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The Action Potential Explained

How Nerve Cells Fire an Electrical Impulse

Nearly everything a nervous system does runs on the action potential — the brief, self-regenerating electrical impulse a nerve or muscle cell fires to carry a signal without it fading.

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IN SHORT

Nearly everything a nervous system does runs on the **action potential** — the brief, self-regenerating electrical impulse a nerve or muscle cell fires to carry a signal without it fading. A cell rests at about **−70 mV**, inside negative; when a stimulus pushes it past **threshold**, voltage-gated **Na^+** channels snap open (depolarisation) and then **K^+** channels restore the charge (repolarisation) — the whole spike in 2–3 ms. Understand that one event and the clinic follows: why **local anaesthetics (anesthetics)** silence nerves, why **hyperkalaemia (hyperkalemia)** stops the heart, and why a calcium-starved cow goes down with milk fever.

KEY POINTS

1. An action potential is an **all-or-none** electrical impulse — once threshold is crossed it fires fully and spreads **without losing amplitude**; strength is coded by **frequency**, not size.
2. At rest the membrane sits near **−70 mV**, inside negative, because it is far more permeable to **K^+** (which leaks out) than to Na^+.
3. The **Na^+/K^+ pump** builds the gradients that make it all possible — 3 Na^+ out for 2 K^+ in — and it is one of the body's biggest energy costs.
4. Crossing **threshold** opens **voltage-gated Na^+ channels** (depolarisation); they inactivate and **voltage-gated K^+ channels** repolarise the cell.
5. The **refractory period** (Na^+ channels inactivated) forces a one-way signal and caps firing frequency.
6. **Myelin** and the **nodes of Ranvier** give **saltatory conduction** — the impulse leaps node to node, far faster than in bare axons.
7. Every step is a **drug and disease target**: local anaesthetics block Na^+ channels, and K^+/Ca^{2+} disturbances change how easily nerves fire.

01 What is an action potential?

Nearly everything a nervous system does — a paw pulling back from a thorn, a heart beating, a memory forming — is carried by the same electrical event: the **action potential**. It is a brief, explosive reversal of the voltage across a cell's membrane that, once triggered, **regenerates itself down the whole length of a nerve or muscle fibre (fiber) without fading**. That last point is its entire reason for existing. The gentle voltage changes a neuron collects at its dendrites die away within a millimetre or two; the action potential is the cell's way of carrying a signal a metre down a leg with the message still intact at the far end.

Two features define it. First, it is **all-or-none**: a stimulus either reaches the trigger point and fires a full-sized spike, or nothing happens — there is no half an action potential. Because every spike is identical, the nervous system cannot signal the *strength* of a stimulus by making a bigger impulse; it codes strength as **frequency**, firing more action potentials per second for a stronger input. Second, it is **self-propagating**: each patch of membrane that fires drags the next patch to threshold, so the impulse marches — or, on myelinated nerves, leaps — to the axon's end at full amplitude.

To see how a quiet cell becomes a firing one, start with the steady state it holds before any signal arrives — the resting membrane potential — and with the cell's own architecture.

02 The neuron: four regions, one job

A typical neuron is built from **four anatomical regions**, each with a distinct part in moving a signal from one end to the other. The **dendrites** — branch-like extensions whose surface area dwarfs the rest of the cell — are the main **receiving** apparatus, studded with receptors that read the chemical signals arriving from other neurons. The **cell body** (soma or perikaryon, about 20 µm across) is the metabolic factory: it holds the nucleus and the machinery that manufactures the cell's proteins.

The **axon** is the **conducting** unit — a single tubular process that can exceed a metre in large animals, carrying the action potential from its base to its far end. Because a mature axon has no ribosomes, everything it needs is built in the cell body and ferried down by **axoplasmic transport**. At its end the axon branches into **presynaptic terminals** (synaptic boutons) that **transmit** the signal across a synapse to the next cell.

One spot deserves special attention: the **initial segment** of the axon, where it leaves the cell body. This is the neuron's decision point — the **axon hillock** and initial segment, together the trigger zone, where all the incoming excitatory and inhibitory signals are added up, and where, if the sum is large enough, the action potential is born. Everything before it is gentle and graded; everything after it is all-or-none.

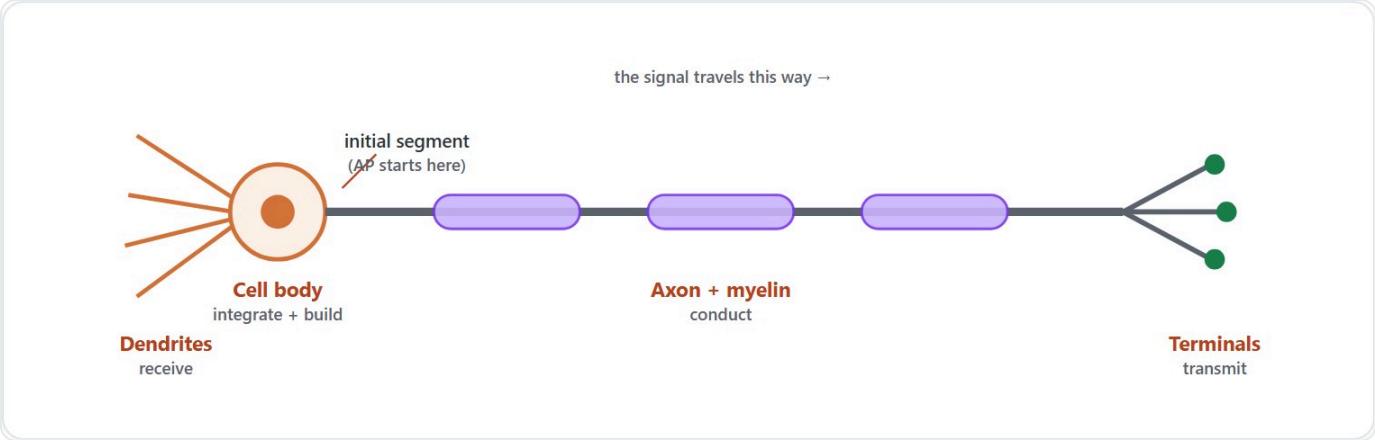


Fig 1 — A neuron's four regions. Signals are received on the dendrites, integrated at the initial segment, conducted along the axon, and passed on at the terminals.

03 The resting membrane potential: −70 mV, inside negative

Before it can fire, every cell holds a steady voltage across its membrane — the **resting membrane potential (RMP)**. Set the outside to zero, and the inside of a typical mammalian neuron sits at about **−70 mV** (the value ranges roughly −40 to −90 mV between cell types, and −70 to −80 mV in neurons). In plain terms, the inside of a resting neuron is **negative** relative to the fluid around it.

That negativity does not come from a large pool of spare charge sloshing about — the bulk of the cytoplasm and the extracellular fluid are each electrically neutral. The imbalance is razor-thin: a single scattered layer of extra **positive** charge clings to the outer face of the membrane and an equal layer of extra **negative** charge to the inner face, so the membrane behaves like a tiny charged capacitor. Across its sub-10-nanometre thickness even that modest voltage is a colossal electric field — on the order of 100,000 volts per centimetre.

The resting potential is the stored energy the cell will spend when it fires — and holding it steady is one piece of the cell's wider cellular homeostasis. Understanding the three things that build it explains not only how a nerve works, but why a disturbance of blood **potassium** can be lethal.

04 What sets the resting potential — the three determinants

1 — The Na^+/K^+ pump. This ATP-driven **sodium–potassium pump** (Na^+/K^+-ATPase) moves **3 Na^+ out of the cell for every 2 K^+ it brings in**, building the steep gradients everything else depends on: K^+ concentrated inside, Na^+ concentrated outside. Because it exports more positive charge than it imports, it is slightly *electrogenic* — but its real job is maintaining the gradients. It is also astonishingly expensive: one source calls outward Na^+ pumping "the single most energy-requiring process in the body," and the pump can consume **50–70% of the brain's energy**. That bill matters clinically, because the pump runs on ATP from glucose and oxygen and neurons store neither.

2 — A permeant ion moving toward equilibrium. Given those gradients, **K^+** tends to leak out down its concentration gradient, carrying positive charge with it and leaving unopposed negative charges (mostly trapped protein anions) behind. As the inside grows negative, that

negativity starts to pull K^+ back in. Where the outward chemical push and the inward electrical pull balance is the ion's **equilibrium potential**, given by the **Nernst equation** — about **−90 mV for K^+**.

3 — A selectively permeable membrane. At rest the membrane holds **far more K^+ leak channels than Na^+ leak channels**, so it is much more permeable to K^+. That is why the RMP settles close to the K^+ equilibrium potential (−90 mV) rather than the Na^+ equilibrium potential (about +70 mV); the small, steady inward Na^+ leak is all that holds it at −70 rather than −90. The **Goldman equation** formalises this by weighting each ion by its permeability.

ION	INSIDE (MM)	OUTSIDE (MM)	EQUILIBRIUM POTENTIAL
K^+	140	4	≈ −90 mV
Na^+	15	145	≈ +70 mV
Cl^-	7	120	passively distributed
Ca^{2+}	0.0001	1	strongly positive

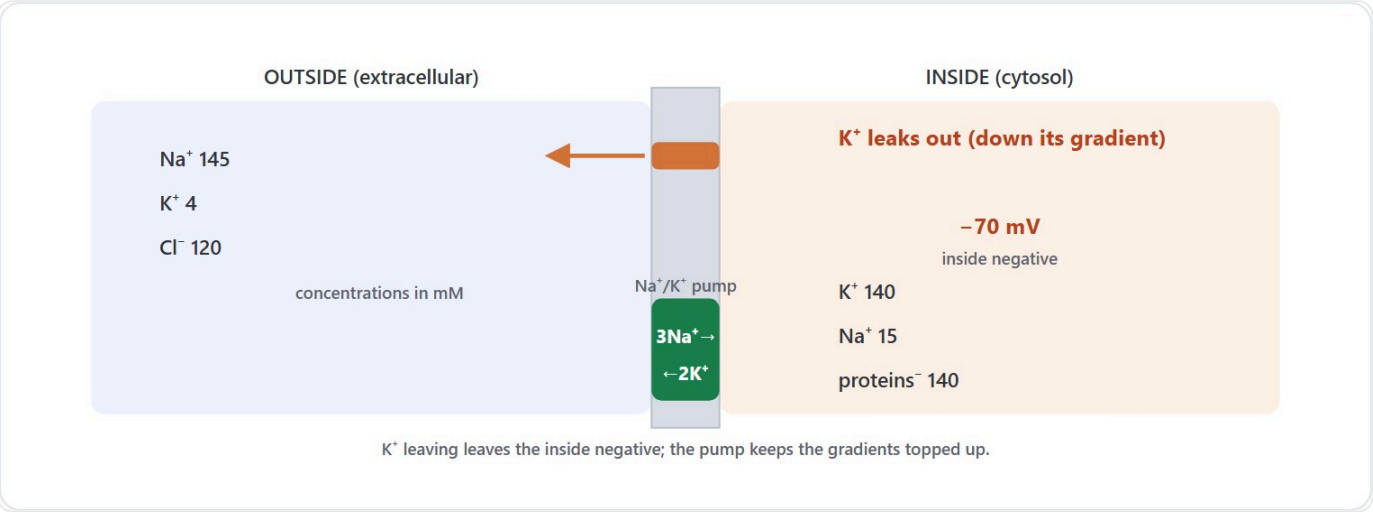


Fig 2 — The resting potential. The pump builds the gradients; K^+ leaking out down its gradient leaves the inside at about −70 mV.

05 Reaching threshold: EPSPs, IPSPs and summation

A resting neuron does not fire on its own; it must be pushed to **threshold** — a voltage roughly **10–20 mV more positive than rest** (around −55 mV). The push arrives from other neurons, through synapses onto the dendrites and cell body.

When a presynaptic terminal releases transmitter, it opens ligand-gated channels on the receiving cell and nudges its voltage one of two ways. Open Na^+ channels let Na^+ in and make the membrane **more positive** — an **excitatory postsynaptic potential (EPSP)**, say a shift from −75 to −65 mV, bringing the cell *closer* to firing. Open K^+ channels move the voltage **more negative** — an **inhibitory postsynaptic potential (IPSP)**, or hyperpolarisation, pushing it *away* from threshold.

These postsynaptic potentials are **graded and decremental**: each is small (often just 2–3 mV), lasts only a few milliseconds, and fades with distance from the synapse, like ripples spreading on a pond. No single EPSP fires the cell. Instead the initial segment continuously **sums** the hundreds of EPSPs and IPSPs arriving at any instant — a running tug-of-war — and only when excitation wins by enough to cross threshold does it launch an action potential. That **integration** is how a neuron "decides".

06 The action potential, step by step

Cross threshold at the initial segment and the response is explosive. The action potential runs through the **sequential opening of voltage-gated channels — Na^+ first, K^+ shortly after** — in a fixed sequence that takes only **2–3 ms** in most neurons.

Depolarisation (the upstroke). Threshold flings open **voltage-gated Na^+ channels**. Na^+ floods in, making the inside rapidly more positive — which opens still more Na^+ channels, a runaway **positive feedback** that drives the membrane from −70 mV up past zero toward the Na^+ equilibrium potential, peaking around **+30 mV** in well under a millisecond. For an instant the inside is positive.

Repolarisation (the downstroke). Two things end the spike: the Na^+ channels **inactivate** — they shut by themselves after about half a millisecond — and the slower **voltage-gated K^+ channels** now open. K^+ pours out, carrying positive charge away and driving the voltage back down toward the K^+ equilibrium potential.

After-hyperpolarisation. Those K^+ channels close sluggishly, so for a few milliseconds K^+ keeps leaving and the membrane dips **slightly more negative than rest** before settling back to −70 mV.

The lag in the Na^+ channels creates the **refractory period**. While they are inactivated the cell *cannot* fire again, however strong the stimulus (the absolute refractory period); as they recover, only a stronger-than-normal stimulus will fire it (the relative refractory period). This does two essential jobs: it forces the impulse to travel in **one direction only**, and it caps how many action potentials a fibre can fire each second.

For students Up = in, down = out. The upstroke is Na^+ coming **in**; the downstroke is K^+ going **out**. If you remember one thing, remember that.

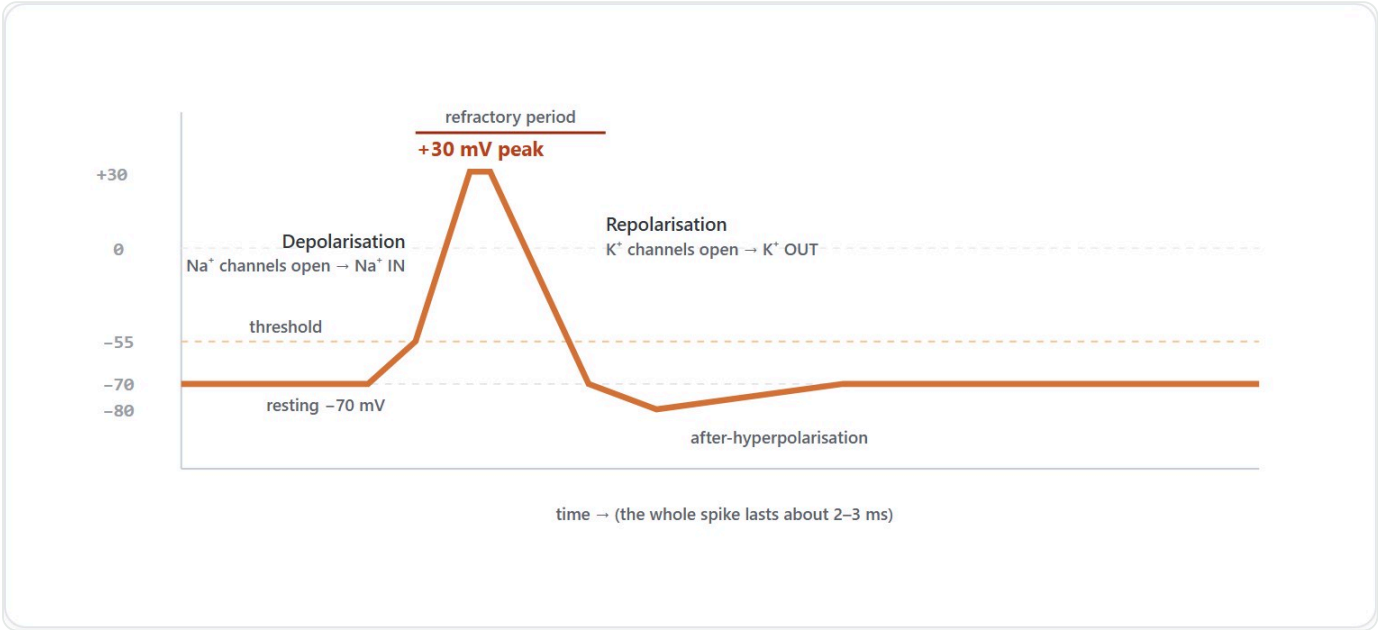


Fig 3 — One action potential: rest → threshold → Na^+-driven depolarisation → K^+-driven repolarisation → after-hyperpolarisation, in 2–3 ms.

07 Propagation: how the spike travels — and why myelin matters

An action potential at the initial segment is only useful if it reaches the axon terminals, and it gets there by **regenerating itself**. The Na^+ pouring in at the firing patch spreads as a local **electrotonic current** to the neighbouring resting patch, dragging it to threshold; that patch fires, triggers the next, and the impulse advances at full size to the very end. Because the patch just behind is refractory, it can only travel forward.

Speed depends on two things: **axon diameter** and **myelination**. A thin, bare axon conducts slowly — around **0.5 m/s**, and bare vertebrate axons rarely top 2 m/s. Wrapping the axon in **myelin** — the fatty insulation laid down by **Schwann cells** in peripheral nerves and **oligodendrocytes** in the brain and cord — transforms it. Myelin insulates long internodes and leaves bare gaps, the **nodes of Ranvier**, packed with voltage-gated Na^+ channels. The impulse regenerates only at the nodes and effectively **jumps from node to node** — **saltatory conduction** — reaching up to about **100 m/s in domestic mammals (≈120 m/s in the cat, versus ~70 m/s in humans)**. A bare axon would need roughly a hundred times the diameter to match that, so myelin lets the body pack many fast fibres into a slim nerve.

It also explains why **demyelinating disease** is so disabling: strip the myelin and conduction slows or fails — producing weakness and incoordination even though the axon itself survives.

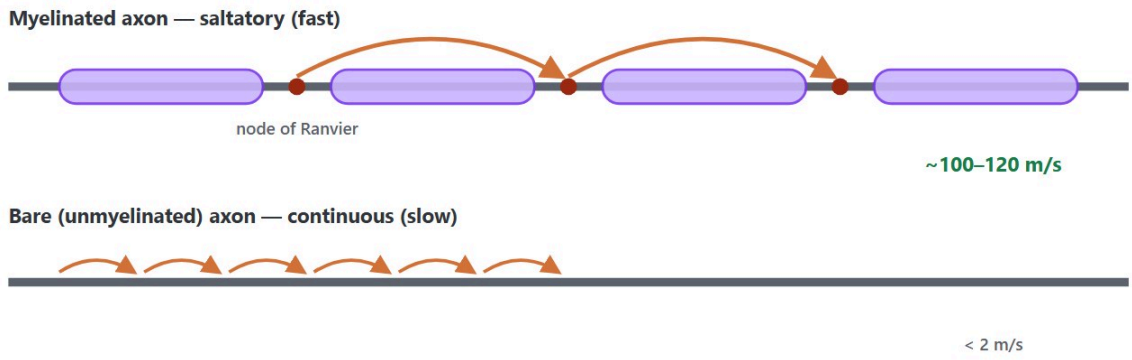


Fig 4 — Myelin turns slow, continuous conduction into fast saltatory conduction that leaps between the nodes of Ranvier.

08 Why the action potential matters in the clinic

Every step of this mechanism is a place where drugs act and disease strikes.

Local anaesthetics. Lidocaine and procaine work by **blocking voltage-gated Na^+ channels**: no Na^+ influx, no depolarisation, so pain fibres can neither fire nor conduct — a nerve block. The same target explains natural poisons such as **tetrodotoxin** (puffer fish) and **saxitoxin** (paralytic shellfish poisoning).

Potassium. Because the resting potential is set by the K^+ gradient, blood K^+ must be held in a narrow range. **Hyperkalaemia** — from a blocked cat's urethra, uroabdomen, hypoadrenocorticism (Addison's) or oliguric kidney injury — raises (depolarises) the resting potential; excitable cells first over-fire, then their Na^+ channels inactivate and cardiac conduction fails. It is a true cardiac emergency. **Hypokalaemia (hypokalemia)** does the reverse, hyperpolarising cells and causing weakness.

Calcium — a classic species split. Low blood calcium destabilises excitable membranes, but the result depends on the species. In **dogs and humans** it causes **tetany** — spontaneous, involuntary firing of motor nerves. In **ruminants** the same hypocalcaemia (hypocalcemia) instead **blocks** transmitter release at the neuromuscular junction, so the cow goes *down*: the flaccid paresis of **milk fever** (periparturient hypocalcaemia). Same electrolyte, opposite clinical picture.

Fuel for the pump. Finally, the whole system fails without ATP. A dog with an insulin-secreting tumour (**insulinoma**) whose blood glucose crashes can seizure, because starved neurons cannot power the Na^+/K^+ pump that maintains the resting potential — a direct clinical read-out of the biology on this page.

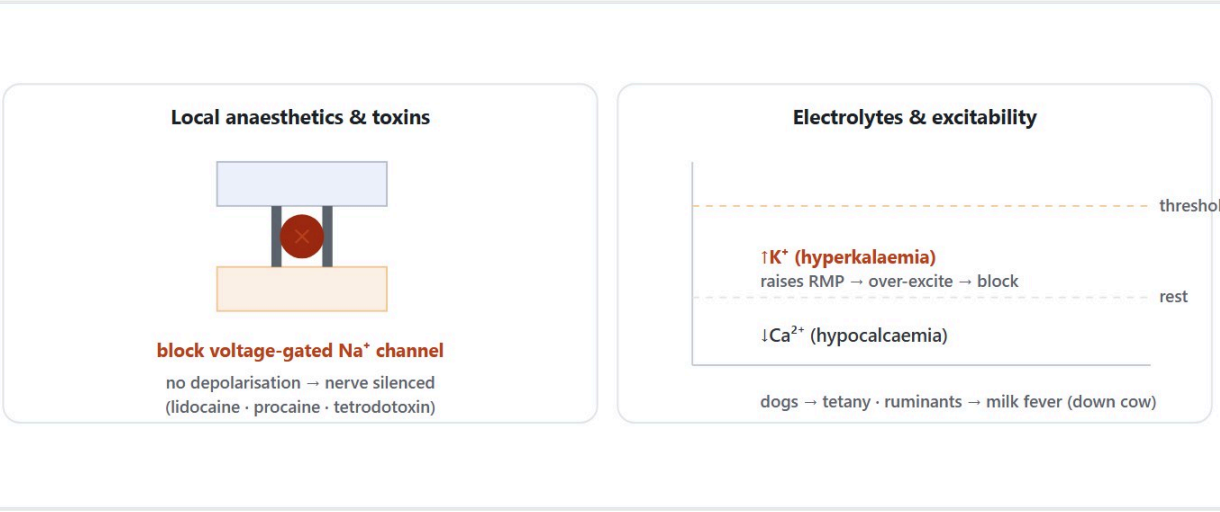


Fig 5 — Two ways the clinic meets the action potential: drugs/toxins that block the Na^+ channel, and electrolytes that shift the membrane toward or past threshold.

> **Red flag**
>
> Sudden flaccid weakness or collapse — suspect a neuromuscular or electrolyte emergency (hyperkalaemia from a blocked cat, hypocalcaemic milk fever in a fresh cow, tick paralysis, botulism). Check an ECG and electrolytes early; caught fast, these are treatable.

09 Quiz: test yourself

Eighteen questions across the neuron, the resting potential, threshold, the spike itself, saltatory conduction and the drugs and electrolytes that break it. Answers are beneath each question — cover them and work down.

1. What is an action potential, in one sentence?**Answer:** A brief, explosive **reversal of membrane voltage** that, once triggered, **regenerates itself** down the whole length of a nerve or muscle fibre without fading.2. If every spike is identical, how does the nervous system signal a stronger stimulus?**Answer:** By **frequency** — more action potentials per second. It cannot make a bigger impulse, so it makes more of them.3. What are the four regions of a neuron?**Answer: Dendrites** (the receiving surface, studded with receptors), the **cell body / soma** (about 20 µm, the metabolic factory), the **axon**, and the **presynaptic terminals** that transmit across the synapse.4. What is the resting membrane potential of a neuron, and what is the range across cell types?**Answer:** About **−70 mV**, inside negative. Neurons sit at roughly −70 to −80 mV; across cell types generally the range is about −40 to −90 mV.5. The resting voltage sounds small. Why is it not?**Answer:** Because it is held across a membrane **under 10 nanometres** thick — an electric field on the order of **100,000 volts per centimetre**.6. What does the Na^+/K^+ pump move, and what does it cost?**Answer: 3 Na^+ out for every 2 K^+ in**, per ATP. It is described as the single most energy-requiring process in the body and can consume **50–70% of the brain's energy**.7. What are the equilibrium potentials for K^+ and Na^+?**Answer:** K^+ about **−90 mV** (140 mM in, 4 mM out); Na^+ about **+70 mV** (15 mM in, 145 mM out). The resting potential sits near K^+ because the resting membrane is most permeable to it.8. Why does the pump's energy bill matter clinically?**Answer:** It runs on ATP from **glucose and oxygen, and neurons store neither**. Interrupt the supply and the gradients collapse within minutes.9. What is threshold, in millivolts?**Answer:** Roughly **10–20 mV more positive than rest** — around **−55 mV**.10. What is the difference between an EPSP and an IPSP?**Answer:** An **EPSP** opens Na^+ channels and moves the membrane more positive (say −75 to −65 mV), toward threshold. An **IPSP** opens K^+ channels and hyperpolarises, away from it.11. How long does an action potential take, and what is its peak voltage?**Answer: 2–3 ms** in most neurons, peaking around **+30 mV** as the membrane drives toward the Na^+ equilibrium potential.12. What makes the upstroke explosive?**Answer: Runaway positive feedback**: Na^+ entry makes the inside more positive, which opens still more voltage-gated Na^+ channels, which admits more Na^+.13. What two events end the spike?**Answer:** The Na^+ channels **inactivate** — they shut by themselves after about half a millisecond — and the slower voltage-gated **K^+ channels** open.14. How fast does a thin unmyelinated axon conduct?**Answer:** Around **0.5 m/s**; bare vertebrate axons rarely exceed **2 m/s**.15. What lays down myelin, and where?**Answer: Schwann cells** in peripheral nerves; **oligodendrocytes** in the brain and spinal cord.16. What are the nodes of Ranvier, and why does the spike leap between them?**Answer:** Bare gaps between myelinated internodes, **packed with voltage-gated Na^+ channels**. Only the nodes can regenerate the spike, so it jumps node to node — saltatory conduction.17. How do lidocaine and procaine work?**Answer:** They **block voltage-gated Na^+ channels**. No Na^+ influx, no depolarisation — so pain fibres can neither fire nor conduct.18. Why is hyperkalaemia an emergency in a blocked cat?**Answer:** A raised extracellular K^+ **depolarises the resting potential**. Excitable cells first over-fire, then their Na^+ channels **inactivate** and cardiac conduction fails.

10 The action potential — frequently asked questions

What is an action potential in simple terms?

It is the brief electrical "spike" a nerve or muscle cell fires to send a signal. The cell flips its membrane voltage from about −70 mV up to +30 mV and back in 2–3 ms, and that flip travels down the fibre without fading.

Why is the resting membrane potential negative?

Because the resting membrane is far more permeable to K^+ than Na^+. K^+ leaks out down its gradient and leaves negatively charged proteins behind, so the inside settles near the K^+ equilibrium potential (about −90 mV), giving a resting value near −70 mV.

Which ions move during an action potential?

Na^+ rushes in first, through voltage-gated Na^+ channels (depolarisation, the upstroke); then K^+ flows out, through voltage-gated K^+ channels (repolarisation, the downstroke). The Na^+/K^+ pump quietly restores the gradients afterwards.

What are the phases of an action potential?

Five, in order. **Resting** — the membrane sits near −70 mV. **Depolarisation** (depolarization) — a stimulus drags the membrane to threshold, voltage-gated Na^+ channels snap open and Na^+ floods in, driving the membrane sharply positive. **Repolarisation** (repolarization) — the Na^+ channels inactivate and slower voltage-gated K^+ channels open, so K^+ leaves and the charge is restored. **Hyperpolarisation** — those K^+

channels are slow to shut, so the membrane briefly overshoots below rest. **Recovery** — the sodium-potassium pump quietly re-establishes the gradients. The whole spike takes 2–3 ms; the pump works on for far longer.

What is the difference between a graded potential and an action potential?

Size and behaviour. A **graded potential** — an EPSP or IPSP at a synapse, or a receptor potential — is proportional to the stimulus, often only 2–3 mV, and it *decrements*: it fades with distance and time, and several can sum. An **action potential** is **all-or-none**: once threshold is reached at the axon hillock the spike is always the same size, and it regenerates itself along the axon without fading. Graded potentials decide *whether* the neuron fires; the action potential is *how* the message travels once that decision is made.

What is the refractory period, and why does it matter?

It is the window after a spike when the neuron cannot fire normally, and it exists because the Na^+ channels need time to reset. During the **absolute refractory period** no stimulus of any size will trigger another action potential — which is what forces the impulse to travel in *one direction* and sets a ceiling on firing frequency. During the **relative refractory period** a stronger-than-normal stimulus can fire the cell, because some Na^+ channels have reset while the membrane is still hyperpolarised. In the heart the same principle is what stops tetany: cardiac muscle has a refractory period so long that it cannot be re-excited until it has relaxed.

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When to refer

- Sudden generalised weakness or flaccid paralysis (suspected coonhound paralysis/polyradiculoneuritis, tick paralysis, botulism) — a neuromuscular emergency; stabilise and refer.
- Collapse, arrhythmia or profound weakness with suspected **hyperkalaemia** (blocked cat, uroabdomen, Addison's, oliguric kidney injury) — treat as a cardiac emergency (ECG, calcium gluconate).
- A recumbent periparturient cow (**milk fever**) or a tetanic/seizuring hypocalcaemic patient — correct calcium promptly.
- Progressive ataxia or paresis suggesting **demyelination** — image and refer for a full neurological work-up.

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3. Ion concentrations from PDA 3e (Fig 2.14); equilibrium potentials and the “three determinants” framing from Cunningham 6e (Ch 4). Species conduction velocities from PDA 3e (Ch 4).

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