

Control and coordination

A-Level Biology 9700 · Topic 15 revision notes

Everything from the 5 lessons of this topic on a few pages: the key idea of each lesson, the diagram to remember, the points to learn and the traps examiners set. Read it, cover it, test yourself.

1. Nervous and endocrine systems

2. Resting and action potentials

3. The cholinergic synapse

4. Striated muscle and contraction

5. Control and coordination in plants

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1 Nervous and endocrine systems

Syllabus 15.1

The **endocrine system** is made of **ductless glands** that secrete **hormones** into the blood; only **target cells** with the matching receptor respond. Examples: **ADH** from the posterior pituitary acts on collecting duct cells; **insulin** (beta cells) and **glucagon** (alpha cells) from the islets of Langerhans in the pancreas act on liver and other cells. The **nervous system** sends **electrical impulses** along **neurones** to specific effectors: it is faster, more precise and shorter-lived. A **sensory neurone** carries impulses from a receptor to the CNS; an **intermediate (relay) neurone** connects it to a **motor neurone**, which carries impulses to an effector. **Sensory receptor cells** are transducers: a stimulus changes the permeability of their membrane, producing a **receptor potential**; if this is big enough, an **action potential** starts in the sensory neurone. In a **taste bud**, Na^+ from salt enters a chemoreceptor cell, depolarising it, so Ca^{2+} channels open and neurotransmitter is released onto a sensory neurone.

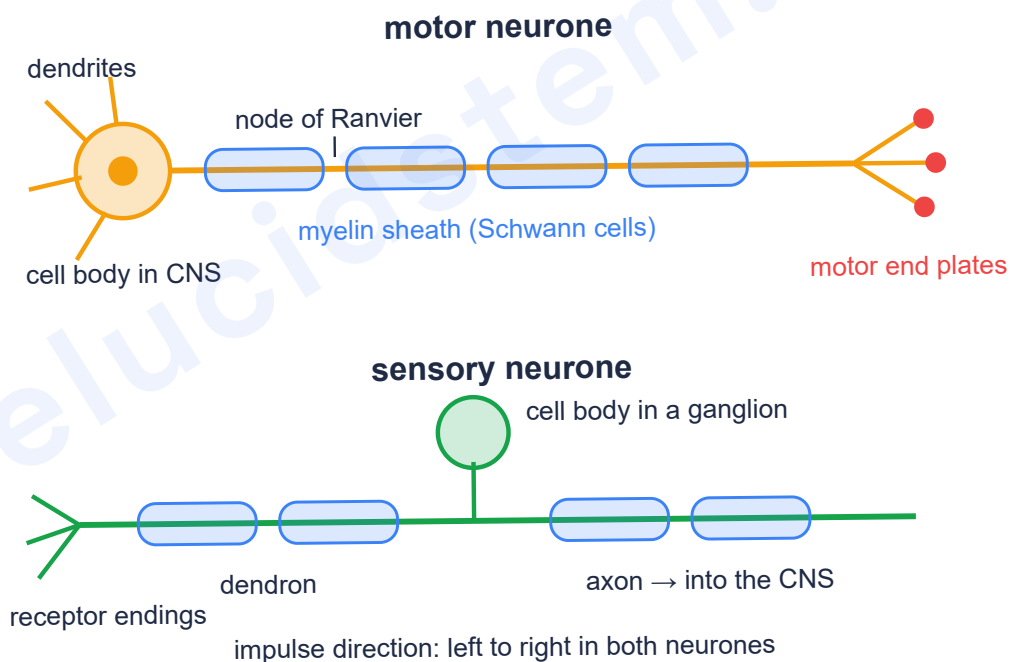


Fig. 1: a motor neurone (cell body in the CNS) and a sensory neurone (cell body in a ganglion outside the CNS)

Gland, neurone, receptor.

- ✓ **Hormones** reach every cell; only target cells with receptors respond.
- ✓ **Motor** cell body in the CNS; **sensory** cell body in a ganglion.
- ✓ **Receptor potential** is graded; **action potential** is all-or-nothing.
- ✓ **Taste:** Na^+ in, depolarise, Ca^{2+} in, transmitter out.

Watch out!

Do not say hormones travel only to their target organ. Hormones are carried in the blood to **every** tissue; only target cells respond, because only they have receptors with a shape complementary to the hormone.

A receptor potential is **graded**: its size depends on the stimulus strength. An action potential is **all-or-nothing**. Only when the receptor potential pushes the sensory neurone past threshold does an action potential start.

2 Resting and action potentials

Syllabus 15.1

At rest the inside of an axon is about -70 mV relative to the outside. The Na^+/K^+ pump uses ATP to move 3 Na^+ out for every 2 K^+ in, and the membrane is more permeable to K^+ , which leaks out; the membrane is relatively impermeable to Na^+ , so the inside stays negative. A stimulus that reaches **threshold** opens **voltage-gated Na^+ channels**: Na^+ floods in (**depolarisation**, to about $+40\text{ mV}$). Then Na^+ channels close and **voltage-gated K^+ channels** open: K^+ diffuses out (**repolarisation**), overshooting slightly (**hyperpolarisation**). During the **refractory period** the Na^+ channels cannot reopen while the resting potential is restored. Local circuits depolarise the next region of membrane. In a **myelinated** neurone this happens only at the **nodes of Ranvier**, so the impulse jumps (**saltatory conduction**), much faster. Because action potentials are all the same size, a stronger stimulus is coded as a **higher frequency**, and the refractory period sets the maximum frequency.

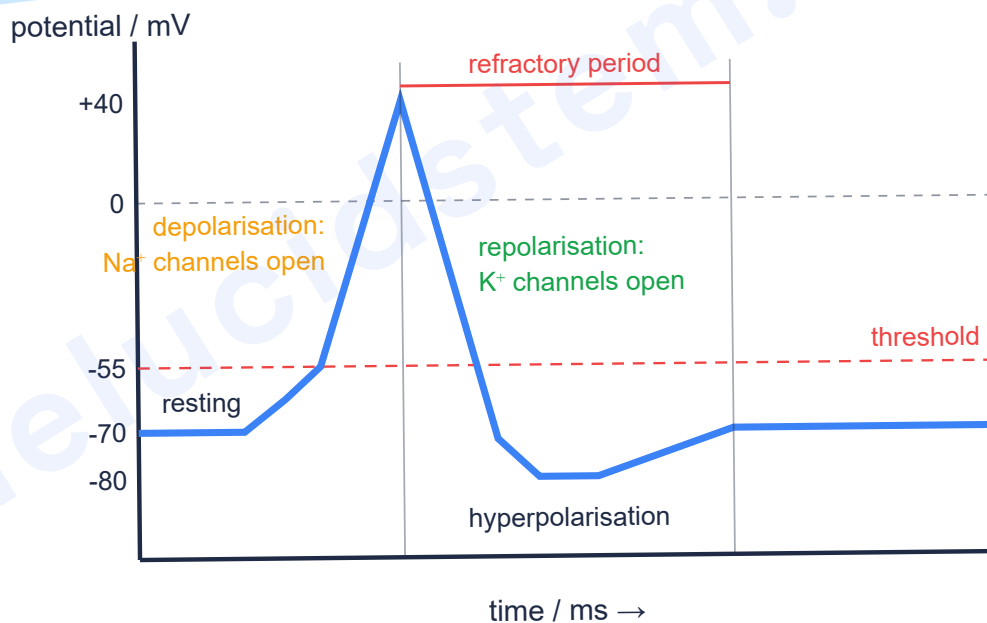


Fig. 1: an action potential recorded at one point on an axon, with the phases and the refractory period

Pump, channel, node.

- ✓ **Rest:** pump 3 Na⁺ out / 2 K⁺ in; K⁺ leaks out.
- ✓ **Up:** Na⁺ channels open; **down:** K⁺ channels open.
- ✓ **Refractory period:** discrete, one-way impulses; caps frequency.
- ✓ **Myelin:** depolarise only at nodes, so the impulse jumps.

Watch out!

During repolarisation K⁺ moves out by **diffusion through channels**, not by the pump. The Na⁺/K⁺ pump keeps the concentration gradients in place, but the fast changes in an action potential are all caused by ions diffusing through voltage-gated channels.

A stronger stimulus does **not** make action potentials bigger or faster. It increases their **frequency** (and may stimulate more neurones). Refer to the refractory period when explaining the upper limit.

3 The cholinergic synapse

Syllabus 15.1

A **cholinergic synapse** uses **acetylcholine (ACh)** as its neurotransmitter. The **presynaptic knob** contains many **mitochondria** and **vesicles** of ACh; it is separated from the **postsynaptic membrane** by a **synaptic cleft** about 20 nm wide. When an action potential arrives, **voltage-gated Ca^{2+} channels** open and Ca^{2+} diffuses into the knob. This makes vesicles move to and fuse with the presynaptic membrane, releasing ACh by **exocytosis**. ACh diffuses across the cleft and binds to **receptor proteins** on the postsynaptic membrane, opening Na^+ channels; Na^+ diffuses in and depolarises the membrane, and if **threshold** is reached an action potential is set up. **Acetylcholinesterase** in the cleft hydrolyses ACh to **choline and ethanoic acid**, which are taken back into the knob and recombined using ATP. Transmission is **one-way** because only the presynaptic side has vesicles and only the postsynaptic side has receptors.

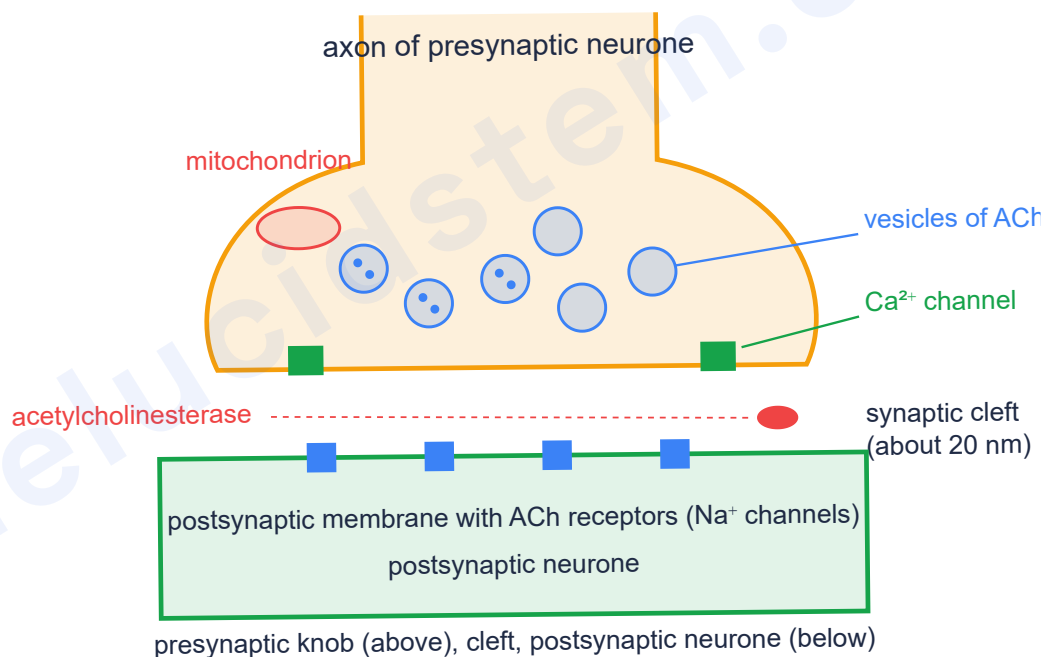


Fig. 1: a cholinergic synapse, with vesicles of acetylcholine in the presynaptic knob and receptors on the postsynaptic membrane

Calcium in, ACh across.

- ✓ **Ca²⁺** enters the knob and triggers exocytosis.
- ✓ **ACh** diffuses across the cleft and binds to receptors.
- ✓ **Na⁺** enters the postsynaptic neurone: threshold?
- ✓ **Acetylcholinesterase** hydrolyses ACh; choline is recycled.

Watch out!

Calcium ions do not cross the cleft and do not bind to the postsynaptic receptors. **Ca²⁺** acts **inside the presynaptic knob**, triggering vesicle fusion. It is ACh that diffuses across the cleft.

Choline is recycled, but it is reabsorbed into the **presynaptic knob**, not the postsynaptic neurone. The postsynaptic neurone does not store or release ACh at this synapse.

4 Striated muscle and contraction

Syllabus 15.1

A striated muscle fibre is a long, multinucleate cell packed with **myofibrils**, each made of repeating **sarcomeres** from one **Z line** to the next. Thin **actin** filaments attach to the Z lines; thick **myosin** filaments are held at the **M line**. The **A band** is the length of the myosin (dark), the **I band** has actin only (light), and the **H zone** has myosin only. At the **neuromuscular junction** ACh depolarises the **sarcolemma**; the action potential spreads down the **T-tubules** and makes the **sarcoplasmic reticulum** release Ca^{2+} . Ca^{2+} binds to **tropoin**, which moves **tropomyosin** off the myosin-binding sites on actin. Myosin heads bind, forming **cross-bridges**, and tilt (the **power stroke**), pulling actin towards the M line. **ATP** binds to detach each head and is hydrolysed to re-cock it. The filaments do not change length: the **I band and H zone shorten**, the **A band stays the same**.

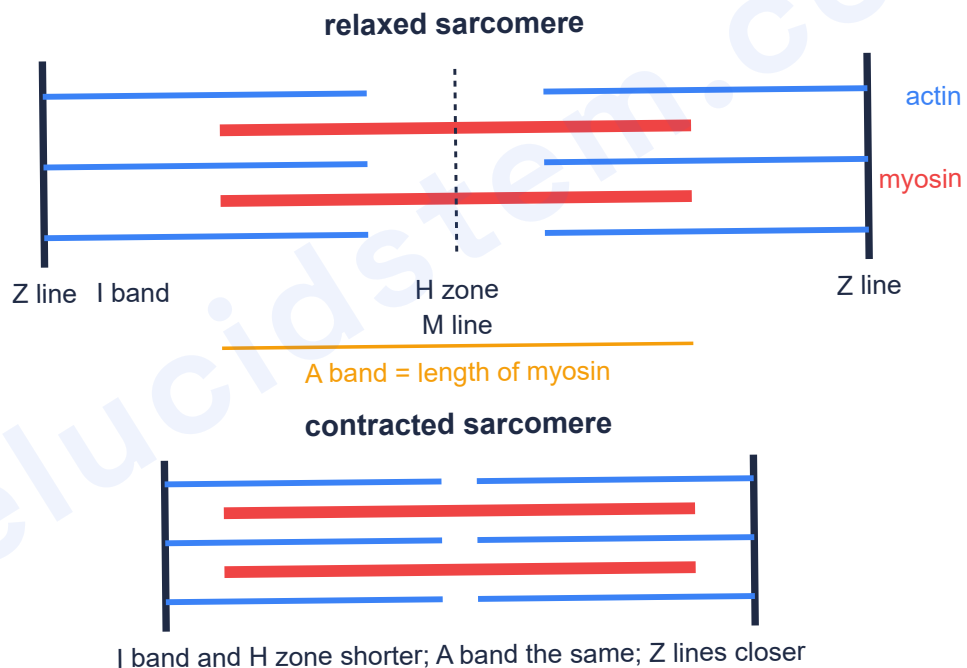


Fig. 1: one sarcomere relaxed and contracted; the actin slides between the myosin so the Z lines move closer

Bands, calcium, ATP.

- ✓ **A band** stays the same; **I band and H zone** shorten.
- ✓ **NMJ** → sarcolemma → T-tubules → SR releases Ca^{2+} .
- ✓ **Ca^{2+} + troponin** moves tropomyosin off the binding sites.
- ✓ **ATP** detaches the head, then is hydrolysed to re-cock it.

Watch out!

The filaments themselves do **not** shorten. Only the overlap changes. If a question asks which band stays the same length, the answer is the **A band**, because it equals the length of the myosin filaments.

ATP is needed to **detach** the myosin head as well as to re-cock it. The power stroke itself happens when ADP and P_i are released. This is why muscles lock in rigor mortis after death: with no ATP, the heads cannot let go of actin.

5 Control and coordination in plants

Syllabus 15.2

The **Venus fly trap** has modified leaves with two lobes, each carrying sensitive **trigger hairs**. Bending a hair produces a **receptor potential**; if two hairs are touched within about 20 seconds, **action potentials** spread across the lobes. These trigger rapid movement of ions and water, changing the turgor of cells in the lobes, so the lobes flip from convex to concave and the trap snaps shut in a fraction of a second. **Auxin**, made in shoot tips, stimulates elongation growth: it binds to a receptor and stimulates **proton pumps** to pump H^+ into the **cell wall**; the **acidic** wall activates **expansins**, which loosen the links between cellulose microfibrils, so water entering by osmosis stretches the wall and the cell **elongates**. In a germinating **barley** grain, the embryo releases **gibberellin**, which travels to the **aleurone layer** and switches on the gene for **amylase**; amylase hydrolyses **starch** in the endosperm to **maltose**, providing sugars for the embryo's respiration and growth.

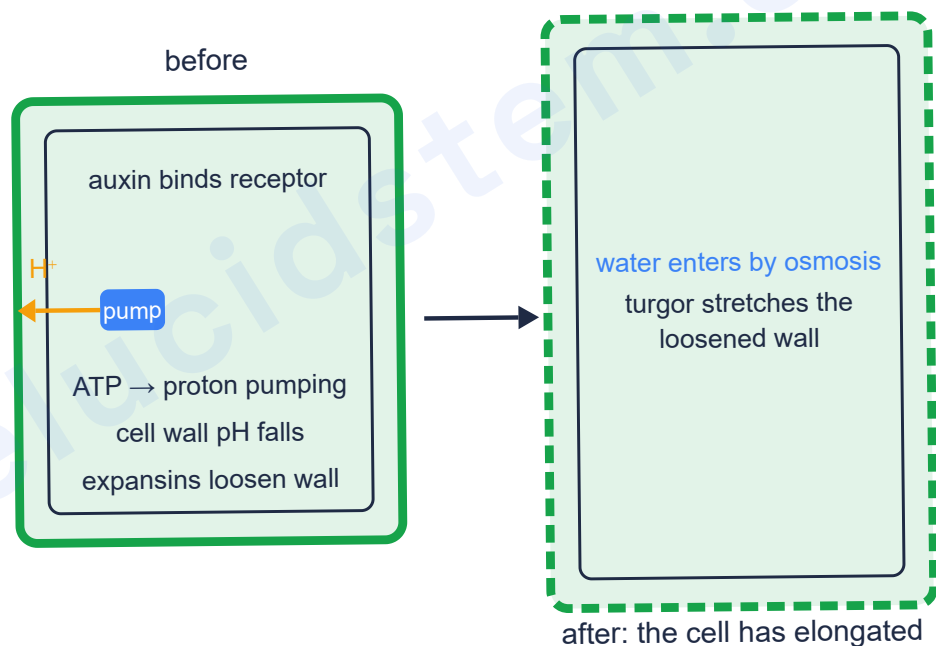


Fig. 1: auxin stimulates proton pumping into the cell wall; the acidified wall loosens and the cell elongates as it takes up water

Snap, stretch, sprout.

- ✓ **Fly trap:** two touches in about 20 s, action potentials, turgor change.
- ✓ **Auxin:** H^+ pumped into the wall, wall loosens, cell elongates.
- ✓ **Gibberellin:** embryo to aleurone layer, amylase gene switched on.
- ✓ **Amylase:** starch to maltose for the embryo's respiration.

Watch out!

The Venus fly trap has no muscles and no nerves. Its rapid movement comes from **action potentials** and changes in **turgor** and cell wall properties, not from contraction.

Auxin does not make the cell divide here, and it is the **cell wall** that becomes acidic, not the cytoplasm. The key steps are **protons pumped into the wall, acidic wall, loosening, then water uptake and elongation.**