EXAM #3 INCLASS/ONLINE LECTURE SUMMARIES

CHAPTER 8 Movement

MUSCLES

1. Smooth Muscle- Found in internal organs. Are long, thin cells

2. Skeletal/Striated Muscle- Are the main movers. Are Long cylindrical fibers w/ stripes

3. Cardiac Muscle- Found in heart. Fused fibers contract together**.** Each fiber innervated by one motor neuron. One motor neuron innervates many fibers.

MOTOR UNIT - smallest functional unit. Neuron meets fiber at ***NEUROMUSCULAR JUNCTION***

- ***acetylcholine - excites muscle/contraction.***
- **Antagonistic muscles** work together to move perfect amount

FLEXORS vs. EXTENSORS

MYASTHENIA GRAVIS - *Autoimmune Disorder. Antibodies act against ACh receptors*

• ***Symptoms*** – progressive weakness. rapid fatigue of skeletal muscles

• ***Physiological Basis*** – fewer ACh receptors. Morphological changes at synapse

– action of : ***ACETYLCHOLINESTERASE*** - *lower likelihood ACh reaches enough receptors*

• **Treatments** – immunosuppressants. Acetylcholinesterase inhibitors

• ***FAST-TWITCH FIBERS*** – Contract and relax rapidly. Anaerobic. Fatigue rapidly. Generate greatest force (EX: sprinting)

• ***SLOW-TWITCH FIBERS*** – longer contraction time. Aerobic. Resistant to fatigue. Generate less force (EX: walking)

PROPRIOCEPTORS - Sensitive to position/movement of muscles. Detect stretch/tension. Allow spinal cord to adjust signal.

• **Two primary types of proprioceptors:** **- Muscle Spindles** **- Golgi Tendon Organs**

MUSCLE SPINDLEs - Parallel to muscle. Senses Stretch::: Muscle stretched -> Signal to motor neuron in spinal cord -> Muscle contraction. • Negative Feedback = *stretch causes contraction.* Stimulates ***STRETCH REFLEX***

GOLGI TENDON ORGANS - Located in tendons (connecting muscles to bone). Senses Tension. Vigorous muscle contraction::: Signal to spinal cord -> inhibitory interneurons -> Inhibit motor neuron (muscle contraction)

• Negative Feedback = *tension causes muscle relaxation.* Protects against too vigorous a contraction

MOVEMENT

- **Reflexes** – consistent, automatic responses to stimuli. EX: Babinski reflex (in babies; when foot touched -> toes curl)

– ***allied reflexes*** - occur together/elicit each other

• **Ballistic Movements** – executed as a whole. cannot be corrected/not sensitive to feedback. (EX: reflexes)

MOTOR PROGRAMS - Neural circuits w/ fixed sequence of movements. ***Built-in*** *(Ex:* rodent grooming, yawning) vs. ***learned*** (EX: gymnast, pianist)

• **Central Pattern Generators**: Neural mechanisms that generate rhythmic motor patterns(EX: wet dog shake. wing flapping)

SOMEMAJOR MOTOR AREAS OF THE BRAIN

FOREBRAIN = • **Cortex** • **Basal Ganglia** • **Cerebellum**

MIDBRAIN = • **Substantia Nigra** • **Red Nucleus**

HINDBRAIN = • **Reticular Formation** • **Reticular Formation** • **Vestibular Nucleus**

MAJOR MOTOR AREAS OF THE BRAIN

• **Primary Motor Cortex** - Involved with coordinated movements in several muscles leading to a specific outcome.

• **Posterior Parietal Cortex** - Involved with coordinating movement through the environment based on visual input.

• **Prefrontal Cortex / Premotor Cortex** - Involved in planning movement.

• **Supplementary Cortex** – Involved in preparation for rapid sequences of movements.

PRIMARY MOTOR PATHWAYS TO SPINAL CORD

DORSOLATERAL TRACT - Carries axons from primary motor cortex & red nucleus(midbrain) to spinal motor neurons.

• Crosses over to the contralateral side at the ***pyramids*** in the ventral portion of the anterior medulla (***pyramidal tract***).

• Controls movements of distal limbs including hands fingers and toes.

• Courses down the dorsolateral portion of the white matter of the spinal cord.

VENTROMEDIAL TRACT - Carries axons from primary motor cortex (& vestibular nucleus/tectum/reticular formation & other cortical areas). Axons synapse on spinal interneurons (and some motor neurons) controlling spinal motor neurons.

• Some of the axons cross and others don't, providing bilateral innervation.

• Controls movements of proximal limbs and axial musculature (neck/shoulders/trunk). Involved w/ movements and posture.

• Courses down the ventromedial portion of the white matter of the spinal cord.

MOTOR FUNCTIONS OF CEREBELLUM - The cerebellum contains more neurons than the rest of the brain combined.

• Involved in: Control of rapid ballistic movements (including saccades) • Timing • Establishment of new motor programs

CEREBELLAR DAMAGE can lead to trouble:

• Tapping a rhythm • Athletics • Speaking and writing • Hand clapping • Typing
• Playing musical instruments • Finger-to-nose test • Simple saccades

BASAL GANGLIA

• **CAUDATE NUCLEUS / PUTAMEN** - Primarily input area. Receives info from thalamus and cortex. Also receives dopaminergic projection from the substantia nigra (in midbrain)
• **GLOBUS PALLIDUS** - Primarily an output area. sends info to thalamus, which sends to motor/prefrontal cortex/midbrain.
* **BASAL GANGLIA** - Role still somewhat unclear. Might be involved in: Organization of action sequences into chunks, inhibition of specific motor responses. Basal ganglia role can be known by examining Parkinson's Disease.
PARKINSON'S DISEASE- MUSCLE RIGIDITY/TREMORS • SLOW MOVEMENTS • COGNITIVE DEFICITS
• DIFFICULTY INITIATING MOVEMENT • DEPRESSION • AFFECTS 1 IN 100 ABOVE AGE 50
* CAUSED BY : Degeneration of the dopaminergic neurons in the substantia. Nigra projecting to caudate nucleus/ putamen.
POSSIBLE CAUSES OF PARKINSON'S DISEASE
• Early onset (< 50 years) has a genetic component.
• Possible environmental influence. Environmental Toxins; e.g.: MPTP converted to MPP; which accumulates in opaminergic neurons and kills them. Cigarette/Caffeine has a protective effect. (less likely w/ smoking/caffeine)
TREATMENT FOR PARKINSON'S DISEASE: Unlike dopamine, **L-dopa** can cross blood-brain barrier where it is absorbed by neurons and can increase dopamine production.
• It can't restore degenerated neurons, however. Effectiveness declines as the disease progresses. Side-effects are a problem.
OTHER POSSIBLE TREATMENTS (largely experimental)
• Antioxidant drugs to decrease further damage. • Dopaminergic agonists that can cross blood-brain barrier.
• Glutamate or adenosine antagonists • Inactivating electrical stimulation of globus pallidus
• Neurotrophins to promote growth and survival of neurons. • Drugs that decrease apoptosis. • Fetal tissue transplants
• Drugs that block certain calcium channels abundant in elderly brains • Drugs that stimulate cannabinoid receptors.

CHAPTER 10 Regulation of Internal States

REGULATION OF INTERNAL STATE
HOMEOSTASIS - Processes that maintain various physiological states within a fixed range.
SET POINT - A specific value of an internal state that the body defends. (Temperature)
FLUID and MINERAL BALANCE
BODY WATER CONTENT- Between 45% - 70% of the body is water. Individuals with more fat have less water.
DISTRIBUTION OF BODY WATER • **1/3 Extracellular** • **2/3 Intracellular**
OSMOSIS - Water moves across membrane from area of **low** solute concentration to an area of **high** solute Concentration.
• occurs until concentration of dissolved particles in the **intracellular** and **extracellular** fluid are **equal**.
• intracellular / extracellular concentration of particles is referred to as ***osmolarity.***
• The normal concentration of dissolved particles in the intracellular and extracellular fluid = ~300 mM.
ISOTONIC: 300 mM, normal concentration of solute
HYPOTONIC: <300 mM, less than normal concentration of solute
HYPERTONIC: >300 mM, greater than normal concentration of solute in extracellular andintracellular fluid.
THIRST : Two Mechanisms • **OSMOTIC THIRST** • **HYPOVOLEMIC THIRST**
OSMOTIC THIRST -Caused when solute concentration in **ECF** increases. This causes **ICF** volume to decrease/cells shrink.
• Osmoreceptor neurons, especially in ***organum vasculosum of the laminae terminalis (OVLT),*** respond. Osmoreceptors in the OVLT stimulate neurons in the ***paraventricuclar nucleus (PVN)*** & ***supraoptic nucleus (SON)*** to release ***vasopressin*** (***antidiuretic hormone (ADH)***) in the circulation of the ***posterior pituitary.***
• Circuits (?) controlling drinking behavior are stimulated and ADH causes kidney to reabsorb water.
HYPOVOLEMIC THIRST - Caused by loss of solutes/water from ECF. This causes blood pressure to drop. Blood pressure change is sensed by **baroreceptors** in several parts of the body. (Kidney/Heart/Aorta)
• Change in [Na+] is sensed in the kidney.
• Baroreceptors in the heart and aorta send input to the brain through the glossopharyngeal and vagus nerves.
* Low Blood Volume => Kidney Releases Hormone **RENIN** (Angiotensin I **ANGIOTENSIN II**) Constricts blood vessels
* **ANGIOTENSIN II** Stimulates adrenal cortex to release **ALDOSTERONE.** Stimulates cells in **SUBFORNICAL ORGAN (SFO)** Stimulates drinking behavior Causes kidney to reabsorb Na+ Stimulates brain to promote Na+ appetite
CONTROLS OF FEEDING - Most mammals eat in discrete bouts called meals.
WHAT MECHANISMS TURN MEALS ON? **WHAT MECHANISMS TURN MEALS OFF?**
PERIPHERAL FACTORS - ORAL FACTORS - Taste/smell/texture/temperature of food can promote/discourage feeding.
• **SHAM FEEDING - SENSORY SPECIFIC SATIETY - PERIPHERAL FACTORS**
STOMACH – *Excitatory* - ***Grehlin*** is an ***orexigenic*** hormone that is released by stomach. Its circulating blood levels increase with fasting and decrease after a meal.

- *Inhibitory* - Gastric filling stretches the stomach and activates sensory receptors in the stomach wall and these signals are transmitted to the brain through two routes: ***vagus nerve*** and ***splanchnic nerves (spinal)***.

PERIPHERAL FACTORS - INTESTINAL FACTORS - Intestines are sensitive to stretch w/ chemoreceptors. It transmits these signals through the **vagus** and **splanchnic** nerves.

• **CHOLECYSTOKININ (CCK)** - Hormone released by **duodenal** cells that closes the **pyloric sphincter** between stomach & intestines & stimulates receptors on vagus nerve. CCK doesn't cross blood-brain barrier, but some brain neurons responsive to neural signals from the intestines release CCK. CCK is involved in regulation of food intake (like intestinal CCK)
- **POSTABSORPTIVE FACTORS** - Fats & complex carbohydrates are broken down to fatty acids & simple monosaccharides then are absorbed through the wall of intestines into the circulation where they contact a variety of tissues & endocrine glands.
• **LIVER (glycogenolysis, glycogenesis)** • **PANCREAS (glucagon, insulin)** • **ADIPOCYTES (leptin)**
Leptin is an ***anorexigenic*** hormone released by **adipose** tissue in proportion to fat mass.
INSULIN - High Blood Glucose. Stimulates *Insulin* Release from Pancreas. Promotes Entry of Glucose into Muscle
*** Affects Neurons in Hypothalamus to Reduce Feeding**
GLUCAGON - Low Blood Glucose. Stimulates *Glucagon* Release from Pancreas. Stimulates Glycogenolysis
CENTRAL FEEDING CIRCUITS
Early views : *Ventromedial Hypothalamus (VMH) = "Satiety Center"• Lateral Hypothalamus (LH) = "Hunger Center"*
VENTROMEDIAL HYPOTHALAMUS = (Satiety Center) | LATERAL HYPOTHALAMUS = (Hunger Center)
LH LESIONS: INTERPRETIVE ISSUES - LH lesions cause sensory-motor neglect and some of the feeding and drinking deficits might be secondary. Damage to dopaminergic systems sending fiber tracts that course through the LH can cause similar effects (However, fiber-sparing lesions of cells bodies are also effective).
• LH lesions causes a decrease in insulin. Thus, some of the weight loss may be secondary to this hormonal effect.
• VMH lesions cause metabolic disturbances including increases in circulating insulin. VMHX rats will still gain weight even if their food intake is restricted.
• Damage must be large, usually including areas surrounding the VMH, to produce consistent effects.
• Rats with VMH lesions will not over eat all food. They eat less of unpalatable foods than control animals do. Thus, it is not as thought VMHX rats are "so hungry they will eat anything."
• Damage to fiber tracts passing through the area, such as the ventral noradrenergic bundle, can also lead to the effects on feeding and weight gain.
+ -**HYPOTHALAMIC INVOLVEMENT IN FEEDING** - LH and VMH and other areas of the hypothalamus play a significant role in feeding and energy regulation. There are a variety of neuropeptides and neuropeptide receptors found in neurons of the hypothalamus that are known to be involved in increasing or decreasing feeding.

CHAPTER 11 Reproductive Behavior

SEX HORMONES - TWO CLASSES OF SEX HORMONES
• **ANDROGENS** - "male hormones" (e.g., ***testosterone***) • **ESTROGENS** - "female hormones" (e.g., ***estradiol***)
PRODUCTION OF SEX HORMONES
• **STERIODS (derived from *cholesterol*)** -- Act either on: 1) Protein receptor sites on target cell membrane activating second messenger cascades, or 2) Intracellular receptor sites that migrate into nucleus and affect gene expression.
• **PRODUCED PRIMARILY IN GONADS (adrenal cortex can make some).** *Males: Testicles *Females: Ovaries
• **MALES & FEMALES PRODUCE BOTH CLASSES.**
* Males: [Androgens] > [Estrogens] * Females: [Estrogens] > [Androgens]
TESTOSTERONE - Maturation of male genitalia. Promotes sperm production. Growth of facial, pubic, and axillary hair (in females this occurs from androgens released by the adrenal cortex).
• **Muscular development (anabolic steroid).** Enlargement of larynx. Associated w/ sexual arousal in males. Increases bone thickness, but inhibits bone length.
ESTROGENS - Maturation of female genitalia. Promotes growth of uterine lining. Breast development. Broadening of hips. Associated with sexual arousal in females. Increases bone thickness, but inhibits bone length.
PRIMARY SEX CHARACTERISTICS - Primary Sex Characteristics Are Present At Birth :
• **GONADS = Testes (males) / Ovaries (females)**
• **INTERNAL REPRODUCTIVE STRUCTURES** - males: vas deferens(duct from testes-penis)/seminal vesicles(hold semen)
* females: oviducts (duct from ovaries to uterus)uterus and upper vagina
• **EXTERNAL GENITALIA – (Penis / Vagina)**
SECONDARY SEX CHARACTERISTICS - Secondary Sex Characteristics Appear At Puberty
• FACIAL ((males)), PUBIC, & AXILLARY HAIR • WIDENED HIPS and BREAST DEVELOPMENT ((females))
• MUSCULAR DEVELOPMENT and LARYNX ENLARGEMENT (males)
SEX HORMONES CHANGE : ORGANIZATIONAL EFFECTS - Permanent alterations in anatomy, physiology, and Behavior. Often times associated with the presence of a specific hormone during a ***sensitive period*** in development.
• **ACTIVATIONAL EFFECTS** - Causes reversible alterations in anatomy, physiology and behavior.
ORGANIZATIONAL EFFECTS: DEVELOPMENT OF INTERNAL REPRODUCTIVE ORGANS :
• Primordial Internal Reproductive Organs are Bisexual. Both systems are present early in gestation.
• ***Mullerian Ducts*** - Primordial Female Internal Reproductive Organs.• ***Wolffian Ducts*** - Primordial Male Internal Reproductive
ORGANIZATIONAL EFFECTS: DEVELOPMENT OF EXTERNAL GENITALIA
• Primordial External Genitalia are Unisexual Can develop into male or female form.