Ch 6: Muscle Physiology

1. Contrast among 3 types muscle (skeletal, cardiac, smooth) Text Pg 108

<table>
<thead>
<tr>
<th>a) Skeletal Muscle</th>
<th>b) Cardiac Muscle</th>
<th>c) Smooth Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Voluntary (somatic motor)</td>
<td>Involuntary (autonomic motor)</td>
<td>Involuntary (autonomic motor)</td>
</tr>
<tr>
<td><strong>Neurotransmitter</strong> = ACh</td>
<td><strong>Parasymp. Neurotrans.</strong> = ACh</td>
<td><strong>ACh with muscarinic cholinergic receptors</strong>,</td>
</tr>
<tr>
<td><strong>Receptor</strong> = nicotinic cholinergic for EPSPs)</td>
<td><strong>Sympath. Neurotrans</strong>= Epineph. receptor = B1 adrenerfic</td>
<td><strong>Epinephrine with B2 &amp; α-adrenergic receptors</strong></td>
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<tr>
<td>&amp; also Glycine &amp; GABA with muscarinic receptors (Ch 4) (for IPSPs – muscle relax)</td>
<td></td>
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<tr>
<td>Requires somatic motor neuron stimulus to contract (not “autorhythmic”)</td>
<td>Is “autorhythmic”, but HR influenced by ACh (↓HR) &amp; epinephrine (↑HR)</td>
<td>Is “autorhythmic” – influenced by ACh or epinephrine</td>
</tr>
<tr>
<td>Fastest contraction speed</td>
<td>Intermediate contraction speed</td>
<td>Slowest contraction speed</td>
</tr>
<tr>
<td>Prone to fatigue</td>
<td>Fatigue resistant</td>
<td>Fatigue resistant</td>
</tr>
</tbody>
</table>

2. Muscle structure (Text pg 107)

> organ > fascicle > fiber (cell) > myofibril > sarcomere

3. Skeletal muscle divided into 2 types of fibers: (Text Pg 109)

1) **slow twitch (red) fibers** for aerobic, long term use (fatigues slowly)
   > many capillaries, much myoglobin, lots of mitochondria

2) **fast twitch (white) fibers** for short bursts of contraction (fatigues quickly)
   > fewer capillaries, less myoglobin and mitochondria

4. The sarcomere

Actin (thin) myofilament
   > has protein troponin (that Ca+2) binds to
   > has tropomyosin (that covers active sites, preventing myosin heads from binding)
   > active sites – where myosin heads want to bind

Myosin (thick) myofilament
   > has heads that can bind to active sites on actin to form crossbridge.

5. Steps involved in skeletal muscle contraction (sliding filament theory of contraction) (Text Pg 109-110)

Neuromuscular junction = between somatic motor neuron and all the muscle fibers it innervates.

1) motor neuron releases ACh into synapse
2) ACh binds to nicotinic cholinergic receptors on muscle fibers, opens Na+ channels, causes EPSP
3) AP travels through muscle cell & causes Ca+2 release from sarcoplasmic reticulum.
4) Ca+2 binds to troponin on Actin myofilament.
5) causes tropomyosin to lift of active sites on actin
6) myosin myofilament heads bind to active sites (form crossbridges) & pull on actin
7) the actin & myosin “slide” past each other until the sarcomere shortens, which shortens the muscle organ

6. The use of ATP and ADP in muscle contraction (shortening of sarcomeres)
   > ATP is needed to break crossbridge between myosin heads and active sites, and put myosin head back into “locked and ready” position. But ATP reduced to ADP.
   > ADP is used for myosin to grip active sites and pull (power stroke), but then all energy is lost.

7. Rigor mortis – stiffening of body, due to whole body muscle tetany, 12-18 hrs post mortem.
   Body loosen after 24-36 hrs due to myofilaments beginning to necrose.

8. Types of muscle contraction: (Text pg 110)
   1) Isotonic – where muscle contraction produces enough force to move a mass (muscle shortens)
   2) Isometric – where muscle has tone but doesn’t shorten.

Review

9. Factors influencing muscle contractile strength (force)
   Motor unit = one motor neuron and all of the muscle fibers it innervates
   Power vs Precision arrangement:
     > muscle power = one motor neuron innervates MANY fibers (as much as 150) for power, but has little precision/control.
     > muscle precision = one motor neuron innervates few fibers. Not much power, but good precision.

Contractile forces depend on:
1) # of fibers responding. More fibers = more power. Less fibers = less power.
2) strength of stimulus. Stronger stimulus (more neurotransmitter) = more power.
   Weaker stimulus (less neurotransmitter = less power. Graded response!

Recruitment = if stimulus is very strong, and one motor unit is not producing enough power, multiple motor units will be recruited. Produce more power than one unit alone.

3) frequency of stimulus
   > muscle twitch = one stimulus and one contraction. (not strong)
   > muscle treppe = repeated low frequency stimuli, muscle can relax in between stimuli, causes increasing contractile force with each stimulus, until plateau reached. Muscle warm-up.
   > muscle summation = repeated higher frequency stimuli, muscle can’t relax in between, causes greater rise in contractile force with each stimulus, until plateau reached.
   > muscle tetani = highest frequency of stim., produces highest force, BUT muscle gives out.

Sleep twitches (myoclonus or myoclonic jerk) – a.k.a. hypnagogic massive jerk. Pg 118
   > involuntary muscle movement (twitches) that wakes you up.
   > might be changes in muscle as enter REM sleep, when go from conscious to unconscious state (due to GABA inhibition of muscles).
Review

10. Energetics of muscle contraction.
   Muscle Fatigue Pg 112
   > Dep;ete O2, ATP, g;lycogen, myoglobin
   > Accumulate CO2, ADP, lactic acid, phosphate

   **Phosphocreatine** = molecule stored in muscle, needed to convert ADP to ATP (donates a phosphate molecule)

   **Creatine phosphokinase (CK or CPK)** = enzyme in skeletal muscle, brain, and heart, which is needed to convert creatine into phosphocreatine.

   **Isoforms of CPK:**
   > CPK MM = increased with diseased/damaged skeletal muscle
   > CPK BB = increased with brain damage
   > CPK MB = increased with heart damage

11. Muscle growth and repair
   > **muscle satellite cells** = muscle stem cells for muscle repair and growth
   > **myostatin** = chemical that inhibits satellite cells to slow muscle growth. It increases w/age.

12. Muscle Atrophy = decrease in muscle mass (due to many factors) Pg 112, 116-117
   > lack of use, injury or disease, injury to somatic nerves

13. Muscle Disorders (Pg 112-113)
   > **Muscle spasm** = muscle seizure or convulsion
   > **Muscle cramp** = results from strenuous activity, involves lactic acid buildup. Could also be due to dehydration & loss of electrolytes.
   > **Muscle sprain** = joint injury (involves over-stretched or torn ligament)
   > **Muscle strain** (pulled muscle) = a muscle, or its tendon, is overstretched or torn.

   Tx for muscle sprain/strain = RICE (Rest, Ice, Compression, Elevation) and anti-inflammatories

   **Dermatomyositis** = disorder of muscle inflammation (**myopathy**) Pg 116
   > 1/100,100, predominantly women
   > presentation = muscle weakness that progresses, affects muscle close to trunk, skin rashes.
   > Tx ; anti-inflammatories (prednisone or NSAIDs)

   **Muscular Dystrophy (MD)** – Duchenne’s most common Pg 117
   > sex-linked recessive (affects males more)
   > loss of protein dystrophin in muscle
   > childhood onset with motor problems & muscle atrophy. Soon wheelchair-bound.

   **Amyotrophic lateral sclerosis (ALS)**
   > loss of motor neurons, leading to muscle atrophy & eventual paralysis
   > onset in 40’s
   > starts in motor neurons of hands & feet
> life expectancy after diagnosis < 5yrs
> thought to be due to loss of antioxidant superoxide dismutase & glutamate toxicity.

**Myasthenia gravis** = autoimmune attack on ACh receptors (both nicotinic and muscarinic)
> loss of motor control and muscle atrophy
Tx with ACh agonists (ACh-EI’s) like neostigmine

**Tetanus (hypertonia) & botulism (hypotonia)**

**Review**

14. **Muscle sensory organs**
   1) **Golgi tendon organs** = sense muscle tension (from pull on tendons)
   2) **Muscle spindle apparatus** = senses muscle stretch (through tendon)
      > **extrafusal fibers** – thick contracting fibers (fast, thick, stronger, more numerous) involved in isotonic contraction.
      > **intrafusal fibers** – thin stretch fibers (slower, weaker, less numerous) involved in isometric contraction. (muscle tone but no shortening)

15. **Upper and lower motor neurons in muscle contraction**
   1) **Upper motor neurons** (originate in primary motor cortex of frontal lobe)
   2) **Lower motor neurons** (somatic motor neuron) in brainstem and ventral spinal cord
      > extend into major nerves of body
   Have A) **alpha neurons** – innervate extrafusal contractile (isotonic) muscle fibers of muscle spindle.
   B) **gamma neurons** – innervated intrafusal (stretch) muscle fibers for muscle tone.
      > are regulated by feedback from golgi tendon and muscle spindle apparatus

16. **Voluntary vs Spinal Reflex Muscle Movement.**
   **Voluntary muscle contraction** = longer neuron pathway (If someone says, “when I touch your shoulder, kick out your leg”).
   > touch receptors respond
   > signal ascends spinal cord
   > signal goes to brain (sensory cortex)
   > Motor cortex responds
   > motor command send down spinal cord to leg
   > leg contracts

   **Spinal Reflex** = involuntary reflex the relays an important signal (life or body-saving) from sensory receptor to spinal cord and out to skeletal muscles.

4 Spinal Reflexes:
**1. Knee jerk reflex (patellar tendon reflex)**
> Tapping patellar tendon stretches tendon & quadriceps muscle - stimulates spindle fiber (stretch receptor) in muscle
> Stimulating spindle fiber evokes action potentials in sensory neuron
> Sensory neuron synapses directly with alpha somatic motor neuron in spinal cord.
> Alpha motor neuron stimulates contractile muscle fibers
This is ex. of monosynaptic reflex > Only one synapse is crossed (in spinal cord)

2. **Inhibitory Stretch Reflex** (protects tendon from excessive muscle contractile force when stretched)
> Muscle is stretched, muscle tendon is stretched, which stimulates AP in Golgi tendon organ (a sensory organ)
> Sensory neuron goes into spinal cord & stimulates (+) an interneuron (spans distance between dorsal to ventral horn)
> Interneuron stim inhibitory (−) neurotransmitter to alpha motor neuron
> Effect = Reduces tension in tendon to prevent damage from excessive stretching

This is ex. of disynaptic stretch reflex = Two synapses are crossed in spinal cord

3. **Reciprocal Innervation** – While the primary muscle contracts (stimulation on one side) the antagonistic muscle on opposite side is inhibited.
> Stretch of primary muscle & tendon stim. sensory neuron. Sensory info enters dorsal spinal cord, crosses over to ventral horn & does two things:
> Positive (+) stim. of primary muscle to contract.
> Inhibition (−) of antagonist muscle (stays relaxed).

4. **Crossed Extensor Reflex or double reciprocal innervation**
Ex. Painful stimulus on right foot stim sensory neuron, goes into dorsal horn spinal cord. Crosses to ventral horn on left and right sides of cord and does two things:

> Right leg Flexors contract (+) and extensors relax (−) to withdraw injured foot on R.
> Left leg, Extensors contract (+) and flexors relax (−) to put leg down & support body weight.