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— STUDY NOTES

Muscle Contraction Physiology

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).

INSIDE THESE NOTES

LEVEL
Vets & veterinary students

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2026-07-01

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Muscle Contraction Physiology

STUDY NOTES · BASIC SCIENCES · PHYSIOLOGY · UPDATED 2026-07-01

CONTENTS

LEARNING OBJECTIVES

After working through these notes you will be able to:

- ✓ Name the three muscle types and their key structural and functional differences.
- ✓ Describe the sarcomere and the sliding-filament theory of contraction.
- ✓ Explain the cross-bridge cycle and the roles of ATP and Ca^{2+} (including rigor).
- ✓ Walk through excitation-contraction coupling from motor nerve to relaxation.
- ✓ Explain how force is graded, and link the biology to malignant hyperthermia, milk fever and equine 'tying-up'.

TL;DR

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).

AT A GLANCE

DEFINITION	Muscle converts ATP into force by sliding actin and myosin filaments past each other
THREE TYPES	Skeletal (striated, voluntary), cardiac (striated, involuntary), smooth (non-striated)
CONTRACTILE UNIT	The sarcomere — thick myosin + thin actin, between two Z-disks
SLIDING-FILAMENT	Filaments slide and overlap more; the sarcomere shortens, the filaments don't
CROSS-BRIDGE CYCLE	Myosin heads bind actin → power-stroke → detach (ATP) → re-cock → repeat
THE SWITCH	Ca^{2+} binds troponin → tropomyosin moves → myosin can grip actin
E-C COUPLING	AP → T-tubules → SR releases Ca^{2+} → contract; pump Ca^{2+} back → relax
CLINICAL	Malignant hyperthermia, milk fever, equine 'tying-up', white muscle disease

01 The three muscle types

- **Skeletal** — striated, **voluntary**, each fibre has its own motor nerve; ~40% of body mass; the model for how contraction works.
- **Cardiac** — striated, involuntary, self-triggering; cells joined by intercalated discs (gap junctions); uses **calcium-induced calcium release**; long AP → **cannot tetanise**.
- **Smooth** — non-striated, involuntary; no sarcomeres, no troponin; slow & sustained; **calmodulin**-driven.

	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 — Same sliding-filament machine, three control schemes.

02 Skeletal muscle structure

- **Hierarchy:** muscle belly → fascicle → **fibre** (multinucleate cell) → myofibril → **sarcomere** (contractile unit, Z-disk to Z-disk).
- **Filaments:** thin = **actin** (anchored to Z-disks); thick = **myosin** (~500 heads, centre); **titin** = elastic spring setting resting length.
- **Bands:** I (actin only) · A (thick filament) · H (myosin only) · M line (centre); Z-disks = the borders.
- **Ca^{2+} machinery:** the **sarcoplasmic reticulum (SR)** stores Ca^{2+}; **T-tubules** carry the AP deep into the fibre to the SR.

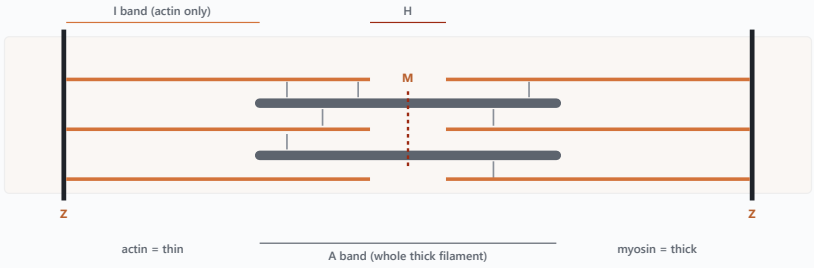


Fig 2 — The sarcomere: actin + myosin, the bands, the M line.

03 Sliding-filament + cross-bridge cycle

- **Sliding-filament:** filaments **slide** and overlap more → the sarcomere shortens; **each filament's length is unchanged**.
- **Cross-bridge cycle:** ATP→ADP+Pi **cocks** the head → head **binds** actin → Pi released → **power stroke** (~10 nm) → new ATP → head **detaches** → repeat.
- **ATP is needed to let go** too → after death, no ATP = heads locked = **rigor mortis**.

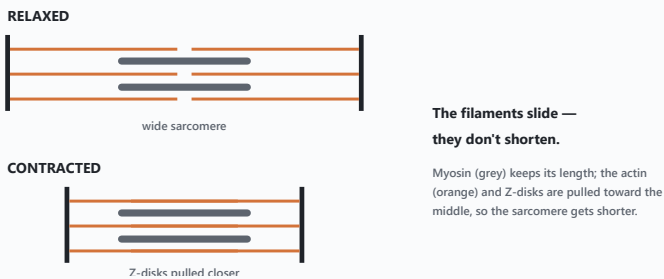
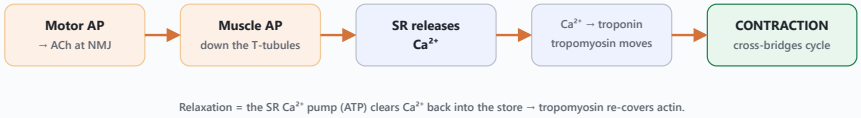


Fig 3 — Filaments slide; the sarcomere shortens.

04 Excitation-contraction coupling (the Ca^{2+} switch)

- At rest, **tropomyosin** (held by **troponin**) covers the myosin-binding sites on actin.
- **Ca^{2+} is the switch:** Ca^{2+} binds troponin → tropomyosin moves → myosin binds actin → contract. Remove Ca^{2+} → relax.
- **Chain:** motor AP → ACh at the NMJ (1:1) → muscle AP → down the **T-tubules** → SR **ryanodine receptors** release Ca^{2+} (mechanically coupled to the T-tubule voltage sensor in skeletal muscle).
- **Relaxation:** ATP-driven SR pumps clear Ca^{2+} back → tropomyosin re-covers actin.



Relaxation = the SR Ca^{2+} pump (ATP) clears Ca^{2+} back into the store → tropomyosin re-covers actin.

Fig 4 — Nerve → T-tubule → SR Ca^{2+} → contraction; pump Ca^{2+} back → relax.

05 Grading force + fibre types

- **Motor unit** = one motor neuron + its fibres. Force graded by **recruitment** (size principle: small units first) + **firing rate**.
- **Twitch** → **summation** → **tetanus** (fused, 3–5× a twitch). Force is best over a narrow **length range** (optimal overlap).

FIBRE	SPEED	FATIGUE	FUEL / COLOUR
Type I (slow)	Slow	Resistant	Oxidative · red
Type IIa (fast)	Fast	Moderate	Oxidative
Type IIx / IIb (fast)	Fastest	Fast	Glycolytic · white

- **Vet numbers:** greyhound ~**97%** fast-twitch, fast horse >90%, camel ~**30%** — speed vs stamina, visible in the animal.

06 Fuel & fatigue

- **ATP spent at 3 steps:** cock the heads · detach the heads · pump SR Ca^{2+}.
- **Fuel layers:** stored ATP (1–2 s) → **creatine phosphate** (~8–10 s) → glycogen (glycolytic + oxidative, s–h) → fat (prolonged). Efficiency ~25% → most energy = **heat** (shivering).

- **Fatigue** = a protective slowdown (↑ inorganic phosphate, ↓ SR Ca^{2+} release) — NOT simple ATP depletion (that would cause rigor).

07 Cardiac & smooth muscle

- **Cardiac:** striated; intercalated discs + gap junctions (syncytium); **calcium-induced calcium release**; long AP → no tetanus; uses troponin.
- **Smooth:** no sarcomeres, dense bodies, **no troponin**; Ca^{2+} → **calmodulin** → **myosin light-chain kinase**; slow, wide length range, cheap **latch** hold; excited OR inhibited by autonomic nerves / hormones / stretch.

08 Clinical — where it breaks

- **Malignant hyperthermia: ryanodine-receptor** defect (halothane trigger) → uncontrolled SR Ca^{2+} → rigidity, soaring temperature, acidosis, muscle damage. **Rx: dantrolene**.
- **Milk fever (hypocalcaemia):** low Ca^{2+} weakens muscle + blocks the NMJ → the fresh cow goes down (paresis in ruminants; tetany in dogs). **Rx: slow IV calcium**.
- **"Tying-up" (exertional rhabdomyolysis):** muscle breakdown after work → stiff horse, **red-brown (myoglobin) urine**, renal risk. Rx: rest, fluids, monitor enzymes.
- **White muscle disease:** selenium / vitamin-E deficiency in lambs & calves → oxidative muscle damage that can strike the heart.

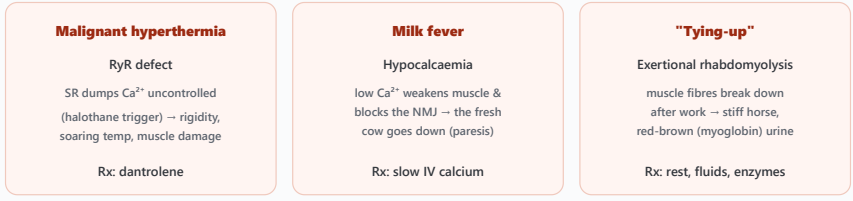


Fig 5 — Too much SR Ca^{2+} (MH), too little blood Ca^{2+} (milk fever), muscle breakdown (tying-up).

RED FLAG

Rigid + hyperthermic under anaesthesia (malignant hyperthermia), a down fresh cow (milk fever), or a stiff horse with red-brown urine (rhabdomyolysis) are muscle-physiology emergencies — act on calcium / temperature / fluids fast.

> *Every crisis here is this one mechanism failing at a point — too much SR calcium, too little blood calcium, or no fuel to relax.*
>
> — muscle physiology, after Cunningham 6e & PDA 3e.

KEY TERMS — QUICK GLOSSARY

Sarcomere	The contractile unit of striated muscle, from one Z-disk to the next — thick myosin overlapping thin actin.
Actin / myosin	The thin and thick filaments; myosin heads bind and pull the actin.
Troponin & tropomyosin	The Ca^{2+}-sensitive switch on the thin filament that hides or exposes the myosin-binding sites.
Sliding-filament theory	Contraction = filaments sliding to overlap more; each filament's length is unchanged.
Cross-bridge cycle	The repeating bind → power-stroke → detach → re-cock action of the myosin heads.
Excitation-contraction coupling	The chain linking a muscle action potential to SR Ca^{2+} release and contraction.
Sarcoplasmic reticulum (SR)	The intracellular Ca^{2+} store; releases Ca^{2+} to contract, pumps it back to relax.
Motor unit	One motor neuron plus every muscle fibre it innervates — the smallest unit of recruitment.
Tetanus	A smooth, maximal contraction from twitches fused at high stimulation frequency (3–5× a single twitch).
Ryanodine receptor (RyR)	The SR calcium-release channel; defective in malignant hyperthermia.

QUICK REVISION — REMEMBER THESE

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.

MEMORY AIDS

Ca^{2+} = GO — Calcium is the green light: Ca^{2+} out of the SR → troponin moves tropomyosin → contract. Ca^{2+} back in the SR → stop.

Slide, don't shrink — In the sliding-filament theory the filaments **slide** and overlap more — they don't get shorter; the **sarcomere** does.

ATP to let go — You need ATP not just to pull but to **release** the head — no ATP = stuck cross-bridges = rigor mortis.

TEST YOURSELF — ACTIVE RECALL

Cover the answers and try to retrieve each one from memory first — self-testing beats re-reading.

1. **In one sentence, how does a muscle shorten?**
2. **What starts and stops contraction at the molecular level?**
3. **Why does muscle need ATP even to relax?**
4. **Trace excitation-contraction coupling.**
5. **How does the nervous system make a muscle contract harder?**
6. **How does cardiac muscle differ from skeletal in triggering contraction?**
7. **Smooth muscle has no troponin — what does it use instead?**
8. **What is malignant hyperthermia?**

ANSWERS

1. Myosin heads repeatedly bind and pull the actin thin filaments, sliding them over the thick filaments so each sarcomere shortens — the filaments themselves stay the same length.
2. Ca^{2+} starts it (binds troponin → tropomyosin moves → myosin binds actin); removing Ca^{2+} — pumped back into the SR — stops it.
3. ATP binding to the myosin head is what detaches it from actin; with no ATP the heads stay locked (rigor mortis), and the SR Ca^{2+} pump also runs on ATP.
4. Motor-neuron AP → ACh at the NMJ → muscle AP → down the T-tubules → SR releases Ca^{2+} → Ca^{2+} binds troponin → cross-bridges cycle → contraction; Ca^{2+} pumped back → relaxation.
5. By recruiting more (and larger) motor units and by firing faster, so twitches summate toward tetanus.
6. It uses calcium-induced calcium release (entering Ca^{2+} triggers SR release), is electrically coupled at intercalated discs, and its long refractory period prevents tetanus.
7. Ca^{2+} binds calmodulin, which activates myosin light-chain kinase to switch the myosin head on; it can also hold force cheaply in a 'latch' state.
8. A defective ryanodine receptor releases SR Ca^{2+} uncontrollably (often triggered by halothane), causing rigidity, a soaring temperature and muscle damage; treat with dantrolene.

WHEN TO REFER OR ESCALATE

- 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 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.

SOURCES

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.
2. Sjaastad ØV, Sand O, Hove K. Physiology of Domestic Animals. 3rd ed. Oslo: Scandinavian Veterinary Press; 2016. Chapter 8 — Muscles (pp 325–362): muscle structure, contraction mechanics, fibre types and species differences, energy metabolism and fatigue, smooth and cardiac muscle.
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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