ECG (EKG)

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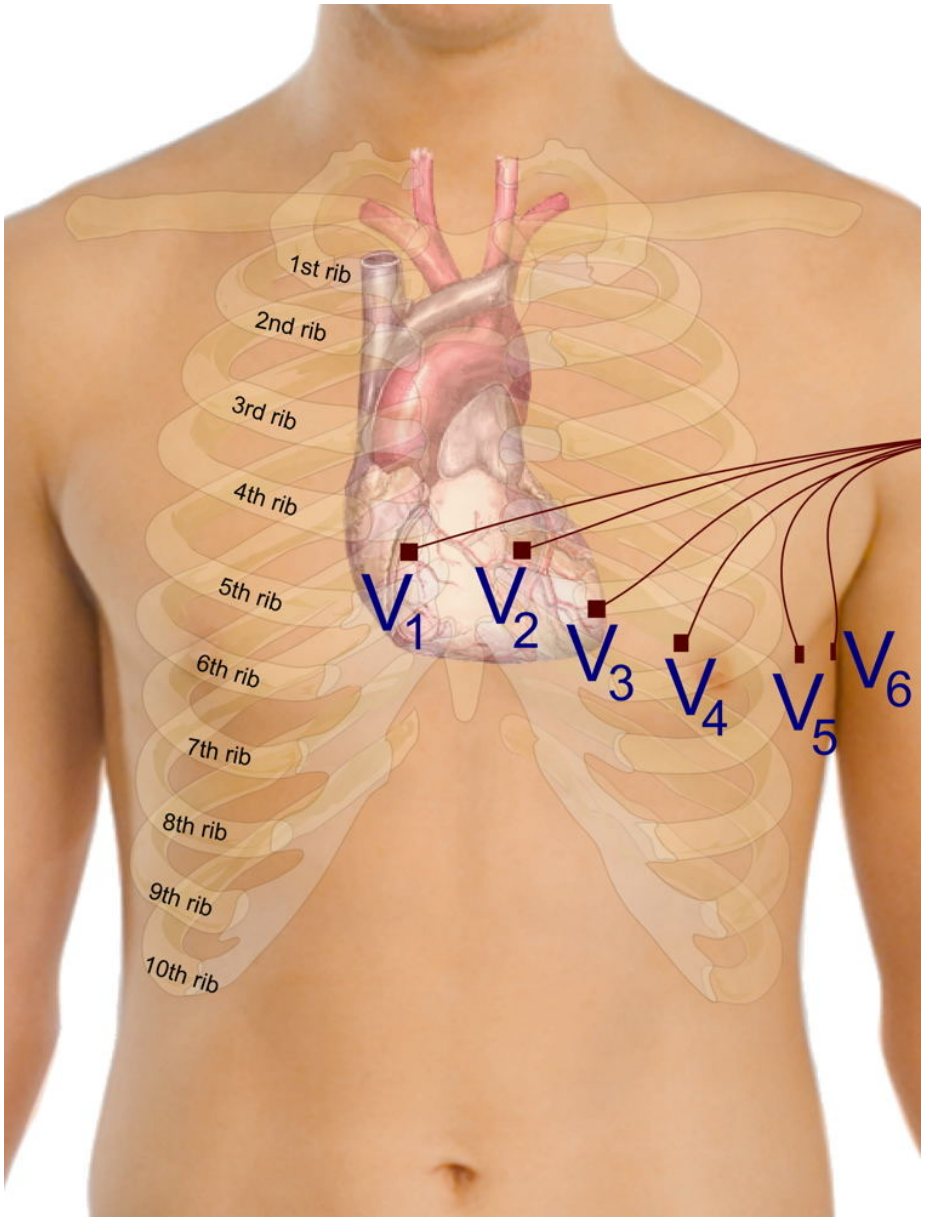
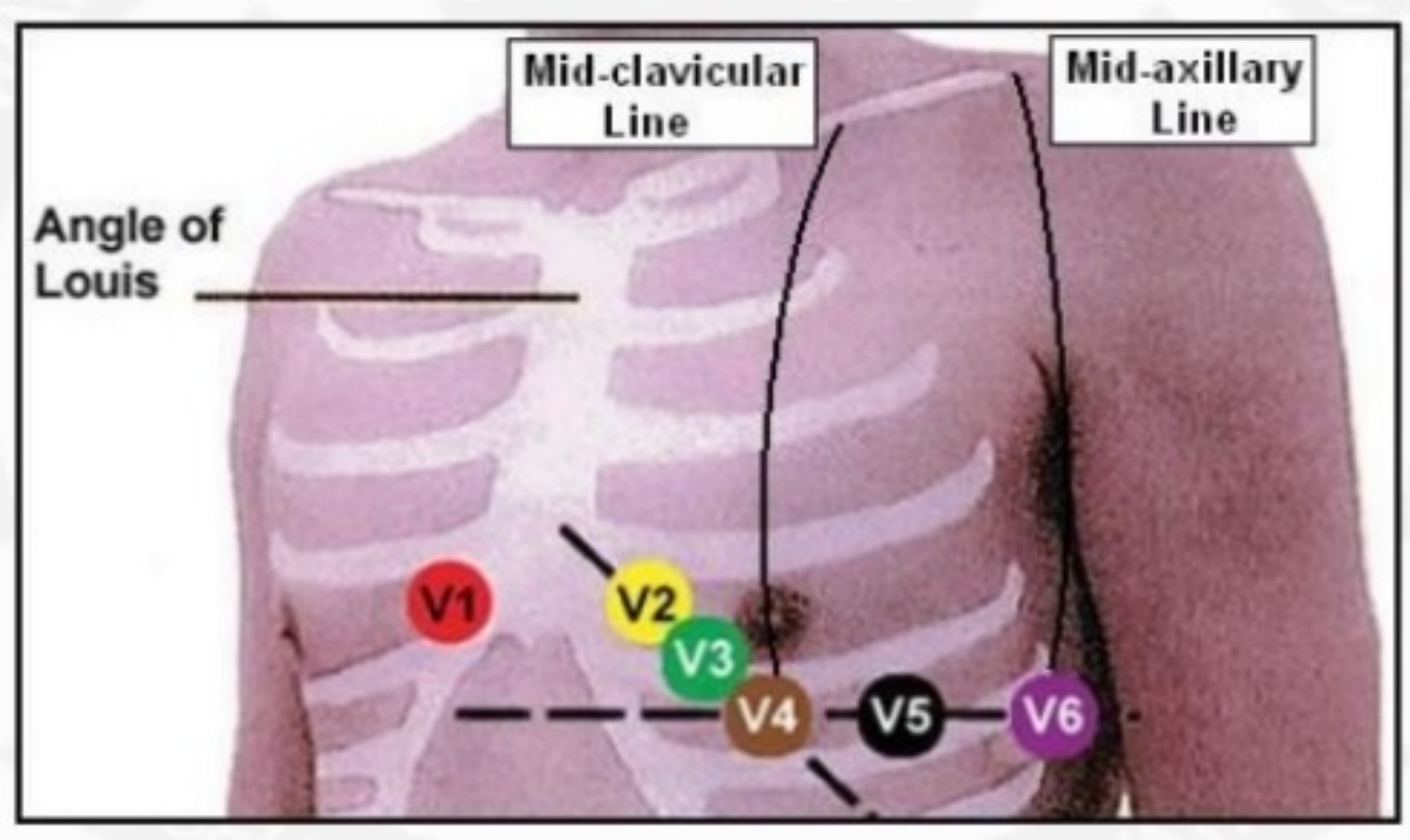
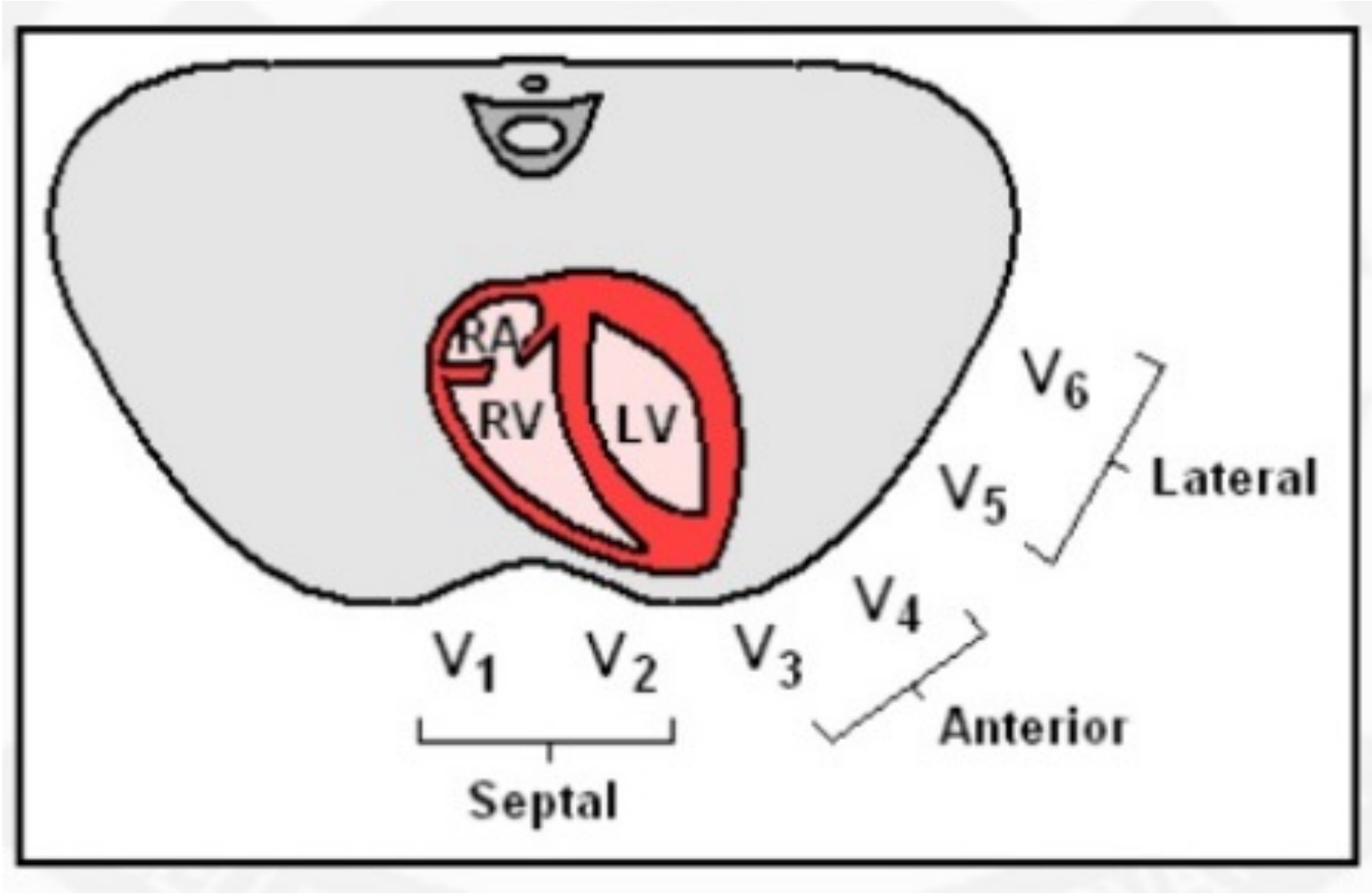
ECG (EKG) Interpretation

The 12-lead ECG misleadingly only has 10 electrodes.
The leads can be thought of as taking a picture of the heart's electrical activity from 12 different positions using information picked up by the 10 electrodes. These comprise 4 limb electrodes and 6 chest electrodes.



Electrode positions on an ECG (EKG).


Mid-clavicular Line
Mid-axillary Line
Angle of Louis
V1
V2
V3
V4
V5
V6


RA
RV
LV
V6
V5
Lateral
V4
V1
V2
V3
Anterior
Septal

I	aVR	V_1	V_4
II	aVL	V_2	V_5
III	aVF	V_3	V_6

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
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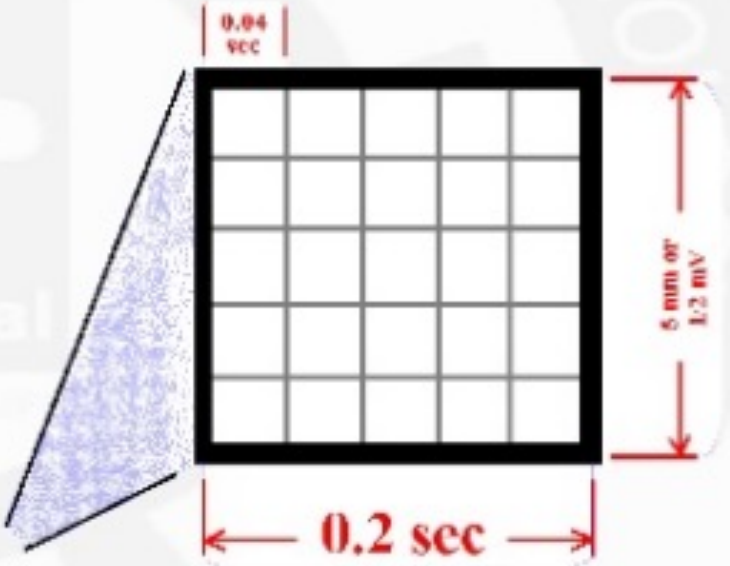
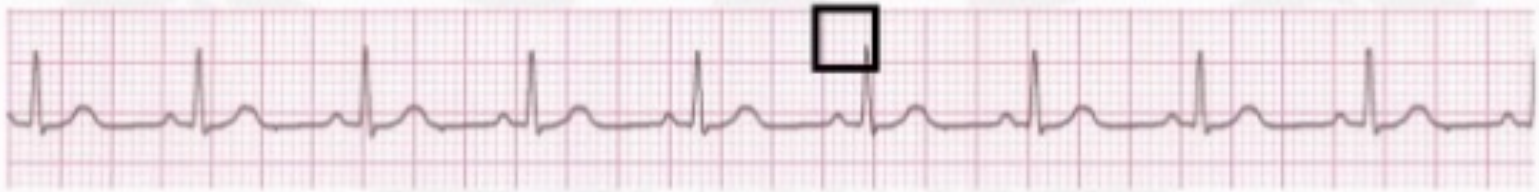
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

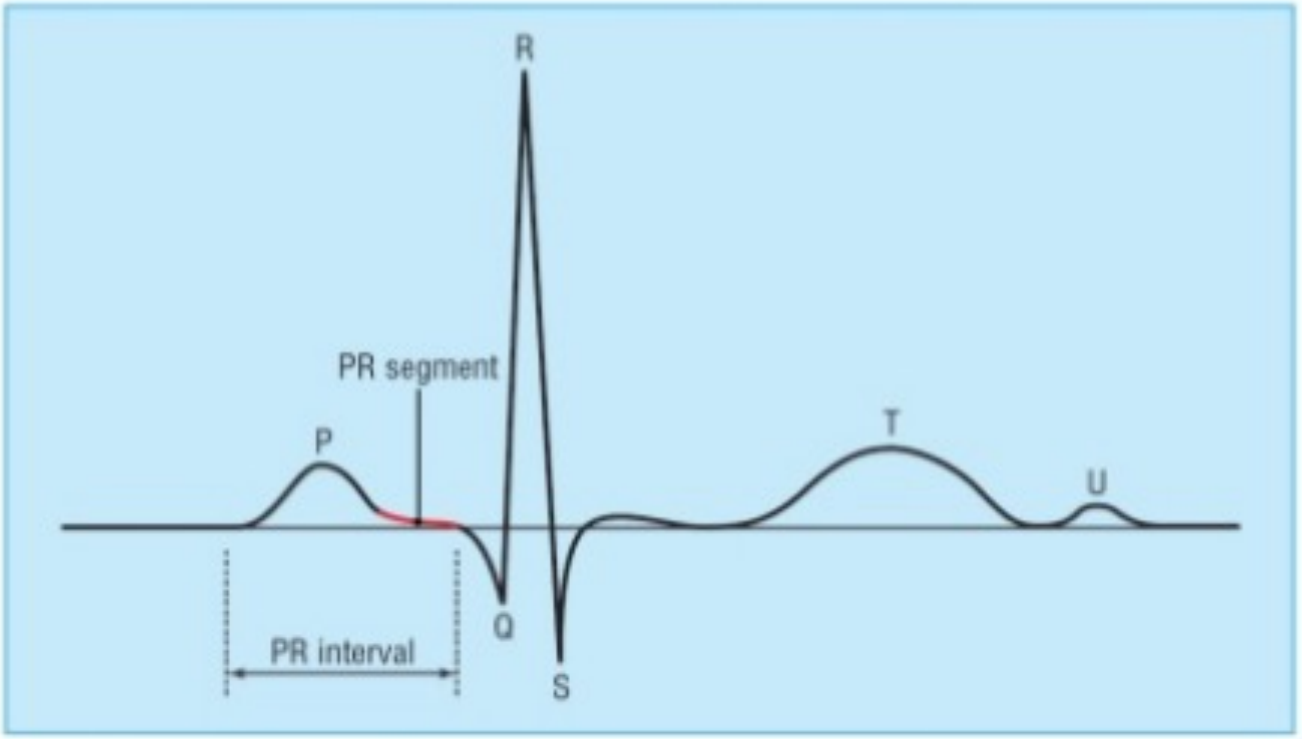
The ECG Paper

- Horizontally
  - One small box - 0.04 s
  - One large box - 0.20 s
- Vertically
  - One large box - 0.5 mV





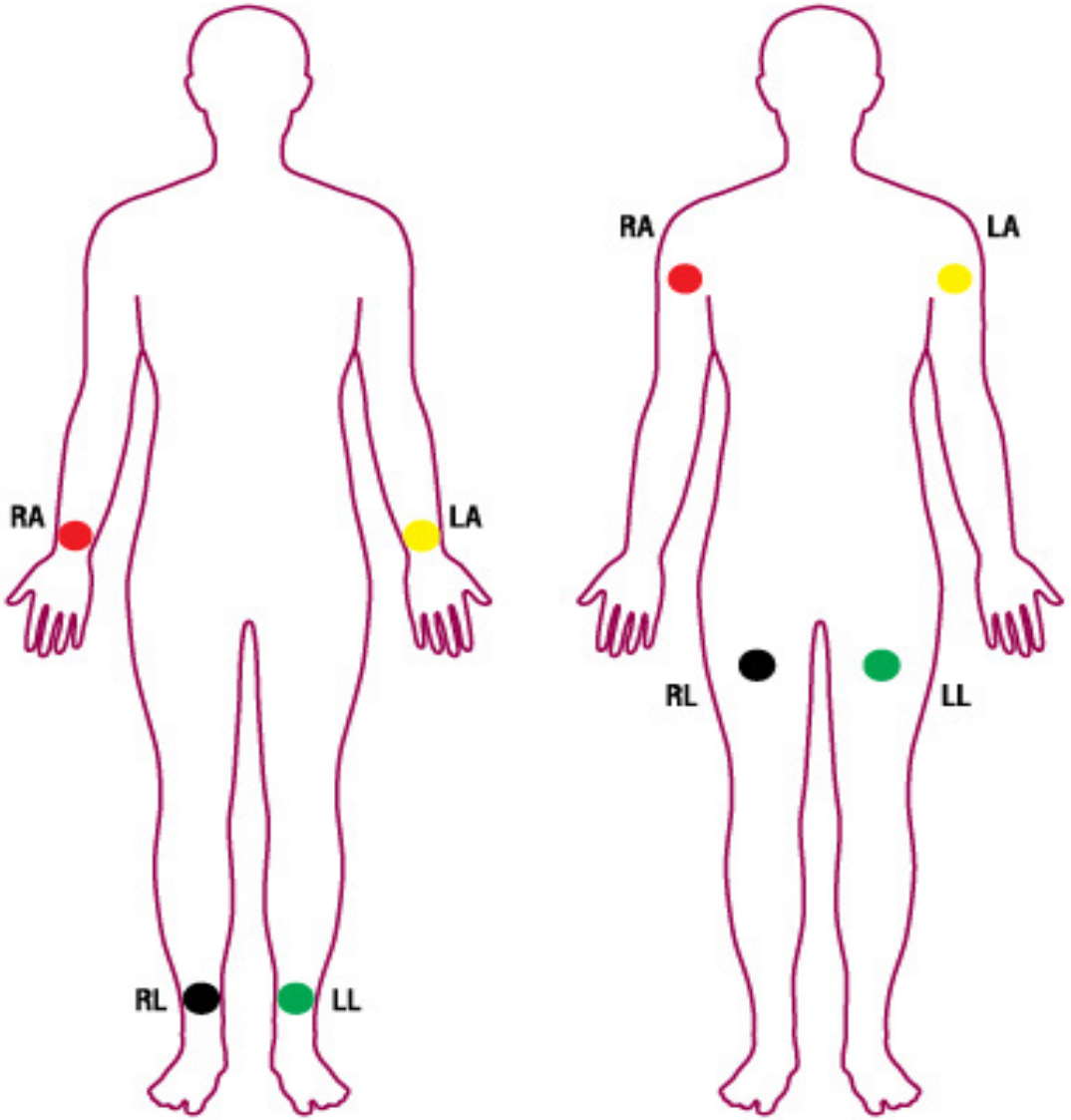
RULE 1



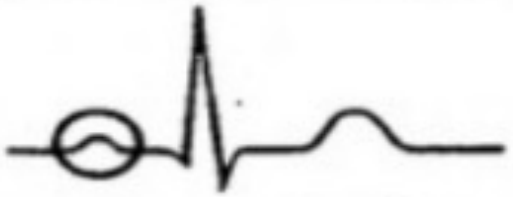
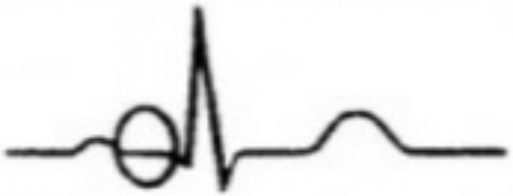
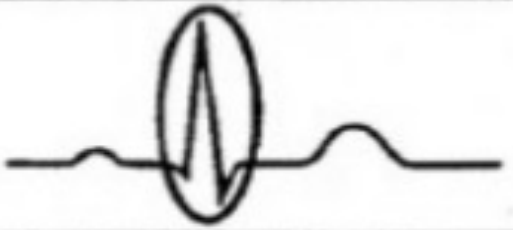
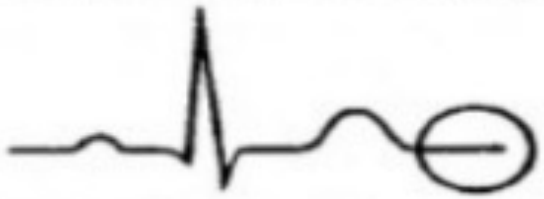
Normal duration of PR interval is 0.12-0.20 s (three to five small squares)

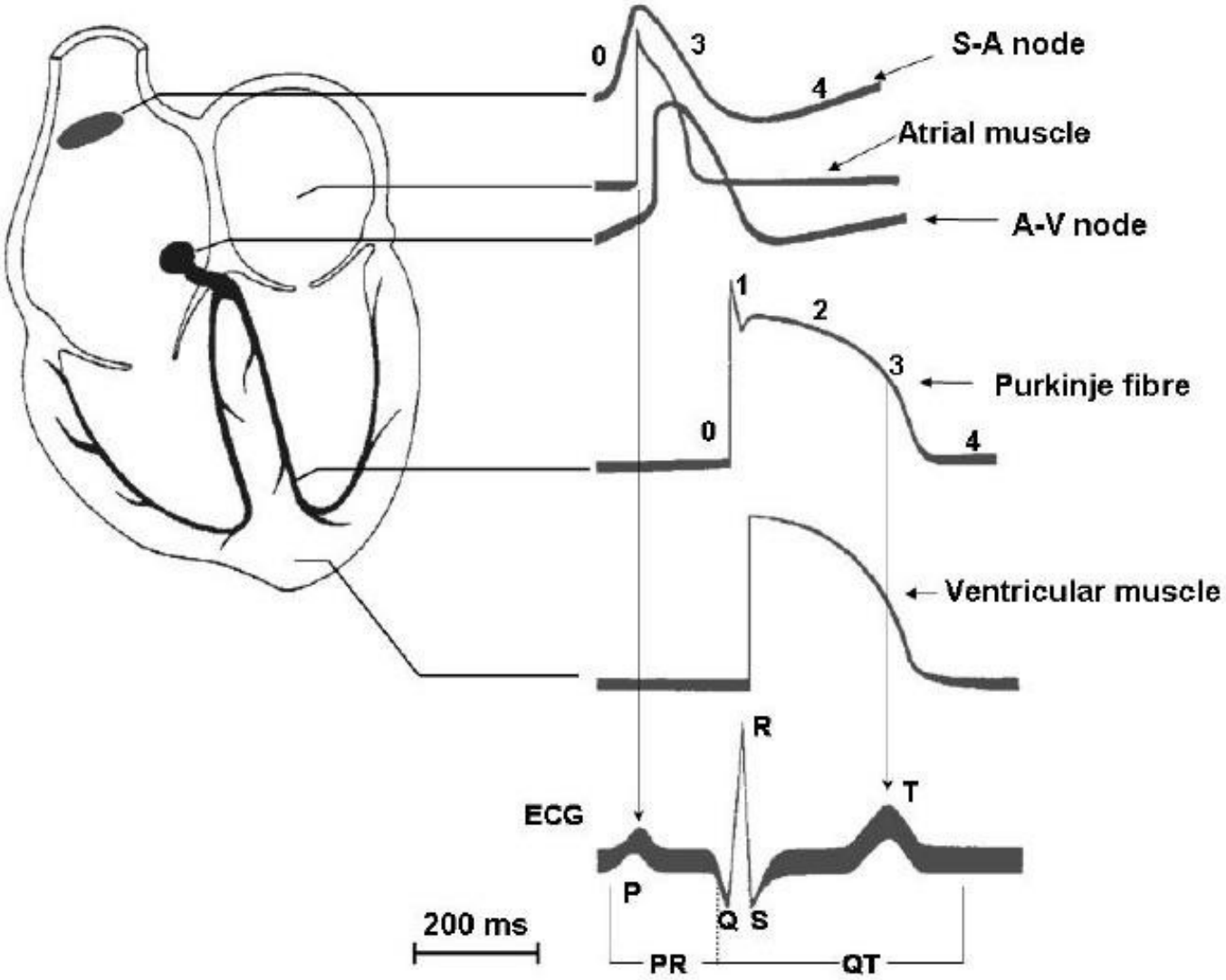
PR interval should be 120 to 200 milliseconds or 3 to 5 little squares

Figure I. Two options for positioning the limb electrodes

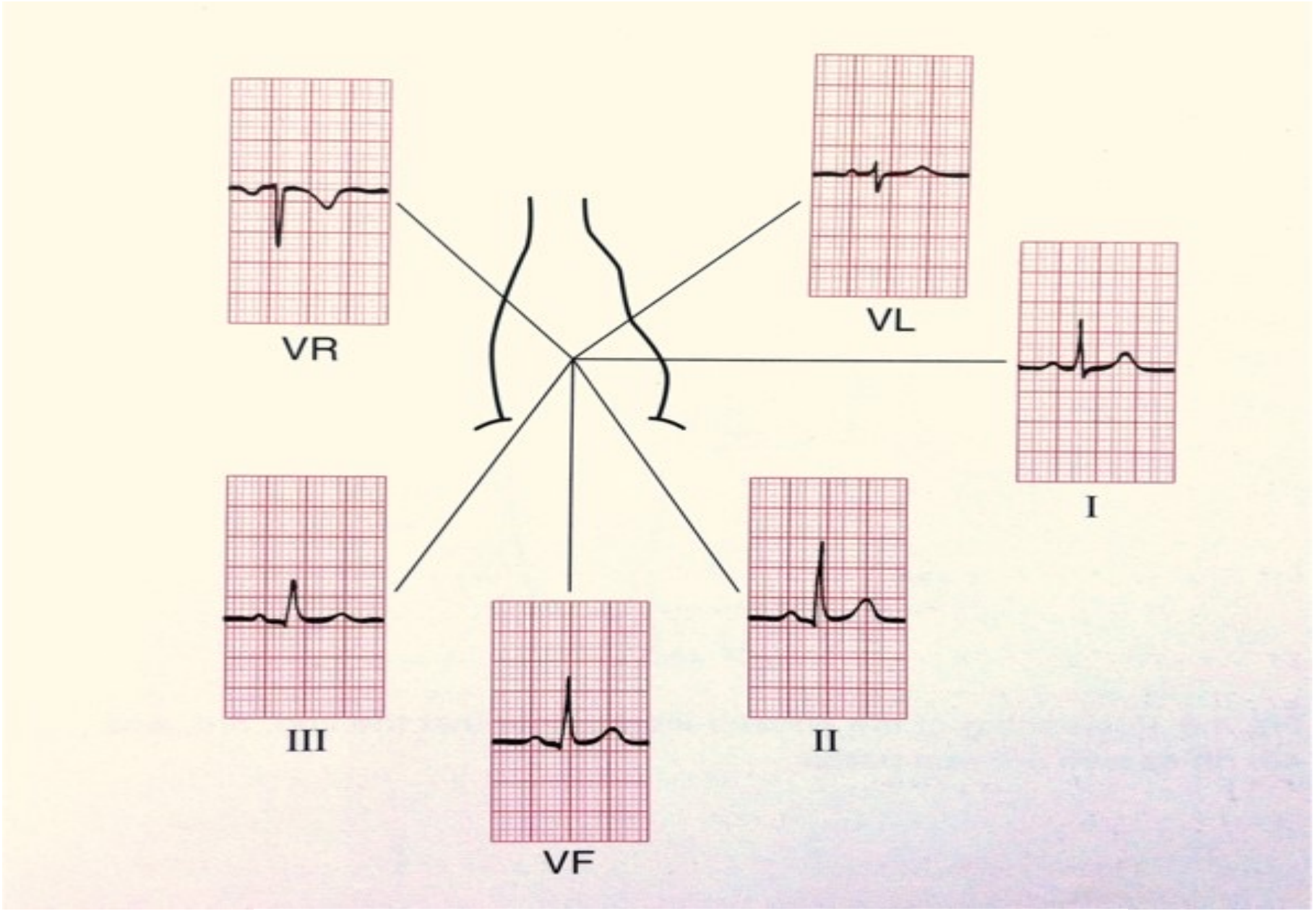


Limb electrodes

Electrical Activity	Graphic Depiction	Associated Pattern
Atrial Depolarization		P Wave
Delay at AV Node		PR Segment
Ventricular Depolarization		QRS Complex
Ventricular Repolarization		T Wave
No electrical activity		Isoelectric Line


0
3
4
S-A node
Atrial muscle
A-V node
1
2
3
0
4
Purkinje fibre
Ventricular muscle
R
T
ECG
P
Q
S
200 ms
PR
QT

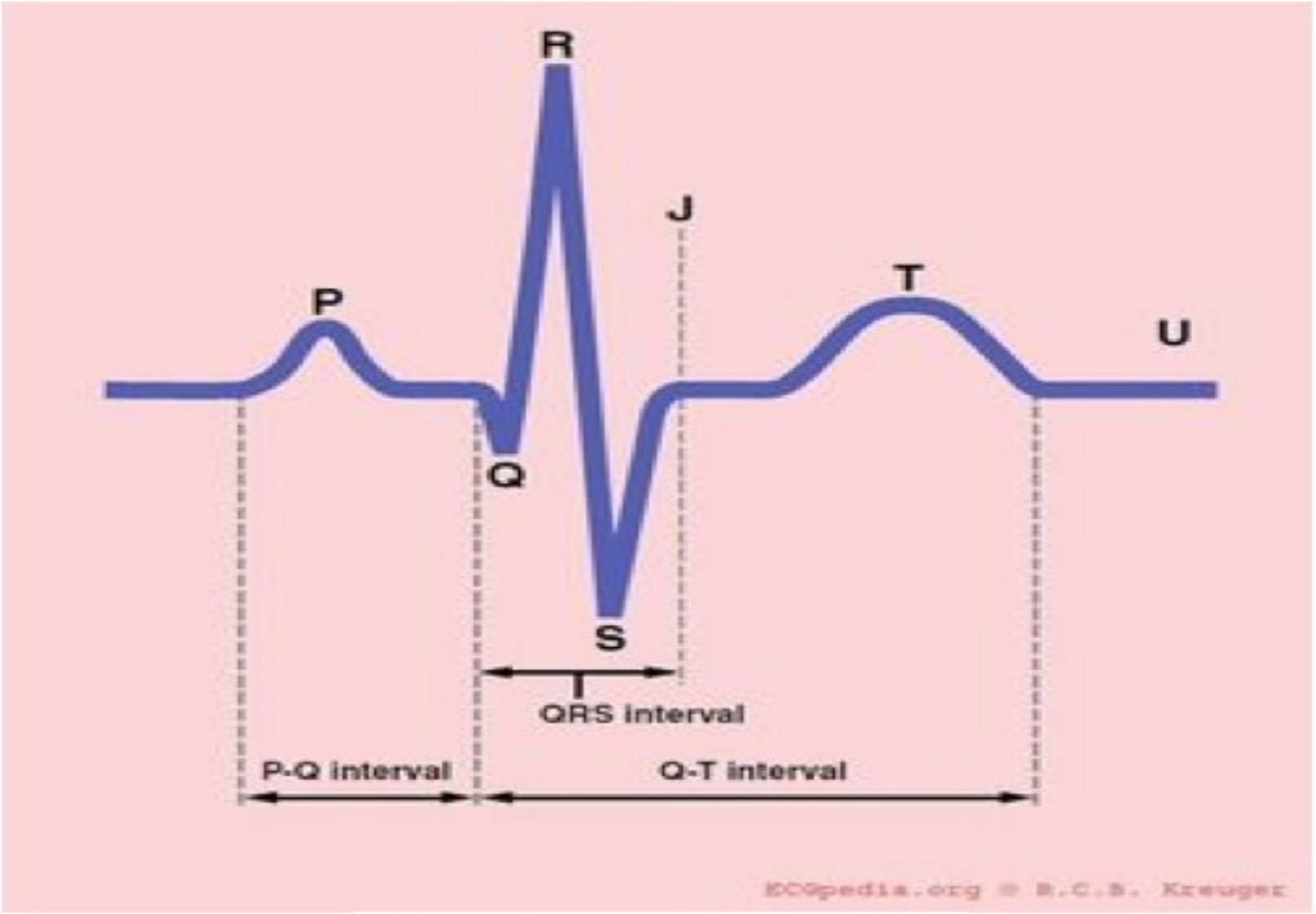
When electrical activity (or depolarisation) travels towards a lead, the deflection is net positive. When the activity travels away from the lead the deflection is net negative.



The electrical activity on an ECG (EKG).

1 large square is equivalent to 0.2 secs and a small square to 0.04 secs

(All boxes are based on the assumption that the paper speed is running at 25mm/sec).



The segments of the ECG.

What do the segments of the ECG represent?

- P-wave: Atrial contraction
- PR interval: Represents the time taken for excitation to spread from the sino-atrial (SA) node across the atrium and down to the ventricular muscle via the bundle of His.
- QRS: Ventricular contraction
- ST segment: Ventricular relaxation
- T-wave: Ventricular repolarisation

Normal duration of ECG segments:

- PR interval: 0.12 – 0.2 secs (3-5 small squares)
- QRS: <0.12 secs (3 small squares)
- QTc: 0.38 – 0.42 secs

How to read an ECG

1. Patient name and details
2. Date
3. Rate
4. Rhthm
5. Axis
6. P-wave and P-R interval
7. Q-wave and QRS complex
8. ST segment
9. QT interval
10. T-wave

These components will now be explained in more detail.

Patient name and details

- Patient's name, date of birth and hospital number
- Location

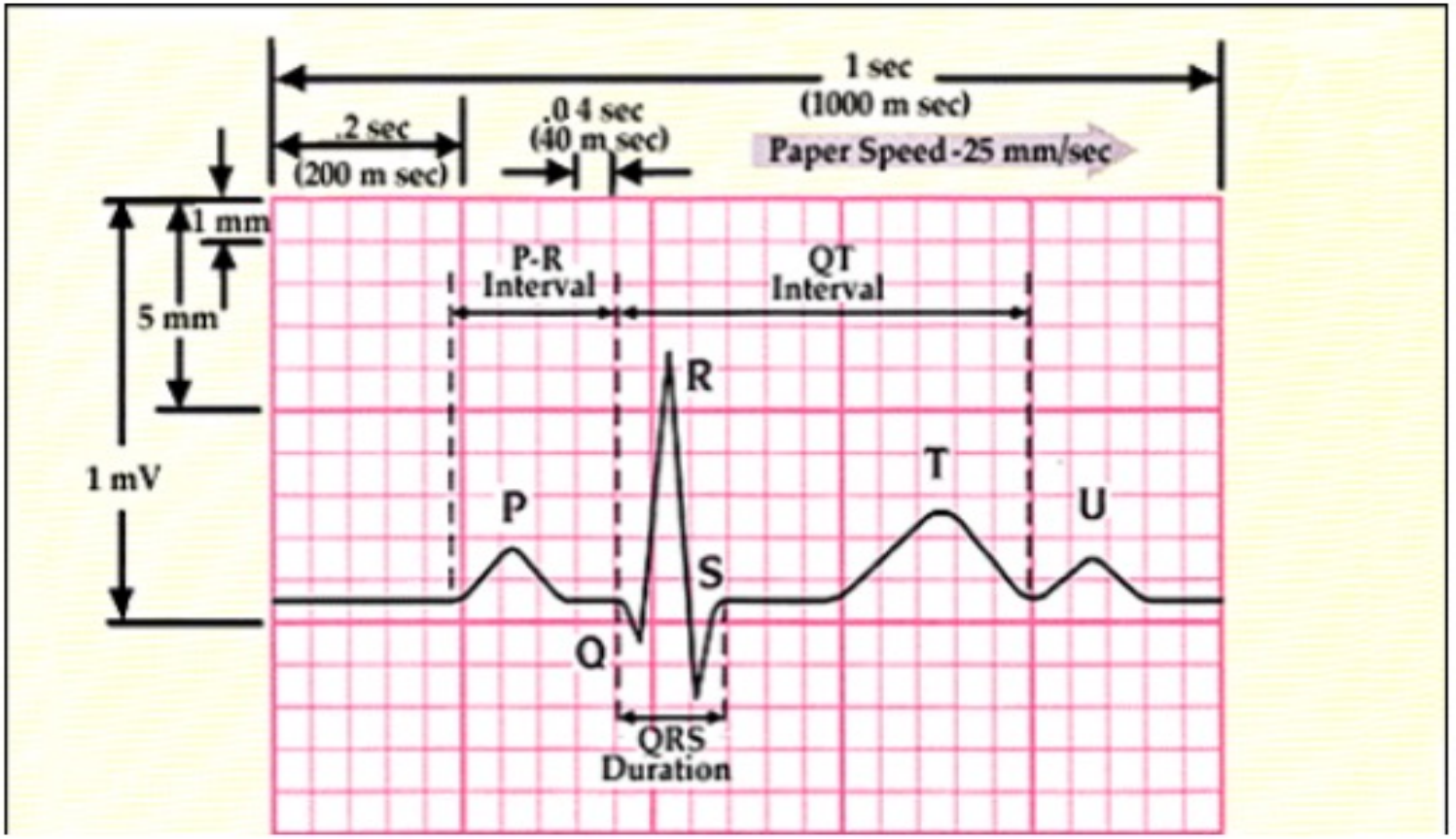
  This becomes important as in the ED or acute medical setting doctors are often shown multiple ECGs. You need to know where your patient is in order to ensure that they can be moved to a higher dependency area if appropriate.

Date and time reference of symptoms

A. When was the ECG done? The time

B. Did the patient have chest pain at the time?

(Or other relevant clinical details. For example, if you are wanted an ECG to look for changes of hyperkalaemia, note the patient's potassium level on the ECG).



ECG paper Key

Thin Lines: 1 mm intervals or 0.04 sec

Thick lines: 5 mm intervals or .2sec

1 thick lined box (5 small boxes) = .20 sec or 5mm

5 thick lines boxes (25 small boxes)= 1 second

10 mm = 1 mV

Tick Marks on ECG paper rhythm strip: 3 secs

There are two different rates that can be determined on an ECG. The atrial rate is indicated by the frequency of the P waves. The ventricular rate is indicated by the frequency of the QRS complexes.

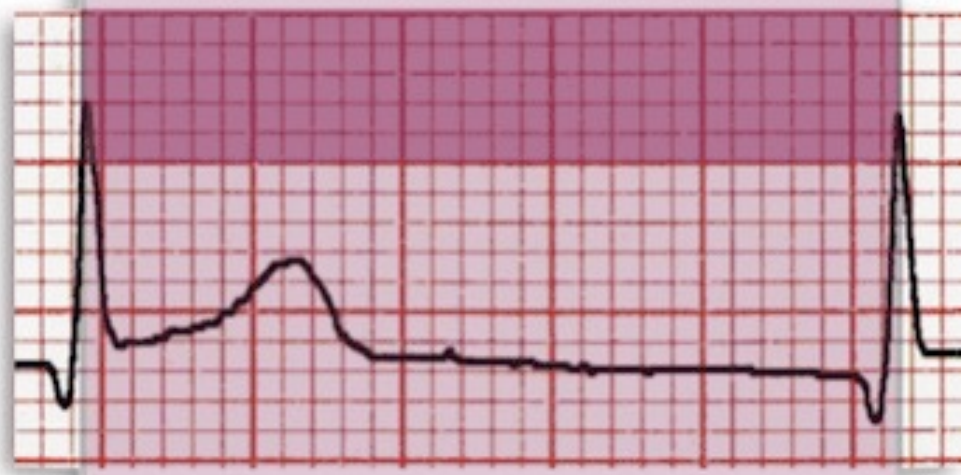
In the absence of disease, the atrial rate should be the same as the ventricular rate. However, certain conditions including third-degree atrioventricular nodal block or ventricular tachycardia can alter this normal relationship, causing “AV dissociation.” In this setting, the atrial rate (P waves) and ventricular rate (QRS complexes) are at different heart rates.

One quick and easy way to measure the ventricular rate is to examine the RR interval — that is, the distance between two consecutive R waves .

HR = 300 / number of large squares between successive R waves

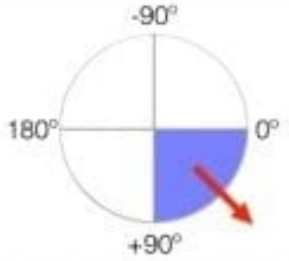
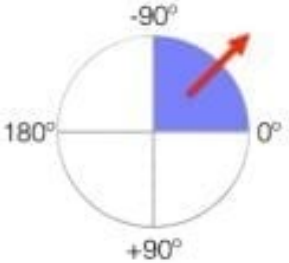
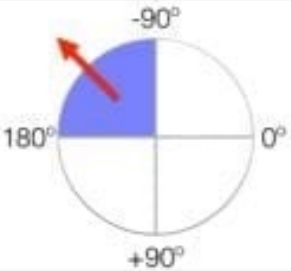
Another quick way is the rule of six seconds to calculate. By counting the number of QRS complexes in 30 large squares and multiplying by 10. (Count the number of RR intervals between two Tick marks (6 seconds) in the rhythm strip and multiply by 10 to get the rate. This method is more effective when the rhythm is irregular).

5.4 **LARGE** squares



$$\frac{\mathbf{300}}{\mathbf{5.4}} = 55.6$$

= 56 bpm

Lead 1	Lead aVF	Quadrant	Axis
POSITIVE	POSITIVE		**Normal Axis** (0 to +90°)
POSITIVE	NEGATIVE		****Possible LAD** (0 to -90°)
NEGATIVE	POSITIVE		**RAD** (+90° to 180°)
NEGATIVE	NEGATIVE		**Extreme Axis** (-90° to 180°)

If you want to work it out more precisely you can use the method below:

- Count the number of small squares of positive or negative deflection in aVF and make a dot on the aVF axis (see Figure 5) moving a mm for each small square counted (e.g. x mm up for negative and x mm down for positive deflections).
- Count the number of small squares of positive or negative deflection in lead 1 and make a dot on the lead 1 axis moving a mm from the centre of the chart for each small square counted (e.g. x mm right for negative and x mm left for positive deflections).
- Draw a vertical line through your lead 1 dot and a horizontal line through your aVF dot then draw a line from this intersection back through 0 and this will give you the accurate axis.

Assessing the axis on an ECG

- Axis is the sum of all the electrical activity in the heart.
- The contraction travels from the atria to the right and left ventricles. As the left ventricle is larger and more muscular normal axis lies to the left (at -30 degrees to 90 degrees – see Figure).
- As a general rule if the net deflections in leads I and aVF are positive then the axis is normal.
  - If lead I has a net negative deflection whilst aVF is positive then there is right axis deviation.
- If lead I has a positive deflection and aVF has a negative deflection then there is left axis deviation

General

Leads: A 12-lead ECG with six **limb leads** (I, II, III, aVL, aVF, aVR) and six **precordial leads** (V_1–V_6) is standard.

Interpretation of the limb leads

I → left ventricle, lateral wall

II, III, and aVF → left ventricle, inferior wall

aVL → left ventricle, high part of the lateral wall

aVR → reciprocal of the left lateral side leads (II, aVL, V_5 and V_6)

Interpretation of the precordial leads

V_1 and V_2 → both ventricles, anterior wall

V_3 and V_4 → anterior wall of the left ventricle and parts of the septum

V_5 and V_6 → lateral wall of the left ventricle and apex of the heart

Paper speed

A paper speed of **25 mm/s** is usually used in the United States: **1 mm** = **0.04 s**

Alternatively, in other countries a paper speed of 50 mm/s is used: 1 mm = 0.02 s

Amplitude: 1 mm (vertical) = 0.1 mV

Interpretation

Compare it with **previous ECGs**.

How to read an ECG

One way of interpreting an ECG:

1. Determine the rhythm (usually best seen in lead II).
2. Measure the frequency (possible in any lead).
3. Determine the heart axis (simple method using leads I–III).
4. Evaluate the morphology and size of the P wave (usually best seen in lead II).
5. Measure the PR interval (usually best seen in lead II).
6. Evaluate the morphology and measure the duration of the QRS complex (look at all leads individually).
7. Evaluate the morphology of the ST segment (look at all leads individually).

Determination of the heart rate

The atrial rate is sometimes calculated (e.g., in assessing some supraventricular arrhythmias).

Implementation

If the QRS rhythm is regular (see determination of the heart rhythm below), then the heart rate can be estimated by **dividing 300** by the number of **large (5 mm) squares** between successive **QRS complexes**, or by counting the number of QRS complexes in 6 seconds and multiplying by 10.

Careful! This method is only a rough estimate.

Only applies if paper speed is 25 mm/s

Interpretation

Normal heart rate: 60–100/min

Tachycardia: > 100/min (see also tachycardic arrhythmias)

Bradycardia: < 50-60/min (see also bradycardic arrhythmias)

Determination of the heart rhythm

The heart rhythm is assessed by evaluating the frequency, regularity, and relationships between the P waves and QRS complexes.

Implementation

1. P wave assessment
   - Are they visible in any lead?
   - Determine the atrial rate (i.e., PP interval).
   - Determine the morphology of the P waves.
2. Relationship of P waves to QRS complexes
   - A 1:1 relationship of P with QRS is normal. If not present:
     - Determine the atrial and ventricular heart rates.
     - Is there an abnormal number of P waves compared to QRS complexes?
   - A P wave before every QRS, and a QRS after every P are normal.
3. QRS morphology
   - Normal duration: **0.07–0.10 seconds**
   - Wide QRS: **> 0.12 seconds** or **3 small squares**

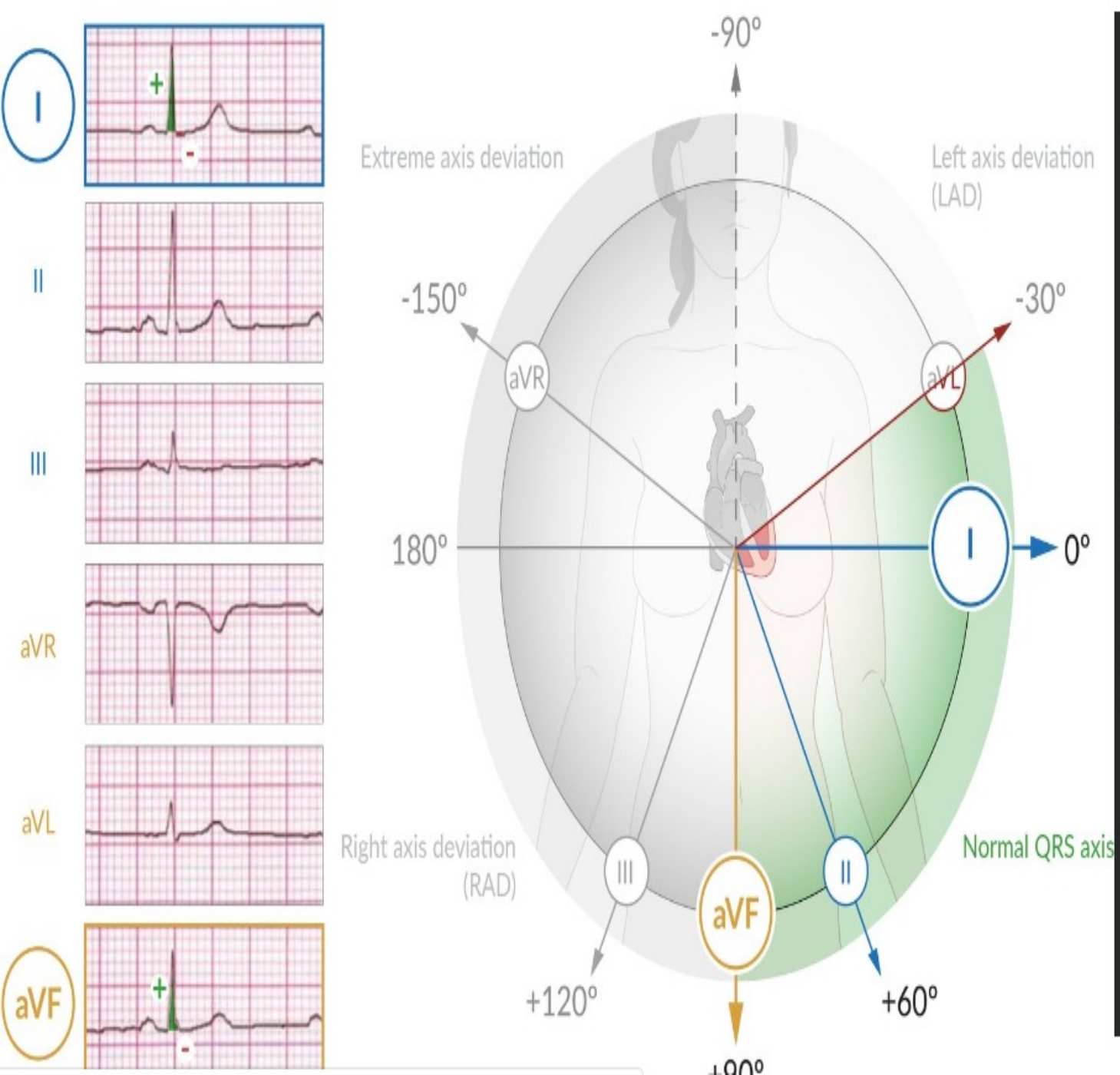
Criteria for a sinus rhythm

1. Normal morphology of the P waves
2. A regular QRS complex follows every P wave.
3. Normal, constant PP and RR intervals

Determination of the axis

- The axis represents the spread of intraventricular electrical activity projected along the frontal plane (determined from limb leads I, II, III, aVR, aVL, aVF).
- The key here is to evaluate the QRS complex, and specifically whether it is positive or negative.
  - **Positive**: if the area above the isoelectric line (i.e., the amplitude) is larger than the area beneath
  - **Negative**: if the area below the isoelectric line is larger than the area above
- The main QRS vector (position of the electrical axis of the heart) is close to the lead with the highest positive QRS amplitude.
- The normal axis of the heart is between -30° and +90°.
- A rapid approximation of the axis may be made by assessing the QRS complexes in leads I and aVF:

Axis	Lead		Degrees	Common causes
	I	aVF		
Left-axis deviation	+	-	(-30°)–(-90°)	Normal variant (especially with age), LVH, LBBB, LAFB, inferior MI
Normal	+	+	(-30°)–(+90°)	Normal axis
Right-axis deviation	-	+	(+90°)–(+180°)	Normal variant, RVH, LPFB, lateral MI, RV strain (e.g., PE), chronic lung disease (e.g., COPD)
Extreme right-axis deviation	-	-	(-90°)–(-180°)	Severe RVH, lateral MI



Determination of the heart axis

Illustration showing the six frontal plane ECG leads and the resulting types of heart axes.

A quick determination of the heart axis (suitable for most clinical circumstances) can be done by comparing the amplitudes of R and S waves in leads I and aVF (although, in principle, other combinations of leads can be used as well).
If a more accurate determination is necessary, additional leads can be employed.

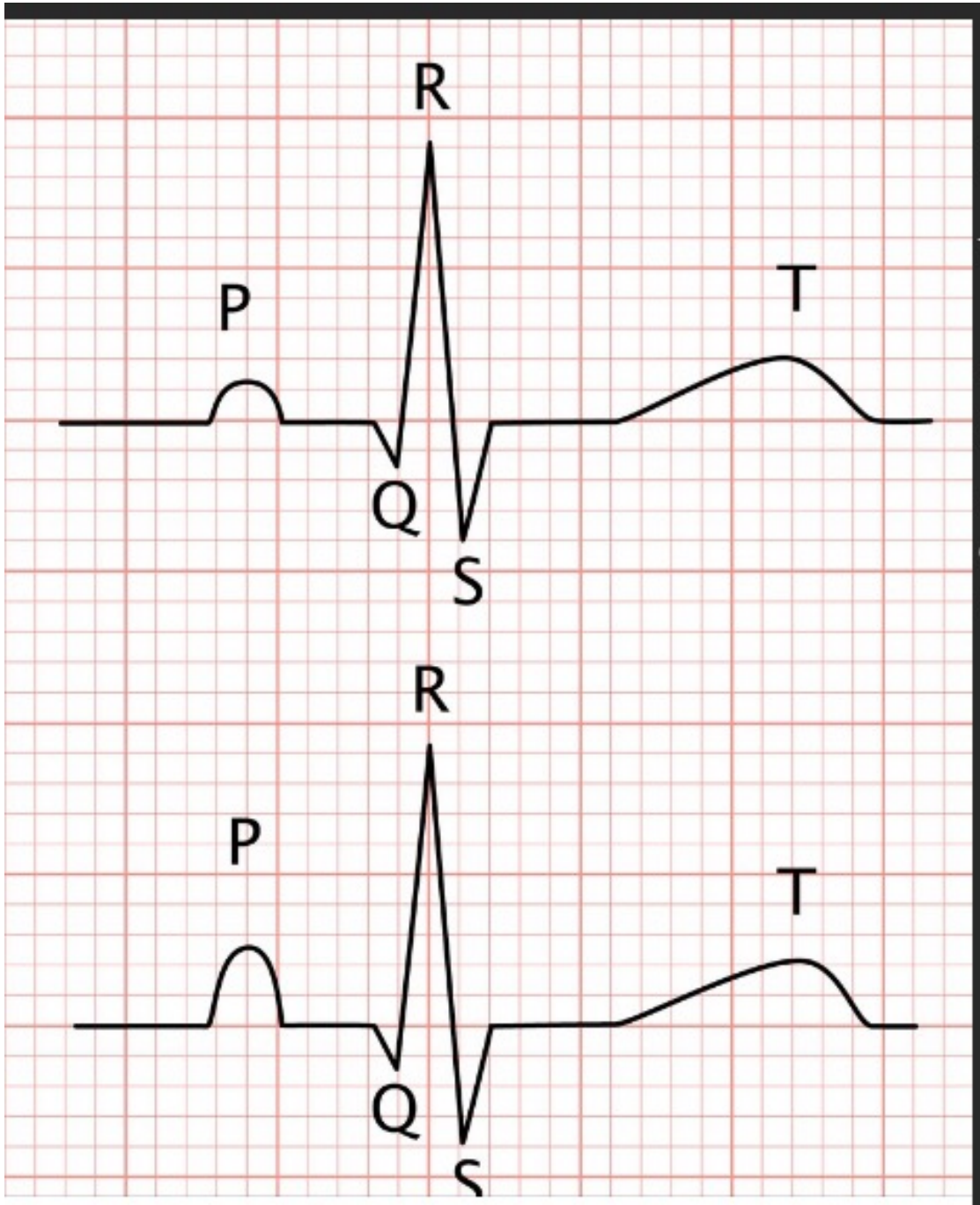
Interpretation of the P wave

P wave	Interpretation	Pathophysiology	Possible etiology

• Elevation of P ≥ 0.25 mV	**P pulmonale**	Effect of **right atrial enlargement**	• Pulmonary disease
			  ◦ **COPD**
			  ◦ **Lung fibrosis**
			  ◦ **Pulmonary hypertension**
			• Other causes of overload of the right atrium (e.g., tricuspid or pulmonary valve stenosis)

• Biphasic P wave
• Prolongation of P > 0.10 s
	P mitrale	Effect of **left atrial enlargement**	• Heart valve defects
			  ◦ **Mitral valve stenosis**
			  ◦ **Severe mitral insufficiency**
			  ◦ **Aortic stenosis**
			• Other causes of overload of left atrium (e.g., cardiomyopathy, myocarditis)

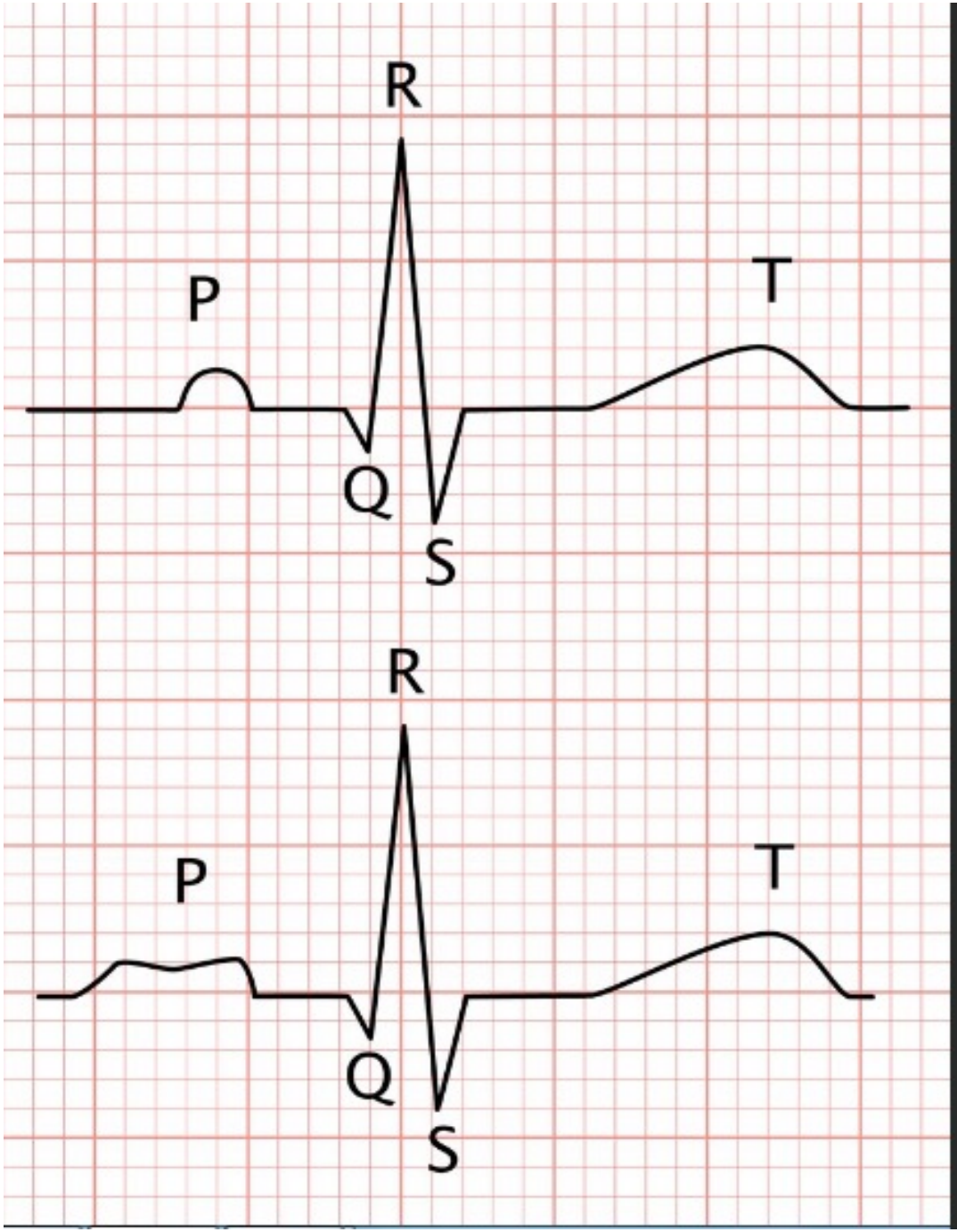
• Biphasic morphology: elevation (≥ 0.25 mV) and prolongation (> 0.10 s)	P biatrial (combination of P mitrale and P pulmonale)	Effect of biatrial enlargement	• Overload of left and right atrium due to global heart strain



P pulmonale

Top: normal ECG

Bottom: Increased P wave (> 0.2 mV in lead II); PR interval approx. 110 ms (paper speed of 50 mm/s); normal QRS complex configuration; no indication of repolarization abnormalities.

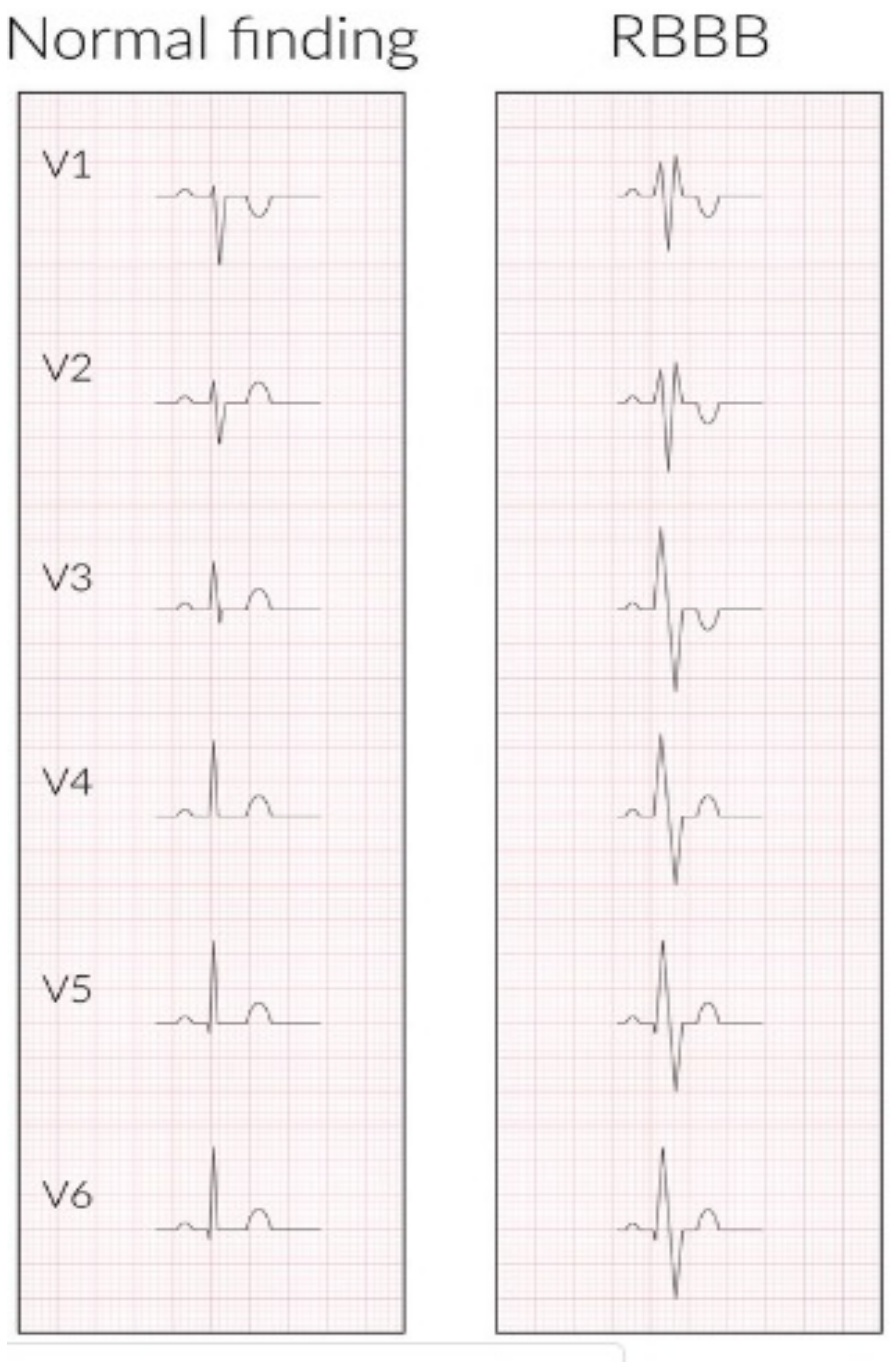


P mitrale

Top: normal ECG. Bottom: prolonged P wave (>0.1 s) with a biphasic wave, normal QRS complex configuration, no indication of repolarization abnormalities

Interpretation of the QRS complex

- The QRS complex represents depolarization of the ventricles and corresponds approximately to ventricular systole.
- **Interpretation of the duration**
  - **≤ 100 ms** = normal
  - 100–110 ms = incomplete bundle branch block (BBB)
  - **≥ 120 ms** = complete bundle branch block (BBB)
    - Signs of **right bundle branch block** (RBBB) are primarily seen in leads **$V_{1,2}$**
      - Prolonged QRS complex
      - rSR' formation (typical M shape/ “rabbit ear” shape)
      - Wide S wave in lead I
      - T wave inversions and ST-segment depression in V_1–-V_3
      - Final negativity (intrinsicoid deflection) in $V_{1,2}$ after > 0.03 s
    - Signs of **left bundle branch block** (LBBB) are primarily seen in leads **I, $V_{5,6}$**
      - Prolonged QRS complex
      - Broad, notched R wave
      - Loss of Q waves
      - Possible rSR' formation in V_5 or $_6$
      - Deep S wave in $V_{1,2}$
      - Final negativity (intrinsicoid deflection) in $V_{5,6}$ after > 0.05



Right bundle branch block (RBBB)

Characteristic changes:

- Prolonged duration of QRS complex ≥ 0.12 s
- rSR' formation in leads V1,2 and sometimes V3 (characteristic M shape)
- Wide S wave in leads V5,6 (and in I und aVL; not shown here)
- Final negativity (intrinsicoid deflection) in V1,2 after > 0.03 s in leads V1 and V2
- T-wave inversion in V1,2 and sometimes in V3

The name William Morrow can help you identify LBBB and RBBB by looking at the QRS morphology in V_1 and V_6. In LBBB the QRS looks like a W in V_1 and an M in V_6 (WiLLiaM), in RBBB the QRS looks like an M in V_1 and a W in V_6 (MoRRoW).

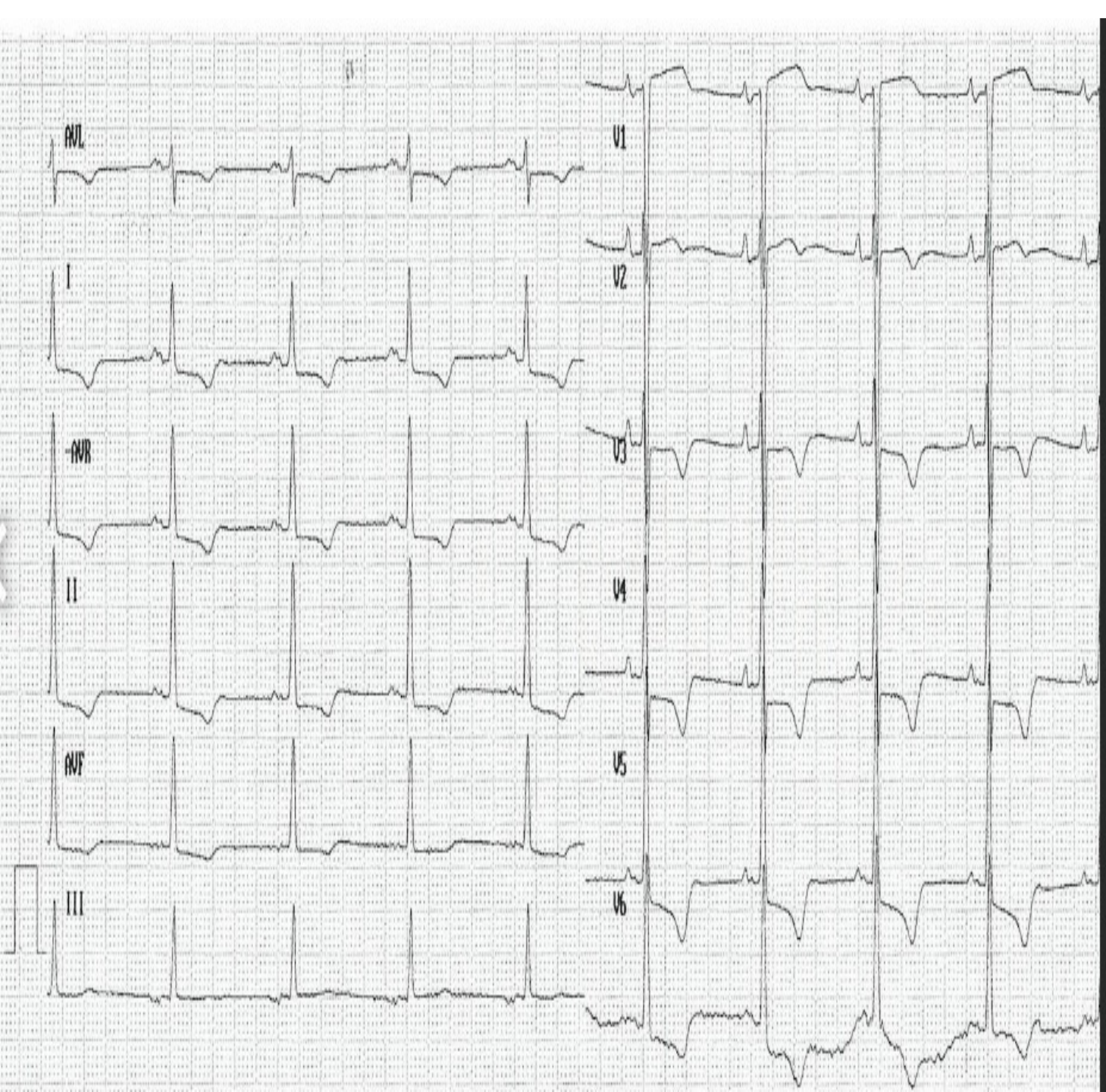
Interpretation of amplitude

Amplitude of the QRS complex in the precordial leads is used to assess for ventricular hypertrophy

Various grading criteria exist for electrocardiographic determination of ventricular hypertrophy.

The **Sokolow-Lyon criteria** are utilized below:

- Left ventricular hypertrophy (LVH): $S_{V1 \text{ or } 2} + R_{V5 \text{ or } 6} \geq 3.5$ mV
- Right ventricular hypertrophy (RVH): $R_{V1 \text{ or } 2} + S_{V5 \text{ or } 6} \geq 1.05$ mV



ECG with left ventricular hypertrophy

12-lead ECG (paper speed 25mm/s) in a 75 year-old patient with left ventricular insufficiency in coronary heart disease: Sinus rhythm with a heart rate of 55-60/min. Normal QRS complex.

, P mitrale

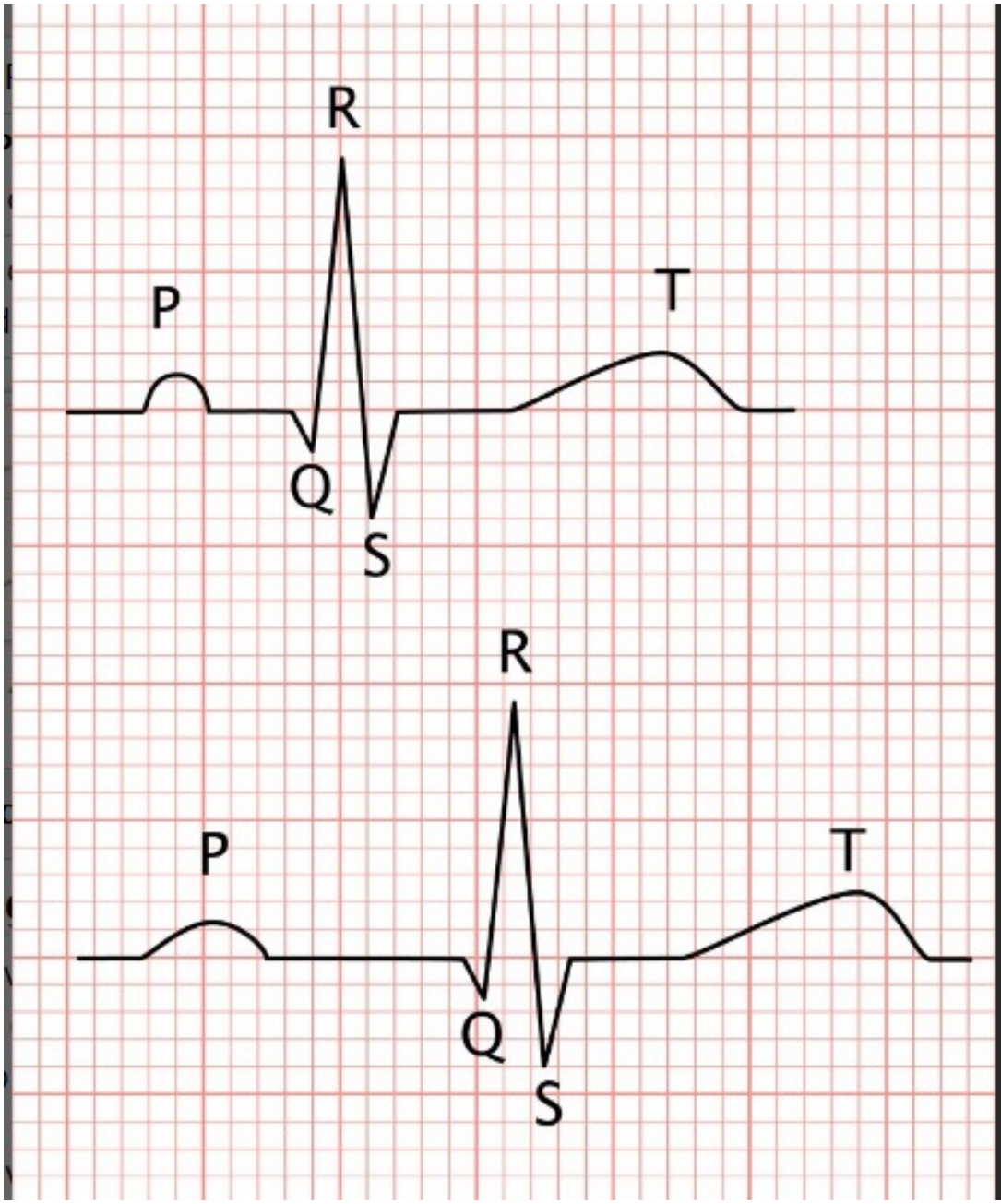
(prolongation of P to 0.12s, typical biphasic P wave), positive Sokolow-Lyon index for left ventricular hypertrophy (SV2 + RV5 > 3.5mV), ST depression with pre-terminal T-wave inversion in I, II, aVF, V4-6. Diagnosis: left ventricular hypertrophy

Interpretation of the PR interval

The time between the beginning of the P wave and the beginning of the Q wave

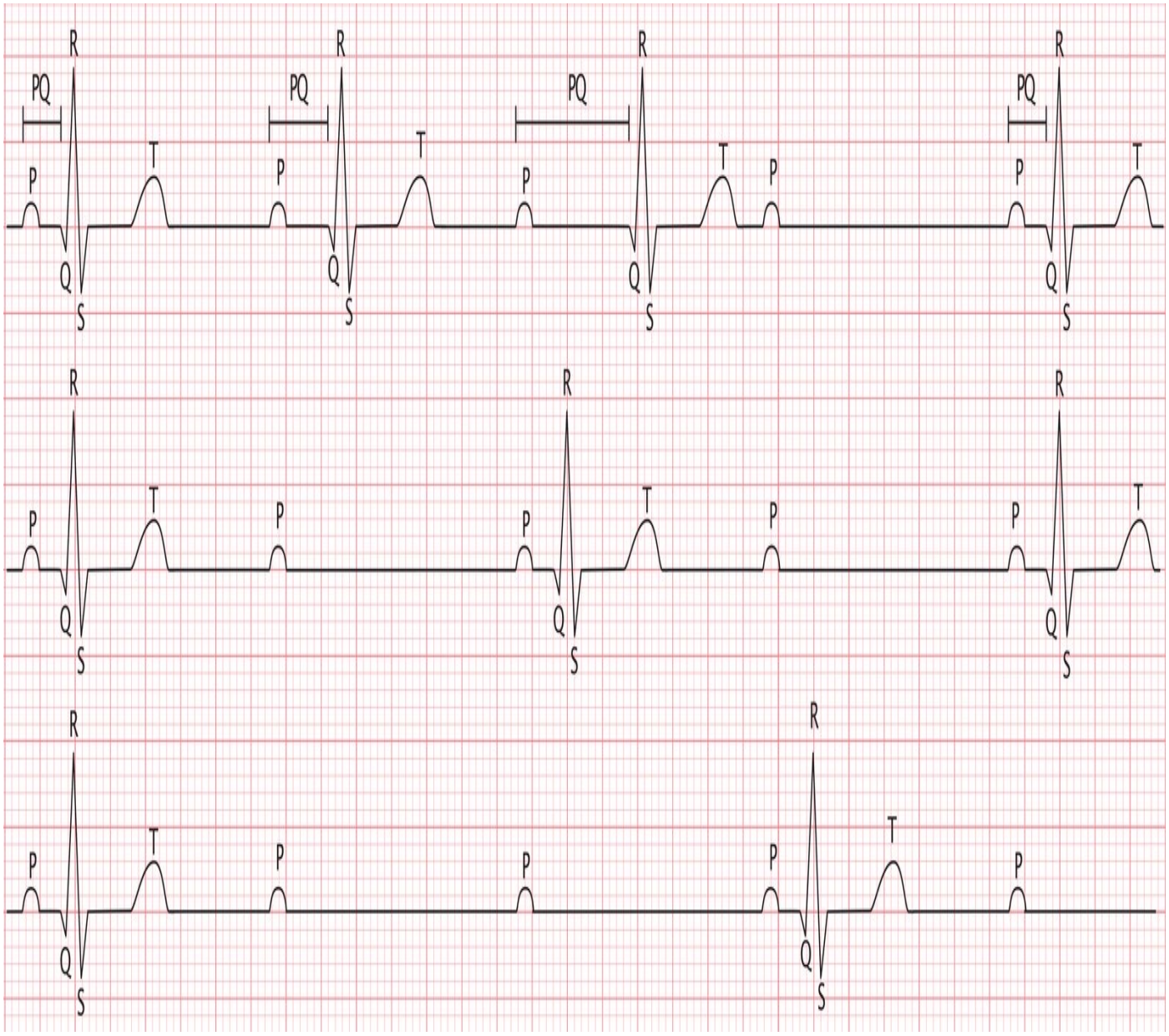
The PR interval represents atrioventricular transmission.

PR interval	Interpretation
PR interval ≤ **0.2 s**	Normal
PR interval > 0.2 s	First-degree atrioventricular block
PR intervals become **progressively longer** (but PP intervals remain constant) until a dropped QRS complex occurs after a regular atrial depolarization.	Second-degree AV block, Mobitz type I (Wenckebach)
Constant PR intervals (which are usually normal but may be prolonged) followed by one or more non-conducted P waves.	Second-degree AV block, Mobitz type II
P waves and QRS complexes occur **independently** of each other, but in regular intervals → complete dissociation of P waves and QRS complexes.	Third-degree AV block



First–degree atrioventricular block

Top: normal ECG. Bottom: PR interval > 200 ms (paper speed of 50 mm/s). Normal QRS complex configuration. No indication of repolarization abnormalities.



Second-degree atrioventricular blocks

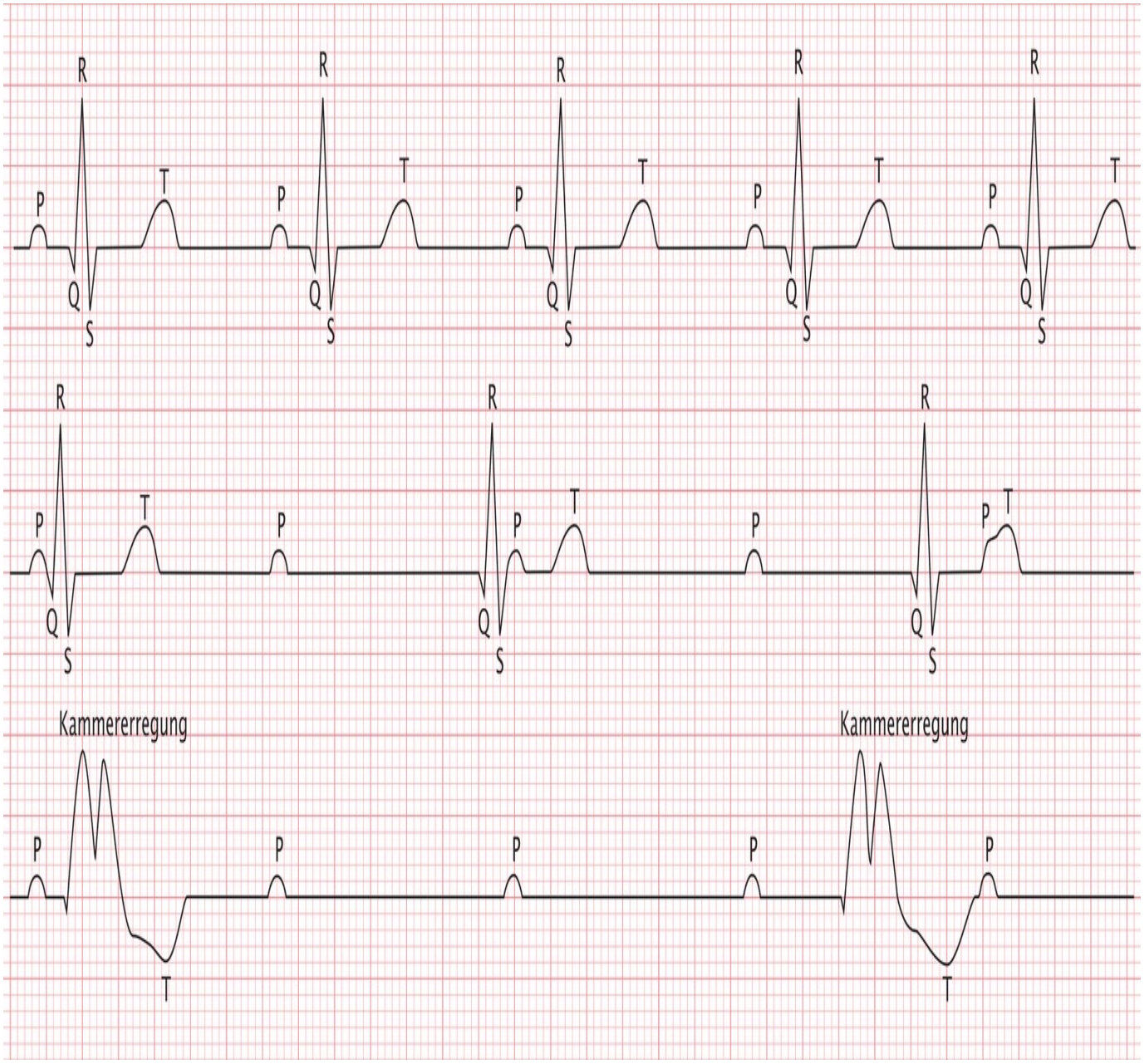
ECG illustration of AV blocks (paper speed: 50 mm/s)

Regular P waves with a frequency of 85/min in all tracings.

Top: second-degree AV block (Mobitz I/Wenckebach). Progressive lengthening of the PR interval with a blocked QRS complex following the fourth P wave. Ventricular rate approx. 74/min.

Middle: second-degree AV block (Mobitz II). PR interval is constant but only every second P wave is conducted to the ventricle, where it triggers a QRS complex (2:1 block). Ventricular rate is approx. 42/min.

Bottom: second-degree AV block (Mobitz II). PR interval is constant but only every third P wave is conducted to the ventricle, where it triggers a QRS complex (3:1 block). Ventricular rate is only approx. 28 bpm.



Third-degree atrioventricular block

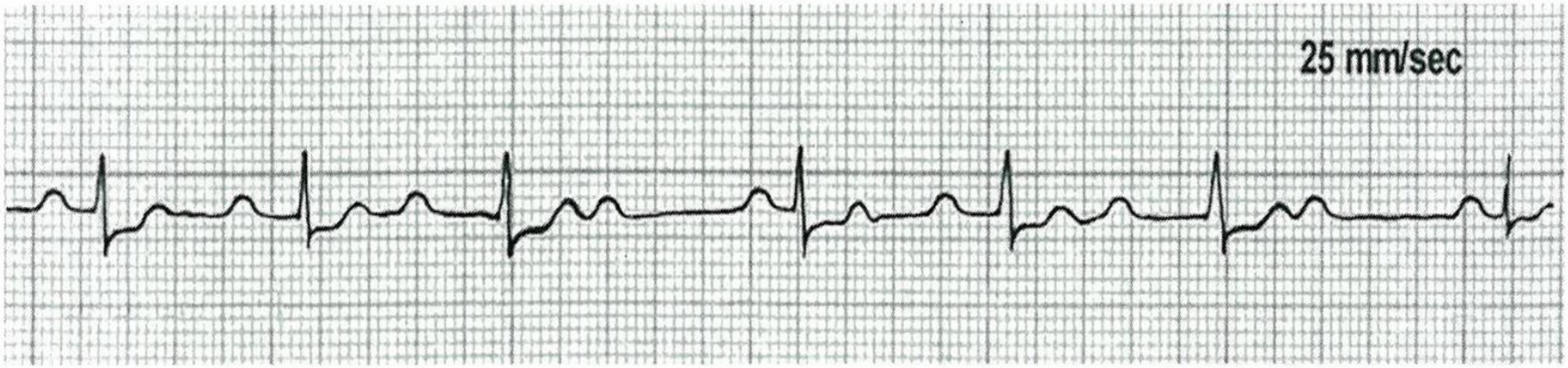
ECG illustration of AV blocks (paper speed: 50 mm/s)

Top: normal ECG with a heart rate of approx. 90/min.

Middle: third-degree AV block with a ventricular escape rhythm (site of origin at the AV node) with a rate of approx. 50/min. QRS complexes are narrow but within normal limits.

Bottom: third-degree AV block with a ventricular escape rhythm (site of origin at the bundle of His or the bundle branches) with a rate of approx. 27/min. QRS complexes are widened, resembling bundle branch block.

In both types of third-degree AV block, the P waves occur at a regular rate of approx. 90/min (the rate of the sinoatrial node). However, the QRS complexes occur in accordance with the rhythm of the abnormal site of origin.

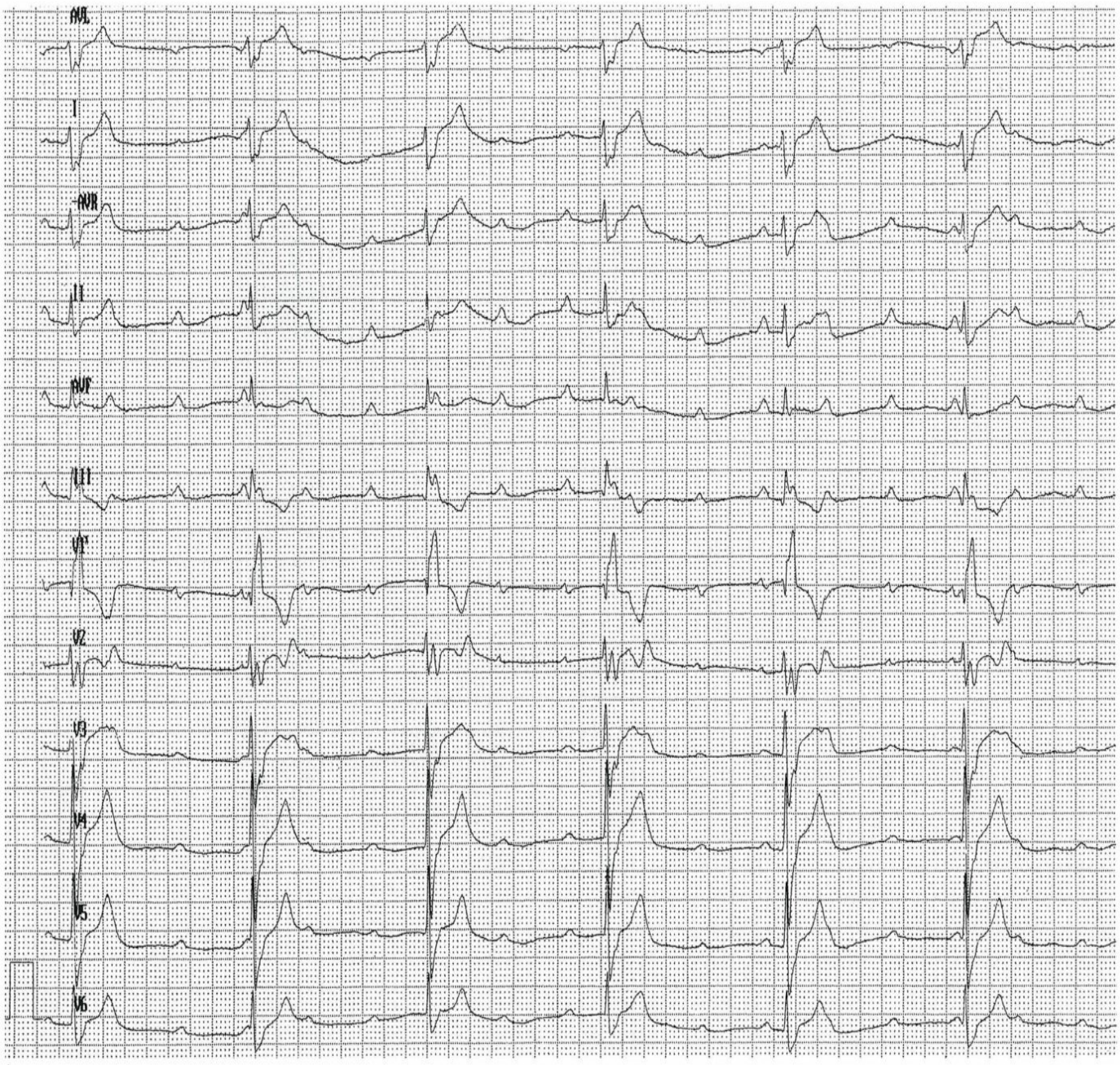


Second-degree AV block (Mobitz I, Wenckebach)

ECG strip (paper speed 25mm/s)

Regular P waves with a frequency of 75–80/min. The PR interval increases steadily until, after the fourth P wave, the QRS complex is missing.
The QRS complexes have normal morphology but occur irregularly (frequency approx. 65/min).

Diagnosis: second-degree atrioventricular block (Mobitz I, Wenckebach)



Third-degree atrioventricular block and ventricular escape rhythm

12-lead ECG (paper speed: 25 mm/s)

P waves occur regularly at a rate of 100/min without conduction to the ventricle; therefore, there is no sinus rhythm. The ventricular rate is approx. 25–40/min (ventricular escape rhythm). There is right axis deviation. QRS complexes are wide and deformed. Repolarization cannot be assessed because of the abnormal depolarizations.

Interpretation of the Q wave

Physiological

The Q wave represents the beginning of ventricular depolarization.

A narrow (**≤ 40 ms**) Q wave is physiological in:

- All limb leads
- aVR
- V_5 and V_6

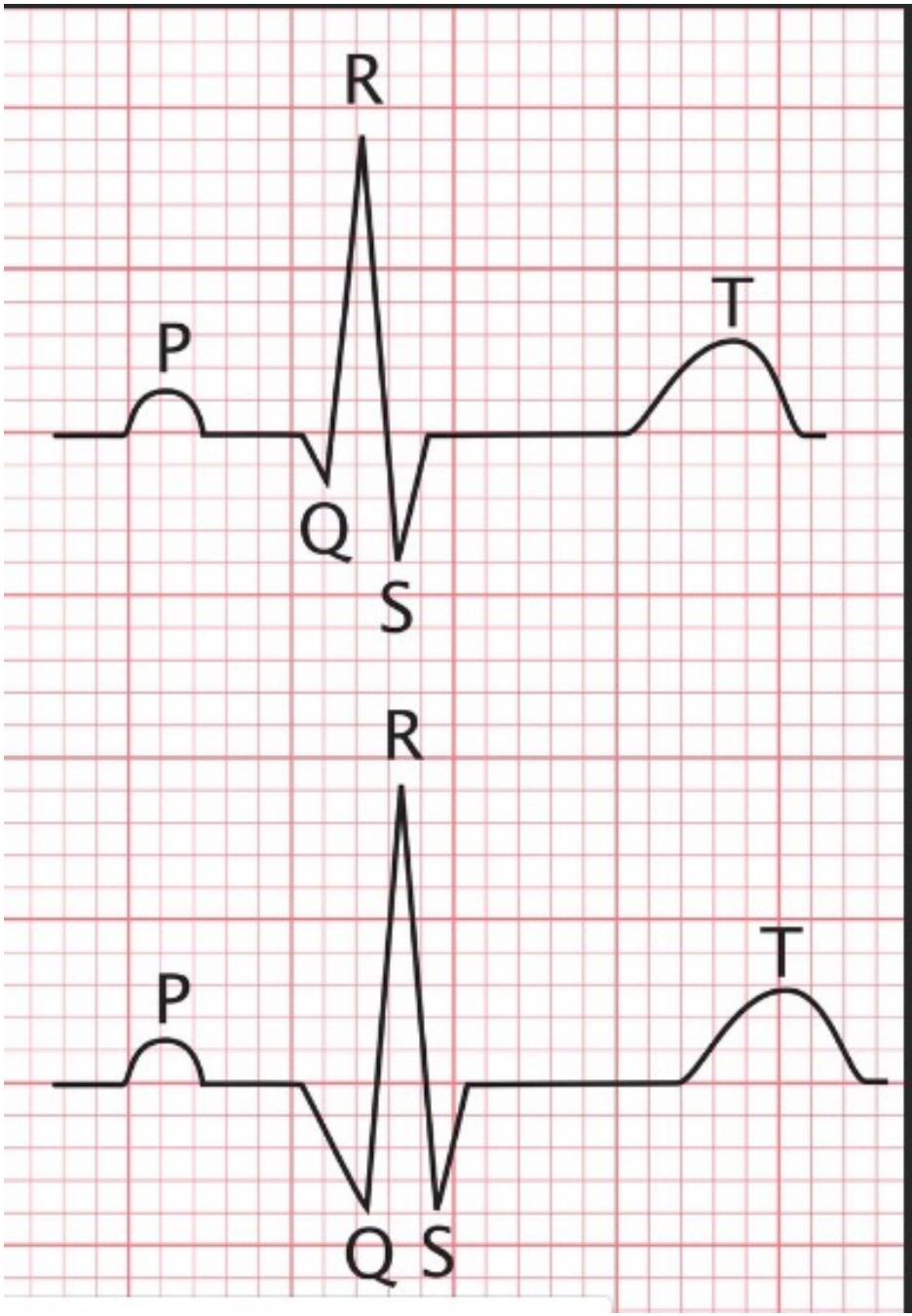
Pathological

Pathological Q waves are characteristically:

- Abnormally wide (**≥ 40 ms**)
- Abnormally deep (**≥ 2 mV** or **> 25%** of the R wave amplitude) or, detectable in V_1–V_3

Etiology

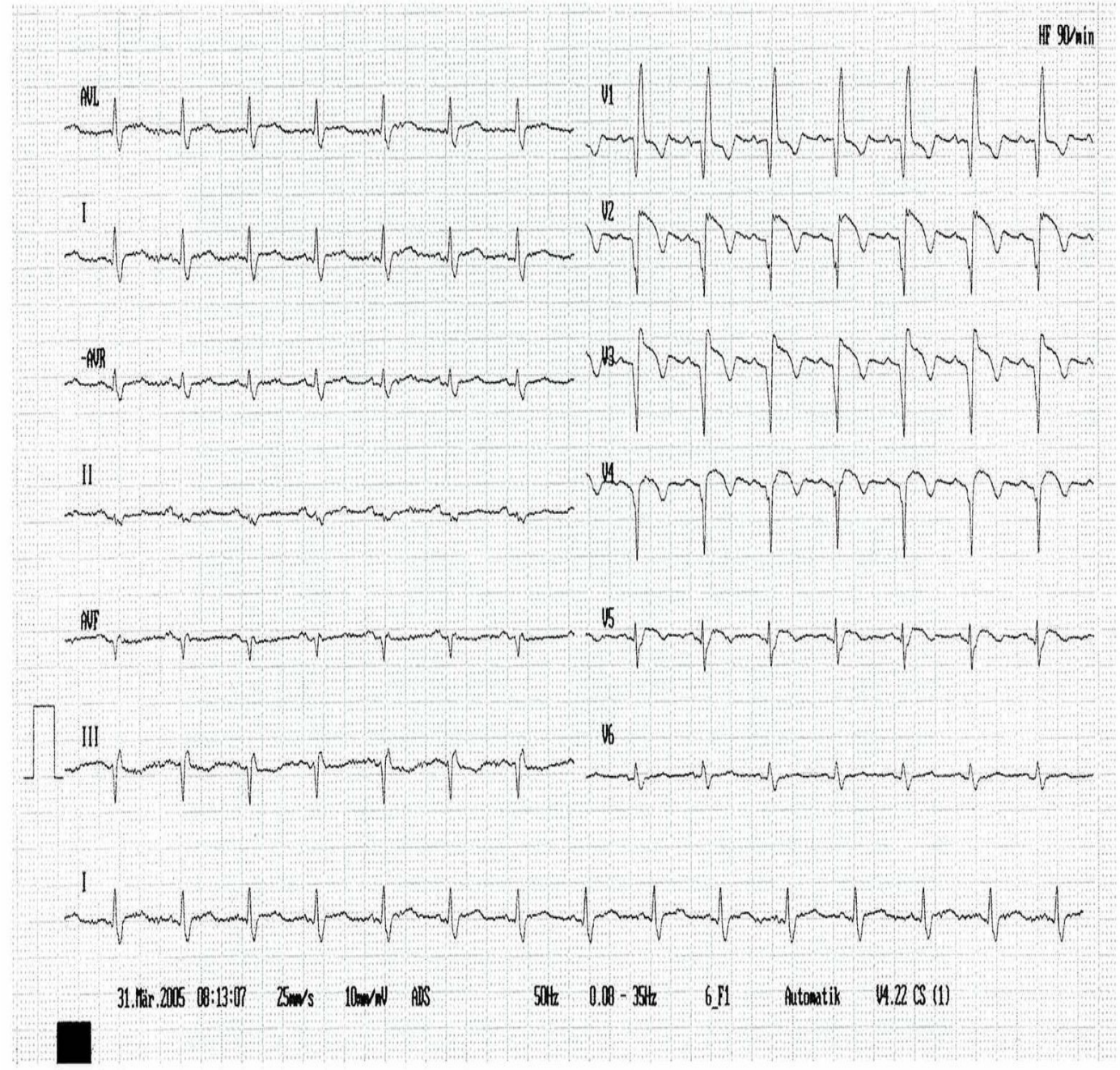
- Myocardial injury or replacement
  - **Myocardial infarction**
  - Cardiac infiltrative disease (e.g., sarcoidosis, amyloidosis)
- Ventricular enlargement
  - Acute pulmonary embolism
  - Hypertrophic cardiomyopathy
- Altered ventricular conduction
  - Left bundle branch block
  - Wolff-Parkinson-White syndrome



Pathologic Q wave

Upper image: normal ECG
Lower image: pathological Q wave (duration ≥ 0.04 s; ≥¼ of preceding R wave)

A pathological Q wave is frequently seen after myocardial infarction or in hypertrophic cardiomyopathy.



ST elevation myocardial infarction on ECG (post-intervention)

12-lead ECG (paper speed: 25 mm/s)

Sinus rhythm with a heart rate of approx. 90/min. Left axis deviation. ST elevation in V2-V4. Pathological Q wave in V1-V4. R wave loses amplitude over the anterior wall. Terminal T-wave inversion in V2-V4, which indicates the intermediate stage of an anterior myocardial infarction.

A new pathological Q wave represents myocardial infarction until proven otherwise!

ST segment

Physiological

The ST segment represents the interval between ventricular depolarization and repolarization

It is physiologically horizontal on the isoelectric line.

Pathological

ST elevation

An ST elevation is significant if:

- **≥ 0.1 mV** in limb leads, or
- **≥ 0.2 mV** in precordial leads!

The hallmark ECG finding of **myocardial infarction!**

- If significant ST elevations are present in **≥ 2 anatomically contiguous leads** (corresponding to occlusion of a specific artery)
- The ischemia can be localized by which leads show ST elevation:
  - **Lateral MI** (left circumflex artery occlusion): I, aVL, V_{5-6}
  - **Anterior MI** (left anterior descending (LAD) artery occlusion): V_{1-4}
  - **Inferior MI** (terminal branches of right or left coronary artery occlusion): II, III, aVF

Widespread ST elevations suggest **pericarditis**

LBBB may cause ST elevations due to repolarization abnormalities, therefore ST elevation cannot be used to diagnose MI in the presence of a LBBB.

Small, concave ST elevations may be a normal finding in young, healthy adults due to early repolarization.

From descending R: The most important cause is a myocardial infarction.

From (deep) S: perimyocarditis

Brugada pattern

Associated with **Brugada syndrome**: rare **autosomal dominant** condition that affects sodium channels and disturbs repolarization

Epidemiology: most common in Asian males

Clinical features

- Often an incidental finding, as patients are mostly asymptomatic
- Syncope
- Sudden cardiac death

Diagnosis

- Brugada pattern on ECG: Pseudo-RBBB with ST elevation in leads V_{1-2}
- Rule out underlying heart disease (e.g., stress test and echocardiography)

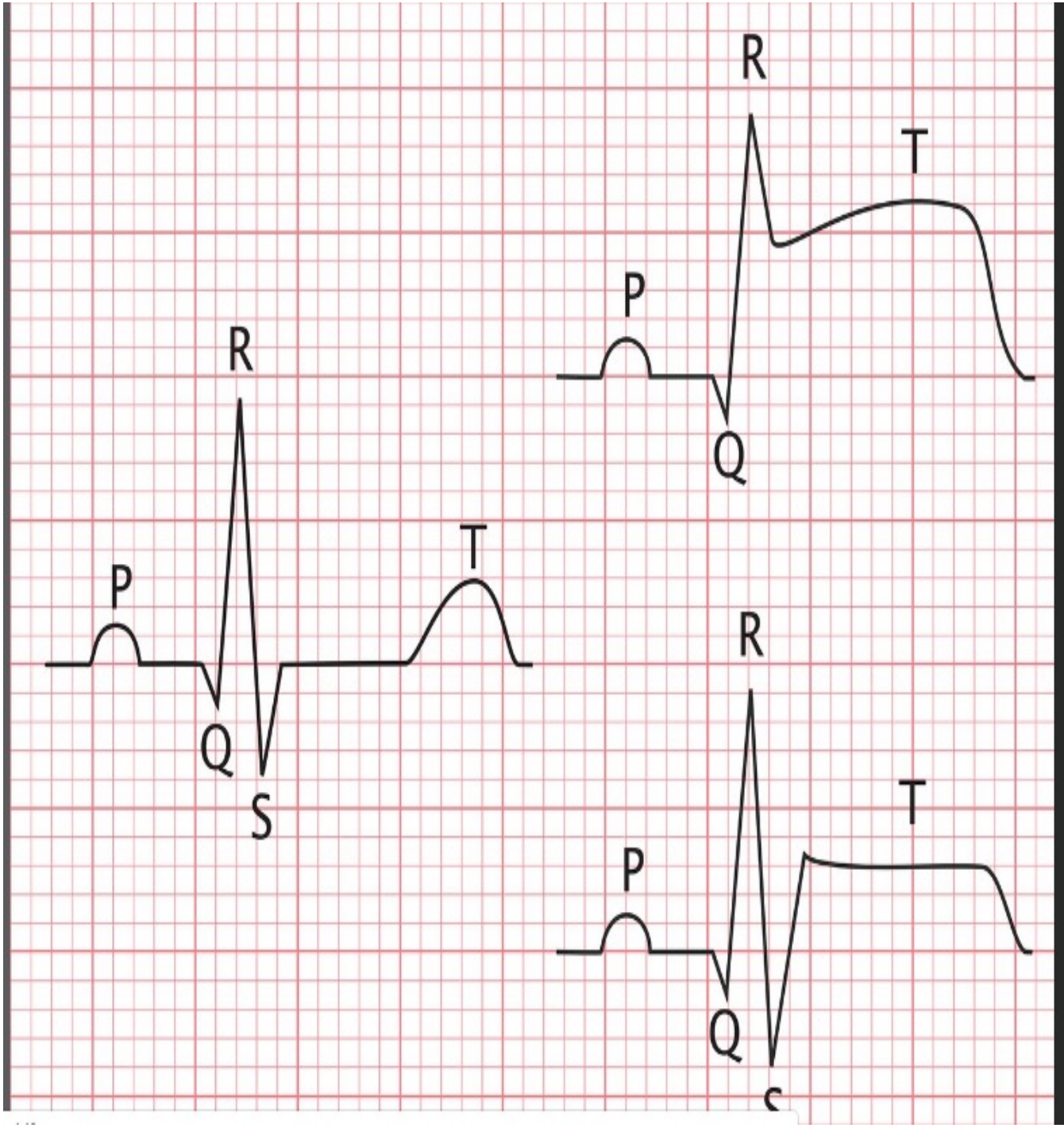
Treatment

- Implantable cardiac defibrillator (ICD) placement
- Screen all 1^{st}-degree relatives annually with clinical exam and ECG

Complications

- Syncope
- Sudden cardiac death
- Increased risk of atrial fibrillation

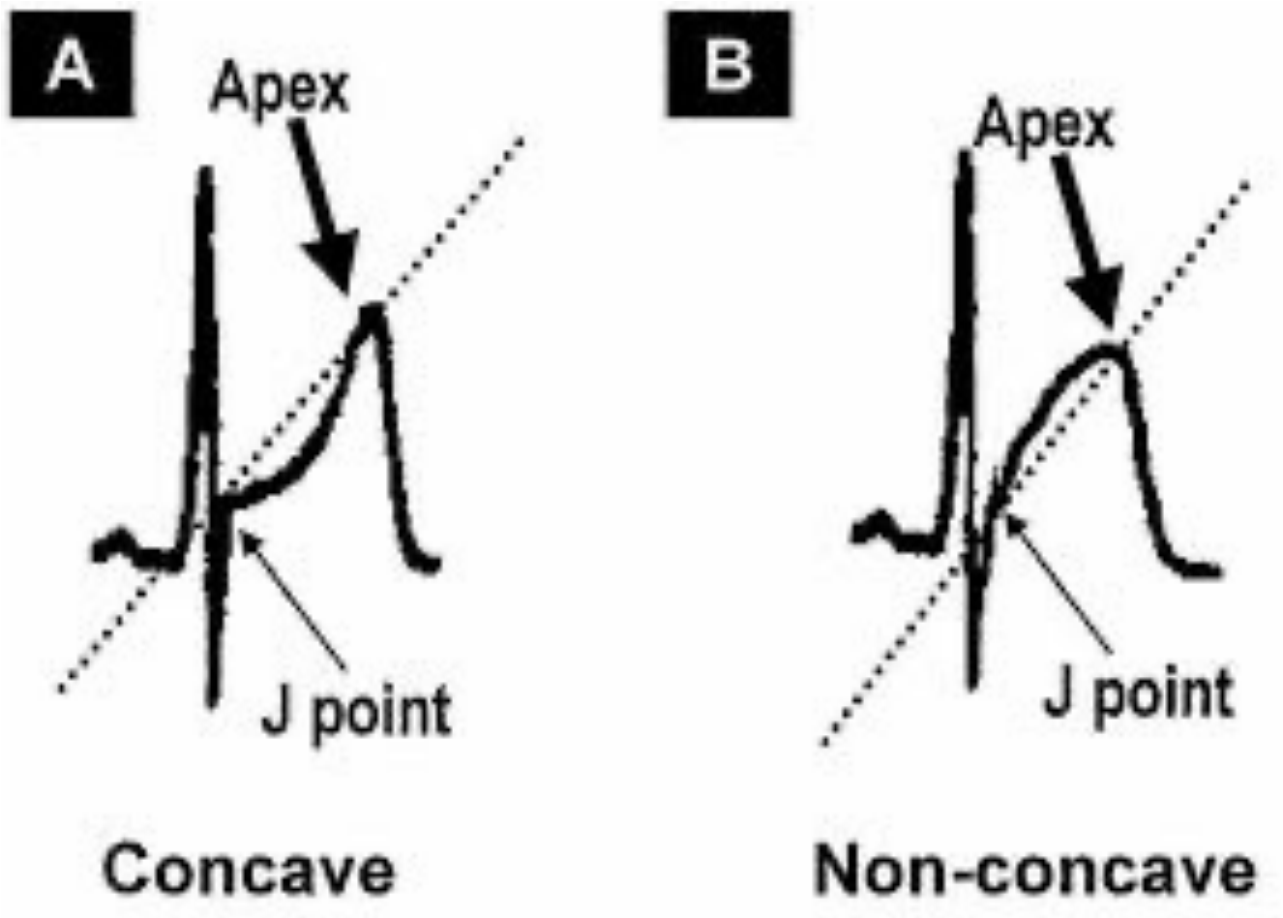
ST elevation from a descending R is likely caused by a myocardial infarction!

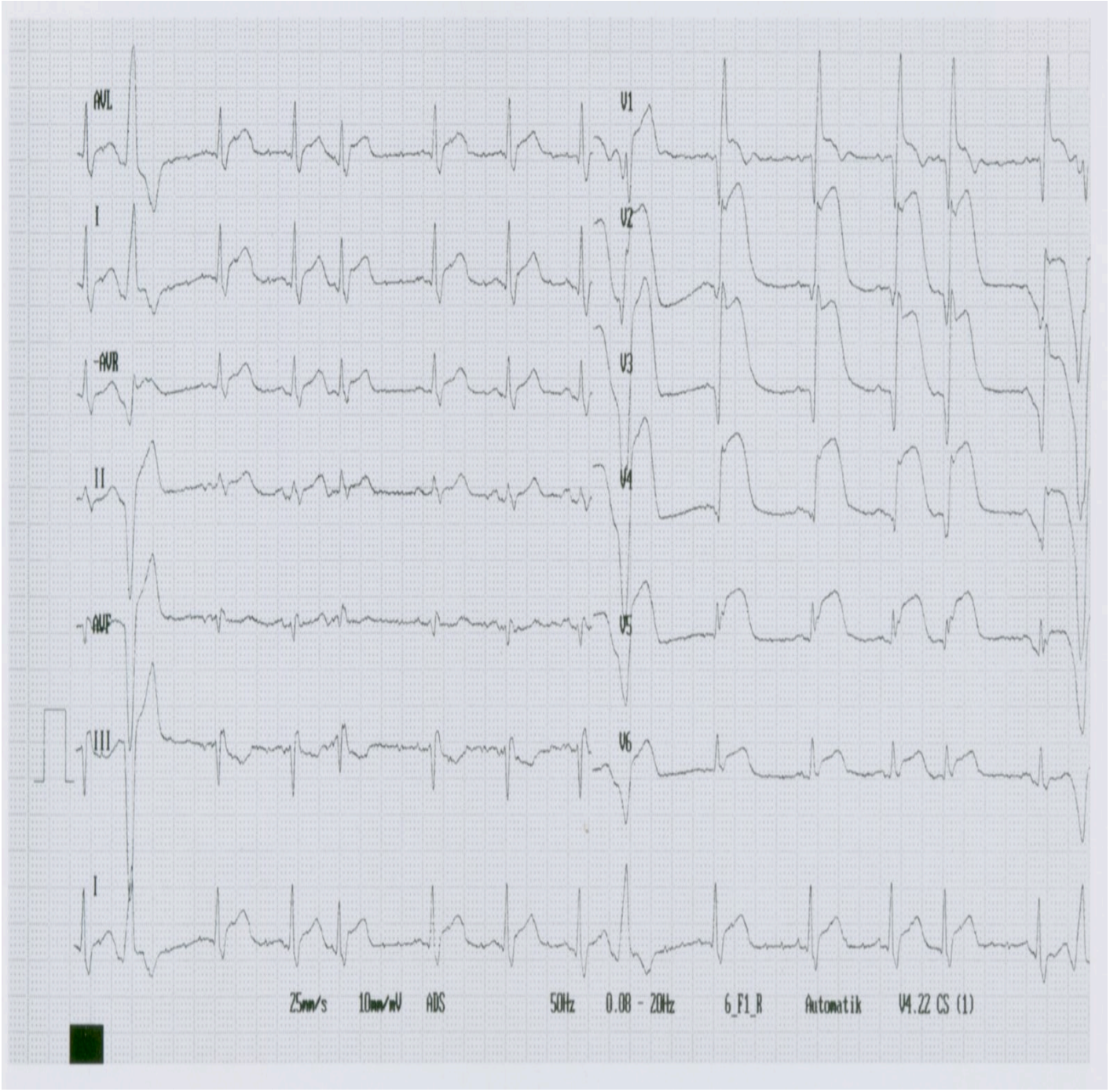


Types of ST–segment elevation

Left: normal ECG
Top right: convex upward ST elevation from descending R
Bottom right: concave ST elevation from deep S

An ST elevation may be visible in various conditions including myocardial infarction, myocarditis, or pericarditis.

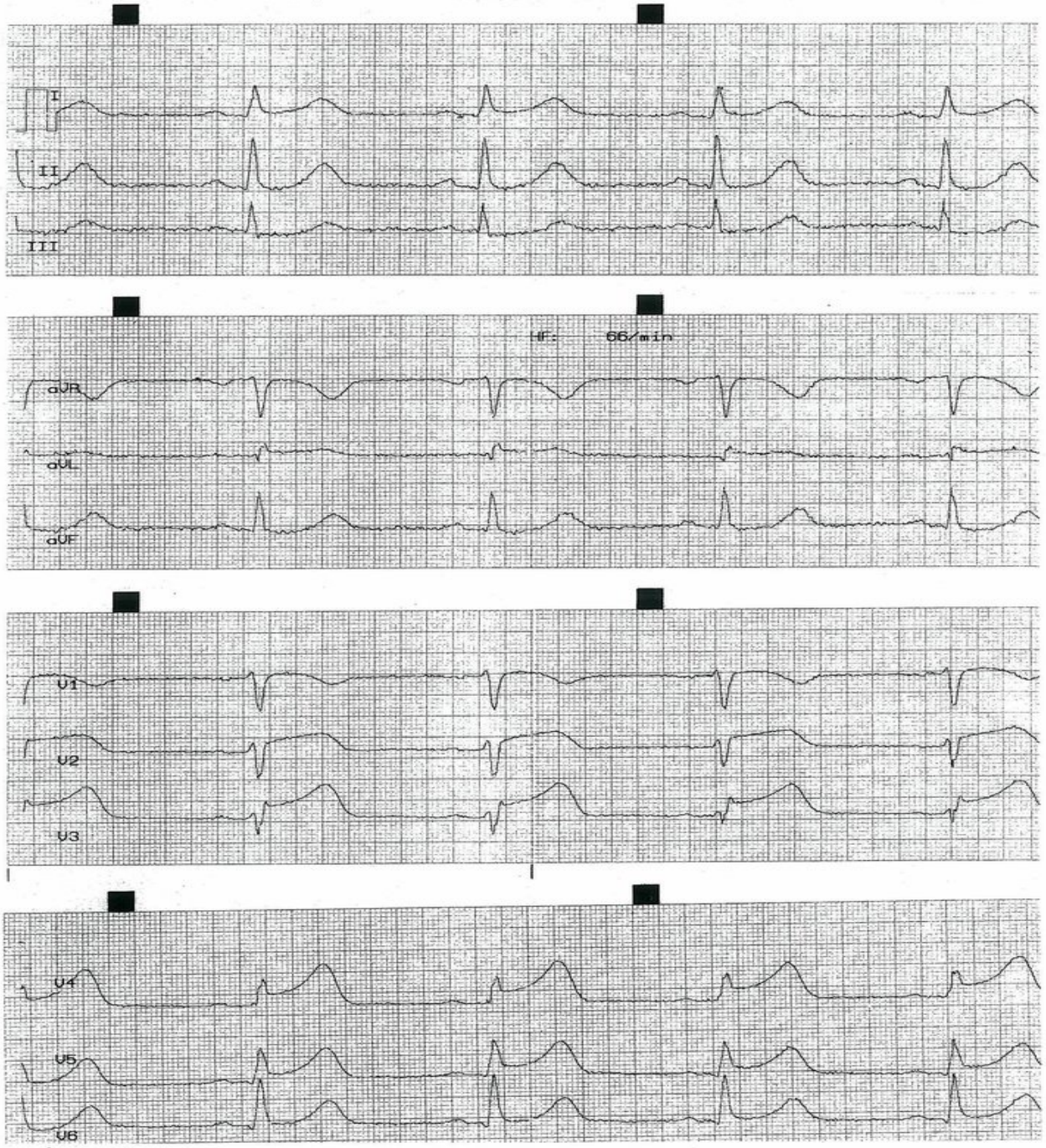

A
Apex
J point
Concave
B
Apex
J point
Non-concave



Acute anterior ST-elevation myocardial infarction (STEMI)

12-lead ECG (paper speed 25 mm/s)

- Sinus rhythm with a heart rate of ~90/min
- Left axis deviation (R > S in I, S > R in aVF)
- There are both ventricular (A) and supraventricular (B) extrasystoles.
- ST elevation in I, aVL and V1-V6. Note the pathological Q in V3.

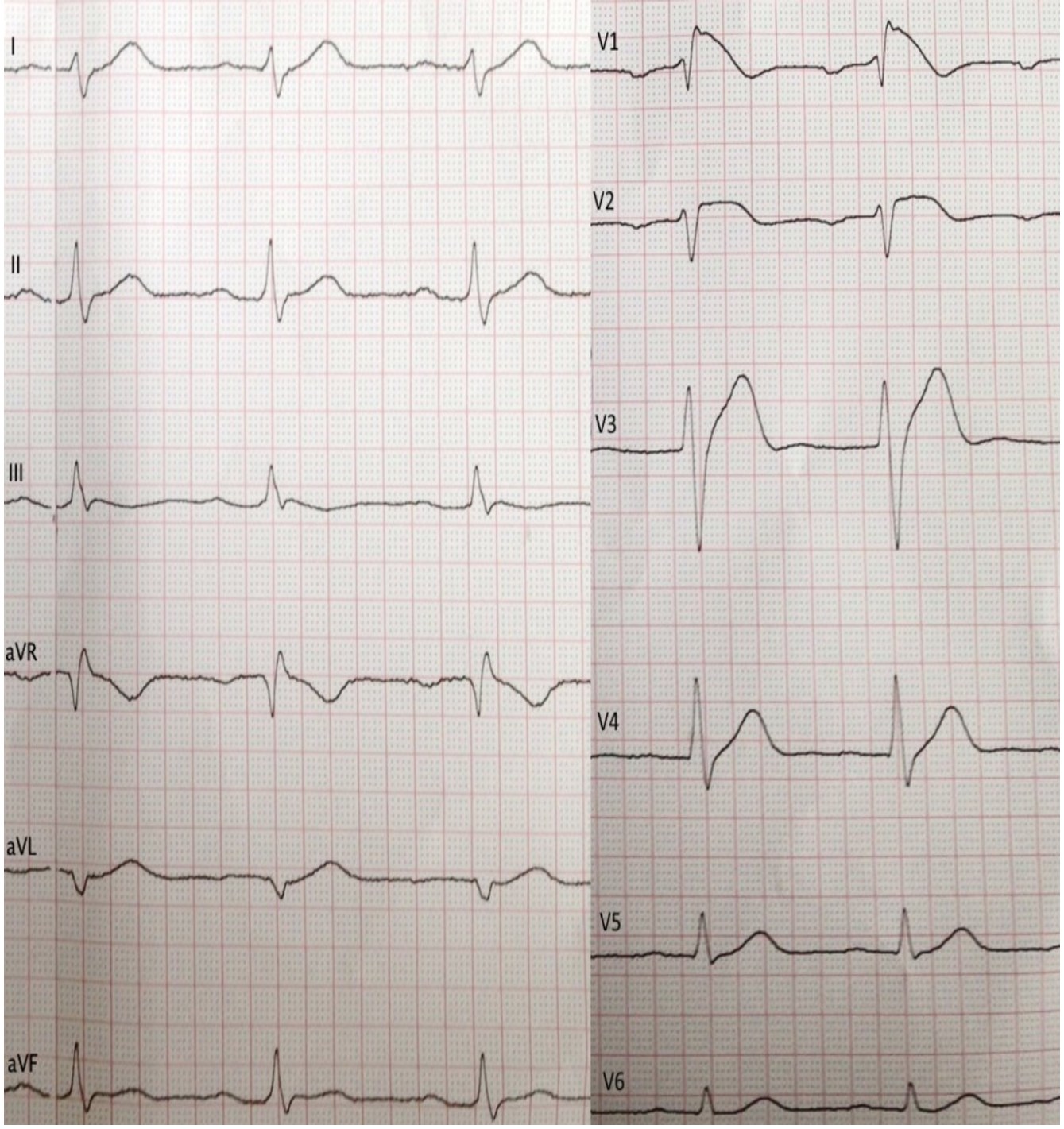


Acute anterior STEMI

12-lead ECG (paper speed: 50 mm/s)

Sinus rhythm with a heart rate of approx. 65/min.
Normal heart axis (R > S in I and aVF).
PQ interval normal (approx. 140 ms).
Elevation of the ST segment in V2–V6 (and, less distinctly, in I and aVL).

Diagnosis: acute anterior ST-elevation myocardial infarction



Brugada syndrome

12-lead ECG (paper speed: 50 mm/s)

Sinus rhythm with normal heart rate (approx. 83/min). Right axis deviation (lead I negative, leads II and III positive). PR interval = 0.16 s, coved-type ST segment, inverted T wave in V1 and V2.

Diagnosis: Brugada syndrome.

ST depression

Differential diagnosis

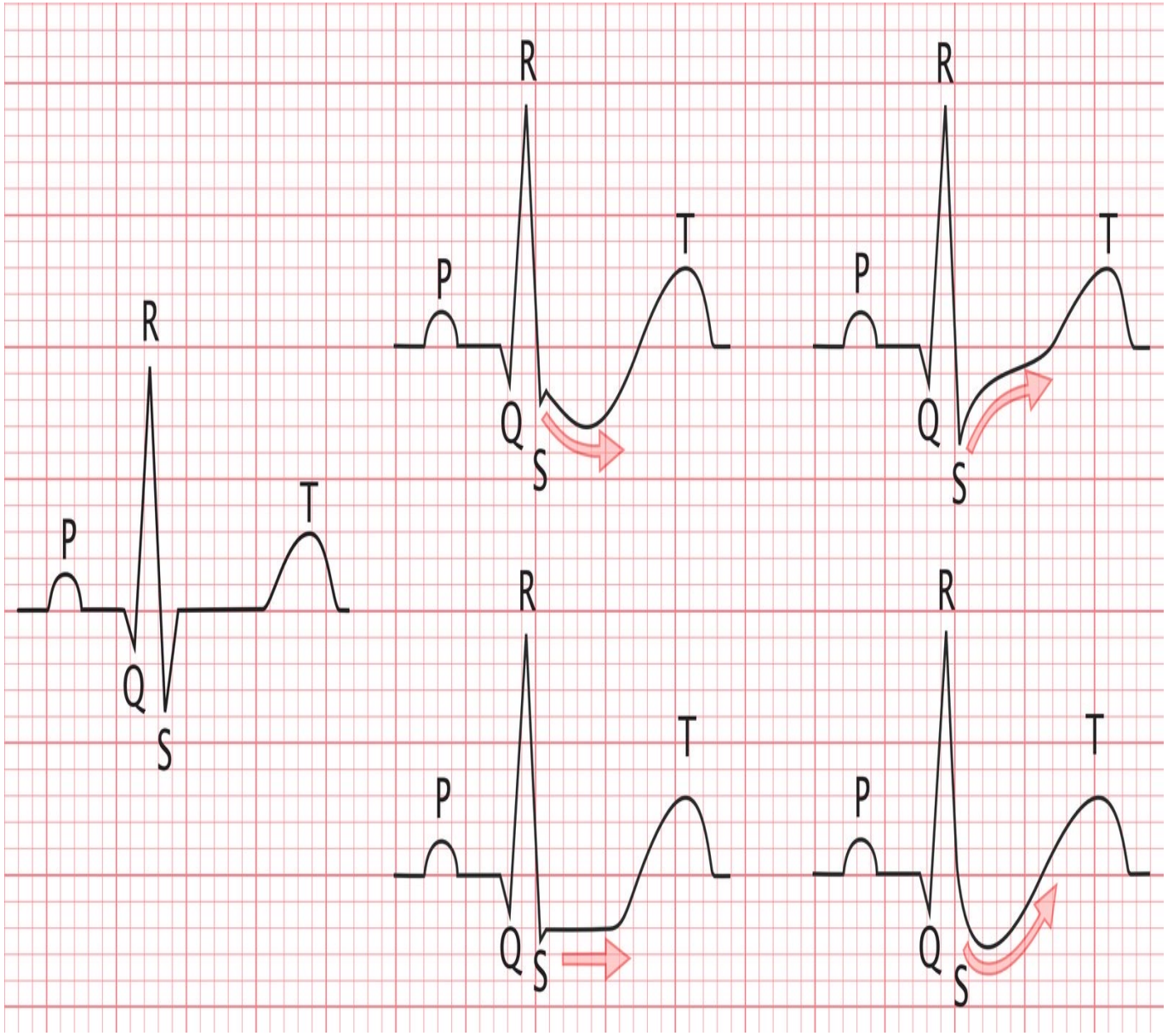
- Subendocardial myocardial ischemia (MI) (i.e., **NSTEMI**)
- Stress-induced MI (sign of coronary artery disease)
- Reciprocal change from MI
- Ventricular hypertrophy
  - Left ventricular hypertrophy: ST depression with pre-terminal T-wave inversion in V_{4-6}
  - Right ventricular hypertrophy: ST depression with pre-terminal T-wave inversion in $V_{1-3(4)}$
- **Digoxin effect**
- **Hypokalemia**
- **LBBB**

The shape of the ST segment suggests the etiology of the depression.

Downsloping ST depression or **horizontal ST depression**: myocardial ischemia

Upsloping ST depression: mild manifestations may be normal, but may also occur in cases of tachycardia; sign of coronary heart disease if significantly manifested

Sagging type ST-segment depression: characteristic of digoxin intake



Types of ST segment depression

Left: normal ECG. Top middle: downsloping ST depression. Bottom middle: horizontal ST depression. Top right: upsloping ST depression. Bottom right: sagging type ST-segment depression.

Progression of ST elevation myocardial infarction (STEMI) on ECG

The stages of myocardial ischemia are associated with characteristic (but variable) ECG findings:

1. **Hyperacute T waves**: very early and transient; usually have disappeared by the time ECG is performed
2. ST elevation at the J point: point at which the QRS complex completes and returns to the isoelectric line (i.e., the intersection of the S wave and the ST segment)
3. Progressive ST segment elevation, with added convexity
4. ST merges with T wave, forming a QRS-T segment (i.e., tombstone): usually with associated reciprocal ST depressions (see ST depression)
5. ST segment returns to isoelectric line, Q wave develops, and R wave loses amplitude
6. T-wave inversion
7. Progressive Q wave deepening and R wave shrinkage
8. T wave may or may not return to upright position

T wave

Physiological

The T wave represents the repolarization of the ventricles

The T wave is physiologically concordant to the QRS complex: positive if the QRS complex is positive or negative if the QRS complex is negative.

Pathological

- **T-wave inversion**
- Small T-wave inversions may be normal in the limb leads
- **Differential diagnosis**
  - Coronary heart disease
  - Ventricular hypertrophy
  - Perimyocarditis
  - Myocardial infarction (STEMI (in the intermediate stage) or NSTEMI)
  - Ventricular aneurysm
  - Intracranial hemorrhage
  - **LBBB**
  - Acid/base disturbance

- The shape of the T wave may help to narrow the differential diagnosis.**Pre-terminal T-wave inversion**: If the T wave is bisected, it points to the left. It may occur in:
  - Perimyocarditis
  - Ventricular hypertrophy
  - Coronary heart disease
- **Terminal T-wave inversion**: If the T wave is bisected, it points either to the right or upwards. It may occur in:
  - Intracranial hemorrhage
  - Perimyocarditis
  - A persistent negative T wave following myocardial infarction may suggest an aneurysm.
  - Myocardial infarction (STEMI (in the intermediate stage) or NSTEMI)

Peaked T wave

Tall, narrow, symmetrically-peaked

Differential diagnosis

- **Hyperkalemia**
- Hypermagnesemia
- High vagal tone

Hyperacute T wave

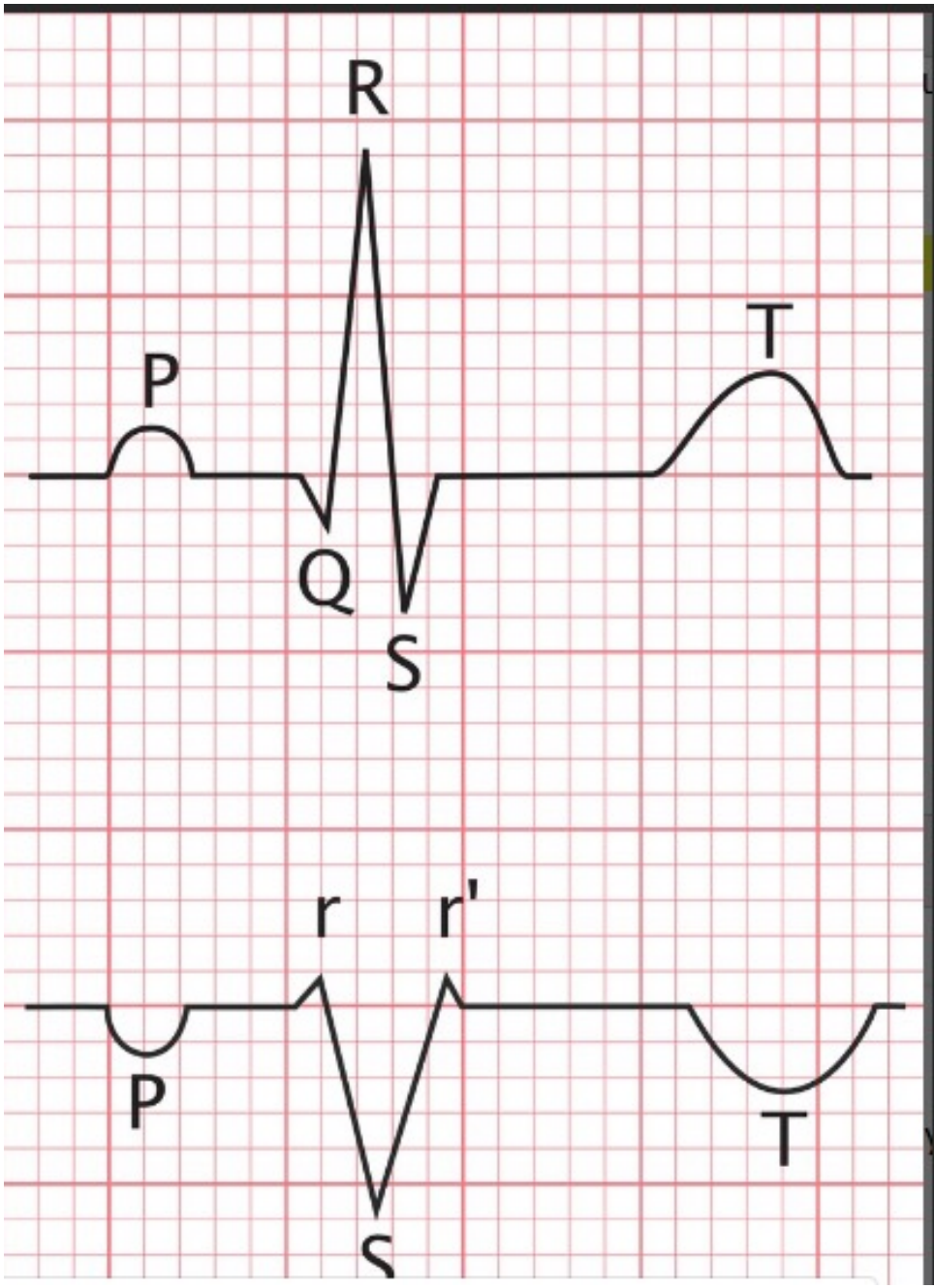
Broad, asymmetrically-peaked

Differential diagnosis

- Early stages of ST (segment) elevation myocardial infarction (**STEMI**)
- Prinzmetal's angina

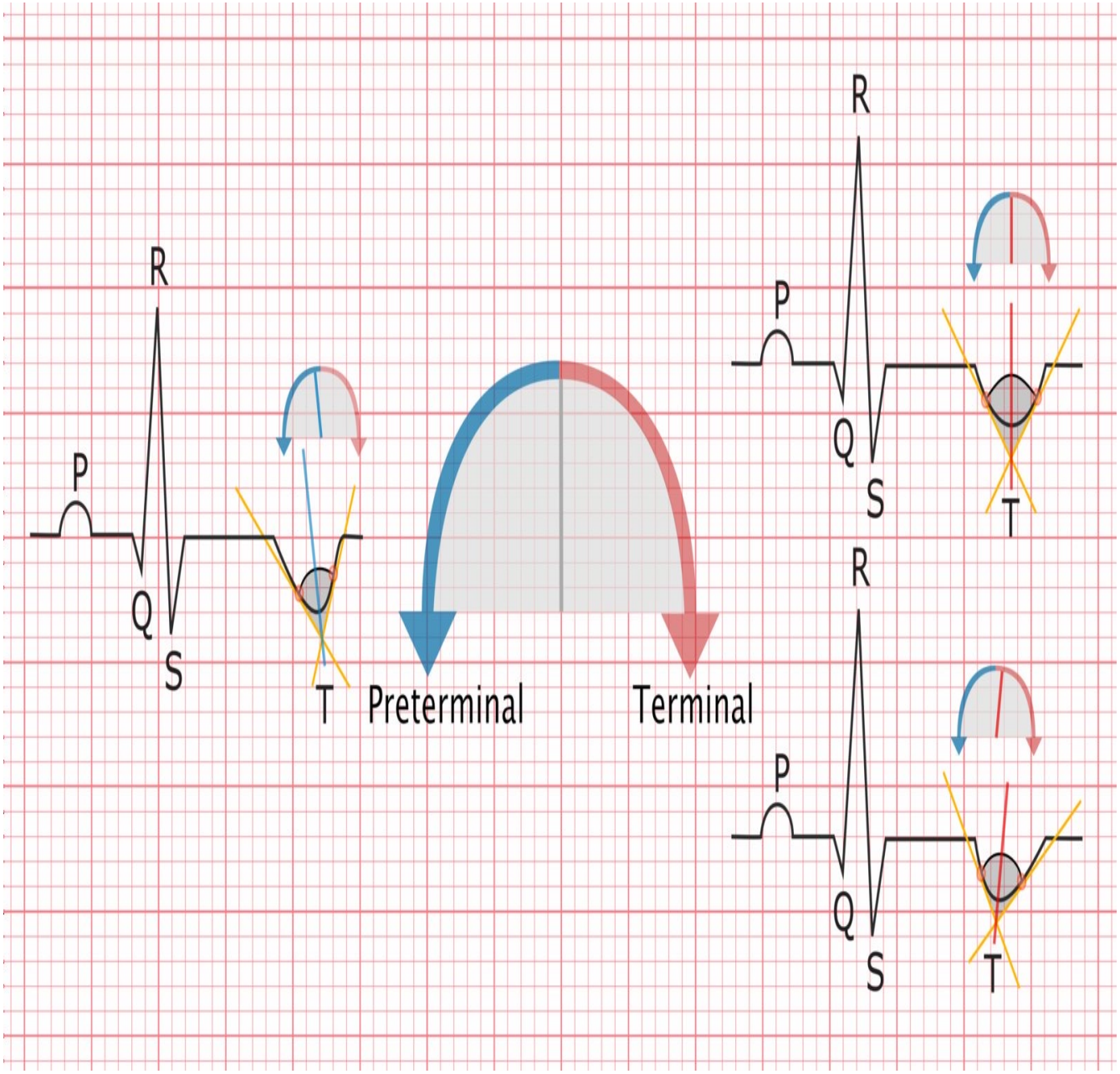
Normally, if electric conduction in the heart is pathological (bundle branch block), repolarization is also disturbed → reliable evaluation of the ST segment or T wave is not possible!

New occurrence of a left bundle branch block associated with angina chest pain is defined as a STEMI!



Concordant negative T wave

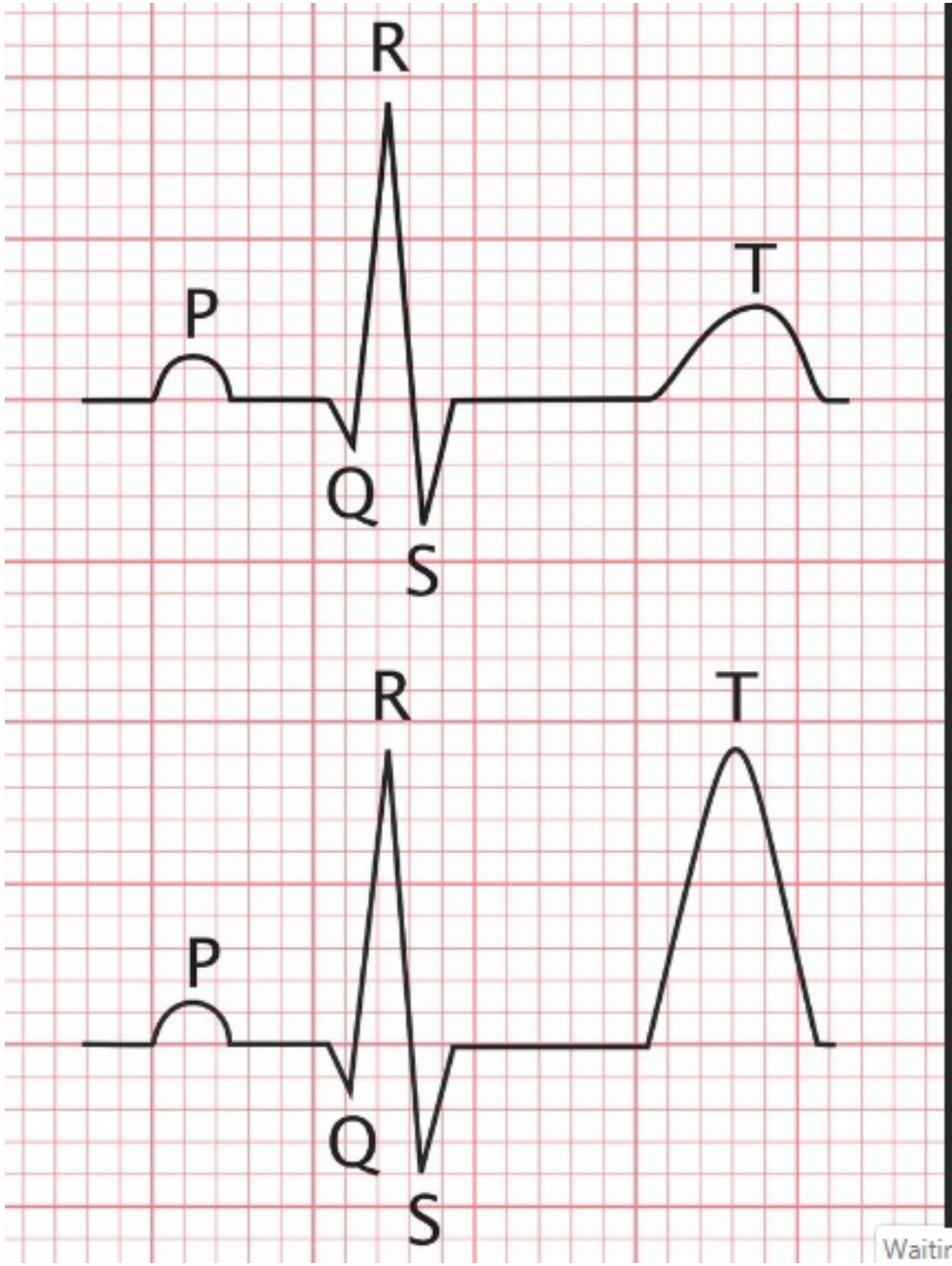
Top: Normal ECG with an overall positive QRS complex and corresponding positive T wave. Bottom: Negative T wave in association with a negative QRS complex, which is seen here, is often a normal finding in AVR and V1.



Types of T-wave inversion

To distinguish between terminal and preterminal T-wave inversion, first apply the tangents (yellow lines) to the legs of the T wave. Next, draw a line bisecting the inner angle (blue/red lines).

The upper end of the bisecting line, which points towards the R wave, indicates a preterminal T-wave inversion. If the bisecting line is perpendicular to the isoelectric line or if its upper end points away from the R wave, this indicates a terminal T-wave inversion.



Peaked T wave

Top: normal ECG

Bottom: significantly elevated, "tent-shaped" T wave. Physiological in patients with a high vagal tone; pathological in, e.g., hyperkalemia or may be an early sign of myocardial infarction.

QT interval

Physiological

- Measured from the beginning of the Q wave to the end of the T wave
- Represents the entire duration of ventricular depolarization
- Varies with heart rate, so correction for the heart rate is necessary (=**QTc**)
  - QTc normally **< 350–440 ms**

Pathological

Prolongation of the QT interval

- Possible differential diagnoses include:
- Hypocalcemia
- Hypokalemia
- Inflammatory heart diseases (myocarditis, pericarditis)
- Bundle branch block
- High vagal tone
- Rare congenital syndromes (e.g., **congenital long QT syndromes** such as Romano-Ward syndrome)
- **Acquired long QT syndrome**
- Hypothyroidism
- **Drug side effect** (e.g., antiarrhythmic agents, **antidepressants**, **phenothiazines**, 1st-generation antihistamines)

Shortening of the QT interval

Possible differential diagnoses include:

- Hypercalcemia
- Hyperkalemia
- **Digoxin effect**
- Increased sympathetic tone
  (e.g., hyperthyroidism or fever)