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PHYSIOLOGY · STUDY GUIDE

The Cardiac Action Potential

How the Heart Generates Every Heartbeat

Every heartbeat begins as an electrical event: a cardiac action potential. It comes in two flavours.

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IN SHORT

Every heartbeat begins as an electrical event: a **cardiac action potential**. It comes in two flavours. **Working muscle cells** of the atria and ventricles fire a long **'fast-response'** action potential with a distinctive **calcium plateau** that holds the cell depolarised for ~200–300 ms — roughly a hundred times longer than a nerve impulse — and a matching long **refractory period** that makes the heart physically unable to tetanise, so it must relax and refill between beats. The **SA node** and the rest of the conduction system fire a **'slow-response'** action potential that has *no resting potential*: it drifts spontaneously up to threshold (the **pacemaker potential**), which is why the heart beats on its own. Read those two waveforms and you understand rate, rhythm, the ECG, and how every antiarrhythmic drug works.

KEY POINTS

1. Every heartbeat starts as a **cardiac action potential**; the heart is an **electrical syncytium** — gap junctions let the impulse spread cell-to-cell so a whole chamber depolarises as one.
2. **Working muscle cells fire a 'fast-response' AP** with five phases: a fast **Na^+** upstroke, a long **Ca^{2+} plateau**, then **K^+** repolarisation back to a stable −80 mV.
3. The plateau makes the AP **~200–300 ms long** (about 100× a nerve impulse) and gives an equally long **refractory period** — so cardiac muscle **cannot tetanise** and always relaxes and refills between beats.
4. The Ca^{2+} that enters during the plateau triggers **calcium-induced calcium release** from the sarcoplasmic reticulum — the link between the action potential and the contraction.
5. **Pacemaker cells fire a 'slow-response' AP** with **no resting potential**: they drift spontaneously to threshold (the **pacemaker potential**, driven by the funny current I_f and Ca^{2+}), and their upstroke is **Ca^{2+}-driven, not Na^+**.
6. The **SA node** drifts fastest, so it sets the pace; the impulse then follows **SA → AV (atrioventricular) node → bundle of His → Purkinje → ventricles**, with an **AV nodal delay** (~0.1 s) that lets the atria finish filling the ventricles.
7. If the SA node fails, slower **escape pacemakers** (AV node, then Purkinje) take over — the further down the hierarchy, the slower the rate.
8. **Sympathetic** nerves (β_1) steepen phase 4 → faster rate and stronger contraction; the **vagus** (M_2) flattens it → slower rate — and **antiarrhythmic drugs** act on the very ion channels that shape the AP.

01 No action potential, no heartbeat

A heart taken out and placed in a warm dish, with every nerve to it cut, keeps beating. Cut a nerve to a skeletal muscle and the muscle falls silent — but the heart marches on. That single observation captures what makes cardiac physiology different: the heartbeat is not commanded from outside, it is generated from within, and it begins every single time as an electrical event — a **cardiac action potential**.

Understand that impulse and you understand the pulse you feel, the trace on the ECG, why some hearts race or block or fibrillate, and how every drug in the cardiology formulary does its job.

The cardiac action potential is a close relative of the nerve and skeletal-muscle action potential — the same ions, the same kinds of channel — but the heart has bent it to two special purposes, and so it comes in two distinct forms. The **working muscle cells** of the atria and ventricles fire a long **"fast-response"** action potential built around a calcium plateau; the specialised cells of the **pacemaker and conduction system** fire a **"slow-response"** action potential that has no resting state and drives itself. This article works through both, then follows the impulse along its wiring and shows how the nervous system and the drug box reach in and change the beat.

02 The heart as an electrical syncytium

Before the impulse, the wiring. Cardiac muscle is striated like skeletal muscle, but its cells are **short and branched** rather than long fibres, and they are joined end-to-end at structures called **intercalated discs**. Packed into those discs are **gap junctions** — protein channels that bridge the membranes of two neighbouring cells and let ions flow directly from one to the next. The consequence is profound: when one cell depolarises, the current spills straight into its neighbours, and the wave sweeps cell-to-cell across the whole muscle mass. The heart therefore behaves as a **functional syncytium** — not one giant cell, but a population wired so tightly that a chamber depolarises and contracts as a single unit. This is the exact opposite of skeletal muscle, where each fibre (fiber) is insulated from its neighbours and must be told to fire individually by its own lower motor neuron.

There are two such syncytia, one for the atria and one for the ventricles, and they are deliberately kept apart. A ring of connective tissue, the **anulus fibrosus**, sits between them and carries no gap junctions at all, so it acts as an **electrical insulator**. The only way an impulse can legally cross from atria to ventricles is through the conduction system — a detail that turns out to be central to the timing of the beat, and to the commonest conduction diseases. Finally, about 99% of cardiac cells are ordinary **contractile cells** that sit quietly at a stable resting potential until they are stimulated; the remaining 1% are **autorhythmic** cells that never sit still — and it is these that give the heart its automatic rhythm.

03 The working-muscle action potential (fast response)

Start with the ordinary muscle cell of a ventricle. At rest its interior sits at about **−80 mV**, held there — exactly as in nerve — by a membrane far more permeable to potassium than to sodium, with the Na^+/K^+ pump maintaining the gradients in the background. When a wave of depolarisation arrives through the gap junctions and lifts the cell to threshold, it fires an action potential in **five phases**:

- **Phase 0 — the upstroke.** Voltage-gated **fast Na^+ channels** fly open and sodium pours in, swinging the interior sharply positive. This is the same explosive sodium spike that drives a nerve impulse.
- **Phase 1 — a brief dip.** Within a couple of milliseconds the fast Na^+ channels inactivate; a small potassium efflux begins to repolarise the cell — and then something unusual interrupts it.
- **Phase 2 — the plateau.** Slow, **L-type Ca^{2+} channels** open, and calcium flows steadily inward at the same time as potassium exit falls. The two roughly balance, and the membrane is held depolarised on a long, near-flat plateau for around **200 ms**. Nothing like this exists in nerve or skeletal muscle, and it is the signature of the cardiac action potential.
- **Phase 3 — repolarisation.** The Ca^{2+} channels close and potassium channels reopen; K^+ efflux now dominates and carries the membrane back down toward rest.
- **Phase 4 — the resting potential.** The cell returns to and holds its stable −80 mV, waiting for the next wave.

The headline number is the duration. Where a nerve impulse is over in **1–2 ms**, the whole cardiac action potential lasts **~200–300 ms — about a hundred times longer** — entirely because of that calcium plateau. (Atrial cells run a briefer, less flat version, which is why the atria can beat faster than the ventricles.) That length is not an accident; it is the key to everything that follows.

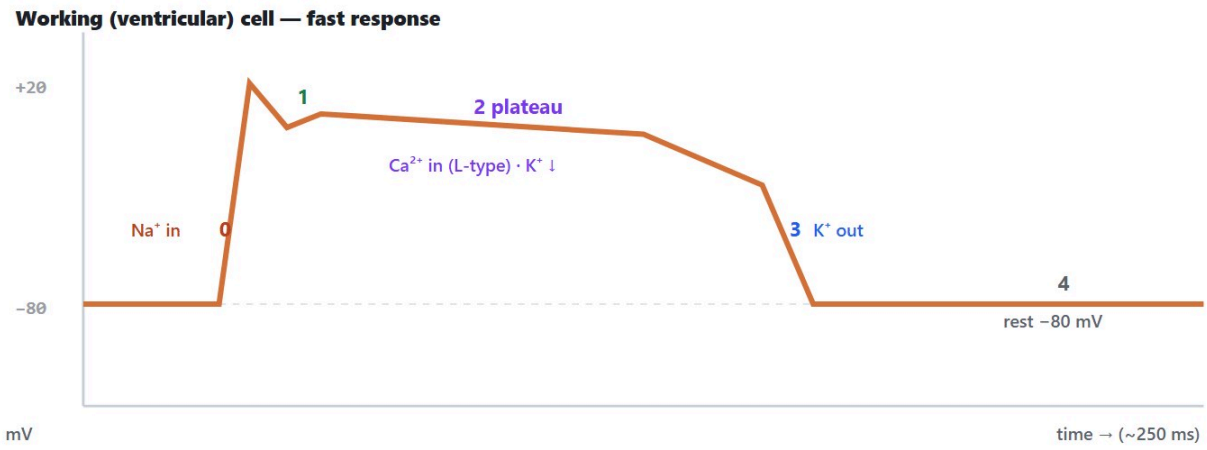


Fig 1 — The fast-response action potential of a working ventricular cell: a fast Na^+ upstroke (phase 0), a brief notch (1), the long L-type Ca^{2+} plateau (2), K^+ repolarisation (3) and the stable −80 mV rest (4).

04 The plateau, the refractory period, and why the heart cannot tetanise

Why build such a long action potential? Because a pump must never seize. Recall that a skeletal muscle can be driven into a smooth, sustained **tetanus**: its action potential is so brief (and its refractory period so short) that a train of impulses arrives before the previous twitch has relaxed, and the twitches fuse into one maintained contraction. That is exactly what you want from a muscle holding a posture — and exactly what would be fatal in a heart, which can only pump if it is allowed to relax and refill between beats.

The cardiac plateau solves this. Because the action potential lasts ~200 ms, so does the **absolute refractory period** — the interval during which the Na^+ channels are inactivated and no new action potential can be started. It runs for almost the entire length of the contraction, so by the time a second impulse could possibly fire, the first beat has already begun to relax. Cardiac twitches therefore **cannot summate and cannot tetanise**: every beat is a single, complete contraction followed by a guaranteed period of relaxation and filling. The very feature that makes the cardiac action potential odd is the safety catch that keeps the animal alive.

The plateau does a second job. The calcium that floods in through the L-type channels is not just electrical — it is the trigger for contraction. That modest influx sets off **calcium-induced calcium release**: it opens the release channels of the sarcoplasmic reticulum, which dumps a much larger store of Ca^{2+} into the cytosol, raising free calcium roughly **a thousand-fold in under a tenth of a second** and driving the crossbridge cycle. Unlike skeletal muscle, where the T-tubule voltage sensor is physically coupled to the SR, the heart depends on this trigger calcium actually entering from outside — which is why cardiac force is so sensitive to extracellular calcium and to drugs that block calcium channels. When the channels close, pumps haul the calcium back into the SR and out of the cell, and the muscle relaxes.

05 The pacemaker action potential (slow response)

Now the cells that make the heart automatic. A pacemaker cell in the **sinoatrial (SA) node** breaks the first rule of the working cell: it has **no stable resting potential**. The moment one action potential ends, at about **−65 mV**, the membrane immediately begins to drift back up on its own, slowly and steadily, until it reaches a threshold of about **−40 mV** and fires again. That spontaneous upward creep is the **pacemaker potential** (phase 4), and it is the reason the heart beats without being told to.

Three ion movements build the drift. As an action potential ends the potassium channels that repolarised the cell gradually **close**, so the outward brake fades. At the same time a special inward current switches on — the aptly named **"funny" current, I_f**, carried by pacemaker channels that let sodium leak in (they are "funny" because, unlike ordinary channels, they open on *hyper*polarisation). And as the cell climbs nearer to threshold, **L-type Ca^{2+} channels** begin to open, adding an inward calcium current that accelerates the final approach. Together they carry the membrane up to threshold, whereupon the cell fires.

The upstroke itself is the other big difference. A pacemaker cell has **no fast Na^+ channels**; its phase-0 depolarisation is carried by the relatively **slow L-type Ca^{2+} channels**. The result is a rounded, unhurried upstroke — which is precisely why these are called **slow-response** action potentials (and the working-cell version the **fast-response**). There is no plateau and no phase 1 or 2 to speak of; a pacemaker action

potential is essentially just phases 4, 0 and 3. Both the pacemaker potential and the upstroke are, at heart, calcium events — a fact that explains why calcium-channel-blocking drugs act so powerfully on the nodes.

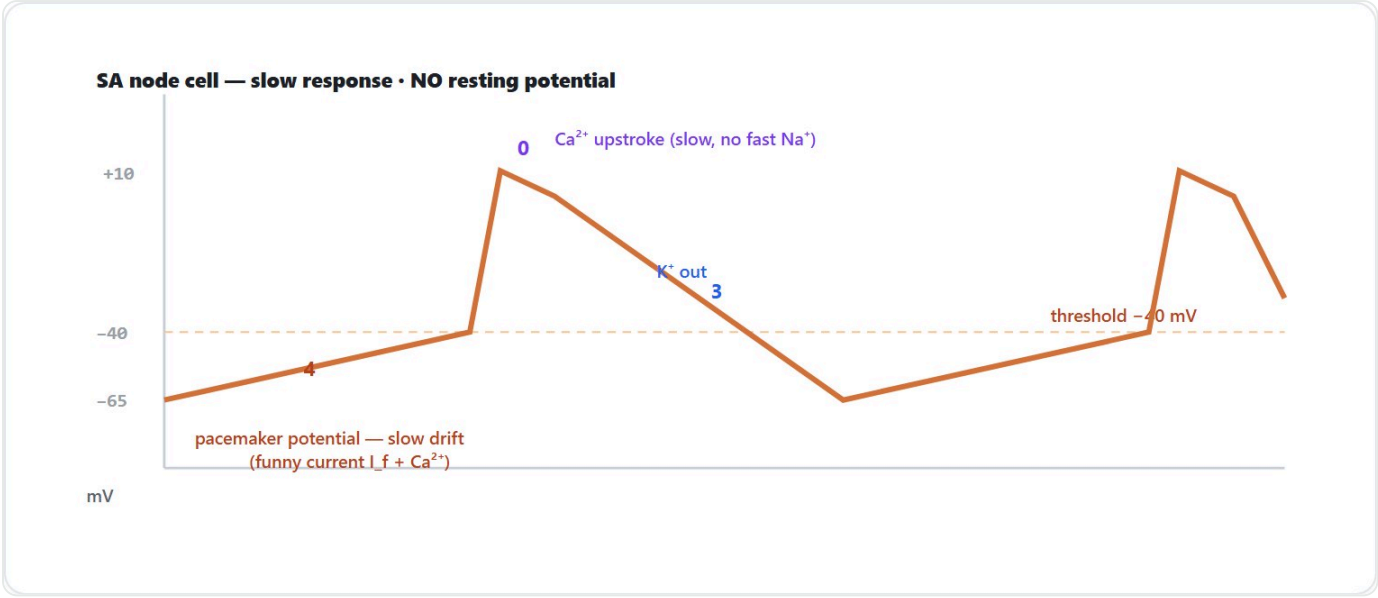


Fig 2 — The slow-response pacemaker action potential of an SA-node cell. With no resting potential, the phase-4 pacemaker potential drifts from ~−65 mV up to the ~−40 mV threshold (the funny current I_f plus calcium), then a slow Ca^{2+}-driven upstroke (0) and K^+ repolarisation (3).

Study tip Two waveforms, one contrast: the working cell has a *stable* baseline and a *Na^+* upstroke; the pacemaker has a *drifting* baseline and a *Ca^{2+}* upstroke. Get that pair straight and the whole chapter unlocks.

06 Fast versus slow response, side by side

It helps to see the two action potentials against each other, because almost every clinical fact about rate and rhythm falls out of their differences. The working cells give a big, fast, plateaued spike from a fixed resting line; the nodal cells give a small, slow, self-starting wave from a line that never settles.

FEATURE	FAST RESPONSE (WORKING MUSCLE)	SLOW RESPONSE (PACEMAKER)
Where	Atrial & ventricular myocytes	SA node & AV node
Resting potential	Stable, ~−80 mV	None — drifts (pacemaker potential)
Phase-0 upstroke	Fast Na^+ channels	Slow L-type Ca^{2+} channels
Upstroke speed	Fast	Slow
Plateau	Yes (long Ca^{2+} plateau)	No
Automaticity	No (must be stimulated)	Yes (fires spontaneously)

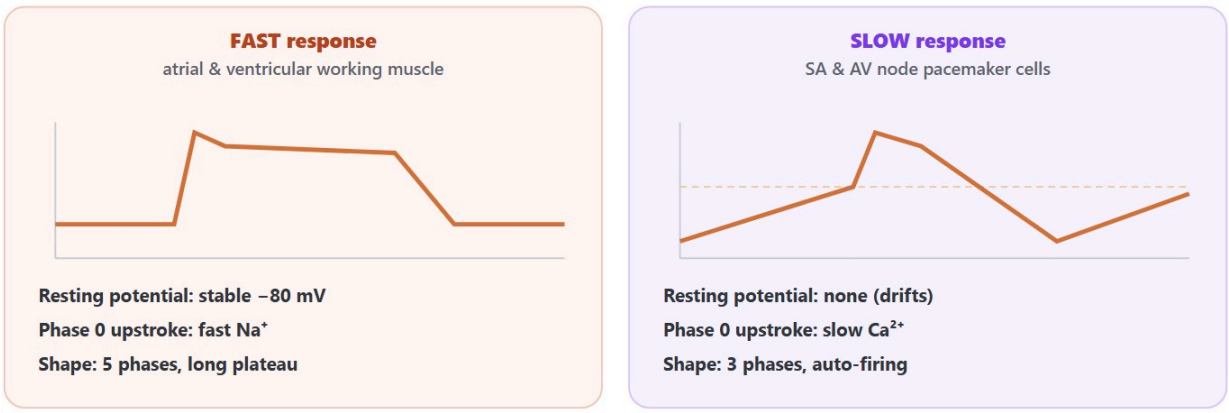


Fig 3 — Fast-response vs slow-response action potentials. The working cell (left) rests at a stable −80 mV and fires a fast Na^+ upstroke with a plateau; the pacemaker cell (right) has no resting potential, drifts to threshold, and fires a slow Ca^{2+} upstroke.

The ion currents beneath the fast-response action potential are worth one more look, because they are exactly what antiarrhythmic drugs grab hold of. The fast Na^+ current is a brief inward spike at phase 0; the L-type Ca^{2+} current is a sustained inward current that holds up the plateau; and the K^+ current is the outward current that finally brings the cell back down.

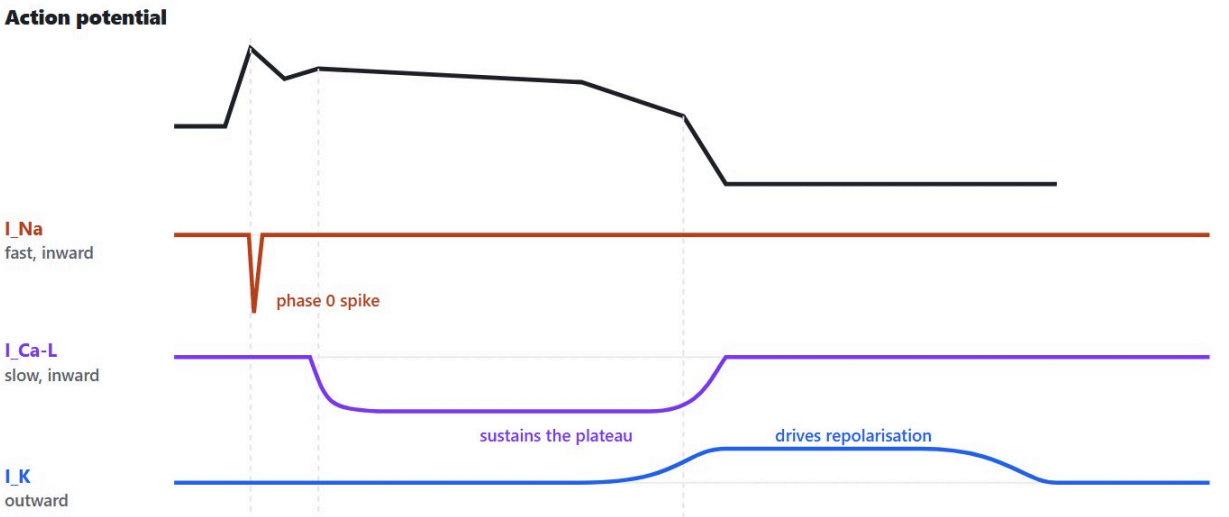


Fig 4 — The three currents that shape the fast-response action potential, aligned in time: the fast inward Na^+ current (phase-0 spike), the inward L-type Ca^{2+} current (sustains the plateau), and the outward K^+ current (drives repolarisation).

07 The conduction system: SA → AV → His → Purkinje

An impulse that started anywhere and spread only by slow cell-to-cell conduction would make the heart writhe rather than pump. So the heart owns a dedicated **conduction system** — itself made of specialised cardiac cells, not nerves — whose job is to launch each beat from one place and deliver it to the ventricles fast, in the right order, and after a deliberate pause.

The beat begins at the **SA node**, a small cluster of pacemaker cells in the wall of the right atrium near the entry of the cranial vena cava. Its impulse spreads across both atria cell-to-cell, contracting them, and converges on the **AV node** at the base of the right atrium. Here the impulse is deliberately **slowed**: the AV nodal cells are small and sparsely coupled by gap junctions, so conduction crawls, imposing an **AV nodal delay** of roughly **0.1 s**. That pause is functional — it gives the atria time to finish contracting and top up ventricular filling before the

ventricles are allowed to fire. From the AV node the impulse enters the **bundle of His**, the only electrical bridge across the insulating anulus fibrosus, which divides into **left and right bundle branches** running down the septum and then fans out into the **Purkinje fibres**. Purkinje fibres are large-diameter and conduct extremely fast — roughly ten times quicker than ordinary muscle — so the ventricular walls are activated almost simultaneously and contract as a coordinated squeeze. Because the network delivers the impulse to the **apex first**, the ventricle empties from the bottom upward, toward the outflow valves at the base.

There is a neat comparative-anatomy point here. In **small mammals** (cats, rabbits) the Purkinje fibres end superficially, just under the endocardium, so the wave still has to spread inward through the wall; in **large mammals** (horses, cattle) the fibres are thicker, conduct even faster, and **penetrate deep into the ventricular muscle**, so activation is more nearly simultaneous throughout the wall. This is one reason the ECG is far less reliable for reading chamber size in large animals — their depolarisation takes a less consistent path — even though it remains excellent for diagnosing arrhythmias.

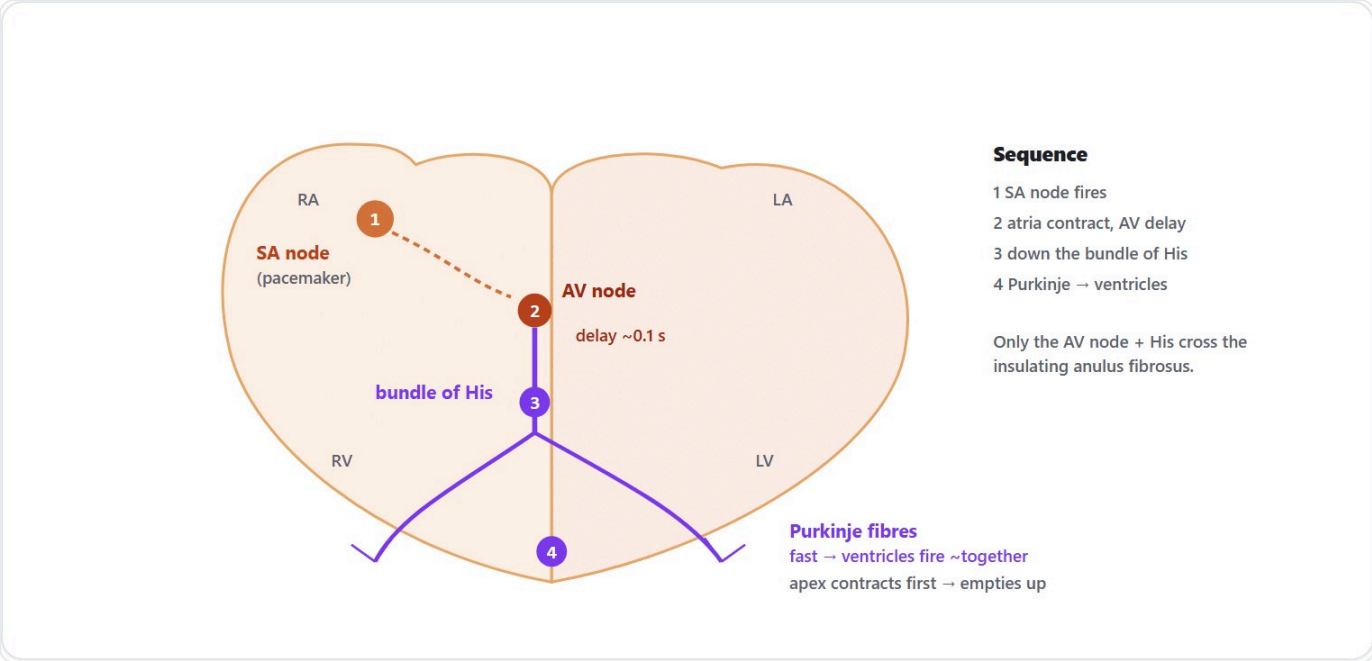


Fig 5 — The conduction system. The SA node fires (1), the atria depolarise and the impulse is delayed at the AV node (2), then races down the bundle of His (3) and the Purkinje network (4) so the ventricles contract almost together, apex first.

08 The pacemaker hierarchy and escape rhythms

Every autorhythmic cell in the conduction system can drift to threshold on its own — but they do not all drift at the same speed, and that difference organises the heart into a strict **pacemaker hierarchy**. The **SA node drifts fastest**, so it always reaches threshold first; its impulse then sweeps through the slower cells below and **resets them** before they can fire on their own. This is **overdrive suppression**: beat after beat, the SA node pre-empts every rival, which is why it is called the pacemaker even though it holds no monopoly on automaticity.

The backups matter when the leader fails. If the SA node stops, or if disease blocks conduction through the AV node, the next-fastest pacemaker **escapes** from suppression and takes over — usually the **AV junction**, which drives the ventricles at a slower intrinsic rate of about **30–40 bpm** in a resting dog, against the ~90 bpm of a normal sinus rhythm. If that too fails, the Purkinje/ventricular cells provide a final, slower safety net. The rule is simple and clinically useful: **the further down the hierarchy a pacemaker sits, the slower the heart it produces**. A dog that suddenly drops to a fixed 40 bpm has very likely lost its SA drive or its AV conduction and is running on an escape rhythm — a finding that sends you straight to the ECG.

09 Autonomic control of rate and force

The SA node sets an *intrinsic* rate — around **90–120 bpm** in a medium dog with no nervous input — but the animal rarely runs at it, because the autonomic nervous system continuously reshapes the pacemaker potential. Both branches work by changing the **slope of phase 4**: steepen it and threshold arrives sooner (a faster rate); flatten it and threshold arrives later (a slower rate).

The **sympathetic** system — noradrenaline from cardiac nerves and adrenaline from the adrenal medulla — acts on **β_1-adrenergic receptors** to **steepen phase 4**, so the SA node reaches threshold faster and the rate climbs (**positive chronotropy**). It also speeds AV conduction, shortening the delay. The **parasympathetic** system — the vagus nerve, releasing acetylcholine onto **muscarinic M_2 receptors** — does the

reverse, **flattening and lowering** the pacemaker drift so the rate falls, and lengthening the AV delay. In a calm, resting animal it is **vagal tone that dominates**, holding the heart *below* its intrinsic rate; much of the everyday rise in rate on excitement or exercise is simply the vagus letting go. (The same push-and-pull produces **respiratory sinus arrhythmia** — the normal, vagally-driven speeding of the heart on inspiration, especially obvious in young dogs.)

Sympathetic stimulation also makes each beat **stronger**, and the mechanism is elegant. β_1 activation works through cyclic AMP to **phosphorylate the L-type Ca^{2+} channels**, holding them open longer so more calcium enters during the plateau; more trigger calcium means more calcium-induced release and a more forceful contraction (**positive inotropy**). The same cyclic-AMP pathway also phosphorylates the SR pump so calcium is cleared faster afterwards — so sympathetic drive makes contractions not only stronger but **quicker and shorter**, which is exactly what a fast-beating heart needs. The parasympathetic system, by contrast, has little direct effect on ventricular force: its fibres are concentrated at the SA node, AV node and atria, so the vagus is a rate-and-conduction control far more than a force control. Different species sit at different set-points of this system — a resting horse cruises near **30–44 bpm** under heavy vagal tone, a cow near 60–70, a dog 70–120, a hen in the hundreds — the general rule being that smaller, more metabolically demanding animals run faster.

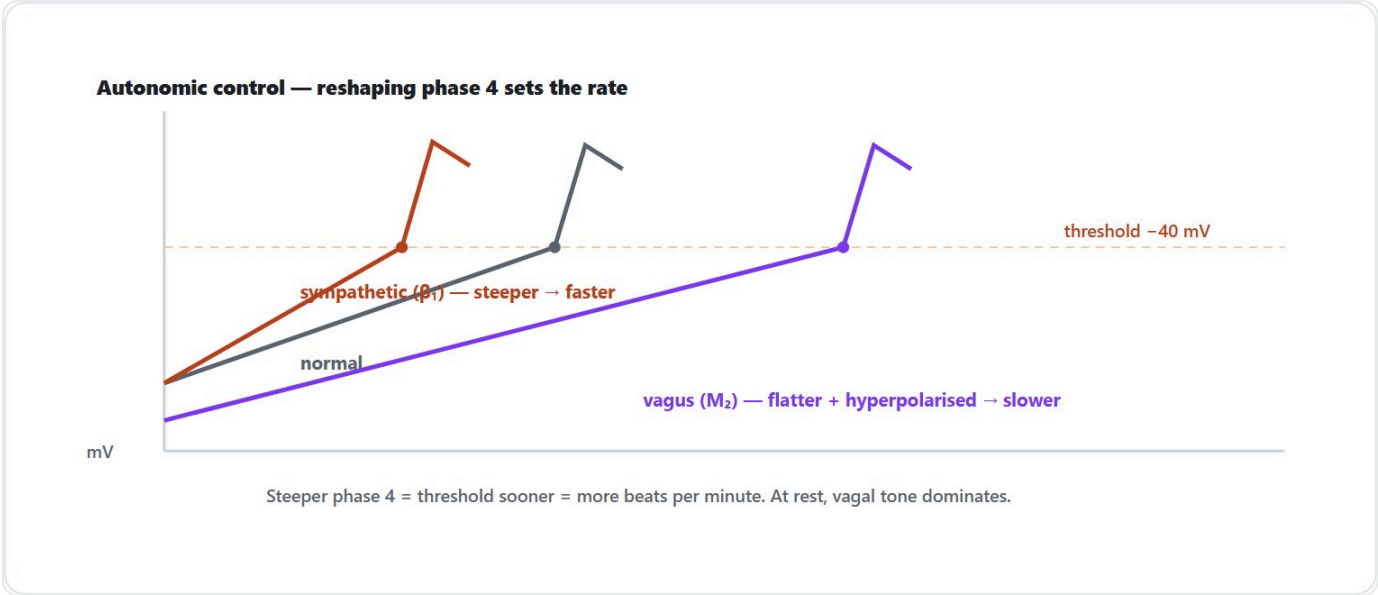


Fig 6 — Autonomic control reshapes the pacemaker potential. Sympathetic β_1 stimulation steepens phase 4 so threshold is reached sooner (faster rate); vagal M_2 stimulation flattens and lowers it (slower rate). At rest, vagal tone dominates.

10 Potassium, and the drugs that move it

Everything above is built on ion gradients, and one ion dominates: the resting membrane potential of cardiac muscle is essentially a potassium equilibrium potential. Change plasma potassium and you change the resting potential itself — which is why **hyperkalaemia (hyperkalemia)** is a cardiac emergency rather than a laboratory curiosity.

As extracellular K^+ rises, the resting potential drifts *less* negative. At first this brings the cell closer to threshold and repolarisation runs faster, which on the ECG is the classic **tall, narrow, peaked T wave**. Push it further and the sustained depolarisation begins to **inactivate the fast Na^+ channels** that drive phase 0: conduction slows, the **QRS widens**, and the P wave flattens and then disappears as the atria become inexcitable. Higher still and the QRS merges with the T wave into a **sine wave**, and the heart arrests. In practice this is the cat with a blocked urethra or the dog with hypoadrenocorticism, and it is the reason intravenous **calcium gluconate** is given first — it does not lower potassium at all, it restores the gap between resting potential and threshold and buys time while insulin, dextrose or fluids move the potassium.

Hypokalaemia does the opposite and is more insidious: repolarisation is prolonged, the cell becomes more excitable between beats, and ectopic activity and re-entrant arrhythmias become likelier — which is also why it potentiates digoxin toxicity.

Digoxin is the clearest example of a drug that works purely through this physiology. It inhibits the **Na^+/K^+-ATPase**, so intracellular Na^+ rises, the Na^+/Ca^{2+} exchanger has less of a gradient to work against, and more Ca^{2+} stays inside the cell — giving a stronger contraction. Its second action is vagal: it slows conduction through the AV node, which is why it is used to control the ventricular rate in atrial fibrillation. Both effects sit on a narrow therapeutic index, and because digoxin competes with potassium for the same pump, **a low plasma potassium leaves more pump free for digoxin to block** — the mechanism behind the classic warning that hypokalaemia precipitates digoxin toxicity.

11 When it goes wrong: arrhythmias and how drugs work

Every arrhythmia is, at bottom, a fault in one of two things: **abnormal impulse formation** or **abnormal impulse conduction** — and both are read directly off the action potentials described above. A pacemaker that fires too slowly or fails gives a **bradyarrhythmia** or an escape rhythm; a patch of muscle that becomes irritable enough to reach threshold on its own becomes an **ectopic pacemaker**, firing premature beats or a tachycardia. When conduction fails at the AV node the result is **AV block** — graded from a merely prolonged delay (first-degree), through intermittently dropped beats (second-degree), to complete dissociation of atria and ventricles (third-degree), where the ventricles limp along on a slow escape rhythm. And where a region conducts slowly in only one direction around a scar, an impulse can circle back and re-excite tissue that has just recovered — a self-sustaining **re-entry** loop that underlies many tachycardias and fibrillation.

Species colour these patterns. A resting horse often shows a **physiological second-degree AV block** — a dropped beat or two — simply because its vagal tone is so strong and its AV node so slow to recover; it vanishes the moment the horse moves and vagal tone falls. **Atrial fibrillation** is common in horses and in large-breed dogs such as Dobermans, and is classically converted with **quinidine**.

That last point opens the whole pharmacology of the heart. Because the action potential *is* a sequence of ion currents, the antiarrhythmic drugs are best understood as **tools that grab specific channels**: the sodium-channel blockers (quinidine, lidocaine) blunt the phase-0 upstroke and quiet ectopic foci and re-entry; the calcium-channel blockers (verapamil, diltiazem) act on the plateau and, powerfully, on the SA and AV nodes; the β-blockers (propranolol, atenolol) cut sympathetic drive and slow the rate and AV conduction; and the potassium-channel blockers lengthen the action potential and refractory period. Even the ECG is nothing more than the summed action potentials of the whole organ, timed out across the body surface — its **P wave** the atrial depolarisation, its **QRS** the ventricular depolarisation, its **T wave** the ventricular repolarisation. Learn the two cardiac action potentials and you have, in effect, learned to read the rhythm.

> **Red flag**
>
> A sudden fixed bradycardia (e.g. a dog at ~40 bpm), collapse or syncope, or a chaotic irregularly-irregular pulse can signal complete AV block, sick sinus syndrome or atrial fibrillation — get an ECG, and refer; some need a pacemaker or urgent rate control.

12 Quiz: test yourself

Nineteen questions across the syncytium, the fast and slow responses, the plateau, the conduction system, the pacemaker hierarchy and potassium. Answers are beneath each question — cover them and work down.

1. A heart is taken out, every nerve cut, and placed in a warm dish. What happens, and what does it prove?**Answer:** It **keeps beating**. The heart's rhythm is **intrinsic** — nerves modulate it, they do not create it.2. What are intercalated discs packed with, and why does it matter? **Answer: Gap junctions** — protein channels bridging neighbouring cells so ions flow directly from one to the next. That is what makes the heart an **electrical syncytium**.3. What proportion of cardiac cells are autorhythmic?**Answer:** About **1%**. The other **99%** are ordinary contractile cells sitting at a stable resting potential until stimulated.4. What is the resting potential of a working ventricular myocyte, and what drives phase 0?**Answer:** About **−80 mV**, held there by a membrane far more permeable to K^+ than Na^+. Phase 0 is the **fast voltage-gated Na^+ channels** flying open.5. How long is the cardiac action potential, and why does that matter?**Answer:** About **200 ms** — and the **absolute refractory period lasts as long**, because the Na^+ channels stay inactivated throughout.6. Why can the heart not tetanise, and why is that essential?**Answer:** The refractory period lasts almost as long as the action potential, so a second contraction cannot be stacked on the first. A tetanised heart could not **relax and refill**, so it could not pump.7. What carries the plateau, and what else does that current do? **Answer:** The **L-type Ca^{2+} current**. The calcium that floods in is not just electrical — it is the **trigger for contraction**.8. What is the pacemaker potential, and between which voltages does it run?**Answer:** After an action potential ends at about **−65 mV**, the membrane **drifts spontaneously upward** (phase 4) until it reaches threshold at about **−40 mV** and fires again.9. What starts the drift?**Answer:** The **K^+ channels that repolarised the cell gradually close**, so the outward brake fades and inward currents carry the membrane up.10. What drives phase 0 in a pacemaker cell, and how does that differ from working muscle?**Answer: Slow L-type Ca^{2+} channels**, not fast Na^+ channels — which is why the pacemaker upstroke is **slow** and there is **no plateau**.11. Where does the AV nodal delay come from, and how long is it?**Answer:** AV nodal cells are **small and sparsely coupled by gap junctions**, so conduction crawls — a delay of roughly **0.1 s**, which lets the atria finish emptying before the ventricles contract.12. What is the pacemaker hierarchy, and what is an escape rhythm?**Answer:** Every autorhythmic cell can drift to threshold, but at different speeds, so the fastest suppresses the rest. If the SA node fails or AV conduction blocks, the next-fastest **escapes** — usually the AV junction, at about **30–40 bpm**.13. A dog suddenly drops to a fixed 40 bpm. What does that suggest?**Answer:** Loss of SA drive or AV conduction, running on an **escape rhythm**. It sends you straight to the **ECG**.14. What is the intrinsic SA rate of a medium dog with no nervous input?**Answer:** About **90–120 bpm**. The animal rarely runs at it, because the autonomic system continuously reshapes the pacemaker potential.15. By what single mechanism do both autonomic branches change heart rate?**Answer:** By changing the **slope of phase 4**. Steepen it and threshold arrives sooner (faster); flatten it and threshold arrives later (slower).16. Which receptor does sympathetic drive act on at the SA node, and what is the effect called?**Answer: β_1-adrenergic** receptors, steepening phase 4 — **positive chronotropy**.17. What does rising plasma potassium do to the ECG, in order?**Answer:** First a **tall, peaked T wave** as repolarisation speeds up. Then, as sustained depolarisation inactivates the fast Na^+ channels, conduction slows, the **QRS widens**, and the **P wave flattens and disappears** as the atria become inexcitable.18. Why does hypokalaemia precipitate digoxin toxicity?**Answer:** Digoxin **competes with potassium for the same pump**. A low plasma potassium leaves **more pump free for digoxin to block**, so the same dose bites harder.19. A dog has a fixed 40

bpm rate, syncope, or a chaotic irregularly-irregular pulse. What should you do?**Answer:** Get an **ECG** and refer. This can be **complete AV block**, sick sinus syndrome or atrial fibrillation — some need a pacemaker or urgent rate control.

13 Cardiac action potential — frequently asked questions

What is the difference between a fast-response and a slow-response cardiac action potential?

Fast-response action potentials belong to the working muscle of the atria and ventricles: they start from a stable −80 mV resting potential and have a fast sodium upstroke and a long calcium plateau. Slow-response action potentials belong to the pacemaker cells of the SA and AV nodes: they have no resting potential, drift spontaneously to threshold, and have a slow calcium-driven upstroke.

Why is the cardiac action potential so long, and why can't the heart tetanise?

The L-type calcium plateau holds the cell depolarised for about 200–300 ms — roughly a hundred times longer than a nerve impulse — and the refractory period lasts almost as long. A new beat cannot begin until the last has relaxed, so cardiac contractions cannot summate into a tetanus; the heart is guaranteed to relax and refill between beats.

What makes the heart beat on its own?

Pacemaker cells in the SA node have no resting potential; they drift spontaneously to threshold (the pacemaker potential, driven by the funny current and calcium) and fire, over and over. Because the SA node drifts fastest, it sets the pace and resets slower would-be pacemakers each beat.

Why is hyperkalaemia dangerous to the heart?

Because the resting potential of cardiac muscle is set by potassium. A rising plasma K^+ makes the resting potential less negative, which first speeds repolarisation — the tall, peaked T wave — and then inactivates the fast sodium channels that generate phase 0, so conduction slows, the QRS widens, P waves vanish and the trace degenerates toward a sine wave and arrest. Intravenous calcium is given first because it restores the gap between resting potential and threshold immediately, without changing the potassium at all.

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When to refer

- A sudden, fixed bradycardia (e.g. a dog at ~30–40 bpm) unresponsive to excitement — suspect **complete AV block** or **sick sinus syndrome**; ECG and refer (may need a pacemaker).
- An **irregularly-irregular** pulse with a pulse deficit — suspect **atrial fibrillation** (common in horses and large-breed dogs); ECG and refer for rate control or conversion.
- Collapse, syncope or exercise intolerance with a rhythm disturbance — a cardiac emergency; stabilise and refer.
- Frequent premature beats or a sustained tachycardia — identify the focus (supraventricular vs ventricular) on ECG before treating; ventricular tachycardia can be life-threatening.
- A resting horse with occasional 'dropped' beats that vanish on exercise is usually **physiological second-degree AV block** (high vagal tone) — confirm it disappears with a rise in rate.

Sources

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2. Klein BG. Cunningham's Textbook of Veterinary Physiology. 6th ed. Elsevier; 2020. Chapter 20 — The Electrocardiogram (pp 203–214): the P–QRS–T relationship to atrial/ventricular depolarisation and repolarisation, the PR and QT intervals, and large-animal ECG variability.
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4. Ware WA. Cardiovascular Disease in Small Animal Medicine. London: Manson/CRC Press — standard clinical reference for the arrhythmias (AV block, sick sinus syndrome, atrial fibrillation, ventricular tachycardia), escape rhythms, and antiarrhythmic drug use referenced here.

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