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Veterinary ECG Interpretation

How to Read an Animal ECG Step by Step

Veterinary ECG interpretation is a skill every clinician can learn, because a rhythm strip only ever shows three things: a P wave (the atria depolarising), a QRS complex (the ventricles depolarising) and a T wave (the ventricles rep

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IN SHORT

Veterinary ECG interpretation is a skill every clinician can learn, because a rhythm strip only ever shows three things: a **P wave** (the atria depolarising), a **QRS complex** (the ventricles depolarising) and a **T wave** (the ventricles repolarising) — the surface record of the heart's action potentials. Read them with a fixed **six-step method** — rate, rhythm, a P for every QRS, the intervals, the waveform shape, then the axis — and you can name the rhythm in almost any species. This guide covers how the ECG is recorded, the normal complex and its intervals, the systematic approach, the common arrhythmias, and the crucial way ECGs differ between dogs, cats and large animals.

KEY POINTS

1. **Veterinary ECG interpretation** rests on three waves: the **P wave** is atrial depolarisation, the **QRS complex** is ventricular depolarisation, and the **T wave** is ventricular repolarisation — the surface trace of the heart's action potentials.
2. The ECG records only **spreading** depolarisation or repolarisation (a moving electrical dipole); when the heart is uniformly at rest or uniformly depolarised the trace is **flat (isoelectric)**.
3. Read the **intervals**: the **PR interval** (~0.13 s in a dog) is AV-node conduction time, the **QRS** (<0.1 s) is ventricular depolarisation, and the **QT** (~0.2 s) approximates the ventricular action-potential duration.
4. Use a fixed **six-step method** on every trace — **rate, rhythm, a P for every QRS, the intervals, the waveform shape, then the axis** — and the rhythm names itself.
5. **Lead II** (right fore negative, left hind positive) is the everyday rhythm lead; standard paper speed is 25 or **50 mm/s** (faster for cats), and 1 mV calibrates to 10 mm.
6. Learn the pattern of each arrhythmia: **AV block** (long PR, dropped or dissociated QRS), **premature complexes** (early beats, wide for ventricular), and **atrial fibrillation** (no P waves, an *irregularly irregular* QRS).
7. In **large animals** the ECG waveforms vary widely between individuals, so the ECG is used to diagnose **arrhythmias, not chamber enlargement** — unlike in dogs and cats.

01 Why ECG interpretation matters in veterinary medicine

Veterinary ECG interpretation looks intimidating until you realise a rhythm strip can only ever tell you three things — and once you can name them, you can read a trace in almost any species. An animal collapses, or its heart sounds irregular on auscultation, and the electrocardiogram (ECG) answers the questions a stethoscope cannot: **how fast is the heart going, is the rhythm regular, and is the electrical impulse travelling normally?** It is the single best tool for diagnosing an arrhythmia, and it does something no other bedside test can: it shows the heart's electrical activity beat by beat.

What the ECG does *not* do is show contraction directly, or reliably measure chamber size — that is the job of auscultation, radiographs and echocardiography. The ECG records **electricity**. Keep that firmly in mind and the whole subject simplifies, because the electricity of every heartbeat produces the same three landmarks on the trace. This guide builds veterinary ECG interpretation from the ground up: what the ECG records, how it is recorded, the normal complex and its intervals, a fixed step-by-step method for reading any strip, the common arrhythmias and how they look, and the important ways the ECG differs between dogs, cats and large animals.

02 What a veterinary ECG records

The heart sits in a body full of salty, conductive fluid, so the electrical currents produced as it beats spread all the way to the skin, where two electrodes can pick up the tiny voltage differences between them. That is all an ECG is: a graph, against time, of the voltage produced as action potentials sweep through the heart muscle. Crucially, a voltage appears only while depolarisation or repolarisation is **spreading** — a moving wave sets up an electrical **dipole** (a separated + and – charge) that the electrodes detect. When the whole myocardium is uniformly at rest, or uniformly depolarised, there is no dipole and the trace sits flat on its **isoelectric** baseline.

Follow one beat and the three waves fall out naturally. The SA node fires and the impulse sweeps across the atria — that spreading atrial depolarisation is the **P wave**. The impulse then crawls through the AV node, which is far too small a mass of tissue to make its own wave, so the trace stays flat (this silent pause is the PR segment). The impulse reaches the ventricles and their large muscle mass depolarises — that is the big **QRS complex**. Finally the ventricles repolarise, producing the **T wave**. Atrial repolarisation happens too, of course, but it is slow, involves little muscle and is buried inside the QRS, so it makes no visible wave of its own. Three events, three waves: **P (atria depolarise), QRS (ventricles depolarise), T (ventricles repolarise)**.

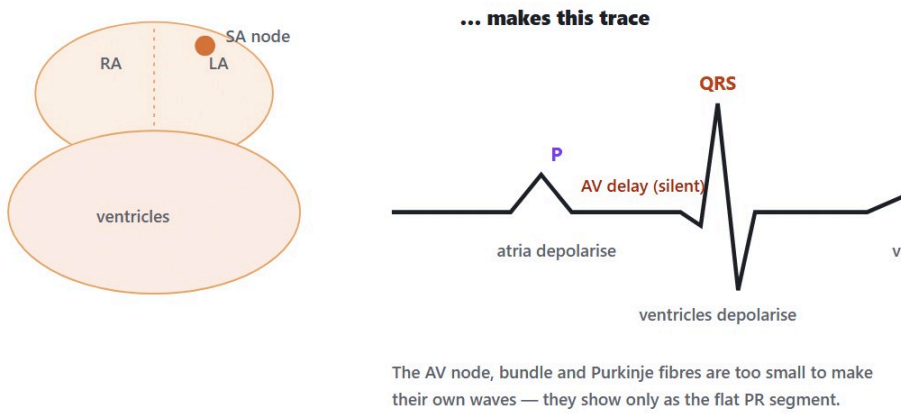


Fig 1 — How the heart's electrical events map to the ECG. Atrial depolarisation makes the P wave, the AV node delays silently, ventricular depolarisation makes the QRS, and ventricular repolarisation makes the T wave.

03 Recording the ECG: leads, Einthoven's triangle & calibration

Because the signal is a difference in voltage between two points, an ECG is always recorded through a **lead** — a specific pair (or arrangement) of electrodes. Attach electrodes to the two forelimbs and the left hind limb and you have the corners of **Einthoven's triangle**, an imaginary equilateral triangle framing the heart. From it come the three **bipolar limb leads**: **Lead I** (right forelimb negative, left forelimb positive), **Lead II** (right forelimb negative, left hind limb positive) and **Lead III** (left forelimb negative, left hind limb positive). Because the limbs behave simply as extensions of the electrode wires, the exact spot on the limb does not matter. Three more **augmented** leads (aVR, aVL, aVF) view the same frontal plane from different angles and are used routinely in veterinary practice; the **precordial** (chest) leads exist too but are used far more in human than in animal medicine.

For everyday rhythm reading, **Lead II is the workhorse**, because the normal depolarisation travels roughly along its axis and produces tall, clear P waves and QRS complexes. Two conventions make the trace measurable. Vertically, the machine is **calibrated** so that 1 millivolt deflects the trace 10 mm — a square calibration pulse is usually printed on the strip. Horizontally, the **paper (or sweep) speed** is standardised at **25 mm/s**, or **50 mm/s** to spread out the complexes in a fast-hearted animal such as a cat. Knowing the speed lets you turn distance into time, and time into rate.

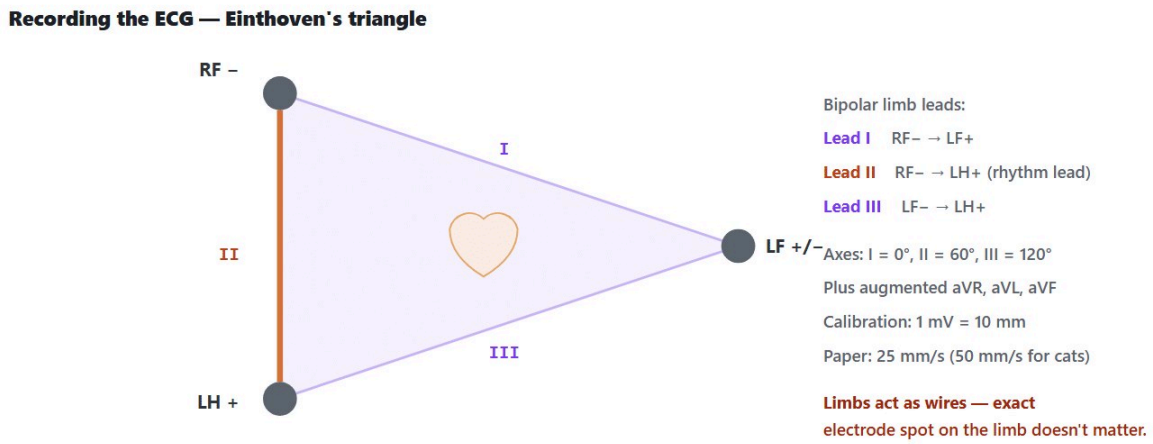


Fig 2 — Recording the ECG. The two forelimb and one hind-limb electrodes form Einthoven's triangle; the bipolar leads I, II and III view the heart from three angles, with Lead II (right fore – to left hind +) the standard rhythm lead.

04 The P–QRS–T complex and what each wave means

Look closely at a single normal complex and each deflection has a physical cause. The **P wave** is a small, usually upright bump as the atria depolarise from right to left. The **QRS complex** is the trio that follows: a small downward **Q** wave as the septum depolarises left-to-right, then the large upright **R** wave — the dominant feature of a normal trace — as both ventricular walls depolarise, and finally a small downward **S** wave as the last of the ventricle activates. The R wave points up and to the left for two reasons: the heart's axis is tilted toward the left chest wall, and the left ventricle is far more muscular than the right, so its electrical dipole dominates. That is why abnormal R-wave size or polarity is such a useful clue.

The **T wave**, ventricular repolarisation, is the one that surprises students: it can be normally positive *or* negative, and it varies from animal to animal, because — unlike depolarisation — the direction repolarisation takes is not fixed. A negative T wave is not automatically abnormal. What matters clinically is a *change* in the T wave, or in the flat **ST segment** between the QRS and the T: an ST shift off the baseline points to ischaemic (ischemic) or injured ventricular muscle that can no longer hold a normal resting charge.

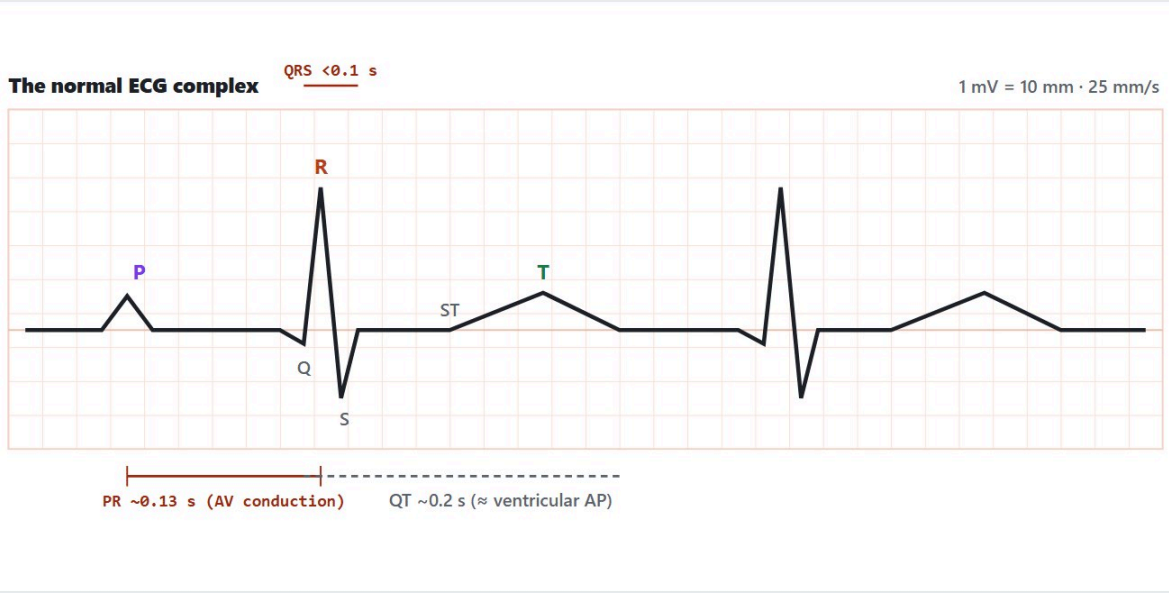


Fig 3 — The normal ECG complex on grid paper: the P wave, the Q, R and S of the QRS, and the T wave, with the PR interval (AV conduction), the QRS duration, the ST segment and the QT interval marked.

05 Intervals, measurements and calculating heart rate

Three intervals turn the ECG from a picture into a set of numbers. The **PR interval**, from the start of the P wave to the start of the QRS, is the time the impulse takes to cross the AV node — normally about **0.13 s in a dog** (under 0.14 s). A long PR interval means slowed AV conduction. The **QRS duration**, normally **under 0.1 s**, is how long the ventricles take to depolarise; a widened QRS means the impulse is travelling slowly or by an abnormal route (as in a ventricular ectopic beat). The **QT interval**, from the start of the Q wave to the end of the T wave, is roughly **0.2 s** and approximates the duration of the ventricular action potential itself.

Heart rate comes straight off the strip. Measure the **R–R interval** (the time between successive R waves) in milliseconds and divide it into 60,000: an R–R of 0.6 s gives 60,000 ÷ 600 = **100 beats per minute**. In a normal heart the atrial rate (measured from the P–P interval) equals the ventricular rate, because every atrial impulse crosses to the ventricles — and one of the first questions an arrhythmia asks is whether that is still true.

The measurements worth carrying in your head, for a dog at 25 mm/s and 10 mm/mV.

MEASUREMENT	NORMAL (DOG)	WHAT IT REPRESENTS	WHAT AN INCREASE MEANS
PR interval	~0.13 s (under 0.14)	Time for the impulse to cross the AV node	AV conduction is slowing — first-degree AV block
QRS duration	under 0.1 s	Time for the ventricles to depolarise	The impulse is travelling slowly or by an abnormal route — a ventricular ectopic, or hyperkalaemia
QT interval	~0.2 s	Approximates the duration of the ventricular action potential	Varies with rate; read it against the heart rate, never alone
Mean electrical axis	about 70°	Average direction of ventricular depolarisation	A shift suggests chamber enlargement or a conduction block. The cat varies widely and the axis is far less useful

06 A systematic approach to reading any veterinary ECG

The secret to confident veterinary ECG interpretation is to abandon pattern-guessing and always work through the **same six steps**, in the same order, on every trace. Do this and even an unfamiliar rhythm reveals itself.

1. **Rate.** Is the ventricular rate normal, fast (tachycardia) or slow (bradycardia) for the species? Calculate it from the R–R interval.
2. **Rhythm.** Is the R–R interval regular, or irregular? If irregular, is it patterned (as in sinus arrhythmia) or chaotic (*irregularly irregular*, as in atrial fibrillation)?
3. **The P–QRS relationship.** Is there a P wave before every QRS, and a QRS after every P? This single question separates most of the important arrhythmias.
4. **Intervals.** Measure the PR, QRS and QT. Is the PR too long (AV block)? Is the QRS too wide (ventricular origin)?
5. **Waveform morphology.** Are the P waves, QRS complexes and T waves normal in shape and amplitude, or are some early, wide or bizarre?
6. **Mean electrical axis.** The average direction of ventricular depolarisation (about 70° in the dog; the cat varies widely). A shifted axis suggests a change in ventricular mass or conduction.

The first three steps — rate, rhythm and the P-for-every-QRS check — identify the great majority of clinical arrhythmias on their own. The intervals, morphology and axis refine the diagnosis. Run the same drill every time and you will not miss the dropped beat, the early complex or the missing P wave.

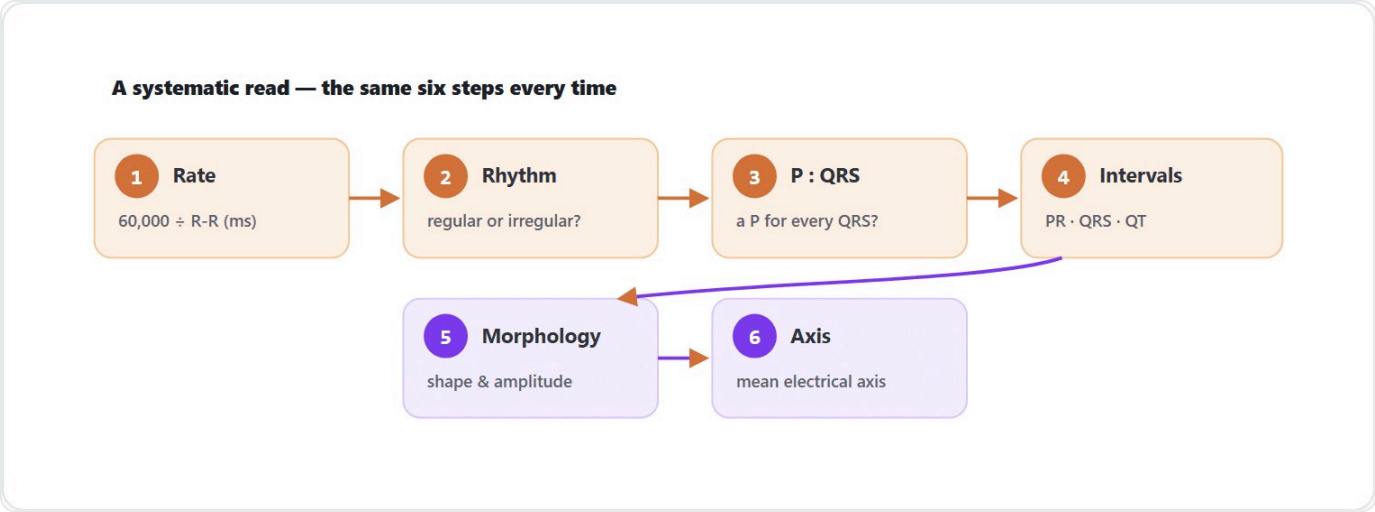


Fig 4 — The six-step systematic method for reading any ECG: rate, then rhythm, then the P-for-every-QRS relationship, then the intervals, the waveform morphology and finally the mean electrical axis.

07 Normal rhythms and normal variants

Before you can spot an abnormal rhythm you must know the normal ones cold. **Normal sinus rhythm** is what you are hoping to see: a regular rhythm at a species-appropriate rate, with an upright P wave before every QRS, a QRS after every P, a fixed PR interval and narrow QRS complexes. The impulse starts in the SA node and travels normally — everything in its place.

The most important *normal variant*, especially in dogs, is **respiratory sinus arrhythmia**: a cyclic speeding of the heart on inspiration and slowing on expiration. It is driven by the vagus nerve — vagal tone falls as the lungs inflate, so the SA node speeds up — and it is a sign of a *healthy*, vagally-toned heart. It is the commonest irregular rhythm in dogs, is especially prominent in young dogs, and it disappears when the animal exercises and sympathetic tone takes over. Mistaking this normal finding for pathology is a classic beginner's error; recognising that the underlying complexes are entirely normal (a P for every QRS) is what tells you it is benign.

08 Bradyarrhythmias: when the ECG slows or blocks

A pathologically slow heart is either failing to *generate* impulses or failing to *conduct* them. **Sinus bradycardia** is simply a slow but otherwise normal sinus rhythm; it is common in very fit animals and with hypothermia. Failure of the SA node itself (sinus arrest, sick sinus syndrome) produces long pauses with no P waves at all.

Failure of conduction at the AV node is **AV block**, and reading its three degrees is a core ECG skill. In **first-degree AV block** every impulse still gets through, but slowly — the trace looks normal except that the **PR interval is abnormally long**. In **second-degree AV block** some P waves are *not* followed by a QRS: you see a P wave with a flat line after it where the beat should be — a dropped beat. In **third-degree (complete) AV block** nothing crosses the AV node, so the P waves and the QRS complexes march completely independently (AV dissociation), and the ventricles are driven by a slow **escape pacemaker** — a dog in complete block may run a ventricular rate of only about 20–40 bpm while its atria fire at a normal 140. Complete block, and second-degree block that drops enough beats to lower cardiac output, are serious and may need a pacemaker.

09 Tachyarrhythmias and ectopic beats

At the other extreme, the ECG can show a heart going too fast or beating out of turn. A **premature complex** is a beat that arrives early from an ectopic focus. A **premature atrial complex (APC)** is an early P wave followed by a normal-looking QRS — because it still uses the normal ventricular conduction pathway. A **premature ventricular complex (VPC)** is more sinister-looking: it arrives early, has *no* preceding P wave, and is **wide and bizarre**, because it starts in ordinary ventricular muscle and spreads the slow way rather than through the Purkinje system; it is usually followed by a compensatory pause. A sustained run of these wide, P-less complexes at speed is **ventricular tachycardia** — a dangerous rhythm that can degenerate into ventricular fibrillation, in which the trace becomes irregular chaos and the heart pumps no blood at all.

Above the ventricles, rapid atrial rhythms include **supraventricular tachycardia** and, importantly, **atrial fibrillation**. In atrial fibrillation the atria are not contracting but quivering chaotically, so there are **no true P waves** — just a wobbly, low-amplitude **f-wave** baseline — and the AV node is bombarded with random impulses, letting some through and blocking others. The result is normally-shaped QRS complexes spaced

irregularly irregularly: the hallmark of AF. It is common in horses and in large-breed dogs, and in horses is classically converted back to sinus rhythm with **quinidine**.

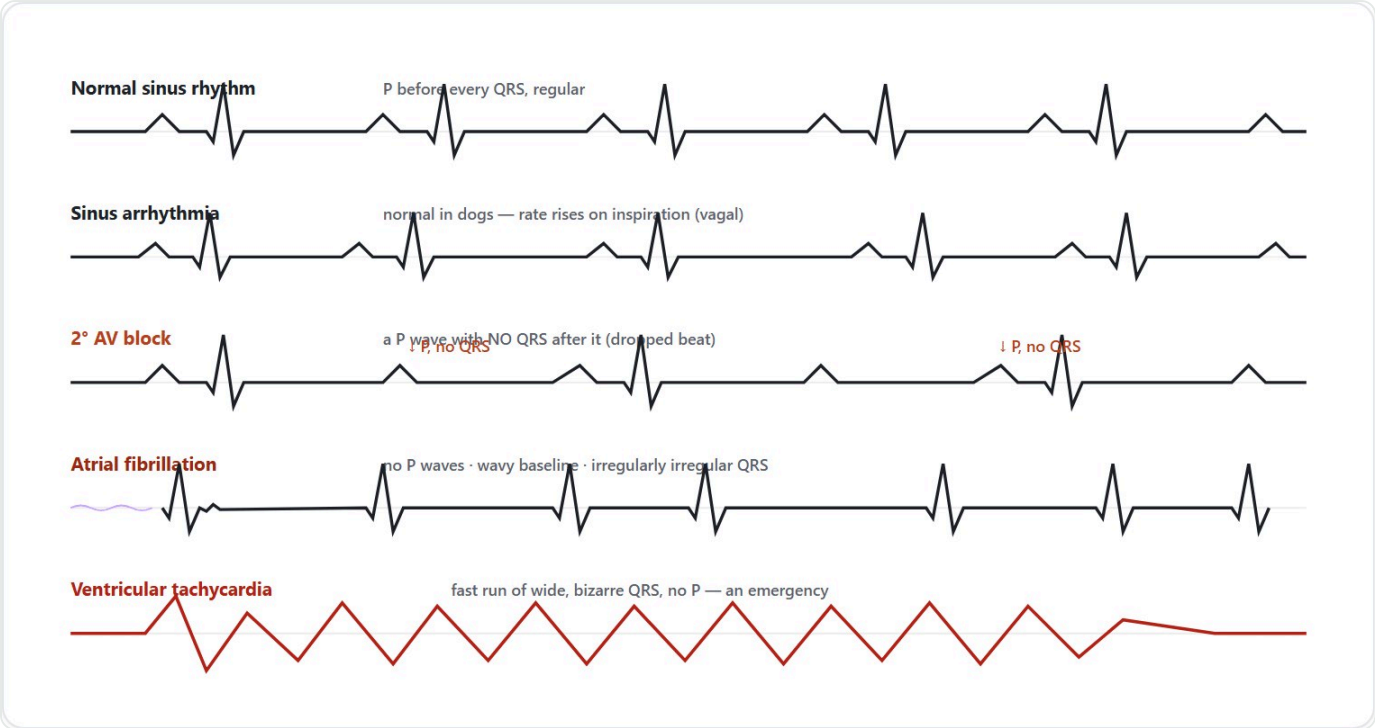


Fig 5 — A rhythm-strip gallery: normal sinus rhythm; respiratory sinus arrhythmia (normal in dogs); second-degree AV block (a P with no following QRS); atrial fibrillation (no P waves, irregularly irregular); and ventricular tachycardia (fast, wide, bizarre complexes).

10 The metabolic ECG: potassium, and what is not a rhythm at all

Not every abnormal trace is an arrhythmia. Two things routinely change the ECG without any primary heart disease: the plasma potassium, and the recording itself.

Hyperkalaemia (hyperkalemia) has a recognisable sequence, and it is worth knowing cold because the ECG is often faster than the laboratory. As potassium climbs the T wave becomes **tall, narrow and peaked**; the P wave then flattens, widens and finally disappears altogether — **atrial standstill**, a slow regular rhythm with no P waves at all, sometimes called a sinoventricular rhythm; the **QRS widens** as conduction slows; and finally QRS and T merge into a **sine wave** before **asystole**. The classic patients are the blocked male cat, the dog with hypoadrenocorticism, and any patient with acute kidney injury or a ruptured bladder. Intravenous **calcium gluconate** is the first move: it does not lower potassium, it protects the myocardium while insulin and dextrose or fluids do the lowering. **Hypokalaemia** is subtler — prolonged repolarisation, ST depression, and a greater tendency to ectopy, particularly alongside digoxin.

Escape beats are a rescue, not a fault. When the sinus node fails or the AV node blocks, a lower pacemaker fires to keep the heart going: a junctional escape at roughly 40–60/min in the dog, a ventricular escape slower still and with a wide, bizarre QRS. The critical point is that an escape beat arrives *late*, after a pause, whereas a premature complex arrives *early*. Suppressing escape beats with an antiarrhythmic because they look ventricular is a genuine way to kill a patient — they are the only thing maintaining cardiac output.

Artefact (artifact) is the commonest reason a practice ECG is misread. Purring in the cat and panting or muscle tremor in the dog produce fine, irregular baseline wobble that can be mistaken for atrial fibrillation; shivering does the same. Movement of a limb or a loose clip gives sudden large deflections that mimic ectopic beats. Regular fine oscillation at 50 or 60 Hz is **mains electrical interference** — check the earthing, unplug nearby equipment, and make sure the patient is not touching metal. The tests are simple: an artefact usually does not disturb the underlying rhythm, so look for the regular R–R marching through the noise, and if in doubt, re-record with better contact and a still, comfortable patient before you diagnose anything.

11 Species differences: dogs, cats, horses and cattle

Here is the point that trips up anyone who learned ECGs on dogs and then meets a horse. In **small animals** the shape of the normal complex is fairly consistent, so an experienced eye can read chamber enlargement from wave amplitudes — a tall R wave suggesting left-ventricular hypertrophy, a right-shifted axis suggesting right-sided disease. In **large animals** that consistency vanishes: in horses and cattle the polarity

and amplitude of the ECG waves vary widely from individual to individual, because cardiac depolarisation follows **less-consistent paths** through their atria and ventricles. Two healthy cows can produce quite different-looking QRS complexes.

The clinical consequence is a rule worth memorising: **in large animals the ECG is used to diagnose arrhythmias, not chamber size.** It is unreliable for detecting hypertrophy or enlargement in horses and cattle — that is what echocardiography is for — but because the *sequence* of electrical events is the same in every mammal, the ECG remains excellent for detecting and characterising rhythm disturbances. For that purpose you do not even need standardised lead placement: any lead that gives clear P waves, QRS complexes and T waves will do. Rates differ too, and you should carry the normals: roughly **60–170 bpm in dogs, 140–220 in cats, 28–44 in horses and 60–80 in cattle** — and remember that a resting horse's strong vagal tone often produces an entirely *normal* dropped beat (physiological second-degree AV block) that vanishes the moment it moves.

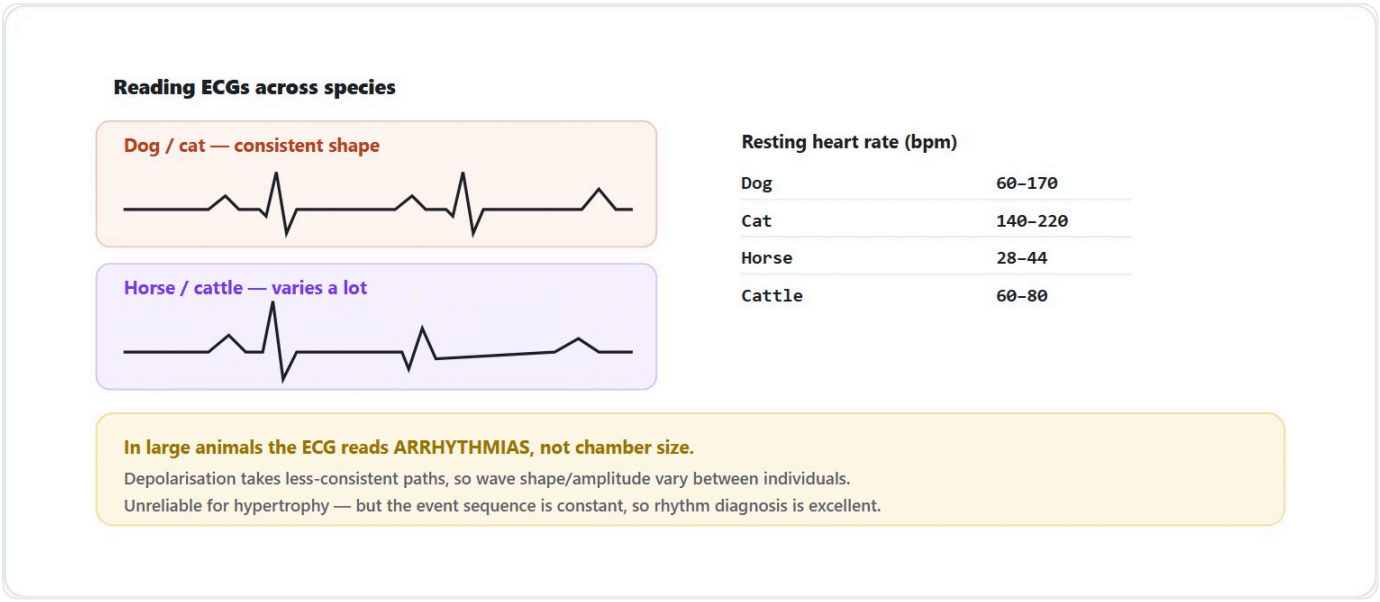


Fig 6 — Reading ECGs across species. Dog and cat complexes are consistent in shape; horse and cattle traces vary between individuals, so the large-animal ECG diagnoses arrhythmias rather than chamber size. Typical resting rates are shown.

12 When to act on an ECG

A systematic read is only useful if it changes what you do. Some findings are true emergencies: **ventricular tachycardia**, especially fast or with beats landing on the previous T wave, can collapse into fibrillation; **complete (third-degree) AV block** or a symptomatic bradycardia can drop cardiac output enough to cause collapse and often needs a pacemaker; and any rhythm accompanied by **syncope, weakness or a pulse deficit** deserves urgent attention. Atrial fibrillation, while rarely an immediate threat itself, usually signals underlying heart disease that needs working up.

Equally, the great value of veterinary ECG interpretation is the confidence to *not* over-react — to recognise respiratory sinus arrhythmia in a bright young dog, or a resting horse's physiological dropped beat, as entirely normal. Read every trace the same way — rate, rhythm, a P for every QRS, then the intervals, the shape and the axis — keep the species normals in mind, and use the ECG for what it does best: naming the rhythm and guiding the next step.

> **Red flag**
>
> Ventricular tachycardia, complete AV block, a sudden fixed bradycardia, or any arrhythmia with collapse, weakness or a pulse deficit is an emergency — obtain an ECG, stabilise the patient and refer for rate/rhythm control or pacing.

13 Quiz: test yourself

Sixteen questions across calibration, the intervals, the systematic read, the arrhythmias and the species normals. Answers are beneath each question — cover them and work down.

1. What is an ECG lead?**Answer:** A **specific pair or arrangement of electrodes**. The signal is a difference in voltage between two points, so it is always recorded through a lead.2. What are the standard calibration settings?**Answer: 1 mV deflects the trace 10 mm** vertically — a square calibration pulse is printed on the strip — and the paper runs at **25 mm/s**, or **50 mm/s** to spread out the complexes in a fast-hearted animal

such as a cat.3. What does the P wave represent?**Answer: Atrial depolarisation** — the impulse spreading from the SA node across the atria.4. What is a normal canine PR interval, and what does it measure?**Answer:** About **0.13 s**, under 0.14. It is the time the impulse takes to **cross the AV node**.5. What is a normal QRS duration, and what does a wide QRS mean?**Answer:** Under **0.1 s**. A widened QRS means the impulse is **travelling slowly or by an abnormal route** — as in a ventricular ectopic beat.6. What does the QT interval approximate?**Answer:** The **duration of the ventricular action potential** itself, roughly **0.2 s**.7. What is the mean electrical axis in a dog?**Answer:** About **70°** — the average direction of ventricular depolarisation. The cat varies widely.8. What is respiratory sinus arrhythmia, what drives it, and what does it signify?**Answer:** A cyclical variation in rate with breathing, driven by the **vagus nerve** — vagal tone falls as the lungs inflate, so the SA node speeds up. It is a sign of a **healthy, vagally-toned heart**.9. What rate does a junctional escape run at in a dog, and a ventricular escape? **Answer:** A junctional escape at roughly **40–60/min**; a ventricular escape is **slower still**, with a wide, bizarre QRS.10. What is ventricular tachycardia, and why does it matter?**Answer:** A sustained run of **wide, P-less complexes at speed**. It can degenerate into **ventricular fibrillation**, in which the trace becomes irregular chaos and the heart pumps no blood at all.11. You see a regular fine oscillation at 50 or 60 Hz. What is it?**Answer: Mains electrical interference** — not a rhythm. Check the earthing, unplug nearby equipment, and make sure the patient is not touching metal.12. What are normal resting heart rates for dog, cat, horse and cattle?**Answer:** Roughly **60–170** bpm in dogs, **140–220** in cats, **28–44** in horses and **60–80** in cattle.13. A resting horse drops a beat. Is that abnormal?**Answer:** Usually **not**. A resting horse's strong vagal tone commonly produces a **physiological second-degree AV block** — an entirely normal dropped beat.14. Why does atrial fibrillation matter even when it is not an immediate threat?**Answer:** Because it **usually signals underlying heart disease** that needs working up.15. Which ECG findings are true emergencies?**Answer: Ventricular tachycardia** — especially fast, or with beats landing on the previous T wave, because it can collapse into fibrillation; **complete (third-degree) AV block** or a symptomatic bradycardia, which can drop cardiac output enough to cause collapse and often needs a pacemaker; and any rhythm with **syncope, weakness or a pulse deficit**.16. What is the other half of the value of reading an ECG well?**Answer:** The confidence **not to over-react** — to recognise respiratory sinus arrhythmia, a physiological block in a horse, or mains interference for what they are, and leave them alone.

14 Veterinary ECG interpretation — frequently asked questions

What do the P, QRS and T waves mean on a veterinary ECG?

The P wave is atrial depolarisation, the QRS complex is ventricular depolarisation, and the T wave is ventricular repolarisation. Atrial repolarisation makes no visible wave — it is small, slow and hidden within the QRS.

How do you read a veterinary ECG step by step?

Use the same six steps every time: check the rate, then the rhythm (regular or irregular), then whether there is a P for every QRS, then measure the intervals (PR, QRS, QT), then the waveform shape, then the mean electrical axis.

Why is the ECG less useful for chamber size in horses and cattle?

In large animals cardiac depolarisation follows less-consistent paths, so wave polarity and amplitude vary widely between individuals — unreliable for detecting hypertrophy. The event sequence is constant, though, so the ECG is still excellent for diagnosing arrhythmias.

What does hyperkalaemia look like on an ECG?

In sequence as the potassium rises: tall, narrow, **peaked T waves**; then flattening and loss of the **P wave** (atrial standstill, giving a slow regular rhythm with no P waves); then a **widening QRS**; then a sine wave and asystole. Think of it immediately in a blocked cat, a collapsed dog with suspected hypoadrenocorticism, or a uroabdomen. Give intravenous calcium first to protect the heart, then lower the potassium.

How do you tell an ECG artefact from a real arrhythmia?

Look for the underlying rhythm marching through the noise. Artefact from purring, panting, tremor or a loose clip usually leaves the R-R intervals regular and the complexes identifiable between the wobbles, whereas a true arrhythmia disturbs the timing itself. Regular fine oscillation at 50 or 60 Hz is mains interference, not the patient. If the trace is ambiguous, re-record it — better contact, a still patient and a quiet surface resolve most of it, and no arrhythmia should be diagnosed from a trace you could not repeat.

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When to refer

- Sustained **ventricular tachycardia** (fast, wide, bizarre QRS), or beats landing on the previous T wave — a pre-fibrillation emergency; treat and refer urgently.
- **Complete (third-degree) AV block** or a symptomatic fixed bradycardia (e.g. a dog at ~30–40 bpm) — often needs a pacemaker; refer.
- An **irregularly-irregular** rhythm with a pulse deficit (atrial fibrillation) — work up the underlying heart disease and refer for rate control or conversion.
- Any arrhythmia with **collapse, syncope, weakness or exercise intolerance** — a cardiac emergency; stabilise and refer.
- Frequent premature complexes (APCs/VPCs) — characterise them on a full ECG and investigate the cause before treating.

Sources

1. Stephenson RB. The Electrocardiogram. In: Klein BG, ed. Cunningham's Textbook of Veterinary Physiology. 6th ed. St Louis: Elsevier; 2020. Chapter 20 (pp 203–215): the ECG as a surface record of spreading depolarisation dipoles; P/QRS/T = atrial depolarisation / ventricular depolarisation / ventricular repolarisation; the PR (~0.13 s), QRS (<0.1 s) and QT (~0.2 s) intervals; Einthoven's triangle and the bipolar limb leads; 25/50 mm/s and 1 mV = 10 mm calibration; heart-rate calculation; sinus tachy/bradycardia, 1°/2°/3° AV block, premature complexes, ventricular tachycardia and atrial fibrillation; the large-animal ECG-variability rule (arrhythmias, not chamber size).
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