

# OVERVIEW of WEEK 8

## ~ PRINCIPLES OF BIOMECHANICS HK\*2270 ~

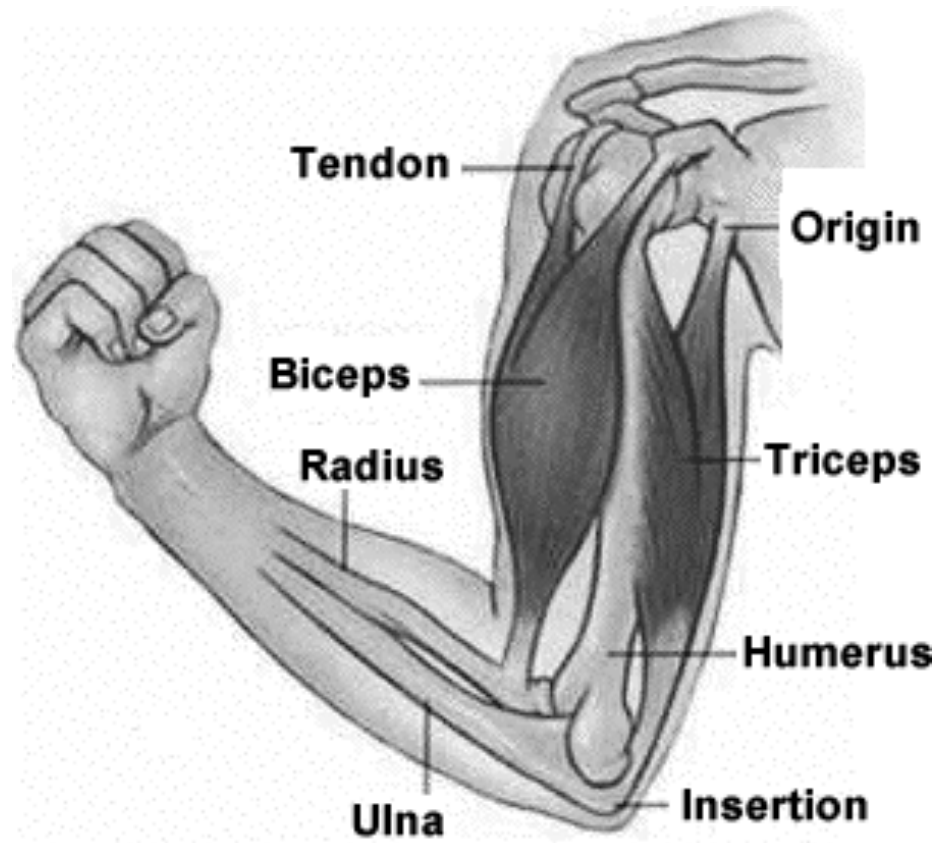
*Lori Ann Vallis, FALL 2016*

**Problem set 6 will be on the quiz for sure, plus some content from this lecture.**

### **Concepts**

- **Muscle Mechanics**
- **Sliding filament theory**
- ***Neurophysiology of muscle contractions***
- ***Force – Length curves***
- ***Force – Velocity curves***

# MUSCLE MECHANICS!



Anatomy of skeletal muscle

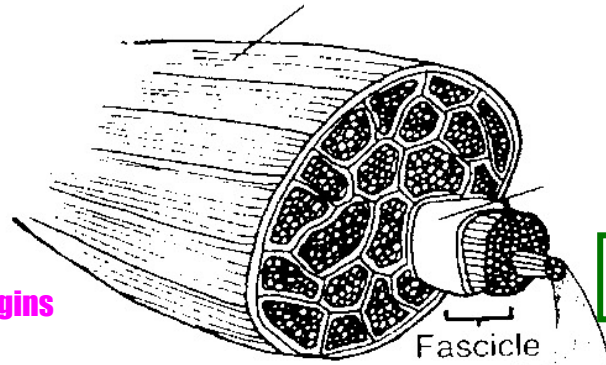
SKELETAL MUSCLE:

Muscle

Epimysium surrounds

Surrounds the muscle tissue itself.

When you cut through this, the muscle begins to fall apart.



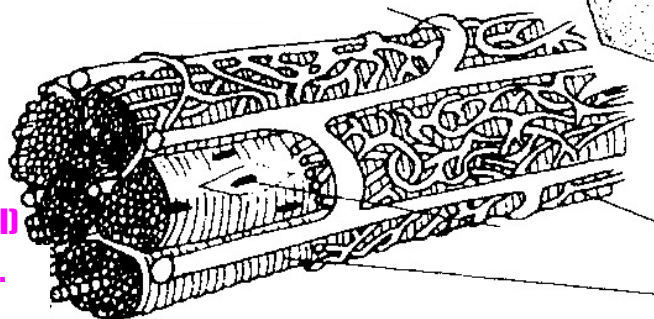
Perimysium surrounds

Endomysium

Single muscle fiber (cell)

Surrounds the single muscle fiber (cell)

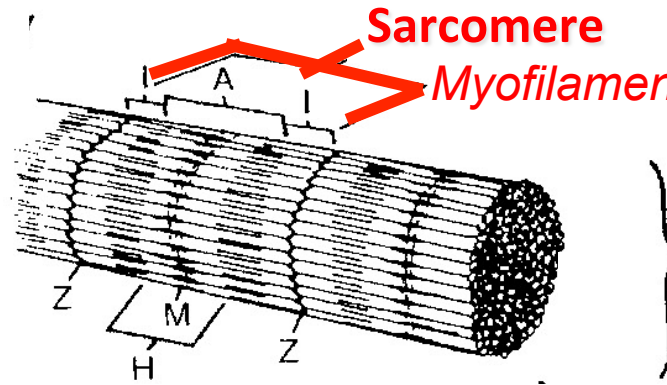
What makes beef juicy is endomysium.



Sarcomere

Myofilaments

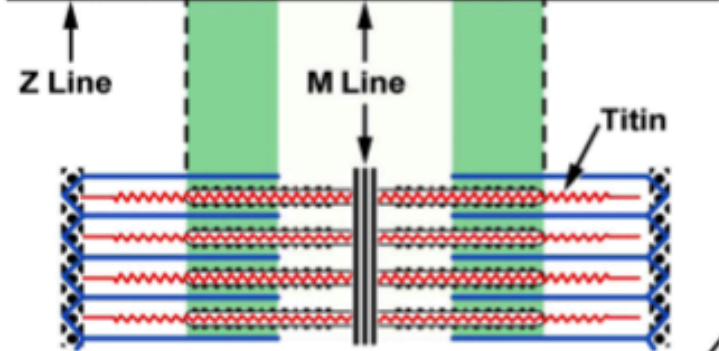
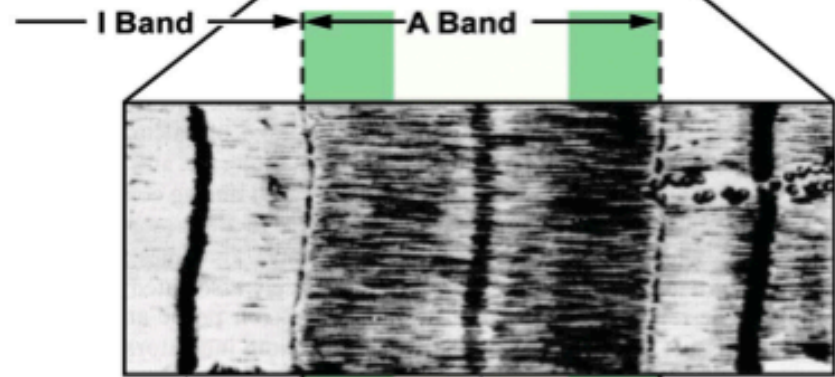
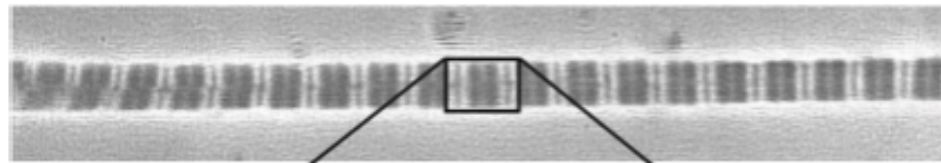
contains the two myofilaments; can measure the level of forces produced by these two



Myofibril

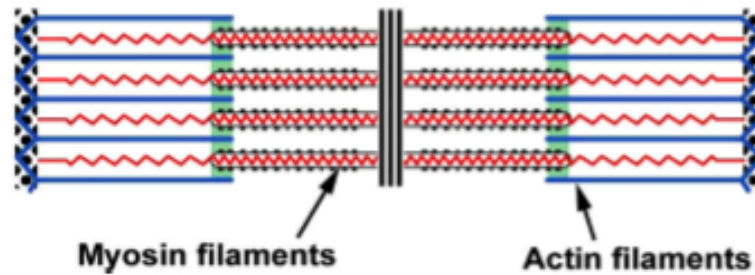
Anatomy of skeletal muscle

This is the sarcomere.



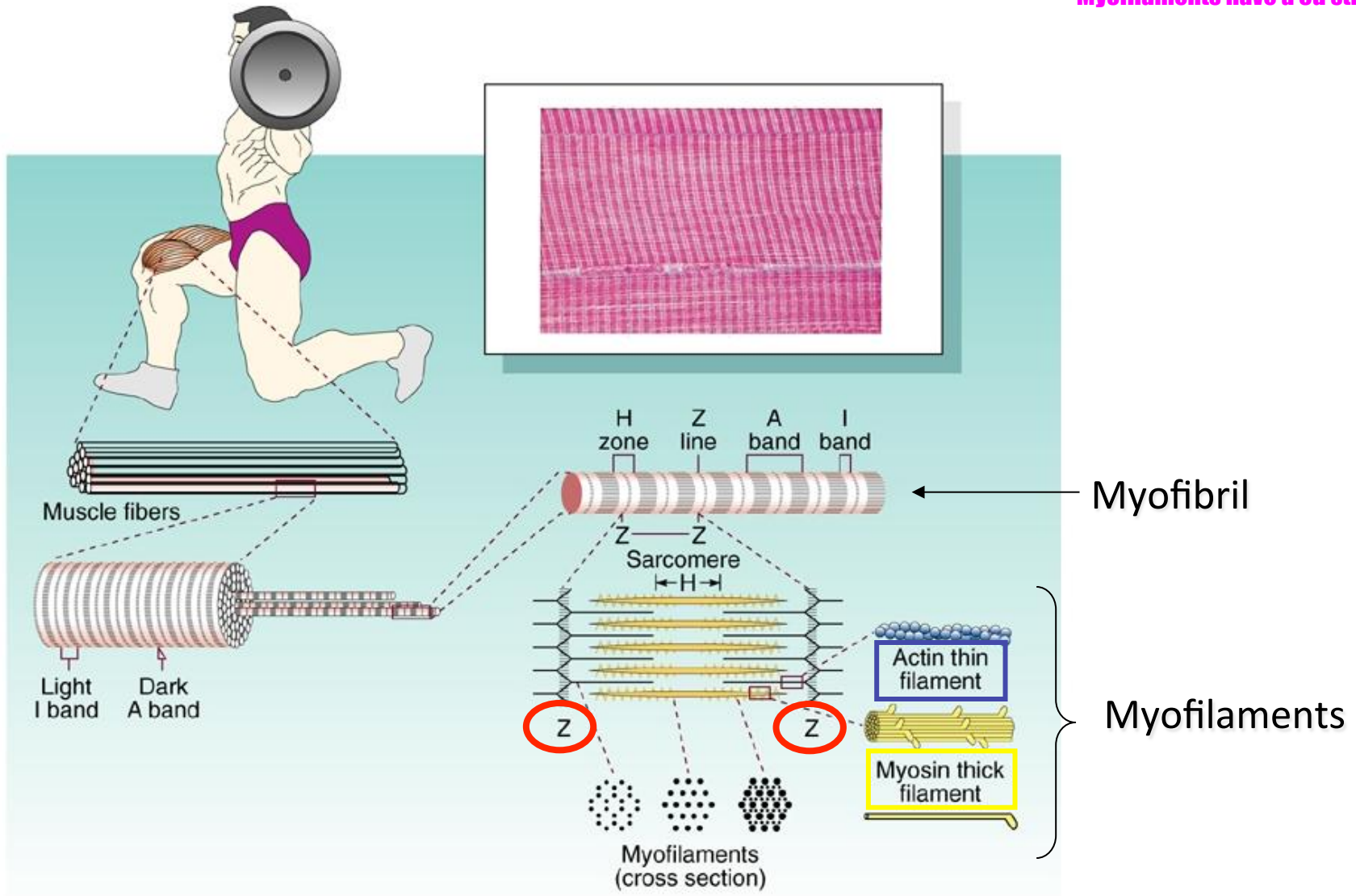
Thick lines in red are the myosin.  
The sarcomere stretches.

Sarcomere stretch



Contractile Mechanism

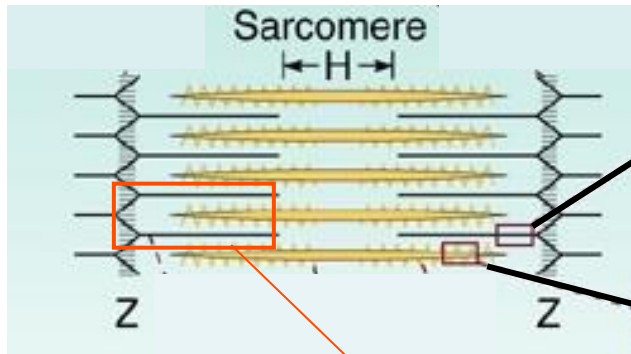
Myofilaments have a 3d structure.



Copyright © 2006 Lippincott Williams & Wilkins.

*How does a muscle contract?*

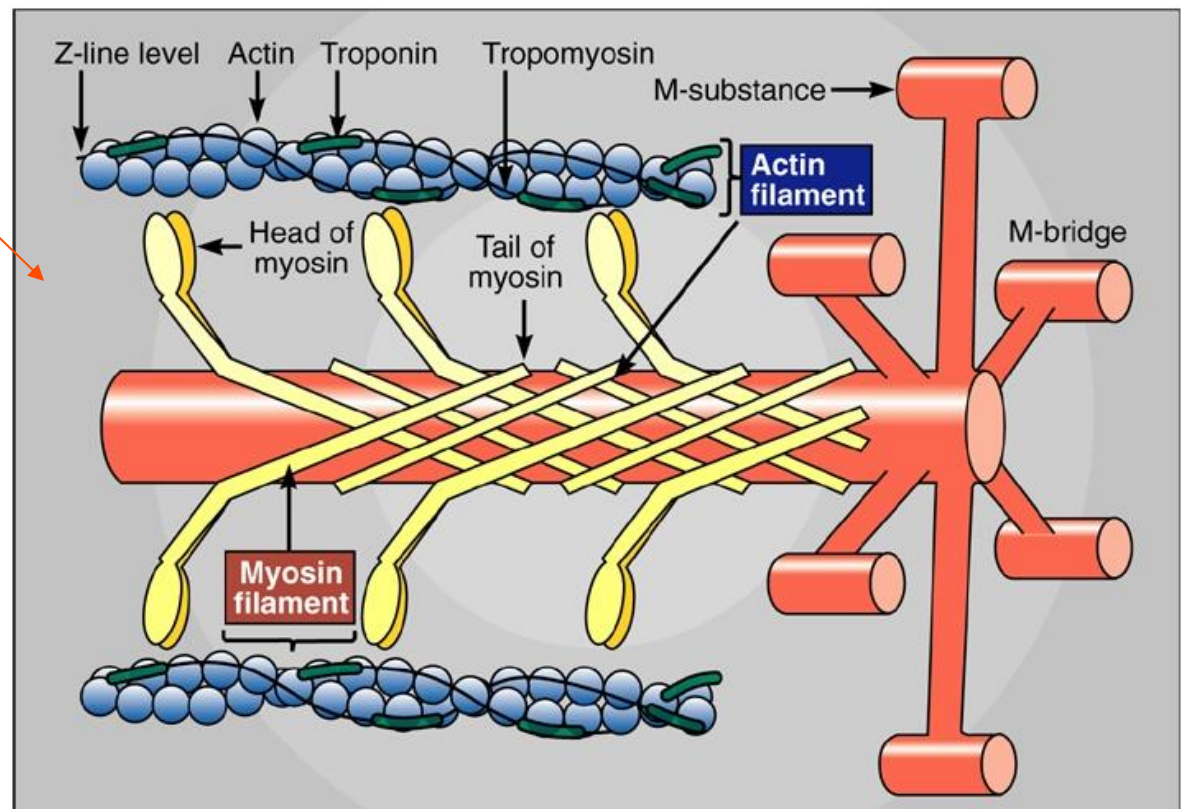
Contractile Mechanism



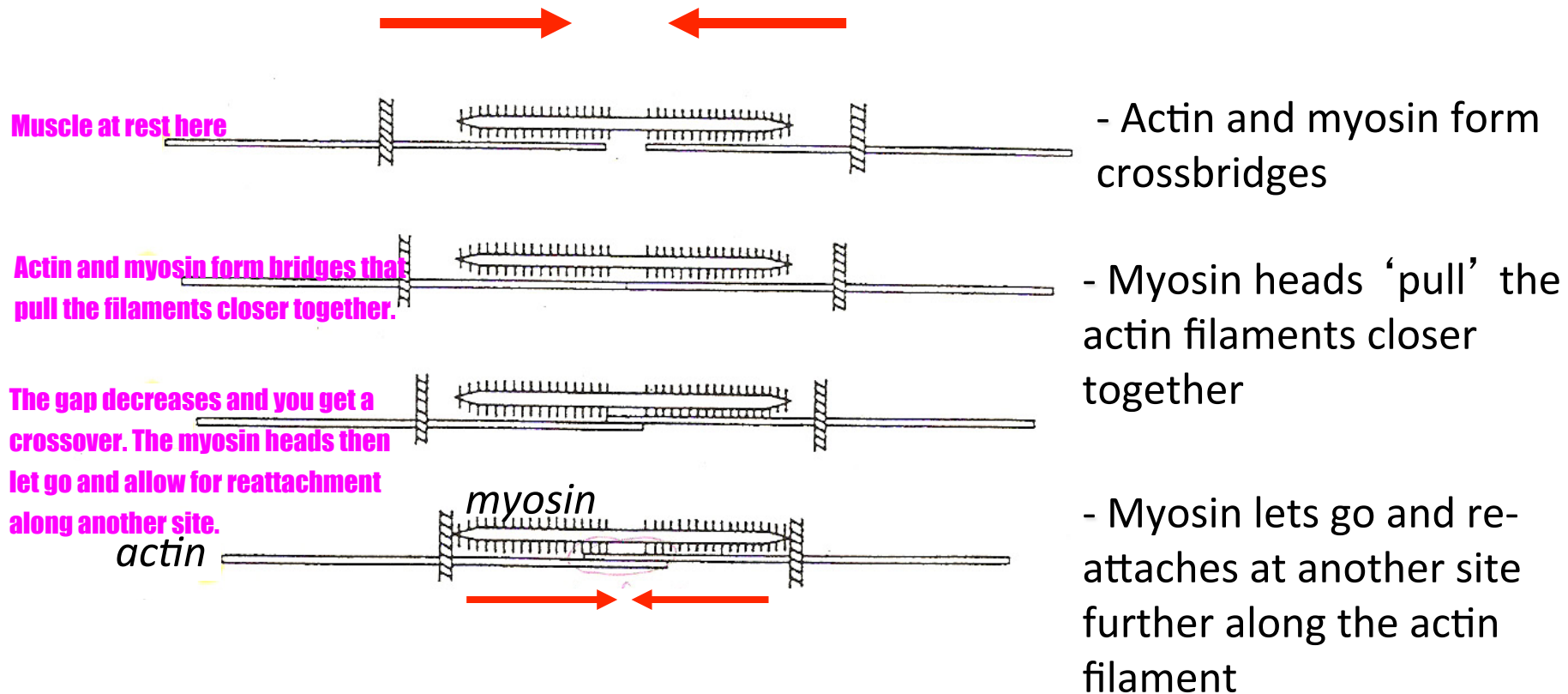
actin

myosin

6 prong fibers on myosin (in red here). The actin surrounds it (in yellow), looks like an oar going into water.



## Contractile Mechanism

Sliding filament theory

**Result?** Z-discs move closer together = sarcomere shortens.

# ***Muscular Contraction and Sliding Filament Theory***

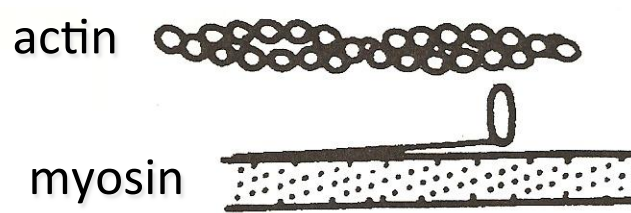
*<http://www.youtube.com/watch?v=EdHzKYDxrKc>*



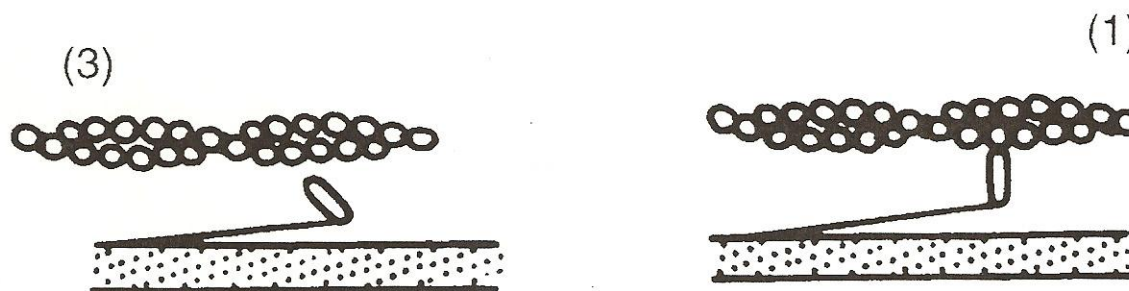
Contractile Mechanism

Cross-bridge cycle

**4. Myosin head in high energy position**



Myosin protein head detaches from actin. Then have an resting state.

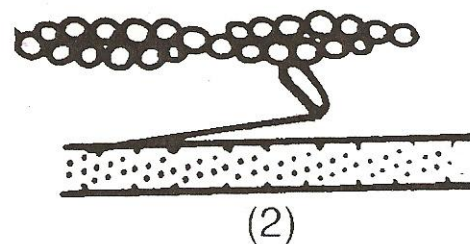


**3. Cross-bridge detachment**

**1. Cross-bridge attachment**

Chemical that causes this

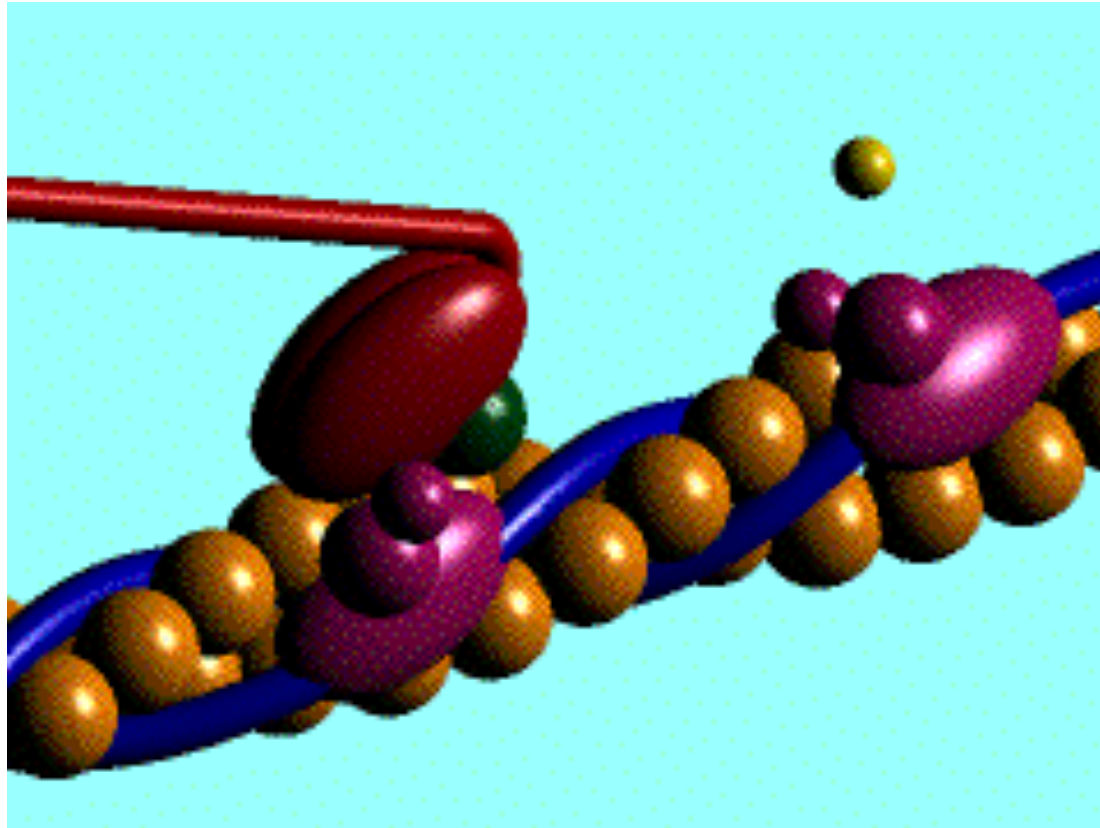
Got myosin head connecting to actin fibers.



**2. Power Stroke**

Literally where you got the connection and it starts to slide (myosin and actin slide on top of each other)

## Contractile Mechanism

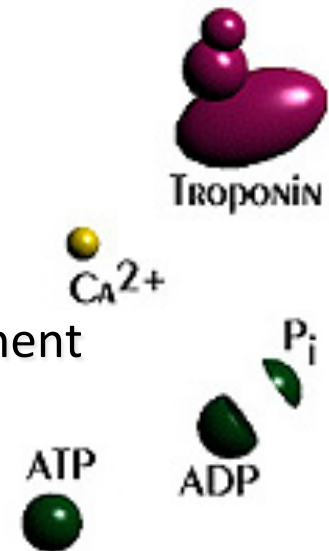


Important players:

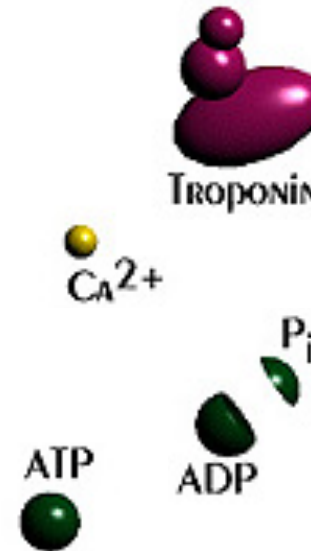
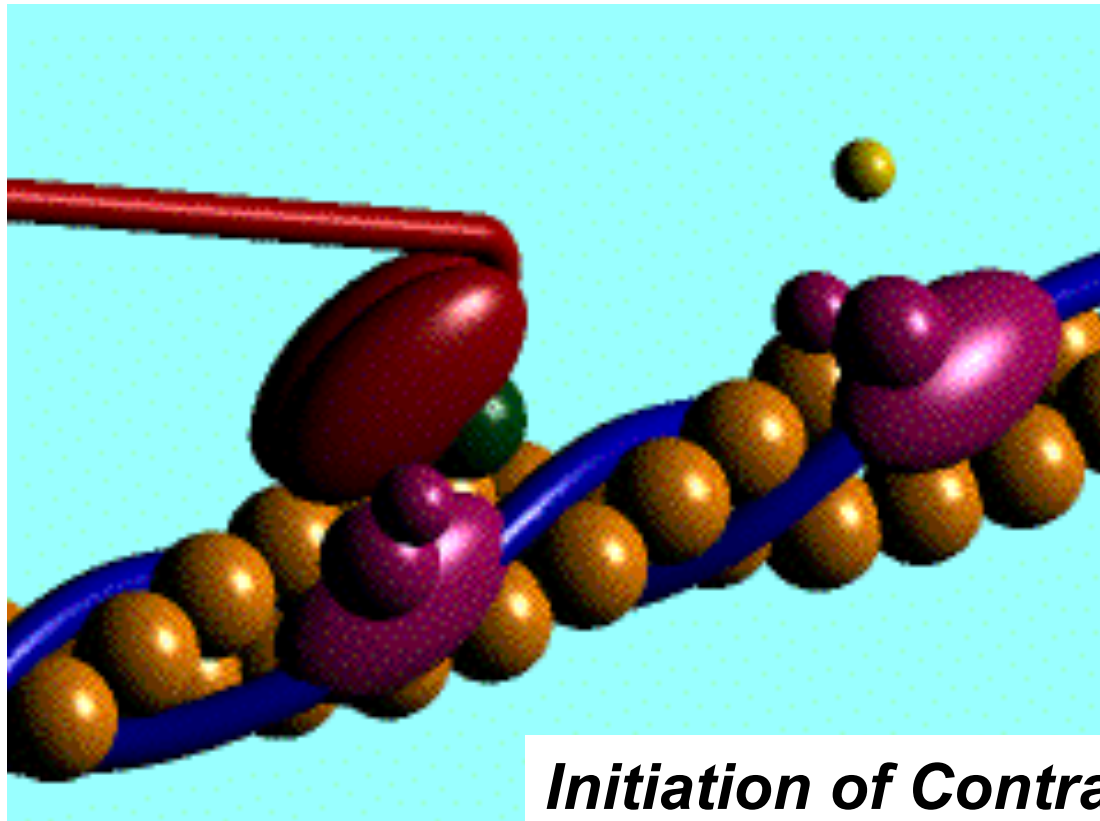
$\text{Ca}^{2+}$  : opens up the myosin-binding sites on the actin filament

ATP : (1) hydrolysis of ATP into ADP +  $\text{P}_i$   
 (2) causes myosin to become unbound from actin

**Called the detachment phase**



Contractile Mechanism



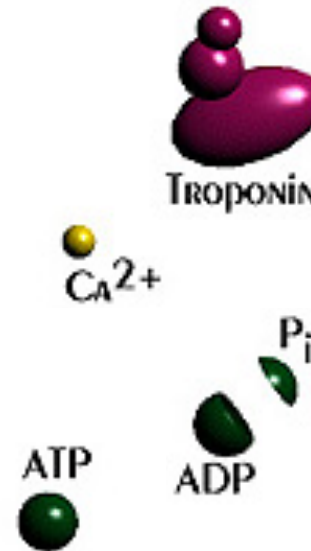
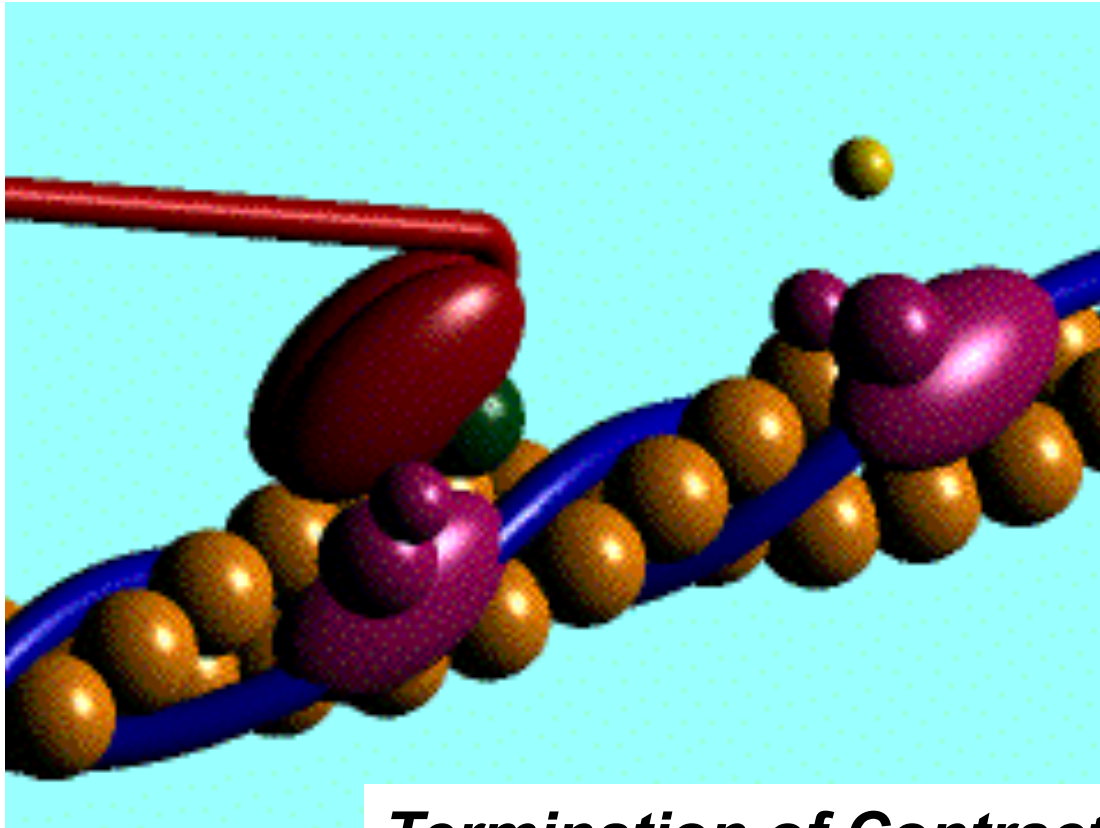
**Initiation of Contraction**

Breaks down of ACh within the structure of the cell (this is how a structure is terminated)

1. Acetylcholine (ACh) released, binding to receptors
2. Action Potential reaches T-tubule **neural control (coming from the brain), reaches the T-tubule; releases calcium.**
3. Sarcoplasmic reticulum releases  $Ca^{2+}$
4. Active site exposure: cross-bridge binding **causes cross bridge binding between myosin and actin.**
5. Contraction begins **Powerstroke happens and shortening slide bone theory happens.**

**Have to have the right chemicals in the right place and the neural contribution. Can be from different input sources (thinking of something then doing it vs reflexes)**

## Contractile Mechanism

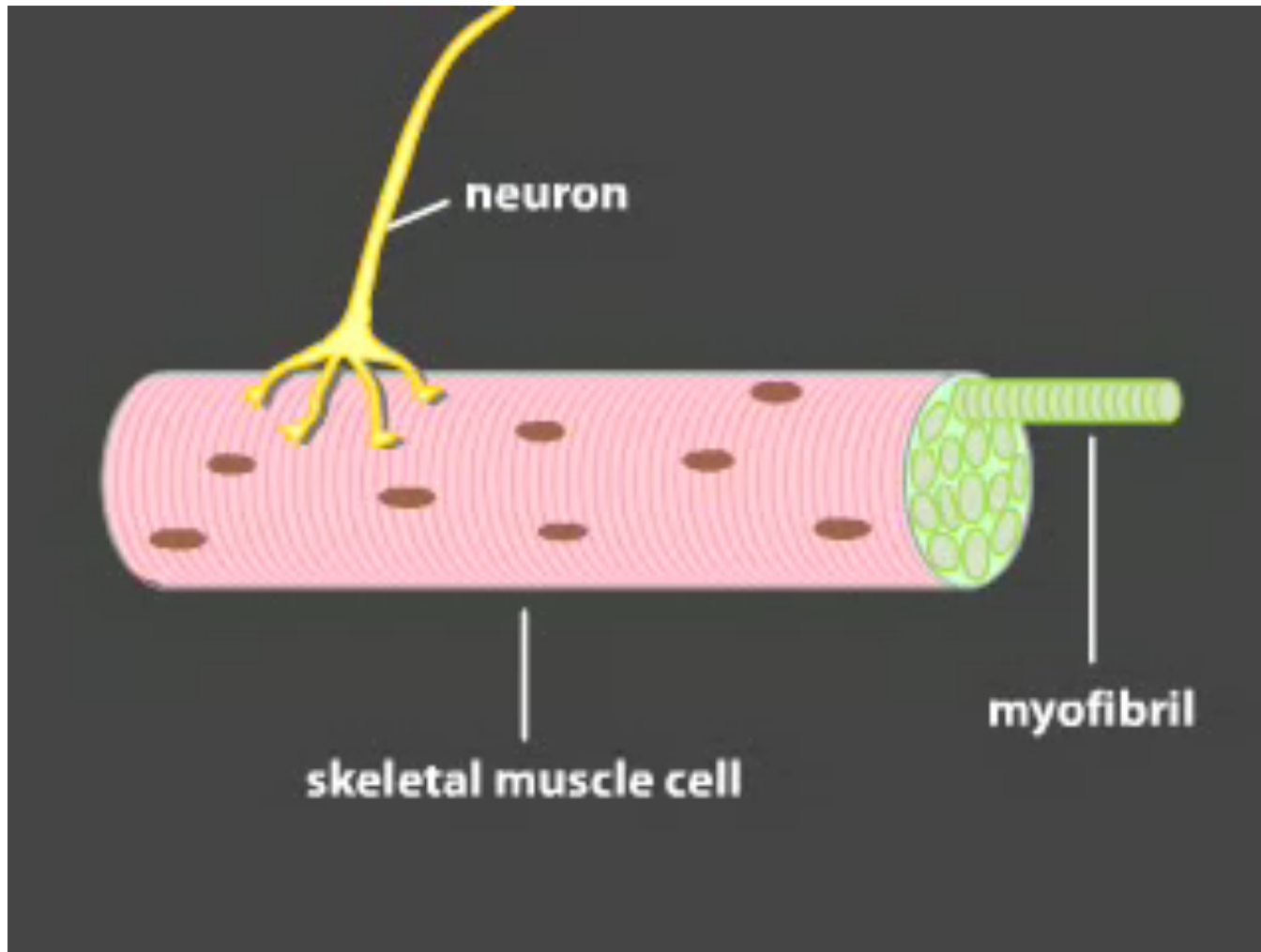
***Termination of Contraction***

1. ACh removed by acetylcholinesterase (AChE)
2. Sarcoplasmic reticulum recaptures  $Ca^{2+}$
3. Active sites covered, no cross bridge formation
4. Contraction ends

***How does cross  
bridge formation  
happen?***

# Sliding Filament Theory: Depolarization & Release of $\text{Ca}^{2+}$

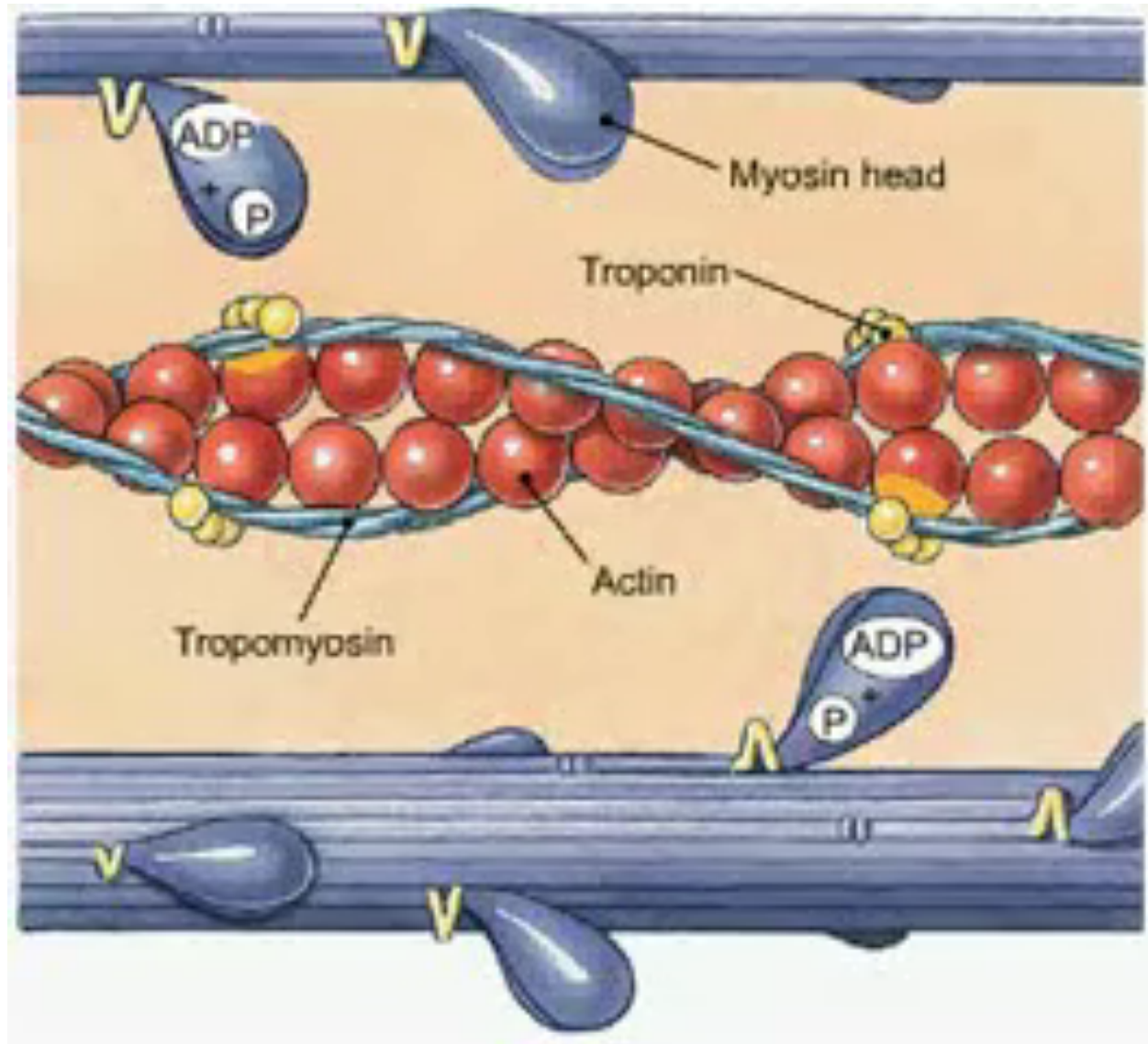
<http://www.youtube.com/watch?v=CepeYFvqmk4>



Impulse from neuron that comes down to the skeletal muscle cell. Watch the video and make notes!

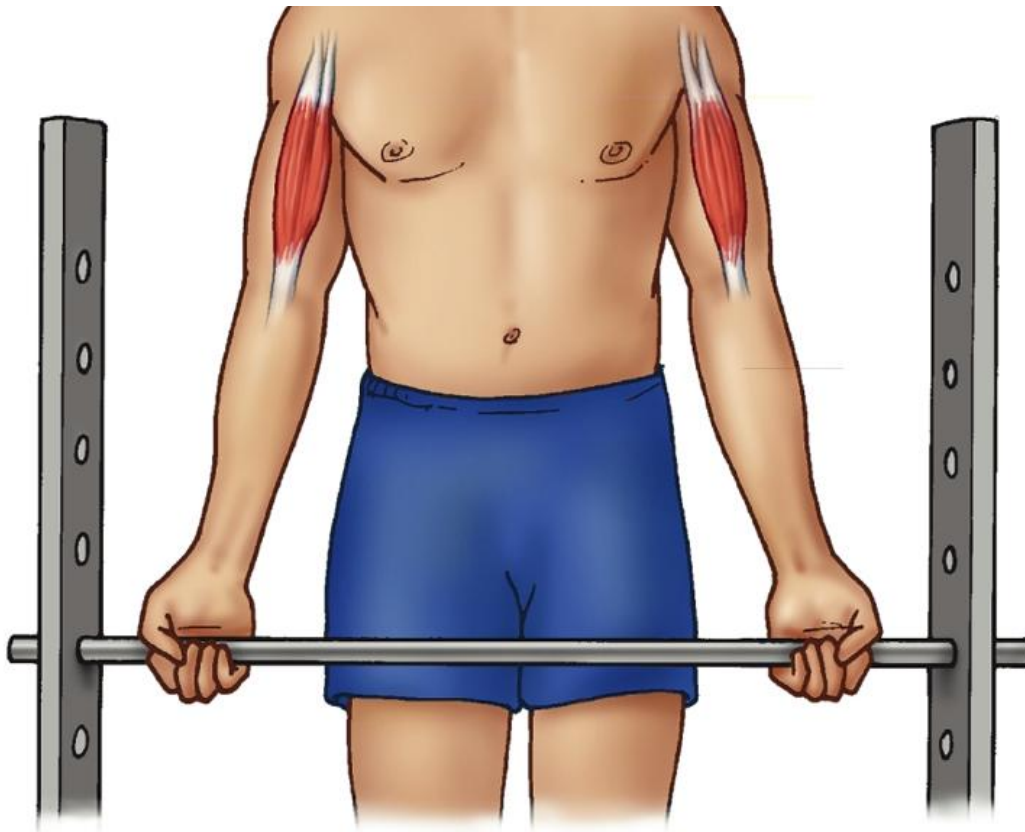
## ***Sliding Filament Theory: Calcium, ATP/ADP + Pi***

<http://www.youtube.com/watch?v=mWPmUqRZYIs>



## Contraction Types

No movement at all is isometric contractions. It means that the muscle fibers generate tension but there is no change in length. An example would be trying to move a fridge and it doesn't budge because it is so big. There is still tension just no shortening of those fibers.



Copyright © 2006 Lippincott Williams & Wilkins.

Movement: **No movement**

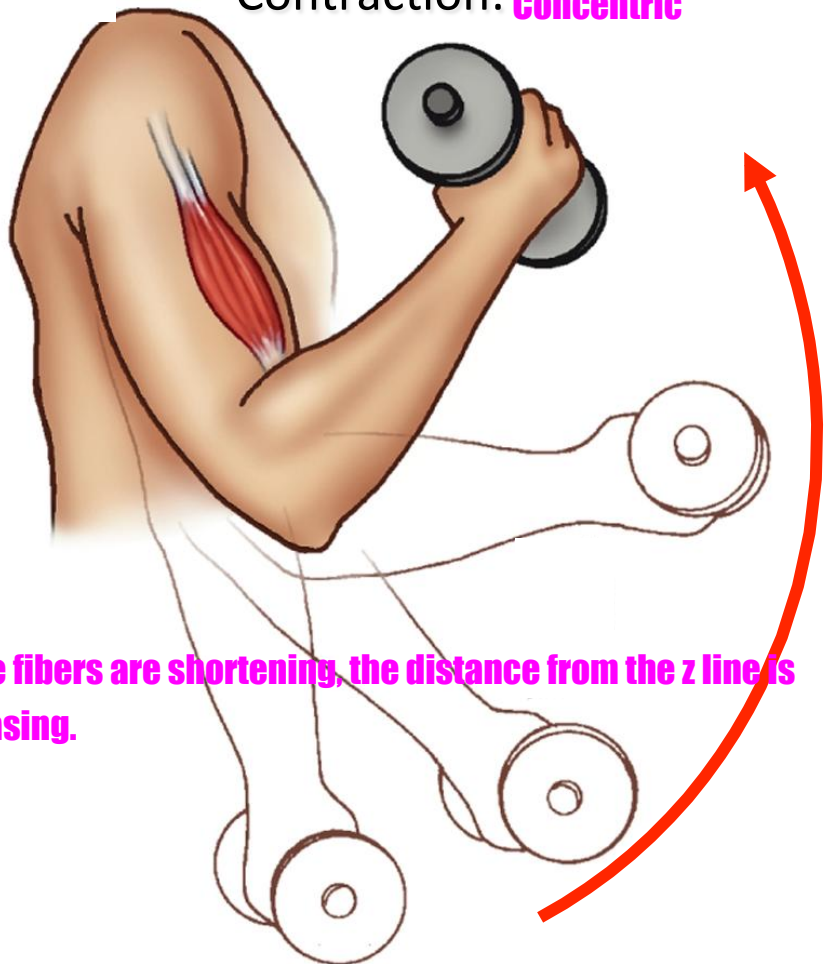
Contraction: **Isometric**

**Isometric** contraction:  
when muscle fibers generate  
tension but there is **no change in length**

Contraction Types

Bicep curl

Movement: Flexion  
Contraction: Concentric



Muscle fibers are shortening, the distance from the z line is decreasing.

Copyright © 2006 Lippincott Williams & Wilkins.

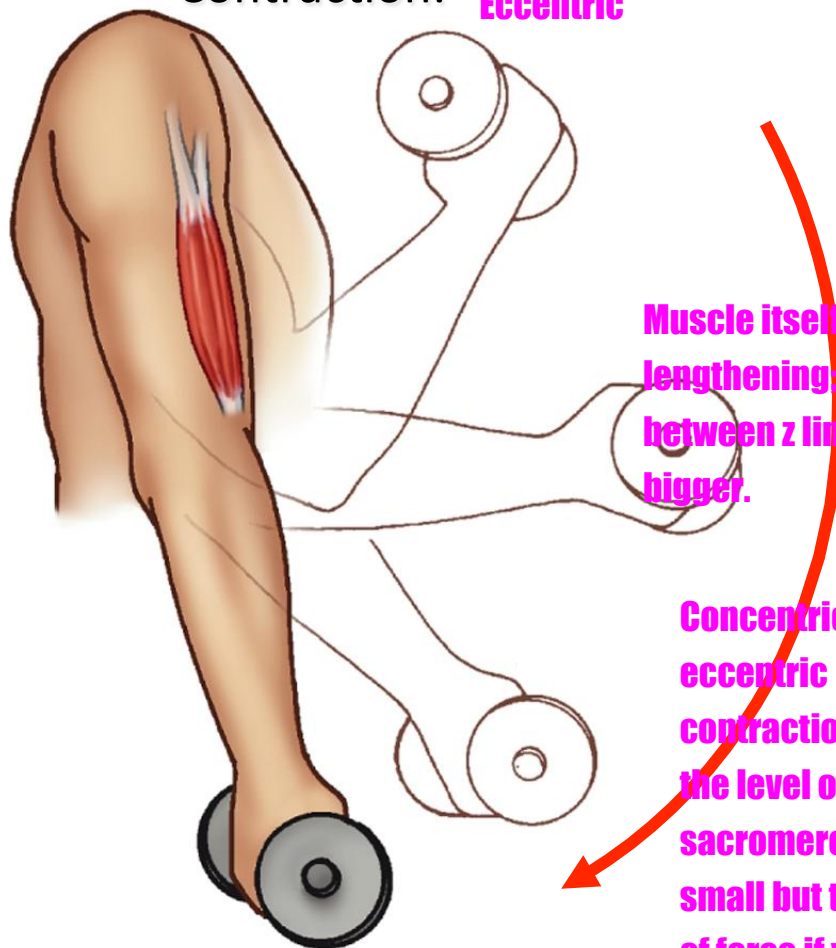
**Concentric**

contraction:

muscle fiber **SHORTENS** while generating tension

**Muscles are ALWAYS contracting!**

Movement: Extension  
Contraction: Eccentric



Muscle itself is lengthening; distance between z lines is getting bigger.

Concentric and eccentric contractions are at the level of the sarcomeres, seem small but there is a lot of force if you look at the whole muscle (all those sarcomeres acting together to get the muscle to contract)

Copyright © 2006 Lippincott Williams & Wilkins.

**Eccentric**

contraction:

muscle fiber **LENGTHENS** while generating tension

# Types of Muscular contraction: At any given instant in time

Note:  $M_m$  = Moment generated by Muscle AND  $M_R$  = Net Joint Moment

## Isometric



Static sol  
 $M_R = M_m$

## Concentric



Dynamic now  
Actually doing a flexion moment, in this situation, when there is motion and a change in acceleration, then:  
 $M_R < M_m$

## Eccentric



Dynamic movements  
Muscles could still be contracting this way but motion is in the opposite direction so:  
 $M_r > M_m$   
Coming back into an extended position.

Now must think of not only the direction of the movement, but also the magnitude of the two muscle components.

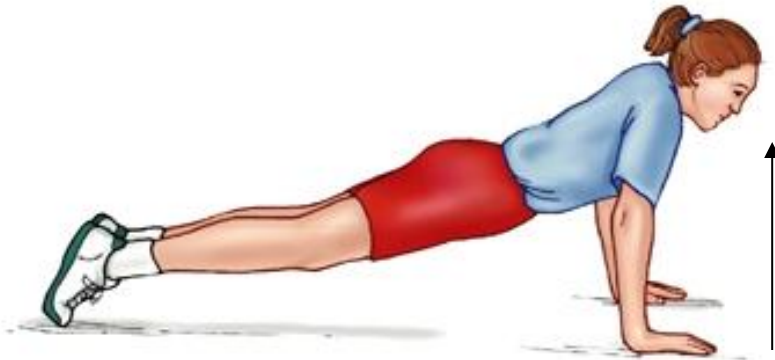
Eccentric actions of muscles are used as brakes! They make sure that this woman doesn't go into hyperextension. Eccentric are critical to prevent going past safe range.

Contraction Types

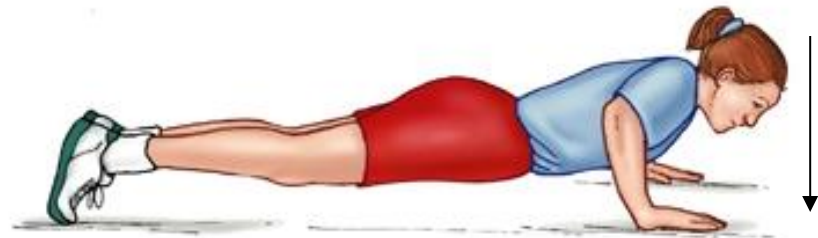
PUSH – UPS:

Triceps – elbow extensors

**Tricep muscles - they are shortening.**



**Concentric - lengthening here.**  
**When one muscle group shortens, the other lengthens**  
**for a controlled action.**



**Eccentric - harder to do at the gym!**  
**Tricep muscle EXTENDS the elbow!**

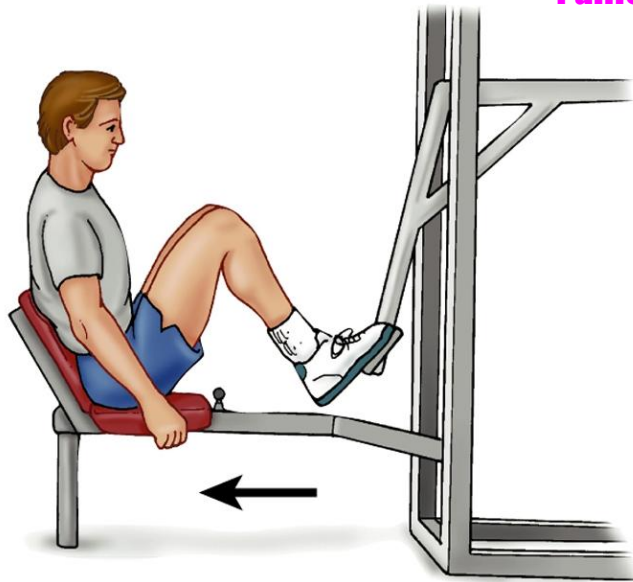
Contraction Types

LEG PRESS:

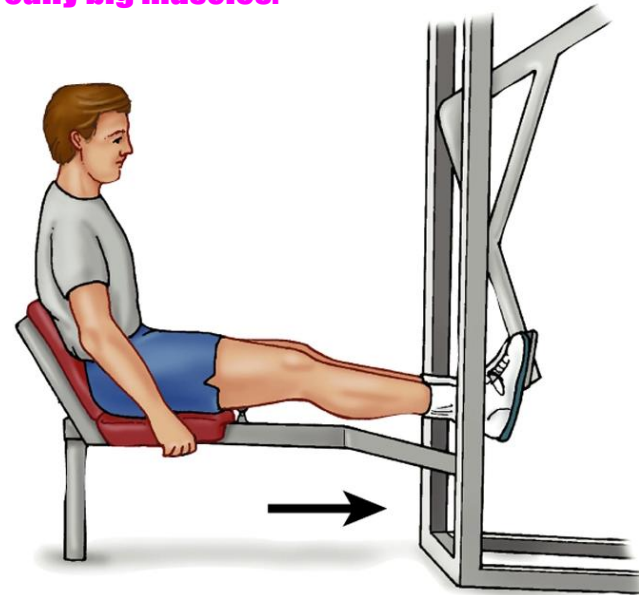
Quadriceps – leg extensors

4 different muscles, really big muscles.

Eccentric



Concentric



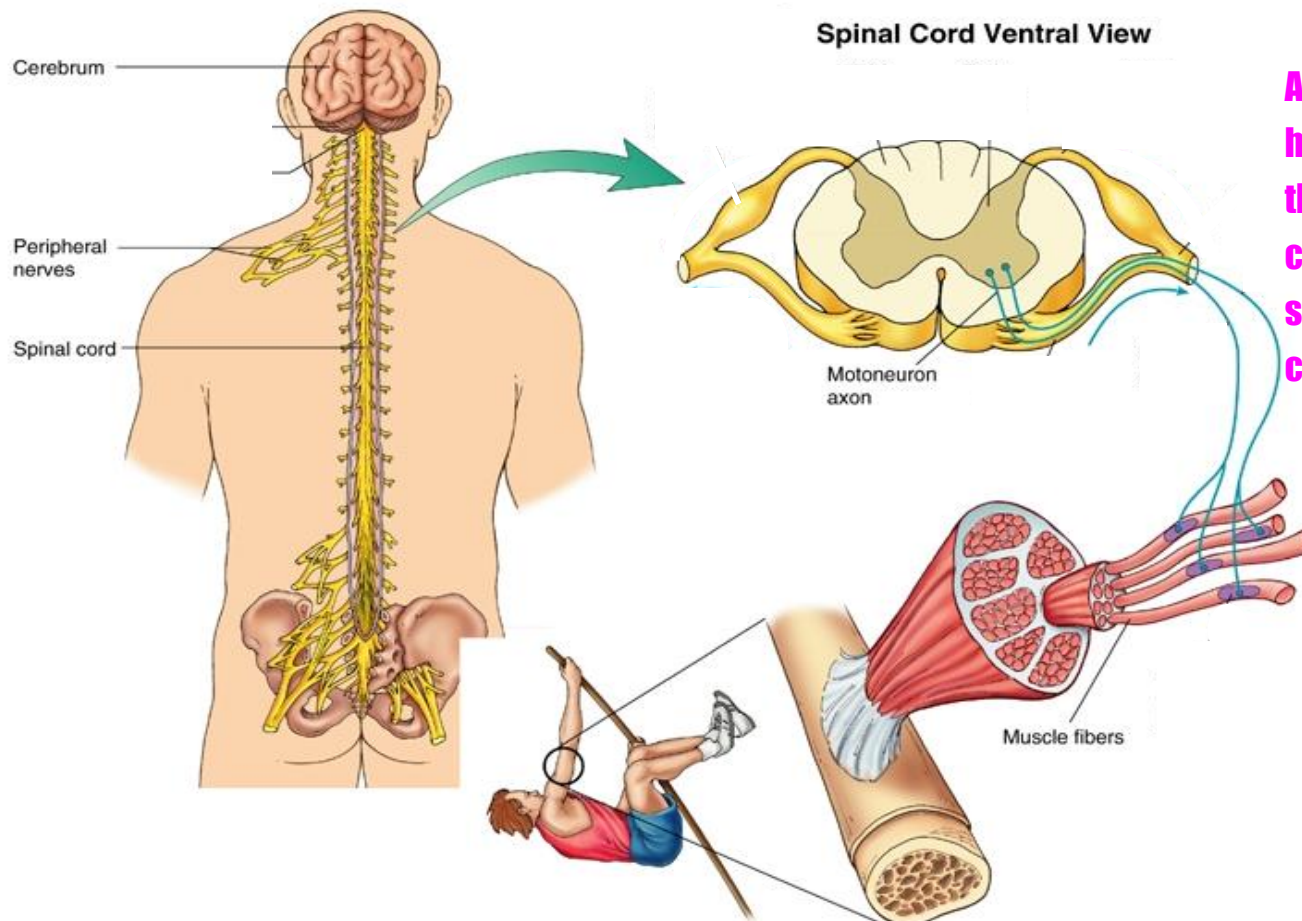
As you push, the reactive force is underneath feet and it is pushing back.

To determine if concentric or eccentric, look at whether the muscle is shortening or lengthening.

## Neurophysiology of Muscle Contraction

How do we initiate a muscle contraction?

With a neural signal: **Action potential**



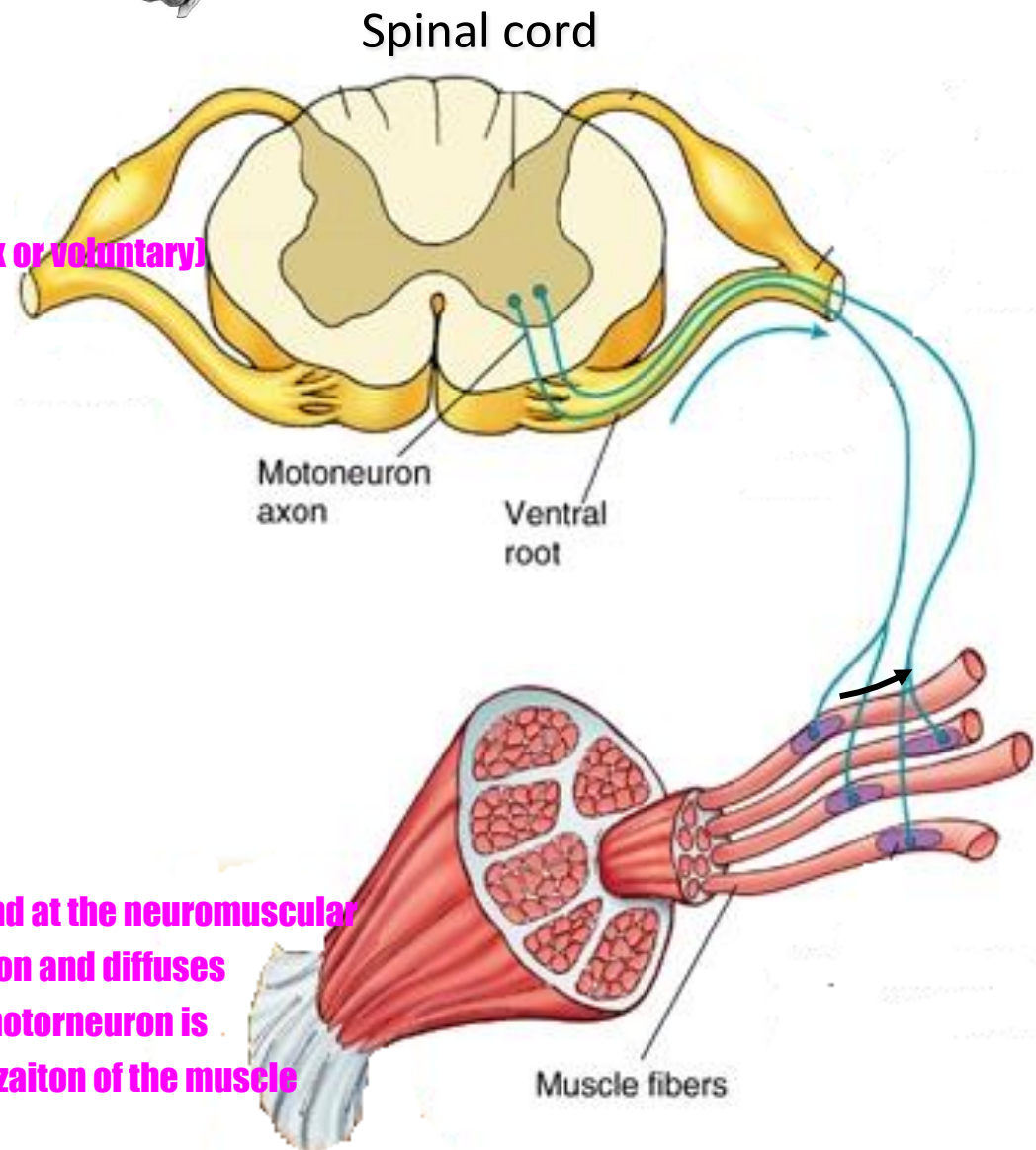
## Neurophysiology of Muscle Contraction



## Initiation of a muscle contraction

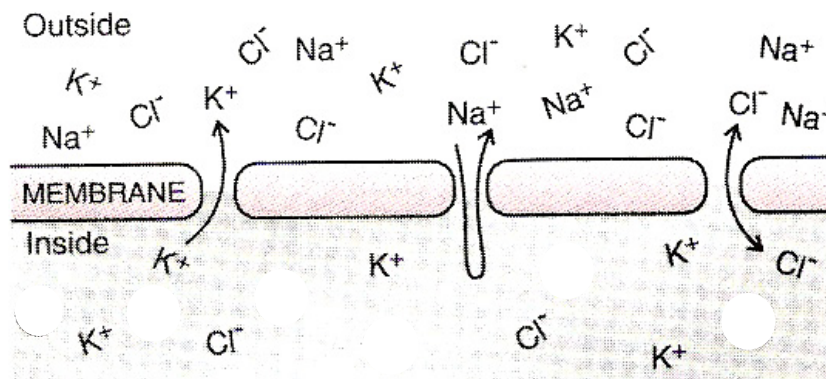
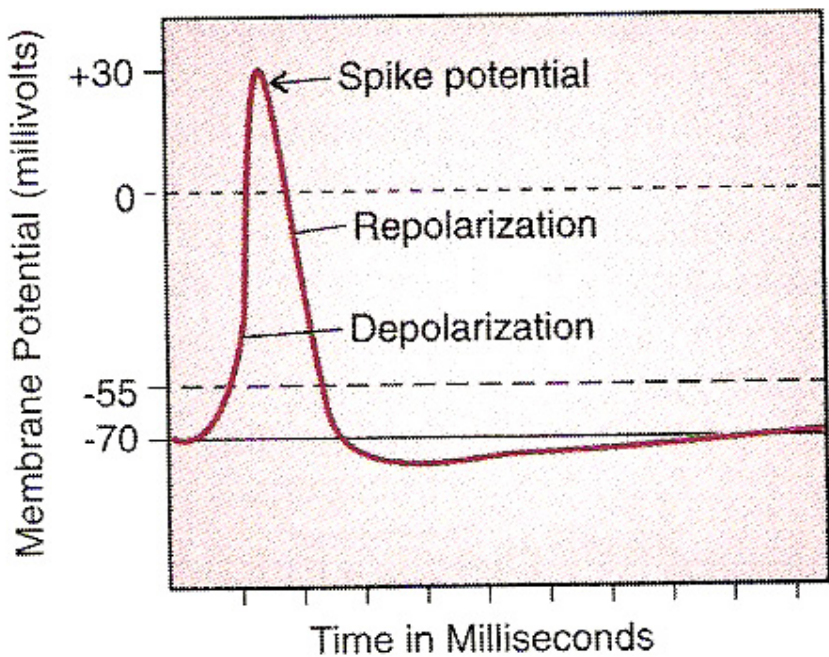
1. CNS initiates depolarization of motor neuron **Have to have some type of input from the brain (reflex or voluntary)**
2. Depolarization is conducted along the motor neuron axon to the muscle fibers
3. At the neuromuscular junction acetylcholine (ACh) is released → diffuses across the synaptic cleft → causes rapid depolarization of the muscle fiber

**Action potentials zip along the axon and at the neuromuscular junction, ACh comes across the junction and diffuses across the synaptic cleft (where the motoneuron is innervating) and causes rapid depolarization of the muscle fiber.**

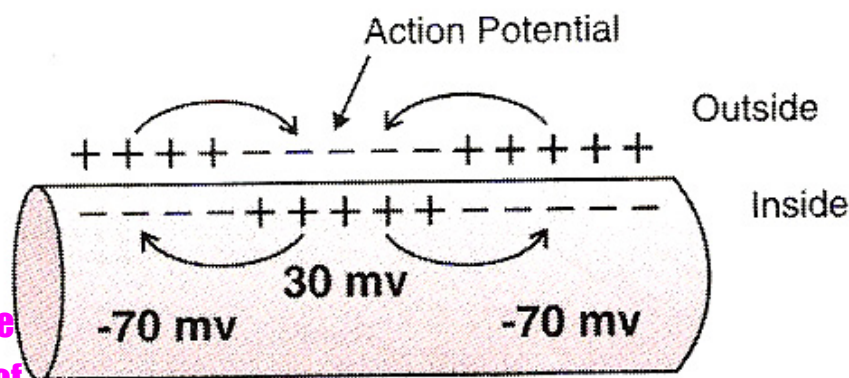


# Neurophysiology of Muscle Contraction

Propagation of an Action Potential down the nerve



Exchange of Ions Across Membrane

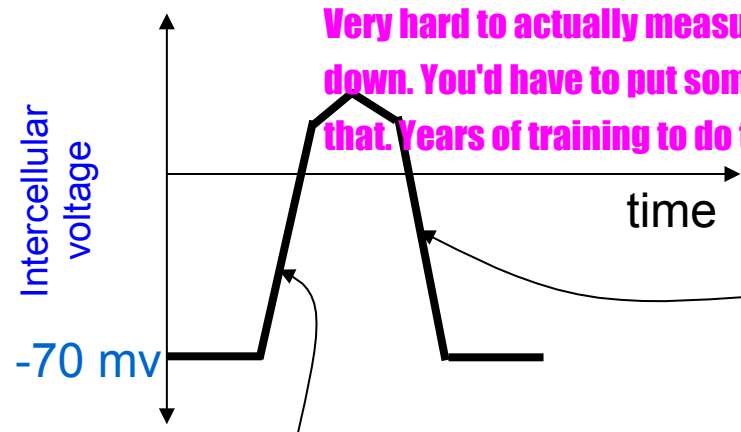
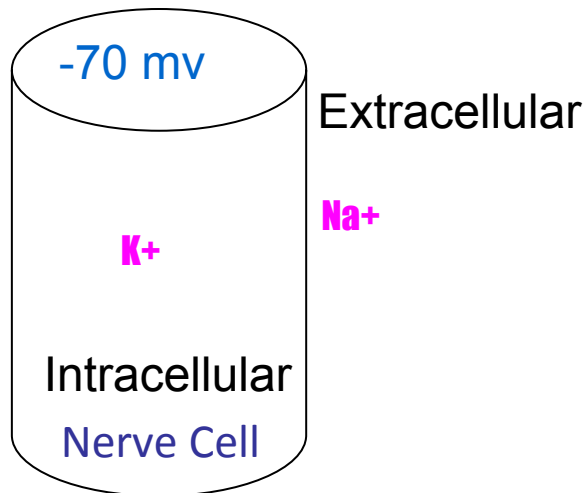


Action Potential Generated Through Change in Electrical Potential

At the action potential, you can measure it. On the y axis, there is the membrane potential vs time in milliseconds, the time of the response changes depending on the muscle and the type of neuron. Exchange of ions across a membrane. Exchange of ions that occur coming in and out of the cell. Across the sodium-potassium gradient, starts to change the polarity of the muscle fibers themselves. It's not that they're positive ions, some are just more positive than the other. Typically inside, it is negative (more negative than outside that is). Have a change in the electrical potential of the cell itself. You have an action potential that is coming down and that's how a muscle flexion is delivered.

## Origin of Electromyography

**EMG** does not measure tension directly....



- Active transport: **Sodium-Potassium pump**
- Equilibrium...until the balance is upset by

**Nerve impulse (stimulation)**

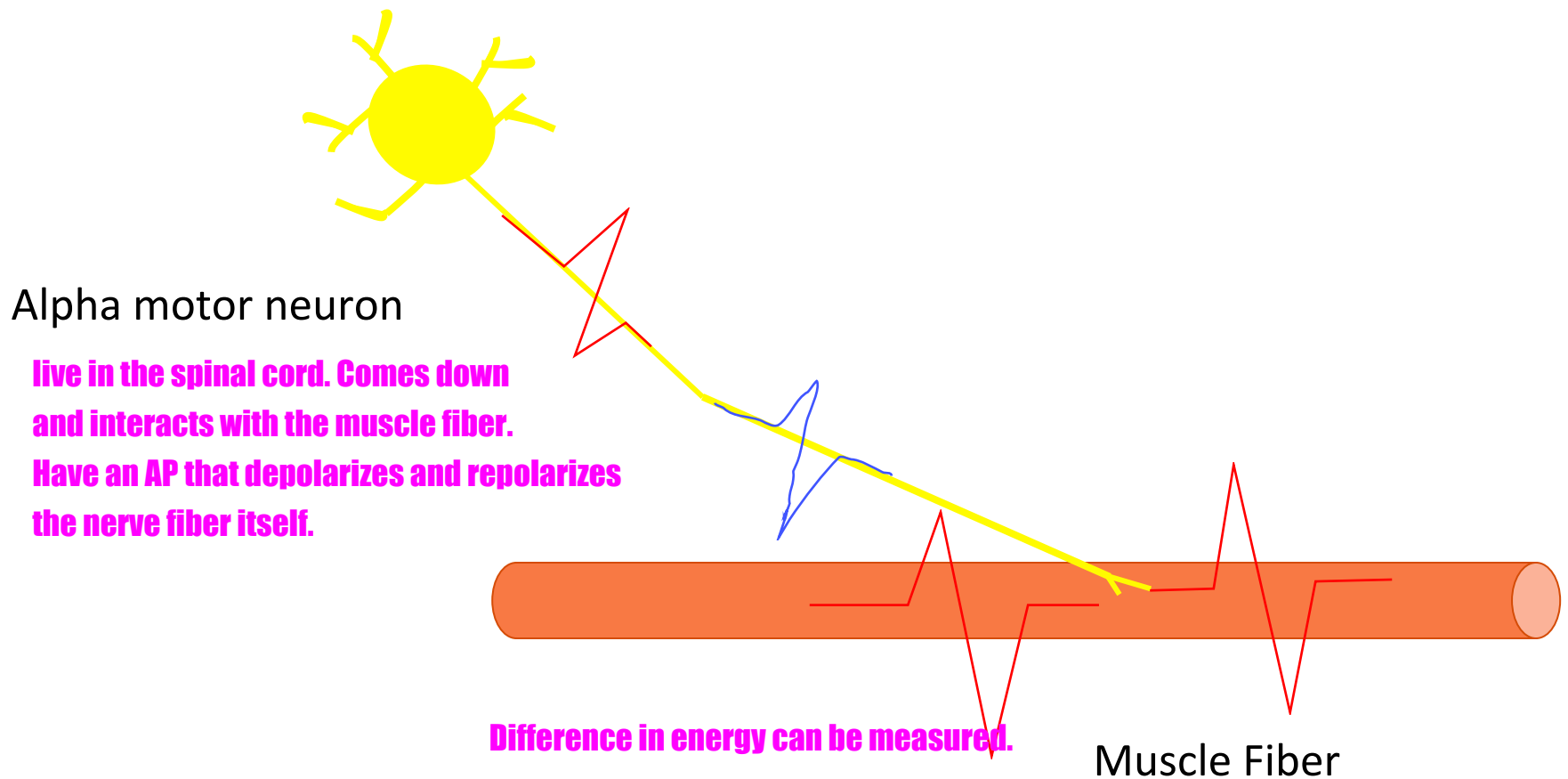
- **Depolarization:** causes increase in permeability to **Na+**; **Na+** flows in, **K+** flows out

Resting Potential is **increased!**  
If stimulus strong enough,  
**Action Potential initiated**

**High amount of K+ inside the cell. Got sodium on the outside of the cell. K+ exits the cell and Na+ floods into the cell when the gates open. Intracellular matrix. Goes from -70 mv and becomes a little more positive. Becomes less negative (+30 mv). Spike potential then a repolarization as it comes back to its resting position. The neural impulse (action potential) needs a strong enough stimulus. If AP is not strong enough, then there is no AP initiator.**

## Neurophysiology of Muscle Contraction

Initiation of a muscle contraction



Action potential (depolarization/repolarization) travels down the nerve axon AND the muscle fiber membrane

## Action at a SINGLE muscle fiber

Twitch Force: Smallest amount of measurable force from a muscle fiber. The amount of tension produced by a muscle fiber from a SINGLE action potential.

The



**Wave summation:** Second contraction is induced before muscle has been completely relaxed; more  $\text{Ca}^{2+}$  is released. Contractions are summed

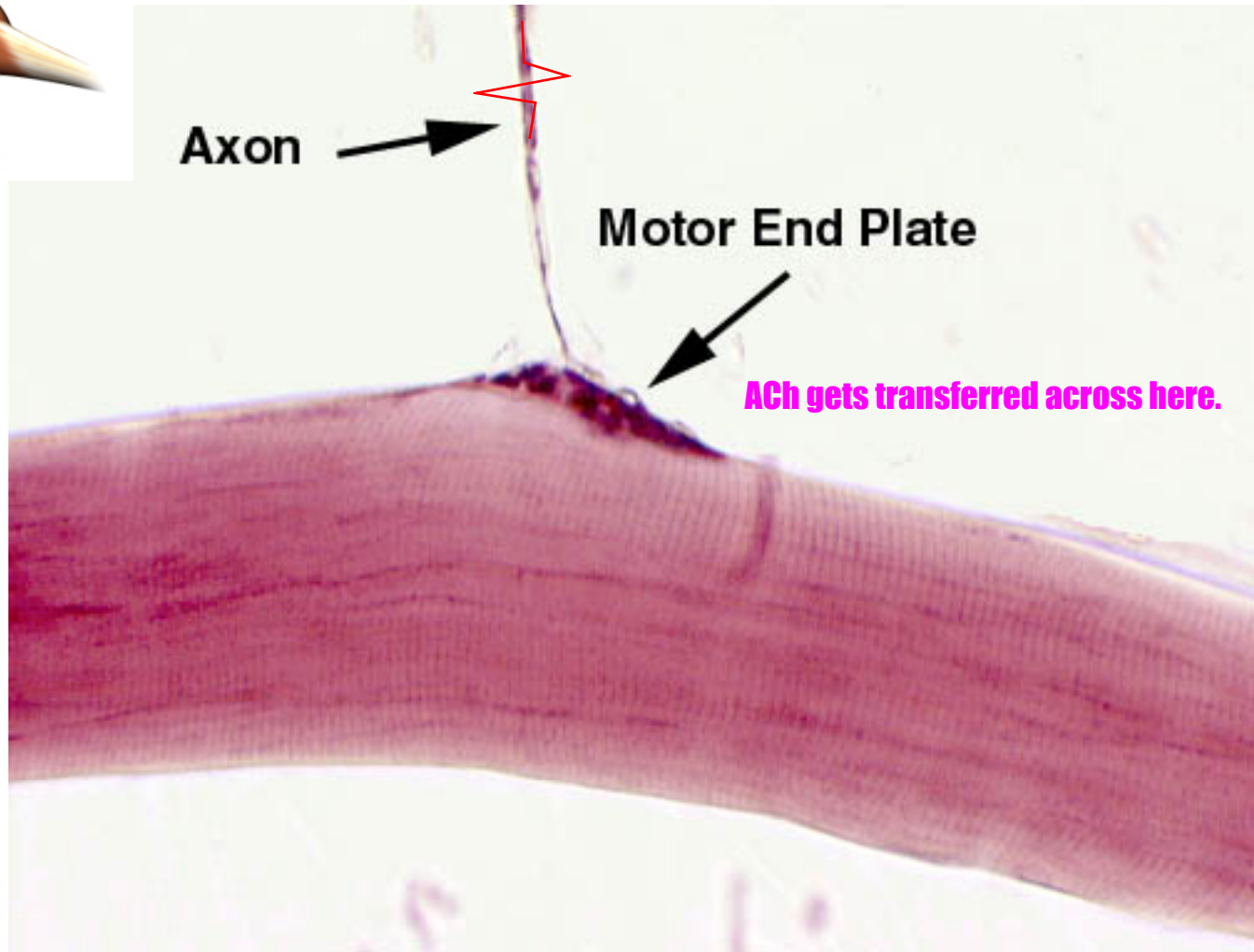
**Tetanus:** the usual manner of muscle contraction ..... volleys of impulses rather than single twitch impulses  
 = smooth continuous contraction

This is typically how we generate forces. An annoying twitch in your eye is an unfused tetanus situation.

Motor Unit



3D SCIENCE.COM



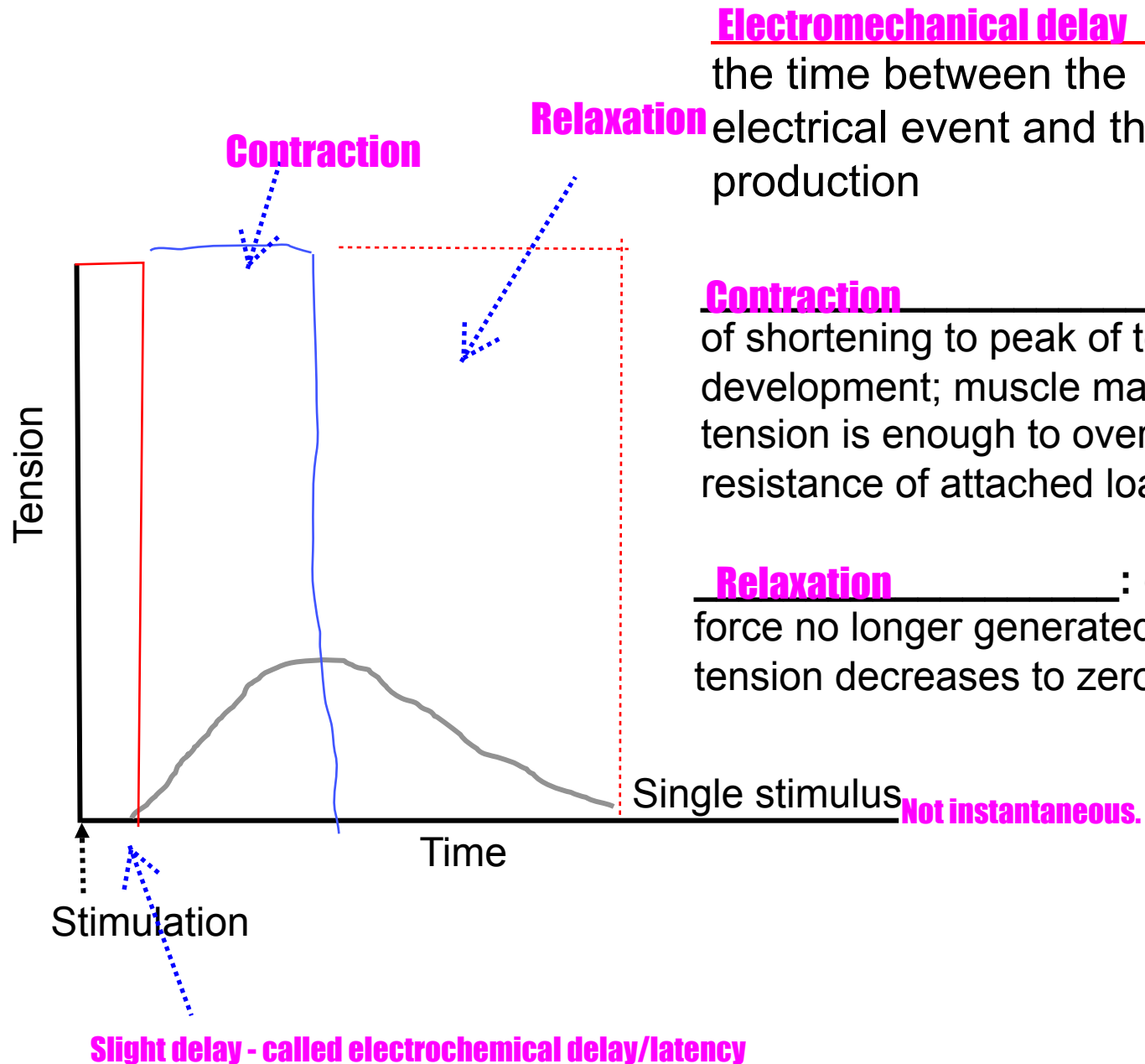
Axon

Motor End Plate

ACh gets transferred across here.

**Muscle Twitch:** response of a muscle to a single brief threshold stimulus

Twitch – **Smallest measurable force. Lower level of activation. Assume twitch does happen.**



**Electromechanical delay**

the time between the electrical event and the force production

**Contraction**: from onset of shortening to peak of tension development; muscle may shorten if tension is enough to overcome resistance of attached load

**Relaxation**: contractile force no longer generated; muscle tension decreases to zero.

Muscle Fiber Types

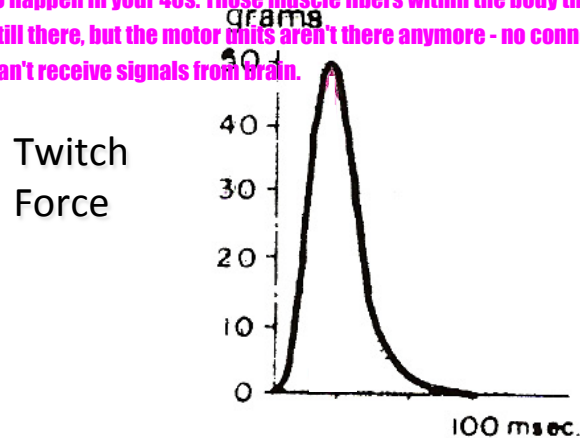
3 different types of MUSCLE FIBERS

**IMPORTANT:** All muscle fibers within a MU are of the same type!

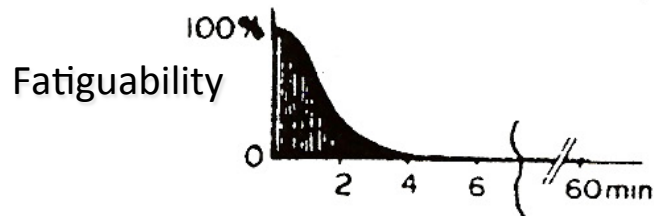
Time is different for each graph. The twitch force is quite small.

Fast twitch - glycolytic

As you get older, you tend to gravitate towards oxidative/glycolytic and oxidative activities. Our motor units as we get older start to die. Starts to happen in your 40s. Those muscle fibers within the body themselves are still there, but the motor units aren't there anymore - no connection from brain, can't receive signals from brain.

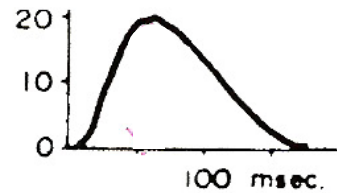


Crash pretty quickly. Can't sustain muscle contractions for more than 100 ms.

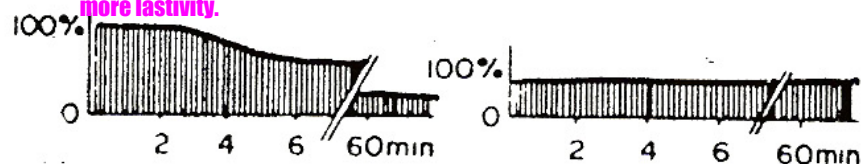


100 means well rested, well hydrated. Everyone has slightly different muscle morphologies. In this initial fiber type, you get high force production, and you fatigue very quickly. Glycogen and sugar are what supply this. At around 50%, this is where lactate acid starts to build up.

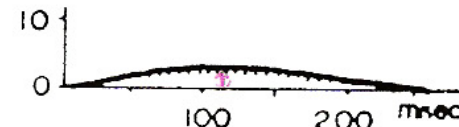
Fast twitch - oxidative/glycolytic



Hybrid between oxygen energy source and glycolytic energy source. Slope is a little less; max peak is about 20. Not nearly as powerful as muscle fiber but more lastivity.



Slow twitch - oxidative

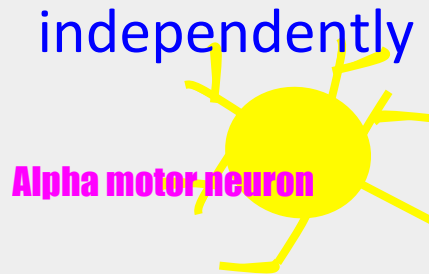


Just standing for x number of hours a day (like a cashier). Can last much longer, using a lot less energy. Collectively, our bodies' genetics determine your type of muscle fibers. Can train it but there is a ceiling effect that you'll hit.



## Motor Units

**Motor Unit** – functional unit of skeletal muscle  
 Smallest part of muscle that can contract  
 independently

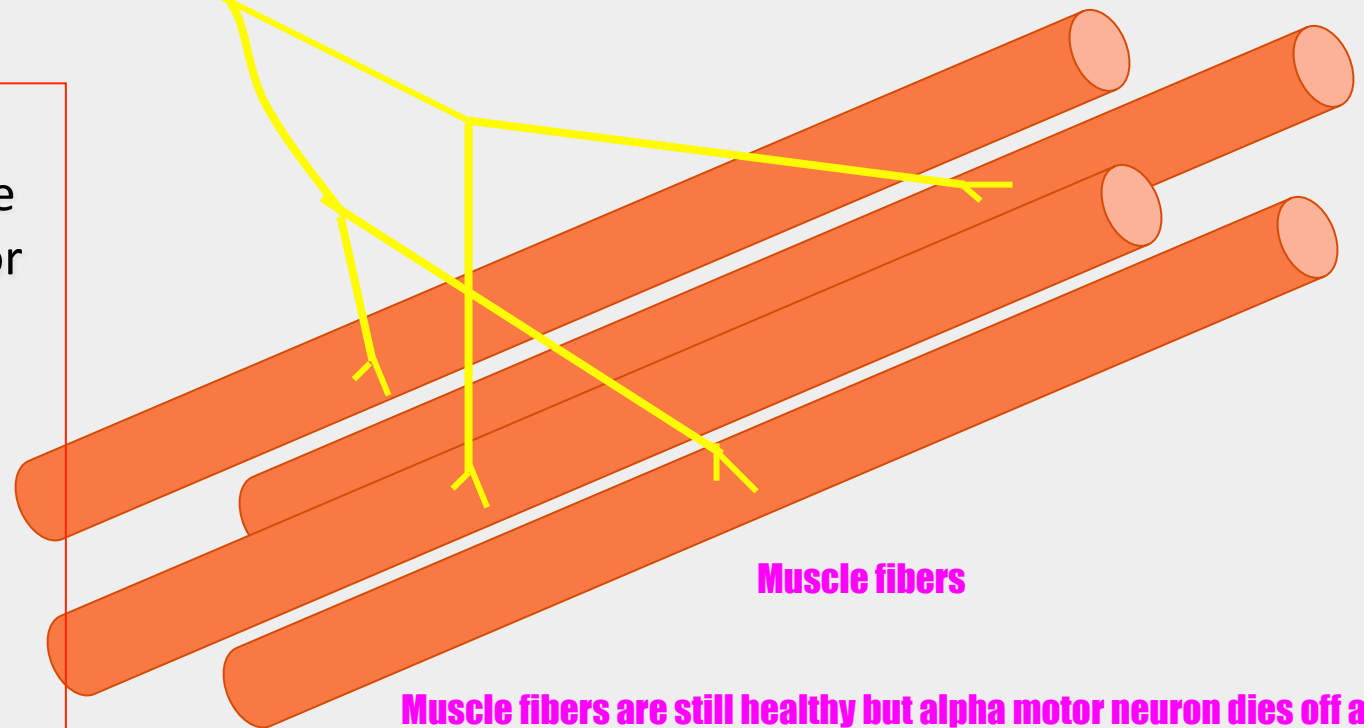
**MU includes:**

- 1.. **cell body of alpha-motor neuron in ventral horn of spinal cord (descending, efferent information)**
- 2.. **All muscle fibers innervated by its axon**

Remember: All muscle fibers are the same type in a motor unit

So...

When motor unit stimulated, ALL muscle fibers respond, “**All or None principle**”



**Muscle fibers are still healthy but alpha motor neuron dies off as you get older. Not sure why it happens but it does.**

## Motor Units

### All or None Principle:

Muscle fiber: contracts when ALL sarcomeres shorten simultaneously in “all-or-none” fashion

Motor Neuron: Cell body containing the nucleus of the nerve cell

Motor Unit: When motor neuron depolarizes, then all muscle fibers that it innervates depolarize and contract.

### Motor Unit Innervation Ratio:

The number of muscle fibers in a motor unit  
 Includes the alpha motor neuron, all the axons, as well  
 as the muscle fibers themselves.

Small # of fibers = Fine movement, control - as few as

3 muscle fibers! E.g. fingers

large # of fibers = Large movements, power - as

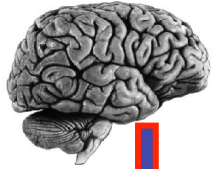
many as 2000 fibers!

Small number of fibers - can turn  
 some on and off.

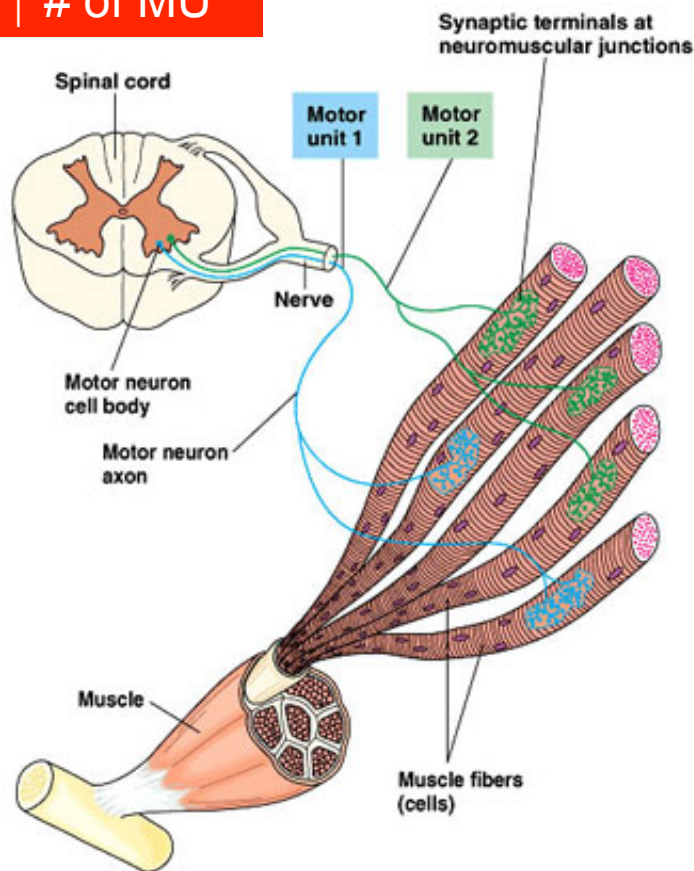
**If a motor unit is sending a pulse to contract, everything contracts at the same time. Lots of sarcomeres living inside just one muscle fiber, and can generate movement in such a controlled way. All muscles (big or small) work essentially the same way but the motor unit innervation ratio is different. Depending on where the muscle is, there is a different ratio.**

# Muscle Tension/Force and Recruitment

## Graded Muscle Responses



↑ # of MU



### 2 ways a muscle response can be graded:

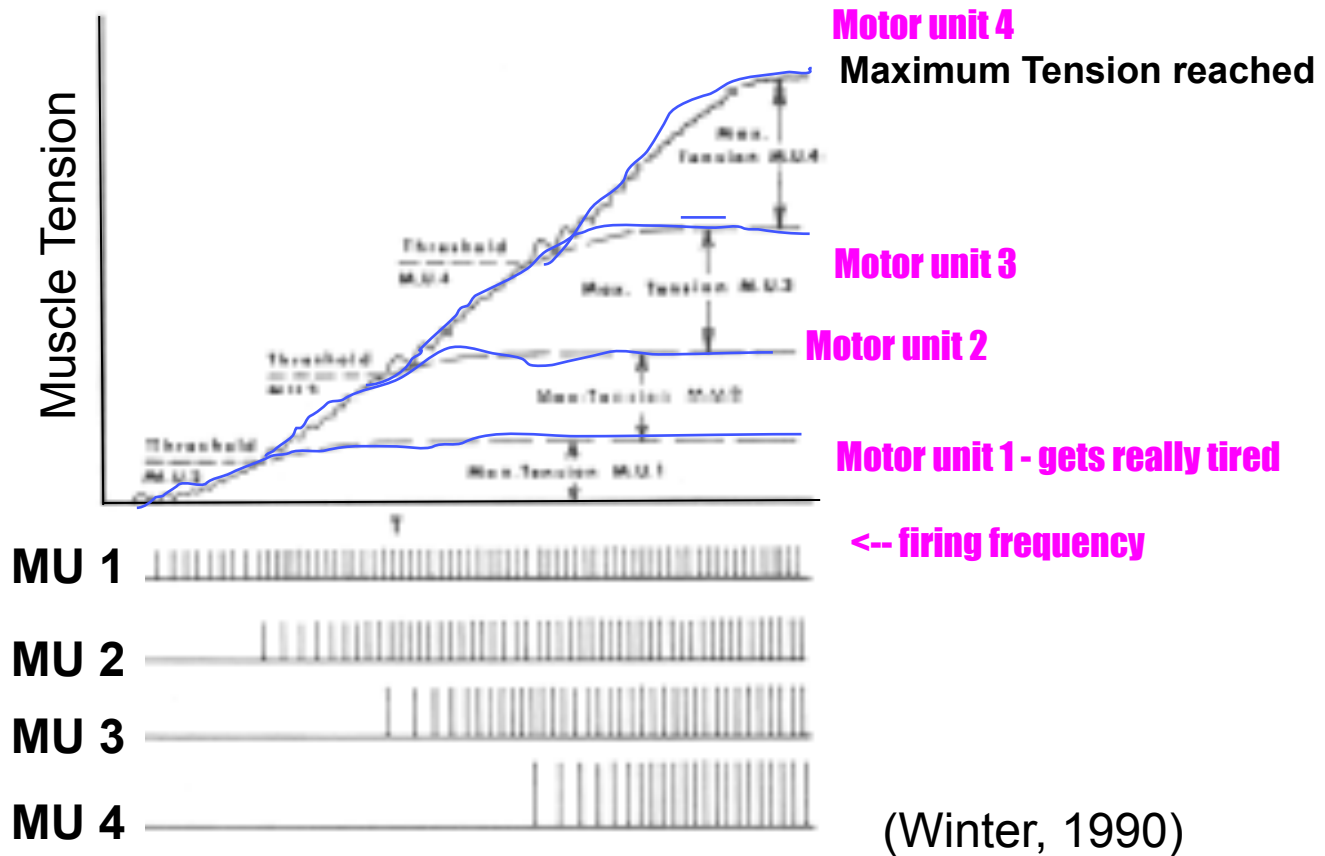
**1. Recruit more motor units to produce multiple motor unit summation. This diagram shows that alpha motor neurons that innervate different muscles. One innervates two, the other innervates three. Can do a small muscle contraction and just turn on motor unit 1 and get a small motor contraction. If need more force, can turn on motor unit 2. If even more force needed, can turn on both to gradually increase force.**

**2. Rate coding: CNS increase nerve stimulation: increase firing rate and therefore wave summation; also called the Cinderella principle; have a certain number of force needed and start with slowly working muscle fibers then increase rapidly.**

(Marieb, 1989)

# Muscle Tension/Force and Recruitment

## Graded Muscle Responses



x-axis: Time

Typically turn on slow twitch (oxidative) muscle fibers first because they can sustain muscle contractions longer (in this example, it would be motor unit 1)

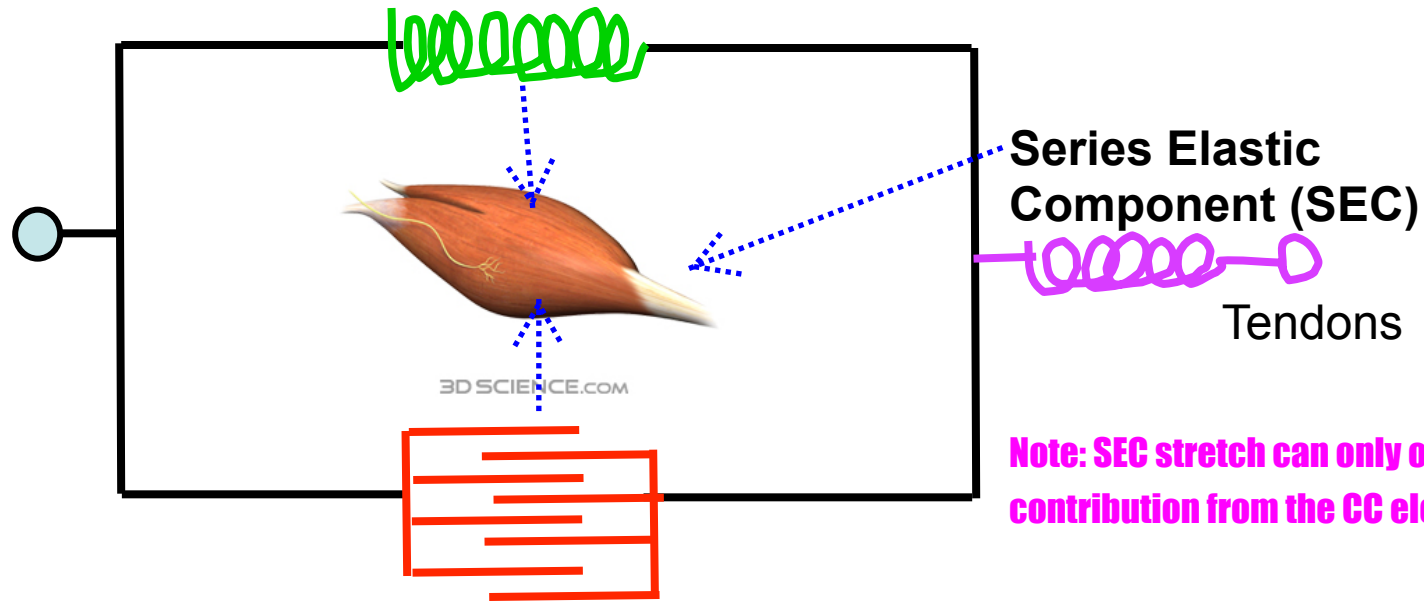
# Force – Length Relationship

## Schematic of Musculo-Tendinous Unit

**Role: Generate tension, independent of CC**

### Parallel Elastic Component (PEC)

Epimyosin, perimyosin, endomyosin...



### Series Elastic Component (SEC)

Tendons

**Note: SEC stretch can only occur if there is a contribution from the CC element**

### Contractile Component (CC)

Myofibrils: Actin/Myosin

**If it was a shortening, tendons would stretch - happens because they are arranged in series. Tendons stretch while muscles lengthen. PEC is like seran wrap that surrounds the muscle - generates tension independently. Muscles have shape and function, even if dead - small amount of force coming even post mortem.**

**Got lots of different passive tissue structures providing support for tissue itself. Got muscle anchor on either side by tendons. On one side, you'd have SEC. If you do not contract muscle, you have no change in series component (so it is like a series circuit). Active component where you can see the rachening happening.**

# Force – Length Relationship

Active Component of Muscle - CC element

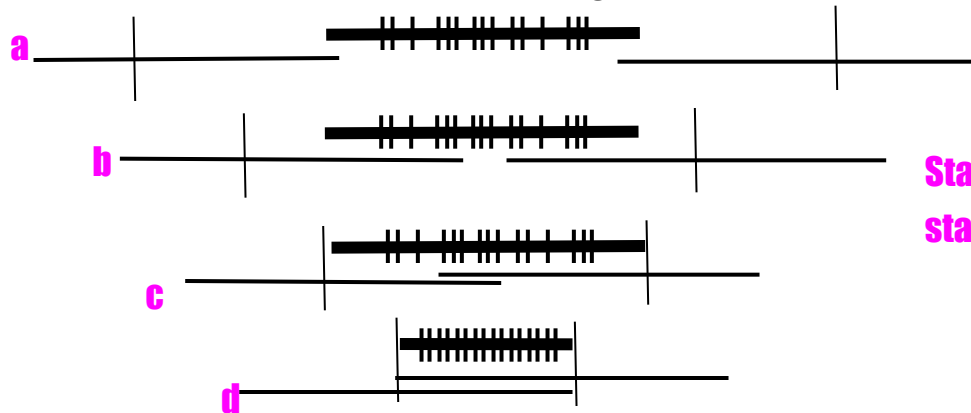
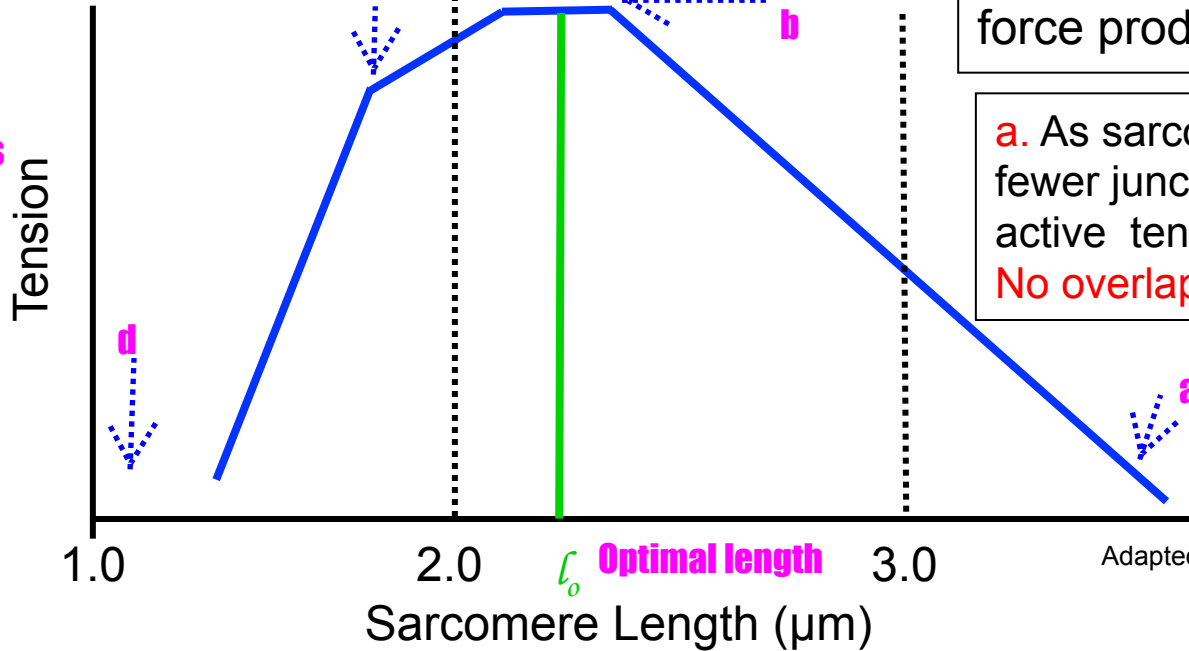
d. As sarcomere shortens less than resting length, ↓ tension because overlapping of thin filaments: **no force generation possible!**

**No overlap**  
No where else for myosin heads to connect.  
Short as the muscle can get.

There is an optimal length for force production =  $l_0$

a. As sarcomere lengthens, fewer junctions b/w filaments: active tension ↓  
**No overlap: no tension**

Backbone of muscle contractions. Sarcomeres, lengths determined by measuring z lines.



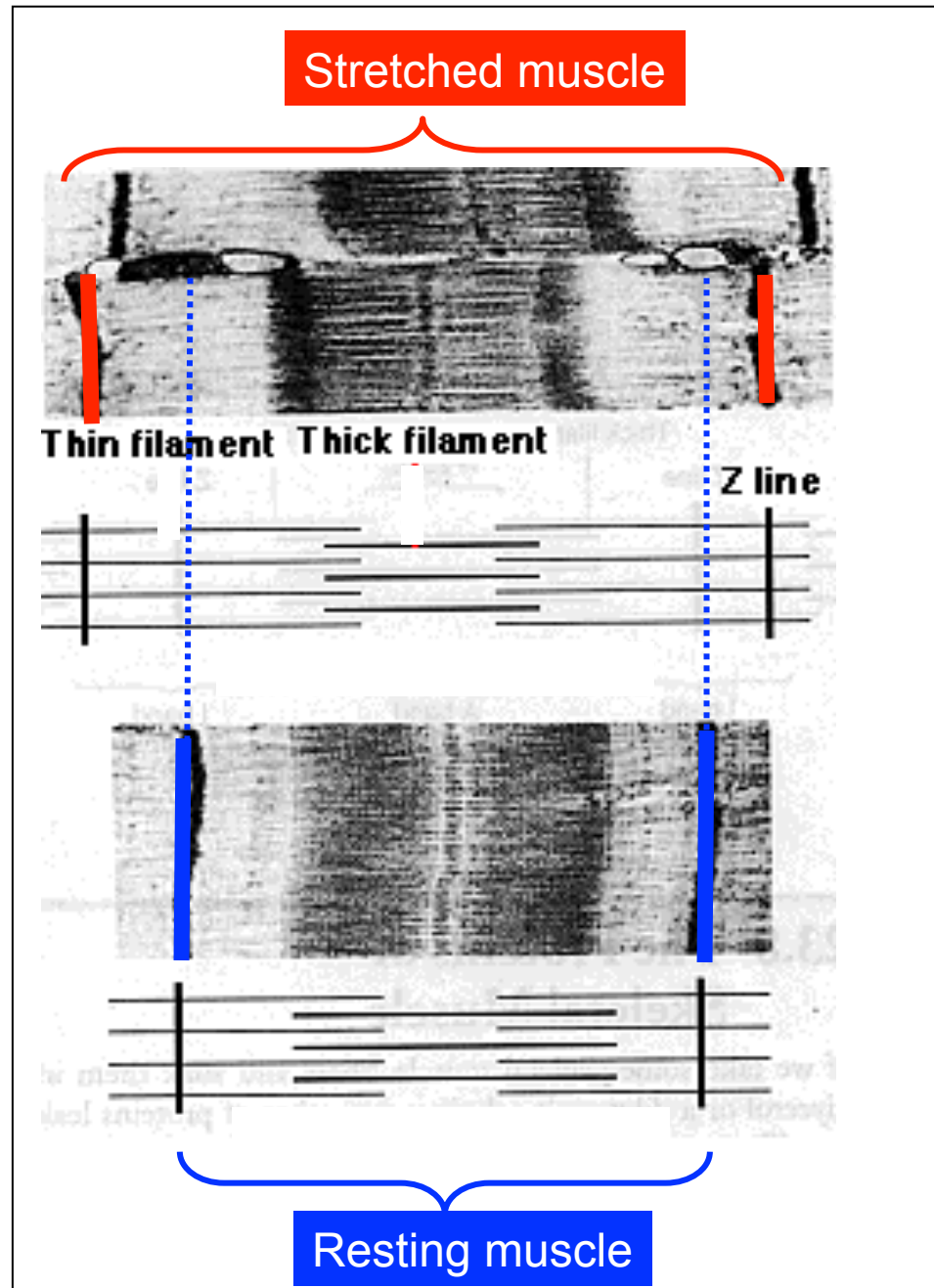
Start to see muscles lengthening but muscles starting to stretch farther away.

# Muscle Mechanics

## - Skeletal Muscle

### Sarcomere Lengthening

Eccentric component of contraction.



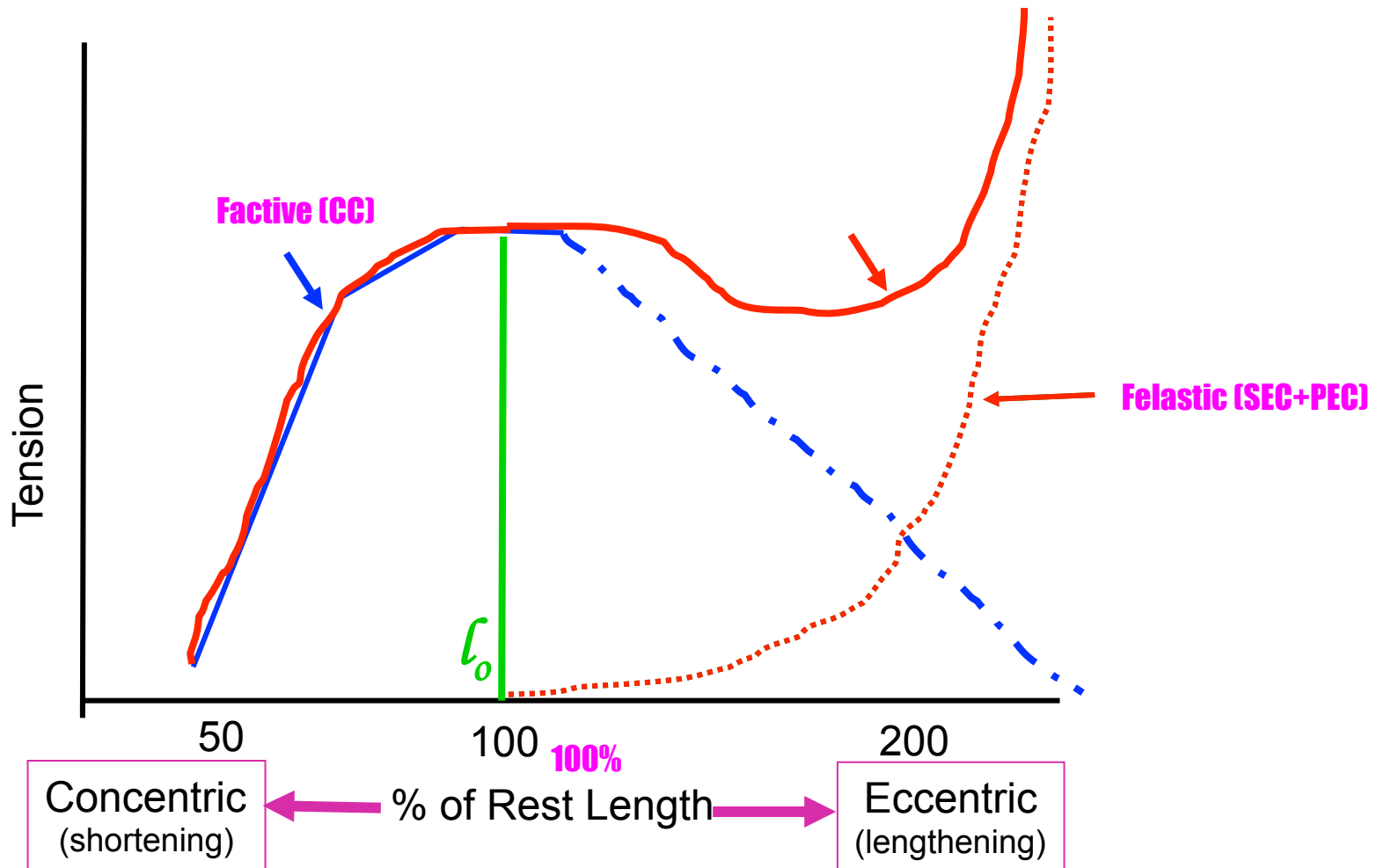
### Sarcomere Shortening

[http://highered.mcgraw-hill.com/olc/dl/120104/bio\\_b.swf](http://highered.mcgraw-hill.com/olc/dl/120104/bio_b.swf)

# Force – Length Relationship

WHOLE MUSCLE

This is what's happening to the elastic components.



Past resting length; tendons start to lengthen as we extend. As muscle lengthens, we increase force production. When you add up the contractile components plus the elastic components, get a slight dip then an increasing rise in force. At this point in time, need high force and range of motion, get hyperextension. Want high force production (eccentric are rate limiting - stop you from injuring yourself). Eccentric provides stability for the joints.

# Force – Velocity Relationship (A.V. Hill)

NOTE: This curve is for 100% activation

We are significantly stronger during eccentric action.

## Eccentric action

Force within muscles  $\uparrow$  with  $\uparrow$  velocity of lengthening

We are much stronger eccentrically. Summation of contractile and elastic.

### Why:

- Eccentric contractions require very little metabolic activity, yet are characterized by high force production!
- 1. takes less energy to break actin/ myosin bonds than to form them --> more energy available to produce force.
- 2. Elastic component increase their tension development
- 3. Force due to viscosity: same direction of application of direction!

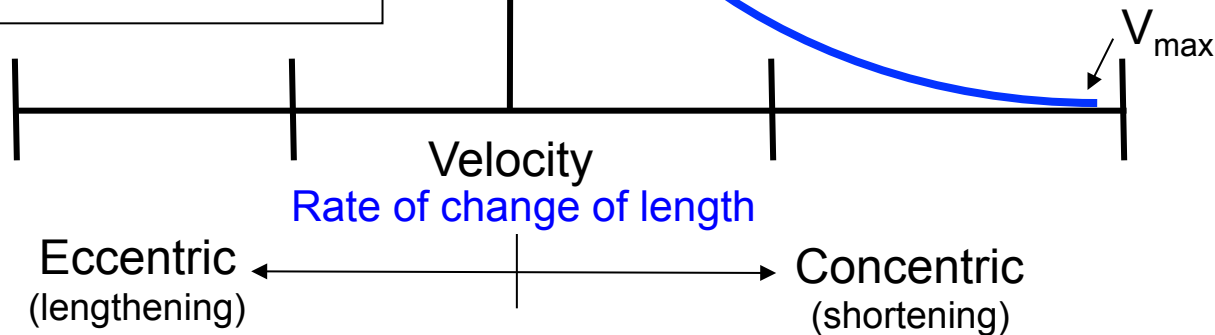
Force

**Isometric:** tension being generated but no shortening (think of flexing your muscles)

## Concentric action

Muscle force potential rapidly  $\downarrow$  with  $\uparrow$  velocity

**Why?** When flexed, no cross bridges for actin and myosin to happen. No elastic component being added to it. Limitation to # of cross bridges that can be formed when velocity increases



NOTE:

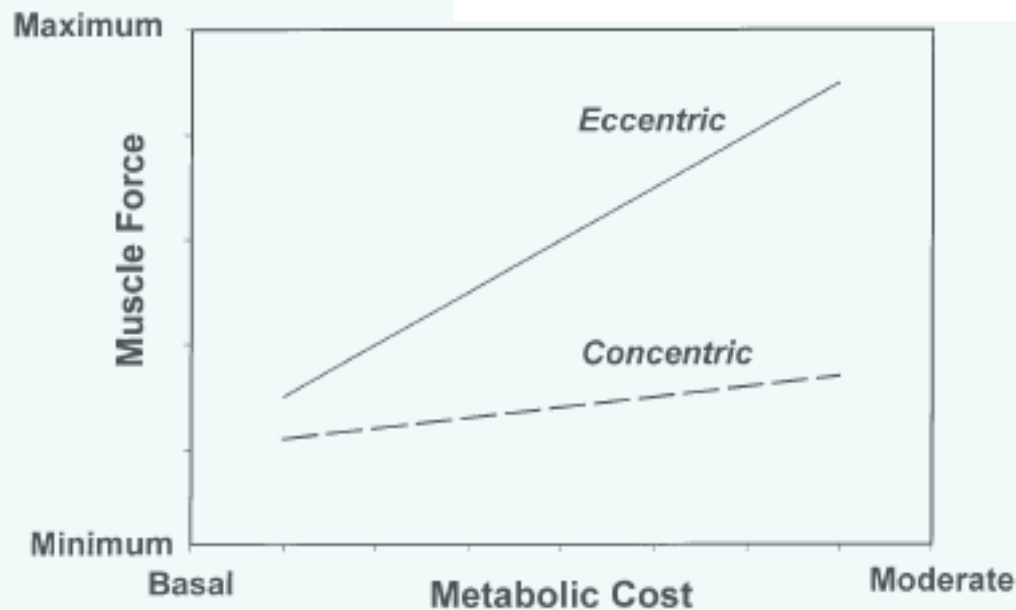
At the same velocity eccentric strength is greater than concentric strength.

# When Active Muscles Lengthen: Properties and Consequences of Eccentric Contractions

S. L. Lindstedt,<sup>1</sup> P. C. LaStayo,<sup>2</sup> and T. E. Reich<sup>1</sup>

<sup>1</sup>Physiology and Functional Morphology Group, Department of Biological Sciences, and <sup>2</sup>Department of Physical Therapy, Northern Arizona University, Flagstaff, Arizona 86011-5640

News Physiol. Sci. • Volume 16 • December 2001



**FIGURE 4.** Bigland-Ritchie (2) first plotted oxygen uptake against work showing 6 to 7 times reduction in metabolic cost to achieve same magnitude of work (negative vs. positive). If we reverse the axes, it is apparent that at identical exercise intensities (measured as oxygen uptake) much more force can be generated eccentrically than concentrically at moderate metabolic cost

Get way more force at moderate metabolic cost.

# **New research in this area- at the cellular level investigating the role of TITIN**

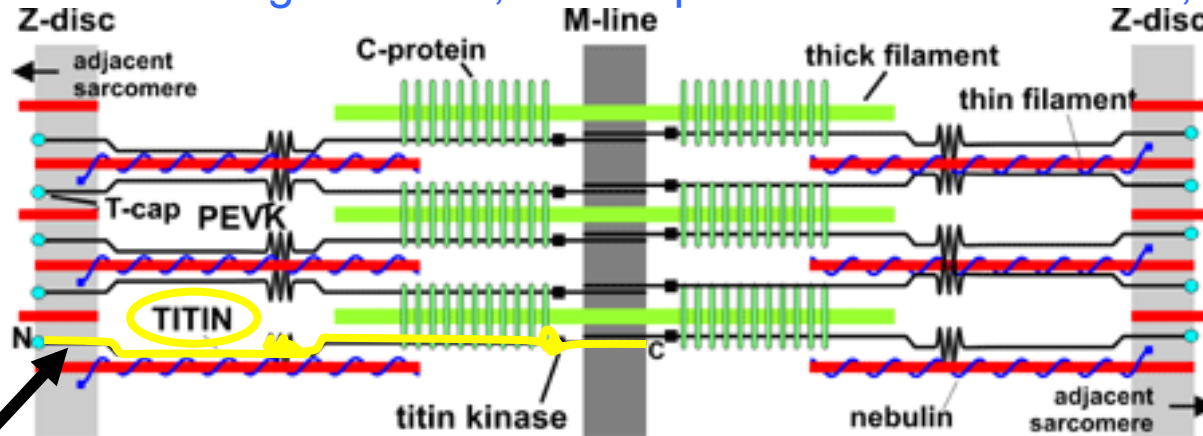
## **Eccentric Contractions—Muscle Actively Lengthening**

For example, the quadriceps (knee extensors) are active just after heel strike while the knee flexes.

The basic mechanics of eccentric contractions are still a source of debate since the [cross-bridge](#) theory that so nicely describes concentric contractions is not as successful in describing eccentric contractions.

## Schematic diagram of the sarcomere

(modified from Gregorio et al., *Curr. Opin. Cell. Biol.* 11: 18-25, 1999).



Titan lives within structure of sarcomere.  
Titan plays a role in elastic recoil storage.

- “Titin forms a continuous filament system along the muscle fiber in vertebrate striated muscle, overlapping in the M-line and in the Z-disc. Titin interacts with a plethora of sarcomeric proteins”.

Titin’s role in lengthening contractions may also include the initiation of cellular signaling to enhance cross-bridge recruitment while decreasing the cost. Hence, a differential expression of titin isoforms could alter the magnitude of elastic recoil storage and subsequent utilization as well as effect cross-bridge cycling and efficiency.

**When Active Muscles Lengthen: Properties and Consequences of Eccentric Contractions**

S. L. Lindstedt,<sup>1</sup> P. C. LaStayo,<sup>2</sup> and T. E. Reich<sup>1</sup>

<sup>1</sup>Physiology and Functional Morphology Group, Department of Biological Sciences, and <sup>2</sup>Department of Physical Therapy, Northern Arizona University, Flagstaff, Arizona 86011-5100

# Mechanoenzymatics of titin kinase

PNAS | September 9, 2008 | vol. 105 | no. 36 | 13385–13390

Elias M. Puchner<sup>†</sup>, Alexander Alexandrovich<sup>‡</sup>, Ay Lin Kho<sup>‡</sup>, Ulf Hensen<sup>§</sup>, Lars V. Schäfer<sup>§</sup>, Birgit Brandmeier<sup>‡</sup>, Frauke Gräter<sup>§¶</sup>, Helmut Grubmüller<sup>§</sup>, Hermann E. Gaub<sup>†</sup>, and Mathias Gautel<sup>‡¶</sup>

<sup>†</sup>Chair for Applied Physics, Center for Integrated Protein Science Munich and Center for Nanoscience, Ludwig-Maximilians-Universität München, 80799 Munich, Germany; <sup>‡</sup>Cardiovascular Division and Randall Division for Cell and Molecular Biophysics, King's College London, London SE1 1UL, United Kingdom; and <sup>§</sup>Department of Theoretical and Computational Biophysics, Max Planck Institute for Biophysical Chemistry, 37077 Göttingen, Germany

Edited by Gregory A. Petsko, Brandeis University, Waltham, MA, and approved July 14, 2008 (received for review May 23, 2008)

**Takes less energy to break bonds but also get recoil**

The 3D animation shows a zoom inside the muscle up to the smallest protein building blocks to visualize how contraction is generated by myosin and actin filaments. In their center, these motor strands are connected and aligned through the elastic titin-network, which contains the molecular force sensor titin kinase.

<http://www.youtube.com/watch?v=HROJU0X1YdQ&feature=related>

# ...Consider Force and viscosity of a visco-elastic material

## In Concentric contractions

Muscle force is in opposite direction ;  
CANCELS => less force for a given velocity

Muscle force is in opposite direction of  $F_{\text{viscosity}}$ , so because of this, can actually cancel out viscosity component.

**And !**

## In Eccentric contraction

Muscle force is in same direction ;  
SUMMATES = greater force, same velocity!

In same direction, so can add up the two forces. Summate the muscle contraction itself and the force of viscosity - can generate higher force with the same velocity.

