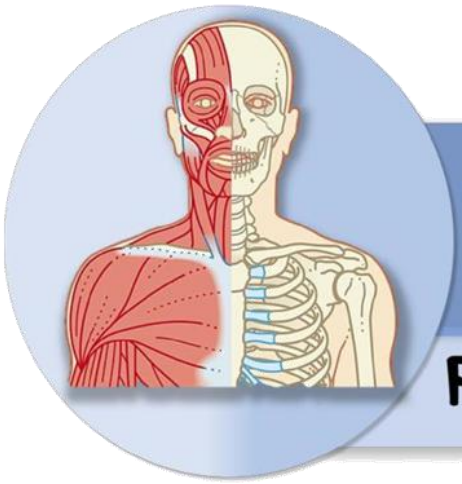

MSS system

Physiology

◉ Sheet ○ Slide

Number: 2

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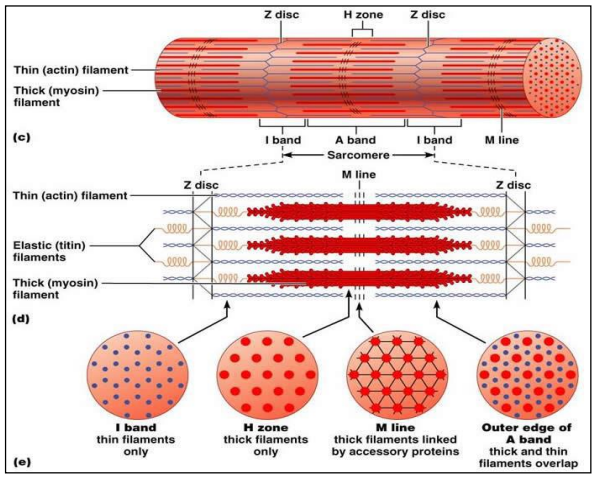
first topic of this sheet is about the structure of actin and myosin filaments after taking a cross section from a myofibril.

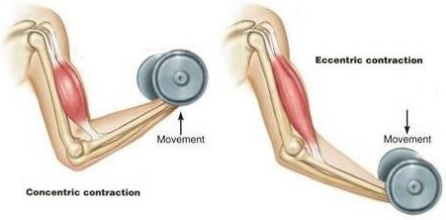


As we see in the figure the picture under the microscope depends on the place in the sarcomere we are taking the cross section from; if we are taking it from the M disc (M line) we have only thick filaments and their linker proteins visible under the microscope , but if it was taken from the edges of the A Band where we have both thick and thin filaments overlap we will be seeing that each thick myosin filament is surrounded by six thin actin filaments AND each thin filament is surrounded by three thick filaments concluding that the ratio between thin filaments to thick one's is 2:1

We know that the interaction between thin and thick filaments "contraction" is shortening **BUT can we have contraction in the muscle without shortening the muscle ?** ***Yes*** and this is known as ***isometric contraction***; length is not changed "by fixing the head of the myofibril" but the tension in that muscle is changed.

Important definitions:

-tension is produced when cross-bridges form between the actin and myosin filaments within each **muscle** fiber.



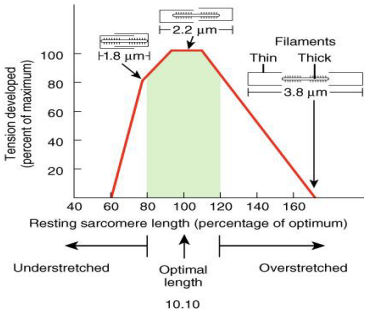
-isotonic contractions generate force by changing the length of the muscle and maintains constant tension ;it can be **concentric contractions** or **eccentric contractions**;

-**concentric contraction** causes muscles to shorten.

-eccentric contraction is the motion of an active muscle while it is lengthening under load.

NOW we are going to discuss the figure below; which is about manipulating the length of the sarcomere, fixing the heads and stimulating it by simply adding ca++ ions to calculate the tension

First imagine we **stretched** the heads of the sarcomere till we have no overlap between the fibrils "**at 3.8 nm**" (length of one thick filaments and 2 thin filaments) and then **stimulating** the sarcomere will result in *no tension*.

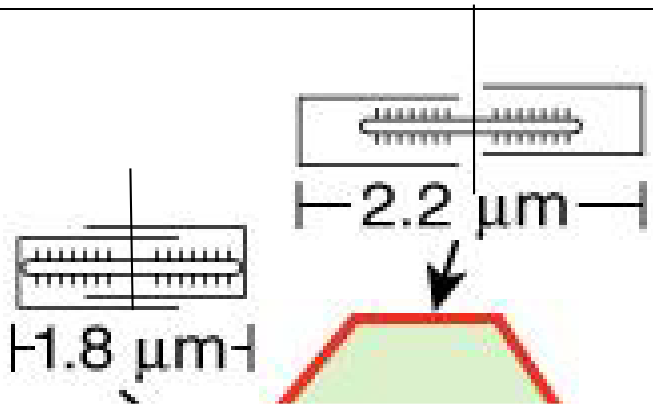


By **decreasing** the length we will start to have some overlapping and the heads of myosin will start to attach thin filaments and tension is recorded . till we reach the **optimal (resting) length "2.2 nm"** where maximum active tension is recorded.

Now imagine you contracted the fibril then fixed it at 1.8 nm then stimulated it again . in this situation you have some overlapping of the thin filaments on one side(half) with the thick filaments of the

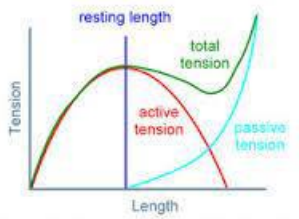
other side " I divided the sarcomere into two half's in this figure to help you understand how two opposite forces will occur on one thin filament " SO tension is decreased

Note : we are using specific transducers to record the tension



Takeaways : **ratio between thin to thick filaments is 2:1 , *isometric contraction*, tension, isotonic contractions , eccentric contractions; concentric contraction , at 3.8 nm no overlapping no tension , at 2.2nm optimal length (where thin filaments is overlapping with half of the thick one's) and max active tension , at 1.8nm overlapping with other half of the thick filaments occur and decreased tension.**

NOW taking the whole muscle or muscle fiber ,it was found that stretching the muscle without stimulating it will result in tension called passive tension(without stimulation) BUT stimulating a stretched muscle beyond 2.2nm will result in less active tension (tension due to stimulation) as we mentioned before. So the total tension increases by stretching but the active tension deacreses.



Length-Tension Curve of a Muscle

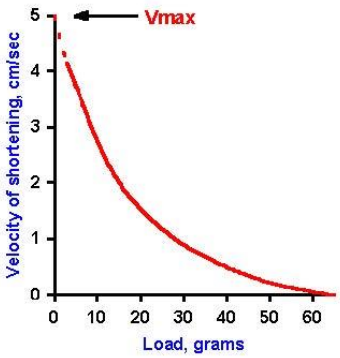
We can calculate the active tension by this equation : (ACTIVE T.=Total T. – Passive T.)
passive tension is DUE TO **the elastic property of the muscles.

Note : stretching a muscle leads to more powerful shortening of it when stimulated

Note: Passive tension is due to the structural component of the myofibrils like “titin proteins”. An example of this would be the pull one feels in their hamstrings while touching their toes

Velocity of contraction depends on the **load** we are producing to the muscle; Skeletal muscle contracts with maximum velocity when it is *not loaded*. By loading the muscle, the velocity of contraction decreases as the load increases till we reach no shortening state but still we have interactions and tension recorded which is called **isometric contraction**

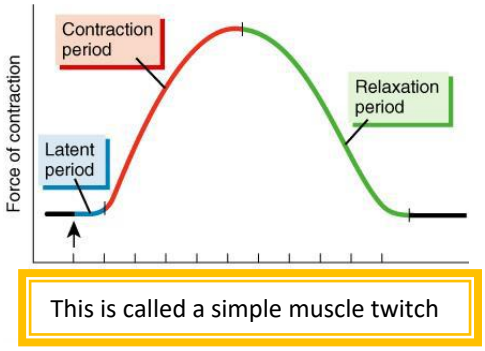
-if you have no load then highest velocity will be recorded but almost no changing in the tension in that muscle so we are having what is called **isotonic contraction**.



Note : we have combination of isotonic and isometric contractions in our body " shortening and changing the tension "

Type of muscle fiber	Slow fibers "soleus muscle"	Fast fibers " ocular muscle"
size	small	large
color	Red" more blood supply and myoglobin"	White" less blood supply"
Sarcoplasmic reticulum	Less extensive	More extensive
composition	More mitochondria , more myoglobin	More glycolytic enzymes, less myoglobin
Energy supplement	By oxidative phosphorylation	By glycolysis

CAN you report the process of isotonic contraction ?
The answer is yes , we have devices that can record the process of contraction and relaxation , or by having a nerve-muscle preparation and a recording drum "a device that measures speed of contraction" we can calculate the time needed for a contraction as **(speed= distance/time)** to get the following graph :
the stylus of the device will raise due to contraction then it will go down by relaxation to get that graph.

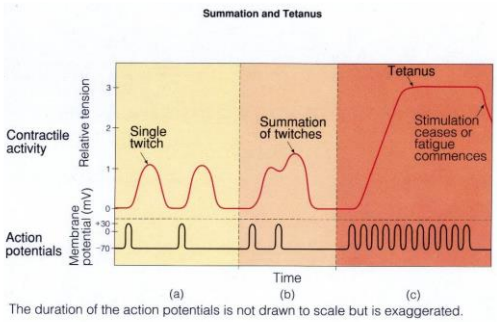


-in the latent period we stimulated the muscle but no change in length; time during which impulse is traveling along sarcolemma & down t-tubules to sarcoplasmic reticulum, calcium is being released. ***"stimulation occurs in the latent period always"***

-And in the contraction period calcium concentration is increased
So any action before the increase in calcium concentration is in the latent period

-In the relaxation period: Powered by ATP, it pumps **calcium** ions back into the sarcoplasmic reticulum, reducing the **calcium** level around the actin and myosin filaments and allowing the **muscle to relax.**

NOW if you stimulated the muscle twice " co stimulation " as in the graph the second stimulus can take place in the **latent period** "KEEP IN MIND THAT THE SECOND STIMULUS (ACTION POTENTIAL) (IN THE NEUROMUSCULAR JUNCTION) TAKES PLACE AFTER THE FIRST ONE HAS FINISHED AND THE MEMBRANE IS AT RESTING POTENTIAL"

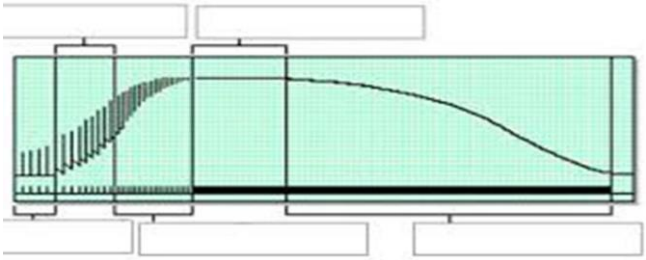


IMPORTANT NOTE : Contractile activity (the upper part of the graph):happens inside the muscle fiber.
Action potential (the lower part of the graph) : happens in the neuromuscular junction.

-if you stimulated the muscle 2 times one after another before the first twitch is ended you will have summation of twitches before the muscle is relaxed which is called wave or frequency summation .

-**if you are having continuous stimuli then contractile activity is increased without any relaxation which know as tetanization**

Now we have increased the frequency to get the wave summation and higher amplitude "shortening" of contraction is achieved by which is called **staircase effect or treppe phenomenon.**



Staircase effect (Treppe): When a muscle contracts after a period of rest, the simple muscle twitch has certain amplitude. After several contractions approximately 10, the amplitude of simple muscle twitches " the amplitude of shortening " **increases**. This is known as Treppe or staircase effect. This effect is probably due to an increase in Ca++ concentration inside the cytosol with each muscle stimulation and inability of sarcoplasmic reticulum to recapture Ca++ immediately.at higher concentration of calcium you have higher probability of the interaction with troponin c and higher probability of sufficient contraction, till we have **tetanization**.***it happens in our body and that the concept of warm ups before exercising.***

Treppe is not a summation…… Treppe is not a summation……. Treppe is not a summation

The concentration of calcium in the sarcoplasm at **resting** without stimulation is about 10^-7 molars and once you have stimulated the preparation the concentration is increased to 10^-3 molars this is an 10000 folds increase " NUMBERS ARE NOT FOR MEMORIZATION"
What's in the handout is : (Ca++ concentration in sarcoplasmic reticulum = 10^-3molar ,in the sarcoplasm during rest = 10^-7 molar, and during excitation of muscle = 2X10^-4molar)
Now assume you continued with stimulation without relaxation your muscles will suffer a **fatigue** " the decrease in the graph after tetanization" which Is characterized by the muscle **not responding** to the stimulus, and it is caused by depletion of ACH neurotransmitters in the presynaptic membrane (neuron) in the neuromuscular junction .

Note: 1- if you stimulated the muscle directly by calcium it will respond and contract.
2-most of the fatigues are caused by Ach depletion but some other conditions called also fatigue caused by lactic acid accumulation in which the muscle can still contract but with pain caused by the low PH. " not a real fatigue because the muscle is still responding"
3- in the fatigue phase we are not having direct relaxation it's like the tetanization but with a lower contraction power till we reach the relaxation again.

Takeaways: maximum velocity of contraction is reached when the muscle is not loaded "isotonic" and minimum with high load "isometric",,, slow and fast fibers ,,,, latent ,contraction and relaxing periods ,,,,, co-stimulation, tetanization and fatigue.

Are all muscle fibers innervated by one neuron ? **NO .**
Is each muscle fiber(cell) innervated by one neuron ? usually **YES** (except for about 2% of the fibers) but not each neuron innervates one muscle fiber only.

SO what happens is that **one motor neuron** having many **terminals** , each **terminal** will end in **one muscle** fiber only , so you will find a **group** of many muscle fibers innervated by one **motor neuron** and other group by other motor neuron.
The group of muscle fibers innervated by the same neuron is called **motor unit**

SO when a specific neuron is stimulated only it's motor unit will be stimulated not all the muscle.

When the number of nerve fibers stimulated increases, this will recruit more motor units in contraction. The increase in contraction will result in an increase in the amplitude of simple muscle twitch which is called motor unit summation . In human body this summation is important for gradation of forces during contraction.

For **example**: holding a paper needs the stimulation of one motor unit but gym weight lifting needs motor unit summation to get high amplitude of contraction.

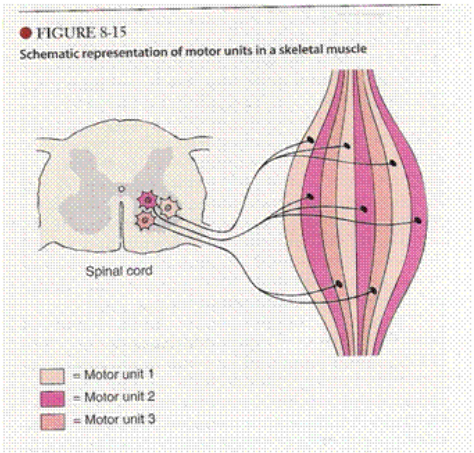


Figure 4. One motor unit can control multiple muscle cells.
Taken from Sherwook, 2004

THE QUSTION FOR THE NEXT LECTURE IS :
ARE ALL MUSCLES HAVING THE SAME NUMBER OF MUSCLE FIBERS FORMING ONE MOTOR UNIT?

DON'T HESITATE TO ASK...THANK YOU