

Muscle Anatomy & Physiology Chapter 9-10

FUNCTIONS OF MUSCULAR SYSTEM

- 1) Movement
- 2) Posture
- 3) Joint stabilization
- 4) Heat generation (shivering when cold)

CHARACTERISTICS OF MUSCLES

Excitability/irritability:

- Only muscles and nerve can do this
- Able to receive and respond to stimulus
- Usually in response to a chemical (neurotransmitter)
- Other conditions stimulate smooth muscle (such as lack of oxygen, pH imbalance)

Contractility:

- Muscles are UNIQUE in their ability to shorten.
- Muscles do not actively expand
- This is an active process

Extensibility:

- Muscles can be passively stretched beyond resting length
- This is a passive process

Elasticity:

- Muscles have the ability to recoil back to their resting length after stretching.
- This is a passive process

GROSS ANATOMY OF MUSCLES

Each muscle is a discreet organ, and there are about 800 muscles in the body. They are composed of various tissues:

- Skeletal muscle
- Blood vessels (in abundance)
- Nerves (muscle HAS to have a nerve to be functional as nerves stimulate the contraction)
- Connective tissue (the wrappings of muscle)
- Vessels/nerves enter and exit near the center of the belly. These are held in place by CT.

CONNECTIVE TISSUE WRAPPINGS

Functions of CT Wrappings:

- Connect muscle to bone
- Reinforce the muscle
- Contribute to elasticity of the muscle
- Serve as attachment sites for blood vessels, nerves, and lymphatics

Layers of CT Wrappings:

-Whole muscle is surrounded by the EPIMYSIUM (DICT)

-Each whole muscle is made of subunits. Each subunit is surrounded by PERIMYSIUM (fibrous CT). The subunits are called FASCICLES.

-Each fascicle has muscle fibers wrapped in ENDOMYSEUM (areolar CT & reticular CT)

ATTACHMENTS

In direct attachments the epimyseum is fused to the periosteum of bone or periosteum of cartilage. In indirect attachments, wrappings extend beyond the muscle mass. They merge together at the ends of muscles to form TENDONS or APONEUROSES. From here they extend to bone, cartilage or other muscles (this last one is common with facial muscles).

MUSCLE INTERACTIONS...no muscle works by itself!

The PRIME MOVER is the agonist...the one that provides the most force for a given moment. The ANTAGONIST is a muscle that opposes a given movement (triceps oppose biceps in elbow flexion). The SYNERGIST assists the primer mover's action by reinforcing the direction of movement (pulling in the same way) or by stabilizing the agonist's origin. When it does this it is the FIXATOR.

NAMING MUSCLES...they are named in a variety of ways:

- Location (temporalis, intercostals)
- Shape (deltoid, trapezius)
- Relative size (~longus, ~brevis, ~maximus, ~medius, ~minimus)
- Direction of fibers (~obliques, ~rectus, ~transversus)
- Number of origins (biceps brachii, triceps brachii, quadriceps)
- Location of attachment (sternocleidomastoid)
- Action (~flexor, ~extensor, ~adductor, ~pronator)
- Combinations of these

MUSCLE FIBER ARRANGEMENTS

PARALLEL muscles (sartorius) run parallel to the long axis and allow the greatest amount of shortening. Larger "bulgy" muscles may be classified as FUSIFORM (biceps).

PENNATE muscles have a feather appearance. The fibers run obliquely to the axis, which can fit more muscle fibers into the space (like tug of war). This allows for more power for the size of the muscle. Shortening is sacrificed, but they have great power (calf muscle) Pennate muscles may be in various forms: unipennate, bipennate and multipennate (deltoid).

CONVERGENT muscles have a broad origin that tapers to a focused insertion. The advantage is that it is versatile and allows for versatile muscle usage (pectoralis major).

CIRCULAR muscles have fibers that are arranged in concentric rings. This type of muscle guards openings and are sphincters. (obicularis oris)

HOW MUSCLES USE THE BONES...AS LEVERS!

The components of a lever system are the:

- Lever rigid bone
- Fulcurum joint (fixed pivot point)
- Load mass of limb or object (resistance to movement)
- Effort muscular action (force applied to lever)

Three classes of levers.

FIRST CLASS L F E
Used when we change direction or force
Ex: seesaw, scissors, skull on axis
Sometimes mechanical advantage, other times disadvantage
Not very common

SECOND CLASS E L F
Used to increase strength at sacrifice of speed and distance
Ex: wheelbarrow, plantar flexion
Always a mechanical advantage
Not very common

THIRD CLASS F E L
Used to increase speed/distance at the sacrifice of strength
Ex: tweezers, elbow flexion
Always a mechanical disadvantage
Common

MICROSCOPIC MUSCLE FIBER ANATOMY (unique features)

Sarcolemma: -Plasma membrane of muscle cell. It lies just inside endomyseum

Sarcoplasm: -The nonfibbular cytoplasm of a muscle fiber
 -Similar to other cells' cytoplasm, but it contains large amounts of glycosomes and myoglobin

Glycosomes: -Glycogen storage granules

Myoglobin: -molecule of oxygen storage

Nuclei: -Nuclei are peripheral

Multinucleate: -Muscles are big so they have lots of nuclei

Mitochondria: -Numerous mitochondria

Myofibrils: -cytoskeletal elements
 -rod-like dense bundles of contractile proteins
 -run parallel to the long axis
 -80% of cellular volume
 -very dense, so nuclei are pushed off to the side and other organelles are squeezed

Terminal cisternae: -Regular expansions of SER
 -Occur in pairs
 -They form triads

Triad =
2 terminal
cisternae

+

1 transverse
t-tubule

Transverse tubules: -a phospholipids bilayer
 -deep extension of sarcolemma
 -encircle myofibrils between terminal cisternae
 -bring the outside world to deep interior of cell
 -forms triads with terminal cisternae

Sarcomere: -Functional unit of skeletal muscle, lined end to end along myofibril. It is a section of muscle fiber between adjacent Z-Discs...runs from z-disc to z-disc.

Striations: -repeating series of various bands and zones (A, I, H, M, Z)
 -A BAND: wide, dark band
 -I BAND: wide, light band
 -H-ZONE: light region through middle of A-BAND
 -M-LINE: dark line bisecting H-Zone and A-Band (middle)
 -Z-DISC: Dark line bisecting I band (at ends)

-ZONE OF OVERLAP is the region of A-Band between the H-Zone and I-Band

-The bands are created by regular arrangement of myofibrils.

Even smaller structures within the sarcomere are MYOFILAMENTS and FILAMENTS.

THICK FILAMENTS

- Extend entire length of A-Band
- Composed primarily of the protein myosin
- Held in hexagonal 3-D array by myomesin at M-Line
- ~300 proteins bundled together
- Has a long filamentous “tail” portion, which forms the shaft of the thick filament
- The head region is made up of 2 globular subunits
 - One globular subunit has a binding site for actin
 - The other one has a binding site for ATPase, which hydrolyzes ATP for energy for contraction)

THIN FILAMENTS

- Made up of actin, tropomyosin and troponin
- The thin filament is made up of two intertwined F-ACTIN chains, which is comprised of singular G-ACTIN globular subunit.
- Each thin filament has two TROPOMYOSIN. TROPOMYOSIN is a filamentous protein that spirals over the surface of the actin, blocking myosin binding sites on actin. This is the state of the muscle when it is inactive.
- TROPONIN is a complex of three regulatory proteins. It is spaced at regular intervals along thin filaments.

G-Actin is one globular subunit

String them together and you get one F-Actin

Two F-Actins make a thin filament.

SLIDING FILAMENT THEORY OF CONTRACTION states that the muscle contracts because the thin filaments slide past thick filaments. The theory was developed by Hugh Huxley in 1954. It was derived from four observations of contracting muscle...three changes and one lack of a change.

- 1) Z-discs move closer (the sarcomere shortens)
- 2) Width of the I-band decreases & H-band disappears
- 3) Width of the Zone of Overlap increases
- 4) A-band does not change (this is the length of the thick filament, it's not shortening)

CROSS-BRIDGE FORMATION

- First Phase: Previously activated myosin heads bind to sites on actin (thin filaments). Now the head is in a good position for pulling.
- Second Phase: The Power Stroke! The myosin head pivots and releases stored energy (ADP and Inorganic Phosphate). As the head pivots, the myofilaments slide past one another. ADP and P are released!
- Third Phase: Cross-Bridge Detachment! ATP binds to myosin heads to cause release from actin.
- Fourth Phase: "Cocking" of the Myosin Head! ATP is hydrolyzed...energy is transferred to myosin head and it is cocked back into its high-energy configuration. ADP & P remain bound
- Cycle Repeats: Activated myosin heads bind to sites on actin. This binding occurs in alternation so that they are all not attached or detached at once. This ensures smooth movement without slippage. One cycle results in about 1% of shortening. In a typical muscle contraction there are about 30-40 cycles.

EXCITATION-CONTRACTION COUPLING is the series of events that cause a muscle to contract and terminate the contraction. In the ends of nerves are vesicles containing a neurotransmitter. Here's how it goes down:

- 1) Nervous signal reaches the MOTOR END PLATE of the nerve cell (where it meets muscle). Acetylcholine (a neurotransmitter) is released into the space between muscle and nerve.
- 2) This creates a wave of electrical stimulation that spreads along the sarcolemma (the surface only)
- 3) Stimulation travels deep into the cell interior via t-tubules and reaches triads.
- 4) This causes changes in the Sarcoplasmic Reticulum (SR). The terminal cisternae release calcium ions into the sarcoplasm.
- 5) Calcium binds to troponin of thin filament. This causes troponin complex to change shape.
- 6) Troponin rolls into cleft of actin double helix and drags the tropomyosin along with it. The binding sites are now available.
- 7) Myosin binding site on the actin is exposed. Myosin cross-bridge is formed and the contraction cycle begins. This cycle continues as long as there is enough ATP available and enough calcium to keep binding site exposed.
- 8) Termination contraction occurs when...
 - a. Nerve stimulus ends
 - b. Impulse along sarcolemma & T-tubule stops
 - c. Release of calcium from SR ends
 - d. Calcium removed from troponin
 - e. Troponin reconforms, bringing tropomyosin back into position
 - f. Myosin no longer binds to actin

MUSCLE ACTIVATION and ENERGY NEEDS!

MOTOR UNITS are comprised of two components:

- 1) A single nerve cell (motor neuron)
- 2) All the muscle fibers to which it attaches and innervates

Sizes of motor neurons/motor units

Small motor units have one neuron to four or five muscle fibers (approximate). Small motor neurons are found in muscles requiring fine control such as the hand, eye and tongue.

Large motor units have one neuron that innervates hundreds or thousands of fibers. Large motor neurons are used for powerful, gross movements such as the shoulder and leg.

ACTIVATION of MUSCLES

Motor units are activated starting with the smallest ones. The larger units are added later as needed.

FIBER DISTRIBUTION

Fibers belonging to any given motor unit are spread throughout the muscle.

ENERGY SOURCES. Muscles need ATP to:

- Contract the myosin head for the power stroke
- For the myosin head to detach from thin filament

Where does the ATP come from?

Each muscle stores some ATP in the fibers, but it's only enough for 4-6 seconds of contraction. This gets the muscle moving, but it runs out pretty fast. The muscle needs more ATP, and it is generated within the cell in three ways:

- 1) Creatine Phosphate is a high energy molecule found in muscle cells. It stores energy in its phosphate bonds.
 - How does the ATP get extracted from the CP?
 - Phosphate group of CP is transferred to ADP, regenerating ATP and forming creatine.
 - This reaction is catalyzed by creatine kinase.
 - Quantity: ~15 seconds worth (about 100 meter dash)
 - Advantage: Very quick
 - Disadvantage: Creatine phosphate stores are rapidly depleted
- 2) ANAEROBIC GLYCOLYSIS is the FIRST PHASE of all cells' energy metabolism pathway, which converts glucose to pyruvic acid and 2 ATP. Oxygen is not needed for anaerobic glycolysis.
 - In absence of oxygen, pyruvic acid is converted to lactic acid, which can be converted back to pyruvic acid in the liver...but this takes place AFTER exercise.
 - Quantity: ~30-40 seconds of contraction
 - Advantages: Fast! Glucose is readily available
 - Disadvantages: Inefficient use of glucose & lactic acid buildup causes muscle fatigue.
- 3) AEROBIC RESPIRATION takes place when oxygen is available. When aerobic respiration occurs, pyruvic acid is shunted into oxidative phosphorylation. This produces water and CO₂ as waste, which is easily removed through pulmonary ventilation. It supplies 32-34 additional ATP per glucose molecule, so it is very efficient and produces a lot of energy!
 - Quantity: Unlimited energy, depending on one's conditioning. It will keep coming as long as you keep breathing. However, lactic acid makes us stop when we're not in shape.
 - Advantage: Does not require "on board" energy stores
 - Disadvantage: Slow. It may not keep up with energy demands.

WHAT IS MUSCLE FATIGUE?

REVIEW: THE FOUR SOURCES OF ENERGY IN ORDER OF USAGE

- 1) ATP stored in muscle fibers
- 2) Creatine Phosphate
- 3) Anaerobic Glycolysis
- 4) Aerobic Respiration

Fatigue is the physiological inability to continue contracting. There are a few possible causes, but the whole process is not fully understood.

- **ATP Depletion** occurs when energy stores are used up. Complete depletion results in contractures.
- **Lactic acid buildup** causes changes in the pH of the cell. This may alter contractile proteins, but is not likely because of homeostasis. It could, however, be significant in extreme exertion. It is more of a factor in psychological fatigue.
- **Ionic imbalances** occur when potassium and inorganic phosphate levels are altered. This interferes with calcium release from SR.

MUSCLE FIBER TYPES

The speed and length of a muscle contraction is affected by:

- Load
- Motor unit recruitment (how many get involved)
- Muscle fiber type

Muscle fibers are CLASSIFIED by:

Speed of the contraction (speed of ATPase) and the dominant ATP formation pathway.

Two types of ATPase...

Slow twitch fibers
Fast twitch fibers

The dominant ATP formation pathways

Oxidative fibers
rely on aerobic respiration (slower)

Glycolytic fiber
rely on anaerobic respiration (faster)

The body uses three of four possible combinations: Slow oxidative, Fast oxidative and Fast glycolytic

Characteristics	Slow Oxidative	Fast Oxidative	Fast Glycolytic
<i>Speed of Contraction</i>	Slow	Fast	Fast
<i>Fiber color</i>	Deep red	Pink-ish	White (pale)
<i>Myoglobin content</i>	High	High	Low
<i>Glycogen stores</i>	Low	Intermediate	High
<i>Metabolic pathway</i>	Aerobic	Aerobic	Anaerobic
<i>Resistant to fatigue</i>	High	Intermediate	Low
<i>Capillaries</i>	Many	Many	Few
<i>Fiber size</i>	Small <i>(dependent on diffusion)</i>	Intermediate	Large

DISTRIBUTION OF MUSCLE FIBERS

- Most muscles have all fiber types and they are distributed evenly throughout the muscle.
- Some have a predominant type (white meat vs. dark meat)
- Motor units consist of a single fiber type.

USAGE OF FIBER TYPES

- Slow oxidative – endurance muscles (postural muscles)
- Fast glycolytic – power muscles (intense, short-term movement such as sprinting)
- Fast oxidative – intermediate use

ADAPTATION TO EXERCISE

- Endurance training (aerobic)
 - Affects slow oxidative fibers most
 - Tissue changes
 - Increased number of capillaries
 - Increased number of mitochondria
 - Increased myoglobin content (gets more red)
 - Some fast glycolytic fibers convert to fast oxidative.
 - Typically does not increase muscle mass dramatically.
 - Oxidative fibers cannot get too big because they are dependent on diffusion of oxygen from blood.
- Resistance training (anaerobic)
 - Affects fast glycolytic fibers most
 - Tissue changes
 - Increased number of myofilaments & myofibrils
 - Increased number of mitochondria
 - Increased glycogen content
 - Increased amount of CT between cells
 - Some fast oxidative fibers can convert to fast glycolytic (the fibers will convert upon cessation of exercise)
 - Typically increases muscle mass dramatically. This is an increase in muscle fiber size, not adding more cells.

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