How fast a muscle can contract?

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Introduction

How fast a muscle can contract? How fast a runner can run? How fast human body can react against a phenomenon? How fast a bird can flap?
There are a lot of questions like these that is important for human to know. Although each question should be investigated individually and for each case all related parameter should be considered, the common question among all of them is “How fast a muscle can contract as an actuator?”
To finding out the answer of this question first of all the structure of muscle should be investigated from different views. Physiological views, chemical reactions, mechanical behaviors will be discussed in the first chapter. Different type of muscle fibers and the architecture of muscle will be explained in the second chapter. In the chapter three modeling and experiments results will be elucidated. Finally in the last chapter by the use of the model and information some calculation will be run to give a sense about the speed of muscle.

1. Muscle in Different Views

1.1 Physiology

Muscle in macroscopic view is a soft tissue of body that is connected to bones by tendons, and it cause the motion by contraction.

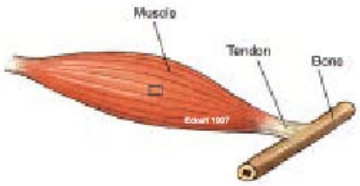


Fig 1: Muscle is connected to the bone via tendon

But in microscopic view it is made up of numerous muscle fibers. Each fiber also is a bundle of thinner fibers, which are called myofibrils. Zooming in to a myofibril, indicates that each myofibril is made up of small sections (Sarcomeres) in series form. Sarcomeres are the smallest anatomical unit that contract like a muscle.

Fig 2: Myofibers

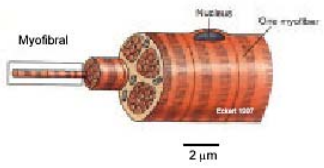


Fig 3: Myofibrils

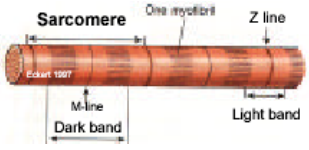


Fig 4: Sarcomere

1.1.1 Inside a sacromere

As the contraction of sarcomeres cause the contraction of muscle it is better to have a look inside a sarcomere.
A sarcomere is approximately cylindrical shape with 1µm diameter and about 2µm length in rest status. Both ends of a sarcomere are two tough tissues which are called z-discs or z-lines. Inside a sarcomere there are two kinds of filaments: Thin filaments or actins and thick filaments or myosins (fig 5).

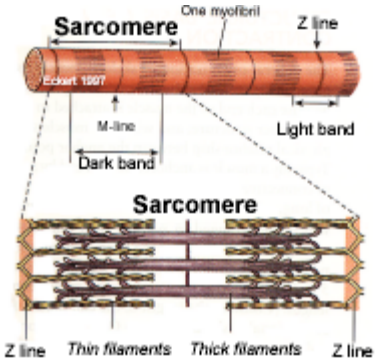


Fig 5: Inside a sarcomere

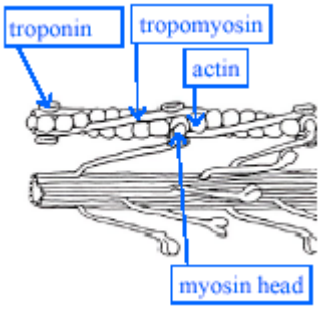


Fig 6: Actin and Myosin

Myosin is resembled to a trunk of a tree with several branches. At the end of each branch there is a myosin head.
Actin has a cylindrical shape that some worm like molecules (tropomyosin) have been twisted along the actin somehow like a screw teeth. There are some seat form molecules along tropomyosin that they are called troponin (fig 6).

In order to have a sense about the size of filaments, table 1 includes some approximate values:

Table 1: The size and distance of several parts in a sarcomere

Sarcomere diameter	1 µm
Sarcomere length	2.28µm
Myosin length	1.65µm
Actin length	1.04µm
The spacing between thick filaments	450 Å
The spacing between thick and thin filaments	130 Å
working stroke of the cross-bridge	14.0 nm

The process of contraction of a sarcomere can be abstracted in four individual steps:

a. Myosin head which is attached to troponin rotates a bit
b. The myosin detaches the actin
c. The myosin head bent back to normal position
d. Myosin head attaches to the troponin

In fig 7 these four steps have been indicated in very simple form

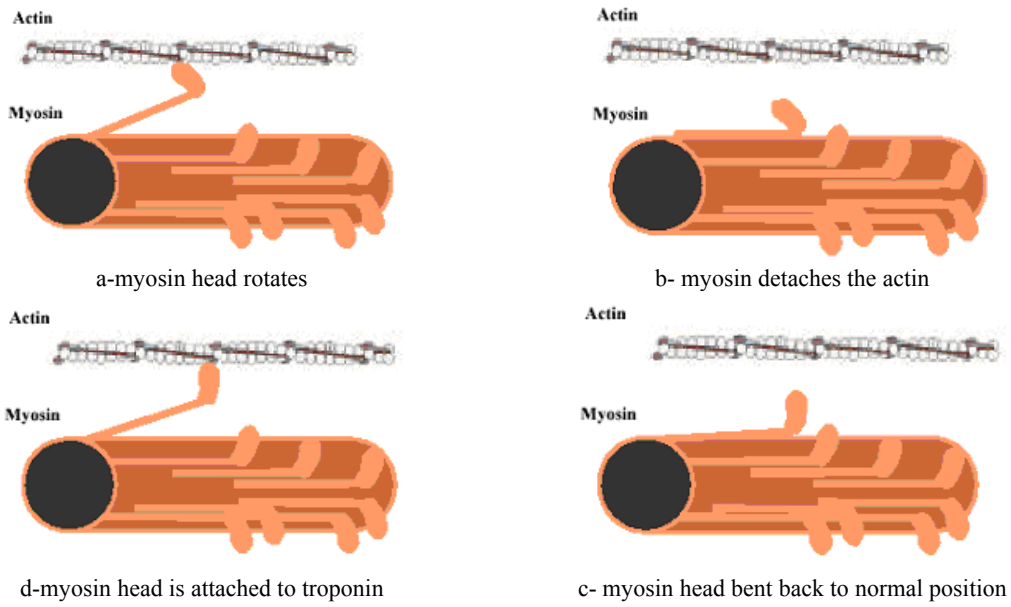


Fig 7: 4 steps of actin and myosin motion

Each step takes place by a chemical reaction that will be discussed shortly in the next few section.

Repeating the four steps cause a large motion of actins along myosins.

1.1.2 Command of contraction

The contraction of muscle is controlled by nerves. A motor nerve enters muscle and split into numerous axons; each axon contacts 10-2000 muscle fibers as each muscle fiber is innervated by only one motor nerve axon. All sarcomeres that are connected to that motor nerve axon via myofibrils and muscle fibers contract in response to an action potential in that axon.

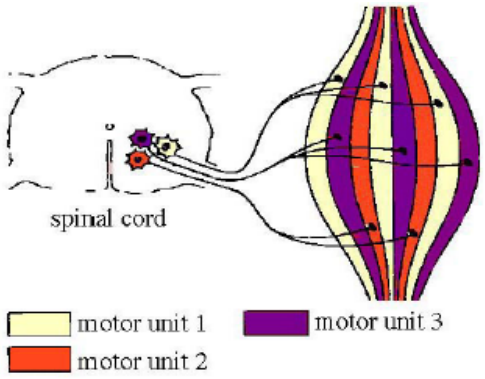


Fig 8: motor units

According to the definition a single motor nerve axon and all muscle fibers it contacts are called as motor unit.
The total contraction of muscle depends on the relative status of myosin and actin of sarcomeres and the number of sarcomere that are connected in series in myofibers. Obviously the longer muscle has a larger contraction. Also the force which produce by this contraction can be increased by increasing the number of muscle fibers which are parallel together. But the trend of contraction is not uniform. This point can be explained by the relative status of myosins and actins in a sarcomere.

1.1.3 Force – Length characteristic of a sarcomere

The force that a sarcomere can generates is depended to the number of cross-bridges between myosins and actins and also the status of these cross-bridges.
The force-length curve of a sarcomere has been indicated in fig9.
As it is indicated there are 5 specific points in the graph.

1. In the rest status (length=2μm) myosins and actins have the maximum cross-bridge so they can produce the maximum force (100%).

2. Contraction of a sarcomere from normal length up to 85% runs by some overlap of actins. This overlap prevents to do all cross-bridges, so the shorter is the sarcomere from its rest length, the less the force generates. At point 2 (length=1.6μm) the myosin reaches to the z disks and the force that it can generate would be about 80% of maximum.

3. The worst case is when myosins touch the z-discs. So for more contraction the myosin should suffer the stress of z-discs addition to less force from less cross-bridges.
   Hence, the force that a sarcomere can produce, collapse rapidly up to 62% of the rest length of sarcomere (length=1.25µm) which force reaches to zero.
   Further contraction is impossible because the z-discs don't allow the actins to pass throw them. So the maximum contraction that a sarcomere can produce is 38%, but the maximum contraction of a muscle is about 20%, because of other tissues among sarcomeres that they are not compressible.

In many cases a muscle should be stretched. And the status of myosins and actins and the force that they can generate is important.

4. While a sarcomere is stretched up to 110% of its rest length (length=2.25µm) the cross-bridges have a normal situation and all myosins participate in attachment to actins. So the force remains constant.

5. But further stretch cause some myosin heads go to the zone that they can't attach to the actins. Therefore the more sarcomere stretches, the less myosin heads can attach. This trend goes on up to 175% of sarcomere rest length (length=3.6µm).
   There is not any attaching force for further stretch.(dangerous for muscle tissue).

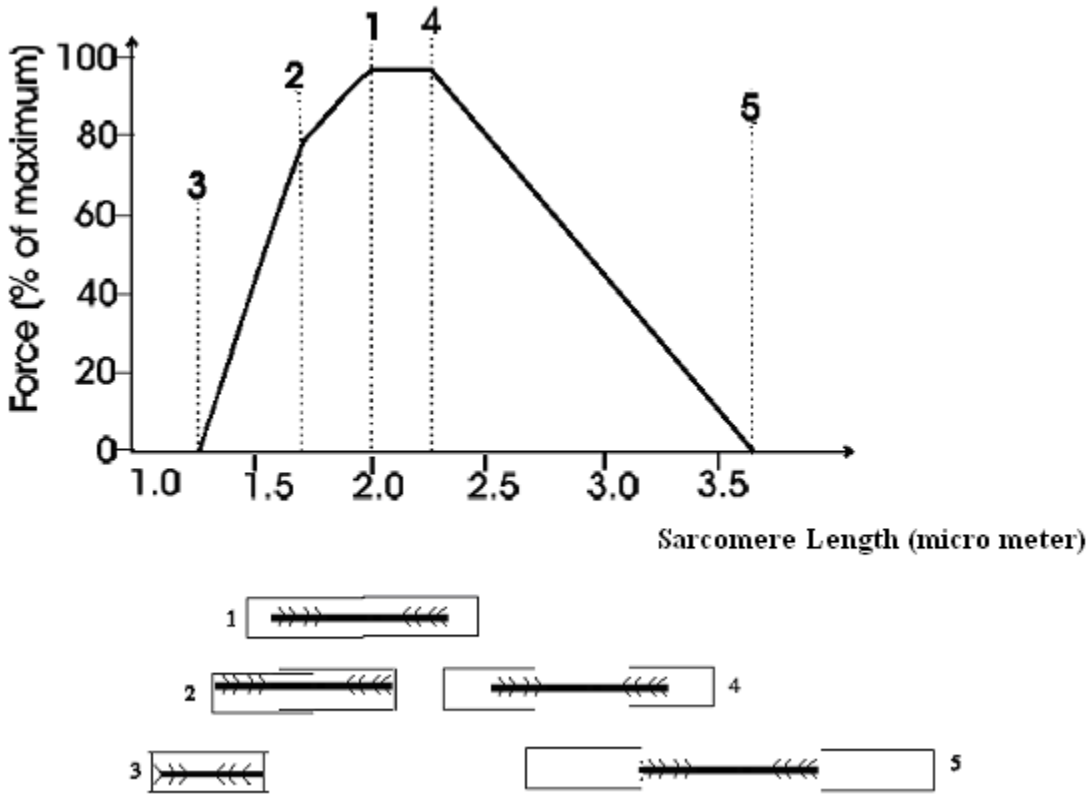


Fig 9: The Force-Length diagram of a sarcomere

The force-length relationship is useful to model the contraction of muscle.
In order to have a bit information about chemical process that cause the interaction between myosin and actin, in the following section some important point has been mentioned.

1.2 Chemical View

The required energy for contraction of a muscle obtains from chemical reaction. The ultimate source of energy resides in the food (for muscle contraction), the energy values of the various food stuffs are known. But how much energy in muscle is obtained is depended on the chemical reaction.
Previously, it was supposed that energy for contraction was produced by glycolysis that is the break down of glycogent lactic acid.

$n\ (C_6H_{12}O_6) \rightarrow 2n\ (C_3H_6O_3) + \text{Energy}$

This theory was called Lactic Acid Theory.
In the base of several experiments, today it is believed that ATP (Adenosine TriPhosphate) is direct energy source for contraction.

The most important factor in the speed of contraction is the rate of the cross-bridge cycling process.
The transition, from attached state to detached states requires ATP hydrolysis. As we will see, rate constant s of the different transitions are different between slow and fast muscle fibers and depend on the velocity of filament sliding and thereby determined the force produced. See fig 10 for a simplified biochemical representation of the cross-bridge cycle. More detail could be found in [1].

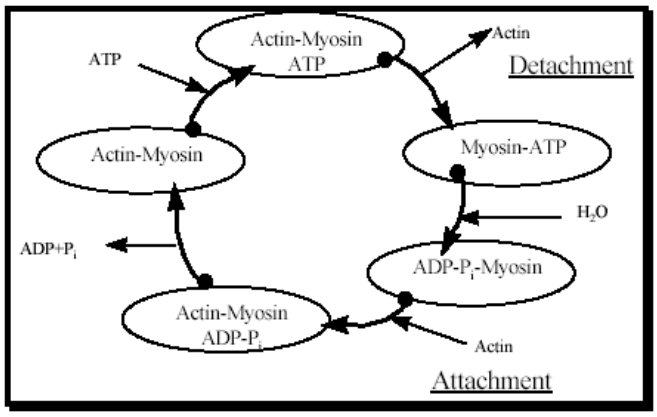


Fig 10: The biochemical representation of cross-bridge cycle

The rates of attachment of cross-bridges (force generating) and detachment (non-force generating phase) depend on the velocity with which actin and myosin slide along each other. The higher this velocity in shortening, the fewer cross-bridges will be in the attached phase and the more will be in the detached phase (to later attach again and pull-in the actin).
The fewer attached cross-bridges are at a particular time, the less the produced force will be. In a fast muscle fiber, the transition rates are such that at a given speed more cross-bridges are attached compare to slow. Further more, the pulling-in of the actin filament by cross-bridges occurs at a higher rate (this pulling-in is in fact a biochemical transition) thus fast muscle fibers can contract at higher maximal speed.

1.3 Mechanical View

Lets have a mechanical view to a muscle. Muscle can be considered as a non-ideal machine which converts chemical energy to mechanical motion, and heat.
The chemical reaction in muscle generates heat that is vital for maintaining body temperature. So the efficiency of muscle is:

Efficiency = Work produced / Free energy of ATP

Measurements indicate that efficiency of muscle is about 50% that is pretty good in compare of other machines.

In order to have a sense of work produced in muscle, we can consider the four step of contraction of a sarcomere like an engine of car that has four steps of operation.
Although each step needs some amount of energy to do the attachment / detachment and binding / unbinding but all of these activities are internal motion and the only part that can produce the relative motion of actin and myosin is in the binding the myosin head while it is attached to troponin.
Regarding the status of myosin head (fig11), it is attached to troponin in t_{on} interval. If d would be the working distance, the amount of work which will be done by a single myosin head is:

$W = F \times d$

d is about 5-14nm and F is 2-5 pN (10^{-12} N). Consequently the work would be in the range of $1\text{-}7 \times 10^{-20}$ J.

If the attached time is t_{on} then the sliding speed of myosin and actin is:

$$v = d / t_{on}$$

So to get faster sliding speed it is necessary to have a short t_{on}.
But the total time ($t_{on} + t_{off}$) can't be less than the time which ATPase rate. Because fuel should feed to the sarcomere .

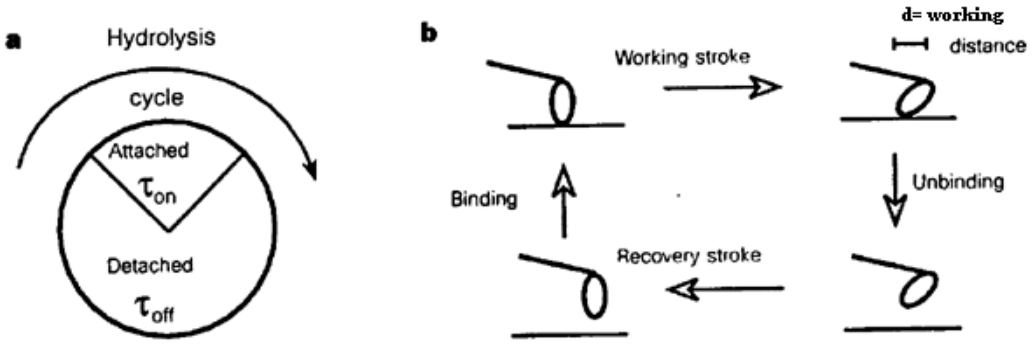


Fig 11: The proportion of attaching time of a myosin to actin

The 4 step motor rhythm is similar to an engine of a car. All useful energy obtains only in one stroke from 4 steps.
So the motion can't be uniform except that there will be other motor proteins in parallel to the first one with some different phases of stroke.

The cross bridge theory is the best theory that can explain the contraction of muscles so far. But it is weak in describing some behaviors. Some of these weak points have been mentioned in appendix1.

2. Structure of Muscle

So far, we have considered the basic of muscle structure is same for any muscle and anybody. But indeed a muscle performance is depended to the geometry and type of different fibers. It is a point that should be considered for muscle speed and we are going through it in this chapter.

2.1 Muscle Fiber Types

The skeletal muscle fibers are not all the same. Traditionally they were categorized depending on their vary color to red fibers and white fibers.
Red fibers contain high levels of myoglobin and oxygen and store proteins. They have a red appearance and tend to have more blood vessels than white ones. While the white fibers have a low content of myoglobin and their appearance is white.

But in our view the speed of muscle is more important. So we use another classification.

Fast twitch fibers

In fast twitch fibers, myosin can split ATP very quickly. Fast twitch fibers demonstrate a higher capability for electrochemical transmission of action potentials and rapid level of calcium release and uptake. They rely on a well developed short term, glycolytic system for energy transfer and can contract and develop tension at 2-3 times the rate of slow twitch fibers.

Slow twitch fibers

The slow twitch fibers generate energy for ATP re-synthesis by means of a long term system of aerobic energy transfer. They tend to have a low activity level of ATPase a slower speed of contraction with a less well developed glycolytic capacity. They contain large and numerous mitochondria and with the high levels of myoglobin that gives them a red pigmentation, they have been demonstrated to have high concentration.

Combining two categorized fibers gives three fundamental types of muscle fibers. In the following paragraphs the type and a brief characteristic of them have been mentioned:

1. **Type I**
   Red fibers, slow twitch (also called slow oxidative or fatigue resistant fibers) contain large amount of myoglobin, many mitochondria, many blood capillaries. Generate ATP by aerobic system. Split ATP at slow rate, slow contraction velocity. It is resistant to fatigue. Needed for aerobic activities like long distance running.

2. **Type IIa**
   Red fibers fast twitch (fast oxidative or fatigue resistant fiber) contain large amount of myoglobin, many mitochondria many blood capillaries. High capacity

for generating ATP by oxidation. Split ATP at a very rapid rate and hence high contraction velocity. Resistant to fatigue but not as much as type I
(This type of fiber is needed for sports such as middle distance running and swimming.)

3. **Type IIb**
   White fibers, fast twitch (fast glycolytic, fatigable fibers) contain low myoglobin few mitochondria few blood capillaries large amount of glycogen. Split ATP very quickly. Fatigue easily
   (This type of fiber is needed for sports like spinting.)

Individual muscles are a mixture of 3 types of muscle fibers (type I, type IIa, type IIb) but their proportions vary depending on the action of that muscle.

The proportion of muscle fibers in different muscle of a body are different too. For example muscle of neck, back and legs have a higher proportion of type I fibers. Muscles of the shoulders and arms are not constantly active but are used intermittently, usually for short periods of time, to produce large amounts of tension such as lifting and throwing These muscles have a higher proportion of type I and IIb fibers.
The interesting point in type of fibers is that various type of exercise can bring about changes in the fibers in the skeletal muscle. Endurance type exercise, such as running or swimming, cause a gradual transformation of type IIb fibers into type IIa fibers the transformed muscle fibers show a slight in crease in diameter mitochondria, blood capillaries, and strength. Endurance exercise. Result in cardiovascular and respiratory changes that cause skeletal muscles to receive better supplies of oxygen and carbohydrates but do not contribute to muscle mass.
On the other hand, exercises that require great strength for short periods of time. Such as weight lifting, produce an increase in the size and strength of type IIb fibers. The increase in size is due to increased synthesis of thin and thick filaments.

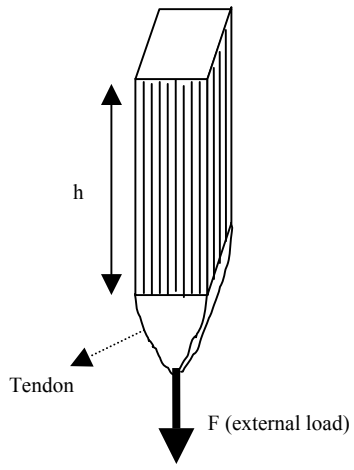
It must be remembered that although skeletal muscles is a mixture, can only have one type of muscle fiber within a motor unit.
The different skeletal muscle fibers in a muscle may be used in various ways, depending on need. If only a weak contraction is needed to perform a task, only type I fibers are activated by their motor units. If a stronger contraction is needed the motor units of type IIa fibers activated. If maximal contraction is required motor units of type IIb fibers are activated as well activation of various motor units is determined in the brain and spinal cord.

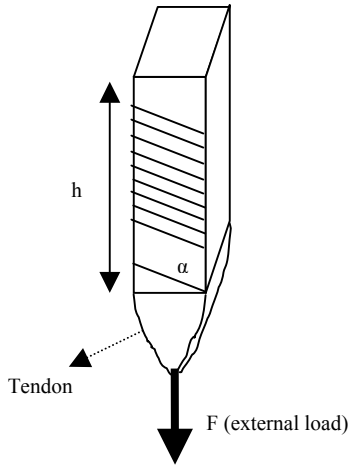
In addition to the type of muscle fibers the muscle geometry affects muscle behavior especially on force and velocity of muscle contraction.

2.2 Geometry

As explained produced force from muscles is obtain from contraction of sarcomeres. In order to have more force, the number of parallel muscle fibers should increase.
But it is not reasonable to have a thicker muscle wherever we need a stronger motion. (It made bodies hulk and out of shape)

Obviously, one of the ways is using different joints to get appropriate amplification of force, this subject is not covered in here.
There is another way to get more force in less volume. To explain it first we should define the Perpendicular Cross Section Area-PCSA. It is the area of a muscle which is perpendicular to muscle fibers. Definitely the wider is the PCSA, the more parallel muscle fibers exist.
To produce larger PCSA in a limited volume some muscles in body are pinnate form. In pinnate muscles fibers are oblique to long axis of the muscle.
Consider two muscles with same type of muscle fibers, same volume and same length but different geometry, one with straight and the other with pinnate geometry.
The stress of muscle fibers are equal ($\sigma = 2\times10^5$ N/m^2) also the strain of muscle fibers are considered equal (ε).
In the following calculation the force which they can generate has been compared.





$$PCSA_{straight} = \frac{V}{h}$$

$$F_{straight} = \sigma \times PCSA_{straight} = \frac{\sigma \times V}{h}$$

$$\varepsilon = \frac{\Delta l_{straight}}{l_{straight}}$$

$$\Delta h = \Delta l_{straight}$$

$$U_{straight} = \frac{\Delta h}{\Delta t} = \frac{\Delta l_{straight}}{\Delta t}$$

$$U_{straight} = \frac{l_{straight} \times \varepsilon}{\Delta t}$$

$$PCSA_{pinnate} = \frac{V}{h \times Cos\alpha}$$

$$F_{pinnate} = \sigma \times PCSA_{pinnate} = \frac{\sigma \times V}{h \times Cos\alpha}$$

$$F_{pinnate} \propto \frac{1}{Cos\alpha}$$

$$\varepsilon = \frac{\Delta l_{pinnate}}{l_{pinnate}}$$

$$\Delta h = \Delta l_{pinnate} \times Cos\alpha$$

$$U_{pinnate} = \frac{\Delta h}{\Delta t} = \frac{\Delta l_{pinnate} \times Cos\alpha}{\Delta t}$$

$$U_{pinnate} = \frac{l_{pinnate} \times \varepsilon \times Cos\alpha}{\Delta t}$$

It is a great advantage to have a higher force in the same volume by the factor of (1/Cosα) while α is the angle of oblique muscle fibers. Obviously the contraction of muscle along the h vertex, decreases by the factor of Cosα. But great disadvantage is the decline in speed of contraction. Consider an equal time interval for contraction of two kinds of muscles (Δt) not only the factor of Cosα but also the shorter length of muscle fibers ($l_{pinnate}<l_{straight}$) cause to have a very slow contraction in pinnate type muscle. So the pinnate muscles are suitable for those part of body which needs high force in limited volume and speed is not so important. There are a lot of pinnate muscles in two sides of substantial joints in body that are in form of ball and bowl like shoulder.

3. Model of Muscle Contraction

In order to describe the behavior of a muscle, it's good to find out a model.
The contraction of a muscle can be modeled as a black box (because we have not any idea about its function yet). The tendons and the elastic property of the myosins and actins and the cross-bridges can be considered as a series elastic element. The connective tissue that runs in parallel to the myofibrils and is ultimately connected to the tendon behaves like a parallel elastic element.
Also muscle cells are essentially bags of jells, when the contractile material is activated. The cell shortens becoming fatter as the cytoplasm redistributes spatially. This viscous fluid is resistant to movement in a velocity dependent fashion. The faster the change in length or shape the larger the resistive force. A visco elastic element is the most suitable component for modeling this effect so the total model would be somehow like Fig12

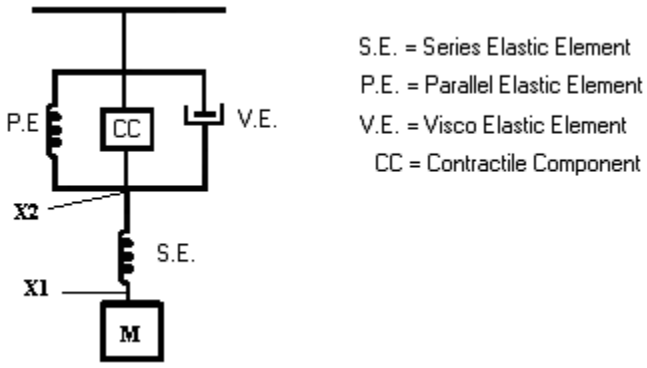


Fig 12: The Model of a muscle

The main problem is that the behavior of contracting element is not modeled yet but gathering the result of several experiments can help to figure out its characteristic.

3.1 Experiments

To obtain the characteristic of the contraction element of muscle it is need to run some experiments. The traditional form of experiment is to use a muscle of frog's leg that has been connected to the apparatus to investigate its behavior. For running these tests on human muscle a special measuring device is needed to measure the force.

3.1.1 Response to an electric pulse – Isometric condition

In this experiment a muscle should be kept in a fixed length then the force that it can generate by stimulation should be measured
The result can be seen on the fig 13.

3.1.2 Response to an electric pulse –Isotonic experiment

The main aim of doing this experiment is to measure the displacement in fix load situation when it is stimulated. The result is somehow like fig 13 but the y-axis is the length of muscle.

Some results can be derived from these two experiments

1. There is a delay between electrical stimulation and reaction of muscle. This delay is in range of few milliseconds. In the first glance it seems that it can be a limited parameter in response of a muscle but as the appropriate pulse for stimulation of a muscle emits from brain, in repetitive activities like running, flapping for birds and so on the command can be sent to the muscle just a few millisecond before the exact time of muscle motion in the form that the motion starts just the time which it should move. Engineers have applied such a technique in sparking system of a combustion engine. The combustion starts a bit sooner than the time that whole fuel should be compressed completely in cylinder. Hence the maximum force will generate at the time that piston starts to go back.
2. The maximum force or maximum shortening occurs at approximately 50msec after stimulation. As explained it is not important in repetitive motion but for single and fast reaction like a reaction of a driver to push the break 50msec may be too much
3. The stimulation of a muscle by a short single pulse (this pulse can be generate by nerve or an electric pulse) produced a twitch.
   This is a short spasm of contraction that rapidly generates a small amount of force and then declines to zero over a large period.

The result of experiments can be a good criteria for muscle behavior but it should be remembered that the response of muscle depends on the length of muscle, type of muscle, the cross section of muscle, the number of motor units that are active while stimulation. The last item itself depends on the amplitude of pulse and its duration. The experiment shows that the strength of the twitch increases with the increase in the amplitude of pulse. This is due to the recruitment of increased numbers of muscle fibers that are involved in the twitch. Electromyography of a stimulate muscle shows that if a muscle is contracting very weakly only a single motor unit may be activated As the tension is increased additional motor units get recruited. The order in which motor units get recruited is not random, smaller motor units get recruited first followed by larger ones. This makes sense because small motor units are used for fine control which is required at low forces. At higher forces small changes in force are not necessary. If big motor units fired off force you would not be able to apply very small forces at all.
By increasing the frequency of pulses if the second pulse is applied before muscle relax completely the effect of second stimulation will add to the pervious one This is called summation. With a faster train of stimulation a higher plateau is reached and the force curve (tension) is now smooth. This is known as tetanus. The frequency of stimulus required for smooth response is the fusion frequency.
The fusion frequency is about Hz 60–90 Hz for the red fibers and at 70–110 Hz for the white fibers.
The electrical pulse which brain sends to muscles includes the train pulses to get smooth force response.

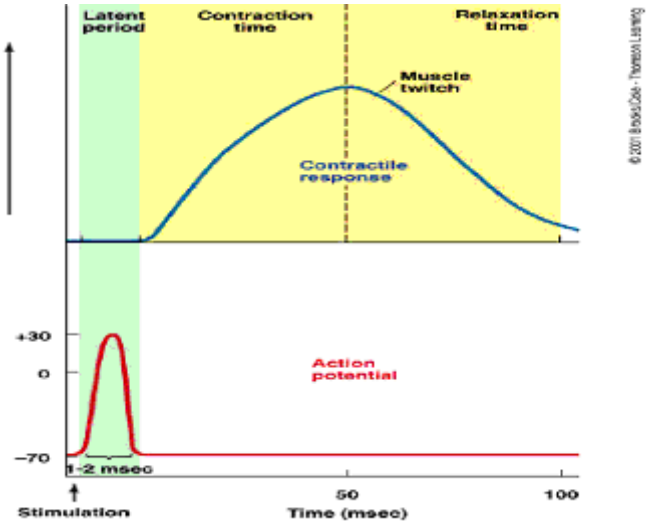


Fig 13: The response of muscle to an electric pulse

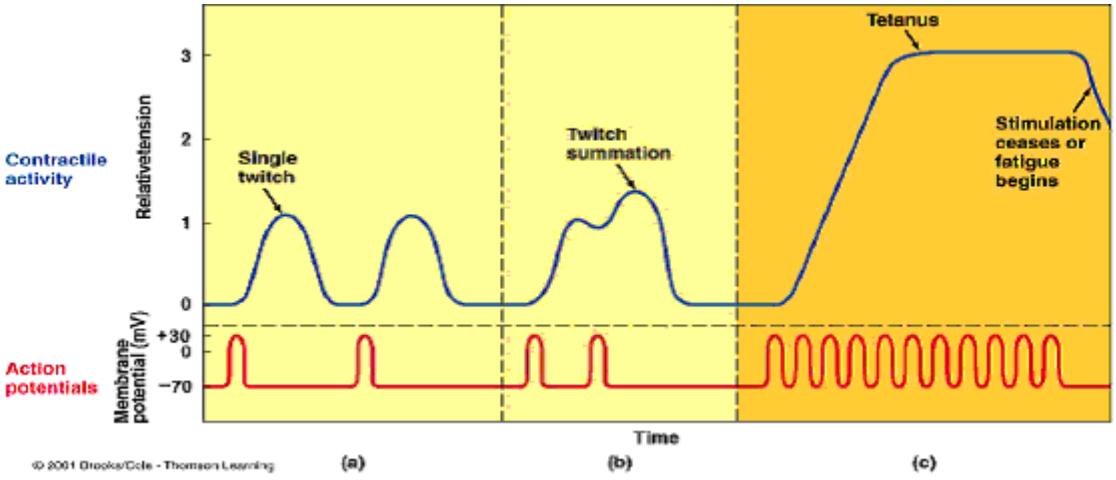


Fig 14: The response of muscle to a train of pulses

Another data which is required is according to the form of contraction. It seems that contraction is equal to shortening but some time lengthening occurs while muscle is contracting.

3.1.3 Concentric contraction (muscle actively shorting) Force-Velocity relation

When a muscle is activated and required to lift a load which is less than the maximum titanic tension it can generate the muscle to shorten are referred to as concentric contraction. In concentric contractions, the force generated by the muscle is always less than the muscle maximum tension. By performing a series of constant velocity shortening contractions, a force-velocity relationship can be determined.

3.1.4 Eccentric contraction (muscle actively lengthening) Force-Velocity relation

During normal activity, muscles are often active while they are lengthening like setting an object down gently (the arm muscles must be active to control the fall of the object) As the load on the muscle increases, it finally reaches a point where the external force on the muscle is greater than the force that the muscle can generate. Thus even though the muscle may be fully activated, it is forced to the lengthening due to the high external load. This is referred to as an eccentric contraction.
In eccentric contraction two points are very important.

1. The absolute tension achieved are very high relative to the muscle's maximum titanic tension generating capacity (you can set down a much heavier object than you can lift)
2. The absolute tension is relatively independent of lengthening velocity.

This suggests that the skeletal muscles are very resistant to lengthening.
The basic mechanics of eccentric contractions can't be explained by theory of cross-bridges of actins and myosins.

The mathematical form of the force-velocity relationship in concentric contraction is a rectangular hyperbola and is given as:

$$(P+a)\ v = b\ (Po-P)$$

Where a and b are constants derived experimentally (usually about 0.25), P is muscle force, Po is maximum tetanic tension, and v is muscle velocity. This equation can be used to determine the relative muscle force that occurs as a muscle is allowed to shorten. It is important to note that the force-velocity relationship is a steep rectangular hyperbola. In other words, force drops off rapidly as velocity increases. For example, in a muscle that is shortening at only 1% of its maximum contraction velocity (extremely slow), tension drops by 5% relative to maximum isometric tension. Similarly, as contraction velocity increases to only 10% maximum (easily attainable physiologically), muscle force drops by 35%. Note that even when muscle force is only 50% maximum, muscle velocity is only 17% Vmax.

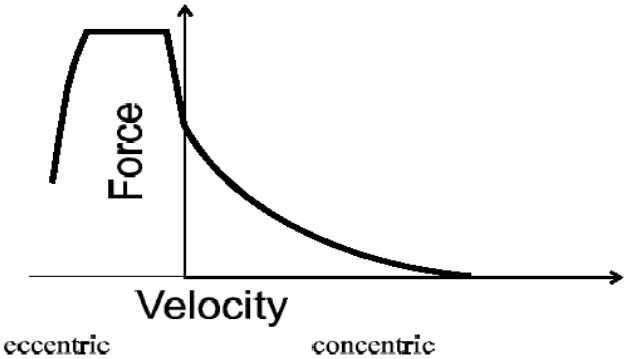


Fig 15: Force-Velocity of muscle in Concentric and Eccentric contraction

Combining the force-length and force-velocity graphs of two experiments give a 3D plot (fig16) that for any amount of load and strain, we can obtain its velocity. But it should be remembered that this graph is a sample muscle with a specific rest length and a mixed of muscle fibers.
Important point in this graph is the point in which we can get the maximum force and that is in zero velocity for concentric contraction and about half of the full strain.

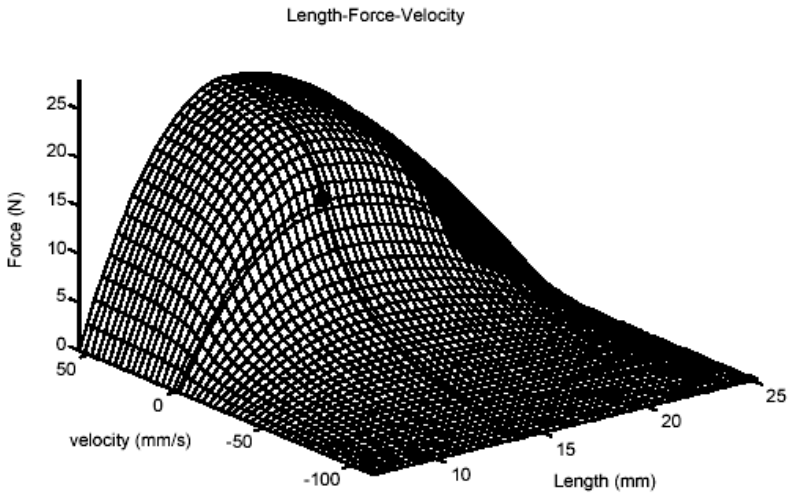


Fig 16: Force- Velocity-Length relation (for a sample muscle)

3.1.5 Passive stretching- Muscle passively lengthening

In passive state muscle is stretching while it is not active. The characteristic of lengthening as indicated in fig17 is exponential form.

The structures responsible for passive tension are outside of the cross bridge itself since muscle activation is not required. The source for passive force bearing in muscle was within normal mitibrillar structure.

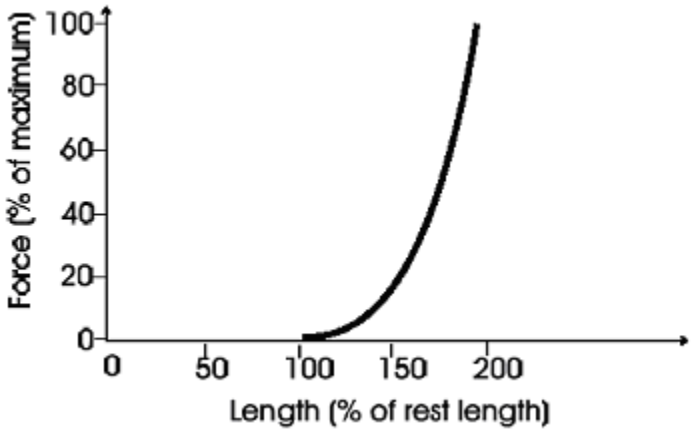


Fig 17: Force- Length plot of a muscle in passive condition

Now we can complete the model of contraction element of muscle the fig18 indicates the characteristic of contractile component in active tension and passive tension

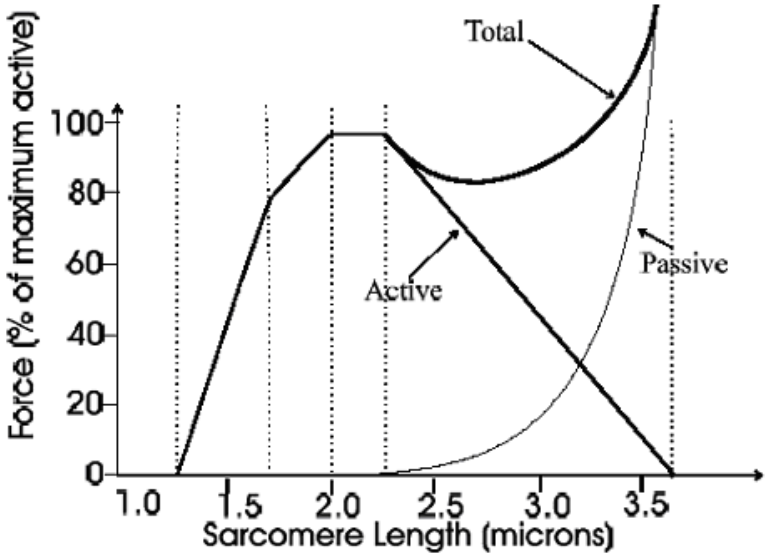


Fig 18: Force- Length plot of a sarcomere in active and passive situation

Obviously close to normal length of a muscle a larger change in tension is needed to get a fixed amount of shortening in compare to pre-contracted muscle.
For a muscle the relation between force and length is like fig18 only the scale of graph changes to the length of muscle.

4. Calculation

4.1 Complete Model

Now we have the characteristic of all components of the model.

To derive the contraction speed of a muscle, a differential equation can be written for the displacement M in fig12. The answer of that gives the x=g(t) then calculating x' would be the speed of contraction and putting x"=0 gives the point that there is the maximum speed.
So the first step is writing the differential equation in point x1 and x2 in base of balance of forces.

Assumption:

- Kp and Ks are the coefficient of parallel and serial springs respectively.
- M is the mass of muscle and the load.
- μ is the coefficient of visco elastic part.
- f(x) would be the force function of contract element versus displacement that has been obtained by experiment (fig18)

Although the f(x) is obtained in the experiment but in order to use for differential equation first we try to derive the equation of f(x).
Looking at fig18 gives an idea to separated the function to two sections:

1. The first part which is according to contraction of sarcomere is some how like a square function with symmetry around zero displacement. For convenience the contraction has considered from half-length of muscle to 1.5 times of muscle length. So the function can be written in the form of:

$$0 \le f_1(x) \le F_{\max} \quad or \quad 0.5l_0 \le l \le 1.5l_0 \quad or \quad -0.5l_0 \le x \le +0.5l_0$$

$$f_1(x) = -A \times x^2 + B$$

$$x = 0 \Rightarrow f_1(0) = F_{\max} = B$$

$$f_1(0.5 \times l_0) = 0 = -A \times (0.5 \times l_0)^2 + F_{\max} \Rightarrow A = \frac{4 \times F_{\max}}{l_0^2}$$

$$f_1(x) = F_{\max}\left(-\left(\frac{2x}{l_0}\right)^2 + 1\right) \qquad -0.5l_0 \le x \le +0.5l_0$$

2. The second part which is like an exponential function can be in form of:
$0 \le f_1(x) \le F_{\max}$ *or* $l_0 \le l \le 1.5l_0$ *or* $0 \le x \le +0.5l_0$

$$f_2(x) = (e^{\frac{x}{\lambda}} - 1)$$
$$f_2(0.7 \times l_0) = e^{\frac{0.7l_0}{\lambda}} - 1 = F_{\max}$$
$$\lambda = \frac{0.7l_0}{Ln(F_{\max} + 1)}$$

The force-displacement function of whole element is the summation of $f_1$and f_2:
$$f(x) = f_1(x) + f_2(x)$$
$$f(x) = -F_{\max}(\frac{2x}{l_0})^2 + e^{\frac{x}{\lambda}} + F_{\max} - 1 = Ax^2 + e^{\frac{x}{\lambda}} + D$$

Now we can write the force balance equations at points x_1 and x_2.
At x_1 :
$$M\ddot{x}_1 = K_S(x_1 - x_2)$$
$$x_1 = x_2 + X \times Sin(\omega t)$$
$$\omega = \sqrt{\frac{K_S}{M}}$$

At x_2:
$$K_S(x_2 - x_1) = K_P x_2 + \mu\dot{x}_2 + f(x_2)$$
$$X \times Sin(\omega t) = K_P x_2 + \mu\dot{x}_2 + Ax_2^2 + e^{\frac{x_2}{\lambda}} + D$$

Solving this equation is too complicated than my expectation
Definitely in order to have a good estimation it is necessary to know the Ks, Kp and μ.
Therefore solving this equation doesn't give much information. Another way is to simplify the model of muscle.

4.2 Simplifying the Model

One easy way to calculate the maximum velocity of muscle contraction is using the Newton's first law. According to the experiments the maximum velocity of muscle obtains in no load condition. Therefore the contraction force which muscle can generate cause to accelerate the mass of muscles and bones. We can consider a muscle out of body in order to reach the maximum velocity by neglecting the mass of bones and other parts.

If the muscle has the volume of V with initial length of L_0, cross section area of A (a straight muscle with PCSA=A has considered in this example because it has the highest speed among different geometry of muscles), and the stress that a muscle can generate will be indicated by σ (approximately for all muscles σ is constant and its value is about 2×10^5 N/m^2 , then:

$$\left.\begin{aligned} F &= ma \\ F &= \sigma \times A \\ m &= \rho \times V = \rho \times L_0 \times A \end{aligned}\right\} \Rightarrow \sigma \times A = \rho \times L_0 \times A \times a \Rightarrow a = \frac{\sigma}{\rho \times L_0}$$

If the motion of muscle is considered to have a constant acceleration (Although it is not a good assumption, the short length of contraction can give a good approximation to have a constant acceleration)

$$\left.\begin{aligned} U &= \sqrt{2 \times a \times \Delta L} \Rightarrow U = \sqrt{\frac{2 \times \sigma \times \Delta L}{\rho \times L_0}} \\ \Delta L_{\max} &\rightarrow U_{\max} \\ \Delta L_{\max} &= 0.4 \times L_0 \end{aligned}\right\} \Rightarrow U_{\max} = \sqrt{\frac{2 \times \sigma \times 0.4}{\rho}} = \sqrt{\frac{0.8 \times \sigma}{\rho}}$$

$$\left.\begin{aligned} \sigma &= 2 \times 10^5 \, N/m^2 \\ \rho &= 1000 \, Kg/m^3 \end{aligned}\right\} \Rightarrow U_{\max} = \sqrt{\frac{0.8 \times 2 \times 10^5}{1000}} = 12.6 m/s$$

Obviously, it is the maximum speed that a muscle can reach for a while. But this value is higher than real value because:

1. Before muscle reaches to the maximum contraction it should decelerate.
2. The effect of elastic parts and visco-elastic have not been considered.
3. At first step we considered that force which cause to accelerate the muscle can have the maximum possible value (σ×A). But according to the 3D plot (fig16) the maximum force obtains while external force would be zero. It is meaning less to consider the maximum force then try to calculate the maximum velocity.

4. The value of force has been considered fix, while fig9 indicates that the maximum force obtains at zero contraction, and more the muscle contracts the less force generates.
5. The effect of mass of bones and external loads degrades the velocity and the cross section area which has been canceled in equation appears again.

$$\left.\begin{aligned} F &= (m + M_e)a \\ F &= \sigma \times A \\ m &= \rho \times V = \rho \times L_0 \times A \end{aligned}\right\} \Rightarrow \sigma \times A = (\rho \times L_0 \times A + M_e) \times a \Rightarrow a = \frac{\sigma \times A}{\rho \times L_0 \times A + M_e}$$

$$U = \sqrt{2 \times a \times \Delta L} \Rightarrow U = \sqrt{\frac{2 \times \sigma \times A \times \Delta L}{\rho \times L_0 \times A + M_e}}$$

As a result of the last equation, speed of contraction decreases by increasing the external load of muscle and initial length of muscle.
Anyway By this calculation we have at least an upper limit that makes sure that there is not any muscle which can contract faster than 12.6 m/s. To make a sense that how close maybe the calculated value would be, we can compare it with the result of twitch experiment.

Consider the bicep muscle in approximate length of 20cm (the tendons have not be considered). It can contract up to 15cm so ΔL=5cm. According to the fig13 it takes about 50msec to reach the maximum tension so the speed may be around
$V = \Delta L / T = 0.05 / 0.05 = 1$ m/s
It is less than of 0.1 times of calculated value. But in final equation that we know that stress is not as high as the maximum stress and the mass of M_e the maximum velocity degrades maybe to a reasonable value.

5. Conclusion

The speed of contraction of a muscle is related to different items.
In this short report, it has been tried to mention these items while each of them is a separated subject with different aspects.

The related items are:

a. Types of muscle fibers
b. The percent of Shortening – $\Delta L/L_0$
c. The initial length of muscle
d. The PCSA of muscle
e. The load or the force which is expected to be generated
f. The geometry of muscle
g. The number of motor units
h. The delay between commands from nerve system and actuation

In the simplified model all related items didn't consider and calculation showed that the maximum speed can be 12.6m/s. The first deduction is that in any case it is impossible to have a muscle with velocity of more than 12.6m/s. In addition actual test indicated that contraction speed is about 1m/s. As contraction by electric pulse generates a high speed contraction while nerve system in body can't stimulate the muscle in such a speed, we can consider the 1m/s as the maximum practical speed of contraction.

Appendix 1: More Questions

Along the study for this report I faced to several points that I didn't find appropriate answer for them. May it would be interesting to work on them, too.

- It is supposed that while a sarcomere is contracting the z discs are getting closer by cross bridge between actins and myosins. It has been mentioned that for a uniform contraction there are four groups of myosins in one sarcomere that they are acting in phase delay. So in each moment there are a group of myosins that are pulling the actins and consequently a uniform force will be produce. But if we accept this theory, then there won't be any contraction at all.
  Consider two adjacent sarcomeres in series, the z-disc between them is pulling to one side by a sarcomere while it is pulling to the opposite side by other sarcomere in the same force. Hence the z-disc doesn't move. Similarly for the other z-disks there won't be any motion. So contraction is impossible.

  This theory may be modified by another assumption. There should be two kinds of myosin heads in a sarcomere each of them in one end of myosin. We call them Odd and Even sides. If we presume that in odd side of myosin, myosin head are in stroke (a) and (c) (fig 3) while in the even side there are only stroke (b) and (d) at the same time.
  The z disc is pulling toward the odd side, because myosin heads which are in stroke (a) are pulling the z disk while the other side of z disk is a sarcomere which myosin head of them is not in phase (a). in the next stroke myosin head in odd side goes to strokes (b) and (d) and in even side there are (a) and (c) phases. So z disc moves toward even side. It seems that z disc is going right and left alternatively but in each stroke the distance between two adjacent z discs has reduced in few nano-meters.
  But it is only a theory that an engineer might be considered in designing of an actuator. It needs more evidence to prove it.

- It is believed that the maximum stress that a muscle can generate is about 2×10^5 N/m^2 . Consider a sarcomere with diameter of 1μm. It can generate this amount of stress, too. So the force which it can generate is about:
  $F = \sigma\times A = 2\times10^5\times0.785\times10^{-12}$ N $= 1.5\times10^{-7}$ N
  We know that the force that myosin head can generate is about 2pN. Hence
  The number of active myosin head in one side of myosin$=1.5\times10^{-7}/2\times10^{-12}$
  Total number of myosin head = 4 × The number of active myosin head
  Total = 3×10^5
  But observation indicates about few thousands myosin heads should be exist in one sarcomere.

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