Basics of ECG

A Systematic Approach that never fails



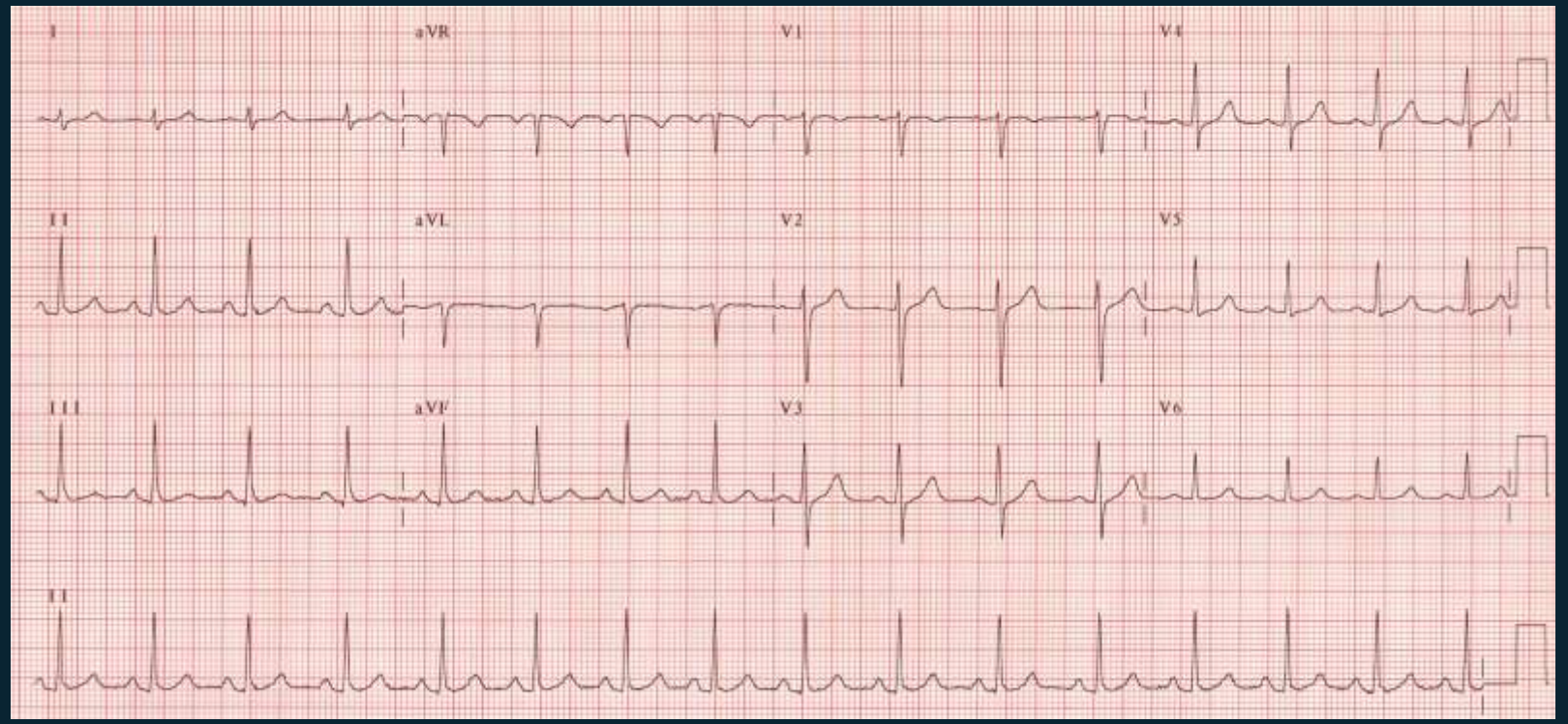
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Medical Superintendent
Rawalpindi Institute of Cardiology

OPENING CHALLENGE

What can you say in 20 seconds?

Describe the tracing before naming a diagnosis.



Rate? Rhythm? QRS width? ST–T changes?

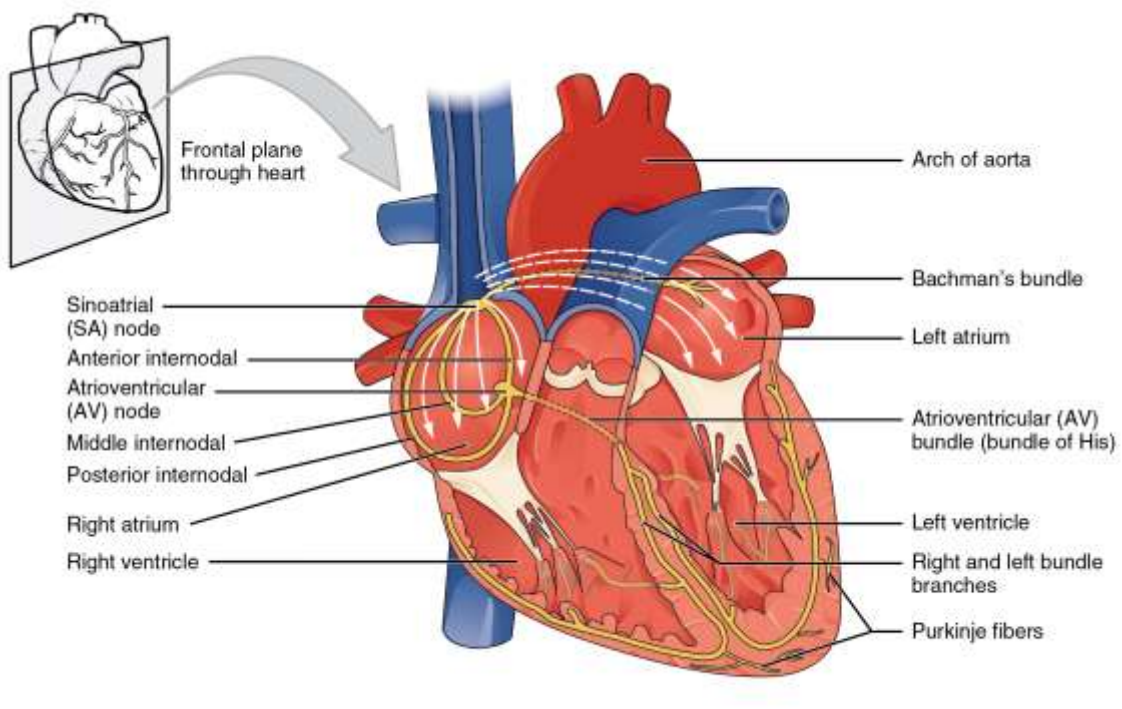
Six questions for every ECG

A repeatable reading sequence reduces omissions.

Step	Question
01 Patient	Is the patient stable?
02 Paper	Is this a trustworthy recording?
03 Rhythm	What are the rate and P–QRS relationship?
04 Conduction	What are the axis, PR, QRS and QTc?
05 ST–T	Are there regional or dynamic changes?
06 Summary	What matters, and what happens next?

The electrical journey

The surface ECG records the net electrical activity of the heart.



Sinus node

The usual impulse begins in the right atrium.

AV node

A delay allows atrial activation to finish.

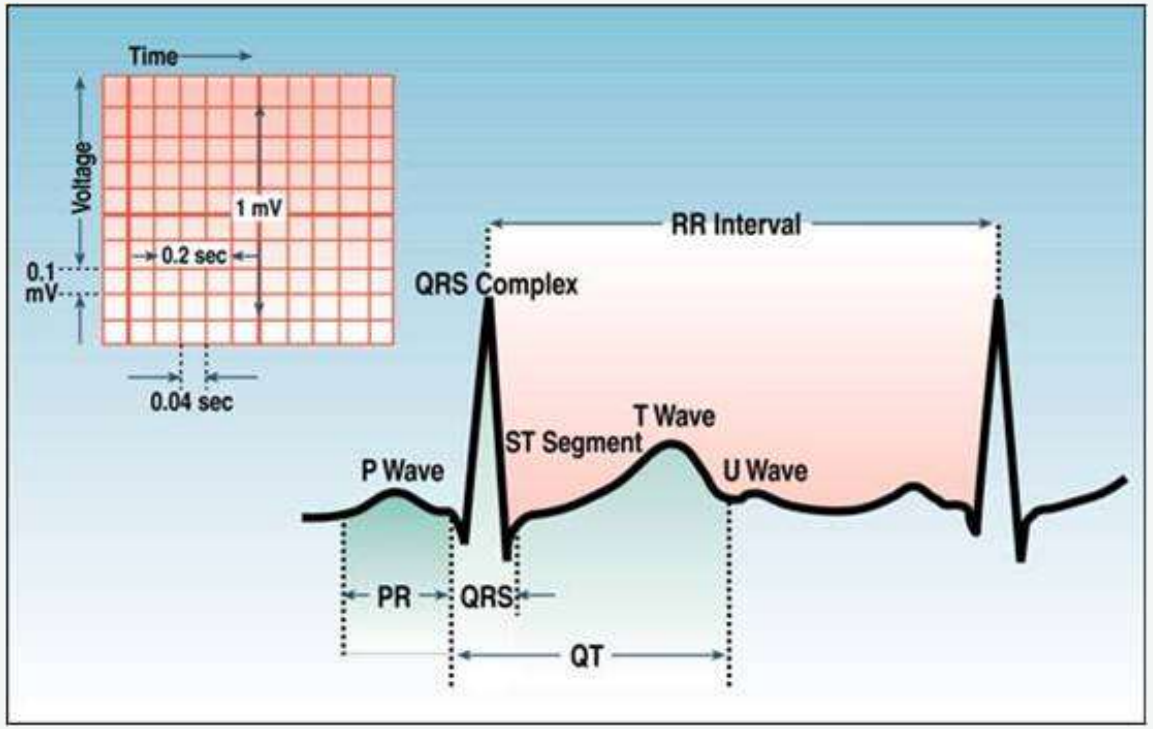
His–Purkinje system

Rapid activation spreads through the ventricles.

The conduction system makes ventricular activation fast.

OpenStax, Anatomy and Physiology, Figure 19.18

One beat, three main events



P wave

Atrial depolarisation

QRS complex

Ventricular depolarisation

T wave

Ventricular repolarisation

P = atrial activation. QRS = ventricular activation. T = recovery.

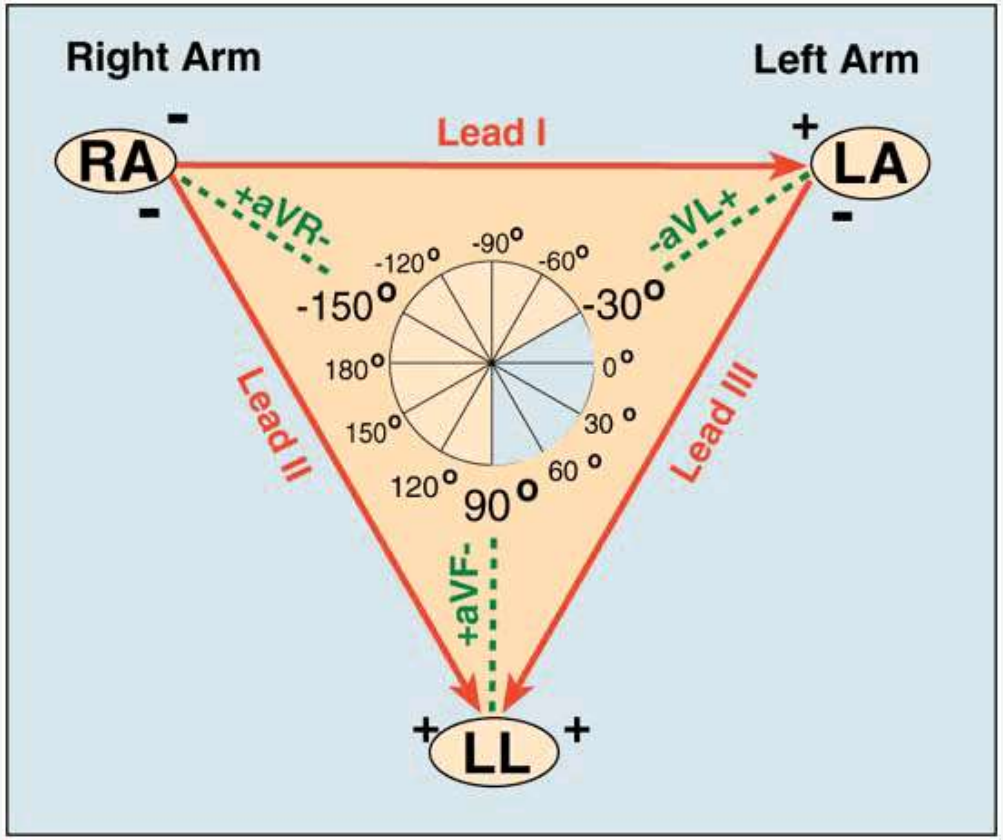
Direction determines the deflection

For a depolarisation wave, think about the positive electrode.

Wave direction	Usual appearance
Towards the positive electrode	Predominantly upward
Away from the positive electrode	Predominantly downward
Approximately perpendicular	Small or biphasic

A different lead sees the same electrical event from a different angle.

10 electrodes create 12 views



Six limb leads

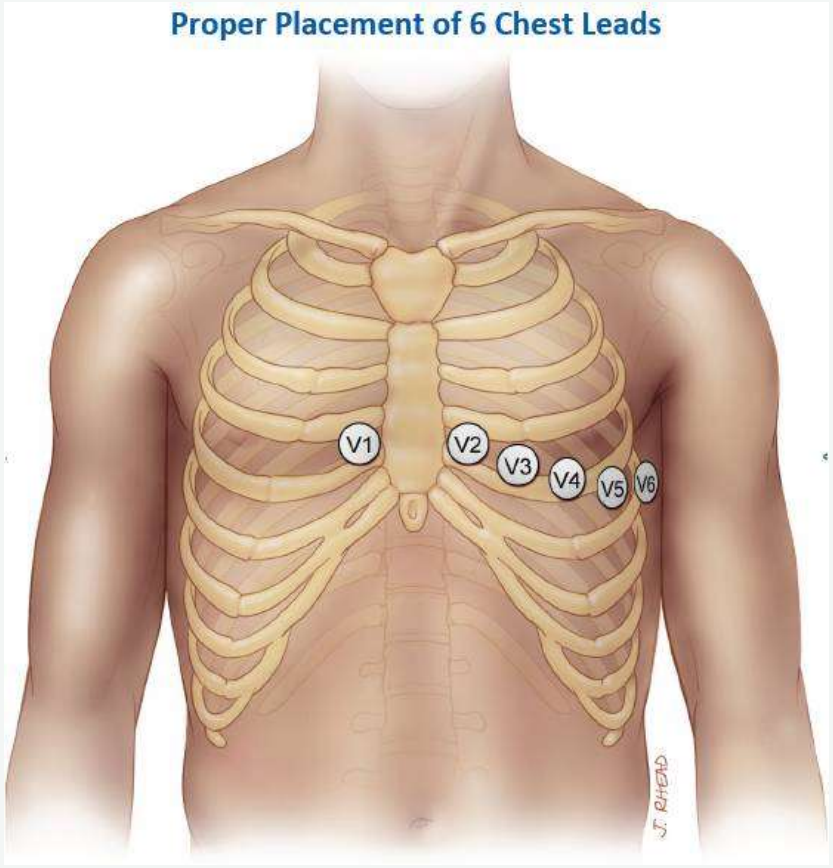
I, II, III, aVR, aVL, aVF
View the frontal plane.

Six chest leads

V1–V6
View the horizontal plane.

An electrode touches the skin. A lead is a voltage view.

Chest electrodes follow landmarks



Lead	Position
V1 / V2	4th intercostal space Right / left sternal border
V4	5th intercostal space Left midclavicular line
V3	Halfway between V2 and V4
V5 / V6	Anterior / midaxillary line Horizontal with V4

V4 before V3. V4, V5 and V6 share a horizontal level.

Neighbouring leads form territories

A regional pattern carries more meaning than one isolated lead.

Region	Lead group
Inferior	II, III, aVF
Lateral	I, aVL, V5, V6
Anterior / septal	V1–V4

These are useful regional groupings, not a one-lead–one-artery map.

Calibration comes before calculation

25 mm/s

10 mm/mV

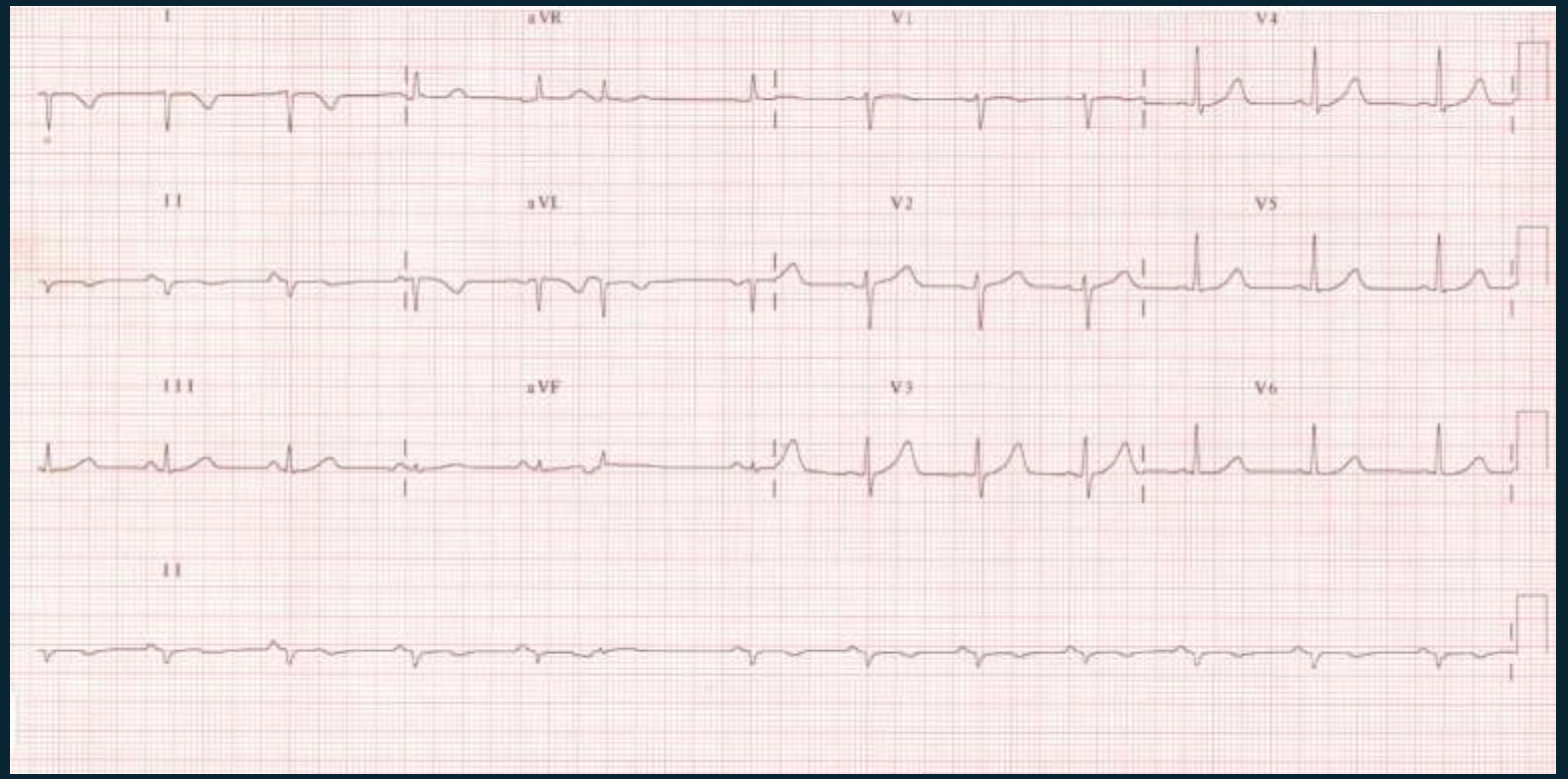
At standard settings	One small box	One large box
Horizontal time	40 ms	200 ms
Vertical voltage	0.1 mV	0.5 mV

A change in speed or gain changes what the boxes mean.

CASE 1

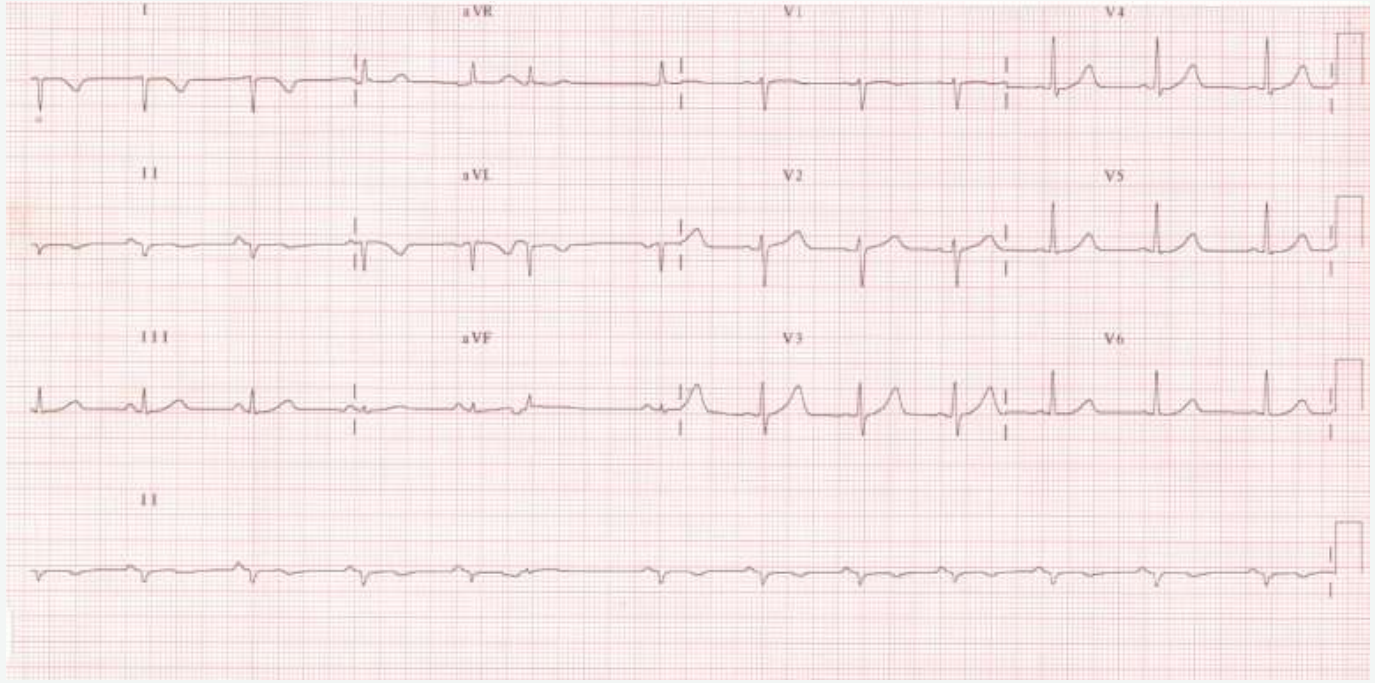
Why does lead I look upside down?

Which technical problem would you check first?



Compare lead I, aVR and the chest leads.

Right-arm / left-arm reversal



Limb-lead clues

Inverted lead I
Unexpectedly positive aVR

Chest-lead clue

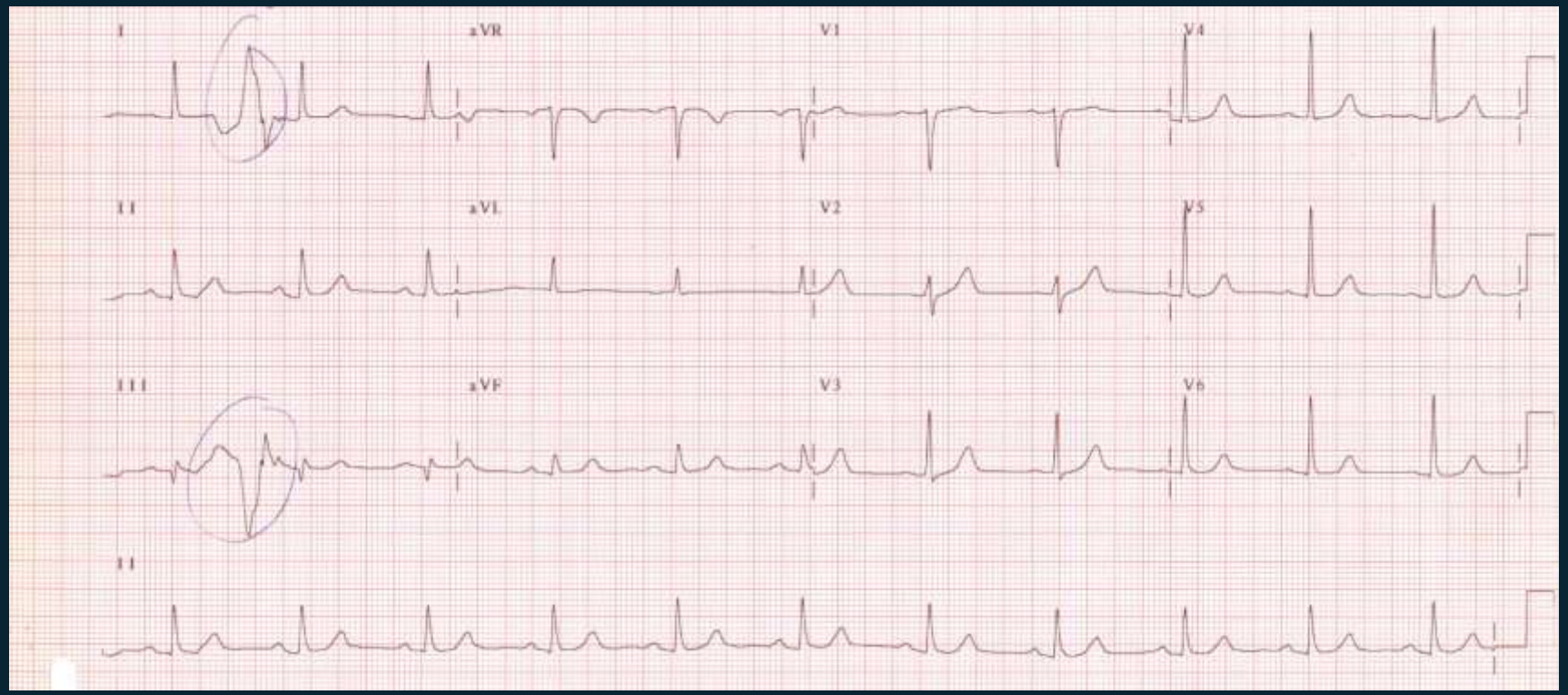
Preserved R-wave progression

Electrode check and repeat recording come before a new diagnosis.

CASE 2

Is every large deflection a heartbeat?

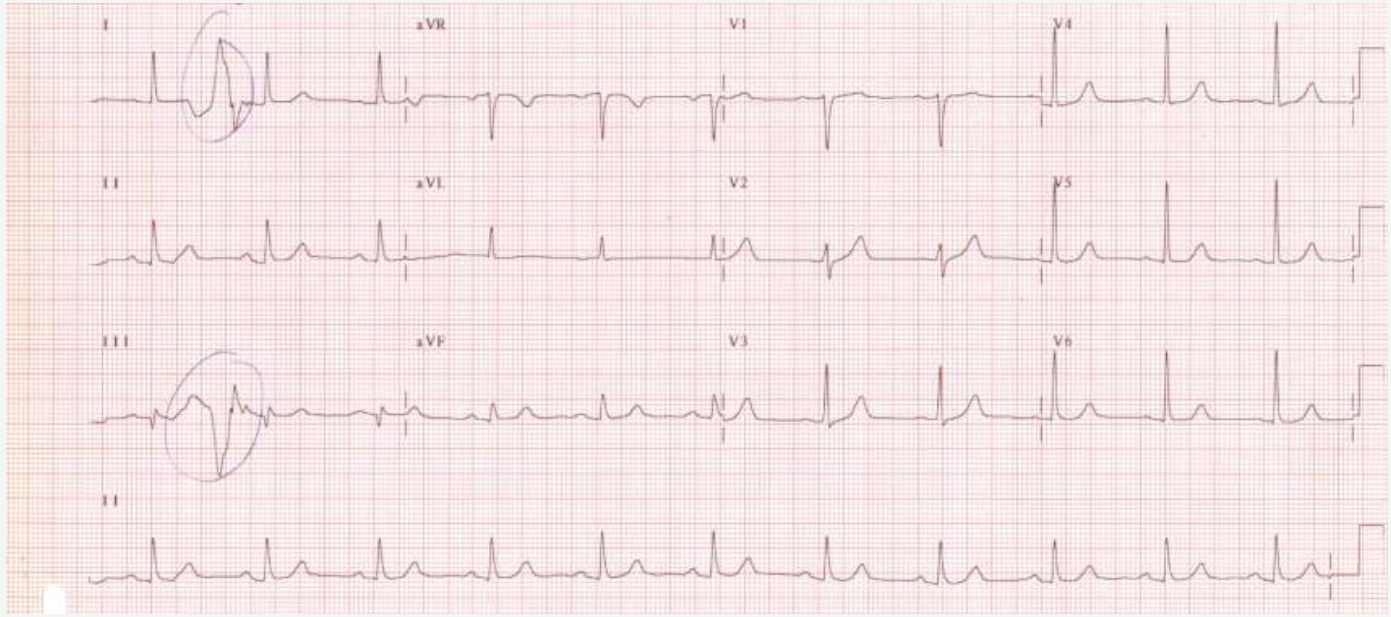
Look across the simultaneously recorded limb leads.



Which lead gives you the clearest underlying rhythm?

CASE 2 EXPLAINED

Artefact can imitate arrhythmia



Electrical consistency

Does the rhythm agree across leads?

Clinical consistency

Does the pulse match the tracing?

Repeat recording

Correct movement or electrode contact.

Patient assessment and trace verification belong together.

Rate depends on the rhythm

Use these methods at 25 mm/s.

REGULAR

300 ÷ large boxes

Between two consecutive R waves

IRREGULAR

10-second count × 6

Count QRS complexes in the labelled 10-second rhythm strip

Large boxes	1	2	3	4	5	6
Rate / min	300	150	100	75	60	50

Count a labelled time interval, not all the beats on the page.

Sinus rhythm begins with the P wave



Sinus P waves

Consistent shape, usually upright in I and II

Usual AV relationship

A P before each QRS and a QRS after each P

P-wave origin and AV conduction are separate questions.

Four questions organise tachycardia reading

First impression	Narrow QRS <120 ms	Broad QRS ≥120 ms
Regular	Sinus tachycardia SVT or flutter with fixed block	VT SVT with aberrancy or pre-excitation
Irregular	AF Variable block or ectopic beats	AF with aberrancy / pre-excitation Polymorphic VT or ectopic beats

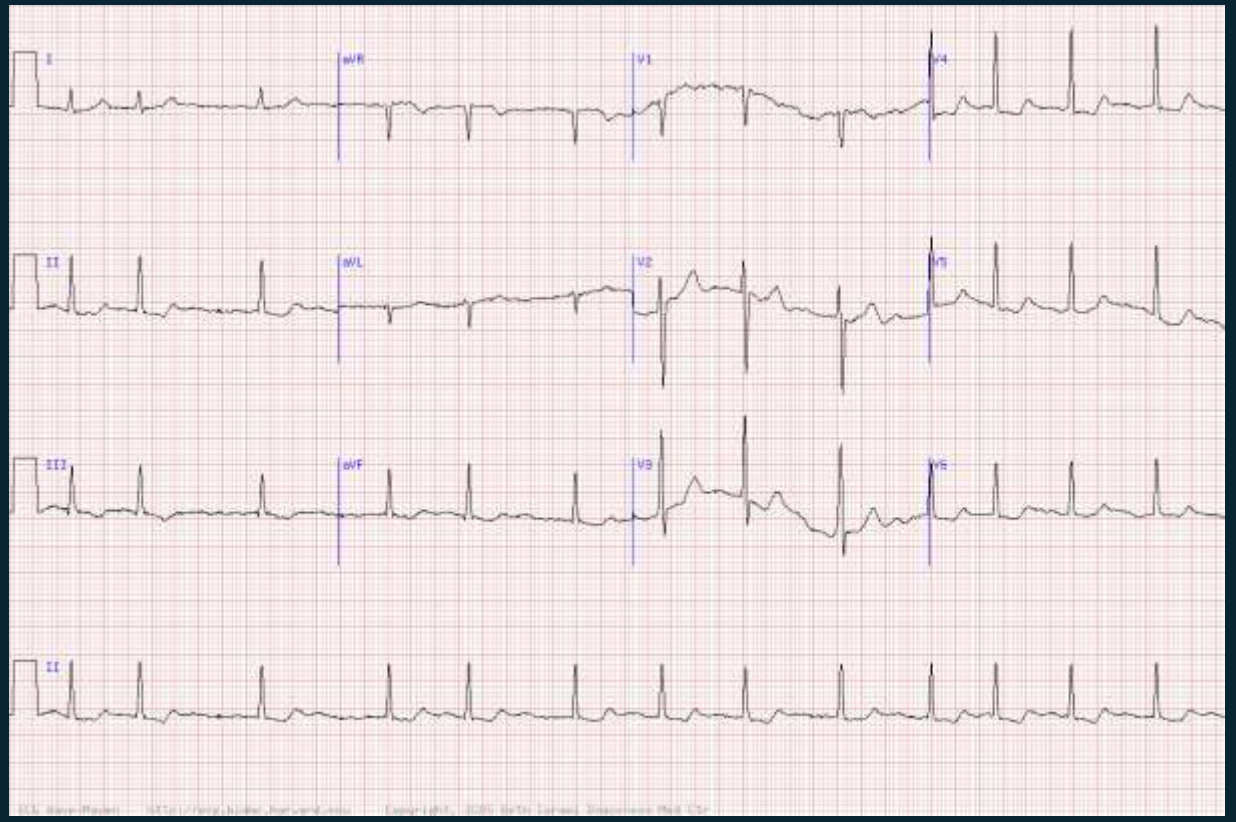
Then ask: Where are the P waves? How do they relate to QRS?

The category narrows the possibilities. P waves refine the diagnosis.

CASE 3

What explains this irregular rhythm?

Assess the R–R intervals and look for organised P waves.



Two observations before one diagnosis.

Atrial fibrillation



Irregular R–R intervals

No predictable repeating pattern

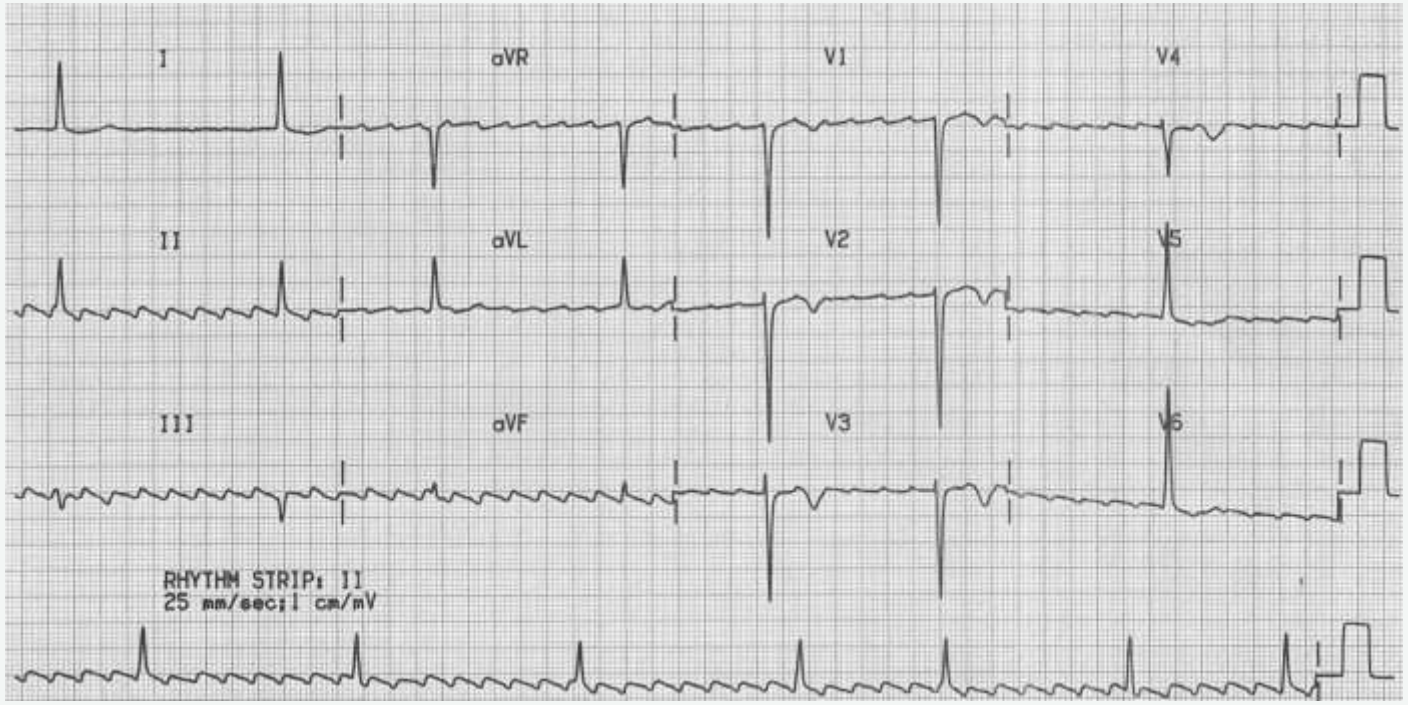
No organised P waves

Confirm the pattern across an adequate recording

AF can be slow, controlled or fast.

Flutter has organised atrial activity

The atria keep a regular rhythm even when ventricular conduction varies.

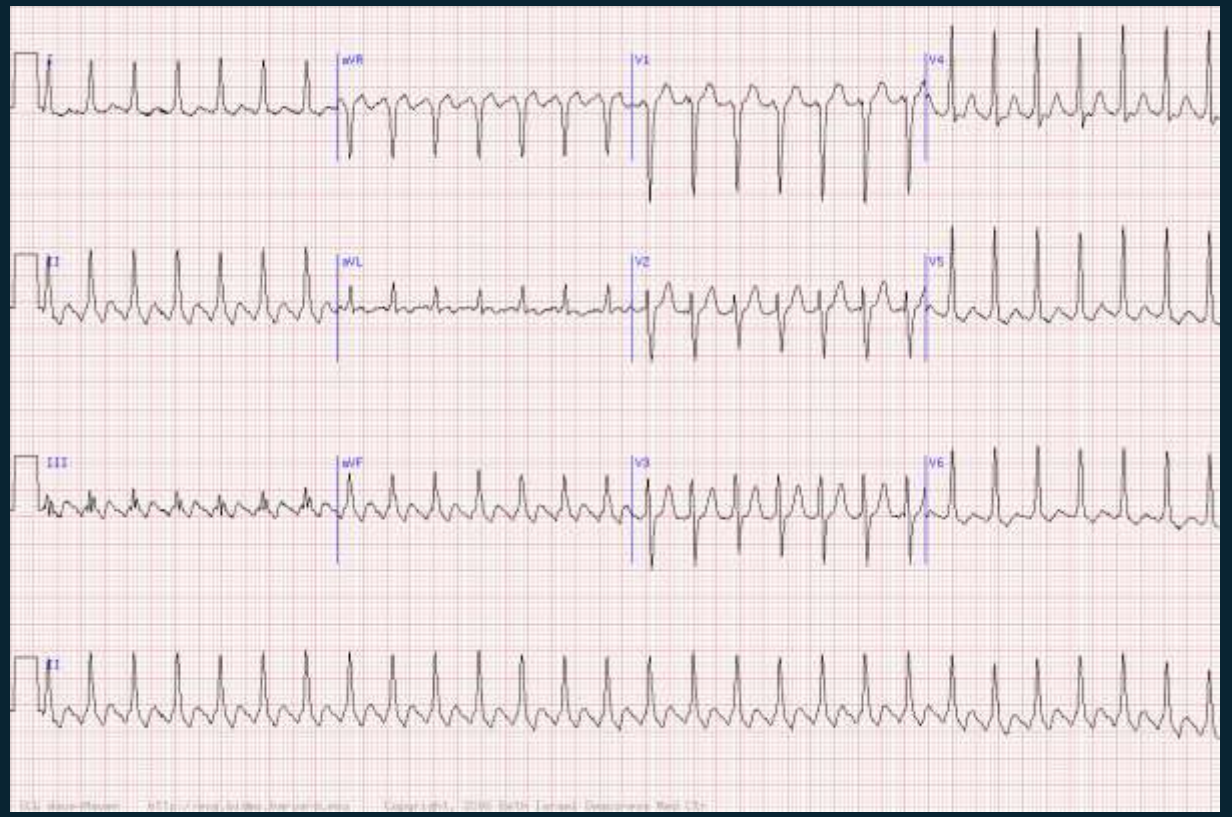


Atrial rate and ventricular rate are different measurements.

QUICK CHALLENGE

A regular tachycardia can hide flutter

What atrial activity is visible in the inferior leads and V1?



With 2:1 conduction, the ventricular rate is half the atrial rate.

Axis is the net QRS direction

Use the overall QRS polarity in I and aVF, then lead II if needed.

Lead I	aVF	Interpretation
Positive	Positive	0° to +90°: normal quadrant
Positive	Negative	II positive: normal II negative: left axis deviation
Negative	Positive	Right axis deviation
Negative	Negative	Extreme axis

I positive + aVF negative? Lead II prevents a common mistake.

Intervals measure conduction and recovery

Interval	What it includes	Adult guide
PR	P onset to QRS onset	120–200 ms
QRS	Ventricular activation	Usually <110 ms ≥120 ms is broad
QT / QTc	Ventricular activation and recovery	QT varies with rate Assess the corrected QT

PR starts at P onset. QT starts at QRS onset.

QT needs a rate correction

QTc

Activation and recovery, adjusted for heart rate

Measure carefully

QRS onset to the end of T

Check the correction

Bazett can mislead at rate extremes

Look for reversible causes

Medicines, potassium and magnesium

QTc above 500 ms is a warning that deserves prompt review.

AV block is a P–QRS relationship

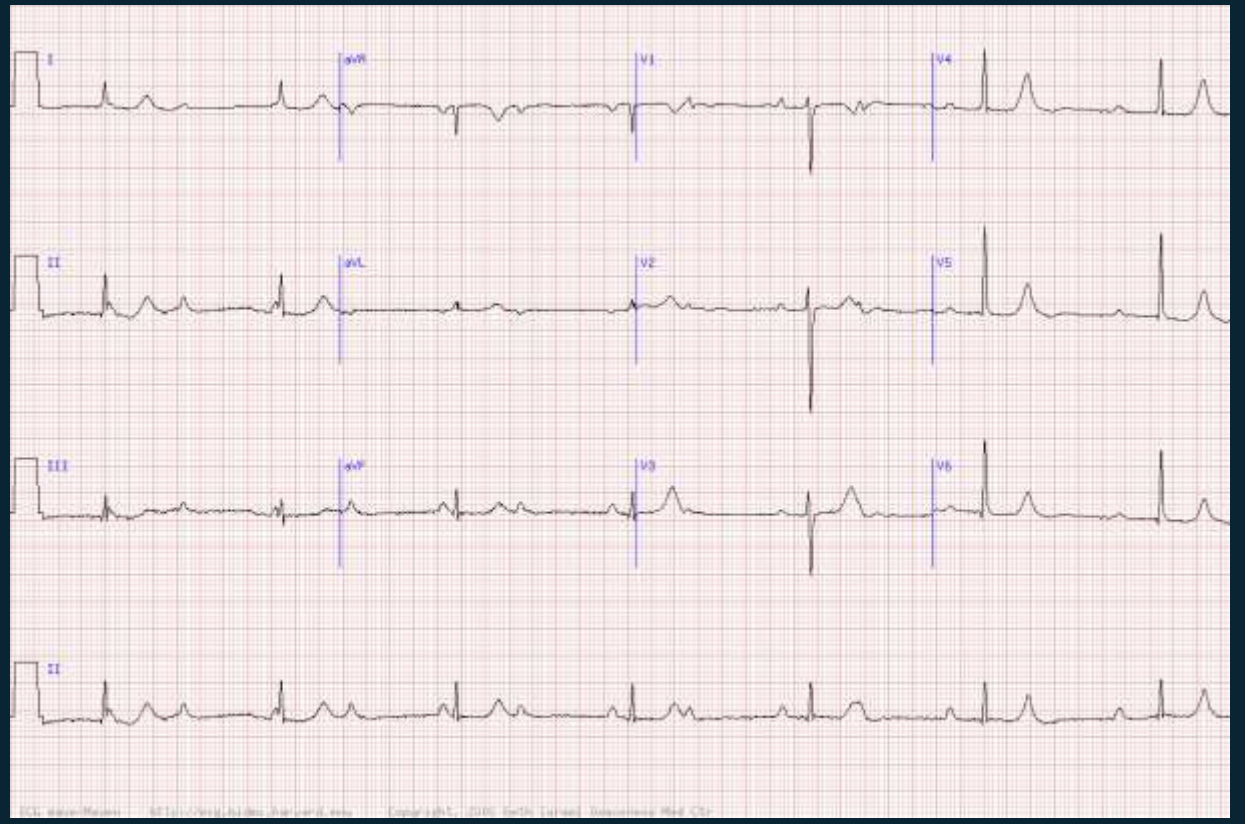
Pattern	What to look for
First degree	Every P conducts, with PR >200 ms
Mobitz I	PR classically lengthens before a blocked P
Mobitz II	Intermittent blocked P with stable conducted PR
Complete block	Atrial and ventricular rhythms are independent

A 2:1 pattern alone does not distinguish Mobitz I from Mobitz II.

CASE 4 · TEACHING SCENARIO

A slow rhythm after syncope

Are the P waves and QRS complexes following the same clock?



Trace the P waves first. Then trace the QRS complexes.

CASE 4 EXPLAINED

Complete AV block: two independent clocks



ATRIAL CLOCK

Regular P waves
Faster rate

VENTRICULAR CLOCK

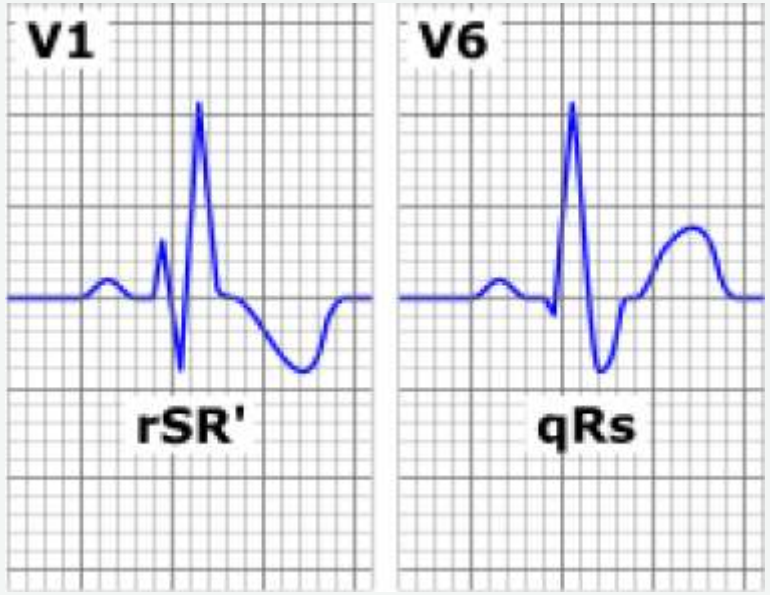
Regular escape rhythm
Slower rate

Syncope with complete block needs urgent monitored assessment.

Bundle branch block changes the route

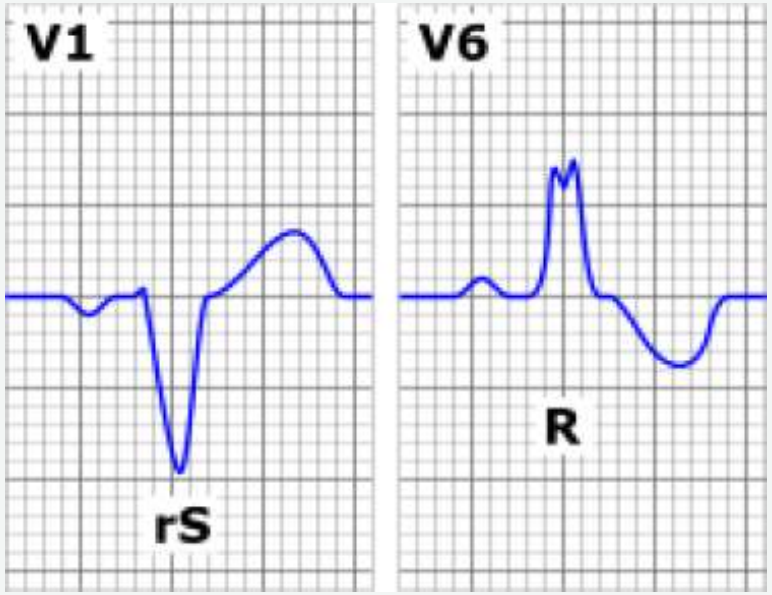
Complete block requires QRS ≥120 ms and the appropriate morphology.

RBBB



Terminal R′ in V1, broad S in I / V6

LBBB

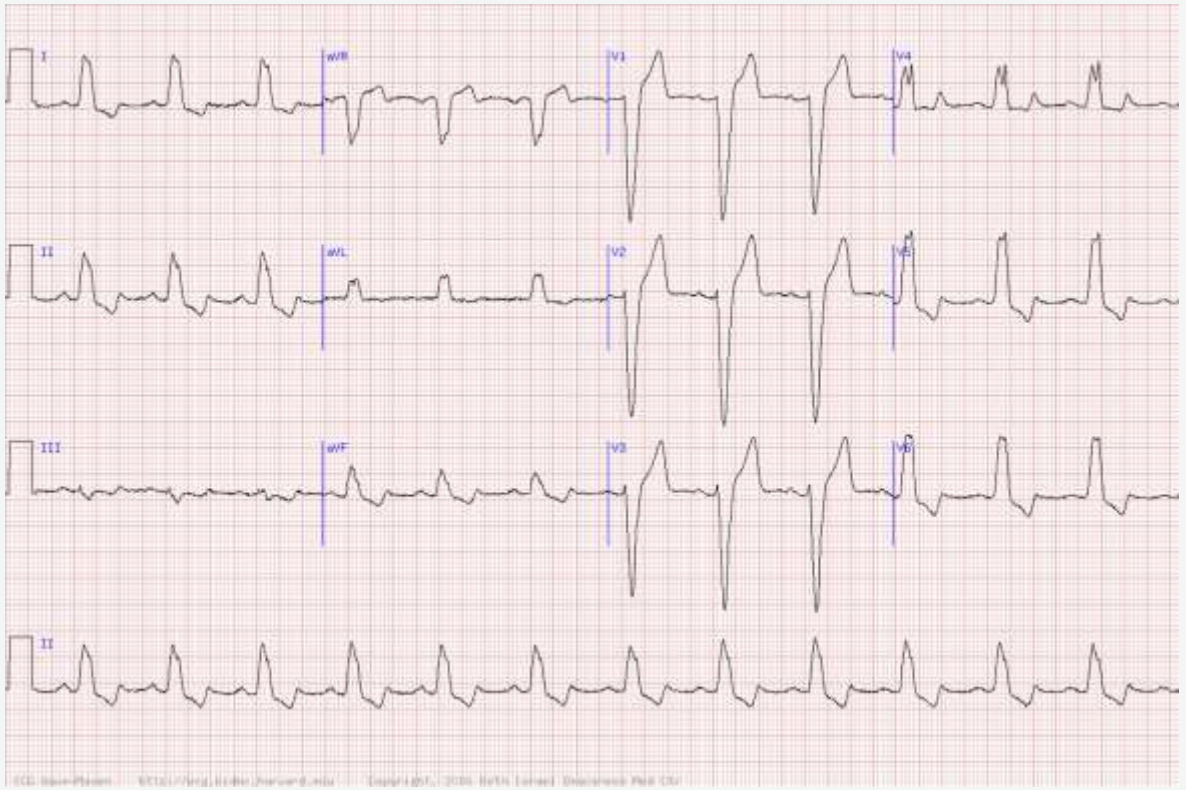


Negative V1, broad lateral R

V1 and the lateral leads explain which ventricle activates late.

LBBB also changes repolarisation

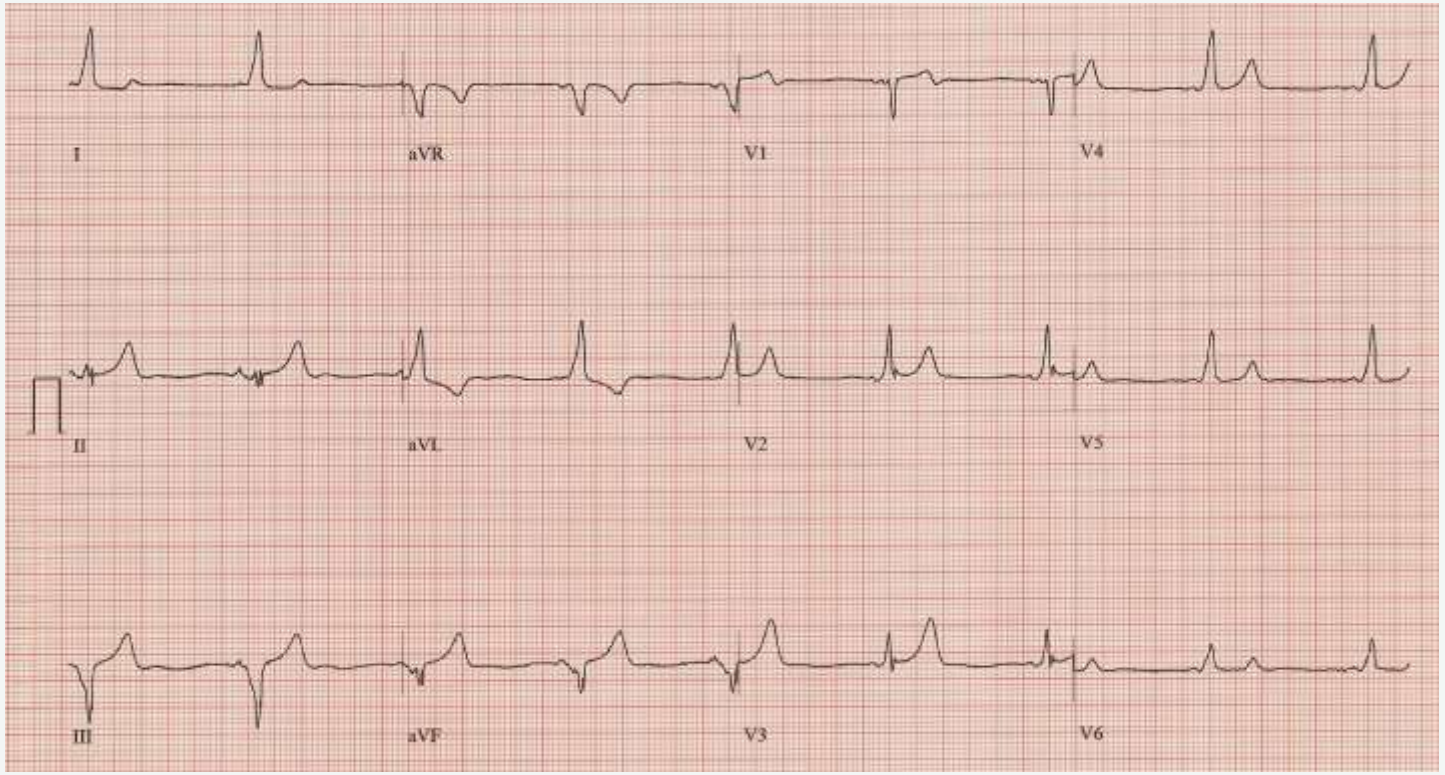
The ST segment and T wave often oppose the main QRS direction.



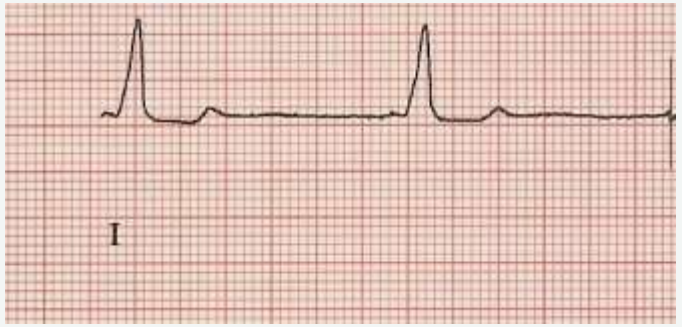
LBBB complicates ischaemia assessment. Clinical context still matters.

Pre-excitation gives the ventricle an early start

Short PR, a delta wave and QRS widening form the classic pattern.



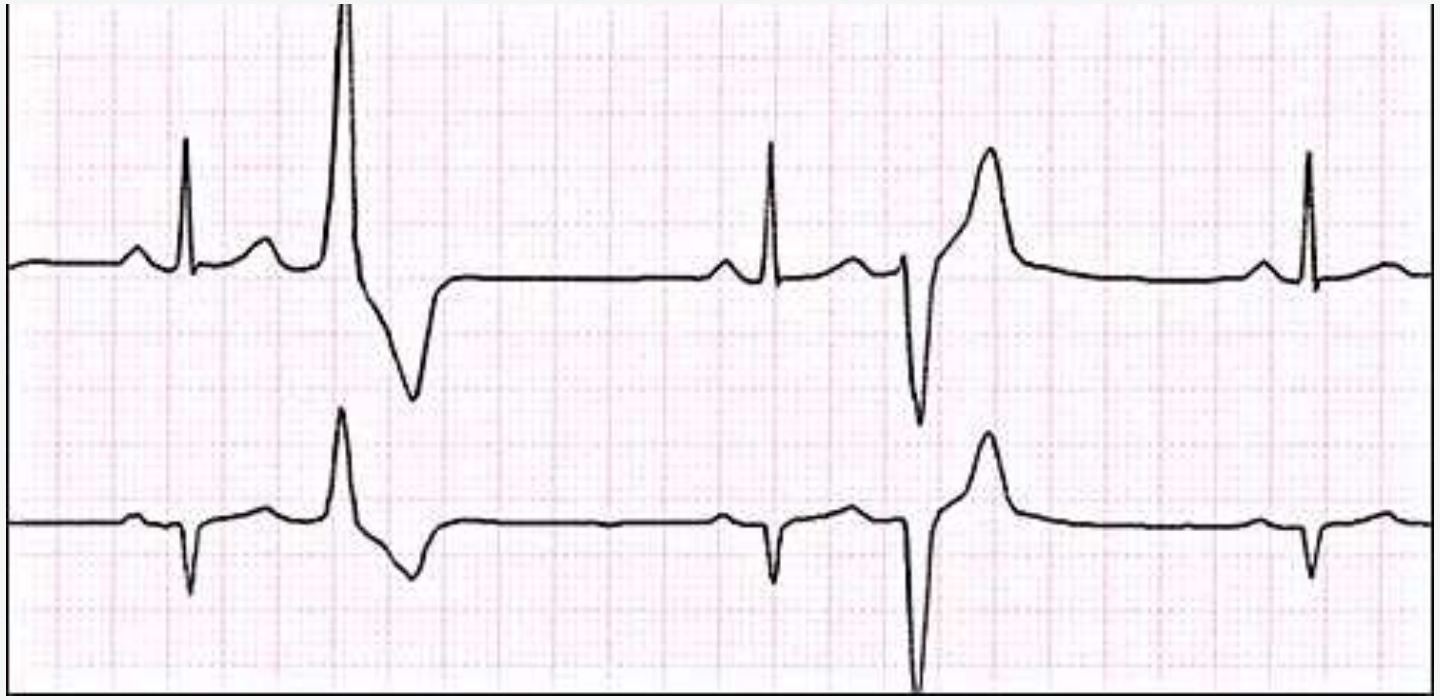
Lead I close-up



Short PR
Slurred QRS onset
(the delta wave)

WPW syndrome = pre-excitation + symptomatic tachyarrhythmia.

A premature beat interrupts the pattern



Timing

The beat arrives early.

Morphology

A broad abnormal QRS suggests a PVC.

Context

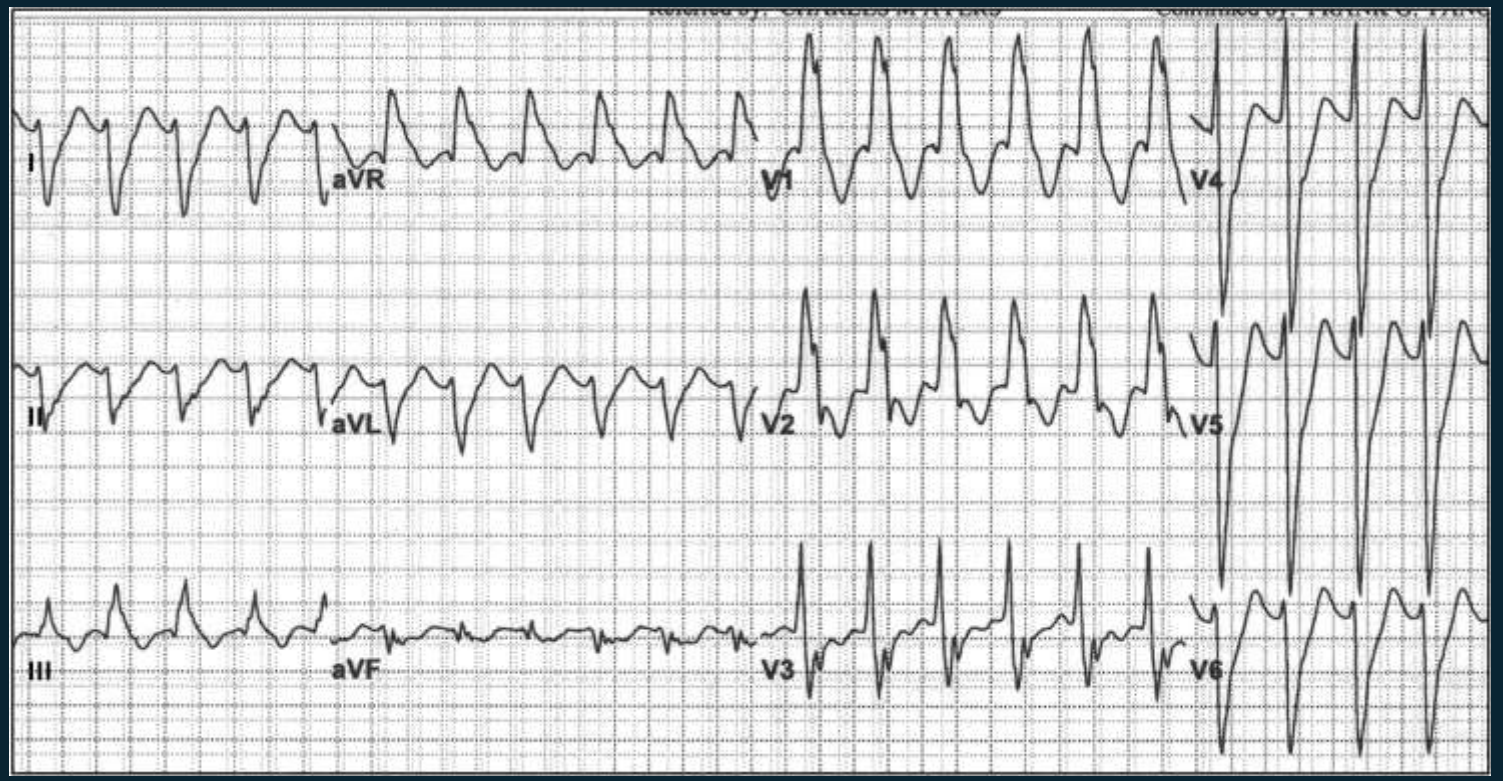
Symptoms and burden guide evaluation.

Describe timing and morphology before judging clinical significance.

CASE 5

A regular broad-complex tachycardia

What is your working diagnosis while help arrives?



Assume VT unless a secure alternative is established.

The patient determines urgency

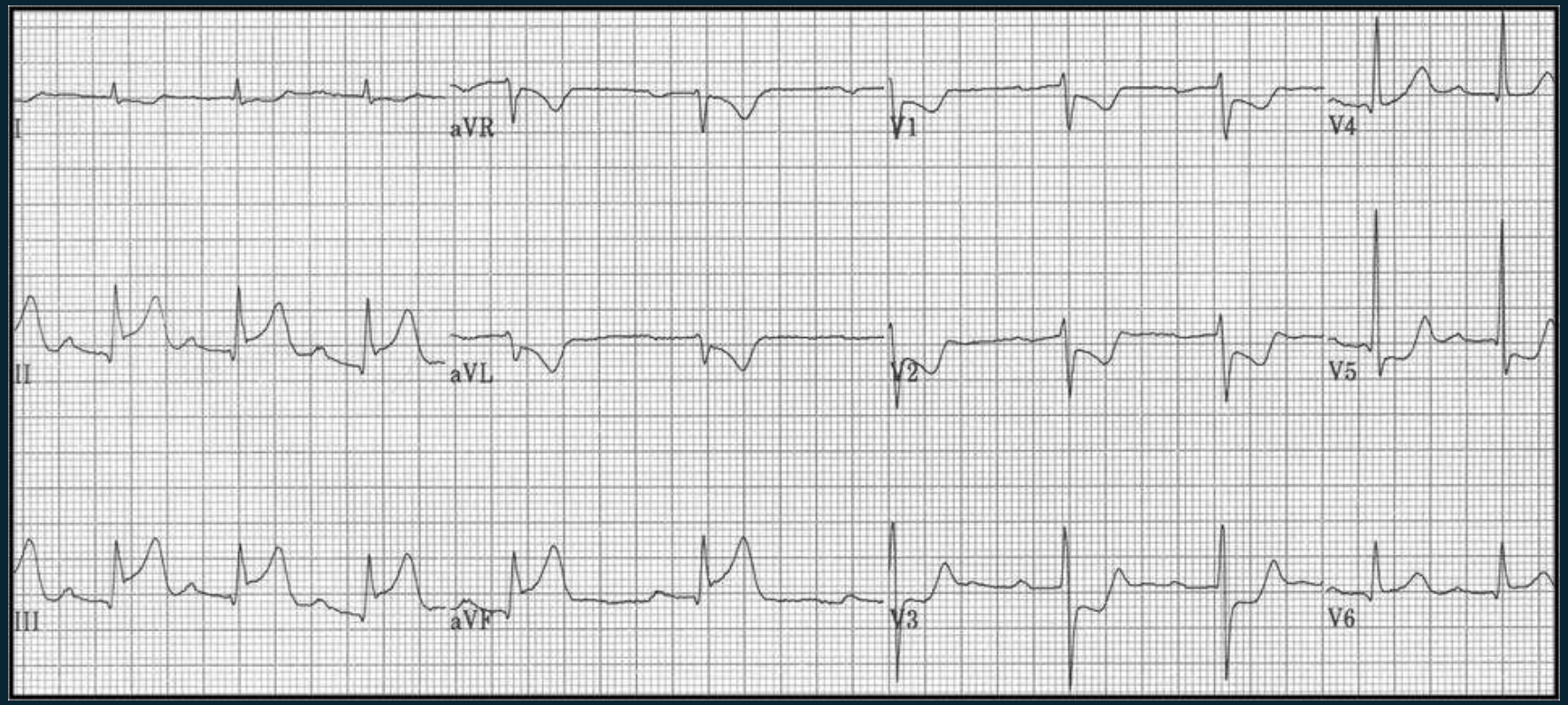
Clinical situation	Immediate principle
Unstable monomorphic tachycardia with a pulse	Synchronised cardioversion
Ventricular fibrillation or pulseless VT	CPR and defibrillation
Sustained polymorphic VT	Immediate unsynchronised shock
Stable broad-complex tachycardia	Monitor and obtain urgent expert assessment

A rhythm name must never delay emergency action.

CASE 6 · TEACHING SCENARIO

Chest pain with a regional ST pattern

Which lead group is affected? Where is the reciprocal change?



A regional pattern plus ischaemic symptoms needs urgent assessment.

ST–T changes need clinical context

Regional or dynamic change

Compare neighbouring leads and earlier recordings.

ST elevation has mimics

Early repolarisation, pericarditis, LVH and LBBB can alter ST–T.

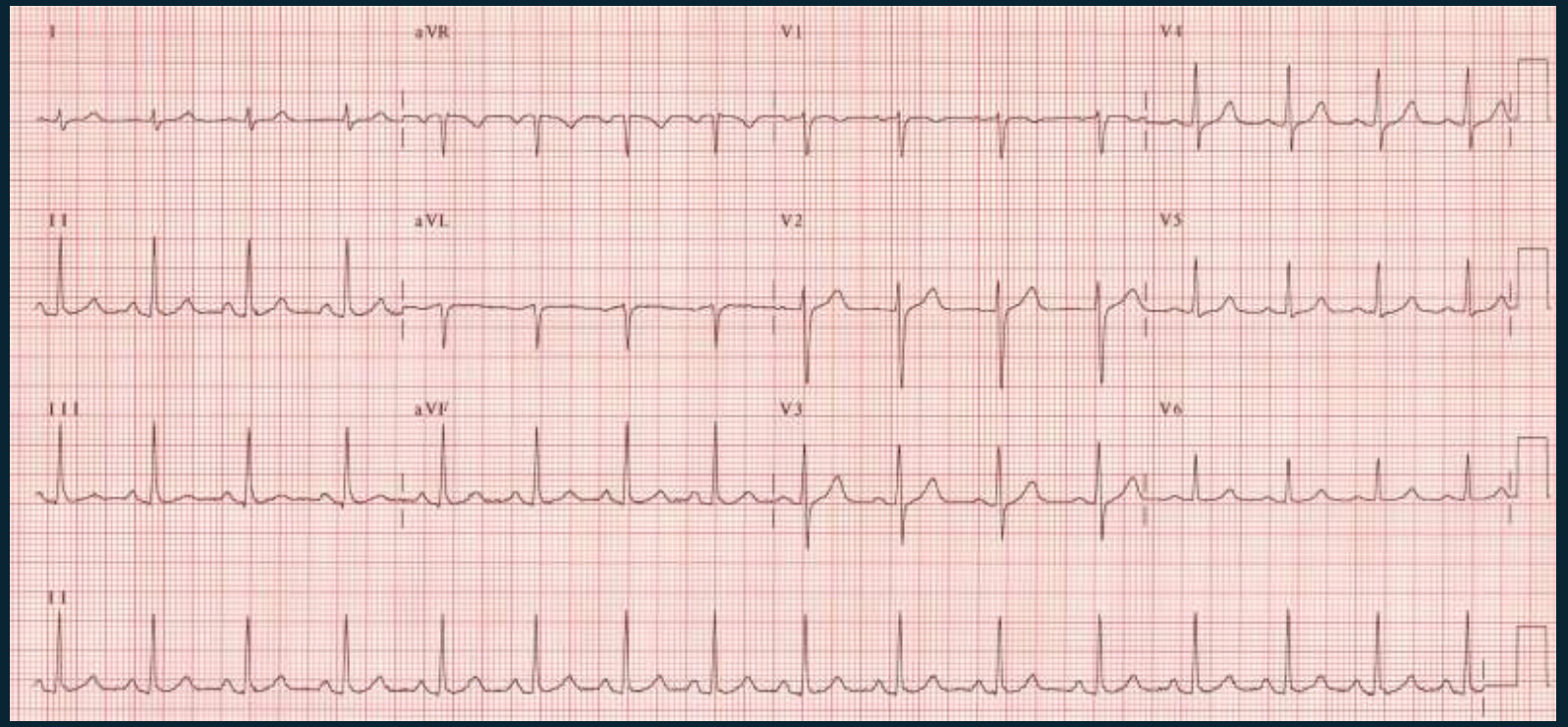
Symptoms can persist despite a non-diagnostic ECG

Repeat the ECG and consider additional leads when indicated.

A normal first ECG does not exclude acute coronary syndrome.

A structured ECG report

Work through the same six questions. What can you now explain?



Sinus rhythm, normal axis, narrow QRS, no acute regional ST–T change.

RETRIEVAL ROUND

Five questions without looking back

1. What do P, QRS and T represent?
2. At 25 mm/s, what does one small box mean?
3. I is positive and aVF negative. Which lead is next?
4. What does a regular rate near 150 make you look for?
5. Can 2:1 AV block alone establish Mobitz II?

Discuss with the person beside you for 45 seconds.

The answers, with the reasoning

Question	Answer
P / QRS / T	Atrial activation / ventricular activation / recovery
One small box	40 ms horizontally at 25 mm/s
I positive, aVF negative	Lead II separates normal leftward axis from LAD
A regular rate near 150	Look for flutter with 2:1 conduction
2:1 AV block	The ratio alone cannot classify Mobitz I or II

A checklist worth keeping

Patient	Pulse, symptoms, perfusion and stability
Paper	Identity, time, speed, gain, placement and artefact
Rhythm	Rate, regularity, P waves and P–QRS relationship
Conduction	Axis, PR, QRS width / morphology and QTc
ST–T	Regional patterns, reciprocal changes and comparison
Summary	Main finding, uncertainty and the next action

The final sentence links the ECG finding to the patient.

ECG essentials

Patient
Paper
Rhythm
Conduction
ST–T
Summary



Questions and discussion

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APPENDIX

Adult reference values

Feature	Usual reference or threshold
Resting sinus rate	60–100/min conventionally; lower rates may be physiological
Frontal QRS axis	Approximately −30° to +90°
PR interval	120–200 ms
QRS duration	Usually <110 ms; ≥120 ms is broad
QTc	Common prolonged thresholds: >450 ms men, ≥460 ms women

A reference range supports interpretation. It does not replace context.

APPENDIX

QTc: a worked example

Illustrative measurements: QT 400 ms, R–R 800 ms (rate 75/min).

BAZETT

$QTc = QT / \sqrt{RR}$

$0.400 / \sqrt{0.800}$
$= 0.447$ s = 447 ms

FRIDERICIA

$QTc = QT / \sqrt[3]{RR}$

$0.400 / \sqrt[3]{0.800}$
$= 0.431$ s = 431 ms

Use seconds for both QT and RR in these calculations.

Always state the formula and check whether the result fits the tracing.

APPENDIX

Q waves, voltage and supporting evidence

Q waves

Small septal q waves can be normal in lateral leads.
A concerning pattern depends on width, depth and distribution.

High voltage

Voltage criteria can support LVH, but do not prove anatomical LVH.
Body habitus, age and athletic training affect voltage.

Poor R-wave progression

Consider placement, conduction and structural causes.
It does not establish an anterior infarct by itself.

An isolated finding is a clue to investigate, not proof of disease.

APPENDIX

Why 2:1 block resists a Mobitz label

P QRS P — P QRS P —

Every alternate P wave conducts.

There are no two consecutive conducted beats
to show whether the PR interval progressively lengthens.

Describe the conduction ratio first, then assess level and risk.

APPENDIX

Conventional ST-elevation thresholds

J-point elevation in at least two contiguous leads, without LVH / LBBB confounding.

Lead / group	Threshold
All leads except V2–V3	≥1 mm
V2–V3: men aged <40 years	≥2.5 mm
V2–V3: men aged ≥40 years	≥2 mm
V2–V3: women	≥1.5 mm

Absence of these thresholds does not exclude acute coronary occlusion.

APPENDIX

Five shortcuts that can mislead

Tempting shortcut	More accurate interpretation
“A P wave means sinus rhythm”	Assess P morphology, axis and the AV relationship
“A broad QRS means VT”	Broad QRS has several causes; undifferentiated WCT needs caution
“A rate of 150 means flutter”	Search for flutter waves and the conduction ratio
“New LBBB proves acute MI”	Use symptoms, comparison and appropriate ischaemia assessment
“The machine says normal”	Review the tracing and the clinical presentation yourself

Selected references: foundations

SCST, 2024

Recording a Standard 12-lead Electrocardiogram

AHA / ACCF / HRS ECG standardisation

Part III: conduction, 2009. Part IV: ST–T, U waves and QT, 2009

ACC / AHA / HRS, 2018

Guideline on Bradycardia and Cardiac Conduction Delay

ECG Learning Center, University of Utah

Frank G. Yanowitz, MD. Waves, leads and clinical examples

Selected references: clinical recognition

ESC, 2019

Guideline on Supraventricular Tachycardia

ACC / AHA / ACCP / HRS, 2023

Guideline for Atrial Fibrillation

ESC, 2023 and ACC, 2022

Acute Coronary Syndromes guideline and Acute Chest Pain pathway

AHA, 2025

Adult Advanced Life Support

AHA / ACCF scientific statement, 2010

Prevention of Torsade de Pointes in Hospital Settings