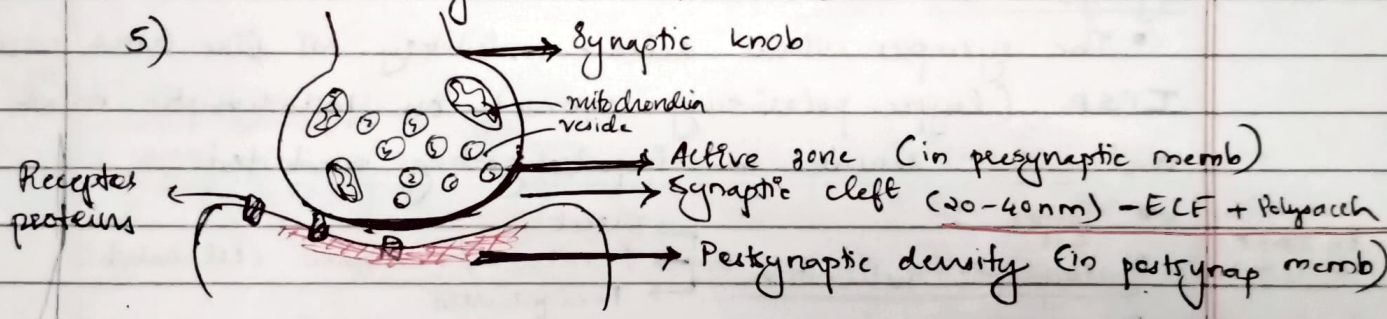
CNS

SYNAPSE (6E)

1) Def
2) Classification → Functional, Structural
3) Functions (6)
4) FUNCTIONAL ANATOMY
   1) Axons of presynaptic neuron divide into many branches
   2) End of each branch → End foot / Synaptic knob
   3) Two imp structures in knob → Vesicle (contain NT) & Mitochondria (for synthesis of neurotransmitter)
   4) KISS-AND-RUN DISCHARGE :-

Synaptic vesicle discharges its contents through a small hole in presynaptic memb, then memb reseals rapidly and main vesicle stays inside the cell.

5)



6) Receptor protein (2 types)

i) Binding component
- binds with transmitter

ii) Intracellular
- Ligand gated channels → ionotropic receptors
- Metabotropic

7) Neurexins - proteins that hold presynaptic and postsynaptic together → provides structural stability to synapse.

PROPERTIES OF SYNAPSE (11)

1) One way conduction (Receptor → receptive & effector)
- Impulse pass only from pre → post membr (Law of forward conduction)
- This is bcoz receptors for neurotransmitter released are present only on post synaptic OR neuro only in presynaptic membr

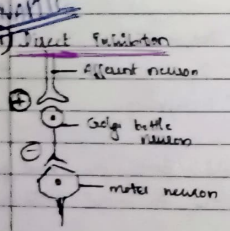
2) Synaptic delay
- When impulse reaches presynaptic terminal of chemo synapse there is an interval of 0.5 ms before response is obtained in postsynaptic neuron

3) Synaptic inhibition
- The synapses which release inhibitory NT like GABA cause IPSP (hyper-polarising response) on post-synaptic membr and this results in impulse getting inhibited
- Types :-

by IPSP → i) Postsynaptic inhibition → Direct / Feedback / Renshaw cell inhib / Feedforward

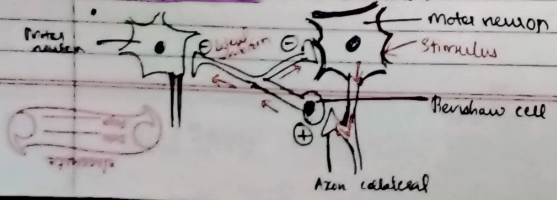
ii) Presynaptic

i) Direct Inhibition



- When postsynaptic neuron directly inhibited by germs of IPSP on post-synaptic neuron
Eg:- Spinal cord
Golgi tendon organ inhibition
- NT → Glycine

ii) Feedback / Renshaw cell Inhibition

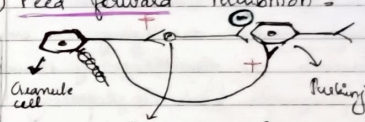


- Neurons may inhibit themselves in a feedback manner

Mechanism
- When motor neuron stimulated
↓
impulse passes through axon, collateral also
↓
Stimulates Renshaw cell
↓
Releases inhibitory NT → Glycine → Produce IPSP in same neuron
(Also there is lateral inhibition of adjacent motor neuron)

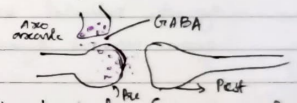
AA → Strychnine poisoning → poison binds to glycine receptors in motor neuron and blocks it
Hence, all muscles remain contracted ← So, no feedback inhibition

iii) Feed forward Inhibition :-



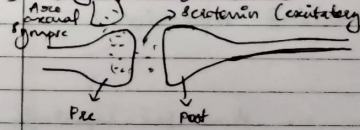
- Seen in cerebellum
- Both basket & purkinje stimulated, but basket in turn inhibits purkinje

2) Presynaptic / Indirect



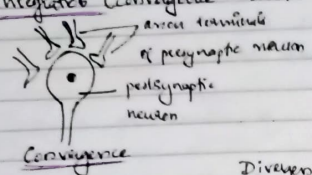
- Inhibition at presynaptic terminal (axo-axonal synapses)

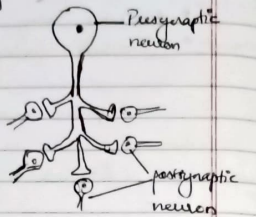
4) Presynaptic facilitation



- A.P. in pre-synaptic membrane is prolonged and L-type Ca^{2+} channels opened for longer period

5) Integration (Convergence and divergence)





6) Synaptic modification / After discharge

- A single input signal is converted to repetitive discharges as it passes through the synapse and there will be prolonged output discharge from circuit.

7) Summation

- Since synaptic potentials are graded, individual potentials (EPSP & IPSP) can be summated.

1) Spatial : No. of synaptic knobs converging on one postsynaptic membs are simultaneously stimulated
2) Temporal : A single presynaptic knob is repeatedly stimulated in such a way that the 2nd synaptic potential arrives before membs recovers from previous synaptic potential.

8) Occlusion

- When 2 excitatory afferents to skele m. are simultaneously stimulated sometimes : tension developed in muscle is less than sum of tension developed when 2 afferent are stimulated separately.

9) Post-tetanic Potentiation

- When excitatory synapses are stimulated repeatedly and rapidly for a short period and a period of rest is allowed → the postsynaptic neuron becomes more responsive to subsequent stimulation and shows enhanced post-synaptic potential.

10) Synaptic fatigue

- If synapses are stimulated repeatedly and rapidly for some time, no. of discharge produced is great for some time but progressively becomes less and finally stops
AA → Protective phenomenon against excess neuronal activity
- Cause for cessation of fits during an epileptic attack without treatment.

11) Synaptic Plasticity

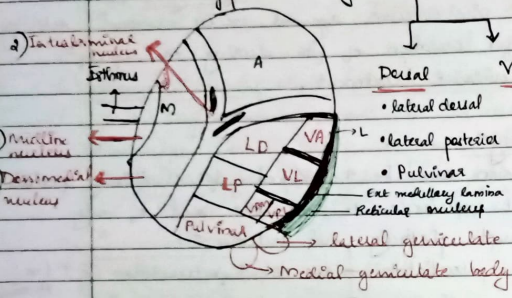
- Syn. transmission can be ↑ed / ↓ed for short/long periods on basis of past experience to suit the need of body
- Thus is synaptic plasticity and includes :-

1) Post-tetanic potentiation → due to ↑ intracellular Ca^{2+} (in presynaptic)
2) Long-term " " " " " (in post neuron)
3) Long-term depression → opposite to LTP
4) Habituation → When neutral stimulus repeated, response ↓ed & finally disappears
5) Sensitization → Enhanced response to known stimulus after presentation of intense/noxious stimulus

THALAMUS

(revised)

1) Anatomical classification of thalamic nuclei
- Ant grp - present blw bifurcation of internal medullary lamina
- Medial grp - medially (in fig)
- Lateral grp - laterally



Dorsal	Ventral
• lateral dorsal	• Ventral anterior
• lateral posterior	• Ventral lateral
• Pulvinar	• Ventral posterior (medial and lateral)

- diffuse sensation
- project to whole cortex

2) Functional classification of thalamic nuclei
- non-specific projection
- specific projection
  - Specific sensation
  - to specific part of cortex
- CONNECTIONS (UR)

I) Non-specific nuclei

Afferent fibers	Nuclei	Efferent fibers
1) Reticular formation	→ Midline	→ Neocortex
2) Corpus striatum, Tectal, Hypo, cerebellum	→ Intralaminar	→ Neocortex
3) Fibers from cerebral cortex	→ Reticular	→ To reticular formation

II) of specific (association) relay nuclei
1) Lateral dorsal group (lateral dorsal, lateral posterior, pulvinar)
2) Dorsomedial nucleus

III) of specific relay nuclei
- all the rest

ANATOMY (Connections table)

NUCLEUS	AFFERENT	EFFERENT	FUNCTIONS
1) Anterior	Mammillary body	Cingulate gyrus	• Limbic, emotions
2) Medial dorsal	Hypo, amygdala	Prefrontal cortex	• Personality
3) Midline	Reticular formation	Hippo, amygdala	• limbic, arousal resp
a) Lateral dorsal gp (Lateral dorsal, posterior, pulvinar)	Ventral thalamic nuclei, Sup colliculus, pretectum	Cing, parahippo, parietal, occipital & temporal lobes	• limbic • Sensory analysis
1) ~~Post~~ VPM	Trigeminal lemniscus, Solitario-thalamic tract	Postcentral gyrus	• SSAI (face area)
2) VPL	Medial, ~~lateral~~ Spinal lemnisci	〃	• SSAI
3) MGB	Auditory fibres from inf colliculus	Area 41, 42 (1° auditory area)	• Relay for auditory impulses
4) LGB	Optic tract	Area 17 (1° visual area)	• Relay for visual impulses
1) Ventral Ant	Globus pallidus	Premotor area	• Relay for striatal impulses • Modify motor activity
2) Ventral lateral	• Cerebellum • Red nucleus	Premotor & motor area	• Relay for Cerebellar activities

FUNCTIONS (Rook) - code - THALAMUS

1) T & The genesis of EEG (Electroencephalogram)
2) H : Hypothalamic connections
   - plays a role in emotional behaviour (mammillothalamic tract)
   - memory
3) A : Arousal and alert
   - Non-specific projection nuclei play important part of ascending reticular activating system (ARAS).

4) L : Language function
- Dorsolateral nucleus is connected to parietal lobe and is concerned with language & speech.

5) A : Autonomic and somatic sensation
- Role in appreciation of sensations like pain, crude touch, crude temp etc.

6) M : Motor function and regulation
- Because of its connection with cerebellum and basal ganglia, it acts as an integrating centre for motor activity

7) U : Urge for sex/ perception of sexual sensation
- connection with papez circuit

8) S : Sensory relay centre
- Most imp subcortical relay centre for all main sensory pathways.

THALAMIC SYNDROME (TOTICS A to H) Physio basis (Degree and Posey features)

1) Thalamic hand — Motor defect due to moderate flexion at wrist and hyperextension of all fingers
2) Over reaction — Subthalamic perception of pain
• pain threshold reduced
3) Ataxia limb — Not able to localise the position of limbs with eyes closed
4) Intention tremors — involuntary movements (cerebellum VA, VL nuclei)
5) Choreoathetosis — " "
6) Sensory ataxia — Ataxia due to loss of kinesthetic sensation
7) Astereognosis — loss of tactile sensation (defect in touch-pressure system)
8) Hemianesthesia — loss of sensations in opp half of the body.

ASCENDING PATHWAYS

- Classification :- 1) Tracts in dorsal / posterior white column
- Fasciculus gracilis
- Fasciculus cuneatus
2) Tracts in lateral white column
- Lateral spinothalamic
- Dorsal spinocerebellar
- Ventral spinocerebellar
3) Tracts in ant white column
- Ant. spinothalamic tract

MEDIAL LEMNISCUS SYSTEM/ POSTERIOR (Dorsal) COLUMN PATHWAY/ Tract of Gall and Burdach

1) Sensations carried :-
- Fine touch
- tactile localisation
- 2-point discrimination
- vibration sense
- Proprioception & kinesthetic sensation
- Stereognosis

2) Receptors :-
- Touch, tactile localisation & discrimination
- Rapidly adapting : Meissner's corpuscle, Pacinian corpuscle
- Slowly adapting : Merkel's disc, Ruffini endings
- Proprioception & kinesthetic sensation
- Slowly adapting spray endings in joints
- Pacinian corpuscle in ligament
- Muscle spindle and golgi tendon organ

3) Neurons :-
1st order neuron
- It is a pseudo-unipolar neuron with 2 processes : peripheral and central. The cell body is located in

dorsal horn of spinal cord. the fibres enter dorsal horn, give minor branches and ascend ipsilateral up in dorsal column of same side (no crossing)

- Sensations from sacral, lumbar and up-to midthoracic level are carried by fasciculus gracilis.
- Sensations above midthoracic are carried by Fasciculus cuneatus
- Fasciculus gracilis begins at lowest level of S.C.
- At higher levels, cuneatus fibres enter S.C. pushing gracilis more medially.
- The fibres from medial to lateral are sacral, lumbar, thoracic and cervical segments.

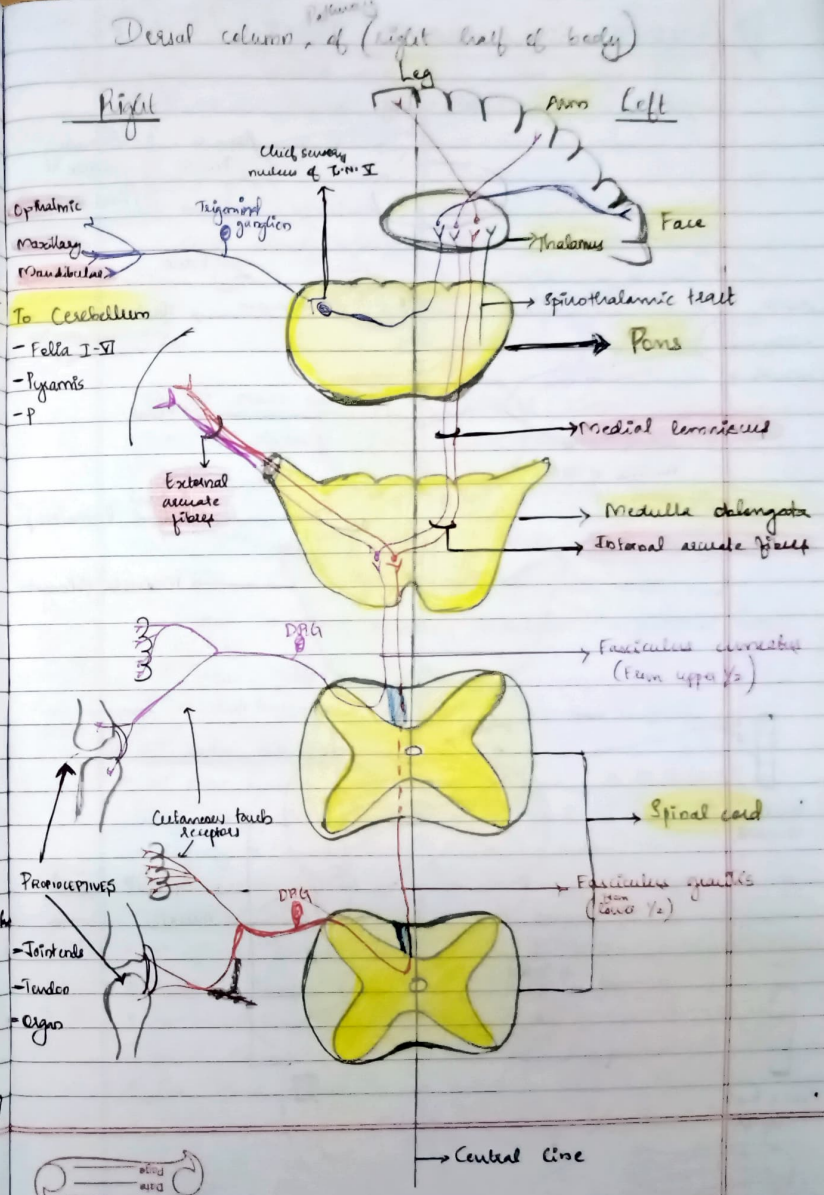
2nd order neurons

Fibres of fasciculus gracilis synapse in nucleus gracilis and fibres of fasciculus cuneatus synapse in nucleus cuneatus in medulla.

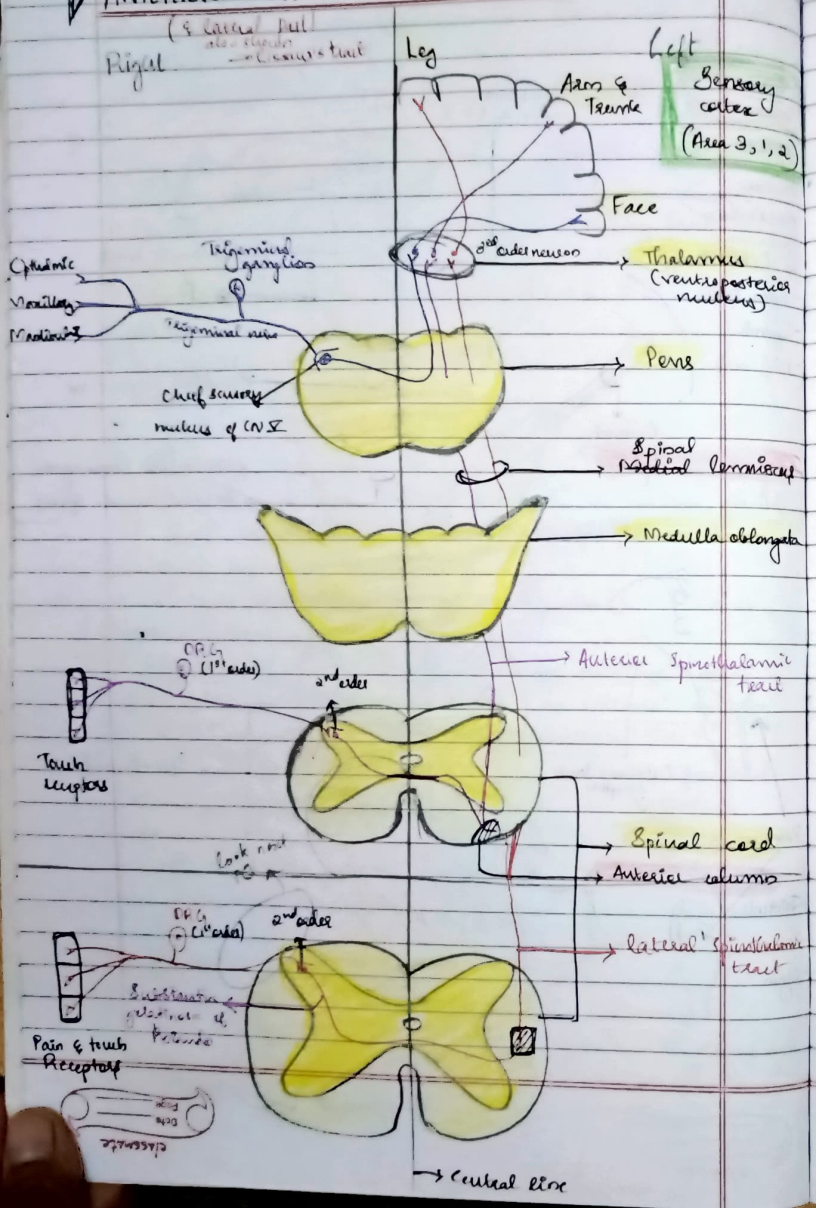
- 2nd order neurons arise from n. gracilis & n. cuneatus (dorsal column nuclei) in medulla.
- Majority of 2nd order neurons cross midline and pass into opp. side of medulla as internal arcuate fibres. They ascend up as medial lemniscus.
- Few of fibres pass through inf. cerebellar peduncle as ext. arcuate fibres to terminate in cerebellum.
  - Concerned with unconscious proprioception
- 2nd order neurons pass through brainstem to terminate in ventroposterior lateral (VPL) nucleus of thalamus.

3rd order neuron

- They arise from VPL nucleus of thalamus. They ascend through internal capsule to somatosensory area of cortex (post central gyrus, area-3, 1, 2) in parietal lobe.



ANTERIOR SPINOTHALAMIC TRACT (Right)



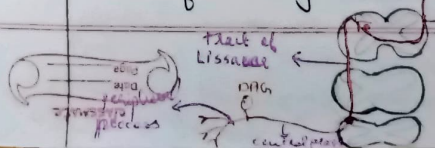
1) Sensations carried :
- crude touch, itch, tickle

2) Receptors
- Mainly : Pacinian corpuscle
- Other slowly adapting : Merkel's disc, Ruffini's end organ, Krause's end bulb

3) Neurons

1st order neurons : (Aδ and C fibres) - also conducting
- The afferent fibres from touch receptors reaching DRG are dendrites of DRG which form 1st order neurons.
- These fibres carrying crude touch sensation enter spinal cord through posterior root and relay in chief sensory nucleus in posterior horn.
- These cells form 2nd order neurons.

2nd order neuron
- 2nd order neuron arise from lamina I, II and V of SC. They cross the opposite side in front of central canal more obliquely to reach ant. column pathway.
- The arrangement of fibres in SC from medial to lateral - cervical, thoracic, lumbar and sacral.
- 2° fibres are joined in brainstem by fibres of trigeminothalamic tract (Pain & temp from face & teeth)
- In medulla, Ant STT joins with Lateral STT to form spinal lemniscus. It ascends close to medial lemniscus.
- The fibres relay in ventroposterior nucleus (VPN) of thalamus.



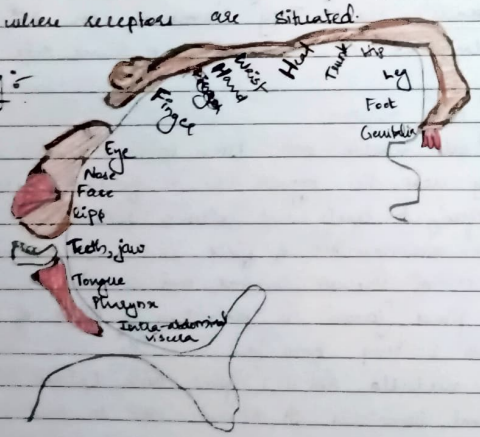
3rd order neuron

They arise from VPL of thalamus. From here, most fibres pass to medial nucleus of thalamus. The fibres ascend via posterior limb of internal capsule, diverge in corona radiata and end in sensory cortex area 3, 1, 2 in post central gyrus.

SENSORY HOMUNCULUS

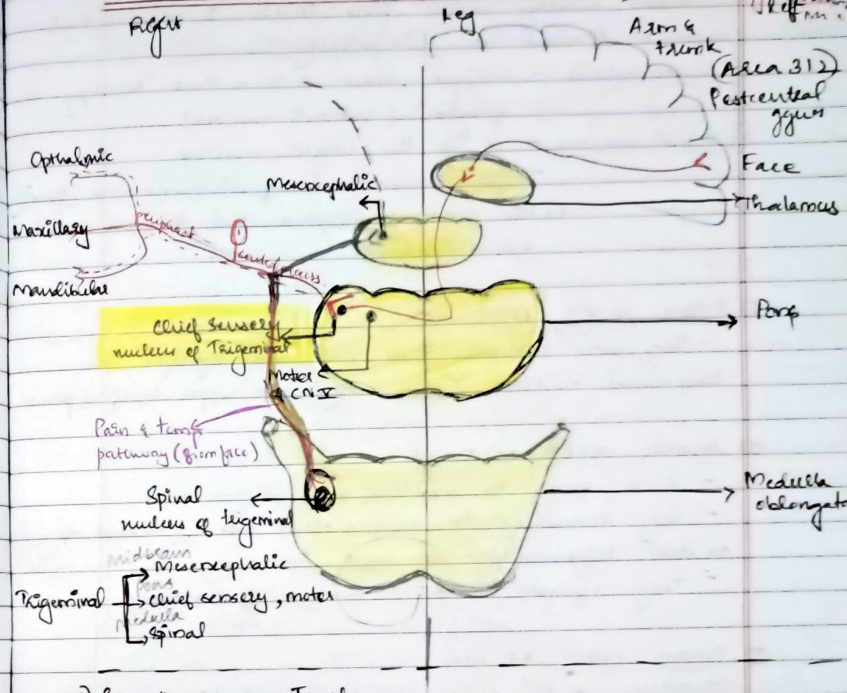
- Diff areas of body have topographical representation post central gyrus. The representation of our body appears upside down.
- The foot area occupies the most medial part of gyrus and face occupies the lateral part of gyrus.
- Areas of body which are very sensitive occupy much bigger space irrespective of actual size. Index finger, face have larger representation compared to trunk.
- When sensory homunculus is stimulated in a conscious man, the sensation is felt like coming from skin where receptors are situated.

Fig:

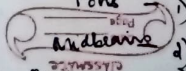


TRIGEMINAL PATHWAY FOR TOUCH

(There are other pathways also.)



1) Sensations - Touch
2) Receptor - Cutaneous mechanoreceptors and muscle proprioceptors on face
3) Neurons:
   - 1st order neuron
   - It is a pseudounipolar neuron with a process - central & peripheral
   - The cell bodies are situated in trigeminal ganglion
   - The peripheral processes radiating from TG, arrive to form 3 separate nerve - ophthalmic, maxillary, mandibular
   - The central processes of TG neuron terminate in three different components of Tg nucleus:-



1) Main sensory
2) Mesencephalic nucleus
3) Spinal

1) Main sensory carries fine touch & pressure sensations and is involved in this pathway

2) Mesencephalic - unique, since it is a true "sensory ganglion" (and not nucleus)

3) Spinal nucleus - It is the largest nucleus
- for transmission of discriminative (fine) tactile sense, nociception and thermal sensation from head

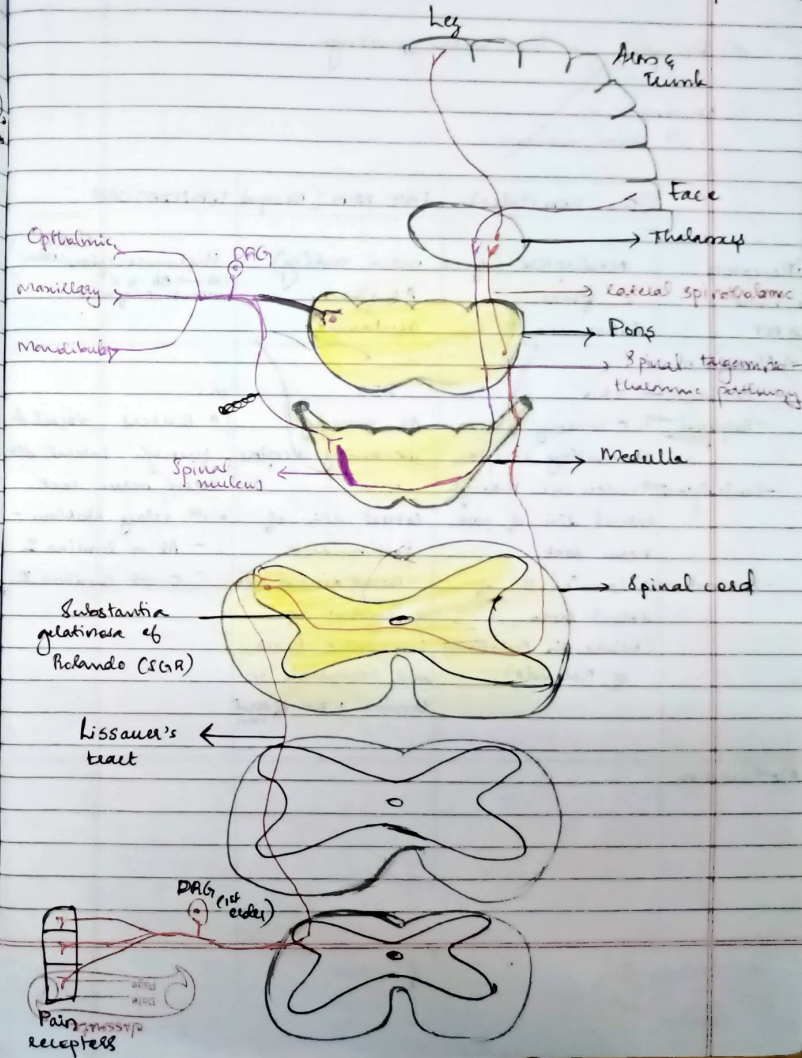
(1) FIRST ORDER NEURONS (Cranial nerve)
- They enter brainstem at pons and synapse in trigeminal nucleus (chief sensory) in upper half of pons.

(2) SECOND ORDER NEURONS
- Arise from TG nucleus
- They decussate to contralateral side, ascend as ventral trigeminothalamic tract.
- They synapse in VPM nucleus of thalamus

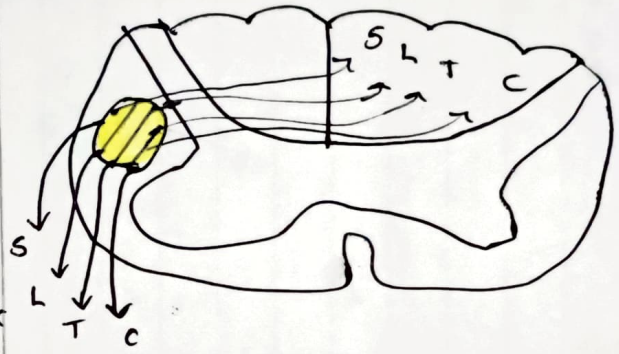
(3) THIRD ORDER NEURON
- Fibres of 3rd order neurons from VPM of thalamus travel through posterior limb of internal capsule, and synapse at 1° somatosensory cortex (Area 3,1,2) Postcentral gyrus

PATHWAY FOR TEMP SENSATION and pain sensation
- From face (spinal trigeminal tract)
- From body (lateral spinothalamic)

1) LATERAL SPINOTHALAMIC (Part of Anterolateral spinothalamic)



	SLOW PAIN	FAST PAIN	TEMPERATURE
1) Receptors	Nociceptors (free nerve endings)		Thermo → Cold → Krause's end bulb → warm → Ruffini's end organ
2) Fibers	C fibers	Aδ fibers	cold → Aδ and C warm → C fibers
3) 1st order n. - cell bodies	- DRG	DRG	• Enters dorsal horn through lateral division of dorsal n. root. • 1st relay station → Dorsal horn of SC - Aδ → lamina I, V - C → lamina II
- Peripheral process	- C sensory fibers innervating receptors	Aδ sensory fibers innervating receptors	
- Central process	- enters SC through lateral division of post- n. root	" "	
- Termination	- lamina II of dorsal horn (Substantia Gelatinosa of Rolando)	- Ascends/descends 1 or 2 spinal in Lissauer's tract and terminate in lamina I and V of dorsal horn.	
4) 2nd order neuron	- Cross to opp side in ant commissure - Reaches lateral column - Ascend as lateral STT tract	- Cross to opp side in ant commissure - Reaches lateral column - Ascend as lateral STT	- Arise from laminae of dorsal horn - Cross to opp side - Ascend as lateral STT - Terminate in posteroventral N. of thalamus

	SLOW PAIN	FAST PAIN	TEMPERATURE
2nd order (contd)	- In brainstem en route way to thalamus, fibres project to 3 nuclear groups:- 1) Medulla - reticular formn (Spinoreticular T) 2) Midbrain - periaqueductal grey (Spinomesencephalic T) 3) Hypothalamus - (Spinohypothalamic T)	- Topographic organisation in SC (medial → lateral) cervical, thoracic, lumbar, sacral →  - In medulla, joins with ventral SFT to form spinal lemniscus - Ascends close to medial lemniscus - Relay in posteroventral n. of thalamus	
3rd order neurons (thalamic radiations)	• Thalamus - Medial nuclear gp, midline & intralaminar n. (non-specific n.) • Project to diff areas of cortex including limbic cortex	• Arise in thalamus • Ascend via post limb of IC • Diverge in corona radiata • End in sensory cortex (A 3,1,2) in post central gyrus	• Arise in thalamus • Ascend via post limb of IC • Diverge in corona radiata • End in sensory cortex (A 3,1,2) in post central gyrus

HYPOTHALAMUS

- It is a part of diencephalon concerned with autonomic, visceral and endocrine functions.

Functions:-

1) Autonomic nervous system regulation
2) Body temp & body water balance
3) Control of sleep-wake cycle
4) Diurnal rhythm
5) Endocrine functions
6) Feeding and satiety centre
7) Gain satisfaction / aversion
8) Having sexual behaviour

1) ANS

Trophotropic effects: ↓ HR, CO, BP ; ↑ GIT, bladder

- Ant & medial hypo :- parasympathetic activities
- Lat & posterior hypo :- sympathetic activities

Ergotropic effects:
- pupillary dilatation
- ↑ HR, ↑ BP
- vasoconstriction, piloerections
- ↓ GIT & bladder

- Homeostasis is a dynamic balance b/w autonomic branches.

2) Body temperature regulation

- Hypothalamus is the thermoregulatory centre
- Maintaining body temp at 37°C via thermoreceptors (in skin) and thermosensitive neurons (in blood vessels).
- There are 2 centres

Heat loss centre	Heat gain centre
- Ant & preoptic hypothalamus	- Post hypothalamus
- Activated by warmth	- Activated by cold

Effects:

↓	↑
- Cutaneous vasodilation	- Cutaneous vasoconstriction
- Sweating	- Piloerections
- ↓ in heat prodn	- ↑ in heat prodn • Shivering, chemical thermogenesis, thyroxine release

Lesions → Hyperthermia (not able to loose heat when body temp ↑es)

Cause - Brings body temp to that of environ (ie, neither can loose heat nor gain heat)

3) Control of sleep-wake cycle

- Biological clock
- A 24-hr periodicity of functions of body exam - Circadian rhythm
  Examples are :- 1) Sleep wake fulness → no def centre (pons)
  2) Adrenocortical activity → suprachiasmatic nucleus
  3) Melatonin secretion
- Applied → lesion of post hypo can induce sleep
  Stimln of dorsal hypo causes sleep.

4) Endocrine fns

Hypothalamus

- (Median eminence) → Releasing & Inhibiting → Ant pit hormone
- (Supraoptic & paraventricular) nucleus → Oxytocin & vasopressin

Hypothalamo-hypophyseal portal system

5) Feeding/hunger and satiety centre

Feeding centre ⇌ Satiety centre (Balance)

↓ stimulated	↓
Lateral hypothalamus	Ventromedian nucleus of hypo
- Desire for food	- No desire for food
- Voracious apetite	- No apetite

Lesions:
- Satiety center overaction
- No desire for food
- No apetite
- Starvation
- Death

- Feeding center overaction
- Desire for food
- Voracious apetite
- Obesity

Theories:

I) Glucostatic theory

↑ in blood glucose → Ventromedial hypo. turned on → Satiety

Hunger ← Lateral hypothalamus turned on ← ↓ in blood glucose

II) Lipostatic theory

Fat deposits → ↑ed leptin synthesis → ↑ plasma leptin

acts on ↓ Hypothalamus (inhibits neuropeptide Y secretion)

↓ food intake and ↑ed lipolysis (↑ed energy expenditure)

- Leptin controls body fat deposits

III) Gut-peptide theory

Ghrelin (from oxyntic cells) → ⊕ Activate appetite centers → ↑ appetite → Release Neuropeptide Y

- food in GIT, releases hormones which inhibit food intake
  - → CCK
  - → glucagon
  - → somatostatin

6) Gain satisfaction / aversion
- Reward centers
- Punishment Centers

(7) BODY WATER BALANCE

- Thirst center is located in hypothalamus
- Water intake is ↑ed when there is ↓ in ECF volume or ↑ in plasma osmolality or both.

↑ in plasma osmolality → ⊕ → osmoreceptors (in supraoptic area) → Thirst Centre stimulated

↓ in Extracellular (ECF) fluid volume → ↓ → R.A.F.G. Mech.

- Renin → Angiotensin → Ang I → Ang II → (In diencephalon) Action on Subfornical organ and OVLT → ⊕ → Thirst centre
- ⊕ → Supraoptic nucleus (& hypo.) → ADH / vasopressin → ↑ reabsorption of water (DCT, collecting duct)

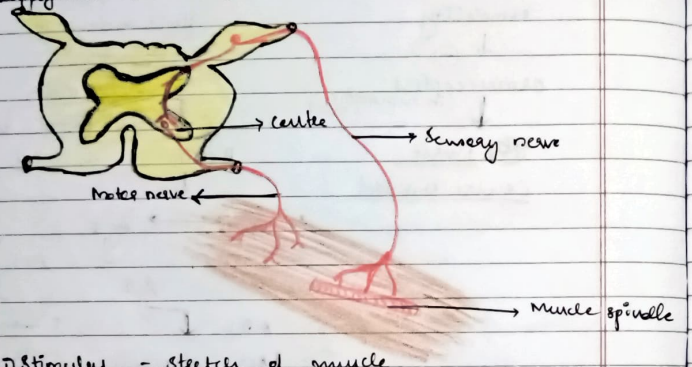
REFLEXES

1) Classify reflexes. Explain each. (Short essay)

ANS:-
1) Monosynaptic / Stretch reflexes - only 1 synapse, low affere & efferent
2) Disynaptic - Eg: Inverse stretch reflex
3) Polysynaptic - Eg: Withdrawal reflex
} more than 1 or more interneuron

STRETCH REFLEX / Myotatic reflex

- When a skeletal muscle with intact nerve supply is stretched, it contracts.



1) Stimulus - Stretch of muscle
2) Receptor - muscle spindle
3) Afferent - Group Ia and II fibres
4) Centre - Ventral grey horn of spinal cord
5) Efferent - axons of α-motor neuron supplying the same muscle
6) Neurotransmitter - Glutamate
7) Reaction time - time b/w application of stimulus and response → 19-24 msec (for knee jerk)
8) Central delay - time taken for reflex activity to traverse the spinal cord → 0.6 - 0.9 msec (for knee jerk)

(Since min. synaptic delay is 0.5 msec, ∴ only one synapse could have been traversed for this reflex activity.

MUSCLE SPINDLE (Short essay)

- Receptor of stretch reflex present in all skeletal muscles, • embryonic in nature

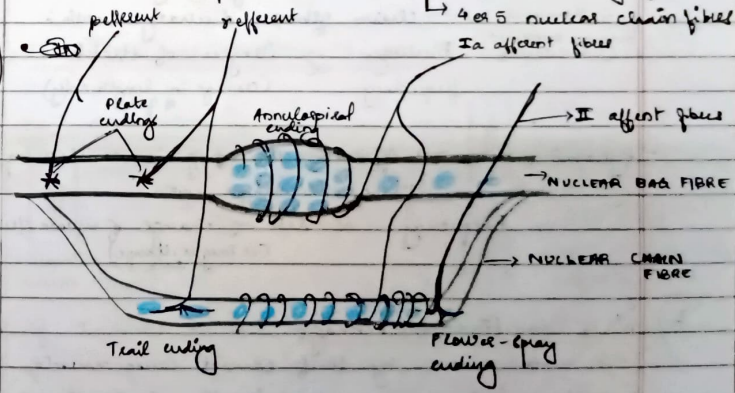
A) • Structure :-

Contain 3-10 muscle fibers enclosed in a connective tissue capsule, seen throughout belly of skeletal muscle hence called → intrafusal fibers

(• the regular contractile elements of muscle are the intrafusal fibres / skeletal muscle fibres)

- Intrafusal arranged parallel to extrafusal fibers.
- It is the non-contractile central part of muscle spindle which is sensitive to stretch. (But ends are contractile)
- There are 2 types of intrafusal fibers :-
  1) Nuclear bag fibers - contain many nuclei in central position → dynamic
  2) Nuclear chain fibers → static
     - nuclei are arranged in a chain, with no central bulging.
- Each muscle spindle contains : → 2 nuclear bag fibers, 4 or 5 nuclear chain fibers

B)



C) • Innervation → Sensory / Motor

I SENSORY

• Type Ia afferent → winds around central portion of nuclear bag and chain fibres called as annulospiral nerve endings / primary endings

• Type II afferent → located near ends of nuclear chain fibres called as flower spray ending / 2° ending

II MOTOR

• γ - efferent endings

Plate endings
- End on motor endplates of nuclear bag fibres
• β - efferents may also be seen

Trail endings
- Form extensive network on nuclear chain fibres

D) • Responses → Static / Dynamic

- Static response → Afferents originating in nuclear chain fibres discharge with ∴ stimulated only by sustained stretch

Discharge frequency ∝ Degree of stretch (change in length only) of fibre

- Dynamic response → Afferents originating in nuclear bag fibres discharge with

Frequency ∝ Rate of change of muscle stretch (or length change rate)

E) ~~α-γ~~ coactivation / linkage

• γ motor neuron by itself cannot cause muscle contraction but can cause contraction indirectly via stretch reflex.

(when α - motor neurons stimulated, γ - motor neurons also stimulated at same time.

Stretch reflex

Stimulation of γ efferents
↓
Contraction of intrafusal fibres
↓
Stretches nuclear bag portion of spindles
↓
Stimulation of Ia sensory fibres
↓
Stimln of α motor neuron
↓
Contractn of extrafusal fibres

α motor neuron
- Supplies extrafusal

γ motor neuron
- intrafusal
- pyramidal tract
- extrapyramidal
- does not affect sensitivity of spindle
- Increases muscle spindle sensitivity

F) FUNCTIONS of muscle spindle

1) Role in stretch reflex
2) Maintains muscle tone
3) Posture maintenance
4) Maintains skeletal muscle at some physiological length via stretch reflex
5) Stabilise body during tense motor action

G) ~~INHIBITION OF STRETCH REFLEX~~ (Reciprocal innervation / Withdrawal reflex)

E) α-γ coactivation

Intrafusal fibres (muscle spindle)
Extrafusal fibres
Normal muscle

Stretched
- EFF stretch
- IFF stretch

Contracted
- EFF contract
- IFF no contraction (if γ motor not present)

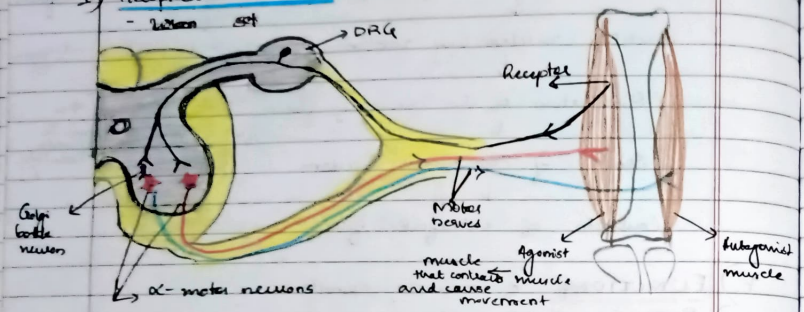
So slacking occurs

- If γ - motor neurons not present, there will be contraction of extrafusal fibres (as usual) (via α - motor neuron), but not intrafusal fibres
- So "slacking" of muscle spindle occurs ∴ Muscle spindle nerve ending becomes insensitive to further stretch.
- To prevent, the γ - motor neuron causes contraction of intrafusal fibres also.

III. BISYNAPTIC REFLEXES → Reciprocal innervation / Inverse stretch reflex

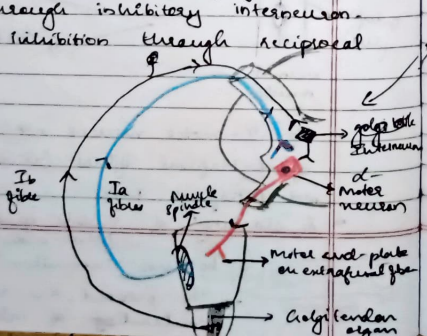
I) Reciprocal innervation

- When set



- Afferent nerve fibres from agonist muscle entering spinal cord gives collateral to an inhibitory interneuron which makes synapse with α-motor neuron that supplies the antagonistic muscle.
- This is reciprocal innervation and the interneuron is golgi bottle neuron.
- When muscle is stretched, the motor neurons supplying skeletal (agonist) muscle is stimulated and the muscle contracts. At the same time, collateral from afferent fibre inhibits the motor neuron supplying the antagonist muscle through inhibitory interneuron.
  - This is reciprocal inhibition through reciprocal innervation.

Interneuron releasing inhibitory mediates (Glycine)



locus of resemblance with pocket knife

II. Inverse stretch reflex

- As strength of stretch increases, reflex contraction also ↑es. But after a certain extent, as strength of stretch ↑es contraction ends and muscle relaxation
- This relaxation in response to strong stretch is called inverse stretch reflex (protective).

1) Stimulus - Strong Stretch
2) Receptor - Golgi tendon organ
3) Afferent - Ib myelinated nerve fibres
4) Centre - Spinal cord, the Ib fibres end on inhibitory interneurons that directly end on motor neurons
5) Efferent - α- motor neuron
6) Neurotransmitter - Glycine (Inhibitory)

GOLGI TENDON ORGAN

- Net like collection of knobby nerve endings among fascicles of a tendon
- 3-25 muscle fibres / Golgi tendon organ
- Effect :- 1) Stimln of Ib fibres causes ↓ion of IPSP on motor neurons that supply same muscle
  2) GTO is in series with muscle fibres, they are stimulated by passive stretch and active contraction of muscle.

SIGNIFICANCE

1) Protective reflex and prevents tearing of muscle by extreme stretch.
2) Important in maintaining tension during normal muscle activity.
3) Clasp-knife effect / lengthening reaction
   The sequence of resistance followed by relaxation when a limb is moved passively is known as clasp-knife effect.

Eg: If triceps is spastic, (Stiff & resistant to movement)
- passive flexion of elbow meets immediate resistance due to initiation of stretch reflex in triceps (causing extension)

→ Passive flexion of elbow
↓
Stretch reflex in triceps
↓
Contraction of triceps } Resistance to initial movement as in clasp-knife
↓
Extension of elbow
↓ further stretch
Inverse stretch reflex
↓
Resistance to flexion collapses
↓
Arm flexes
↳ Sequence may be repeated.

POLYSYNAPTIC REFLEX (Withdrawal reflex)

1) Stimulus - noxious or painful stimuli on skin, tissue, muscle
2) Response - Contraction of flexor and inhibition of extensor

Effect → the part stimulated is flexed and withdrawn from the stimulus

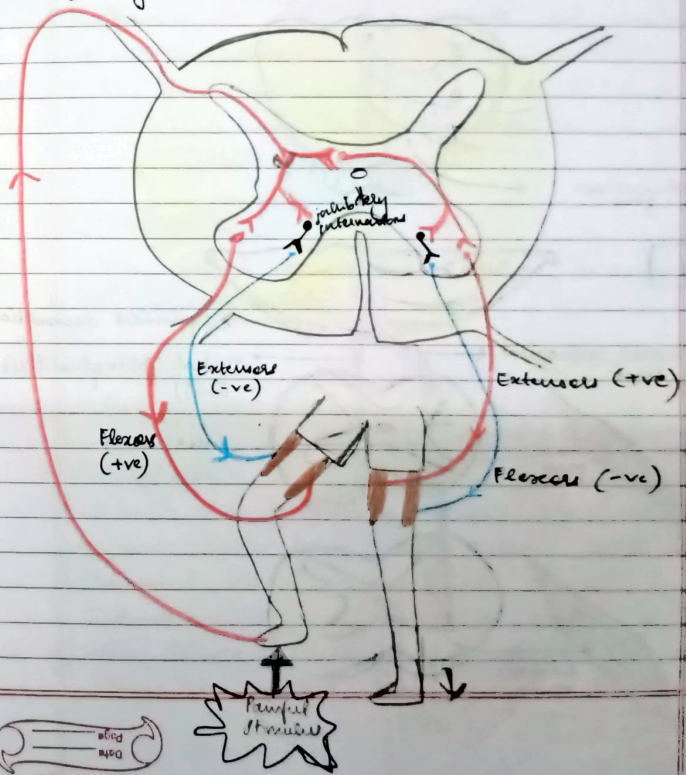
Crossed extensor reflex:
→ If stimulus is stronger, response includes
- flexion & withdrawal of that limb
- Extension of opposite limb.
↳ This maintains balance and posture.

3) Mechanism - the interneurons form several pathways and ultimately end on α-motor neuron
- Reciprocal inhibition
~~- After discharge, reverberating circuits~~

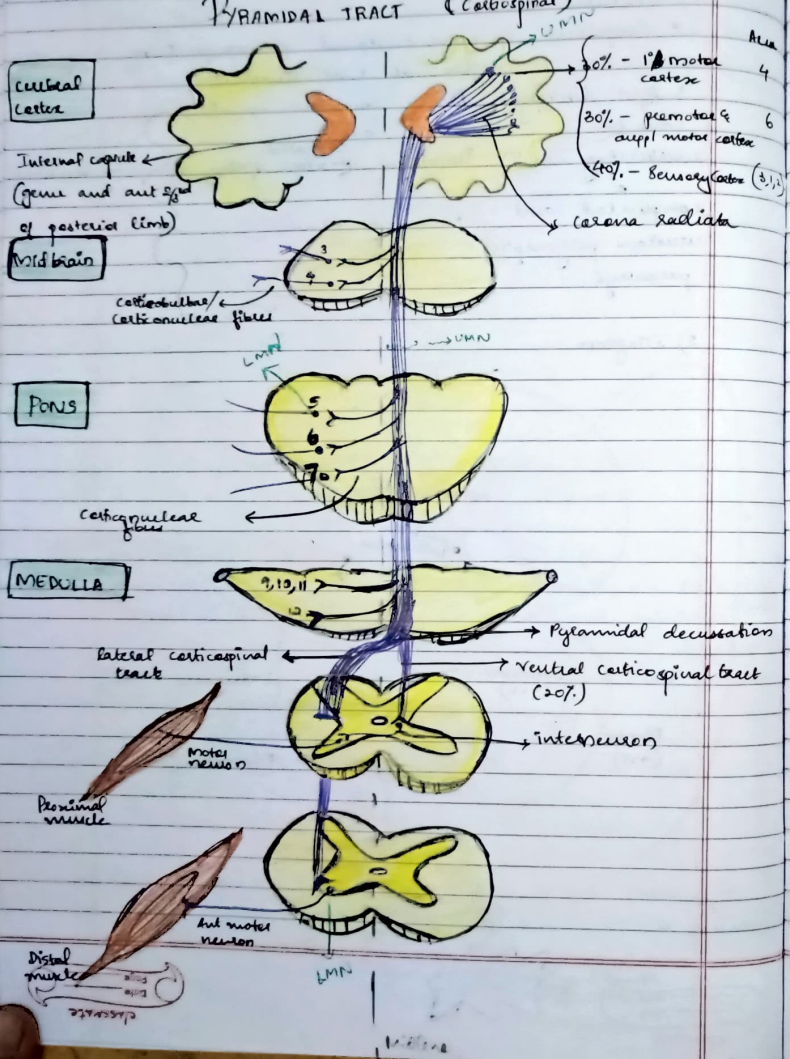
4) Features
- Prepotent - prevent spinal pathways from any other reflex at that moment
- After discharge - Strong stimulus (painful)
↓
Prolonged repeated firing of motor neurons
↓
Prolonged flexion

(due to reverberating circuits)
↓
complicated and circuitous polysynaptic pathways

5) Diagram



PYRAMIDAL TRACT (Corticospinal)



Pyramidal tract is the longest tract starting from motor cortex and reaches last segment of spinal cord.

1) Corticospinal tract : PT fibres to spinal ventral horn cells
2) Corticobulbar tract : PT fibres to motor cranial nuclei (3 and 4 in midbrain, 5,6,7 in pons, 9-12 in medulla)

COURSE

1) Arise from Betz cells of cerebral cortex
2) Converge towards brainstem as a radiating mass of fibres (corona radiata) to reach internal capsule.
3) In internal capsule :-
   - It lies in the knee (genu) and anterior 2/3rd of posterior limb.
   - PT fibres get intermingled with EP (Extrapyramidal fibres)

   APPLIED - ∴ lesion in internal capsule includes both PT and EPT.
4) In midbrain :-
   lies ventral to substantia nigra, occupying middle 3/5th of this region.
5) In pons :-
   Occupy most ventral aspect of trapezium. (commonly involved)
6) In medulla :-
   - lower part of medulla, 80-85% fibres cross to opp side, enters lateral white column called lateral corticospinal tract (crossed / indirect)
   - About 15-20%, do not cross and enter anterior white column called ant. corticospinal tract (uncrossed / direct)

   They cross midline when it reaches level of anterior (motor) interneurons.

II FUNCTIONS

1) Lateral corticospinal → fine & precise movement of fingers and hands to carry out skilled work

2) Ant corticospinal → Muscles of trunk & proximal limb to carry out postural adjustments and gross movements.

3) Corticospinal fibres from Area 1 and 2 (sensory) → sensory-motor coordination

4) Corticobulbar → voluntary control of muscles of larynx, pharynx, palate, upper and lower face

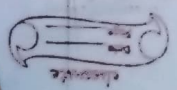
5) Corticospinal may end on → excitatory synapses / inhibitory

III Effects of lesions at various levels

a) INTRO

- Pyramidal system (PS) can be injured by cerebrovascular accidents.
- CVA forms → 1) Infarction, 2) Hemorrhage
- CVA can damage PS at various levels:-

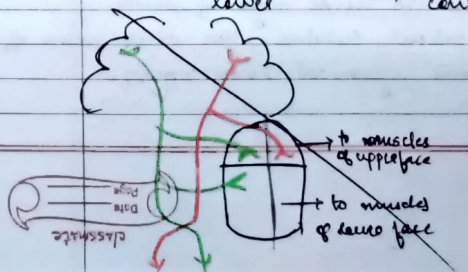
SITE	FEATURES	PHYSIOLOGICAL BASIS
Area 4	Monoplegia (contralateral side)	- PT fibres are scattered here - ∴ To cause hemiplegia, lesion in Area 4 has to be very extensive - PT fibres are crowded here that even a slight injury will cause damage to large no. of fibres (in severe → hemianesthesia → homonymous hemianopia)
Corona radiata	Hemiplegia (contralateral)	

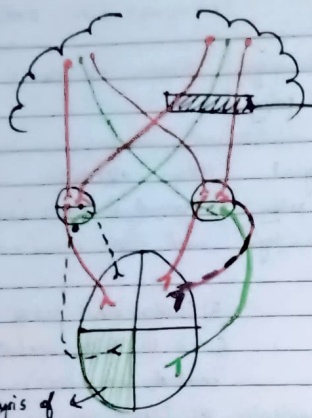


SITE	FEATURES	PHYSIOLOGICAL BASIS
Internal capsule	Contralateral hemiplegia and UMN facial palsy	Reason as above
Midbrain	Crossed hemiplegia (ipsilateral cranial n. palsy and contralateral hemiplegia)	lesion affects :- 1) Cranial n fibres (which don't cross and affect same side) 2) Corticospinal tract fibres (cross in lower medulla and control opp side)
Pons	Contralateral hemiplegia and ipsilateral (LMN) facial palsy (Cr VI may be affected)	lesion :- 1) VII, Cortico tract
Medulla	Fatal, ipsilateral n, 10, 11, 12 → (Tox), (Here no decussation, so no contralateral hemiplegia)	
Upper spinal cord level	Quadriplegia + resp paralysis	
Thoracic & lumbar level	paraplegia, resp normal	

IV) At internal capsule level → UMN facial palsy

- Motor nucleus of CN VII, has upper and lower divisions which supply upper and lower part of face
- Upper face → bilateral innervation (now)
  Lower → contralateral (but ami)





IMP TERMS

1) Hemiplegia (2) → paralysis of one half of body
- Dense hemiplegia : weakness of whole opp side
- Crossed hemiplegia : Ipsilateral cranial n. involved (LMN type of CN lesion) but contralateral weakness (UMN type)

2) Paraplegia → paralysis of both lower limbs
3) Monoplegia → paralysis of single limb
4) Quadriplegia → " " all 4 limbs

(2) Stroke → cerebrovascular accident induced damage of pyramidal system is stroke / apoplexy
- Onset of stroke is sudden
- Initially, there will be visual signs of shock
- After few days, if patient survives, the shock passes off and stage of chronic pyramidal lesion appear.

Brief answers :-

1) Differentiate UMN & LMN

	LMN lesion	UMN lesion
Def 1)	due to L of LMN neurons ie spinal and cranial motor neurons which directly innervate muscle	due to L of UMN ie, neurons that originate in cortical motor (also called) and terminate on anterior horn cells of SC. (or LMN)
2)	Usually single muscle affected	Involves grp of muscles
3)	Flaccid paralysis (hypotonia) - due to :- complete muscle tone loss as integrity of reflex arc is lost	Spastic paralysis (hypertonia) - due to :- i) Release phenomenon → loss of inhibition ii) Denervation hypersensitivity
4)	All sup and deep reflexes (tendon) are lost	• Sup reflex lost • Deep reflex exaggerated because of ↑ed γ motor discharge
5)	Disuse atrophy of muscle (- grey wasting of muscles as all are paralysed	Minimal disuse atrophy → muscles though not used in as such are paralysed voluntary movements are continuously in action to maintain posture
6)	Fasciculation and fibrillation	No fasciculation & fibs
7)	Babinski sign / Plantar reflex not elicited (Here no plantar, no extensor reflex)	Babinski sign +ve (Abnormal) (Here, extensor reflex occurs) → Normally in people plantar reflex occurs

2) Babinski sign and significance

I) Def : It is obtained by stroking outer edge of sole of foot with firm tactile stimulus.

Direction → from heel towards little toe
then medially across metatarsus

Normal (healthy) (-ve) → Produces a downward movement (plantar flexion) of great toe and small toes (-ve sign)
• Due to contn of Flexor hallucis longus

UMN lesion (+ve) → First, an upward movement (dorsiflexion) of great toe and then
Fanning out (abduction) of small toes
• Due to cont of Extensor hallucis longus
(wrong bc given physiologically → as withdrawal response)

CLINICAL SIGNS

1) Babinski once +ve in life, remains +ve for rest of life.

2) Causes :
• lesion of pyramidal tracts
• Infants (below 1yr) (ie before child starts walking)
- Develop and myelienation of pyramidal tract occur only after child starts walking
• deep sleep
• Cheyne-Stokes resp due to hypoxia

NOTE

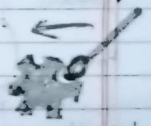
In pyramidal tract lesion
• Muscle tone is controlled by pt through α neurons and extrapyramidal (Basalganglia) and are in balance.
• In PT lesion,
inhibitory control over extrapyramidal is lost
↓
Over activn of EP system
↓
↑ed neuronal discharge (γ motor) → Hypertonia, exaggerated deep tendon reflexes

3) Differentiate spasticity (clasp-knife phenomenon) and rigidity

	SPASTICITY	RIGIDITY
Seen in	• lesion of PT (most common → internal capsule)	• lesion of basal ganglia (∴ called extrapyramidal rigidity)
2)	Only one group of muscle is involved (either agonist or antagonist)	• Involve both agonist and antagonist
3)	Hypertonia (depends on direction and velocity) ↳ ∴ clasp-knife phenomenon	• Hypertonia (but not direction or velocity dependent) ↳ 1) 2) i) Lead pipe rigidity ii) Cog-wheel rigidity

Lead pipe rigidity
- Passive movement of extremity meets with plastic clay feeling resistance as through bending a lead pipe

Cog wheel rigidity
- Resist to passive movement of extremity where there is series of catches
- Similar to a lever rubbing on teeth of a cogwheel



4) Differentiate slow pain and fast pain.

Ans:

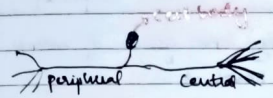
SLOW PAIN (paleo)	FAST PAIN (neo) → localised
1) C fibres	A δ fibres
2) Burning, aching, dull type	Sharp, pricking type
3) Poorly localised (non specific projections)	Well localised (topographic organisation in thalamus and cortex is precise)

• Sensory cortex → neurons arranged in modality specific columns

3) Associated with tissue destruction — Acc with cut or electrical injury

4) Occurs both in skin and deeper tissue — Do not occur in deep tissues (usually skin)

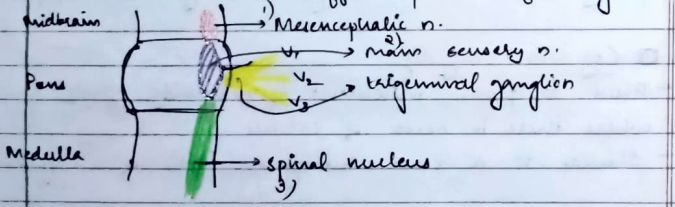
5) Trigeminal pathway



SENSATIONS carried
- Touch (tactile)
- Pain & temp
- Proprioceptive impulses from jaw muscles

} from face, oral cavity including teeth

1st order neurons
- Cell body - in trigeminal ganglion
- Peripheral process - 3 divisions of trigeminal n. (ophthalmic, maxillary, mandibular)
- Central processes
  - divide into ascending / descending branches
  - terminates in diff components of trigeminal n.



1) Mesencephalic : From Pons to midbrain
   - Proprioception
2) Main sensory : From pons
   - Tactile sensation
3) Spinal nucleus : Medulla and extends to SC
   - Pain & temp

2nd order neurons
- In three components of trigeminal n.
- Axons cross to opp side
- Ascend as trigeminal lemniscus
- Terminates in posteroventral n. of thalamus

3rd order neurons (Thalamic radiations)
- Arise in thalamus
- Ascend via post limb of IC
- Diverge in corona radiata
- End in sensory cortex (A 3,1,2) in post central gyrus

TOPOGRAPHIC REP
- Face region : most medial part of n.
- Arm " : middle part of n
- Leg " : lateral most part of n.

REFERRED PAIN

- Pain felt in an area away from the actual side of its origin
- A characteristic of visceral pain
- Pain from a visceral structure is felt in some somatic str. that may be at considerable dist away
- Aids to diagnose various diseases.
- Deep somatic pain may also be referred
- ~~Radiation of pain~~ → both local and referred

Eg:-

1) Cardiac pain	Inner aspect of left arm
2) Diaphragmatic	Tip of shoulder
3) Ureteric	Testicles
4) Maxillary sinus	Nearby teeth
5) Appendicitis	Umbilicus

MECHANISM of referred pain

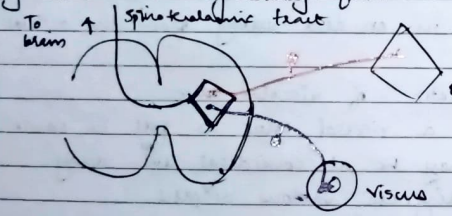
1) Dermatomal rule
2) Convergence theory
3) Facilitation "

1) Dermatomal rule

- Visceral pain is referred to a structure that developed from same embryonic segment / dermatome as the str in which pain originate
  - Diaphragm, tip of shoulder ⟶ (C_3-C_4 → Phrenic n.)
  - Heart; arm ⟶ (C_3-T_5)

2) Convergence theory

- Convergence of somatic (peripheral) and visceral pain fibres on same 2nd order neuron that project to brain.
- Since the somatic pain pathway is more frequently stimulated and pain originating from viscera will be interpreted by brain as originating from the somatic str.



3) Facilitation theory

- Fibres from visceral organs give collateral to neurons of pathway of pain from cutaneous region
- Subliminal fringe effect
- When visceral pain stimulus is activated, because of subliminal fringe effect the excitability of dorsal horn neuron which receive impulses from somatic str is Ted
- Causes sensation of pain in skin

MODIFICATION OF PAIN at spinal and supraspinal level

1) Stress analgesia

- Soldiers wounded in heat of battle often feel no pain till battle is over
- Amygdala —send→ motivational and affective response to pain
  ⟶ Release of NE from brainstem catecholaminergic neuron
- Euphoria ⟶ Release of cannabinoids

2) Descending analgesic sys / Endogenous pain relief system

(Body's intrinsic mech)

Activated by pain, stress
↓
Periaqueductal grey (midbrain)
↓
Raphe magnus n. (medulla)
↓
Sends serotonergic and enkephalinergic projections to SC
↓
Presynaptic and postsynaptic inhibition of Aδ and C fibres by release of → opiods, serotonin, norepinephrine in dorsal horn of SC
↓
Inhibit 2nd order pain neuron
↓
Reduces pain transmission to brain

BASAL GANGLIA - essay

I) INTRO

Basal ganglia is a group of nuclei that influences pyramidal system (motor) activity.

II) COMPONENTS

1) Caudate nucleus } Striatum
2) Putamen
3) Globus pallidus → External seg (by medial medullary lamina)
   → Internal seg
   (lenticular nucleus)
4) Subthalamic nucleus
5) Substantia nigra → Pars compacta
   → Pars reticularis

dopamine and connects to striatum

INPUTS

1) Corticostriate projection : cerebral cortex → Striatum
2) Thalamostriate // : Centromedian n. of thalamus → Striatum
3) Raphestriate // : Dorsal raphe n. → Striatum

OUTPUTS

- Efferent connects from basal ganglia are mainly to
  - Thalamus
  - Reticular form
  - Hypothalamus
  - Red nucleus
  - Substantia nigra
  - Subthalamic nucleus

III) CONNECTIONS (within basal ganglia)

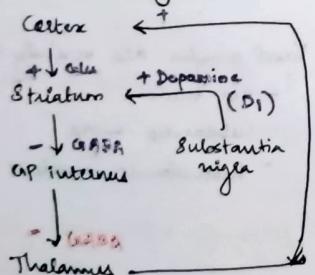
A) Nigrostriatal projections
- Substantia nigra → Striatum
- Dopaminergic
- Degeneration of this system → Parkinsonism

B) Striatonigral projections
- Striatum → Substantia nigra
- GABAergic (Inhibitory)
- Degeneration of this → Huntington's disease

C) Intrastriatal projections
- Btw cells of striatum
- Cholinergic (ACh → excitatory effect)

IV) NEURAL PATHWAY THROUGH BASAL GANGLIA — Direct / Indirect

I) Direct pathway – stimulated during movement
Lesion → Parkinsonism



Cortical–basal ganglia–thalamic–cortical loop

- To initiate voluntary movements, motor cortex must inhibit basal ganglia
- Motor cortex does this by activating GPi (GABA release)
- Since GPi is inhibited, it can't inhibit thalamus
- ∴ Activation of striatum finally excites the thalamic neurons (disinhibition)
- Now, thalamus can now activate target neurons in motor cortex for all voluntary movements via thalamocortical projection.

II) Indirect pathway → Huntington disease
lesion

- It is to terminate the initiated movement.
- GPi is activated here
- ∴ There is inhibition of thalamus → hence inhibit the thalamocortical projections

III) Modulation by nigrostriatal projections
- Dopaminergic projections from pars compacta of substantia nigra to striatum appear to have excitatory influence on direct pathway (D_1 receptor) and inhibitory on indirect pathway (D_2 receptor)

V) FUNCTIONS

1) Role in planning of motor activity
Mech → Activity in the circuit btw subthalamic n. and globus pallidus

2) Control of muscle tone

Basal ganglia inhibits stretch reflex (muscle tone) by stimulation of striatum and inhibitory cortex.

3) Cognitive function

- Caudate nucleus plays a role in cognitive process (thinking of brain)
- Lesion of head of left caudate nucleus is associated with ⟶ dysarthric aphasia (difficulty in articulating words)

4) Subconscious execution of movements

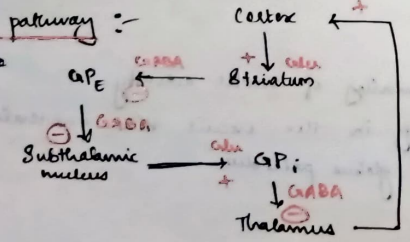
- BG controls normal automatic and associated movements like swinging of arms during walking.

5) The activity of BG ↑es during slow, steady movement and is silent during rapid movements.

6) Gp provides muscle tone for skilled movements.

7) Substantia nigra is centre for coordination of impulses essential for skilled movements

Indirect pathway :-



DISEASES OF BASAL GANGLIA

1) Parkinsonism (Paralysis Agitans / Shaking Palsy)

It is caused due to degeneration of substantia nigra dopaminergic neurons in substantia nigra pars compacta

(a) CAUSES

1) Primary or idiopathic
2) Complication of drugs such as phenothiazine which blocks dopamine (D_2) receptors
3) Complication of encephalitis

Symptoms appear when 60–70% of nigrostriatal dopaminergic neurons are lost.

(b) FEATURES ⟶ Both hyperkinetic and hypokinetic

I) Hypokinetic features (Direct pathway not excited by dopamine)

- Bradykinesia : slowness of movement
- Akinesia : Difficulty initiating movements and spontaneous movements
- Mask like face : Due to ↓ in facial muscle movement (expressionless)
- loss of subconscious : As activity of BG decreases movements (swinging of arms during walking)
- Tendon reflex difficult to elicit ⟶ as rigidity ↑es

II) Hyperkinetic features (indirect pathway not inhibited by dopamine)

1) Rigidity

- Due to ↑activity of neostriatum is influenced by excitatory effect discharge of cholinergic interneurons and inhibitory dopaminergic input in striatum.
- Lack of dopaminergic activity
  ↓
  Shift to excitatory cholinergic
  ↓
- Increased discharge of ↑ efferents
  ↓
  large proximal gp of muscles (both agonists and antagonists)
- Posture is → flexion attide
  - Back flexed
  - Arms adducted and flexed
  - Knees bent (Stooped posture)

→ Cog wheel rigidity → Series of "catches" during passive motion
→ Lead pipe rigidity → plastic dead feeling resistance as though bending a lead pipe (during passive motion)

2) Tremor

- Consists of regular, rhythmic alternate contraction of antagonist and agonist muscle at 6-8 times/sec.
- Seen as :- Pill-rolling movements
  i.e, rhythmic contraction of thumb over 1st two fingers
- It is present only during rest, hence called resting tremor.
- During sleep it disappears (due to ↓ activity of PLAS)

3) Retropulsion :

If patient pulled back, he begins to walk backwards and is unable to stop himself

III) Gait (Festinant type / shuffling gait)

Patient is bent forwards and walks quickly with short steps as if trying to catch up the centre of gravity / preventing himself from falling down.

TREATMENT

1) Administration of L-dopa + carbidopa (peripheral decarboxylase inhibitor)
   - Dopamine cannot cross BBB. But L-dopa can cross and enters brain tissue where it is converted to dopamine

2) Transplant of fetal basal ganglia (from aborted fetus)
   - Dopamine secreting cells have been implanted into caudate nucleus and putamen.

3) Monoamine oxidase (MAO) inhibitors

4) Anticholinergics

5) Surgical treatment by making lesions in Gpi or subthalamic nucleus to restore output balance

(B) Interruption of inhibitory pathway

2) CHOREA
- due to caudate n. involvement
- characterised by rapid irregular involuntary "dancing" movements of short duration
- ↓ed muscle tone and muscle weakness

3) ATHETOSIS
- lesion of lenticular n.
- characterised by continuous slow twisting / writhing movements

4) BALLISM
- lesion : ~~contralateral~~ subthalamic n.
- Involuntary flailing, intense and violent movements

5) Hemiballism
- lesion : contralateral subthalamic n.
- It is ballism that affects only one side

6) Wilson's disease / Hepatolenticular degeneration
- Autosomal recessive disease where the Cu content of substantia nigra is high.
- Reason :- Deficiency of Ceruloplasmin (Cu-binding protein)
- Thus, Cu accumulates
↓
Degeneration of lenticular n. + cirrhosis of liver
Also, tremors and rigidity

7) Huntington's disease
- Autosomal dominant
- due to damage of GABA-ergic and cholinergic neurons that project to putamen.
- Damage to this inhibitory pathway, results in hyperkinetic features :-
  - hyperkinetic choreiform movements
  - slurred speech
  - Progressive loss of memory (dementia)

(D) No treatment → death.

Note :- cholinergic is generally excitatory in basal ganglia, but certain areas inhibitory

CEREBELLUM

Cerebellum is situated posterior to brainstem and influences motor activities both voluntary (via pyramidal) and involuntary (via extrapyramidal).

I) Functional divisions / lobes

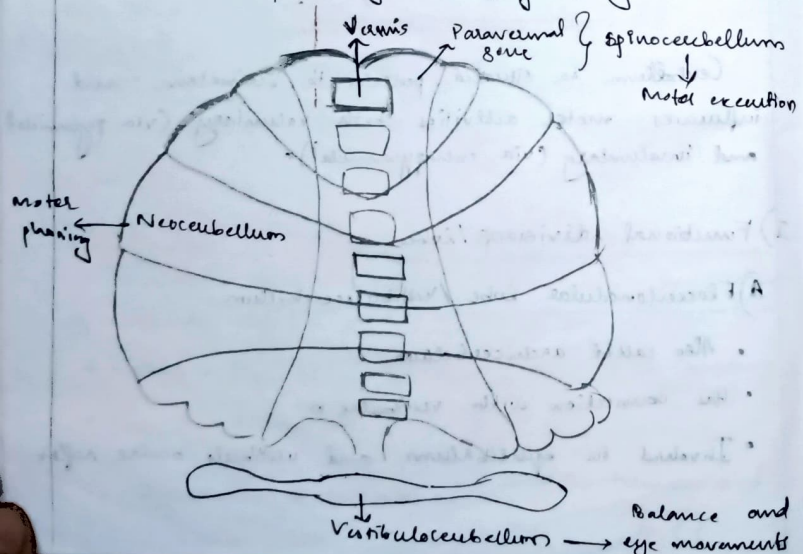
A) Flocculonodular lobe / Vestibulocerebellum
- Also called archicerebellum
- Has connection with vestibular n.
- Involved in equilibrium and vestibulo ocular reflex

b) Spinocerebellum / Paleocerebellum

- Consists of vermis and paravermal regions
- Receives proprioceptive and other sensory inputs from motor cortex.
- Vermal portion → controls axial and proximal limb muscle and controls posture
- Paravermal region → influences distal limb muscles
  - controls skilled voluntary movements

c) Cerebrocerebellum / Neocerebellum

- Consists of 2 main cerebral hemispheres
- Also called corticopontocerebellum since cerebral cortex projects to neocerebellum through pontine nuclei
- Involved in planning and programming of movements.



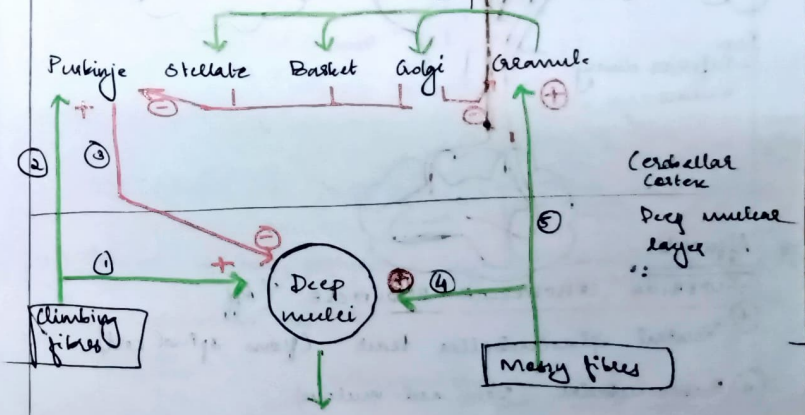
II MICROSCOPIC STRUCTURE

- 5 types of neurons : Stellate, Basket, Purkinje, Granule, Golgi
- These neurons are in 3 layers :-

1) Molecular layer : Most superficial and is made of Stellate and Basket cells.
2) Purkinje cell layer : a single layer of large flask-shaped Purkinje cells and dendrites of Purkinje cells.
3) Granule cell layer : Contains granule and golgi cells.

III NEURONAL CIRCUITS

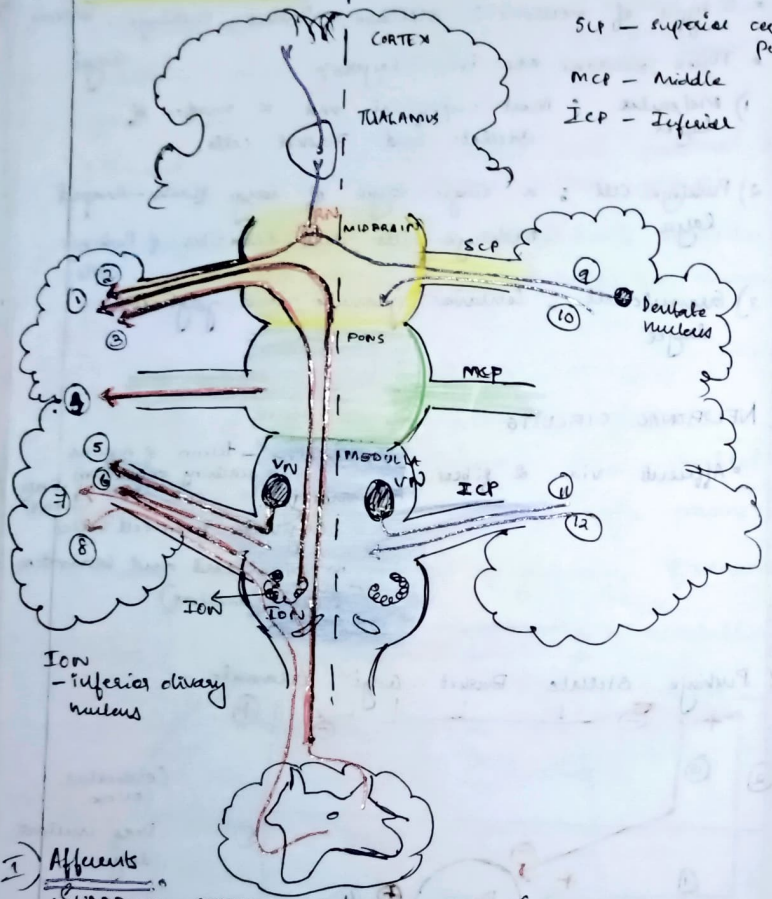
- Afferents via 2 fibers
  - climbing – terms of axons reaching cerebellum (from inferior olivary nucleus)
  - mossy – originates from cell bodies in spinal cord and brainstem (spinocerebellar)



IV CONNECTIONS

Afferents | Efferents

VN – 3 nuclei
RN – red nucleus
SCP – superior cerebellar peduncle
MCP – Middle
ICP – Inferior



ION – inferior olivary nucleus

1) Afferents

SUPERIOR CEREBELLAR PEDUNCLE (Aff)

1. Ventral spinocerebellar tract (from spinal cord)
2. Rubrocerebellar (from red nucleus)
3. Olivocerebellar (from inf olivary nucleus in medulla)

MIDDLE CEREBELLAR PEDUNCLE

4. Pontocerebellar (from pontine n.) (also pontocerebellar)

INFERIOR CEREBELLAR PEDUNCLE

5. Vestibulocerebellar (from vestibular n. in medulla)
6. Reticulocerebellar
7. cuneocerebellar (from accessory cuneate nuclei)
8. Posterior spinocerebellar (from ipsilateral thoracic nuclei – clarke's column of SC)

II Efferents

SCP → 9. Dentato-rubro thalamo cortical tract
10. Cerebello reticular

MCP → Nil

ICP → 11. cerebello vestibular
12. cerebello reticular

V FUNCTIONS OF CEREBELLUM

Tracts involved :-

- Spino (ventral part) → unconscious proprioception
- Vestibulo and cerebello vestibular → posture
- Dentato rubro thalamo cortical → error detection and correction

1) Control of body posture and equi

- Flocculonodular lobe
- Vestibular apparatus → A → Vestibular nuclei → vestibulospinal tract → Spinal motor neurons
- Vestibular nuclei → Flocculonodular lobe → E → Vestibular nuclei

2) Control of muscle tone and stretch reflexes

- Spinocerebellum
  ↓ facilitates
  γ motor neuron release (SSC)
  ↓ modify
  Motor neurons ⟶ Regulate muscle tone

3) Control of voluntary movements

- Cannot initiate movement, but coordinates
- Coordination of movements is a result of appropriate regln of time, rate, range, force and direction of muscular activity

4) Control of eyeball movements

- Vestibular nuclei ~~III, IV~~, VI nerves that supply extraocular muscles are under cerebellar control (by vestibular nuclei)

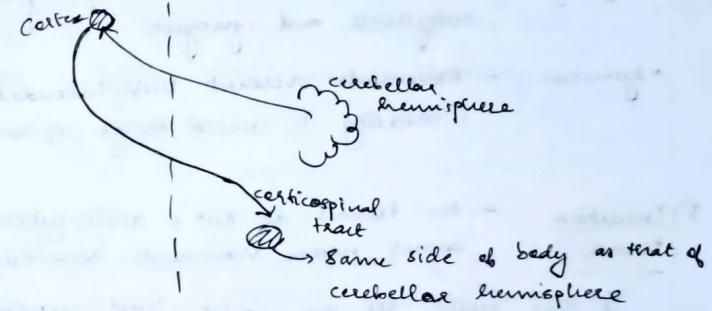
5) Balancing action and damping effect

→ Cerebellum is capable of producing suppressor effect on cerebral facilitating level

LESIONS OF CEREBELLUM

• Lesion site	Area
1 hemisphere	↦ dysfunction on same side of body
Vermis	↦ Affects both sides (due to double crossing)

DOUBLE CROSSING



Characteristic features

1) Disturbance of posture (Ataxia)

- Atonia → muscle tone completely lost
- hypotonia → markedly led muscle tone

} due to less of facilitatory effect on vestibular nuclei

ATTITUDE

- Trunk bent with concavity toward affected side
- Face rotated towards opp
- Homolateral shoulder lowered

- Nystagmus
- Pendular knee jerk → after the initial reflex response, the leg on falling continues to swing to and fro. This is due to hypotonia of Quadriceps

II Disturbance of voluntary movements

A) Ataxia – Lack of coordination of movements due to errors in rate, range force and direction of movement.

- Decomposition of movement – movement occurs in stages
- Asynergia – Lack of coordination btw protagonists, antagonists and synergists
- Dysmetria – Movements overshoot their intended mark (Inability to control range of motion)

B) Intention tremor – No tremor at rest, such patients cannot perform movements smoothly

If they reach for an object, their movements are jerky and accompanied by oscillating to and fro tremors that become more marked as hand approaches the object.

TESTS :- 1) Finger nose test
2) Rebound phenomenon
3) Adiadochokinesis → inability to perform rapid, alternating movements
4) Heel-knee test

C) Gait – clumsy manner, feet wide apart.
– Drunken gait → walk in zigzag line and deviates to affected side due to hypotonia

D) Speech – slurred, scanning type.
(due to imperfect use of movements of laryngeal muscle and tongue).

GATE CONTROL THEORY

- The theory explains how a non-noxious/non-painful input can suppress pain perception at level of SC, like a "gate" that modulates incoming pain signals after reaching brain.

I) MECHANISM

Components

"GATE" locas :- dorsal horn of SC (especially in substantia gelatinosa) in lamina II

- Controls : Flow of pain from peripheral nerves to brain
- Non-painful stimuli (like rubbing) : Closes the gate
- GATE is an → Inhibitory interneuron

Fibers	Fn	Effect on gate
Aδ and C	carries pain sensation	Open the gate
Aβ fibers	Touch, pressure, vib	close the gate

II) Working :-
1) Painful stimuli activate Aδ and C, send pain signal to SC (like bump your elbow)
2) Non-painful stimuli (Eg: rubbing the area) activate

A-β fibres.

- Aβ fibres activate inhibitory interneurons in dorsal horn.
- Interneurons inhibit transmission of pain (by closing the gate)

SIGNIFICANCE

1) Rubbing the skin near painful areas can relieve pain.
2) Counter irritant appl
3) Acupuncture
4) Transcutaneous electrical nerve stimulation (TENS)
   - use electrodes to activate A-α, Aβ fibres in vicinity of injury.

FEATURES

1) 1st theory to propose the effect of psychological factors in pain perception

Eg:- depression can open the gate
- Unhealthy life choices open

LESIONS OF SPINAL CORD

Exam

1) Transection of spinal cord (esp. hemisection)

Ans:- It can be divided into - complete, incomplete and hemisection

I) COMPLETE TRANSECTION

- Causes → motor vehicle accident, gunshot, spasticity etc
- Most common site → mid thoracic level

STAGES (3)

i) Stage of spinal shock (/flaccidity stage)

It is the 1st stage of effects that occurs immediately after injury. This stage depends on site of injury:-

- CT at C_3 is fatal
- below C_3 → deprived of sensations and motor activity

Cause of this stage → sudden removal of impulses from higher centres

Duration & severity of shock depends
- evolution of animal
- degree of encephalisation → oligaschinsis
- duration in humans → 3 weeks

Effects → motor, sensory, visceral and vasomotor effects

I) Motor effects

- Paresis - depending on level of section; paralysis are of following types:-
  - paraplegia → paralysis of both lower limbs
  - Quadriplegia → " " all 4 limbs
  - hemiplegia → " of ½ body

- Tone → loss of tone
- Reflexes → superficial and deep reflexes are lost

II) SENSORY EFFECTS

- loss of all sensations below level of lesion

III) VISCERAL EFFECTS

- Urinary bladder → retention of urine
- Rectum → constipation
- Sexual reflexes → not present

IV) AUTONOMIC / VASOMOTOR

- lesion to L_2 → no effect on BP
- lesion at or above T_1 segment → marked fall in BP (M.A.BP → 40 mm Hg)
  - Retards circulation and venous return (due to bed muscle tone)
  - No sweating → ∴ skin dry
    ∴ skin becomes cold and cyanotic (blue)
  - Bed sores develop (later)

ii) Stage of Reflex activity

It is the stage of recovery. After 3 weeks occurs depending on the general health of patient.

Recovery pattern :-

Tone (in following order)

i) Smooth muscles of bowel and bladder
ii) " " of blood vessels
iii) Skeletal muscles

~ Reflex activity (after few weeks of muscle tone return)

1) Flexor reflexes → i) Babinski appears first (unusually)
2) Mass reflex
3) Tendon jerk → i) Knee jerk (unusually)
   ii) Ankle jerk

TONE

i) Smooth muscles of bowel and bladder

- Reflex emptying of rectum (defecation)
- Reflex " " urinary bladder (but its partial, so some amt of urine remains in bladder (contaminated bladder) → recurrent cystitis)

ii) Smooth muscles of blood vessels

- BP restored to normal (feedback reflex absent) ∴ fluctuations in BP may occur
- Sweating
- Bed sores heal up rapidly

iii) Skeletal muscle recovery

- Tone of flexors return 1st, so now both flexors and extensors are hypotonic. But flexors are less hypotonic. Hence limbs adopt a position of slight flexion and the paralysis is called paraplegia in flexion

MASS REFLEX (BA)

- developed after several weeks or months
- Stimuli (scratching, pricking, pinching) below level of lesion :-
  - Spasm of flexor muscles of both limbs
  - Reflex evacuation of bladder and bowel
  - Profuse sweating

→ Cause : Denervation hypersensitivity

- Spinal neurons become hypersensitive to NTs due to increase in the no. of Ach receptors.
- Development of collaterals providing additional synaptic connections.

→ Importance of this reflex : Patient can use this reflex to get some degree of control on bowel and bladder

iii) Stage of reflex failure

- Occurs when general condn of patient deteriorates

Here,
- Muscles become flaccid → undergoes wasting
- Reflexes become difficult to elicit
- Threshold for stimulus ↑es
- Mass reflex is abolished

Management
- Good nursing care
- Good nutrition and maintenance of fluids
- Prevention of infections
- Rehabilitation
- Physiotherapy

II) INCOMPLETE TRANSECTION

- Has 3 stages. 1st and last stages are similar to complete transection.

II) Stage of Reflex activity (Features of UMN lesion)

- Tone appears in extensors first
- Vestibulospinal, reticulospinal tracts escape injury
- Paraplegia in extension
- Extensors (stretch reflex) return 1st :-
  - Extensor thrust reflex
  - Crossed extensor reflex

Sensory disturbances (other than transection)

1) Tabes dorsalis / Syphilis
   - In this condn, degeneration of post (dorsal) nerve roots occur.
   - Affects fibres which convey pain

FEATURES (Both sensory and motor fn affected)
- Numbness or paraesthesia and lightning pains
- Atonicity of bladder
- Loss of position sense, vibrations, passive movements
- Deep tendon reflexes lost (knee, ankle)
- Marked dist of voluntary movements
- Loss of pain sensations particularly in joints leads to deformities of joints. There is no proper support and movements at joints become uncontrollable → charcot's joint

2) Syringomyelia → Grey matter around central canal of SC shows excess growth of neuroglial tissue with cavity formation

Essay

Hemisection of Spinal cord (Brown-Sequard Syndrome)

- Lesion involving one lateral half of SC

Causes :- tumor, trauma (gun shot, stab wound), Infection, inflammations

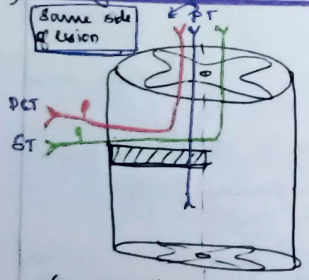
Symptoms

- Autonomic fun are usually normal
- the other functional changes that take place can be divided into 3 categories:-

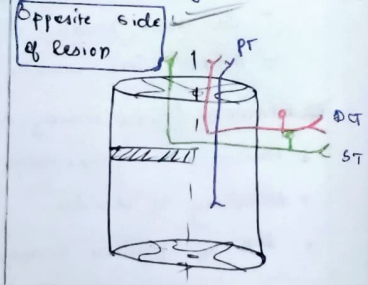
1) changes below / above level of lesion
2) " at level of lesion
3) " below level of lesion

DCT → dorsal column tract
ST → spinothalamic tract
PT → Pyramidal tract

1) Changes above level of lesion

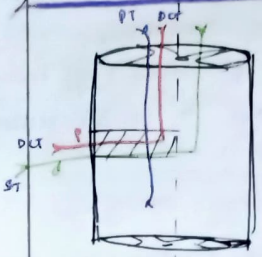


- **Sensory changes**
  - DCT & ST intact (while the sensations ascend)
  - But since they send collaterals downwards → results in a band of hyperaesthesia
- **Motor changes**
  - PT affected → twitching of muscles



- **Sensory changes** → No
- **Motor changes** → No (PT intact)

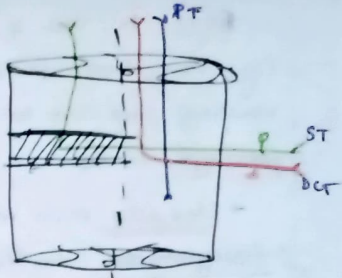
2) At level of lesion



(Same side of lesion)

- **Sensory** - DC → all sensation (Crude vibration, touch) lost
  - ST → " (crude pain/temp)
- **Motor** - Complete LMN type of paralysis

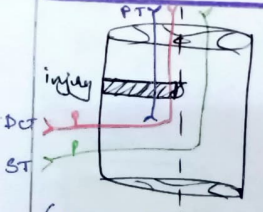
↝ Permanent vasomotor paralysis



(Opp side of lesion)

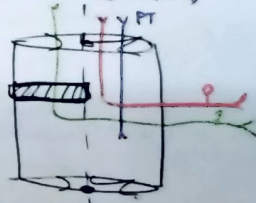
- **Sensory** - ST damages → loss of pain, temp, crude touch
  - DCT intact
- **Motor** → no changes

3) Below level of injury (Ipsilateral DCT lesion and contralateral spinothalamic tract lesion)



(Same side of lesion)

- **Sensory**
  - loss of DC sensation
  - Pain, temp, crude touch not lost
- **Motor**
  - UMN type of paralysis



(Opp side of lesion)

- **Sensory**
  - No loss of DC sensation
  - Pain, temp, crude touch lost
- **Motor**
  - No changes

- **Vasomotor changes** → temporary loss of vasomotor tone

DISSOCIATED ANAESTHESIA

Refers to loss of some type of sensations (Eg: Pain, temp) with preservation of others (Eg: touch, vibration) → due to selective damage to specific sensory pathways in SC

- Classically seen in :-

- Syringomyelia → (Here, damage occurs to decussating fibres of ST
- Intramedullary SC lesions (like central cord syndrome)

→ So in syringomyelia → pain & temp lost
→ touch, vibration, position intact

LIMBIC SYSTEM

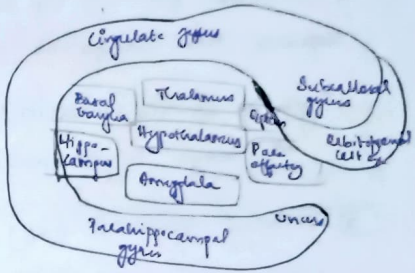
PA

1) Functions and connections of limbic system

ANS:- The limbic system consists of 4 parts :-

I) Limbic lobe / cortex (COPS UP)

- Cingulate gyrus
- Orbitofrontal cortex
- Parahippocampal gyrus
- Subcallosal gyrus
- Uncus
- Paraolfactory area

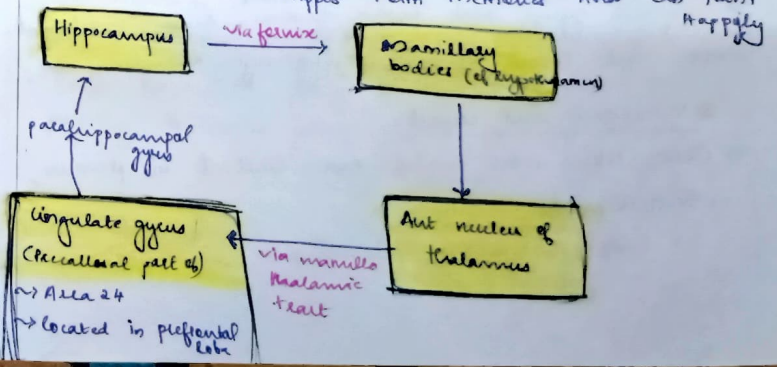


II) Subcortical structures (HATS B)

- Hypothalamus
- Amygdala
- Thalamus (Ant nucleus)
- Septum area
- Basal ganglia (portions)

III) CONNECTIONS ~ Papez circuit

Hippos Form Memories And Can Paint Happily



- Papez circuit refers to a closed circuit formed by connections blw cingulate gyrus, hippocampus, mammillary bodies and ant nucleus of thalamus

FUNCTIONS

1) Autonomic fns → eg amygdala (stimln) produces autonomic responses like changes in cardiovascular, resp, GIT (through hypothalamus)

2) Regln of feeding behaviour
→ through hypothalamus and amygdala

3) Emotional behaviour (BA)

- Emotion recognition and generation

~ amygdala and prefrontal cortex helps identify emotional stimuli (Eg:- fear, anger)

- Emotional memory

~ hippocampus links emotions with past experiences
Eg:- Fear seeing a snake

- Behavioural response

~ Cingulate gyrus and hypothalamus (prefrontal cortex) helps coordinate motor and social behaviour (Eg:- crying, laughing etc)

- Motivation and reward

~ Areas like septal nuclei are involved in pleasure, motivation and addiction.
→ Imp in positive emotions

4) Regulation of sexual behaviour

- the basic sex drive is a fn of limbic system and hypothalamus which in turn are influenced by gonadal hormone and cerebral cortex.

RETICULAR FORMATION

BA

1) ~~ARAS~~

Reticular formn is scattered through central part of brainstem. There are reticular excitatory (activating) and inhibitory areas

The Reticular activating System (RAS) ≈ (ARAS)

- is a complex polysynaptic pathway
- the system is non-specific
- has ascending and descending fibres → reticulospinal tracts (inhibitory, facilitatory)

BA

1) ARAS (Ascending Reticular activating System) OR RAS

I) Components

- Fibres from reticular formn (bulboreticular facilitatory area) directly projecting to cerebral cortex → cholinergic
- Fibres from RF going through thalamus and projecting to entire cortex (non-specific thalamic projections) → mainly adrenergic
- RF also contain serotonergic fibres.

II) Functions (No specific sensation is carried)

- Maintains cortical neuronal excitability
- Maintains state of wakefullness, alertness and consciousness of an individual
  - ARAS can be stimulated by sensory stimuli and by muscular activity.
  - Fibres from limbic system can cause ↑ed activity of ARAS.

REVERBERATING CIRCUIT

→ this sets in due to afferent and efferent connections btw reticular formn and cortex.

Sensory input stimulates ARAS (visual, auditory etc)

↓

Reticular formn in Brainstem (midbrain & pons)

↓

Actvn of ARAS

↓

Thalamic relay (esp intralaminar nuclei)

↓

Cerebral cortex actvn

↓

Consciousness, wakefulness and alertness maintained

- Role in appreciation of sensations
  - Many of the ascending sensory pathways send collaterals to reticular formn. So, when a stimulus is applied, it not only evokes conscious perception of sensation, but also activates ARAS.
  - During sleep, all sensations are dull (ARAS inactive)
  - The most imp stimulus for activating ARAS is pain and this can arouse a drowsy/sleepy person

- Ability to concentrate/focus

The field of awareness is narrowed and this is done by → facilitating certain cortical areas and → simultaneously suppressing certain other areas

Eg: Soldiers do not feel pain in battle field unless fight is over.

REASON- due to suppression of pain fibres by corticofugal fibres and fibres from reticular formn that suppresses pain pathway at the 1st synapse (ie, substantia gelatinosa of Rolando) (Periaqueductal gray matter)

BA

2) Functions of reticulospinal tract

- Control of muscle tone
  - γ-motor neuron has connection from bulboreticular facilitatory area of reticular formations directly and from caudal inhibitory area indirectly through interneurons.
  - Normally, there is balance b/w facilitatory and inhibitory impulses and muscle tone is maintained.

Pontine form

- Relay centre for extrapyramidal fibre → Reticular formn

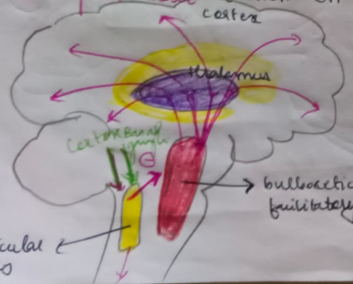
RT

- Decides background tone and posture
  - by maintaining tone of antigravity muscles
- Caudal inhibitory area inhibits extensors.

~~reticulospinal tract~~

The neurons of reticular formn are organized into 2 columns ↔ Medial / lateral

- The descending fibres from medial part form the reticulospinal tract (action on γ-motor neurons) & α " "



VESTIBULAR APPARATUS

BA

1) Functions of vestibular apparatus

ANS:-

1) Posture and eqlbm:
   - Medial VT → antigravity muscles of neck and UL
   - lateral VT → " " of trunk and LL

2) Macula in utricle and saccule detect linear acceleration
   - utricle → horizontal acc
   - saccule → vertical acc
   - These macules help to detect orientation of head with respect to gravity and maintain static eqlbm.

3) Crista ampullaris of semicircular canal can detect → Angular acceleration (Head rotation)

4) Reflexes and functions
   - Vestibular righting reflex → maintains erect position of head
   - tonic labyrinthine reflexes → Adjust the relative position of head with trunk and limbs
   - Vestibulo-ocular reflex → keeps visual fixation unchanged (function of semicircular canal)

2) Components

Vestibular apparatus is encased in a system of bony labyrinth. Within this system, there are membranous tubes and chambers called membranous labyrinth. (Full part)

ML

Consists of:
1) Three semicircular canal
2) The otolith organ - Saccule and utricle

VIII SEMICIRCULAR CANAL → detect angular acceleration

I) Structure

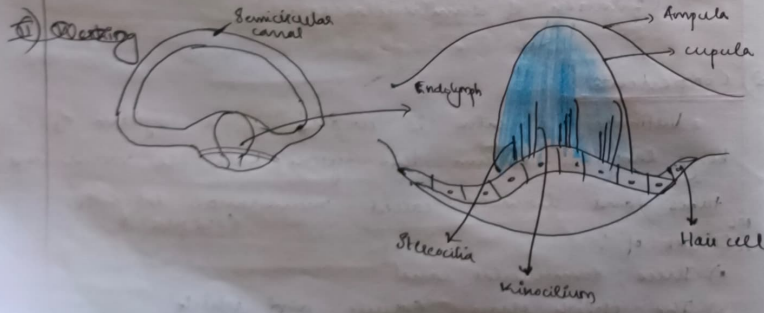
- SC are 3 tubular structures placed at right angles to each other.
- They represent 3 axis of rotations: vertical, anteroposterior and transverse axis and the canals are:

1) Horizontal (lateral)
2) Superior (anterior)
3) Inferior (posterior)

} contain endolymph and separated from bony canal by perilymph

- Each canal begins as a dilatation (ex ampulla), containing a projection ridge (on its inner aspect) called crista ampullaris (Cristae are receptors)
- Each crista contains hair cells and sustentacular cells surmounted by gelatinous partition (cupula) that closes off the ampula.
- The processes of hair cells are embedded in cupula and bases are in contact with afferent fibres of vestibular division of vestibulocochlear n.

II) Working



II) Working

When the head rotates

1) Head moves → Bony canal of SC moves
2) But endolymph lags behind (due to inertia)
3) Cupula bends due to the lagging fluids, thereby bending the hair cells within it.
4) If the hair cell bends:
   - towards kinocilium → depolarisation (↑ed firing)
   - away from " → hyperpolarisation (↓ed firing)
5) Signal sent to brain
   - vestibular n. carries the info

NOTE :- 1) • horizontal canal → detects 'no' motion
• Ant & post → detect nodding and tilting head to side

2) Paired system → Rt and Lt SC work together
→ When 1 excited, other is inhibited

VIII X/E

VIII Otolith organ (BA)

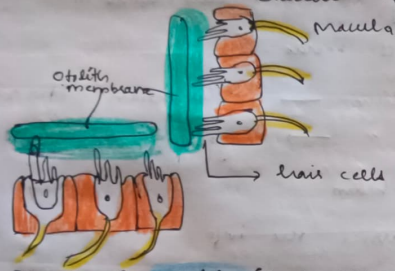
- Formed by utricle and saccule

I) STRUCTURE

- Both utricle and saccule contain a projection ridge called the macula (like crista)
  ↓
  Receptors of otolithic organ
- Macula is formed by hair and supporting cells which project into a firm gelatinous material, cupula
- This cupula contains many chalky ($CaCO_3$) particles

called otoliths (ie otoconia) hence the name otolith organ.

- The hairs + gelatinous material = otolith membrane
- When head in normal erect position
  - Macula of each utricle is in horizontal plane
  - " " Saccule is in vertical plane



II) Working → detect linear acc
- vert → saccule (Eg:- elevator)
- hori → utricle (Eg:- car motion)

- Otoliths shift due to gravity / inertia (during motion).
- This cause otolith membrane to move and hence bending the hair cells
- Towards kinocilium → depolarisation (opp hyper)
- Causes changes in firing of vesti n.

2) VESTIBULO-OCULAR REFLEX (VOR)

- keeps your eye fixed on an object when head is moving.

Stimulus: Head movement (angular/linear)

Sensor:- Semicircular canals, otolith organs } Vestibular apparatus

Pathway: Vestibular n. → Vestibular nuclei → Oculomotor nuclei (act on extraocular muscles)

Effect: Eye movement in opp direction

Fn:- Keeps gaze stable during head movement

DISORDERS

1) Nystagmus - involuntary, rhythmic oscillation of eye
- In vestibular disease, one side of vestibular apparatus may get weak (either Rt or Lt)
- ∴ Uneven signals reach brain
(Eg:- one ear says "I'm moving"
other ear says "I'm still")
- This confuses the brain and makes it think you are moving and activates vestibulo-ocular reflex.
- So, eyes starts to
  - slowly drift in one direction (as brain thinks you are moving)
  - Then quickly back to reset (fast phase)

This, keeps repeating → causing oscillatory motion

2) Motion sickness

- While in a car, bus, flight etc, our ears (vestibular apparatus) detects motion
- But, the visual input and proprioceptive input if that we are seated and are not using our muscles or joints to move.
- This causes a mismatch and confuses the brain hence causing: nausea, vomiting, cold sweats

(For Eg: Reading book in car
- Ears feel the bumps and motion → "we are moving"
- But eyes are focused on book → "we are still")

TREATMENT / Prevention

1) Habituation / Repeated exposure

2) Suppressing the vestibular inputs
   - use of pharmacological agents like
   - Scopolamine, promethazine etc

3) Stabilising visual field
   - looking at stable object or horizon helps synchronize vestibular and visual inputs.

3) Manicre's disease
- Vestibular dysfunction with progressive hearing loss

POSTURE AND EQUILIBRIUM

1) Differentiate decerebrate and decerticate preparations

Ans:	DECORTICATE	DECEREBRATE
1) Level of lesion:	- Above red nucleus (ie cerebral cortex and internal capsule)	B/w superior and inf colliculi (ie below red nucleus)
2) Mechanism:	• Red nucleus and rubrospinal tract intact → Flexor tone in UL • Vestibulospinal and reticulospinal tract active → Extensor tone in LL	• Red nucleus damaged / disconnected so rubrospinal tract inactive → no flexor tone in arms • Vestibulospinal and reticulospinal are overactive (as inhibitory influence of cortex & basal ganglia removed) → Extensor hypertonia
3) Posture	• Flexion of arms • Extension of legs	Rigid extension of all four limbs (arms and legs)
4) Gamma motor activity	↑ (due to loss of cortical inhibition)	↑↑↑ (more severe)
5) Postural and righting reflexes	P → ✓ R → ✓ But certain postural reflexes are disrupted → hopping, placing rxn.	P (at spinal cord centre) → stretch, positive supporting rxn, crossed extensor, tonic neck and labyrinthine R → absent

Righting reflexes

	Stimulus	Response	Receptor	Integrated in
1) Labyrinthine Rt reflex	Gravity	Head kept level / upright	Otolith organ	Midbrain
2) Body on head Rt reflex	Pressure on side of body	Righting of head	Exteroceptors	"
3) Neck Rt reflex	Stretch of neck muscles	Righting of trunk and shoulders, then pelvis	Muscle spindle	"
4) Optical Rt reflex	Visual cues	Rt of head	Eyes	Cerebral cortex (Callosing cortex)
5) Body on body Rt reflex	Pressure on side of body	Rting of body over when head held sideways	Exteroceptors	Midbrain

POSTURAL REFLEXES

1) Stretch (V. imp) - main	Stretch	Contraction of muscle	Muscle spindle	Spinal cord, medulla
2) + supporting reflex	Contact with sole / palm	Foot extended to support body	Proprioceptors	SC
3) -ve supporting reflex	Stretch	Relaxation of +ve supporting reflex	Proprioceptors	SC
4) Tonic labyrinthine reflexes	Gravity	Contraction of limb extensor	Otolithic organs	medulla
5) Tonic neck reflexes	Head turned → side, up or down	Change in extensor pattern - Extension of limbs on side to which head is turned	Neck proprioceptors	Medulla
6) Placing reflex	Various visual, exteroceptive, proprioceptive	Footing placed on supporting surface to support body	Various	Cerebral cortex
7) Hopping reflex	Lateral displ while standing	Hops to maintain limb in position to support body	Muscle spindle	Cerebral cortex

RELEASE PHENOMENON (BA)

The release phenomenon refers to the appearance of exaggerated reflexes or abnormal movement when higher brain centres (like cerebral cortex) that normally inhibit lower centres (like brain stem) are damaged.

Release = loss of inhibition

↓

Overactivity of primitive/reflex actions

EXAMPLES :-

1) Decerebrate rigidity
Loss of cortical control → brainstem extensor patterns exaggerative

2) Babinski sign
Cortical inhibition gone → primitive reflex reappears

3) Mass reflex (in spinal shock recovery)
Spinal reflexes become exaggerated due to loss of brain control

4) Return of primitive reflexes
Infantile reflexes (like grasp, -ve supporting reflex) reappears in adults

RA

EEG - Electroencephalogram (Imp)

It is the recording of spontaneous neuronal electrical activities of brains → brain waves. It is due to summated post-synaptic potentials

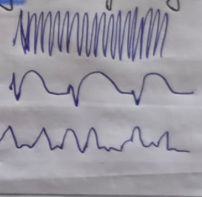
WAVES	Freq	Amp (μV)	Area of brain from where recorded	Condn of subject
1) α-wave	8-12	30-50	Occipital	Awake, closed eyes
2) β-wave	15-30	5-10	Parietal	Awake, active state of brain (open eyes)
3) δ-wave	1-5	20-200	Frontal	Deep sleep, newborn, deep anesthesia
4) θ-wave	4-7	5-100	Temporal	Awake, normal in children and early sleep

Variation of EEG

1) Freq of α-rhythm at rest varies according to age
2) Fast, β-like rhythm seen in infants
3) Adult pattern gradually appears during adolescence
4) Freq of α-rhythm decreased by
   - low body temp
   - low blood glucose
   - low glucocorticoid
   - high arterial pCO_2

Applications of EEG

1) Subdural hematoma leads to trd EEG activity over that area
2) Localisation of brain tumors
   - tumor may compress neurons in that area
   - ↑ing their excitability → epileptic attack may be seen
3) Brain death → flat EEG for more than 24 hrs
4) Diagnosis of epilepsy (distinguishing diff types)
   - Grand mal epi
   - Petit mal epi
   - Psychomotor epi

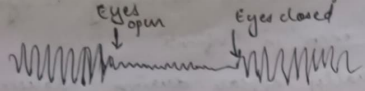


high freq, high amp
spike and dome pattern
low amp, abnormal pattern

α-block

- α-rhythm is recorded when a person is at rest, awake with eyes closed.
- This can be blocked by opening of eyes, sensory stimuli or mental concentration such as solving a arithmetic problems.

- Waves became fast irregular waves (β type)
- Also called arousal resp / alerting resp / desynchronization



Changes in EEG during different stages of sleep

Stage	Behavioural observation	EEG changes
NREM (Slow wave sleep)		
Stage 1	Stage of drowsiness and falling asleep i) Easily aroused by moderate stimuli ii) Continuous lack of awareness	α-waves reduced in frequency and amplitude
Stage 2	i) True sleep ii) Further lack of sensitivity to activation and arousal	• Sleep spindles appear (due to reverberating activity b/w thalamus and the cerebral cortex) • There are bursts of regular α-like waves • Appearance of α-rhythm in the β rhythms is sleep spindles.
Stage 3	Sleep deepens	Sleep spindles are occasional and are superimposed by background of δ waves
Stage 4	• Stage of deep sleep • difficult to arouse the person • Arousal only when vigorously stimulated and when awakened person does not report of dreaming	Slow high voltage δ waves

REM sleep (paradoxical sleep)

- Deepest sleep
- → called paradoxical because sleeping person is difficult to arouse despite having a desynchronized EEG that is characteristic of alert, awake state
- Skeletal muscle tone is markedly reduced except in eye, where REM occurs
- REM sleep occurs more towards morning hours and hence dreams are frequent towards waking hours
- EEG desynchronised i.e low voltage fast activity (resembling awake state with eyes open)
- Large spikes are seen in EEG called PGO spikes (ponto-geniculo-occipital) which are characteristic of REM sleep

NREM	REM (Paradoxical sleep)
1) Hypotonia	1) More hypotonia
2) Slow-high voltage δ waves	2) Fast low voltage 'β' waves
3) No rapid movement of eye ball	3) Rapid movement of eyeball
4) Dreams cannot be recalled or vague recalling	4) Dreams can be recalled easily
5) No PGO spikes	5) PGO spikes present.
6) Pulse, BP, resp are lowest and regular	6) Pulse, BP, resp increased and are irregular
7) Serotonin content of Raphe nucleus is associated with SWS (slow wave sleep)	7) Noradrenaline content of locus coeruleus is associated with paradoxical sleep
8) Threshold for arousal by the sensory stimulus is elevated	8) Threshold is further elevated

9) 75% of total sleep	9) 25% of total sleep
10) Brain activity is less	10) Brain activity is more
11) O_2 consumption is less	11) O_2 consumption more

Physiological changes during sleep

1) CVS : HR, CO, vasomotor tone, BP decrease
2) Resp : TV, RR, PV decreases
3) BMR : decreases
4) Nervous system :
   - EEG shows δ waves
   - deep reflexes reduced
   - sup reflexes unchanged
   - light reflex is retained
5) Eye : Eyeballs roll up and out due to flaccidity of external ocular muscles
   Eyelids comes closer due to drooping of upper eyelid
   Pupils constrict
6) Blood volume : increases resulting in dilution of plasma
7) Muscles : completely relaxed and tone is minimum
8) Secretions :
   - Salivary and lacrimal secretion decreases
   - Sweat secretion ↑es
   - Gastric sec either remains unaltered or ↑es
9) Urine volume ~~volume~~ decreases
   ~~concentration~~ Phosphate content and specific gravity ↑es
   ∴ More concentrated urine is formed.