Cell Biology

Unit 6 · Lesson 1 · Nerve and muscle cells

The notes for Lesson 1 of Unit 6: chapters 6.1 to 6.2 of the unit book, with their figures, worked examples, exam traps and quizzes, in one file. The full unit book, with every chapter, the exam question bank and the glossary, is linked at the end.

Lesson 1 of 4 | 2 chapters | Chapters 6.1 to 6.2

About these notes

These are the notes for **Lesson 1 · Nerve and muscle cells** of the University free n easy video lessons on Cell Biology Unit 6, Specialized Cells and Cell Study Methods. They are chapters 6.1 to 6.2 of the unit book, unchanged, so a page here is the same page in the full book.

6.1 Nerve cells: structure and function

A nerve cell, or neuron, is a cell built to carry a message fast and far: from your toe to your spinal cord in a few hundredths of a second. It sends an electrical signal along a long, thin axon and then a chemical signal across a small gap, the synapse. Other universities meet neurons in physiology courses.

You need: ion channels and the Na^+/K^+ pump (Unit 2, Ion channels: ligand-gated and voltage-gated; Active transport) and exocytosis (Unit 2, Endocytosis and exocytosis).

Specialised cells: one genome, many jobs

Every cell in your body carries the same DNA, yet a neuron, a muscle fibre and a pancreatic β cell work nothing alike. Specialisation (differentiation) comes from which genes are switched on: each cell type makes its own proteins and builds its shape around one job. Chapters 6.1 to 6.3 look at four such cells.

Parts of a neuron

- **Dendrites**: short branches that receive signals from other cells.

- **Cell body (soma)**: holds the nucleus and most ribosomes; its clumps of rough ER are called Nissl bodies.
- **Axon hillock and initial segment**: where the axon leaves the soma. Voltage-gated Na^+ channels are densest here, so the action potential starts here.
- **Axon**: one long process, up to about a metre in human leg nerves, that carries the signal away from the soma.
- **Myelin sheath**: layers of membrane wrapped round the axon by Schwann cells (peripheral nerves) or oligodendrocytes (brain and spinal cord). The bare gaps between wraps are the **nodes of Ranvier**.
- **Axon terminals**: end in synapses, where vesicles of neurotransmitter wait.

By shape, neurons are multipolar (many dendrites, one axon; motor neurons), bipolar (one dendrite, one axon; retina) or pseudounipolar (one process that splits in two; sensory neurons).

Resting potential and the action potential

At rest the inside of a neuron is negative, about −70 mV. The Na^+/K^+-ATPase pumps 3 Na^+ out and 2 K^+ in per ATP, so K^+ is high inside and Na^+ high outside. The resting membrane is leaky mainly to K^+, so K^+ flows out until the negative charge left behind holds it back. The voltage at which one ion is in balance is its equilibrium potential, given by the Nernst equation:

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E = (RT / zF) · ln(C_out / C_in)
At 37 °C and z = +1:  E = 61.5 mV × log10(C_out / C_in)
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Here R is the gas constant (8.314 J mol^{-1} K^{-1}), T the temperature in kelvin, z the ion's charge, F the Faraday constant (96,485 C mol^{-1}) and C the ion concentration outside and inside.

1. A stimulus depolarises the membrane to threshold, about −55 mV.
2. Voltage-gated Na^+ channels open, Na^+ rushes in, and the inside swings to about +30 mV.
3. Within about a millisecond the Na^+ channels inactivate and voltage-gated K^+ channels open. K^+ leaves and the membrane repolarises.
4. The K^+ channels close slowly, so the voltage briefly undershoots below rest (after-hyperpolarisation) before leak channels and the pump restore −70 mV.

While the Na^+ channels are inactivated (the refractory period) they cannot reopen, so the spike moves only forward. The action potential is all-or-none: a stronger stimulus gives more spikes per second, not a taller spike. In an unmyelinated axon the spike creeps along at about 1 m/s. In a myelinated axon it jumps from node to node (saltatory conduction), up to about 100 m/s, and only the nodes exchange ions, which saves ATP.

The synapse

At a chemical synapse the spike opens voltage-gated Ca^{2+} channels in the terminal. Ca^{2+} entry makes vesicles fuse with the membrane, and transmitter (acetylcholine, glutamate, GABA) crosses a cleft of about 20 nm. It binds ligand-gated channels on the next cell, which either excite it (cations in) or inhibit it (Cl^- in). The transmitter is then removed: acetylcholinesterase splits acetylcholine, and other transmitters are pumped back. Electrical synapses use gap junctions (Unit 5, Cell junctions and cell adhesion) and pass current directly.

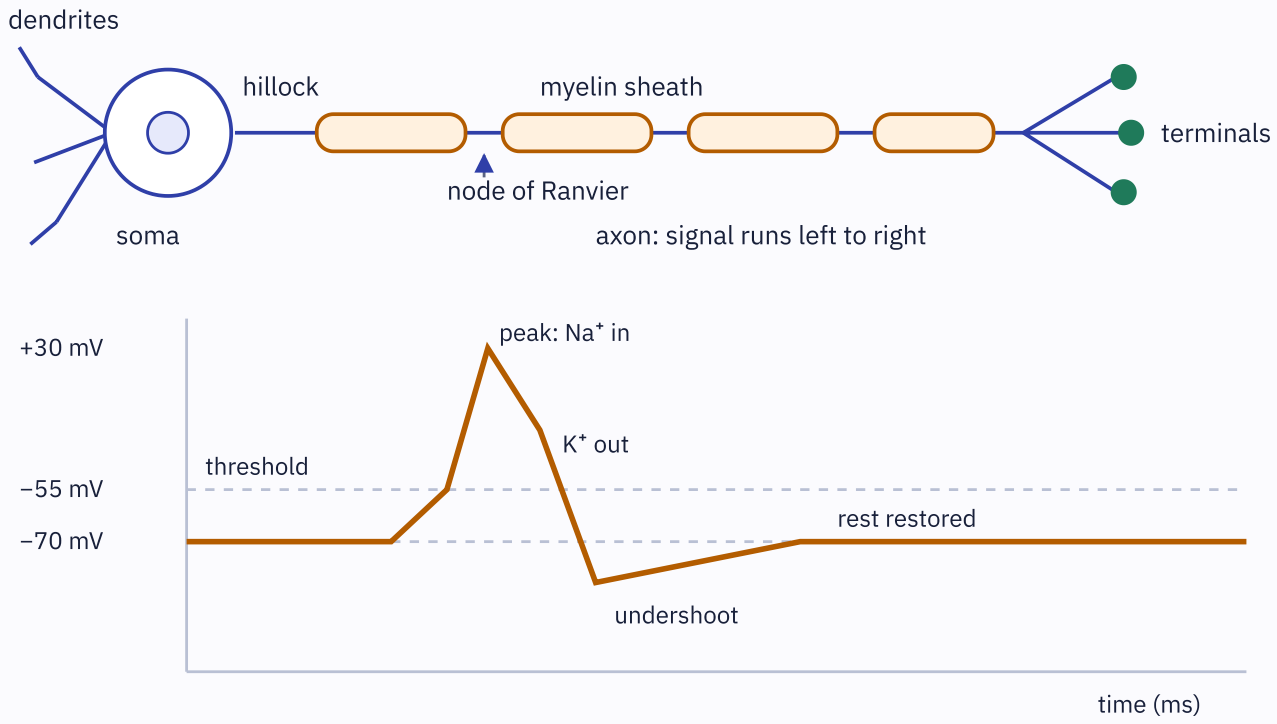


Figure 6.1.1 Top: the parts of a myelinated neuron; the spike starts at the hillock and jumps between the nodes. Bottom: one action potential, about 2 ms long. Notice the threshold line: below it nothing happens, above it the full spike always fires.

WORKED EXAMPLE

Question (6 marks). A mammalian neuron has K^+ = 140 mM inside and 5 mM outside, Na^+ = 15 mM inside and 145 mM outside, at 37 °C. Find E_K and E_{Na}. Explain why the resting potential (−70 mV) lies close to E_K and the spike peak moves towards E_{Na}.

1. $E_K = 61.5 \times \log_{10}(5/140) = 61.5 \times \log_{10}(0.0357) = 61.5 \times (-1.447) =$ **−89 mV**.
2. $E_{Na} = 61.5 \times \log_{10}(145/15) = 61.5 \times \log_{10}(9.67) = 61.5 \times 0.985 =$ **+61 mV**.
3. At rest the membrane is far more permeable to K^+ than to Na^+, so the voltage sits near E_K. A small Na^+ leak pulls it up from −89 to about −70 mV.
4. When voltage-gated Na^+ channels open, Na^+ permeability briefly dominates and the voltage heads towards E_{Na}. It peaks below +61 mV (about +30 mV) because Na^+ channels inactivate and K^+ channels open first.

EXAM TRAPS

- The pump moves 3 Na^+ out and 2 K^+ in, not the reverse, and not 1:1.
- Schwann cells myelinate peripheral axons; oligodendrocytes myelinate in the brain and spinal cord. Examiners swap them.
- A stronger stimulus does not make a bigger spike; it makes more spikes per second.
- Repolarisation is K^+ leaving the cell, not Na^+ being pumped out. The pump is far too slow to shape one spike.

CHECK YOURSELF

– REMEMBER **Name the cells that form myelin in the PNS and in the CNS.** 2 marks

Schwann cells in the peripheral nervous system; oligodendrocytes in the central nervous system.

– UNDERSTAND **Why does an action potential travel only away from the soma?** 3 marks

Behind the spike the Na^+ channels are inactivated (refractory) and cannot reopen, so only the membrane ahead can be pushed to threshold.

– UNDERSTAND **Explain the role of Ca^{2+} at a chemical synapse.** 3 marks

The spike opens voltage-gated Ca^{2+} channels in the terminal; Ca^{2+} entry triggers synaptic vesicles to fuse with the membrane and release transmitter by exocytosis.

– APPLY **Outside K^+ rises from 5 to 10 mM (inside K 140 mM). Find the new E and say what happens to the resting potential.** 4 marks

$E_K = 61.5 \times \log_{10}(10/140) = 61.5 \times (-1.146) =$ **−70 mV**. E_K becomes less negative, so the cell depolarises, sits nearer threshold and becomes more excitable; this is why high blood K^+ disturbs heart rhythm.

– ANALYZE **Myelin raises conduction speed and saves energy. Explain both from the same mechanism.** 5 marks

Myelin insulates the internode, so current spreads quickly inside the axon to the next node instead of leaking out. The spike is regenerated only at nodes: it jumps (faster), and ions cross the membrane only at nodes, so the pump has far fewer ions to move back (less ATP).

BEYOND THE SYLLABUS

Not for the exam. Optogenetics puts light-gated channels into chosen neurons so a researcher can switch them on with a flash of blue light. Brain-computer interfaces read spikes from electrode arrays to move a cursor or a robotic arm. Search next: "patch clamp" and "optogenetics channelrhodopsin".

6.2 Muscle cells: types and function

A muscle cell turns chemical energy (ATP) into pulling force. Myosin motors walk along actin filaments, and because thousands of them pull at once, the whole cell shortens. Your body has three kinds of muscle cell, skeletal, cardiac and smooth, built on the same actin–myosin engine but switched on in different ways.

You need: actin filaments and myosin motors (Unit 3, Cytoskeleton; Molecular motors and centrioles) and the action potential (Chapter 6.1).

Three types of muscle cell

FEATURE	SKELETAL	CARDIAC	SMOOTH
Shape	Long cylindrical fibre, many nuclei at the edge	Short, branched, usually one central nucleus	Spindle-shaped, one central nucleus

FEATURE	SKELETAL	CARDIAC	SMOOTH
Striations	Yes (sarcomeres)	Yes (sarcomeres)	No
Control	Voluntary, by motor neurons	Involuntary, own pacemaker	Involuntary, nerves and hormones
Cell links	Each fibre separate	Intercalated discs with gap junctions	Gap junctions in many tissues
Ca^{2+} switch	Troponin on thin filament	Troponin on thin filament	Calmodulin and myosin light-chain kinase
Where	Limbs, trunk, face	Heart wall	Gut, blood vessels, uterus, bladder

A skeletal muscle fibre is a single huge cell formed when many myoblasts fuse during development; that is why it has many nuclei. Inside, it is packed with myofibrils, each a chain of repeating units called sarcomeres.

The sarcomere and the sliding filament model

A sarcomere runs from one Z line (Z disc) to the next. Thin filaments of actin are anchored at the Z lines and point inwards; thick filaments of myosin sit in the middle, held at the M line. Under the microscope this gives bands:

- **A band**: the length of the thick filaments (dark, anisotropic). It does not change during contraction.
- **I band**: the part with thin filaments only (light, isotropic). It shortens.
- **H zone**: the middle of the A band, with thick filaments only. It shortens and can vanish.

The sliding filament model (A. F. Huxley and R. Niedergerke; H. E. Huxley and J. Hanson, both 1954) says the filaments do not shorten; they slide past each other, pulling the Z lines closer.

Cross-bridge cycle and excitation–contraction coupling

1. A motor neuron releases acetylcholine at the neuromuscular junction; the fibre fires an action potential.
2. The spike runs down T-tubules (inward folds of the plasma membrane). A voltage sensor there (the dihydropyridine receptor) opens ryanodine receptors in the sarcoplasmic reticulum (SR), and Ca^{2+} floods the cytosol.
3. Ca^{2+} binds troponin C; troponin shifts tropomyosin off the myosin-binding sites on actin.

4. A myosin head carrying ADP + Pi binds actin. Releasing Pi triggers the power stroke, pulling the thin filament towards the M line; ADP leaves.
5. A fresh ATP binds and the head lets go of actin. Hydrolysis of that ATP re-cocks the head for the next cycle.
6. When nerve firing stops, the SR Ca^{2+} pump (SERCA) takes Ca^{2+} back, tropomyosin covers the sites and the muscle relaxes.

ATP is needed both to detach myosin and to pump Ca^{2+} back. After death ATP runs out, heads stay locked on actin, and the muscles go stiff: rigor mortis. In smooth muscle, Ca^{2+} binds calmodulin, which activates myosin light-chain kinase; the phosphorylated myosin then binds actin. Cardiac cells add a Ca^{2+} entry step from outside and pass the spike to neighbours through gap junctions, so the heart contracts as one unit.

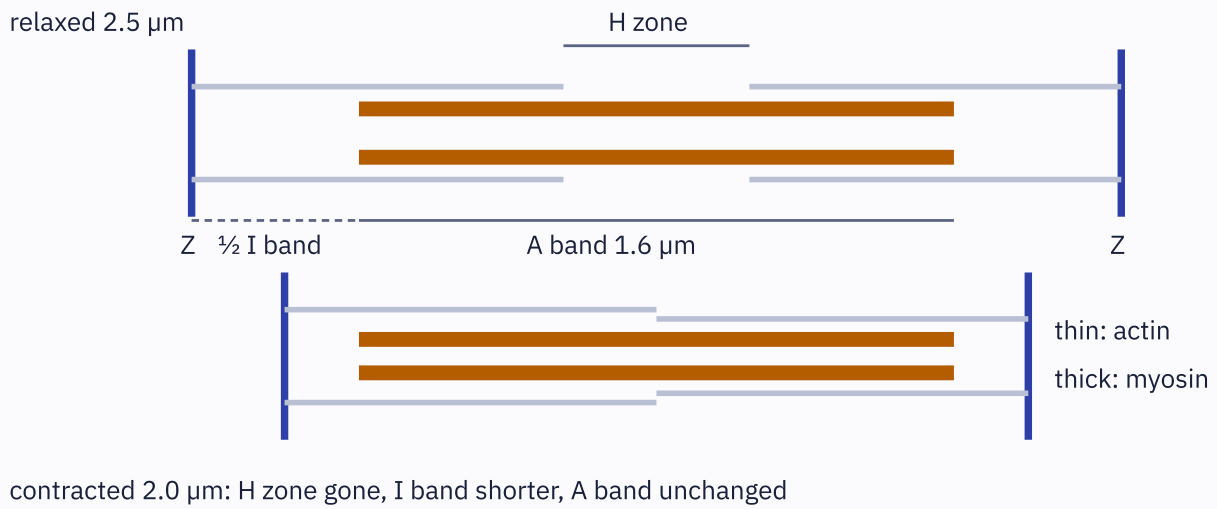


Figure 6.2.1 One sarcomere before and after contraction, drawn to scale with the lengths of the worked example (200 units = 1 µm). The thick filaments (orange) keep their length, so the A band is the same; the thin filaments slide inwards, so the I band and H zone shrink and the Z lines move closer.

WORKED EXAMPLE

Question (5 marks). In a frog muscle, thick filaments are 1.6 µm long and each thin filament (from a Z line) is 1.0 µm long. A sarcomere shortens from 2.5 µm to 2.0 µm. Find the A band, I band and H zone before and after.

1. A band = thick filament length = **1.6 µm** both times.
2. I band = sarcomere − A band. Before: 2.5 − 1.6 = **0.9 µm**. After: 2.0 − 1.6 = **0.4 µm**.
3. H zone = sarcomere − 2 × thin filament length. Before: 2.5 − 2.0 = **0.5 µm**. After: 2.0 − 2.0 = **0 µm**; the thin filaments now meet at the centre.
4. Check: the I band lost 0.5 µm and the H zone lost 0.5 µm, equal to the 0.5 µm shortening, as sliding predicts.

EXAM TRAPS

- The A band never changes length. Only the I band and H zone shorten.
- ATP binding makes myosin let go of actin; the power stroke follows Pi release. Rigor is the state with no ATP.
- Smooth muscle has actin and myosin but no troponin and no sarcomeres; Ca^{2+} works through calmodulin.
- Cardiac muscle is striated but involuntary; do not pair "striated" with "voluntary".

CHECK YOURSELF

– REMEMBER **Name the protein that Ca^{2+} binds in skeletal muscle and in smooth muscle.** 2 marks

Troponin C in skeletal (and cardiac) muscle; calmodulin in smooth muscle.

– UNDERSTAND **Why are skeletal muscle fibres multinucleate?** 2 marks

Each fibre forms by the fusion of many myoblasts during development, and their nuclei are kept.

– UNDERSTAND **Explain rigor mortis using the cross-bridge cycle.** 3 marks

After death ATP runs out. Myosin heads that have done their power stroke cannot detach without ATP, so they stay bound to actin and the muscle stays stiff until proteins break down.

– APPLY **With the filament lengths of the worked example, what is the I band at a sarcomere length of 3.0 µm?** 2 marks

I band = 3.0 − 1.6 = **1.4 µm**.

– ANALYZE **Why must heart cells be electrically coupled while skeletal fibres are not?** 4 marks

The heart must pump as one unit, so the spike from the pacemaker spreads cell to cell through gap junctions in the intercalated discs. Skeletal muscle needs graded force, so each fibre is driven by its own motor neuron and the brain chooses how many motor units to fire.

BEYOND THE SYLLABUS

Not for the exam. Duchenne muscular dystrophy comes from loss of dystrophin, a protein linking the actin cytoskeleton to the membrane, and gene therapies for it are now in clinical use. Cultured meat grows skeletal muscle cells on scaffolds in bioreactors, a growing biotech industry. Search next: "dystrophin gene therapy" and "cultivated meat bioreactor".

Key terms of this lesson

TERM	MEANING OR FORMULA	CHAPTER
Nernst equation	$E = 61.5\ \text{mV} \times \log_{10}(C_{out} / C_{in})$ for a +1 ion at 37 °C	6.1
Action potential	All-or-none spike: Na^+ in (to about +30 mV), then K^+ out; threshold about −55 mV	6.1
Saltatory conduction	Spike jumps between nodes of Ranvier in myelinated axons	6.1
Sarcomere bands	A band = thick filament (constant); I band = sarcomere − A band; H zone = sarcomere − 2 × thin filament	6.2
Cross-bridge cycle	ATP binding detaches myosin; Pi release drives the power stroke	6.2

Glossary (terms used in this lesson)

Action potential	A brief, all-or-none reversal of membrane voltage that travels along an axon or muscle fibre.
Axon hillock	Where the axon leaves the cell body; the action potential starts there.
Cross-bridge	A myosin head bound to actin during contraction.
Myelin	Wrapped membrane layers around an axon that insulate it.
Rhodopsin	The rod visual pigment: opsin plus 11-cis-retinal.
Sarcomere	The contractile unit of striated muscle, from one Z line to the next.

The full unit book

Every chapter of Unit 6, the unit summary, the exam question bank with model answers and the full glossary: Cell Biology · Unit 6 · Specialized Cells and Cell Study Methods (one file, works offline).

Sources and further reading

- Alberts B. et al., *Molecular Biology of the Cell*, 7th edition (2022), W. W. Norton: chapters on visualising cells, analysing cells and molecules, and the cytoskeleton (from memory; check).
- Lodish H. et al., *Molecular Cell Biology*, W. H. Freeman: chapters on culturing, visualising and fractionating cells, and on nerve cells (from memory; check).
- Karp G., *Cell and Molecular Biology: Concepts and Experiments*, Wiley: chapter on techniques in cell and molecular biology (from memory; check).
- Cooper G. M., *The Cell: A Molecular Approach*, Sinauer/Oxford University Press: chapter on tools of cell biology (from memory; check).
- Hall J. E., *Guyton and Hall Textbook of Medical Physiology*, Elsevier: chapters on membrane potentials, skeletal muscle contraction, the retina and insulin (from memory; check).
- Wikipedia, "Cell fractionation": en.wikipedia.org/wiki/Cell_fractionation (overview of homogenisation and isotonic, cold buffers).
- Abcam, "Cell fractionation: a complete overview": abcam.com knowledge center (differential and density-gradient centrifugation, purity checks by Western blot).

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