MEGA LECTURE

IB DP BIOLOGY

Theme B: Form and Function

B3.3 Muscle and Motility

Revision Notes · Higher Level only

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What the syllabus requires

B3.3 Muscle and Motility is an **HL-only** sub-topic. Everything in this booklet is examinable at Higher Level and only at Higher Level. Use the checklist below as a final revision sweep.

Understanding	You should be able to...
Movement in animals	Explain how muscles, the skeleton and joints act together to produce movement, and why muscles work in antagonistic pairs.
Skeletal muscle structure	Describe the hierarchy muscle → fibre → myofibril → sarcomere and label the banding pattern.
Myofilaments	Identify thick (myosin) and thin (actin, tropomyosin, troponin) filaments and their roles.
Sliding filament model	Explain sarcomere shortening in terms of filaments sliding, and predict changes to the I band, H zone and A band.
Cross-bridge cycle	Sequence attachment, power stroke, detachment and re-cocking, and state the roles of ATP.
Role of calcium	Trace the pathway from action potential to Ca^{2+} release, troponin binding and exposure of binding sites.
Motor units and fibre types	Explain recruitment, summation and the difference between fast and slow twitch fibres.
Other motility	Outline microtubule sliding in cilia and flagella and amoeboid movement.

> **Exam note:** This is a data-rich topic. Expect labelled electron-micrograph diagrams of a sarcomere, sequencing questions on the cross-bridge cycle, and extended-response questions linking Ca^{2+}, ATP and the sliding filament model.

1. Movement in animals: muscles, bones and joints

Animals move by converting the chemical energy of ATP into mechanical work. Three components act together as a lever system: **muscles** supply the force, **bones** act as rigid levers, and **joints** act as the pivots about which the bones turn. Muscle can only actively generate force by *shortening* (contracting) and pulling; it cannot actively push or lengthen itself.

Antagonistic muscle pairs

Because a muscle can only pull, a single muscle can move a bone in one direction only. To return the bone, a second muscle that pulls in the opposite direction is needed. Two muscles that produce opposite movements at a joint form an **antagonistic pair**: as one (the agonist) contracts, the other (the antagonist) relaxes.

- At the elbow, the **biceps** (a flexor) contracts to bend the arm while the triceps relaxes.
- To straighten the arm, the **triceps** (an extensor) contracts while the biceps relaxes.
- The biceps and triceps attach to bones by tendons; the two bones are joined at the elbow by ligaments.

Synovial joint structure

Freely movable joints such as the elbow, knee and hip are **synovial joints**. Their structure reduces friction and allows a wide range of movement while remaining stable.

Structure	Function
Cartilage (articular)	Smooth layer covering the ends of the bones; reduces friction and absorbs shock/compression.
Synovial fluid	Lubricates the joint and reduces friction between the cartilage surfaces; also supplies nutrients to the cartilage.
Synovial membrane	Lines the joint capsule and secretes the synovial fluid.
Joint capsule	Tough fibrous sleeve that encloses and seals the joint, holding the synovial fluid in.
Ligaments	Connect bone to bone across the joint, providing stability and limiting the range of movement to prevent dislocation.
Tendons	Connect muscle to bone so that muscle contraction is transmitted to the bone as movement.

> **Key distinction: Tendons** join muscle to bone; **ligaments** join bone to bone. Mixing these up is one of the most common errors in this topic.

2. Structure of skeletal (striated) muscle

Skeletal muscle is also called **striated** muscle because under the microscope it shows regular light and dark cross-bands (striations). It is organised as a set of structures nested inside one another, each level smaller than the last.

Level	Description
Muscle	A whole organ (e.g. the biceps), made of many bundles of parallel muscle fibres.
Muscle fibre (muscle cell)	A single, very long, **multinucleate** cell formed by the fusion of many cells during development; contains many myofibrils. Surrounded by the sarcolemma (cell membrane); the cytoplasm is the sarcoplasm.
Myofibril	A cylindrical bundle of myofilaments running the length of the fibre; the contractile machinery.
Sarcomere	The repeating functional (contractile) unit of a myofibril, running from one Z line to the next.

Why are muscle fibres multinucleate?

Each fibre is formed by the fusion of many precursor cells, so it retains many nuclei. A single fibre is far too large and too metabolically demanding to be serviced by one nucleus, so multiple nuclei allow rapid synthesis of the large amounts of contractile protein required.

The banding pattern of the sarcomere

The striations arise from the ordered, overlapping arrangement of thick and thin filaments within each sarcomere. Learn each band precisely.

Feature	What it is
Z line (Z disc)	The boundary at each end of a sarcomere; thin filaments are anchored here. One sarcomere = Z line to Z line.
I band	A **light** band containing only thin (actin) filaments, no overlap with thick filaments. Straddles a Z line, so it is shared between two adjacent sarcomeres.
A band	A **dark** band, the full length of the thick (myosin) filaments; includes the region where thick and thin filaments overlap.
H zone	A lighter region in the middle of the A band containing only thick filaments (no thin-filament overlap).
M line	A line down the centre of the H zone that anchors the thick filaments and holds them in register.

> **Memory aid:** The l**I**ght band is the **I** band (actin only); the d**A**rk band is the **A** band (myosin, including overlap). The **H**ole in the middle is the **H** zone (myosin only).

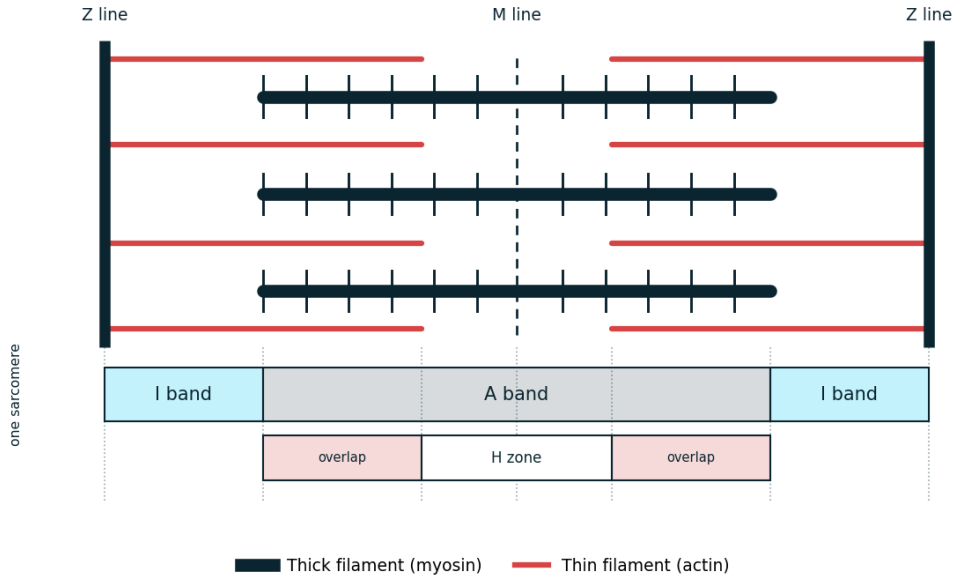

Figure 1. Structure of a sarcomere (Z line to Z line). Thick (myosin) filaments make up the dark A band; thin (actin) filaments make up the light I band; the myosin-only centre is the H zone (M line at its middle). Drawn to scale for a relaxed sarcomere of 2.6 µm: A band 1.6 µm, I band 1.0 µm, H zone 0.6 µm.

3. The myofilaments

Each sarcomere contains two kinds of protein filament arranged in a highly ordered, overlapping lattice.

Filament	Composition and features
Thick filament	Made of many **myosin** molecules. Each myosin has a long tail and a globular **head** that projects outward. The heads form the cross-bridges: they can bind actin, hydrolyse ATP and swivel.

Filament	Composition and features
Thin filament	A double helix of **actin** monomers, each of which has a myosin-binding site. Wound along the actin is the protein **tropomyosin**, which blocks the binding sites at rest. Attached to tropomyosin at intervals is **troponin**, a protein that binds Ca^{2+}.

Roles of the four proteins

- **Myosin** → thick filament; its heads generate force in the power stroke and act as ATPase enzymes.
- **Actin** → thin filament; provides the binding sites that the myosin heads pull on.
- **Tropomyosin** → blocks the myosin-binding sites on actin when the muscle is relaxed.
- **Troponin** → binds Ca^{2+}; when it does, it pulls tropomyosin aside to expose the binding sites.

4. The sliding filament model

The sliding filament model explains how a sarcomere shortens. The filaments themselves do **not** change length. Instead, the thin (actin) filaments are pulled inward, sliding *past* the thick (myosin) filaments toward the centre of the sarcomere. Because thin filaments at both ends slide inward, the two Z lines are drawn closer together and the whole sarcomere shortens.

What changes and what stays the same during contraction

Feature	During contraction	Why
Sarcomere length	Shortens	Z lines pulled closer together.
I band	Shortens	Overlap increases, so the actin-only region shrinks.
H zone	Shortens	Thin filaments move inward into the H zone, reducing the myosin-only region.
A band	Stays the SAME	Its length equals the length of the thick filaments, which do not change.
Zone of overlap	Increases	Actin and myosin overlap more as filaments slide together.

> **Examiner favourite:** If asked what happens to the A band during contraction, the answer is: **it stays constant**. The A band length equals the thick-filament length, and filaments slide rather than shorten. Many students wrongly say all bands shorten.

When a muscle is stretched, the same logic runs in reverse: the I band and H zone widen, the overlap decreases, but the A band still stays constant.

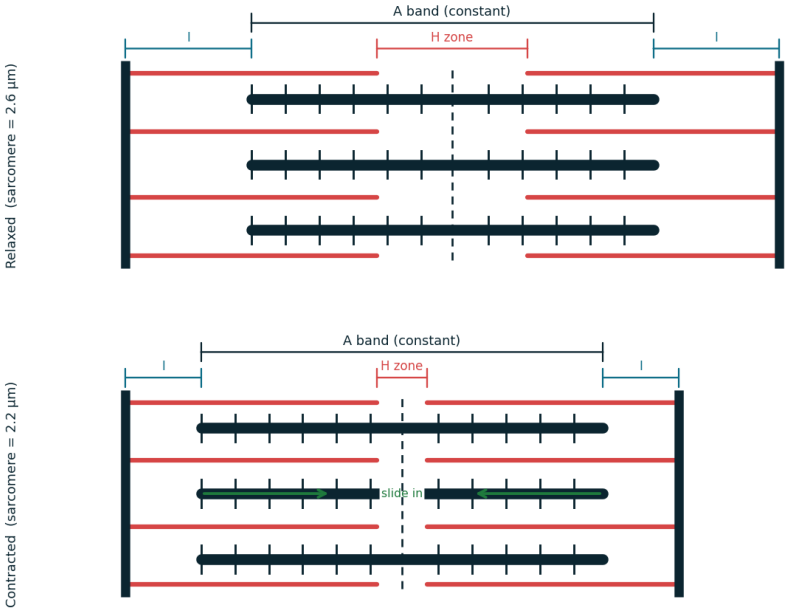

Figure 2. Sliding-filament model. On contraction the sarcomere shortens 2.6 → 2.2 µm (a 15.4% decrease). Thin filaments slide inward, so the I band narrows (1.0 → 0.6 µm, -40%) and the H zone narrows (0.6 → 0.2 µm, -66.7%), while the A band stays constant at 1.6 µm.

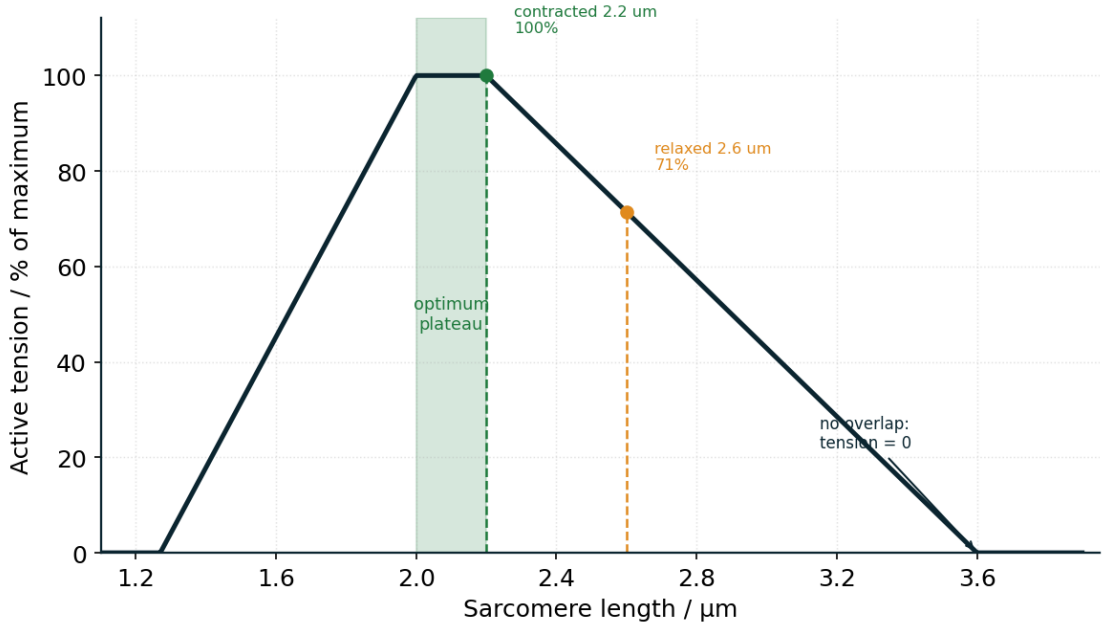

Figure 3. Length-tension relationship. Active tension tracks the thick-thin filament overlap: it is maximal over the optimum plateau (2.0-2.2 µm) and falls to zero at 3.6 µm where overlap is lost. The relaxed length (2.6 µm) sits on the descending limb at 71%; shortening toward 2.2 µm raises tension to 100%.

5. The cross-bridge cycle

Sliding is driven by the myosin heads repeatedly attaching to actin, pulling, releasing and re-attaching further along. One complete round is the **cross-bridge cycle**; each cycle moves the thin filament a small step, and millions of cycles occurring asynchronously produce a smooth, sustained contraction. Start from a myosin head already 'cocked' and carrying the products of ATP hydrolysis (ADP + P_i).

Step	Event
1 Attachment	The energised myosin head binds to an exposed binding site on actin, forming a cross-bridge.
2 Power stroke	The head releases ADP and P_i and swivels/tilts, pulling the thin filament toward the centre of the sarcomere. This is the force-generating step.
3 Detachment	A new **ATP** molecule binds to the myosin head. This binding lowers the head's affinity for actin, so the cross-bridge breaks and the head detaches.
4 Re-cocking (hydrolysis)	The head hydrolyses ATP → ADP + P_i; the energy released re-cocks the head back to its high-energy position, ready to bind again further along the actin.

The role of ATP

ATP is needed at **two** distinct points, and in muscle physiology it has **three** essential roles overall. Being explicit about all of these scores full marks.

- ATP **binding** to the myosin head causes it to **detach** from actin (step 3).
- ATP **hydrolysis** provides the energy to **re-cock** the head for the next power stroke (step 4).
- ATP also powers the calcium pump that returns Ca^{2+} to the sarcoplasmic reticulum during relaxation (section 6).

> **Rigor mortis link:** After death ATP production stops. Without ATP the myosin heads cannot detach from actin, so the cross-bridges lock and the muscle becomes rigid — this is rigor mortis. It is direct evidence that ATP is needed for **detachment**, not for the power stroke.

6. Calcium ions and the sarcoplasmic reticulum

The cross-bridge cycle can only run when the actin binding sites are exposed, and this is controlled by **calcium ions**. Ca^{2+} is stored in the **sarcoplasmic reticulum** (SR) — a specialised endoplasmic reticulum that forms a network around each myofibril. Excitation of the muscle releases Ca^{2+} and switches contraction on.

From nerve impulse to contraction

1. A motor neuron releases neurotransmitter at the neuromuscular junction, triggering an **action potential** that spreads across the sarcolemma and down the T-tubules into the fibre.

2. The action potential causes the SR to release stored Ca^{2+} into the sarcoplasm, so the Ca^{2+} concentration around the myofibrils rises sharply.

3. Ca^{2+} **binds to troponin**, changing its shape.

4. The troponin change pulls **tropomyosin** away from the actin, **exposing the myosin-binding sites**.

5. Myosin heads can now bind actin, and the cross-bridge cycle runs — the muscle contracts (as long as ATP and Ca^{2+} are present).

Relaxation

When the nerve stimulation stops, Ca^{2+} is actively pumped back into the SR by calcium pumps in the SR membrane, using **ATP**. As the sarcoplasmic Ca^{2+} concentration falls, Ca^{2+} leaves troponin, tropomyosin

moves back to cover the binding sites, cross-bridges can no longer form, and the muscle relaxes. Elastic recoil and the contraction of the antagonistic muscle then return the sarcomeres to their resting length.

> **Two jobs for ATP:** Contraction needs ATP for the cross-bridge cycle (detachment + re-cocking); relaxation *also* needs ATP to pump Ca^{2+} back into the SR. Both processes are active and energy-consuming.

7. Motor units, control of force and fibre types

A single contraction of a whole muscle can be weak or powerful. The nervous system grades the force using motor units.

Motor units

A **motor unit** is a single motor neuron together with all the muscle fibres it stimulates. When the neuron fires, every fibre in its unit contracts together. Small motor units (few fibres) allow fine control (e.g. eye muscles); large motor units (many fibres) give powerful but coarse contractions (e.g. thigh muscles).

Grading the force of contraction

- **Recruitment** – to increase force, the nervous system activates a greater number of motor units at the same time.
- **Summation (frequency)** – to increase force, motor neurons fire action potentials more frequently, so successive twitches add together before the fibre can fully relax, giving a larger, sustained (tetanic) contraction.

Fast-twitch and slow-twitch fibres

Feature	Slow-twitch (Type I)	Fast-twitch (Type II)
Contraction speed	Slow	Fast
Main ATP source	Aerobic respiration	Anaerobic respiration (glycolysis)
Mitochondria	Many	Few
Myoglobin / capillaries	High (red in colour)	Low (paler)
Fatigue resistance	High – resists fatigue	Low – fatigues quickly
Best suited to	Endurance / posture (e.g. marathon)	Short, powerful bursts (e.g. sprinting)

> **Application:** Most muscles contain a mix of both fibre types; the proportion varies between people and muscles. Endurance athletes tend to have a higher proportion of slow-twitch fibres, sprinters more fast-twitch.

8. Other forms of motility

Muscle is not the only way cells and organisms move. The syllabus requires a brief outline of two further mechanisms, both driven by motor proteins and cytoskeletal filaments.

Cilia and flagella

Eukaryotic cilia and flagella move by the **sliding of microtubules**. Their core (the axoneme) has the classic **9 + 2** arrangement: nine pairs of microtubules around two central ones. The motor protein **dynein** uses ATP to make adjacent microtubule doublets slide against one another; because the doublets are anchored, this sliding is converted into **bending**, producing the beating movement that propels a cell (e.g. a sperm flagellum) or moves fluid past it (e.g. ciliated cells of the airways).

Amoeboid movement

Cells such as *Amoeba* and human white blood cells crawl by **amoeboid movement**. The cell extends a projection called a **pseudopodium** ('false foot') by the controlled assembly (polymerisation) of **actin** filaments at the leading edge, coupled with sol-gel transformations of the cytoplasm. The cell anchors the pseudopodium and flows its contents forward. This ATP-dependent, actin-based crawling underlies phagocytosis and the movement of white blood cells toward sites of infection.

> **Common thread:** All three systems – skeletal muscle, cilia/flagella and amoeboid movement – use **motor proteins** (myosin or dynein) walking along **cytoskeletal filaments** (actin or microtubules), powered by **ATP**.

9. Worked examples and exam skills

Worked example 1 – labelling a sarcomere

A micrograph shows one repeating unit of a myofibril. Identify: (a) the boundary marking the ends of the unit, (b) the light band with actin only, (c) the dark band, (d) the lighter central region of the dark band.

(a) The **Z line** – a sarcomere runs from one Z line to the next.

(b) The **I band** – light, thin (actin) filaments only, straddling the Z line.

(c) The **A band** – dark, the full length of the thick (myosin) filaments including the overlap region.

(d) The **H zone** – thick filaments only, with the **M line** down its centre.

Worked example 2 – bands during contraction

Explain what happens to the width of the I band, the H zone and the A band as a sarcomere contracts, and why.

The thin filaments slide inward past the thick filaments, so the overlap increases. The **I band shortens** (actin-only region shrinks) and the **H zone shortens** (thin filaments intrude into the myosin-only region).

The **A band stays the same width**, because it equals the length of the thick filaments, and the filaments *slide* rather than change length.

Worked example 3 — sequence the cross-bridge cycle

Put the following into the correct order, starting from an energised myosin head: power stroke; ATP hydrolysis re-cocks the head; myosin binds actin; ATP binds and head detaches.

1. Myosin head **binds actin** (cross-bridge forms).
2. **Power stroke** — head swivels, releasing ADP + P_i and pulling the thin filament inward.
3. **ATP binds** the head, which then **detaches** from actin.
4. **ATP hydrolysis re-cocks** the head, ready to bind again further along.

Worked example 4 — the role of calcium

Explain how a rise in Ca^{2+} concentration switches on contraction.

An action potential causes the sarcoplasmic reticulum to release Ca^{2+}. The Ca^{2+} **binds to troponin**, which changes shape and pulls **tropomyosin** off the actin. This **exposes the myosin-binding sites**, allowing cross-bridges to form and the cycle to run. When Ca^{2+} is pumped back into the SR (using ATP), tropomyosin re-covers the sites and the muscle relaxes.

Common pitfalls

- Saying the filaments **shorten**. They do not — they **slide** past each other; the sarcomere shortens.
- Claiming the **A band shortens**. It stays constant during both contraction and stretching.
- Thinking ATP is only for the power stroke. ATP is needed for **detachment** (binding) and for **re-cocking** (hydrolysis), and again to **pump** Ca^{2+} back into the SR.
- Confusing **tropomyosin** (blocks the sites) with **troponin** (binds Ca^{2+}).
- Confusing **tendons** (muscle to bone) with **ligaments** (bone to bone).

10. Quick reference

Item	Key point
Antagonistic pair	Biceps (flexor) and triceps (extensor); one contracts while the other relaxes.
Tendon vs ligament	Tendon: muscle → bone. Ligament: bone → bone.
Muscle hierarchy	Muscle → fibre (multinucleate) → myofibril → sarcomere.
Banding	I band = actin only (light); A band = full thick filament (dark); H zone = myosin only; M line centres it; Z line bounds the sarcomere.
Sliding filament	Actin slides past myosin; I band and H zone shorten; A band constant.
Cross-bridge cycle	Bind → power stroke → ATP binds, detach → ATP hydrolysed, re-cock.
Roles of ATP	Detachment; re-cocking; pumping Ca^{2+} back into the SR.
Calcium switch	AP → SR releases Ca^{2+} → troponin → tropomyosin moves → sites exposed.

Item	Key point
Force control	Recruitment (more motor units) and summation (higher firing frequency).
Fibre types	Slow twitch: aerobic, fatigue-resistant, endurance. Fast twitch: anaerobic, powerful, fatigues fast.
Other motility	Cilia/flagella: microtubules slide via dynein. Amoeboid: actin polymerises to form pseudopodia.

11. Test yourself

Attempt these without notes; full answers follow.

1. Explain why skeletal muscles must operate in antagonistic pairs, using the biceps and triceps as an example.

2. State one function each of synovial fluid, cartilage and ligaments in a synovial joint.

3. Name the repeating contractile unit of a myofibril and state which structures mark its boundaries.

4. Describe what happens to the I band, H zone and A band when a sarcomere contracts, and explain why the A band does not change.

5. Outline the cross-bridge cycle in four steps, starting from an energised (cocked) myosin head.

6. Explain the two distinct roles of ATP in the cross-bridge cycle, and state a third role of ATP in muscle relaxation.

7. Trace the sequence of events from an action potential arriving at a muscle fibre to the exposure of the myosin-binding sites.

8. Distinguish between recruitment and summation as ways of increasing the force of a muscle contraction.

Answers

1. A muscle can only generate force by contracting (shortening) and pulling; it cannot push. So one muscle can move a bone in only one direction. The biceps contracts to flex (bend) the arm; to extend (straighten) it, the triceps must contract while the biceps relaxes. The two produce opposite movements, so they are antagonistic.

2. Synovial fluid: lubricates the joint / reduces friction. Cartilage: reduces friction and absorbs shock at the bone ends. Ligaments: join bone to bone, stabilising the joint and limiting movement.

3. The **sarcomere**; it runs from one **Z line** to the next Z line.

4. The I band shortens and the H zone shortens (thin filaments slide inward, increasing overlap). The **A band stays constant** because its width equals the length of the thick (myosin) filaments, and the filaments slide past each other rather than shortening.

5. (i) The myosin head binds an exposed site on actin. (ii) Power stroke: the head swivels, releasing ADP + P_i and pulling the thin filament toward the sarcomere centre. (iii) ATP binds the head, which detaches from actin. (iv) ATP is hydrolysed to ADP + P_i, re-cocking the head for the next cycle.

6. In the cross-bridge cycle, ATP **binding** to the myosin head causes it to **detach** from actin, and ATP **hydrolysis** provides energy to **re-cock** the head. A third role: ATP powers the pumps that return Ca^{2+} to the sarcoplasmic reticulum during relaxation.

7. Action potential spreads across the sarcolemma and down the T-tubules → the sarcoplasmic reticulum releases Ca^{2+} → Ca^{2+} binds troponin → troponin changes shape and moves tropomyosin → the myosin-binding sites on actin are exposed.

8. **Recruitment**: increasing force by activating a greater number of motor units simultaneously. **Summation**: increasing force by stimulating the fibres more frequently, so twitches overlap and add together before relaxation, producing a larger sustained contraction.