



بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

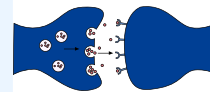


Cerebral Cortex Intellectual Functions (Pt.2)

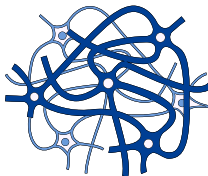
FINAL | Lecture 8

﴿إِنِّي تَوَكَّلْتُ عَلَى اللَّهِ رَبِّي وَرَبِّكُمْ مَا مِنْ دَابَّةٍ إِلَّا هُوَ آخِذٌ بِنَاصِيَتِهَا إِنَّ رَبِّي عَلَى صِرَاطٍ مُسْتَقِيمٍ﴾

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Language Areas

- Located in a large area surrounding the left (or language-dominant) lateral sulcus
- Major parts and functions:
 - Wernicke's area (Parieto-occipito-temporal area) – involved in sounding out unfamiliar words, **speech is fluent but nonsensical**—sensory aspect of speech- damage—→ sensory aphasia (Receptive aphasia)
 - Broca's area (Most inferior part of premotor cortex) – speech preparation and production, can't speak but maybe can write **their ideas**- motor aspect of speech – damage—→ motor aphasia (expressive aphasia)
 - Both Wernicke's and Broca's area damage —→ global aphasia (**severe impairment of both comprehension and speech production**).
 - Lateral prefrontal cortex – language comprehension and word analysis
 - Lateral and ventral temporal lobe – coordinate auditory and visual aspects of language
 - **Conduction aphasia occurs when there is isolated damage to the arcuate fasciculus.**
 - **Patients with conduction aphasia have impaired repetition, while other language functions may be relatively preserved**

Brain Organization and Handedness

- Close to 90% of people are right-handed and close to 10% are left-handed and a small number are ambidextrous (use both hands)
- 95% of right-handers process speech primarily in the left hemisphere
 - left-handers: around **65% in left hemisphere**, 15- 20% in right hemisphere, 15-20% in both
- More than 90% of people are born with the left hemisphere area that controls the movement of the right hand is bigger
 - They tend to use the right hand, this area grows and become dominant, **This applies to lower limbs too**

Doctor said that someone is right handed because the area that controls their right hand is bigger, causing them to have "more tendency" to use it.

But technically, this is not the "cause" of someone's handedness; this is more of a "consequence". It is just a classical example of synaptic plasticity: somehow (due to some genetic and prenatal developmental factors) that will cause someone to be, for example, right handed, so they start using their right hand consistently, driving synaptic plasticity, creating new connections, and overall developing a larger cortical representation as a result.

If you read Guyton you might get confused. It mentions how the theory for cerebral dominance is due to a structural difference at birth: at birth the left posterior temporal lobe (planum temporale) is larger, and because it is larger the brain directs its attention towards it, causing it to get used more for language, undergoes more neuroplastic development, and becomes the dominant "language hemisphere."

This concept of "larger leads to higher tendency to be used" is true here but not for handedness, because newborns who end up becoming right handed are not born with their area controlling the right hand being larger. Instead, it is not until they start using their right hand that it ends up getting bigger.

I couldn't find any papers discussing changes in asymmetry at birth for handedness, although subcortical asymmetries might be present, but if you find anything, please let us know

Brain Organization and Handedness

...cont

- Left handed people have their right cerebral hemisphere area that controls the movement of the left hand bigger
 - If they use the left hand then this area grow and become dominant
 - They still can convert and the younger the more easier if they start to use their right hand instead and then they become right handed
 - Same applies for using the legs

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
 - damage causes an inability to keep track of simultaneous bits of information, easily distracted

Intellectual Functions of the Prefrontal Association Area (Cont'd)

- 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
 - Destruction to that area will cause the opposite, and that is what we see with Phineas Gage (he became easily distractible, socially disinhibited, etc..)

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

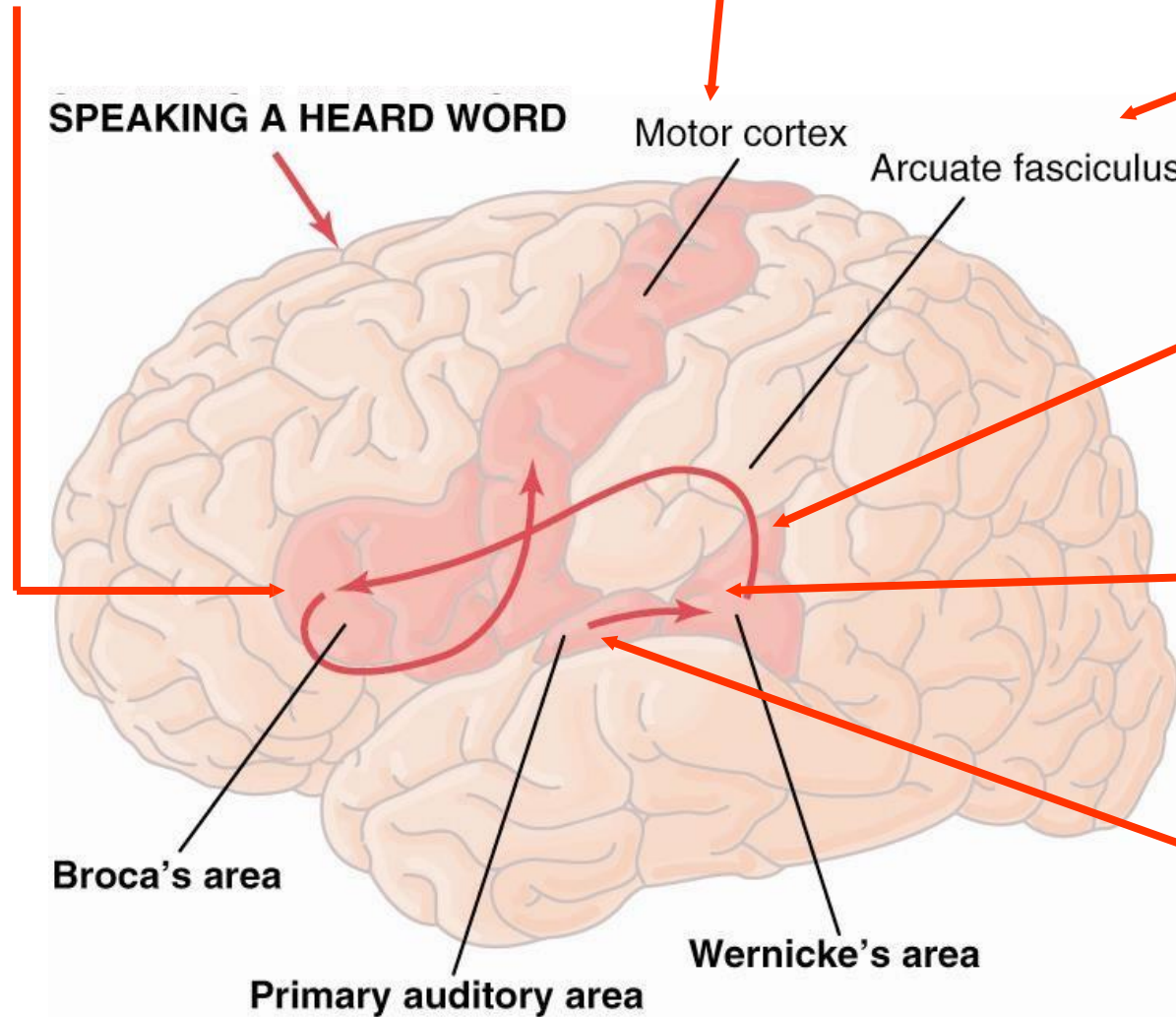
4. transmission via the arcuate fasciculus to Broca's area

3. formation of the word that expresses a particular thought *Now you've an idea*

2. interpretation of the word and the thought that the word expresses in Wernicke's area

1. primary auditory area recognition of the sound as a word

2nd Association area



- The left hemisphere is typically the dominant hemisphere for language.
- Sensory and motor pathways follow contralateral organization, meaning that information from the right side of the body is processed in the left hemisphere (through early crossing of neural pathways).
- Right hemiplegia indicates a lesion in the left hemisphere → high likelihood of aphasia.
- Left hemiplegia indicates a lesion in the right hemisphere → language is usually preserved, but there may be impairment in the emotional aspect of speech (dysprosody).

Key concept:

Left hemiplegia → **right hemisphere** lesion

Right hemiplegia → **left hemisphere** lesion (**contralateral** representation – **possible exam question!**)

- The **auditory association cortex** is responsible for interpreting sounds, including recognizing speech, music, and environmental noises.

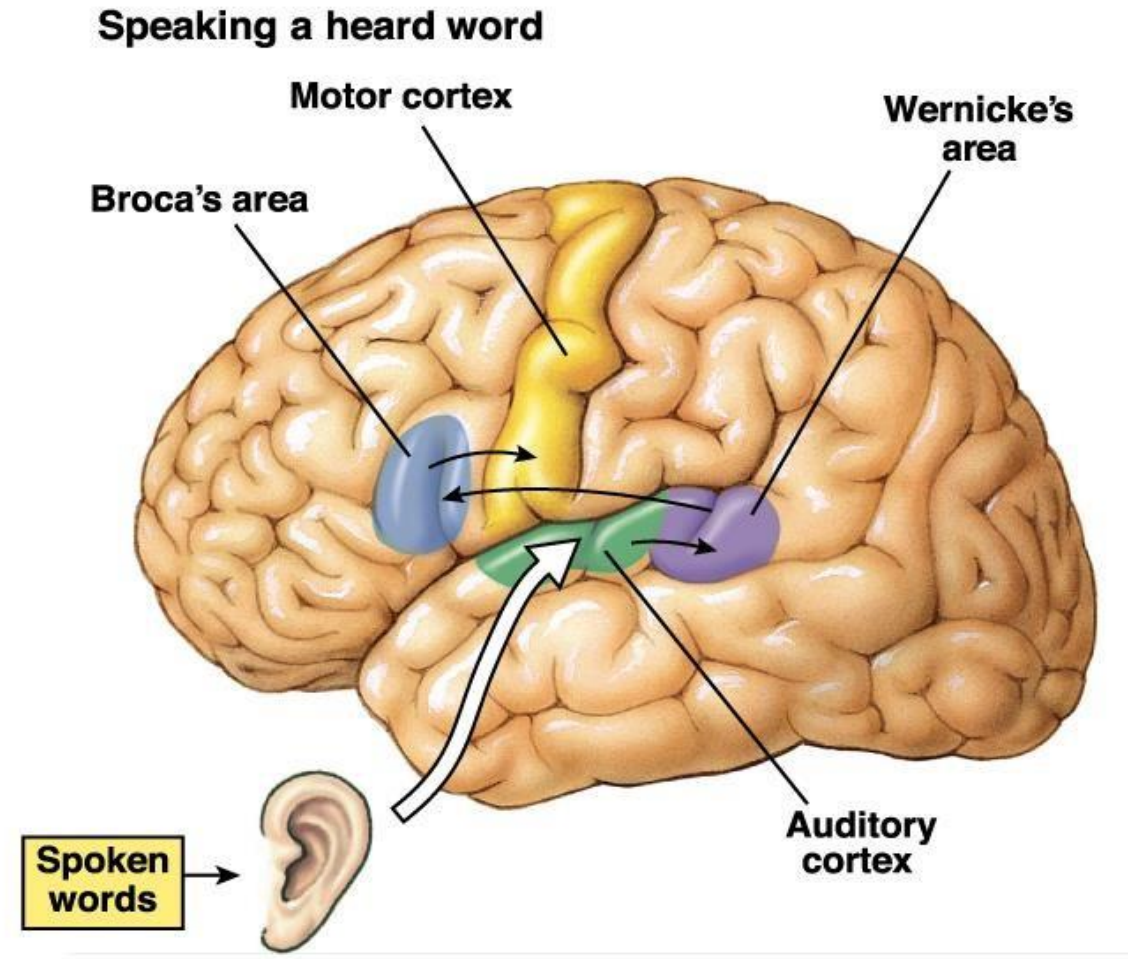
- The **Wernicke's area** is responsible for understanding language.

It integrates auditory and visual inputs and converts them into meaningful language

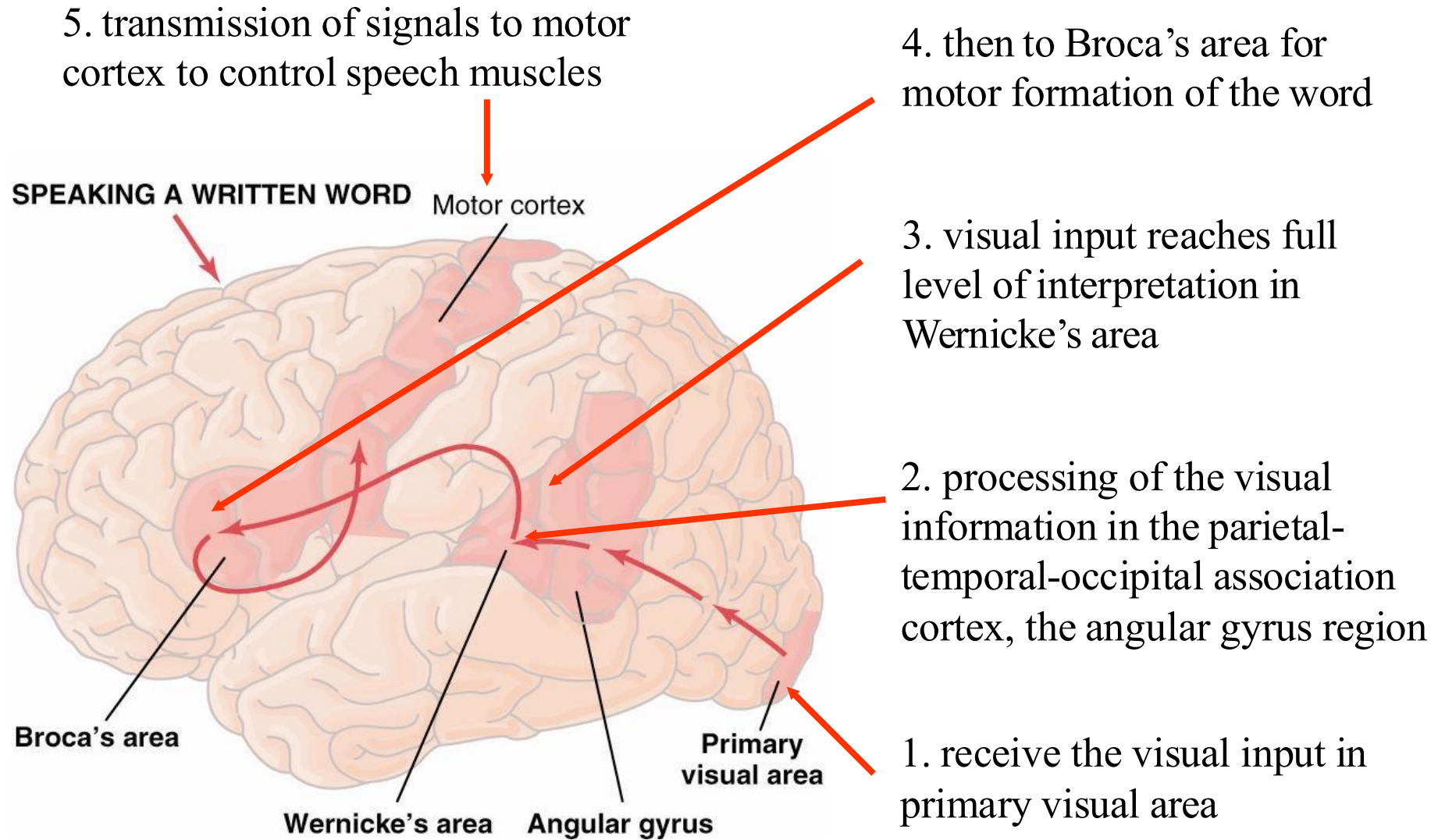
- If the auditory cortex is damaged → word deafness:

You hear sounds, but **don't understand speech**

You won't become completely deaf because hearing pathways are bilateral.

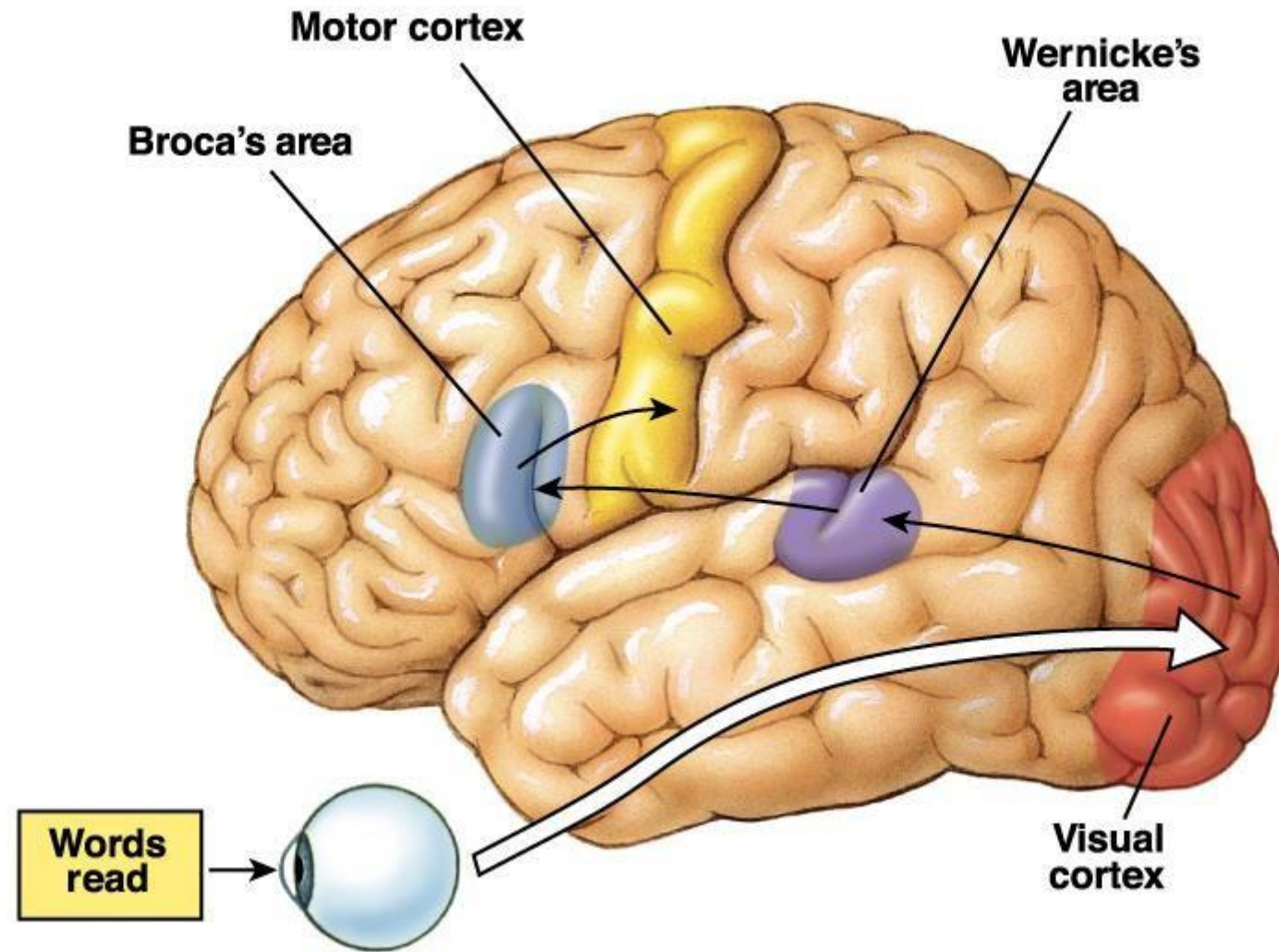


Pathways for Visual Communication

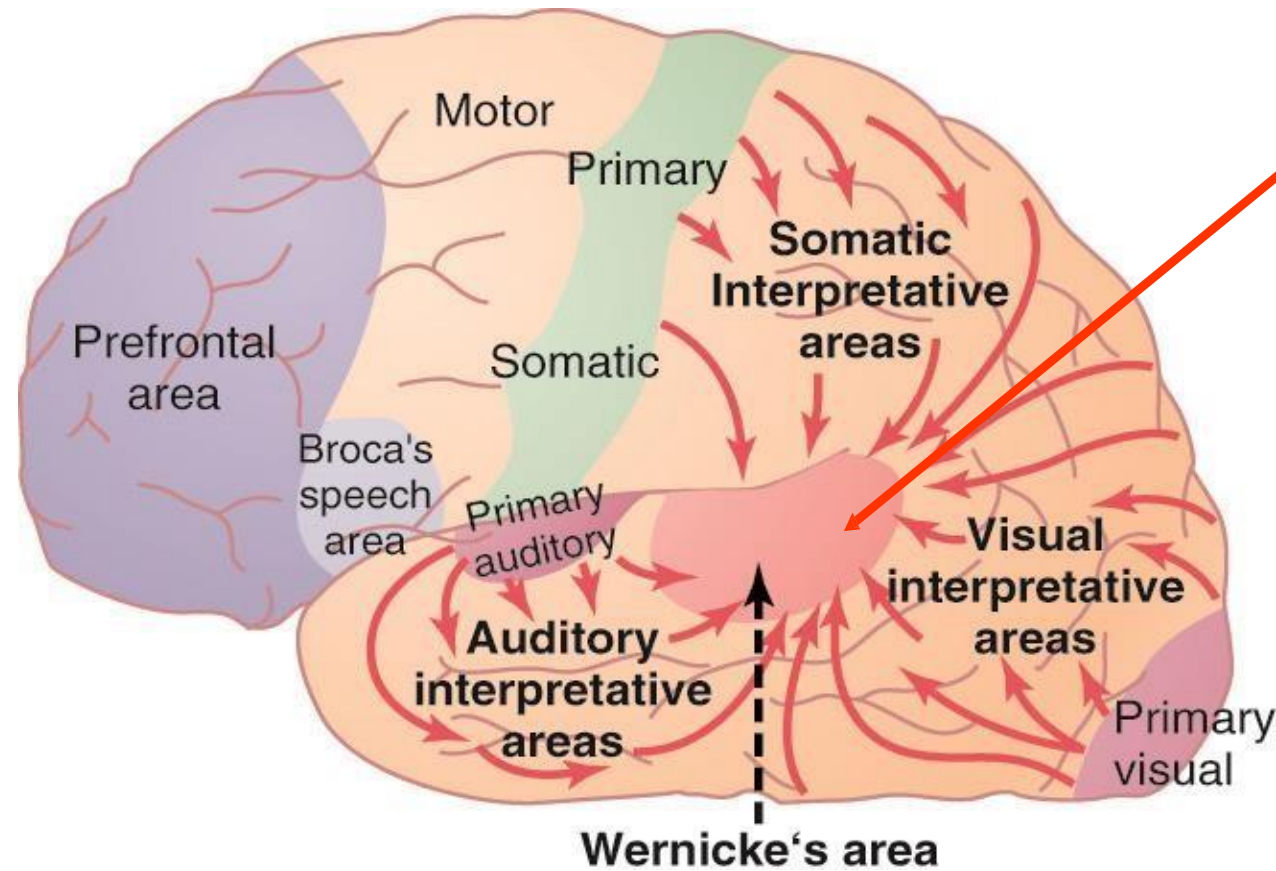


- Area 18/19 lesion → can't explain what you see → visual agnosia. For the language part, it would be word blindness (alexia), where you can see the words but they don't make sense.

Speaking a written word



Sensory Aspects of Communication



Wernicke's aphasia

Destruction of the visual and auditory association areas results in an inability to understand the written or spoken word.

Sensory association area → located posterior to the postcentral gyrus → responsible for interpretation of tactile sensations. Lesion there causes inability to recognize objects by touch (Amorphosynthesis). So you can still perceive touch, but you cannot identify what the object is.

Motor Aspects of 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.

Function of the Corpus Callosum

- connects the two hemispheres and allows transfer of information
- interruption of these fibers can lead to bizarre types of anomalies
 - dominant hemisphere understands spoken word
 - non dominant hemisphere understands written word and can elicit motor response without dominant side knowing why response was performed
- If the Corpus Callosum is cut, it can cause Split-brain syndrome, where communication between the hemispheres is lost. As a result, each hemisphere processes information independently rather than true loss of coordination. For example, the left hand may perform actions that the right hand (and the language-dominant left hemisphere) is unaware of, because information can no longer cross between hemispheres.

Thoughts 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, **cause it's not one area responsible for thinking but many.**
 - However, depth of thought and level of awareness may be less.
- **The Basal ganglia have multiple functional circuits, not just motor. For example, the Caudate nucleus has cognitive functions due to its connections with the association (prefrontal) cortex, whereas the Putamen is primarily involved in motor control and movement patterns. Therefore, the basal ganglia are not purely motor, and parts of them also contribute to thinking and executive functions, so it's not limited to one area.**

Memory and learning

- Learning is acquiring new sensory information or motor skills, **on the contrary, memory is about recalling or retrieving what you've learned.**
- Change in the capability of synaptic transmission from neuron to neuron as a result of prior stimulation.
- Memory trace is a specific pattern or pathway of signal transmission.
- Once established they can be activated by the thinking mind to reproduce the pattern and thus the memory.
Remembering something and where that piece of information will go in the brain (to what type of memory) depends heavily on how important that piece of information is.

3 Types of Memory

- immediate memory
 - lasts for seconds or minutes (remembering 10 digit phone number or a stranger's name)
- short-term memory
 - lasts for days to weeks
- long-term memory
 - lasts for years or for a lifetime, such as personal information (your name, your parents' names, etc.).

For short-term memory, neuronal circuits are transiently activated, and neurotransmitters are briefly released, producing temporary changes in synaptic activity. These neurotransmitters are then rapidly cleared by enzymatic degradation, reuptake, or diffusion, leading to the short duration of the memory trace.

An example of short-term memory can be exams. The capacity in the brain for it, is limited, so if the information isn't stored in your long term memory it will end up being permanently removed. The advantage of it is the quick retrieval of information.

Long term memory is formed mostly through:

Structural changes -> by forming new synapses or strengthening existing synapses. It can also happen through formation of new neurons (neurogenesis), but this is mostly limited to the hippocampus, and most of the mechanisms relating to long term memory rely on strengthening or modifying existing neural circuits.

Functional changes -> this is specifically for consolidation of memory (turning short term memory into long term memory), and it involves gene expression where certain transcription factors will be activated, leading to the synthesis of new proteins.

Mechanism of Memory

- Immediate memory may result from synaptic potentiation through the accumulation of calcium in the presynaptic membrane.
 - would promote neurotransmitter release
- Short-term memory may result from a temporary physical or chemical change in the pre- or postsynaptic membrane.

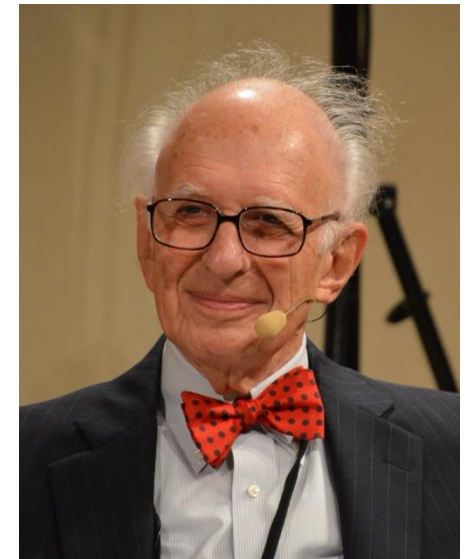
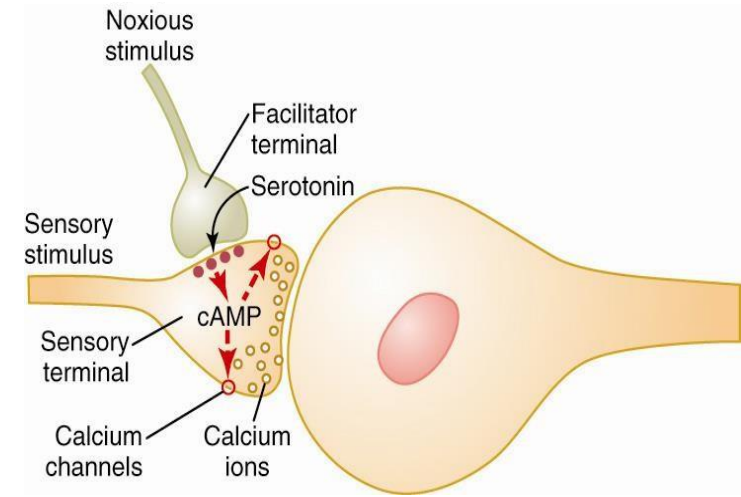
Cellular Basis for Memory

- repetitive stimulation causes a progressive decline in sensitivity called *habituation*
- Habituation is a decrease in response to a repeated harmless or irrelevant stimulus.
- results from progressive decline in the number of active calcium channels
- less calcium entry less transmitter released
- stimulation of facilitator terminal prevents habituation

There are two synaptic inputs. One comes from a **sensory** (input) neuron, which terminates on a postsynaptic neuron. The second is an **axo-axonic** synapse from a facilitator interneuron that acts on the presynaptic terminal of the sensory neuron.

If only the sensory neuron is active while the facilitator input is inactive, synaptic transmission is initially strong but gradually decreases with repeated stimulation due to reduced neurotransmitter release. This process is called **Habituation**, and it allows the nervous system to ignore irrelevant repeated stimuli.

If a **noxious stimulus** activates the facilitator interneuron, it releases **serotonin** onto the presynaptic terminal of the sensory neuron. This enhances neurotransmitter release via intracellular signaling pathways, leading to a progressively stronger synaptic response. This process is called **Sensitization**, and in its **long-term form**, it can persist for days to weeks due to gene expression and structural synaptic changes.



Molecular Basis for Memory

Transmitter activates G protein 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 prolongs the action potential which increases transmitter release.

Prolonged effects → mediated by metabotropic receptors → activation of second messenger systems, leading to slower onset but longer-lasting responses.

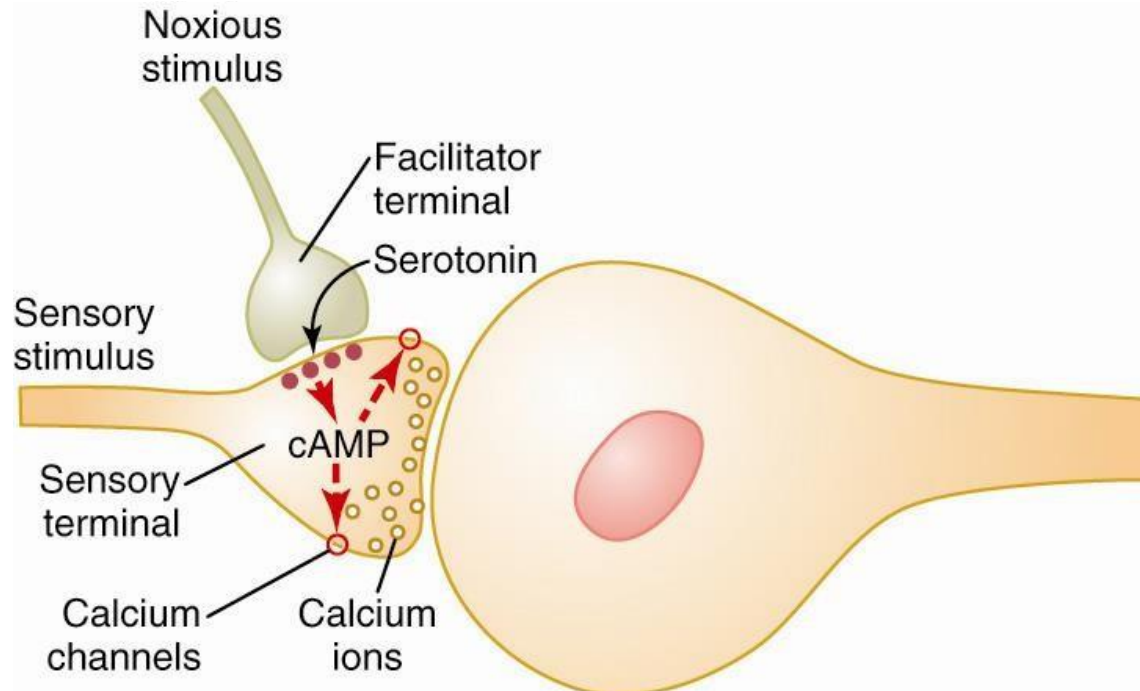


Figure 57-9; Guyton & Hall

- Release serotonin, which is a neuropeptide, causing prolonged duration of action.

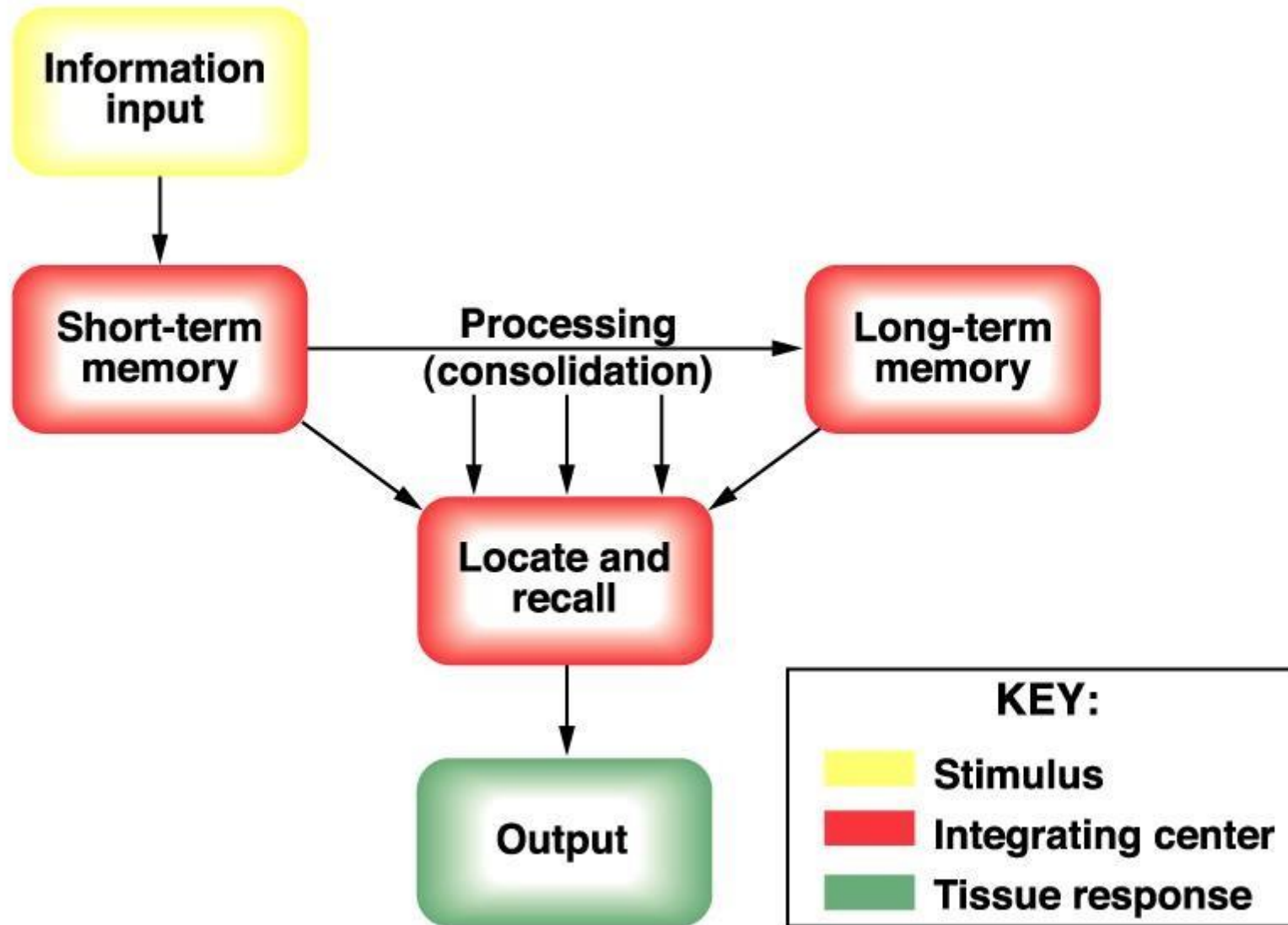
Long-Term Memory

Structural + functional

- results from a structural change in the synapse
- increase in the area for vesicular release
therefore, more transmitter is released
- during periods of inactivity the area decreases
in size
- enlargement of the release site area results
from synthesis of release site proteins

Consolidation of Memory

- converting immediate into short and long-term memory
- results from chemical, physical and anatomical changes in the synapse
- requires time, it differs from when person to another but mostly 24 hours
- interruption of the process by electrical shock or by anesthesia will prevent memory development
- rehearsal enhances consolidation
- Repetition, practice and Good filing system (organization, structure, categorization) help with memory consolidation

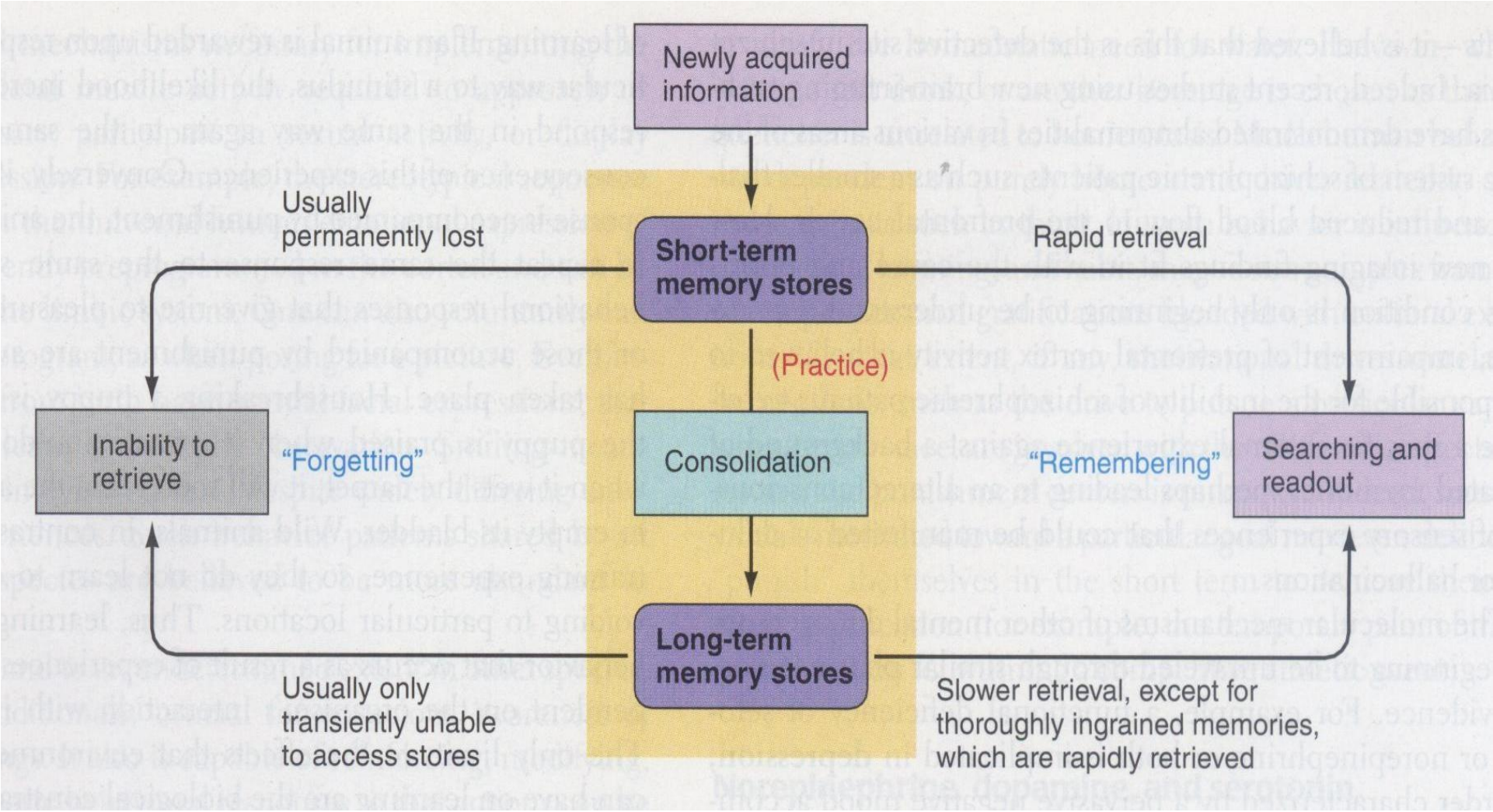


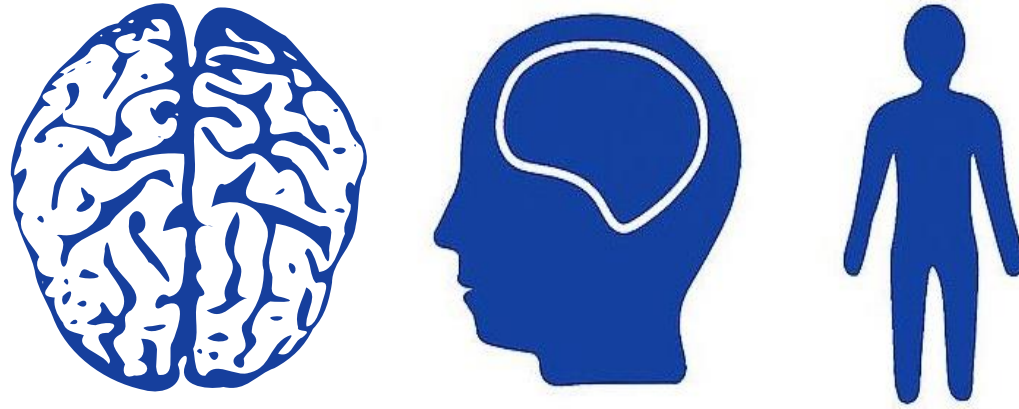
Brain Centers and Memory

- Thalamic structures are important for recalling memories.
- Damage to thalamus causes **retrograde amnesia** or the inability to recall stored experiences.
- Thalamus scans the cortex for the area and the circuit for the stored memory.
- Lesion in the **Thalamus** can cause anterograde amnesia, making it difficult to form new memories, and may also produce retrograde amnesia, especially affecting recent past memories. The extent of memory loss generally depends on the severity and location of the lesion, with recent memories more vulnerable than older ones.
- **Hippocampus** injury causes **anterograde amnesia** where you can't form new memories.

Comparison of 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	<p>weeks</p> <p>Lasts for seconds to hours</p>	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 limited	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





**PHYSIOLOGY
QUIZ
LECTURE 8**

External Resources

رسالة من الفريق العلمي

اللهم إن عمر عطية في ذمتك وحبل جوارك، فقه من فتنة القبر وعذاب النار،
أنت أهل الوفاء والحق، فاغفر له وارحمه إنك أنت الغفور الرحيم.

Additional sources:

1. <https://youtu.be/rNoRadeNrqM?si=BASuWjlWF4tR0lak>

Scan the QR code or click it for FEEDBACK



Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			