

Chapter 9: Introduction to the 12-Lead ECG and Infarction Patterns

Dr. Alice Matthews

amatthews@concorde.edu · Concorde Career College Portland

After this chapter you should be able to Describe the layout of the 12-lead electrocardiogram and name the plane each lead group views; apply the deflection rule and the vector concept to explain the normal appearance of every lead; assign each lead group to a wall of the heart and to the artery that usually feeds it; recognize the ischemia, injury and infarction progression and confirm it with reciprocal change; identify right and left bundle branch block from V1; recognize atrial enlargement and left ventricular hypertrophy; recognize the potassium and calcium patterns; acquire a correctly placed 12-lead; and analyze a 12-lead in a fixed order every time. Reading: Aehlert, *ECGs Made Easy*, 7th ed., Chapter 9 (p. 225 to 258).

Note to students: Values in this guide follow **Aehlert, 7th edition**, because that is the edition this guide follows. This chapter is the broadest in the book and the one where the internet disagrees with itself most. Two examples you will meet within a week of searching. First, the **normal QRS axis**: this guide and your textbook use -30° to $+90^{\circ}$, while many teaching sites publish 0° to $+90^{\circ}$, and that single difference is why so many quick axis methods over-call left axis deviation. Second, the **lead group mnemonic ISAL**: several widely-copied versions leave lead I out of the lateral group. Lead I is a lateral lead. Answer from the textbook, and read the corrections in this guide carefully, because they mark the exact places where a confident wrong answer is easy to find. **What the first eight chapters give you:** Chapter 2 gave you the deflection rule and the camera analogy, and this week is that one rule applied twelve times; Chapter 1 gave you the coronary arteries, and this week is where that anatomy finally tells you something about a patient.

At a Glance: this chapter in one box

Numbers you must know

- 10 electrodes produce **12 leads**. Limb leads view the **frontal** plane; chest leads the **horizontal** plane.
- Normal QRS axis -30° to $+90^{\circ}$.
- Pathologic Q wave: wider than **0.04 s** (1 small box) *or* deeper than **one third** the R wave beside it.
- ST deviation is measured at the **J point** against the **TP segment**. One small box is **1 mm, 0.1 mV**.
- STEMI requires elevation in **two or more anatomically contiguous** leads, not one.
- Bundle branch block: QRS **0.12 s or more** (3 small boxes) in a *supraventricular* rhythm.
- LVH, Sokolow-Lyon: S in V1 plus R in V5 or V6, **35 mm or more**.
- P pulmonale: P taller than **2.5 mm** inferiorly. P mitrale: P **0.12 s or wider**, notched.

The two ideas this chapter is built on

- **Twelve cameras**. Each lead is a camera on a tripod aimed at the heart from a different angle. Which way the camera points tells you which wall it photographs.
- **The deflection rule, from Chapter 2**. Depolarization moving *toward* a positive electrode writes **up**; moving *away* writes **down**; moving *perpendicular* writes little or nothing.

Top 3 traps

- Believing a normal 12-lead rules out a myocardial infarction. It does not, ever.
- Calling ST elevation in one lead a STEMI. Two contiguous leads, plus reciprocal change.
- Calling every Q wave pathologic. Small septal q waves in the lateral leads are normal.

You Already Know This: Chapters 1 and 2, Cashed In

Nothing here is a new physical principle. It is old principles applied to twelve views instead of one strip. Find the row you already own, and the right-hand column is this week's work.

What you already have	What Chapter 9 does with it
Deflection rule (Chapter 2)	Explains aVR, R wave progression, and the Q wave of dead muscle
Electrode versus lead (Chapter 2)	Ten stickers, twelve camera angles, one event
QRS wide at 0.12 s (Chapters 2, 6)	The entry gate for bundle branch block
Lead groups (Chapter 2)	Now attached to a wall <i>and</i> to an artery
Coronary arteries (Chapter 1)	Which vessel, and which rhythm problem to expect (Chapter 7)

Foundation One: Twelve Cameras on One Event

An electrode is a sticker. A lead is a view. Picture each lead as a **camera on a tripod**, planted at a fixed spot around the patient and aimed at the heart. Whichever way that camera points tells you which wall it photographs, and nothing else about it matters.

That one image collapses two lists into one idea. **"Twelve leads from ten electrodes"** stops being a paradox, because six of the twelve camera positions are calculated rather than wired. And **"different leads see different walls"** is the same fact stated from the other end: a camera at the left leg looking up photographs the **inferior** wall, and a camera on the left flank looking across photographs the **lateral** wall.

Now the payoff. A blocked artery starves the muscle it feeds and nothing else. The cameras aimed at that wall record the damage; the cameras aimed anywhere else record a completely normal heart, **because from where they are standing the heart is normal**. That is why one region of a 12-lead can be dramatically abnormal while the other nine leads look unremarkable, and why you read a 12-lead group by group rather than glancing at it whole.

Two planes, one printout. The six limb leads (I, II, III, aVR, aVL, aVF) view the **frontal plane**, top to bottom and side to side, like a photograph taken from in front of the patient. The six chest leads (V1 through V6) view the **horizontal plane**, front to back, like a slice through the chest seen from below. A standard 12-lead prints them as **four columns of three leads**, read left to right: I, II, III, then aVR, aVL, aVF, then V1, V2, V3, then V4, V5, V6, with one or more **continuous rhythm strips** along the bottom. Each of the twelve short segments is only about two and a half seconds long, nowhere near enough to judge regularity.

Take **rhythm** from the bottom strip. Take **anatomy** from the twelve short segments above it.

A full 12-lead runs **10 seconds**, so counting QRS complexes across the bottom strip means multiplying by **6**, not by 10. That is the Chapter 2 rule doing its job: multiply by however many of these windows fit into a minute.

Foundation Two: The Deflection Rule, and What a Vector Actually Is

Depolarization moving toward a positive electrode writes an upward deflection. Moving away from it writes a downward deflection. Moving perpendicular to it writes little or nothing, often a small biphasic wiggle.

You met that sentence in Chapter 2. Two payoffs prove it inside a single paragraph. **Why aVR is normally inverted:** the positive electrode of aVR sits at the right shoulder, and normal depolarization sweeps down and to the left, away from the right shoulder in every phase. So the P wave, the QRS and the T wave are all normally negative in aVR, and nothing is wrong with that patient. **Why R waves grow across the chest:** ventricular depolarization travels mostly leftward and posteriorly, largely away from V1 and largely toward V6. Apply the rule six times and the answer falls out. V1 shows a small r and a deep S, the complexes get progressively more upright as you walk left, and V6 shows a tall R. That is **R wave progression**, with the transition from mostly negative to mostly positive normally landing at **V3 or V4**. You did not memorize six appearances; you used one rule six times.

A vector is an arrow with two properties, and that is all it is. Its **direction** is where the electricity is heading at that instant. Its **length** is how much muscle is depolarizing, because more muscle means more current means a

bigger deflection. The tracing records the sum of thousands of those tiny arrows. The left ventricle has by far the most muscle, so its arrows dominate the sum, and that is the entire explanation for **why the normal axis points down and to the left**, toward the left leg and the apex. The **axis** is nothing more than the direction of that one summed arrow.

Third payoff, and it is the one this chapter is built on. Infarcted muscle is electrically dead and silent. A camera aimed at a dead wall no longer sees current coming toward it; it sees straight through the hole to the **opposite** wall depolarizing **away** from it. Away writes down, so the first deflection points downward, and that is a **pathologic Q wave**. A Q wave is a hole knocked in a wall.

Lead Groups and the Walls They Watch

Wall	Leads	Usual artery
Septal	V1, V2	LAD
Anterior	V3, V4	LAD
Lateral	I, aVL, V5, V6	Circumflex
Inferior	II, III, aVF	RCA in about 80 percent

aVR watches no wall at all, which is exactly what makes it useful as a quality check rather than an anatomic view.

Memory hook: “I See All Leads,” or ISAL: Inferior, Septal, Anterior, Lateral.

The mnemonic is worth far more as a **search order** than as a list. Look at each group in that fixed order, write down what you find, and only **then** go looking for reciprocal changes. Students who read a 12-lead by scanning for whatever catches the eye find the dramatic finding and miss the second one sitting next to it.

Correction you must carry with ISAL. Several published versions of this mnemonic list the lateral group as “aVL, V5, V6” and drop lead I. **Lead I is a lateral lead.** Include it. Leaving it out costs you the single most useful lead in the reciprocal-change pair you are about to learn.

The column-reading trick. Because the printout is always laid out the same way, reading straight down the four columns and skipping aVR gives you a fixed sequence of walls, every time, on every tracing:

Lateral, Inferior, Inferior, Lateral, Inferior, Septal, Septal, Anterior, Anterior, Lateral, Lateral

Memory hook: “Lucky Irish Immigrants Landed In Seattle, Seven Adventurous Alaskans Left Late.” Eleven words, eleven leads, in printout order with aVR skipped.

Coronary Mapping: Probability, Not Law

The **LAD** supplies the anterior wall and the anterior portion of the septum. The **circumflex** supplies the lateral wall. The **RCA** supplies the inferior wall in roughly **80 percent** of inferior infarcts, with the circumflex accounting for most of the rest in left-dominant people.

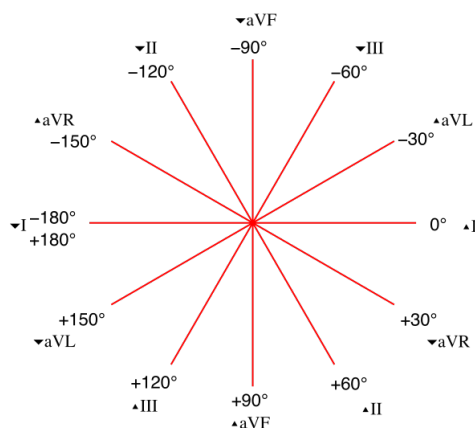
Say **usually**. Never say **always**. Coronary anatomy varies between people, and naming the occluded vessel is a clinical claim you cannot make from a tracing alone. What you can say is “the inferior leads are involved, so this is most likely the right coronary artery.”

Now the part that changes patient care. The RCA does not only feed the inferior wall. It also feeds the **SA node** in roughly 60 percent of people and the **AV node** in about 9 out of 10. So an **inferior infarct is not only a wall problem, it is a rate and rhythm problem**: the same occluded vessel starving inferior muscle is starving the tissue that sets the pace and the tissue that passes the impulse along. High-grade AV block occurs in about **19 percent** of acute inferior infarcts and is usually transient. When you see ST elevation in II, III and aVF, look at the monitor next and expect bradycardia, a junctional rhythm, or AV block. That is Chapters 1, 7 and 9 arriving at the same bedside.

Axis: Use Three Leads, Not Two

Normal axis runs from -30° to $+90^{\circ}$. The bedside method most students are taught uses two leads, I and aVF, and sorts the answer into four quadrants. It is fast, and in one of its four answers it is **wrong often enough to matter**.

Lead I	Lead aVF	Answer
Positive	Positive	Normal. Stop here.
Positive	Negative	Normal or left axis deviation. Check lead II.
Negative	Positive	Right axis deviation
Negative	Negative	Extreme axis deviation



The hexaxial reference system: six limb leads and their angular relationships. MoodyGroove, Public Domain, via Wikimedia Commons.

Why the second row needs a third lead. aVF sits at $+90^{\circ}$, so it flips negative the moment the axis passes 0° . But normal extends to -30° . That leaves a 30 degree band where aVF is already negative and the axis is still perfectly normal, and the two-lead method calls every one of those patients left axis deviation.

The fix is one more look. Lead II sits at $+60^{\circ}$, and the line perpendicular to it is exactly -30° , the real boundary. So when lead I is positive and aVF is negative, check lead II. **Positive lead II means still normal. Negative lead II means true left axis deviation.** Three leads, three seconds, and the over-call disappears.

Ischemia, Injury, Infarction: One Bullseye, Three Rings

Picture the damaged region as a **bullseye**. At the dead center is **infarction**, tissue that is gone. Around it, a ring of **injury**, tissue hurt but still alive. Around that, a ring of **ischemia**, tissue merely starved of oxygen. Each ring writes its own signature, and because all three exist at the same moment, **one lead can show a Q wave, ST elevation and T wave changes simultaneously**. That is not a contradiction; it is the bullseye seen from one camera angle.

Zone	ECG finding	Meaning	Timing
Ischemia (outer)	ST depression, T inversion	Starved but alive	Reversible
Injury (middle)	ST elevation	Happening right now	Acute
Infarction (center)	Pathologic Q wave	Established tissue death	Usually permanent

The burn. Red skin that still hurts and still heals is ischemia. A blister forming while you watch is injury. A scar that never goes away is the pathologic Q wave.

One honest addition your textbook diagram does not emphasize. The very first change in an acute coronary occlusion is usually **neither** ST elevation nor a Q wave. It is a **hyperacute T wave**: broad, tall, and asymmetrically peaked, appearing **before** the ST segment lifts. In a chest pain patient with big, fat, oddly prominent T waves and an otherwise unimpressive tracing, you may be looking at the first ten minutes of an infarct. It is also the finding most often dismissed as normal.

Not every Q wave is pathologic, and students over-call this all week. A Q wave qualifies as pathologic when it is **wider than 0.04 s** (one small box) **or deeper than one third the height of the R wave** in the same complex. Small, narrow septal q waves in the lateral leads (I, aVL, V5, V6) are normal on healthy people.

STEMI versus NSTEMI. A **STEMI** shows ST elevation in **two or more anatomically contiguous leads** and reflects a fully occluded artery, which is a cath lab activation. An **NSTEMI** shows no elevation, but has ST depression, T wave inversion, or even an unremarkable tracing, together with a positive troponin, and reflects a partial occlusion. The ECG separates them; the troponin confirms that an infarct happened at all.

Measuring ST Deviation: The J Point and the TP Segment

An ST measurement is a comparison between two places on the tracing, and you have to be right about both. **Measure at the J point**, the junction where the QRS ends and the ST segment begins. **Compare it against the TP segment**, the flat line from the end of the T wave to the start of the next P wave, which is the true isoelectric baseline. When the rate is fast enough that the TP segment has been squeezed out of existence, fall back on the **PR segment**. One small box of deviation is **1 mm**, which at standard gain is **0.1 mV**.

★ Measuring from the wrong baseline is how a normal tracing becomes an abnormal one on a lab sheet. Eyeball ST deviation against whatever line looks flattest and a wandering baseline will hand you 2 mm of elevation that is not there.

Reciprocal Changes: The See-Saw

Reciprocal change is the **mirror image**. A camera on the far side of the heart, looking at the same injured wall from the opposite direction, sees the identical event travelling the other way, so it writes the opposite deflection. Push one end of a see-saw up and the other end has to go down. Reciprocal ST **depression** in the opposite group is the confirmation that your ST **elevation** is real.

Rather than memorizing a whole system, learn **one pair properly**, because it does most of the clinical work:

Lead III and lead aVL are the truly reciprocal pair. They sit almost directly opposite one another in the frontal plane. Inferior ST elevation in II, III and aVF appears as reciprocal ST **depression in aVL** (and often lead I). Run the see-saw the other way and lateral elevation in I, aVL, V5 and V6 appears as reciprocal ST **depression in the inferior leads**.

aVL is the most sensitive lead for inferior infarction. Reciprocal depression in aVL shows up in nearly every inferior STEMI, frequently while the inferior elevation is still small enough to argue about. That is exactly why dropping lead I and aVL out of your lateral group costs you something real.

Reciprocal depression also tells you what this is not. Acute pericarditis classically produces **diffuse** ST elevation across many lead groups **without** reciprocal depression anywhere, often with PR segment depression. Elevation plus reciprocal depression argues for occlusion; elevation everywhere with nothing going down argues against it. If you cannot find reciprocal change anywhere, take a hard second look before anyone says the word STEMI.

Common Trap

Your patient has crushing chest pain and diaphoresis. The 12-lead is normal. Have you ruled out a myocardial infarction?

No. A normal 12-lead never rules out an acute myocardial infarction. This is the single most dangerous belief in this chapter. Reasons a genuinely infarcting patient prints a normal tracing:

- **Too early.** You caught it before the ST segment moved. Hyperacute T waves may be the only clue, and they are easy to call normal.
- **NSTEMI.** By definition there is no ST elevation; the infarct is confirmed by troponin, not by the tracing.
- **A blind spot.** The standard twelve leads do not directly face the posterior wall or the right ventricle, so an isolated posterior infarct can look like nothing at all.
- **Existing LBBB or a paced rhythm**, which distorts the ST segments so much that ordinary criteria no longer apply.

This is the camera analogy earning its keep. **A camera that photographs nothing does not prove nothing happened. It**

proves that camera could not see that corner. It is the entire reason serial ECGs and serial troponins exist.

Common Trap

You find a mnemonic called PAILS for predicting reciprocal changes. Should you learn it?

No. PAILS (Posterior, Anterior, Inferior, Lateral, Septal, each supposedly giving reciprocal change in the next) circulates widely and is partly wrong, which is worse than being obviously wrong because it fails quietly.

- “Lateral gives Septal” is contradicted by primary sources, which state that lateral infarcts produce reciprocal depression in the inferior leads.
- “Anterior gives Inferior” holds only sometimes, depending on where the lesion sits along the LAD.
- It mixes the frontal and horizontal planes as though leads in different planes were opposite one another, which teaches a wrong mental model that is hard to unlearn.

Learn the two solid pairs instead, as one see-saw read in both directions: inferior elevation gives depression in aVL and lead I, and lateral elevation gives depression in the inferior leads. Two facts, anatomically true, same plane, covering the great majority of what you will be asked.

Intraventricular Conduction Delays: The Bundle Branch Blocks

Gate the question before you answer it. Bundle branch block is only on the table when **both** of these are true:

1. The rhythm is supraventricular, meaning it started above the ventricles, so there is a P wave driving it.
2. The QRS is 0.12 s or wider, which is 3 small boxes or more.

If the rhythm is ventricular, the wide QRS is already fully explained by Chapter 6 and bundle branch terminology does not apply. Students who skip the gate end up labelling ventricular tachycardia as “left bundle branch block,” which is a serious error.

Why you look at V1. V1 sits on the chest wall over the **right** side of the heart. When one bundle is blocked, that ventricle cannot be activated down its own highway; it has to wait for the impulse to cross over from the other side and crawl through muscle. **The blocked ventricle therefore depolarizes last, and the terminal portion of the QRS points toward it.** Apply the deflection rule once more and it resolves: terminal current heading toward V1 writes up, terminal current heading away from V1 writes down.

The turn signal method. In V1, find the J point and step back **one small box** into the QRS. If the tracing there sits **above** the baseline, the terminal deflection is up: **right** bundle branch block. If it sits **below** the baseline, the terminal deflection is down: **left** bundle branch block. Up is right, down is left, exactly like a turn signal.

Three caveats, or the turn signal will mislead you. It works in V1 **only**, not in lead II, V6, or whatever monitoring lead you happen to have up. You must read the **terminal** portion of the QRS rather than the tallest wave, because in right bundle branch block the second R prime is often shorter than the first R and students who look for “the big one” get the answer backwards. And it sits **downstream of the two gates**: supraventricular rhythm first, 0.12 s second, turn signal third.

About WiLLiaM MaRRoW. You will meet this mnemonic, so here it is: WiLLiaM says a W in V1 and an M in V6 is LBBB; MaRRoW says an M in V1 and a W in V6 is RBBB. Now the accuracy caveat, which matters because students try to shape-match literally and then decide they cannot read ECGs. **In left bundle branch block, V1 is typically an rS or QS, which looks far more like a V than a W. In right bundle branch block, the lateral leads show a wide slurred S that looks more like an N than a W.** Use it only as a hook for **which lead carries the late, messy complex**, never as a literal shape test.

★ Right bundle branch block is common and is often found in people with entirely normal hearts. Left bundle branch block is more often a marker of structural disease, and it distorts the ST segments enough that ordinary infarct criteria no longer apply to that tracing.

Chamber Abnormalities: Tall Means Right, Wide Means Left

The atrial half is one timing story, not two rules. **The right atrium depolarizes first and the left atrium second**, and the P wave you see is the two of them overlapping.

Finding	What changes	Criterion	Name
Right atrial enlargement	The first half gets taller	Greater than 2.5 mm in II, III, aVF	P pulmonale
Left atrial enlargement	The second half gets longer	0.12 s or wider , notched	P mitrale

Right atrial enlargement is an **amplitude** criterion because the change lands on the first hump, the peak of the P wave. Left atrial enlargement is a **duration** criterion because the change lands on the second hump, pushing the tail out and separating the humps into a visible notch. **P mitrale is shaped like an M, and the M is literally in the name.**

Left ventricular hypertrophy. The classic voltage criterion is **Sokolow-Lyon**: the depth of the S in V1 plus the height of the R in V5 or V6, **35 mm or more**. More muscle means a longer vector arrow means taller complexes.

★ **Voltage alone is not hypertrophy.** Amplitude depends on how far the electrode sits from the heart, so a thin, athletic 19-year-old routinely meets voltage criteria with an entirely normal heart. What separates real hypertrophy from a thin chest is the **strain pattern**: down-sloping ST depression with asymmetric T wave inversion in the **lateral** leads (I, aVL, V5, V6). Voltage plus strain is meaningful. Voltage alone in a slim young person is usually just a slim young person.

Common Trap

You see a tall R wave in V1. Is that right ventricular hypertrophy?

Not automatically, and this is the single most consequential over-call in the chapter. A tall R in V1 means the terminal or dominant forces are heading anteriorly and to the right. Right ventricular hypertrophy is only one of the things that does that. The differential you must be able to list:

- **Posterior infarction.** V1 and V2 are the mirror leads for the posterior wall, so a posterior Q wave shows up as a tall R and posterior ST elevation shows up as ST depression. **This is the one that hurts patients**, because it is an infarct being read as a hypertrophy.
- **Right bundle branch block**, which puts a late R prime in V1 by design.
- **Wolff-Parkinson-White**, where a right-sided accessory pathway pre-excites the ventricle from the wrong direction.
- **Hypertrophic cardiomyopathy**, where septal hypertrophy redirects early forces.
- **Normal variant**, particularly in young people and children.
- **Lead misplacement**, above all V1 and V2 placed too high on the chest.

Teaching the finding without the differential is how you get a student who confidently misreads a posterior infarct. **A tall R in V1 is a question, not an answer.**

Electrolyte Disturbances: One Gesture, Both Directions

Here is the whole potassium chapter in one movement of your hand. **Hyperkalemia PULLS the T wave up.** **Hypokalemia PUSHES the T wave down.** Make the gesture while you say it; that single image is correct in both directions and will survive an exam room.

Hyperkalemia pulls, and as potassium climbs it drags the rest of the tracing with it in a fixed order. Learn the order, because the order tells you how sick the patient is:

1. **Peaked T waves.** Tall, narrow, symmetric, pointed. The earliest change.
2. **PR prolongation, then loss of P waves.** The atria go quiet.
3. **QRS widening.** The complex smears out and loses its edges.
4. **Sine wave.** The widened QRS merges into the T wave into one continuous rolling waveform. This immediately precedes cardiac arrest.

Peaked T waves in a dialysis patient are an emergency, not a curiosity, because every stage above is on the same escalator.

Hypokalemia pushes. The T wave flattens, then inverts, ST depression appears, and as the T wave sinks the U wave emerges from behind it. The U wave has always been there; low potassium simply removes the T wave that was hiding it.

Calcium sits on a see-saw with the QT interval. Calcium up, QT down. Calcium down, QT up. **Hypocalcemia prolongs the QT specifically by stretching the ST segment**, leaving the T wave itself normal in shape and duration, so the T just sits further from the QRS than it should. Hypercalcemia does the reverse and shortens the ST segment until the T seems to start almost on top of the QRS.

Honest caveat: the ECG is not a potassium meter. The *rate of rise* matters more than the absolute level. A dialysis patient who lives at a chronically high potassium may show an unremarkable tracing, while a patient whose potassium climbed quickly can show dramatic changes at a much more modest number. **A normal ECG never excludes hyperkalemia.** Treat the tracing as a reason to act, never as a reason to relax.

Common Trap

Two traps live in the electrolyte section, and both of them get tested.

Trap one: steeple or balloon? Two different emergencies both produce a tall, pointed T wave, and the difference is the *shape of the base*, not the height.

- **Hyperkalemia:** tall, **narrow, symmetric**, pointed, **narrow base**. A **church steeple**. You could pitch a tent on it.
- **Hyperacute infarction:** **broad, asymmetric, wide base**. A **hot air balloon**. Fat rather than sharp.

Steeple sends you toward potassium. Balloon sends you toward the cath lab. Look at the base of the T wave, not the tip.

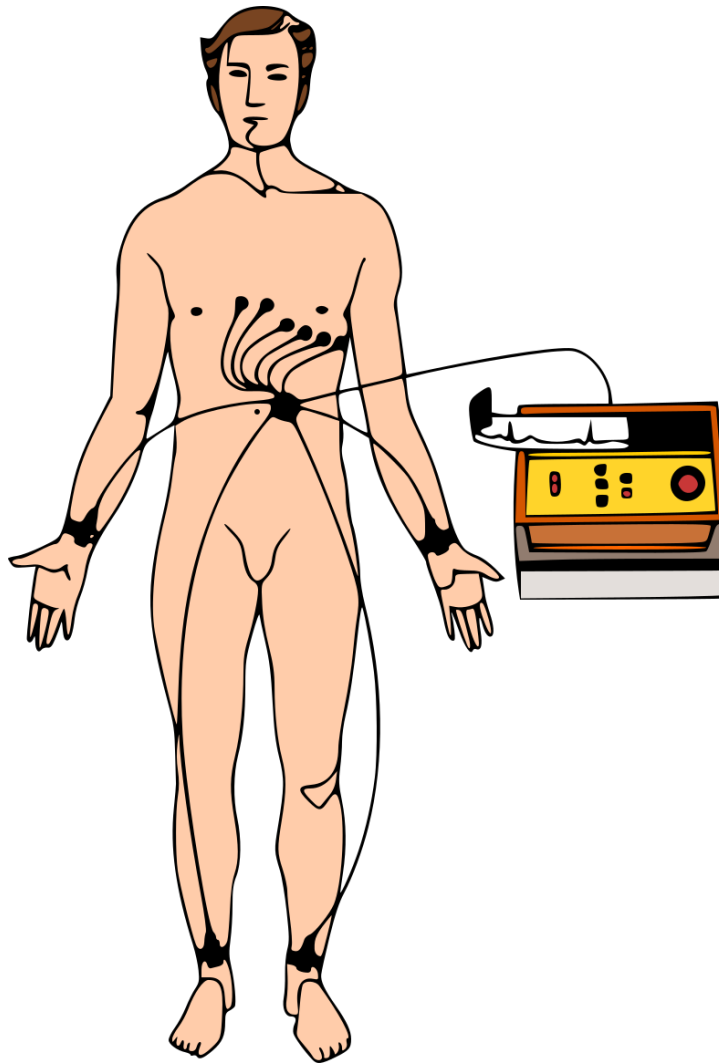
Trap two: is that a prolonged QT, or a QU? In hypokalemia the QT does **not** truly lengthen. The flattened T wave and the emerging U wave **fuse**, and the fused pair looks like one very long T wave. Measure to the end of that and you have measured a **QU interval** and reported it as a prolonged QT. This is one of the most common student errors on a lab sheet. Find the notch where the T ends and the U begins, and measure to the end of the T.

Electrode Placement: The Most Job-Relevant Section in This Course

Everything above assumes the cameras are where you think they are. They frequently are not.

Find the sternal angle first, every time. Put a finger in the **suprasternal notch**, the soft dip at the top of the sternum, and slide **down about an inch and a half** until you feel a horizontal ridge across the bone. That ridge is the **sternal angle**, also called the **Angle of Louis**, and it is where the **second rib** attaches. Slide **laterally** along that rib, and the gap immediately **below** it is the **second intercostal space**. Now walk down: third space, fourth space. Counting down from the clavicle instead is the classic way to end up one whole space off.

Lead	Location	Order
V1	Fourth intercostal space, right sternal border	1st
V2	Fourth intercostal space, left sternal border	2nd
V4	Fifth intercostal space, midclavicular line	3rd
V3	Midway between V2 and V4	4th
V5	Anterior axillary line, <i>level with V4</i>	5th
V6	Midaxillary line, <i>level with V4</i>	6th



Twelve-lead electrode placement: limb electrodes and precordial leads V1 through V6. Madhero88, Public Domain, via Wikimedia Commons.

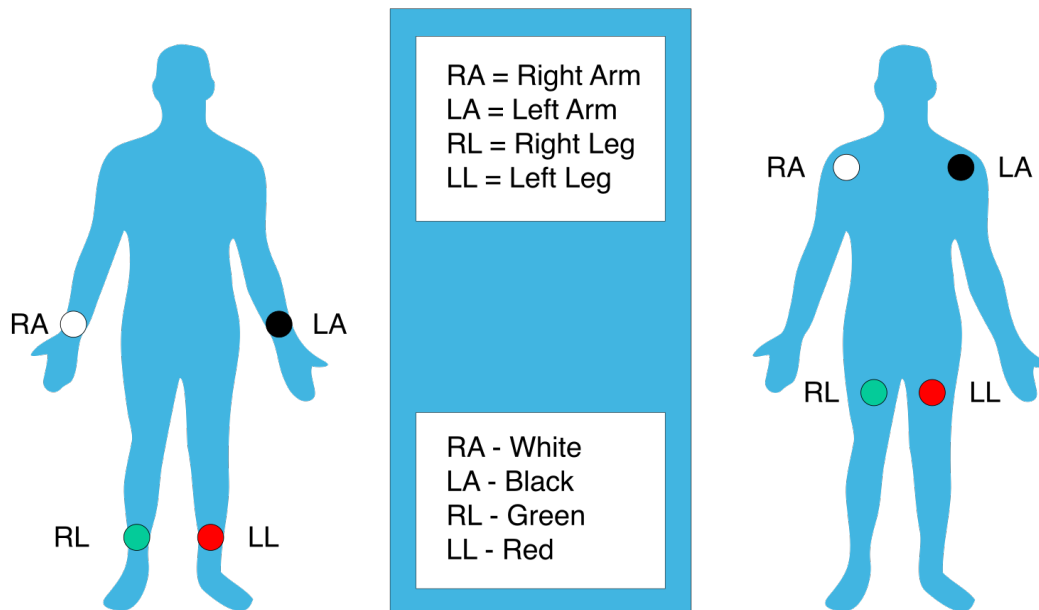
- **Place V4 before V3.** V3 is *defined* as the midpoint between V2 and V4, so it cannot be placed until V4 exists. Placing V3 first is how students end up with V3 in the wrong space and a distorted transition zone.
- **V5 and V6 go level with V4, horizontally.** They follow the rib line out toward the axilla; they do not step down. Students who count down another rib space for each one build a **staircase** across the chest and change the horizontal plane the leads are supposed to share.
- **Place V3 through V6 under the left breast, on the chest wall, not on top of breast tissue.** Tissue between the electrode and the heart attenuates the signal and shifts the apparent position.
- **Do not use nipples as landmarks.** Nipple position varies enormously between people and tells you nothing reliable about which intercostal space you are in. Limb electrodes go on **fleshy tissue, off bony prominences**, symmetrically on the two sides.

The consequence that should make you care: pseudo-septal infarction. Placing V1 and V2 **too high**, in the second or third intercostal space, fabricates findings on a completely healthy heart: **false Q waves, ST elevation**, false rSR prime patterns, an inverted P wave in V1 and V2, and poor R wave progression. Those get read as an acute or old anteroseptal infarct, and the patient inherits that reading in their chart. **Studies have found electrodes misplaced in up to 64 percent of cases, and one study found fewer than 20 percent of cardiologists placed V1 and V2 correctly.** Treat those as “studies have found,” not fixed rates, but treat the message as settled: misplacement is common, and experienced people commit it too.

★ **Lead reversal, and the fastest quality check you own.** An **upright P-QRS-T in aVR** on an otherwise ordinary tracing means the arm electrodes are probably reversed. Do not begin interpreting. Fix the electrodes and reacquire.

Separating arm reversal from dextrocardia takes one more look, at the chest leads. Arm reversal does not touch the chest electrodes, so **R wave progression stays normal**. Dextrocardia moves the heart itself, so **progression reverses too** and the R waves shrink from V1 out to V6. Limb leads abnormal with chest leads normal means somebody swapped two wires; both reversed means the heart is genuinely on the other side.

Know the four limb electrodes by colour, because that is how you catch a reversal. The figure below shows both accepted placements: distal on the limbs, or up on the torso at the shoulders and hips. Either is acceptable so long as you are **symmetrical**, but the two are not interchangeable within one tracing, and moving electrodes between recordings on the same patient makes those tracings unusable for comparison.



Limb electrode placement and the **AHA** colour code: RA white, LA black, RL green, LL red. *Left*: distal placement. *Right*: torso placement. **Caution**: this is the AHA code used in the United States. The **IEC** code used in much of the rest of the world is different (RA red, LA yellow, RL black, LL green), so never identify a lead by colour alone on unfamiliar equipment. MoodyGroove; Twisp (SVG), Public Domain, via Wikimedia Commons.

Memory hook: **White on the right**, and **smoke over fire** (black above red) down the left side. Green is the ground, and it goes on the leg that is left over.

Analyzing the 12-Lead: The Same Order, Every Single Time

A dramatic ST segment pulls your eye across the page and you will not notice the wide QRS sitting beside it. The only defense is a fixed order, run whether the tracing looks interesting or not.

1. **Check the tracing before you read it.** Standardization pulse two large boxes tall, paper speed 25 mm/s, no wandering baseline or tremor, aVR inverted. Upright aVR means stop and fix the electrodes.
2. **Rate and rhythm from the bottom strip**, using the Five-Question Method, not from the short segments above.
3. **Axis**, with the three-lead method. Lead I, then aVF, then lead II if you need it.
4. **Intervals.** PR, QRS, QT. Write values in seconds, not adjectives.
5. **Chambers.** P waves for enlargement, voltage for hypertrophy.
6. **ST segments and T waves, group by group, in ISAL order.** Write down what each group shows before you move on.
7. **Q waves**, applied against the two criteria, not by impression.
8. **Only now, reciprocal changes**, and then one summary sentence: which wall, which artery, and what rhythm problem to watch for on the monitor.

That last sentence is the whole book in one line, and it is exactly what a good interpretation sounds like: “inferior wall, most likely right coronary artery, and I am watching for bradycardia and AV block.”

Before You Move On

You should now be able to:

- say what each lead group looks at and which artery usually supplies it
- apply the deflection rule to a twelve lead and explain R wave progression
- localize an infarct by lead group and recognize reciprocal change
- recognize the potassium and calcium patterns, and tell a hyperkalemic T wave from a hyperacute one
- place chest electrodes correctly and explain what a high V1 and V2 do to the tracing

The last chapter pulls the whole book together into one comparison you can revise from.

Dr. Alice Matthews · amatthews@concorde.edu · alice@deepneuro.ai · Concorde Career College Portland