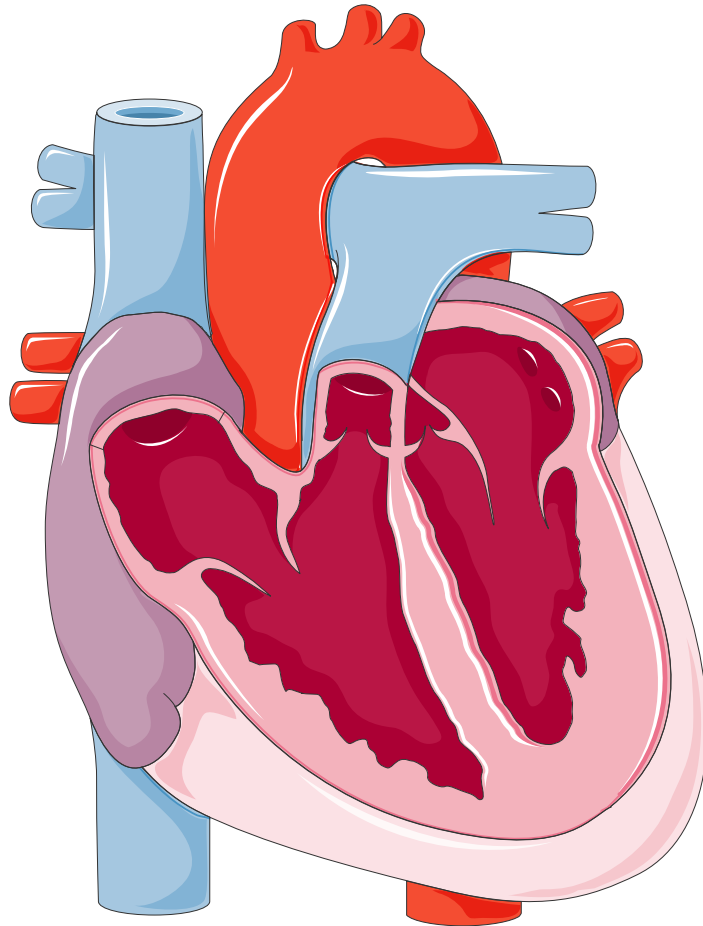


Clinical Electrocardiography

A Case-Based Visual Guide



From fundamentals to advanced interpretation



PTB-XL 00033 | 0.35-5.35 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Dr Qurban Hussain Khan

Cardiologist and Cardiac Electrophysiologist

HOW TO USE THIS GUIDE

Learn from the patient and the tracing

The central question. Which electrical process explains the tracing, and what does it mean for this patient?

A simple reading rhythm

1

Start with the short clinical presentation.

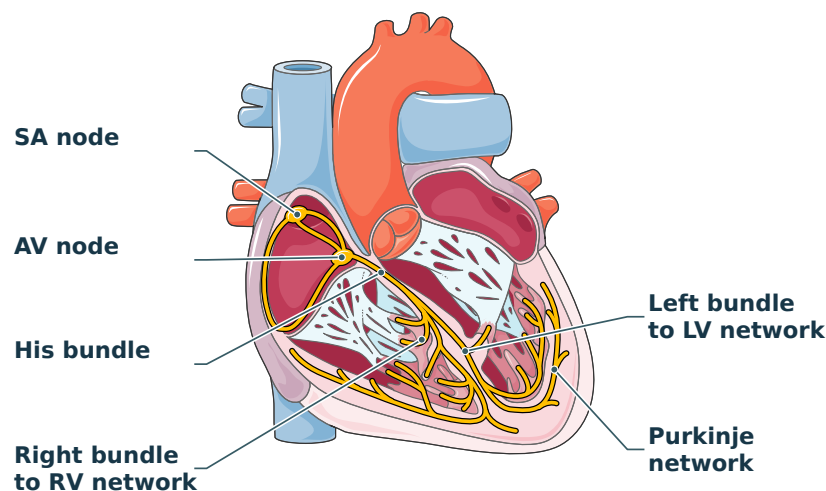
2

Read the ECG before the highlighted findings.

3

Follow the anatomy to understand the pattern.

From pacemaker to ventricular network



The specialised conduction pathway is shown in yellow.
Atrial lines and ventricular branching are simplified.

- Use the close-up and comparison ECGs to test what you see.
- Turn the mechanism into a clinical action, then check the pitfall.

All ECGs come from published recordings or clinical reports. Teaching vignettes are composites unless marked “Published case”. Figure credits and references identify the original sources.

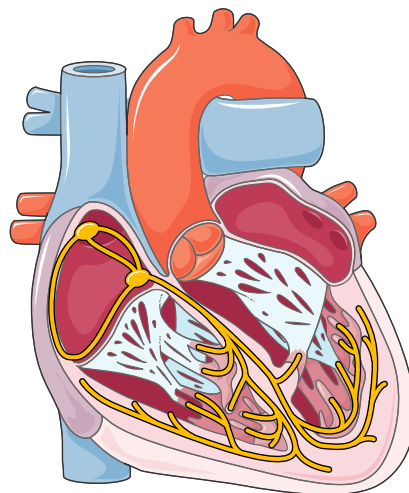
ECG plots use standard 25 mm/s and 10 mm/mV proportions. Enlargement is equal in both directions; each square represents 40 ms by 0.1 mV. Published ECG images retain their original proportions. Upper twelve-lead panels show the same 2.5-second window, with a ten-second lead II strip.

This educational guide supports interpretation alongside the patient’s clinical assessment and current local protocols.

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Foundations

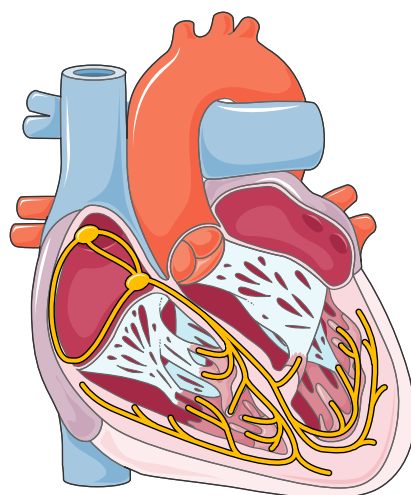
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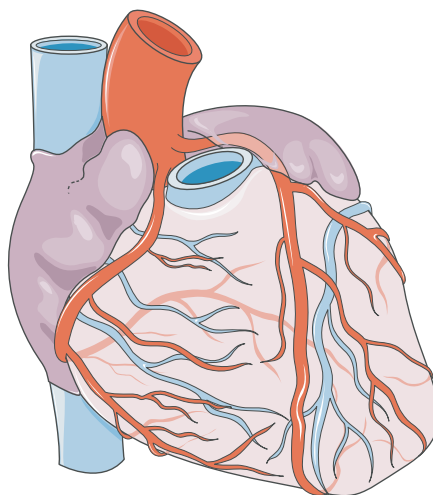
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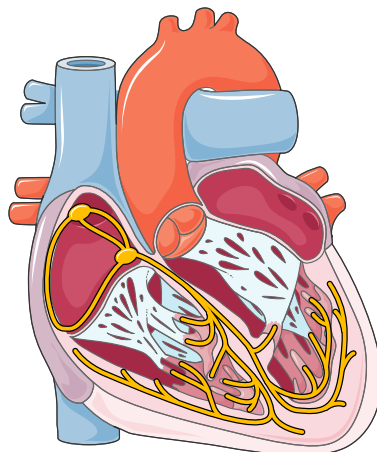
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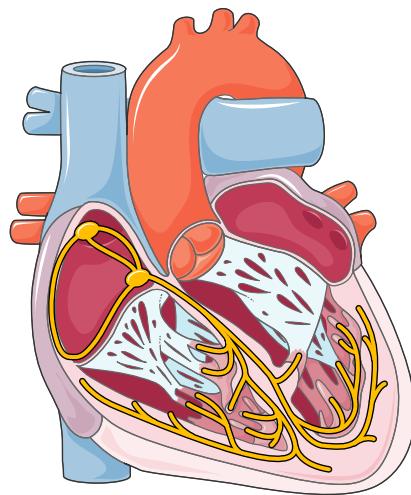
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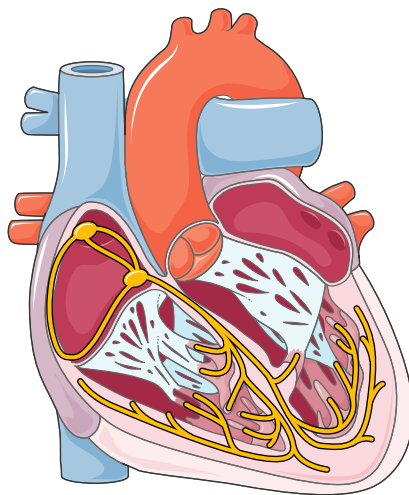
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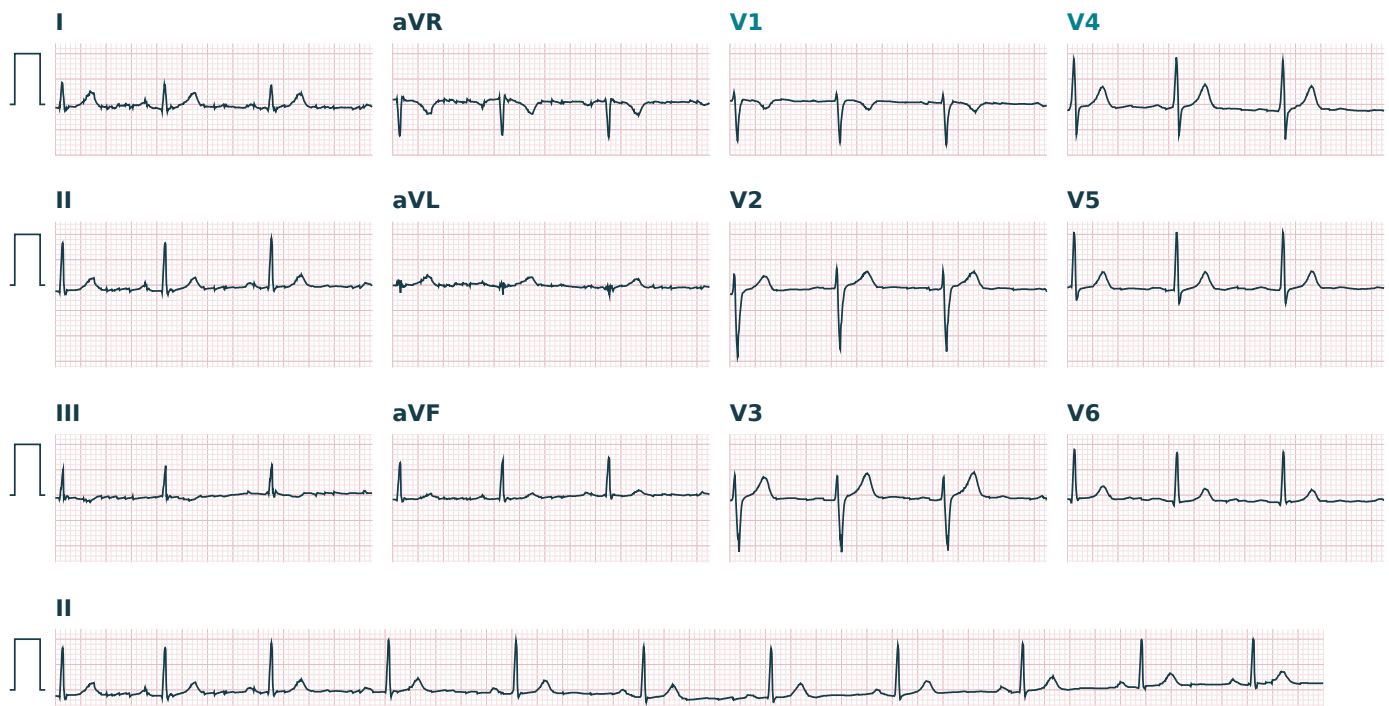


CASE 01 | FOUNDATIONS

One Heart, Different Electrical Views

CLINICAL PRESENTATION

A well patient has a regular pulse. A student notices an upward QRS in V4 but a downward QRS in V1 and asks whether one must be abnormal.

Can opposite shapes describe the same heartbeat?

PTB-XL 00010 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise**1**

V1 is mainly negative; V4 is mainly positive.

2

The QRS complexes occur together across the aligned leads.

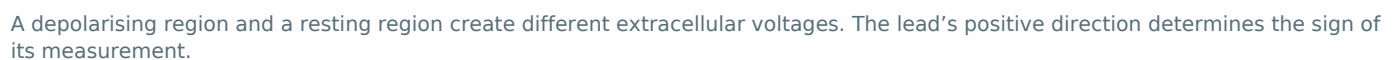
3

Amplitude changes with the viewing direction.

Remember: One event; several views.

From anatomy to clinical meaning

Why an activation front creates a voltage difference



-

In practice

- Avoid.** A downward QRS is not automatically abnormal.

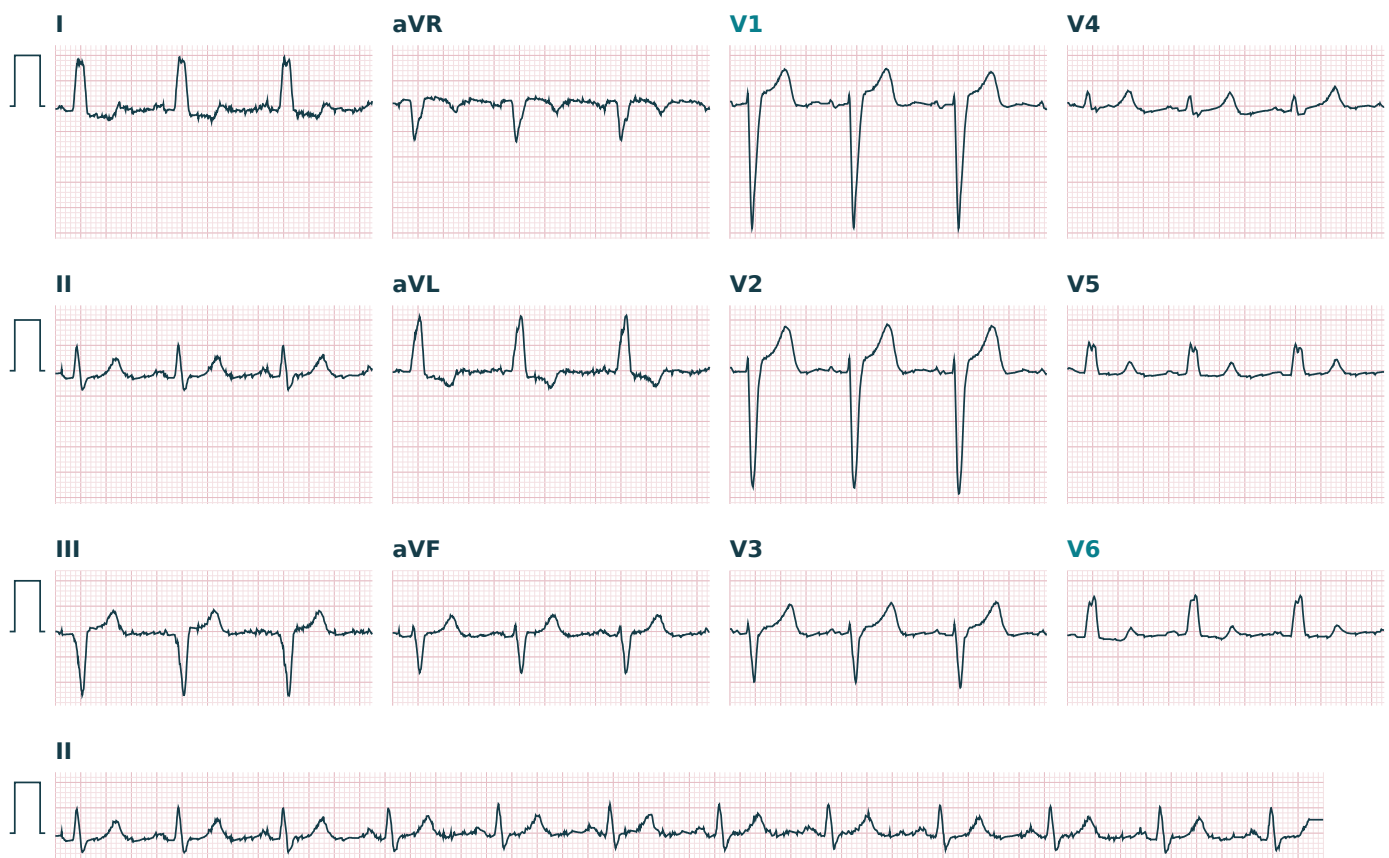
CASE 02 | FOUNDATIONS

Activation Shapes Recovery

CLINICAL PRESENTATION

A stable patient has longstanding bundle-branch block. Broad QRS complexes are followed by unfamiliar T waves. A trainee wonders whether the T waves necessarily mean a second disease.

Could the changed activation route explain the T waves?



PTB-XL 00618 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

V1 has a broad, mainly negative QRS.

2

Lateral leads show broad, notched positive complexes.

3

The following ST-T direction differs from the main QRS.

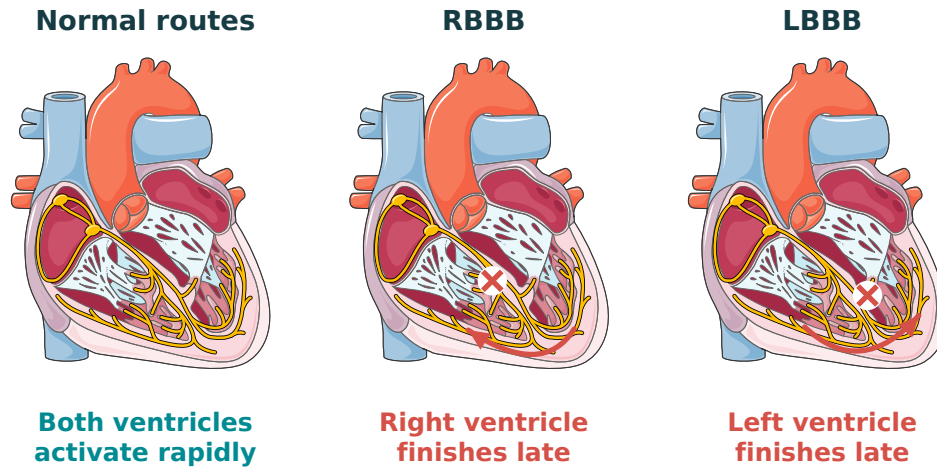
Remember: Read activation before recovery.

CASE 02 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Left bundle-branch block with secondary ST-T changes

Bundle-branch block: which ventricle finishes last?



Anatomy: Servier Medical Art, CC BY 4.0. Routes and labels added.

The intact branch activates its ventricle first; slower myocardial spread reaches the delayed ventricle. Routes are simplified.

- The QRS records ventricular activation; T records ventricular recovery.
- Delayed activation changes when different regions begin their electrical cycle.
- Their changed recovery sequence can produce secondary ST-T changes.



PTB-XL 00618 | 0-5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
 PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Read QRS duration and morphology before judging the T wave.
- Compare the ST-T pattern with a previous ECG.
- New chest pain still needs acute assessment despite longstanding bundle-branch block.

Avoid. A conduction abnormality does not explain every new ST change.

CASE 02 | ANOTHER REAL ECG

Compare the patterns

Left bundle-branch block with secondary ST-T changes



PTB-XL 00618 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Compare a narrower activation sequence.



PTB-XL 00033 | 0.35-5.35 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- QRS complexes are narrower than in the main example.
- Read T-wave direction together with its preceding QRS.

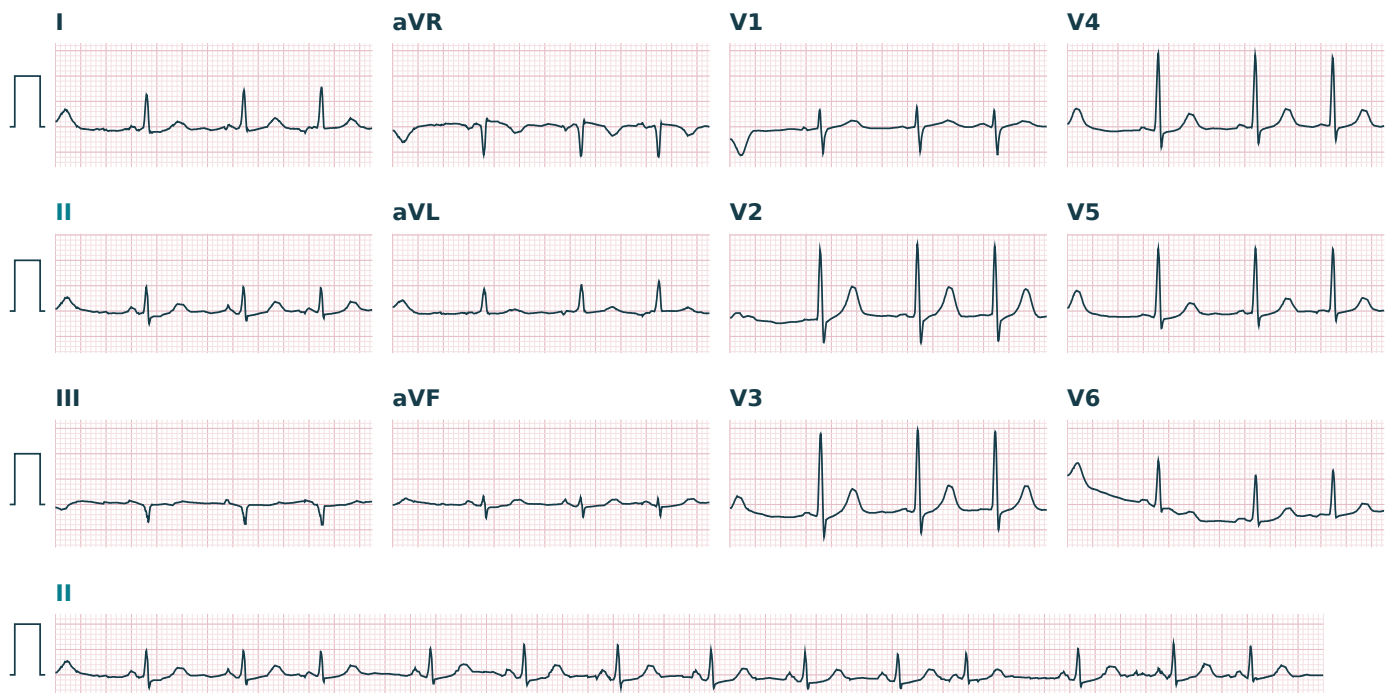
Remember. Read activation before recovery.

CASE 03 | FOUNDATIONS

Follow One Beat Through the Heart

CLINICAL PRESENTATION

A student feels a patient's radial pulse while watching the monitor. They can recognise the large spike but cannot connect the smaller waves with the heart's electrical route.

Which events produce P, QRS and T?

PTB-XL 00687 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise**1**

A small P wave precedes each QRS in II.

2

The ST segment follows QRS.

3

The T wave follows the ST segment.

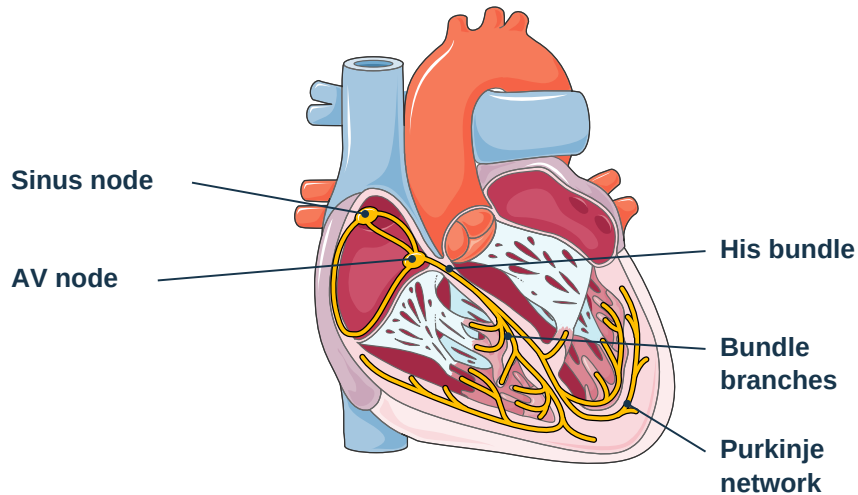
Remember: P: atria. QRS: ventricles activate. T: ventricles recover.

CASE 03 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

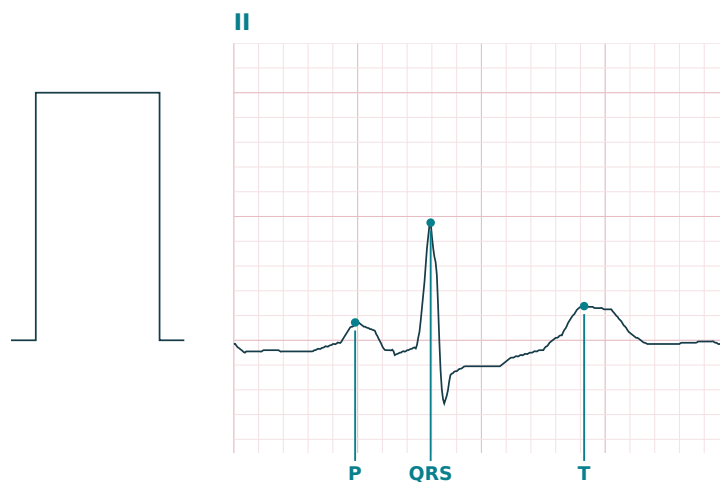
Atrial activation, ventricular activation and ventricular recovery

The electrical route through the heart



The impulse travels from sinus node through atria and AV conduction into the ventricular network. Adapted from Servier Medical Art, CC BY 4.0.

- The sinus node initiates atrial activation, recorded as P.
- AV conduction reaches His-Purkinje tissue, which rapidly activates ventricles: QRS.
- Ventricles recover electrically during the period ending with T.



PTB-XL 00687 | 0.4-1.2 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

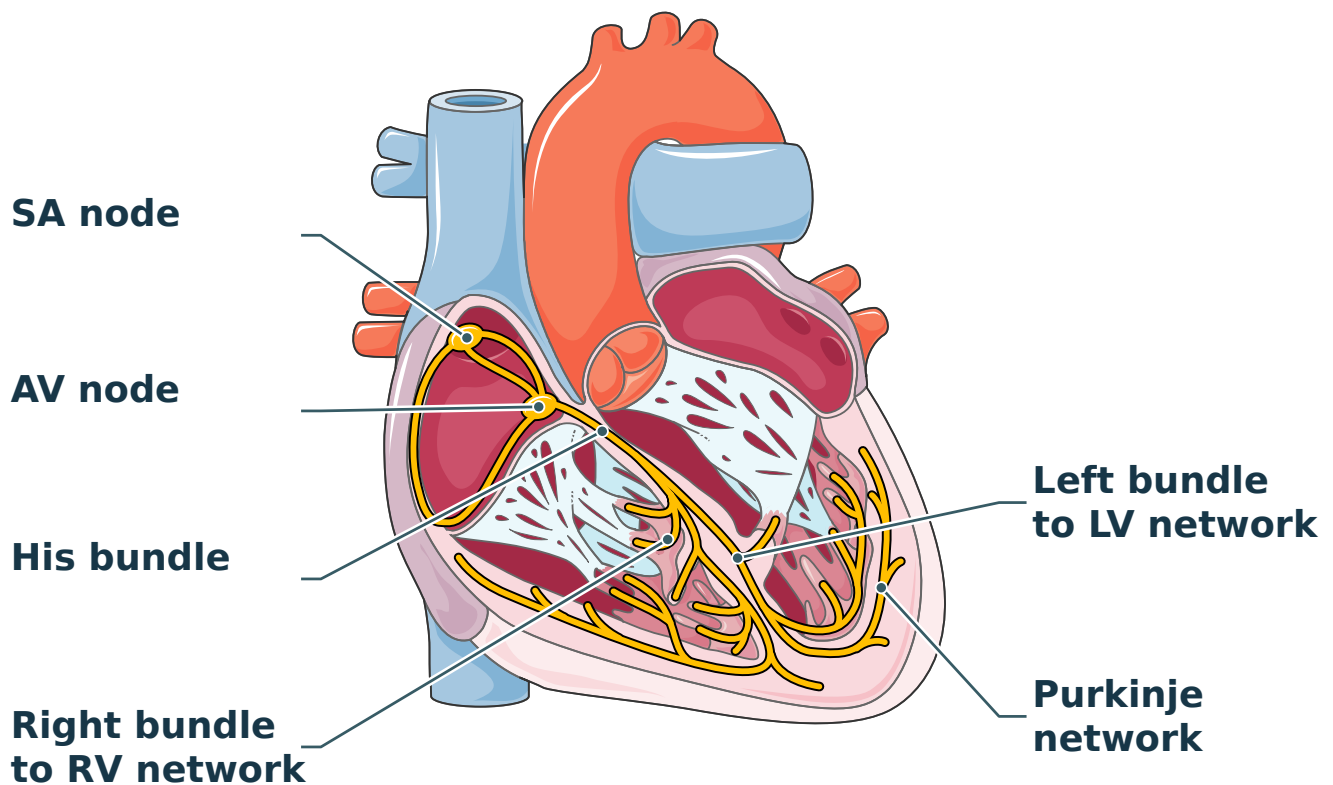
- Identify P, QRS and T on one clean beat.
- Check whether the same P-QRS relationship repeats.
- Check the pulse: electrical activity alone does not establish effective circulation.

Avoid. The radial pulse follows electrical activation; it is not the QRS itself.

ANATOMY AT A GLANCE

SA node to Purkinje network

From pacemaker to ventricular network



The specialised conduction pathway is shown in yellow.
Atrial lines and ventricular branching are simplified.

Connect it to the ECG. P: atria. QRS: ventricles activate. T: ventricles recover.

- The sinus node initiates atrial activation, recorded as P.
- AV conduction reaches His-Purkinje tissue, which rapidly activates ventricles: QRS.
- Ventricles recover electrically during the period ending with T.

Adapted from Servier Medical Art, CC BY 4.0. Teaching labels added.

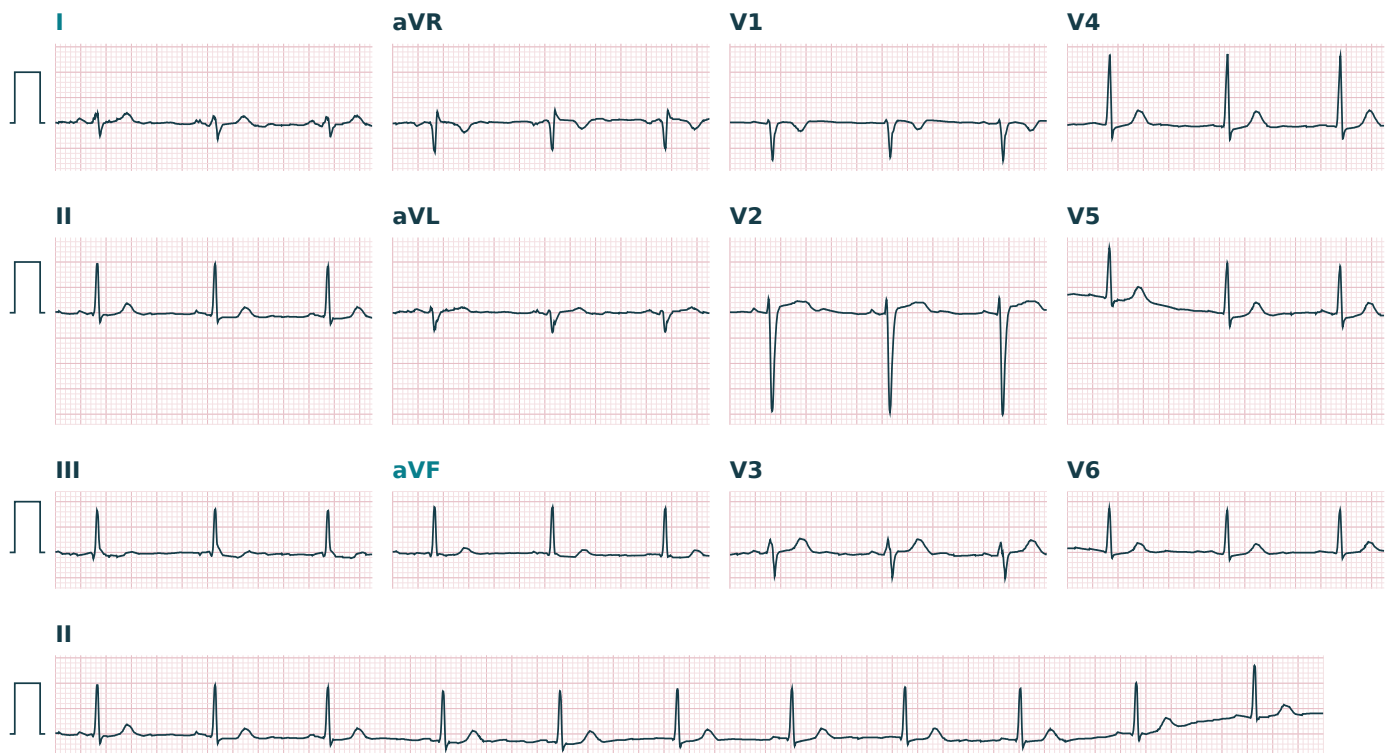
CASE 04 | FOUNDATIONS

Place Electrodes; Understand the Views

CLINICAL PRESENTATION

Before a procedure, ten skin electrodes produce twelve printed leads. Lead I is mainly negative while the inferior leads are positive. The trainee checks positioning before interpreting direction.

How do ten electrodes provide twelve electrical views?



PTB-XL 07031 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Lead I is predominantly negative.

2

II, III and aVF are predominantly positive.

3

These limb leads show simultaneous views of the same activation.

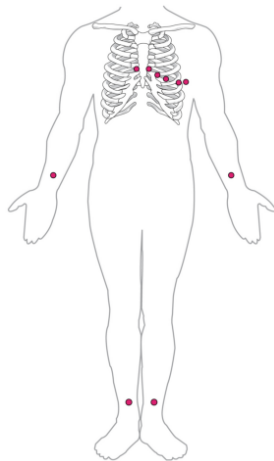
Remember: Ten contacts; twelve views.

CASE 04 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

A rightward frontal QRS direction requiring clinical interpretation

Ten electrodes, twelve electrical views



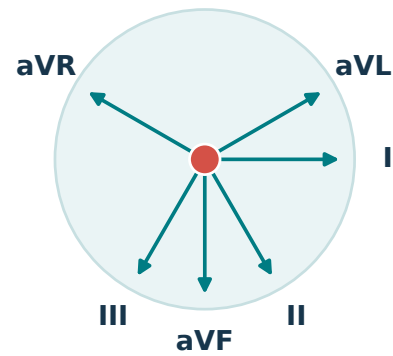
6 limb leads

Frontal plane

6 chest leads

Transverse plane

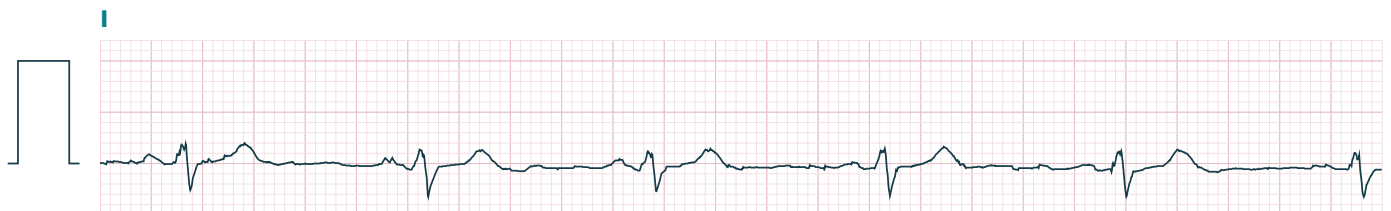
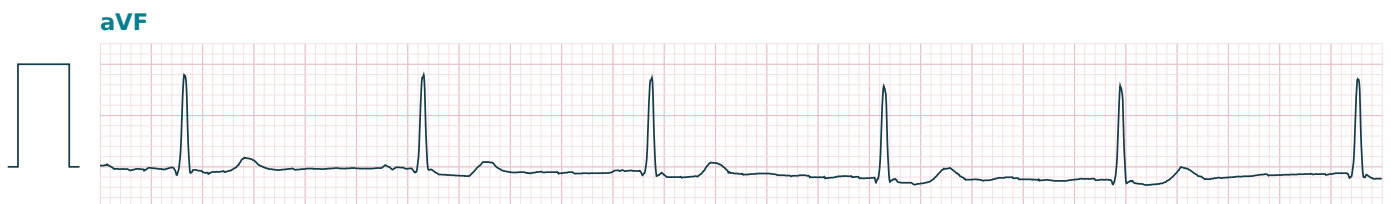
Frontal lead directions



Patient's left →

Ten electrodes provide twelve views. Torso adapted from OpenStax College (2013), CC BY 3.0; lead directions drawn for this course.

- Limb electrode comparisons provide six frontal-plane leads.
- Six chest electrodes provide six additional transverse-plane views.
- Moving an electrode changes the voltage comparison and can change morphology.



PTB-XL 07031 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Locate chest electrodes using anatomical landmarks.
- Place V5 and V6 at V4's horizontal level.
- Check connections before diagnosing an unexpected axis pattern.

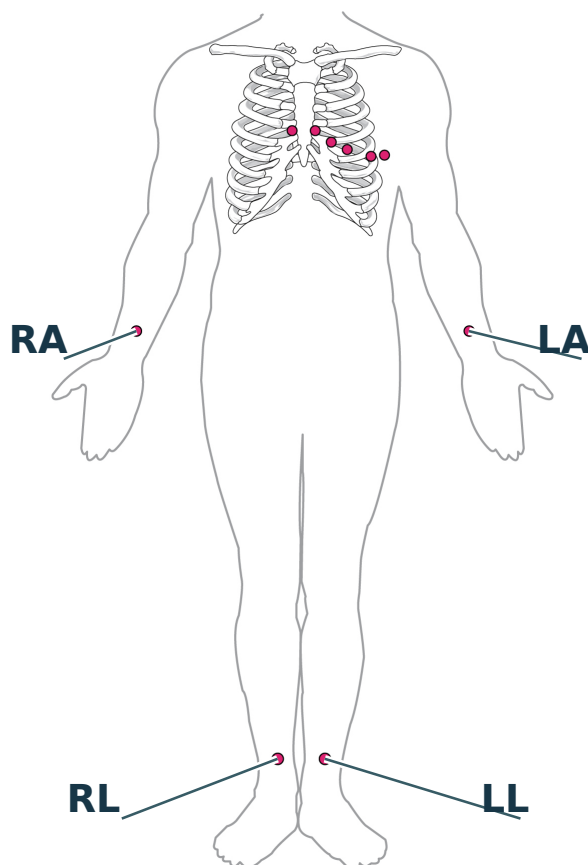
Avoid. Do not follow the downward rib slope when placing V5 and V6.

ANATOMY AT A GLANCE

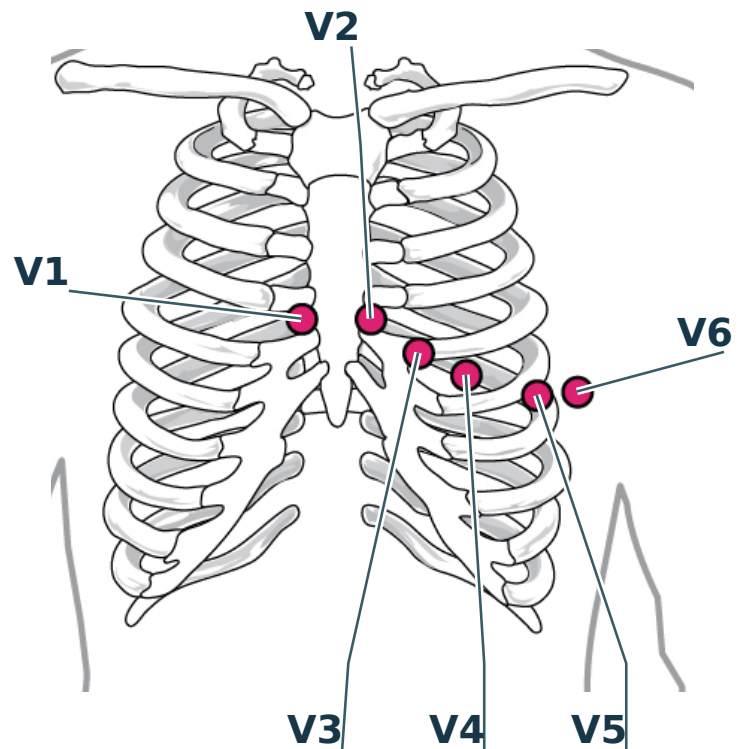
Placement of ten ECG electrodes

Ten electrodes produce twelve leads

Limb electrodes



Chest electrodes



V4-V6: use one horizontal level.

Right and left refer to the patient. Place electrodes using anatomical landmarks.
V3 lies between V2 and V4; do not follow the downward slope of the ribs.

Connect it to the ECG. Ten contacts; twelve views.

- Limb electrode comparisons provide six frontal-plane leads.
- Six chest electrodes provide six additional transverse-plane views.
- Moving an electrode changes the voltage comparison and can change morphology.

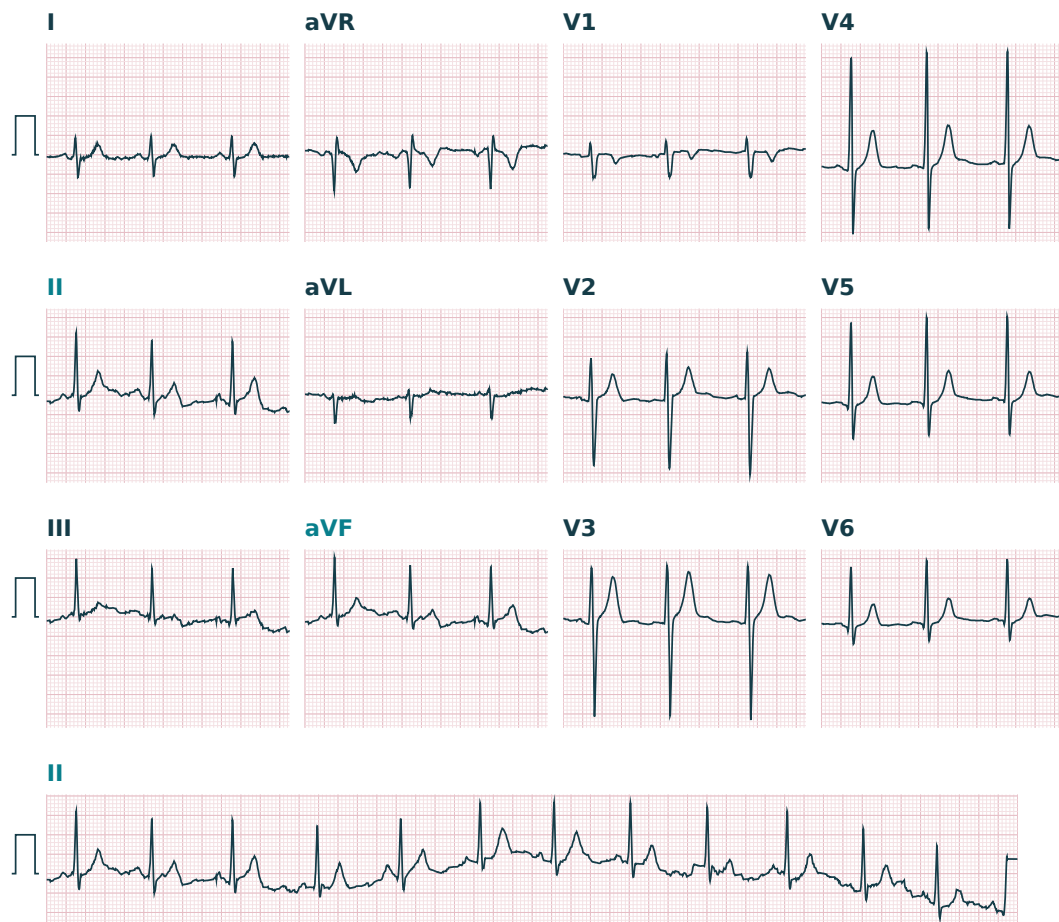
OpenStax College (2013), CC BY 3.0. Teaching labels added.

CASE 05 | FOUNDATIONS

Check the Recording First

CLINICAL PRESENTATION

A comfortable ward patient has an ECG with marked baseline movement. QRS complexes remain visible through the drift. The apparent ST displacement must be checked before it drives treatment.

Is the reference baseline stable enough for measurement?

PTB-XL 00004 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise**1**

The inferior-lead baseline moves slowly.

2

QRS complexes continue through the drift.

3

A nearby baseline differs from the page's horizontal edge.

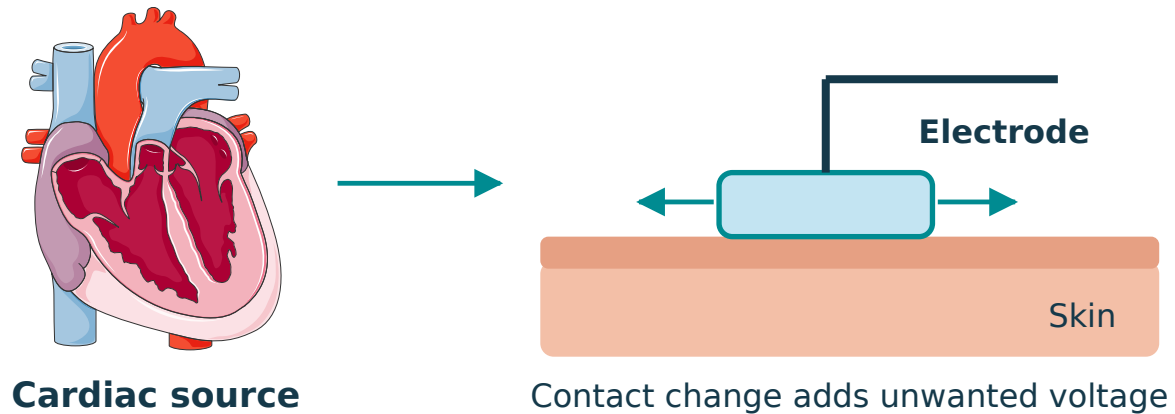
Remember: Check the patient; then check the recording.

CASE 05 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

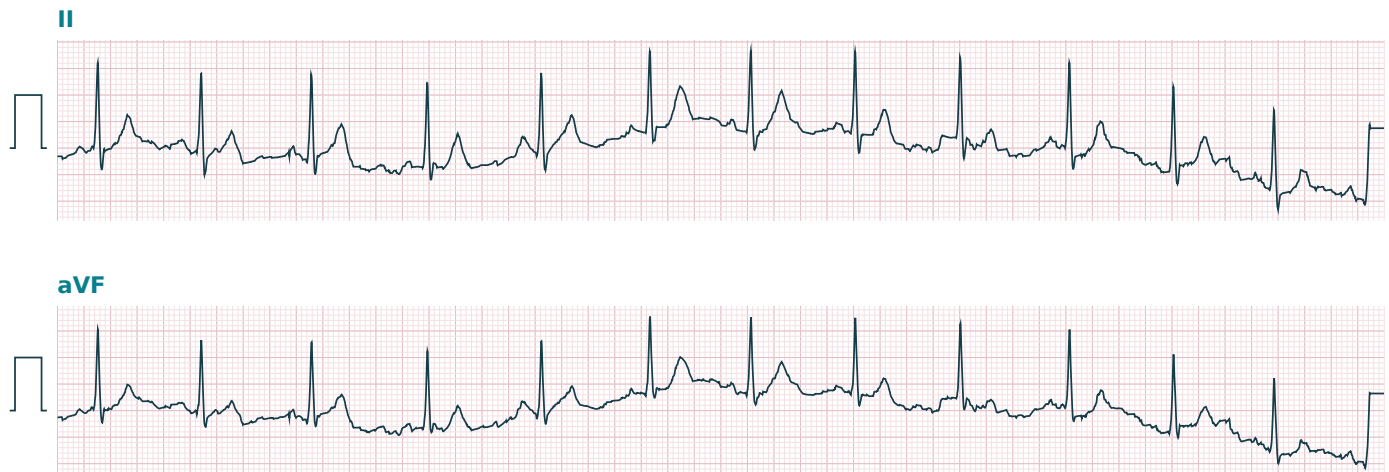
Baseline drift superimposed on a clinical ECG

Movement can change the electrode signal



Movement changes skin contact, adding unwanted voltage to the cardiac signal.

- Skin contacts detect cardiac signals plus unwanted electrical changes.
- Movement or unstable contact can shift the reference baseline.
- Gain and recording speed determine the displayed voltage and time scales.



PTB-XL 00004 | 0-10 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Assess the patient, then check identity, connections, speed and gain.
- Improve skin contact and repeat a poor-quality recording.
- Measure ST displacement from a nearby baseline; retain the original ECG.

Avoid. Artefact and genuine disease can coexist.

CASE 05 | ANOTHER REAL ECG

Compare the patterns

Baseline drift superimposed on a clinical ECG

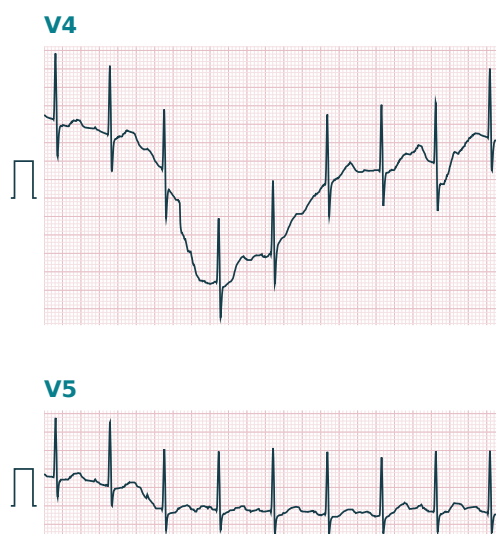


PTB-XL 00004 | 0-10 s | 25 mm/s, 10 mm/mV proportions

Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Compare drifting V4 with its neighbouring lead.



PTB-XL 00849 | 0-5 s | 25 mm/s, 10 mm/mV proportions

Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- V4 has a slow baseline excursion.
- V5 provides a simultaneous, more stable comparison.

Remember. Check the patient; then check the recording.

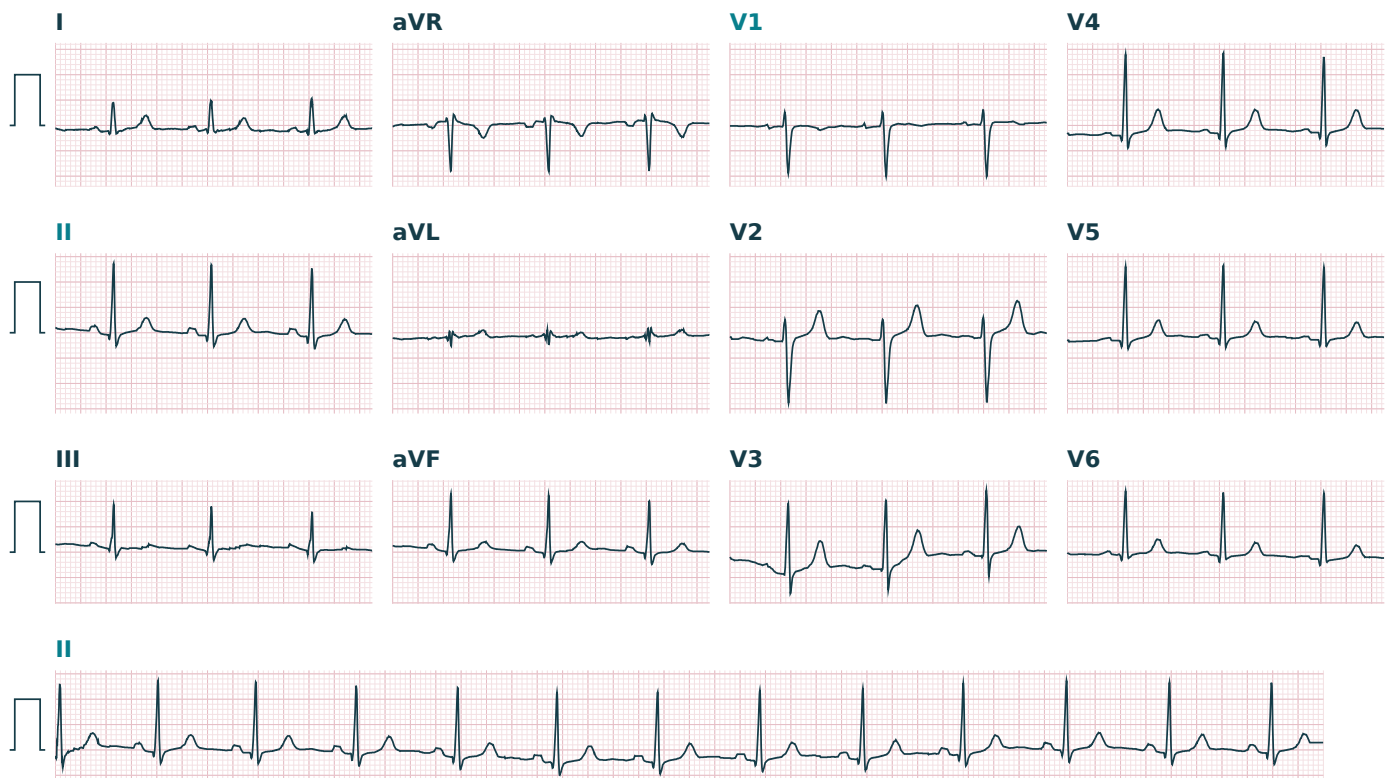
CASE 06 | FOUNDATIONS

Read and Measure a Normal Beat

CLINICAL PRESENTATION

A patient's preoperative ECG looks familiar and the machine calls it normal. A trainee must still identify the beginning and end of each interval before reporting it.

Where does each measurement start and finish?



PTB-XL 00033 | Upper leads: 0.35-2.85 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Lead II shows P, QRS and T in sequence.

2

V1 shows the same event from another direction.

3

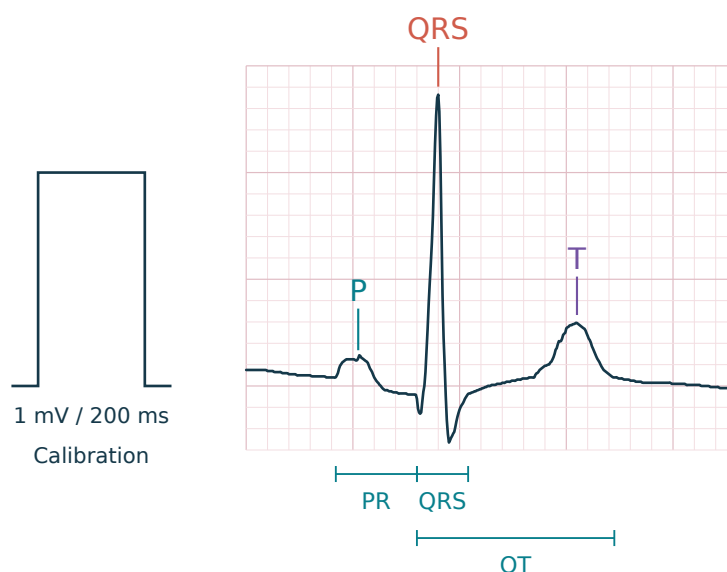
The T wave returns towards the baseline before the next beat.

Remember: Find the landmarks before measuring the distance.

CASE 06 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Visible P, QRS and T landmarks



P

Atria activate

QRS

Ventricles activate

T

Ventricles recover

PTB-XL 00033 | Lead II

25 mm/s | 10 mm/mV proportions

Grid: 40 ms × 0.1 mV

Lead II from PTB-XL 00033. Approximate landmarks show where intervals begin and end. Wagner et al., CC BY 4.0.

- PR begins at P onset and ends at QRS onset.
- QRS spans ventricular activation; the J point marks its end.
- QT begins at QRS onset and ends at T end.



PTB-XL 00033 | 0.35-5.35 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms × 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Measure from true waveform boundaries, not from the largest peaks.
- Use aligned leads to check an uncertain onset or offset.
- Report rate, rhythm, intervals and relevant ST-T findings.

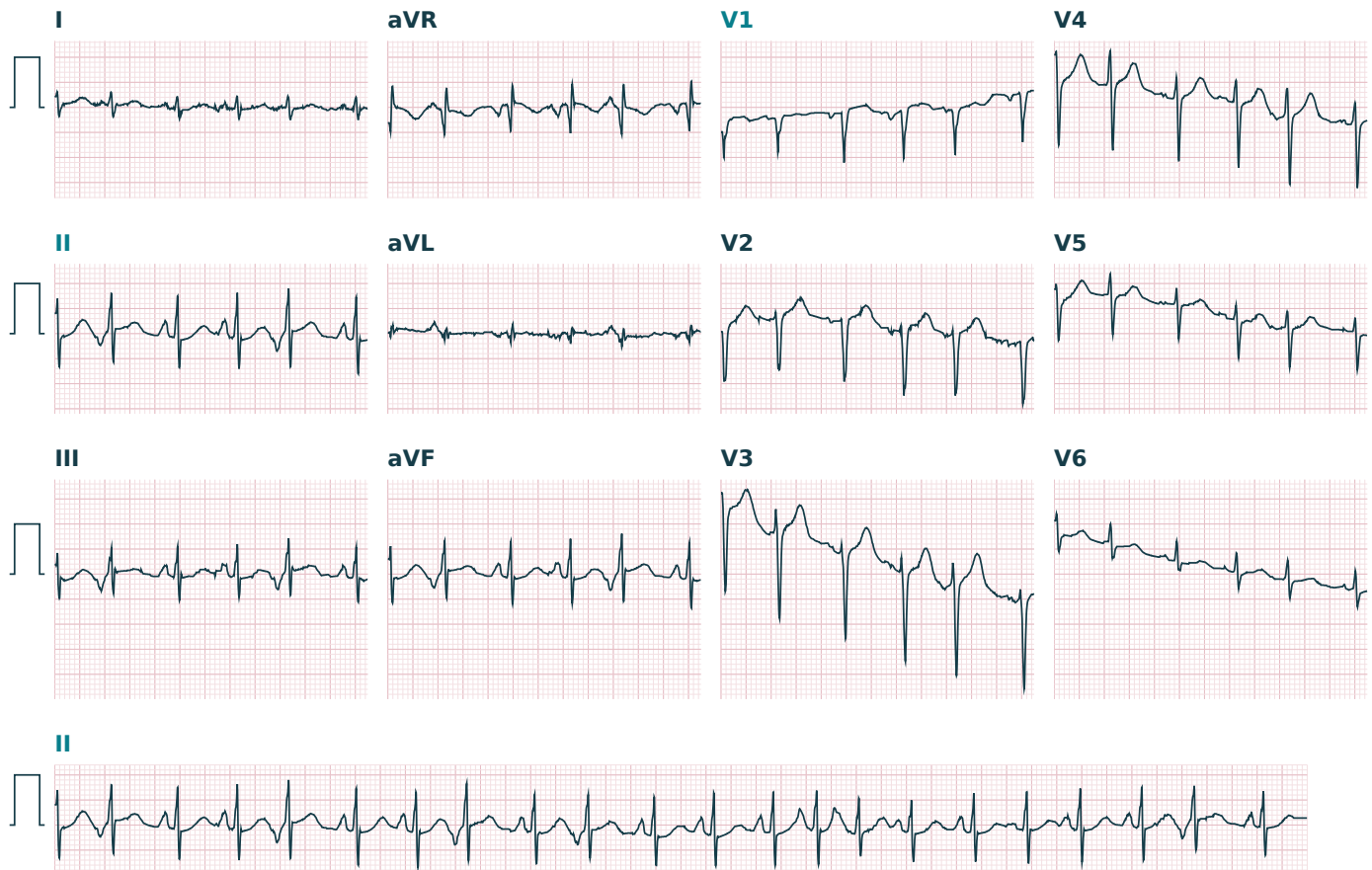
Avoid. The PR segment is only part of the PR interval.

CASE 07 | FOUNDATIONS

Separate Rate from Rhythm

CLINICAL PRESENTATION

A patient with fever and poor intake has a fast regular pulse. The ECG is labelled “tachycardia”. The clinician must determine what is fast, where it starts and why.

Is the fast rhythm sinus, and what is driving it?

PTB-XL 04408 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise**1**

QRS complexes occur rapidly.

2

Atrial deflections recur before QRS complexes in II.

3

Compare consecutive cycles rather than one isolated beat.

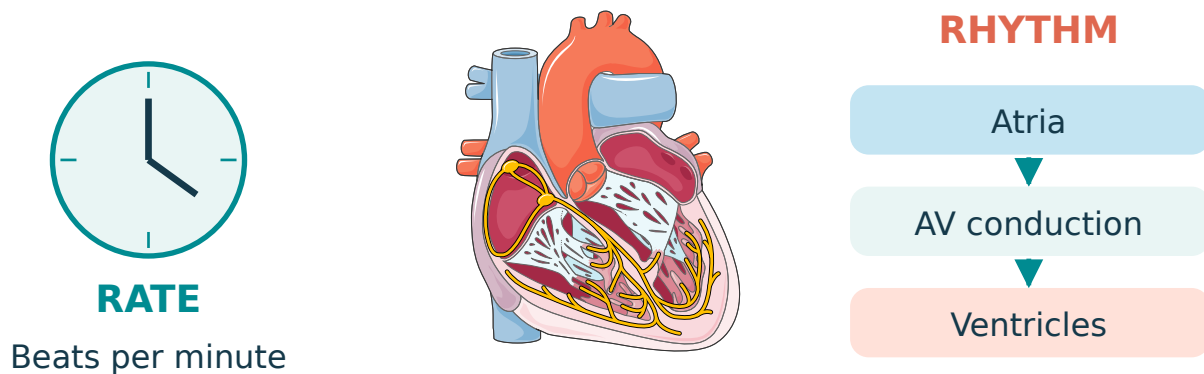
Remember: Rate counts beats; rhythm explains their sequence.

CASE 07 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

A rapid rhythm with a recurring P-QRS sequence

Rate counts; rhythm explains the sequence



The P-QRS relationship reveals the sequence.

Rate measures frequency; atrial activity and its relationship to QRS reveal rhythm. Anatomy: Servier Medical Art, CC BY4.0.

- Sinus-node impulses activate atria before reaching the ventricles.
- The R-R interval sets ventricular rate; the P-QRS relationship identifies sequence.
- A faster sinus rate can be a response to physiological stress.



PTB-XL 04408 | 0-10 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Assess blood pressure, perfusion and symptoms immediately.
- For regular rhythms, calculate rate as 60 divided by RR seconds.
- If sinus tachycardia is present, investigate its clinical driver.

Avoid. Fast and regular does not automatically mean sinus rhythm.

CASE 07 | ANOTHER REAL ECG

Compare the patterns

A rapid rhythm with a recurring P-QRS sequence



PTB-XL 04408 | 0-10 s | 25 mm/s, 10 mm/mV proportions

Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Compare an irregular rhythm without a stable atrial clock.



PTB-XL 00330 | 0-6 s | 25 mm/s, 10 mm/mV proportions

Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- R-R intervals vary substantially.
- No consistent P-before-QRS sequence is visible.

Remember. Rate counts beats; rhythm explains their sequence.

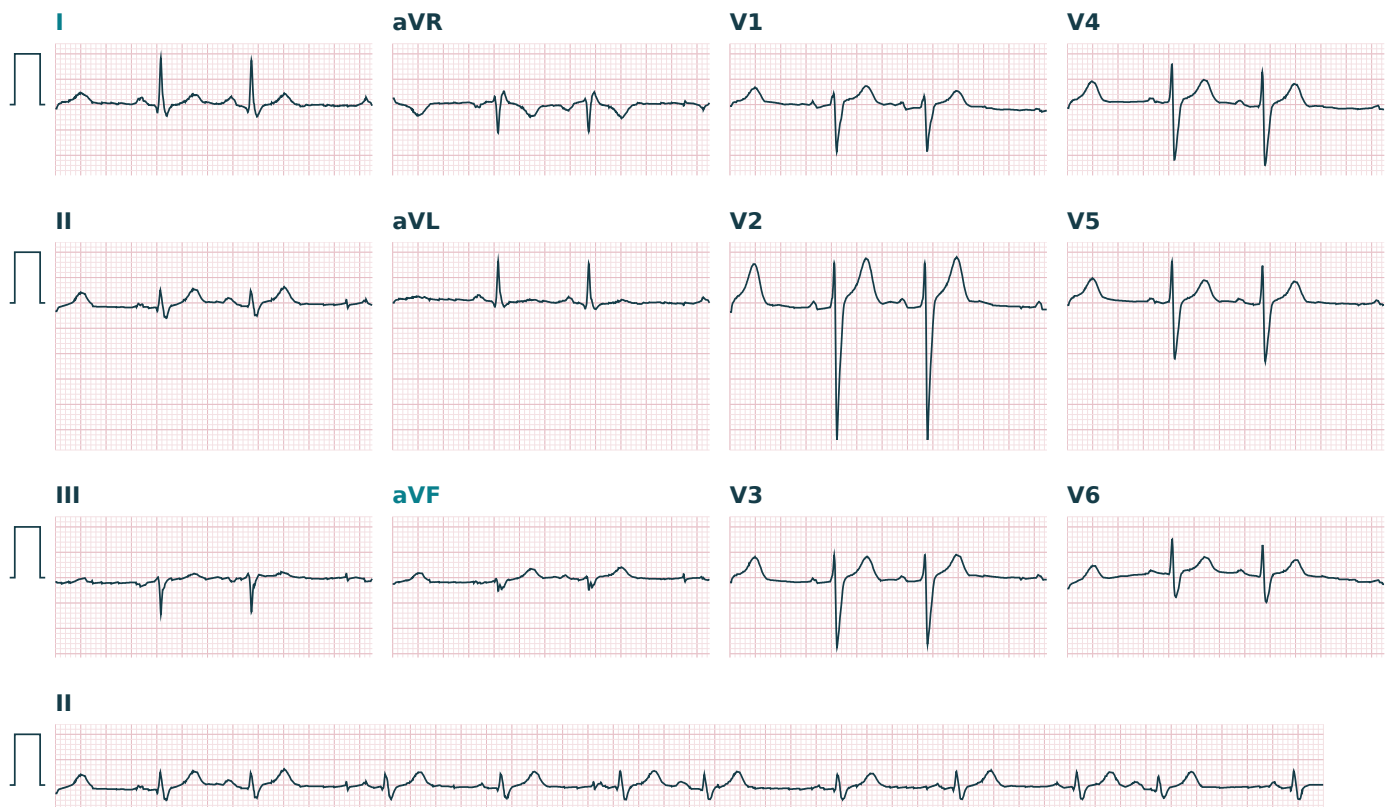
CASE 08 | FOUNDATIONS

Axis: Follow the Net Direction

CLINICAL PRESENTATION

A stable patient's ECG is flagged "left axis deviation". Lead I is positive and inferior leads are negative. The clinician first describes direction before deciding what caused it.

Which quadrant contains the net ventricular activation?



PTB-XL 02252 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

I and aVL are mainly positive.

2

II and aVF are mainly negative.

3

Use the whole QRS balance, not one small deflection.

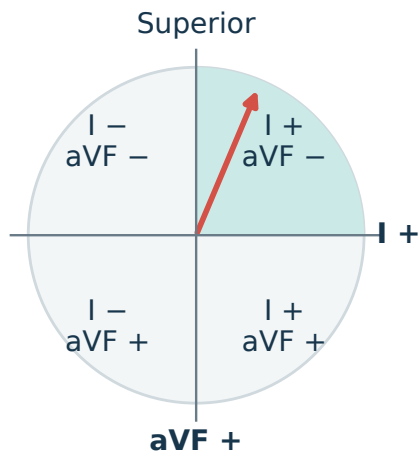
Remember: I: left or right. aVF: down or up.

CASE 08 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

A left-superior frontal QRS direction

Axis: combine the signs to find the direction



Left-superior pattern

I positive

→ Leftward

aVF negative

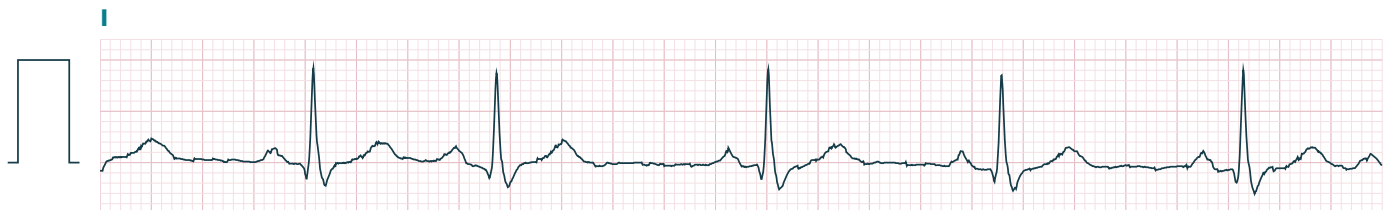
→ Superior

II negative

→ Beyond -30°

Positive I and negative aVF identify the left-superior quadrant. A negative net QRS in II places the axis beyond -30 degrees.

- Positive I points towards the patient's left.
- Negative aVF points superiorly.
- Negative II places this direction beyond the -30 -degree boundary.



PTB-XL 02252 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Confirm electrode placement before accepting an unexpected axis.
- Combine axis with QRS width and morphology.
- Consider age, previous tracings and the clinical setting.

Avoid. Axis describes direction; it does not by itself identify disease.

CASE 08 | ANOTHER REAL ECG

Compare the patterns

A left-superior frontal QRS direction



PTB-XL 02252 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Compare the opposite frontal quadrant.



PTB-XL 07031 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- I is mainly negative.
- aVF is positive: net activation points rightward and inferiorly.

Remember. I: left or right. aVF: down or up.

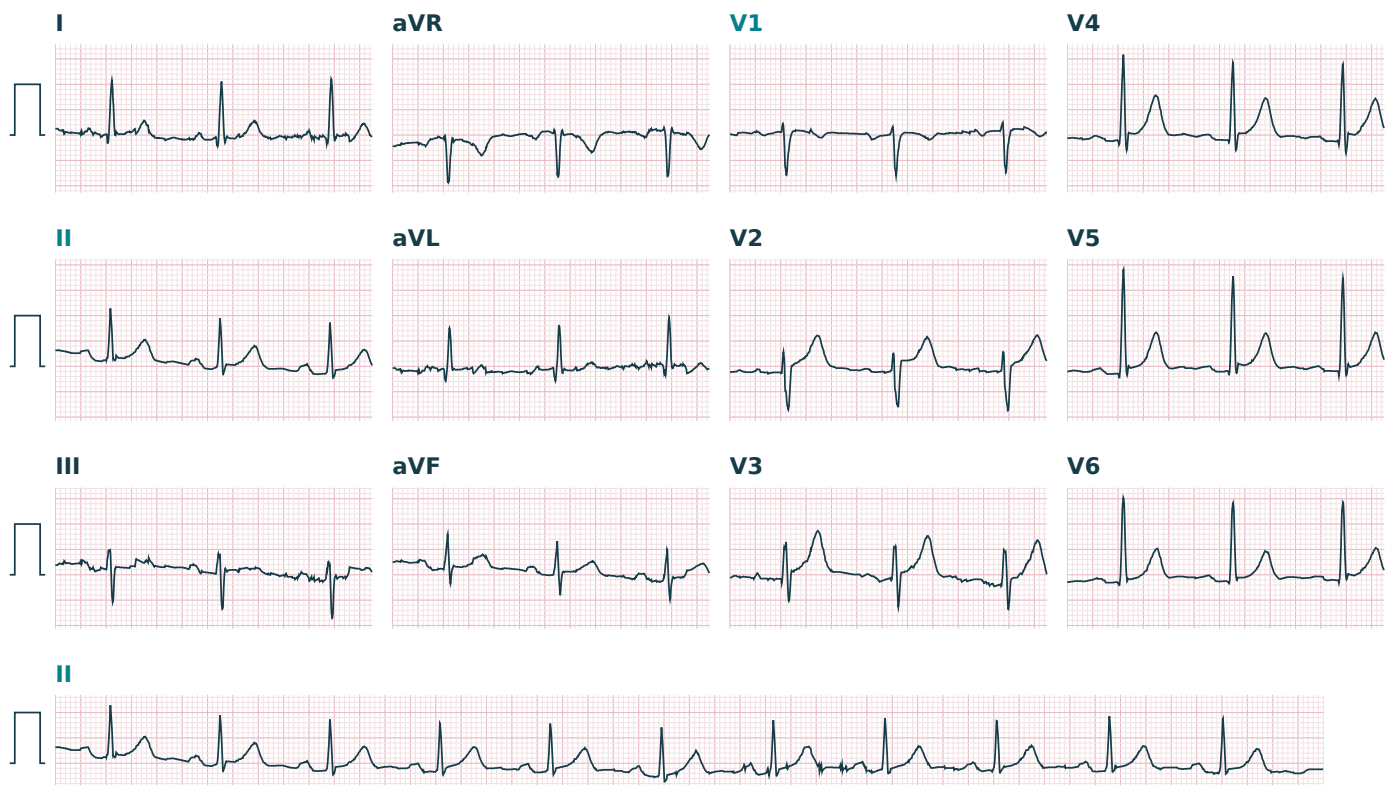
CASE 09 | FOUNDATIONS

PR and QRS Time Different Journeys

CLINICAL PRESENTATION

An asymptomatic patient taking a rate-slowing medicine has a long-looking PR interval. Every P is followed by a QRS. The clinician measures delay without assuming that beats are blocked.

Is conduction delayed before or during ventricular activation?



PTB-XL 03948 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Small P waves precede QRS complexes in II.

2

The gap from P onset to QRS onset is conspicuous.

3

Every visible P is followed by a ventricular complex.

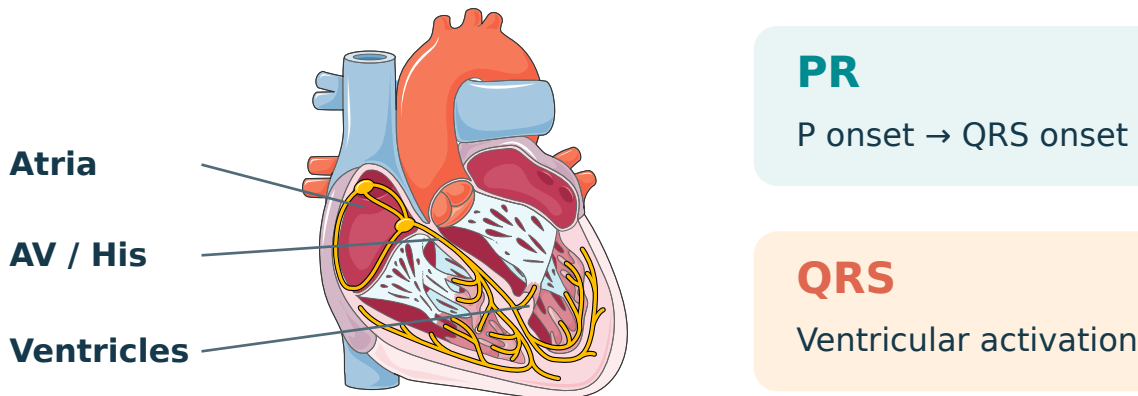
Remember: PR: arrival. QRS: ventricular spread.

CASE 09 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Prolonged atrioventricular conduction with a preserved 1:1 relationship

PR and QRS time different journeys



PR includes more than AV-node delay.

PR and QRS time different portions of the conduction journey. Anatomy: Servier Medical Art, CC BY4.0.

- PR includes atrial activation and conduction to the start of ventricular activation.
- QRS measures spread through the ventricles themselves.
- PR >200 ms with every P conducted defines adult first-degree AV block.



PTB-XL 03948 | 0-5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
 PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Measure several beats and compare previous recordings.
- Review symptoms, rate-slowing medicines and QRS width.
- Syncope, dropped beats or marked conduction disease changes urgency.

Avoid. A long PR does not localise the delay precisely.

CASE 09 | ANOTHER REAL ECG

Compare the patterns

Prolonged atrioventricular conduction with a preserved 1:1 relationship



PTB-XL 03948 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Compare a different ventricular activation contour.



PTB-XL 00658 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- Follow the terminal QRS component in V1.
- Measure PR and QRS separately; their boundaries answer different questions.

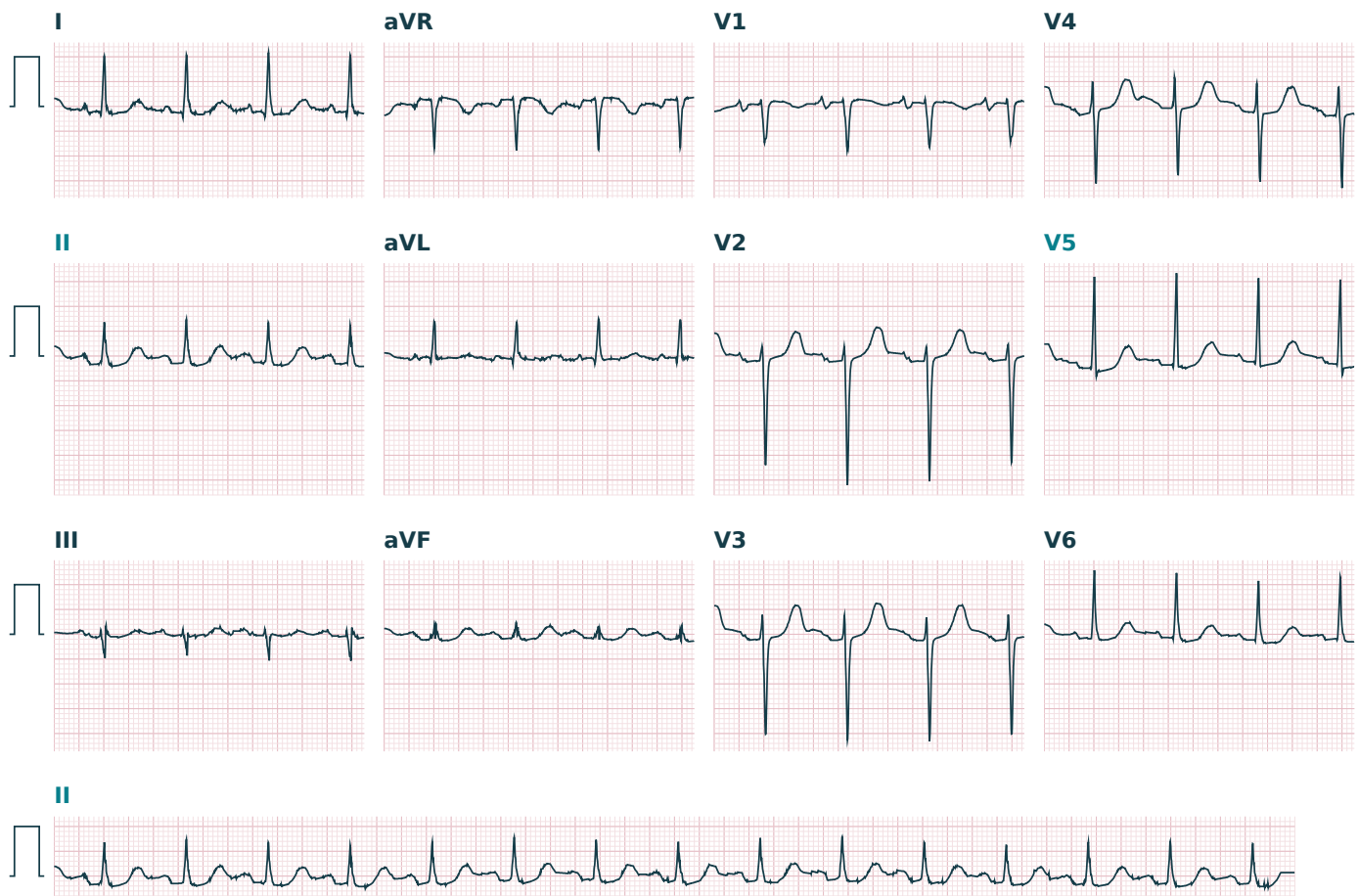
Remember. PR: arrival. QRS: ventricular spread.

CASE 10 | FOUNDATIONS

QT: Find the End Before Correcting

CLINICAL PRESENTATION

A medicine review identifies an unexpectedly long automated QTc. The T wave is small. Before changing treatment, the clinician checks the measured interval, heart rate and correction formula.

Is the QT endpoint correct?

PTB-XL 09894 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise**1**

Compare the terminal T wave in II and V5.

2

Start QT at QRS onset, not QRS end.

3

Inspect several beats and their preceding R-R intervals.

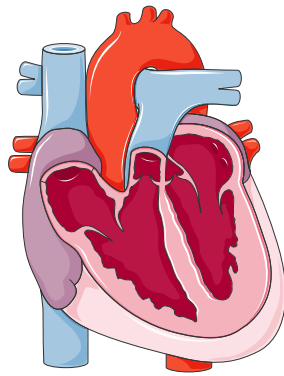
Remember: Correct the measurement before correcting for rate.

CASE 10 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

A QT measurement that needs visual verification

QT includes activation and recovery



Ventricles

QT

QRS

Activation

JT

To the end of T

A wider QRS can lengthen QT without longer repolarisation.

QT includes ventricular activation; delayed activation can lengthen QT. Anatomy: Servier Medical Art, CC BY4.0.

- QT includes ventricular activation and recovery.
- A wider QRS lengthens QT even without longer recovery.
- Rate correction estimates a comparison value; it cannot repair a wrong endpoint.



PTB-XL 09894 | 0-5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Choose a clear T end and exclude a distinct U wave.
- Record QT, RR, QRS width and the correction formula.
- Review symptoms, electrolytes and medicines when QT is prolonged.

Avoid. One automated QTc does not diagnose congenital long-QT syndrome.

CASE 10 | ANOTHER REAL ECG

Compare the patterns

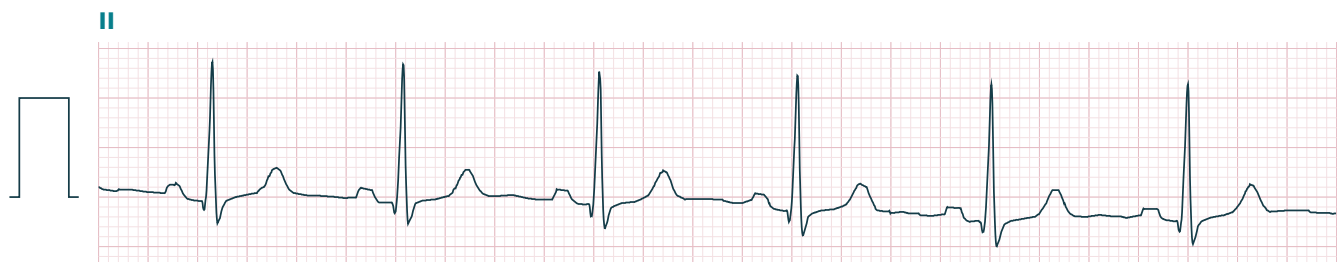
A QT measurement that needs visual verification



PTB-XL 09894 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

A clearer beat helps identify the measurement landmarks.



PTB-XL 00033 | 0.35-5.35 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- Locate QRS onset and the end of T.
- The QT interval includes QRS; it is not the ST segment.

Remember. Correct the measurement before correcting for rate.

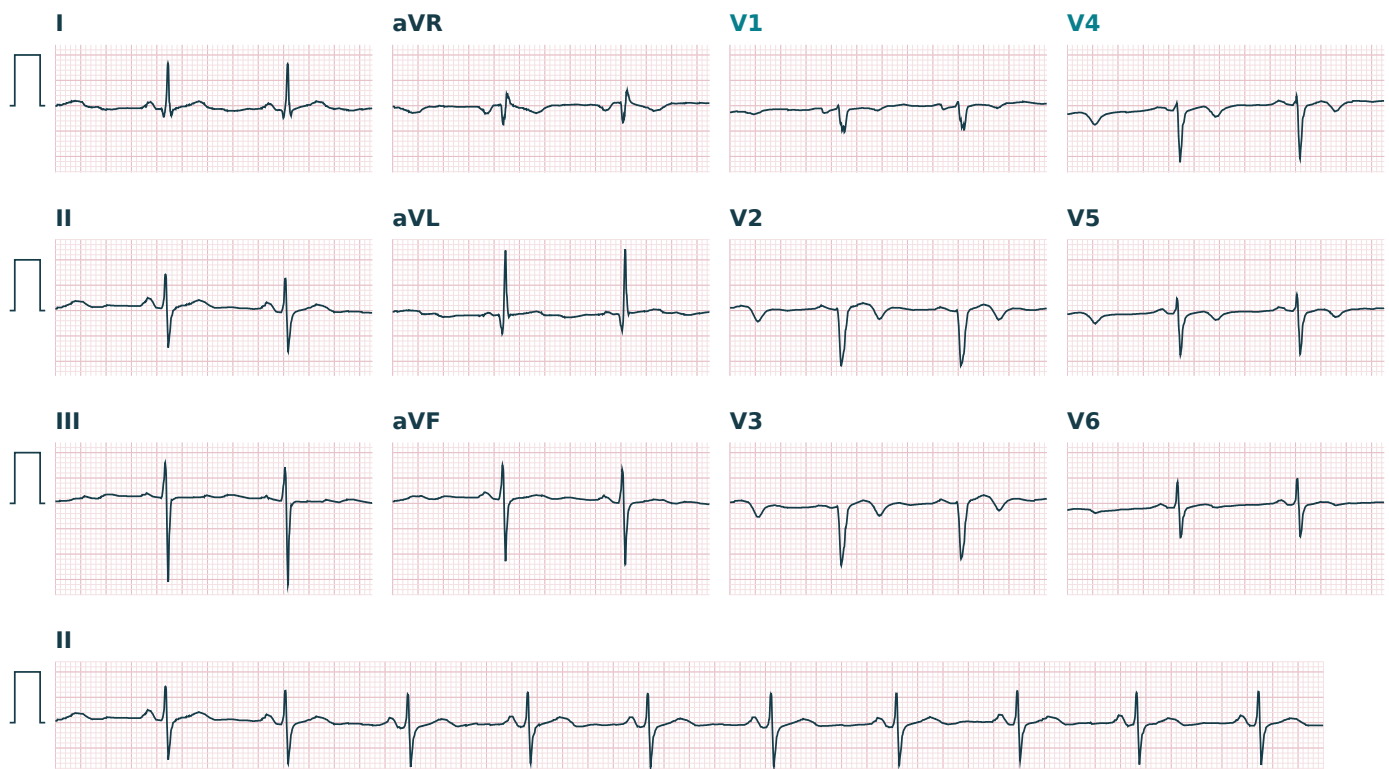
CASE 11 | FOUNDATIONS

R-Wave Progression and Q Waves

CLINICAL PRESENTATION

A machine reports “possible old anterior infarction” in a patient without a recalled infarction. Small anterior R waves prompt a careful description, positioning check and comparison with earlier ECGs.

What does the pattern show, and what remains unproven?



PTB-XL 07790 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Anterior QRS complexes are predominantly negative.

2

Initial R waves are tiny or absent in V1-V4.

3

Anterior T-wave inversion accompanies the QRS pattern.

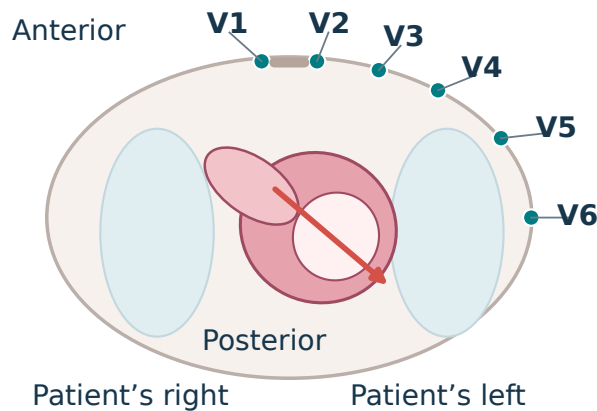
Remember: Describe the forces before naming the disease.

CASE 11 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Poor anterior R-wave progression with regional ST-T changes

R-wave progression: the viewpoint changes



Usual net forces

Away from V1

Towards V6

Same beat.

Different views.

Projected chest-lead views explain why usual net forces point away from V1 and towards V6. The transition varies between individuals.

- Chest leads view ventricular activation from successive directions.
- Loss or redirection of early forces can reduce the initial R wave.
- Infarction, altered conduction and electrode position can each change this pattern.



PTB-XL 07790 | 0.5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

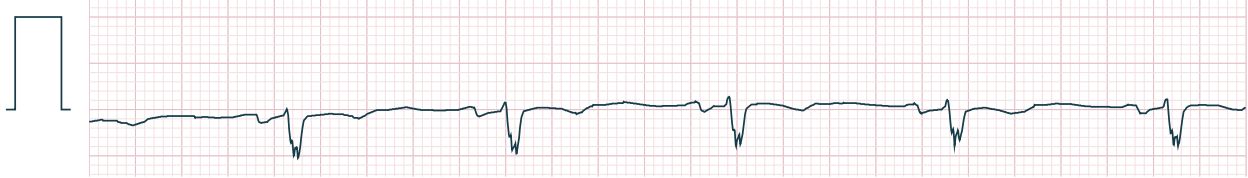
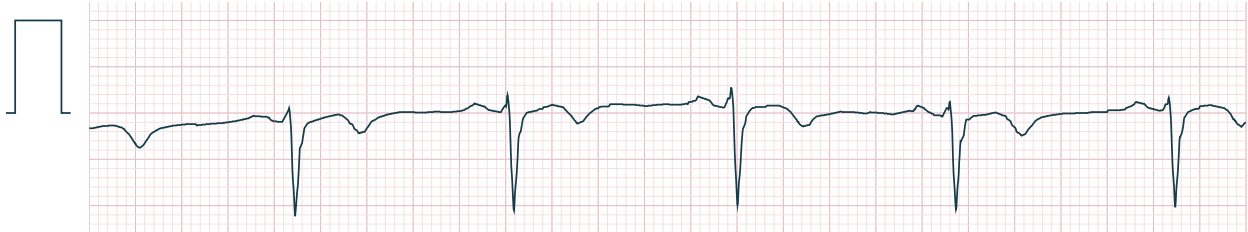
- Check V1-V2 positions and compare a correctly recorded previous ECG.
- Describe Q, QS and ST-T findings in their lead distribution.
- Use symptoms and appropriate imaging or acute investigations to determine the cause.

Avoid. Poor R-wave progression alone does not prove or date an infarction.

CASE 11 | ANOTHER REAL ECG

Compare the patterns

Poor anterior R-wave progression with regional ST-T changes

V1**V4**

PTB-XL 07790 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Compare an initial negative deflection in inferior leads.

III**aVF**

PTB-XL 01577 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- Lead III has a conspicuous initial negative deflection.
- Assess related leads before assigning a cause.

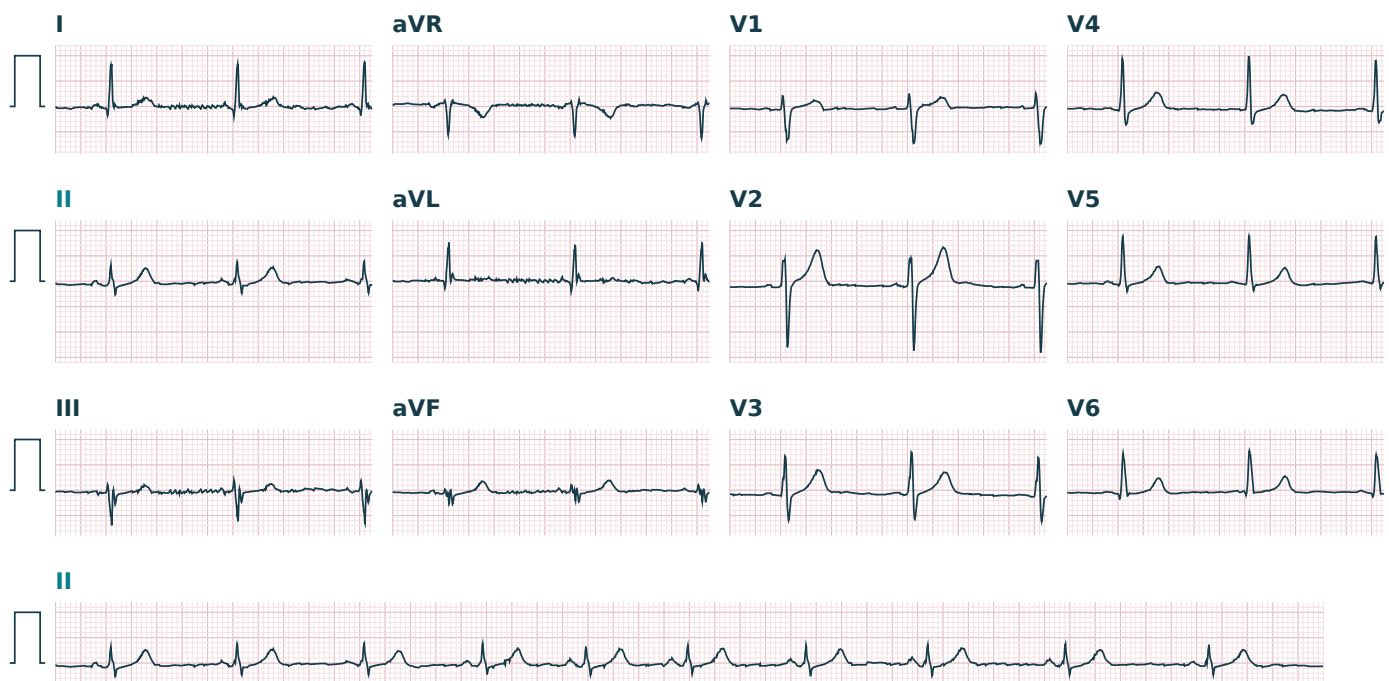
Remember. Describe the forces before naming the disease.

CASE 12 | FOUNDATIONS

A Normal ECG Has Limits

CLINICAL PRESENTATION

A young adult's palpitations have stopped before clinic. The resting ECG shows P waves with gently changing R-R intervals. The clinician must decide whether today's recording explains the episodes.

Has an intermittent arrhythmia been excluded?

PTB-XL 00122 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise**1**

P waves remain visible before QRS complexes.

2

Successive R-R intervals change gradually.

3

The P-QRS sequence persists despite the changing rate.

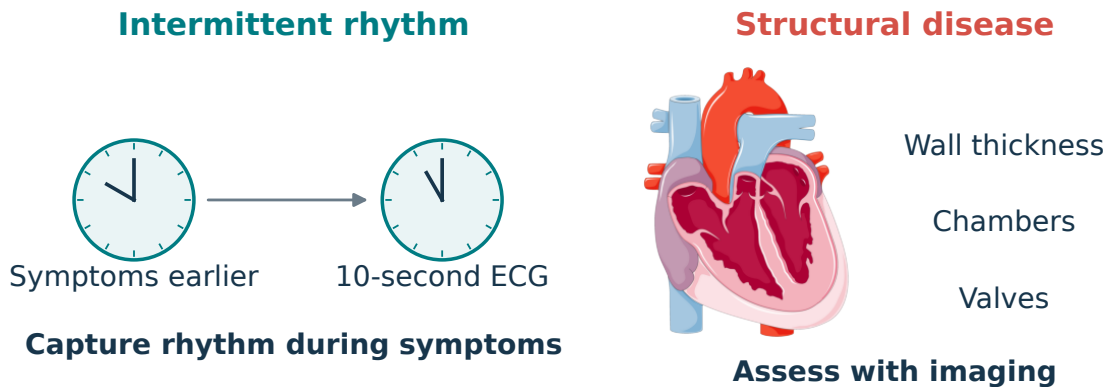
Remember: A recording answers only the time it captures.

CASE 12 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Sinus rhythm with variable cycle length during this recording

A normal resting ECG answers a limited question



Resting ECGs sample electrical activity; symptom-linked recording tests rhythm, and imaging tests structure. Heart adapted from Servier Medical Art, CC BY 4.0.

- Autonomic input changes sinus-node timing.
- A resting ECG samples only a brief period.
- An intermittent rhythm can be absent throughout that sampling window.



PTB-XL 00122 | 0-10 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Ask about onset, duration, frequency, triggers and associated symptoms.
- Match ambulatory monitoring to episode frequency when indicated.
- Exertional syncope or a sudden-death family history warrants prompt assessment.

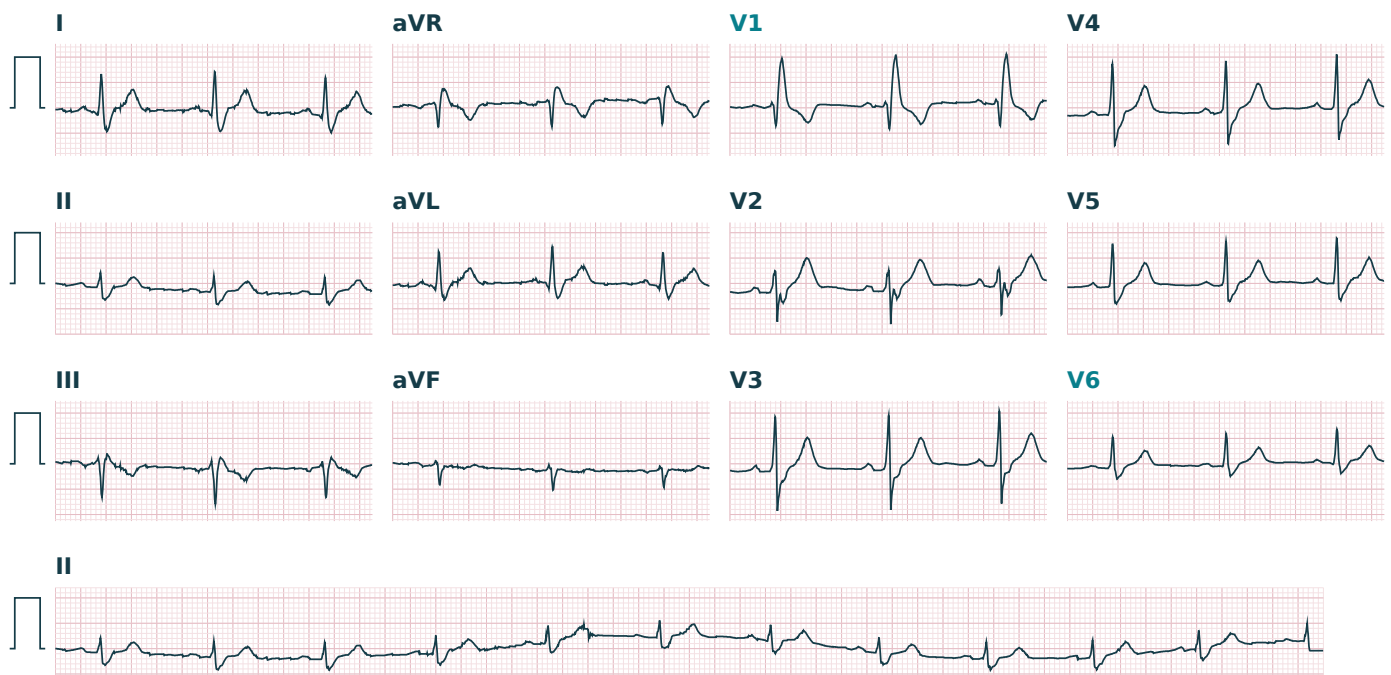
Avoid. A normal ECG between episodes cannot exclude a rhythm occurring only during symptoms.

CASE 13 | CONDUCTION AND STRUCTURE

Right and left bundle-branch block

CLINICAL PRESENTATION

A 62-year-old feels well at a hypertension review. The machine flags a broad QRS. V1 ends upwards; the lateral leads end downwards.

Which ventricle finishes activation late?

PTB-XL 00195 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise**1**

V1 has a prominent terminal positive deflection.

2

I and V6 finish with broad terminal S waves.

3

Assess atrial rhythm separately from the ventricular conduction pattern.

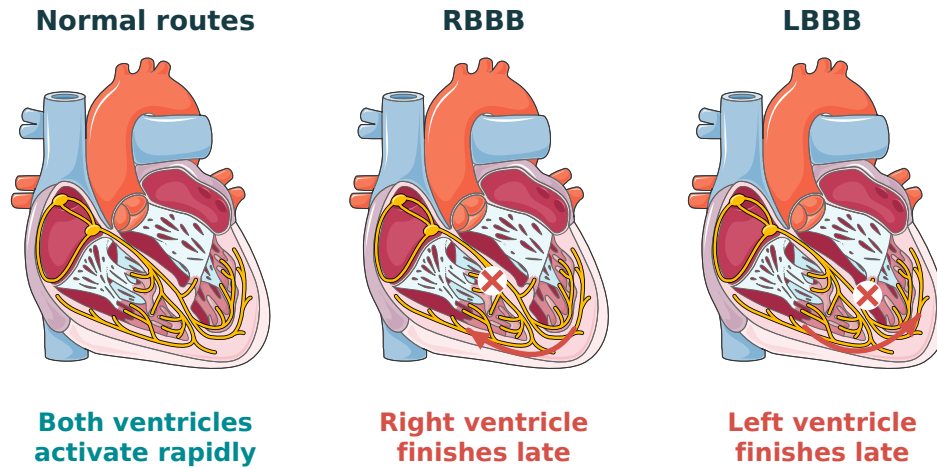
Remember: Rhythm tells when; bundle branches determine how.

CASE 13 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Right bundle-branch block

Bundle-branch block: which ventricle finishes last?



Anatomy: Servier Medical Art, CC BY 4.0. Routes and labels added.

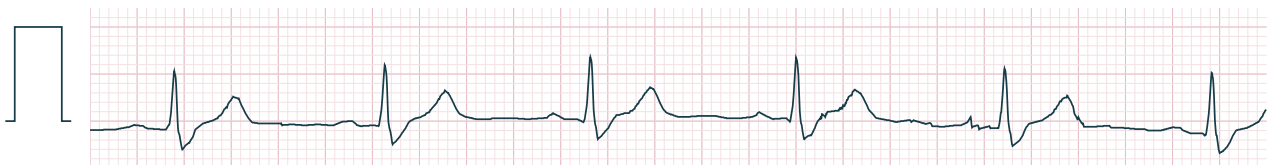
The intact branch activates its ventricle first; slower myocardial spread reaches the delayed ventricle. Routes are simplified.

- The intact left bundle activates the left ventricle first.
- Slower myocardial spread reaches the right ventricle afterwards.
- Late rightward forces point towards V1 and away from lateral leads.

V1



V6



PTB-XL 00195 | 0.5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
 PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Measure QRS: complete adult RBBB requires ≥ 120 ms plus characteristic morphology.
- Compare earlier ECGs; assess symptoms and associated structural disease.
- Syncope, chest pain or haemodynamic compromise requires prompt monitored assessment.

Avoid. A small second peak in V1 alone does not establish RBBB.

CASE 13 | ANOTHER REAL ECG

Compare the patterns

Right bundle-branch block



PTB-XL 00195 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

LBBB reverses the delayed ventricle.



PTB-XL 00618 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

- V1 is predominantly negative.
- V6 has a broad positive QRS.

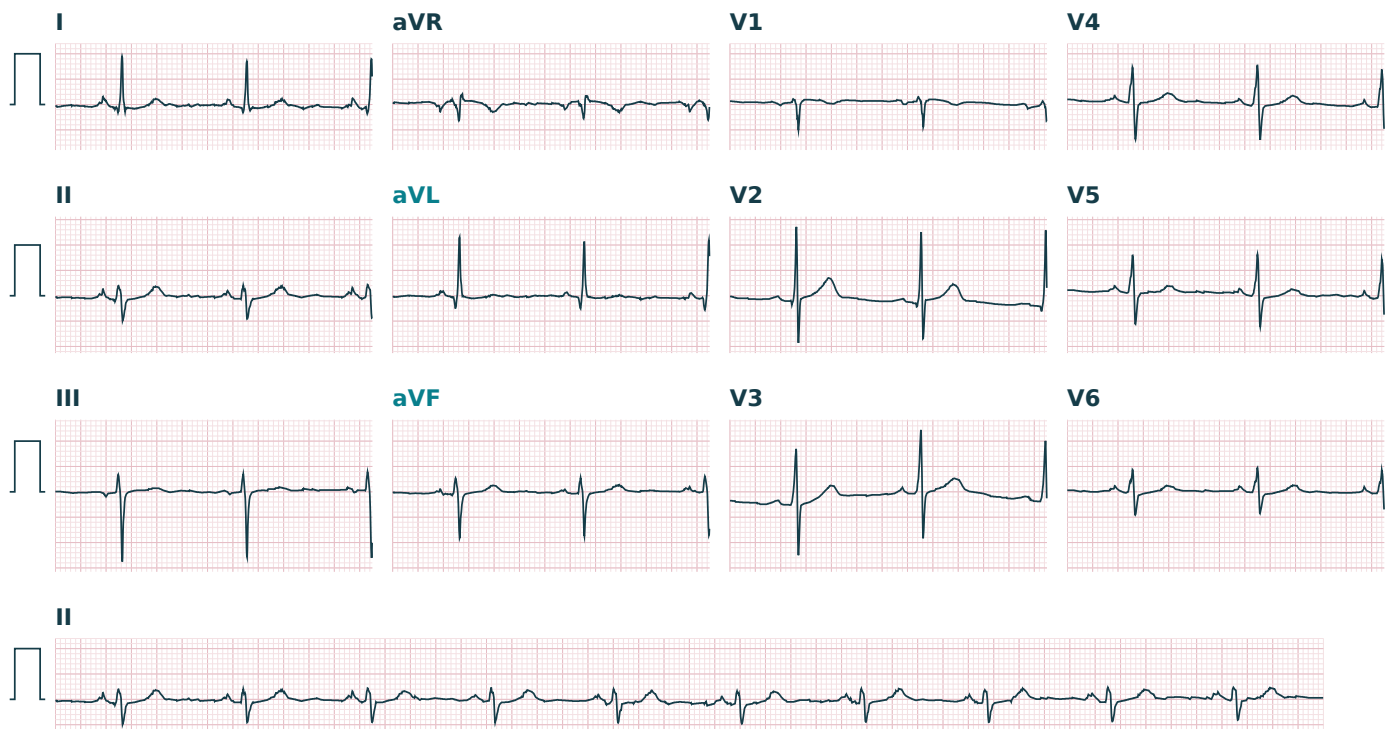
Remember. Rhythm tells when; bundle branches determine how.

CASE 14 | CONDUCTION AND STRUCTURE

Fascicular block: explain the axis

CLINICAL PRESENTATION

A 69-year-old has no fainting or chest pain. Her ECG points leftward, with negative inferior complexes but no striking QRS widening.

Are the inferior deflections Q waves or terminal S waves?

PTB-XL 04444 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise**1**

I and aVL are mainly positive.

2

Inferior complexes begin with small r waves, then deep S waves.

3

Judge the whole QRS direction, not one negative deflection.

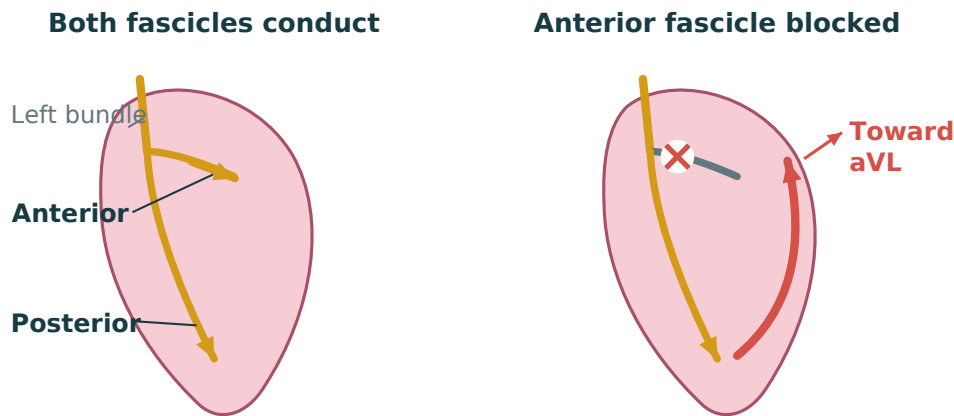
Remember: Read the beginning before naming the negative wave.

CASE 14 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Left anterior fascicular block pattern

LAFB: late activation moves superiorly and leftward



Intact posterior route first → late superior-leftward activation

Original simplified left ventricular pathways in frontal projection.

Posterior fascicular conduction activates the inferior region first; delayed superior-leftward activation produces the characteristic leftward QRS axis.

- The anterior fascicle conducts poorly.
- The intact posterior route activates the inferior ventricle first.
- Late superior-leftward activation shifts the frontal axis towards aVL.



PTB-XL 04444 | 0.5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Confirm the axis and morphology; exclude recording error and alternative causes.
- Compare previous ECGs and assess associated cardiovascular disease.
- Syncope with bifascicular disease warrants prompt rhythm and specialist assessment.

Avoid. RBBB plus LAFB plus long PR does not prove three-fascicle block.

CASE 14 | ANOTHER REAL ECG

Compare the patterns

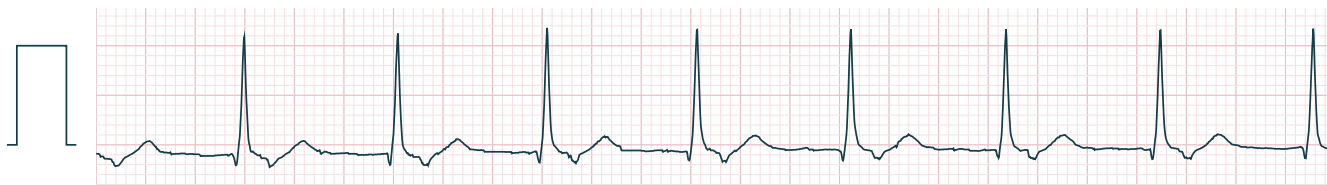
Left anterior fascicular block pattern

aVL**aVF**

PTB-XL 04444 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

A rightward pattern needs different exclusions.

I**aVF**

PTB-XL 00377 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- I is mainly negative; aVF mainly positive.
- Exclude RV disease before calling posterior fascicular block.

Remember. Read the beginning before naming the negative wave.

CASE 15 | CONDUCTION AND STRUCTURE

2:1 AV block: count the missing responses

CLINICAL PRESENTATION

A 76-year-old feels faint while walking. His pulse is slow. Small atrial deflections continue between broad, widely spaced ventricular complexes.

Are the atria slow, or are alternate impulses blocked?



MIT-BIH 231 | source 1211 s | 0-12 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise**1**

P waves occur more often than QRS complexes.

2

Alternate atrial impulses have no following ventricular response.

3

The conducted ventricular complexes are broad.

Remember: Count P waves before counting missing QRS complexes.

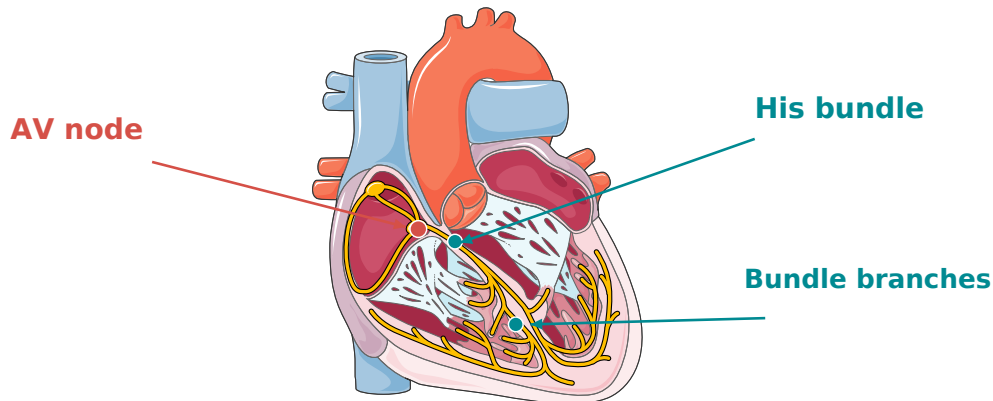
References: C08, X13A, C07, V4-MIT-DIR

CASE 15 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

2:1 AV block with broad ventricular complexes

AV block can interrupt conduction at different levels

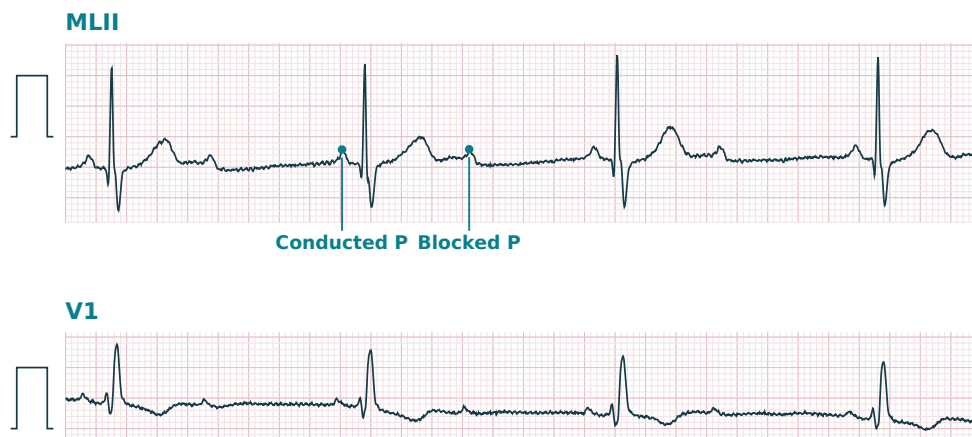


Atrial impulses may continue while ventricular responses fail.

Anatomy: Servier Medical Art, CC BY 4.0. Routes and labels added.

The AV node and His-Purkinje system are different possible sites of failed atrioventricular conduction.

- The atria generate regular impulses.
- Only alternate impulses reach the ventricles.
- Ventricular rate falls to half the atrial rate.



MIT-BIH 231 | source 1211 s | 0-6 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

- Assess perfusion, monitor continuously and obtain urgent senior assistance.
- Review rate-slowing medicines, electrolytes and possible acute ischaemia.
- Treat haemodynamic compromise through the emergency bradycardia pathway without delaying for subtype labels.

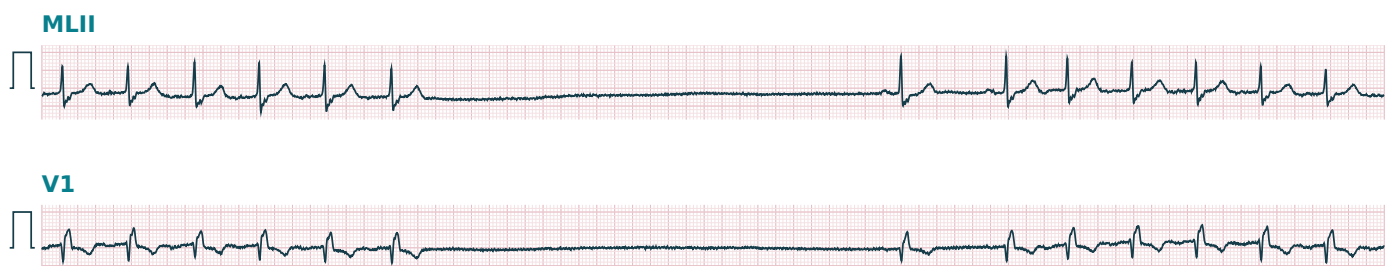
Avoid. An isolated 2:1 pattern cannot reliably distinguish Mobitz I from Mobitz II.

CASE 16 | CONDUCTION AND STRUCTURE

A pause: look upstream before naming it

CLINICAL PRESENTATION

A 73-year-old reports dizziness after palpitations. A monitor records a long pause. The symptom diary has not yet been matched to the event.

Do P waves continue through the ventricular pause?

MIT-BIH 232 | source 1414 s | 0-15 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise**1**

The long interval appears in both recorded channels.

2

Inspect atrial activity before, during and after the gap.

3

The longest displayed R-R interval is approximately 5.69 seconds.

Remember: Look for the atria inside the gap.

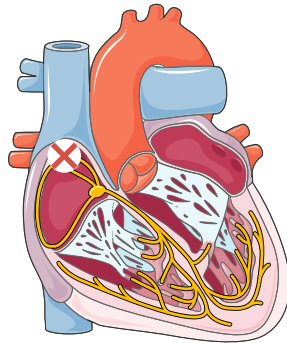
CASE 16 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Prolonged ventricular pause; identify its atrial mechanism

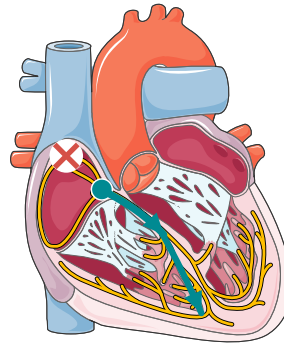
Sinus pause: escape starts from a slower pacemaker

Sinus activation pauses



No sinus impulse reaches the atria

A junctional focus escapes

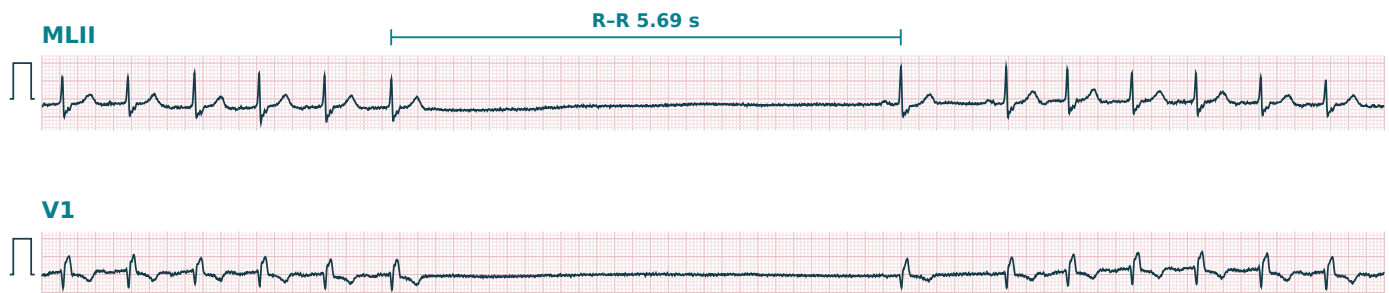


A slower backup impulse activates the ventricles

Anatomy: Servier Medical Art, CC BY 4.0. Routes and labels added.

If sinus firing fails, a slower subsidiary pacemaker may generate an escape beat through the downstream conduction system.

- Sinus failure removes the usual initiating impulse.
- Alternatively, continuing atrial impulses may fail to conduct.
- A recovering pacemaker or escape focus restores ventricular activation.



MIT-BIH 232 | source 1414 s | 0-15 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

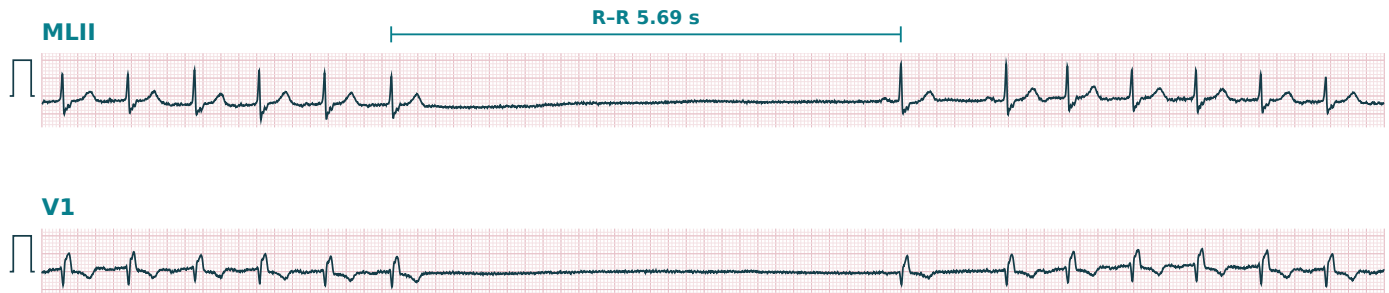
- Match the symptom time to the actual recording.
- Review rate-slowing medicines, electrolytes and other reversible causes.
- Symptomatic prolonged pauses or compromised circulation require prompt monitored assessment.

Avoid. A pause duration alone neither proves sinus arrest nor determines the need for pacing.

CASE 16 | ANOTHER REAL ECG

Compare the patterns

Prolonged ventricular pause; identify its atrial mechanism



MIT-BIH 232 | source 1414 s | 0-15 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Slow sinus rhythm still conducts every visible P wave.



PTB-XL 00611 | 0-10 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- P precedes each QRS.
- Slowness alone does not establish sinus-node disease.

Remember. Look for the atria inside the gap.

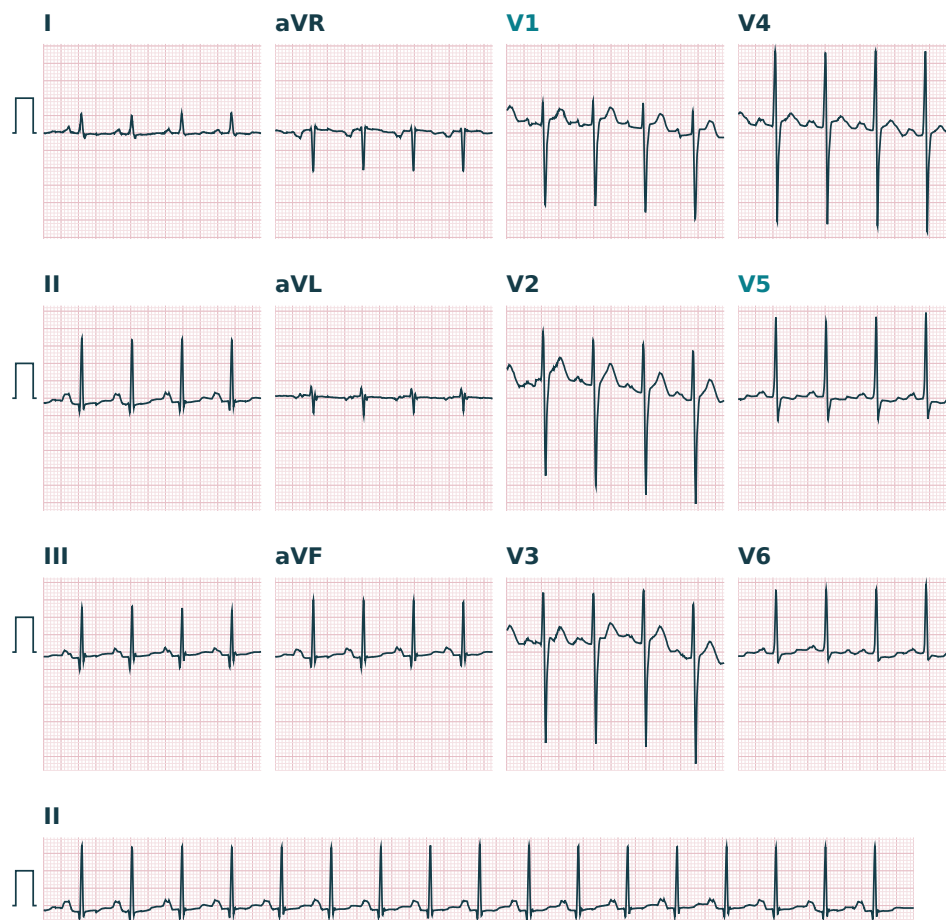
CASE 17 | CONDUCTION AND STRUCTURE

High voltage: electrical clue, anatomical question

CLINICAL PRESENTATION

A 58-year-old with poorly controlled hypertension becomes breathless on stairs. The chest leads show deep S waves and tall lateral R waves.

Does high voltage directly measure ventricular wall thickness?



PTB-XL 05911 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

V1 has a deep S wave.

2

V5 and V6 have tall positive R waves.

3

Inspect lateral ST segments and T waves separately.

Remember: Voltage suggests; imaging measures.

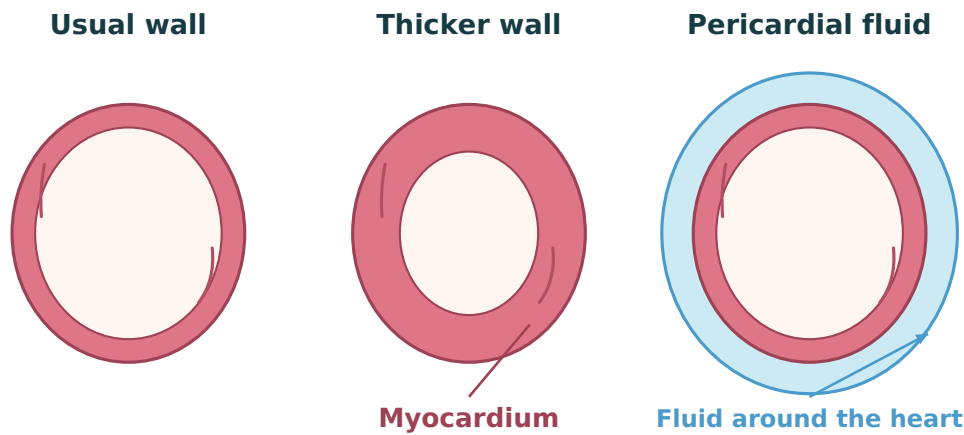
References: C02, C04, S01, S06, PTBXL

CASE 17 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

High QRS voltage consistent with LVH criteria

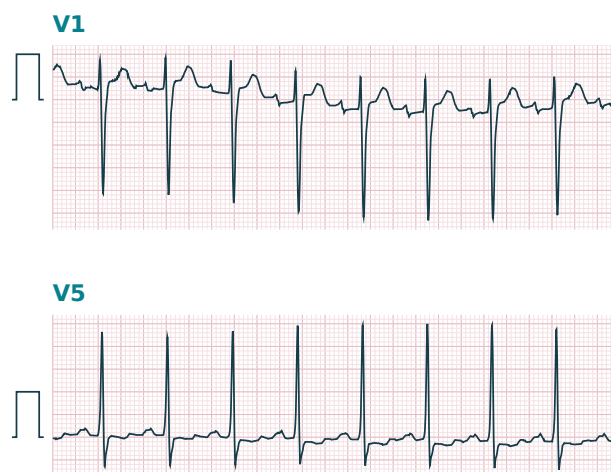
Wall thickness and fluid affect the recorded signal



Original simplified short-axis sections; dimensions are illustrative.

Hypertrophy changes myocardial thickness. Pericardial fluid changes the tissue surrounding the heart. ECG voltage is influenced by both source and transmission.

- Ventricular muscle generates the depolarisation forces.
- More muscle and different geometry can increase surface voltage.
- The chest and electrode position also affect the recorded amplitude.



PTB-XL 05911 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

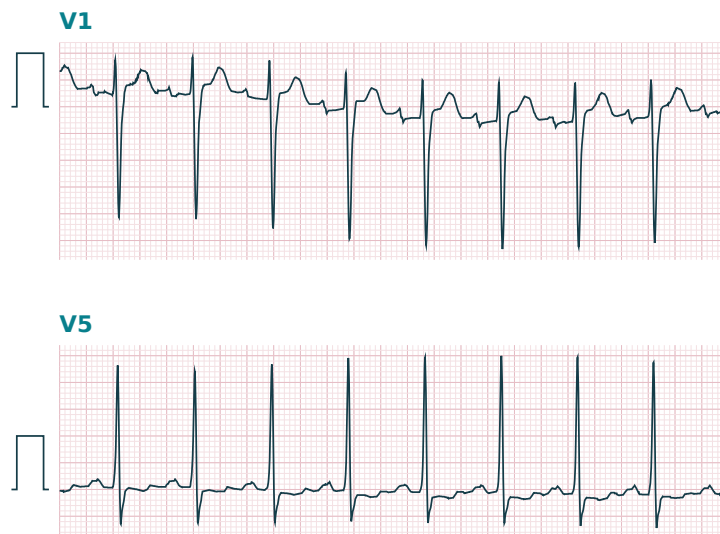
- Check gain before applying a voltage threshold.
- Assess blood pressure, symptoms and the complete ECG.
- Use echocardiography to assess wall thickness, chamber size, function and valve disease.

Avoid. High voltage does not by itself diagnose hypertrophic cardiomyopathy.

CASE 17 | ANOTHER REAL ECG

Compare the patterns

High QRS voltage consistent with LVH criteria



PTB-XL 05911 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

A normal reference at the same magnification.



PTB-XL 00033 | 0.35-5.35 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

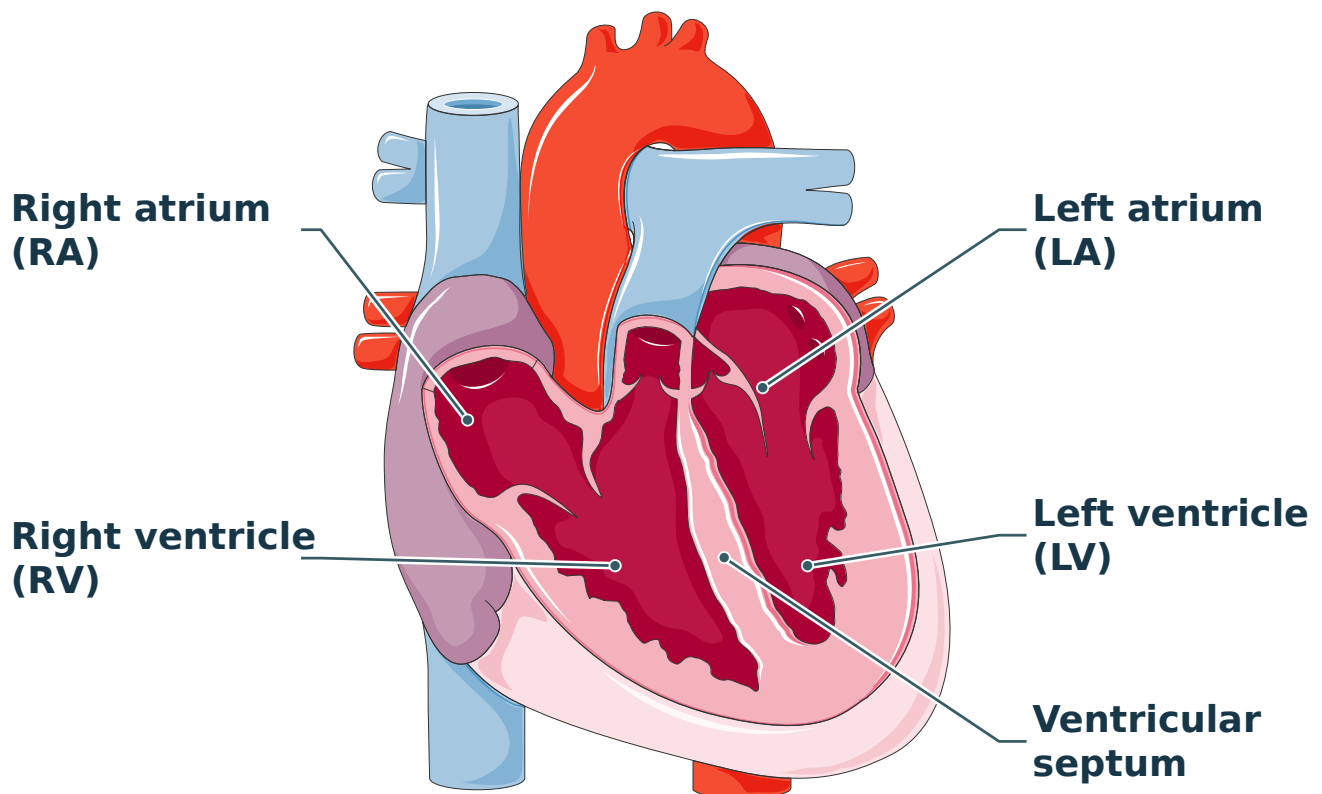
- The normal reference has smaller precordial voltages.
- High voltage alone does not establish ventricular hypertrophy.

Remember. Voltage suggests; imaging measures.

ANATOMY AT A GLANCE

Four-chamber heart cutaway

Four chambers; one electrical sequence



Anatomical right is on the viewer's left.
Chamber shape and wall thickness are schematic.

Connect it to the ECG. Voltage suggests; imaging measures.

- Ventricular muscle generates the depolarisation forces.
- More muscle and different geometry can increase surface voltage.
- The chest and electrode position also affect the recorded amplitude.

Adapted from Servier Medical Art, CC BY 4.0. Teaching labels added.

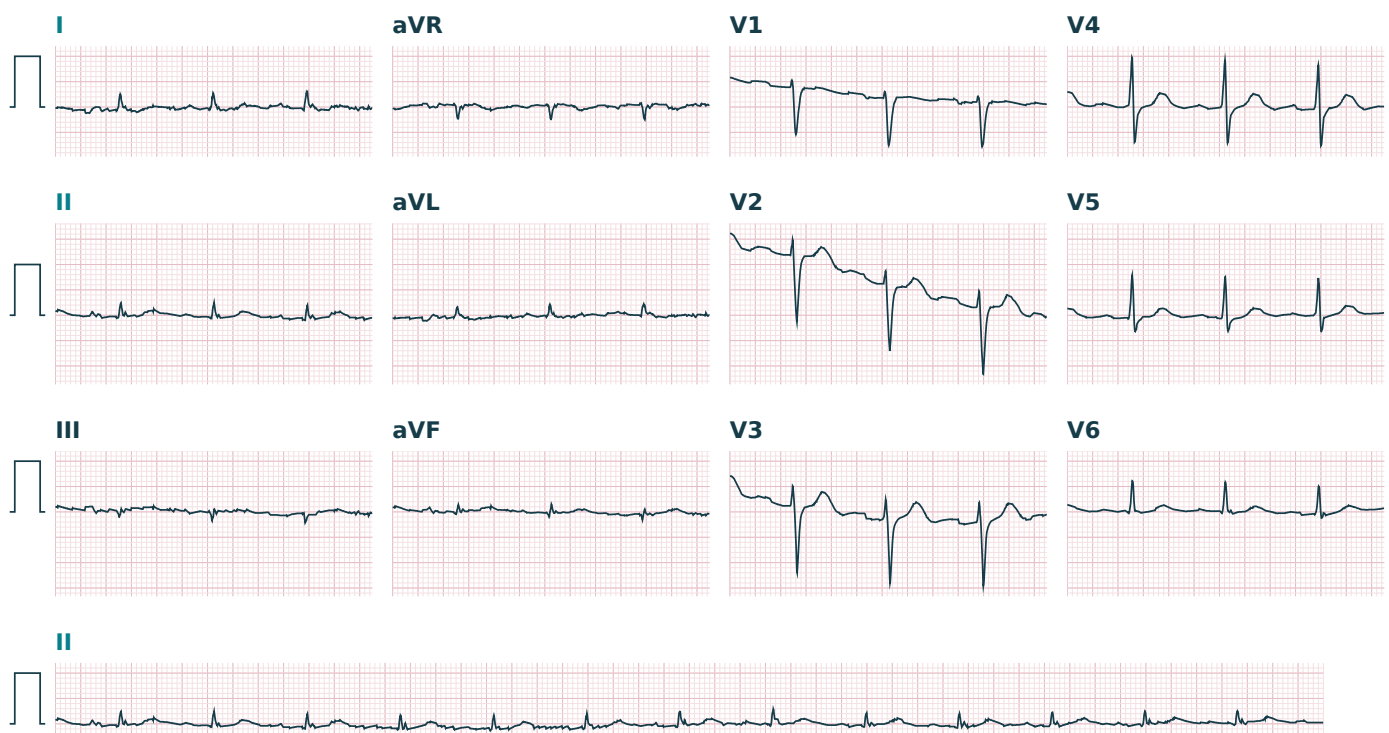
CASE 18 | CONDUCTION AND STRUCTURE

Low voltage: examine the source and surroundings

CLINICAL PRESENTATION

A 67-year-old develops oedema and breathlessness. Echocardiography shows thick ventricular walls, yet limb-lead QRS complexes are unexpectedly small.

What might explain the voltage-wall thickness mismatch?



PTB-XL 00442 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

QRS amplitudes are small across the limb leads.

2

Measure peak to trough, not only the R wave.

3

Compare chest-lead voltage and the previous ECG.

Remember: Small signals demand a bigger clinical picture.

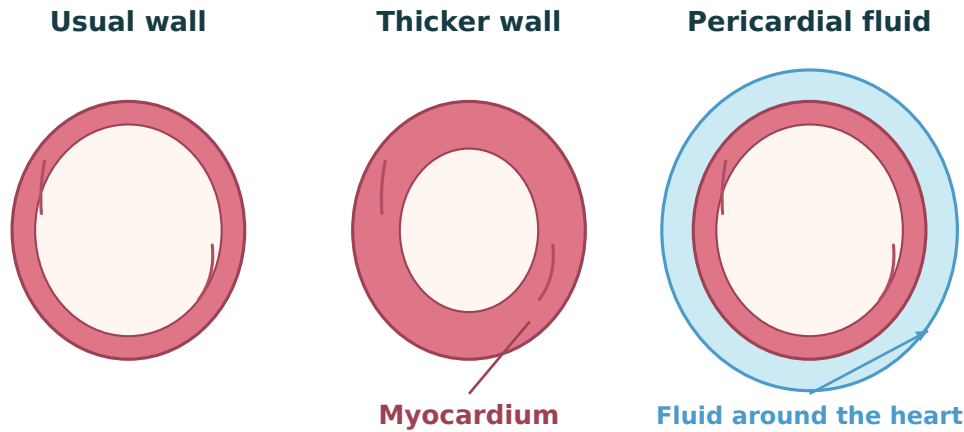
References: C02, C11, E25-MYO, P36D, PTBXL

CASE 18 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Low limb-lead QRS voltage

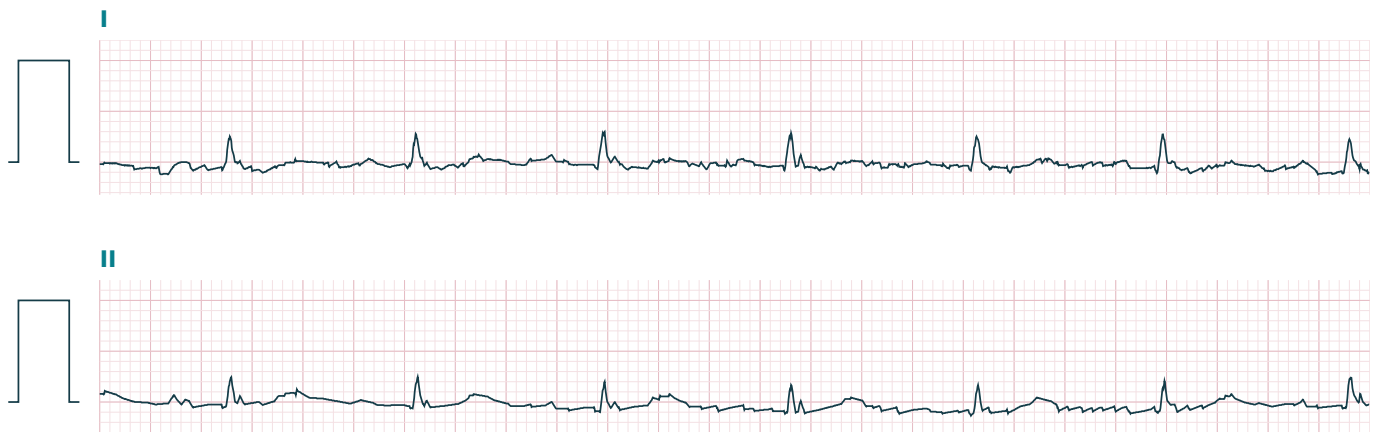
Wall thickness and fluid affect the recorded signal



Original simplified short-axis sections; dimensions are illustrative.

Hypertrophy changes myocardial thickness. Pericardial fluid changes the tissue surrounding the heart. ECG voltage is influenced by both source and transmission.

- The myocardium generates the signal.
- Fluid or intervening tissue can reduce its surface expression.
- Infiltrative myocardial disease can combine thick walls with small electrical signals.



PTB-XL 00442 | 0.5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

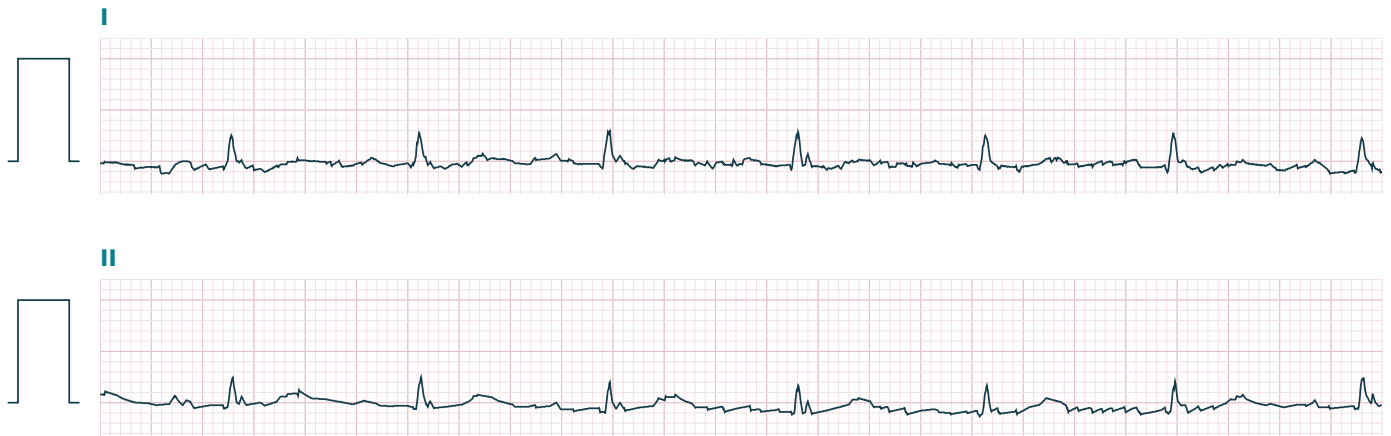
- Confirm calibration and electrode placement.
- Assess ventricular structure, function and pericardial fluid with echocardiography.
- Hypotension with worsening breathlessness requires urgent assessment, including possible tamponade.

Avoid. Low voltage is a pattern with several causes, not a diagnosis of amyloidosis.

CASE 18 | ANOTHER REAL ECG

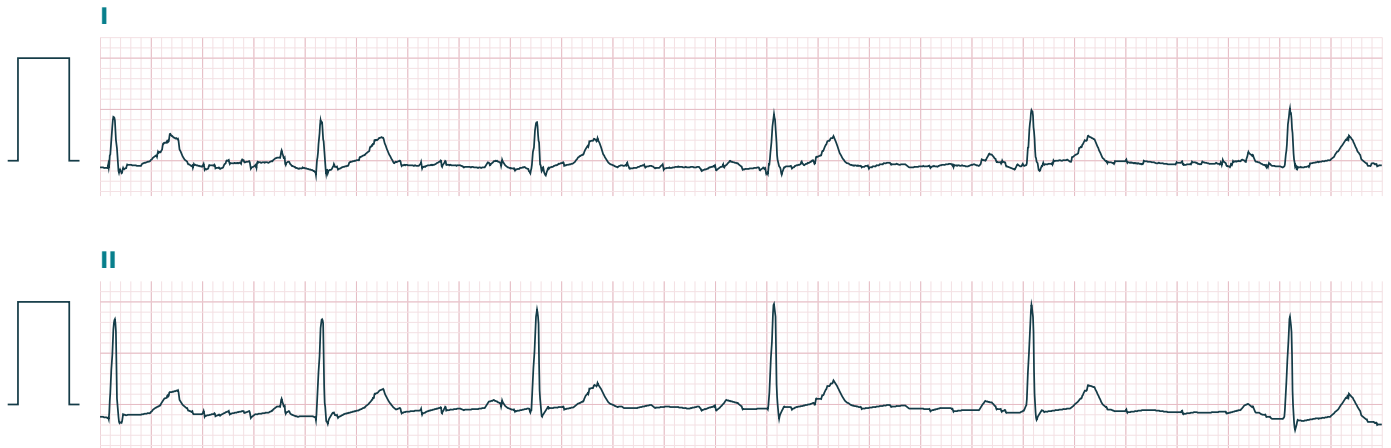
Compare the patterns

Low limb-lead QRS voltage



PTB-XL 00442 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

A normal comparison shows larger limb-lead complexes.



PTB-XL 00010 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

- Normal limb-lead complexes are larger at the same magnification.
- Check all six limb leads before reporting low limb-lead voltage.

Remember. Small signals demand a bigger clinical picture.

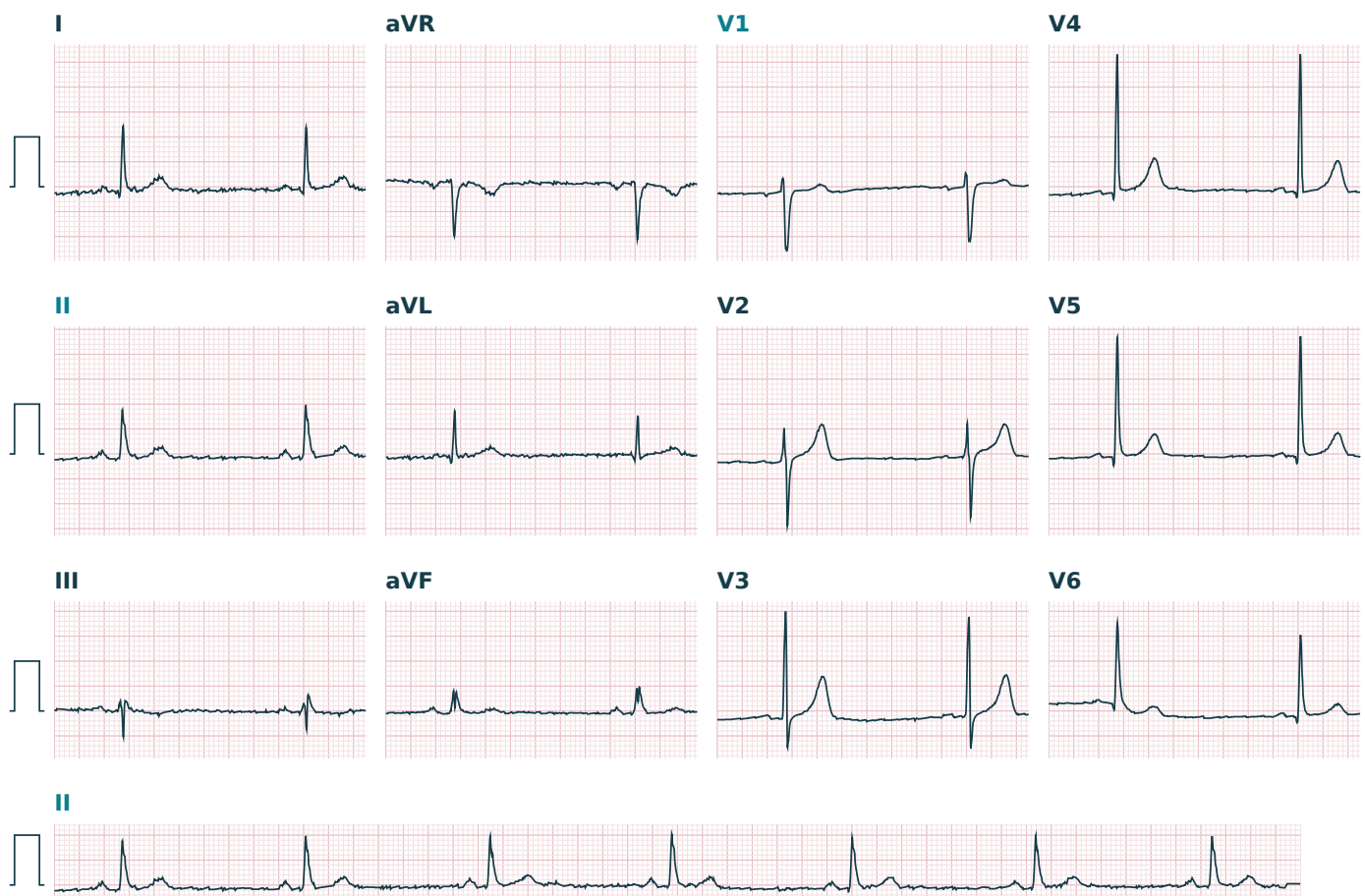
CASE 19 | CONDUCTION AND STRUCTURE

Sinus bradycardia: rate is not the diagnosis

CLINICAL PRESENTATION

A trained 28-year-old has a resting pulse of 42/min during a routine examination. There is no syncope, exercise intolerance or rate-slowing medication.

Is this an appropriate sinus response or symptomatic disease?



PTB-XL 00611 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Upright P waves precede each QRS in lead II.

2

Ventricular complexes are widely spaced.

3

The P-QRS relationship remains consistent.

Remember: A slow clock can still work correctly.

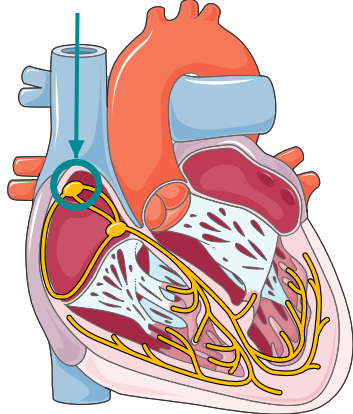
CASE 19 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Sinus bradycardia

Sinus bradycardia: a slower sinus clock

Sinus node



**Longer interval
between impulses**

The P-QRS sequence is preserved.

A slower sinus clock can preserve one-to-one conduction. Anatomy adapted from Servier Medical Art, CC BY 4.0.

- The sinus node remains the initiating pacemaker.
- Autonomic input slows impulse formation.
- AV conduction preserves one atrial impulse for each ventricular response.



PTB-XL 00611 | 0-10 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Ask about syncope, exertional symptoms and medicines.
- Assess reversible causes when the clinical context suggests disease.
- Correlate symptoms with rhythm before diagnosing symptomatic sinus-node dysfunction.

Avoid. A low resting rate alone is not a pacing indication.

References: C08, X13A, S06, PTBXL

CASE 19 | ANOTHER REAL ECG

Compare the patterns

Sinus bradycardia



PTB-XL 00611 | 0-10 s | 25 mm/s, 10 mm/mV proportions

Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Sinus arrhythmia changes timing while preserving atrial origin.



PTB-XL 00122 | 0-10 s | 25 mm/s, 10 mm/mV proportions

Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- P waves still precede QRS complexes.
- R-R intervals change gradually.

Remember. A slow clock can still work correctly.

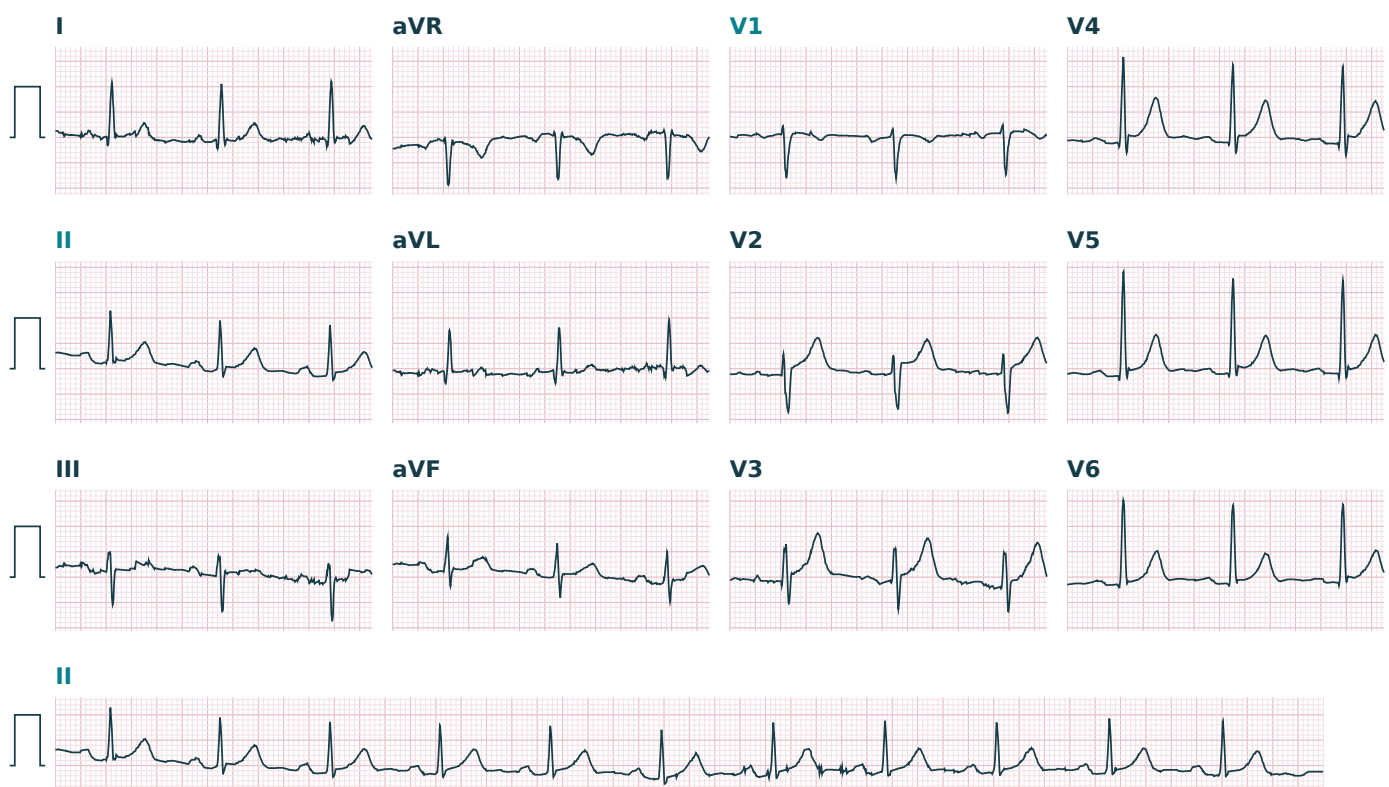
CASE 20 | CONDUCTION AND STRUCTURE

First-degree AV block: delayed, not dropped

CLINICAL PRESENTATION

A 64-year-old taking a rate-slowing medicine attends review. Each P wave conducts, but the interval before ventricular activation is long. There is no syncope.

Is conduction delayed or intermittently failing?



PTB-XL 03948 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Each visible P wave is followed by a QRS.

2

The PR interval is prolonged.

3

No dropped ventricular response defines this pattern.

Remember: Delayed delivery is not a missing delivery.

References: C02, C08, X13A, PTBXL

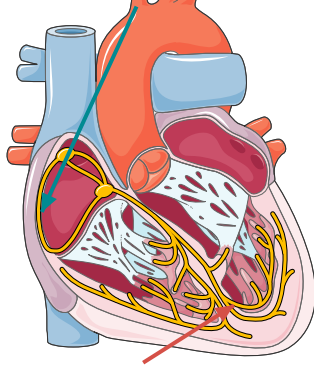
CASE 20 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

First-degree AV block

First-degree block: delayed AV conduction

P: atrial muscle



Ventricles start QRS

Atrial activation



AV node + His-Purkinje



Ventricular activation

Delay can occur at more than one level.

PR spans atrial and AV-system conduction; it does not locate delay. Servier Medical Art, CC BY 4.0.

- The atria activate first.
- Conduction takes longer between atrial onset and ventricular onset.
- Every impulse still reaches the ventricles.



PTB-XL 03948 | 0-5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
 PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Measure PR from P onset to QRS onset; >200 ms is prolonged in adults.
- Review symptoms, medicines and the previous ECG.
- Assess marked delay or associated conduction disease in clinical context.

Avoid. A prolonged PR does not locate the delay specifically within the AV node.

CASE 20 | ANOTHER REAL ECG

Compare the patterns

First-degree AV block



PTB-XL 03948 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

Normal PR timing provides a useful comparison.



PTB-XL 00033 | 0.35-5.35 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

- P precedes every QRS.
- Measure the interval using the time grid.

Remember. Delayed delivery is not a missing delivery.

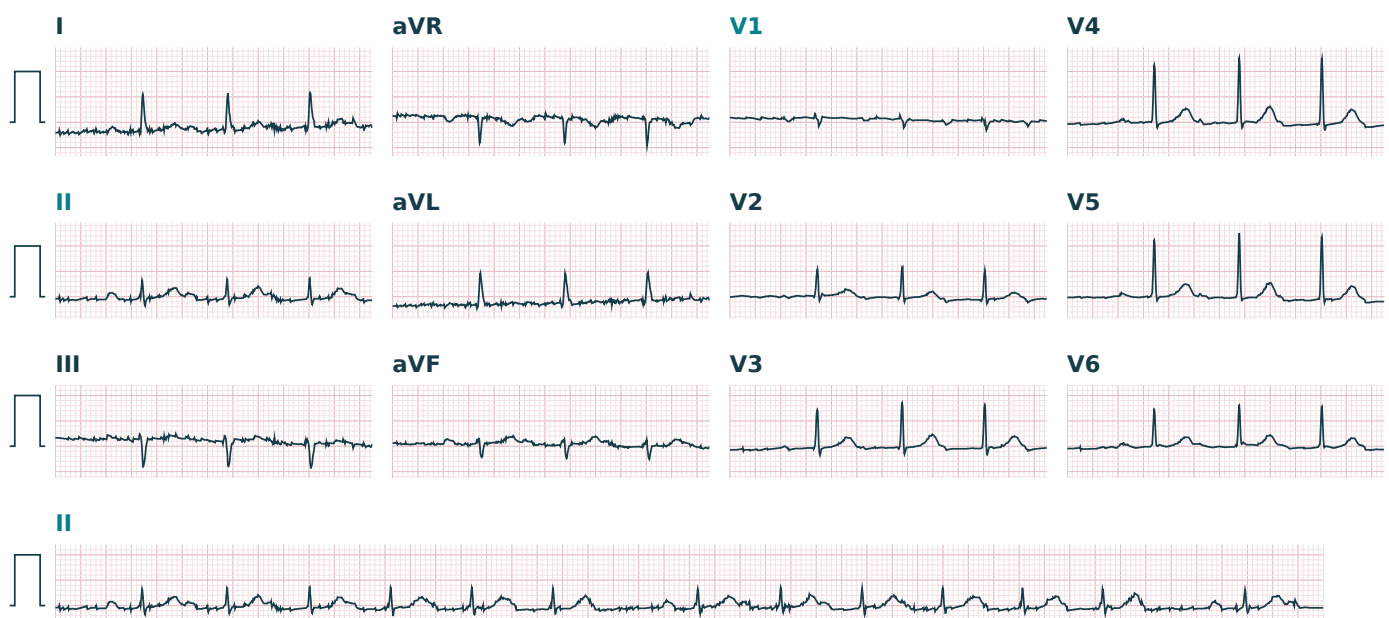
CASE 21 | CONDUCTION AND STRUCTURE

Wenckebach: delay increases, then conduction fails

CLINICAL PRESENTATION

A young adult's overnight monitor shows grouped beats. The team finds changing PR intervals and one atrial impulse without a ventricular response. There are no daytime symptoms.

What happens to conduction before and after the dropped beat?



PTB-XL 14009 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Ventricular beats occur in groups separated by longer intervals.

2

PR delay increases within the conducted sequence.

3

After a nonconducted P wave, the next PR becomes shorter.

Remember: Longer, longer, lost; then shorter.

References: C08, X13A, PTBXL

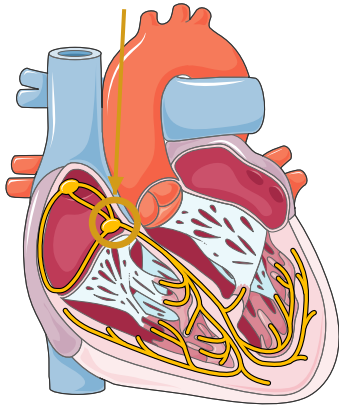
CASE 21 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Second-degree AV block: Wenckebach pattern

Wenckebach: delay, block, recovery

Commonly AV nodal



- → Conduction slows
- → Delay increases
- → One impulse fails
- → Conduction recovers

Typical nodal Wenckebach delays successive impulses before one fails. Anatomy adapted from Servier Medical Art, CC BY 4.0.

- The AV node commonly conducts successive impulses progressively more slowly.
- One impulse arrives before conduction has sufficiently recovered and fails.
- The pause permits recovery, so the next conducted PR shortens.



PTB-XL 14009 | 0-5.5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Confirm the sequence across several beats and inspect P waves carefully.
- Review symptoms, medications and whether the episode occurred during sleep.
- Wide QRS, exercise-related block or symptoms require closer specialist assessment.

Avoid. Wenckebach is often nodal, but its anatomical site cannot always be assumed.

CASE 22 | CONDUCTION AND STRUCTURE

Mobitz II: an impulse fails without warning

CLINICAL PRESENTATION

A 72-year-old reports abrupt light-headedness. A rhythm strip shows a dropped ventricular response among broad conducted complexes. The surrounding atrial rhythm remains regular.

Does PR lengthen before the blocked impulse?



MIT-BIH 231 | source 2 s | 0-12 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise

1

A P wave lacks a following QRS.

2

Conducted PR intervals do not progressively lengthen before the drop.

3

Sinus timing remains stable through the nonconducted P wave.

Remember: A constant PR does not guarantee the next response.

References: C08, X13A, C07, V4-MIT-DIR

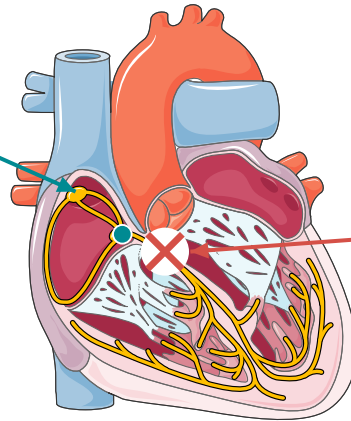
CASE 22 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Second-degree AV block: Mobitz II pattern

Mobitz II: sudden His-Purkinje failure

Atrial impulses
continue regularly



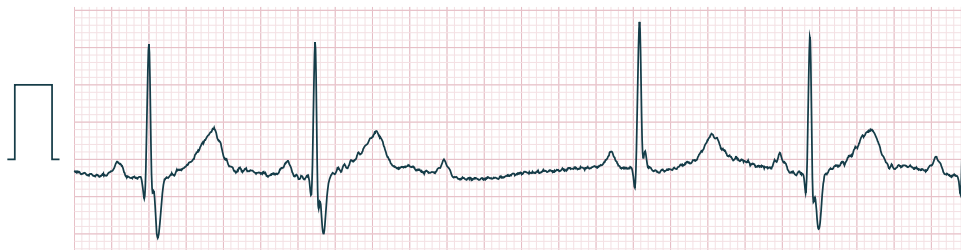
Intermittent
conduction failure

The conducted PR can stay similar before an impulse is blocked.

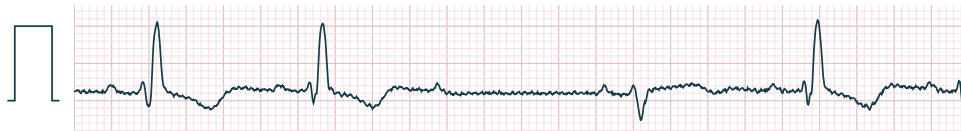
Mobitz II usually reflects diseased His-Purkinje conduction. Anatomy adapted from Servier Medical Art, CC BY 4.0.

- Atrial impulses continue regularly.
- Diseased His-Purkinje tissue can fail abruptly despite stable preceding conduction.
- Loss of ventricular responses can progress to advanced or complete block.

MLII



V1



MIT-BIH 231 | source 2 s | 1.8-6.6 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

- Monitor continuously and seek prompt specialist assessment.
- Check reversible causes, including medicines, electrolyte disturbance and ischaemia.
- Acquired Mobitz II without a reversible cause warrants permanent pacing assessment.

Avoid. Exclude blocked premature atrial beats and vagal slowing before assigning Mobitz II.

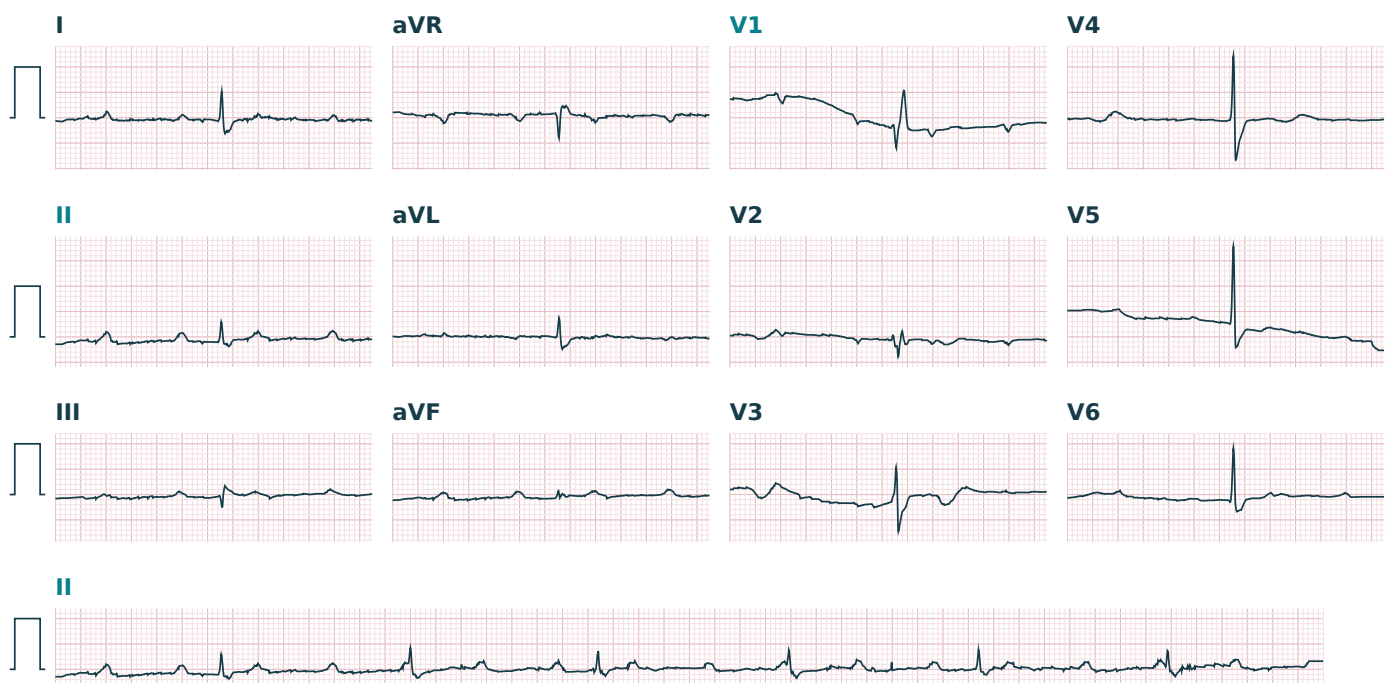
CASE 23 | CONDUCTION AND STRUCTURE

Complete AV block: two independent clocks

CLINICAL PRESENTATION

A 78-year-old collapses while standing. The ECG shows slow ventricular complexes while atrial deflections continue at a faster rate. The team begins monitored assessment.

Is any consistent P-QRS relationship preserved?



PTB-XL 00959 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

P waves occur more frequently than QRS complexes.

2

P waves change position relative to the ventricular complexes.

3

The slower ventricular rhythm runs independently.

Remember: Two clocks; no reliable electrical bridge.

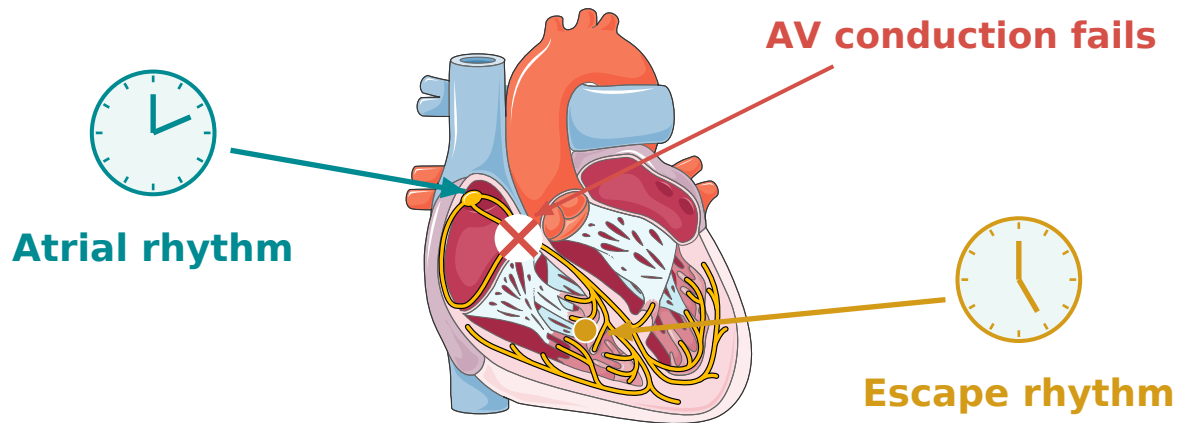
References: C08, X13A, C07, PTBXL

CASE 23 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Complete atrioventricular block

Complete AV block: two independent clocks



Block may be nodal or infranodal; a subsidiary focus sustains ventricular activation. Servier Medical Art, CC BY 4.0.

- Atrial impulses cannot reach the ventricles.
- A subsidiary pacemaker generates an escape rhythm below the block.
- Atrial and ventricular timing become independent.



PTB-XL 00959 | 0-10 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
 PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Assess perfusion and initiate the emergency bradycardia pathway if unstable.
- Arrange urgent specialist care and pacing capability while evaluating reversible causes.
- Document atrial rate, ventricular rate, QRS width and AV relationship.

Avoid. AV dissociation alone is not synonymous with complete AV block.

CASE 23 | ANOTHER REAL ECG

Compare the patterns

Complete atrioventricular block



PTB-XL 00959 | 0-10 s | 25 mm/s, 10 mm/mV proportions

Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Another complete-block tracing has a broader escape pattern.



PTB-XL 08620 | 0-8 s | 25 mm/s, 10 mm/mV proportions

Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- Ventricular activation is markedly broad.
- Escape morphology and rate affect clinical urgency.

Remember. Two clocks; no reliable electrical bridge.

CASE 24 | CONDUCTION AND STRUCTURE

Junctional escape: a backup beat arrives late

CLINICAL PRESENTATION

A monitor records a pause followed by an isolated beat identified as junctional escape. The patient's symptom diary is available for comparison.

Is the extra-looking beat early, or is it rescuing a pause?



MIT-BIH 232 | source 1250 s | 0-12 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise

1

The escape event follows a longer interval, not premature timing.

2

Inspect for a preceding sinus P wave and retrograde atrial activity.

3

Compare both channels and surrounding conducted complexes.

Remember: Premature beats interrupt; escape beats rescue.

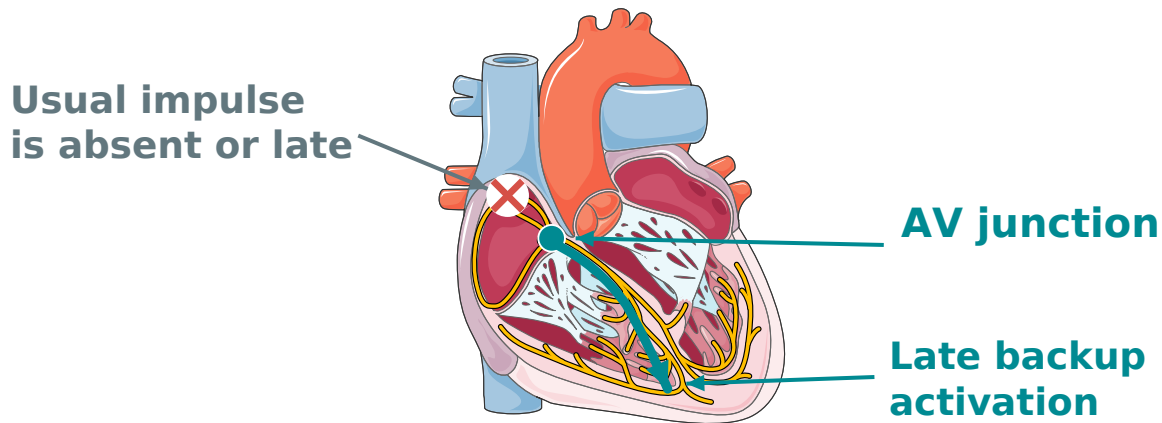
References: C08, X13A, V4-MIT-DIR

CASE 24 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

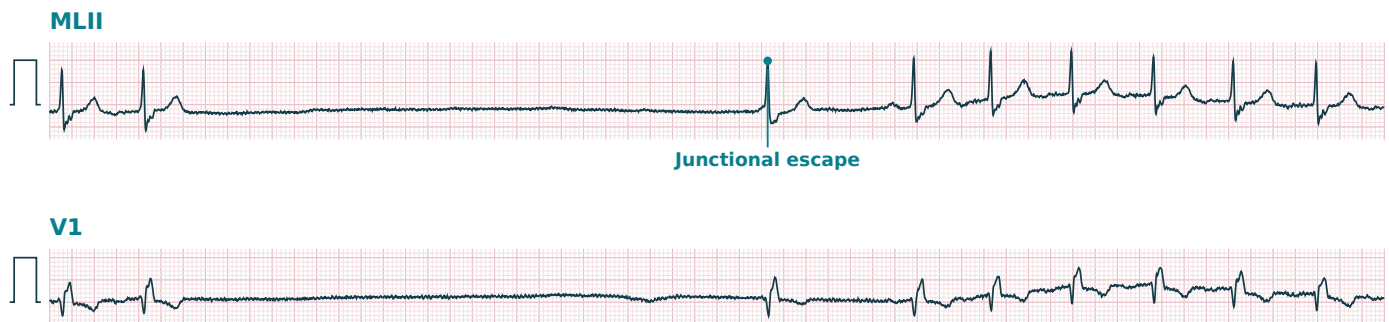
Junctional escape beat

Junctional escape: the backup pacemaker



A slower AV-junction focus generates a backup impulse. Anatomy: Servier Medical Art, CC BY 4.0.

- The usual faster pacemaker fails to activate the ventricles in time.
- A subsidiary focus near the AV junction reaches its own threshold.
- The backup impulse enters the ventricular conduction system.



MIT-BIH 232 | source 1250 s | 0-12 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

- Assess symptoms and circulation during the event.
- Investigate why the dominant rhythm paused or failed to conduct.
- Review medicines and reversible causes; do not treat the escape beat as the primary problem.

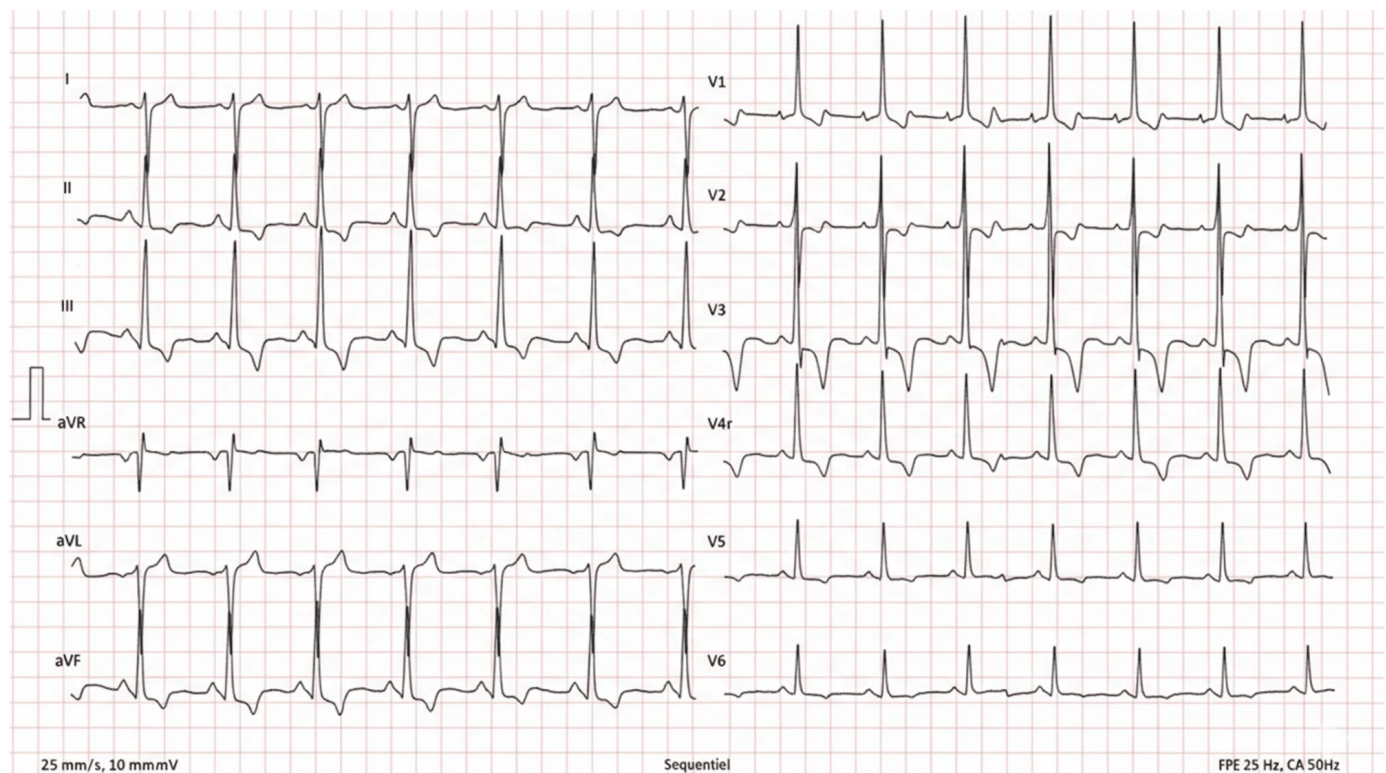
Avoid. Absent visible P waves alone do not establish a junctional origin.

CASE 25 | CONDUCTION AND STRUCTURE

Right ventricular hypertrophy: pressure leaves a trace

PUBLISHED CASE

A 21-year-old man has six months of exertional breathlessness and chest discomfort. A systolic ejection murmur is heard at the left upper sternal border.

How does an obstructed pulmonary valve reshape the ECG?

Moujahid et al. · Figure 1 · CC BY 4.0

Three findings to recognise**1**

Lead I is predominantly negative; inferior leads are positive.

2

V1 has a dominant tall R wave.

3

Deep S waves are visible in I and aVL.

Remember: The ECG suggests the load; imaging identifies its source.

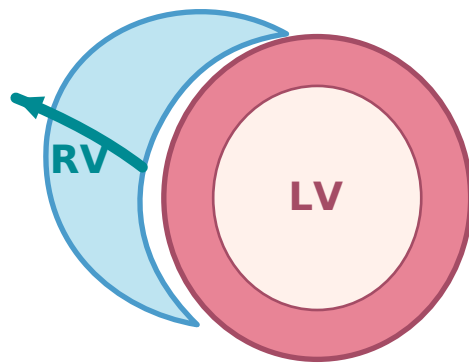
CASE 25 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Right ventricular pressure loading from severe pulmonary valve stenosis

Pulmonary valve obstruction loads the right ventricle

Transverse ventricular view



Patient's right ←

Tight pulmonary valve



Higher RV pressure



Stronger rightward forces

Pulmonary valve obstruction loads the right ventricle and can increase rightward electrical forces. Original anatomical schematic.

- A narrowed pulmonary valve obstructs right ventricular outflow.
- The right ventricle works against higher pressure and hypertrophies.
- Stronger rightward electrical forces change the axis and right-precordial QRS.



Moujahid et al. · Figure 1 · CC BY 4.0

In practice

- Examine for right-heart disease and an outflow murmur.
- Use echocardiography to assess the pulmonary valve, gradient and right ventricle.
- Refer severe congenital valve obstruction for specialist assessment and intervention planning.

Avoid. Right-axis deviation alone does not prove hypertrophy; this patient's diagnosis was confirmed by echocardiography.

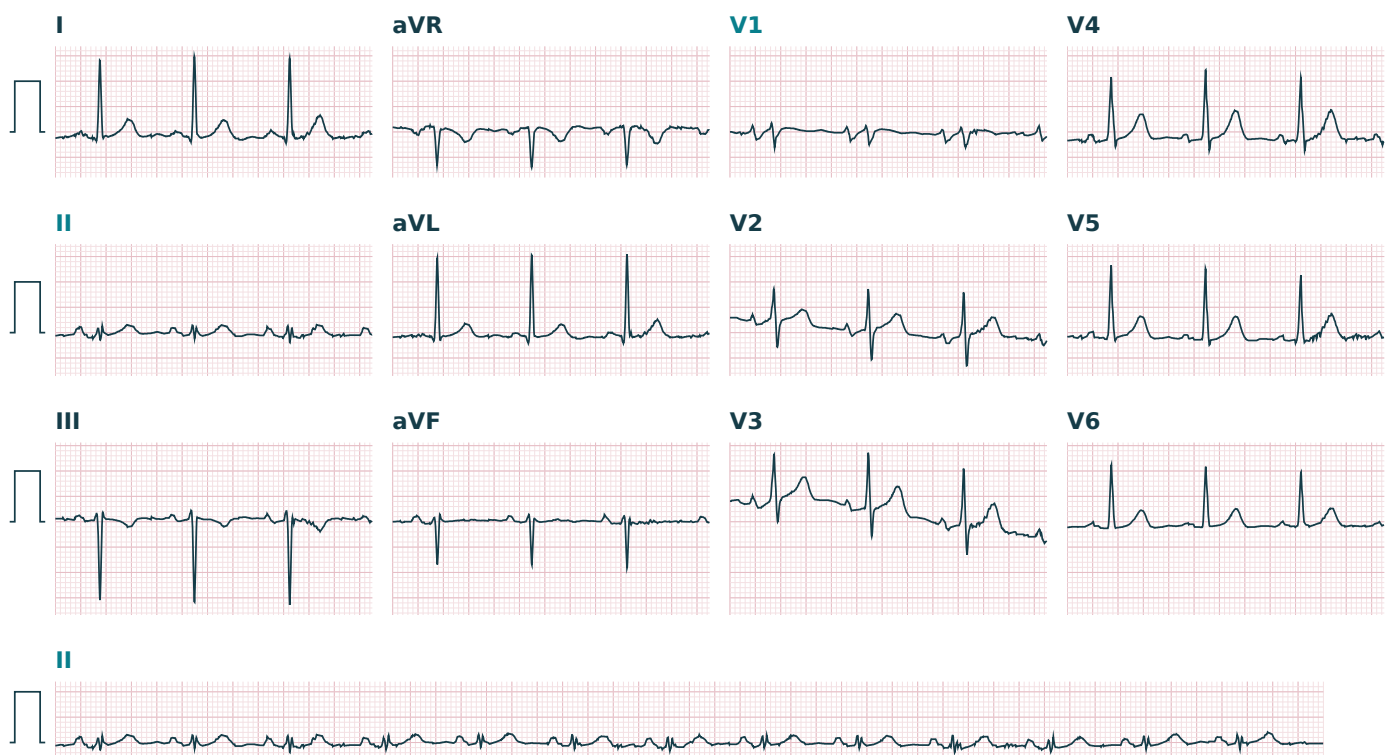
CASE 26 | CONDUCTION AND STRUCTURE

Atrial abnormality: read the P wave in two views

CLINICAL PRESENTATION

A 66-year-old with hypertension and exertional breathlessness has a broad-looking P wave. In V1, the terminal atrial deflection is conspicuously negative.

Which atrium contributes to the late part of atrial activation?



PTB-XL 06685 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

V1 has a conspicuous terminal negative P-wave component.

2

Lead II provides a simultaneous view of atrial duration and contour.

3

Interpret the entire P wave rather than one isolated notch.

Remember: The P wave is a journey through both atria.

References: C02, S01, B17-ATRIAL, PTBXL

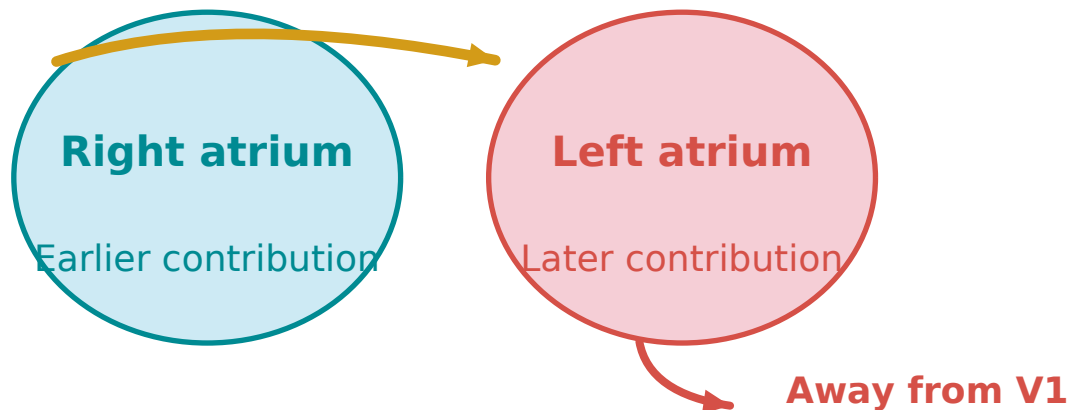
CASE 26 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Left atrial electrical abnormality

Atrial activation shapes the P wave

Activation spreads across both atria



Late left atrial activation contributes to terminal P-wave negativity in V1. Original anatomical schematic.

- Right atrial activation usually contributes to the early P wave.
- Left atrial activation contributes later and points partly away from V1.
- Changes in atrial activation can widen or deepen the terminal component.



PTB-XL 06685 | 0-3 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Assess hypertension, valve disease and symptoms.
- Use echocardiography when structural atrial disease is suspected.
- Report the electrical finding without treating it as a direct measurement of atrial size.

Avoid. ECG atrial criteria have imperfect agreement with measured atrial enlargement.

CASE 26 | ANOTHER REAL ECG

Compare the patterns

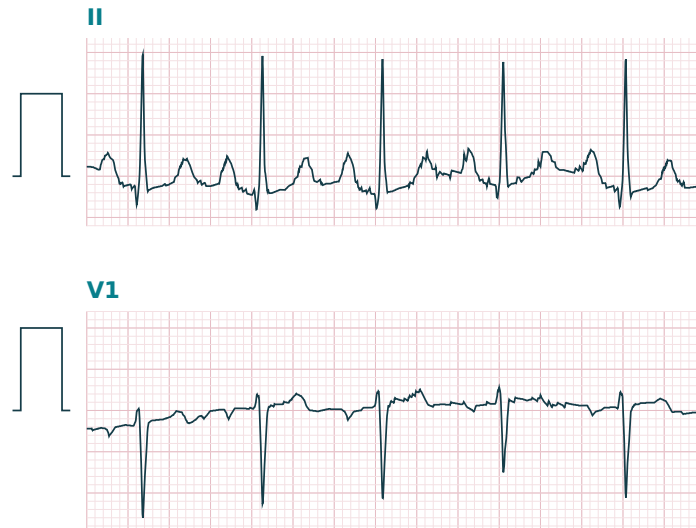
Left atrial electrical abnormality



PTB-XL 06685 | 0-3 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

A right atrial pattern emphasises the earlier positive component.



PTB-XL 05955 | 0-3 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- Inferior P waves are prominent and upright.
- Confirm the anatomical cause with clinical assessment.

Remember. The P wave is a journey through both atria.

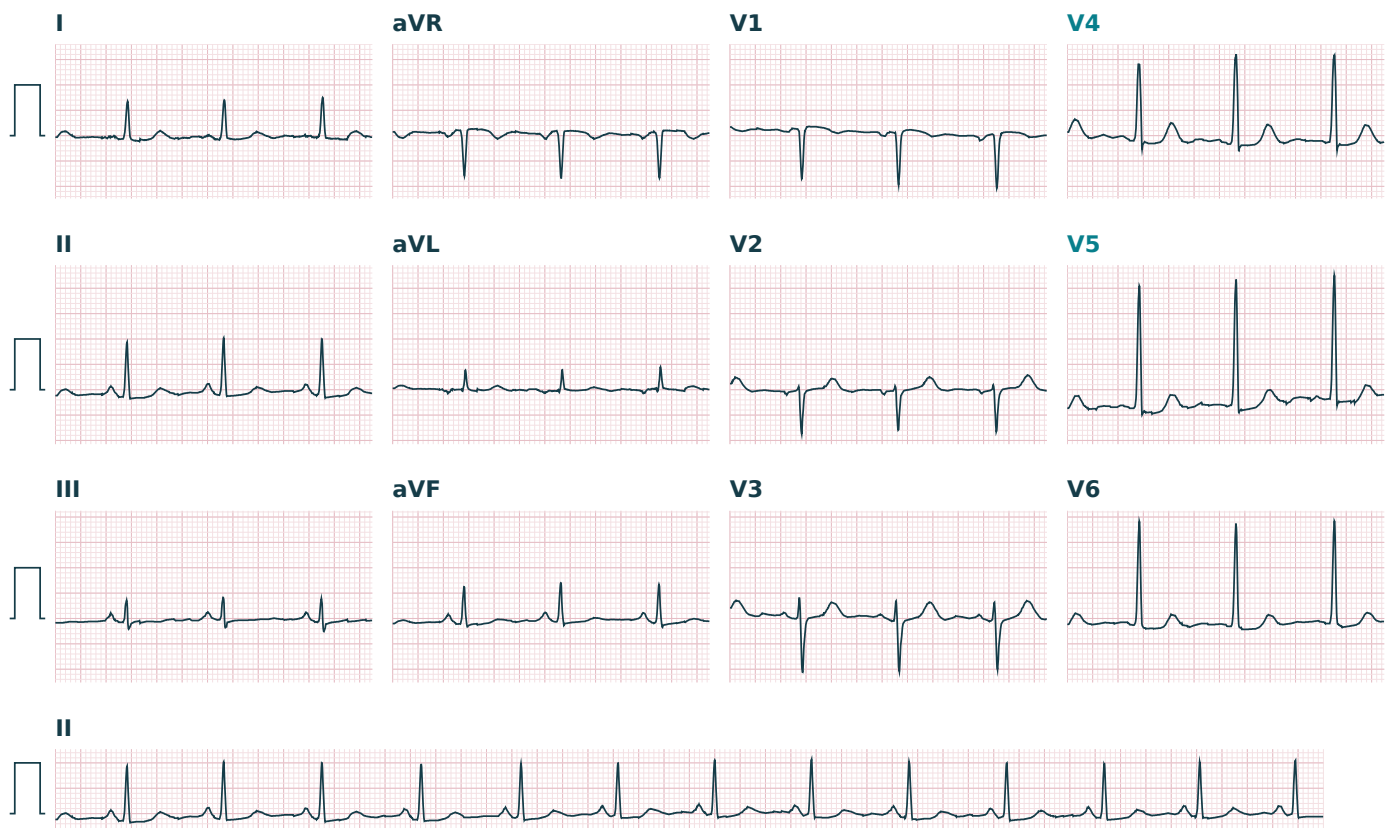
CASE 27 | ISCHAEMIA AND ACUTE ILLNESS

ST depression: supply fails to meet demand

CLINICAL PRESENTATION

A 64-year-old develops chest pressure on walking. Symptoms persist at rest. The ECG has ST depression in adjacent chest leads.

What makes these ST changes clinically important?



PTB-XL 00341 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

The ST segment lies below its baseline.

2

Adjacent leads show a related change.

3

The QRS is narrow; assess ST changes in context.

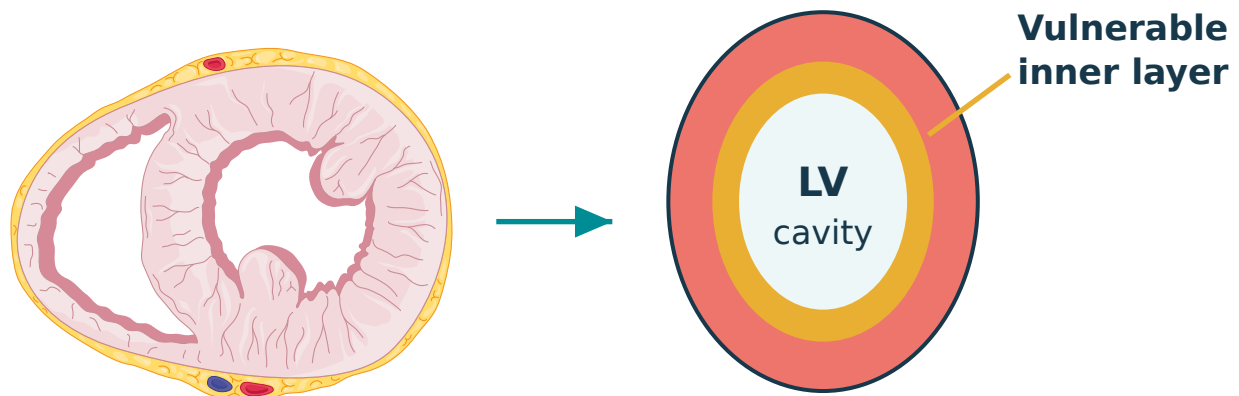
Remember: The ST segment is a clinical clue, not a coronary map.

CASE 27 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

ST depression concerning for myocardial ischaemia

Ventricular cross-section



Low supply or high demand → uneven voltages

Subendocardial ischaemia changes voltage relationships across the ventricular wall.

- Reduced coronary supply or increased demand stresses ventricular muscle.
- Uneven membrane voltages create an injury-current gradient.
- The lead records the resulting ST shift.



PTB-XL 00341 | 0.5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

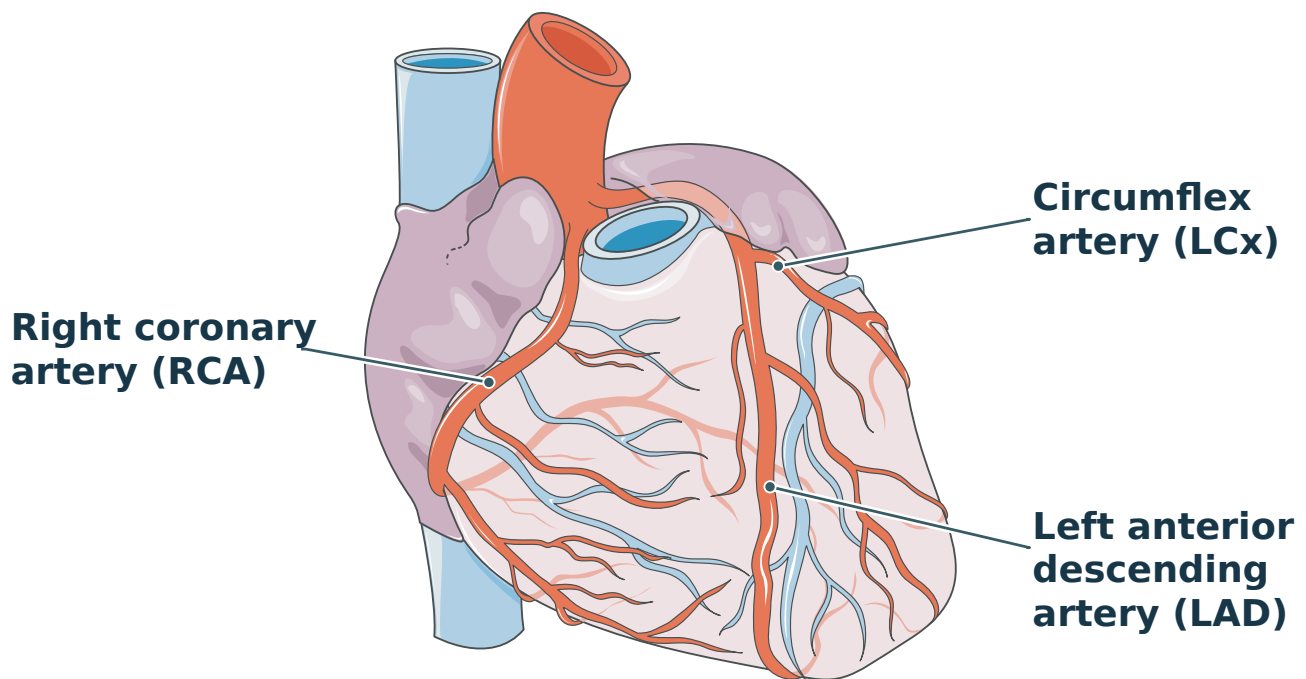
- Assess symptoms, vital signs and haemodynamic stability immediately.
- Compare previous ECGs; repeat ECGs and obtain serial high-sensitivity troponin.
- Use the acute coronary syndrome pathway; ongoing symptoms require urgent review.

Avoid. ST depression alone cannot identify a culprit artery or distinguish all mechanisms.

ANATOMY AT A GLANCE

Epicardial coronary circulation

Three coronary arteries to orient the ECG



Orange-red vessels are arteries; blue-gray vessels are veins.
Coronary dominance and supplied territories vary.

Connect it to the ECG. The ST segment is a clinical clue, not a coronary map.

- Reduced coronary supply or increased demand stresses ventricular muscle.
- Uneven membrane voltages create an injury-current gradient.
- The lead records the resulting ST shift.

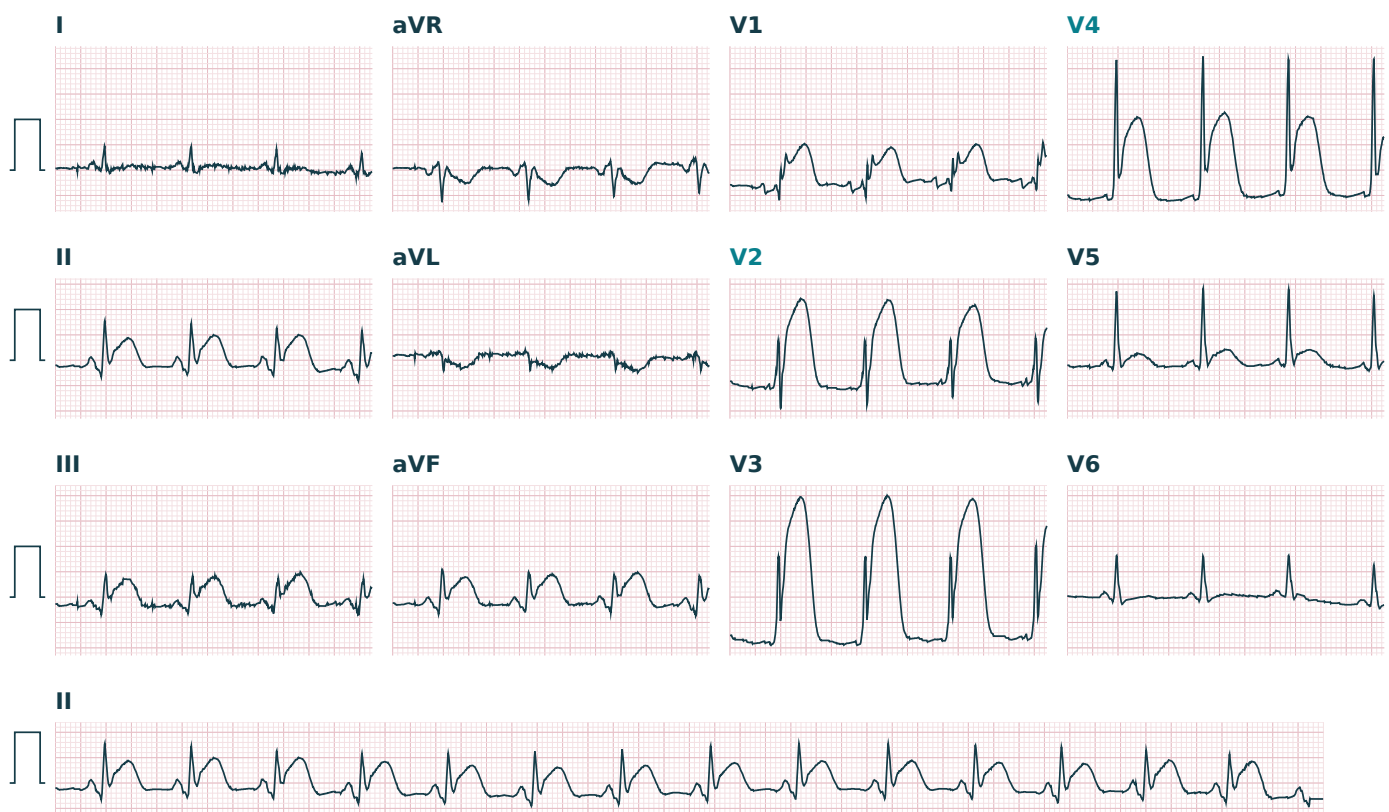
Adapted from Servier Medical Art, CC BY 4.0. Teaching labels added.

CASE 28 | ISCHAEMIA AND ACUTE ILLNESS

Anterior injury: recognise the urgent pattern

CLINICAL PRESENTATION

A 58-year-old has persistent central chest pressure, sweating and nausea. Contiguous anterior leads show substantial ST elevation.

Which myocardial territory is threatened?

PTB-XL 01063 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise**1**

ST elevation involves neighbouring anterior leads.

2

The ST-T contour is markedly abnormal.

3

Inspect the complete ECG for reciprocal and additional changes.

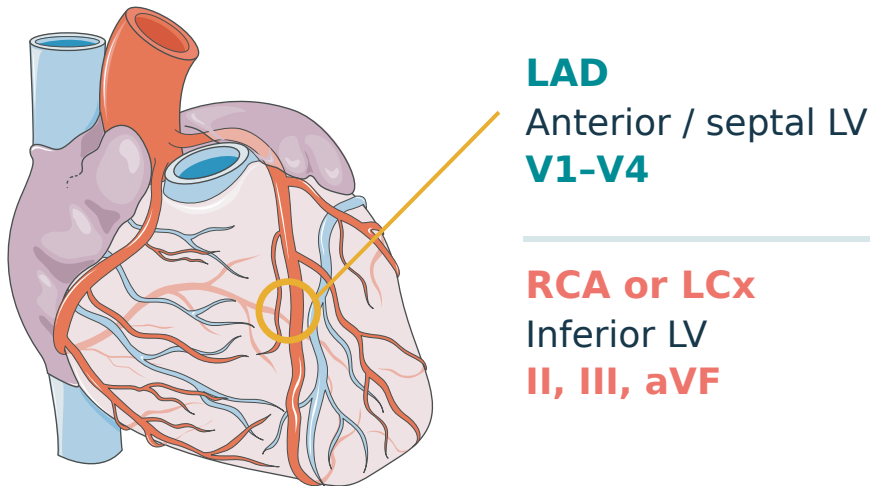
Remember: Adjacent leads, a matching story, urgent action.

References: C06, S02, E09-VI

CASE 28 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Anterior ST-elevation pattern requiring emergency coronary assessment



Lead territories guide attention; coronary dominance varies.

Coronary supply and lead territories explain related changes; angiography determines the culprit artery.

- Anterior leads face the anterior left ventricle.
- Acute ischaemic injury creates a voltage gradient.
- LAD disease often threatens this territory; angiography defines anatomy.



PTB-XL 01063 | 0.5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Activate the local reperfusion pathway when the clinical diagnosis is STEMI.
- Monitor continuously and prepare for haemodynamic or electrical deterioration.
- Do not delay reperfusion for troponin when the diagnosis is established.

Avoid. ST-elevation thresholds support the diagnosis; they do not detect every acute coronary occlusion.

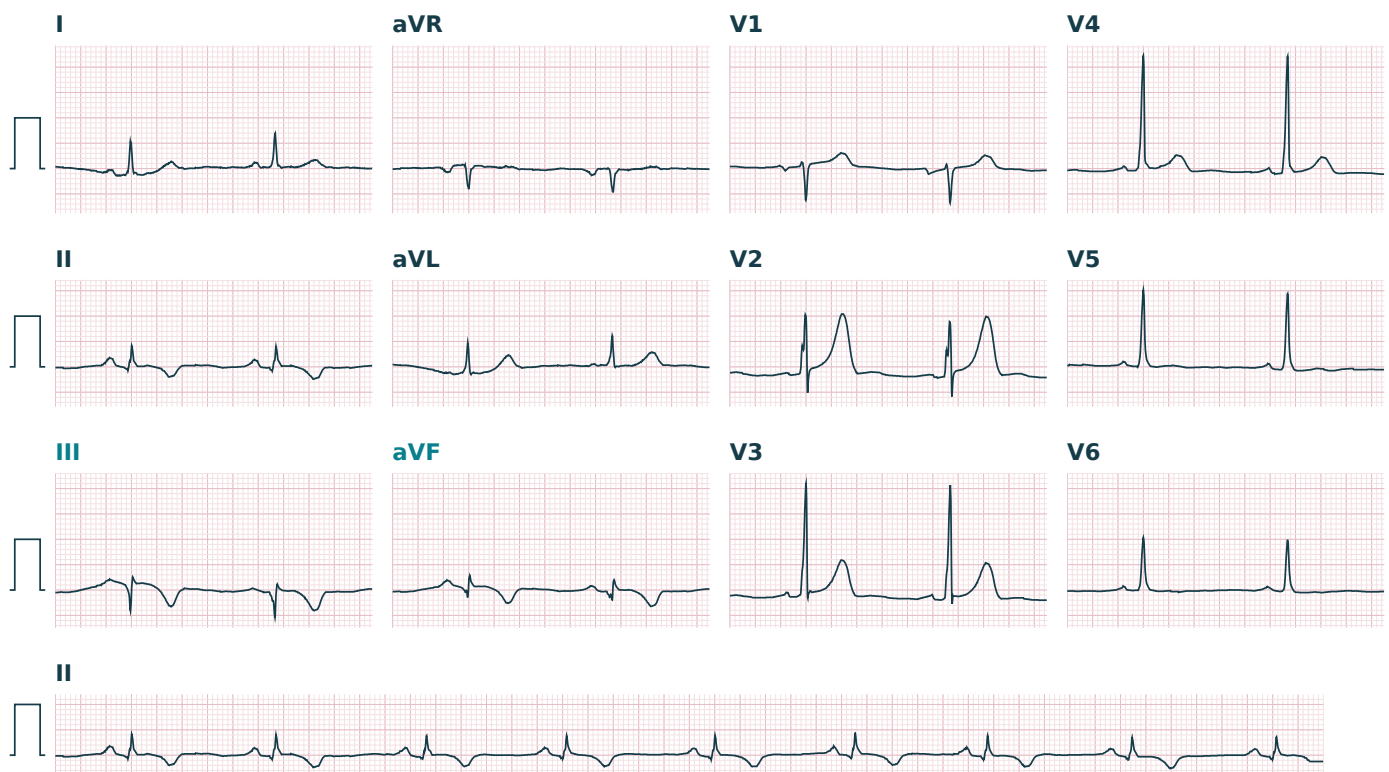
CASE 29 | ISCHAEMIA AND ACUTE ILLNESS

Look beyond the standard twelve leads

CLINICAL PRESENTATION

A patient with inferior ECG abnormalities develops chest pain and hypotension. You need to examine territories poorly covered by the standard tracing.

Which extra leads answer the next clinical question?



PTB-XL 12941 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise**1**

Inferior Q-wave and T-wave abnormalities are visible.

2

These findings alone do not establish acute infarction.

3

The standard ECG does not directly show posterior or right-sided leads.

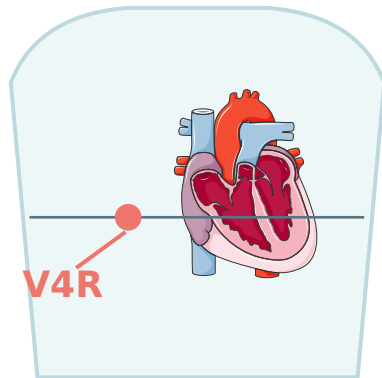
Remember: When the viewpoint is incomplete, move the camera.

CASE 29 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

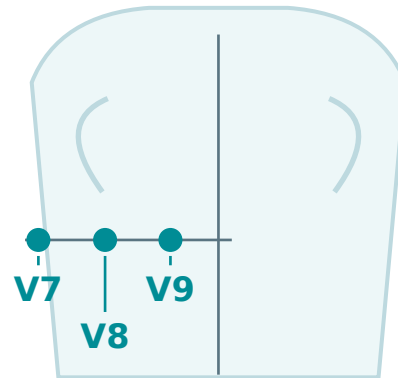
Inferior abnormalities: consider posterior and right-sided recordings

Front: patient facing you



5th intercostal space
Right midclavicular line

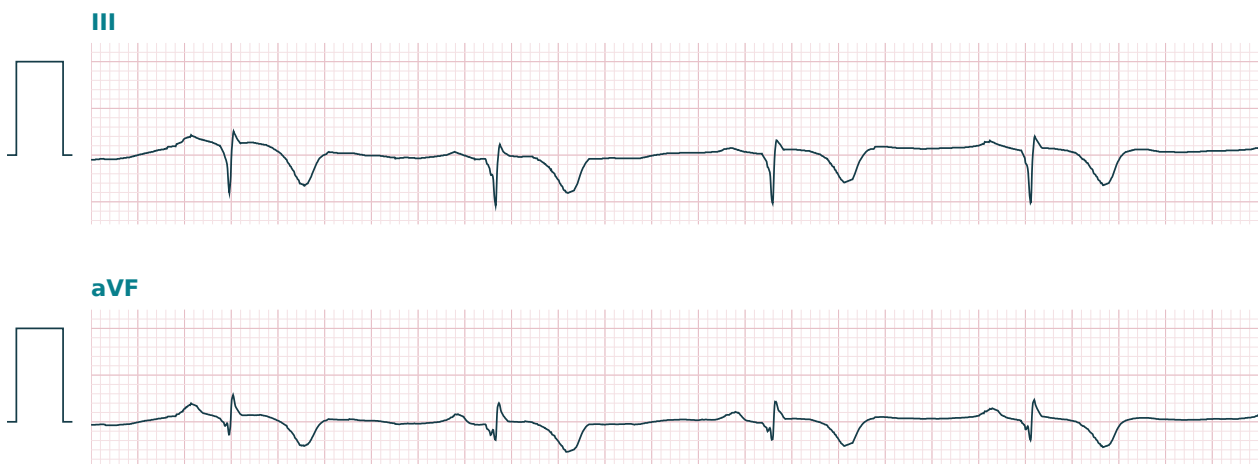
Back: patient facing away



Posterior axillary → scapular → paraspinal
All at the horizontal level of V6

V4R faces the right ventricle; V7-V9 face the posterior left ventricular wall.

- Posterior leads V7-V9 face the posterior left ventricle.
- Right-sided V3R-V4R examine the right ventricle.
- Moving the electrodes changes the viewpoint, not the underlying injury.



PTB-XL 12941 | 0.5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Record V7-V9 when posterior occlusion is suspected.
- Record right-sided leads with inferior STEMI or suspected right ventricular involvement.
- Repeat ECGs during symptoms and compare with earlier recordings.

Avoid. This tracing contains inferior abnormalities; it does not itself prove acute posterior or right ventricular infarction.

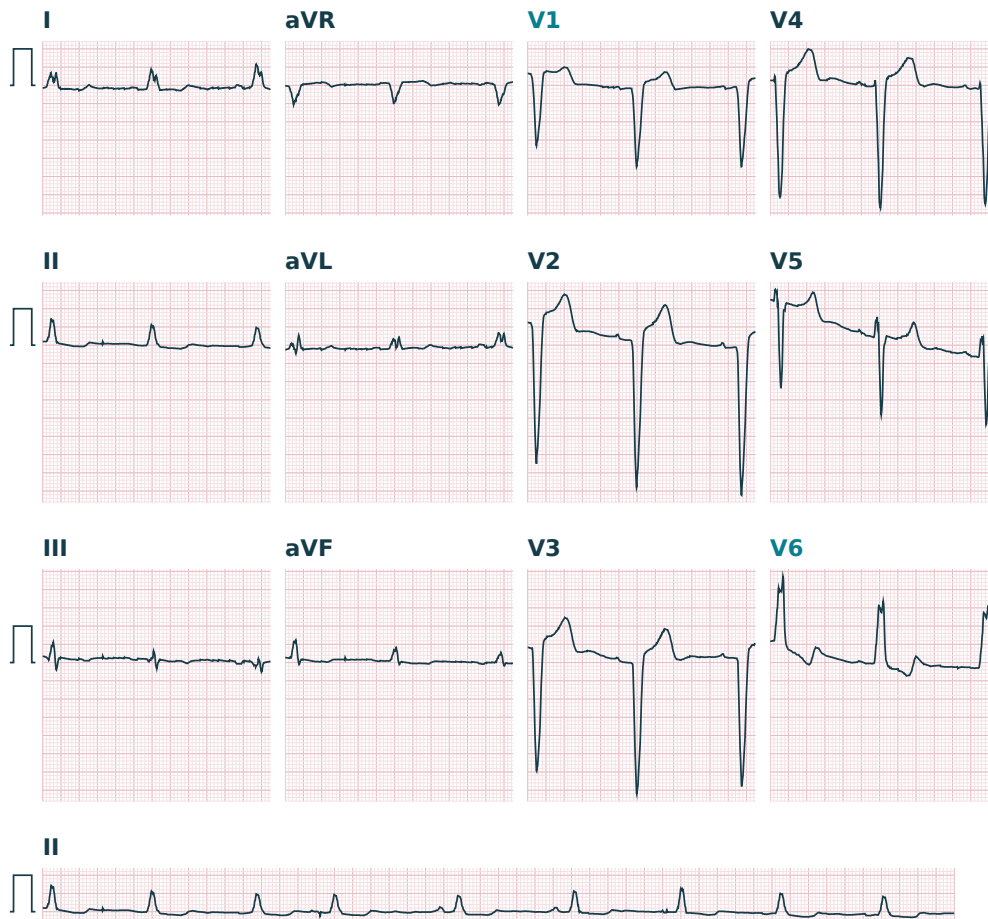
CASE 30 | ISCHAEMIA AND ACUTE ILLNESS

LBBB: judge ST shifts against the QRS

CLINICAL PRESENTATION

A 72-year-old with known left bundle branch block develops chest pressure. Broad QRS complexes make ordinary ST-elevation rules unreliable.

Is the ST shift expected or disproportionate?



PTB-XL 01564 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Broad QRS complexes reflect abnormal ventricular activation.

2

ST-T direction is often opposite the dominant QRS.

3

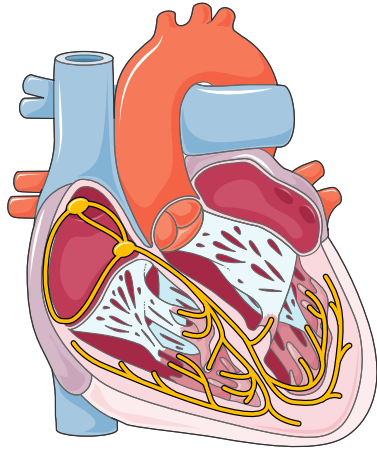
Look for concordance or excessive discordance in every lead.

Remember: In LBBB, judge the ST segment beside its QRS.

CASE 30 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Left bundle branch block: assess for superimposed ischaemia



Activation sequence changes
Broad QRS



Recovery sequence changes
ST-T usually opposes QRS

Left ventricular delay

LBBB changes both activation and recovery; assess ST deviation relative to its QRS complex.

- Delayed left ventricular activation changes the QRS direction.
- Recovery changes secondarily, producing expected discordant ST-T segments.
- Ischaemia may create shifts beyond that expected relationship.



PTB-XL 01564 | 0.5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Assess symptoms, instability and previous ECGs promptly.
- Apply modified Sgarbossa criteria; see the compact reference page.
- Escalate persistent suspected occlusion even when criteria are absent.

Avoid. New LBBB alone is not diagnostic of acute coronary occlusion.

CASE 30 | ANOTHER REAL ECG

Compare the patterns

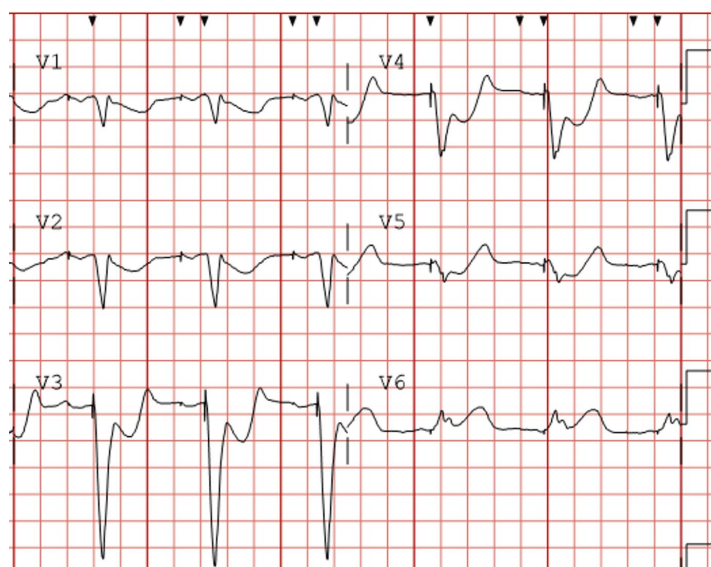
Left bundle branch block: assess for superimposed ischaemia



PTB-XL 01564 | 0.5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Chest pain during ventricular pacing: infarction confirmed at angiography.



Precordial leads V1-V6. O'Rorke et al. · Figure 1 · CC BY 4.0

- V6 has concordant ST elevation.
- V1-V3 show concordant ST depression.

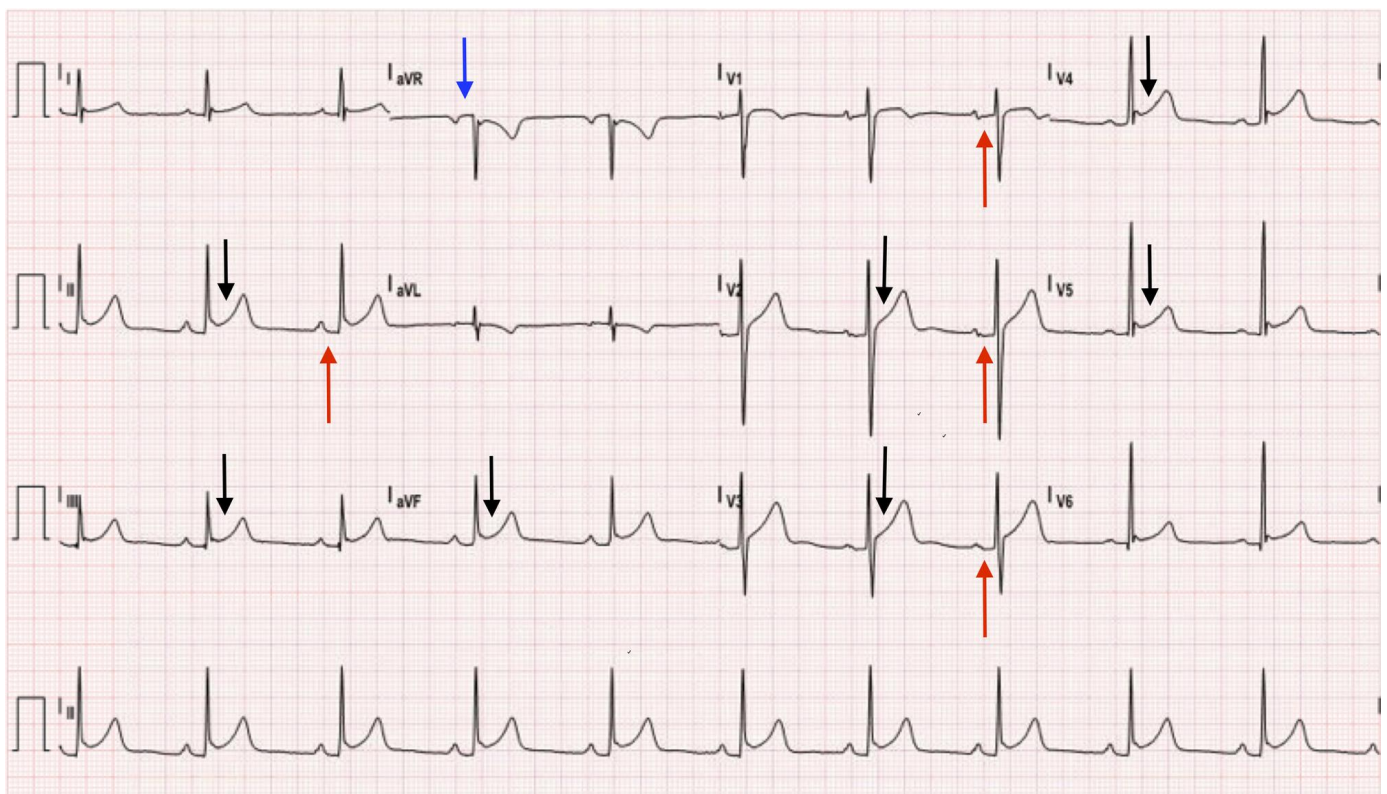
Remember. In LBBB, judge the ST segment beside its QRS.

CASE 31 | ISCHAEMIA AND ACUTE ILLNESS

Pericarditis: inflammation around the heart

PUBLISHED CASE

A 22-year-old man has pleuritic chest pain after a respiratory illness. Leaning forward helps. A pericardial rub is heard.

Does the ECG fit the clinical inflammatory picture?

Jason Mefford and Shannon L Toohey et al. | 2016 | CC BY 4.0

Three findings to recognise**1**

ST elevation is widespread rather than one coronary territory.

2

PR depression supports pericardial involvement.

3

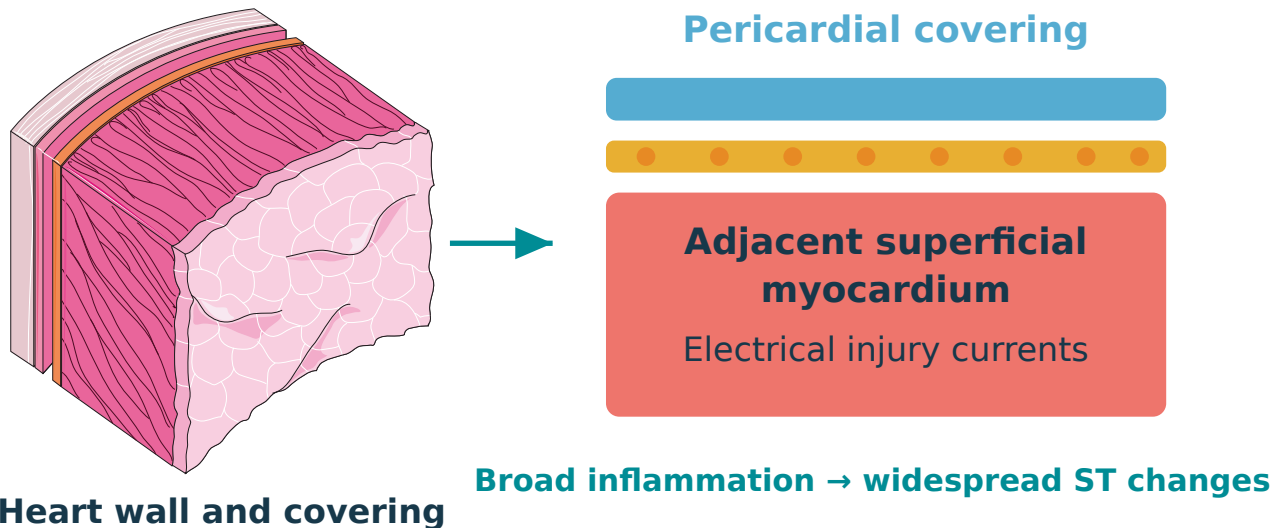
Interpret aVR and reciprocal changes with the complete pattern.

Remember: Widespread inflammation needs a whole-patient diagnosis.

CASE 31 | UNDERSTAND AND APPLY

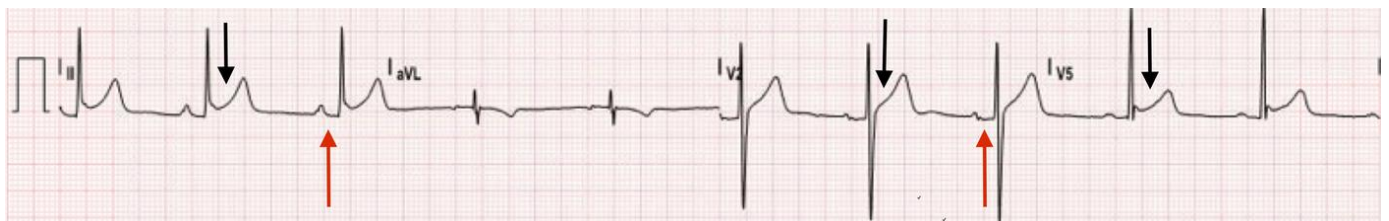
From anatomy to clinical meaning

Acute pericarditis with widespread ST elevation



Pericarditis-associated ST shifts arise from electrical effects in adjacent superficial myocardium.

- Inflammation involves the pericardium and adjacent superficial myocardium.
- Widespread injury currents produce diffuse ST changes.
- Atrial involvement can shift the PR segment.



Published middle-row detail: inferior and precordial ST elevation with PR depression; original arrows retained. Jason Mefford and Shannon L Toohey et al. | 2016 | CC BY 4.0

In practice

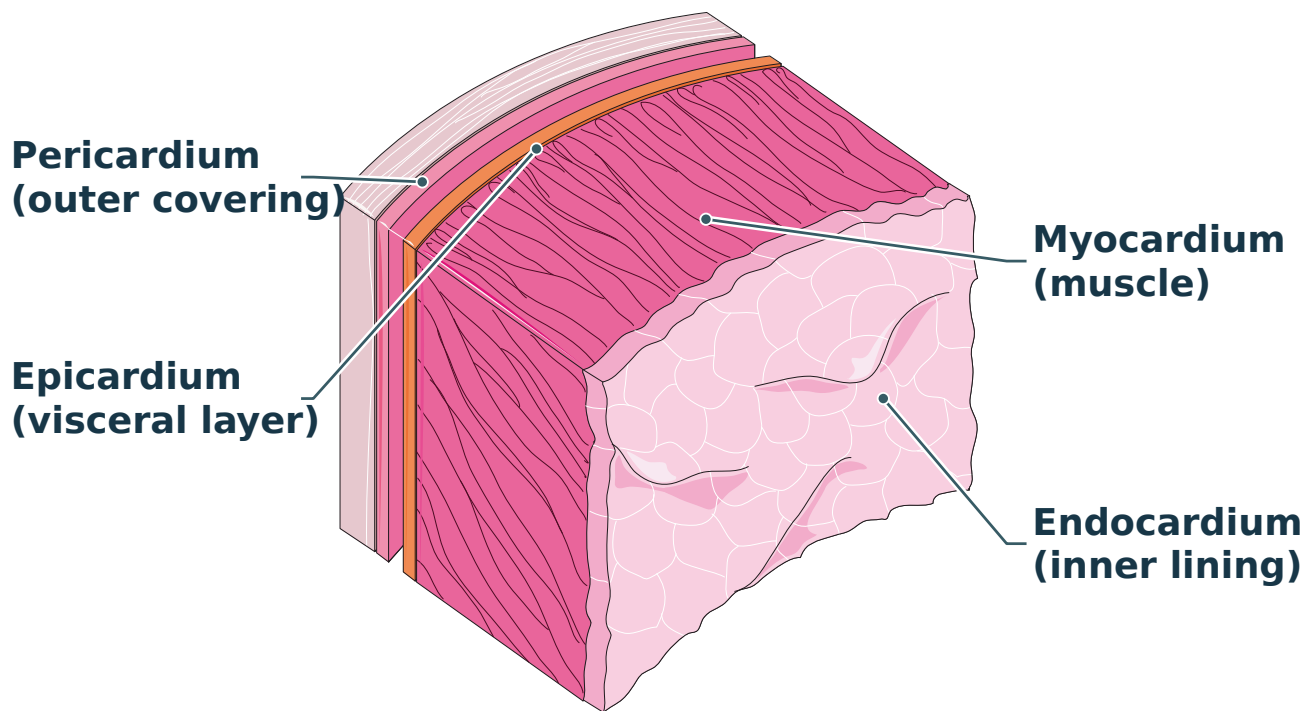
- Assess for myocardial involvement, effusion and haemodynamic compromise.
- Use symptoms, inflammatory markers, troponin and echocardiography together.
- Seek urgent assessment for high-risk features or diagnostic uncertainty.

Avoid. Concave ST elevation alone cannot distinguish pericarditis from acute coronary occlusion.

ANATOMY AT A GLANCE

Heart wall and pericardial layers

From pericardium to chamber lining



A schematic wall block; layers are not drawn to scale.
The pericardial cavity lies outside the epicardium.

Connect it to the ECG. Widespread inflammation needs a whole-patient diagnosis.

- Inflammation involves the pericardium and adjacent superficial myocardium.
- Widespread injury currents produce diffuse ST changes.
- Atrial involvement can shift the PR segment.

Adapted from Servier Medical Art, CC BY 4.0. Teaching labels added.

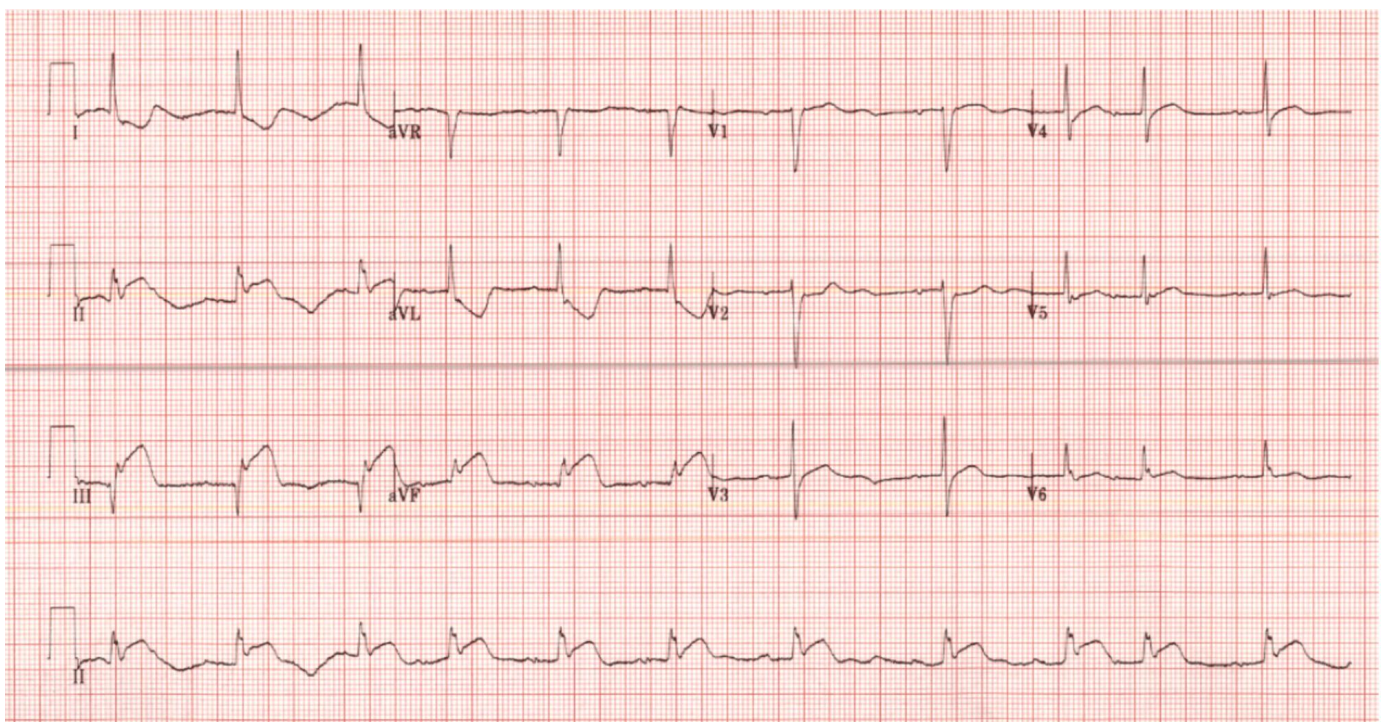
CASE 32 | ISCHAEMIA AND ACUTE ILLNESS

Inferior infarction can present without chest pain

PUBLISHED CASE

A 70-year-old woman develops severe nausea, vomiting and left arm weakness after several days of ear pain. Her ECG changes redirect the assessment.

Which changes identify an urgent inferior injury pattern?



Bactawar et al. · Figure 2 · CC BY 4.0

Three findings to recognise

1

ST elevation involves II, III and aVF.

2

Reciprocal ST depression appears in I and aVL.

3

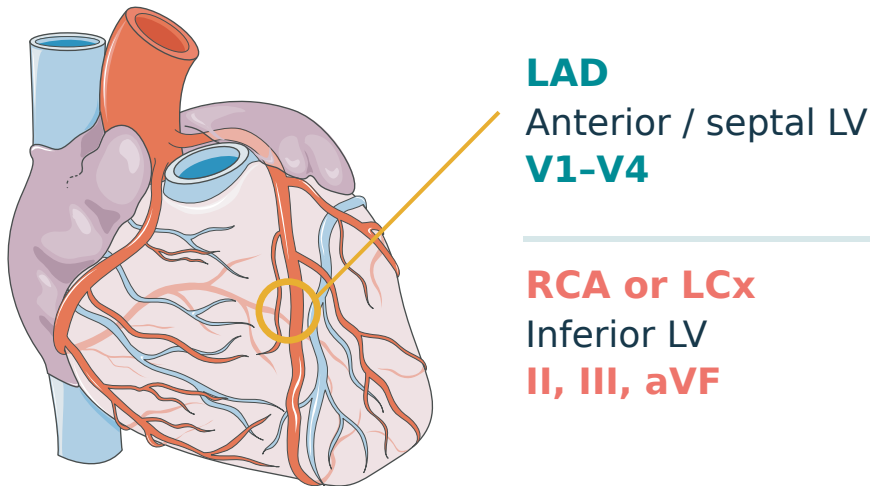
The clinical presentation is atypical, but the ECG is high risk.

Remember: The inferior wall may announce itself with nausea.

CASE 32 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

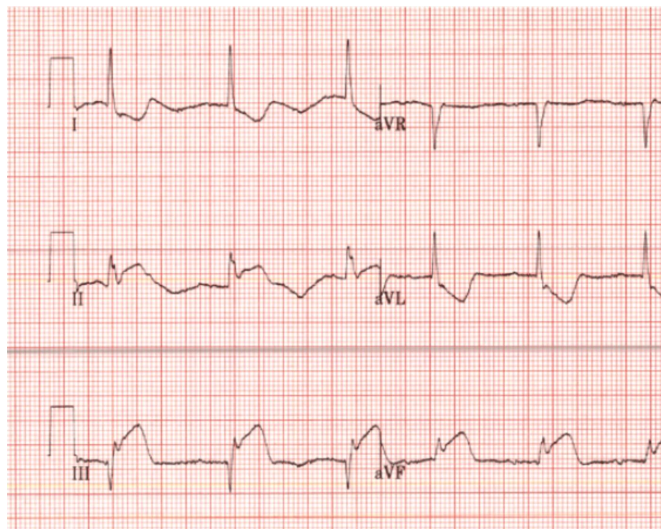
Acute inferior ST-elevation myocardial infarction



Lead territories guide attention; coronary dominance varies.

Coronary supply and lead territories explain related changes; angiography determines the culprit artery.

- Inferior leads face the diaphragmatic left ventricular surface.
- Acute inferior injury directs the ST vector towards these leads.
- Angiography confirmed right coronary artery occlusion in this patient.



Bactawar et al. · Figure 2 · CC BY 4.0

In practice

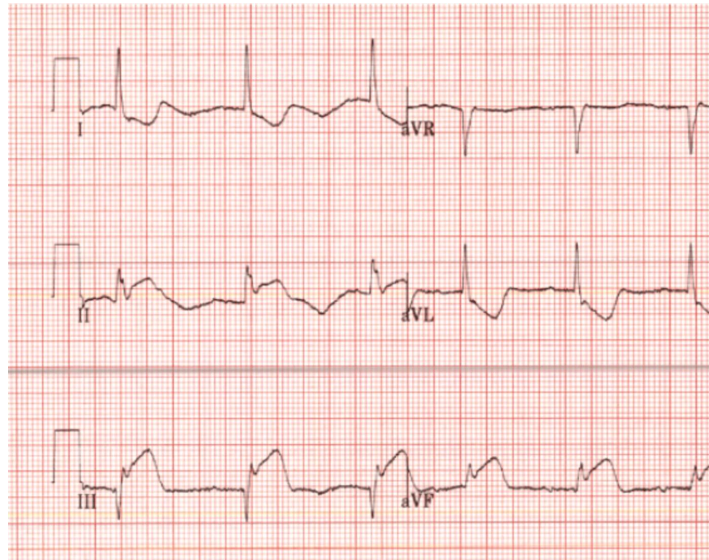
- Activate the emergency coronary reperfusion pathway.
- Record right-sided leads and assess for right ventricular involvement.
- Monitor rhythm, blood pressure and signs of shock continuously.

Avoid. An inferior pattern does not always mean right coronary occlusion; coronary anatomy varies.

CASE 32 | ANOTHER REAL ECG

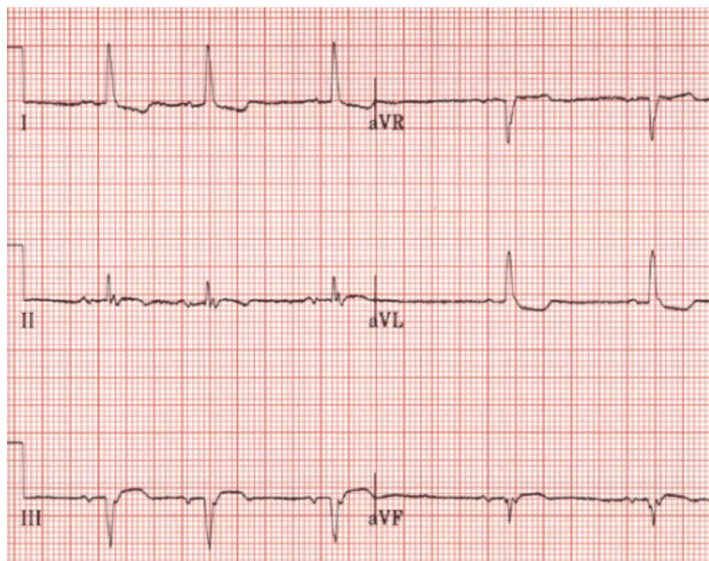
Compare the patterns

Acute inferior ST-elevation myocardial infarction



Bactawar et al. · Figure 2 · CC BY 4.0

The same patient after right coronary reperfusion



Bactawar et al. · Figure 6 · CC BY 4.0

- Inferior ST elevation resolves.
- Reciprocal depression improves.

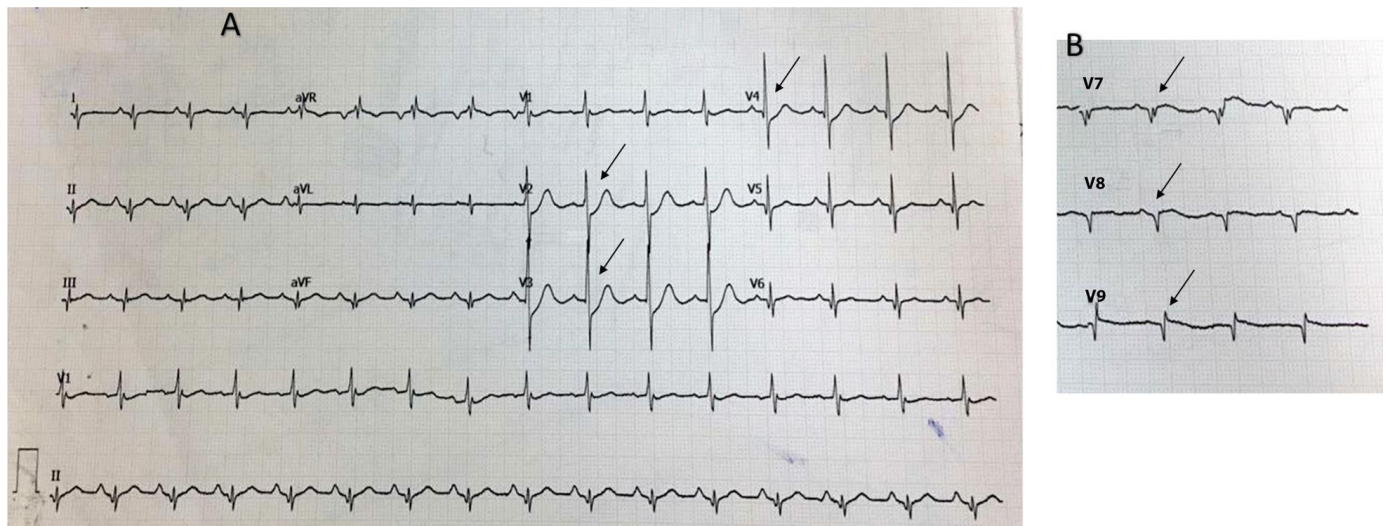
Remember. The inferior wall may announce itself with nausea.

CASE 33 | ISCHAEMIA AND ACUTE ILLNESS

Posterior infarction: reveal the hidden elevation

PUBLISHED CASE

A 57-year-old man has persistent chest pain and dyspnoea. ST depression in anterior leads prompts a recording from the posterior chest.

What do V7-V9 add to the diagnosis?

Alsagaff et al. (2022), Figure 1; CC BY 4.0.

Three findings to recognise**1**

Anterior leads show ST depression.

2

Posterior leads V7-V9 reveal ST elevation.

3

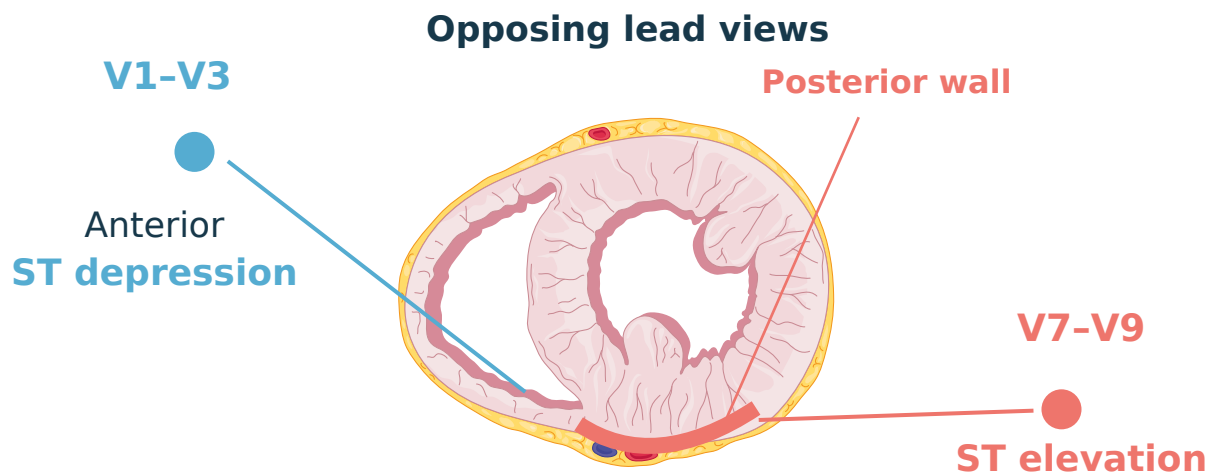
The two views describe the same posterior injury.

Remember: Front: depression. Back: elevation. One injured wall.

CASE 33 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

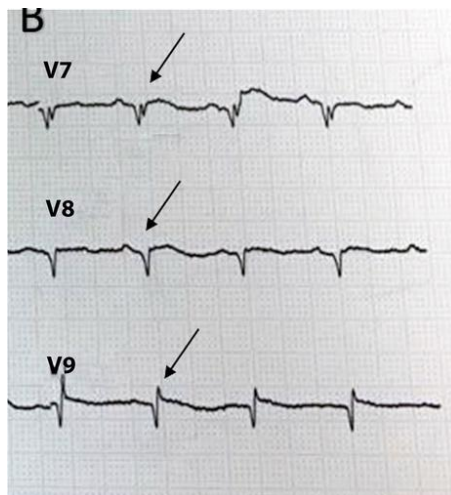
Posterior ST-elevation myocardial infarction



Posterior injury: opposing views of one electrical gradient

Posterior injury may depress anterior ST segments while elevating directly facing posterior leads.

- The injured posterior wall lies behind the left ventricle.
- Anterior leads see its injury vector moving away.
- Posterior leads face the injury and record elevation.



Actual V7-V9 show posterior ST elevation; original arrows retained. Alsagaff et al. (2022), Figure 1; CC BY 4.0.

In practice

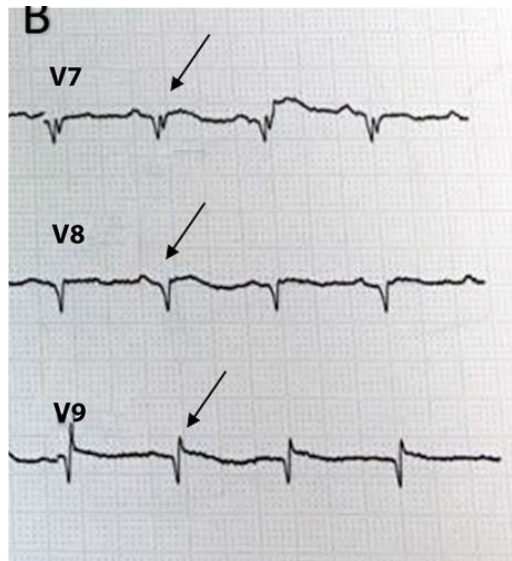
- Record posterior leads promptly when this pattern raises suspicion.
- Use the emergency coronary reperfusion pathway in the appropriate clinical setting.
- Compare serial ECGs; do not dismiss persistent symptoms after one tracing.

Avoid. Anterior ST depression has several causes; posterior leads and the clinical context resolve the question.

CASE 33 | ANOTHER REAL ECG

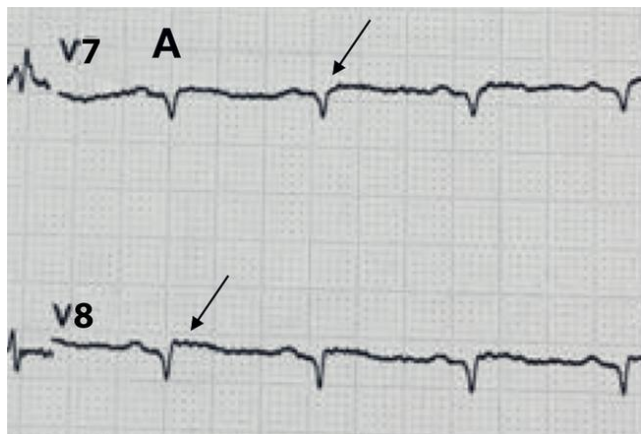
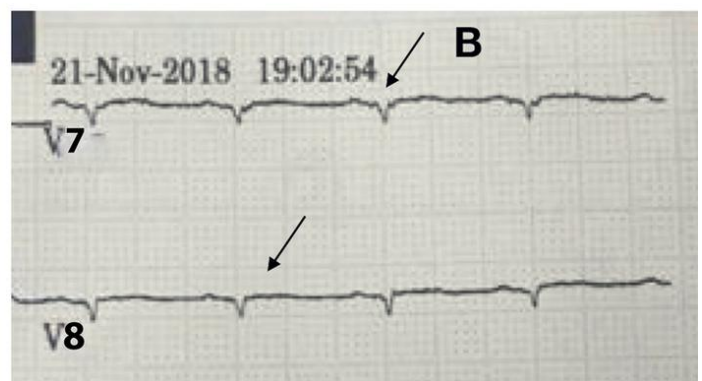
Compare the patterns

Posterior ST-elevation myocardial infarction



Actual V7-V9 show posterior ST elevation; original arrows retained. Alsagaff et al. (2022), Figure 1; CC BY 4.0.

Posterior leads before and after reperfusion



Source panels A and B: posterior ST elevation before PCI and resolution after PCI. Alsagaff et al. (2022), Figure 5; CC BY 4.0.

- Posterior elevation before PCI.
- ST segments settle after reperfusion.

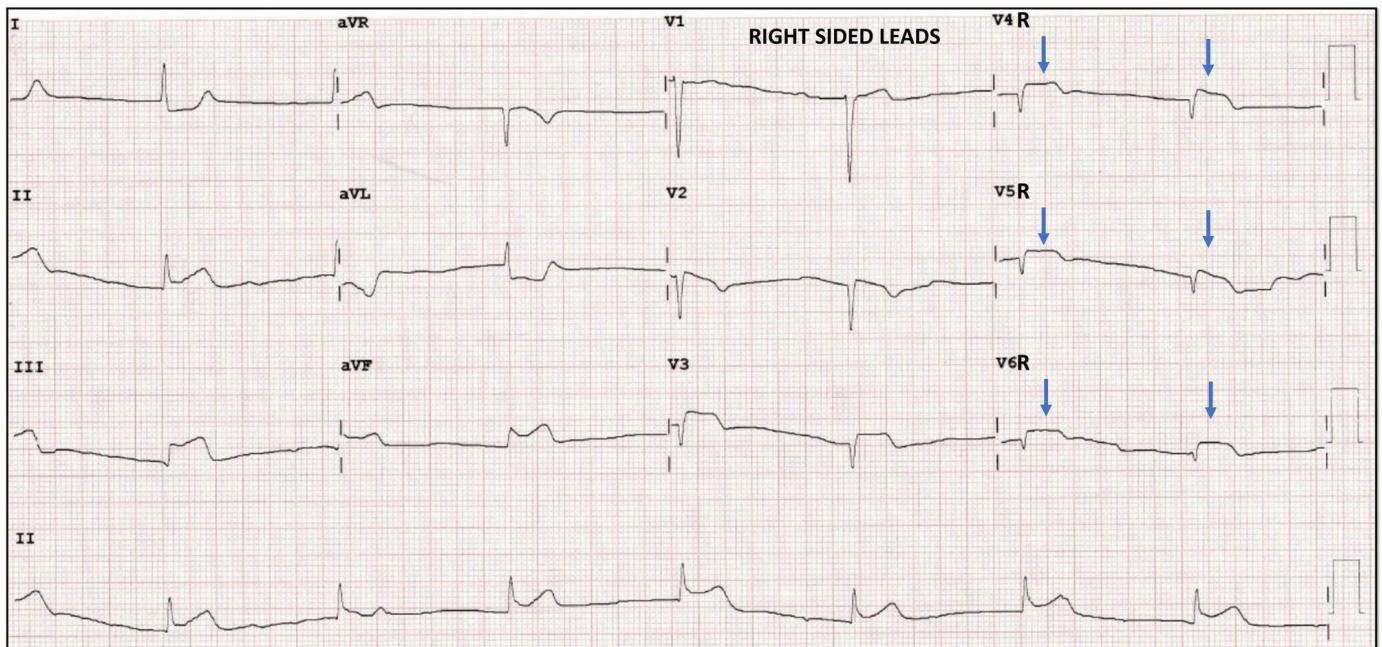
Remember. Front: depression. Back: elevation. One injured wall.

CASE 34 | ISCHAEMIA AND ACUTE ILLNESS

Right ventricular infarction: check the right side

PUBLISHED CASE

A 65-year-old man has chest pain, sweating, bradycardia and raised jugular venous pressure. His lungs are clear; hypotension develops during transfer.

What does the right-sided recording reveal?

Kidambi et al. (2023), Figure 7; CC BY 4.0.

Three findings to recognise**1**

Right-sided V4R-V6R show ST elevation.

2

The ventricular rhythm is slow.

3

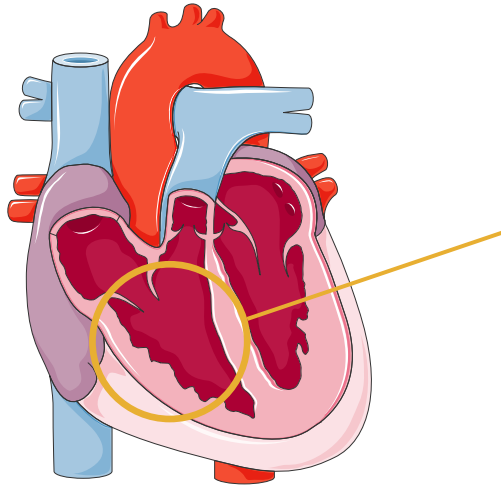
The companion standard ECG shows inferior injury.

Remember: Clear lungs plus shock: examine the right ventricle.

CASE 34 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Right ventricular infarction with inferior involvement



Right ventricular injury

Right ventricular injury reduces forward flow, left ventricular filling and potentially systemic pressure.

- Proximal right coronary occlusion can compromise right ventricular perfusion.
- The injured right ventricle pumps less blood through the lungs.
- Reduced left ventricular filling can worsen systemic hypotension.

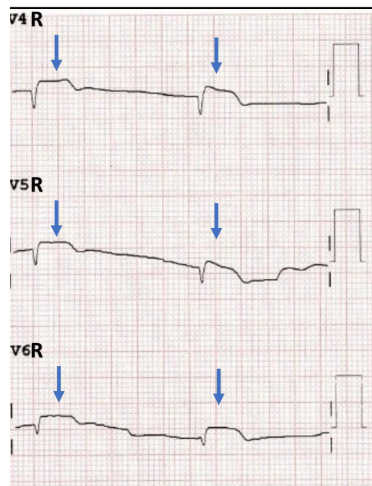
Right ventricular output falls



Left ventricular filling falls



Blood pressure may fall



Actual V4R-V6R show ST elevation; original arrows retained. Kidambi et al. (2023), Figure 7; CC BY 4.0.

In practice

- Activate urgent reperfusion and manage shock with expert support.
- Review preload, rhythm and right ventricular function at the bedside.
- Avoid nitrates in suspected right ventricular infarction or hypotension.

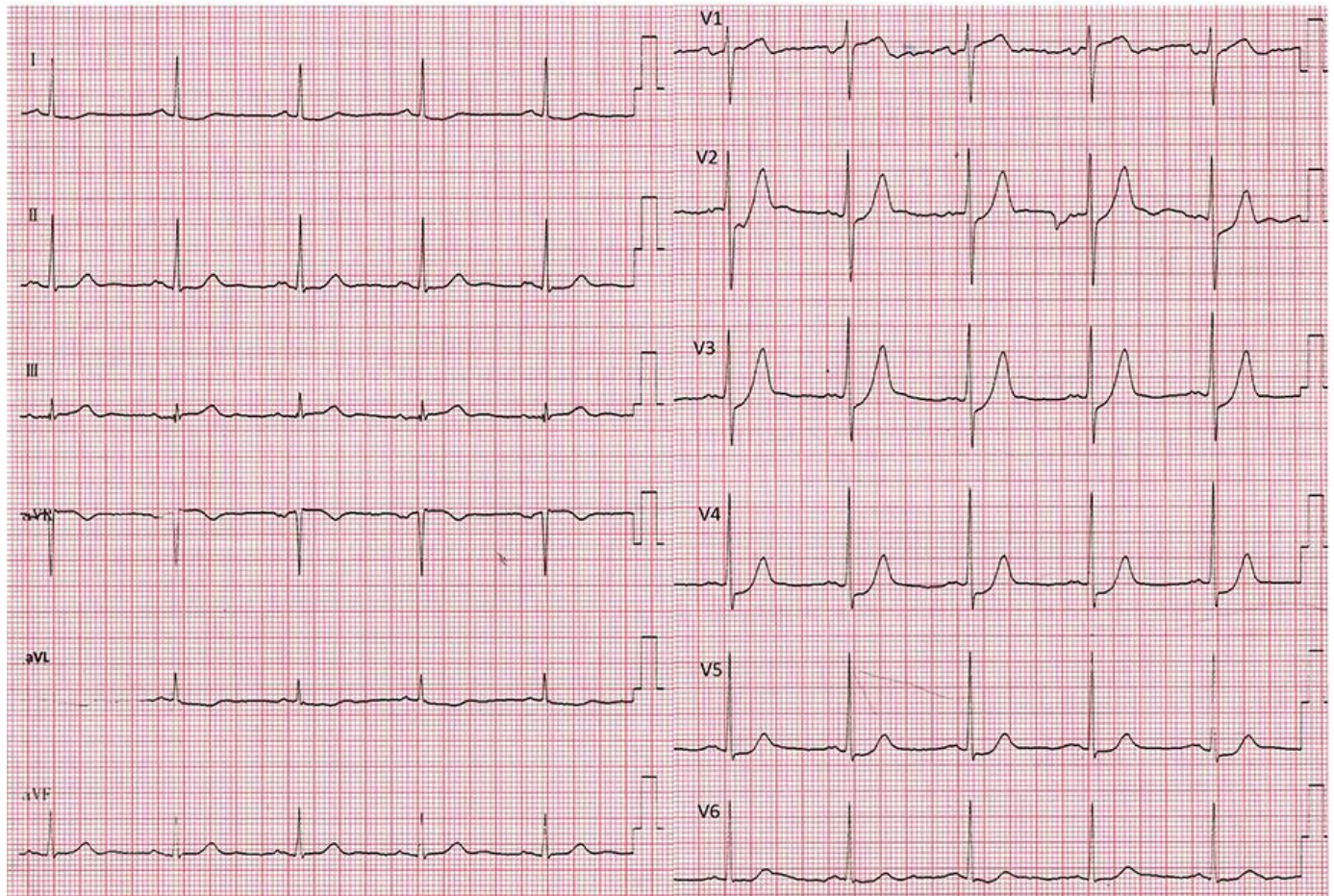
Avoid. Right-sided ST elevation can disappear early; a delayed negative recording does not exclude involvement.

CASE 35 | ISCHAEMIA AND ACUTE ILLNESS

De Winter pattern: an occlusion warning

PUBLISHED CASE

A 77-year-old man develops prolonged chest tightness during activity. His anterior leads show upsloping ST depression with strikingly tall, symmetric T waves.

Can dangerous coronary disease occur without classic ST elevation?

Wang et al. (2025), Figure 3B; CC BY 4.0.

Three findings to recognise**1**

Upsloping ST depression begins at the J point.

2

Tall, symmetric anterior T waves follow.

3

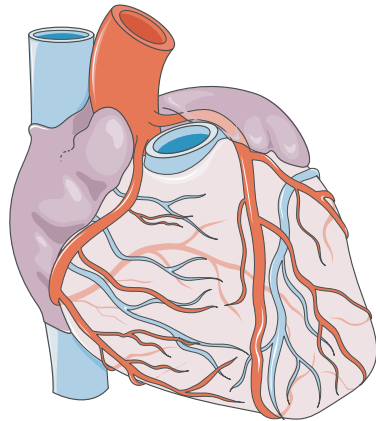
aVR may show modest ST elevation.

Remember: No classic elevation does not mean no occlusion.

CASE 35 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

De Winter pattern suggesting acute coronary occlusion



During ischaemia
ST depression + tall T waves



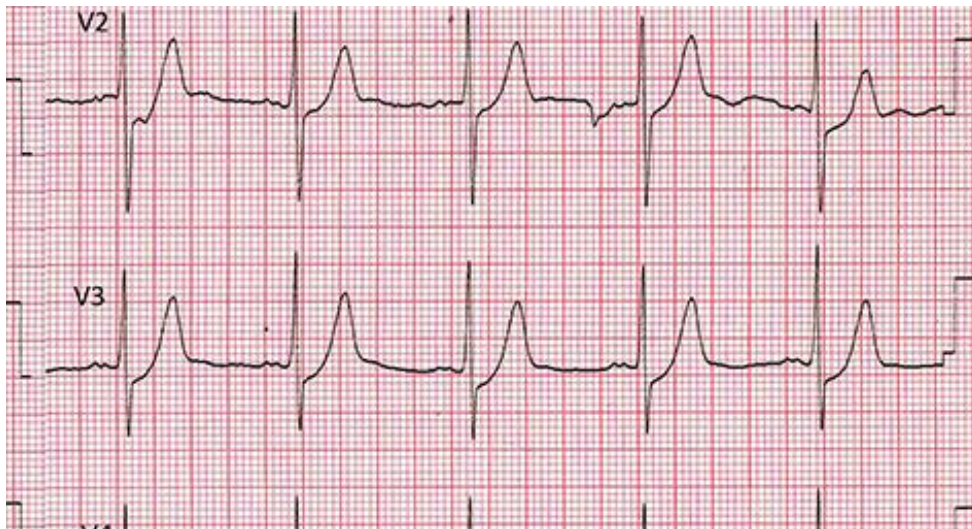
After pain eases
Biphasic / inverted T waves

Severe coronary disease

ECG evolves; coronary risk can persist.

Serial ECGs can reveal changing ischaemia and reperfusion; symptom relief does not establish coronary safety.

- Severe regional ischaemia alters ventricular activation and recovery.
- The pattern often accompanies severe LAD disease.
- Its exact mechanism is uncertain; the urgent clinical implication is clearer.



V2-V3: upsloping J-point depression followed by tall symmetric T waves. Wang et al. (2025), Figure 3B; CC BY 4.0.

In practice

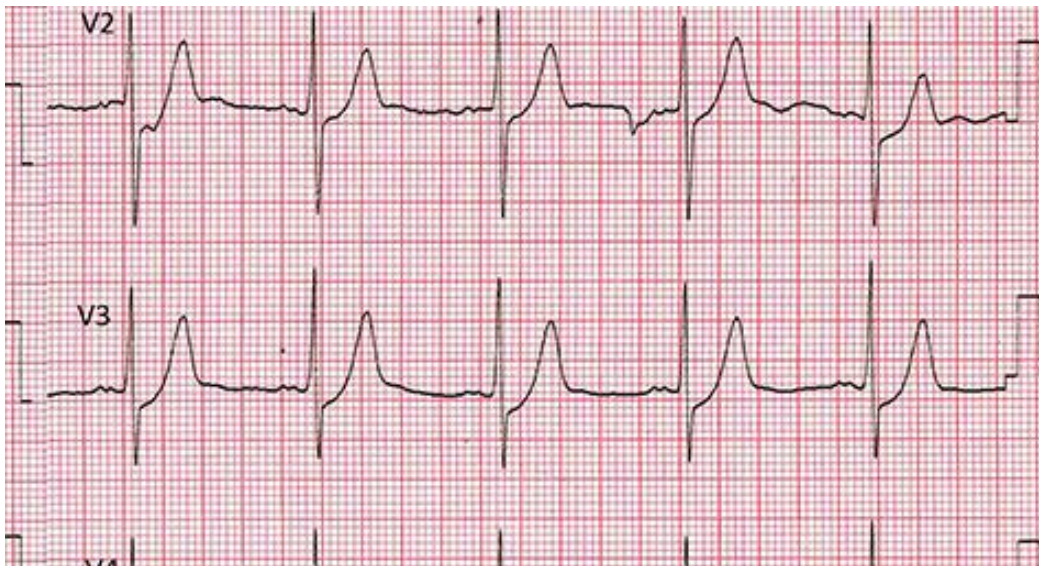
- Arrange immediate expert assessment for possible coronary occlusion.
- Use urgent coronary angiography and reperfusion pathways as clinically indicated.
- Repeat ECGs during changing symptoms; do not wait for conventional ST elevation.

Avoid. Tall T waves alone are not de Winter; assess the J point, distribution and symptoms.

CASE 35 | ANOTHER REAL ECG

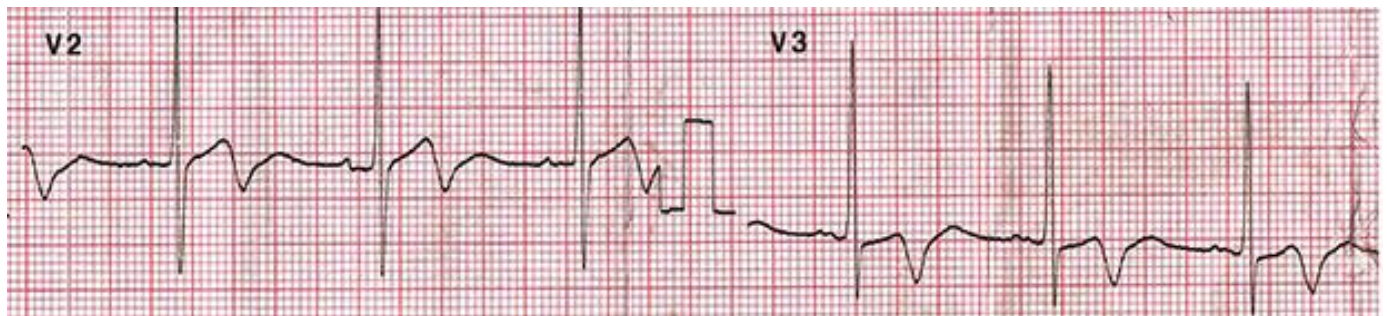
Compare the patterns

De Winter pattern suggesting acute coronary occlusion



V2-V3: upsloping J-point depression followed by tall symmetric T waves. Wang et al. (2025), Figure 3B; CC BY 4.0.

The same patient after pain eased



V2-V3 after pain relief: biphasic and inverted T waves. Wang et al. (2025), Figure 3C; CC BY 4.0.

- Anterior T waves become biphasic or inverted.
- Symptom relief does not remove coronary risk.

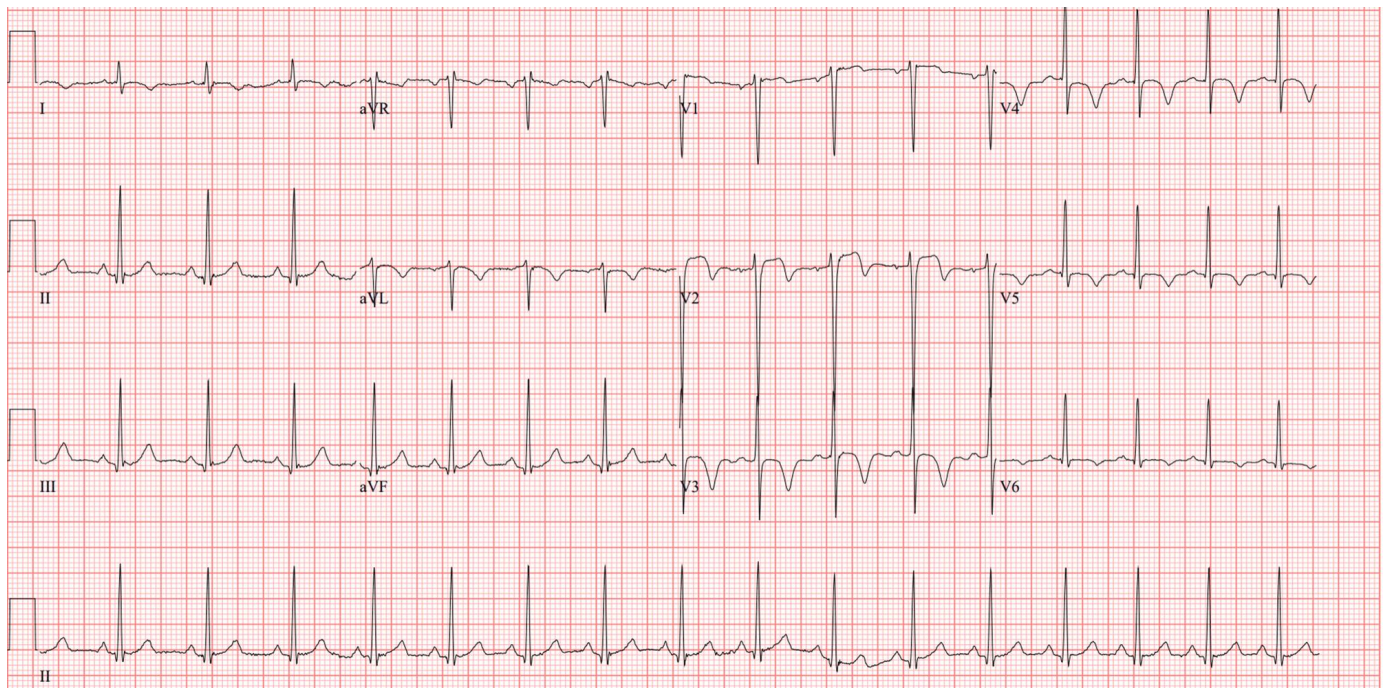
Remember. No classic elevation does not mean no occlusion.

CASE 36 | ISCHAEMIA AND ACUTE ILLNESS

Wellens pattern: danger between pain episodes

PUBLISHED CASE

A 43-year-old man has recurrent exertional chest pressure radiating to his jaw and arm. An ECG between painful episodes shows anterior T-wave abnormalities.

Why is this pain-free ECG still concerning?

Kim et al. (2023), Figure 1; CC BY 4.0.

Three findings to recognise**1**

V2 has a biphasic T wave.

2

V3-V5 show deep T-wave inversion.

3

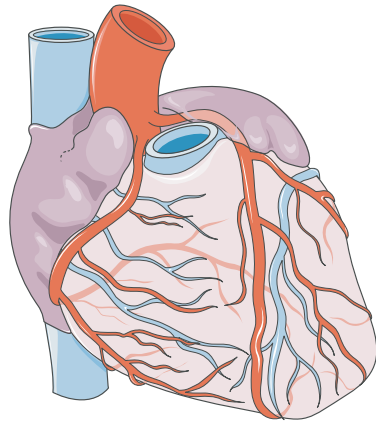
The pattern occurs after recurrent anginal symptoms.

Remember: Pain can disappear before the danger does.

CASE 36 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Wellens pattern associated with severe coronary disease



**Transient coronary
ischaemia**



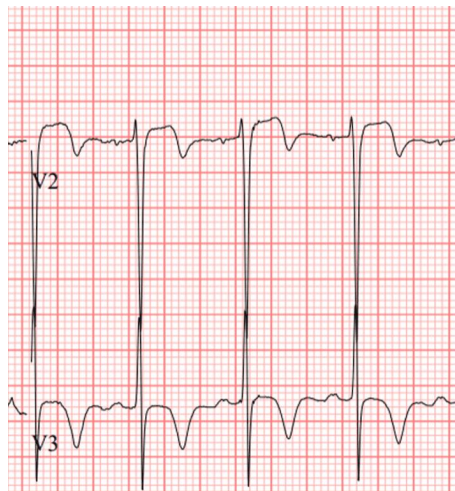
**After pain eases
Biphasic / inverted T waves**

Severe coronary disease

ECG evolves; coronary risk can persist.

Transient ischaemia and reperfusion can leave biphasic or inverted T waves after pain eases.

- Transient ischaemia and reperfusion alter regional ventricular recovery.
- Anterior T-wave inversion may remain after pain resolves.
- Angiography showed severe proximal LAD disease in this patient.



V2-V3: biphasic T in V2 and deep T inversion in V3. Kim et al. (2023), Figure 1; CC BY 4.0.

In practice

- Arrange urgent cardiology assessment and coronary evaluation.
- Avoid exercise stress testing when Wellens syndrome is suspected.
- Continue monitoring and serial ECG/troponin assessment through the acute coronary pathway.

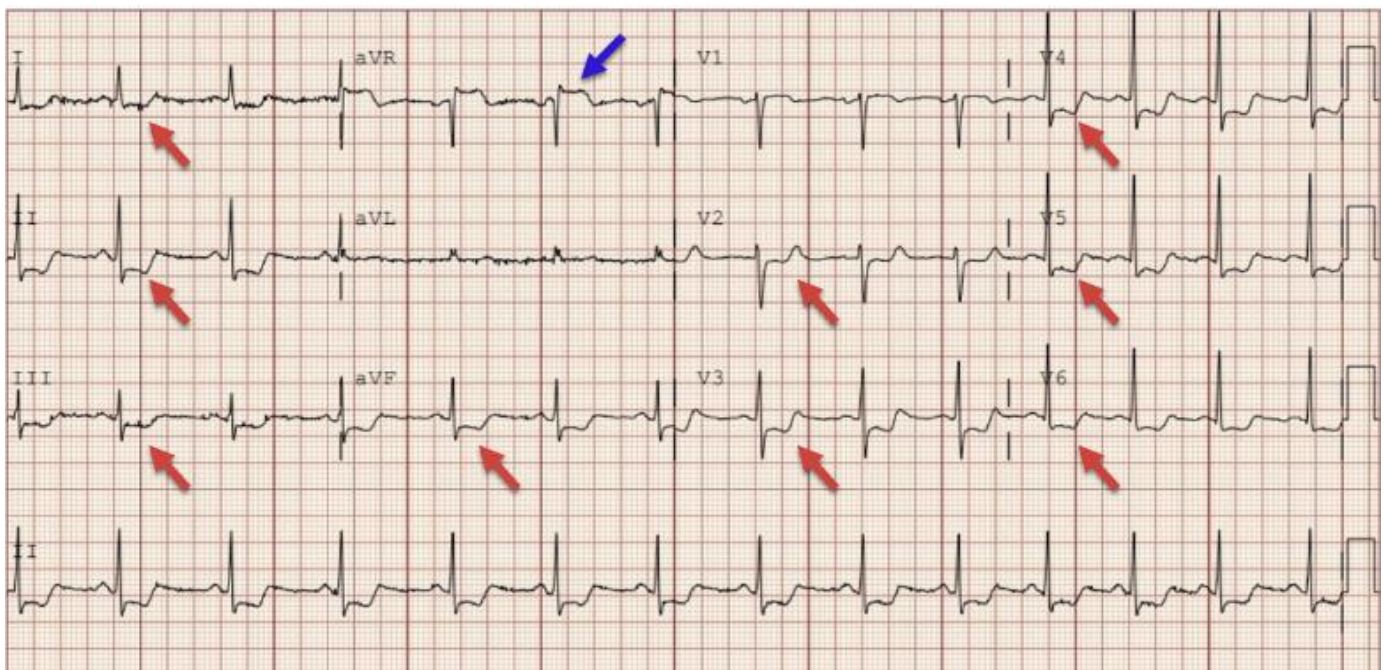
Avoid. Anterior T-wave inversion has mimics; the pattern must fit the history and remaining ECG.

CASE 37 | ISCHAEMIA AND ACUTE ILLNESS

Diffuse ST depression with aVR elevation

PUBLISHED CASE

A 33-year-old woman with antiphospholipid syndrome develops exertional chest pain and dyspnoea. Her ECG shows widespread ST depression with elevation in aVR.

What does this global pattern demand?

Lo et al. · Image 1 · CC BY 4.0

Three findings to recognise**1**

ST depression spans several lead territories.

2

aVR shows reciprocal ST elevation.

3

The pattern requires assessment of symptoms and circulatory status.

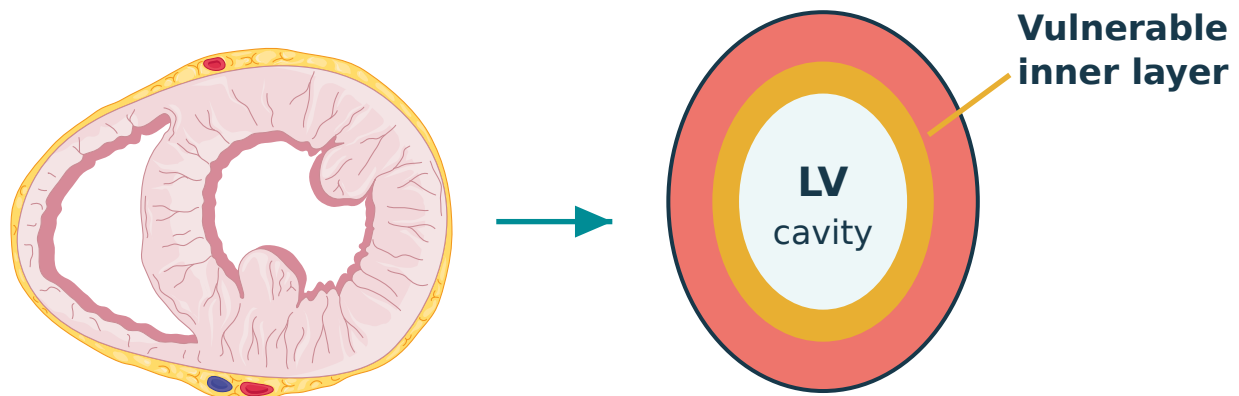
Remember: aVR is a warning light, not an angiogram.

CASE 37 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

High-risk pattern of widespread subendocardial ischaemia

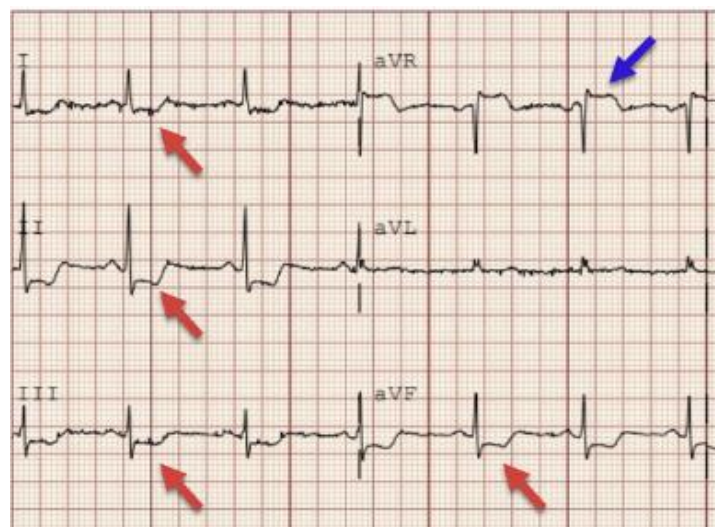
Ventricular cross-section



Low supply or high demand → uneven voltages

Subendocardial ischaemia changes voltage relationships across the ventricular wall.

- Widespread subendocardial ischaemia creates a global injury-current gradient.
- The net ST vector points towards aVR.
- Coronary disease or severe supply-demand imbalance can produce this pattern.



Lo et al. · Image 1 · CC BY 4.0

In practice

- Assess haemodynamic stability and seek urgent expert review.
- Investigate acute coronary syndrome and important supply-demand triggers together.
- Persistent ischaemia or instability warrants urgent invasive assessment.

Avoid. This pattern is not specific for left main occlusion; this patient had severe ostial left main disease.

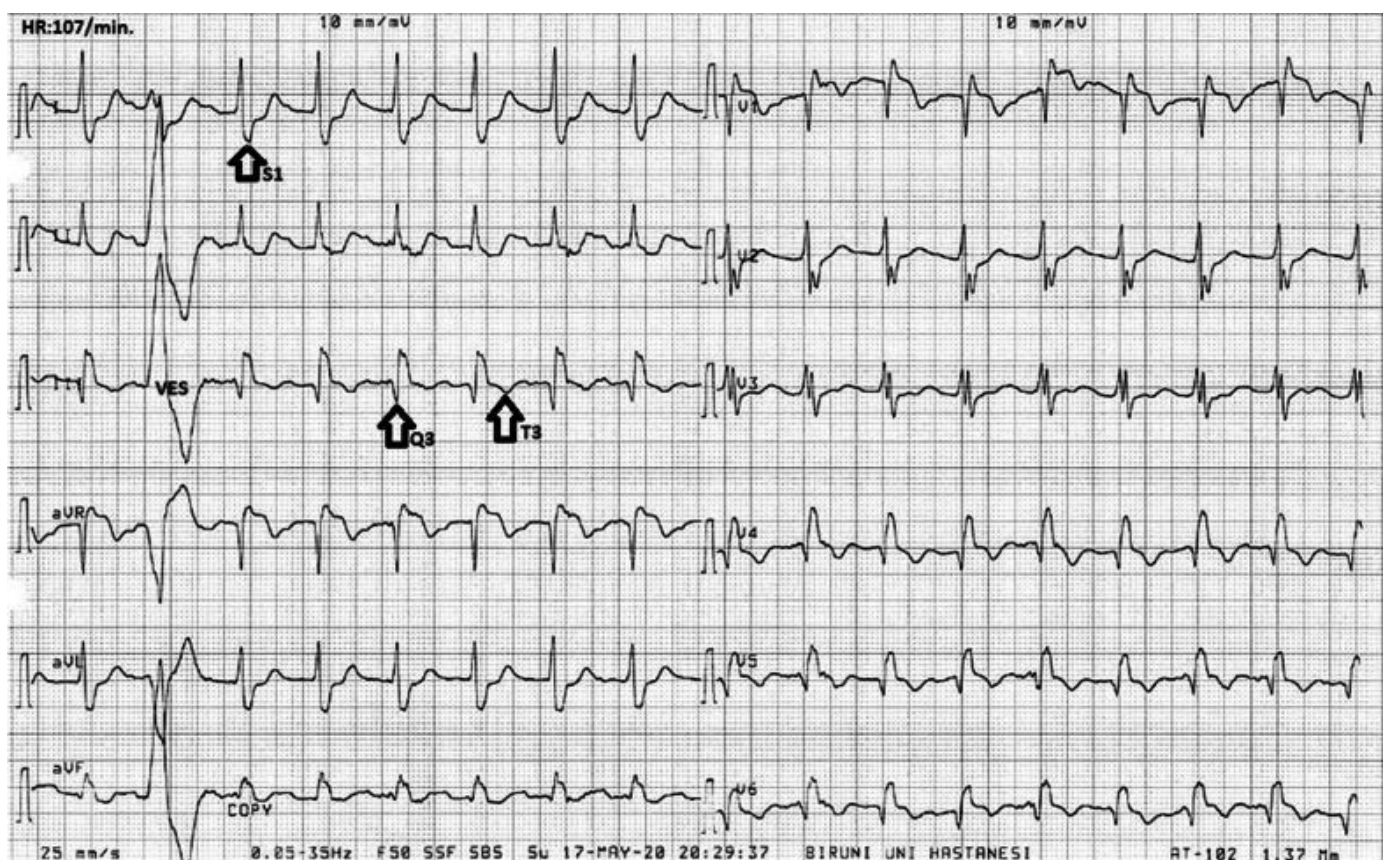
CASE 38 | ISCHAEMIA AND ACUTE ILLNESS

Pulmonary embolism: the strained right ventricle

PUBLISHED CASE

A 61-year-old man presents with dyspnoea, chest pain and haemoptysis. He is tachycardic, hypoxaemic and hypotensive; CT subsequently confirms bilateral pulmonary emboli.

How does pulmonary obstruction change the ECG?



Islamoglu et al. (2021), Figure 1; CC BY 4.0.

Three findings to recognise

1

A deep S in I accompanies Q and inverted T in III.

2

Right bundle branch block is present.

3

Additional repolarisation changes reflect acute right ventricular stress.

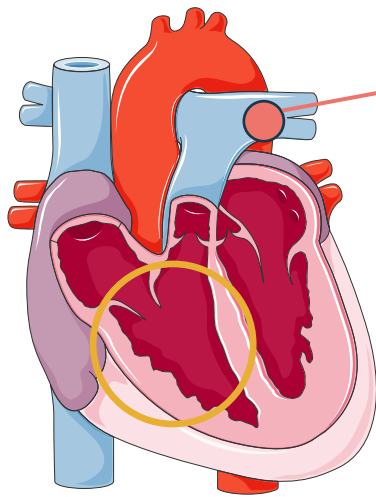
Remember: The ECG may reveal the strain, not the clot.

References: V4-PE-2021, V4-PE-GUIDELINE-2026, V4-RCUK-SPECIAL-2025

CASE 38 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Acute right ventricular strain in pulmonary embolism



Pulmonary arterial clot

Pulmonary resistance rises



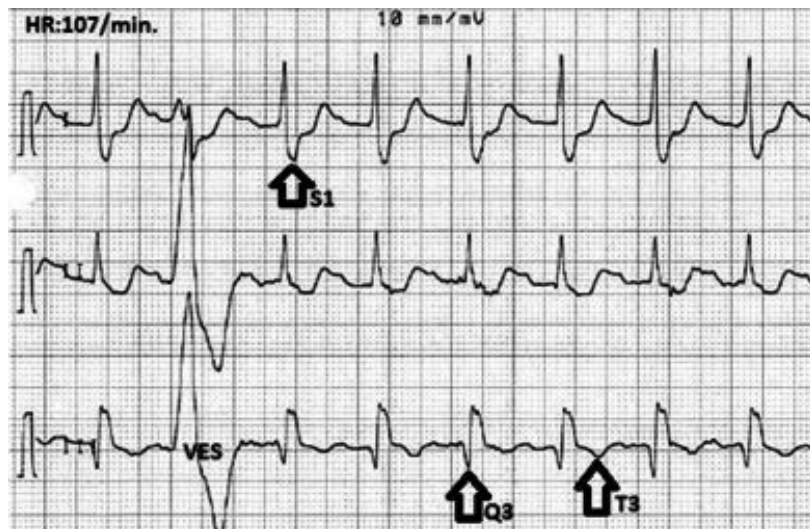
RV pressure and wall stress rise

QRS and T-wave changes may follow

Acute right ventricular strain

A pulmonary arterial obstruction acutely increases right ventricular afterload and may produce electrical strain.

- Emboli obstruct the pulmonary arterial circulation.
- Right ventricular pressure and wall stress rise abruptly.
- Dilatation, ischaemia and delayed activation can change QRS and T waves.



Limb-lead detail: deep S in I with Q and inverted T in III. Islamoglu et al. (2021), Figure 1; CC BY 4.0.

In practice

- Assess haemodynamic stability and activate the local pulmonary embolism pathway.
- Use clinical probability and appropriate imaging; do not diagnose from the ECG alone.
- Instability requires urgent senior assessment and a rapid bedside diagnostic strategy.

Avoid. S1Q3T3 is neither sensitive nor specific for pulmonary embolism; a normal ECG cannot exclude it.

CASE 39 | ARRHYTHMIAS

Premature ventricular beats

CLINICAL PRESENTATION

A 34-year-old feels a missed beat followed by a thump. There is no syncope or exertional chest pain. Recording during symptoms catches an early complex.

What caused the early beat and the stronger beat afterwards?



MIT-BIH 100 | source 1514 s | 0-10 s, simultaneous | MLII, V5
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise**1**

An early complex has a different shape in both channels.

2

A longer interval follows the early beat.

3

The preceding rhythm resumes afterwards.

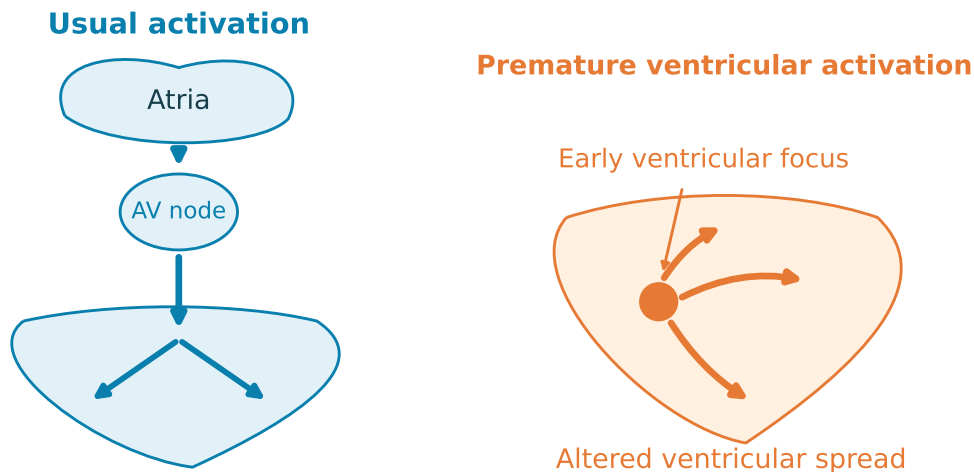
Remember: Read the beat before, the early beat and the beat after.

CASE 39 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

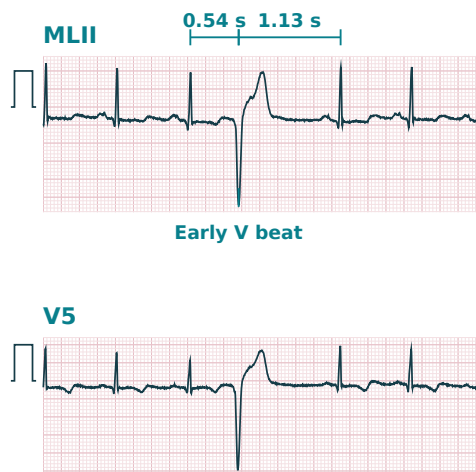
Premature ventricular complex

An early ventricular focus changes activation



An early ventricular focus can activate the ventricles through a different route, changing QRS shape and beat timing.

- An early ventricular impulse takes a different activation route.
- Short filling time can make its pulse difficult to feel.
- The next contraction may feel stronger after the longer interval.



MIT-BIH 100 | source 1514 s | 2.7-7.6 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

- Ask about syncope, exertional symptoms, heart disease and family history.
- Use monitoring to relate symptoms to rhythm and measure recurrent ectopy.
- Assess structure when frequency, symptoms or other findings suggest disease.

Avoid. A broad early QRS can also be an aberrantly conducted PAC.

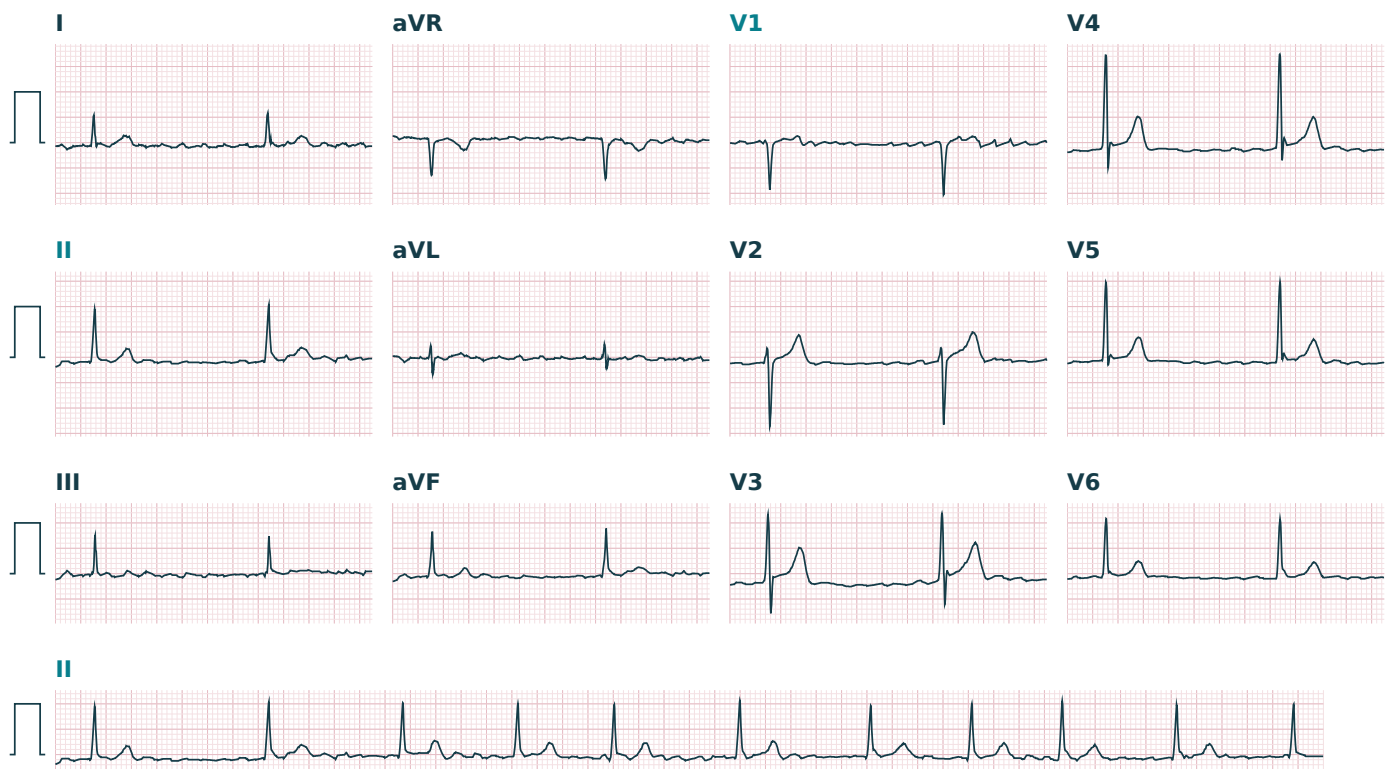
CASE 40 | ARRHYTHMIAS

Atrial fibrillation

CLINICAL PRESENTATION

A 72-year-old with hypertension reports fatigue and palpitations. The pulse is irregular, blood pressure is preserved and there is no pulmonary oedema.

Is there a consistent atrial rhythm before the QRS complexes?



PTB-XL 00330 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Successive R-R intervals vary substantially.

2

No consistent P-before-QRS sequence can be followed.

3

Most QRS complexes retain a similar shape.

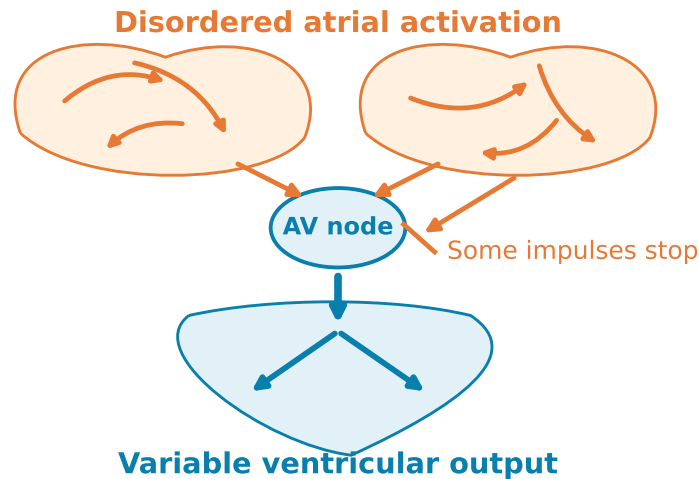
Remember: Control symptoms and prevent stroke: two separate decisions.

CASE 40 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Atrial fibrillation

Atrial fibrillation: disordered input, filtered output



Disordered atrial activation meets an AV node whose changing recovery allows only some impulses to reach the ventricles.

- Disordered activation replaces coordinated atrial depolarisation.
- The AV node conducts only some arriving impulses.
- Variable transmission produces an irregular ventricular rhythm.



PTB-XL 00330 | 0-10 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

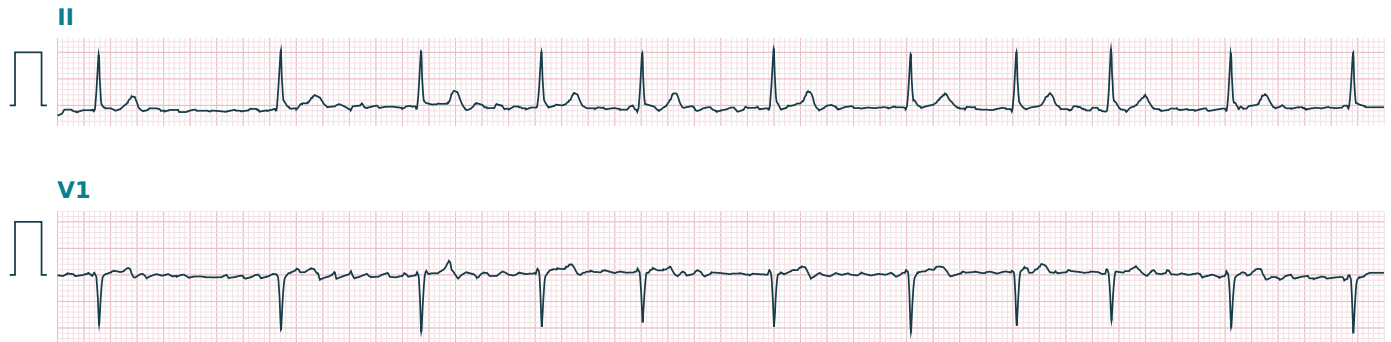
- Assess whether AF is causing haemodynamic instability.
- Plan rate or rhythm control around symptoms and ventricular function.
- Assess stroke prevention separately, including the cardioversion anticoagulation strategy.

Avoid. A comfortable patient with a controlled rate may still need anticoagulation.

CASE 40 | ANOTHER REAL ECG

Compare the patterns

Atrial fibrillation



PTB-XL 00330 | 0-10 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

AF with a faster ventricular response



PTB-XL 04117 | 0-10 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- The ventricular rate changes; the atrial diagnosis remains AF.
- Assess haemodynamic consequences rather than the rate alone.

Remember. Control symptoms and prevent stroke: two separate decisions.

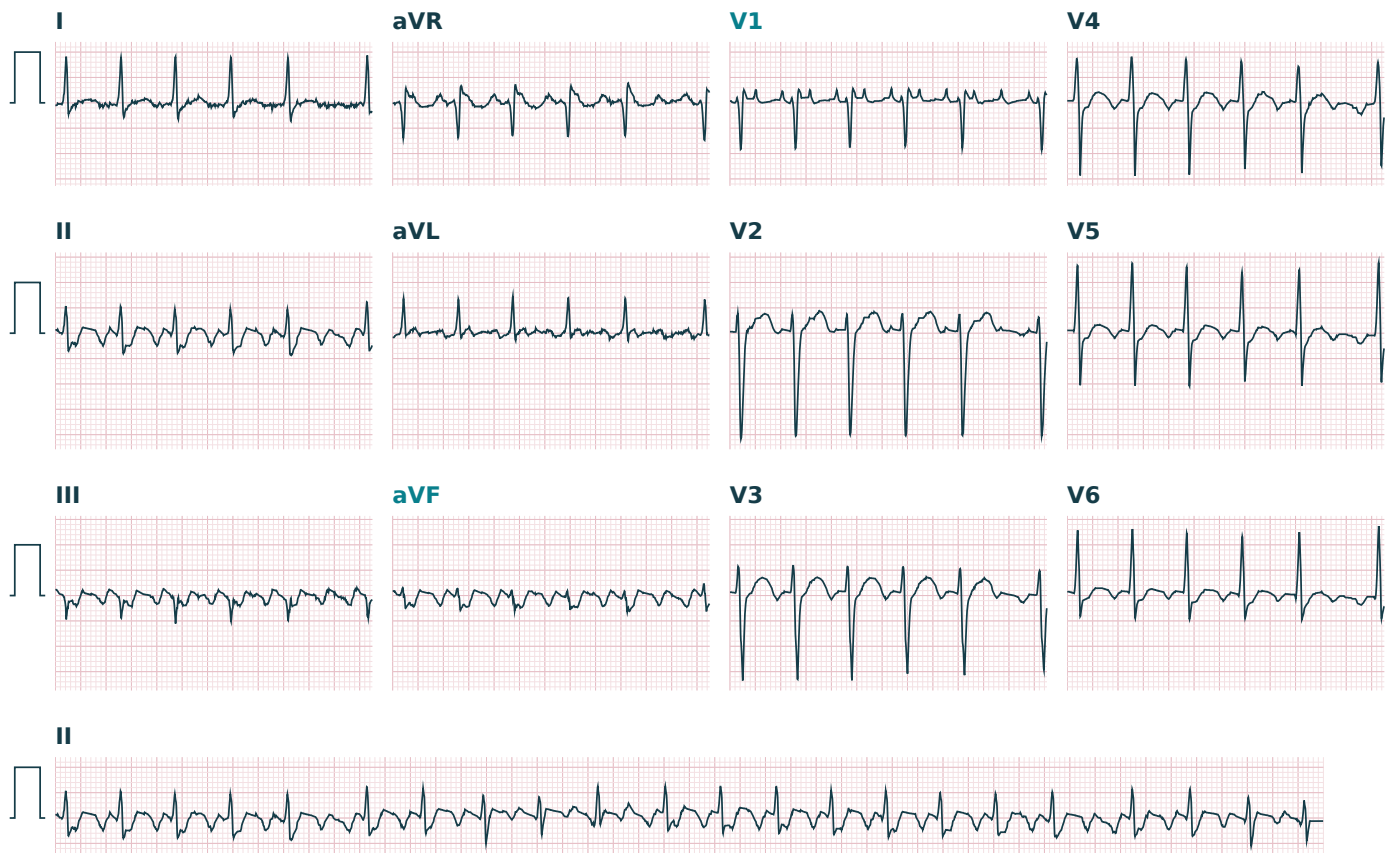
CASE 41 | ARRHYTHMIAS

Atrial flutter

CLINICAL PRESENTATION

A 61-year-old presents with persistent palpitations and reduced exercise tolerance. The pulse is rapid and regular, but repeated atrial waves appear between the ventricular complexes.

Are the ventricles showing only part of the atrial rhythm?



PTB-XL 00018 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Repeated atrial deflections continue between QRS complexes.

2

The ventricular sequence is comparatively regular.

3

Inferior leads and V1 show different views of the atrial waves.

Remember: Count the atria first; the ventricles may follow only alternate impulses.

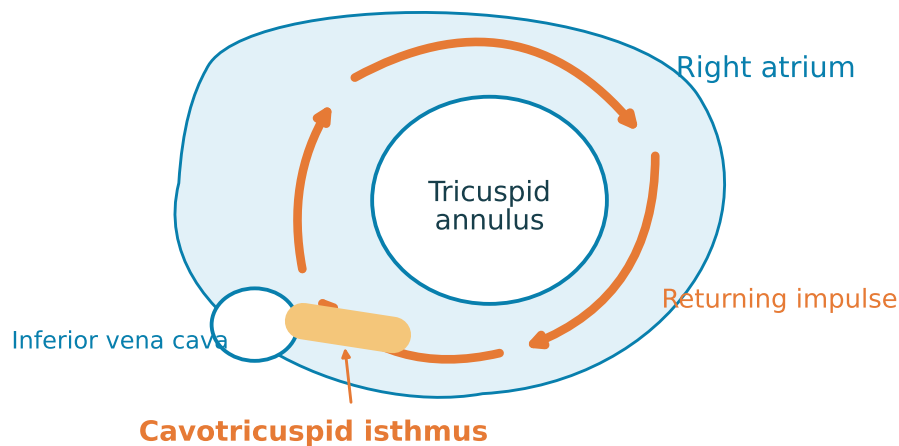
References: C09

CASE 41 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Atrial flutter

Typical flutter: a right-atrial circuit



A typical flutter circuit circles the tricuspid annulus and crosses the cavotricuspid isthmus. The AV node controls ventricular transmission.

- Organised re-entry repeatedly activates the atria.
- Typical flutter circulates around the tricuspid annulus through the isthmus.
- The AV node determines how many atrial impulses reach the ventricles.



PTB-XL 00018 | 0-4 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Assess stability and preserve a twelve-lead tracing.
- Review episode duration and thromboembolic protection before elective cardioversion.
- Discuss ablation for recurrent symptomatic typical flutter.

Avoid. A ventricular rate near 150 does not establish sinus tachycardia.

CASE 41 | ANOTHER REAL ECG

Compare the patterns

Atrial flutter



PTB-XL 00018 | 0-4 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

A second recording of organised flutter activity



PTB-XL 00023 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

- Follow atrial waves through several ventricular cycles.
- Atrial organisation distinguishes flutter from AF.

Remember. Count the atria first; the ventricles may follow only alternate impulses.

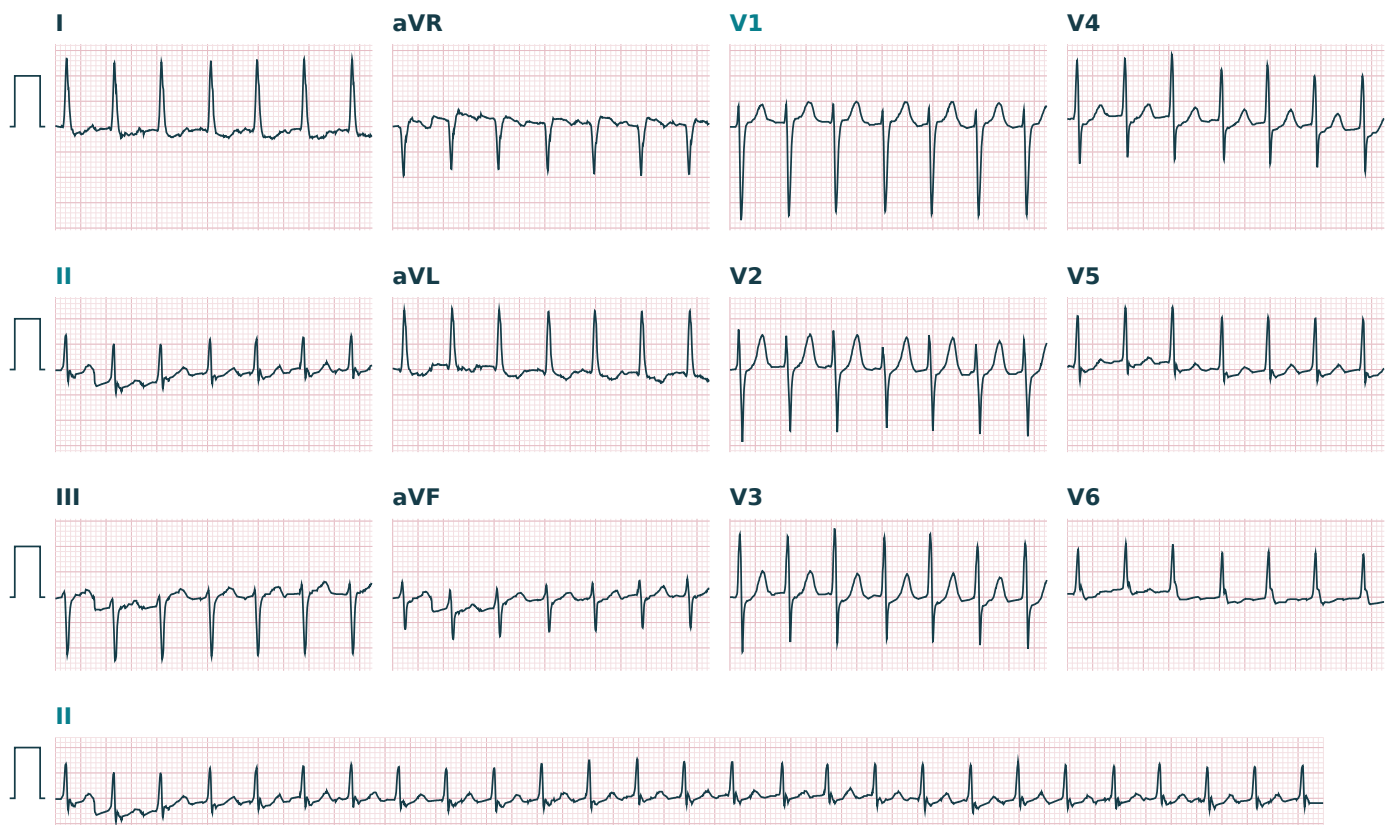
CASE 42 | ARRHYTHMIAS

Regular narrow-complex tachycardia

CLINICAL PRESENTATION

A 27-year-old develops sudden, steady palpitations. There is no fever or bleeding. Consciousness and blood pressure remain normal; the ECG shows a regular narrow-complex tachycardia.

Which observations could distinguish a returning circuit from an atrial focus?



PTB-XL 07889 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

QRS complexes recur rapidly at similar intervals.

2

Ventricular activation remains relatively narrow.

3

A separate P wave is difficult to identify consistently.

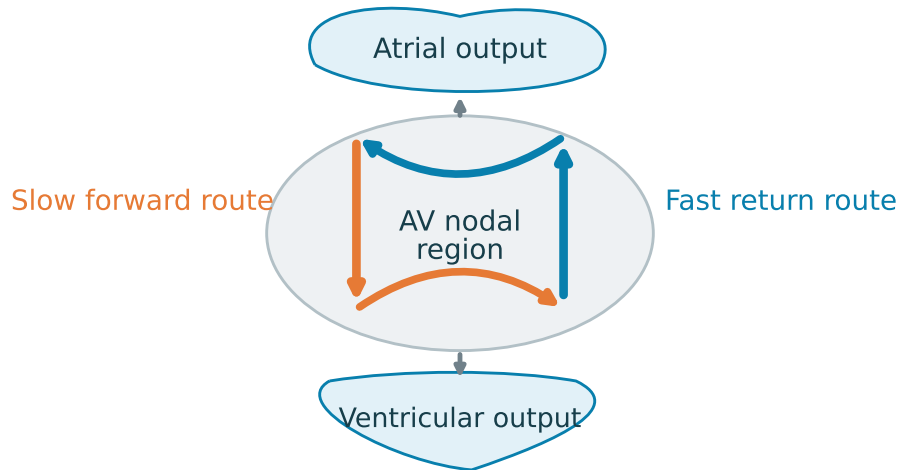
Remember: Save the onset and termination: they can reveal the circuit.

CASE 42 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

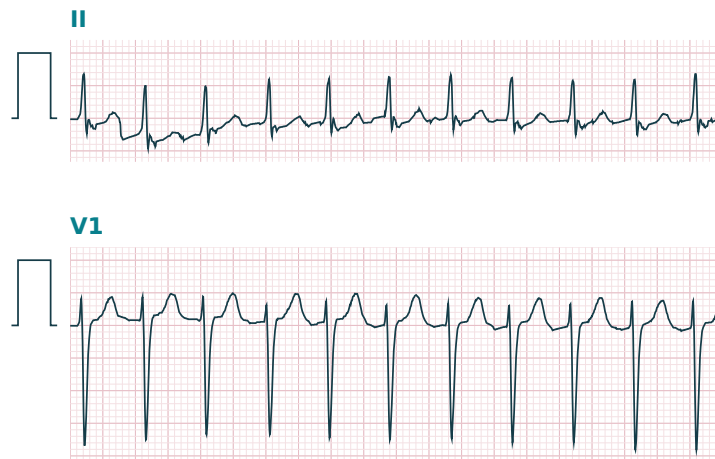
Regular supraventricular tachycardia

AVNRT: slow forward, fast return



Typical AVNRT uses slow forward and fast returning conduction in the AV nodal region; atrial and ventricular activation are outputs.

- AVNRT can use slow forward and fast returning nodal conduction.
- AVRT returns through an accessory pathway after ordinary forward AV conduction.
- Surface timing supports a mechanism but does not always prove it.



PTB-XL 07889 | 0-4 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Assess circulation and record the twelve-lead ECG.
- For stable regular narrow SVT, use vagal manoeuvres then monitored protocol-based treatment.
- Preserve the treatment strip; arrange specialist review for recurrent symptomatic episodes.

Avoid. A short RP interval does not prove AVNRT.

CASE 42 | ANOTHER REAL ECG

Compare the patterns

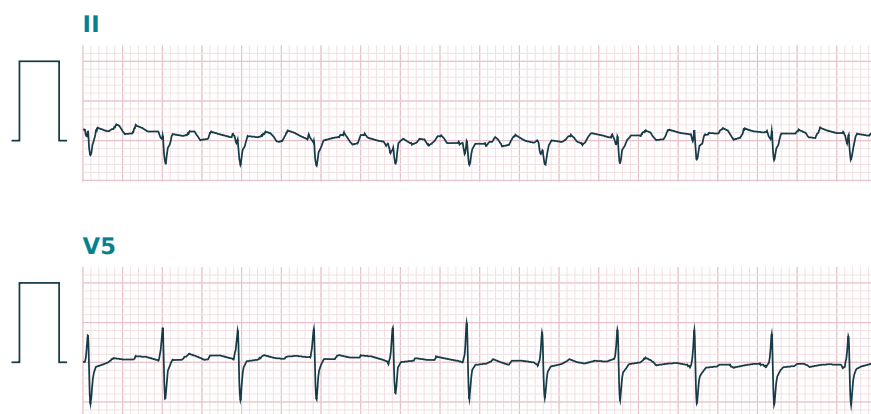
Regular supraventricular tachycardia



PTB-XL 07889 | 0-4 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Another regular SVT: similar rhythm, potentially different mechanism



PTB-XL 02051 | 0-4 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- A regular narrow rhythm does not specify the circuit.
- Inspect P-wave timing and the response to AV block.

Remember. Save the onset and termination: they can reveal the circuit.

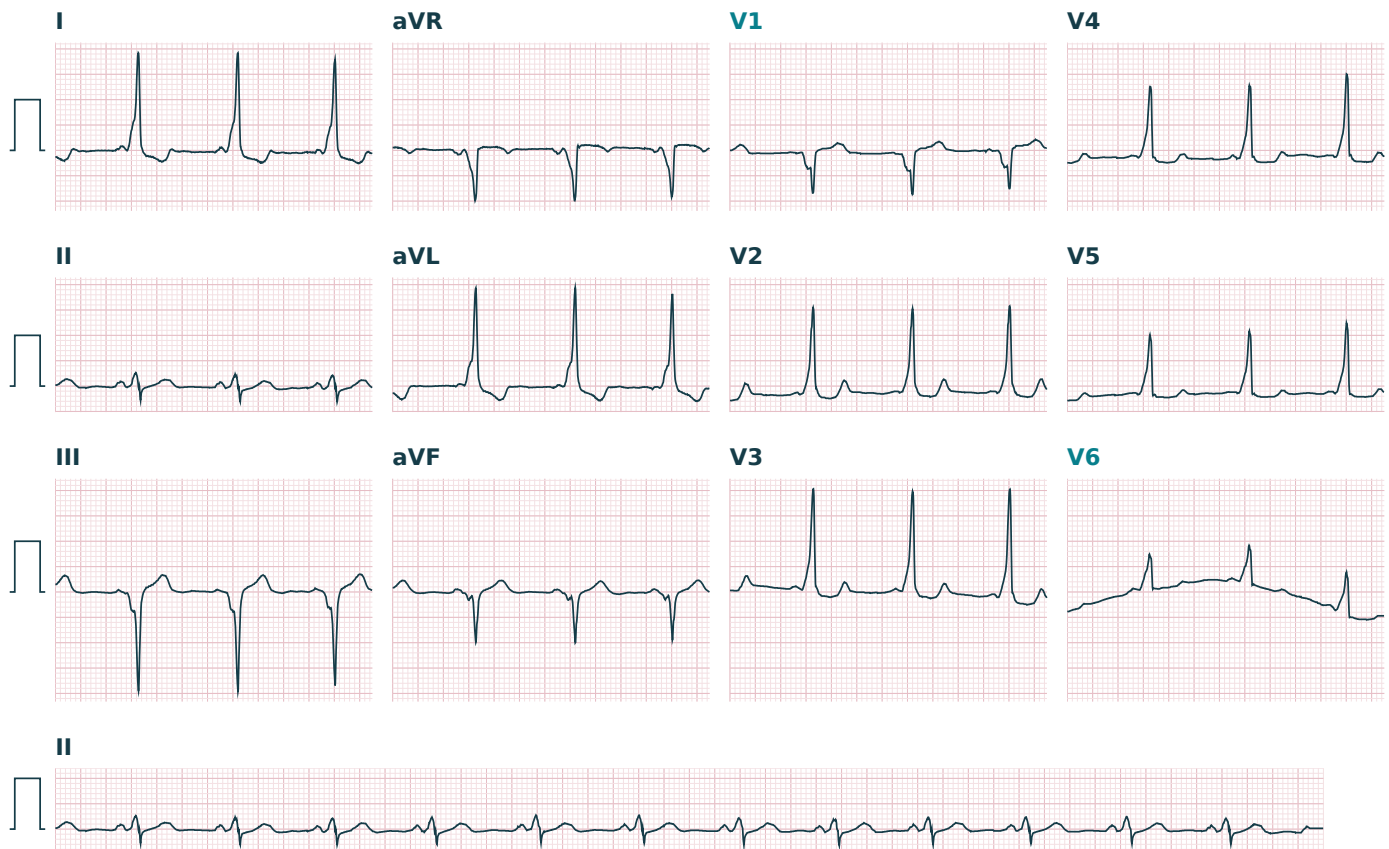
CASE 43 | ARRHYTHMIAS

Ventricular pre-excitation

CLINICAL PRESENTATION

A 23-year-old describes recurrent abrupt palpitations. Between episodes, the ECG has a subtle slurred QRS onset. The clinician examines the first part of ventricular activation carefully.

Does ventricular activation begin before the sharp R-wave upstroke?



PTB-XL 02145 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

The initial QRS transition is slurred.

2

Different leads show different projections of the early activation.

3

Measure QRS duration from the earliest delta-wave onset.

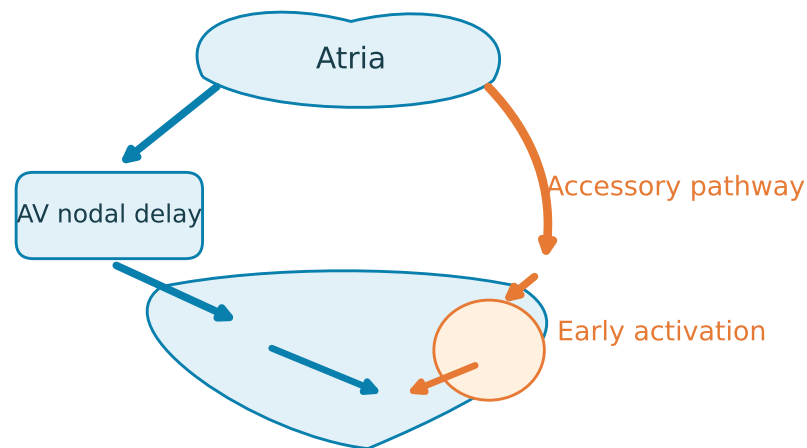
Remember: The QRS starts at the delta wave, not the sharp R.

CASE 43 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Manifest ventricular pre-excitation

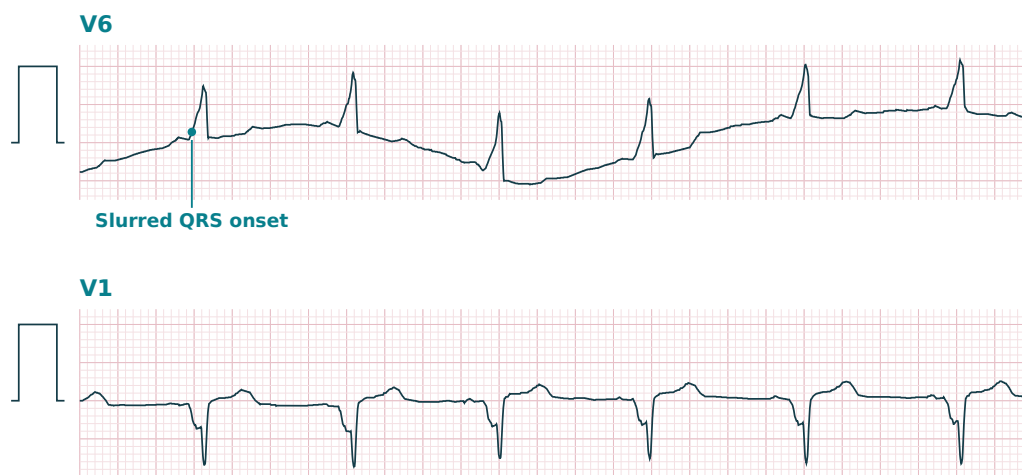
Pre-excitation: an early ventricular head start



Fusion of activation fronts

An accessory route activates part of the ventricle early; later His–Purkinje activation joins it to form the fused QRS.

- An accessory pathway allows early atrium-to-ventricle activation.
- Early slow muscle activation forms the delta wave.
- Later His–Purkinje activation joins it to complete the QRS.



PTB-XL 02145 | 0.5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
 PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Document the pattern and the history of symptomatic episodes.
- Arrange specialist assessment of pathway properties and treatment options.
- Recognise rapid irregular broad tachycardia as possible pre-excited AF.

Avoid. AV nodal blockers are hazardous in pre-excited AF, including intravenous amiodarone.

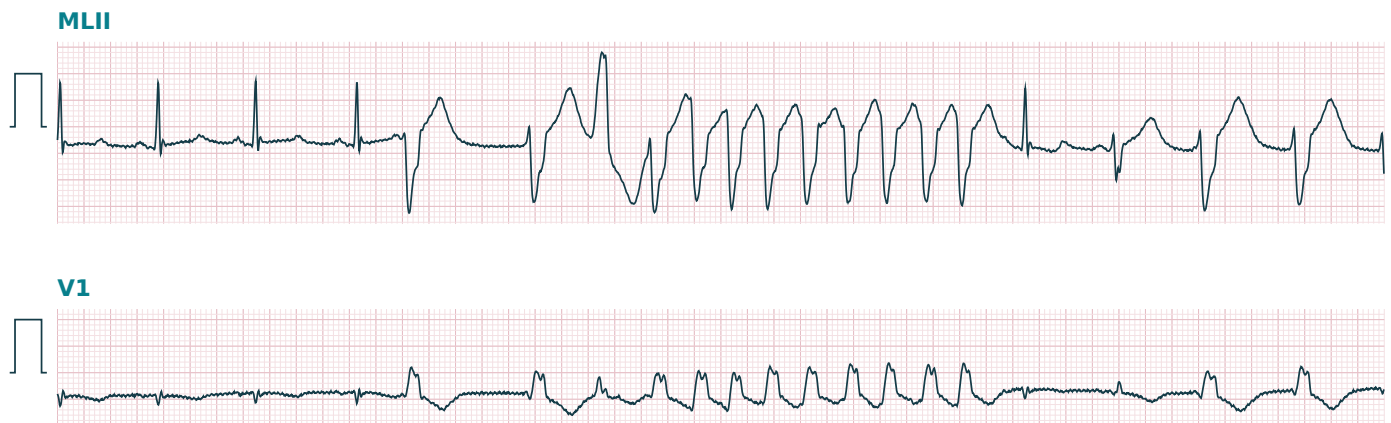
CASE 44 | ARRHYTHMIAS

Wide-complex tachycardia

CLINICAL PRESENTATION

A patient with previous myocardial infarction develops rapid palpitations and dizziness. The monitor changes from ordinary complexes to a broad tachycardia; a pulse is still present.

Does a preserved pulse make ventricular tachycardia unlikely?



MIT-BIH 205 | source 1458 s | 0-10 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise**1**

The rhythm accelerates with a major change in QRS shape.

2

The recording preserves the onset and return to the preceding rhythm.

3

One changing complex does not establish capture or fusion.

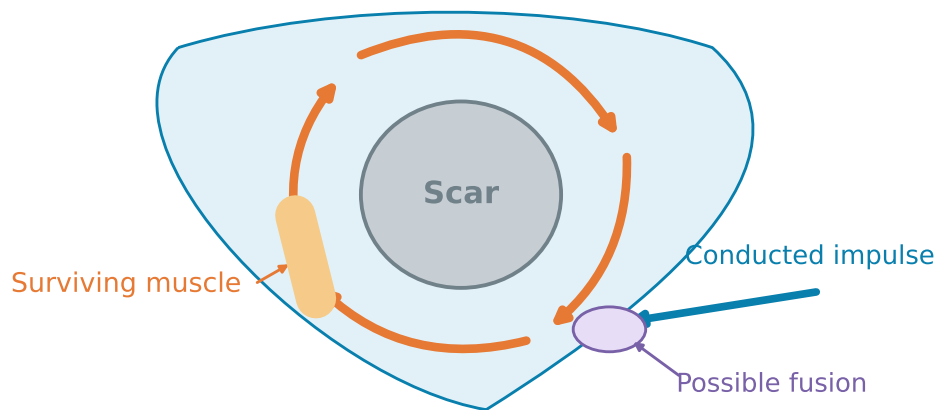
Remember: Tolerance describes the patient; it does not identify the rhythm.

CASE 44 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

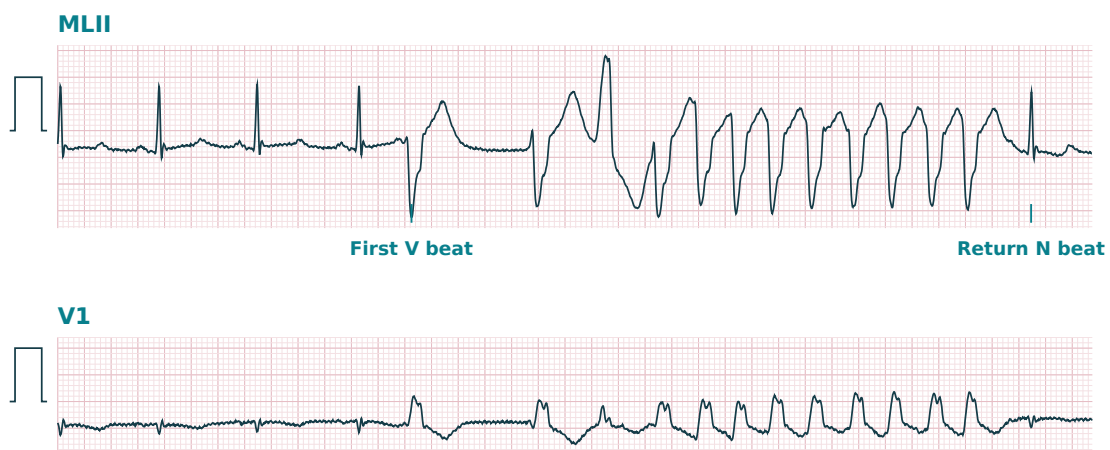
Treat undifferentiated wide-complex tachycardia as VT

Ventricular re-entry: a route around scar



Surviving muscle can support ventricular re-entry around scar. A conducted impulse meeting ventricular activation can produce fusion.

- Ventricular activation can start outside the usual supraventricular route.
- Scar-related re-entry is one possible mechanism of monomorphic VT.
- Slower muscle-to-muscle spread produces broad ventricular complexes.



MIT-BIH 205 | source 1458 s | 0-7.75 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

- Assess pulse and circulation immediately; treat an uncertain broad tachycardia as VT.
- If sustained monomorphic VT causes instability, use urgent synchronised cardioversion.
- Preserve the tracing and investigate structural, ischaemic and reversible causes.

Avoid. Alertness and a maintained blood pressure do not exclude VT.

CASE 45 | ARRHYTHMIAS

Brief ventricular runs

CLINICAL PRESENTATION

A patient admitted after near-syncope has two brief broad-complex runs on telemetry. Both stop before review. The ward asks whether spontaneous termination makes the episodes harmless.

What does the lead-in and recovery add to the diagnosis?

MIT-BIH 205 | source 295 s | 0-14 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise**1**

Two separate rapid ventricular runs are visible.

2

The second onset contains a source-labelled fusion beat.

3

Both runs return to the preceding ventricular sequence.

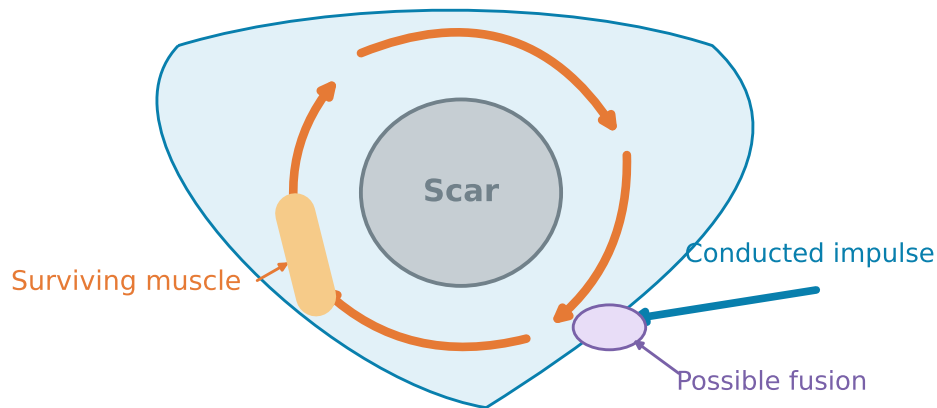
Remember: Keep the lead-in, the run and the recovery.

CASE 45 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

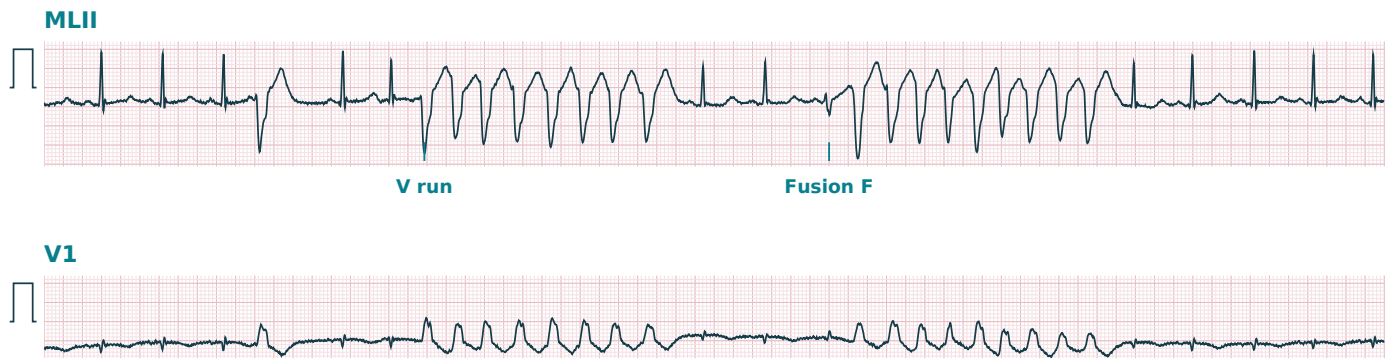
Recurrent non-sustained ventricular tachycardia

Ventricular re-entry: a route around scar



Surviving muscle can support ventricular re-entry around scar. A conducted impulse meeting ventricular activation can produce fusion.

- Repeated ventricular activation can follow one stable route.
- A conducted impulse meeting ventricular activation may create fusion.
- Termination reveals baseline conduction and repolarisation.



MIT-BIH 205 | source 295 s | 0-14 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

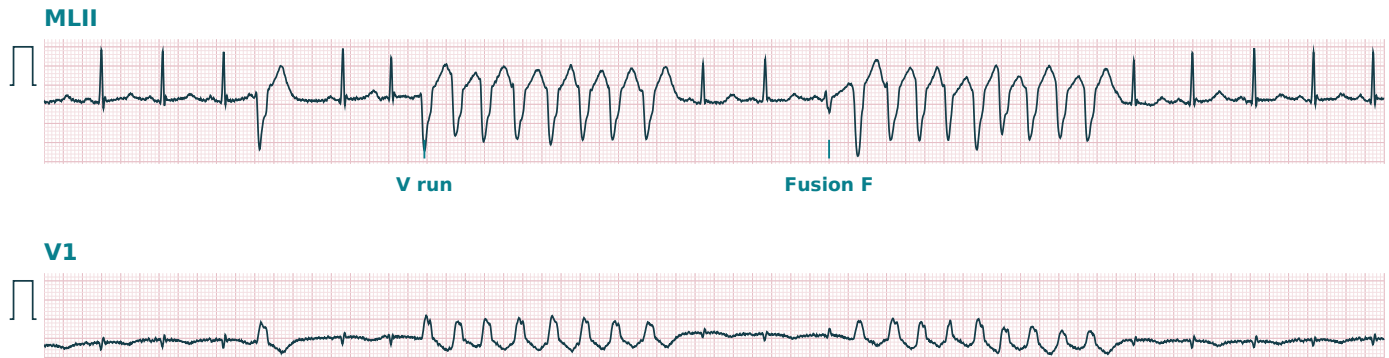
- Match event times to symptoms, especially syncope or near-syncope.
- Assess ventricular function, medicines, electrolytes and possible ischaemia.
- Use monitoring and specialist assessment to define recurrence and risk.

Avoid. Short duration alone does not establish low risk.

CASE 45 | ANOTHER REAL ECG

Compare the patterns

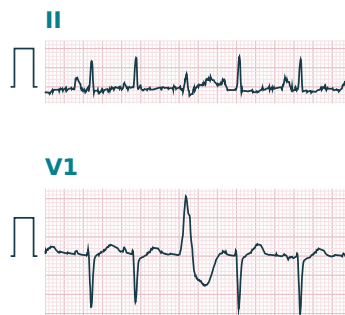
Recurrent non-sustained ventricular tachycardia



MIT-BIH 205 | source 295 s | 0-14 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

An isolated PVC is different from a ventricular run



PTB-XL 04953 | 3.8-7 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- One altered early complex interrupts the baseline rhythm.
- Count consecutive ventricular beats before describing a run.

Remember. Keep the lead-in, the run and the recovery.

CASE 46 | ARRHYTHMIAS

A missing ventricular beat

CLINICAL PRESENTATION

A 74-year-old with light-headedness has a sudden pause on monitoring. A small atrial deflection remains inside the gap, prompting review of atrial and ventricular timing separately.

Did atrial activity stop, or did conduction fail?



MIT-BIH 231 | source 2 s | 0-12 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise**1**

The ventricular interval is longer than neighbouring intervals.

2

A small atrial deflection remains visible inside the gap.

3

Conducted complexes before and afterwards have similar shapes.

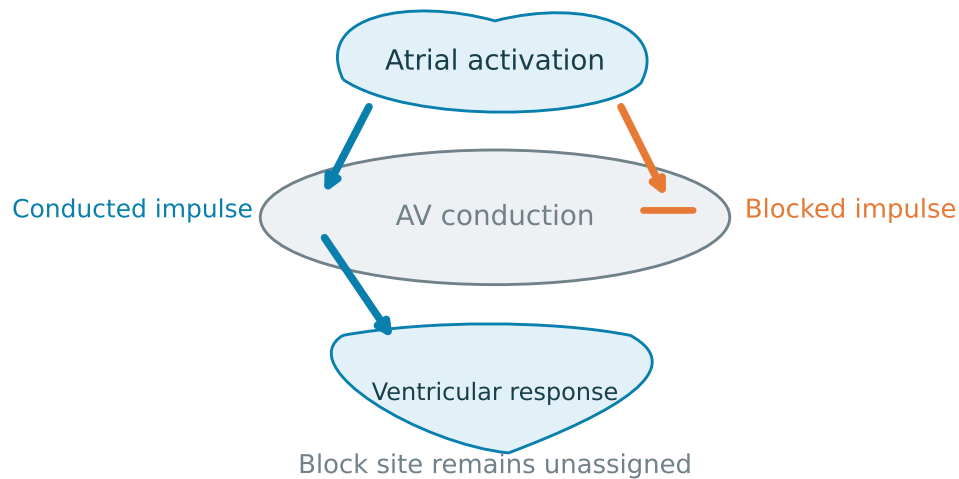
Remember: A missing QRS is a question about its preceding P wave.

CASE 46 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

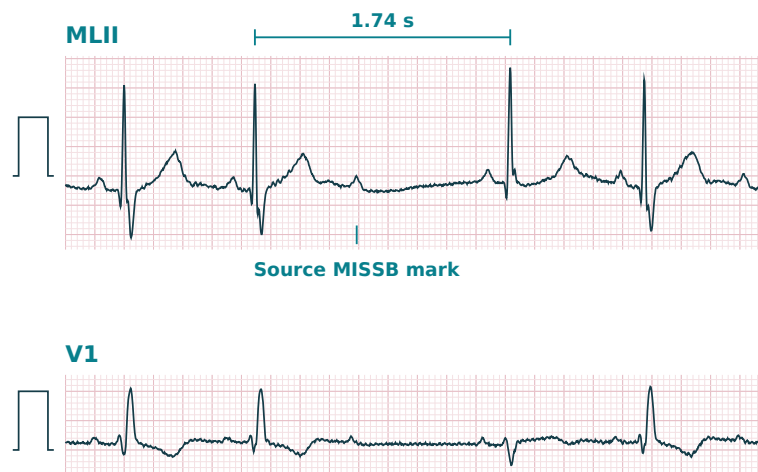
Intermittent failure of AV conduction

AV conduction: one impulse is blocked



The atrial event within the gap has no QRS response; this surface sequence does not establish the anatomical block site.

- Atrial depolarisation can continue while ventricular activation pauses.
- A nonconducted atrial impulse leaves a P wave without its QRS.
- An early blocked PAC causes a different atrial timing pattern.



MIT-BIH 231 | source 2 s | 1.8-6.6 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

- Obtain a longer recording and correlate the episode with symptoms.
- Review medicines, reversible causes and baseline conduction disease.
- Significant symptomatic block needs urgent monitoring and readiness to support the rate.

Avoid. An isolated 2:1 sequence cannot distinguish Mobitz I from Mobitz II.

CASE 47 | ARRHYTHMIAS

Blocked premature atrial beats

CLINICAL PRESENTATION

An older patient has pauses and intermittent conduction failure. One small atrial deflection arrives early, without a following QRS. The tracing contains more than one cause of a pause.

Is the missing QRS preceded by an early atrial impulse?



MIT-BIH 231 | source 142 s | 0-12 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise**1**

The marked V1 deflection is an early atrial impulse.

2

That premature impulse has no following QRS.

3

Other pauses in this recording reflect coexisting conduction disease.

Remember: Sometimes a pause starts with an extra beat.

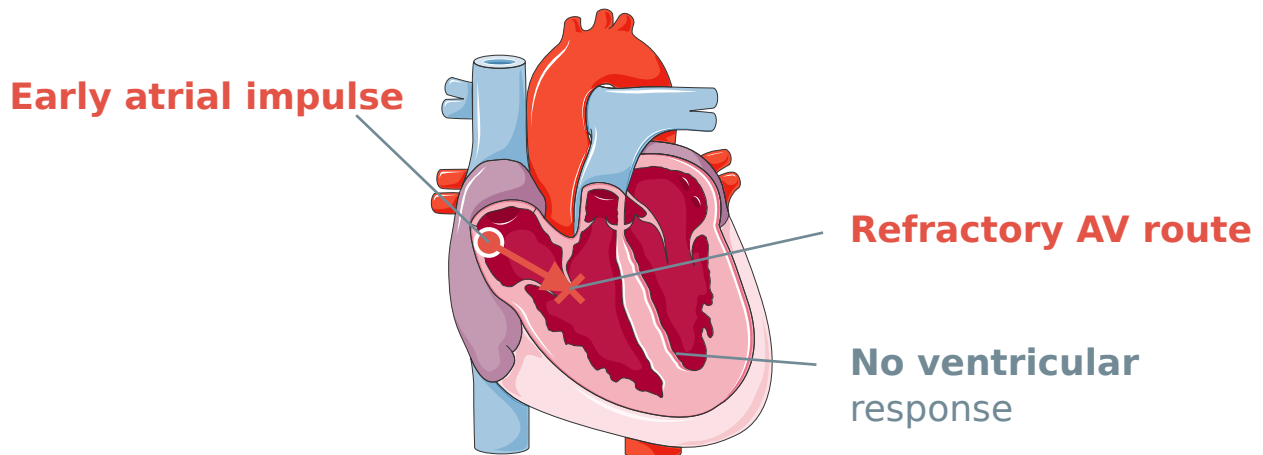
References: C08, C09

CASE 47 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

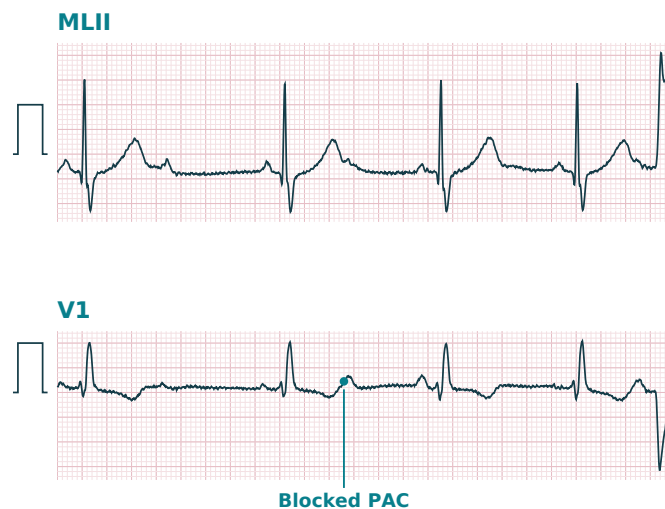
Blocked premature atrial complexes

An early atrial impulse can block



An early atrial impulse may meet refractory conduction tissue and produce no ventricular beat.

- An ectopic atrial focus fires before the next sinus impulse.
- The premature impulse meets refractory AV conduction tissue.
- Blocked transmission creates a ventricular pause despite extra atrial activity.



MIT-BIH 231 | source 142 s | 1-6 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

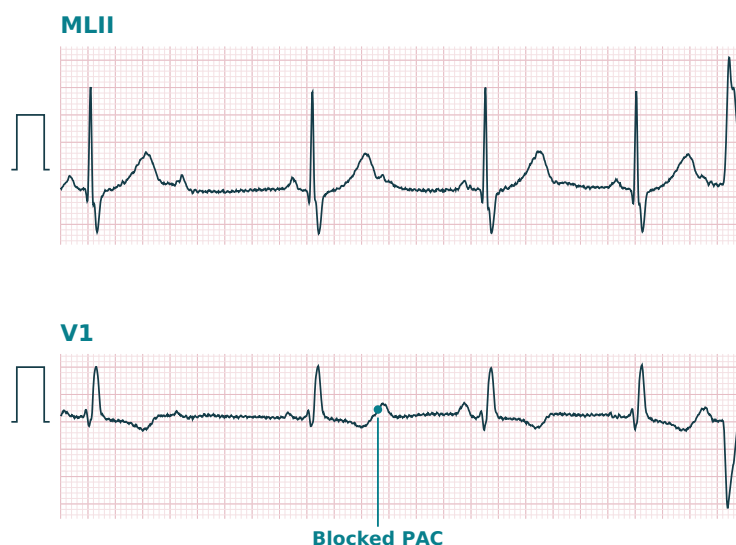
- Compare P-wave timing with the surrounding sinus rhythm.
- Correlate symptoms and obtain a longer tracing if the mechanism remains uncertain.
- Do not diagnose AV block from a pause alone.

Avoid. Blocked PACs and AV block can coexist; judge the marked P wave by timing.

CASE 47 | ANOTHER REAL ECG

Compare the patterns

Blocked premature atrial complexes



MIT-BIH 231 | source 142 s | 1-6 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

A conducted premature atrial beat



PTB-XL 05434 | 0-3.5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- An early narrow complex interrupts the usual rhythm.
- The premature atrial impulse successfully reaches the ventricles.

Remember. Sometimes a pause starts with an extra beat.

CASE 48 | ARRHYTHMIAS

Ventricular bigeminy and couplets

CLINICAL PRESENTATION

A patient feels repeated skipped beats. The monitor alternates ordinary complexes with early broad complexes. Another recording captures two consecutive ventricular ectopic beats.

Are the ectopic beats isolated, alternating or occurring together?



MIT-BIH 200 | source 0 s | 0-10 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise**1**

Ordinary and early altered complexes alternate.

2

The early complexes have a repeatable abnormal contour.

3

Read the whole sequence rather than counting pulse deficits.

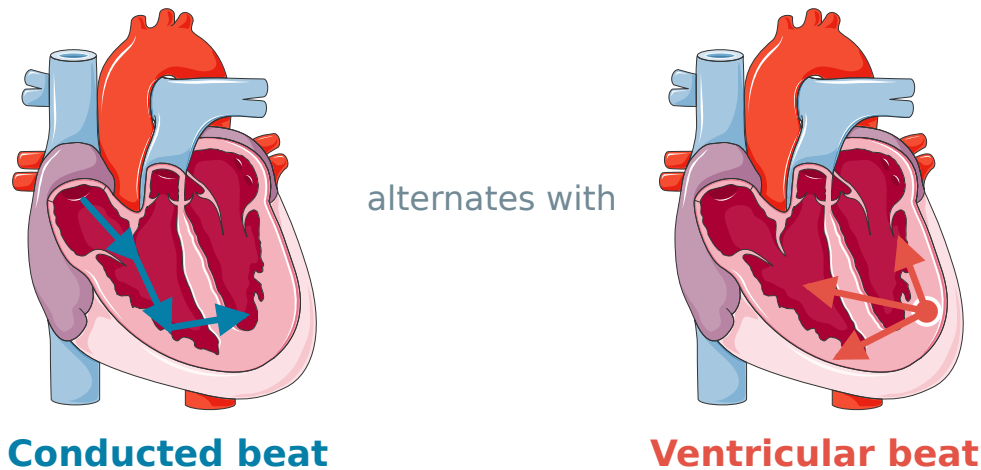
Remember: One, two, then a run: count consecutive ventricular beats.

CASE 48 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Ventricular bigeminy; compare with a ventricular couplet

Bigeminy alternates two activation routes



Alternating conducted and premature ventricular activations create bigeminy; two consecutive ventricular ectopic beats form a couplet.

- A ventricular focus interrupts ordinary conducted activation.
- Alternation after each conducted beat creates bigeminy.
- Two successive ectopic ventricular activations form a couplet.



MIT-BIH 200 | source 0 s | 0-10 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

- Assess symptoms, haemodynamic effect and possible reversible triggers.
- Use ambulatory monitoring to quantify ectopic burden when indicated.
- Consider structural assessment for frequent ectopy or concerning clinical features.

Avoid. Bigeminy describes a sequence; it does not establish the cause or risk.

CASE 48 | ANOTHER REAL ECG

Compare the patterns

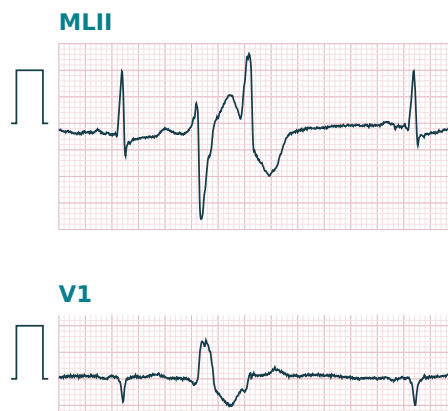
Ventricular bigeminy; compare with a ventricular couplet



MIT-BIH 200 | source 0 s | 0-10 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Two consecutive ventricular premature beats form a couplet



MIT-BIH 200 | source 1574 s | 2.2-5.2 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

- Read the beats immediately before and after the pair.
- Consecutive ectopy differs from alternating bigeminy.

Remember. One, two, then a run: count consecutive ventricular beats.

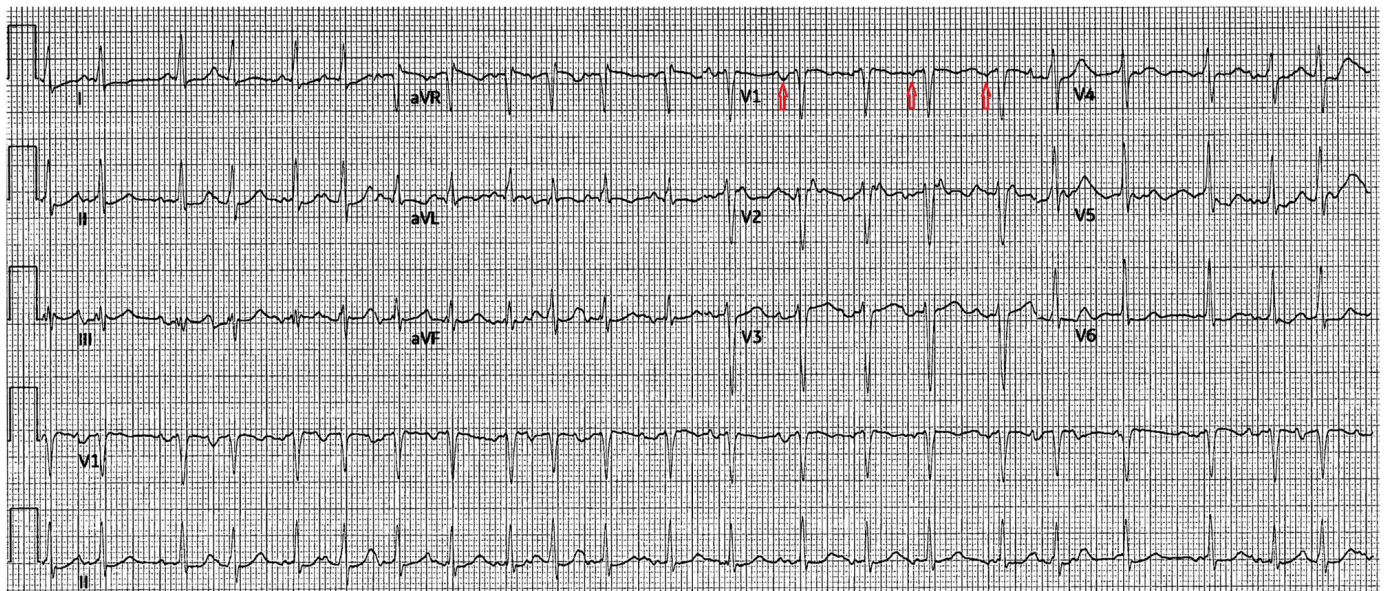
CASE 49 | ARRHYTHMIAS

Multifocal atrial tachycardia

PUBLISHED CASE

A 69-year-old woman with COPD is admitted with hypoxaemia and worsening breathlessness. An irregular tachycardia is labelled AF by the machine, but discrete P waves remain visible.

Are there several distinct P-wave shapes in the same lead?



Harvey · Figure 1 · CC BY 4.0

Three findings to recognise**1**

Discrete P waves have differing shapes, especially in V1.

2

Atrial and ventricular intervals vary.

3

Look for at least three P morphologies and intervening isoelectric baseline.

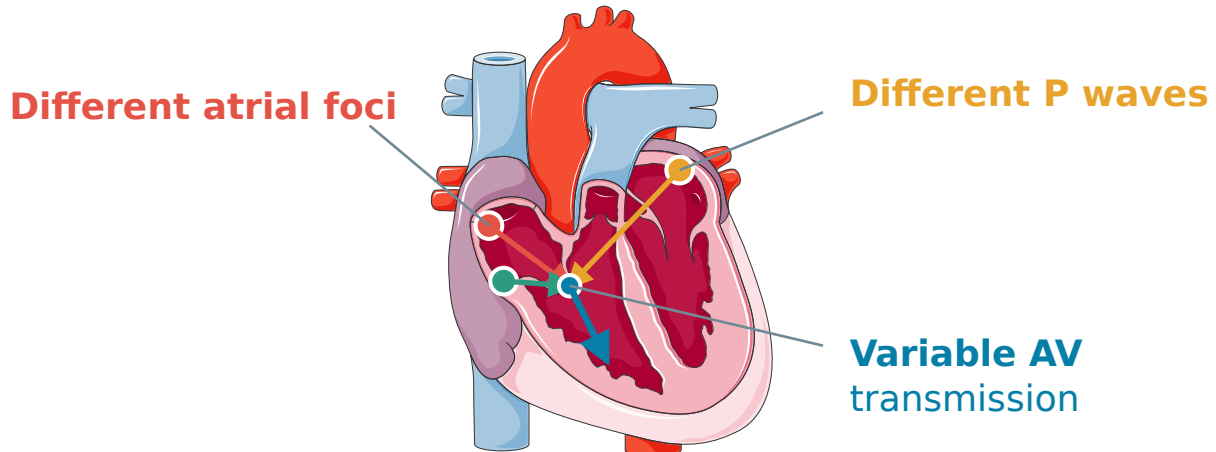
Remember: Irregular does not mean AF: look for distinct P waves.

CASE 49 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Multifocal atrial tachycardia

Multiple atrial origins change the P wave



Changing atrial origins alter P-wave morphology; AV conduction determines the ventricular response.

- Several atrial sites can initiate impulses.
- Changing activation direction produces different P-wave shapes.
- Illness and atrial stress can sustain the irregular tachycardia.



V1 detail; original arrows identify differing P waves. Harvey · Figure 1 · CC BY 4.0

In practice

- Treat hypoxaemia and the underlying pulmonary or systemic illness.
- Review electrolytes and medicines that increase atrial irritability.
- Choose rate treatment according to ventricular function and respiratory context.

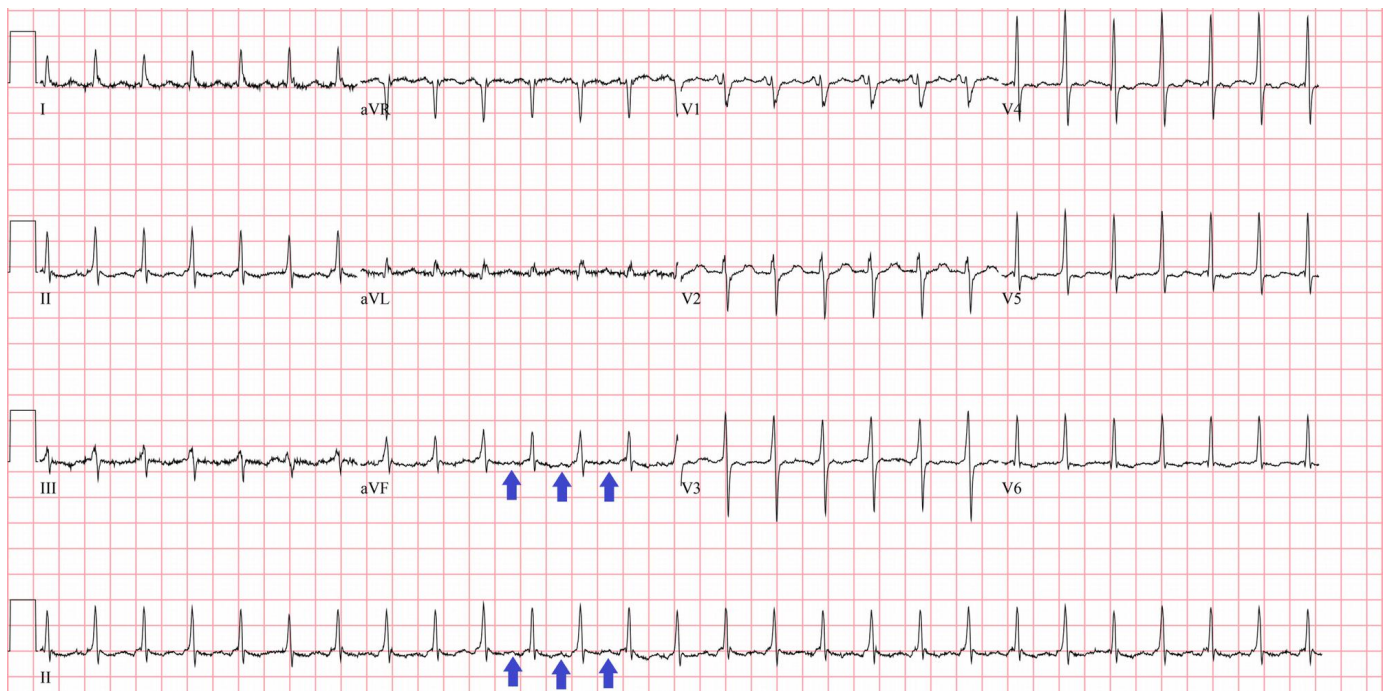
Avoid. MAT alone does not create the AF indication for anticoagulation.

CASE 50 | ARRHYTHMIAS

Atrial tachycardia through AV block

PUBLISHED CASE

A 69-year-old woman develops two hours of persistent palpitations. Regular P waves accompany narrow-complex tachycardia. During monitored adenosine testing, atrial activity continues despite transient AV block.

Does the atrial rhythm depend on conduction through the AV node?

Stahl et al. · Figure 1 · CC BY 4.0

Three findings to recognise**1**

The original tracing shows regular narrow-complex tachycardia.

2

P-wave morphology differs from the previous sinus tracing.

3

Regular atrial activity continues while ventricular conduction is blocked.

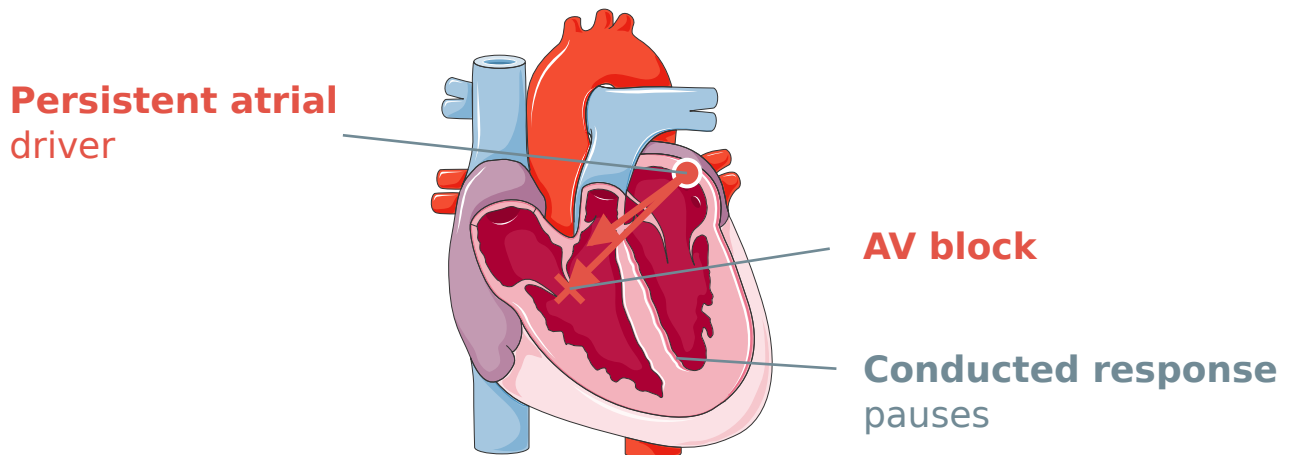
Remember: Watch the atria when the ventricles pause.

CASE 50 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

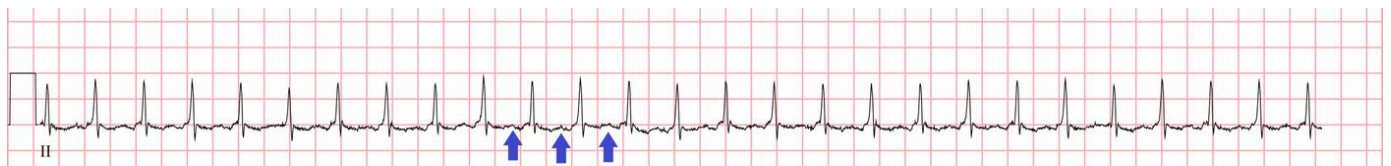
Atrial tachycardia revealed by transient AV block

The atrial rhythm can continue through AV block



Atrial tachycardia may continue while transient AV block interrupts ventricular transmission.

- Atrial tachycardia is driven within the atria.
- The AV node transmits impulses but need not sustain the atrial rhythm.
- Transient AV block can expose continuing atrial activity.



Initial ECG: lead II rhythm strip. Stahl et al. · Figure 1 · CC BY 4.0

In practice

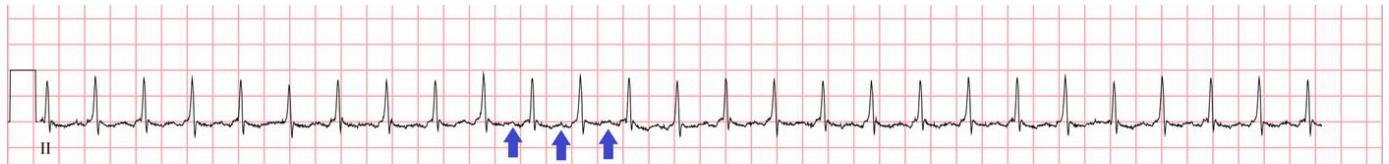
- Assess stability and reversible causes before pursuing the exact mechanism.
- Record the response to monitored treatment, including atrial activity during AV block.
- Persistent symptomatic tachycardia warrants specialist assessment and consideration of ablation.

Avoid. Failure to terminate after adenosine is not, by itself, proof of focal AT.

CASE 50 | ANOTHER REAL ECG

Compare the patterns

Atrial tachycardia revealed by transient AV block



Initial ECG: lead II rhythm strip. Stahl et al. · Figure 1 · CC BY 4.0

During adenosine: regular atrial activity continues through AV block



During adenosine: V1 detail. Stahl et al. · Figure 4 · CC BY 4.0

- The blue box highlights continuing atrial deflections.
- The atrial driver persists when ventricular transmission pauses.

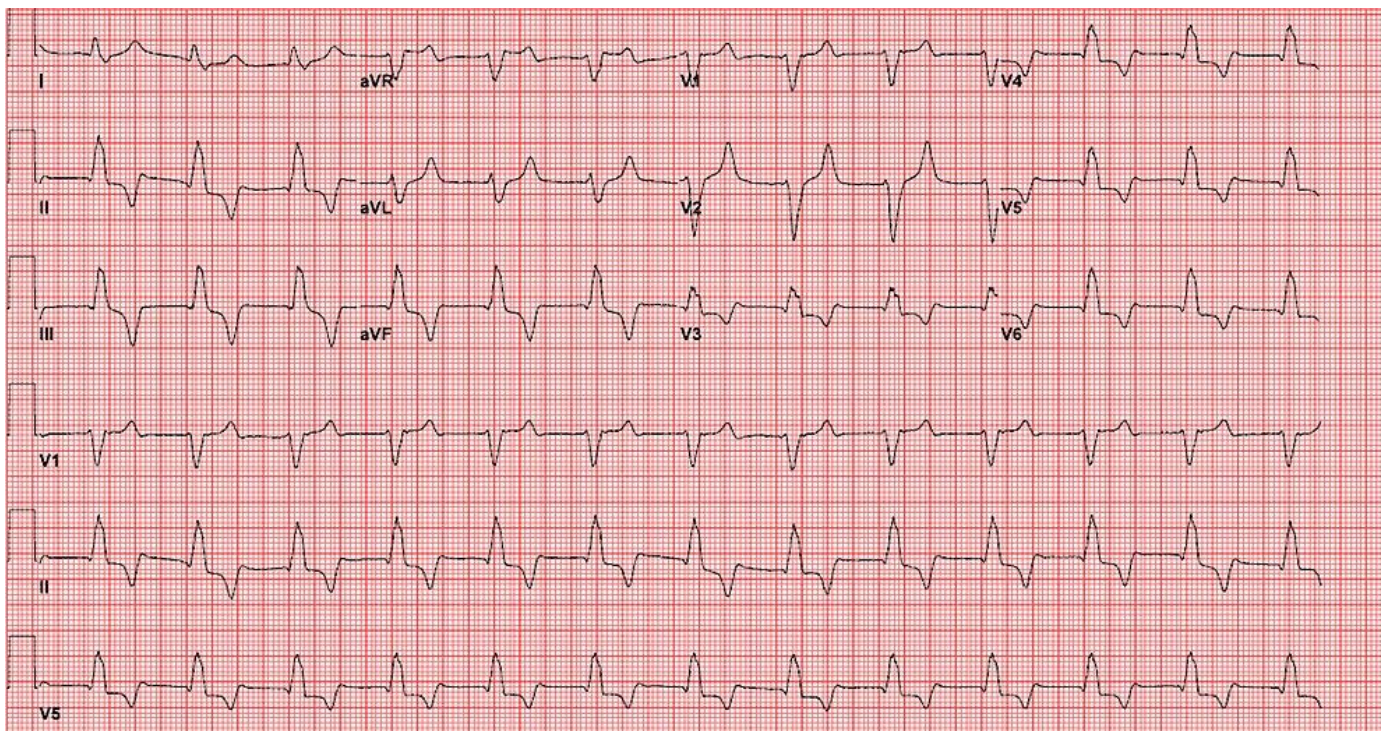
Remember. Watch the atria when the ventricles pause.

CASE 51 | ARRHYTHMIAS

Accelerated idioventricular rhythm

PUBLISHED CASE

A 73-year-old woman regains circulation after cardiac arrest. She has anuric renal failure and hyperkalaemia. The post-resuscitation ECG shows a regular broad-complex rhythm at 77/min.

Why can a ventricular rhythm occur without a very fast rate?

Koo et al. · Figure 2 · CC BY 4.0

Three findings to recognise**1**

Broad ventricular complexes occur regularly at 77/min.

2

No consistent preceding P-wave sequence is discernible.

3

Tall T waves accompany the metabolic disturbance in this published case.

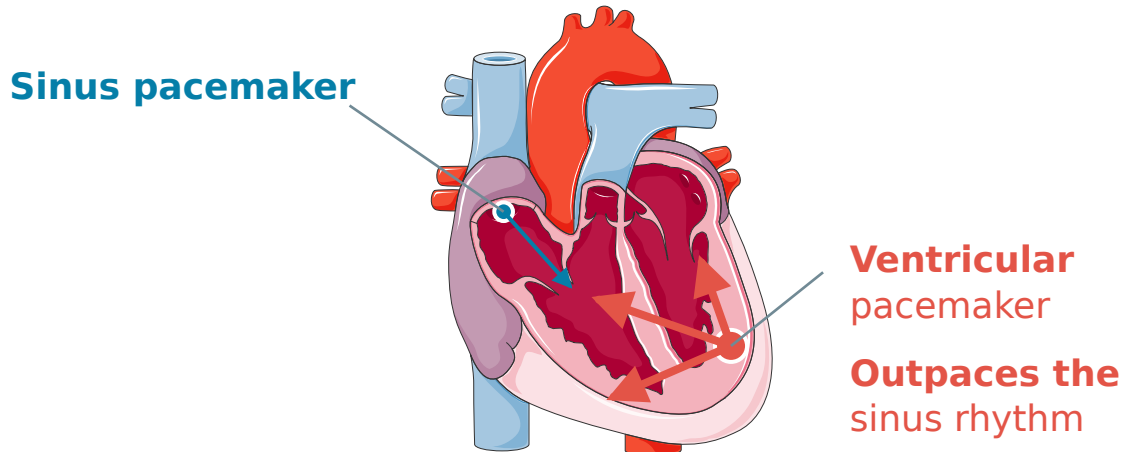
Remember: A ventricular rhythm can be slow enough to resemble an escape rhythm.

CASE 51 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Accelerated idioventricular rhythm

A ventricular pacemaker can take the lead



Enhanced ventricular automaticity can overtake sinus timing, producing a regular broad-complex rhythm.

- An ectopic ventricular pacemaker can outpace the sinus node.
- Ventricular-origin activation produces a broad QRS.
- Metabolic disturbance, drugs or reperfusion can alter ventricular automaticity.



V1 and II rhythm-strip detail. Koo et al. · Figure 2 · CC BY 4.0

In practice

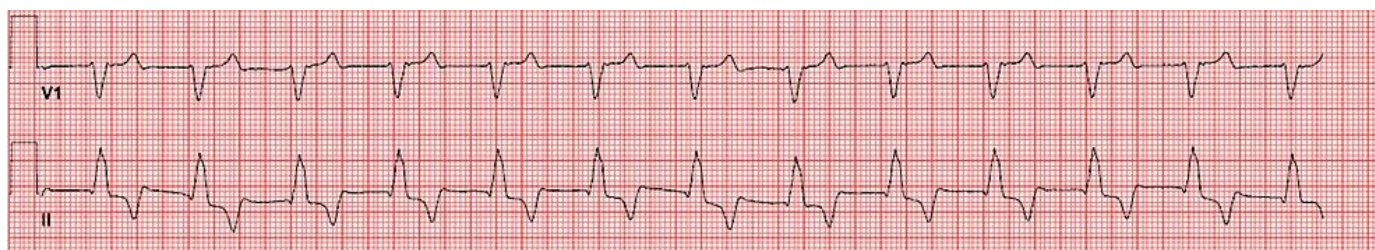
- Assess circulation and identify the clinical trigger.
- Correct urgent metabolic abnormalities and review toxic exposures.
- Do not suppress a stable AIVR merely because its complexes are broad.

Avoid. AIVR is not proof of successful coronary reperfusion.

CASE 51 | ANOTHER REAL ECG

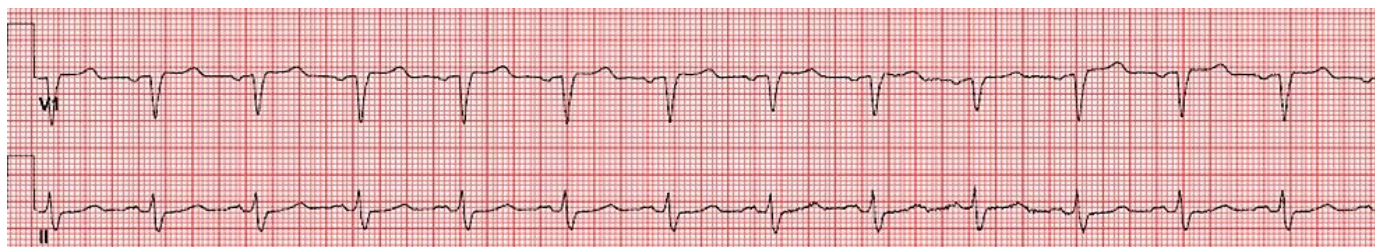
Compare the patterns

Accelerated idioventricular rhythm



V1 and II rhythm-strip detail. Koo et al. · Figure 2 · CC BY 4.0

After dialysis: sinus rhythm and resolution of peaked T waves



After dialysis: rhythm-strip detail. Koo et al. · Figure 3 · CC BY 4.0

- P waves now precede the ventricular complexes.
- Compare the rhythm and T waves with the first ECG.

Remember. A ventricular rhythm can be slow enough to resemble an escape rhythm.

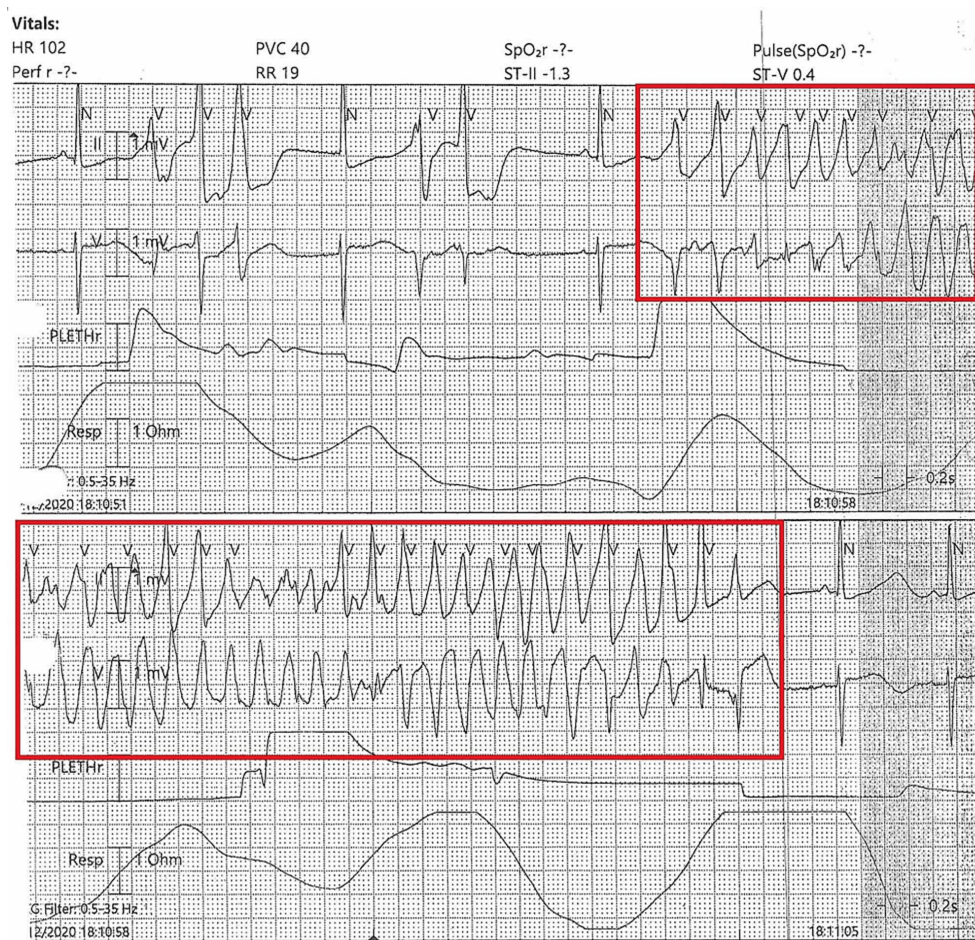
CASE 52 | ARRHYTHMIAS

Torsades de pointes

PUBLISHED CASE

A 57-year-old woman has recurrent palpitations after abdominal surgery. Telemetry shows polymorphic ventricular runs; subsequent sinus ECGs confirm persistent QT prolongation.

What recovery abnormality distinguishes torsades from other polymorphic VT?



Kommineni et al. · Figure 1 · CC BY 4.0

Three findings to recognise

1

QRS shape and amplitude change during the ventricular run.

2

The displayed run terminates spontaneously.

3

The accompanying sinus ECG demonstrates prolonged ventricular recovery.

Remember: Read the QT before naming the polymorphic rhythm.

CASE 52 | UNDERSTAND AND APPLY

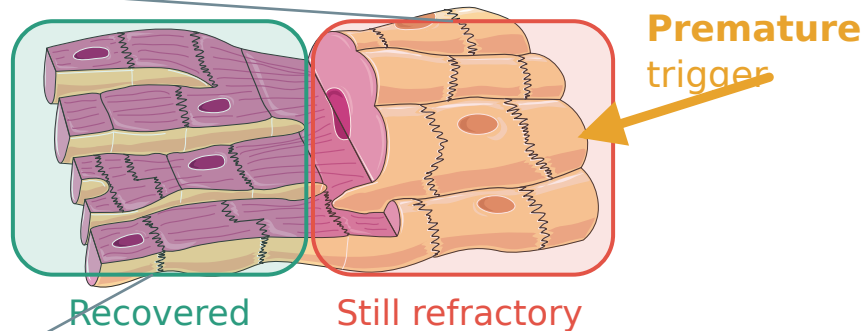
From anatomy to clinical meaning

Torsades de pointes in a prolonged-QT setting

Uneven recovery makes a trigger more dangerous

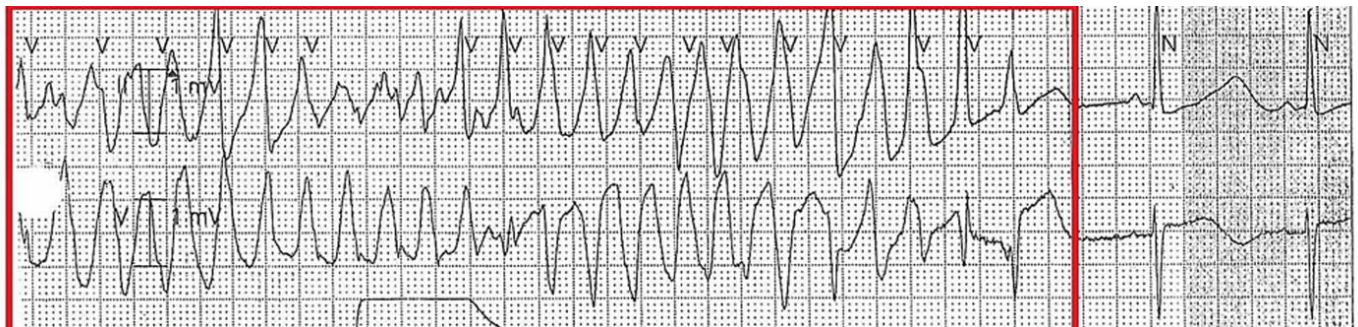
Delayed recovery

Uneven readiness



Delayed, uneven ventricular recovery can permit premature triggers and unstable activation in torsades.

- Delayed recovery increases susceptibility to triggered ventricular beats.
- Uneven recovery allows unstable ventricular activation patterns.
- A premature trigger can initiate polymorphic VT.



Continuation: polymorphic run and spontaneous termination. Kommineni et al. · Figure 1 · CC BY 4.0

In practice

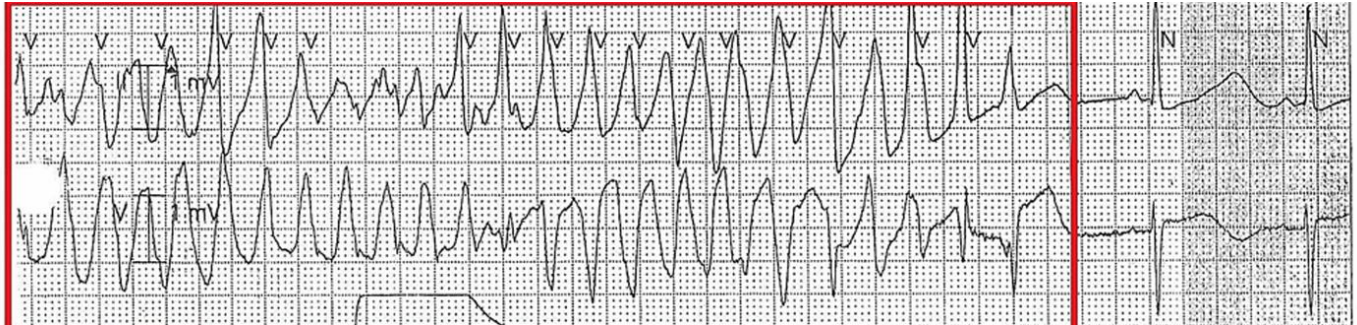
- Sustained polymorphic VT requires immediate unsynchronised shock.
- For torsades, give protocol-based magnesium and correct potassium and other contributors.
- Stop QT-prolonging triggers; seek expert treatment for recurrent pause-dependent episodes.

Avoid. A twisting appearance alone does not establish torsades without QT context.

CASE 52 | ANOTHER REAL ECG

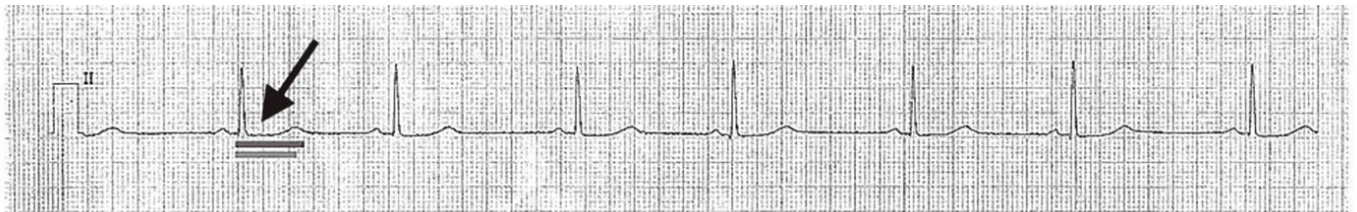
Compare the patterns

Torsades de pointes in a prolonged-QT setting



Continuation: polymorphic run and spontaneous termination. Kommineni et al. · Figure 1 · CC BY 4.0

Later sinus ECG: the prolonged QT context



Later sinus rhythm: lead II and original QT annotation. Kommineni et al. · Figure 3 · CC BY 4.0

- Interpret polymorphic VT alongside repolarisation outside the run.
- The publication reports persistent QT prolongation.

Remember. Read the QT before naming the polymorphic rhythm.

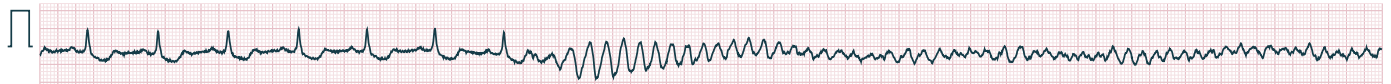
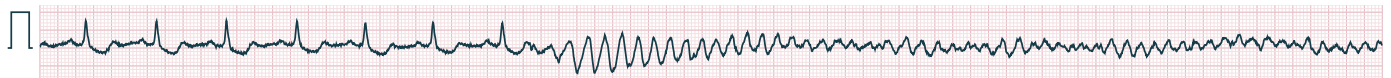
CASE 53 | ARRHYTHMIAS

Ventricular fibrillation

CLINICAL PRESENTATION

A monitored patient suddenly collapses and has no palpable pulse. Organised ventricular complexes give way to an irregular waveform in both recorded channels.

Has coordinated ventricular activation been lost?

ECG 1**ECG 2**

VFDB 426 | 10:40 | 0-15 s, simultaneous | ECG 1, ECG 2
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

VFDB | Albrecht, Moody & Mark | ODC Attribution 1.0

Three findings to recognise**1**

Organised complexes are followed by a rapid irregular waveform.

2

No stable repeating P-QRS-T sequence remains.

3

Both channels show the same transition.

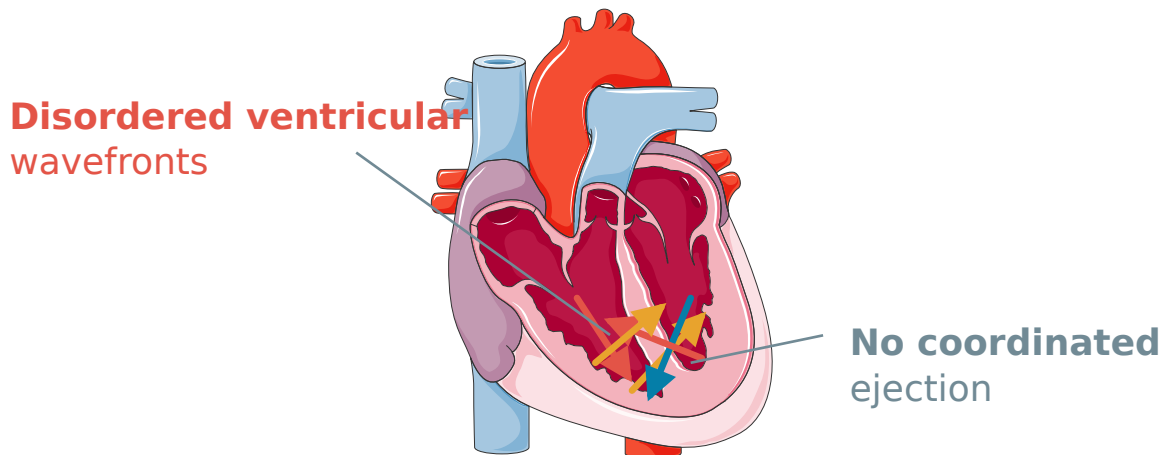
Remember: Treat the patient and the arrest rhythm together.

CASE 53 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Ventricular fibrillation

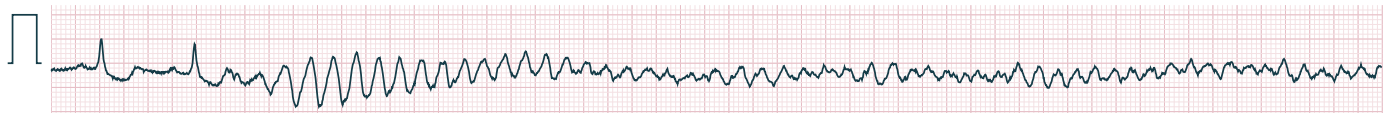
Ventricular fibrillation loses coordinated activation



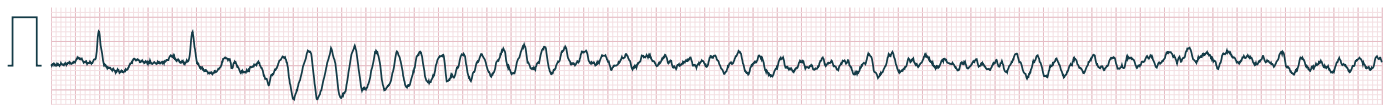
Disordered ventricular activation prevents effective coordinated contraction; cardiac arrest requires immediate CPR and defibrillation.

- Disordered wavefronts activate different ventricular regions at different times.
- Effective coordinated ventricular contraction is lost.
- Defibrillation aims to stop the disordered electrical activity.

ECG 1



ECG 2



VFDB 426 | 10:40 | 4-15 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

VFDB | Albrecht, Moody & Mark | ODC Attribution 1.0

In practice

- Recognise cardiac arrest, summon help and start high-quality CPR.
- Deliver prompt unsynchronised defibrillation according to the resuscitation protocol.
- Resume CPR immediately; address reversible causes during organised resuscitation.

Avoid. Do not delay CPR or defibrillation while attempting detailed waveform analysis.

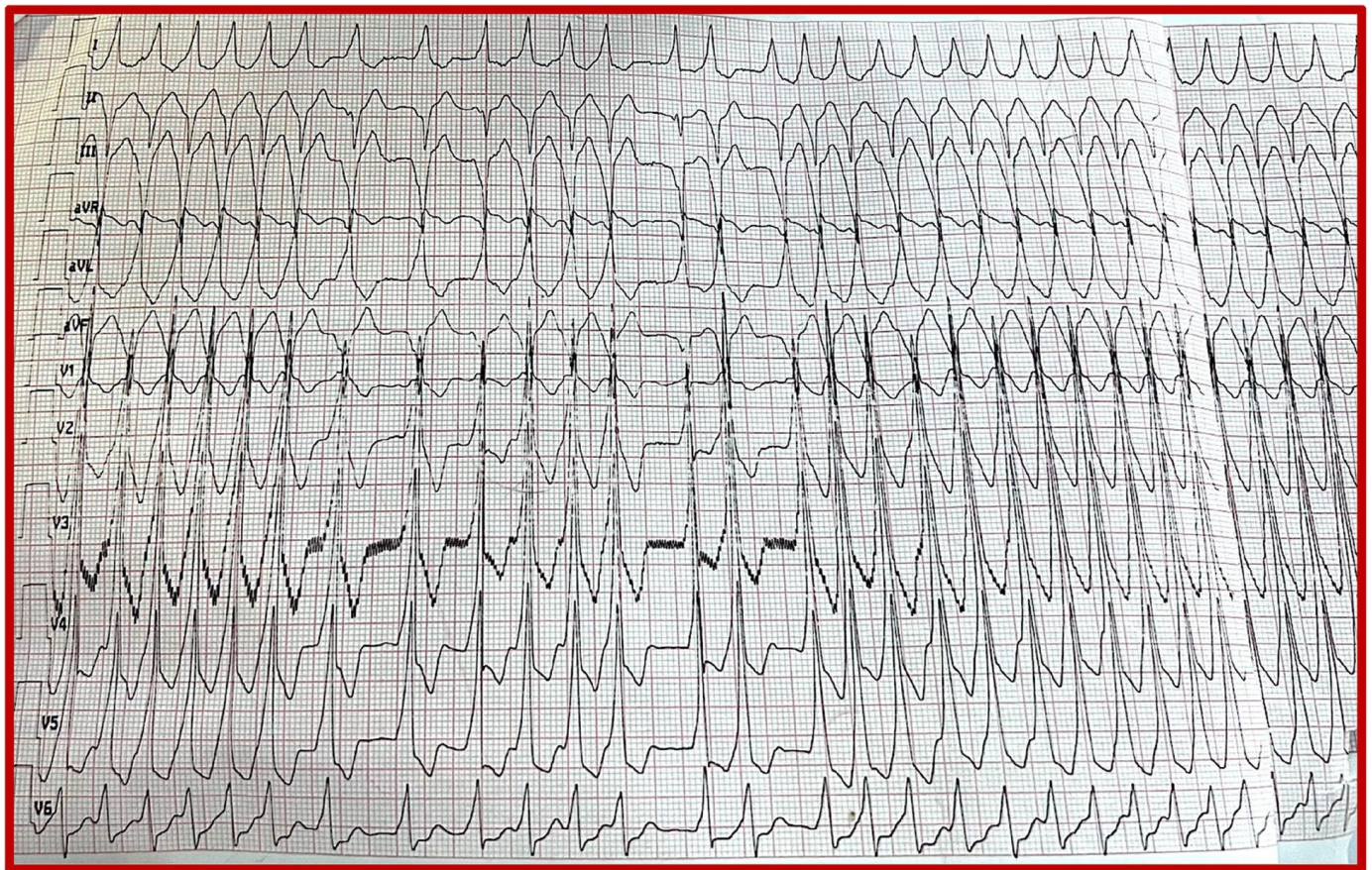
CASE 54 | ARRHYTHMIAS

Pre-excited atrial fibrillation

PUBLISHED CASE

A 72-year-old man develops abrupt palpitations, confusion and cold extremities. Blood pressure is 80/60 mmHg. The ECG shows rapid irregular broad complexes whose shape varies.

Could atrial impulses be bypassing the usual AV nodal filter?



Diallo et al. · Figure 1 · CC BY 4.0

Three findings to recognise**1**

The ventricular rhythm is very rapid and irregular.

2

QRS width and shape vary between beats.

3

After cardioversion, sinus rhythm reveals ventricular pre-excitation.

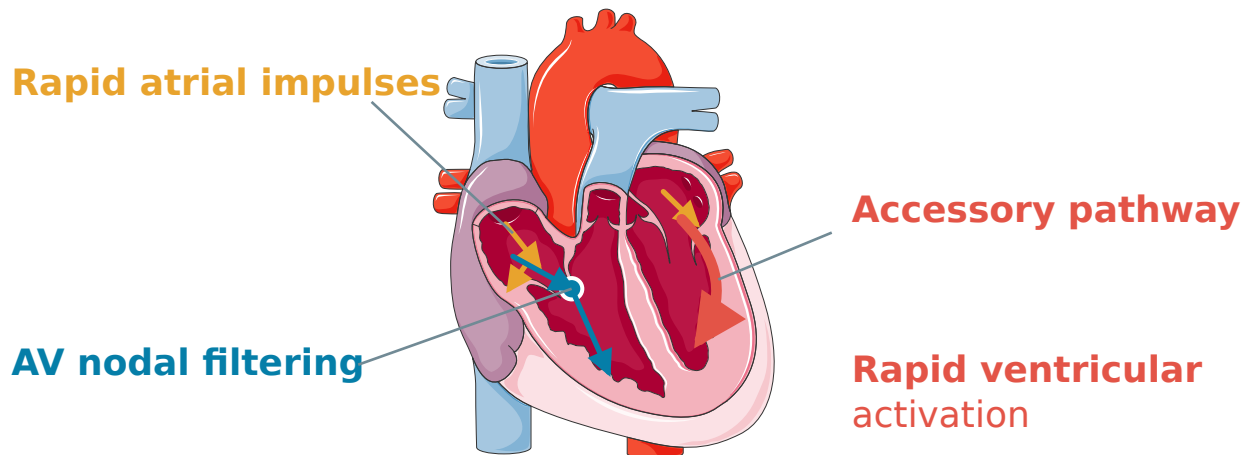
Remember: Irregular, rapid and broad: think beyond ordinary AF.

CASE 54 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

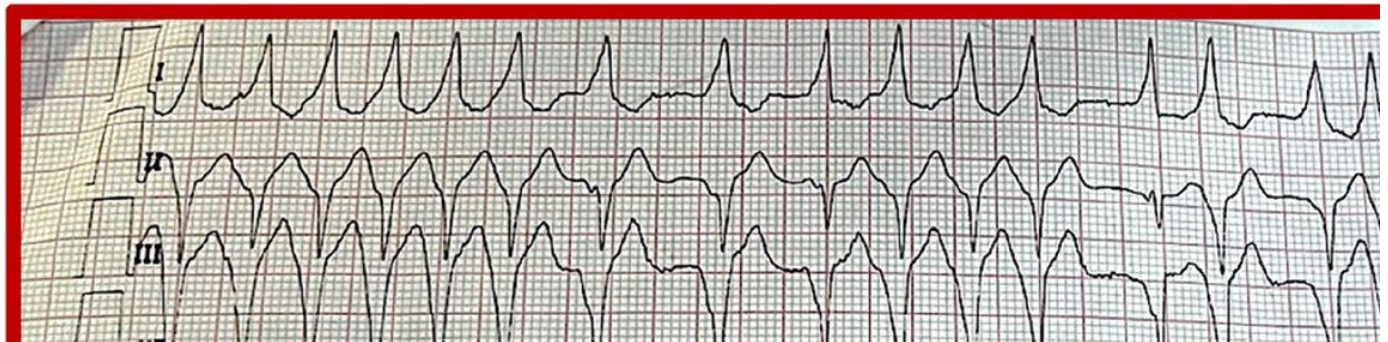
Pre-excited atrial fibrillation

An accessory pathway bypasses the AV filter



During AF, an accessory pathway can transmit atrial impulses rapidly to the ventricles.

- Fibrillating atria generate rapid, disordered impulses.
- An accessory pathway can conduct them directly to ventricular tissue.
- Rapid ventricular activation may deteriorate into ventricular fibrillation.



Limb-lead detail of the presenting tachycardia. Diallo et al. · Figure 1 · CC BY 4.0

In practice

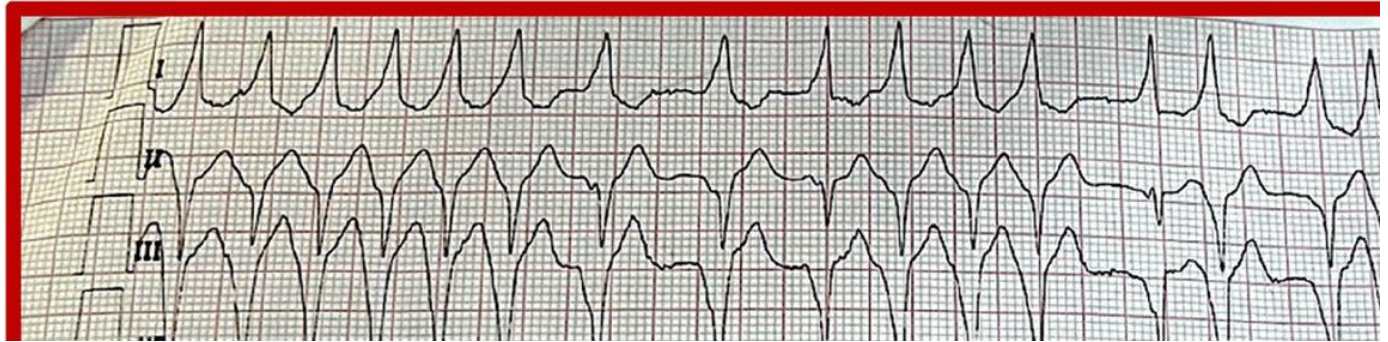
- If unstable with a pulse, perform urgent synchronised electrical cardioversion.
- Avoid AV nodal blockers, including intravenous amiodarone.
- Use an appropriate pre-excited-AF pathway with specialist support; arrange definitive pathway assessment.

Avoid. Do not apply the ordinary narrow-SVT treatment sequence to irregular broad tachycardia.

CASE 54 | ANOTHER REAL ECG

Compare the patterns

Pre-excited atrial fibrillation



Limb-lead detail of the presenting tachycardia. Diallo et al. · Figure 1 · CC BY 4.0

After cardioversion: pre-excitation becomes easier to recognise



After cardioversion: V1-V2 detail. Diallo et al. · Figure 2 · CC BY 4.0

- Sinus rhythm reveals a short PR and delta wave.
- The underlying pathway explains the earlier rapid ventricular response.

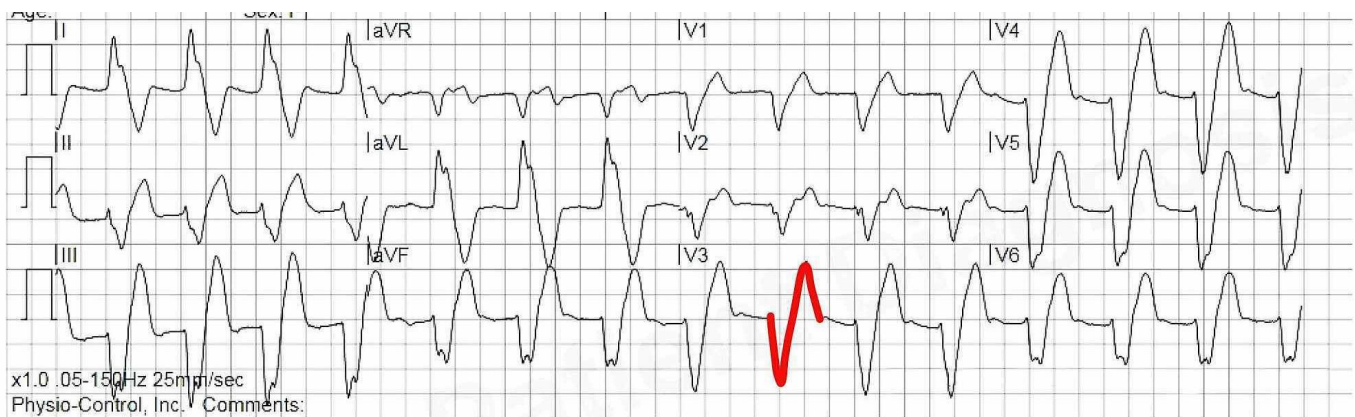
Remember. Irregular, rapid and broad: think beyond ordinary AF.

CASE 55 | METABOLIC AND TOXIC EFFECTS

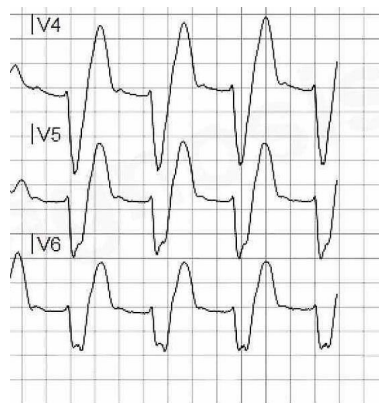
Hyperkalaemia: electrical danger may escalate

PUBLISHED CASE

A 62-year-old woman with chronic kidney disease develops weakness while taking potassium-raising medicines. Her ECG has severely broadened, merging complexes.

What immediately threatens this patient?

Amr Elmoheen et al. | 2020 | CC BY 4.0 (publisher PDF)



Recorded V4-V6 detail: broad merging ventricular complexes. Amr Elmoheen et al. | 2020 | CC BY 4.0 (publisher PDF)

Three findings to recognise**1**

Ventricular complexes are markedly broad.

2

QRS and T contours become difficult to separate.

3

Severe electrical disturbance requires immediate clinical action.

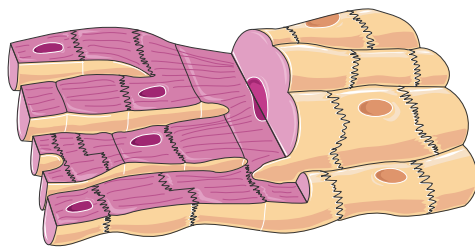
Remember: Protect the heart, shift potassium, remove potassium.

CASE 55 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Severe hyperkalaemia with dangerous conduction disturbance

Ventricular muscle cells



High extracellular K⁺



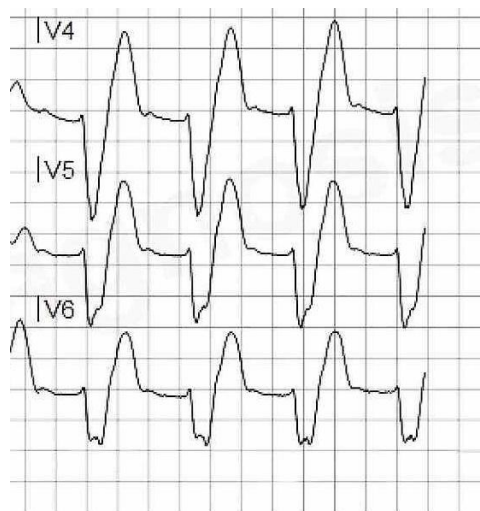
Less negative resting voltage

Fewer available Na⁺ channels

Slower conduction → broad QRS

Extracellular potassium changes membrane behaviour and can destabilise ventricular electrical activity.

- High extracellular potassium alters the resting membrane potential.
- Sodium-channel availability falls as depolarisation becomes sustained.
- Ventricular conduction slows and dangerous arrhythmias can follow.



Recorded V4–V6 detail: broad merging ventricular complexes. Amr Elmoheen et al. | 2020 | CC BY 4.0 (publisher PDF)

In practice

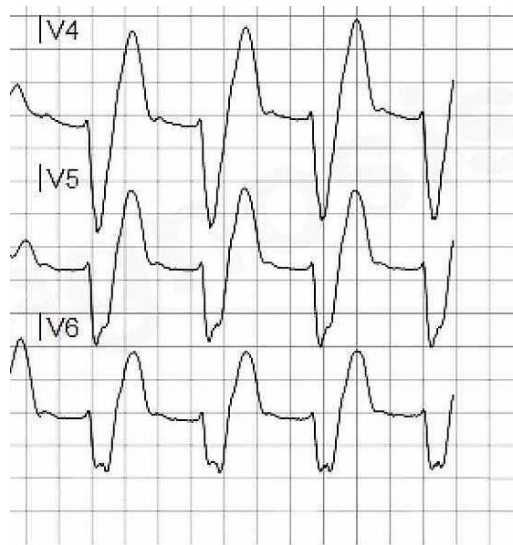
- Use the emergency hyperkalaemia pathway and continuous cardiac monitoring.
- Give intravenous calcium for toxic ECG changes according to local protocol.
- Shift potassium into cells, remove potassium and recheck ECG and laboratory values.

Avoid. A reassuring ECG does not exclude dangerous hyperkalaemia; calcium does not remove potassium.

CASE 55 | ANOTHER REAL ECG

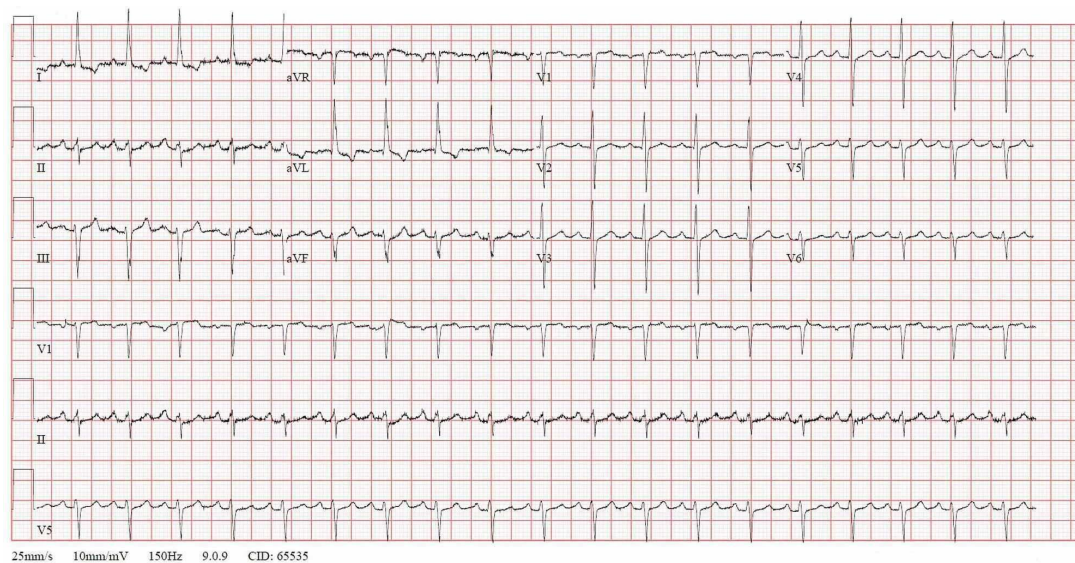
Compare the patterns

Severe hyperkalaemia with dangerous conduction disturbance



Recorded V4-V6 detail: broad merging ventricular complexes. Amr Elmoheen et al. | 2020 | CC BY 4.0 (publisher PDF)

After calcium: electrical improvement before potassium removal



Elmoheen et al. (2020), Figure 2; CC BY 4.0.

- The QRS narrows markedly.
- Potassium remained high; definitive removal was still required.

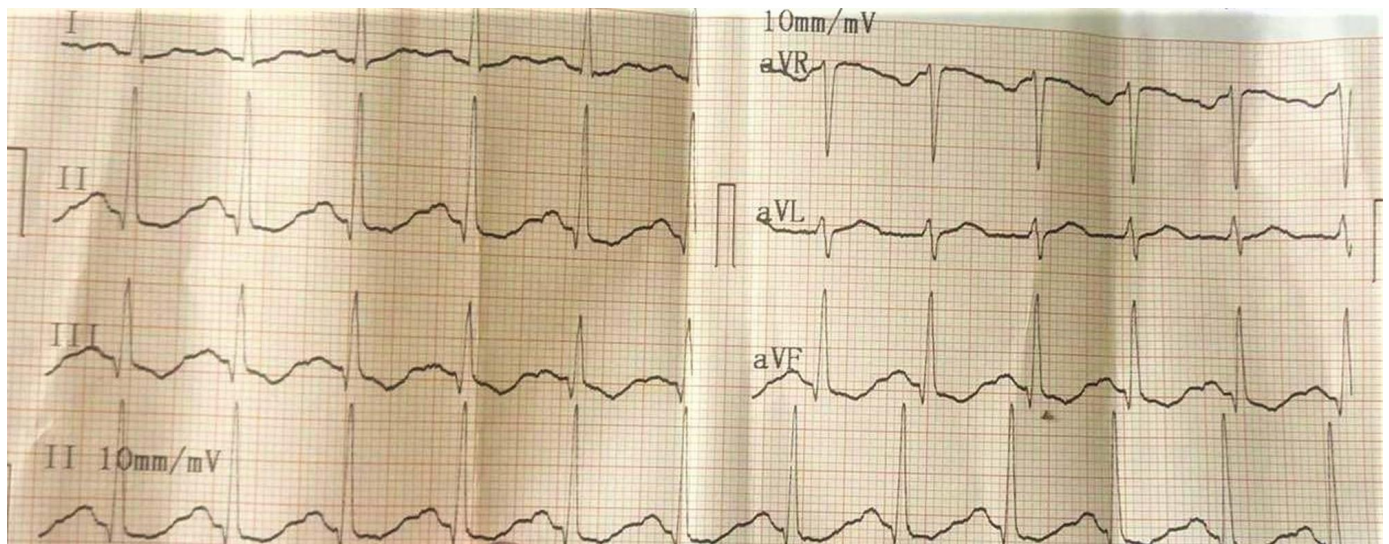
Remember. Protect the heart, shift potassium, remove potassium.

CASE 56 | METABOLIC AND TOXIC EFFECTS

Hypokalaemia: separate the T wave from the U

PUBLISHED CASE

A 38-year-old woman is treated for supraventricular tachycardia. After conversion, her ECG has small T waves and prominent U waves; potassium is 2.7 mmol/L.

Is the late deflection part of the T wave?

Thu et al. (2022), Figure 5; CC BY 4.0.

Three findings to recognise**1**

T waves are small and ST segments depressed.

2

Prominent U waves follow the T waves.

3

T-U fusion can create apparent QT prolongation.

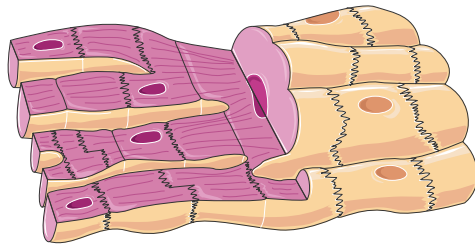
Remember: Find the T-wave end before measuring QT.

CASE 56 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Hypokalaemia with ST depression and prominent U waves

Ventricular muscle cells



Low extracellular K⁺

+

+

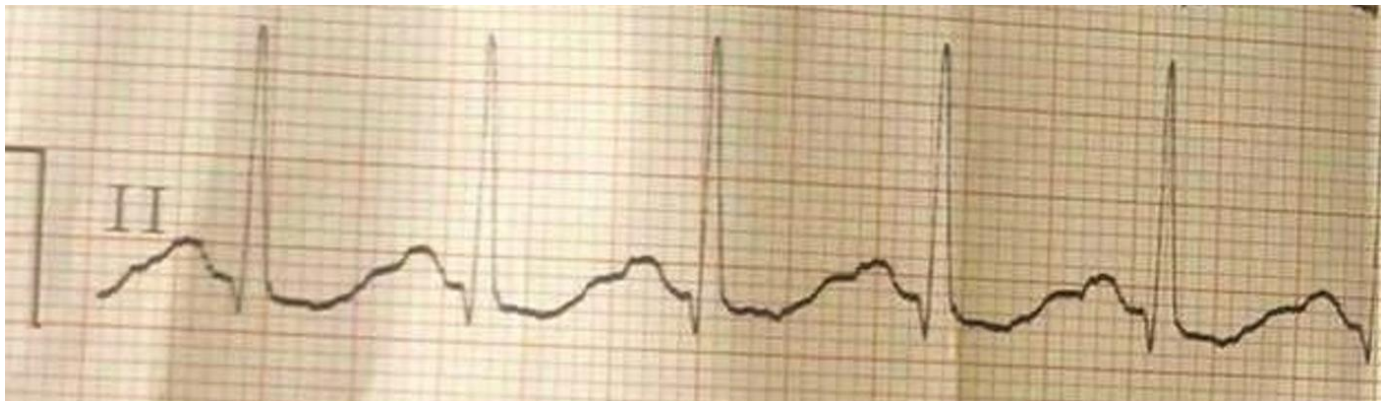
**Repolarisation becomes uneven**

Delayed recovery; triggered activity

T-U changes; arrhythmia risk

Extracellular potassium changes membrane behaviour and can destabilise ventricular electrical activity.

- Low extracellular potassium disrupts normal ventricular repolarisation.
- Repolarisation becomes prolonged and less uniform.
- Triggered activity and dangerous ventricular arrhythmias become more likely.



Small T waves and prominent U waves in the published limb-lead recording. Thu et al. (2022), Figure 5; CC BY 4.0.

In practice

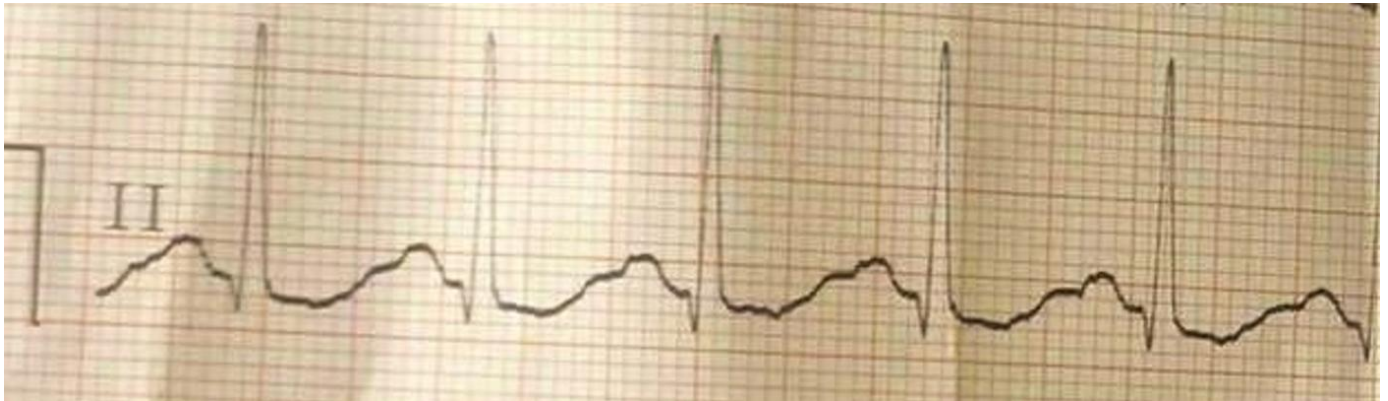
- Measure potassium, magnesium and renal function; assess ongoing losses.
- Replace potassium using severity-based local protocols and appropriate monitoring.
- Review QT-prolonging medicines; correct magnesium deficiency when present.

Avoid. This ECG followed amiodarone; medication effects may also influence the measured QT interval.

CASE 56 | ANOTHER REAL ECG

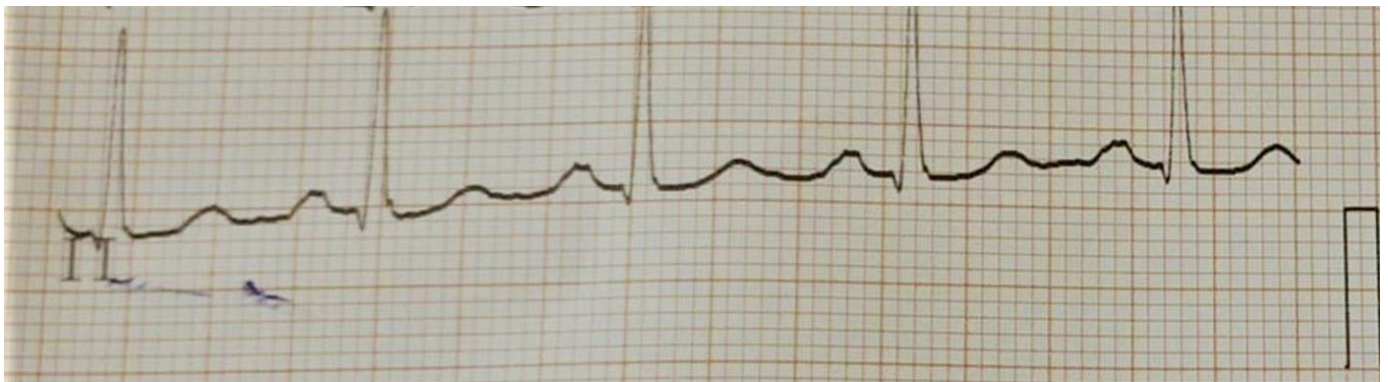
Compare the patterns

Hypokalaemia with ST depression and prominent U waves



Small T waves and prominent U waves in the published limb-lead recording. Thu et al. (2022), Figure 5; CC BY 4.0.

After potassium replacement in the same patient



Limb leads after potassium replacement: ST-T and U-wave changes improve. Thu et al. (2022), Figure 6; CC BY 4.0.

- U-wave prominence reduces.
- ST-T changes improve.

Remember. Find the T-wave end before measuring QT.

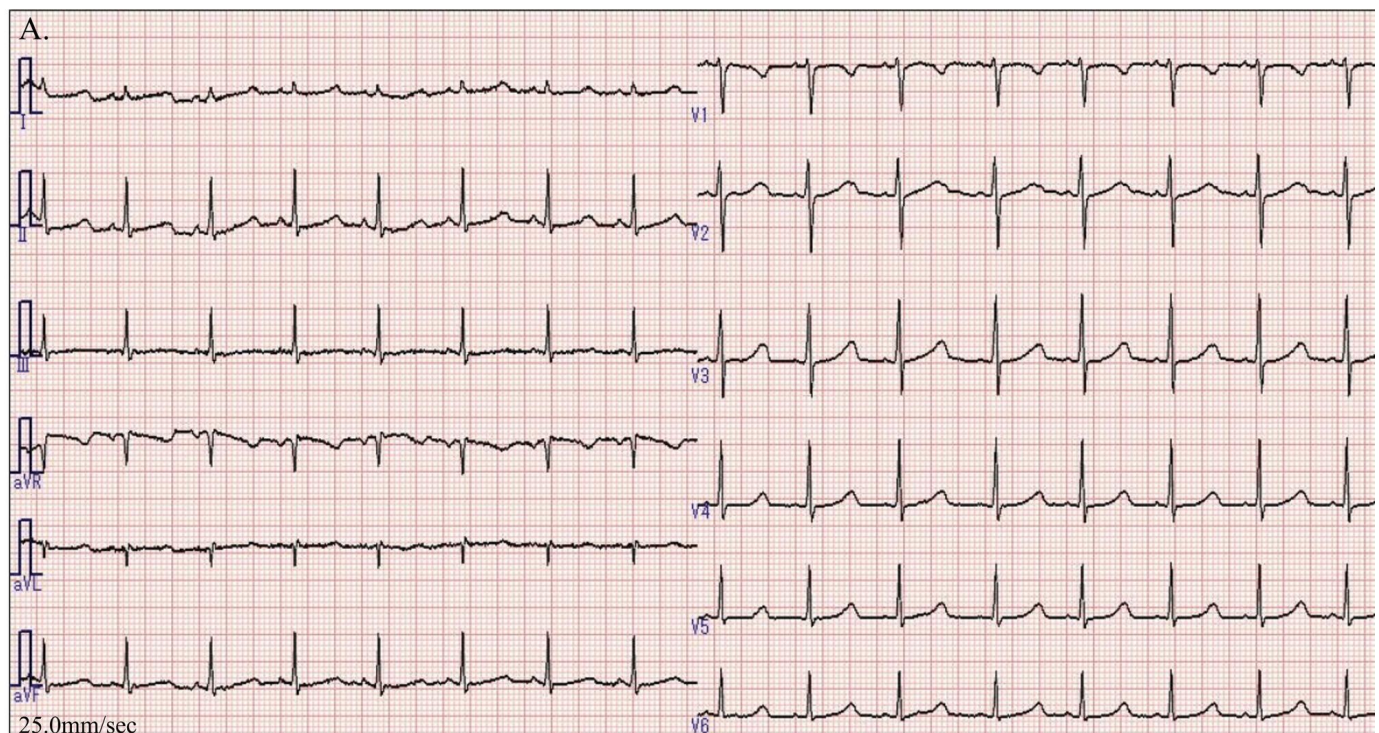
CASE 57 | METABOLIC AND TOXIC EFFECTS

Low calcium: a prolonged recovery phase

PUBLISHED CASE

A 13-year-old girl briefly loses consciousness. Tingling follows. Corrected calcium is 1.40 mmol/L; the ECG shows a reported QTc of 524 ms.

Which part of ventricular recovery is prolonged?



Miyao M et al. · Figure 1A · CC BY 4.0

Three findings to recognise

1

The ST segment is prolonged.

2

QRS complexes remain narrow.

3

After calcium replacement, the reported QTc falls to 465 ms.

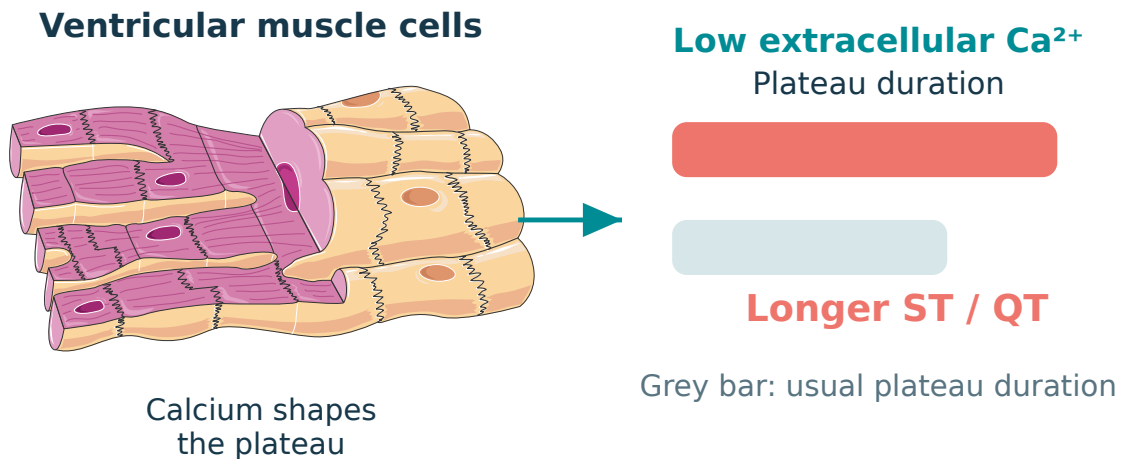
Remember: Low calcium can stretch the ST.

CASE 57 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Hypocalcaemia-associated QT prolongation

Calcium changes the duration of ventricular recovery



Calcium changes the ventricular plateau and the ST portion of the QT interval.

- Low extracellular calcium alters ventricular action-potential timing.
- The plateau lengthens, delaying completion of repolarisation.
- The ECG usually shows a longer ST segment and QT.



Miyao M et al. · Figure 1A · CC BY 4.0

In practice

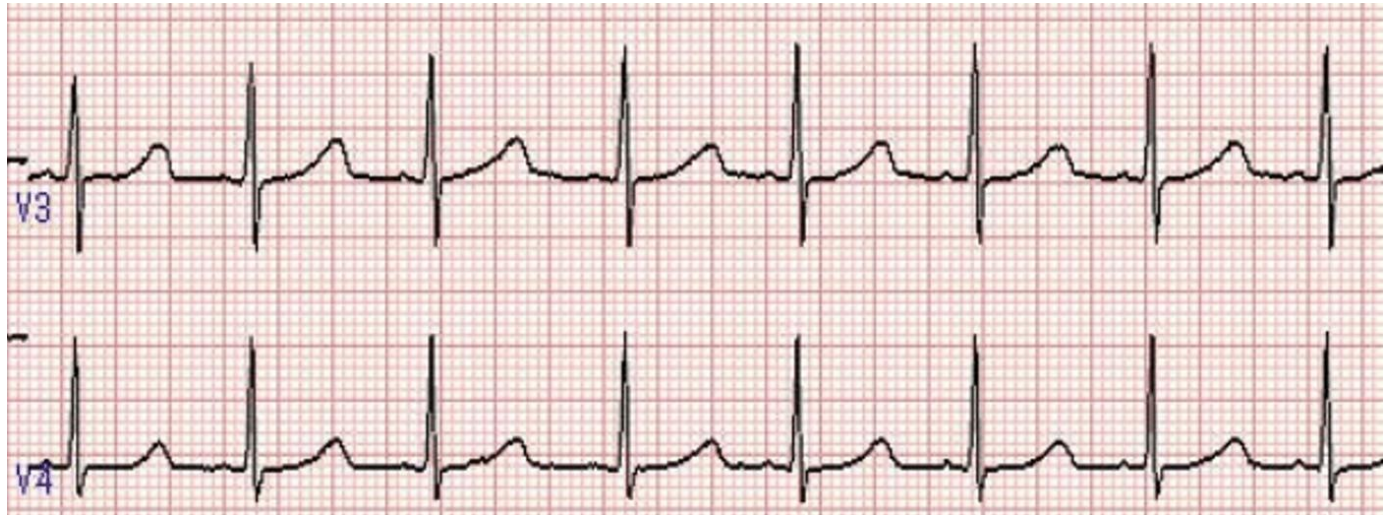
- Monitor symptomatic patients and assess calcium, magnesium and potassium.
- Correct severe symptomatic hypocalcaemia with monitored treatment under the local protocol.
- Investigate the cause; this patient had primary hypoparathyroidism.

Avoid. Syncope plus QT prolongation does not prove that an arrhythmia caused the episode.

CASE 57 | ANOTHER REAL ECG

Compare the patterns

Hypocalcaemia-associated QT prolongation



Miyao M et al. · Figure 1A · CC BY 4.0

The same patient after calcium replacement.



Miyao M et al. · Figure 1B · CC BY 4.0

- QT is shorter on day two.
- Use QTc cautiously when heart rate changes.

Remember. Low calcium can stretch the ST.

CASE 58 | METABOLIC AND TOXIC EFFECTS

High calcium: a shortened recovery phase

PUBLISHED CASE

A 20-year-old woman reports palpitations and previous exertional syncope. Familial hypocalciuric hypercalcaemia is confirmed. Calcium reaches 2.95 mmol/L; her reported QTc is 340 ms.

Could a biochemical abnormality explain the short QT?

Cuny T et al. · Figure 1, daughter before treatment · CC BY 4.0

Three findings to recognise**1**

T waves follow the QRS with a short intervening ST segment.

2

The source reports a QTc of 340 ms.

3

After treatment, the reported QTc is 420 ms.

Remember: High calcium can compress the ST.

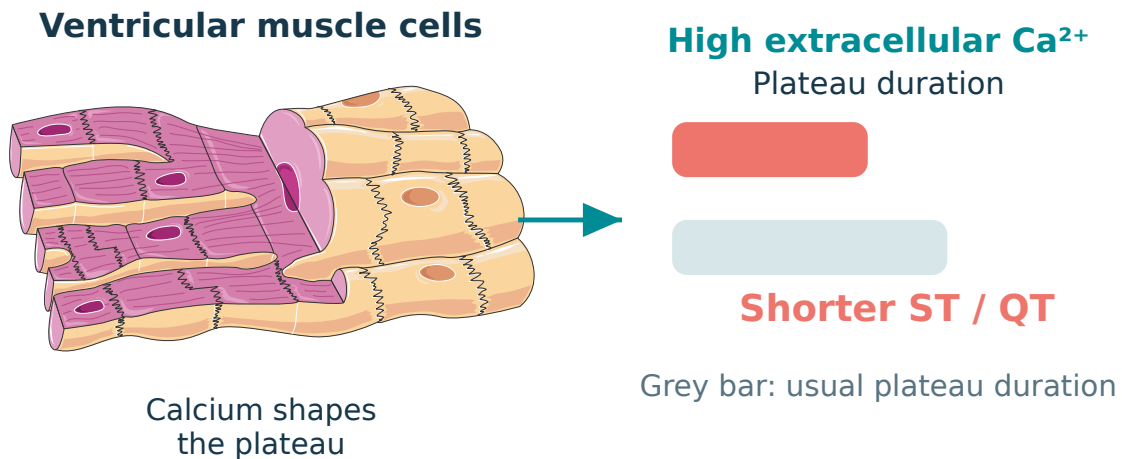
References: V4-ADV-HYPERCALC, C04

CASE 58 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

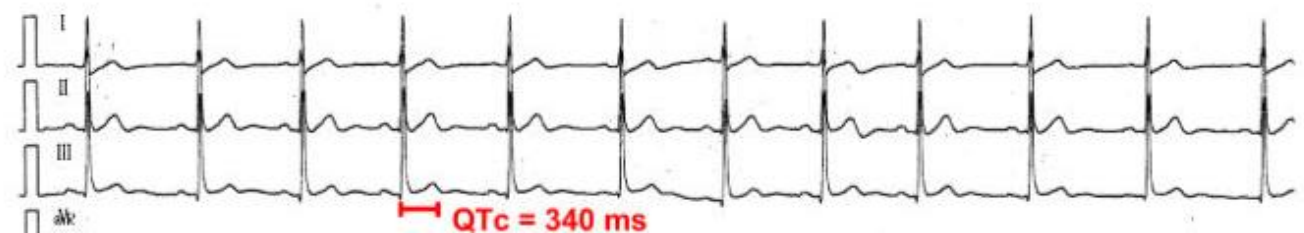
Hypercalcaemia-associated short QT

Calcium changes the duration of ventricular recovery



Calcium changes the ventricular plateau and the ST portion of the QT interval.

- High extracellular calcium can shorten the ventricular plateau.
- Earlier repolarisation reduces the ST segment and QT duration.
- A short QT therefore has acquired as well as inherited causes.



Cuny T et al. · Figure 1, daughter before treatment · CC BY 4.0

In practice

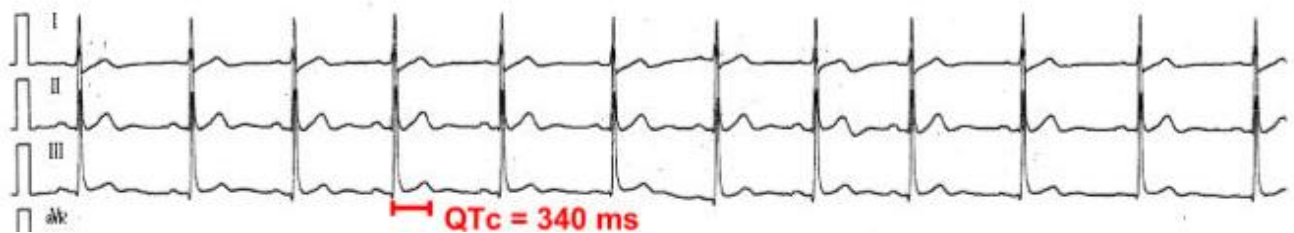
- Confirm calcium, albumin or ionised calcium, and renal function.
- Review symptoms, medicines and other causes of QT shortening.
- Treat the cause with specialist input; management depends on the underlying disorder.

Avoid. Do not label an inherited short-QT syndrome before excluding acquired causes.

CASE 58 | ANOTHER REAL ECG

Compare the patterns

Hypercalcaemia-associated short QT



Cuny T et al. · Figure 1, daughter before treatment · CC BY 4.0

The same patient after three months of specialist treatment.



Cuny T et al. · Figure 1, daughter after treatment · CC BY 4.0

- The QTc has lengthened.
- This case does not establish treatment for every cause of hypercalcaemia.

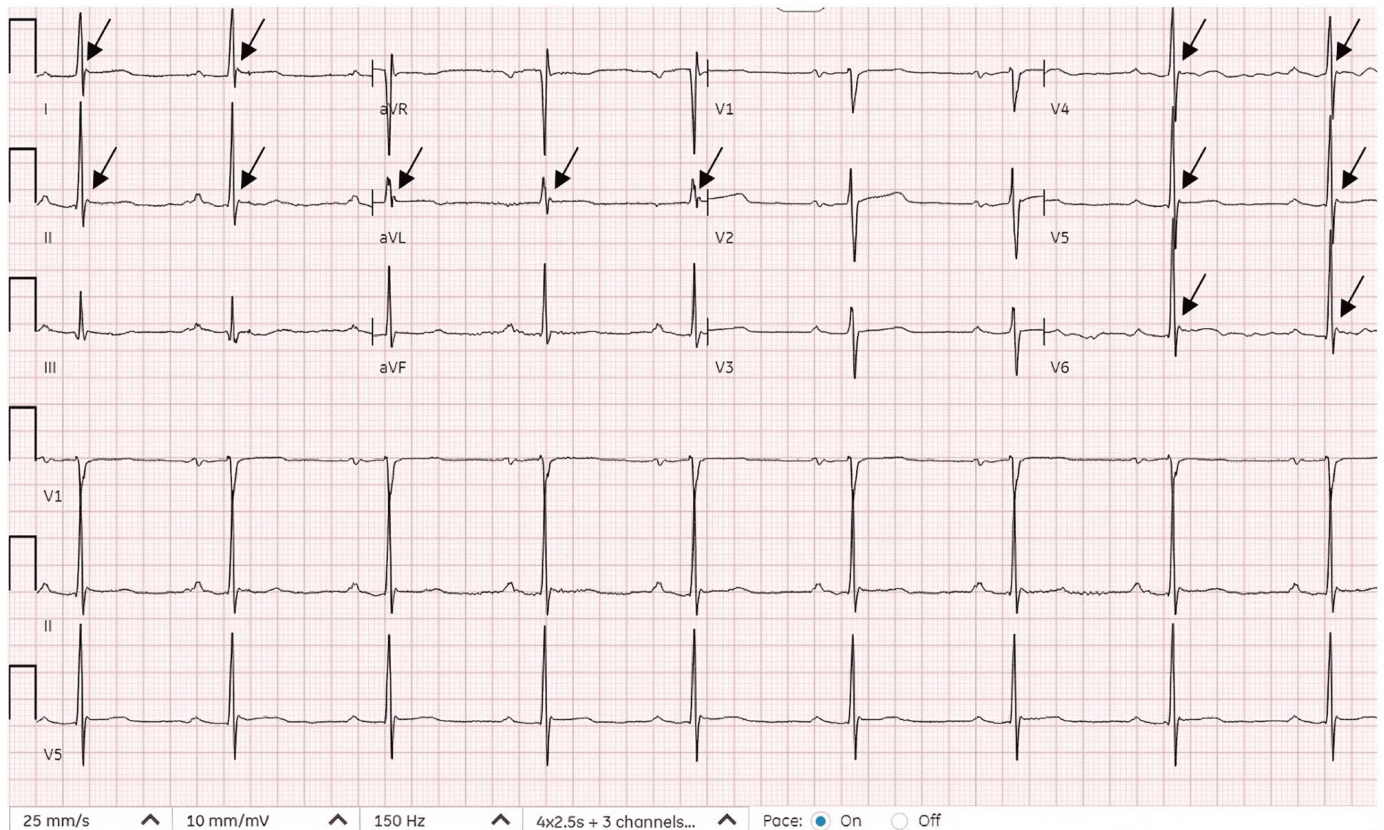
Remember. High calcium can compress the ST.

CASE 59 | METABOLIC AND TOXIC EFFECTS

Cold exposure: recognise the J wave

PUBLISHED CASE

A 78-year-old woman is found confused near an open window. Rectal temperature is 31.1°C and pulse is approximately 53/min.

What is the notch at the QRS-ST junction?

Batikyan A et al. · Figure 1 · CC BY 4.0

Three findings to recognise**1**

The rhythm is slow.

2

Source arrows identify J-point deflections in several leads.

3

The deflections disappear after rewarming.

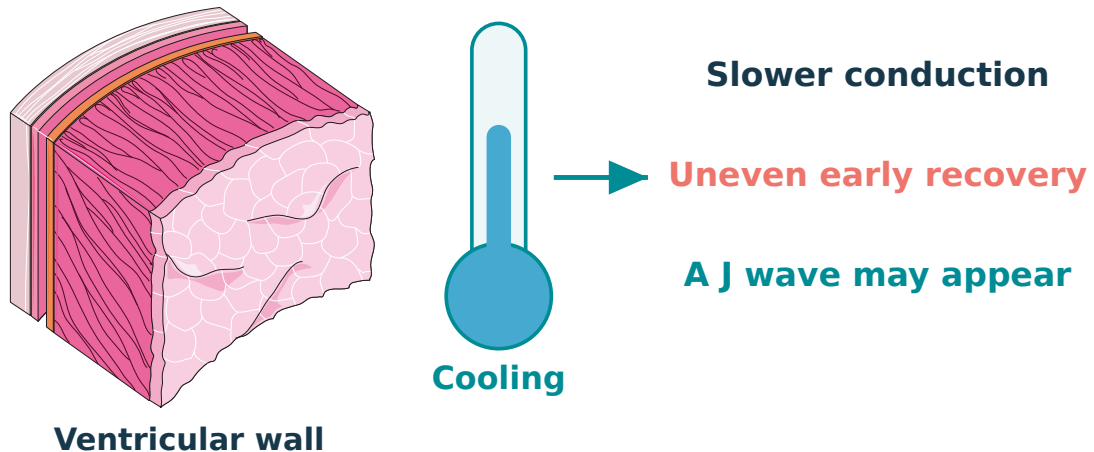
Remember: A cold heart may add a J.

CASE 59 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Hypothermia with Osborn waves

Cold changes both conduction and recovery



Cooling slows conduction and accentuates early-recovery voltage differences across the ventricular wall.

- Cooling slows impulse formation and conduction.
- Unequal recovery across the ventricular wall produces a J wave.
- Greater cooling increases vulnerability to serious arrhythmias.



Batikyan A et al. · Figure 1 · CC BY 4.0

In practice

- Measure core temperature and monitor cardiac rhythm.
- Handle gently and follow a severity-based rewarming pathway.
- Seek associated exposure, metabolic disease, infection or drug effects.

Avoid. J waves have several causes; temperature and the complete clinical picture determine their meaning.

CASE 59 | ANOTHER REAL ECG

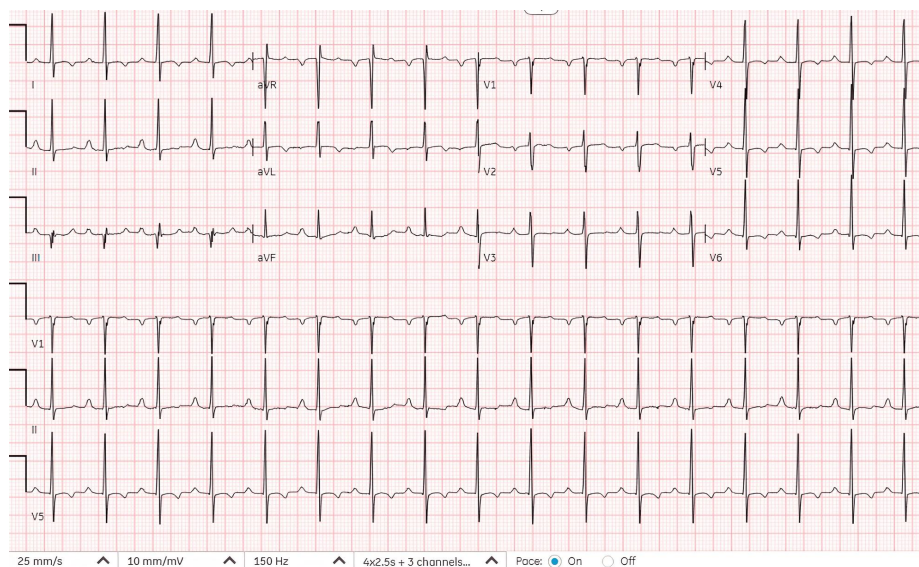
Compare the patterns

Hypothermia with Osborn waves



Batikyan A et al. · Figure 1 · CC BY 4.0

After rewarming, the J waves resolve.



Batikyan A et al. · Figure 2 · CC BY 4.0

- Compare the QRS-ST junction.
- The heart rate is also faster.

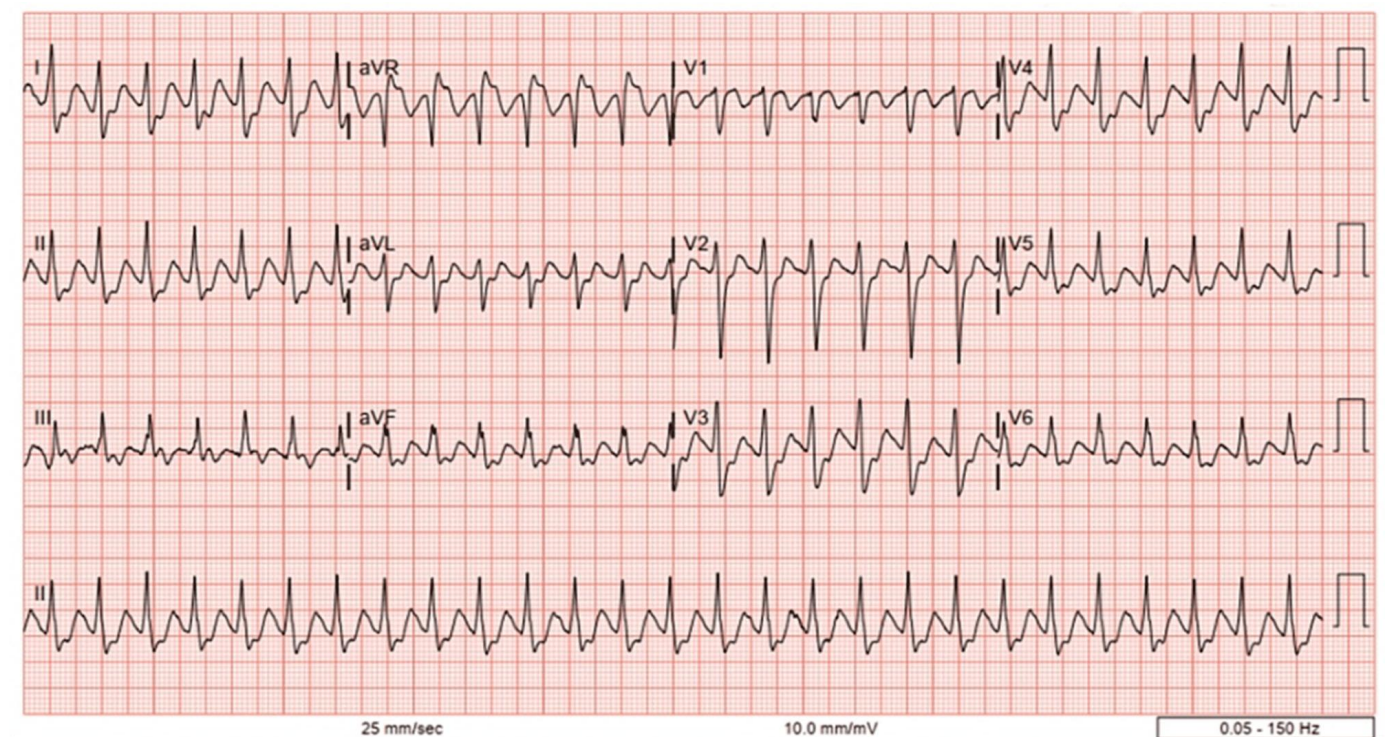
Remember. A cold heart may add a J.

CASE 60 | METABOLIC AND TOXIC EFFECTS

Drug toxicity: the widening QRS

PUBLISHED CASE

A 15-year-old girl is found unresponsive after amitriptyline ingestion. She requires airway support. The initial ECG shows tachycardia and a reported QRS duration of 128 ms.

What does the wide QRS reveal about conduction?

Navarro AW et al. · Figure 1 · CC BY 4.0

Three findings to recognise**1**

The ventricular rate is approximately 163/min.

2

QRS complexes are widened.

3

Later recordings show QRS narrowing during recovery.

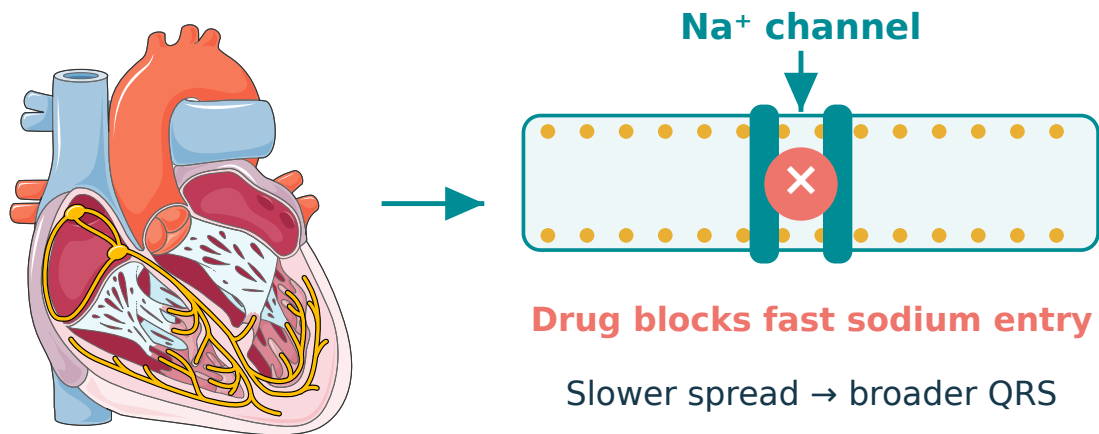
Remember: A wider QRS can signal a blocked sodium route.

CASE 60 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Sodium-channel blocker cardiotoxicity

Sodium-channel block delays ventricular activation



Ventricular activation slows

Blocking fast sodium entry slows activation and broadens the QRS complex.

- Fast sodium channels normally support rapid myocardial activation.
- Channel blockade slows ventricular conduction and broadens the QRS.
- Acidosis can worsen toxicity and cardiovascular instability.



Navarro AW et al. · Figure 1 · CC BY 4.0

In practice

- Stabilise airway and circulation; monitor continuously and contact toxicology urgently.
- Use sodium bicarbonate for significant sodium-channel blocker cardiotoxicity under the emergency protocol.
- Follow serial QRS duration, blood pressure, pH and electrolytes.

Avoid. A negative routine drug screen does not exclude sodium-channel blocker poisoning.

CASE 60 | ANOTHER REAL ECG

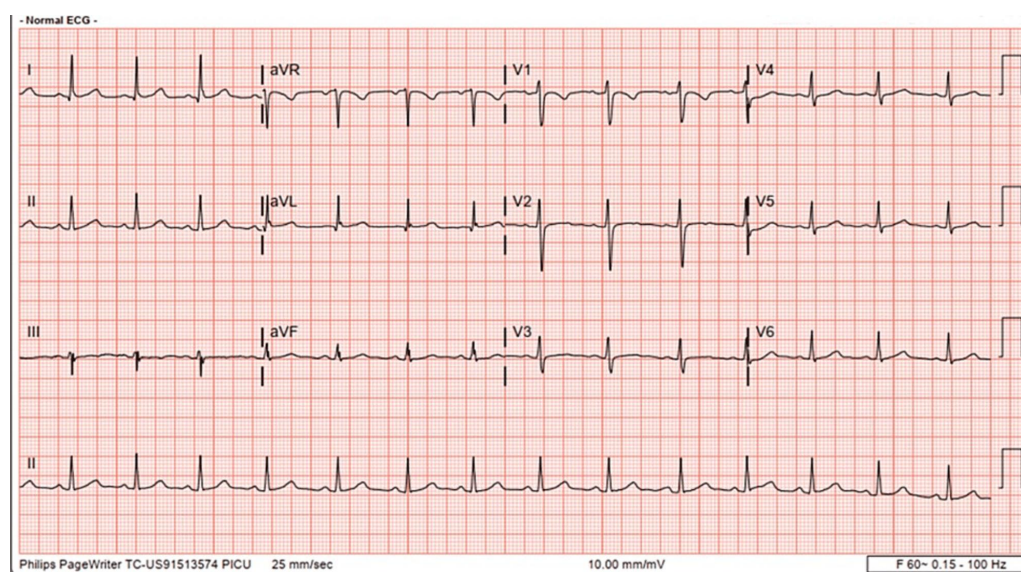
Compare the patterns

Sodium-channel blocker cardiotoxicity



Navarro AW et al. · Figure 1 · CC BY 4.0

Day nine: ventricular conduction has recovered.



Navarro AW et al. · Figure 3 · CC BY 4.0

- QRS duration is approximately 92 ms.
- Recovery time depends on the drug and clinical circumstances.

Remember. A wider QRS can signal a blocked sodium route.

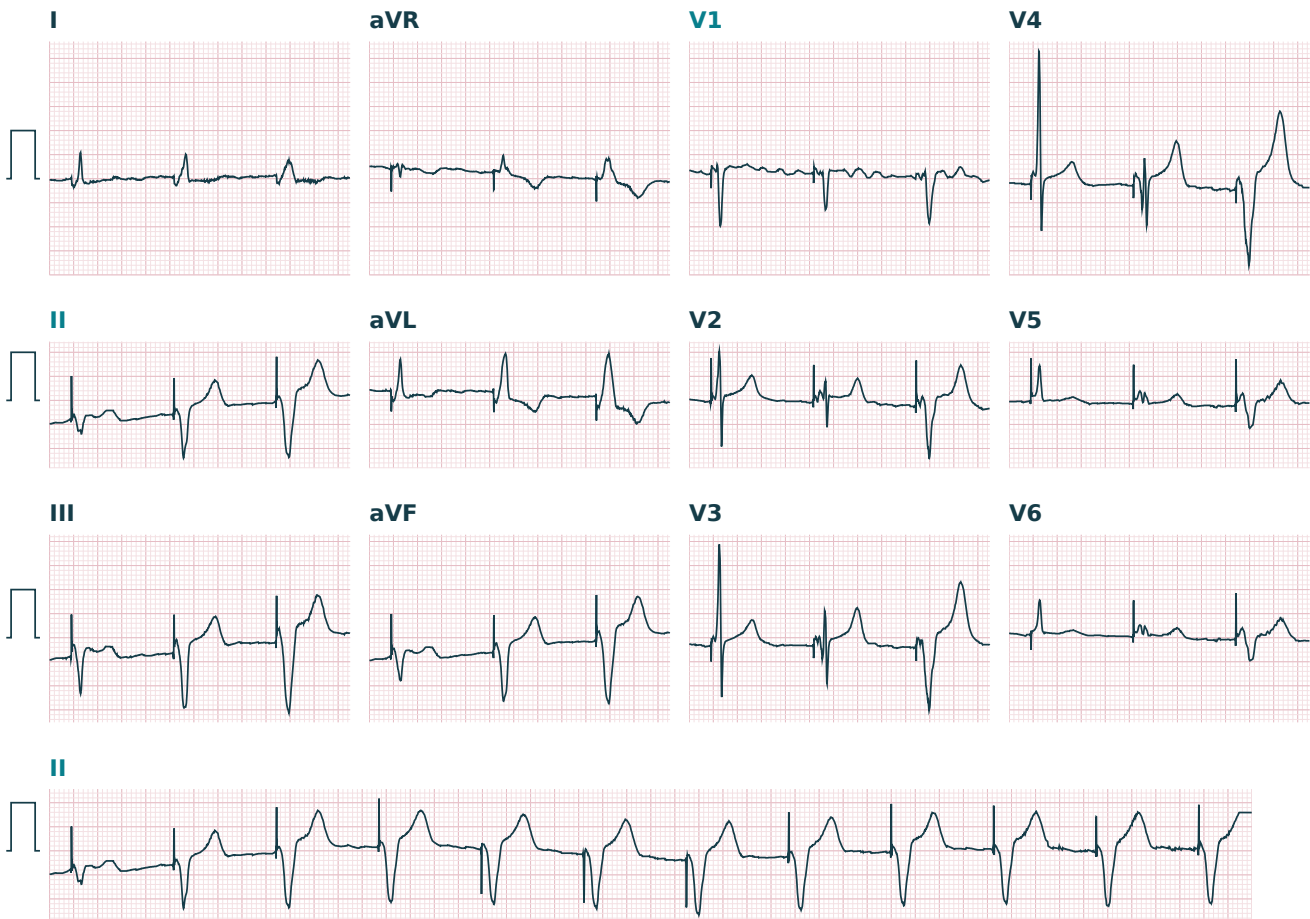
CASE 61 | PACING AND SPECIAL SETTINGS

Pacing: a spike is only the first clue

CLINICAL PRESENTATION

A 79-year-old with a pacemaker reports brief dizziness. He is now stable. Sharp pacing artefacts precede broad ventricular complexes on the ECG.

Does a visible pacing spike prove normal device function?



PTB-XL 00144 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

Pacing artefacts precede several broad QRS complexes.

2

Look for a reproducible ventricular response after each stimulus.

3

Compare pacing visibility across simultaneous leads.

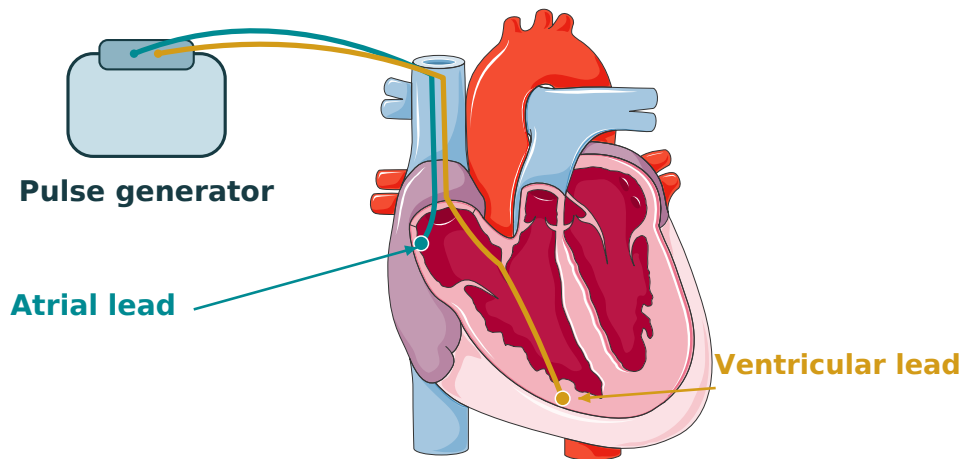
Remember: Stimulus, response and circulation are separate observations.

CASE 61 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Ventricular pacing with visible captured complexes

Dual-chamber pacing: where the leads go



Anatomy: Servier Medical Art, CC BY 4.0. Routes and labels added.

The atrial lead reaches the right atrium; the ventricular lead crosses the tricuspid valve into the right ventricle.

- The generator delivers a stimulus through the lead.
- Sufficient local current depolarises myocardium: capture.
- Activation spreads from the pacing site, shaping the QRS.



PTB-XL 00144 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Assess the patient and compare the prior paced ECG.
- Interrogate the device for sensing, capture, programming and stored events.
- Uncaptured stimuli with haemodynamic compromise require urgent bradycardia care and pacing capability.

Avoid. A missing visible spike does not prove failure to pace.

CASE 61 | ANOTHER REAL ECG

Compare the patterns

Ventricular pacing with visible captured complexes



PTB-XL 00144 | 0-5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Fusion mixes intrinsic and paced ventricular activation



MIT-BIH 102 | source 288 s | 5.2-10.2 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

- QRS shapes change as activation routes combine.
- Fusion alone does not establish device failure.

Remember. Stimulus, response and circulation are separate observations.

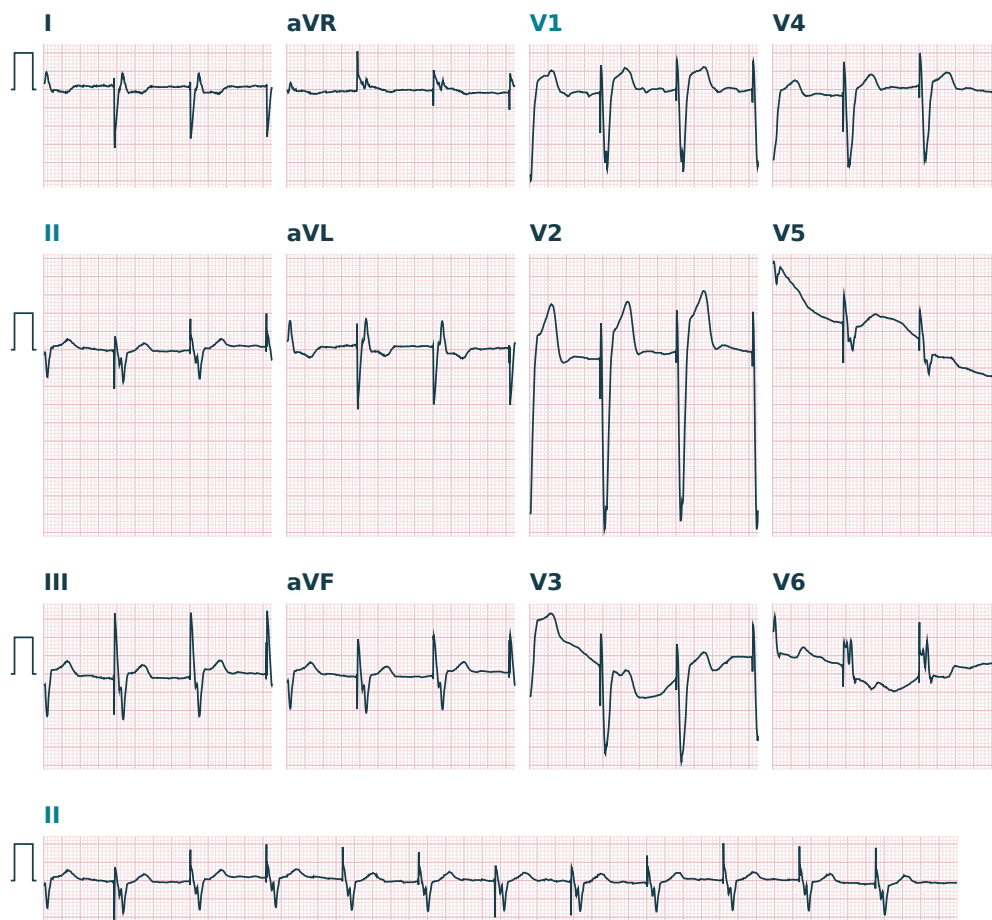
CASE 62 | PACING AND SPECIAL SETTINGS

A device alert needs its stored electrograms

CLINICAL PRESENTATION

A 72-year-old receives an atrial high-rate alert. She feels well; the clinic ECG is paced. The stored episode has not yet been reviewed.

What evidence confirms the device's rhythm label?



PTB-XL 02205 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

The surface ECG shows the rhythm recorded now.

2

Pacing artefacts and ventricular responses can be compared.

3

This tracing cannot establish the earlier alert's mechanism.

Remember: Read the signal behind the label.

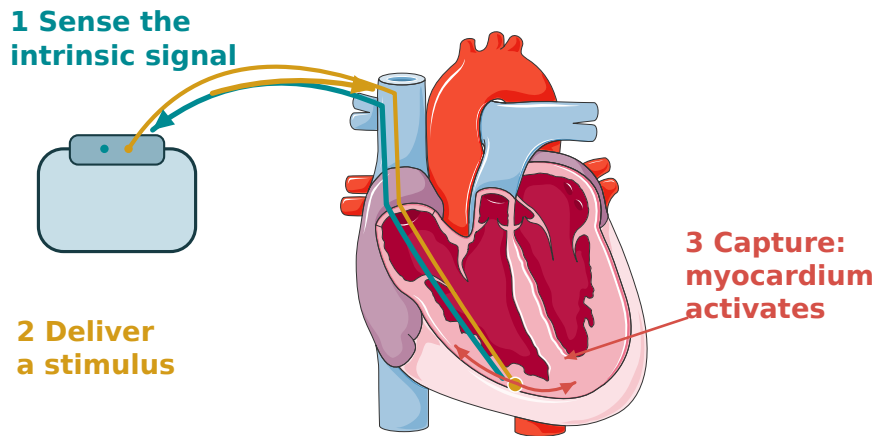
References: C08, C10, C12, P34, PTBXL

CASE 62 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Paced surface rhythm; alert requires electrogram verification

Sensing, output and capture are separate steps



Anatomy: Servier Medical Art, CC BY 4.0. Routes and labels added.

A sensed signal informs device timing; output delivers energy; capture means that cardiac tissue depolarises.

- A sensing electrode detects local and sometimes distant electrical activity.
- Extra detections can make a device count an artificial high rate.
- Stored electrograms reveal timing relationships behind the alert.



PTB-XL 02205 | 0-5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
 PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Review the episode's time, duration, atrial and ventricular electrograms.
- Check marker channels, sensing and programming with the device team.
- Base AF treatment decisions on confirmed rhythm evidence and the patient's clinical risk.

Avoid. Neither an alert count nor a normal clinic ECG settles an earlier rhythm diagnosis.

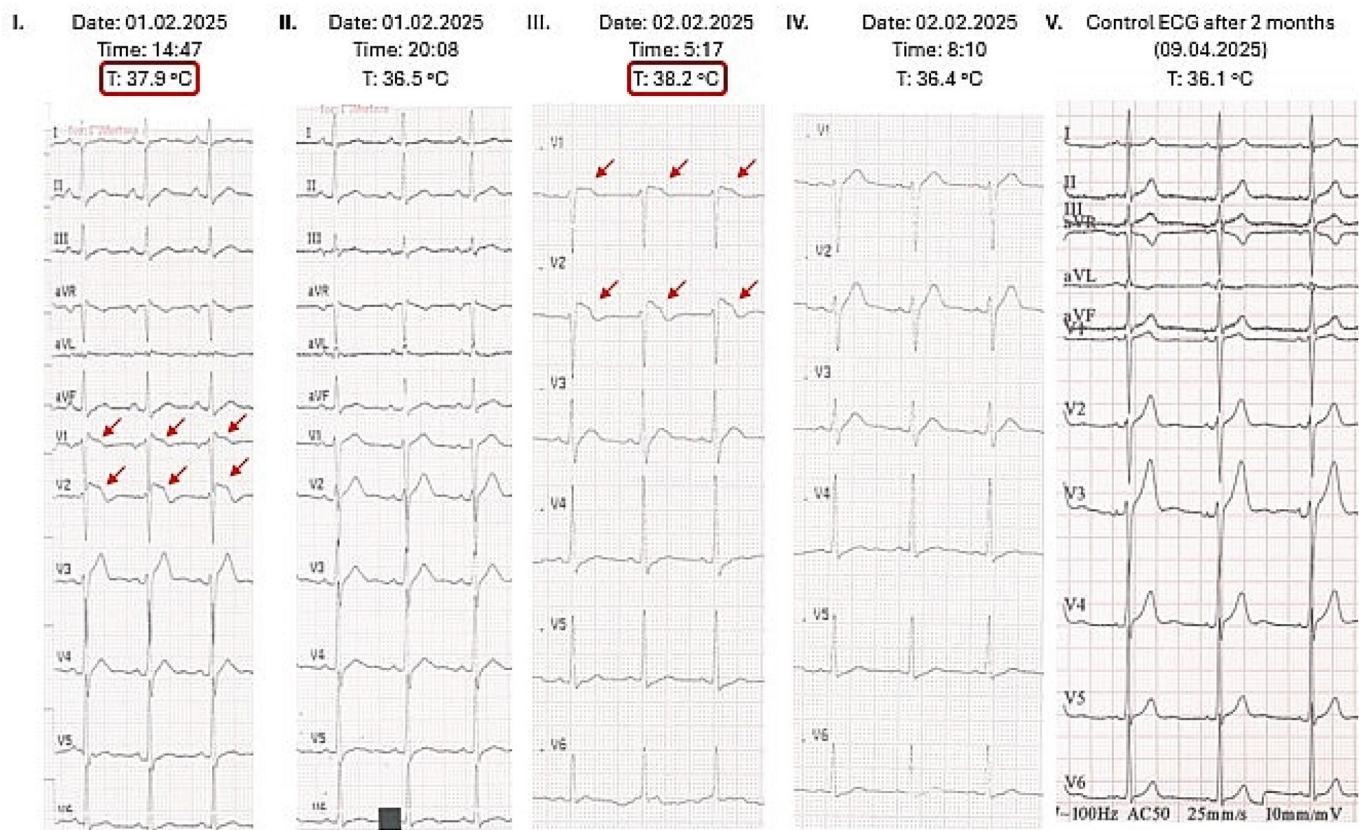
CASE 63 | PACING AND SPECIAL SETTINGS

Fever unmasks a Brugada pattern

PUBLISHED CASE

A 19-year-old man with influenza develops a type 1 ECG pattern during fever. He reports no syncope. The pattern recedes as his temperature falls.

Which ST-T contour matters?



Hamza I et al. | 2026 | Creative Commons Attribution 4.0 International

Three findings to recognise

1

Coved ST elevation appears in the right-precordial leads.

2

Negative T waves follow the elevated ST segments.

3

Serial recordings change with temperature.

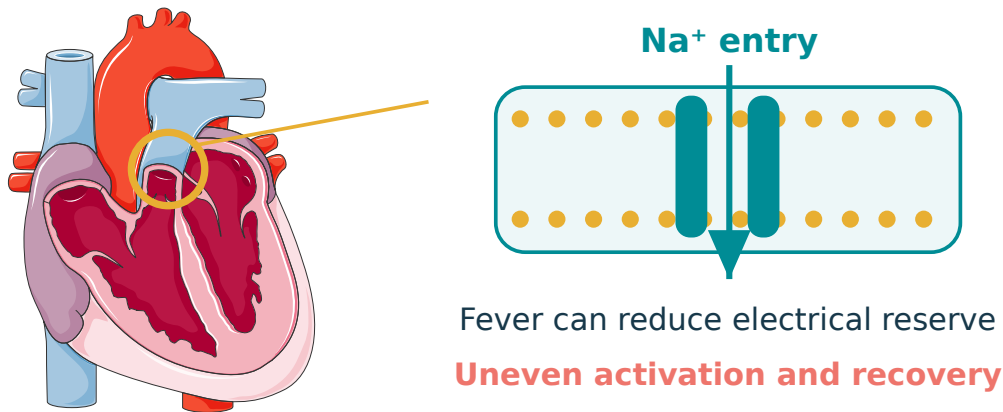
Remember: Fever can reveal a concealed electrical vulnerability.

CASE 63 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Fever-associated type 1 Brugada pattern

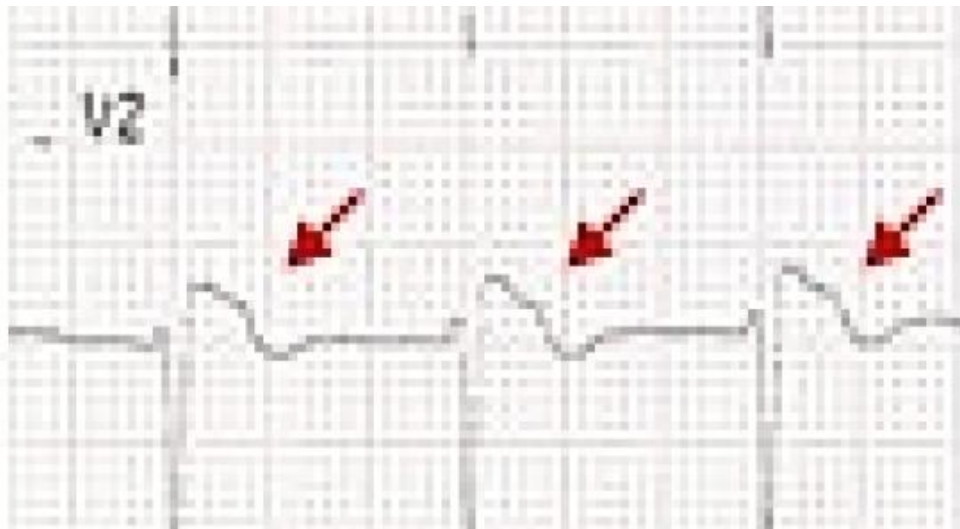
Fever can expose a vulnerable right ventricle



Right ventricular outflow region

Fever alters channel behaviour in susceptible myocardium; right-ventricular activation and recovery may become uneven.

- Fever can reduce electrical reserve in susceptible myocardium.
- Right-ventricular outflow tissue develops uneven activation and recovery.
- The surface ST-T pattern can therefore change with temperature.



Hamza I et al. | 2026 | Creative Commons Attribution 4.0 International

In practice

- Treat fever promptly and review aggravating medicines.
- Ask about syncope, ventricular arrhythmia and familial sudden death.
- Preserve the abnormal ECG and arrange specialist risk assessment.

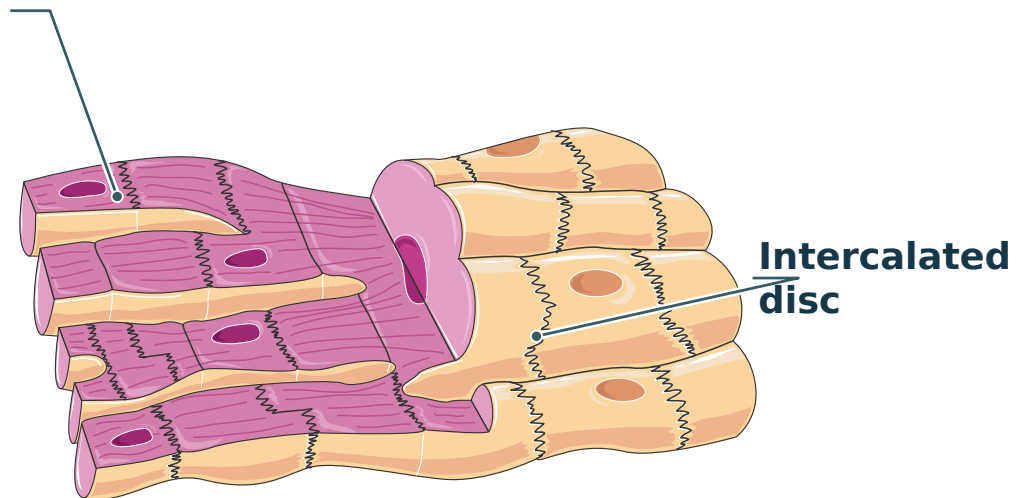
Avoid. A later normal ECG does not erase a documented type 1 pattern.

ANATOMY AT A GLANCE

Interconnected cardiac muscle cells

Connected cells propagate depolarisation

Cardiac muscle cell



Gap junctions within discs electrically couple neighbouring cells. Individual gap-junction channels are not resolved in this artwork.

Connect it to the ECG. Fever can reveal a concealed electrical vulnerability.

- Fever can reduce electrical reserve in susceptible myocardium.
- Right-ventricular outflow tissue develops uneven activation and recovery.
- The surface ST-T pattern can therefore change with temperature.

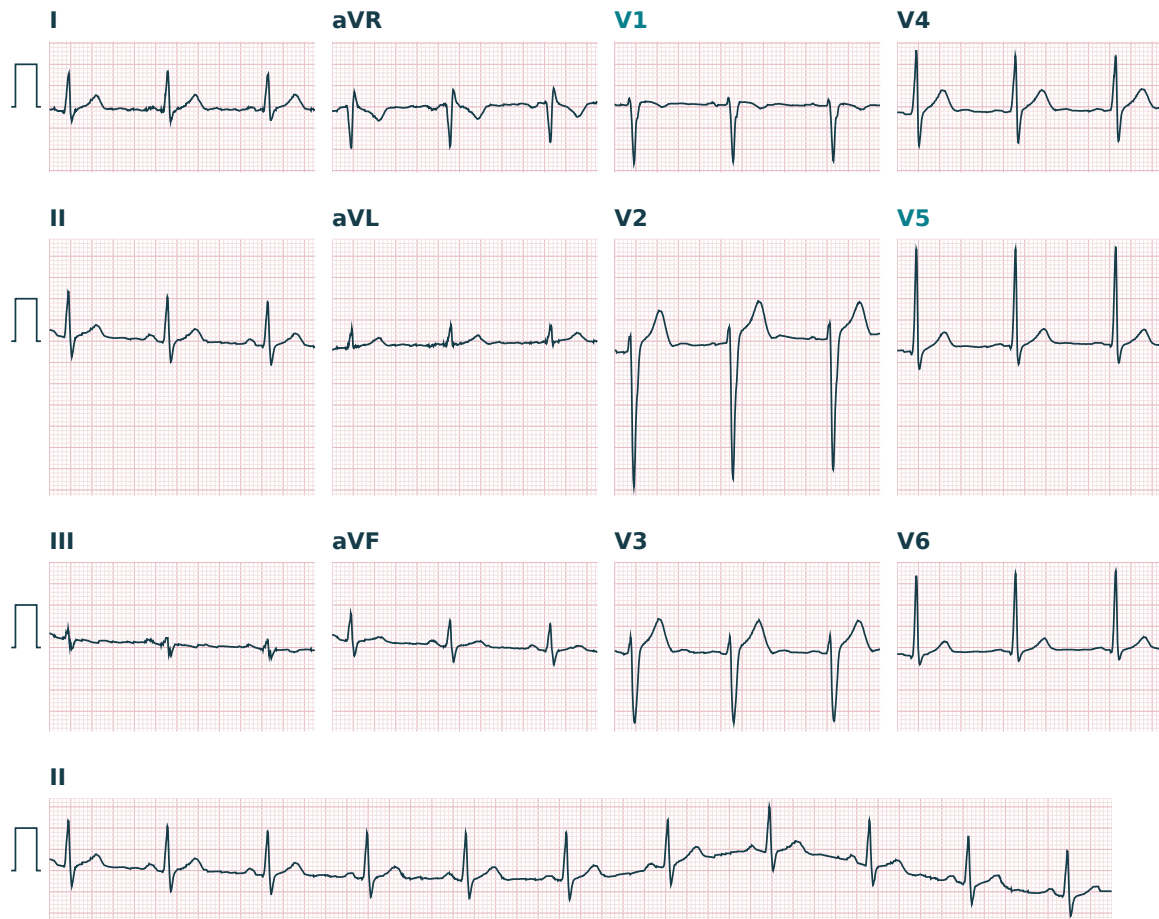
Adapted from Servier Medical Art, CC BY 4.0. Teaching labels added.

CASE 64 | PACING AND SPECIAL SETTINGS

High voltage: training or disease?

CLINICAL PRESENTATION

A 48-year-old runner has hypertension and new breathlessness on hills. The ECG has large chest-lead complexes.

Is voltage alone a structural diagnosis?

PTB-XL 00030 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

- 1**
V1 has a deep S wave.
- 2**
V5 and V6 have tall R waves.
- 3**
Assess lateral ST-T changes separately from voltage.

Remember: Voltage suggests; symptoms and imaging decide.

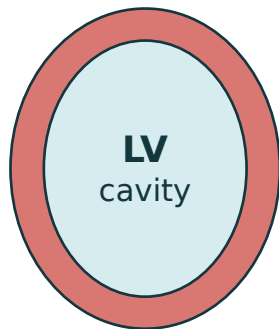
CASE 64 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

High QRS voltage requiring clinical assessment

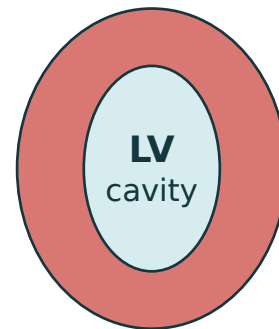
Similar voltage can have different causes

Training adaptation



Proportionate enlargement

Pathological pressure loading



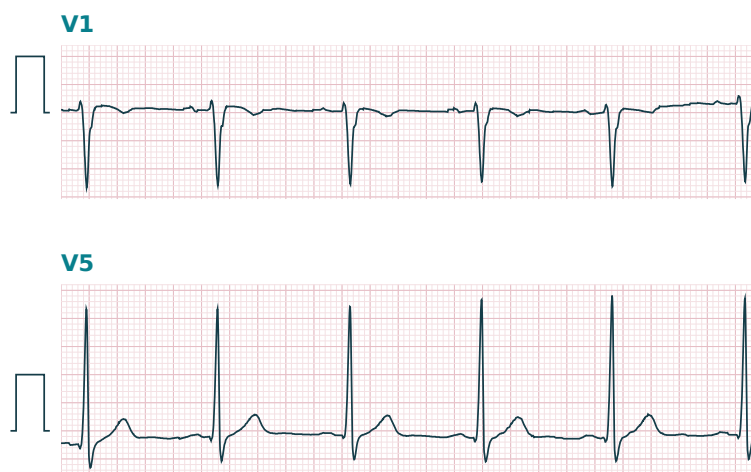
Wall thickening

Symptoms
→ Repolarisation ←
Imaging

Interpret the complete clinical pattern

Similar voltage can accompany athletic adaptation or pathological loading; accompanying findings and imaging identify the clinically important difference.

- Ventricular activation produces forces directed towards the lateral chest.
- Muscle mass, body build and electrode distance affect surface voltage.
- Training and pathological loading can both increase voltage.



PTB-XL 00030 | 0.5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

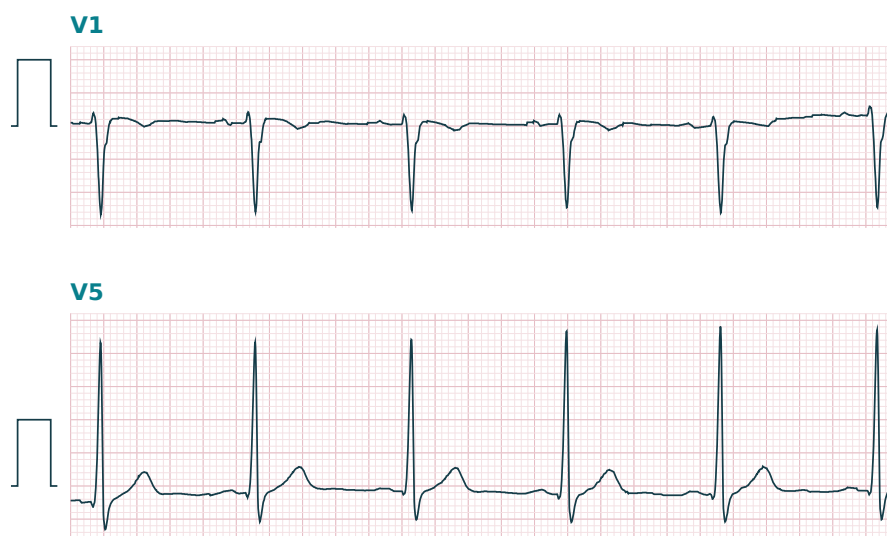
- Recheck blood pressure and assess exertional symptoms.
- Use echocardiography to establish chamber size, wall thickness and function.
- Investigate syncope, familial sudden death or concerning repolarisation changes.

Avoid. Do not diagnose hypertrophic cardiomyopathy from a voltage threshold alone.

CASE 64 | ANOTHER REAL ECG

Compare the patterns

High QRS voltage requiring clinical assessment



PTB-XL 00030 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Compare with this ordinary-voltage recording.



PTB-XL 00033 | 0.35-5.35 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- Use the printed voltage grid.
- Compare morphology as well as amplitude.

Remember. Voltage suggests; symptoms and imaging decide.

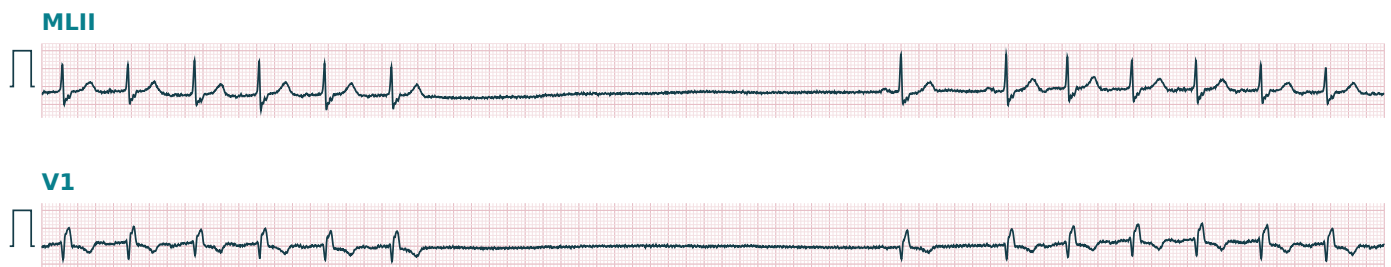
CASE 65 | PACING AND SPECIAL SETTINGS

Capture the cause of intermittent collapse

CLINICAL PRESENTATION

An adult reports sudden light-headedness and one collapse. Clinic ECGs have been unremarkable. Ambulatory monitoring captures a long pause.

Does the pause explain the symptoms?



MIT-BIH 232 | source 1414 s | 0-15 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise

1

The marked ventricular interval is approximately 5.69 seconds.

2

Both channels show the same interruption.

3

Inspect atrial activity before assigning the mechanism.

Remember: Find the pause, then look upstream.

References: S08, C08, C11

CASE 65 | UNDERSTAND AND APPLY

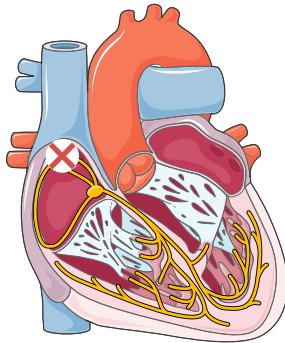
From anatomy to clinical meaning

Prolonged ventricular pause

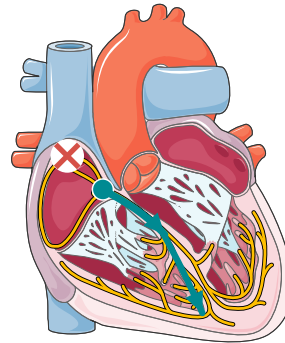
Sinus pause: escape starts from a slower pacemaker

Sinus activation pauses

A junctional focus escapes



No sinus impulse reaches the atria

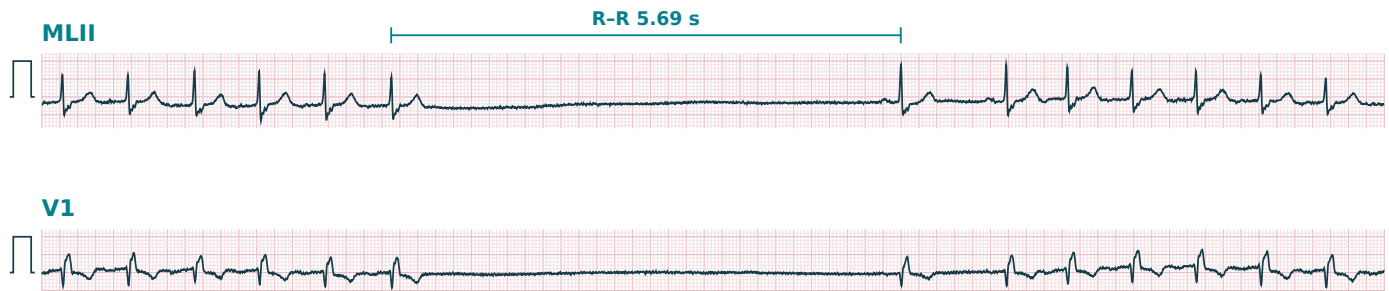


A slower backup impulse activates the ventricles

Anatomy: Servier Medical Art, CC BY 4.0. Routes and labels added.

If sinus firing fails, a slower subsidiary pacemaker may generate an escape beat through the downstream conduction system.

- Reduced ventricular activation can interrupt effective cardiac output.
- Sinus failure and AV block create different atrial patterns.
- A brief resting ECG can miss an intermittent event.



MIT-BIH 232 | source 1414 s | 0-15 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

- Compare the recording time with the symptom diary.
- Review medicines, electrolytes and reversible contributors.
- Assess significant bradyarrhythmia promptly and select further monitoring by event frequency.

Avoid. Pause duration alone does not establish its mechanism or the indication for pacing.

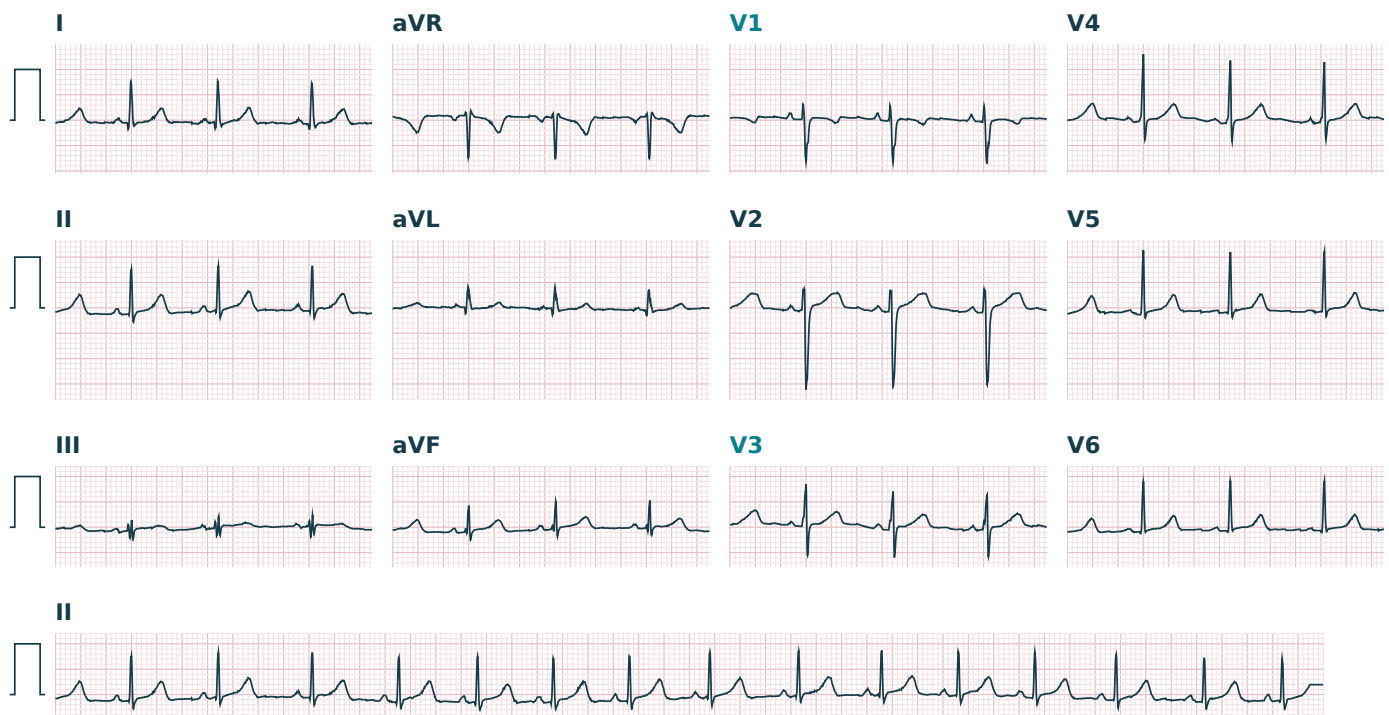
CASE 66 | PACING AND SPECIAL SETTINGS

The child's ECG: start with age

CLINICAL PRESENTATION

An eight-year-old has brief non-exertional chest discomfort, a normal examination and no exercise-related collapse. The computer flags a negative T wave in V1.

Is an adult reference appropriate?



PTB-XL 06598 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

P waves precede successive QRS complexes.

2

V1 shows a small R followed by a deeper S.

3

T is negative in V1 but upright in the displayed V2-V3.

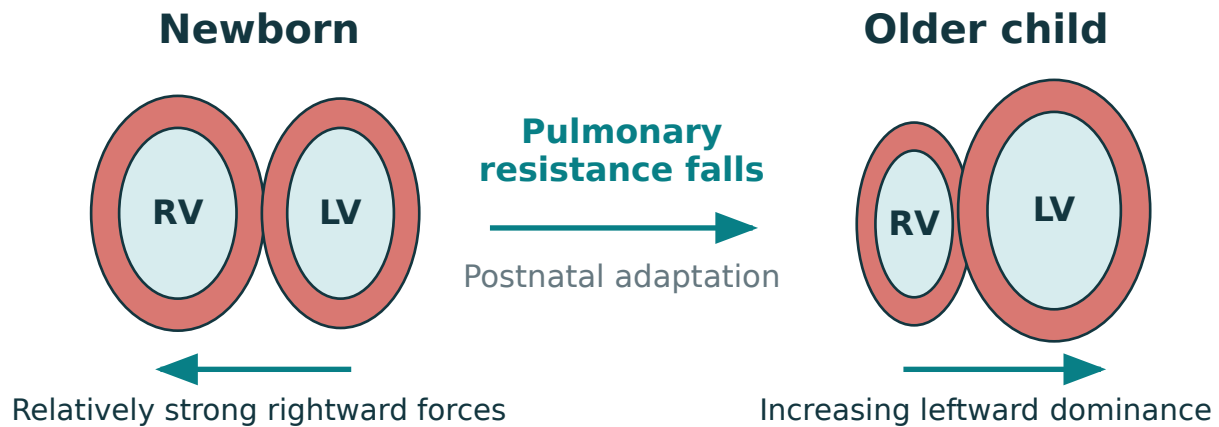
Remember: Read the age before the abnormality.

CASE 66 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Age-dependent right-precordial T-wave pattern

Age changes the electrical reference



Ventricular forces and recovery mature differently

After birth, ventricular electrical balance shifts leftward; paediatric T-wave expectations also change with age.

- Postnatal ventricular forces gradually become more leftward.
- Right-precordial repolarisation also changes during childhood.
- Adult criteria can misclassify age-appropriate findings.



PTB-XL 06598 | 0.5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Verify age, symptoms and electrode positions.
- Use paediatric reference ranges for rate, intervals and axis.
- Investigate exertional syncope or a concerning family history.

Avoid. An age-appropriate ECG does not dismiss a high-risk clinical presentation.

CASE 66 | ANOTHER REAL ECG

Compare the patterns

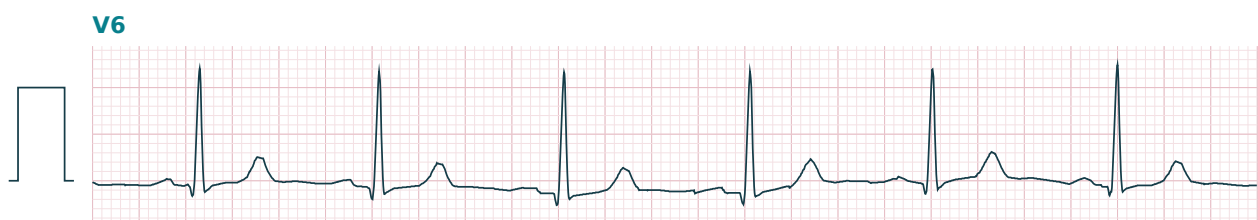
Age-dependent right-precordial T-wave pattern



PTB-XL 06598 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Adult comparison: developmental reference ranges differ.



PTB-XL 00033 | 0.35-5.35 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- Compare the right-precordial T wave.
- Do not transfer adult thresholds unchanged.

Remember. Read the age before the abnormality.

CASE 67 | PACING AND SPECIAL SETTINGS

Presyncope with 2:1 AV block

CLINICAL PRESENTATION

An older adult becomes presyncopal. The pulse is slow and regular, and blood pressure is falling. Two atrial cycles occur for each ventricular complex.

Which atrial impulse fails to conduct?



MIT-BIH 231 | source 1211 s | 0-12 s, simultaneous | MLII, V1
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

Three findings to recognise**1**

Atrial deflections continue between the QRS complexes.

2

Alternate P waves have no following QRS.

3

The ventricular response is slow and the complexes are broad.

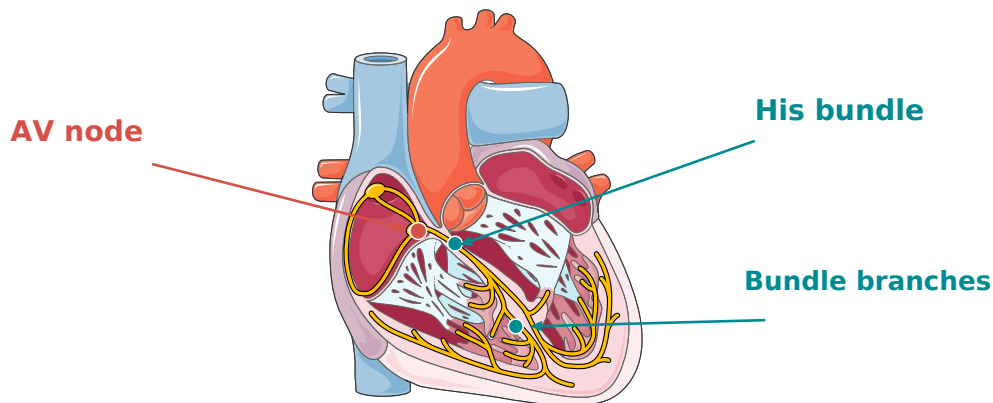
Remember: Count atrial events before naming the slow rhythm.

CASE 67 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Symptomatic 2:1 atrioventricular block

AV block can interrupt conduction at different levels

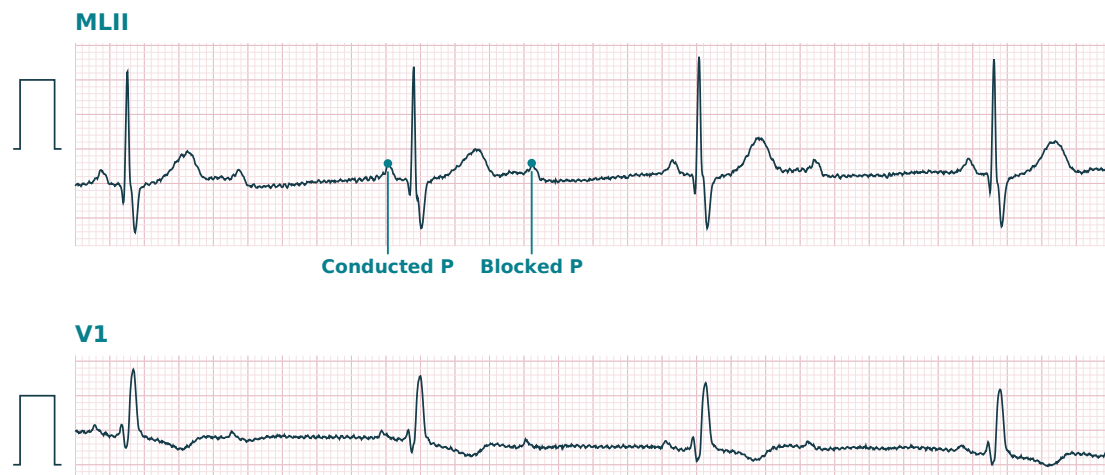


Atrial impulses may continue while ventricular responses fail.

Anatomy: Servier Medical Art, CC BY 4.0. Routes and labels added.

The AV node and His-Purkinje system are different possible sites of failed atrioventricular conduction.

- Atrial impulses continue regularly.
- Alternate impulses fail to reach the ventricles.
- Reduced ventricular output can compromise cerebral perfusion.



MIT-BIH 231 | source 1211 s | 0-6 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

In practice

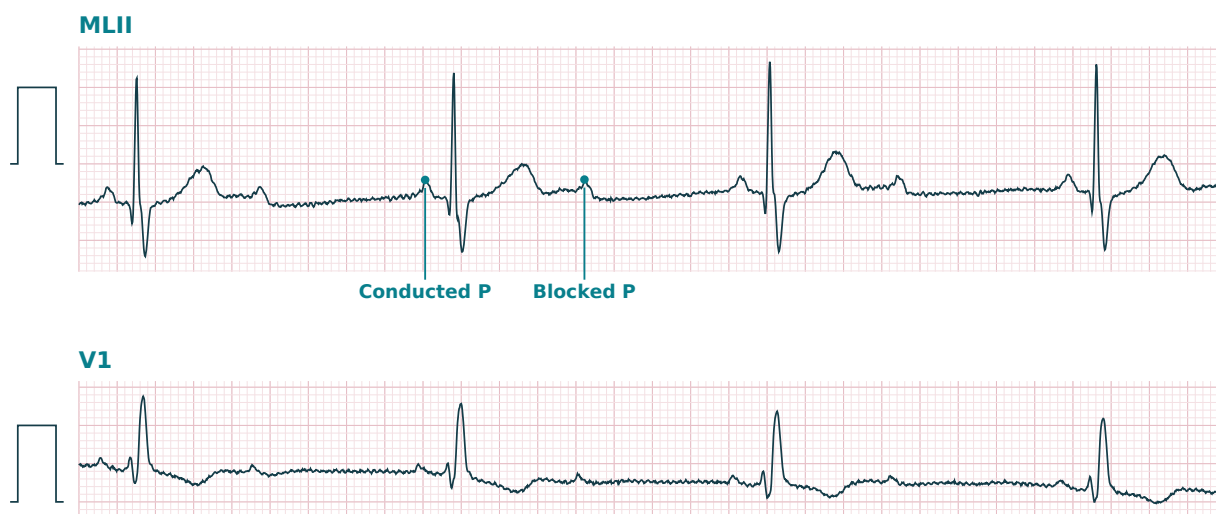
- Assess circulation, monitor continuously and summon immediate clinical help.
- Prepare pacing capability while following the current symptomatic-bradycardia pathway.
- Investigate ischaemia, drugs and electrolyte abnormalities without delaying stabilisation.

Avoid. An isolated 2:1 pattern cannot usually be assigned Mobitz I or II.

CASE 67 | ANOTHER REAL ECG

Compare the patterns

Symptomatic 2:1 atrioventricular block



MIT-BIH 231 | source 1211 s | 0-6 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

MIT-BIH | Moody & Mark | ODC Attribution 1.0

First-degree block: delayed conduction, without dropped ventricular responses.



PTB-XL 03948 | 0-3 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- Each P is followed by a QRS.
- PR is prolonged, but conduction remains one-to-one.

Remember. Count atrial events before naming the slow rhythm.

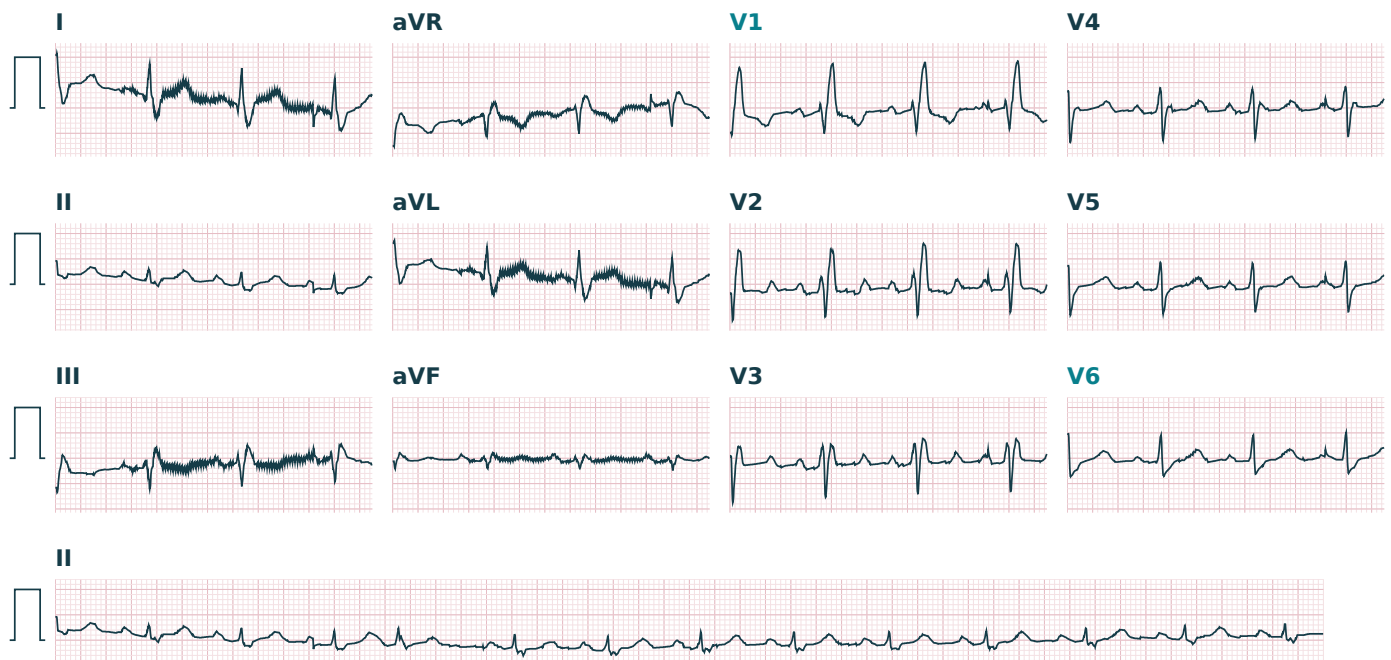
CASE 68 | PACING AND SPECIAL SETTINGS

Write a report that guides the next clinician

CLINICAL PRESENTATION

An ECG is obtained for intermittent palpitations. Ventricular intervals vary, while the right-precordial QRS has a prominent terminal positive component.

Which finding concerns timing, and which concerns activation?



PTB-XL 05212 | Upper leads: 0-2.5 s, simultaneous | Lead II: 0-10 s
25 mm/s, 10 mm/mV proportions | Square grid: 40 ms x 0.1 mV | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Three findings to recognise

1

V1-V2 have prominent terminal positive QRS components.

2

I and V6 show broad terminal negative components.

3

Variable R-R intervals require a separate rhythm assessment.

Remember: Separate the clock from the route.

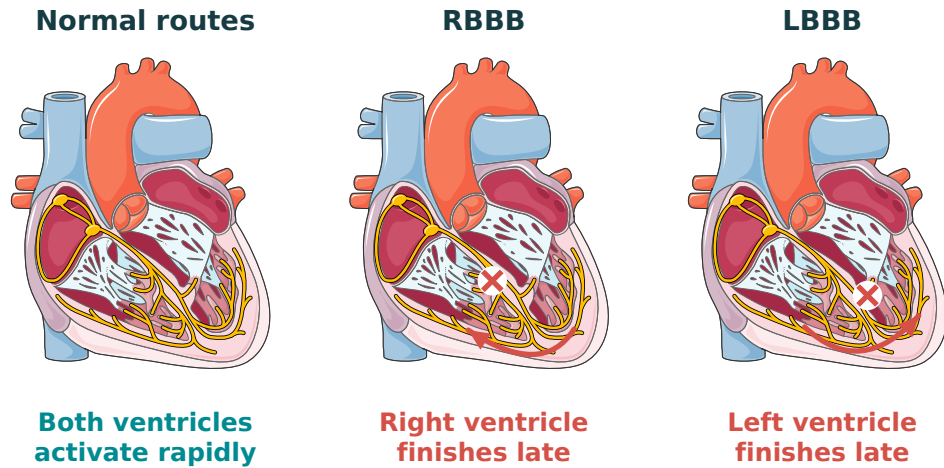
References: C02, C03, C10, S08

CASE 68 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Right bundle-branch pattern with variable ventricular timing

Bundle-branch block: which ventricle finishes last?

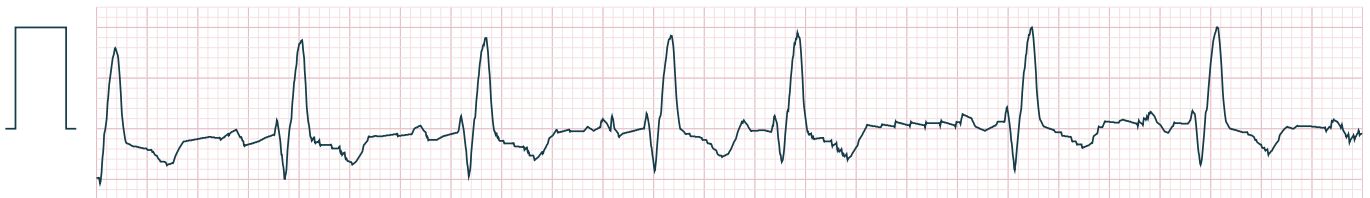


Anatomy: Servier Medical Art, CC BY 4.0. Routes and labels added.

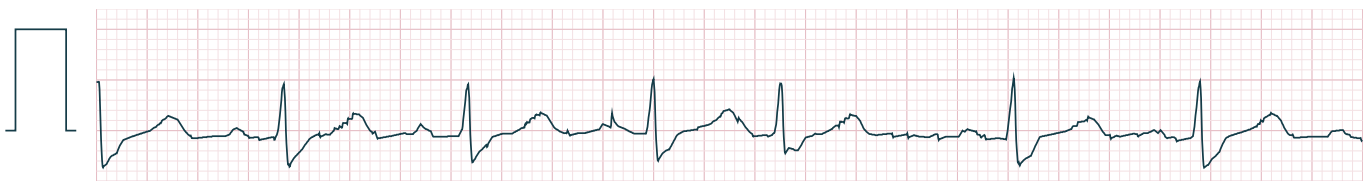
The intact branch activates its ventricle first; slower myocardial spread reaches the delayed ventricle. Routes are simplified.

- The atrial rhythm determines when impulses arrive.
- Right-bundle delay changes how the ventricles activate.
- Late rightward forces point towards V1 and away from lateral leads.

V1



V6



PTB-XL 05212 | 0-5 s | 25 mm/s, 10 mm/mV proportions
 Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

In practice

- Report rate and atrial-ventricular relationship separately from QRS morphology.
- Describe the supporting leads and compare the previous ECG.
- State the clinical priority and the evidence still needed.

Avoid. A bundle-branch label does not explain an irregular ventricular rhythm.

CASE 68 | ANOTHER REAL ECG

Compare the patterns

Right bundle-branch pattern with variable ventricular timing



PTB-XL 05212 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

Compare left-bundle activation with the right-bundle pattern.



PTB-XL 01564 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom

PTB-XL | Wagner et al. | CC BY 4.0

- The lateral ventricular contour differs.
- Interpret rhythm separately in either conduction pattern.

Remember. Separate the clock from the route.

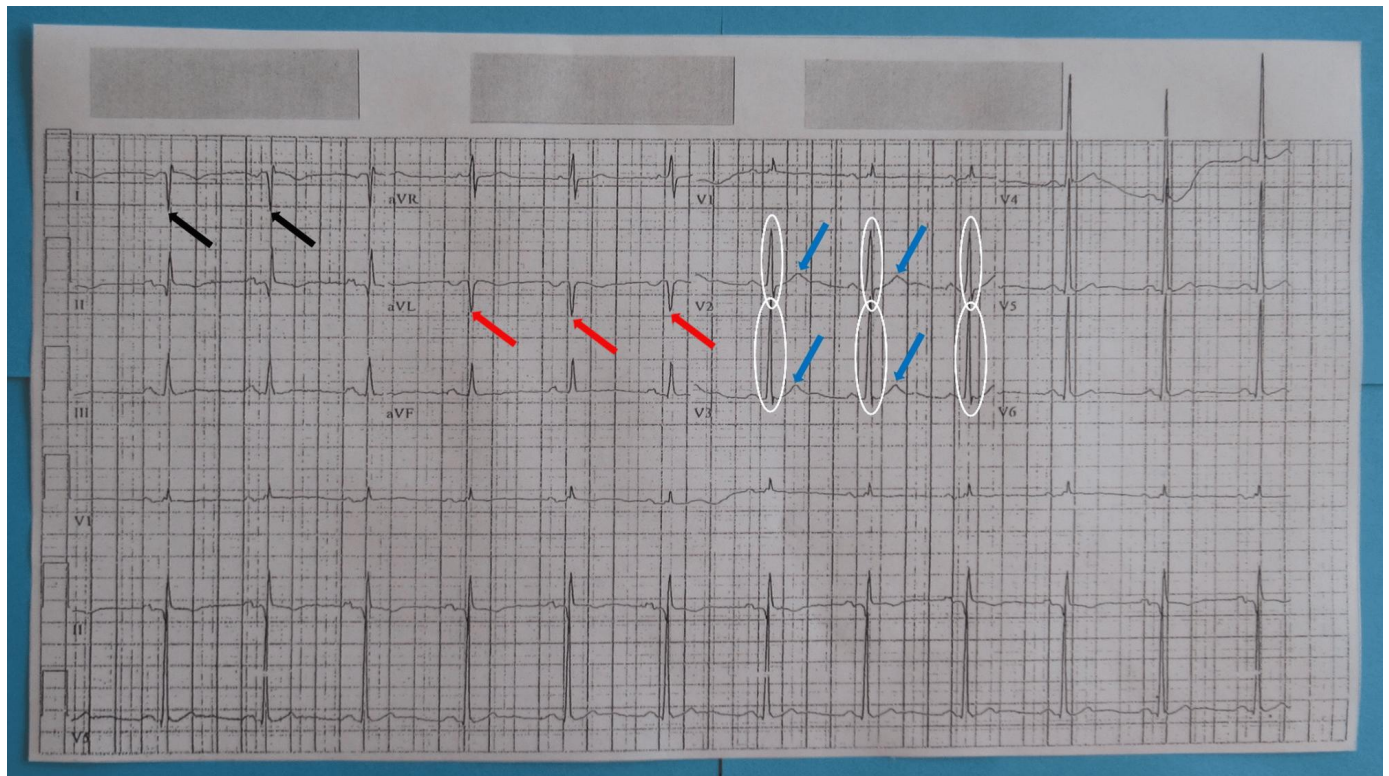
CASE 69 | PACING AND SPECIAL SETTINGS

Arm-lead reversal: check the patient, then the cables

PUBLISHED CASE

A 58-year-old man recovering from spinal surgery has an ECG labelled as infarction. He has no matching ischaemic symptoms; repeat correctly placed electrodes resolve the apparent abnormalities.

Do the limb and chest leads tell the same story?



Cohen PR et al. · Figures 2 and 3 · CC BY 4.0

Three findings to recognise

1

Lead I has inverted P, QRS and T deflections.

2

aVR becomes unexpectedly positive.

3

Chest-lead R-wave progression is preserved.

Remember: Upside-down I: inspect the arms.

References: V4-ADV-REVERSAL, C02

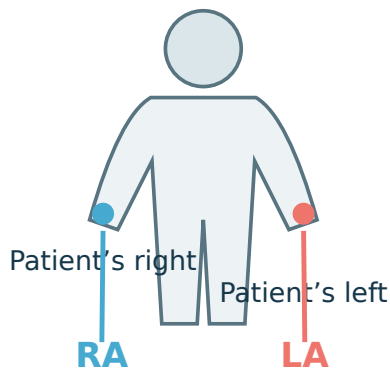
CASE 69 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

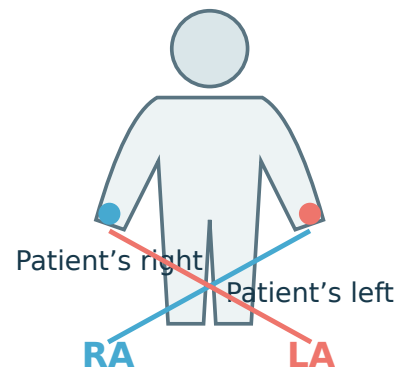
Right-arm/left-arm electrode reversal

Check the patient-to-cable connection

Correct arm cables



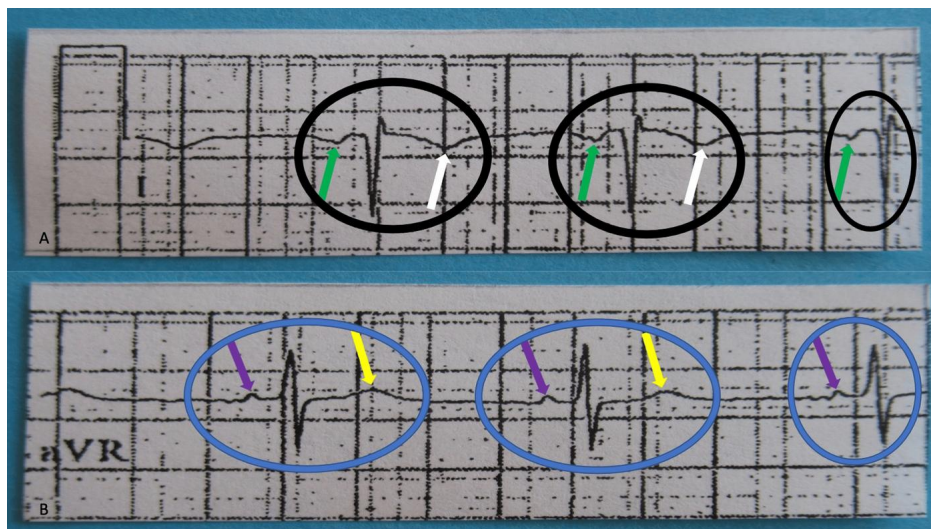
Arm cables exchanged



Exchanging RA and LA reverses the polarity of lead I

Exchanging the arm cables reverses lead I and alters the other limb-lead relationships.

- Swapping the arm electrodes reverses lead I.
- Leads II and III swap, as do aVR and aVL.
- Chest leads retain their usual orientation.



Cohen PR et al. · Figures 2 and 3 · CC BY 4.0

In practice

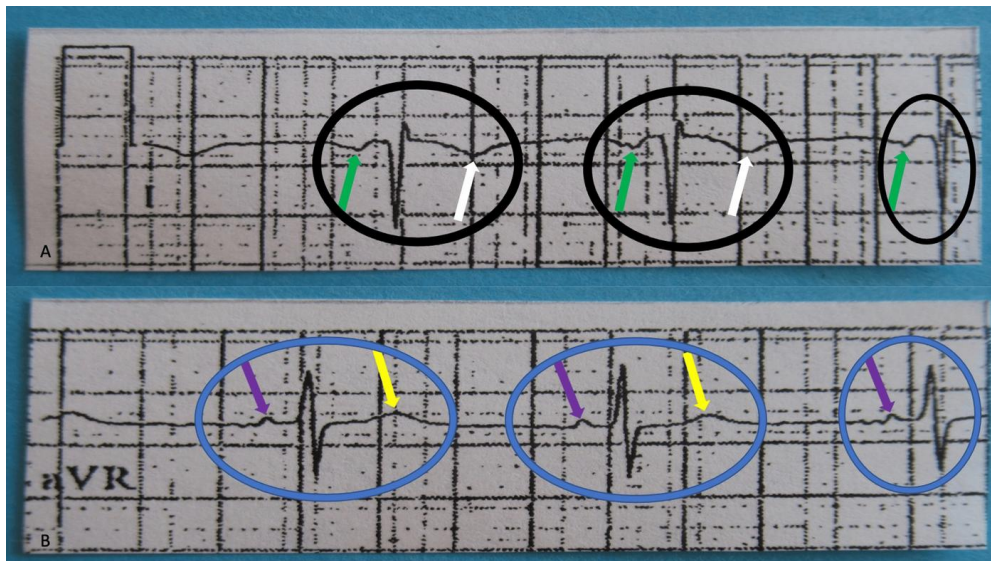
- Inspect electrode labels and locations.
- Repeat the ECG with verified placement and compare prior recordings.
- Continue clinical assessment if symptoms or abnormalities remain concerning.

Avoid. An acquisition error can coexist with real disease; do not dismiss persistent symptoms.

CASE 69 | ANOTHER REAL ECG

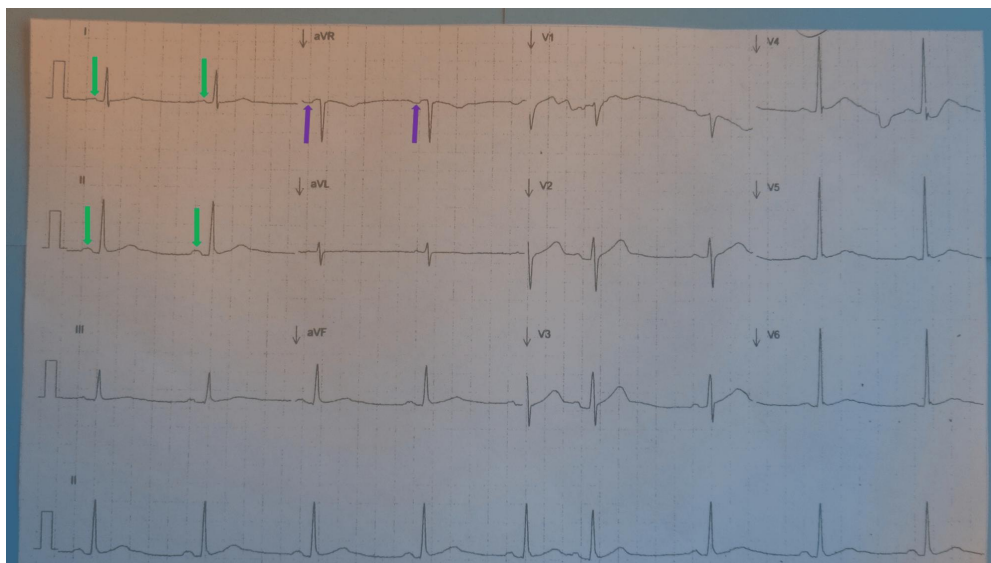
Compare the patterns

Right-arm/left-arm electrode reversal



Cohen PR et al. · Figures 2 and 3 · CC BY 4.0

Repeat recording after correcting the electrode placement.



Cohen PR et al. · Figure 4 · CC BY 4.0

- Lead I returns to its usual orientation.
- Apparent infarction features resolve.

Remember. Upside-down I: inspect the arms.

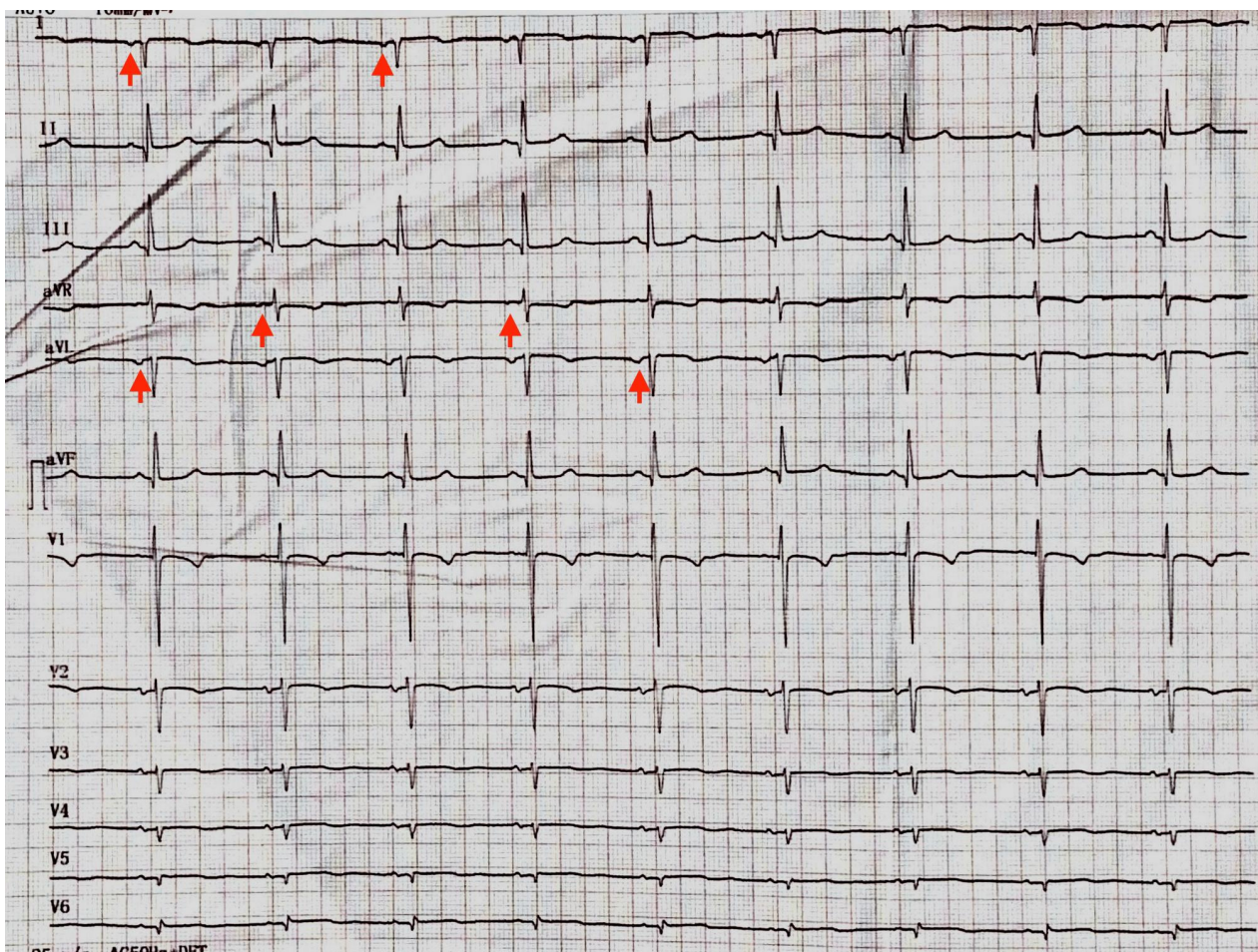
CASE 70 | PACING AND SPECIAL SETTINGS

Dextrocardia: when the heart points right

PUBLISHED CASE

A 60-year-old woman undergoes assessment before thyroid surgery. Heart sounds are louder on the right. Chest imaging confirms a right-sided cardiac apex and situs inversus.

What distinguishes this from an isolated arm-lead swap?



Solórzano CE et al. · Figure 1 · CC BY 4.0

Three findings to recognise

1

Lead I has a predominantly negative QRS.

2

R waves diminish across the usual left-sided chest leads.

3

The small lateral complexes match a heart directed away from them.

Remember: Reversed limb forces plus reversed chest progression: check the anatomy.

References: V4-ADV-DEXTRO, C02

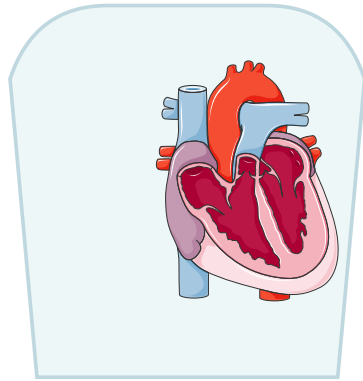
CASE 70 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Dextrocardia with situs inversus

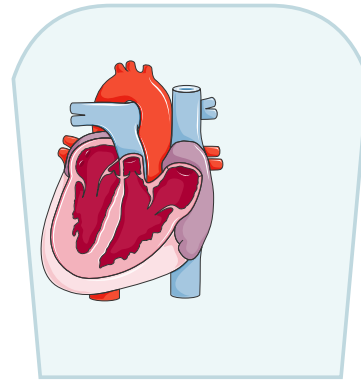
The leads must be interpreted around the actual heart

Usual position



Apex towards patient's left

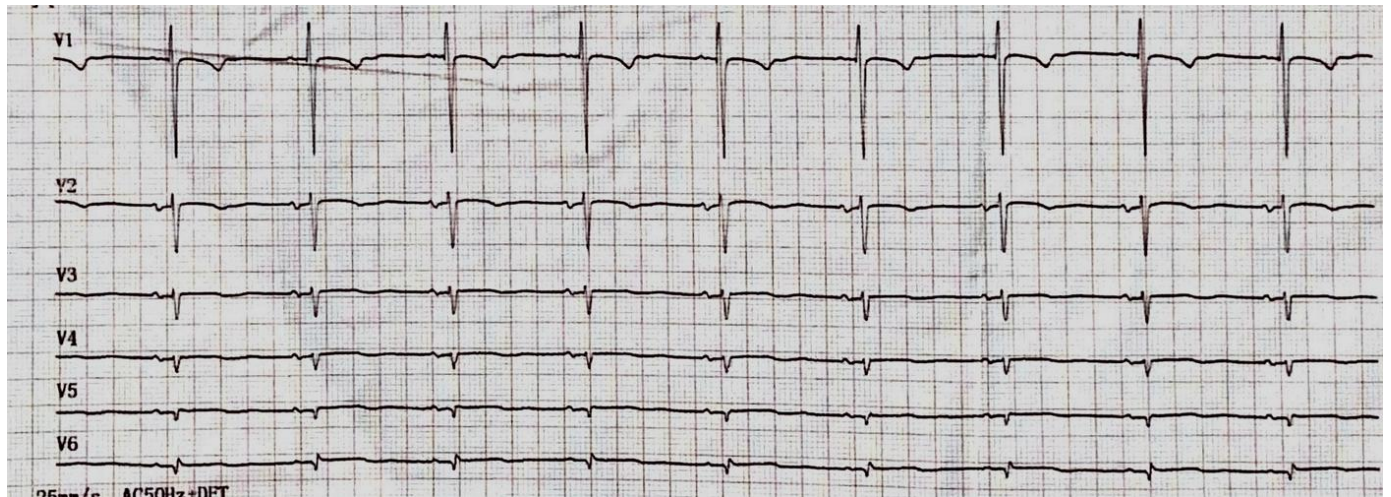
Mirror-image dextrocardia



Apex towards patient's right

Mirror-image anatomy changes limb-lead direction and left-sided chest-lead progression.

- Mirror-image anatomy directs ventricular forces towards the right chest.
- Conventional left-sided chest electrodes face away from much of this activity.
- Right-sided chest recording restores a useful view of the ventricular forces.



Solórzano CE et al. · Figure 1 · CC BY 4.0

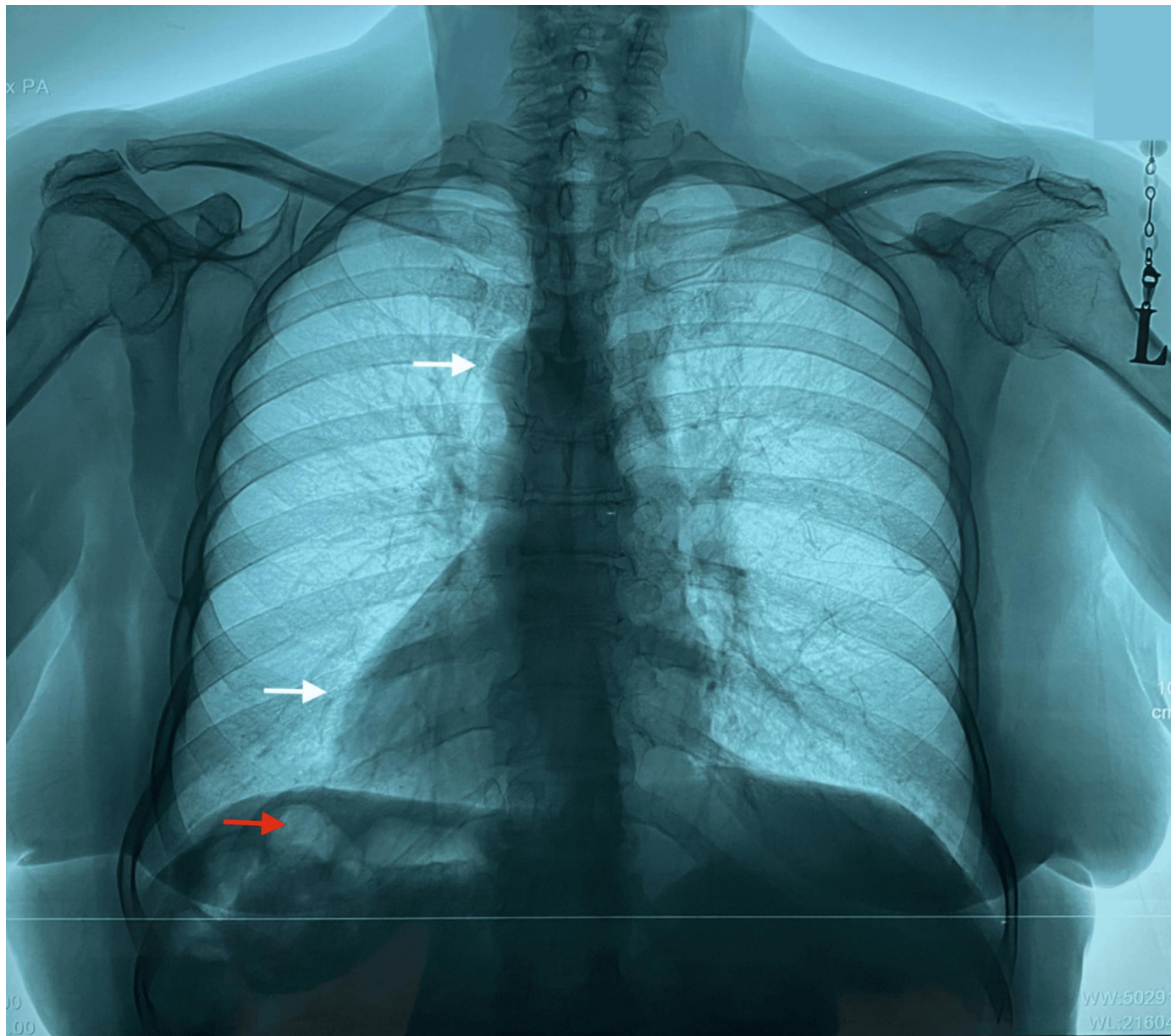
In practice

- Check electrode placement and examine the position of the cardiac apex.
- Confirm anatomy with appropriate imaging.
- Obtain a clearly labelled mirror-image ECG using the local acquisition protocol.

Avoid. Poor R-wave progression alone is not diagnostic of dextrocardia.

CASE 70 | CONNECT ECG AND ANATOMY

The ECG matches the right-sided cardiac apex



The left-side marker orients this radiograph; the cardiac apex and gastric bubble lie on the patient's right.

- The L marker identifies the patient's left, on the image's right.
- White arrows show the right-sided aortic contour and cardiac apex.
- The red arrow marks a right-sided gastric bubble.

Solórzano CE and Leyva Matos O (2025), Figure 2, CC BY 4.0; proportionally resized.

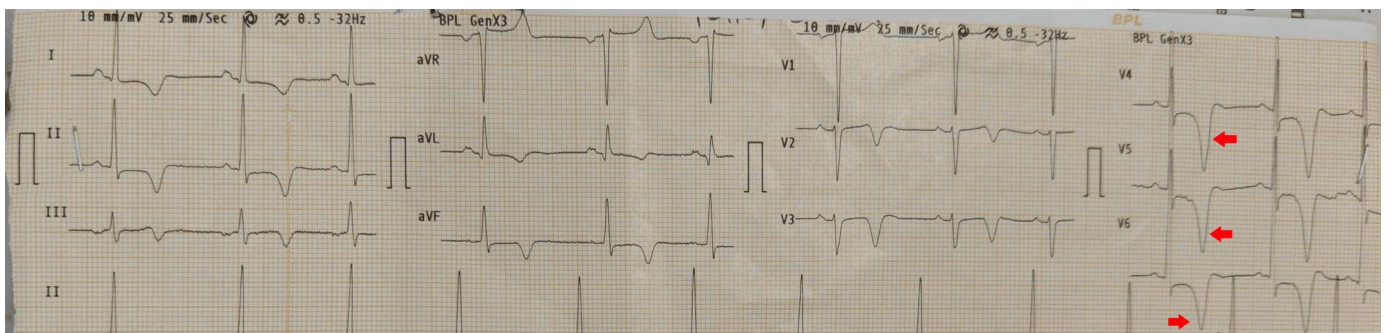
CASE 71 | PACING AND SPECIAL SETTINGS

Deep T inversion: look at the ventricular apex

PUBLISHED CASE

A 46-year-old woman presents with chest pain. Deep lateral T-wave inversion prompts an acute coronary assessment. Subsequent echocardiography demonstrates apical hypertrophy; coronary angiography shows no obstructive disease.

Which structural diagnosis can produce this repolarisation pattern?



Shirbhat L · Figure 1 · CC BY 4.0



Shirbhat L · Figure 1 · CC BY 4.0

Three findings to recognise**1**

T waves are deeply inverted in V4–V6.

2

The abnormalities involve adjacent lateral chest leads.

3

The ECG suggests a structural or ischaemic problem; it cannot settle which.

Remember: The T wave points to a question; imaging answers it.

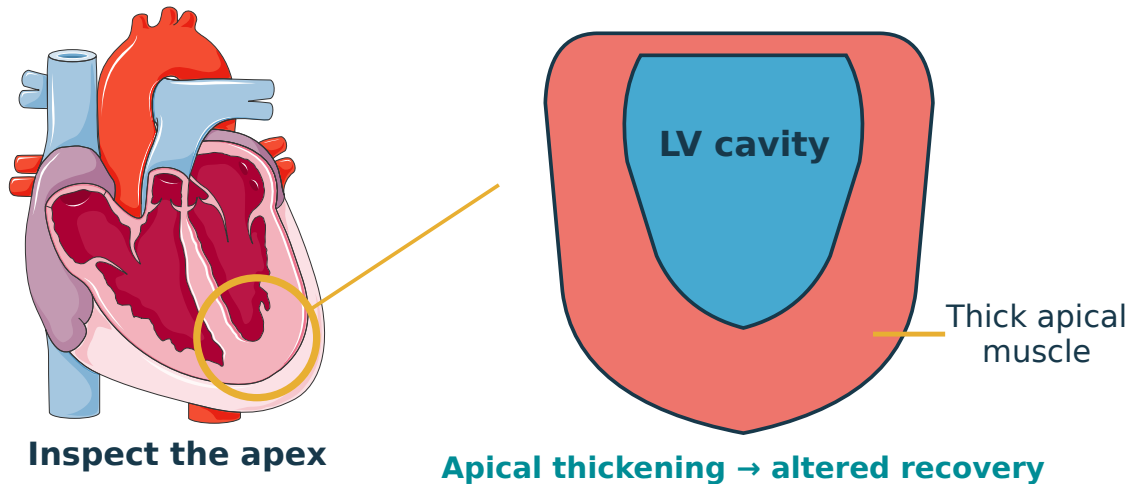
References: V4-ADV-HCM, V4-ADV-ESC-CM, C11

CASE 71 | UNDERSTAND AND APPLY

From anatomy to clinical meaning

Apical hypertrophic cardiomyopathy

Apical hypertrophy changes ventricular recovery



Apical hypertrophy can produce deep T-wave inversion; imaging establishes the anatomical diagnosis.

- Hypertrophied apical myocardium changes ventricular activation and recovery.
- Abnormal recovery can produce deep precordial T-wave inversion.
- Imaging defines the hypertrophy and any associated obstruction or scar.



Shirbhate L · Figure 1 · CC BY 4.0

In practice

- Assess acute chest pain for coronary disease using the appropriate pathway.
- Use echocardiography; consider cardiac MRI when the apex is unclear.
- Arrange cardiomyopathy assessment, family evaluation and individual sudden-death risk assessment.

Avoid. Deep symmetrical T inversion is not specific to apical hypertrophic cardiomyopathy.

CASE 71 | CONNECT ECG AND ANATOMY

The ECG clue leads to structural imaging



Source long-axis view: arrows highlight thick apical myocardium around a narrowed left-ventricular cavity.

- The left-ventricular cavity narrows towards the apex, at image left.
- Source arrows mark the reported apical myocardial thickening.
- This structural finding complements the deep lateral T-wave inversion.

Shirbhate L (2026), Figure 3, CC BY 4.0; proportionally resized.

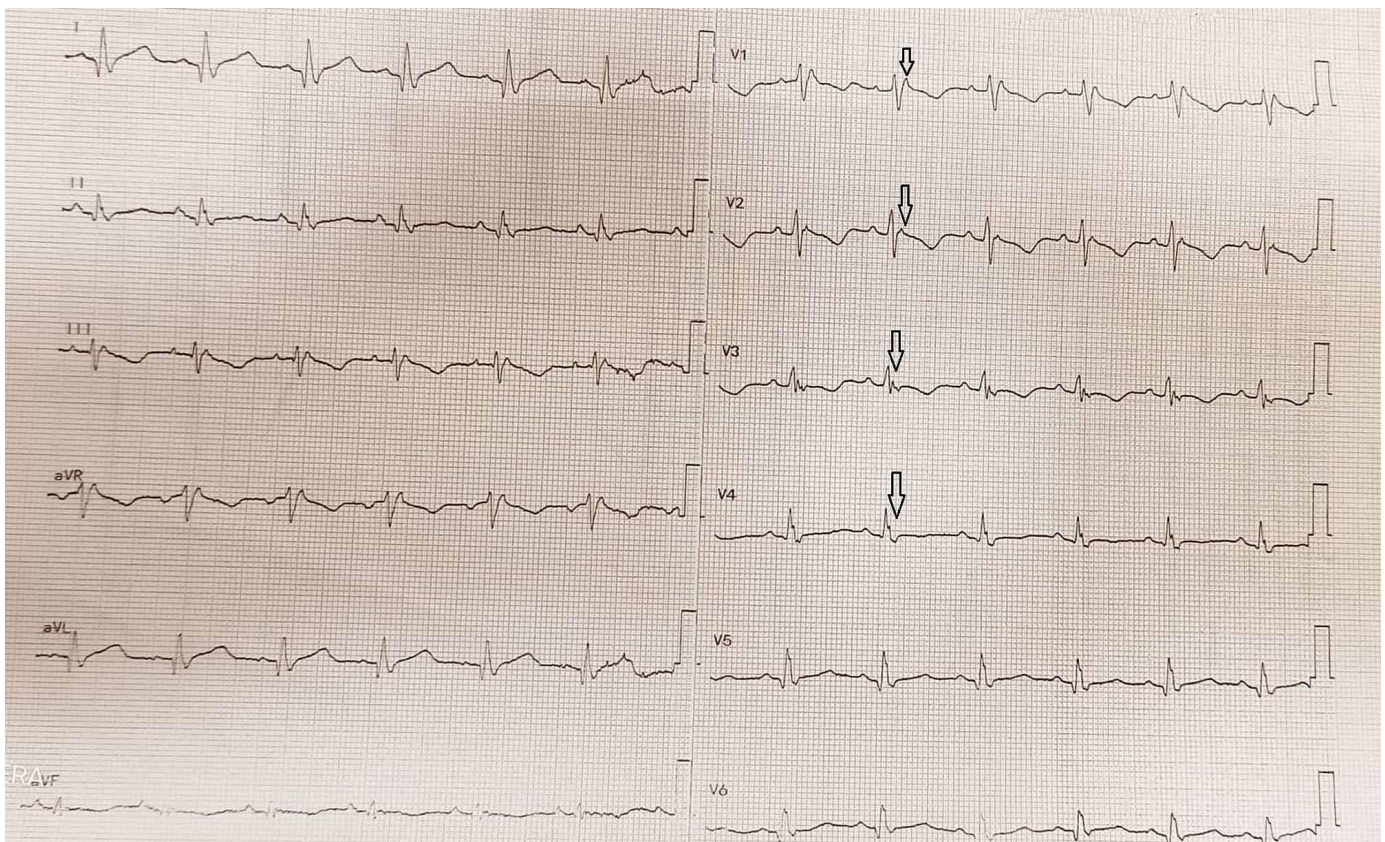
CASE 72 | PACING AND SPECIAL SETTINGS

Arrhythmogenic cardiomyopathy: ECG and structure

PUBLISHED CASE

A 30-year-old man presents with palpitations and recurrent unstable ventricular tachycardia. After stabilisation, the ECG is abnormal in V1-V4. Imaging shows severe right-ventricular disease.

What links delayed activation to the ventricular arrhythmia?



Mushtaque RS et al. · Figure 1 · CC BY 4.0

Three findings to recognise

1

T waves are inverted in the right-precordial leads.

2

Source arrows identify small late deflections around the QRS end.

3

The reported diagnosis also depends on ventricular imaging and clinical findings.

Remember: Combine the rhythm, the right ventricle and the family.

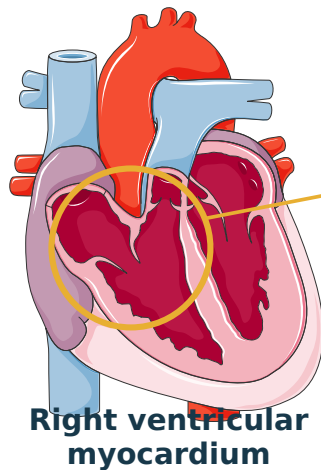
References: V4-ADV-ARVC, V4-ADV-ESC-CM, C11

CASE 72 | UNDERSTAND AND APPLY

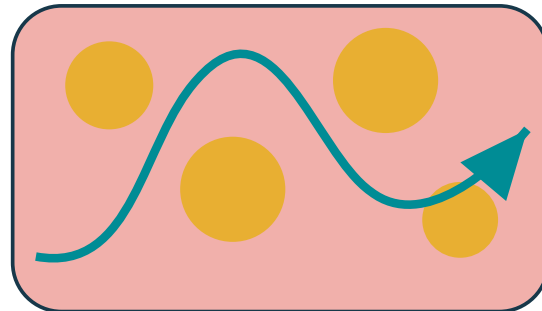
From anatomy to clinical meaning

Arrhythmogenic right-ventricular cardiomyopathy

A patchy substrate delays activation



Patchy myocardial replacement

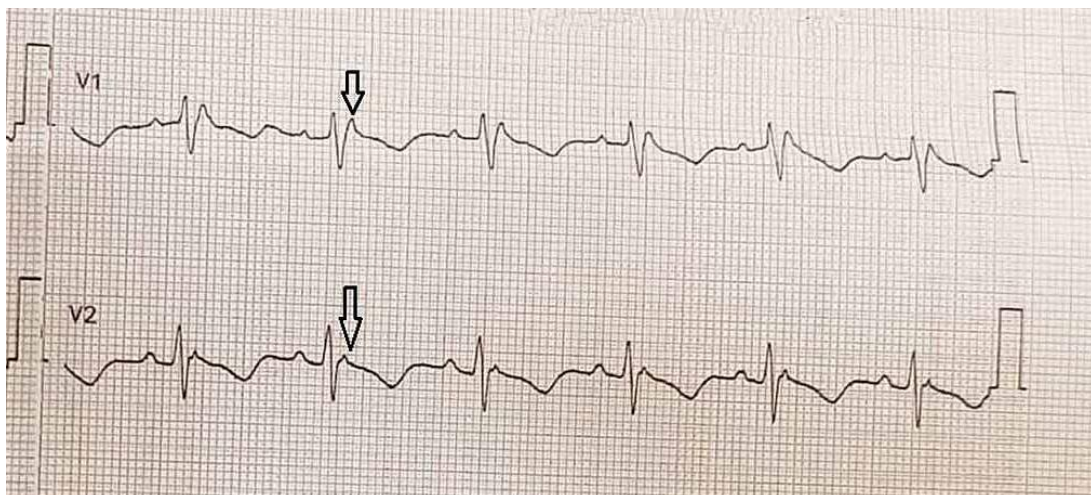


Slow surviving conduction paths

Late potentials and re-entry can result

Patchy myocardial replacement can create delayed activation and re-entrant ventricular arrhythmia.

- Patchy myocardial replacement can slow and fragment activation.
- Late local activation may appear as a small post-QRS deflection.
- Surviving pathways through diseased tissue can support re-entry.



Mushtaque RS et al. · Figure 1 · CC BY 4.0

In practice

- Treat unstable ventricular tachycardia immediately using the resuscitation pathway.
- Arrange specialist imaging, rhythm assessment and evaluation for diagnostic mimics.
- Discuss exercise, inherited-disease testing and family screening with a specialist team.

Avoid. An epsilon-like deflection alone is unreliable; neither its presence nor absence establishes the diagnosis.

QUICK REFERENCE

Measurements that matter



PTB-XL 00033 | 0.35-5.35 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
PTB-XL | Wagner et al. | CC BY 4.0

Measure	Adult reference	How to use it
Grid	25 mm/s; 10 mm/mV	Small square: 40 ms × 0.1 mV. At 50 mm/s: 20 ms.
Resting rate	Usually 60–100/min	Regular rhythm: 60/RR (seconds). Sleep and training can lower rate.
PR interval	Usually 120–200 ms	P onset to QRS onset. >200 ms with every P conducted: first-degree AV block.
QRS duration	Usually ≤110 ms	Earliest onset to latest offset. Complete BBB: ≥120 ms plus its characteristic morphology.
QTc	Review ≥450 ms (men); ≥460 ms (women)	QRS onset to T end. Exclude a distinct U wave; state the correction formula.

QTc screening thresholds alone do not diagnose congenital long-QT syndrome. Children need age-specific ranges. With a wide QRS or ventricular pacing, consider QRS-adjusted QT or JT; delayed activation itself lengthens QT.

ECG CRITERIA AT A GLANCE

ST elevation: use the correct J-point threshold



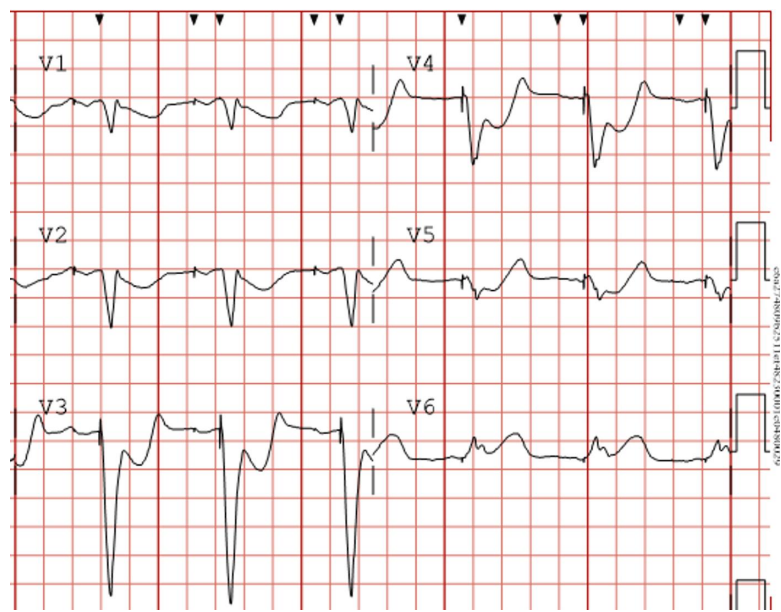
PTB-XL 01063 | 0-5 s | 25 mm/s, 10 mm/mV proportions
Square grid: 40 ms x 0.1 mV | Calibration: 1 mV, 200 ms | Uniform zoom
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Leads / group	J-point elevation
V2-V3: men younger than 40	≥2.5 mm
V2-V3: men aged 40 or older	≥2.0 mm
V2-V3: women	≥1.5 mm
Other standard leads	≥1.0 mm

Interpret in context. At 10 mm/mV, require two contiguous leads and compatible symptoms, without confounding LVH/BBB. Subthreshold changes do not exclude occlusion.

ECG CRITERIA AT A GLANCE

Modified Sgarbossa: three warning patterns



Precordial leads V1-V6. O’Rorke et al. · Figure 1 · CC BY 4.0

Criterion	What to identify
Concordant ST elevation	≥1 mm elevation with a predominantly positive QRS
Concordant ST depression	≥1 mm depression in V1-V3
Excessively discordant ST elevation	Negative QRS: ≥1 mm elevation and ≥25% of S depth ($ST/S \leq -0.25$)

Interpret in context. In symptomatic LBBB or ventricular pacing, any criterion is concerning. Negative criteria do not exclude coronary occlusion.

REFERENCE NOTES

Sources and image credits

Recorded ECG data

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Anatomical illustrations

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Editorial note

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Published clinical ECG figures

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