Neuromechanics of Human Motion

Motor Units and Muscle Function

Joshua Cashaback, PhD

Recap — Bayesian Integration

$$p(A|B) = \frac{p(B|A) \cdot p(A)}{p(B)}$$

x Likelihood
(new evidence)

Prior

Posterior

Update Belief

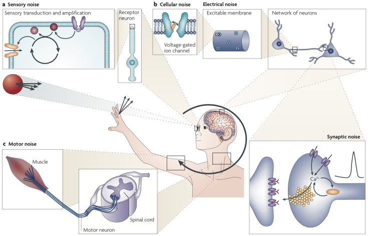
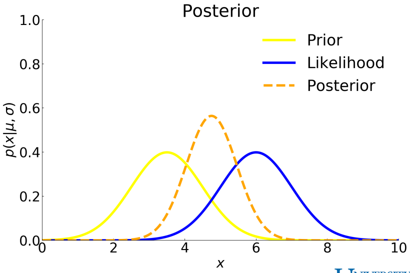


Figure 1 | **Overview of the behavioural loop and the stages at which noise is present in the nervous system.** **a** | Sources of sensory noise include the transduction of signals. This is exemplified here by a photoreceptor and its signal-amplification cascade. **b** | Sources of cellular noise include the ion channels of excitable membranes, synaptic transmission and network interactions (see BOX 2). **c** | Sources of motor noise include motor neurons and muscle. In the behavioural task shown (catching a ball), the nervous system has to act in the presence of noise in sensing, information processing and movement.



MODULE 2
Muscle and Limb Dynamics (Outputs)

Lecture Objectives — Motor Units and Muscle Function

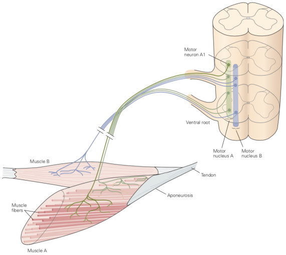
1. Motor Unit and Muscle Contraction
   a. Motor Unit
   b. Neuromuscular junction, sarcoplasmic reticulum
   c. Cross-bridges
   d. Motor Neuron
   e. Recruitment and discharge rate
   f. Force-Length curve (active and passive)
   g. Force-Velocity curve
   h. Tendon compliance

Muscle Contraction

Motor Unit

Motor Unit

A motor neuron and the skeletal muscle fibers innervated by that motor neuron's axonal terminals.

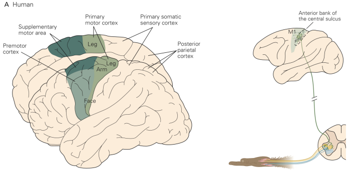


One motor neuron will innervate a few to 1000s of muscle fibers

Motor neurons dictate muscle force

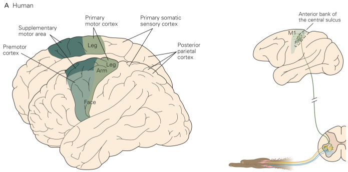
Origin of Motor Neuron Action Potentials

Motor Cortex - Voluntary Actions

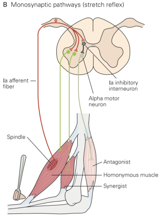


Origin of Motor Neuron Action Potentials

Motor Cortex - Voluntary Actions

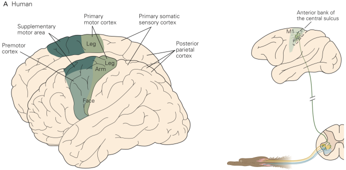


Spinal Reflexes - Involuntary Actions

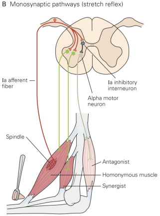


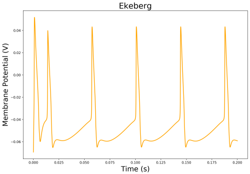
Origin of Motor Neuron Action Potentials

Motor Cortex - Voluntary Actions



Spinal Reflexes - Involuntary Actions

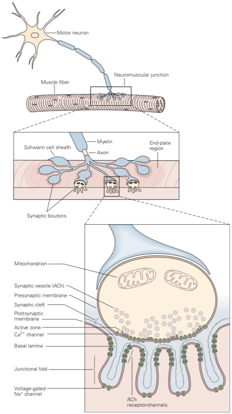




MN action potentials travels toward the muscle

Neuromuscular Junction

Synapse from Motor Neuron to Muscle



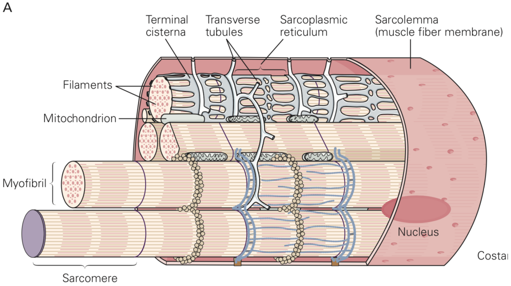
Acetylcholine (ACh) is a neurotransmitter

Neuromuscular Junction

From Motor Neuron to Muscle

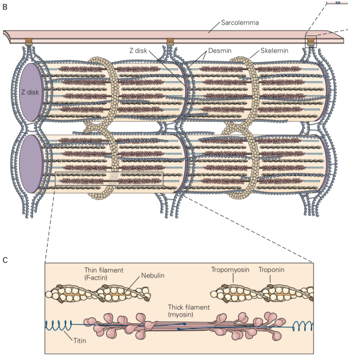
1. The action potential reaches the axon terminal.
2. Calcium ions flow into the axon terminal.
3. Acetylcholine is released into the synaptic cleft.
4. Acetylcholine binds to postsynaptic receptors.
5. This binding causes ion channels to open and allows sodium ions to flow into the muscle cell.
6. The flow of sodium ions causes an action potential along the sarcolemma (EMG).

Sarcoplasmic Reticulum & T-Tubules



1. Sarcoplasmic Reticulum & transverse tubules absorb and store Ca2+
2. AP along the sarcolemma causes the SR and TT to release Ca2+
3. Ca2+ allows cross-bridges to move and generate muscle contraction

Sarcomere — Cross Bridges



1. Myosin (thick filament) binds to Actin (thin filament)
2. Myosin heads move and cause muscle contraction

Cross Bridge Cycle

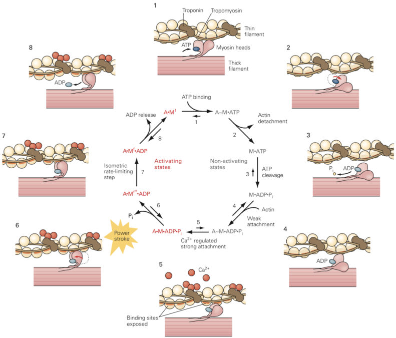


Figure 34–9 The cross bridge cycle. Several nonactivating states are followed by several activating states triggered by Ca^{2+}. The cycle begins at the top (**step 1**) with the binding of adenosine triphosphate (ATP) to the myosin head. The myosin head detaches from actin (**step 2**), ATP is hydrolyzed to phosphate (P_i) and ADP (**step 3**), and the myosin becomes weakly bound to actin (**step 4**). The binding of Ca^{2+} to troponin causes tropomyosin to slide over actin and enables the two myosin heads to close (**step 5**). This results in the release of P_i and the extension of the myosin neck, the power stroke of the cross bridge cycle (**step 6**). Each cross bridge exerts a force of about 2 pN during a structural change (**step 7**) and the release of adenosine diphosphate (ADP) (**step 8**). (•, strong binding; ~, weak binding; **M†**, cross bridge force of myosin; and **M†***, force-bearing state of myosin.) (Adapted, with permission, from Gordon, Regnier, and Homsher 2001.)

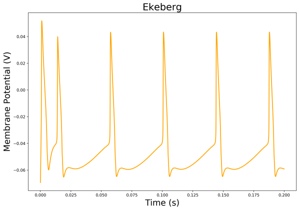
Ca^{2+} from the SR allows myosin to strongly bind to Actin

P_i release causes power stroke — myosin head moves (muscle contraction)

Motor Neurons Modulate Muscle Force

Motor Neuron — Modulating Muscle Force

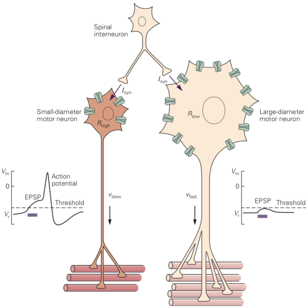
1. Recruitment
    - thresholds
    - size principle
2. Discharge Rate
    - low vs. high firing rates



Motor Neuron — Recruitment

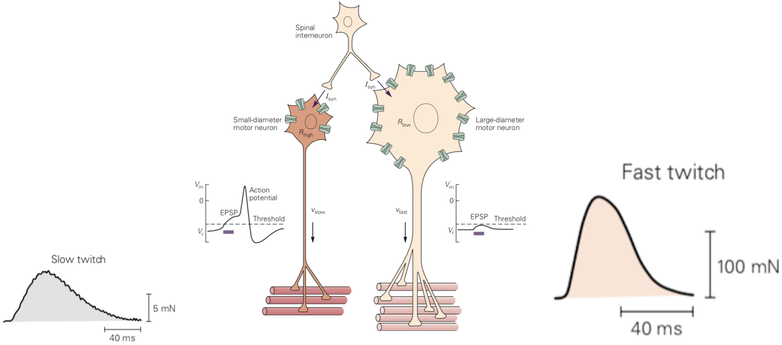
Henneman's Size Principle

Motor neurons with small cell bodies generate action potentials (are recruited) before motor neurons with large cell bodies



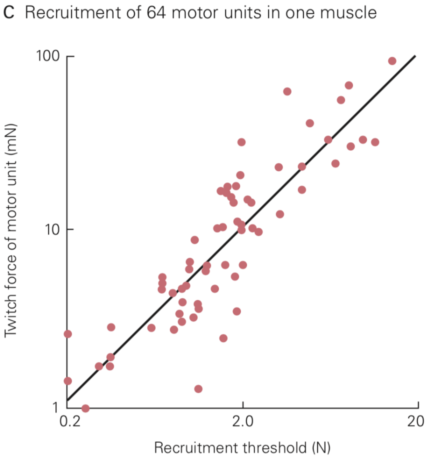
Inverse relationship between excitability and size

Motor Neuron — Slow vs. Fast Fibers



1. Small diameter motor neurons recruit slow-twitch muscle fibers
2. Large diameter motor neurons recruit fast-twitch muscle fibers

Motor Neurons — Threshold vs Force



Motor Neurons—Modulating Muscle Force

1. Recruitment
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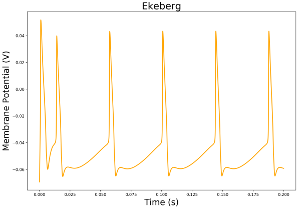
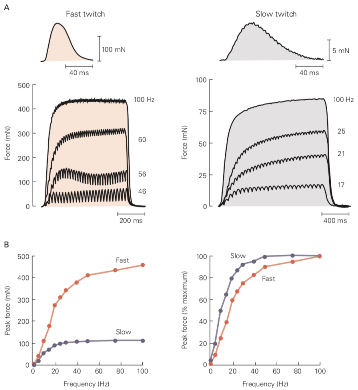


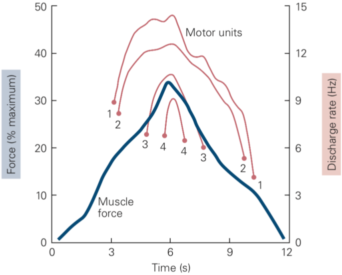
Motor Neuron — Discharge Rate



Greater motor neuron discharge rate leads to greater force

Recruitment and Discharge Rate

Figure 34–6 Muscle force can be adjusted by varying the number of active motor units and their discharge rate. A gradual increase and then a decrease in the force (**blue line**) exerted by the knee extensor muscles involved the concurrent activation of four (out of many) motor units. The muscle force was changed by varying both the number of motor units that were active and the rate at which the motor neurons discharged action potentials. Motor unit 1 was activated when muscle force reached 20% of maximum. Initially the motor neuron discharged action potentials at a rate of 9 Hz. As force increased, the discharge rate increased up to 15 Hz, when both the force and discharge rate declined, and the motor unit was inactivated at 14% of maximal force. Motor units 2, 3, and 4 were activated at greater forces but discharge rate was modulated similarly. (Reproduced, with permission, from Person and Kudina 1972.)



Muscle force modulated by both the recruitment and discharge rate of motor neurons

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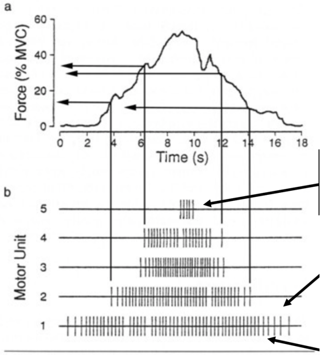
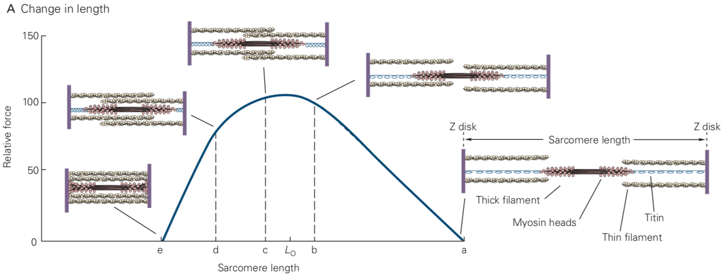
Recruitment and Discharge Rate



Muscle force modulated by both the recruitment and discharge rate of motor neurons

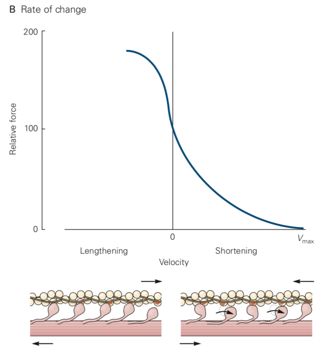
Cross Bridge Binding Influences Muscle Force

Active Force Length Curve



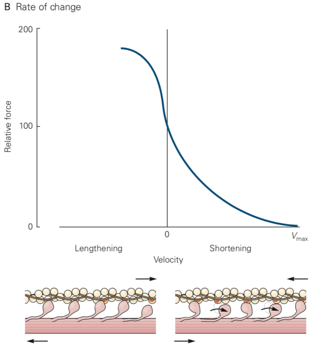
1. Optimal length (L_0) = maximal actin and myosin overlap
2. Stretched muscle = cross bridges can't form
3. Shortened muscle = extreme overlap reduces number of potential attachment sites

Force Velocity Curve



1. Shortening: myosin heads spend more time near the end of their power stroke, causing less force (active state) and more time detaching, reposition, and reattaching (inactive state)

Force Velocity Curve

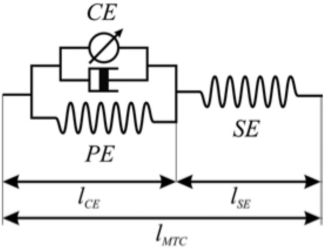


1. Lengthening: myosin heads spend more time stretched and little time unattached because they do not need to reattach

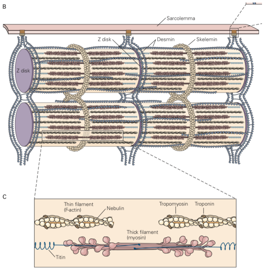
Introduction to Muscle Modelling

Different Types of Muscle Models

Hill (Phenomenological) vs Cross-bridge (Mechanistic)



Schematic of a Hill-type muscle model.



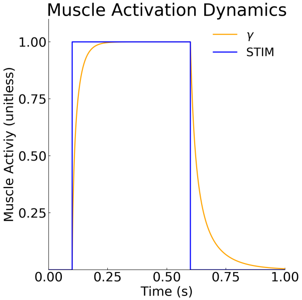
Fundamental difference relates to how the contractile element generates force

Considerations

Factors to consider:

1. Electrical
   - Stim-driven vs. EMG-driven
   - Activation dynamics
2. Mechanical
   - active force length curve
   - passive force length curve
   - force velocity curve
   - tendon compliance
   - pennation angle

Stim Driven



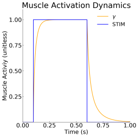
STIM: stimulating the muscle

$\cdot$neuromuscular junction via αmn OR electrical stimulation

γ: free Ca^{2+} concentration (drive cross-bridge cycling)

$\cdot$rise = release, fall = uptake into sarcoplasmic reticulum

Stim Driven



$$\dot{\gamma} = \frac{(STIM - \gamma)}{\tau} \qquad (1a),$$

$$\tau = \begin{cases} \tau_{act}(0.5 + 1.5\gamma); & STIM > \gamma \qquad (1b), \\ \tau_{deact}/(0.5 + 1.5\gamma); & STIM \leq \gamma \qquad (1c). \end{cases}$$

$$\tau_{act}(0.015), \tau_{deact}(0.05)$$

note: minimal STIM set to 0.0001 for numerical stability

EMG Driven

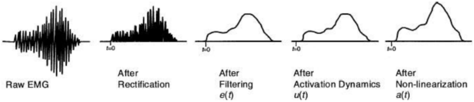
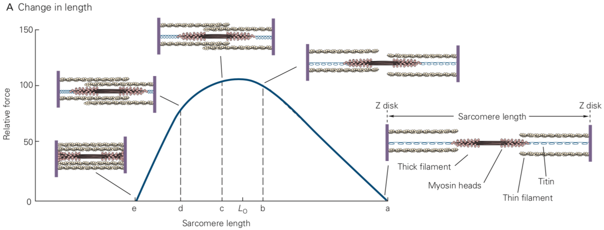


Figure 3.
Muscle activation dynamics: Transformation from EMG to muscle activation.

1. EMG is a measure of the electrical activity spreading across the muscle, causing it to activate.
2. electromechanical delay
3. non-linear EMG to Force relationship
4. Buchanan et al., 2004
5. $\gamma = a(t)$

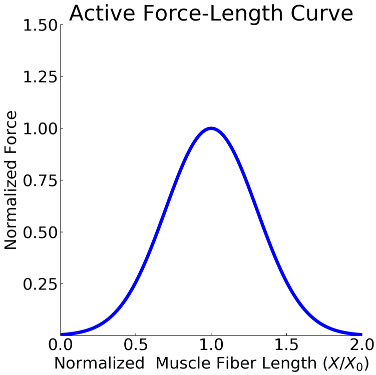
Active Force Length Curve



Modelling:

1. piece-wise linear
2. polynomial
3. Gaussian

Active Force Length Curve



$$\alpha = e^{-(X/X_0 - 1)^2/v}$$

α: normalized active force capacity

$v(0.185)$: width parameter

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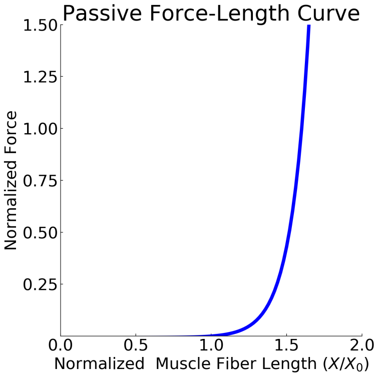
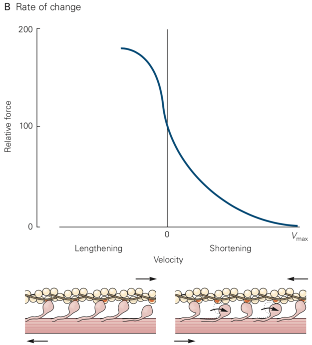
Passive Force Length Curve



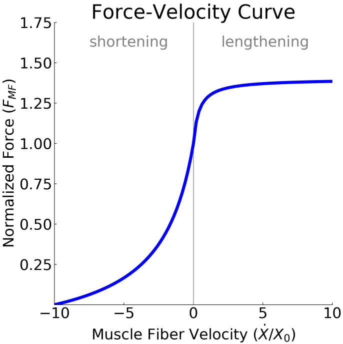
$$F_{PE} = \frac{e^{k_{PE}(X/X_0-1)/\varepsilon_{PE}}-1}{e^{k_{PE}}-1}$$

$$k_{PE}(5.0); \varepsilon_{PE}(0.6)$$

Force Velocity Curve



Force Velocity Curve



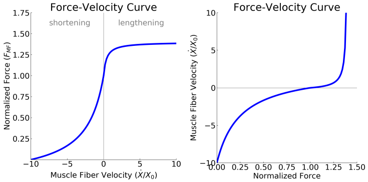
Defined in terms of normalized muscle fiber velocity

$$\left(\frac{\dot{X}}{X_0}\right) = (0.25 + 0.75\gamma) V_{MF}^{max} \frac{F_{MF} - \gamma\alpha}{b}$$

$V_{MF}^{max}(10.0)$: maximum shortening velocity

Note: $\gamma = 1$, $\alpha = 1$, $\frac{\dot{X}}{X_0}$ is an input $[-10, 10]$ for plotting

Force Velocity Curve



$$\left(\frac{\dot{X}}{X_0}\right) = (0.25 + 0.75\gamma)\, V_{MF}^{max}\, \frac{F_{MF} - \gamma\alpha}{b} \qquad (1a),$$

$$b = \begin{cases} \gamma\alpha + \frac{F_{MF}}{A_f}; & F_{MF} \leq \gamma\alpha \qquad (1b), \\ \frac{(2+\frac{2}{A_f})(\gamma\alpha F_{MF}^{max} - F_{MF})}{(F_{MF}^{max} - 1)}; & F_{MF} > \gamma\alpha \qquad (1c). \end{cases}$$

$F_{MF}^{max}(1.4)$; influences peak lengthening force

$A_f(0.25)$: shape parameter

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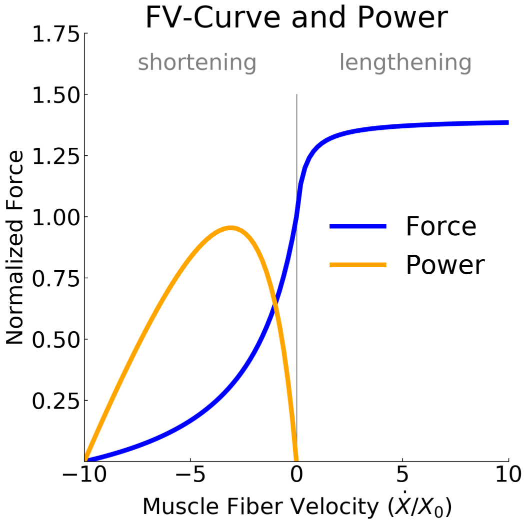
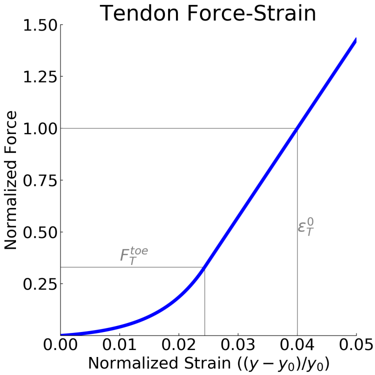
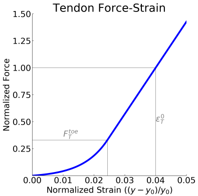
Power


FV-Curve and Power
shortening
lengthening
Force
Power
Normalized Force
Muscle Fiber Velocity ($\dot{X}/X_0$)
1.75
1.50
1.25
1.00
0.75
0.50
0.25
−10
−5
0
5
10

Tendon Strain-Force Curve



Tendon Strain-Force Curve

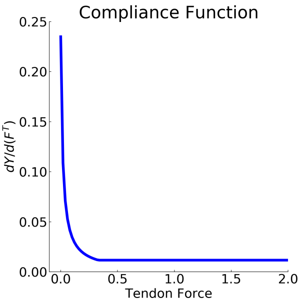


$$F_T = \begin{cases} \frac{F_T^{toe}}{e^{k^{toe}}-1}\left(e^{k^{toe}[(Y-Y_0)/Y_0]/\varepsilon_T^{toe}} - 1\right); & \frac{Y-Y_0}{Y_0} \leq \varepsilon_T^{toe} \\ k^{lin}([(Y-Y_0)/Y_0] - \varepsilon_T^{toe}) + F_T^{toe}; & \frac{Y-Y_0}{Y_0} > \varepsilon_T^{toe} \end{cases}$$

Y_0: tendon slack length

$F_T^{toe}(0.33); k^{toe}(3.0); e_T^0(0.04); \varepsilon_T^{toe}(0.609 e_T^0); k^{lin}(1.712/e_T^0)$

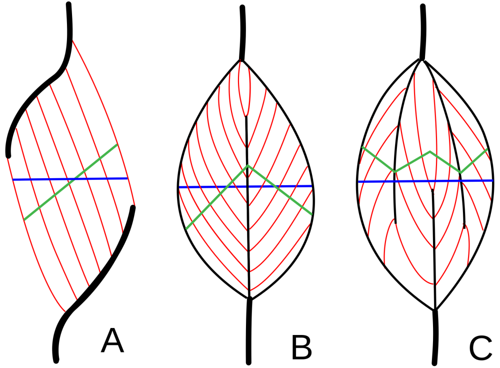
Tendon Compliance



$$\frac{dY}{dF_T}$$

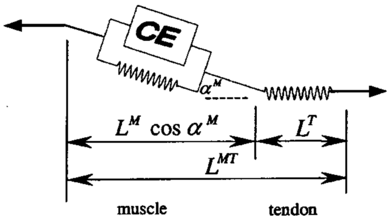
some muscle models want tendon mechanics defined in terms of compliance

Muscle Pennation



unipennate, bipennate, multipennate

Pennation Angle



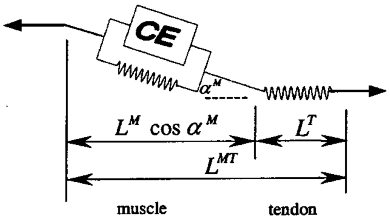
Don't confuse α here with active FL curve in previous slides

I personally use θ for pennation angle

Models: no, static, dynamic pennation angle

$$L_{MT} = L_T + L_{MF} cos(\theta)$$

Pennation Angle



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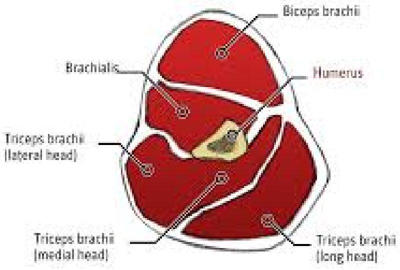
I personally use θ for pennation angle

Models: no, static, dynamic pennation angle

$$L_{MT} = L_T + L_{MF} cos(\theta)$$

$$L_{MT} = Y + Xcos(\theta)$$

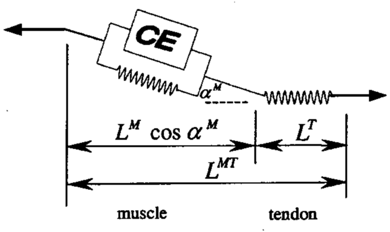
Physiological Cross-Sectional Area & Stress



PCSA: bigger muscles generate more force

σ (muscle stress, N/cm^2): quite variable (35.0)

Muscle-Tendon Force



$$F_{MT} = F_T = F_{MF} cos(\theta) + F_{PE} cos(\theta)$$

$$F_{MT} = F_T = PCSA \cdot \sigma(\gamma \cdot \alpha \cdot FV + F_{PE}) cos\theta$$

Summary

1. Motor Unit and Muscle Contraction
    a. Motor neuron
    b. Recruitment and discharge rate
    c. Neuromuscular junction, sarcoplasmic reticulum
    d. Cross-bridges
    e. Muscle and Tendon Mechanics

Questions???

Homework

Plot:

1. Active Force Length Curve
2. Passive Force Length Curve
3. Velocity-Force (x-axis) Curve
4. Force (x-axis)-Velocity Curve
5. Tendon Strain-Force Relationship
6. Tendon Compliance
7. Activation Dynamics
8. Thelen, D. G. (2003). J Biomech Eng, 125(1), 70-77.

Next Class

1. Hill-type Model
2. Distribution Moment Approximation Model

Acknowledgements

Dinant Kistemaker