Nervous coordination

Neurones, reflex arcs, the action potential and saltatory conduction, synapses, the effects of drugs, the retina and rod cells, habituation, and the nervous system.

The nervous system

The **mammalian nervous system** has the **central nervous system (CNS)** (brain and spinal cord) and the **peripheral nervous system (PNS)** (the nerves linking the CNS to the rest of the body).

Coordination in an animal works through **nervous control** (fast, short-lived, specific, by impulses) and **hormonal control** (slower, longer-lasting, widespread). A **stimulus** is detected by a **receptor**, and the nervous system causes an **effector** (muscle or gland) to respond.

Neurones

Neurone	Structure	Function
Sensory	Long dendron, a cell body outside the CNS (in a ganglion)	Carries impulses **from receptors to the CNS**
Relay (intermediate)	Short, in the CNS	Links sensory and motor neurones
Motor	Cell body in the CNS, a long axon	Carries impulses **from the CNS to effectors**

A **motor neurone** has: a **cell body** (with the nucleus), **dendrites** (receive impulses), a long **axon**, and **Schwann cells** wrapped round the axon, forming a **myelin sheath** (a fatty insulating layer), with gaps called **nodes of Ranvier**.

The reflex arc

A **spinal reflex** is a rapid, automatic, protective response.

stimulus → **receptor** → **sensory neurone** (enters the spinal cord through the **dorsal root**) → **relay neurone** in the **grey matter** → **motor neurone** (leaves through the **ventral root**) → **effector** (muscle) → response.

The spinal cord has **grey matter** (cell bodies and synapses, in the centre, in an H shape) and **white matter** (myelinated axons, on the outside). Reflexes are fast because there are only **few synapses** and the brain is not involved, and so they protect the body from harm.

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The action potential

Resting potential

At rest, the inside of an axon is **negative** (about −70 mV) relative to the outside. This is maintained by the **sodium–potassium pump**, which uses ATP to move **3 Na^+ out** and **2 K^+ in**, and by the greater permeability of the membrane to K^+ (through open potassium channels) than to Na^+, so K^+ leaks out.

Stages of the action potential

1. A **stimulus** causes some voltage-gated **sodium channels** to open, and Na^+ ions diffuse in, **depolarising** the membrane.
2. If the **threshold** (about −55 mV) is reached, many more sodium channels open (**positive feedback**) and the inside becomes **positive** (about +40 mV): the **action potential**.
3. The sodium channels **close** and **voltage-gated potassium channels open**. K^+ diffuses out, **repolarising** the membrane.
4. There is a slight **hyperpolarisation** (more negative than resting) before the resting potential is restored by the sodium–potassium pump.

The refractory period

After an action potential, the sodium channels cannot open again for a short time. This means that:

- impulses are **discrete** (separated) and cannot overlap,
- impulses travel in **one direction** only,
- the **frequency** of impulses is limited.

The **all-or-nothing law**: an action potential is either fired or not. The **size** is always the same, and a stronger stimulus gives a **higher frequency** of impulses.

Conduction along the axon

A local circuit of current spreads to the next region, which depolarises to threshold, and the action potential is **propagated**.

Saltatory conduction

In **myelinated** neurones, the myelin **insulates** the axon, so ions can only cross the membrane at the **nodes of Ranvier**. The action potential **jumps from node to node** (saltatory conduction), which makes

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conduction **faster** (up to 100 m s^{-1}, against 1 m s^{-1} in unmyelinated) and uses less energy.

The speed also increases with a **greater axon diameter** (less resistance) and a **higher temperature**.

Synapses

A **synapse** is the junction between two neurones. A **cholinergic synapse** uses **acetylcholine** as the neurotransmitter.

1. An action potential reaches the **presynaptic knob**, which opens **voltage-gated calcium channels**, so Ca^{2+} ions **enter**.
2. Ca^{2+} causes **vesicles** containing **acetylcholine** to fuse with the presynaptic membrane and release it by **exocytosis** into the **synaptic cleft**.
3. Acetylcholine **diffuses** across the cleft and binds to **receptors** on the postsynaptic membrane, which opens **sodium channels**.
4. Na^{+} enters, causing **depolarisation**. If the threshold is reached, a new action potential is generated.
5. **Acetylcholinesterase** breaks down acetylcholine into choline and ethanoate, so the response stops. The choline is taken up and re-synthesised using energy from the mitochondria.

Roles of synapses: they make sure impulses pass in **one direction only**, allow **many neurones to converge** (summation), allow **integration**, and filter out low-level stimuli. A **neuromuscular junction** is a synapse between a motor neurone and a muscle.

The pupil reflex

The pupil **constricts** in bright light, to protect the retina (circular muscles of the iris contract, radial muscles relax) and **dilates** in dim light (radial muscles contract, circular relax). The two muscle sets are controlled by the autonomic nervous system.

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Drugs and synapses

Drug	Effect on the synapse
Nicotine	Mimics acetylcholine and **binds to its receptors**, causing a response. Stimulates dopamine release
Lidocaine	A local anaesthetic: **blocks voltage-gated sodium channels** in sensory neurones, so no action potential passes
Cobra venom α-toxin	**Binds to acetylcholine receptors** and blocks them, so muscles cannot contract (paralysis)
L-DOPA	Converted to **dopamine** in the brain, to treat **Parkinson's disease** (low dopamine)
MDMA (ecstasy)	Causes the release of **serotonin** and blocks its re-uptake, leading to feelings of well-being and then to depression and damage

Detecting light: rod cells

The **retina** has **rods** and cones. **Rod cells** work in dim light and are very sensitive, with a pigment called **rhodopsin**, which is made of **opsin** and **retinal**.

1. In the **dark**, **sodium (cation) channels** in the rod's membrane are **open**, so Na^+ flows in, and the cell is **depolarised** (about −40 mV), releasing an **inhibitory** neurotransmitter onto the bipolar neurone.
2. In **light**, the retinal changes shape, which makes **rhodopsin split** into opsin and retinal (**bleaching**). This causes **sodium channels to close**.
3. So Na^+ no longer enters. The rod is **hyperpolarised** (more negative), and releases **less inhibitory neurotransmitter**.
4. The bipolar neurone is **no longer inhibited**, so it **depolarises** and the signal passes to the **ganglion cells**, which generate **action potentials** in the optic nerve.

> **NOTE**
>
> The surprising point: **light closes** the channels. The signal is generated by **reduced** release of inhibitory transmitter.

Habituation

Habituation is the **decrease in response** to a repeated, harmless stimulus. For example, ignoring a clock ticking. At the synapse, there is a **reduction of calcium channels opening**, so less neurotransmitter is released. It allows the animal to **ignore** irrelevant stimuli and **save energy**, while remaining sensitive to new ones.

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