Biomechanics of skeletal muscles

Akhtar Rasul
Muscles

- cardiac muscle: composes the heart
- Smooth muscle: lines hollow internal organs
- skeletal (striated or voluntary) muscle: attached to skeleton via tendon & perform movement
- Skeletal muscle 40-45% of body weight
  - more than 430 muscles
  - 80 pairs produce vigorous movement
- Dynamic & static work
  - Dynamic: locomotion & positioning of segments
  - Static: maintains body posture
Composition & structure of skeletal muscle

- Structure and organization:
  - The structural unit of skeletal muscle is the muscle fiber = multinucleated, thickness: 10-100 micro m
  - Length: 1-30 cm

- Sarcolemma

- The myofibril is made up of several sarcomeres that contain thin (actin), thick (myosin), elastic (titin), and inelastic (nebulin) filaments
- Muscle fiber → endomysium → fascicles → perimysium → many fascicles → Epimysium (fascia)

- The forces produced by the contracting muscles are transmitted to bone through these connective tissues and tendons
Thin filament (actin 5nm) + thick filament (myosin 15 nm)
Bands of myofibrils

**A bands:** thick filaments in central of sarcomeres

**Z line:** short elements that links thin filaments

**I bands:** thin filaments not overlap with thick filaments

**H zone:** gap between ends of thin filaments in center of A band

**M line:** transverse & longitudinally oriented linking proteins for adjacent thick filaments
Sarcoplasmic reticulum

- Network of tubules & sacs
- Parallel to myofibrils
- Enlarged & fused at junction between A & I bands: transverse sacs (terminal cisternae)
- Triad \{terminal cisternae, transverse tubule\}
- T system: duct for fluids & propagation of electrical stimulus for contraction (action potential)
- Sarcoplasmic reticulum store calcium
Molecular composition of myofibril

- Myosin composed of individual molecules each has a globular head and tail
- Cross-bridge: actin & myosin overlap (A band)
- Actin has double helix; two strands of beads spiraling around each other
- Troponin & tropomysin regulate making and breaking contact between actin & myosin
Molecular basis of muscle contraction

- Sliding filament theory: relative movement of actin & myosin filaments yields active sarcomere shortening
  - Myosin heads or cross-bridges generate contraction force
  - Sliding of actin filaments toward center of sarcomere: decrease in I band and decrease in H zone as Z lines move closer
Motor unit

**Diagram A:**
- Axon
- Motor end plate
- Muscle fiber

**Diagram B:**
- Sarcolemma (muscle fiber membrane)
- Terminal nerve sheath
- Myelin sheath
- Axon

**Diagram C:**
- Axon terminal in synaptic trough
- Synaptic vesicles
- Subneural
Parallel fiber arrangements

Pennate fiber arrangements
A muscle fiber contracts when all sarcomere shorten simultaneously in an all-or-nothing fashion, which is called a Twitch.

The mechanism by which the electric signal trigger's the chemical events of contraction is known as Excitation-contraction coupling.
An **action potential** is initiated and propagated in a motor axon.

This action potential causes the release of **acetylcholine** from the axon terminals at the neuromuscular junction.

Acetylcholine is bound to **receptor sites** on the motor end plate membrane.

Acetylcholine increases the permeability of the motor end plate to sodium and potassium ions, producing an **end-plate potential**.

The end-plate potential depolarizes the muscle membrane sarcolemma), generating a **muscle action potential** that is propagated *over* the membrane surface.
1. Action potential generated is propagated along the sarcolemma and down the T tubules.

2. Action potential triggers Ca\(^{2+}\) release from terminal cisternae of SR.

3. Calcium ions bind to troponin; troponin changes shape, removing the blocking action of tropomyosin; actin active sites exposed.

4. Contraction; myosin cross bridges alternately attach to actin and detach, pulling the actin filaments toward the center of the sarcomere; release of energy by ATP hydrolysis powers the cycling process.

5. Removal of Ca\(^{2+}\) by active transport into the SR after the action potential ends.

6. Tropomyosin blockage restored; blocking actin active site; contraction ends and muscle fiber relaxes.

7. Neurotransmitter released diffuses across the synaptic cleft and attaches to Ach receptors on the sarcolemma.
1. Myosin cross bridge attaches to the actin myofilament

2. Working stroke—the myosin head pivots and bends as it pulls on the actin filament

3. As new ATP attaches to the myosin head, the cross bridge detaches

4. As ATP is split into ADP and P_i, cocking of the myosin head occurs

ATP hydrolysis

Myosin head (high-energy configuration)

Thin filament

Myosin head (low-energy configuration)

New ATP attaches to myosin head

ADP and P_i (inorganic phosphate) released
**Muscle Differentiation (types of fibers)**

<table>
<thead>
<tr>
<th></th>
<th>I (slow-twitch oxidative)</th>
<th>IIA (fast-twitch oxidative glycolytic)</th>
<th>IIB fast-twitch glycolytic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contraction speed</td>
<td>Slow</td>
<td>fast</td>
<td>fast</td>
</tr>
<tr>
<td>Myosin-ATPase activity</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Primary source of ATP production</td>
<td>Oxidative phosphorylation</td>
<td>Oxidative phosphorylation</td>
<td>Anaerobic glycolysis</td>
</tr>
<tr>
<td>Glycolytic enzyme activity</td>
<td>Low</td>
<td>Intermediate</td>
<td>High</td>
</tr>
<tr>
<td>No. of mitochondria</td>
<td>Many</td>
<td>Many</td>
<td>Few</td>
</tr>
<tr>
<td>Capillaries</td>
<td>Many</td>
<td>Many</td>
<td>Few</td>
</tr>
<tr>
<td>Myoglobin contents</td>
<td>High</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Muscle Color</td>
<td>Red</td>
<td>Red</td>
<td>White</td>
</tr>
<tr>
<td>Glycogen content</td>
<td>Low</td>
<td>Intermediate</td>
<td>High</td>
</tr>
<tr>
<td>Fiber diameter</td>
<td>small</td>
<td>Intermediate</td>
<td>Large</td>
</tr>
<tr>
<td>Rate of fatigue</td>
<td>slow</td>
<td>Intermediate</td>
<td>Fast</td>
</tr>
</tbody>
</table>
The Musculotendinous Unit

- Tendon- spring-like elastic component in series with contractile component (proteins)
- Parallel elastic component (epimysium, perimysium, endomysium, sarcolemma)

PEC: parallel elastic component
CC: contractile component
SEC: series elastic component
The Musculo-tendinous Unit

- The tendons and the connective tissues in and around the muscle belly are Viscoelastic structures.
- When stretched during active contraction or passive extension of a muscle, tension is produced and energy is stored; when they recoil with muscle relaxation, this energy is released.
- The series elastic fibers are more important in the production of tension than are the parallel elastic fibers.
Dispensability and elasticity of the elastic components

- Keep the muscle in **readiness** for contraction smooth production and transmission of tension during contraction.
- Assure that the contractile elements **return to their original** (resting) positions when contraction is terminated.
- Prevent the passive **overstretch** thereby lessening the danger of muscle injury.
Absorb energy proportional to the rate of force application and to dissipate energy in a time dependent manner.

when a person attempts to stretch and touch the toes???
Mechanics of Muscle Contraction

- Use of Electromyography
- **Time relationship** between the onset of electrical activity in the muscle and actual contraction of the muscle or muscle fiber.
Summation and Tetanic Contraction

- Neural stimulation – **impulse**
- Mechanical response of a motor unit – twitch—single stimulus
- **Tonic type:** motor units that require more than a single stimulus before the initial development of tension.
- Following stimulation there is an interval of a few milliseconds known as the **latency period** before the tension in the muscle fibers begins to rise.
The time from the start of tension development to peak tension is the **contraction time**.

Time from peak tension until the tension drops to zero is the **relaxation time**.

Muscle fiber makeup

Some muscle fibers contract with a speed of only 10 m sec others may take 100 m sec or longer.

An action potential lasts only approximately 1 to 2 m sec.
When mechanical responses to successive stimuli are added to an initial response, the result is known as **summation**.

If a second stimulus occurs during the latency period of the first muscle twitch, it produces no additional response and the muscle is said to be **completely refractory**.
The greater **the frequency of stimulation** of the muscle fibers, the greater the tension produced in the muscle as a whole.

A **maximal frequency** will be reached beyond which the tension of the muscle no longer increases.

When this maximal tension is sustained as a result of summation, the muscle is said to contract **tetanically**.

Rapidity of stimulation outstrips the contraction relaxation.
Summation and tetanic contraction
Generation of muscle tetanus

Note: muscle is controlled by frequency modulation from neural input very important in functional electrical stimulation
Wave summation & tetanization

Critical frequency
Motor unit recruitment
- All-or-nothing event

- 2 ways to increase tension:
  - Stimulation rate
  - Recruitment of more motor unit

- Size principle
  - Smallest Motor Units recruited first
  - Largest Motor Units last
Types Of Muscle Contraction

- Isometric muscle work
- Isotonic muscle work
  - Concentric M. W
  - Eccentric M.W
The total force that a muscle can produce is influenced by its mechanical properties:

- Force–length characteristics
- Force–velocity characteristics
- Muscle Modeling
- Neuromuscular system dynamics
Length-tension Relationship

![Graph showing the relationship between relative tension and sarcomere length (µm)].

- **Relative Tension**
  - Range: 0.5 to 1.0

- **Sarcomere Length (µm)**
  - Range: 1.27 to 3.6

- **Length-Tension Relationship**
  - 2.25–3.6 µm
  - 2.0–2.25 µm
  - <1.65 µm

The diagram illustrates the increased tension with increased sarcomere length, reaching a maximum at around 2.25 µm, after which tension decreases again.
Single muscle fiber

- Resting 2.0-2.25 um max. no. of cross bridges; max. tension
- 2.25-3.6 um no. of cross bridge ↓
- < 1.65 um overlap of actin no. of cross bridge ↓
Maximal tension is produced when the muscle fiber is approximately at its "slack," or resting, length.

- If the fiber is held at shorter lengths: the tension falls off slowly at first and then rapidly.
- If the fiber is lengthened beyond the resting length: tension progressively decreases.
whole muscle contracting isometrically and tetanically

- The tension produced by both active components and passive components must be taken into account

  - **Active tension**: represents the tension developed by the contractile elements of the muscle

  - **Passive tension**: muscle surpasses its resting length and the non contractile muscle belly is stretched.
Passive tension is mainly developed in the parallel and series elastic components.

When the belly contracts, the combined active and passive tensions produce the total tension exerted.

When a muscle is progressively stretched beyond its resting length, the passive tension rises and the active tension decreases.

One joint muscles normally are not stretched enough for the passive tension to play an important role, but the case is different for two-joint muscles.
Load-velocity Relationship

- Shortening is less with increasing load = concentric contraction
- Lengthening is more rapid with increasing load in eccentric contractions.
Force-time Relationship

- The longer the contraction time, the greater is the force developed, up to the point of maximum tension.
- Slower contraction leads to greater force production because time is allowed for the tension produced.
- Active contraction process is of sufficient duration.
Effect of skeletal muscle architecture

The arrangement of the contractile components affects the contractile properties of the muscle

- The more sarcomeres lie in series, the longer is myofibril
- The more sarcomeres lie parallel the larger the cross-sectional area of the myofibril will be
The **force** the muscle can produce is proportional to the cross-section of the myofibril.

The velocity and the excursion (working **range**) that the muscle can produce are proportional to the length of myofibril.
Muscles with shorter fibers and a larger cross-sectional area are designed to produce force e.g. **quadriceps muscle**

Muscles with long fibers are designed for excursion and velocity. The **Sartorius muscle** has longer fibers and a smaller cross-sectional area and is better suited for high excursion.
Effect Of Pre-stretching

- VARIED RESULTS
- Strong evidences that stretching (Static) may reduce the performance or force production in the muscles.
- ??????
Effect Of Temperature

- A **rise in muscle temperature** causes an increase in conduction velocity across the sarcolemma.

- Increasing **the frequency of stimulation** and hence the production of muscle force.

- Rising of the muscle temperature from **6 to 34°C** results in an almost linear increase of the tension/stiffness ratio.


- **Increased temperature** = **Increased enzymatic activity** of muscle metabolism, **Increased efficiency** of muscle contraction.

- **Increased elasticity** of the collagen in the series and parallel elastic components.

- **Increased extensibility** of the muscle-tendon unit. Results in pre-stretch increases the force production of the muscle??
Muscle temperature increase

- Increase in **blood flow**, which occurs when an athlete "warms up" his or her muscles.
- Production of the heat of reaction generated by **metabolism**, by the release of the **energy of contraction**, and by **friction** as the contractile components slide over each other.
At low temperature 10°C, it has been shown that the maximum shortening velocity and the isometric tension are inhibited significantly.

Decreased pH (acidosis) in the muscle.

At physiological temperature range the role of pH is insignificant.
Effect Of Fatigue

- The ability of a muscle to contract and relax is dependent on the availability of adenosine triphosphate (ATP).
- ATP breakdown should balance the ATP Synthesis.
- Imbalance results in muscle fatigue.
- Chances of Fatigue are even greater in the tetanic phase.
Sources of ATP in muscle

- Creatine phosphate
- Oxidative Phosphorylation in the mitochondria.
- Substrate Phosphorylation during anaerobic glycolysis.
When contraction begins myosin ATPase rapidly breaks down ATP.

The increase in adenosine diphosphate (ADP) and phosphate (Pi) concentrations resulting from this breakdown ultimately leads to increased rates of oxidative phosphorylation and glycolysis.
After a **short lapse** metabolic pathways begin to deliver ATP at a high rate.

During this interval the energy for ATP formation is provided by **Creatine phosphate**, which offers the most rapid means of forming ATP in the muscle cell
- At **moderate rates of muscle activity**, most of the required ATP = process of oxidative Phosphorylation.

- **During intense exercise** = ATP is broken down rapidly = limited cell's ability to replace ATP by oxidative Phosphorylation = **inadequate delivery of oxygen** to the muscle by the circulatory system.
The **glycolytic pathway** = much smaller amounts of ATP from the breakdown of glucose = operates at a much faster rate.

It can also proceed in the **absence of oxygen** = formation of **lactic acid** as its end product.

During **intense exercise**, anaerobic glycolysis becomes an additional source for rapidly supplying the muscle with ATP.
The **glycolytic pathway** has the disadvantage of requiring large amounts of glucose for the production of small amounts of ATP.

After a period of intense exercise, Creatine phosphate levels have become low and much of the muscle glycogen may have been converted to **lactic acid**.
when muscle is operating in its **most efficient state**, a maximum of only approximately **45%** of the energy is used for contraction

**Dissipation energy**
Consequences of fatigue

- Muscle fatigue is first observed by the lack of coordination of movement.
- **Skill** of the person in performing a given action is affected by fatigue.
- Decrease in knee extension observed in runners.
- Reduction in **accuracy, control and speed** of contraction which may predispose an individual to **injury**.
<table>
<thead>
<tr>
<th>Muscle Differentiation (types of fibers)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I (slow-twitch oxidative)</strong></td>
</tr>
<tr>
<td>Contraction speed</td>
</tr>
<tr>
<td>Myosin-ATPase activity</td>
</tr>
<tr>
<td>Primary source of ATP production</td>
</tr>
<tr>
<td>Glycolytic enzyme activity</td>
</tr>
<tr>
<td>No. of mitochondria</td>
</tr>
<tr>
<td>Capillaries</td>
</tr>
<tr>
<td>Myoglobin contents</td>
</tr>
<tr>
<td>Muscle Color</td>
</tr>
<tr>
<td>Glycogen content</td>
</tr>
<tr>
<td>Fiber diameter</td>
</tr>
<tr>
<td>Rate of fatigue</td>
</tr>
</tbody>
</table>
Muscle Fiber Differentiation

- Endurance athletes have type I fibers in abundance e.g. Marathon Runners
- Sprinters have Type II fibers in abundance
In the average population, approximately 50 to 55% of muscle fibers are **type I**

Approx. 30 to 35 percent are **type II A**, and approx. are 15 percent **type II B**, but these percentages vary greatly among individuals.

Genetically determined

Type of training
Muscle Injuries

- Muscle injuries comprise contusion, laceration, ruptures, ischemia, compartment syndromes, and denervation

- These injuries weaken the muscles and can cause **significant disability**.
- **Blunt trauma** can diminish muscle strength, limit joint motion, and finally lead to myositis ossificans.

- Muscle laceration, surgical incisions, and traumatic lesion to muscle tissue and denervation weaken the muscle.
- **Ruptures** in muscles also can cause weakness
- Direct trauma
- Muscle contractions **against resistance** also can lead to tears in muscle tissue
- Acute muscle ischemia and compartment syndromes can cause extensive muscle necrosis.
The many potential causes of compartment syndrome all result in increased pressure within a confined muscle compartment.

- Failure to relieve the pressure rapidly may cause complications that range from weakness and decreased motion to loss of an entire limb.
- Repair of the muscle depends upon many factors.
Muscle Remodeling

- The remodeling of muscle tissue is similar to that of other skeletal tissues such as bone, articular cartilage, and ligament.
Effects of disuse and immobilization

Detrimental effects

- Muscle **atrophy**es on a microstructural and macrostructural level, such as decreased numbers and size of fibers
- **Biochemical** changes occur and affect aerobic and anaerobic energy production.
- Immobilization in a **lengthened position** has a less deleterious effect
Program of **immediate or early motion** may prevent muscle atrophy after injury or surgery

- Cannot be reversed through the use of only **isometric exercises** in case of applied plasters

- Partly **mobile casts** in used
Human muscle biopsy: the type I fibers that atrophy with immobilization; their cross-sectional area decreases and their potential for oxidative enzyme activity is reduced.

Early motion

When muscle is placed under tension: afferent (sensory) impulses from the intrafusal muscle spindles will increase, leading to increased stimulation of the type I fiber.
Although **intermittent isometric exercise** may be sufficient to maintain the metabolic capacity of the type II fiber

- Type I fiber (the postural fiber) requires a more **continuous impulse**.

- **Electric stimulation** may prevent the decrease in type I fiber size and the decline in its oxidative enzyme activity caused by immobilization

- Fibers affected may be in accordance with the sports involved
Effects of physical training

- Physical training increases the cross-sectional area of all muscle fibers: increase in muscle bulk and strength

- Relative percentage of fiber types composing a person's muscles may also change with physical training
The **cross-sectional area** of the fibers affected by the athlete's principal activity increases.

In **endurance athletes**, the area of muscle taken up by type I and type IIA fibers increases at the expense of the total area of type IIB.

**Stretching** increases muscle flexibility, maintains and augments the range of joint motion, and increases the elasticity and length of the Musculotendinous unit.
Relative Role of muscle spindles: extrafusal fibers and intrafusal fibers

Role of Golgi tendon Organs: GTO’S