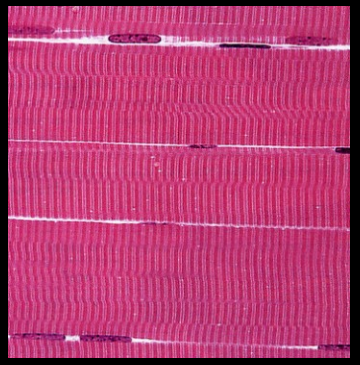
MUSCLES AND HOW THEY MOVE

Types of Muscles

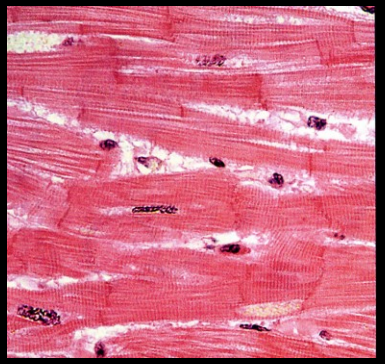
Skeletal

- Voluntary muscle; controlled consciously
- Over 600 throughout the body



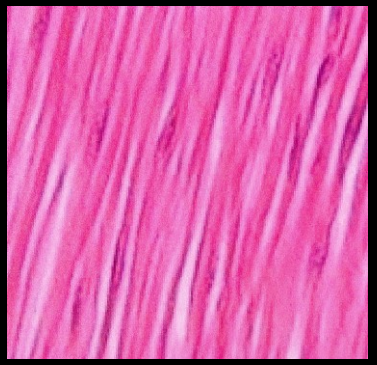
Cardiac

- Controls itself with assistance from the nervous and endocrine systems
- Only in the heart

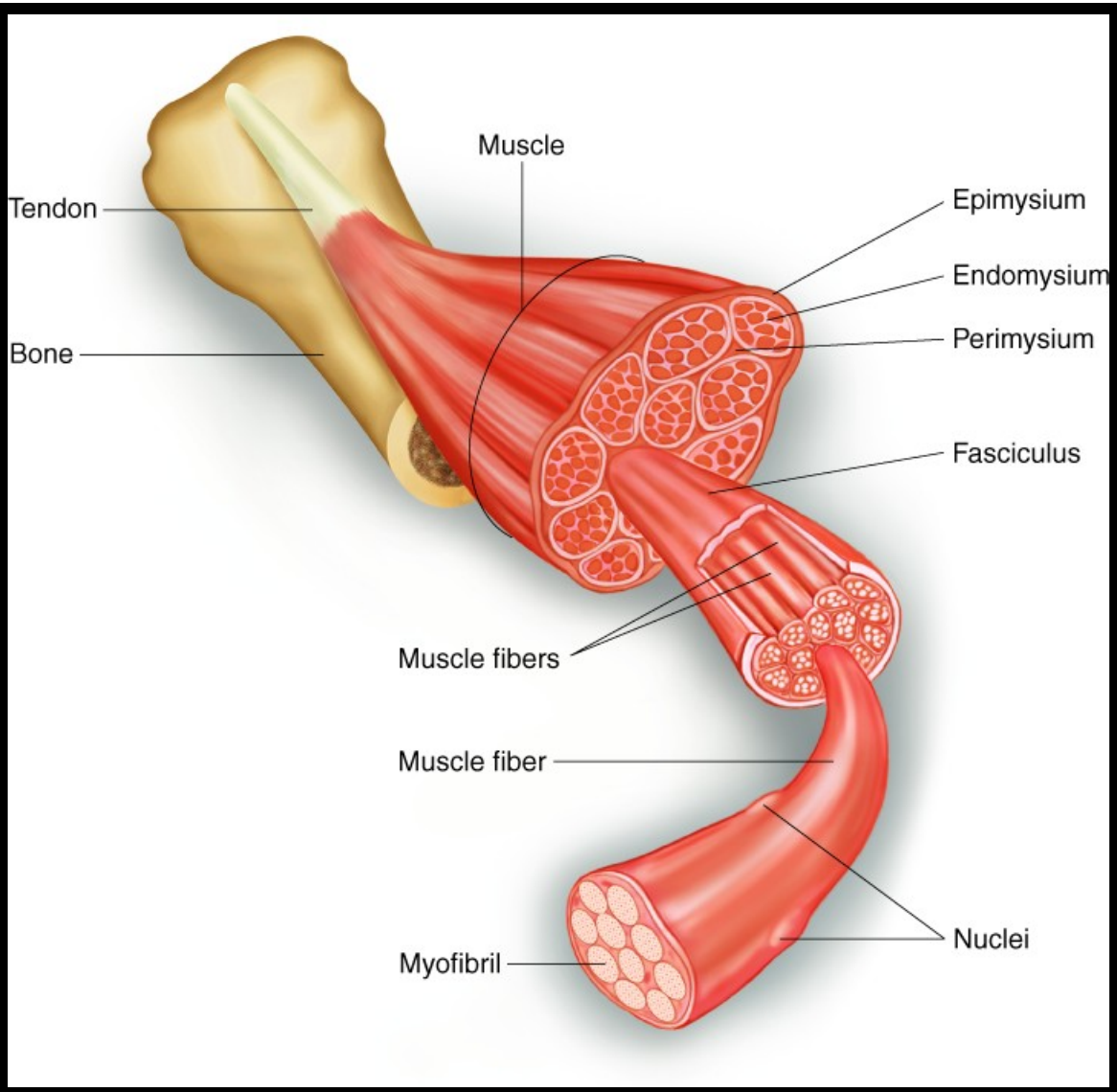


Smooth

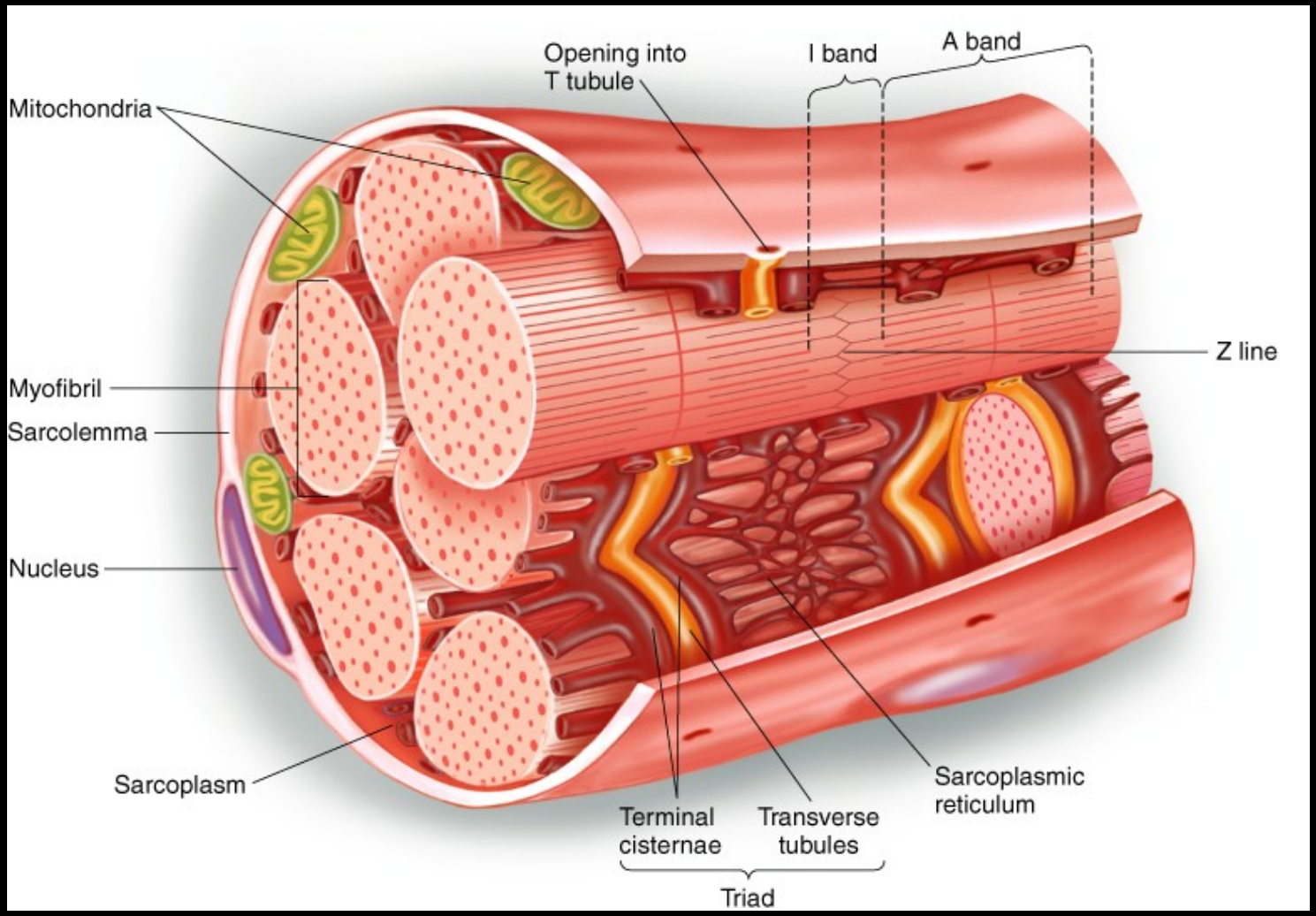
- Involuntary muscle; controlled unconsciously
- In the walls of blood vessels and internal organs



SKELETAL MUSCLE STRUCTURE

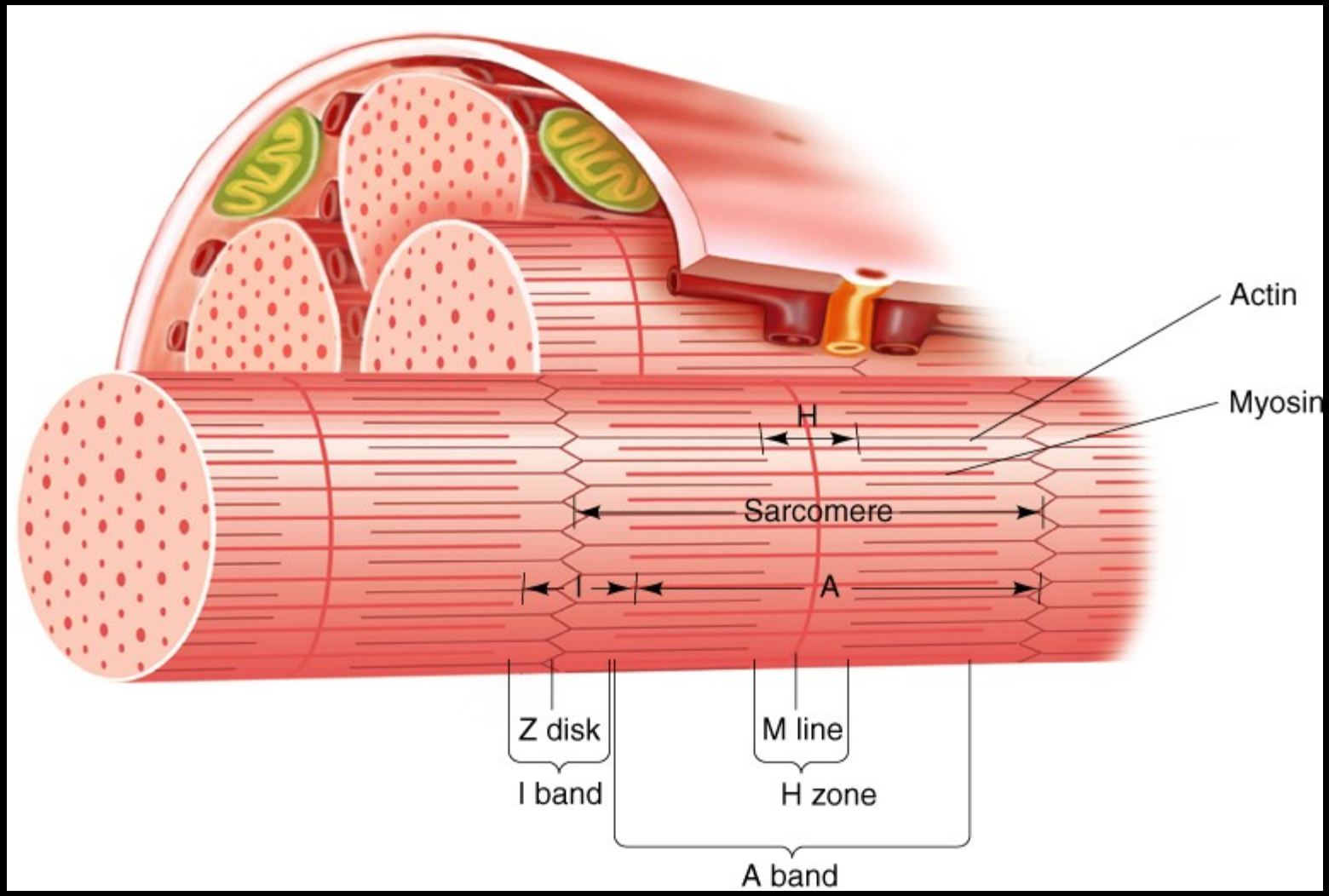


MUSCLE FIBER

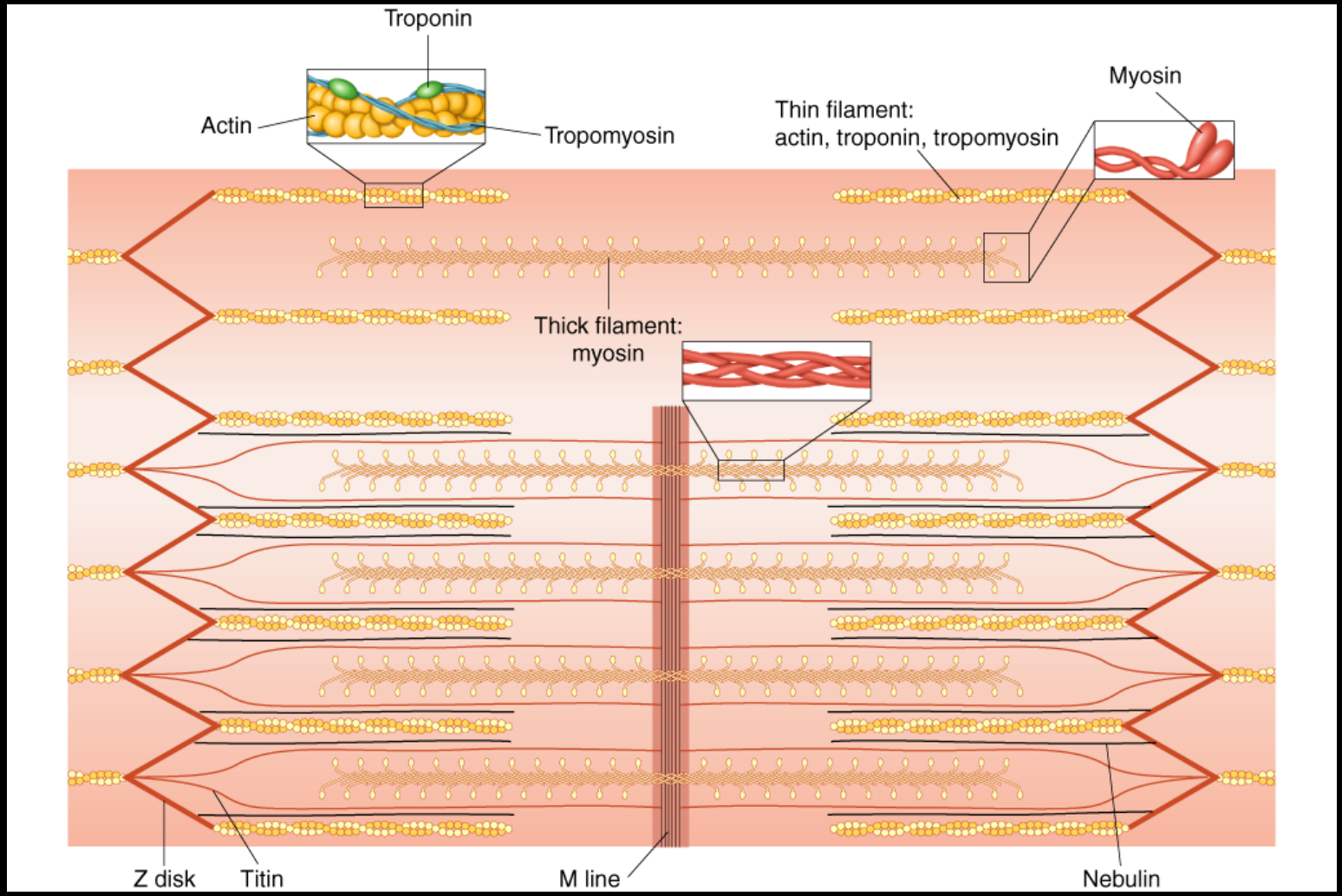


MICROGRAPH OF MYOFIBRILS

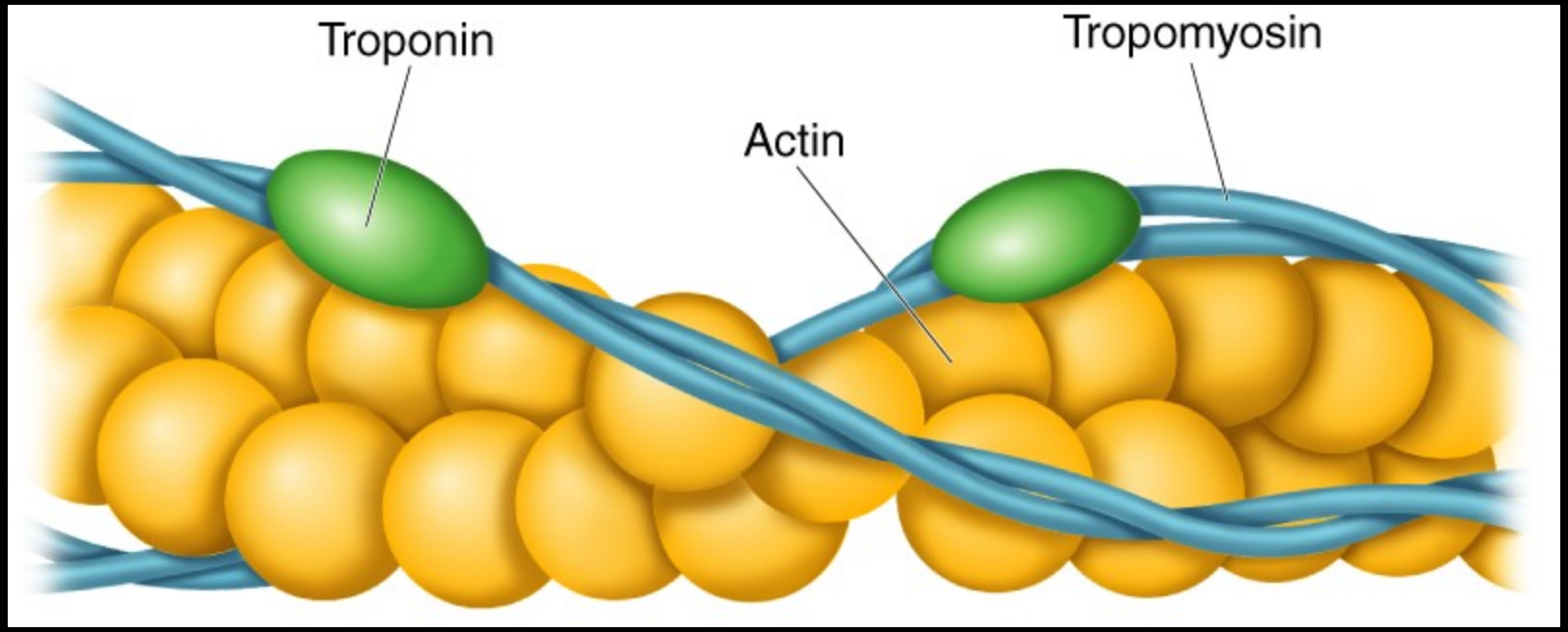
ARRANGEMENT OF FILAMENTS



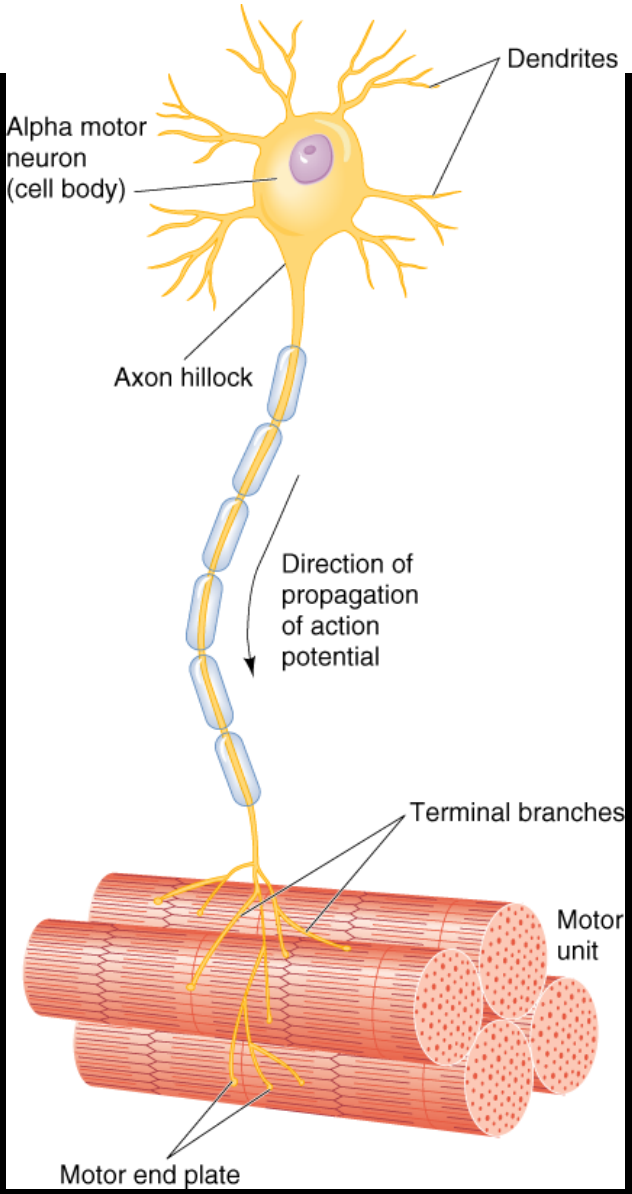
ARRANGEMENT OF FILAMENTS IN A SARCOMERE



ACTIN FILAMENT

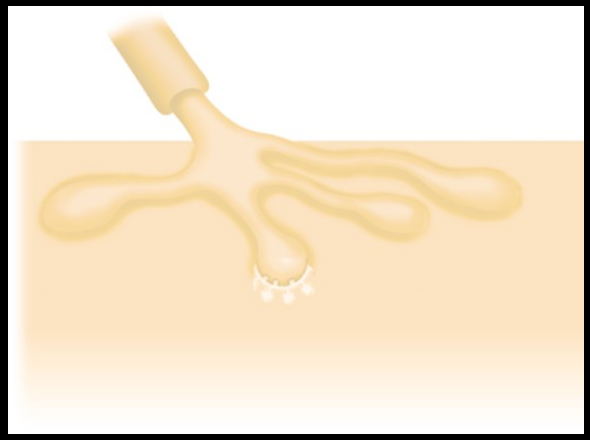


MOTOR UNIT


Dendrites
Alpha motor neuron (cell body)
Axon hillock
Direction of propagation of action potential
Terminal branches
Motor unit
Motor end plate

All-Or-None-Response

- For a motor unit to be recruited into activity the motor nerve impulse must meet or exceed the threshold.
- When this occurs, all muscle fibers in the motor unit act maximally.
- If the threshold is not met no fibers in that unit act.
- More force is produced by activating more motor units.



Excitation/Contraction Coupling

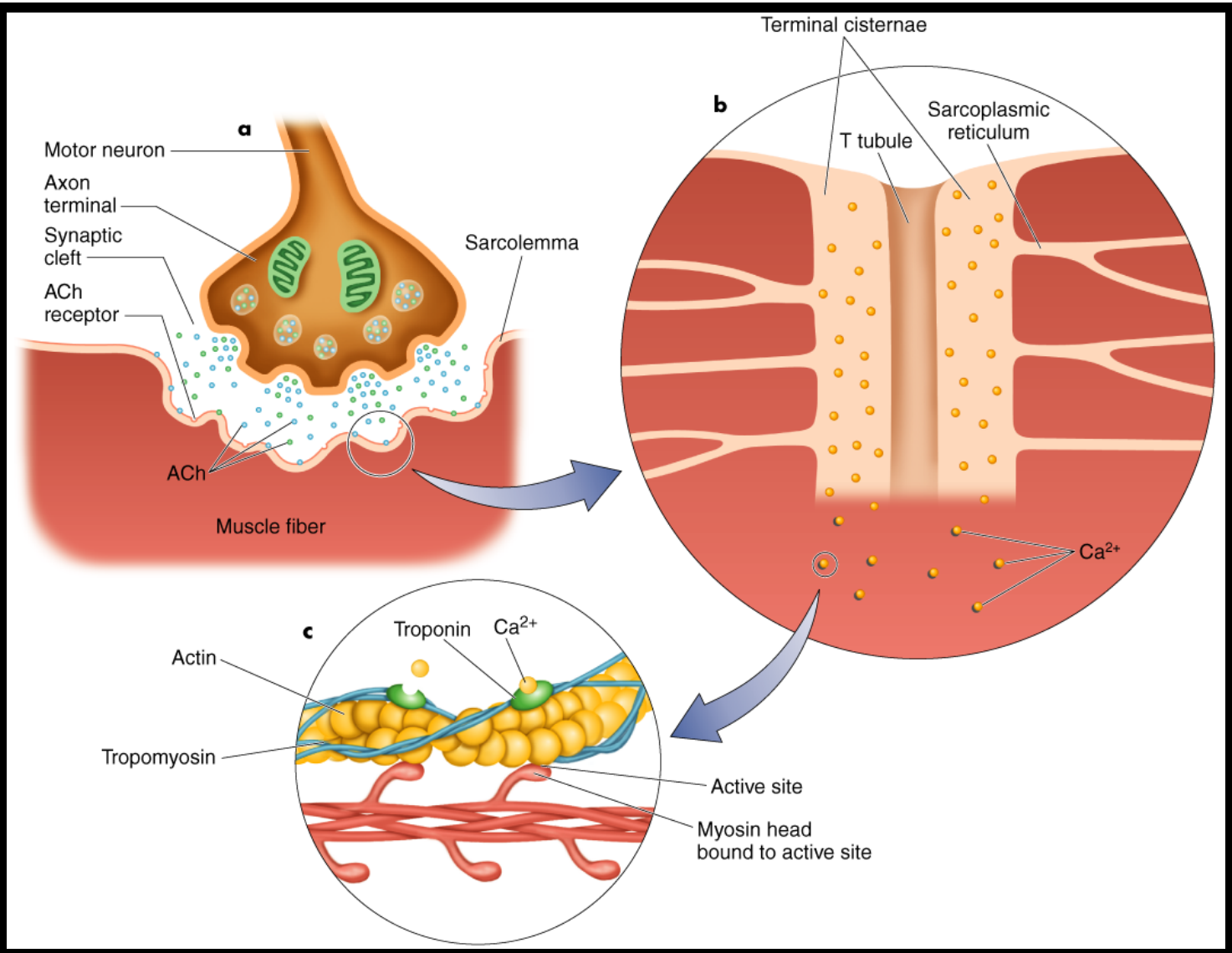
1. A motor neuron, with signals from the brain or spinal cord, releases the neurotransmitter acetylcholine (ACh) at the neuromuscular junction.
2. ACh crosses the junction and binds to receptors on the sarcolemma.
3. This initiates an action potential, providing sufficient ACh.
4. The action potential travels along the sarcolemma and through the T tubules to the SR releasing Ca^{2+}.
5. The Ca^{2+} binds to troponin on the actin filament, and the troponin pulls tropomyosin off the active sites, allowing myosin heads to attach to the actin filament.

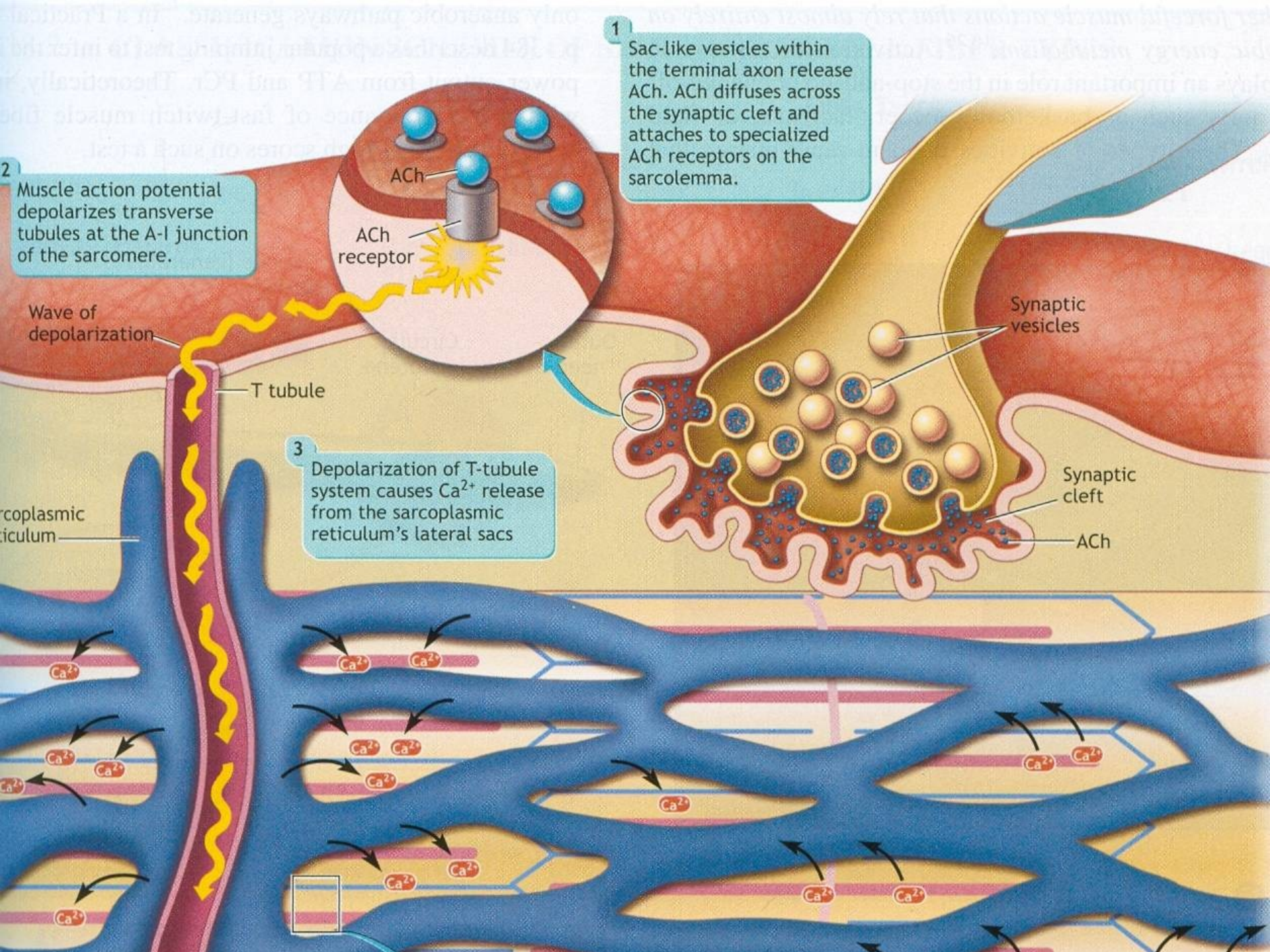
(continued)

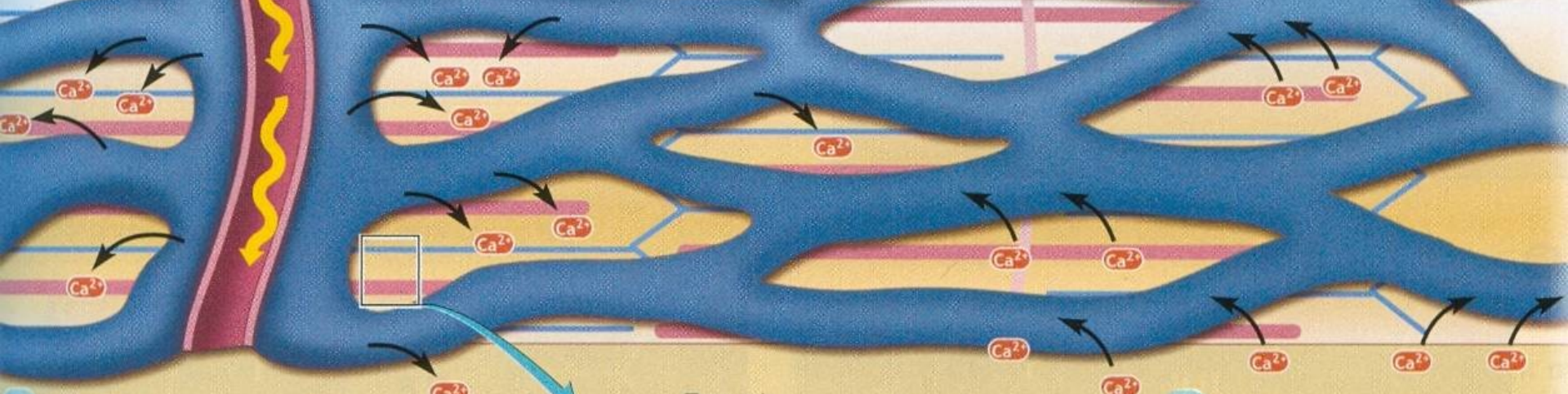
Excitation/Contraction Coupling

6. Once a strong binding state is established with actin, the myosin head tilts, pulling the actin filament (power stroke).
7. The myosin head binds to ATP, and ATPase found on the head splits ATP into ADP and P_i, releasing energy.
8. The new ATP binding releases the myosin head from the actin molecule.
9. Muscle action ends when calcium is actively pumped out of the sarcoplasm back into the sarcoplasmic reticulum for storage.

EVENTS LEADING TO MUSCLE ACTION



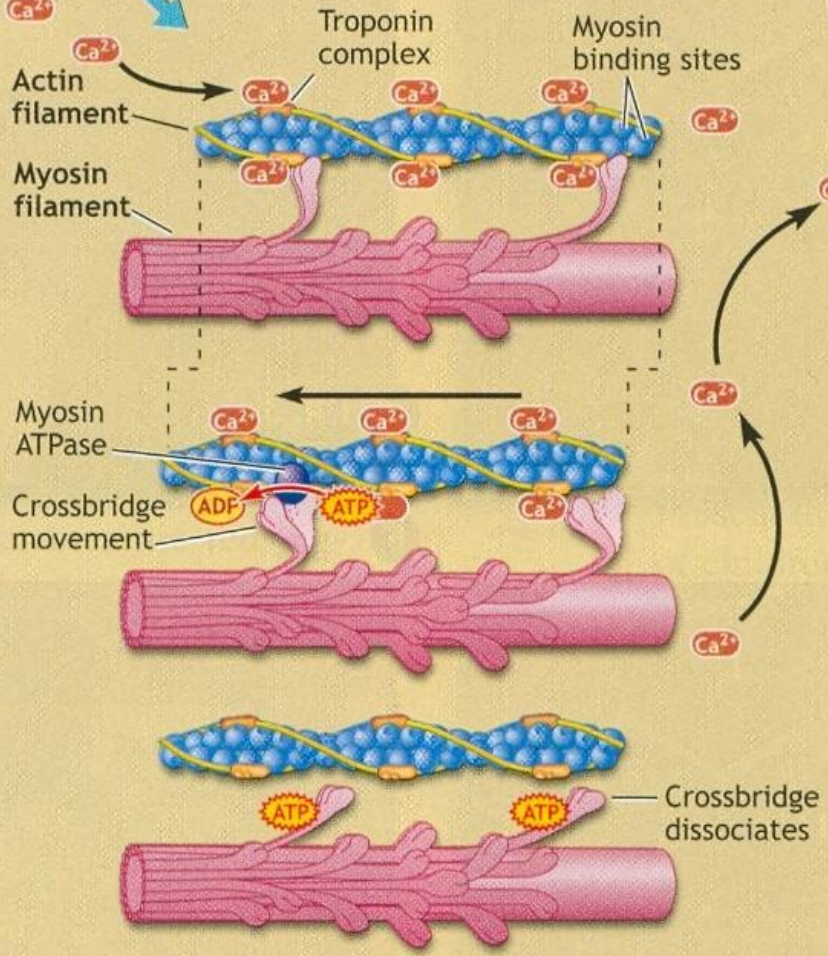

1
Sac-like vesicles within the terminal axon release ACh. ACh diffuses across the synaptic cleft and attaches to specialized ACh receptors on the sarcolemma.
2
Muscle action potential depolarizes transverse tubules at the A-I junction of the sarcomere.
3
Depolarization of T-tubule system causes Ca^{2+} release from the sarcoplasmic reticulum's lateral sacs
ACh
ACh receptor
Wave of depolarization
T tubule
Synaptic vesicles
Synaptic cleft
ACh
rcoplasmic
ticulum
Ca^{2+}



4 Ca^{2+} binds to troponin-tropomyosin in the actin filaments. This releases the inhibition that prevented actin from combining with myosin.

5 During muscle action, actin combines with myosin ATPase to split ATP with energy release. Tension created from the energy release produces myosin crossbridge movement.

6 ATP binds to the myosin crossbridge, breaking the actin–myosin bond allowing the crossbridge to dissociate from actin. This leads to sliding of thick and thin filaments, causing muscle shortening.



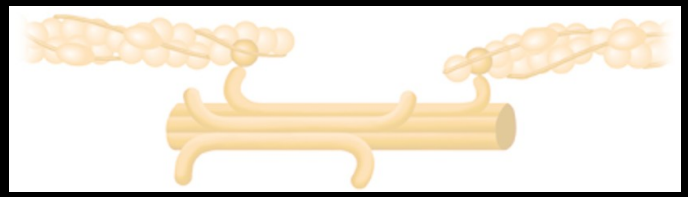
9 Ca^{2+} removal restores inhibitory action of troponin–tropomyosin. In the presence of ATP, actin and myosin remain in the dissociated relaxed state.

8 When muscle stimulation ceases, Ca^{2+} moves back into the sarcoplasmic reticulum's lateral sacs through active transport requiring ATP hydrolysis.

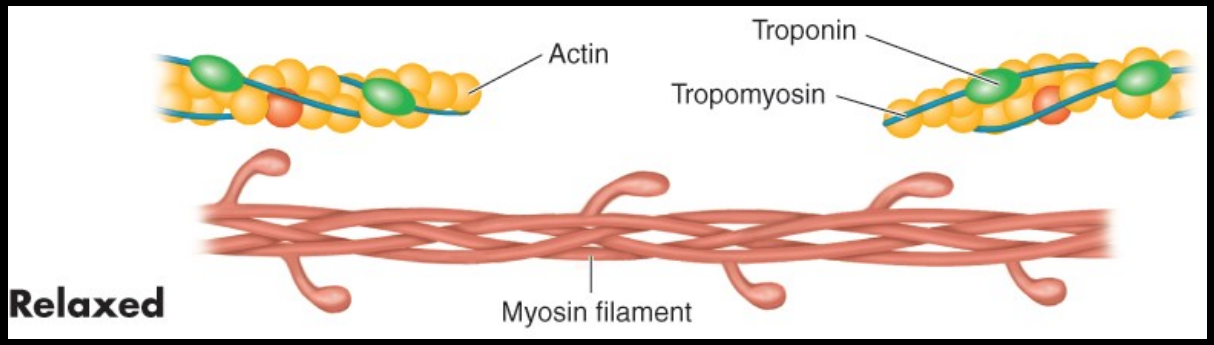
7 Crossbridge activation continues when the concentration of Ca^{2+} remains high (from membrane depolarization) to inhibit action of the troponin–tropomyosin complex.

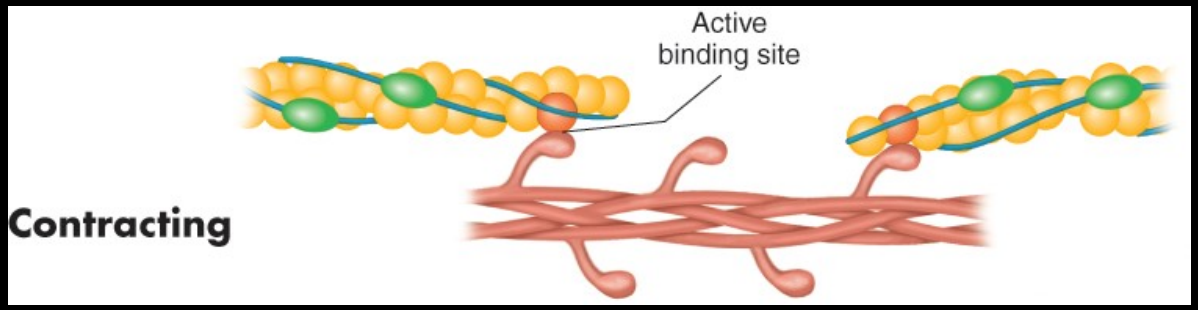
Sliding Filament Theory

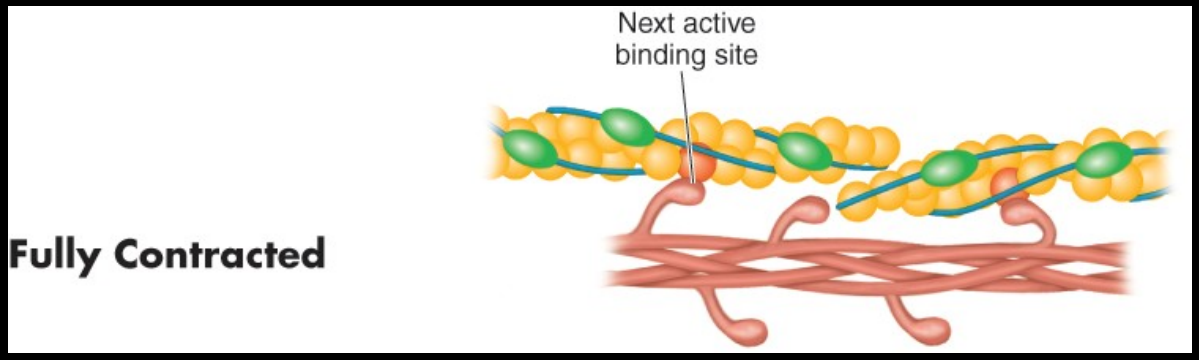
- When myosin cross-bridges are activated, they bind strongly with actin, resulting in a change in the cross-bridge.
- The change in the cross-bridge causes the myosin head to tilt toward the arm of the cross-bridge and drag the actin and myosin filaments in opposite directions.
- The tilt of the myosin head is known as a *power stroke*.
- The pulling of the actin filament past the myosin results in muscle shortening and generation of muscle force.



CONTRACTING MUSCLE FIBER







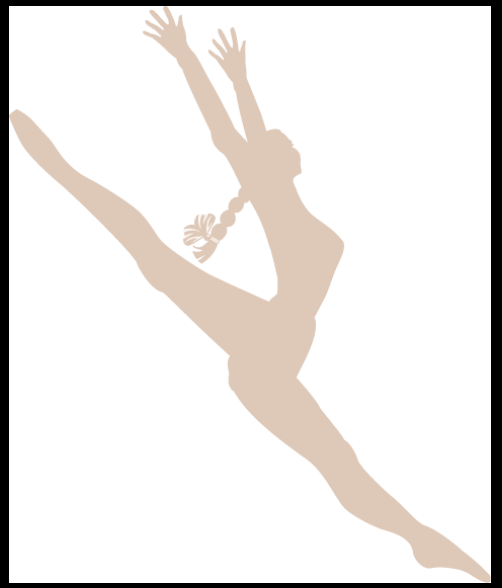
https://www.youtube.com/watch?v=BVcgO4p88AA

Functional Classification of Muscles

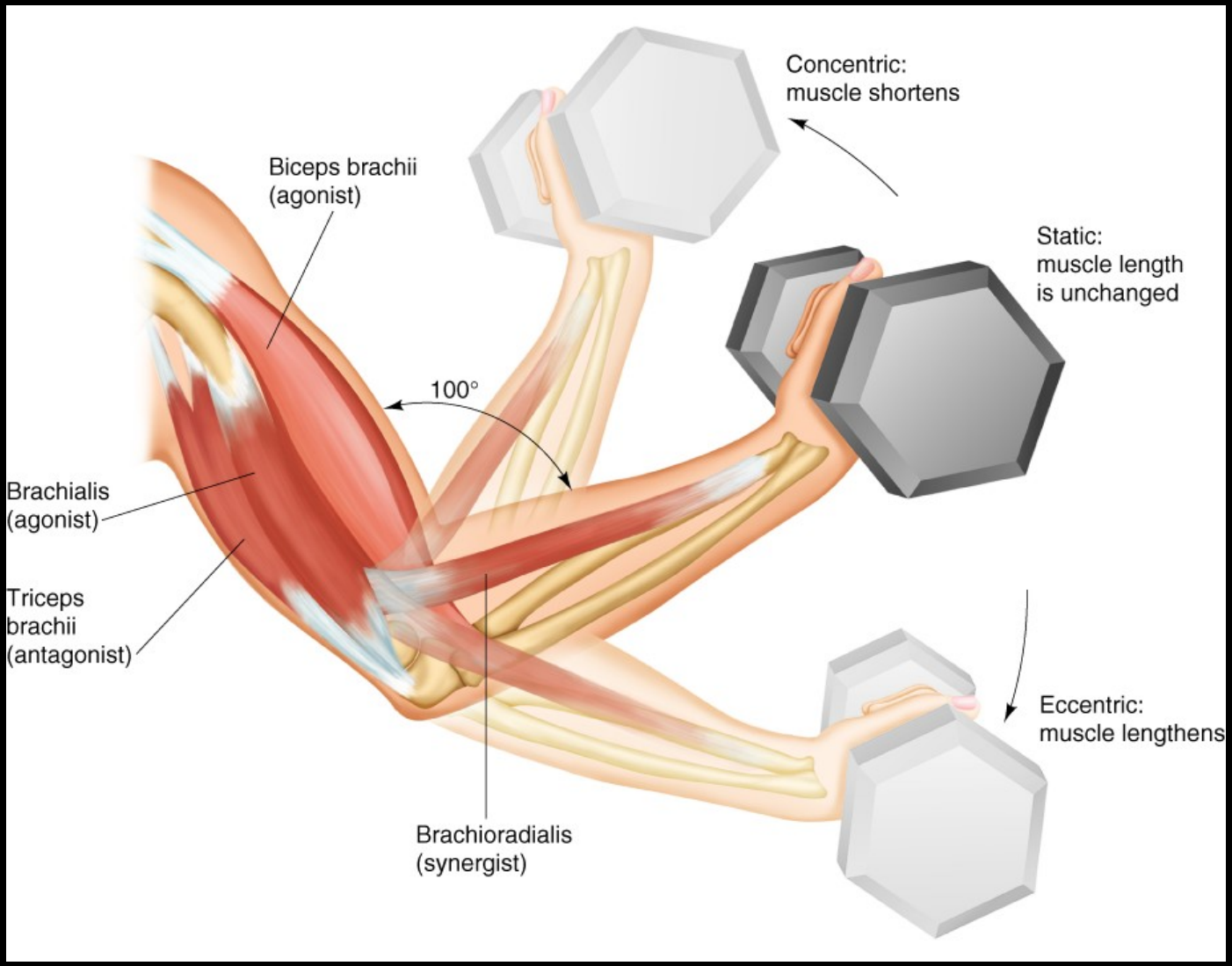
Agonists—prime movers; responsible for the movement

Antagonists—oppose the agonists to prevent overstretching of them

Synergists—assist the agonists and sometimes fine-tune the direction of movement

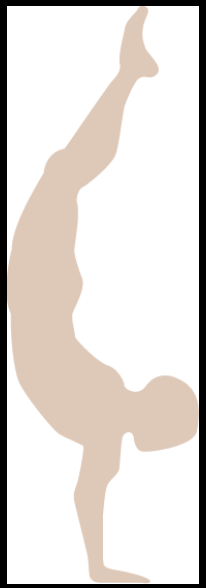


TYPES OF MUSCLE ACTION



Factors Influencing Force Generation

- Number of motor units activated
- Type of motor units activated (FT or ST)
- Muscle size
- Initial muscle length
- Joint angle
- Speed of muscle action (shortening or lengthening)



MUSCLE FIBRE TYPES

Slow-Twitch (ST; red) Muscle Fibers

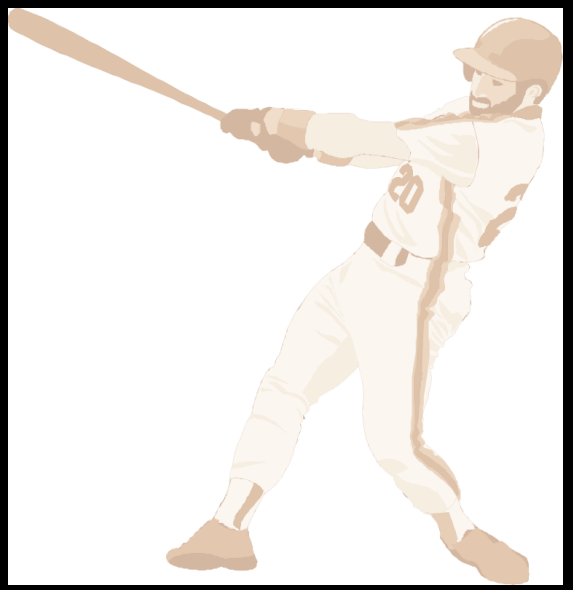
- High aerobic (oxidative) capacity and fatigue resistance
- Low anaerobic (glycolytic) capacity and motor unit strength
- Slow contractile speed (110 ms to reach peak tension) and myosin ATPase
- 10–180 fibers per motor neuron
- Low sarcoplasmic reticulum development

Fast-Twitch (FT_a; white) Muscle Fibers

- Moderate aerobic (oxidative) capacity and fatigue resistance
- High anaerobic (glycolytic) capacity and motor unit strength
- Fast contractile speed (50 ms to reach peak tension) and myosin ATPase
- 300–800 fibers per motor neuron
- High sarcoplasmic reticulum development

Fast-Twitch (FT_b; white) Muscle Fibers

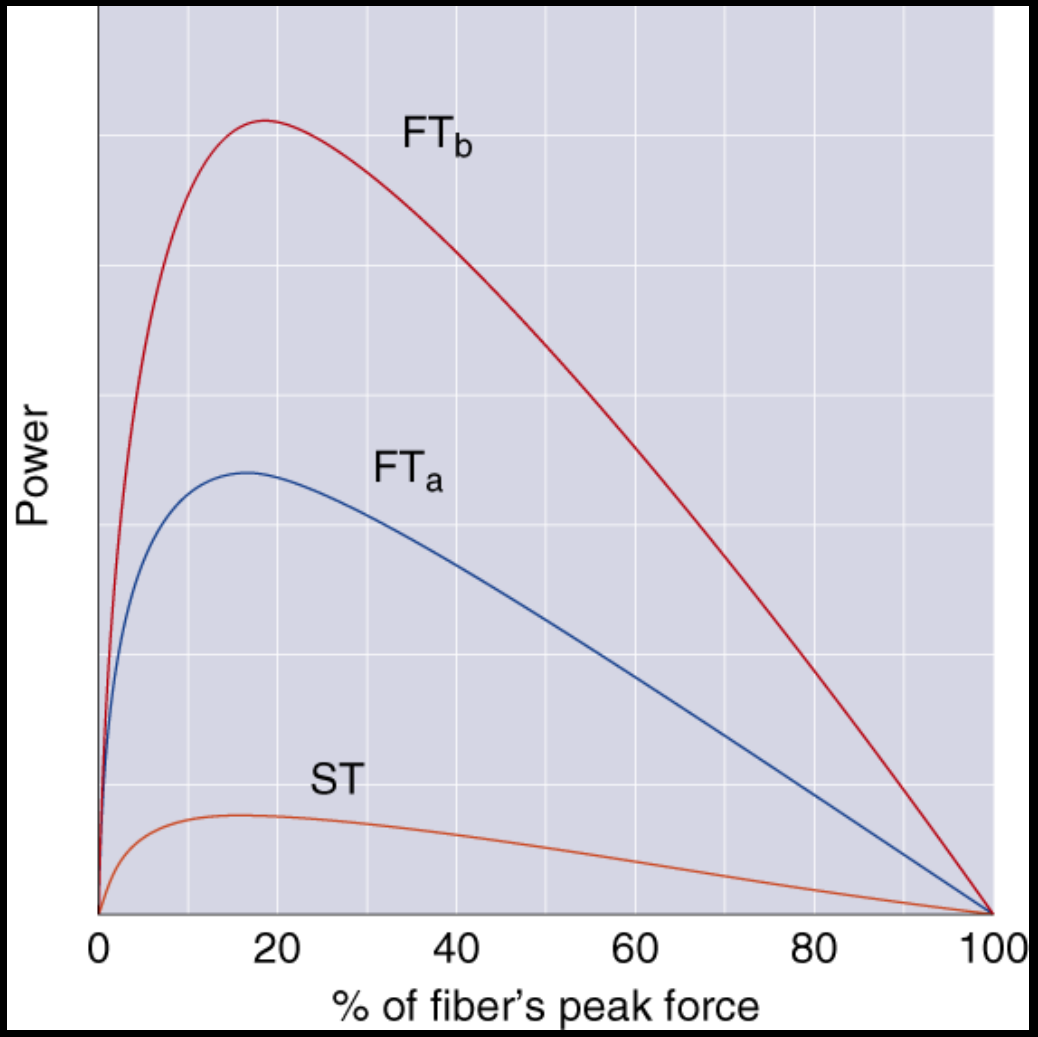
- Low aerobic (oxidative) capacity and fatigue resistance
- Highest anaerobic (glycolytic) capacity and motor unit strength
- Fast contractile speed (50 ms to reach peak tension) and myosin ATPase
- 300–800 fibers per motor neuron
- High sarcoplasmic reticulum development



Main characteristics of muscle fibre types

	Slow-twitch (ST)	Fast-twitch (FTa)	Fast-twitch (FTb)
Contraction speed	low	high	high
Contraction power	low	medium	high
Fatigue resistance	high	medium	low
Glycogen volume	low	high	high
Diameter	low	medium	high
Mitochondrial density	high	high	low
Capillary density	high	high	low
ATPase activity	low	high	high
Glycolytic capacity	low	high	high

PEAK POWER GENERATED BY FIBERS

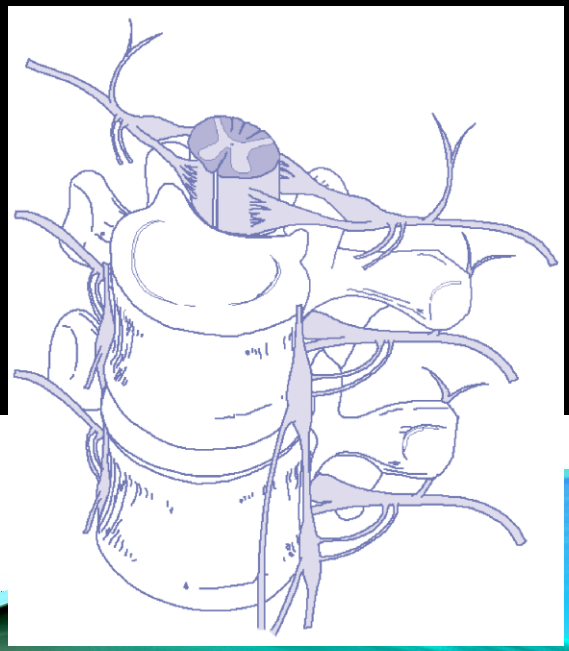


What Determines Fiber Type?

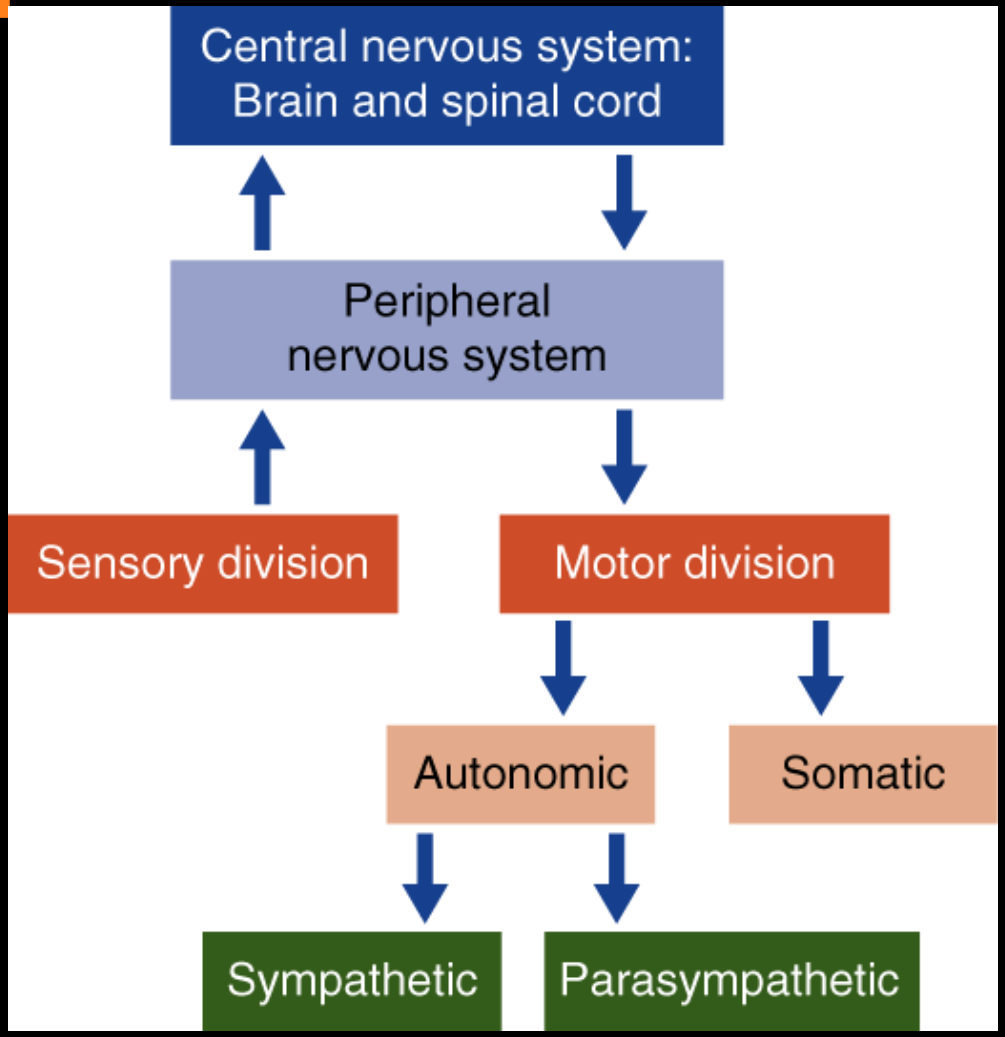
- Genetics determine which type of motor neurons innervate our individual muscle fibers.
- Muscle fibers become specialized according to the type of neuron that stimulates them.
- Endurance training, strength training, and muscular inactivity may result in small changes (less than 10%) in the percentage of FT and ST fibers.
- Endurance training has been shown to reduce the percentage of FT_b fibers, while increasing the fraction of FT_a fibers.
- Aging may result in changes in the percentage of FT and ST fibers.

CHAPTER

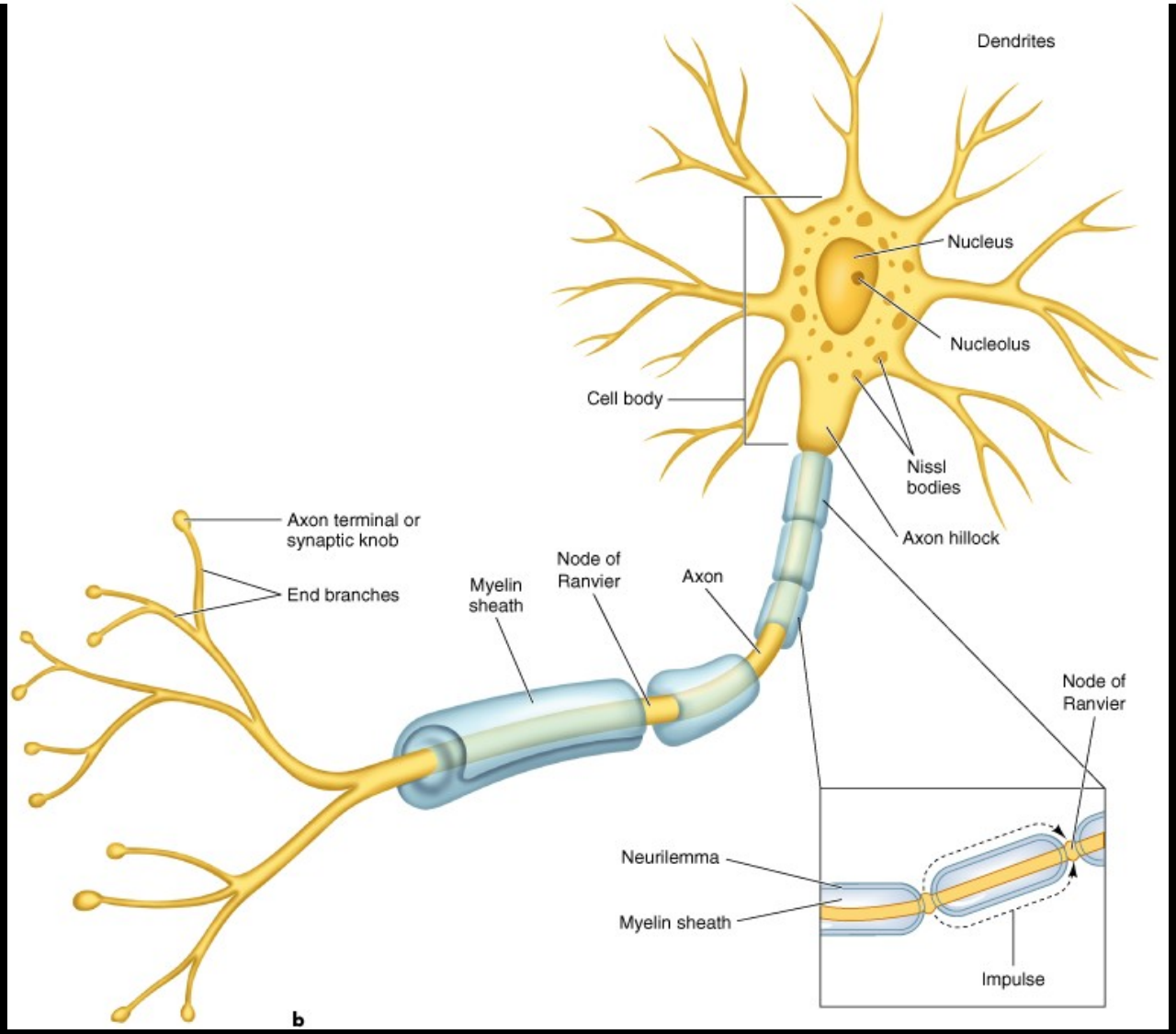
NEUROLOGICAL CONTROL OF MOVEMENT



ORGANIZATION OF THE NERVOUS SYSTEM

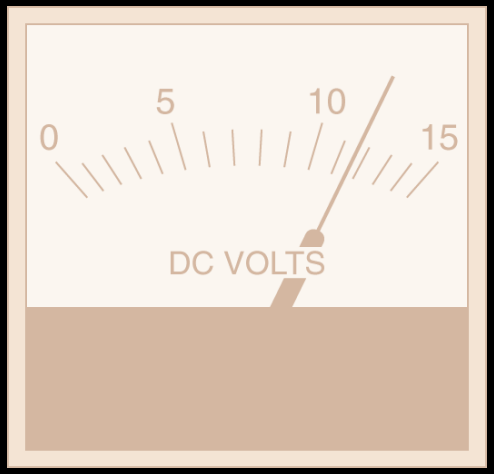


STRUCTURE OF A NEURON



Nerve Impulse

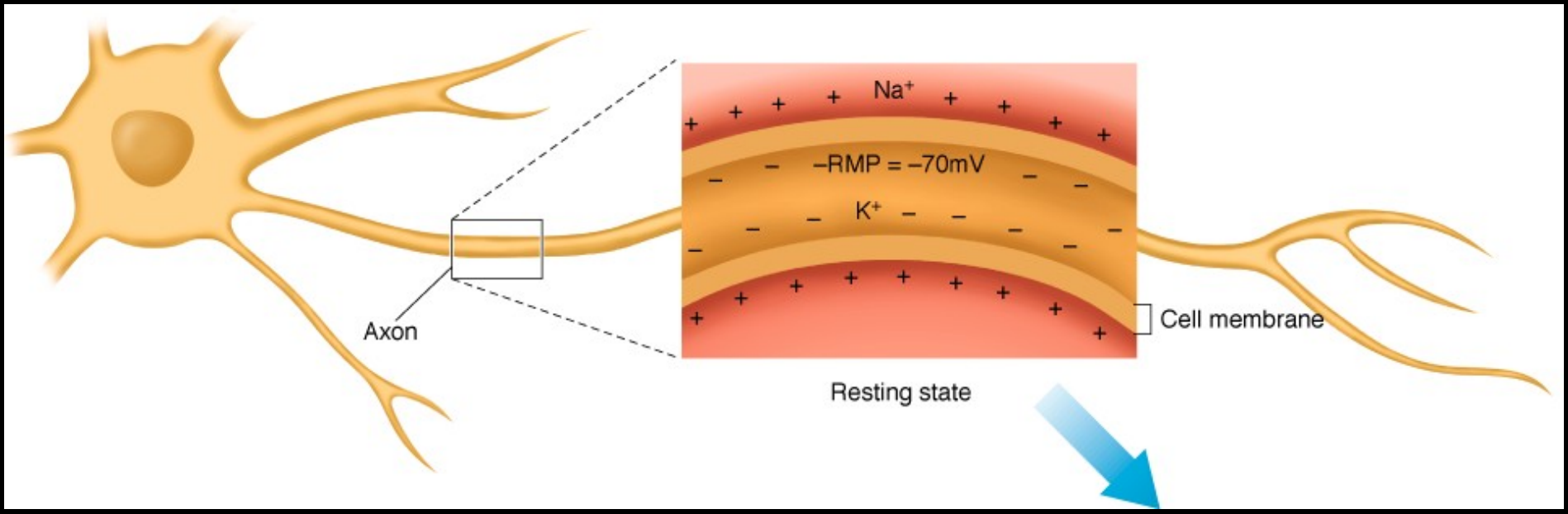
An electrical charge that passes from one neuron to the next and finally to an end organ, such as a group of muscle fibers.



Resting Membrane Potential (RMP)

- Difference between the electrical charges inside and outside a cell, caused by separation of charges across a membrane
- High concentration of K^+ inside the neuron and Na^+ outside the neuron
- K^+ ions can move freely, even outside the cell to help maintain imbalance
- Sodium-potassium pump actively transports K^+ and Na^+ ions to maintain imbalance
- The constant imbalance keeps the RMP at –70 mV

RESTING STATE



Changes in Membrane Potential

Depolarization—inside of cell becomes less negative relative to outside (> –70 mV)

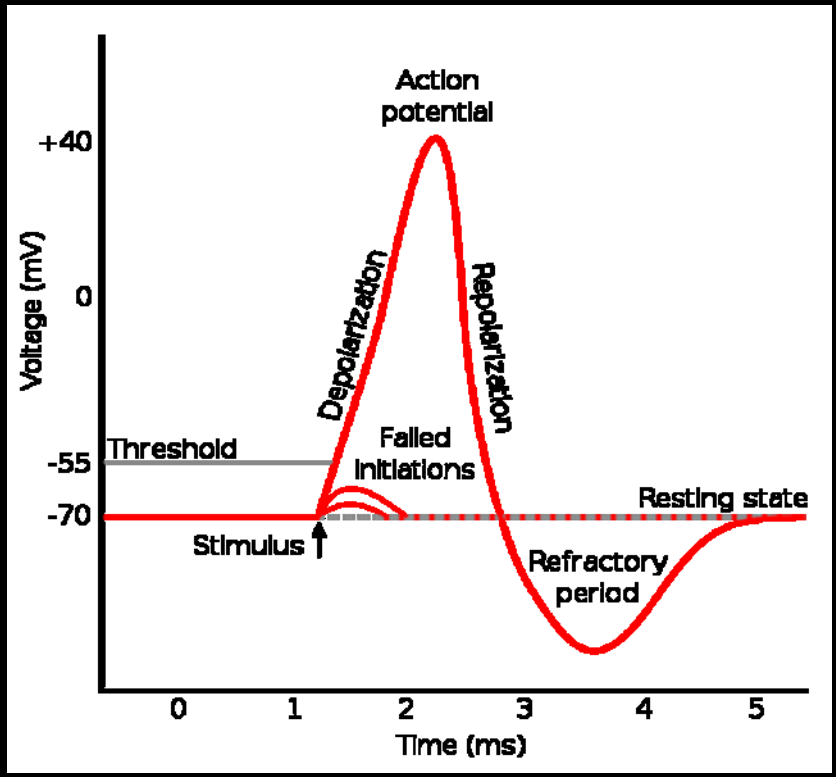
Hyperpolarization—inside of cell becomes more negative relative to outside (< –70 mV)

Graded potentials—localized changes in membrane potential (either depolarization or hyperpolarization)

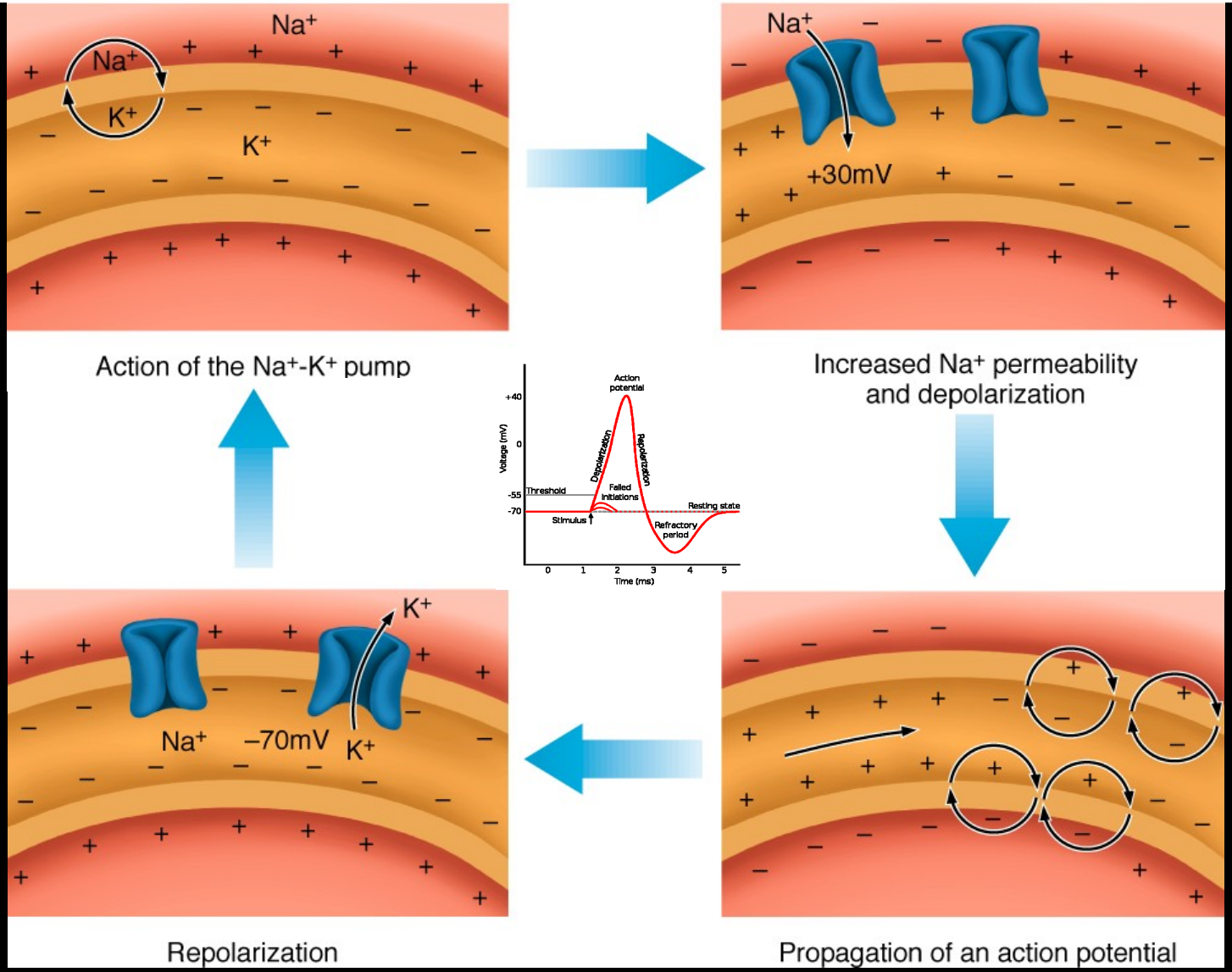
Action potentials—rapid, substantial depolarization of the membrane (–70 mV to +30 mV to –70 mV all in 1 ms)

What Is an Action Potential?

- Starts as a graded potential
- Requires depolarization greater than the threshold value of 15 mV to 20 mV (e.g., –50 to –55 mV)
- Once threshold is met or exceeded, the all-or-none principle applies



ACTION POTENTIAL



Events During an Action Potential

1. The resting state
2. Depolarization
3. Propagation of an action potential
4. Repolarization
5. Return to the resting state with the help of the sodium-potassium pump

Velocity of an Action Potential

Myelinated fibers

- Saltatory conduction—action potential travels quickly from one break in myelin to the next.
- Action potential is 5 to 150 times faster in myelinated compared to unmyelinated axons.

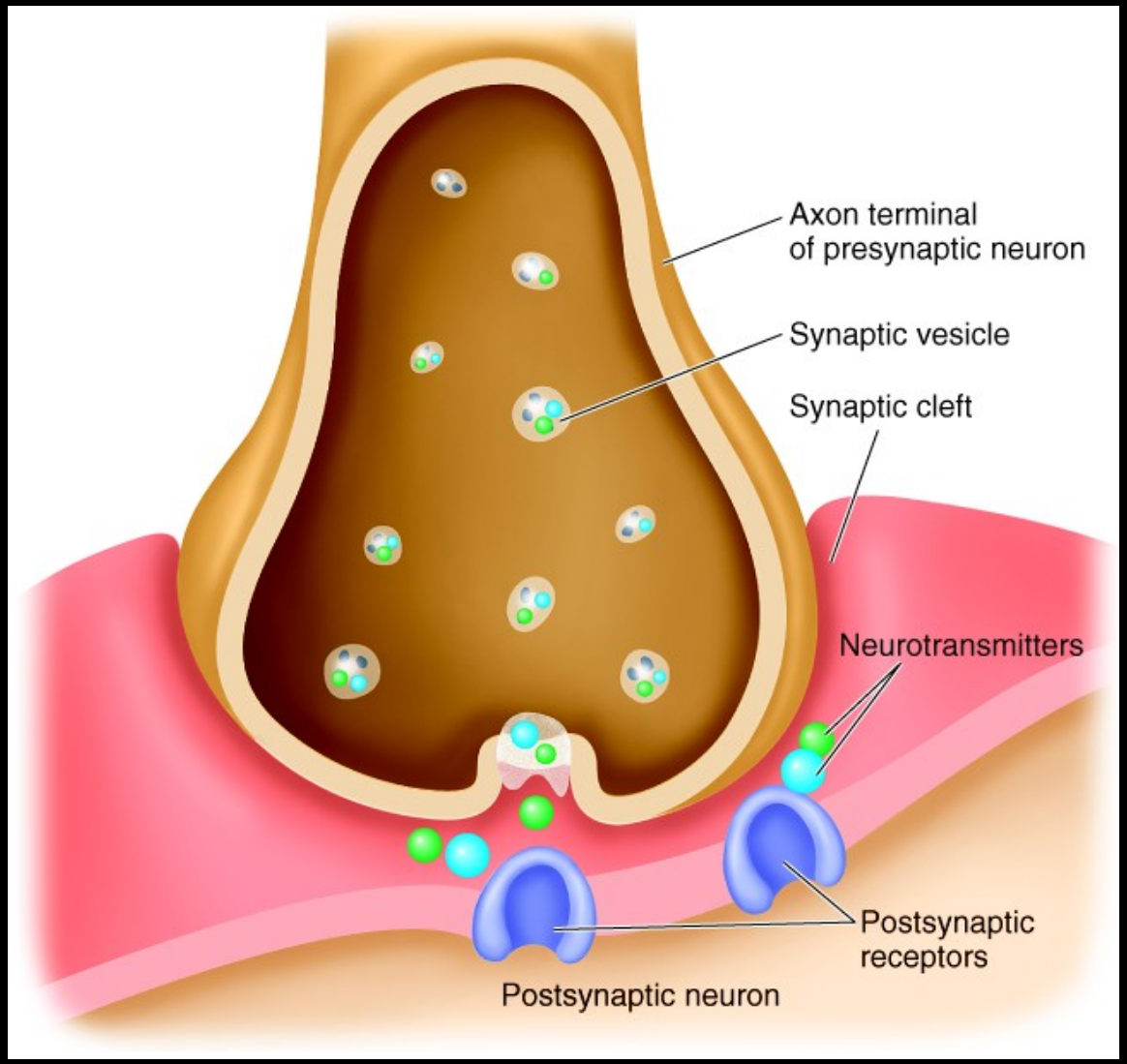
Diameter of the neuron

- Larger diameter neurons conduct nerve impulses faster.
- Larger diameter neurons present less resistance to current flow (remember FT muscle fibers!).

Synapse

- A synapse is the site of an impulse transmission between two neurons.
- An impulse travels to a presynaptic axon terminal where it causes synaptic vesicles on the terminal to release chemicals (neurotransmitters) into the synaptic cleft.
- The neurotransmitters bind to postsynaptic receptors on an adjacent neuron usually on the dendrites (80–95%).
- Neural impulses can only be transmitted from the dendrite or cell body through the axon to the adjacent neuron since the neurotransmitters are released only from the terminal end of the axon.

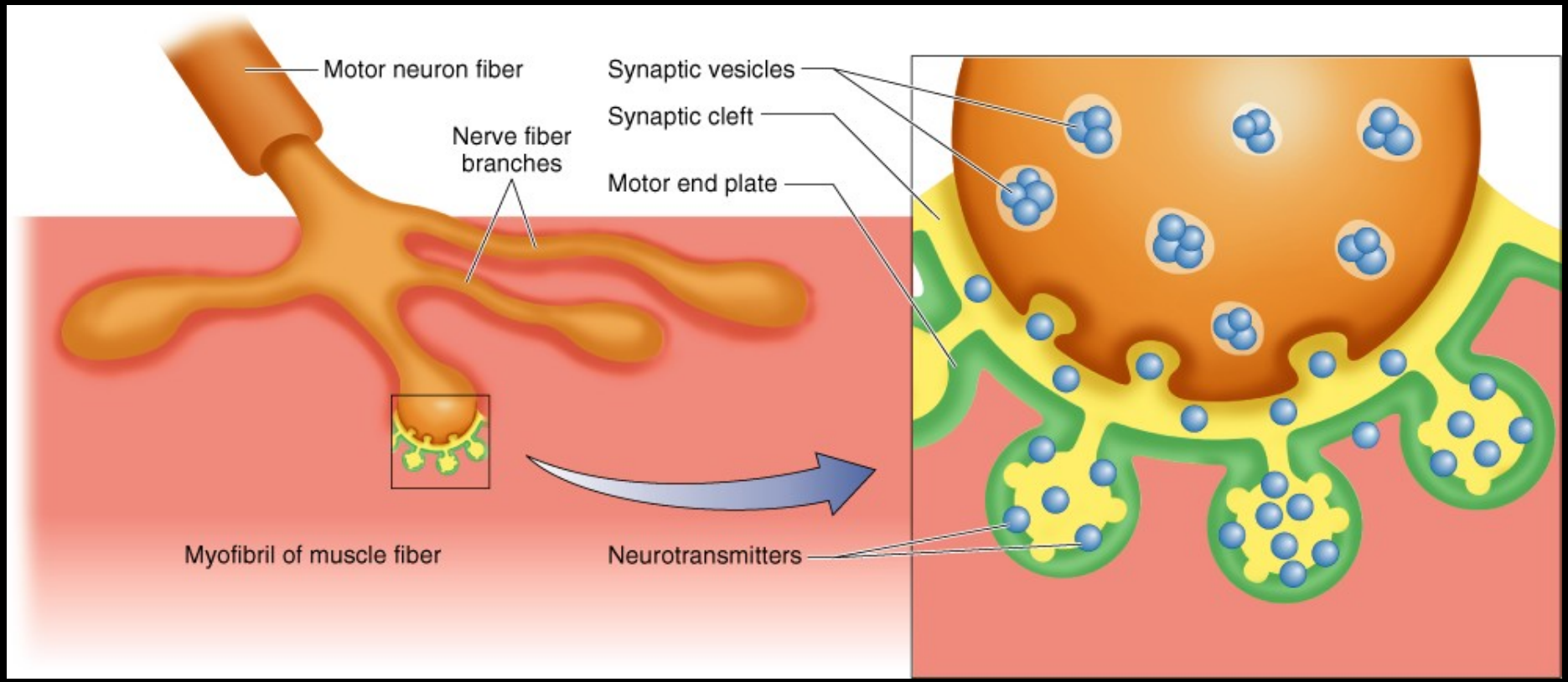
CHEMICAL SYNAPSE



Neuromuscular Junction

- The junction is a site where a motor neuron communicates with a muscle fiber.
- Motor axon terminal releases neurotransmitters (such as acetylcholine or norepinephrine) which travel across a synaptic cleft and bind to receptors on a muscle fiber.
- This binding causes depolarization, thus possibly causing an action potential.
- The action potential spreads across the sarcolemma into the T tubules causing the muscle fiber to contract.

NEUROMUSCULAR JUNCTION



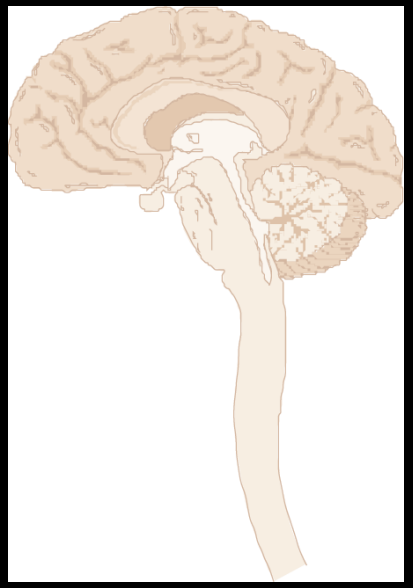
Refractory Period

- Period of repolarization.
- The muscle fiber is unable to respond to any further stimulation.
- The refractory period limits a motor unit's firing frequency.

Central Nervous System

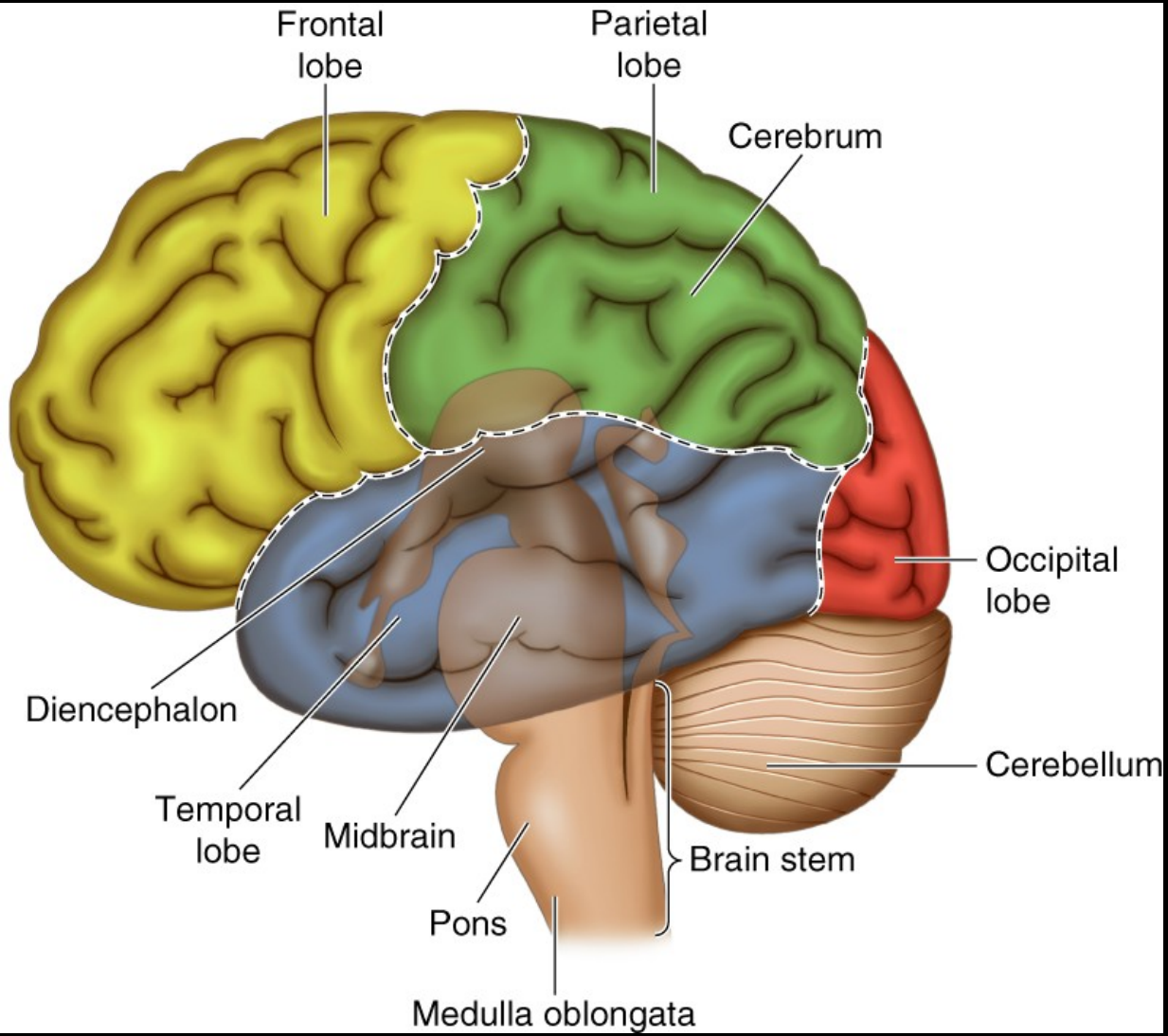
Brain

- Cerebrum—Site of the mind and intellect.
- Diencephalon—Site of sensory integration and regulation of homeostasis.
- Cerebellum—Plays crucial role in coordinating movement.
- Brain stem—Connects brain to spinal cord; coordinates skeletal muscle function and maintains muscle tone; contains regulators of respiratory and cardiovascular systems.



Spinal cord

REGIONS OF THE BRAIN



Peripheral Nervous System

- 12 pairs of cranial nerves connected with the brain.
- 31 pairs of spinal nerves connected with the spinal cord.
- Sensory division—carries sensory information from the body via afferent fibers to the CNS.
- Motor division—transmits information from CNS via efferent fibers to target organs.
- Autonomic nervous system—controls involuntary internal functions.

Sympathetic Nervous System

Fight-or-flight—prepares you for acute stress or physical activity

Facilitates your motor response with increases in

- Heart rate and strength of heart contraction
- Blood pressure
- Blood supply to the heart and active muscles
- Metabolic rate and release of glucose by the liver
- Rate of gas exchange between lungs and blood
- Mental activity and quickness of response

Parasympathetic Nervous System

Housekeeping (rest-or-digest)—digestion, urination, glandular secretion, and energy conservation

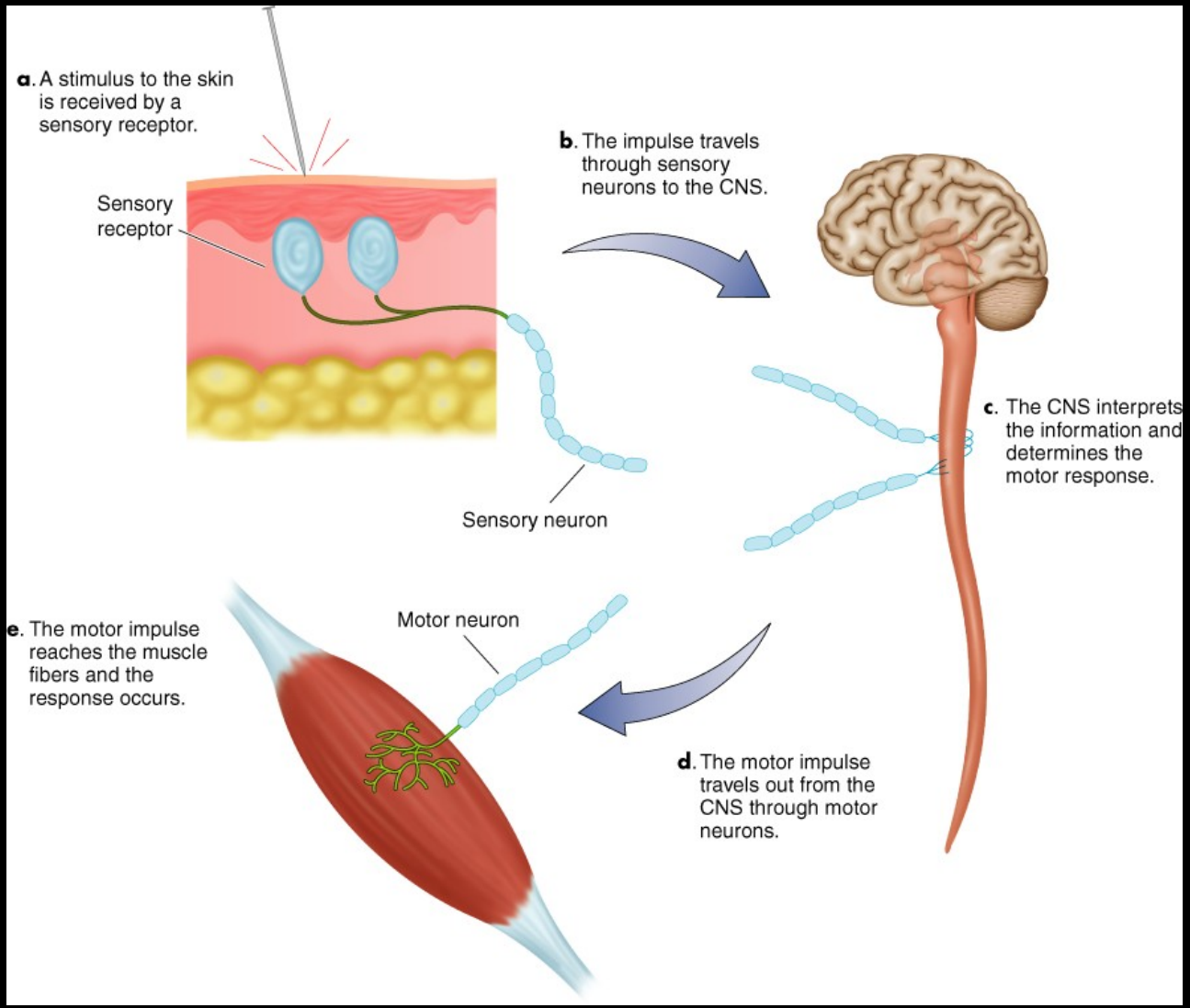
Actions oppose those of the sympathetic system

- Decreases heart rate
- Constricts coronary vessels
- Constricts tissues in the lungs
- Stimulates sexual functions
- Digestion

Motor Control

- Sensory impulses evoke a response through a motor neuron.
- The closer to the brain the impulse stops, the more complex the motor reaction.
- A motor reflex is a preprogrammed response that is integrated by the spinal cord without conscious thought.

SENSORY-MOTOR INTEGRATION



Types of Sensory Receptors

Mechanoreceptors—respond to mechanical forces such as pressure, touch, vibration, or stretch.

Thermoreceptors—respond to changes in temperature.

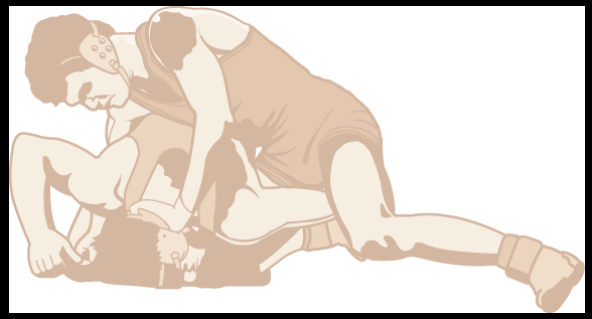
Nociceptors—respond to painful stimuli.

Photoreceptors—respond to light to allow vision.

Chemoreceptors—respond to chemical stimuli from foods, odors, and changes in blood concentrations of gases and substances.

Muscle and Joint Nerve Endings

- Joint kinesthetic receptors in joint capsules sense the position and movement of joints.
- Muscle spindles sense how much a muscle is stretched.
- Golgi tendon organs detect the tension of a muscle on its tendon, providing information about the strength of muscle contraction.



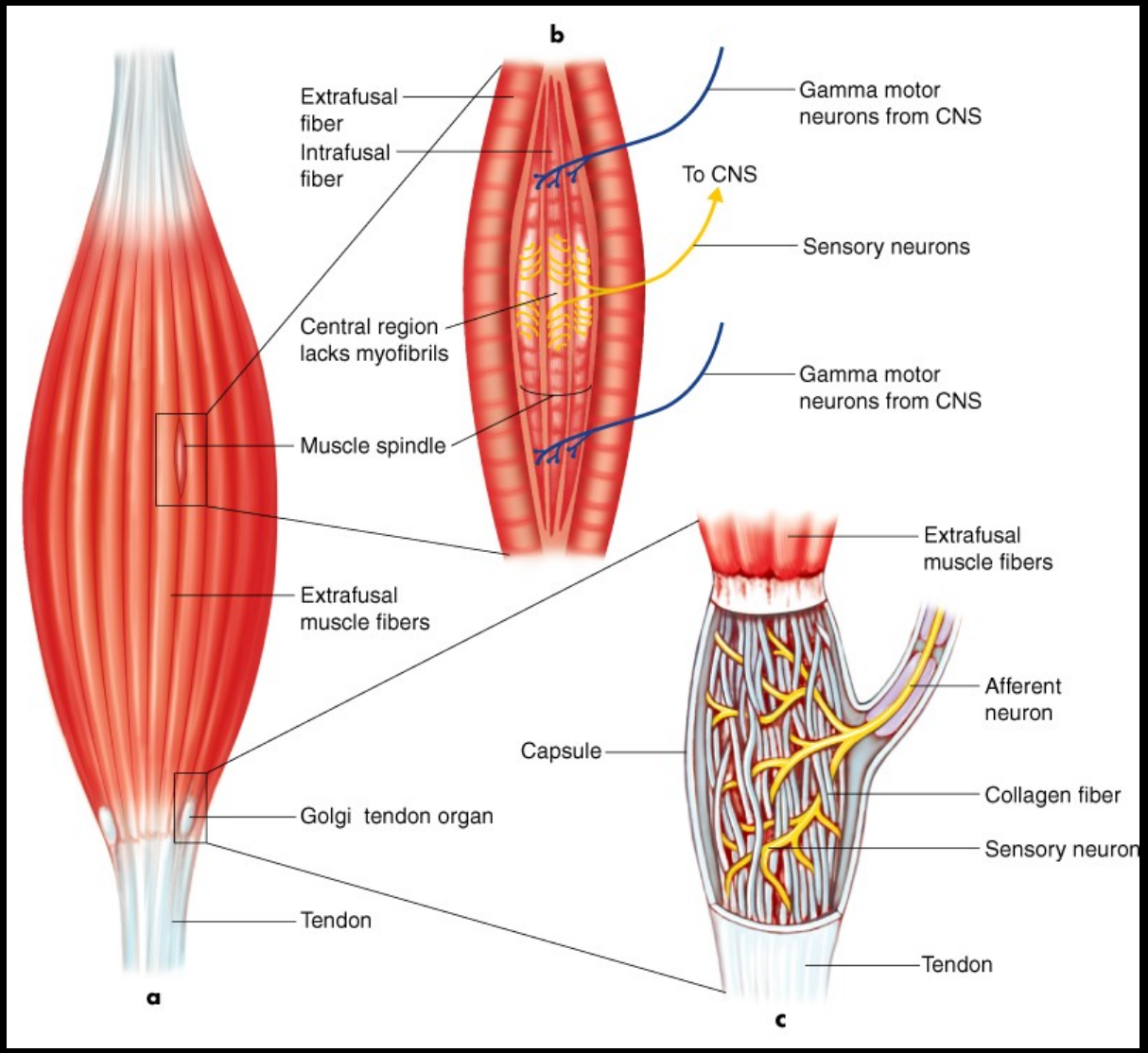
Muscle Spindles

- A group of 4 to 20 small muscle fibers (intrafusal) with sensory and motor nerve endings, covered by a connective tissue sheath, and connected to extrafusal (or regular) muscle fibers.
- The middle of the spindle can stretch but cannot contract as it contains little or no actin and myosin.
- When extrafusal fibers attached to the spindle are stretched, sensory neurons on the spindle transmit information to the CNS about the muscle's length.
- Reflexive muscle contraction is triggered through the alpha motor neuron to resist further stretching.
- Gamma motor neurons activate intrafusal fibers, causing the middle of the spindle to stretch, making the spindle sensitive to small degrees of stretch.

Golgi Tendon Organs (GTOs)

- Encapsulated sensory organs through which muscle tendon fibers pass
- Located close to the tendon's attachment to the muscle
- Sense small changes in tension
- Inhibit contracting (agonist) muscles and excite antagonist muscles to prevent injury

MUSCLE BODY, MUSCLE SPINDLE, AND GTO

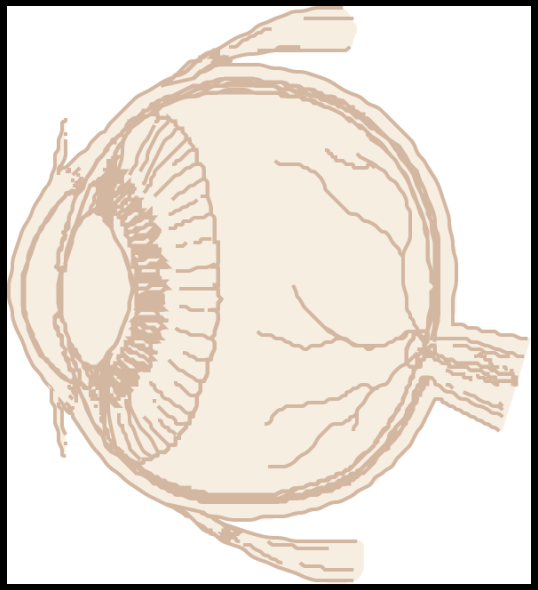


Conscious Control of Movement

- Neurons in the primary motor cortex control voluntary muscle movement.
- Clusters of nerve cells in the basal ganglia initiate sustained and repetitive movements—walking, running, maintaining posture, and muscle tone.
- The cerebellum controls fast and complex muscular activity.

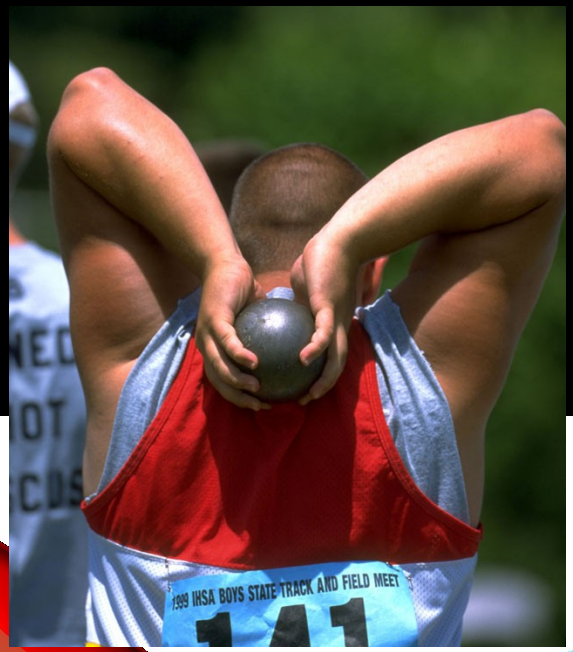
Did You Know…?

Muscles controlling fine movements, such as those controlling the eyes, have a small number of muscle fibers per motor neuron (about 1 neuron for every 15 muscle fibers). Muscles with more general function, such as those controlling the calf muscle in the leg, have many fibers per motor neuron (about 1 neuron for every 2,000 muscle fibers).



CHAPTER

NEUROMUSCULAR ADAPTATIONS TO RESISTANCE TRAINING



Defining Muscular Performance

Strength—the maximal force a muscle or muscle group can generate (dynamometer).

Power—the product of strength and the speed of movement.

Muscular endurance—the capacity to sustain repeated muscle actions.

Mechanisms of Gains in Muscle Strength

Neural Adaptations

- Synchronization and recruitment of additional motor units
- Autogenic inhibition
- Coactivation of agonist and antagonist muscles
- Rate coding—the firing frequency of motor units

Muscle Hypertrophy

- Fiber hypertrophy
- Fiber hyperplasia

Neural Activation and Fiber Hypertrophy

- Early gains in strength appear to be more influenced by neural factors.
- Long-term strength increases are largely the result of muscle fiber hypertrophy.

Muscle Size

- **Hypertrophy** refers to increases in muscle size.
- **Atrophy** refers to decreases in muscle size.
- Muscle strength involves more than just muscle size.

Muscle Hypertrophy

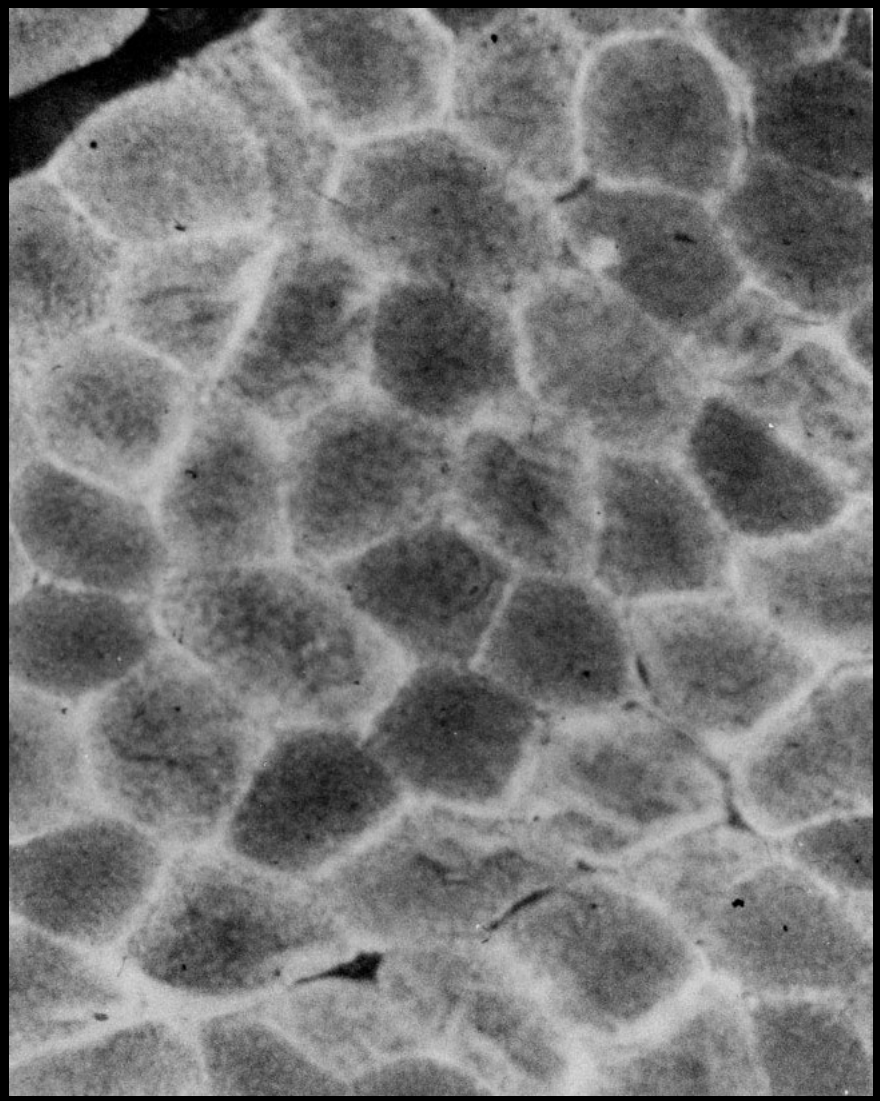
Transient—pumping up of muscle during a single exercise bout due to fluid accumulation from the blood plasma into the interstitial spaces of the muscle.

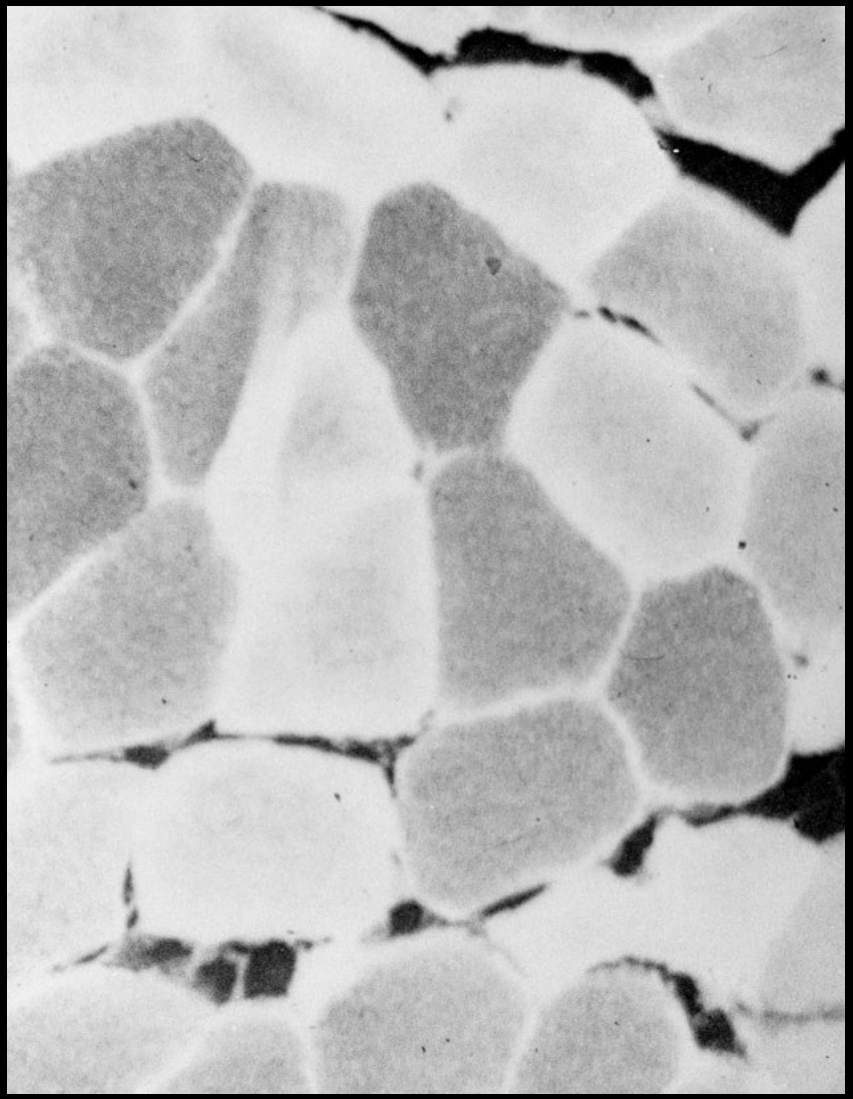
Chronic—increase of muscle size after long-term resistance training due to changes in muscle fiber number (fiber hyperplasia) or muscle fiber size (fiber hypertrophy).

Fiber Hypertrophy

- The numbers of myofibrils and actin and myosin filaments increase, resulting in more cross-bridges; sarcoplasm and connective tissue increase.
- Muscle protein synthesis increases during the postexercise period.
- Testosterone plays a role in promoting muscle growth.
- Training at higher intensities appears to cause greater fiber hypertrophy than training at lower intensities.

FIBER HYPERTROPHY AFTER TRAINING

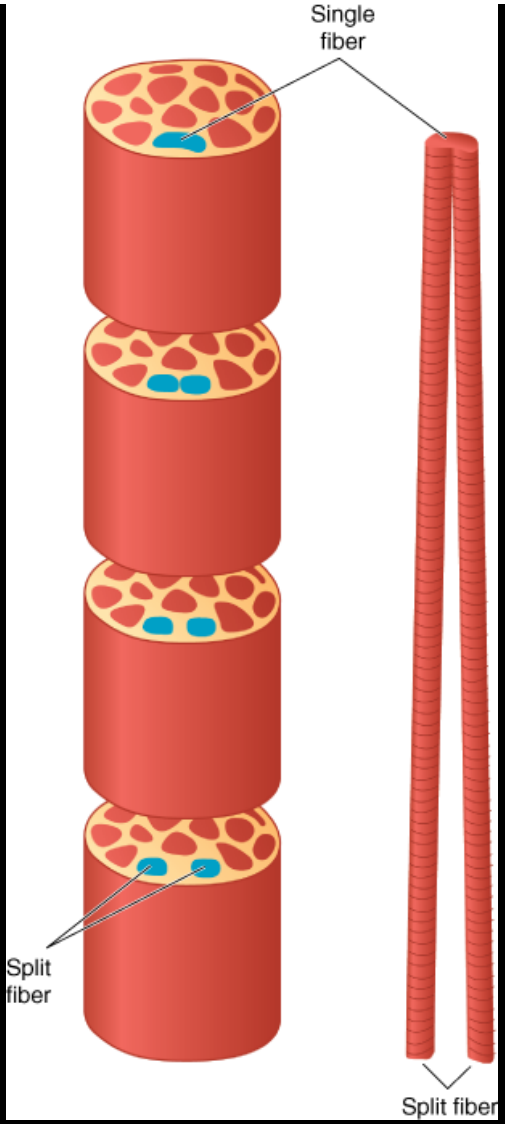




Fiber Hyperplasia

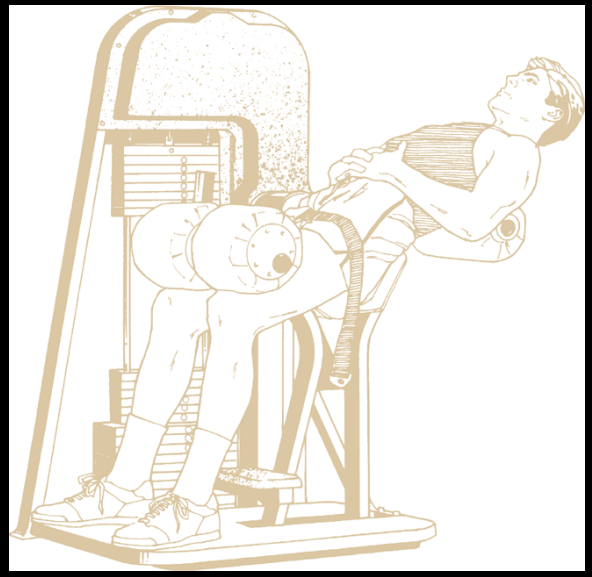
- It has been proposed that muscle fibers can split in half with intense weight training.
- Each half then increases to the size of the parent fiber.
- Satellite cells may also be involved in skeletal muscle fiber generation.
- It has been clearly shown to occur in animal models; only a few studies indirectly suggest this occurs in humans too.

SPLITTING MUSCLE FIBER



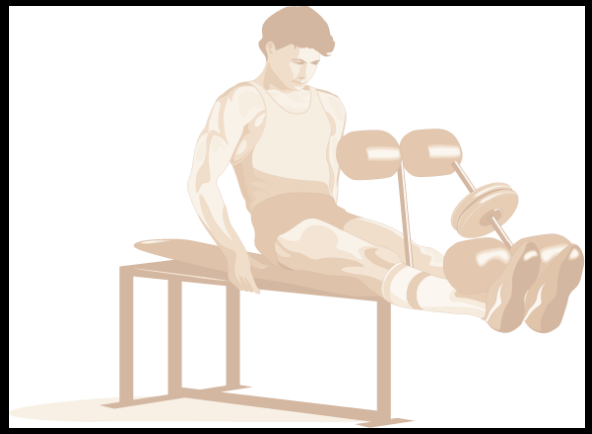
Results of Resistance Training

- Increased muscle size (hypertrophy).
- Alterations of neural control of trained muscle.
- Studies show strength gains can be achieved without changes in muscle size, but not without neural adaptations.



Acute Muscle Soreness

- Results from an accumulation of the end products of exercise in the muscles
- Usually disappears within minutes or hours after exercise



Delayed-Onset Muscle Soreness (DOMS)

- Results primarily from eccentric action
- Is associated with damage or injury within muscle
- May be caused by inflammatory reaction inside damaged muscles
- May be due to edema (accumulation of fluid) inside muscle compartment
- Is felt 12 to 48 hours after a strenuous bout of exercise

MUSCLE FIBERS AFTER A MARATHON