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Cardiac Action Potential

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.

INSIDE THESE NOTES

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Vets & veterinary students

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2026-07-02

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Cardiac Action Potential

STUDY NOTES · BASIC SCIENCES · PHYSIOLOGY · UPDATED 2026-07-02

CONTENTS

LEARNING OBJECTIVES

After working through these notes you will be able to:

- ✓ Explain why the heart behaves as an electrical syncytium and beats without nervous input.
- ✓ Draw and label the five phases of the fast-response (working-myocyte) action potential and the ion current behind each.
- ✓ Explain the calcium plateau, the long refractory period, and why cardiac muscle cannot tetanise.
- ✓ Draw the slow-response (pacemaker) action potential and explain the phase-4 pacemaker potential and the funny current.
- ✓ Trace the conduction pathway SA→AV→His→Purkinje, explain the AV nodal delay and the pacemaker hierarchy.
- ✓ Explain how the sympathetic and parasympathetic systems change heart rate and force, and how antiarrhythmic drugs act on the AP.

TL;DR

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.

AT A GLANCE

WHAT IT IS	The electrical impulse that triggers each heartbeat — recorded, summed, as the ECG
TWO FLAVOURS	Fast-response (atria/ventricles) · slow-response (SA & AV nodes)
FAST-RESPONSE AP	5 phases (0–4); phase-0 Na^+ upstroke, a long Ca^{2+} plateau, K^+ repolarisation, stable −80 mV rest
THE PLATEAU	L-type Ca^{2+} influx holds the cell depolarised ~200 ms → Ca-induced Ca release drives contraction
REFRACTORY PERIOD	Almost as long as the contraction → no summation, **no tetany**; the heart must refill
PACEMAKER AP	No resting potential; phase-4 drift (funny current I_f + Ca^{2+}) to a ~−40 mV threshold; phase-0 is Ca^{2+}-driven
CONDUCTION PATH	SA node → atria → AV node (delay) → bundle of His → Purkinje → ventricles
AUTONOMIC CONTROL	Sympathetic (β_1) steepens phase 4 → faster + stronger; vagus (M_2) flattens it → slower
WHY IT MATTERS	Rate, rhythm, the ECG and every antiarrhythmic drug are read off these two waveforms

01 No action potential, no heartbeat

- **Every heartbeat begins as a cardiac action potential.** The heart is a **functional syncytium** — gap junctions at the intercalated discs let the impulse spread cell-to-cell, so a chamber fires as one.
- **Two flavours:** a **fast-response** AP (working atrial/ventricular muscle) and a **slow-response** AP (SA & AV node pacemakers).
- Atria and ventricles are separated by the **insulating anulus fibrosus**; only the conduction system crosses it.

02 The working-muscle AP (fast response) — 5 phases

- Rest ~**−80 mV**. **0** fast **Na^+** upstroke · **1** brief K^+ dip · **2** L-type **Ca^{2+} PLATEAU** (~200 ms) · **3 K^+** repolarisation · **4** rest.
- **~200–300 ms long — about 100× a nerve impulse**, entirely because of the plateau. Atrial APs are shorter than ventricular.

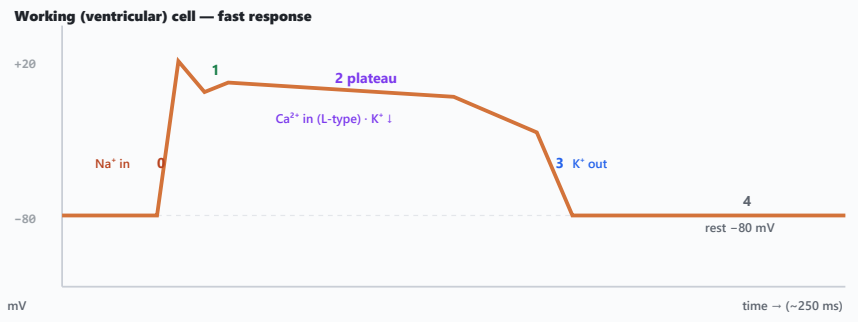

Fig 1 — The fast-response AP: Na^+ upstroke (0), notch (1), Ca^{2+} plateau (2), K^+ repolarisation (3), rest (4).

03 The plateau, refractory period & no tetany

- The **Ca^{2+} plateau** = a long AP = a long **refractory period** (~200 ms) → beats **cannot summate** → **NO TETANY** → the heart always relaxes and refills. The safety catch.
- The plateau Ca^{2+} triggers **calcium-induced calcium release** from the SR (cytosolic Ca^{2+} ~1000×) → contraction. Unlike skeletal muscle, the heart needs **trigger Ca^{2+} from outside**.

04 The pacemaker AP (slow response)

- **SA node: NO resting potential.** The phase-4 **pacemaker potential** drifts from ~**−65 mV** up to a ~**−40 mV** threshold — driven by decaying K^+, the **funny current I_f** (Na^+) and a late Ca^{2+} current.
- The phase-0 upstroke is **Ca^{2+}-driven (slow), no fast Na^+** → a 'slow response'. Essentially just phases 4, 0, 3.

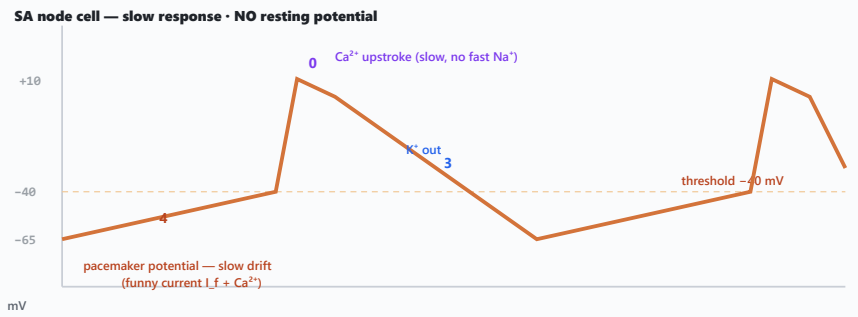

Fig 2 — The pacemaker AP: a phase-4 drift (funny current + Ca^{2+}) to a −40 mV threshold, then a slow Ca^{2+} upstroke.

05 Fast vs slow — side by side

FEATURE	FAST RESPONSE (MUSCLE)	SLOW RESPONSE (PACEMAKER)
Where	Atrial & ventricular myocytes	SA & AV node
Resting potential	Stable ~−80 mV	None — drifts
Phase-0 upstroke	Fast Na^+	Slow Ca^{2+}
Plateau	Yes (Ca^{2+})	No
Automaticity	No (stimulated)	Yes (self-firing)

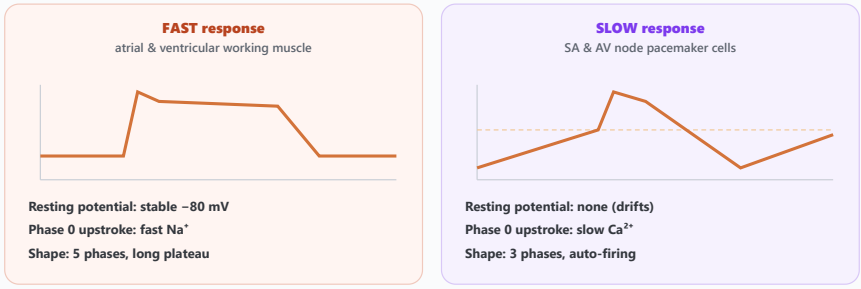

Fig 3 — Fast vs slow: a stable baseline + Na^+ upstroke vs a drifting baseline + Ca^{2+} upstroke.

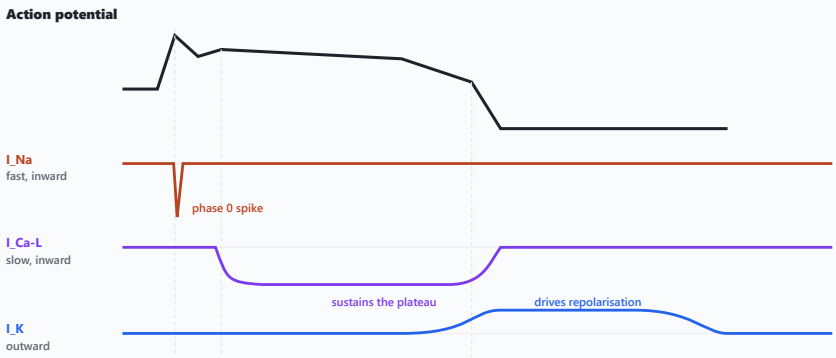

Fig 4 — The currents under the AP: Na^+ spike (phase 0), Ca^{2+} (plateau), K^+ (repolarisation).

06 Conduction system: SA → AV → His → Purkinje

- **SA node** (right atrium) → atria → **AV node** (**delay ~0.1 s** so the atria fill the ventricles) → **bundle of His** (the only path across the anulus fibrosus) → L/R branches → **Purkinje** (fast, ~10× muscle) → ventricles fire ~together, **apex first**.
- **Species:** in small mammals Purkinje fibres end superficially; in large mammals (horse, cattle) they are thicker, faster and **penetrate deep** into the wall.

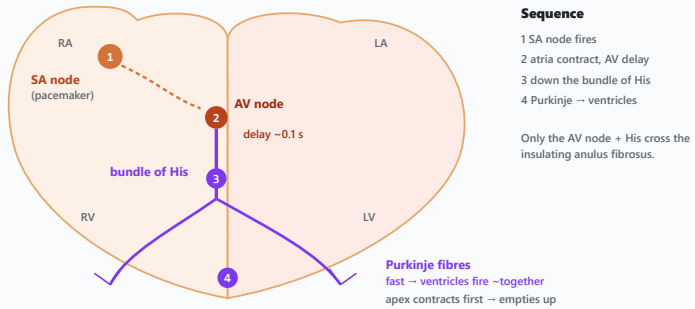

Fig 5 — The conduction pathway: SA (1) → atria + AV delay (2) → bundle of His (3) → Purkinje (4).

07 Pacemaker hierarchy & escape rhythms

- The **SA node drifts fastest**, so it sets the pace and **resets** slower pacemakers each beat (**overdrive suppression**). Normal sinus ~90 bpm in a dog.
- If it fails, an **escape** pacemaker takes over: **AV junction ~30–40 bpm**, then Purkinje slower still. **The further down, the slower the rate.**

08 Autonomic control of rate & force

- Intrinsic SA rate ~**90–120 bpm** (dog). **Sympathetic (β_1)** steepens phase 4 → **faster** (chronotropy) + speeds AV conduction; via cAMP it opens more L-type Ca^{2+} channels → **stronger + shorter** contraction (inotropy).
- **Vagus (M_2)** flattens phase 4 → **slower**. At rest, **vagal tone dominates** (hence respiratory sinus arrhythmia). Species HR: horse ~30–44, cow 60–70, dog 70–120, hen 200–400.

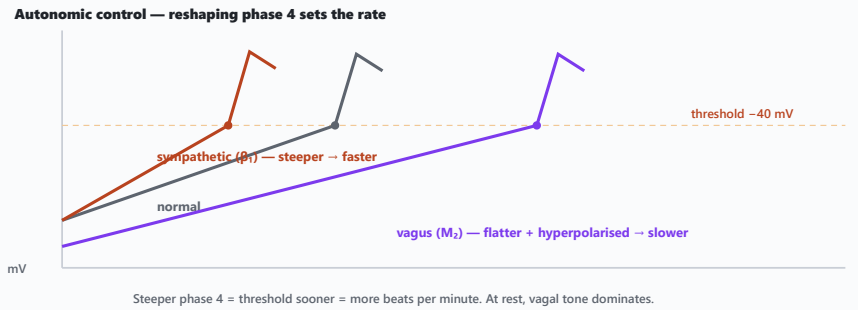

Fig 6 — Sympathetic β_1 steepens phase 4 (faster); vagal M_2 flattens it (slower). At rest the vagus wins.

09 Arrhythmias & how drugs work

- Every arrhythmia = **abnormal impulse formation** (brady / ectopic / tachy) or **abnormal conduction** (AV block 1°/2°/3°, re-entry) — read straight off the two APs.
- **Species:** a resting horse often shows **physiological 2nd-degree AV block** (high vagal tone), gone on exercise; **atrial fibrillation** is common in horses & large-breed dogs (Dobermans) — converted with **quinidine**.
- **Antiarrhythmics grab the AP's channels:** Na^+ blockers (quinidine, lidocaine), Ca^{2+} blockers (verapamil, diltiazem — the nodes), β-blockers (propranolol), K^+ blockers. The **ECG** = summed APs: **P** = atrial depol, **QRS** = ventricular depol, **T** = ventricular repol.

RED FLAG

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

> *Learn the two cardiac action potentials — fast-response and slow-response — and you have, in effect, learned to read rate, rhythm and the ECG.*
>
> — after Cunningham 6e Ch 19–20 & PDA 3e Ch 11.

KEY TERMS — QUICK GLOSSARY

Cardiac action potential	The electrical impulse that triggers a heartbeat; long (~200–300 ms) because of a Ca^{2+} plateau.
Functional syncytium	Myocytes joined by gap junctions at the intercalated discs, so current spreads cell-to-cell and a chamber depolarises as a unit.

Fast-response AP	The working-myocyte (atrial/ventricular) action potential — fast Na^+ upstroke, 5 phases, stable resting potential.
Slow-response AP	The pacemaker (SA/AV node) action potential — Ca^{2+}-driven slow upstroke, no stable resting potential.
Plateau (phase 2)	The sustained depolarisation from L-type Ca^{2+} influx that makes the cardiac AP so long.
Refractory period	The ~200 ms during which no new AP can start — almost as long as the contraction, so no tetany.
Calcium-induced calcium release	Ca^{2+} entering during the AP triggers a much larger Ca^{2+} release from the sarcoplasmic reticulum, driving contraction.
Pacemaker potential	The spontaneous phase-4 drift of a pacemaker cell up to threshold; the reason the heart is automatic.
Funny current (I_f)	The inward Na^+ current through pacemaker ('funny') channels that starts the phase-4 drift.
SA node	The sinoatrial node in the right atrium — drifts to threshold fastest, so it is the normal pacemaker.
AV nodal delay	The slow conduction through the AV node (~0.1 s) that lets the atria fill the ventricles before they contract.
Overdrive suppression	The faster SA node resets slower would-be pacemakers each beat, keeping itself in charge.
Escape rhythm	A slower backup pacemaker (AV node ~30–40 bpm in a dog) taking over when the SA node or AV conduction fails.
Positive chronotropy / inotropy	A faster rate / a stronger contraction — both produced by sympathetic β_1 stimulation.

QUICK REVISION — REMEMBER THESE

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 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.

MEMORY AIDS

0-1-2-3-4 — Fast-response phases: **0** Na^{+} in (up), **1** brief dip, **2** Ca^{2+} plateau, **3** K^{+} out (down), **4** rest. 'Na up, Ca holds, K down.'

Plateau = no tetany — The Ca^{2+} **plateau** = a long AP = a long **refractory period** = the heart **can't tetanise** = it always relaxes and refills. The safety catch.

Funny phase 4 — Pacemakers have **no rest** — the **funny current** drifts phase 4 **up** to threshold. Fastest drifter (SA node) wins.

SA beats AV beats Purkinje — Pacemaker hierarchy & rate: **SA** (~90) > **AV** (~40) > **Purkinje** (~20–40). Further down = slower escape.

Sympathetic Steepens — **S**ympathetic (β_1) **S**teepens phase 4 → faster + stronger. Vagus (M_2) flattens it → slower. Rest = vagus wins.

TEST YOURSELF — ACTIVE RECALL

Cover the answers and try to retrieve each one from memory first — self-testing beats re-reading.

1. **Why does a whole cardiac chamber contract as a unit, and what makes the heart beat without nerves?**
2. **List the five phases of the fast-response action potential and the main ion movement in each.**
3. **Why is the cardiac action potential so long, and why can't cardiac muscle tetanise?**
4. **How does the calcium plateau lead to contraction?**
5. **What is the pacemaker potential, and which currents drive it?**
6. **Trace the conduction pathway and explain why the AV nodal delay matters.**
7. **What happens if the SA node stops, and why is the resulting rate slow?**
8. **How do the sympathetic and parasympathetic systems change the heartbeat, and how do antiarrhythmic drugs work?**

ANSWERS

1. Myocytes are joined by gap junctions at the intercalated discs, so the impulse spreads cell-to-cell (a functional syncytium); autorhythmic pacemaker cells depolarise spontaneously, giving the heart its own automatic rhythm.
2. Phase 0 = fast Na^+ influx (upstroke); phase 1 = brief K^+ efflux (Na^+ channels inactivate); phase 2 = plateau, L-type Ca^{2+} influx with reduced K^+ efflux; phase 3 = K^+ efflux (repolarisation); phase 4 = stable ~−80 mV resting potential.
3. The L-type Ca^{2+} plateau holds the cell depolarised ~200 ms (~100× a nerve), and the refractory period lasts almost as long as the contraction — so a new beat cannot start until the last has relaxed. No summation means no tetany, guaranteeing the heart relaxes and refills.
4. Ca^{2+} entering through the L-type channels during the plateau triggers calcium-induced calcium release from the sarcoplasmic reticulum; cytosolic Ca^{2+} rises ~1000-fold, driving the crossbridge cycle.
5. The spontaneous phase-4 drift of a pacemaker cell from ~−65 mV up to a ~−40 mV threshold. It is driven by the inward funny current (I_f, Na^+), a decaying K^+ current, and a late L-type Ca^{2+} current; the phase-0 upstroke is then Ca^{2+}-driven, not Na^+.
6. SA node → atria → AV node → bundle of His → left/right bundle branches → Purkinje fibres → ventricles. The AV node conducts slowly (~0.1 s delay) so the atria finish contracting and top up ventricular filling before the ventricles fire.
7. A lower escape pacemaker takes over — usually the AV junction (~30–40 bpm in a resting dog), or the Purkinje system if that fails. The further from the SA node, the slower the intrinsic rate.
8. Sympathetic nerves and adrenal adrenaline act on β_1 receptors to steepen phase 4 (faster rate) and raise Ca^{2+} entry (stronger contraction); the vagus (ACh, M_2) flattens phase 4 for a slower rate. Antiarrhythmic drugs block the Na^+, Ca^{2+}, K^+ or β targets that shape the AP.

WHEN TO REFER OR ESCALATE

- 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.

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