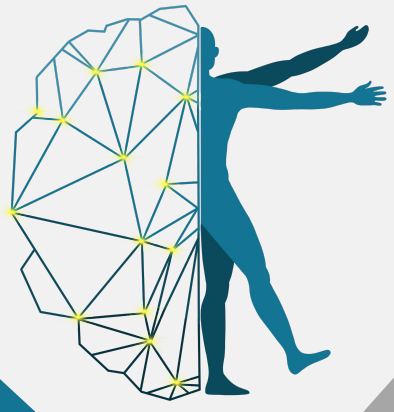
Physiology Team 439

MED439
KING SAUD UNIVERSITY



Dr.Salah's Final Exam Revision

Dr.Faten & Dr.laila

DISCLAIMER!

This file is completely a personal effort, it is not by any mean a primary source to study and prepare for the exam, study your slides or the team work first then check this file, some points I want to clarify:

- I suggest opening this file hand by hand while reading the lectures and focusing on the important things mentioned by doctor to have a full picture and imagination.

- I tried to do all my best to make this file summarized as possible, but the revision was almost an audio and I could not be sure %100 what slides were skipped or not, so I wrote what had been said and what is important for the exam according to what the doctor said.

- Any slide number mentioned in this file it would be according to Dr.Salah's slides

 means highly potential to be SAQs lecture

Good luck

Color Vision

R

Cone system	Blue cone system	Green cone system	Red cone system
Pigment	**S** pigment Blue sensation pigment	**M** pigment Green sensation pigment	**L** pigment Red sensation pigment
Wavelength	SHORT wavelength (**440** nm)	MIDDLE wavelength (**535** nm)	LARGE wavelength more than (**535** nm)- (**565** nm)
Color sensation	senses the **blue color**	senses the **green color & less to yellow**, & absorb light at the green portion.	senses the **red & yellow color** & absorb light at the red portion.

Color	Cone stimulation percentage **imp**	Ratio
Orange	**99% of red cones** **42% of green cones** **0% of blue cones**	(99:42:0)
Yellow	**50% of red cones** **50% of green cones** **0% of blue cones**	(50:50:0)* (83:83:0)*
Blue	**0% of red cones** **0% of green cones** **97% of blue cones**	(0:0:97)
White	Equal stimulation	

Red-Green Blindness

- **Green** & **Red** cones see color between the wavelengths of **525-675 nm**.
- If either of these cones are absent, the person won't be able to distinguish between: **Red Green -Yellow-Orange.** and he can not distinguish red from green
- (primary colors) so called (**Red-Green blindness**).
- It's **X-Linked (recessive disease*)** disease that is transmitted
- by females to their male children (sons*), never occurs in females
- because they have 2X Chromosomes.
- 8% of female are carrier*.

Have or carry the disease

Male

Males have one x & one y chromosome so **If this one x chromosome miss the gene for color vision , he will get red-green color blindness**(their gene is on x chromosome).

Female

Female show the disease only if **both of their X chromosomes lack the gene.**

Female from color-blind fathers are only **carriers and transmit the disease to half (1/2) of their children.**

Color Vision

R

- Individuals classified into three groups:

A: Trichromats (Completely normal or has weakness in one cone, e.g Protanomaly)
B: Dichromats (Loss of one of primary cones)
C: Monochromat (Individual has only one primary cone)

Nopia = blindness/total loss
Nomaly =weakness
Nomaly: يشوف الغامق بس ما يشوف الفاتح كويس

Types Of Color Blindness MCQ

Trichromats	have **3** cone pigments normal or have slight weakness in detecting red or green or blue color.* See the 3 primary colors.*	**Protanomaly**	If only weakness in red color vision
		Deuteranomaly	If only weakness in green color vision
		Tritanomaly	If only weakness in blue color vision
Dichromats	have only **2** cone pigments systems only so he is completely blind to red or green or blue, (so they may have protanopia, deuteranopia, or tritanopia) they get color by mixing only 2 of the primary colors.* Blind to one primary color.*	**Protanopia (red-blindness)**	no red cones system so person has shortened spectrum wave length*
		Deuteranopia (green-blindnes)	no green cones system so person see only **long & short** wave length*
		Tritanopia (blue-blindness)	no blue cones system*
Monochromats	have only **one** cone system or loss of all so see only black or grey or have no color perception.* Have only one color pigment.*		

★ Postural Reflexes

R

Note from both doctors:Know the center and receptor of each reflex

❖ **Static reflexes**

➢ 1-**Spinal cord reflexes**

A- **Local** (stretch reflex) and the receptors are muscle spindles.

B-**Segmental** (Cross extensor reflex) and the receptors are proprioceptors of extensors of the released limb.

➢ 2-**Medulla Oblongata reflexes**

A- Neck static reflexes

1-Ventroflexion of head: Flexion of forelimbs and extension of hindlimbs.

2-Dorsiflexion of head: Extension of forelimbs and flexion of hindlimbs.

B- Labyrinthine static reflex

➢ 3- **Righting reflexes**

center is midbrain EXCEPT the visual reflex is cerebral cortex.

❖ **Phasic reflexes**

➢ 1-**Hobbing reaction**

When animal is pushed laterally, reflex hopping to keep limbs in position to support body.

receptors are the muscle spindles.

➢ 2-**Placing reaction**

Blind folded animal suspended in air and moved towards a supporting surface, the feet will be placed firmly on the supporting surface.

-Receptors: 1-touch receptors 2-proprioceptors in sole of feet.

Postural Reflexes

R

Very Important table Slide number 32

Reflexes	Stimulus	Response	Receptor	Integrated in
Stretch reflexes	Stretch	Contraction of muscle	Muscle spindles	Spinal cord, medulla
Positive supporting (magnet) reaction	Contact with sole or palm	Foot extended to support body	proprioceptos in distal flexors	Spinal cord
Negative supporting reaction	Stretch	Release of positive supporting reaction	Proprioceptors in extensor	Spinal cord
Tonic labyrinthine reflexes	Gravity	Construction of limb extensor muscles	Otolithic organs	medulla
Tonic Neck reflexes	Head turned: (1) To side (2) UP (3) Down	Change in pattern of extensor contraction (1) Extension of limbs on one side to witch head is turned (2) Hind legs flex (3) Forlegs flex	Neck proprioceptors neck	medulla
Labyrinthine righting reflexes	Gravity	Head keept level	Otolithic organs	Midbrain
Neck righting reflexes	Stretch of neck muscles	Righting of thorax and shoulders, then pelvis	Muscle spindles	Midbrain
Body on head righting reflexes	Pressure on side of body	Righting of head	Exteroreceptors	Midbrain
Body on Body righting reflexes	Pressure on side of body	Righting of body even when head held sideways	Exteroreceptors	Midbrain
Optical righting reflexes	Visual cues	Righting of head	Eyes	Cerebral cortex
Placing reactions	Various visual, extero-ceptive, and proprio-ceptive cues	Foot placed on supporting surface in position to support body	Various	Cerebral cortex
Hopping reactions	Lateral displacement while standing	Hops, maintaining limbs in position to support body	Muscle spindles	Cerebral cortex

Postural Reflexes

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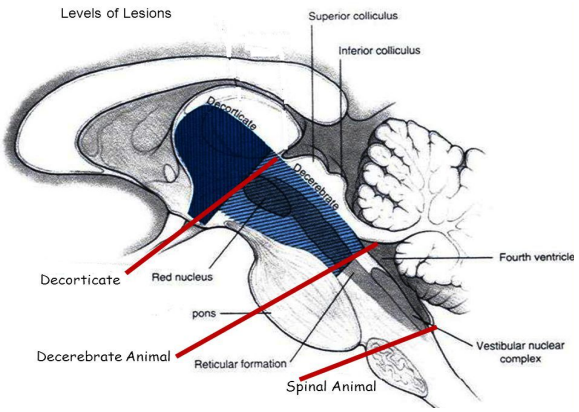
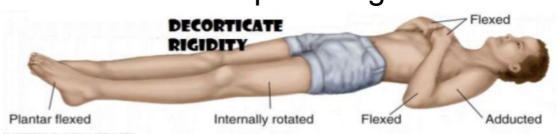


	Definition	★ features
Decerebrate rigidity	★it is a mid-collicular lesion • In Decerebrate rigidity **below** red nucleus (section between superior& inferior colliculi of midbrain) • block inhibitory signals from brain & red nucleus to tonically active pontile reticular formation & Vestibular N 	• Maintained tonic static postural reflexes that support animal against gravity (Medullary tonic neck R & Medullary labyrinthine R) • Absent midbrain righting R • Extension of head & 4 limbs extensors (as in labyrinthine static R) The jaw may be clenched with the neck hyperextended due to increased extensor tone from vestibulospinal & reticulospinal tracts to extensor motor neuron • spasticity &rigidity & extension in antigravity muscles
Decorticate rigidity	★ it is a lesion in cerebral cortex but brain stem is intact • In decorticate rigidity the lesions is **above** the red nucleus so rubrospinal are intact together with pontine reticulospinal and the vestibulospinal this leads to the characteristic flexion posturing of the upper extremities and extensor posturing of the lower 	• Full extension of the legs • Arm lying across the chest • Semiflexoin at the elbow • Slight pronation of forearm • Flexion of wrist and fingers

★ Postural Reflexes

R

- You should know what are the neurophysiological basis of the two mentioned types of rigidity in the lecture, and what are the retained and lost reflexes.

- Decerebrate rigidity:

Reflexes that are lost/absent imp	**Reflexes that are retained /still present** (those which have their centers in SC, medulla or pons)
Righting Reflex	-Stretch reflex -positive&negative supporting reaction -crossed extensor reflex
	Tonic Labyrinthine reflexes
	Tonic Neck Reflexes

- Decorticate rigidity:

Reflexes that are lost/absent imp	**Reflexes that are retained /still present**
Placing Reaction	Tonic Labyrinthine reflexes
Hopping Reaction	Tonic Neck Reflexes
Visual righting reflex	Other Righting Reflexes

The two table above are only to demonstrate the retained and lost reflexes, you should back to your slides or the team work of this lecture and know each type (definition, lesion, and manifestations).

Spasticity and increase muscle tone

R

❖ **You should know the different between spasticity and rigidity.**

	Spasticity	Rigidity
Character	❖ Unidirectional, agonist either antagonist ❖ pyramidal lesion ❖ Velocity dependent. ❖ clasp-knife ❖ is often associated with UMNL	❖ present in both agonist and antagonist muscles. ❖ Rigidity is usually **extra-pyramidal** in origin ❖ **not** velocity dependent ❖ lead pipe rigidity or coag-wheel rigidity. ❖ It is often associated with basal ganglia disease such as Parkinson's disease
Type	❖ (UMNS) syndrome include : ➢ Cerebral palsy ➢ Stroke ➢ Spinal cord injury ➢ Multiple Sclerosis ➢ Acquired brain injury (trauma , etc)	❖ Rigidity in Parkinsonism: ➢ Lead-pipe rigidity ➢ Cog-wheel rigidity ❖ Decerbrate rigidity ❖ Decorticate rigidity

❖ You should know the spinal cord lesions:
complete transection of spinal cord and its phases you should know them in **DETAILS**!
- What are the phases?
- what are the features of each phase? E.g,
- What are the features of spinal shock stage?

❖ Know the presentation and manifestation according to the site of lesion, e.g, lesion in the upper level of the cervical area patient will die, and so on.

Hemisection of the Spinal Cord (Brown- Sequard syndrome)

R

1

At the level of the lesion :

All manifestations occur on the same side

1. Paralysis of lower motor neuron involving only the muscle supplied by the damaged segments
2. Loss of **all** sensations in the areas supplied By the afferent fibers that enter the spinal cord in the damaged segments +/- band of hyperesthesia or anaesthesia
3. Vasodilation of the vessels that receive Vasoconstrictor fibers from the damaged segments

2

Ipsilaterally below the level of the lesion:

1. Paralysis of UMN due to interruption of Pyramidal and extrapyramidal tracts

2. UMNL/spastic lower limb (spasticity why? &CLONUS

As we know corticospinal tract supply the opposite side

Dr najeeb explain at 56:20

الديسيندريق تاركت اللي جايه من الناحية التانية وهي نازلة في الناحية السليمة بتيقي قبل لا تنتهي بتعمل كورس للاوبيزت ساييد فذي شغالة كويس فتغذي الجهه اللي فيها الكت وبنفس الوقت كل الانهيبيتوري سقنل مقطوعه فاللي ماخد اليد العليا هي الاكسيتيبيشن

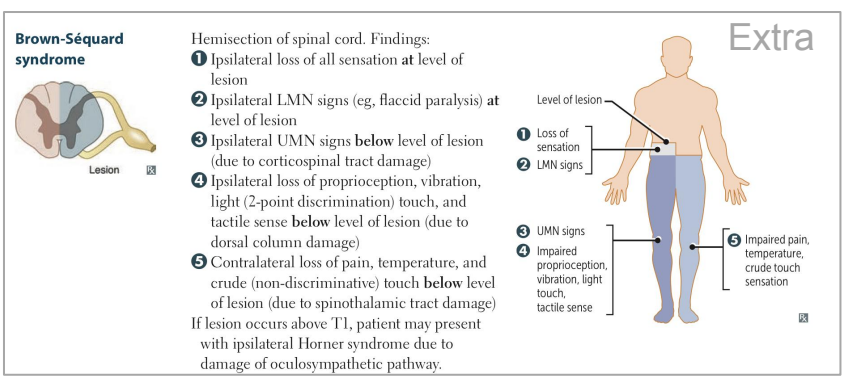
The corticospinal lesion produces spastic paralysis on the same side of the body **below the level of the lesion** (due to loss of moderation by the UMN).
At the level of the lesion, there will be flaccid paralysis of the muscles supplied by the nerve of that level (since Lower motor neurons are affected at the level of the lesion).

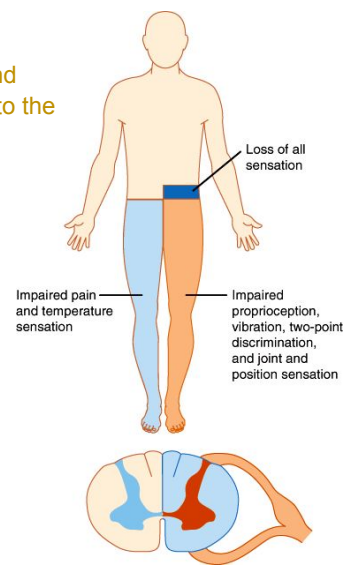
3. Loss of fine touch, two-point discrimination, Position and vibration sensations **why?** (Below the level of the lesion, there are tracts from the spinal cord (dorsal column/gracile and cuneate tracts) carry the sensation of the same side and ascend to the brain, which will be cut off)

3

Contralaterally below the level of the lesion:

- Loss of temperature and pain sensations **Why?** (The lateral and ventral spinocortical tracts that cross below the level of the lesion and ascends to the opposite side of the brain will be cut off)





Proprioceptive pathways

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- how do we classify receptors? Location, adequate stimulus, speed of adaptation
- What do we mean by adaptation? When a continuous sensory stimulus is applied, the receptor responds at a high impulse rate at first and then at a progressively slower rate until finally the rate of action potentials decreases to very few or often to none at all.
- What are the types of Rapidly adapting receptors? Pacinian receptors.
- What are the slowly adapting receptors? Pain receptors & Muscle spindles.
- Know the receptor potential and action potential and the different between them.

The difference between generator potential and Action potential*

Receptor or Generator potential	Action potential
- In the receptor	- In the sensory nerve fiber
- Graded	- Not Graded
- Doesn't obey all or none role	- Obeys all or none role
- Can be summated	- Not summated
- Unpropagated	- propagated

- **Increase intensity of receptor potential will lead to increase amplitude.**
- **Increase intensity of action potential will lead to increase frequency.**

Proprioceptive pathways

R

- What are the major sensory pathways?
  1-**Conscious** proprioception reach the cerebral cortex, usually through:
  A- dorsal column medial lemniscus pathway. (High degree of localization, and high intensity of discrimination i.e fine), crossing at medulla oblongata, Fibres aB which have VERY FAST velocity.

  B- Anterolateral system which consist of ventral and lateral spinothalamic tracts. (Pain, Thermal, Crude, Pressure, Tickle, Itch, and sexual sensations), crossing in the spinal cord, Fibres c fibres which are slow, and aD which have RELATIVELY slow velocity.

  2-**Unconscious proprioception** reach the cerebellum, usually through:
  A-Dorsal Spinocerebellar tract: Inter through ICP, end in the vermis & intermediate zone of the cerebellum, usually stimulated and carry impulses from the peripheral parts of the body.

  B-Ventral Spinocerebellar tract: usually stimulated by the corticospinal and rubrospinal tract, and this usually inform the cerebellum about what types of motor copies which sent by the motor cortex to the spinal cord.
- What do we mean by proprioception? The awareness of body position and movements of body parts
- What are the static and dynamic proprioception?
  A-Static: conscious perception of the orientation of the different parts of the body with respect to one another.
  B-Dynamic: rate of movement sense. (Kinesthesia)

Cerebral Hemispheres functions

R

- You should know the cerebral hemisphere layers.
- **First**: All incoming impulses IV first; then the signal spreads toward the surface of the cortex and also toward deeper layers.
- **Second**: Layers I, II, and III perform most of intracortical association function.
- **Third**: The neurons in layers II and III making short horizontal connections with adjacent cortical areas.
- **Fourth**: The neurons in layers V and VI send output signals to brain stem,Spinal cord (V) and Thalamus (VI).
- **Association areas**:
  - **ParietoOccipitotemporal association areas**:

1-Spatial coordinates of the body. Parietal & Occipital lobes
2-Language Comprehension. Behind auditory area superior gyrus of temporal lobe
3-Initial processing. Angular gyrus
4-Naming objects. Lateral portion of anterior occipital lobe & posterior temporal lobe

Slide 13 and 14 were skipped

  - **Area of recognition of faces**

- Inability to recognize faces is called prosopagnosia
- Located on the underside of the brain on the medial occipital and temporal lobes.

Cerebral Hemispheres functions

R

- Know the different lobes of the brain and the functions of each lobe.
- You should know what do we mean by Dominant (Categorical) and Non-Dominant (Representational) hemispheres, and functions of each one.

Dominant (categorical hemisphere) & Non-dominant (representational) Hemisphere

- **Functional differences between left and right hemispheres**
- **In most people, left hemisphere (dominant hemisphere)controls:**

reading, writing, and math, decision-making, logic, speech and language (usually)

- **Right cerebral hemisphere relates to:**

1. understanding & interpreting music
2. Non verbal visual Experience (facial expression , gesture)
3. Spatial relation between the person & their surroundings
4. Body language and intonation of peoples voices

Physiology of Consciousness

R

Definition of RF

Set of interconnected nuclei that are located throughout the brainstem (Pons, Midbrain, Upper medulla) AND thalamus

Functions of reticular formation:

1

Somatic motor control

(Reticulospinal tracts)

They maintain the amount of muscle contraction needed for the body to function normally & to be in a good state with self & environment.

2

Cardiovascular control

Through cardiac and vasomotor centers of the medulla oblongata

3

Pain modulation raphe nuclei

- Pain signals from the lower body >> >> RF >> >> cerebral cortex
- RF is origin of the descending analgesic pathways
- (act on the spinal cord to block the transmission of some pain signals to the brain)

4

Sleep and consciousness

The reticular formation has projections to the thalamus and cerebral cortex . It plays a central role in states of consciousness like alertness and sleep. Injury to the reticular formation can result in irreversible coma *.

5

Habituation

This is a process in which the brain learns to ignore repetitive, meaningless stimuli while remaining sensitive to others. A good example of this is when a person can sleep through loud traffic in a large city, but is awakened promptly due to the sound of an alarm .

If we have ascending pain sensation through the ascending sensory tract traveling towards the cerebral cortex of the spinal cord, it must travel through the Raphe Nuclei.
When pain becomes severe, the Raphe Nuclei will send *inhibitory signals to inhibit the transmission of pain to the cerebral cortex causing the activation of the Internal Analgesic Tract.

What do we mean by RAS?

A group of neuronal circuits connecting the brainstem to the cortex Originate in the upper brainstem reticular core and project through synaptic relays in the thalamic nuclei to the cerebral cortex.

Enumerate RAS

The Reticular Activating System is composed of the:
Bulboreticular Facilitatory (excitatory) area + Thalamus

- **You should know what are the levels of consciousness**.
  1- Normal consciousness
  2-Clouded consciousness
  3-Sleep
  4-Coma

- **What are the structures important for our consciousness?**
  1-Reticular formation
  2-Thalamus
  3-hypothalamus

- He skipped slides from 29 to 32.

R

If RAS is depressed All functions related to the cerebral cortex will be depressed

-An under-aroused cortex
-Difficulty in learning
-Poor memory
-Little self-control
-lack of consciousness or even coma.

RAS Dysfunction: important

-Over aroused cortex
-Hyper-vigilance (sensory sensitivity)
-Touching everything
-Talking too much
-Restless
-Hyperactive

If RAS is Excited

People with excited RAS have exaggerated cerebral cortex activities. So they will become sensitive to touch, light, sound and other stimuli. It is seen in patients with ADHD.
ADHD: Attention deficit hyperactivity disorder is a disorder that causes above normal levels of hyperactive & impulsive behaviors.

Electroencephalogram

a test that detects electrical activity in your brain using small, metal discs (electrodes) attached to your scalp. Your brain cells communicate via electrical impulses and are active all the time, even when you're asleep. This activity shows up as wavy lines on an EEG recording. **Here are 4 waves that you may find in an EEG recording:**

Alpha

-Recorded from the parietal & occipital regions
-Awake and relaxed + eyes closed
-Frequency of 10 to 12 cycles/second
Most common wave

Delta

-From the cerebral cortex
-Frequency of 1 to 5 cycles/second (Very low).
-Sleep in adults and awake infants.
-In awake adults indicates brain damage.
Slowest wave

Beta

-Recorded from the Frontal lobe
-Produced by visual stimuli and mental activity.
-Frequency of 13 to 25 Cycles/second.
Wave with the highest frequency

Theta

-Temporal and occipital lobes.
-Frequency of 5 to 8 cycles/second (low).
-**Normal in newborn.**
*Theta waves in adults indicates severe emotional stress awake adults, can indicate neurodegenerative diseases
Slower than alpha & beta waves

How we assess a comatose patient? **(VERY important)**

Indices of Level of Consciousness

Appearance and behavior

- Posture (sitting ,standing?)
-eyes (Open?)
-Facial expression.
-Responds to stimuli (including the examiner's questions about name, orientation in time & place. And other general Qs like: who is the president?)

Vital Signs

-Pulse
-BP
-respiration
-pupils fixed and dilated can indicated death
-reflexes, particularly brainstem reflexes e.g. Vestibulo-ocular reflex & cephalo ocular reflex etc. calorie test, gag reflex, and other spinal reflexes

EEG

Each of these states (wakefulness, sleep, coma and death) has specific EEG patterns.

Evoked potentials (in cases of brain death).

* Stimulation of a sense organ can evoke a cortical response that can be recorded by scalp electrode over the primary receiving cortical area for that particular sense. *

Brain Neurotransmitters

R

- What are the types of receptors? Metabotropic and Ionotropic
- What are the types of receptors for each neurotransmitter?

  A-Acetylcholine:
  1-Nicotinic (Ionotropic) 2-Muscarinic (Metabotropic)

  B-Glutamate:
  1-Metabotropic
  2-Ionotropic (AMPA,Kainate,NMDA)

  C-GABA:
  1-GABAa
  2-GABAb
  3-GABAc

  D-NE:
  1-Alpha
  2-Beta

  E-Dopamine:
  D1-D5
- What are the functions of each neurotransmitter?
- What are the disorder associated with increase or decrease of each neurotransmitter?
- Many disease can caused by more than one neurotransmitter abnormality.
- Most important factor for Schizophrenia is over stimulation of D2 receptor.
- Alzheimer's disease due to loss of Ach.
- Serotonin has a role in mod regulation.
- Pathways of dopamine

Dr.Laila
Pathways of dopamine
Domapamine receptors
Function of dopamine
Dopamine disorders

R

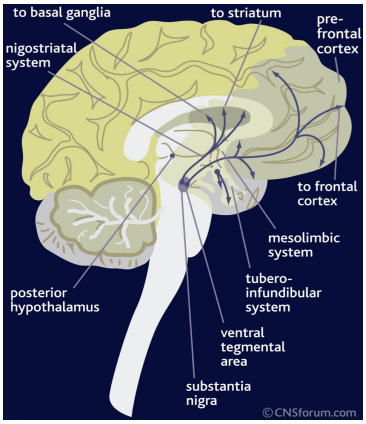
5. Dopamine System:
Dopamine

Overview

- Dopamine is a catecholamine that is synthesized from tyrosine
- Five dopaminergic receptors (D1-D5).
- Overstimulation of D2 receptors is thought to be related to schizophrenia.
- Dopamine is transmitted via three major pathways:

Pathways

1	The first (nigro striatal system) extends from the substantia nigra to the caudate nucleus-putamen (neostriatum) and is involved in motor control and concerned with sensory stimuli and movement.
2	The second pathway project to the mesolimbic forebrain and Related to cognitive, reward and emotional behavior in reward and emotional behavior and addiction. Dysfunction is connected to hallucinations and schizophrenia.
3	The third pathway, known as the tuberoinfundibular system It is concerned with: - Regulation of secretion of prolactin from the anterior pituitary endocrine systems. - Maternal behavior (nurturing).



Dopamine Functions & Disorders

Functions	❖ Reward ❖ Pleasure, euphoria ❖ Motor function (fine tuning) ❖ Compulsion ❖ Perseveration
Disorders	❖ Schizophrenia ❖ Parkinson's Disease. Cocaine elevate activity at dopaminergic synapses.

Summary

R

NT	Ach	Glutamate	GABA	serotonin (5HT)
Postsynaptic effect	Excitation	Excitatory 75% of excitatory transmission in the brain.	Major inhibitory mediator	Excitatory
From	Acetyl co-A + choline	By reductive amination of kreb's cycle intermediate α-ketoglutarate.	Decarboxylation of glutamate by glutamate decarboxylase (GAD) by GABAergic neuron.	Tryptophan
Site of Synthesis	Cholinergic nerve endings Cholinergic pathways of brainstem.	Brain & spinal cord e.g. hippocampus.	CNS	CNS, Gut (chromaffin cells) Platelets & retina.
Postsynaptic Receptor	1.Nicotinic. 2.Muscarinic.	Ionotropic and metabotropic receptors. Three types of ionotropic receptors e.g. NMDA, AMPA and kainate receptors.	GABA – A increases the Cl - conductance, GABA – B is metabotropic works with G – protein GABA transaminase catalyzes. GABA – C found exclusively in the retina.	5-HT1 to 5-HT 7 5-HT 2 A receptor mediate platelet aggregation & smooth muscle contraction.
Fate not imp	Broken by acetyl cholinesterase.	It is cleared from the brain ECF by Na + dependent uptake system in neurons and neuroglia.	Metabolized by transamination to succinate in the citric acid cycle.	Inactivated by MAO to form 5-hydroxyindoleacetic acid(5-HIAA) in pineal body it is converted to melatonin.
Function	Cognitive functions e.g. -memor -peripheral action e.g. cardiovascular system.	Long term potentiation involved in memory and learning by causing Ca^{++} influx.	GABA – A causes hyperpolarization (inhibition) Anxiolytic drugs like benzodiazepine cause increase in Cl- entry into the cell & cause soothing effects. GABA – B cause increase conductance of K+ into the cell.	Mood control, sleep, pain feeling, temperature, BP, & hormonal activity.

Summary

R

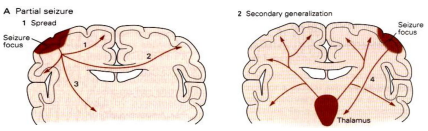
Catecholamines			
NT	**Epinephrine (adrenaline)**	**Norepinephrine**	**Dopamine**
Postsynaptic effect	Excitatory in some but inhibitory in other	Excitatory	Excitatory
From	Tyrosine produced in liver fromphenylalanine	Tyrosine, found in pons. Reticular formation, Locus coeruleus, Thalamus, Midbrain	Tyrosine
Site of Synthesis	Adrenal medulla and some CNS cells	Begins inside axoplasm of adrenergic nerve ending is completed inside the secretory vesicles	CNS, concentrated in basal ganglia and dopamine pathways e.g. -nigrostriatal -mesocorticolim bic and tubero -hypophyseal pathway
Postsynaptic Receptor	Excites both alpha α and beta β receptors	α1 α2 β1 β2	D1 to D5 receptor
Fate not imp	1. Catabolized to inactive product through COMT & MAO in liver. 2. Reuptake into adrenergic nerve endings. 3. Diffusion away from nerve endings to body fluid.		
Function	For details refer ANS. e.g. fight or flight, on heart, BP, gastrointestinal activity etc. Norepinephrine controls attention & arousal, sleep/wake cycle.		Sensory motor Cognitive/emotional behavior Endocrine Hypothalamic Decreased dopamine in Parkinson's disease. Increased dopamine 36 concentration.

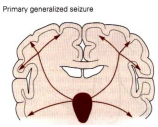
Pathophysiology of Epilepsy
Classification of seizures

R

Seizures

Seizures affect all ages,most cases of epilepsy are identified in childhood, and several seizure types are particular to children





1 **Partial (focal):**
Involves only one part of cerebral hemisphere

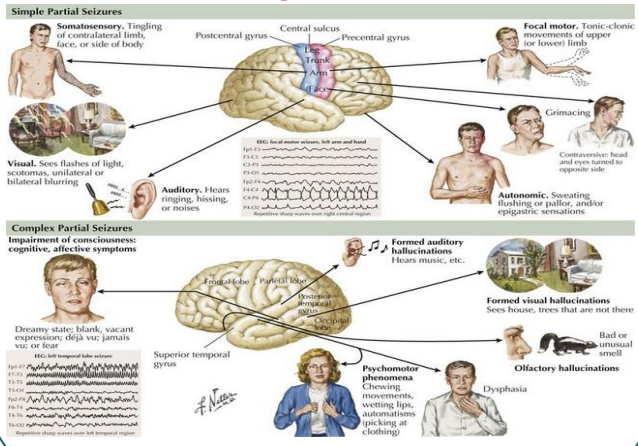
Primary Generalized:
Involves the entire cerebral cortex.(whole of it) **2**

A) **Simple**:
Awareness is retained

B) **Complex**:
Altered consciousness subconsciousness

C) **Partial with secondary generalization:**
Seizures may spread to involve adjacent areas of the brain and progress to complex partial seizures, and complex partial seizures can secondarily become generalized.

important



A) **Tonic-clonic:** Stiffness followed by violent contractions & relaxation (1-2 minute).

B) **Tonic**: Muscle stiffness

C) **Clonic**: Spasms of contraction & relaxation

D) **Atonic (loss of tone)**: Patients legs give under him & drop down

E) **Myoclonic**: Jerking movement of the body

F) **Absence(Petit mal):**Brief loss of consciousness with minor muscle twitches. and eye blinking

- Typical
- Atypical

in typical absence seizures there is a characteristic pattern in EEG, unlike atypical absence seizures

G) **Status epilepticus**: Recurring tonic-clonic seizure (30 min or more), or two or more seizures within a five-minute period without the person returning to normal between them. “life-threatening condition”

Epilepsy

VERY IMPORTANT

R

Other types of focal (partial) seizure	
Partial psychomotor (temporal lobe)	-Epileptic seizures which originate in the temporal lobe of the brain. -The seizures involve sensory changes, for example smelling an unusual odour that is not there, and disturbance of memory. -Visual , auditory , olfactory or visceral hallucinations, déjà vu (over familiarity), feelings of unreality (jamais vu) -The most common cause is mesial temporal sclerosis deep scarring of the temporal lobe
Partial Jacksonian epilepsy	-Focal motor seizures begin in motor areas of cerebral cortex, usually begins with twitching of the thumb or finger , toe or the angle of the mouth. -Spreading to involve the limbs on the side opposite the epileptic focus. -Clinical evidence of this spread of activity is called the march of the seizure.

Generalized tonic-clonic (grand mal) seizure

-Aura(+/-) It's like a warning sign

peculiar sensation or dizziness; then sudden onset of seizure **with loss of consciousness**.

like an abnormal smell

-Tonic phase : مرحلة التصلب

Rigid muscle contraction in which clenched jaw and hands, eyes open with pupils dilated, **lasts 30 to 60 seconds**

in tonic phase there might be an epileptic cry or/and cyanosis due to spasm of respiratory muscles

-Clonic phase: مرحلة الانقباض والانبساط

Rhythmic, jerky contraction and relaxation of all muscles in with incontinence and frothing at the lips; may bite tongue or cheek, lasts **several minutes**.

any seizure with biting of the tongue is usually epileptic in origin

-Postictal state: مرحلة الراحة والتعافي

Sleeping or dazed for up to **several hours.** depression of CNS

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Pathophysiology of Epilepsy (at molecular level)

Cortical cell membrane level:

01 Instability of the nerve cell membrane

02 Polarization abnormalities (excessive polarization , ,hypopolarization , or lapses in repolarization)

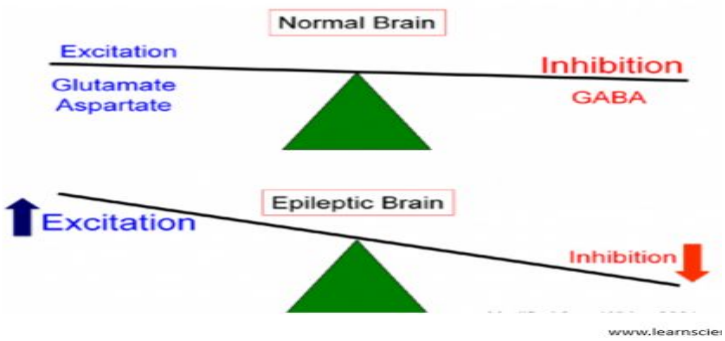
03 Allowing the cell to be more susceptible to activation

04 Hypersensitive neurons with lowered thresholds for firing and firing excessively , related to

- **4.1** **Excess of Excitatory** (acetylcholine- or Glutamate– related activity)
- **4.2** **Decreased inhibitory** (GABA –related activity)

05 Both or any one of 4.1 & 4.2 can lead to instability of cell- membrane & lowered threshold for excitation

06 Excessive polarization, hypopolarization allowing the cell to be more susceptible to activation spontaneously or by any ionic imbalances in the immediate chemical environment of neurons



Thank you

Done by:
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