LEARNING AND MEMORY

Chapter 11
Learning and Memory

Learning and Memory
• You cannot have learning without memory, vice versa

Learning Studies
• Studied with classical or operant conditioning, rats in lab

Memory Studies
• Studied with recall of words or events
Theories of Learning

Classical Conditioning

- Learning to make a reflex response to a stimulus (other than the original stimulus that produced the reflex)
- Explains involuntary stimulus-response relationships
<table>
<thead>
<tr>
<th>Classical Conditioning</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Unconditioned Stimulus (UCS)</strong></td>
</tr>
<tr>
<td>- A naturally occurring stimulus that leads to an involuntary (reflex) response</td>
</tr>
<tr>
<td>Dog food</td>
</tr>
<tr>
<td><strong>Unconditioned Response (UCR)</strong></td>
</tr>
<tr>
<td>- An involuntary (reflex) response to a UCS</td>
</tr>
<tr>
<td>Salivation</td>
</tr>
<tr>
<td><strong>Conditioned Stimulus (CS)</strong></td>
</tr>
<tr>
<td>- A stimulus that becomes able to produce a learned reflex response by being paired with the UCS</td>
</tr>
<tr>
<td>Bell</td>
</tr>
<tr>
<td><strong>Conditioned Response (CR)</strong></td>
</tr>
<tr>
<td>- A learned reflex response to a CS</td>
</tr>
<tr>
<td>Salivation (at the sound of the bell)</td>
</tr>
</tbody>
</table>
Theories of Learning

**Operant Conditioning**
- Learning based on the consequence of an action

**Reinforcer**
- A stimulus that increases the frequency of a behavior

**Punishment**
- A stimulus that decreases the frequency of a behavior
Examples of Learning

A 12 year old steals a soda from a grocery store. Outside the store his friends tell him that was “awesome.”

When a shock is given 5.6 seconds after a masked picture of a spider. People have increased sympathetic arousal after the picture is shown.

When you order take-out Chinese, the doorbell rings at your house/apt. and you begin to salivate.

Giving your dog a treat every time it sits when you say “sit.”
Karl Lashley’s Search For The Engram

Does learning depend on connections among the cortex?

**Method**
Train rats on mazes – then made cuts in various areas of the cortex

**Results**
No knife cut significantly impaired the rats performances
Major Findings

• Learning depends on many cortical areas – dif. areas may contribute in dif. ways
Types of Memory

**Short-Term Memory (STM)**
- 1 sec., to minutes, to hours
- Limited capacity

**Long-Term (LTM)**
- Days, weeks, to lifetime
- Capacity hard to measure

**Working Memory**
- Keeping info accessible for short periods of time
- Better conceptualization than STM

Should there be a distinction between STM and LTM?
Types of Memory

Explicit Memories

- Readily conscious, are capable of putting into words/images

1. Semantic Memory
   - Explicit memories for general facts or information

2. Episodic Memory
   - Memories for events or experiences in life

Implicit Memories

- Not consciously available, but info influences thoughts, judgments, behaviors

Procedural Memories

- Motor skills – how to tie shoe
- These types are implicit
Memory: Structures and Types

We now embark on an investigative search for structures that underlie the formation of different types of memory and learning...

...brought to you by the findings from Neuropsychology
The Case of H.M.

During the 11 years preceding his surgery, H.M. suffered an average of one generalized seizure each week and many partial seizures each day, despite massive doses of anticonvulsant medication. Electroencephalography suggested that H.M.’s convulsions arose from foci in the medial portions of both his left and right temporal lobes.

Because the removal of one medial temporal lobe had proven to be an effective treatment for patients with a unilateral temporal lobe focus, the decision was made to perform a bilateral medial temporal lobectomy – the removal of the medial portions of both temporal lobes, including most of the hippocampus, amygdala, and adjacent cortex.
In several respects, H.M.’s bilateral medial temporal lobectomy was an unqualified success. His generalized seizures were all but eliminated, and the incidence of partial seizures was reduced to one or two per day, even though the level of his anticonvulsant medication was substantially reduced. Furthermore, H.M. entered surgery a reasonably well-adjusted individual with normal perceptual and motor abilities and superior intelligence, and he left it in the same condition. Indeed, H.M.’s IQ increased from 104 to 118 as a result of his surgery, presumably because of the decline in the incidence of his seizures. Be that as it may, H.M. was the last patient to receive a bilateral medial temporal lobectomy – because of it’s devastating amnesic effects.
The Case of H.M.

Like his intellectual abilities, H.M.’s memory for events predating his surgery remained largely intact. Although he had a mild retrograde amnesia for those events that occurred in the 2 years before his surgery, his memory for more remote events (e.g., the events of his childhood) was reasonably normal. H.M.’s short-term memory also remained normal: For example his digit span test score was 6 – well within the normal range. In contrast, H.M. had an almost total inability to form new long-term memories: once he stopped thinking about a new experience, it was lost forever. In effect, H.M. became suspended in time on that day in 1953 when he regained his health, but lost his future.

-Pinel, p. 270
The Case of H.M.

**Retrograde Amnesia**
- Loss of memory for events that occurred before the brain damage

**Anterograde Amnesia**
- Inability to form memories for events that happened after brain damage

**Medial Temporal Lobe Amnesia**
1. Difficulty in forming explicit and episodic long-term memories
2. Retain the ability to form implicit and semantic long-term memories
The Case of H.M.

Graph showing the errors by H.M. on each set of the incomplete pictures test. Initial test and retest results are compared, with a 1-hour interval between the two tests.
Evidence

1. Tetris: Patients with amnesia train playing Tetris for 1 hour. The next day they have no recollection of playing it the previous day, but demonstrate better performance and faster learning that amnesia patients that have never played.
Evidence

2. Nice, Neural, and Stern Nurses

- Pleasant, to neutral, to refusal of all requests
- Shown picture of each nurse and asked, “Which one would you like to be friends with?”
  He never chose the stern nurse, even though she was an attractive woman who was smiling in the photo.
Hippocampus
• Crucial for explicit episodic, important for explicit semantic (but not implicit)
• Appears to store memories for an extended period of time

Hippocampus
• Important for spatial memory

Evidence
• London cab drivers have larger than average posterior hippocampus
Memory: Structures and Types

Basal Ganglia

- Plays a more prominent role in procedural memories (how to type, etc.)
- Diff. types of memories do utilize more than one system
Memory: Types and Structures

**Amygdala**
- Memory for emotional events
- Fear responses

**Cerebellum**
- Learned sensorimotor skills
- Conditioned responses
Karl Lashley’s Search For The Engram

Does the removal of specific cortical regions impair memory?

Method
Trained rat on mazes, then removed large portions of the cortex

Results?
Ablations and lesions impaired performance, but the amount of retardation depended more on the amount of brain damage than on its location. That is, no matter what location was removed, the rats were still able to perform moderately well.
Memory: Structures and Types

Cortex

1. Memories seem to be stored diffusely throughout the cortex

2. Memories become more resistant to destruction over time
Memory Consolidation

- Conversion of STM into LTM – stabilizes the memory
- When first learned, memories are changeable and unstable
- Consolidation corresponds to synthesis of proteins triggering growth of new synaptic connections
**Standard Consolidation Theory**

- Memories are temporarily stored in the hippocampus

- Then transferred to the cortex and other more stable cortical storage systems
Memory Consolidation

**Evidence**

A. Cases of retrograde amnesia from patients with brain damage (H.M.)

B. Electroconvulsive shock studies

C. Protein synthesis-inhibiting drug studies
Introduction

• Many studies have found that protein-inhibiting injections prevent the long-term retention of learning but not the acquisition of learning or short-term retention (e.g., 3 hrs)
• These studies suggest that there is a short term protein synthesis-independent stage and a long-term protein synthesis-dependent stage
**Introduction**

- The time of onset of the amnesia-producing effects of protein synthesis-inhibitors has varied from study to study.
- Studies have found that the effect can begin from minutes, to hours, to days of the learning.
Canal & Gold (2007)

**Method**

**Drug Infusions**

- 20 min. before training trials rats were infused with either:
  - *Anisomycin*
  - *Phosphate buffered saline (PBS)*

- To control for motivational or sensorimotor explanations, a different group of amygdala rats were injected immediately after training.
Canal & Gold (2007)

**Method**

**Inhibitory Avoidance Training**

- Rats placed in a box with a divider
- Divider is removed through the floor, when rats cross over, it is raised back up
- Rats would then receive a shock in the “shock compartment” half of the box
Method

Dependent Variable

• Rats placed in the box again at differing times
  * 30 min. (amygdala)
  * 4 hrs. (amygdala & hippocampus)
  * 48 hrs. (amygdala & hippocampus)

• Latency time of second trial – how much time is spend on the “non shock” side of the box (300 sec. maximal)
Canal & Gold (2007)

The chart shows the latency to cross (in seconds) for two groups, PBS and ANI, at two different time points after training: 4 hours and 48 hours. The bars indicate the median ± interquartile ranges. At 4 hours, both groups show similar latencies, while at 48 hours, the ANI group has a significantly lower latency compared to the PBS group, as indicated by the asterisk.
Canal & Gold (2007)
Discussion

• Learning task involved both the hippocampus and amygdala memory systems

• Hippocampus injections suggest that the rats learned and remembered the trials 4hrs after learning, but experienced memory impairment 48 hrs after training, relative to controls

• Amygdala injected rats exhibited memory impairment at all times, suggesting that they did not retain the learning
Conclusions

• Protein synthesis appears to play a critical role in the process of memory consolidation

• Different memory systems have different temporal properties for consolidation

• The amygdala system consolidates memories faster than the hippocampus system

• Different memory systems may somehow be interdependent on each other
Does sleep affect memory consolidation?
Research Question

How do periods of wakefulness and periods of sleep affect performance on a motor-sequence task?
Consolidating the Effects of Waking and Sleep on Motor-Sequence Learning

Brawn and Colleagues (2010)

**Introduction**

“The acquisition of a new skill initiates a process of memory formation wherein the newly formed memory trace is consolidated into a more stable and strengthened form. The consolidation of memories is widely believed to benefit from sleep”
Participants
• 85 right-handed U. of Chicago students

Motor Sequence Task
• 4-1-3-2-4
• Press with left hand
• Each key press produced an "*" on the screen to indicate the key press had been recorded without providing accuracy feedback.
Experimental Conditions

1. Training in A.M.
2. Training in P.M.

- Each condition included a training session and two posttest sessions that occurred 12 and 24 h after training.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Training</th>
<th>Retention 1</th>
<th>Posttest 1</th>
<th>Retention 2</th>
<th>Posttest 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>AM-massed</td>
<td>8:30–9:30</td>
<td>12 h</td>
<td>9:00–9:30</td>
<td>12 h</td>
<td>9:00–9:30</td>
</tr>
<tr>
<td>($n = 15$)</td>
<td>A.M.</td>
<td>wake</td>
<td>P.M.</td>
<td>sleep</td>
<td>A.M.</td>
</tr>
<tr>
<td>PM-massed</td>
<td>8:30–9:30</td>
<td>12 h</td>
<td>9:00–9:30</td>
<td>12 h</td>
<td>9:00–9:30</td>
</tr>
<tr>
<td>($n = 14$)</td>
<td>P.M.</td>
<td>sleep</td>
<td>A.M.</td>
<td>wake</td>
<td>P.M.</td>
</tr>
</tbody>
</table>
Consolidating the Effects of Waking and Sleep on Motor-Sequence Learning

<table>
<thead>
<tr>
<th>Schedule of Training and Testing Sessions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Training Session</strong></td>
</tr>
<tr>
<td>Warm-up</td>
</tr>
<tr>
<td>Pretest</td>
</tr>
<tr>
<td>Training</td>
</tr>
<tr>
<td>Posttrain Test</td>
</tr>
<tr>
<td>Rest Period</td>
</tr>
<tr>
<td>Warm up</td>
</tr>
<tr>
<td>Postrest test</td>
</tr>
<tr>
<td><strong>Posttest Session 1</strong></td>
</tr>
<tr>
<td>Warm-up</td>
</tr>
<tr>
<td>Posttest 1</td>
</tr>
<tr>
<td><strong>Posttest Session 2</strong></td>
</tr>
<tr>
<td>Warm-up</td>
</tr>
<tr>
<td>Posttest 2</td>
</tr>
</tbody>
</table>
Consolidating the Effects of Waking and Sleep on Motor-Sequence Learning
“We found that performance deteriorated significantly across the day and then recovered after a night of sleep when participants were trained in the morning. In contrast, performance remained stable across both a night of sleep and subsequent waking when training occurred in the evening. Therefore, sleep restored motor-sequence performance after it had deteriorated during a period of wakefulness before sleep, and sleep stabilized the motor memory against degradation during a subsequent day of wakefulness.”
Memory Reconsolidation

- When memories are recalled, they become malleable (capable of change)
- **New memories and reactivated memories** exist in similar states

*Video: How your memory works part 3, 4:00*
Long-Term Potentiation

• One or more axons bombard a dendrite with a rapid series of stimuli (100 messages/sec. for 4 sec.)
• Leaves the synapse “potentiated” (more responsive)
• = easier communication between the two neurons

Long-Term Potentiation

• A major mechanisms by which information is stored in the nervous system
Long-Term Potentiation

**Biochemical Mechanisms (Hippocampus)**
- Most LTP occurs at glutamate synapses
- AMPA receptors
- NMDA receptors
Biochemical Mechanisms (Hippocampus)

- Glutamate causes AMPA receptors open sodium (Na+) channels
- NMDA channels blocked by Magnesium (Mg++)
Long-Term Potentiation

**Biochemical Mechanisms (Hippocampus)**

- Major AMPA stimulation causes depolarization
- Removes the Mg++
- Allows Calcium (Ca++) to enter post synaptic dendrite
- Ca++ causes a protein to move to migrate to the synapse, sets in motion a series of events
Long-Term Potentiation

**Biochemical Mechanisms (Hippocampus)**

- More AMPA receptors are built and dendritic branching is increased
- Increase later responsiveness to incoming glutamate

**Biochemical Mechanisms (Hippocampus)**

- Drugs that block NMDA synapses prevent the establishment of LTP, but they do not interfere with the maintenance of LTP that was already established
Long-Term Potentiation

**Presynaptic Changes**

- Changes in presynaptic neuron can also cause LTP.
- Extensive stimulation of a postsynaptic cell causes the release of a retrograde transmitter that travels back to the presynaptic cell to cause the following changes:
  - Decrease in action potential threshold
  - Increase neurotransmitter release of glutamate
  - Expansion of the axons.
  - Transmitter release from additional sites.