States of Consciousness

• Refers to whether a person is awake, asleep, in a coma, etc.

• This state is determined by observing a person’s behavior or by measuring the electrical activity of the brain.
  – A recording of brain electrical activity is called an electroencephalogram.
EEG

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Fig. 8-1
What Makes the EEG?

• EEG patterns are largely due to graded potentials; in this case, summed postsynaptic potentials in the many hundreds of thousands of brain neurons that underlie the recording electrodes.

• The majority of the electrical signals recorded in the EEG originate in the pyramidal cells of the cortex.
Clinical Uses for EEG

- EEGs are used to detect wave patterns that are abnormal over diseased or damaged brain areas (e.g., because of tumors, blood clots, hemorrhage, regions of dead tissue, and high or low blood sugar).

- Shifts from a less synchronized pattern of electrical activity (low-amplitude EEG) to a highly synchronized pattern can be a preceed an epileptic seizure.
Epilepsy

- Epilepsy is a common neurological disease, occurring in mild, intermediate and severe forms, and is associated with abnormally synchronized discharges of cerebral neurons.

- Epilepsy is also associated with involuntary muscle contraction and a temporary loss of consciousness.

- In most cases the cause of epilepsy cannot be determined. Among the known triggers are traumatic brain injury, abnormal prenatal brain development, diseases that alter brain blood flow, heavy alcohol and illegal drug use, infectious diseases like meningitis and viral encephalitis, extreme stress, sleep deprivation, and exposure to environmental toxins such as lead or carbon monoxide.
EEG for Seizures

Onset of seizure

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Fig. 8-2
The Waking State

- The most prominent EEG wave pattern of an awake, relaxed adult whose eyes are closed, is the alpha rhythm.

- The alpha rhythm is recorded best over the parietal and occipital lobes and is associated with decreased levels of attention. When alpha rhythms are generated, subjects commonly report that they feel relaxed and happy.

- When people are attentive to an external stimulus or are thinking hard about something, the alpha rhythm is replaced by the beta rhythm.
The Waking State

(a) *Alpha rhythm* (relaxed with eyes closed)

(b) *Beta rhythm* (alert)
Sleep

- The EEG pattern changes profoundly in sleep. As a person becomes increasingly drowsy, their wave pattern transitions from a beta rhythm to one that is predominantly alpha.

- When sleep actually occurs, the EEG shifts toward slower-frequency, higher-amplitude wave patterns known as the theta rhythm (4–8 Hz) and the delta rhythm (slower than 4 Hz).

- There are two phases of sleep: NREM (non-rapid eye movement) and REM (rapid eye movement) sleep.
NREM Sleep

• During NREM sleep the EEG waves are referred to as slow-wave sleep. The initial phase of sleep—NREM sleep—is itself divided into four stages:

  In **stage 1** sleep theta waves begin to be interspersed among the alpha pattern.

  In **stage 2** sleep high frequency bursts called **sleep spindles** and very large-amplitude **K complexes** occasionally interrupt the theta rhythm. Delta waves first appear along with the theta rhythm.

  In **stage 3** sleep delta waves first appear along with the theta rhythm.

  In **stage 4** sleep the dominant pattern is a delta rhythm.

• Sleep begins with the progression from stage 1 to stage 4 of NREM sleep, which normally takes 30 to 45 min.
REM Sleep

• REM sleep is also called paradoxical sleep because the sleeper is difficult to arouse, despite having an EEG characteristic of the alert, awake state.

• When awakened during REM sleep, subjects generally report that they have been dreaming.

• If uninterrupted, sleep occurs in a cyclical fashion, tending to move from NREM stages 1, 2, and 3, to 4, then back up from 4 to 3, 2, and then an episode of REM sleep.
Why do we need sleep?

• Many lines of research suggest that it is a fundamental necessity of a complex nervous system.

• Studies of sleep deprivation in humans and other animals suggest that sleep is a homeostatic requirement, similar to the need for food and water. Deprivation of sleep impairs the immune system, causes cognitive and memory deficits, and ultimately leads to psychosis and even death.

• Much of the sleep research on humans has focused on the importance of sleep for learning and memory formation. EEG studies show that during sleep, the brain experiences reactivation of neural pathways stimulated during the prior awake state, and that subjects deprived of sleep show less effective memory retention.
Fig. 8-4

**Sleep**

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### Awake
- Alert, beta rhythm

![Awake EEG](image)

### NREM (slow-wave) sleep
- **Stage 1, theta rhythm**
  - Theta waves

![Stage 1 EEG](image)

- **Stage 2, sleep spindles and K complexes**
  - Sleep spindle
  - K complex

![Stage 2 EEG](image)

### Stages 3 and 4, delta rhythm

![Stage 3-4 EEG](image)

### REM (paradoxical) sleep
- REM pattern, similar to awake beta rhythm

![REM EEG](image)
Sleep

Fig. 8-5
Neural Substrates of State of Consciousness

Brainstem nuclei that are part of the reticular activating system

↑ Norepinephrine and serotonin

↓ Acetylcholine

Waking ↔ NREM sleep → REM sleep

↑ Activation of the thalamus and cortex

↑ Histamine

↓ GABA

Hypothalamus with circadian and homeostatic centers

↓ Norepinephrine and serotonin

↑ Acetylcholine

Fig. 8-6
Coma and Brain Death

• Coma is a severe decrease in mental function due to structural, physiological, or metabolic impairment.

• Brain death occurs when the brain no longer functions and has no possibility of functioning again.
### Table 8-2  Criteria for Brain Death

<table>
<thead>
<tr>
<th>I. The nature and duration of the coma must be known.</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Known structural damage to brain or irreversible systemic metabolic disease.</td>
</tr>
<tr>
<td>b. No chance of drug intoxication, especially from paralyzing or sedative drugs.</td>
</tr>
<tr>
<td>c. No severe electrolyte, acid–base, or endocrine disorder that could be reversible.</td>
</tr>
<tr>
<td>d. Patient is not suffering from hypothermia.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>II. Cerebral and brainstem function are absent.</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. No response to painful stimuli other than spinal cord reflexes.</td>
</tr>
<tr>
<td>b. Pupils unresponsive to light.</td>
</tr>
<tr>
<td>c. No eye movement in response to stimulation of the vestibular reflex or corneal touch.</td>
</tr>
<tr>
<td>d. Apnea (no spontaneous breathing) for 8 minutes when ventilator is removed, and carbon dioxide levels over 60 mmHg.</td>
</tr>
<tr>
<td>e. Systemic circulation may be intact.</td>
</tr>
<tr>
<td>f. Purely spinal reflexes may be retained.</td>
</tr>
<tr>
<td>g. Confirmatory neurological exam after 6 hours.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>III. Supplementary (optional) criteria:</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Flat EEG for 30 min (wave amplitudes less than 2 μV).</td>
</tr>
<tr>
<td>b. Responses absent in vital brainstem structures.</td>
</tr>
<tr>
<td>c. Greatly reduced cerebral circulation.</td>
</tr>
</tbody>
</table>

Conscious Experiences

• Things we are aware of, such as sensory stimuli, the passage of time, and past events.
Selective Attention

• Seeking out and focusing on stimuli that are momentarily important while avoiding the distraction of irrelevant stimuli.

• Some insights into neural mechanisms of selective attention are being gained from the study of individuals diagnosed with attention deficit hyperactivity disorder (ADHD).
ADHD

- This condition typically begins early in childhood and is the most common neurobehavioral problem in school-aged children.

- ADHD is characterized by abnormal difficulty in maintaining selective attention, and/or impulsiveness and hyperactivity.

- Functional imaging studies of the brains of children with ADHD have indicated dysfunction of brain regions in which catecholamine signaling is prominent, including the basal nuclei and prefrontal cortex.

- In support of this, the most effective medication used to treat ADHD is methylphenidate (Ritalin®), a drug that increases synaptic concentrations of dopamine and norepinephrine.
Neural Mechanisms of Conscious Experiences

• Conscious experience requires neural processes within the brain.

• Conscious experience resides not in a single anatomical cluster, but in a set of neurons that are temporarily functioning together.

• This grouping of neurons varies as our attentions shifts among visual or auditory stimuli, memories, emotions, etc.
Motivation and Emotion

• Motivations are the processes responsible for goal-directed behavior.

• Emotions result from the relationship between an individual and the environment.
Motivation

• Motivation can lead to hormonal, autonomic, and behavioral responses.

• Primary motivated behavior is behavior related directly to homeostasis—that is, the maintenance of a relatively stable internal environment, such as getting something to drink when you are thirsty.

• In many kinds of behavior, however, the relation between the behavior and the primary goal is indirect. For example, the selection of a particular flavor of soft drink has little if any apparent relation to homeostasis. The motivation in this case is secondary.
Motivation

• Much of human behavior fits into this latter category and is influenced by habit, learning, intellect, and emotions—factors that can be lumped together under the term “incentives.”

• The concepts of reward and punishment are inseparable from motivation. Rewards are things that organisms work for or things that make the behavior that leads to them occur more often—in other words, positive reinforcement. Punishments are the opposite.

• The neural system subserving reward and punishment is part of the reticular activating system.
Motivation: Chemical Mediators

• Dopamine is a major neurotransmitter in the pathway that mediates the brain reward systems and motivation.

• Amphetamines are an example of a drug that is abused because they increase the presynaptic release of dopamine (positive reinforcement).

• Conversely, drugs such as chlorpromazine, an antipsychotic drug that blocks dopamine receptors and lowers activity in the catecholamine pathways, are negatively reinforcing.
Motivation: the Mesolimbic-Dopamine Pathway
Emotions

• Emotional behavior can be studied more easily than the anatomical systems or inner emotions because it includes responses that can be measured externally (in terms of behavior).

• Certain anatomical structures in the brain are responsible for emotional responses; for example, the amygdala = fear.

• The cerebral cortex plays a major role in directing many of the motor responses during emotional behavior (e.g., to approach or avoid a situation).
Emotions

• Moreover, forebrain structures, including the cerebral cortex, account for the modulation, direction, understanding, or even inhibition of emotional behaviors.

• Although limbic areas of the brain seem to handle inner emotions, there is no single “emotional system.”

• The limbic areas have been stimulated in awake human beings undergoing neurosurgery. These patients reported vague feelings of fear or anxiety during periods of stimulation to certain areas. Stimulation of other areas induced pleasurable sensations, which the subjects found hard to define precisely.
Emotions: the Limbic System

Fig. 8-11
Altered States of Consciousness

• Distinct experiences commonly associated with mind-altering drugs or psychiatric illnesses.
Schizophrenia

• Schizophrenia is a disease in which information is not properly regulated in the brain.

• The amazingly diverse symptoms of schizophrenia include hallucinations, especially “hearing” voices, and delusions, such as the belief that one has been chosen for a special mission or is being persecuted by others.

• Schizophrenics become withdrawn, are emotionally unresponsive, and experience inappropriate moods. They may also experience abnormal motor behavior, which can include total immobilization (catatonia). The symptoms vary from person to person.
Schizophrenia

• The causes of schizophrenia remain unclear. Recent studies suggest that the disease reflects a developmental disorder in which neurons migrate or mature abnormally during brain formation.

• One explanation is that some dopamine pathways are overactive. This hypothesis is supported by the fact that amphetamine-like drugs, which enhance dopamine signaling, make the symptoms worse, and by the fact that the most therapeutically beneficial drugs used in treating schizophrenia block dopamine receptors.

• Schizophrenia affects approximately one in every 100 people and typically appears in the late teens or early twenties, just as brain development nears completion.
Mood Disorders: Depression and Bipolar Disorder

- Depression is characterized by pervasive sadness, loss of energy, irritability, disturbed sleep, and thoughts of death or suicide.

- Bipolar disorder is characterized by episodes of depression and mania.
  - Mania is characterized by an abnormally and persistently elevated mood.
Depressions

- In the *depressive disorders (depression)*, some of the prominent features are a pervasive feeling of emptiness or sadness; a loss of energy, interest, or pleasure; anxiety; irritability; a marked increase or decrease in appetite; disturbed sleep; and thoughts of death or suicide.

- Depression can occur on its own, independent of any other illness, or it can arise secondary to other medical disorders. It is associated with decreased neuronal activity and metabolism in the anterior part of the limbic system and nearby prefrontal cortex.

- Although the major biogenic amine neurotransmitters (norepinephrine, dopamine, and serotonin) and acetylcholine have all been implicated, the causes of the mood disorders are unknown.
Depressions

- Current treatment of the mood disorders emphasizes drugs and psychotherapy. The classical anti-depressant drugs are of three types.

1. **The tricyclic antidepressant drugs** such as Elavil®, Norpramin®, and Sinequan® interfere with serotonin and/or norepinephrine reuptake by presynaptic endings.

2. **The monoamine oxidase inhibitors** interfere with the enzyme responsible for the breakdown of these same two neurotransmitters.

3. **The serotonin-specific reuptake inhibitors (SSRIs)** are the most widely used antidepressant drugs and include Prozac®, Paxil®, and Zoloft®. As their name—SSRI—suggests, these drugs selectively inhibit serotonin reuptake by presynaptic terminals.
Depressions

• Alternative treatments used when drug therapy and psychotherapy are not effective include electrical and electromagnetic stimulation of the brain.

• *Electroconvulsive therapy (ECT).* A series of ECT treatments alters neurotransmitter function by causing changes in the sensitivity of certain serotonin and adrenergic postsynaptic receptors.

• *Repetitive transcranial magnetic stimulation (rTMS).* Neural activity is transiently disordered or sometimes silenced in that brain region.
SADD

• Another nondrug therapy used for the type of annual depression known as *seasonal affective depressive disorder (SADD)* is *phototherapy*.

• *Phototherapy* exposes the patient to bright light for several hours per day during the winter months.

• Although light is thought to relieve depression by suppressing melatonin secretion from the pineal gland, as yet there is little evidence to support this claim.
Bipolar Disorder

• The term *bipolar disorder* describes swings between mania and depression. Episodes of *mania* are characterized by an abnormally and persistently elated mood, sometimes with euphoria, racing thoughts, excessive energy, overconfidence, and irritability.

• A major drug used in treating patients with bipolar disorder is the chemical element lithium, sometimes given in combination with anticonvulsant drugs.

• It is highly specific, normalizing both the manic and depressing moods and slowing down thinking and motor behavior without causing sedation.
Psychoactive Substances

- Psychoactive substances are also used as “recreational” drugs in a deliberate attempt to elevate mood and produce unusual states of consciousness ranging from meditative states to hallucinations.

- Virtually all the psychoactive substances exert their actions either directly or indirectly by altering neurotransmitter-receptor interactions in the biogenic amine (particularly dopamine) pathways.

- For example, the primary effect of cocaine comes from its ability to block the reuptake of dopamine into the presynaptic axon terminal.
Dependence

• Substance dependence has two facets that may occur either together or independently:

  1. A *psychological dependence* is experienced as a craving for a substance and an inability to stop using the substance at will.

  2. A *physical dependence* that requires one to take the substance to avoid *withdrawal*, which is the spectrum of unpleasant physiological symptoms that occurs with cessation of substance use.

• Several neuronal systems are involved in substance dependence, but most psychoactive substances act on the mesolimbic dopamine pathway.

• Although the major neurotransmitter implicated in substance dependence is dopamine, other neurotransmitters, including GABA, enkephalin, serotonin, and glutamate, are also involved.
Tolerance

• *Tolerance* to a substance occurs when increasing doses of the substance are required to achieve effects that initially occurred in response to a smaller dose.

• Tolerance can develop to another substance as a result of taking the initial substance, a phenomenon called *cross-tolerance*. Cross-tolerance may develop if the physiological actions of the two substances are similar.

• Tolerance may develop because the presence of the substance stimulates the synthesis of the enzymes that degrade it.

• Tolerance can develop as a result of changes in the number and/or sensitivity of receptors that respond to the substance, the amount or activity of enzymes involved in neurotransmitter synthesis, the reuptake transport molecules, or the signal transduction pathways in the postsynaptic cell.
Psychoactive Substances, Dependence, & Tolerance

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Fig. 8-13

Serotonin (5-hydroxytryptamine)

Psilocybin (some mushroom species)  Dimethyltryptamine (DMT)

Dopamine

Amphetamine

Methamphetamine (speed)

Mescaline (peyote)

Dimethoxymethylamphetamine (DOM-STR)
Learning and Memory

• Learning is the acquisition and storage of information.

• Memory is the relatively permanent storage form of the learned information.
Memory

• Memory can be viewed in two broad categories, called declarative and procedural memory.

• Declarative memory (sometimes also referred to as “explicit” memory) is the retention and recall of conscious experiences that can therefore be put into words (declared) i.e., facts and figures.

• One example is the memory of having perceived an object or event and, therefore, recognizing it as familiar and maybe even knowing the specific time and place when the memory originated.

• The hippocampus, amygdala, and other parts of the limbic system are required for the formation of declarative memories.
Memory

• The second broad category of memory, procedural memory, can be defined as the memory of how to do things.

• This is the memory for skilled behaviors independent of any conscious understanding, as for example, riding a bicycle.

• The category of procedural memory also includes learned emotional responses, such as fear of spiders, and the classic example of Pavlov’s dogs.

• The primary areas of the brain involved in procedural memory are regions of sensorimotor cortex, the basal nuclei, and the cerebellum.
Short-term Memory

• Another way to classify memory is in terms of duration—does it last for a long or only a short time?

• Working memory, also known as short-term memory, registers and retains incoming information for a short time—a matter of seconds to minutes—after its input.

• Short-term memories may be converted into long-term memories, which may be stored for days to years and recalled at a later time. The process by which short-term memories become long-term memories is called consolidation.
The Neural Basis of Learning and Memory

- The neural mechanism and parts of the brain involved vary for different types of memory.

- Short-term encoding and long-term memory storage occur in different brain areas for both declarative and procedural memories.

- Working memory is susceptible to external interference, such as an attempt to learn conflicting information. On the other hand, long-term memory can survive deep anesthesia, trauma, or electroconvulsive shock, all of which disrupt the normal patterns of neural conduction in the brain.
The Neural Basis of Learning and Memory

• Based on current data it is assumed that working memory requires ongoing graded or action potentials.

• Working memory is interrupted when a person becomes unconscious from a blow on the head, and memories are abolished for all that happened for a variable period of time before the blow, a condition called retrograde amnesia. (Amnesia is defined as the loss of memory.)

• Another type of amnesia is referred to as anterograde amnesia. It results from damage to the limbic system and associated structures, including the hippocampus, thalamus, and hypothalamus.
The Neural Basis of Learning and Memory

• Patients with anterograde amnesia lose their ability to consolidate short-term declarative memories into long-term memories. While they can remember stored information and events that occurred before their brain injury, they can only retain anything that happens from that point forward in time as long as it exists in working memory.

• This type of amnesia is sometimes intentionally induced pharmacologically during medical procedures for which patients are required to remain conscious, such as colonoscopy. The most common drugs used to produce this “conscious sedation” are of the type that stimulates GABA receptors.
The Neural Basis of Learning and Memory

- The problem of exactly how memories are stored in the brain is still unsolved. One model for memory is long-term potentiation (LTP), in which certain synapses undergo a long-lasting increase in their effectiveness when they are heavily used.

- It is generally accepted that long-term memory formation involves processes that alter gene expression and result in the synthesis of new proteins. This is achieved by a cascade of second messengers and transcription factors that activate cellular gene expression resulting in the production of new protein synthesis (translation).

- These new proteins may be involved in the increased number of synapses that have been demonstrated after long-term memory formation. They may also be involved in structural changes in individual synapses (e.g., by an increase in the number of receptors on the postsynaptic membrane). This ability of neural tissue to change because of activation is known as plasticity.
The Neural Basis of Learning and Memory

• Certain types of learning depend not only on factors such as attention, motivation, and various neurotransmitters, but also on certain hormones.

• For example, the hormones epinephrine, ACTH, and vasopressin affect the retention of learned experiences. These hormones are normally released in stressful or even mildly stimulating experiences, suggesting that the hormonal consequences of our experiences affect our memories of them.

• Two of the opioid peptides, enkephalin and endorphin, interfere with learning and memory, particularly when the lesson involves a painful stimulus. They may inhibit learning simply because they decrease the emotional (fear, anxiety) component of the painful experience associated with the learning situation, thereby decreasing the motivation necessary for learning to occur.
# The Neural Basis of Learning & Memory

## Declarative memory

<table>
<thead>
<tr>
<th>Short-term</th>
<th>Long-term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hippocampus &amp; other</td>
<td>Many areas of association cortex</td>
</tr>
<tr>
<td>limbic system</td>
<td></td>
</tr>
<tr>
<td>structures</td>
<td></td>
</tr>
</tbody>
</table>

## Procedural memory

<table>
<thead>
<tr>
<th>Short-term</th>
<th>Long-term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Widely distributed</td>
<td>Basal nuclei</td>
</tr>
<tr>
<td></td>
<td>Cerebellum</td>
</tr>
<tr>
<td></td>
<td>Sensorimotor cortex</td>
</tr>
</tbody>
</table>
Cerebral Dominance

• The two cerebral hemispheres appear to be nearly symmetrical, but each has anatomical, chemical, and functional specializations.

• In 90 percent of the population, the left hemisphere is specialized to produce language—the conceptualization of what one wants to say or write, the neural control of the act of speaking or writing, and recent verbal memory.

• Language is a complex code that includes the acts of listening, seeing, reading, and speaking. The major centers for language function are in the left hemisphere in the temporal, parietal, and frontal cortex next to the Sylvian fissure, which separates the temporal lobe from the frontal and parietal lobes.
Cerebral Dominance

• Males and females typically use different brain areas for language processing, with females tending to involve areas of both hemispheres for some language tasks, while males generally show activity only on the left side.

• The cerebellum is also important in speaking and writing, because those tasks involve coordinated muscle contractions.

• Much of our knowledge about how language is produced has been obtained from patients who have suffered brain damage and, as a result, have one or more defects in language, known as aphasias.

• For example, in most people, damage to the left cerebral hemisphere, but not to the right, interferes with the capacity for language manipulation, and damage to different areas of the left cerebral hemisphere affects language use differently.
Cerebral Dominance

• Damage to the temporal region known as Wernicke’s area generally results in aphasias that are more closely related to *comprehension*—the individuals have difficulty understanding spoken or written language even though their hearing and vision are unimpaired. Although they may have fluent speech, they scramble words so that their sentences make no sense, often adding unnecessary words, or even creating made-up words.

• In contrast, damage to Broca’s area, the language area in the frontal cortex responsible for the articulation of speech, can cause *expressive* aphasias. Individuals have difficulty carrying out the coordinated respiratory and oral movements necessary for language, even though they can move their lips and tongues. They understand spoken language and know what they want to say, but have trouble forming words and sentences.
Cerebral Dominance

• The potential for the development of language-specific mechanisms in the left hemisphere is present at birth, but the assignment of language functions to specific brain areas is fairly flexible in the early years of life.

• Differences between the two hemispheres are usually masked by the integration that occurs via the corpus callosum and other pathways that connect the two sides of the brain.

• Although language skills emerge spontaneously in children in all societies, there is a critical period during childhood when exposure to language is necessary for these skills to develop, just as the ability to see depends upon effective visual input early in life.
Cerebral Dominance

- Memories are handled differently in the two hemispheres.

- Verbal memories are more apt to be associated with the left hemisphere, and nonverbal memories (e.g., visual patterns or nonverbal memories that convey emotions) with the right.

- Even the emotional responses of the two hemispheres seem to be different; for example, the left hemisphere has more ability to understand the emotional states of oneself or others.

- When electroconvulsive therapy is administered in the treatment of depression, however, better effects are often obtained when the electrodes are placed over the right hemisphere. The two sides of the brain also differ in their sensitivity to psychoactive drugs.
Cerebral Dominance and Language

Fig. 8-15

Broca’s area

Parietal lobe

Frontal lobe

Occipital lobe

Sylvian fissure

Temporal lobe

Wernicke’s area

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