The Action Potential

"Spikes"

Computational Neuroscience

University of Bristol

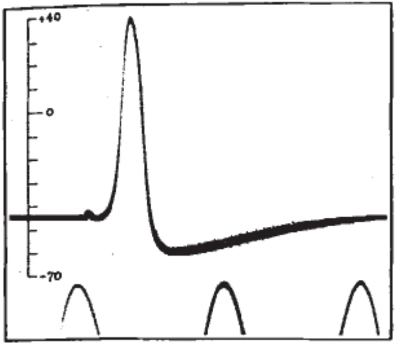
M Rule

Learning outcomes:

- Describe what an action potential is, what challenges it solves, etc.
- Describe the phases of an action potential, and the roles of various ionic currents and channels in each phase
- Distinguish mechanisms of relative vs. absolute refractory period

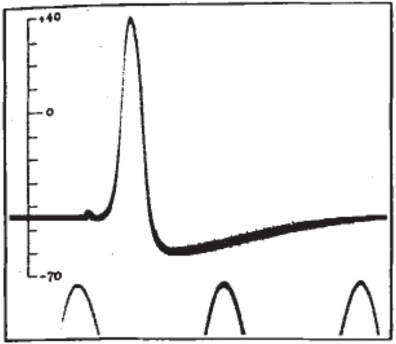
Spikes

Neurons use spikes for reliable long-range communication



Oscilloscope trace from *Action Potentials Recorded from Inside a Nerve Fibre*, Hodgkin and Huxley (1939): ***"Action potential recorded between inside and outside of axon. Time marker 500 cycles/sec. The vertical scale indicates the potential of the internal electrode in millivolts, the sea water outside being taken at zero potential."***

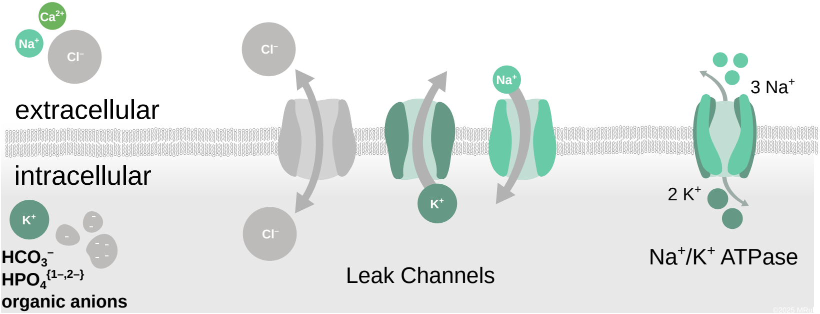
Neurons use spikes for reliable long-range communication



The action potential ("spike") is a sharp rise and fall in cell's membrane voltage that's produced when the cell exceeds a **threshold voltage**.

All-or-nothing, binary $\{0, 1\}$

- Robust: regenerative wave that can travel long distances along axons/dendrites without decay


Ca2+
Na+
Cl−
extracellular
intracellular
K+
HCO3−
HPO4{1−,2−}
organic anions
Cl−
Cl−
K+
Na+
Leak Channels
3 Na+
2 K+
Na+/K+ ATPase

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The Action Potential

Initiation Voltage-gated Na^+ channels have a threshold of $\sim$ -55 mV; If the v_m exceeds this, Na^+ channels open. Na^+ flows *into* cell, pulling v_m toward $E_{\mathrm{Na}^+} \sim +60$ mV

Depolarisation The cell spikes if positive feedback from voltage-gated Na^+ channels overwhelms other currents: v_m, crossing 0 mV and reaching a peak at $\sim +30$–40 mV

Peak v_m stops rising because the depolarisation…

- Inactivates voltage-gated Na^+ channels (Na^+ current stops)
- Opens voltage-gated K^+ channels (threshold $\sim$ -40 – -30 mV), leading to …

Repolarisation K^+ efflux pulls v_m toward $E_{\mathrm{K}^+} \sim$ -95 mV;

Hyperpolarization Voltage-gated K^+ channels close slowly, continue to pull cell below v_{rest} to $\sim$ -80 – -75 mV.

Na^+ inactivation threshold -80 – -60 mV (near rest!), surprising?

ask me why the peak is at $\approx +40$ mV?

Refractory period

Absolute

- $\sim$ 1–2 ms
- Enough voltage-gated Na^+ channels are inactive that no physiological input can trigger a new spike

Relative

- $\sim$ 2–5 ms
- Voltage-gated Na^+ channels have started to de-inactivate
- Some voltage-gated K^+ channels remain open
- Cell remains hyperpolarized, and requires a strong input to spike.

Exam confusions

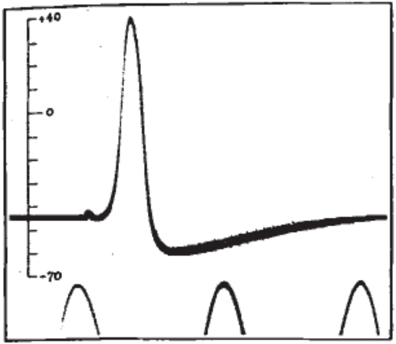
The Na^+/K^+ ATPase is *not* immediately required to repolarise the neuron after an action potential. Each spike uses a small amount of the electrical potential stored in Na^+ and K^+ gradients.

Exam confusions

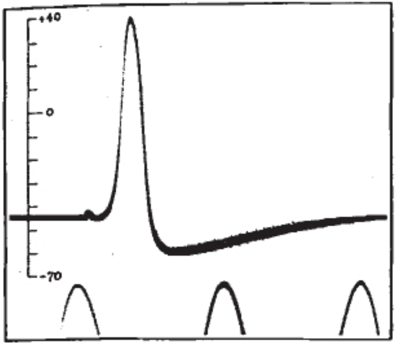
Try not to confuse

1. *voltage-gated* Na^+ and K^+ channels involved in the *action potential*
2. *ligand-gated* ionotropic neurotransmitter receptors
   (synapse: chemical $\rightarrow$ electrical)
3. *voltage-gated* CA^{2+} channels in axonal terminals
   (synapse: electrical $\rightarrow$ chemical)

label the phases of this action potential



what voltage gates and ion fluxes are involved in each phase?



Draw an action potential from member, including realistic voltages ±15 mV, and label its key phases

More realism

Action potentials *travel* out along axons

- Regenerative soliton wave
- Refractory period ensures directed propagation

Cable equation

Saltatory conduction

- Myelin
- Nodes of Ranvier

and also sometimes back into dendrites

soliton = nonlinear wave that holds a fixed shape

Neural computation and communication is implemented via electrochemical reactions

Signal transmission via spikes

1. *Spike initiation and propagation:* via voltage-gated ion channels
2. *Pre-synaptic:* axonal terminal depolarises $\longrightarrow$ neurotransmitter release
3. *Post-synaptic:* chemical signals $\longrightarrow$ electrical currents in the dendrite
4. (repeat)

end