



# **CNS revision (Part 2)**

**By**

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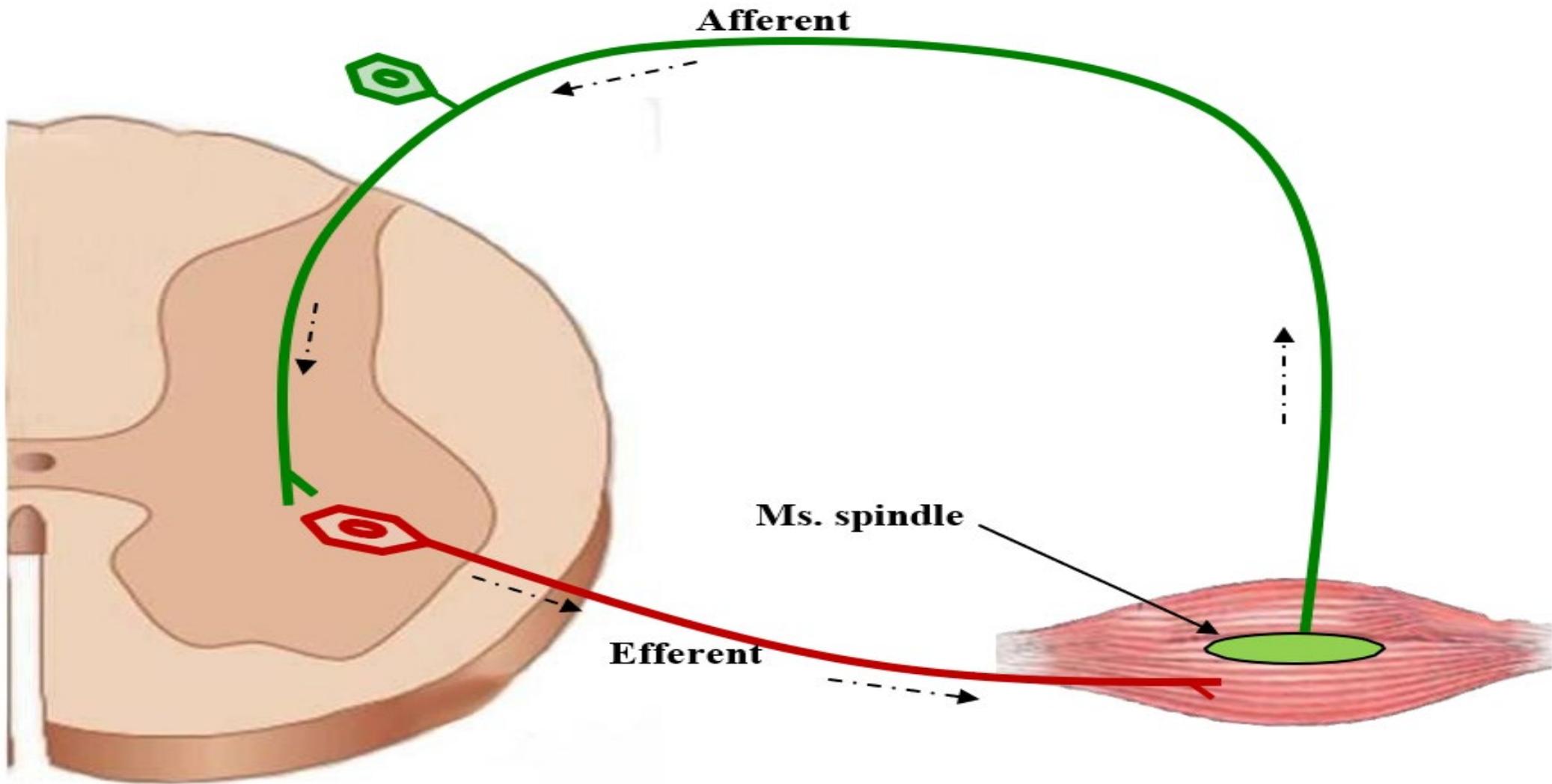
**Professor of Medical Physiology**



# Stretch reflex

# Stretch reflex

- Def of stretch reflex: It is a reflex contraction of a skeletal muscle when it is passively stretched
- Pathway of stretch reflex:
  - Stimulus: Passive **stretch** of the muscle
  - Receptor: Muscle spindle
  - Afferent: **A- $\alpha$**  & **A- $\beta$**  (type Ia & II) nerve fibers
  - Center: A.H.Cs ( $\alpha$  motor neurons)
  - Efferent:  $\alpha$ -motor nerves
  - Response: contraction of the muscle



Pathway of stretch reflex

## □ Receptor of stretch reflex (muscle spindle):

### • Def of muscle spindle:

- Specialized fusiform capsulated structures located in the fleshy part of the muscles.
- They act as length receptors i.e. detect the changes in the muscle length.
- They are parallel to the extrafusal muscle fibers.

- Structure of muscle spindle:

- Each muscle spindle consists of 4-12 modified muscle fibers called intrafusal muscle fibers.

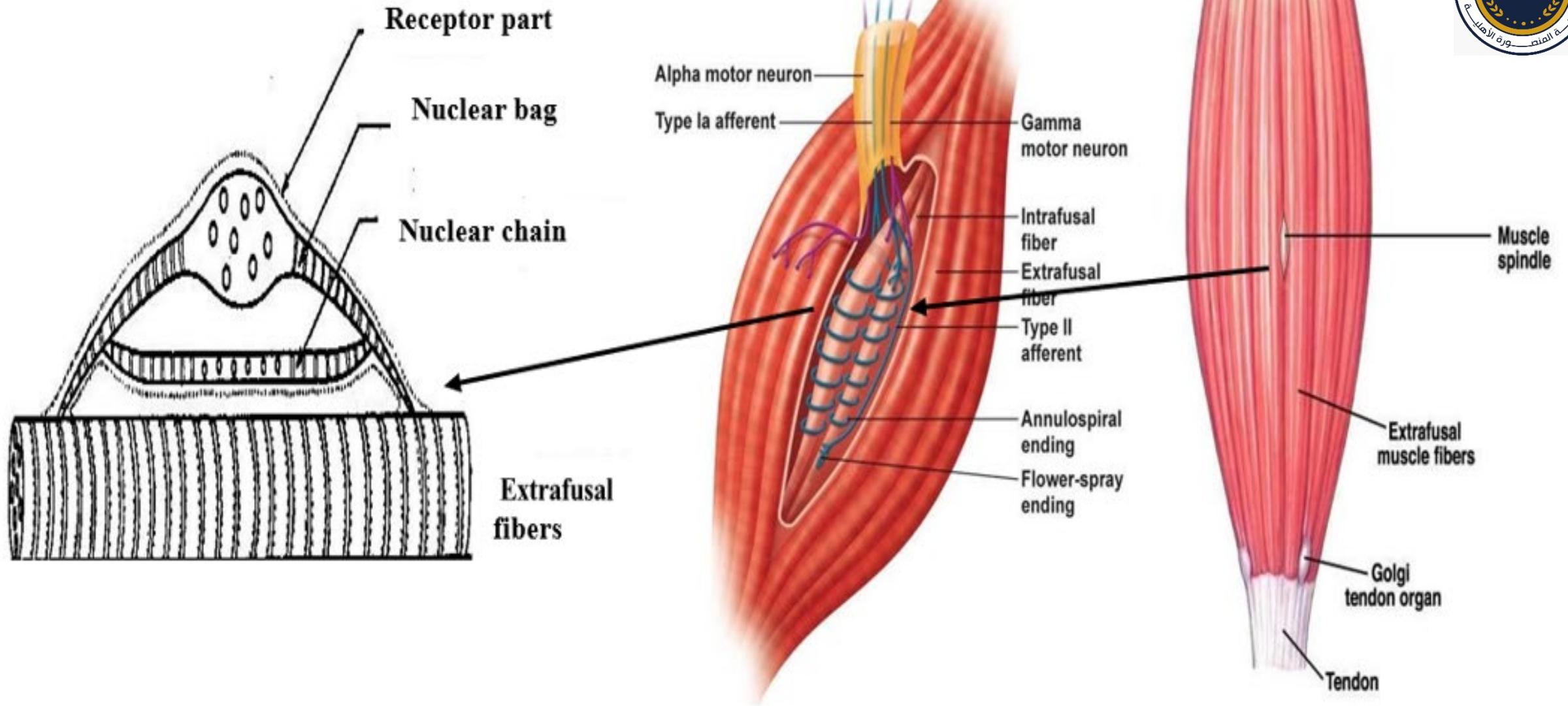
- Intrafusal muscle fibers:

- Each intrafusal muscle fiber consists of 2 parts:

i) central receptor area	ii) peripheral contractile parts
From which the <b>afferent</b> nerve endings arise.	Which can <b>contract</b> and produce stretching of the <b>central receptor area</b> .

▪ Types of intrafusal muscle fibers:

	Nuclear bag fibers	Nuclear chain fibers
<b>Number</b>	<b>1-2</b> in each spindle	<b>5-8</b> in each spindle
<b>Length</b>	<b>Long</b> fibers	<b>Short</b> fibers
<b>Central part</b>	<b>dilated</b> and contains the nuclei i.e. “like a <b>bag</b> ”.	<b>Straight</b> , and the nuclei arranged as a <b>chain</b> .



Innervation of muscle spindle

• Innervation of muscle spindle:

A. Afferent innervation of muscle spindle:

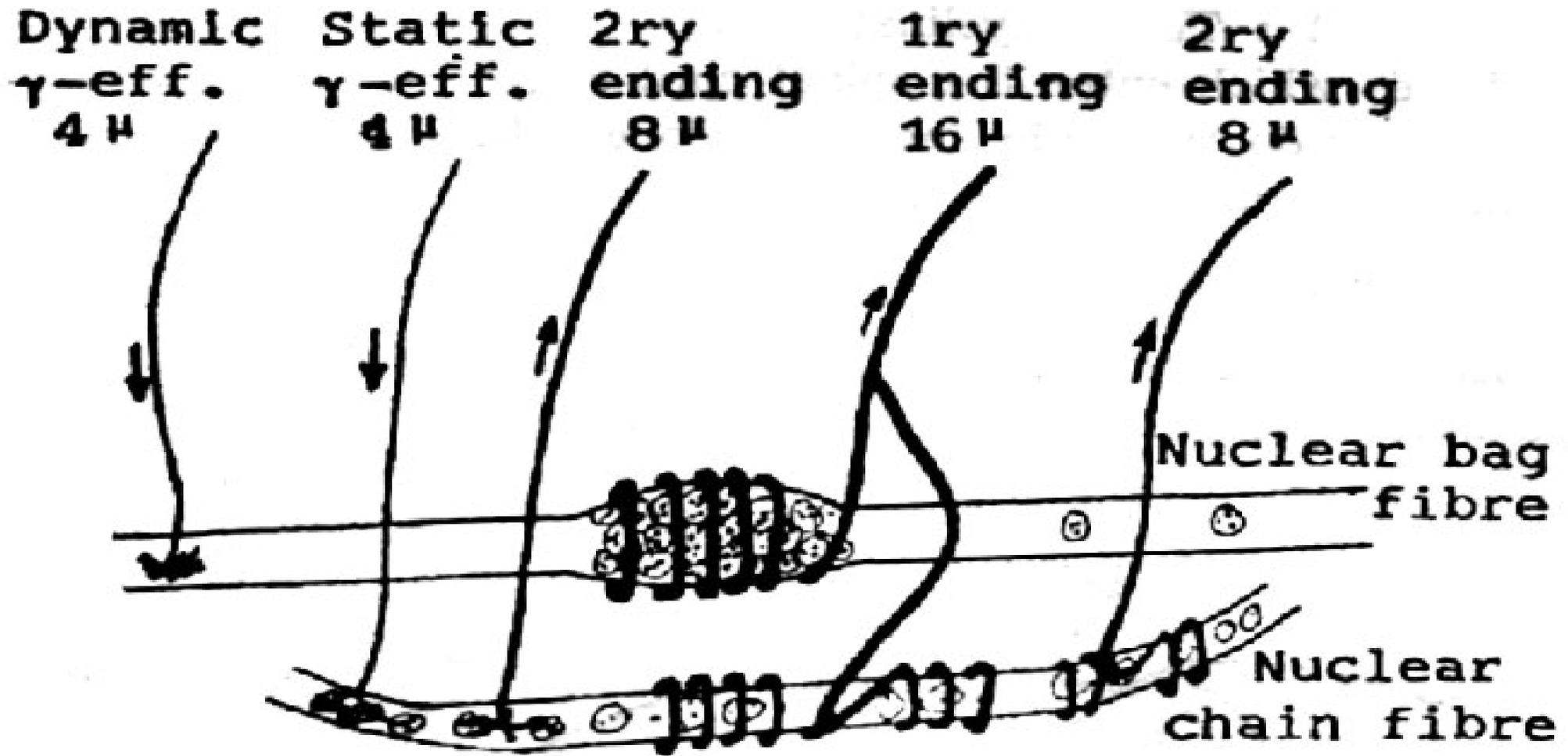
	Primary afferent (Ia)	Secondary afferent (II)
<b>Give rise to:</b>	thick myelinated rapidly conducting type A $\alpha$ ( <b>type Ia</b> ) fibers.	thin myelinated less rapidly conducting type A $\beta$ ( <b>type II</b> ) fibers.
<b>Its ending:</b>	Arise from central receptor area of <b>both nuclear bag &amp; nuclear chain.</b>	Arise from central receptor area of <b>nuclear chain fibers only</b> on both sides of 1ry endings.
	Annulospiral	Flower spray



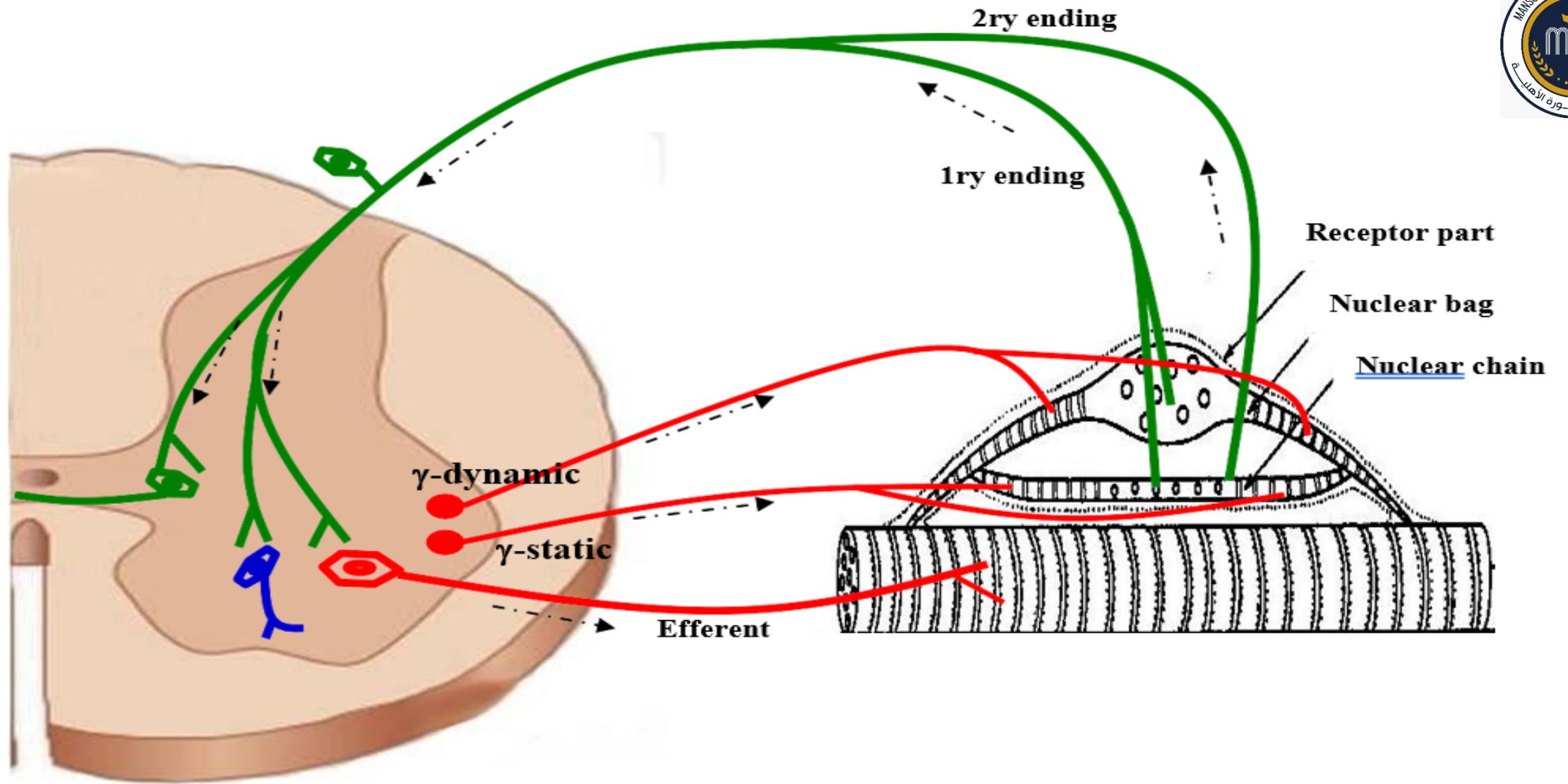
	Primary afferent (Ia)	Secondary afferent (II)
<b>Response &amp; adaptation:</b>	Rapidly responding & rapidly adapting.	Slowly responding & slowly adapting.
They are stimulated by stretch of the central receptor area		

## B. Efferent innervation of muscle spindle:

	<u><math>\gamma</math>-dynamic nerve fibers:</u>	<u><math>\gamma</math>-Static nerve fibers:</u>
<b>Supply</b>	the peripheral contractile parts of the <b>nuclear bag</b> fibers.	the peripheral contractile parts of the <b>nuclear chain</b> fibers.



**Innervation of the muscle spindle.**



Innervation of muscle spindle

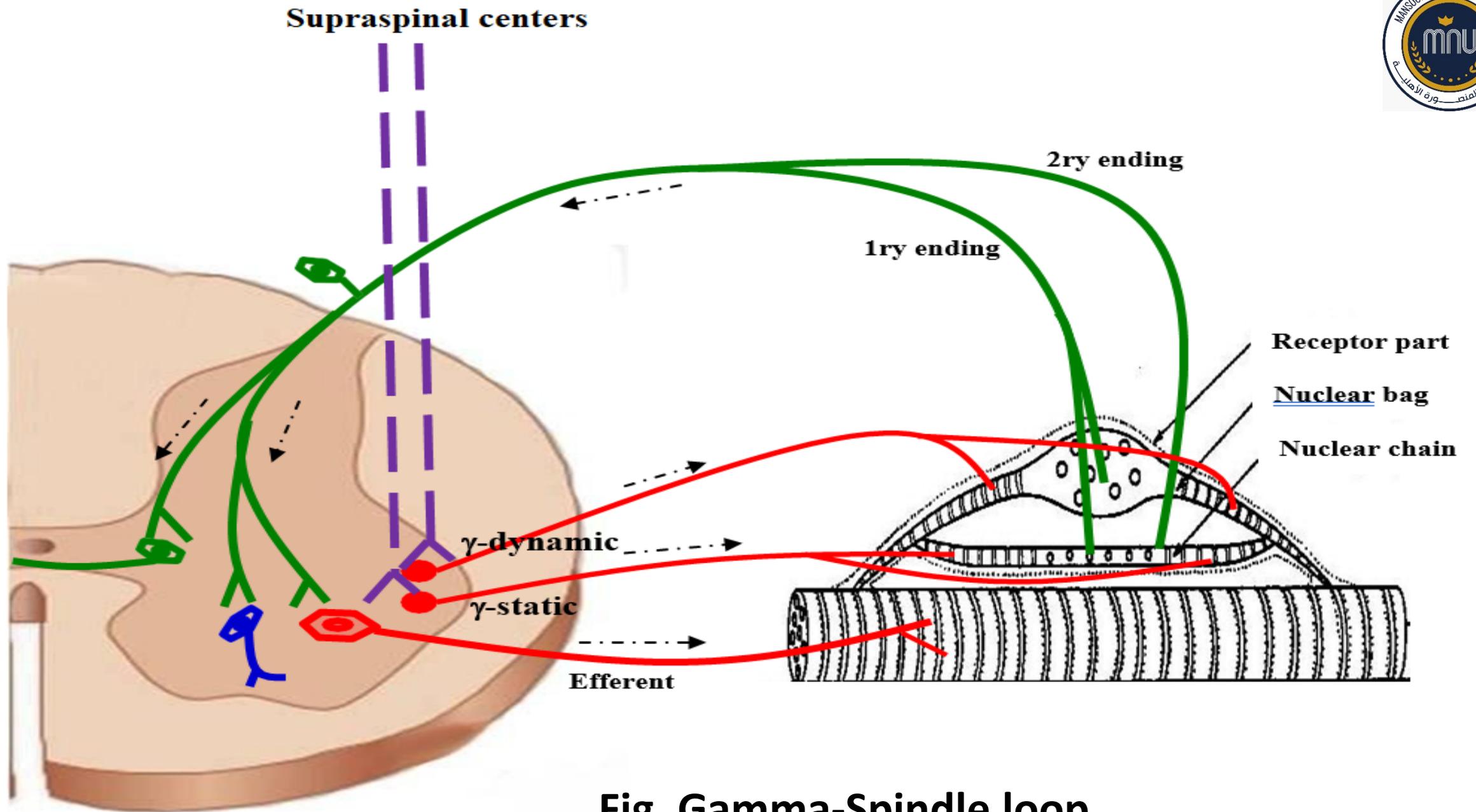


- **Methods of stimulation of muscle spindle:**

- 1) **Direct passive stretching** of the muscle.

- 2) **Stimulation of the efferent  $\gamma$ -motor neurons:**

**Stimulation of  $\gamma$ -motor neurons by supraspinal facilitatory impulses.** → contraction of peripheral contractile regions of intrafusal MFs → stretch of the central receptor area → discharge of impulses through 1ry & 2ry endings → stimulation of  $\alpha$  MNs → muscle contraction (**Gamma-spindle loop**).



**Fig. Gamma-Spindle loop**



## □ Types of stretch reflex:

	Dynamic stretch reflex	Static stretch reflex
<b>Stimulus</b>	Sudden stretch	Maintained stretch of the muscle
<b>Receptor</b>	Nuclear bag intrafusal muscle fiber	Nuclear chain intrafusal muscle fiber
<b>Afferent</b>	Type A- $\alpha$ (type Ia) nerve fibers (1 <sup>ry</sup> afferent)	Type A- $\beta$ (type II) nerve fibers (2 <sup>ry</sup> afferent) (Mainly).
<b>Center</b>	$\alpha$ -motor neurons	$\alpha$ -motor neurons
<b>Efferent</b>	Axon of $\alpha$ -motor neurons	Axon of $\alpha$ -motor neurons
<b>Response</b>	Rapid contraction followed by relaxation	Continuous contraction as long as the stretch is maintained
<b>Example</b>	Tendon jerks	Muscle tone

# □ Functions of stretch reflex: (Functions of muscle spindle):

## 1) Generation of muscle tone:

- **Muscle spindles**, through the **stretch reflex**, are responsible for generation of **skeletal muscle tone**.
- CNS adjusts muscle tone by varying the level of  **$\gamma$ -motor neurons** activity which by its turn affect the **frequency of spindle sensory discharge**.



## 2) Smoothing of muscle contraction (Damping function):

- Is the ability of muscle spindle to **prevent oscillation and jerkiness** of body movements.
- **Signals** are sent from brain to motor neurons controlling muscles in an **unsmooth form**, ( $\uparrow$  in intensity for a few milliseconds, then  $\downarrow$  in intensity for a few milliseconds & so on).
- **However**, muscles oscillations or jerkiness **does not occur** due to presence of muscle spindle.

### 3) Load Reflex:

- **Def:** It is a reflex that is responsible for keeping the hand or foot in position when a **moderate or heavy load** is applied.
- **e.g.** when a person holding up a cup while someone is filling it with tea, as the load gets bigger and bigger, the force required to keep the hand in position must be continually increased.

### 4) Proprioceptive functions:

- Muscle spindles provide **proprioceptive information** to the brain and cerebellum for keeping them continually informed about muscle length and changes in that length.

# □ Inverse stretch reflex (Golgi tendon reflex) (lengthening reaction):

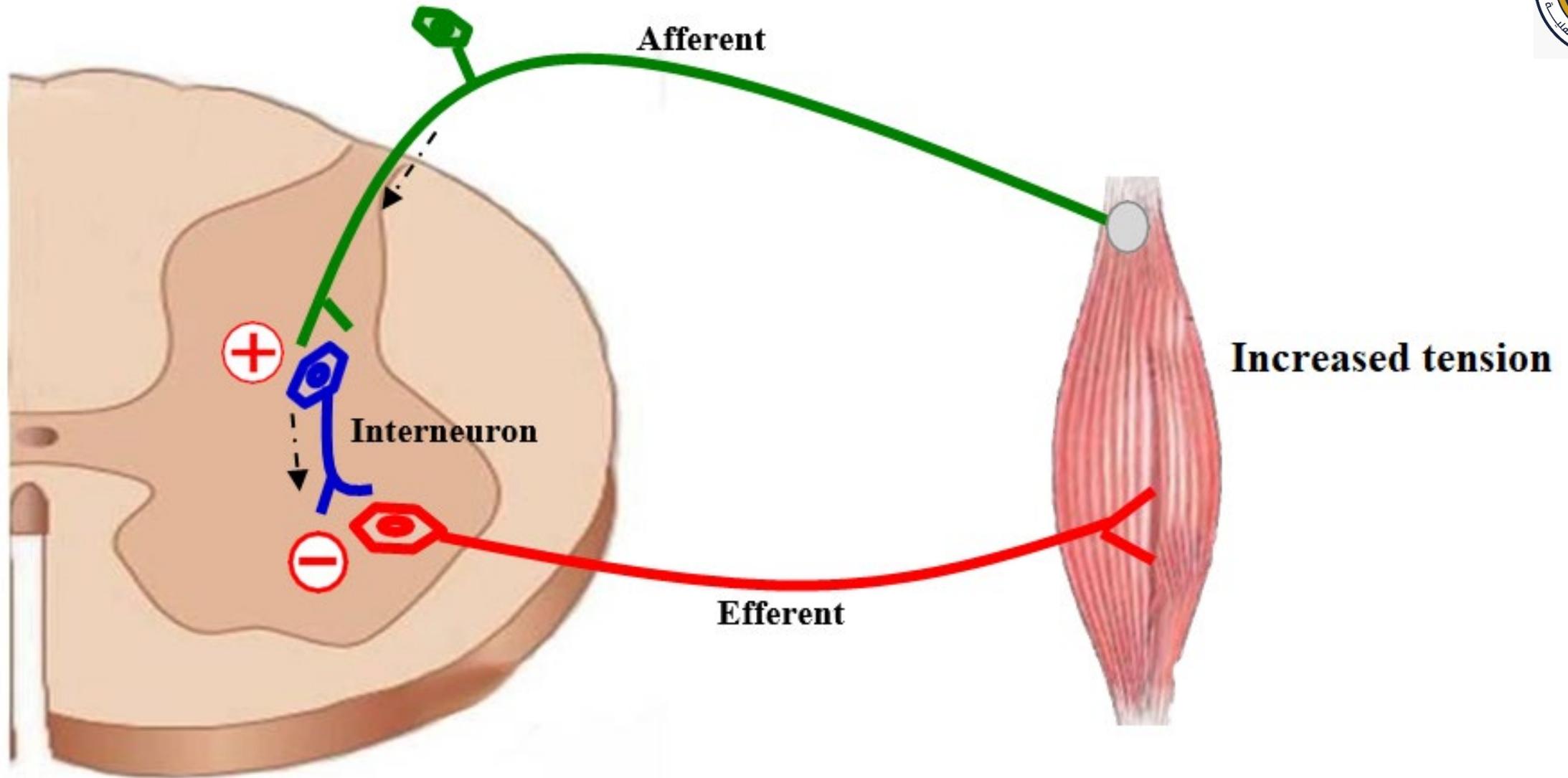
**\* Def:** Reflex relaxation of a contracting skeletal muscle (lengthening) when it is exposed to excessive stretch (overstretch) or severely contracted.

**\* Pathway:**

- ✓ **Stimulus:** Increased tension in the tendon of the muscle due to:
- i) Severe contraction OR
  - ii) Overstretch.



- ✓ **Receptors:** Golgi tendon organs (GTO):
  - Present in the **tendon of the muscle.**
  - Each organ consists of 6-20 modified fibers mostly **elastic fibers.**
  - Stimulated by **↑ tension** in the tendon “**Tension Receptors**”.
- ✓ **Afferent:** A- $\alpha$  (Ib) nerve fibers.
- ✓ **Center and response:** The impulses from GTO inhibit the A.H.Cs via interneurons → relaxation of the muscle.
- \* **Significance:** it is a **protective** reflex which prevents:
  - a) Muscle tearing.
  - b) Tendon avulsion



**Figure : inverse stretch reflex |**

# Stretch reflex and inverse stretch reflex



	Stretch reflex	Inverse stretch reflex
<b>Type</b>	Monosynaptic	Di-synaptic
<b>Receptors</b>	Muscle spindles	Golgi tendon organs
<b>Stimulus</b>	Muscle stretch (↑ muscle length).	↑ muscle tension due to excessive muscle stretch or severe contraction.
<b>Effect</b>	Muscle contraction	Muscle relaxation
<b>Functions</b>	1- Generation of muscle tone 2- Damping function. 3-Load reflex. 4-proprioceptive functions.	Prevention of: 1-Muscle tear 2-Tendon avulsion.

	<b>Muscle spindle</b>	<b>Golgi tendon organ</b>
<b>Found in</b>	fleshy part of muscle	tendons of muscle
<b>Formed of</b>	intrafusal muscle fibers	modified fibers mostly elastic fibers “number 6-20”.
<b>Stimulated by</b>	muscle stretch	↑ muscle tension
<b>Its stimulation elicits</b>	stretch reflex.	inverse stretch reflex.
<b>Effect on muscle</b>	muscle contraction	muscle relaxation
<b>Afferent:</b>	A $\alpha$ (Ia) & A $\beta$ (II) nerve fibers	A- $\alpha$ (Ib) nerve fibers

# Skeletal muscle tone



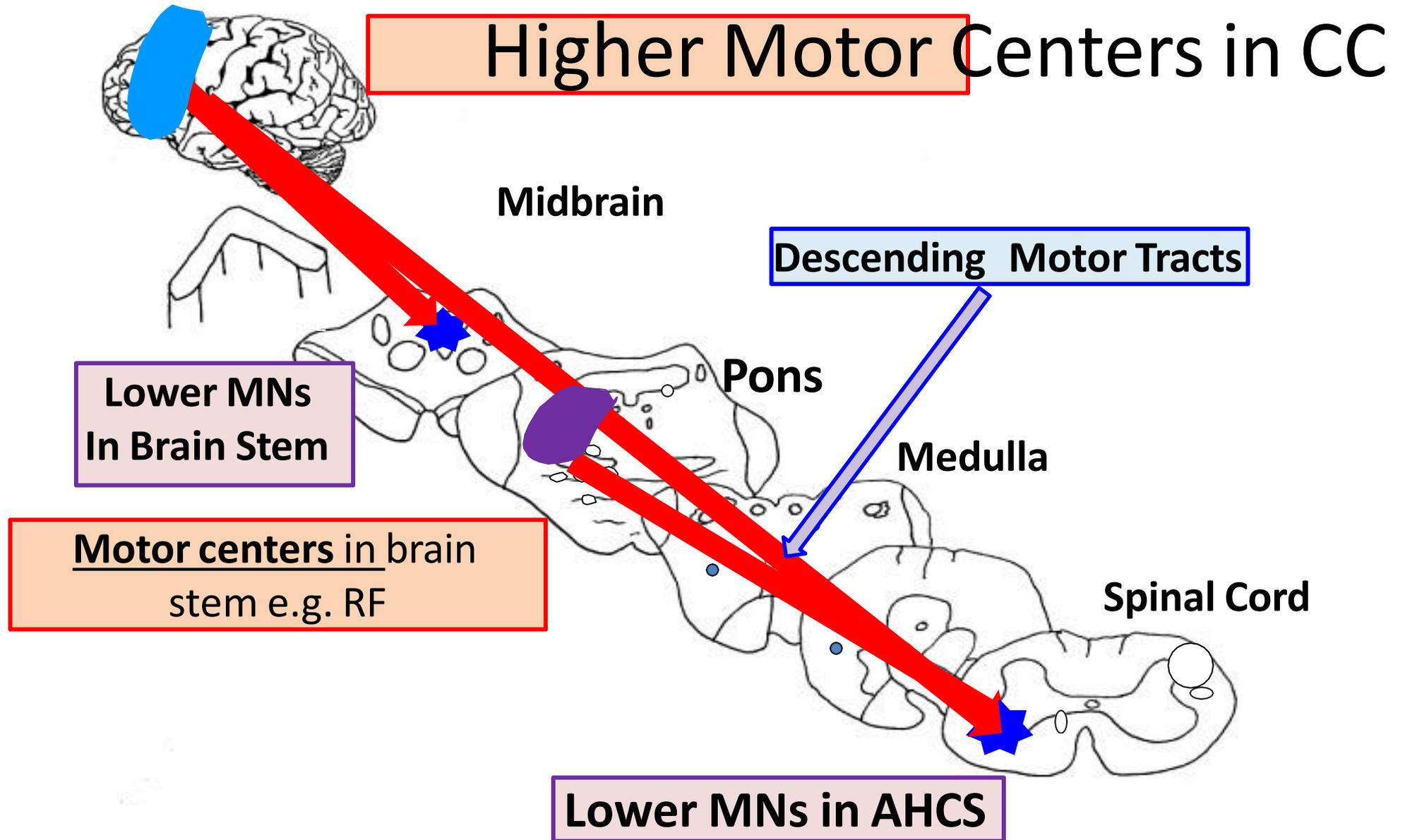
- **Def:** It is a state of continuous partial (Subtetanic) contraction present in all skeletal muscles during rest.
- **Pathway:** Static stretch reflex
- **Distribution:**
  - The muscle tone is more prominent in the antigravity muscles (extensors of the lower limb, flexors of the upper limb, extensors of the back and neck, anterior abdominal wall muscles, and elevators of the lower jaw).
  - This occurs because these muscles are exposed to more stretch under the effect of gravity.



- Functions:

- 1) It is the basic mechanism for **postural reflexes which regulate the body posture and equilibrium.**
- 2) Helps production and maintenance of **body temperature.**
- 3) Helps **venous and lymphatic return.**
- 4) **Keeps viscera** in position and prevents visceroptosis

# Somatic Motor System



# Upper Motor vs Lower Motor Neurons

- Consists of 2 sets of motor neurons;

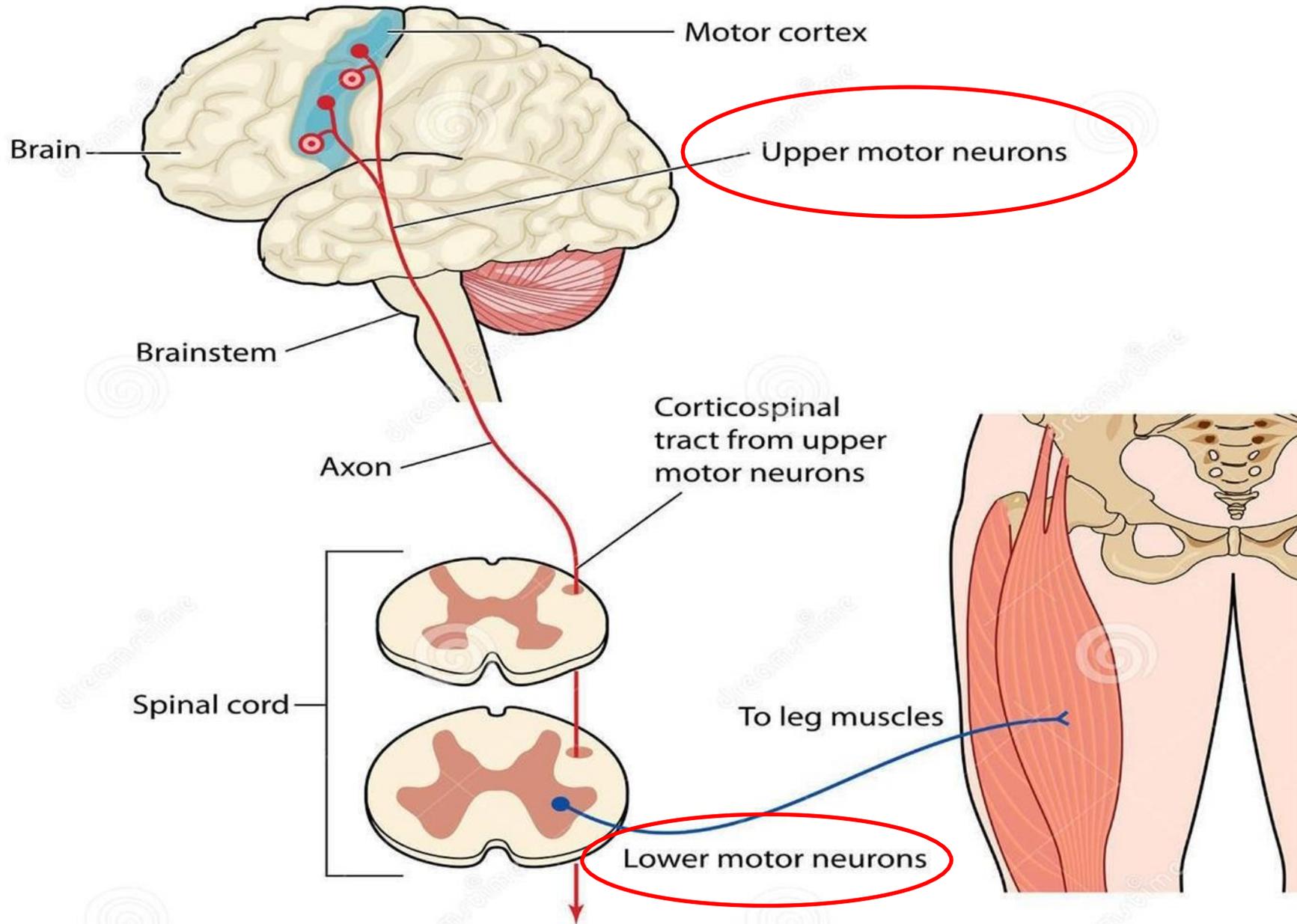
## 1. Upper Motor neurons (UMNs):

- Their **cell bodies** are present in **cortical motor areas** of cerebral cortex and other **motor centers** in brain stem
- Their **axons** form **descending motor tracts**

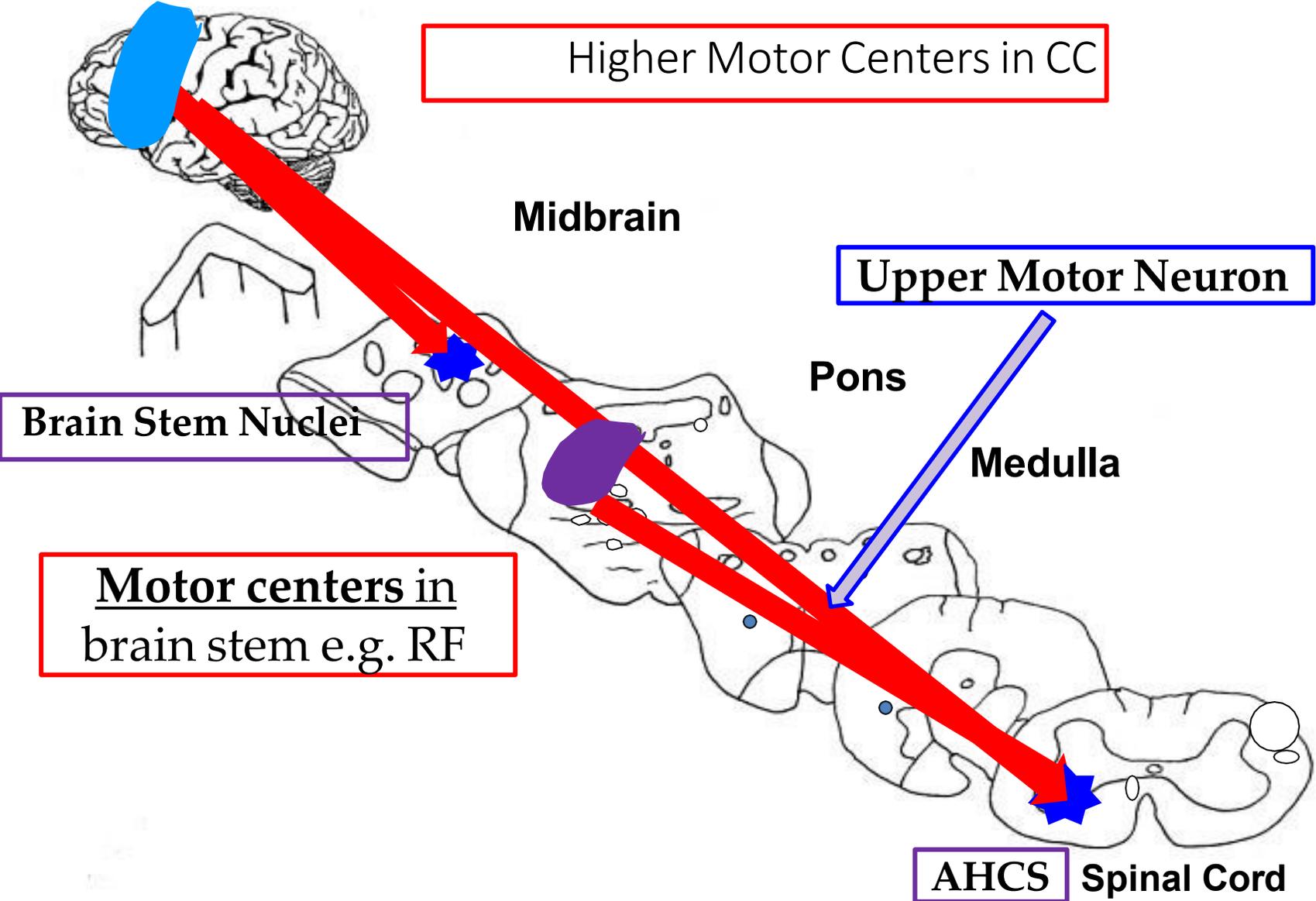
## 2. Lower motor neurons (LMNs):

- Their **cell bodies** are present in **AHCs** of spinal cord or **motor nuclei** of cranial nerves
- Their **axons** form peripheral nerves that supply **skeletal ms**

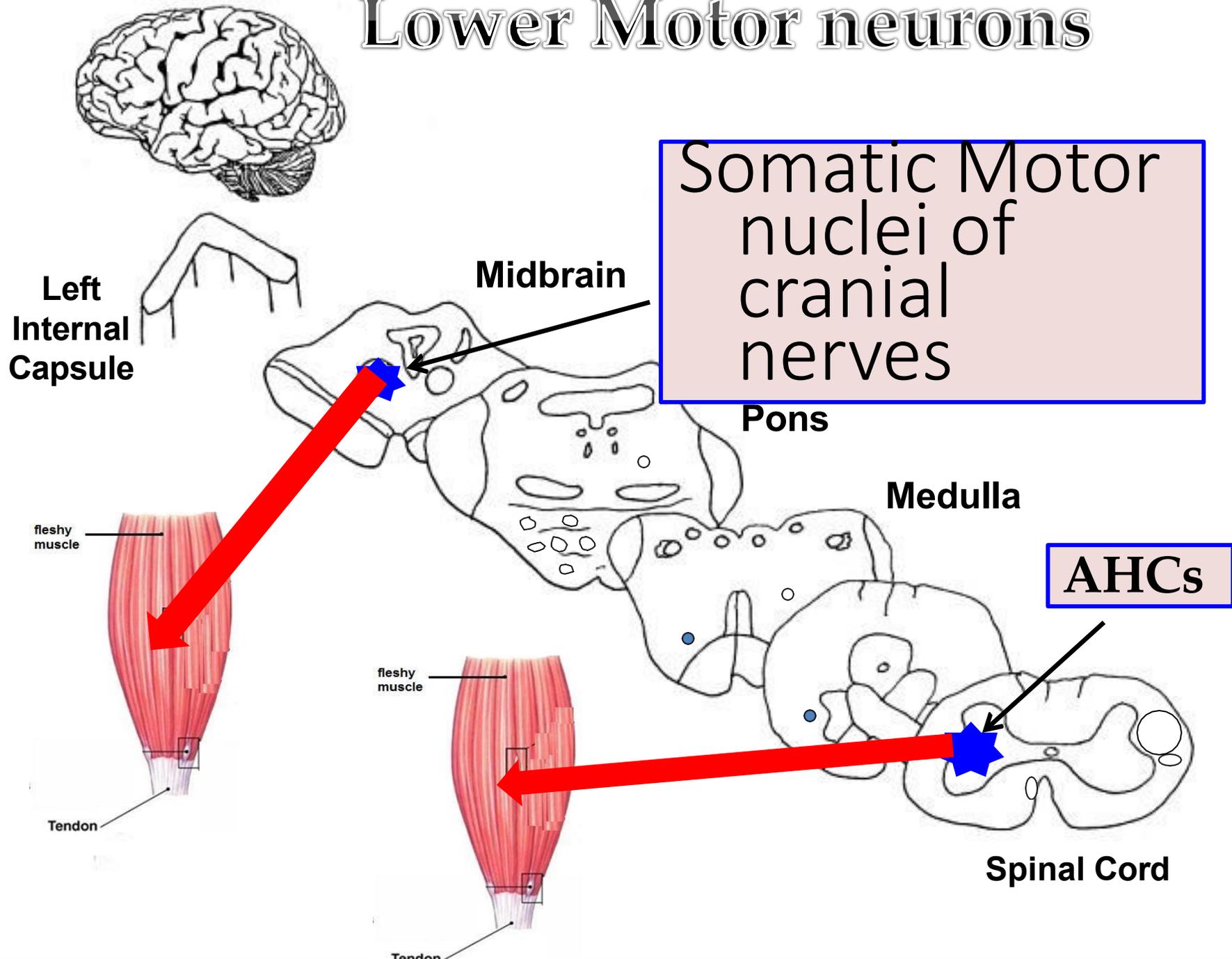
# UMN vs LMN



# Upper Motor Neuron



# Lower Motor neurons



# Motor Areas

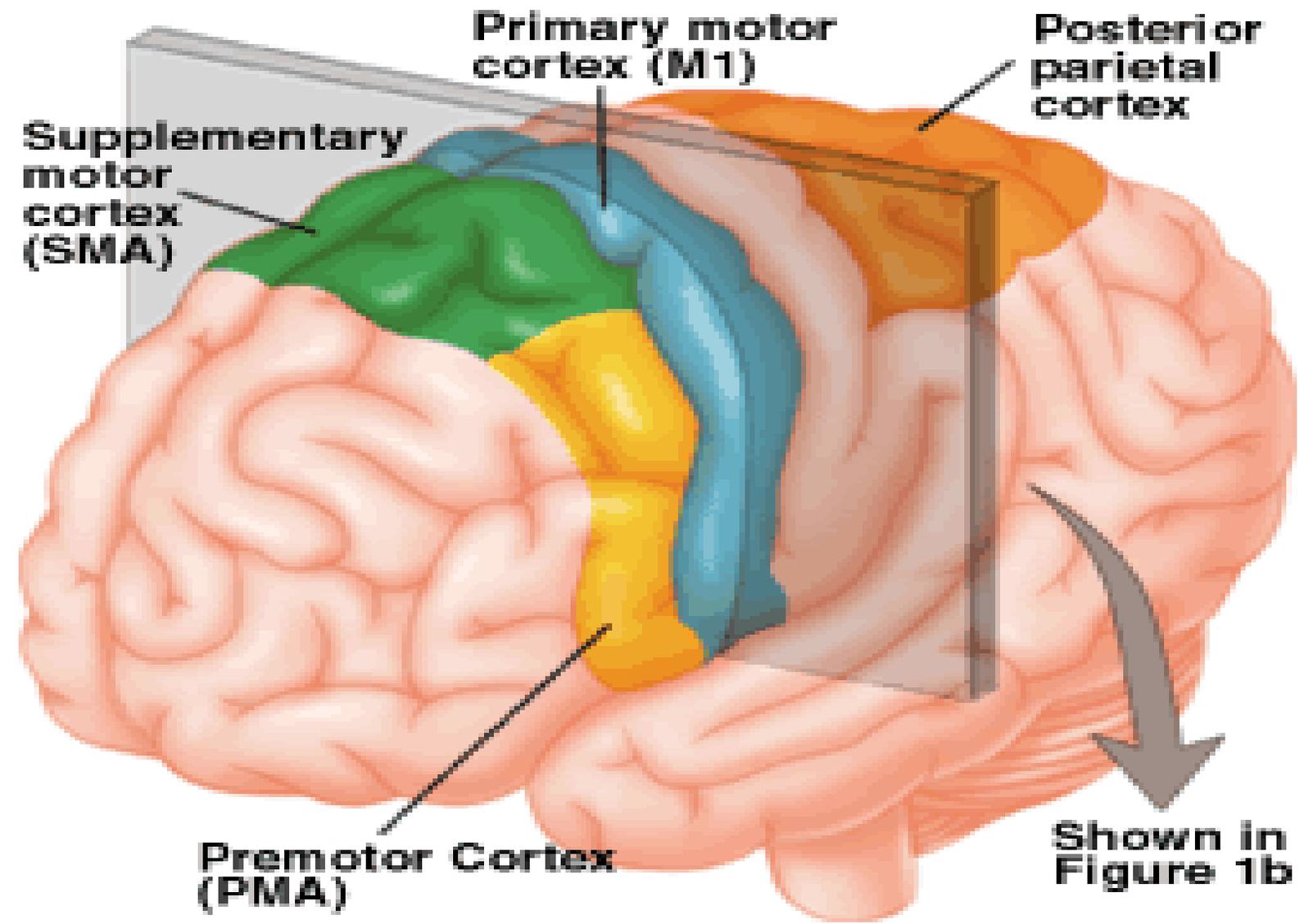
- **Initiation and performance** of voluntary movements result from **motor** signals from the **motor cortex** to the **lower motor centers** via the **descending motor tracts**
- **Motor cortex** is located in the **frontal lobe**, and comprises;

1. Primary motor area (area 4)

2. Premotor area (area 6)

3. Supplemental motor area

# Motor Areas



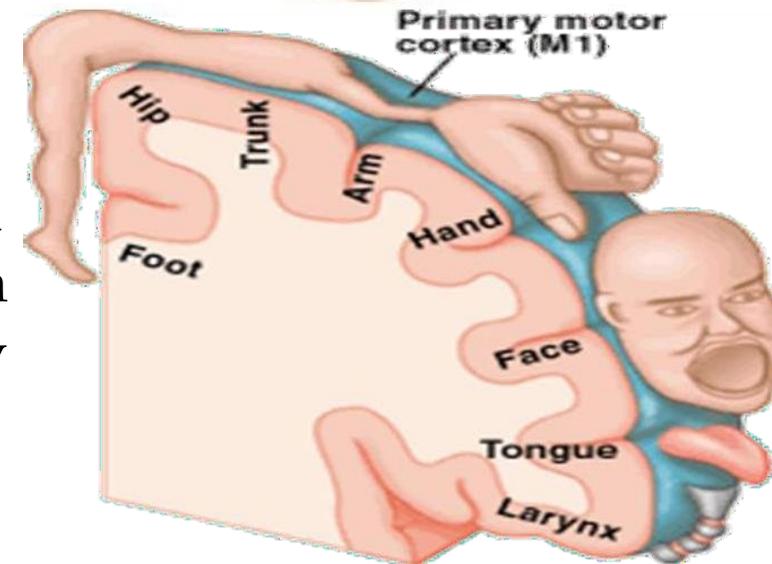
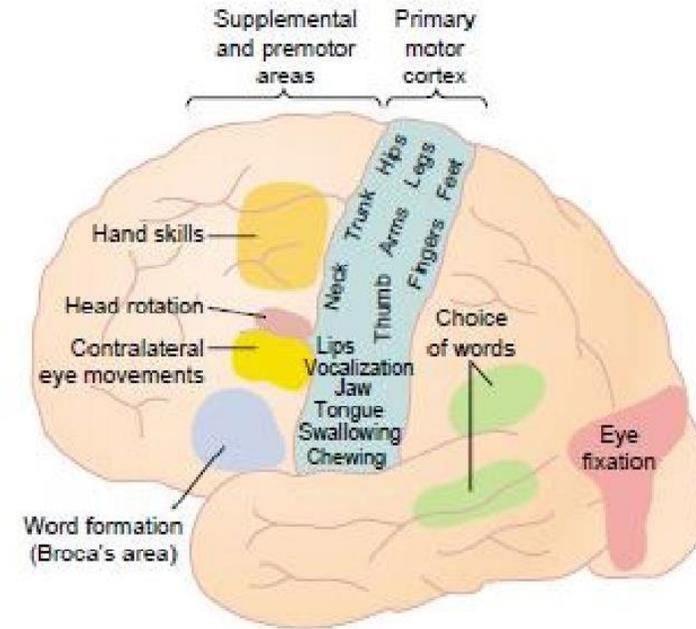
# Primary Motor Area (4)

Site:

- Precentral gyrus in frontal lobe

## Body representation:

1. **Contralateral (crossed)** : i.e. body ms from the Rt side of the body is represented in the left hemisphere and Lt half of body is represented in Rt hemisphere
2. **Inverted**
3. The size of the represented part in the cortical areas depends on complexity of movements done by this part



# Primary Motor Area (4)

Functions:

**1. Initiation of voluntary movements done by the distal limb muscles**

**2. Facilitates stretch reflex and the tone of the distal muscles**

# Primary Motor Area

Lesion:

1. loss of fine skilled movements in one limb (monoplegia) on the opposite side

2. Hypotonia and weak tendon jerks

3. Babinski's sign with dorsiflexion of the big toe only

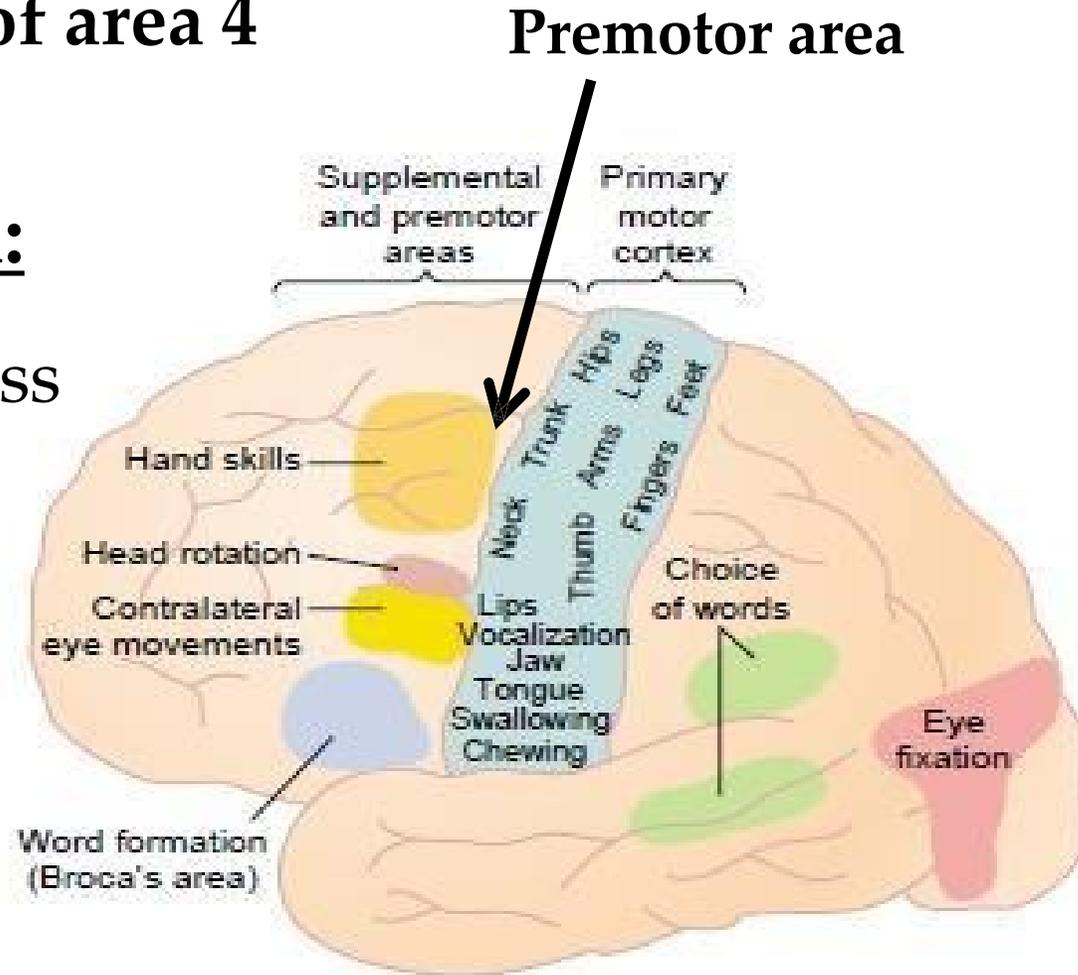
# Premotor Area (6)

## Site:

- Frontal lobe in front of area 4

## Body representation:

- As area 4 but less excitable



# Premotor Area

Functions:

1. Initiation of gross movements as those done by the trunk

2. Shares in planning of complex movements together with area supplemental motor area

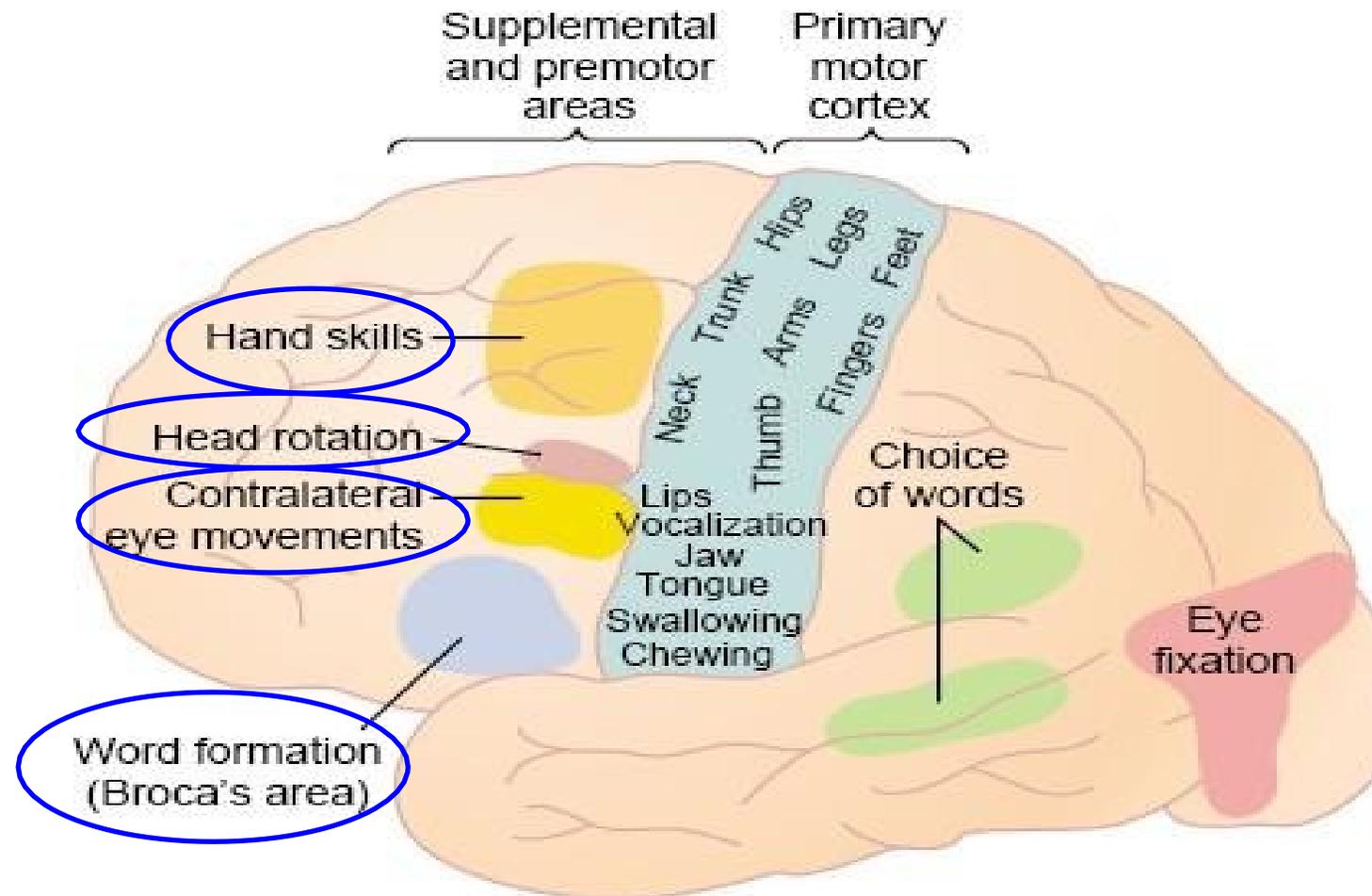
3. With basal ganglia, involved in postural adjustment during voluntary distal movements

4. Inhibitory effect on muscle tone

5. With basal ganglia, initiate & control the automatic associative movements e.g. swinging of arms during walking

# Premotor Area

Functions: contains special areas



# Premotor Area

Lesion:

1. Impairment of complex movements or paresis

2. Hypertonia and exaggerated tendon jerks

3. Babinski's sign with fanning of the outer toes

4. Loss of automatic or associative movements

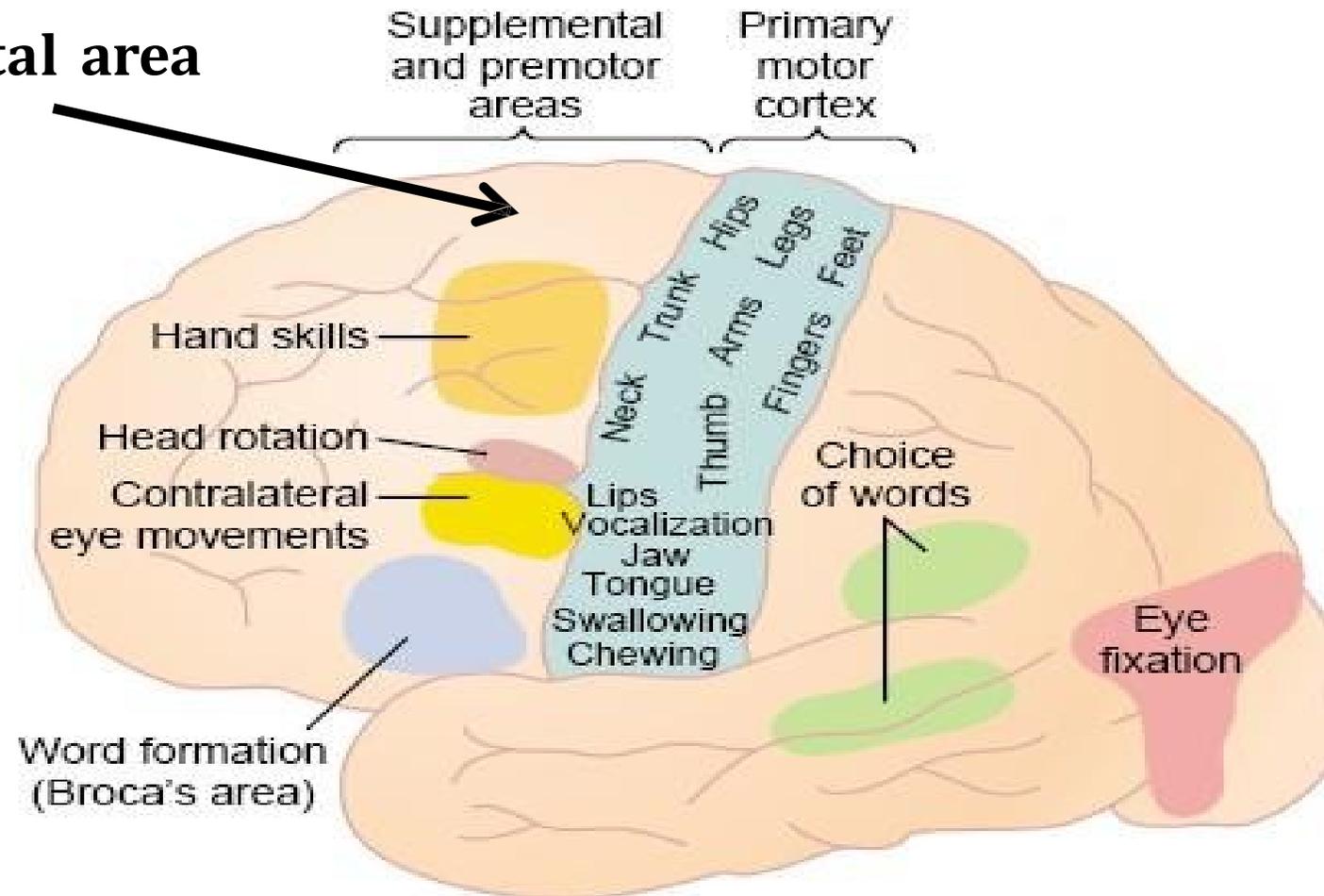
5. Motor aphasia and motor apraxia

# Supplemental Motor Area

Site:

- Upper medial side of frontal lobe above area 6

## Supplemental area

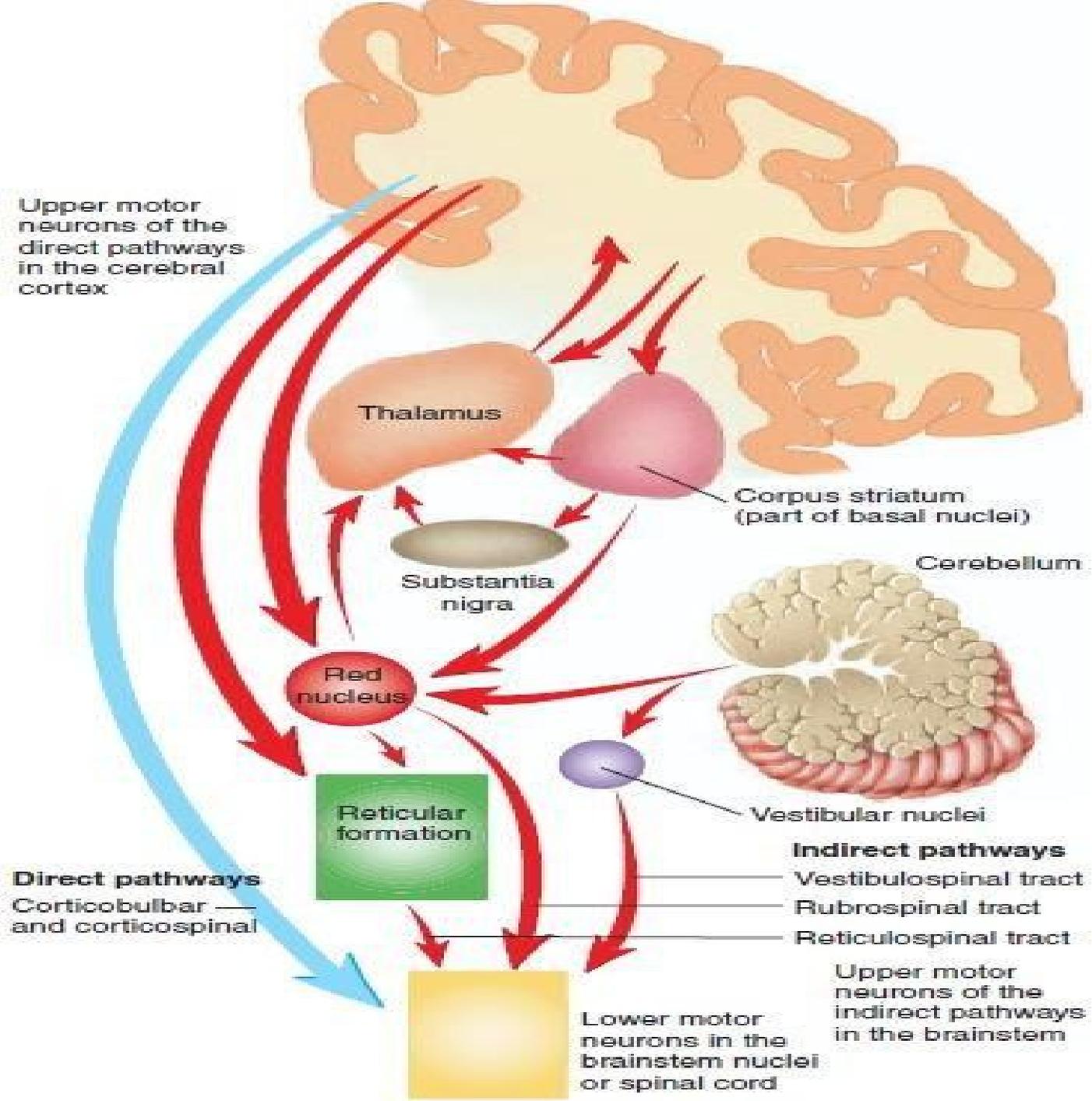


# Supplemental Motor Area

## Functions:

- 1) With the premotor area (6) in provide postural background for the performance of the fine skilled movements by hands and fingers
- 2) **It functions with the premotor area (6) in providing suitable background** for the performance of the fine skilled movements by hands and fingers that are mediated by the C.B.S tract.
- 3) Shares in the planning and programming of the complex movements with area 6.

# Descending Motor Tracts



# Descending Motor Tracts

Classified by **2 ways** ;

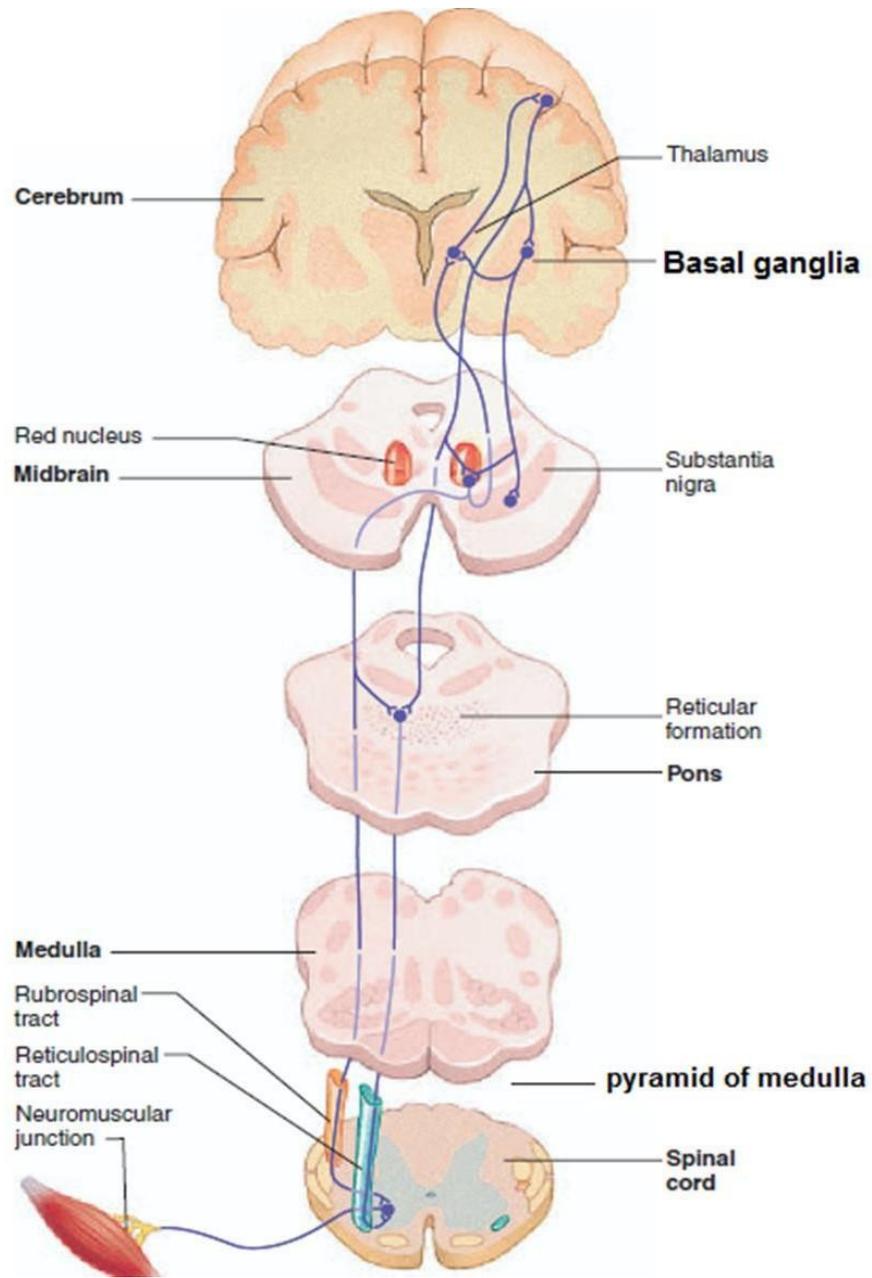
## a) Pyramidal and extrapyramidal tracts:

- If the tract **passes through** the **pyramids** of the medulla → **pyramidal tract.**
- If the tract **does not pass** through the **medullary pyramids** → **extrapyramidal tract**

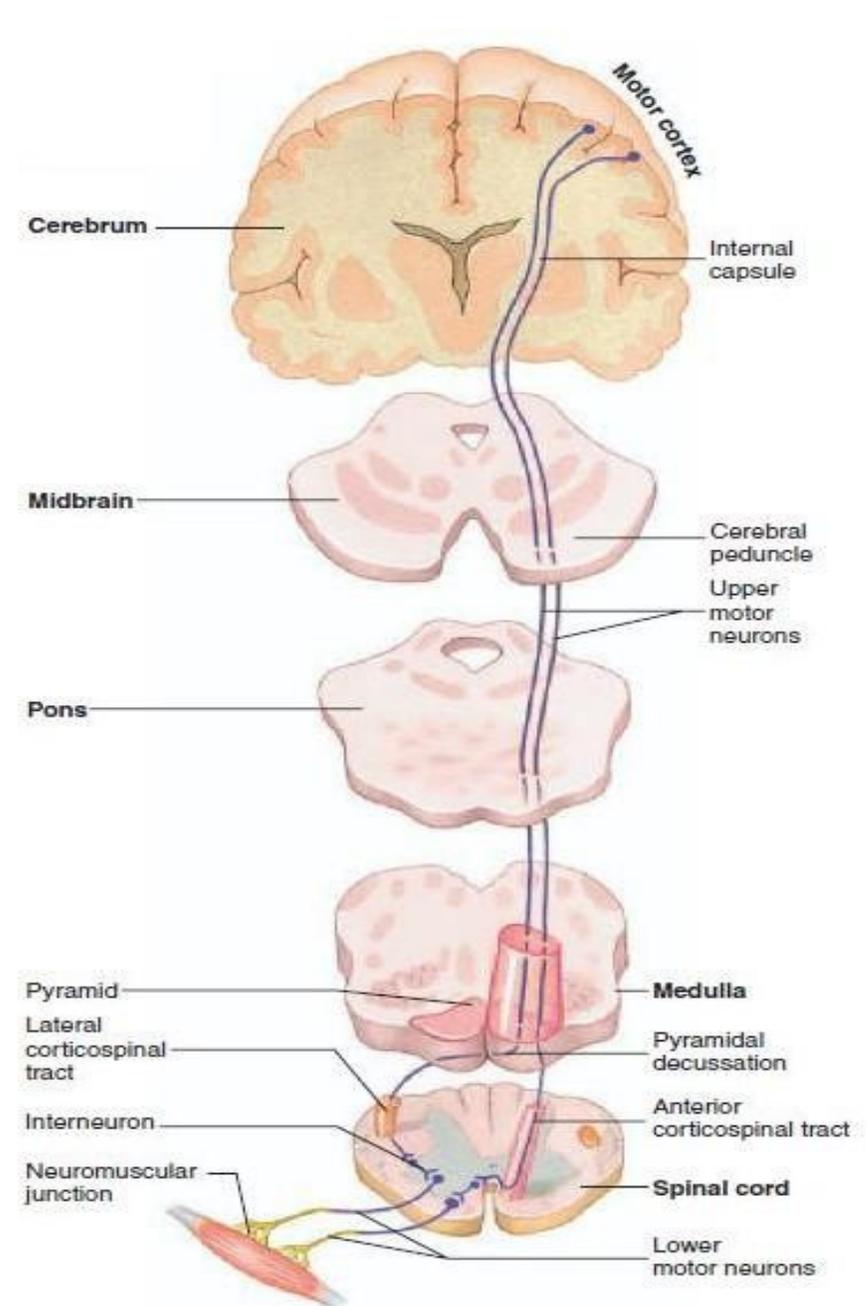
## b) Medial and lateral motor system:

i) **Medial motor system** → include tracts that terminate primarily on the **ventromedial AHCs neurons** → innervate **axial and girdle ms** → concerned with **postural control.**

ii) **Lateral motor system** → include tracts that terminate primarily on the **lateral AHCs neurons** → innervate **distal ms of the limbs** → concerned with control of **fine voluntary**

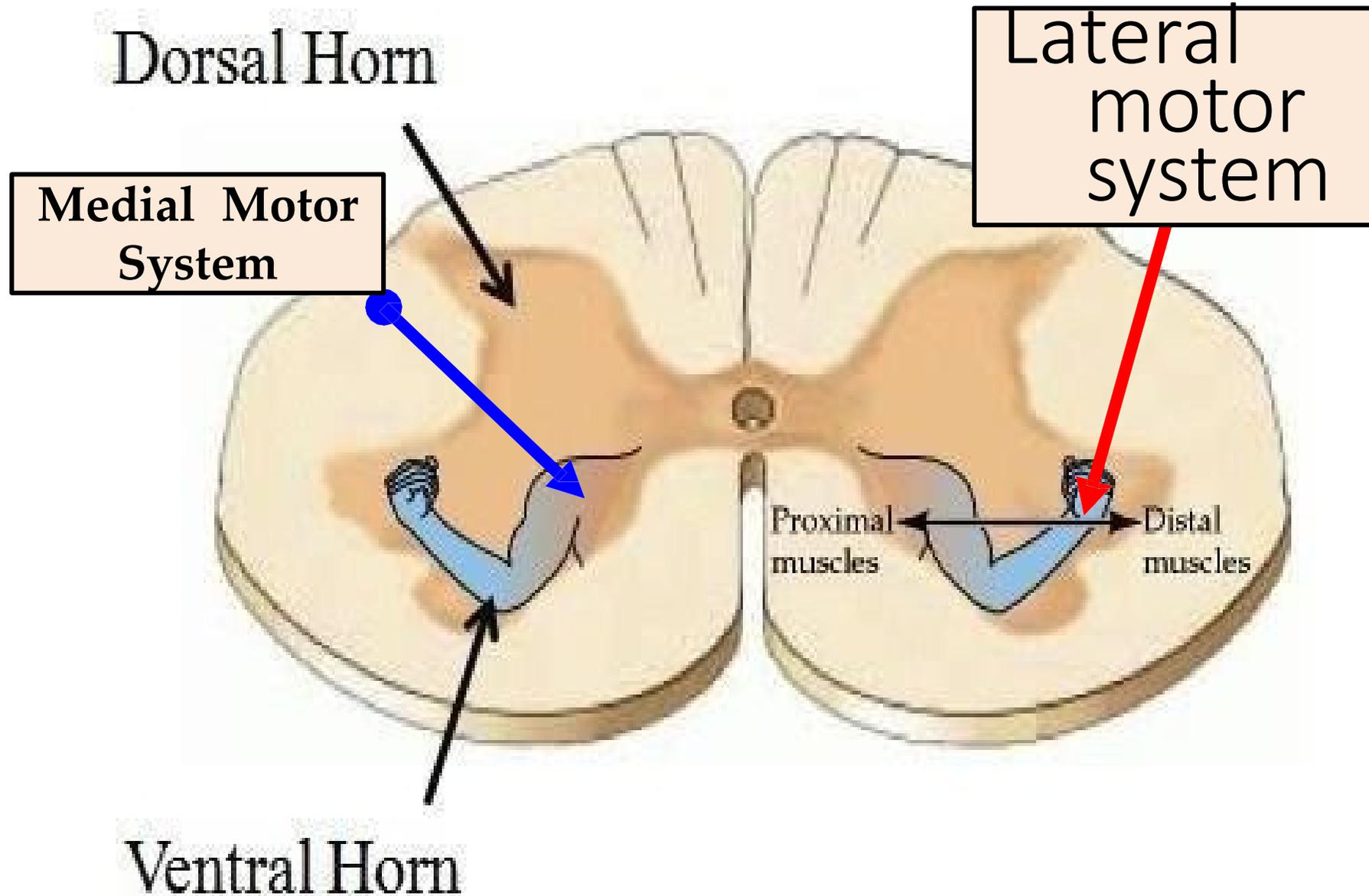


**Extrapyramidal Tracts**



**Pyramidal Tract**

# Medial and Lateral Motor System



# Descending Motor Tracts

□ **Descending motor tracts are 5 tracts** (named according to their origin and termination)

1) **Corticobulbospinal tract**

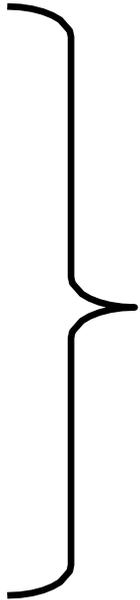
**(= Pyramidal tract)**

1) **Rubrospinal tract**

2) **Reticulospinal tracts**

3) **Vestibulospinal tracts**

4) **Tectospinal tract**



**Extrapyramidal tracts**

# Medial and Lateral Motor System

	a) Tracts of medial motor system	b) Tracts of lateral motor system
<b>Terminate on</b>	Medial motor neurons in the ventral horns of spinal cord.	Lateral motor neurons in the ventral horns of spinal cord.
<b>Innervate</b>	the antigravity muscles	distal muscles of the limbs
<b>concerned with</b>	control of the body posture	fine voluntary movements
<b>Include</b>	<ul style="list-style-type: none"> <li>1 Ventral corticospinal tract.</li> <li>2 Reticulospinal tract.</li> <li>3 Vestibulospinal tract.</li> <li>4-tectospinal tract.</li> </ul>	<ul style="list-style-type: none"> <li>1 Lateral corticospinal tract.</li> <li>2 Rubrospinal tract.</li> </ul>

# Pyramidal Tract

Motor areas, somatic sensory areas

**Corticospinal tract**

Left  
Internal  
Capsule

Midbrain

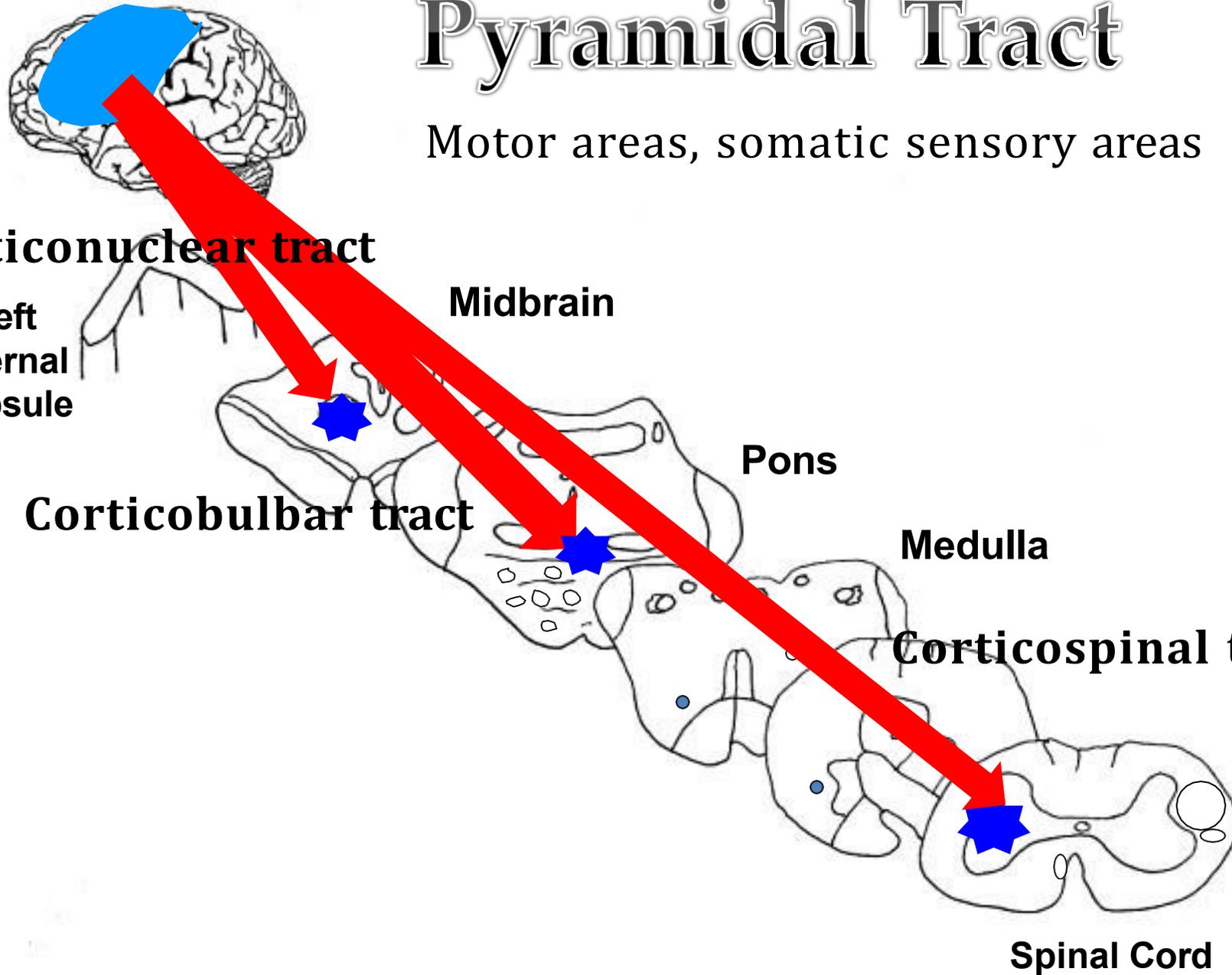
**Corticobulbar tract**

Pons

Medulla

**Corticospinal tract**

Spinal Cord



# Pyramidal Tract

Origin:

1. Primary motor area (area 4) → 30%

2. Premotor and supplemental areas → 30%

3. Somatic Sensory areas → 40%

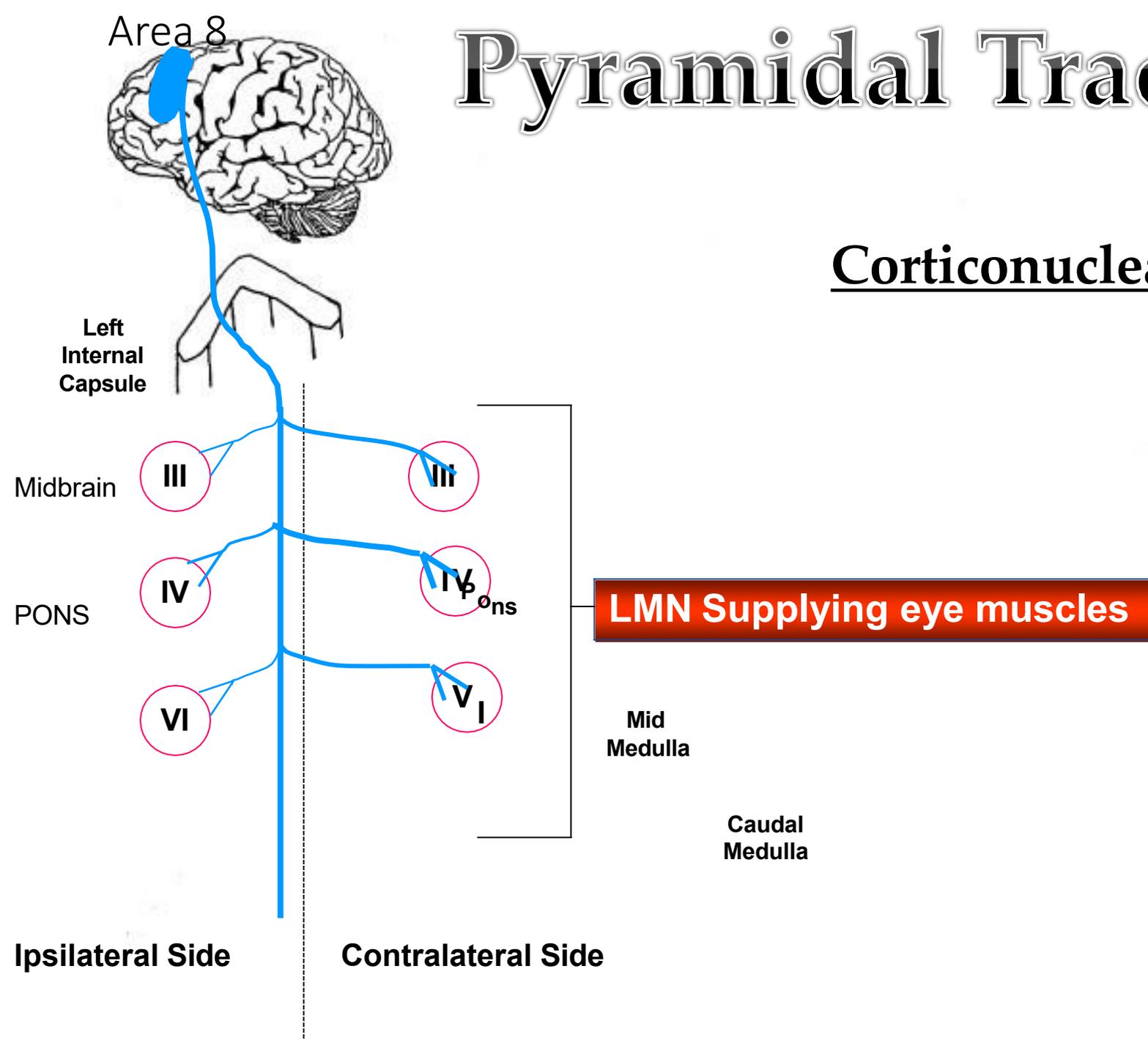
# Pyramidal Tract

Divisions:

- 1) Corticospinal tract** : from cortex to spinal cord
- 2) Corticonuclear tract**: from cortex to cranial nerves nuclei (**3,4,6**) that supply extraocular ms
- 3) Corticobulbular tract**: from cortex to **other cranial nerves (5,7,9,10,11,12)** of brain stem

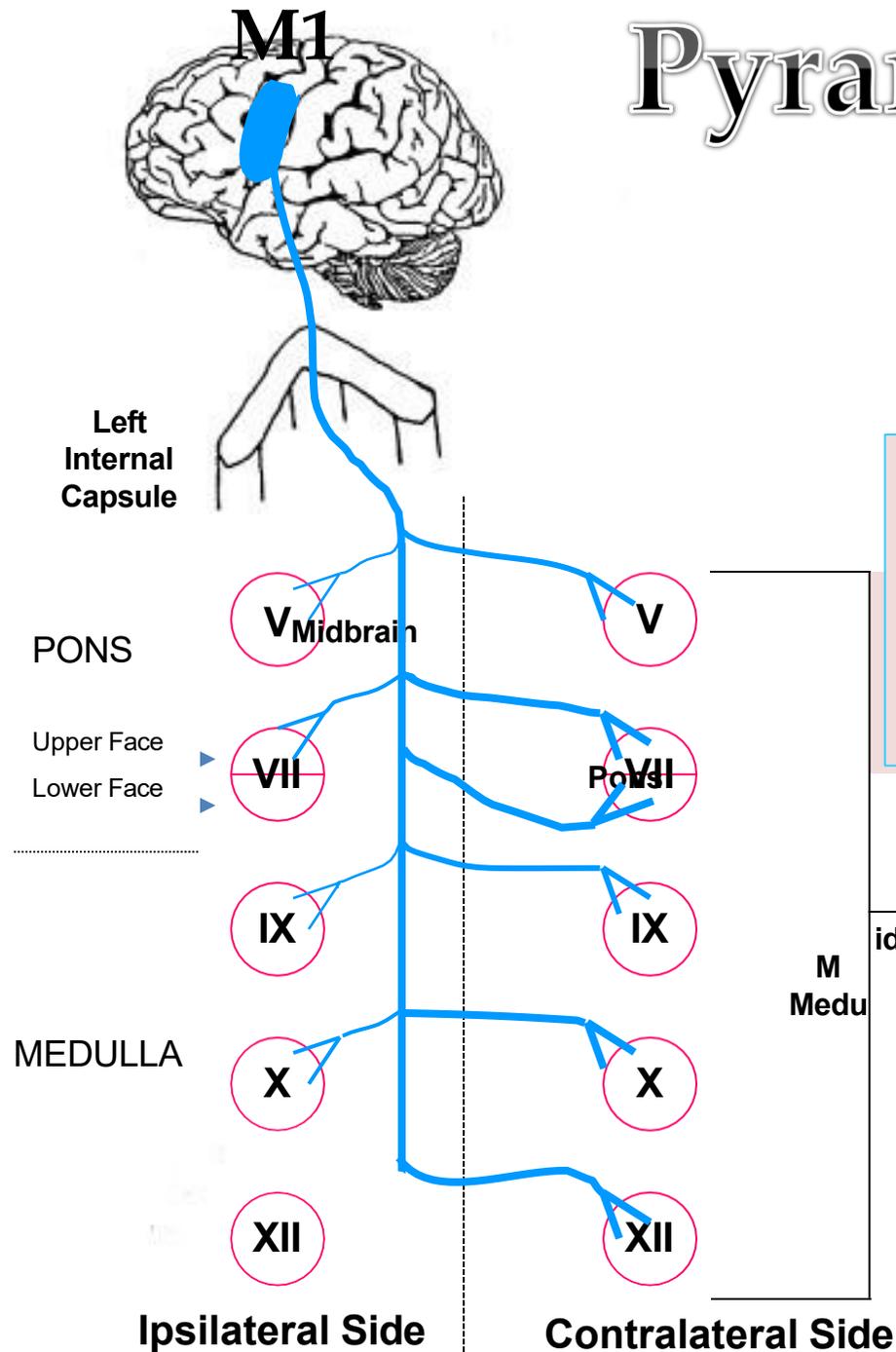
# Pyramidal Tract

## Corticonuclear Tract



# Pyramidal Tract

## Corticobulbar Tract



Corticobulbar Tract is crossed and uncrossed EXCEPT to lower motor neurons which control lower half of the face and tongue are crossed only.

**LOWER MOTOR NEURON NUCLEI**

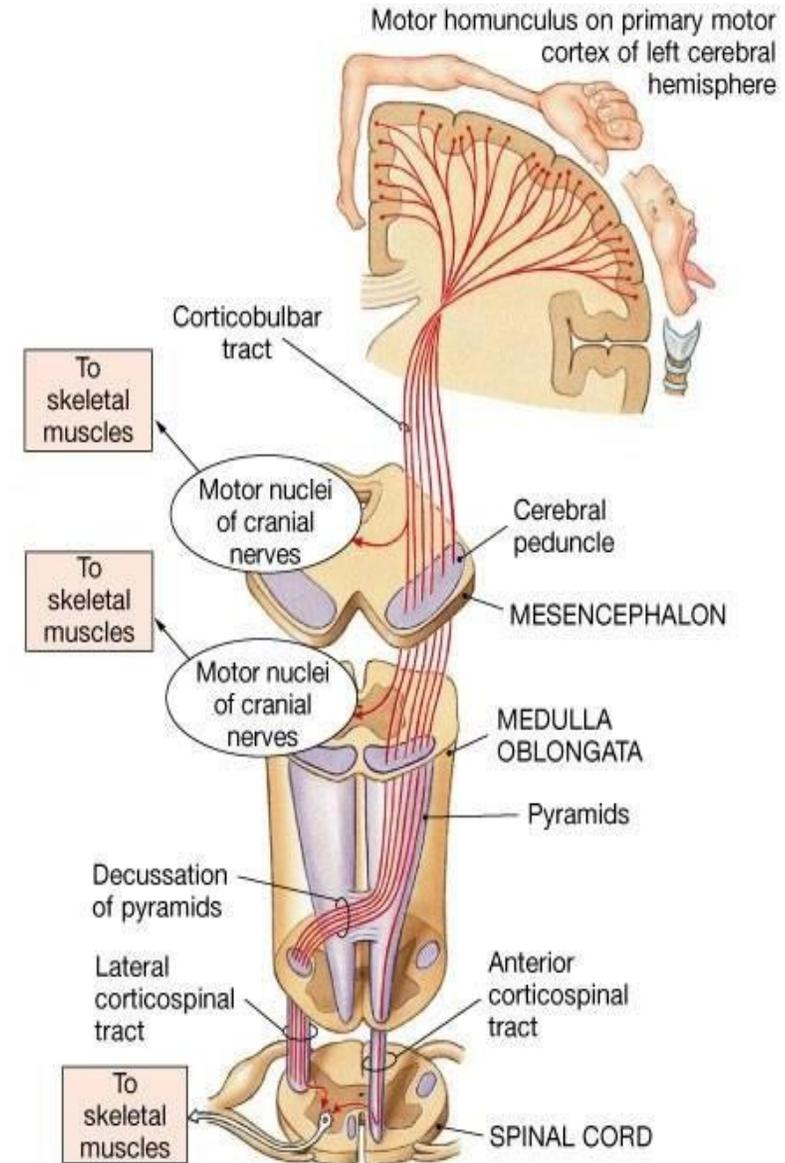
Caudal Medulla

Spinal

# Pyramidal Tract

## Corticospinal Tract

- (a) **90% cross to the opposite side** and descend in the lateral column of the spinal cord as **lateral corticospinal tract** → terminate on lateral AHCs and innervate **distal ms**
- (b) **10% not cross** → descend ipsilaterally as **ventral corticospinal tract**.
- **About 9%** of these fibers cross and terminate on medial AHCs → innervate the axial ms of the opposite side
- **Only 1%** of the fibers **terminate on the A.H.Cs** of the same side (bilateral innervations as respiratory ms).



# Pyramidal Tract

## Functions:

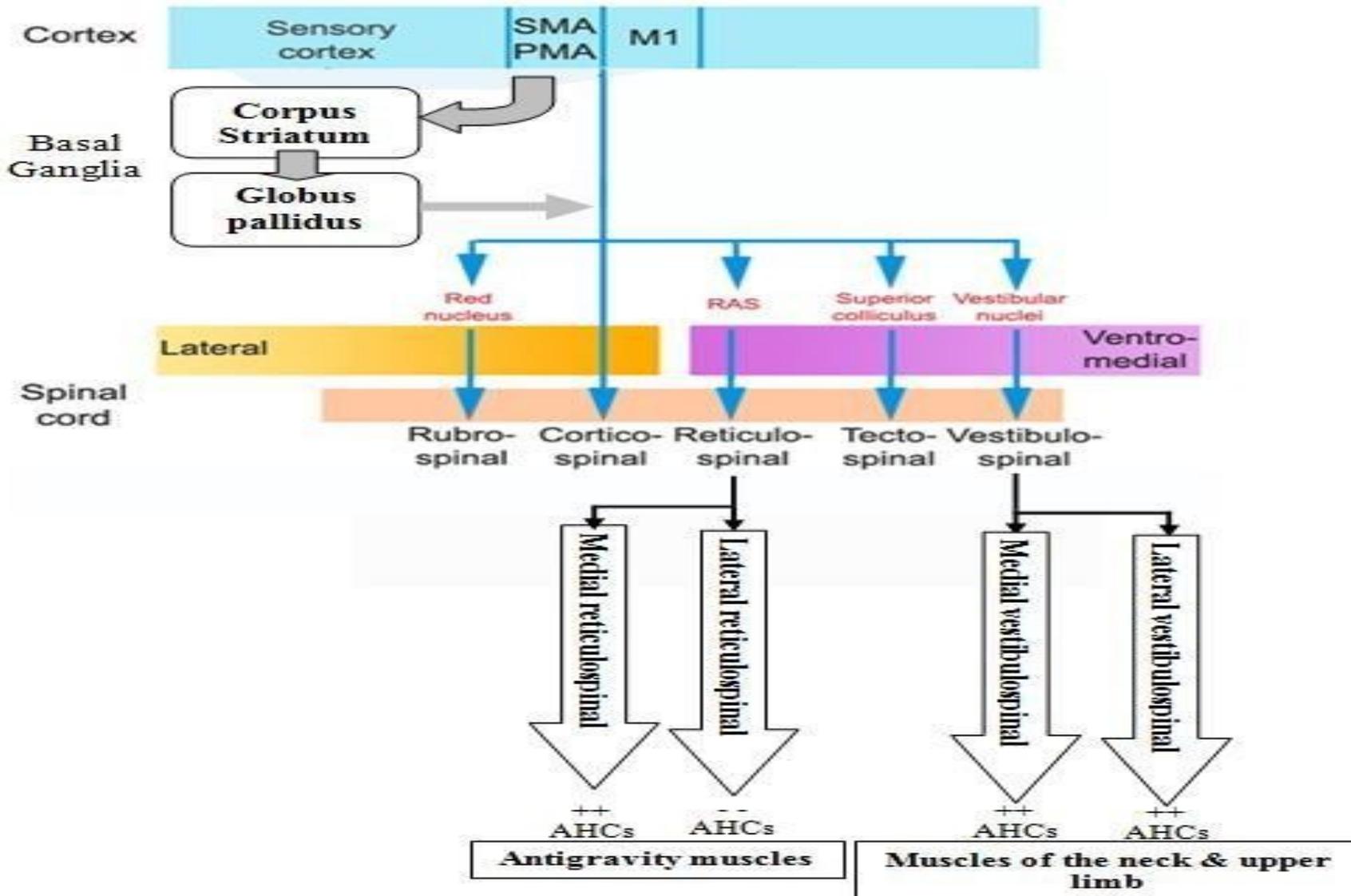
- 1) Initiation of voluntary movements: mediated by;
  - a) Corticobulbar tract; high skilled movements in face e.g. speech.
  - b) Lateral corticospinal tract; fine skilled movements of hand and fingers
- 2) A Role in automatic and postural movements: e.g. chewing, swallowing via **corticobulbar and medial corticospinal tracts**
- 3) Facilitatory to ms tone: of distal flexor ms of the limbs
- 4) Inhibits primitive withdrawal reflex i.e. **Babinski sign**

# Extrapyramidal Tracts

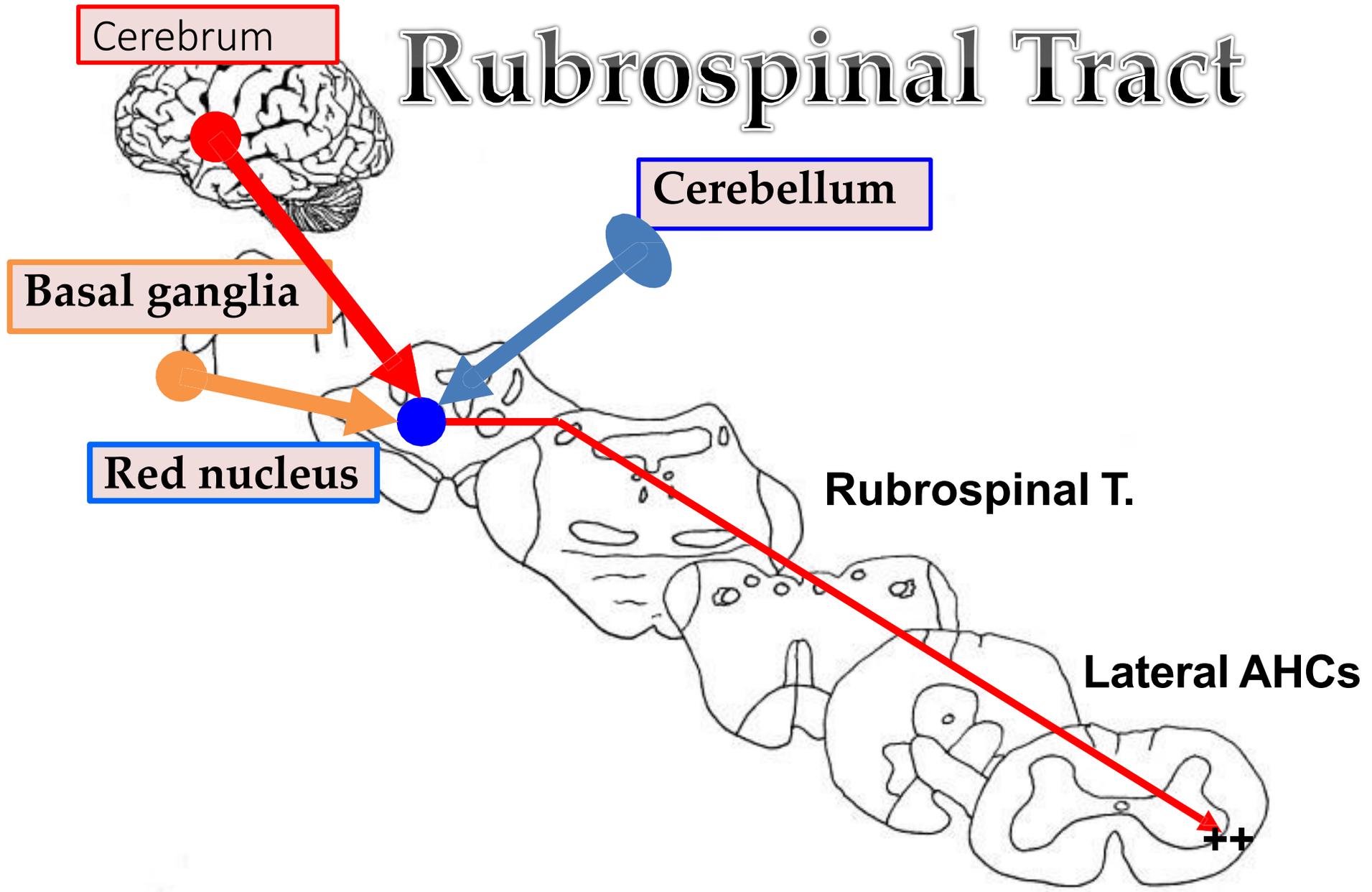
## Origin:

- From area (6) and area (4) → descends to **corpus striatum** → **Globus pallidus** → from the globus pallidus fibers pass to;
  1. **Reticular formation**
  2. **Vestibular nuclei**
  3. **Red nucleus**
  4. **Tectum of midbrain**
  5. **Inferior olivary nucleus**
- From these nuclei the following extrapyramidal tracts

# Extrapyramidal Tracts



# Rubrospinal Tract



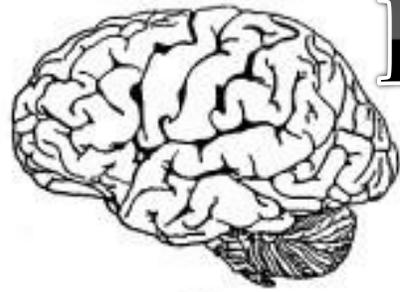
# Rubrospinal Tract

Functions:

**Terminates primarily in the cervical spinal cord, suggesting that it functions in upper limb.**

1. Has a role in **voluntary control of distal muscles** of upper limb responsible for skilled movements (together with lateral corticospinal tract).
2. Also **facilitates** motor neurons of the distal limb muscles.

# Reticulospinal Tracts



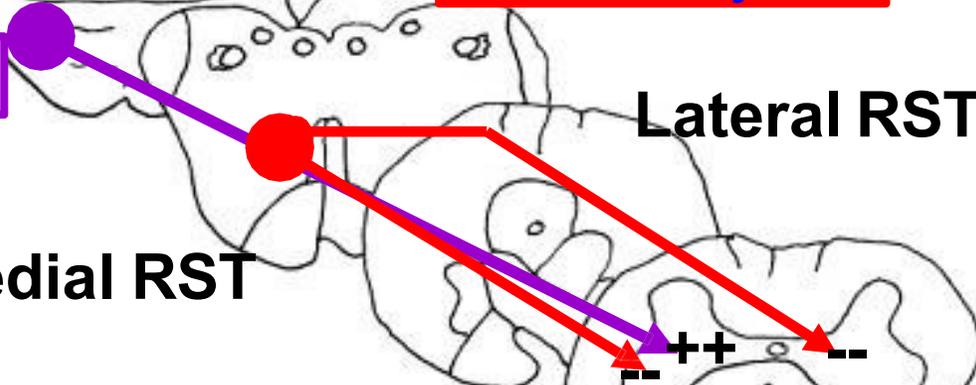
Pontine RF

Medullary RF

Lateral RST.

Medial RST

Medial AHCs



# Reticulospinal Tracts

## Functions:

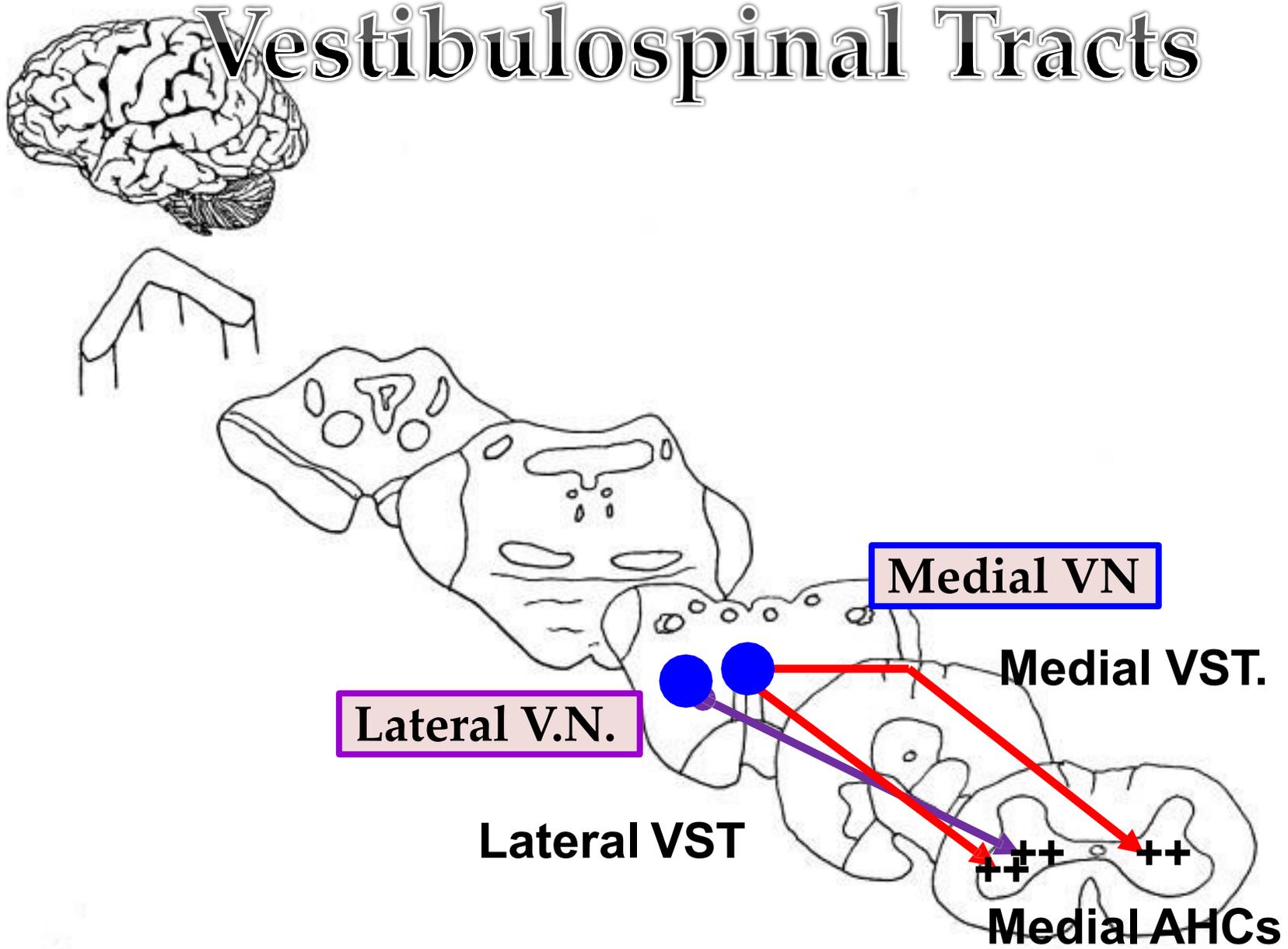
### a. Medial R.S.T.

- It mediates **facilitatory** effect on the motor neurons of the **antigravity ms** to maintain **postural** support.

### b. Lateral R.S.T.

- It **inhibits** the tone in the **antigravity ms** under some **postural conditions** e.g. **during walking**

# Vestibulospinal Tracts



# Vestibulospinal Tracts

Functions:

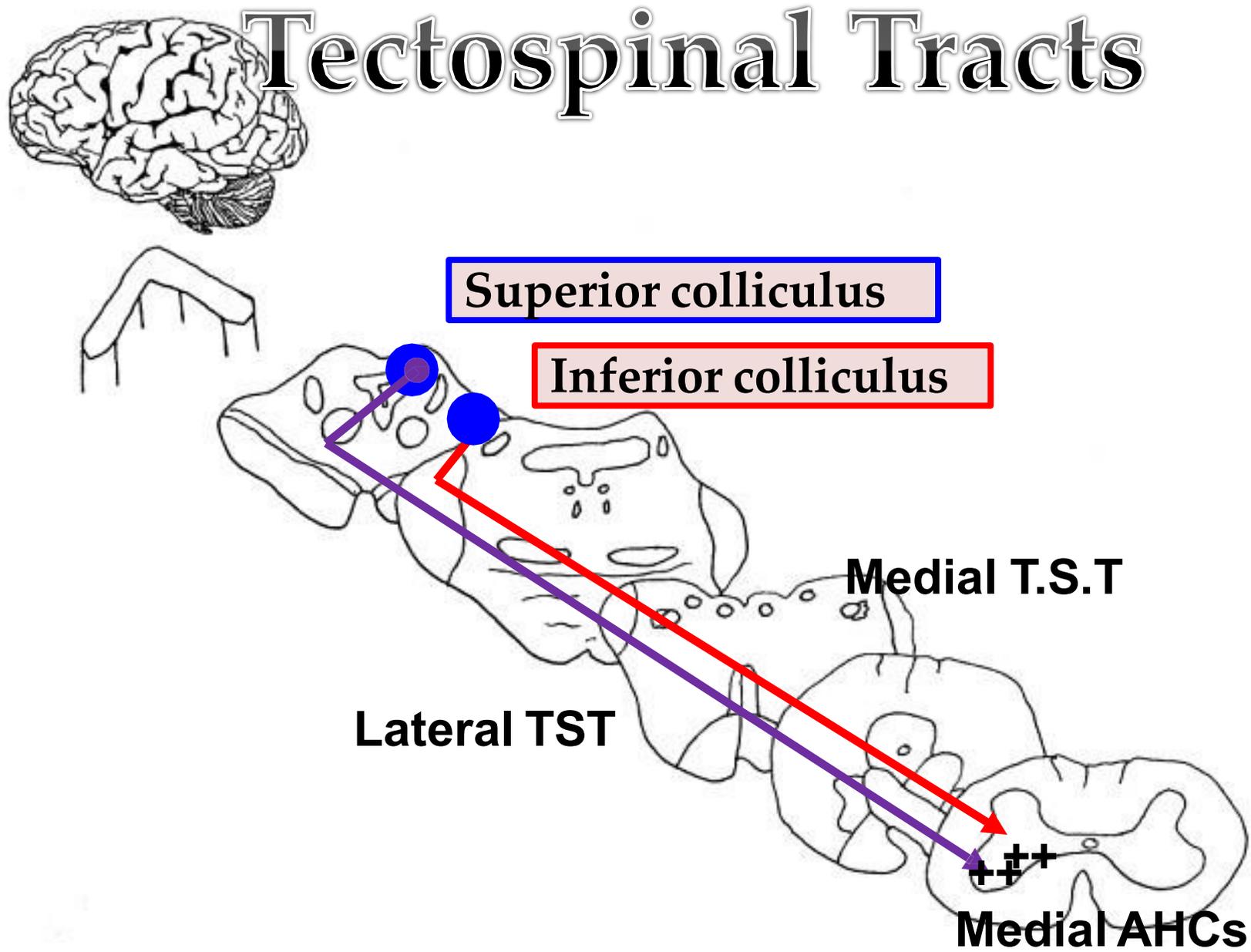
## a. Lateral V.S.T.

- Is **facilitatory to motor neurons of the antigravity ms** to maintain **body posture & equilibrium** in response to impulses from the **vestibular apparatus**
- Keep **equilibrium and posture** during changes in **head position** or **exposure to acceleration**

## b. Medial V.S.T.

- Is **facilitatory to motor neurons of the neck and upper limb ms** that are involved in regulation of the **head & upper limb position** during **exposure to acceleratory movements**

# Tectospinal Tracts



# Tectospinal Tracts

- ❑ **Origin:** Superior (Mainly) & inferior colliculi in the midbrain.
- ❑ **Course:** Descends contralateral.
- ❑ **Termination:** On the spinal motor neurons of the neck muscles only
- ❑ **Functions:** Reflex turning of the head in response to sudden visual or auditory stimuli

# Functions of extrapyramidal system

1. **Coordination** of isolated skilled muscle movements (with pyramidal pathway), **but cannot** initiate them.
2. **Coordination of gross**, involuntary movements of the big joints which require the activity of bulky proximal group of ms
3. **Keeping equilibrium** and adjustment of body posture
4. **Regulation of muscle tone** (some are excitatory other are inhibitory) **but inhibitory predominates**

# Lesion of Extrapyramidal system

- Mostly combined with **pyramidal tract** in internal capsule (but sometimes it may be **pure extra-pyramidal** lesion; e.g. lesion in the basal ganglia)

## Effects:

1. **Difficulty** in **initiation** of voluntary movements but not paralysis.
2. **Impairment** of orienting and balancing of reflexes.
3. Alternation in **muscle tone** (may increase or decrease).
4. Characteristic **appearance** of abnormal involuntary movements.
5. **Babinski's sign** reappear (Fanning of toes)



# Upper and Lower Motor Neuron Lesions

**By**

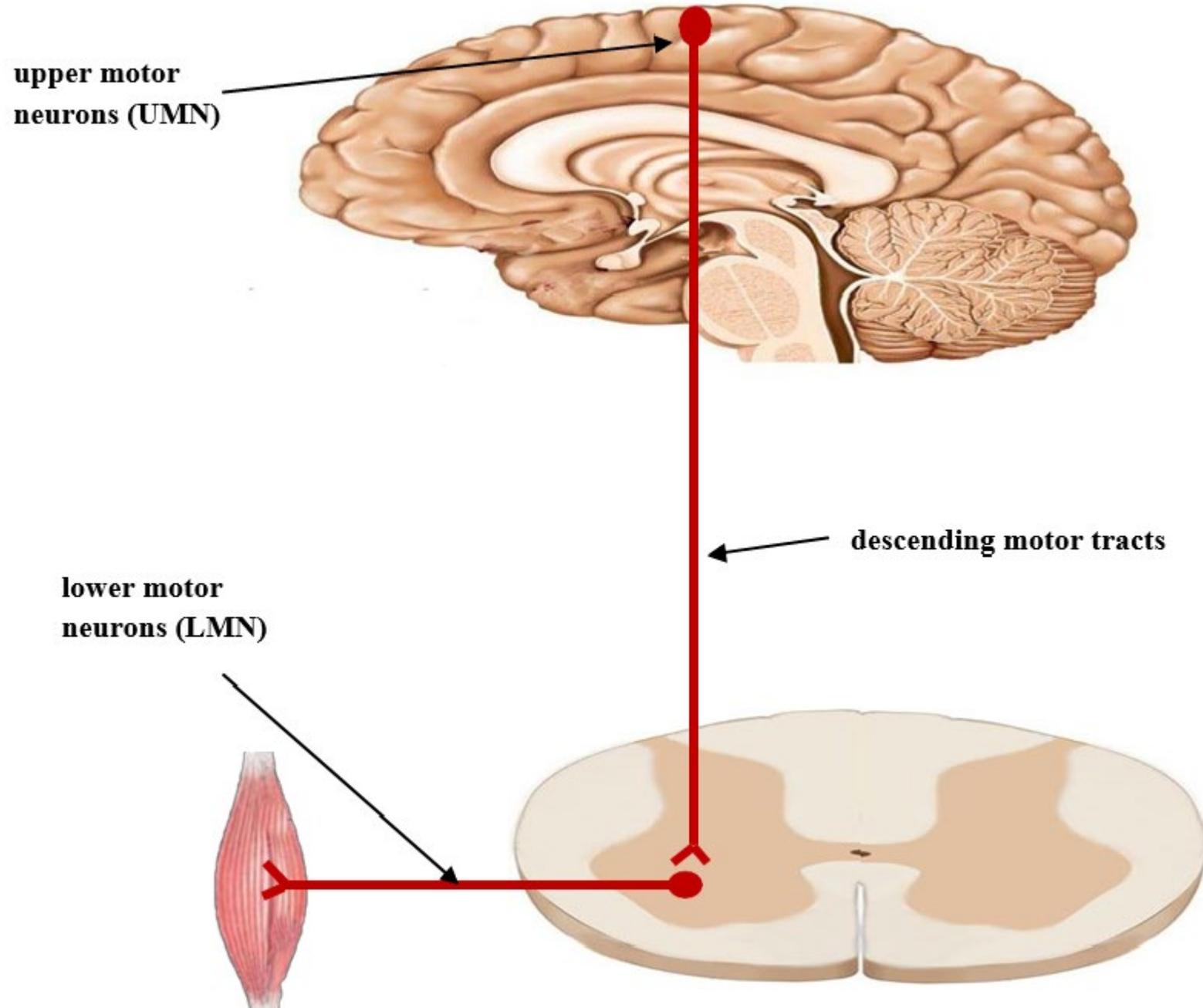
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# Upper and lower motor neurons



	Upper motor neuron (UMN)	Lower motor neuron (LMN)
<b>cell bodies lie in</b>	higher motor centers	spinal ventral horns or the corresponding cranial nuclei
<b>Their axons constitute the</b>	descending motor tracts	efferent motor fibers in the peripheral somatic nerves



**upper motor  
neurons (UMN)**

**lower motor  
neurons (LMN)**

**descending motor tracts**

**Upper and  
lower motor  
neurons**

# Upper motor neuron lesion (UMNL)



- **Def:** damage of upper motor neuron in the higher center or the descending motor tract.
- **Site:** Most common site of UMNL is the internal capsule.
- **The main cause of internal capsule lesion** is hemorrhage or thrombosis of the lenticulo-striate artery.

# Effects of UMNL at internal capsule



## (I) Motor loss:

### 1-Contralateral paralysis

\* Means complete loss of voluntary movements.

\* Occurs in:

a- Distal limb muscles particularly those of hand & fingers (performing voluntary skilled movements) due to damage of lateral corticospinal tract.



**b- Muscles of the lower face & tongue due to damage of contralateral corticobulbar tract.**

**2-Contralateral paresis:** weakness(i.e., incomplete loss of movements). **It occurs in:**

**a) Muscles of the upper face on the paralyzed side** of the body as they receive **bilateral** supply from the **cortico-bulbar tract.**

**b) The axial muscle** controlling posture & equilibrium as they receive **bilateral** supply from the **ventral cortico-spinal tract.**

**3-Spasticity (hypertonia of skeletal muscles)**: due to ↑ supraspinal facilitation (by damage of **cortico-reticular tract**).

• **Mechanism:**

➤ Normally the **net effect of cortico-reticular tract** (from areas 4 & 6) is:

**i) Inhibitory** to pontine (facilitatory) reticular formation & vestibular nuclei (facilitatory areas).

**ii) Facilitatory** to medullary (inhibitory) reticular formation.

➤ Accordingly, **damage of the cortico-reticular fibers** in UMNL → ↑ supraspinal **facilitation** → ↑ stretch reflex → hypertonia or spasticity.

**4-Exaggerated tendon jerk & clonus:** due to ↑  
supraspinal facilitation (by damage of **cortico-reticular tract**).

**5-Positive Babinski's sign:**

- Abnormal planter response characterized by **dorsiflexion of the big toe & fanning (separation) of the lateral 4 toes** on scratching the skin of the lateral edge of the sole of foot.

➤ Normally, scratching the lateral margin of the foot → plantar flexion of all toes & this is called **plantar reflex**

➤ It is a withdrawal reflex that is normally inhibited by impulses from area 4 & 6.

# ➤ Causes of Babinski's sign:

## A) Physiologically in:

**1-Infants below 1 year** age because corticospinal tract is **not yet myelinated**.

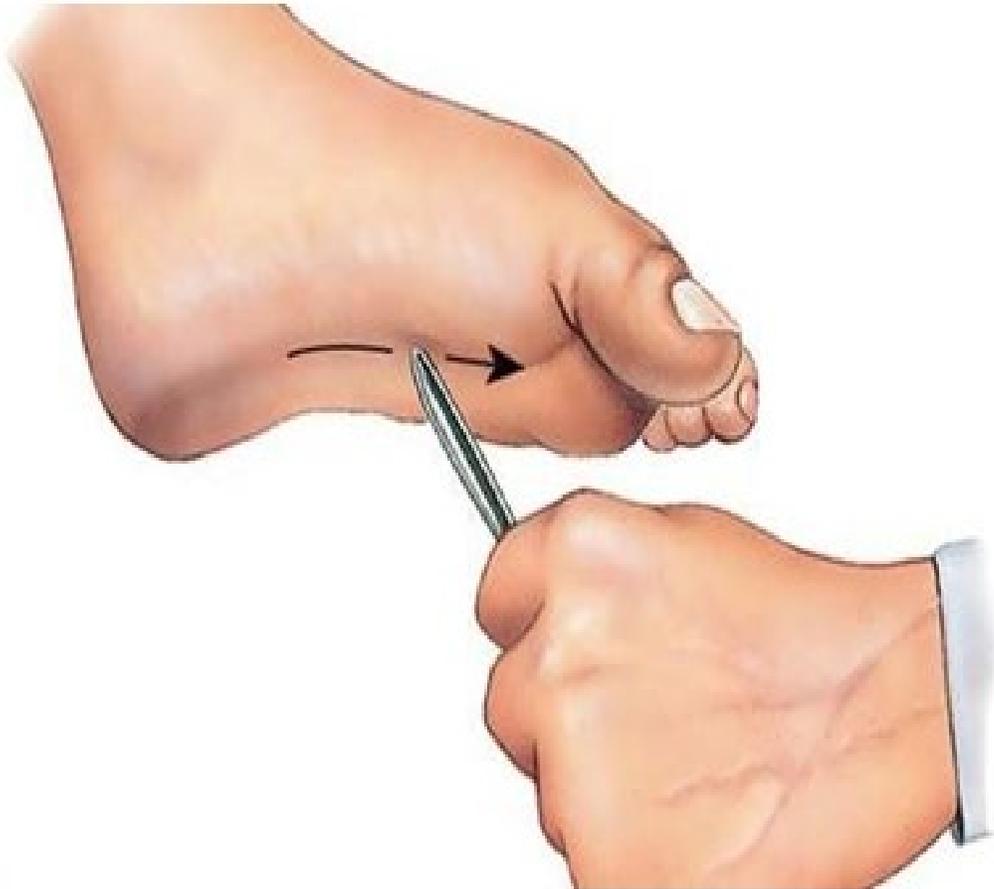
**2-Normal adults** during sleep & general anaesthesia due to **depression of cerebral cortex**

## B) Pathologically in:

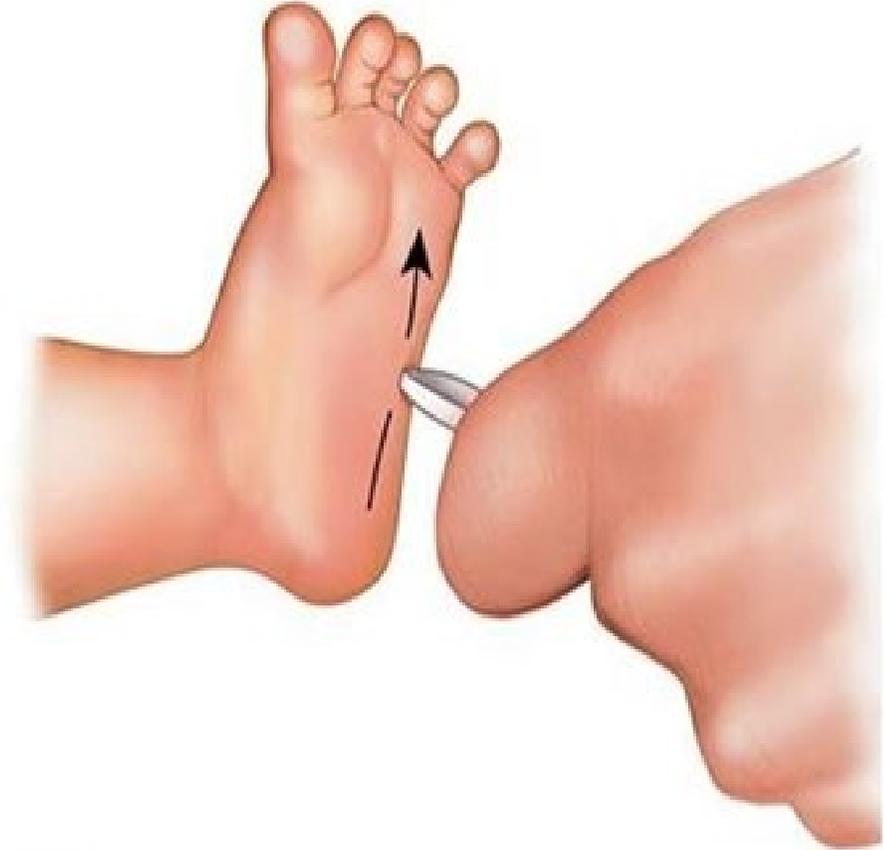
1-Coma  
2-UMNL

### \*Mechanism:

- It is a withdrawal reflex that is **normally inhibited** by impulses from area 4&6.
- So, it appears when these areas are **damaged or inhibited**.
- Isolated lesions in area 4 → **dorsiflexion of big toe** only
- Isolated lesions in area 6 → **fanning** of the lateral four toes only.



**Plantar reflex**



**Babiniski sign**



## **6-No or minimal muscle atrophy because:**

- i. The LMN is intact and the muscle **contracts reflexely.**
- ii. The muscle is still supplied with **trophic substance** from LMN.

**N.B.** after long period the muscle undergoes "**disuse atrophy**".



## 7- Normal response to electrical stimulation:

- In UMNL the electrical response of muscle is **identical to normal muscle.**
- Normal muscle response to electric stimuli:
  - a) **Faradic current** produces **clonic or tetanic** contractions depending upon the frequency of action potentials.
  - b) **Galvanic current** produces contractions that occur only **at closing (make) and opening (break) of the circuits.**

- The magnitude of response differs whether the stimulating electrode was cathode or anode and whether the circuit is opened or closed.
- **Normally: CCC > ACC > AOC > COC** (due to unknown cause)
- **CCC** = Cathodal closing contraction, **ACC** = Anodal closing contraction, **AOC** = Anodal opening contraction, **COC** = Cathodal opening contraction.



## (II) Sensory loss:

*Contralateral hemi-anaesthesia* i.e., loss of all sensations on the **opposite** side of the body.



# Lower motor neuron lesion (LMNL)

## ➤ Causes:

- Damage of AHCs or cranial motor nuclei e.g. by poliomyelitis.
- Damage of motor axons e.g. by trauma or diabetes mellitus.

## ➤ Effects of LMNL:

A) Structural changes.

B) Functional changes.

## (A) Structural changes:



### 1- Atrophy of denervated muscles due to:

- a) Deprivation of the muscle from **trophic factors** secreted by the cut motor neuron.
- b) Lack of movement.



## 2-Degeneration of the motor neuron due to:

a) **Wallerian degeneration** of the injured axon **distal to** the site of lesion.

b) **Retrograde degeneration: proximal to** the site of lesion.

➤ **Regeneration** of the neuron may occur provided the cell body is intact.



## (B) Functional changes:

### 1- Flaccid paralysis: characterized by:

- a) Complete loss of all types of movements: including voluntary, postural & reflex movements.
- b) Hypotonia of paralyzed muscles (flaccidity) due to interruption of motor component of stretch reflex.
- c) Lost of tendon jerk in the denervated muscle.

d) Lost all reflexes in the denervated muscle.

e) The extent of paralysis is limited to a small group of muscles

## 2- Denervation supersensitivity:

➤ Def: Is marked  $\uparrow$  in the sensitivity of the denervated muscle to acetylcholine.

➤ Cause: It's due to **spread out of acetylcholine receptors** which nearly cover the entire surface of the muscle cell membrane.

### 3-Fasciculation and fibrillations:

Fasciculations	Fibrillations
Appear <b>few days</b> after denervation.	after <b>few weeks</b> after denervation
<b>Visible</b> contractions	<b>non visible</b> recorded by electromyogram ( <b>EMG</b> ).
<b>Synchronous</b> contraction of <b>all muscle Fibers</b> in certain motor unit caused by <b>injury potentials</b> generated in <b>the main axon</b> .	<b>A synchronous</b> contraction of <b>individual muscle fibers</b> in certain motor units caused by injury potentials generated in <b>their terminal axon branches</b> .

*Fasiculations & fibrillations **disappear** when the motor nerves are either:*

- i) **completely degenerated** or*
- ii) **successful re-innervation of the muscle occur.***



## 4-Reaction of degeneration(R.D):

- **Def:** Is abnormal electric response of the denervated muscle to electrical stimuli.
- **Cause:** It occurs only in **LMNL**.

➤ **Types:** depending on the severity of the condition:

<b>Incomplete R.D</b>	<b>Complete R.D</b>
<p><b>1-No response to faradic current.</b></p> <p><b>2-Response to galvanic current is weak &amp; shows that ACC &gt; CCC</b></p>	<p><b>Absent response to both faradic &amp; galvanic current as the muscle becomes atrophied &amp; fibrous</b></p>



# Vestibular apparatus

# Structure of vestibular apparatus



• It consists of 2 parts:

**1- Semicircular canals**

**2- Utricle and saccule**

# Semicircular canals (S.C.C)



- **Number:** 3 on each side.
- **Ends:** Each S.C.C has **two ends**: One of them is dilated called **ampullary end** which contains the receptors of S.C.C (Crista).
- **Contains:** Each S.C.C contains **endolymph**.



- **Arrangement:** Arranged with right angles in between (represent 3 planes of the space):
  - One **anterior vertical.**
  - One **Horizontal.**
  - One **posterior vertical.**
- **They open into** the **utricle** by **5 openings** only because the non ampullary ends of anterior vertical & posterior vertical open into a common opening.



- **Co-planner S.C.Cs:**

- Each semicircular canal on one side **correspond to** one SCC on the opposite side (lie with it in the **same or parallel** plane), and both act as a **functional unit** (i.e. act together).
- **For example:**
  - \* **Horizontal** canals on both sides.
  - \* The **anterior vertical** canal on one side and **posterior vertical** one on the opposite side.

- **The structure of the crista:**

➤ It consists of an elevated ridge of epithelium covered by a wedge-shaped gelatinous mass called a cupula.

➤ **Epithelium consists of 2 types of cells:**

**1-Hair cells:** its upper border show **ciliary projections** which are embedded into the **cupula**. Its base and lateral borders are surrounded by the **vestibular nerve endings**.

**2-Supporting cells:** (*columnar in between the hair cells*).



# Utricle and saccule

- 2 small sacs or cavities.
- Each one contains specific receptor called **Macula**.
- **Macula (Otolith organ):**
  - Similar in structure to crista but the copular or gelatinous part is rich in **calcium carbonate crystals** which make it hard.
  - These crystals are called **Otoconia** and the covering is called **otolith membrane**.



# Vestibular hair cell

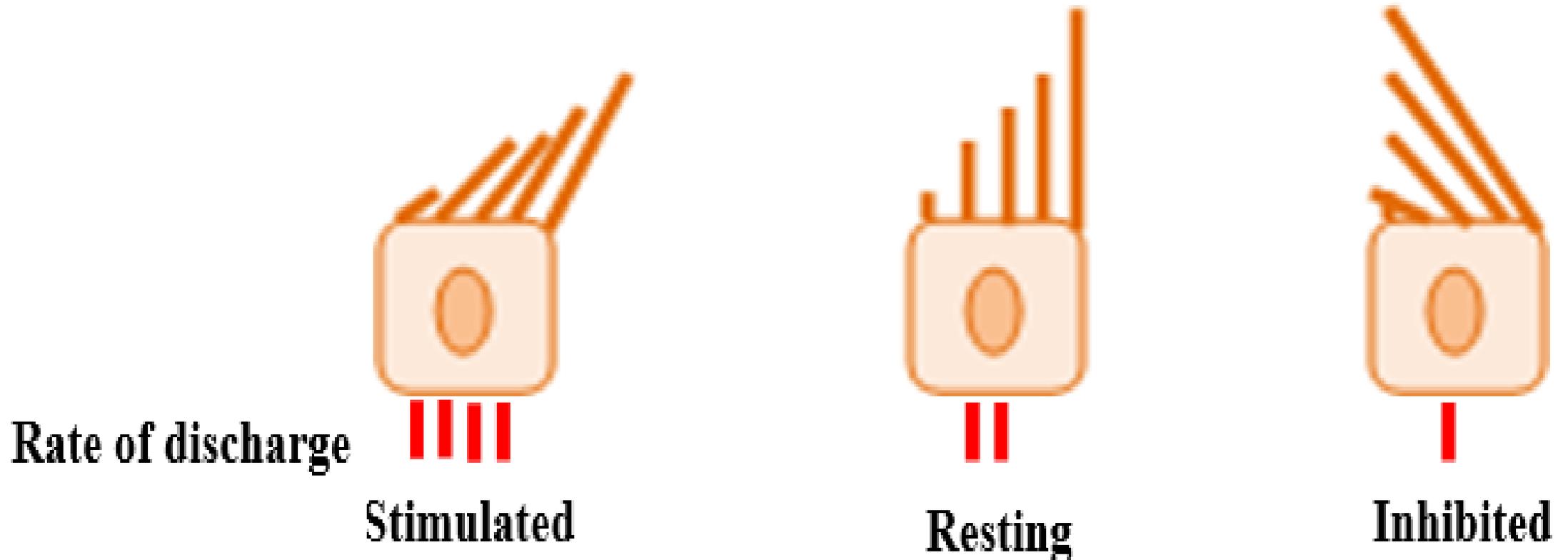
- Has upper border which shows 2 types of ciliary projections:
  - Stereocilia: short, numerous (60-100), thin. and motile
  - Kinocilium: long, single, thick. and tough.
- The **kinocilium** is located at **one side** & the **stereocilia** become **progressively shorter** towards the **other side** of the cell.

- **Stimulation of vestibular hair cell:**

- **During Rest (no deflection of cilia):** there is a basal rate of impulse discharge from the hair cells along the vestibular nerve.
- **Bending of the stereocilia towards the kinocilium:** → opening of mechanosensitive  $K^+$  channels (↑  $K^+$  permeability) → depolarization of hair cells → ↑ release of chemical transmitter → ↑ impulse discharge from the hair cells.

- **Bending of stereocilia away from the kinocilium:**

→ closing of  $K^+$  channels ( $\downarrow$   $K^+$  permeability) → hyperpolarization of hair cells →  $\downarrow$  release of chemical transmitter →  $\downarrow$  impulse discharge.



# • Orientation hair cells in the of vestibular apparatus:

## 1- In cristae:

- The position and arrangement of stereocilia in relation to the kinocilium is the same in all hair cells of the same crista.
- In Horizontal S.C.Cs: the Kinocilium towards the utricle
- In Vertical S.C.Cs: the Kinocilium away from the utricle

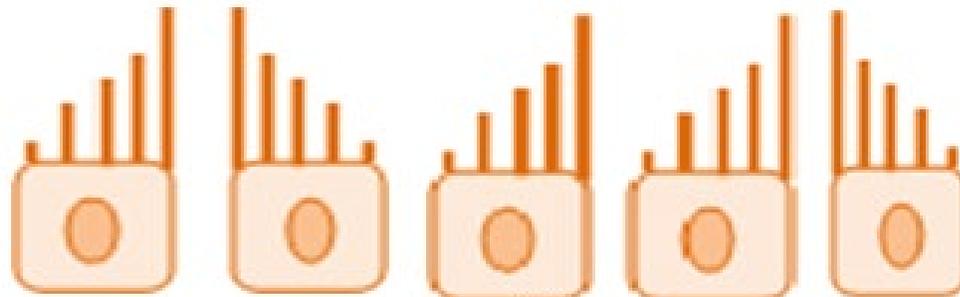


## 2-In maculae:

The position of the kinocilium and stereocilia **differ**  
**from one group of hair cells to another in the same**  
**macula.**



**Hair cells in the crista**



**Hair cells in the macula**

# Functions of the vestibular apparatus

## A) Functions of the utricle and saccule (Maculae):

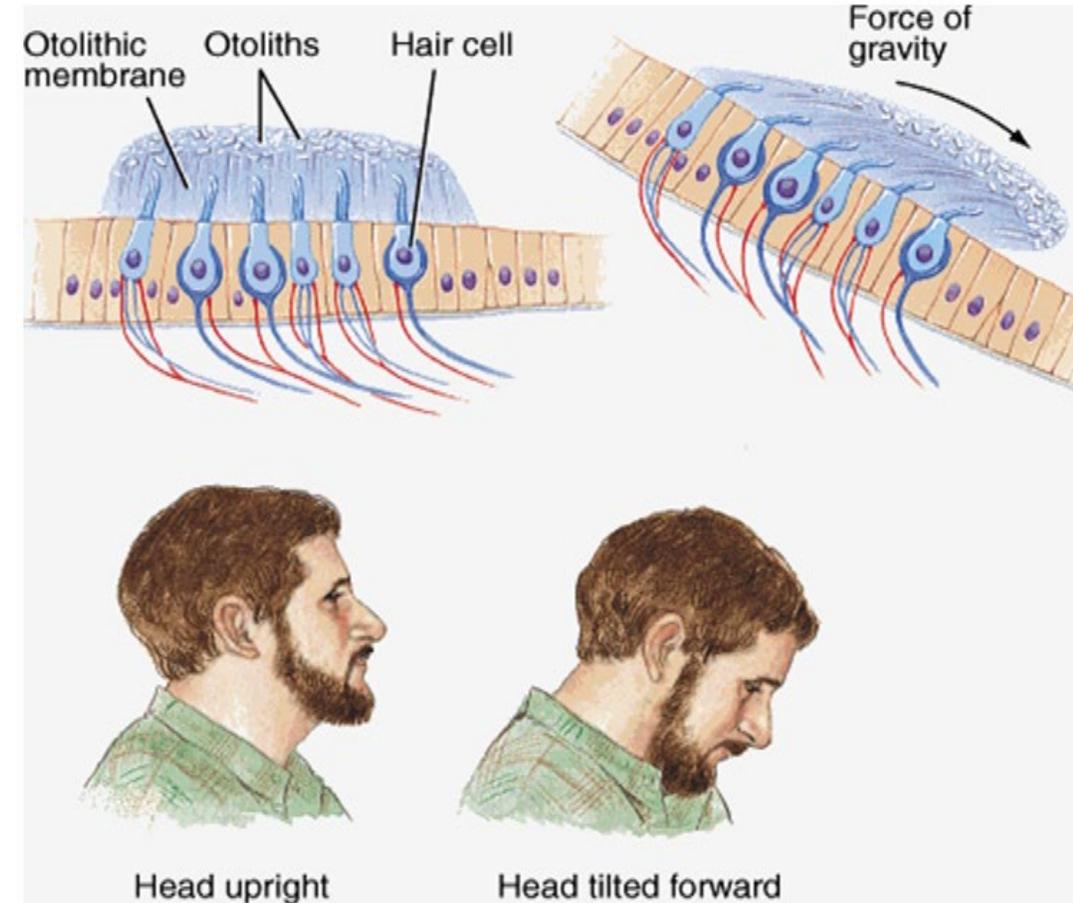
### 1- Detection of the absolute position of head in space:

a) When the head is in erect position, equal discharge from the maculae of both sides because both are exposed to the same gravitational force.

-The **symmetrical** discharge from both maculae interpreted by the **cortex** that the head is in **erect** position.

## b) If the head is tilted to one side:

- The macula on that side (otolith membrane) is **pulled more** under the effect of gravity and the macula on the opposite side is less exposed to the gravitational force → **asymmetrical** discharge from both maculae, interpreted by the **cortex** that the head is **tilted** to that side.





## **2- Initiation of reflexes that maintain body posture.**

## **3- Detection of linear acceleration:**

Responsible for perception of **linear acceleration** as regard:

**a) Onset of acceleration.**

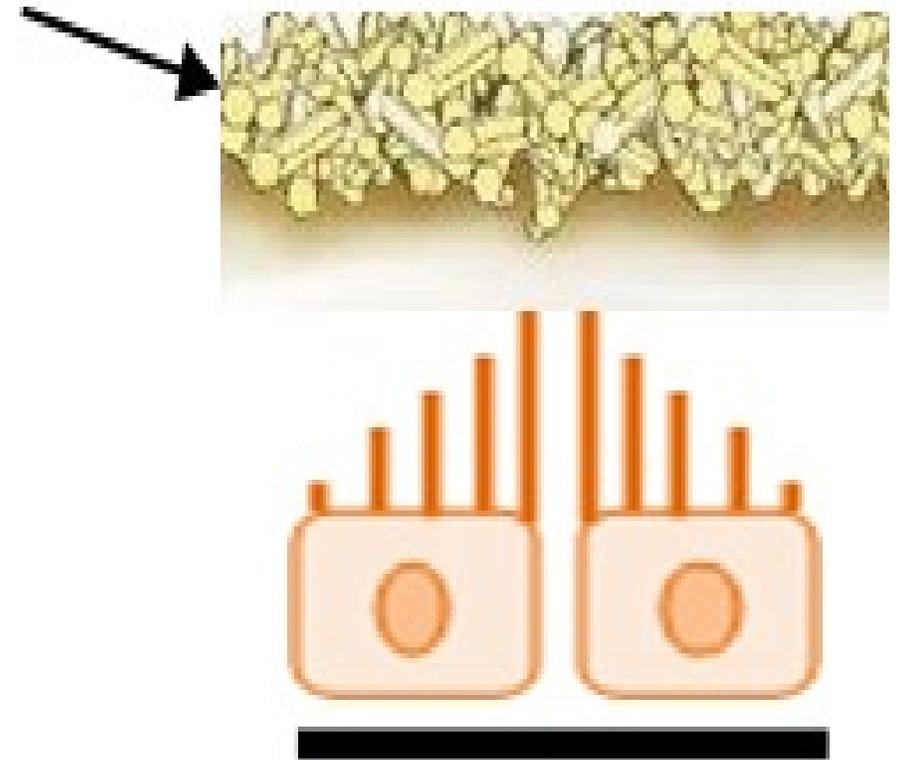
**b) Deceleration (Stoppage).**

**c) Change in the rate of acceleration.**

a) Before the start of acceleration (resting):

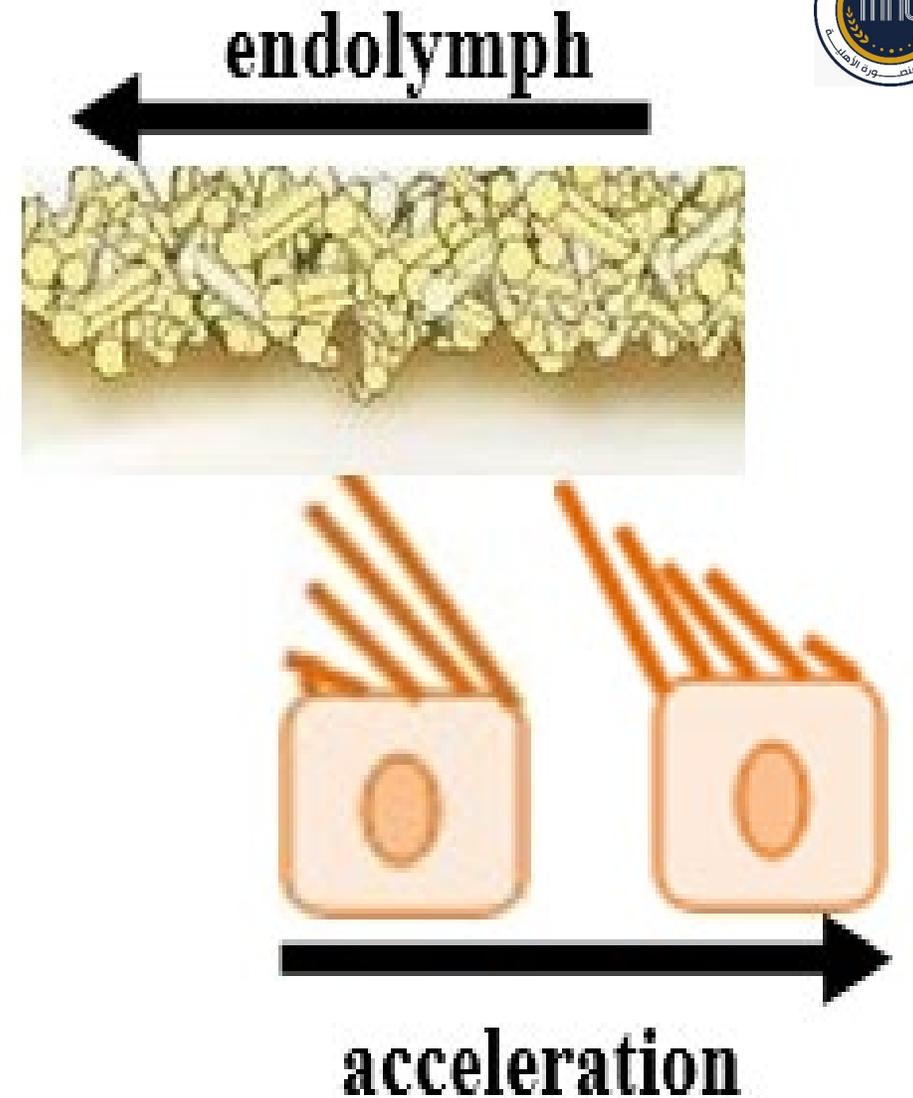
Maculae show **symmetrical** basal discharge.

Otolith



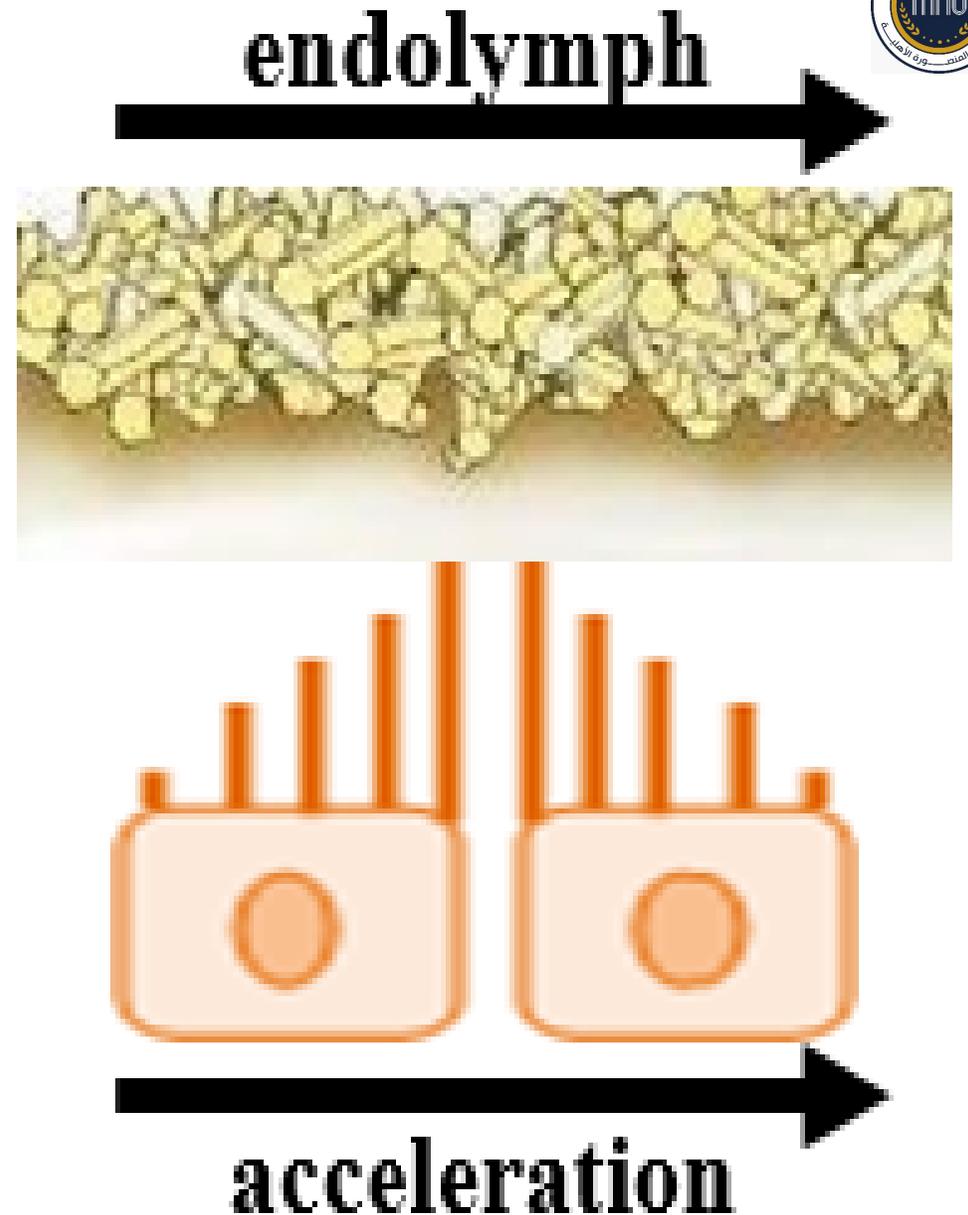
## b) At the onset of acceleration:

Due to **inertia**, the maculae displaced in opposite direction  
→ bending of the cilia →  
peculiar pattern of discharge →  
when this pattern reaches the  
cortex, the cortex interprets it →  
feeling of onset of acceleration.



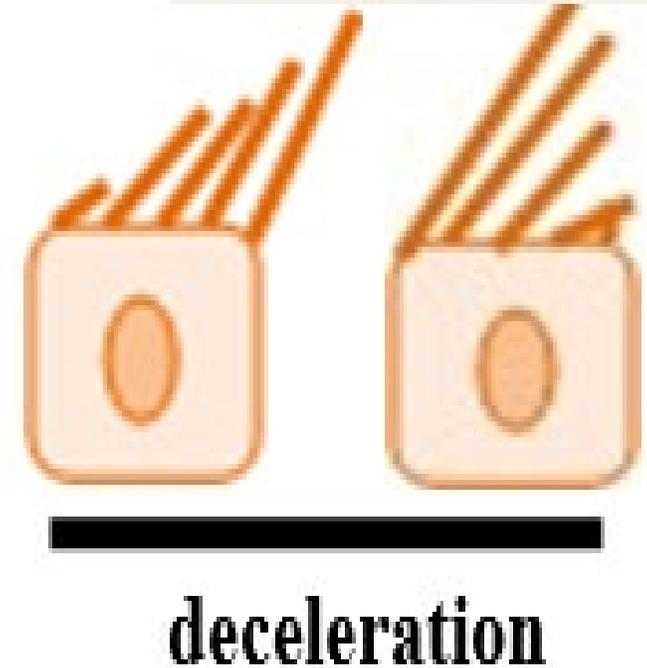
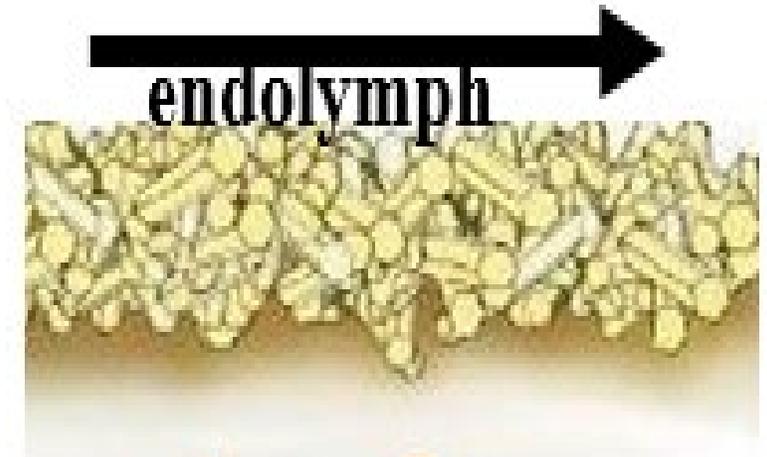
### c) With constant speed:

When the linear acceleration reaches a constant speed, the otolith membrane moves with the same speed as the head → cilia straighten again → discharge becomes basal → no feeling of movement occurs.



## d) At Deceleration:

As movement stop, the otolith membrane is displaced in the same direction of previous acceleration due to **momentum** → bending of the cilia → peculiar pattern of discharge → when this pattern reaches the cortex, the cortex interprets it → feeling of stoppage of acceleration.





## **B) Functions of the S.C.Cs:**

**1- Perception of angular acceleration (Rotation):** as regard:

- 1- **Onset** of acceleration.
- 2- **Stoppage** of acceleration.
- 3- **Change** in velocity of rotation.

➤ **HOW? by the following mechanism of action:**

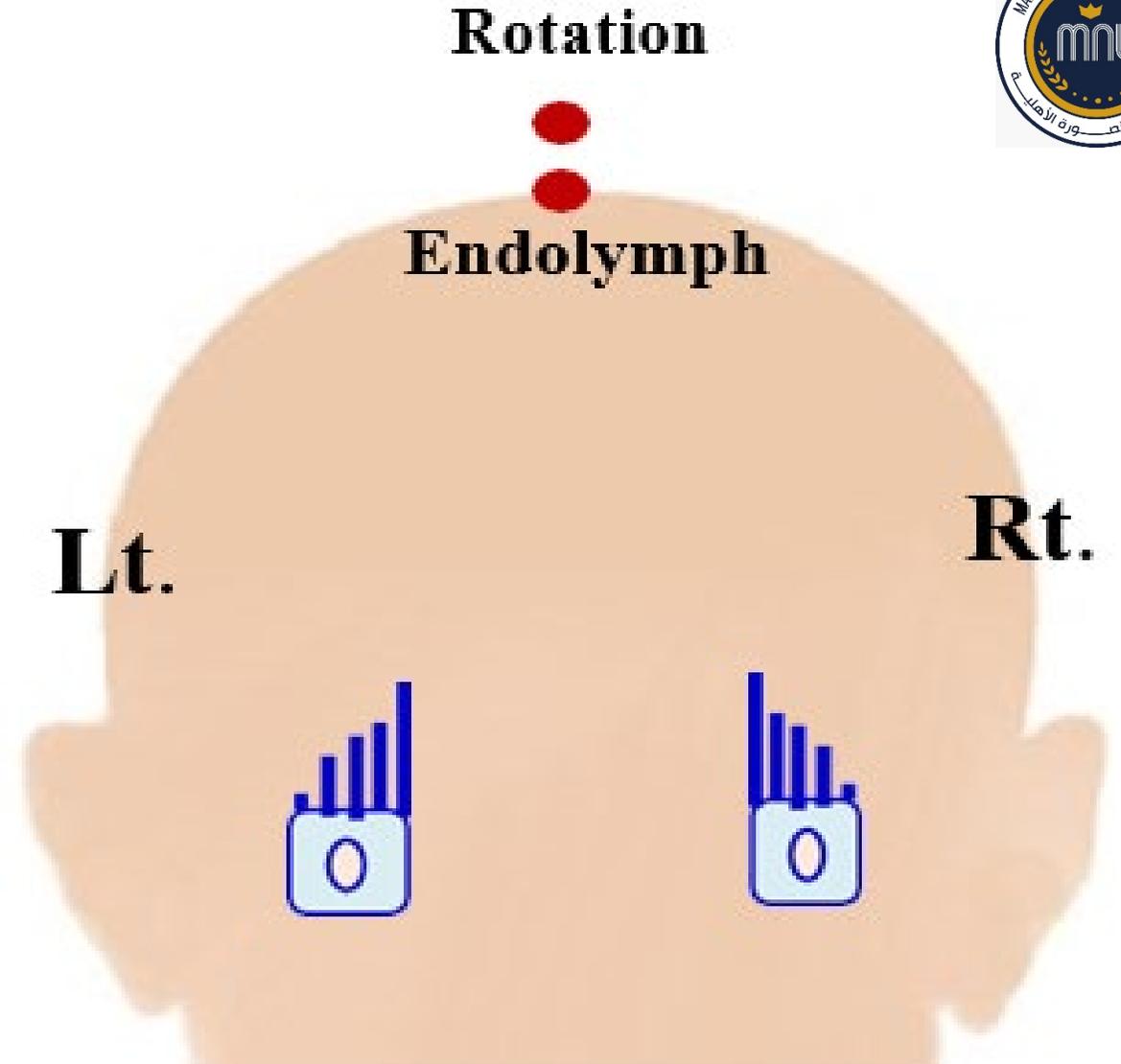
- The rotation of the body in certain plane, stimulate the 2 corresponding S.C.Cs acting on that plane.

\* **For Example:**

- Rotation in the horizontal plane from the **left to right**, the 2 horizontal SCCs will act together, **how?**

## a) Without rotation (at rest):

- The rate of discharge from both S.C.Cs is equal (i.e. **symmetrical**) → no feeling of movement



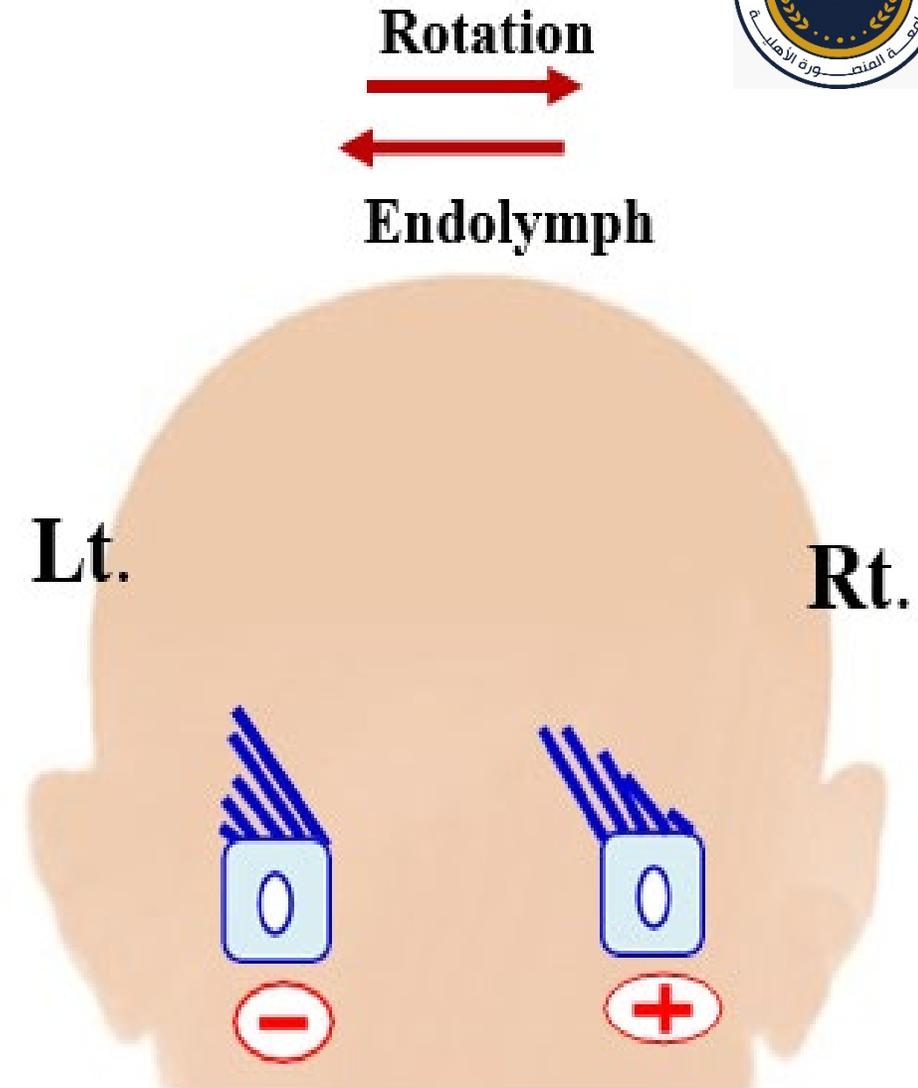
**b) With onset of rotation:** e.g: from left → right.

-The endolymph is displaced by its **inertia** to the opposite direction of rotation (**left side**) leading to:

\*Bending of the cupula of **right crista towards the utricle**. i.e. bending the stereocilia towards the kinocilium → ↑ **the rate** of discharge from the **right crista**.

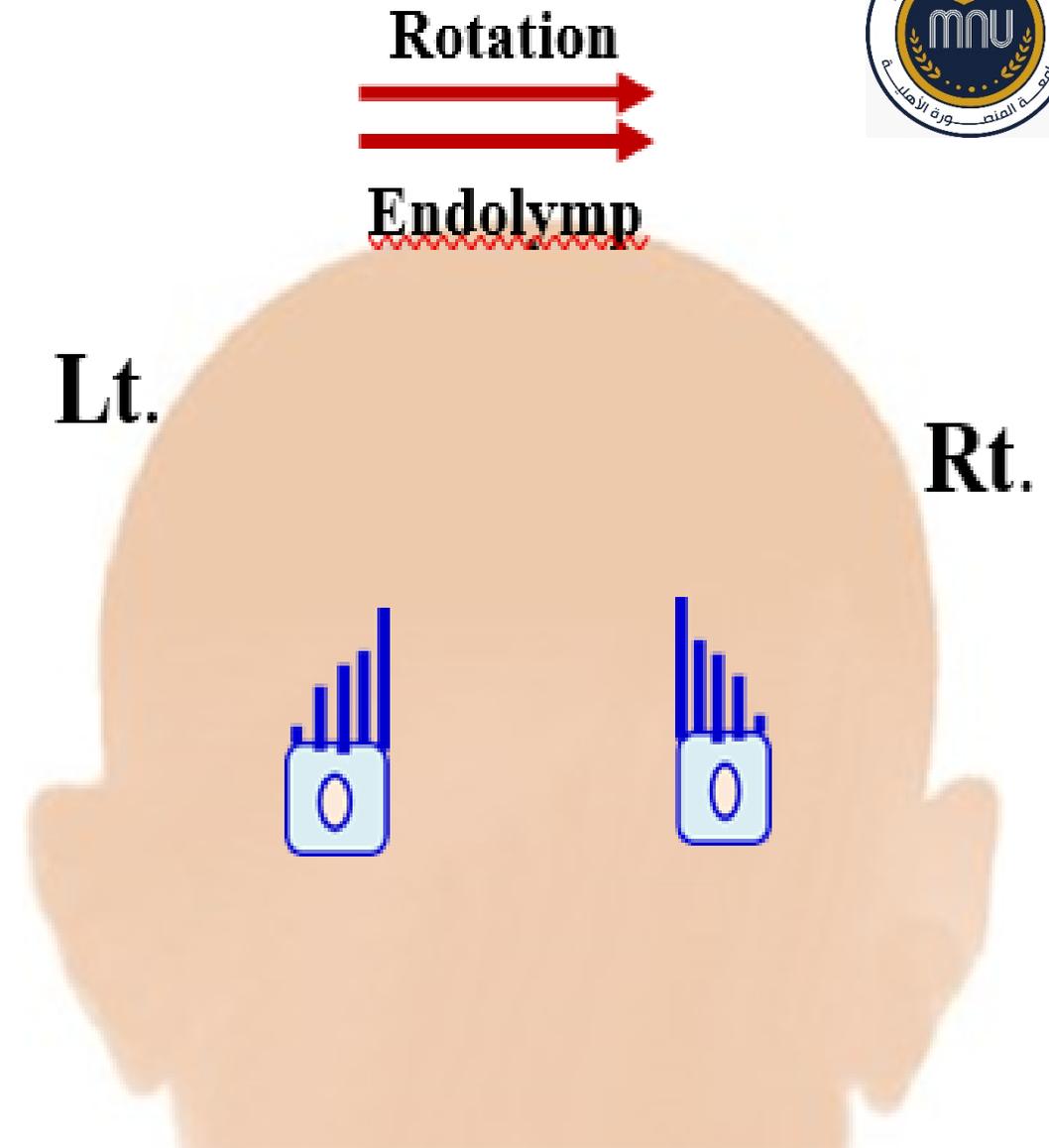
\*Bending of the cupula of the **left crista away from the utricle** i.e. bending the stereocilia away from the kinocilium → ↓ **the rate** of discharge from the **left crista**.

- **Asymmetrical discharge from both horizontal S.C.Cs with excessive discharge from the right** → when this pattern of discharge reaches the **CNS**, it evokes **sense of rotation to the right side**.



## c) When the rotation speed become constant:

**After 20 seconds, endolymph will gain the same speed as the SCC & the cupula return to the original position, and the rate of discharge return to symmetrical basal level → no feeling of rotation.**



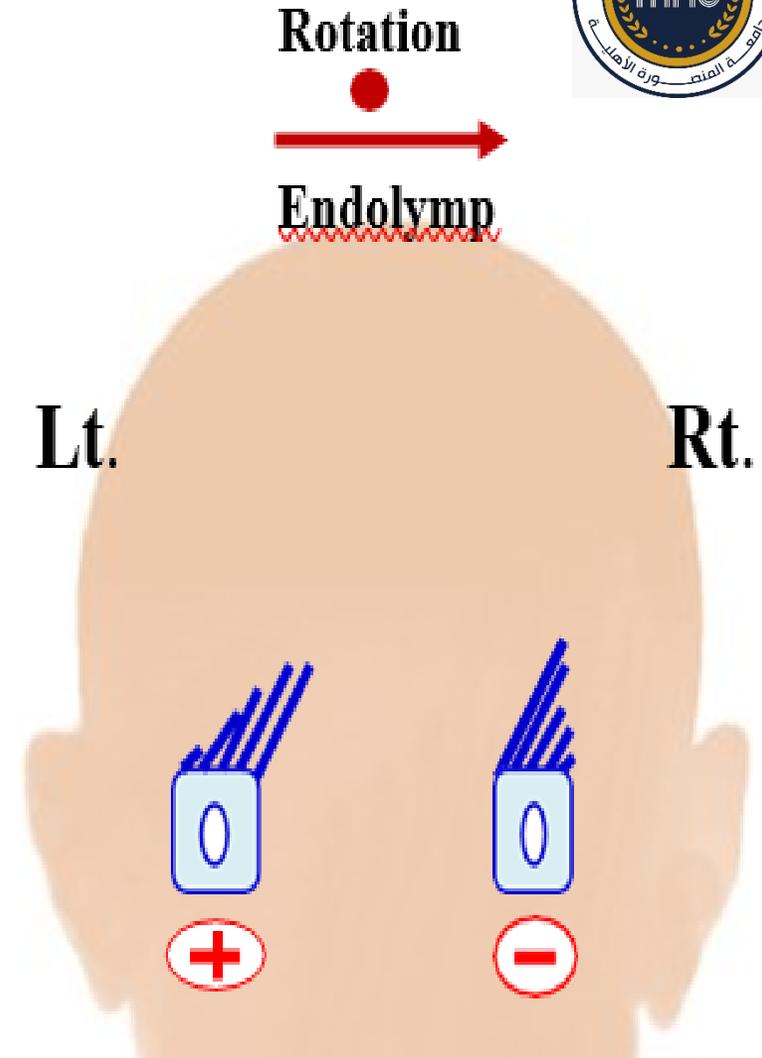
## d) On deceleration:

- The endolymph continues to move, due to its **momentum**, in the same direction of the **original rotation (Right side)** leading to:

\*Deflection of the cupulae of the **right crista away from the utricle** → bending of the stereocilia away from Kinocilium → ↓ the rate of discharge from the **right crista (inhibited)**.

\*Deflection of the cupulae of the **left crista away from the utricle** → bending of the stereocilia towards Kinocilium → ↑ the rate of discharge from the **left crista (stimulated)**.

- **Asymmetrical discharge from both Horizontal S.C.Cs with excessive discharge from the left** → when this pattern of discharge reaches the **CNS**, it evokes **sense of rotation to the left side** → **false sensation of counter-rotation to the left (Vertigo)**.





# Effects of stimulation of S.C.Cs

➤ If the S.C.Cs are stimulated **asymmetrically** and **excessive** as in: (1) exposed to unusual pattern of motions e.g. after prolonged & rapid rotation, (2) in certain diseases e.g. Menier's disease, **the following manifestations will occur:**

1- Vertigo

2- Nystagmus

3 Autonomic reactions

4- Changes in the muscle tone.



# 1- Vertigo

➤ **Def:** sense of rotation of either the subject or his surroundings.

➤ **Causes:**

## 1- Post- rotational vertigo:

- Remains for about 20-30 seconds.
- Disturb the equilibrium.

## 2- Spontaneous (i.e. without rotation); It may be due to:

- a) Excessive irritation of S.C.Cs. e.g. in labyrinthitis
- b) Irritation of pathway of vertigo (e.g by brain tumours).
- c) Exposure to unusual pattern of movement.



## 2- Nystagmus

➤ Def: oscillatory movement of the eye balls.

➤ Causes:

### A. Rotational nystagmus:

- Due to rotation and occurs:

- a) At the **onset** of rotation (for 20-30 sec.)
  - b) **Post-Rotational nystagmus** (for 20-30 sec.)
- } due to asymmetrical discharge from SCCs.

- Significance:

- a) At the onset of Rotation: help in maintaining clear vision during rotation → maintaining the body equilibrium.
- b) Post-Rotational nystagmus: - It may disturb the equilibrium.



## B. Spontaneous nystagmus:

- Due to:

- \* irritation of the labyrinth as in Minier's disease.
- \* Neocerebellar Syndrome.
- \* Severe defective vision (e.g. in blind)

## C. Optokinetic nystagmus:

- Occurs during looking out from a moving vehicle, e.g. train.



## 3- Autonomic reactions

- The exposure to unusual motion pattern → a group of manifestations called (Motion Sickness). These manifestations include:
  - 1- Bradycardia.
  - 2- Hypotension.
  - 3- Nausea & vomiting.
  - 4- Profuse sweating.
- This is due to the effect of vestibular apparatus on the autonomic centers in the brain stem reticular formation.



# Thalamus



# Thalamus

- ❑ It is an ovoid gray mass located in the diencephalon.
- ❑ The two thalami of both sides are separated by the 3rd ventricle with interconnection by a narrow band called interthalamic adhesion.
- ❑ The gray matter of the thalamus is divided internally by lamina of white matter into many separate nuclei.



# Thalamic nuclei

Functionally, thalamic nuclei are divided into:

1- Specific nuclei: Receive well defined afferents and project to specific areas in the cerebral cortex. They include:

i- Anterior nuclei.

ii- Medial nuclei.

iii- Lateral nuclei



**2- Non-specific nuclei:** **Receive** input mainly from **R.A.S**, and **project** diffusely to **all parts** of the neocortex. **They include:**

**i- Intralaminar nuclei.**

**ii- Midline nuclei.**

**iii-Reticular nuclei.**

# Functions of thalamus



## 1-Sensory functions

### A) Thalamus acts as a sensory relay station:

**All ascending sensory pathways** before reaching the cerebral cortex relay in the thalamus especially those carrying **fine epicritic sensations** from **opposite** side of the body.

*i) Somatic sensory pathways* relay in **ventral posterior (VP)** nucleus of the **opposite** side, where those



- from **body** → relay in **lateral part of VP** nucleus
- from **face & taste** → relay in **medial part of VP** nucleus

*ii) Special sensations:*

- **Vision:** the optic fibers from both **retinae** reach **L.G.B.** and projected to the **visual cortex**.
- **Hearing:** auditory fibers from both **ears** relay in **M.G.B.** before reaching the **auditory cortex**.

## **B) Gating of the ascending sensory information:**

- **Corticofugal impulses** are transmitted back from the **cerebral cortical sensory areas** to the **thalamic relay nuclei** which already project to these areas.
- **This cortical-feedback is inhibitory**, and it **decreases** transmission through these nuclei particularly when the **sensory input is very high**.



## C) Thalamus as a sensory center:

Discrimination of many sensory impulses occurs in the thalamus but the sensations felt are of crude protopathic nature e.g: diffuse pain & crude touch.

## 2- Motor functions:

- Ventral anterior (VA) and ventral lateral (VL) nuclei relay motor signals from the **basal ganglia** and **cerebellum** to the **motor** and **premotor areas** of the frontal lobe to control their functions.
- V.P.L nucleus relays **proprioceptive** signals to the **motor** cortex. This provides sensory information about **position** and **movements** of the different parts of the body.

### 3- Association & integration functions:

- Anterior and medial thalamic nuclei together with hypothalamus and limbic system play a role in integrating the visceral and somatic motor responses evoked during **emotions**.
- The reciprocal connection between the dorsomedial (D.M) nucleus of the thalamus and prefrontal areas may play a role in the coding, storing and recalling of memory.



## 4- Arousal function:

- The non-specific thalamic nuclei receive excitatory signals from the R.A.S of brain stem, and project it to almost all areas of the cerebral cortex, producing **arousal** and **wakefulness**.



# Wakefulness (Arousal)

➤ Def.: The state of **consciousness** in which the person is aware of the various **sensory stimuli**, as well as, **his feelings, thoughts and ideas**. It also enables the person to **pay attention to external stimuli**.

## ➤ Reticular activating system (RAS):

- The RAS consists of facilitatory reticular formation (in **pons**) with its upward projections to the non-specific thalamic nuclei, from which the excitatory signals project diffusely to all areas of the neocortex, causing generalized activation of the brain.
- This activation of the brain produces arousal and wakefulness.



# ➤ Factors affecting activity of R.A.S

## 1-Sensory signals:

- Sensory signals from every sensory system in the body produce activation of RAS neurons.
- **Pain and proprioceptive** signals are **stimulatory** than others.



## 2-Feedback signals:

- Feedback signals from the cerebral cortex are sent back to RAS, whenever the cortex becomes activated by **thinking or motor activity**.
- This corticofugal feed-back activates **R.A.S** which in turn sends more excitatory signals to the **cortex** to enhance its activity.



## 3-Neurotransmitters:

- Epinephrine and norepinephrine: Lower the threshold for excitation of neurons of the R.A.S.
- Serotonin: Released mainly by the neurons of raphe nuclei in medulla. It suppresses R.A.S and could play a role in sleep. Administration of anti-serotonin drugs → insomnia.

## 4-Reticular inhibitory area (RIA):

- Located in medulla oblongata.
- When this area becomes excited, it inhibits RAS by increased secretion of serotonin.
- ❖ The normal wakefulness-sleep cycles may be regulated through **alternating reciprocal** activity in between **RAS** and **RIA**.



## 5-Drugs:

- Sympathetic drugs: as amphetamine and adrenaline increase the activity of RAS (explain alert state during emergency and stress).
- Anesthetic drugs: ↓ conduction through RAS synapses → unconsciousness.



# Cerebellum

# Internal structure of cerebellum



## Anatomically,

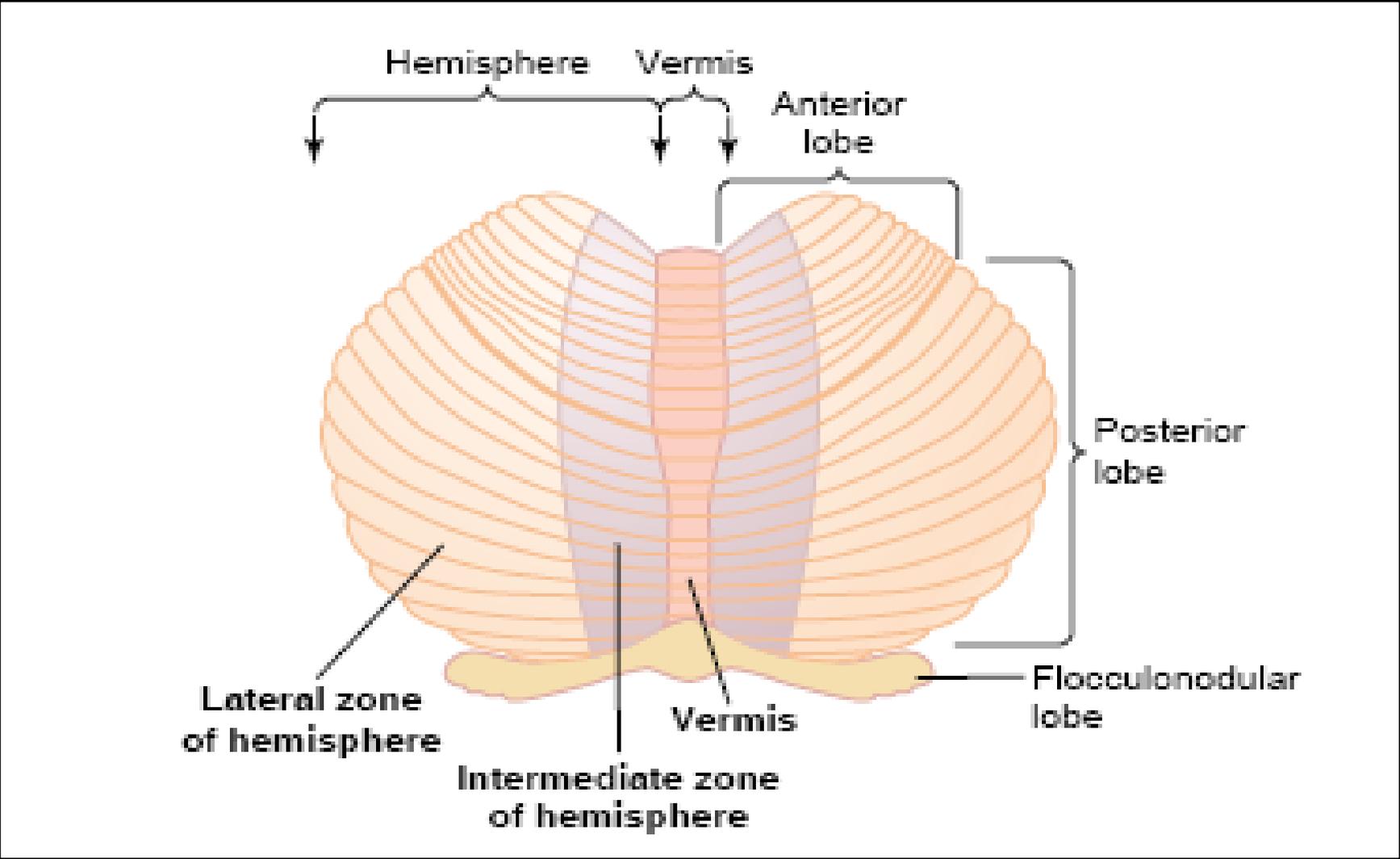
- The cerebellum is composed of an outer cortex surrounding a core of white matter which consists of deep cerebellar nuclei (Dentate nucleus, Globose & emboliform nuclei and fastigial nucleus).

➤ Cerebellum is divided by **2 fissures** (primary and postero-lateral fissures) into **3 lobes:**

***1. Anterior lobe.***

***2. Posterior lobe.***

***3. Flocculo-nodular lobe.***





## Functionally,

Cerebellum divided into:

1- Vestibulocerebellum (flocculonodular lobe; **archicerebellum**): Composed of the flocculo-nodular lobe (F.N.L) which is closely related in its functions to the vestibular system

**2- Spinocerebellum** (vermis + intermediate zone; **paleocerebellum**): Consists of the vermis and the intermediate zones of the cerebellar hemispheres. Most of its sensory information comes from the spinal cord.

**3- Cerebrocerebellum** (lateral zone; **neocerebellum**): Composed of the lateral zones of the cerebellar hemisphere. Almost all of its afferent signals originate from the cerebral cortex and reach it through the pons.

# Vestibulo-cerebellum



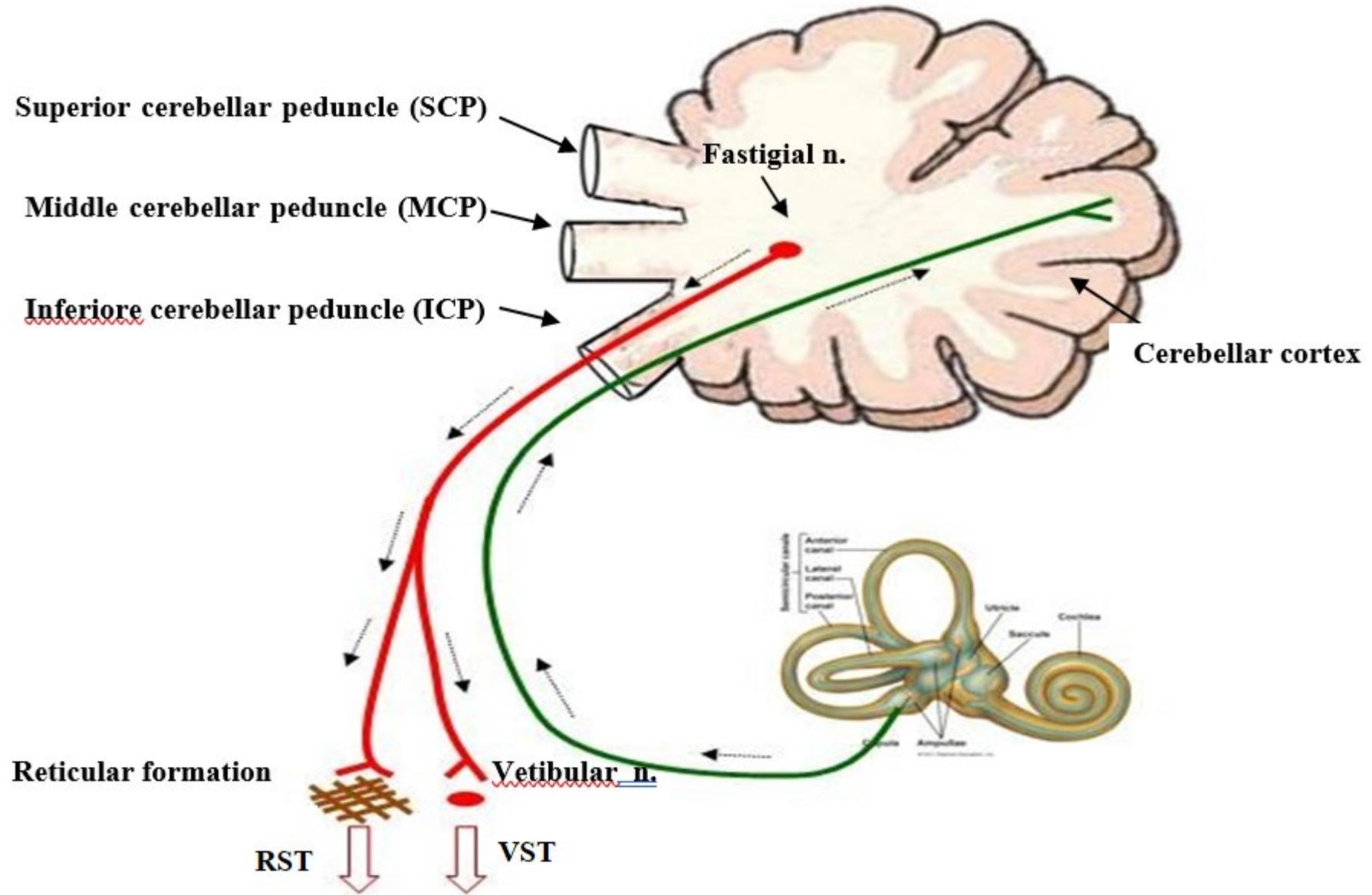
## □ Neural connections:

### ➤ Input or afferent:

Vestibular apparatus → **vestibular nuclei** → inferior cerebellar peduncle → **cortex of F.N.L.**

### ➤ Output or efferent:

Cortex of F.N.L → **Fastigial nucleus** → inferior cerebellar peduncle → **Vestibular nuclei** → **Vestibulo-spinal and reticulospinal tracts** → adjust **tone of postural muscles.**





## □ Functions:

### 1. Control of equilibrium:

➤ Disturbed equilibrium or altered head position → stimulation of vestibular receptors → **Vestibulo-cerebellum**.

➤ Vestibulo-cerebellum interpretes these impulses then immediately sends **corrective signals** → **Fastigeal N** → then through:

*a) Vestibulospinal tract (VST) & reticulospinal tract (RST)* → motor neurons of **axial muscles & proximal limb muscles** → affect their **muscle tone** → **maintain body posture**.

*b) Medial longitudinal bundle (M.L.B)* → **III, IV & VI cranial nuclei** → **coordinate eye movement** with head movement → **maintain clear vision** → **keep equilibrium**.

## □ Cerebellar lesions:

### Flocculo-nodular Lobe disorders:

- These disorders impair equilibrium.
- This is manifested by swaying during standing and unsteady wide based gait (**waddling gait**).



# Spino-cerebellum

## □ Neural connections:

### ➤ Input or afferent:

#### a- Brain & brain stem centers:

- 1- Cerebral cortex.
- 2- Red nucleus.
- 3- Reticular formation

Impulses from these centers inform cerebellum about the **plan of movement ordered by the higher centers.**

**b- Proprioceptors:** Ventral spinocerebellar tract (VSCT) & lateral spinocerebellar tract (LSCT) carry impulses from proprioceptors regarding the **actual performance of muscles.**



## ➤ Output or efferent:

### i. From vermis:

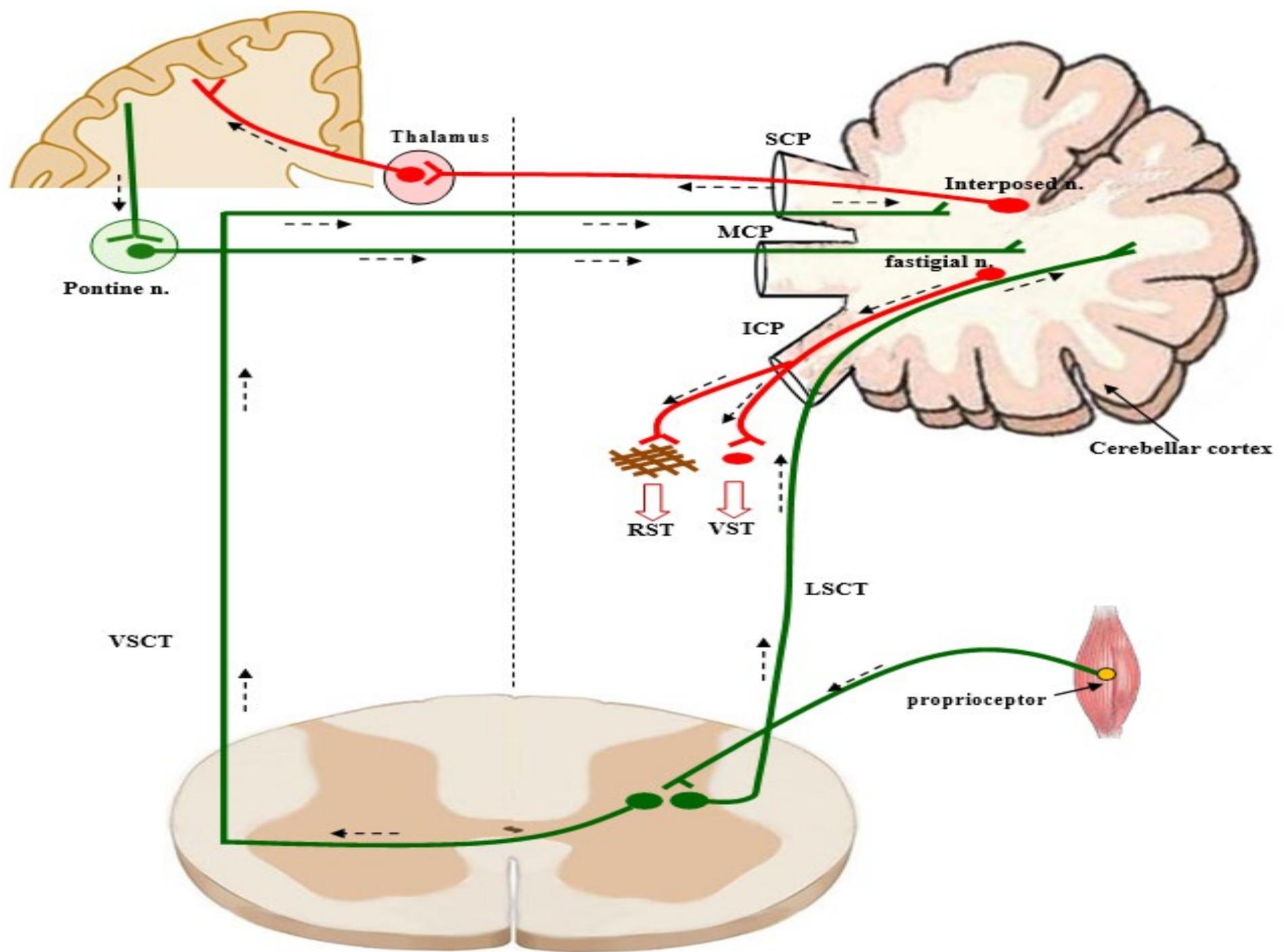
vermis → fastigial nucleus → reticular formation & vestibular nuclei  
→ vestibulo-spinal (VST) & reticulo-pinal tracts (RST) → antigravity muscles.

### ii. From paravermal (intermediate) zone:

• Intermediate zone → interposed nucleus (globose and emboliform)  
→ superior cerebellar peduncle →

**1) Contralateral thalamus** → motor cortex → corticospinal tract → distal limb muscles.

**2) Contralateral red nucleus** → rubro-spinal tracts → distal limb muscles.



## □ Functions:



### I. Vermal zone: Regulation of body posture:

- It receives information from proprioceptors about position and movements of every part of the body .
- Disturbed body posture → stimulation of proprioceptors → vermis.
- Vermis interpretes these impulses, then immediately sends **corrective impulses** → **fastigial N.** → then through **VST & RST** → adjusts the **tone of antigravity muscles** → maintain posture against the effect of gravity.

## II. Paravermal (intermediate) zone: *Regulation of voluntary movement:*



### 1. Servo-comparator function (Fine-tuning)

#### ➤ *Spino-cerebellum is informed about:*

- a) The intended plan of movement from the motor cortex (via cortico-ponto-cerebellar tract), and
- b) The performance of movement from muscles (via spino-cerebellar tracts).

- Spino-cerebellum compares the intention of the motor cortex with the actual performance of the muscles.
- If not appropriate, the spino-cerebellum sends corrective signals to the **motor cortex**, which by its turn through descending **CBS** tract adjusts the **muscle activity**.



## 2. Predictive & damping function:

- *Damping means* ending of the movements without oscillation at the proper site
- The cerebellum assesses the rate of movement, calculates the time needed to reach the intended point & then transmits inhibitory impulses to the motor cortex to stop the movements at the exact intended point.

## □ Cerebellar lesions:

### Vermal disorders:

- These disorders cause inability to sustain the upright posture, due to failure to adjust the tone of antigravity muscles.

# Cerebro-cerebellum



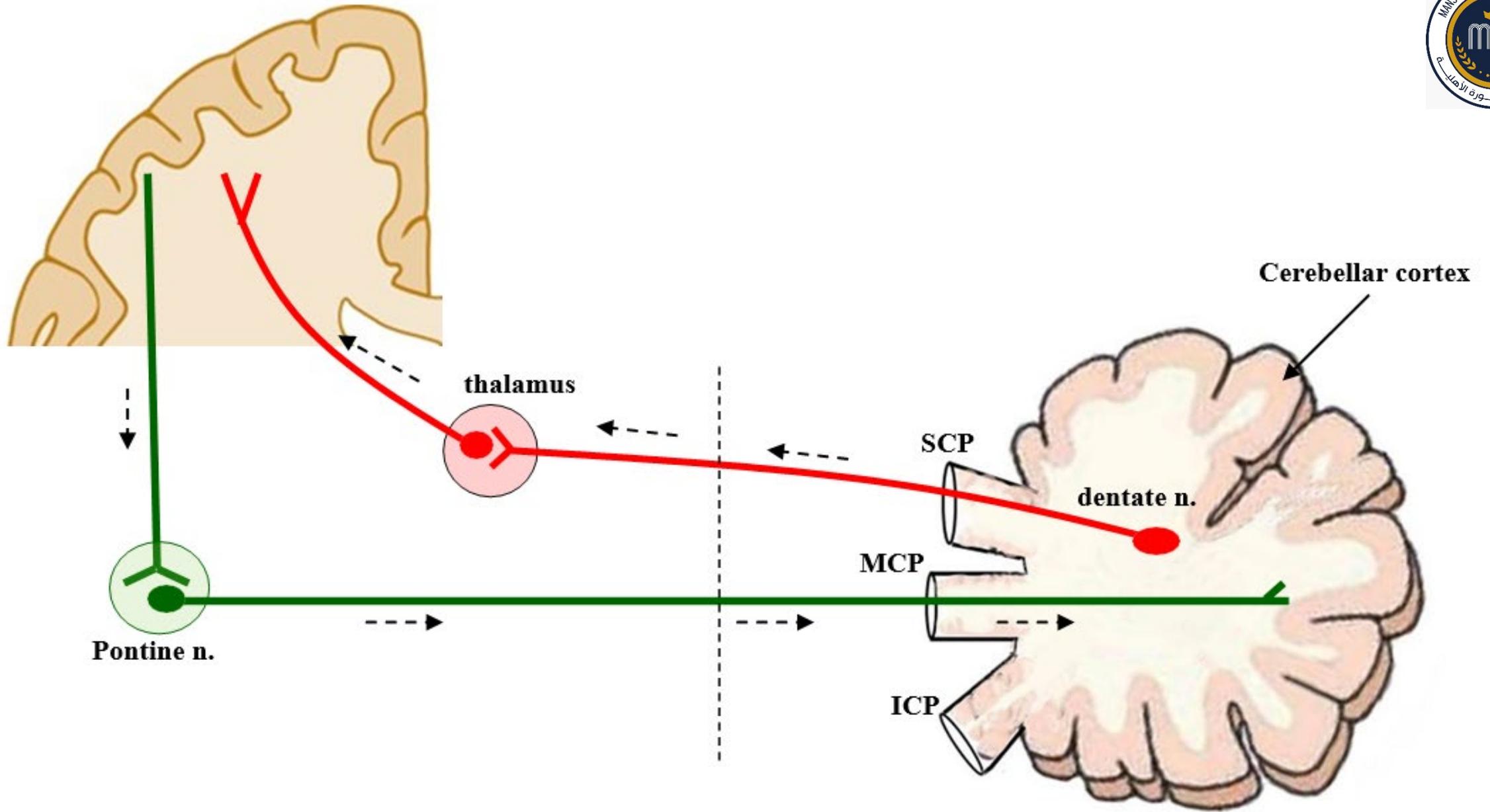
## □ Neural connections:

### ➤ Input or afferent:

- Cerebral cortex → pontine nuclei → middle cerebellar peduncle via cortico-ponto-cerebellar pathway.
- *This pathway provides the cerebellum with:*
  - 1) The plan of movement.
  - 2) Sensory information regarding postural state of the body.

## ➤ Output or efferent:

- Cerebellar cortex of lateral zone → dentate nucleus → superior cerebellar peduncle → VL nucleus of contra-lateral thalamus → cortical motor areas.
- *This pathway mediates* the role of the cerebellum in **adjustment of motor commands** before being discharged from cerebral cortex.



# □ Functions: Regulation of voluntary movement:



## (I) Predictive (planning and timing) function:

### a) Sequence planning:

- The cerebro-cerebellum receives afferent impulses from the **cortical association areas** (the site of ideas for voluntary movements) about the **intension** of performing a certain voluntary movement **before** the movements starts.

- Then, the cerebro-cerebellum sends to the **motor area of cerebral cortex** (that initiate movements) the **plan of the sequential movements** required to execute the intended voluntary movement.
- The cerebro-cerebellum **plans for the next movement** while the present movement is occurring.
- Thus, it is not involved with what is happening at a given moment, but with what will occur in the **subsequent moment**.

## **b) Timing planning:**

- The cerebro-cerebellum provides **proper timing for each movement.**
- Such function is necessary for **smooth transition** from one movement to the next and **joining** of sequential movements.

# Neocerebellar syndrome

- ❑ Cause: cerebellar hemispheric (paravermal and lateral zones) lesion.
- ❑ The principal motor dysfunctions caused by cerebellar hemispheric lesions include:
  - I. Hypotonia: Due to decreased supraspinal facilitation of stretch reflex (muscle tone).
  - II. Asthenia: Leading to weakness of movements
  - III. Ataxia (asynergia): refers to the **incoordination** of voluntary movements in **absence** of UMNL and LMNL.

## Manifestations of ataxia:

### 1-Rebound phenomenon:

- When the patient flexes his forearm strongly against a resistance, then the resistance is **suddenly removed**, the patient **cannot stop** inward movement of his forearm in proper time, and may thus strike his body.
- This is due to **failure of the “damping” function.**

### 2-Dysmetria:

- The moving limb usually **overshoots** the intended point (**hypermetria**), due to **failure of “damping” functions.**



### **3-Intention (Kinetic) Tremors:**

- Occur during voluntary movements.
- Due to hypermetria of the acting muscles.
- Thus, the limb oscillates throughout the whole movement.

### **4-Nystagmus:**

- Represents the kinetic tremors of the extra-ocular muscles during movements of the eye.

## **5-Unsteady Gait:**

- **Unsteady drunken gait** due to **dysmetria** and **kinetic tremors** of the lower limb muscles.

## **6-Decomposition of complex movements:**

- A voluntary motor act is carried out as **several successive steps**, due to **failure of sequence and timing planning**.
- The patient cannot perform movement that involve **two joints** at the same time **e.g.** touching the knee of one leg with the heel of the opposite foot (**heel-knee test**).

## **7-Dysdiadochokinesia:**

- The patient is **unable to perform rapid successive opposite movements**, e.g. supination & pronation of the hands.
- Movements become **slow** and **irregular**, due to **failure of sequence and timing planning**.

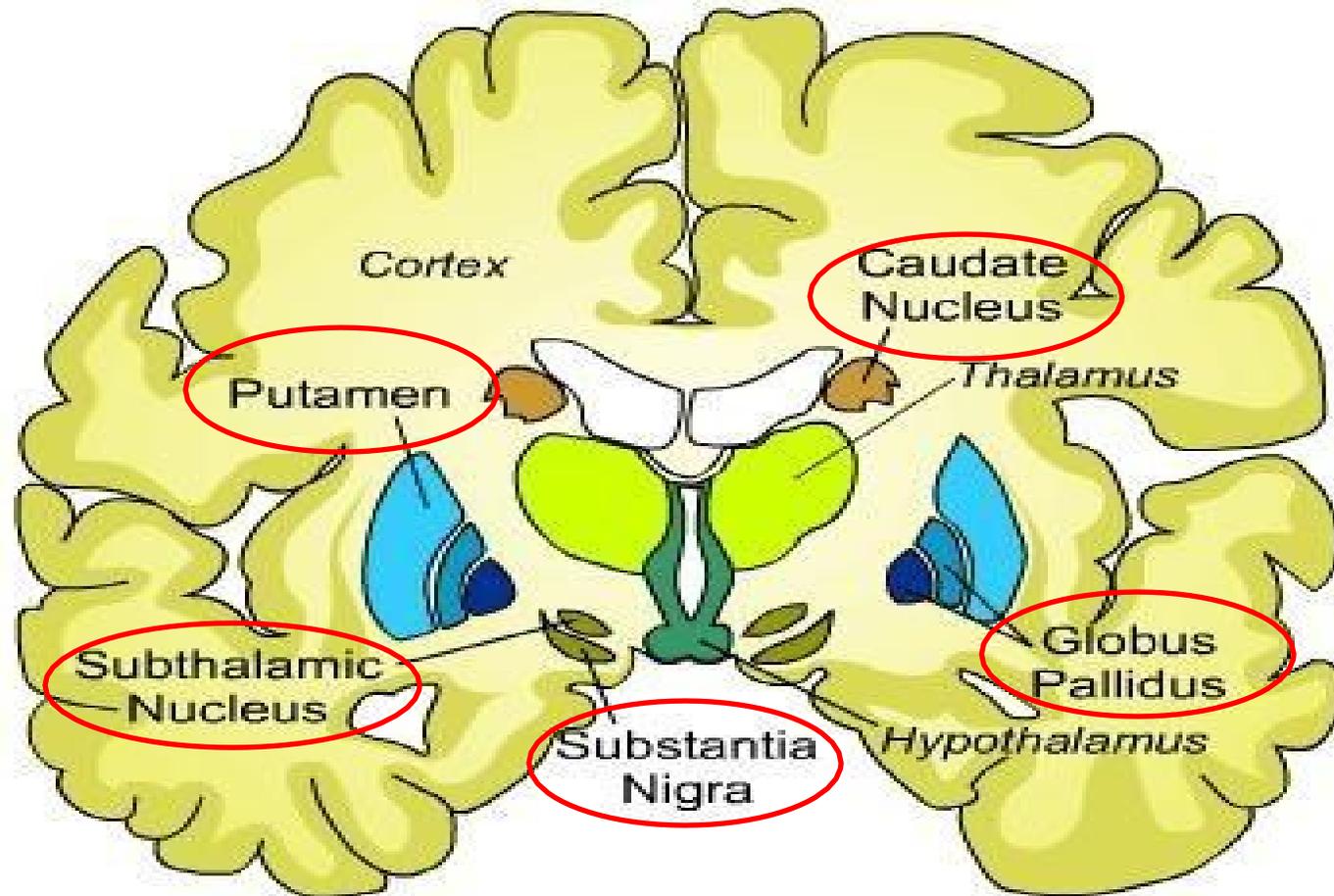
## **8-Scanning speech (Dysarthria).**

- Speech becomes **slow** and **decomposed**.
- Each word is fragmented and pronounced as **several separate syllables**.
- Due to **failure of sequence and timing planning**.

# Basal Ganglia

## Def,

- The BG are elements of the **motor system ??** that play a principal role in the **initiation and regulation** of motor activity.



# Basal Ganglia

**Functionally**, the basal ganglia consist of the following nuclei ;

## 1) Telencephalic nuclei:

a) Caudate Nucleus

b) Putamen

c) Globus Pallidus

- The caudate and putamen → together are called **Corpus Striatum**.
- **Globus Pallidus** is subdivided into **internal (Gpi)** and **external (Gpe)** segments

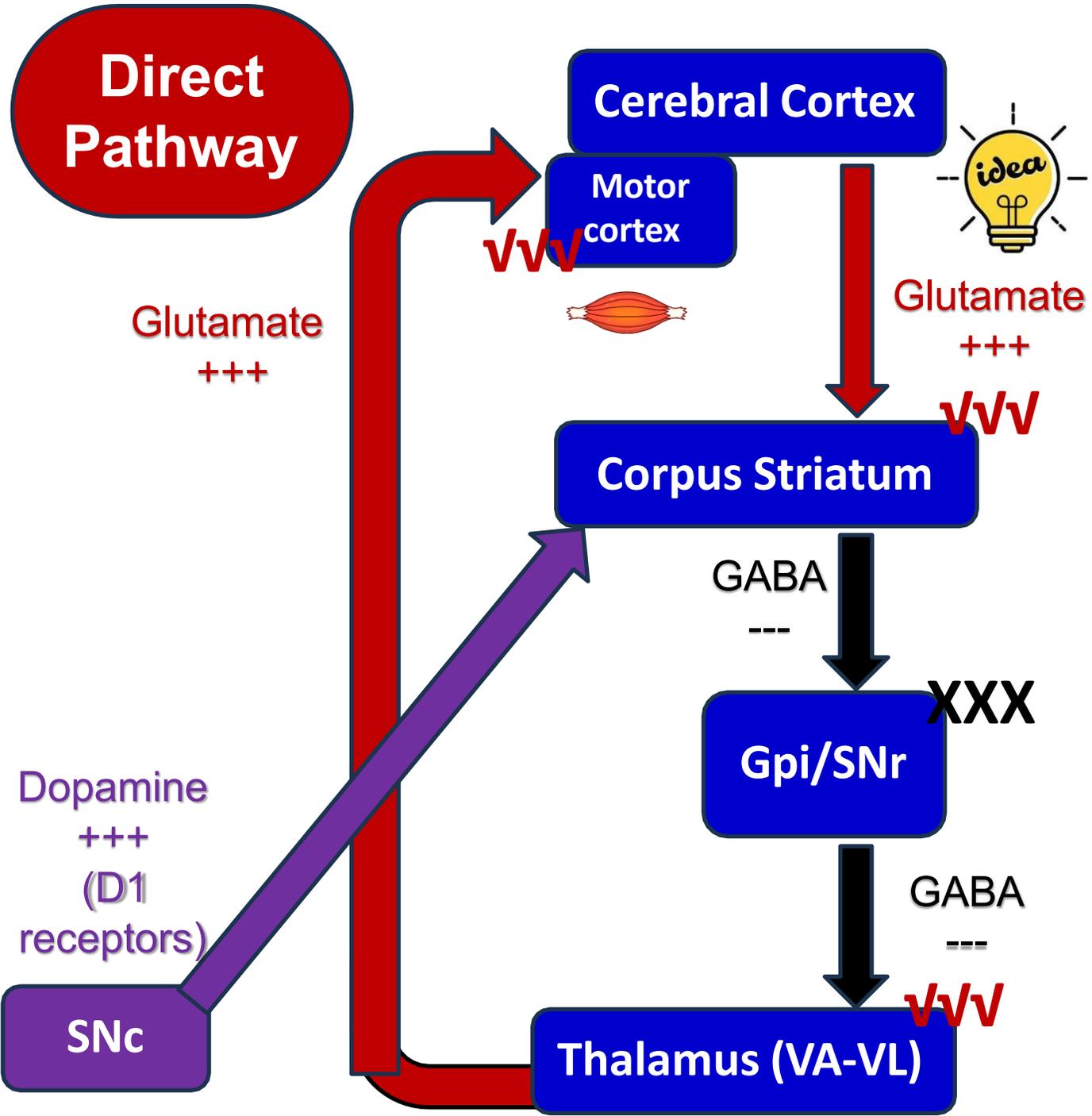
# Basal Ganglia

## 2) Diencephalic nuclei:

**Subthalamic Nucleus** is located in the diencephalon below the thalamus.

## 3) Mesencephalic nuclei:

- **Substantia nigra**; is located in the mesencephalon (midbrain)
- Histologically, it is subdivided into 2 regions,
  - a) **Substantia nigra pars compacta (SNc) secrete dopamine**
  - b) **Substantia nigra pars reticulata (SNr) secrete GABA and act as Gpi**

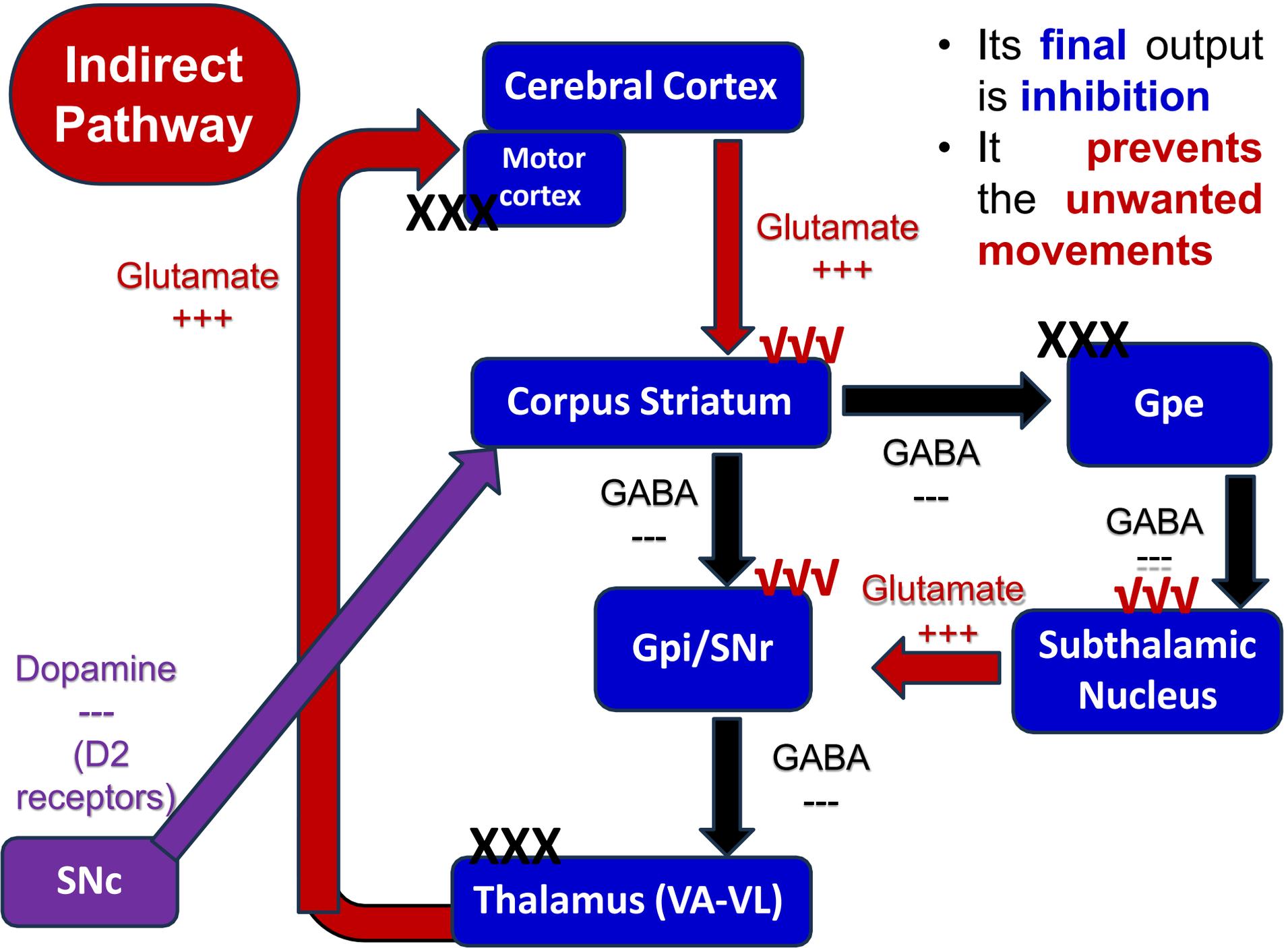


- Its **final** output is **excitation**
- It **initiates** the **movements**

## Direct Pathway

In summary, we have the following connections:

- **The cortex stimulate the corpus striatum.** Corpus striatum **suppresses** the activity of **the globus pallidus interna** .
- The result of this is **a reduction of the inhibitory influence** that the globus pallidus has **over the thalamus**, so-called disinhibition of the thalamus, which is equivalent to the **excitation** of **the motor cortex**.
- So, **the final function** of the direct pathway of the basal ganglia is to **excite the motor cortex to initiate certain motor activity**.
- **Dopamine** from **Substantia nigra pars compacta (SNc)** **stimulate D1 receptors on the corpus striatal** neurons of the direct pathway leading to **more stimulation of the direct pathway**.



## Indirect Pathway

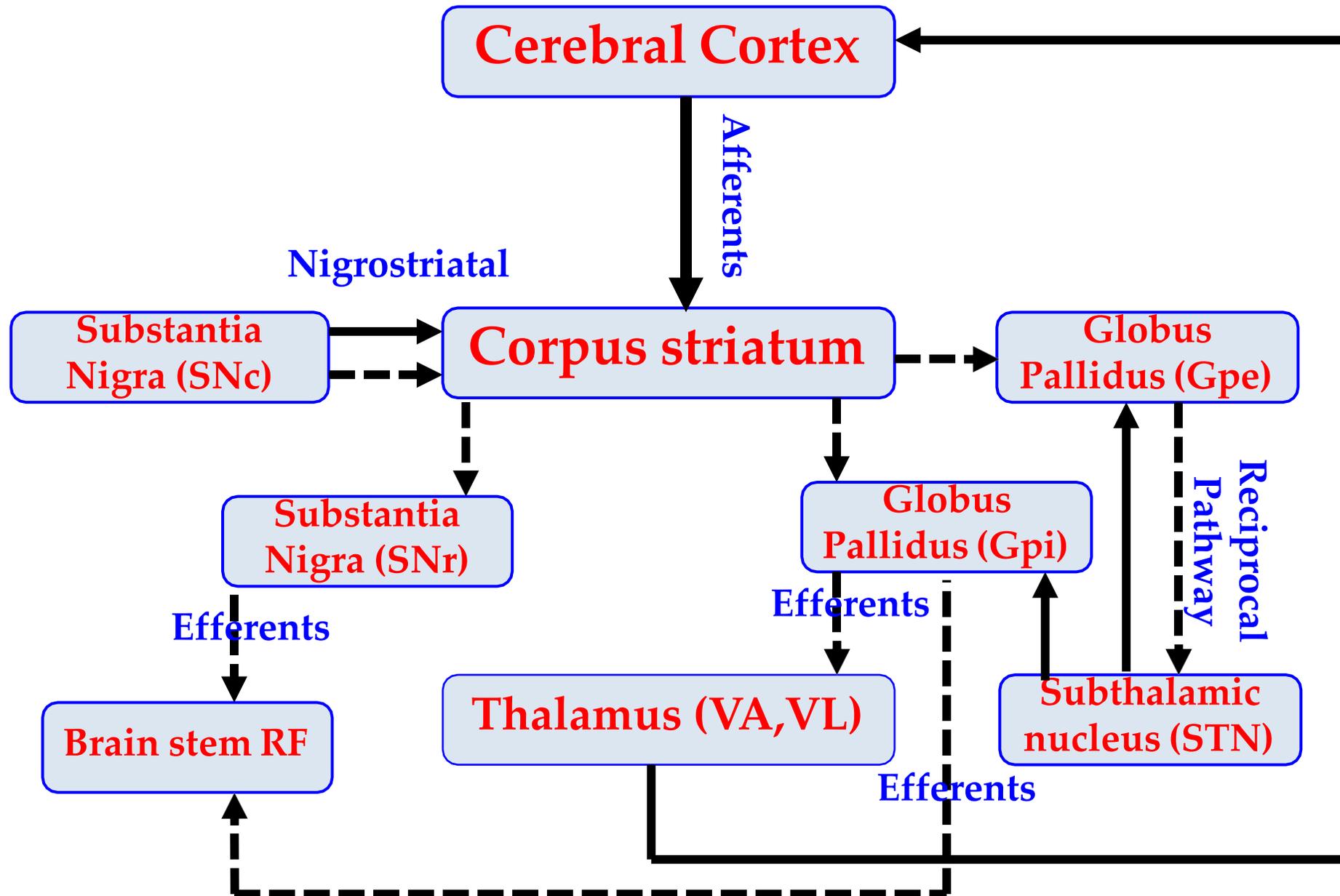
In summary, we have the following connections:

- **The cortex stimulate the corpus striatum.** Corpus striatum **suppresses** the activity of **the globus pallidus externa** .
- The result of this is **a reduction of the inhibitory influence** that the globus pallidus has **over the subthalamus**, so-called disinhibition of the subthalamus, which stimulate **the globus pallidus interna**. Leading to **inhibition** of the **thalamus and the motor cortex**
- So, **the final function** of the indirect pathway of the basal ganglia is to **inhibit the motor cortex to suppress the unwanted movement**.
- **Dopamine** from **SNC stimulate D2 receptors on the corpus striatal** neurons of the indirect pathway leading to **inhibition of the indirect pathway ???**.

# Neural connections of the Basal Ganglia

	From	To
Afferent connections	Cerebral cortex	Corpus striatum (the principal entry gate) → Corticostriatal pathway
Efferent connections	<p>i) Main efferent pathway → from internal segment of the globus pallidus (GPi) and SNr</p> <p>ii) Efferent pathways from the Gpi and SNr</p>	<p>i) To the VA and VL nuclei of the thalamus → in turn project to the frontal motor areas and the prefrontal region of the CC.</p> <p>ii) To brain stem reticular formation</p>
Internal connections	<p>i) A pathway from the Striatum</p> <p>ii) A reciprocal pathway ( ) the globus pallidus &amp; the subthalamic nucleus.</p> <p>iii) A nigrostriatal pathway → from the substantia nigra</p>	<p>i) To the globus pallidus</p> <p>ii) A reciprocal pathway ( ) the globus pallidus &amp; the subthalamic nucleus.</p> <p>iii) To the striatum</p>

# Neural connections of the Basal Ganglia



# Neurotransmitters of BG

Transmitter	<u>Source</u>	<u>Action</u>
<b>Glutamate</b>	Secreted from; i) <u>Neurons</u> which arise from the <b>CC</b> and terminate in <b>striatum</b> ii) <u>Neurons</u> from <b>subthalamic nucleus</b> that terminate in the <b>GPI and SNr</b>	<b>Excitatory transmitter</b>
<b>Dopamine</b>	Secreted from <b>neurons of the SNc</b> that terminate mainly in the <b>corpus striatum</b> → the nigrostriatal pathway	Has <b>2 different actions (modulatory)</b> : i) <b>Is excitatory</b> to neurons having <b>D1 receptors</b> → <b>potentiate</b> the effect of cortical glutamatergic input to the striatum in <b>the direct pathway</b> . ii) <b>Is inhibitory</b> to striatal neurons having <b>D2 receptors</b> → <b>antagonize</b> the effect of glutamatergic input to the striatum in <b>the indirect pathway</b>

# Neurotransmitters of BG

Transmitter	<u>Source</u>	<u>Action</u>
<b>Acetylcholine</b>	Found mainly in interneurons in the striatum ( <b>inhibit D1 neurons via M4 receptors and stimulate D2 neurons via M1 receptor</b> )	<b>modulatory</b>
<b>GABA</b>	<ul style="list-style-type: none"> <li>• Found in <b>striatal neurons</b> that project to the <b>Gpe , Gpi and SNr</b>.</li> <li>• Found in the neurons of <b>Gpe , Gpi and SNr</b></li> </ul>	<b>Inhibitory</b>
<b>Substance P</b>	Co-transmitter with <b>GABA</b> from <b>striatal GABA-ergic neurons</b>	<b>Inhibitory</b>
<b>Enkephalins</b>	Co-transmitter with <b>GABA</b> from <b>striatal GABA-ergic neurons</b>	<b>Inhibitory</b>

# Function of Basal Ganglia

1. Planning and programming of voluntary movements

2. Execution of learned patterns of movements

3. Regulation of posture

4. Regulation of muscle tone

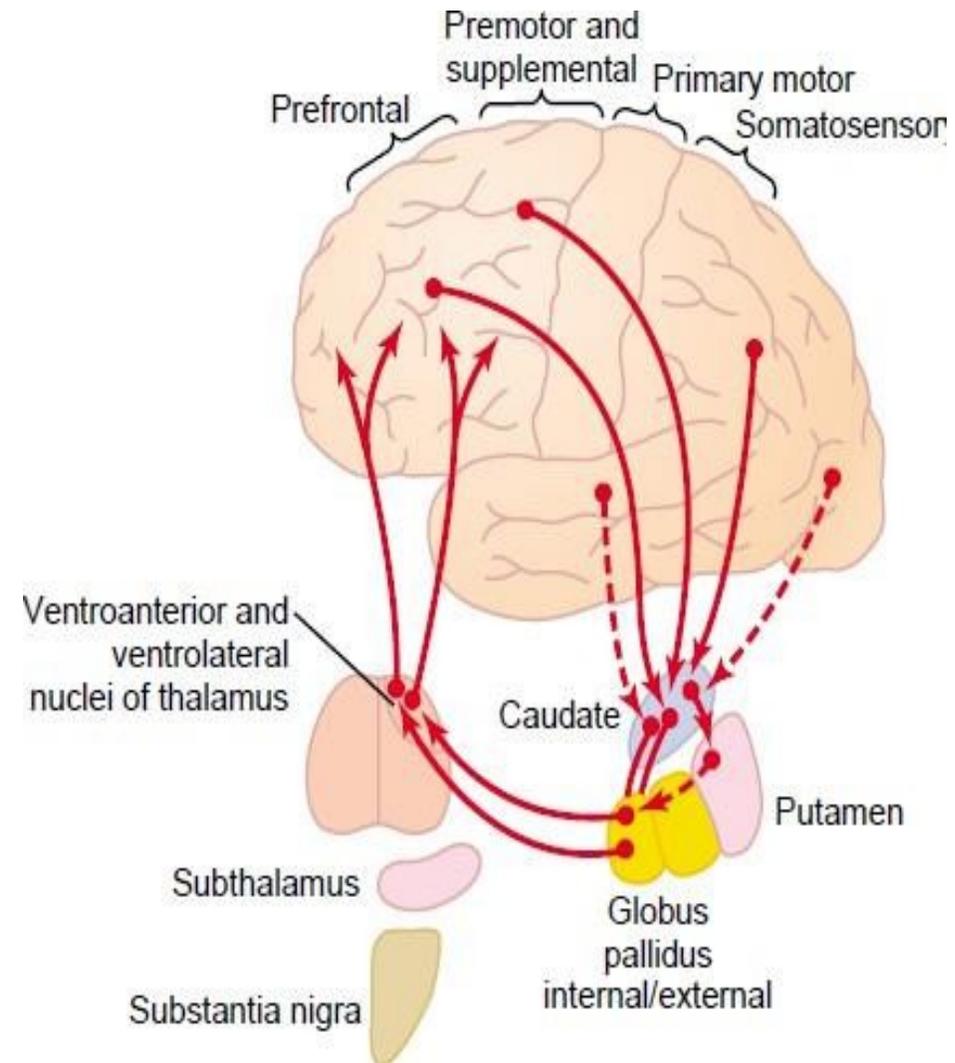
5. Initiation of automatic movements

# 1. Planning and programming of voluntary movements

- The BG plays a major role in **converting thoughts into voluntary motor acts**;
- This is done by **determining**:
  1. When move
  2. Where to move
  3. What ms to use for moving
  4. What is the direction of the movement
- The BG **does not function alone**, but in **close association** with;
  - a) **Cortical motor areas**: provide a preliminary plan of motor act
  - b) **Cortical association areas**: provide information about the **spatial relationship** of the different part of the body relative to each other and the **spatial orientation** of the body to the surrounding environment

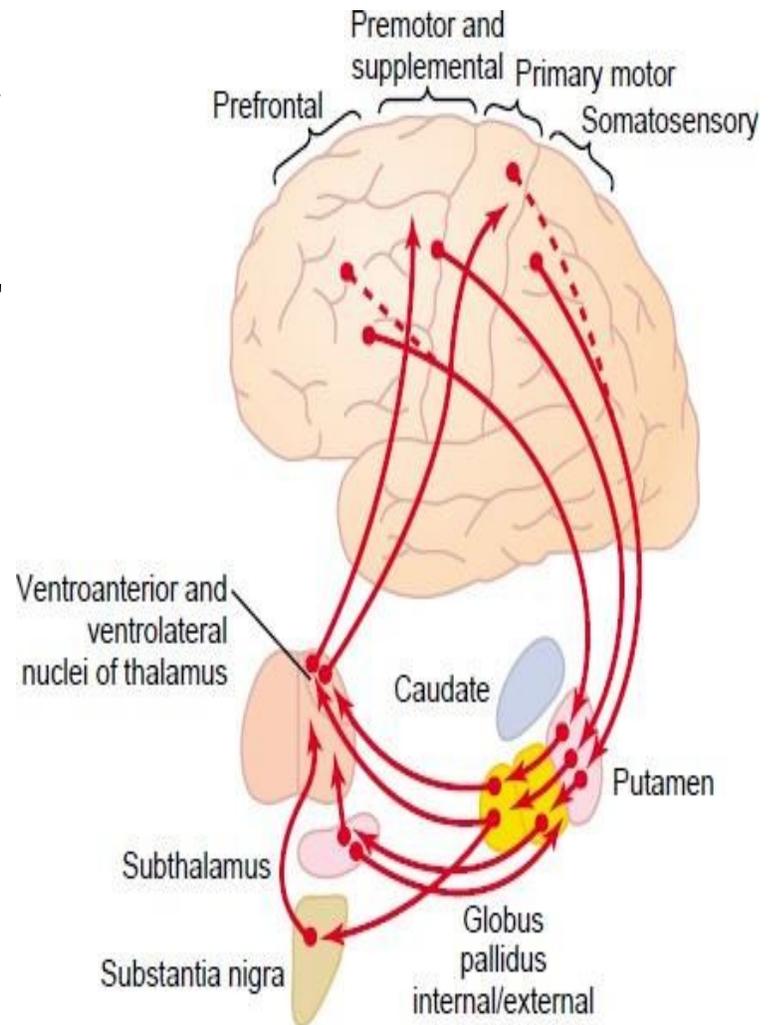
# 1. Planning and programming of voluntary movements

- This function is usually performed via **caudate circuit**
- Loss of this **cognitive function** of the BG makes the person **unable to make the proper decisions** about planning his motor acts under different circumstances



## 2. Execution of learned patterns of movements

- Many patterns of **learned or skilled movements** such as **writing , typing , and many hand skills** in addition to certain aspects of speech are performed so rapidly that it **could not allow for sensory feed-back control**.
- The patterns of these **rapid skilled movements** are established, after several trials (**training period**), in the motor system, then **stored in the BG**.
- **Execution of these movements** are controlled by BG through **putamen circuit**
- Damage of **BG** make **these movements crude** and as they done for the **first time**



### 3. Regulation of posture

- **BG provides a postural background**, which is **necessary** for the performance of voluntary **movements by** the **distal parts** of the limbs.
- That is, if the person wishes to perform a **skilled movement** by the hand, initiated by motor impulses mediated through the **corticospinal tract**, then the BG adjust the tone of the **trunk ms** and **proximal limb ms**, by way of its controlling influence on the **reticulospinal tracts**, to provide appropriate positioning of the shoulder and elbow as a **stable basis** for voluntary movements of the hand
- So, the **BG** constitute an **important bridge that link** the **lateral motor system**, with the **medial motor system????**



## 5. Initiation of automatic movements

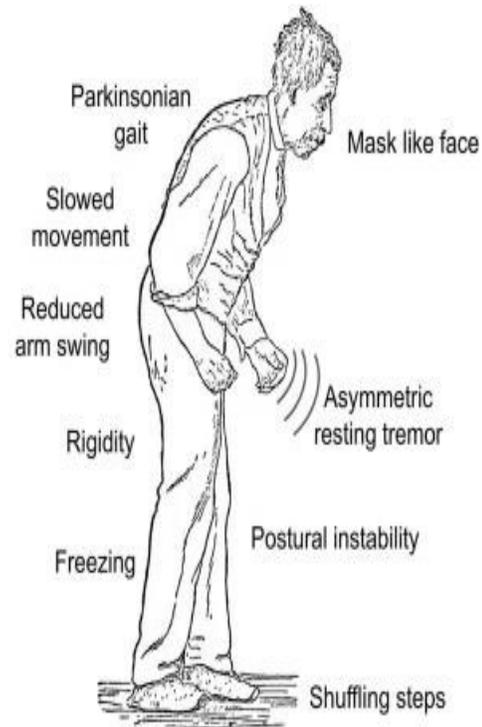
- The BG seems to initiate and regulate several patterns of **automatic movements** that are usually performed subconsciously.

-Such movements include e.g.

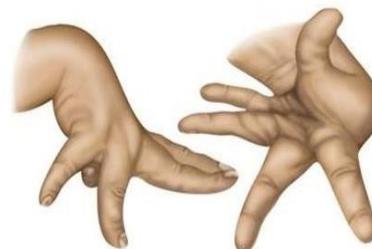
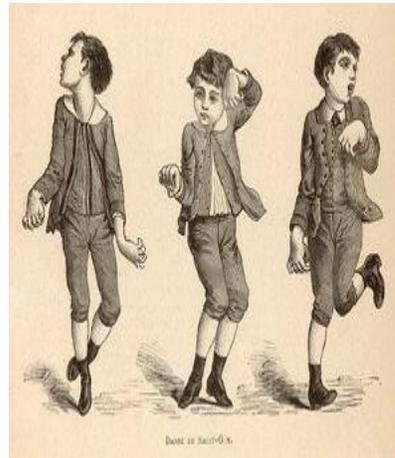
1. **Swinging movements** of the arms **during walking**,
2. **Facial expression** that reflect the **emotional state** of the subject.

# Disorders of Basal Ganglia

## Parkinsonism



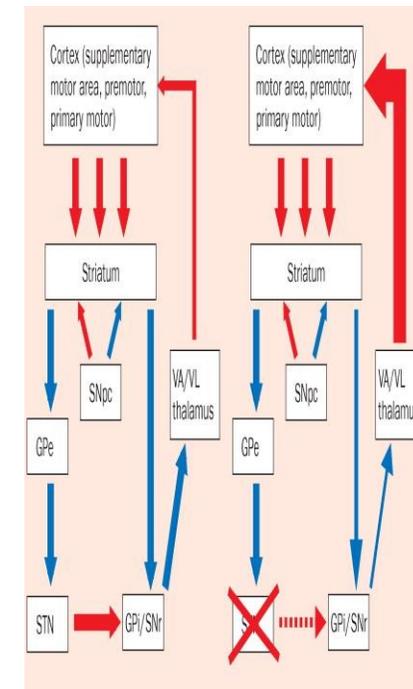
## Chorea



## Athetosis



## Ballismus



# Parkinsonism

- It is a **common degenerative disease** of the brain caused by a lesion in **nigrostriatal pathway** that characterized by

- a) **Tremors**
- b) **Akinesia**
- c) **Rigidity**

## Causes:

-Caused by **degeneration of the dopaminergic nigrostriatal neurons** → marked ↓ of dopamine release

-It may be;

- a) **Idiopathic (spontaneous)** → more common in old age (**Parkinson's disease**).
- b) **Non -idiopathic Parkinsonism** → Stroke, Viral encephalitis, CO and manganese poisoning, repeated head trauma and tranquilizers that block D2 receptors

# Parkinsonism

## Mechanism

- Degeneration of SNc will result in loss of dopamine and activation of inhibitory pathway and **inhibition of the motor cortex** resulting in **difficulty to initiate the movements** (hypokinesia) and **increased muscle tone**.

# Clinical signs of Parkinsonism

## 1) Tremors:

- Occur at rest i.e. **static tremors**
- ↑ by emotional excitement.

## 2) Akinesia and Bradykinesia:

- The voluntary movements show difficulty in initiating them**, when initiated become slow, of limited range stiff, and exhausting.
- The automatic movements are absent** e.g. facial expression lost (**a mask-like face**).
- Speech** becomes **slow and monotonous**.
- The gait** becomes **short shuffling**

## 3) Rigidity: (lead pipe and cogwheel)

- It is due to hypertonia of all skeletal ms (**alpha rigidity**)
- Rigidity affects flexors than extensors → **flexion attitude or gorilla like attitude**



# Disorders of Basal Ganglia

	B)Chorea	C)Athetosis	D) Ballismus
<b>Def.</b>	<p><b>Rapid</b> uncontrolled shaking or jerky (<b>dancing</b>) movements, which may involve <b>distal</b> parts of the body , e.g. hand &amp; face</p> <p>i) <b>Sydenham's Chorea</b> occurs in children with rheumatic fever</p> <p>ii) <b>Huntington's Chorea</b> : inherited disease occurs at the age of 30-50 years</p>	<p>Continuous <b>slow</b> writhing (twisting) movements which are particularly evident in the hands and fingers.</p>	<p>Spontaneous attacks of <b>violent</b> involuntary movements involving <b>large areas of the body</b></p>
<b>Cause</b>	<p>Atrophy and degeneration of the <b>caudate nucleus</b></p>	<p>Traumatic damage to the basal ganglia at birth (cerebral palsy)→involves <b>the striatum and globus pallidus</b></p>	<p>Damage of <b>the subthalamic nucleus</b></p>