A COMPLETE BEDSIDE REFERENCE

ECG Complete Guide From Basics to Advanced

Reading the electrocardiogram from first principles to confident diagnosis — with annotated rhythm strips, infographics and bedside checklists.



Prepared for Dr. Seema Parvin, MBBS
General Physician

October 2026

Disclaimer — please read

This guide is an **educational resource**. It is not a substitute for clinical judgment, formal cardiology training, or institutional protocols. Always correlate the ECG with the patient's history, examination and prior tracings, and seek senior/cardiology input for any tracing you are unsure about — especially suspected acute coronary occlusion, unstable arrhythmias and paediatric ECGs. Drug doses and emergency algorithms change; verify against your hospital's current resuscitation protocol.

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CHAPTER 1

What the ECG Records

The electrocardiogram (ECG or EKG) is a recording of the heart's **electrical activity** as seen from the body surface. It does not directly show contraction, blood flow or anatomy — it shows the waves of **depolarization** (electrical activation) and **repolarization** (electrical recovery) sweeping through cardiac muscle. Because mechanical contraction follows electrical activation, the ECG is our best bedside window into rhythm, conduction and ischaemia.

The cardiac dipole

At rest, each cardiac muscle cell is polarized: negative inside, positive outside. When a wave of depolarization passes, the boundary between depolarized and resting tissue forms a **dipole** — a separation of charge with a positive and a negative pole. The ECG lead records a **positive (upward) deflection** when the wave of depolarization travels *toward* the lead's positive electrode, and a **negative (downward) deflection** when it travels *away*. Repolarization produces deflections of the opposite polarity to the direction of travel, because the charge orientation is reversed. This single principle explains the shape of every wave you will ever see.

Depolarization and repolarization in sequence

A normal heartbeat begins in the sinoatrial (SA) node. Atrial depolarization produces the **P wave** (small, because the atria are thin). The impulse pauses briefly at the atrioventricular (AV) node, then races down the His–Purkinje system to depolarize the ventricles almost simultaneously — the tall, sharp **QRS complex** (large, because ventricular mass is large). Ventricular repolarization follows as the **T wave**. Atrial repolarization also occurs, but it is buried inside the QRS and invisible.

The 12-lead concept

One lead is one "electrical viewpoint." The standard 12-lead ECG uses ten electrodes to create **twelve viewpoints**: six limb leads looking at the heart in the frontal (vertical) plane and six chest leads looking in the horizontal (transverse) plane. Together they surround the heart, so an electrical event — an infarct, a chamber enlargement, a conduction block — casts a characteristic shadow across specific leads. Learning the ECG is largely learning to read these shadows (Chapter 3 and Chapter 14).

Indications: when to record an ECG

- **Chest pain or suspected acute coronary syndrome** — record within 10 minutes of arrival; repeat if pain persists or evolves.
- **Palpitations, syncope, presyncope or documented arrhythmia** — including during symptoms if possible.
- **Dyspnoea, heart failure or unexplained hypotension/shock.**
- **Before starting drugs** that affect conduction or the QT interval (e.g. antiarrhythmics, antipsychotics, macrolides), and for monitoring on them.
- **Electrolyte disturbances** — especially potassium and calcium disorders.

- **Pre-operative assessment** in at-risk patients, and **screening** in athletes or family members of inherited arrhythmia syndromes when indicated.
- **Any critically ill patient** — continuous monitoring plus a 12-lead tracing.



Figure 1.1 — ECG grid. At the standard paper speed of 25 mm/s, horizontal distance = time (1 small square = 40 ms; 1 large square = 200 ms) and vertical distance = voltage (1 small square = 0.1 mV; calibration pulse = 1 mV).

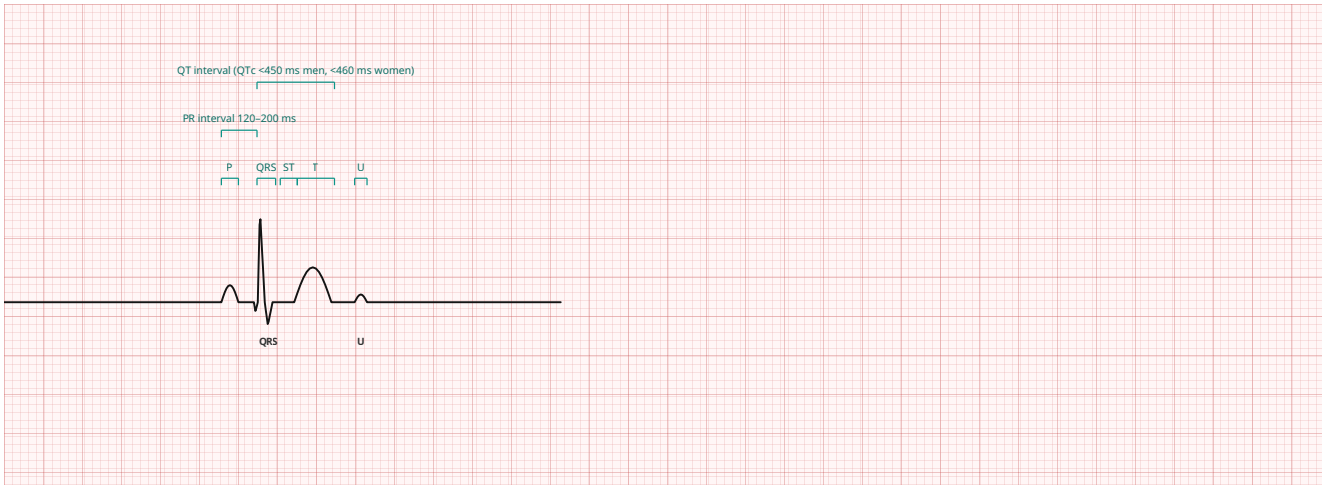


Figure 1.2 — The normal ECG waveform and key measurements. P wave = atrial depolarization (<120 ms wide, <2.5 mm tall); QRS = ventricular depolarization (<120 ms); ST segment is normally isoelectric; T wave = ventricular repolarization; U wave = late Purkinje repolarization (normal variant). PR interval 120–200 ms; QTc <450 ms (men) / <460 ms (women).

Clinical Pearl

The ECG is a map of **electrical vectors**, not anatomy. A “normal” ECG never excludes heart disease (e.g. unstable angina often has a normal resting ECG), and an “abnormal” ECG must always be read in the context of the patient in front of you.

Red Flag

Never let a normal ECG overrule a convincing history of cardiac-sounding chest pain. Serial ECGs and troponins are the safety net — ischaemia is dynamic.

CHAPTER 2

The Cardiac Conduction System

The heart beats on its own because specialized pacemaker cells generate rhythmic electrical impulses. Understanding the conduction hierarchy — who fires, who conducts, and who takes over when the leader fails — is the foundation of every rhythm diagnosis.

The normal sequence

1. Sinoatrial (SA) node, high in the right atrium near the superior vena cava: the dominant pacemaker, firing at **60–100/min** at rest. Sympathetic stimulation speeds it; vagal (parasympathetic) stimulation slows it. The impulse spreads across both atria (P wave) and converges on the AV node.

2. Atrioventricular (AV) node, in the low interatrial septum: the gatekeeper. Conduction slows here (≈0.12 s delay), giving the ventricles time to fill. Its intrinsic rate is **40–60/min**.

3. Bundle of His, penetrating the fibrous skeleton into the ventricles — the only normal electrical connection between atria and ventricles.

4. Right and left bundle branches, running down either side of the interventricular septum; the left bundle divides further into anterior and posterior fascicles.

5. Purkinje fibres, the fine terminal network that delivers the impulse to ventricular muscle almost simultaneously, producing a narrow QRS. Ventricular escape rhythm is **20–40/min**.

Why intrinsic rates matter

Faster pacemakers suppress slower ones (overdrive suppression). If the SA node fails or its impulses cannot reach the ventricles — as in complete heart block — a lower pacemaker escapes and takes over at its own slower intrinsic rate. That is why a junctional escape rhythm runs at 40–60/min and a ventricular escape rhythm at 20–40/min: the rate itself tells you *where* the rhythm is coming from. A regular bradycardia at 45/min with no preceding P waves is behaving exactly like a junctional escape; a wide-complex regular rhythm at 30/min is behaving like a ventricular escape.

Autonomic and drug influences

Sympathetic drive (exercise, fever, pain, anxiety, thyrotoxicosis, β-agonists) accelerates the SA node and AV conduction. Vagal drive (sleep, athletes, vomiting, carotid pressure) and drugs such as β-blockers, non-dihydropyridine calcium-channel blockers (verapamil, diltiazem), digoxin and amiodarone slow the SA node and prolong AV nodal conduction — the pharmacologic basis of both therapy and toxicity.

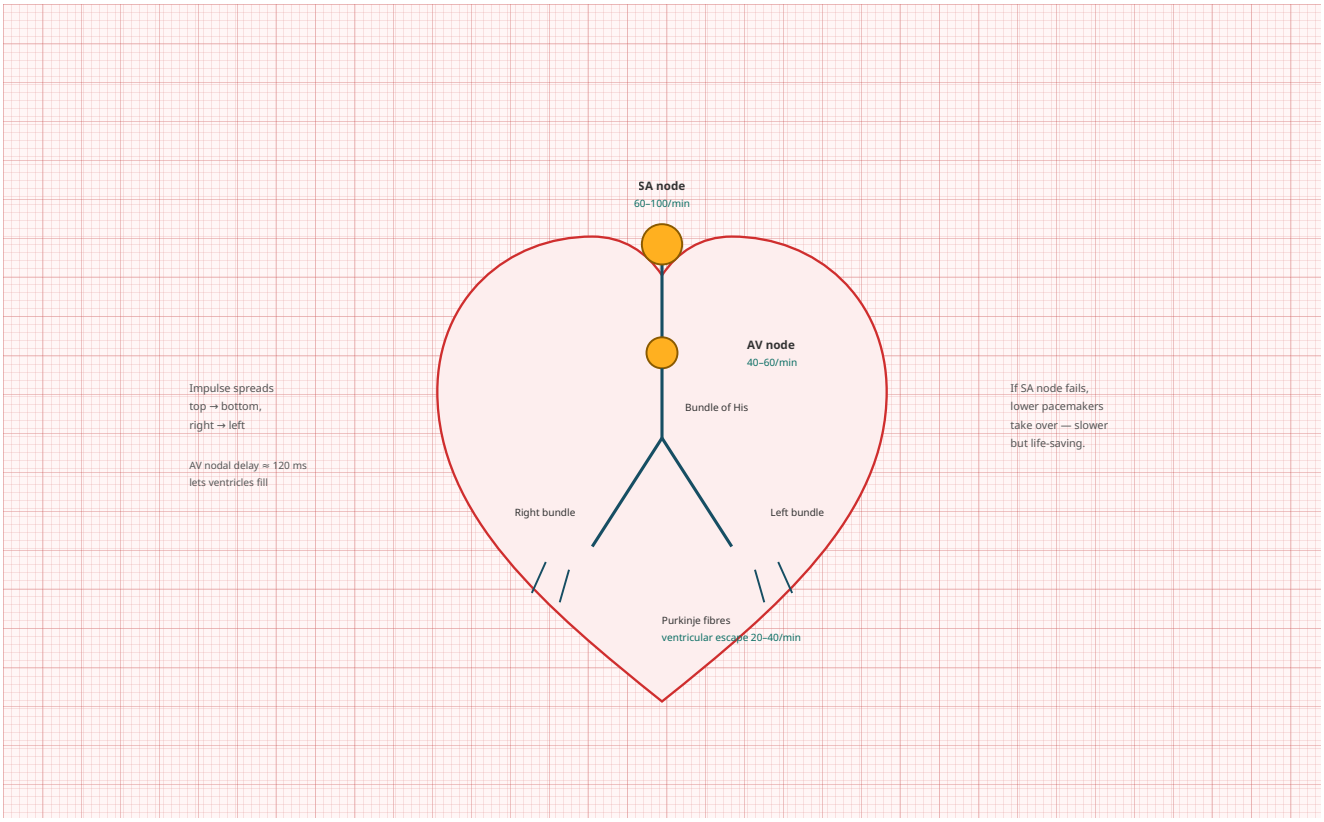


Figure 2.1 — Cardiac conduction system and intrinsic rates. The SA node is the normal pacemaker (60–100/min). Each lower level has a slower intrinsic rate and acts as a backup if the level above fails.

Clinical Pearl

When you see a slow regular rhythm, ask: *what is the rate, and are there P waves married to each QRS?* The intrinsic-rate ladder (60–100 → 40–60 → 20–40) localizes the pacemaker before you measure anything else.

CHAPTER 3

The 12-Lead System

Twelve leads, ten electrodes, two planes. Once you can picture where each lead "looks from," localization becomes intuitive.

Limb leads (frontal plane)

Electrodes go on the right arm (RA), left arm (LA) and left leg (LL); the right leg is an earth/ground. The three **bipolar limb leads** record the voltage difference between two limbs:

Lead	Positive pole → negative pole	Views
I	LA (+) − RA (−)	Lateral wall, left–right axis (0°)
II	LL (+) − RA (−)	Inferior wall (+60°); best P-wave lead
III	LL (+) − LA (−)	Inferior wall (+120°)

The three **augmented unipolar leads** compare one limb against the average of the other two (Goldberger's central terminal), "augmenting" the small signal:

Lead	Positive electrode	Views
aVR	Right arm (−30°)	Right upper; normally negative — a useful polarity check
aVL	Left arm (−30°)	High lateral wall
aVF	Left leg/foot (+90°)	Inferior wall

Einthoven's triangle and Einthoven's law

The three limb electrodes form **Einthoven's triangle** around the heart. Because lead II = lead I + lead III (Einthoven's law), the limb leads are mutually redundant — a sanity check: if the math does not roughly hold, suspect lead misplacement or artefact.

Wilson central terminal

The six **unipolar chest leads** each compare one chest electrode against **Wilson's central terminal** — the average of RA + LA + LL, which approximates zero potential at the body's electrical centre. They view the heart in the horizontal plane, sweeping from the right ventricle (V1) around to the lateral left ventricle (V6).

Chest lead positions (memorize these)

Lead	Position
V1	4th intercostal space, right sternal edge
V2	4th intercostal space, left sternal edge
V3	Midway between V2 and V4
V4	5th intercostal space, left midclavicular line
V5	Left anterior axillary line, same horizontal level as V4
V6	Left midaxillary line, same horizontal level as V4

Right-sided and posterior leads

V4R (mirror of V4 on the right chest) is recorded when **inferior STEMI** is present to detect **right ventricular infarction** (ST elevation ≥1 mm in V4R) — critical because RV infarct needs fluids, not nitrates. **Posterior leads V7–V9** (posterior axillary line, scapular tip line, paraspinal line, same level as V6) uncover **posterior MI**, which hides as ST *depression* in V1–V3 on the standard tracing (ST elevation ≥0.5 mm in V7–V9, or ≥1 mm in men under 40, confirms it).

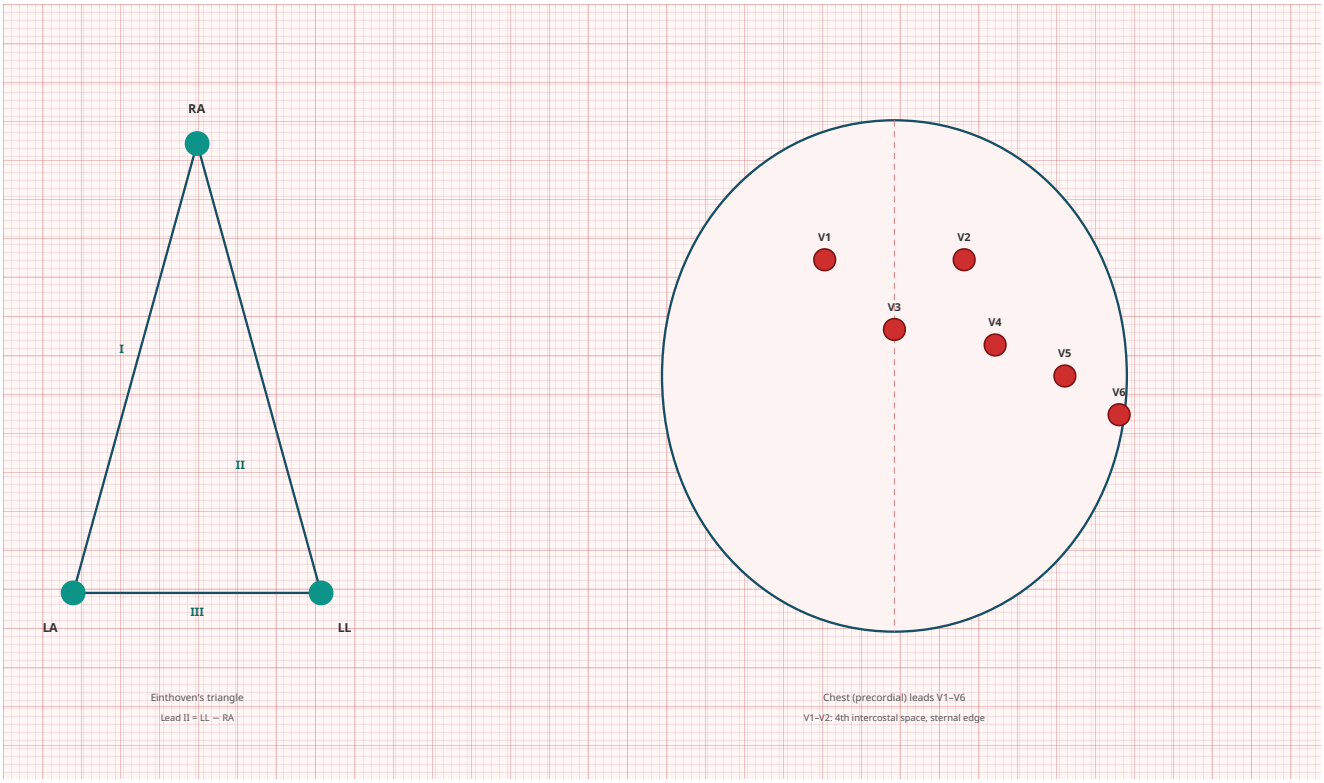


Figure 3.1 — Limb leads (Einthoven's triangle) and precordial lead positions. Limb leads record the frontal plane; chest leads record the horizontal plane. Together they give a 3-D electrical view of the heart.

Clinical Pearl

Lead II is roughly parallel to the normal P-wave vector, so P waves are tallest and most visible in lead II — always check rhythm there first. aVR points at the cardiac “cave” (right upper shoulder) and is normally negative; if aVR is broadly positive, think dextrocardia or limb-lead reversal (Chapter 20).

Red Flag

In every inferior STEMI (STE in II, III, aVF), record V4R. Missing an RV infarct — then giving nitrates or diuretics — can precipitate catastrophic hypotension.

CHAPTER 4

ECG Paper and Calibration

Before interpreting a single complex, confirm the recording conditions. A misread calibration or paper speed silently corrupts every measurement that follows.

Paper speed: 25 mm/s

The international standard is **25 mm per second**. On this grid:

Unit	Size	Time at 25 mm/s
1 small square	1 mm	**40 ms** (0.04 s)
1 large square	5 mm (5 small)	**200 ms** (0.2 s)
5 large squares	25 mm	**1 second**

If the machine was set to 50 mm/s (sometimes used for detailed measurement), everything is stretched — halves all your time readings unless you correct. Always glance at the speed printed on the tracing.

Calibration: 10 mm/mV

Standard gain is **10 mm per millivolt**, so 1 small square vertically = 0.1 mV and 1 large square = 0.5 mV. The rectangular **calibration pulse** at the start of the tracing should be 10 mm tall (1 mV). If gain was halved (5 mm/mV, e.g. very tall QRS complexes), voltage criteria such as LVH lose their meaning.

Heart-rate calculation

Method 1 — the 300-150-100-75-60-50 sequence (regular rhythms). Count large squares between two consecutive R waves: 1 large square = 300/min, 2 = 150, 3 = 100, 4 = 75, 5 = 60, 6 = 50. Each small square refines the estimate (e.g. 3.5 large squares ≈ 85/min).

Method 2 — the 6-second strip (any rhythm, including irregular). Count QRS complexes in 6 seconds (30 large squares, usually marked) and multiply by 10. For atrial fibrillation, count over a full 6 seconds — never trust a single R–R interval.

Method 3 — the 1500 method. Count small squares between R waves and divide 1500 by that number (1500 small squares = 1 minute).

Checklist before you read

- Patient name, date/time correct? Right patient?
- Paper speed 25 mm/s and calibration 10 mm/mV?
- Baseline stable, or wandering (breathing, tremor, poor contact)?
- All 12 leads present and labelled?
- Compare with the **old ECG** — the single most valuable step in ischaemia assessment.

Clinical Pearl

Tachycardia shortcut: if the R–R interval is under 3 large squares (600 ms), the rate is over 100/min. Under 2 large squares (400 ms) — over 150/min, think SVT/VT until proven otherwise.

CHAPTER 5

The Normal Waveform: Waves and Intervals

Every deflection has a cellular meaning. Learn the normal limits cold — pathology is simply these numbers exceeded, in recognizable patterns.

Waves

P wave — atrial depolarization. Small, smooth, upright in leads I, II and aVF; inverted in aVR. Limits: **duration <120 ms** (3 small squares) and **height <2.5 mm** (2.5 small squares). Best seen in lead II. A P wave before every QRS with a QRS after every P = sinus rhythm until proven otherwise.

QRS complex — ventricular depolarization. Naming follows strict rules: the first negative deflection is **Q**, the first positive deflection is **R** (a second positive is R′, read "R prime"), and any negative deflection after an R is **S**. Case matters: a small deflection gets a lowercase letter (qRs), a tall one uppercase. Duration **<120 ms** (3 small squares) means the ventricles depolarized via the fast His–Purkinje system; ≥120 ms means abnormal ventricular activation (bundle branch block, ventricular origin, pre-excitation or hyperkalaemia). A **pathologic Q wave** — evidence of dead, electrically silent myocardium — is **>40 ms wide** or **>25% of the following R-wave height**, in ≥2 contiguous leads. (Small septal q waves in I, aVL, V5–V6 are normal.)

ST segment — early ventricular repolarization. Normally **isoelectric** (flat at the baseline) between the end of the S wave (J point) and the start of the T wave. Elevation or depression here is the hallmark of acute ischaemia and pericarditis — measured at the J point + 60–80 ms.

T wave — ventricular repolarization. Broad, asymmetric (gentler upslope, steeper downslope), concordant with the QRS (same direction) in most leads; inverted in aVR and often in V1. Tall/peaked T waves suggest hyperkalaemia or hyperacute ischaemia; flat or inverted T waves suggest ischaemia, hypokalaemia or strain.

U wave — a small deflection after the T, same direction, thought to reflect Purkinje repolarization. Prominent in **hypokalaemia**, bradycardia and hypothermia; a normal variant in young people.

Intervals and segments

Interval	Measured	Normal	Meaning
PR interval	Start of P to start of QRS	**120–200 ms** (3–5 small squares)	Atrial depolarization + AV nodal delay
QRS duration	Start of Q to end of S	**<120 ms** (<3 small squares)	Ventricular depolarization time
QT interval	Start of Q to end of T	Rate-dependent; **QTc <450 ms men, <460 ms women**	Total ventricular electrical systole

Correcting the QT: Bazett's formula

QT shortens as heart rate rises, so it must be rate-corrected: **QTc = QT / √RR**, with QT and RR in seconds. Example: QT 400 ms at RR 800 ms (75/min) → QTc = 0.40 / √0.80 ≈ 447 ms — borderline in a man, normal in a woman. Bazett overcorrects at fast rates and undercorrects at slow ones, but it remains the bedside

standard. A prolonged QTc (>500 ms is high-risk) warns of **torsades de pointes**, especially with QT-prolonging drugs or hypokalaemia/hypomagnesaemia.

> **Clinical Pearl**
>
> Measure intervals in the lead where the wave is clearest, and always recheck the QT in a second lead — U waves and flat T waves make the T-end notoriously easy to misjudge. When in doubt, the longest QT you can defend is the one to use.

CHAPTER 6

The Electrical Axis

The **electrical axis** is the net direction of ventricular depolarization — the average of all instantaneous QRS vectors. It is reported as an angle in the frontal plane, measured from lead I (0°), with inferior angles positive.

Normal ranges

Axis	Range	Quadrant (I / aVF)
Normal	−30° to +90°	I positive, aVF positive
Left axis deviation (LAD)	−30° to −90°	I positive, aVF negative
Right axis deviation (RAD)	+90° to +180°	I negative, aVF positive
Extreme ("northwest") axis	−90° to ±180°	I negative, aVF negative

The quadrant method (bedside shortcut)

Look at the **net QRS deflection** (is it mostly up or mostly down?) in leads **I** and **aVF** only. The table above gives the answer in seconds. To refine within the normal range, find the most isoelectric limb lead — the axis is perpendicular to it.

Causes of axis deviation

Left axis deviation: left anterior fascicular block (commonest pathological cause), left ventricular hypertrophy, left bundle branch block, inferior MI (Q waves in inferior leads pull the axis left), hyperkalaemia, emphysema/ COPD (less common), and normal variant in the elderly and in pregnancy (horizontal heart).

Right axis deviation: right ventricular hypertrophy or strain (pulmonary embolism, chronic lung disease, pulmonary hypertension), right bundle branch block, left posterior fascicular block, lateral MI, and normal variant in children and young adults (vertical heart).

Extreme axis: consider **limb-lead reversal or dextrocardia** first (Chapter 20), then ventricular tachycardia or severe hyperkalaemia.

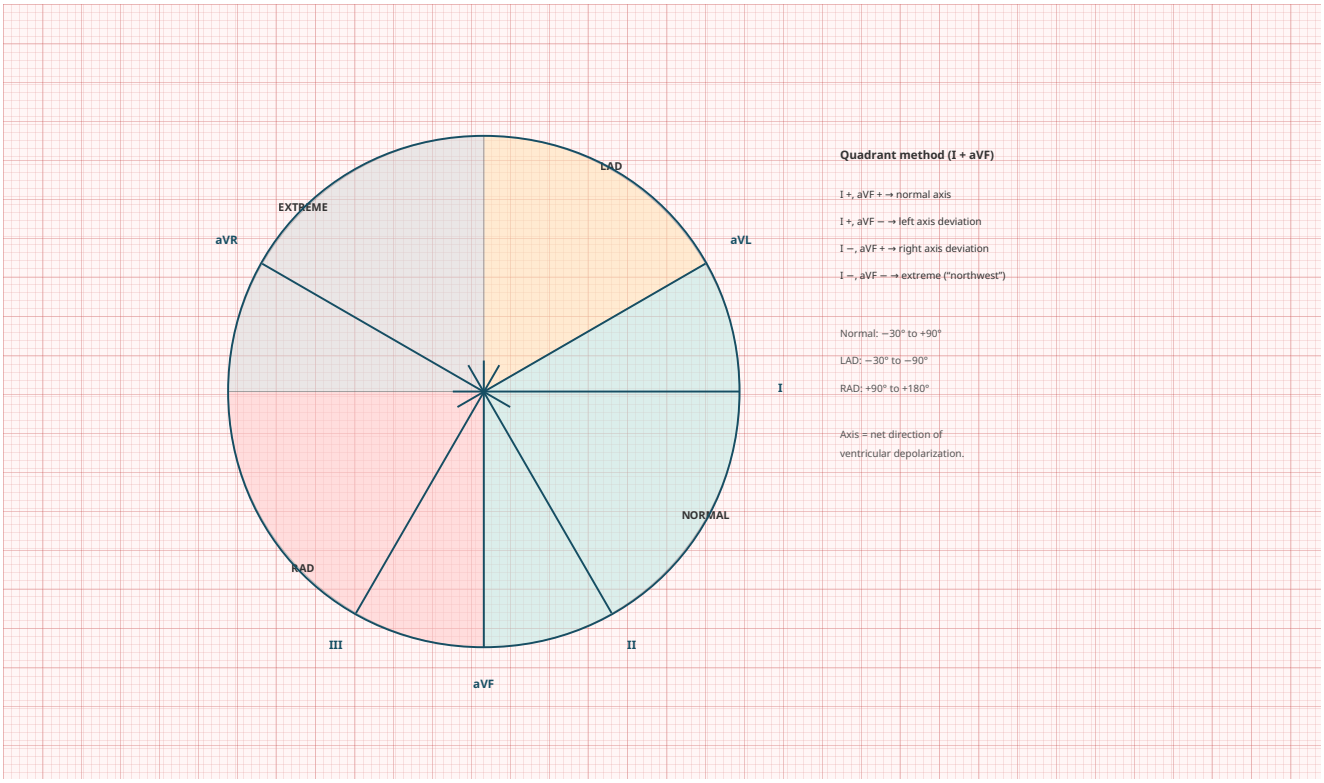


Figure 6.1 — Hexaxial reference system. The six limb leads are rearranged around a common centre. The QRS axis is the lead direction with the tallest net-positive QRS; the quadrant method (I and aVF) is the fastest bedside shortcut.

Clinical Pearl

New left axis deviation in a patient with chest pain points toward left anterior fascicular block from LAD ischaemia; new right axis deviation with tachycardia and T-wave inversion in V1–V4 raises pulmonary embolism. Axis is most powerful read as a *change* from the old ECG.

CHAPTER 7

Systematic 8-Step Interpretation

Read **every** ECG the same way, every time. A fixed sequence prevents the classic error of spotting one abnormality and stopping. Work through these eight steps in order, and compare with the old tracing at the end.

The 8-Step ECG Checklist

1. **Rate** — atrial and ventricular rates (300-150-100-75-60-50 or 6-second method). Bradycardia <60, tachycardia >100.
2. **Rhythm** — regular or irregular? P wave before every QRS, QRS after every P? If irregular: regularly irregular (patterned — e.g. Wenckebach) or irregularly irregular (e.g. AF)?
3. **P wave** — present? Morphology normal (upright in II, inverted in aVR)? One P per QRS? Atrial rate vs ventricular rate?
4. **PR interval** — 120–200 ms and constant? Prolonged, shortening, or progressively lengthening?
5. **QRS complex** — narrow (<120 ms) or wide? Morphology (RBBB/LBBB pattern? pathological Q? voltage — LVH criteria?).
6. **ST-T changes** — ST elevation or depression (≥2 contiguous leads)? T-wave peaking, flattening or inversion? Compare territories.
7. **QTc** — prolonged (>450 ms men / >460 ms women)? Shortened? Check drugs and electrolytes.
8. **Axis** — normal, LAD, RAD or extreme (quadrant method: I and aVF)?

Final step: put it together — *rate + rhythm + conduction + ST-T + QTc + axis + comparison with old ECG = diagnosis and plan.*

How the steps fit together

Steps 1–4 answer *"what is the rhythm?"* Steps 5–7 answer *"what is the conduction and repolarization doing?"* Step 8 adds *"where is the electricity going?"* Ischaemia lives mostly in step 6, blocks in steps 4–5, chamber enlargement in steps 5 and 8, and danger (QT, Brugada, hyperkalaemia) in steps 6–7. With practice the sequence takes under a minute — speed comes from order, not from skipping.

> **Clinical Pearl**
>
> Dictate or write your read in the 8-step order. A structured read is auditable, teachable, and far less likely to miss a second pathology hiding behind the first — e.g. AF *with* an inferior STEMI.

CHAPTER 8

Sinus Rhythms

Sinus rhythms originate in the SA node: every QRS is preceded by a normal P wave (upright in II, inverted in aVR), every P is followed by a QRS, and the PR interval is normal and constant. Only the **rate** differs.

Normal sinus rhythm

Criteria: regular rhythm, **60–100/min**, P before every QRS, QRS after every P, normal PR (120–200 ms), narrow QRS. Minor sinus arrhythmia (rate varying with breathing, common in the young) is still sinus rhythm.

Sinus bradycardia (<60/min)

Causes: athletic training and sleep (high vagal tone), hypothyroidism, hypothermia, raised intracranial pressure, and drugs — β-blockers, verapamil/diltiazem, digoxin, amiodarone. **Management:** treat the cause; asymptomatic bradycardia needs nothing. Symptomatic (hypotension, syncope, ischaemia, heart failure) → **atropine 0.5–1 mg IV** (up to 3 mg), then transcutaneous pacing or adrenaline/dopamine infusion while arranging **transvenous pacing** if unresponsive.

Sinus tachycardia (>100/min)

Sinus tachycardia is almost always **secondary** — it is the heart responding appropriately to demand or stress. **Causes:** pain, anxiety, fever, anaemia, hypovolaemia, dehydration, hyperthyroidism, pulmonary embolism, heart failure, sepsis, sympathomimetics (β-agonists, cocaine), caffeine and alcohol withdrawal. **Management:** find and treat the cause; do not "slow the sinus node" with β-blockers while the patient is bleeding or septic. A useful rule: *sinus tachycardia has a reason — your job is to find it.*

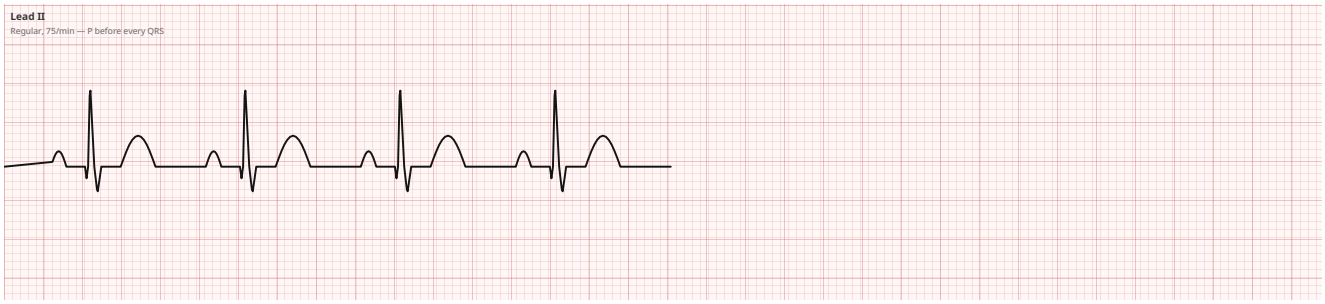


Figure 8.1 — Normal sinus rhythm. Regular at 75/min; normal P waves (upright in II), constant PR interval, narrow QRS.

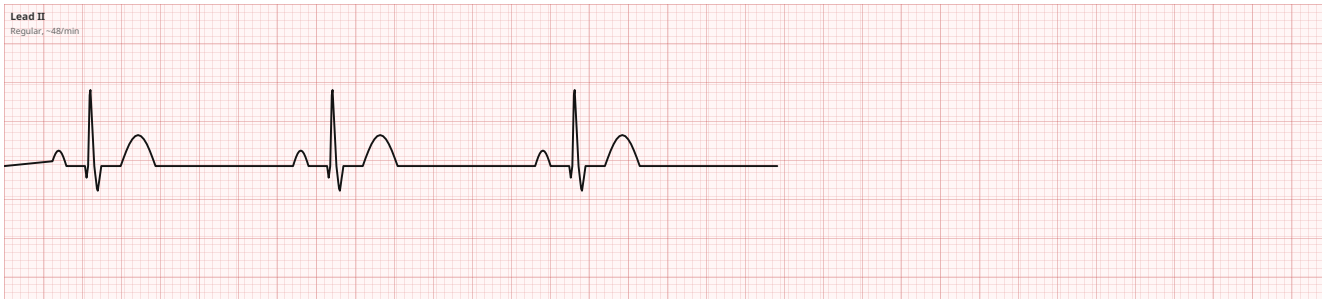


Figure 8.2 — Sinus bradycardia. Sinus morphology preserved; rate <60/min. Correlate with symptoms, drugs and thyroid status.

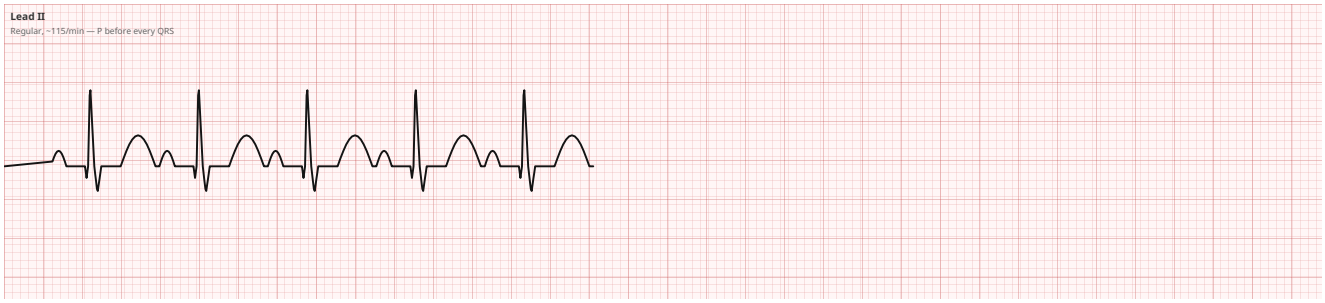


Figure 8.3 — Sinus tachycardia. Regular narrow-complex tachycardia with visible P waves. Seek the underlying cause (fever, volume loss, anaemia, PE, thyrotoxicosis).

Red Flag

A regular narrow-complex tachycardia at ~150/min **without visible P waves** is SVT, not sinus tachycardia. Gradual onset/offset favours sinus; abrupt onset/offset favours SVT.

CHAPTER 9

Atrioventricular (AV) Blocks

AV blocks are delays or failures of impulse transmission from atria to ventricles. Classify by **what happens to the PR interval** and **what happens to the QRS** — that determines danger and management.

First-degree AV block

PR interval >200 ms (5 small squares), but **constant**, with every P followed by a QRS. Usually benign — seen in athletes, with AV-nodal drugs, or ageing conduction tissue. No treatment; review drugs and monitor.

Second-degree AV block, Mobitz type I (Wenckebach)

Progressive PR lengthening until a P wave fails to conduct (dropped QRS), then the cycle restarts — **grouped beating**. The block is at the **AV node**, so the QRS is usually narrow and the rhythm, though grouped, is haemodynamically tolerated. Often transient (inferior MI, drugs, high vagal tone). Usually needs observation, not pacing — unless symptomatic.

Second-degree AV block, Mobitz type II

Fixed (constant) PR interval with **sudden, unexpected dropped QRS complexes**. The block is **infranodal** (His–Purkinje), so the QRS is usually **wide**. This is dangerous: it can progress without warning to complete heart block. **Needs pacing** (transcutaneous bridge → transvenous/permanent pacemaker), especially if symptomatic or with wide QRS.

2:1 AV block

Every second P wave conducts — cannot be classified as Mobitz I or II from a single tracing (need to see consecutive conducted PR intervals). Treat as **Mobitz II until proven otherwise** if the QRS is wide; if narrow, it may be nodal. When in doubt, pace-ready.

Third-degree (complete) AV block

Complete AV dissociation: P waves march through at their own (faster) atrial rate, QRS complexes escape at their own (slower) ventricular rate, with **no relationship** between them. The escape may be junctional (narrow QRS, 40–60/min — more stable) or ventricular (wide QRS, 20–40/min — unstable). Causes: inferior/anterior MI, drugs, calcific degeneration (Lenègre/Lev disease), Lyme carditis, post-cardiac surgery.

Management pointers

Situation	Action
Symptomatic bradycardia (hypotension, syncope, ischaemia, HF)	**Atropine 0.5–1 mg IV** (repeat to 3 mg max); if no response → **transcutaneous pacing** ± adrenaline or dopamine infusion; arrange **transvenous pacing**
Mobitz II / 2:1 with wide QRS / complete heart block	**Pacing indicated** — do not rely on atropine (infranodal blocks respond poorly)
Asymptomatic Mobitz I / first-degree	Observe; review AV-nodal drugs; cardiology follow-up
Drug-induced (e.g. β-blocker overdose)	Withhold drug; consider glucagon, high-dose insulin–glucose, calcium per toxicology advice



Figure 9.1 — First-degree AV block. Every P conducts, but the PR interval is prolonged (>200 ms) and fixed. Compare with the normal PR in Figure 1.2.

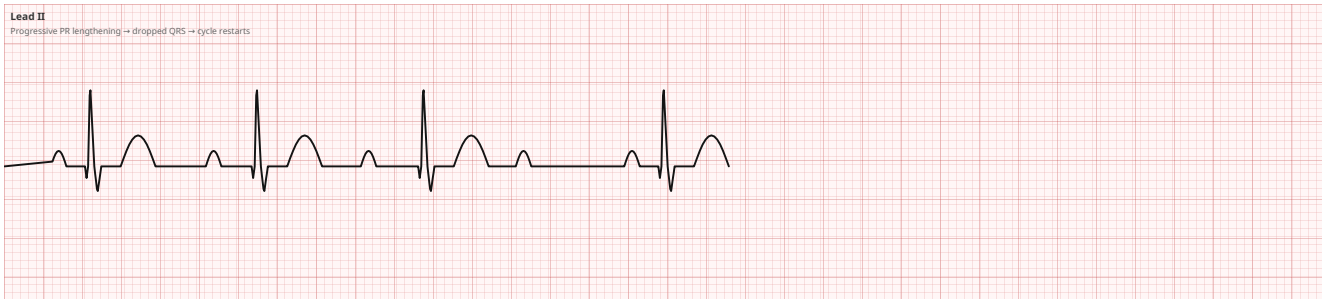


Figure 9.2 — Mobitz I (Wenckebach). Note the grouped beating: clusters of conducted beats separated by pauses. Narrow QRS favours an AV-nodal block — usually benign and reversible.

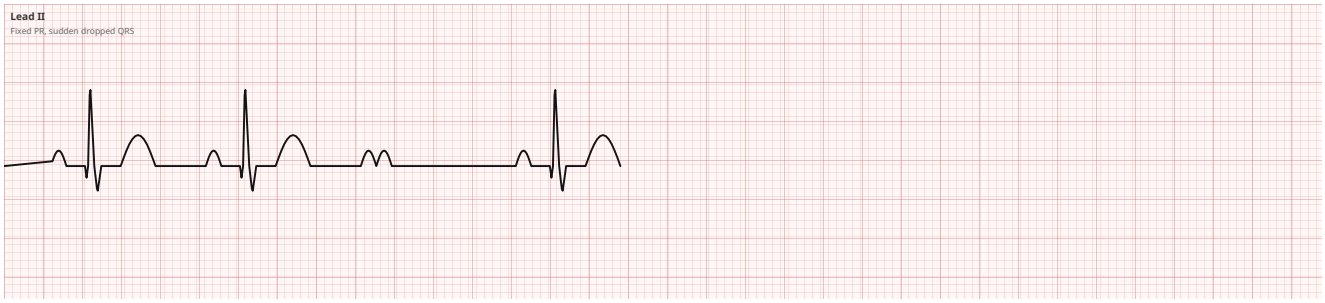


Figure 9.3 — Mobitz II. The PR of conducted beats is constant, then a P wave abruptly fails to conduct. Infranodal disease — high risk of progression to complete block; needs pacing.

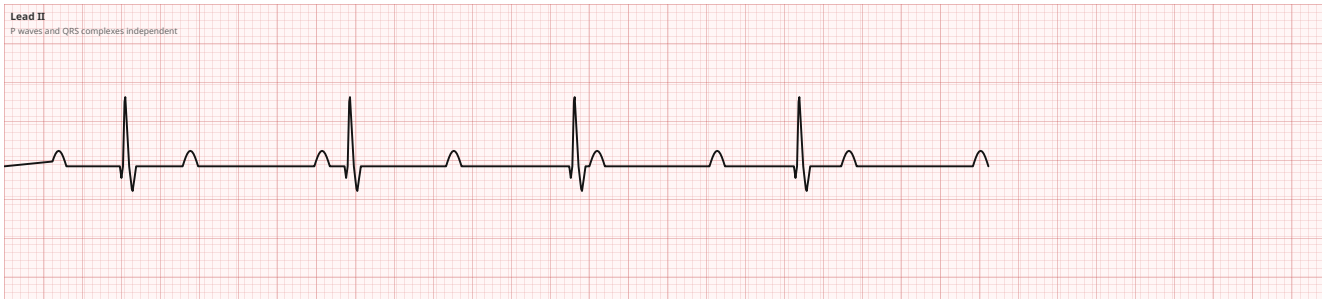


Figure 9.4 — Third-degree (complete) AV block. P waves march through regularly (faster) while a slower ventricular escape paces the QRS — AV dissociation. Atropine rarely helps infranodal escape; pace.

Red Flag

Wide-QRS bradycardia with AV dissociation = ventricular escape at 20–40/min. This patient can arrest without warning — attach pacing pads **now**, call cardiology, and do not wait for atropine to work.

CHAPTER 10

Bundle Branch and Fascicular Blocks

When one bundle branch fails, the ventricles depolarize sequentially instead of simultaneously: the impulse must travel muscle-to-muscle to the blocked side. Result: **QRS ≥120 ms** with a characteristic two-peaked ("rabbit ears") or broad monophasic shape.

Right bundle branch block (RBBB)

Criteria: **QRS ≥120 ms**, **rSR′ pattern in V1–V3** (tall second R, "rabbit ears"), and a **wide, slurred S wave in leads I, V5–V6**. T-wave discordance (opposite to the terminal QRS) is expected. Causes: right ventricular strain (PE, pulmonary hypertension), ischaemia, degenerative conduction disease; incomplete RBBB can be a normal variant in the young.

Left bundle branch block (LBBB)

Criteria: **QRS ≥120 ms**, **broad monophasic R waves in I, aVL, V5–V6**, **absent septal q waves** in those leads, and **discordant ST-T** (ST-T opposite the QRS). New LBBB was historically treated as a STEMI equivalent — current guidance: use the **Sgarbossa/Smith criteria** (Chapter 14) and, more importantly, the clinical picture. New LBBB with ischaemic symptoms warrants urgent cardiology input.

Fascicular blocks (hemiblocks) — QRS stays narrow (<120 ms)

Block	Axis	Key morphology	Common cause
Left anterior fascicular block (LAFB)	**Left axis deviation** (−45° to −90°)	**qR in aVL**, rS in II, III, aVF	LAD ischaemia, LVH, degenerative
Left posterior fascicular block (LPFB)	**Right axis deviation**	rS in I/aVL, qR in III/aVF	Rare — exclude RVH, PE first

Bifascicular and trifascicular block

Bifascicular = RBBB + LAFB (or LPFB), or LBBB alone (both fascicles blocked). **Trifascicular** = bifascicular block **plus** first-degree AV block (the AV node as the "third fascicle") — a marker of extensive conduction disease; symptomatic patients need pacing evaluation. Asymptomatic bifascicular block needs cardiology follow-up, not emergency pacing.

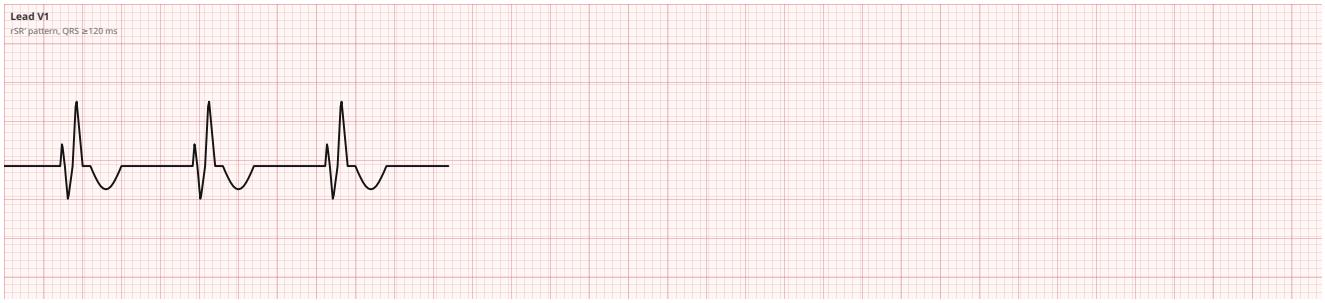


Figure 10.1 — RBBB in V1. Classic “rabbit ears”: small r, deep S, tall broad R′. Look for the reciprocal wide slurred S in I and V6.



Figure 10.2 — LBBB in V6. Wide, notched monophasic R wave with discordant (opposite-direction) ST-T changes. Absent q waves in lateral leads.

Clinical Pearl

LBBB **hides** ischaemia: you cannot read ST segments by normal rules. Apply the Sgarbossa criteria (Chapter 14) — concordant ST elevation ≥1 mm is worth 5 points and is highly specific for MI.

Red Flag

New bifascicular block (RBBB + LAFB) with syncope, or any Mobitz II / complete block, = pacing territory. Do not discharge these patients without cardiology input.

CHAPTER 11

Atrial Arrhythmias

Atrial arrhythmias arise above the ventricles, so the QRS is usually **narrow** (normal His–Purkinje conduction) — the atria, not the ventricles, are misbehaving. Read the P waves (or their absence) and the regularity first.

Premature atrial contractions (PACs)

An early beat with an **abnormal (non-sinus) P wave** followed by a usually narrow QRS, then a **non-compensatory pause** (the next sinus beat is not fully delayed). PACs are extremely common and usually benign — caffeine, alcohol, stress, fatigue. Frequent PACs can trigger AF; investigate if very frequent or symptomatic, but isolated PACs need reassurance, not drugs.

Atrial flutter

A macro-reentrant circuit around the tricuspid annulus produces **sawtooth flutter (F) waves at 250–350/min** (typically ~300), best seen in II, III, aVF. The AV node cannot conduct that fast, so conduction is usually **2:1** — giving the classic regular tachycardia at **~150/min**. Clues: ventricular rate suspiciously fixed near 150, sawtooth baseline, narrow QRS. Vagal manoeuvres or adenosine slow AV conduction transiently to *reveal* the flutter waves (they do not terminate flutter). Management mirrors AF: rate control, anticoagulation by CHA_2DS_2-VASc, and rhythm control — flutter responds excellently to **cavotricuspid isthmus ablation**.

Atrial fibrillation (AF)

Chaotic atrial activity (400–600/min) with **no discrete P waves**, **fibrillatory f waves** (fine baseline wobble), and an **irregularly irregular** narrow-complex rhythm. The ventricular rate depends on AV nodal conduction — fast AF (>100) is common.

Rate vs rhythm control: rate control (β-blocker or diltiazem/verapamil; digoxin if heart failure/sedentary) suits most — especially older, asymptomatic patients. Rhythm control (cardioversion, antiarrhythmics, ablation) suits the young, symptomatic, first-episode, or heart-failure patient where sinus rhythm clearly helps.

Anticoagulation — CHA_2DS_2-VASc: score 1 point each for Congestive heart failure, Hypertension, Age 65–74, Diabetes, Vascular disease, Sex female; 2 points each for Age ≥75 and prior Stroke/TIA/thromboembolism. **Men ≥2, women ≥3 → anticoagulate** (usually a DOAC); men 1 / women 2 → consider; 0 → no anticoagulation. Bleeding risk (HAS-BLED) guides caution, not denial.

Multifocal atrial tachycardia (MAT)

Irregular rhythm with **≥3 different P-wave morphologies** in the same lead, rate >100. Classic in **severe COPD exacerbations** and theophylline toxicity. Treat the lungs, not the rhythm — verapamil/diltiazem only if needed; cardioversion does not work.

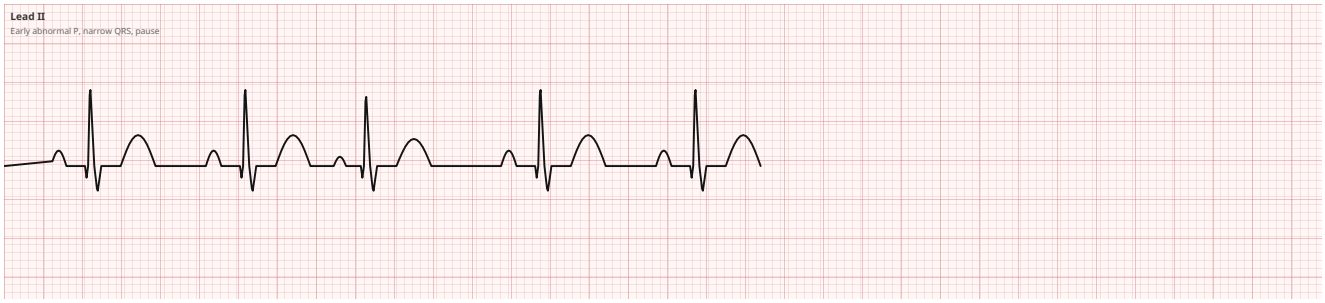


Figure 11.1 — Premature atrial contraction. The premature P wave looks different from sinus P waves; the QRS is narrow (normal ventricular conduction). Note the incomplete compensatory pause.



Figure 11.2 — Atrial flutter with 2:1 conduction. Regular ventricular rate ~150/min with sawtooth flutter waves marching through. Adenosine slows the ventricles to unmask the F waves.

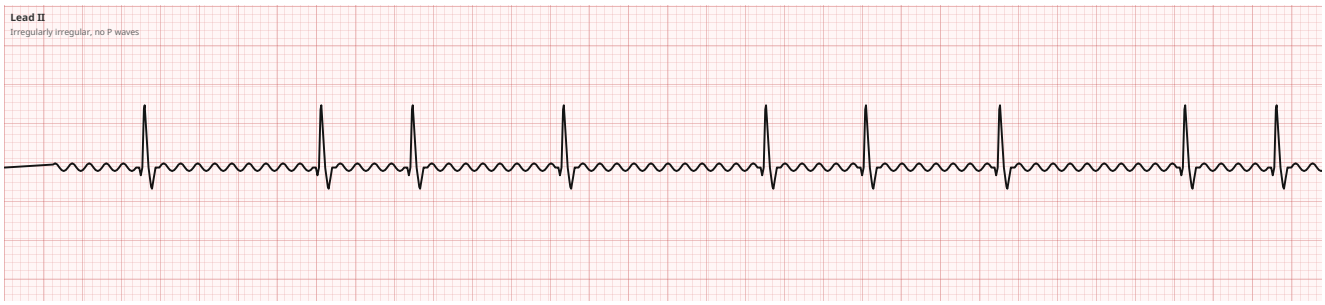


Figure 11.3 — Atrial fibrillation. Irregularly irregular narrow-complex rhythm, absent P waves, fine fibrillatory baseline. Assess rate, anticoagulation need, and cause.

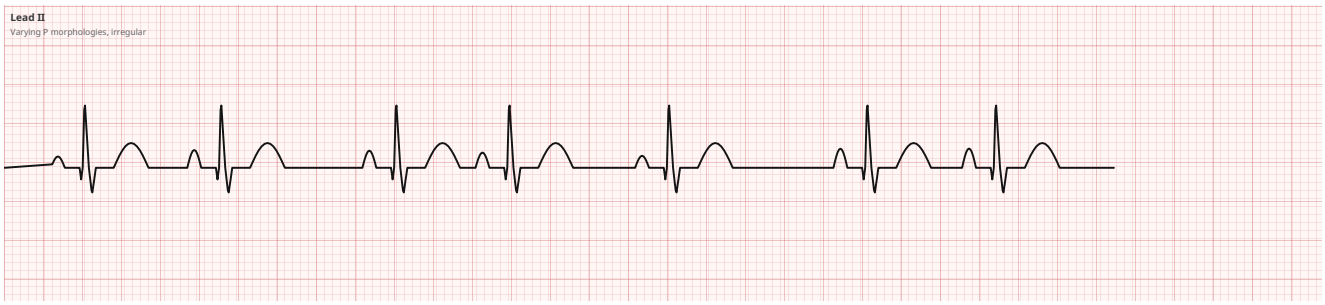


Figure 11.4 — Multifocal atrial tachycardia. Irregular rhythm with at least three distinct P-wave shapes. Think COPD exacerbation — treat the lungs.

> **Clinical Pearl**
>
> “Irregularly irregular” = AF until proven otherwise. But always exclude **complete heart block with variable conduction** and frequent PACs mimicking irregularity — march out the P waves before you commit.

CHAPTER 12

Junctional Rhythms and SVT

Junctional rhythms

When the SA node fails or AV conduction blocks, the **AV junction** escapes at **40–60/min**. Hallmarks: **narrow QRS**, **absent or inverted (retrograde) P waves** — appearing just before, during (hidden), or just after the QRS — and a slow regular rate. A junctional escape rhythm is a *rescue* rhythm: ask why the sinus node is not in charge (drugs, ischaemia, sick sinus syndrome) rather than suppressing the junction.

Supraventricular tachycardia (SVT)

AVNRT (AV nodal re-entry — dual AV nodal pathways, commonest regular SVT) and **AVRT** (accessory pathway, e.g. WPW) both present as **regular narrow-complex tachycardia at 150–250/min** with **P waves hidden inside the QRS** (AVNRT: pseudo-R′ in V1, pseudo-S in inferior leads) or just after it (AVRT). Onset and termination are **abrupt** — patients often describe a switch being flipped.

Acute management of stable regular narrow-complex tachycardia

1. **Vagal manoeuvres first:** modified Valsalva (semi-recumbent, blow hard into a syringe for 15 s, then lay flat and lift legs) — terminates ~40% of SVT.
2. **Adenosine 6 mg rapid IV push** (with 20 mL saline flush, arm raised); if no effect after 1–2 min, **12 mg**; a third **12 mg** dose may follow. Warn of transient flushing, chest tightness and a few seconds of asystole — it feels alarming but is brief. Avoid in asthma/COPD (bronchospasm) and use cautiously in transplanted hearts.
3. If adenosine fails: **verapamil/diltiazem** or a β-blocker IV; then consider **synchronized cardioversion** under sedation.

Unstable (hypotension, pulmonary oedema, ischaemic chest pain, altered consciousness) → **synchronized DC cardioversion immediately** — do not delay for drugs.

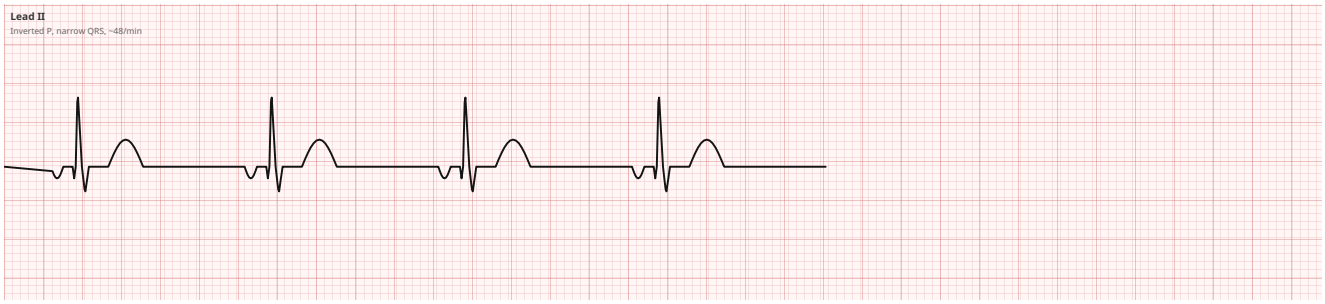


Figure 12.1 — Junctional escape rhythm. Slow regular narrow-complex rhythm with inverted retrograde P waves. A backup pacemaker doing its job — find out why the SA node is silent.

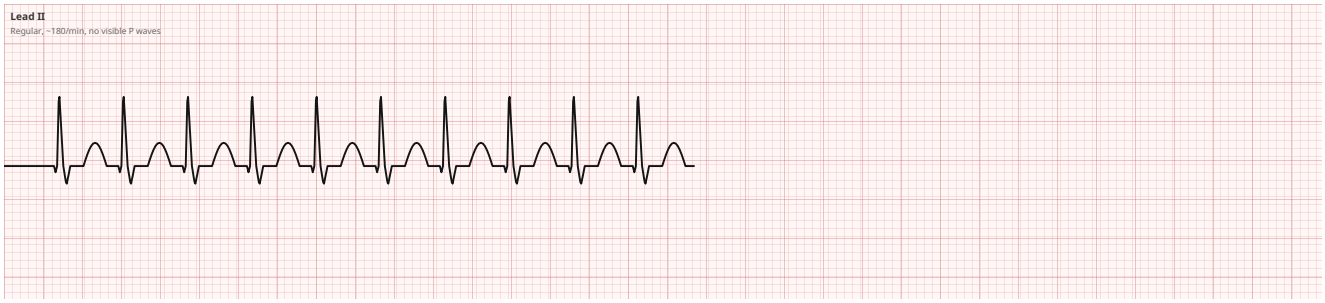


Figure 12.2 — SVT (AVNRT/AVRT). Abrupt-onset regular narrow-complex tachycardia; P waves buried in the QRS. Vagal manoeuvres → adenosine if stable; synchronized cardioversion if unstable.

Red Flag

Never give verapamil/diltiazem or adenosine to a **wide-complex** tachycardia you cannot confidently call SVT with aberrancy — treat as VT. And never cardiovert AF of >48 h (or unknown duration) without anticoagulation cover/TOE guidance.

CHAPTER 13

Ventricular Arrhythmias and Cardiac Arrest Rhythms

Ventricular rhythms are wide, bizarre and dangerous. **Any regular wide-complex tachycardia is VT until proven otherwise** — this single rule saves lives, because treating VT as SVT (with verapamil) can cause fatal hypotension.

Premature ventricular contractions (PVCs)

Early, **wide bizarre QRS** (≥120 ms) with **discordant T wave**, **no preceding P wave**, and usually a **full compensatory pause**. **Unifocal** PVCs look identical each time (one ectopic focus); **multifocal** PVCs vary (multiple foci — more concerning). Patterns: **bigeminy** (every other beat a PVC), trigeminy, **couplets** (two in a row), triplets. Frequent PVCs (>10–15% burden) can cause cardiomyopathy — refer. Isolated PVCs in a structurally normal heart are usually benign; correct K^+/Mg^{2+}, reduce stimulants, reassure.

Ventricular tachycardia (VT)

≥3 consecutive ventricular beats at ≥100/min, wide QRS. **Monomorphic VT** (uniform QRS — scar re-entry, e.g. old MI) vs **polymorphic VT** (changing QRS — acute ischaemia, channelopathies). Clues favouring VT over SVT with aberrancy: AV dissociation, capture/fusion beats, extreme axis, concordance across precordials, and a history of structural heart disease.

Torsades de pointes

A **polymorphic VT** with QRS complexes that appear to **twist around the baseline** (waxing–waning amplitude), occurring on a **prolonged QT**. Triggers: QT-prolonging drugs, hypokalaemia, hypomagnesaemia, bradycardia. Treatment is **magnesium sulphate 1–2 g IV** (even if Mg^{2+} normal), correct K^+ to high-normal, stop offending drugs, and consider overdrive pacing/isoprenaline for pause-dependent torsades. **Amiodarone prolongs QT — avoid it here.**

Ventricular fibrillation, asystole, PEA

VF: chaotic, irregular baseline with no identifiable QRS — no cardiac output. **Asystole:** flat line (confirm in two leads, check gain, exclude loose lead). **PEA:** organized electrical rhythm but no pulse. All three are cardiac arrest rhythms — start CPR immediately.

Brief ACLS pointers (adult)

- **Unstable tachycardia with a pulse** (hypotension, shock, ischaemia, altered) → **synchronized cardioversion** under sedation.
- **Pulseless VT / VF → unsynchronized defibrillation** (biphasic 120–200 J) + high-quality CPR; **adrenaline 1 mg IV every 3–5 min**; **amiodarone 300 mg IV** after the third shock (second dose 150 mg).

- **Asystole / PEA** → CPR + adrenaline 1 mg every 3–5 min; **no shock**; hunt reversible causes — the **4 Hs** (Hypoxia, Hypovolaemia, Hypo/Hyperkalaemia, Hypothermia) and **4 Ts** (Tamponade, Tension pneumothorax, Toxins, Thrombosis).

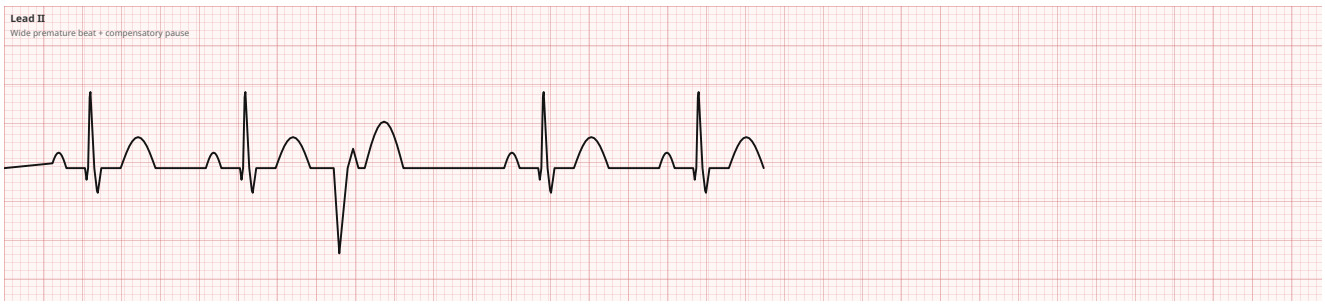


Figure 13.1 — Premature ventricular contraction. Wide bizarre premature QRS with discordant T wave and a full compensatory pause. Unifocal here (identical morphology).

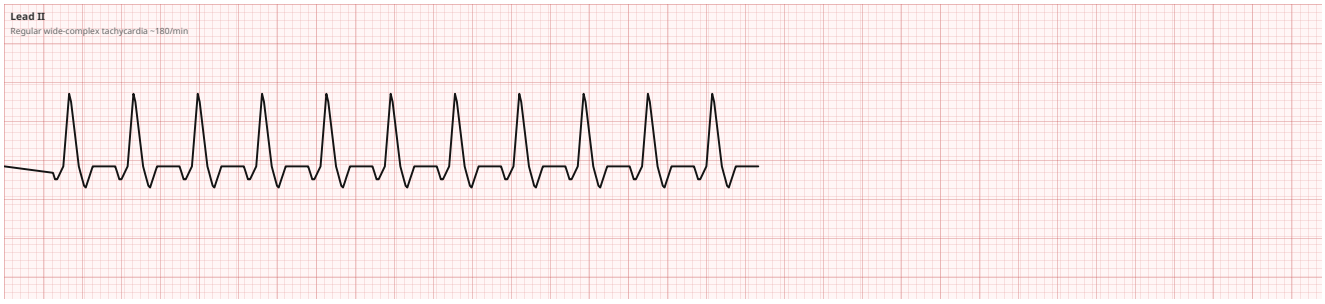


Figure 13.2 — Monomorphic ventricular tachycardia. Regular, wide, uniform QRS complexes. Treat any regular wide-complex tachycardia as VT until proven otherwise.

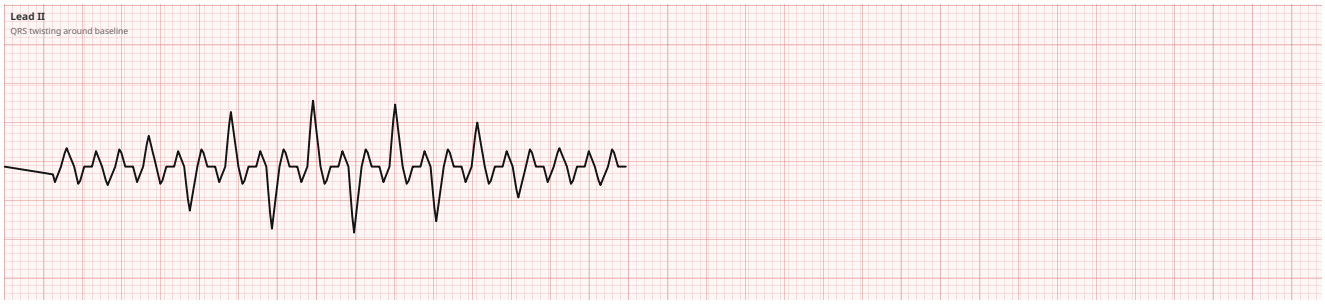


Figure 13.3 — Torsades de pointes. Polymorphic VT with waxing–waning amplitude on a prolonged QT. Give magnesium, correct potassium, stop QT-prolonging drugs.

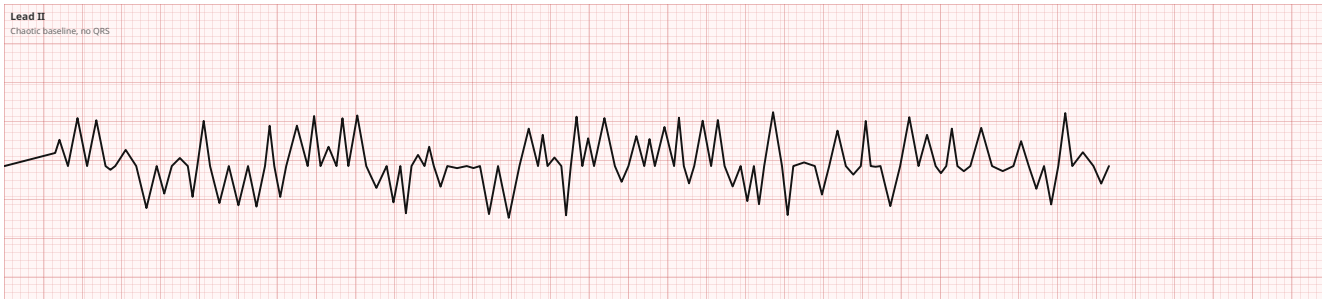


Figure 13.4 — Ventricular fibrillation. Coarse chaotic activity, no organized complexes, no output. Defibrillate immediately with ongoing CPR.



Figure 13.5 — Asystole. Confirm in more than one lead, check connections and gain — then CPR, adrenaline, and reversible causes. Never shock asystole.

Red Flag

A **regular wide-complex tachycardia at ~150/min** in a patient with prior MI is VT until proven otherwise — even if they look well. Do not trial verapamil; cardiovert if unstable, amiodarone/procainamide pathway if stable per local protocol.

CHAPTER 14

Acute Coronary Syndromes and STEMI

Acute coronary syndrome is where ECG reading saves the most lives. The tracing must be acquired **within 10 minutes** of arrival for suspected ACS — and a single normal ECG never rules it out.

STEMI criteria (4th Universal Definition)

ST elevation at the J point in **≥2 contiguous leads**:

- **Limb leads:** ≥1 mm (0.1 mV).
- **Precordial V2–V3:** ≥2.5 mm in men <40 years, ≥2.0 mm in men ≥40, ≥1.5 mm in women.
- **Other precordials / posterior V7–V9:** ≥1 mm (≥0.5 mm in V7–V9, except ≥1 mm in men <40).
- **V4R (RV infarct):** ≥1 mm.

Measure at the J point (or J+60 ms). The morphology matters: **convex ("tombstone") elevation strongly favours MI**; concave elevation is more typical of early repolarization or pericarditis.

Evolution of infarction

Minutes: hyperacute T waves — tall, broad, symmetric (the earliest sign). **Minutes–hours:** convex ST elevation. **Hours: pathologic Q waves** develop (>40 ms wide or >25% of R) as myocardium dies. **Hours–days:** ST segments settle, T waves invert. **Days–weeks:** T waves may normalize; Q waves often persist permanently as the scar's signature. **Reciprocal ST depression** in electrically opposite leads is the hallmark that distinguishes true STEMI from mimics.

Localization: which artery?

STE territory	Wall	Likely culprit
II, III, aVF	Inferior	**RCA** (usually); LCx possible
V1–V4	Anterior/septal	**LAD**
I, aVL, V5–V6	Lateral	**LCx** (or diagonal)
STD V1–V3 + tall R	Posterior (mirror)	**LCx/RCA** (posterior descending)
STE in V4R	Right ventricle	**Proximal RCA**
STE aVR (+ diffuse STD)	Left main or triple-vessel disease pattern — ominous, urgent angiography	

NSTEMI and unstable angina

Same clinical syndrome **without** persistent ST elevation: **ST depression, transient STE, or T-wave inversion** — or a completely normal ECG. **Troponin** separates NSTEMI (positive) from unstable angina (negative). Manage with antiplatelets, anticoagulation, statin and early invasive strategy per risk scores (GRACE/TIMI).

High-risk ischaemic patterns you must not miss

Wellens syndrome: deeply inverted (type B) or biphasic (type A) T waves in **V2–V3** when pain-free — critical proximal LAD lesion; "do not stress test, go to cath." **de Winter pattern: upsloping ST depression with tall symmetric T waves** in the precordials — a STEMI equivalent of proximal LAD occlusion; activate reperfusion. **Posterior MI:** ST depression + tall R in V1–V3 (the mirror of posterior STE) — confirm with V7–V9.

Sgarbossa criteria (LBBB or ventricular-paced rhythm)

Criterion	Points
Concordant ST elevation ≥1 mm in a lead with a positive QRS	**5**
ST depression ≥1 mm in V1–V3	**3**
Discordant ST elevation ≥5 mm (Smith: ≥25% of QRS amplitude)	**2**

Score ≥3 = MI (high specificity). Any concordant ST deviation in LBBB is pathological — normal LBBB is always discordant.

Brugada vs early repolarization

Brugada type 1: coved ST elevation ≥2 mm in V1–V3 followed by an inverted T ("shark-fin") — sodium-channel disease with risk of VF; refer for electrophysiology (fever and sodium-blockers unmask it). **Early repolarization:** concave STE with notched J point, usually in young athletic men, without reciprocal change — benign. When uncertain in a symptomatic patient, treat as ischaemia first.

Takotsubo (stress) cardiomyopathy

Typically postmenopausal women after emotional/physical stress: ECG mimics anterior STEMI (STE or deep T inversion, long QT), troponin mildly raised, but coronaries are clean and the LV shows apical ballooning. Manage supportively; most recover — but acute complications (shock, VT) need full ACS-level care initially.

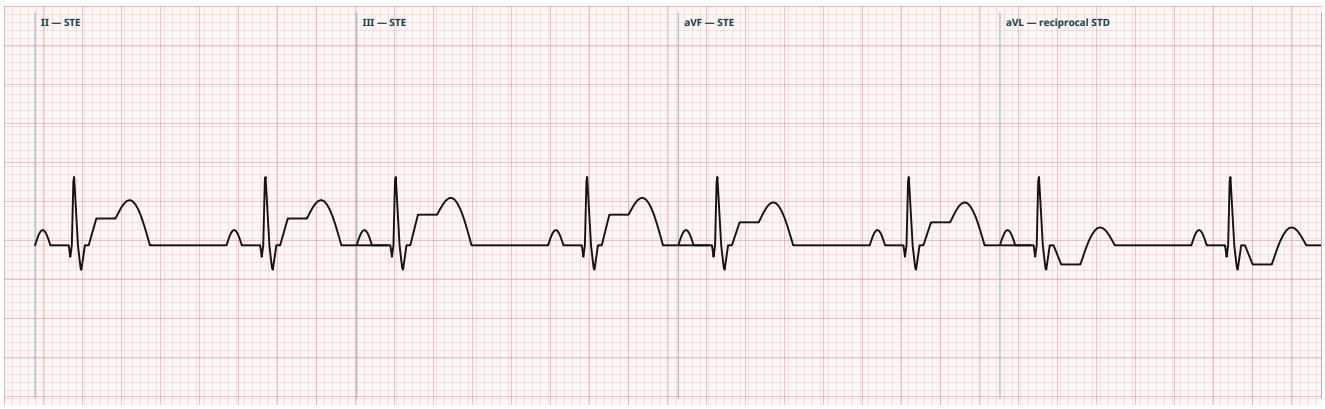


Figure 14.1 — Inferior STEMI. Convex ST elevation in the inferior leads (II, III, aVF) with reciprocal ST depression in aVL — the reciprocal change confirms true ischaemia rather than early repolarization. Culprit usually the RCA; record V4R for RV infarct.

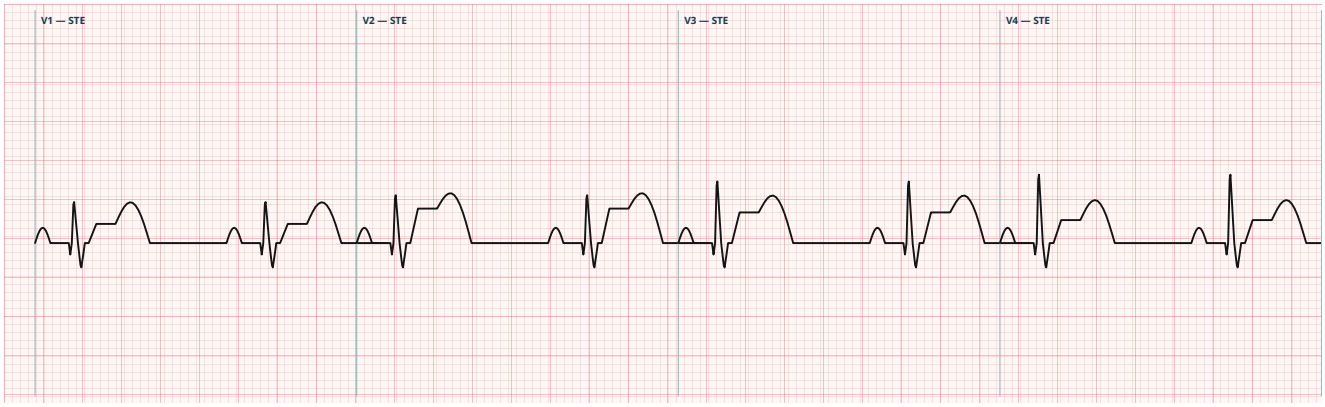


Figure 14.2 — Anterior STEMI. ST elevation across V1–V4 (LAD territory). Anterior MI jeopardizes a large muscle mass — higher risk of heart failure, VT/VF and bundle-branch blocks; activate reperfusion immediately.

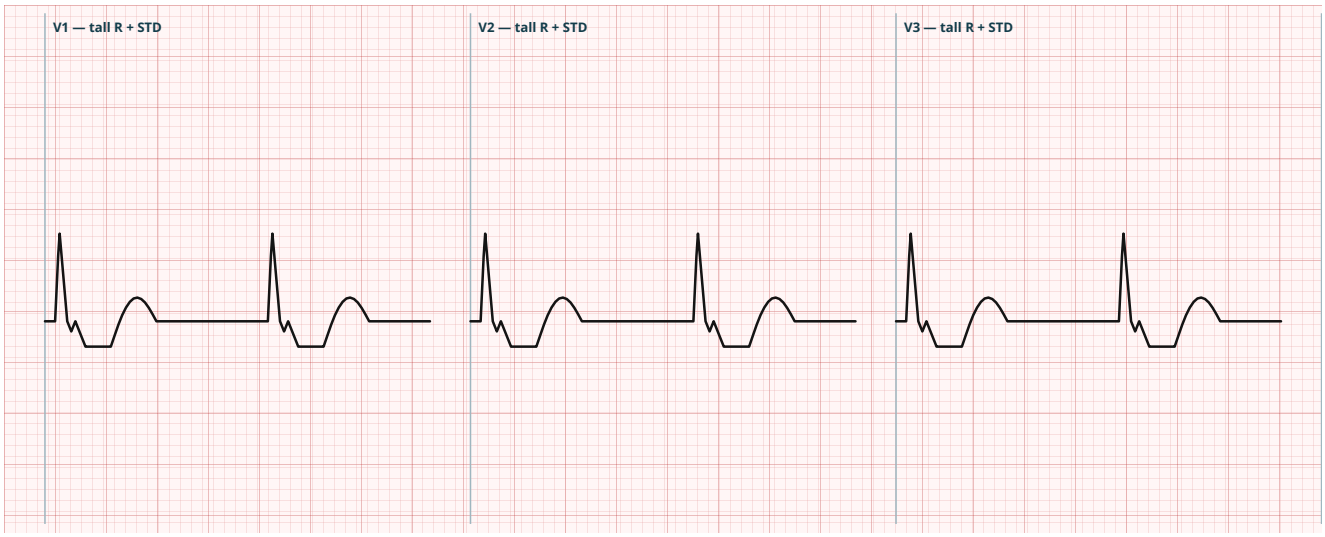


Figure 14.3 — Posterior MI (mirror pattern). The posterior wall is not seen directly: its STEMI appears as ST *depression* with tall R waves and upright T waves in V1–V3. Confirm with posterior leads V7–V9 (STE ≥0.5 mm).

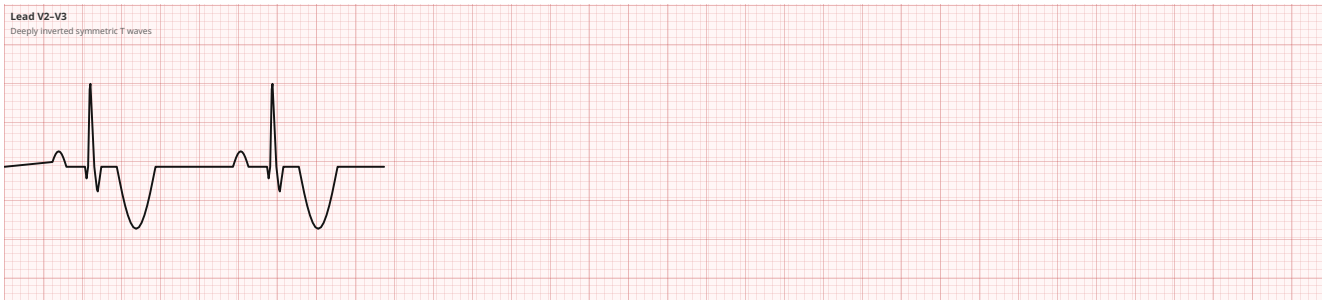


Figure 14.4 — Wellens syndrome type B. Deeply inverted, symmetric T waves in V2–V3 in a pain-free patient = critical proximal LAD stenosis. Type A shows biphasic T waves. Do **not** stress-test — refer urgently for angiography; these patients infarct within days.

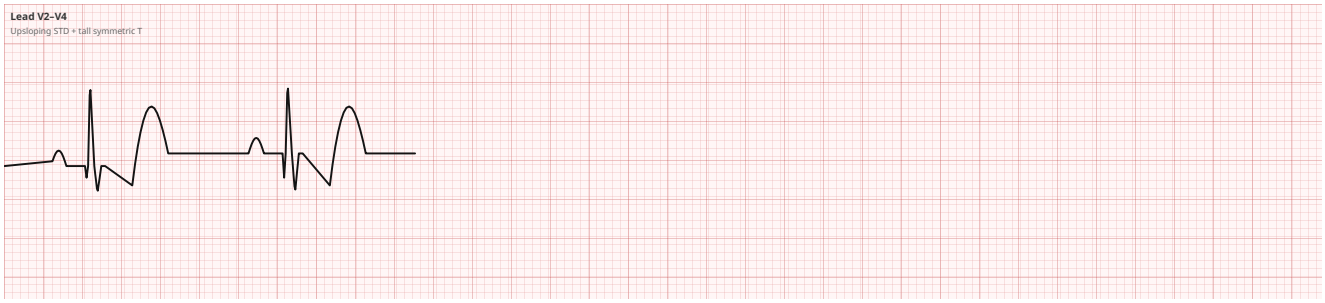


Figure 14.5 — de Winter pattern. A STEMI equivalent: upsloping ST depression with tall, symmetric T waves in the precordials = proximal LAD occlusion. Activate reperfusion as for STEMI.



Figure 14.6 — Brugada type 1 pattern. Coved ("shark-fin") ST elevation ≥2 mm in V1–V3 followed by an inverted T. Sodium-channel disease with VF risk — fever unmasks it; refer to electrophysiology.

Clinical Pearl

STE criteria are **sex- and age-specific** in V2–V3 (2.5 / 2.0 / 1.5 mm). Using a flat 1-mm rule everywhere misses anterior MIs in young men and overcalls them in women — apply the right number.

Red Flag

aVR STE with diffuse ST depression = left main or severe triple-vessel disease until proven otherwise. This patient needs urgent cardiology — do not wait for troponin.

CHAPTER 15

Acute Pericarditis vs STEMI

Both acute pericarditis and STEMI elevate ST segments — but one needs reperfusion and the other needs anti-inflammatories. Telling them apart is a core bedside skill.

Feature	Acute pericarditis	STEMI
ST elevation	**Diffuse** (most leads), **concave** ("smiley")	**Territorial** (anatomical group), **convex** ("tombstone")
Reciprocal ST depression	**Absent** (except aVR/V1 may show reciprocal change)	**Present** in opposite leads
PR segment	**PR depression** (especially II, aVF, V5–V6) — atrial injury current	PR usually normal
Q waves	None (no necrosis)	Pathologic Q waves evolve
T waves	Remain upright while ST elevated	Invert as ST settles
Spodick's sign	**Downsloping TP segment** — specific for pericarditis	Absent
Clinical	Pleuritic, positional pain (better sitting forward); pericardial rub; diffuse	Pressure-like, exertional; territorial radiation

Stages of pericarditis ECG

Stage 1 (hours–days): diffuse STE + PR depression. **Stage 2:** ST normalizes, T flattens. **Stage 3:** T-wave inversion. **Stage 4** (weeks): normalization. Unlike MI, **Q waves never form** and the ST/T evolution is diffuse.

Management notes

Pericarditis: **NSAIDs + colchicine**, rest, treat the cause (viral most common; consider TB in endemic settings, uraemia, post-MI/Dressler, malignancy). Avoid anticoagulants if a significant effusion is present. Red flags for **tamponade**: hypotension, raised JVP, muffled heart sounds (Beck's triad), pulsus paradoxus, electrical alternans, low voltage — urgent echocardiography and pericardiocentesis.



Figure 15.1 — Pericarditis vs STEMI. Pericarditis: diffuse *concave* STE with PR depression and **no reciprocal change**. STEMI: territorial *convex* STE **with reciprocal depression** in opposite leads.

Clinical Pearl

PR depression is the most specific ECG sign of pericarditis — look for it in lead II whenever you see diffuse STE. STEMI essentially never depresses the PR segment.

Red Flag

Pericarditis pain can be severe and troponin mildly raised (myopericarditis) — but **territorial convex STE with reciprocal change is MI until proven otherwise**. When the ECG is ambiguous, urgent cardiology review beats regret.

CHAPTER 16

Electrolytes, Temperature and Drug Effects

The ECG is a real-time electrolyte monitor. Potassium reshapes the tracing progressively; calcium moves the QT; temperature and drugs add their own signatures.

Hyperkalaemia — the classic progression

As K^+ rises: **(1)** tall, peaked, tented T waves (earliest); **(2)** flattened P waves with PR prolongation; **(3)** QRS widening; **(4)** sine-wave pattern merging QRS-T; then VF/asystole. **Management: calcium** (gluconate or chloride IV) immediately if QRS wide or arrhythmic — it stabilizes the membrane without lowering K^+; then **insulin + glucose**, **salbutamol nebulization**, **bicarbonate** if acidotic, and **definitive removal** (calcium resonium, dialysis). Recheck K^+ and the ECG continuously.

Hypokalaemia

Prominent U waves, **flattened T waves**, **ST depression**, prolonged QU, and arrhythmia tendency (PACs/PVCs, torsades risk with long QT). Replace potassium (oral if mild and no ECG changes; IV with cardiac monitoring if severe or ECG changes), and **check magnesium** — hypomagnesaemia makes hypokalaemia refractory.

Calcium

Hypercalcaemia: short QT (shortened ST segment). **Hypocalcaemia: long QT** (prolonged ST segment) — torsades risk. Calcium disorders move the ST segment; potassium disorders move the T wave and QRS.

Disturbance	ECG signature
Hyperkalaemia	Peaked T → flat P/long PR → wide QRS → sine wave
Hypokalaemia	U waves, flat T, ST depression, long QU
Hypercalcaemia	Short QT (short ST)
Hypocalcaemia	Long QT (long ST)
Hypothermia	**Osborn (J) wave** — hump at the J point; bradycardia, long QT
Digoxin effect	**Scooped ("Salvador Dalí") ST depression**, short QT; toxicity → almost any arrhythmia + AV block

QT-prolonging drugs (watch the QTc)

Antiarrhythmics: amiodarone, sotalol, quinidine. **Antibiotics:** macrolides (erythromycin, azithromycin), fluoroquinolones. **Antipsychotics:** haloperidol, quetiapine, ziprasidone. **Others:** ondansetron, methadone, some antifungals. Risk multiplies with hypokalaemia, hypomagnesaemia and bradycardia — check the ECG before and after starting these drugs.

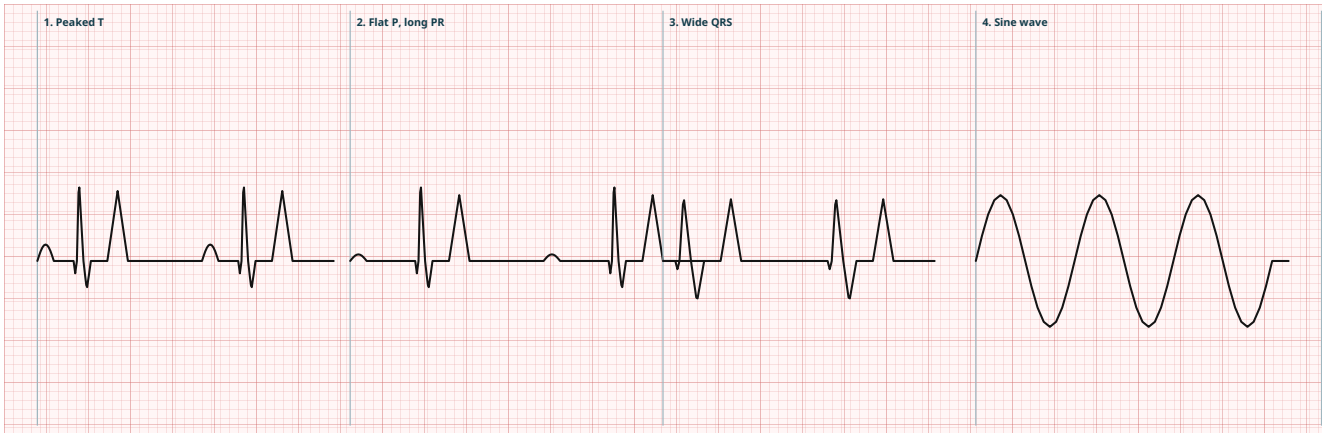


Figure 16.1 — Hyperkalaemia progression. Rising K^+: tall peaked T waves → flattened P with PR prolongation → widened QRS → sine-wave pattern → asystole. Treat on the ECG, not the lab slip — give calcium first if the QRS is wide.

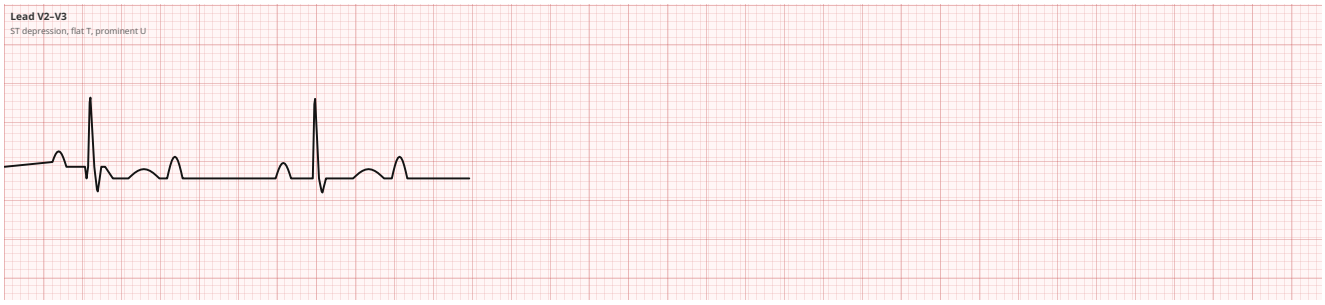


Figure 16.2 — Hypokalaemia. Sagging ST depression, flattened T waves and a prominent U wave (the "hump" after the T). Prolonged QU predisposes to torsades — replace K^+ and check Mg^{2+}.

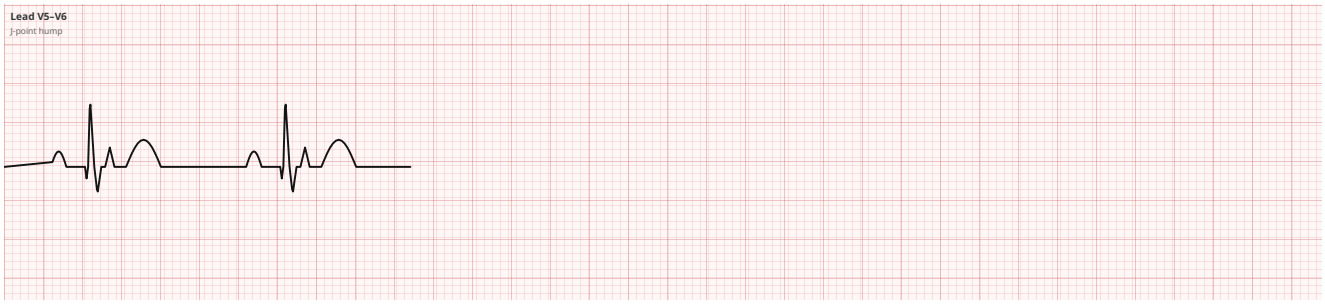


Figure 16.3 — Hypothermia: Osborn (J) wave. A positive deflection at the J point, best in lateral precordials; height tracks the degree of cooling. Handle gently — the cold heart fibrillates easily.

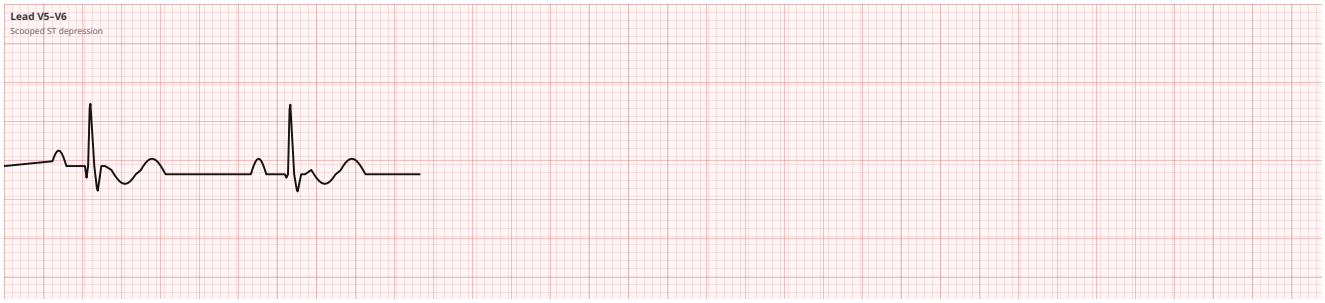


Figure 16.4 — Digoxin effect. Downsloping "scooped" ST depression (the "Salvador Dalí moustache") with a short QT. This is drug *effect*, not toxicity — toxicity shows as arrhythmias and AV block.

Red Flag

A **sine-wave pattern** is pre-arrest hyperkalaemia. Do not wait for the lab: give IV calcium immediately, then insulin–glucose, and prepare for dialysis. Similarly, a QTc >500 ms on a QT-prolonging drug needs same-day action.

CHAPTER 17

Chamber Enlargement

Hypertrophied chambers generate larger electrical forces, shifting QRS voltage and axis. Voltage criteria are **specific but insensitive** — echocardiography confirms; the ECG screens.

Left ventricular hypertrophy (LVH)

Criterion	Rule
Sokolow–Lyon	**S in V1 + R in V5 (or V6) ≥35 mm**
Cornell	**R in aVL + S in V3 >28 mm (men) / >20 mm (women)**
Strain pattern	Downsloping ST depression + asymmetric T inversion in lateral leads (I, aVL, V5–V6) — suggests pressure overload

Supportive signs: left axis deviation, left atrial enlargement, prolonged QRS. Causes: hypertension (commonest), aortic stenosis, hypertrophic cardiomyopathy. Note: voltage criteria overcall LVH in young, thin, athletic chests — interpret with the patient, not just the numbers.

Right ventricular hypertrophy (RVH)

R > S in V1 (dominant R), **right axis deviation**, and strain pattern in V1–V3. Causes: pulmonary hypertension, chronic lung disease, pulmonary stenosis, mitral stenosis (via pulmonary hypertension).

Atrial enlargement

Left atrial enlargement (P mitrale): P wave **>120 ms**, often **notched** (bifid) in II, with a deep terminal negative component in V1 — classic in mitral stenosis and LV diastolic dysfunction. **Right atrial enlargement (P pulmonale):** P wave **>2.5 mm tall**, peaked, in II, III, aVF — pulmonary disease, pulmonary hypertension, tricuspid stenosis.



Figure 17.1 — Left ventricular hypertrophy. Sokolow–Lyon: S in V1 (22 mm) + R in V5 (20 mm) = 42 mm ≥ 35 mm. Note the “strain” pattern — discordant ST-T in the lateral leads, a marker of pressure overload.

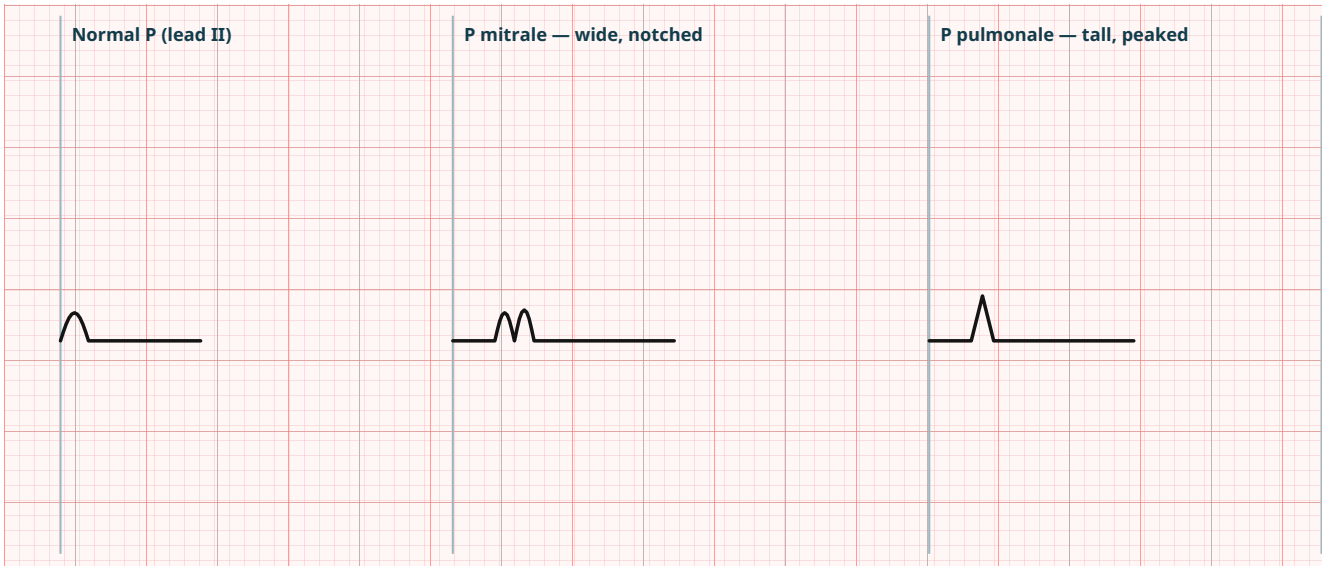


Figure 17.2 — Atrial enlargement P-wave patterns. P mitrale: P >120 ms, notched (left atrial enlargement). P pulmonale: P >2.5 mm tall, peaked (right atrial enlargement).

Clinical Pearl

The **strain pattern** (lateral ST depression + T inversion) in a hypertensive patient with LVH voltage is a red flag for severe pressure overload — tighten blood-pressure control and look for aortic stenosis.

Red Flag

New LVH strain pattern with chest pain can mask ischaemia — compare with the old ECG and check troponin. Repolarization abnormalities from LVH do not cause troponin rise.

CHAPTER 18

Pulmonary Embolism

Pulmonary embolism (PE) strains the right heart acutely, and the ECG reflects right-sided stress. **No ECG finding rules PE in or out** — the ECG's role is to raise suspicion and exclude STEMI.

ECG clues in PE

- **Sinus tachycardia** — commonest finding, but entirely non-specific.
- **S1Q3T3:** S wave in lead I, Q wave + inverted T in lead III — the famous pattern of acute right-heart strain. Memorable but **insensitive and non-specific**.
- **T-wave inversions in V1–V4** — the most **sensitive** ECG sign of PE-related right ventricular strain (still not specific).
- Right bundle branch block (new), right axis deviation, P pulmonale, clockwise rotation.

Bedside approach

In a patient with pleuritic chest pain, dyspnoea or syncope: tachycardia + right-strain pattern + T inversion V1–V4 should push PE up your differential — apply a clinical score (Wells/Geneva), check D-dimer appropriately, and go to **CT pulmonary angiography** when indicated. Massive PE with hypotension is a resuscitation diagnosis: bedside echo showing a dilated, hypokinetic RV justifies thrombolysis per local protocol when CT is not feasible.

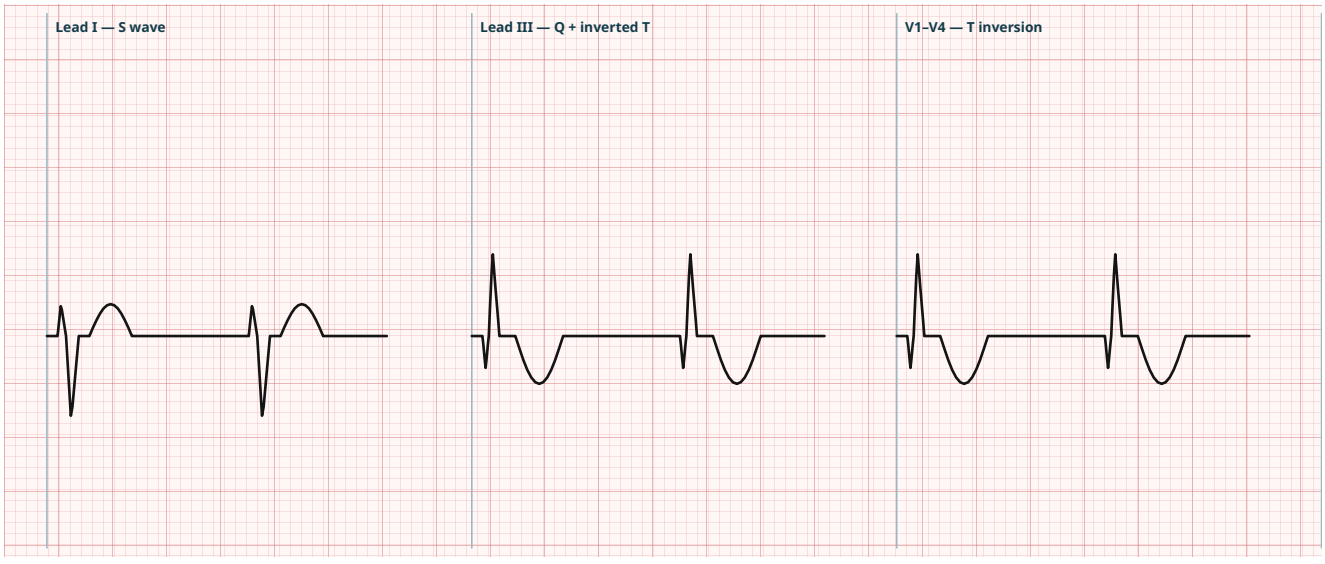


Figure 18.1 — The S1Q3T3 pattern of acute right-heart strain. S in lead I, Q with inverted T in lead III. Sensitive but **not specific** — sinus tachycardia and T inversion in V1–V4 are the more useful bedside clues in suspected PE.

Clinical Pearl

T-wave inversion that is **deepest in V1–V3 and shallows toward V6** suggests right ventricular strain (PE); inversion deepest in V5–V6 suggests LV ischaemia. The *gradient* across the precordials localizes the strain.

Red Flag

Anterior T-wave inversion + tachycardia + hypotension after long immobility, surgery or in pregnancy = PE until proven otherwise. Do not anchor on “anterior ischaemia” and delay anticoagulation workup.

CHAPTER 19

Pre-excitation (WPW)

In pre-excitation, an **accessory pathway** (bundle of Kent) connects atria to ventricles directly, bypassing the AV node's protective delay.

Wolff–Parkinson–White (WPW)

The triad: **(1) PR interval <120 ms**, **(2) delta wave** — slurred upstroke of the QRS (early ventricular activation via the accessory path), **(3) wide QRS** (≥120 ms) with discordant ST-T changes that can mimic ischaemia or BBB. The delta wave's polarity hints at the pathway's location (used by electrophysiologists for ablation planning).

Orthodromic AVRT (commonest SVT in WPW): impulse goes *down* the AV node and *up* the accessory pathway — **narrow-complex** regular tachycardia. **Antidromic AVRT**: down the accessory pathway, up the AV node — **wide-complex** regular tachycardia, easily mistaken for VT.

The dangerous scenario: pre-excited AF

If AF develops in WPW, atrial impulses bombard the ventricles via the accessory pathway **without AV-nodal rate limiting** — producing an **irregular, very fast, wide-complex** rhythm that can degenerate into VF. **Never give AV-nodal blockers** (verapamil, diltiazem, β-blockers, digoxin, adenosine, amiodarone is debated) in pre-excited AF — they block the AV node and leave the accessory pathway as the only route, accelerating the ventricles. Unstable → **synchronized cardioversion**; stable → urgent cardiology (procainamide/ibutilide per protocol, then ablation).

Lown–Ganong–Levine (LGL)

Short PR (<120 ms) with **normal QRS and no delta wave** — enhanced AV nodal conduction rather than an accessory pathway. Usually benign; SVT can occur but the malignant AF risk of WPW does not apply.

Definitive management

Symptomatic WPW (SVT, syncope, pre-excited AF) → **electrophysiology study and radiofrequency ablation**, which is curative in >95%. Asymptomatic pre-excitation found incidentally needs cardiology risk stratification (especially athletes).

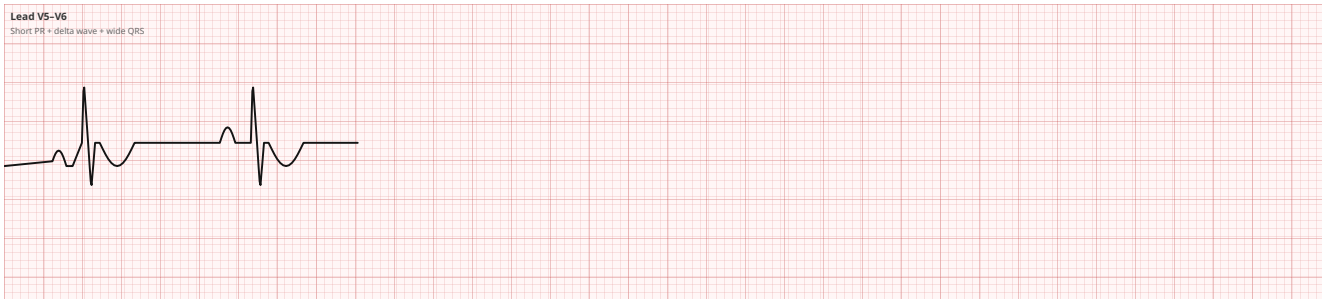


Figure 19.1 — Wolff–Parkinson–White (WPW). The classic triad: PR <120 ms, slurred upstroke (δ wave) of the QRS, and wide QRS with discordant T. The accessory pathway bypasses the AV node — never give AV-nodal blockers in pre-excited AF.

Red Flag

Irregular wide-complex tachycardia at 200+/min in a young patient = **pre-excited AF** until proven otherwise. AV-nodal blockers can kill here — cardiovert if unstable, call cardiology immediately if stable.

CHAPTER 20

Paced Rhythms and Artefacts

Paced rhythms

A **pacing spike** — a sharp vertical artefact — precedes the depolarization it triggers. **Atrial pacing:** spike before each P. **Ventricular pacing:** spike before each wide, bizarre QRS (LBBB-like with left axis, since the lead is usually in the RV apex). **AV sequential (dual-chamber) pacing:** spikes before both P and QRS. Check: is there a spike before every expected complex (capture)? Is every spike followed by a complex (capture vs failure)? Is the device sensing intrinsic beats (no inappropriate spikes)? Pacemaker malfunction + symptoms = urgent cardiology/device check; a magnet or reprogramming may be needed per protocol.

Limb-lead reversal vs dextrocardia

LA–RA reversal (commonest lead error): **lead I shows a negative P-QRS-T** (global negativity), **aVR becomes positive**, and leads II/III swap. The precordials look normal. Fix: swap the arm electrodes and repeat.

Dextrocardia (mirror-image heart): same limb-lead pattern **plus** poor R-wave progression and dominant S waves across **all** precordials (the chest leads are looking at the wrong side). Confirm by repeating with reversed limb leads *and* right-sided chest leads (V1R–V6R) — the tracing then normalizes.

Feature	Limb-lead reversal	Dextrocardia
Lead I	Negative P-QRS-T	Negative P-QRS-T
aVR	Positive	Positive
Precordials	**Normal** R progression	**Reversed** — dominant S throughout
Fix	Swap arm electrodes	Right-sided chest leads

Other artefacts

Wandering baseline: breathing or poor skin contact — reposition electrodes, clean skin. **Muscle tremor:** fine irregular noise (Parkinson's, shivering, anxiety) — warm and reassure the patient. **AC interference:** regular 50 Hz thickening of the baseline — check earthing, move away from electrical devices. **Lead misplacement:** V1–V2 placed too high mimics Brugada or incomplete RBBB — verify the 4th intercostal space.

> **Bedside Check**
>
> Before diagnosing an exotic rhythm, run the artefact drill: **patient still and warm? electrodes on clean dry skin? correct limb and chest positions? calibration 10 mm/mV, speed 25 mm/s?** Most "bizarre" tracings are technical, not cardiac.

Red Flag

A paced patient who is symptomatic with **spikes not followed by QRS complexes** has loss of capture — treat as an emergency: pads on, cardiology/device team immediately, transcutaneous pacing standby.

CHAPTER 21

The Rapid Bedside Read

When the patient is crashing, you do not have time for the full 8 steps. This is the distilled version — sixty seconds, ten questions.

10-Point Rapid Read

1. **Rate** — too fast (>150), too slow (<40), or okay?
2. **Regular?** — irregularly irregular = AF until proven otherwise.
3. **Wide or narrow QRS?** — wide + regular + fast = **VT until proven otherwise**.
4. **P waves** — present and married to QRS? Marching alone (block)?
5. **ST elevation?** — ≥2 contiguous leads, convex, with reciprocal change = STEMI.
6. **ST depression / T inversion** — ischaemia, strain, or Wellens in V2–V3?
7. **QRS width + T shape** — BBB? Peaked T (hyperkalaemia)? Sine wave?
8. **QTc** — >500 ms or on QT-prolonging drugs = torsades risk.
9. **Axis** — extreme axis in tachycardia favours VT.
10. **Compare with old ECG** — new vs old changes everything.

Red Flags — Never Miss

- **STEMI pattern** (territorial convex STE + reciprocal change) — activate reperfusion.
- **aVR STE + diffuse STD** — left main disease.
- **Wellens / de Winter** — LAD occlusion in disguise.
- **Complete heart block / Mobitz II** — pacing.
- **VT / VF / torsades** — cardioversion/defibrillation/magnesium.
- **Sine wave / severe hyperkalaemia pattern** — IV calcium now.
- **Brugada type 1** in a syncope patient — VF risk.
- **Pre-excited AF** — no AV-nodal blockers.
- **Posterior MI** hiding as anterior STD — check V7–V9.

When to urgently get cardiology help

- Any suspected **acute coronary occlusion** (STEMI, Wellens, de Winter, posterior MI, left-main pattern).
- **Unstable arrhythmia** — hypotension, ischaemia, pulmonary oedema, altered consciousness.
- **High-grade AV block**, symptomatic bradycardia unresponsive to atropine.
- **Wide-complex tachycardia** you cannot confidently diagnose.
- **QTc >500 ms**, Brugada pattern with syncope, or any inherited-arrhythmia suspicion.
- Any tracing where **history and ECG disagree** — trust the patient, escalate, repeat the ECG.

Common pitfalls

- **Stopping at the first abnormality** — AF *and* STEMI coexist; always finish the 8 steps.
- **Calling VT "SVT with aberrancy"** without proof — default to VT.
- **Forgetting the old ECG** — "new" LBBB, "new" Q waves and "new" T inversions change management.
- **Misplaced chest leads** (V1–V2 too high) mimicking Brugada/RBBB — recheck anatomy.
- **Limb-lead reversal** mimicking dextrocardia or lateral MI — check lead I polarity.
- **Normal ECG ≠ no ACS** — unstable angina and early STEMI can have unremarkable tracings; serial ECGs + troponins.
- **Treating the monitor, not the patient** — artefact, not arrhythmia, is the commonest cause of a sudden "bizarre" rhythm on telemetry.

> **Clinical Pearl**
>
> The three questions that resolve most bedside dilemmas: **(1)** Is the patient stable? **(2)** What did the *old* ECG look like? **(3)** Does the ECG fit the story? If any answer worries you, escalate — a cardiologist would rather see ten false alarms than one missed occlusion.

APPENDIX A

Normal Values Quick Reference

Parameter	Normal value
Paper speed	25 mm/s (1 small square = 40 ms)
Calibration	10 mm/mV
Heart rate	60–100/min
P wave	<120 ms wide, <2.5 mm tall; upright in I, II, aVF
PR interval	120–200 ms
QRS duration	<120 ms
Pathologic Q	>40 ms wide or >25% of R, in ≥2 contiguous leads
ST segment	Isoelectric
QTc (Bazett)	<450 ms (men), <460 ms (women); >500 ms = high risk
QRS axis	−30° to +90°
STEMI STE (limb)	≥1 mm in ≥2 contiguous leads
STEMI STE (V2–V3)	≥2.5 mm men <40; ≥2.0 mm men ≥40; ≥1.5 mm women
SA node intrinsic rate	60–100/min
AV node / junctional rate	40–60/min
Ventricular escape rate	20–40/min
LVH Sokolow–Lyon	S(V1) + R(V5/V6) ≥35 mm
LVH Cornell	R(aVL) + S(V3) >28 mm men / >20 mm women
Atrial flutter rate	250–350/min (usually ~300; 2:1 → ventricular ~150)
SVT rate	150–250/min, regular, narrow
VT definition	≥3 consecutive ventricular beats ≥100/min, wide QRS
Sgarbossa (LBBB)	≥3 points = MI (5 / 3 / 2)

APPENDIX B

Abbreviations Glossary

Abbreviation	Meaning
ACLS	Advanced Cardiovascular Life Support
ACS	Acute coronary syndrome
AF	Atrial fibrillation
AV	Atrioventricular
AVNRT / AVRT	AV nodal / AV re-entrant tachycardia
BBB	Bundle branch block (RBBB/LBBB)
CHA_2DS_2-VASc	Stroke-risk score in AF
DOAC	Direct oral anticoagulant
LAD / LCx / RCA	Left anterior descending / left circumflex / right coronary artery
LAFB / LPFB	Left anterior / posterior fascicular block
LVH / RVH	Left / right ventricular hypertrophy
MAT	Multifocal atrial tachycardia
NSTEMI / STEMI	Non-ST-elevation / ST-elevation myocardial infarction
PAC / PVC	Premature atrial / ventricular contraction
PE	Pulmonary embolism
PEA	Pulseless electrical activity
SA	Sinoatrial
SVT	Supraventricular tachycardia
VF / VT	Ventricular fibrillation / tachycardia
WPW	Wolff–Parkinson–White syndrome