2 Memory & learning
Functions of specific cortical areas:
Associated areas:

A. Parieto-occipito-temporal association area
The parieto-occipito-temporal association area provides a high level of interpretative meaning for signals from the ①somatosensory cortex anteriorly, ②the visual cortex posteriorly, and ③the auditory cortex laterally.

The parieto-occipito-temporal association area has its own functional sub-areas

(1) Analysis of the Spatial Coordinates of the Body.
Spatial coordinates of the body and surrounding area provides continuous analysis of a. the spatial coordinates of all parts of the body, b. as well as of the surroundings of the body.

Spatial coordinates of the body and surrounding area receives visual sensory information from the posterior occipital cortex and simultaneous somatosensory information from the anterior parietal cortex.

Spatial coordinates of the body and surrounding area computes the coordinates of the visual, auditory, and body surroundings.

(2) Wernicke’s Area
Wernicke’s area lies behind the primary auditory cortex
Wernicke’s area is important for language comprehension فهم through hearing
Wernicke’s area is named as language comprehension intelligence in the dominant hemisphere
Wernicke’s area functions:

1. Wernicke’s area **process sensory information** from somatic sensory, visual, and auditory cortices
2. Wernicke’s area is **speech understanding area**
3. Wernicke’s area is **interpretation and determination** of the words

(3) **Angular gyrus area** (Dejerine's area)
The angular gyrus sub-serves perception of written language.

The angular gyrus appears to process information from words that are read in such a way that they can be converted into the auditory forms of the words in Wernicke’s area.

المعلومات التي ترسل منها الى (Wernicke’s area) هي ادراك الكلمات المقرؤة ولكن على شكل مخارج صوتية

In its absence, a person can still have excellent language comprehension through hearing but not through reading.

Damage to angular gyrus area the person may be able to see words and even know that they are words but not be able to interpret their meanings. This condition is called **dyslexia**, or word blindness.

(4) **Area for Naming Objects.**
The objects names are learned mainly through auditory input physical natures of the objects are learned mainly through visual input.

In turn, the names are essential for both auditory and visual language comprehension (functions performed in Wernicke’s area which is located near auditory and visual area).
(B) Prefrontal association area

1. The prefrontal association area functions in close association with the motor cortex to
   a. plan complex patterns
   b. sequences of motor movements.

2. The prefrontal association area is essential to carrying out “thought” processes.
   People without prefrontal cortices are easily distracted from their central theme of thought,
   Decreased aggressiveness and inappropriate social responses probably result from loss of the ventral parts
   of the frontal lobes on the underside of the brain.

(1) Broca’s area
Broca’s area is located partly in the posterior lateral prefrontal cortex and partly in the premotor area.
Broca’s area functions:

1. Broca’s area responsible for speech synthesis (control of word formation): is responsible for planning and motor patterns are initiated and executed for expressing individual words or even short phrases.

التخطيط و التنفيذ لتحريك الحركات العضلية لأخراج الكلمات أو الجمل

2. Broca’s area works in close association with the Wernicke language comprehension center

3. Broca’s area regulate the function of muscles of lips, tongue, and larynx

An especially interesting discovery is the following:

When a person has already learned one language and then learns a new language, the area in the brain where the new language is stored is slightly removed from the storage area for the first language. If both languages are learned simultaneously، they are stored together in the same area of the brain.
Limbic association area is concerned primarily with behavior, emotions, and motivation.

The limbic system

a. provides most of the emotional drives for activating other areas of the brain.

b. provides motivational drive for the process of learning itself.

1. Area for recognition of faces

Area for recognition of faces damage causes abnormality called Prosop-agnosia or face blindness (is the inability to recognize faces).

a. The occipital portion of this facial recognition area is contiguous with the visual cortex.

b. The temporal portion is closely associated with the limbic system that has to do with emotions, brain activation, and control of one’s behavioral response to the environment.
Complementary specialization of the hemispheres versus "cerebral dominance"

Dominant hemisphere (the categorical hemisphere) sequential-analytical process:

- Left hemisphere in right handed people (9 of 10 persons)
- Wernike's and Broca's area is larger than the other hemisphere
- Is related to language, that is,
  1. understanding the spoken and printed word
  2. expressing ideas in speech and writing (i.e. written and spoken language)
- Mathematical and scientific skills
- Analytical Reasoning

Non-dominant hemisphere (the representational hemisphere) visuo-spatial relation

- Right hemisphere in right handed people
- For the identification of objects by their form (i.e. three dimensional awareness)
- The significance of "body language: muscle awareness"
- Plays a primary role in the recognition of people faces and people voices
- Art and music awareness (understanding and interpreting)
- Insight (nonverbal visual experiences)
- Imagination
- Spatial relations between the person and their surroundings
- Probably many somatic experiences related to use of the limbs and hands
Language:
to understand spoken and printed words and to express ideas in speech and writing

Writing: language expressed by visual symbols

Reading: visual symbols or writing words expressed verbally

Speech: expression of thought by producing of articulated sound, bearing a definite meaning.

Speech involved integration of 3 cerebral cortex areas: Wernike's area, Broca area and Motor area: activation of peripheral speech apparatus (the laryngeal, respiratory, and mouth muscles)

Speech major recognized types:
Spoken speech: understand the spoken words and expressing ideas in speech

Written speech: understand the written words and expressing ideas in writing
Speech in response to hearing:

Primary auditory area (area 41, 42): receive auditory nerve impulse

Auditory associated area (area 21, 20) concerned with interpretation and integration of auditory impulses

Wernike's area: interpretation and comprehension of speech ideas

Arcuate fasciculus

Broca area

Primary motor area

Brain stem

Muscles of speech
Speech in response to reading:

- **Primary Visual cortex (area 17):** perception of written words
- **Visual association area (area 18 and 19):** recognition and identification of written words
- **Dejerine area:** generation of thought/ideas in written speech
- **The angular gyrus area** (needed to make meaning out of the visually perceived words)
- **Wernike's area:** interpretation and comprehension of written words

**Pathway in the process of reading**

- Exner's area
- Primary Motor cortex
- Dejerine area
- Brain stem
- Broca area
- Wernicke's area
- Visual Association cortex
- Muscles of speech

**Speaking a written word**

- Motor cortex
- Primary visual cortex
- Angular gyrus
- Wernicke's area
- Broca's area
- Muscles of larynx
- Brainstem
Auditory receptive aphasia and visual receptive aphasia or, more commonly, word deafness and word blindness (also called **dyslexia**).

Dyslexia, which is a broad term applied to **impaired ability to read**, is characterized by difficulties with

1. learning how to decode at the word level
2. to spell, and
3. to read accurately
4. to read fluently

Dyslexia is frequently due to an **inherited abnormality that affects 5% of the population**.

Many individuals with dyslexic symptoms also have

a. problems with **short-term memory skills** and
b. problems **processing** spoken language.

Dyslexia precise cause is unknown (**maybe destruction of portions of the auditory or visual association areas of the cortex**)
Aphasias caused by lesions in the categorical hemisphere

Motor aphasia or (non-fluent aphasia)

Motor aphasia results from damage to Broca’s speech area

Motor aphasia a person is capable of deciding what he or she wants to say

المريض قادر على تحديد الكلمات التي يريد النطق بها

Motor aphasia cannot make the vocal system emit words instead of noises

المريض غير قادر على تطوع والسيطرة الناضم الصوتي مما يؤدي إلى النطق بكلمات غير مفهومة

Speech is

① slow, الكلام بطيء

② words are hard to come صعوبة اخراج الكلمات

③ limited to two or three words with which to express the whole range of meaning and emotion. اختصار الإجابة بأقل عدد من الكلمات
Wernicke’s aphasia or (fluent aphasia) results from damage to Wernicke’s area in the dominant hemisphere. Wernicke’s aphasia a person is capable of understanding either the spoken word or the written word, but are unable to interpret the thought that is expressed.

Wernicke’s aphasia

1. Speech itself is normal and sometimes the patients talk excessively.
2. What they say is full of jargon and neologisms that make little sense.
3. The patient also fails to comprehend the meaning of spoken or written words, so other aspects of the use of language are compromised.

The person normally loses almost all intellectual functions associated with language or verbal symbolism, such as:

1. The ability to read,
2. The ability to perform mathematical operations,
3. The ability to think through logical problems.
Anomic aphasia: When a lesion damages the angular gyrus in the categorical hemisphere without affecting Wernicke’s or Broca’s areas.

There is no difficulty with speech or the understanding of auditory information; instead there is trouble understanding written language or pictures, because visual information is not processed and transmitted to Wernicke’s area.

Learning

Learning is acquisition of the information that makes ability to alter behavior on the basis of experience possible.

Learning is a process that will modify a subsequent behavior.

Memory

Memory is the ability to store, retain and recall personal experiences, information, and various skills and habits.

Memory is an active system that store, organizes, alters and recovers (retrieves) information

Memory process Just like computer
Encoding: Converting information into a useable form
Storage: Holding this information in memory
Retrieval: Taking memory out of storage
Neuronal basis of memory:
Memories are stored in the brain by changing the basic sensitivity of synaptic transmission between neurons as a result of previous neural activity.
We cannot store all memories
Some memories are erased; or ignore.
This capability results from inhibition of the synaptic pathways for this type of information; the resulting effect is called habituation, which is a type of negative memory.

Some memories are enhanced or stored.
This capability results from facilitation of the synaptic pathways, and the process is called memory sensitization, which is a type of positive memory.
The new or facilitated pathways are called memory traces.
Multi-store model memory:

Is a model of memory proposed that human memory has three separate components: sensory, short-term (or working), and long-term.

Typically from 10 to 15 seconds, or sometimes up to a minute.

Most of information is forgotten someway along the way.

Information that you pay attention to and process will move to next store of memory.
Sensory memory

Sensory memory is the **shortest-term** element of memory.

Sensory memory is the ability to **retain impressions** of sensory information after the original stimuli have ended.

Sensory memory acts as a kind of **buffer** for stimuli received through the **five senses** of sight, hearing, smell, taste and touch, which are retained accurately, but very briefly. For example, the ability to look at something and remember what it looked like with just a second of observation is an example of sensory memory.

Sensory memory unlike other types of memory, **cannot be prolonged via rehearsal**.

Sensory memory is an ultra-short-term memory and **decays** or degrades very quickly, typically in the region of 200 - 500 milliseconds (1/5 - 1/2 second) after the perception of an item.
Sensory memory often considered part of the process of perception.

Sensory memory represents an essential step for storing information in short-term memory.

Sensory memory is usually unlimited capacity.

Sensory memory is usually considered to be totally outside of conscious control. The brain is designed to only process information that will be useful at a later date, and to allow the rest to pass by unnoted.

The stimuli detected by our senses can be either:

a. deliberately ignored, in which case they disappear almost instantaneously, or

b. Perceived محسوس و ملحوض, in which case they enter our sensory memory.

As information is perceived, it is therefore stored in sensory memory automatically and unbidden.
2. Short-term memory

Short-term memory can be thought of as

A. the ability to **remember**

B. **process** معالجة **information** at the same time.

Short-term memory is **very fragile and information can be lost with distraction or passage of time.**

Short term memory has three key aspects:

1. **Limited capacity** (only about 7 items can be stored at a time)

2. **Limited duration** short-term memory, which lasts seconds to hours, during which processing in the hippocampus and elsewhere lays down long-term changes in synaptic strength.

3. **Encoding** (primarily acoustic صوتي, even translating visual information into sounds).

4. **Conscious** process of information
However, this information will quickly disappear forever unless we make a conscious effort to retain it.

Short term memory is a necessary step toward the next stage of retention, long-term memory.

The transfer of information to long-term memory for more permanent storage can be facilitated or improved via:

1. The process of attention (i.e. selectivity of information) and a first step of learning is attention.

2. Mental repetition of the information or, even more effectively, by giving it meaning and associating it with other previously acquired knowledge.

3. Motivation is also a consideration, in that information relating to a subject of strong interest to a person, is more likely to be retained in long-term memory.
Working memory

Some consider working memory as a form of short-term memory that keeps information available, usually for very short periods, while the individual plans action based on it. Other considered working memory is short-term memory. Instead of all information going into one single store, there are different systems for different types of information. Evidence suggests that working memory uses two different systems for dealing with visual and verbal information. A visual processing task and a verbal processing task can be performed at the same time. It is more difficult to perform two visual tasks at the same time because they interfere with each other and performance is reduced. The same applies to performing two verbal tasks at the same time. This supports the view that the phonological loop and the sketchpad are separate systems within working memory.
1. Central Executive (the boss of working memory)

The central executive is responsible for monitoring and coordinating the operation of the slave systems (i.e. visuo-spatial sketch pad and phonological loop).

The central executive relates the slave systems to long term memory (LTM).

The central executive decides which information is attended to (memory pays attention to) and which parts of the working memory to send that information to be dealt with.

Central Executive deals with cognitive tasks such as mental arithmetic and problem solving.

Working Memory Model – Baddeley and Hitch
Visuo-Spatial Sketchpad stores and processes information in:

1. **Visual form** (what things look like or counting the windows on a house, or imagining images or backtracking to pick up an item you skipped while browsing in a new store)
2. **Spatial form** (helping us keep track of where we are in relation to other objects as we move through our environment, or judging distances), so visuo-Spatial Sketchpad is used for navigation.
3. Displays and manipulates visual and spatial information held in long-term memory.

Try answering this question: How many windows are there in the front of your house?

You probably find yourself picturing the front of your house and counting the windows.

An image has been retrieved from LTM and pictured on the sketch pad.
3. Phonological Loop

Phonological Loop is a part of working memory that deals with spoken and written material. Phonological Loop stores auditory information by silently rehearsing sounds or words in a continuous loop: the articulatory process (for example the repetition of a telephone number over and over again). Then, a short list of data is easier to remember.

Phonological Loop consists of two parts:

a. Phonological Store (inner ear)

The phonological store (linked to speech perception) acts as an inner ear and holds information in speech-based form (i.e. spoken words) for 1-2 seconds. Spoken words enter the store directly.

b. Articulatory control process (inner voice)

Linked to speech production. Used to rehearse and store verbal information from the phonological store.

Written words must first be converted into an articulatory (spoken) code before they can enter the phonological store, so articulatory control process converts written material into an articulatory code and transfers it to the phonological store.
(3) Intermediate long-term memories,
It last for days to weeks but then fade away (Not all agrees that here is intermediate long term memory)

Molecular mechanism of intermediate memory
a. Mechanism for Habituation.
   Closure of calcium channels through the terminal membrane
       decrease release of neurotransmitter
b. Mechanism for Facilitation.
   In the case of facilitation, at least part of the molecular mechanism is believed to be the following:
1. Stimulation of the facilitator presynaptic terminal

2. Serotonin release at the facilitator synapse on the surface of the sensory terminal

3. Serotonin acts on serotonin receptors

4. Activate the enzyme adenyl cyclase inside the membrane

4. Formation of cyclic adenosine monophosphate (cAMP)

   - Activates a protein kinase

   - Phosphorylation of a protein that is part of the potassium channels

   - Blocks the channels for potassium conductance (can last for minutes up to several weeks)

   - Prolonged action potential in the synaptic terminal because flow of potassium ions out of the terminal is necessary for rapid recovery from the action potential

   - Prolonged activation of the calcium channels

   - Increased transmitter release by the synapse, thereby markedly facilitating synaptic transmission to the subsequent neuron.
Long-term memory, Place where data is stored for a long time once stored, can be recalled up to years or even a lifetime late

Long term memory characterizes by:

1. Unlimited capacity,
2. Semantically encoded
3. Storage presumed permanent,
4. Information highly organized
5. Robust

Storage: Holding this information in memory
Retrieval: Taking memory out of storage
Encoding: Converting information into a useable form

Encoding types:

1. Semantic encoding: encoding of meaning like meaning of word
2. Acoustic encoding: encoding of sound like sound of word
3. Visual encoding: encoding of picture like picture of word
4. Tactile encoding: is the encoding of how something feels, normally through the sense of touch

During short-term memory, the memory traces are subject to disruption by trauma and various drugs, whereas long-term memory traces are remarkably resistant to disruption.
Long-term memory types:

A. Explicit or declarative memory: is associated with consciousness—or Explicit memory at least awareness—and is dependent on the hippocampus and other parts of the medial temporal lobes of the brain for its retention.

Explicit memory is divided into:

1. Episodic memory is based on specific events (experienced events), or "episodes" that are part of your personal history. Some examples: The name of your pet bird growing up, your sister’s wedding, the name of your fifth-grade teacher.

2. Semantic memory for facts (knowledge and concepts) eg, words, rules, and language. It is the ability to recall facts and concepts, often referred to as common knowledge. Some examples: Understanding the difference between a dog and a cat, being able to associate letters with their sounds, recalling how to use a phone.

Explicit memories initially required for activities such as riding a bicycle can become implicit once the task is thoroughly learned.
B. Implicit or non-declarative memory

Implicit memory does not involve awareness (unconscious or automatic memory).

Implicit memory uses past experiences to remember things without thinking about them.

Implicit memory retention does not usually involve processing in the hippocampus.

One common example of the differences between implicit and explicit memory is that implicit memory allows you to type on a keyboard without looking at the keys, while you need explicit memory to remember that the A-S-D-F keys are on the left and J-K-L-; keys are on the right in the "home" row.
Implicit memory is subdivided into four types.

1. **Procedural memory** (الذاكرة الإجرائية): 

Procedural memory includes (skill and action: how to do things), which, once acquired, become unconscious and automatic so enables us to carry out ordinary motor actions essentially on autopilot.

Procedural memory is typically acquired through repetition and practice, sometimes described as muscle memory or body memory.

Some examples of procedural memory are: Driving a car, walking, riding a bicycle, and swimming. These are typically tasks that you can go months or even years without performing and pick them up again quickly.

Usually, anterograde amnesia impacts declarative memory only and has no effect on procedural memory. An amnesiac can remember how to talk on the phone, but can't recall with whom they spoke earlier that day.

Anterograde amnesia (عدم المدية لاضافة ذاكية جديدة) and retrograde amnesia (عدم المدية لاسترجاع ذاكية قديمة) are two types of amnesia that affect memory differently.
Priming: إعداد (شخص ما) لحالة أو مهمة، عادة عن طريق تزويدهم بالمعلومات ذات الصلة.

Priming is facilitation of recognition of words or objects by prior exposure to them. Priming involves using pictures, words or other stimuli to help someone recognize another word or phrase in the future. Examples include using green to remember grass and red to remember apple, improved recall of a word when presented with the first few letters of it.

Priming is an effect in which exposure to one stimuli influence the response to another stimulus due to prior experience. Example if some lets you to give a name of animal begins with "D" everybody says" Dog" not" Deer" or "Dolphin" because we are seen and familiar with dog more ; people elsewhere says "Deer"

Non-associative learning, the organism learns about a single stimulus.

A signal stimulus is given repeatedly and nervous system learn about the characteristics of the stimulus:

a. habituation: decrease response to stimuli e.g. exercise to treat dizziness in patients
b. Sensitization: increase response to stimuli e.g. training to enhance awareness of loss of balance
Associative learning (classical conditional learning), the organism learns about the relation of one stimulus to another.

a. skeletal muscular: Dog + meat + ringing bell
   Dog + meat + ringing bell $\Rightarrow$ Dog + ringing bell (conditional learning); Dog + meat (unconditional learning)

b. emotional response: snake + fear

Short term memory and Long-term potentiation

Short-term memory is generally believed to result from only chemical changes.

Long-term potentiation

Long-term potential is the gradual strengthening of the connections among neurons from repetitive stimulation.

Long-term potentiation is a type of synaptic learning, in that synapses that are first stimulated at high frequency will subsequently exhibit increased excitability.

Long-term potentiation is a long lasting enhancement in signal transmission that result from stimulating them synchronously.
Short and weak stimuli:
- Glutamate release from pre-synapse
- Attach to AMPA (alpha-Amino-3-hydroxy-5-Methyl-4-isoaxoleproionic-Acid) receptor that allow sodium to enter
- Depolarization for short duration

Strong and prolonged stimuli:
First: open of AMPA receptor (as above)
- Glutamate release from pre-synapse
- Attach to NMDA receptor (N-methyl-D-aspartate)
- Removal of Magnesium that block the receptor
- Allow Calcium entry
- Calcium attach to calmodulin
- Stimulate enzymes as Calmodulin-dependent-protein kinase II, Phosphokinase, tyrosine kinase

This will cause multiple effect that will prolong action potential
a. retrograde release of nitric oxide so more glutamate release
b. express more AMPA receptor of Post-synapsis so more sodium enter

Both of those effect will prolong and strengthen the signal (Long duration potential)
Number of neurons and their connectivity often change significantly during learning. During the first few weeks, months, and perhaps even year or so of life, many parts of the brain produce a great excess of neurons, and the neurons send out numerous axon branches to make connections with other neurons. If the new axons fail to connect with appropriate neurons, muscle cells, or gland cells, the new axons will dissolve within a few weeks. Thus, the number of neuronal connections is determined by specific nerve growth factors released retrograde from the stimulated cells. Furthermore, when insufficient connectivity occurs, the entire neuron that is sending out the axon branches might eventually disappear.

Consolidation of memory:
For short-term memory to be converted into long-term memory that can be recalled weeks or years later, it must become “consolidated.” That is, the short-term memory, if activated repeatedly, will initiate chemical, physical, and anatomical changes in the synapses that are responsible for the long-term type of memory. This process requires 5 to 10 minutes for minimal consolidation and 1 hour or more for strong consolidation. Studies have shown that rehearsal of the same information again and again in the mind accelerates and potentiates the degree of transfer of short-term memory into long-term memory and therefore accelerates and enhances consolidation.
During consolidation, the new memories are not stored randomly in the brain but are stored in direct association with other memories of the same type.

Short term memory ► Rehearsal ► structural changes ► consolidation ► Long term memory

Long-term memory consolidation of memory depends on relatively permanent changes in the chemical structure of neurons and their synapses.

**Synaptic plasticity (i.e. morphological (or structural) changes) occurs in the postsynaptic neuron (i.e. dendritic spine) as a result of Long-term potentiation (i.e. that permit transmission of stronger signals)**

Long term memory is associated with Long-term potentiation as described above and this will cause Synaptic plasticity due to prolong DNA stimulation and release nerve growth factor.
Growth of Dendritic spine and formation of new synaptic connection:
Most excitatory synapses in the adult brain occur at the heads of tiny, spine like extensions from the dendrites called dendritic spine.

During synaptic stimulation that induces Long-term potentiation, these spines enlarge and change shape ▶️ connected to other neuron ▶️ formation of new synaptic connection.

This may increase the area of contact between the pre- and post neuronal cell. Increase entrance of Calcium induces Long-term potentiation.
3. Neural Stem Cells in Learning and Memory

- Neuronal stem cell produce new neural cell by (neurogenesis) and is involved in learning and memory.
- Neuronal stem cell and neurogenesis is related to hippocampus.

Therefore, soon after birth, the principle of “use it or lose it” governs the final number of neurons and their connectivities in respective parts of the human nervous system. This is a type of learning. For example, if one eye of a newborn animal is covered for many weeks after birth, neurons in alternate stripes of the cerebral visual cortex—neurons normally connected to the covered eye—will degenerate, and the covered eye will remain either partially or totally blind for the remainder of life.

Role of different areas in brain in relation to memory:

The hippocampus is important for maintain recent memories, it is no longer needed once the memory has become consolidated into a more stable. Working memory areas are connected to hippocampus and the adjacent portion of the medial temporal cortex.
The amygdula appears to be particularly important in the memory of fear responses.

The cerebral cortex is thought to store factual information, with verbal memories lateralized to the left hemisphere and visuo-spatial information to the right hemisphere. Apparently, the various parts of the memories (visual, olfactory, auditory, etc.) are located in the cortical regions concerned with these functions.

The inferior temporal lobes are sites for the storage of long-term visual memories and ability to recall names and categories (semantic memory).

1. The left inferior frontal lobe participate in performing exact mathematical calculations.
2. Anterior frontal lobes (prefrontal cortex) are involved in complex problem-solving and planning activities.

When subjects recall words, there is increased activity in the frontal lobe and their left para-hippocampal cortex, but when they recall pictures or scenes, there is activity in their right frontal lobe and the para-hippocampal cortex on both sides.

Lesions of the prefrontal cortex interfere with memory in a less dramatic way than lesions of the medial temporal lobe.
Emotion relation to memory:
A. Strengthening memory: amygdale plays a role in that.
B. Hinder memory: hippocampus plays a role in that such as people with (post-traumatic stress disorder).

The mechanisms by is by increased secretion of "stress hormones" (cortisol) that the hippocampus and amygdala are rich in receptors for these hormones.

2. Prefrontal Cortex relation to memory:
The pre-frontal cortex is involved in:
1. higher cognitive functions, including memory, planning, and anti-judgment
2. normal motivation
3. interpersonal skills
4. sexual desire.

Electroencephalogram (EEG):
EEG is an electrophysiological monitoring method to record electrical activity of the brain.
EEG can be recorded with scalp electrodes through unopened skull or with electrodes on or in the brain.
EEG records may be bipolar or unipolar.
EEG Source: rhythmically discharge cell bodies in the most superficial layer of the cortical gray matter.
Types of waves recorded by EEG:

1. Alpha wave:
   - Alpha wave is a fairly regular pattern of waves at a frequency of 8 to 12 Hz and an amplitude of 50 to 100 μV; occur during wakefulness and periods of relaxation.
   - Alpha wave is most marked in the parieto-occipital area.
   - Alpha wave is seen in adult humans who are awake but at rest with mind wandering and eyes closed.
   - Alpha wave results from spontaneous feedback oscillation in the thalamo-cortical system, possibly including the brain stem activity system as well.
   - Alpha wave frequency decreases with low blood glucose level, low body temperature, low adrenal glucocorticoid hormone, and high arterial PCO₂.
   - Alpha rhythm is replaced by fast, somewhat irregular low voltage activity called as: Alpha block, Arousal, or alternating response (because it is correlated with arousal, after state) or Desynchronization; When attention is focused on something or when sensory stimulation is applied or when mental concentration such as solving arithmetic problems.

![Graph of different EEG wave types](image-url)
• 2. Beta wave:
  It occurs at frequency 18 to 30 Hz. Occur during daily wakefulness, high levels of arousal.
  It is seen over frontal region during extra-activation of CNS or during tension.

• 3. Gamma wave
  ✓ It occurs at frequency 30 to 80 Hz.
  ✓ It seen when individual focuses attention on something, mentally challenged

• 4. Theta wave:
  ❖ It occurs at frequency 4 to 7 Hz.
  ❖ They occur normally in parietal and temporal regions in children, but they also occur during emotional stress in some adults particularly during disappointment and frustration.

• 5. Delta wave:
  - It occurs at frequency less than 4 Hz.
  - They occur in very deep sleep, in infancy and in serious organic brain disease.
  - Synchronizing mechanism can occurs in the cortical neuron themselves (mainly independently of lower structure in the brain) to cause delta wave.

1. Delta Waves
2. Theta Waves
3. Alpha Waves
4. Beta Waves
5. Gamma Waves
Sleep

Sleep is a reversible behavioral state of perceptual disengagement from environment and unresponsive to the environment. Sleep is a person can be aroused while unconsciousness or coma a person cannot be aroused.

Sleep Patterns:

A. Rapid eye movement (REM) sleep (Paradoxical or Desynchronous sleep):

REM sleep the eyes undergo rapid movements despite the fact that the person is still asleep. REM sleep is also called Paradoxical sleep because it is a paradox that a person can still be asleep despite marked activity in the brain. REM sleep occurs in episodes lasting 5 to 30 minutes usually appear on average every 90 minutes in young adults.

REM sleep is generated by cholinergic mediated REM on neurons at the junction of the pons and midbrain (in the upper brain stem reticular formation) begins to discharge before the onset of this phase of sleep. Cholinergic projections to the thalamus promote EEG activation.
REM sleep is thought to play a role in memory consolidation, the synthesis and organization of cognition, and mood regulation. REM sleep is the occurrence of large phasic potentials, in group of three to five that originate in the pons and pass to the lateral geniculate body and from there to the occipital cortex. For this reason, they are called (Ponto-geniculo-occipital (PGO) spikes) with decrease activity in the prefrontal and parietal cortex. REM associated with increased activity in visual association area but there is a decrease in the primary visual cortex. This is consistent with increased emotion and operation of a closed neural system cut off from the related brain activity to the external world.
B. Non-rapid eye movement (NREM) or slow-wave sleep:

NREM is an activity state that is maintained partly through oscillation between the thalamus and cortex. It represents about 75% of the sleep cycle.

It is divided into stages:

N1 (formerly “stage 1”):
N1 is a time of drowsiness or transition from being awake to falling asleep.
N1 is characterized by low-amplitude, high-frequency EEG activity (alpha wave).

N2 (formerly “stage 2”):
N2 is a period of light sleep during which eye movements stop.

Theta waves occur during stages 1 and 2 and are slower in frequency and greater in amplitude than alpha waves. As a person moves from stage 1 to stage 2 sleep, theta wave activity continues; every few minutes, sleep spindles (sudden increase in wave frequency: 10 to 14 Hz, 50 μV wave) and K-complexes (sudden increase in wave amplitude) occur.
N3 (formerly “stages 3 and 4”) is called “slow wave sleep” (SWS). N3 is characterized by the presence of slow brain waves called “delta waves” interspersed with smaller, faster waves.

The amount of slow wave sleep a person gets is directly related to accumulated sleep need — the longer a person has been awake, the more slow wave sleep he or she gets when sleep occurs.

The difference between the dreams that occur in slow-wave sleep and those that occur in REM sleep is

1. REM sleep dreams are associated with more bodily muscle activity
2. slow-wave sleep dreams are usually not remembered because consolidation of the dreams in memory does not occur.

The main difference between REM sleep and wakefulness dreams is that

Dream consciousness is characterized by bizarre imagery and illogical thoughts, and dreams are generally not stored in memory. The reason for this difference is unknown.

The tooth-grinding (bruxism) that occurs in some individuals is also associated with dreaming.
There are two different kinds of sleep:

<table>
<thead>
<tr>
<th>Sleep activity</th>
<th>Rapid eye movement (REM) sleep (Paradoxical or Desynchronized sleep)</th>
<th>Non-rapid eye movement (NREM) or slow-wave sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye movement</td>
<td>Rapid</td>
<td>Slow (drowsiness)</td>
</tr>
<tr>
<td>Vital sign</td>
<td>Fluctuating</td>
<td>stable</td>
</tr>
<tr>
<td>Muscle tone</td>
<td>Decrease or no movement Paralysis (voluntary muscle suppressed)</td>
<td>Some tone in postural muscle Sleep walking and talking (3 and 4)</td>
</tr>
<tr>
<td>Penile erection</td>
<td>common</td>
<td>rare</td>
</tr>
<tr>
<td>Dream</td>
<td>Common Night mares</td>
<td>Rare “dreamless sleep” night terrors</td>
</tr>
<tr>
<td>EEG</td>
<td>low-voltage rapid rhythm, De-synchronized</td>
<td>Sleep Spindles, V-wave, K-complex, slow wave</td>
</tr>
<tr>
<td>Percentage adult</td>
<td>20-25%</td>
<td>75-80%</td>
</tr>
<tr>
<td>Ability to arouse</td>
<td>difficult</td>
<td>easy</td>
</tr>
<tr>
<td>Heart rate, Blood pressure</td>
<td>increase</td>
<td>Decrease (10 to 30%)</td>
</tr>
<tr>
<td>Sympathetic activity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood flow to brain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain activity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body temperature</td>
<td>No regulation</td>
<td>Regulated at lower point</td>
</tr>
<tr>
<td>Duration</td>
<td>Increase as the night progress</td>
<td>decrease as the night progress</td>
</tr>
<tr>
<td>Dream</td>
<td>Vivid, colorful, bizarre,</td>
<td>Vague, partial images</td>
</tr>
</tbody>
</table>
Physiological function of sleep:
The essential role of sleep in homeostasis is perhaps most vividly demonstrated by the fact that rats deprived of sleep for 2 to 3 weeks may actually die.

Sleep causes two major types of physiological effects: first, effects on the nervous system, and second, effects on other functional systems of the body.

Various studies imply that sleep is needed to maintain
1. metabolic-caloric balance,
2. thermal equilibrium
3. immune competence.

In experimental animals, sleep is necessary for learning and memory consolidation.

Learning sessions do not improve performance until a period of slow-wave or slow-wave plus REM sleep has occurred.

Sleep has been postulated to serve many functions, including
(1) neural maturation,
(2) facilitation of learning or memory,
(3) cognition,
(4) clearance of metabolic waste products generated by neural activity in the awake brain, and
(5) conservation of metabolic energy.

We might postulate that the principal value of sleep is to restore natural balances among the neuronal centers. The specific physiological functions of sleep, however, remain a mystery and are the subject of much research.
An earlier theory of sleep was that the excitatory areas of the upper brain stem, the reticular activating system, simply became fatigued during the waking day and became inactive as a result. Sleep is caused by an active inhibitory process by the "sleep centers" which is required to cause sleep by inhibiting other parts of the brain.

"Sleep centers" are located in:
1) A center located below the mid-pontile level of the brain stem

Cycle between sleep and wakefulness:

When the mid-pontile level of the brain stem is not activated:
- the mesencephalic and upper pontile reticular activating nuclei are released from inhibition, which allows the reticular activating nuclei to become spontaneously active.
- excites both the cerebral cortex and the peripheral nervous system
- both of which send numerous positive feedback signals back to the same reticular activating nuclei to activate them still further.

Therefore, once wakefulness begins, it has a natural tendency to sustain itself because of all this positive feedback activity.

After the brain remains activated for many hours, even the neurons in the activating system presumably become fatigued. Consequently, the positive feedback cycle between the mesencephalic reticular nuclei and the cerebral cortex fades and the sleep-promoting effects of the sleep centers take over, leading to rapid transition from wakefulness back to sleep.
2) Raphe nuclei in the lower half of the pons and in the medulla is required to cause sleep by inhibiting other parts of brain. Nerve ending of fibers from these raphe neurons secret serotonin

3) Stimulus area of nucleus of tractus solitaries

4) Diencephalon
(a) Hypothalamus, mainly in the suprachiasmal area
(b) Thalamus.

Destruction of 3 and 4 causes lead to a high state of wakefulness

Possible transmitter substances related to sleep.

a) Serotonin related to raphe nuclei in the lower half of the pons and in the medulla and causes sleep

b) Muramyl peptide, causes sleep

c) Orexin (also called hypocretin) is produced by neurons in the hypothalamus that provide excitatory input to many other areas of the brain where there are orexin receptors. Orexin neurons are most active during waking and almost stop firing during slow wave and REM sleep
Sleep stages:
In a typical night of sleep, a young adult first enters NREM sleep, passes through stages 1 and 2, and spends 70–100 minutes in stages 3 and 4. Sleep then lightens, and a REM period follows. This cycle is repeated at intervals of about 90 minutes throughout the night.

The cycles are similar, though there is less stage 3 and 4 sleep and more REM sleep toward morning. Thus, four to six REM periods occur per night.

REM sleep occupies 80% of total sleep time in premature infants and 50% in full-term neonates. Thereafter, the proportion of REM sleep falls rapidly and plateaus at about 25% until it falls further in old age. Children have more total sleep time and stage 4 sleep than adults.
Seizures and Epilepsy
Seizures are temporary disruptions of brain function caused by uncontrolled excessive neuronal activity.
Depending on the distribution of neuronal discharges, seizure manifestations can range from experiential phenomena that are barely noticeable to dramatic convulsions.
Approximately 5 to 10 percent of the population will have at least one seizure in their lifetime.
Secondary Epilepsy
Secondary Epilepsy can be caused by multiple neurological or medical conditions, such as
- acute electrolyte disorders,
- hypoglycemia,
- drugs (e.g., cocaine),
- eclampsia,
- kidney failure,
- hypertensive encephalopathy,
- meningitis, and so forth.
These temporary *symptomatic* seizures usually do not persist if the underlying disorder is corrected.
In cases in which a person has brain injury due to trauma, stroke, or infection, there may be a delay of several months or years after the injury before the seizures begin.
Primary Epilepsy

Epilepsy is a chronic condition of *recurrent seizures* that can also vary from brief and nearly undetectable symptoms to periods of vigorous shaking and convulsions.

Epilepsy is not a single disease.

Epilepsy clinical symptoms are heterogeneous and reflect multiple underlying causes and pathophysiological mechanisms that cause cerebral dysfunction and injury, such as trauma, tumors, infection, or degenerative changes.

Hereditary factors appear to be important, although a specific cause cannot be identified in many patients and several factors may coexist, reflecting an acquired brain pathology and genetic predisposition.

Epilepsy is estimated to affect approximately 1 percent of the population, or 65 million people worldwide.

Pathophysiology

At a basic level an epileptic seizure is caused by a disruption of the normal balance between inhibitory and excitatory currents or transmission in one or more regions of the brain. Drugs or pathological factors that increase neuronal excitation or impair inhibition tend to be *eliptogenic* (i.e., predisposing a person to epilepsy), whereas effective antiepileptic drugs attenuate excitation and facilitate inhibition.
Epileptic seizures can be classified into two major types:

Focal (Partial) Epileptic Seizures

Focal epileptic seizures begin in a small localized region of the cerebral cortex or deeper structures of the cerebrum and brain stem and have clinical manifestations that reflect the function of the affected brain area. Most often, focal epilepsy results from some localized organic lesion or functional abnormality, such as:

1. scar tissue in the brain that pulls on the adjacent neuronal tissue,
2. a tumor that compresses an area of the brain,
3. a destroyed area of brain tissue, or
4. congenitally deranged local circuitry.

These lesions can promote extremely rapid discharges in the local neurons; when the discharge rate rises above several hundred per second, synchronous waves begin to spread over adjacent cortical regions. These waves presumably result from localized reverberating circuits that may gradually recruit adjacent areas of the cortex into the epileptic discharge zone.

The process spreads to adjacent areas at a rate as slow as a few millimeters a minute to as fast as several centimeters per second.

Focal seizures can spread locally from a focus or more remotely to the contralateral cortex and subcortical areas of the brain through projections to the thalamus, which has widespread connections to both hemispheres.
When such a wave of excitation spreads over the motor cortex, it causes a progressive “march” of muscle contractions throughout the opposite side of the body, beginning most characteristically in the mouth region and marching progressively downward to the legs but at other times marching in the opposite direction. This phenomenon is called *jacksonian march*.

Focal seizures are often classified as *simple partial* when there is no major change in consciousness or as *complex partial* when consciousness is impaired. Simple partial seizures may be preceded by an *aura*, with sensations such as fear, followed by motor signs, such as rhythmic jerking or tonic stiffening movements of a body part. A focal epileptic attack may remain confined to a single area of the brain, often the temporal lobe, but in some instances strong signals spread from the focal region and the person may lose consciousness. Complex partial seizures may also begin with an aura followed by impaired consciousness and strange repetitive movements (*automatisms*), such as chewing or lip smacking. After recovery from the seizure the person may have no memory of the attack, except for the aura. The time after the seizure, prior to the return of normal neurological function, is called the *postictal period*. 
Psychomotor, temporal lobe, and limbic seizures are terms that have been used in the past
to describe many of the behaviors that are now classified as complex partial seizures. However, these terms are not synonymous. Complex partial seizures can arise from regions other than the temporal lobe and do not always involve the limbic system. Also, automatisms (the “psychomotor” element) are not always present in complex partial seizures. Attacks of this type frequently involve part of the limbic portion of the brain, such as the hippocampus, the amygdala, the septum, and/or portions of the temporal cortex.

A typical EEG during a psychomotor seizure, showing a low-frequency rectangular wave with a frequency between 2 and 4 per second and with occasional superimposed 14-per-second waves.
Generalized Seizures

Generalized epileptic seizures are characterized by diffuse, excessive, and uncontrolled neuronal discharges that at the outset spread rapidly and simultaneously to both cerebral hemispheres through interconnections between the thalamus and cortex. However, it is sometimes difficult clinically to distinguish between a primary generalized seizure and a focal seizure that rapidly spreads. Generalized seizures are subdivided primarily on the basis of the ictal motor manifestations, which, in turn, depend on the extent to which subcortical and brain stem regions participate in the seizure.

[Diagrams showing neuronal connections and spread of seizures]
Generalized Tonic-Clonic (Grand Mal) Seizures

1. **Tonic-clonic**: Characterized by:
   The duration of the seizure is usually 1 to 3 minutes.
   These seizures are often described as “grand mal.”

   The seizures are divided into two phases, the tonic phase and the clonic phase, hence the name of the seizure, though a tonic–clonic seizure will often be preceded by an aura.

**Aura**

   The person may feel lightheadedness and/or dizziness, unusual (and possibly inappropriate) emotions, intense feelings of discomfort or foreboding, altered vision and hearing (which may or may not include hallucinations).

   An aura may last as little as a few minutes or as long as several hours, though some with epilepsy do not experience them at all. Many auras are followed by a tonic–clonic seizure.
Tonic phase

The person will quickly lose consciousness, and the skeletal muscles will suddenly tense, often causing the extremities to be pulled towards the body or rigidly pushed away from it, which will cause the person to fall if standing.

The tonic phase is usually the shortest part of the seizure, usually lasting only a few seconds. The person may also express vocalizations like a loud moan or scream during the tonic stage, due to air forcefully expelled from the lungs.
Clonic phase

The person's muscles will start to contract and relax rapidly, causing convulsions. These may range from exaggerated twitches of the limbs to violent shaking or vibrating of the stiffened extremities. The person may roll and stretch as the seizure spreads.

The eyes typically roll back or close and the tongue often suffers bruising sustained by strong jaw contractions.

The typical EEG from almost any region of the cortex during the tonic phase of generalized tonic-clonic seizure demonstrates that high-voltage, high-frequency discharges occur over the entire cortex. Furthermore, the same type of discharge occurs on both sides of the brain at the same time, demonstrating that the abnormal neuronal circuitry responsible for the attack strongly involves the basal regions of the brain that drive the two halves of the cerebrum simultaneously.
What Initiates a Generalized Tonic-Clonic Seizure?

The majority of generalized seizures are idiopathic, which means that the cause is unknown. Many people who have generalized tonic-clonic attacks have a hereditary predisposition to epilepsy, a predisposition that occurs in about 1 of every 50 to 100 persons. In these people, factors that can increase the excitability of the abnormal “epileptogenic” circuitry enough to precipitate attacks include (1) strong emotional stimuli, (2) alkalosis caused by overbreathing, (3) drugs, (4) fever, and (5) loud noises or flashing lights.

Even in people who are not genetically predisposed, certain types of traumatic lesions in almost any part of the brain can cause excess excitability of local brain areas, as we discuss shortly; these local brain areas also sometimes transmit signals into the activating systems of the brain to elicit tonic-clonic seizures.

What Stops the Generalized Tonic-Clonic Attack?

The extreme neuronal overactivity during a tonic-clonic attack is presumed to be caused by massive simultaneous activation of many reverberating neuronal pathways throughout the brain. Although the factors that terminate the attack are not well understood, it is likely that active inhibition occurs by inhibitory neurons that have been activated by the attack.
Absence Seizures (Petit Mal Seizures)

Absence seizures, formerly called petit mal seizures, usually begin in childhood or early adolescence and account for 15 to 20 percent of epilepsy cases in children. Absence seizures almost certainly involve the thalamocortical brain activating system. They are usually characterized by 3 to 30 seconds of unconsciousness or diminished consciousness, during which time the person often stares and has twitchlike contractions of muscles, usually in the head region, especially blinking of the eyes; this phase is followed by a rapid return of consciousness and resumption of previous activities. This total sequence is called the absence syndrome or absence epilepsy.

The patient may have one such attack in many months or, in rare instances, may have a rapid series of attacks, one after the other. The usual course is for the absence seizures to appear first during childhood or adolescence and then to disappear by the age of 30 years. On occasion, an absence seizure will initiate a generalized tonic-clonic (grand mal) attack.
3. Myoclonic (infantile spasm):

Seen in children or infants, caused by cerebral pathology, often with mental retardation. Infantile spasms usually disappear by age 4, but child may develop other types of seizures.

Characterized by:

A. Single and very brief jerks of all major muscle groups. It is of two types:
   1. Extensor type – infant extends head, spreads arms out, bend body backward in “spread eagle” position.
   2. Mixed flexor and extensor types may occur in clusters or alternate.

B. May cause children to drop or throw something.

C. Infant may cry out, grunt, grimace, laugh, or appear fearful during an attack.

D. Patients with these may not lose consciousness, due to the seizure lasting less than 3 to 4 seconds.

E. Patients may describe these seizures as shoulder shrugs or spinal chills.

F. Myoclonic seizures may cluster and build into a generalized tonic-clonic seizure.

G. Myoclonic type is often transient may occur repetitively.
4. Atonic:
Characterized by:
A. The patient loses consciousness and muscle tone.
B. No muscle movements are typically noted, and the patient will fall when they are not lying down or sitting in a chair.
C. These seizures may be described as “falling out.”
D. Usually accruing in children, this type is associated with complete loss of consciousness and muscle tone.