CHAPTER 10

LECTURE OUTLINE

I. OVERVIEW OF MUSCLE TISSUE

A. Types of Muscular Tissue

1. Three types of muscle tissue.
    a. Skeletal Muscle:
        1) Location: skeleton
        2) Function: movement heat, posture
        3) Appearance: striated, fibers parallel
        4) Control: voluntary
    b. Cardiac Muscle
        1) Location: Cardiac muscle tissue is found only in the heart wall
        2) Function: pump blood
        3) Appearance: Striated, central nucleus
        4) Control: Involuntary
        5) Cardiac muscle fibers contract when stimulated by their own autorhythmic fibers.
        6) Several hormones and neurotransmitters can adjust heart rate by speeding or slowing the pacemaker
    c. Smooth Muscle
        1) Location: GI tract, uterus, eye, blood vessels
        2) Function: Peristalsis
        3) Appearance: no striations, central nucleus
        4) Control: Involuntary SMOOTH MUSCLE TISSUE

B. Functions of Muscular Tissue
1. Producing body movements
2. Stabilizing body positions
3. Storing and moving substance within the body
4. Generating heat

C. Properties of Muscular Tissue
1. Electrical excitability
2. Contractility
3. Extensibility
4. Elasticity

II. SKELETAL MUSCLE TISSUE

A. Connective Tissue Components

a. Endomysium, Perimysium, epimysium (figure 10.1)

b. Epimysium, perimysium and endomysium are continuos with and form the tendons and ligaments

c. Fascia is a sheet or band of fibrous connective tissue that is deep to the skin and surrounds muscles and other organs of the body.

d. If, the connective tissue layers extend beyond the muscle to form a rope-like structure it is called a tendon, if they form a flat sheet it is called an aponeurosis.

e. Clinical Connection: Fibromyalgia

1. Nerve and Blood supply

a. Skeletal muscles are well supplies with nerves and blood vessels.

b. Capillaries are plentiful in muscular tissue (Figure 10.9d)

B. Microscopic Anatomy of a Skeletal Muscle Fiber

1. Microscopi anatomy of a skeletal muscle fiber.

a. Sarcolemma, Tranverse Tubule and sarcoplasm

1) Figure 10.2b,c

2) Transverse tubules are tiny invaginations of the sarcolemma that quickly spread the muscle action potential to all parts of the muscle fiber.

b. Triad: transverse tubule, sarcoplasmic reticulum

1) terminal cisternae

2) The sarcoplasmic reticulum encircles each myofibril. It is similar to smooth endoplasmic reticulum in non-muscle cells and in the relaxed muscle, functions to store calcium ions.

3) Sarcoplasm is the muscle cell cytoplasm and contains a large amount of glycogen for energy production and myoglobin for oxygen storage.

4) Clinical Connection: Muscular hypertrophy, fibrosis and atrophy.

2. Filaments and the sarcomere (Table 10.1).

a. A band, Z disc, M line, I band, H zone

1) The darker middle portion is the A band consisting primarily of the thick filaments with some thin filaments overlapping the thick ones.

2) The lighter sides are the I bands that consist of thin filaments only.

3) Z disc passes through the center of the I band.

(a) Exercise can result in torn sarcolemma, damaged myofibrils, and disrupted Z discs

4) the narrow H zone in the center of each A band contains thick but no thin filaments.

5) Myomesin forms the M line.

C. Anatomy of the myofibril

1. Muscle Proteins (table 10.2)
   a. Titan, M line, thick and thin filaments
   b. Contractile proteins (figure 10.4)
      1) actin
      2) myosin
   c. Regulatory proteins
      1) troponin
      2) tropomyosin
         (a) In relaxed muscle, tropomysium, which in held in place by troponin, blocks the myosin-binding sites on actin preventing myosin from binding to actin.
   d. Structural proteins
      1) titin
         (a) Titin helps a sarcomere return to its resting length after a muscle has contracted or been stretched.
      2) Nebulin helps maintain alignment of the thin filaments in the sarcomere.
      3) Alpha-actinin: binds actin to titin
      4) Myomesin: form the M-line
      5) Dystrophin reinforces the sarcolemma and helps transmit the tension generated by the sarcomeres to the tendons (figure 10.2d).

III. CONTRACTION AND RELAXATION OF SKELETAL MUSCLE FIBERS

A. **The Sliding Filament Mechanism**: During muscle contraction, myosin cross bridges pull on actin filaments, causing them to slide inward toward the H zone; Z discs come toward each other and the sarcomere shortens, but the myosin and actin filaments do not change in length. The sliding of filaments and shortening of sarcomeres causes the shortening of the whole muscle fiber and ultimately the entire muscle. This is called the sliding filament mechanism.

A. Illustrate the progressive overlap of the thick and thin filaments as they pull the A disc toward the center of the sarcomere, and the result on the length of the fibril, fiber, and muscle (figure 10.5).

B. Define the steps involved in the sliding filament mechanism of muscle contraction (figures 10.5 and 10.6).
   a. ATP hydrolysis
   b. Attachment
   c. Power Stroke
   d. Detachment

C. Illustrate the structural relationships of the Sarcoplasmic Reticulum, Transverse tubules, and sarcomere and how they generally function as part of the excitation-coupling mechanism. Relate the role of calcium.

D. Illustrate the relationship of sarcomere length (myofilament overlap) and discuss the amount of tension that results from changes in length (figure 10.5).

B. Excitation-Contraction coupling

A. And increase in intracellular Ca++ starts the contraction

B. Ca++ increase occurs because of release from the sarcoplasmic reticulum (figure 10.7a,b)

C. Ca++ active transport pumps restore the Ca++

D. Calesequestrin helps concentrate the Ca++ near the site for release for the sarcoplasmic reticulum

E. Clinical connection: Rigor Mortis

C. Length-Tension Relationship

A. Figure 10.8. length-tension indicates how the forcefulness of muscle contraction depends on the length of the sarcomeres within a muscle before contraction begins.

D. The Neuromuscular Junction

A. The muscle action potential releases calcium ions from the sarcoplasmic reticulum that combine with troponin, causing it to pull on tropomyosin to change its orientation, thus exposing myosin-binding sites on actin (Figure 10.9a) and allowing the actin and myosin to bind together.
   a. The use of calcium ions to remove the contraction inhibitor and the joining of actin and myosin constitute the excitation-contraction coupling, the steps that connect excitation (a muscle action potential propagation through the T tubules) to contraction of the muscle fiber.

b. Calcium ion active transport pumps return calcium ions to the sarcoplasmic reticulum.
c. Clinical Connection: Electromygraphy – measures the electrical activity of muscle cells.

B. Show the general features of the neuromuscular junction that allows signals coming from the brain to be conveyed across the gap between the neuron motor cell and the sarcolemma of the muscle cell.
a. Synaptic vesicles containing acetylcholine
b. Motor end plate
c. Sarcolemma

C. Describe the steps and components in the mechanism that cause a motor neuron action potential to result in a muscle cell action potential (figure 10.10).
a. Acetylcholine released from vesicles
b. Binding to receptor
c. Graded potential is elicited and then an action potential
d. Acetylcholine is broken down

D. Describe the interaction of acetylcholine with its membrane receptor

E. Describe how binding by acetylcholine results in the simultaneous movement of Na+ and K+

F. Describe the movement of the action potential along the sarcolemma

G. Several plant products and drugs selectively block events at the NMJ.

H. Review the components of the action potential
a. Depolarizing phase
b. Repolarizing phase
c. After-hyperpolarizing phase

I. Connect the action potential phases to the open and closed status of membrane channels in the sarcolemma.

J. Review the initiation of the action potential to the sarcolemma and subsequent propagation to the T-tubules.

K. Summarize the major role players in the excitation-coupling mechanism
a. Brain and associate neurons
b. Acetycholine, receptors and enzymes
c. Na+ and K+ channels and permeabilities
d. Sarcolemma components

e. Myofibril components (troponin, tropomyosin, myosin)

f. ATP

L. The inability of a muscle to maintain its strength of contraction or tension is called muscle fatigue; it occurs when a muscle has low calcium, creatine phosphate, oxygen and other nutrients.

a. Elevated oxygen use after exercise is called recovery oxygen uptake (rather than the formerly used term oxygen debt).

IV. MUSCLE METABOLISM

A. Skeletal Muscle Metabolism

A. Describe energy use in muscle cells

a. Stored ATP

b. Creatine Phosphate

1) Creatine phosphate and ATP can power maximal muscle contraction for about 15 seconds and is used for maximal short bursts of energy (e.g., 100-meter dash) (Figure 10.11).

2) Creatine phosphate is unique to muscle fibers.

3) Clinical Connection: Creatine Supplementation. There is a controversy regarding the effectiveness of creatine supplementation

c. Anaerobic Glycolysis

1) Glycolysis

(a) The partial catabolism of glucose to generate ATP occurs in anaerobic cellular respiration. This system can provide enough energy for about 30-40 seconds of maximal muscle activity (e.g., 300-meter race).

2) Lactic acid

(a) Describe the chemical fate of lactic acid when exercise has ceased and oxygen is being delivered as fast as it is needed (aerobic metabolism)

d. Muscle Fatigue

1) Central fatigue: caused by changed in the central nervous system

B. Oxygen Consumption after Exercise

a. Oxygen debt – extra oxygen used to pay back or restore metabolic conditions in three ways

b. Replenish creatine phosphate stores

c. Convert lactate into pyruvate

d. Muscular activity lasting more than 30 seconds depends increasingly on aerobic cellular respiration (reactions requiring oxygen). This system of ATP production involves the complete oxidation of glucose via cellular respiration (biological oxidation).

e. Muscle tissue has two sources of oxygen: diffusion from blood and release by myoglobin inside muscle fibers.

f. The aerobic system will provide enough ATP for prolonged activity so long as sufficient oxygen and nutrients are available.

V. CONTROL OF MUSCLE TENSION

A. The Motor Unit

A. A motor neuron plus all the muscle cells that innervate it (Figure 10.12)

a. A single motor unit may innervate as few as 10 or as many as 2,000 muscle fibers, with an average of 150 fibers being innervated by each motor neuron.

B. Twitch contraction

A. The brief contraction of all muscle fibers in a motor unit in response to a single action potential.

B. Muscle contractions are recorded by a **myogram** (figure 10.13)

C. **Latent period**: the delay following an action potential and the onset of contraction

D. **Contraction period**: Ca++ binds to troponin and peak contraction is developed

E. **Relaxation period**: time that Ca++ is transported back into the sarcoplasmic reticulum.

F. **Refractory period**: the period of lost excitability

C. Frequency of Stimulation

A. The second contraction will occur stronger than the first (figure 10.14b)

B. Wave summation: stronger contractions caused by stimuli arriving at different times.

C. Tetanus: unfused or incomplete – sustained but wavering contraction. Fused or complete tetanus – individual twitches cannot be detected (figure 10.14d).

D. Motor Unit Recruitment

A. Motor unit recruitment – the process in which the number of active motor units increases.

B. Clinical connection: Anaerobic training vs Aerobic training

E. Muscle Tone

A. Even at rest, a skeletal muscle exhibits muscle tone, or a small amount of tension

B. Clinical Connection: Hypotonia and Hypertonia

F. Isotonic and Isometric Contractions

A. Isotonic contraction – tension is constant while the muscle changes length

a. Concentric isotonic contraction – when the length of the muscle shortens and the object is moved (figure 10.15a)

b. Eccentric Isotonic contraction – when the length of the muscle increases or lengthens during contraction (figure 10.15b)

B. Isometric contraction – muscle contracts but does not change length (figure 10.15c)

VI. SKELETAL MUSCLE FIBER TYPES

A. Types of skeletal muscle fibers (Table 10.4)

A. Slow oxidative fibers

a. Small, dark red, fatigue resistant

B. Fast oxidative-glycolytic fibers

a. Intermediate size, dark red, moderately resistant to fatigue

b. Use the cross section to show how must skeletal muscles are mixtures of all types.

C. motor units are recruited in specific orders depending on the task required.

D. Color varies according to the content of myoglobin, an oxygen-storing reddish pigment. Red muscle fibers have a high myoglobin content while the myoglobin content of white muscle fibers is low.

E. Fiber diameter varies as do the cell's allocations of mitochondria, blood capillaries, and sarcoplasmic reticulum.

F. Contraction velocity, method of ATP production and resistance to fatigue also differ between fibers.

B. Exercise and Skeletal Muscle Tissue (table 10.4)

A. Effective stretching

a. Stretching is most effective when the muscles are warm, it is important to warm up before stretching

B. Strength Training

a. The process of exercising a muscle and adding resistance for the purpose of strengthening the musculskeletal system.

b. Clinical Connection: Anabolic Steroids

C. Cardiac Muscle Tissue (figure 20.9)

A. Intercalated discs – contain desmosones and gap junctions that allow muscle action potentials to spread from one muscle fiber to another (figure 4.2e)

B. Contractions last 10 15 times longer than skeletal muscle (figure 20.11)

C. Mitochondria are larger and more numberous

D. Smooth muscle Tissue

A. Usually activated involuntarily

B. Single unit – contract as one single unit, similar to cardiac muscle (figure 10.16a)

C. Multiunit – consists of individual fibers that contract independently

D. Microscopic Anatomy of Smooth Muscle

a. Thick in the middle and tapered at the end (Figure 10.16c)

b. Contain intermediate filaments in addition to thick and think filaments

c. Filaments are connected to structures called dense bodies which are functionally similar to Z discs (figure 10.16c).

E. Physiology of Smooth Muscle

a. Contraction and relaxation are regulated by calmodulin and myosin light chain kinase.

b. Smooth muscle cells can stay contracted long term

c. Hormones can also influence the state of contraction (Table 15.2)

d. Smooth muscle fibers will contract in response to stretch

E. Regeneration of Muscular Tissue

A. Hypertrophy – the enlargement of existing cells

B. Hyperplasia – an increase in the number of fibers

a. Table 10.5 is a summary of the major features of the three types of muscular tissue

F. Development of Muscle (figure 10.17)

A. Mesoderm – all muscle of the body are derived from mesoderm

B. Somites – segmentation of the mesoderm

a. Cells of somite differentiate into three regions

1) Myotome – forms head, neck and limbs

2) Dermatome – forms the dermis of the skin

3) Sclerotome – gives rise to the vertebrae

VII. Aging and Muscular Tissue

A. Between the ages of 30 and 50, humans undergo a slow, progressive loss of skeletal muscle mass that is replaced by fibrous connective tissue and adipose tissue

B. Lower limb muscle atrophy before the upper limbs

VIII. DISORDERS: HOMEOSTATIC IMBALANCES

A. Neuromuscular disease involves problems involving somatic motor neurons, neuromuscular junctions, or muscle fibers. Myopathy signifies a disease or disorder of the skeletal muscle tissue itself.
B. Myasthenia gravis is an autoimmune disorder characterized by great muscular weakness and caused by antibodies directed against ACh receptors at the neuromuscular junction; more ACh receptors are affected as the disease progresses, making the muscle increasingly weaker.
C. Muscular dystrophies are inherited muscle-destroying diseases that are characterized by degeneration of individual muscle fibers, leading to progressive atrophy of the skeletal muscle. The most common form is Duchenne muscular dystrophy, for which there is hope that gene therapy may someday be employed to replace the responsible gene with one that halts muscle loss.
D. Skeletal muscle fibers cannot divide and have limited powers of regeneration; growth after the first year is due to enlargement of existing cells, rather than an increase in the number of fibers (although new individual cells may be derived from satellite cells).
E. The number of new skeletal muscle fibers formed from satellite cells is minimal.
F. Extensive repair results in fibrosis, the replacement of muscle fibers by scar tissue.
G. Cardiac muscle fibers cannot divide or regenerate.
H. Smooth muscle fibers have limited capacity for division and regeneration.