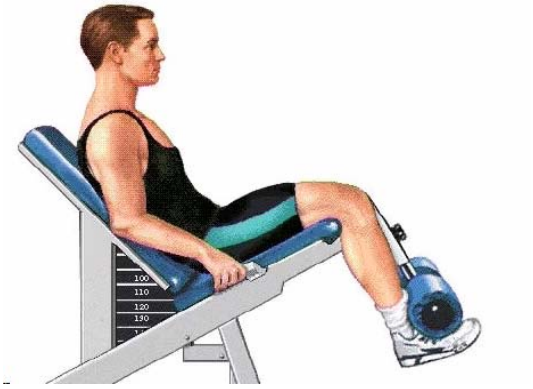
REVISED
11:10 am, Feb 25, 2008

Entire PDF Audio File In Real Media Format

The Muscular System



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3 Types of Muscle Tissue

Muscle Type	Location	Characteristics	Control
Skeletal	Attached to the bones for movement	Long, cynlindrical cells; multinucleated, striated	Voluntary
Cardiac	Muscle of the Heart	Short, branching cells, mononucleated, faintly striated. Forms functional syncytia.	Involuntary myogenic
Smooth Muscle	**Single Unit**: GI, Respiratory, & Genitourinary tract mucous membranes. **Multi-unit:** smooth muscle in blood vessel walls.	Small oblong cells, mononucleated, also may form a functional syncytium.	Involuntary myogenic

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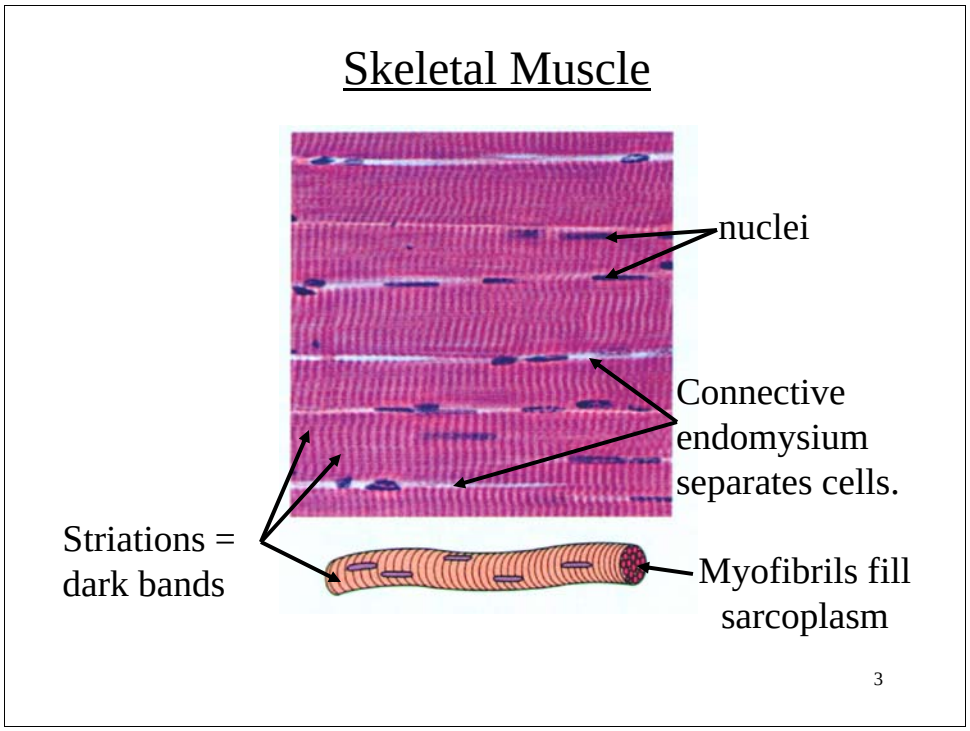
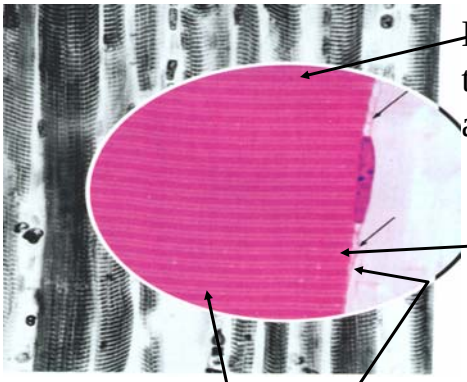


The nuclei and other organelles of skeletal muscle cells are found next to the sarcolemms and the majority of the **sarcoplasm** is filled with the contractile machinery of the cell, the **myofibrils**. Skeletal muscle cells are derived from individual **myocytes** which fuse to produce a mature multinucleated muscle fiber. There are few if any of the precursor myocytes found in a mature muscle, and so muscles produce no new cells after maturity. Individual cells respond to training by enlarging and building myofibrils and other components.

Skeletal Muscle photomicrographs



Dark striations = A-bands, the light areas between are the I-bands.

Z-line. From one Z line to the next is a sarcomere.

Z-line

The sarcolemma is the cell membrane

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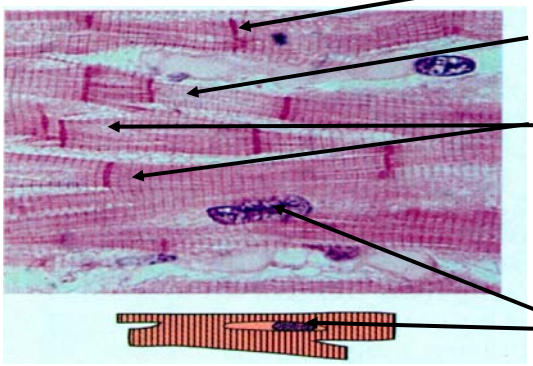
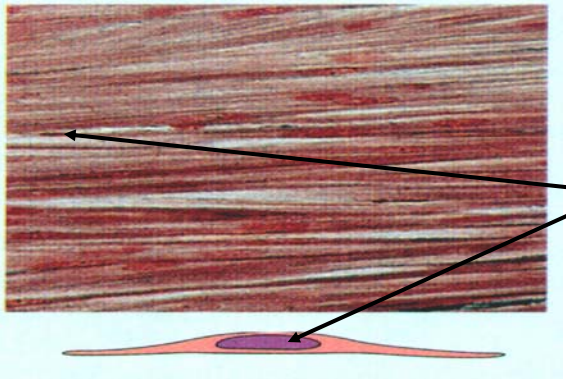
Cardiac Muscle



The action potential travels through all cells connected together in a syncytium causing them to function as a unit.

Cardiac muscle cells are much shorter than cells in skeletal muscle and they branch to connect to neighboring cells through specialized membranes called **intercalated disks** to form a network called a **syncytium**.

Smooth Muscle



Spindle -shaped mononucleated smooth muscle cell.

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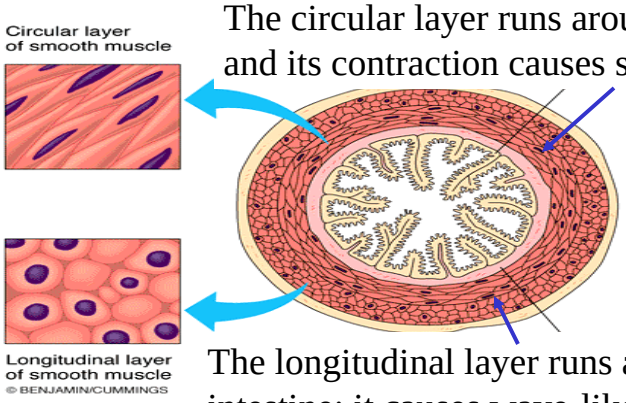
Smooth muscle cells connect to form single-unit **syncytia** similar to cardiac muscle. But impulses and contractions occur much more slowly in smooth than in cardiac muscle.

Smooth Muscle Arrangement

In the intestine smooth muscle forms two distinct layers, one running along, the other running around the organ. Together these layers cause movements which propel the contents.



The circular layer runs around the intestine and its contraction causes segmentation

The longitudinal layer runs along the intestine; it causes wave-like contractions.

Types of Smooth Muscle Fibers

Single unit smooth muscle – cells connected to function as a single unit (syncytium)

e.g. in GI tract

Multiunit smooth muscle – cells grouped into many contractile units controlled by the nervous system.

e.g. in blood vessel walls and sphincters in GI tract.

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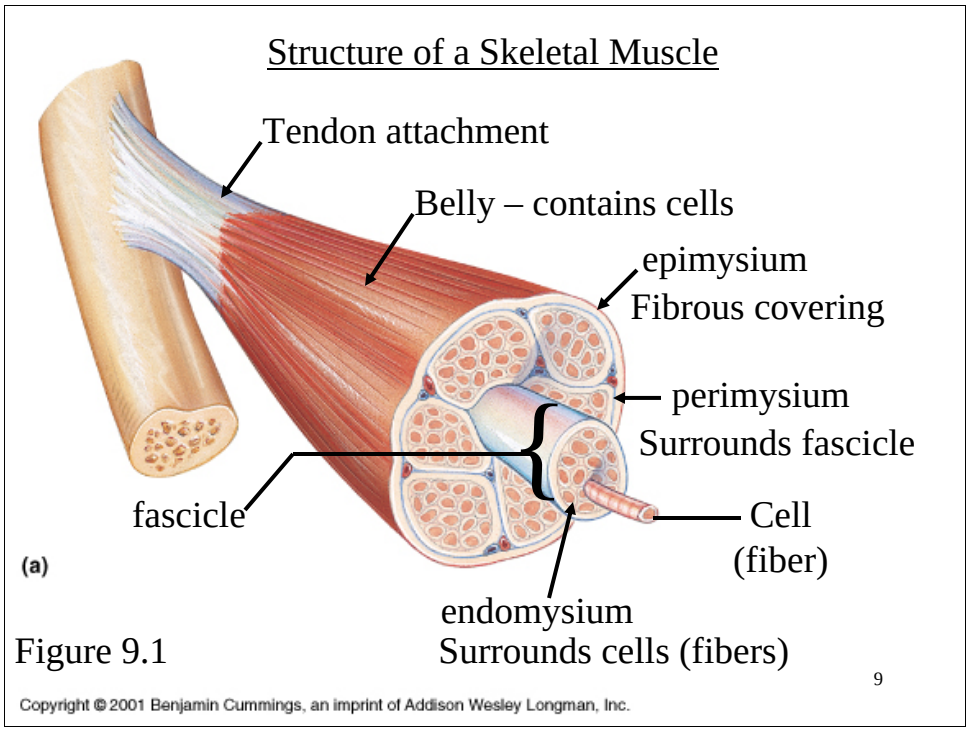

The hierarchy of connective tissues associated with a skeletal muscle provide a continuous connection between muscle cells and their action on a bone or other attachment. At the same time cells are effectively separated from one another and each is controlled by a separate nerve fiber.

Functional Characteristics of Skeletal Muscle

Excitability (responsiveness) – muscles can be stimulated by electrical, chemical, and physical means.

Contractility – a muscle responds to stimuli by contracting.

Elasticity – muscles tend to recoil to their resting length.

Extensibility – muscles can be stretched beyond their resting length.

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Muscle Attachments

Tendons – attach muscle to bone.

Aponeuroses – broad, flat, tendinous attachment.

Origin – more fixed point of attachment.

Insertion – more movable point of attachment.

Muscle action pulls insertion toward the origin.

A muscle can only pull, it cannot push.

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Types of Muscle Contractions

Agonist – the prime mover; the muscle which performs the movement in question.

Antagonist – the muscle that performs the opposing movement to that of the agonist.

Both muscles contract (exert tension) regardless of which is the agonist or antagonist.

On-center movement – that of the agonist

Off-center (eccentric) movement – that of the antagonist

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The antagonist may actually be stretching while it is generating tension (contracting).

Knee Extension

Agonist: the rectus femoris (quadriceps femoris)

Antagonist: the biceps femoris (hamstrings)

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Knee Flexion

Agonist: the biceps femoris (hamstrings)

Antagonist: the rectus femoris (quadriceps femoris)

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Synergists – muscles which work together to perform a movement; often differs from the movement either performs when working alone.

Fixators – muscles which work to keep a part from moving; stabilizers, neutralizers.

Assignment: make a list of 5 each antagonists, synergists, and fixators.

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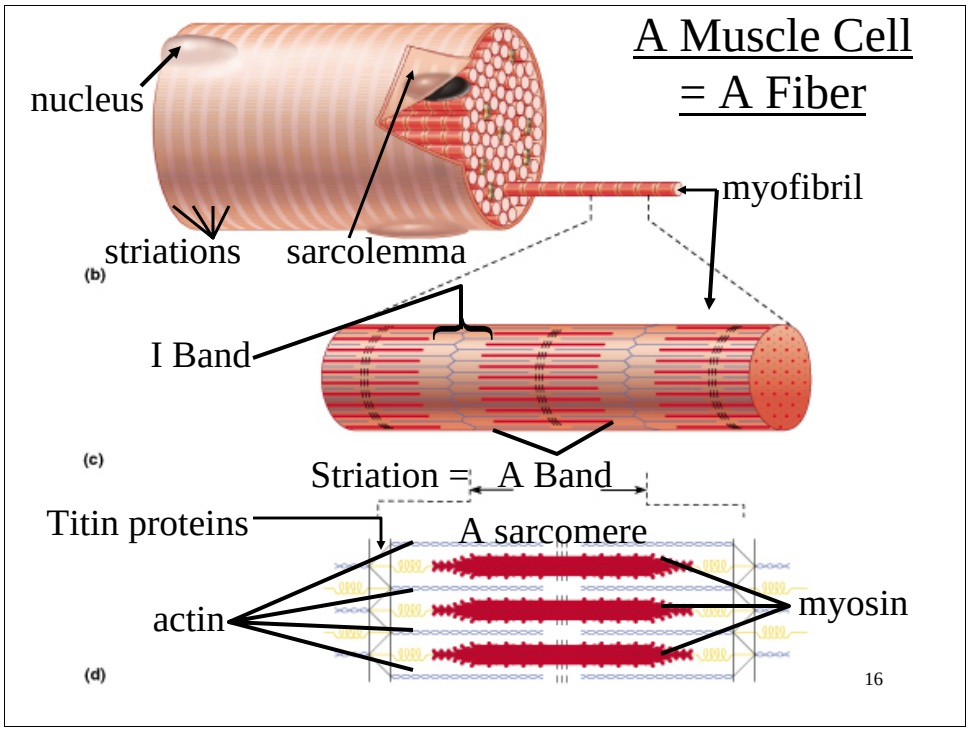
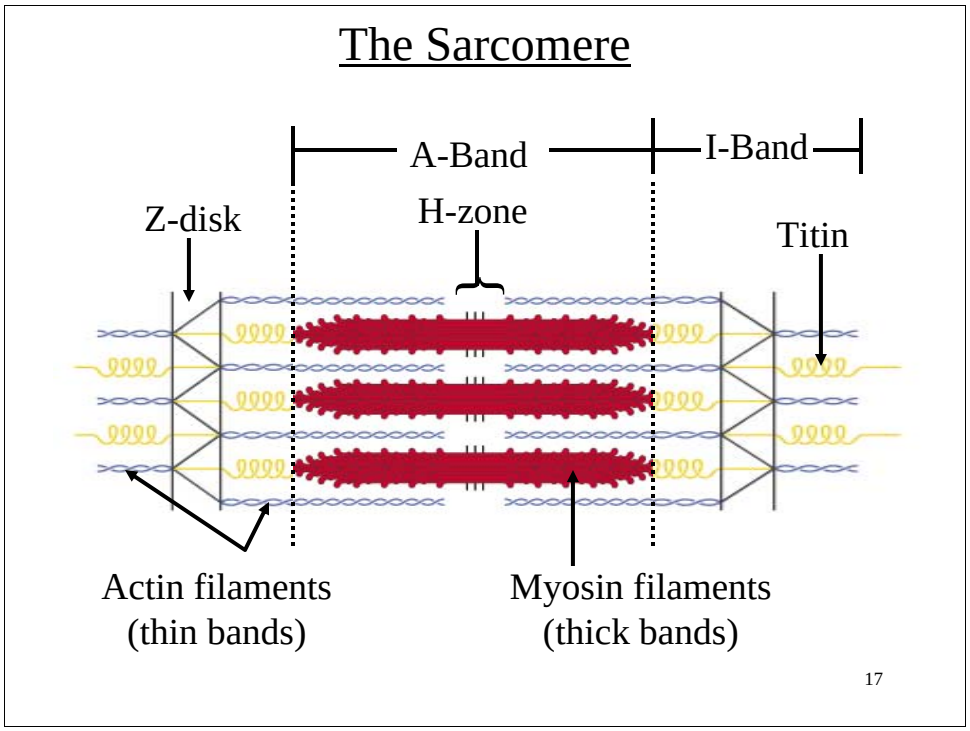
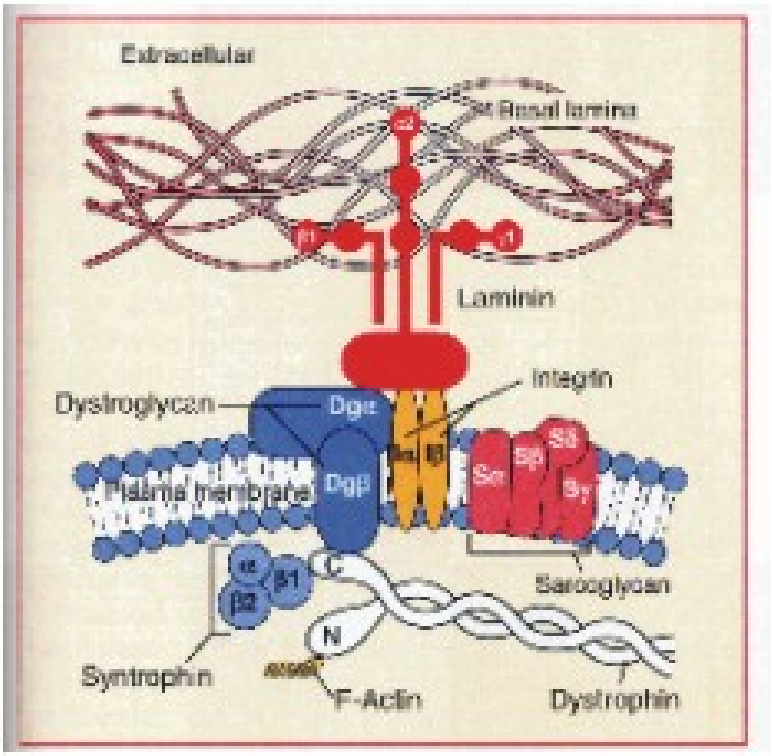
For each example you will need a couple of muscles: for antagonists you will need the two muscles which are antagonistic, for synergists the two or more muscles which work together, and for fixators the part of the body which is fixed and under what conditions.

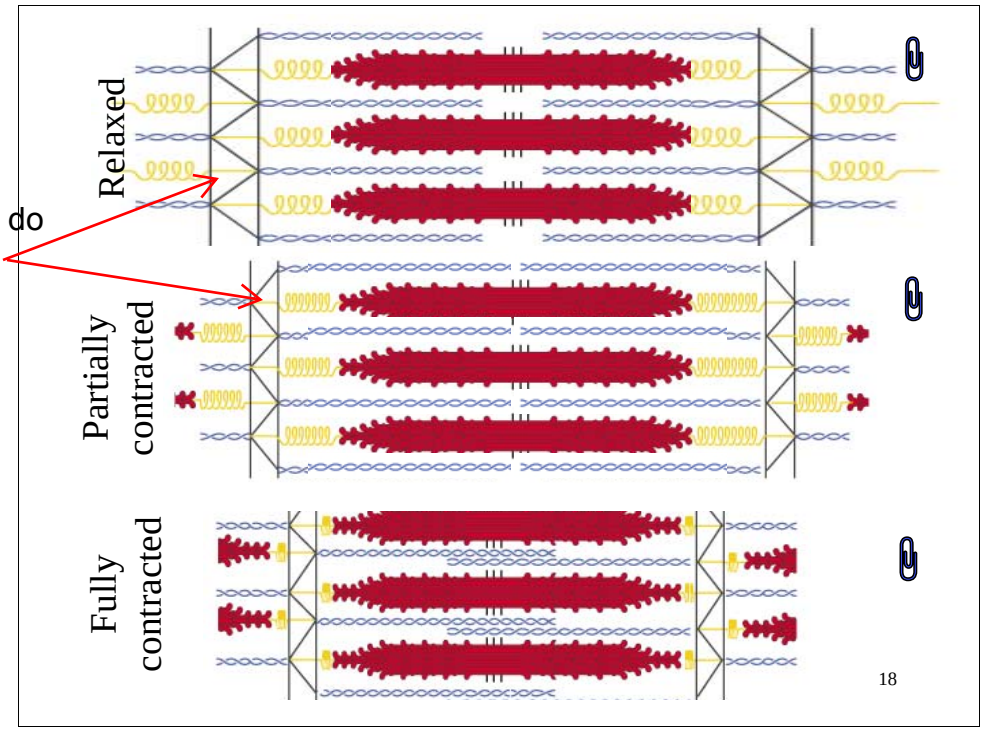


Look at the video clip showing muscle hierarchy. From the largest to smallest the hierarchy is as follows: whole **muscle** is composed of bundles of cells called **fasciculi**, individual **cells** are composed of **myofibrils**, which are organized of **myofilaments** of **actin** and **myosin** and other proteins arranged in a specific way.



Titin proteins are part of the structural support for the myofilaments of the sarcomere and also a part of the “series elastic elements” along with other proteins and connective tissues.





The Z-disks do not actually change.

Click on the paper clip icons for photo-micrographs of the sarcomere at various degrees of shortening. Note how the I bands disappear and, as a result, the sarcomere darkens.

In the relaxed state you can see the resting-length overlap of actin and myosin fibers. Note how the actin myofilaments move together in the partially contracted muscle, and themselves overlap when fully contracted. As the muscle contracts the z-disk of the sarcomere move toward one another and the sarcomere shortens. The actin and myosin myofilaments themselves do not shorten, nor does the Z-disk, despite its appearance in this view which is an artifact from making the graphic.

The Sliding Filament Mechanism
of Muscle Contraction

The shortening of sarcomeres, and the resulting muscle contraction, is due to the sliding of the actin and myosin myofilaments against one another.

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The Myosin Molecule

(a) Myosin molecule

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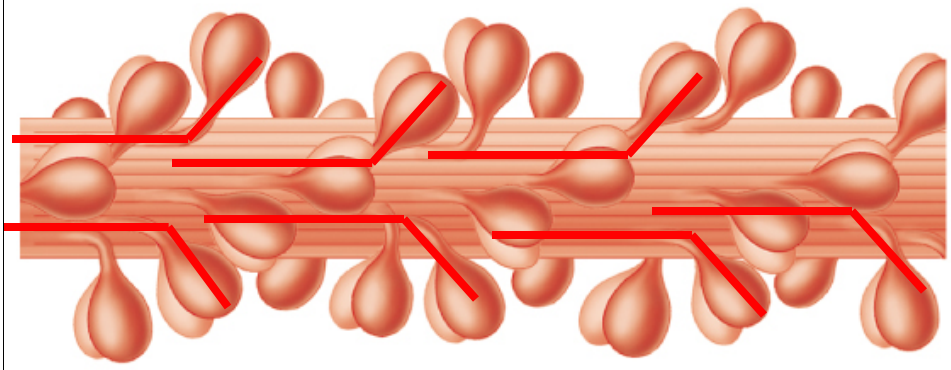
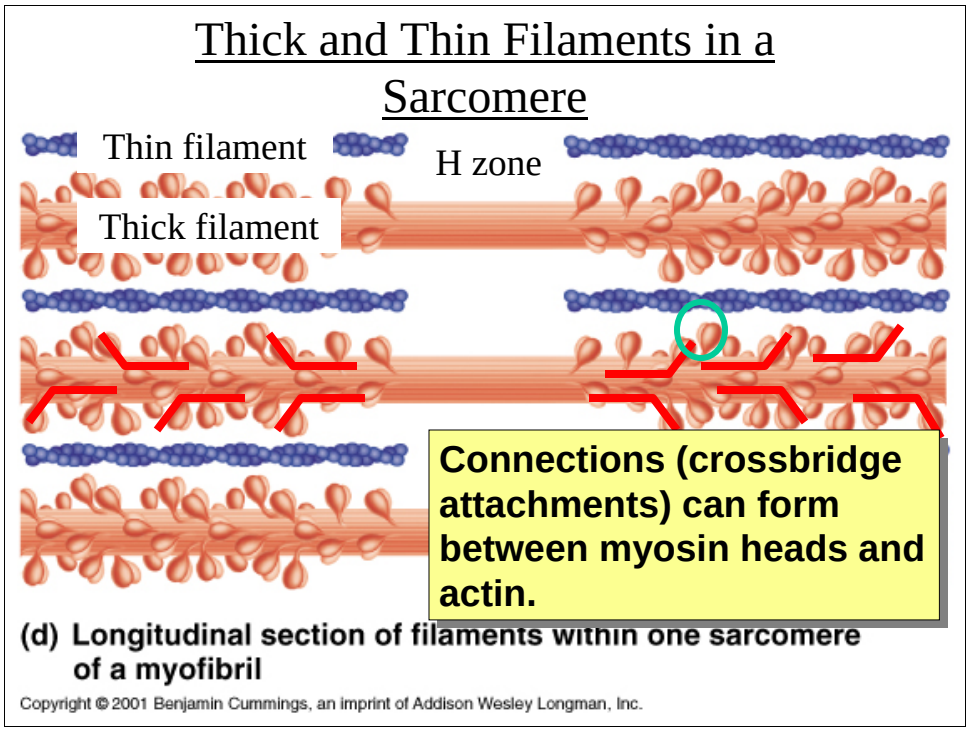
Each myosin molecule is actually composed of two “tails” and two “heads”.

The Thick Filament



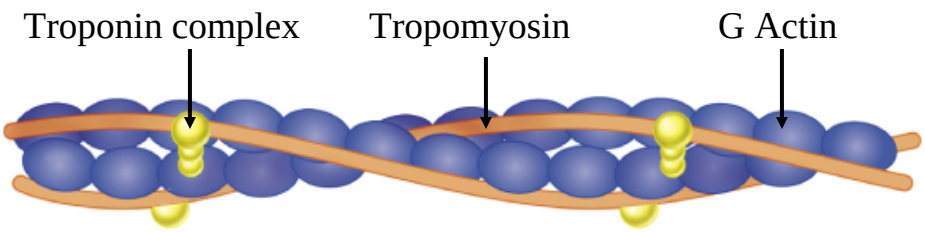
(b) Portion of a thick filament

Myosin molecules are arranged into the thick filaments with their tails parallel and their heads projecting toward the adjacent actin filaments.



Myosin heads project toward the acting filaments, with the heads angled toward the Z-disks on each side of the sarcomere. In the center of the thick myofilaments the H zone is bare of heads, with myosin tails only.

The Thin Filament



(c) Portion of a thin filament

Tropomyosin blocks the crossbridge attachment sites on actin.

Troponin shifts to move tropomyosin and expose the active sites.

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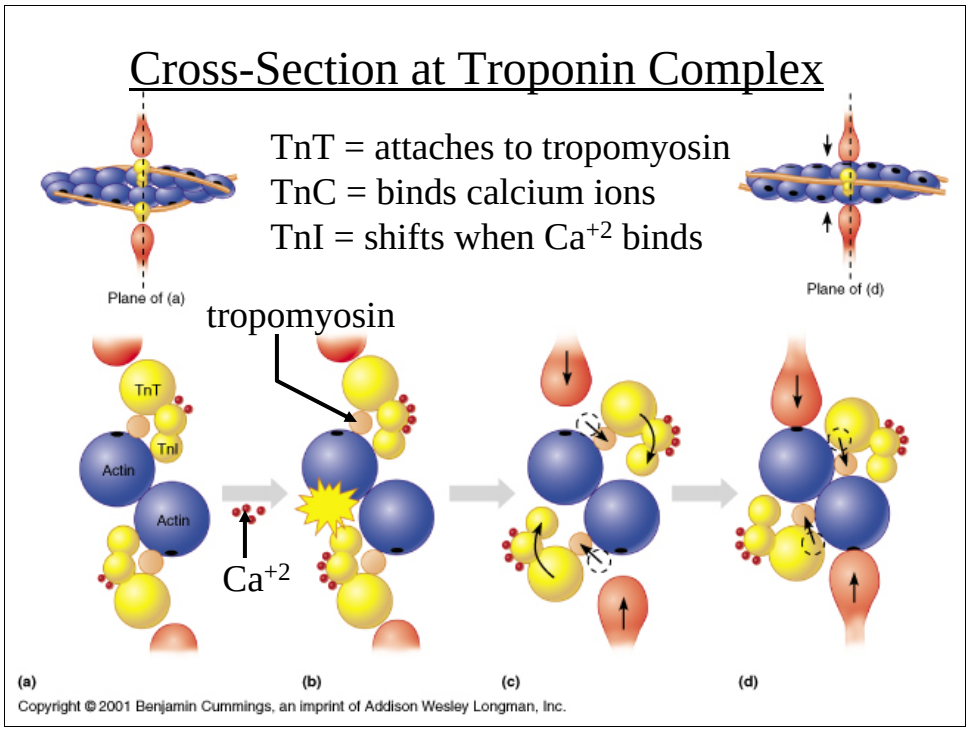
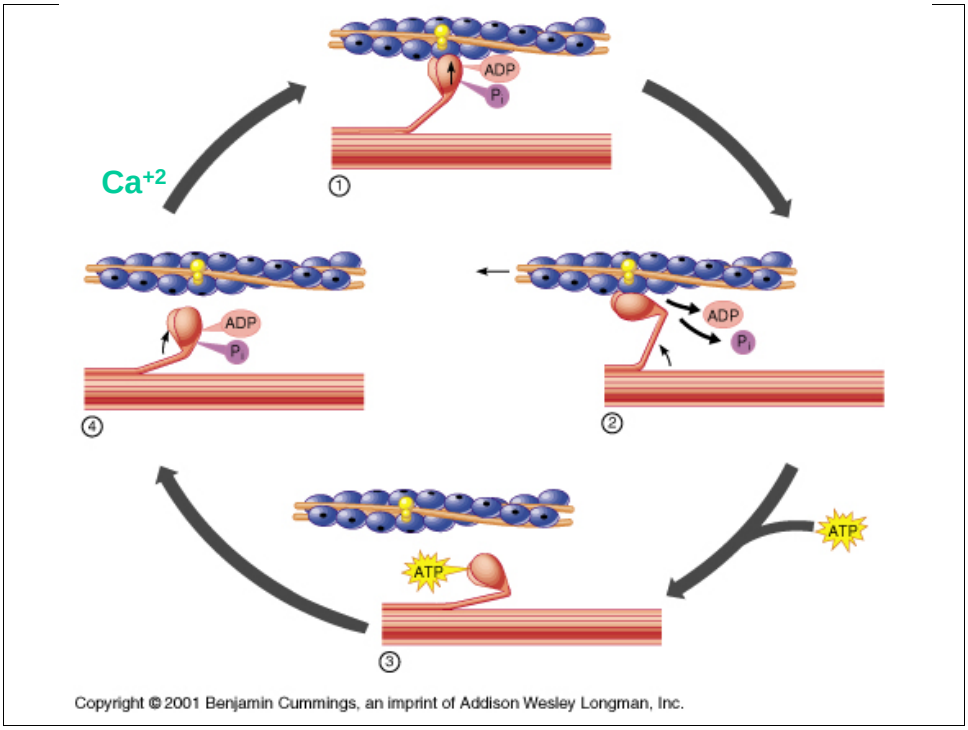
Tropomyosin molecules wrap around the fibrous actin myofilaments blocking the active sites where myosin heads could attach. **Troponin** complexes hold the tropomyosin in position, and when stimulated by the presence of Ca^{+2} ions the troponin complex moves, causing tropomyosin to shift and expose the active sites.



1) Ca^{+2} binds to TnC

2) Troponin comlex shifts, moving tropomyosin, and exposing the active sites on actin.

3) Crossbridges attached from myosin heads to actin molecules.



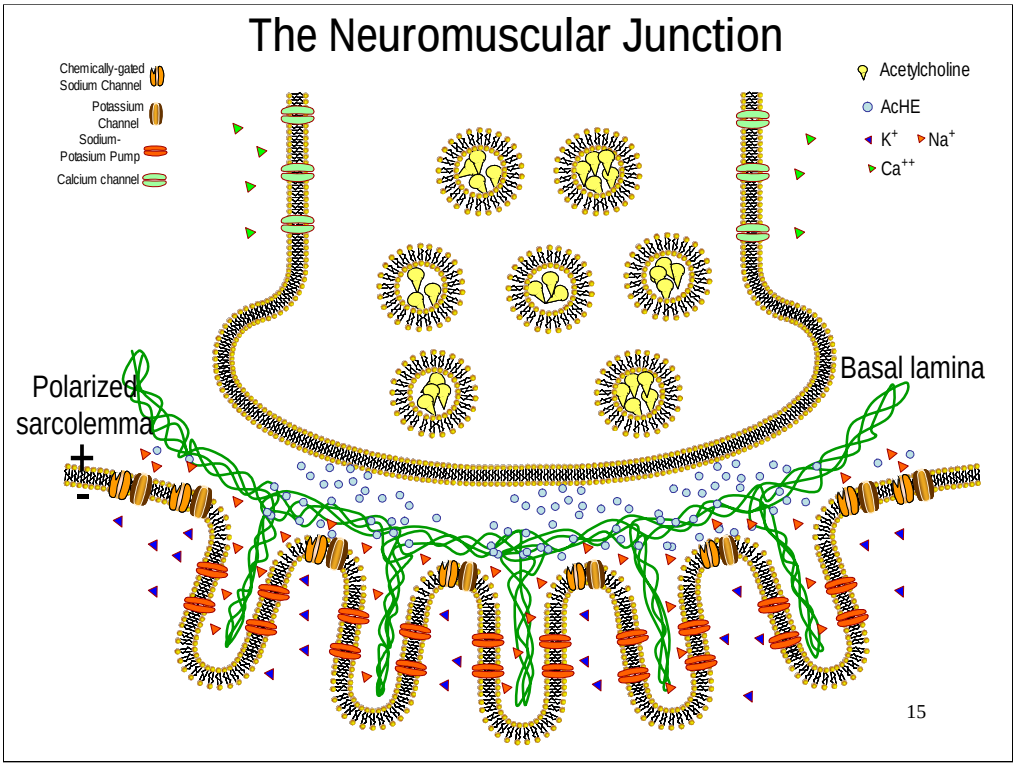
1) In response to Ca^{+2} release into the sarcoplasm, the troponin-tropomyosin complex removes its block from actin, and the myosin heads immediately bind to active sites.

2) The myosin heads then swivel, the **Working Stroke**, pulling the Z-lines closer together and shortening the sarcomeres. As this occurs the products of ATP hydrolysis, ADP and Pi, are released.

3) ATP is taken up by the myosin heads as the crossbridges detach. If ATP is unavailable at this point the crossbridges cannot detach and release. Such a condition occurs in rigor mortis, the tensing seen in muscles after death, and in extreme forms of contracture in which muscle metabolism can no longer provide ATP.

4) ATP is hydrolyzed and the energy transferred to the myosin heads as they cock and reset for the next stimulus.

Neuromuscular Junction Flash Video



Na+ is pumped out, K+ in at 3:2 producing unequal distribution which leads to a polarized membrane, ~ -65mv.1) An impulse arrives at the neuromuscular junction causing Ca+2 to enter the axon terminus. 2) Ca+2 causes exocytosis of Ach vesicles into synaptic cleft. 3) Ach diffuses across the synapse to contact post-synaptic receptors on the sarcolemma. Ach causes Na+ to enter sarcolemma causing depolarization. 4) If depolarization is threshold a new impulse is produced on the sarcolemma by depolarization of voltage-regulated ion gates. 5) Sarcolemma repolarizes, K+ leaves the cell, pump restores distribution.6) Achase (a.k.a. ACh-E) breaks down Ach so the NMJ can function again.

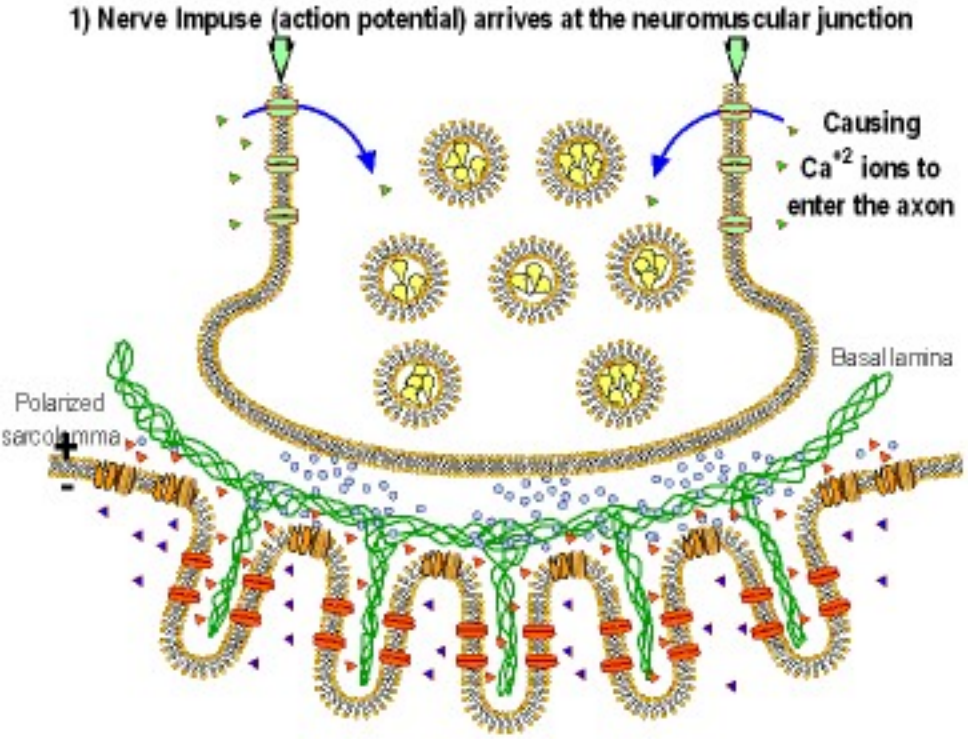
See the next slide for illustrations.

Events at The Neuromuscular Junction

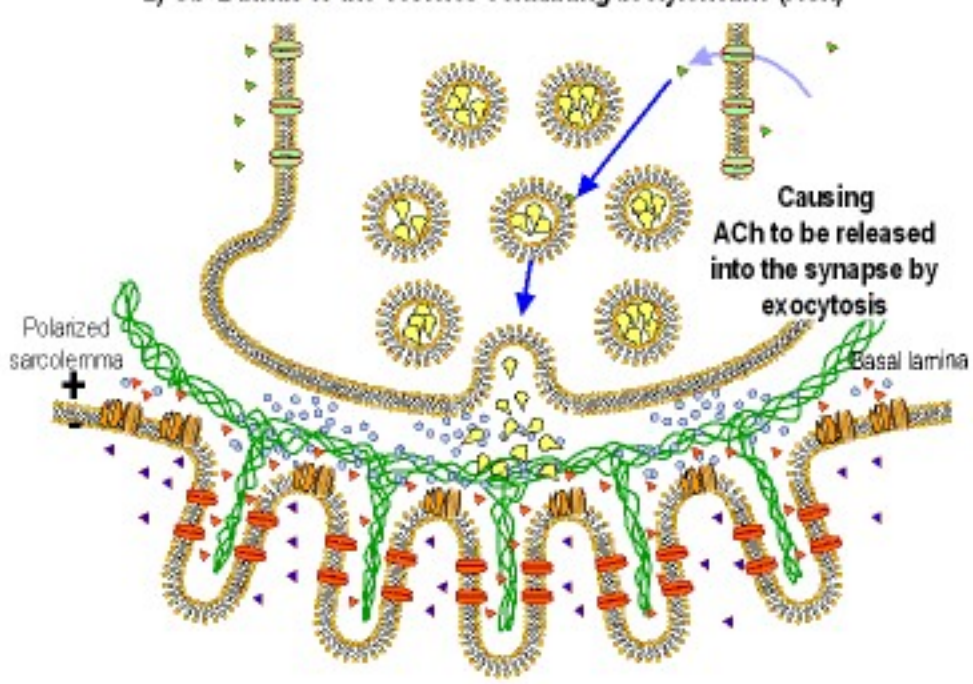
Chemically-gated Sodium Channel | Potassium Channel | Sodium-Potasium Pump | Calcium channel

Acetylcholine | AcHE | K^+ | Na^+ | Ca^{++}

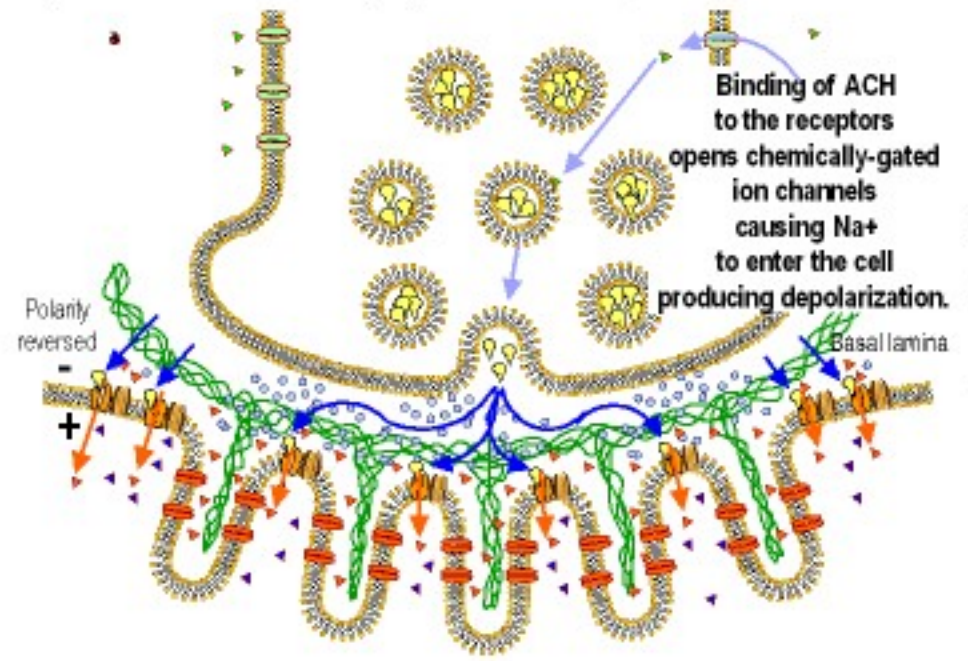
1) Nerve Impuse (action potential) arrives at the neuromuscular junction



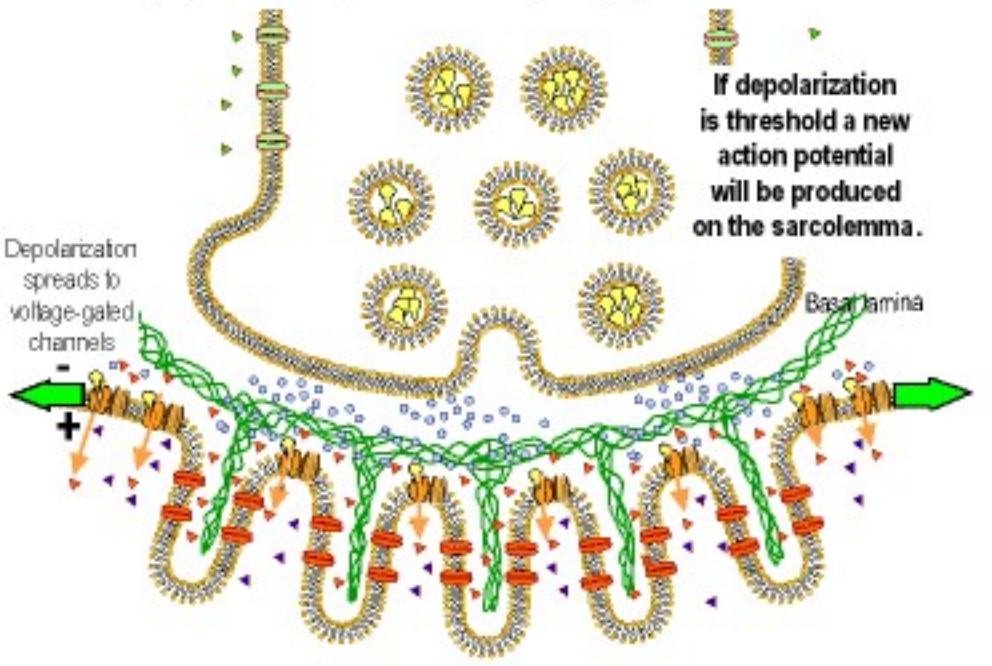
2) Ca+2 binds to the vesicles containing acetylcholine (ACh)



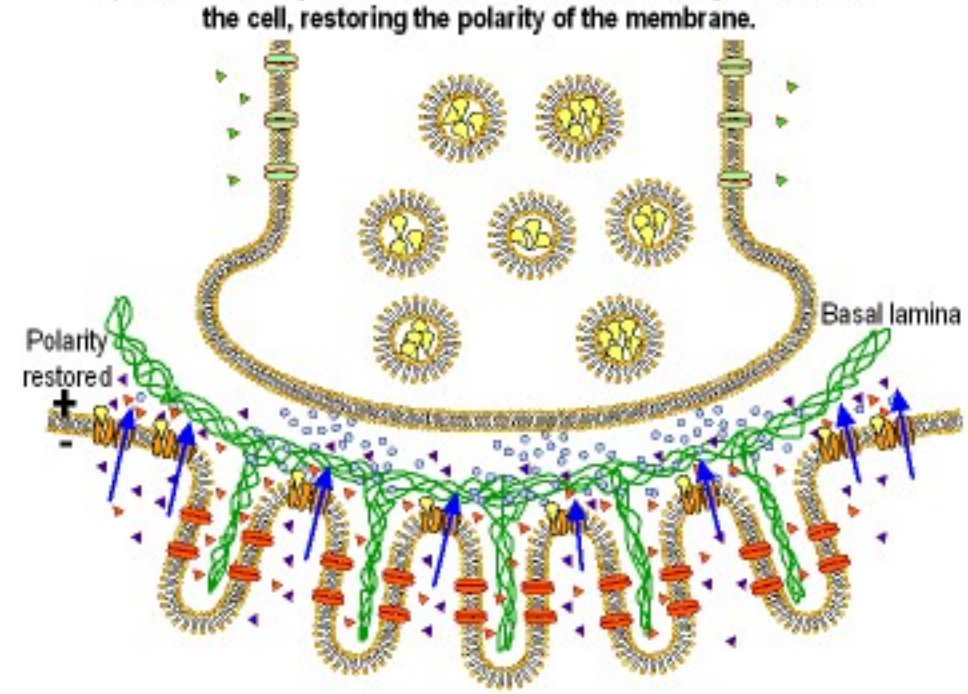
3) ACH diffuses across the synapse to bind to receptors on the sarcolemma.



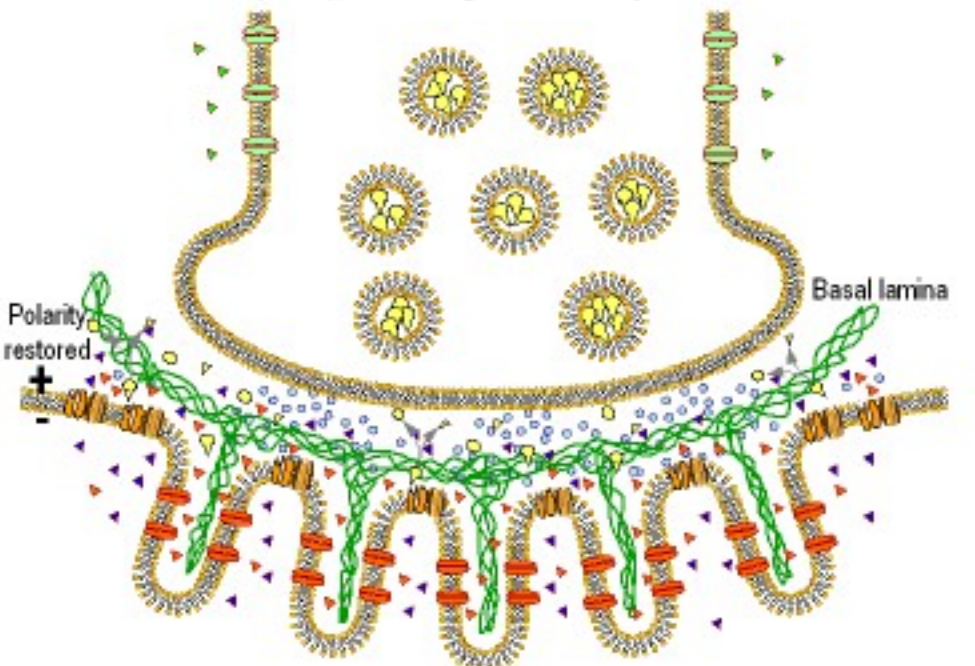
4) Depolarization spreads to nearby voltage-gated channels.



5) K+ channels open as Na+ channels close causing K+ to leave the cell, restoring the polarity of the membrane.



6) Acetylcholine is broken down by acetylcholinesterase (AChE), unblocking the ACh receptors.



Synaptic Blockers

chlinesterase inhibitors: cause paralysis by leading to blockage of receptors by ACh, e.g. insecticides and nerve gas.

The antidote to these toxins is **atropine** which blocks ACh.

Curare is an ACh competitor derived from plants, which has been used to relax muscles.

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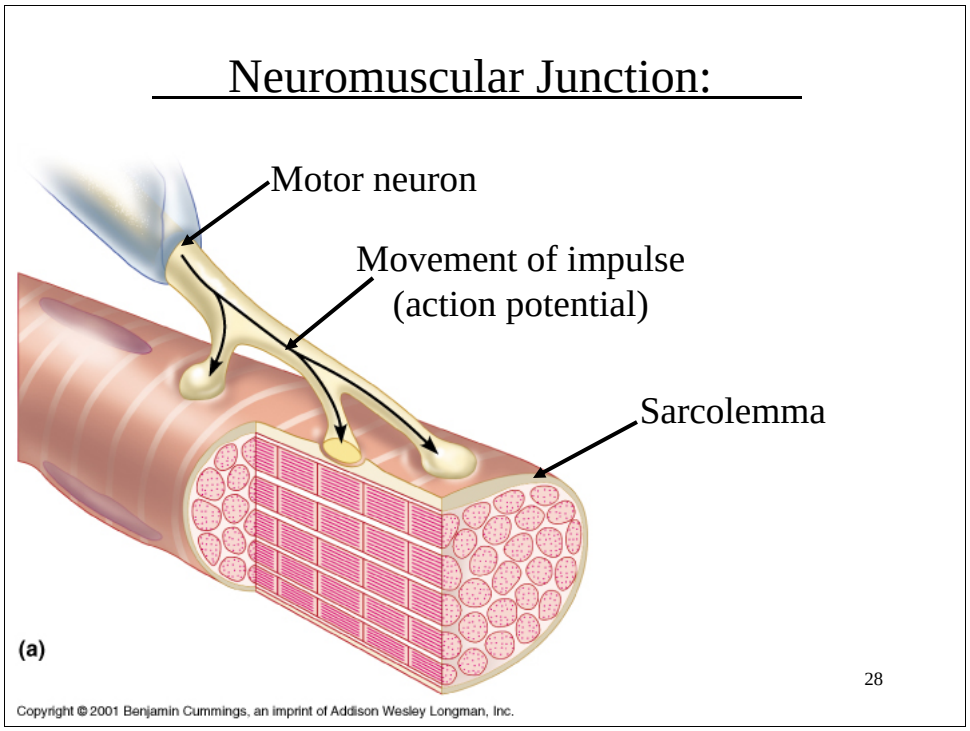
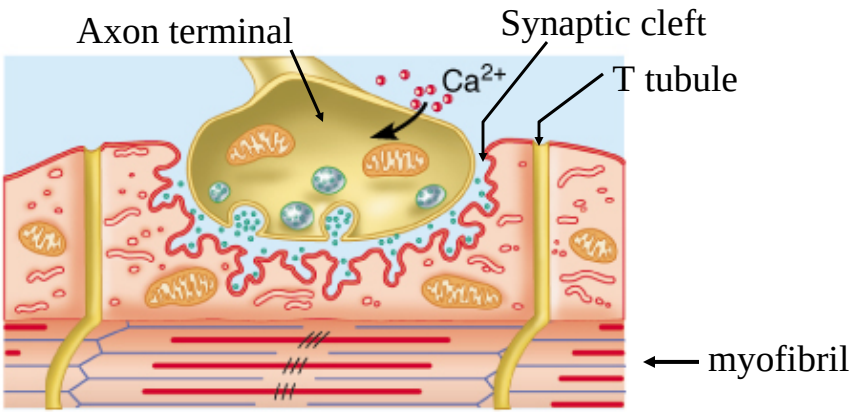


Each skeletal muscle cell is stimulated by branches from a neuron's axon.

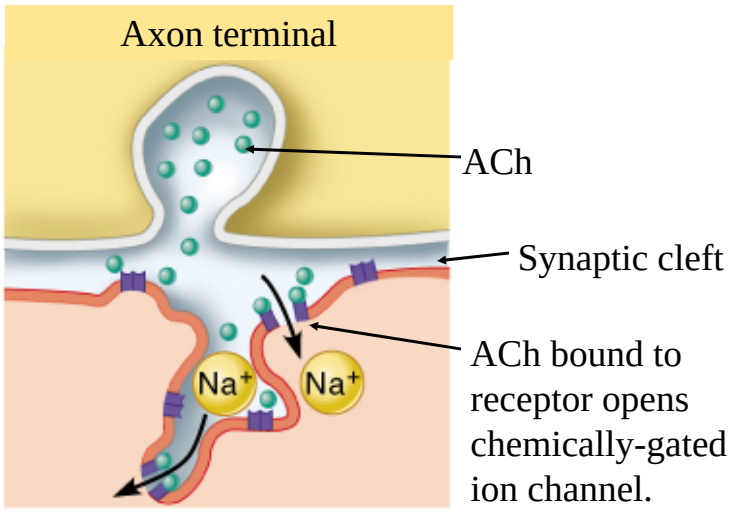
The Axon Terminal at the Motor End Plate



(b)

At each axon terminal is a **motor end plate** at which the **neuromuscular junction** occurs.

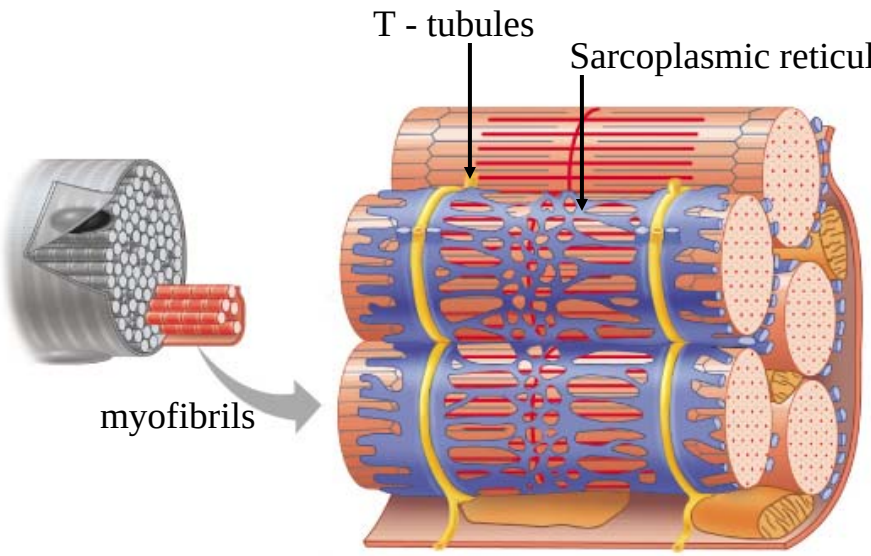
The Motor End Plate



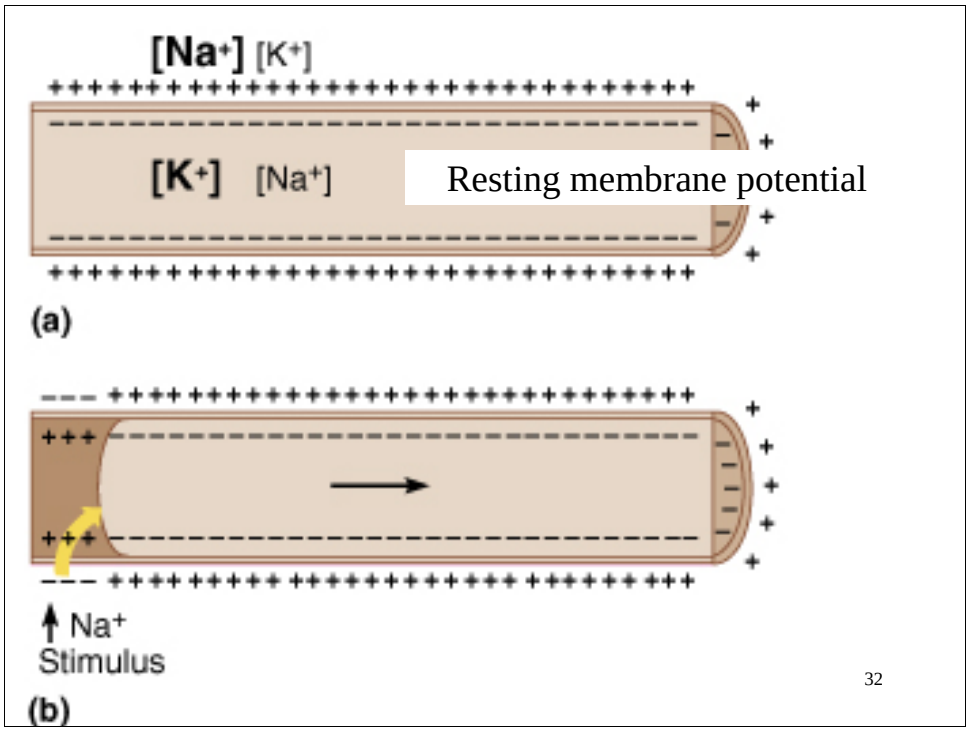
(c)

The ACh receptor binds to a **chemically-gated ion channel** which opens due to contact with ACh.

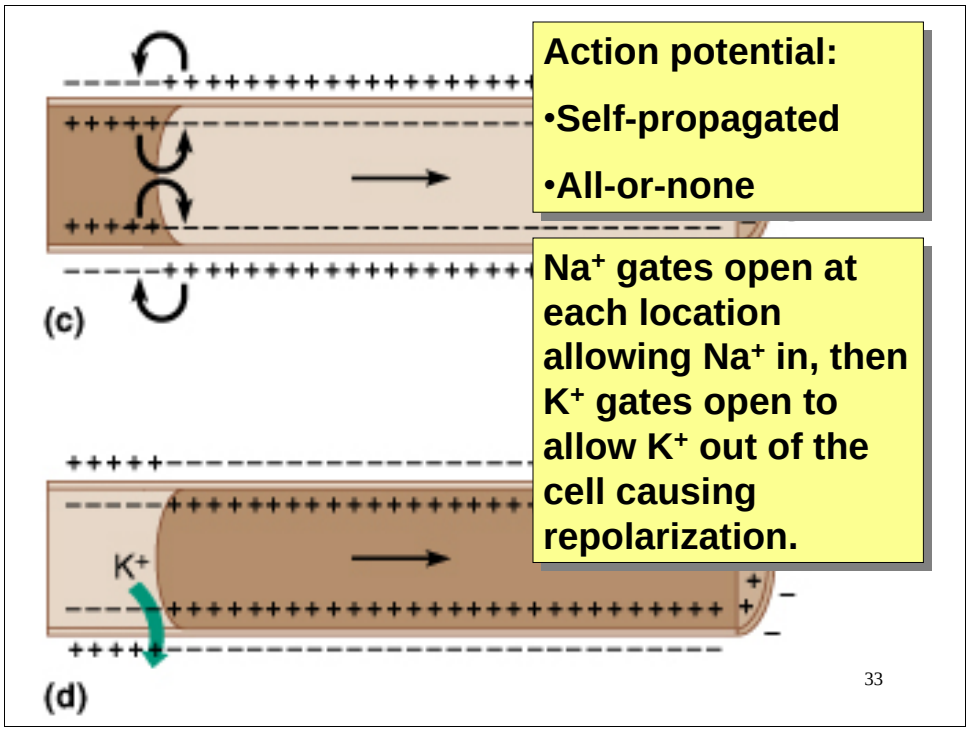
Skeletal Muscle Impulse Conduction



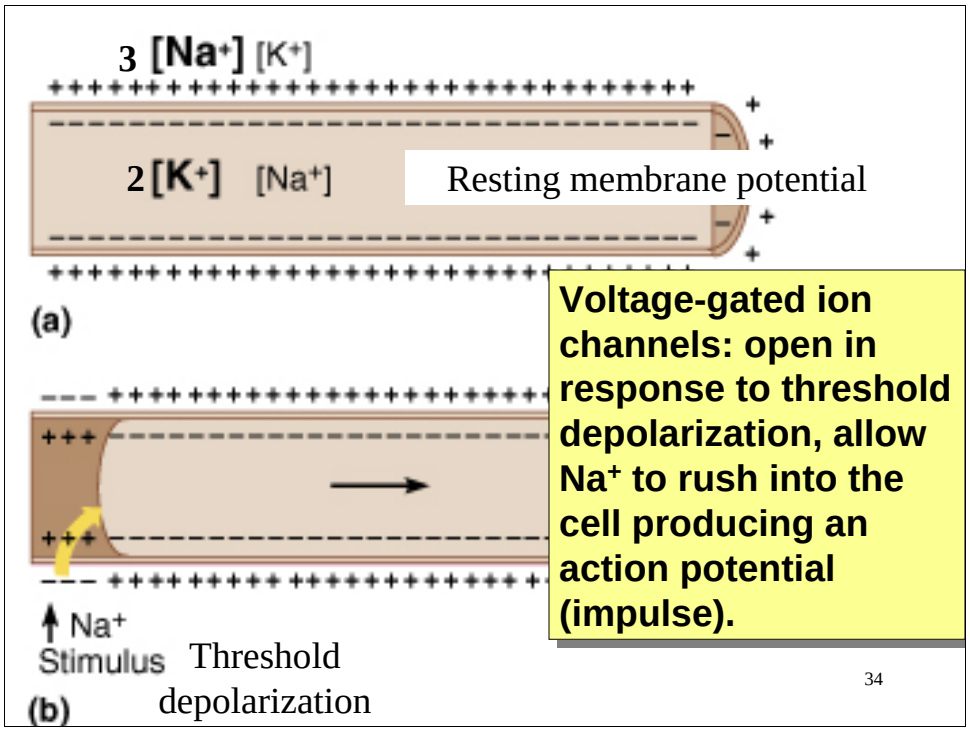
The **T-tubules** and **sarcoplasmic reticulum** represent membranes which penetrate the sarcoplasm of the cell, taking the action potential to the inside.



An action potential is produced when **voltage-gated ion channels** open in response to threshold depolarization, causing Na^+ ions to rush into the cell.

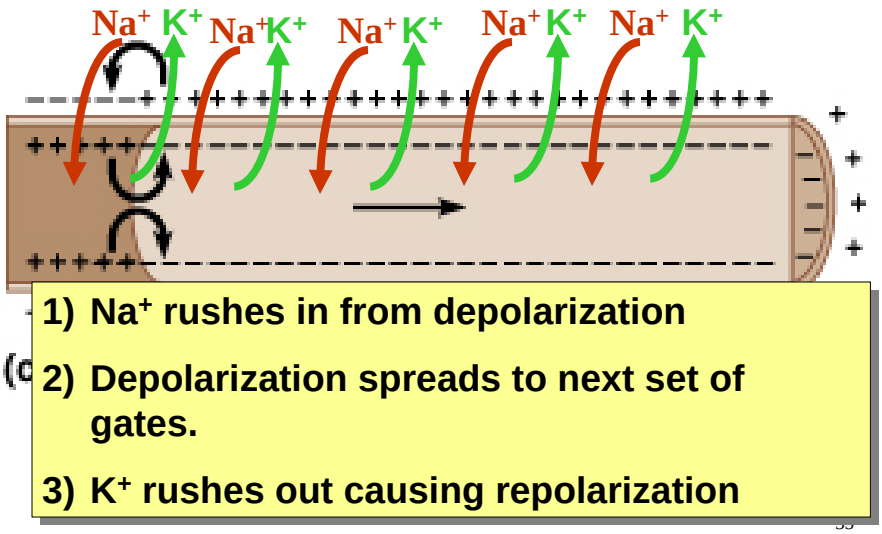


The action potential is a **self-propagated, all-or-none movement of depolarization along the membrane**. All-or-none means that there are not different size action potentials. You either have one or you don't. As the action potential passes along the sarcolemma it causes release of Na^+ into the cell by voltage-regulated ion gates, just as at the chemically-regulated gates when stimulated by ACH. Then K^+ gates open to repolarize that section of the membrane. The opening of Na^+ gates then K^+ gates happens at each location along the sarcolemma to propagate the action potential.



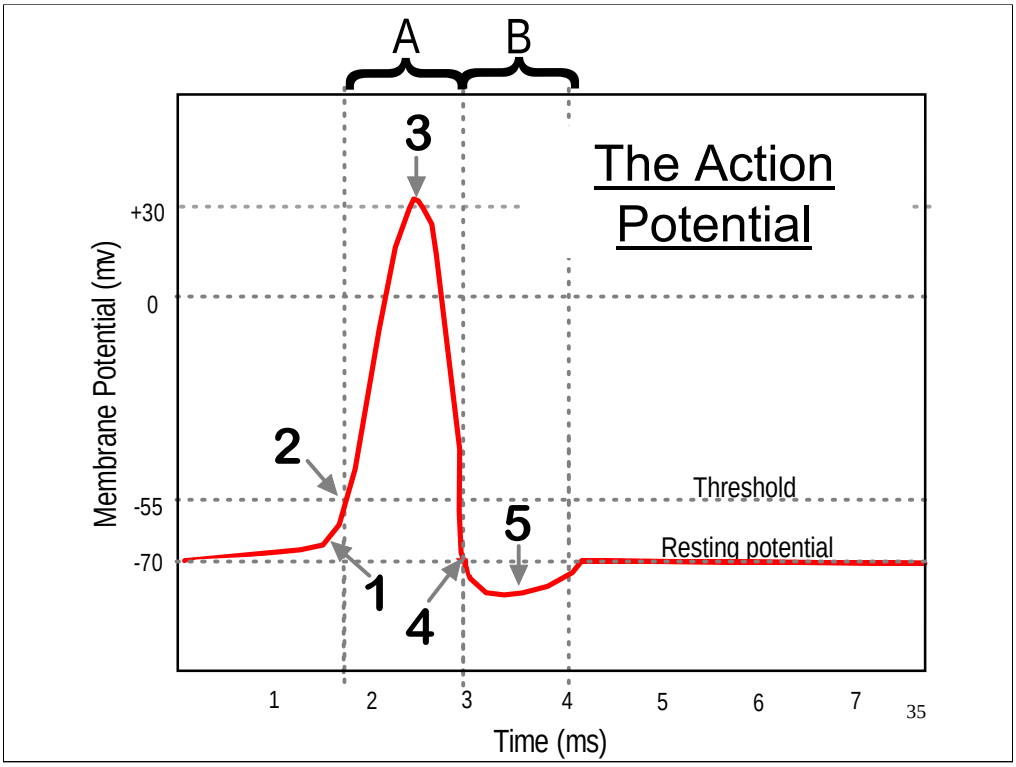
The resting membrane potential occurs because of the unequal distribution of sodium and potassium ions (a). An action potential is produced when **voltage-gated ion channels** open in response to threshold depolarization, causing Na^+ ions to rush into the cell (b).

Propagation of the Action Potential



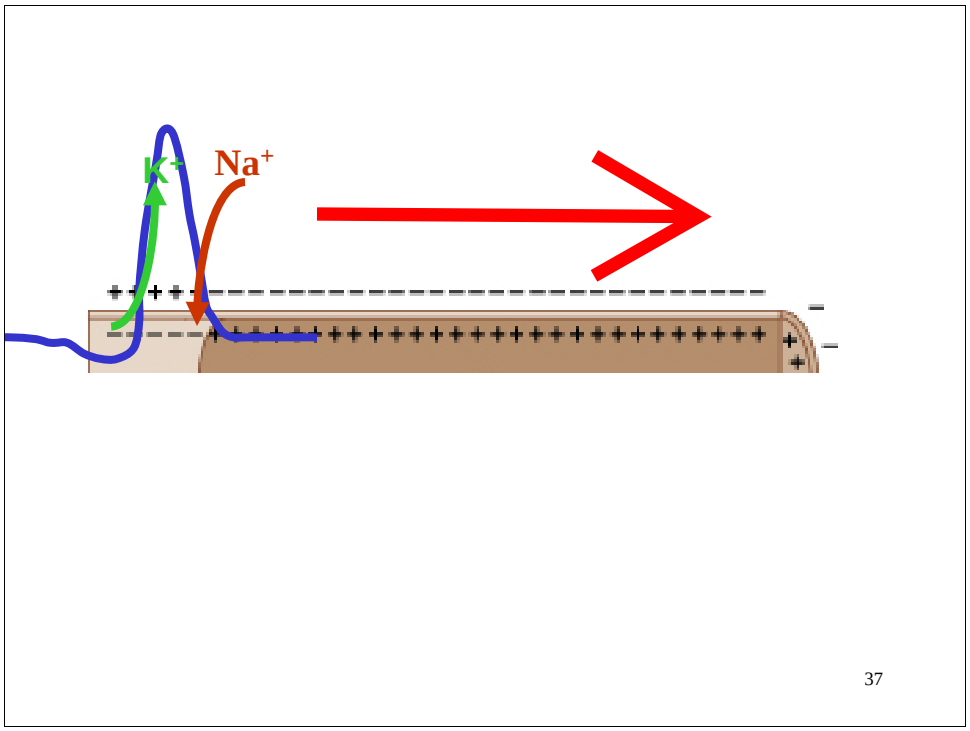
As the action potential passes along the sarcolemma it causes release of Na^+ into the cell by voltage-regulated ion gates, just as at the chemically-regulated gates when stimulated by ACH. This depolarization spreads to the next set of gates causing them to open, and so forth all along the membrane. K^+ gates open as the Na^+ gates close to repolarize each section of the membrane. The opening of Na^+ gates then K^+ gates happens at each location along the sarcolemma to propagate the action potential.

Action Potential Flash Video



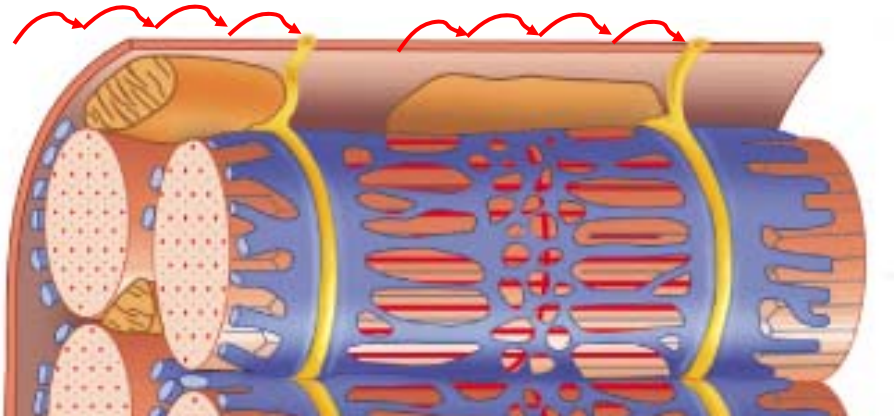
The action potential is a self-propagated, all-or-none movement of depolarization along the membrane.All-or-none means that there are not different size action potentials. You either have one or you don't.

1) Depolarization speads from previous ion channel.
2) When this depolarization reaches threshold, the Na+ channel opens, allowing Na+ ions into the cell and causing a reversal of potential.
3) As the Na+ channels close, K+ channels open allowing K+ ions to leave the cell causing repolarization.
4) Resting potential is reached, but so much K+ leaves the cell that there is a brief hyperpolarization
5) The hyperpolarization ends as K+ channels close.



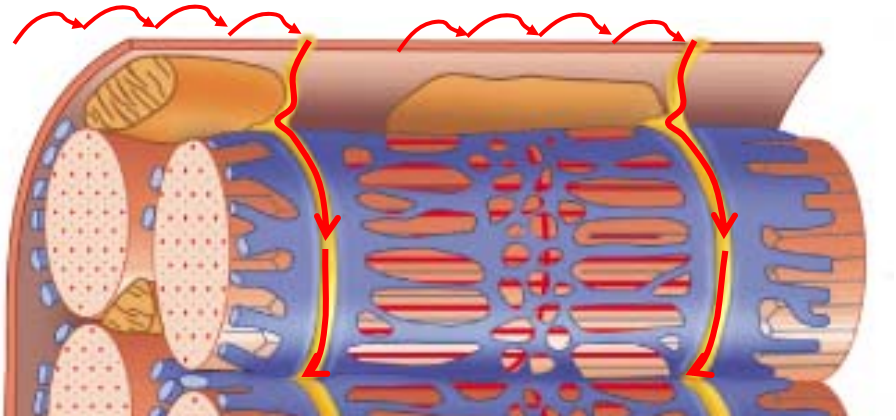
The sequential opening of first sodium, then potassium, ion channels produces depolarization which spreads along the membrane as an action potential.

• Impulse travels along the sarcolemma.



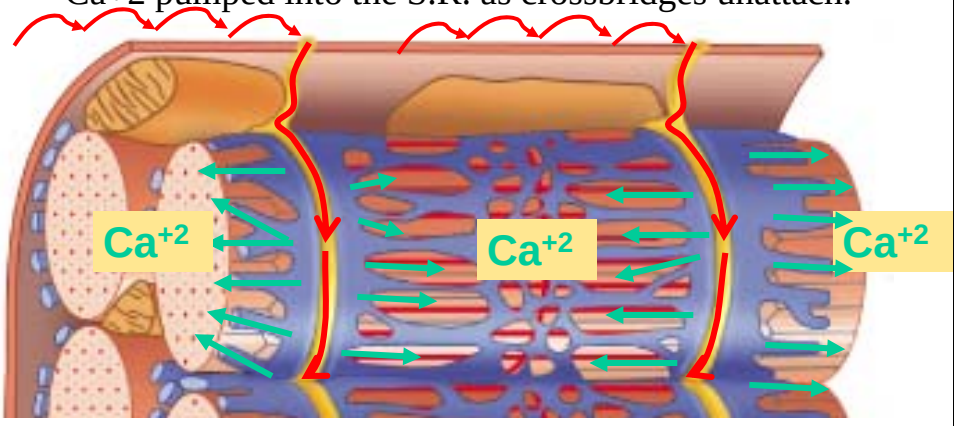
As the action potential passes along the sarcolemma it enters the **T-tubules** which occur at each Z-line. (See Figure 9.4) The T-tubules are membranes which run across the cell (T for *transverse*) connecting to the sarcolemma. The T-tubules allow the action potential to continue into the cell interior.

- Impulse travels along the sarcolemma,
- enters the T-tubules.

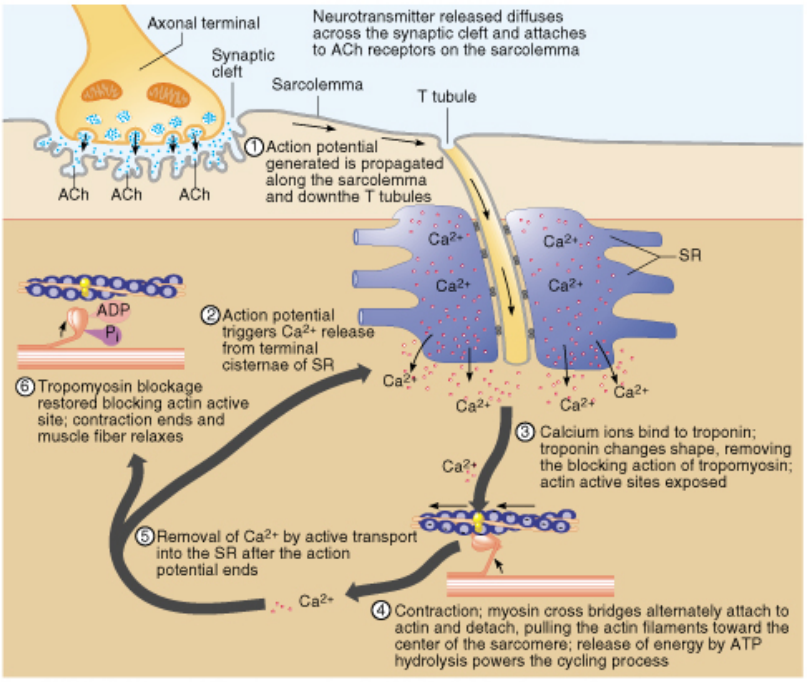


At points along the T tubules they attach to the **sarcoplasmic reticulum**, a system of membrane channels inside the sarcoplasm.

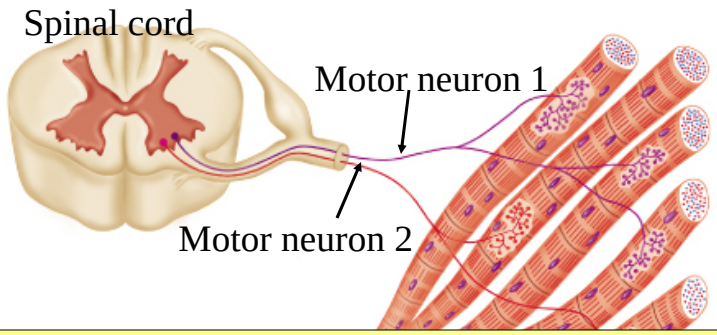
- Impulse travels along the sarcolemma,
- enters the T-tubules,
- and triggers the release of Ca^{+2} from the S.R.
- Ca+2 pumped into the S.R. as crossbridges unattach.



When the action potential moves along the T tubules it causes the sarcoplasmic reticulum to release Ca^{+2} which is sequestered by the SR. The SR pumps calcium like the sarcolemma pumps sodium and releases it into the sarcoplasm when stimulated by the action potential. This causes the sliding of filaments as outlined earlier. The SR then pumps the Ca^{+2} back out of the sarcoplasm.



Here is shown **Excitation-Contraction Coupling** from the beginning to end, including each of the components examined previously.

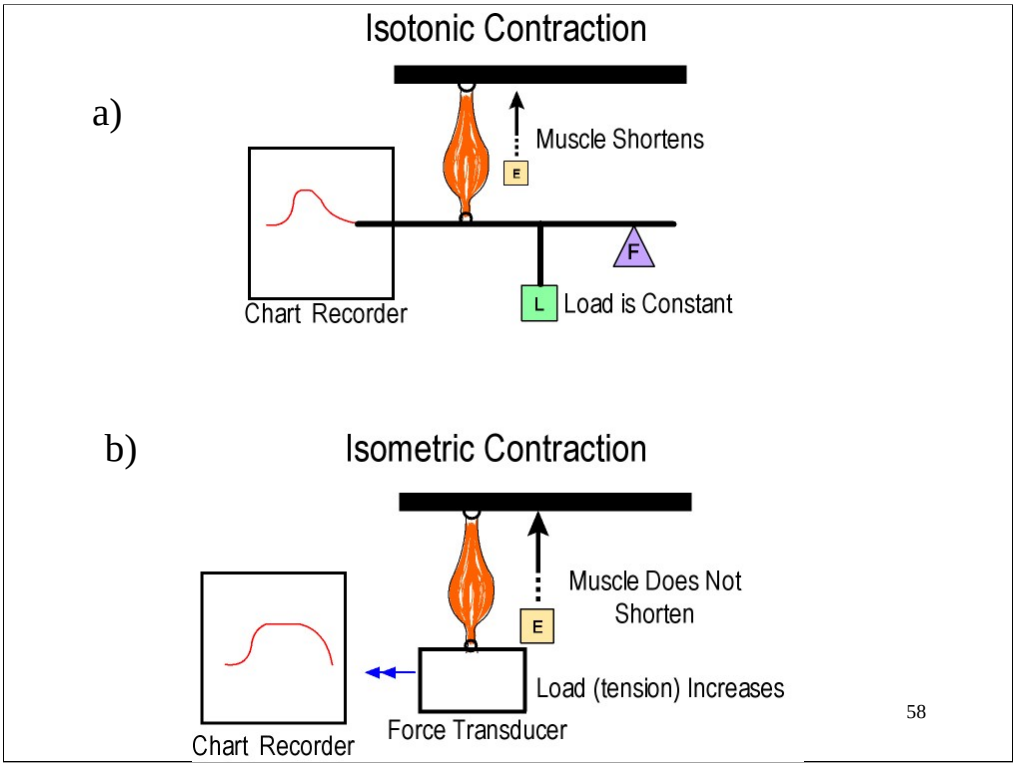


Motor unit = a group of muscle cells stimulated by the same neuron.

< 10 cells per motor unit = very precise control e.g. extraocular muscles

10 to 100 cells per unit = most muscles, average control

>100 cells per unit = gross movements, e.g. postural muscles



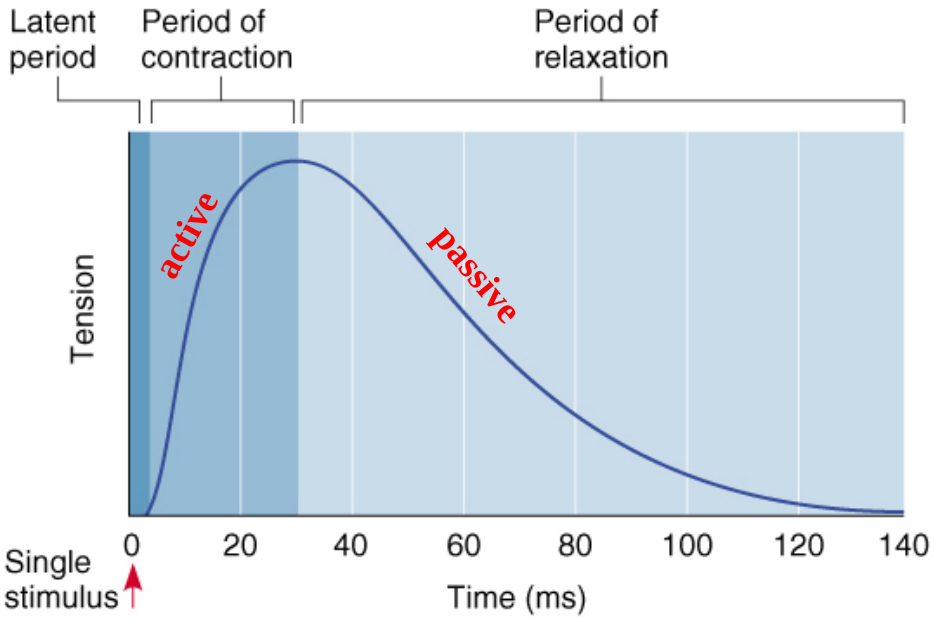
There are two ways in which a muscle can contract: isotonically and isometrically.

Isotonic contraction involves a shortening of the muscle as tension is generated, exhibited by diagram a) above, in which the muscle is moving a load. The tension remains constant as long as the load doesn't change. Isotonic contractions are the majority of our muscular movements.

In b) above, the muscle cannot shorten but may increase its tension continuously against the force transducer. Isometric contractions are seen when a muscle generates tension against an immovable object.

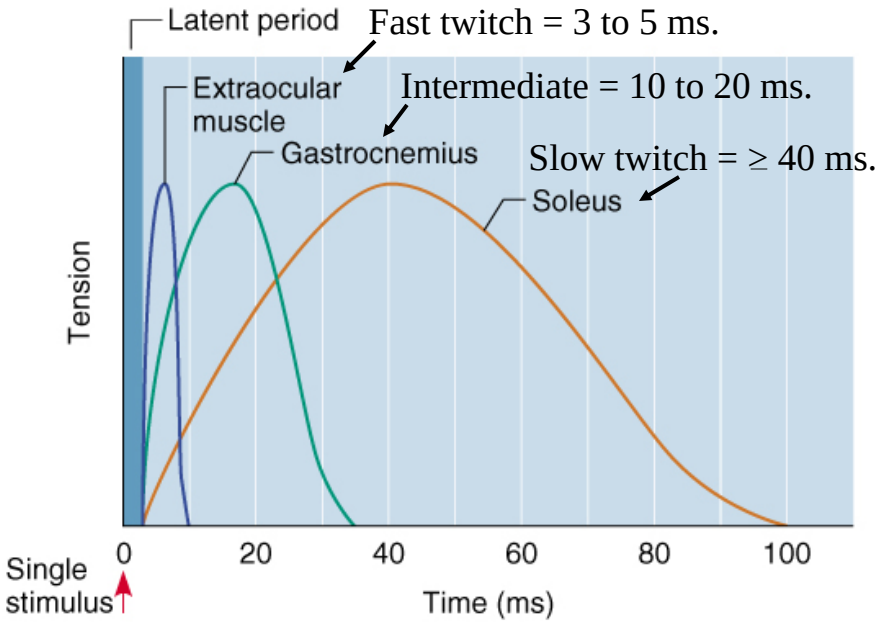
Physiologically the contractions are identical. The heat produced by isotonic contractions is greater due to the action of movement of the internal myofilaments, but not significantly. Isotonic contractions provide a more complete workout of both the muscle and supportive systems such as the cardiovascular and respiratory. Isometric contractions can be effective in building muscle tone and size.

We do not use the muscle twitch as part of our normal muscle responses. Instead we use graded contractions, contractions of whole muscles which can vary in terms of their strength and degree of contraction. In fact, even relaxed muscles are constantly being stimulated to produce muscle tone, the minimal graded contraction possible. The above illustration shows how a muscle from a frog is used to produce graded contractions experimentally.



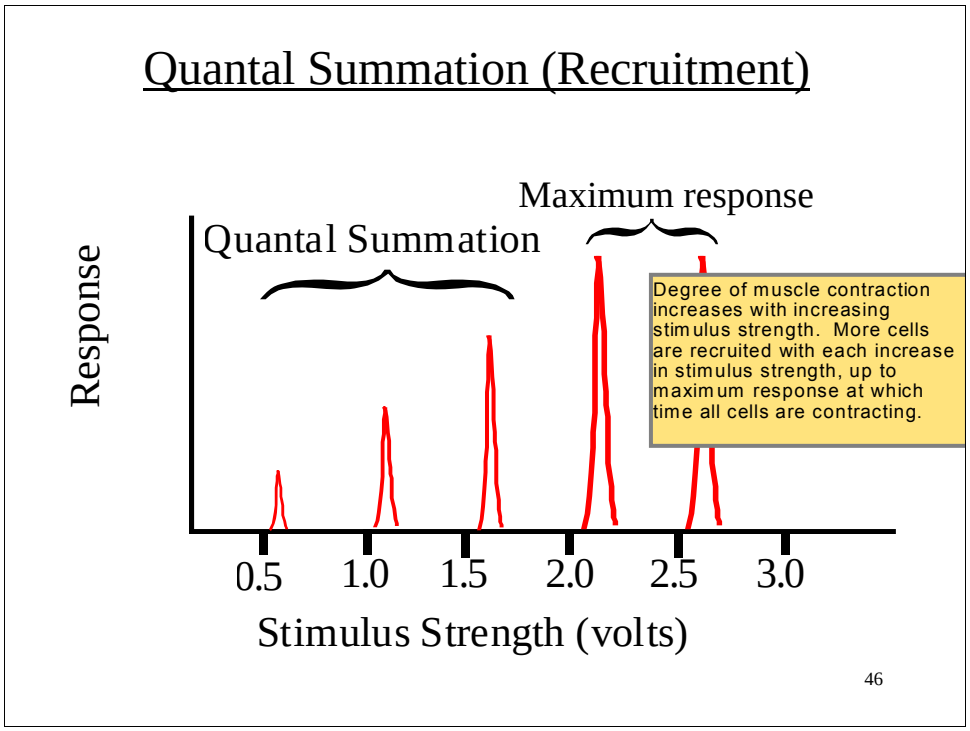
The Muscle Twitch Figure 9.12

The **muscle twitch** is a single response to a single stimulus. In a diagram of the muscle twitch can be seen the **latent period**, the period of a few ms encompassing the chemical and physical events preceding actual contraction. This is **not the same** as the **absolute refractory period**, the even briefer period when the sarcolemma is depolarized and cannot be stimulated. The **relative refractory period** occurs after this when the sarcolemma is briefly hyperpolarized and requires a greater than normal stimulus. [See Refractory Periods Diagram in Slide #34] Following the latent period is the contraction phase in which the shortening of the sarcomeres and cells occurs. Then comes the relaxation phase, a longer period because it is passive, the result of recoil due to the series elastic elements of the muscle.



Twitch Lengths

Muscle twitches vary in length according to the type of muscle cells involved. **Fast twitch** muscles such as those which move the eyeball have twitches which reach maximum contraction in 3 to 5 ms (milliseconds). See [superior eye] and [lateral eye] These muscles were mentioned earlier as also having small numbers of cells in their motor units for precise control. The cells in **slow** twitch muscles like the postural muscles (e.g. back muscles, soleus) have twitches which reach maximum tension in 40 ms or so. The muscles which exhibit most of our body movements have **intermediate** twitch lengths of 10 to 20 ms. These three types also represents different metabolic patterns as will be discussed later.



Quantal Summation or **Recruitment** - this refers to increasing the number of cells contracting. This is done experimentally by increasing the voltage used to stimulate a muscle, thus reaching the thresholds of more and more cells. In the human body quantal summation is accomplished by the nervous system, stimulating increasing numbers of cells or motor units to increase the force of contraction.

Frequency Summation

Muscle is stimulated before it relaxes from the previous stimulus.

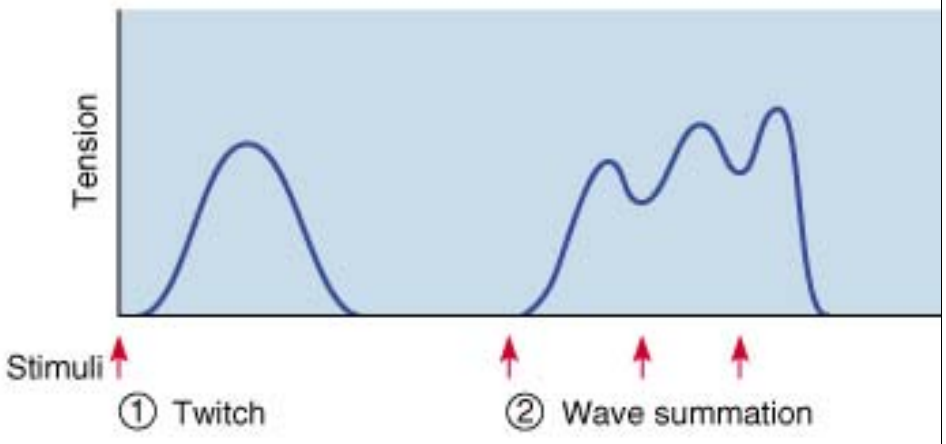


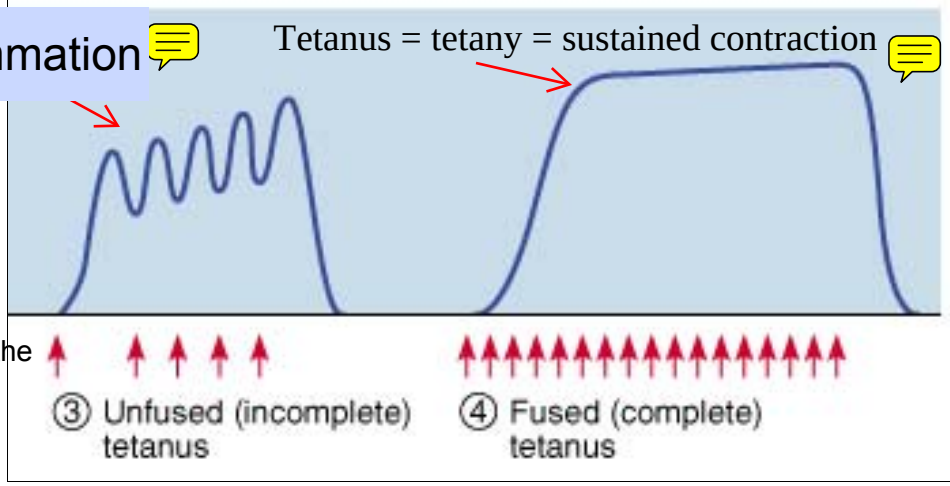
Figure 9.13

If you stimulate a muscle cell, or muscle, before it has relaxed from a previous stimulus, it will contract some more. This is called frequency summation.

Incomplete and Complete Tetanus

High frequency stimuli don't permit the muscle to relax, producing a sustained contraction.

Wave Summation



Wave Summation (a.k.a. frequency summation) and **Tetanization**- this results from stimulating a muscle cell before it has relaxed from a previous stimulus. This is possible because the contraction and relaxation phases are much longer than the refractory period. This causes the contractions to build on one another producing a wave pattern or, if the stimuli are high frequency, a sustained contraction called **tetany** or **tetanus**. (The term *tetanus* is also used for an illness caused by a bacterial toxin which causes contracture of the skeletal muscles.) This form of tetanus is perfectly normal and in fact is the way you maintain a sustained contraction.

Two Types of Summation

Quantal summation – the number of cells or motor units varies to produce contractions of different strengths.

a.k.a. recruitment

Frequency summation – a given set of cells or motor units increases the degree of contraction to produce a sustained contraction.

Wave summation or incomplete tetanus

Complete tetanus.

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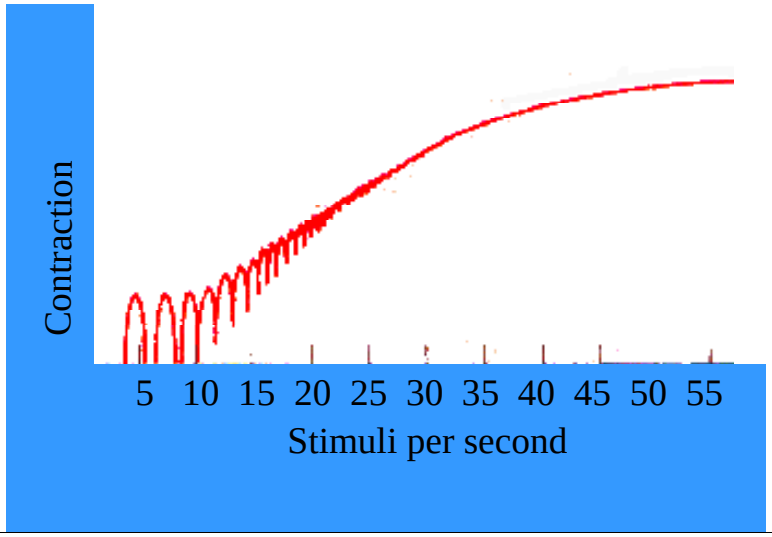
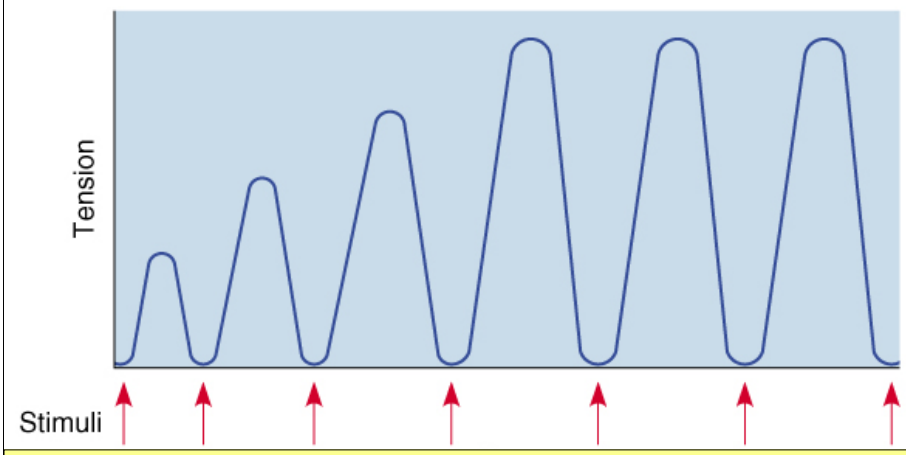
Example of Tetanus



Treppe: Warmup

Figure 9.14



muscle cells initially stimulated when cold will exhibit gradually increasing responses until they have warmed up.

Treppe is **not a way muscles exhibit graded contractions**. It is a warmup phenomenon in which when muscle cells are initially stimulated when cold, they will exhibit gradually increasing responses until they have warmed up. The phenomenon is due to the increasing efficiency of the ion gates as they are repeatedly stimulated. Treppe can be differentiated from quantal summation because the strength of stimulus remains the same in treppe, but increases in quantal summation.

Length-Tension Relationship in a Sarcomere

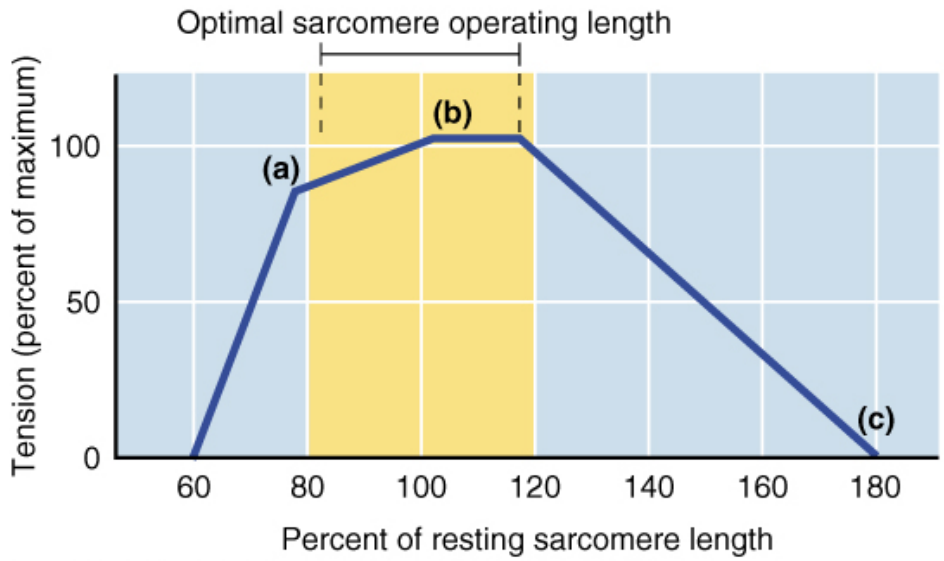
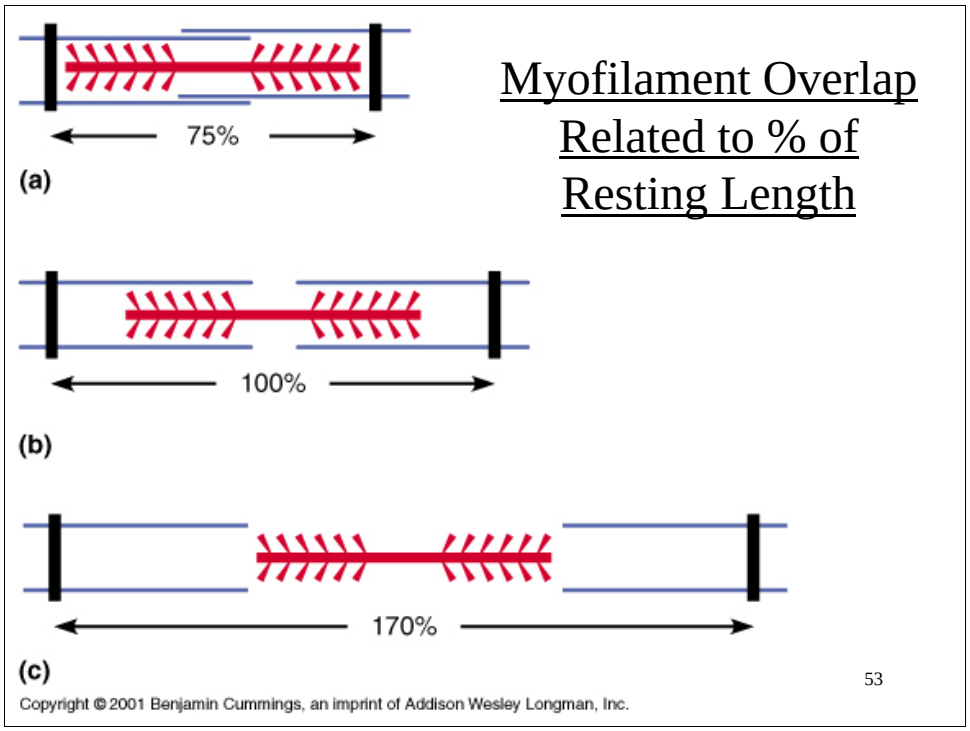
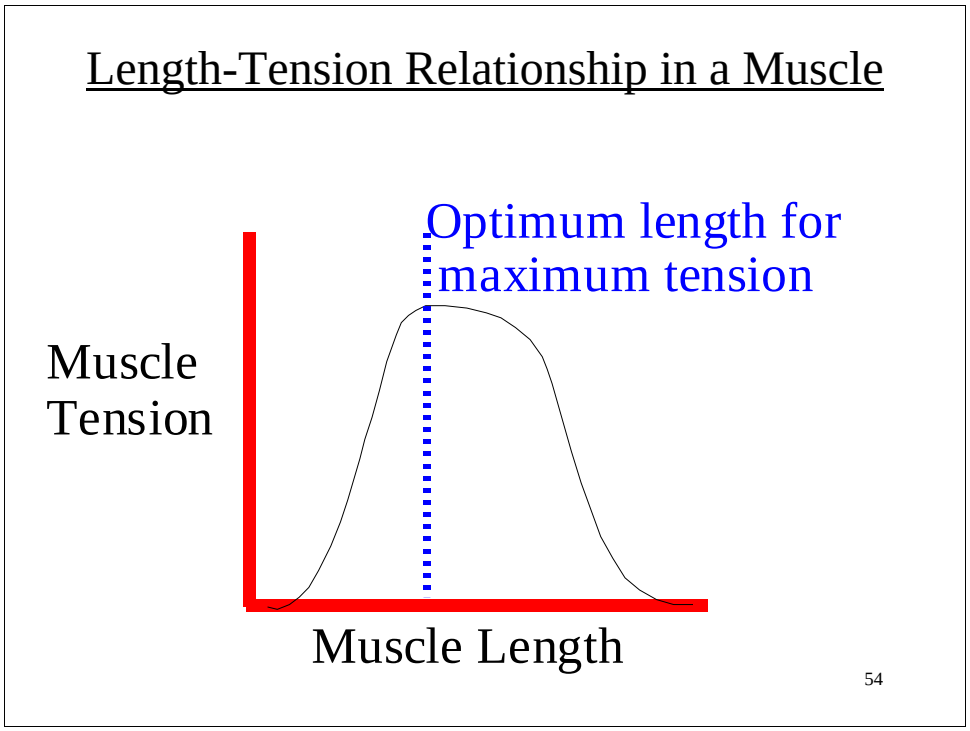


Figure 9.21

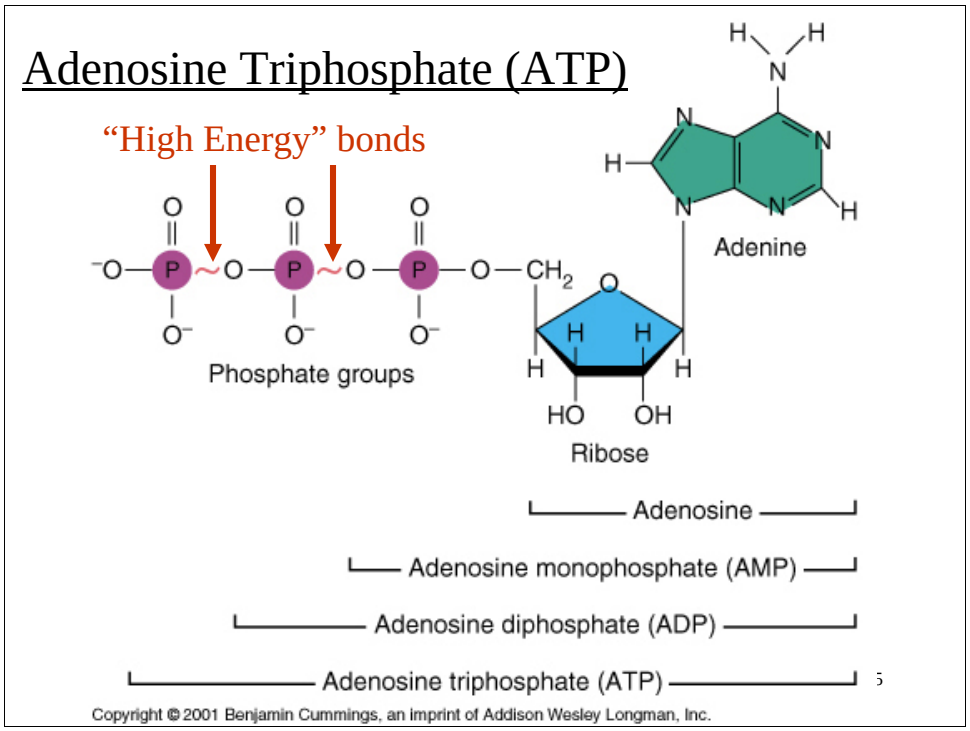
Another way in which the tension of a muscle can vary is due to the length-tension relationship. This relationship expresses the characteristic that within about 10% the resting length of the muscle, the tension the muscle exerts is maximum. At lengths above or below this optimum length the tension decreases. In practical terms a muscle will be its strongest at midpoint in its extensibility. For the heart, you will later learn, it means the muscle will adjust its output to normal increases in blood supply.



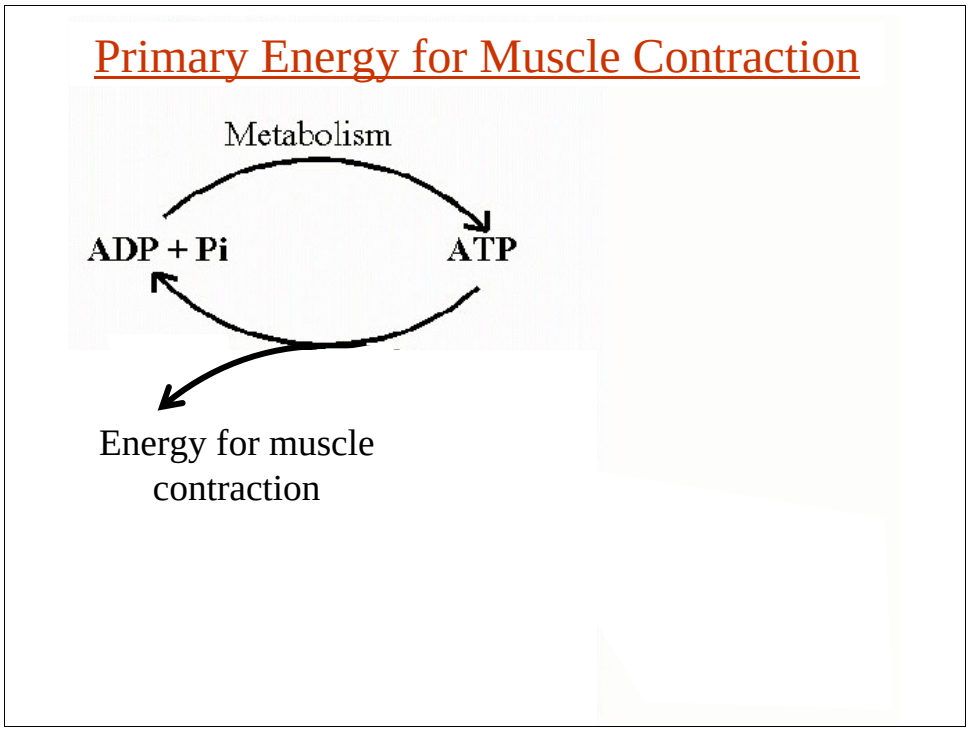
Note how the overlap of actin and myosin is maximized near the resting length, and reduced when the sarcomere is fully contracted or fully stretched. The overlap of myosin and actin produces the ability to generate tension.



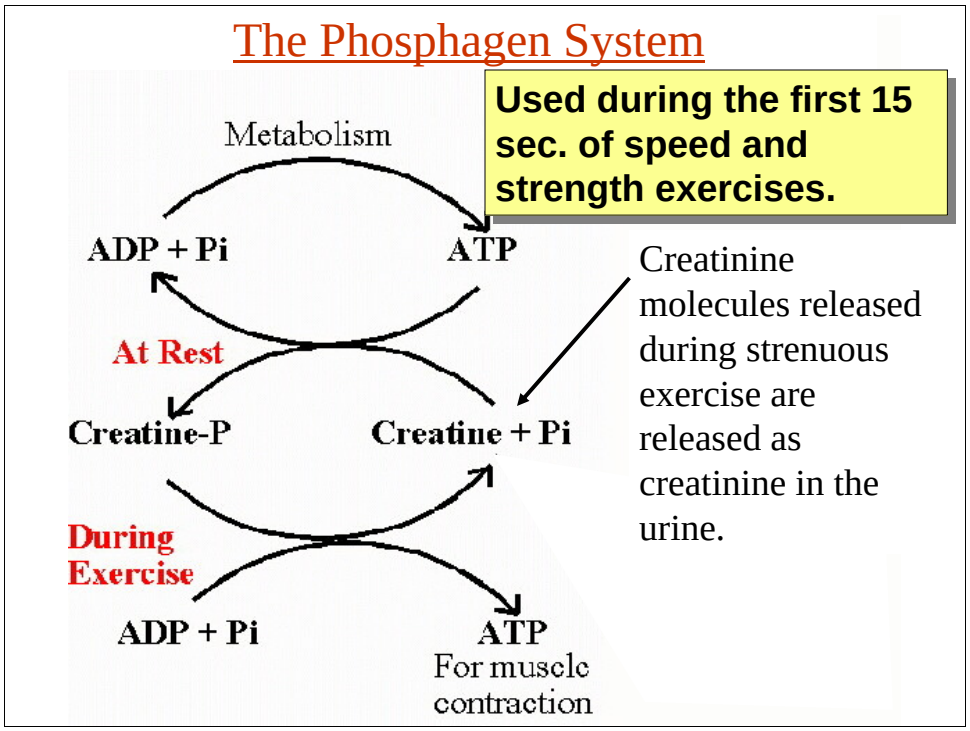
The length-tension relationship is derived from the anatomy of the sarcomere, but it can be applied to the whole muscle as well. A muscle can exhibit its greatest tension within 10% of its resting length.



Muscle cells, like all others, use ATP as their energy currency. During periods of rest ATP is built from ADP and Pi (inorganic phosphate), then during energy-requiring activities the ATP is hydrolyzed to release the energy for doing work, along with the ADP and Pi. But some muscle cells must exhibit activity levels in which they cannot make ATP as fast as it is consumed. So muscle cells have several mechanisms to provide the ATP they need.



The **phosphagen system** - this is the use of immediately available ATP. This is **not** from stored ATP itself, muscle cells can store only very limited amounts. It is from energy stored as the related molecule **CP**, **creatine phosphate**. Creatine phosphate **can** be stored and is made from ATP during periods of rest. Then during periods of high activity CP is broken down quickly and its energy converted to ATP. But this source of ATP can only supply a cell for 8 to 10 seconds during the most strenuous exercise. Creatine released during muscle activity shows up in the urine as **creatinine**, a combination of two creatine molecules.



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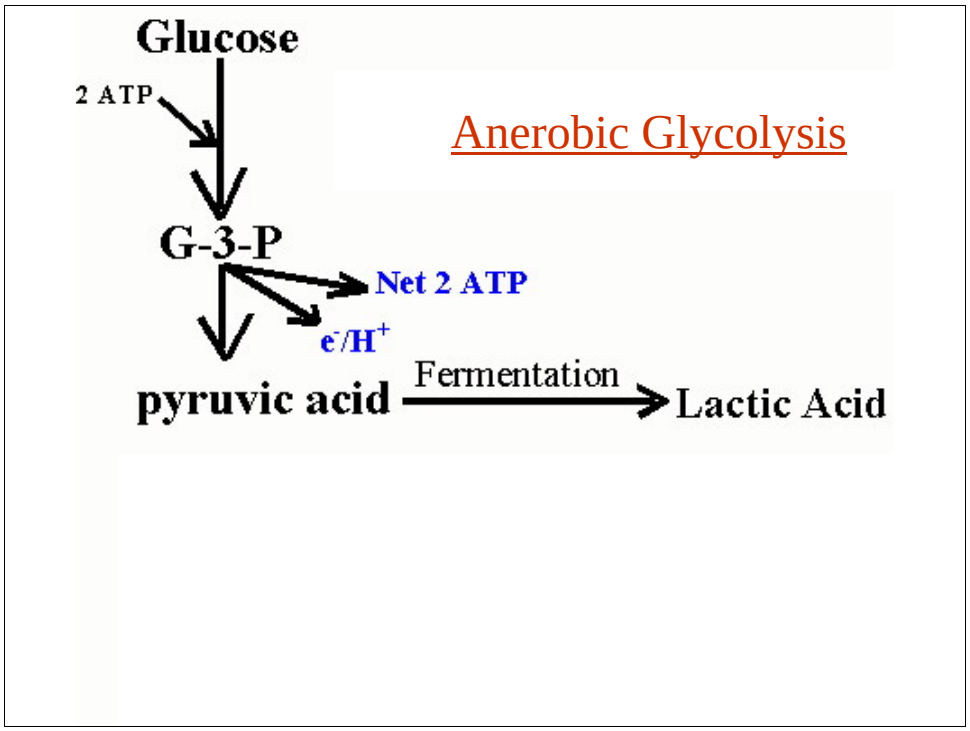
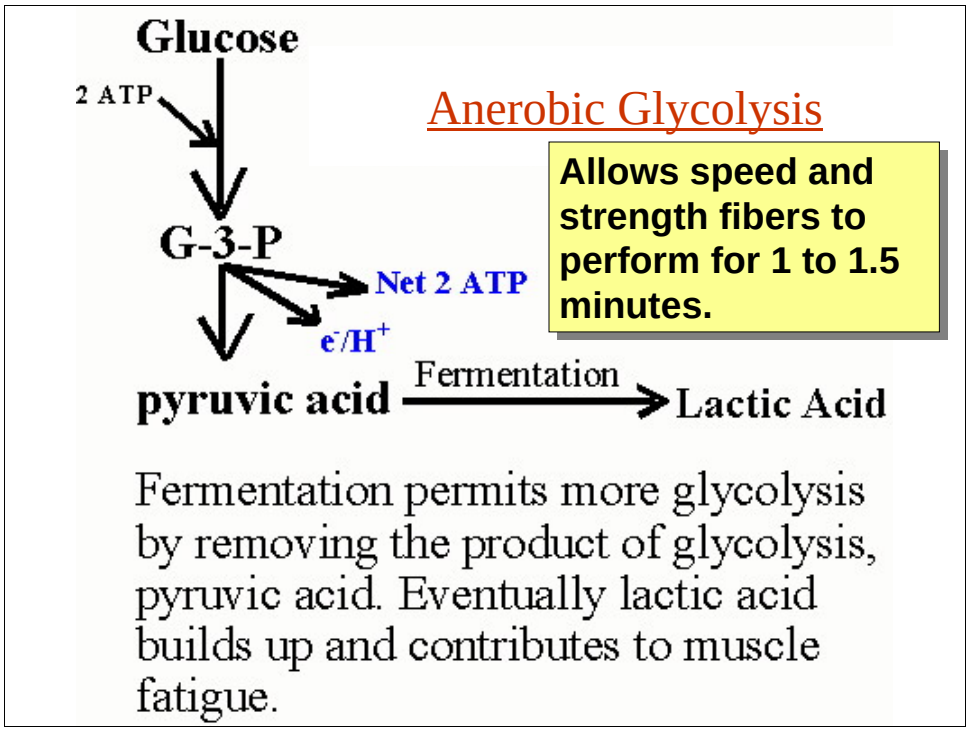
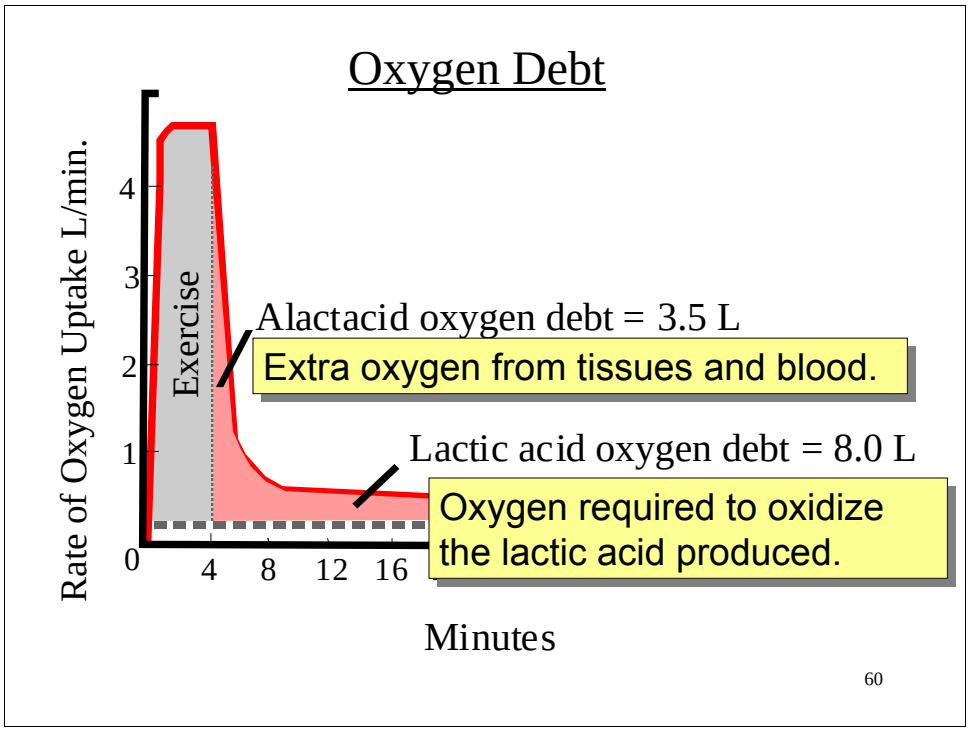


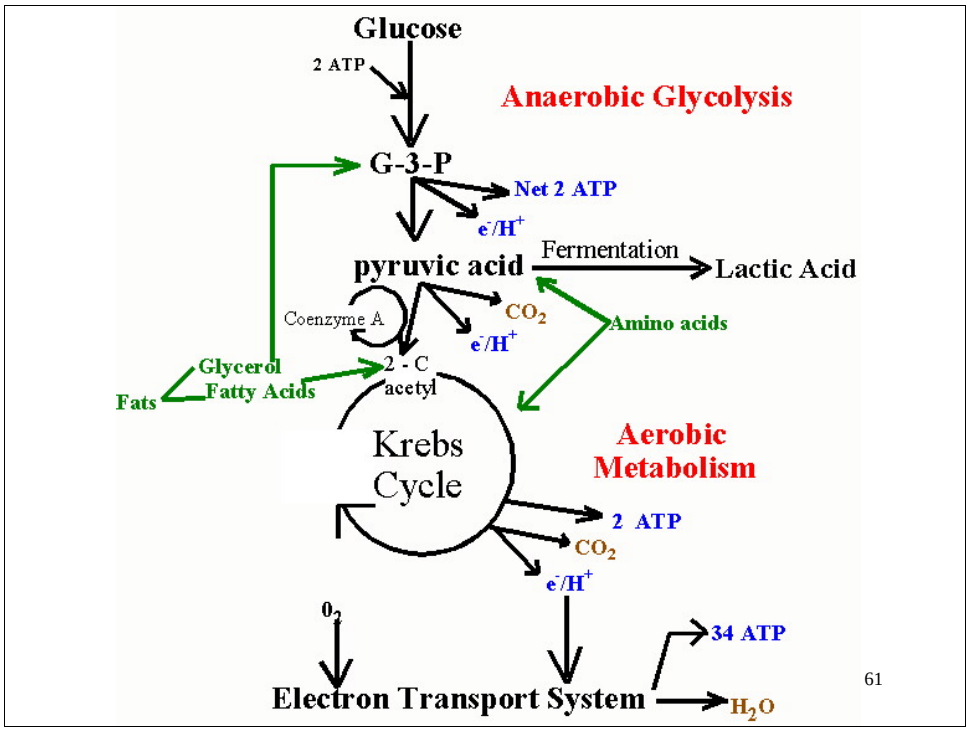
Glycolysis is the initial way of utilizing glucose in all cells, and is used exclusively by certain cells to provide ATP when insufficient oxygen is available for aerobic metabolism. Glycolysis doesn't produce much ATP in comparison to aerobic metabolism, but it has the advantage that it doesn't require oxygen. In addition, glycolysis occurs in the cytoplasm, not the mitochondria. So it is used by cells which are responsible for quick bursts of **speed** or **strength**. Like most chemical reactions, glycolysis slows down as its product, pyruvic acid, builds up. In order to extend glycolysis the pyruvic acid is converted to lactic acid in a process known as fermentation. Lactic acid itself eventually builds up, slowing metabolism and contributing to muscle fatigue.



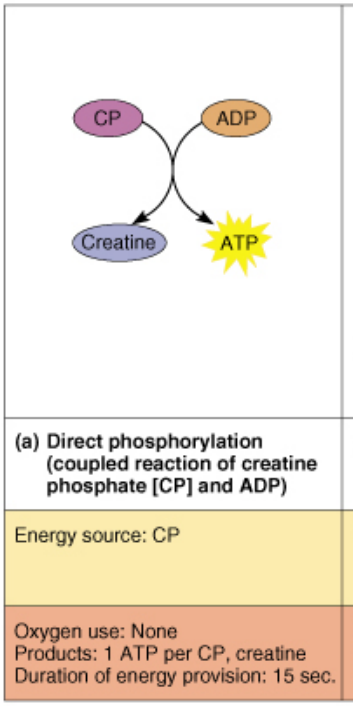
Eventually the lactic acid must be reconverted to pyruvic acid and metabolized aerobically, either in the muscle cell itself, or in the liver. The oxygen which is "borrowed" by anaerobic glycolysis is called **oxygen debt** and must be paid back.



This graph shows the rate of oxygen uptake by the lungs during maximal exercise for 4 minutes and then for one hour after the exercise is over. This figure demonstrates the principle of oxygen debt. Oxygen debt is partly oxygen reserves in the lungs, tissues, and myoglobin in the lungs (**alactacid oxygen debt**). But mostly it is the amount of oxygen which will be required to metabolize the lactic acid produced, the **lactic acid oxygen debt**.



Ultimately, the product of glycolysis, pyruvic acid, must be metabolized aerobically. Aerobic metabolism is performed exclusively in the mitochondria. Pyruvic acid is converted to a molecule called an acetyl group and put into a pathway known as the Krebs Cycle. Energy is released in the form of ATP and, especially, as high energy electrons. These high energy electrons are sent to a process within the mitochondria known as the electron transport system which produces the vast majority of the ATP. The waste products of aerobic metabolism are CO_2 and H_2O. The reactant other than glucose is O_2.



Summary of Metabolic Processes in Muscles: The Phosphagen System

See [Facts About Performance Boosters]

Figure 9.16 a

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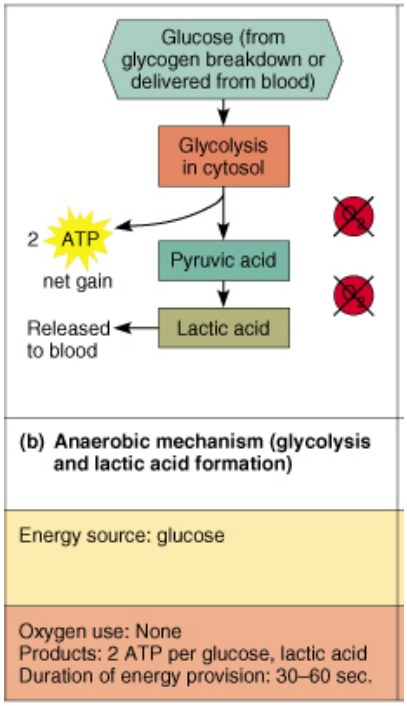
Training can increase the amount of creatine phosphate stored, but this alone does not increase the strength of a muscle, just the length of time before it runs out of CP, and that by only a few seconds. See [Facts About Performance Boosters]

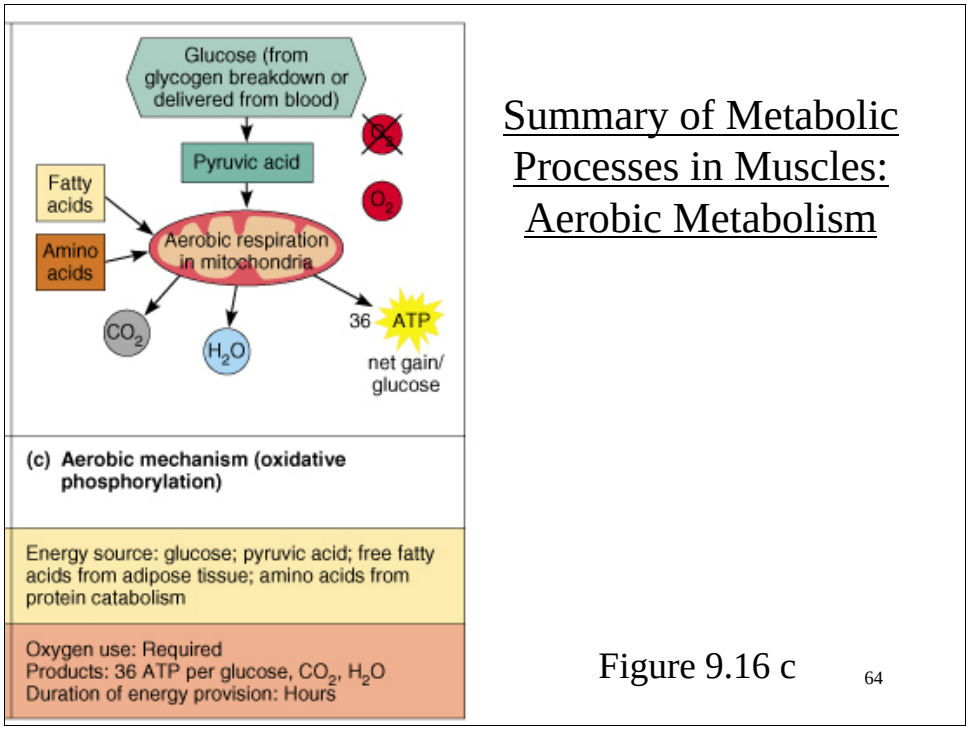


Summary of Metabolic Processes in Muscles: Anerobic Glycolysis

Figure 9.16 b

63

Strength training increases the myofilaments in muscle cells and therefore the number of crossbridge attachments which can form. Training **does not increase the number of muscle cells** in any real way. (Sometimes a cell will tear and split resulting in two cells when healed). Lactic acid removal by the cardiovascular system improves with training which increases the anaerobic capacity. Even so, the **glycolysis-lactic acid system** can produce ATP for active muscle cells for only about a minute and a half.



Summary of Metabolic Processes in Muscles: Aerobic Metabolism

Figure 9.16 c

64

Aerobic metabolism is used for endurance activities and has the distinct advantage that it can go on for hours. Aerobic training increases the length of endurance activities by increasing the number of mitochondria in the muscle cells, increasing the availability of enzymes, increasing the number of blood vessels, and increasing the amount of an oxygen-storing molecule called **myoglobin**.

Types of Muscle Fibers: White Fibers

Fast twitch

Large diameter, used for speed and strength.

Depends on the phosphagen system and on glycolysis-lactic acid.

Stores glycogen for conversion to glucose.

Fewer blood vessels.

Little or no myoglobin.

Training increases the number of myofibrils and, as a result, the muscle size.

65

Different types of cells perform the differing functions of endurance activities and **speed-** strength activities. There are three types, red, white, and intermediate. The main differences can be exemplified by looking at red and white fibers and remembering that intermediate fibers have properties of the other two.

Red Fibers

Slow twitch

Small diameter, used for endurance.

Depends on aerobic metabolism.

Utilize fats as well as glucose.

Little glycogen storage.

Many blood vessels and much myoglobin give this muscle its reddish appearance.

Training increases the number of mitochondria and enzymes for aerobic metabolism. Also increases cardiovascular and respiratory capacity.

Intermediate Fibers: sometimes called "fast twitch red", these fibers have faster action but rely more on aerobic metabolism and have more endurance. Most muscles are mixtures of the different types. Muscle fiber types and their relative abundance cannot be varied by training, although there is some evidence that prior to maturation of the muscular system the emphasis on certain activities can influence their development.

Energy Systems Used in Various S ports

Phosphagen System, almost entirely:	4 M ATP/Min for 8 to 10 Sec.
100 meter dash	
jumping	
weight lifting	
diving	
football dashes	
Phosphagen and Glycogen-lactic Acid systems:	
200 meter dash	
basketball	
baseball home run	
ice hockey dashes	
Glycogen-lactic Acid System, mainly	2.5 M ATP/Min for 1.3 to 1.6 Min.
400 meter dash	
100 meter swim	
tennis	
soccer	
Glycogen-lactic Acid System and Aerobic System	
800 meter dash	
200 meter swim	
1500 meter run	
boxing	
2000 meter rowing	
1500 meter run	
1 mile run	
400 meter swim	
Aerobic System:	
10,000 meter skating	
cross-country skiing	
marathon run	
jogging	

Pathways Used in Various Activities

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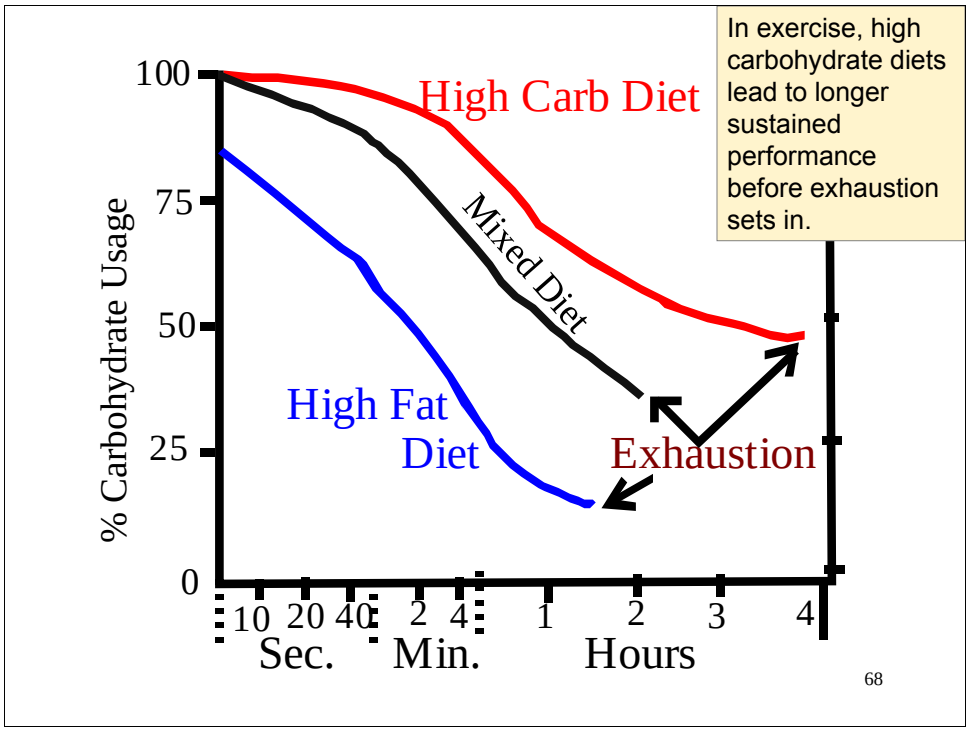
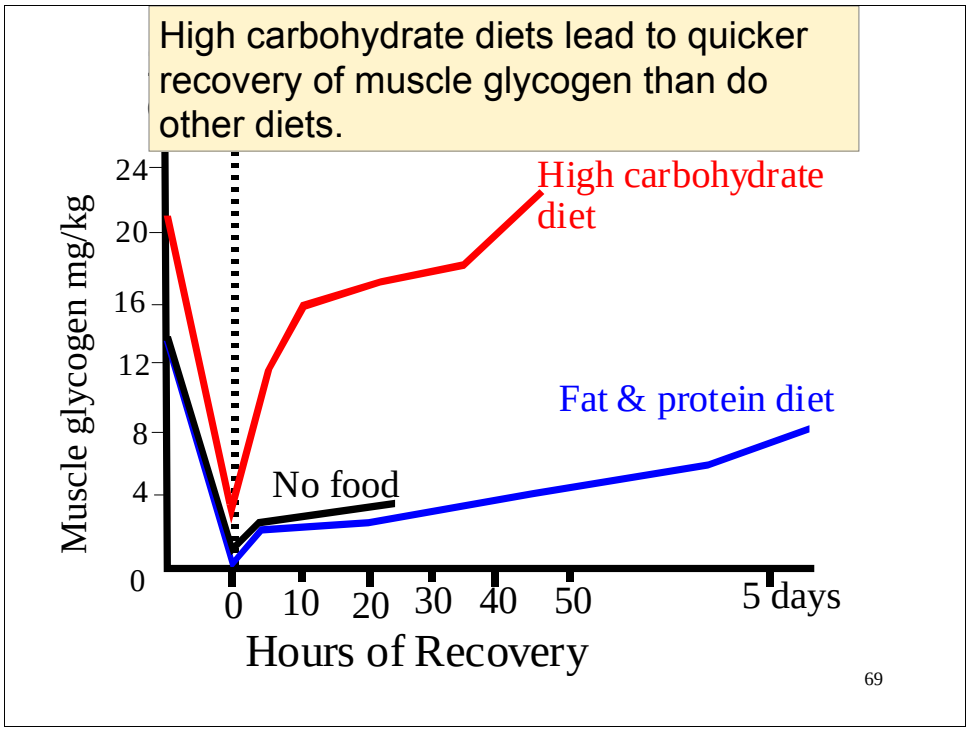


Effect of duration and type of diet on relative percentages of carbohydrate or fat used for muscle energy. Percentage carbohydrate used is proportional to glycogen reserves and inversely proportional to fat usage.



Effect of diet on the rate of muscle glycogen replenishment after prolonged exercise. This study compares athletes who have had a high carb diet with those who have had high fat & protein diets and with those who have fasted.