MEGA LECTURE

IB DP BIOLOGY

Theme C: Interaction and Interdependence

C2.2 Neural Signalling

Revision Notes · Standard and Higher Level

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What the syllabus requires

By the end of C2.2 you should be able to work confidently with each of the understandings below. Use this checklist for a final revision sweep. Items marked **(HL)** are examined only at Higher Level; everything else is common to SL and HL.

Understanding	You should be able to...
Neurons and nerve impulses	Describe neurons as cells specialised for the rapid transmission of electrical signals as nerve impulses.
Neuron structure	Label dendrites, cell body, axon, myelin sheath, nodes of Ranvier and axon terminals, and relate each to function.
Resting potential	Explain the resting potential of about −70 mV in terms of the sodium−potassium pump and membrane permeability.
Action potential	Sequence depolarisation, repolarisation and the return to rest using voltage-gated ion channels, and state the all-or-nothing principle.
Propagation	Explain how local currents move an impulse along an axon and how the refractory period makes travel one-way.
Saltatory conduction	Explain how myelination speeds conduction and state the factors affecting conduction speed.
Synaptic transmission	Sequence chemical transmission across a synapse from Ca^{2+} influx to the postsynaptic potential and neurotransmitter removal.
Excitation and inhibition	Distinguish excitatory from inhibitory synapses and describe the neuromuscular junction.
Drugs and integration (HL)	Describe how drugs and toxins act at synapses and explain spatial and temporal summation and integration.

> **Exam note:** SL and HL share the core of C2.2 — neuron structure, the resting and action potentials, propagation and basic synaptic transmission. HL candidates additionally study the action of drugs and toxins at synapses, spatial and temporal summation, and integration. HL-only material is flagged (HL) throughout.

1. Neurons and the nervous system

Multicellular animals must coordinate the activity of trillions of cells and respond to changes in their surroundings within milliseconds. The **nervous system** provides this rapid, precisely targeted communication. It gathers information through receptors, processes it, and sends instructions to effectors (muscles and glands). The messages travel as electrical **nerve impulses** along specialised cells called **neurons**.

Neurons carry signals quickly and along a defined path, which is why the nervous system is suited to responses that must be fast and localised, such as pulling a hand from a hot surface. This contrasts with the endocrine system (C2.1), whose hormones travel in the blood and act more slowly and more widely.

The parts of a neuron

A neuron is built for one-way transmission of a signal from one end to the other. Its structure reflects this job.

Structure	Description	Function
Dendrites	Short, branched extensions of the cell body	Receive signals from receptors or other neurons and carry them toward the cell body
Cell body (soma)	Contains the nucleus and most organelles	Site of metabolism and protein synthesis; integrates incoming signals
Axon	A single long fibre extending from the cell body	Conducts the nerve impulse away from the cell body toward the terminals
Myelin sheath	Insulating layers of membrane from Schwann cells wrapped around the axon	Electrically insulates the axon and greatly speeds conduction
Nodes of Ranvier	Short unmyelinated gaps between Schwann cells	Sites where the impulse is regenerated; allow the impulse to jump along the axon
Axon terminals	Fine branched endings of the axon	Form synapses with the next cell and release neurotransmitter

Schwann cells are the glial cells that produce the myelin sheath in peripheral nerves. Each wraps repeatedly around a section of axon, leaving the gaps called nodes of Ranvier between adjacent cells.

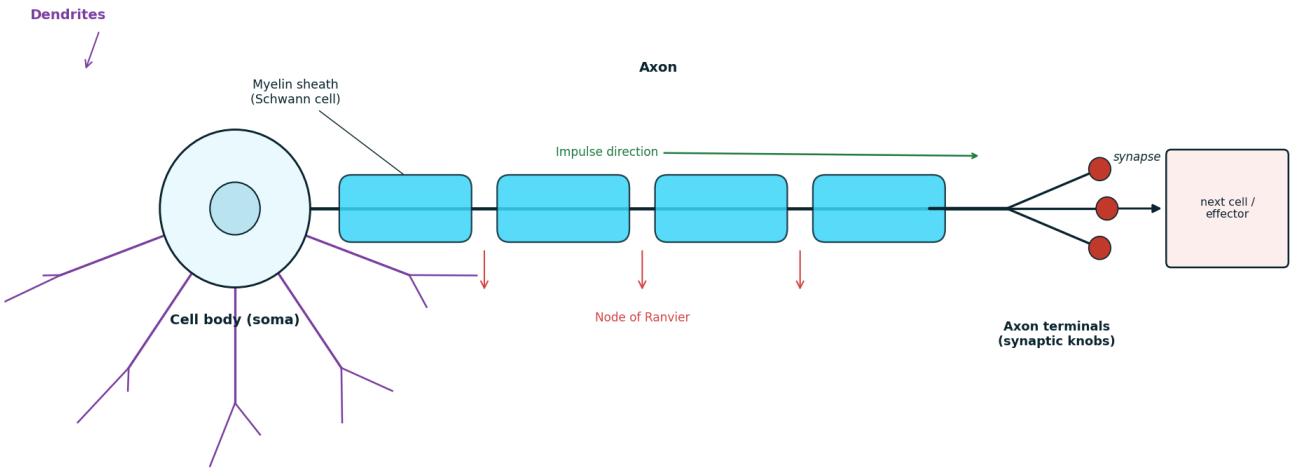

Figure 1. Structure of a neuron: dendrites receive signals and carry them to the cell body; the myelinated axon (here 4 Schwann-cell segments separated by 3 nodes of Ranvier) conducts the impulse toward the axon terminals, which form a synapse with the next cell.

Three functional types of neuron

- **Sensory (afferent) neurons** carry impulses from receptors, such as those in the skin, eye or ear, toward the central nervous system.
- **Relay (interneuron) neurons** lie within the brain and spinal cord and connect neurons to one another, allowing processing and integration.
- **Motor (efferent) neurons** carry impulses from the central nervous system to effectors, so that muscles contract or glands secrete.

Putting the neurons together: the reflex arc

The three neuron types form a pathway. A rapid, automatic response to a stimulus, such as withdrawing a hand from a sharp object, follows a **reflex arc** that runs through the spinal cord without waiting for the brain, which is why it is so fast.

- A **receptor** detects the stimulus and generates an impulse.
- A **sensory neuron** carries the impulse to the spinal cord.
- A **relay neuron** in the spinal cord passes it on (and can signal the brain).
- A **motor neuron** carries the impulse to an effector.
- The **effector** (a muscle or gland) carries out the response.

Each junction between these neurons is a synapse, so the reflex arc is also a chain of synapses — a useful reminder that impulses pass both along axons and across synapses on their way from stimulus to response.

> **Exam tip:** A nerve impulse is not a flow of electrons like current in a wire. It is a wave of electrical change (a moving region of membrane depolarisation) produced by ions crossing the membrane. Describe it in those terms in extended answers.

Nervous compared with hormonal signalling

Both systems coordinate the body, but they differ in speed, route and duration. This comparison is a common short-answer question.

Feature	Nervous system	Endocrine system
Signal	Electrical impulse, then chemical at synapses	Chemical (hormone) only
Transport route	Along neurons to a specific target	In the blood to all tissues
Speed	Very fast (milliseconds)	Slower (seconds to hours)
Duration of effect	Short-lived	Often longer-lasting
Specificity	Precisely targeted to one effector	Any cell with the right receptor responds

2. The resting potential

A neuron that is not transmitting an impulse is not electrically “off.” It actively maintains a voltage difference across its membrane called the **resting potential**, typically about −70 mV. The minus sign means the inside of the axon is negative relative to the outside. A membrane in this state is said to be **polarised**.

How the resting potential is set up

Two things acting together produce and maintain the resting potential:

- **The sodium–potassium pump.** This membrane protein uses ATP to pump 3 Na^+ out of the cell for every 2 K^+ it pumps in. Because it is an active process moving unequal numbers of ions, it builds concentration gradients (high Na^+ outside, high K^+ inside) and removes more positive charge than it brings in.

- **Membrane permeability.** At rest the membrane is far more permeable to K^+ than to Na^+, because more potassium leak channels are open. K^+ therefore diffuses out down its gradient faster than Na^+ leaks in, leaving the inside more negative. Large negatively charged proteins trapped inside the cell add to this negativity.

The net result is a steady negative charge inside relative to outside. The pump consumes energy continuously; without ATP the gradients would run down and the resting potential would collapse.

> **Common misconception:** The resting potential is maintained by **both** the sodium−potassium pump **and** the differential permeability of the membrane. Answers that mention only the pump, or only diffusion of K^+, lose marks. Name both.

3. The action potential

An **action potential** is a rapid, temporary reversal of the membrane potential that travels along the axon as a nerve impulse. It is produced by **voltage-gated ion channels** that open and close in response to changes in membrane voltage. The whole event lasts only a few milliseconds.

Stages of an action potential

- **Stimulus and threshold.** A stimulus makes the membrane slightly less negative. If this reaches the **threshold** (around −55 mV), voltage-gated Na^+ channels open and an action potential is triggered. A stimulus too weak to reach threshold produces no impulse.
- **Depolarisation.** Voltage-gated Na^+ channels open and Na^+ floods into the axon down its gradient. The inside rapidly becomes positive, reaching about +30 mV. The influx of Na^+ makes nearby membrane reach threshold too, so the process is self-propagating.
- **Repolarisation.** The Na^+ channels close and voltage-gated K^+ channels open. K^+ flows out of the axon down its gradient, so the inside becomes negative again and the membrane returns toward the resting value.
- **Hyperpolarisation.** The K^+ channels are slow to close, so a little too much K^+ leaves and the potential briefly dips below −70 mV.
- **Return to rest.** The K^+ channels close and the sodium−potassium pump, with the leak channels, restores the resting distribution of ions and the −70 mV resting potential.

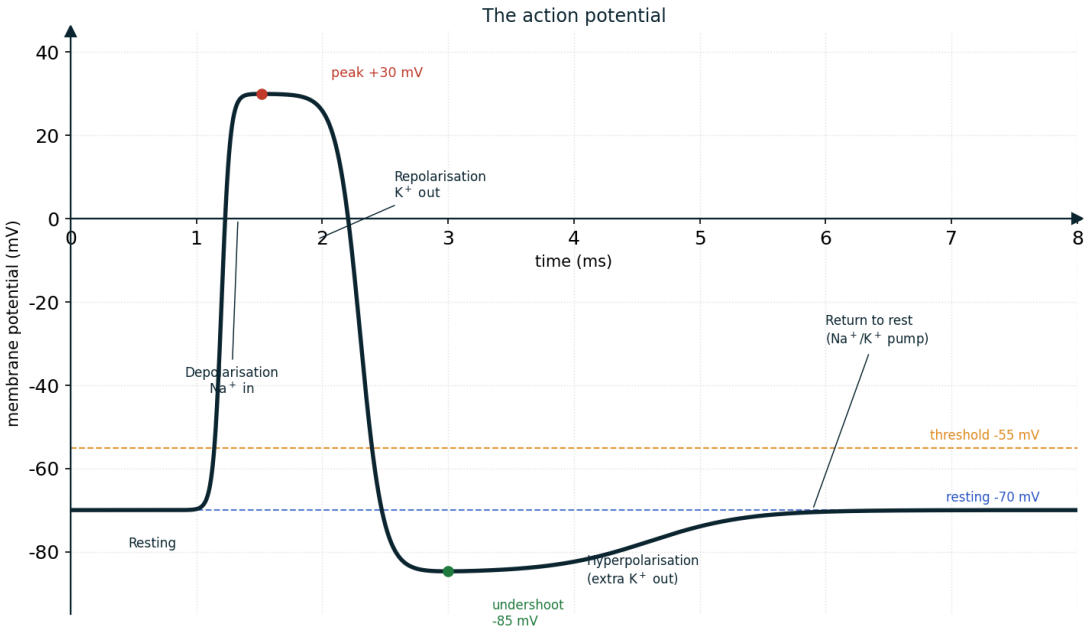

Figure 2. The action potential (voltages audited against the text): resting −70 mV → threshold −55 mV triggers depolarisation (Na^+ in) to a peak of +30 mV → repolarisation (K^+ out) → brief hyperpolarisation (modelled −85 mV) → return to resting via the Na^+/K^+ pump.

Summary of the stages

Stage	Channel event	Ion movement	Membrane potential
Resting	Na^+ and K^+ voltage-gated channels closed; pump active	Pump: 3 Na^+ out, 2 K^+ in	About −70 mV
Depolarisation	Voltage-gated Na^+ channels open	Na^+ rushes in	Rises to about +30 mV
Repolarisation	Na^+ channels close; K^+ channels open	K^+ moves out	Falls back toward rest
Hyperpolarisation	K^+ channels slow to close	A little extra K^+ out	Dips below −70 mV
Recovery	Channels closed; pump and leak channels active	Ion gradients restored	Returns to −70 mV

The all-or-nothing principle

An action potential is **all-or-nothing**: provided the stimulus reaches threshold, an action potential of the same size and shape is always produced; below threshold, nothing happens. A stronger stimulus does not make a bigger action potential. Instead, the strength of a stimulus is coded by the **frequency** of action potentials (and by how many neurons fire), not by their size.

The refractory period

Immediately after an action potential the membrane cannot easily be excited again. This is the **refractory period**. During it the Na^+ channels are temporarily inactivated. The refractory period limits the maximum firing frequency and, crucially, ensures the impulse travels in one direction only.

Worked example 1 — reading the graph

An action-potential graph plots membrane potential (mV) on the y-axis against time (ms) on the x-axis. Identify the region where (a) Na^+ channels are open and (b) K^+ channels are open, and state the value at rest.

(a) The **steep upstroke** from about −55 mV up to +30 mV is depolarisation, when voltage-gated Na^+ channels are open and Na^+ enters.

(b) The **downstroke** from +30 mV back toward rest is repolarisation, when voltage-gated K^+ channels are open and K^+ leaves. The dip just below the resting line is hyperpolarisation.

The flat baseline before and after is the resting potential at −70 mV.

4. Propagation along the axon

An action potential at one point on the axon must trigger the next region so the impulse travels the whole length. This is achieved by **local currents**.

- Where the membrane is depolarised, the inside is momentarily positive while the region just ahead is still at resting potential (inside negative).
- This difference makes ions flow sideways within the axon — a local current — which depolarises the next patch of membrane to threshold.
- Voltage-gated Na^+ channels there open, a new action potential is generated, and the sequence repeats along the axon. The signal is therefore regenerated at full size at each point and does not fade.

One-way travel

Local currents spread in both directions, so why does the impulse not travel backward? Because the region just behind the action potential is in its **refractory period**: its Na^+ channels are inactivated and cannot reopen immediately. The impulse can therefore only move forward, into membrane that has not yet fired. This is why nerve impulses are unidirectional along an axon.

> **Exam tip:** If asked why an impulse travels in one direction, the marking point is the **refractory period behind the action potential**, not the structure of the synapse. Synapses enforce one-way transmission between neurons; the refractory period enforces it within a single axon.

5. Saltatory conduction and conduction speed

In a **myelinated** neuron the axon is insulated by the myelin sheath except at the nodes of Ranvier. Because the myelin prevents ions crossing the membrane, action potentials can only be generated at the nodes. The local currents therefore flow from one node to the next, and the impulse appears to **jump** from node to node. This is called **saltatory conduction** (from the Latin *saltare*, to leap).

Because the membrane only has to depolarise at the nodes rather than continuously along its whole length, saltatory conduction is much **faster** and uses less energy than conduction in an unmyelinated axon of the same diameter.

Factors affecting conduction speed

Factor	Effect on speed	Reason
Myelination	Myelinated axons conduct much faster	Saltatory conduction lets the impulse jump between nodes instead of moving continuously
Axon diameter	Wider axons conduct faster	A larger diameter offers less resistance to the local currents flowing along the axon
Temperature	Higher temperature (up to a point) increases speed	Ions diffuse faster and channel proteins operate more quickly when warmer; in endotherms this is stable

These three factors explain why some animals achieve fast responses in different ways: vertebrates rely mainly on myelination, whereas some invertebrates such as squid use very wide (giant) axons to carry escape signals quickly.

Myelinated compared with unmyelinated axons

Feature	Myelinated axon	Unmyelinated axon
Conduction	Saltatory: impulse jumps node to node	Continuous along the whole membrane
Speed	Fast (can exceed 100 m s^{-1})	Slower
Energy use	Lower: only nodes repolarise	Higher: whole membrane repolarises
Where found	Most vertebrate motor and sensory neurons	Many short or slow-signalling neurons

Worked example 2 — explaining saltatory conduction

Explain why a myelinated neuron conducts an impulse faster than an unmyelinated one of the same diameter. (3 marks)

1. In a myelinated axon the sheath insulates the membrane, so ion movement and action potentials can only occur at the nodes of Ranvier.

2. Local currents flow directly from one node to the next, so the impulse jumps between nodes (saltatory conduction) instead of depolarising every part of the membrane.

3. Fewer regions have to depolarise, so the impulse travels along the axon much more quickly than the continuous conduction seen in an unmyelinated axon.

6. Synapses and chemical transmission

A **synapse** is the junction between two neurons, or between a neuron and an effector. The two cells do not touch: they are separated by a tiny gap, the **synaptic cleft**. Because an electrical impulse cannot cross the gap directly, the signal is carried across by a chemical messenger, a **neurotransmitter**. This is **chemical synaptic transmission**.

Structure of a synapse

- The **synaptic knob (presynaptic terminal)** is the swollen end of the axon; it contains many mitochondria and **vesicles** filled with neurotransmitter.
- The **synaptic cleft** is the narrow gap between the presynaptic and postsynaptic membranes.

- The **postsynaptic membrane** carries **receptor** proteins that are specific to the neurotransmitter and are often themselves ligand-gated ion channels.

Steps of transmission

- **1. Arrival of the impulse.** An action potential arrives at and depolarises the presynaptic membrane.
- **2. Calcium influx.** Depolarisation opens voltage-gated Ca^{2+} channels, and Ca^{2+} flows into the synaptic knob.
- **3. Vesicle fusion.** The Ca^{2+} causes vesicles to move to and fuse with the presynaptic membrane.
- **4. Release (exocytosis).** Neurotransmitter is released into the synaptic cleft by exocytosis.
- **5. Diffusion.** The neurotransmitter diffuses across the cleft.
- **6. Binding.** It binds to specific receptors on the postsynaptic membrane, opening ligand-gated ion channels.
- **7. Postsynaptic potential.** Ions flow through the opened channels, changing the postsynaptic membrane potential; if the change reaches threshold, a new action potential is triggered in the postsynaptic neuron.

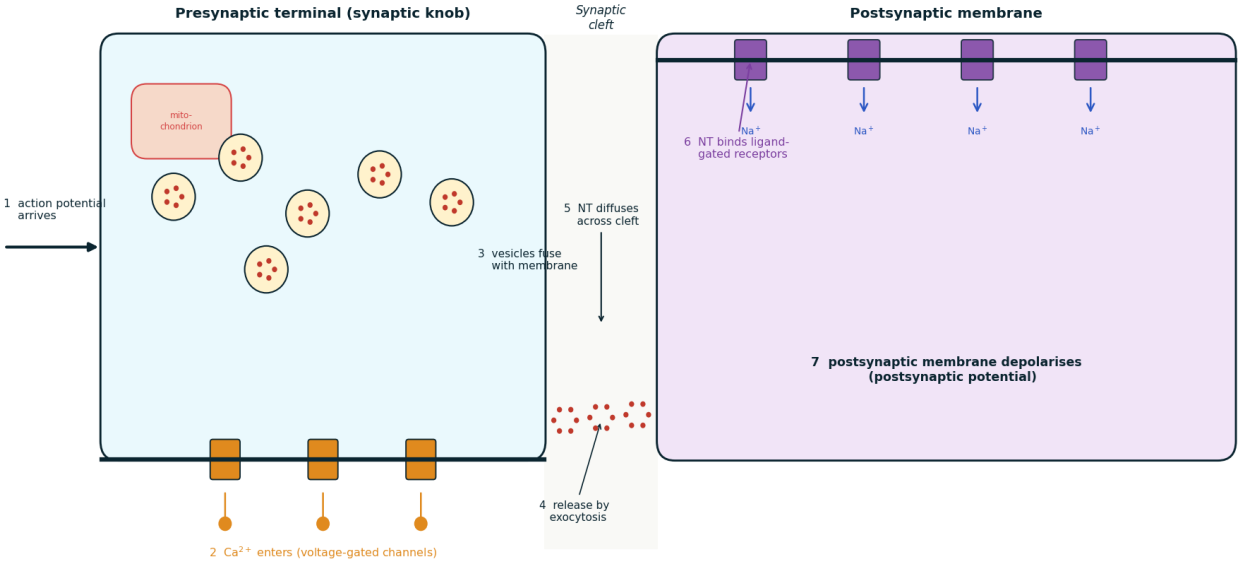

Figure 3. Chemical synaptic transmission: Ca^{2+} influx (2) triggers vesicle fusion (3) and exocytosis of neurotransmitter (4), which diffuses across the cleft (5), binds postsynaptic receptors (6) and opens ion channels (e.g. Na^{+}), depolarising the postsynaptic membrane (7). All 7 numbered steps match the sequence described in the text.

Removing the neurotransmitter

The signal must be switched off so the synapse can respond to new impulses. The neurotransmitter is quickly removed from the cleft, either by an **enzyme** that breaks it down (for example acetylcholinesterase breaking down acetylcholine) or by **reuptake** into the presynaptic neuron. This stops continuous stimulation of the postsynaptic cell.

Different synapses use different neurotransmitters — for example acetylcholine at the neuromuscular junction, and others such as dopamine, serotonin, GABA and glutamate within the central nervous system. Whether a neurotransmitter is excitatory or inhibitory depends on the receptor it binds to, not on the neurotransmitter alone.

> **Exam tip:** Synapses transmit in one direction only: vesicles of neurotransmitter are on the presynaptic side and receptors on the postsynaptic side. This structural asymmetry — not the refractory period — is the reason signals cross a synapse one way.

7. Excitatory and inhibitory synapses; the neuromuscular junction

Not every synapse pushes the postsynaptic neuron toward firing.

- At an **excitatory synapse** the neurotransmitter opens channels that let positive ions (such as Na^+) into the postsynaptic cell. This **depolarises** the membrane, making it more likely to reach threshold and fire.
- At an **inhibitory synapse** the neurotransmitter opens channels that let Cl^- in or K^+ out. This **hyperpolarises** the membrane, making it less likely to reach threshold.

Feature	Excitatory synapse	Inhibitory synapse
Ion movement	Positive ions (e.g. Na^+) enter	Cl^- enters or K^+ leaves
Effect on membrane	Depolarises the postsynaptic membrane	Hyperpolarises the postsynaptic membrane
Effect on firing	More likely to reach threshold and fire	Less likely to reach threshold

Summation (in brief)

A single excitatory input is usually too small to reach threshold on its own. The postsynaptic neuron **adds up** the excitatory and inhibitory inputs it receives; only if the total depolarisation reaches threshold does it fire. This adding-up is called **summation** (developed further at HL, section 8).

The neuromuscular junction

The **neuromuscular junction** is the synapse between a motor neuron and a skeletal muscle fibre. It works by the same steps as a neuron-to-neuron synapse. The neurotransmitter released is **acetylcholine**. When it binds to receptors on the muscle fibre membrane, the membrane depolarises and this triggers the muscle fibre to contract. Acetylcholine is then broken down by the enzyme acetylcholinesterase so the muscle can relax and respond to the next impulse.

8. Drugs, toxins and integration (HL)

Because synapses are chemical, they are the main site where drugs and toxins alter the nervous system. Any chemical that changes neurotransmitter release, receptor binding or neurotransmitter removal will change the message that crosses the synapse.

How chemicals act at synapses (HL)

- Some chemicals **mimic** a neurotransmitter by binding to and activating its receptors (agonists).
- Some **block** receptors so the neurotransmitter cannot act (antagonists).
- Some **block the enzyme** that breaks the neurotransmitter down, or block reuptake, so the neurotransmitter stays in the cleft and overstimulates the postsynaptic cell.

Neonicotinoid pesticides illustrate this well. They bind to acetylcholine receptors in insect synapses and activate them, but they are not broken down by acetylcholinesterase. The receptors are held open, the insect's nervous system is continuously overstimulated, and the insect is paralysed and dies. Neonicotinoids bind far more strongly to insect than to vertebrate receptors, which is why they are selectively toxic to insects — though concern about their effect on bees and other pollinators has led to restrictions.

Summation (HL)

Summation is how a neuron combines many small inputs that individually cannot reach threshold.

- **Spatial summation:** several different presynaptic neurons fire at the same time onto one postsynaptic neuron; their small depolarisations add together and may reach threshold.
- **Temporal summation:** a single presynaptic neuron fires rapidly in quick succession, so its depolarisations arrive close together and add up before each has faded.

Integration at synapses (HL)

Because a postsynaptic neuron receives many excitatory and inhibitory inputs at once, it does not simply relay a signal — it **integrates** them. It continuously sums the depolarising (excitatory) and hyperpolarising (inhibitory) effects, and fires an action potential only if the net effect reaches threshold. This integration lets the nervous system weigh competing information and make decisions, and it is the basis of processing in the brain and spinal cord.

> **Exam tip (HL):** Distinguish the two kinds of summation by their cause: **spatial** = many neurons in different *places* firing together; **temporal** = one neuron firing repeatedly over *time*.

Worked example 5 (HL) — summation and integration

A postsynaptic neuron has a threshold at −55 mV and rests at −70 mV. One excitatory input alone depolarises it by 8 mV. Explain, using summation, how the neuron could still be made to fire. (3 marks)

1. One input gives only 8 mV of depolarisation, reaching −62 mV, which is short of the −55 mV threshold, so alone it cannot trigger an action potential.

2. By **spatial summation**, if two or more excitatory neurons fire together their depolarisations add (for example 8 + 8 = 16 mV), reaching threshold and firing the neuron.

3. By **temporal summation**, one neuron firing rapidly can also add successive depolarisations before each fades. Any inhibitory (hyperpolarising) input would subtract from the total, which is how the neuron integrates competing signals.

9. Skills, worked examples and pitfalls

The examples below rehearse the exact skills examiners test: sequencing events, reading graphs and giving cause-and-effect explanations.

Worked example 3 — sequence synaptic transmission

Put the following events of synaptic transmission in the correct order: neurotransmitter binds to receptors; Ca^{2+} enters the synaptic knob; action potential reaches the presynaptic membrane; vesicles fuse and release neurotransmitter; postsynaptic membrane depolarises.

Correct order: (1) action potential reaches the presynaptic membrane → (2) Ca^{2+} enters the synaptic knob → (3) vesicles fuse and release neurotransmitter → (4) neurotransmitter binds to receptors → (5) postsynaptic membrane depolarises.

Remember the trigger for release is always Ca^{2+} influx, and release is by exocytosis of vesicles.

Worked example 4 — all-or-nothing and stimulus strength

A neuron is given a weak stimulus, then a strong one. Explain what happens to the size and frequency of action potentials. (3 marks)

1. Provided each stimulus reaches threshold, every action potential is the **same size** — the all-or-nothing principle; a stronger stimulus does not give a bigger action potential.

2. A stronger stimulus makes the neuron fire action potentials at a **higher frequency**.

3. The brain interprets the higher frequency (and the number of neurons firing) as a stronger stimulus.

Common pitfalls

- **Resting potential:** it is maintained by the sodium−potassium pump **and** the membrane being more permeable to K^+. Name both, not just the pump.
- **All-or-nothing:** a bigger stimulus increases the **frequency** of action potentials, never their size. Do not write that action potentials get “bigger.”
- **One-way travel:** within an axon this is due to the **refractory period**; across a synapse it is due to vesicles being only on the presynaptic side and receptors only on the postsynaptic side. Match the reason to the situation.
- **Ion movements:** in depolarisation Na^+ moves *in*; in repolarisation K^+ moves *out*. Keep the ion and the direction paired correctly.
- **The impulse is not electron flow:** describe it as a moving wave of depolarisation caused by ions crossing the membrane.

10. Quick reference

Key term	One-line summary
Resting potential	About −70 mV inside; set by the Na^+/K^+ pump (3 out, 2 in) and higher K^+ permeability
Threshold	About −55 mV; the potential that must be reached to trigger an action potential
Depolarisation	Voltage-gated Na^+ channels open; Na^+ in; inside reaches about +30 mV

Key term	One-line summary
Repolarisation	Na^+ channels close, voltage-gated K^+ channels open; K^+ out; return toward rest
All-or-nothing	Above threshold every action potential is the same size; stimulus strength is coded by frequency
Refractory period	Brief period after an action potential when the axon cannot refire; makes travel one-way
Saltatory conduction	In myelinated axons the impulse jumps node to node; faster and more energy-efficient
Synapse	Ca^{2+} in → vesicles fuse → neurotransmitter released → binds receptors → postsynaptic potential
Neuromuscular junction	Motor neuron to muscle synapse using acetylcholine; broken down by acetylcholinesterase
Summation (HL)	Spatial = many neurons together; temporal = one neuron firing repeatedly

11. Test yourself

Attempt these without notes; full worked answers follow.

1. State the approximate value of the resting potential and name the two factors that maintain it.

2. Describe the movement of ions during depolarisation and repolarisation of an action potential.

3. Explain what the all-or-nothing principle means and how the nervous system codes the strength of a stimulus.

4. Explain why an action potential travels in one direction along an axon.

5. Explain how myelination increases the speed of nerve conduction.

6. Describe the sequence of events by which an action potential in a presynaptic neuron leads to depolarisation of a postsynaptic neuron.

7. Distinguish between an excitatory and an inhibitory synapse.

8. (HL) Explain how neonicotinoid pesticides disrupt synaptic transmission in insects.

Answers

1. About −70 mV. It is maintained by (i) the sodium−potassium pump, which actively moves 3 Na^+ out for every 2 K^+ in, and (ii) the membrane being more permeable to K^+ than to Na^+, so K^+ diffuses out and leaves the inside negative.

2. During depolarisation voltage-gated Na^+ channels open and Na^+ moves into the axon, making the inside positive (about +30 mV). During repolarisation the Na^+ channels close and voltage-gated K^+ channels open, so K^+ moves out and the inside becomes negative again.

3. All-or-nothing means that once the stimulus reaches threshold an action potential of a fixed size is produced; a stronger stimulus does not produce a larger action potential. Stimulus strength is coded by the **frequency** of action potentials and by the number of neurons firing.

4. The membrane just behind the action potential is in its refractory period, with Na^+ channels temporarily inactivated, so it cannot be re-excited. Local currents can therefore only depolarise the membrane ahead, so the impulse moves forward only.

5. The myelin sheath insulates the axon so action potentials only form at the nodes of Ranvier. Local currents flow from node to node and the impulse jumps between them (saltatory conduction). Fewer regions of membrane have to depolarise, so conduction is faster than continuous conduction in an unmyelinated axon.

6. The action potential depolarises the presynaptic membrane → voltage-gated Ca^{2+} channels open and Ca^{2+} enters → vesicles fuse with the presynaptic membrane and release neurotransmitter by exocytosis → neurotransmitter diffuses across the cleft → it binds to receptors on the postsynaptic membrane, opening ion channels → ions enter and depolarise the postsynaptic membrane, which may reach threshold and fire an action potential.

7. At an excitatory synapse the neurotransmitter opens channels that let positive ions (Na^+) in, depolarising the postsynaptic membrane and making it more likely to reach threshold. At an inhibitory synapse the neurotransmitter opens channels that let Cl^- in or K^+ out, hyperpolarising the membrane and making it less likely to fire.

8. (HL) Neonicotinoids bind to and activate acetylcholine receptors at insect synapses but are not broken down by acetylcholinesterase. The receptors stay activated, so the postsynaptic membrane is continuously depolarised. The insect's nervous system is overstimulated, leading to paralysis and death. They are selectively toxic because they bind insect receptors far more strongly than vertebrate ones.