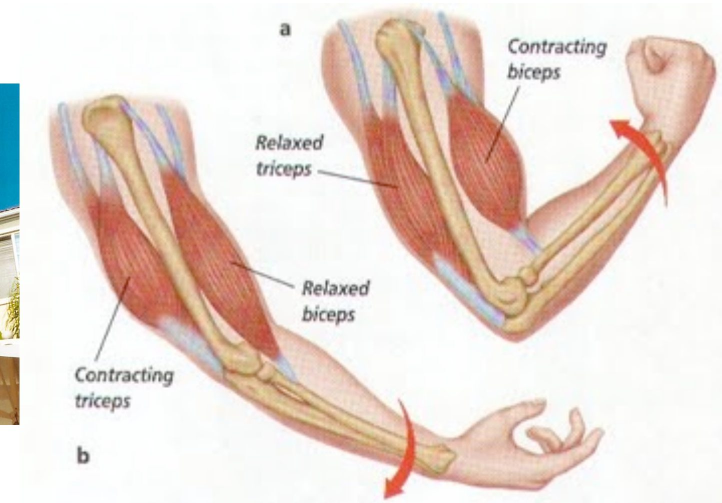
Biomechanics of the musculoskeletal system

Prof. Dominique Pioletti
Laboratory of Biomechanical Orthopedics
EPFL

Muscle biomechanics

- How do muscles work?
    - Muscles functional anatomy
    - Modes of muscle function
    - Structure and molecular functioning of the active part of the muscle
- How can we measure muscle activity and mechanical properties
    - Force-EMG relationship
    - Passive and active force-elongation curves
    - Force-velocity curve
- How can we incorporate muscle function in biomechanics
    - Spring and active elements
    - Force/cross-sectional relationship

The muscles are the engines of the MSS

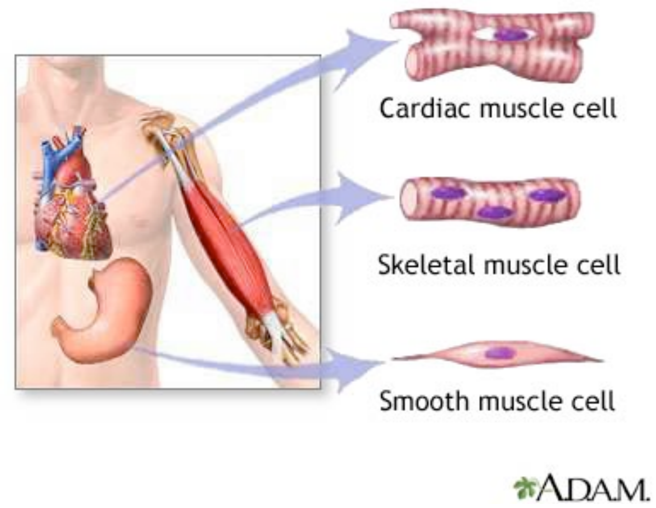


source: Bartel et al, Orthopaedic Biomechanics, 2006

The only way we can move, push things is through the action of muscles which are really the engine of our body. The flexion and extension of the elbow are presented and we can observed that the biceps (flexor muscle) and triceps (extensor muscle) are antagonist muscles: when one is contracting, the other is relaxing. Despite generating motion, muscles also allow to adjust the stiffness at the joints. Skeletal muscle accounts for approximately 40% of the body weight and is the largest organ of the body.

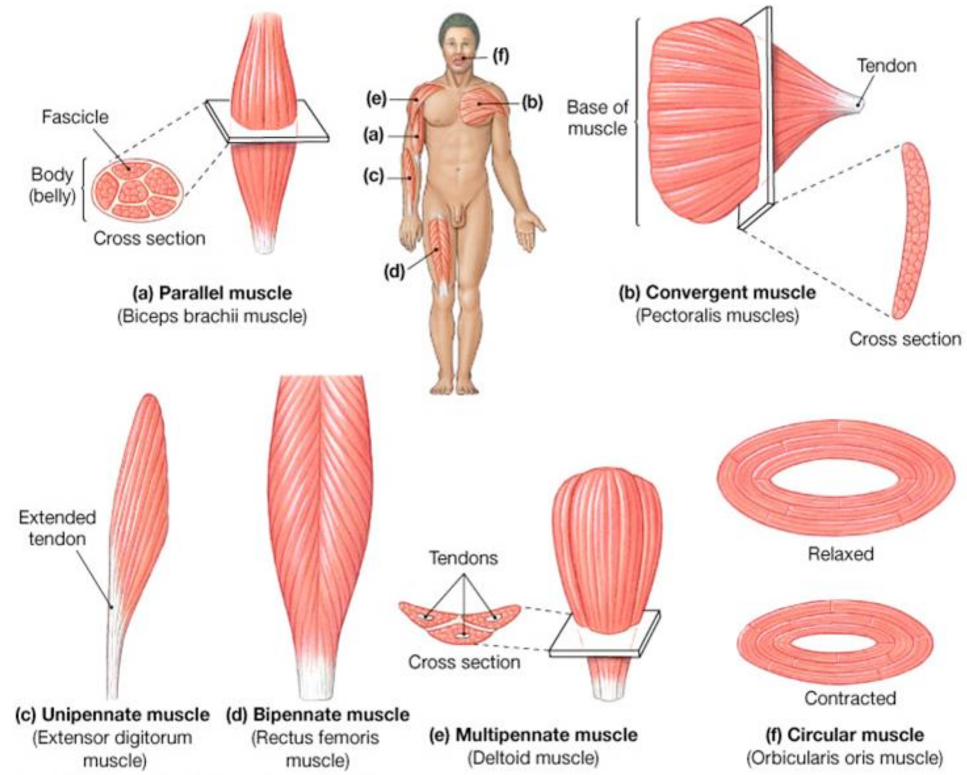
From a biological point of view, muscles can be seen as a machine converting chemical energy obtained from food substrate and oxygen into mechanical work and heat.

In our body, we have three types of muscles,



There are approximately 700 different muscles in the human body, divided into 3 different types: skeletal, cardiac, and smooth or visceral muscles. Smooth muscles (which control is mostly involuntary) are typically found surrounding the lumen of tubes within the body such as blood vessels, urinary tract, and gastrointestinal track. Cardiac muscle is also under involuntary control. Skeletal muscles (36% total body weight in women and 42% in men) are attached to bones via tendons and are usually under voluntary control.

different shapes of skeletal muscles,



To fulfil all their different functions for the locomotion or force transmission, the skeletal muscles present different shapes and accordingly different cross-sections too. The shape and arrangement of the fascicles are directly related to the direction of pull and strength of the muscle:

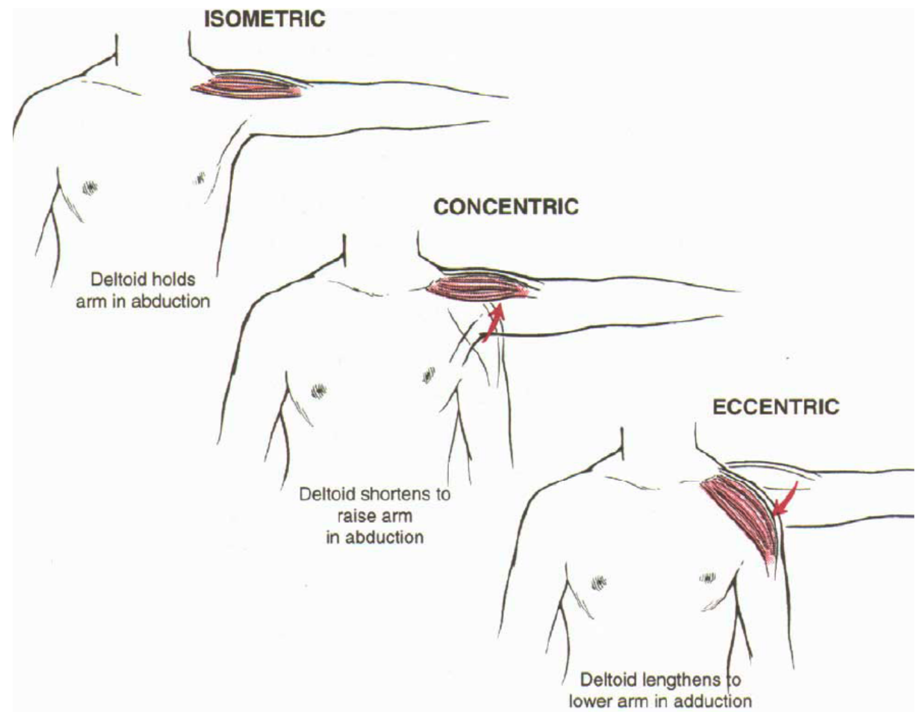
- parallel: fascicles parallel to long axis of muscle;
- convergent: fascicles spread over a larger area converge to a single tendon;
- unipennate: all fascicles converge to one side of a tendon;
- bipennate: fascicles converge to both sides of a tendon;
- multipennate: tendon branches converge to a single tendon;
- circular: concentrically arranged fibres surrounding an opening.

as well as different types of muscle fibres.

- Slow twitch (Type I)
- Fast twitch (Type II)

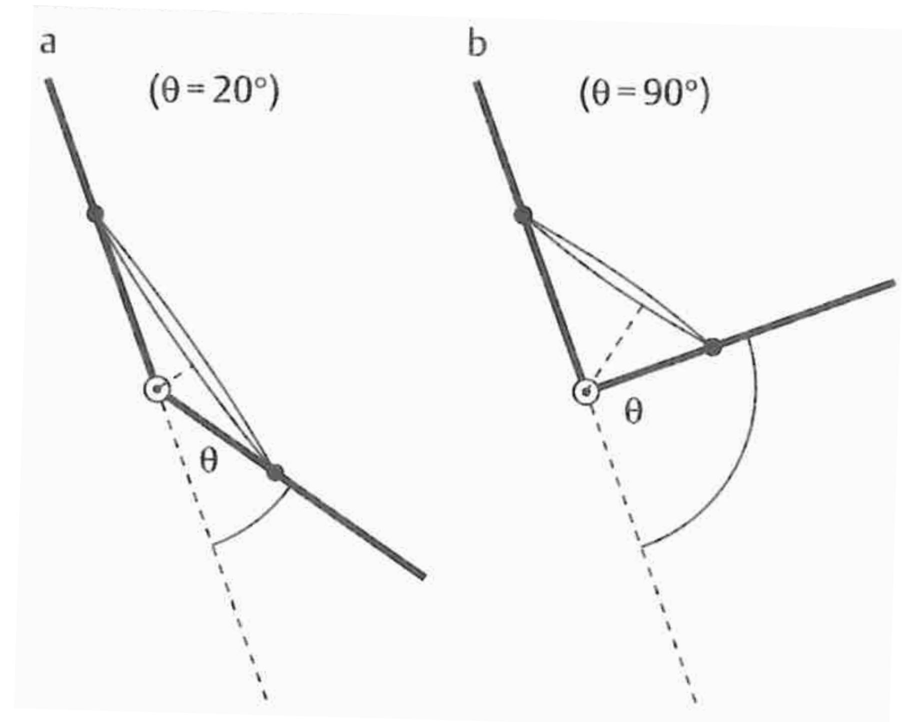
In our muscles, we have varying percentages of fast twitch and slow twitch muscle fibres (in gastrocnemius muscle: higher preponderance of fast twitch fibres -> rapid contraction used in jumping; in soleus muscle: higher preponderance of slow twitch fibres -> greater extent for prolonged lower leg muscle activity). Slow twitch fibres, as the name suggests, contract more slowly than fast twitch muscle fibres and they can contract for a longer period of time. We often call them 'endurance' muscles because they're great for aerobic activities that require endurance like long distance running, cycling and swimming. Fast twitch muscle fibres are activated during short bursts of speed and strength such as in sprinting or bodybuilding.

The muscles can work under different modes



The muscles can work under three different modes:
- Isometric: muscle contraction in which there is little or no change in fibres length -> used to maintain positions against the force of gravity or other resistances;
- Concentric: muscle contraction in which muscle fibres get shorter -> used to move body or body segments against gravity or other resistances;
- Eccentric: muscle contraction in which muscle fibres get longer -> used to control movement due to gravity or other external forces.

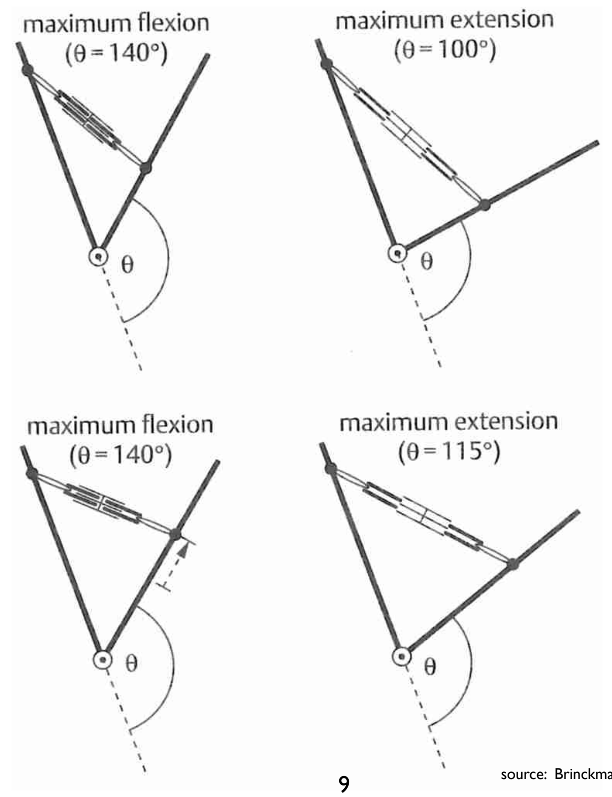
Finally, there is a macroscopical way of playing with the forces, through variation of the arm lever



source: Brinckmann et al, Musculoskeletal biomechanics, Thieme, 2002

During flexion of the joint, the arm lever of the muscular force varies. If we need to maximise the muscular force to lift an heavy object, it would be more effective to start the motion with the joint having a 90° flexion as shown on the example given here.

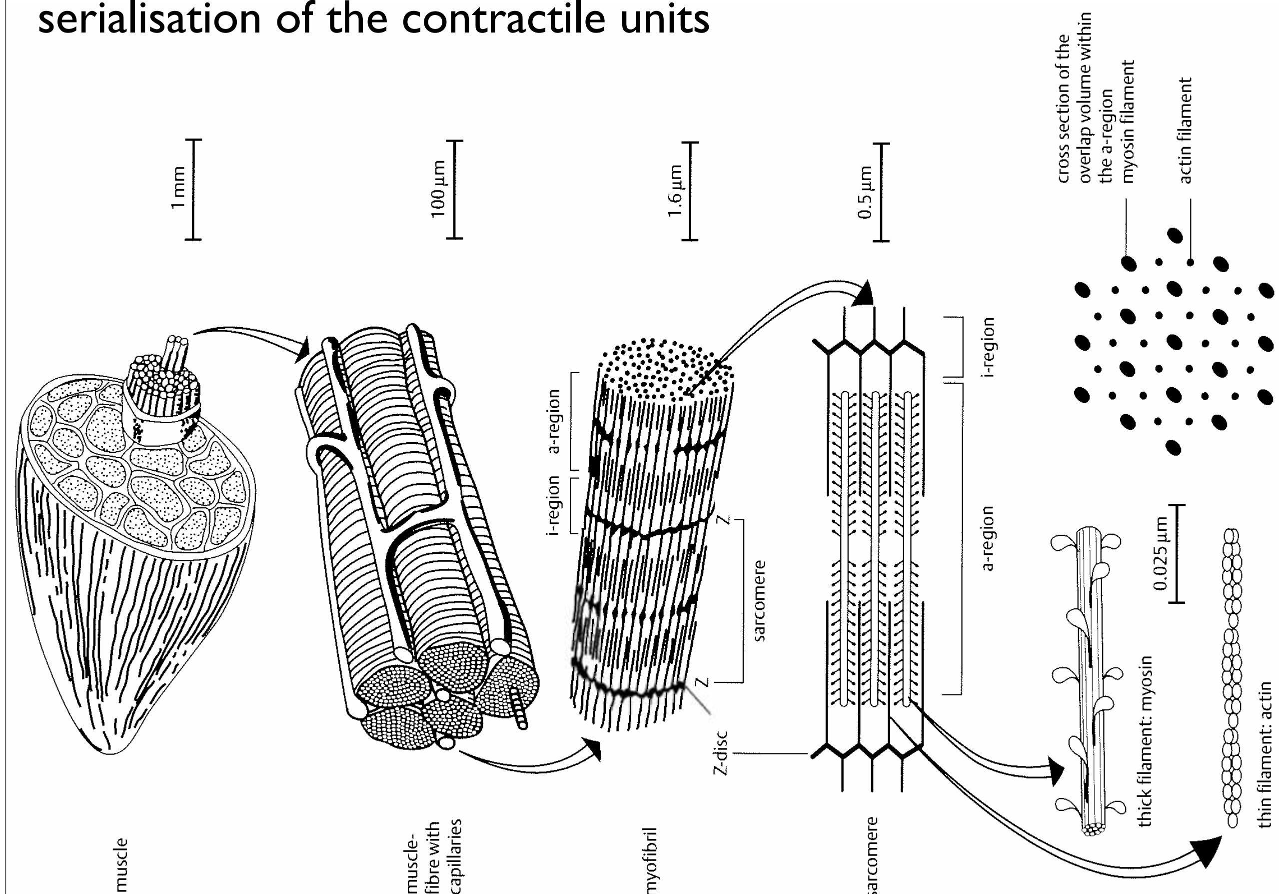
By the way, the attachment site of the tendon influences the range of motion of the joint



source: Brinckmann et al, Musculoskeletal biomechanics, Thieme, 2002

The range of motion of a joint is influenced by the distance of the tendon attachment to the joint rotation centre as this parameter influences the elongation of the muscle. The closer to the center of rotation is the tendon attachement, the larger is the range of motion.

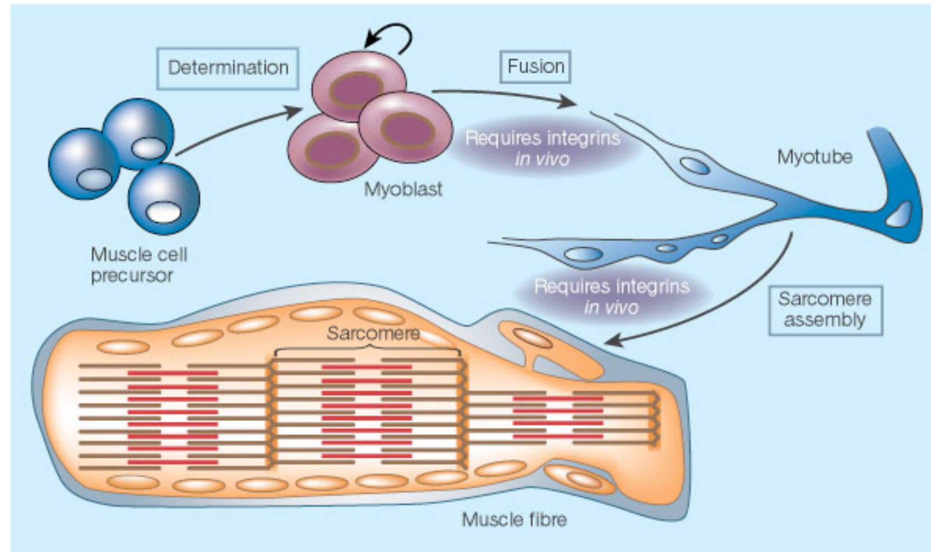
The hierarchically arranged structure of the muscles allows a serialisation of the contractile units



source: Brinckmann et al, Musculoskeletal biomechanics, Thieme, 2002

In the adult, approximately 2000 myofibrils join to constitute a muscle fibre. Myofibrils are sarcomeres arranged in series.

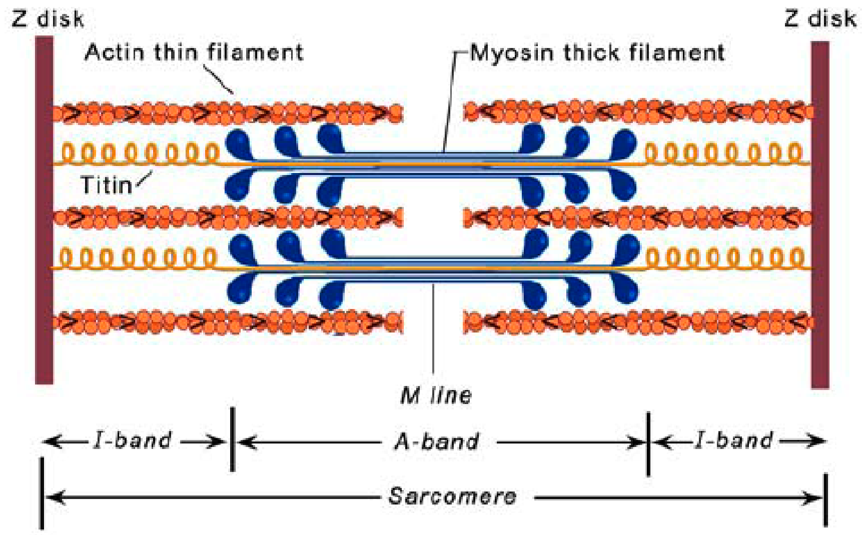
Cellular aspect of sarcomere creation ...

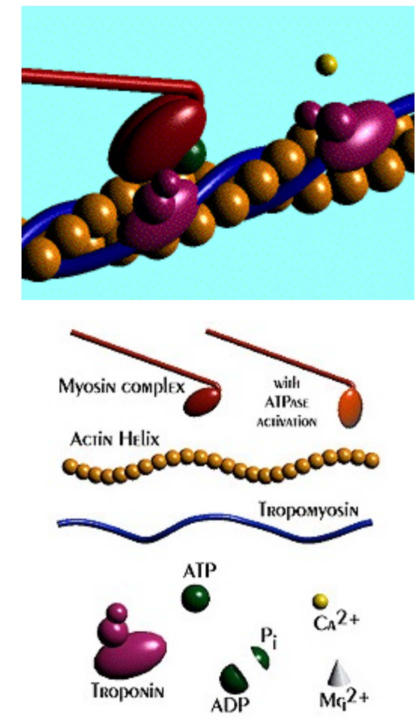


source: Gullberg, Cell biology: The molecules that make muscle, Nature, 424, 2003

The process begins with muscle cell precursors becoming 'destined', or 'determined', to form muscle, at which point they are called myoblasts. The myoblasts then proliferate and migrate to the site of future muscle formation, where they fuse to produce myotubes. As myotubes mature, the sarcomere develops; this array of myosin and actin fibres forms the contractile unit of muscle.

Molecular explanation of the force generated in muscles

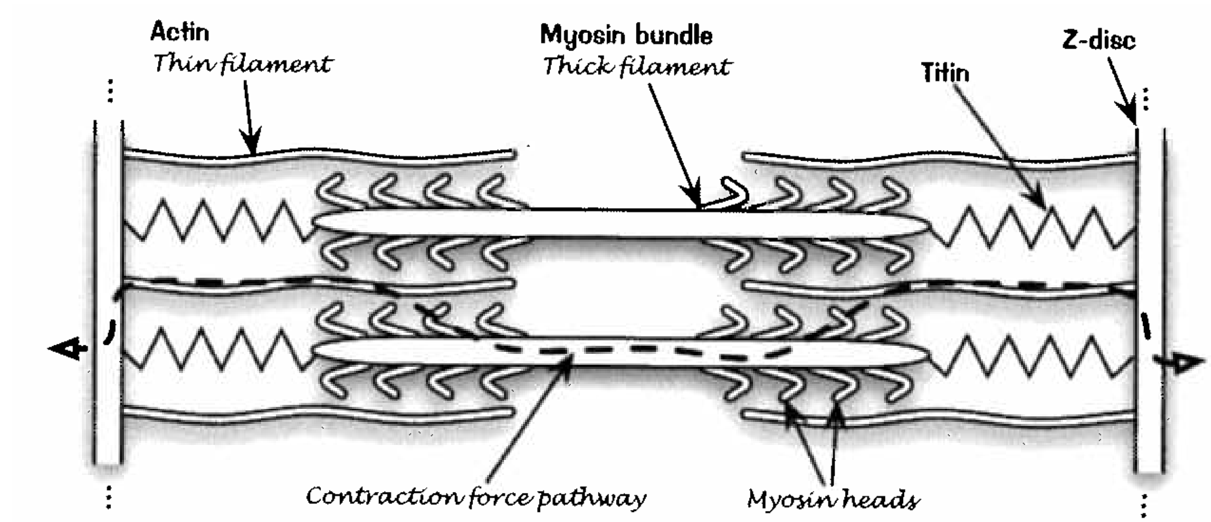




source: http://www.sci.sdsu.edu

The length of the myosin filament is assumed to be constant and not to contribute to the shortening of the sarcomere. Therefore, the shortening of a sarcomere is assumed to occur only via the sliding of the filaments relative to each others. The total shortening of the myofibril is the sum of the length changes occurring in the sarcomeres arranged in series. To make a long story short, the chemical energy transformation into mechanical energy and the slip of the filaments are the basic explanation for the contraction of the muscles.

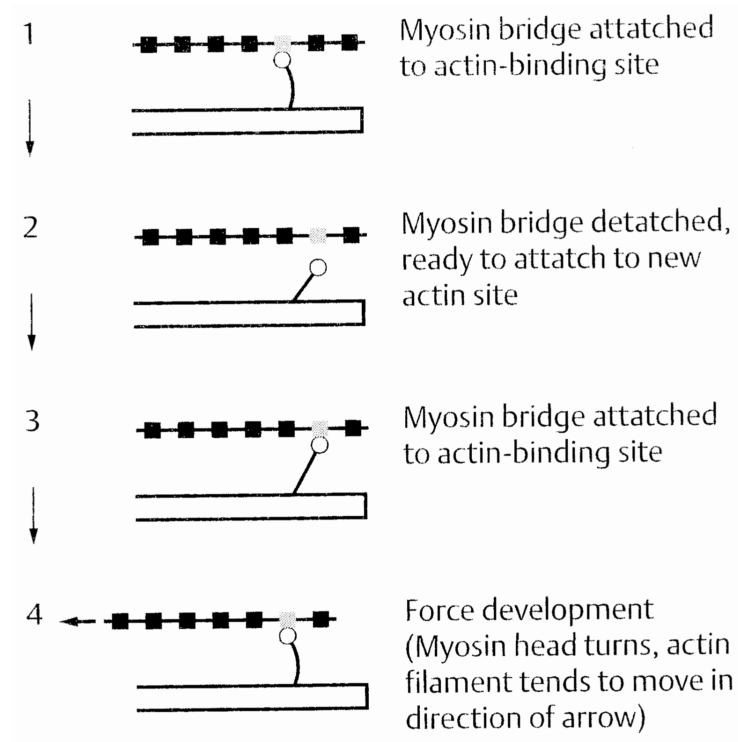
The “force pathway” follows the actin and myosin filaments



source: A.Pexidier Travaux pratiques de physiologie, EPFL

The “force pathway” of the sarcomere follows the thick and thin filaments as illustrated for one path in the figure. Under no load conditions, the stimulation of the muscle will cause it to contract to its smallest length without development of any active tension. In contrast, when contraction takes place against a resistance, a force is generated and mechanical work is done.

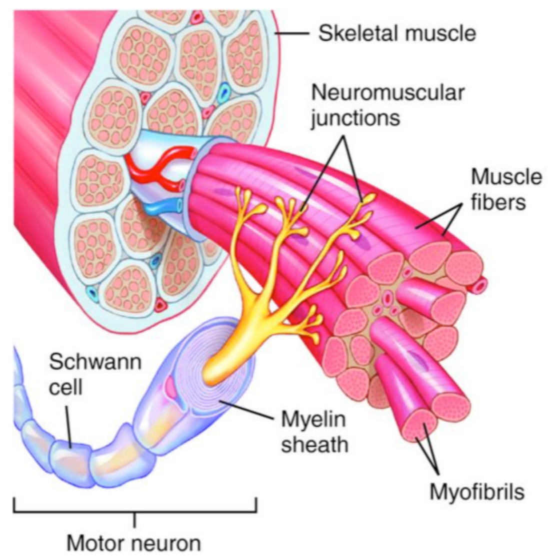
The principle of force generation in muscles



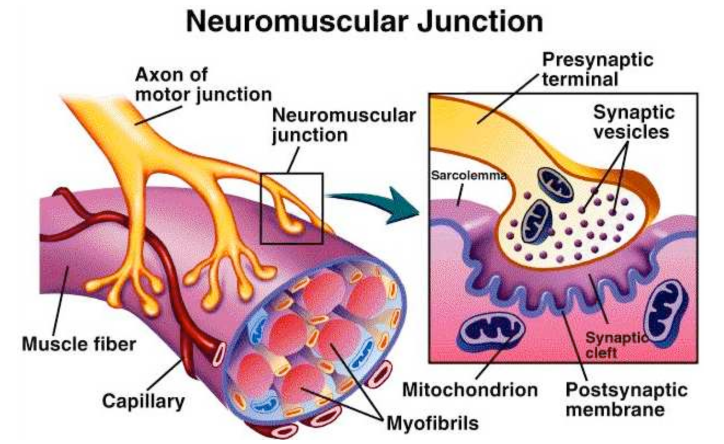
source: Brinckmann et al, Musculoskeletal biomechanics, Thieme, 2002

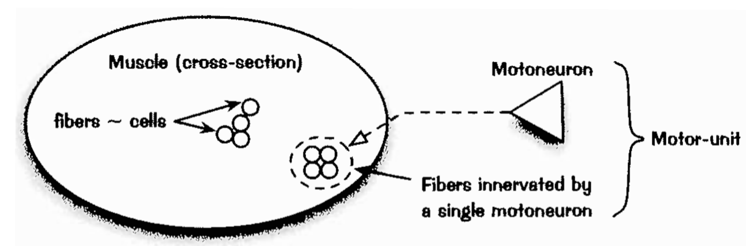
The basic hypothesis is to consider that active muscle force originates exclusively from cross-bridges.

Nervous system controls the muscle contraction



source: Mosby's Medical Dictionary, 8th edition. © 2009, Elsevier.

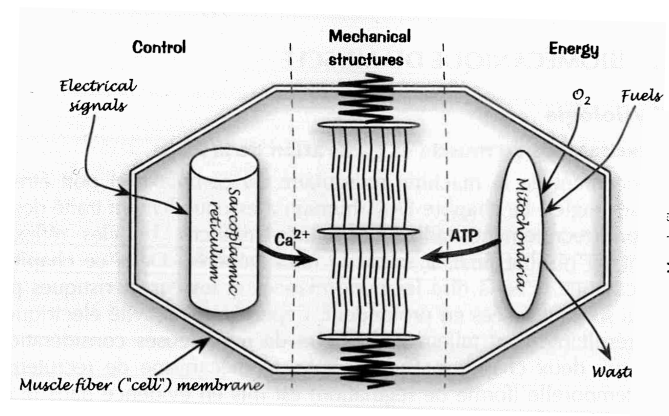




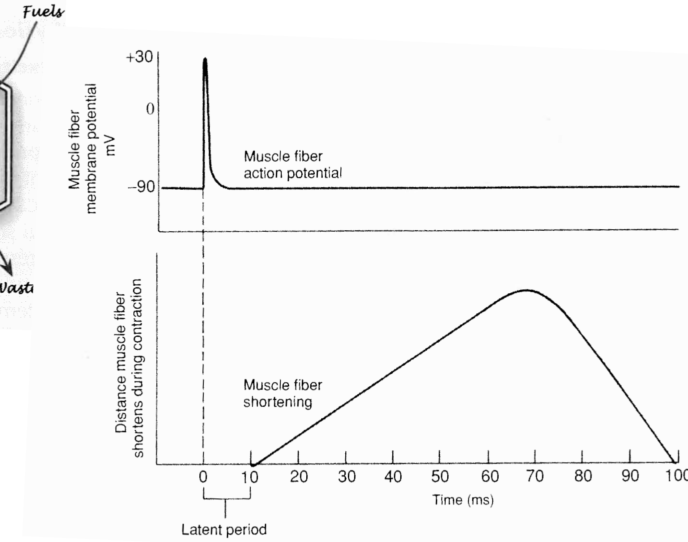
source: A.Pexidier Travaux pratiques de physiologie, EPFL

Indeed, the contraction force of skeletal muscles is under the control of the nervous system. Each neuron can be attached to several muscles fibres. A motor-unit is made up of a motor neuron and the skeletal muscle fibres innervated by that axon. We can find a distribution (small to big) of motor-units in one muscle, with corresponding small to large generated forces. The control of the total force generated by one muscle can then be obtained by recruiting more or less of motor-units (small to large). This process is called spatial summation. It is key to understand that the global force of a muscle is given by the addition of contraction of muscle fibres working alternately.

Delay between nervous system signal and muscle contraction



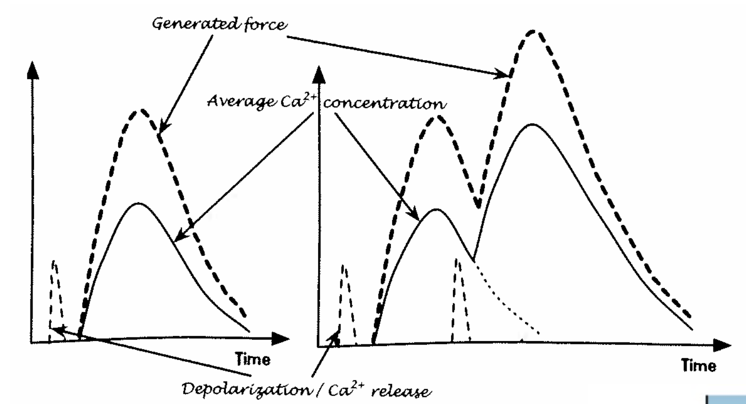
source: A.Pexidier Travaux pratiques de physiologie, EPFL

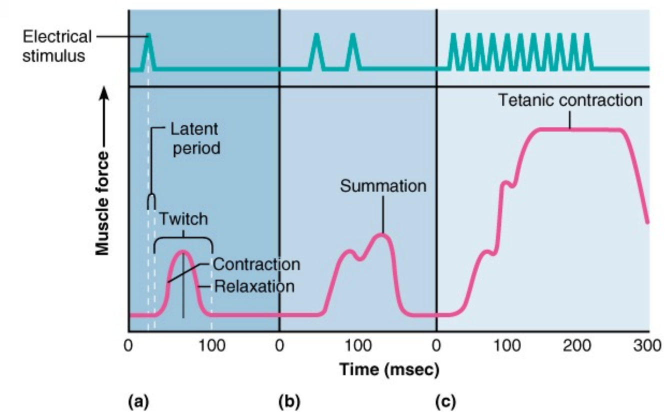


source: Ethier and Simmons, Introductory biomechanics, 2007

As the signal from the nervous system reaches the neuromuscular junction, a release of Ca^{2+} is induced. The Ca^{2+} takes some time to diffuse to the actin, a delay is then observed between the depolarisation (electrical signal) and the force generation. This delay is called the latent period.

A temporal summation of depolarization increases the resulting force





The temporal summation consists in activating several times the same unit before the cycle of contraction/relaxation is finished so that the generated force can be slightly increased (as with only one depolarisation, a part of the Ca^{2+} stock can be released). The frequency of the depolarisation can be increased until a saturation is obtained meaning that the amount of Ca^{2+} presents is in excess so that all the available myosin heads form a bridge with the actin-binding site generating a maximal muscular constant force called tetanus.

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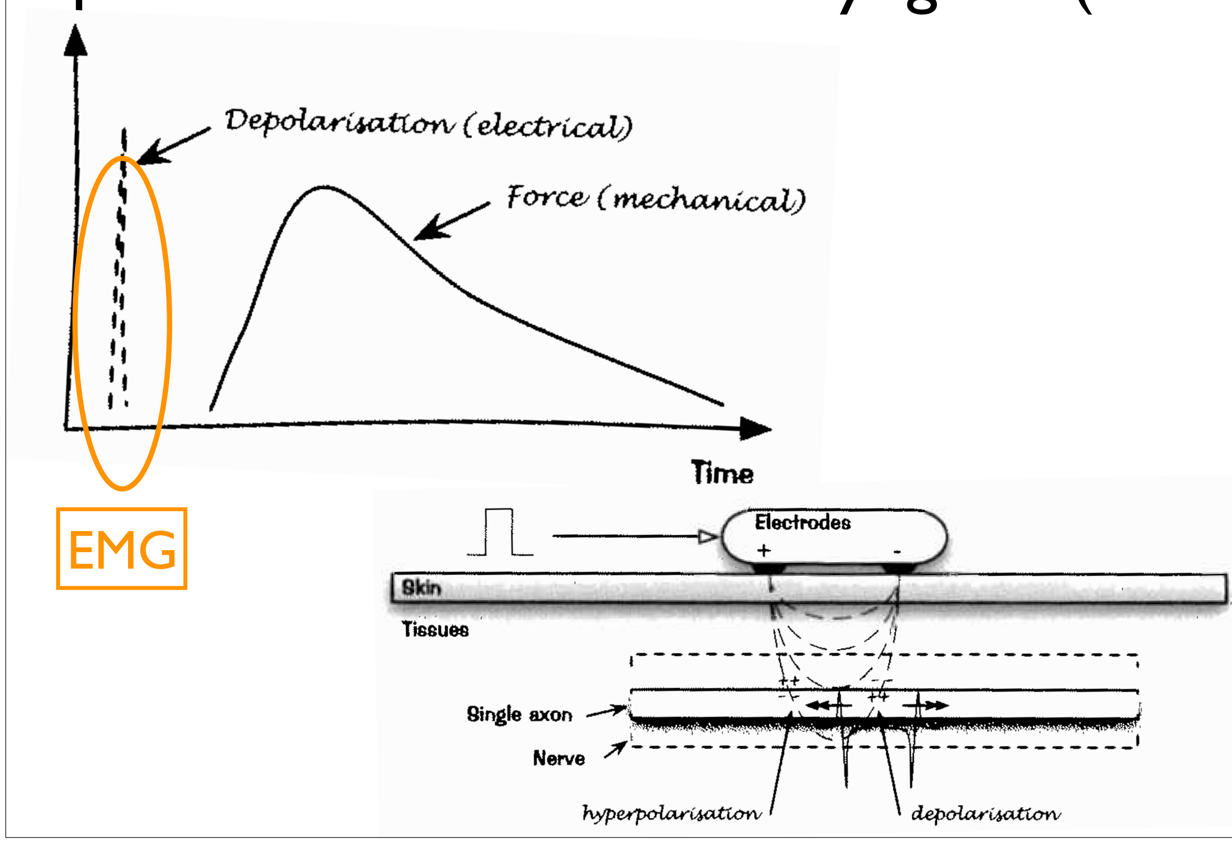
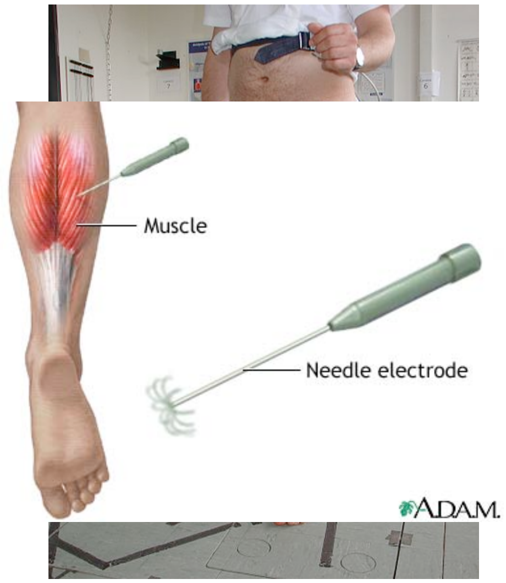
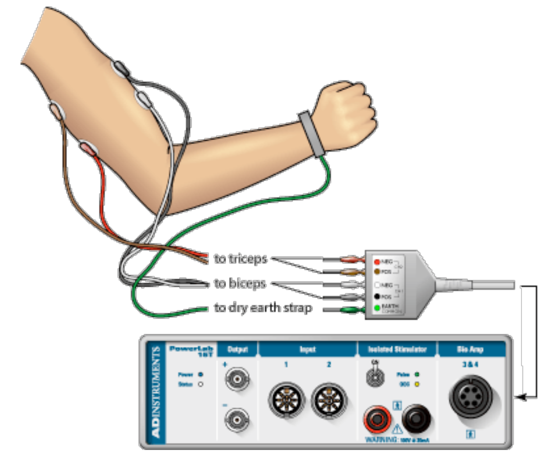
The electrical activity of the muscle can be quantified with an electromyogram (EMG)



The polarisation/depolarisation generates an electrical activity which can be monitored with a technique called electromyography. It is important to realise that an EMG does not indeed measure directly the mechanical behaviour of the muscle, but its electrical activity which is somehow linked. The different electrical signals generated by the motor-units can be added (if in phase) or subtracted (if not in phase). In order to synchronise the electrical signals, either a reflex can be induced in the muscle or an electrical signal can be generated by an electrode and the induced electrical activity of the muscle can be obtained.

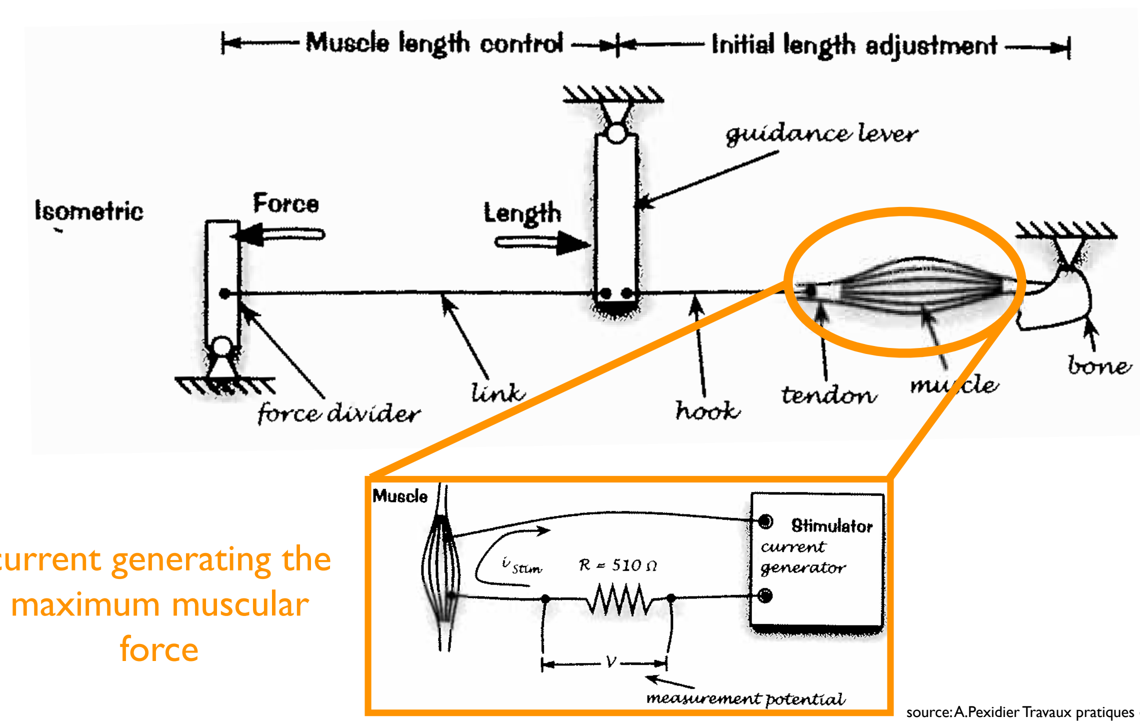
The electrical activity of the muscle can be quantified with an electromyogram (EMG)





One of the major limitation of the EMG is that only activity of surface muscles can be measured ... unless volunteers agree to have needles (used as electrodes) implanted in their deep muscles.

The force-elongation curve of a muscle is measured by superposing passive and active parts

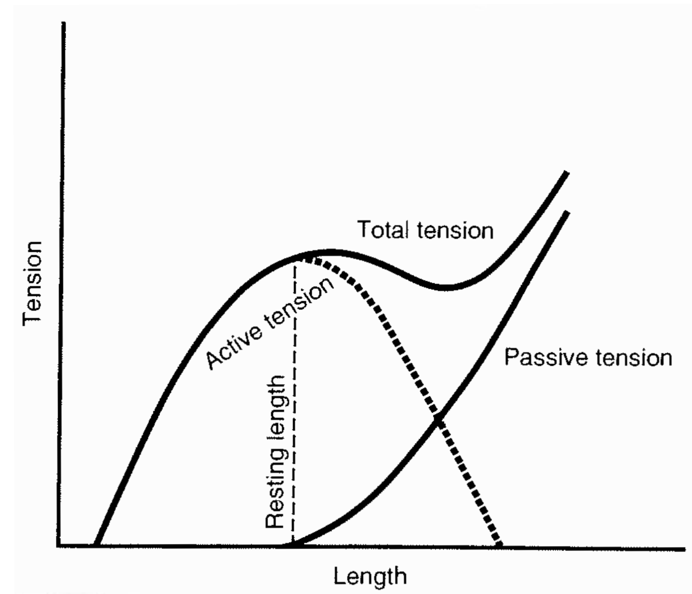


current generating the maximum muscular force

source: A.Pexidier Travaux pratiques de physiologie, EPFL

The passive length-tension curve of the muscle is obtained as usually done for any material. The active length-tension curve of the muscle is obtained in isometric condition. Indeed, it has been known since more than 100 years that the force developed by a muscle of constant length (= isometric contraction) varies with its initial length. In a very shortened or in a very lengthened state, the muscle generates less force than in mid-ranges. The isometric length-tension relationship can be measured directly when a muscle is maximally stimulated at a variety of discrete lengths and the resulting force is recorded.

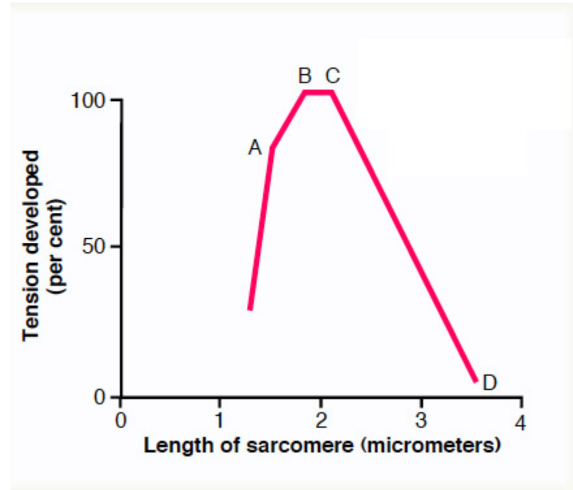
The force-elongation curve of a muscle is composed of passive and active parts

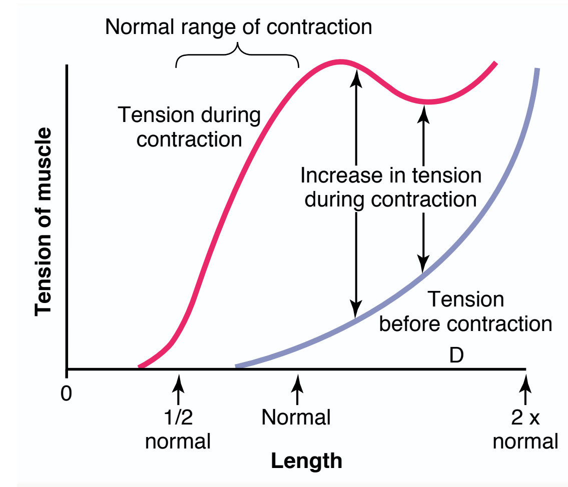


source: Basic biomechanics of the musculoskeletal system, M. Nordin and V. Frankel, 2001

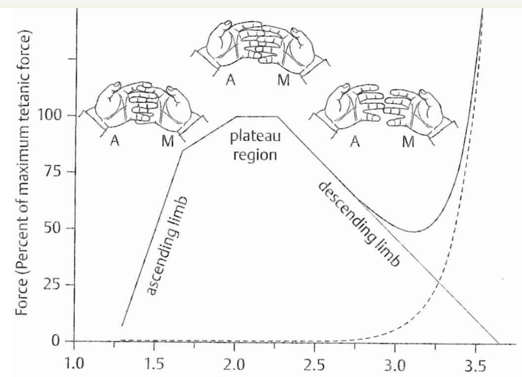
The length-tension curve of a muscle represents the result of many experiments plotted on the same graph, i.e. an artificial connection of individual points from isometric experiments added with its passive behaviour. It is important to realise that the length-tension curve was obtained in “static” condition as the muscle was not allowed to shorten. Therefore, the length-tension relationship is strictly valid only for isometric contractions.

Active contraction force depends on sarcomere length





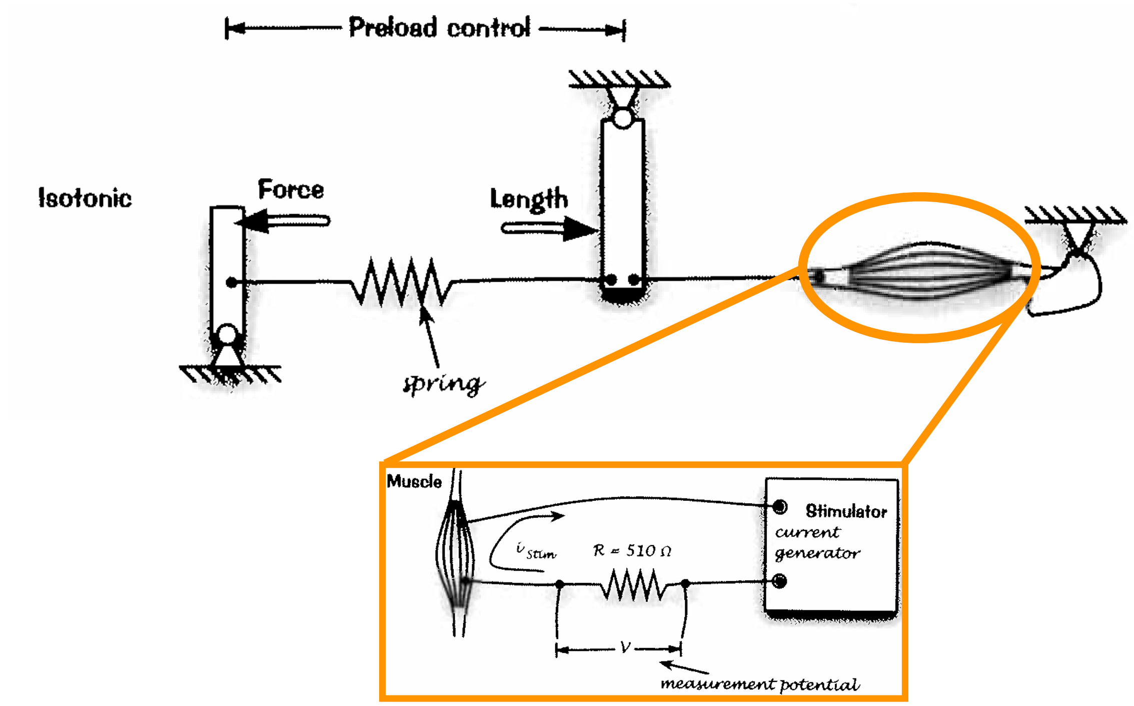
source: Medical physiology, Guyton and Hall, 2006



source: Brinckmann et al, Musculoskeletal biomechanics, Thieme, 2002

At point D on the diagram, the actin filament has pulled all the way out to the end of the myosin filament, with no actin-myosin overlap. At this point, the tension developed by the activated muscle is zero. Then, as the sarcomere shortens and the actin filament begins to overlap the myosin filament, the tension increases progressively until the sarcomere length decreases to about 2.2 micrometers. At this point, the actin filament has already overlapped all the cross-bridges of the myosin filament but has not yet reached the centre of the myosin filament. With further shortening, the sarcomere maintains full tension until point B is reached, at a sarcomere length of about 2 micrometers. At this point, the ends of the two actin filaments begin to overlap each other in addition to overlapping the myosin filaments. As the sarcomere length falls from 2 micrometers down to about 1.65 micrometers, at point A, the strength of contraction decreases rapidly.

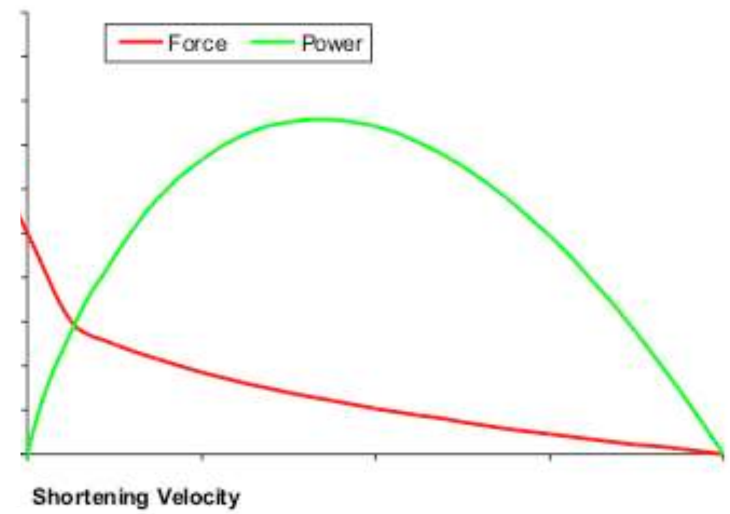
The velocity-force curve of a muscle



source: A.Pexidier Travaux pratiques de physiologie, EPFL

The velocity-force relationship, like the length-tension relationship, is a curve that actually represents the results of many experiments plotted on the same graph. Experimentally, a muscle is allowed to shorten against a constant load. The muscle velocity during shortening is measured and then plotted against the resistive force.

The velocity-force curve of a muscle



The idealised graphic is represented without absolute value as the values would vary between individuals (or due to training).
What is the physiologic basis of the velocity-force relationship? The force generated by a muscle depends on the total number of cross-bridges attached. Because it takes a finite amount of time for cross-bridges to attach, as filaments slide past one another faster and faster (i.e., as the muscle shortens with increasing velocity), force decreases due to the lower number of cross-bridges attached. Conversely, as the relative filament velocity decreases (i.e., as muscle velocity decreases), more cross-bridges have time to attach and to generate force, and thus force increases.

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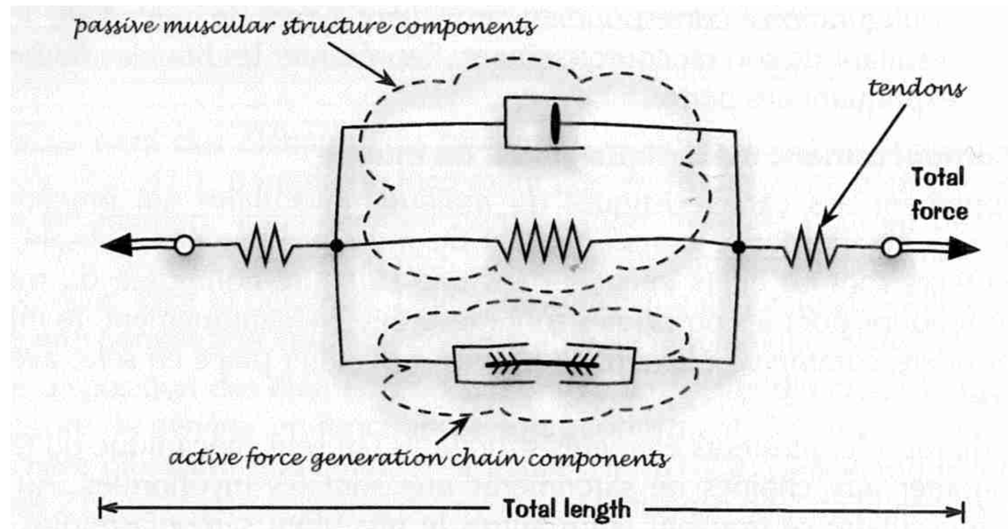
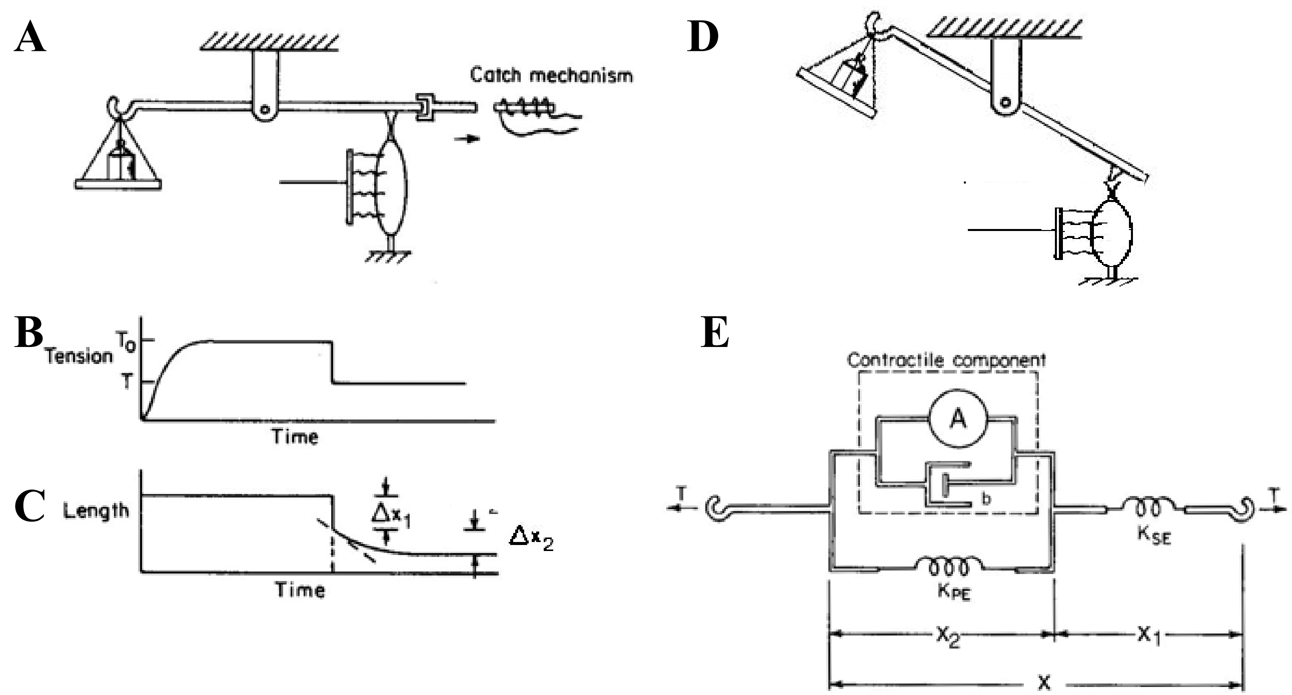
Spring, viscous, and active elements



source: A.Pexidier Travaux pratiques de physiologie, EPFL

Prof Hill (Nobel Prize for Physiology or Medicine, 1922) has proposed to model the muscular force with spring, dashpot (=viscous) and active elements placed in series and in parallel. This phenomenological model was derived from his experimental observations that activated muscles produce more force when held isometrically (i.e., at a fixed length) than when they shorten. Moreover, when muscles shorten, they appear to waste some of their active force in overcoming an inherent resistance.

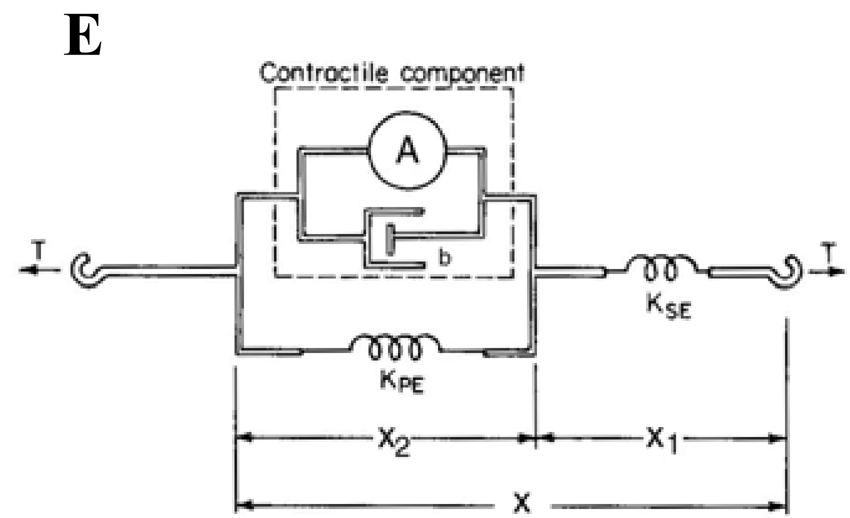
Spring, viscous, and active elements



source: McMahon TA (1984) Muscles, reflexes, and locomotion. Princeton University Press: Princeton, N.J.

Initially, the bar in (A) can not pivot and the muscle is maximally stimulated generating a force T_0 as can be seen in (B). The length of the muscle stays constant as the bar can not pivot (C). After a certain time, the catch mechanism of the bar is removed so that the bar can pivot (D). In (B), you can notice the force drops from T_0 to T and the length decreases immediately, then continues to decrease at a slower rate. The immediate release of the force is described by a spring in series with a stiffness called K_{SE}. (By definition the stiffness is given by K = F/L; SE: series elastic and PE: parallel elastic). The slow gradual changed in length can be described by a spring (with stiffness called K_{PE}) and dashpot in series (Kelvin-Voigt model). Finally, in (E) the active element of the muscle, called A, is a force acting against the passive elements of the model to produce the force allowing to pivot the bar. Based on this description with basic elements, the force evolution of the muscle can be theoretically calculated if the parameters (stiffness, viscosity) of the basic elements are known.

Hill model in equations



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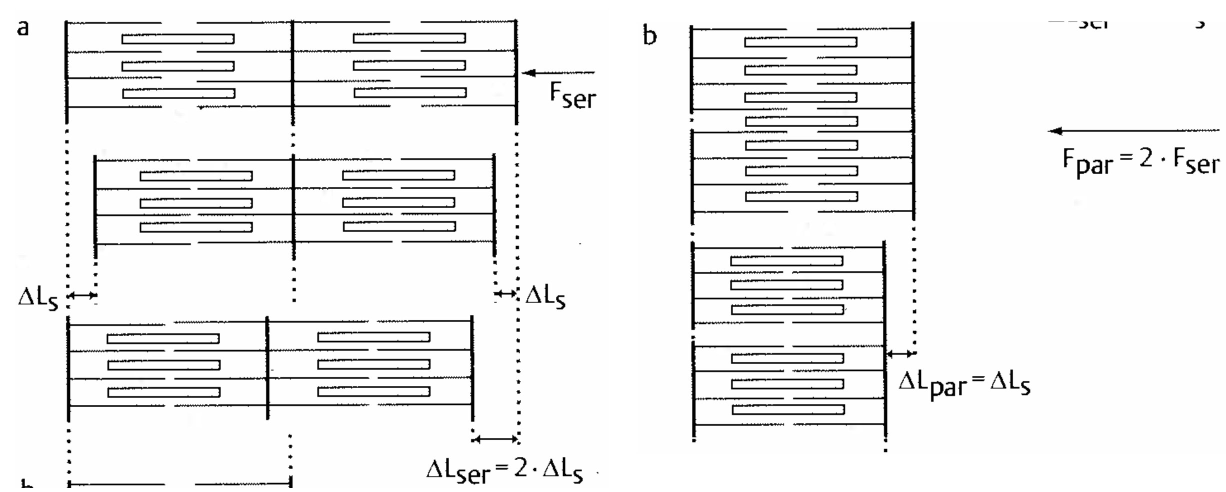
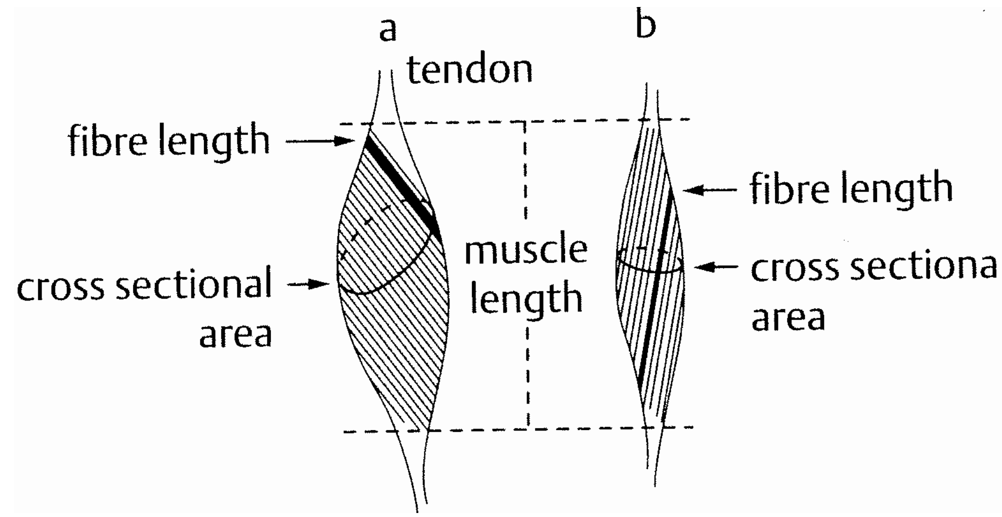
Muscle force is proportional to the quantity of myofibrils arranged in parallel



source: Brinckmann et al, Musculoskeletal biomechanics, Thieme, 2002

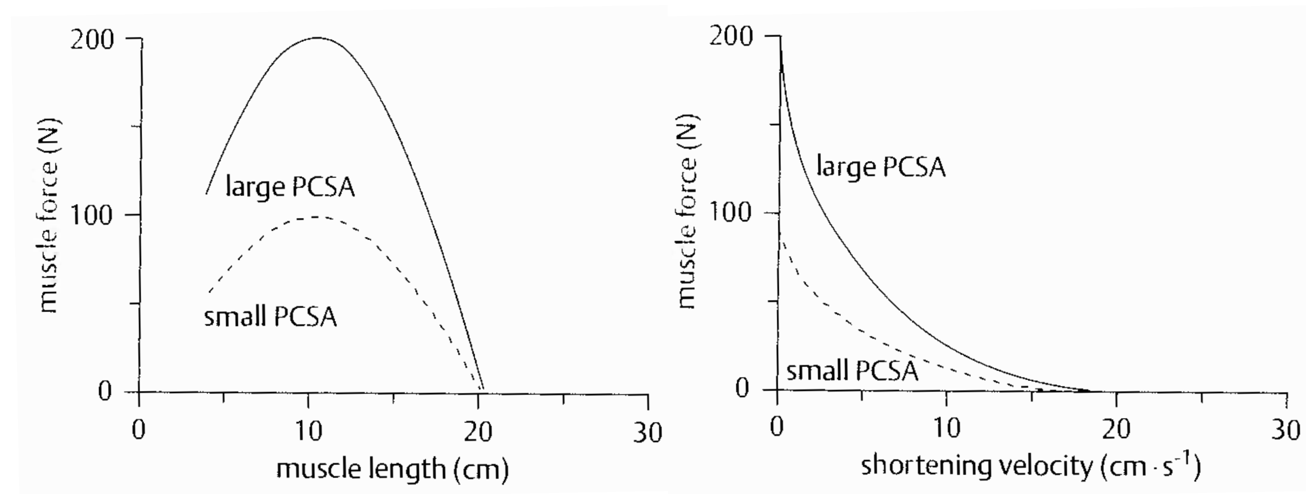
At the level of the sarcomere, in case a), the maximum force is half of the maximum force b), but in case a) the maximum change in length is twice the maximum change in length in b). We can deduced that the maximum force developed in a muscle fibre will increase proportionally to the quantity of myofibrils arranged in parallel, thus we can extrapolate that the muscular force is proportional to the cross-sectional area of the muscle.

Indeed, muscle force is proportional to its physiological cross-sectional area (PCSA)



The physiological cross-sectional area (PCSA) is the total area of the cross-section perpendicular to the muscle fibres. This is then a measure of the number of sarcomere in parallel in the muscle and it gives an indirect measure of the muscle's force-generating capacity.

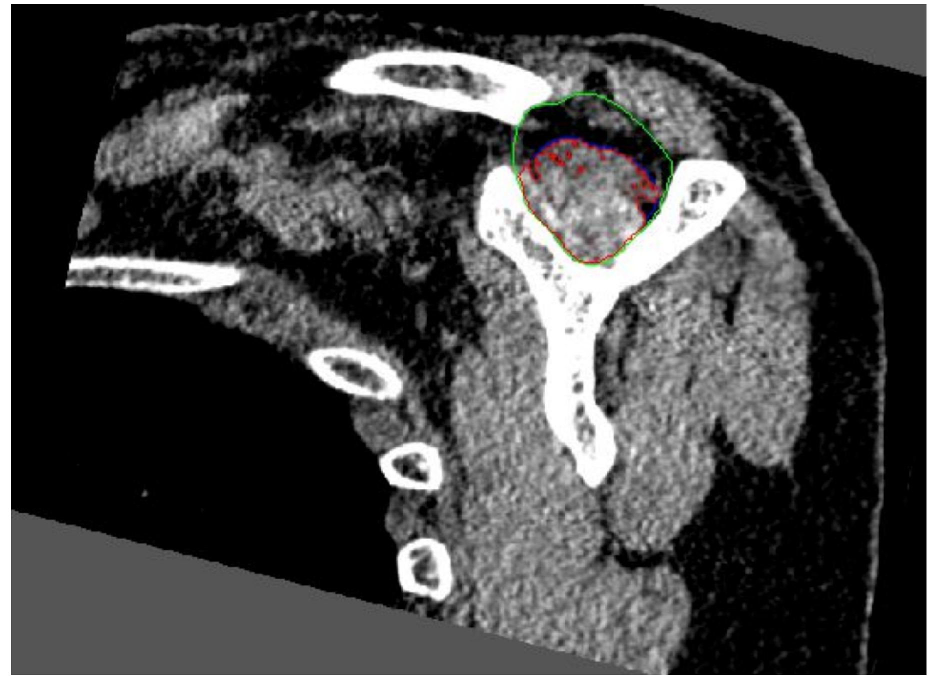
Muscle force is proportional to its physiological cross-sectional area (PCSA)



source: Brinckmann et al, Musculoskeletal biomechanics, Thieme, 2002

Suppose that two muscles had identical fibre lengths and pennation angles, but one muscle had twice the mass (equivalent to saying that one muscle had twice the number of fibres and thus twice the PCSA). What would be the difference in their mechanical properties? How would the length-tension and force-velocity curves be affected? This schematic demonstrates that the only effect is to increase maximum tetanic tension so that the length-tension curve has the same basic shape but is simply amplified upward in the case of the stronger muscle. Similarly, the force-velocity curve simply changes the location of F_0, but the curve retains the same basic shape.

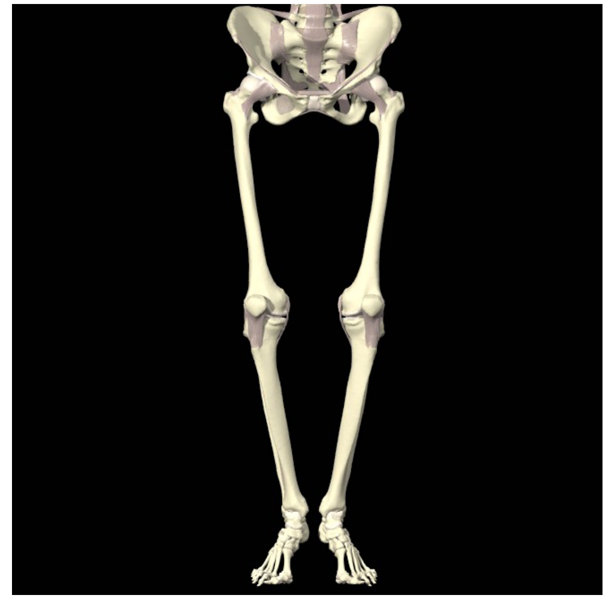
Muscle force is proportional to its physiological cross-sectional area (PCSA)



source: A. Terrier, LBO

The PCSA can be obtained from MRI imaging. In this particular example from the shoulder of a patient, we can observe that a muscle (supra spinatus) is atrophied because it should have filled all the surface delimited by the green curve. The actual PCSA of the patient is delimited by the red curve and fatty insertions (red dots) are also removed from its calculation. The Y-shape bone is the scapula and the bone above is the clavicle. The possible generated force by this patient to move his arm will obviously be decreased due to the muscle atrophy.

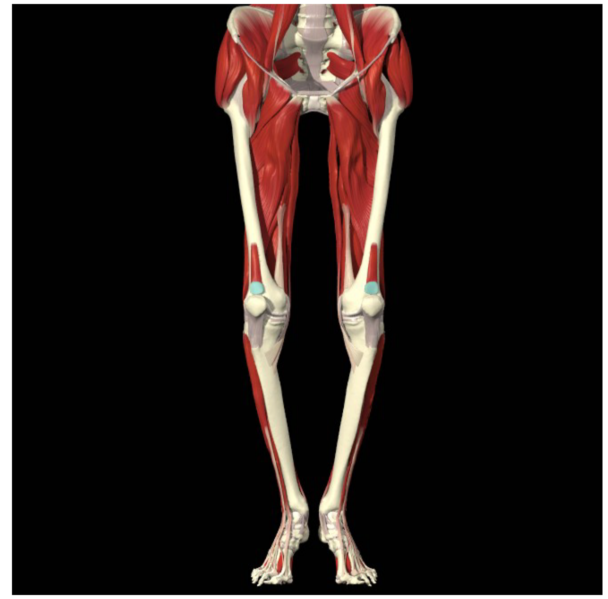
The skeleton alone seems quite unstable



A powerful equipment of muscles stabilizes the knee

Adducteur magnus (below) et brevis (above)

A powerful equipment of muscles stabilizes the knee



Adductor longous et gluteus medius (hip external), gluteus minimus (touch the medius internally)

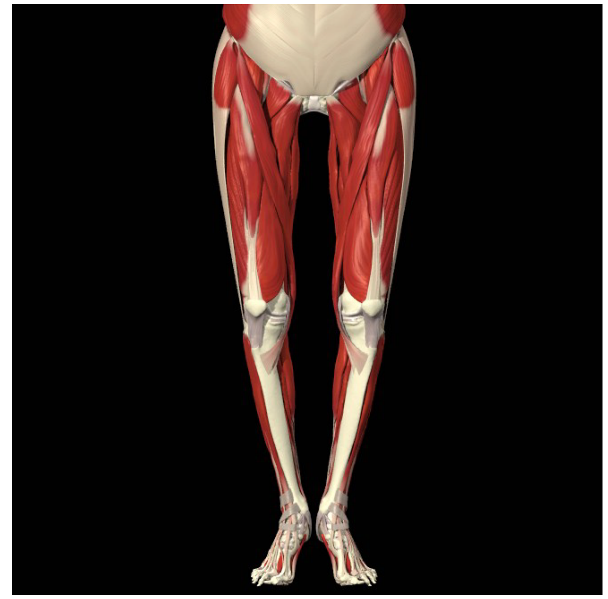
A powerful equipment of muscles stabilizes the knee

Vastus intermedius (external), vastus medialis (internal)

A powerful equipment of muscles stabilizes the knee

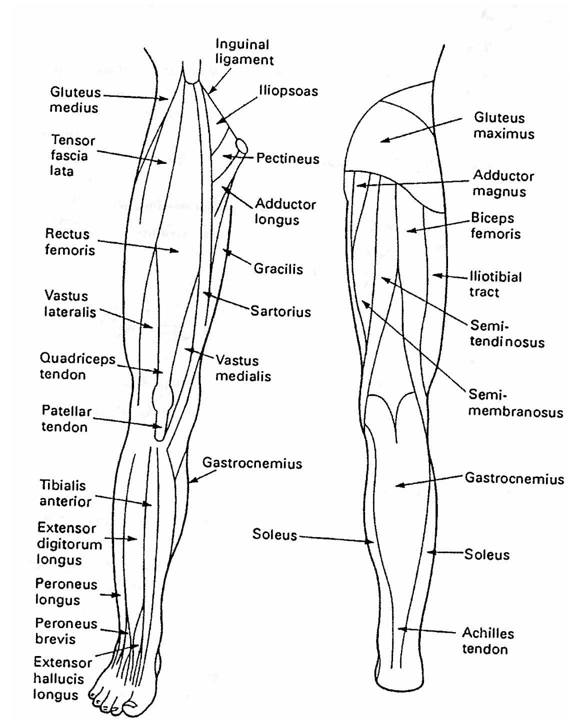
Vastus lateralis (external), rectus femoris (middle), sartorius (band in the middle)

A powerful equipment of muscles stabilizes the knee

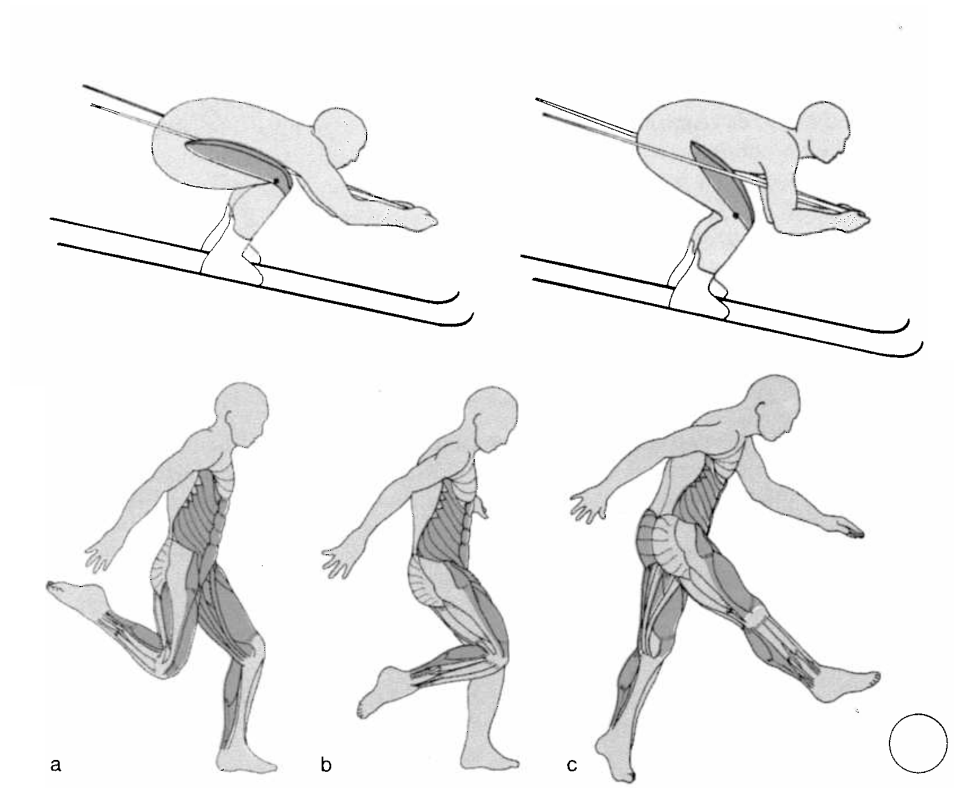


Tenseur fascia (cover like a socket), gastrocnemius (mollet)

A powerful equipment of muscles stabilizes the knee



A powerful equipment of muscles stabilizes the knee



Each muscle has a different role

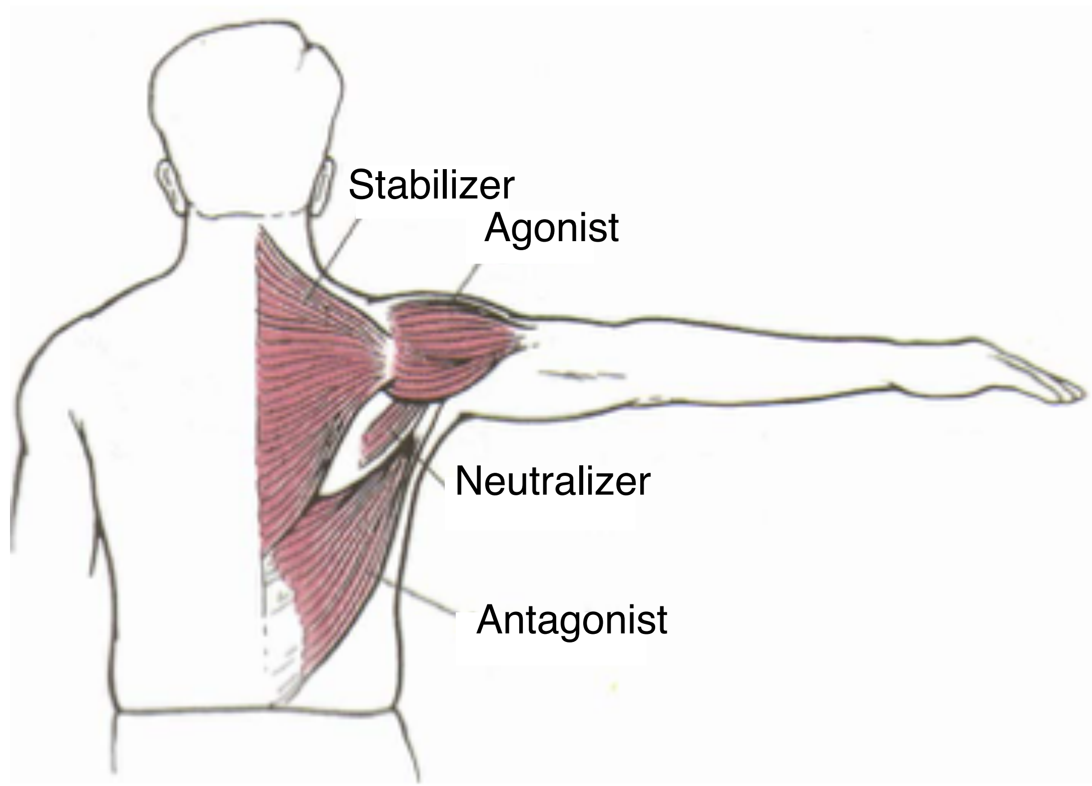
Agonist
Muscle that produces desired movement

Antagonist
Oppose motion of agonist

Stabilizer
Muscles that act in one segment so that
movement in an adjacent segment can occur

Neutralizer
Muscles that eliminate an undesired action of another muscle

Each muscle has a different role



Muscle biomechanics

- How do muscles work?
    - Muscles functional anatomy
    - Modes of muscle function
    - Structure and molecular functioning of the active part of the muscle
- How can we measure muscle activity and mechanical properties
    - Force-EMG relationship
    - Passive and active force-elongation curves
    - Force-velocity curve
- How can we incorporate muscle function in biomechanics
    - Spring and active elements
    - Force/cross-sectional relationship