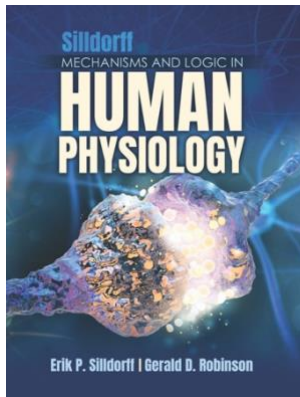


# Chapter 7: Muscle Physiology

## Skeletal, Cardiac, and Smooth Muscle, Instructor Guide

Mechanisms and Logic in Human Physiology | Silldorff & Robinson | Kendall Hunt

*Format: Left column = instructor narrative in the author's voice. Right column = structured causal chains, key distinctions, common student errors (red), and prediction prompts (gold italic). The chapter is organized around a single mechanism: a muscle cell harnesses ATP energy into directed force by cycling crossbridges between overlapping filaments. Every section does one of three things, it shows how that mechanism is switched on, how the tiny pull of each crossbridge is summed and conveyed out of the cell onto bone, or how the same machinery is re-tuned for a different job. Cardiac muscle is introduced here; its detailed treatment is developed in Chapter 9.*



Companion to **Mechanisms and Logic in Human Physiology** by Erik P. Silldorff and Gerald D. Robinson (Kendall Hunt Publishing). The half-year and full-year web subscriptions and the print version are available at <https://he.kendallhunt.com/product/mechanisms-and-logic-human-physiology-2>. For those teaching A&P more classically in a 2-semester sequence, the book is also packaged with Visible Body to support the anatomical portions of those courses (365-day subscriptions): <https://he.kendallhunt.com/product/mechanisms-and-logic-human-anatomy-and-physiology-visible-body-1>.

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### Section 1: Muscle as the Body's Force Specialist — One Mechanism Behind Every Movement

| Instructor Narrative                                                                                                                                                                                                                                                                                     | Structured Outline / Causal Chain                                                                                                |
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| <b>The move:</b> <i>Open not with a list of muscle facts. State one claim and hold it: a muscle cell is a machine that employs energy from ATP to generate directed force, and it does the job with one mechanism repeated billions of times. All of muscle physiology revolves around this process.</i> | <b>The Singular Fundamental Mechanism</b> Muscle = a cell that uses energy derived from ATP to produce directed mechanical force |

## Only muscle generates force that moves other things

Plenty of cells produce force, but in tiny increments. A white blood cell crawls, cilia beat, the cytoskeleton drags vesicles around inside a cell. What sets muscle apart is scale and purpose. This is the one tissue adapted to convert the chemical energy stored in ATP into mechanical force large enough to move the whole body, or to drive blood, air, food, and urine along the tubes that carry them. Frame muscle this way and the three muscle types stop looking like three separate memorization exercises. These are three solutions to one engineering problem: force on demand, each tuned to a different job.

Keep this purpose in mind because it predicts the functional hierarchy before even a single protein is named. Any cell charged with generating force needs to solve three problems in sequence. It must switch the force-making machinery on at the exact instant, it must generate that force in such a way that it can be dialed from gentle to maximal, and it must get that internally-generated force out of the cell and onto something rigid, in the case of skeletal muscle, bone.

## One repeating mechanism: filaments that pull past each other

Here is the single concept that unifies skeletal, cardiac, and smooth muscle. Inside every muscle cell, two sets of protein filaments overlap, and on stimulation the thick filaments grab the thin filaments and pull them past, spending ATP, so the overlapping region shortens. This is the sliding filament theory. If there is no filament overlap no force can be generated. Notice what the cell does *not* do: the filaments themselves do not shorten. They keep their length and slide, like two hands hauling a rope past one another.

Because filament pull is nothing more than the sum of countless tiny molecular interactions, grading force becomes simple in principle. If greater force is required, more filaments must interact at the same moment. We'll find later that this reality explains wave summation, the length-tension relation, fiber diameter effects, and motor-unit recruitment. Different levers, identical outcome: change the number of crossbridges working at once.

- Sliding filament theory: thick filaments pull thin filaments past them; filaments slide, they do not shorten
- No zone of overlap, no crossbridges, no tension
- Force is graded by changing the *number* of crossbridges working at once

## The Three Problems Every Muscle Solves

- Switch on: couple a stimulus to the contractile machinery
- Generate and grade force: sum many tiny filament pulls
- Transmit force: convey internal tension out of the cell onto bone

## Three Tissues, One Logic

- Skeletal: voluntary, neurogenic trigger, pulls on the skeleton
- Cardiac: involuntary, myogenic trigger, pushes blood (detail in Ch. 9)
- Smooth: involuntary, force on the walls of hollow organs

## Causal Chain: Why Training Makes a Muscle Stronger

Repeated high-force use mechanically loads the fiber → that load signals increased protein synthesis → more contractile protein is synthesized → more thick and thin filaments are now present per fiber (greater cross-sectional area) → more crossbridges can form simultaneously → greater maximal force

## Common Errors

- **Error:** Thinking that filaments themselves shorten. They keep their length and simply slide past one another.

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|  | <ul style="list-style-type: none"> <li>• <b>Error:</b> Treating skeletal, cardiac, and smooth as unrelated. They are one contractile mechanism tuned to three jobs.</li> <li>• <b>Error:</b> Assuming force per cell is fixed. Force scales with the number of active crossbridges.</li> </ul> <p><b>Prediction Prompt</b></p> <ul style="list-style-type: none"> <li>• <b>Predict:</b> Predict the effect on maximal force if a drug has blocked half of a fiber's myosin heads so they can't bind actin. Justify your response using crossbridge number.</li> <li>• <b>Predict:</b> A muscle is stretched until its thick and thin filaments no longer overlap. Predict the active force it can generate and explain why.</li> </ul> |
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## Section 2: Whole-Muscle Architecture — Fascicles, Fascia, and the Path Force Must Travel

| Instructor Narrative                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    | Structured Outline / Causal Chain                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |
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| <p><b>The move:</b> Teach the force transmission sequence as a force-delivery route, not a vocabulary list. Ask your students this: the contractile proteins are locked inside cells, yet the load is a bone outside every cell, so how does the pull get from inside to outside? Every connective-tissue layer plays a part in the answer.</p> <p><b>A muscle is a bundle of bundles, wrapped at every level</b></p> <p>Start at the outside and work in. A whole muscle attaches to bone at each end through a tendon. The meaty middle of the muscle, the belly, is packed with cells, also called muscle fibers because they are long and thread-like. Those fibers are gathered into bundles called fascicles, and each fascicle, each fiber, and the entire muscle is sheathed in its own layer of connective tissue, collectively the fascia. About fifteen percent of a typical muscle is composed of this connective tissue, and it is not</p> | <p><b>The Nested Wrapping (Outside In)</b></p> <ul style="list-style-type: none"> <li>• Whole muscle (wrapped) → fascicles (wrapped) → fibers (wrapped)</li> <li>• All fascial layers are continuous and funnel into the tendon</li> <li>• ~15% of muscle mass is connective tissue, the force cabling</li> </ul> <p><b>Why Continuity Matters</b></p> <ul style="list-style-type: none"> <li>• Contractile proteins are intracellular; the load (bone) is extracellular</li> <li>• Continuous fascia, tendon → a single route for force out of every cell</li> </ul> |

exclusively packing material. It serves as the cabling that will deliver force to the tendon.

The key structural purpose here is to provide continuity. The wrapping layers just mentioned do not stop at the ends of the muscle. They funnel together and become continuous with the tendon itself. A tendon, then, is not “glued” like a cap onto the end of the muscle. Its fibers run on *into* the muscle as the fascial sheaths, making it integral to the muscle. It’s this continuity that allows force generated deep inside thousands of separate muscle cells to end up pulling on one shared bone.

### Fiber arrangement trades range of motion against force

Skeletal muscles come in two broad fiber layouts, and that arrangement is very much adaptive. In fusiform (parallel) muscles the fibers run the long axis of the muscle, so each fiber is long, the muscle shortens dramatically, and the result is a large range of motion. In pennate muscles the fibers attach at an angle to a central tendon, like the barbs of a feather. Angling of these fibers permits packing many more, shorter fibers into the same mass, substantially increasing the total cross-sectional area.

Cross-sectional area is a key variable setting maximal force generated, so pennate muscles are much stronger than fusiform muscles of the same size. The tradeoff is range and speed: because the shorter fibers pull at an angle, the muscle shortens less. This is exactly why large load-moving muscles, like the gluteals that lift you out of a chair, tend to be pennate. Muscles involved in sweeping a limb through a wide arc are generally fusiform. Form is matched to the job at the level of fiber geometry.

- Tendon fibers run *into* the muscle; they are not merely attached at the end

### Key Distinction: Fusiform vs. Pennate

- Fusiform (parallel): long fibers, more shortening, greater range of motion, less force generated
- Pennate (angled): more, shorter fibers, greater cross-sectional area, less range, more force produced
- Cross-sectional area, not muscle length, sets maximal force

### Derivation Chain: Why Pennate Muscles Are Stronger

Angled fibers pack more, shorter fibers per unit mass → greater physiological cross-sectional area (more myofilaments now lying in parallel) → more crossbridges can form at once → greater force generated

### Common Errors

- **Error:** Thinking the tendon is just stuck to the muscle's end. Its fibers are continuous with the internal fascia.
- **Error:** Assuming a longer muscle is automatically a stronger one. Force tracks cross-sectional area; length governs distance shortened.
- **Error:** Treating fascia as inert packaging. Fascia is the load-bearing path from cell to bone.

### Prediction Prompt

- **Predict:** Predict why a bipennate muscle can be stronger than a unipennate muscle of the same mass.
- **Predict:** Compartment syndrome swells a muscle inside its tight fascia. Predict the consequence for the capillaries threading the muscle and for the tissue.

## Section 3: The Muscle Fiber — A Giant Cell Wired for Deep, Synchronous Stimulation

| Instructor Narrative                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 | Structured Outline / Causal Chain                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      |
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| <p><i><b>The move:</b> Establish the size problem first. This cell can be a foot long and packed solid with contractile machinery, yet stimulation arrives only at the surface. The T-tubule and SR layout is the cell's answer to getting one signal everywhere almost at once. Teach that the structural layout provides that solution.</i></p> <p><b>Skeletal muscle fibers are giant cells built by myoblast fusion</b></p> <p>A skeletal muscle fiber is enormous, up to a hundred micrometers across and often more than ten centimeters long. It possesses hundreds of nuclei and thousands of mitochondria and grew that way via fusion. During development many embryonic myoblasts merged into one giant cell and there's adaptive logic to that. Generating force takes billions of copies of the contractile proteins, so just one nucleus could never transcribe and distribute protein across a cell this size. Consequently, many nuclei, scattered along that one fiber, keep the protein supply local. Leftover unfused myoblasts persist as satellite cells, the repair and growth reserve that lets adult muscle regenerate even though mature fibers do not divide.</p> <p>The fiber's membrane has its own name, sarcolemma, and it behaves like an axon membrane. It's replete with voltage-gated channels, so it can readily fire and propagate action potentials. A somatic motor neuron synapses with the sarcolemma near its middle (at the neuromuscular junction or NMJ), and the impulse then races toward both ends of the fiber at once. This last detail is functionally significant: firing from the center outward is how the cell triggers all of its sarcomeres in synchrony rather than in a slow wave from one end to the other.</p> <p><b>T-tubules and the SR solve the deep-delivery problem</b></p> <p>Here is a problem solved by myofiber architecture. The action potential is initiated at the cell surface, but the contractile machinery fills the whole interior. If electrical stimulation remained only at the surface, the core of this large fiber would never receive the signal. The solution is the transverse tubule, a deep invagination of the sarcolemma that carries the action potential down into the fiber, perpendicular to its length. Because</p> | <p><b>Why the Fiber Is Built the Way it is</b></p> <ul style="list-style-type: none"> <li>• Myoblast fusion builds one huge fiber → many nuclei distributed along its length → protein synthesis stays local to each region</li> <li>• Satellite cells = leftover myoblasts, the repair/growth reserve</li> <li>• Sarcolemma is excitable; NMJ sits mid-fiber → AP fires toward both ends</li> </ul> <p><b>The Deep-Delivery Apparatus</b></p> <ul style="list-style-type: none"> <li>• T-tubule: invagination of sarcolemma; carries the AP deep into the fiber</li> <li>• SR: stores and releases <math>\text{Ca}^{2+}</math>; SERCA pump refills the SR continuously</li> <li>• Triad = one T-tubule + two SR terminal cisternae (signal meets <math>\text{Ca}^{2+}</math> store)</li> </ul> <p><b>Causal Chain: Why Surface Excitation Reaches the Core</b></p> <p>AP on sarcolemma at the NMJ → propagates along surface and down the T-tubules → T-tubule depolarization reaches the triad deep in the fiber → depolarization there opens the SR release channels (mechanism detailed in Section 9) → adjacent SR releases <math>\text{Ca}^{2+}</math> throughout the cell → all sarcomeres activate in synchrony</p> <p><b>Common Errors</b></p> <ul style="list-style-type: none"> <li>• <b>Error:</b> Picturing the T-tubule lumen as cytoplasm. This lumen is continuous with extracellular fluid, so it's separate from the sarcoplasm and filled with ECF</li> <li>• <b>Error:</b> Thinking the SR "makes" calcium. <math>\text{Ca}^{2+}</math> is simply stored in the SR and released during activation. After this, the <math>\text{Ca}^{2+}</math> is returned to the SR via SERCA pumping.</li> </ul> |

T-tubule membrane also has voltage-gated channels, the impulse travels deep into the cell's interior.

Running alongside T-tubules is the sarcoplasmic reticulum (SR), the muscle cell's specialized endoplasmic reticulum, whose one job is to store  $\text{Ca}^{2+}$  and release it on cue. Since any single T-tubule is flanked by two expanded SR terminal cisternae in close apposition, the trio is called a triad. This arrangement places the surface-borne electrical signal directly next to the  $\text{Ca}^{2+}$  release site. A SERCA pump in the SR membrane runs continuously to quickly transport the  $\text{Ca}^{2+}$  back inside, ending activation, and concluding the so-called calcium surge. This event and the contraction it triggers, is brief. Later on we'll see how that warrants very careful consideration.

- **Error:** Assuming the AP starts at one end of the fiber. It actually begins mid-fiber at the NMJ and is simultaneously conducted toward both ends of the fiber.

#### Prediction Prompt

- **Predict:** Myotubular myopathy malforms T-tubules. Predict the effect on the fiber's ability to contract and explain the mechanism.
- **Predict:** Predict what happens to relaxation speed if SERCA pumps are partly inhibited, and why.

## Section 4: The Sarcomere — Building a Functionally Important Zone of Overlap

| Instructor Narrative                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          | Structured Outline / Causal Chain                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            |
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| <p><b>The move:</b> Build the sarcomere on the board to show that it satisfies a very critical requirement: there must exist a region where thick and thin filaments overlap because mechanical force can only be generated there. Identify all proteins that either contribute to or protect that zone.</p> <p><b>The contractile unit is a repeating, end-to-end machine</b></p> <p>Inside the fiber, the contractile proteins are organized into myofibrils, long cylindrical bundles divided up into sarcomeres, repeating block-like structures that are laid end-to-end and run the length of the cell. The sarcomere is the functional contractile unit. It's made up of partially overlapping thick and thin filaments and extends from one Z-disc to the next. Each sarcomere shortens only about a millionth of a meter, which sounds trivial, but a single myofibril can hold a hundred thousand sarcomeres in series. That means collective shortening of these structures adds up to centimeters across a whole muscle. Understanding one sarcomere means understanding them all; contraction itself is just</p> | <p><b>Anatomy of One Sarcomere</b></p> <ul style="list-style-type: none"> <li>• Sarcomere = Z-disc to Z-disc, the contractile unit</li> <li>• Thick filament (myosin) central; thin filament (actin) anchored at Z-discs</li> <li>• Zone of overlap = the only site of tension generation</li> <li>• A-band = thick-filament length (dark); I-band = thin only (light)</li> </ul> <p><b>Who Holds What in Place</b></p> <ul style="list-style-type: none"> <li>• M-line proteins suspend thick filaments (like pencils penetrating through a cardboard disc)</li> <li>• Z-disc proteins anchor thin filaments</li> <li>• Nebulin: thin-filament molecular ruler (length)</li> <li>• Titin: M-line to Z-disc spring; centers thick filament, stores elastic recoil</li> </ul> |

repetition and summation as the thin filaments slide toward the centers of sarcomeres over stationary thick filaments.

The two filaments have distinct jobs and positions. Thick filaments, made of myosin, cluster in the center of the sarcomere. Thin filaments, built mainly of actin, anchor at the Z-discs and project inward from both sides. Where these filaments overlap, A-bands are visible and crossbridges can form and it's here that tension is generated. The striations (alternating dark and light stripes) seen under the microscope, are just the repeating alternation of the dense A-bands (same length as each sarcomere's aggregation of thick filaments) and the lighter I-bands (thin filaments only with no myosin overlap and extending to the Z-discs).

### Scaffolding proteins secure the filaments in proper position

Soft protein filaments cannot just float in position, so structural proteins are needed to hold the array in space. M-line proteins suspend the thick filaments at their centers. Think of thick filaments as like pencils pushed through a sheet of cardboard, the M-line, which holds these filaments parallel and spaces them so thin filaments can interdigitate in between. Z-disc proteins do the same job for the thin filaments at the sarcomere ends. Keeping the two filament sets suspended and separate is what makes consistent overlap, and therefore force, possible.

Two molecular rulers finish the picture, and one of them warrants extra attention. Nebulin runs along the thin filament and helps set its length. Titin, the largest protein in the body, spans from the M-line to the Z-disc along the thick filament, keeping it centered and adding a spring-like elastic region. That spring action is important: a stretched sarcomere recoils partly because titin was stretched, and titin's elasticity is the molecular basis of the passive tension we will visit in relation to the length-tension relationship and again in our discussion of eccentric contractions.

### Key Distinction: Contractile vs. Structural

- Contractile (myosin, actin): generate the pulling force
- Regulatory (troponin, tropomyosin): controls when crossbridges form and contraction is initiated
- Structural (M-line, Z-disc, titin, nebulin): hold the geometry that makes overlap possible

### Derivation Chain: Why a Train of Tiny Shortenings Adds Up to Significant Shortening

Sarcomeres sit end-to-end in series along each myofibril  
→ each shortens ~1 micrometer → the shortenings add along the fiber → whole fiber, and muscle, shortens by centimeters

### Common Errors

- **Error:** Thinking the whole myofibril is the contractile unit. The unit is the sarcomere; myofibrils are sarcomeres in series.
- **Error:** Thinking tension is made all along the filaments. It is made only in the zone of overlap.
- **Error:** Treating titin as mere scaffolding. It is also the elastic spring behind passive and recoil tension.

### Prediction Prompt

- **Predict:** A biceps fiber can shorten 10 cm and each sarcomere shortens 1 micrometer. Predict how many sarcomeres lie in series along one myofibril.
- **Predict:** Predict what happens to a fiber's recoil to resting length if its titin springs were cut, and explain.

## Section 5: Contractile Proteins — Myosin, Actin, and the Crossbridge Engine

## Instructor Narrative

***The move:** Muscle contraction can be better understood via a car-and-driver analogy. The thick and thin filaments are like an engine. Think of myosin and actin as being built around two functional sites on the myosin head, an ATPase and an actin-binding site, with contraction taking place because of those two sites doing their jobs in a repeating cycle.*

### Myosin is a polymer of two-headed molecules, heads facing out

A thick filament is a polymer of several hundred myosin molecules. Each myosin molecule has a long tail and two heads. A golf club analogy may be useful here: take a bundle of golf clubs, point all the handles toward the center, and let the heads stick out at both ends. The tails form the backbone, the heads project outward toward the surrounding thin filaments. There is a bare central zone (the H-zone) with no heads and an M-line at the very center. Each head has an ATPase molecule that hydrolyzes ATP for energy, and also a binding site for actin.

Those two sites are the car's engine. The head uses ATP hydrolysis to cock itself like a loaded spring, binds actin, then releases the stored energy in a power stroke that drags the thin filament toward the center of the sarcomere. Because each crossbridge contributes a fixed small amount of force, total tension is, once again, just a count of how many crossbridges are formed and cycling at that instant. Myosin also comes in several isoforms with different cycling speeds, which is the molecular root of the fast-versus-slow fiber distinction and of power output that will be discussed later.

### Actin is a twisted strand of myosin head binding sites

The thin filament is built mainly of actin. Globular G-actin monomers link into a strand of filamentous F-actin, and two F-actin strands twist around each other. The F-actin strands look like two strings of pearls (the G-actin molecules), wound together into a helix. Each G-actin pearl carries a myosin-binding site, so the thin filament is essentially a long row of attachment points for the myosin heads.

Myosin and actin have a strong chemical affinity, which the cell must actively manage. Left to themselves these molecules would tightly bind,

## Structured Outline / Causal Chain

### Myosin (Thick Filament)

- Polymer of ~ hundreds of myosin molecules; tails in, heads out (like a bundle of golf-clubs)
- Each head = ATPase site + actin-binding site
- Bare central zone at the M-line (no heads)
- Isoforms differ in cycling speed → fast vs. slow fibers, power

### Actin (Thin Filament)

- G-actin monomers → F-actin strand; two strands twist (pearl necklace)
- Each G-actin carries one myosin-binding site
- Plus regulatory proteins troponin and tropomyosin (next section)

### Key Distinction: Force vs. Power

- Force per crossbridge is fixed; total force = number of crossbridges
- Power adds speed: faster myosin ATPase → faster cycling → more power at a given load
- Two equal-size fibers can match force yet differ in power (ATPase isoform)

### Causal Chain: Why Rigor Mortis Happens

Death stops ATP production → no fresh ATP binds the myosin head (ATP binding is what releases a head from actin) → heads already cross-bridged cannot detach → crossbridges stay locked → rigid muscle until proteins degrade

### Common Errors

- **Error:** Thinking the myosin head has just one job. It has two: ATPase activity and possessing an actin binding site.

the cell would lock into permanent contraction, that's what actually occurs in rigor mortis. Accordingly, the thin filament also carries regulatory proteins that keep binding switched off until the cell is stimulated. In other words, the cell employs a special molecular mechanism to keep it relaxed while still in the resting state.

- **Error:** Believing myosin and actin need some form of “coaxing” to bind. They bind strongly and spontaneously; at rest, the cell works to keep them apart.
- **Error:** Equating force with power. Force is crossbridge number; power adds shortening speed (mediated by an ATPase isoform).

#### Prediction Prompt

- **Predict:** A mutation lowers the myosin head's binding affinity for actin. Predict the effect on force and the likely symptom.
- **Predict:** Predict why a fiber expressing a faster myosin ATPase generates more power than an equal-size slow fiber lifting the same load.

## Section 6: Regulatory Proteins — Why Resting Muscle Stays Off

### Instructor Narrative

**The move:** Teach relaxation as an active, defended state, not the absence of activity. The default for myosin and actin is to bind; tropomyosin is like a bouncer standing in the doorway. Release of  $Ca^{2+}$  from the SR and its subsequent protein binding will pull that bouncer aside. Excitation-contraction coupling now follows directly.

#### Tropomyosin physically blocks actin's myosin binding sites

Because myosin and actin bind avidly, an unstimulated muscle would contract on its own without some kind of inhibiting action. That inhibition is provided sterically by tropomyosin, a long string-like protein that lies in the groove of the thin filament directly over the myosin-binding sites on G-actin. Put simply, tropomyosin is in the way of myosin binding to actin under resting conditions. While tropomyosin is in that configuration, myosin heads cannot reach their binding sites, no crossbridges form, and

### Structured Outline / Causal Chain

#### The Two Regulatory Proteins

- Tropomyosin: string-like protein blocking myosin-binding sites (steric inhibition)
- Troponin: rides on tropomyosin; one subunit binds  $Ca^{2+}$  (the sensor/switch)
- Together they keep resting muscle off by default

#### The Strict Activation Order

- $Ca^{2+}$  rises → binds troponin → troponin changes shape
- Tropomyosin dragged off the binding sites → sites exposed
- With ATP present, crossbridges form and cycle

no sliding filament action is taking place. Relaxation is something the cell *maintains*, not something that just happens.

Sitting on top of tropomyosin is a second regulatory protein, troponin, and troponin acts as a  $\text{Ca}^{2+}$  sensor. It's a complex of subunits, one of which (troponin-C) binds  $\text{Ca}^{2+}$ . When intracellular  $\text{Ca}^{2+}$  is low, the troponin complex holds tropomyosin in its blocking position. The two proteins, then, act as a linked unit: troponin as the switch, tropomyosin as the gate it controls.

### Calcium is the single trigger that lifts the block

When the myofiber is stimulated,  $\text{Ca}^{2+}$  floods the sarcoplasm and binds troponin. That binding changes troponin's shape, and because troponin is tied to tropomyosin, it drags tropomyosin out of the groove, off the myosin-binding sites. Now those sites are exposed, and if ATP is present the myosin heads start their repetitive bind-pull-release cycle. This sequence has a highly predictive order:  $\text{Ca}^{2+}$  is released from the SR,  $\text{Ca}^{2+}$  binds to troponin-C, troponin shape change occurs, tropomyosin moves so it no longer blocks the myosin binding sites on actin, then crossbridges form. Note that  $\text{Ca}^{2+}$ -troponin binding is highly random, each event occurs independently of other like events. This contributes to smoothness of muscle contractions even at low tension. Relaxation is achieved by the same steps, but run in reverse: Stimulus stops, SERCA pumps lower sarcoplasmic  $\text{Ca}^{2+}$  by transporting it back into the SR,  $\text{Ca}^{2+}$  diffuses off troponin and troponin regains its resting shape, troponin lets tropomyosin slide back over the binding sites, and crossbridge formation is once again blocked. This complete sequence is reminiscent of how the throttle and brake are used when driving a car:  $\text{Ca}^{2+}$  binding troponin and dragging tropomyosin aside acts as the throttle, and SERCA pulling  $\text{Ca}^{2+}$  back into the SR functions as the brake.

### Key Distinction: Throttle vs. Brake

- Throttle:  $\text{Ca}^{2+} \rightarrow$  troponin  $\rightarrow$  tropomyosin moves  $\rightarrow$  crossbridges allowed
- Brake: SERCA lowers  $\text{Ca}^{2+} \rightarrow$  troponin releases it  $\rightarrow$  tropomyosin re-blocks
- Relaxation is active (it costs ATP at SERCA), not passive

### Causal Chain: How a Calcium Surge Turns On Contraction

Sarcoplasmic  $\text{Ca}^{2+}$  rises  $\sim 10$ -fold  $\rightarrow$   $\text{Ca}^{2+}$  binds troponin  $\rightarrow$  troponin conformational change shifts tropomyosin  $\rightarrow$  myosin-binding sites on G-actin exposed  $\rightarrow$  crossbridge cycling begins (ATP permitting)

### Common Errors

- **Error:** Thinking relaxation is just the absence of stimulus. It is actively maintained by tropomyosin blocking myosin binding sites and SERCA pumping action.
- **Error:** Believing that  $\text{Ca}^{2+}$  binds tropomyosin.  $\text{Ca}^{2+}$  binds *troponin*, which then moves tropomyosin.
- **Error:** Scrambling the step order. It's  $\text{Ca}^{2+} \rightarrow$  troponin  $\rightarrow$  tropomyosin  $\rightarrow$  crossbridges, never the reverse.

### Prediction Prompt

- **Predict:** Predict the contractile state of a fiber whose troponin cannot bind  $\text{Ca}^{2+}$ , and explain.
- **Predict:** Predict what happens to relaxation if  $\text{Ca}^{2+}$  cannot be cleared from the sarcoplasm, and identify which protein stays in the wrong position.

## Section 7: Structural Proteins and the Costamere — Conveying Tension Out of the Cell

*This section deserves extra time. The chapter and most courses move quickly past force transmission, but what happens here is the crux of the tissue: filaments generate tension inside a cell and do not directly touch bone, so the tension must be transferred externally to the fascia and then onward to the target.*

| Instructor Narrative                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 | Structured Outline / Causal Chain                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               |
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| <p><i>Sarcomeres generate tension inside myofibers, but that tension must be transferred externally (ultimately to the bone) for contractions to be meaningful. This is achieved, at least in part, by Z-discs relaying that tension to the sarcolemma via costameres. Make sure students understand that the transfer of internal tension to external tension is essential and that they know exactly how it takes place.</i></p> <p><b>Internal tension is useless until it crosses the membrane</b></p> <p>Everything discussed so far generates internal tension, force between sliding filaments inside the fiber. But the load is extracellular, at the bone. Contractile proteins never touch the tendon. So, how does tension generated inside thousands of separate cells get conveyed across the cell membranes and out to the shared connective tissue that pulls on bone? Without this transfer all the crossbridge cycling in the world moves nothing.</p> <p>Here's the sequence in a nutshell: sliding filaments pull the Z-discs, Z-disc proteins are tied to the intracellular cytoskeleton, the cytoskeleton links to dystrophin (a rod-shaped protein just under the membrane), dystrophin connects to transmembrane proteins that, on the outside, bind proteins of the extracellular matrix and fascia. It's through this path that tension is finally relayed to the tendon and bone. The joint complex spanning the Z-disc to the sarcolemma is called the costamere, and this is the route by which tension leaves the cell.</p> <p><b>Titin and nebulin keep the geometry; dystrophy illustrates what happens if cabling is compromised</b></p> <p>Two intracellular structural proteins keep the contractile geometry intact so that internal force can be produced. Titin spans the thick filament from M-line to Z-disc, centering it and adding a spring; nebulin runs the thin filament as a length ruler. Hold titin in mind, because its spring will reappear later as passive tension, a load-bearing element during lengthening contractions. These structural proteins build and preserve the</p> | <p><b>The Inside-to-Outside Chain</b></p> <ul style="list-style-type: none"> <li>• Sliding filaments pull the Z-disc</li> <li>• Z-disc → cytoskeleton → dystrophin (sub-membrane rod)</li> <li>• Dystrophin → transmembrane proteins → extracellular fascia</li> <li>• The Z-disc-to-sarcolemma complex = the costamere</li> </ul> <p><b>Why This Step Is Essential</b></p> <ul style="list-style-type: none"> <li>• Contractile proteins are intracellular; bone is extracellular</li> <li>• Internal tension must be transferred across the membrane to do external work</li> <li>• Continuous fascia then carries tension to tendon and bone</li> </ul> <p><b>Key Distinction: Generating Force vs. Transmitting Force</b></p> <ul style="list-style-type: none"> <li>• Contractile defect: less internal tension generated</li> <li>• Transmission defect (e.g., dystrophin pathology): internal tension normal at first, whole-muscle force fails</li> <li>• Two separable steps, two disparate failure modes</li> </ul> <p><b>Causal Chain: Why Duchenne Muscular Dystrophy Weakens Muscle</b></p> <p>Defective dystrophin (a transmission, not contractile, protein) → Z-disc force cannot be conveyed across the sarcolemma → whole-muscle force falls even though sarcomere force is initially normal; separately, the unsupported sarcolemma tears during contraction → fiber death, progressive loss of function</p> |

interdigitation without which no overlap could occur and, therefore, no force could be generated.

A clinical illustration that internal-to-external force transmission is a separate, essential step in muscle contraction is Duchenne muscular dystrophy (DMD). The defect here is in the costamere protein dystrophin. In DMD the sarcomeres still generate internal tension normally, yet the muscle is weak as a whole because the force cannot be conveyed across the membrane to the tendons. Over time the unsupported sarcolemma tears during contraction, fibers die, and function is lost progressively. That dissociation, normal internal tension but failing whole-muscle force, clearly demonstrates that making force and transmitting it are two different events.

#### Common Errors

- **Error:** Assuming force-making and force-transmission are the same. They are separate steps and can have separate potential failures.
- **Error:** Thinking dystrophin generates force. Dystrophin *conveys* force; its loss disables transmission, not contraction itself.
- **Error:** Forgetting the load is extracellular. The costamere is what conveys tension across the membrane.

#### Prediction Prompt

- **Predict:** Predict the early effect of dystrophin loss on (a) tension within a single sarcomere and (b) tension measured at the tendon; explain the difference.
- **Predict:** Predict why a costamere defect could cause weakness even when troponin, tropomyosin, myosin, and actin are all normal.

## Section 8: The Neuromuscular Junction — Engineering a Guaranteed Twitch

### Instructor Narrative

**The move:** Frame the NMJ as a specialized synapse with a large safety factor. Unlike a typical synapse that may or may not reach threshold, this one is built so that a single motor-neuron action potential unfailingly produces an action potential at the sarcolemma. Present each feature as a contributor to this reliability.

### One neuronal impulse must reliably trigger one muscle impulse

The neuromuscular junction is the synapse between a somatic motor neuron and the sarcolemma, sitting near the middle of the fiber. Its design

### Structured Outline / Causal Chain

#### Features That Guarantee a Twitch

- NMJ = motor neuron-to-sarcolemma synapse, mid-fiber
- Transmitter is always acetylcholine (ACh)
- Folded end-plate + many receptors + large ACh release → EPP overshoots threshold

#### Why the EPP Is $\text{Na}^+$ -mediated

- ACh opens nicotinic  $\text{Na}^+/\text{K}^+$  channels

goal is reliability. A normal central synapse contributes a small push that may or may not bring the next cell to threshold. The NMJ is the opposite: it is built so that a single motor neuron action potential is essentially guaranteed to trigger a muscle action potential. The motor neuron releases acetylcholine, always acetylcholine, onto a specialized, deeply folded patch of sarcolemma called the motor end-plate.

Acetylcholine binds nicotinic ACh-gated channels that pass both  $\text{Na}^+$  and  $\text{K}^+$ . The net current is strongly inward  $\text{Na}^+$  because the electrical driving force on sodium is large ( $E_{\text{Na}}$  is far from  $V_m$ ) while the driving force on potassium is tiny ( $E_{\text{K}}$  is near  $V_m$ ). An ion's driving force is simply the gap between the membrane voltage and that ion's equilibrium potential, which is why  $\text{Na}^+$ , sitting far from its own equilibrium, is pushed hard while  $\text{K}^+$ , near its own, is barely driven. The result is a large depolarization, the end-plate potential. A safety margin exists throughout this sequence: the folded membrane holds large numbers of receptors, and the terminal releases large amounts of transmitter, so the end-plate potential reliably overshoots threshold. This EPP results in opening of neighboring voltage-gated  $\text{Na}^+$  channels and launches a self-propagating sarcolemmal action potential toward both ends of the fiber.

### All-or-nothing twitches, and a fast off-switch

Because threshold is essentially always reached during transmission at the NMJ, the muscle fiber twitch is an all-or-nothing event. Its strength is not dependent on how strongly the motor neuron was stimulated, only that it was excited at least sufficiently to fire. Since one motor neuron branches to many myofibers, that neuron and all the fibers it controls constitute a motor unit. All the unit's fibers twitch together as a group. This is why we can later treat the motor unit, not any individual fiber, as the practical building block of whole-muscle force.

The NMJ also needs a clean off-switch. Acetylcholinesterase, bound at the synapse, degrades acetylcholine within milliseconds, ending the end-plate potential almost as fast as it began. That speed is what keeps twitches brief and, therefore, separable. The NMJ is also a favorite target for drugs and toxins: blocking the receptor (curare), destroy receptors (myasthenia gravis), blocking transmitter release (botulinum toxin), or

- Driving force large for  $\text{Na}^+$  ( $E_{\text{Na}}$  far from  $V_m$ ), tiny for  $\text{K}^+$  ( $E_{\text{K}}$  near  $V_m$ )
- Net inward  $g_{\text{Na}^+}$  → large depolarizing EPP → opens voltage-gated  $\text{Na}^+$  channels → sarcolemmal AP

### Key Distinction: Reliable NMJ vs. Graded Central Synapse

- Central synapse: one input may or may not reach threshold
- NMJ: one motor-neuron AP reliably yields one muscle AP
- Result: fiber twitch is all-or-nothing; whole motor unit fires together

### Causal Chain: From Motor Neuron AP to Muscle AP

Motor-neuron AP reaches terminal → ACh released, binds nicotinic receptors → net  $\text{Na}^+$  influx → end-plate potential (EPP) → EPP opens adjacent voltage-gated  $\text{Na}^+$  channels → sarcolemmal AP propagates toward both ends and down T-tubules

### Common Errors

- **Error:** Thinking  $\text{K}^+$  flux drives the EPP. Net current is inward  $\text{Na}^+$ ;  $\text{K}^+$  contributes little ( $E_{\text{K}}$  near  $V_m$ ).
- **Error:** Believing twitch strength depends on stimulus size. The fiber twitch is all-or-nothing.
- **Error:** Forgetting acetylcholinesterase. Without this fast off-switch, contraction would not end cleanly.

### Prediction Prompt

- **Predict:** Curare blocks ACh receptors without opening them. Predict the effect on the EPP, the muscle AP, and breathing.
- **Predict:** An acetylcholinesterase inhibitor (nerve agent) is absorbed. Predict the effect on end-plate potential duration and on muscle contraction.

inhibiting the esterase (nerve agents) results in predictable, and sometimes lethal, paralysis or spasm.

## Section 9: Excitation–Contraction Coupling — From Sarcolemmal Voltage to Released Calcium

### Instructor Narrative

**The move:** Consider the events of ECC as a sequence, not a list to be memorized. The idea here is to order events so each one triggers the next just like dominos in a line falling into each other one after another. Try putting the eleven steps on the board scrambled and have students reorder them by logic alone.

### The first task is to convert a membrane voltage into a $\text{Ca}^{2+}$ signal

Excitation-contraction coupling is the named bridge between an electrical event, the action potential, and a mechanical one, contraction. The deliverable is  $\text{Ca}^{2+}$ . The sarcolemmal action potential, already running deep into the fiber along the T-tubules, has to be transduced into a flood of sarcoplasmic  $\text{Ca}^{2+}$  at the right place and time. The triad geometry is what makes this possible: a T-tubule impulse will be carried directly between two adjacent terminal cisternae of the SR - the SR's  $\text{Ca}^{2+}$  storage depot.

The electrical impulse to  $\text{Ca}^{2+}$  release succession is facilitated through actions of two linked triad proteins. Residing in the T-tubule membrane is the dihydropyridine receptor, which here acts purely as a voltage sensor. This protein is mechanically coupled to the ryanodine receptor, a  $\text{Ca}^{2+}$  channel in the SR membrane. When the T-tubule depolarizes, the voltage sensor changes shape, that shape change opens the ryanodine receptor, and  $\text{Ca}^{2+}$  escapes the SR into the sarcoplasm, causing a tenfold increase in intracellular free  $\text{Ca}^{2+}$ . The direct mechanical connection between these two receptors is how the impulse -  $\text{Ca}^{2+}$  release succession is handled in skeletal muscle; cardiac muscle, instead, lets  $\text{Ca}^{2+}$  enter the sarcoplasm directly through the DHPR to trigger even more substantial

### Structured Outline / Causal Chain

#### The Two Coupling Proteins at the Triad

- DHPR in the T-tubule membrane: voltage sensor (in skeletal muscle, not a  $\text{Ca}^{2+}$  source itself)
- Ryanodine receptor in SR membrane: the  $\text{Ca}^{2+}$  release channel
- DHPR is mechanically linked to RyR; voltage opens calcium release

#### The Deliverable

- Sarcoplasmic free  $\text{Ca}^{2+}$  rises ~10-fold
- Enough to bind nearly all troponin
- Tropomyosin shifts → crossbridges permitted

#### Causal Chain: The Ordered ECC Sequence

Motor-neuron AP → ACh release at NMJ → ACh binds nicotinic receptors →  $\text{Na}^+$  influx → EPP → sarcolemmal AP down the T-tubules → DHPR voltage-sensor shape change → ryanodine receptor opens →  $\text{Ca}^{2+}$  floods sarcoplasm →  $\text{Ca}^{2+}$  binds troponin → tropomyosin moves off binding sites → crossbridge formation occurs and cycling begins

#### Reversal (Relaxation)

- Membrane repolarizes → DHPR resets → RyR closes
- SERCA pumps  $\text{Ca}^{2+}$  back into SR

SR  $\text{Ca}^{2+}$  release. This pathway is developed further in Chapter 9. The  $\text{Ca}^{2+}$  surge is intense enough to bind nearly all the troponin, pulling tropomyosin strands aside and permitting crossbridge formation.

### The impulse: $\text{Ca}^{2+}$ surge linkage is highly ordered and reversible

The steps taken during relaxation occur in reverse order. The membrane repolarizes, the voltage sensor returns to rest, the ryanodine receptor closes, and SERCA pumps drive  $\text{Ca}^{2+}$  back into the SR. Free  $\text{Ca}^{2+}$  falls rapidly,  $\text{Ca}^{2+}$  dissociates from troponin and that protein is reconfigured to its resting state, tropomyosin re-blocks the myosin binding sites, and tension subsides. The speed at which intracellular  $\text{Ca}^{2+}$  clearance occurs at the onset of relaxation is one reason fast fibers can relax quickly between contractions.

The reason to teach ECC as an ordered chain is that this greatly improves student understanding of the process as a whole and its integral steps. From motor-neuron action potential to ACh release to receptor binding to end-plate  $\text{Na}^+$  influx to sarcolemmal action potential to T-tubule conduction to voltage-sensor shape change to ryanodine receptor opening to  $\text{Ca}^{2+}$  flood to troponin binding to tropomyosin movement to crossbridge formation, each step exists only because the one before it happened. A student who can reconstruct that order on the basis of logic can predict the effect of any malfunction in it; a student who simply memorized the list cannot.

- Troponin releases  $\text{Ca}^{2+}$  → tropomyosin re-blocks → tension falls

#### Common Errors

- **Error:** Thinking the DHPR conducts the calcium that triggers contraction in skeletal muscle. It is the voltage sensor; the RyR releases SR calcium.
- **Error:** Treating ECC as a list to memorize. Each step exists only because the prior one occurred; the order is the logic.
- **Error:** Forgetting relaxation is reversal of the same chain, gated by SERCA lowering calcium.

#### Prediction Prompt

- **Predict:** Given the events scrambled, predict the correct order using cause and effect, then identify which step a T-tubule defect would block.
- **Predict:** Predict the effect on contraction if the link between the DHPR and the ryanodine receptor were broken.

## Section 10: Crossbridge Cycling — Spending ATP to Slide Filaments

### Instructor Narrative

**The move:** Crossbridge cycling is like a physiological tug of war in real time: grab, pull, let go, reach ahead, grab again... Tie each phase of that motion to a specific use of ATP, considering exactly where that molecule (and the energy released by its hydrolysis) is utilized.

### Structured Outline / Causal Chain

#### One Crossbridge Cycle (Tug of War)

- ATP → ADP +  $\text{P}_i$ ; energy cocks the head
- Head binds G-actin

## The bind-pull-release cycle, phase by phase

Once tropomyosin is off the binding sites, the myosin head runs a repeating cycle. In tug-of-war fashion, the myosin head grabs the thin filament, pulls, lets go, recocks, reaches further along, and grabs again. Mechanistically, the head hydrolyzes ATP to ADP and  $P_i$  and uses that energy to recock itself into a high-energy position, like loading a spring. It binds a G-actin site. Releasing  $P_i$  triggers the power stroke, which swings the head and drags the thin filament a few nanometers toward the M-line. Then a fresh ATP binds the head, which releases it from actin, and the cycle repeats.

Two specific substances are absolutely necessary for cycling:  $Ca^{2+}$  and ATP.  $Ca^{2+}$  keeps the binding sites exposed by way of troponin and tropomyosin. ATP does double duty, providing the energy to cock the head and, separately, being the molecule whose binding detaches the head after a stroke. If ATP is absent, the head cannot detach and a rigor state ensues, dramatically visible as rigor mortis after death. So, ATP is needed not only to pull but to release. Students routinely miss this point.

## Tiny strokes, summed and staggered, result in smooth tension

A single power stroke moves the actin filament only nanometers, yet a sarcomere shortens about a micrometer. That means each crossbridge must cycle many times, and billions of heads must work to produce a meaningful contraction. The strokes are also deliberately asynchronous. If every head released at once, tension would stutter. Instead, the randomness of  $Ca^{2+}$ -troponin binding events staggers the cycles, so while some heads are mid-stroke, others are reattaching, and the fiber holds steady tension. This is the molecular reason a contraction feels smooth rather than shuddering.

These summed, staggered events generate internal tension, force between sliding filaments inside the cell. Remember, though, this is not yet force on a bone. That is the handoff previously covered under the key role of structural proteins and developed next in the Hill model. The crossbridge cycle is the source of force; the elastic and structural elements determine how and when that force extends to external sites.

- $P_i$  release → power stroke: filament dragged toward M-line
- New ATP binds → head detaches → cycle repeats

### Two Things Cycling Cannot Do Without

- $Ca^{2+}$ : keeps binding sites exposed (via troponin/tropomyosin)
- ATP: both cocks the head *and* detaches it after the stroke
- No ATP → head cannot release → rigor

### Key Distinction: Pull vs. Release Roles of ATP

- ATP hydrolysis energy: cocks the head for the next stroke
- ATP binding: detaches the head from actin after a stroke
- Rigor mortis = ATP gone, heads stuck bound

### Causal Chain: Why Contractions Are Smooth, Not Stuttering

Each power stroke moves the filament only nanometers, so many cycles are needed for sarcomere shortening; independently, random  $Ca^{2+}$ -troponin binding staggers those cycles in time → some heads pull while others reattach (asynchronous). Summed over billions of heads, the tension is steady and smooth

### Common Errors

- **Error:** Thinking ATP is only for pulling. ATP binding is also what detaches the head; without it, rigor occurs.
- **Error:** Imagining all heads stroke in unison. Asynchronous cycling is what keeps tension smooth.
- **Error:** Assuming crossbridge force is applied directly to bone. Cross bridge cycling generates internal tension; transmission to external tissues is a separate step.

### Prediction Prompt

- **Predict:** Predict the state of the crossbridges if the ATP supply is suddenly depleted mid-contraction. When does this generally occur in the real world?
- **Predict:** Predict why a fiber needs many cycles per sarcomere to produce a 1-micrometer shortening from nanometer strokes.

## Section 11: The Hill Model — How Internal Tension Becomes External Tension

| Instructor Narrative                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   | Structured Outline / Causal Chain                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            |
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| <p><b>The move:</b> Draw the three-element Hill model as an engineering diagram and put it to work: understanding this model explains why just a single twitch can barely move a load. Explain why action of the series elastic element (SEE) must be carefully considered in any study of applied skeletal muscle contraction.</p> <p><b>Three elements: a motor and two springs</b></p> <p>The Hill model strips muscle to three mechanical elements in order to explain something the crossbridge cycle alone cannot: the timing of force applied at the bone. In this model, the sarcomeres are the contractile element (CE), the active force generator. In series with the CE, between it and the bone, is the series elastic element (SEE), the springy tendon and fascia plus the elasticity of the filaments themselves. In parallel with the CE and SEE is the parallel elastic element (PEE), the sarcolemma and surrounding connective tissue. The PEE simply resists length change. Think of a motor connected to its load through a spring, with a second spring alongside resisting any stretch or squeeze.</p> <p>The SEE has a decisive role because it sits between the force generator and the bone. When the CE pulls, it must first stretch the SEE before significant force reaches the bone. This is exactly where internal tension and external tension diverge: internal tension, <math>T_i</math>, rises promptly as crossbridges form, but external tension, <math>T_e</math>, lags because contractile</p> | <p><b>The Three Hill Elements</b></p> <ul style="list-style-type: none"> <li>• Contractile element (CE): the sarcomeres, active force</li> <li>• Series elastic element (SEE): tendon, fascia, filament elasticity, in line with CE</li> <li>• Parallel elastic element (PEE): sarcolemma + connective tissue, resists length change</li> </ul> <p><b>Internal vs. External Tension</b></p> <ul style="list-style-type: none"> <li>• <math>T_i</math> rises promptly as crossbridges form</li> <li>• <math>T_e</math> lags because the SEE must be stretched first</li> <li>• <math>T_e</math> also lingers after <math>T_i</math> falls (SEE recoil)</li> </ul> <p><b>Key Distinction: Twitch vs. Sustained Contraction</b></p> <ul style="list-style-type: none"> <li>• Single twitch: too brief to fully stretch the SEE → little external work</li> <li>• Sustained contraction: SEE becomes taut (cable-like) → <math>T_e</math> can match <math>T_i</math></li> <li>• Rubber band + book: one quick tug vs. a sustained pull</li> </ul> <p><b>Causal Chain: Why One Twitch Barely Moves a Load</b></p> |

energy is going into first stretching the SEE rather than pulling on the tendon.

### Why a single twitch does almost no useful work

A twitch lasts only fractions of a second, far too brief to fully stretch the series elastic element. Thus, during a single twitch, most of the contractile energy is expended stretching that SEE spring.  $T_e$  doesn't climb to match  $T_i$ , and the load barely moves. A rubber-band-and-book analogy may be useful here: tape a rubber band to a book and give one quick, weak tug. The band stretches, the book does not budge. Only a sustained pull, which fully tautens the band, moves the book. The twitch is the quick tug.

Two practical consequences follow. First, useful work generally requires contractions of greater duration than a single twitch, which is why summation and tetanus, discussed in the next section, are so significant. Second, the SEE is protective and energy-storing: it dampens the rate of tension change at the tendon and recoils as it relaxes, helping return the fiber to resting length and sparing the connective tissue from sudden loads. Note that titin's elasticity contributes to the SEE as well, which sets up the larger role titin plays during lengthening contractions.

Crossbridges generate internal tension ( $T_i$ ) quickly → energy first goes into stretching the SEE → because a twitch is too brief to fully stretch the SEE, it ends before the spring is taut → internal tension is never fully converted to external tension ( $T_e$  stays well below  $T_i$ ) → load is moved very little or not at all

### Common Errors

- **Error:** Thinking  $T_i$  and  $T_e$  are the same during a twitch. The SEE makes  $T_e$  lag and stay lower than  $T_i$ .
- **Error:** Treating the SEE as a flaw. This elasticity protects tissue and stores recoil energy.
- **Error:** Assuming a single twitch does meaningful work against a real load. It usually cannot.

### Prediction Prompt

- **Predict:** Predict the relationship between  $T_i$  and  $T_e$  during a single twitch and explain it with the SEE.
- **Predict:** Predict why external tension lingers briefly after internal tension has fallen at the end of a twitch.

## Section 12: Isometric and Isotonic Contractions — Same Length or Same Tension?

### Instructor Narrative

***The move:** An arm-wrestling analogy can be used to illustrate differences between the various modes of muscle contraction. It can also make it clear these forms lead to one another in a very predictable way in real life scenarios.*

### Isometric and isotonic are defined by what stays constant

Two pure contraction types are defined by the prefix “iso-“, meaning same. An isometric contraction keeps the same length: the muscle

### Structured Outline / Causal Chain

#### The Definitions (Iso = Same)

- Isometric: same length; tension does not exceed load; no shortening
- Isotonic: same tension (constant load); length changes
- Concentric isotonic: shortening (tension > load)

generates tension but does not shorten, because the tension developed does not exceed the load. Postural muscles do this all day. An isotonic contraction keeps the same tension while the length changes, because the muscle is moving a constant load. Real movements actually blend the two. Use the arm-wrestling image: at the start both wrestlers strain without movement and isometric contraction takes place, with the CE shortening exactly as much as the SEE stretches.

Isotonic contractions come in two varieties, depending on whether the muscle shortens or lengthens while it pulls. When one wrestler wins, generating more tension than the load, that muscle shortens in an isotonic concentric contraction. Picking up a bag is also concentric once the muscle actually shortens and the bag leaves the ground. If a new balance is reached (while either arm wrestling or trying to lift an object), the contraction is once again isometric, with the muscle just at a shorter length. The order is important in all this: lift a bag and you go isometric, then concentric, then isometric, then, on lowering it under control, isotonic eccentric.

### **Eccentric contraction: lengthening while generating force**

The isotonic eccentric contraction is challenging for some students to grasp. Here, the muscle lengthens even though it is actively generating tension, because the load exceeds the force produced. As we just saw, lowering a heavy bag slowly is eccentric. In the arm-wrestling story, it occurred in the arm wrestler losing while still straining, and is even observed in the rope slipping through one's hands during an ongoing tug of war. Crossbridges keep forming and trying to pull, but the filaments are being dragged the wrong way. Eccentric contractions are how muscles absorb and control loads, and they are a major source of the soreness after unfamiliar downhill or lowering exercise.

There is a subtlety in eccentric contractions that's often underdiscussed, but still significant. Eccentric force is not only about crossbridges losing ground. Titin (an important structural protein mentioned previously) behaves as a tunable molecular spring, and there's current evidence that its stiffness can increase when the fiber is activated. In this way, a stretched, activated sarcomere stores and bears real passive tension through titin. The proposed mechanisms, still being worked out, are that  $\text{Ca}^{2+}$  binds titin directly at specific sites and that, on activation, a region of

- Eccentric isotonic: lengthening (load > tension, force still generated)

### **The Arm-Wrestling Sequence**

- Clasp (rest) → strain, no movement (isometric)
- Winning: shortening (concentric)
- New balance: isometric, shorter length
- Losing: lengthening while straining (eccentric)

### **Eccentric Force: More Than Crossbridges**

- Crossbridges keep cycling while the filament is dragged backward (tug of war being lost)
- Titin acts as a tunable spring; stiffness rises with activation
- Stretched, activated sarcomere bears real titin-borne tension
- Helps explain high lengthening strength and eccentric micro-damage

### **Causal Chain: Lifting Then Lowering a Bag**

Strain before liftoff: isometric (unshortened) → tension exceeds load: concentric, bag rises → hold it still: isometric (shortened) → lower under control: eccentric, muscle lengthens while generating force

### **Common Errors**

- **Error:** Thinking eccentric contractions are passive relaxation. The muscle is actively generating force while lengthening.
- **Error:** Explaining eccentric force as crossbridge events alone. An activated titin spring carries part of the lengthening load.
- **Error:** Assuming isotonic always means shortening. Eccentric isotonic is lengthening at near-constant tension.

### **Prediction Prompt**

titin binds the thin filament, shortening titin's free spring length so that a stretch loads only its stiff segment. That extra titin-borne tension helps explain why muscles are unusually strong during lengthening and why eccentric loading is effective for building strength and, at the same time, implicated in making muscles contracting under eccentric conditions susceptible to micro-damage.

- **Predict:** Predict which contraction type dominates while one walks downstairs, and explain using load versus force.
- **Predict:** Predict why titin behaving as an activation-stiffened spring would make a muscle stronger when lengthening than when shortening.

## Section 13: Frequency–Tension — Wave Summation and Tetanus

| Instructor Narrative                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               | Structured Outline / Causal Chain                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              |
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| <p><b>The move:</b> Tie tension directly to <math>\text{Ca}^{2+}</math>, the CE, and SEE. Higher stimulus frequency does two things at once: it keeps sarcoplasmic <math>\text{Ca}^{2+}</math> high and it keeps the SEE taut. Students need to acknowledge both contributions rather than just assuming that twitches “add up”.</p> <p><b>Faster stimulation stacks twitches into summation</b></p> <p>Action potentials rarely arrive alone. They come as a train, and their frequency partially controls force. If stimuli occur at low frequency, <math>\text{Ca}^{2+}</math> is fully pumped back into the SR between them and the twitches are separate and distinct. As frequency rises, <math>\text{Ca}^{2+}</math> does not have time to return to baseline before the next stimulus, so sarcoplasmic <math>\text{Ca}^{2+}</math> accumulates, more crossbridges form and stay formed, and successive twitches add on top of one another. This stacking of twitch tension is wave summation, a stronger and longer-lasting contraction than any single twitch.</p> <p>There is also a second mechanism working in parallel, and it comes straight from the Hill model. Each twitch begins to stretch the SEE, but a single twitch ends before the SEE is taut. With rapid repeated stimulation, the SEE gets pulled fully tight and stays tight. As a result, subsequent twitch tension is conveyed to the load instead of being absorbed by the spring. Summation therefore reflects both rising <math>\text{Ca}^{2+}</math> and a tautened SEE, two reasons external tension climbs with frequency.</p> | <p><b>How Frequency Raises Force (Two Mechanisms)</b></p> <ul style="list-style-type: none"> <li>• <math>\text{Ca}^{2+}</math> accumulates in the sarcoplasm: too little time to pump it back between stimuli</li> <li>• More crossbridges form and persist → twitches stack (wave summation)</li> <li>• SEE stays taut → twitch tension reaches the load instead of stretching the spring</li> </ul> <p><b>Tetanus</b></p> <ul style="list-style-type: none"> <li>• Very high frequency → <math>\text{Ca}^{2+}</math> maximized, SEE fully taut</li> <li>• Twitches fuse into smooth, maximal, sustained tension</li> <li>• Fast fibers need higher frequency to fuse than slow fibers</li> </ul> <p><b>Key Distinction: Skeletal vs. Cardiac on Summation</b></p> <ul style="list-style-type: none"> <li>• Skeletal: short AP/refractory period → summation and tetanus possible</li> <li>• Cardiac: long AP/refractory period → tension falls before re-stimulation; no tetanus</li> <li>• Same machinery, opposite design goal (steady pull vs. separate beats)</li> </ul> |

### Push frequency high enough and tetanus occurs

At very high stimulus frequency,  $\text{Ca}^{2+}$  is maximized, all the slack is out of the series elastic element, and twitches fuse into a smooth, maximal, sustained contraction called complete tetanus. This is the most force a single fiber can produce. The frequency needed to get to this point depends on fiber type: long-duration slow twitches summate at modest frequencies, while brief fast twitches need very high frequencies to fuse. In practice, true complete tetanus is fairly rare because muscle action potential rates are capped, so most real contractions live in partial summation.

The situation with heart contractions is different and makes the logic behind summation vivid. Skeletal muscle can summate and tetanize precisely because its action potential and refractory period are short. Cardiac muscle deliberately cannot, because its action potential and refractory period are of long enough duration that tension falls before another stimulus can arrive. This single design difference, treated in the cardiac section, is why heart beats always occur in separate strokes but a quadriceps can maintain a steady maximal pull.

### Causal Chain: From Higher Frequency to Greater Tension

Stimulus frequency rises → two effects follow in parallel: sarcoplasmic  $\text{Ca}^{2+}$  stays elevated between stimuli, driving more persistent crossbridges; and, independently, successive twitches restretch the SEE before it can recoil, holding it taut → twitch tensions sum (wave summation) → at maximal frequency the twitches fuse into maximal tetanus

### Common Errors

- **Error:** Explaining summation only as twitches adding. Both rising  $\text{Ca}^{2+}$  and a tautened SEE drive the tension increase.
- **Error:** Thinking all fibers tetanize at the same frequency. Fast fibers need much higher rates than slow fibers.
- **Error:** Believing the heart can tetanize. Its long refractory period adaptively prevents summation.

### Prediction Prompt

- **Predict:** Predict the difference in stimulus frequency needed to tetanize a fast-twitch versus a slow-twitch fiber, and explain.
- **Predict:** Predict what would happen to cardiac pumping if the cardiomyocyte refractory period were shortened to skeletal-muscle length.

## Section 14: Length–Tension — Overlap Sets the Ceiling

### Instructor Narrative

*The move:* Remind students about the zone of overlap and its importance. Active CE tension is just a function of possible crossbridge interactions, and crossbridge number depends on how much thick and

### Structured Outline / Causal Chain

#### Why Length Sets Active Tension

- Active tension is made only in the zone of overlap

*thin filament overlap exists at the starting length. Have students sketch overlap at three hypothetical lengths before predicting differences in tension generated.*

### Starting length sets how many crossbridges are possible

How much tension a fiber can generate depends on its length before it contracts, and the reason is overlap. Active tension is produced only in the zone of overlap, and its magnitude is proportional to the number of crossbridges that can form. Stretch a fiber too far and the thick and thin filaments barely overlap, so few crossbridges are possible and active tension falls. At extreme stretch there is no overlap at all and no active tension can be generated, regardless of effort. This is the same no-overlap-no-force rule from the sarcomere section, now plotted against length.

Shortening too much causes a different problem. If a sarcomere is compressed below about seventy percent of normal resting length and the thin filaments from opposite ends start to collide, or the thick filaments run into the Z-discs, both of which interfere with effective crossbridge formation. That means a sweet spot must exist. Active tension peaks at an optimal resting length, where overlap is maximal, and falls off when the fiber is either too stretched or too compressed. Joints normally keep muscles within the favorable mid-range, which is why muscles feel strongest at mid-range positions.

### Passive tension and a preview of the heart

There is a second, related, curve to consider when considering length – tension relationships. If a resting fiber is stretched, its elastic elements, including titin, resist, producing passive tension that rises steadily. Total tension is the sum of active tension (from crossbridges, peaking at the optimum) and passive tension (from elastic recoil, always climbing with stretch). At very long myofiber lengths, active tension falls to zero (no overlap), but total tension keeps rising because passive tension continues to rise. Distinguishing the active and passive curves (and comprehending differences between them) prevents a lot of confusion.

This relationship just brought up is not just a skeletal-muscle curiosity. The same logic governs the heart. When cardiac chambers fill with more

- Tension is proportional to crossbridge number
- Myofibers too stretched → little/no overlap → little/no active tension
- Myofibers too short (<~70%) → filament/Z-disc interference → tension falls

#### Active vs. Passive vs. Total

- Active: from crossbridges; peaks at optimal length
- Passive: from elastic elements (incl. titin); rises with stretch
- Total = active + passive (keeps rising at long lengths via passive)

#### Key Distinction: Overlap-Limited vs. Interference-Limited

- Long lengths: limited by lost overlap
- Short lengths: limited by thin-filament collision / thick-filament-Z-disc contact
- Peak active tension sits between, at optimal resting length

#### Causal Chain: Why a Fuller Heart Beats with Greater Strength

More venous return fills the chamber → cardiomyocytes stretched toward optimal overlap → more crossbridges can form → stronger contraction ejects the extra blood (basis of Ch. 9 length-tension)

#### Common Errors

- **Error:** Thinking more stretch always means more force. Past the optimum, lost overlap drops active tension.
- **Error:** Merging active and passive tension. They are separate curves; total tension is their sum.
- **Error:** Treating length-tension as applying only to skeletal muscle. This concept underlies the heart's force-filling relationship.

#### Prediction Prompt

blood between heartbeats, the cardiomyocytes are stretched toward better overlap, so they contract more forcefully and eject the extra blood. That is the cellular basis of this key relationship developed in Chapter 9 as the mechanism by which the heart matches output to filling. Teaching length-tension here pays off directly when students reach cardiac function.

- **Predict:** Predict the active tension of a fiber stretched until thick and thin filaments no longer overlap, and explain.
- **Predict:** Predict how increased ventricular filling changes contraction strength, using overlap as the rationale.

## Section 15: Diameter, Work, and Power — Keeping Three Measures Straight

| Instructor Narrative                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 | Structured Outline / Causal Chain                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   |
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| <p><b>The move:</b> Force, work, and power are three different entities, and students blur them constantly. Define each by its equation and use a few daily examples to illustrate key differences between them.</p> <p><b>Diameter sets force; force is not the whole story</b></p> <p>Fiber diameter is the structural lever for force. A larger-diameter fiber holds more myofibrils and therefore more contractile protein, so more crossbridges can form at once and more force is generated. This is why exercise that thickens fibers raises strength, and why a thicker muscle is a stronger muscle. But force, the instantaneous pull, is only a measure of tension generated. Considered alone, force tells us very little about what the muscle contraction actually achieved. What really needs to be looked at is how much work was done as a result of a muscle contraction, how much power that contraction generated, or both. These are easily calculated using some very simple math.</p> <p>Work is computed as the product of force applied times distance the load is moved, so a muscle that generates great force but moves nothing, a pure isometric hold, does zero work. Furthermore, a muscle moving a tiny load (very minimal force needed) over a huge distance also does little work. Maximal work comes from moving a moderate load a moderate distance. Because distance depends on how much the fiber shortens,</p> | <p><b>Three Quantities, Three Equations</b></p> <ul style="list-style-type: none"> <li>• Force: the instantaneous pull; set by fiber diameter (crossbridge number)</li> <li>• Work = Force x Distance; distance depends on muscle length</li> <li>• Power = Work / Time = Force x Velocity; depends on fiber type (ATPase speed)</li> </ul> <p><b>When Work Is Zero</b></p> <ul style="list-style-type: none"> <li>• Isometric hold: big force, no distance → zero work</li> <li>• Tiny load over a long distance → little work</li> <li>• Maximal work: moderate load, moderate distance</li> </ul> <p><b>Key Distinction: Same Work, Different Power</b></p> <ul style="list-style-type: none"> <li>• Mow the lawn in 1 h or 4 h: same work, more power in 1 h</li> <li>• Two equal-strength fibers: faster ATPase isoform → more power</li> <li>• <math>V_{\max}</math> is highest in fast fibers; power peaks at moderate load and velocity</li> </ul> <p><b>Derivation Chain: Why a Taller Lifter Generates More Power at Equal Weight</b></p> |

and shortening depends on how many sarcomeres sit in series, work output depends heavily on muscle length.

### Power adds time (velocity, actually) into the mix, and that is where fiber type plays a major role

Power is work per unit time(or, if you will, force times velocity). Imagine mowing the same lawn in either one hour or four hours. The same work is performed either way, but the one-hour job requires generation (and use!) of more power because the same work is done faster. That means two fibers can do identical work and differ greatly in power if they function at different speeds. The faster myosin ATPase isoform allows fast fibers to cycle crossbridges quickly and, therefore, generate high power.

The dumbbell curl example separates the three cleanly. If a tall person and a short person curl the same weight, they generate the same force, because force is set by the load. But the taller person's longer muscles, with more sarcomeres in series, move the weight through a greater range, so each rep does more work, and if both finish a rep in the same time, the taller person also generated more power (the load moved at a greater velocity). Keep the three parameters straight: force tracks diameter, work tracks distance (and thus length), power tracks how fast the work is done (and thus fiber type).

The taller lifter's longer muscle → more sarcomeres in series → greater shortening distance per rep, at the same weight and therefore the same force → force acting over a greater distance → more work per rep → at the same rep time, more power

#### Common Errors

- **Error:** Using force, work, and power interchangeably. They are distinct entities and computed with different equations.
- **Error:** Thinking an isometric hold does work. Distance is zero, so work is zero, however strong the pull.
- **Error:** Believing more force always means more power. Power requires speed; ATPase isoform sets that.

#### Prediction Prompt

- **Predict:** Predict the work done during a maximal isometric contraction and justify with  $W = F \times D$ .
- **Predict:** Two equal-diameter fibers move the same load; one is type IIx (fast twitch), one is type I (slow twitch). Predict which generates more power and why.

## Section 16: Fiber Types and Metabolism — Matching Machinery to the Job

### Instructor Narrative

***The move:** Muscle fiber types are derived based on two functional considerations: how fast does the myosin ATPase cycle, and how does the fiber make ATP. Once students see that a fiber's contractile speed and its metabolic machinery match each other, these “dark-meat” and “white-meat” differences become easily predictable.*

**Two axes generate three useful fiber types**

### Structured Outline / Causal Chain

#### Two Axes, Three Types

- Type I (SO): slow ATPase, oxidative, small, weak, endurance
- Type IIa (FOG): fast ATPase, both oxidative and glycolytic, fatigue-resistant

Fiber type is really based on two traits. First, how fast is the myosin ATPase, which sets twitch speed and power. Second, how does the fiber regenerate ATP, aerobically or anaerobically, which sets endurance. Combine these and three practical types emerge. Slow oxidative (type I) fibers are slow, weak, small, and fatigue-resistant. Fast oxidative-glycolytic (type IIa) fibers are fast and reasonably fatigue-resistant, a middle ground. Fast glycolytic (type IIx) fibers are the fastest and strongest but fatigue quickly. The naming literally encodes the two axes: speed first, metabolism second.

The metabolic equipment is matched to the contractile task. A slow oxidative fiber that must work for hours needs a large oxygen supply and aerobic machinery, so it has dense capillaries, abundant mitochondria, plentiful myoglobin to store oxygen, and stores fat, which can only be burned aerobically. A fast glycolytic fiber that fires in short, intense bursts will outrun its oxygen supply almost immediately, so being good at aerobic metabolism really isn't necessary. Instead this fiber stockpiles glycogen and glycolytic enzymes to drive anaerobic ATP production hard and fast, accepting the inefficiency in exchange for speed.

### Why dark meat and white meat differ

The dark-meat and white-meat contrast makes the logic involved here concrete. Myoglobin and mitochondria give slow oxidative fibers their dark color, so the endurance muscles, those in the legs of a chicken that walks all day, are dark. Fast glycolytic fibers lack myoglobin and are pale, so the sprint muscle, a chicken's breast used for short bursts of flapping, is white. Predict from there: a duck flies long distances, so its flight (breast) muscle must be aerobic and endurance-adapted, which is why duck breast is dark. Function predicts color (along with other characteristics) in these examples.

Finally, energy sources are tapped in a sensible order based on speed and capacity. Stored ATP lasts only a second or two. Phosphocreatine instantly refills ATP for another several seconds, a fast buffer found mainly in skeletal muscle precisely because that tissue is most likely to outrun its ATP supply. Glycolysis then provides rapid but inefficient ATP from glucose and glycogen, dominating in fast fibers. Oxidative

- Type IIx (FG): fastest ATPase, glycolytic, strong, fatigues fast

### Metabolism Matched to Job

- Oxidative (slow): dense capillaries, many mitochondria, myoglobin, fat stores
- Glycolytic (fast): high glycogen + glycolytic enzymes, few mitochondria, little myoglobin
- Color follows myoglobin/mitochondria: slow = dark, fast = white

### Energy Sources, In Order

- ATP: seconds (must be resupplied)
- Phosphocreatine: instant buffer, ~ 5-10 s more (mainly skeletal)
- Glycolysis: fast, inefficient, glucose/glycogen (fast fibers)
- Oxidative: slow, high yield, glucose + fat (slow fibers); liver/fat = the grocery store

### Causal Chain: Why a Fast Glycolytic Fiber Stores Glycogen, Not Fat

Fast ATPase uses ATP faster than oxygen can be delivered → fiber goes anaerobic almost immediately when active, and only glucose and glycogen fuel anaerobic ATP synthesis (fat cannot). A fiber built this way therefore stockpiles glycogen and glycolytic enzymes, not fat

### Common Errors

- **Error:** Treating fiber type as one trait. It has two: ATPase speed *and* metabolic strategy.
- **Error:** Thinking color causes function. Myoglobin and mitochondria (function) determine color.
- **Error:** Believing fat fuels sprinting. Fat is aerobic only; anaerobic bursts run on glucose/glycogen.

### Prediction Prompt

metabolism of glucose and fats yields far more ATP but more slowly, dominating in slow fibers and during prolonged work.

- **Predict:** Predict the dominant fiber type in an elite marathoner's quadriceps versus that in a sprinter's, and justify.
- **Predict:** A duck's breast meat is dark while a chicken's is white. Predict the reason from flight versus flapping behavior.

## Section 17: Motor-Unit Recruitment and the Size Principle — Assembling Whole-Muscle Tension

### Instructor Narrative

**The move:** Shift the unit of focus from fiber to motor unit and note that the size principle follows logically. Ask why it would be a disaster to recruit the strongest units first, then derive the small-to-large order from fatigue and control.

### The motor unit, not the fiber, is the practical building block

Whole-muscle force is built by recruiting motor units, where a motor unit is one motor neuron plus all the fibers it controls. Because all unit fibers fire together as an all-or-nothing group, the size of a motor unit sets the increment of force it adds. Small units, a handful of fibers, allow fine grading, which is why eye muscles use tiny units. Large units, hundreds of fibers, add big chunks of force, which is why leg muscles use large ones. A muscle grades its output by choosing how many units to switch on and how fast to fire them, not by partially contracting individual fibers.

The fibers of a single motor unit are all one fiber type and are scattered throughout the muscle belly rather than clumped. Such dispersion means that activating a unit produces an even, balanced pull across the whole muscle and a smooth line of force applied to the tendon, even when only a few units are active. This spreading is an important design feature.

### The size principle orders recruitment small to large

### Structured Outline / Causal Chain

#### Motor Unit Basics

- Motor unit = one motor neuron + all fibers it innervates (all one fiber type)
- Fibers fire together (all-or-nothing); unit size = force increment
- Small units → fine control (eyes); large units → big force (legs)
- Unit fibers are dispersed → even, balanced pull

#### The Size Principle

- Recruit smallest (type I) units first, largest (type IIx) last
- Small neurons = low threshold; large neurons = high threshold
- Force grades by recruiting more units + firing faster

#### Key Distinction: Recruitment vs. Rate Coding

- Recruitment: how many units are switched on
- Rate coding: how fast and frequently each unit fires (summation within units)

Recruitment is not random. As effort rises, the smallest motor units are recruited first, then progressively larger ones are brought into play; this is the size principle. The mechanism involved here is electrical: small motor units are driven by small motor neurons that are easier to bring to threshold, so they fire at the slightest effort, while bigger units have large, high-threshold neurons that fire only under strong drive. The first units recruited are the small, slow, fatigue-resistant type I units; the last are the large, fast, fatigable type IIx units.

Ask why this order is adaptive and the reasoning follows directly. Recruiting weak, endurance units first is logical because light tasks are rarely tiring and finely controllable. The powerful, fatigable units are held in reserve and added only when force demands are high, so they are not wasted and their fatigue is not incurred unnecessarily. Adding big units late, once the series elastic elements are already stretched and a load may already be moving, also avoids sudden violent jerks that could injure muscle or tendon. The same principle explains why jogging can continue for hours while a maximal sprint exhausts you in seconds.

- Both recruitment and rate coding raise whole-muscle force; the size principle orders recruitment

#### Derivation Chain: Why Small Units Fire First

Small motor units are driven by small motor neurons → a small neuron reaches threshold at low synaptic input (due to its cable properties) → those units fire first as effort rises, and the large, fatigable units are recruited only at high force

#### Common Errors

- **Error:** Thinking a muscle grades force by partially contracting fibers. Fibers are all-or-nothing; grading is accomplished by unit recruitment and adjusting firing rate.
- **Error:** Assuming strong units fire first. The smallest, most fatigue-resistant units fire first (size principle).
- **Error:** Picturing a motor unit's fibers clumped together. They are dispersed for an even pull.

#### Prediction Prompt

- **Predict:** Predict the recruitment pattern (SO, FOG, FG) for quiet standing versus a maximal vertical jump.
- **Predict:** Explain why activating large fast units only after a load is already moving protects the muscle and tendon.

## Section 18: Muscle Plasticity — How Training Rewrites the Tissue

### Instructor Narrative

**The move:** Treat training-induced plasticity as muscle changes invoked to solve a functional problem. Strength training poses force demands; endurance training poses sustained-ATP demands. Have students predict

### Structured Outline / Causal Chain

#### Strength (Anaerobic) Adaptations

- Hypertrophy, fastest in type II fibers (more myofibrils → more force/power)

*the adaptations that should logically occur in muscle to make it more capable of engaging in a particular mode of training.*

### **Strength training builds force-making and force-delivery machinery**

Muscle is highly plastic, remodeling its structure and metabolism to match the stress placed on it. Anaerobic strength training, lifting heavy, poses a force problem, so the tissue adapts to make and deliver more force. All fiber types can enlarge, but the fast type II fibers hypertrophy most, adding myofibrils through protein synthesis, which raises cross-sectional area and therefore force, and because they are fast, capable of generating needed power. There is also a shift toward faster ATPase expression and also from hybrid toward purer fast types, all directing the tissue toward greater force and speed.

Not all early strength gains are bigger fibers, which surprises many students. The first improvements are neural and excitation-related: faster maximal motor-neuron firing and enhanced  $\text{Ca}^{2+}$  release, which accelerate force development even before any hypertrophy appears. Hypertrophy is slower to arrive but accounts for the continued strength gains after the neural adaptations have plateaued. This explains why a beginner gets noticeably stronger in a couple of weeks without visibly larger muscles.

### **Endurance training builds the aerobic supply chain**

Aerobic endurance training, running and cycling, poses a sustained-ATP problem, so the adaptations target oxidative capacity rather than raw force. Fibers increase the number and size of mitochondria, raise oxidative and fat-metabolizing enzymes, add capillaries to shorten diffusion distances for oxygen, and increase myoglobin. The slow type I fibers gain moderate size mainly from added mitochondrial volume, and there is again a shift of hybrid fibers toward the type best suited to the stimulus, in this case, toward more oxidative profiles.

The unifying idea is that the muscle builds whatever the demand requires and lets the rest atrophy. Strength stimulus produces force machinery; endurance stimulus produces aerobic supply. This is the same theme observed in almost all of physiology, function shaping structure, now

- Shift toward faster ATPase; hybrid → purer fast types
- Early gains are neural + faster  $\text{Ca}^{2+}$  release, before hypertrophy

#### **Endurance (Aerobic) Adaptations**

- More and larger mitochondria; more oxidative + fat enzymes
- Increased capillary density and myoglobin
- Type I moderate hypertrophy (mostly mitochondrial volume)

#### **Key Distinction: Neural vs. Structural Gains**

- First strength gains: neural (firing rate,  $\text{Ca}^{2+}$  release); fast onset
- Later strength gains: hypertrophy; slower onset
- Explains rapid early strength with no visible size change

#### **Causal Chain: Why Endurance Training Raises Oxidative Capacity**

Repeated sustained low-force work demands continuous ATP → repeated energy stress activates signaling that drives mitochondrial biogenesis and enzyme expression → more mitochondria, capillaries, myoglobin → greater aerobic ATP supply → improved endurance

#### **Common Errors**

- **Error:** Thinking early strength gains come from bigger muscles. The first gains are neural; hypertrophy follows.
- **Error:** Expecting endurance training to build large, powerful fibers. This kind of training builds aerobic supply, not maximal force.
- **Error:** Assuming adaptations persist without use. Detraining reverses costly, unused changes.

#### **Prediction Prompt**

operating over weeks instead of milliseconds. This same reasoning, but in reverse, also explains detraining: remove the demand and costly adaptations regress because there is no reason to maintain machinery that is not being used.

- **Predict:** Predict the specific fiber and metabolic changes after months of marathon training versus months of heavy powerlifting.
- **Predict:** Predict why a novice can gain measurable strength in two weeks without visible muscle growth.

## Section 19: Proprioceptive Control — Spindles, Golgi Tendon Organs, and Reciprocal Inhibition

| Instructor Narrative                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          | Structured Outline / Causal Chain                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   |
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| <p><i><b>The move:</b> Frame each of the two different proprioceptors as either a length detector or a tension detector, then observe it in action. The spindle reflex seems tidy in isolation, but consider how real movement complicates it and how reciprocal inhibition resolves an obvious paradox</i></p> <p><b>Two sensors: one for length, one for tension</b></p> <p>Muscles are not just actuators; they are instrumented. Two proprioceptors report on what the muscle is doing. The muscle spindle, built from intrafusal fibers lying parallel to the main contractile fibers, senses length and the rate of length change. Stretch the muscle and the spindle fires faster, telling the central nervous system the muscle is lengthening. The Golgi tendon organ, sitting near the muscle-tendon junction, senses active tension, because the tendon stretches mainly when the muscle is actively pulling on it, not from passive stretch. The length and tension detectors work in complementary directions.</p> <p>The spindle drives the stretch reflex, which also maintains muscle tone. When a muscle is stretched, spindle sensory neurons synapse directly onto the motor neurons of that same muscle and trigger a contraction that resists the stretch. Tone, the low-grade tension a resting muscle always carries, comes from the same loop firing the lowest-threshold units, which takes up slack in the SEE and improves filament overlap, keeping the muscle ready to respond. Gamma motor neurons keep the spindle taut and sensitive even as the muscle shortens, so it never goes slack.</p> | <p><b>Two Proprioceptors</b></p> <ul style="list-style-type: none"> <li>• Muscle spindle (intrafusal, parallel): senses length and rate of stretch</li> <li>• Golgi tendon organ (at muscle-tendon junction): senses active tension</li> <li>• Spindle = length detector; GTO = tension detector</li> </ul> <p><b>Stretch Reflex and Tone</b></p> <ul style="list-style-type: none"> <li>• Stretch → spindle afferents fire → they excite the same muscle's motor neurons → contraction that resists the stretch</li> <li>• Tone = low-threshold units kept active (takes up SEE slack, improves overlap)</li> <li>• Gamma motor neurons keep the spindle sensitive as the muscle shortens</li> </ul> <p><b>Key Distinction: Reciprocal Inhibition vs. GTO Feedback</b></p> <ul style="list-style-type: none"> <li>• Reciprocal inhibition: inhibit the antagonist so it does not fight the mover</li> <li>• GTO reflex: inhibit the <i>same</i> muscle when its tension gets too high (protection)</li> <li>• Together they coordinate movement and prevent self-damage</li> </ul> |

### Putting the reflexes in a moving body

Students should spot a problem with the stretch reflex the moment it is generalized. Contracting one muscle stretches its antagonist, which would trigger that antagonist's stretch reflex and fight the intended movement. The nervous system solves this with reciprocal inhibition: while it excites the prime mover, it simultaneously inhibits the motor neurons of the antagonist, so the antagonist does not interfere. The same stretch that would have started a counterproductive reflex instead lengthens the antagonist's sarcomeres, priming it to contract more strongly when its turn comes.

The Golgi tendon organ adds protection and fine tuning. As contraction tension rises, the GTO fires and, through inhibitory interneurons, dials down the motor neurons of the same muscle, forming a negative feedback loop that limits dangerously strong contractions and smooths tension. So the spindle regulates length while the GTO regulates tension, and the two, plus reciprocal inhibition, plus input from one another, make even a simple movement a coordinated, self-correcting act rather than a single reflex arc.

### Causal Chain: Why a Mover Is Not Fought by Its Antagonist

Prime mover contracts and shortens → antagonist is stretched → its stretch reflex would resist, but the same descending command that excites the mover simultaneously inhibits the antagonist's motor neurons (reciprocal inhibition), overriding that reflex → antagonist relaxes and lengthens, priming it for its own later contraction

### Common Errors

- **Error:** Confusing the two sensors. Spindle = length (parallel); GTO = active tension (at the tendon).
- **Error:** Thinking the GTO responds well to passive stretch. It mainly responds to active contraction tension.
- **Error:** Treating reflexes as isolated arcs. Reciprocal inhibition and cross-talk make movement coordinated.

### Prediction Prompt

- **Predict:** Predict the spindle and GTO outputs when one isometrically flexes the biceps with eyes closed, and what each tells the brain.
- **Predict:** Predict what movement would look like if reciprocal inhibition failed during elbow flexion.

## Section 20: Cardiac Muscle — Twitching Forever (Well, Almost) Without Summation

Cardiac excitation and the conduction system are developed fully in Chapter 9. Note how the cardiac muscle physiology presented here contrasts with that of skeletal muscle.

| Instructor Narrative                                                                                                                                                                                                | Structured Outline / Causal Chain |
|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------|
| <b>The move:</b> Teach cardiac muscle by difference, not from scratch. This tissue shares the sarcomere and ECC with skeletal muscle, so spend the time on the three changes that matter most: cells coupled into a | Shared With Skeletal Muscle       |

*syncytium, a self-starting pacemaker, and an action potential long enough to prevent summation and tetanus.*

### Same contractile core, three decisive differences

Cardiac muscle is striated and uses the same sarcomere, troponin and tropomyosin, SR  $\text{Ca}^{2+}$ , and T-tubule arrangements as skeletal muscle, so the contractile mechanism is familiar. The main differences relate to organization and timing, and they all serve one job: pump blood reliably for a lifetime. First, cardiomyocytes are small, branching, single-nucleus cells joined end to end by intercalated discs rather than long fused fibers, and they pull on each other rather than on bone. Those discs possess desmosomes that transmit force between cells and gap junctions that pass depolarizing current, so the tissue is structurally and electrically linked into a functional syncytium that contracts as a unit.

Second, the heart initiates its own beat. Nodal pacemaker cells are not contractile; they have an unstable membrane potential that drifts spontaneously to threshold and fires, and those action potentials spread through the gap junctions to the cardiomyocytes. These gap junctions are substitutes for the neuromuscular junctions found in skeletal muscle: there is no need for a synapse on every cell, because the signal readily travels cell-to-cell. The heart is myogenic, its rhythm originating from within, while skeletal muscle is neurogenic and silent without its motor neuron.

### The long duration action potential is what prevents tetanus

Third, and most important functionally, the cardiomyocyte action potential is long, roughly 250 milliseconds, with an equally long refractory period during which no new action potential can fire. Because tension develops during that prolonged action potential and the refractory period outlasts the twitch, tension falls before another stimulus can arrive, so wave summation and tetanus are physically impossible. This is functionally necessary, not a limitation. A heart that could tetanize would lock into sustained contraction, fail to relax and refill, and stop pumping. Separate twitches are precisely what this pump needs.

Metabolically, cardiac muscle is essentially the type I slow oxidative specialist, because it can never afford to fatigue. It is packed with

- Sarcomeres, striations, troponin/tropomyosin, SR  $\text{Ca}^{2+}$ , T-tubules
- Same basic crossbridge ECC mechanism

### Three Decisive Differences

- Branching single-nucleus cells joined by intercalated discs (desmosomes + gap junctions) → functional syncytium
- Myogenic: nodal pacemaker cells self-fire; gap junctions replace the NMJ
- Long AP (~250 ms) and long refractory period → no summation or tetanus

### Key Distinction: Why the Heart Cannot Tetanize

- Skeletal: short AP/refractory → summation, tetanus possible
- Cardiac: long AP/refractory → tension falls before re-stimulation
- Separate twitches let the heart relax and refill (essential for a pump)

### Derivation Chain: Why a Long Refractory Period Protects the Pump

Cardiomyocyte AP and refractory period are long (~250 ms), so no new AP can fire until the refractory period ends → the twitch's tension has already risen and fallen well within that same window → summation/tetanus impossible → heart relaxes and refills each cycle

### Common Errors

- **Error:** Thinking cardiac muscle needs a synapse on every cell. Gap junctions pass the signal cell-to-cell.
- **Error:** Assuming the heart can be driven to tetanus like skeletal muscle. Its long refractory period prevents it.

mitochondria, fed by a dense capillary bed, and stocked with myoglobin to ride out brief oxygen shortfalls, which is why it relies on aerobic metabolism. This is why narrowed or blocked coronary arteries that limit oxygen delivery are so dangerous. Although the heartbeat is self-generated, the autonomic nervous system and circulating hormones modulate rate and contraction strength, tuning output to the body's needs, with the full story reserved for Chapter 9.

- **Error:** Treating the absence of summation and tetanus in the heart as a weakness. This trait is what lets the heart refill and keep pumping.

#### Prediction Prompt

- **Predict:** Predict what would happen to cardiac output if a drug shortened the cardiomyocyte refractory period toward skeletal-muscle values.
- **Predict:** Predict why cardiac muscle, like type I skeletal fibers, is loaded with mitochondria and myoglobin.

## Section 21: Smooth Muscle — One Contractile Logic, Many Behaviors

### Instructor Narrative

***The move:** Smooth muscle utilizes the same actin-myosin mechanism as do the other muscle tissues, but the regulatory machinery is swapped out. Sarcomeres and troponin are missing and what replaces them are calmodulin and MLCK. Smooth muscle's single-unit and multiunit behaviors are results of how cells are coupled.*

#### Same engine, no sarcomeres, a different switch

Smooth muscle uses the same fundamental engine, actin and myosin sliding past each other, but reorganizes everything around it for slow, sustained, involuntary work in the walls of hollow organs. Because the filaments are not arranged into repeating sarcomeres, there are no striations. Instead, actin filaments anchor to cytoplasmic dense bodies and to membrane dense plaques, tied together by cytoskeletal intermediate filaments, so when myosin pulls, the whole cell shortens and even takes on a corkscrew shape as the tension generated is conveyed to neighboring cells. The arrangement of cytoplasmic dense bodies, membrane dense plaques, and cytoskeletal intermediate filaments found

### Structured Outline / Causal Chain

#### Same Engine, Reorganized

- Actin + myosin, but no sarcomeres → no striations
- Actin anchors to dense bodies (cytoplasm) and dense plaques (membrane)
- Intermediate filaments tie it together; force passes to adjacent cells

#### A Different Regulatory Switch

- No troponin;  $\text{Ca}^{2+}$  binds calmodulin instead
- $\text{Ca}^{2+}$ -calmodulin activates myosin light-chain kinase (MLCK)
- MLCK phosphorylates myosin heads → cycling (a thick-filament switch)
- Phosphatase reverses it; latch-bridges hold tension cheaply

#### Key Distinction: Single-Unit vs. Multiunit

in smooth muscle solve the same problem handled by costameres in skeletal muscle.

The smooth muscle regulatory switch is entirely different from the one described previously. Smooth muscle lacks troponin. Instead, when  $\text{Ca}^{2+}$  in the sarcoplasm of these cells rises it binds calmodulin, and the  $\text{Ca}^{2+}$ -calmodulin complex activates an enzyme, myosin light-chain kinase (MLCK), which phosphorylates the myosin heads. Only phosphorylated heads can cycle. So where skeletal muscle regulates by uncovering of actin's myosin head binding sites (a thin-filament switch), smooth muscle regulates by activating myosin (a thick-filament, enzyme-based switch). This also lets smooth muscle integrate many inputs, since anything that raises or lowers  $\text{Ca}^{2+}$  or alters the kinase and its opposing phosphatase changes the contraction.

### Coupling determines whether cells contract as a unit or independently

How smooth muscle behaves mechanically depends on how its cells are wired together. Single-unit smooth muscle is richly coupled by gap junctions, so action potentials, often triggered by stretch or by pacemaker-like cells, spread cell-to-cell and the tissue contracts as a coordinated whole. This is the smooth muscle of the gut wall and small blood vessels, where large sheets must act together. It behaves a bit like cardiac muscle: pacemaker input, gap-junction spread, no need for a synapse on each cell.

Multiunit smooth muscle has few gap junctions, so its cells respond individually to autonomic neurotransmitters and hormones, and force is graded by recruiting more cells, much like motor-unit recruitment in skeletal muscle. This tissue is found where localized, finely controlled contraction is needed, such as the muscles adjusting the eye's lens. A second axis sorts smooth muscle by contraction pattern: tonic smooth muscle holds prolonged tension, often using slow-cycling latch-bridges that maintain force with little energy, while phasic smooth muscle, as in the gut, contracts in rapid, intermittent bursts. The unifying message for this entire chapter is that one contractile engine, re-regulated and re-coupled, produces the entire range of muscle function in the body.

- Single-unit: many gap junctions → acts as a whole (gut, small vessels); like cardiac
- Multiunit: few gap junctions → cells act individually (eye lens); graded by recruitment, like skeletal
- Tonic (sustained, latch-bridges) vs. phasic (rapid, intermittent)

### Causal Chain: How Calcium Turns On Smooth Muscle

$\text{Ca}^{2+}$  rises (membrane channels and/or SR) →  $\text{Ca}^{2+}$  binds calmodulin →  $\text{Ca}^{2+}$ -calmodulin activates MLCK → MLCK phosphorylates myosin heads → crossbridge cycling and contraction

### Common Errors

- **Error:** Thinking smooth muscle regulates contraction with troponin. It has none; it uses calmodulin and MLCK.
- **Error:** Assuming smooth muscle regulation is on the thin filament. It is a thick-filament (myosin) switch.
- **Error:** Treating all smooth muscle alike. Single-unit vs. multiunit and tonic vs. phasic behave very differently.

### Prediction Prompt

- **Predict:** Predict how blocking myosin light-chain phosphatase would affect smooth-muscle tension, and explain.
- **Predict:** Predict whether vascular wall smooth muscle behaves more like single-unit or multiunit, and justify from its job.