Mechanics of Skeletal Muscle

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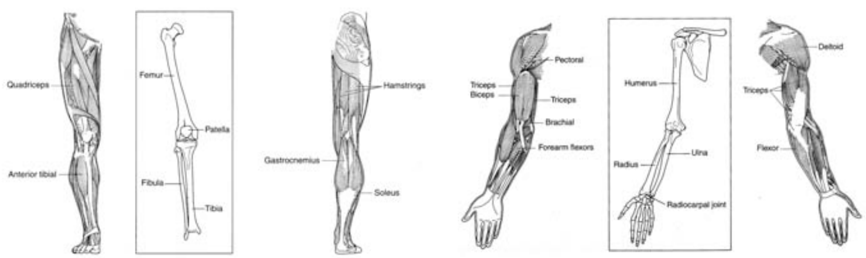
Chapter 9, pages 213-214

Outline

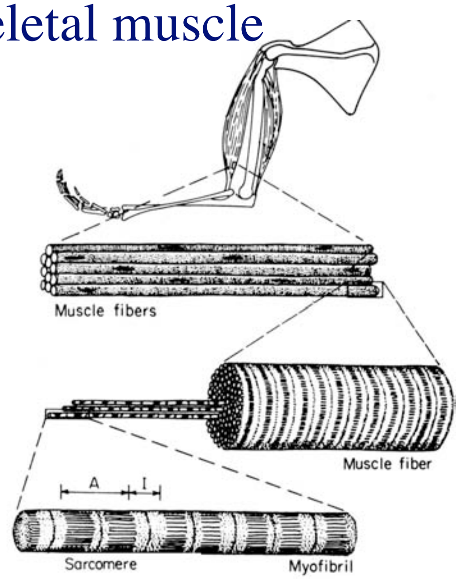
- anatomical structure
- physiological basis of muscle contraction (muscle nerve interaction, sliding filament theory of force development)
- effect on muscle force of stimulation frequency, muscle fibre type, muscle length, velocity of shortening/ lengthening, muscle geometry (PCSA, angle of pennation)
- quick-release experiments
- Hill's active state model of muscle contraction

Muscle anatomy



Fibers, fibrils, and filaments; the architecture of skeletal muscle

- each muscle is made up of numerous muscle *fibers*
- each fiber is made up of many *myofibrils*
- each myofibril is made of many *sarcomeres*, the smallest anatomical unit that contracts like a muscle

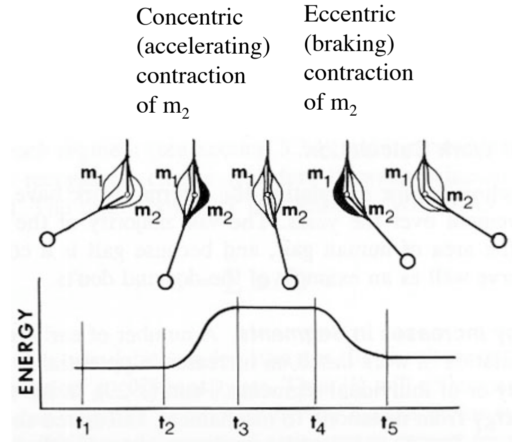


Muscle contraction may occur with or without a change in muscle length

- muscle “contraction” refers to force development in activated muscle, rather than decrease in length
- contractions may be:
  - *isometric*: constant muscle length
  - *isokinetic*: constant muscle velocity
  - *isotonic:* constant muscle load
  - *concentric*: shortening
  - *eccentric*: lengthening
- strains are greatest during concentric contractions, where muscle may shorten by 50-70 percent

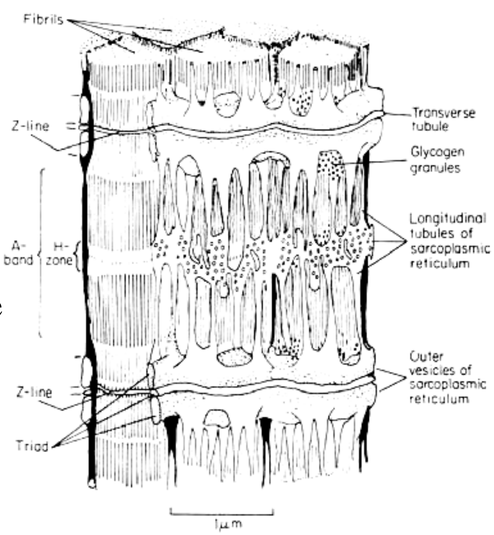
Eccentric and concentric contractions

- *Concentric (energy generating, positive work) contractions* tend to increase joint angular velocity, and increase the total energy of the system
- *Eccentric (energy absorbing, negative work) contractions* tend to decrease (or brake) joint angular velocity, and reduce the total energy of the system



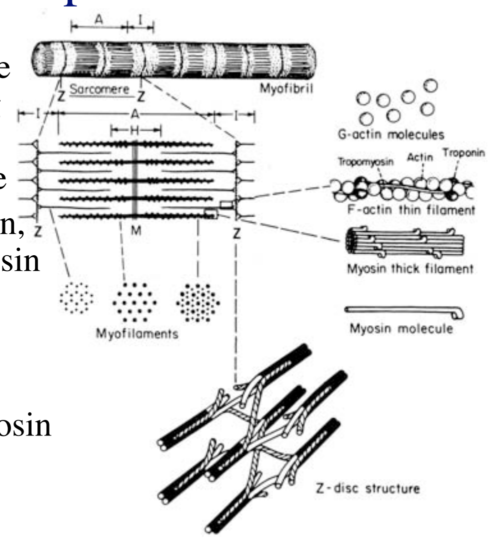
Calcium is needed for muscle contraction

- the sarcoplasmic reticulum (SR) is a membrane that surrounds myofibrils
- at the onset of an action potential, the SR releases calcium
- calcium then binds to troponin, triggering muscle contraction
- the SR re-sequesters calcium at the end of the action potential, thereby inducing muscle relaxation



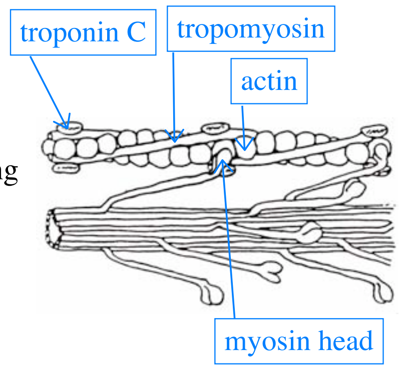
Muscle force results from interaction between contractile proteins

- 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



Muscle force results from interaction between contractile proteins (cont)

- during the action potential, calcium binds to troponin C, inducing a conformal change in tropomyosin
- simultaneously, adenosine triphosphate (ATP) is hydrolyzed by ATPase in the myosin head, providing the metabolic energy required for cross-bridge attachment
- at the end of the action potential, calcium re-uptake causes a reconfiguration of tropomyosin to a position which prevents cross-bridge attachment

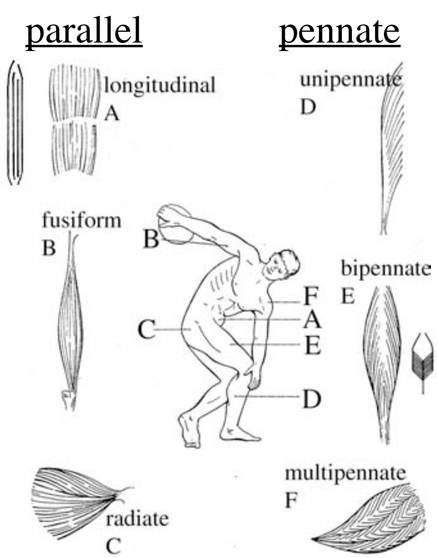


Factors affecting muscle force development (a partial list)

- muscle geometry (e.g., physiological cross-sectional area (PCSA), angle of pennation)
- number of activated motor neurons, frequency of discharge
- muscle fibre type
- muscle length
- velocity of shortening/ lengthening

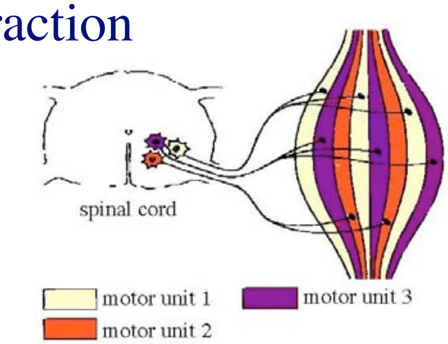
gross muscle geometry affects muscle force

- peak muscle force increases linearly with physiologic cross-sectional area (PCSA)
- *pennate muscles*: have fibers that run oblique to long axis of the muscle
- pennate muscles have larger PCSA (and muscle force), but smaller length and shortening velocity, than parallel fiber muscles



Muscle-nerve interaction

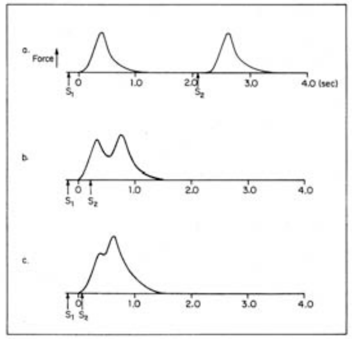
- *motor unit*: a single motor nerve axon and all the muscle fibers it contacts
- a motor nerve enters muscle and splits into numerous axons; each axon contacts 10-2000 muscle fibers
- each muscle fiber is innervated by only one motor nerve axon, and contracts in response to an action potential in that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

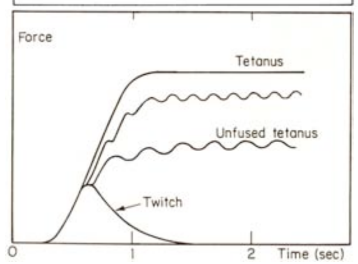


muscle	# muscle fibers	# motor units	av. fibers per motor u.
platysma	27,100	1,100	25
Brachioradialis	130,000	330	410
Tibialis anterior	250,000	450	600
gastrocnemius	1,120,000	580	2,000

stimulation frequency affects muscle force: twitch and tetanus

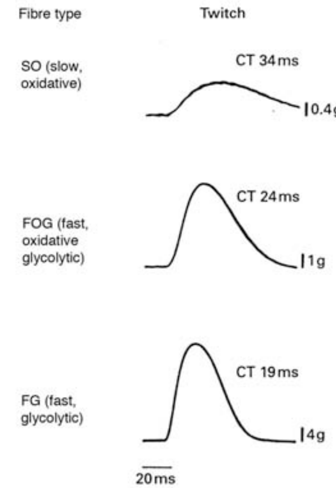
- muscle force can be modulated by varying: (1) the number of recruited motor neurons, and (2) the frequency of discharge (i.e., stimulation rate) in motor neurons
- a single action potential (S_1) produces a *twitch* contraction, a quick rise and slow fall in force
- a *tetanus* occurs when a new action potential (S_2) arrives before the previous twitch has dissipated, and there is force summation
- at stimulation frequencies >30/s, there are no twitch transients (fused tetanus)





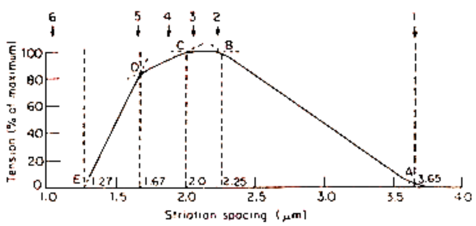
muscle fiber type affects the speed and strength of muscle force

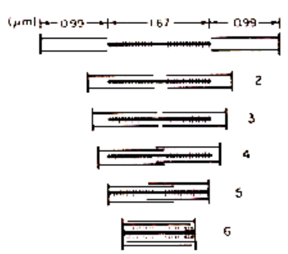
- fast glycolytic (FG, type IIB, white meat) fibers: twitch contraction times less than 55 ms, can generate 2-3 times more force than slow fibers, but highly fatigable, ATP generation through conversion of glucose to lactic acid (glycolysis)
- slow oxidative (SO, type I, red meat): fatigue resistant, ATP generation through oxidative phosphorylation of blood glucose and free fatty acids
- fast oxidative glycolytic (FOG, type IIA): intermediate degree of fatigue resistance, ATP generation through oxidative phosphorylation and glycolysis



active force development in the sarcomere depends on actin-myosin overlap

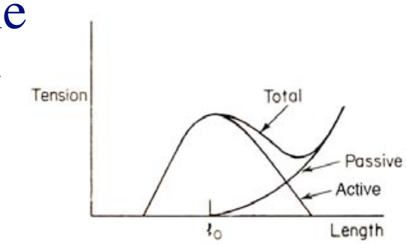
- (A): no overlap between actin and myosin, zero developed tension
- between (A) and (B): tension increases linearly as overlap increases
- between (B) and (C): maximum overlap & maximum tension
- left of (C): interference between actin filaments reduces ability of crossbridges to develop tension
- left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

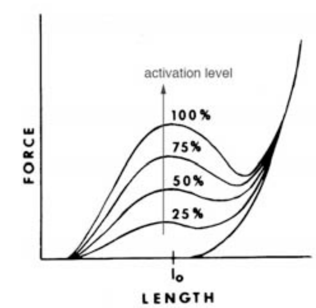




muscle length affects force development in whole

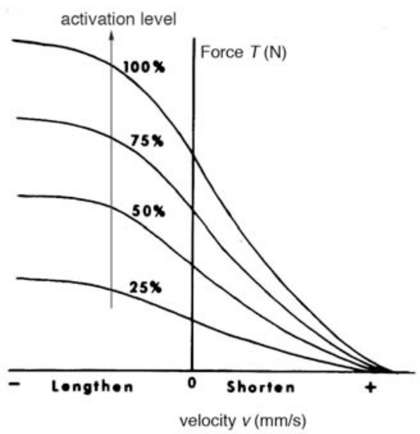
- the tension developed in a whole muscle is the sum of active force due to muscle contraction and passive force due the passive stiffness of tendon and muscle
- the passive force is negligible for lengths less that the normal resting length (l_0)
- the active force follows the tension-length behaviour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



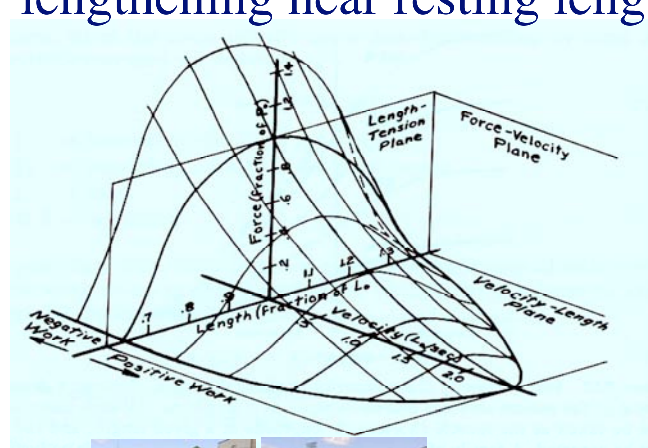


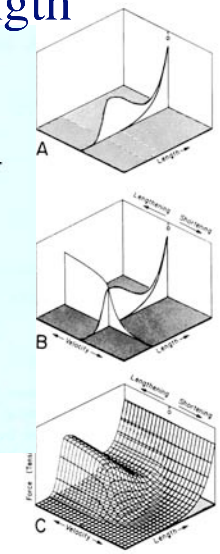
muscle velocity affects force development in whole muscle

- 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



Greatest force is developed when lengthening near resting length





Muscle force-velocity behaviour is described by the Hill Equation

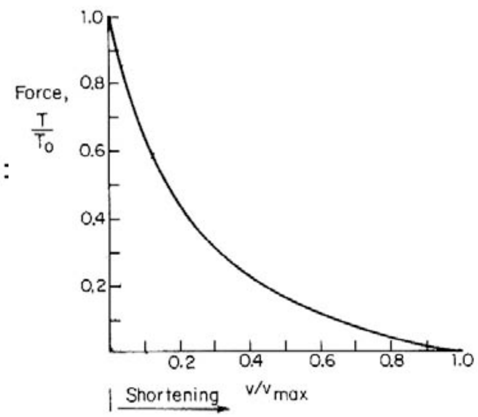
An empirical relation that describes the force-velocity behaviour of muscle during shortening is the Hill Equation.

The equation can be written as:

$$\boxed{(T+a)(v+b)=(T_o+a)b}$$

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 $P = T \cdot v$.

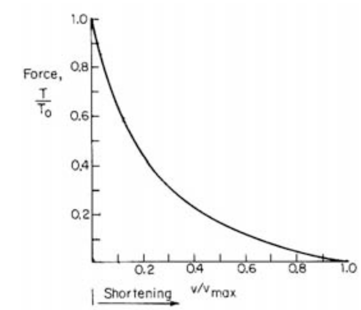


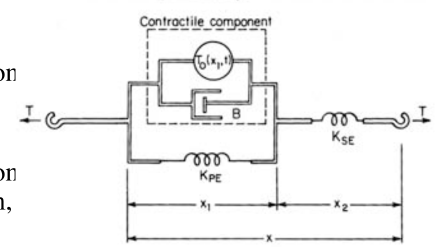
Hill's active state model of muscle contraction

- Hill assumed:

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

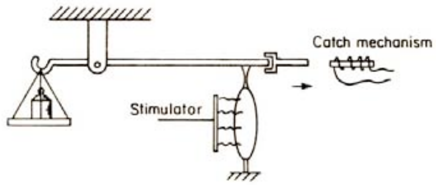
  (2) if the muscle is shortening, some force is dissipated in overcoming inherent viscous resistance
- B: muscle damping constant, which must be a nonlinear function of shortening velocity and temperature
- K_{SE}: stiffness of the series elastic component; represents force-deflection properties of tendon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

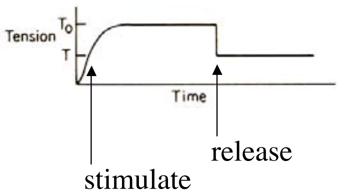




Quick-release experiments

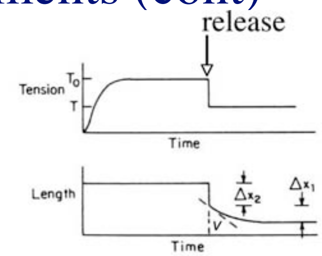
- hold muscle length fixed with the catch
- stimulate muscle to produce peak (isometric) force T_0
- instantly release catch
- at the instant of release, muscle force changes to a value (T) that depends on weight in pan
- in this example, $T < T_0$ so the muscle shortens, rather than lengthens

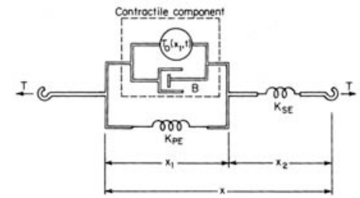


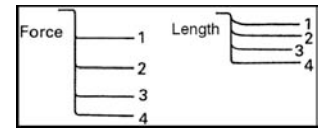


Quick-release experiments (cont)

- there is an instant change (Δx_2) in total muscle length following release
- this occurs in the tendon, which is relatively elastic and in series with the muscle (K_{SE})
- this is followed by a more gradual change (Δx_1) in total muscle length
- 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







Review Questions

- What is a motor unit?
- What is the role of calcium, ATP, troponin, tropomyosin, actin, and myosin in muscle contraction?
- What structure(s) contribute to the passive force-length behaviour of muscle?
- What characteristics of muscle might make it easier for us to quickly stop than to quickly start a movement?
- Why is there an optimal muscle length, above or below which there is a decrease in the force developed by activated muscle?
- During shortening, what combination of force and velocity is approximately maximizes muscle power?
- What are quick release experiments, and what data do they provide?