The Muscle physiology

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LEARNING OUTCOMES

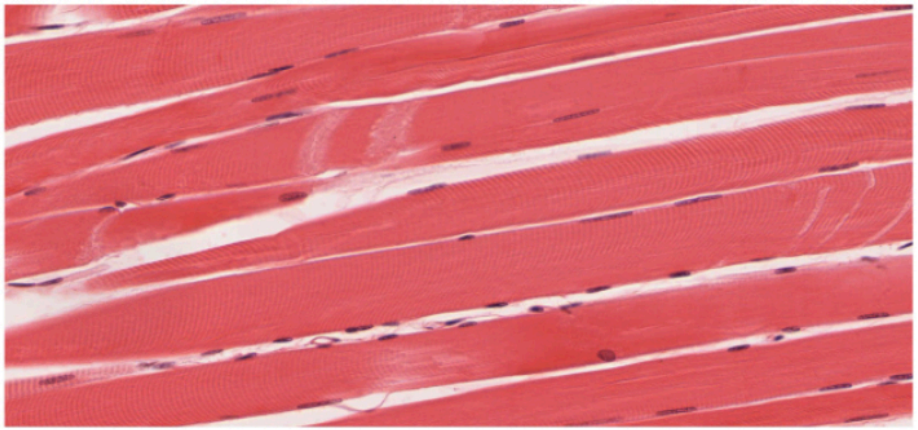
As a result of the lesson you will be able to:

- *Summarize the functions of muscular tissue;*
- *Describe the structure of a skeletal muscle fiber and relate this to its function; and describe the nerve–muscle relationship in skeletal muscle.*
- *Describe the physiological properties that all muscle types have in common;*
- *Explain the mechanisms of muscle contraction and relaxation;*
- *Describe similarities and differences in the structure and function of the three types of muscle tissue, and indicate where they are found in the body.*

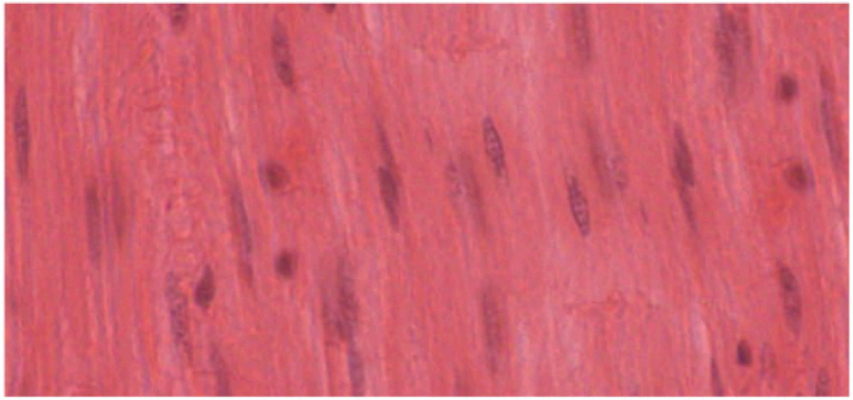
Characteristics of Muscle

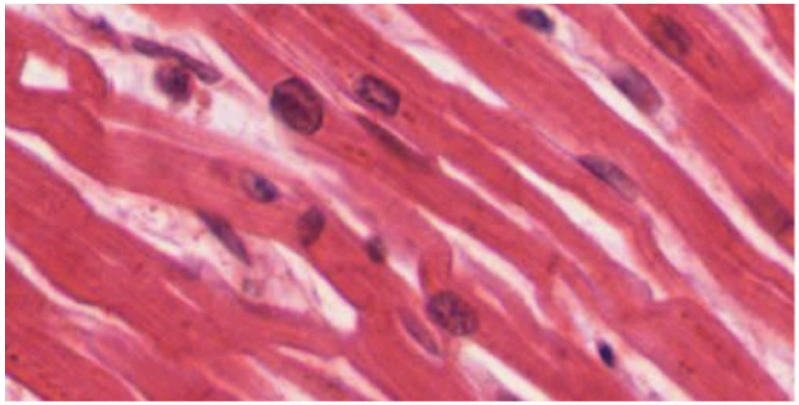
- **responsiveness (excitability)**
  - to chemical signals, stretch and electrical changes across the plasma membrane
- **conductivity**
  - local electrical change triggers a wave of excitation that travels along the muscle fiber
- **contractility**
  - shortens when stimulated
- **extensibility**
  - capable of being stretched between contractions
- **elasticity**
  - returns to its original resting length after being stretched

Characteristics of Muscle



(a)





(b)

FUNCTION OF MUSCLE

- **Skeletal muscle**
- **-** Ability to contract and cause movement. stop movement, hold a body upright or balanced in any position.
- - Prevent excess movement of the bones and joints, maintaining skeletal stability and preventing skeletal structure damage or deformation.
- - Located throughout the body at the openings of internal tracts to control the movement of various substances AND allow functions, such as swallowing, urination, and defecation, to be under voluntary control.
- - Protect internal organs (particularly abdominal and pelvic organs) by acting as an external barrier or shield to external trauma and by supporting the weight of the organs.
- - Skeletal muscles contribute to the maintenance of homeostasis in the body by generating heat. Muscle contraction requires energy, and when ATP is broken down, heat is produced. This heat is very noticeable during exercise, when sustained muscle movement causes body temperature to rise, and in cases of extreme cold, when shivering produces random skeletal muscle contractions to generate heat.

FUNCTION OF MUSCLE

- **Cardiac muscle**
- **-** to generate force and build pressure gradients to drive blood flow throughout the body.

- **Smooth muscle**
- **-**Smooth muscle in the walls of arteries is a critical component that regulates blood pressure and blood flow through the circulatory system.
- -Smooth muscle in the skin, visceral organs, and internal passageways is also essential for moving materials through the body.

Structure of a Skeletal Muscle Fiber

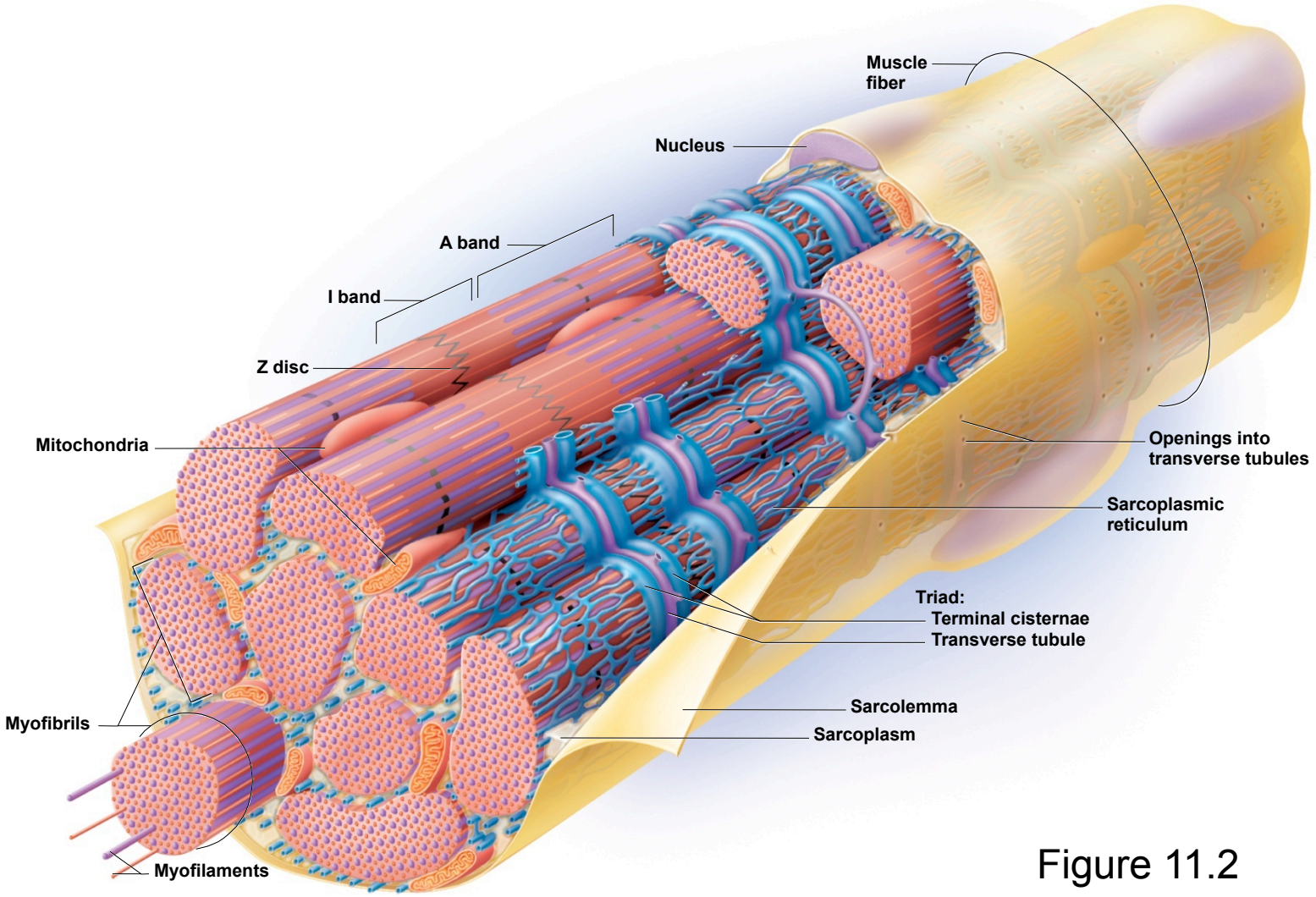
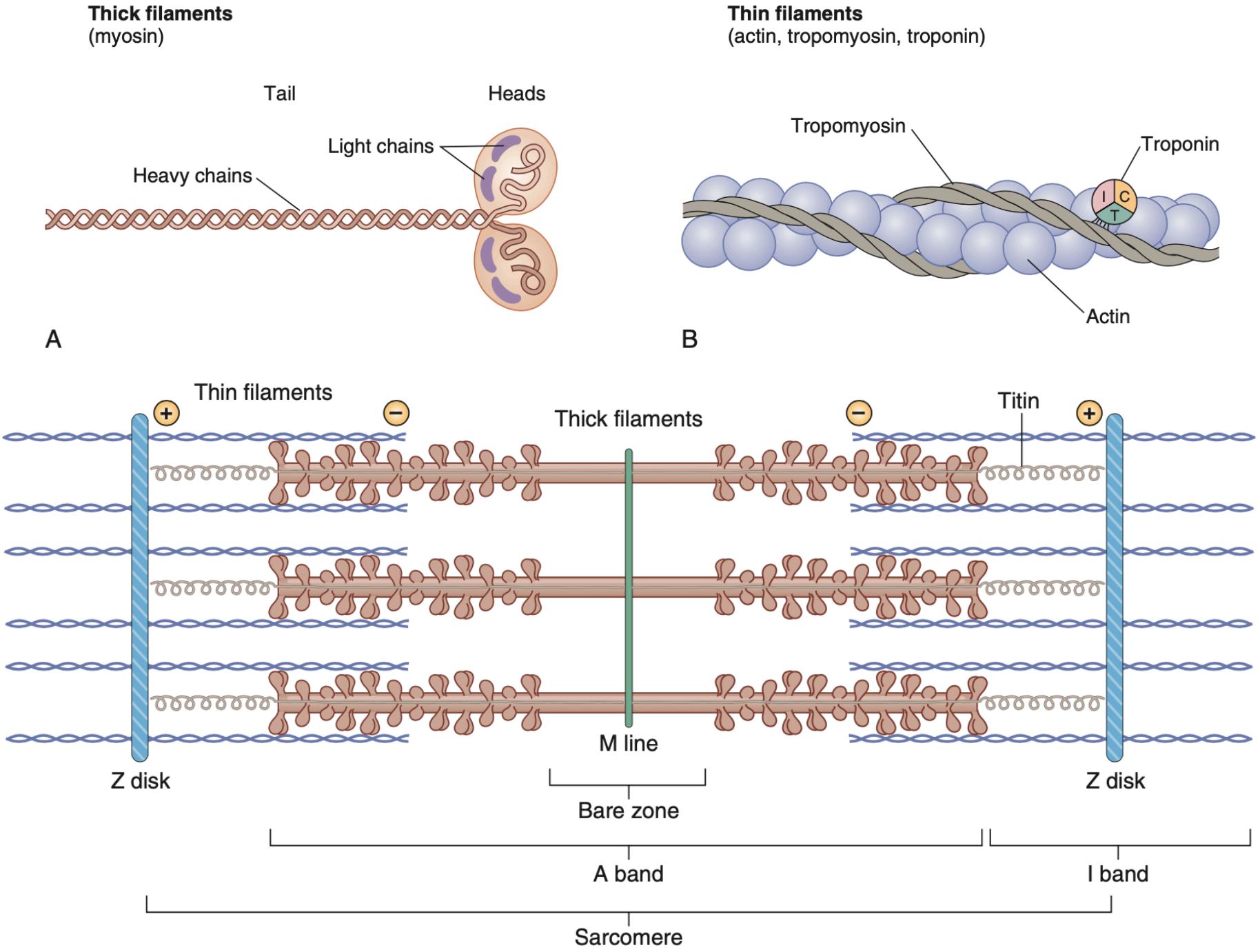


Figure 11.2


Thick filaments
(myosin)
Tail
Heads
Light chains
Heavy chains
A
Thin filaments
(actin, tropomyosin, troponin)
Tropomyosin
Troponin
I
C
T
Actin
B
Thin filaments
Thick filaments
Titin
M line
Z disk
Z disk
Bare zone
A band
I band
Sarcomere

Connective Tissue Elements

- **tendons** are attachments between muscle and bone matrix
  - **endomysium** – connective tissue around **muscle cells**
  - **perimysium** – connective tissue around **muscle fascicles**
  - **epimysium** – connective tissue surrounding **entire muscle**
  - continuous with collagen fibers of tendons
  - in turn, with connective tissue of bone matrix
- **collagen** is somewhat extensible and elastic
  - stretches slightly under tension and recoils when released
    - resists excessive stretching and protects muscle from injury
    - returns muscle to its resting length
    - contribute to power output and muscle efficiency

Structure of a Skeletal Muscle Fiber

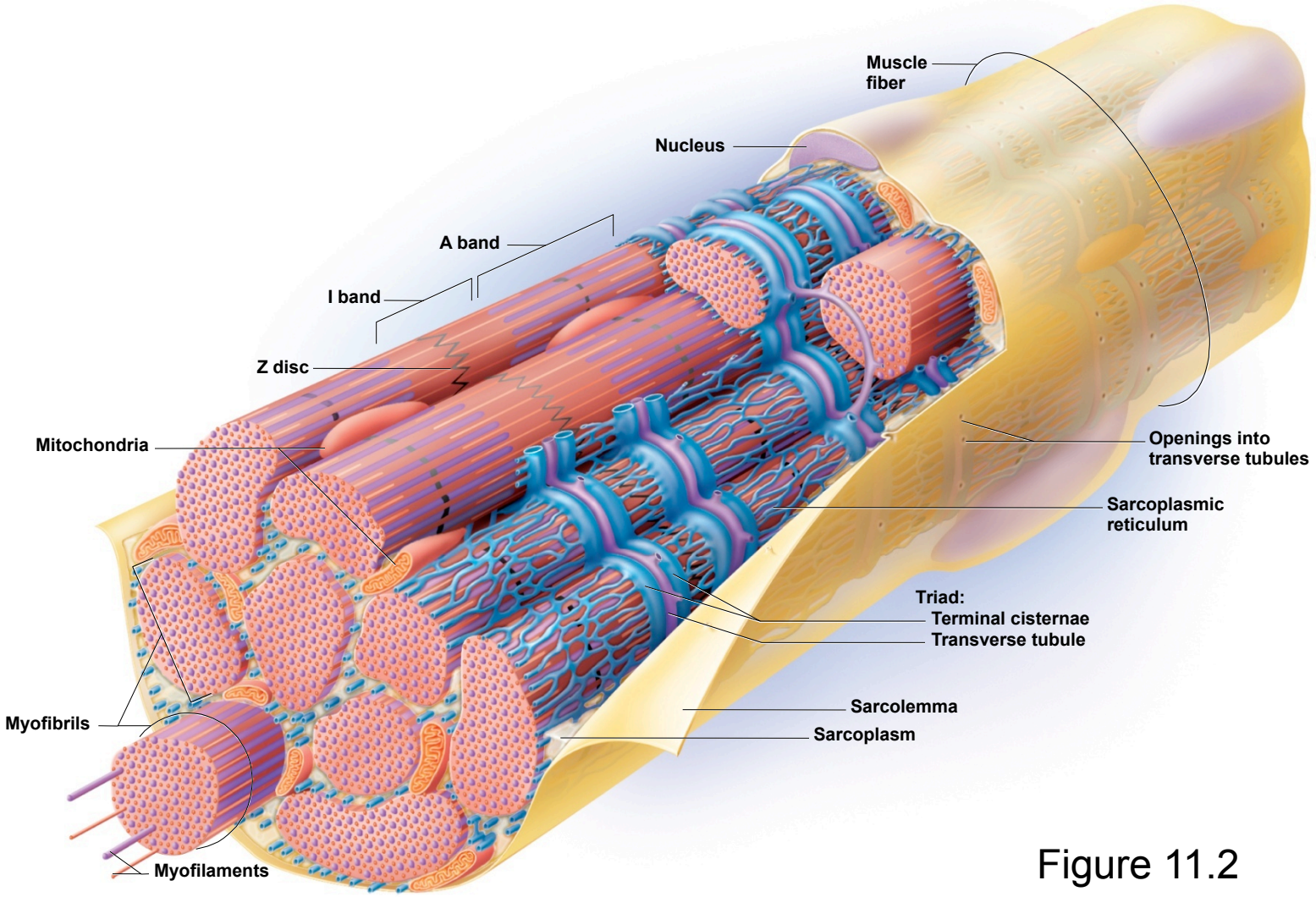


Figure 11.2

The Muscle Fiber

- **sarcolemma** – plasma membrane of a muscle fiber
- **sarcoplasm** – cytoplasm of a muscle fiber
- **myofibrils** – long protein bundles that occupies the main portion of the sarcoplasm
  - **glycogen** – stored in abundance to provide energy with heightened exercise
  - **myoglobin** – red pigment – stores oxygen needed for muscle activity
- **multiple nuclei** – flattened nuclei pressed against the inside of the sarcolemma
  - **myoblasts** – stem cells that fuse to form each muscle fiber
  - **satellite cells** – unspecialized myoblasts remaining between the muscle fiber and endomysium
    - may multiply and produce new muscle fibers to some degree
- **repair by fibrosis** rather than regeneration of functional muscle
- **mitochondria** – packed in spaces between myofibrils
- **sarcoplasmic reticulum** (SR) - smooth ER that forms a network around each myofibril – **calcium reservoir**
  - calcium activates the muscle contraction process
- **terminal cisternae** – dilated end-sacs of SR which cross muscle fiber from one side to the other
- **T tubules** – tubular infoldings of the sarcolemma which penetrate through the cell and emerge on the other side
- **triad** – a T tubule and two terminal cisterns

Thick Myofilaments

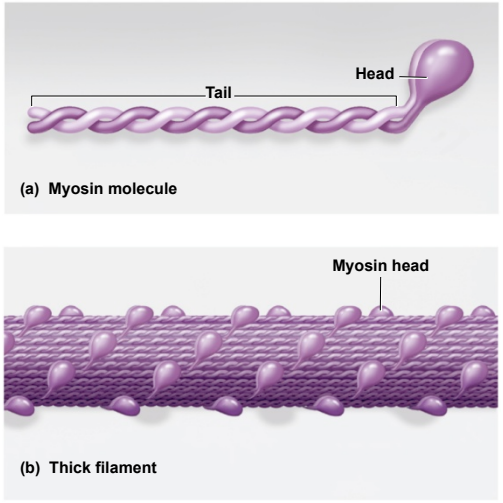


Figure 11.3 a-b

- made of several hundred **myosin** molecules
  - shaped like a golf club
    - two chains intertwined to form a shaft-like tail
    - double globular head
  - heads directed outward in a helical array around the bundle
    - heads on one half of the thick filament angle to the left
    - heads on the other half angle to the right
    - bare zone with no heads in the middle

Thin Myofilaments

- **fibrous (F) actin -** two intertwined strands
  - string of **globular (G) actin** subunits each with an **active site** that can bind to head of myosin molecule
- **tropomyosin** molecules
  - each blocking 6 or 7 active sites on G actin subunits
- **troponin** molecule - small, calcium-binding protein on each tropomyosin molecule

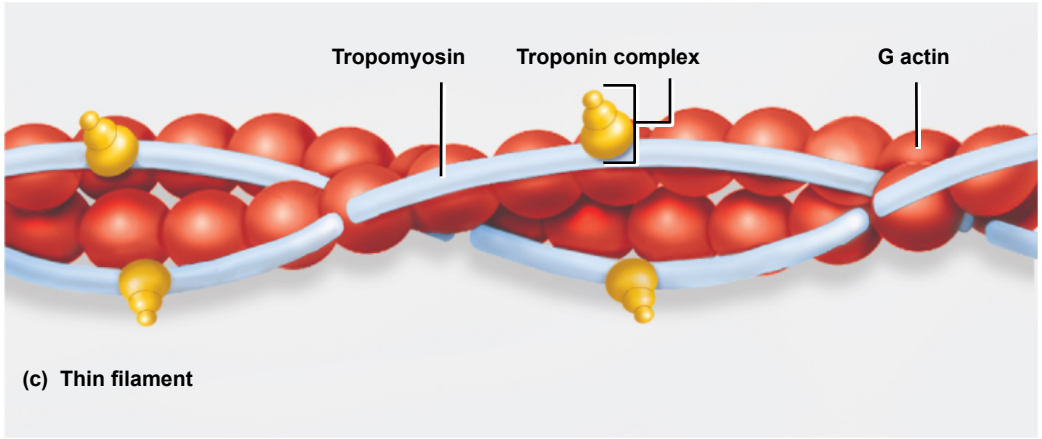


Figure 11.3c

Elastic Myofilaments

- **titin (connectin)** – huge springy protein
  - flank each thick filament and anchor it to the Z disc
  - helps stabilize the thick filament
  - center it between the thin filaments
  - prevents over stretching

Regulatory and Contractile Proteins

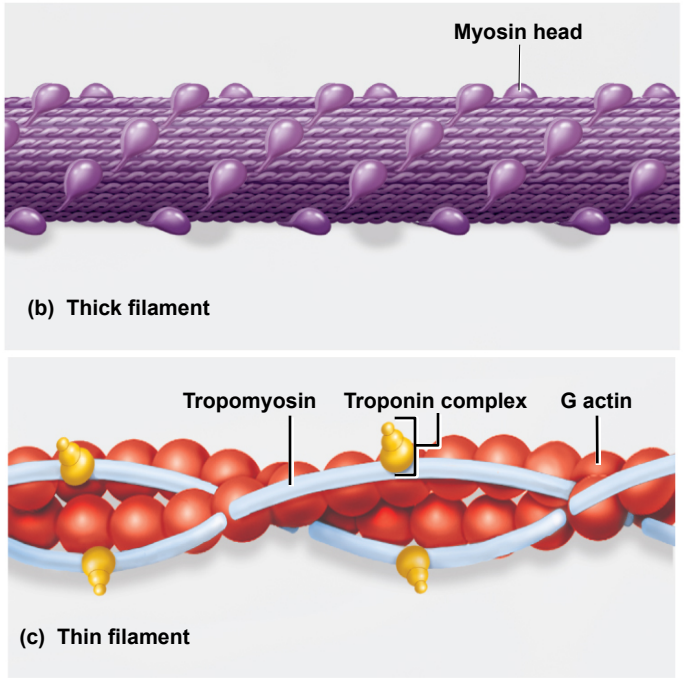


Figure 11.3 b-c

- **contractile proteins - myosin and actin**
  - do the work
- **regulatory proteins - tropomyosin and troponin**
  - like a switch that determine when the fiber can contract and when it cannot
  - contraction activated by release of calcium into sarcoplasm and its binding to troponin,
  - troponin changes shape and moves tropomyosin off the active sites on actin

Overlap of Thick and Thin Filaments

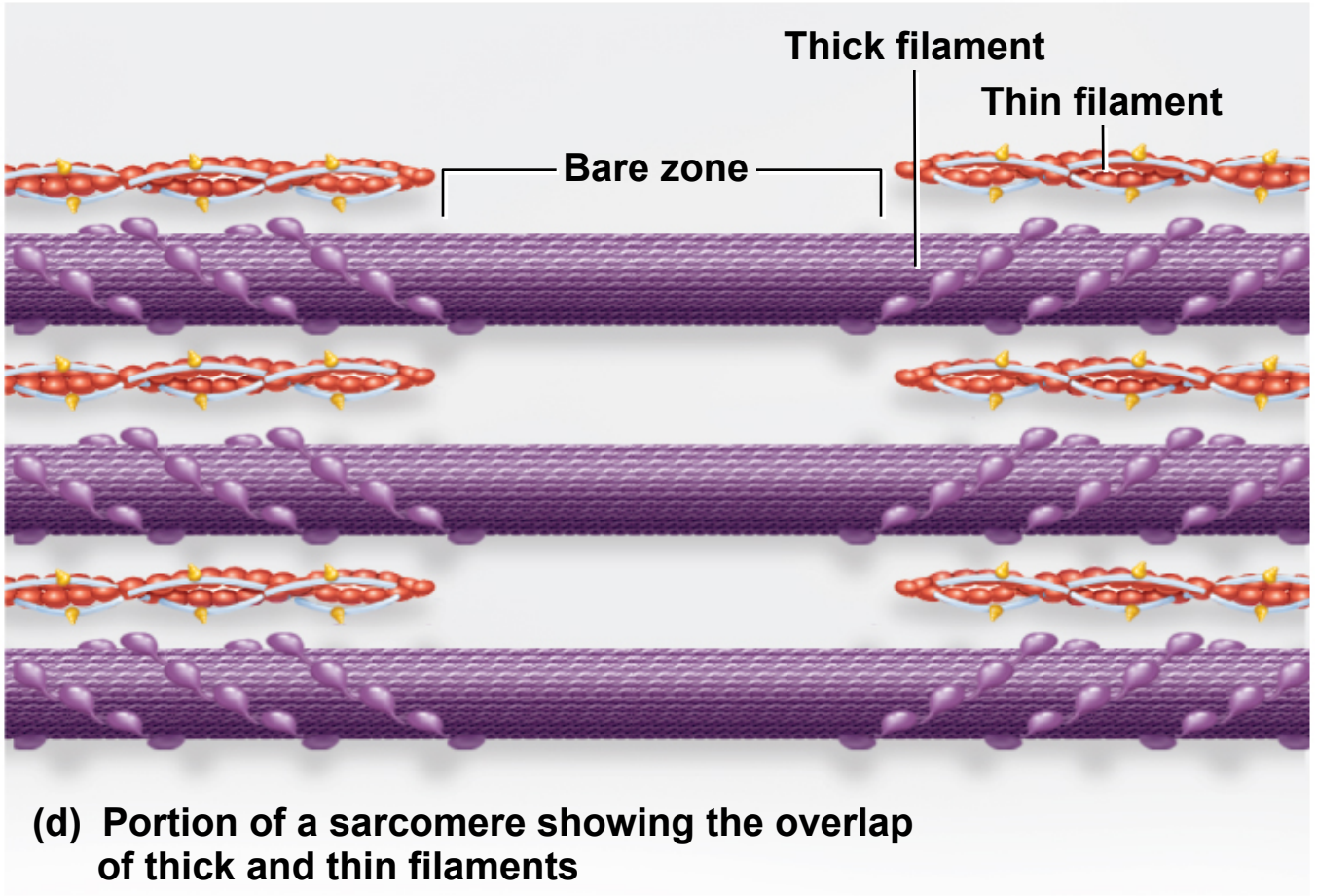


Figure 11.3d

Accessory Proteins

- at least seven other accessory proteins in or associated with thick or thin filaments
  - anchor the myofilaments, regulate length of myofilaments, alignment of myofilaments for maximum effectiveness

- **dystrophin** – most clinically important
  - links actin in outermost myofilaments to transmembrane proteins and eventually to fibrous endomysium surrounding the entire muscle cell
  - transfers forces of muscle contraction to connective tissue around muscle cell
  - genetic defects in dystrophin produce disabling disease **muscular dystrophy**

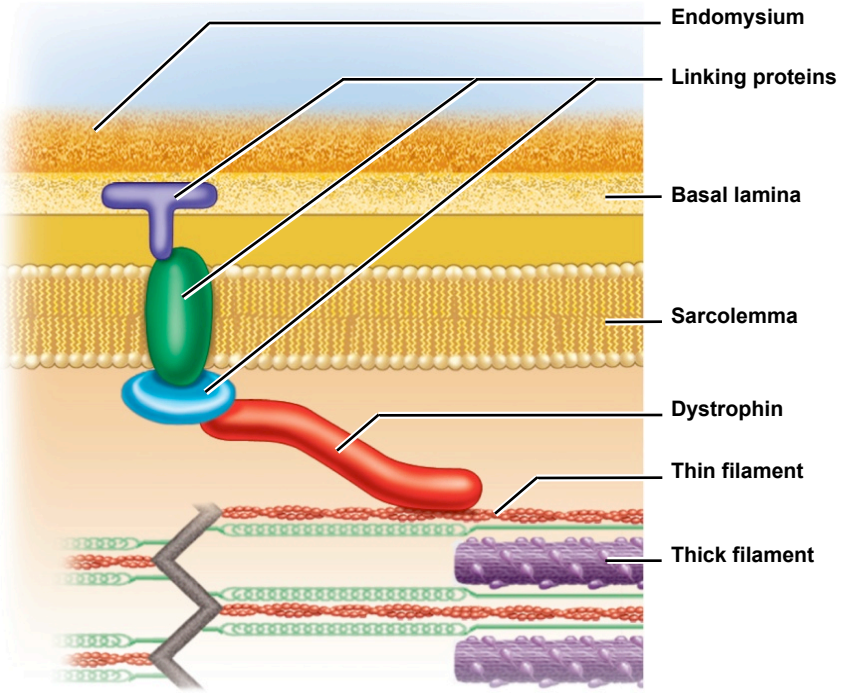


Figure 11.4

Striations

- **myosin** and **actin** are proteins that occur in all cells
  - function in cellular motility, mitosis, transport of intracellular material
- organized in a precise way in **skeletal** and **cardiac** muscle
  - **A band** – dark – A stands for anisotropic
    - part of A band where thick and thin filaments overlap is especially dark
    - **H band** in the middle of A band – just thick filaments
    - **M line** is in the middle of the H band
  - **I band** – alternating lighter band – I stands for isotropic
    - the way the bands reflect polarized light
  - **z disc** – provides anchorage for thin filaments and elastic filaments
    - bisects I band
  - **sarcomere –** the segment of the myofibril from one z disc to the nex**t**

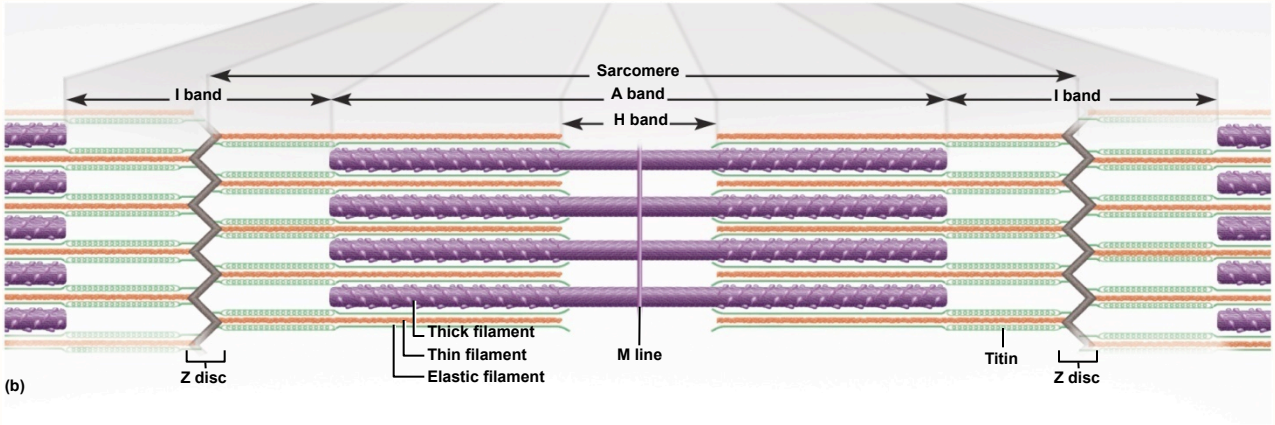
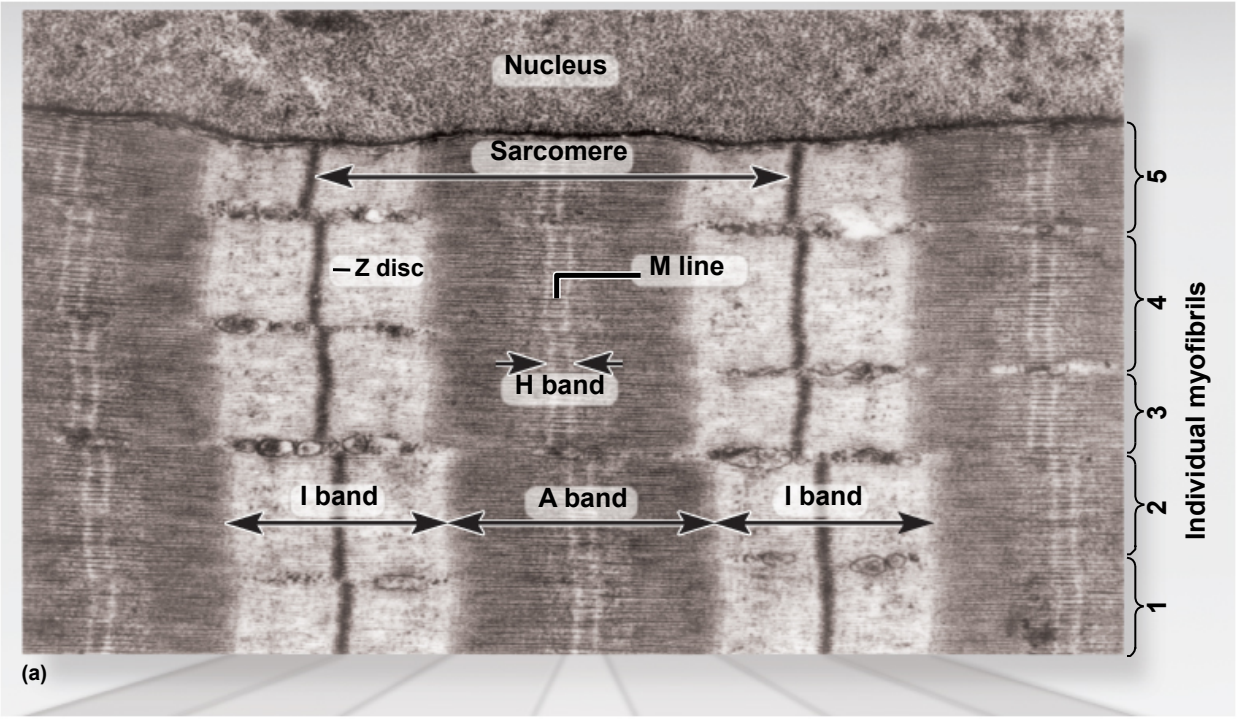


Figure 11.5b

Striations and Sarcomeres

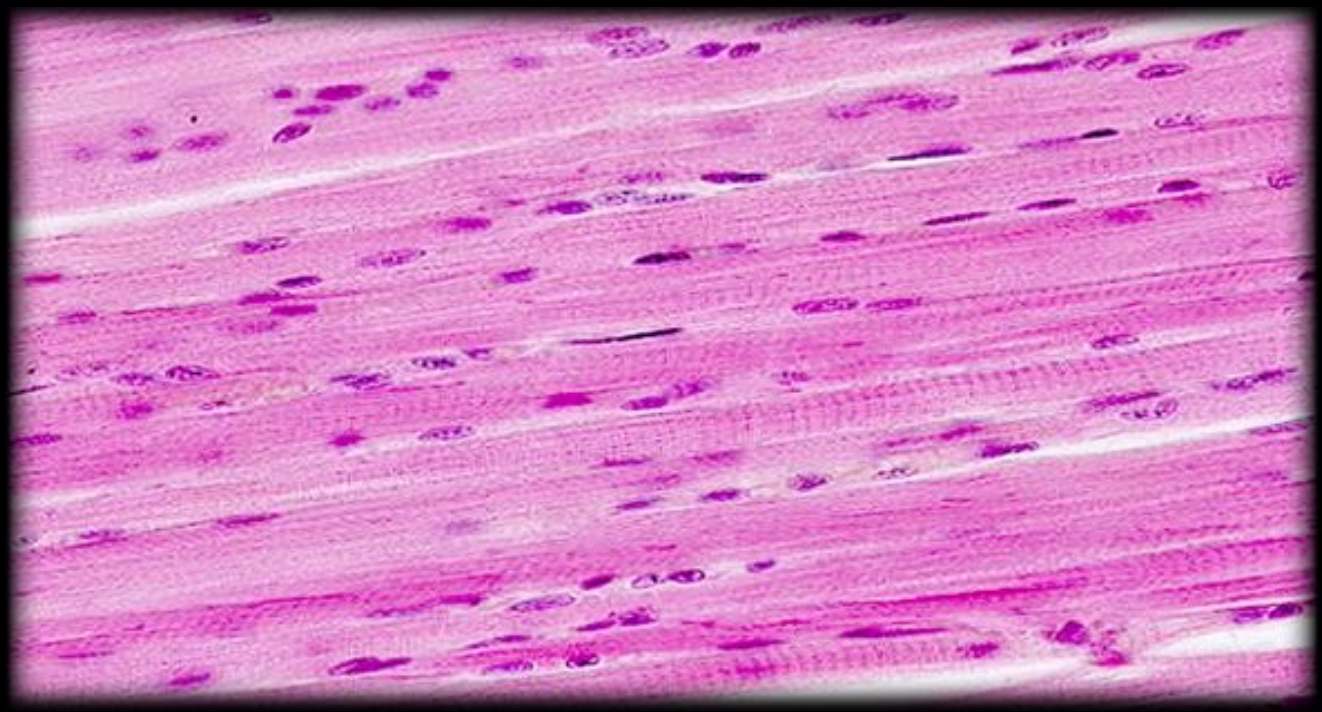


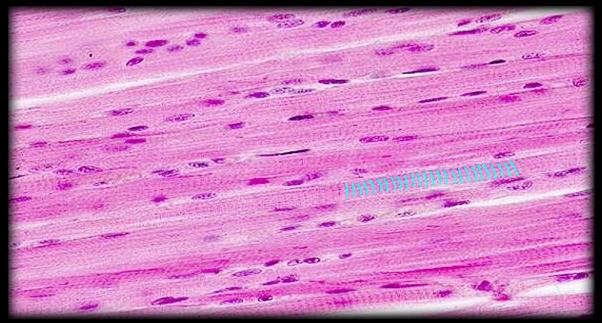
Visuals Unlimited

Figure 11.5a

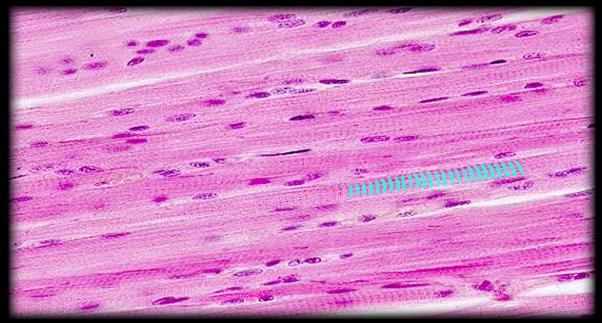
- **sarcomere** – ***functional contractile unit of the muscle fiber***
  - muscle shortens because individual sarcomeres shorten
  - pulls z discs closer to each other

Skeletal Muscle

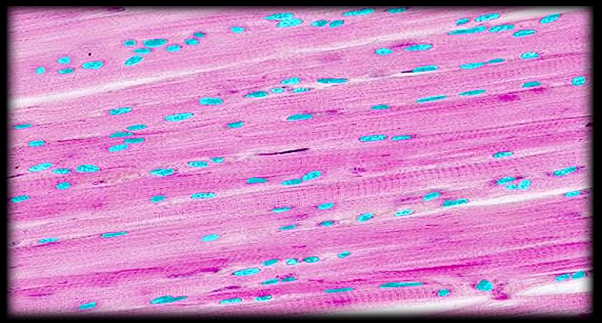




A band



I band



nuclei

Sarcomeres

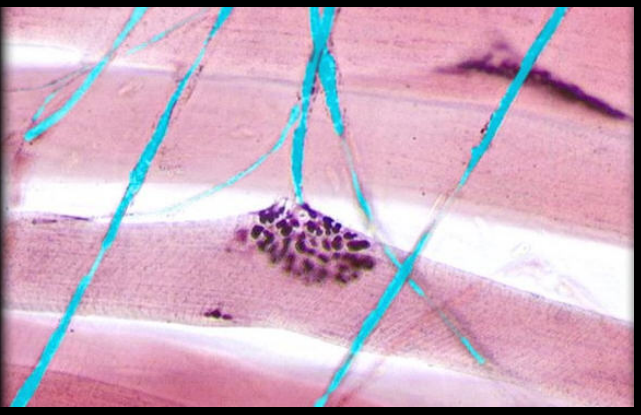
- **sarcomere** - segment from Z disc to Z disc
  - functional contractile unit of muscle fiber
- muscle cells shorten because their individual sarcomeres shorten
  - Z disc (Z lines) are pulled closer together as thick and thin filaments slide past each other
- neither thick nor thin filaments change length during shortening
  - only the amount of overlap changes
- during shortening dystrophin & linking proteins also pull on extracellular proteins
  - transfers pull to extracellular tissue

The Nerve-Muscle Relationship

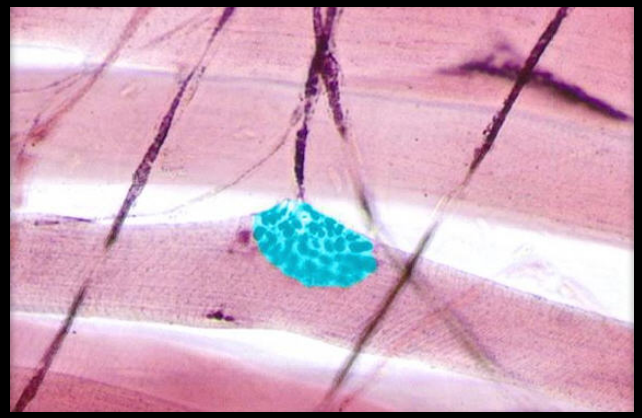
- skeletal muscle never contracts unless stimulated by a nerve
- if nerve connections are severed or poisoned, a muscle is paralyzed
- **denervation atrophy** – shrinkage of paralyzed muscle when connection not restored
- **somatic motor neurons** – nerve cells whose cell bodies are in the brainstem and spinal cord that serve skeletal muscles
- **somatic motor fibers** –their **axons** that lead to the skeletal muscle
  - each nerve fiber branches out to a number of muscle fibers
  - each muscle fiber is supplied by only one motor neuron

Motor Neurons

axon



axon terminal



muscle fiber

Motor Units

- **motor unit** – one nerve fiber and all the muscle fibers innervated by it

- **muscle fibers of one motor unit**
  - dispersed throughout the muscle
  - contract in unison
  - produce weak contraction over wide area
  - provides ability to sustain long-term contraction as motor units take turns contracting (postural control)
  - effective contraction usually requires the contraction of several motor units at once

- **average motor unit** – 200 muscle fibers for each motor unit

- **small motor units** - fine degree of control
  - 3-6 muscle fibers per neuron
  - eye and hand muscles

- **large motor units** – more strength than control
  - powerful contractions supplied by large motor units – gastrocnemius – 1000 muscle fibers per neuron
  - many muscle fibers per motor unit

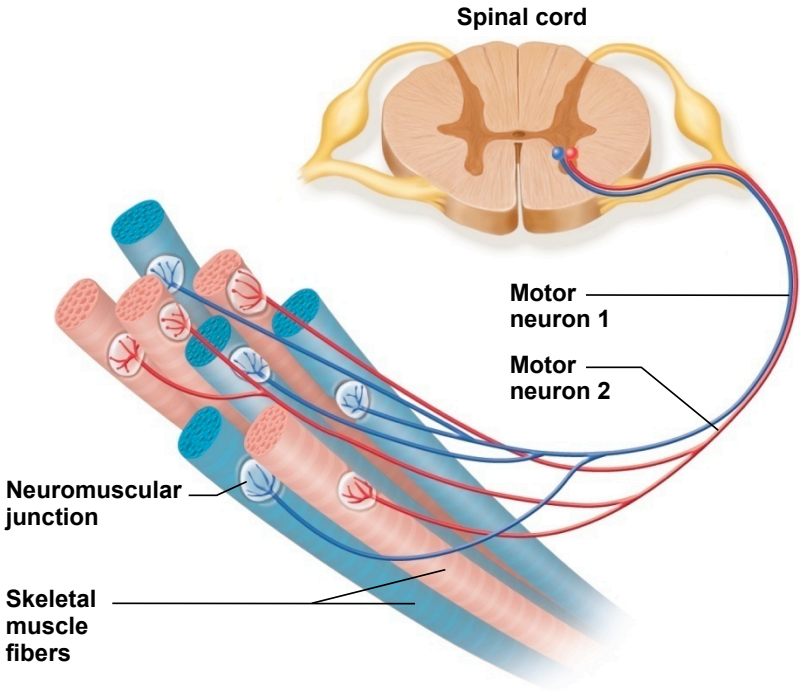


Figure 11.6

The Neuromuscular Junction

- **synapse** – point where a nerve fiber meets its target cell
- **neuromuscular junction (NMJ) -** when target cell is a muscle fiber
- each terminal branch of the nerve fiber within the NMJ forms separate synapse with the muscle fiber
- one nerve fiber stimulates the muscle fiber at several points within the NMJ

Components of Neuromuscular Junction

- **synaptic knob** - swollen end of nerve fiber
  - contains **synaptic vesicles** filled with **acetylcholine** (ACh)
- **synaptic cleft** - tiny gap between synaptic knob and muscle sarcolemma
- **Schwann cell** envelops & isolates all of the NMJ from surrounding tissue fluid
- synaptic vesicles undergo **exocytosis** releasing ACh into synaptic cleft
- 50 million **ACh receptors** – proteins incorporated into muscle cell plasma membrane
  - **junctional folds** of sarcolemma beneath synaptic knob
    - increases surface area holding ACh receptors
      - lack of receptors leads to paralysis in disease myasthenia gravis
- **basal lamina** - thin layer of collagen and glycoprotein separates Schwann cell and entire muscle cell from surrounding tissues
  - contains **acetylcholinesterase** (AChE) that breaks down ACh after contraction causing relaxation

Neuromuscular Junction

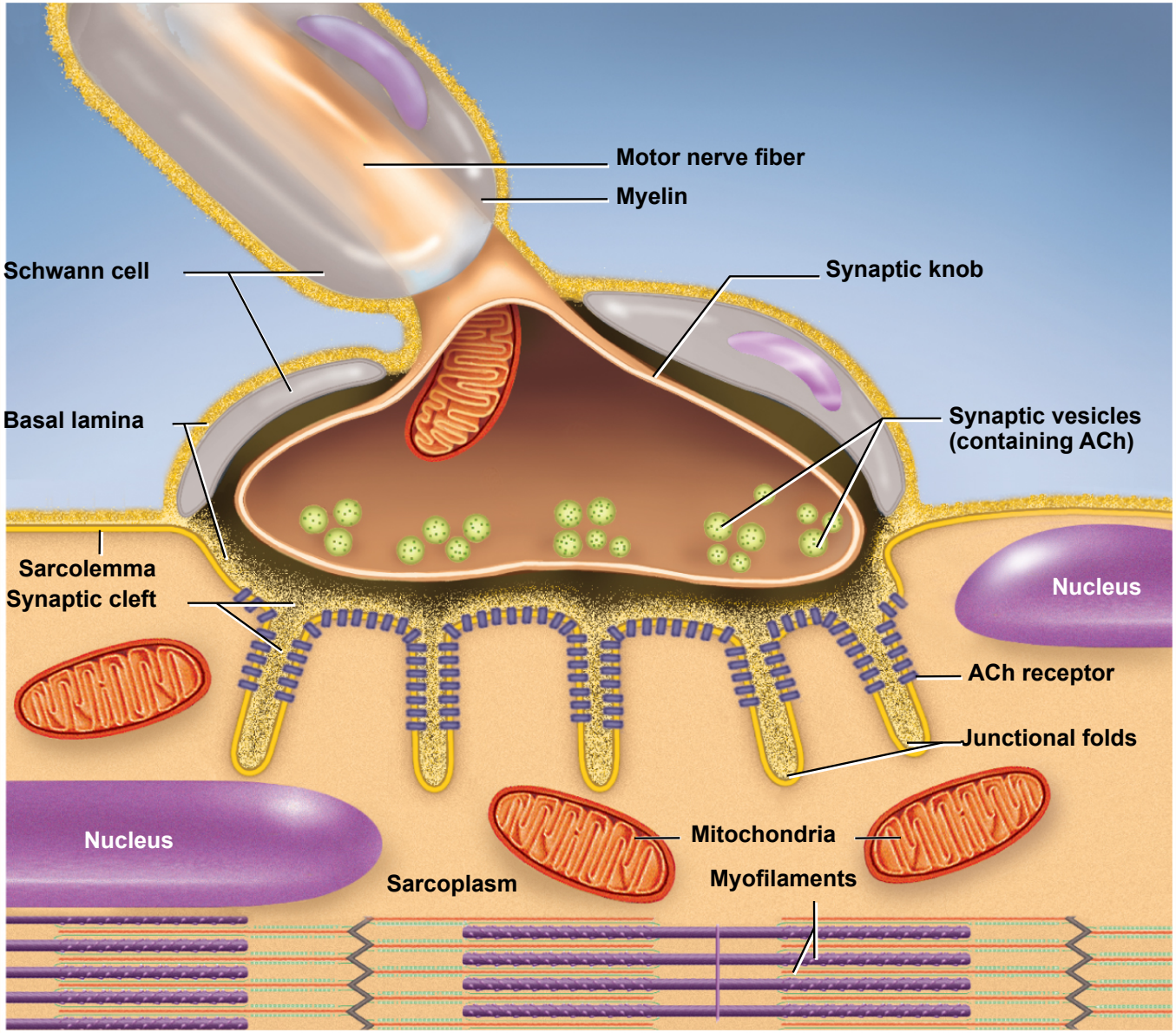


Figure 11.7b

Neuromuscular Junction - LM

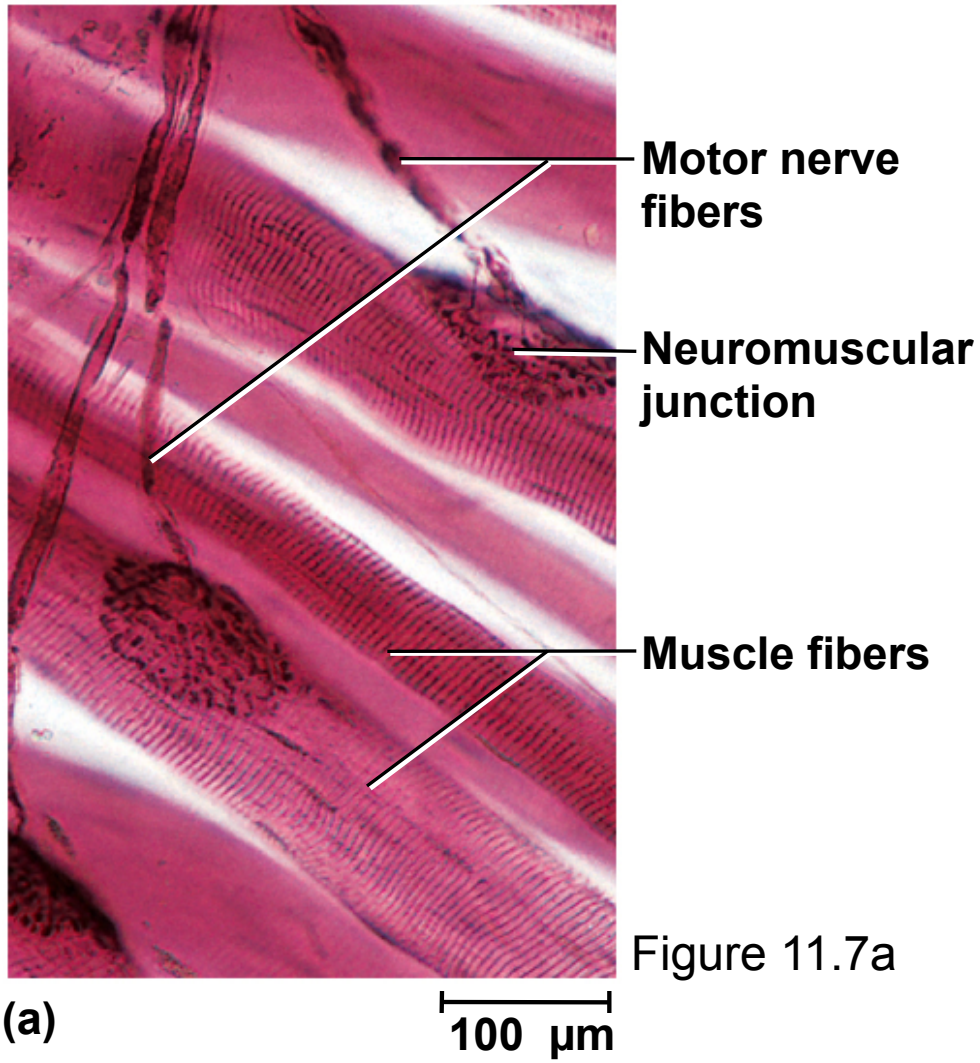


Figure 11.7a

Neuromuscular Toxins

- toxins that interfere with synaptic function can paralyze the muscles
- some **pesticides** contain **cholinesterase inhibitors**
  - bind to acetylcholinesterase and prevent it from degrading ACh
  - **spastic paralysis** - a state of continual contraction of the muscles
  - possible suffocation
- **tetanus** (lockjaw) is a form of spastic paralysis caused by toxin of *Clostridium tetani*
  - **glycine** in the spinal cord normally stops motor neurons from producing unwanted muscle contractions
  - tetanus toxin blocks glycine release in the spinal cord and causes overstimulation and spastic paralysis of the muscles
- **flaccid paralysis** – a state in which the muscles are limp and cannot contract
  - **curare** – compete with ACh for receptor sites, but do not stimulate the muscles
  - plant poison used by South American natives to poison blowgun darts
- **botulism** – type of food poisoning caused by a neuromuscular toxin secreted by the bacterium *Clostridium botulinum*
  - blocks release of ACh causing flaccid paralysis
  - **Botox Cosmetic** injections for wrinkle removal

Electrically Excitable Cells

- **muscle fibers** and **neurons** are electrically excitable cells
  - their plasma membrane exhibits voltage changes in response to stimulation
- **electrophysiology -** the study of the electrical activity of cells
- in an **unstimulated (resting) cell**
  - there are more anions (negative ions) on the inside of the plasma membrane than on the outside
  - the plasma membrane is electrically **polarized** (charged)
  - there are **excess sodium** ions (Na^+) in the **extracellular fluid** (ECF)
  - there are **excess potassium** ions (K^+) in the **intracellular fluid** (ICF)
  - also in the ICF, there are anions such as proteins, nucleic acids, and phosphates that cannot penetrate the plasma membrane
  - these anions make the inside of the plasma membrane negatively charged by comparison to its outer surface
- **voltage (electrical potential)** – a difference in electrical charge from one point to another
- **resting membrane potential** – about -90mV
  - maintained by sodium-potassium pump

Electrically Excitable Cells

- **stimulated (active) muscle fiber or nerve cell**
  - **ion gates open** in the plasma membrane
  - **Na^+** instantly diffuses down its concentration gradient into the cell
  - these cations override the negative charges in the ICF
  - **depolarization** - inside of the plasma membrane becomes briefly positive
  - immediately, **Na^+ gates close** and **K^+ gates open**
  - **K^+ rushes out** of cell
  - repelled by the positive sodium charge and partly because of its concentration gradient
  - loss of positive potassium ions turns the membrane negative again **(repolarization)**
  - **action potential –** quick up-and-down voltage shift from the negative RMP to a positive value, and back to the negative value again.
  - **RMP** is a stable voltage seen in a **waiting** muscle or nerve cell
  - **action potential** is a quickly fluctuating voltage seen in an **active stimulated cell**
  - an action potential at one point on a plasma membrane causes another one to happen immediately in front of it, which triggers another one a little farther along and so forth

Muscle Contraction & Relaxation

- **four major phases of contraction and relaxation**
  - **excitation**
    - the process in which nerve action potentials lead to muscle action potentials
  - **excitation-contraction coupling**
    - events that link the action potentials on the sarcolemma to activation of the myofilaments, thereby preparing them to contract
  - **contraction**
    - step in which the muscle fiber develops tension and may shorten
  - **relaxation**
    - when its work is done, a muscle fiber relaxes and returns to its resting length

Excitation of a Muscle Fiber

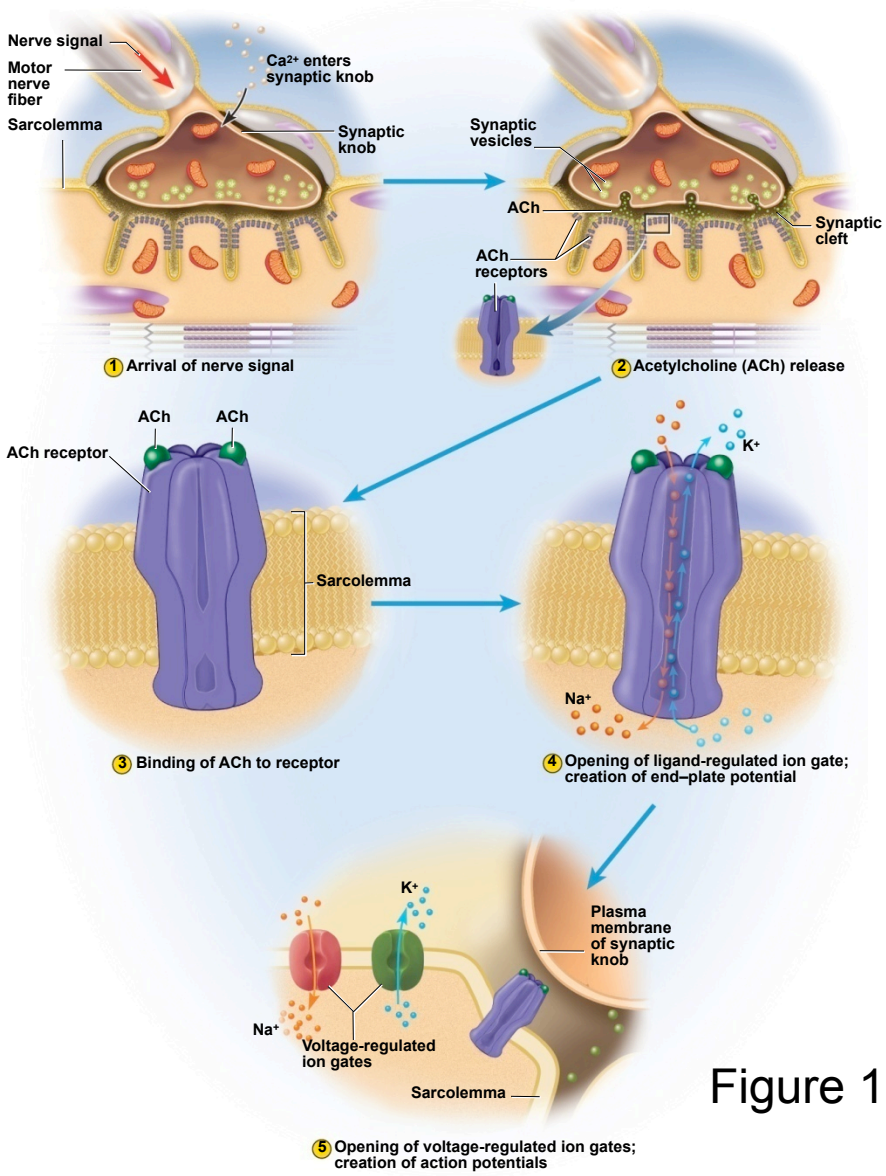


Figure 11.8

Excitation (steps 1 and 2)

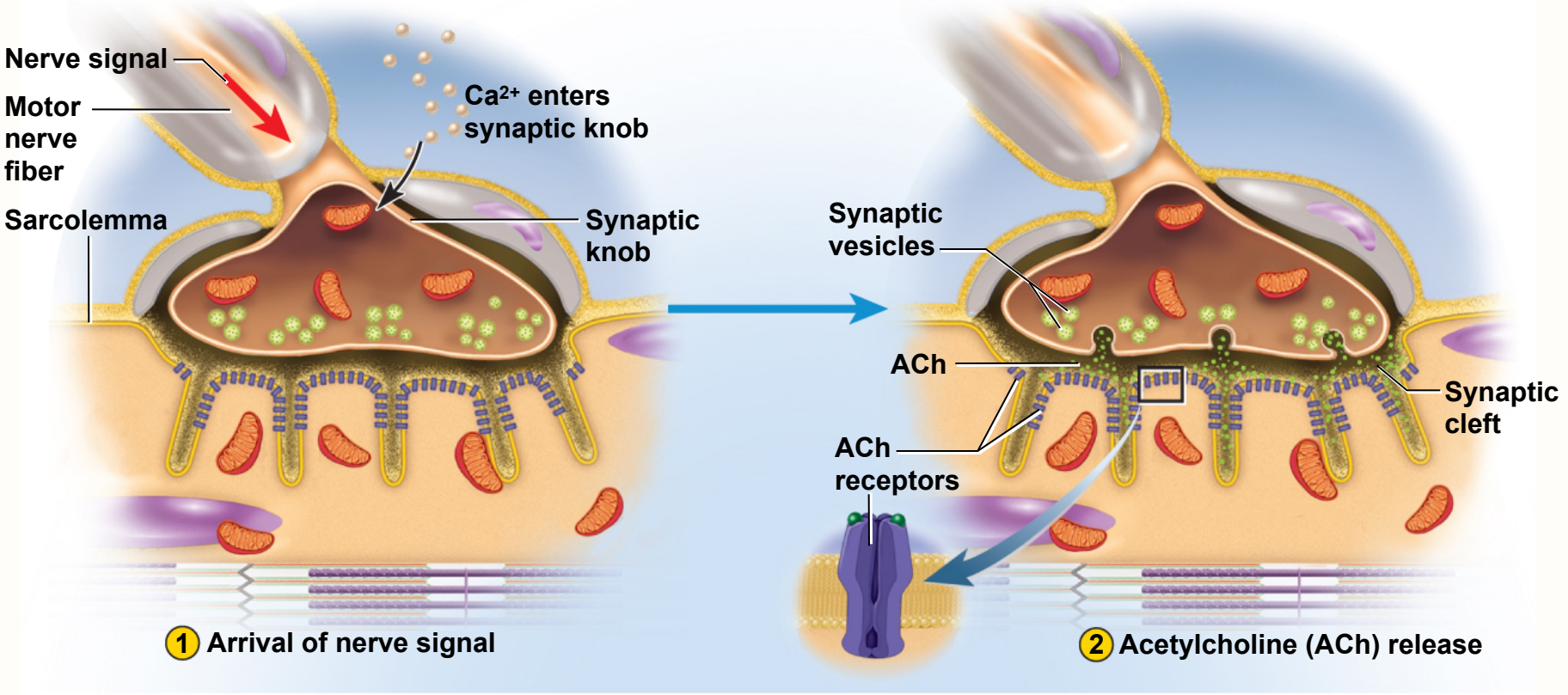
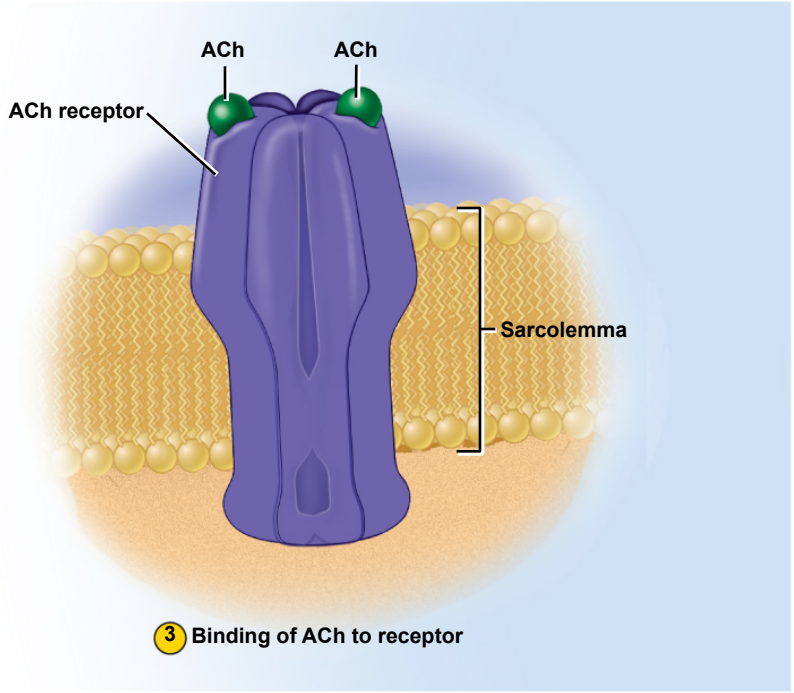


Figure 11.8 (1-2)

- nerve signal opens voltage-gated calcium channels in synaptic knob
- calcium stimulates exocytosis of ACh from synaptic vesicles
- ACh released into synaptic cleft

Excitation (steps 3 and 4)



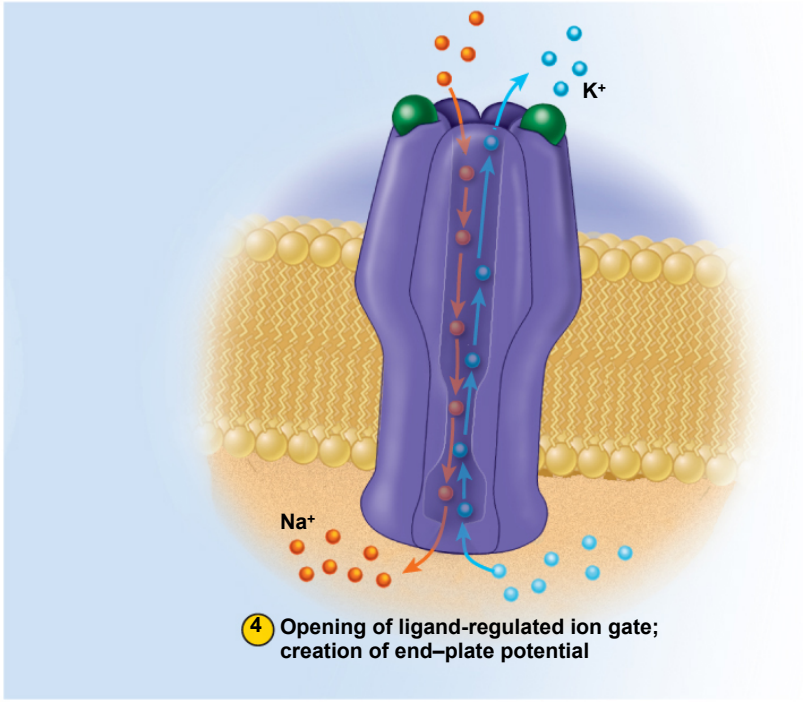


Figure 11.8 (3-4)

- two ACh molecules bind to each receptor protein, opening Na^+ and K^+ channels.
- Na^+ enters shifting RMP goes from -90mV to +75mV, then K^+ exits and RMP returns to -90mV - quick voltage shift is called an **end-plate potential (EPP).**

Excitation (step 5)

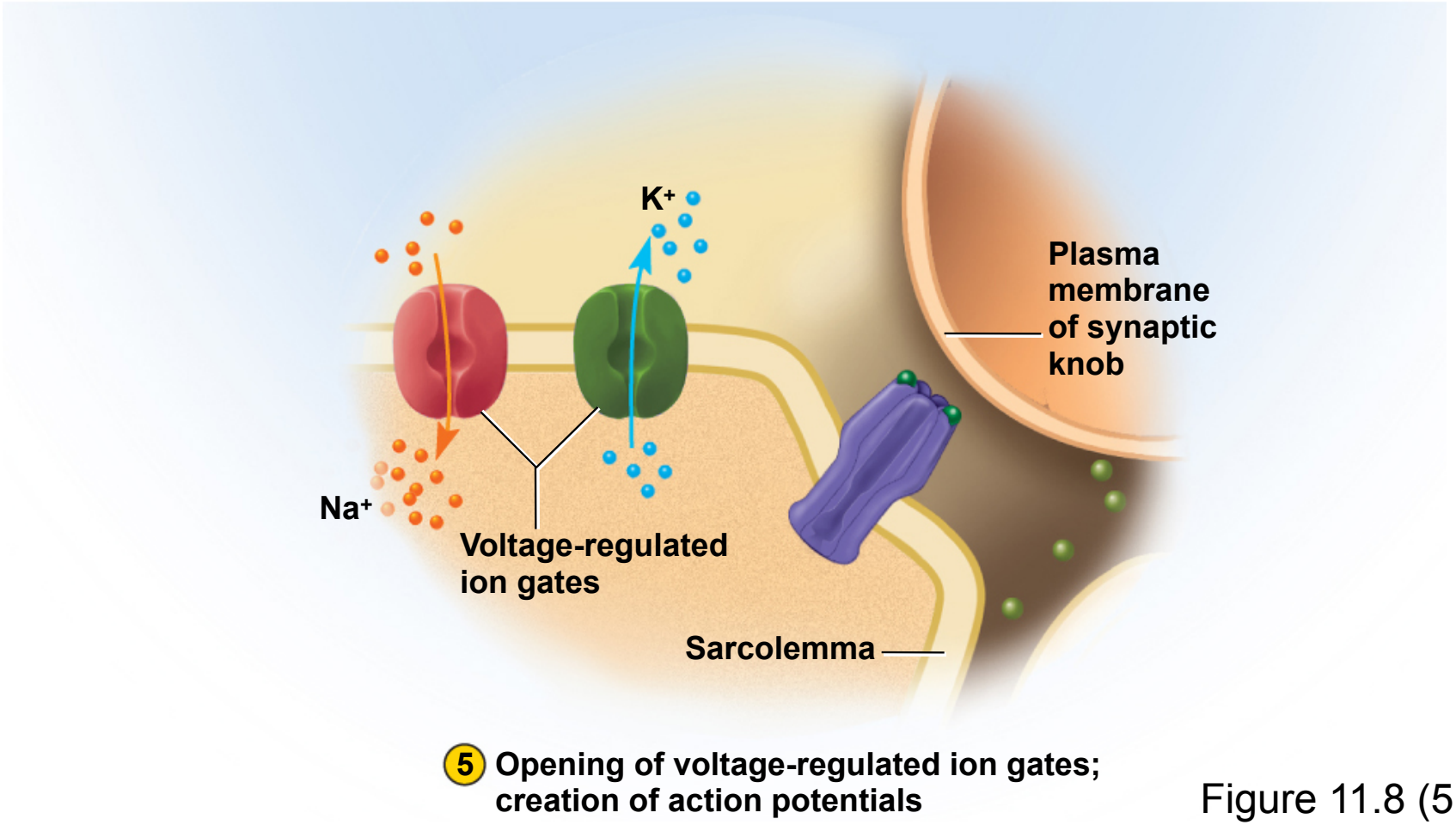


Figure 11.8 (5)

- voltage change (EPP) in end-plate region opens nearby voltage-gated channels producing an action potential that spreads over muscle surface,

Excitation-Contraction Coupling in Skeletal Muscle

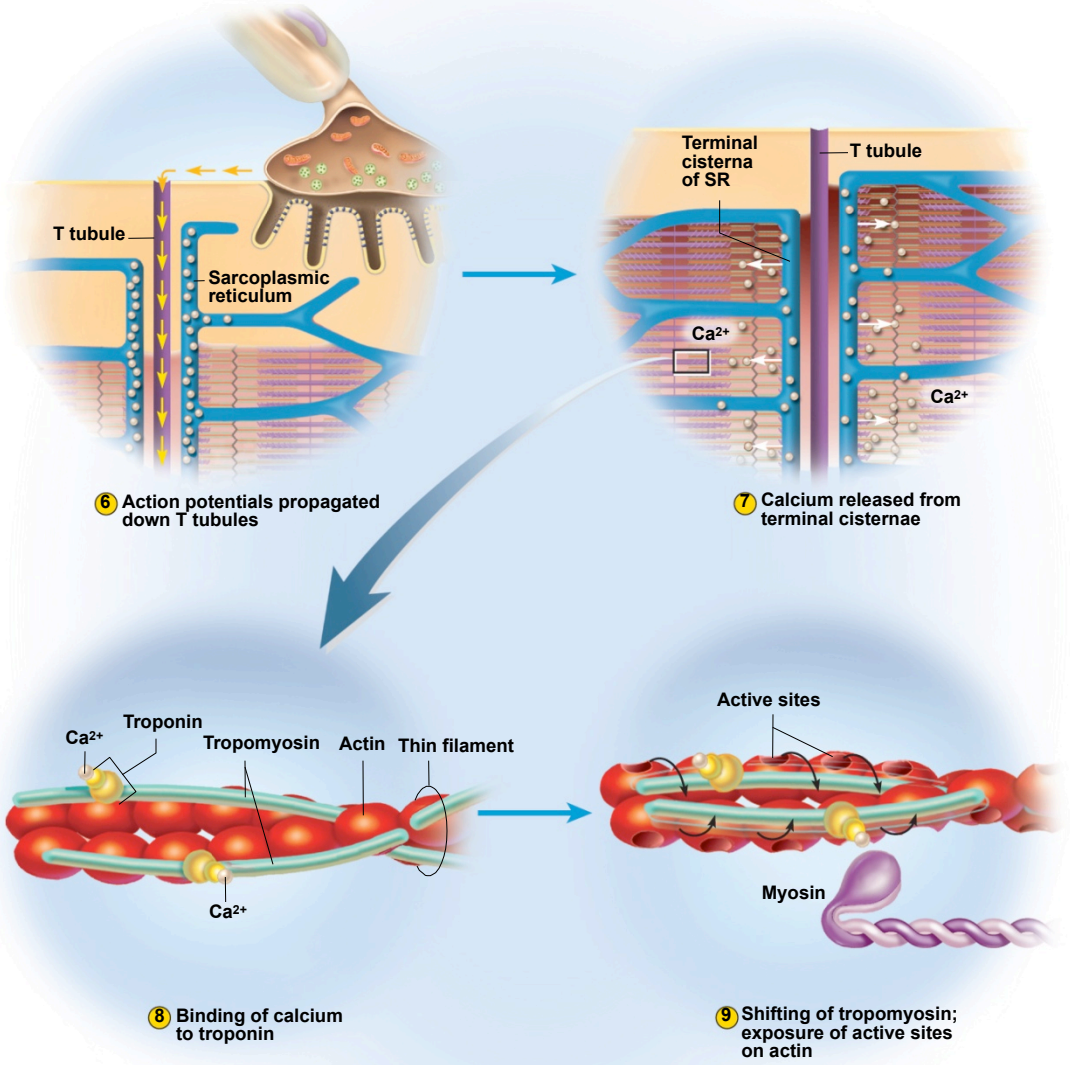


Figure 11.9 (6-9)

Excitation-Contraction Coupling (steps 6 and 7)

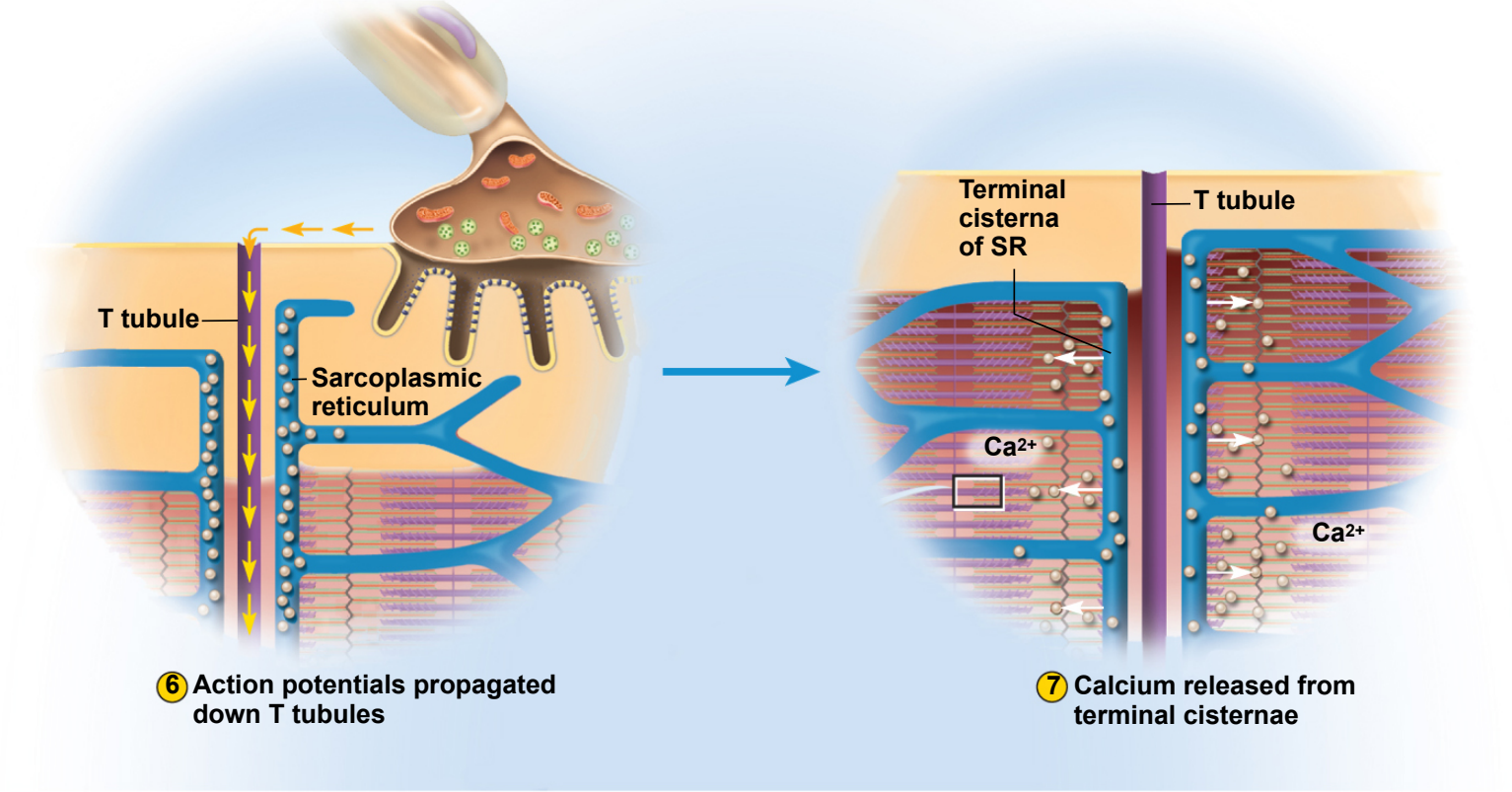


Figure 11.9 (6-7)

- action potential spreads down into T tubules
- opens voltage-gated ion channels in T tubules and Ca^{+2} channels in SR
- Ca^{+2} enters the cytosol

Excitation-Contraction Coupling (steps 8 and 9)

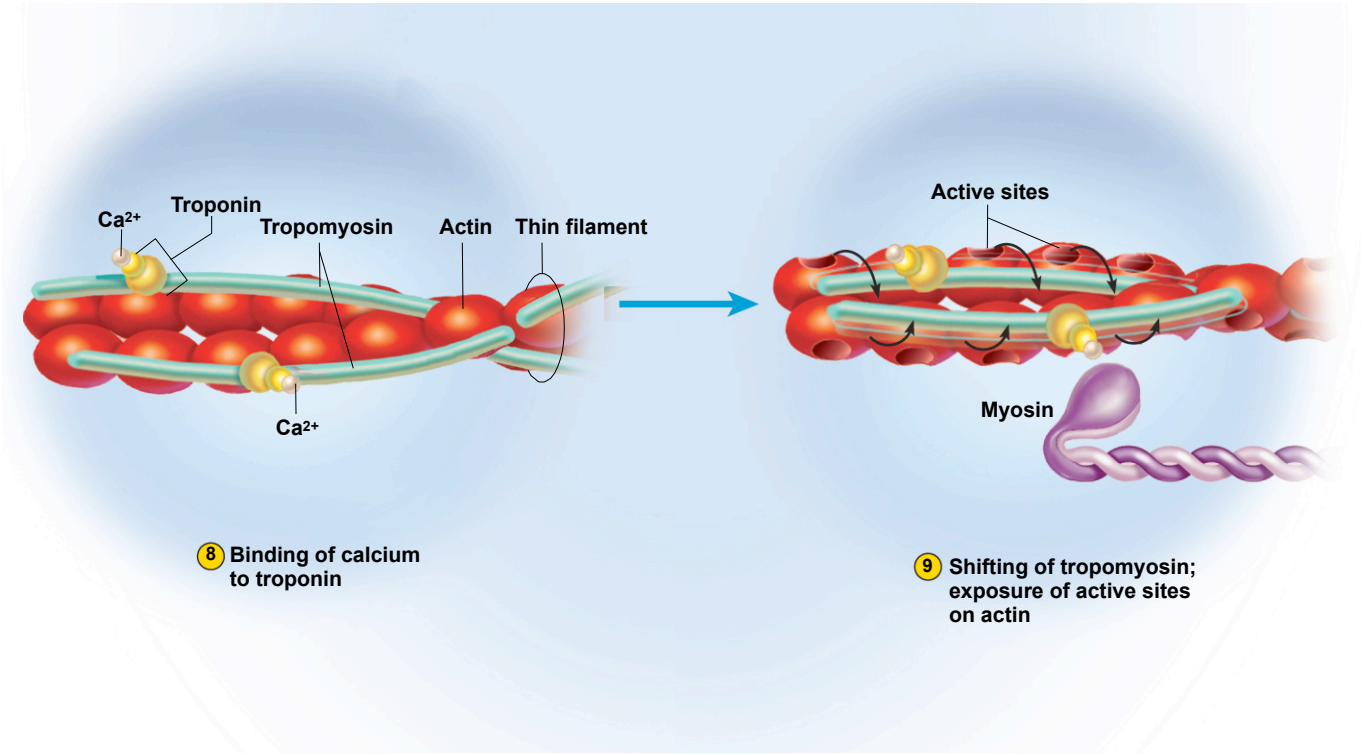
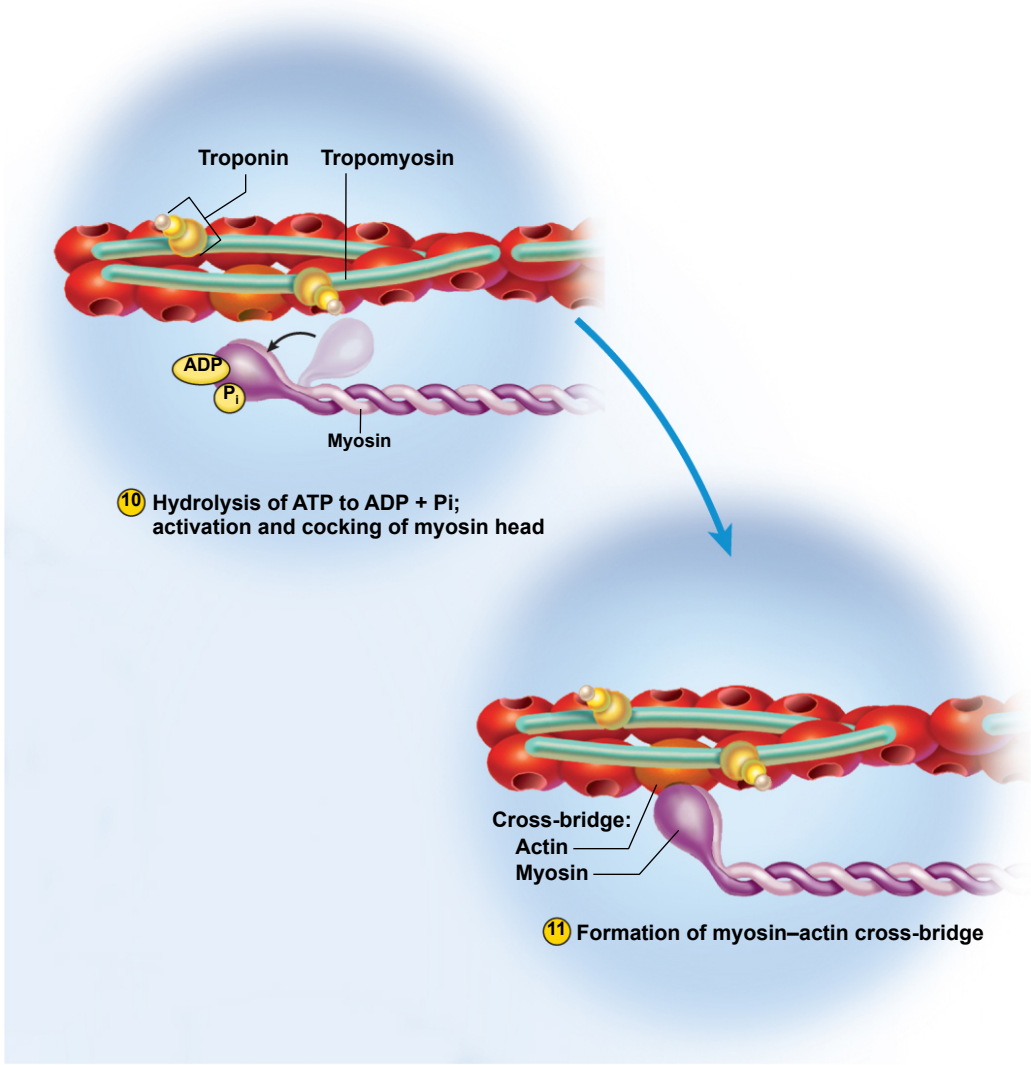


Figure 11.9 (8-9)

- calcium binds to troponin in thin filaments
- troponin-tropomyosin complex changes shape and exposes active sites on actin

Contraction (steps 10 and 11)



- myosin ATPase enzyme in myosin head hydrolyzes an ATP molecule
- activates the head "cocking" it in an extended position
  - ADP + P_i remain attached
- head binds to actin active site forming a myosin - actin cross-bridge

Figure 11.10 (10-11)

Contraction (steps 12 and 13)

- myosin head releases ADP and P_i, flexes pulling thin filament past thick - **power stroke**

- upon binding more ATP, myosin releases actin and process is repeated
  - each head performs 5 power strokes per second
  - each stroke utilizes one molecule of ATP

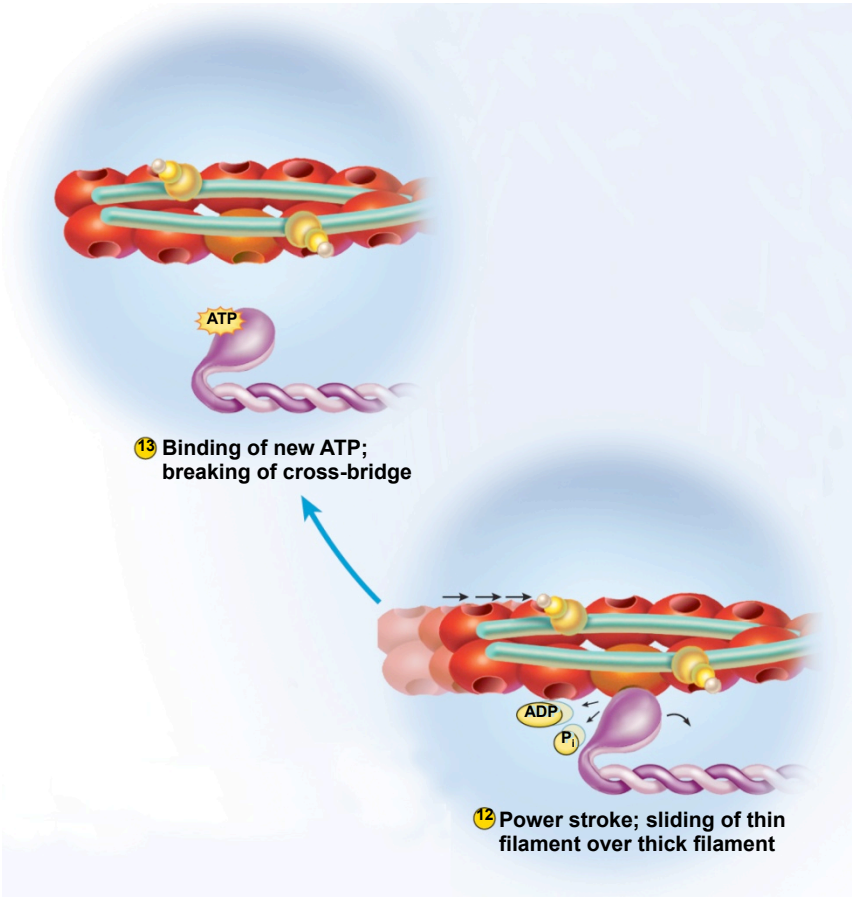


Figure 11.10 (12-13)

Relaxation (steps 14 and 15)

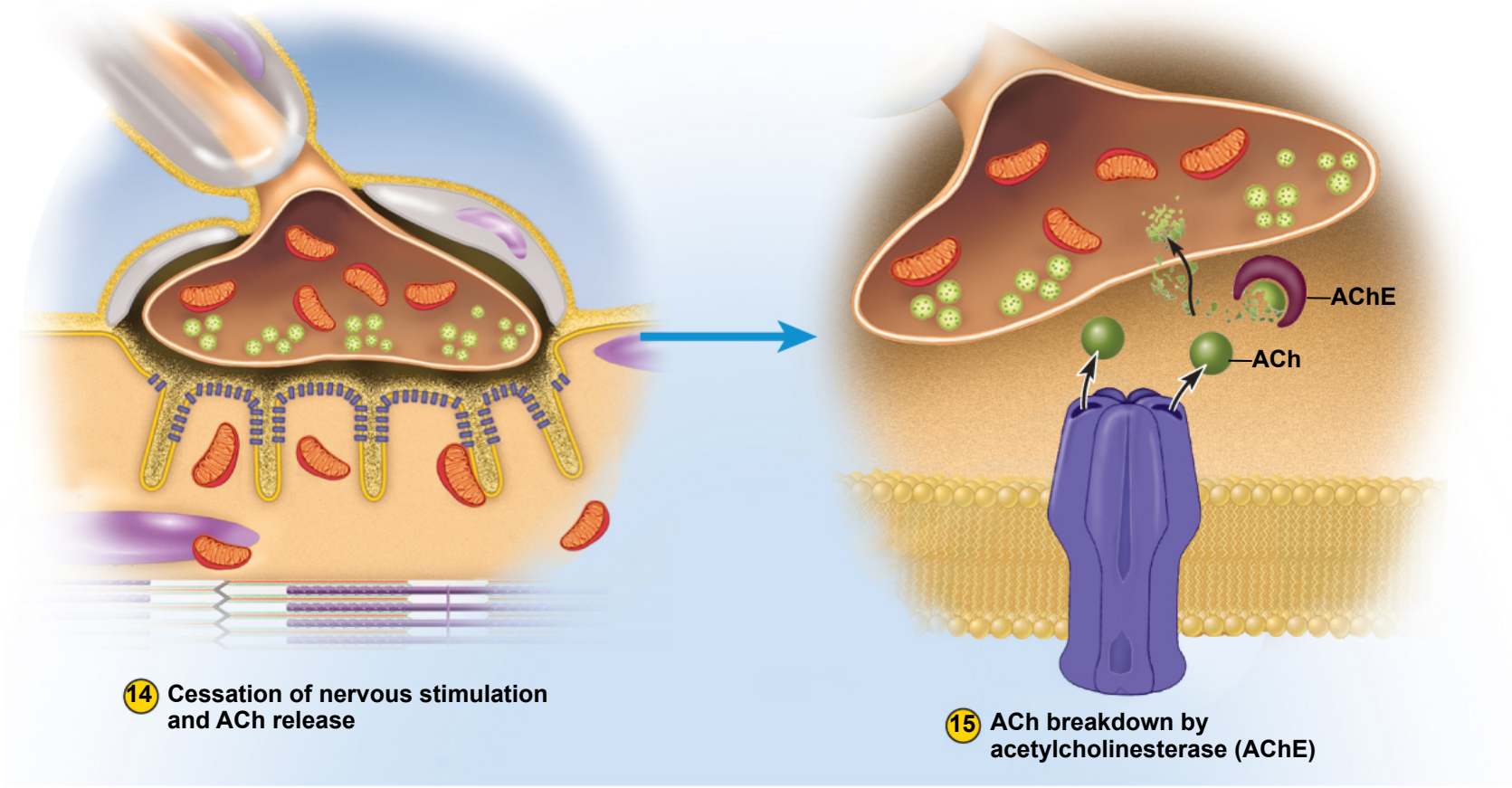


Figure 11.11 (14-15)

- nerve stimulation & ACh release stop
- AChE breaks down ACh & fragments reabsorbed into synaptic knob
- stimulation by ACh stops

Relaxation (step 16)

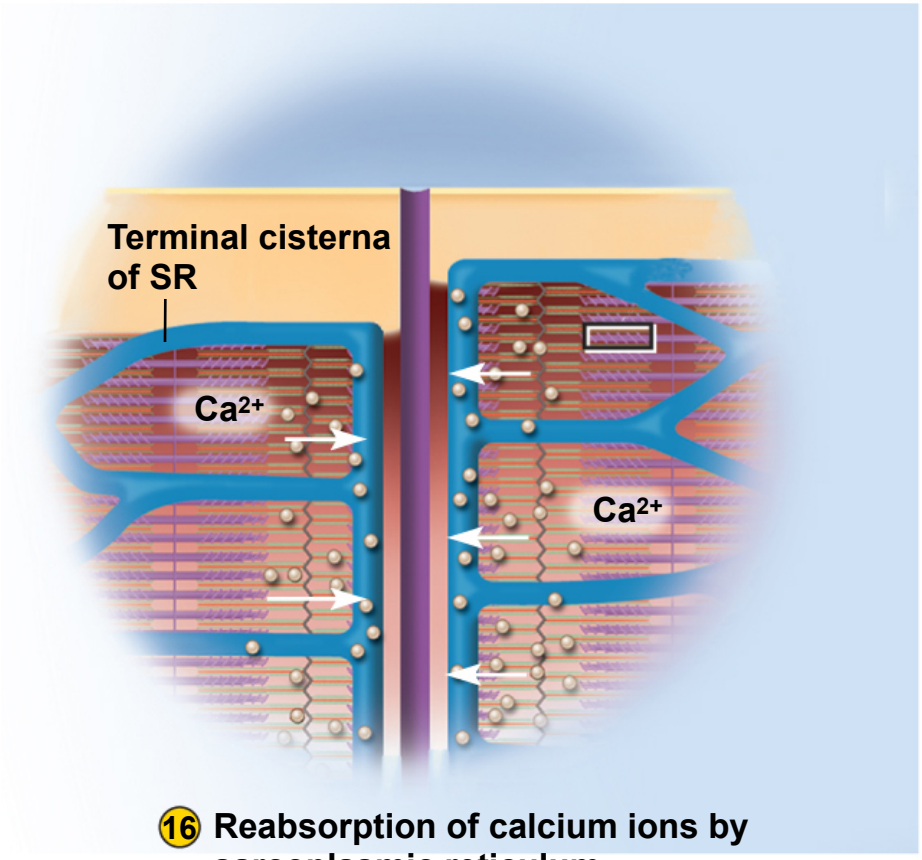


Figure 11.11 (16)

- Ca^{+2} pumped back into SR by active transport. Ca^{+2} binds to calsequestrin while in storage in SR
- ATP is needed for muscle relaxation as well as muscle contraction.

Relaxation (steps 17 and 18)

- Ca^{+2} removed from troponin is pumped back into SR
- tropomyosin reblocks the active sites
- muscle fiber ceases to produce or maintain tension
- muscle fiber returns to its resting length
  - due to recoil of elastic components & contraction of antagonistic muscles

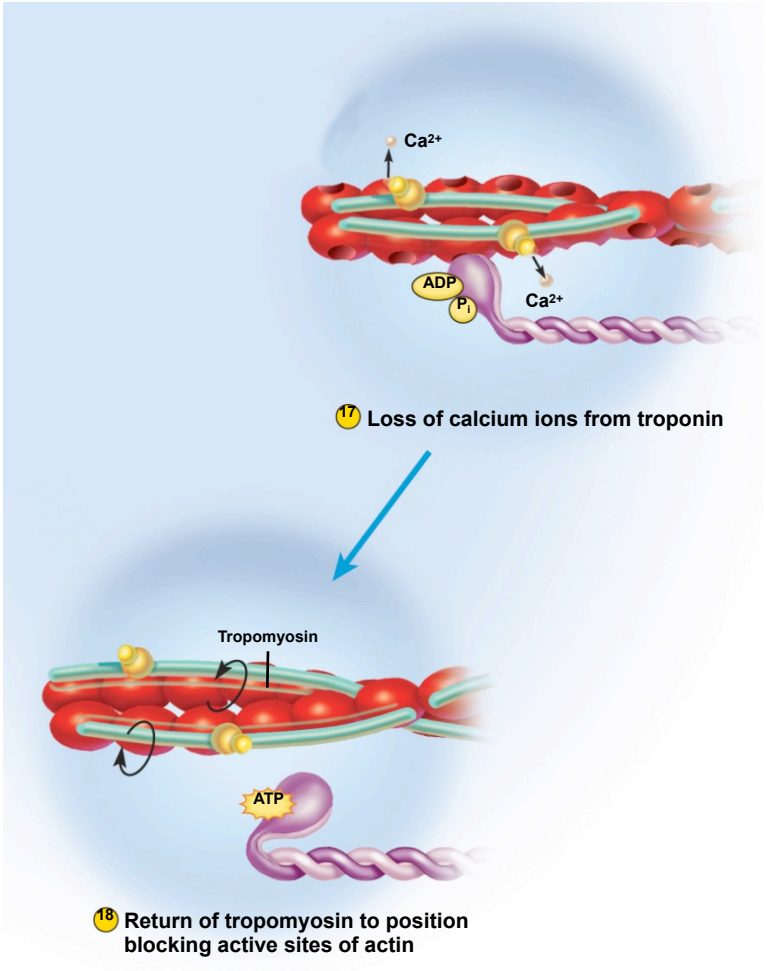


Figure 11.11 (17-18)

Rigor Mortis

- **rigor mortis** - hardening of muscles and stiffening of body beginning 3 to 4 hours after death
  - deteriorating sarcoplasmic reticulum releases Ca^{+2}
  - deteriorating sarcolemma allows Ca^{+2} to enter cytosol
  - Ca^{+2} activates myosin-actin cross-bridging
  - muscle contracts, but can not relax.
- muscle relaxation requires ATP, and ATP production is no longer produced after death
  - fibers remain contracted until myofilaments begins to decay
- rigor mortis peaks about 12 hours after death, then diminishes over the next 48 to 60 hours

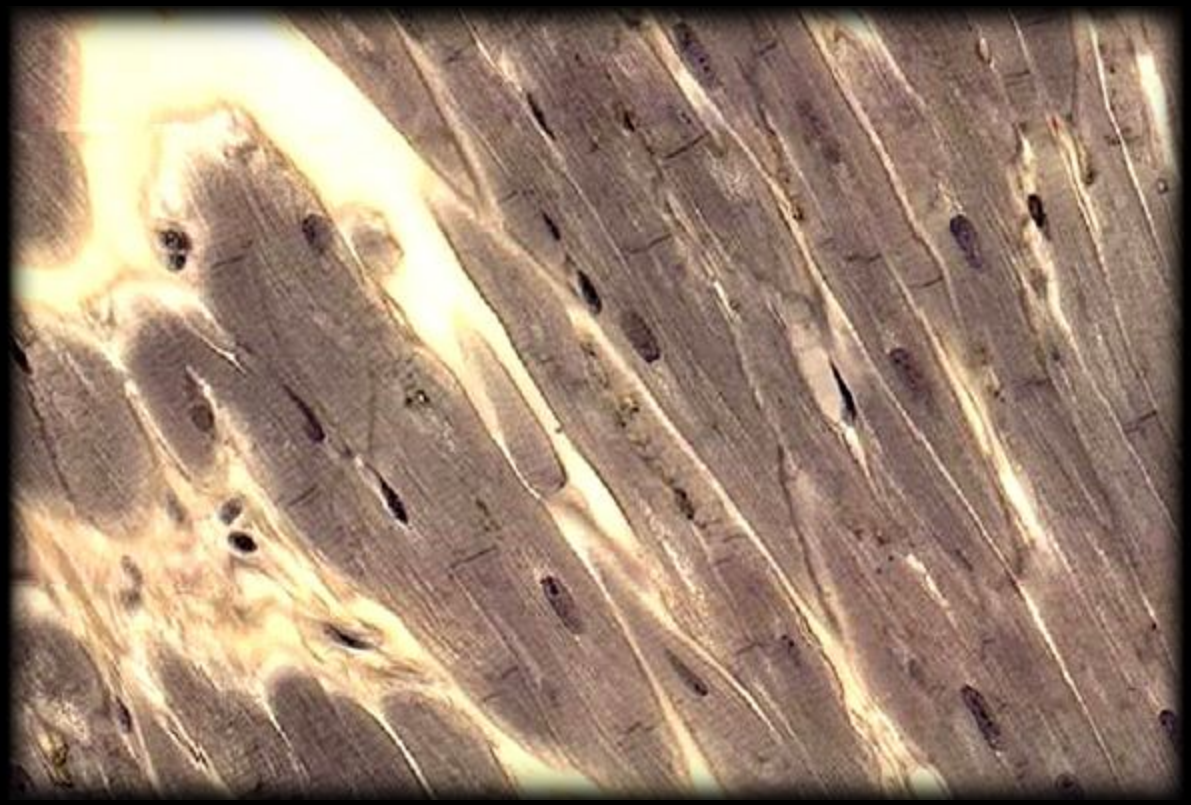
Cardiac Muscle

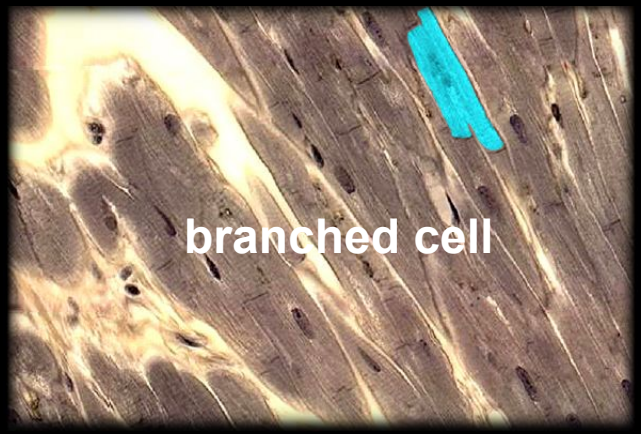
- limited to the heart where it functions to pump blood
- required properties of cardiac muscle
  - contraction with regular rhythm
  - muscle cells of each chamber must contract in unison
  - contractions must last long enough to expel blood
  - must work in sleep or wakefulness, with out fail, and without conscious attention
  - must be highly resistant to fatigue

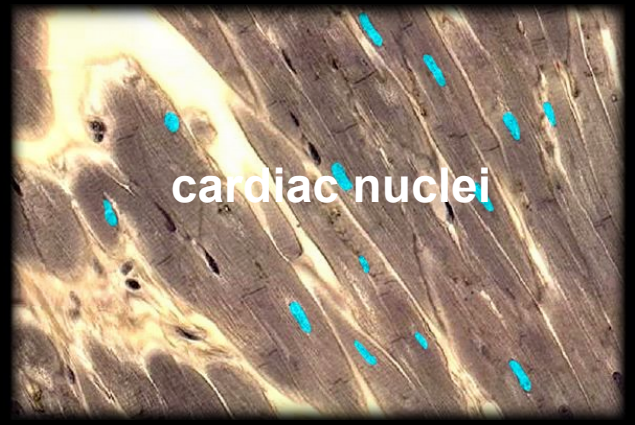
Cardiac Muscle

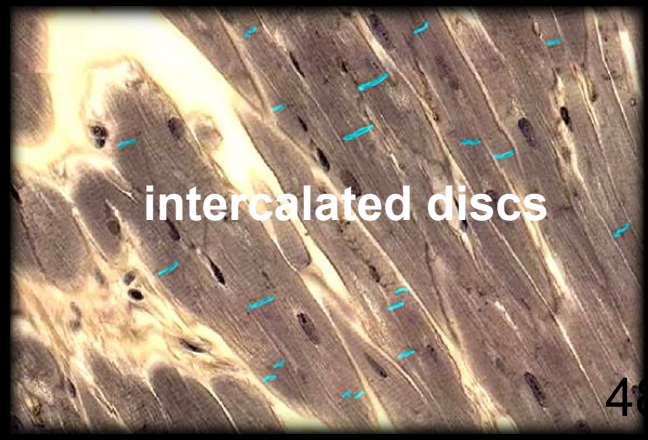
- characteristics of cardiac muscle cells
  - **striated** like skeletal muscle, but myocytes (cardiocytes) are **shorter and thicker**
  - each myocyte is joined to several others at the uneven, notched linkages – **intercalated discs**
    - appear as thick dark lines in stained tissue sections
    - electrical **gap junctions** allow each myocyte to directly stimulate its neighbors
    - mechanical junctions that keep the myocytes from pulling apart
  - sarcoplasmic reticulum less developed, but T tubules are larger and admit supplemental Ca^{2+} from the extracellular fluid
  - damaged cardiac muscle cells repair by **fibrosis**
    - a little mitosis observed following heart attacks
    - not in significant amounts to regenerate functional muscle

Cardiac Muscle





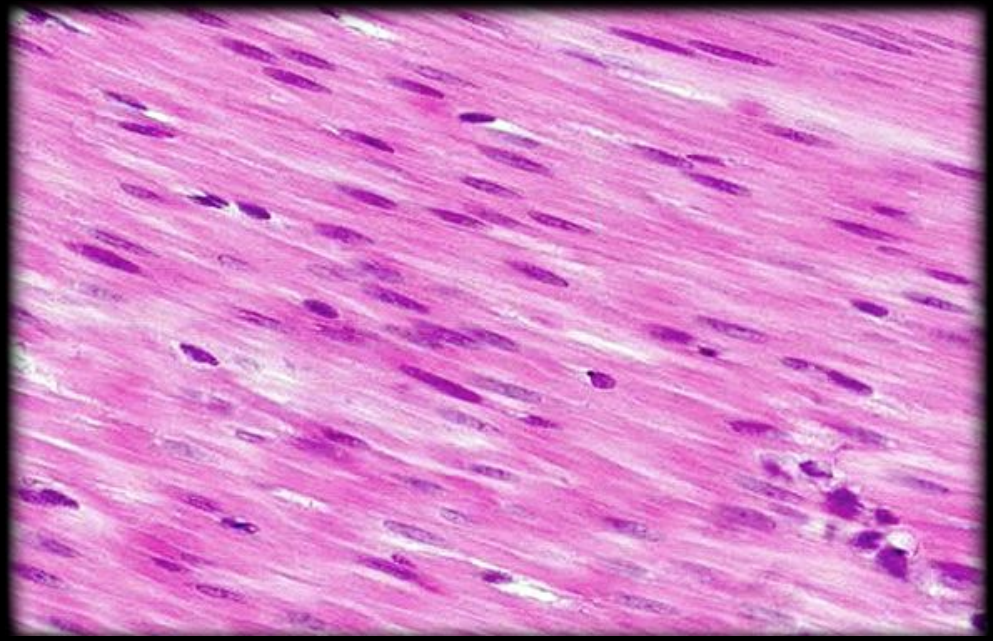


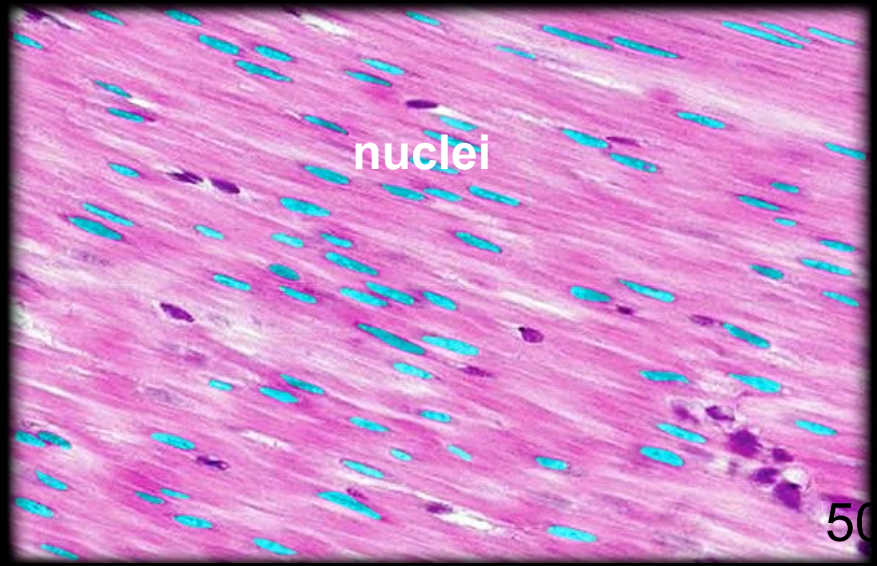


Cardiac Muscle

- can contract without need for nervous stimulation
  - contains a built-in **pacemaker** that rhythmically sets off a wave of electrical excitation
  - wave travels through the muscle and triggers contraction of heart chambers
  - **autorhythmic** – because of its ability to contract rhythmically and independently
- **autonomic nervous system** does send nerve fibers to the heart
  - can increase or decrease heart rate and contraction strength
- very slow twitches - does not exhibit quick twitches like skeletal muscle
  - maintains tension for about 200 to 250 msec
  - gives the heart time to expel blood
- uses **aerobic respiration** almost exclusively
  - rich in myoglobin and glycogen
  - has especially **large mitochondria**
    - 25% of volume of cardiac muscle cell
    - 2% of skeletal muscle cell with smaller mitochondria
- very adaptable with respect to fuel used
- very vulnerable to interruptions of oxygen supply
- highly fatigue resistant

Smooth Muscle



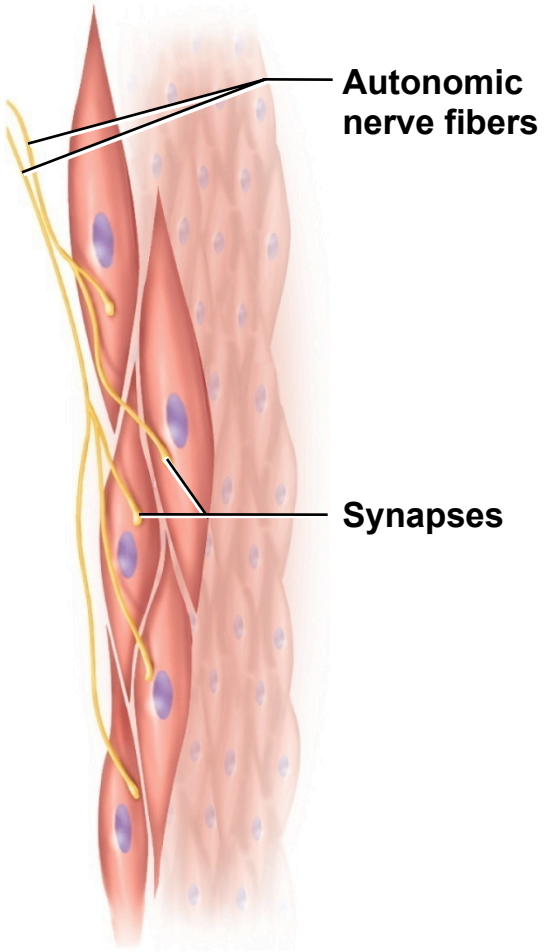


Smooth Muscle

- composed of myocytes that have a **fusiform shape**
- there is only **one nucleus**, located near the middle of the cell
- **no visible striations**
  - reason for the name ‘smooth muscle’
  - thick and thin filaments are present, but not aligned with each other
- z discs are absent and replaced by **dense bodies**
  - well ordered array of protein masses in cytoplasm
  - **protein plaques** on the inner face of the plasma membrane
- cytoplasm contains extensive cytoskeleton of intermediate filament
  - attach to the membrane plaques and dense bodies
  - provide mechanical linkages between the thin myofilaments and the plasma membrane
- sarcoplasmic reticulum is scanty and there are no T tubules
- Ca^{2+} needed for muscle contraction comes from the ECF by way of Ca^{2+} channels in the sarcolemma
- some smooth muscles lack nerve supply, while others receive autonomic fibers, not somatic motor fibers as in skeletal muscle
- capable of mitosis and hyperplasia
- injured smooth muscle regenerates well

2 Types of Smooth Muscle

- **multiunit smooth muscle**
  - occurs in some of the largest arteries and pulmonary air passages, in piloerector muscles of hair follicle, and in the iris of the eye
  - autonomic innervation similar to skeletal muscle
    - terminal branches of a nerve fiber synapse with individual myocytes and form a motor unit
    - each motor unit contracts independently of the others

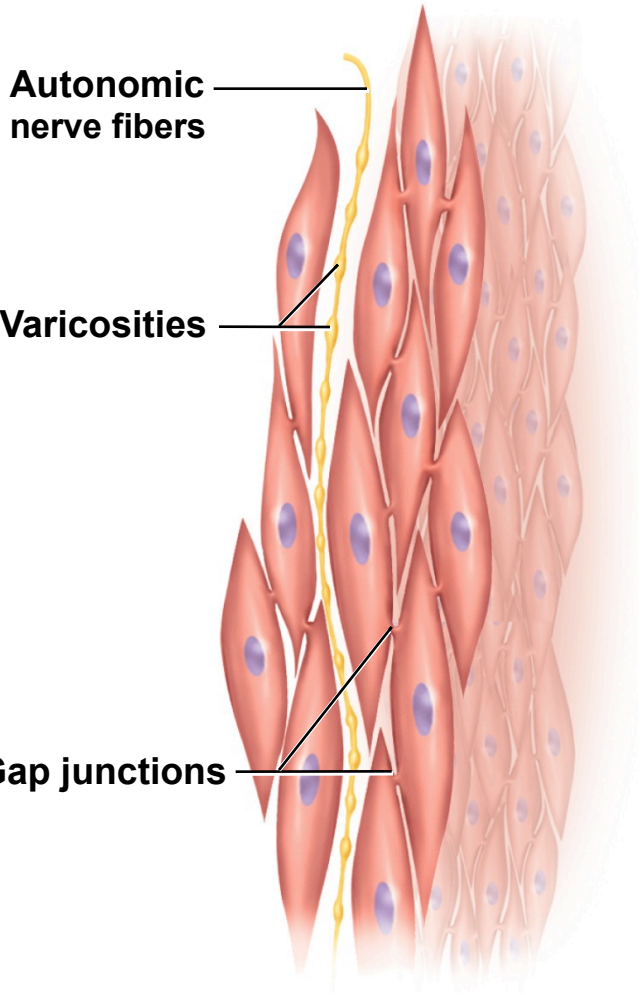


(a) Multiunit smooth muscle

Figure 11.21a

2 Types of Smooth Muscle

- **single-unit smooth muscle**
  - more widespread
  - occurs in most blood vessels, in the digestive, respiratory, urinary, and reproductive tracts – also called **visceral muscle**
    - often in two layers
      - inner circular
      - outer longitudinal
  - myocytes of this cell type are electrically coupled to each other by **gap junctions**
  - they directly stimulate each other and a large number of cells contract as a **single unit**



(b) Single-unit smooth muscle

Figure 11.21b

Layers of Visceral Muscle

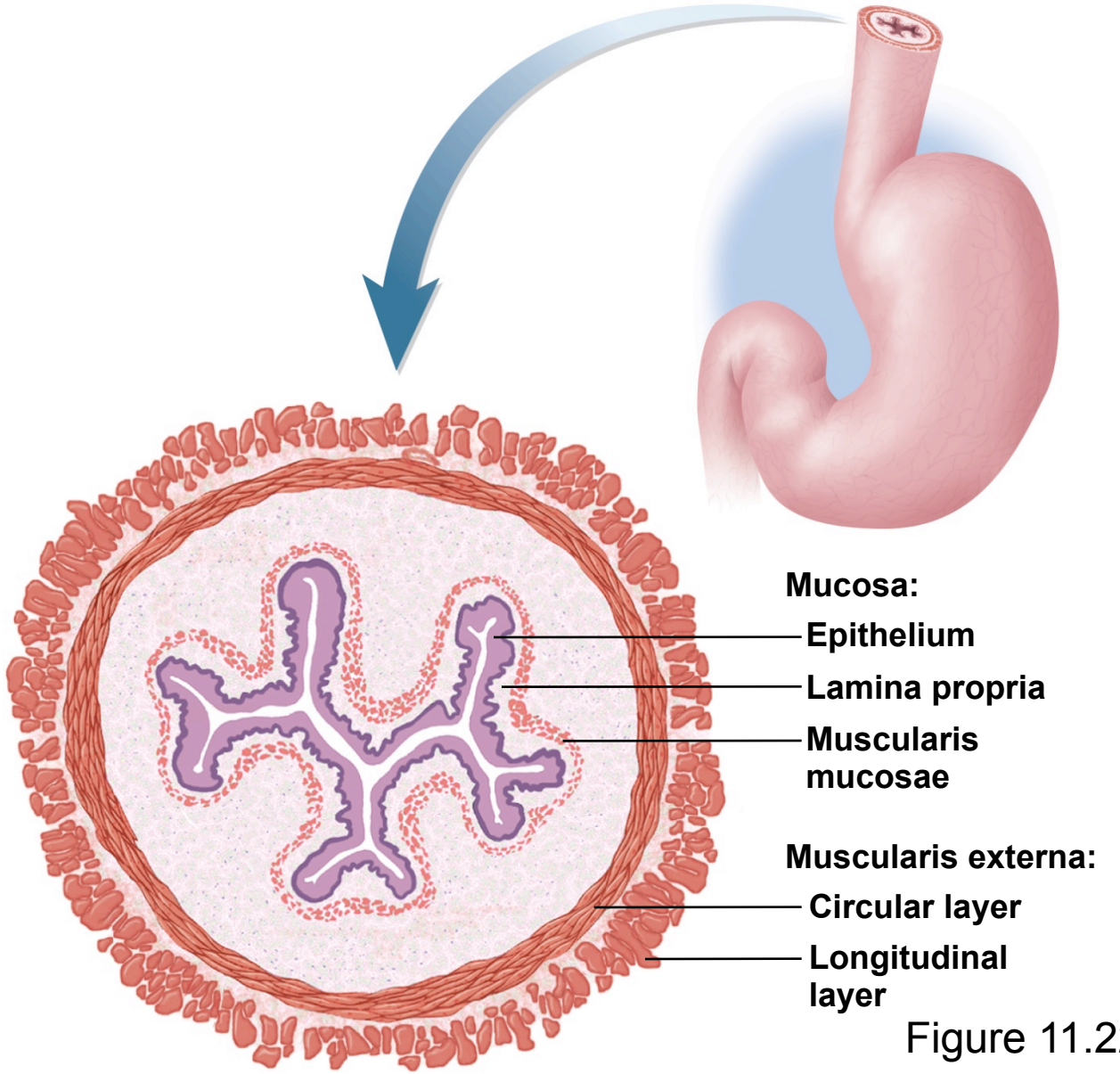


Figure 11.22

Stimulation of Smooth Muscle

- smooth muscle is **involuntary** and can contract without nervous stimulation
  - can contract in response to chemical stimuli
    - hormones, carbon dioxide, low pH, and oxygen deficiency
    - in response to stretch
    - single unit smooth muscle in stomach and intestines has **pacemaker cells** that set off waves of contraction throughout the entire layer of muscle
- most smooth muscle is innervated by **autonomic nerve fibers**
  - can trigger and modify contractions
  - stimulate smooth muscle with either acetylcholine or norepinephrine
  - can have contrasting effects
    - relax the smooth muscle of arteries
    - contract smooth muscles of the bronchioles
- in single unit smooth, each autonomic nerve fibers has up to 20,000 beadlike swelling called **varicosities**
  - each contains synaptic vesicles and a few mitochondria
  - nerve fiber passes amid several myocytes and stimulates all of them at once when it releases its neurotransmitter
    - no motor end plates, but receptors scattered throughout the surface – **diffuse junctions –** no one-to-one relationship between nerve fiber and myocyte

Stimulation of Smooth Muscle

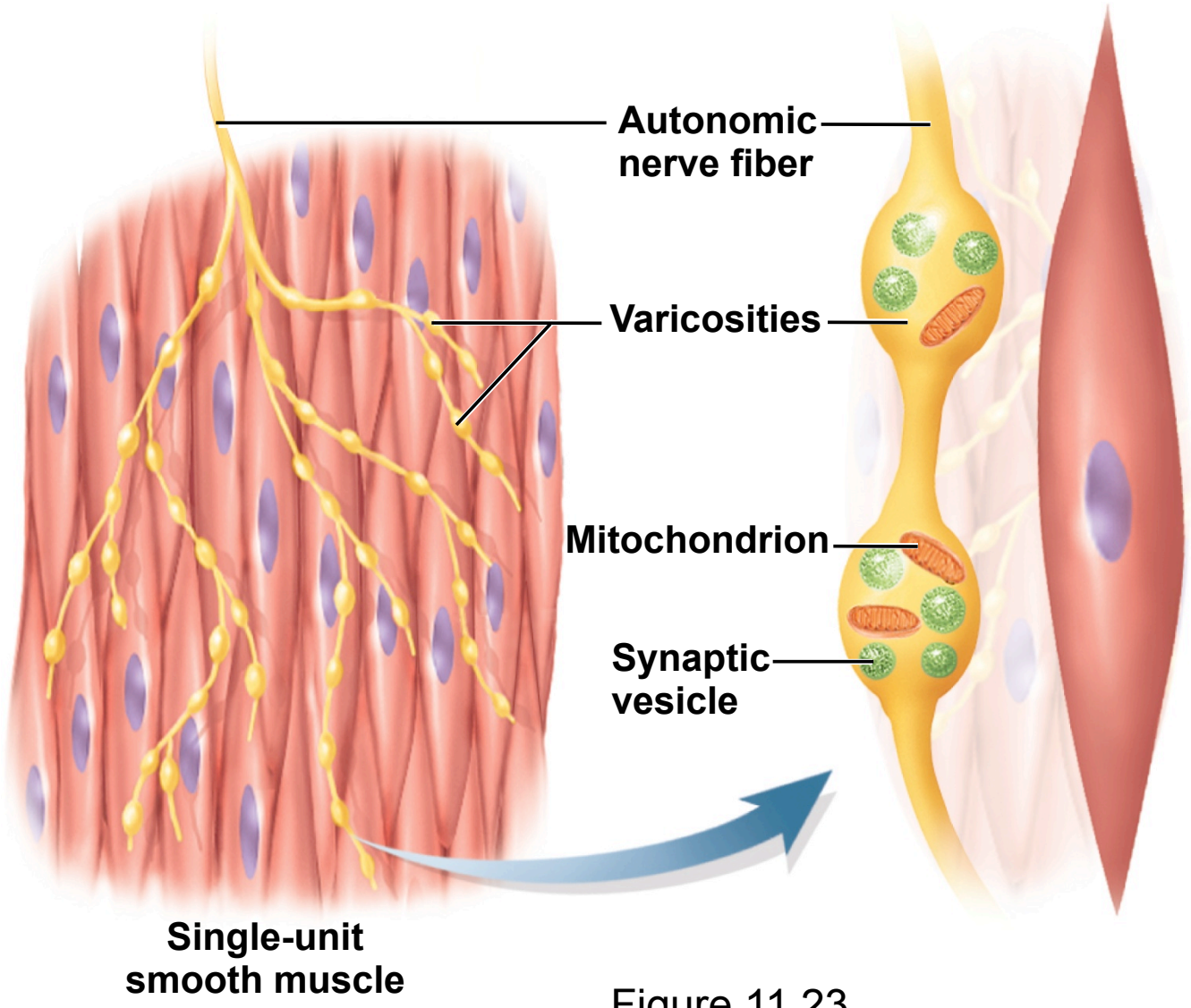


Figure 11.23

Contraction and Relaxation

- contraction is triggered by Ca^{+2}, energized by ATP, and achieved by sliding thin past thick filaments
- contraction begins in response to Ca^{+2} that enters the cell from ECF, a little internally from sarcoplasmic reticulum
  - voltage, ligand, and mechanically-gated (stretching) Ca^{+2} channels open to allow Ca^{+2} to enter cell
- calcium binds to **calmodulin** on thick filaments
  - activates **myosin light-chain kinase –** adds phosphate to regulatory protein on myosin head
  - and **myosin ATPase**, hydrolyzing ATP
    - enables myosin similar power and recovery strokes like skeletal muscle
  - thick filaments pull on thin ones, thin ones pull on dense bodies and membrane plaques
  - force is transferred to plasma membrane and entire cell shortens
  - puckers and twists like someone wringing out a wet towel

Contraction and Relaxation

- contraction and relaxation very slow in comparison to skeletal muscle
  - latent period in skeletal 2 msec, smooth muscle 50 - 100 msec
  - tension peaks at about 500 msec (0.5 sec)
  - declines over a period of 1 – 2 seconds
  - slows myosin ATPase enzyme and slow pumps that remove Ca^{+2}
  - Ca^{+2} binds to calmodulin instead of troponin
    - activates kinases and ATPases that hydrolyze ATP

- **latch-bridge mechanism** is resistant to fatigue
  - heads of myosin molecules do not detach from actin immediately
  - do not consume any more ATP
  - maintains tetanus tonic contraction (smooth muscle tone)
    - arteries – vasomotor tone intestinal tone
  - makes most of its ATP aerobically

Contraction of Smooth Muscle

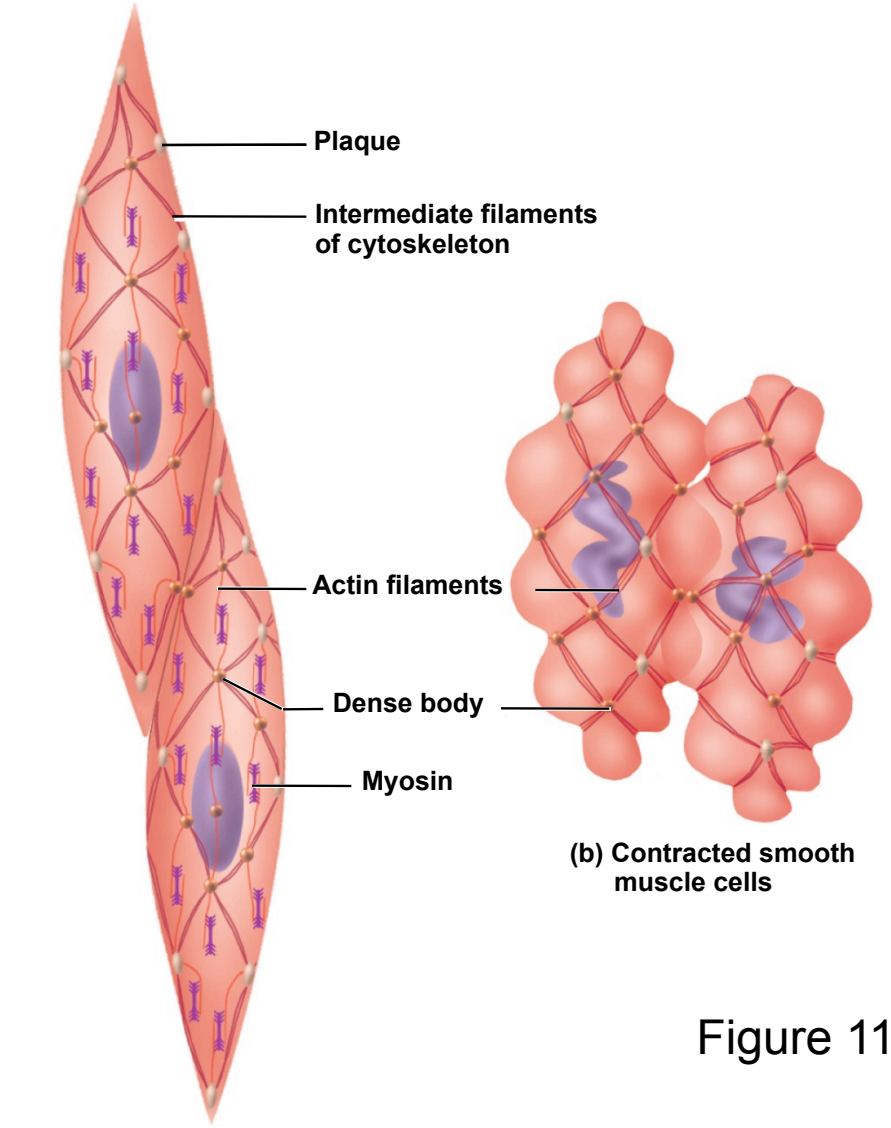


Figure 11.24

Stretching Smooth Muscle

- **stretch** can open mechanically-gated calcium channels in the sarcolemma causing contraction
  - **peristalsis** – waves of contraction brought about by food distending the esophagus or feces distending the colon
    - propels contents along the organ
- **stress-relaxation response** (receptive relaxation) -helps hollow organs gradually fill (urinary bladder)
  - when stretched, tissue briefly contracts then relaxes – helps prevent emptying while filling

Contraction and Stretching

- skeletal muscle cannot contract forcefully if overstretched
- smooth muscle contracts forcefully even when greatly stretched
  - allows hollow organs such as the stomach and bladder to fill and then expel their contents efficiently
- smooth muscle can be anywhere from half to twice its resting length and still contract powerfully
- three reasons:
  - there are no z discs, so thick filaments cannot butt against them and stop contraction
  - since the thick and thin filaments are not arranged in orderly sarcomeres, stretching does not cause a situation where there is too little overlap for cross-bridges to form
  - the thick filaments of smooth muscle have myosin heads along their entire length, so cross-bridges can form anywhere
- **plasticity** – the ability to adjust its tension to the degree of stretch
  - a hollow organ such as the bladder can be greatly stretched yet not become flabby when it is empty

Muscular Dystrophy

- **muscular dystrophy** - group of hereditary diseases in which skeletal muscles degenerate and weaken, and are replaced with fat and fibrous scar tissue

- **Duchenne muscular dystrophy** is caused by a sex-linked recessive trait (1 of 3500 live-born boys)
  - most common form
  - disease of males – diagnosed between 2 and 10 years of age
  - mutation in gene for muscle protein **dystrophin**
    - actin not linked to sarcolemma and cell membranes damaged during contraction, necrosis and scar tissue results
  - rarely live past 20 years of age due to affects on respiratory and cardiac muscle – incurable

- **facioscapulohumeral MD** - autosomal dominant trait affecting both sexes equally
  - facial and shoulder muscles more than pelvic muscles

- **limb-girdle dystrophy**
  - combination of several diseases of intermediate severity
  - affects shoulder, arm, and pelvic muscles

Myasthenia Gravis

- autoimmune disease in which antibodies attack neuromuscular junctions and bind ACh receptors together in clusters
  - disease of women between 20 and 40
  - muscle fibers then removes the clusters of receptors from the sarcolemma by endocytosis
  - fiber becomes less and less sensitive to ACh
  - effects usually first appear in facial muscles
    - drooping eyelids and double vision, difficulty swallowing, and weakness of the limbs
  - **strabismus** – inability to fixate on the same point with both eyes
- treatments
  - **cholinesterase inhibitors** retard breakdown of ACh allowing it to stimulate the muscle longer
  - **immunosuppressive agents** suppress the production of antibodies that destroy ACh receptors
  - **thymus removal** (thymectomy) – helps to dampen the overactive immune response that causes myasthenia gravis
  - **plasmapheresis** –technique to remove harmful antibodies from blood plasma