Slide 1

Neuromusculoskeletal Modeling

Slide 2

Why are muscle forces important?

- Moving (walking, running, waving, …)
- Talking
- Breathing
- Seeing
- Hearing
- Digesting (smooth muscle)
- Pumping (cardiac muscle)

Description of muscle functions.

Slide 3

Muscle and Tendon Properties Influence Performance

Muscle strength and the rate at which muscles contract are major determinants of running speed

Kangaroos can run more efficiently at fast speeds than at slow speeds, partly because of the compliance of their tendons

Understanding how muscles can impact an action is crucial for many applications. When we talk about a muscle we mean the whole structure (fibers, and tendon).

What is the role of the fibers?
What is the role of the tendon?
Why we have many muscles spanning a single joint?
Why does the musculo-tendon path maters (tendon routing)?
How muscles are controlled by the Central Nervous System (CNS)?
What muscles contribute to joint acceleration (training)?

These are some question that we can answer by modeling and simulation.

Slide 4

Facts

- Strong quadriceps can lift a small car off the ground

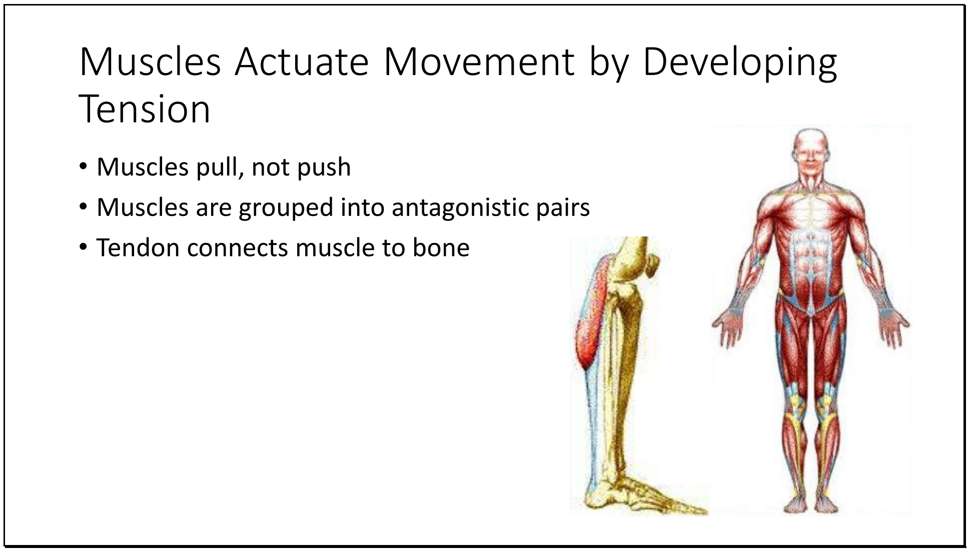
$$F_o^M \approx 10{,}000\,N \approx 1{,}000\ kg$$

$$VW\ \ Bug \approx 1{,}180\ kg.$$

- Joint contact forces:
  - 3 * Body Weight during walking
  - 5 * Body Weight during running

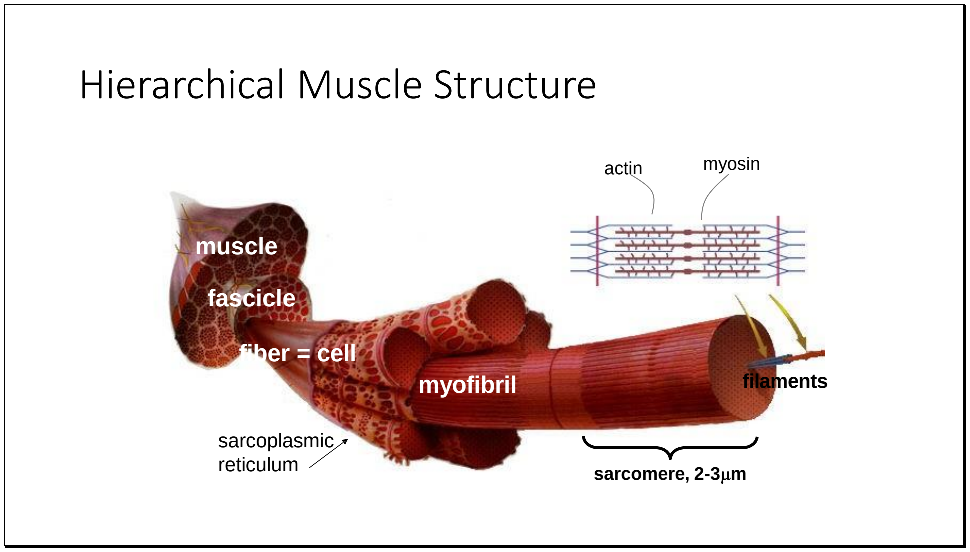
Muscles are strong and efficient.

Slide 5



Muscles can pull, not push (different from artificial actuators). Is this a problem and how we solve it (our body)? -> agonist antagonist

Slide 6



Muscle hierarchy:
Muscle (*whole organ*)
Fascicle (*portion of muscle*): Bunch of Fascicles = Muscle
Muscle Fiber (*single muscle cell*): Fascicle = Bunch of Muscle Fibers
Myofibril (*muscle cell organelle*)
Sarcomere (*portion of myofibril*)
Myofilament (*part of sarcomere*)

Sacromere are the contractile units and contain thin filaments with actin and myosin

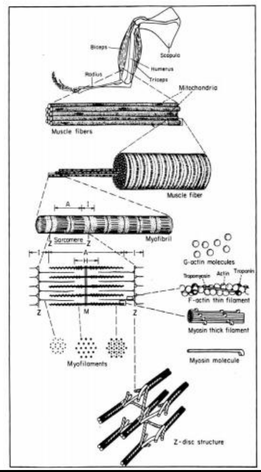
Muscle cells contain protein filaments of **actin** and **myosin** that slide past one another, producing a contraction that changes both the length and the shape of the cell.

The sarcoplasmic reticulum is a specialized type of smooth endoplasmic reticulum that regulates the calcium ion concentration, which is responsible for the initiation of the contraction.

Slide 7

Muscle Force Results From Interaction Between Contractile Proteins

- Sarcomere: the smallest anatomical unit that contracts like a muscle
- Sliding filament model proposes that muscle force arises from cyclic binding between thick and thin filaments of the sarcomere
- Thin filaments contain actin, troponin C, and tropomyosin
- Thick filaments contain myosin
- In the absence of calcium, tropomyosin prevents myosin from attaching to actin

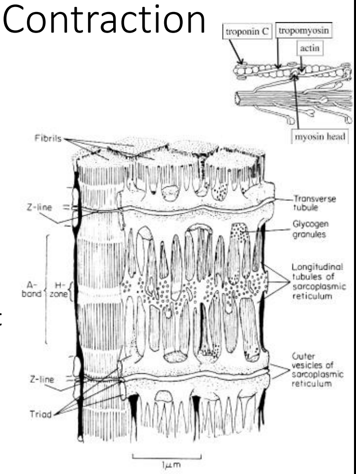


How a muscle produces force

Slide 8

Calcium is Needed for Muscle Contraction

- At the onset of an action potential, the sarcoplasmic reticulum (SR; a membrane that surrounds the myofibrils) releases calcium
- Calcium binds to troponin, causing a conformal change in tropomyosin which reveals myosin binding sites on the actin
- Simultaneously, adenosine triphosphate (ATP) is hydrolyzed by ATPase in the myosin head, providing the energy for cross-bridge attachment
- The SR re-sequesters calcium at the end of the action potential, thereby inducing muscle relaxation



The chemical process of activation dynamics

Slide 9

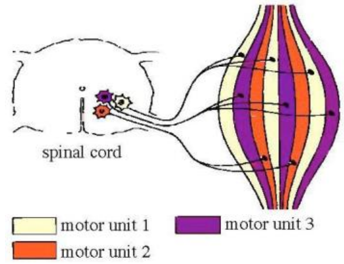
Factors Affecting Muscle Force Development

- Muscle fiber type
- Number of activated motor neurons, frequency of discharge
- Muscle length
- Velocity of shortening/ lengthening
- Muscle geometry (physiological cross-sectional area (PCSA), angle of pennation)

Slide 10

Muscle-Nerve Interaction

- A motor nerve enters muscle and splits into numerous axons; each axon contacts 10- 2000 muscle fibers
- Each muscle fiber is innervated by only one motor nerve axon, and contracts in response to an action potential in that axon
- Motor unit: a single motor nerve axon and all the muscle fibers it contacts

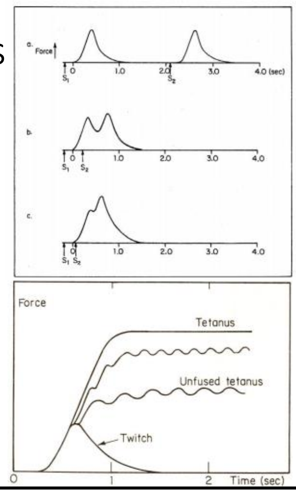


muscle	# muscle fibers	# motor units	av. fibers per motor u.
platysma	27,100	1,100	25
Brachioradialis	130,000	330	410
Tibialis anterior	250,000	450	600
gastrocnemius	1,120,000	580	2,000

Slide 11

Stimulation Frequency Affects Muscle Force: Twitch and Tetanus

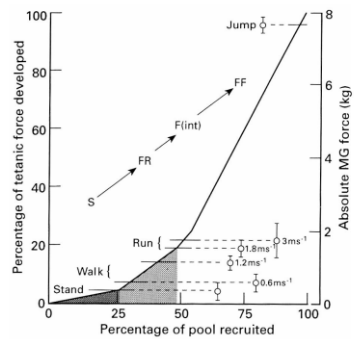
- Muscle force can be modulated by varying: (1) the number of recruited motor neurons, and (2) the frequency of discharge (i.e., stimulation rate) in motor neurons
- A single action potential (S1) produces a twitch contraction, a quick rise and slow fall in force
- A tetanus occurs when a new action potential (S2) arrives before the previous twitch has dissipated, and there is force summation
- At stimulation frequencies >30/s, there are no twitch transients (fused tetanus)



Slide 12

Size Principle

- When a stimulus is applied to the ventral aspect of the spinal cord, the smallest and most excitable motor units are activated first. These tend to be slow (S) motor units which innervate slow oxidative (SO) muscle fibers. Larger FR and FF motor units that innervate FOG and FG fibers are recruited only at high levels of force
- Sequence is reversed when force level falls, with largest motor units dropping out first



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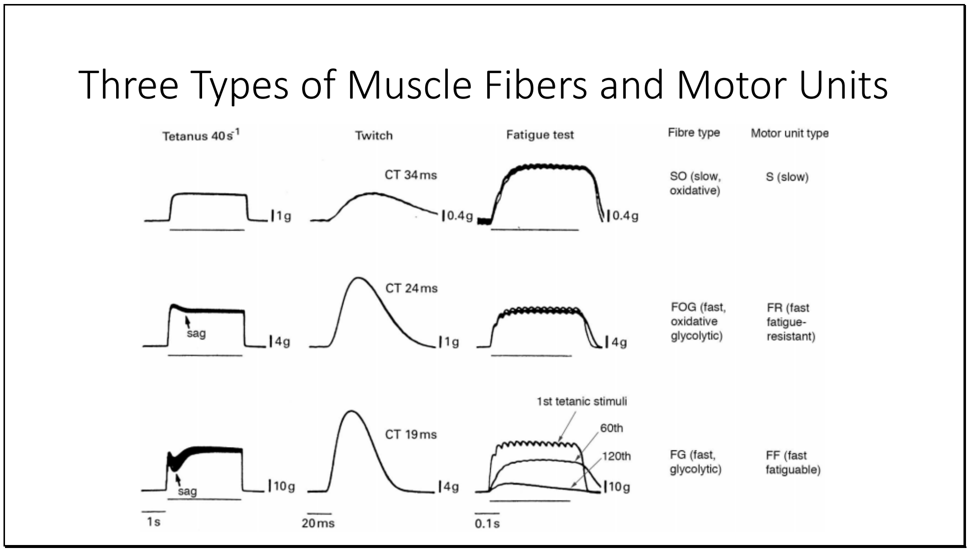

The importance of the different muscle fiber types is to understand what kind of fibers are recruited and why for each action.

Muscle twitch: a brief, contractile response of a skeletal muscle elicited by a single maximal volley of impulses in the neurons supplying it
Tetanus (Tetanic contraction): a tetanic contraction (also called tetanized state, tetanus, or physiologic tetanus, the latter to differentiate from the disease called tetanus) is a sustained muscle contraction evoked when the motor nerve that innervates a skeletal muscle emits action potentials at a very high rate. During this state, a motor unit has been maximally stimulated by its motor neuron and remains that way for some time. This occurs when a muscle's motor unit is stimulated by multiple impulses at a sufficiently high frequency. Each stimulus causes a twitch. If stimuli are delivered slowly enough, the tension in the muscle will relax between successive twitches. If stimuli are delivered at high frequency, the twitches will overlap, resulting in tetanic contraction. When tetanized, the contracting tension in the muscle remains constant in a steady state. This is the maximal possible contraction. During tetanic contractions, muscles can shorten, lengthen or remain constant length. Muscles may exhibit some level of tetanic activity, leading to muscle tone, in order to maintain posture.

S or SO — Slow (oxidative) — low force, slower contraction speed, highly fatigue resistant.
FR — Fast fatigue resistant — intermediate force, fatigue resistant — fast contraction speed and resistant to fatigue
FF — Fast fatigable — high force, fast contraction speed but fatigue in a few seconds.

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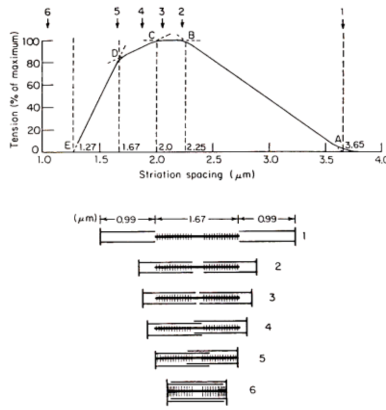
Active Force Development in the Sarcomere Depends on Actin-Myosin Overlap

- (A): no overlap between actin and myosin, zero developed tension
- Between (A) and (B): tension increases linearly as overlap increases
- Between (B) and (C): maximum overlap & maximum tension
- Left of (C): interference between actin filaments reduces ability of crossbridges to develop tension
- Left of (D): myosin filaments collide with Z-lines and fold, and force declines rapidly



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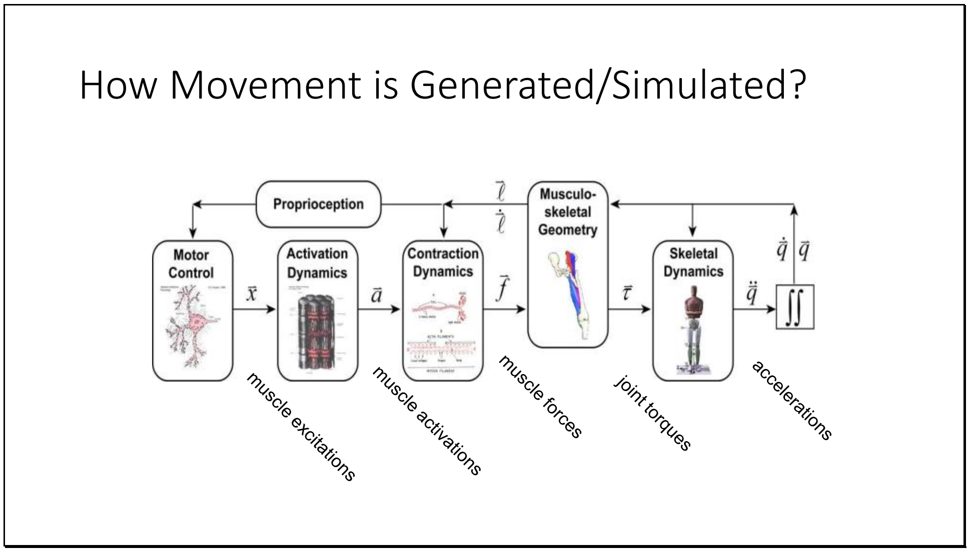

1) Movements are generated by the neural excitation to the muscles.
2) These excitations trigger some chemical reactions which cause the muscle to contract.
3) Muscles are wrapped on the bones through tendons. When the muscle contracts it pull and a force is applied on the bones.
4) These forces correspond to applied torques on the joint and produce accelerations (Newton's second law).
5) For simulation purposes we integrate the acceleration to derive the velocity and position (numerical integration).

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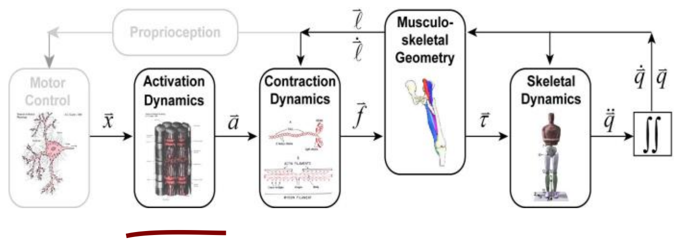
Muscle Activation Dynamics



A muscle can neither generate force nor relax instantaneously. The development of force is a complex sequence of events that begins with the firing of motor units and culminates in the formation of actin–myosin cross-bridges within the myofibrils of the muscle. When the motor units of a muscle depolarize, action potentials are elicited in the fibers of the muscle and cause calcium ions to be released from the sarcoplasmic reticulum. The increase in calcium ion concentrations then initiates the cross-bridge formation between the actin and myosin filaments. In isolated muscle twitch experiments, the delay between a motor unit action potential and the development of peak force has been observed to vary from as little as 5 milliseconds for fast ocular muscles to as much as 40 or 50 milliseconds for muscles comprised of higher percentages of slow-twitch fibers. The relaxation of the muscle depends on the re-uptake of calcium ions into the sarcoplasmic reticulum. This re-uptake is a slower process than the calcium ion release, so the time required for muscle force to fall can be considerably longer than the time for it to develop.

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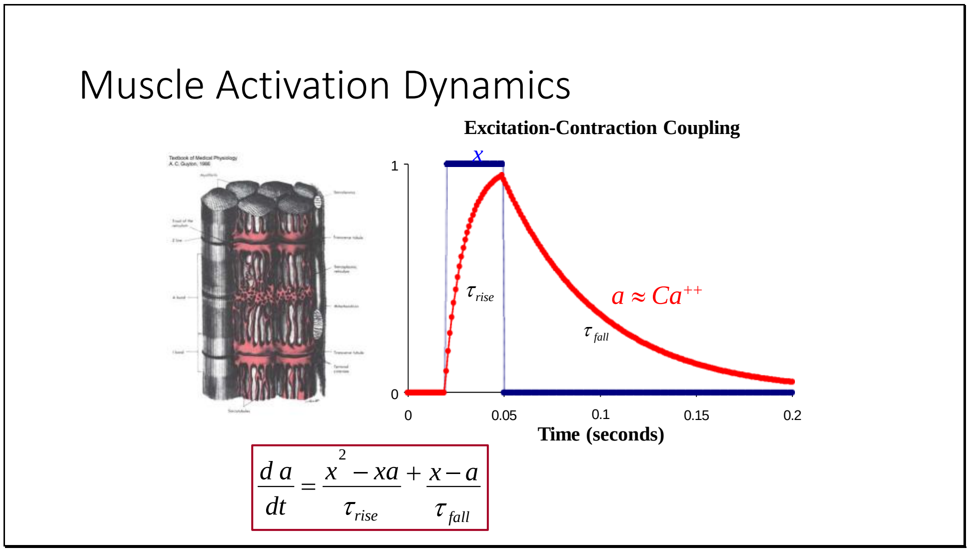

The activation dynamics of muscle can be modeled with a first-order differential equation. This equation relates the rate of change of muscle activation (i.e., the concentration of calcium ions within the muscle) to the muscle excitation (i.e., the firing of motor units).

trise and tfall are the time constants, x is the excitation and a is the activation

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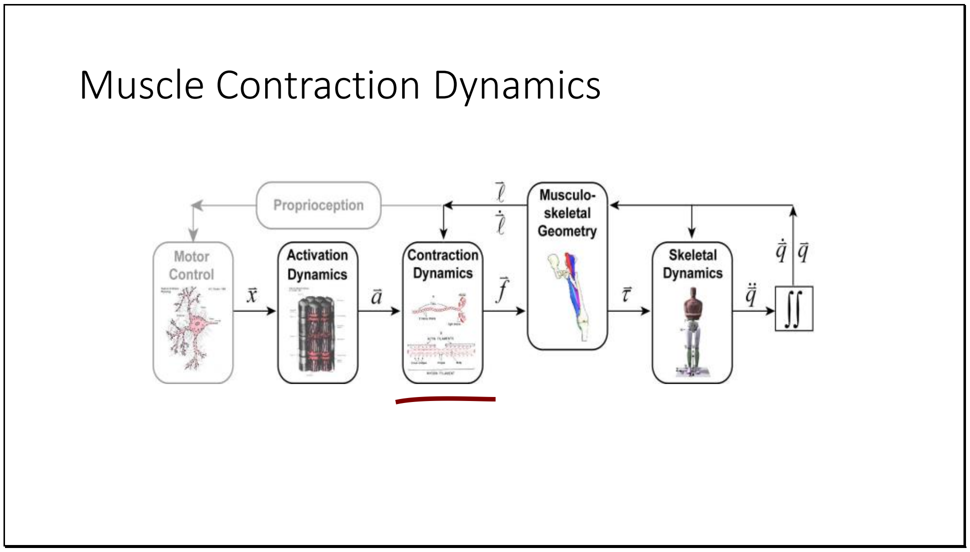

Contraction dynamics represents how the muscle produces a force. It is a complex process which is a function of multiple aspects.

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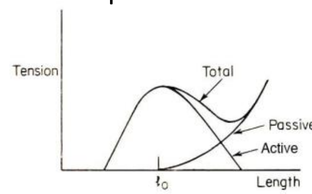
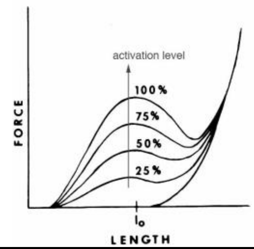
Muscle Length Affects Force Development

- The tension developed in a whole muscle is the sum of active force due to muscle contraction and passive force due the passive stiffness of tendon and muscle
- The passive force is negligible for lengths less that the normal resting length (l_0)
- The active force follows the tension-length behavior of the sarcomere, and scales with muscle activation

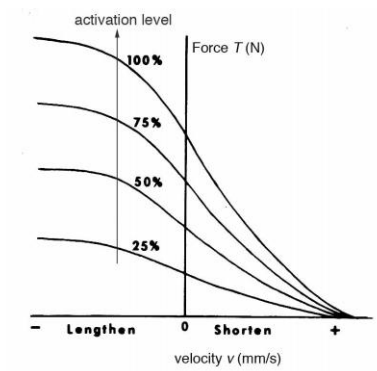




Slide 20

Muscle Velocity Affects Force Development

- Force (T) is greater during lengthening than shortening contractions
- The greater the shortening velocity (v), the smaller the force (explains why we cannot lift heavy objects quickly)
- In the shortening regime, mechanical power output is maximum when T and v are around one-third their maximum values



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Muscle Force-Velocity Behavior is Described by the Hill Equation

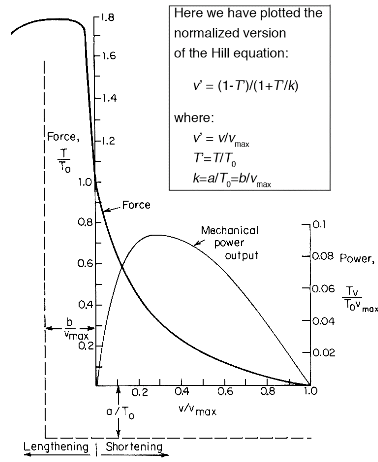
An empirical relation that describes the force-velocity behaviour of muscle during shortening is the Hill Equation

The equation can be written as:

$$(T + a)(v + b) = (T_o + a)b$$

where T_o is the isometric (zero velocity) tension, and v_{max} is the maximum (zero tension) velocity $= {bT_o}/{a}$.

The instantaneous power is given by $P = T \cdot v$.



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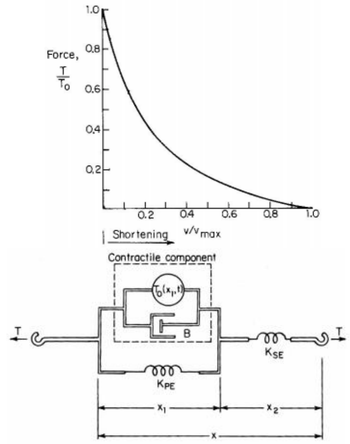
Hill’s Active State Model of Muscle Contraction

- Hill assumed:

  (1)for a given length, muscle always develops the same peak force $T_0(x_1, t)$;

  (2)if the muscle is shortening, some force is dissipated in overcoming inherent viscous resistance
- B: muscle damping constant, which must be a nonlinear function of shortening velocity and temperature
- K_{SE}: stiffness of the series elastic component; represents force- deflection properties of tendon
- K_{PE}: stiffness of the parallel elastic component; represents force- deflection properties of sarcolemma, epimysium, perimysium, and endomysium



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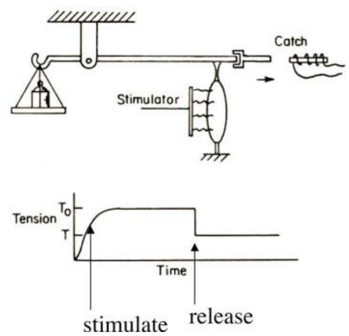
Quick Release Experiments for Determining the Hill Model Parameters

- Hold muscle length fixed with the catch
- Stimulate muscle to produce peak (isometric) force T_0
- Instantly release catch
- At the instant of release, muscle force is reduced to a value T (where $T < T_0$) that depends on weight in pan



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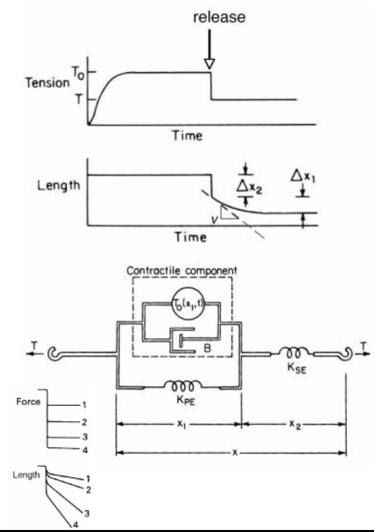
Quick Release Experiments

- There is an instant change (Δx_2) in the length of K_{SE} following release
- this is followed by a more gradual change (Δx_1) in the length of the muscle
- As T increases, there is a decrease in v (slope of dashed line), reflecting that muscle cannot shorten quickly under high loads
- combinations of T and v reflect the force-velocity properties of a given muscle



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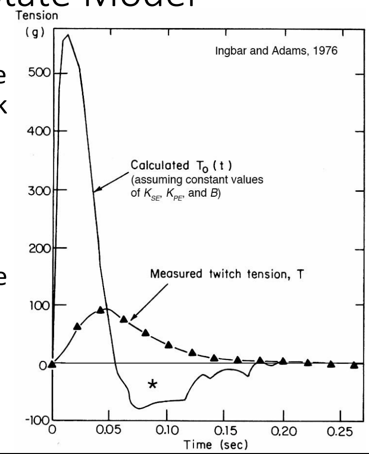
Shortcomings of The Active State Model

- The model predicts a negative T_0 during the end phase of the twitch (marked by asterisk in diagram at right)
- the liquid in muscle (water) does not have the required nonlinear damping characteristics
- the model cannot accurately predict muscle force during lengthening (the slope of the T-v curve is about 6-fold greater for lengthening than shortening, and muscle length increases rapidly when the load exceeds $1.8T_0$)



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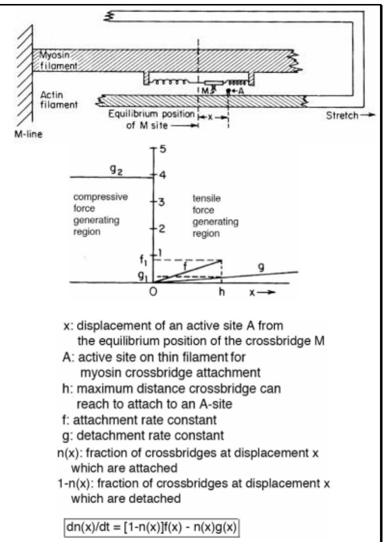
Huxley’s Sliding Filament Model

- Force development is due to stretch of elastic myosin crossbridges, which can form bonds with actin for $x < h$
- Bonds can be maintained for $x > h$ (tensile) and $x < 0$ (compressive)
- For $(0 < x < h)$, rate of attachment (f) exceeds rate of detachment (g)
- Rate of detachment (g) is slower during lengthening than shortening, thus accounting for greater force under eccentric conditions



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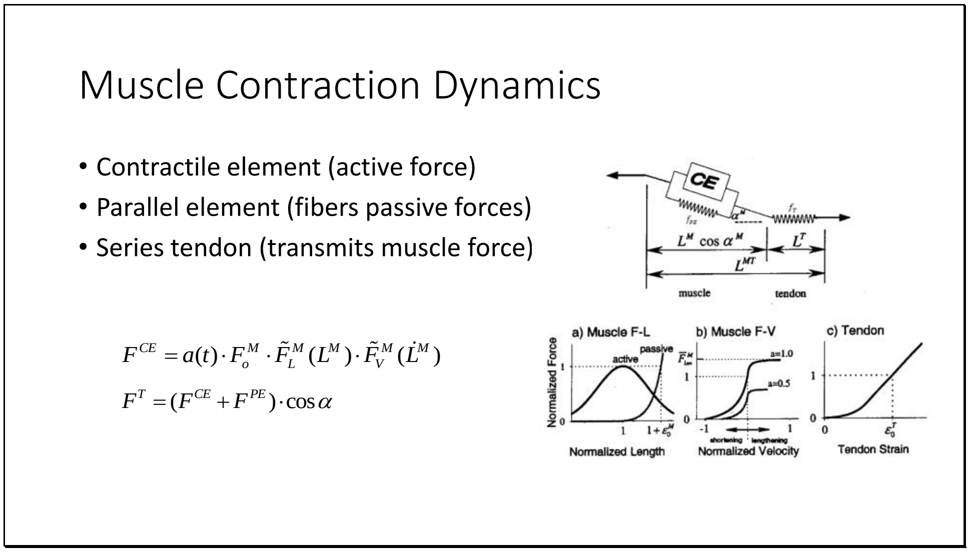

Musculotendon actuators consist of an active contractile element, a passive elastic element, and an elastic tendon. The maximum active force a muscle can develop varies nonlinearly with its length, represented by the active-force–length curve F_L, peaking at a force of F_o^M at a length of ~l_o^M (the tilde is used to denote forces, velocities, muscle lengths, and tendon lengths that are normalized by F_o^M, v_{max}^M, l_o^M and l_s^T, respectively). During non-isometric contractions, the force developed by muscle varies nonlinearly with its rate of lengthening, which is represented by the force–velocity curve F_V. Force is also developing when a muscle is stretched beyond a threshold length, regardless of whether the muscle is activated, which is represented by the passive-force–length curve F_{PE}.

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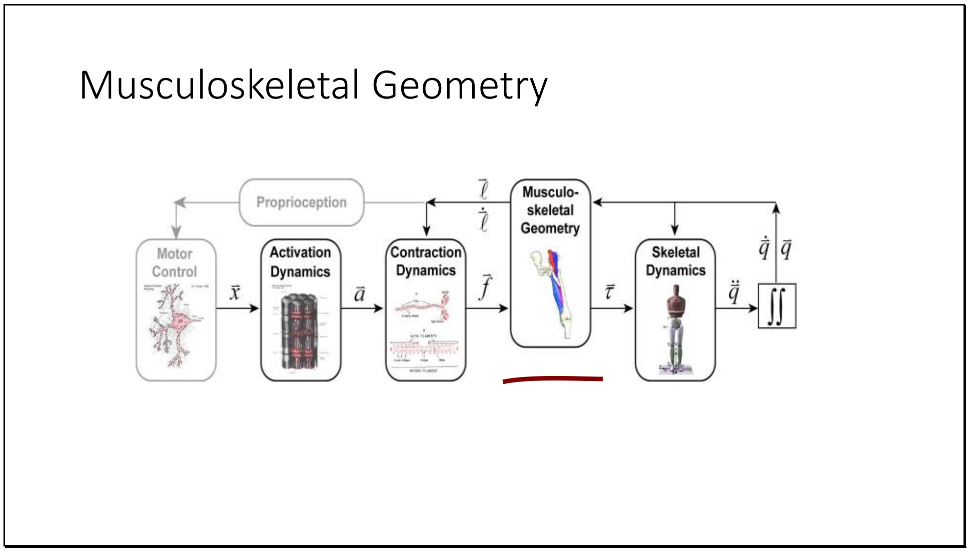
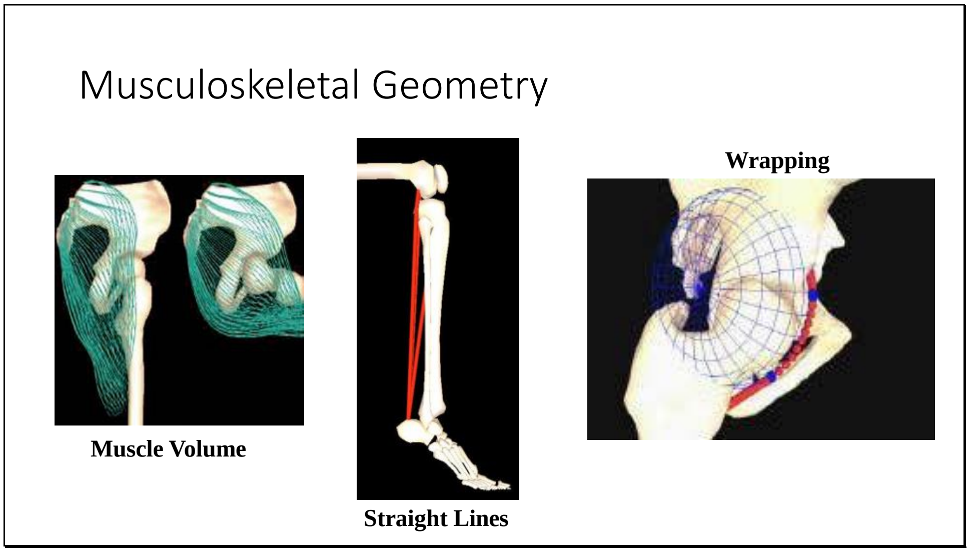


Musculoskeletal geometry models the muscle routing and the ability of a muscle to accelerate a joint.

Slide 29



A muscle occupies a volume, but for simulation reasons it is approximated by one or more strings (fibers). The string connects to the bones and when the muscle contracts force is applied on the bodies. The muscles may not follow a straight line and may have complex geometry, thus wrapping geometries are used.

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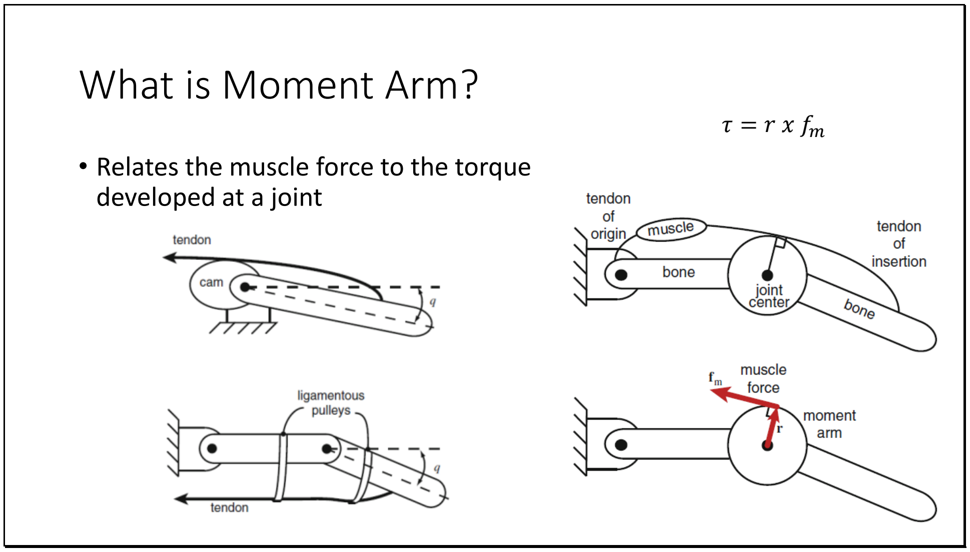

How a muscle is able to produce a torque around a joint and accelerate it?

The muscle force **f***m* transmitted by the tendon produces a torque **τ** at the DOF because the tendon crosses the joint at a distance **r** from its joint center. The torque can be calculated by the cross product of the moment arm (r) and the muscle force f.

It is important to note that Eq $\tau = r \; x \; f_m$ apply only to the instantaneous posture of the limb. That is, the moment arm of a tendon can either be constant throughout the range of motion of the joint, or be dependent on the joint angle.

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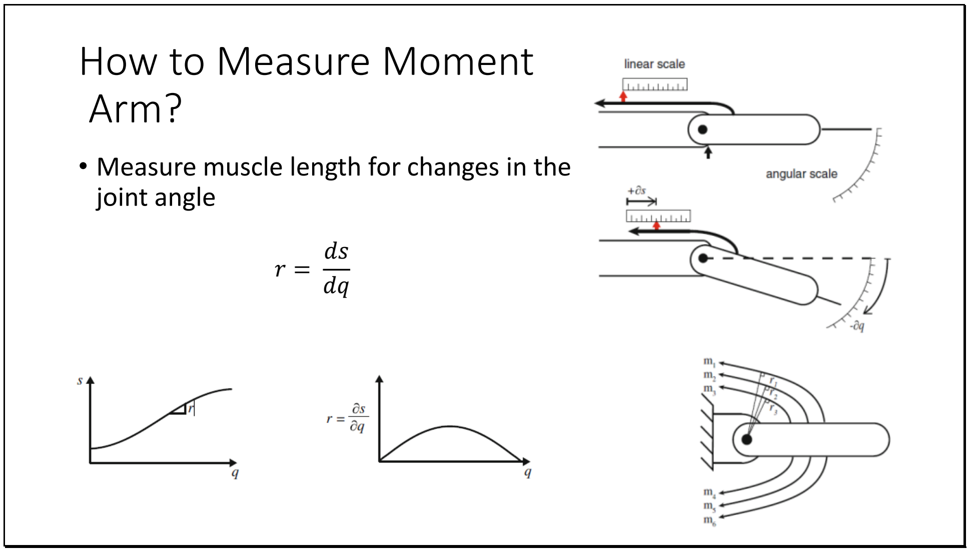
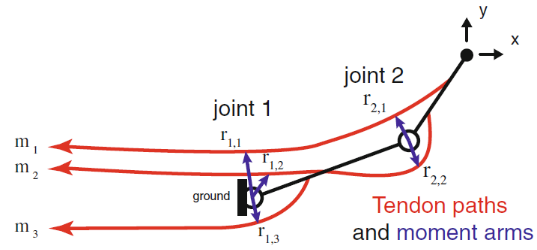


The geometry of tendon actuation also has important implications to muscle force production. This comes from the fact that the force a muscle can produce depends on its length and velocity. Thus, it is important to calculate the length and velocity of a muscle for any posture of the limb.

Consider the figure where we see that the change in angle δq induces an excursion, or travel, of the tendon. Call this *tendon excursion* δs. The question is, what is δs as a function of δq?

Muscles Can Spann Multiple Joints

- A muscle can contribute to the acceleration of multiple joints (How?)
- The moment arm of a muscle can depend on multiple joint angles (Why?)



- Muscles that span multiple joints can accelerate them (muscle 1, 2).
- When we change pose, the moment arm changes so do the torque that the muscle can induce.

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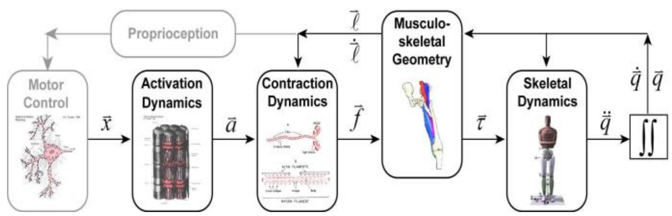
Skeletal Dynamics



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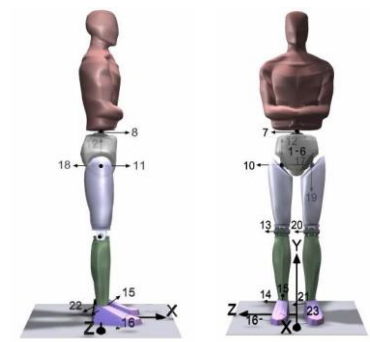
Musculoskeletal Dynamics

- 10 Rigid Bodies
  - mass
  - center of mass
  - inertia tensor
- 10 Joints
- 23 Degrees of Freedom



$M(q)\ddot{q} = \tau + V(q,\dot{q}) + G(q) + F(q,\dot{q}) \rightarrow$

$M(q)\ddot{q} = R(q)F_M + V(q,\dot{q}) + G(q) + F(q,\dot{q})$

M: $mass\ matrix$, V: $Coriolis\ and\ centrifugal$ torques,
G: gravity torques, F: other forces (ligaments), τ: active forces (muscles)
R: moment arm matrix, FM a vector of muscle forces

The equations of motion are the product of complex derivations. When we use different tools, we may be able to access an **analytical** (pydy, autolev) or a **numerical** (OpenSim) form of these equations. Each type has its advantages and disadvantages and depends on the application that we are interested.

Moment arm matrix R maps muscle forces to joint torques. Each column describes how a muscle can alter the joint torques. A muscle can accelerate multiple joints!

For each joint the total torque is equal to the sum of the torques that the spanning muscles can induce. Some muscles rotate the joint in the positive direction, while other in the negative. This is reflected by the sign of the moment arm elements.

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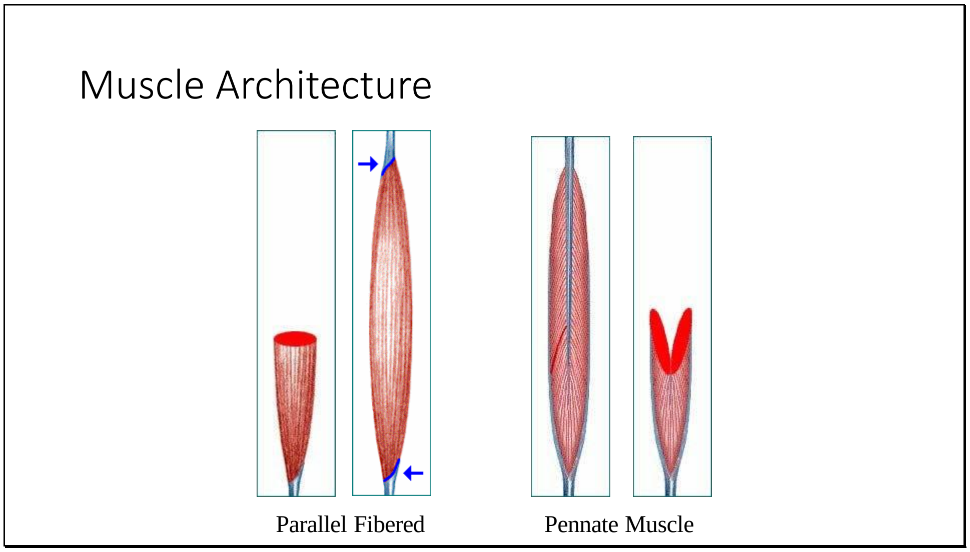

There are many muscle fiber types, but the most common are parallel fiber and pennate muscles.

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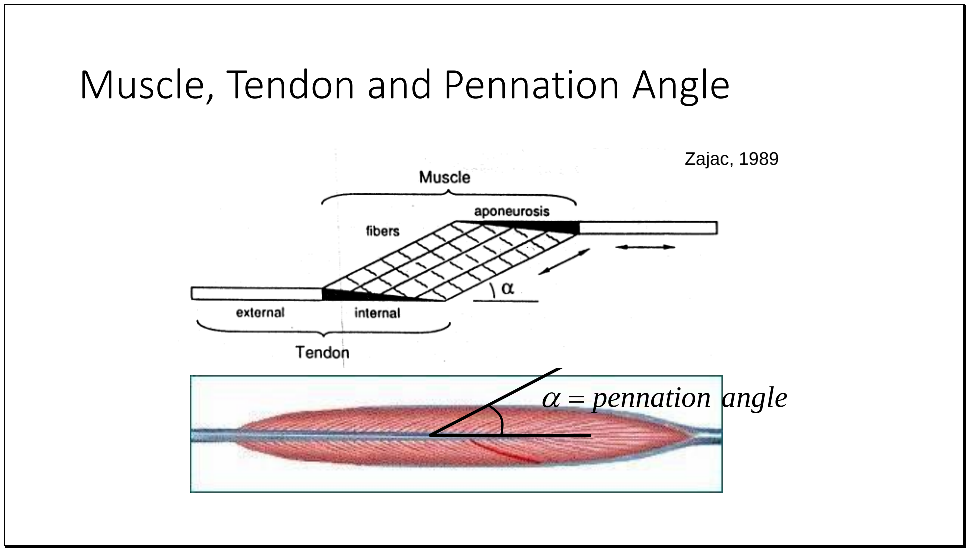

Pennate fibers are modeled with the pennation angle

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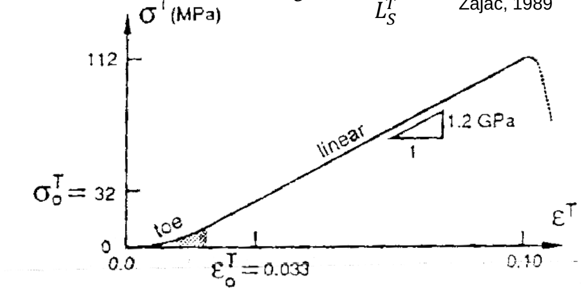
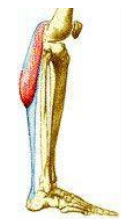
Tendon Stress-Strain Properties

$$\varepsilon^{\mathrm{T}} = \frac{\mathrm{L}^{T} - L_{S}^{T}}{L_{S}^{T}}$$

Zajac, 1989





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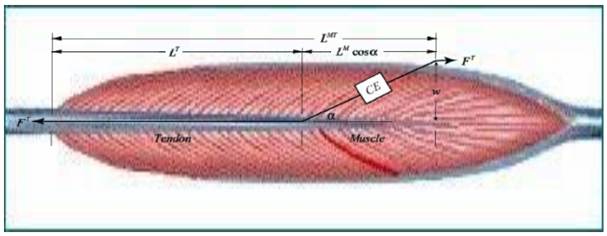
Lumped-Parameter Model

- The distributed properties of all muscle fibers are lumped into a single ideal fiber characterized by parameters appropriate for the whole muscle.
  - All fibers are the same length, at the same pennation angle, etc
  - The strength of the muscle is the summed strength of the individual fibers



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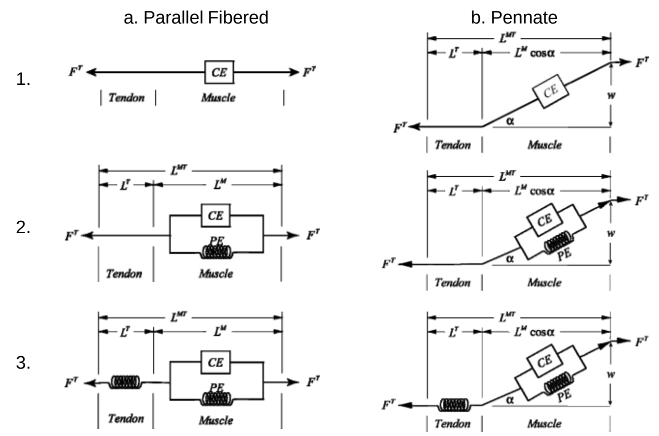
Muscle Models



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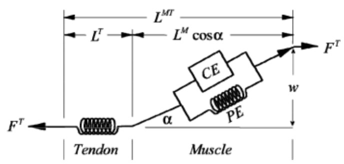
Definitions



L = Length
F = Force

T = Tendon
M = Muscle
α = Pennation angle

L^T = Length of tendon
L^M = Length of muscle
L^{MT} = Length of actuator

CE = Contractile Element.
Models the active force generating properties of muscle.

PE = Parallel Elastic Element
Models the passive force generating properties of muscle.

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Five Parameters

F_o^M Optimal muscle force.
Maximum isometric strength of muscle.

$$F_o^M = \sigma \frac{Volume}{L_o^M}$$

L_o^M Optimal muscle fiber length
Length at which F_o^M is generated

α_o Optimal pennation angle
Pennation angle when the fibers are at L_o^M

V_{max}^M Maximum shortening velocity of muscle normalized by fiber length

$$\underbrace{2.0 \cdot L_o^M}_{slow\ twitch} < V_{max}^M < \underbrace{10 \cdot L_o^M}_{fast\ twitch}$$

L_s^T Slack length of tendon.
Length at which tendon starts to develop force

$$L_s^T = L_{external}^T + L_{internal}^T$$

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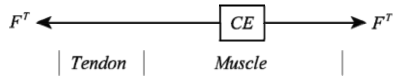
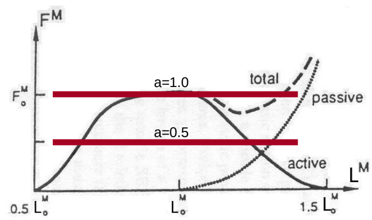
Simplest

- Assumptions
  - Tendon is inelastic
  - No dependence on length or velocity
  - Parallel fibered
- Parameters
  F_o^M
- Time-varying inputs
  a

$0\ (off) \le a \le 1.0\ (fully\ on)$



$$F^T = F^{CE} = a(t) \cdot F_o^M$$



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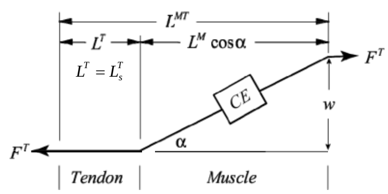
Simplest with Pannate Fibers

- Assumptions
  - Tendon is inelastic
  - No dependence on length or velocity
  - Pennate
- Parameters
  $F_o^M \quad L_o^M \quad \alpha_o \quad L_s^T$
- Time-varying inputs
  $a \quad L^{MT}$



$$F^T = F^{CE} \cdot \cos\alpha = a(t) \cdot F_o^M \cdot \cos\alpha$$

But, α changes with the length of the muscle!

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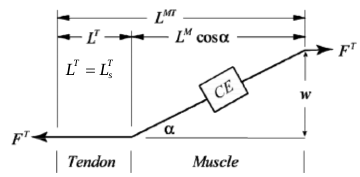
Simplest with Pannate Fibers

$$L^{MT} = L_s^T + L^M \cos\alpha$$

$$w = L_o^M \sin\alpha_o$$

Width is assumed to be constant.

Some algebra and trig…



$$\cos\alpha = \sqrt{\frac{\left(\frac{L^{MT} - L_s^T}{w}\right)^2}{1 + \left(\frac{L^{MT} - L_s^T}{w}\right)^2}}$$

$$F^T = F^{CE} \cdot \cos\alpha = a(t) \cdot F_o^M \cdot \cos\alpha$$

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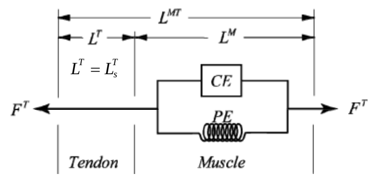
Force-Length-Velocity Properties and Inelastic Tendon

- Assumptions
  - Tendon is inelastic
  - Dependence on length or velocity
  - Parallel fibered
- Parameters
  $F_o^M \; L_o^M \; L_S^T \; V_{max}$
- Time-varying inputs
  $a \;\; L^{MT} \; \dot{L}^{MT}$



$$F^T = F^{CE} + F^{PE}$$

$$F^{CE} = a(t) \cdot F_o^M \cdot \tilde{F}_L^M(L^M) \cdot \tilde{F}_V^M(\dot{L}^M)$$

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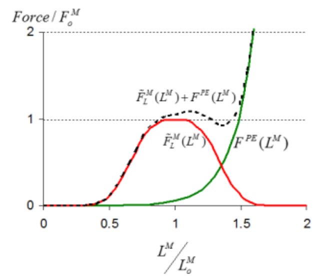
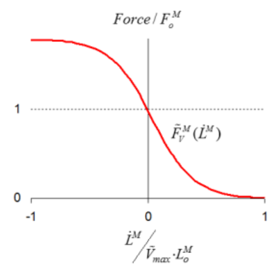
Length, Velocity and Passive Forces





$$F^{PE} = F_o^M \cdot 3 \cdot 10^4 \cdot \exp[6 \cdot (\frac{L^M}{L_o^M} - 3.2)]$$

$$\tilde{F}_L^M(L^M) = \exp[\ 17.33 \cdot \left|\frac{L^M}{L_o^M} - 1.0\right|^3\ ]$$

$$\tilde{F}_V^M(\dot{L}^M) = 1.8 - \frac{1.8}{1.0 + \exp[\ \frac{0.04 - \frac{\dot{L}^M}{\tilde{V}_{max} \cdot L_o^M}}{0.18}\ ]}$$

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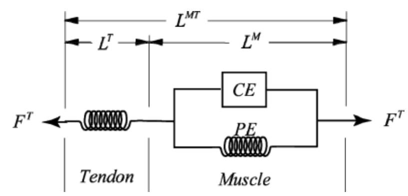
Force-Length-Veloctiy Properties and Elastic Tendon

- Assumptions
  - Tendon is elastic
  - Dependence on length or velocity
  - Parallel fibered
- Parameters
  F_o^M L_o^M L_S^T V_{max}
- Time-varying inputs
  F_o^M L_o^M L_S^T V_{max}



$$L^{MT} = L^T + L^M$$
$$\dot{L}^{MT} = \dot{L}^T + \dot{L}^M$$

A closed-form expression for F^T is generally not possible.