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— STUDY NOTES

The Action Potential

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** silence nerves, why **hyperkalaemia** stops the heart, and why a calcium-starved cow goes down with milk fever.

INSIDE THESE NOTES

- What an action potential is
- Reaching threshold
- Propagation & myelin
- The resting membrane potential (~−70 mV)
- The action potential, phase by phase
- Clinical — where it bites

LEVEL
Vets & veterinary students

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2026-07-01

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The Action Potential

STUDY NOTES · BASIC SCIENCES · PHYSIOLOGY · UPDATED 2026-07-01

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1. What an action potential is
2. The resting membrane potential (~−70 mV)
3. Reaching threshold
4. The action potential, phase by phase
5. Propagation & myelin
6. Clinical — where it bites

LEARNING OBJECTIVES

After working through these notes you will be able to:

- ✓ Define the resting membrane potential and explain the three factors that create it.
- ✓ Explain why selective K^+ permeability and the Na^+/K^+ pump set the −70 mV resting potential.
- ✓ Describe the phases of the action potential in terms of voltage-gated Na^+ and K^+ channels.
- ✓ Explain threshold, the all-or-none principle and the refractory period.
- ✓ Describe saltatory conduction and link the action potential to clinical problems (local anaesthetics, hyperkalaemia, hypocalcaemia).

TL;DR

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** silence nerves, why **hyperkalaemia** stops the heart, and why a calcium-starved cow goes down with milk fever.

AT A GLANCE

DEFINITION	A brief, all-or-none, self-propagating reversal of membrane voltage that carries a signal
RESTING POTENTIAL	~−70 mV, inside negative; set mainly by K^+ leaking out
THREE DETERMINANTS	Na^+/K^+ pump · K^+ moving to equilibrium · K^+-permeable membrane
THRESHOLD	~10–20 mV above rest (≈ −55 mV)
DEPOLARISATION	Voltage-gated Na^+ channels open → Na^+ in → toward +30 mV
REPOLARISATION	Na^+ inactivates, voltage-gated K^+ opens → K^+ out → back to rest
CONDUCTION	Saltatory — jumps node to node along myelinated axons
CLINICAL	Local anaesthetics, hyperkalaemia, hypocalcaemia, demyelination

01 What an action potential is

- **The nerve's signal:** a brief, **all-or-none**, self-regenerating reversal of membrane voltage that runs down an axon **without fading**.
- **All-or-none:** above threshold it always fires full-size; stimulus strength is coded by **frequency**, not amplitude.
- **Four neuron regions:** dendrites (receive) → cell body/soma (integrate + build) → axon (conduct) → presynaptic terminals (transmit). The AP starts at the **axon initial segment**.

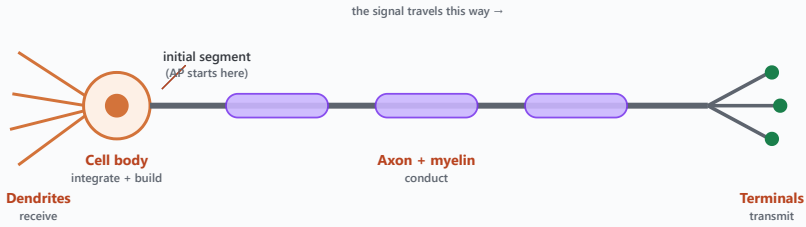

Fig 1 — The neuron's four regions; the action potential begins at the initial segment.

02 The resting membrane potential (~−70 mV)

- **Value:** about **−70 mV**, inside negative (−70 to −80 mV in neurons).
- **Three determinants:** (1) the **Na^+/K^+ pump** (3 Na^+ out / 2 K^+ in) builds the gradients; (2) **K^+ moves toward equilibrium** (leaks out, leaves protein anions behind); (3) the membrane is **far more permeable to K^+** than Na^+ → RMP sits near E_K.
- **Equilibrium potentials:** $E_K \approx -90$ mV, $E_{Na} \approx +70$ mV (Nernst); the small Na^+ leak holds RMP at −70, not −90.
- **Expensive:** the pump can use ~**50–70% of brain ATP** — it runs on glucose + O_2, which neurons don't store (see insulinoma).

ION	IN (MM)	OUT (MM)	EQUILIBRIUM
K^+	140	4	≈ −90 mV
Na^+	15	145	≈ +70 mV
Cl^-	7	120	passive
Ca^{2+}	0.0001	1	strongly +

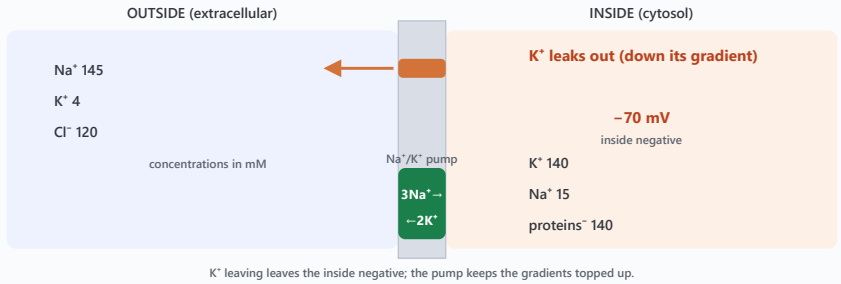

Fig 2 — The pump builds the gradients; K^+ leaking out sets the inside negative.

03 Reaching threshold

- **Threshold ≈ −55 mV** (~10–20 mV above rest).
- **EPSP** = depolarising (Na^+ in) → toward firing; **IPSP** = hyperpolarising (K^+ out) → away from firing.
- Postsynaptic potentials are **graded & decremental** (small, brief, fade with distance); the **initial segment sums** them (integration) and fires only if excitation wins.

04 The action potential, phase by phase

- **Sequence (2–3 ms):** rest → **depolarisation** (voltage-gated **Na^+** in, positive feedback, peak ~+30 mV) → **repolarisation** (Na^+ inactivates; voltage-gated **K^+** out) → **after-hyperpolarisation** → rest.
- **Up = Na^+ in, down = K^+ out.**
- **Refractory period:** Na^+ channels inactivated → **absolute** (can't fire) then **relative** (needs a stronger stimulus). Forces **one-way** travel and caps firing rate.

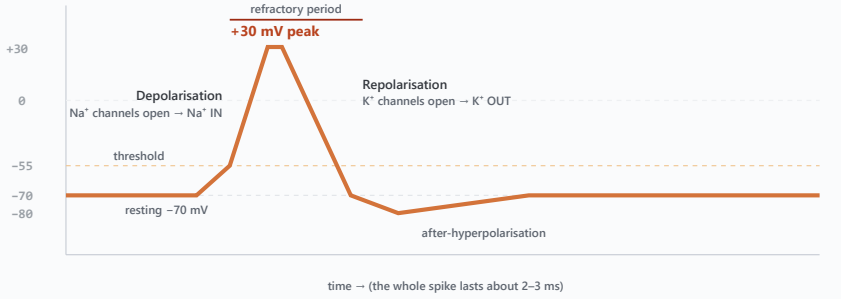

Fig 3 — One spike: threshold → Na^+ up-stroke → K^+ down-stroke → after-hyperpolarisation.

05 Propagation & myelin

- **Regenerates itself:** Na^+ influx spreads as a local **electrotonic current** that drags the next patch to threshold; refractoriness behind keeps it **one-way**.
- **Speed ∝ diameter + myelination:** bare axon ~0.5 m/s (<2 m/s); myelinated up to **~100 m/s (cat ~120, human ~70)**.
- **Saltatory conduction:** myelin (Schwann cells PNS / oligodendrocytes CNS) insulates internodes; the AP **jumps node to node** at the **nodes of Ranvier** (dense Na^+ channels).
- **Demyelination** slows or blocks conduction → weakness, ataxia (axon intact).

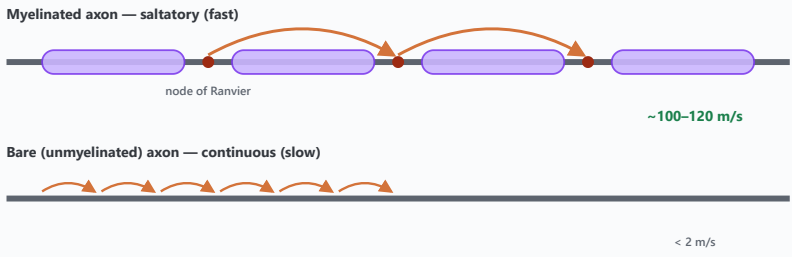

Fig 4 — Myelin turns slow continuous conduction into fast saltatory conduction.

06 Clinical — where it bites

- **Local anaesthetics** (lidocaine, procaine) and **TTX / saxitoxin** block **voltage-gated Na^+ channels** → no depolarisation → nerve silenced.
- **Hyperkalaemia** (blocked cat, Addison's, kidney injury): raises/depolarises RMP → over-excite then Na^+-channel block → **cardiac emergency**. **Hypokalaemia**: hyperpolarises → weakness.
- **Hypocalcaemia — species split:** dogs/humans → **tetany** (spontaneous firing); ruminants → NMJ block → **milk fever** (down, flaccid cow).
- **No ATP, no potential:** insulinoma (hypoglycaemia) starves the pump → seizures.

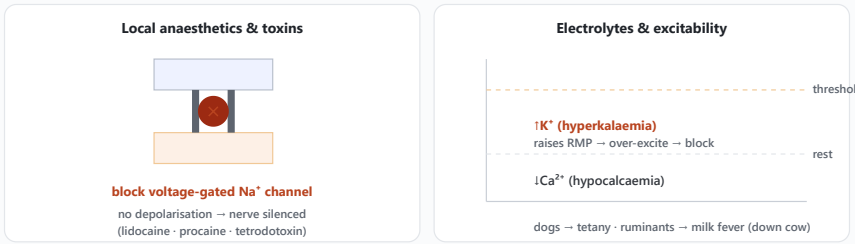

Fig 5 — Na^+-channel blockers silence nerves; K^+/Ca^{2+} shifts change excitability.

RED FLAG

Sudden flaccid weakness or collapse — think neuromuscular / electrolyte emergency (hyperkalaemia from a blocked cat, hypocalcaemic milk fever, tick paralysis, botulism). Check an ECG and electrolytes early.

> *Every step of the action potential is a place a drug can act or a disease can strike — which is why this one mechanism underlies so much of the clinic.*
>
> — neurophysiology, after Cunningham 6e & PDA 3e.

KEY TERMS — QUICK GLOSSARY

Resting membrane potential (RMP)	The steady voltage across a resting cell membrane — about −70 mV, inside negative.
Depolarisation	A shift of the membrane potential toward zero / positive (less negative).
Repolarisation	Return of the membrane potential toward its negative resting value.
Hyperpolarisation	A shift to a more negative potential than rest.
Threshold	The critical voltage (~10–20 mV above rest) that triggers an action potential.
All-or-none	An action potential either fires fully or not at all; stimulus strength is coded by frequency, not amplitude.
Voltage-gated channel	An ion channel that opens or closes in response to membrane voltage (Na^+, K^+, Ca^{2+}).
Refractory period	The brief interval after an AP when Na^+ channels are inactivated and a new AP is hard or impossible to fire.

Equilibrium (Nernst) potential	The voltage at which an ion's chemical and electrical forces balance — $E_K \approx -90$ mV, $E_{Na} \approx +70$ mV.
Saltatory conduction	Node-to-node 'jumping' of the impulse along a myelinated axon, greatly speeding conduction.

QUICK REVISION — REMEMBER THESE

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.

MEMORY AIDS

Na^+ in, K^+ out — **Na^+ rushes IN** to depolarise (the up-stroke); **K^+ flows OUT** to repolarise (the down-stroke). Up = in, down = out.

−70 → threshold → +30 → −70 — Rest at −70, climb to threshold, spike toward +30, fall back — one lap in 2–3 ms.

Salt jumps — **Salt**atory conduction = the impulse **jumps** the myelin gaps, node to node.

TEST YOURSELF — ACTIVE RECALL

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

1. **Why is the inside of a resting neuron negative?**
2. **Name the three determinants of the resting membrane potential.**
3. **Which ion moves first in an action potential, and which restores rest?**
4. **What does 'all-or-none' mean, and how is signal strength encoded?**
5. **What causes the refractory period and why does it matter?**
6. **How does myelin speed conduction?**
7. **How do local anaesthetics work?**

8. **Why is hyperkalaemia dangerous to the heart?**

ANSWERS

1. The membrane is far more permeable to K^+ than Na^+; K^+ leaks out down its gradient and leaves trapped protein anions behind, so the RMP sits close to the K^+ equilibrium potential (~−90 mV), around −70 mV.
2. The Na^+/K^+ pump (builds the gradients), movement of a permeant ion (K^+) toward its equilibrium, and the differentially (K^+-)permeable membrane.
3. Na^+ enters first (depolarisation) through voltage-gated Na^+ channels; K^+ then exits (repolarisation) through voltage-gated K^+ channels.
4. Above threshold the AP always fires at full size; a stronger stimulus doesn't make a bigger spike — intensity is coded by the frequency of action potentials.
5. Voltage-gated Na^+ channels are inactivated for about a millisecond; this forces the impulse to travel one way and limits the maximum firing rate.
6. It insulates the internodes so the impulse regenerates only at the nodes of Ranvier, jumping node to node (saltatory conduction) — up to ~100–120 m/s versus <2 m/s in bare axons.
7. They block voltage-gated Na^+ channels, so pain fibres cannot depolarise, fire or conduct.
8. High extracellular K^+ raises (depolarises) the resting potential, first over-exciting then inactivating Na^+ channels — disrupting cardiac action potentials and risking arrest.

WHEN TO REFER OR ESCALATE

- 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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