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PHYSIOLOGY · STUDY GUIDE

How Skeletal Muscle Contracts

The Sliding-Filament and Cross-Bridge Cycle

Every movement an animal makes — a heartbeat, a breath, a gallop — is a muscle turning ATP into force. It does it by sliding two sets of filaments, actin and myosin, past each other so the sarcomere shortens.

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IN SHORT

Every movement an animal makes — a heartbeat, a breath, a gallop — is a **muscle turning ATP into force**. It does it by sliding two sets of filaments, **actin** and **myosin**, past each other so the **sarcomere** shortens. The switch is **calcium**: a nerve impulse makes the muscle release Ca^{2+} from its internal store, Ca^{2+} frees the actin, and myosin heads ratchet along it. Understand that one mechanism and the clinic follows — why a pig goes rigid under anaesthesia (malignant hyperthermia), why a fresh cow goes down (milk fever), and why a hard-worked horse passes red-brown urine (tying-up).

KEY POINTS

1. Muscle turns **ATP into movement** by sliding **actin (thin)** and **myosin (thick)** filaments past each other — the sarcomere shortens, the filaments themselves do not.
2. The trigger is **calcium**: Ca^{2+} binds **troponin**, shifts **tropomyosin** off the actin, and lets the myosin heads grab on.
3. The **cross-bridge cycle** — bind → power-stroke → detach → re-cock — repeats while Ca^{2+} and ATP last; ATP is needed even to *let go*, which is why muscle stiffens in **rigor mortis**.
4. **Excitation-contraction coupling** links nerve to force: a muscle AP runs down the **T-tubules**, the **SR** dumps Ca^{2+} → contraction; pumping Ca^{2+} back = relaxation.
5. Force is graded two ways: **recruiting more motor units** and **firing faster** (summation → tetanus).
6. The same machine is retuned per type: **cardiac** uses calcium-induced calcium release and cannot be tetanised; **smooth** has no troponin and works through **calmodulin**.
7. It is a **clinical hotspot** — the ryanodine receptor (malignant hyperthermia), calcium (milk fever) and muscle fuel (equine rhabdomyolysis) all live in this pathway.

01 Muscle: three types, one job — turning ATP into force

Muscle is the body's motor. It is the one tissue built to **convert chemical energy (ATP) into mechanical force and movement**, and it makes up an enormous share of the animal — roughly **40% of body mass** is skeletal muscle alone. Whatever the animal does that moves — walk, breathe, pump blood, push food through the gut, shiver to keep warm — a muscle is doing it.

There are **three types**, and telling them apart is the first step. **Skeletal muscle** is the big striped (striated) muscle bolted to the skeleton; it is under **voluntary** control, each fibre driven by its own motor nerve, and it is the model we use to learn how contraction works. **Cardiac muscle** is also striated but **involuntary** and self-triggering — its cells are wired together so the heart beats as one. **Smooth muscle** lines the hollow organs and vessels; it is **non-striated**, involuntary, and specialised for slow, sustained squeezing over a wide range of lengths.

All three run on the same core trick — sliding filaments powered by ATP and switched on by calcium — but each is tuned differently, and those differences explain a great deal of clinical medicine. We'll build the whole story from the skeletal muscle model, then see how cardiac and smooth muscle re-use it.

	SKELETAL	CARDIAC	SMOOTH
Striations	Striated	Striated	Non-striated
Control	Voluntary	Involuntary	Involuntary
Trigger	Nerve → SR Ca^{2+}	CICR	calmodulin
Tetanus?	Yes	No (long AP)	Sustained tone
Example	Limb muscles	Heart	Gut, vessels

Fig 1 — The three muscle types at a glance. Same sliding-filament machine, three different control schemes.

02 Inside a skeletal muscle: from belly to sarcomere

A skeletal muscle is a set of nested bundles. The fleshy **belly** is wrapped in connective tissue (epimysium) and split into **fascicles** (perimysium), each holding many **muscle fibres** — the individual cells, sheathed in endomysium. Those sheaths all merge into the **tendon**, so the pull of a single fibre is transmitted straight to bone. A muscle fibre is a remarkable cell: **multinucleate** (formed by fusion of many precursor cells), 10–100 µm across, and in some muscles up to tens of centimetres long.

Each fibre is packed with hundreds to thousands of parallel **myofibrils**, and each myofibril is a chain of **sarcomeres** — the actual contractile unit, repeated end to end tens of thousands of times. A sarcomere runs from one **Z-disk** to the next and contains two interleaved filaments: **thin filaments (actin)** anchored to the Z-disks, and **thick filaments (myosin)** suspended in the middle, each thick filament bristling with roughly 500 myosin heads. The precise alignment of these filaments gives skeletal and cardiac muscle their striped look; the springy giant protein **titin** holds everything centred and sets the muscle's resting length.

Two more structures make electrical control possible. The cell's Ca^{2+} store, the **sarcoplasmic reticulum (SR)**, wraps every myofibril. And the surface membrane dives into the fibre as **T-tubules** — tunnels of membrane that carry the electrical signal deep inside, right up against the SR. Hold those two in mind; they are where excitation becomes contraction.

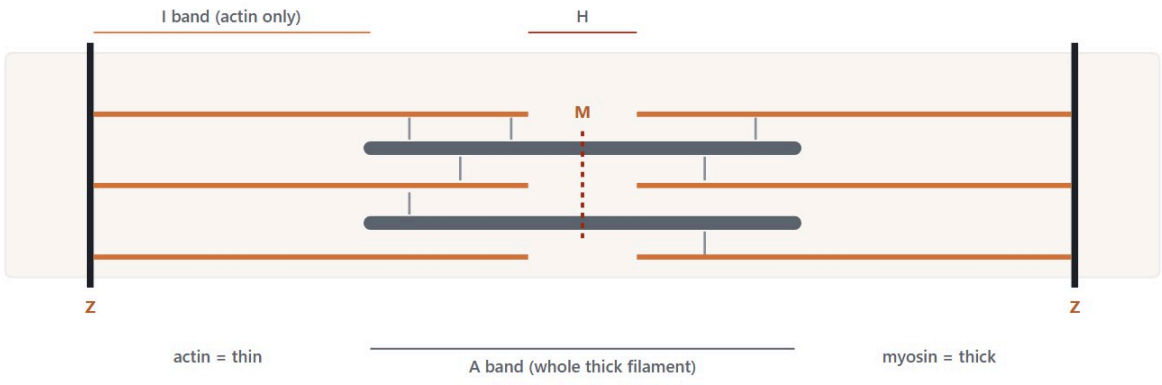


Fig 2 — The sarcomere: thin actin filaments anchored to the Z-disks interdigitate with central thick myosin filaments (A band; actin-only I band; myosin-only H zone; M line at the centre).

03 How it shortens: the sliding-filament theory & cross-bridge cycle

Here is the single most important idea in muscle physiology: **the filaments do not get shorter — they slide.** When a muscle contracts, the myosin heads reach out, grab the actin, and haul the thin filaments in toward the centre of the sarcomere. The actin and myosin overlap more, the Z-disks are pulled closer, and the sarcomere shortens; multiply that across tens of thousands of sarcomeres in series and the whole muscle shortens. This is the **sliding-filament mechanism**.

The pulling is done by the **cross-bridge cycle**, a four-beat action each myosin head performs over and over. (1) An ATP is hydrolysed to ADP + phosphate, which **cocks** the head into a high-energy position. (2) The cocked head **binds** an exposed site on actin, forming a cross-bridge. (3) It releases the phosphate and snaps back — the **power stroke** — dragging the actin ~10 nm. (4) A fresh ATP binds, the head **lets go**, and the cycle repeats. Thousands of heads doing this out of step, like a rope-team hauling hand over hand, produce a smooth pull.

Notice that ATP is needed twice — to cock the head *and* to detach it. That second requirement has a famous consequence: after death, when ATP runs out, the heads bind actin and cannot release, so the whole body stiffens — **rigor mortis**. The same "no ATP, no release" rule is why a living muscle must keep its energy supply up just to relax.

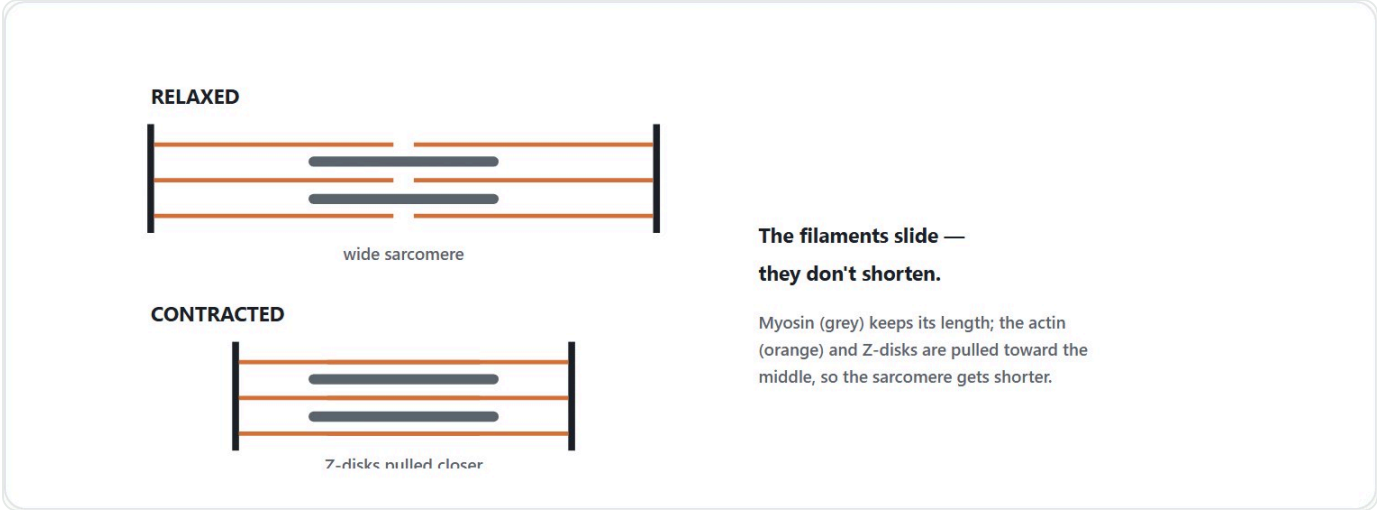


Fig 3 — Sliding-filament theory: from relaxed to contracted, the Z-disks are pulled closer and overlap increases — but each filament is exactly as long as before.

04 The calcium switch: excitation-contraction coupling

A muscle only contracts when told to, and the messenger that carries "go" from the nerve to the filaments is **calcium**. At rest, the actin's binding sites are physically covered by **tropomyosin**, held in place by **troponin**, so myosin cannot grip. Contraction begins the instant Ca^{2+} appears: Ca^{2+} binds troponin, troponin changes shape, tropomyosin swings aside, and the myosin-binding sites are exposed. Remove the Ca^{2+} and the cover slides back — the muscle relaxes. Calcium is the whole switch.

Getting that Ca^{2+} to appear on cue is **excitation–contraction coupling**. A motor neuron fires and releases **acetylcholine** at the neuromuscular junction, which triggers an action potential on the muscle fibre (reliably one-for-one). That impulse races along the surface and dives down the **T-tubules** into the fibre's core. There, the voltage sensor in the T-tubule membrane is **mechanically linked** to the SR's **ryanodine-receptor** calcium channels — so the electrical signal pops those channels open and the SR floods the sarcomere with stored Ca^{2+}. Cross-bridges cycle, the fibre contracts.

Relaxation is not passive: dedicated **ATP-driven pumps** constantly haul Ca^{2+} back into the SR. When the action potentials stop, reuptake wins, cytosolic Ca^{2+} falls, tropomyosin re-covers the actin, and the muscle lets go — a process that takes tens of milliseconds. That ryanodine receptor and this Ca^{2+} handling are not academic details: they are exactly where **malignant hyperthermia** and **milk fever** strike.

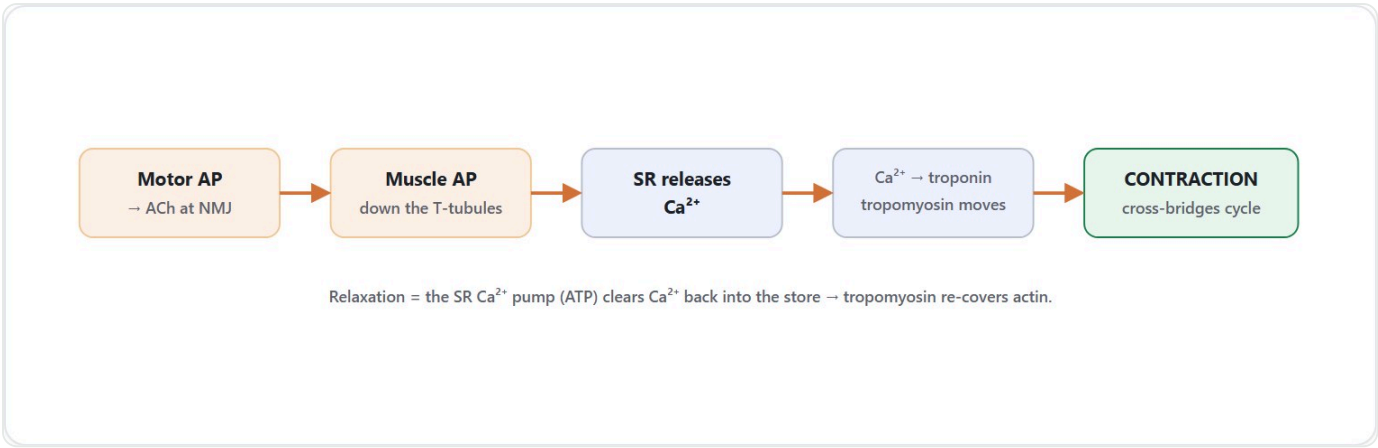


Fig 4 — Excitation-contraction coupling: nerve impulse → muscle AP → T-tubules → SR releases Ca^{2+} → troponin/tropomyosin switch → contraction. Pumping Ca^{2+} back = relaxation.

05 Grading the force: motor units, twitches & fibre types

A single nerve impulse to a fibre gives a single brief **twitch**. Whole-animal movement needs far finer control than that, and the body grades force in two ways. First, the **motor unit** — one motor neuron plus all the fibres it drives — is the smallest switch, and the nervous system **recruits** more of them as more force is needed. By the **size principle**, small, fatigue-resistant units switch on first for delicate work, and larger, more powerful units join in for heavy effort. Second, firing a unit **faster** makes each twitch pile onto the last (**summation**) until they fuse into a smooth, maximal **tetanus** — three to five times the force of a single twitch. Force is also best in a narrow **length range** where filament overlap is optimal, which is why muscles sit at close to their ideal length on the skeleton.

Not all fibres are alike. **Slow-twitch (type I, "red")** fibres are packed with mitochondria and myoglobin, run on oxygen, and resist fatigue — ideal for posture and endurance. **Fast-twitch (type II, "white")** fibres contract quickly and powerfully but tire fast. The mix is genetically set and strikingly species- and breed-specific.

FIBRE TYPE	SPEED	FATIGUE	FUEL / COLOUR	BEST FOR
Type I (slow)	Slow	Very resistant	Oxidative · red	Posture, endurance
Type IIa (fast)	Fast	Moderate	Oxidative · red-ish	Sustained fast work
Type IIx / IIb (fast)	Fastest	Fatigues fast	Glycolytic · white	Sprint, jump, power

For vets The mix is dramatic across species: a greyhound's propulsion muscles are ~**97% fast-twitch** and a fast horse's are >90%, while a camel — built for stamina, not speed — runs about **30%**. It's muscle physiology you can see in the animal in front of you.

06 Fuelling the machine — and why it fatigues

Muscle fatigue is not one thing, and it is almost never the muscle "running out of ATP" — a fibre that truly exhausted its ATP would go into rigor, not go slack. It is the accumulation of the by-products of hard work and the failure of the calcium machinery to keep cycling at full speed.

Contraction is expensive, and a working muscle can raise its energy use a thousandfold in a fraction of a second. ATP is spent at **three** steps — cocking the heads, detaching them, and pumping Ca^{2+} back into the SR — so a steady supply is essential. But the fibre only stores enough ATP for a second or two, so it layers its fuel: **creatine phosphate** instantly tops ATP back up (good for about **8–10 seconds** of all-out effort); **glycogen** broken down through glycolysis and oxidative metabolism covers seconds to hours; and prolonged work shifts increasingly to **fat**. Overall the muscle is only ~25% efficient, so most of the energy becomes **heat** — which is exactly why shivering warms an animal up.

Fatigue is not, as long assumed, simply running out of ATP — fibres keep ATP nearly constant until the very end, because a true energy crash would lock them in rigor. Instead fatigue is a protective slowdown driven by the build-up of **inorganic phosphate** (from spent creatine phosphate), a fall in SR calcium release, and disturbed ion gradients. It is a safety brake, not a failure.

07 The same machine, retuned: cardiac & smooth muscle

Cardiac muscle keeps the striated sarcomere but is rewired for a pump. Its cells are short, branched, and joined end-to-end at **intercalated discs** whose **gap junctions** let the impulse jump cell to cell, so the heart contracts as one. Its calcium trigger is different too: a little Ca^{2+} entering from outside during the action potential sets off a bigger release from the SR — **calcium-induced calcium release**. And crucially its action potential is *long*, giving a long refractory period, so cardiac muscle **cannot be tetanised** — it must relax and refill between beats.

Smooth muscle throws out the sarcomere altogether: its actin and myosin are arranged irregularly and anchored to **dense bodies**, and it has **no troponin**. So it switches on a different way — Ca^{2+} (much of it from outside the cell) binds **calmodulin**, which activates **myosin light-chain kinase** to turn the myosin heads on. It is slow, works over a huge range of lengths, and can hold tension for long periods on very little ATP (a "latch"-like state) — perfect for a gut wall or a blood vessel that must stay contracted for hours. It can be told to contract *or* relax by the autonomic nerves, hormones, and even stretch.

08 When muscle physiology goes wrong

Because so much hinges on calcium and ATP, muscle is a rich source of clinical disease — and each condition is this chapter's biology failing at a specific point.

Malignant hyperthermia is the ryanodine receptor gone rogue: a genetic defect (classically in pigs, triggered by the anaesthetic **halothane**) lets the SR pour out Ca^{2+} uncontrollably. The muscle contracts without stopping — rigidity, a rocketing temperature, acidosis and muscle breakdown — and the antidote, **dantrolene**, works by blocking that SR calcium release.

Milk fever (periparturient hypocalcaemia) is the mirror image: too *little* calcium. The fresh cow's demand for calcium in colostrum outstrips supply, blood Ca^{2+} falls, and because Ca^{2+} is needed both to release acetylcholine at the neuromuscular junction and to run the contractile switch, the cow weakens and goes down. Slow intravenous calcium can reverse it dramatically. (Note the species twist: the same low calcium causes flaccid *paresis* in ruminants but *tetany* in dogs, because their neuromuscular safety margins differ.)

Myasthenia gravis is the mirror image again, one synapse further on. In milk fever the problem is *releasing* acetylcholine; in myasthenia the transmitter is released normally but there is nothing left to receive it. In the **acquired** form — much the commonest in dogs — autoantibodies destroy the **nicotinic acetylcholine receptors** on the postsynaptic membrane, so each nerve impulse produces a smaller endplate potential than it should. The signature is **weakness that worsens with exercise and recovers with rest**: a dog that walks well for two minutes and then sinks. Because the oesophagus of the dog is striated muscle along its whole length, roughly 90% of canine cases also develop **megaoesophagus**, and it is often the regurgitation — and the aspiration pneumonia that follows — rather than the weakness that brings the dog in. Diagnosis rests on a serum **anti-acetylcholine-receptor antibody titre**, and treatment on an anticholinesterase such as **pyridostigmine**, which leaves more acetylcholine in the cleft to find the receptors that remain. A congenital form, with too few receptors from birth rather than antibodies against them, occurs in Jack Russell terriers, springer spaniels and smooth fox terriers.

Exertional rhabdomyolysis ("tying-up") in horses is muscle fibres literally breaking down after strenuous work — a stiff, painful, sweating horse that may pass **red-brown urine** as released myoglobin reaches the kidneys, risking kidney injury. And **white muscle disease** — selenium/vitamin-E deficiency in lambs and calves — lets oxidative damage destroy muscle, including the heart. In every case, knowing the normal physiology tells you what has failed and what to do.

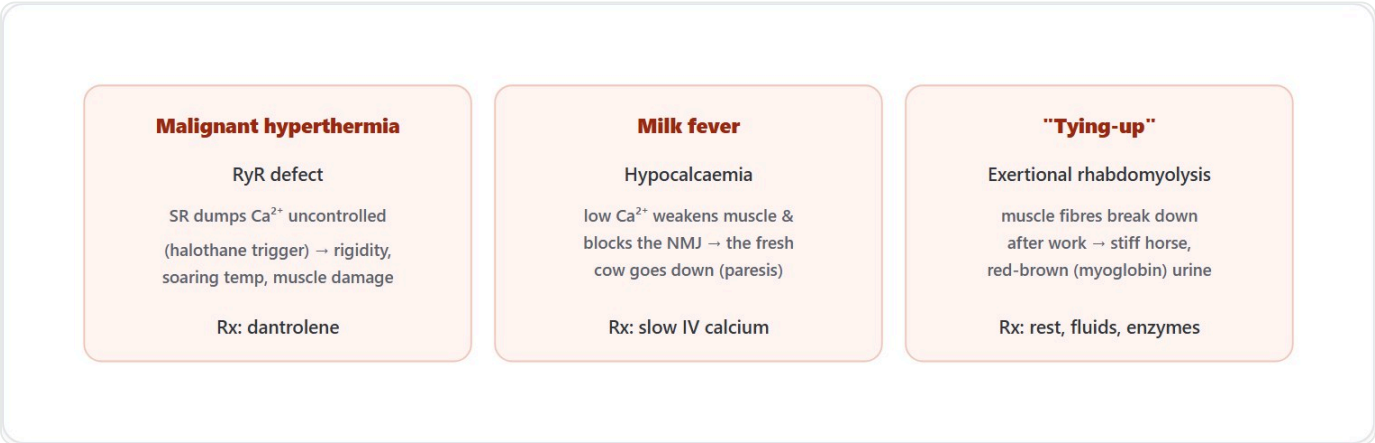


Fig 5 — Three ways the biology fails in practice: too much SR calcium (malignant hyperthermia), too little blood calcium (milk fever), and muscle breakdown after exertion (tying-up).

> **Red flag**
>
> A patient turning rigid + hyperthermic under anaesthesia (malignant hyperthermia), a down periparturient cow (milk fever), or a horse stiff with red-brown urine after work (rhabdomyolysis) are muscle-physiology emergencies — act on the calcium/temperature/fluids fast.

09 Quiz: test yourself

Seventeen questions across the sarcomere, the cross-bridge cycle, the calcium switch, fibre types and the diseases that break each step. Answers are beneath each question — cover them and work down.

1. What share of body mass is skeletal muscle, and what does muscle uniquely do?**Answer:** Roughly **40%**. It is the one tissue built to convert **chemical energy (ATP) into mechanical force**.2. What kind of cell is a muscle fibre, and how big?**Answer: Multinucleate** — formed by fusion of many precursor cells — **10–100 µm** across, and in some muscles up to tens of centimetres long.3. What defines a sarcomere, and what is in it?**Answer:** It runs from one **Z-disk** to the next. **Thin filaments (actin)** anchored to the Z-disks interleave with **thick filaments (myosin)** suspended in the middle, each thick filament carrying roughly **500 myosin heads**.4. What are T-tubules for?**Answer:** They are tunnels of surface membrane diving into the fibre, carrying the **electrical signal deep inside**, right up against the sarcoplasmic reticulum.5. What are the four beats of the cross-bridge cycle?**Answer:** (1) ATP is hydrolysed, **cocking** the myosin head. (2) The head **binds** an exposed site on actin. (3) It releases phosphate and snaps back — the **power stroke**, dragging actin about **10 nm**. (4) A new ATP binds and the head **detaches**.6. What links the electrical signal to calcium release?**Answer:** The **voltage sensor in the T-tubule membrane is mechanically linked** to the SR's **ryanodine-receptor** calcium channels, so depolarisation pops them open and the SR floods the sarcomere with Ca^{2+}.7. Is relaxation passive?**Answer: No.** Dedicated **ATP-driven pumps** constantly haul Ca^{2+} back into the SR. Relaxation costs energy just as contraction does.8. How do type I and type II fibres differ?**Answer: Type I (slow, red)** are packed with mitochondria and myoglobin, run on oxygen and resist fatigue — posture and endurance. **Type II (fast, white)** contract quickly and powerfully but tire fast.9. How does fibre-type mix vary across species?**Answer:** A greyhound's propulsion muscles are about **97% fast-twitch** and a fast horse's over **90%**, while a camel — built for stamina — runs about **30%**.10. At which three steps is ATP spent in a contracting muscle?**Answer: Cocking** the heads, **detaching** them, and **pumping Ca^{2+}** back into the SR.11. How does a fibre fuel itself beyond its stored ATP?**Answer:** It stores only a second or two of ATP, so it layers fuel: **creatine phosphate** tops ATP straight back up for about **8–10 seconds** of all-out effort, then **glycogen** through glycolysis and oxidative metabolism.12. How efficient is muscle, and what follows from that?**Answer:** Only about **25%**. Most of the energy becomes **heat** — which is exactly why shivering warms an animal.13. How is smooth muscle retuned for its job?**Answer:** It is slow, works over a **huge range of lengths**, and can hold tension for long periods on very little ATP — a **latch**-like state, ideal for a gut wall or a vessel that must stay contracted for hours.14. What is malignant hyperthermia, and what is the antidote?**Answer:** The **ryanodine receptor gone rogue** — classically in pigs, triggered by **halothane** — letting the SR pour out Ca^{2+} uncontrollably: rigidity, rocketing temperature, acidosis, muscle breakdown. The antidote is **dantrolene**, which blocks SR calcium release.15. Why does a fresh cow with milk fever go down?**Answer:** Demand for calcium in colostrum outstrips supply, so blood Ca^{2+} falls. Calcium is needed both to **release acetylcholine** at the junction and to run the **contractile switch**, so she weakens and becomes recumbent.16. The same hypocalcaemia causes flaccid paresis in a cow but tetany in a dog. Why?**Answer:** Their **neuromuscular safety margins differ**. The same electrolyte fault expresses itself in opposite directions depending on species.17. How does myasthenia gravis differ from milk fever, mechanistically?**Answer:** One synapse further on. In milk fever the problem is **releasing** acetylcholine; in myasthenia the transmitter is released normally but **autoantibodies have destroyed the nicotinic receptors** that should receive it.

10 Muscle contraction physiology — frequently asked questions

What is muscle contraction in simple terms?

Myosin (thick) filaments grab actin (thin) filaments and pull them inward, so the contractile units (sarcomeres) shorten and the whole muscle shortens. It runs on ATP and is switched on by calcium.

What role does calcium play in muscle contraction?

Calcium is the on-switch. Released from the sarcoplasmic reticulum, it binds troponin and moves tropomyosin off the actin, letting myosin bind and pull. Pumping calcium back into the store switches the muscle off (relaxation).

Why does the body stiffen after death (rigor mortis)?

Myosin heads need a fresh ATP to detach from actin. After death ATP production stops, so the heads stay bound and the muscle locks — rigor mortis — until enzymes break the tissue down.

How do muscles contract?

By pulling, not by shortening. Myosin heads reach out, grip the actin filament, and swivel — the **power stroke** — dragging the thin filaments past the thick ones so the sarcomere shortens while the filaments themselves stay exactly the same length. That is the **sliding-filament theory**. Each head then binds a fresh ATP to let go, splits it to re-cock itself, and grips again a little further along. Thousands of heads repeat this **cross-bridge cycle** out of step with one another, so the pull is smooth rather than jerky. The whole thing is gated by calcium: without Ca^{2+}, tropomyosin covers the binding sites and no cross-bridge can form.

Why do muscles get tired?

Several mechanisms at once, and the mix depends on the work. In short, intense effort the limit is metabolic: phosphate released from ATP breakdown accumulates and interferes with cross-bridge force, hydrogen ions build up as lactate is produced, and the sarcoplasmic reticulum becomes slower at both releasing and recapturing calcium. In prolonged submaximal work the limit is fuel — muscle glycogen is depleted. Fatigue is, in that sense, protective: it stops the fibre before it destroys itself. When an animal *does* push past that point, the result is exertional rhabdomyolysis.

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When to refer

- A pig or dog turning rigid, hyperthermic and tachycardic under anaesthesia (esp. halothane) — suspect **malignant hyperthermia**: stop the trigger, cool actively, give **dantrolene**.
- A recumbent periparturient cow (**milk fever**) — hypocalcaemia (hypocalcemia) weakens muscle and blocks the neuromuscular junction; treat with slow IV calcium and monitor for relapse.
- A horse with a stiff, painful gait and red-brown urine after exercise — suspect **exertional rhabdomyolysis ('tying-up')**; stop work, give fluids, check muscle enzymes and renal function.
- Weak, stiff lambs or calves from selenium/vitamin-E-deficient dams — suspect **white muscle disease**, which can strike cardiac muscle and kill; supplement and prevent in the dam.

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1. Klein BG. Cunningham's Textbook of Veterinary Physiology. 6th ed. St Louis: Elsevier; 2020. Chapter 6 — The Physiology of Muscle (pp 73–81): the sarcomere, sliding-filament mechanism, cross-bridge cycle, excitation-contraction coupling, motor units, and cardiac vs smooth muscle.
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3. Clinical correlations (malignant hyperthermia / ryanodine receptor + dantrolene; hypocalcaemic paresis / milk fever; equine exertional rhabdomyolysis; selenium/vitamin-E white muscle disease) per Cunningham 6e Ch 6 & PDA 3e Ch 8.

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