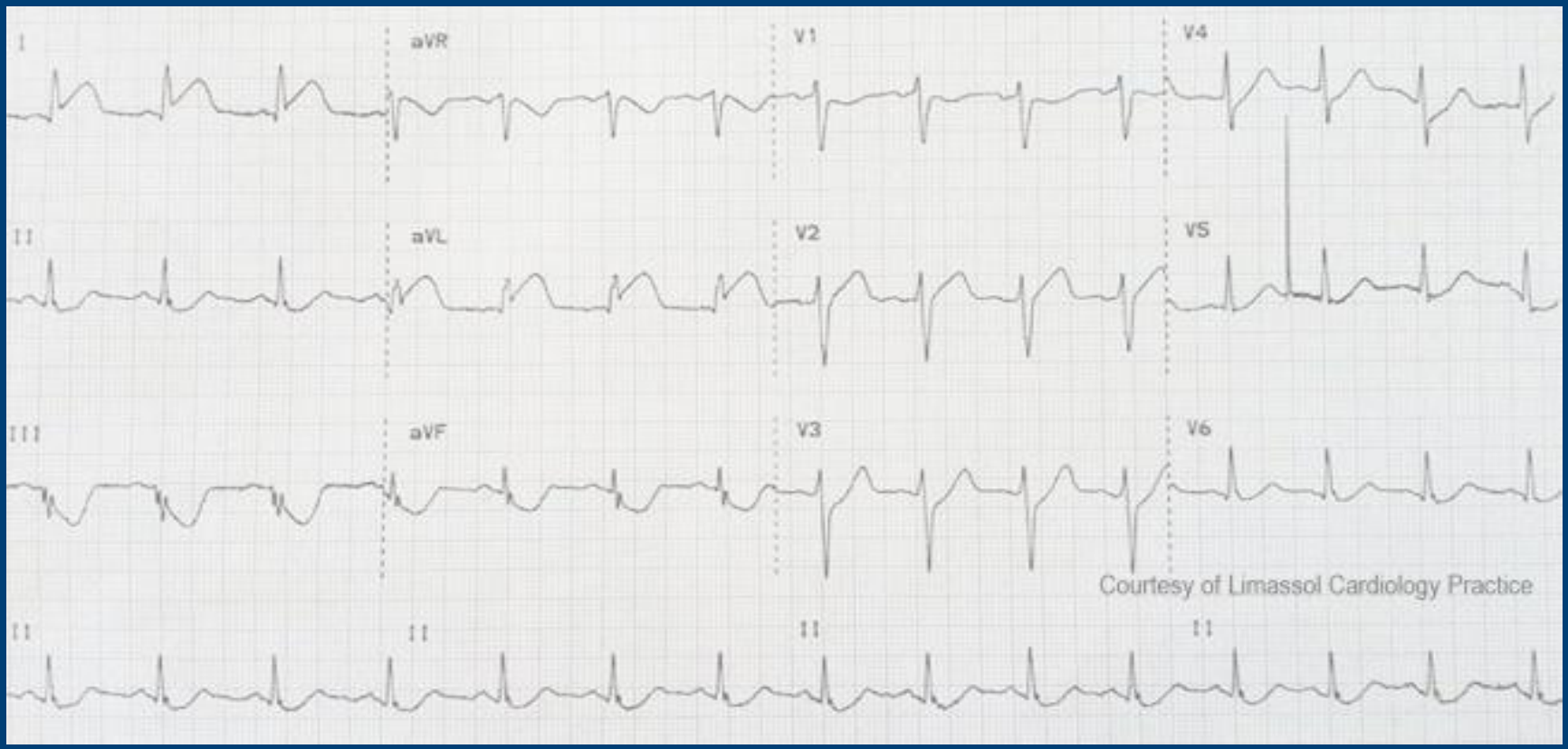
Antero-septal MI



Anterio-lateral MI

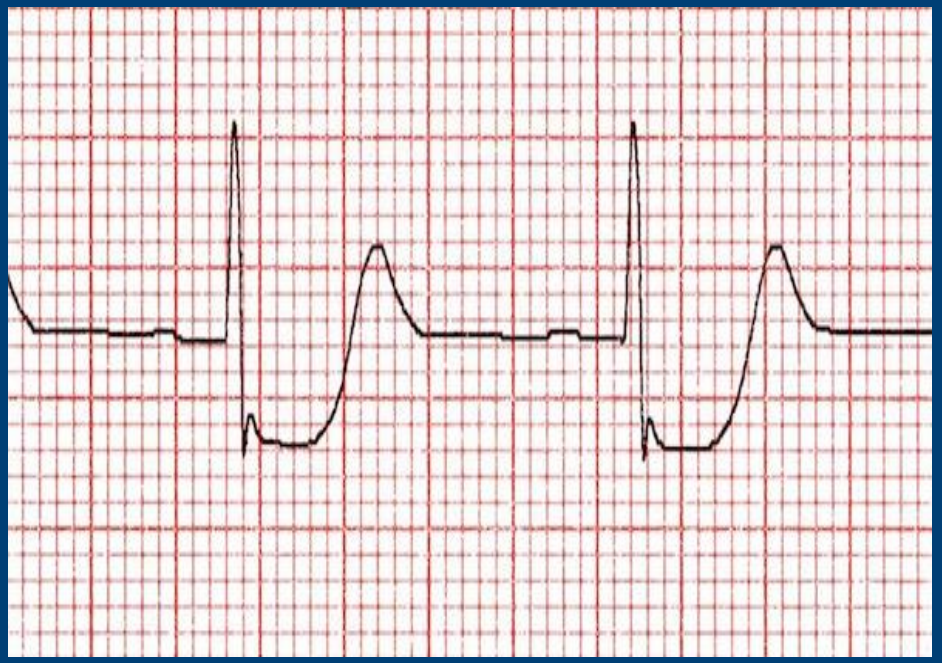


Lateral MI

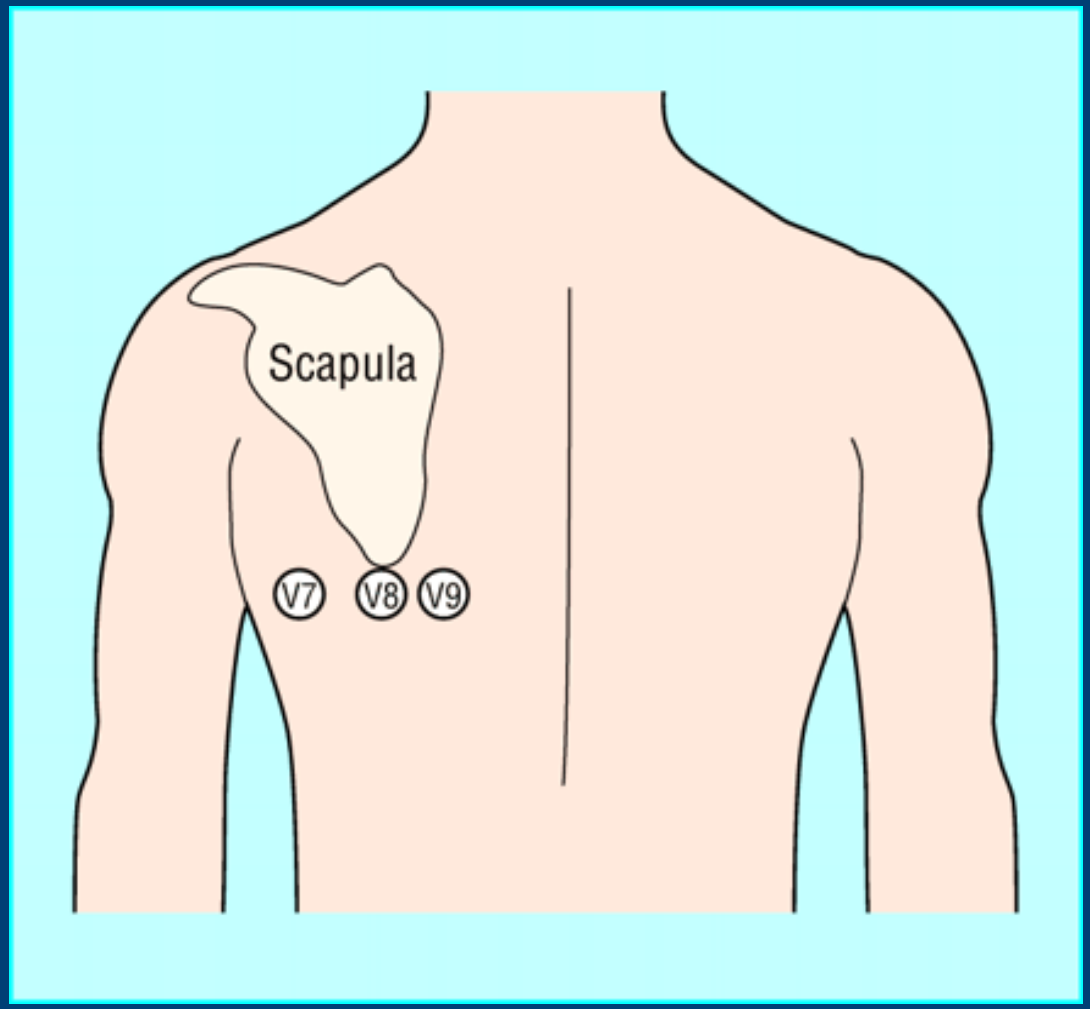

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
II
II
II
Courtesy of Limassol Cardiology Practice

Posterior MI

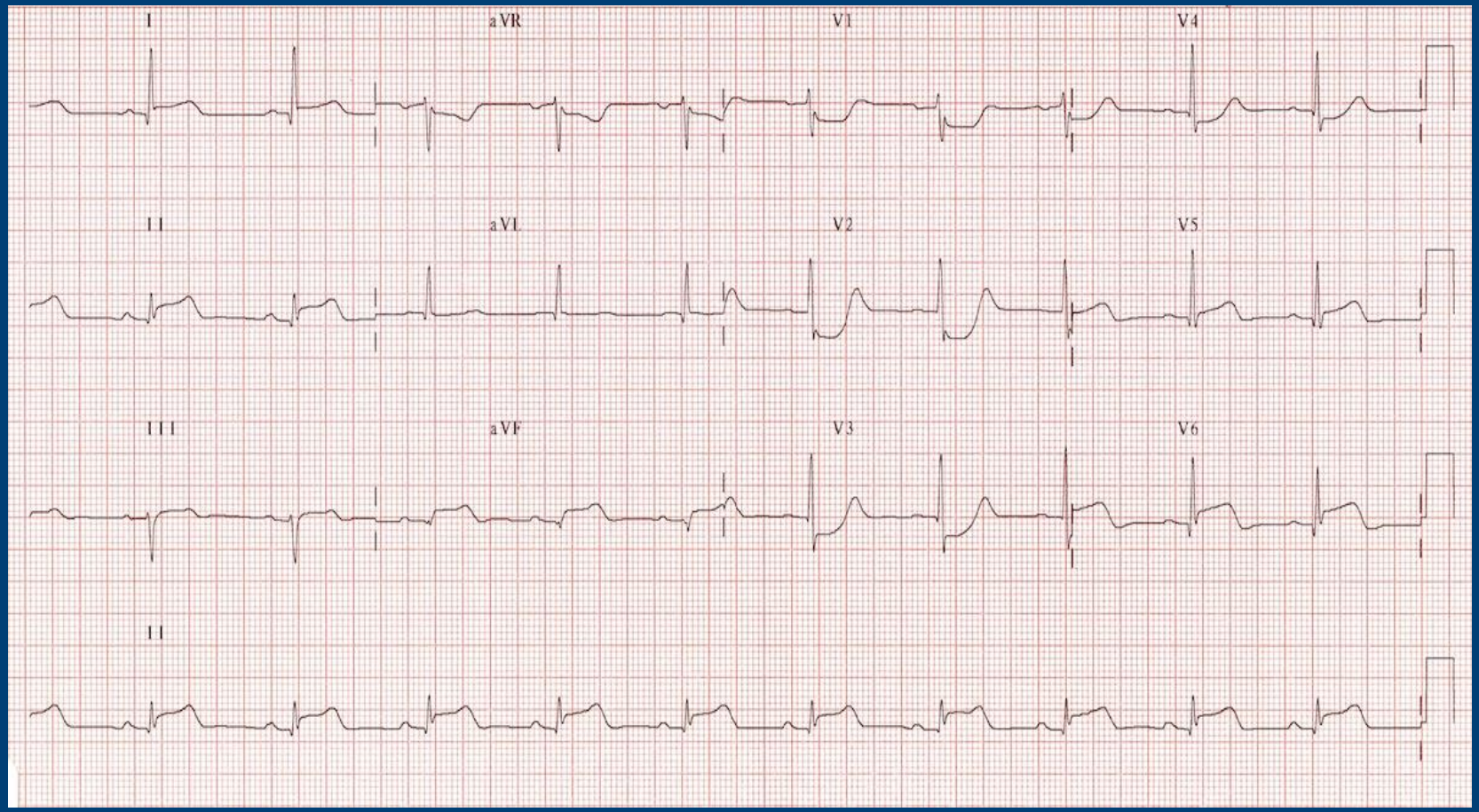
- Posterior MI is suggested by the following changes in V1-3:
  - Horizontal ST depression
  - Tall, broad R waves (>30ms)
  - Upright T waves
  - Dominant R wave (R/S ratio > 1) in V2



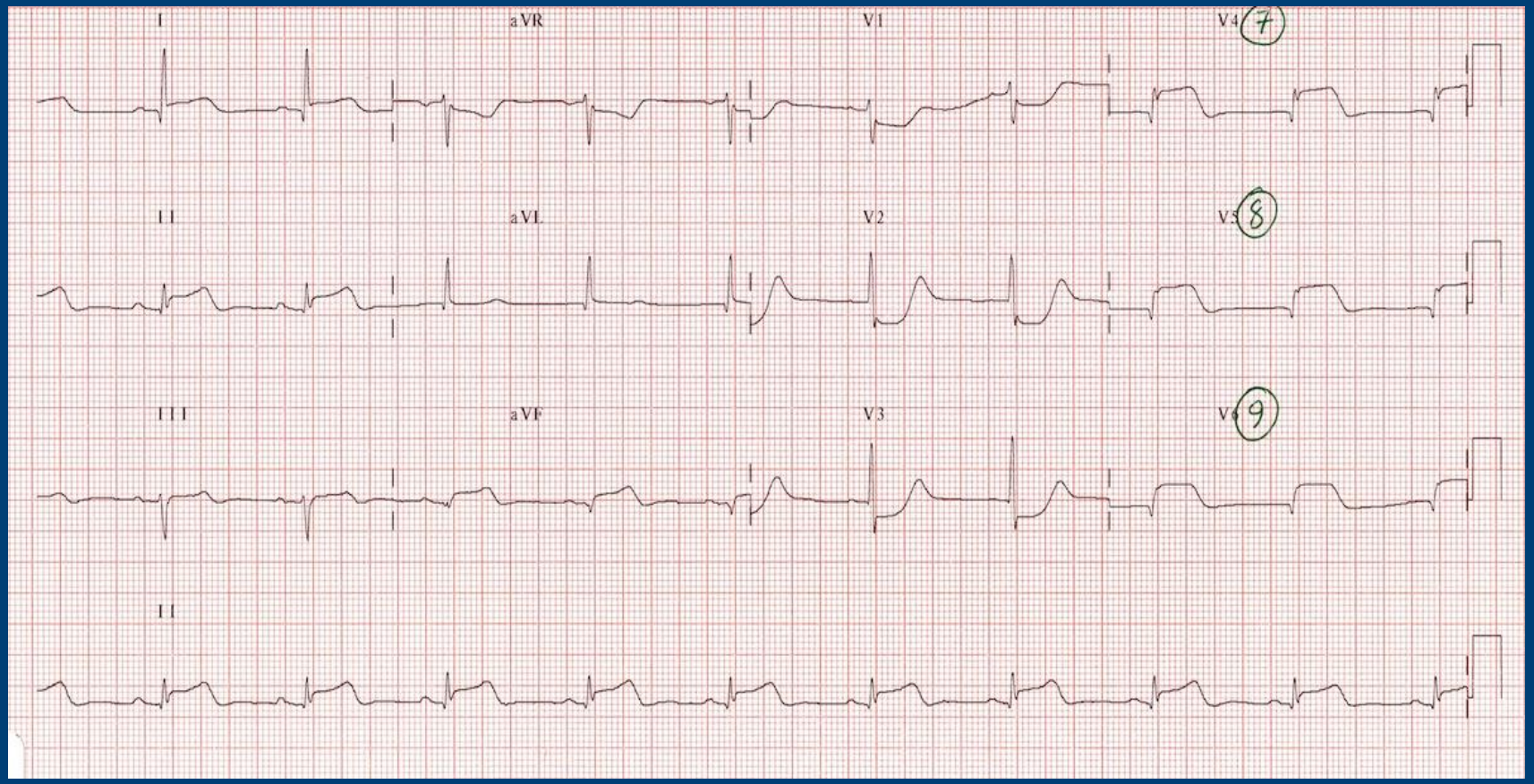
Posterior MI(V7,V8,V9)



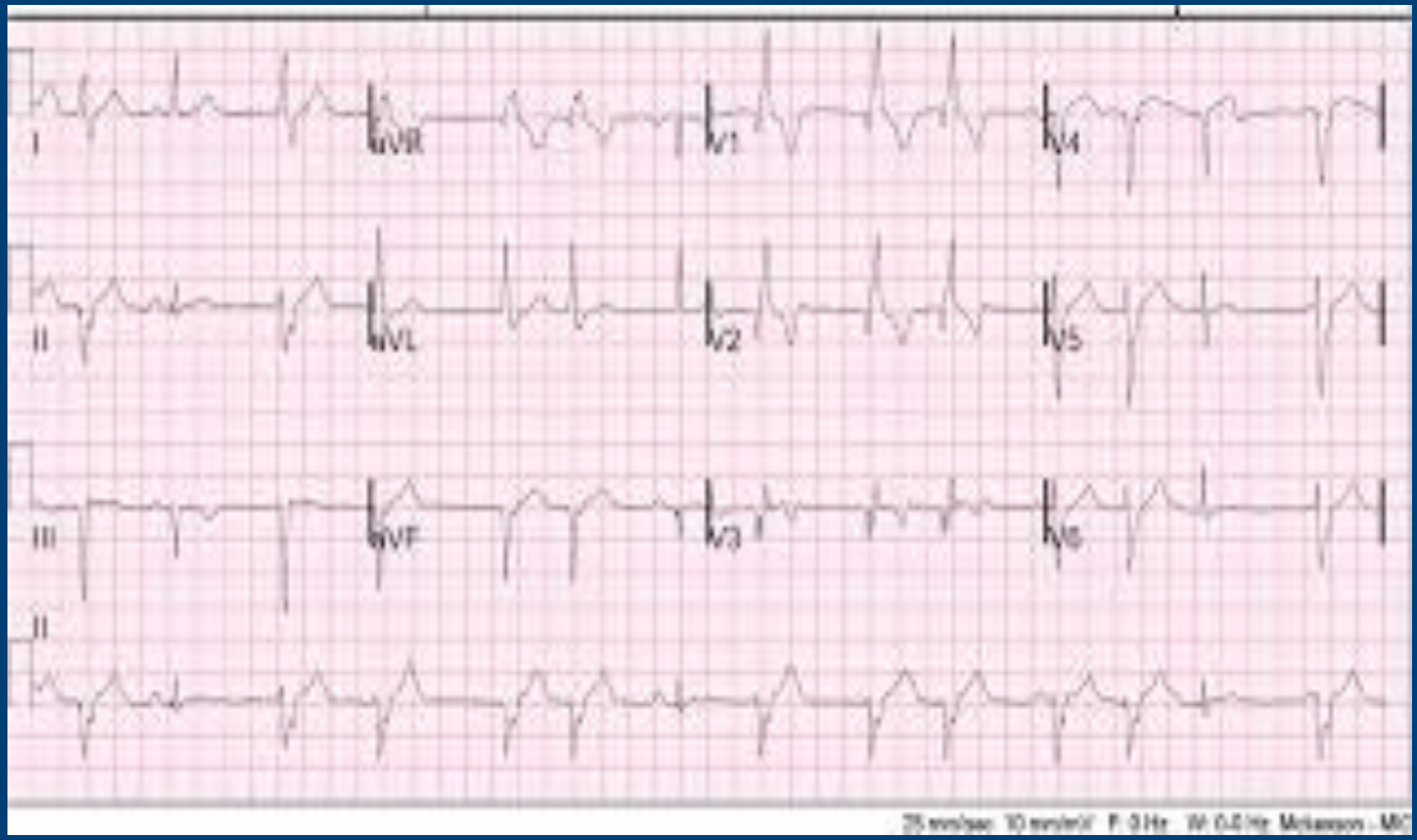
Suspeceted Posterior MI



ECG after V7,V8,V9



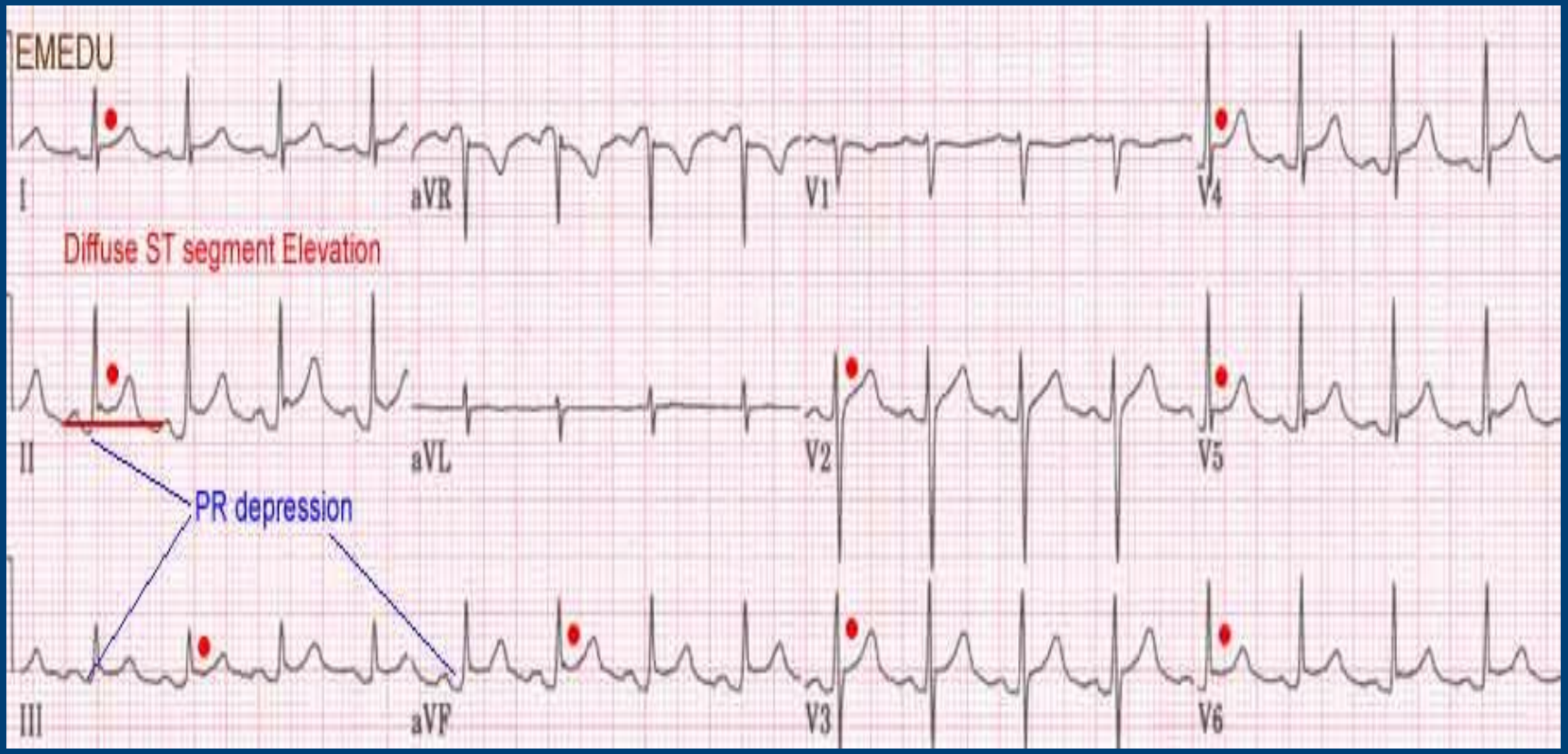
Posrterior MI with tall R in v1



Pericarditis

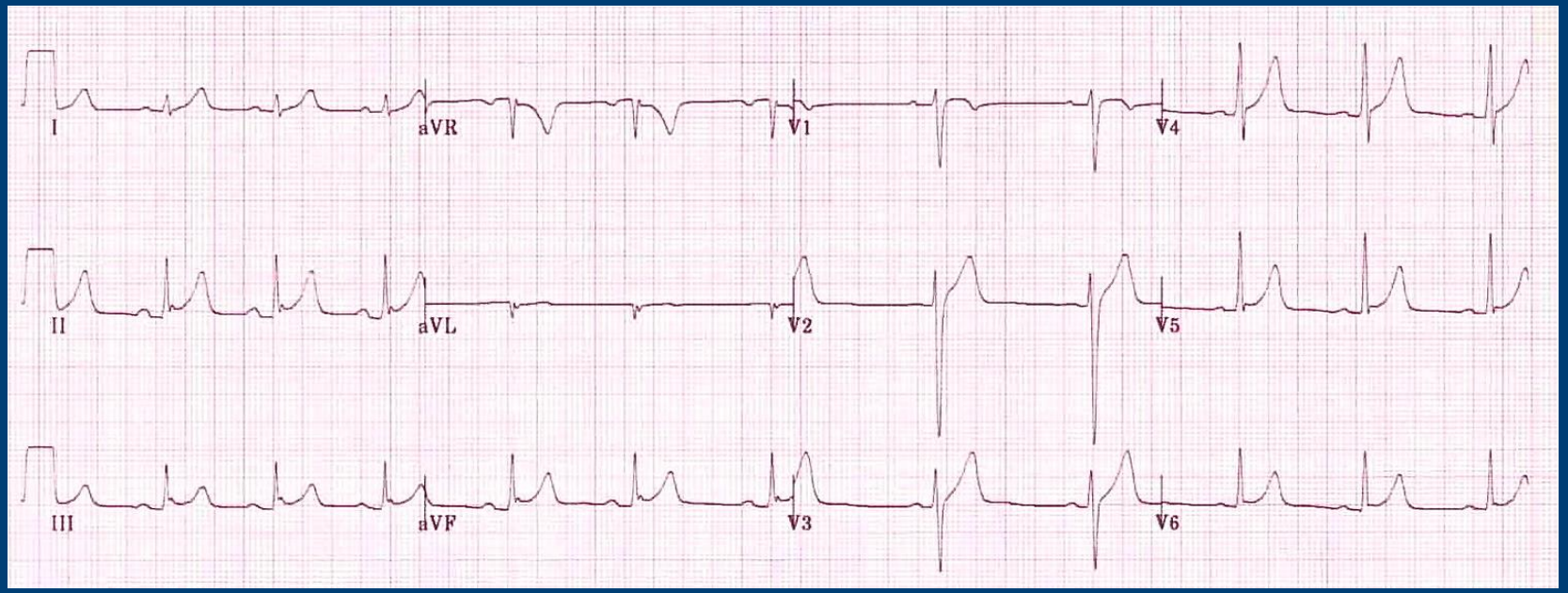
- Widespread concave ST elevation and PR depression throughout most of the limb leads (I, II, III, aVL, aVF) and precordial leads (V2-6).
- Reciprocal ST depression and PR elevation in lead aVR (± V1).
- Sinus tachycardia is also common in acute pericarditis due to pain and/or pericardial effusion.

Pericarditis ECG

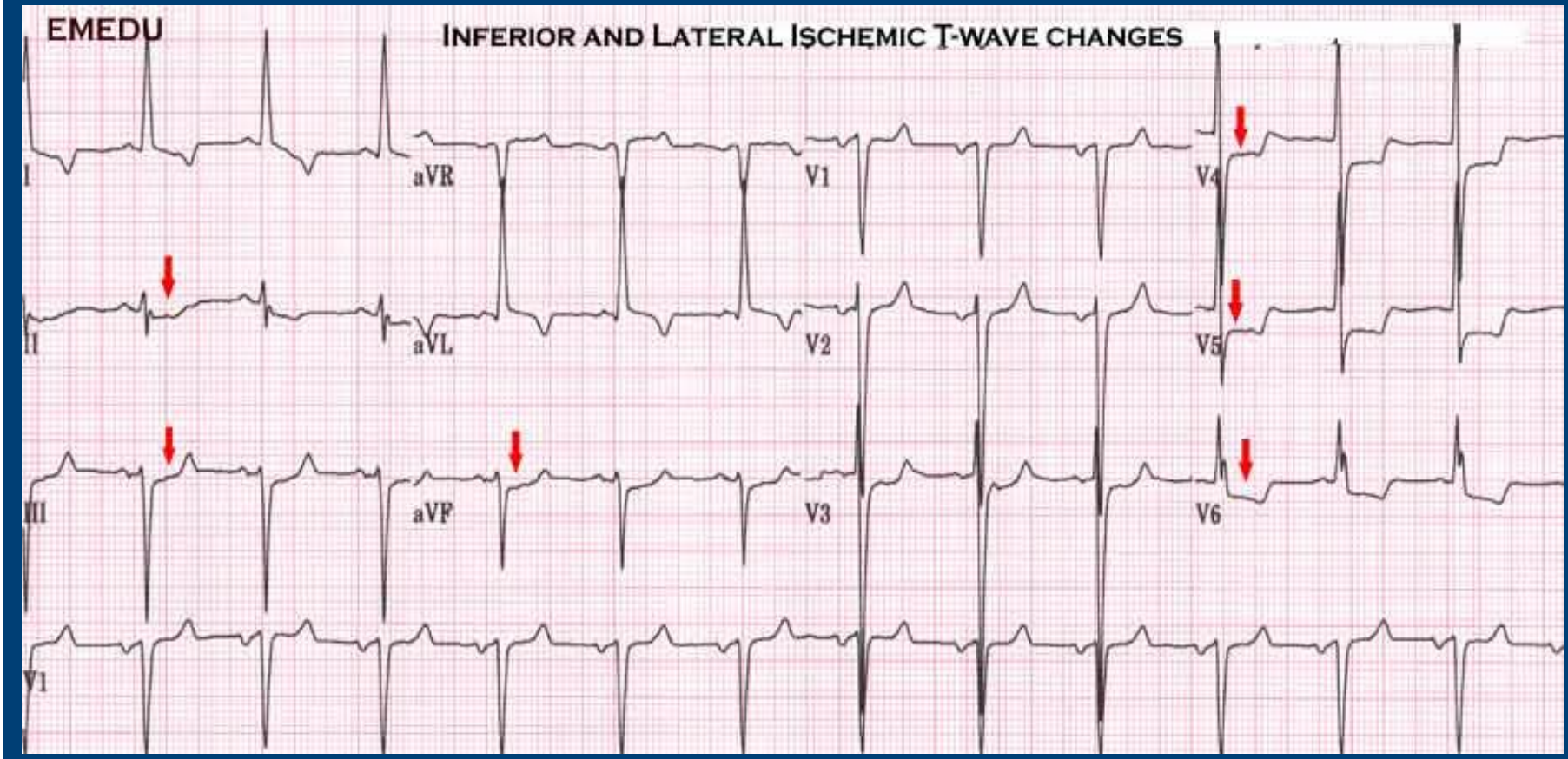


Early repolarization

- ST elevation is usually < 2mm in the precordial leads and < 0.5mm in the limb leads, although precordial STE may be up to 5mm in some instances.



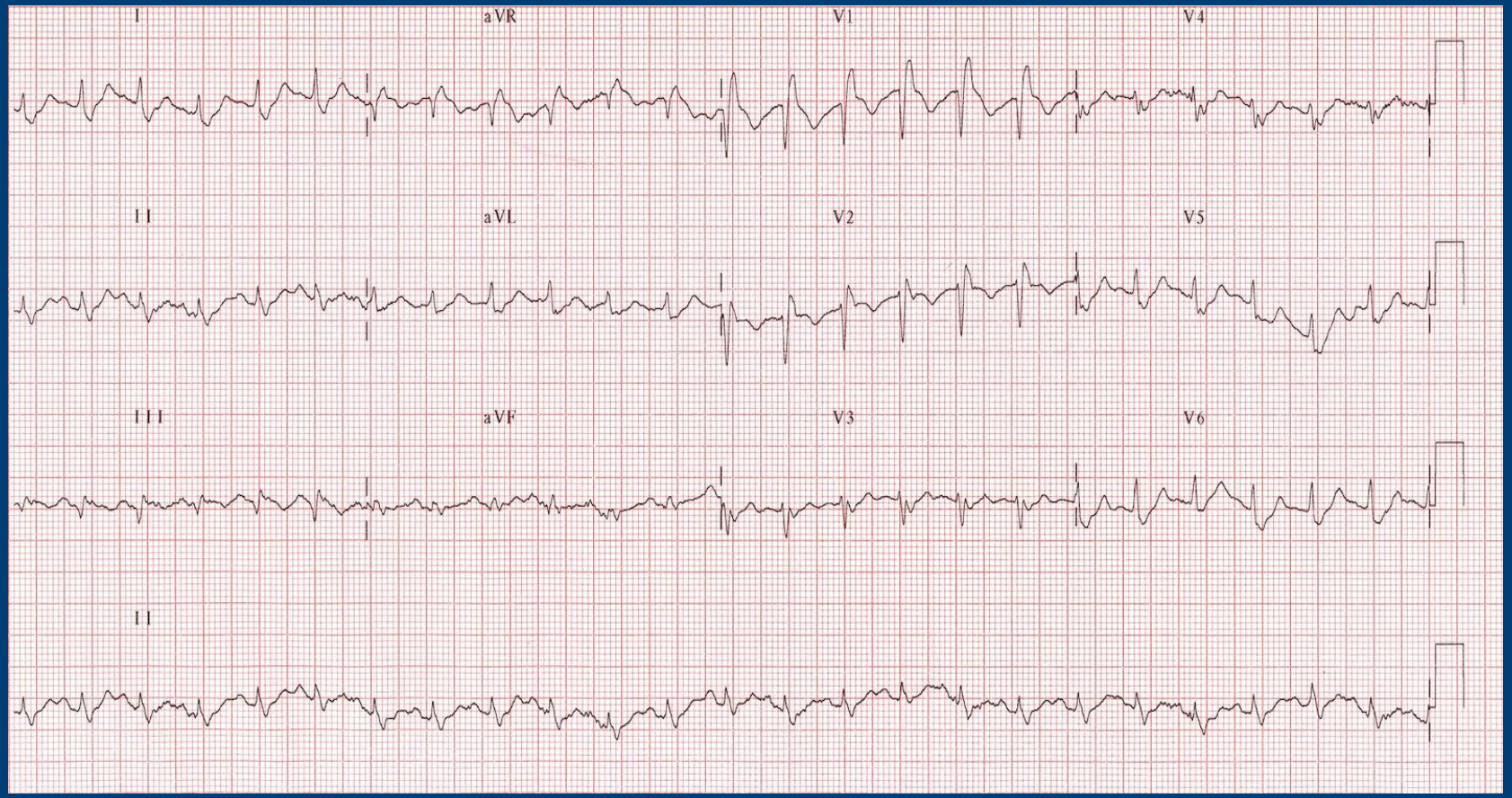
ST depression MI ECG



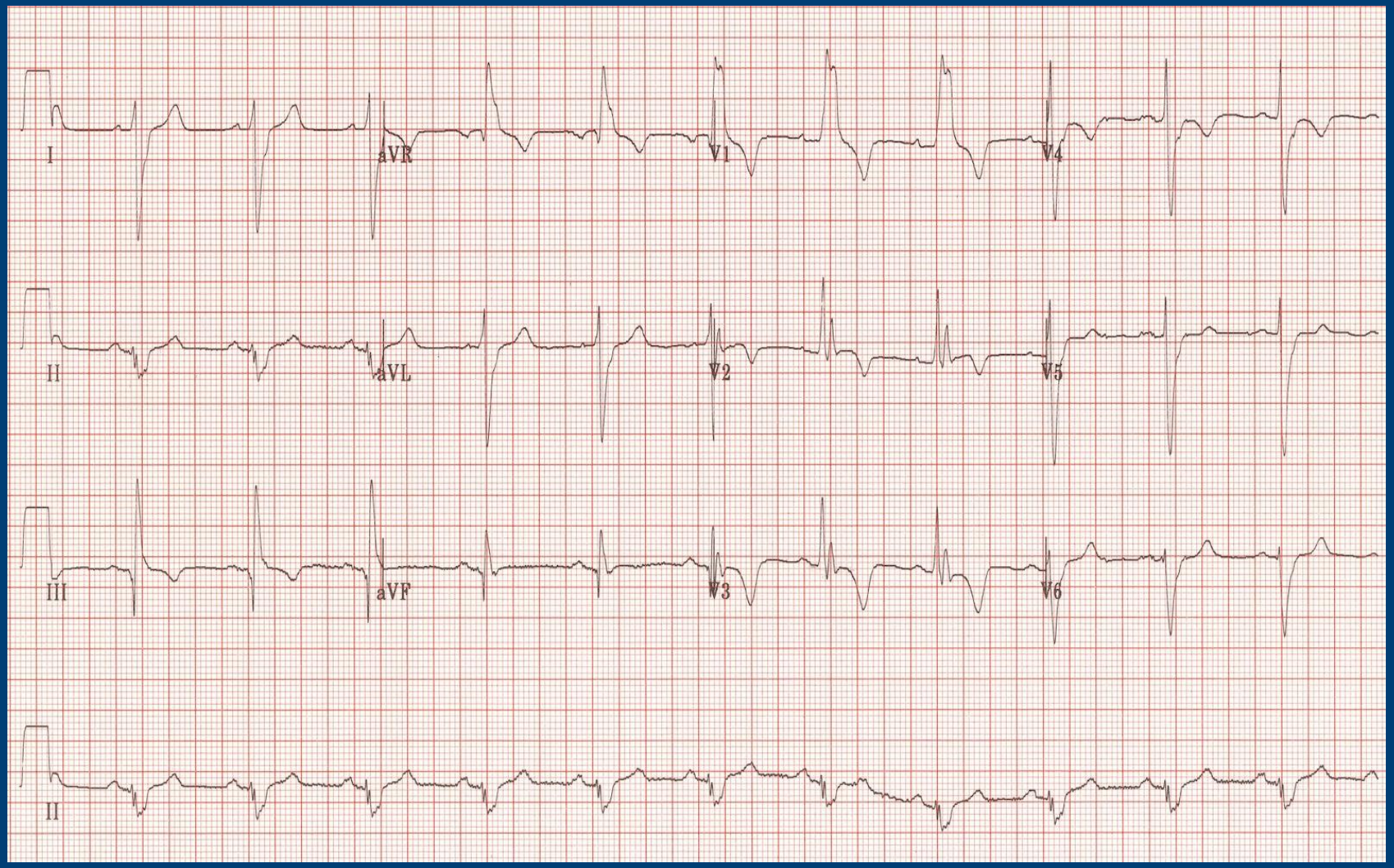
Electrocardiographic abnormalities found in acute pulmonary embolism

- Sinus tachycardia
- Atrial flutter or fibrillation
- S_1, Q_3, T_3 pattern
- Right bundle branch block (incomplete or complete)
- T wave inversion in the right precordial leads
- P pulmonale
- Right axis deviation

PE ECG



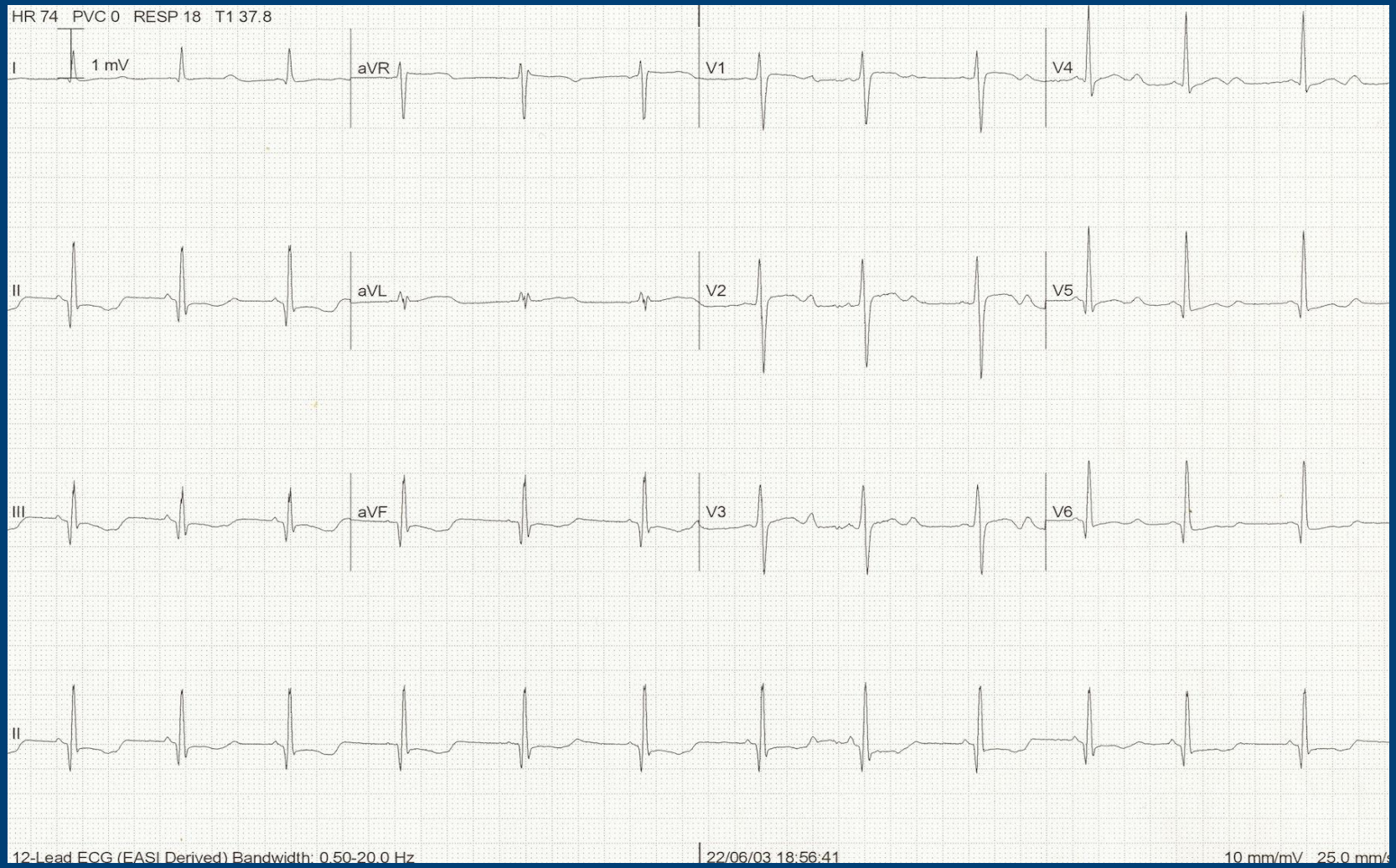
PE ECG



Electrocardiographic features of hypokalaemia

- Broad, flat T waves
- ST depression
- QT interval prolongation
- Ventricular arrhythmias (premature ventricular contractions, torsades de pointes, ventricular tachycardia, ventricular fibrillation)

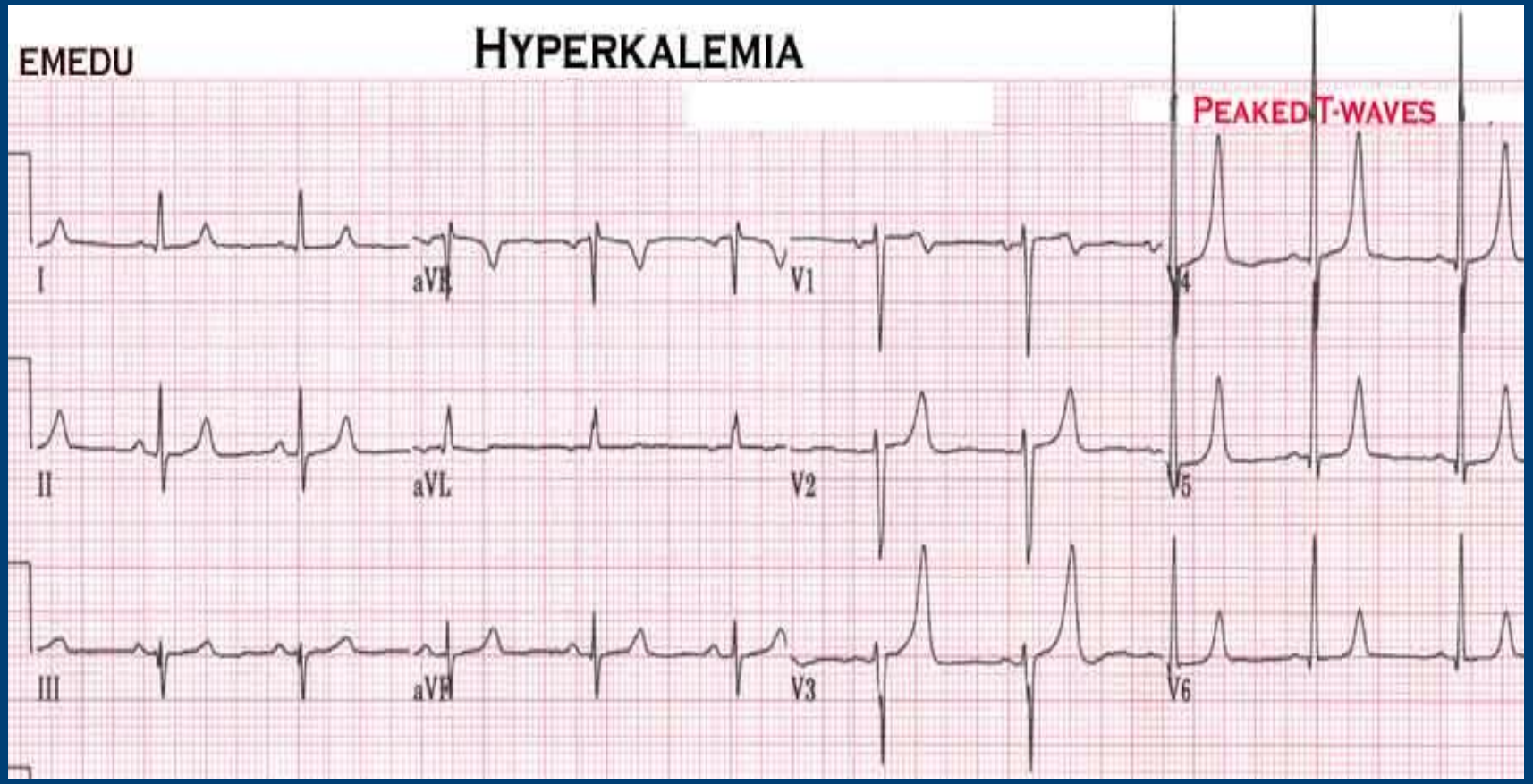
Hypokalemia ECG

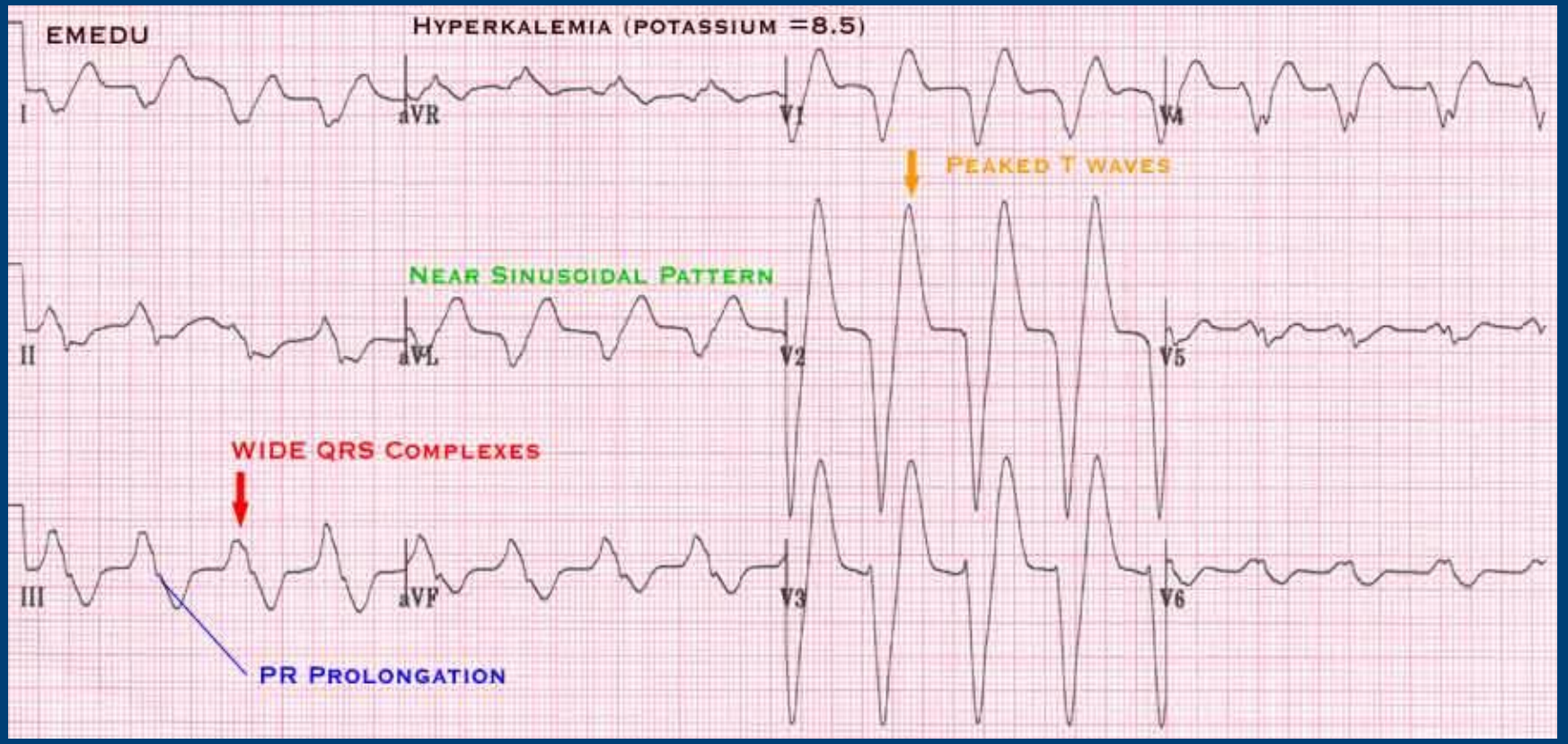


Electrocardiographic features of hyperkalaemia

- K > 5.7 Tall , symmetrical T wave.
- K>7.0 reduce P wave, prolongation of PR Interval.
- K>8.4 dissapperance of P wave
- K 9-11 widening of QRS
- K >12 VF

Hyperkalemia ECG




EMEDU
HYPERKALEMIA (POTASSIUM =8.5)
I
aVR
V1
V4
PEAKED T WAVES
NEAR SINUSOIDAL PATTERN
II
aVL
V2
V5
WIDE QRS COMPLEXES
III
aVF
V3
V6
PR PROLONGATION

Digoxin Toxicity

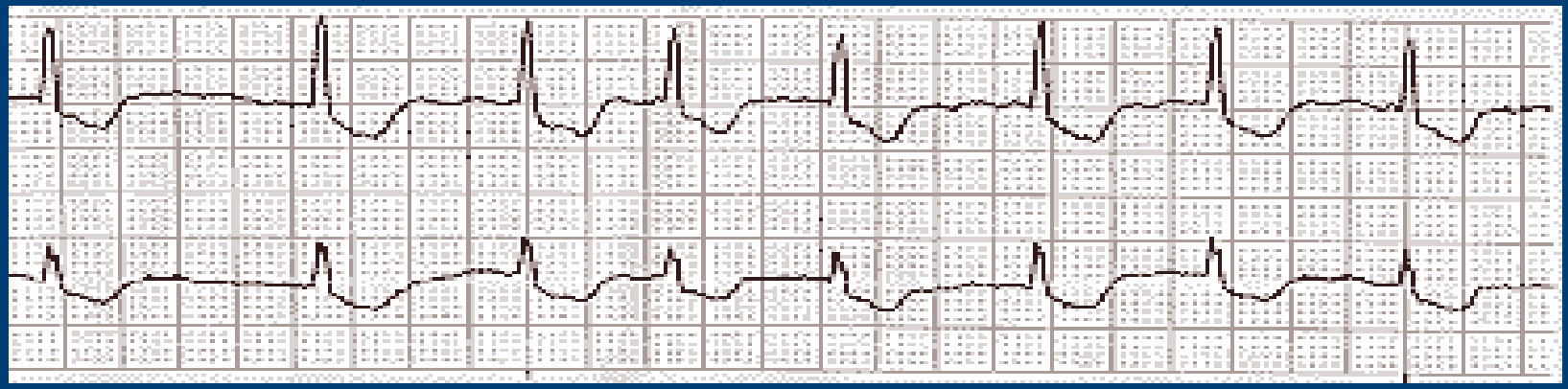
Clinical features

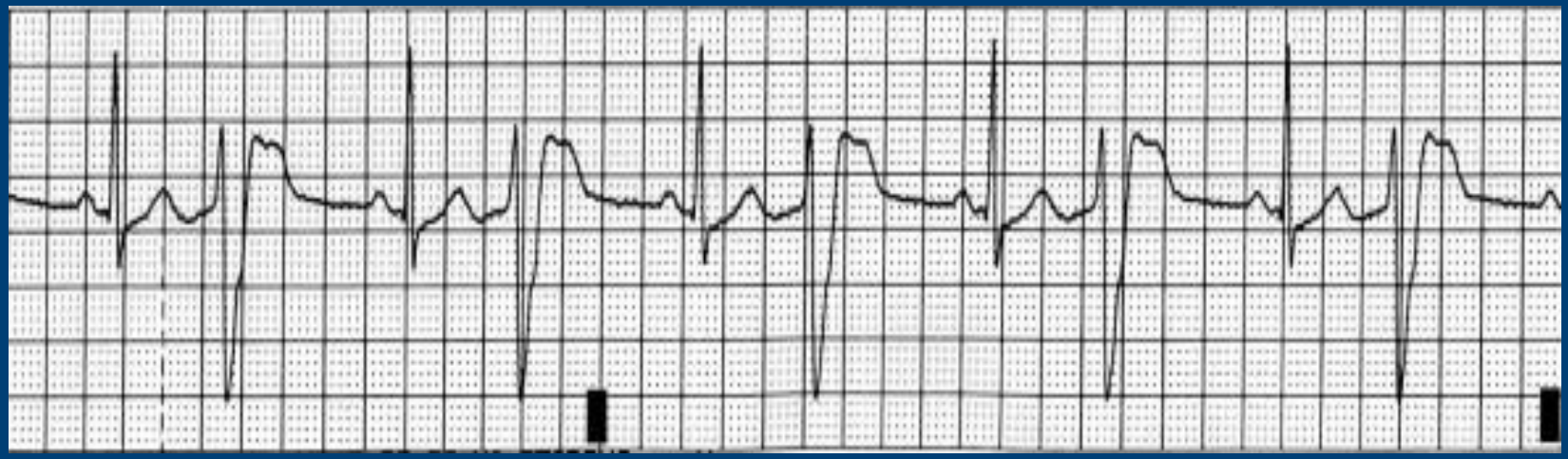
- GIT: Nausea, vomiting, anorexia, diarrhoea
- Visual: Blurred vision, yellow/green discolouration, haloes
- CVS: Palpitations, syncope, dyspnoea
- CNS: Confusion, dizziness, delirium, fatigue

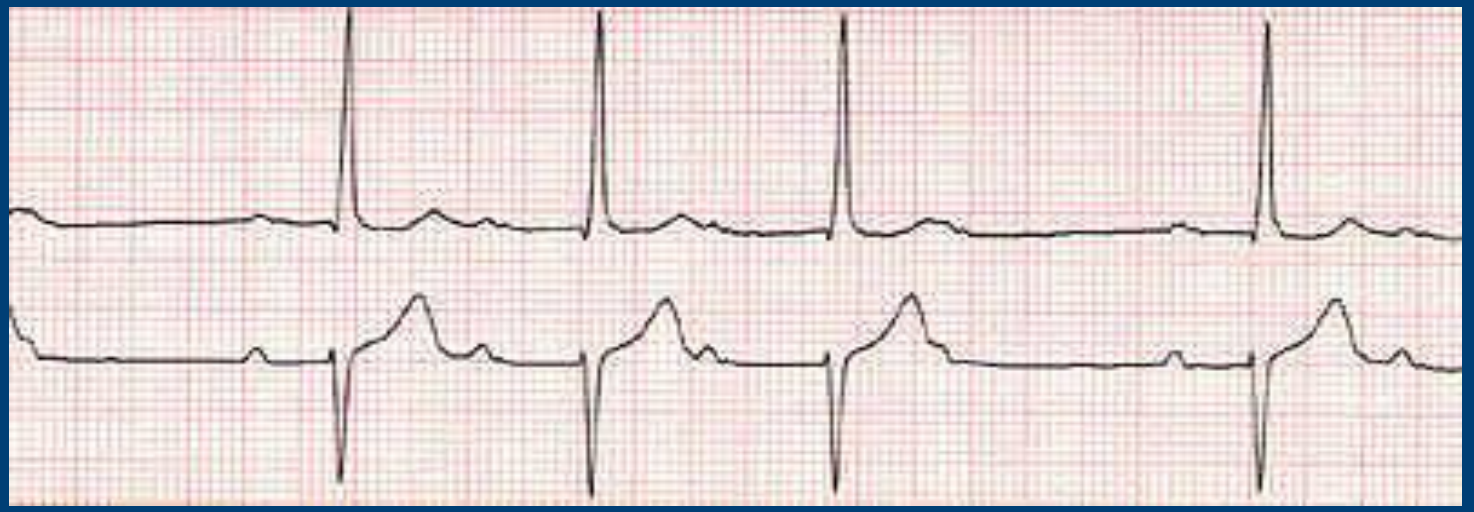
ECG criteria

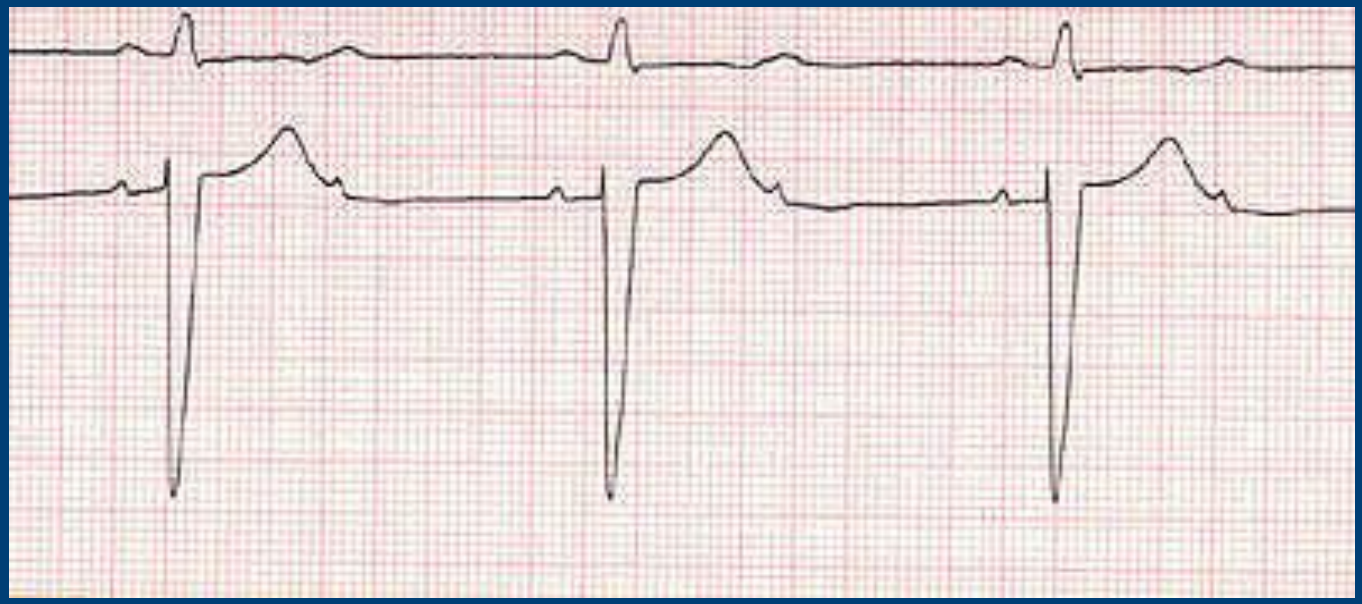
- Downsloping ST depression with a characteristic "sagging" appearance (*see below*).
- Flattened, inverted, or biphasic T waves.
- Shortened QT interval.
- Frequent PVCs (the most common abnormality), including ventricular bigeminy and trigeminy
- Sinus bradycardia or slow AF
- Any type of AV block (1st degree, 2nd degree & 3rd degree)
- *Regularised AF* = AF with complete heart block and a junctional or ventricular escape rhythm
- Ventricular tachycardia, including polymorphic and bidirectional VT

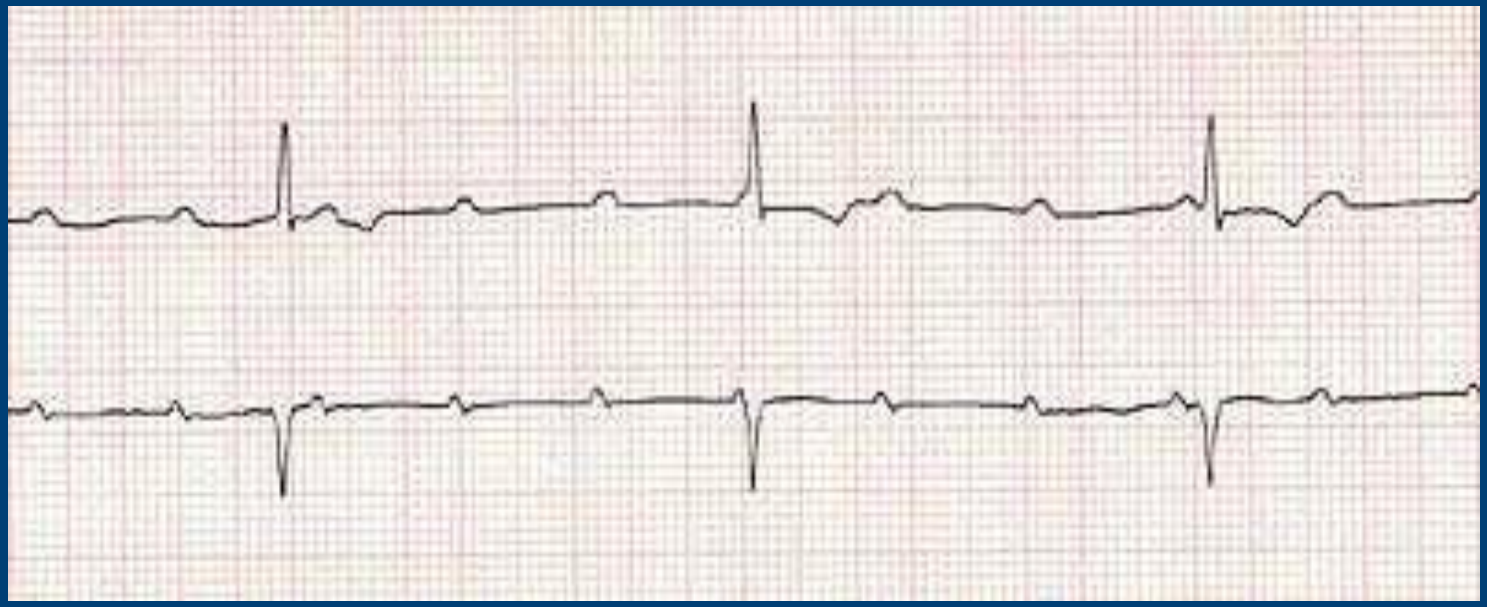
Digoxin toxicity ECG

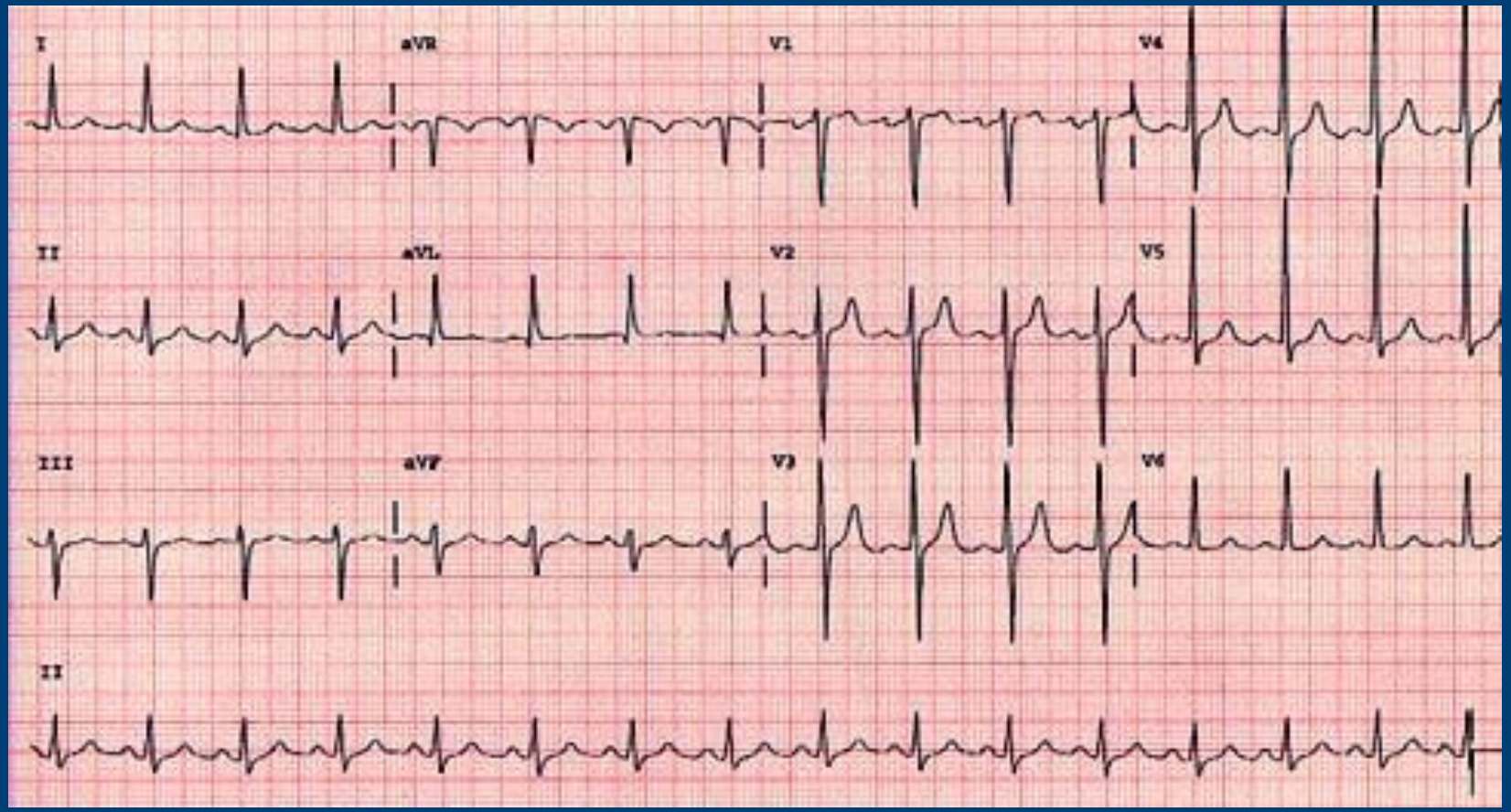


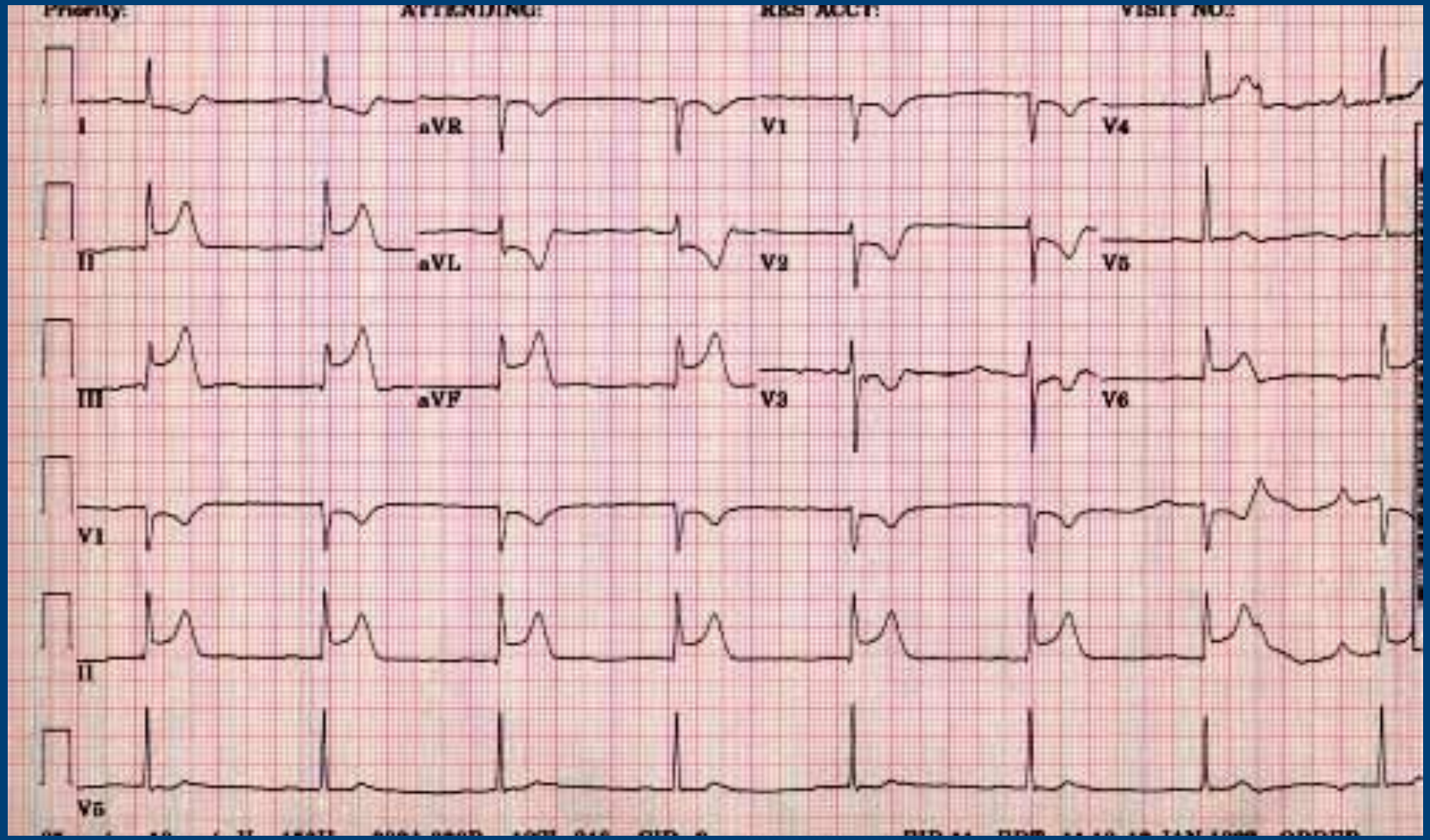


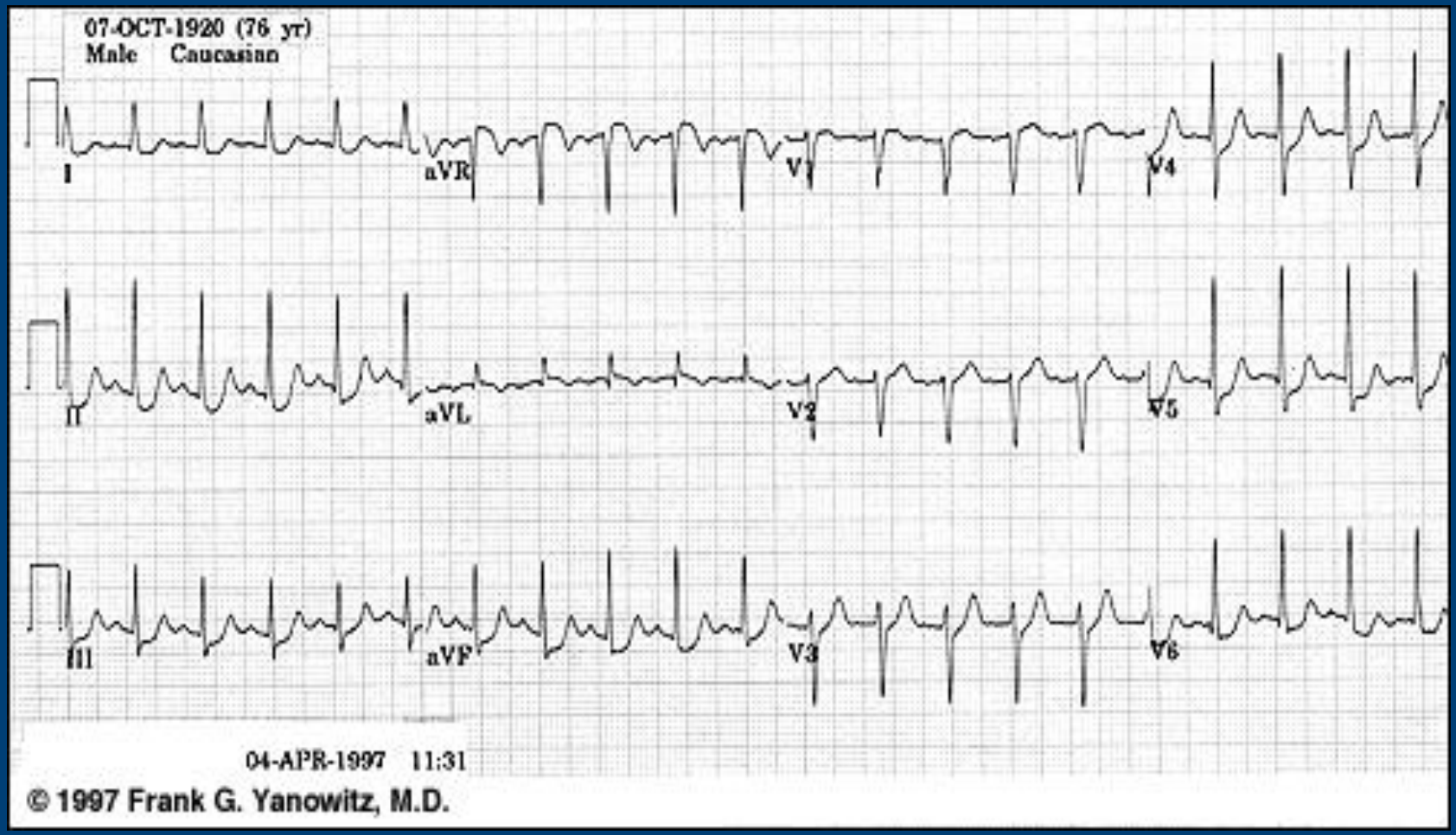


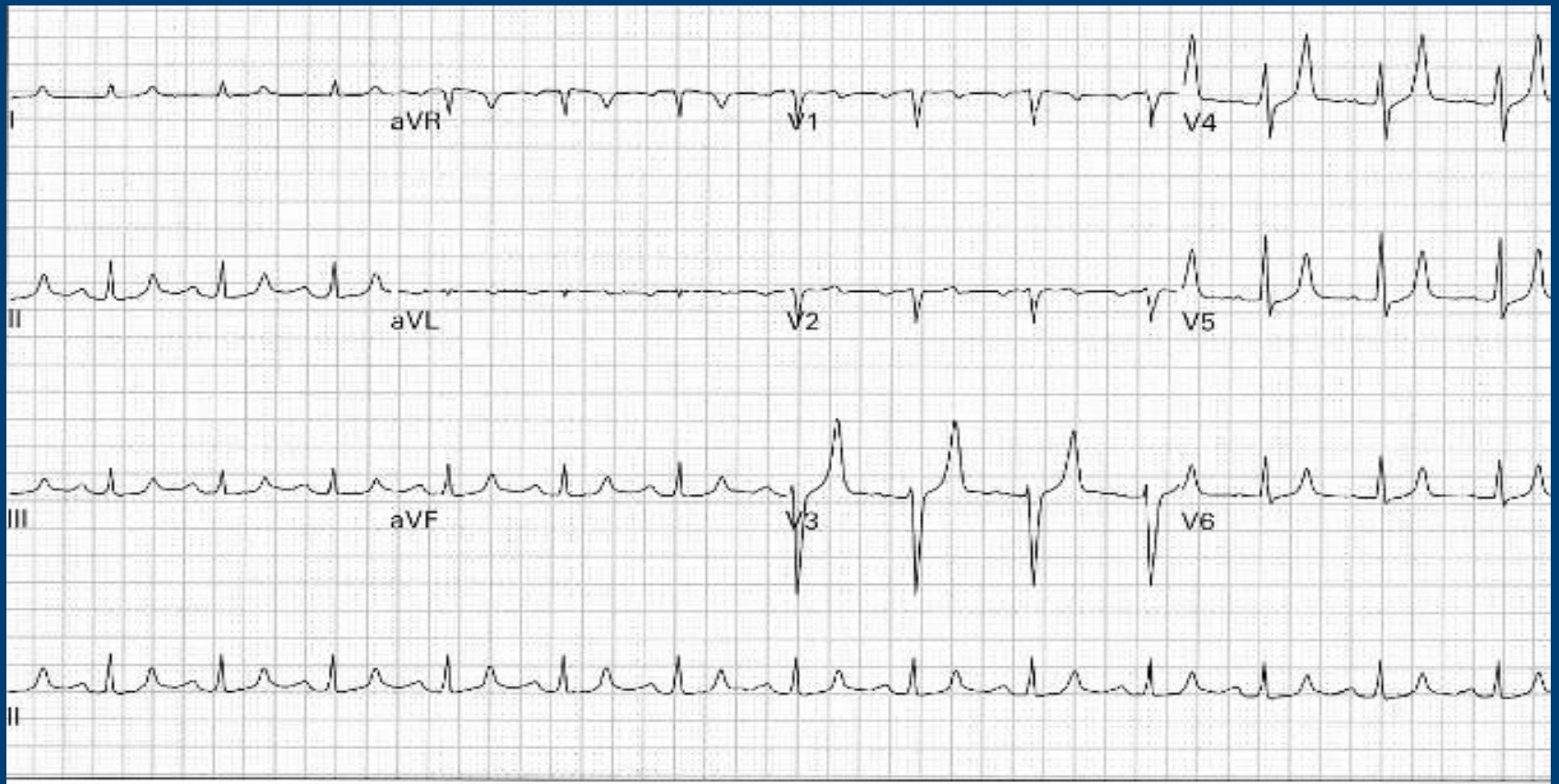




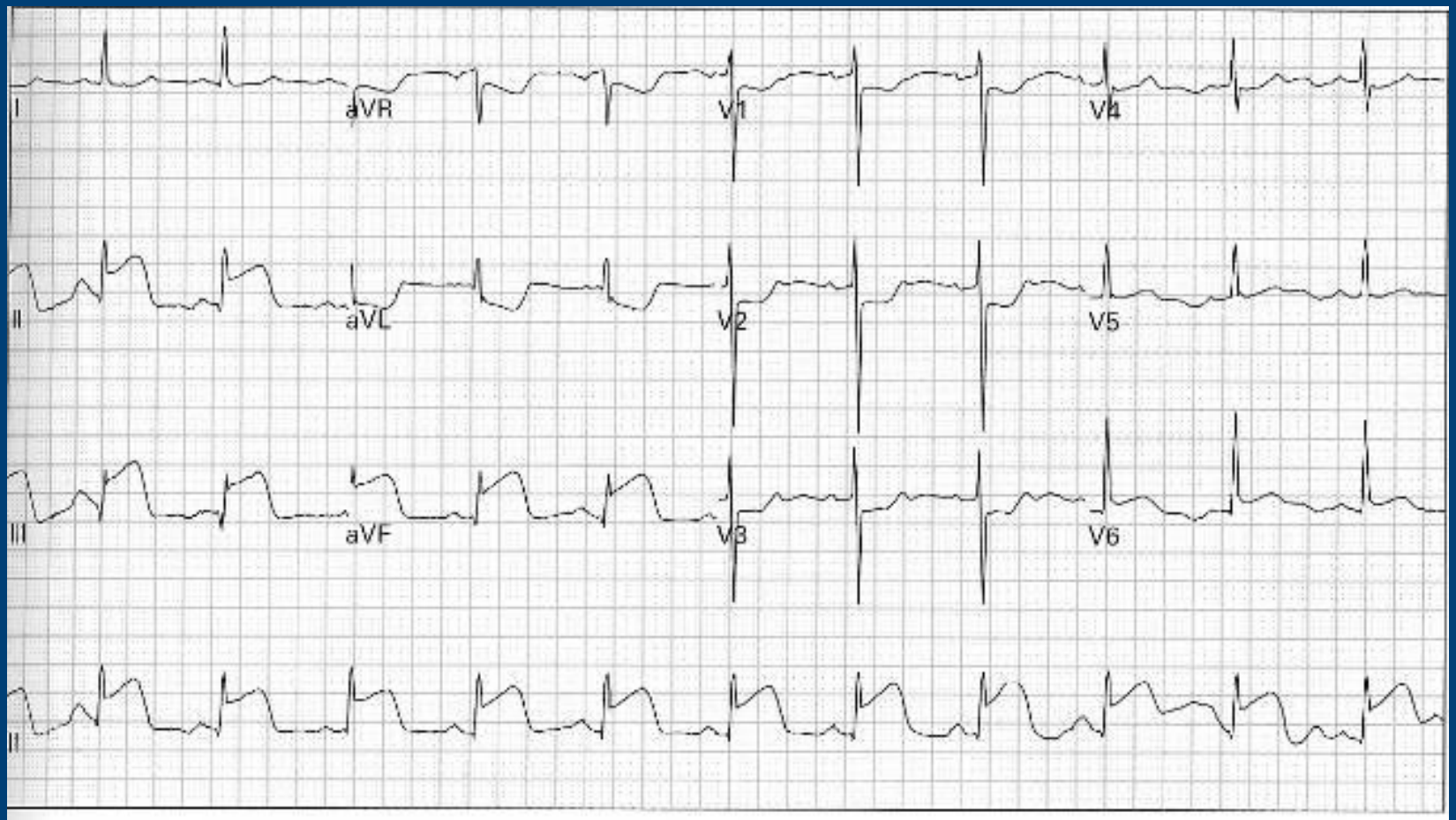

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II


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

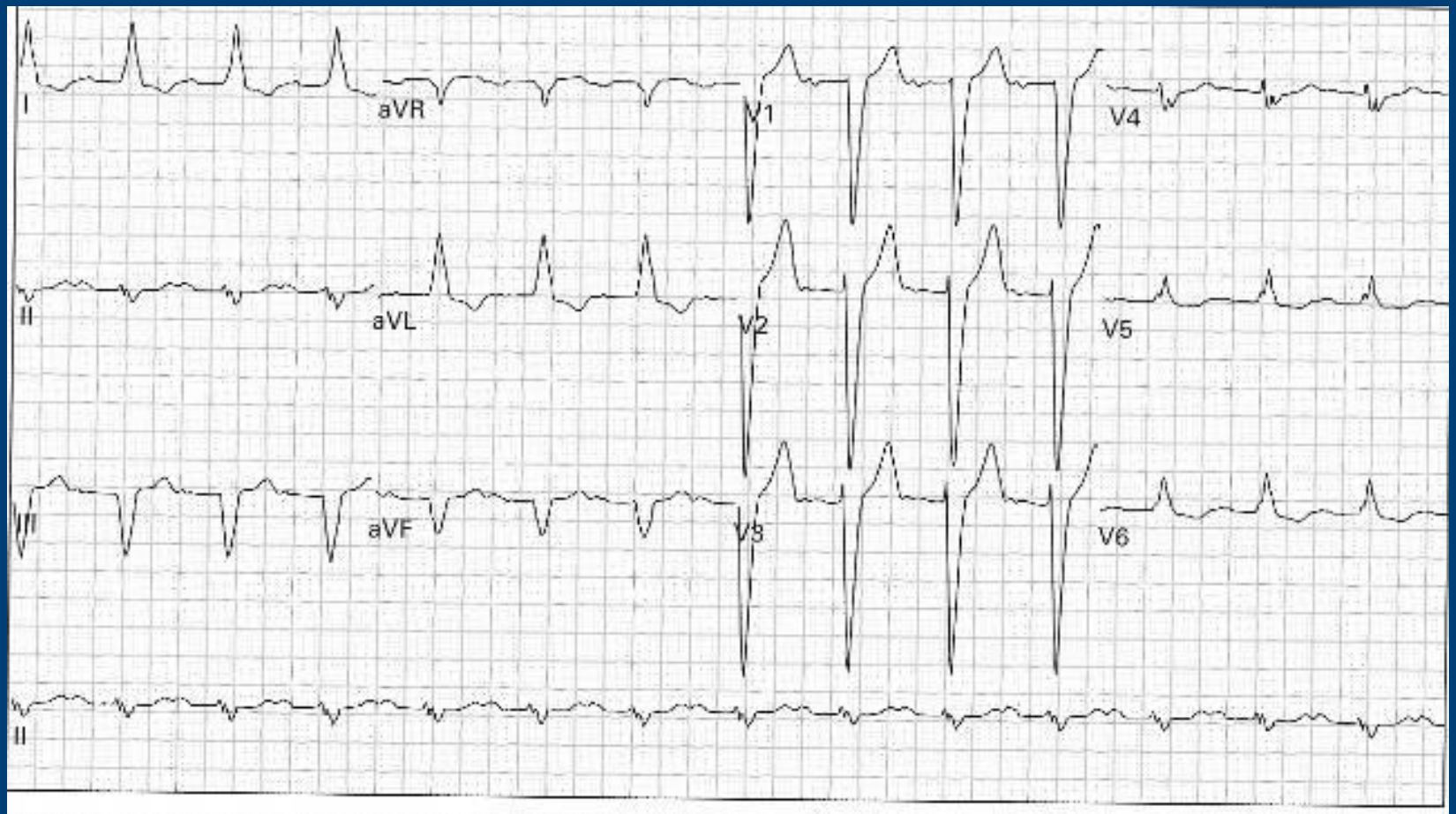

07-OCT-1920 (76 yr)
Male Caucasian
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
04-APR-1997 11:31
© 1997 Frank G. Yanowitz, M.D.



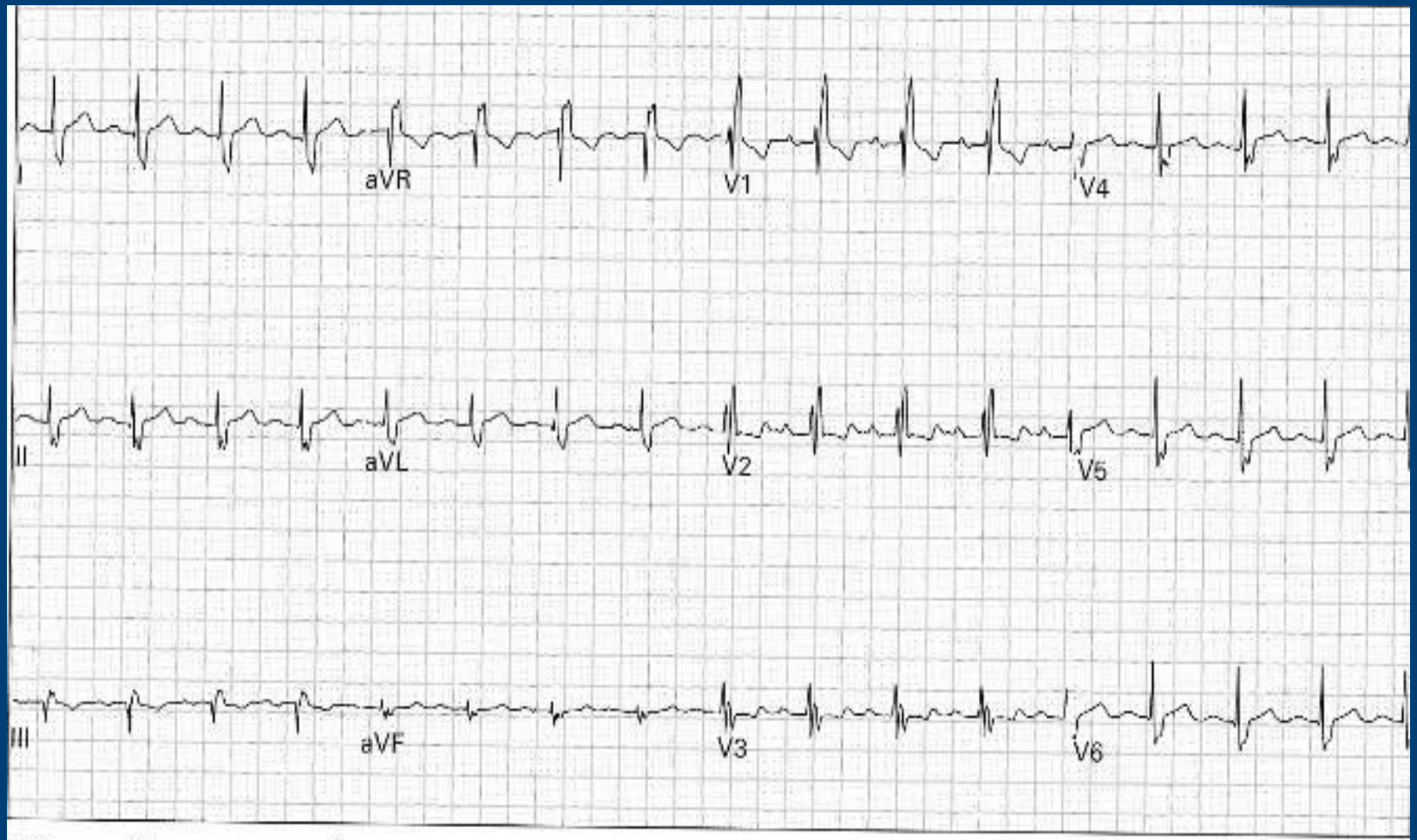
52. 62 year old man with renal failure complains of progressive dyspnea and orthopnea after missing his last two hemodialysis sessions



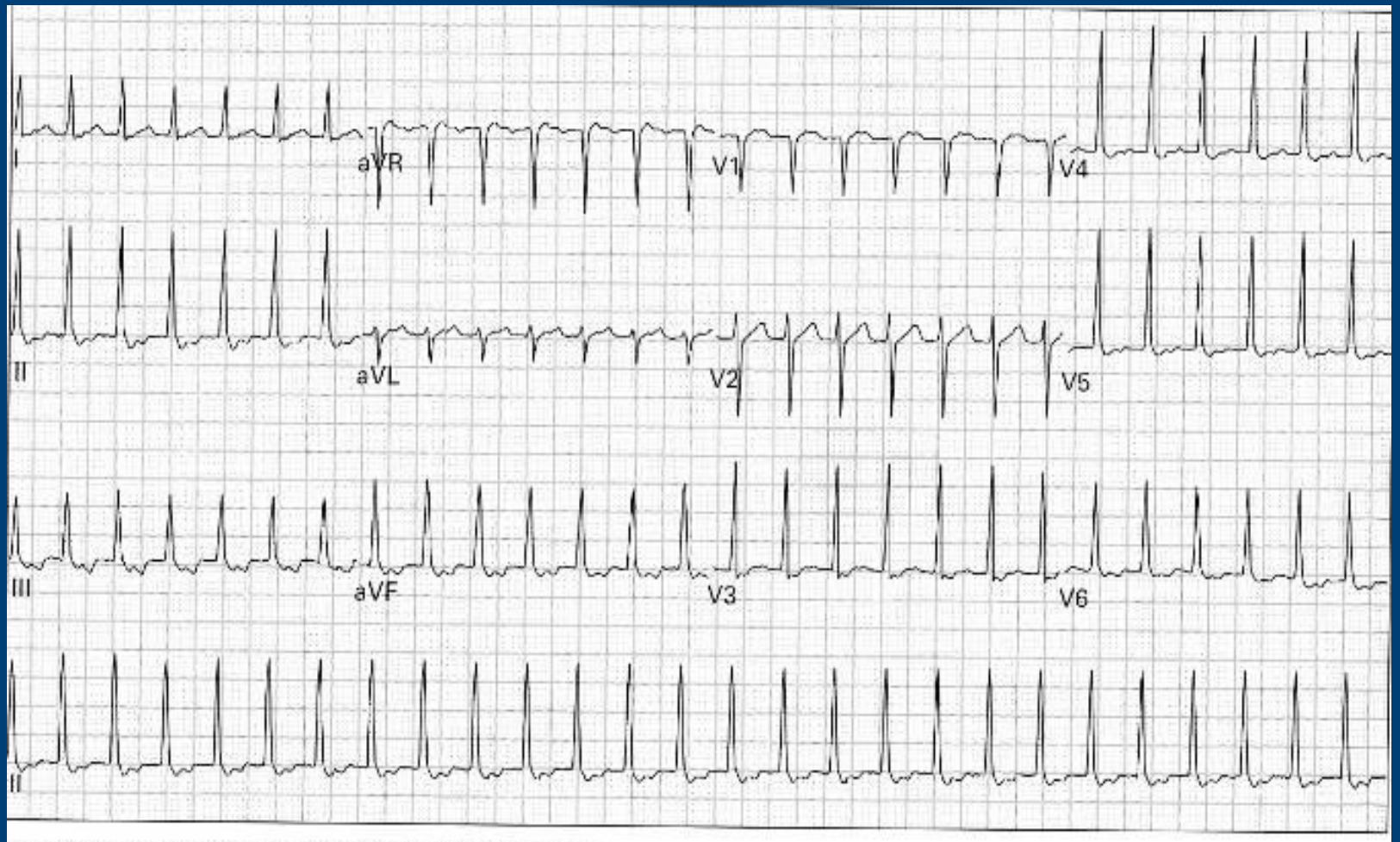
37. 38 year old man with chest pain, nausea, and diaphoresis



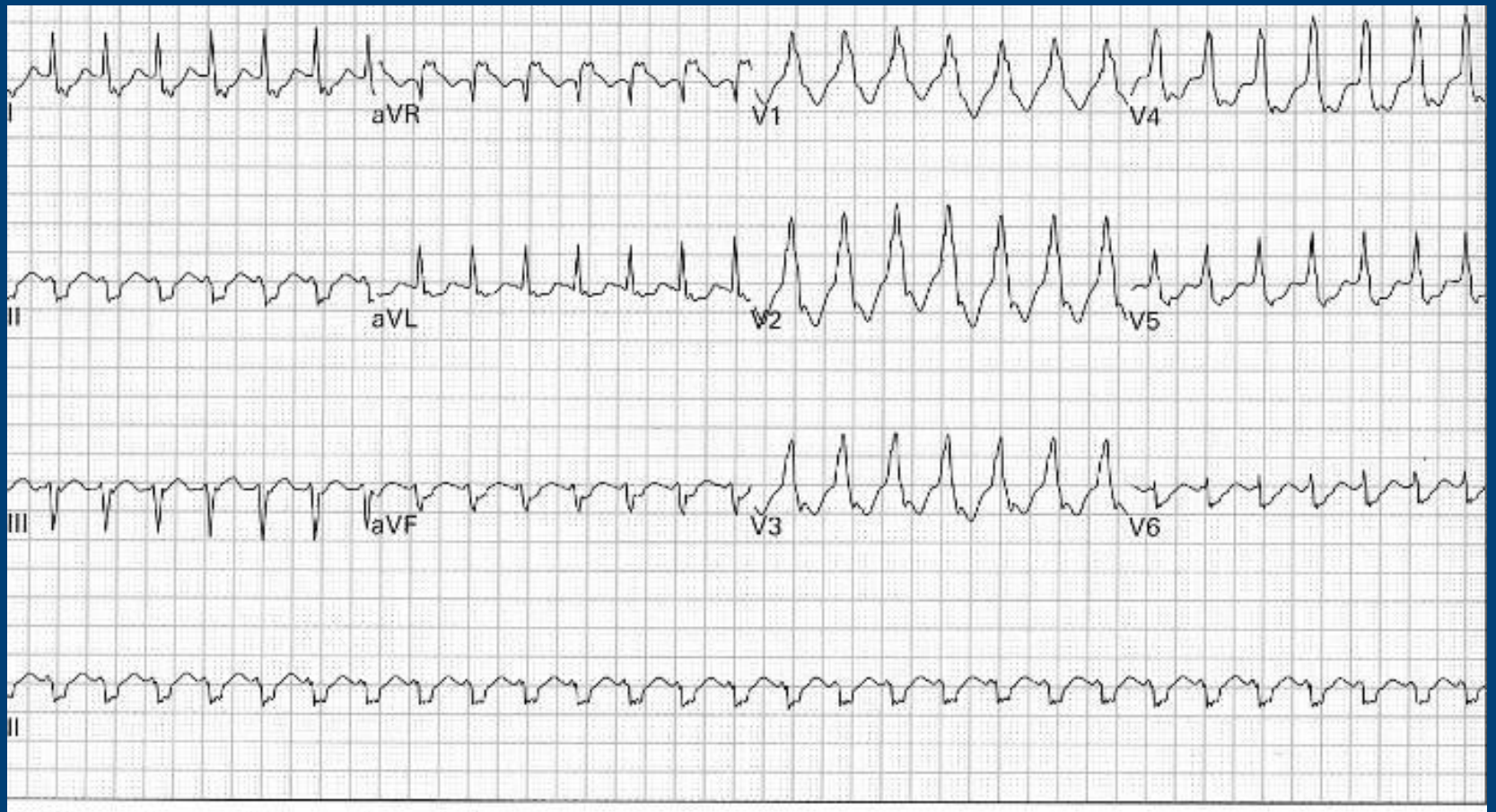
8. 82 year old man recently increased his dose of a beta-receptor blocking medication; he now reports exertional lightheadedness



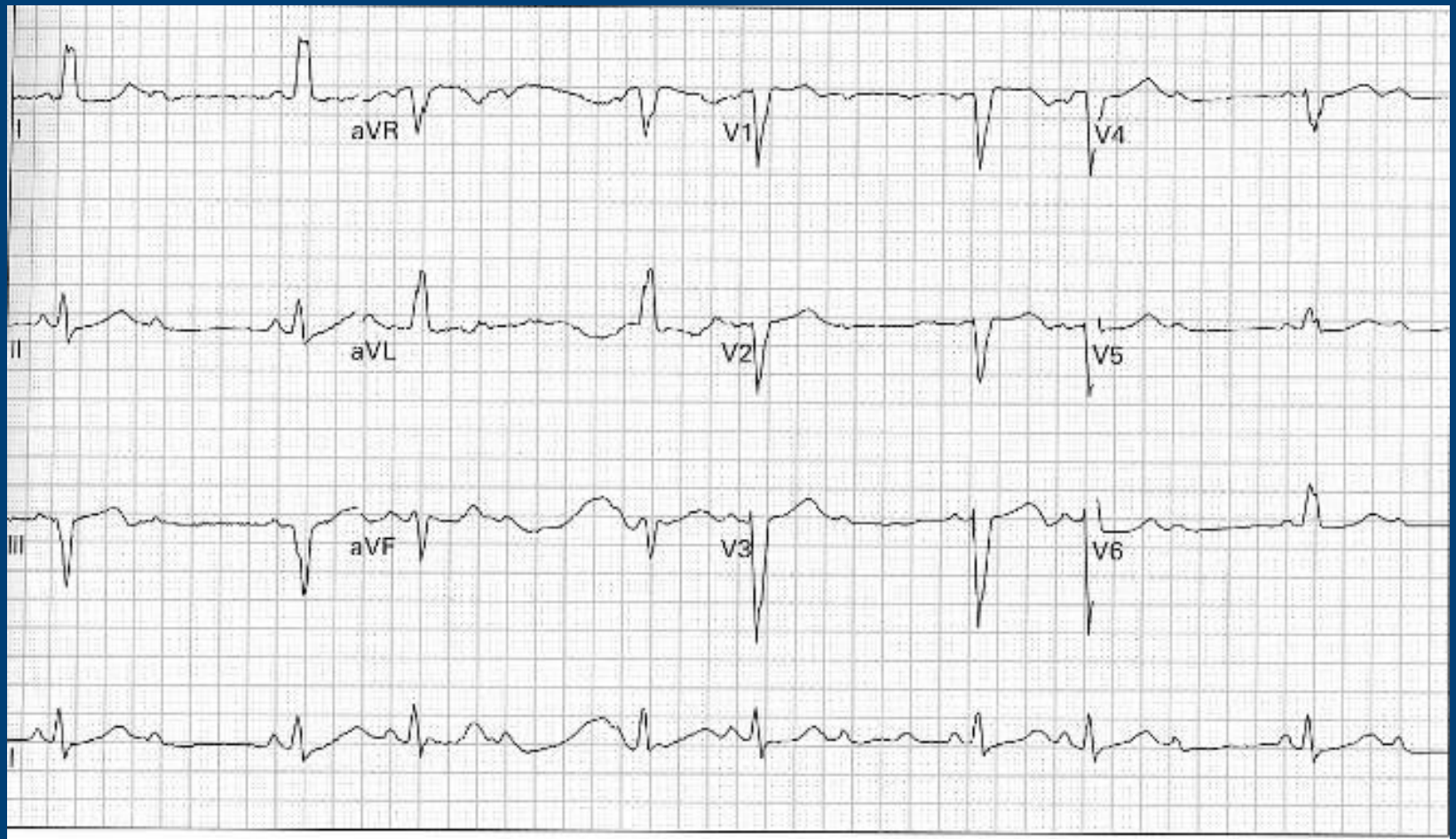
7. 43 year old man, asymptomatic



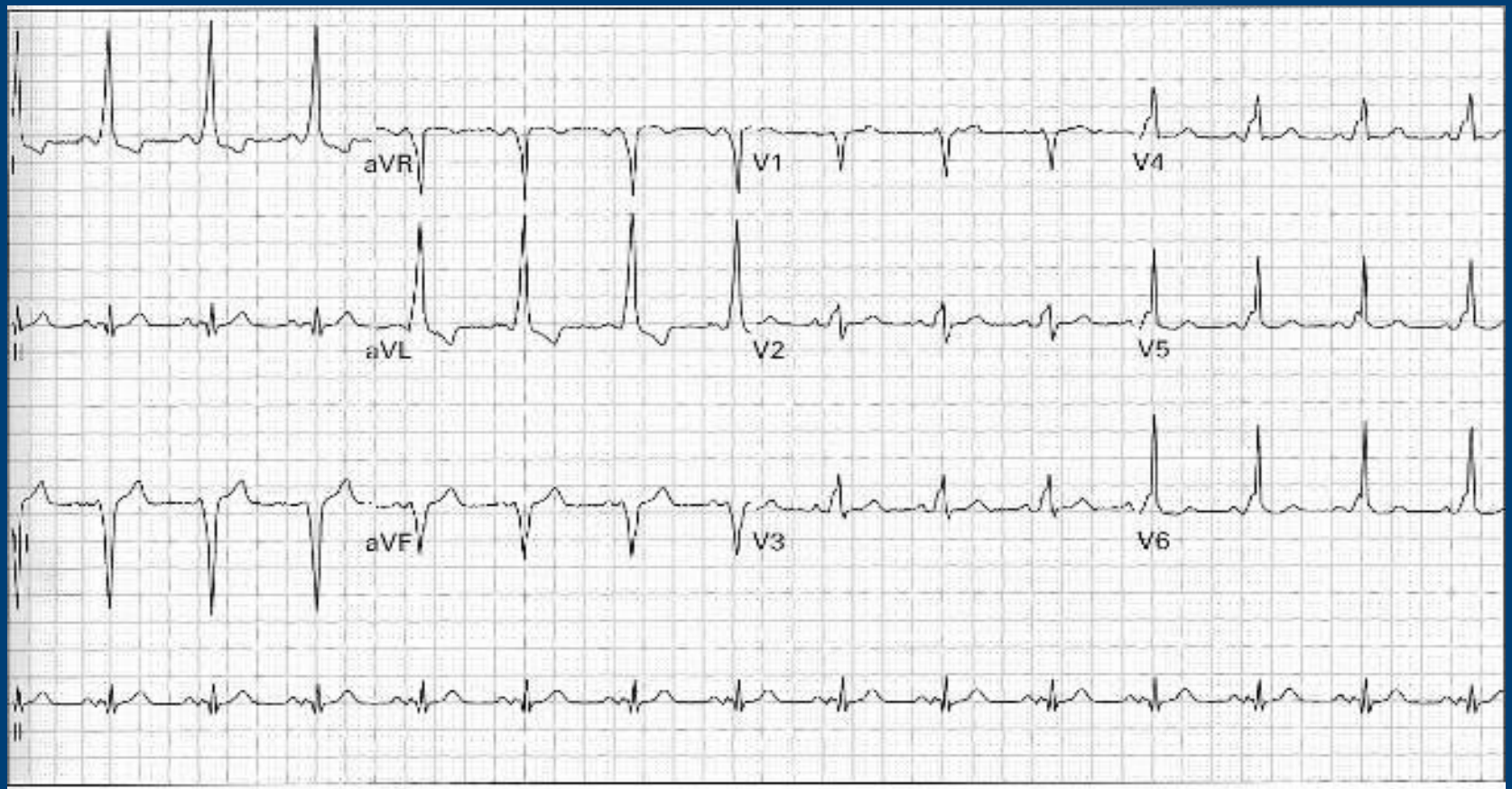
27. 40 year old woman with palpitations and lightheadedness



9. 74 year old man with chest pain and palpitations



2. 85 year old woman presents after a syncopal episode, still reports lightheadedness



14. 44 year old woman with intermittent episodes of palpitations

- الدكتور بشارة بقاعين
- استشاري الامراض الباطنية
- مستشفى البشير