

## Learning

- **HM's Tests:**
  1. Digital span +1 test: *declarative* memory (explicit)
    - Short-term memory: retain phone number long enough for one use
      - HM could barely do this
    - Tests long-term memory: retain phone number for a whole day
      - HM could *not* do this
  2. Block Tapping Memory Span Test: *spatial* memory (explicit)
    - HM was required to remember a tapping sequence to see if his lesion affected non-declarative memory as well.
      - He could only remember the tapping sequence up to 5 blocks and could not remember how to get to his house
  3. Mirror drawing test: *long-term sensorimotor* skill (implicit)
    - Sensorimotor tasks could be recalled from day to day
      - He remembered how to preform the task, but could not remember actually taking the test.
  4. Incomplete pictures test: *visual perceptual skill* (implicit memory)
    - Implicit visual memory also called priming memory
      - He preformed very well with these tasks, indicating that MTL is independent of priming memory
  5. Classical conditioning: associative learning (implicit memory)
    - CS = tone; UCS=air puff; Response = eye blink
      - HM was slow to learn, but retained it for years
        - MTL is independent of associative learning
- **Conclusions from HM**
  1. Medial Temporal lobes play an important role in memory
    - Memory functions are not equally distributed throughout the brain
  2. Different structures are involved in forming working and long term memories
    - HM could retain long term memories and suffered almost no retrograde amnesia and could also retain several forms of short term memory
  3. Memories of different nature (implicit and explicit)
    - HM maintained implicit memory, but lost long-term explicit memory
- **Generalization of MTL Amnesia:**
  - o *Loss of long-term explicit memory formation and retention of long-term implicit memory formation*
- Other types of amnesia
  1. Korsakoff's syndrome
    - Caused by: Alcoholism/vitamin B1 deficiency/diffuse damage to neocortex, hippocampus, and cerebellum

- Early stage: anterograde amnesia for episodic memory
  - Later stages: severe retrograde amnesia, personality changes, and extreme confusion
2. Alzheimer's disease:
- Initially mild deterioration of memory and intellect leads to confusion, anxiety, irritability, major anterograde and retrograde amnesia resulting in widespread neural death
  - Reduced ACh level due to deterioration of basal forebrain and other diffuse damage
  - Narrowed gyri, wider sulci, enlarged ventricles
  - Amyloid plaques: large extracellular sheers of peptides arranged in sheets block cell-to-cell signaling
  - Neurofibrillary tangles: paired helical threads of proteins in neurons that disrupt vital cell transport systems
    - i. Both are prevalent in rhinal cortex, hippocampus, and neocortex and are probable cause of memory deficits
3. Post-traumatic amnesia:
- A blow to the head produces coma, followed by a period of confusion after the victim regains consciousness
    - i. Victim has period of retrograde amnesia for events hat occurred during the period right before the blow and anterograde amnesia for events that occurred during the period of confusion

## Memory

- Hippocampus and consolidation:
  - o **Standard consolidation theory**: affected memories transferred to a more stable storage during a set time period (STM to LTM)
  - o **Continuous Consolidation theory**: Initial memory encoded in hippocampus and/or neocortex and with each recall, a physical change in the brain occurs that stores memory (*engram*) that makes disruption more difficult
    - Consolidation continues indefinitely and some memories will be consolidated preferentially over time
  - o **Reconsolidation**: each time a memory is recalled from LTM, it is in an easily-altered state in STM and must be reconsolidated
- **Reference memory**: the general principle required to perform a task (ex: go to the baited arms)
- **Working (short-term) memory**: the memory necessary for successful performance of the task
  - o Both are affected by hippocampal lesions in rats (short-term memory not dependent on hippocampus in humans)
- **Hippocampal place cells and entorhinal grid cells**

- Grid cells establish a grid representation of a location so that during movement place cells can fire according to their new location while orienting according to the reference grid of their external environment.
  - Grid cells allows species to understand their position in space
  - A place cell becomes active when an animal enters a particular space in the environment (place field)
- **Hippocampal role in learning/memory:**
  - **Hippocampal cognitive map theory:** Hippocampus is specialized for spatial location memory and constructs/maintains maps of the external world
  - Also involved in the organization and flexible expression of **non-spatial memory**
    - Ex: hippocampal neurons fire in a place field, but only when a non-spatial stimulus is there also
- **Rhinal cortex role:**
  - *Object recognition*
    - Mumby box – rat learns that food is next to a specific object/visual cue and remembers that visual stimulus even when placed in a different setting with other arbitrary visual cues
- **Inferotemporal cortex:**
  - *Memories of visual input*
- **Prefrontal cortex:**
  - *Temporal (timely) order of events and working memory* (attention and task management)
    - Phineas Gage
- **Cerebellum:**
  - Pavlovian conditioning and sensorimotor skills
- **Amygdala:**
  - *Role in memory for emotional significances of experiences* (it itself does not store memories, rather strengthens emotionally significant memories stored in other areas)
  - Fear conditioning.
- **Striatum** (caudate and putamen): *habit formation*
- **Long-term Potentiation**
  - Long-lasting enhancement in signal transmission between two neurons that results from stimulating them synchronously; modification of synaptic strength.
  - Synaptic plasticity demonstrated in a number of areas implemented in learning and memory
    - Protein synthesis required for maintenance
    - Increased number and thickness of dendritic spines
    - Changes in dendritic branching