

جانی

PHYSIOLOGY

SHEET NO.

3

WRITER :

Doctor 019

CORRECTOR :

Malek Absia

DOCTOR :

Faisal Mohammad

★ Language areas

□ Located in large area surrounding the left (or language dominant) lateral sulcus. □
Major parts and functions:

✦ Wernicke's area

Damage ⑦ **sensory aphasia** (receptive aphasia), in which individuals have difficulty understanding written and spoken language. They demonstrate **fluent** speech, but their speech lacks content or meaning.

✦ Broca's area

Damage ⑦ **motor aphasia** (expressive aphasia), in which individuals can understand written and spoken language and form ideas, but they have difficulty expressing their ideas by speech and their speech is **non-fluent**.

□ If both Wernicke's and Broca's areas are damaged ⑦ **global aphasia** (both expressive and receptive language skills are reduced).

✦ **Lateral prefrontal cortex**: for language comprehension and word analysis.

✦ **Lateral and ventral temporal lobe**: coordinate auditory and visual aspects of language.

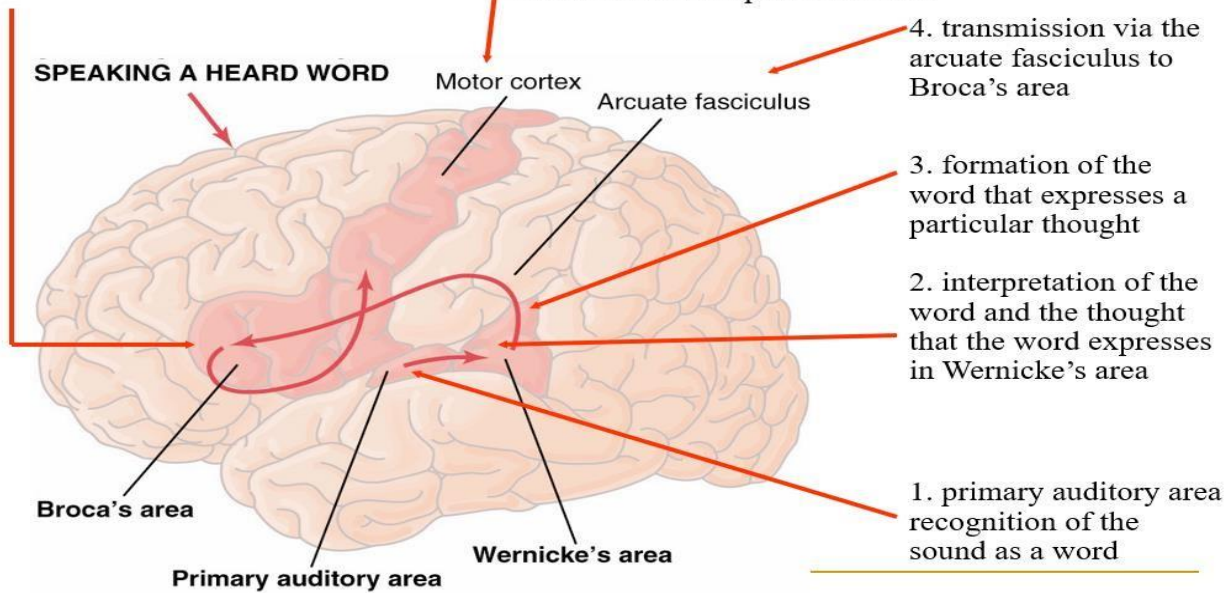
★ Intellectual functions of the prefrontal association area

- responsible for calling forth stored information and using it to obtain a goal.
- responsible for concerted thinking in a logical sequence.
- elaboration of thought.
- prognosticate, plan, consider consequences of motor actions before they are performed.
- correlate widely divergent information, control one's activities.
- Personality trait and behavior that confines to values and manners of the culture.
- damage causes an inability to keep track of simultaneous bits of information, easily distracted.

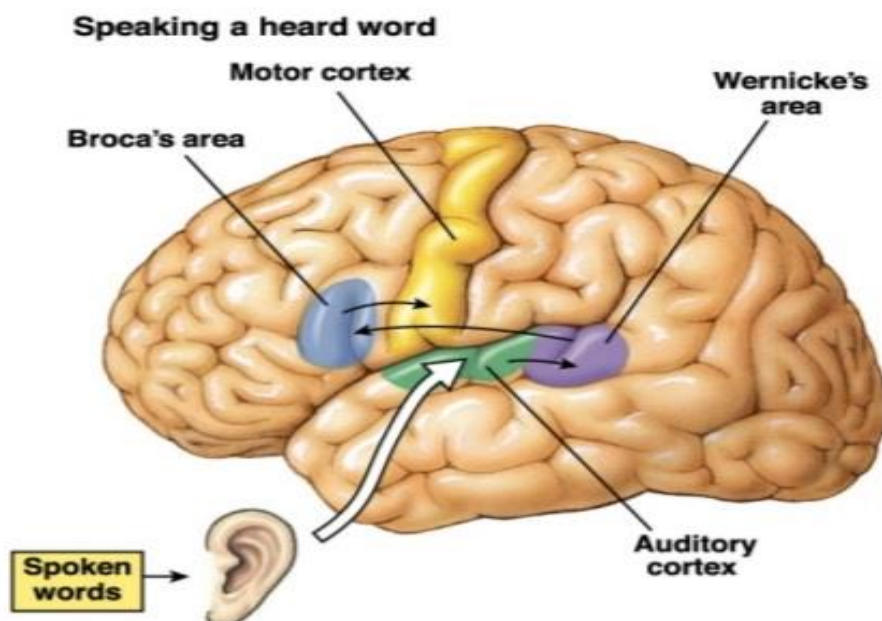
Pathways for auditory communication

5. activation of motor programs in Broca's area for control of word formation

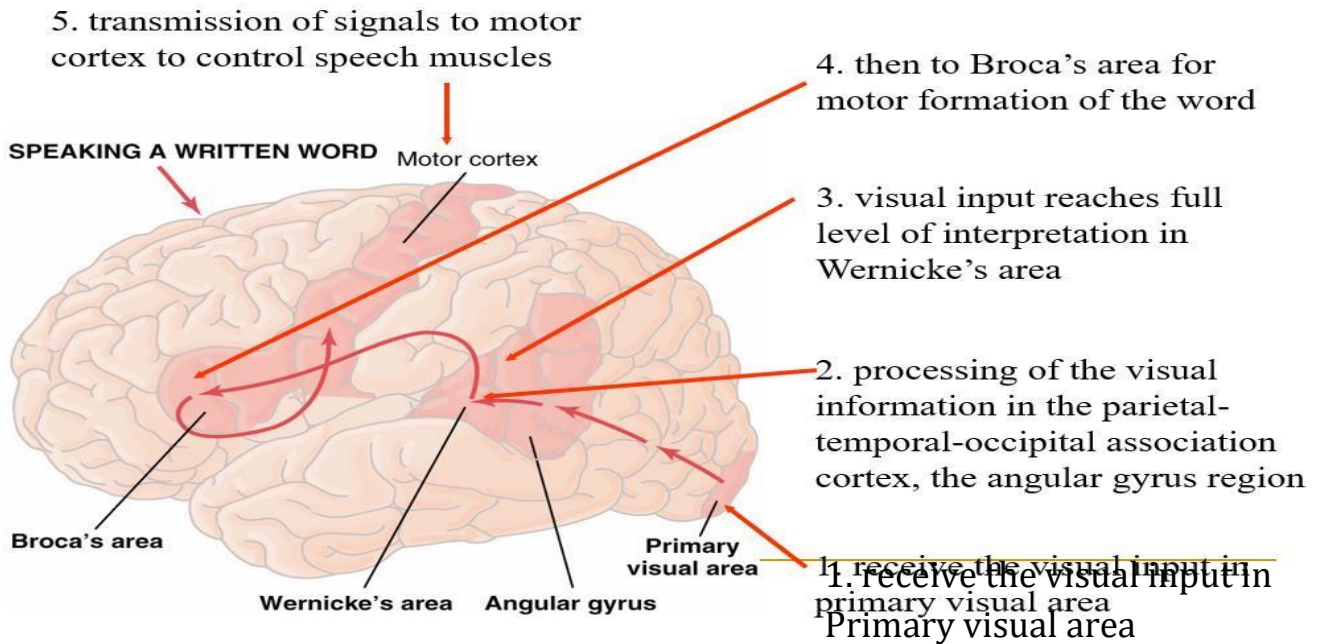
6. transmission of signals to motor cortex to control speech muscles



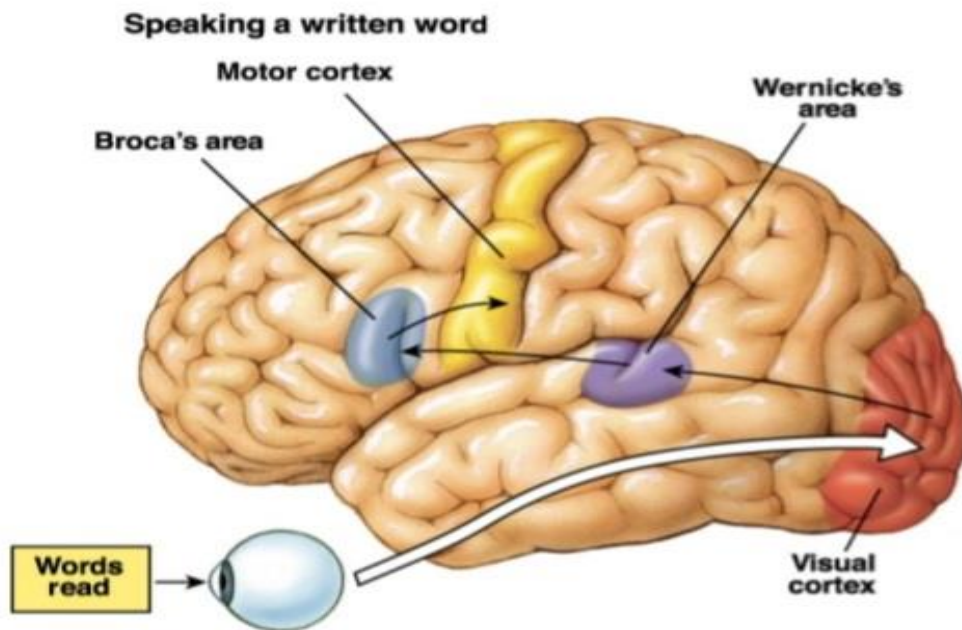
□ Note: after the primary auditory area it's transmitted to the auditory association area and then to Wernicke's area.



Pathways for visual communication



- Speech involves two things:
formation in the mind of thoughts to be expressed and the choice of words.
motor control of vocalization and the act of vocalization.

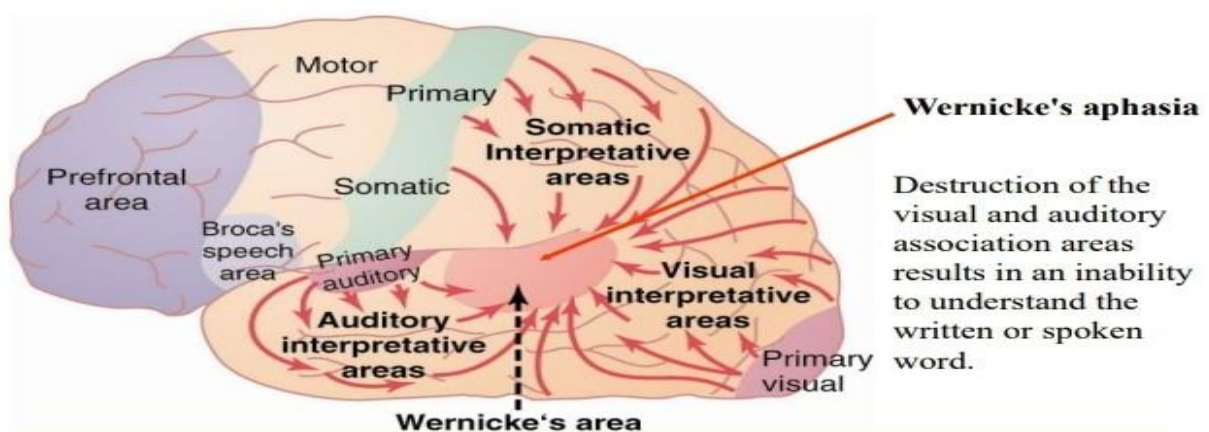


- Formation of word, thought and choice of words is function of **Wernicke's area**.
- **Broca's area** controls the motor coordination required for speech.
- ➔ Destruction of the visual and auditory association areas results in an inability to understand the written words (word blindness) or spoken words (word deafness).

What is the difference between sensory aphasia and word deafness?

Word deafness results from damage to the auditory association area, and it leads to total loss of the ability to understand spoken words. Sensory aphasia results from damage to the Wernicke's area, some people might be capable of understanding spoken words (auditory association area is intact) but are unable to put together the appropriate sequences of words to express the thought, but in more severe damage they might be unable to formulate the thoughts.

Sensory Aspects of Communication



***There are fibers that connect the two cerebral hemispheres called commissural fibers**

***The largest fiber is called corpus callosum**

✓ Corpus callosum

- Connects the two hemispheres and allows transfer of information.
- Interruption of these fibers can lead to bizarre types of anomalies.

People whose corpus callosum is completely sectioned have two entirely separate conscious portions of the brain.

In a person with a sectioned corpus callosum, only the dominant hemisphere could understand the spoken word, whereas the non-dominant hemisphere could understand only the written word but not the spoken word*. The non-dominant hemisphere can elicit motor response to the written word without the dominant hemisphere ever knowing why the response was performed!

*non-dominant hemisphere has the angular gyrus, which is partly responsible for the interpretation of written words, but it doesn't have the Wernicke's area because it's only present in the dominant hemisphere.

✓ Thoughts, learning and memory

- ✦ Neural mechanism for **thought** is not known.
 - Most likely a specific pattern of simultaneous neural activity in many brain areas.
 - Destruction of cerebral cortex does not prevent one from thinking. However, depth of thought and level of awareness may be less.
- ✦ **Learning** is acquiring new sensory information or motor skills.
 - Learning results from the change in the capability of synaptic transmission from neuron to neuron as a result of prior stimulation.
- ✦ **Memory** is the ability to recall specific information.
 - **Memory trace** is a specific pattern or pathway of signal transmission.
 - the more important the information is, the more easily you remember it.

There are three types of memory:

1) Immediate memory: lasts for seconds or minutes (remembering 10 digit phone number).

Mechanism: may result from **synaptic potentiation** through the accumulation of calcium in the presynaptic membrane, which promotes neurotransmitter release.

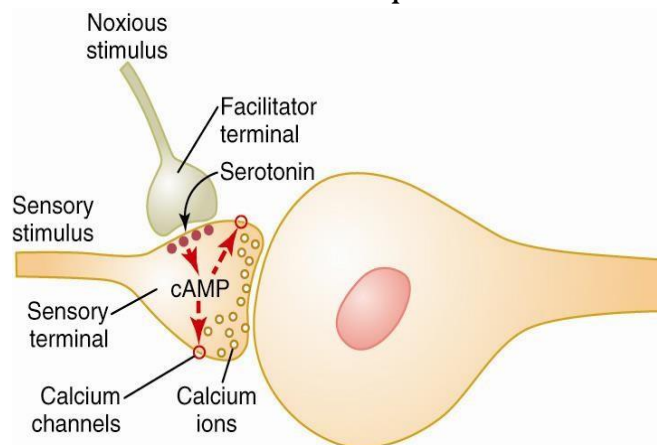
2) Short term memory: lasts for days to weeks (remembering exam material 🧠).

Mechanism: result from a **temporary physical or chemical change** in the pre- or postsynaptic membrane.

3) Long term memory: lasts for years or for a lifetime (your name, date of birth..). Results from **permanent changes** (to be discussed after a while).

★ **Molecular basis for intermediate memory** now let's talk about the molecular mechanism in which our memory is formed and even how we forget things!

- There are two synaptic terminals, one is **sensory terminal** from a sensory input neuron, and the other is **facilitator terminal** which is activated by noxious stimulus (when you **try** to remember something).
- The facilitator terminal is connected presynaptically with the sensory terminal and stimulates it by releasing a transmitter (serotonin).
- The transmitter activates G protein in the sensory terminal which in turn activates adenylate cyclase resulting in an increase in cAMP.
- cAMP activates a protein kinase that phosphorylates a component of the K⁺ channel blocking its activity.
- This causes prolonged depolarization and prolonged action potential which results in prolonged activation of calcium channels allowing high amounts of calcium to enter the sensory synaptic terminal.
- These calcium ions cause greatly increase transmitter release by the sensory terminal, thereby markedly facilitating synaptic transmission to the subsequent neuron.



➡ Important notes:

Even when the facilitator neuron is not stimulated (that is, you don't try to remember something again, like when you don't revise the material before the exam), there is continuous stimulation for the sensory terminal. However, without the stimulation of the facilitator terminal, signal transmission at first is great, but it becomes less and less intense with repeated stimulation until transmission almost ceases. This phenomenon is called **habituation**. It's a form of **negative memory** that causes the neuronal circuits to lose its response to repeated events that are insignificant, or a bad experience that you don't like to remember.

So habituation is a progressive decline in sensitivity. But how does this occur? This results from progressive decline in the number of active calcium channels ⑦ less calcium entry ⑦ less transmitter released.

On the other hand, the associative effect of stimulating the facilitator terminal at the same time that the sensory terminal is stimulated causes **prolonged** increase in the excitatory sensitivity of the sensory terminal, which establishes the **memory trace**, and this prevents habituation. (isn't that crazy? 😳)

★ Mechanism of Long-term memory

Long term memory is believed to result from actual **structural and functional changes (permanent)**, instead of only chemical changes.

- An increase in the **area for vesicular release**, therefore, more transmitter is released. (the number of neurons can't increase, rather, the number of synapses and spikes is increased) 🔗 **permanent anatomical (structural) change**.
- This enlargement of the release site area results from synthesis of **release site proteins**. (long term memory involves genetic changes. some genes are dormant, when you activate these genes you activate the formation of new proteins) 🔗 **permanent physiological (functional) change**.
during periods of inactivity the area decreases in size.

★ Consolidation of memory

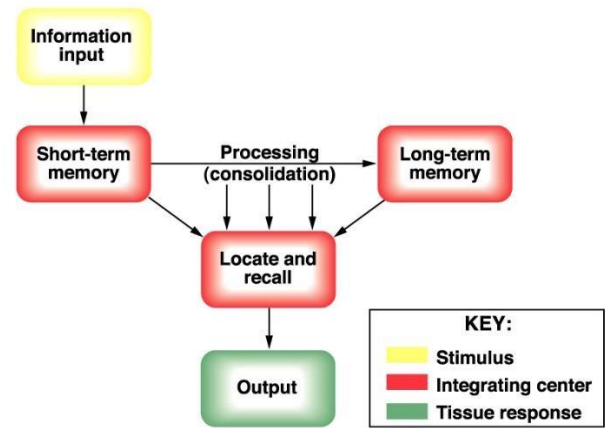
- It means converting immediate into short and long-term memory.
- There are two ways to speed up consolidation:
 1. Rehearsal (repetition of the information).
 2. Filing of information.

It results from chemical, physical and anatomical changes in the synapse (as we discussed).

It requires time.

interruption of the process by electrical shock or by anesthesia will prevent memory development.

- The first stage of memory is called **registration**, when you receive the information into the short-term memory. **Retrieval** is recalling back the stored information in response to some cues. You can convert short-term memory to long-term memory through consolidation.
 - Immediate memory doesn't need registration.



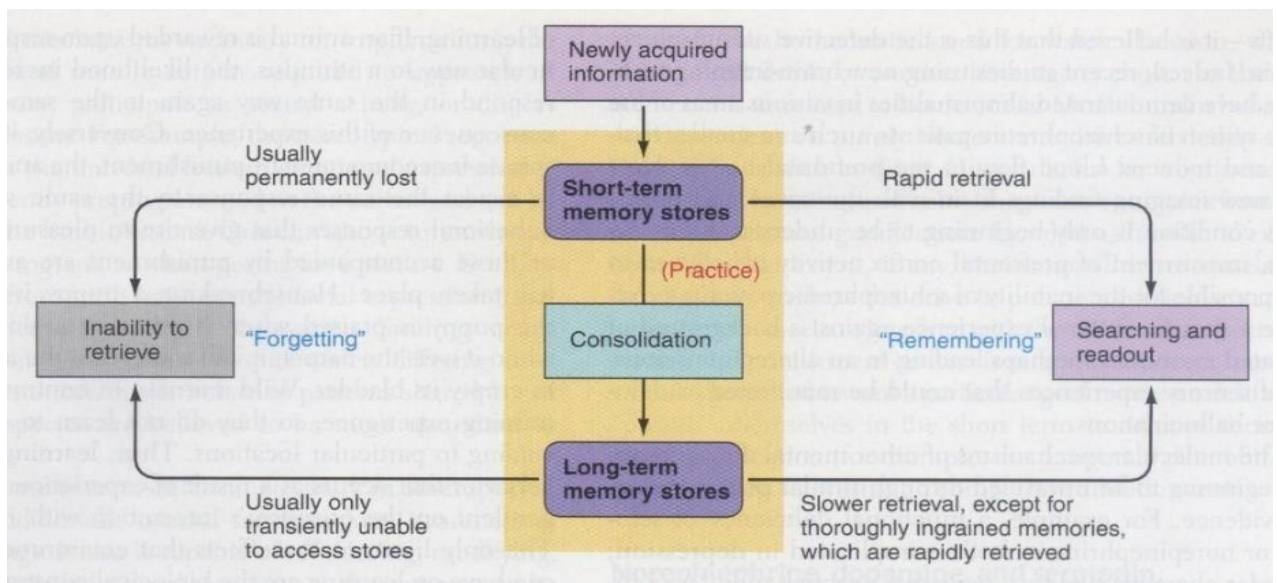
✓ **Brain centers and memory**

- ✦ One of the areas in the brain that is important for recalling memory is **thalamus**. Thalamus scans the cortex for the area and the circuit for the stored memory (search). Damage to thalamus causes **retrograde amnesia** or the inability to recall stored experiences from the **recent past events**. When the damage is more severe, more distant past memories will be lost.
- ✦ **Hippocampus** also has a very important role in memory.
- ✦ **Destruction in hippocampus cause anterograde amnesia**

★ **Comparison between long-term and short-term memory**

| CHARACTERISTIC | SHORT-TERM MEMORY | LONG-TERM MEMORY |
|---|--|--|
| Time of Storage after Acquisition of New Information | Immediate | Later; must be transferred from short-term to long-term memory through consolidation; enhanced by practice or recycling of information through short-term mode |
| Duration | Lasts for seconds to hours to days and weeks | Retained for days to years |
| Capacity of Storage | Limited | Very large |
| Retrieval Time (remembering) | Rapid retrieval | Slower retrieval, except for thoroughly ingrained memories, which are rapidly retrieved |
| Inability to Retrieve (forgetting) | Permanently forgotten; memory fades quickly unless consolidated into long-term memory | Usually only transiently unable to access; relatively stable memory trace |
| Mechanism of Storage | Involves transient changes in functions of pre-existing synapses, such as altering amount of neurotransmitter released | Involves relatively permanent functional or structural changes between existing neurons, such as formation of new synapses; synthesis of new proteins plays key role |

- ➔ The **capacity** of short-term memory is limited, that's why we forget (delete :p) much of the information after an exam to leave a space for the next exam material, but if these information were consolidated into the long-term memory, it has a very large capacity which is enough to huge amounts of information.
- ➔ On the other hand, the **retrieval time** (remembering) of the information from the short-term memory is rapid, whereas remembering information from the long-term memory takes longer time, that's why sometimes you try hard to remember something and you give up, but after a while the information pops into your head.



This slide will be discussed in the next sheet

