SPASTICITY AND INCREASED MUSCLE TONE

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Note: Pure corticospinal tract lesion cause hypotonia instead of spasticity The reason is that pure pyramidal tract lesion is very very rare, and spasticity is due to loss of inhibitory control of extrapyramidal tracts.

OBJECTIVES

At the end of this lecture you should be able to

- Define spasticity ,rigidity and dystonia
- Describe the neurophysiology of spasticity
- Describe the features of upper and lower motor neuron lesions
- Describe the causes of spaticity
- Enumarate the effects of spasticity
- List the treatments to reduce spasticity

NEXT

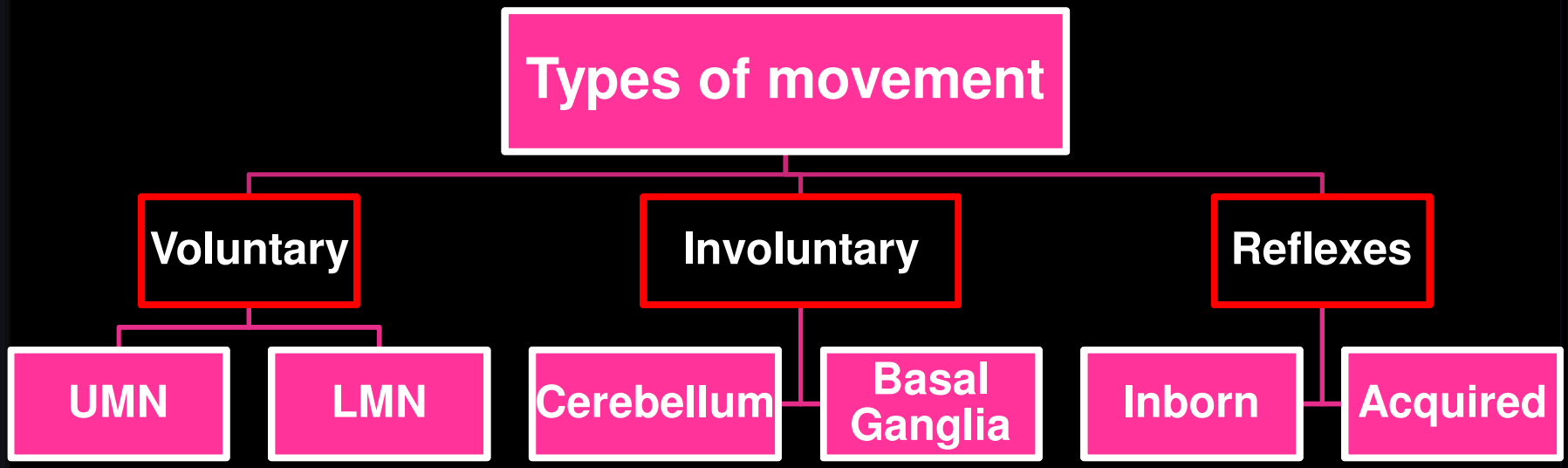
MOTOR SYSTEM LAYOUT

Motor = movement

For movement we need nervous system and muscles.

Components of motor system are:

- Motor cortex
- Upper and lower motor neurons
- Cerebellum
- Basal ganglia
- Spinal cord
- Muscles



INITIATOR OF MOTOR ACTIVITY

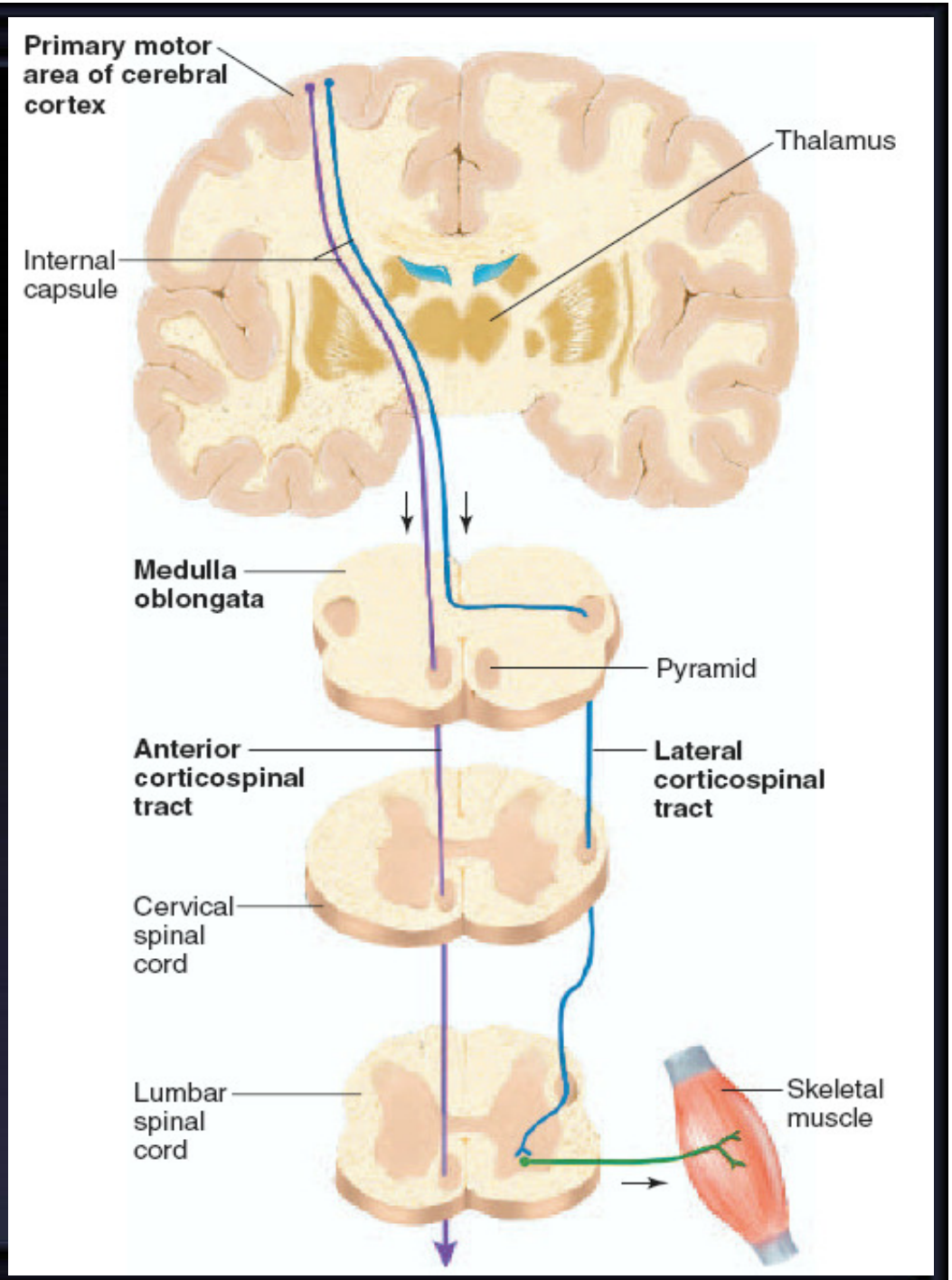
Corticospinal tract (Pyramidal tract)

REGULATOR OF MOTOR ACTIVITY

Cerebellum (Extrapyramidal tracts)

PYRAMIDAL TRACTS

- Originating Cells: Giant pyramidal cells
- 80% to 90% of the pyramidal fibers then cross in medulla to the opposite side and descend into the lateral corticospinal tracts
- Anterior or ventral corticospinal tract is formed by uncrossed fibers are primarily concerned with the control of fine movements that require dexterity.



MUSCLE TONE ➔🡻🡹

The resistance of a muscle to stretch is often referred to as its **tone or tonus**.

If the motor nerve to a muscle is severed, the muscle offers very little resistance and is said to be **flaccid.**

A **hypertonic (spastic)** muscle is one in which the resistance to stretch is high because of hyperactive stretch reflexes

COMAPRISON BETWEEN UPPER & LOWER MOTOR NEURON LESIONS

UMN LESION

- Paralysis affect movements
- Wasting not pronounced.
- Spasticity Muscles hypertonic (Clasp Knife).
- Tendon reflexes increased.
- Superficial reflexes diminished
- Babinski's sign +ve,

LMN LESION

- Individual muscle or group of muscles are affected.
- Wasting pronounced.
- Flaccidity. Muscles hypotonic.
- Tendon reflexes diminished or absent.

What is Spasticity?

"Spasticity is a motor disorder characterized by a velocity-dependant increase in tonic stretch reflexes with exaggerated tendon jerk, resulting from hyperexcitability of the stretch reflex" **James Lance (1980)**

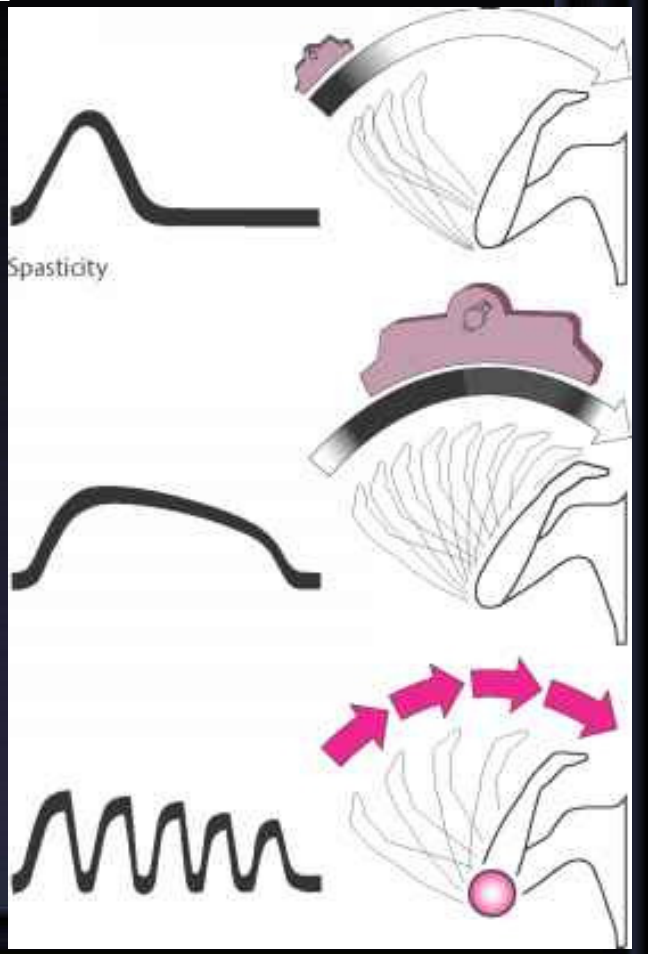
In UMN lesions Spasticity is of Clasp Knife Type

Rigidity is increased neural activity throughout the range of muscle movement and is not velocity dependent. Rigidity is present in both agonist and antagonist muscles. It is often associated with basal ganglia disease such as Parkinson's disease

Rigidity in Parkinsonism

Lead-pipe rigidity . Passive movement of an extremity meets with a constant dead feeling resistance like a lead pipe throughout the range of movement.

Sometimes a series of "catches" takes place during passive motion like a cogwheel



Cog-wheel rigidity

In cogwheel rigidity one feels that resistance varies rhythmically when applying a passive movement. It is because of an underlying resting tremor associated with rigidity.

CAUSES OF SPASTICITY

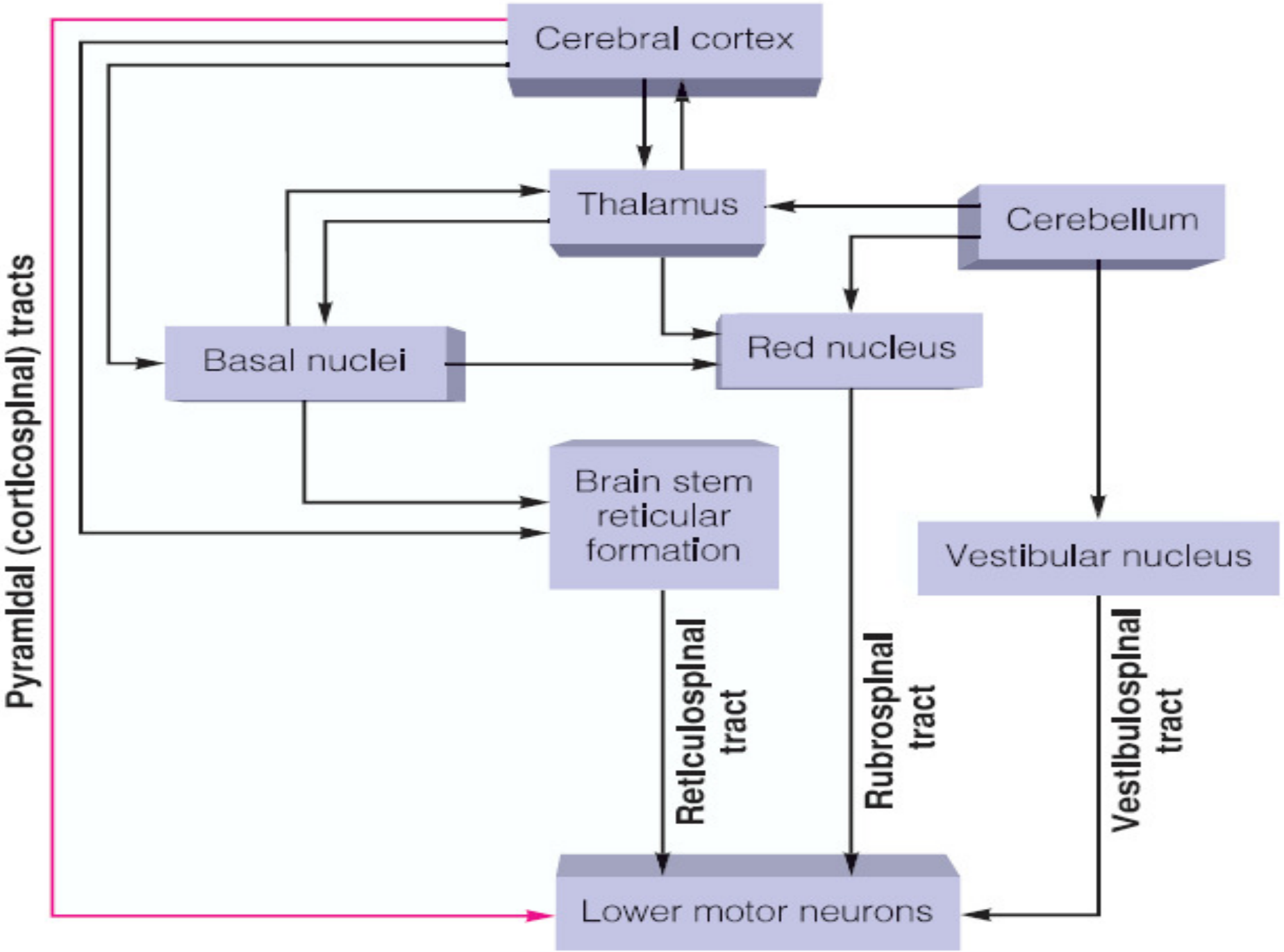
A-(UMNS) syndrome include :

- **(1) Cerebral palsy**
- **(2) Stroke**
- **(3) Spinal cord injury**
- **(4) Multiple Sclerosis**
- **(5) Acqiured brain injury (trauma , etc)**

B-Parkinsonism

C- Decerebrate & decorticate rigidity

Patients complain of pain, stiffness & inability to relax
Prolonged stiffness leads to bone & joint deformities with disability and contractures & its consequences


Cerebral cortex
Thalamus
Cerebellum
Basal nuclei
Red nucleus
Brain stem reticular formation
Vestibular nucleus
Lower motor neurons
Pyramidal (corticospinal) tracts
Reticulospinal tract
Rubrospinal tract
Vestibulospinal tract

Muscle Spasticity Caused by Lesions That Damage Large Areas Adjacent to the Motor Cortex.

The primary motor cortex normally exerts a continual tonic stimulatory effect on the motor neurons of the spinal cord; when this stimulatory effect is removed, *hypotonia* results.

-Most lesions of the motor cortex, especially those caused by a *stroke,* involve not only the primary motor cortex but also adjacent parts of the brain such as the basal ganglia. In these instances, *muscle spasticity* almost invariably occurs in the afflicted muscle areas on the o*pposite side* of the body.

This spasm results mainly from damage to accessory nonpyramidal pathways. These pathways normally inhibit the vestibular and reticular brain stem motor nuclei. When these nuclei cease their state of inhibition (i.e., are "disinhibited"), they become spontaneously active and cause excessive spastic tone in the involved muscles.

Two types of reticulospinal tracts

(1)Pontine (Medial) Reticulospinal Tract(anterior column of spinal cord)

- Transmit excitatory signals downward into the cord→medial anterior motor neurons that excite the axial muscles of the body, which support the body against gravity. In addition, they receive strong excitatory signals from the vestibular nuclei & from deep nuclei of the cerebellum.
- Axons descend in anterior(ventral)white column of spinal cord
- **Functions:** Excite the antigravity muscles of the body and increases Gamma efferent activity

(2)Medullary (Lateral) Reticulospinal Tract:

Axons descend in lateral white column of spinal cord on both sides

It receive strong input from (1) the corticospinal tract, (2) the rubrospinal tract, and (3) other motor pathways→ activate the medullary reticular inhibitory system to counterbalance the excitatory signals from the pontine reticular system

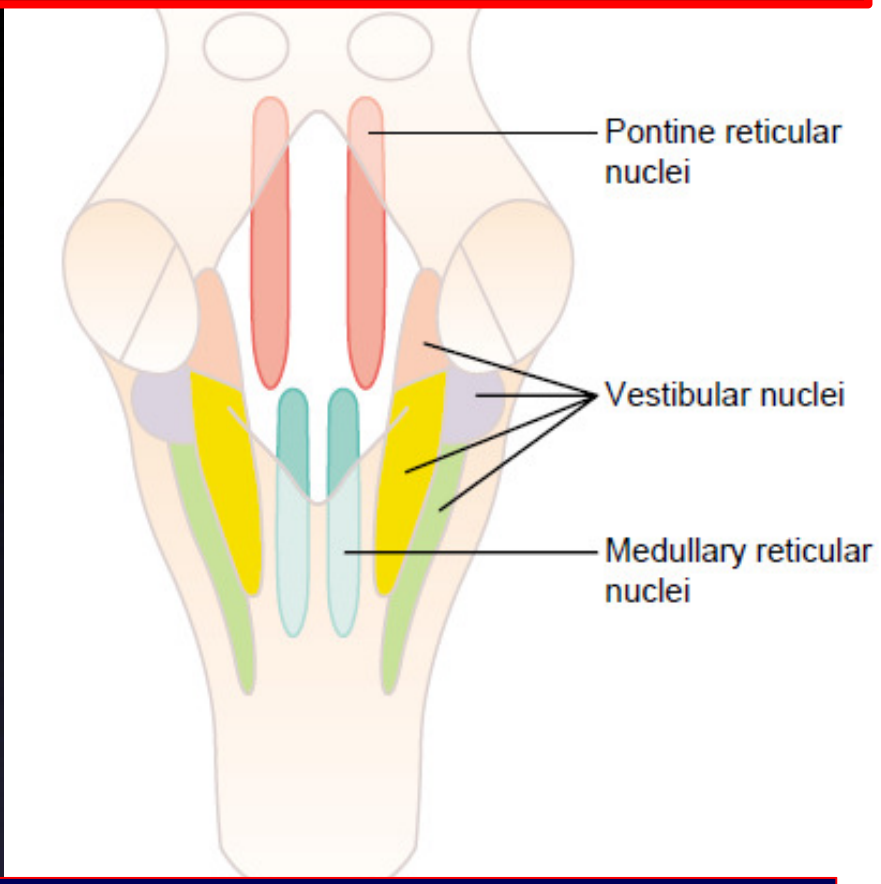
- **Functions:** counterbalance the excitatory signals from the pontine reticular system and inhibits Gamma efferent activity

The excitatory and inhibitory reticular nuclei constitute a controllable system that is manipulated by motor signals from the cerebral cortex to provide necessary background muscle contractions for standing against gravity

Support of Body Against Gravity is the role of the Reticular and Vestibular Nuclei

Pontine Reticular System. Transmit excitatory signals downward into the cord through the pontine reticulospinal tract in the anterior column of the cord

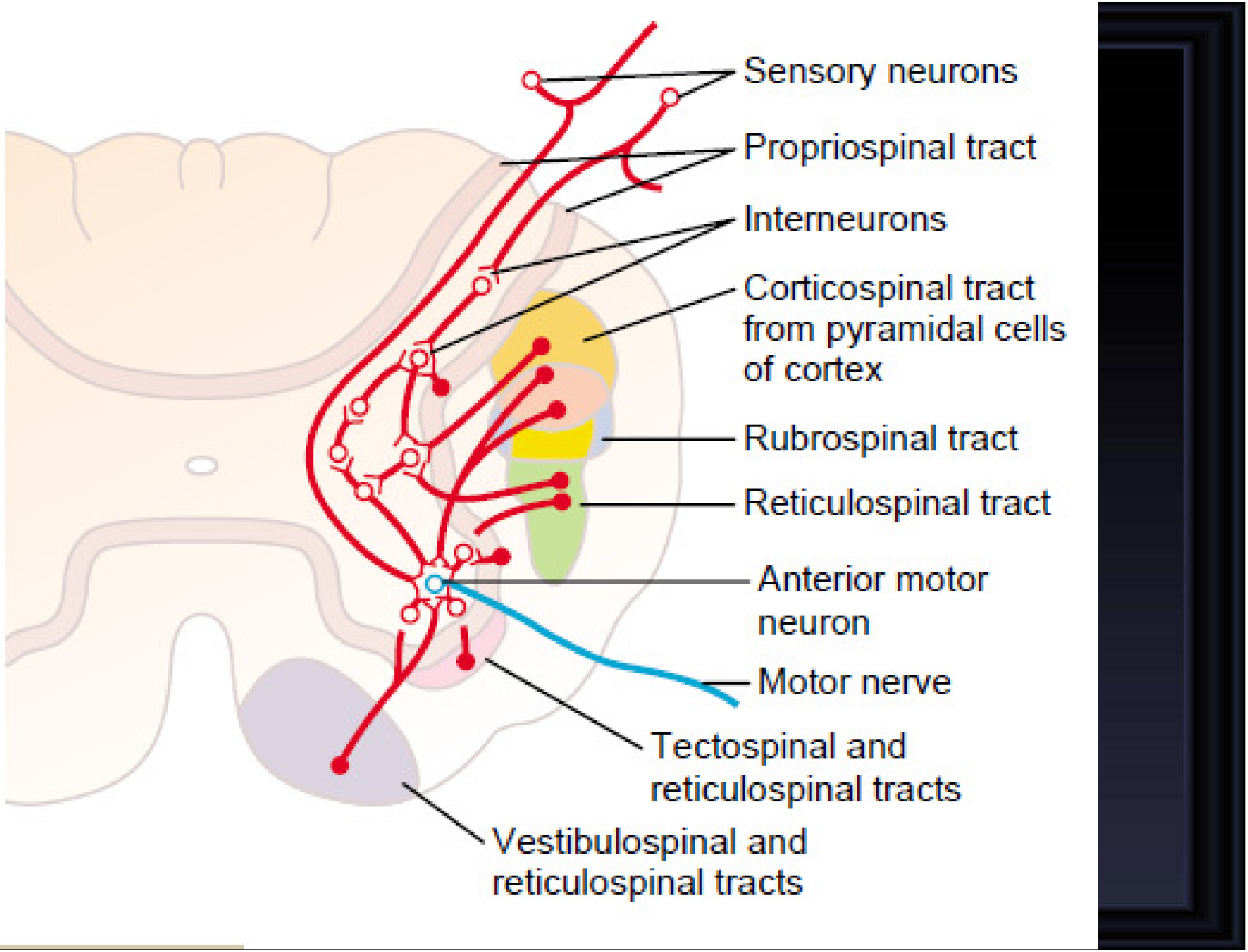
Medullary Reticular System. Transmit inhibitory signals to the same antigravity anterior motor neurons by way of a different tract, the medullary reticulospinal tract, located in the lateral column of cord



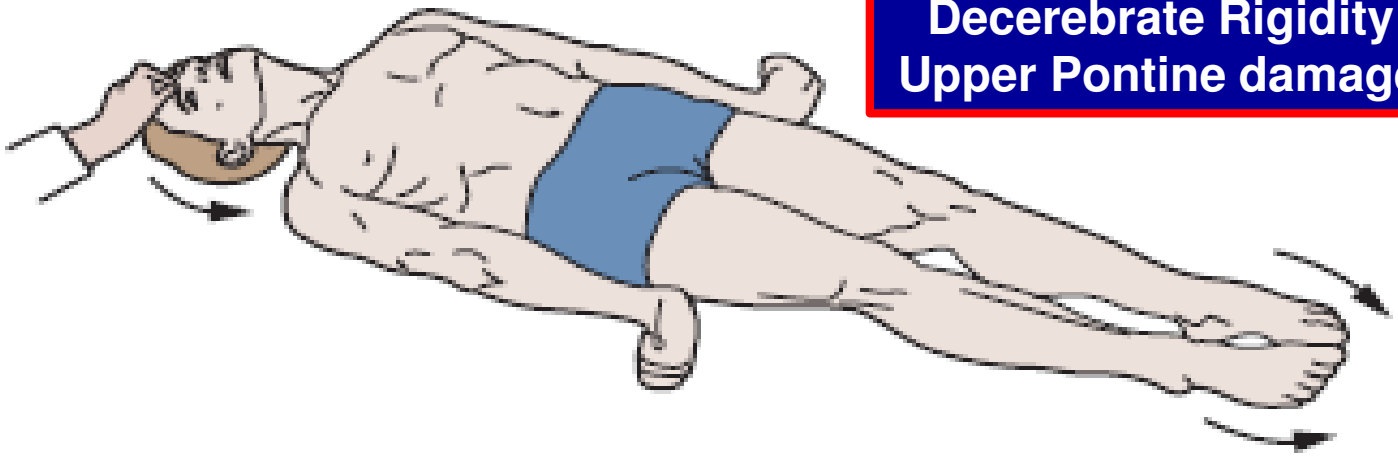
Decerebrate rigidity results from blockage of normally strong input to the medullary reticular nuclei from the cerebral cortex, the red nuclei, and the basal ganglia.

Medullary reticular inhibitor system becomes nonfunctional

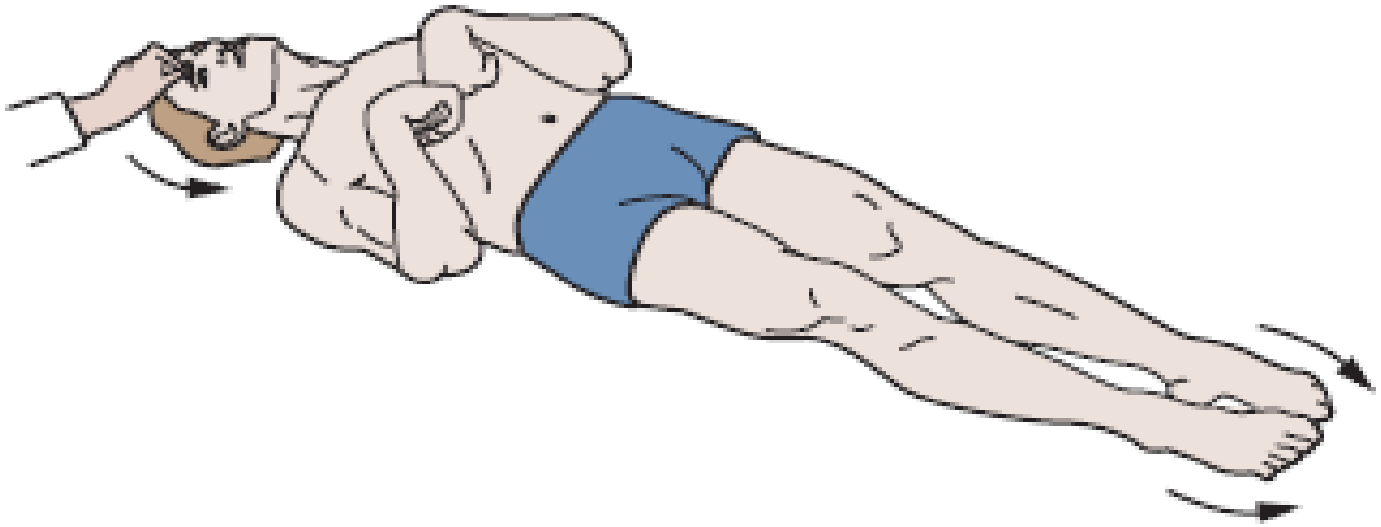
Pontine excitatory system becomes overactive


Sensory neurons
Propriospinal tract
Interneurons
Corticospinal tract from pyramidal cells of cortex
Rubrospinal tract
Reticulospinal tract
Anterior motor neuron
Motor nerve
Tectospinal and reticulospinal tracts
Vestibulospinal and reticulospinal tracts

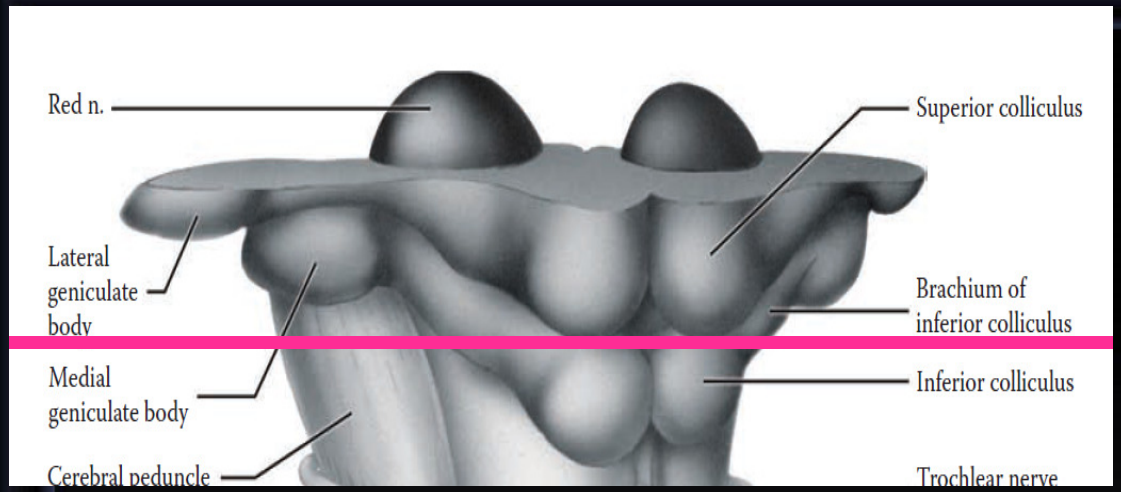
Decerebrate Rigidity
Upper Pontine damage

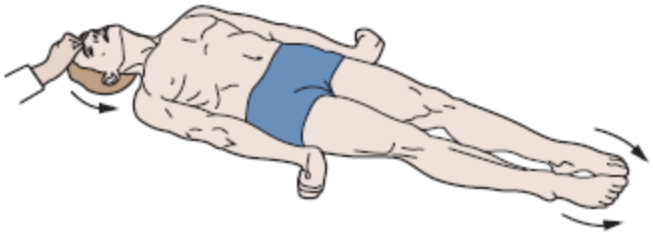


Decorticate Rigidity
Upper midbrain damage



Decerebrate posturing

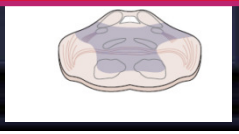


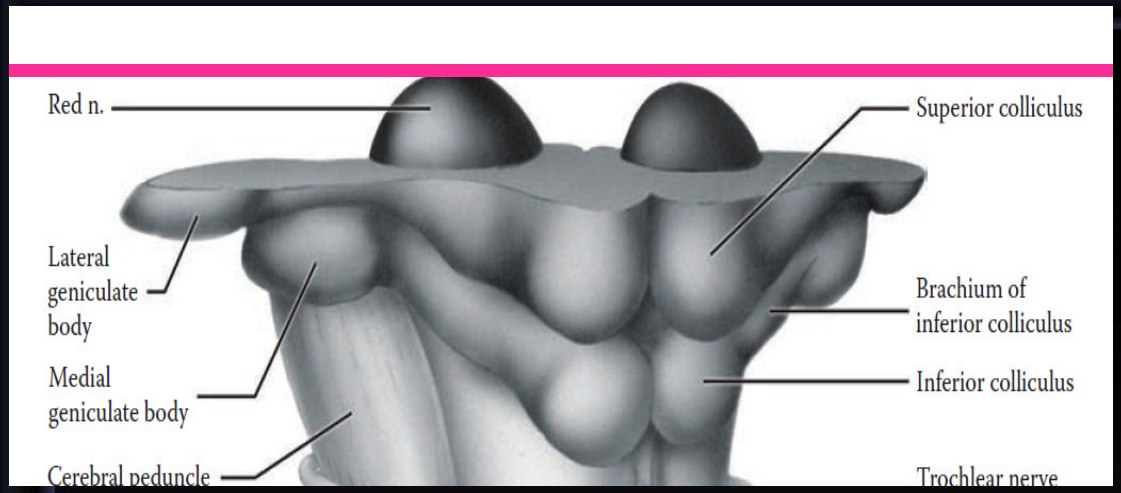


- midcollicular decerebration ➔decerebrate rigidity
- Clinical Example: Uncal herniation due to a supratentorial lesion (tumors, hemorrhages, strokes, or abscesses in the cerebral hemisphere)
  - Signs:
  - compressing the ipsilateral cranial nerve III
  - hyperactive reflexes
  - bilateral Babinski sign

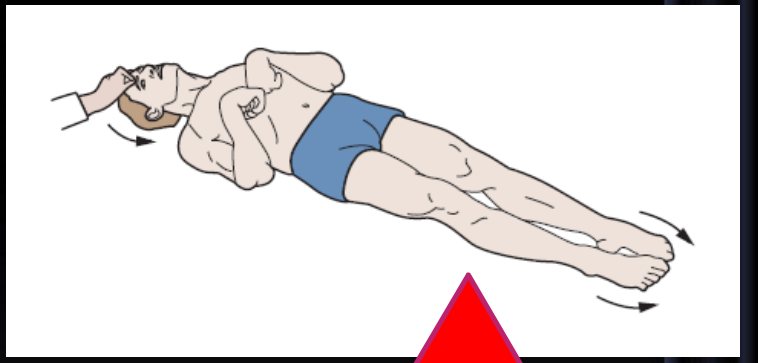
After brain herniates: the patients are decerebrate and comatose, have fixed and dilated pupils, and eye movements are Absent. Once damage extends to the midbrain, a Cheyne–Stokes respiratory pattern starts. Decerebrate posture occurs

Lower extremities are extended with toes pointed inward and upper extremities extended with fingers flexed and forearms pronate. Neck and head are extended.





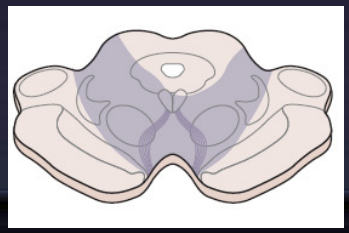
Decorticate posturing



Upper limbs are Flexed, lower limbs are extended with toes pointed slightly inward, and head is extended.

Removal of the cerebral cortex (decortication)

- **Flexion can be explained by rubrospinal excitation of flexor muscles in the upper extremities; the**
- **hyperextension of lower extremities is due to the same changes that occur after midcollicular decerebration.**
- **because of their anatomy, the small arteries in the internal capsule (60% lesions) are especially prone to rupture or thrombotic obstruction, so this type of decorticate rigidity is fairly common**

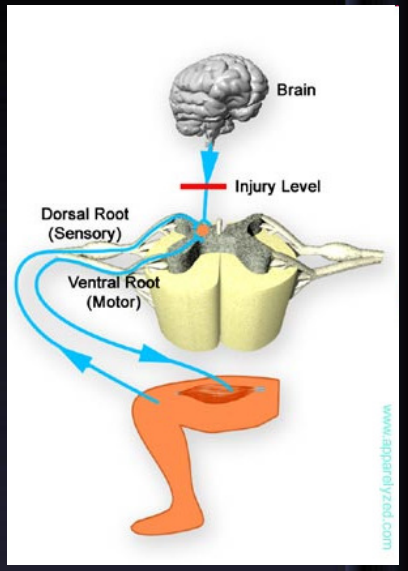


Mechanism of Spasticity

- The absence of an upper motor neuron's inhibitory control on the spinal reflex. Loss of descending inhibition from the brain to BRAIN STEM EXCITATORY CENTERS (pontine RF + vestibular N).
- Vestibulospinal & reticulospinal EXCITATORY signals to muscles to become spastic
- Hyperactive stretch reflexes that are mediated by muscle spindle stretch receptors
- Decreased threshold of the alpha lower motor neurons

Mechanism of Spasticity

- When Central Nervous System Lesion occurs at that time there is
  - Hypotoncity
  - Alpha Lower motor neuron firing ↓



- Days to weeks later
  - Denervation hypersensitivity
    - Alpha lower motor neurons lower threshold
    - An increase in the number of Ach receptors

Pure corticospinal tract lesion cause hypotonia instead of spasticity The reason is that pure pyramidal tract lesion is very very rare, and spasticity is due to loss of inhibitory control of extrapyramidal tract.

Increased Gamma efferent discharge is the main cause of increased muscle tone.

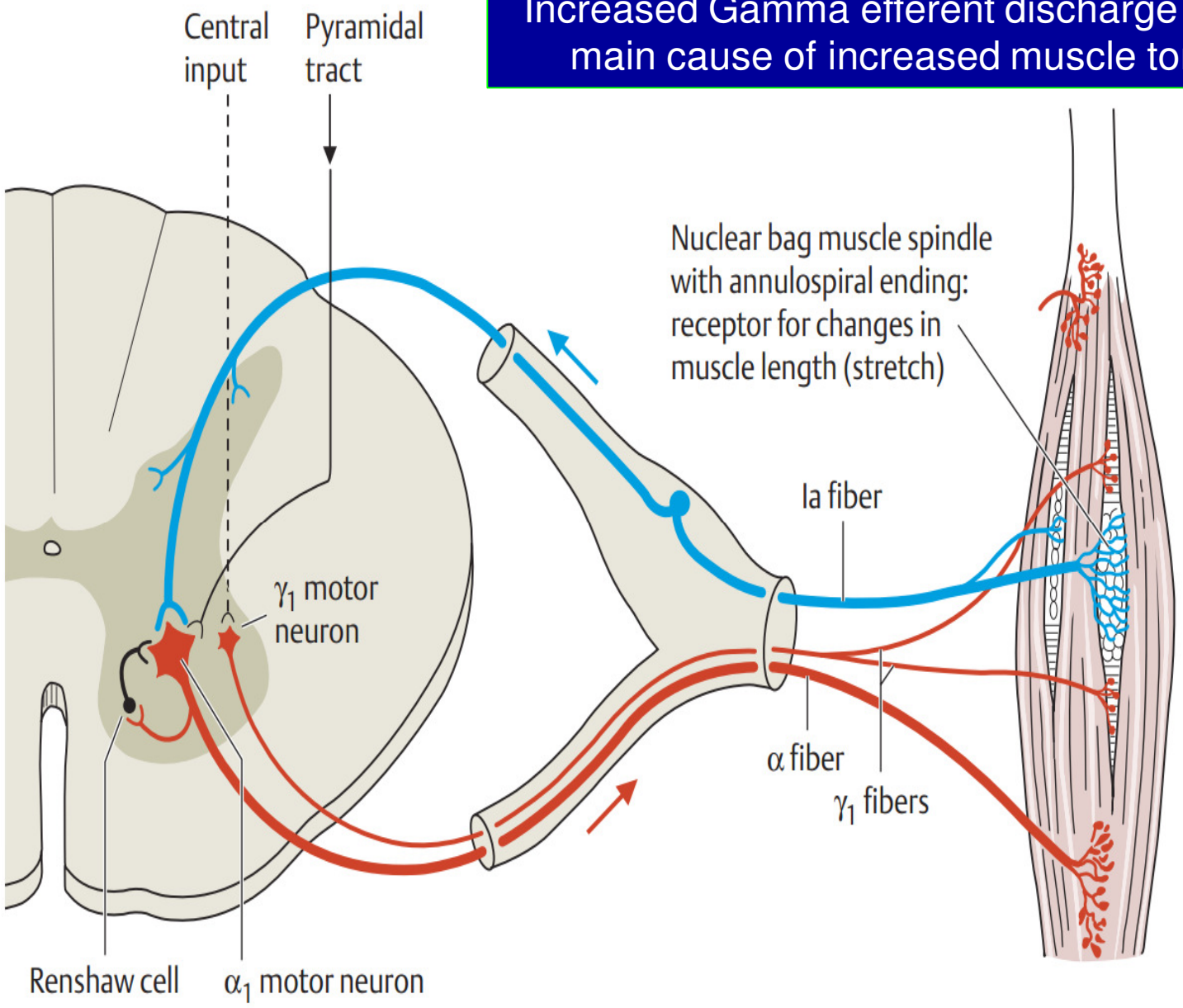


Fig. 2.11 **Regulatory circuit for muscle length**

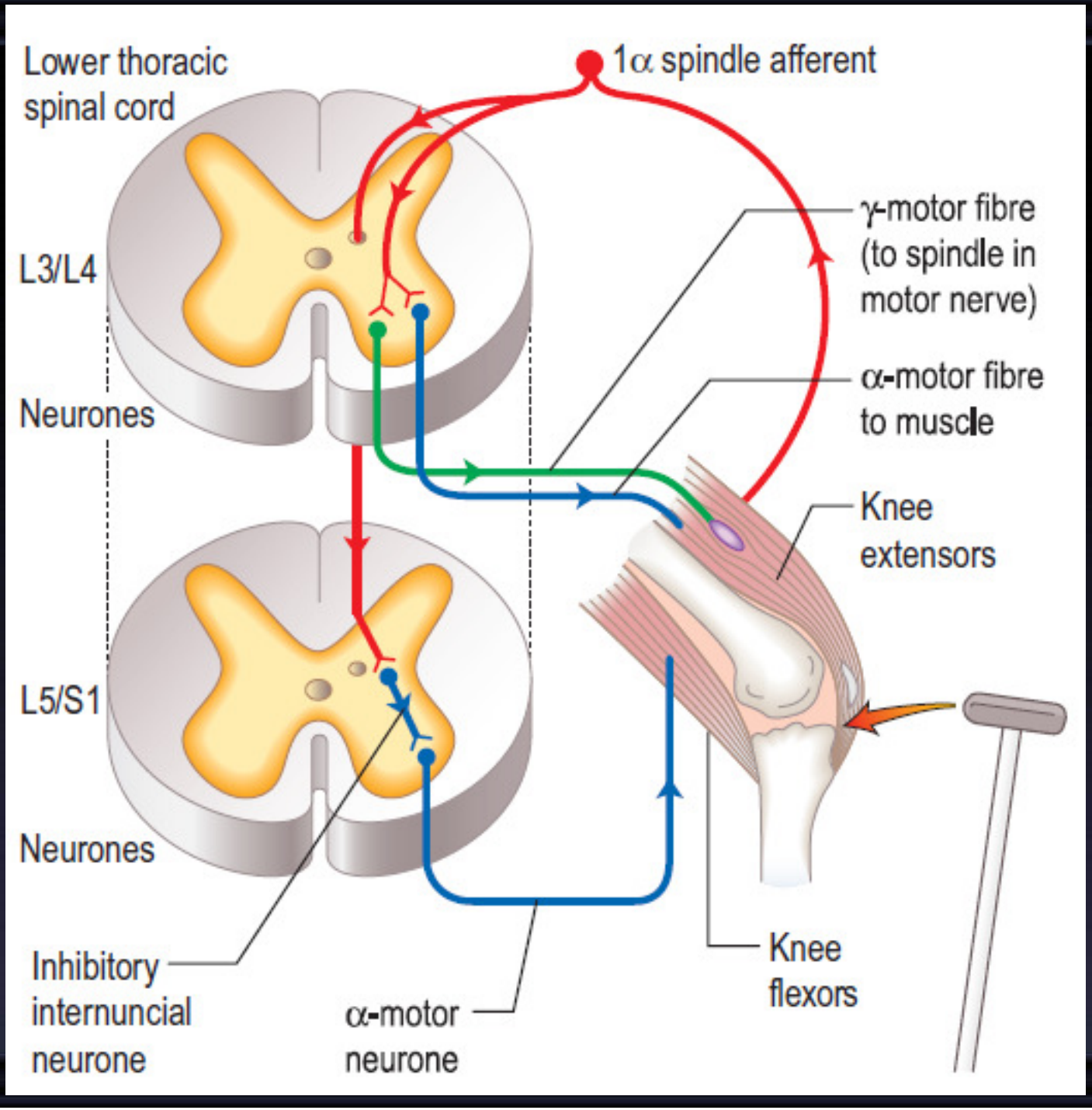

Lower thoracic spinal cord
1α spindle afferent
γ-motor fibre (to spindle in motor nerve)
α-motor fibre to muscle
L3/L4
Neurones
Knee extensors
L5/S1
Neurones
Inhibitory internuncial neurone
α-motor neurone
Knee flexors

Table 21.17 Spinal levels, recording/interpretation of tendon reflexes

Level	Reflex	Symbol for reflex	Meaning
C5–6	Supinator	0	Absent
C5–6	Biceps	+/–	Present with reinforcement
C7	Triceps	+	Normal
L3–4	Knee	++	Brisk, normal
S1	Ankle	+++	Exaggerated (abnormal)
		CL	Clonus

Ashworth Scale

- **Physical therapist need clarity to realize how spasticity can have affects on a patients ability to move**
- **A valid & reliable measure scale for spasticity**

	Modified Ashworth Scale
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end range of motion when the part is moved in flexion or extension/abduction or adduction, etc.
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM
2	More marked increase in muscle tone through most of the ROM, but the affected part is easily moved
3	Considerable increase in muscle tone, passive movement is difficult
4	Affected part is rigid in flexion or extension (abduction ,adduction, etc.)

Spasticity vs Dystonia

- Increased muscle tone
- Resistance to passive muscle stretch at a joint
- Velocity and joint-angle dependent
- Has to be palpated – ie you need to lay on hands
- Other features: clonus, increased reflexes, upgoing plantars

- Sustained and repititive muscle contractions & abnormal postures
- Visible Involuntary movements
- Suggests active rather than passive movements
- Not usually velocity-dependent
- Dependent on mental state: alertness, emotion

Types of dystonia

Basal Ganglia and Cerebellum damage

Excessive movements:

chorea, athetosis, dystonic tremor, myoclonus, "spasms"

Decreased movements:

extrapyramidal rigidity, hypokinesia

Oropharyngeal: mouth, tongue, pharynx, larynx

Axial muscles: cervical, erector spinae (opisthotonus)

Upper Limbs: Elbow extensor rigidity, fisting

Lower limbs: Ankle evertors as well as invertors

Interventions/Treatments

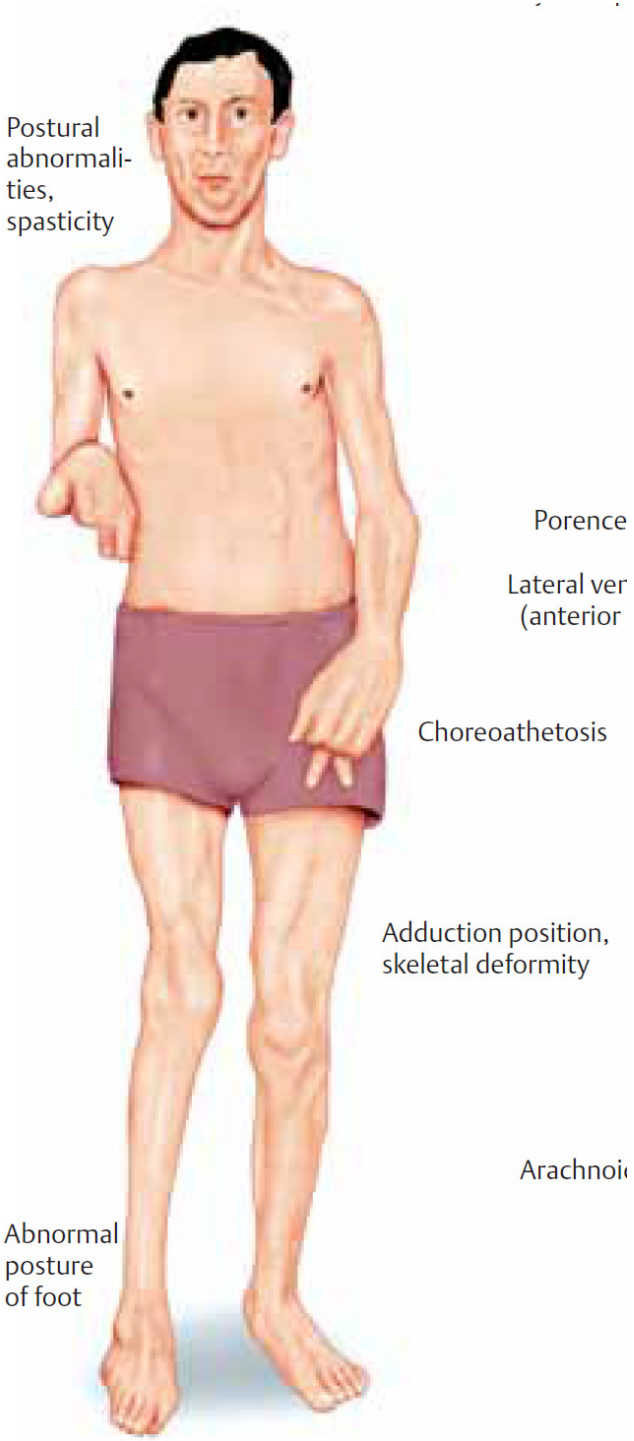
Effective Treatments for reducing Spasticity

- **PT Intervention**
- **Electrical and Functional Electrical Stimulation**
- **Casting**
- **Mental Imagery**
- **Pharmacological Interventions**
- **Surgery**

CEREBRAL PALSY (CP)

CP includes disorders apparent at birth or in childhood due to intra-uterine or neonatal brain damage mainly motor control centers; deficits are non-progressive

- hypoxia in utero and/or during parturition
- neonatal cerebral haemorrhage and/or infarction
- trauma, neonatal or during parturition
- prolonged seizures – status epilepticus
- Hypoglycaemia
- kernicterus



MULTIPLE SCLEROSIS

- Auto-immune demyelinating disease , in which the body's own immune system attacks the myelin sheath of nerves mainly of brain, spinal cord & optic nerve
- Demyelination affects saltatory conduction and cause muscle weakness& wasting.
- Onset usually in young adults (F>M)
- When it causes demyelination of descendindg motor tracts in the brainstem & spinal cord , the patient develops spasticity and other signs of UMN lesions .
- The disease frequently remits and relapses
- Treated by steroids

STROKE

- Haemorrhagic stroke & Ischaemic stroke
- Results in paralysis in the opposite half of the body
- A lesion in Corona Radiata on one side can cause Monoplegia in a contralateral limb (UL or LL).
- A lesion in the Internal Capsule on one side may cause Hemiplegia or Hemiparesis on the contralateral side
- Presents with features of upper motor neuron syndrome UMNL .

Complete transection of spinal cord

- in **lower cervical region → quadriplegia**
- **down in the thoracic region → paraplegia (both lower limbs)**

3 Stages :-

A/ Spinal shock (2-6 weeks)
B/ Recovery of reflex activity
C/ Paraplegia in extension

Voluntary movements and sensations are permanently lost

A/ Spinal shock: Immediately following transection there is loss of all sensations (anaesthesia), reflexes, tone (flacciduty) and voluntary movement below the level of the lesion , due to interruption of all
sensory and motor tracts.
Bladder urinary retention with overflow) and Loss of vasomotor tone occurs causes a fall in blood pressure;

B/ Stage of return of reflex activity due to increase in degree of excitability of the spinal cord neurons

- Gradual rise of arterial blood pressure
- Exaggerated tendon reflexes, spasticity, viceral reflexes return (micturition, defecation & erection reflexes)
- Mass reflex A minor painful stimulus to the skin of the lower limbs causes withdrawal and evoke autonomic reflexes (bladder and rectum emptying, sweating , blood pressure rise)

C/ Stage of extensor paraplegia

The lower limbs become spastically extended.

-Extensor reflexes become exaggerated, as shown by tendon jerks and by the appearance of clonus.

-The positive supportive reaction returns and the patient can stand on his feet with appropriate support.

The flexor withdrawal reflex and crossed extensor reflex returns