CNS Physiology

- Final Summary -

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Vestibular system:

1. What Does the Vestibular Apparatus Detect?

- Head **position in space**
- **Linear** and **rotational/angular** acceleration/deceleration

⚠️ *NOT constant motion! Only changes in movement are detected.*

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2. Utricle vs. Saccule (together are called otolithic) – Differences

Feature	Utricle	Saccule
Orientation in upright position	Horizontal	Vertical
Function	Horizontal linear acceleration (e.g. walking, car)	Vertical linear acceleration (e.g. elevator)

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3. Semicircular Canals

- Detect **rotational/angular acceleration**
- Three canals: anterior, posterior, lateral — arranged **perpendicular**
- Imp. structures:
  - **Ampulla**: Swelling at the base
  - **Crista ampullaris**: Contains **hair cells**
  - **Cupula**: Gelatinous structure where hairs are embedded

🌀 **Mechanism:**

- Head rotates → endolymph lags behind بسبب القُصور الذاتي→ bends **cupula** opposite direction
- Start of movement = depolarization
- Constant speed = no signal
- End of movement = opposite bend → signal again

⚠️ Common MCQ: *"Vestibular apparatus is stimulated when?"* → *Beginning & end of movement only*

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4. Hair Cells: Stereocilia & Kinocilium

- **Kinocilium** = the longest hair

- **Toward kinocilium = Depolarization → excitation**
- **Away from kinocilium = Hyperpolarization → inhibition**

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5. Otolithic Membrane *It's gelatinous* & Otoliths (Statoconia)

- Located in **macula** of utricle & saccule
- **Otoliths (calcium carbonate crystals)** increase membrane weight → respond to gravity
- The **pull** on stereocilia helps detect **head tilt & linear motion**

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6. Integration of Vestibular Info

Vestibular input goes to:

1. **Spinal cord** – muscle tone & posture
2. **Neck muscles** – align head/body
3. **Cerebellum** – coordination & balance
4. **Visual centers (CN III, IV, VI)** – eye movements
5. **Thalamus → Cerebral cortex (parietal lobe)** – conscious awareness

🧠 Also related:
Vestibulo-ocular reflex– eyes move opposite head direction to maintain focus

- **Slow phase**: Eyes move opposite to head
- **Fast phase**: Reset eyes in same direction

🔥 Remember : **"Nystagmus"** refers to **fast phase**

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7. Bilateral Vestibular Dysfunction

- Loss of both sides = visual compensation
- If **eyes open + slow movement → balance OK**
- If **eyes closed or rapid movement → fall over**

🧠 Know the difference between:

- **Sensory ataxia** (visual compensation helps)
- **Cerebellar ataxia** (visual input does NOT help)

🚨 **Few MCQ Examples**

"What structure detects vertical linear acceleration?"
→ **Saccule**

"During constant head rotation, semicircular canals are..."
→ **Inactive (not stimulated)**

"When stereocilia bend away from kinocilium, the result is..."
→ **Hyperpolarization**

"When moving in a car, which organ is active?"
→ **Utricle**

"Which phase defines nystagmus direction?"
→ **Fast phase**

Past21: true about the vestibular system:

A. utricle and saccule are concerned with rotational movement

B. otoliths are fluid filled sacs

C. when the head rotates stereocilia bend to the opposite direction

D. hair cells in the copula detect linear motion

E. destruction of the vestibular apparatus causes loss of balance mainly when eyes are open

Answer: C

Motor Cortex:

🧠 Motor Cortex Functional Areas : مهم

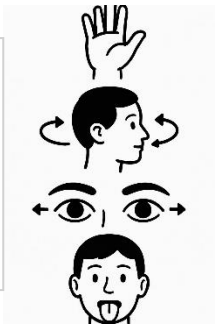
Area	Main Function	Features
Primary Motor Cortex	**Execution** of movement	Fine control (hands, face), homunculus mapping
Premotor Area	Planning movement	Uses mirror neurons هاي المعلومة ركزت عليها - anterior/posterior regions, indirect + direct pathways then to primary motor area
Supplementary Area	Bilateral coordination	Coordinates both sides, background movements e.g: catch the laptop with ur both hands.
Broca's Area	Speech production	Lesion → expressive aphasia
Contralateral Eye Movement	Voluntary eye tracking/blinking	Lesion → inability to shift gaze When the visual cortex fixes the eye on a certain object by controlling the eye movement .. This area unlocks this fixation ether by Voluntary eye movement or by blinking
Head Rotation Area	Head turning coordination	Works with eye movement area
Hand Skills Area	Complex hand movement (e.g. writing)	Lesion → motor apraxia (normal power but can't perform skilled complex movements)

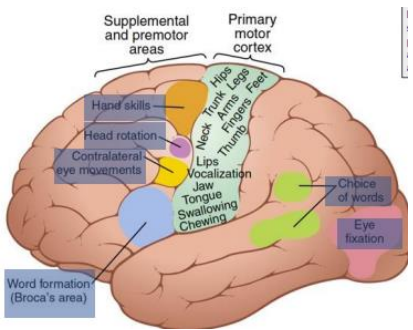
💪 Cortical Organization + Output

- **Motor Homunculus:**
  - 50% of cortex for face/hands

Past: microstimulation of which of the following would lead to contraction of individual muscle fibers.
A: Primary Motor Cortex

Pic on Rt. Shows these areas' sites, and the lt. one is only to make it easier for u to remember their sequence (last four in table)
ع الترتيب فوق بعض





    - **Each column in cotrex functions as a unit, usually stimulating a group of synergistic muscles, or just a single muscle.**

- **Cortical Columns:**
    - Each controls synergistic muscle groups
    - Pyramidal cells in Layer V
    - Functions: integration + amplification (past21: each column of the motor cortex can function as an amplifying system)

⚖️ Neuron Types

Type	Firing Pattern	Function
Dynamic	Rapid bursts	Initiate contraction
Static	Sustained firing	Maintain contraction force

🚪 Motor Pathways Overview

Pathway	Decussation Point	Function
Lateral Corticospinal	Medulla pyramids	Controls distal limb muscles
Anterior Corticospinal	Spinal cord segment	Controls trunk/proximal limbs
Corticobulbar	No spinal decussation	Head/neck muscles via CN nuclei - Motor nuclei no. = 9 why? Olfactory, Optic and Vestibulocochlear are pure sensory

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🧠 Incoming Sensory Pathways to the Motor Cortex

Source
1. Adjacent regions of the cerebral cortex
2. Opposite cerebral hemisphere via corpus callosum
3. Different thalamic nuclei

🤔 Quick Facts + MCQ Triggers

- Mirror neurons = Premotor area (posterior/anterior regions)
- Broca's area controls **speech production**, lesion → expressive aphasia
- Motor apraxia = hand skills area lesion

- Cortical movement activation = **movement**, not muscle
- Eye movement area lesion = eyes fixed
- Supplementary area = background + bilateral movement
- Decussation of most corticospinal fibers = **medulla**

Motor signals are transmitted directly from the cortex to the spinal cord through the corticospinal tract Or to the cranial nuclei In the Brain stem through corticobulbar tract and indirectly through multiple accessory pathways that involve the basal ganglia, cerebellum, and various nuclei of the brain stem

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Basal Nuclei :

🧠 Core Structures of the Basal Nuclei (Ganglia)

Component	Subdivisions / Function
Striatum	Caudate nucleus + Putamen → main input from cortex
Globus Pallidus	External (GPe) → intrinsic relay Internal (GPi) → main output to thalamus
Subthalamic Nucleus (STN)	Excitatory input (glutamate) → boosts GPi to suppress movement
Substantia Nigra	Pars Compacta (SNc) → dopamine modulation Pars Reticulata (SNr) → output partner to GPi
Thalamus (VA/VL nuclei)	Motor relay to cortex; under inhibition or disinhibition from basal nuclei but NOT part of it.

🔁 Functional Classification

Group	Includes	Role
Input nuclei	Striatum (caudate + putamen)	Receive signals from cortex
Intrinsic nuclei	GPe, STN, SNc	Process signals internally
Output nuclei	GPi الدكتورة ركزت عليها اكتر من الثانية, SNr	Project signals to thalamus → modulate movement

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⚡ Neurotransmitters in the Basal Nuclei

Neurotransmitter	Released By	Effect
GABA	Striatum, Gpi, SNr	Inhibitory (past: GABA= the main inhibitory)
Glutamate	Subthalamic nucleus *past*	Excitatory
Dopamine	Substantia nigra pars compacta (SNc)	Excitatory (D1) / Inhibitory (D2)

🧠 **Rule of thumb:** It’s GABA unless you're in STN (glutamate) or SNc (dopamine).

🧠 هاي قاعدة، فـ بالشرح الجاي اي اشي inhibitory فهو GABA تلقائيًا باستثناء ال2 المكتوبات بالسطر فوق

Motor Pathways Through Basal Nuclei: *Dr said that striatum is the one who decides which pathway the signal should go, according to signal arrived from cortex *

✅ Direct Pathway – Facilitates Movement (input → output)

1. Cortex excites Striatum (putamen , caudate)
2. Striatum inhibits (by GABA) GPi/SNr → less inhibition on thalamus
3. Thalamus activates motor cortex
4. **Result: Movement initiated**

🧠 *Disinhibition = excitation*

❌ Indirect Pathway – Inhibits Movement (input → Intrinsic→ output)

1. Cortex excites Striatum
2. Striatum inhibits GPe
3. GPe normally inhibits STN → STN now more active
4. STN excites GPi → GPi inhibits thalamus
5. **Result: Suppression of movement**

مثال من الدكتورة بالمحاضرة

basal nuclei control the balance between different types of movements and determine if this action is appropriate to do or not. Example : if I have a glass of water and a fly around it And I have 2 thoughts 1- I am thirsty and willing to drink it 2- I must move away the fly These 2 types of thoughts and the sensory information sent to my cerebral cortex then arrived to my premoter area which will plan each action (like I must do this to hold the glass and then …etc.) And send these plans to basal nuclei and then to the striatum (input) Putamen will Review the data and find these actions are contradictory so will send the first movement in the direct pathway And the other on in the indirect pathway (Previous experiences and priorities will determine which action will go direct or indirect)

🔄 Role of Dopamine – <u>Nigrostriatal Projection</u>

Pathway	Receptor	Dopamine Effect	Result
Direct	D1	Excitatory	Enhances movement
Indirect	D2	Inhibitory	Reduces inhibition

→ **Dopamine from SNc overall promotes movement** by:

- Activating direct pathway (via D1)
- Inhibiting indirect pathway (via D2)

Loss of SNc → **Parkinson's disease** symptoms:* in this disease, balance is tipped in favor of the indirect pathway*

- Bradykinesia
- Rigidity
- Tremor

Past: A 75 year old man gradually presented with left sided tremor especially at rest, and slowness of movement. On clinical examination of this patient the following is TRUE:
A: he exhibits a mask face

🧠 Functional Circuits

Circuit	Origin/Input Area	Output Target	Role
Putamen Circuit	supplementary, premotor, somatosensory areas	Primary motor cortex	Pure motor control
Caudate Circuit	Association areas of cortex, prefrontal	Supplementary/premotor cortex	Cognitive motor planning

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🌀 Basal Nuclei – Overall Functions

- Initiation & scaling of movement
- Suppression of unwanted movements
- Regulation of muscle tone (via reticular formation)
- Cognitive planning of motor behavior (esp. caudate circuit)
- Emotional & behavioral modulation (via limbic connections)

❌ Disorders

Disorder	Feature(s)	Pathophysiology
Parkinson's	Bradykinesia, tremor, rigidity	SNc degeneration → ↓ dopamine
Huntington's	Chorea (jerky, involuntary movement), mental decline	Striatal neuron death → ↓ GABA, Ach ما حكته بالتفصيل
Schizophrenia/OCD لسا الموضوع قيد البحث وما ركزت عليه كتير	Thought/emotion dysfunction	Imbalance in basal nuclei-limbic connections

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Spinal Cord & Reflexes :

🧠 Reflex Arc

Component	Function
Sensory receptor	Detects stimulus
Afferent neuron	Sends info to spinal cord
Integrating center	Usually **gray matter** in spinal cord
Efferent neuron	Alpha motor neuron
Effector	Skeletal muscle (contraction or inhibition)

Reflex arc = **fast, involuntary, unplanned**

🧬 Muscle Fiber Types

Fiber Type	Function	Innervation
Extrafusal	Main contractile muscle fibers	Alpha motor neurons
Intrafusal	Sensory role (in spindles)	Gamma motor neurons

🧠 Intrafusal Fiber Types + Afferents

Intrafusal Type	Afferents	Function
Nuclear bag	Type Ia	Detects **rapid** length change
Nuclear chain	Type Ia & Type II	Detects **slow** length change

- **Ia** = Primary, fast response
- **II** = Secondary, slow response
- **Gamma motor neurons** control **ends** of intrafusal fibers to keep spindle sensitive.

🔥 1.Stretch Reflex (Deep Tendon Reflex, don't confuse its name with the next one!)

Feature	Explanation
Type	**Monosynaptic**, **Ipsilateral**, **Dynamic (via Ia)**
Stimulus	Sudden muscle stretch (hammer tap)
Receptor	Muscle spindle
Pathway	Ia → spinal cord → alpha motor neuron → same muscle contracts
Function	Prevent overstretching
Additional phase	Type II fibers for **static, sustained contraction**
Coactivation	Alpha + gamma fire together ***past21***→ spindle remains sensitive

Reciprocal innervation: inhibition of antagonist via interneuron (also seen in tendon reflex)

🚨 2.Golgi Tendon Reflex (Tendon Reflex)

Feature	Explanation
Receptor	Golgi Tendon Organ (GTO) in tendon
Stimulus	Excess tension (muscle pulling too hard)
Reflex Type	**Polysynaptic**, **Ipsilateral**, **Inhibitory**
Pathway	GTO → afferent → inhibitory interneuron → inhibits alpha motor
Effect	**Muscle relaxation** (protection from overcontraction)
Reciprocal effect	**Antagonist contracts** (for balance/stability)

🔥 Stretch vs. Tendon Reflex – Compare them Always! مقارنة بين الأول والثاني

Feature	Stretch Reflex	Tendon Reflex

Receptor	Muscle spindle	Golgi tendon organ
Stimulus	Stretch	Tension
Response	Same muscle contracts	Same muscle relaxes
Synapse type	Monosynaptic	Polysynaptic
Protection from	Overstretch	Overcontraction
Reciprocal?	Yes	Yes

🚩 3. Flexor (Withdrawal) Reflex

Feature	Explanation
Trigger	Pain or skin stimuli
Reflex type	**Polysynaptic**, **Ipsilateral**, **Reciprocal, Intersegmental** = يقدر يحرّك أكثر من عضلة من أكثر من مستوى فقري
Function	Pull away from painful stimulus
Interneurons	Heavily involved

🚩 Crossed Extensor Reflex تابع لِما قبله

Feature	Explanation
Side	**Contralateral**
Function	Maintains balance on opposite limb
Reflex type	Polysynaptic, intersegmental, after-discharge

🧠 Might be imp. for MCQs

- **Ia fibers**: fast, sudden stretch
- **II fibers**: slow, sustained
- **Alpha**: extrafusal
- **Gamma**: intrafusal
- **Muscle spindle**: stretch detection
- **GTO**: tension detection
- **Monosynaptic = Stretch**, **Polysynaptic = Tendon, Flexor**

- **Ipsilateral**: all except **Crossed Extensor = Contralateral**

Brainstem – (Midbrain, Pons, Medulla) & reticular formation

🧠 **Brainstem :**

- **Main Parts: Midbrain, Pons, Medulla Oblongata**
- **All ascending/descending tracts between brain & spinal cord pass through the brainstem**
- **Contains cranial nerve nuclei, vital reflex centers (CV, respiratory), and reticular formation**

🧬 **Medulla Oblongata**

Structure/Nucleus	Function
Pyramids	**Contain corticospinal tracts (motor)**
Olives	**Contain inferior olivary nucleus → Sends input to cerebellum**
Gracile & Cuneate nuclei	**Relay touch, pressure, vibration, conscious proprioception**
CV Center	**Controls HR, vessel diameter**
Respiratory Center	**Controls basic respiratory rhythm**
Reflex Centers	**Vomiting, swallowing, coughing, hiccuping**
Cranial Nerve Nuclei	**Includes nuclei for multiple cranial nerves**

Injury to the medulla is life-threatening

🧱 **Pons**

Region	Function
Dorsal	**Contains cranial nerve nuclei, ascending & descending tracts**
Ventral	**Pontine nuclei: relay motor info between cerebral cortex ↔ cerebellum**

Pontine relay supports motor coordination (voluntary movement fine-tuning)

🧠 **Midbrain**

Structure	Function
Cerebral Peduncles	**Contain corticospinal, corticobulbar, corticopontine tracts**

Substantia Nigra	**Dopamine release; part of basal nuclei (damaged in Parkinson's)**
Red Nucleus	**Receives motor cortex input → relays to cerebellum, rubrospinal tract**
Tectum	**Contains superior & inferior colliculi**
Superior Colliculi	**Visual reflexes: eye/head movements in response to visual stimuli**
Inferior Colliculi	**Auditory reflexes: startle reaction to loud sounds**
Periaqueductal Gray	**Part of analgesic (pain suppression) system**

🔁 Reticular Formation (RF)

- **Netlike structure spanning spinal cord → brainstem → diencephalon**
- **Reticular Activating System (RAS) = ascending RF controlling:**
  - **Consciousness, attention, alertness**
  - **Filters sensory input → prevents sensory overload**
- **Descending RF controls:**
  - **Muscle tone, HR, BP, respiration**

RAS has no olfactory input → explains why gas leaks don't wake sleeping people

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🔄 Reticular Nuclei: Excitatory vs Inhibitory

Nucleus Type	Fx.
Pontine Reticular Nuclei	**Excite antigravity muscles (via anterior column)**
Medullary Reticular Nuclei	**Inhibit same muscles (via lateral column)**

Past: rigidity of the axial and antigravity muscles when cortical control over the brain stem is integrated (decerebrate) is due to:

Over activity of pontine reticulospinal tract

- **Pontine nuclei receive input from vestibular & cerebellar nuclei → strong excitatory tone**
- **Medullary nuclei are activated by cortex & rubrospinal tract → inhibition when needed**
- **Together: create balance for postural tone & movement**

⚖️ Vestibular Nuclei

- **Support pontine reticular system by enhancing antigravity muscle tone**
- **Control equilibrium via input from vestibular apparatus (ear) & cerebellum**
- **Involved in eye movement control via medial longitudinal fasciculus**
- **Some fibers go directly to reticular formation, cerebellum (fastigial & flocculonodular lobes)**

Injury → impaired posture, balance, and coordination during rapid movement

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🔴 **Motor Function – Red Nucleus & Backup Tract**

- **Red nucleus (midbrain) receives motor cortex input → acts via corticorubrospinal tract**
- **Functions as backup for corticospinal tract (main motor pathway)**
- **Especially supports rapid movement control, less fine finger motion**

This backup system is part of the lateral motor system

Diencephalon & Limbic System :

🔄 **Diencephalon Overview:**

Structure	Fx.
Thalamus	Relay for **sensory**, **motor**, **pain**, **emotions**, **consciousness**
Hypothalamus	**Homeostasis** controller: ANS, hormones, behavior, emotions
Epithalamus	Pineal gland (melatonin) + Habenular nuclei (olfaction/emotion link)

💡 **Thalamus – Major Nuclei مهم**

Nucleus	Fx.
Ventroposterior (basal)	Somatic sensation to cortex
Lateral geniculate body	Vision relay
Medial geniculate body	Auditory relay
Intralaminar nuclei	Pain + diffuse input (from reticular formation)→ consciousness
Ventroanterior + Ventrolateral	Motor relay (from cerebellum & basal nuclei)
Anterior, medial, lateral	Emotion (connects to limbic system)

🔮 **Epithalamus**

- **Pineal gland**: Secretes **melatonin** which controls circadian rhythm (light-dark cycle)
- **Habenular nuclei**: Connect **olfaction** to **emotions**, especially via memory and smell.

👀 **Melatonin Inhibition by Light – Pathway**

1. Light hits retina → melanopsin-containing cells detect it

2. Signal sent via **retinohypothalamic tract** to SCN (suprachiasmatic nucleus)
3. SCN inhibits pineal gland via GABA release → **melatonin drops**
4. At night, SCN stops inhibition → melatonin rises → sleep induced

SCN = biological clock → coordinates sleep-wake, hormones, metabolism, body temp

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🌊 Hypothalamus – Big Boss of Homeostasis

Function Category	Roles
Autonomic regulation	Controls ANS → HR, BP, GI motility, etc.
Temperature control	Body thermostat, maintains set point
Hunger & Satiety	Feeding center (hunger), Satiety center (fullness)
Thirst regulation	Triggered by **high osmolarity** → thirst sensation
Hormonal control	→ Releasing hormones (to ant. pituitary)
Posterior pituitary hormones	→ Oxytocin + ADH *vasopressin* (from post. pituitary)
Circadian rhythm	→ SCN synchronizes with light/dark to regulate pineal melatonin
Emotions & behavior	→ Rage, sexual arousal, pain/pleasure patterns (via limbic links) مثلًا مراكز الجوع مرتبطة مع مشاعر الغضب ومراكز الشبع مع مشاعر الراحة والهدوء

💔 Limbic System – Emotional Brain

Component	Fx.
Hypothalamus	Central integrator: emotions, homeostasis, endocrine links
Hippocampus	**Memory consolidation**, learning, context for emotion
Amygdala	Fear, anxiety, appropriate behavioral response
Paraolfactory area	Smell-emotion link
Anterior thalamic nuclei	Emotional relay to cortex

Limbic cortex : Connects cortex to limbic core, coordinates **behavior + cognition**

Past21: A lesion in which of the following will cause memory impairment and olfactory and other types of hallucination and sense of fear? Hippocampus

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🤔 Reward vs Punishment System

- **Reward centers (hypothalamus)** → Weak stimulation = pleasure
- **Punishment centers** → Stronger stimuli = rage, fear, aversion
- Located in **periventricular hypothalamus(mainly)**, **amygdala**, **hippocampus**

Punishment can inhibit reward → Fear overrides pleasure

📊 Learning & Memory: Habituation vs Reinforcement:

Process	Outcome
Habituation	Repeated neutral stimuli (does not elicit a sense of either reward or punishment) = reduced brain response
Reinforcement	Stimuli with emotional value (reward/punish) = stronger cortical encoding

Motivation requires emotional tag → Otherwise, info is ignored

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🌟 Hippocampus

- Converts **short-term to long-term** memory → Damage = **anterograde amnesia**
- Sends output via **fornix** to thalamus, hypothalamus, limbic system
- It's hyperexcitable so Can trigger seizure focus→ **hallucinations**

😱 Amygdala & Fear Response

- Processes fear, anxiety, danger assessment
- If damaged → no fear = **risky behavior**
- Sends signals to: hypothalamus, thalamus, hippocampus, cortex

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Memory :

🤔 Thought & Learning

- Thought involves **simultaneous signaling** between cerebral cortex, thalamus, limbic system, and reticular formation.
- Learning occurs via **observation** (language, social cues) or **experimentation** (trial & error).

⏳ Memory Types by Duration

Type	Duration	Description
Short-term	Seconds to minutes	Temporary storage of information (e.g phone number)
Intermediate	Days to weeks	Transient memory, fades unless reinforced
Long-term	Weeks to lifetime	Stable storage via structural synaptic changes
Working memory *defect in dementia pts.*	Active while working	Used in reasoning, problem-solving (prefrontal cortex)

🌟 Memory Types by Content:

Type	Description	Brain Area
Declarative	Facts, events (explicit, verbalizable)	Hippocampus, cortex
Procedural	Skills, habits (implicit)	Cerebellum

🧠 Memory Trace Formation

- **Memory trace** = neural pattern formed by repeated activity.
- Short-term → chemical change (↑ Ca^{2+} influx → more NT release).
- Long-term → structural changes in synapses.

🔁 Transition from Short-term to Long-term involves:

1. **↑ Presynaptic terminals**
2. **↑ Postsynaptic receptors**
3. **↑ Dendritic branching**
4. **↑ Vesicle release zones**

These structural changes make synaptic transmission more effective and stable.

Past: short term memories can involve all of the following processes except: regulation of gene expression

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🧬 Synaptic Mechanisms Summary

Feature	Short-Term Memory	Long-Term Memory
Duration	Seconds–minutes	Days–lifetime
Basis	Chemical changes	Structural/synaptic remodeling
Neurotransmission	↑ Ca^{2+} influx, ↑ NT release	↑ Dendrites, receptors, synaptic terminals
Reversibility	Easily lost	Retained with reinforcement

🧪 Molecular Events

Process	Mechanism
Habituation	↓ Ca^{2+} entry → ↓ NT release → weaker synapse
Sensitization	Emotional/reward → ↑ NT release via presynaptic facilitation
Consolidation * means conversion from short to long term memory*	Structural changes: more synapses, receptors, branches = stronger trace

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📚 Factors Promoting Consolidation:

Factor	Role
Repetition	Reinforces pathways
Emotion	Tags memory as important via limbic system
Context	Sensory + situational cues enhance encoding

Hippocampus: critical for consolidation and spatial memory.
Dorsomedial thalamus: prioritizes which inputs become memories.

🧩 Brain Regions & Memory

Region	Role
Hippocampus	Short → long-term conversion; damaged in anterograde amnesia
Dorsomedial Thalamus	Retrieval & prioritization; damaged in retrograde amnesia
Cerebellum	Procedural memory (skills, habits)

🧠 Amnesia Types 🧠

Type	Loss of...	Caused by...
Anterograde Amnesia	New memories post-injury	Hippocampus damage
Retrograde Amnesia	Memories before injury	Dorsomedial thalamus

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🏆 Motor vs Verbal Memory

- **Hippocampal lesions**: impair declarative memory (facts, names)
- **While Procedural memory** (e.g riding bike, swimming) remains intact
- Skill-based tasks depend on cerebellum

- Our brain organize memories in categories to make remembering easier.

Cerebral Cortex

General Cortex Concepts

- The **cerebral cortex** is the highest, most complex brain region.
- Needs activation from **reticular formation** + **thalamic input** to function.
- Cortex + thalamus = **Thalamocortical system**: they operate as one unit.
- **Damage to both cortex + thalamus** = more severe than cortex alone.

😄 Thalamocortical Connections

Thalamic Nucleus	Cortical Target
Medial Geniculate Body	Auditory Cortex
Lateral Geniculate Body	Visual Cortex
Ventroposterior Nucleus	Somatosensory Cortex

- Cutting these connections → severe cortical functional loss.

🔄 Types of Cortical Areas

Area Type	Fx Description
Primary areas	Directly receive/send sensory/motor info (e.g primary motor/sensory/visual/auditory)
Secondary areas	Interpret info from primary areas (e.g orientation, color, movement pattern)
Association areas	Complex integration from multiple sources → cognition, language, personality

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📏 Major Association Areas

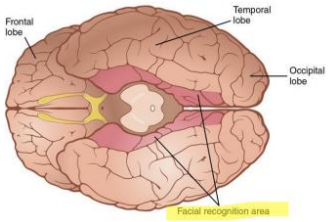
Association Area	Functions
Parieto-occipito-temporal	Spatial awareness, object naming, face recognition
Prefrontal	Planning, working memory, behavior, logic
Limbic Association	Emotion-related processing

🌎 Parieto-Occipito-Temporal Sub-Areas:

1. **Spatial Coordinates Area**

- Integrates input from: **Visual cortex** + **Somatosensory (proprioception)**
- Purpose: Body & environmental orientation

2. **Object Naming Area**
   - Auditory input = name
   - Visual input = object identity
   - Needs: **Auditory, Visual, Language areas, Memory**
3. **Face Recognition Area**
   - Connected to **Visual cortex** + **Limbic system** (emotional response to faces)



🔊 Language Areas

Area	Fx	Damage =
Broca's	Motor expression (word formation) by exciting simultaneously the laryngeal muscles, respiratory muscles, and muscles of the mouth.	Expressive aphasia
Wernicke's	Comprehension, converts words into meaning or thought ***past***	Receptive (sensory) aphasia ***past***, loss of intellectual function
Angular gyrus	Links visual info to language (reading/writing)	Alexia (Can't read) , dyslexia, agraphia بكون الطفل لو سمع الكلمة بفهمها بس لو شافها وقرأها ما بقدر يفهمها

😄 Language Processing Pathways

- **Auditory input**: Hearing → Auditory Cortex → Wernicke's → Broca's → Primary Motor Cortex → Speech
- **Visual input**: Reading → Visual Cortex → Angular Gyrus → Wernicke's → Broca's → Motor Cortex
- **Angular gyrus** is the extra step for visual language مهمة

🤔 Hemispheric Specialization

Hemisphere	Function Focus
Left (Dominant)	Language مهم , logic, math
Right	Music, art, spatial reasoning, body language

If a patient comes to you with a right motor deficit, you should consider a lesion in language area (left hemispheres).

past : Aphasia + hemiplegia → Lt. hemisphere

Corpus callosum connects hemispheres → synchronizes sensory, motor, and emotional info.

- **Lesion**: Split-brain syndrome → disconnect in memory, language, coordination (many symptoms can occur)

- **Anterior commissure**: Emotion link across hemispheres (especially limbic), so not be affected in Split-brain syndrome.
  Remember that in Anatomy it was told that its fx is olfaction & acute pain

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🧠 Prefrontal Association Cortex

Function Category	Role
Working memory	Short-term storage of active thoughts
Planning	Future action, task organization, motor coordination
Behavior & Emotion	Personality, inhibition, social responses
Decision-making	Logic, attention, multitasking ability

Damage → disinhibition, poor planning, change in mood& personality, inability to finish complex tasks

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🚩 Prefrontal Damage Effects (e.g: lobotomy pts.) الدكتورة ما قرأتهم كلهم بس مذكورين بالسلايدات

1. Can't solve complex problems
2. Fail to plan/sequence multi-step tasks
3. Poor parallel processing
4. ↓ aggression, ↓ motivation
5. Inappropriate social responses **جرأة، اندفاع، تصرف غير محسوب**
6. Mood instability, scattered thoughts
7. Motor patterns intact but purposeless

Cerebellum

🧠 Functions of the Cerebellum

- Timing of muscle activity, smooth coordination of movement
- Adjusts muscle force to match load changes
- Coordinates agonist/antagonist switching
- Makes real-time corrections to movement
- Assists cortex in sequencing future movements
- **Silent area**: stimulation doesn't cause movement, but damage causes severe incoordination

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🚑 Functional Anatomy

Region	Function / Representation
Vermis	Axial muscles (spine, shoulders, hips)
Intermediate	Limbs (upper/lower), face
Lateral zones	No body map; connected to planning centers (motor cortex)

- Deep Nuclei:
    - **Fastigial** → vermis
    - **Interposed** → intermediate
    - **Dentate** → lateral

🚪 Input Pathways

Tract	Source & Function
Corticopontocerebellar	From cortex (premotor, somatosensory, motor) → via pons
Reticulocerebellar	From reticular formation → motor state feedback
Vestibulocerebellar	From vestibular apparatus or nuclei → head position
Olivocerebellar	From inferior olive → integrates cortex, basal ganglia, RF, spinal cord
Dorsal spinocerebellar	Muscle spindle, GTOs, joints → body position, contraction, force sense
Ventral spinocerebellar	"Efference copy" of motor commands from anterior horn → actual command sent

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🧰 Neural Circuit Core

- Each cerebellar unit = **Purkinje cell + corresponding deep nuclear cell**
- Inputs:
    - **Mossy fibers** → widespread from brainstem, spinal cord <u>except</u> from inferior olive
    - **Climbing fibers** → only from inferior olive

Climbing Fibers

- <u>Directly</u> excite deep cerebellar nuclei and Purkinje cells in the molecular layer. *Past21*
- Subsequently, they induce a complex spike in Purkinje cells, characterized by a <u>strong initial spike followed by a period of weaker</u> <u>(NOT the opposite, this is PP question)</u>, delayed inhibition.

Mossy Fibers

- Synapse on **granule cells** → granule cells → parallel fibers → weakly excite many Purkinje cells
- Trigger **simple spikes** (weak, brief)

All inputs → excite deep nuclei directly, then Purkinje inhibition modulates it

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🐂 Deep Cerebellar Nuclei

Nucleus	Connected Zone	Function
Fastigial	Vermis, flocculonodular	Equilibrium, posture via vestibular system + RF past21: Equilibrium disturbances during rapid movement are associated with lesion in: Folocculonodular lobe
Dentate	Lateral zone	Plans sequential movement → cortex via thalamus
Interposed	Intermediate	Coordinates distal limb movement → cortex, red nucleus, basal nuclei

- **All outputs from cerebellum pass through deep nuclei**
- Output = excitation (deep nuclei) → fine-tuned by delayed Purkinje inhibition

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📁 Learning & Adaptation

- Cerebellum **learns motor timing** via repeated feedback correction
- **Delay line inhibition** → prevents overshooting & oscillation
- **Basket + stellate cells** → lateral inhibition → sharpen signals

Both Purkinje & deep nuclei fire continuously → can be modulated

Past: The **motor** cortex controls the motor orders while the **cerebellum** compares the intended movement with actual movement for the improvement of movement skill

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↻ Turn-on / Turn-off System الدكتورة ما ركزت عليه ابدا بس احتياط يعني

Phase	Action
Start of movement	Agonist ON, Antagonist OFF → sharp initiation
End of movement	Agonist OFF, Antagonist ON → smooth stop

- Controlled precisely by cerebellum for coordination & stability

Past: A 70 year old man with a history of hypertension went to work and had sudden onset of nausea and vomiting. He was taken to ER, where his exam was notable for slurred speech (dysarthria), and dysmetria on finger-to-nose testing on the left side. His gait was normal with normal equilibrium. Where is the lesion?
A: **left cerebellar hemisphere**

🚀 Functional Subdivisions

Subdivision	Zone/Nucleus	Role
Vestibulocerebellum	Vermis + Flocculonodular	Balance, posture, rapid correction using vestibular input
Spinocerebellum	Intermediate + Interposed	Coordinates distal limbs via sensory feedback + motor plan بقارن بين الحركة المطلوبة والحركة يلي تمّ تنفيذها
Cerebrocerebellum	Lateral + Dentate	Plans sequential complex movement with cortex & timing

Last Note: Impaired rapid alternating movements = cerebellar lesion,
Not basal ganglia ← it's p.p question .. Also, lesion in cerebellum causes Dysmetria (impaired coordination) & Dysarthria (slurred/slow speech due to poor muscle control)

Last Lec :) | Brain Activity, Sleep, EEG & Epilepsy .

🧠 Brain Activation Systems:

System	Mechanism
1.Reticular Formation	Direct neuronal activation from brainstem (pons & mesencephalon)
2.Neurohormonal System	Neurotransmitters modulate brain regions (excitatory/inhibitory)

🏋️ 1. Reticular Formation :

- **Excitatory Area (Bulboreticular)**
  - Activates cortex via **thalamus** (fast → specific, slow → diffuse)
  - Increases **antigravity muscle tone** via spinal cord
  - Driven by **sensory input**, especially **pain**
  - Receives **positive feedback** from cortex → amplification loop
- **Inhibitory Area**
  - Located inferiorly, uses **inhibitory NTs** like serotonin
  - Suppresses excitatory area → promotes **sleep**

Pain is the strongest activator of the reticular system.

🔶 Thalamus Role

- Every cortical region is linked to a specific thalamic area
- Thalamus and cortex work as a **reverberating circuit**
- Important for maintaining **conscious cortical activity**

🔍 2. Neurohormonal Systems

Neurotransmitter	Source Location	Effect on Brain Activity

Serotonin	Raphe nuclei *past*	Inhibitory → promotes **sleep**, ↓ pain
Acetylcholine	Gigantocellular RF neurons	Excitatory → promotes **wakefulness**
Dopamine	Substantia nigra → cortex/limbic	Excitatory or inhibitory (depends on receptor)

- They Control brain states for **minutes to hours**
- Each system affects different **qualities of function**

🏠 Sleep Basics

State	Definition
Sleep	Reversible unconsciousness, responsive to sensory input
Coma	Non-reversible unconsciousness, **not** responsive to sensory stimuli

- Sleep is regulated by RF, hypothalamus, nucleus tractus solitarius, and raphe nuclei.

😴 Types of Sleep

Type	Description
REM	Active sleep (not restful), dreams, ↑ metabolism, irregular HR/resp, ↓ muscle tone
NREM	Deep restful sleep, ↓ vegetative function, ↓ memory formation

- REM = 25% of sleep (5–30 min every 90 min)
- NREM = Majority of sleep time (especially first hours)
- As the person becomes more rested through the night, the durations of the REM bouts increase

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🧠 EEG & Brain Waves

Wave Type	Description
Alpha	Relaxed, awake state (eyes closed)
Beta	Active mental work, alertness, desynchronized waves
Theta	Children or adults under emotional stress/frustration
Delta	Deep sleep, infancy, or pathological brain states

- This electrical activity that's recorded by the EEG reflects the number of neurons that fire **synchronously**, not the total degree of neurons that are active مهمة
- **Alpha → Beta**: when opening eyes or focusing (occipital shift)
- **REM sleep** EEG resembles alert wakefulness → paradoxical

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🔍 EEG & Sleep Stages Summary

Stage	EEG Pattern Description
Wake (quiet)	Alpha waves (relaxed)
Wake (alert)	**Beta waves** (active, alert, **REM-like*past21***)
NREM Stage 1	Transition stage, alpha-like waves/spindles
NREM Stage 2	Slower frequency, theta waves emerge
NREM 3 & 4	Deep sleep → Delta waves dominate

⚡ Epilepsy Overview

- Caused by **uncontrolled, excessive neuronal activity**
- Triggers: electrolyte imbalance, hypoglycemia, drugs, infection, trauma

Type	Description
Focal	Localized, may or may not affect consciousness
Generalized	Bilateral, spreads via thalamocortical loops

⚡ Focal Seizures

Type	Features
Simple Partial	No loss of consciousness; may include aura (sensation)
Complex Partial	Impaired consciousness, repetitive strange movements
Postictal Period	Phase after seizure before full recovery

🚗 Generalized Seizures

اللهم عَافِ كل مبتلى واشفِ كل مريض يتألم من مرض جسدي أو نفسي أو علة من العلل لا يقدر على شفائها إلا أنت، آمين يارب العالمين.

- لا تنسوا أهلنا في غزة من الدعاء . .

1. **Tonic-Clonic (Grand Mal)**
    - Sudden LOC, tonic stiffness → clonic jerks
    - Can be triggered by emotion, fever, drugs, sensory overload
    - May include cyanosis, tongue biting, postictal drowsiness
2. **Absence (Petit Mal)**
    - Brief LOC (3–30s), blank stare, eyelid blinking then they return to their normal activity
    - Seen in children/adolescents , and it involves **thalamocortical system**
    - Often missed unless carefully observed يعني صعب تمييز هاد النوع فـ لازم الأهل ينتبهوا

The End