Student Notes

PHYSIOLOGY

1st Edition

Unit 5

Central Nervous System

Preface

Dear Medicos,

Agam Medical Organization has been dedicated to fostering a culture of academic excellence and knowledge sharing among medical students. Our organization has always recognized the importance of passing on the torch of learning to the next generation. Today, we are proud to present the culmination of years of dedication and hard work: the Agam Medical Organization's Physiology Study Material.

This comprehensive study material is not just a book; it's a testament to the spirit of collaboration and the unwavering commitment of our fellow students to their academic pursuits. As we navigate the challenging journey of medical education, we understand the significance of having reliable and concise resources at our disposal. This study material has been meticulously crafted with this understanding in mind.

The material within these pages is the result of countless hours of research, study group sessions, and the collective wisdom of our seniors. It has been designed to aid you in comprehending the intricacies of physiology and, more importantly, to empower you to excel in your undergraduate university exams.

Each chapter within this book delves into various aspects of physiology, offering not just theoretical knowledge but also practical insights. We have strived to simplify complex concepts, making them accessible to all readers, regardless of their level of familiarity with the subject.

Furthermore, we have incorporated summaries, and diagrams to facilitate effective revision. We firmly believe that learning should be a dynamic process, and this study material encourages active engagement with the subject matter.

As you embark on your journey through the pages of this book, remember that you are not alone. You have the collective wisdom and support of Agam Medical Organization and its members behind you. We are here to help you succeed.

We extend our heartfelt gratitude to all the contributors, without whom this project would not have been possible. Their dedication and passion for the subject shine through in every page.

We hope that this Physiology Study Material serves as a valuable companion on your academic path, guiding you toward a deeper understanding of the human body and fostering a love for the subject. May it be a beacon of knowledge that illuminates your path to success.

Best wishes,

Agam

Acknowledgment

In the journey of creating the Agam Medical Organization's Physiology Study Material, we have been fortunate to receive support, guidance, and inspiration from numerous sources. We would like to extend our heartfelt gratitude to:

Contributors:

We are deeply appreciative of all the individuals who contributed to this material, whether through research, content creation, or editorial work. Your collective efforts have resulted in a resource that will benefit countless students. A special thanks to **Jwala S**, for leading the team to bring the material in perfect form. We would like to express our deepest appreciation to:

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God:

We begin by expressing our gratitude to the Almighty for bestowing upon us the wisdom, strength, and determination to undertake this endeavour.

Pioneers in the Field of Physiology:

We stand on the shoulders of giants—those pioneering scientists and researchers who paved the way for our understanding of the human body's intricate workings. Their dedication to advancing the field of physiology has been our guiding light.

Doctors and Professors:

To the medical professionals and educators who have tirelessly imparted their knowledge and expertise to generations of students, we owe a debt of gratitude. Your dedication to teaching and healing has inspired us to strive for excellence.

Seniors:

A heartfelt thanks to our senior colleagues who generously shared their wisdom and experiences with us. Your mentorship and guidance have been invaluable in shaping this study material.

Supportive Families and Friends:

Behind every student is a network of family and friends who offer unwavering support. We thank our loved ones for their patience, encouragement, and understanding throughout this journey.

The entire Agam Medical Organization:

To our dedicated members and the organization itself, thank you for providing a platform where students can collaborate, learn, and grow together. Your commitment to knowledge-sharing is the driving force behind this initiative.

Fellow Students:

Last but not least, we extend our gratitude to our fellow students who will use this study material. It is for you that we embarked on this journey, and we hope that this resource empowers you to excel in your academic pursuits.

This study material is a testament to what can be achieved through collective effort and a shared passion for learning. As we move forward, let us remember the importance of giving back and supporting one another in our pursuit of knowledge.

With sincere thanks,

Agam

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5. tectospinal tract
6. rubrospinal tract
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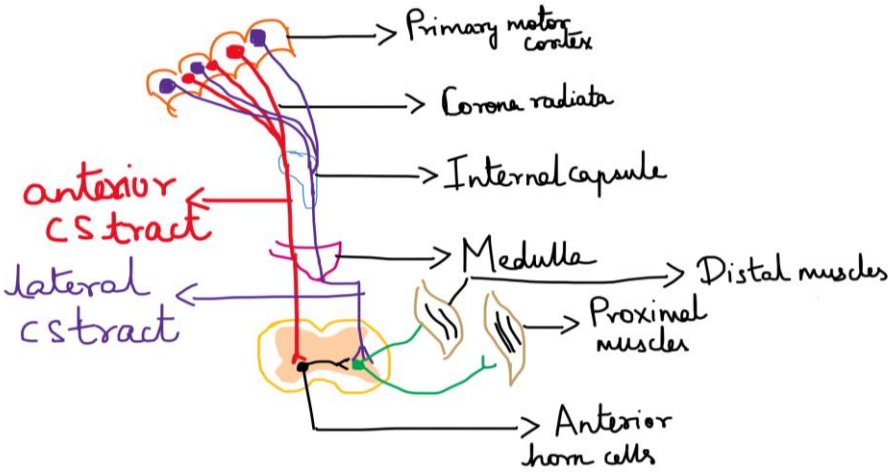


PYRAMIDAL TRACT

- Pyramidal tracts are the descending tracts concerned with voluntary motor activities of the body
- Also known as corticospinal tract.
- There are two corticospinal tracts- the anterior and lateral corticospinal tracts.
- While running from cortex to spinal cord, the fibers give the appearance of pyramid.

ORIGIN:

Fibers of pyramidal tract arise from:

1. Primary motor area (area 4)
2. Premotor area (area 6)
3. Supplementary motor area
4. Somatosensory area

All these fibers form the upper motor neurons of motor pathway.

COURSE:

Corona radiata:

- After origin, the nerve fibers run downwards through the white matter of cerebrum and converge in the form of fan like structure.
- This fan like structure is called corona radiata.
- Corona radiata contains both ascending and descending fibers.

Internal capsule:

- Corona radiata converge in the form of internal capsule while passing through the brain stem.
- It is situated between thalamus and caudate nucleus on medial side and thalamus and lenticular nucleus on lateral side.

Pons:

- The fibers are divided into different bundles at the upper part of pons which are then grouped into a compact bundle at the lower border.

Medulla:

- The fibers give the appearance of pyramid in the medulla region.
- At the lower border of medulla, the fibers are divided into bundles of unequal sizes.
- 80% of fibers cross the opposite site. While crossing, they form pyramidal decussation.

Spinal cord:

- The fibers which crossed the midline are called crossed pyramidal tract or lateral corticospinal tract or indirect corticospinal tract.
- The uncrossed fibers are called uncrossed pyramidal tract or anterior corticospinal tract or direct corticospinal tract.

TERMINATION:

- The fibers terminate both on the alpha and gamma motor neurons.
- Neurons giving origin to the pyramidal tract are called upper motor neurons.
- Alpha and gamma motor neurons are the lower motor neurons.

FUNCTIONS:

- Pyramidal tracts are concerned with voluntary movements of the body.
- Responsible for fine skilled movements.

EFFECTS OF LESION:

- Lesion in the neurons of the motor cortex and pyramidal tract is called upper motor neuron lesion.
- Loss of voluntary movements in the extremities initially, later it involves other parts of the body like hip and shoulder.
- Muscle tone is increased due to spasticity.
- Groups of muscles are affected
- Spastic paralysis of muscles.
- Hypotonia occurs in pure pyramidal tract lesions which is rare because extrapyramidal tracts are also damaged during lesions.
- Superficial reflexes are lost.
- Deep reflexes are exaggerated.
- Abnormal plantar reflex called Babinski sign is present.

EFFECTS OF LESION AT DIFFERENT LEVELS:

Cerebral cortex:

- Hypertonia
- Spasticity
- Contralateral monoplegia (paralysis of one limb) or contralateral hemiplegia.

Internal capsule:

- Contralateral hemiplegia.

Brainstem:

- Contralateral hemiparesis (weakness of muscles in one side of the body)
- Sixth and seventh cranial nerve palsies.

Spinal cord:

- Unilateral lesion of lateral corticospinal fibers at upper cervical segment causes ipsilateral hemiplegia.
- Bilateral lesion of lateral corticospinal fibers at upper cervical segment causes quadriplegia (paralysis of all four limbs) and paralysis of respiratory muscles.
- Bilateral lesion in thoracic and lumbar segment results in paraplegia (paralysis of both lower limbs)

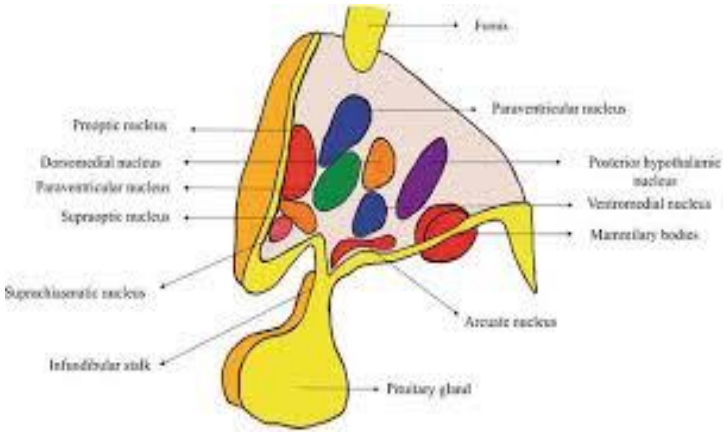
HYPOTHALAMUS

NUCLEI OF HYPOTHALAMUS:

Hypothalamus consists of large number of nuclei and nuclear groups

4 Main Nuclear Groups are

- Anterior group: includes supraoptic, preoptic**,** paraventricular nuclei (spp)
- Middle group: Tuberal, ventromedial, arcuate, dorsomedial nuclei (TV ad)
- Posterior group: Supramammilary, mammilary, posterior hypothalamic nuclei (psm)
- Lateral group: lateral preoptic area, lateral hypothalamic nuclei



Connections of hypothalamus:

- Through fornix it is connected to the limbic system
- Through the median forebrain bundle it is connected to the brainstem
- Through periventricular system it is connected to the sensory pathways and midbrain
- Through the mamillothalamic tract it is connected to the anterior nucleus of thalamus
- Retinohypothalamic fibers connect the retina to the suprachiasmatic nucleus
- Connected to the tegmental nucleus through the mamillotegmental tract
- The hypothalamohypophyseal tract connects the supraoptic and paraventricular nuclei to the posterior pituitary
- Arcuate and venteromedian nuclei are connected to the infundibulum via the tuberoinfundibular tract
- Locus ceruleus and dorsal hypothalamus are connected by the dorsal noradrenergic pathway
- Serotonergic pathway connects raphe nucleus to hypothalamus
- Mesolimbic dopaminergic system connects the hypothalamus to the third ventricle
- Corticohypothalamic fibers connect the cerebral cortex to the hypothalamus

FUNCTIONS OF HYPOTHALAMUS:

1. Endocrine Functions
2. Autonomic Functions
3. Temperature Regulation
4. Circadian rhythm
5. Regulation of food
6. Regulation of water intake
7. Reproductive Functions
8. Immunological Functions
9. Influence on emotions
10. Role in sleep

Endocrine Functions

- Controls anterior and posterior pituitary functions.
- Connected with anterior pituitary via portal hypophyseal vessels and posterior pituitary through hypothalamohypophyseal tract.
- **Control of anterior pituitary function**

  By secreting various releasing and inhibiting hormones

The main releasing and inhibiting hormones are

- Growth hormone releasing hormones (GRH) - controls GH secretion
- Somatostatin- inhibits GH, TSH, prolactin secretion
- TRH - stimulate TSH secretion
- Corticotropin releasing hormones (CRH)- regulate ACTH secretion

Control of posterior pituitary functions

Neurons of supraoptic and paraventricular nuclei of Hypothalamus secretes 2 hormones:

- Antidiuretic hormone
- oxytocin

They are stored and released from posterior pituitary.

Autonomic functions

- **Sympathetic control**

Hypothalamus has profound influence on sympathetic functions

Stimulation of lateral area results in rise in BP, heart rate, sweating, pupillary dilation

Stimulation of posterior hypothalamus results in activation of emotional behavior pattern

- **Parasympathetic control:**

Stimulation of anterior hypothalamus results in parasympathetic response

Temperature regulation

- Depends on balance between mechanism that controls heat loss and heat gain
- Anterior hypothalamus- activates mechanism that promotes heat loss
- Posterior hypothalamus- activates the mechanism that increases heat production and causes heat gain

Circadian rhythm

- Means 24hrs fluctuations in body function i;e. day- night variation
- Most of these circadian fans are regulated by hypothalamus
- Suprachiasmatic nucleus (SCN)- regulate and maintain circadian rhythm
- SCN aka biological clock - its accuracy of execution is achieved through retinohypothalamic fibres that convey the retinal information of light and darkness via optic chiasma to SCN
- Nocturnal secretion of melatonin
- Provide important hormonal signal for other functions

Regulation of food intake

By maintaining balance between intake and energy expenditure hence body weight is maintained in normal range

Neural factors

- Feeding center - lateral hypothalamus

Satiety center- ventromedial hypothalamus

Hormonal factors

- Hormones that increase food intake- Neuropeptide Y, orexins, gherkin, MCH, GHRH
- Hormones that decrease food intake- estrogen, dopamine, peptide YY, leptin, CRH, gut hormones, CCK

Metabolic factors

- Plasma glucose
- Malonyl CoA
- Amino acid and fatty acids
- Body temperature

Regulation of water intake

- Change in osmolality
- Change in fluid volume

Reproductive functions

Hypothalamus secretes Gonadotropin releasing hormone-crucial role

Immunological functions

Influence immunity by controlling the secretion of ACTH, cortisol via hypothalamo pituitary adrenal axis

Influence on emotions

Hypothalamus forms one of the output pathways of limbic system (principal seat of emotions)

Role in sleep

- Diencephalic sleep zone (major part of posterior hypothalamus) induces slow wave sleep
- Preoptic area also induces sleep.

PAIN

DEFINITION:

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage

PAIN PATHWAYS

Pain is transmitted to the higher centers in the brain in the lateral spinothalamic tract of the anterolateral system. The pain pathways are divided into two types:

1. Paleospinothalamic pathway
2. Neospinothalamic pathway

Paleospinothalamic Pathway

This pathway mainly carries the sensation of slow pain. The fibers are mostly C fibers.

First Order Neurons: enter the spinal cord and terminate mainly in the laminae II of the dorsal horn.
Second Order Neurons: decussate and ascend up in the contralateral spinothalamic pathway. In the brain-stem on their way to thalamus, fibers project to three major nuclear groups forming three subsystems:
1. At the level of medulla: spinoreticulothalamic pathway.

2. Fibers also project heavily to the midbrain nuclei: (spino-mesencephalic fibers)

3.Fibers also project to hypothalamus forming spino-hypothalamic fiber system.

Third Order Neurons
In the thalamus, the fibers terminate mainly in the medial nuclear group (the midline and intralaminar nuclei; also called as non-specific nuclei) from where the third order of neurons arise and project to different areas of the cortex including limbic cortical areas.

Neospinothalamic Pathway:

This pathway carries mainly the fast pain. The fibers are mostly Aδ fibers.

First Order Neurons: terminate mainly in the lamina I and V in the dorsal horn of the spinal cord. The neurotransmitters released at the terminals of primary nociceptive afferents (1st order of neurons) are glutamate and neuropeptides (substance P).

Second Order Neurons: cross over to the opposite side in the same segment of the spinal cord and ascend in the lateral spinothalamic tract. In the spinal cord - topographic organization of

fibers: The fibers from lower body parts are placed laterally and fibers from upper body parts are located more medially in the lateral fasciculus.

Third Order neurons: originate from specific thalamic nuclei and project to the postcentral gyrus. The topographic organization of these fibers in the thalamus and cortex is very concrete, and in the sensory cortex, the neurons are organized in modality specific columns. Therefore, the fast pain is better localized.

Types of Nociceptors

Receptors for pain are called nociceptors. Nociceptors are the free nerve endings. They are distributed widely throughout most parts of the body. However, there are few tissues like brain (only the neural tissue of the brain) that are devoid of nociceptors.

The nociceptors are broadly divided into three categories:

1. Aδ mechanical nociceptors
2. Multimodal C fiber nociceptors
3. Other nociceptors.

Aδ Mechanical Nociceptors:

These are the terminals of Aδ fibers. The Aδ fibers are small myelinated axons that discharge only in response to intense mechanical stimuli (but, not to thermal or chemical stimuli). Aδ fibers conduct the fast pain.

Polymodal C Fiber Nociceptors:

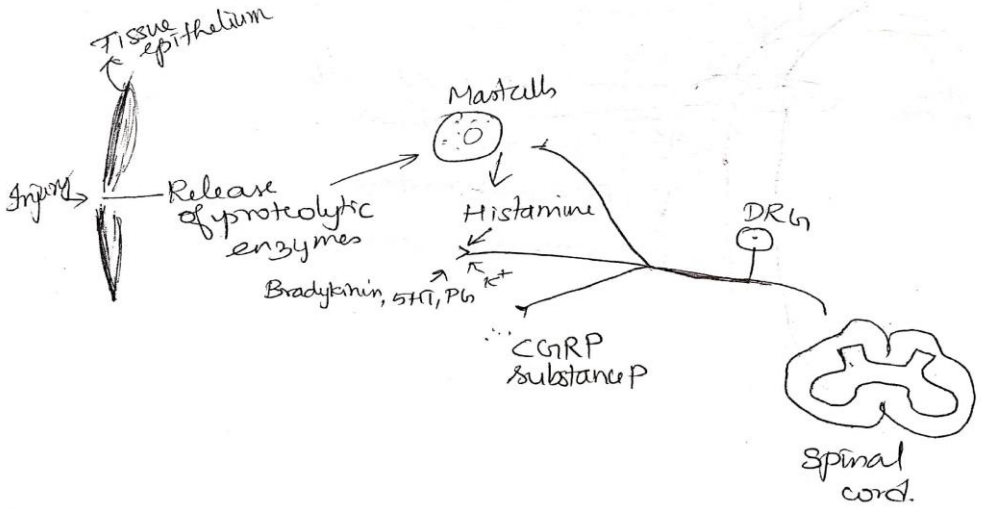
Activated by high-intensity mechanical, chemical and thermal (both hot and cold) stimuli. These are terminals of C fibers. The C fibers carry the slow pain.

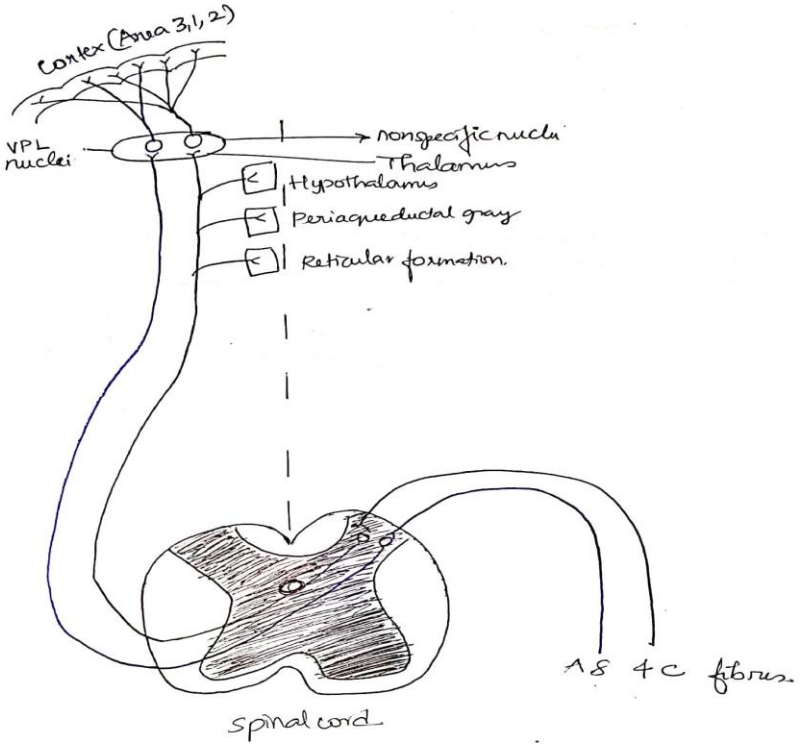
Other Nociceptors: These include thermal nociceptors (Aδ and C fiber terminals that respond to very low and high temperature, i. e. < 5°C and > 45°C respectively), Aδ fibers responding to heat and non-multimodal C fibers responding to strong mechanical stimuli:

Vanilloid Receptors: Vanilloid receptor–1 (VR–1) at C fiber terminals - they respond to vanillin, a group of pain producing compounds that include capsaicin and also responds to increase in temperature > 43°C, and change in pH.

Local anesthetics and many centrally acting analgesics act by raising the pain threshold.

Mechanism of pain recognition





Paleospinothalamic & Neospinothalamic pain pathways.

ASCENDING TRACTS OF SPINAL CORD

Tracts which carry sensory information from external and internal environment to higher center

Various ascending tracts

1.Dorsal columns or posterior column

A.Tract of gracilis

B.Tract of cuneatus

2.Spinothalamic tracts

A.Lateral spinothalamic tract

B.Anterior or ventral spinothalamic tract

3.Spinocerebellar tract

A.Dorsal spinocerebellar tract

B.Ventral spinocerebellar tract

4.Spinotectal tract

5.Spino-olivary tract

6.Spinovestibular tract

7.Spinopontine tract

8.Spinoreticular tract

9.Comma tract

Dorsal column pathway

Other names

- Medial leminiscal pathway
- Tract of Goll and Burdach
- Posterior column
- Fasciculus of gracilis and cuneatus

Sensations carried

- Fine touch
- Pressure
- Vibration
- Proprioception

- Tactile localization
- Tactile discrimination
- Graphesthesia
- Stereognosis

First order Neuron

Peripheral nerves carrying sensations from various mechanoreceptors and proprioceptors

Enter the spinal cord through dorsal root (Bell Magendie law)

Cell bodies in the DRG; Axons after entering the spinal cord immediately ascends up in the posterior white column of same side

Ascend up and end in the Nucleus gracilis and Nucleus cuneatus in the medulla

- Fibres carrying sensation from the lower limb and trunk occupy the medial side (Fasciculus gracilis or Tract of Goll);
- Fibres carrying sensation from the upper limb occupy the lateral side (Fasciculus cuneatus or Tract of Burdach)

Second order Neuron

Second order neurons arise from nucleus gracilis and nucleus cuneatus

Most of the fibres arising from the second order neuron crosses to the opposite side and form the internal arcuate fibres

Internal arcuate fibres decussate with opposite side internal arcuate fibre and form the Great sensory decussation at the lower level of medulla

After the decussation it is also called as medial lemniscal pathway

It ascends up and synapse in thalamus (VPL Nucleus)

*Few external arcuate fibres which take origin from accessory cuneatus and ascend up in the same side carrying unconscious tactile and proprioceptive sensations to cerebellum

Third order Neuron

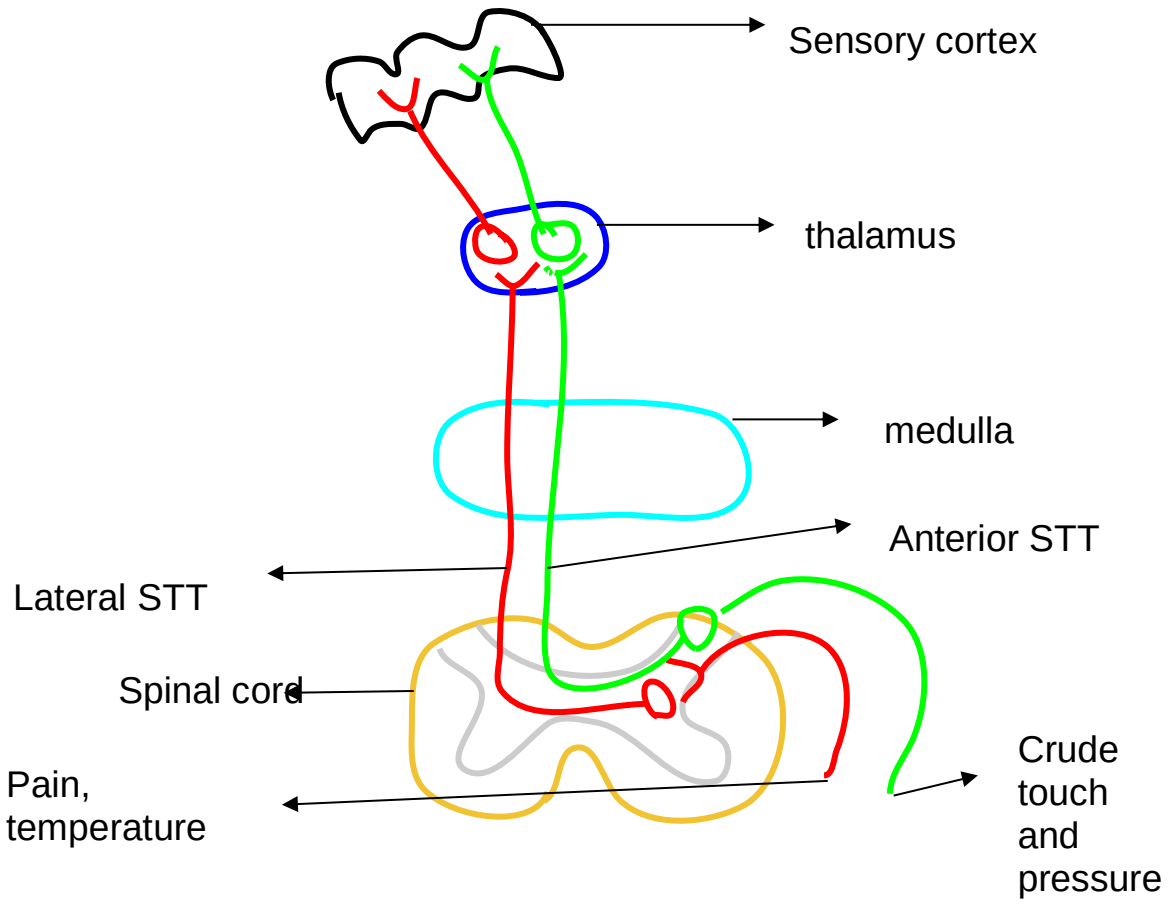
Third order neurons are present in the VPL nucleus of thalamus

Axons ascend through the internal capsule and reach the somatosensory area I (3,1,2) and somatosensory area II

Some fibres also reach hypothalamus, limbic system and reticular formation

Diseases of posterior column:

- Subacute combined degeneration of spinal cord (B12 def)
- NeuroSyphilis –Tabes dorsalis
- Multiple sclerosis
- Gullian –barre syndrome
- Brown –Sequard syndrome



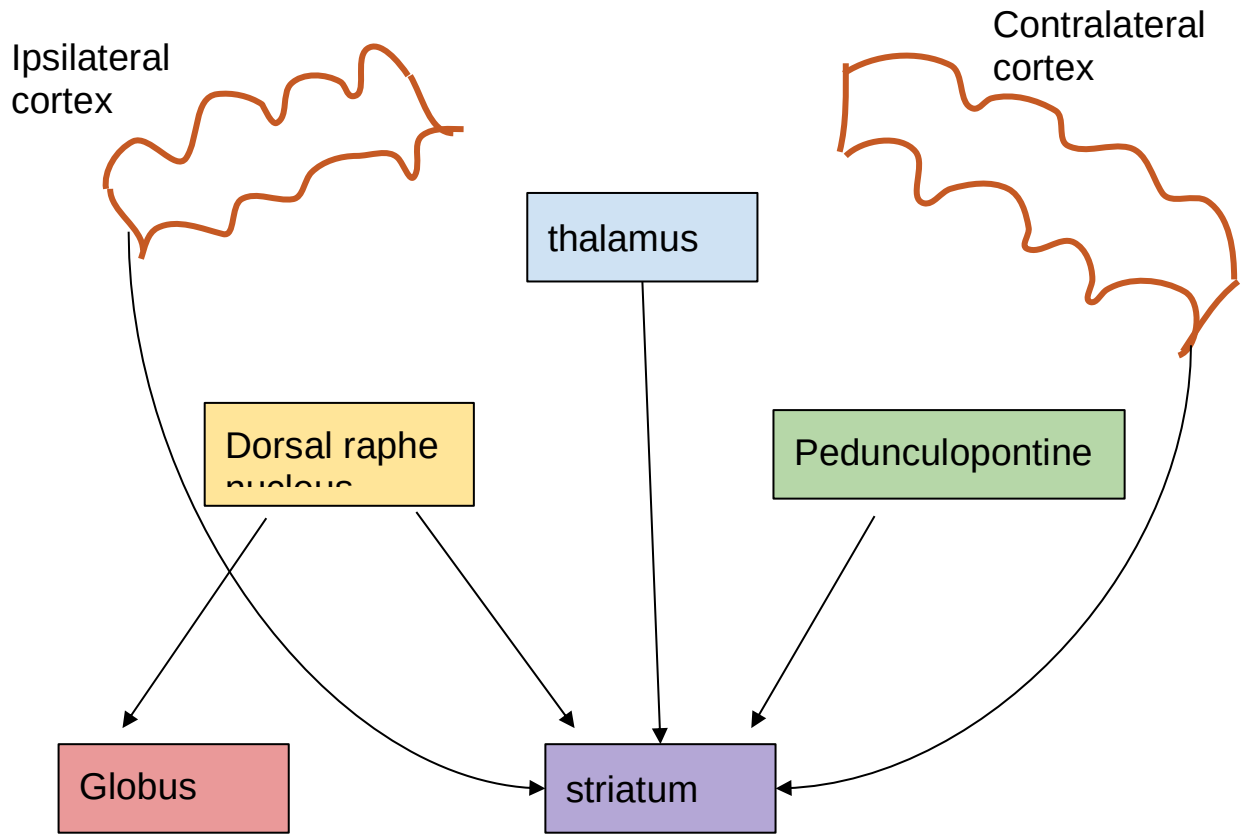
BASAL GANGLIA

They are a group of deep subcortical nuclei located at the base of the forebrain that are primarily involved in the control of posture and movement.

Anatomical organization:

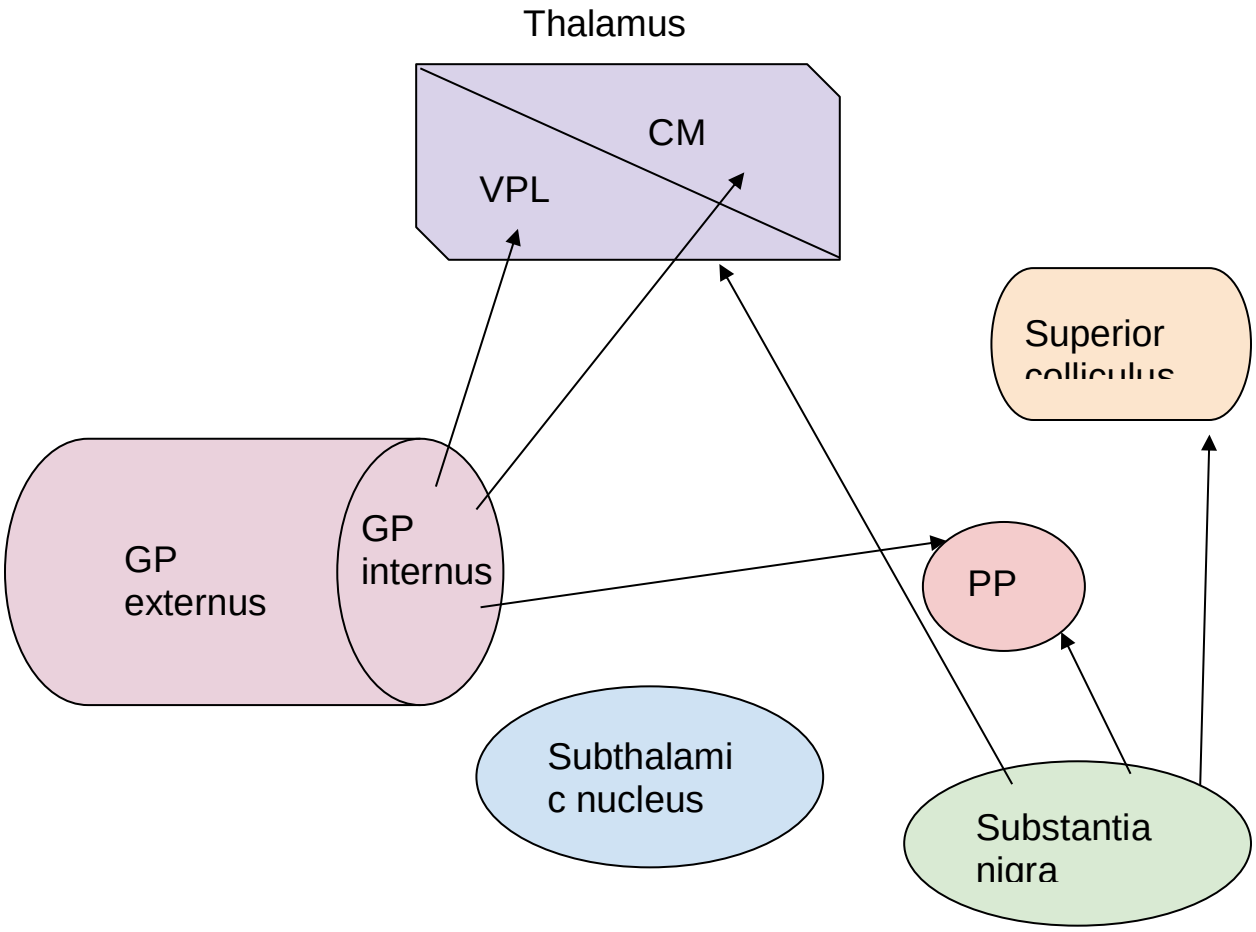
- Caudate nucleus
- Putamen
- Globus pallidus: divided into internus and externus
- Subthalamic nucleus
- Substantia nigra

Inputs to the basal ganglia:



- The main inputs come from the cerebral cortex
- Most afferent information enter the basal ganglia through the striatum
- The afferent fibers are corticostriate projection, thalamostriate projection, raphestriate projection and pedunculostriate projection.

Outputs of the basal ganglia:



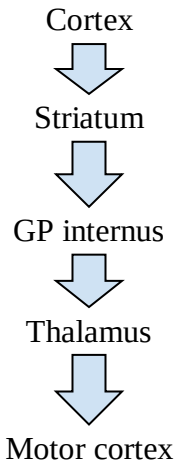
- The principal outputs of the basal ganglia is from the GP internus
- The fibers mainly project into the ventral and centeromedian nuclei of the thalamus that further projects into the cortices
- The output from the GP to thalamus is inhibitory, the output from thalamus to cortex is excitatory.
- The main feature of input and output of basal ganglia is that the cerebral cortex projects to the striatum, striatum projects to the GP, GP projects to the thalamus and the thalamus projects back to the cortex that completes the motor loop.

Connections within the basal ganglia:

- **Nigrostriatal projection:** dopaminergic in nature, projects from the substantia nigra to the striatum
- **Striatonigral pathway:** GABA-ergic in nature, projects from the striatum to the substantia nigra.

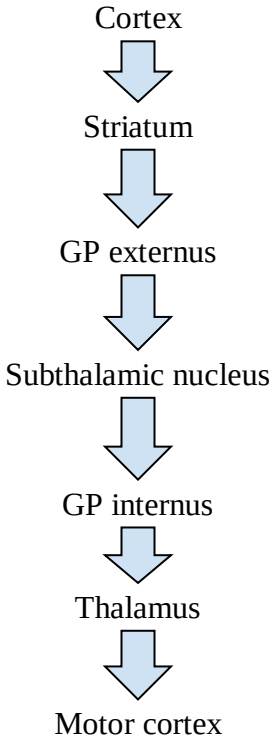
Neural pathways through basal ganglia:

Direct pathways:



- The projection from cortex to striatum is excitatory, the further projections are inhibitory
- Stimulation of the striatum results in stimulation of thalamus by disinhibition

Indirect pathway:



- Striatum inhibits globus pallidus externus that further inhibits subthalamic nucleus
- Subthalamic nucleus activates globus pallidus internus
- Stimulation of striatum activates the GP internus and the final pathway is inhibitory.

Functions of basal ganglia:

- Involved in planning and programming of movements
- Controls posture
- Inhibits stretch reflex by stimulation of caudate nucleus
- Regulation of subconscious gross movements
- Role in cognitive functions
- Skilled movement regulation

Applied aspect:

Parkinson's disease:

A disease of the old age

Characterized by the degeneration of the nigrostriatal pathway that leads to excessive loss of dopamine and dopamine receptors

Features:

- Akinesia
- Bradykinesia
- Mask face
- Rigidity (cogwheel and lead pipe rigidity)
- Resting tremors
- Festinant gait

Treatment: anticholinergics, dopamine agonists, dopamine precursors

Huntington's chorea:

Autosomal dominant type that occurs due to a defective gene on chromosome 4

Caused by the degeneration of the striatonigral pathway

Features:

- Chorea: rapid and involuntary movements
- Dementia

Treatment: no definitive treatment.

CEREBELLUM

- It is also called as little brain
- It is responsible for the integration and regulation of motor function

- It determines the rate, range, force and direction to the termination of movement

Cerebellar organisation:

Located in the posterior cranial fossa behind the brainstem
Connected to midbrain through superior cerebellar peduncle
Connected to the pons through middle cerebellar peduncle
Connected to the medulla through inferior cerebellar peduncle

Functional divisions of cerebellum:

Vestibulocerebellum:

- It is the oldest part, also called as archicerebellum
- Consists of flocculonodular lobe
- Involved in equilibrium and learning induced changes in the vestibulo-ocular reflex

Spinocerebellum:

- Also called as palaeocerebellum
- Consists of the vermis and the paravermal regions
- Also called as spinocerebellum as it receives inputs from the spinal cord
- It is involved in smooth and coordinated voluntary movements.
- Vermal portion projects into the brainstem area that control posture
- Paravermal region controls skilled voluntary movements

Cerebrocerebellum:

- Also called as neocerebellum, newest phylogenetically
- Projects into the cortex
- Involved in planning and programming of movements.

Cerebellar connections:

- Vestibulocerebellar tract: receives inputs from the vestibular apparatus and from vestibular nuclei
- Dorsal spinocerebellar tract: receives inputs from spinal cord
- Cuneocerebellar tract: originates from lateral cuneate nucleus, provides inputs from head and neck
- Tectocerebellar tract: conveys visual and auditory input to the cerebellum
- Pontocerebellar tract: motor cortex inputs
- Olivocerebellar tract: proprioceptive inputs that reach from the whole body through the inferior olive.

Mode of inputs:

- Mossy fibers: proprioceptive inputs
- Climbing fibers: project to purkinje cells

Mode of output:

- Vestibulospinal tract
- Reticulospinal tract
- Rubrospinal tract
- Corticospinal tract

Internal connections of the cerebellum:

- There are 2 sources of input: mossy fibers and climbing fibers
- Purkinje cells are directly stimulated directly by the climbing fiber input
- Purkinje cells are indirectly stimulated by the mossy fibers via granule cell parallel fiber pathways
- Basket and stellate cells are activated by mossy fibers and finally inhibit purkinje cells. This is an example of feed forward inhibition.
- Granule cells also stimulate golgi cells which in turn inhibit the activity of granule cells, this is an example of feedback inhibition.

Functions of cerebellum:

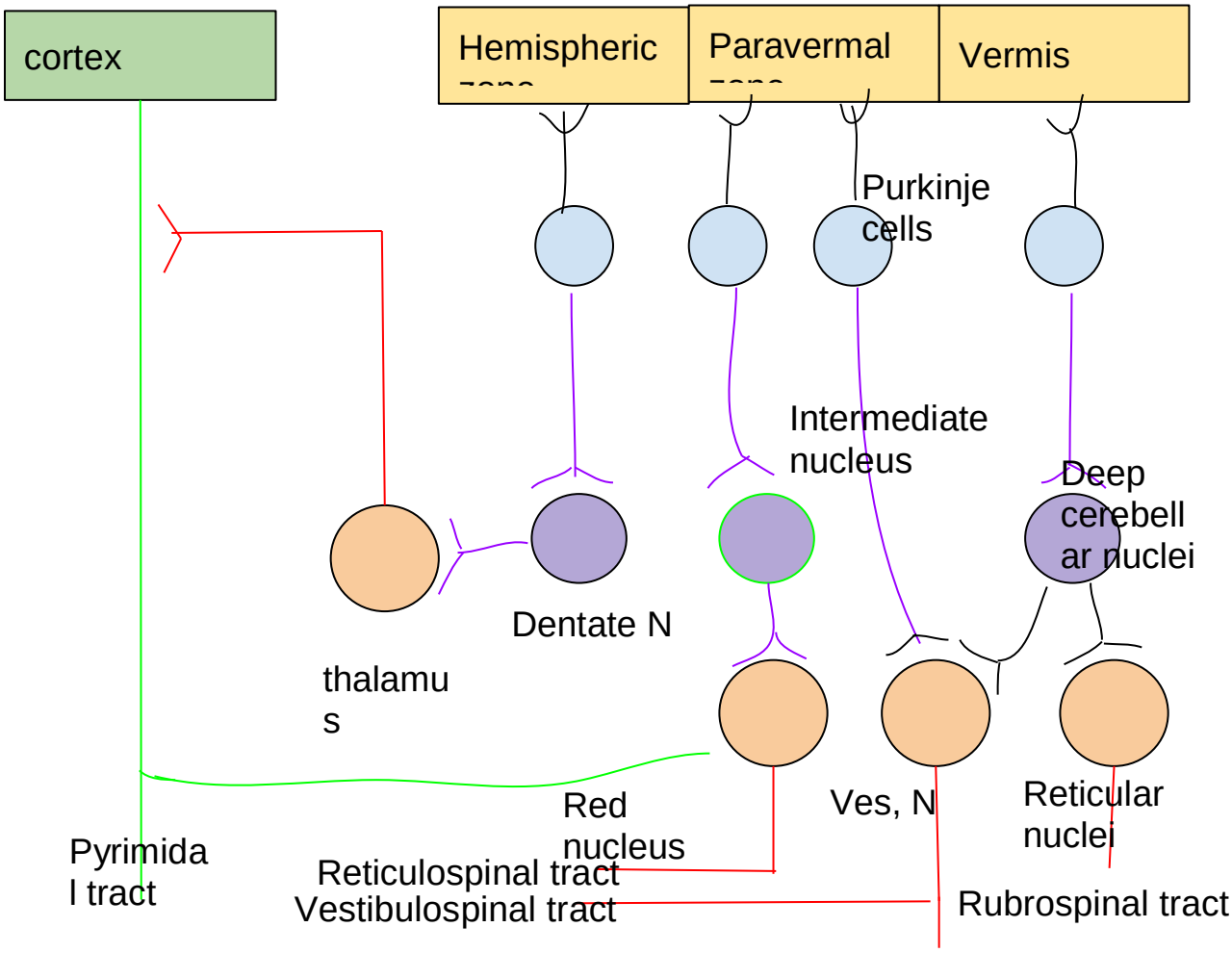
- Control of posture and equilibrium
- Vestibulo-ocular reflex
- Smoothening and coordination of movement
- Control of skilled voluntary movements
- Planning and programming of movements
- Control of muscle tone and stretch reflex
- Control of movement of one side of the body
- Learning and improvement of motor skills
- Eyeball movement
- Vestibular functions.

Cerebellar disorders:

No paralysis, normal reflexes

- Pendular knee jerk
- No sensory deficit
- Hypotonia is present
- Ataxia: defect in coordination due to errors in rate, range, force and direction of movement
- Drunken gait, scanning speech, intention tremor, dysmetria, rebound phenomenon, adiadochokinesia, decomposition of movement.
- Pathological nystagmus

- Charcot's triad: nystagmus, intention tremor, scanning of speech
- Friedrich's ataxia: hereditary ataxia because of the degeneration of spinocerebellar tract.



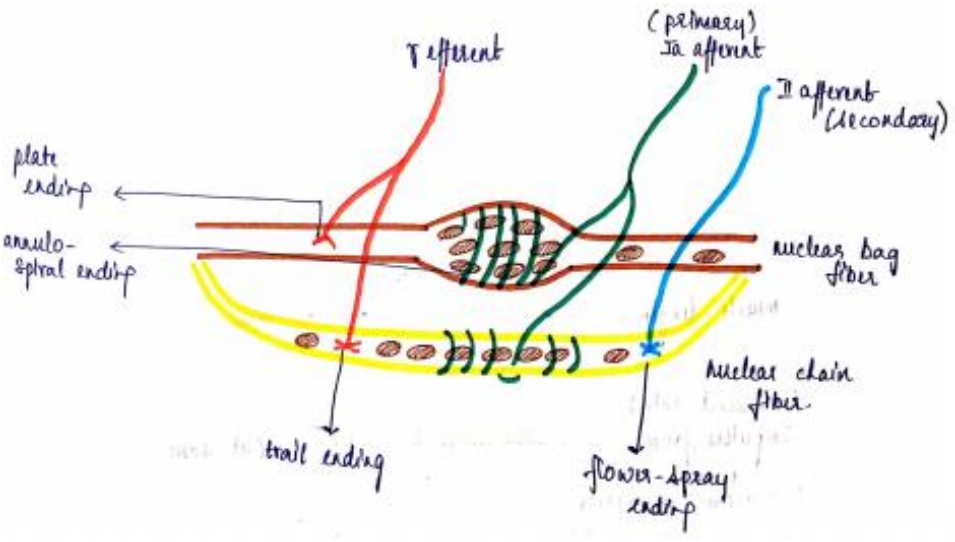
MUSCLE SPINDLE AND GOLGI TENDON ORGAN

Definition -

- Muscle spindles are the receptors that respond to change in muscle length and the velocity of lengthening.
- Present in all skeletal muscles.
- Fibers of the muscle spindle are known as **intrafusal fibers** as they are present inside the fusiform capsules.
- They lie parallel to the extrafusal fibres (extrafusal fibres are the fibres involved in muscle contraction)

Structure -

1. Intrafusal fibers are of **two types**: the nuclear bag fibers, and the nuclear chain fibers.
2. Named so because of their **spindle/fusiform shape**.



- **Innervation of Muscle Spindle** The muscle spindles have both afferent (sensory) and efferent (motor) innervations.

  ✓ **Afferent Innervation** Sensory Fibers are Ia and II fibers.
  There are two types of sensory endings in each muscle spindle: the primary endings, and the secondary endings.

Primary Endings	Secondary Endings
terminals of type **Ia** afferent fiber	terminals of type **II** afferent fiber
also called **annulospiral endings** as they are coiled spirally around the center of the intrafusal fibers	also called **flower-spray endings**, as they appear like flowers.

✓ **Efferent Innervation (Motor Fibers)**
✓ The spindles are innervated by the **γ motor neurons**. They are also called **fusimotor fibers,** constituting 30% (70% of fibers are α motor neurons).

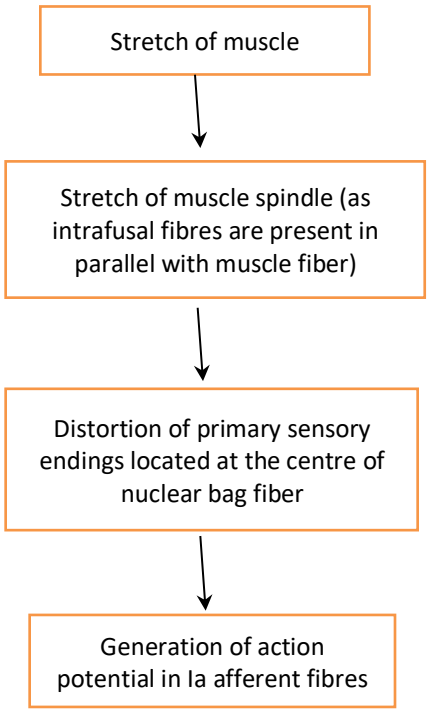
✓ The γ motor neurons are of **two types**: the dynamic γ motor neurons and the static γ motor neurons.

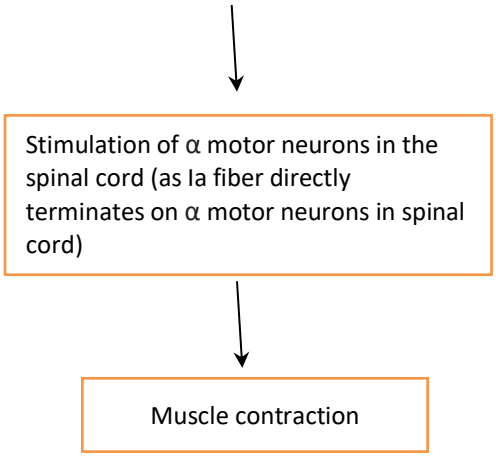
Dynamic γ motor neurons	Static γ motor neurons
Terminate on the nuclear bag fiber 1	Terminate on the nuclear bag fiber 2 and nuclear chain fiber
Stimulation of dynamic γ motor neurons increases response in type Ia afferent fibers (only during the dynamic phase of muscle stretch; i.e. during change in muscle length)	**Stimulation of static γ motor neurons** increases response in type II afferent fibers (only during the static phase of muscle stretch; i.e. the maintained stretch

Functions of Muscle Spindle

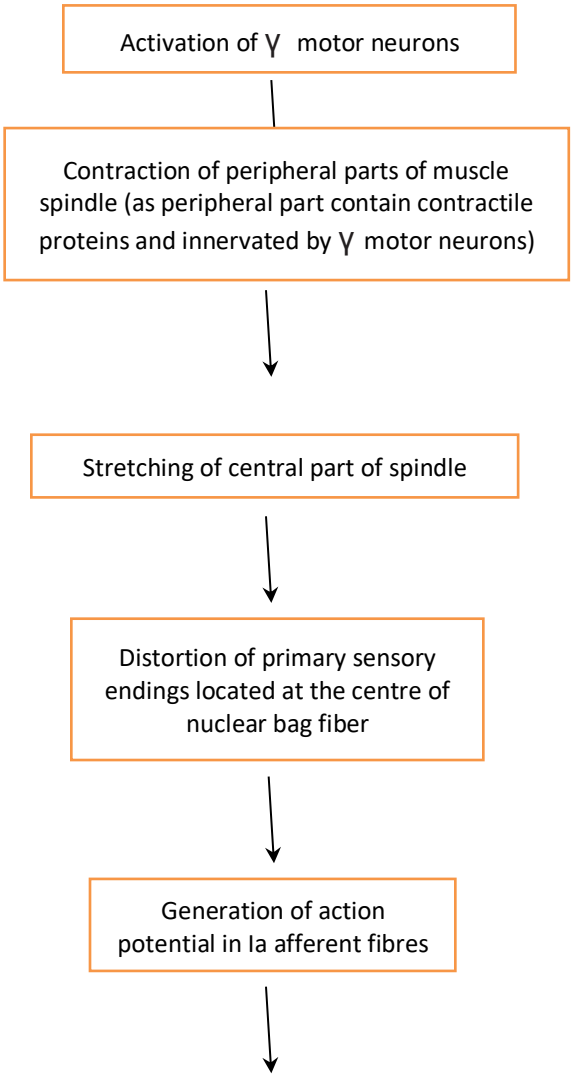
- Muscle spindles are receptors that are sensitive to stretch.
- Spindle is stretched by the stretch of the muscle, as the spindle is present in parallel with the extrafusal fibers (actual contractile fibres).
- **Thus, stretch of the muscle causes muscle contraction by stretching the muscle spindle whereas contraction of the muscle causes muscle relaxation by inhibiting the spindle.**

MECHANISM OF ACTION IN RESPONSE TO MUSCLE SPINDLE





- **MECHANISM OF MUSCLE CONTRACTION IN RESPONSE TO γ MOTOR NEURON STIMULATION**



Stimulation of α motor neurons in the spinal cord (as Ia fiber directly terminates on α motor neurons in spinal cord)

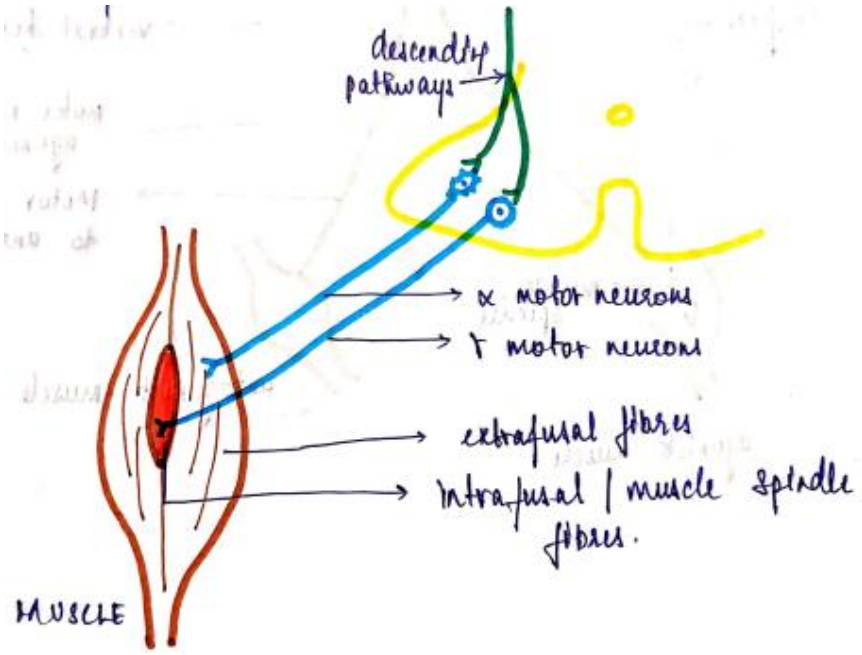
↓

Muscle contraction

- STATIC AND DYNAMIC RESPONSES

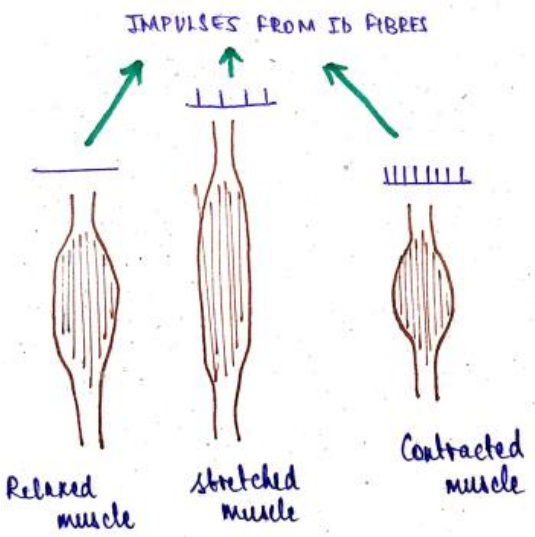
Static response	Dynamic response
Stimulus - Muscle stretch (change in muscle length at maintained stretch)	Stimulus - Rate at which muscle is stretched (velocity of muscle lengthening)
Nuclear chain fiber involved	Nuclear bag fiber involved
Impulse rate is the same throughout stretch	Impulse rate is increased i.e., dynamic

- α–γ coactivation - The descending pathways that stimulate the γ motor neurons also stimulate the α motor neurons.
  1. Therefore, γ efferent discharge increases along with the increased discharge of α motor neurons. This is called a-γ colinkage.
  2. Thus, **spindle adjusts motor neurons discharge throughout the period of muscle contraction**.



- GOLGI TENDON ORGAN

1. Location - Golgi tendon organs (GTO) are **found in the tendon** of the muscles.
2. Structure The GTOs are formed by the terminals of the group Ib afferent fibers. The Ib fibers from GTO terminate indirectly on a motor neurons via interneurons.
   As the interneurons are inhibitory, stimulation of Ib fibers inhibit the motor neuron activity.
3. Function - Because of their arrangement (in series) with the muscle, GTO can be activated either by muscle stretch or by contraction of the muscle.
4. Passive stretch does not effectively stimulate GTO. But, the afferents from GTO discharge actively in response to muscle contraction as muscle contraction stretches the tendon to a greater extent.

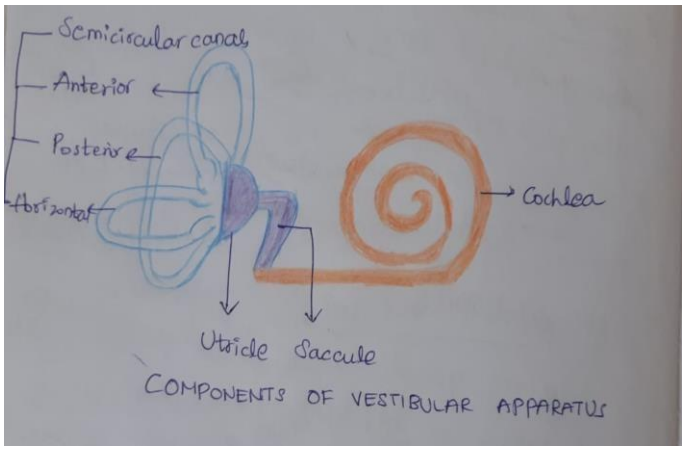


5.. The actual stimulus for activation of GTO is **the force that develops in the tendon** by muscle contraction or muscle stretch.
Therefore, GTO provides the force feedback whereas muscle spindle provides the length feedback

VESTIBULAR APPARATUS

Ear is popularly known for its sensory function of audition, but equally important for its non -auditory function like maintenance of equilibrium at rest and balance during movement.

FUNCTIONAL ANATOMY:

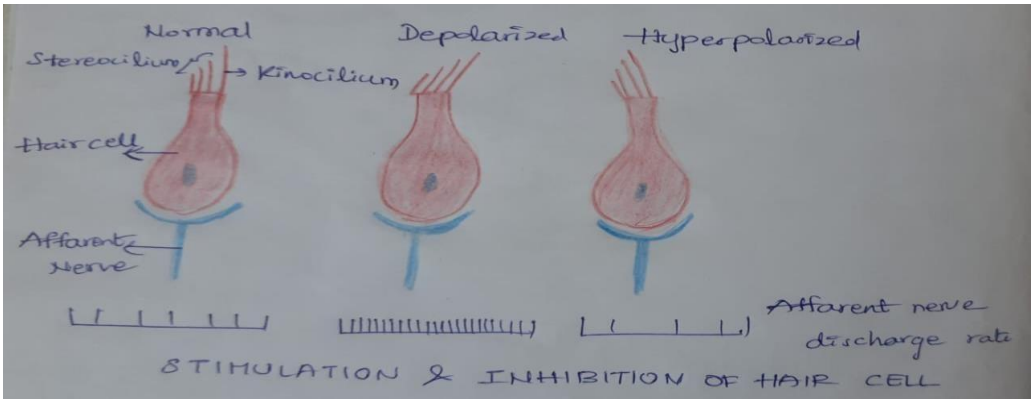


- Vestibular apparatus also known as membranous labyrinth is enclosed in bony labyrinth
- Membranous labyrinth-contains endolymph rich in potassium similar to intracellular fluid.
- Space between membranous labyrinth and bony labyrinth-resembles ECF.

Vestibular apparatus consists of:

- Otolith organ-Saccule and Utricle
- 3 semicircular canals
- Hair cells are the receptors in the vestibular apparatus

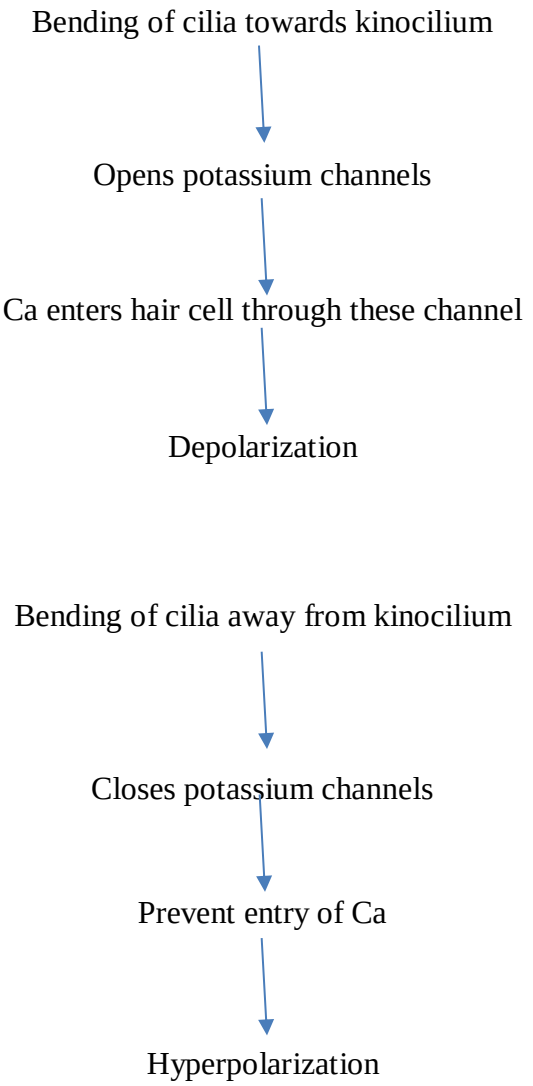
HAIR CELLS:



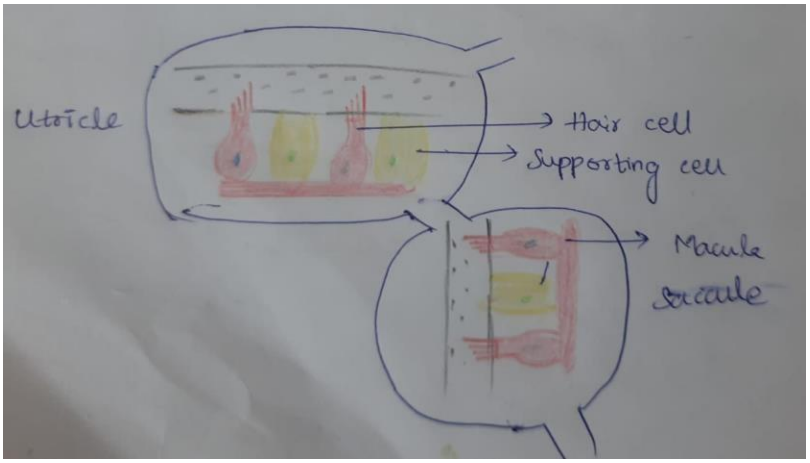
- Large number of cilia arranged according to length
- Kinocilium-longest cilium.
- Stereocilium-other cilia arranged in graded length.

Hair cells have directional sensitivity

Hair cell activity conveyed through eighth cranial nerve.



OTOLITH ORGANS:



- Receptors-Hair cells located in macula.
- Macular hair cells are covered with otolith membrane containing $CaCO_3$ crystal.
- Macula of saccule-oriented vertically-detects linear acceleration in vertical direction
- Macula of the utricle-oriented horizontally-detects linear acceleration in horizontal direction.
- Also detect change in head position.

MECHANISM OF ACTION:

Otolith organs are heavier than endolymph, thus specific gravity is more than endolymph.

CHANGE IN HEAD POSITION:

Tilting of head

↓

Change in the direction of gravitational pull in otolith membrane

↓

As cilia are entrenched in otolith membrane

↓

Bends cilia of some of the hair cells towards kinocilium.

↓

Action potential generated.

Linear acceleration of head

↓

Due to higher specific gravity of otolith membrane

↓

Membrane lags behind because of inertia

↓

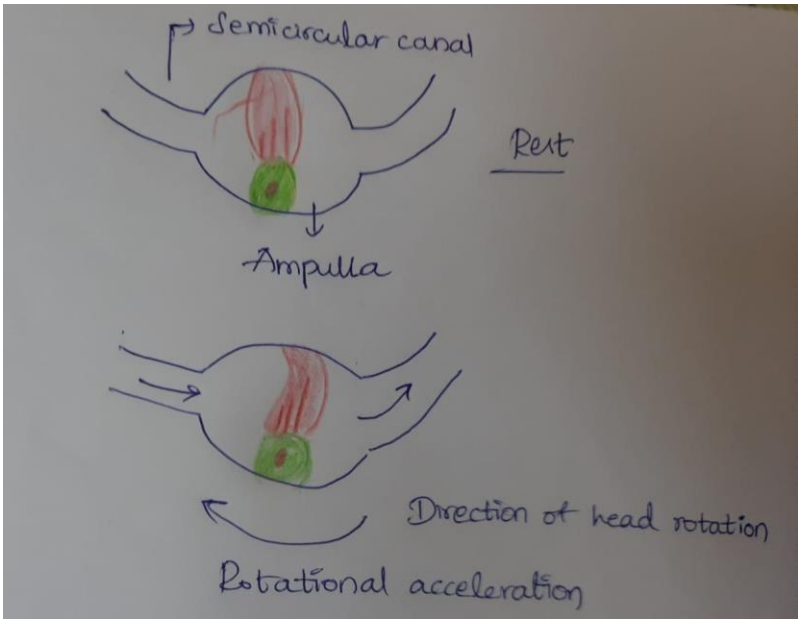
This causes bending of cilia

↓

Action potential generated.

SEMICIRCULAR CANALS:

- Hair cells located in ampulla
- Cilia of ampullary hair cell located in cupula.
- Specific gravity of cupular fluid is same as endolymph.



MECHANISM OF ACTION

When head rotates to one side

↓

Semicircular canals rotate to that side

↓

For 20 sec due to natural inertia endolymph does not move

↓

Initially endolymph lags behind

↓

As if endolymph moves in opp direction to canal

For example, if head rotates to right

Initially endolymph practically rotates to left

↓

Cupular fluid having same specific gravity moves with endolymph

↓

Cupular deflection bends cilia of hair cell

↓

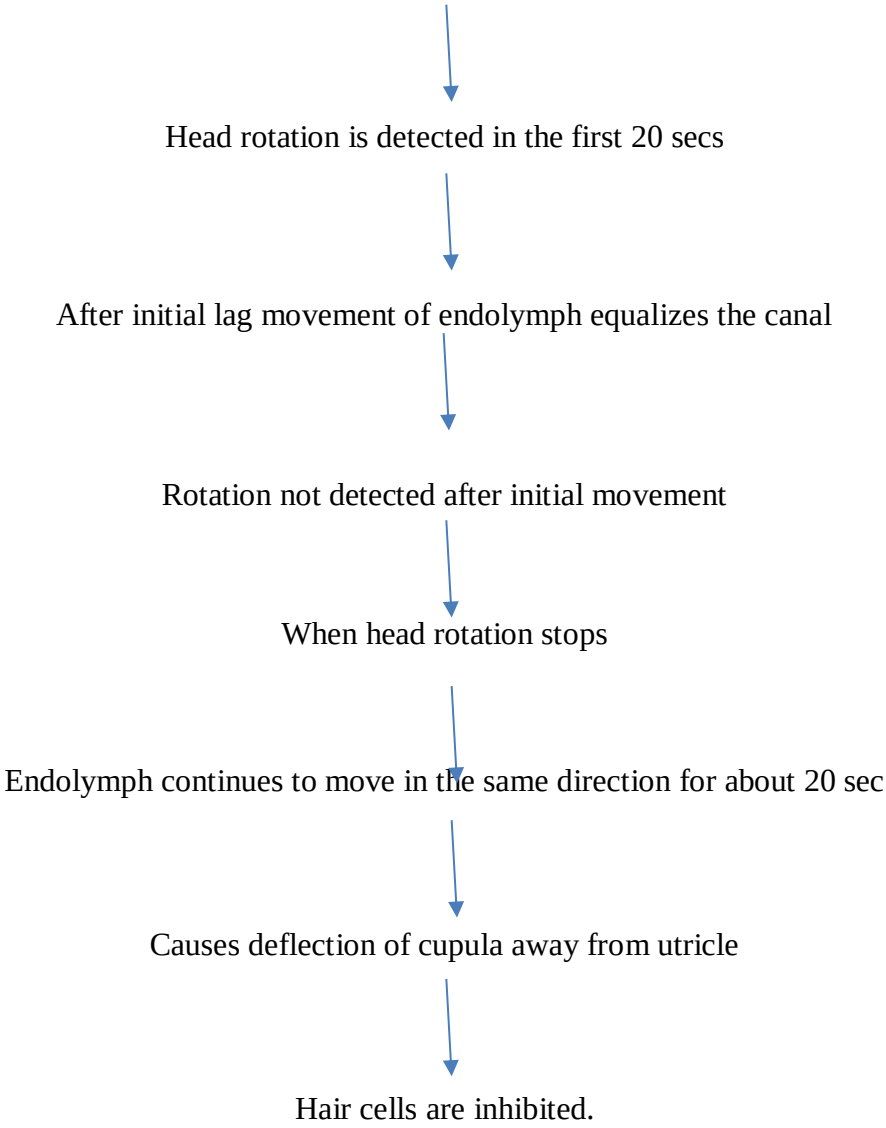
In ampulla kinocilium located towards utricle

↓

Hence displacement of cupula towards utricle

↓

Stimulates hair cell.



Arrangement of hair cells in the canals of both the ear is such that the beginning of rotation is detected by hair cells in the ear toward which rotation takes place and termination of rotation is detected by hair cells in the opposite ear.

APPLIED PHYSIOLOGY:

- VERTIGO-illusion of motion
- Physiological vertigo-MOTION SICKNESS due to overstimulation of vestibular apparatus.
- Central positional vertigo-lesion of the 8th cranial nerve

- Peripheral or labyrinthine vertigo
- Benign positional vertigo.

OTOLITH ORGANS AND THEIR ROLE IN ACCELERATION

There are 2 otolith organs:

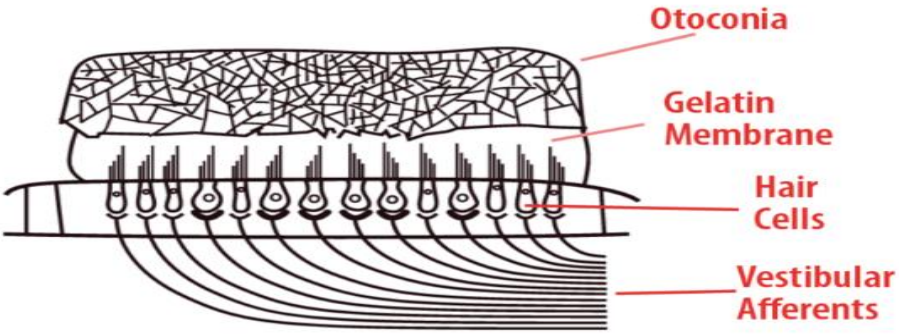
  - Utricle
  - Saccule

- Utricle lies in posterior part of bony vestibule.
- Saccule lies anterior to the utricle opposite the footplate of stapes
- It receives the 5 openings of 3 semicircular canals
- It is also connected to the saccule through utriculosaccular duct.
- Sensory epithelium of utricle and saccule is macula.
- Macula is made of hair cells similar to hair cells of crista amupllaris
- A gelatinous membrane lies over the hair into which the hair gets embedded.
- Crystals of calcium carbonate called otoliths or otoconia are present in the membrane
- The gelatinous membrane with otoliths is called otolith membrane
- The otoliths increase the specific gravity of otolith membrane to about twice that of the endolymph, hence it tends to respond to gravity changes.
- In utricle, macula is situated in horizontal plane so that hair cells are in vertical position
- In saccule, macula is in vertical position so that hair cells are in horizontal position
- They respond to linear acceleration (utricle to horizontal; saccule to vertical)
  - Movement of head in horizontal/vertical axis
  - Otoconia moves in opposite direction
  - Stimulation of hair cells
- Utricle also detects dorsiflexion and ventrifelxion
- Saccule also detects side to side movement of neck.
- A tilt of 2.5 degree is enough to stimulate appropriate maculae.

INNERVATIONS AND CONNECTIONS OF VESTIBULAR APPARATUS

- Vestibular or scarpa's ganglion is situated in lateral part of internal acoustic meatus
- It contains bipolar cells
- The distal part of bipolar cells innervates sensory epithelium while the central processes aggregate to form vestibular nerve.
- The fibers of vestibular nerve end in vestibular nuclei and some goes to cerebellum directly
- Vestibular nuclei are 4 in number (superior, medial, lateral, descending)
- Afferents to these nuclei comes from:
  - Peripheral vestibular receptors
  - Cerebellum
  - Reticular formation
  - Spinal cord
  - Contralateral vestibular nuclei
- Efferent from vestibular nuclei goes to

- Nuclei of CN 3, 4, 6 via medial longitudinal bundle. It is the pathway for vestibulo-occular reflexes and the reason for nystagmus
- Motor part of spinal cord. This coordinates the movements of head, neck and body in maintenance of balance
- Cerebellum. It helps to coordinate input information to maintain the body balance
- Automatic nervous system. Reason for nausea, vomiting, palpitation, sweating and pallor seen in vestibular disorders.
- Vestibular nuclei of opposite side
- Cerebral cortex. Responsible for subjective awareness of motion



APPLIED ASPECT:

MENIERE'S DISEASE

- Also called endolymphatic hydrops
- It is a disorder of inner ear where endolymphatic system is distended with endolymph.
- It is characterized by vertigo, sensorineural hearing loss, tinnitus and aural fullness.

SYNAPSE AND ITS PROPERTIES

Definition: Physiological junction without anatomical union two neuron or between a neuron and an effector such as muscle or a gland.

Usually a neuron may receive 10,000 synaptic inputs.

Classification

Anatomical classification

1. Axodendritic: most common, formed between axon of presynaptic neuron and dendrite of postsynaptic neuron, often excitatory
2. Axosomatic: synapses between axon and the cell body, mostly inhibitory
3. Axoaxonic: synapses between two axon, rare, often modulatory in nature
4. Dendro-dendritic: between dendrite of one neuron and dendrites of other neuron, seen in olfactory bulb

Based on type of transmission

1.Chemical:

Transmission of impulse takes place by release of chemicals, unidirectional, present in vertebrates including humans

2.Electrical:

Transmission of impulse by transfer of ions directly in both directions, seen in invertebrates. In mammals,

a. Lateral vestibular nucleus, hippocampus and cerebral cortex

b. Cardiac and smooth muscle

c. Respiratory neuron in mammals

3.Conjoint:

Transmission is both chemical and electrical

Based on response

1.Excitatory synapse

2.Inhibitory synapse

3.Modulatory synapse

Structure of synapse(Axodendritic)

Junction between two neuron

- 1st neuron presynaptic neuron(axon)
- 2nd neuron postsynaptic neuron(dendrite)

1.Presynaptic nerve terminal

This is dilated terminal nerve endings. This contains

a. Vesicles that store the neurotransmitters
b. Mitochondria
c. Microtubules

Membrane present called presynaptic membrane, contains dense bar or active zone at which vesicles fuse, rupture and release of neurotransmitters into the synaptic cleft

2.Post synaptic nerve terminal

- Membrane present called the postsynaptic membrane
- Part of postsynaptic membrane that is in opposition with presynaptic membrane called subsynaptic membrane, contains a number of protein receptor

3.Synaptic cleft

- Space between presynaptic and postsynaptic terminal
- Measures about 200-400 deg A
- Filled with amorphous tissue, containing enzymes that destroys the released neurotransmitters after its action is over

Mechanism of transmission of impulse(excitatory) :

Impulse (AP) is conducted along the nerve fibre to the presynaptic nerve terminal causing its depolarization.

⬇

Voltage-gated Ca++ channels open and Ca++ enters the presynaptic nerve terminal.

⬇

Vesicles fuse (docking) with the presynaptic mem brane and rupture, releasing the neurotransmitter into the synaptic cleft.

⬇

Neurotransmitter combines with the receptor sites present on the subsynaptic membrane.

⬇

This leads to the opening of ligand-gated Na+ channels & Na moves into postsynaptic nerve terminal.

⬇

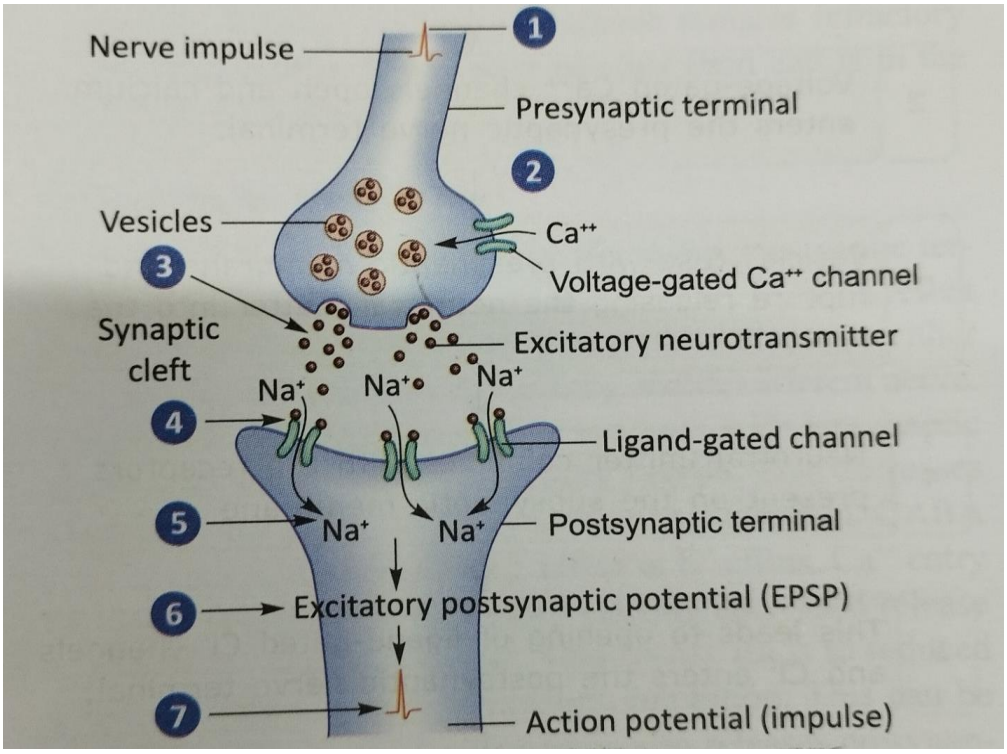
A local potential called Excitatory Post Synaptic Potential develops (amplitude 15 mV).

⬇

This triggers an action potential at the initial segment of postsynaptic neuron, which is more sensitive due to the presence of more number of voltage-gated ionic channels.

⬇

This is conducted in both directions, i.e., towards somato dendritic tree and along the axon of the postsynaptic neuron.



INHIBITION OF THE TRANSMISSION AT SYNAPSE

Conduction of nerve impulse along the nerve fibre to the presynaptic nerve terminal causing its depolarization.

⬇

Voltage-gated Ca++ channels open and calcium enters the presynaptic nerve terminal.

⬇

Vesicles fuse with presynaptic membrane and rupture releasing the neurotransmitter into the synaptic cleft.

⬇

Neurotransmitter combines with the receptors present on the subsynaptic membrane.

⬇

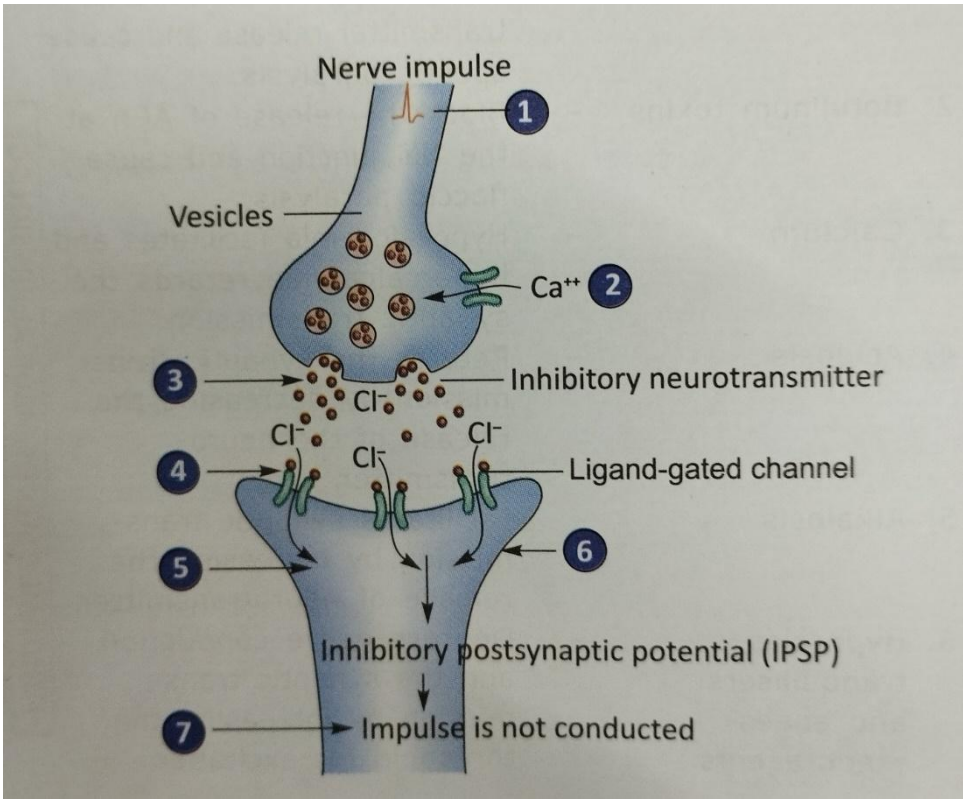
This leads to opening of ligand-gated Cl channels and Cl enters the postsynaptic nerve terminal.

⬇

Inhibitory Post Synaptic Potential develops due to hyperpolarization (IPSP).

⬇

Impulse conduction is blocked.



Properties of synapse

1.One-way conduction:

- Conducts impulse in one direction only
- Reason: presence of receptor only on the postsynaptic membrane to the neurotransmitter released from the presynaptic nerve terminal
- Useful for orderly function of neural function

2.Synaptic delay:

- Due to various mechanism involved in the synaptic transmission, some time is lapsed at the synapse
- For one synapse the delay is 0.5msec
- Synaptic delay depends on the number of synapse present in the pathway

3.Summation:

A facilitated response to repeated stimuli applied simultaneously or one after the other is called summation. It is of two types,

- **Spatial summation:** When one subthreshold stimulus is applied to one afferent nerve fibre no response is obtained but it produces a local EPSP on the post

synaptic new When subthreshold stimuli are applied to two or afferents, which make synaptic connections with the motor neuron, simultaneously, then a response is obtained due to summation of EPSPs on the postsynaptic ne This is referred to as spatial summation.

- **Temporal summation:** When a single subthreshold stimulus is applied to an afferent nerve fibre synapsing with a motor neuron, only an EPSP is produced. When afferent fibre is stimulated with same subthreshold repeatedly at a rapid rate, a propagating spike potential develops on the motor neuron due to summation of the previous stimuli. This is called temporal summation.

4.Fatigue:

On repeated stimulation, the synapse goes into easy fatigue due to exhaustion of neurotransmitters

5.Occlusion:

- Decline in response than normally expected
- Reflex response that is obtained by stimulating two afferent neuron is less than the response obtained when they are stimulated separately
- This is due to presence of common neuron in both groups.

6.Subliminal fringe:

- Partial state of excitation
- Reflex response that is obtained by stimulating two afferent neurons together is more than the response when they are stimulated separately

7.Facilitation:

- When a stimulus is applied to an afferent nerve, some response is obtained.
- When a second and third stimuli are applied immediately the response obtained are better than first one
- Due to beneficial effects, increased Ca++ influx into the presynaptic neuron

8.Divergence:

- One neuron connecting to several neuron

Present in

a. Reticular activating system which is required for wide spread activation of brain
b. SNS for wide spread activation of body
c. Focal epileptic seizures

9.Convergence:

Several neuron connecting to single neuron

a. Anterior horn cell receiving impulse from several sources
b. Several sensory neuron converging on a single relay neuron in the sensory tracts
c. Retinal to visual cortex

10.Reverbertory circuit:

Activity in the neuronal circuit continues for longer period even after cessation of stimulus.

This is basis for after discharge

a. Short term memory
b. Smooth decline and termination of a movement.

SHORT NOTES

ROLE OF HYPOTHALAMUS IN SATIETY AND HUNGER

Role of hypothalamus in satiety

- **Satiety center:** ventromedial hypothalamus, its stimulation causes cessation of eating
- Decreases food intake by inhibiting feeding center

Role of hypothalamus in feeding

- **Feeding center:** lateral hypothalamus, its stimulation greatly increases food intake
- Remains chronically active and gets inhibited by satiety center once satiety is achieved

THERMOSTAT MECHANISM OF HYPOTHALAMUS

- Thermosensor cells in the hypothalamus detect changes in the temperature of blood and receive inputs from thermo sensitive receptors in the skin which is matched with set point of body temperature and initiate temperature regulating mechanisms
- Done depending on balance between mechanism that controls heat loss and heat gain

Anterior hypothalamus- activates mechanism that promotes heat loss by causing cutaneous vasodilation & sweating

Posterior hypothalamus- activates the mechanism that increases heat production and promotes heat gain by vasoconstriction and piloerection

Applied aspect:

Global hypothalamic syndrome:

- The disease involves either all parts or a large part of hypothalamus
- Primary tumors or metastatic carcinomas, lymphoma or granulomatous diseases like sarcoidosis results in such syndromes
- Patient dies due to visceral homeostatic mechanism failure

Partial hypothalamic syndrome:

Occur due to specific lesions of a specific part of hypothalamus resulting in deficiency or overproduction of a single hormone. Some of the important syndromes are

- Diabetes insipidus
- Neurogenic salt wasting
- Precocious puberty
- Body weight alteration
- Anorexia nervosa and bulimia
- Disturbance in temperature regulation
- Hypothalamic cardiovascular disorder
- Neurogenic pulmonary edema

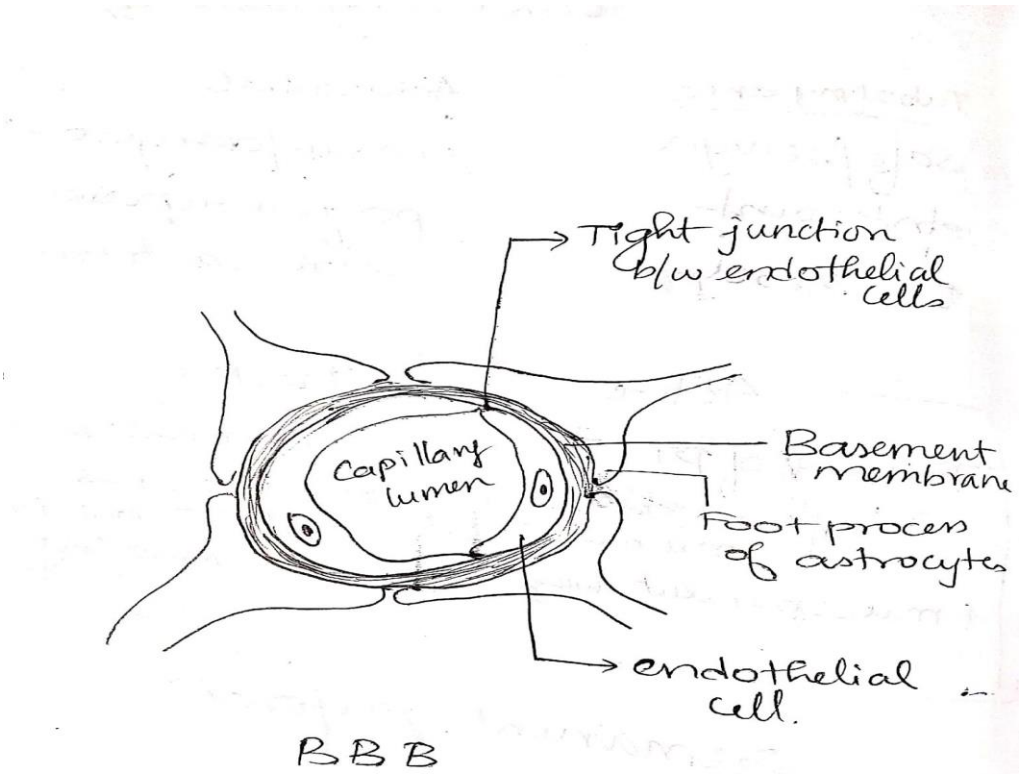
BLOOD BRAIN BARRIER

A special barrier that exists between the blood and the brain tissue is the blood-brain barrier (BBB). This was first demonstrated by Paul Ehrlich.

Anatomical basis

BBB is formed by two special structures

1. In the endothelium of capillaries of the brain, cells are joined by tight junctions. This significantly decreases the permeability of the capillaries.
2. The capillaries are surrounded by the foot processes of astroglia. These glial processes form a complete sheath around the capillaries.

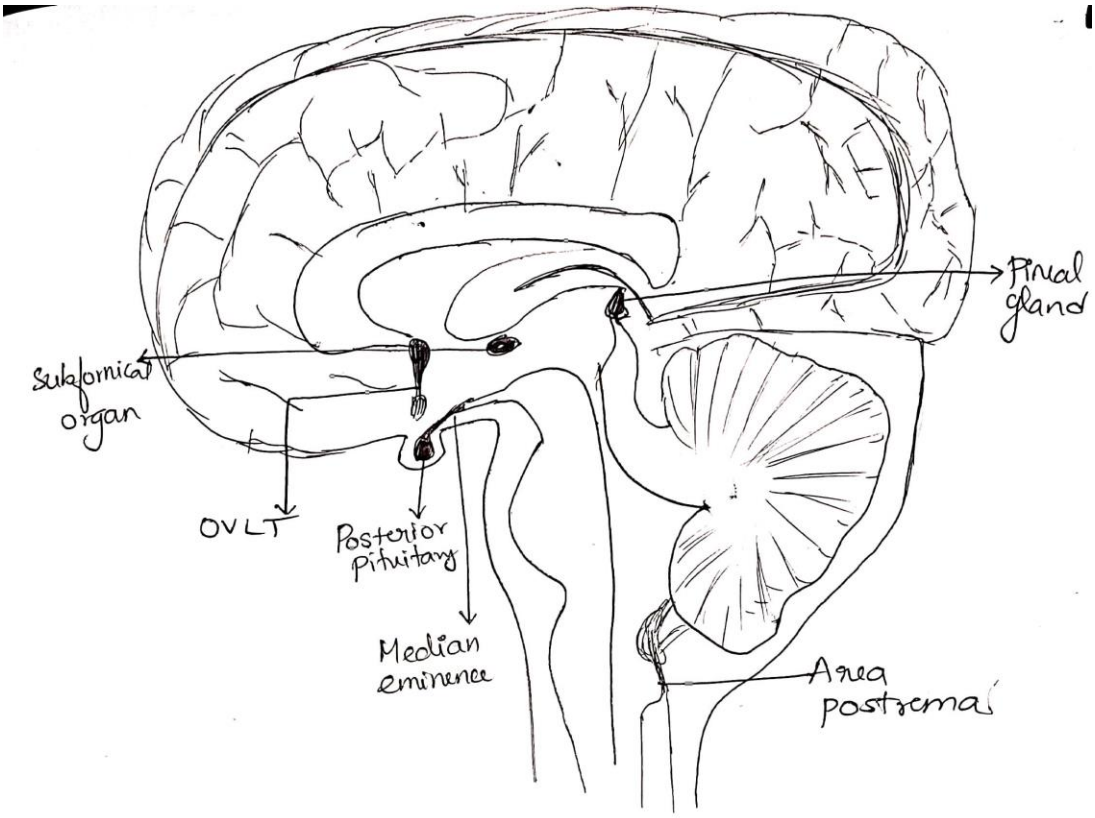


CIRCUMVENTRICULAR ORGANS

Normally, BBB is deficient in some regions of the brain that are collectively called as circumventricular organs

The important circumventricular organs are:

1. Organum vasculosum of lamina terminalis (OVLT)
2. Subfornical organ (SFO)
3. Area postrema in medulla oblongata
4. Posterior pituitary and median eminence (ventral part) of hypothalamus
5. The pineal gland


Pineal gland
Subfornical organ
OVLT
Posterior Pituitary
Median eminence
Area postrema

Functions of BBB:

1. Neurons depend on a normal concentration of various ions in the fluid bathing them, especially Na+, K+, Ca++, H+ and Mg++. Alteration in these ion concentrations in ECF of brain tissue results in severe consequences. B**BB maintains constancy of these ions in brain fluids.**

2. Many toxins either produced endogenously or admini-stered exogenously circulate in blood. These toxins are harmful to the neurons that are highly sensitive. BBB **protects neurons from these harmful substances.**
3. BBB prevents escape of neurotransmitters from brain into circulation.
4. Disruption of BBB helps in identifying the location and extent of lesions in the brain.

5. BBB influences drug permeability into the brain.

Clinical Importance

- Kernicterus
- Brain Tumor
- Infection and Injury

PARKINSON'S DISEASE

A disease of the old age
Characterized by the degeneration of the nigrostriatal pathway that leads to excessive loss of dopamine and dopamine receptors
Features:

- Akinesia
- Bradykinesia
- Mask face
- Rigidity (cogwheel and lead pipe rigidity)
- Resting tremors
- Festinant gait

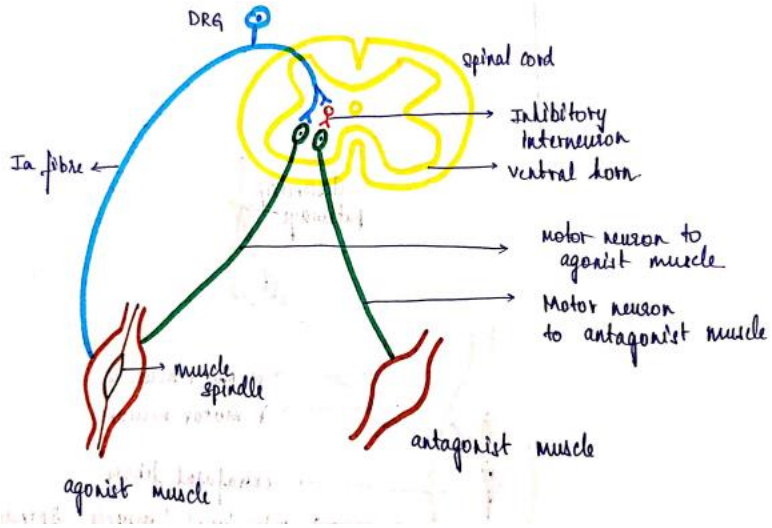
Treatment: anticholinergics, dopamine agonists, dopamine precursors

STRETCH REFLEX

- **DEFINITION -** The reflex contraction of the muscle to stretch when a skeletal muscle with its intact nerve supply is stretched is called the stretch reflex.
- **STIMULUS** - **stretch** of the muscle
- **RESPONSE**- **contraction** of the stretched muscle.
- There are two types of stretch reflexes: the phasic stretch reflex and the tonic stretch reflex.

■ Phasic Stretch Reflex

1. Stimulus - sudden stretch of the muscle.
2. Receptor - muscle spindle
3. Afferent group - Ia afferent fiber (2 branches)



4. Function -

One branch terminates monosynaptically on the motor neuron (homonymous) supplying protagonist muscle

Other branch terminates disynaptically on motor neuron (heteronymous) supplying antagonist muscle **via inhibitory interneuron**

✓ Thus, activity in the Ia afferent fiber **stimulates the homonymous motor neuron** that causes contraction of the protagonist muscle, and **inhibits the heteronymous motor neuron** that causes relaxation of the antagonist muscle.

✓ Such an innervation that causes activation of a set of motor neuron and inhibition of another set of motor neuron is called **reciprocal innervation.**

Therefore, when the agonist muscle contracts, simultaneously the antagonist muscle relaxes.

■ Tonic Stretch Reflex

1. Stimulus - sustained stretch of the muscle.
2. Receptor - Muscle spindle
3. Afferents - both Ia and II afferent fibers.
4. Function -

- ✓ Tonic stretch reflex **contributes to the muscle tone**.
- ✓ Tonic stretch reflex is also important for **regulation of posture**.
- ✓ To maintain standing position, the extensors of knees should contract so that knees remain extended and legs do not bend. This is achieved by the **action of gravity on medial extensors of the thigh**.
- ✓ The **sustained stretch of extensors** results in sustained contraction of these antigravity muscles that maintains extension at knee joint. Thus, the standing position is maintained.
- ✓ After assuming standing posture for a longer duration, **due to fatigue, gradually the knees bend**

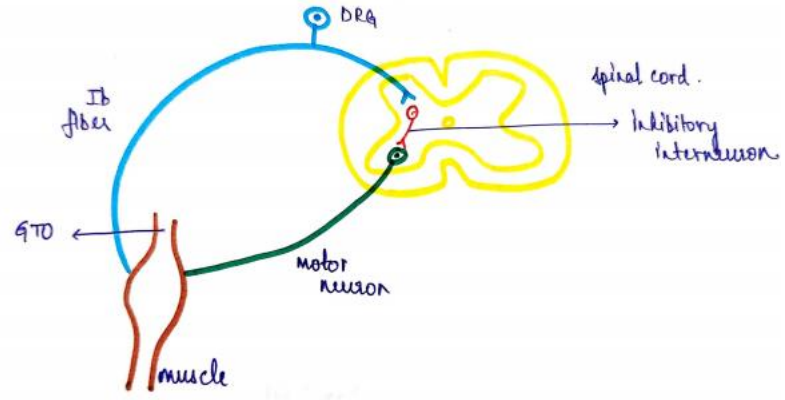
that further stretches the quadriceps muscles. The flexion at knee joints elicits additional tonic stretch reflex that, **causes added sustained contraction** of quadriceps. This maintains further extension of knees and prevents the person from falling.
Thus, stretch reflex helps **to restore and maintain the posture for a very long period**.

INVERSE STRETCH REFLEX

- DEFINITION -Relaxation of the muscle in response to a strong stretch is called **inverse stretch reflex**.

- **REFLEX ARC**

1. Receptors - Golgi tendon organs
2. Stimulus - **stronger stretch** or An active muscle contraction .
3. Afferent fibers **Ib fibers** that terminate on the **inhibitory interneurons** that, in turn, project to the homonymous α motor neurons.
4. This results in inhibition of the agonist muscle.



- **FUNCTIONS** - Golgi tendon organ **monitors force developed in the muscle**.
  1. The force is detected either by strong stretch or by an active contraction. Stimulation of the GTO **inhibits the agonist muscle** through its reflex connections.
  2. The agonist muscle relaxes in response to activation of GTO.
  3. Thus, a stronger stretch imparted on the muscle automatically inhibits the muscle. Therefore, the reflex is also called **autogenic inhibition**.

- **PHYSIOLOGICAL SIGNIFICANCE** -
  Muscle spindle (stretch reflex) monitors muscle length and GTO (inverse stretch reflex) monitors muscle tension, i.e. the force of contraction.
  Inverse stretch reflex, by allowing the muscle to relax, **prevents rupture of muscle** when the muscle is stretched to greater extents.

- **EFFECT OF γ MOTOR NEURON ON STRETCH**
  1. Descending fibers from supraspinal segments increase the discharge of γ motor neurons, which, in turn, increases the sensitivity of the spindle to stretch.
  2. **In upper motor neurons paralysis**, the increased γ motor neuron discharge (due to loss of inhibitory suprasegmental inputs on the γ motor neurons) increases the reflex activity.
  3. The muscle becomes **hyper-reactive due to increased phasic stretch reflex activity** and becomes **hypertonic due to increased tonic stretch reflex activity**.

- **MUSCLE TONE** - **Resistance of the muscle to stretch** is called tone.
  1. Muscle becomes hypertonic or spastic due to hyperactive stretch reflexes (increased γ motor neuron discharge).
  2. Muscle becomes hypotonic or flaccid when the motor neurons supplying the muscle are damaged or when the discharge of γ motor neurons is decreased.

- **CLASP KNIFE PHENOMENON**
  - ✓ When a hypertonic muscle as seen in upper motor neuron paralysis is stretched, muscle contracts, but if the stretch is continued then muscle relaxes.
  - ✓ This type of **high resistance followed by sudden collapse** is known as clasp-knife phenomenon as it resembles the closing a clasp-knife.

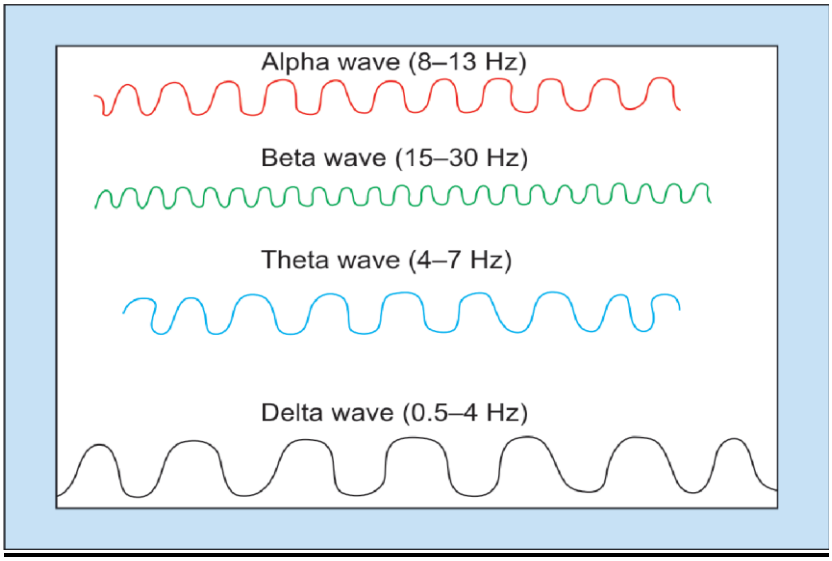
EEG

(electroencephalogram)

- Record of spontaneous electrical activities generated in cerebral cortex
- Electroencephalography- procedure
- Electroencephalograph- instrument
- Unipolar and bipolar leads are used for recording
- Unipolar lead consists of one exploring electrode and one indifferent, which is kept at zero potential
- Bipolar lead consists of two exploring electrodes and potential difference between the two electrodes is recorded

EEG PATTERN

- Alpha rhythm:
  - Frequency:8-13Hz
  - Amplitude: 50-100µV
  - Most prominent wave
  - Can be recorded with closed eyes from parieto-occipital area in resting man
  - When eyes are opened alpha is replaced by beta. This is referred to as alpha block
- Beta rhythm:
  - Frequency:12-32Hz
  - Amplitude: 5-10µV
  - Faster rhythm with lowest voltage
  - Indicates alert stage
  - Recorded from parietal and frontal region
- Theta rhythm:
  - Frequency:4-7Hz
  - Amplitude: 10µV
  - Seen normally in children and during moderate sleep in adults
  - Recorded from parietal and temporal region
- Delta wave
  - Frequency:0.5-4Hz
  - Amplitude: 20-200µV
  - Seen during deep sleep in adults
  - Recorded from occipital area
  - Appearance in alert state indicates serious organ damage.



Clinical use of EEG:

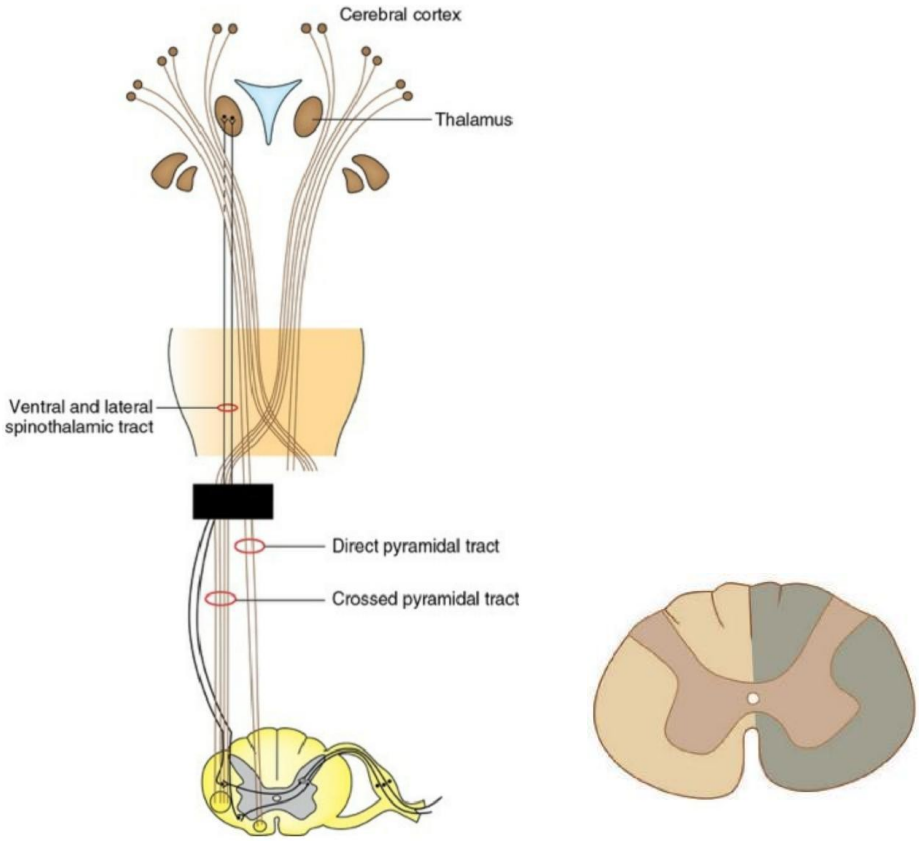
- Diagnose epilepsy
- Confirm type of epilepsy
- Identify cortical site of abnormal discharge
- Intracranial space occupying lesion
- Sleep analysis
- Diagnose sleep disorders

Applied physiology:

- Epilepsy: Intermittent disorder characterized by sudden uncontrolled discharge of cerebral neurons with or without loss of consciousness
- Types:
  - Generalized:
    - Grand mal epilepsy (generalized tonic clonic seizure)- discharge from both hemispheres
    - Petit mal epilepsy (absence seizures)
  - Focal (temporal lobe and jacksonian epilepsy)

BROWN SEQUARD SYNDROME

- Brown Séquard Syndrome is a functional hemisection of the spinal cord.
- It is usually seen in injury to the spinal cord or in tumors of the spinal cord that affects only half of the cord.
- Damage occurs to ipsilateral dorsal-column pathway, contralateral spinothalamic tract and ipsilateral descending motor (corticospinal tract) pathways.



Sensory deficit:

- On the side of lesion, the fine touch sensation, proprioceptive sensations (sensations from tendons, muscles, joints and vibration sense) and tactile discrimination are lost.
- On the opposite side, pain and thermal sensations are lost.
- This occurs because sensation for fine touch, proprioception, and two point discrimination ascend up in the dorsal column of the same side, whereas the sensation for pain and temperature ascend up in the anterolateral system in the opposite side of the spinal cord.

Motor deficit:

There is also damage to corticospinal tract on the side of hemisection of spinal cord. This causes paresis (muscle weakness) and spasticity of muscles of the same side of the body.

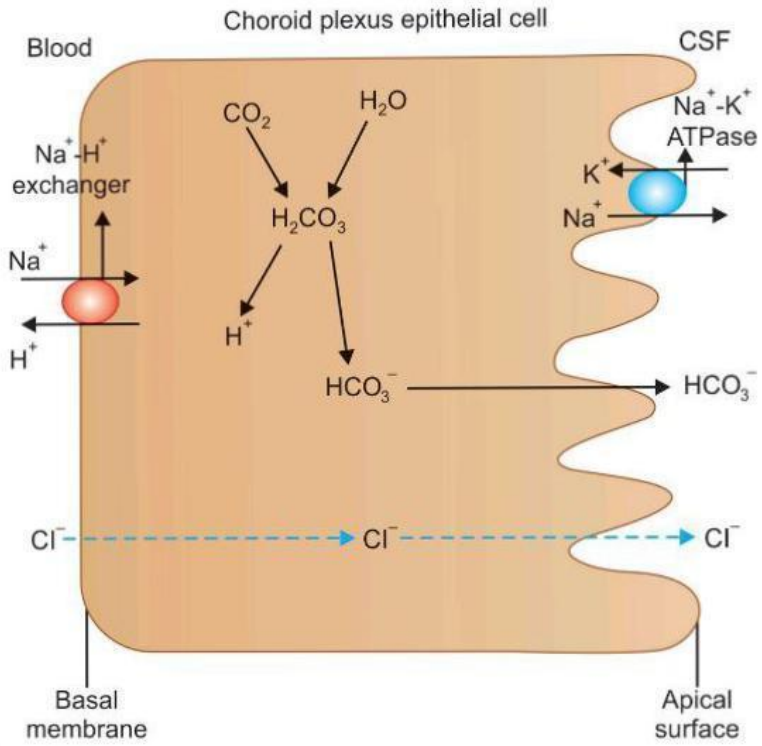
CEREBROSPINAL FLUID

Formation of CSF:

- Choroid plexuses, located in the floor of lateral, third and fourth ventricles, are the major source of CSF. The other sources of CSF are the blood vessels of subependymal regions and pia mater.
- CSF accounts for about 70–160 ml. CSF occupies less than 10% of the volume of total intracranial space.
- The rate of formation of CSF is about 0.35 mL/min or, 20 mL/hour or, 500 mL/day. As a whole, CSF is totally replaced four to five times daily

Mechanism of CSF formation:

CSF is formed by a combination of passive diffusion, active transport and facilitated diffusion.



- Formation occurs in two steps namely ultrafiltration and active secretion.
- Ultrafiltration of plasma occurs across the fenestrated capillary wall into the ECF that baths the basal surface of epithelial cells of choroid plexus.
- Choroidal epithelial cells transport ions and solutes into CSF, mainly by active secretion.

Circulation and Absorption of CSF:

Circulation of CSF was described by Harvey Cushing as the third circulation in the body.

- ❖ The CSF is mainly formed in the lateral ventricle, from where it flows downward into the third ventricle through foramen of Monro, and from the third ventricle into the fourth ventricle though aqueduct of Sylvius.
- ❖ Finally, CSF comes out of fourth ventricle through the foramen of Magendie and Luschka to enter the subarachnoid space.
- ❖ In the subarachnoid space, CSF moves upward toward the cerebral hemispheres and downward toward the spinal cord (Fig. 141.3).
- ❖ Thus, obstruction of foramen of Monro results in distension of lateral ventricles, occlusion of aqueduct of Sylvius causes distension of third ventricle, and blockage of foramina of Magendie and Luschka initially distends fourth ventricle and later the entire ventricular system is distended.
- ❖ Absorption of CSF occurs through the arachnoid villi.The main factor that facilitates the movement of the fluid is the oncotic pressure .Other factor promoting this mechanism is the hydrostatic pressures of CSF.

Functions of CSF:

- ✓ Protection from mechanical injury: A major function of CSF is to protect brain from mechanical injury. Due to higher specific gravity, brain floats freely in CSF rather than resting heavily on the skull box. Thus, the risk of routine acceleration-deceleration injuries is eliminated and also the impact of major injuries is greatly diminished.
- ✓ Provides microenvironment for brain cells: Brain is metabolically fragile. Neurons in the brain are highly sensitive to changes in oxygen, glucose, pH, temperature, etc. in their external environment. However, the CSF ensures constancy in the external environment of neurons. CSF accomplishes this by buffering the changes in blood on one side and with the brain interstitial fluid on the other.
- ✓ Role in homeostasis: CSF is indirectly involved in regulation of respiration, blood pressure, water intake and visceral function by bringing about the chemical changes like hydrogen ion concentration (pH), osmolality, etc. in cerebral interstitial fluid. The changes in blood PO2, PCO2 and pH are transmitted to chemosensitive respiratory neurons and central chemoreceptors via CSF for appropriate homeostatic responses.
- ✓ Removal of proteins and waste products: There are no lymphatic channels in brain and spinal cord. CSF removes proteins and waste products of metabolism, especially H+,

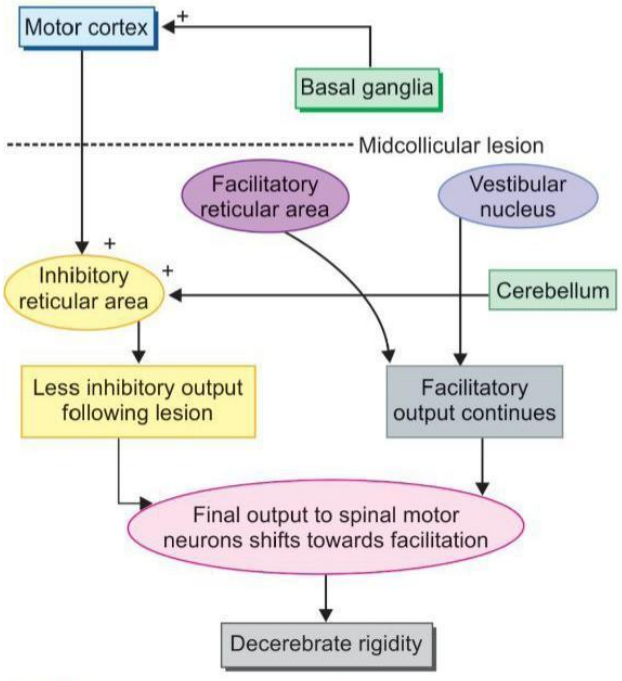
lactate and CO2 through its sink action. In the brain, small amount of protein that leaks into the interstitial fluid is drained by the CSF and returned to the blood stream. Thus, the CSF serves the function of lymphatics in brain.

DECEREBRATE RIGIDITY

After a mid-collicular section (section between superior and inferior colliculi in an experimental animal), severe spasticity is immediately observed in the extensor group of muscles of the body. This is called decerebrate rigidity. Rigidity is so prominent that the limbs are fully extended and the spine is hyperextended.

Mechanism of Decerebrate Rigidity:

- In midcollicular lesion, the influence of cortex and basal ganglia on inhibitory reticular area is abolished (only cerebellar drive remains).
- Therefore, inhibitory output of the medullary reticulospinal tract becomes less inhibitory, whereas facilitatory area continues to discharge spontaneously.
- Consequently, the net output of reticulospinal tract becomes more facilitatory. As motor neurons are primarily driven by reticulospinal tract influence, decerebration causes severe rigidity.



Importance of Decerebrate Rigidity:

Rigidity observed in decerebrate animal is more marked in the extensor muscles.

- Extensor muscles are the most important components of posture regulating system as they maintain erect posture of the body by keeping the limbs extended.
- The tone of these muscles, which is a static postural reflex, is highly essential to support the animal against gravity. Therefore, these muscles are called antigravity muscles.
- In humans, the major antigravity muscles are the extensors of the lower limbs.
- The increased extensor rigidity in decerebrate preparation indicates that medulla controls the tone of the antigravity muscles that are involved in maintaining posture.

UMN AND LMN PARALYSIS

UMN paralysis:

It is the paralysis that results from the lesion of descending fibres between their origin from cortical motor areas and their termination on the anterior horn cells of the spinal cord.

Features:

- Increased muscle tone (spasticity).
- No muscle atrophy, over prolonged time **disuse atrophy can occur.**
- Muscles affected in groups.
- Exaggerated tendon reflexes.

Physiological basis:

- Spasticity occurs due to **increased motor neuron discharge and increased excitability of the motor neurons**
- There is a normal physiological inhibition that is exerted on the reticulospinal pathway by the corticoreticular fibres which is interrupted in UMN paralysis, Thereby, there is facilitatory action o the reticulospinal fibres causing hypertonia and spasticity
- Because of the loss of inhibition of the reticulospinal pathway, there is an increased gamma motor neuron discharge that increases the sensitivity of the muscle spindle to stretch **exaggerated deep tendon reflex.**
- **Superficial reflexes are abolished** in UMN paralysis as the efferent pathway is abolished.

LMN paralysis:

- This type of paralysis occurs in diseases that cause destruction of anterior horn cells or their axons in dorsal root.
- Examples are poliomyelitis, motor neuron disease and lesion of nerve roots.

Features:

- Hypotonic muscles **(flaccidity)**
- Pronounced **muscle atrophy**
- **Individual muscles** affected
- Tendon reflexes are absent
- **Babinski sign is negative**
- Fibrillation, fasciculation and sharp waves (defibrillation potentials) seen
- Nerve conduction either decreased or absent

Physiological basis:

- Individual innervation to the muscle is lost, thereby muscles are affected individually. **Pronounced muscle atrophy** is seen
- Loss of reflex arc is seen so superficial and deep reflexes are lost
- **Decreased gamma motor neuron discharge** causes the hypotonia of muscles
- Babinski sign is not elicited due to loss of motor neuron activity.

	UMN paralysis	**LMN paralysis**
Muscles affected	In groups	Individual
Size of muscles	No atrophy	Atrophy is pronounced
Type of paralysis	Spastic paralysis	Flaccid paralysis
Tendon reflexes	Exaggerated	Diminished/absent
Superficial reflexes	Absent	Absent
Babinski sign	Extensor plantar response (sign positive)	Flexor plantar response (sign negative)
Involuntary movements	Absent	Present (in form of defibrillator potentials)
EMG changes	No denervation potential	Denervation potential seen
Nerve conduction study	No abnormalities	Decreased nerve conduction

REM AND NREM SLEEP

- ☒ In the normal sleep cycle, the sleep begins with a phase of **NREM** sleep, also called as the slow wave sleep.
- ☒ There are 4 stages in slow wave sleep after which the person progresses to **REM** sleep
- ☒ With the completion of REM phase, sleep cycle completes.

NREM sleep:

- There are 4 stages of progressive deepening of sleep.
- It is difficult to wake the subject in this phase and the EEG pattern progresses to slower frequency waves.
- Stage 1: the subject becomes drowsy, there is a change from beta to alpha rhythm, light sleep begins
- Stage 2: the amplitude of EEG waves slightly increases, **sleep spindles and k complexes** are the characteristic features
- Stage 3: stage of moderate sleep, low frequency and high amplitude
- Stage 4: stage of deep sleep, delta rhythm is prominent, maximum slowing, very difficult to wake the subject up, decrease in all the autonomic functions

REM sleep:

- Also called paradoxical sleep.
- Rapid low voltage EEG activity is seen, the activity resembles the beta rhythm as seen in awakened state

Features:

- Rapid eyeball movements visible under the closed eyelids
- EEG pattern shown **desynchronised rapid beta rhythm,** subject likely to wake up spontaneously from REM sleep
- **PGO spikes**: phasic potentials that originate from pons and through LGB travel to occipital cortex (ponto geniculo occipital spikes)
- Dream seen in this stage is easily remembered
- Stimulation of sympathetic system is seen, increase in the autonomic functions
- In males, penile erection occurs – important to diagnose erectile dysfunction
- Profoundly depressed muscle tone, widespread hypotonia except **extraocular and middle ear muscles**

	NREM sleep	**REM sleep**
Timing in sleep cycle	Occurs first	Occurs after NREM
Duration	75% of total sleep	25% of sleep
Autonomic functions	Sympathetic inhibition	Sympathetic excitation
Eyeball movement	No eye movement	Rapid eye movement
Dreams	Not memorised	Memorized
Muscle tone	Inhibited	Depressed
Type of sleep	Enters into deep sleep	Sleep lightens
EEG waves	Slow wave amplitude	High frequency low voltage
Mechanism	Inhibition of RAS	Activation of PGO axis

PROPERTIES OF RECEPTORS

Definition:

- Receptors are transducers that convert various forms of energy in the environment to action potentials in sensory neurons
- They are the **ending of afferent nerve fibres**
- The receptor is usually associated with non-neural cells that surround it, together they are known as a **sense organ**
- They are **specific for a particular stimulus,** the form of energy that the receptor is most sensitive to is called **adequate stimulus**

Properties of receptors:

Specificity: they are specific to a particular type of stimulus

Adequate stimulus: the form of energy to which the receptor is most sensitive to is called adequate stimulus

Adaptation: when a stimulus of constant strength is applied continuously, frequency of action potential in the sensory nerve decreases, also called as **desensitisation,** forms the basis of classification of receptors as phasic and tonic receptors.

☒ **Phasic receptors:** adapt rapidly, eg., touch and pressure receptors
☒ **Tonic receptors:** adapt slowly, eg., baroreceptors in carotid sinus

Acuity: the precision of stimulus localisation, **depends on the number of receptors present in the area of application of stimulus**

Intensity: receptors discharge based on the strength of stimulus.

☒ If low strength stimulus is applied, receptors with less threshold are activated

☒ If more strength is applied, neurons are activated quickly and receptors present further are also activated- **receptor recruitment**

Weber Fechner law: the magnitude of sensation is directly proportional to the log of intensity of stimulus

Law of projection: no matter where a specific sensory pathway is stimulated along the course, the sensation formed is referred to the location of the receptors. **Forms the basis of phantom limb**

Muller's Doctrine of specific nerve energy: the sensation evoked by a stimulus that generates impulse in the pathway depends on the precise area of the brain that is activated by the stimulus.

Sensory unit and receptive field:

☒ Sensory unit is defined as every single sensory axon and all of its peripheral branches
☒ Receptive field of a sensory unit is the area from which a stimulus produces a response in that unit.

WALLERIAN DEGENERATION

Degenerative changes take place at three levels:

1. Changes in the distal-stump

2. Changes in the proximal stump (at the site of injury)

3. Changes in the cell body

CHANGES IN DISTAL STUMP

- When a peripheral nerve is cut, the part of the nerve separated from the cell body shows a series of chemical and physical degenerative changes. These constitute Wallerian degeneration.
- They appear within few hours and continue for several days.
- Degenerative changes also appear in the proximal part and in the cell body.
- These changes are called retrograde degeneration. Regeneration starts from the proximal stump and proceeds distally at a slower rate.

The degenerative period may be divided into:

A. Early phase

B. Late phase

A. Early phase (1-7th day):

- During an early phase, only functional changes appear without physical changes.
- This phase lasts for about a week.

The following changes appear during an early phase in a sequential order:

1. Changes in the enzymatic activity of choline acetylase and ACh esterase.

2. Changes in the activity of ionic channels (decrease).

3. Change in amplitude and conduction velocity of nerve impulse appears within 2-3 days.

4. Ultrastructural changes in the organelles appear.

5. Failure of conduction of nerve impulse by 5th day. "B" fibres are the first to fail to conduct impulses followed by "A" and finally "C" type fibres.

6. No gross structural changes take place until 7 days.

B. **Late phase (8-32 days):**

During this phase, histochemical and physical changes appear.

The changes are:

1. Neurofibrils swell and then disappear.

2. Axis cylinder breaks up into short lengths.

3. Myelin sheath disintegrates into droplets of fat.

4. Axon debris and disintegrated myelin are removed by macrophages

5 Neurilemmal sheath with its nucleus and endoneural tubes are tent intact at the end of 32 day

The entire degenerative process is completed by 4 weeks.

CHANGES IN PROXIMAL STUMP

In the central stump (proximal stump), where the nerve fibres are still attached to their cell bodies, degeneration is usually confined to a centimeter or so next to the point of section. These changes are very similar to the changes that take place in the distal stump.

CHANGES IN CELL BODY

Degenerative changes also appear in the cell body.

changes depend on:

1. Severity of injury

2. Proximity of lesion to the cell body

The changes are:

1. Chromatolysis: Nissl granules which represent endoplasmic reticulum and ribosomes break up in to fine dust and lose their staining reaction. Hence the name chromatolysis. This process is a reaction to injury and is necessary for synthesis of proteins required for neuronal survival.

2. Golgi apparatus, mitochondria and neurofibrils disappear.

3. The cell draws in more fluid and swells up.

4. The nucleus is pushed to one side. In severe cases, it may be totally extruded leading to the death of the neuron.

These changes, which are associated with an alteration in the excitability of the cells begin on the first day and reach the maximum in the third week, after which the cell regains its normal appearance.

WALLERIAN REGENERATION

Regeneration begins at the central end while the degeneration of peripheral stump is proceeding.

Regeneration is seen in peripheral nerves due to the following factors.

1. Neurilemma along with its nucleus is present in peripheral nerves. This is essential for regeneration of axon.
2. Schwann cells, fibroblasts, macrophages and injured peripheral neurons release growth promoting factors (neurotrophins), which stimulate regeneration.
3. Schwann cells multiply and form continuous cords. This bridges the gap between the cut ends and guides the growing filaments from the proximal stumps.

CHANGES IN CELL BODY DURING REGENRATION

The regenerative changes start within 3 weeks and will be completed in 80 days.

1. Nissl granules reappear.
2. Golgi apparatus, neurofibrillae and other organelles reappear.
3. The nucleus occupies central position again.

Regeneration in the cell body may occur even when the axon does not regenerate.

MECHANISM OF REGENERATION OF AXON

1. A large number of thin filaments called fibrils (upto hundred) sprout from the cut fibres in all directions.
2. One of these fibrils enters the peripheral stump successfully, while the other branches disappear.

3. Schwann cells proliferate and form continuous cords of cells within the endoneurial tubes. This bridges the gap between the proximal and distal stumps, and also guides the growing filaments from the central end towards periphery.
4. The fibres grow at a rate of 1-4 mm/24 hrs towards the denervated muscle fibres due to some chemical attraction called "neurotrophism." The chemical may be released by the denervated muscle fibres and macrophages.
5. The growing filament reaches the muscle fibre and establishes contact with it and forms a functional neuromuscular junction. The formation of a junction (synapse) between axonal ending and the muscle membrane is guided by several chemicals released from both the structures.
6. Myelin sheath begins to appear in about 15 days and proceeds peripherally at a slower rate, and takes a long time (1) year) for a complete functional recovery. Final diameter attained is 80-85% of the normal.
7. Occasionally, when the regeneration fails the fibres from the central end intertwine and form an expanded mass called "neuroma." If neuroma contains sensory fibres, it is highly painful.

TYPES OF MEMORY

- Memory is defined as the retention of learned information and experiences.
- Physiologically, according to the type of information that is stored, the memory can be classified as:

1. **EXPLICIT** OR **DECLARATIVE MEMORY:** the memory of facts (sematic) and memory of events (episodic)
    - Short term memory
    - Long term memory
2. **IMPLICIT** OR **NONDECLARATIVE MEMORY**
    - Procedural memory: skills and habits
    - Priming
    - Non associative learning: habituation and sensitisation
    - Associative learning: classical and operant conditioning

EXPLICIT OR DECLARATIVE MEMORY

- Explicit Memory, (Declarative or Recognition Memory) refers to the factual knowledge of people, places or things.

- Associated with consciousness or at least awareness.
- The hippocampus and parts of medial temporal lobes – for its retention.

Episodic memory: for events

- Examples: recollection of a birthday party celebrated three days ago; events taken place while taking breakfast etc

Semantic (factual) memory:

- Knowledge of objects, facts and concepts, words and their meaning
- Form of long-term explicit memory

For example, If you think about the word alarm clock,

VISUAL MEMORY reminds us about its shape etc.

AUDITORY MEMORY reminds us about its sound.

SOMATOSENSORY MEMORY reminds us about that it is made of a plastic, glass etc.

Short term memory:

- It is the memory that lasts for seconds to hours, processing in the hippocampus and other circuit changes take place.
- Working memory is a type of short- term memory. Repeated training can lead to short term memory getting converted to long- term memory which is called **consolidation**

Long term memory:

There is storage of information for years together in long term memory. They are resistant to disruption and is broadly divided into explicit and implicit memory.

IMPLICIT OR **NONDECLARATIVE MEMORY**

- Does not involve awareness, also called as reflexive or nondeclarative memory
- For its retention there is no **processing in the hippocampus**

- **Procedural memory** includes skills and habits. refers to the information about how to perform a task. **Eg.** motor skills, habits, behavioural reflexes and the learning of certain types of procedures and rules which, once acquired, become unconscious and automatic
- **Priming** is facilitation of recognition of words or objects by prior exposure to them
- **Non-associative learning -** the organism learns about a single stimulus. It includes: Habituation and Sensitization.

On repeated stimulation, habituation produces less and less response, but sensitisation produces a greater response.

- **Associative learning -**organism learns about the relation of one stimulus to another. It includes: Classical conditioning and Operant conditioning.

APHASIA

It is defined as the loss or impairment of production and comprehension of spoken or written language due to an acquired lesion of the brain

Causes

1. Cerebral thrombosis
2. Cerebral infarction
3. Injury to the brain during accidents
4. Inadequate blood flow to the parts of the brain due to vascular changes
5. Tumors of the brain.
6. Congenital

The following faculties of the speech are affected:

1. Speech production
2. Speech comprehension
3. Writing
4. Reading

TYPES OF APHASIA:

1) Pure word blindness

2) Pure word deafness

3) Wernicke's aphasia (sensory aphasia)

4) Broca's aphasia (motor aphasia)

5) Global aphasia

1. Pure word blindness: The person is unable to read printed matter or comprehend visual signs. He cannot name the colors or objects. He is able to understand spoken words and can write and repeat on dictation. The lesion is in the visuo-psychic area or angular gyrus in the dominant hemi-sphere.

2. Pure word deafness: The person is unable to comprehend spoken word. He is unable to repeat or write on dictation. Comprehension of visual signs and written matter is normal. The lesion is present in the audito-psychic areas in the dominant cerebral hemisphere. Pure word blindness and pure word deafness is called conduction aphasia.

3. Wernicke's aphasia:

- It is also called sensory/fluent/receptive aphasia.
- Speech is fluent but totally incomprehensible due to errors in word usage, structure and tense. There is impairment of comprehension of speech and usage of paraphasic speech (malformed words used)
- The lesion is in Wernicke's area of dominant hemisphere.
- There is no lesion in the motor apparatus of speech, but written letters are often combined into meaningless words

4.Broca's aphasia (non-fluent motor aphasia):

- The lesion is in Broca's areas (45,44) of dominant hemisphere.
- Speech output is sparse, slow, poorly articulated and telegraphic.
- There is a severe writing impairment.
- Comprehension of written and spoken language is intact.

5.Global aphasia:

- The lesion around Wernicke's area involving the frontal (Broca's area), parietal, occipital and temporal lobes is responsible for this type of severe form of aphasia.
- All aspects of speech and language are impaired.
- The patient cannot read, write, repeat and has poor auditory comprehension.
- Speech output is non-fluent and is minimal

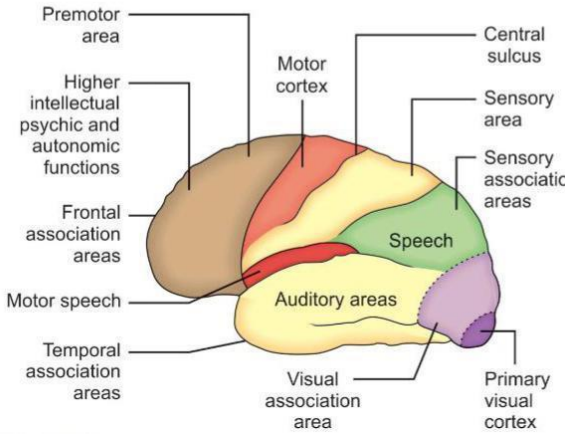
KLUVER BUCY SYNDROME

Klüver Bucy syndrome (was first described by H Klüver and PC Bucy) is experimentally induced in rhesus monkey by bilateral temporal lobectomy, particularly involving the amygdala.

- Animal exhibits placidity and inability to recognize object visually in spite of good vision **(visual agnosia)**, but will pick up almost all objects and explore them orally.
- They also show **hypersexuality and hyperphagia** (omniphagic)**.**
- The striking abnormality is to examine everything orally.
- Animal fails to ignore peripheral stimuli **(hypermetamorphosis)**, and therefore, respond to every stimulus and explore everything.
- Similar picture is observed in human beings following bilateral surgical removal of temporal lobes, cerebral atrophies and meningoencephalitis following toxo plasmosis, herpes simplex or AIDS.

FUNCTIONS OF FRONTAL LOBE

- The part of the cortex rostral to central sulcus (precentral sulcus) and medial to sylvian fissure is the frontal lobe.
- It is divided into three parts: precentral region, transitional region and prefrontal association cortex.

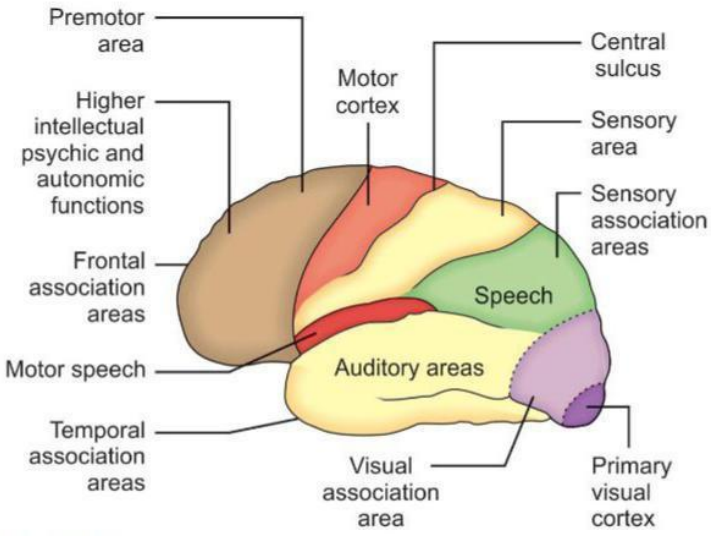


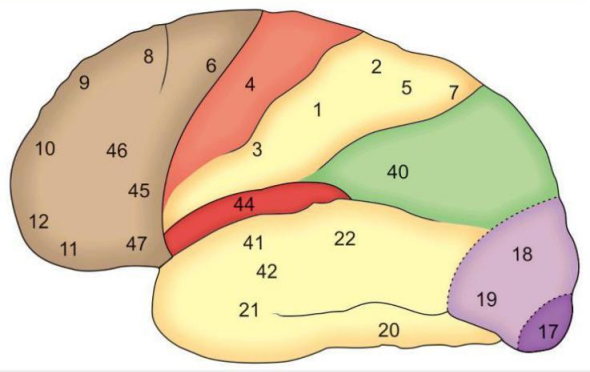
- ✓ **Precentral region:** contains primary motor cortex (area 4) and premotor cortex (area 6). These areas control movements, both skilled and postural.
- ✓ **Transitional region:** it is the area between precentral gyrus and prefrontal cortex. This includes area 44 and area 8. Area 44 is the motor speech area that controls motor activities of speech apparatus, and area 8 is the frontal eye field that controls eye movement.
- ✓ **Prefrontal Association Cortex**: (Prefrontal Lobe) includes area 9–14. This is subdivided into orbital region (area 11–14 and 47), and dorsolateral region (area 9, 10, 45, and 46). Area 24 of prefrontal lobe (in cingulated gyrus) is a part of Papez circuit. Orbital region

is connected with temporal lobe, olfactory cortex and limbic structures. The dorsolateral region receives inputs from various sensory modalities that include visual inputs and auditory inputs. The prefrontal cortex provides powerful neocortical connections to basal forebrain structures including hypothalamus that are involved in control of visceral functions and emotional behaviours. Prefrontal cortex is the seat of human personality.

FUNCTIONS OF PARIETAL LOBE

- ✓ Parietal lobe lies posterior to the central sulcus and rostro dorsal to lateral sulcus (sylvian fissure). It is divided into two regions:
- ✓ The anterior region that mainly contains primary somatosensory cortex (SI), i.e. the Brodmann's area 3, 1, and 2.
- ✓ The posterior region that contains other sensory areas, which is considered to be the association sensory cortex. This includes the area 5 and 7 in the upper part, and area 39 and 40 in the lower part.



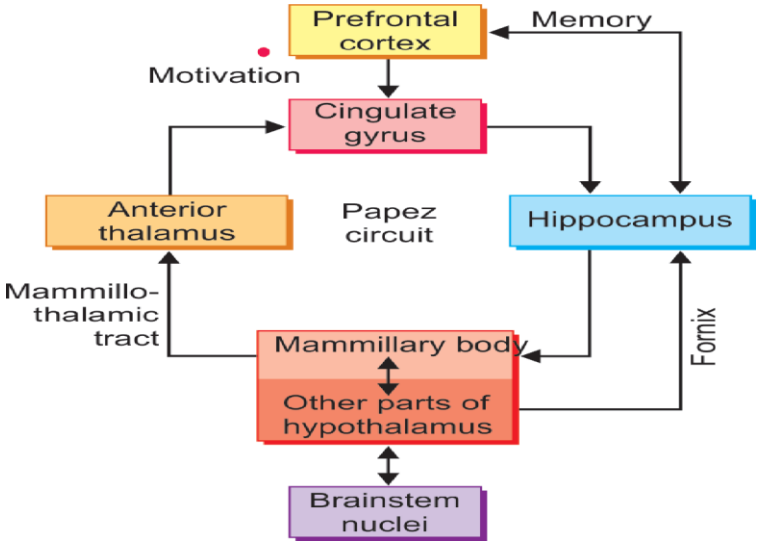


Functions

- Area 3, 1 and 2 are meant for perception of sensations, especially the cortical sensations (stereognosis, tactile localization and two-point discrimination), fine touch, proprioception and vibration
- Area 5 is more involved in processing of somatosensory information to produce movement.
- Area 7 primarily processes visual information in order to produce not only movement, but also arousal, attention and emotion.

PAPEZ CIRCUIT

Papez circuit is a fundamental component of the limbic system. It is a closed neural circuitry that starts and ends in the hippocampus. It plays an important role in genesis and control of emotion.



- The major circuit connects hippocampus to the mamillary body of the hypothalamus, the hypothalamus to the anterior thalamic nuclei via the mammillothalamic tract and the anterior thalamus to the cingulate gyrus by thalamic projections.
- The circuit is complete by the cingulate gyrus projecting to the hippocampus

Functions: information about learning and memory from cortex, especially from the prefrontal cortex is referred to the limbic system through the cortical hippocampal connections