

Ascending Pathway

1) Dorsal column tract and brown sequard's syndrome and Effects of Tabes dorsalis?

Fasciculus Gracilis and Fasciculus cuneatus:

- ⇒ Fasciculus gracilis and Fasciculus cuneatus are together called ascending posterior column tracts.
- ⇒ These tracts are formed by the fibres from posterior root ganglion.
- ⇒ Thus, both the tracts are constituted by fibres of 1st order neurons of sensory pathway.

Situation :-

- ⇒ Tracts of Goll and Burdach are situated in posterior white column of spinal cord, hence the name posterior column tracts.
- ⇒ In the cervical and upper thoracic parts of spinal cord, the posterior white column is divided by post intermediate septum into medial fasciculus gracilis and lateral fasciculus cuneatus.
- ⇒ The fasciculus gracilis is situated medially in b/w posterior median sulcus & post median septum on one side and post intermediate septum and post intermediate septum on other side.
- ⇒ The fasciculus cuneatus is situated laterally. It is bound by
 - Medially - post intermediate septum & sulcus.
 - Laterally - post gray horn, post nerve root, tract of Lissauer.

Origin :-

- ⇒ Fibres of these two tracts are axons of 1st order neurons. Cell body of these neurons is in the post root ganglia and their fibres from medial division of post nerve root.

Course :-

- 1) After entering the spinal cord, the fibres ascend through post white column.
- 2) These fibres do not synapse in the spinal cord. Some fibres of medial division of post nerve root descend through post white



column in the form of Fasciculus interfascicularis.

3) Fasciculus gracilis contains fibres from lower extremities and lower parts of the body i.e from sacral, lumbar and lower thoracic ganglia of post nerve root.

4) Fasciculus cuneatus contains fibres from upper extremities i.e from upper thoracic and cervical ganglia of post nerve root.

Termination :-

1) Tracts of Goll and Burdach terminate in the medulla oblongata.

2) Fasciculus gracilis and Fasciculus cuneatus fibres terminate on the nucleus gracilis and nucleus cuneatus respectively.

3) Neurons of these medullary nuclei form 2nd order neurons.

4) Axons of 2nd order neurons forms internal arcuate fibres.

Internal arcuate fibres from both sides cross the midline forming sensory decussation.

5) Then ascend through pons and midbrain as medial lemniscus.

6) Fibres of medial lemniscus terminate in ventral posterolateral nucleus of thalamus.

7) From thalamus fibres of 3rd order neurons relay to sensory area of cerebral cortex.

Functions :-

Tracts of posterior white column convey impulses of following sensations -

i. Fine tactile sensation

ii. Tactile localization (Ability to locate the area of skin where tactile stimulus is applied with closed eyes)

iii. Tactile or 2 point discrimination (Ability to recognize the 2 stimuli applied over skin with closed eyes)

iv. Vibratory sensation

v. Conscious kinesthetic sensation - sensation of various muscular activities in our body.

vi. Stereognosis - (Ability to recognize the object by touch with closed eyes. It is a sense produced by combination of both tactile & kinesthetic sensations)



Effect of lesion :-

Lesion of ~~tracts~~ of nerve fibres in tracts of Golt and Burdach or lesion in the posterior column tracts causes following symptoms.

- i. Loss of fine tactile sensation
- ii. Loss of tactile localization
- iii. Loss of tactile discrimination
- iv. Loss of vibratory sensation.
- v. Astereognosis
- vi. Loss of ability to recognize the wt of different objects.
- vii. Loss of proprioception.
- viii. Sensory ataxia or posterior column ataxia.

Hemisection of Spinal cord / Brown sequard's syndrome :-

* Lesion involving one lateral half of the spinal cord is called hemisection or Brown sequard's syndrome. It can occur due to injury during accidents.

Signs and symptoms :-

• Signs and symptoms which occur after hemisection of spinal cord constitute Brown sequard's syndrome.

⇒ If hemisection is due to injury, spinal shock occurs immediately. Muscles lose the tone & becomes flaccid. The reflexes are abolished.

⇒ In case the patient survives, this stage gradually passes off and certain signs and symptoms develop.

⇒ Symptoms are seen at the level of lesion and below the level of lesion.

⇒ Effects in these areas differ on same side ^{and} on opposite side. The changes in sensory and motor functions.

Tabes Dorsalis :-

⇒ Tabes Dorsalis is another disease of spinal cord. It is slowly progressive nervous disorder affecting both sensory & motor



functions of spinal cord.

Cause:-

- ⇒ It occurs due to degeneration of posterior nerve roots. It usually occurs in syphilis.
- ⇒ Posterior nerve roots are affected proximal to posterior root ganglion. Ganglion are not affected.
- ⇒ Among the fibres of posterior root, the fibres of lateral dorsal root are more affected.
- ⇒ Along with lateral fibres of posterior cord, fibres of posterior white column of spinal cord are also affected.

Sensations:-

- 1) During onset of degenerative changes, there is exaggeration of pain sensation.
- 2) Impairment and loss of all sensations.
- 3) Loss of sensations, particularly pain sensation leads to joint deformities. There is no proper support & movements at this joint become uncontrolled. It is called Charcot joint.
- 4) Joints enlarge due to inflammation by the development of osteoarthritis.
- 5) Both superficial and deep reflex are lost.
- 6) Ataxia - lack of coordination of movements.
- 7) Stamping gait
- 8) Atonic bladder - Micturition reflex are lost & urinary bladder becomes atonic bladder.

Dissociated anesthesia:-

- ii Loss of some sensations while other sensations are intact
- iv ⇒ Dissociated anesthesia is a form of anesthesia characterized by catalepsy, catatonia, analgesia and amnesia.
- v ⇒ It does not necessarily involve loss of consciousness and does not always imply a state of general anesthesia.



Level

Same side

Sensory changes

Sensations lost & (crossed tracts)

- 1) Fine touch
- 2) Tactile localization
- 3) Tactile discrimination
- 4) Vibratory sensation
- 5) Stereognosis
- 6) Conscious Kinesthetic sensation

Sensations retained :-

1. Crude touch
2. Pain
3. Temperature

Complete anesthesia

Motor changes

Upper motor neuron lesion type
Increased tone

Paralysis (Spastic)

Rigidity in limbs

Babinski positive sign

No muscle wastage

Lower motor neuron lesion type

1. Loss of muscle tone
2. Loss of vasomotor tone
3. Flaccid paralysis
4. Muscle wastage

Opposite side

Sensory changes

Sensations lost :-

1. Crude touch
2. Pain
3. Temperature

Sensations retained :-

1. Fine touch
2. Tactile localization
3. Tactile discrimination
4. Vibratory sensation
5. Stereognosis
6. Conscious Kinesthetic sensation

Same of the above

Motor changes

No paralysis

If it occurs

i. Very mild

ii. resembles upper motor neuron lesion type

No paralysis

If it occurs

i. Very mild

ii. resembles lower motor neuron lesion

Effects of Hemisection

At the level of lesion



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Physiology of Pain

Pain :- Protective phenomenon of the nature.

Defination :- Pain defined as a unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of damage.

Nociceptive stimuli :-

⇒ Stimulus that elicits pain is called nociceptive stimulus.

⇒ Nociceptive means potentially damaging.

For Skin :- Pricking, cutting, crushing, burning, freezing etc.

For GIT :- Inflammation of mucosa, spasm of smooth muscle.

In skeletal muscle :- Ischemia, necrosis, hemorrhage

In cardiac muscle :- Ischemia of cardiac muscles.

In brain :- cerebral arteries or meningeal structures.

For blood vessels :- Artery or vein pierced by needle.

In nerves :- Compression of nerve root or sensory ganglia.

Types of Pain :-

Fast pain / First pain → It is due to Aδ fibres.

Slow pain / Second pain → It is due to C fibres.

Superficial pain → Pain in superficial structures like skin and subcutaneous tissues. There will be fast pain in superficial structures.

Deep pain → Pain in deeper structures like bone, muscles, connective tissue etc. There will be slow pain in deeper structures.

Somatic pain → Originating in somatic structures

^{V/s}
Visceral pain → originating in visceral structures.

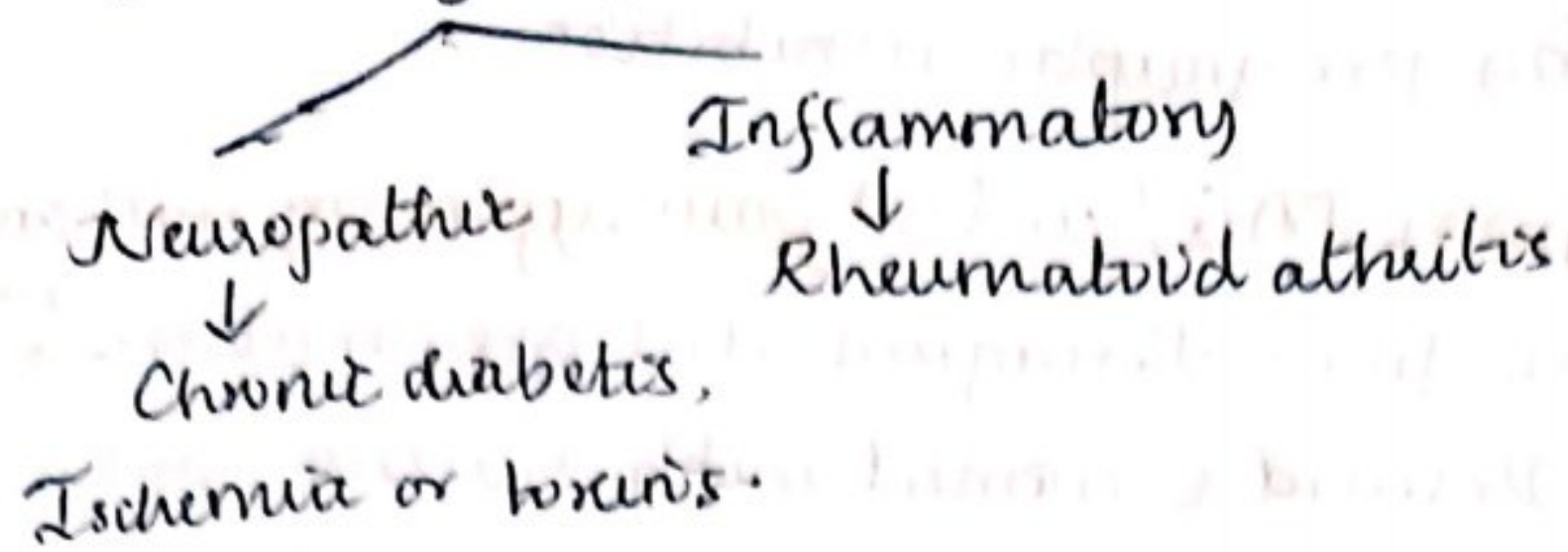
Peripheral pain → occurring due to direct stimulation of receptors

^{V/s}
Eg: neurogenic or neurogenic
Central pain → central pain fibres → pain below the level of lesion in spinal transection

Physiologic pain → Acute and good pain



Pathologic pain → chronic and bad pain.



Pain pathway :-

Pain is carried by two pathways

- i) Neospinothalamic pathway
- ii) Paleospinothalamic pathway.

Neospinothalamic tract :- Carries fast pain.

1st order neuron :- Aδ fibres from receptors to spinal cord.

2nd order neuron :- From dorsal horn of spinal cord → cross to opposite side → ascent in lateral white column → end on ventro-postero lateral & ventro postero medial (VPM) nuclei of thalamus.

3rd order neuron :- From thalamus to somatosensory cortex.

Paleospinothalamic tract :- carries slow pain

1st order neuron :- C fibres from receptors to spinal cord.

2nd order neuron :- From dorsal horn of spinal cord → cross to opposite side → ascend in lateral white column → end in intralaminar & midline nuclei of thalamus.

3rd order neuron :- From thalamus to entire cerebral cortex.

⇒ Neospinothalamic tract is concerned with localization and interpretation of quality of pain.

⇒ Paleospinothalamic tract concerned with perception of pain, arousal and alertness.

2. Modulation of Pain / Endogenous Pain Relief system :-

Gate control theory of Pain :-

⇒ Large myelinated A fibres interact with small unmyelinated C fibres via inhibitory cells of the substantia gelatinosa of the spinal cord

→ stimulation of C fibres inhibits SG cells & favours passage of pain



⇒ Stimulation of large 'A' fibres

block pain transmission by presynaptic inhibition.

Endogenous pain relief from PAG / central pain suppressing mechanism

⇒ Descending pathways arise from Periaqueductal grey matter.

Release → Enkephalin → Descend & connect with nucleus raphe

magnus of medulla → release of serotonin → posterior horn

⇒ of spinal cord → inhibits the release of substance 'P' from the pain

⇒ fibres.

Opioid peptides :-

Enkephalins & Endorphins

Two sites of action

- Block the pain receptors

- At spinal level - binds to opioid receptors & decreases pain transmission.

Referred Pain :-

Visceral pain felt at the somatic structures is called referred pain

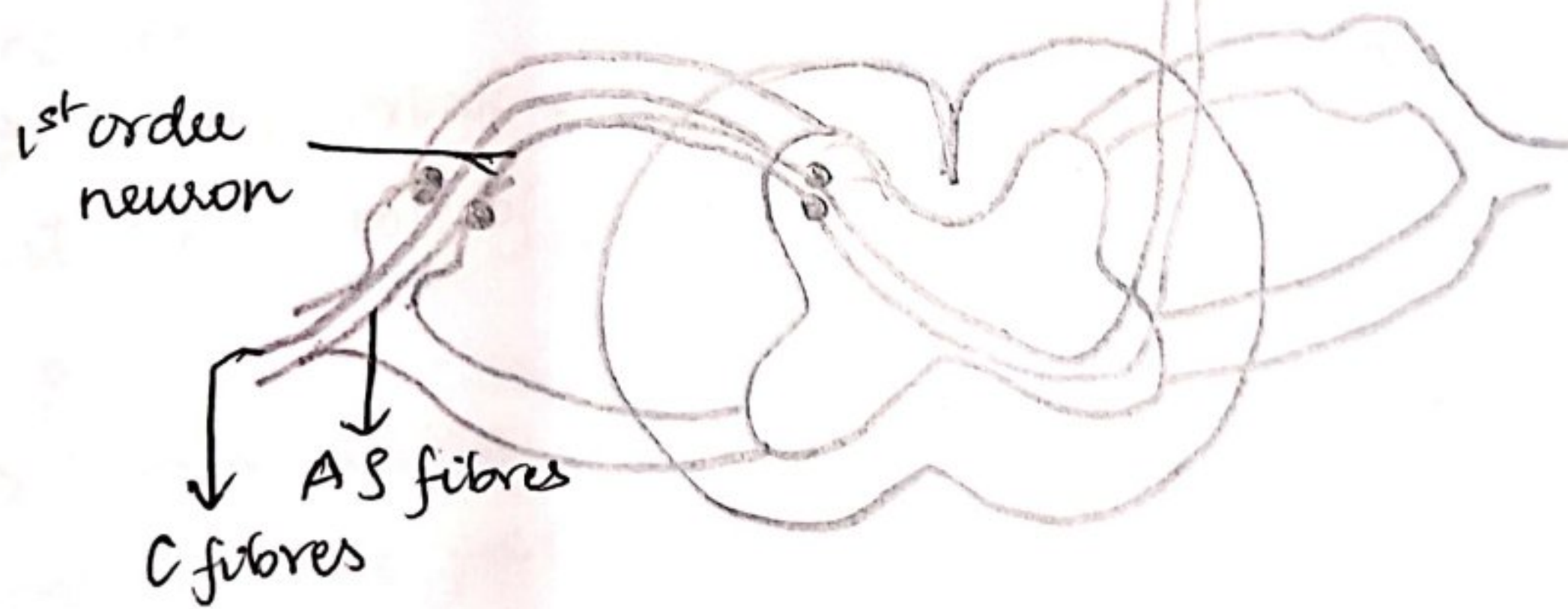
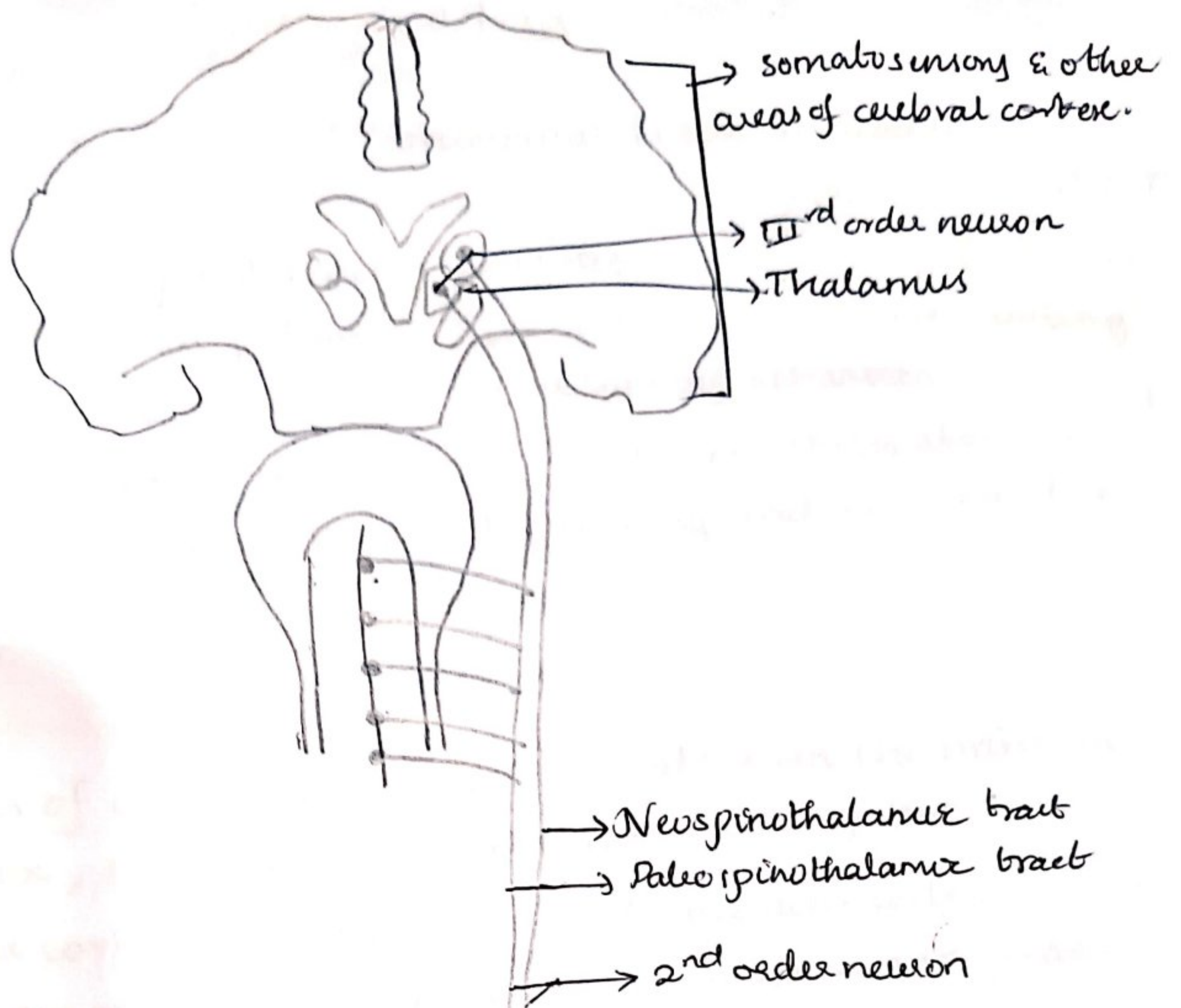
Eg:- Appendicitis pain at the umbilicus

Cardiac pain at the inner aspect of left arm.

Theories of referred pain :-

1. Convergence theory - Fibres carrying pain from the viscera & corresponding dermatome (somatic structures) converge on the same pathway to the cortex.

2. Facilitation theory - The visceral pain facilitates substantia Gelatinosa Rolando (SGR) cells which receive somatic pain nerves



Corticospinal tract

Introduction:-

- ⇒ There are two groups of corticospinal tracts 1) Lateral CST
2) Ant or ventral CST
- ⇒ Lateral corticospinal tract contributes about 80% of the fibres in the corticospinal pathway. This is the most important descending pathway for the control of skilled voluntary activities.
- ⇒ Anterior or ventral corticospinal tract contributes about 20% of the fibres in the corticospinal pathway and is involved in control of posture.

Origin:-

- ⇒ The fibres of corticospinal tract originate from the primary motor cortex, especially from large motor cells of Betz.
- ⇒ The other cortical motor areas include premotor cortex, supplementary motor area, primary somatosensory cortex and parietal cortex.
- ⇒ Motor cortex contributes to 60% of fibres and sensory cortex contributes to 40% of fibres in corticospinal tract.

Course:-

- ⇒ Fibres arising from the different parts of the cortical areas converge through the posterior limb of internal capsule.
- 1. As the fibres in the subcortical areas appear in a radiating pattern, they are collectively known as corona radiata.
- 2. After passing through the internal capsule, fibres descend down in the ventral brainstem as cerebral peduncles.
 - In the medulla, fibres pass through the pyramid.
- 3. About 80% of the fibres after passing through the pyramid immediately decussate and descend down in the lateral funiculus of the spinal cord.
 - This forms the lateral corticospinal tract.
 - The lateral corticospinal fibres have monosynaptic connections with anterior horn cells.
- 4. The remaining 20% of the fibres descend down in the

- ipsilaterally on the anterior. functioning of the corticospinal tract.
- This constitutes the ventral or anterior cortico-spinal tract.
 - The fibres cross over to the opposite side only at the spinal cord segments through termination on the interneurons.

Termination:

1. Fibres of LCST terminate on lateral group of motor neurons.
 - About 30% of lateral CST fibres terminate directly on motor neurons and 70% fibres terminate through ~~two~~ interneurons.
 - These motor neurons innervate the distal limb muscles.
2. The fibres of VCST do not directly terminate on motor neurons they end in interneurons in the same side of spinal cord.
 - These interneurons turn opposite side and terminate on medial group of motor neurons.
 - These motor neurons supply proximal limb muscles & axial muscles of the body.

Functions:

- Motor cortex is mainly involved in initiation, planning and control of movement.
- CST transmit central command signal from the motor cortex to the spinal cord interneurons and motor neurons.
- Lateral CST controls skilled voluntary movements of the body.
- Ventral CST controls posture.

Effects of lesions:-

- 1) Lesion of LCST :- It results in impairment of skilled voluntary activities like writing, painting.
 - Isolated lesion of LCST is very uncommon in humans.
 - In addition, disease which affect the CST also affect the corticobulbar tracts that influence the activities of extrapyramidal tracts.
 - There is no pure CST disease in humans.
- 2) Lesion of VCST :- It results in inability to maintain posture while walking, climbing... etc.

- This tract is not well developed in humans.
- Other major posture regulating pathways, especially the reticulospinal tract & vestibulospinal tract, are still intact.

Hemiplegia:-

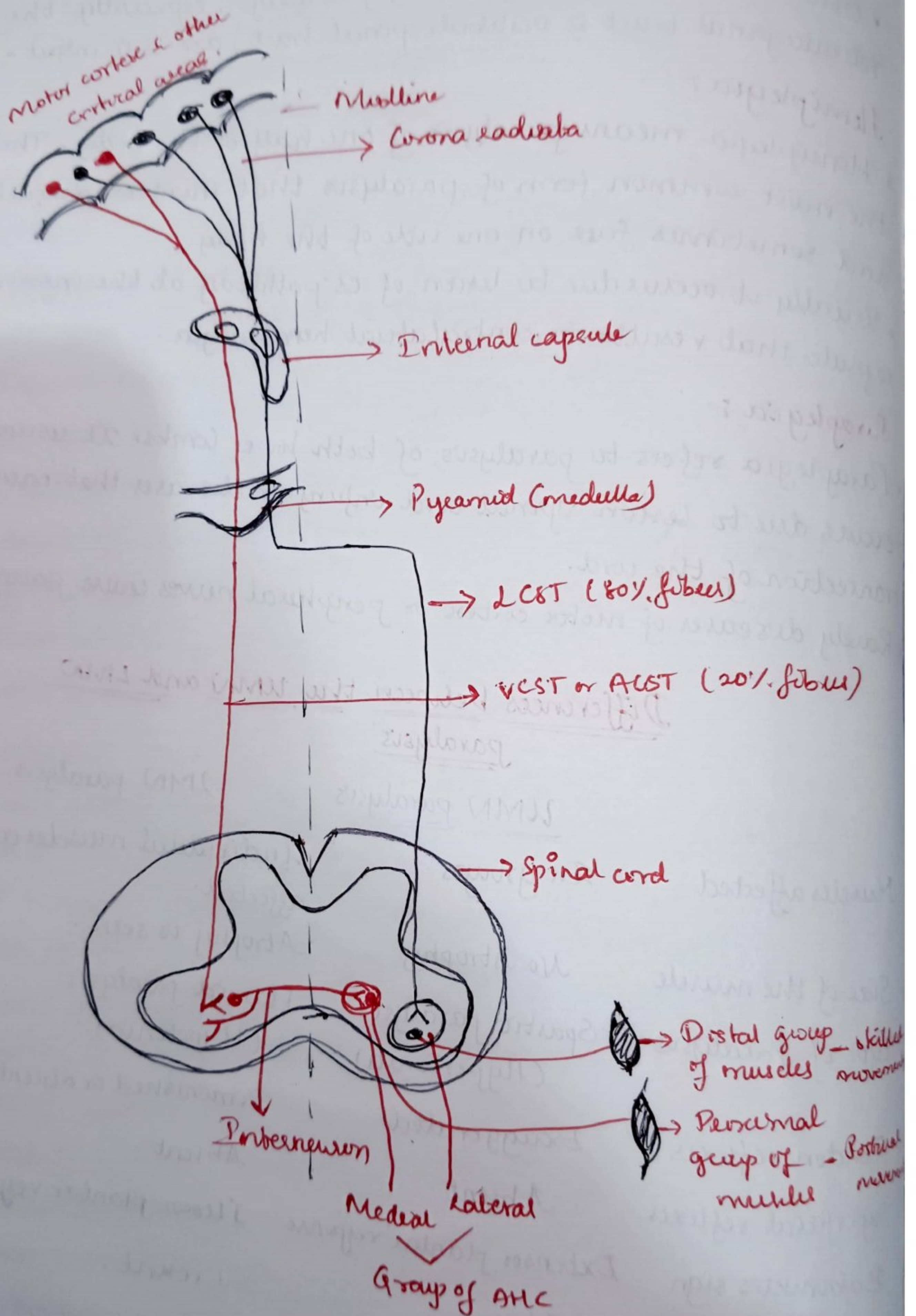
- Hemiplegia means paralysis of one half of the body. This is the most common form of paralysis that involves arm, leg and sometimes face on one side of the body.
- Usually it occurs due to lesion of CS pathway at the internal capsule that results in contralateral hemiplegia.

Paraplegia:-

- Paraplegia refers to paralysis of both lower limbs. It usually occurs due to lesion spinal cord injury or diseases that cause transection of the cord.
- Rarely diseases of motor cortex or peripheral nerves cause paraplegia.

Differences between the UMN and LMN paralysis

	<u>UMN paralysis</u>	<u>LMN paralysis</u>
1. Muscles affected	In groups	Individual muscles are affected.
2. Size of the muscle	No Atrophy	Atrophy is seen.
3. Type of Paralysis	Spastic paralysis (Hypertonia)	Flaccid paralysis (Hypotonia)
4. Tendon reflexes	Exaggerated	Diminished or absent.
5. Superficial reflexes	Absent	Absent.
6. Babinski's sign	Extensor plantar response	Flexor plantar response.
7. Involuntary movements	Absent	Present.
8. EMG changes	No denervation potential	Denervation potential seen
9. Nerve conduction study	No abnormalities	Decreased nerve conduction.



Cerebellum

Functional divisions :-

Based on functions, the cerebellum is divided into 3 divisions.

1. Vestibulocerebellum
2. Spinocerebellum
3. Cerebocerebellum.

1. Vestibulocerebellum :-

- ⇒ Vestibulocerebellum is connected with the vestibular apparatus and so, it is known as vestibulocerebellum.
- ⇒ Since it is phylogenetically oldest part of cerebellum, hence it is called as archicerebellum.
- ⇒ It is concerned with maintenance of posture and equilibrium.

Components :-

- ⇒ Vestibulocerebellum includes the flocculonodular lobe that is formed by the nodulus of vermis and its lat extensions called flocculi.
- ⇒ Utricle of vermis is also considered as the part of vestibulocerebellum.

Connections :-

Afferent connections :-

Vestibulocerebellar tract :-

- It is formed by the fibres of arising from the vestibular nuclei, situated in pons and medulla.
- It passes through the inferior cerebellar peduncle of the same side and reaches cerebellar nuclei, nucleus globosus, nucleus emboliformis and nucleus fastigi.
- Fibres from these nuclei reach the vestibulocerebellum.
- Vestibular nuclei in turn receives fibres from vestibular apparatus situated in the inner ear, through vestibular division of cochlear nerve.

Efferent connections :-

1. Cerebellovestibular tract :-

Fibres of cerebellovestibular tract arise from the flocculonodular

- lobe, pass through inf cerebellar peduncle on same side and terminate on the vestibular nuclei in brainstem.
- Fibres from vestibular nuclei form lat and med-vestibulospinal tracts, which terminate on α -motor neurons.

2. Fastigiobulbar tract:-

- Fibres of fastigiobulbar tract arise from fastigial nucleus pass through inf cerebellar peduncle of the same side & terminate on vestibular nuclei & reticular formation in medulla oblongata.
- From vestibular nuclei, vestibulospinal tracts arise and terminate on α -motor neurons.
- From reticular formⁿ, reticulospinal tracts arise & terminate on gamma motor neurons.

Functions:-

- Vestibulocerebellum regulates tone, posture and equilibrium by receiving impulses from vestibular apparatus.
- Vestibular apparatus sends information regarding gravity, linear movement, and angular acceleration to vestibulocerebellum through vestibulocerebellar tract.
- Vestibulocerebellum sends signals to spinal cord through vestibulospinal tract and reticulospinal tract.

Mechanism of action of Vestibulocerebellum:-

- Normally vestibular nuclei facilitates the movements of neck, trunk and limbs through vestibulospinal tract & α -motor neurons.
- Medullary reticular formation inhibits muscle tone through the reticulospinal tract & gamma motor neurons.
- Vestibulocerebellum inhibits both vestibular nuclei & medullary reticular formation.
- As a result, movements of neck, trunk and limbs are checked & the muscle tone increases.
- Because of these effects, any disturbance in posture & equilibrium is corrected.

In the lesion of vestibulocerebellum, there is a reduction in muscle tone (hypotonia) and failure to maintain posture & equilibrium.

③ Spinocerebellum:-

- Spinocerebellum is connected with spinal cord & hence the name.
- It is the major area of cerebellum for receiving sensory input.
- It is concerned with maintenance of muscle tone & anticipatory adjustment of muscle contraction during movement.
- Spinocerebellum is also phylogenetically older part of cerebellum. It is also called as paleocerebellum.

Components:-

- Spinocerebellum consists of medial portions of cerebellar hemisphere, vermis, paraflocculi and the parts of vermis except lingula, uvula, central lobe, culmen, lobulus simplex, declive, lobus & pyramid.

Functions:-

- Spinocerebellum regulates tone, posture and ^{equilibrium} ~~cerebellum~~ by receiving sensory impulses from tactile receptors, proprioceptors, visual receptors and auditory receptors.
- Spinocerebellum is the receiving area for tactile, proprioceptive, auditory & visual impulses. It also receives cortical impulses from pontine nuclei.
- Tactile & proprioceptive impulses are localized on spinocerebellum.
- Spinocerebellum regulates the postural reflexes by modifying muscle tone.
- Increased discharge from gamma motor neurons increases the muscle tone.
- Lesion, destruction or abolishing the function of spinocerebellum by coding causes stoppage of discharge of gamma motor neurons resulting in hypotonia and disturbances in posture.

Corticocerebellum:-

Corticocerebellum is the largest part of cerebellum. Because of its connection with cerebral cortex, it is called corticocerebellum or cerebrocerebellum.

- It is phylogenetically newer part of cerebellum. So, it is called neocerebellum.
- It is concerned with planning, programming and coordination of skilled movements.

Functions :-

- Cerebrocerebellum is concerned with integration and regulation of well-coordinated muscular activities.
- It is because of its afferent-efferent connection with cerebral cortex through the cerebro-cerebellar-cerebral circuit.
- Apart from this, it also receives feedback signals from the muscles through the nerve fibres of proprioceptors.

Mechanism of action of Cerebrocerebellum :-

1. Damping action.
2. Control of ballistic movements.
3. Timing and programming the events.
4. Servomechanism
5. Comparator function.

① Chorea :-

Chorea is the abnormal involuntary movement. Chorea means rapid jerky movements. It mostly involves the limbs. Chorea is due to the lesion in caudate nucleus and putamen.

② Alpha gamma Colinkage :-

Muscle tone can also be due to tonic discharge of gamma motor neurons. The activation to these neurons are mostly from the descending fibres of the facilitatory reticular formation.

- This leads to stretching of muscle spindle, activation of alpha motor neurons and finally partially contracted muscle.
- The cerebellum is the alpha-gamma motor neuron linkage.
- Therefore, with the cerebellum, the muscle tension is maintained via alpha motor neurons as well as gamma motor neurons.

Muscle Spindle & Golgi tendon Organ

① Structure of Muscle Spindle :-

- Muscle spindle has a central bulged portion and two tapering ends.
- Each muscle spindle is formed by 5-12 intrafusal muscle fibres.
- All these fibres are enclosed by a capsule, which is formed by connective tissue.
- Intrafusal fibres are attached to the capsule on either end. The capsule is attached to either side of extrafusal fibres or tendon of the muscle.
- The intrafusal fibres are placed parallel to extrafusal fibres. Intrafusal fibres are thin and striated.
- Central portion of intrafusal fibres does not contract because it has only few or no actin and myosin filaments. So, this portion acts only as a receptor.
- Only end portion of intrafusal fibres contract. The discharge from gamma motor neurons causes the contraction of intrafusal fibres.

Types of Intrafusal fibres :-

- Muscle spindle is formed by 2 types of intrafusal fibres :

1. Nuclear bag fibres
2. Nuclear chain fibres.

Nuclear bag fibres :- Central portion of this fibre is enlarged like a bag and contains many nuclei. Hence it is called the Nuclear bag fibre.

Nuclear chain fibres :-

In nuclear chain fibre, central portion is not bulged and nuclei are arranged in the centre in the form of a chain. Nuclear chain fibre is attached to the side of end portion of nuclear bag fibre.

Nerve Supply to Muscle spindle :-

- Muscle spindle is innervated by both sensory and motor nerves.

- It is only receptor on the body, which has motor nerve supply.
- Sensory nerve supply :-

Each muscle spindle receives two types of sensory nerve fibres.

1. Primary sensory nerve fibre
2. Secondary " " "

① Primary sensory nerve fibre :-

- It belongs to type Ia nerve fibre. Each sensory nerve fibre has 2 branches. One of the branches supplies the central portion of nuclear bag fibre. The other branch ends in central portion of nuclear chain fibre.
- These branches end in the form of rings around central portion of nuclear bag fibres and nuclear chain fibres.
- Therefore, these nerve endings are called annulospiral endings.

② Secondary sensory nerve fibres :-

- It is a type II (Aβ) nerve fibre. It innervates only the nuclear chain fibre and ends near the end portion of nuclear chain fibre. So this nerve ending is called flower spray ending.

(Motor nerve supply +

- Motor nerve fibres supplying the muscle spindle belongs to gamma motor neuron (Aγ) type.

③ Motor nerve supply to nuclear bag fibre :- Gamma motor nerve fibres supplying the nuclear bag fibres end as motor end plate. This nerve ending is called plate ending.

- Functionally, it is known as dynamic gamma efferent nerve fibre.

Motor nerve supply to nuclear chain fibre :- Gamma motor nerve fibre supplying nuclear chain fibres divides into many branches which form a network called trail ending. 1

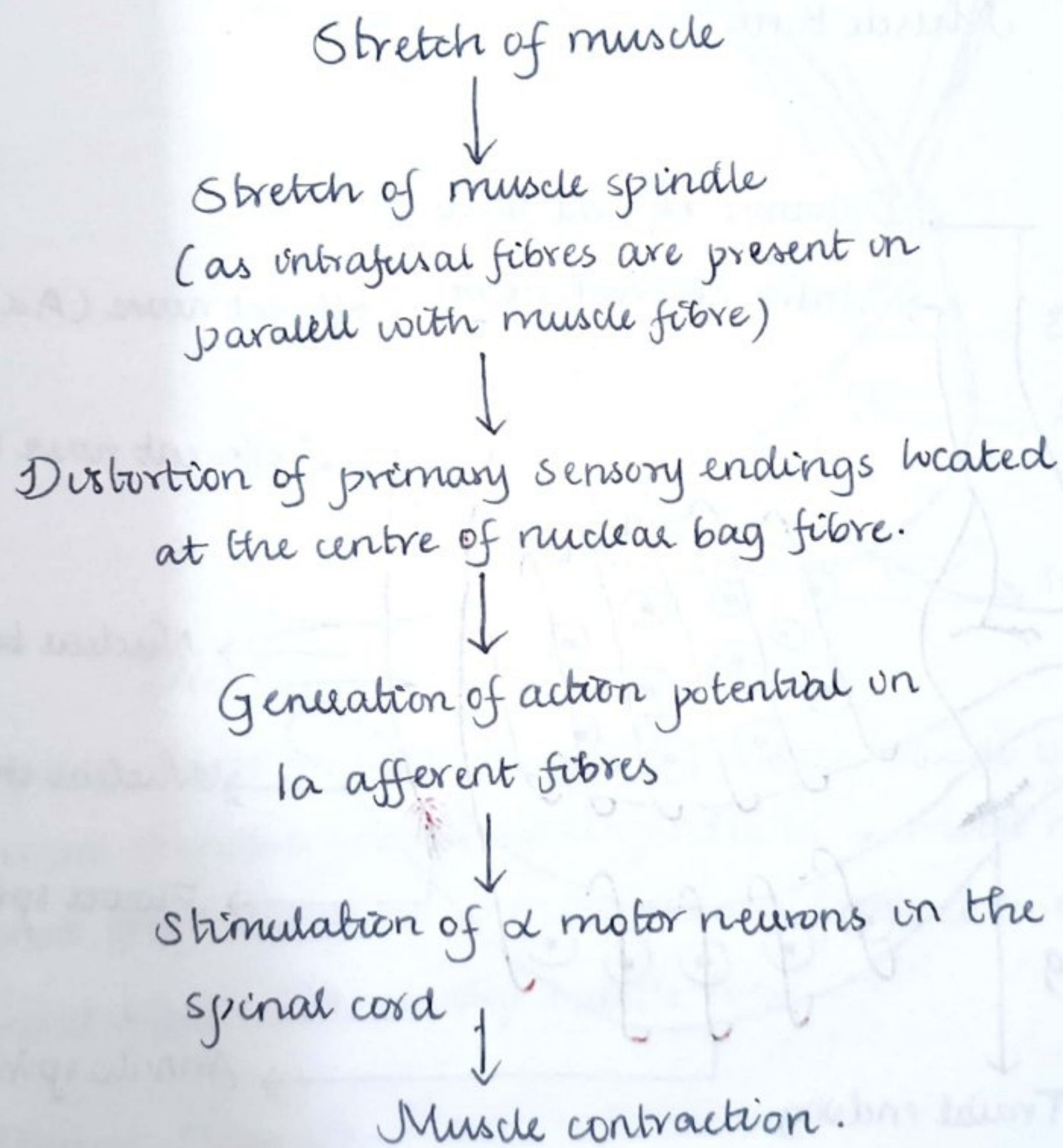
- Functionally it is known as static gamma efferent nerve fibre.
- Sometimes it also gives branch to nuclear bag fibre.

Functions of muscle spindle :-

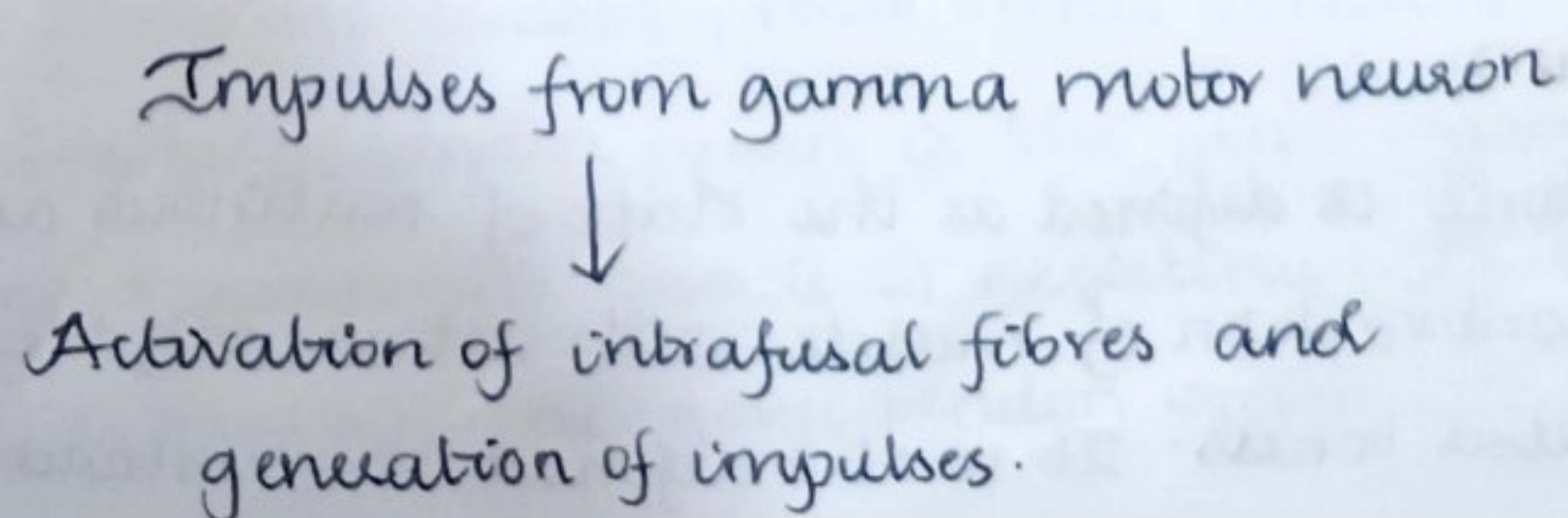
- Muscle spindle gives response to change in the length of the muscle.
- It detects how much the muscle being stretched and sends this information to central nervous system via sensory nerve fibres.
- The information is processed in CNS to determine the position of different parts of the body
- By detecting change in length of muscle, the spindle plays an imp role in preventing the overstretching of the muscles. Muscle spindle has two functions.

- It forms receptor organ for stretch reflex.
- It plays an imp role in maintaining muscle tone.

③ Role of muscle spindle in stretch reflex :-



④ Role of muscle spindle in Muscle tone :-



Transmission of impulses to spinal cord
via primary sensory nerve fibres. (afferent fibres)

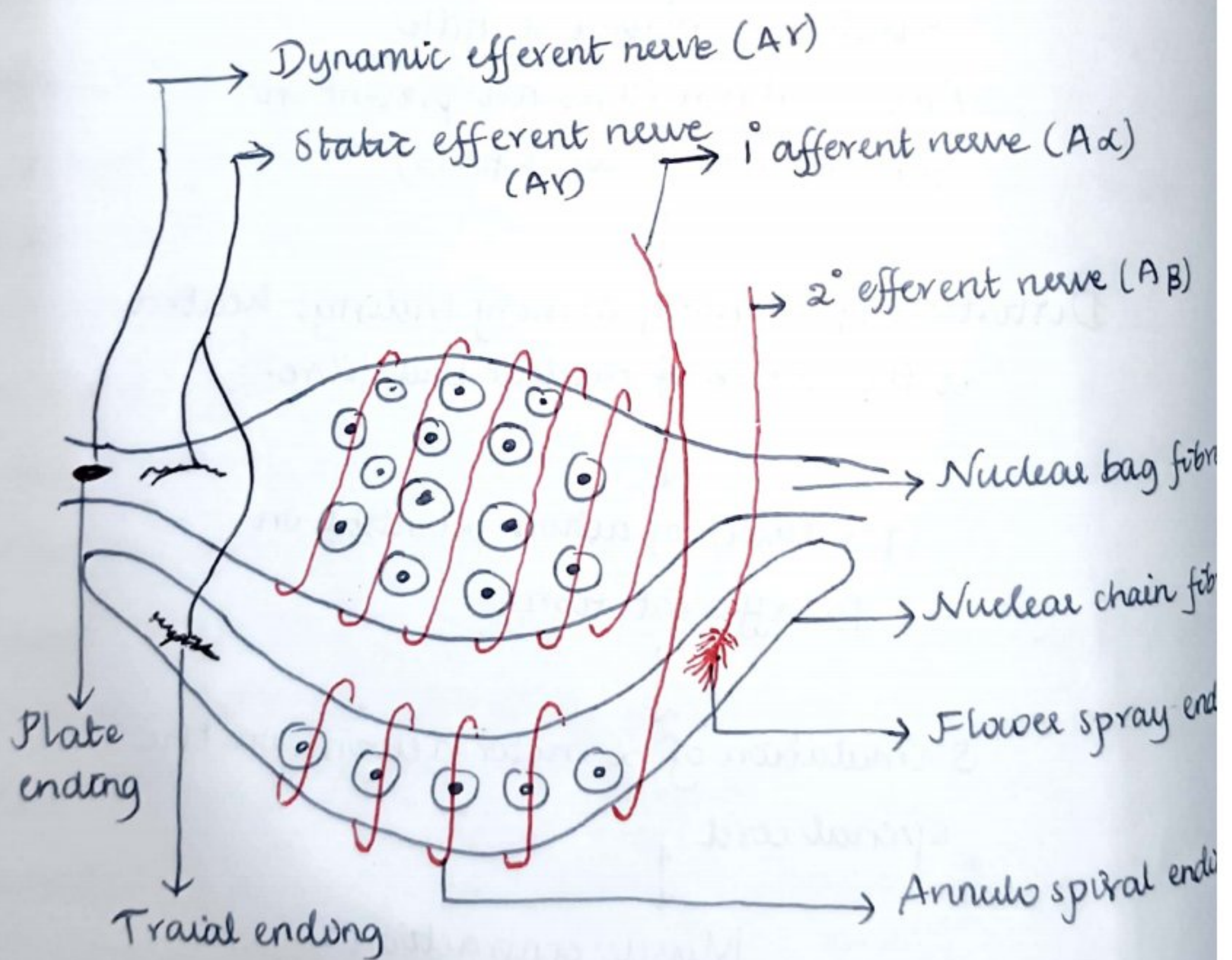
↓
Stimulation of alpha motor neurons

↓
Transmission of impulses via alpha motor
neuron fibres (efferent fibres)

↓
Stimulation of extrafusal fibres

↓
Partial contraction of muscle

↓
Muscle tone.



Muscle Spindle.

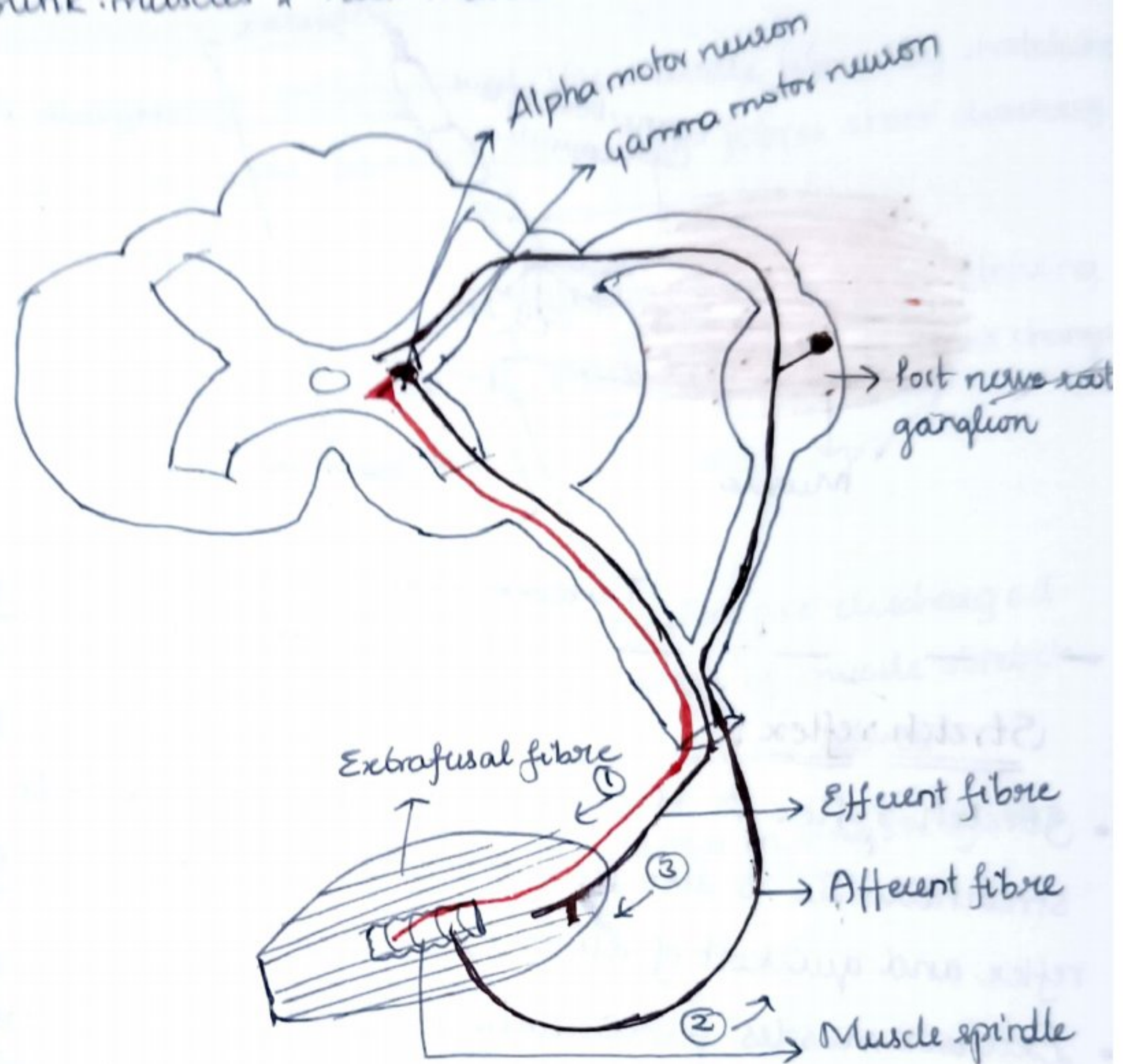
Muscle tone :-

Muscle tone is defined as the state of continuous and passive partial contraction of muscle with certain vigor and tension. It is also called tonus. It is also defined as resistance offered by

the muscle to stretch.

It plays an important role in maintenance of posture. It is present in all skeletal muscles. Change in muscle tone enables movement of different parts of the body.

• Muscle tone is more in antigravity muscles such as extensors of lower limb, trunk muscles & neck muscles.



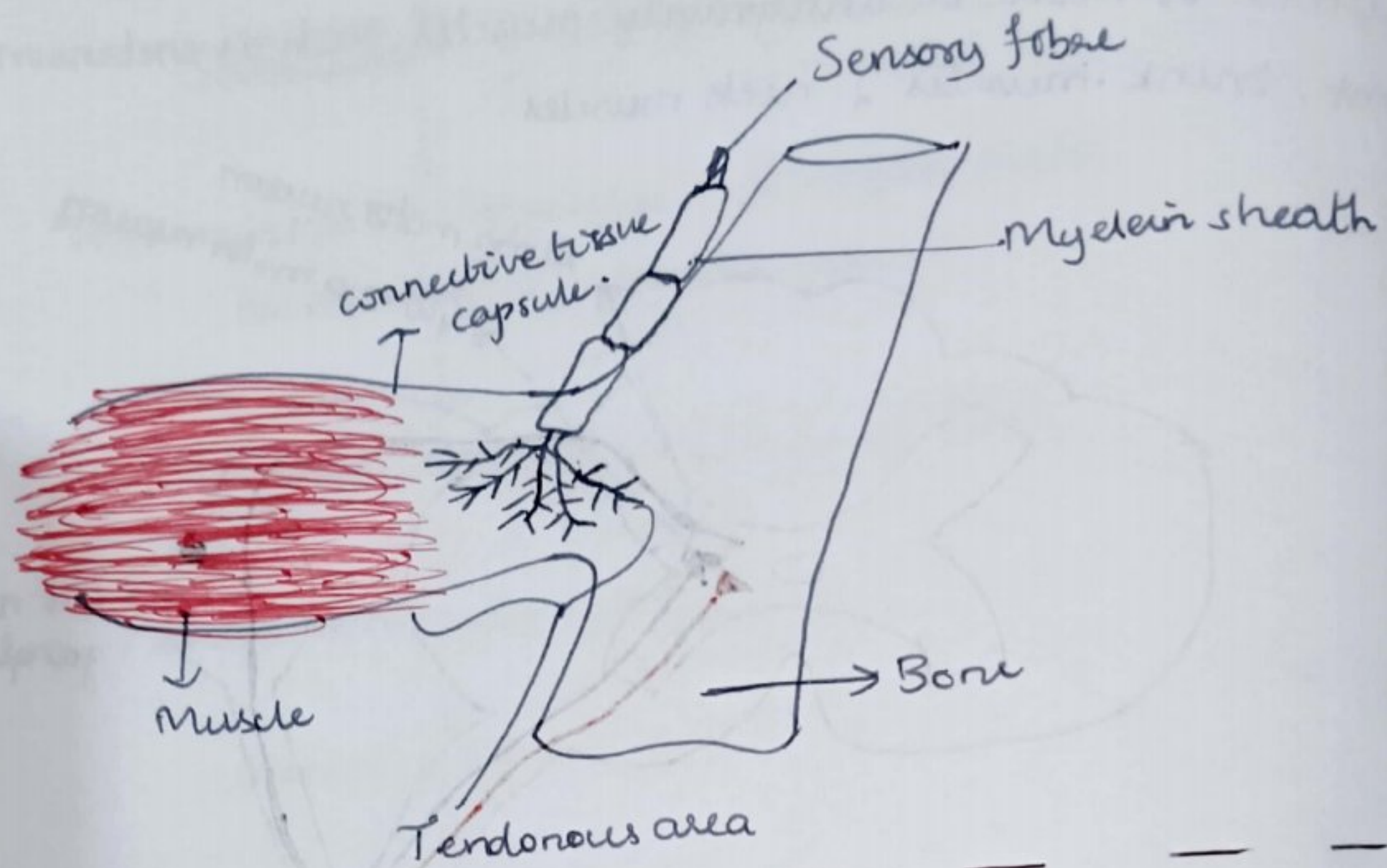
Development of Muscle tone.

- ① Impulses from γ -motor neuron stimulate muscle spindle
- ② Afferent impulses from muscle spindle to α -motor neuron.
- ③ Efferent impulses from α -motor neuron produce contraction of extrafusal fibres and develop muscle tone.

Golgi Tendon Organ :-

The Golgi Tendon Organ is a proprioceptor, sense organ that receives information from the tendon, that senses TENSION. When you lift weights, the golgi tendon organ is the sense organ that tells you how much tension the muscle is exerting. If there is too much muscle tension the golgi tendon organ will inhibit

the muscle from creating
injuring itself.



Stretch reflex:

- Stretch reflex is the reflex contraction of muscle when it is stretched. It is also called myotatic reflex. It is a monosynaptic reflex and quickest of all the reflexes.
- Extensor muscles particularly the antigravity muscles exhibit a severe and prolonged contraction during stretch reflex.
- Stretch reflex plays an imp role in maintaining posture. Muscle spindle is a receptor organ for stretch reflex.
- Stimulation of muscle spindle elicits the stretch reflex. Intrafusal muscle fibres are situated parallel to extrafusal muscle fibres and are attached to tendon of the muscle by means of capsule.
- So, stretching of muscle causes stretching of the muscle spindle also. This stimulates the muscle spindle and it discharges the sensory impulses.
- These impulses are transmitted via the primary & secondary sensory fibres to alpha motor neurons in spinal cord.
- Alpha motor neurons in turn send motor impulses to muscles through their fibres and cause contraction of extrafusal fibres.

Response of muscle spindle to stretch:-

When muscle is stretched, primary sensory nerve fibres from muscle spindle discharge impulses. The response is of 2 types.

i) Dynamic response :-

- Dynamic response is the response in which the primary sensory nerve fibres discharge rapidly.
- When there is a change in length of the muscle fibre by stretching primary sensory nerve fibres from nuclear bag fibres start discharging impulses very rapidly.
- But the discharge become less or nil during continuous stretching of the muscle. Discharge of impulses start whenever there is change in degree of stretching of the muscle.

ii) Static response :-

- Static response is the response in which impulses are discharged rapidly and continuously throughout the period of muscle stretch by primary sensory nerve fibres of nuclear chain fibres.
- Thus, the muscle spindle gives response to change in length of the muscle as well as rate of change in length.

Physiologic tremor:-

- Physiologic tremor is the continuous discharge of action potentials with low voltage and ineffective frequency from primary and secondary nerve fibres of muscle spindle at resting condition.
- Physiological tremor plays an imp role in the feedback regulation of muscle length.

Formation of CSF :-

- Site of formation :- CSF is formed by choroid plexuses, situated within the ventricles. Choroid plexuses are tuft of capillary projects present inside the ventricle and are covered by ventricle pia mater and ependymal covering. A large amount of CSF formed in the lateral ventricles.

• Mechanism of formation :-

- CSF is formed by the process of secretion that involves active transport mechanism.
- Formation of CSF does not involve ultrafiltration or dialysis.

Substances affecting the CSF formation :-

- ① Pilocarpine, ether & extracts of pituitary gland stimulate the secretion of CSF by stimulating choroid plexuses.
- ② Injection of isotonic saline also stimulates CSF formation.
- ③ Injection of hypotonic saline causes greater rise in capillary pressure & intracranial pressure and fall in osmotic pressure, lead to increase in CSF formation.
- ④ Hypertonic saline decreases CSF formation & CSF pressure.

Circulation of CSF :-

- Major quantity of CSF is formed in lateral ventricles and enters third ventricle by passing through foramen of Monro.
- From here it passes to 4th ventricle through aqueductus Sylvius.
- From 4th ventricle, CSF enters the cisterna magna and cisterna lateralis through foramen of Magendie (central opening) and foramen of Luschka (lat opening). From cisterna magna and cisterna lateralis, CSF circulates through subarachnoid space over spinal cord and cerebral hemispheres.
- It also flow into central canal of spinal cord.
- It is absorbed by the arachnoid villi into dural sinuses and spinal veins. Small amount is absorbed along perineural spaces.

Pressure exerted by CSF :-

Pressure exerted by CSF in man varies in diff position. Ex.

Lat recumbent position - 10-18 cm of H_2O

Lying π - 13 cm of H_2O

Sitting " - 30 cm of H_2O

- Certain events like coughing & crying increase the pressure by decreasing absorption.
- Compression of internal jugular vein also raises the CSF pressure.

Functions of CSF :-

1. Protective function :-

- CSF acts as a fluid buffer and protects the brain from shock. Since, the specific gravity of brain and CSF is more or less same, brain floats in CSF.
- When head receives a blow, CSF acts like a cushion and prevents the movement of brain against the skull bone and thereby prevents the damage of brain.
- However, if head receives a severe blow, the brain moves forcefully and hits against the skull bone, leading to damage of brain tissues.
- Brain strikes against the skull bone at a point opp to the point, where the blow was applied. So, this type of damage is called counter coup injury.

2. Regulation of cranial content volume :-

- Regulation of cranial content volume is essential because, brain may be affected if the volume of cranial content increases.
- It happens in cerebral hemorrhage and brain tumors. Increase in cranial content volume is prevented by greater absorption of CSF to give space for increasing cranial contents.

3. Medium of Exchange :-

CSF is the medium through which many substances, particularly nutritive substances and waste materials are exchanged b/w blood & brain tissue.



Functions :-

① Regulation of food intake :-

Food intake is controlled by 2 nuclei. They are

i) Ventromedial nucleus (VMN) - Satiety centre

Inhibits the feedings centre and produces satiety (satisfaction) after taking food.

Destruction of VMN - Hyperphagia & obesity

Stimulation of VMN - Hypophagia ($\downarrow$ food intake)

ii) Lateral nucleus (LN) - feeding centre

Stimulates appetite and produces hunger

Destruction of LN - aphagia & starvation

Stimulation of LN - Hyperphagia ($\uparrow$ food intake)

Hypothesis about food intake :-

① Glucostatic Hypothesis :-

Hyperglycemia in ^{bl} food $\rightarrow \downarrow$ VMN activity $\rightarrow \downarrow$ food intake

Hypoglycemia in blood $\rightarrow \uparrow$ VMN activity $\rightarrow \uparrow$ food intake

② Lipostatic hypothesis :-

Adipose tissue secretes "leptin" $\rightarrow$ inhibits hypothalamus $\rightarrow \downarrow$ food intake

③ Gut peptide hypothesis :-

Presence of food in GI tract $\rightarrow$ release of intestinal peptides (GRP, ghrelin, somatostatin & CCK) $\rightarrow$ acts on brain $\rightarrow$ satiety.

④ Thermostatic hypothesis :-

$\downarrow$ Body temperature $\rightarrow \uparrow$ food intake

$\uparrow$ Body temperature $\rightarrow \downarrow$ food intake.

② Regulation of water intake :-

• Mainly involves thirst centre. Dorsal or lateral hypothalamus acts as thirst centre.

• Water intake mainly depends upon the stimulation of thirst centre.

• Thirst centre is stimulated under 2 conditions.

a) in the increased tonicity of the body fluid

b) in the decreased ECF volume.

a) Increase in the tonicity of body fluid

↓
Stimulation of osmoreceptors

↓
Stimulation of thirst centre

↓
Water intake.

b) Decrease in ECF volume

↓
Baroreceptors

↓
Stimulation of Thirst centre

↓
↑ Water intake

↓
Angiotensin II

↓
Subfornical organ

↓
Thirst centre.

③ Regulation of Body temperature :-

- Hypothalamus is called 'as biological thermostat' as it controls the body temperature.

- Involves preoptic region of the ant and post hypothalamus.

Preoptic nucleus of ant hypothalamus - heat loss centre

" " " post " - heat gain centre.

Stimulation of the centres occurs through 2 mechanisms :

a) Cutaneous thermoreceptors

b) Blood flowing through hypothalamus.

Stimulation of $\left\{ \begin{array}{l} \text{ant hypothalamus - Vasodilation, sweating \& panting} \\ \text{post hypothalamus - Vasoconstriction, shivering \& piloerection.} \end{array} \right.$

④ Control of ANS :-

Stimulation of ant hypothalamus

↓

Increased HR, BP & HCL secretion
Micturition & erection of penis

Stimulation of post hypothalamus

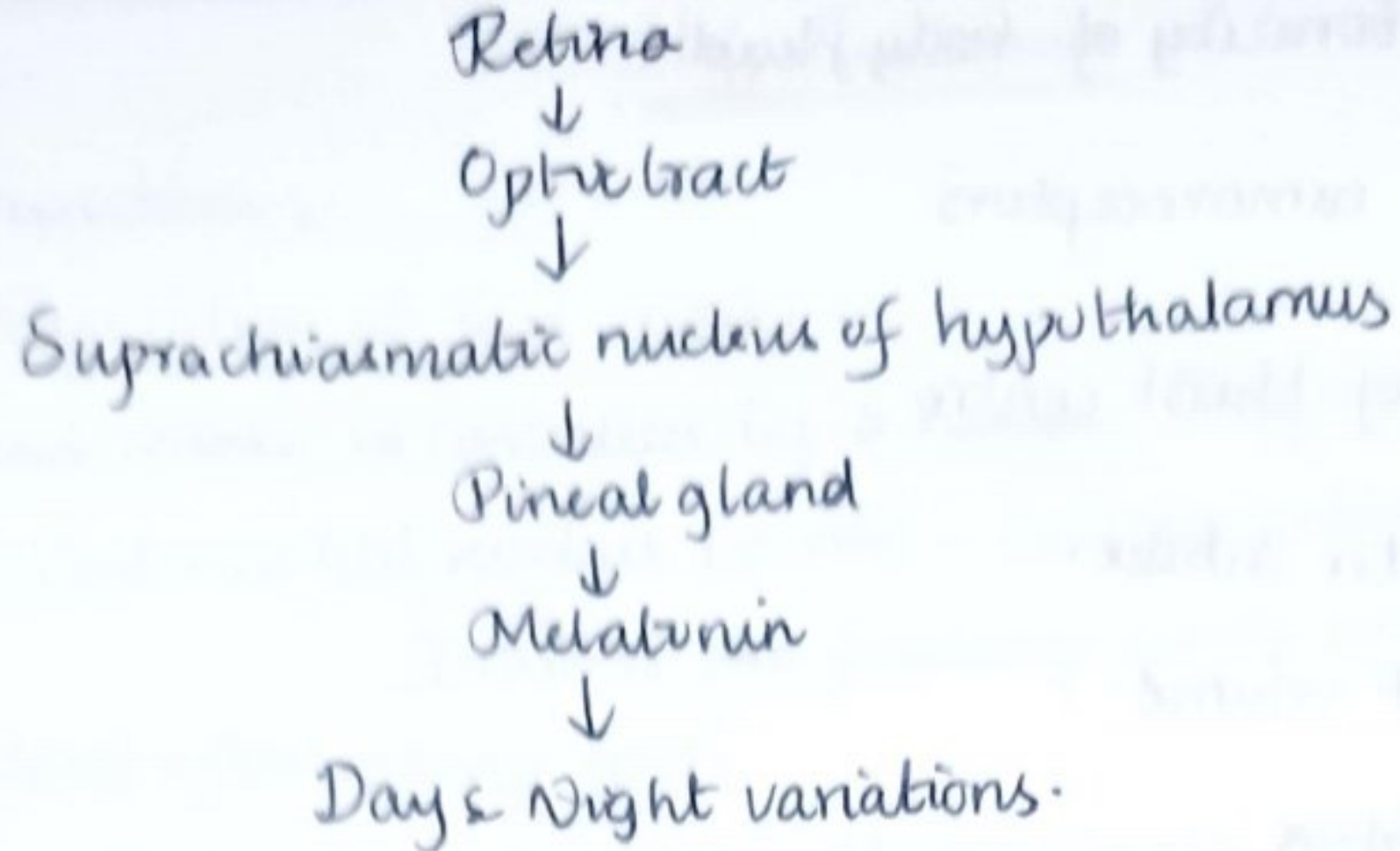
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Decreased HR, BP, Pupillary dilation, sweating & piloerection.

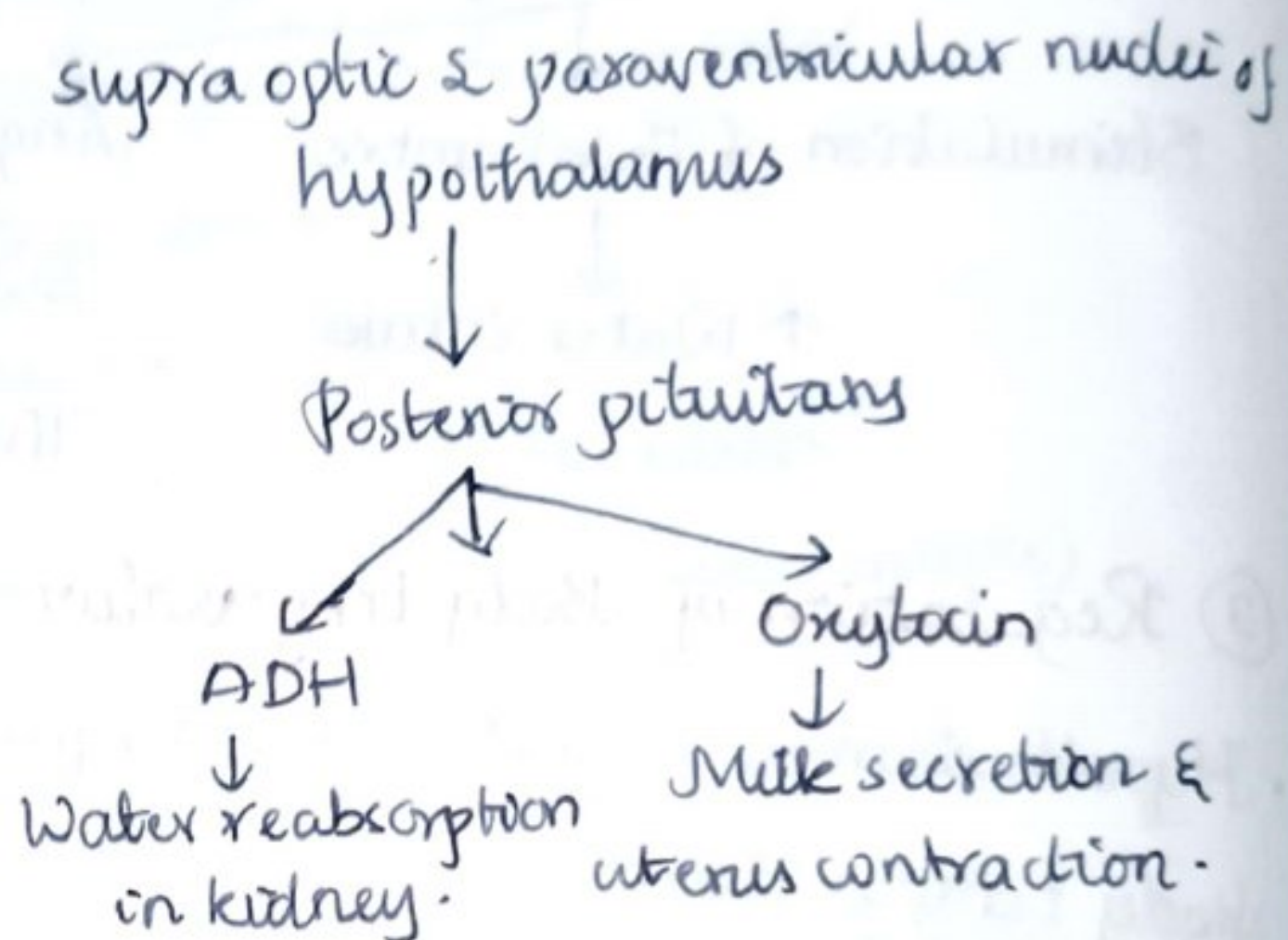
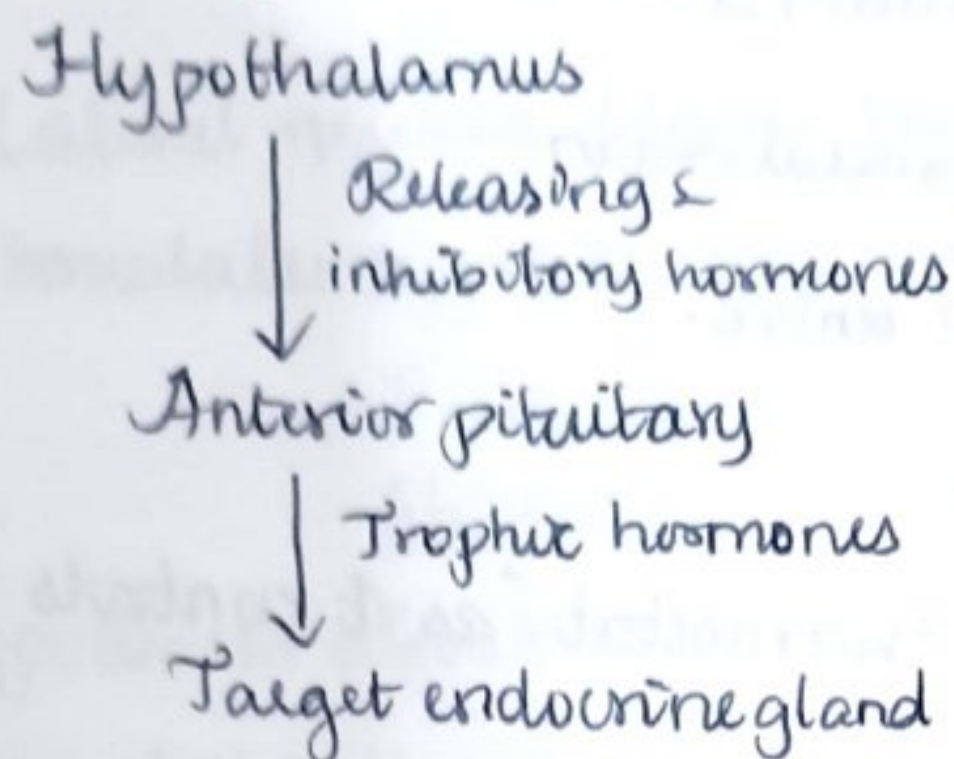
⑤ Role in Circadian Rhythm :-

Hypothalamus play a role in influencing the changes in body functions tuned to the day & night cycle.



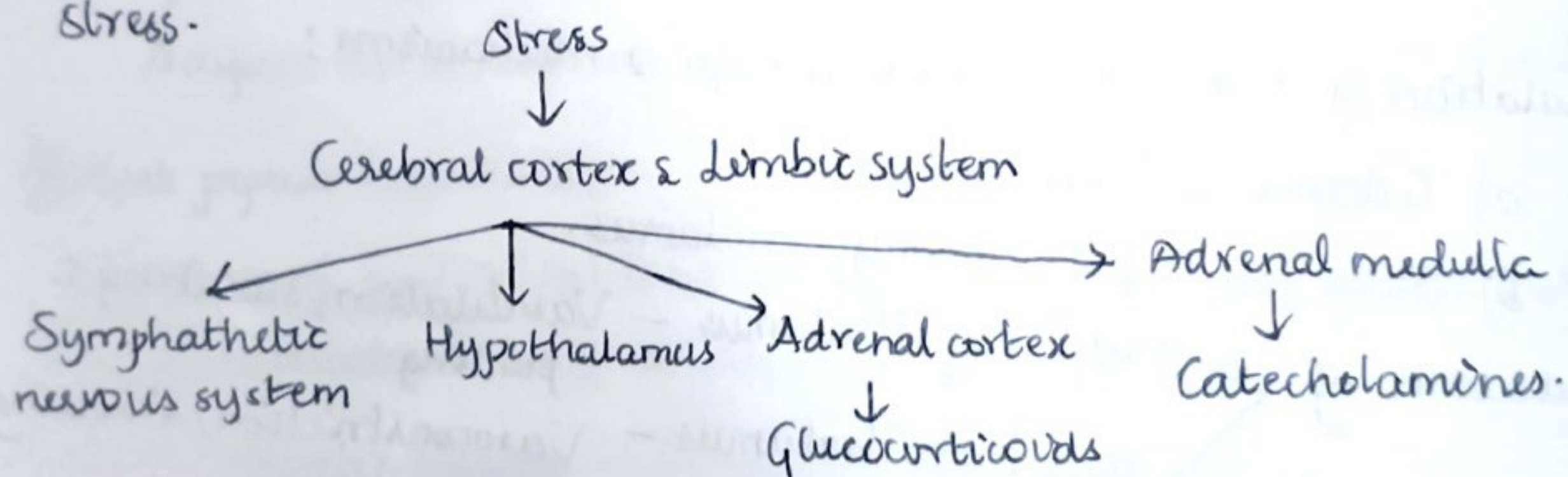


⑥ Control of Pituitary :-



⑦ Role in stress :-

Hypothalamus helps to protect the body from damaging effects of stress.



⑧ Role in emotions :-

Hypothalamus is a part of the limbic system which is responsible for emotional behaviour.

⑨ Role in sexual behaviour :-

Hypothalamic areas are responsible for sexual behaviour like mating, attracting the opposite sex.

⑩ Role in sleep wakeful cycle :-

Hypothalamus has 2 sleep centres
 - diencephalic sleep zone.
 - basal fore brain " "

Electroencephalography (EEG)

- Electroencephalography is the study of electrical activities of brain. It is the graphical recording of electrical activities of brain.
- Electrical activity of brain is complicated when compared to that of single nerve fibre or neuron.
- It is due to involvement of large no. of neurons and synapses.
- Hans Berger was the first one to analyze EEG waves systematically and hence the EEG waves are referred as Berger waves.

Significance of EEG :-

- Electroencephalogram is useful in the diagnosis of neurological disorders and sleep disorders.
- EEG pattern is altered in following conditions or disorders.
 1. Epilepsy - which occurs due to excessive discharge of impulses from cerebral cortex.
 2. Disorders of midbrain affecting ascending reticular activating system.
 3. Subdural hematoma during which there is collection of blood in subdural space over the cerebral cortex.

Method of recording EEG :-

- The electrodes called scalp electrodes from instrument are placed over unopened skull or over the brain after opening the skull or by piercing into brain.
- Electrodes are of 2 types, unipolar and bipolar electrodes. While using bipolar electrodes, both the terminals are placed in diff parts of brain.
- When unipolar electrodes are used, the active electrode is placed over cortex and the ⁱⁿdifferent electrode is kept on some part of the body away from cortex.

Waves of EEG :-

- Electrical activity recorded by EEG may have synchronized or desynchronized waves.
- Synchronized waves are the regular and invariant waves, whereas desynchronized waves are irregular and variant.

In normal persons, EEG has 5 types of waves.

① Alpha rhythm :-

- Alpha rhythm consists of rhythmical waves. Alpha waves are synchronized waves. Alpha rhythm is obtained in inattentive brain or mind as in drowsiness, light sleep, or narcosis with closed eyes.
- It is abolished by visual stimuli or by any other type of stimuli or by mental effort. So, it is diminished when eyes are open.
- Waves of alpha rhythm are most marked in parietooccipital area.

Alpha block :-

- Alpha block is the replacement of synchronized alpha waves in EEG by desynchronized and low voltage waves when the eyes are opened.
- The desynchronized waves do not have specific frequency. It occurs due to any form of sensory stimulation or mental concentration such as solving arithmetic problems.
- Desynchronization is the common term used for replacement of regular alpha waves with irregular low voltage waves.
- It is due to loss of synchronized activity in neural elements that are responsible for regular pattern.

Beta rhythm :-

- Beta rhythm includes high frequency waves. Beta waves are desynchronized waves.
- Beta rhythm is recorded during mental activity or mental tension or arousal state. It is not affected by the opening of eyes.
- During higher mental activity or peak performance state like conscious activity, problem solving and fear, very high frequency waves appear.
- Some controversy exists that high frequency waves are gamma rhythm but many scientists consider these waves as beta rhythm.

Delta rhythm :-

- Delta rhythm includes waves with low frequency and high amplitude. It is common in early childhood during waking hours.
- In adults, it appears mostly during deep sleep. Presence of delta waves in adults during conditions other than sleep indicates the pathological process in brain like tumor, epilepsy, increased intracranial pressure & mental deficiency or depression.
- These waves are not affected by opening the eyes.

Theta waves :-

Theta waves are obtained in children generally below 5 yrs of age. These waves are of low frequency & low voltage.

Stages of sleep & EEG pattern :-

Rapid Eye Movement sleep :- During REM sleep, EEG shows irregular waves with high frequency & low amplitude. These waves are desynchronized waves.

Non Rapid Eye Movement Sleep :-

The NREM sleep is divided into 4 stages based on EEG pattern. During the stage of wakefulness i.e. while lying down with closed eyes & relaxed mind, the alpha waves of EEG appear.

Stage ① Stage of Drowsiness :- Alpha waves are diminished & abolished. EEG shows only low voltage fluctuations & infrequent delta waves.

② Stage of Light Sleep :- Characterized by spindle bursts and superimposed by low voltage delta waves.

③ Stage of medium sleep :- During this stage, the spindle bursts disappear. Frequency of delta waves $\uparrow$ s & amplitude $\uparrow$ s.

④ Stage of Deep sleep :- Delta waves become prominent with low frequency and high amplitude.

- Klüver-Bucy syndrome
- It is observed in animals, particularly monkeys after the bilateral ablation of temporal lobe along with amygdala & uncus.
 - It occurs in human beings during bilateral lesions of these structures.

1. Aphasia (disturbance in speech)

- 2. Auditory disturbances such as frequent attacks of tinnitus, auditory hallucinations with sounds like buzzing, ringing or humming.

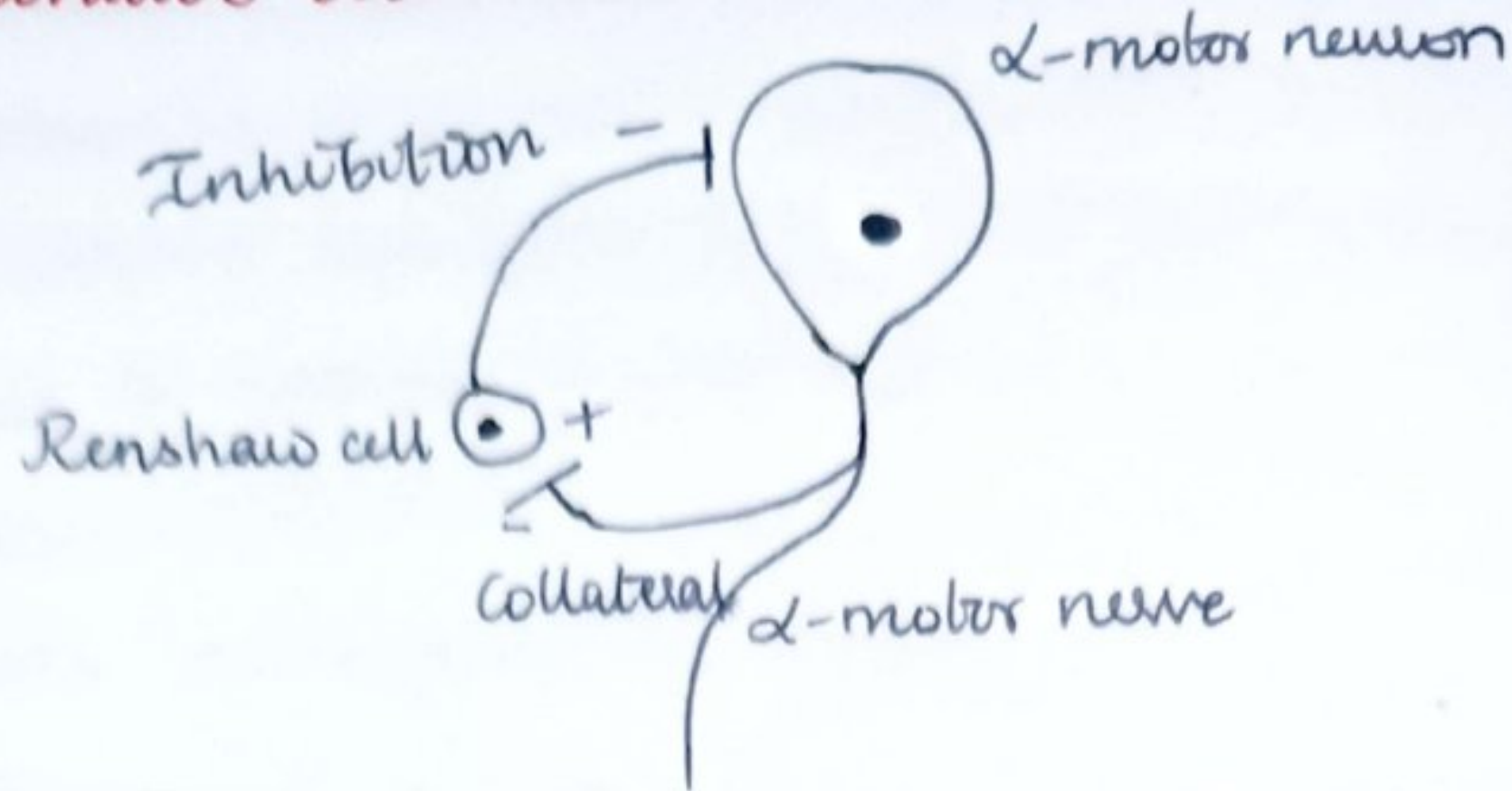
3. Disturbances in smell and taste sensations.

4. Dreamy states: The patients are not aware of their own activities and have the feeling of unreality.

5. Visual hallucinations associated with hemianopia.

Paradoxical Sleep:

- Rapid eye movement sleep is the type of sleep associated with rapid conjugate movements of the eyeballs, which occurs frequently.
- Though the eyeballs move, the sleep is deep. So, it is also called paradoxical sleep.
- It occupies about 20% - 30% of sleeping period. Functionally REM sleep is very imp because, it plays imp role in consolidation of memory. Dreams occur during this period.



Renshaw cell or Negative Feedback inhibition.

- Negative feedback inhibition is the type of synaptic inhibition which is caused by Renshaw cells in spinal cord.
- Renshaw cells are small motor neurons present in anterior gray horn of spinal cord. Ant nerve root consists of nerve fibres, which leave the spinal cord.
- The nerve fibres arise from α motor neurons in ant gray horn of the spinal cord and reach the effector organ (muscles).
- Some of the fibres called collaterals fibres terminate on Renshaw cells instead of leaving the spinal cord.
- When motor neurons send motor impulses, some of the impulses reach the Renshaw cell by passing through collaterals.
- Now, Renshaw cell is stimulated. In turn, it sends inhibitory impulses to α motor neurons. So that, the discharge from motor neurons is reduced.
- In this way, Renshaw cell inhibition represents negative feedback mechanism.
- A Renshaw cell may be supplied by more than one alpha motor neuron collateral and it may synapse on many motor neurons.

Stereognosis :-

- Stereognosis is defined as ability to recognize the known objects by touch with closed eyes.
- It is also a synthetic sense produced by combination of touch and pressure sensations.

Receptor Potential

Defination:-

- Receptor potential is a nonpropagated transmembrane potential difference that develops when a receptor is stimulated. It is also called generator potential.
- Receptor potential is short lived and hence it is called transient receptor potential. It is not action potential. It is graded potential.
- It is similar to excitatory postsynaptic potential in synapse, endplate potential in neuromuscular junction & electrotonic nerve potential in the nerve fibre.

Properties:-

- Receptor potential has 2 imp properties
- i. Receptor potential is nonpropagated, it is confined within the receptor itself.
- ii. It does not obey all or none law.

Significance:-

- When receptor potential is sufficiently strong, it causes development of action potential in the sensory nerve.

Mechanism of Development of Receptor Potential:-

- Pacinian corpuscles are generally used to study the receptor potential because of their large size and anatomical configuration.
- These corpuscles are easily dissected from experimental animals.
- In the pacinian corpuscle, the tip of nerve fibre is unmyelinated.
- The unmyelinated nerve tip extends through the corpuscle as centre core fibre.
- Pacinian corpuscles give response to pressure stimulus. When pressure stimulus is applied, the pacinian corpuscle is compressed.
- The compression causes elongation or change in shape of the corpuscle.
- The change in shape of the corpuscle leads to deformation of centre core fibres of the corpuscle. This results in opening of gated sodium channels.
- So, the positively charged sodium ions enter the interior of nerve fibre. This produces mild depolarization, i.e. receptor potential.

- Generation of action potential
- Receptor potential causes development of a local circuit of current flow, which spreads along the unmyelinated part of nerve fibre within the corpuscle.
 - When this local circuit of current reaches the 1st node of Ranvier within the corpuscle, it causes opening of voltage gated sodium channels and entrance of sodium ions into the nerve fibre.
 - This leads to development of action potential in nerve fibre.

⇒ Phantom limb & its basis

- When a sensory pathway from receptor to cerebral cortex is stimulated on any particular site along its course, the sensation caused by stimulus is always felt at the location of receptor, irrespective of site stimulated.
- This phenomenon is known as Law of Projection.

Examples of Law of Projection :-

- If somesthetic area in right cerebral cortex, which receives sensation from left hand is stimulated, sensations are felt in left hand and not in head.
- Sensation complained by amputated patients in the missing limb (phantom limb) is the best example of law of projection. For if a leg has been amputated, the cut end heals with scar formation.
- The cut ends of nerve fibre are merged within the scar.
- If cut end of sensory fibres are stimulated during movement of thigh, the patient feels as the sensation is originating from non-existent leg.
- Sometimes, the patient feels pain in non-existent leg or limb. This type of pain is called phantom limb pain.

⇒ Synaptic delay :-

- Synaptic delay is the short delay that occurs during the transmission of impulses through the synapse. It is due to the time taken for

1. Release of neurotransmitter



(i) Passage of neurotransmitter from axon terminal to postsynaptic membrane.

(ii) Action of the neurotransmitter to open ionic channels in postsynaptic membrane.

(iv) Normal duration of synaptic delay is 0.3-0.5 millisecond.

Synaptic delay is one of the causes for reaction time of reflex activity.

Significance of Determining Synaptic Delay:-

Determination of synaptic delay helps to find out whether the pathway for reflex is monosynaptic or polysynaptic.

⇒ GABA

- Gamma-aminobutyric acid (GABA) is an inhibitory neurotransmitter in synapses particularly in CNS. It is responsible for pre-synaptic inhibition.

- It is secreted by nerve endings in the following structures:-

1. Cerebral cortex.
2. Cerebellum
3. Basal ganglia.
4. Spinal cord.
5. Retina.

- GABA causes synaptic inhibition by opening potassium channels and chloride channels. So potassium comes out of synapse & chloride enters in.

- This leads to hyperpolarization which is known as inhibitory post synaptic potential.

⇒ Role of Synaptic Inhibition:-

Synaptic inhibition in CNS limits the no. of impulses going to muscle and enables the muscles to act properly & appropriately. Thus, the inhibition helps to select exact no. of impulses & to omit/block excess ones.

- When poison like strychnine is introduced into the body, it destroys



- The inhibitory functioning of the basal ganglia is impaired, leading to involuntary and convulsive contraction even with slight stimulation.
- In the nervous disorders like parkinsonism, the inhibitory system is impaired resulting in rigidity.

Basal Ganglia

Components :-

Basal ganglia include 3 primary components

1. Corpus striatum
2. Substantia nigra
3. Subthalamic nucleus of Leys.

Corpus Striatum :-

- Corpus striatum is a mass of gray matter situated at the base of cerebral hemispheres in close relation to thalamus.
- Corpus striatum is incompletely divided into 2 parts by internal capsule

i. Caudate nucleus :-

- Caudate nucleus is an elongated arched gray mass, lying medial to internal capsule.
- Throughout its length, the caudate nucleus is related to lateral ventricle. It consists head portion and tail portion.
- Head is bulged into lateral ventricle and situated dorsal to thalamus. The tail is long and arched.
- It extends along the dorsolateral surface of thalamus & ends in amygdaloid nucleus.

ii. Lenticular nucleus :-

- Lenticular nucleus is a wedge shaped gray mass, situated lateral to internal capsule.
- A vertical plate of white matter called external medullary lamina, divides lenticular nucleus into 2 portions.
 - a. Outer putamen.
 - b. Inner globus pallidus
- Putamen and caudate nucleus are the phylogenetically newer

parts of corpus striatum and these 2 parts are together called neostriatum or striatum.

- Globus pallidus is phylogenetically older part of corpus striatum. And it is called pallidum or palaeostriatum.

Substantia Nigra :-

- Substantia Nigra is situated below red nucleus. It is made up of large pigmented and small non pigmented cells.
- The pigment contains high quantity of iron.

Subthalamic Nucleus of Luys :-

- Subthalamic nucleus is situated lateral to red nucleus and dorsal to substantia nigra.

Connections :-

In corpus striatum :-

⇒ Afferent connections from

- Thalamic nuclei to caudate nucleus & putamen.
- Cerebral cortex to " "
- Substantia nigra to " "
- Subthalamic nucleus to globus pallidus.

⇒ Efferent connections to

- Thalamic nuclei
- Subthalamic nucleus
- Red nucleus
- Substantia nigra
- Hypothalamus.

In substantia nigra :-

Afferent connections from

- Putamen
- Red nucleus
- Frontal lobe of cerebral cortex.
- Medial & lateral lemnisci

Efferent connections to

- Putamen.

In Subthalamic nucleus of Luys :-

Afferent connections from

Efferent connections to



a) Globus pallidus

a) Globus pallidus.
b) Red nucleus.

• In addition to afferent and efferent connections, diff components of corpus striatum of the same side are interconnected by intrinsic fibres

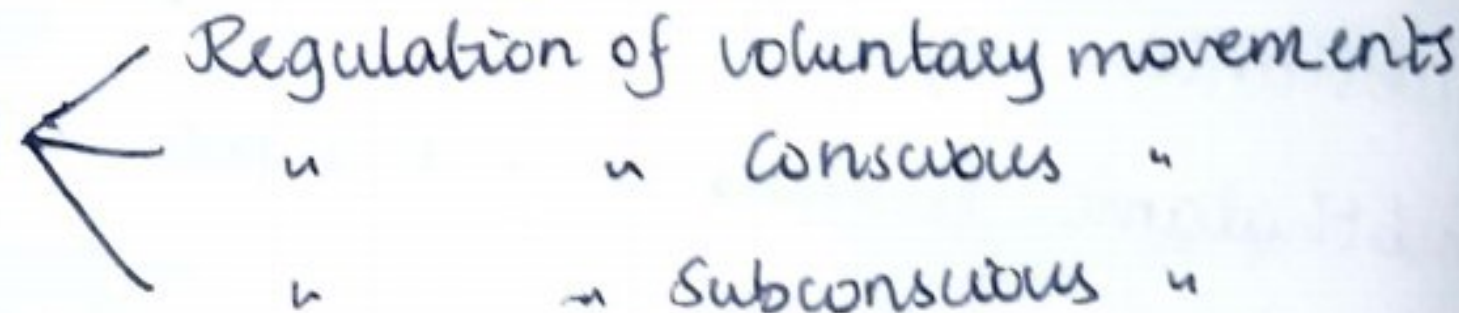
1. Putamen to globus pallidus
2. Caudate nucleus to putamen
3. " " " to globus pallidus.

• Different components of corpus striatum on each side are connected to those of the opposite side by commissural fibres.

Functions :-

• Basal ganglia form the part of extrapyramidal system, which is concerned with integration & regulation of motor activities.

① Control of muscle tone :- Basal ganglia decrease the muscle tone by inhibiting gamma motor neurons. During the lesion of basal ganglia, muscle tone increases leads to rigidity.

② Control of motor activity 

③ Control of Reflex muscular activities :- Some reflex muscular activities, like visual & labyrinthine reflexes are imp in maintaining posture.

④ Control of Automatic associated movements :

Ex: Swing of arms while walking, appropriate facial expression while talking or doing any work.

⑤ Role in Arousal mechanism - Globus pallidus & red nucleus are involved in arousal mechanism.

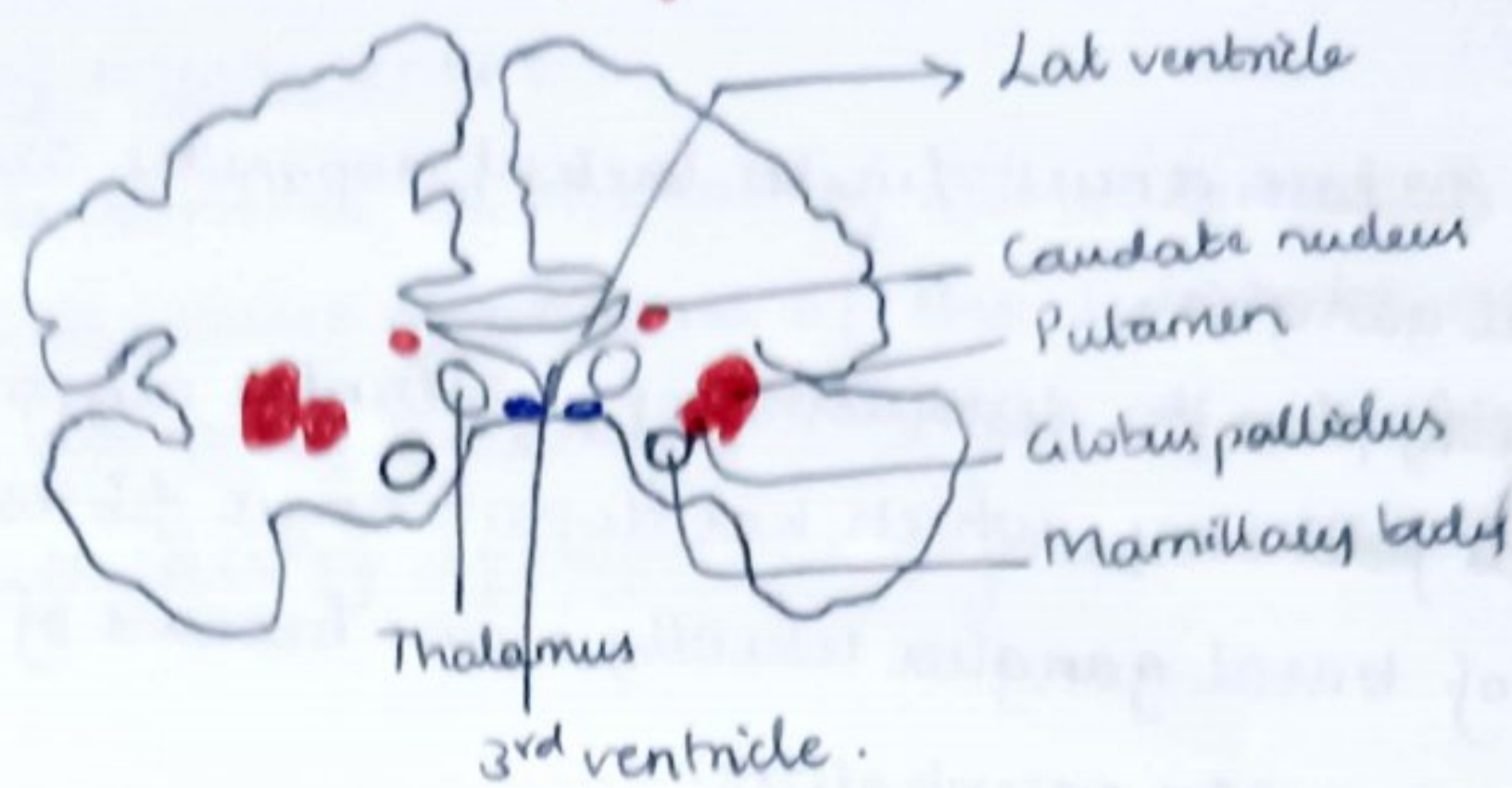
Neurotransmitters involved :-

Dopamine and GABA → Inhibition

Acetylcholine, Substance P, Enkephalins,
Noradrenaline, Glutamic acid → Excitation.



Basal Ganglia.



⇒ Referred pain :-

- Referred pain is the pain that is perceived at a site adjacent to or away from the site of origin. Deep pain & some visceral pain are referred to other areas. But superficial pain is not referred.

Examples :-

1. Cardiac pain is felt at inner part of left arm and left shoulder.
2. Pain in ovary is referred to umbilicus.
3. Pain from testis is felt in abdomen.
4. Pain on diaphragm is referred to shoulder.
5. Pain in gall bladder is referred to epigastric region.
6. Renal pain is referred to loin.

Mechanism of Referred pain :-

- According to dermatomal rule, pain is referred to a structure, which is developed from same dermatome from which the pain producing structure is developed.
- A dermatome includes all the structures or parts of body, which are innervated by aff nerve fibres of one dorsal root.
- For example, the heart & inner aspect of left arm originate from same dermatome. So, the pain in heart is referred to left arm.

⇒ Parkinsonism :-

- Parkinson disease is a slowly progressive degenerative disease of nervous system associated with destruction of brain cells, which produce dopamine.

Causes :-

- Parkinson disease occurs due to lack of dopamine caused by damage of basal ganglia.
- It is mostly due to destruction of substantia nigra and the nigrostriatal pathway, which has dopaminergic fibres.
- Damage of basal ganglia usually occurs because of following causes:
 - i. Viral infection like encephalitis.
 - ii. Cerebral atherosclerosis.
 - iii. Injury to basal ganglia.
 - iv. Destruction or removal of dopamine in basal ganglia. It occurs mostly due to long treatment with anti-HTN drugs like reserpine. Parkinsonism due to drugs is known as drug-induced parkinsonism.
 - v. Destruction of basal ganglia due to unknown reasons called idiopathic parkinsonism.

Signs & symptoms :-

- Parkinson disease develops very slowly & the early signs & symptoms may be unnoticed for months or even years.
- Often symptoms start with a mild noticeable tremor in just one hand.
- When the tremor becomes remarkable the disease causes slowing or freezing of movements followed by rigidity.
- i. Tremor :-
 - In parkinson disease, tremor occurs during rest, but it disappears while doing any work. So, it is called static tremor or resting tremor. Also called drum beating tremor.
 - Thumb moves rhythmically over the index & middle fingers. These movements are called pill-rolling movements.
- ii. Slowness of movements :-
 - Over the time, movements start slowing down (bradykinesia) and it takes a long time even to perform a simple task.
 - Gradually the patient becomes unable to initiate the voluntary activity (akinesia) or the voluntary movements are reduced.

(hypokinesia). It is because of hypertonicity of muscles.

iii. Poverty of movements :-

Poverty of movements is the loss of all automated associated movements. Because of absence of the automatic associated movements, the body becomes statue like, face becomes mask like due to absence of appropriate expressions like blinking and smiling.

iv. Rigidity :-

- Stiffness of muscles occurs in limbs resulting in rigidity of limbs.
- Muscular stiffness occurs because of increased muscle tone. It affects both flexor & extensor muscles equally.
- So, the limbs become more rigid like pillars. The condition is called lead-pipe rigidity.
- In later stages, the rigidity extends to neck & trunk.

v. Gait :-

Gait refers to manner of walking. The patient loses the normal gait. Gait in parkinsonism is called festinant gait.

vi. Speech problems

vii. Emotional changes

viii. Dementia

Treatment :-

- It is treated by Dopamine injection. But dopamine does not cross the blood brain barrier.
- So, another substance called L-dopa which crosses the blood brain barrier is injected. L-dopa moves into brain & there it is converted into dopamine.
- Since, L-dopa can be converted into dopamine in liver, some side effects occur due to excess dopamine in liver & blood. So along with L-dopa another substance called carbidopa is administered.
- Tremor in parkinson disease is abolished by surgical destruction of basal ganglia & thalamic nuclei.

1) Aphasia :-

- Aphasia is defined as the loss or impairment of speech due to brain damage.
- It is an acquired disorder and it is distinct from developmental disorders of speech or other speech disorders like dysarthria.
- Aphasia is not due to paralysis of muscles of articulation.
- It is due to damage of speech centres. Damage of speech centres impairs the expression and understanding of spoken words.
- It also affects reading and writing. Speech function is localised to left hemisphere in most of the people.
- Aphasia may be associated with other speech disorders, which also occur due to brain damage.

Causes :-

- Usually aphasia occurs due to damage of one or more speech centres which are situated in cerebral cortex.
- Damage of speech centres occurs due to
 1. Stroke
 2. Head injury
 3. Severe blow to head
 4. Cerebral tumours
 5. Brain infections
 6. Degenerative disorders.
- In children, traumatic aphasia can develop by exposure to a horrifying event, without any brain damage. It may be cured with psychological treatment.

Types :-

Aphasia is classified by diff methods. The simple & convenient clinical classification divides aphasia into 5 types.

1. Broca aphasia
2. Wernicke "
3. Global "
4. Nominal "
5. Other types of aphasia.

Dyslexia:

Visual aphasia: Inability to understand written symbols - It is also called word blindness or dyslexia and it occurs due to the lesion in secondary visual area.

→ Broca's area and its role in speech:-

Role of Broca area: Speech synthesis

- It is situated adjacent to the motor area, responsible for the movements of tongue, lips and larynx, which are necessary for speech.
- By receiving information required for production of words from Wernicke area, the Broca area develops the pattern of motor activities required to verbalize the words.
- The pattern of motor activities is sent to motor area.
- Role of motor area - Activation of peripheral speech apparatus.
- By receiving the pattern of activities from Broca area, motor area activates the peripheral speech apparatus.
- It results in initiation of movements of tongue, lips and larynx required for speech. Later, when child is taught to read, auditory speech is associated with visual symbols.
- Then, there is an association of auditory & visual areas with the motor area for the muscles of hand.
- Now, the child is able to express auditory & visual impressions in the form of written words.

Broca area is also called speech centre, motor speech area or lower frontal area.

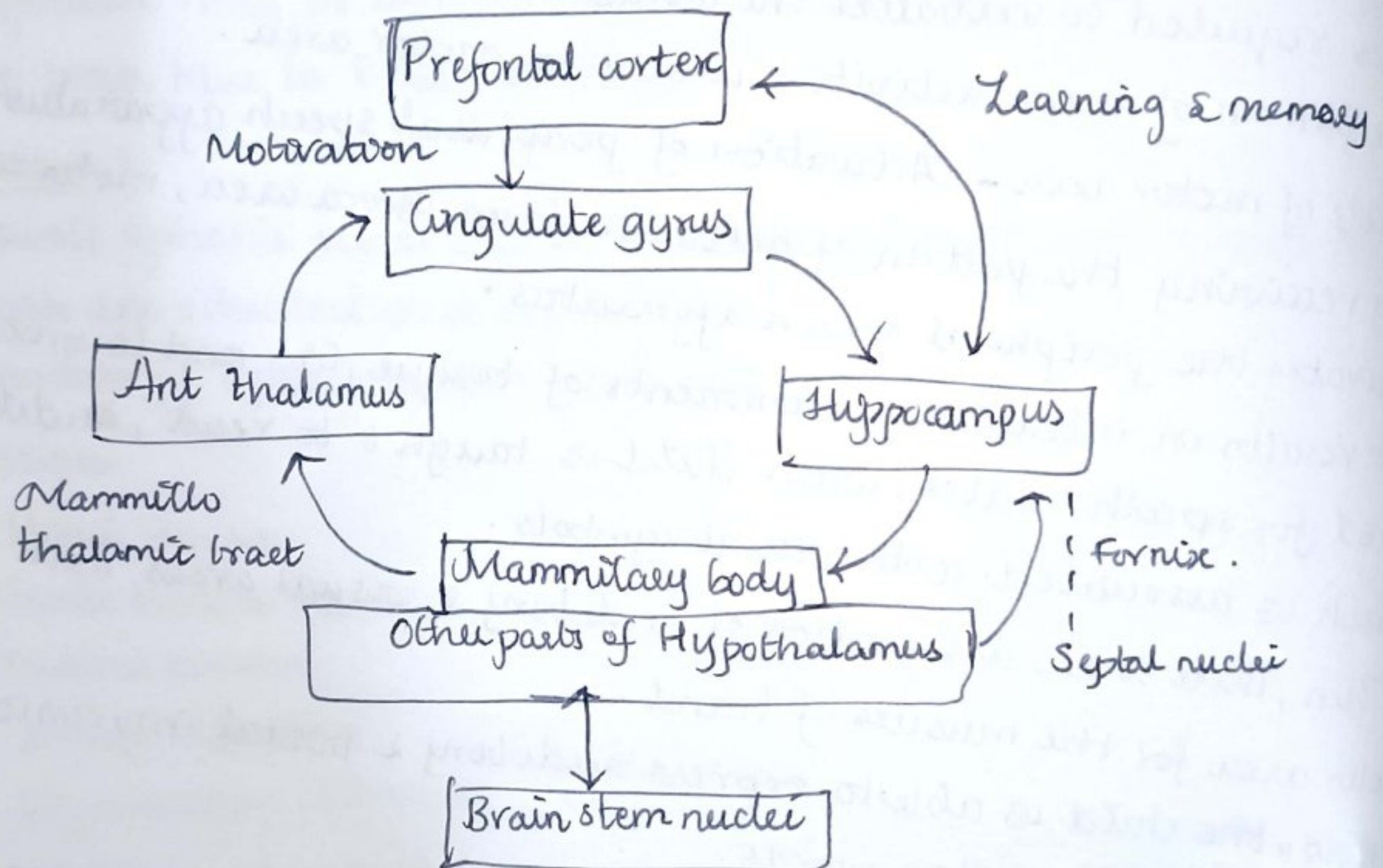
Broca area controls the movements of structures involved in vocalization.

Dissociated Anesthesia:-

- Loss of some sensations while other sensations are intact.
- Dissociative anesthesia is a form of anesthesia characterized by catalepsy, catatonia, analgesia and amnesia.
 - It does not necessarily involve loss of consciousness and thus does not always imply a state of general anesthesia.

⇒ Papez circuit :-

- The limbic system comprises limbic lobe of cortex & subcortical structures associated with it.
- The limbic lobe includes cingulate gyrus, subcallosal and part hippocampal gyri
- The limbic structures are interconnected by circuitous tracts described by Papez and known as Papez circuit.
- Major circuitous tracts of limbic system connect hippocampus, mammillary body of hypothalamus, hypothalamus to ant hypothalamic nuclei, ant thalamus to cingulate gyrus.
- Circuit is completed by cingulate gyrus projecting to hippocampus.



The major circuitous tracts of limbic system connect other structures within the major circuit.

⇒ Amnesia :-

- Amnesia is a neurological condition characterized by a partial or complete loss of memory.
- It can affect a person's ability to remember past events, personal information or even form new memories.
- This condition can be temporary or permanent and may result from various factors, including brain injury stroke, certain medication, psychological trauma or neurological disorders such as Alzheimer's disease.

Types

1. Retrograde Amnesia.
2. Anterograde "
3. Transient Global "
4. Post-Traumatic "

⇒ Reciprocal Innervations :-

- It refers to the contraction of agonist muscle and relaxation of the antagonist muscles in response to the stimulation of afferent nerve.

Circuit :-

Afferent fibres :- Group Ia and II fibres

Centre :- Spinal cord

Efferent fibres :- motor neurons supplying ^{muscle} agonist & antagonist muscle

Response :- contraction of agonist muscle & relaxation of antagonist muscle (relaxation due to stimulation of an inhibitory interneuron).

Importance :-

- Essential for normal physiological movements like walking.
- Useful in eliciting crossed extensor reflex b/w 2 upper limbs.

⇒ Inverse Stretch Reflex :-

- Refers to relaxation of muscle in response to stretch reflex. Also called as lengthening reaction and clasp knife reflex.

Receptors :- Golgi tendon organ.

Afferent fibres :- Group Ib fibres.

Centre :- Corresponding spinal segment

Efferent fibres :- α motor neuron to the corresponding muscle.

Response :- Inhibition of α -motor neuron by the inhibitory interneuron and relaxation of the corresponding muscle.

Functions :-

- Monitors the force generated in muscle.
- Monitors muscle tension and prevents rupture of muscle.
- along with stretch reflex maintains optimal motor responses for postural adjustments.
- reflex is called autogenic inhibition.

- Referred to the withdrawal of body parts by flexion of limbs when a painful stimulus is applied.
- It is a polysynaptic reflex.

Receptors: Nociceptors

Afferent fibres: Type III & IV somatic afferents

Centre: Spinal cord.

Efferent fibres: Somatomotor neuron supplying the flexor muscles same side & extensor muscles of opposite side.

Response:

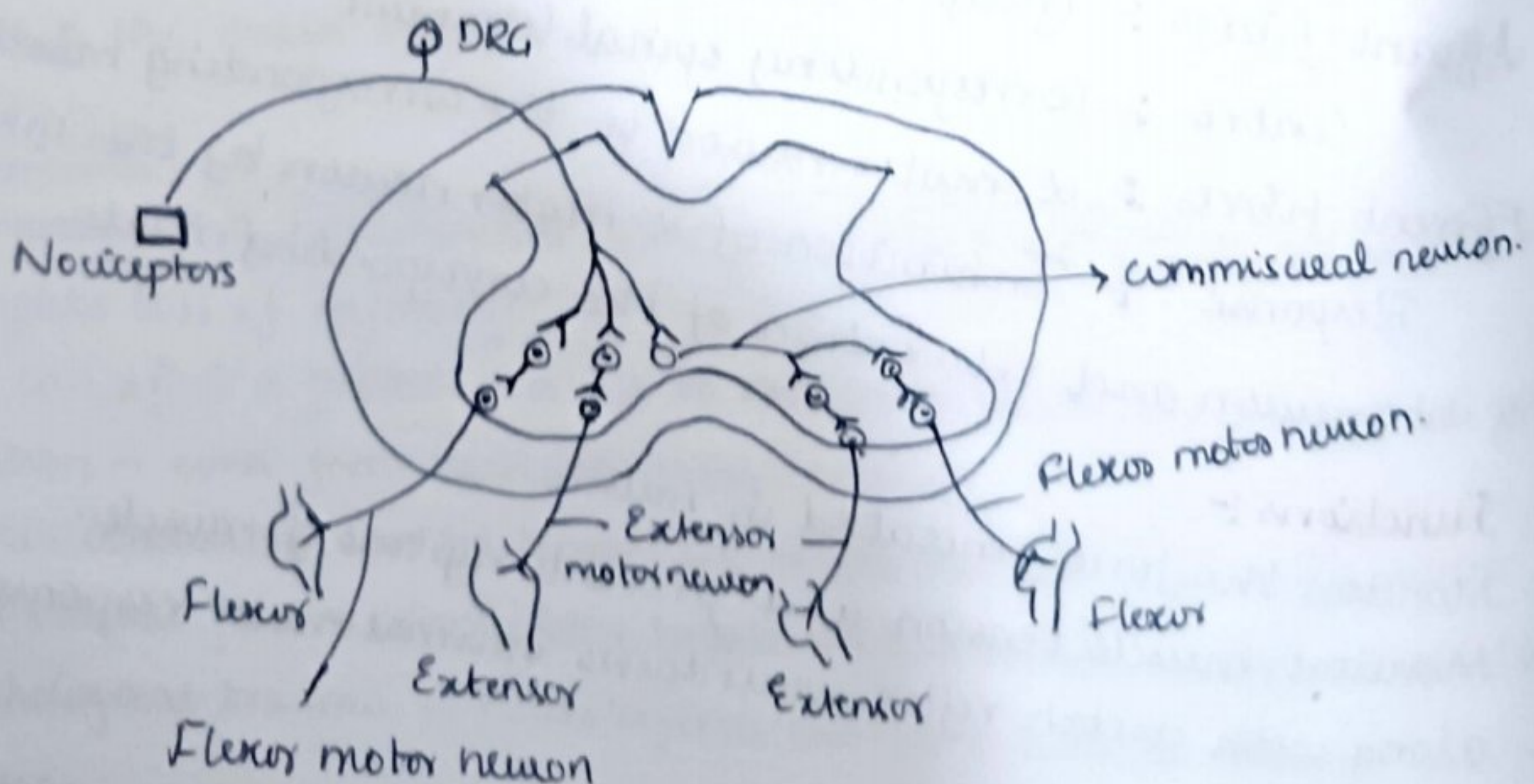
Mild stimulus: flexion of limb on same side and extension of limb of opposite side.

Stronger stimulus: response in all four limbs.

Reason a) Irradiation of impulse b) Recruitment of ^{more} motor units

Special features:-

- Withdraw reflex is a protective reflex (protects the tissue from damage)
- Pre potent (stops all other spinal reflexes temporarily).
- Shows local sign i.e response depends upon the location of the stimulus.
- Stronger stimulus causes wide spread and prolonged response (Causes: After discharge due to involvement of many interneuronal pathways & reverberatory circuits in the spinal cord).



- Along with amygdaloid complex, the feeding centre and a centre present in hypothalamus regulate food intake.

5. Control of Circadian rhythm

Hypothalamus is taking major role in the circadian fluctuation of various physiological activities.

6. Regulation of sexual functions

Hypothalamus is responsible for maintaining sexual function both man and animals.

7. Role in emotional state

Emotional state of human beings is maintained by hippocampus along with hypothalamus.

8. Role in Memory

Hippocampus & Papez circuit play an imp role in memory.

9. Role in motivation

- Reward & punishment centres present in hypothalamus & other structures of limbic system are responsible for motivation & behaviour pattern of human beings.

Babinski sign

- Abnormal plantar reflex is called Babinski sign. It is also called Babinski reflex or phenomenon.
- In normal plantar reflex, a gentle scratch over the outer edge of the sole of the foot causes plantar flexion & adduction of toes.
- But in Babinski sign, there is dorsiflexion of great toe and fanning of other toes.
- When Babinski reflex is present, the condition commonly called Babinski positive sign and when it is negative, the condition is called Babinski negative sign.
- Babinski sign is present in upper motor neuron lesion.
- Physiological conditions when Babinski sign is present are infancy & deep sleep. It is present in infants because of non myelination of pyramidal tracts.

Physiology of Memory

- Memory is defined as retention of learned information and experiences.
- The stored information should be retrieved and utilized at any time in life whenever needed.
- Thus, memory has four stages:
 - 1) Registration of memory that includes proper perception & attention
 - Failure of learning & memory occurs due to impaired perception & attention because the material to be learned is never registered & assimilated
 - 2) Integration & retention :- Learned experiences are processed and integrated by various structures in CNS & then retained at appropriate place in the brain in the form of short term or long term memory
 - 3) Recognition & Recall :- At the appropriate time and place, memory is recalled for proper use.
 - 4) Reutilization :- Memory is utilized for improvement of further learning.

Types of memory :-

- Memory is broadly divided into explicit or declarative memory and implicit or non-declarative memory.
 - 1) Explicit or declarative memory
 - i) memories of events
 - ii) memories of facts.
 - It depends on the hippocampus.
 - a. Short term memory
 - b. Long term memory.
- 2) Implicit or non-declarative memory :- Includes skills, habits, priming & conditioned reflexes.
 - a) Priming
 - b) Procedural memory - skills & habits
 - c) Associative learning - Classical & operant conditioning.
 - d) Non-associative learning - Habituation & Sensitization.

Clinical :

Amnesia :- loss of memory. It is 2 types.

Retrograde - Defect in recall & reproduction of memory.

Anterograde - Information cannot be registered & cannot be retained



- Definition:-
- Reflex response to a stimulus that hardly elicited any response in past, but presently the response to the stimulus is acquired by pairing the stimulus with another stimulus, that normally produces the response is called conditioned response.

Types:-

- There are two types of conditioned reflexes: Classical conditioning and operant conditioning.
- Conditioned reflexes have two components that are associated with emotional responses & motor responses.
- Emotional responses are regulated by amygdala and the motor responses are controlled by cerebellum.

Classical conditioning:- It is the conditioning in which organism learns something through association i.e. Conditioned and Unconditioned stimuli

- It involves associating an involuntary response & stimulus.

Operant Conditioning:- Operant conditioning is the type of learning in which the organism learns by way of modification in behaviour or pattern through reinforcement or punishment.

- It is associating a voluntary behaviour & a consequence.

Anterograde Amnesia:-

- Inability to recall the memory or to form new memories after the event (head injury, mental shock or disease) is called Anterograde amnesia.
- Medial temporal lobe and medial diencephalon of brain are damaged.
- Individual can recall the memories before the onset of amnesia.
- Causes:- Brain damage due to the effects of long term alcoholism, severe malnutrition, stroke, head trauma, anoxia, encephalitis, surgery, cerebrovascular events & Wernicke-Korsakoff syndrome. etc.
- It is difficult to treat with pharmacological methods due to neuronal loss.

- It can be temporary or permanent.

Alzheimer's disease

- Alzheimer's disease is the common degenerative disease of brain characterized mainly by premature & progressive dementia.

Etiology :-

- There is severe loss of cholinergic neurons projecting from basal forebrain to neocortex, amygdala & hippocampus.
- Fibres projecting from nucleus basalis of Meynert r severely affected.
- Pronounced neuronal loss occurs in hippocampus, parahippocampal gyr & entorhinal cortex.

Pathology :-

1. Presence of Neurofibrillary tangles in the nerve cell cytoplasm.
2. Appearance of neuritic / amyloid plaques scattered throughout the cerebral cortex.
3. Granulovacuolar degeneration of neurons.
4. Mitochondrial dysfunction.
5. Autophagy
6. Neuro inflammation & cholinergic insufficiency.

Features :-

- Usually the patient is above 50 yrs.
- Progressive development of forgetfulness is the major symptom.
- Disease starts with loss of short term memory & later followed by loss of other cognitive functions.
- Dysnomia and paranoia.

Treatment :-

- Cerebral vasodilators, stimulants & high dose of vitamins B₁, C, E & have beneficial effects.
- Trials of oral physostigmine, choline, leathin & cholinergic precursor & agonists have some results.

1. Aphasia or Dysphasia :- Loss or impairment of production & prehension of spoken or written language due to an acquired lesion of the brain is called Aphasia.
2. Dysarthria :- Defect in articulation of speech with intact mental function and comprehension of spoken & written language. This occurs purely due to disorder of muscles of articulation, which may be due to flaccid or spastic paralysis.
3. Aphonia :- Loss of voice due to disorder of larynx or its innervation.
4. Stuttering.
5. Apraxia
6. Lisp
7. Selective mutism
8. Tongue tie (ankyloglossia)
9. Articulation errors.

Functions of Prefrontal lobe

- Earlier, this area was considered as inexcitable to electrical stimulation. Hence it was called the silent area or association area.
- But now it is known that stimulation of this area with low voltage electrical stimulus causes changes in the activity of digestive, cardiovascular, respiratory, excretory & other autonomic functions.
- It also causes fear. Various functions of prefrontal cortex are :
 1. It forms the centre for the higher functions like emotion, learning, memory & social behaviour. Short term memories are registered here.
 2. It is the centre for planned actions.
 3. This area is the seat of intelligence ; so it is also called as organ of mind.
 4. It is responsible for the personality of the individuals.
 5. Prefrontal cortex is responsible for the various autonomic changes during emotional conditions, because of its connections with hypo-

Prefrontal Lobotomy Effects:-

- When the connection of thalamus with prefrontal lobe is cut (this is called prefrontal lobotomy), the major areas of prefrontal lobe are disconnected.
- This is associated with frontal lobe syndrome, which is characterized by
 1. Difficulty in planning.
 2. Euphoria & failure to understand the seriousness of others feeling.
 3. Loss of recent memory with impairment of moral & sexual sense.
 4. Emotional instability.
 5. Loss of attention & ability to concentrate.
 6. Decreased mental drive & lack of initiation.

Decerebrate Rigidity

- Decerebrate rigidity is the rigid extension of all the limbs due to decerebration. This type of rigidity is well pronounced in extensor muscles.
- Reason for decerebrate rigidity is the release of centres, situated below the section from higher inhibitory controls.
- The inhibitory area is 45. It is situated in the motor cortex of cerebrum, just anterior to area 4 and behind the area 6.
- In this area, Betz cells are absent. From here, the fibres are projected to spinal cord via reticular formation.
- So in decerebration the discharge from neurons of area 45 cannot reach the spinal motor neurons.
- This leads to exaggeration of spinal motor neurons resulting in rigidity.
- Decerebrate rigidity is also produced by stopping blood flow to the forebrain.
- It is done by occlusion of common carotid artery & the basilar artery at the centre of pons.