Sensorimotor system:

- **What are skeletal muscles?**
  - Large organization of several muscle cells (fibers).
  - Muscle cells rich in myosin (including cross bridges) and actin filaments (bind the heads of cross-bridges).
  - Extrafusal muscle fibers contacted by alpha motor neurons.

- **How do muscle fibers contract?**
  - When alpha motor neurons fire, produce endplate potential in muscle fiber at neuromuscular junction through the release of acetylcholine.
  - Endplate potential produces action potential in muscle fiber = calcium release from sarcoplasmic reticulum via voltage-dependent calcium channels.
  - This induces “ratcheting” movement of myosin cross bridges along actin filaments and the muscle “twitches”.

- **What is a motor unit?** A single (one) alpha motor neuron with its associated axon, neuromuscular junction, and the muscle fibers it innervates (touches).

- **What is a motor pool?** All motor neurons that innervate fibers of a single muscle.

- **How is muscular contraction controlled?**
  - Weak muscular contraction = few motor units activated;
  - Strong muscular contraction = lots of motor units activated;
  - For fine muscular control - use small motor units = single alpha motor neurons connected to only a few muscle fibers;
  - For gross muscular control - use big motor units = single alpha motor neurons connected to hundreds of muscle fibers.

- **Where are alpha motor neurons located?** Motor neurons are localized to the ventral horn of the spinal cord and some of the nuclei in the brainstem giving rise to motor cranial nerves of the neck and above.

- **What's the difference between glabrous and hairy skin?** Glabrous skin is hairless, whereas hairy skin is located throughout the rest of the skin.

- **What are the 3 groups of somatosensory receptors?**
  1. Nocioceptors: involved in temperature and pain sensation (free nerve endings).
  2. Hapsis: involved in fine touch and pressure sensation (several including Pacinian and Ruffini corpuscles, etc).
  3. Proprioceptors: involved in the perception of body in space (body awareness – muscle spindles, Golgi tendon organs, joint receptors).

- **What is the phenomenon of “referred pain”?** Some interneurons in the spinal cord receive pain information from both superficial (skin) tissues and deep tissues (abdominal and thoracic organs). Thus, when an internal organ sends pain information, some of that pain is felt as if coming with the associated skin areas. A good example is the aches and pains felt in the upper left arm when an individual experiences a heart attack.
- **What are the major pathways via which mechanosensory information reaches the brain?**

  1. The dorsal columns provide touch and pressure sensation from skin which synapse in the dorsal column nuclei (gracile and cuneate nuclei) in the medulla (same side of the body as information entered spinal cord). Dorsal column nuclei then send their axons, via the medial lemniscus, to the contralateral (cross midline) ventrolateral thalamus. The thalamic neurons send their axons to the primary somatosensory cortex (post-central gyrus).

  2. The ventral spinothalamic tract provide pain and temperature sensation directly to the thalamus. This information is ascending the spinal tract contralateral to the side of the body it entered. Thalamic neurons carrying this information also send their axons to the primary somatosensory cortex.

  3. The spinocerebellar tract provide proprioceptive information to the cerebellum, but some of this information also reaches the thalamus and primary somatosensory cortex.

- **What is the monosynaptic stretch reflex?**

  Uses muscle spindle receptor which signals muscle length via spindle afferent neuron (sensory neuron).

  Muscle spindle length controlled by intrafusal muscle fibers and intrafusal (gamma) motor neuron. Once length of spindle receptor is set, if muscle gets stretched, spindle afferent neuron contacts alpha motor neurons directly to increase firing and restore muscle length (involves a single synapse = monosynaptic reflex).

- **What is a polysynaptic reflex?** Example is the withdrawal reflex from a painful stimulus.

  Involves at least two synapses.

  Concept of antagonist muscles = flexor and extensor muscles at different joints.

  Painful stimulus triggers activity in pain sensory neurons that contact both excitatory and inhibitory interneurons in the spinal cord.

  Excitatory interneurons excite motor neurons involved in flexor contraction.

  Inhibitory interneurons inhibit motor neurons contacting extensor muscle.

  Produces concerted action to allow withdrawal of limb from painful stimulus.

- **What is the crossed extensor reflex?**

  It is an extension of the polysynaptic reflex by which the contralateral (crossed) extensor reflex is triggered when the flexor reflex occurs in one limb (particularly the legs).

- **What is the Golgi tendon reflex?**

  Golgi tendon organ is located at junction of tendon and muscle and is sensitive to stretch.

  Sensory neurons relaying information from Golgi tendon organ only contacts inhibitory interneurons in the spinal cord, and can only inhibit alpha motor neuron activity.

  This reflex is triggered if tension on muscles and tendon are extreme enough to possibly produce damage.

- **What is the part of the brain that controls voluntary movements?** The primary motor cortex. This region of the brain, like the somatosensory cortex, is organized somatotopically (i.e., from head to toes and disproportionate amounts of cortex are devoted to different parts of the body (lots of cortex devoted to fine motor control – fingers, lips – while relatively little cortex devoted to back muscles).

- **What tells the primary motor cortex what to do?**

  Prefrontal cortex, which is important for the planning of movements.

  This information is passed to the premotor areas, which include the supplementary motor area, the premotor cortex and the cingulate motor area, which in turn “tell” the primary motor cortex what sequence of muscle contractions to perform.

  The supplementary motor area is involved in guiding behavior from internally generated stimuli – intentions.
The premotor cortex is involved in guiding behavior from external stimuli – example from visual guidance of walking around. Cingulate motor area guides behaviors based on emotional states. Feedback about movement is also obtained from the somatosensory cortex.

- **How does the primary motor cortex “talk” to the muscles?**
  1. Corticospinal tract (direct inputs from motor cortex to contralateral spinal cord).
  2. Corticobulbar tract (direct inputs from motor cortex to cranial nuclei innervating contralateral neck and above).
  3. Corticorubral and rubrospinal tracts (indirect inputs to spinal cord from motor cortex with a synapse in red nucleus – important for feedback from cerebellum).
  4. Vestibulospinal tract (involved in the control of balance and innervates upper legs and lower trunk muscles).
  5. Tectospinal tract (involved in visual pursuit – innervates upper trunk and neck muscles).
  6. Reticulospinal tract (involved in programmed motor control such as walking and running – innervates especially leg muscles).

- **What is the basal ganglia and what is its role in the control of movement?**
  Basal ganglia consists mainly of the caudate/putamen, globus pallidus and part of the thalamus. Mostly regulates the force of voluntary movements.

- **What are some diseases associated with basal ganglia dysfunction?**
  1. Parkinson’s disease: loss of dopaminergic inputs from substantia nigra to caudate/putamen. Leads to hypokinetic (reduced force) movements.
  2. Huntington’s disease: loss of neurons in the caudate and putamen. Leads to hyperkinetic (too much movements - involuntary) disruptions.

- **What is the role of the cerebellum in the control of movements?**
  Does not initiate movement. Uses inputs from primary motor cortex, vestibular nuclei and sensorimotor inputs (proprioception) from body. Compares intentions (planned movements) with performance as sensed from proprioceptors. Predicts future body positions. Involved in ballistic body movements which are too rapid for feedback to be effective.

**Sleep and wakefulness:**

- **What are the stages of sleep?**
  Non-REM Stage 1 (light sleep) – Stage 4 (deep sleep)
  REM (rapid eye movement or paradoxical sleep – vivid dreaming)
  Normal progression of sleep goes 1>2>3>4>3>2>1>REM>1>2 . . . and lasts about 90 minutes per episode. REM sleep time increases as night progresses.

- **What is the EEG and how does it correspond to the different stages of sleep?**
  The electroencephalogram is used to measure gross electrical activity of the entire brain. EEG shows different brain activity with different levels of wakefulness and sleep:
  1. Awake = Beta (BBuzzy) waves; fast and low amplitudes;
  2. Drowsy = Alpha (AAhhh) waves; slower and higher amplitudes;
  3. Stage 1 = slower waves; Theta waves
  4. Stage 2 = slower waves with sleep spindles and K complexes (NOT SAME AS PGO SPIKES);
  5. Stage 3&4 = Delta (DDeeep sleep) waves (1-2 Hz); slowest, largest amplitudes;
  6. REM = fast and low amplitudes Beta waves; paradoxical.
**What are the different characteristics of deep, slow-wave vs. REM sleep?**
- more slow-wave sleep early in the night but more REM sleep late in the night;
- synchronized vs. desynchronized EEG;
- absent vs. present rapid eye movements;
- normal vs. flaccid muscle tone (muscle atonia);
- absent vs. present sexual activation;
- absence vs. presence of PGO waves;
- static vs. vivid, action packed dreams.

**What are the main theories of why we sleep?**
1. Passive mechanism: decrease in sensory stimulation associated with evening is simply conducive to sleep.
2. Adaptive (circadian) mechanisms: mostly evolved to keep organisms “out-of-trouble” during certain parts of the day.
   - Evidence from very different sleep patterns in different species.
3. Restorative mechanisms: mostly evolved as a necessity for the body to “restore” itself.
   - It goes like this: wakefulness promotes chemicals that may accumulate in our body and signal the brain regions involved in sleep to become active and initiate sleep to rid the body of these chemicals.
   - So far, no single molecule has been unambiguously associated with the induction of sleep. Like food, and water, sleep is a necessity.
   - Evidence include higher rates of “restorative” hormones and protein synthesis during sleep.
4. Sleep and memory storage: Proposal that either REM or non-REM sleep is important for memory storage (remember video and idea that different types of memory may be affected by sleep deprivation).

**What are the main sleep disorders?**
1. Problems in initiating/maintaining sleep:
   a) Insomnia - inability to fall asleep;
   b) Sleep apnea - cessation of breathing which can wake people up;
   c) SIDS - related to apnea in infants;
   d) Sleeping pills - although help to fall asleep, are very addictive and disrupt normal sleeping patterns - leave users with feeling of sleepiness.
2. Excessive somnolence:
   Narcolepsy - sleep attacks during any time of the day, related to REM.
   - Symptoms of narcolepsy:
   - Extreme daytime sleepiness and sleep attacks;
   - Cataplexy - sudden muscle weakness;
   - Sleep paralysis - complete loss of muscle tone while remaining conscious;
   - Vivid hypnagogic hallucinations - goes into REM sleep quickly - difficult to tease reality from dreaming apart.
3. Sleep-wake cycle disturbances: Involves disturbances of circadian body clock located in suprachiasmatic nucleus of the hypothalamus; even without time cues, body keeps 24-25 hrs rhythm.
   a) Delayed sleep phase syndrome - like living in a different time zone.
   b) Jet lag and work shifts - sleep disturbances produced by traveling to a different time zone, or shifting circadian cycle.
4. Parasomnias - various behaviors associated with sleep - some disturbing, others not.
   a) Sleepwalking (somnambulism) - not really a problem, normally fades by puberty.
   b) Bed-wetting (enuresis) - often wakes child up, can produce anxiety.
   c) Night terrors - awakening in a panic, normally forgotten by morning, subsides with age.
and different from nightmares.

d) sleep talking - can be associated with REM or non-REM sleep.

- **What are lucid dreams?**
  Dreams in which the dreamers are “aware” of their dreams and can even influence the course of the dream (in REM sleep).

- **How does the brain control sleep?**
  1. Bremer suggested that sleep is a “passive” process whereby the lack of sensory stimulation = sleep.
  2. Moruzzi and Magoun suggested that sleep is an “active” process whereby activity in specific regions of the brain is responsible for sleep.
     Evidence from electrical stimulation of pontine reticular formation (known as the ascending reticular activating system) in sleeping cats wakes them up.
     Evidence from partial cut only through midbrain reticular formation (which leaves sensory input relatively intact) produces sleeping EEG in forebrain.
     Reasoned that area between the two sections contain brain regions important for actively inducing sleep and wakefulness.

- **What are the brain regions involved in sleep?**
  1. Basal forebrain region – activates basal forebrain cholinergic system (which contains the neurotransmitter acetylcholine) – also important for circadian rhythm (example - temperature control) - important in producing desynchronized EEG (BUZZ, waking waves).
  2. Pontine reticular formation (ascending reticular activating system) – involved in waking state.
  3. Median Raphe nuclei (serotonin containing neurons) also involved in desynchronizing EEG (waking Beta waves).
  4. Peribrachial area – cholinergic neurons – active during REM sleep – lesions in this area reduced REM sleep.
     - projects to medial pontine reticular formation (MPRF).
  5. MPRF – also an area where cholinergic agonists (carbachol) increases the duration of REM sleep.
     - projects to subcoerulear nucleus which activates magnocellular nucleus of the medulla – involved in muscle atonia (loss of muscle tone).
     - also projects to basal forebrain cholinergic area – involved in EEG desynchronization.
  6. Subcoerulear nucleus - if damaged , loss of muscle atonia (cats “act” their dreams).

**Neuroplasticity, Learning and Memory**

**What is neuroplasticity?** It is a reorganization of the brain (strengthening, weakening, formation or elimination of synapses).

**When does neural plasticity occur?**
- during development;
- during learning and memory;
- during injury and recovery from brain injury.

**How is plasticity expressed during development?**
  1. **Neural proliferation** - cell division (in humans occurs from week 5 to the fifth month of gestation).
  2. **Cell migration** - undifferentiated nerve cells (neuroblasts) need to reach their final destination after their birth.
  3. **Differentiation** - neuroblasts start growing neurites (axons and dendrites).
  4. **Axon growth** - neurites extend growth cones with associated filopodia, probing chemical environment of developing brain.
5. **Synapse formation** – pre- and post-synaptic specialization takes place, begin releasing neurotransmitters.

6. **Neuron death**: too many neurons produced during development; neurons compete to obtain neurotrophic factors (ex., NGF, BDNF) – if a neuron does not get enough NGF, it dies.

7. **Synaptic capacity** – each neuron can make a finite number of synapses based on its total area; this number is greatly reduced during the first few years of adolescence.

**What are examples of the effects of Experience (environment) on Neural Development?**

1. **Early visual experience**: early (during so called “critical period”) monocular visual deprivation (covering of one eye) produces the loss of ability of the deprived eye to activate visual cortex. - idea of competition (active open eye “competes” out axons from deprived eye, so open eye controls all visual cortex – normally, each eye controls about 50% of visual cortex). - these are produced by the loss of axons to the visual cortex that were activated by the deprived eye.

2. **Environmental effects**: enriched environments (many toys and exercisers) vs. impoverished environments (simple cage) make huge difference on cortical development. Enriched environments produce:
   - thicker cortex;
   - better capillary supply;
   - larger dendritic fields;
   - more glial cells;
   - larger postsynaptic zones;
   - larger protein content

**What is learning?** A relatively permanent change in behavior that results from experience and/or practice with the environment.

**N.B.** This excludes maturation processes, damage or injury, or fatigue processes.

**What is memory?** Ability to recall or recognize previous experience in the form of behavioral change.

**What are the types of learning?**

1. **Non-associative learning**: simple forms of learning that do NOT require the formation of associations.
   A. **Habituation**: a decrease in responding that occurs with repeated exposures to the same stimulus.
      Example: habituation of the gill-withdrawal reflex in *Aplysia Californica* (type of sea snail).
   B. **Sensitization**: an increase in responding that occurs with repeated exposures to the same stimulus.
      Example: facilitation of the gill-withdrawal reflex in *Aplysia*.

**N.B.** Short-term habituation is produced by repeating a stimulus only a few times; if the stimulus is repeated many times over days, long-term habituation can be observed. Similarly, a single electric shock (in the case of *Aplysia*) normally produces short-term sensitization, but several electric shocks can produce long-term sensitization.
2. **Associative learning**: more complex forms of learning that do require the formation of associations.

   **A. Classical (Pavlovian) conditioning**: the process by which a neutral stimulus acquires meaning through association with another stimulus (often a biologically relevant stimulus).

   **Terminology**:
   - UCS: unconditioned stimulus (food, water, electric shock, etc.)
   - UCR: unconditioned response (salivation, etc.)
   - CS: conditioned stimulus (bell, light, etc.)
   - CR: conditioned response (salivation to bell, etc. - CR is the “learned” response).

   **Examples**:  
   
<table>
<thead>
<tr>
<th>UCS</th>
<th>UCR</th>
</tr>
</thead>
<tbody>
<tr>
<td>pinprick</td>
<td>withdrawal</td>
</tr>
<tr>
<td>food</td>
<td>salivation</td>
</tr>
<tr>
<td>sudden loud noise</td>
<td>startle</td>
</tr>
<tr>
<td>airpuff to the eye</td>
<td>eyeblink</td>
</tr>
</tbody>
</table>

   **How is Classical conditioning produced?**
   - repeated pairings of CS + UCS -> UCR
     (bell) + (food) (salivation)
   - CS (bell) eventually comes to elicit salivation WITHOUT presenting the food.
   - when the CS produces salivation, it is now called the conditioned response (CR).

   **N.B.** Conditioning works best when the CS immediately precedes the UCS; it does not work if the CS is presented after the UCS.

   **What is the neural basis of classical conditioning?**
   - Classical conditioning of **emotional responses** such as obtained by the repeated pairings of an auditory stimulus with footshock (produce CR of “freezing” and increased heart rate (can be obtained after only a few pairings).
   - **Amygdala** is necessary to produce classical conditioning of **emotional responses**.
   - Classical conditioning of **motor responses** such as obtained by the repeated pairings of a visual stimulus with airpuffs to the eye (produce CR of eyeblink in response to visual CS - normally requires 100s of pairings).
   - **Cerebellum** is necessary to produce classical conditioning of **motor responses**.

3. **Instrumental conditioning (Operant conditioning)**: process by which motor responses are learned when associated with a favorable outcome (reinforcer - B.F. Skinner).

   **Terminology**:
   - **Discriminative stimulus**: cue that triggers the motor response (ex., sight of level bar).
   - **Favorable outcome**: - positive reinforcers (food, water, etc.).
     - negative reinforcers (termination of electric shock, restraint, etc.).

   **Example of instrumental conditioning**: Rats learn to press a bar in order to obtain food or water.

   **What is the neural basis of instrumental conditioning?**
   - **Basal ganglia** (caudate/putamen) necessary for instrumental conditioning.
   - **Nucleus accumbens** necessary for “detection” of reinforcers.
Human Memory:

What are the processes involved in memory formation?
1. Sensory registers (high capacity, low duration) with attention - produce
2. Short-term memory (low capacity, low duration) with consolidation - produce
3. Long-term memory (high capacity, high duration).

What are the types of memory retrieval?
1. Recognition memory (ex., multiple choice type exam).
2. Recall memory (ex., essay type exam).

What are the main types of human memory?
1. Declarative (explicit): accessible to conscious awareness
   A. Episodic (memory for time and place).
   B. Semantic (memory of facts and knowledge).
2. Procedural (implicit): accessible only through performance of tasks in which knowledge is embedded (ex., riding a bicycle, walking, etc.).

What were some of the early hypotheses about the neural basis of memory?
1. Concept of engram: Carl Lashley championed the study of the engram – the biological substrate of memory (neural memory trace).
   - Principle of mass action – memories are stored diffusely all over neocortex;
   - Principle of equipotentiality – neocortex all over the brain plays an equal role in memory storage.
   - studies of human amnesia proved these principles wrong.
2. Concept of consolidation: Donald Hebb proposed a neural mechanism for the transfer of short-term memories into long-term storage.
   - concept of cell assemblies and reverberation – neural activity in collection of neurons reverberates (keeps going around) and produces strengthening of synaptic connections between those neurons = long-term memory.

What are some of the lessons from human amnesics (explicit memory)?
1. H.M.: bilateral removal of medial temporal lobes (lobectomy – including parts of temporal cortex, amygdala, and most of the hippocampus).
   Importance of H.M.’s case for field of memory research:
   1. Implicated medial temporal lobe structures in memory processes (direct challenge to view of diffuse memory storage from Lashley).
   2. Supports distinct short-term and long-term memory processes.
   3. Demonstrated the existence of explicit vs. implicit memory.
   4. Suggested medial temporal lobe important for consolidation.

What are some of the brain regions associated with implicit memory?
1. Amygdala: Responsible for emotional memories.
2. Cerebellum: Responsible for motor learning.
3. Basal ganglia: Responsible for many unconscious motor and verbal memories.