محاضرات د. مزهر الضالعي
2016 لطلبة السنة الثالثة طب بشري
Neuroscience

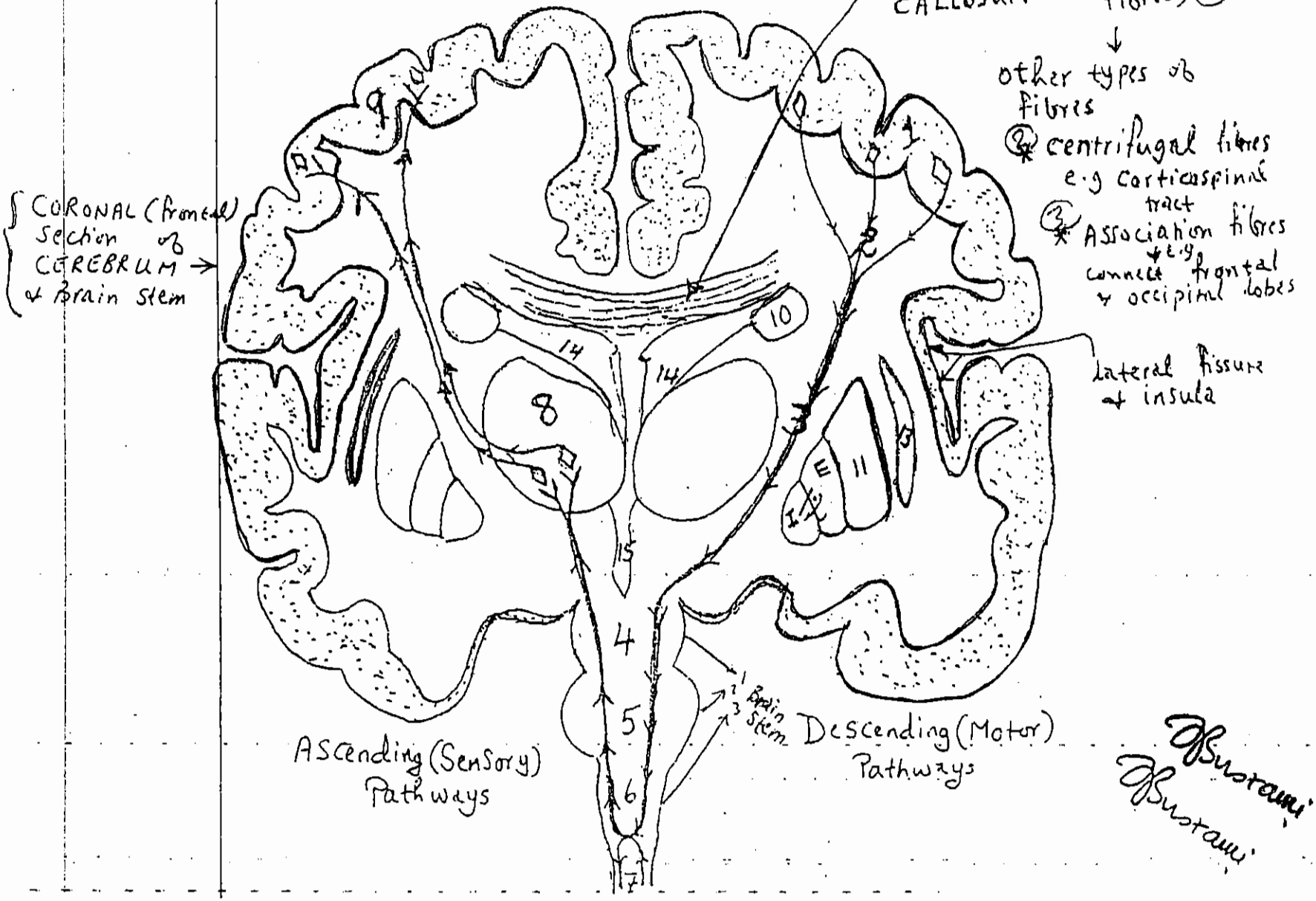

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Diagram 2

Explanation for the diagram

No. (1) → Cerebral cortex → formed of gray matter i.e Neurons → The axons of these neurons may descend forming projection fibres (Centrifugal fibres) or cross from one cerebral hemisphere to the other forming commissural fibres (e.g corpus callosum). Note how the cortex is thrown into gyri separated by sulci or fissures → this is in order to increase the surface area.

No. (2) the descending axons form part of the corona radiata and continue down to form (3) → the corticospinal tract (a major motor pathway) which continues in the brainstem and most of its fibres decussate (cross) to the opposite side at the lower part of the medulla.

N.B. a - Notice that the descending fibres in the corticospinal tract CROSS at the lower medulla oblongata so that the right cerebral cortex will control muscles of the left half of the body and the left cerebral cortex will control muscles of the right side of the body.

b - The corticospinal tract is one component of the pyramidal tract, the other component is the corticobulbar tract which also begins at the motor cortex but ends at the motor nuclei of certain cranial nerves.

(2) Axons of motor neurons of the cerebral cortex forming the corona radiata.

(3) This is the internal capsule which represents the gate to the cerebral cortex i.e. all the fibres that come from (i.e. motor) or go to (i.e. sensory) the cerebral cortex will run here in a compact bundle. As a result lesion in this part of the brain (e.g. obstruction of the blood vessels which supply this region by embolism or thrombosis will lead to widespread disturbances e.g. hemiplegia (paralysis of the contralateral half of the body and hemianaesthesia (loss of sensations in the contralateral half of the body)

(4) Midbrain : ~~This is a part of the brainstem which~~ contains ascending (sensory) and descending (motor) tracts. In addition it contains the nuclei of the third and fourth cranial nerves. ⊕ Red nucleus ⊕ Substantia nigra

(5) Pons : This is another part of the brain stem which contains ascending and descending tracts. In addition it contains the nuclei of the 5th, 6th, 7th and 8th cranial nerves.

6. Medulla oblongata: this is the lower part of the brain stem. In addition the ascending and descending tracts it contains the nuclei of the 9th, 10th, 11th and 12th cranial nerves. The medulla oblongata has the following centres which control vital visceral activities:

a. cardiac centre: Impulses from this centre run along the vagus nerves and can cause the heart to beat more slowly or more rapidly

b. Vasomotor centre (VMC) : impulses from this centre travel to smooth muscles in the walls of arterioles and causes either vasoconstriction (and a rise in blood pressure) or vasodilation (or drop in blood pressure)

c. Respiratory centre: it functions, together with the respiratory centre in the pons to regulate the rate and depth of breathing.

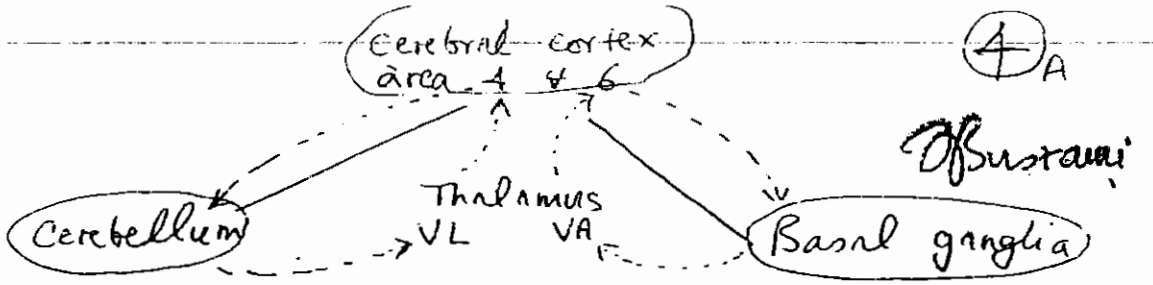
d. Other centres : for reflexes associated with coughing, sneezing, swallowing and vomiting.

N.B. Scattered throughout the medulla oblongata, pons and midbrain a complex network of nerve cells and nerve fibres known as the RETICULAR FORMATION (RF). It is connected to most of the ascending (sensory) pathways as well as to the cerebrum, basal ganglia and cerebellum. When sensory impulses reach the reticular formation, it responds by signaling the cerebral cortex activating it into a state of wakefullness (hence the name reticular activating system RAS)

Without this arousal, the cortex remains unaware of stimulation and cannot interpret sensory information or carry on thought processes. Thus if the reticular formation ceases to function as in certain injuries, the person remains unconscious. In addition many drugs e.g. anaesthetics and tranquilizers are believed to have some effect on the reticular formation.

7. beginning of spinal cord

8. Thalamus : This is the upper part of the diencephalon. The lower part of this regionn is called the hypothalamus. It lies below the thalamus and contains many nuclei. The thalamus serves as a central relay station for sensory impulses traveling upward from other parts of the nervous system to the cerebral cortex. It receives all sensory impulses (except smell) and send them to appropriate regions of the cortex for interpretation. In other words it acts as a SECRETARY to the sensory cortex. In addition it has a motor part connected to the motor cortex, basal ganglia and cerebellum and concerned with the regulation of voluntary motor activity



4 A

Mustafa

VA = Ventral anterior nucleus of Thalamus
VL = Ventral lateral >> >> >>

Remember that the cerebral cortex (motor areas) initiates voluntary movement but inorder that this movement is smooth and accurate the cerebral cortex need the presence of functioning cerebellum and basal ganglia

The Hypothalamus : plays key roles in maintaining HOMEOSTASIS by regulating a variety of visceral activities and by serving as a link between the nervous and endocrine systems.

Among the many important functions of the hypothalamus are the following:-

1. Regulation of heart rate and arterial blood pressure
2. Regulation of body temperature
3. Regulation of water and electrolyte balance
4. control of hunger and regulation of body weight
5. control of movements and glandular secretions of the stomach and intestines
6. Production of neurosecretory subetances that stimulate the pituitary gland to release various hormones
7. Regulation of sleep and wakefulness.

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(9) Sensory part of cerebral cortex which interpret impulses that arrive from various sensory receptors.

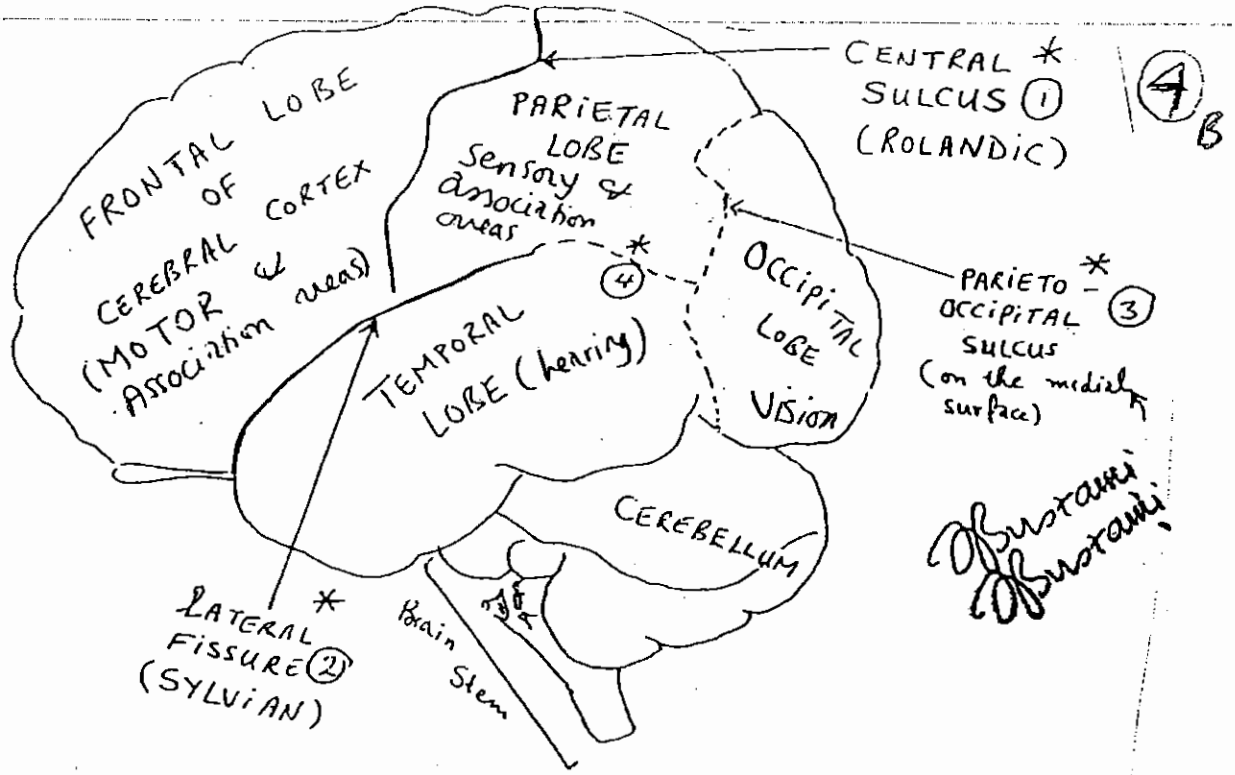
(10) Caudate nucleus

(11) Putamen
(12) globus pallidus → Lentiform nucleus (lenticular nucleus)
(12) globus pallidus → external segment
(12) globus pallidus → internal >>
(13) Claustrum

N.B. 10+ 11 + 12 + 13 = basal ganglia or basal nuclei → Contribute to motor activity (Planning & Programming of movement)

(14) Lateral ventricle i.e. cavity of cerebrum
(15) third ventricle

(Both ventricles contain cerebrospinal fluid - CSF).



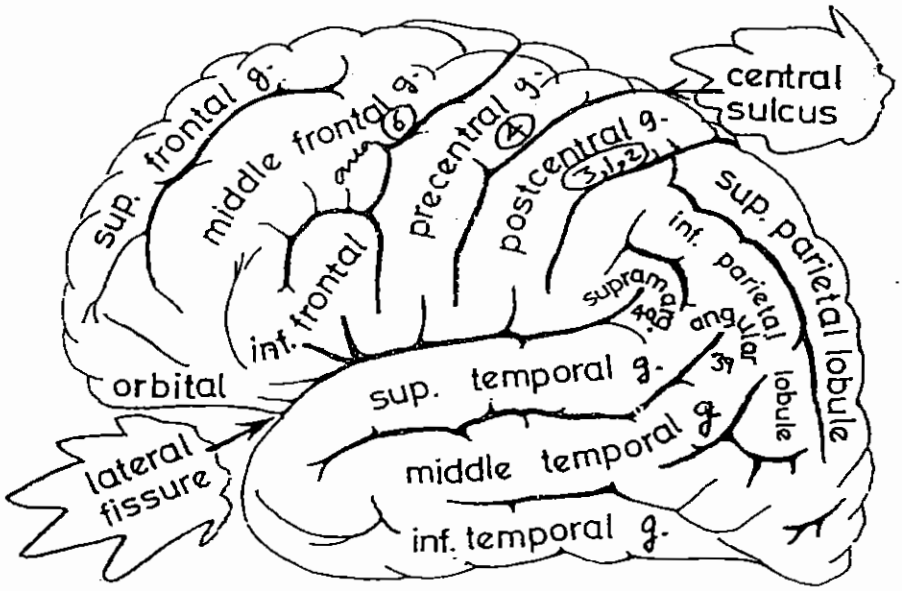


Fig. 48 The gyri of the lateral surface of the cerebral hemisphere.

Precentral gyrus (Frontal lobe) = area 4 = Primary motor cortex

Premotor cortex (~~frontal lobe~~) = area 6

Supplementary motor area (SMA) = medial extension of area 6

Postcentral gyrus (parietal lobe) = area 3, 1, 2 = Somatic sensory cortex or Somasthetic cortex

ORIENTATION

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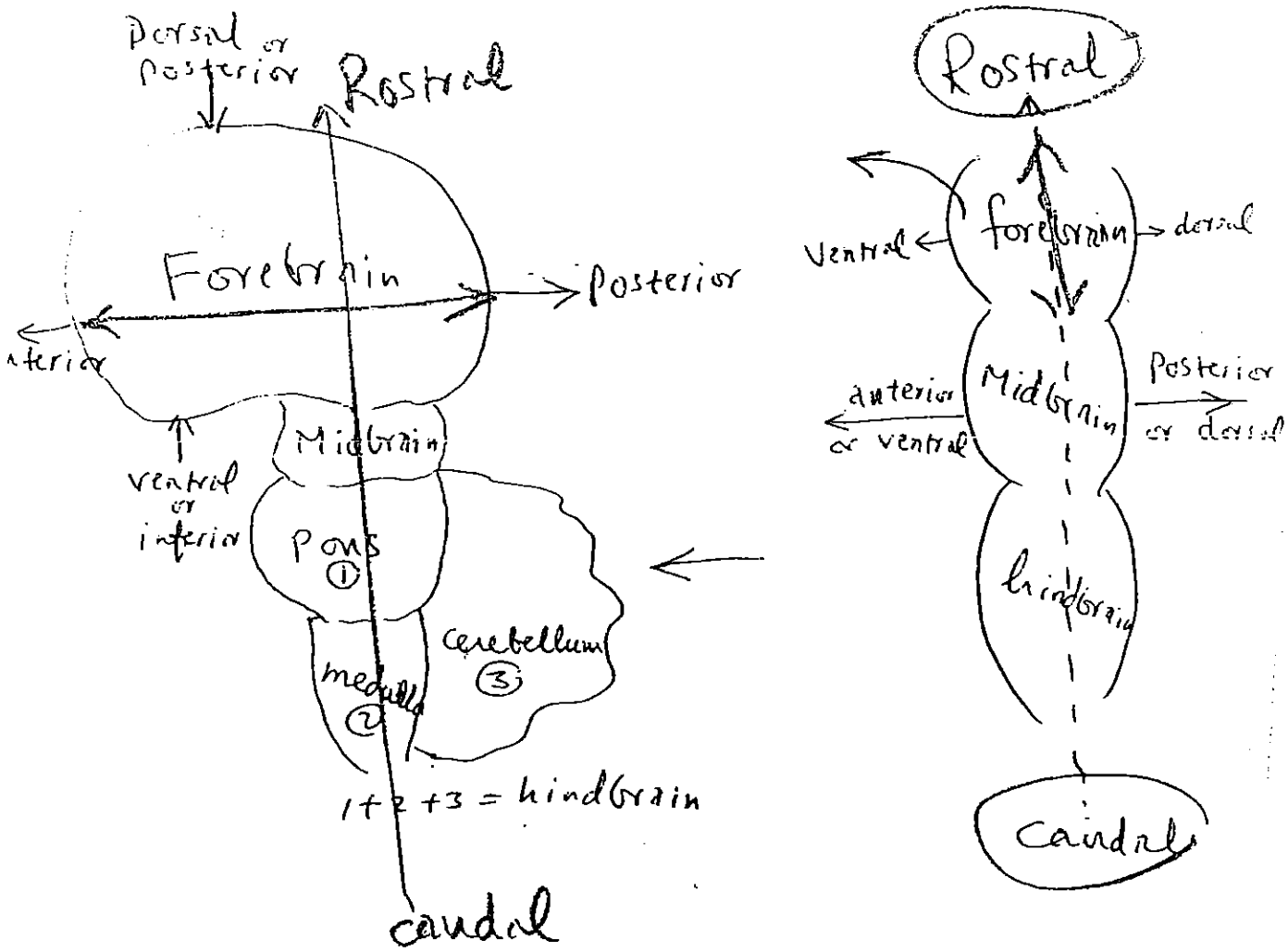


The midbrain, hindbrain, spinal cord → oriented almost vertically

whereas forebrain → oriented horizontally

{ A change in orientation at midbrain - forebrain junction (i.e)

↓

the terms dorsal & ventral have different use }

Table 1. Motor and Sensory Classification of Nerve Fibers

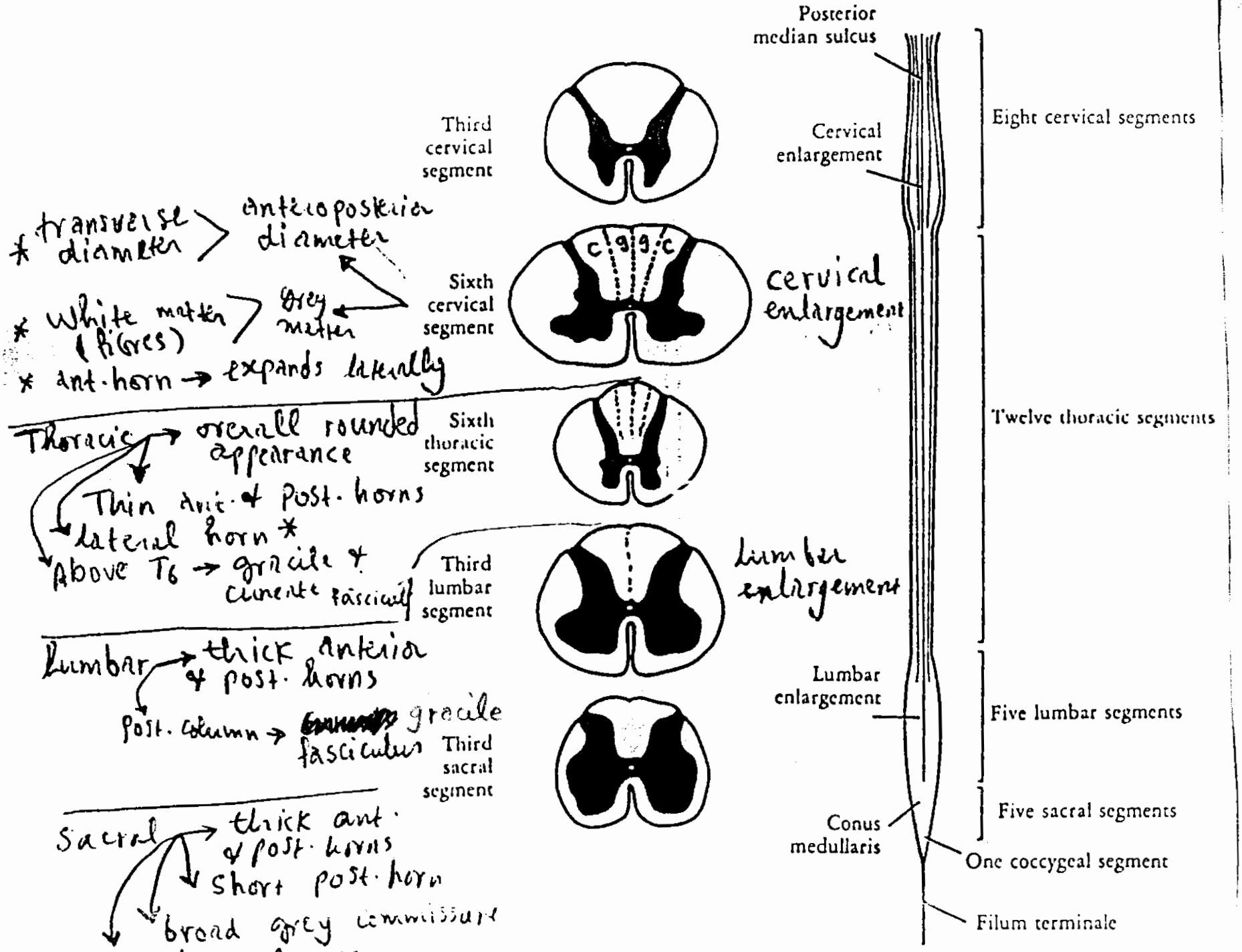
Sensory (Groups)		Sensory and Motor	Greatest Fiber Diameter (μ)	Greatest Conduction Velocity (meters/sec)	General Comments
Ia	=	Aα	22	120	Motor—the large alpha motor neurons of lamina IX Sensory—the primary afferents (annulospiral) of muscle spindles
Ib	=	Aα	22	120	Sensory—Golgi tendon organs, touch, and pressure receptors
II	=	Aβ	13	70	Sensory—the secondary afferents (flower spray) of muscle spindles, touch, and pressure receptors, Pacinian corpuscles (vibratory sensors)
		Aγ	8	40	Motor—the small gamma motor neurons of lamina IX innervate muscle spindles
III	=	Aδ	5	15	Sensory—small lightly myelinated fibers, touch, pressure, pain, and temperature
		B	3	14	Motor—small lightly myelinated preganglionic autonomic fibers
IV	=	C	1	2	Motor—all postganglionic autonomic fibers (all are unmyelinated) Sensory—unmyelinated pain and temperature fibers

Notice → Afferents from muscle spindle are Ia & II

Afferents from Golgi tendon organ is → Ib

Pain fibres run in two types of afferents → Aδ & C

Remember that the greater the diameter of the nerve fibre → the thicker the myelin sheath and the faster the conduction velocity



* transverse diameter > anteroposterior diameter
* White matter (fibres) > Grey matter
* ant. horn → expands laterally

Thoracic → overall rounded appearance
Thin ant. & post. horns
lateral horn *
Above T6 → gracile & cuneate fasciculi

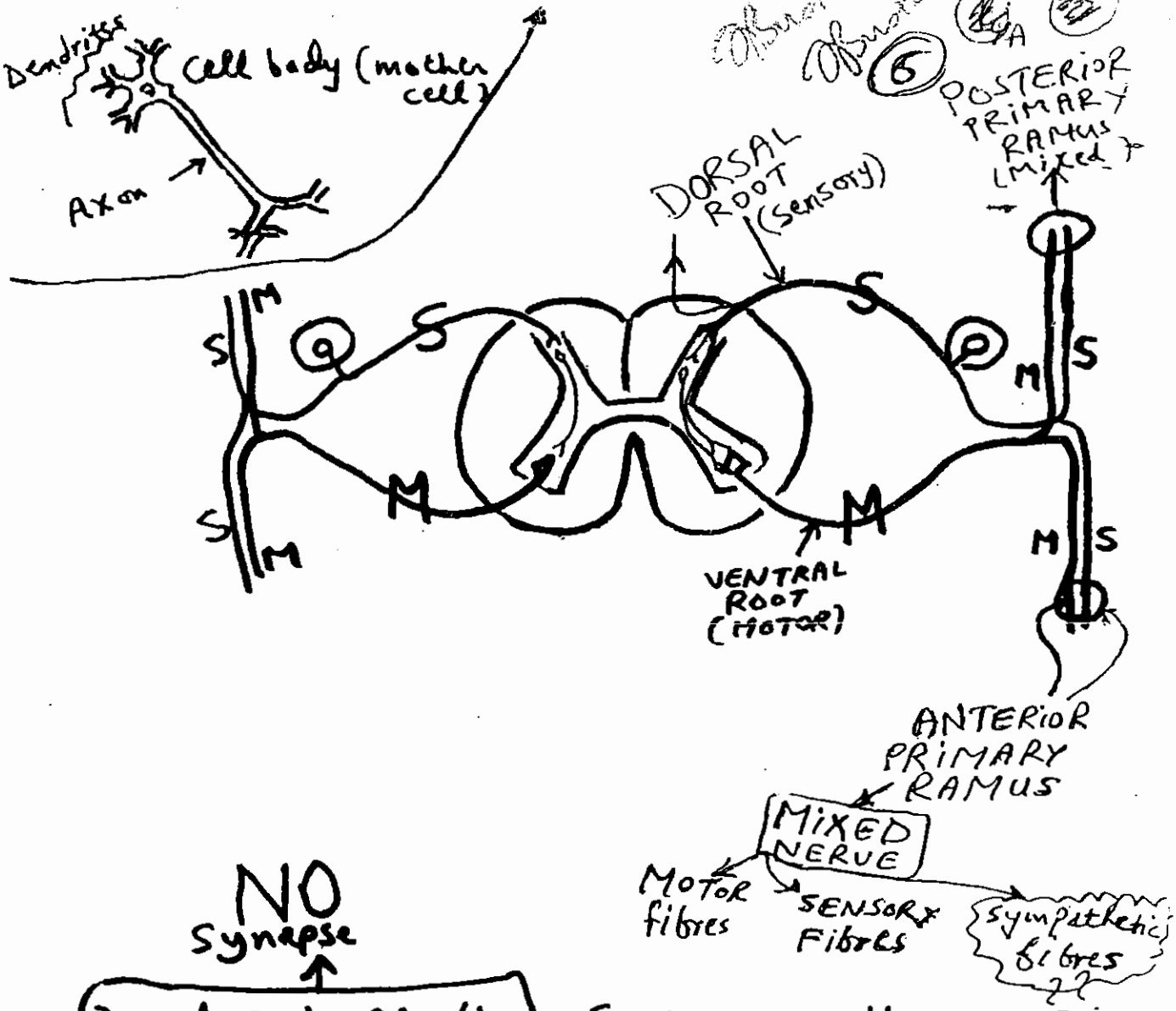
Lumbar → thick anterior & post. horns
post. column → gracile fasciculus

Sacral → thick ant. & post. horns
short post. horn
broad grey commissure
central canal → slit-like

cervical enlargement

lumbar enlargement

9(9)


Dendrites
Cell body (mother cell)
Axon
POSTERIOR PRIMARY RAMUS (Mixed)
DORSAL ROOT (Sensory)
VENTRAL ROOT (MOTOR)
ANTERIOR PRIMARY RAMUS
MIXED NERVE
Motor fibres
SENSORY Fibres
sympathetic fibres
NO Synapse
Dorsal root ganglion = Sensory ganglion = spinal ganglion
dendrite
axon
Dendrid-axon
Receptor
Sp. cord
central Process (axon)
peripheral Process (dendrite)
Pseudo-unipolar neuron

I. Overview—The Cerebral Cortex

–consists of the **neocortex** (90%) and the **allocortex** (10%).

6 B

A. Neocortex (isocortex; homogenetic cortex)

–is a six-layered cortex.

B. Allocortex (heterogenetic cortex)

–is three-layered and includes two types:

1. Archicortex

–includes the hippocampus and the dentate gyrus.

2. Paleocortex

–includes the olfactory cortex.

II. Six Layers of the Neocortex

–are expressed as Roman numerals I through VI:

I) **A. Molecular layer (I)**

–is the superficial layer located below the pia mater.

II) **B. External granular layer (II)**

III) **C. External pyramidal layer (III)**

–gives rise to association and commissural fibers.

IV) **D. Internal granular layer (IV)**

–receives thalamocortical fibers from the thalamic nuclei of the ventral tier (e.g., ventral posterolateral [VPL] and ventral posteromedial [VPM] nuclei).

–in the striate cortex (area 17), receives input from the lateral geniculate body.

* –myelinated fibers of this layer form the stripe of Gennari, which is visible to the naked eye.

V) **E. Internal pyramidal layer (V)**

–gives rise to corticobulbar, corticospinal, and corticostriatal fibers.

pyramidal

–contains the giant cells of Betz, which are found only in the motor cortex (area 4) of the precentral gyrus and the anterior paracentral lobule.

VI) **F. Multiform layer (VI)**

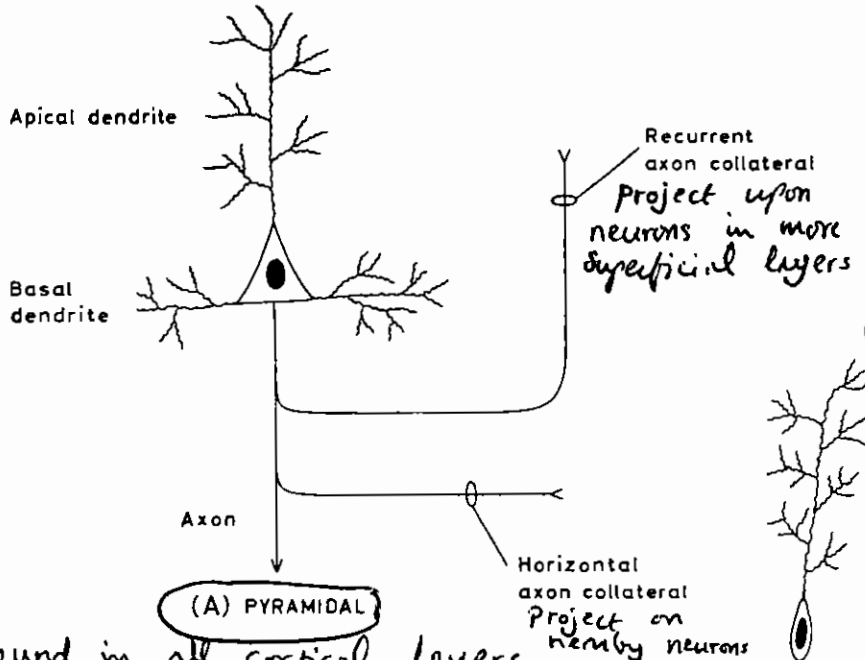
–is the deepest layer of the cortex. It gives rise to projection, commissural, and association fibers.

* Layers I, V, VI → present in all types of cortex ← Neo...
* layers II, III, IV → present only in NEOCORTEX ← Archi... Paleo

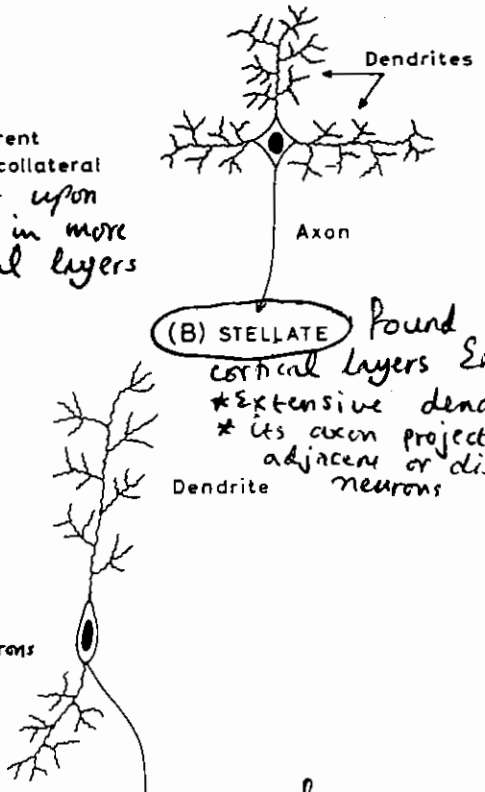
– In general layers I, II, III, IV are Receptive (Afferent)
layers V VI are Efferent

– pyramidal, stellate & Martinotti cells are present in all layers Except first layer

6c

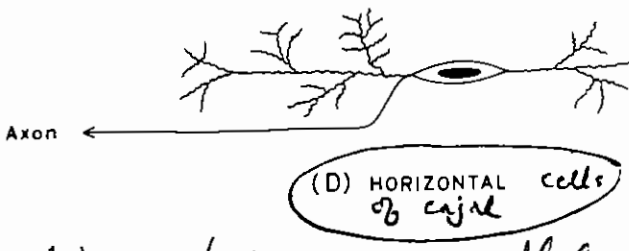


- found in all cortical layers Except Layer I
- the largest → Giant Pyramidal cells of Betz in layer V of motor cortex
- Its axon projects upon neurons in the <u>same</u> or <u>contralateral hemisphere</u> OR leave the cortex to project upon subcortical regions (projection fibres)

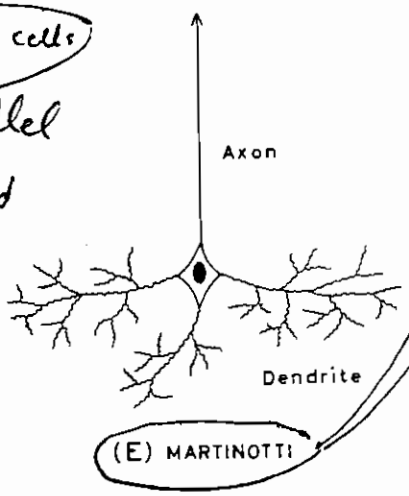


Found in all cortical layers Except I
* Extensive dendrites
* its axon project on adjacent or distant neurons

found in deep cortical laminae
the axon enters the deep white matter



→ dendrites and axon are parallel to the cortical surface
→ found ONLY in lamina I and disappear after the neonatal period



Multipolar
found in all cortical laminae Except layer I

Cortical neurons

Notice ⇒ <u>Golgi type I neurons</u>: with long axons are either { Pyramidal or fusiform

<u>Golgi type II neurons</u>: with short axons are either { Stellate, horizontal, Martinotti (é ascending axons)

OUTPUT OF CEREBRAL CORTEX

E. Efferent outflow from the cerebral cortex is grouped into three categories (Fig. 12.5). These are the association fiber system, commissural fiber system, and corticofugal fiber system. The association and commissural fiber systems have been described in the section on input to the cortex. Essentially they represent intrahemispheric and interhemispheric connections.

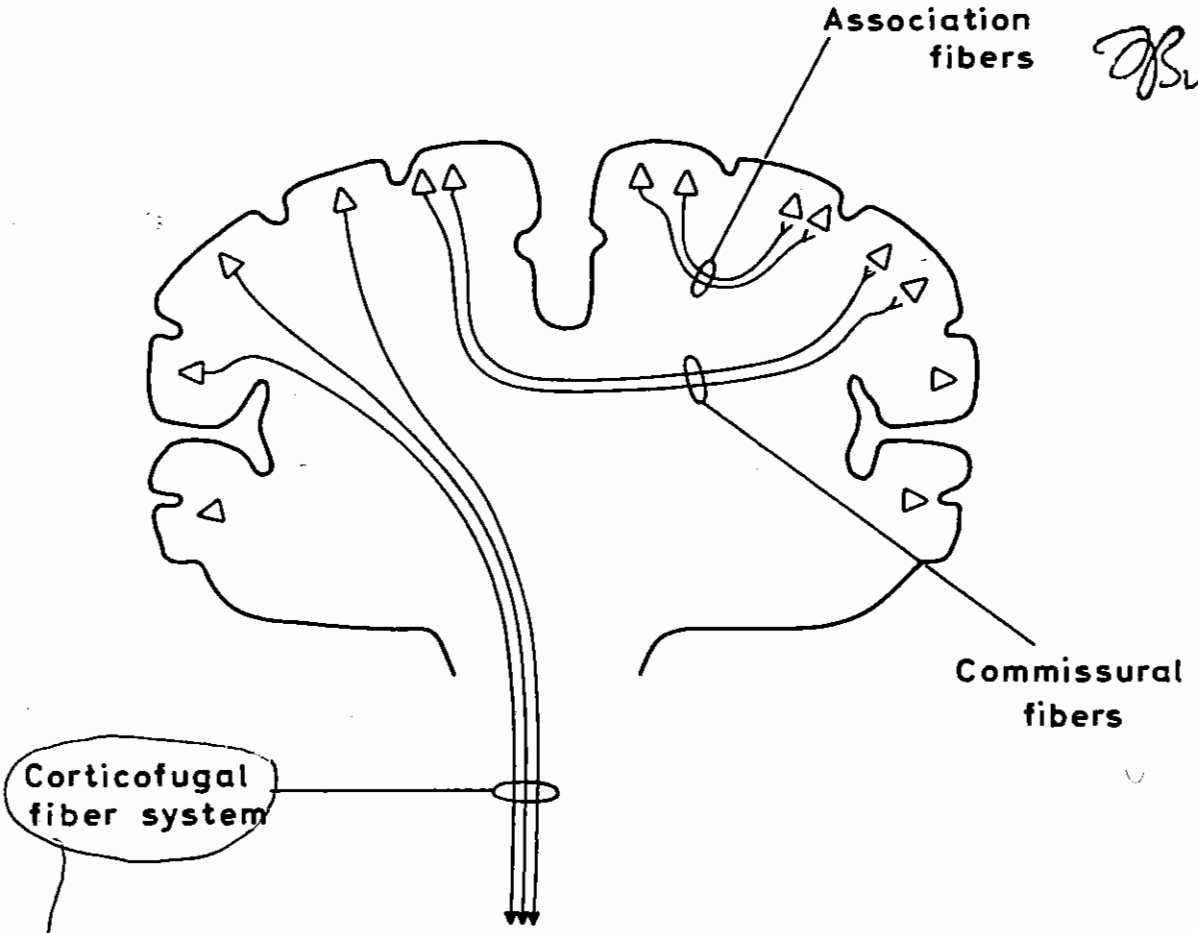


Figure 12.5. Schematic diagram of the major groups of cortical output.

- Corticospinal pathway (contains 1 million fibre → 9-22 μm size and arise from the giant cells of Betz in lamina V of the motor cortex)
- Corticobulbar pathway
- Corticotectal "
- Corticopontine "
- Corticothalamic "

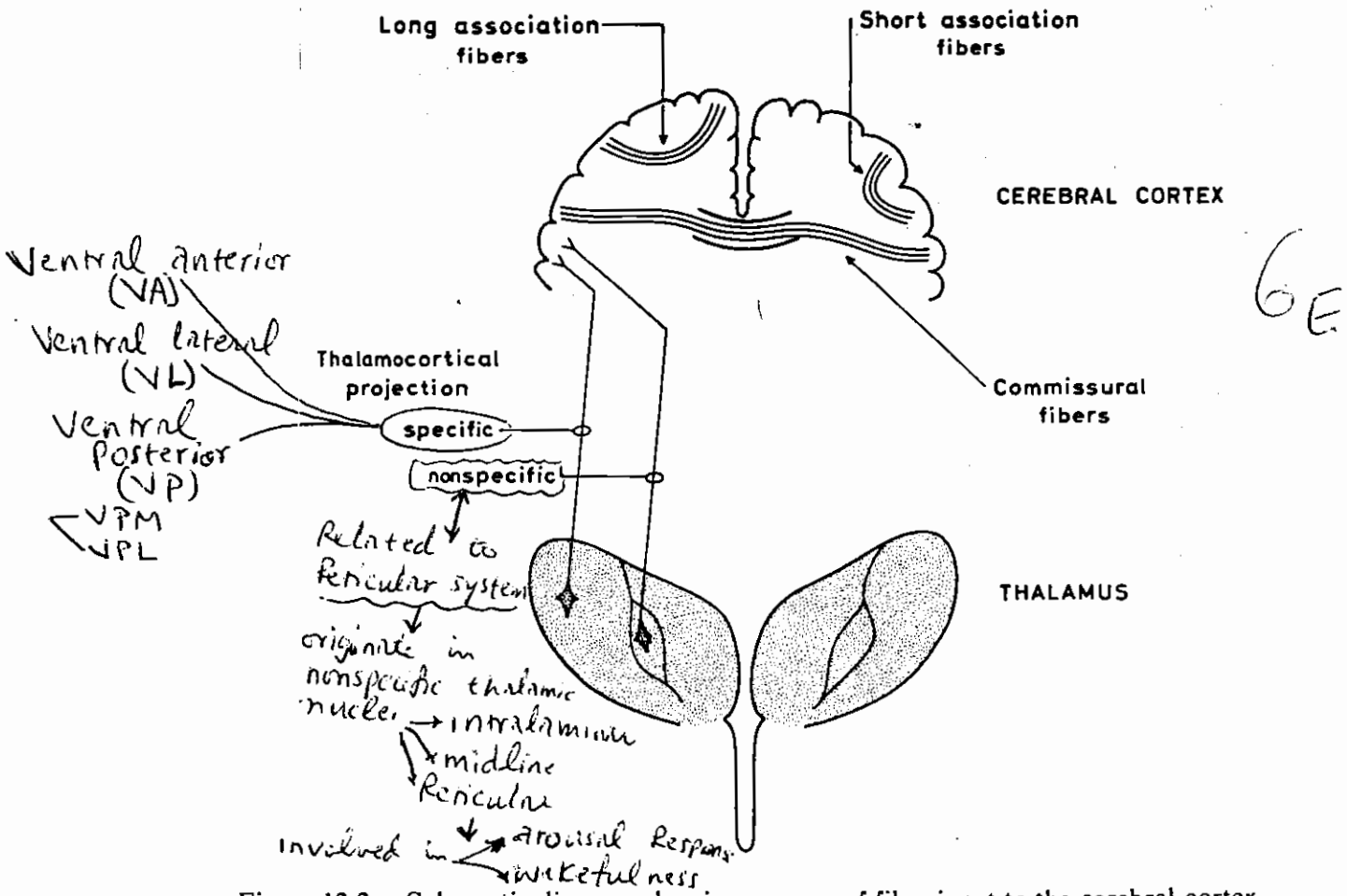


Figure 12.2. Schematic diagram showing sources of fiber input to the cerebral cortex.

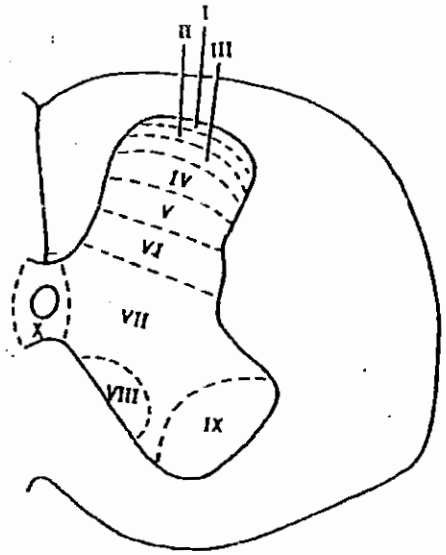
INPUT TO CEREBRAL CORTEX

The input to the cerebral cortex originates in three sites (Fig. 12.2).

1. Thalamus
2. Cortex of the same hemisphere (association fibers)
3. Cortex of the contralateral hemisphere (commissural fibers)

The input from the thalamus travels via two systems. 1) The specific thalamocortical system originates in specific thalamic nuclei (ventralis anterior, ventralis lateralis, ventralis posterior, etc.) and projects upon specific cortical areas. This fiber system reaches the cortex as an ascending component of the internal capsule. The majority of fibers in this system project upon neurons in lamina IV, with some projecting upon neurons in lamina III (Fig. 12.3). 2) The nonspecific thalamocortical system is related to the reticular system and originates in nonspecific thalamic nuclei (intralaminar, midline, and reticular nuclei). In the cortex, fibers of this system project diffusely upon all laminae (Fig. 12.3) and establish mostly axodendritic types of synapses. This fiber system is intimately involved in the arousal response and wakefulness.

The association fibers arise from nearby (short association fibers) and distant (long association fibers) regions of the same hemisphere. They too project diffusely in all laminae (Fig. 12.3), but mostly in laminae I to III. The long association fiber system includes such bundles as the uncinate fasciculus, the cingulum, the superior and inferior longitudinal fasciculi, and the occipitofrontal fasciculus (Fig. 12.4).



Schematic diagram of half of the spinal cord showing the location of Rexed

Laminae I to IV are concerned with exteroceptive sensations, whereas laminae V and VI are concerned primarily with proprioceptive sensations, although they respond to cutaneous stimuli. Lamina VII acts as a relay between midbrain and cerebellum. Lamina VIII modulates motor activity, most probably via the gamma neuron. Lamina IX is the main motor area of the spinal cord. It contains large alpha and smaller gamma motor neurons. The axons of these neurons supply the extrafusal and intrafusal muscle fibres respectively.

Table 5.1. Cellular Organization of Spinal Cord

Rexed terminology	Older terminology
Lamina I	Posteromarginal nucleus
II	Substantia gelatinosa
III, IV	Nucleus proprius
V	Neck of posterior horn
VI	Base of posterior horn
VII	Intermediate zone, intermediolateral horn
VIII	Commissural nucleus
IX	Ventral horn
X	Grisea centralis

The motoneurons of the spinal cord are arranged in *columns* which supply muscle groups having similar functions. The individual muscles are supplied from cell groups (nuclei) within the columns. Medially placed columns supply the axial (trunk) musculature. Laterally placed columns, present only in the cervical and lumbar enlargements, supply the limb musculature. Finally, motoneurons innervating extensor muscles lie in front of motoneurons innervating flexors (Fig. 10-1, Table 10-1).

Table 10-1 Motor cell columns

Cell column	*Muscles*
Ventromedial (all segments)	Erector spinae
Dorsomedial (T1–L2)	Intercostals, abdominals
Ventrolateral (C5–8, L2–S2)	Arm/thigh
Dorsolateral (C6–8, L3–S3)	Forearm/leg
Retrodorsolateral (C8, T1, S1, S2)	Hand/foot
Central (C3, C4, C5)	Diaphragm

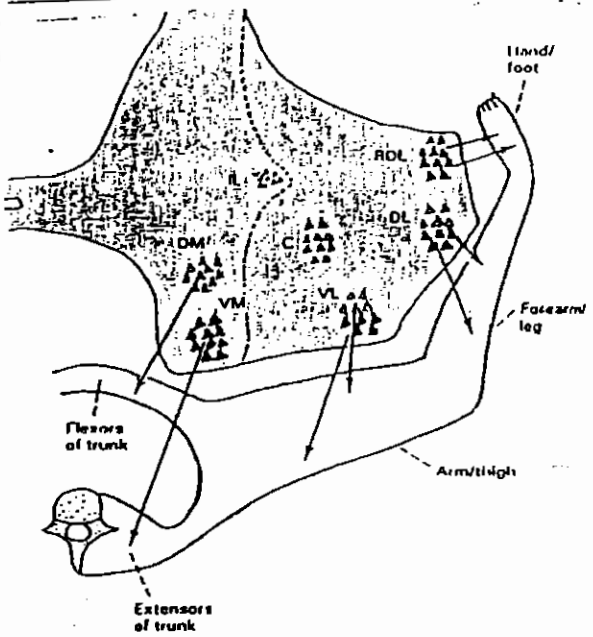


Fig. 10-1 Cell columns in the gray matter. Dotted line indicates limit of gray matter at thoracic level. C, central; DL, dorsolateral; DM, dorsomedial; IL, intermediolateral (autonomic); RDL, retrodorsolateral (for intrinsic muscles); VL, ventrolateral; VM, ventromedial nucleus.

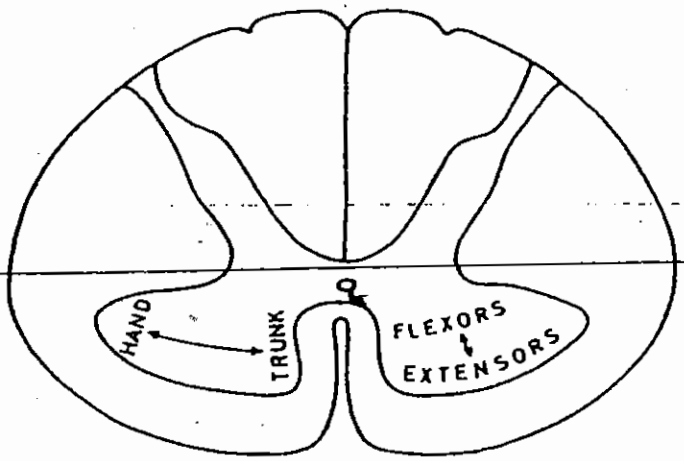
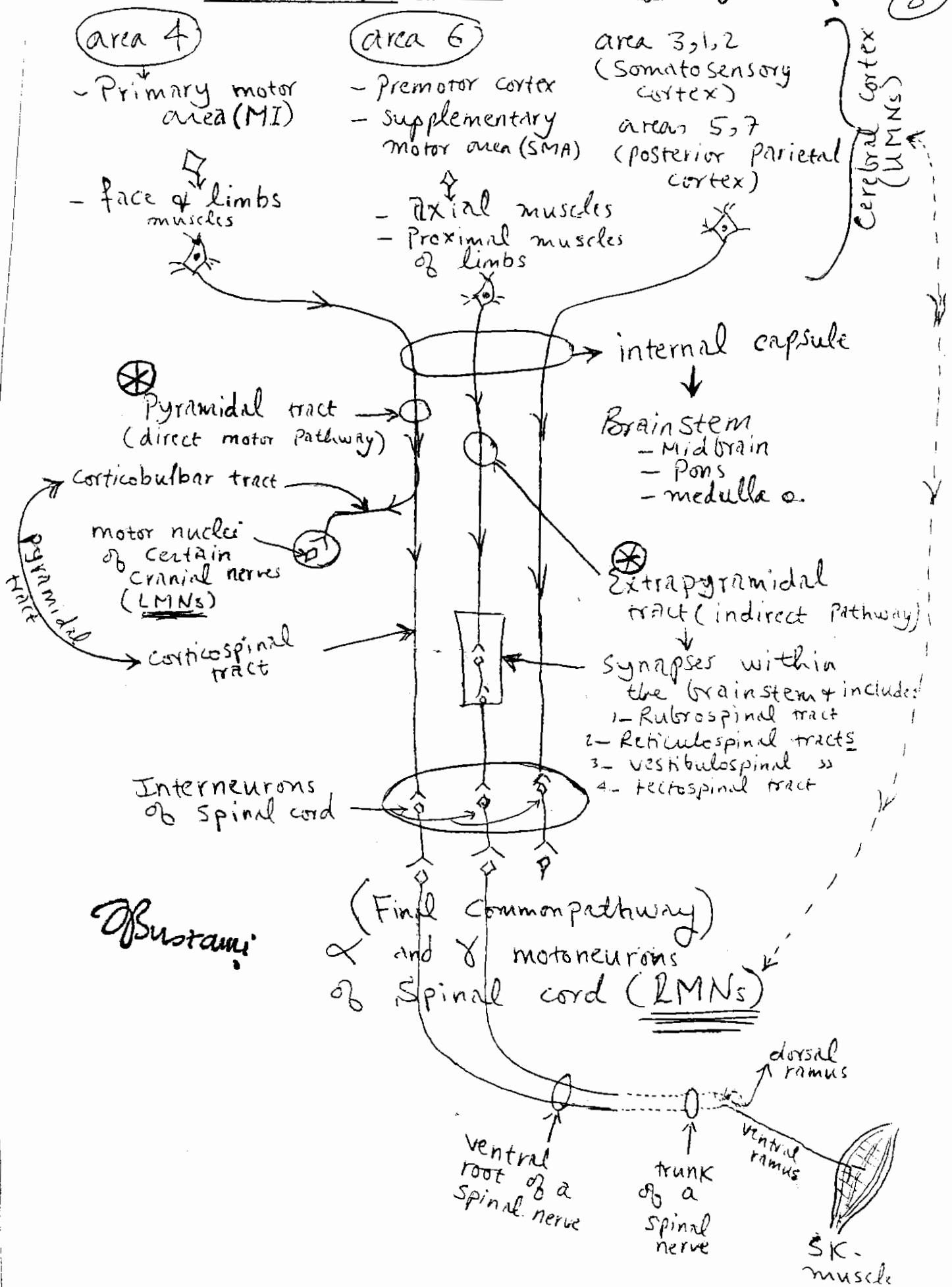


Figure 5.6.—Schematic diagram of the spinal cord showing somatotopic organization of ventral horn neurons.

Descending Motor pathways ©Bustami



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Descending Tracts (Motor Pathways) (9)

- A tract or pathway ? a bundle of nerve fibres that have the same origin, same termination and carry same function. ©Bustami
- The descending tracts or the motor pathways START at Supraspinal neurons present in the cerebral cortex (e.g areas (4) & (6)) or in the medulla, Pons or midbrain (e.g neurons of reticular formation or red nucleus or superior colliculus). All these neurons which give rise to the descending motor pathways are called (UPPER MOTOR NEURONS)
- The (LOWER MOTOR NEURONS) are present in:-
  - → Brainstem ← medulla, pons, midbrain } where they give rise to motor fibres in certain cranial nerves
  - → Spinal cord → these are the alpha (α) and gamma (γ) motor neurons present in the ventral horn of grey matter of Spinal cord and give rise to motor fibres in every spinal nerve
- The motor pathways run between the upper and lower motor neurons, however they do not synapse directly with the lower motor neurons (→ (α and γ) ←) but mostly through interneurons

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Descending (motor) tracts

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- PYRAMIDAL
  - Corticospinal
  - Corticobulbar (Corticonuclear)
- Extra Pyramidal

* Corticospinal tract → its name indicate that it begins in the cerebral cortex (areas 4, 6, 3, 1, 2) & terminates in the lower motor neurons of spinal cord (α and γ) mostly through interneurons. This pathway is concerned with voluntary skilled movement especially those of the distal parts of the limbs (hands & feet)
* Corticobulbar (Corticonuclear) tract → has the same origin as the corticospinal tract however it DOES NOT reach the spinal cord → it terminates in the brainstem by synapsing on the motor nuclei of certain cranial nerves Mostly Bilaterally
* Extra Pyramidal tract: a group of descending tracts that arise (or is) in the brainstem but are under the influence of the cerebral cortex e.g

① Reticulospinal tract < medial (Pontine) / lateral (medullary)
they arise from the reticular formation in the pons and medulla and terminate on α and γ motoneurons mostly through interneurons in laminae VII and VIII

② Rubrospinal tract from the Red nucleus in the midbrain to the spinal cord. It runs very close to the lateral corticospinal tract

③ Vestibulospinal tract < medial / lateral from the vestibular nuclei in the brainstem to the spinal cord

④ Tectospinal tract: from the Superior Colliculus of the midbrain to the spinal cord (cervical and upper thoracic regions)

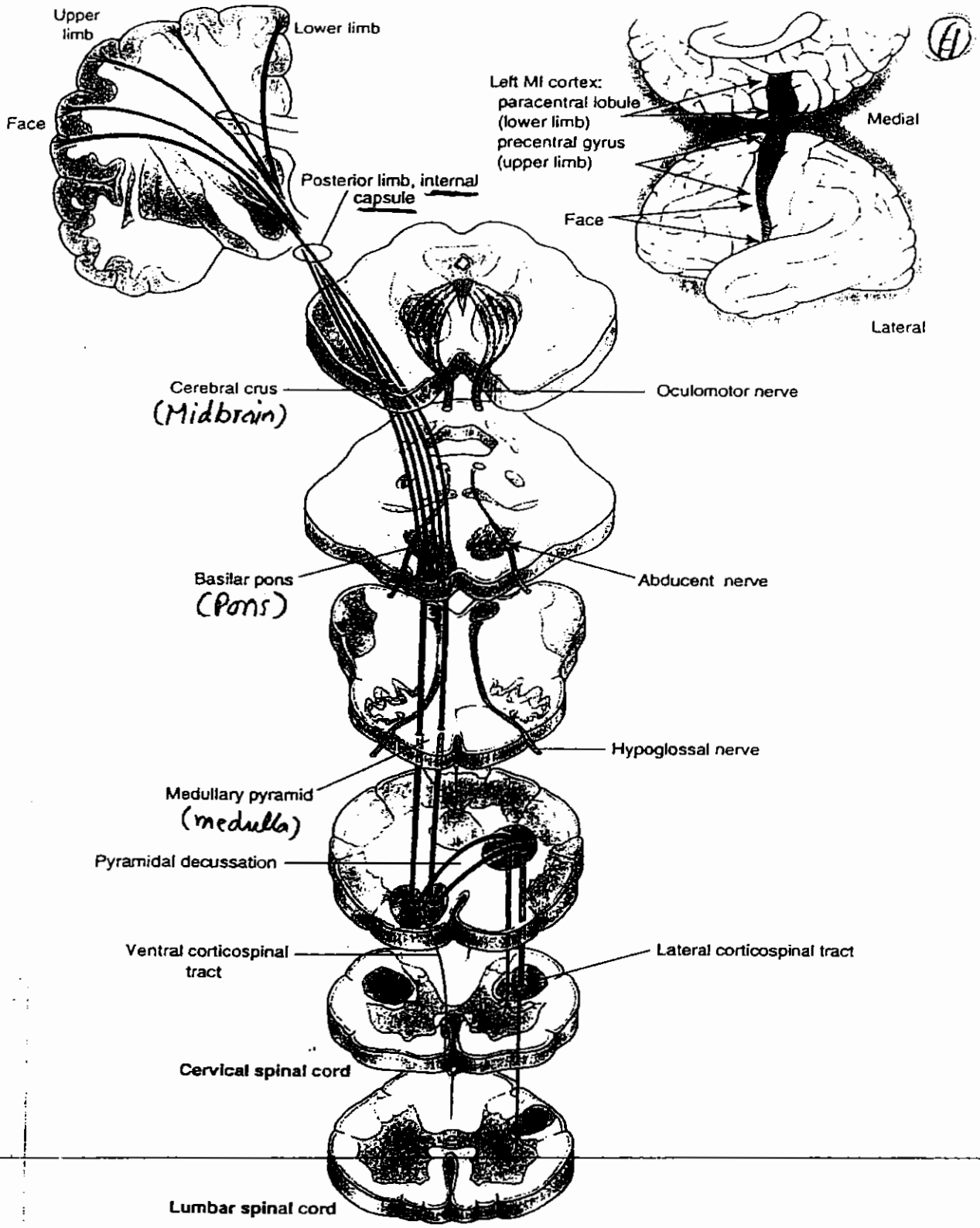


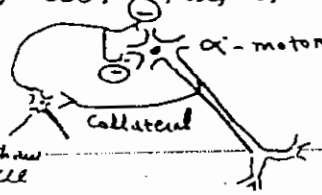
FIGURE 6–2. Schematic diagram of the pyramidal tract, showing its origin, course, and relations.

Alpha-Motoneurons: The only way in which the central nervous system can cause skeletal muscle fibers to contract is by evoking discharges in α-motoneurons. Therefore all motor acts DEPEND ON NEURAL CIRCUITS ⇒ that eventually impinge on α-motoneuron. This is why the α-motoneuron is called → the FINAL COMMON PATHWAY

Spinal cord interneurons: most of the synapses on α-motoneurons originate from spinal cord interneurons. By definition interneurons are neurons interposed between primary afferent neurons and motoneurons. Interneurons whose processes are confined to the spinal cord are often called propriospinal neurons.

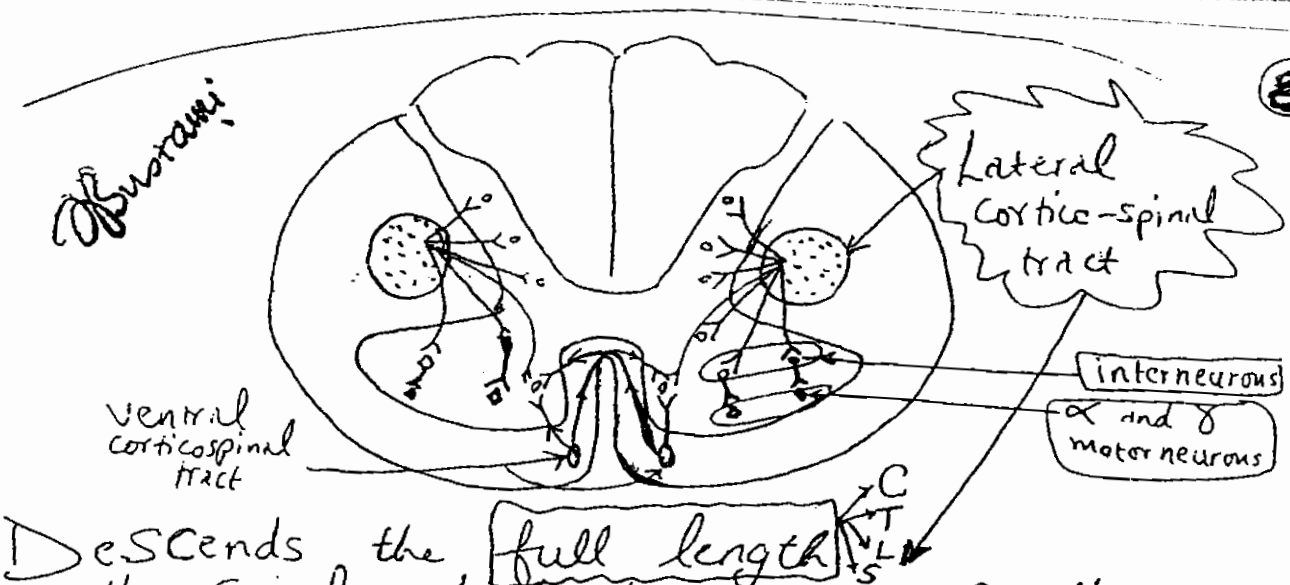
- Most spinal cord interneurons are located in the dorsal horn. Many of these are involved in sensory processing and contribute directly or indirectly to the transmission of sensory information to the brain. However, neurons in the dorsal horn also project to the ventral horn and affect the discharges of motoneurons. Furthermore ⇒ axons of descending pathways from the brain RARELY TERMINATE DIRECTLY ON MOTONEURONS → Axons in descending pathways usually end on interneurons and alter motor output by changing the level of activity in spinal cord circuits.

Renshaw cells → are inhibitory interneurons located in the part of Lamina VII that protrudes ventrally between the lateral part of lamina IX and lamina VII. RECURRENT COLLATERALS FROM α-Motor axons SYNAPSE ON RENSHAW CELLS → When the motor axons discharge, they release acetylcholine at the synapses on Renshaw cells and excite these cells. The Renshaw cells in turn synapse on and *inhibit α-motoneurons; thus when motoneurons discharge, this causes an inhibitory feedback by way of Renshaw cells. This is called → Recurrent inhibition.



α-motoneurons are always under tonic inhibition by Renshaw cells → Loss of this inhibition (by strychnine) ← Excessive firing of α-cells ← convulsions etc.

Anatomy



Descends the full length of the spinal cord, beside the base of the posterior horn → It is the principle voluntary motor pathway of has facilitatory effect on α & γ motoneurons that supply distal flexor muscles

Termination of axons of lateral corticospinal tract

On interneurons within laminae VI – VIII

→ Direct projection of a small number of axons on the α and γ motor neurons in lamina IX (9) ???

The lateral corticospinal tract synapses on:

① interneurons of the dorsal horn where it modulates sensory transmission

② α MNs and γ MNs (mostly through interneurons)

- 55% of its fibres eventually influence the α and γ MNs of Cervical part of Spinal cord
- 20% ------ thoracic part ------
- 25% ---------- lumbosacral part ----

The lateral corticospinal tract acts primarily on the distal muscles of the limbs while the ventral corticospinal tract acts on the Proximal muscles

The ventral corticospinal tract acts on the Proximal muscles of the upper limb (shoulder muscle) of the ipsilateral (same) and contralateral sides.

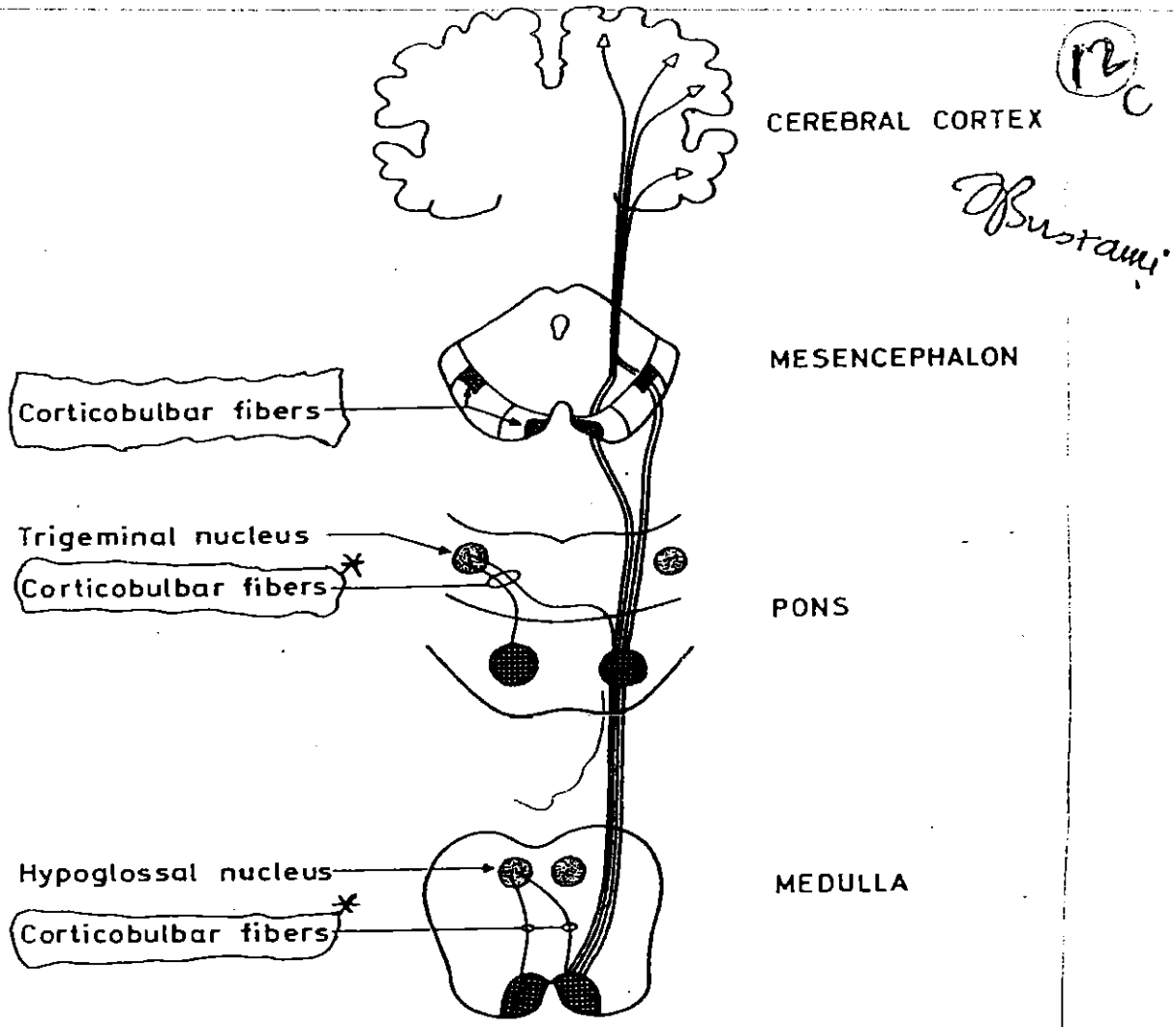


Figure 12.8. Schematic diagram of the corticobulbar pathway.

* CORTICOBULBAR Pathway:
FROM the cerebral cortex to → MOTOR Nuclei of certain cranial nerves → 5, 7, 9, 10, 11, 12

* The Corticobulbar input to (5) part of (7) which supplies upper facial muscles, 9, 10, 11, (12) ?? → Bilateral

* The input to that part of the facial nucleus which supplies lower facial muscles is from the contralateral hemisphere only.

* Bilateral interruption of the corticobulbar fibres Results in Paresis (weakness) but Not paralysis of the muscles supplied by the corresponding cranial nerve nucleus → Pseudobulbar palsy

* Bulbar palsy → complete paralysis ..... as a result of lesion of nucleus

most dorsolaterally and are limited to the most caudal segments of the cervical and lumbosacral enlargements, respectively.

SPINAL MOTOR NEURONS

The spinal alpha motor neurons innervating an individual muscle or a particular group of muscles are arranged in longitudinal columns extending for various distances in a specific part of the anterior horn. The medial cell column extends the entire length of the spinal cord and innervates the paravertebral or axial muscles. The lateral cell column, which is found at the spinal cord enlargements, innervates the muscles of the limbs. Within the lateral cell column further somatotopic organization exists: the proximal limb muscles are represented medially and the distal muscles, laterally (Figs. 5-11, 5-12, 7-1). The most distal muscles (in the fingers and toes) are represented

THE PROPRIOSPINAL SYSTEM OF NEURONS

All movements require the activity of lower motor neurons in more than one spinal cord segment. The number of segments involved in a movement varies. Because axial movements depend on the activity of muscles that extend for great distances along the vertebral column, the paravertebral muscles are innervated by numerous spinal nerves. In contrast, individual finger movements are controlled by the intrinsic muscles of the hand that are innervated by only spinal nerves C8 and T1.

The intersegmental activity required for any particular movement is integrated by the propriospinal system of neurons. The propriospinal

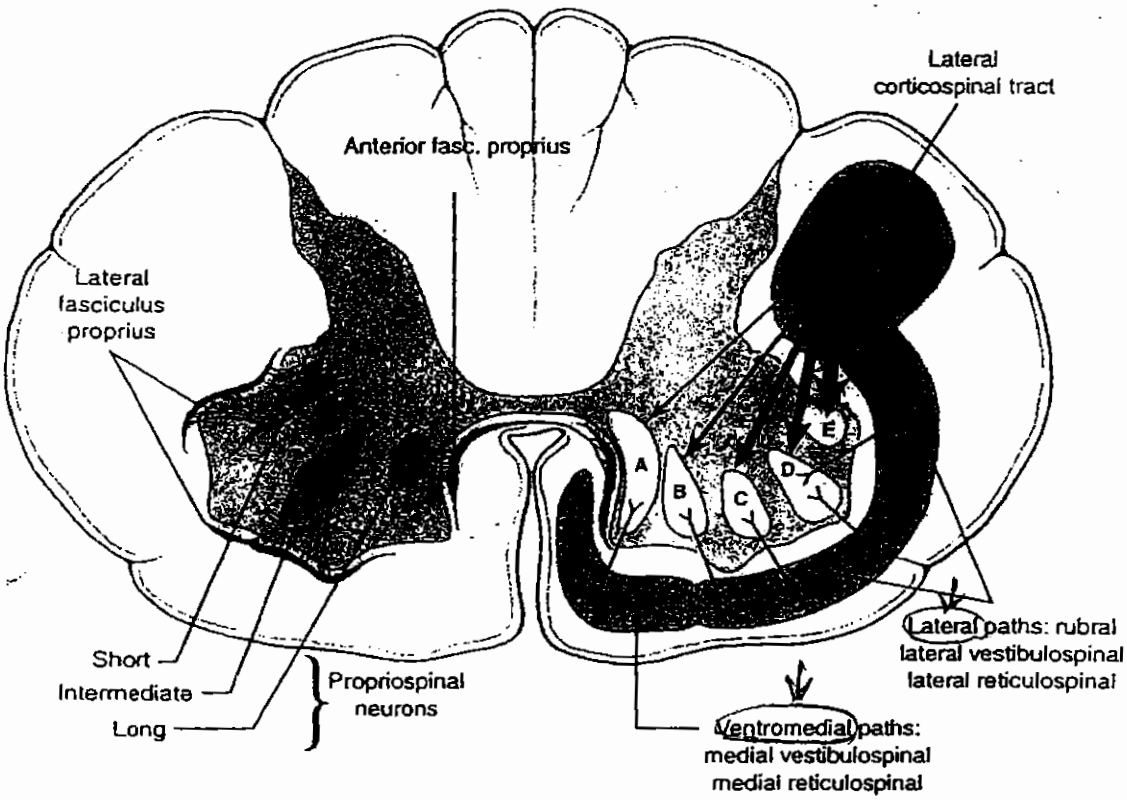


Figure 7-1 Motor organization of a spinal cord segment in the cervical enlargement (A, axial; B, shoulder; C, arm; D, forearm; E, hand; fasc, fasciculus).

(12) E

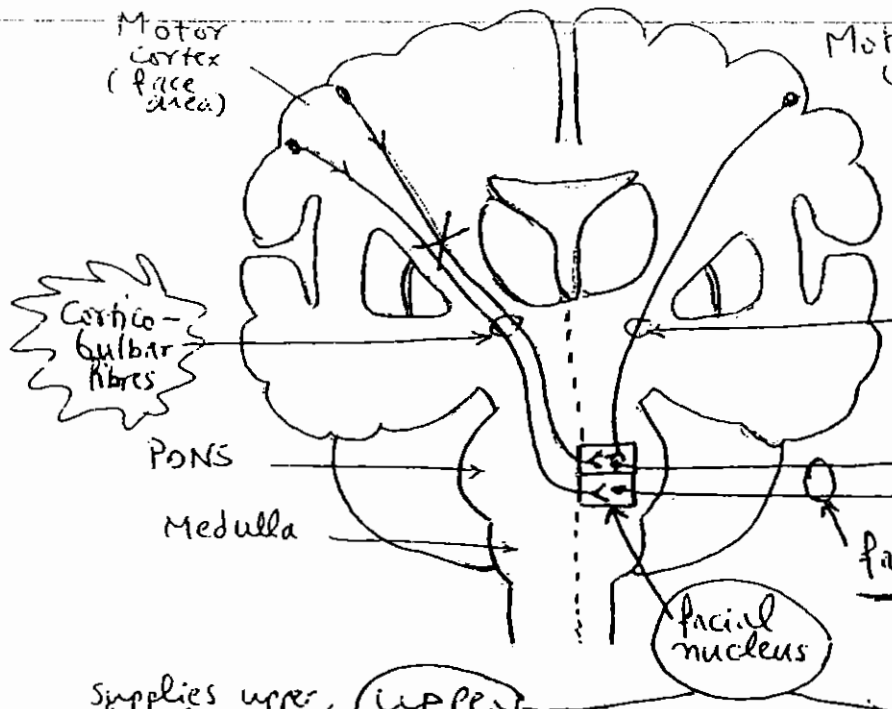
Bastani

system includes three groups of intraspinal neurons whose axons influence homologous areas of the spinal cord gray matter at different levels by traveling through the fasciculi proprii bordering the gray matter (Fig. 7-1):

1. The long propriospinal neurons have axons that ascend and descend in the anterior fasciculus proprius to all levels of the spinal cord. These neurons have a bilateral influence on the more medial motor neurons subserving movements of the axial muscles.
2. The intermediate propriospinal neurons have axons that extend for shorter distances in the ventral part of the lateral fasciculus proprius and influence the motor neurons that innervate the more proximal muscles of the limbs.
3. The short propriospinal neurons are limited to the cervical and lumbosacral enlargements. Their axons travel in the lateral fasciculus proprius and terminate within several segments of their origin. These propriospinal neurons influence the motor neurons that innervate the more distal muscles of the limbs.

Propriospinal Neurons

	Ascend & descend	Influence on which motor neurons?
1. Long	at all levels of the sp. cord (in anterior fasciculus proprius)	Bilateral influence on more medial motor neurons affecting movements of AXIAL MUSCLES
2. Intermediate	extend for short distance in the ventral part of the lateral fasciculus proprius	Influence motor neurons that innervate proximal muscles of limbs
3. Short	- limited to cervical & lumbosacral enlargements - extend for short distance - travel in the lateral fasciculus proprius	Influence motor neurons that innervate more distal muscles of the limbs



Obeikandi 13

supplies upper facial muscles ← upper part

Receives contralateral & ipsilateral corticobulbar (corticonuclear) fibres

lower part (supplies peri-oral muscles of lower part of face)

↓

receives only contralateral corticobulbar fibres

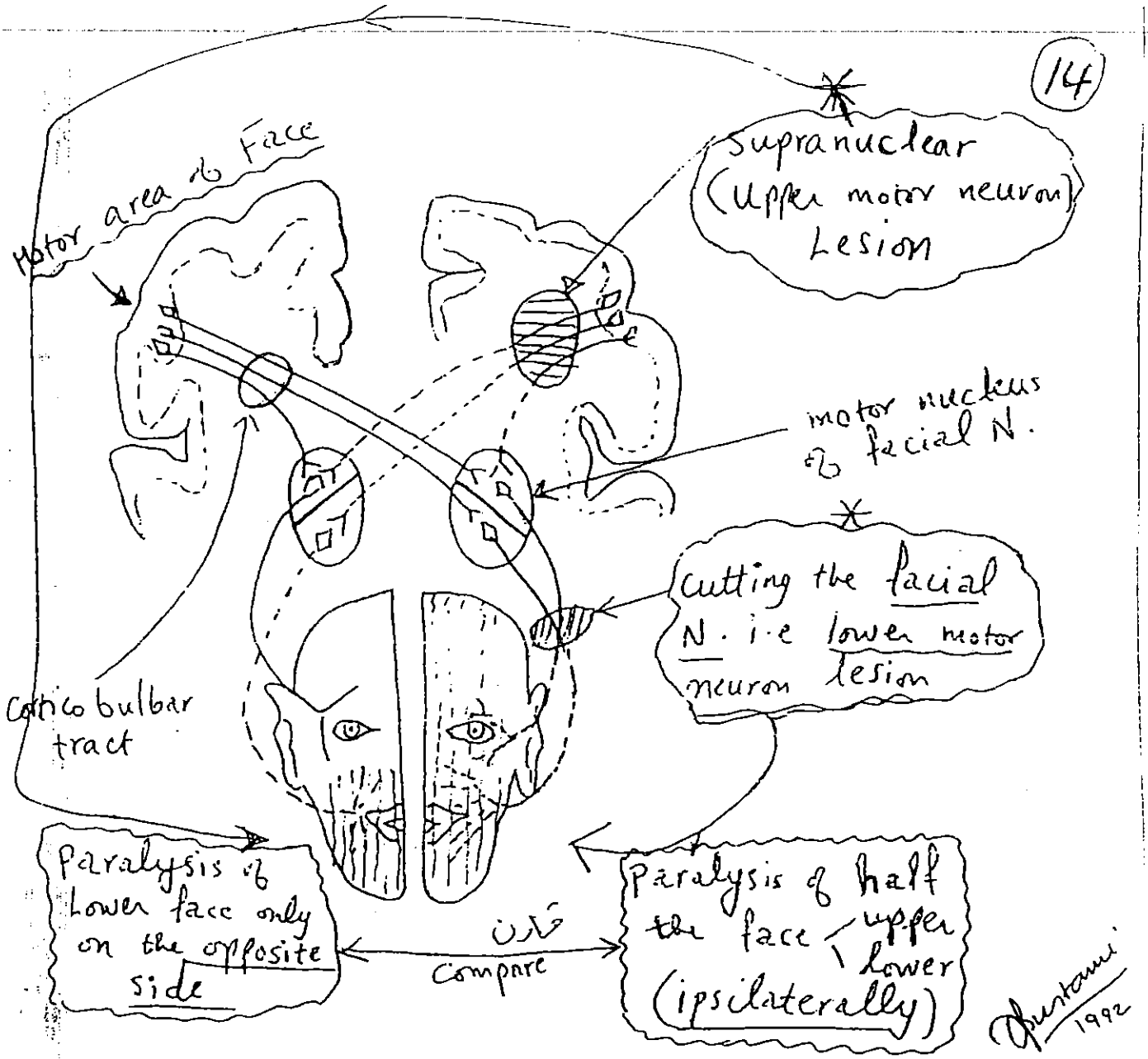
↓

Lesion affecting the corticobulbar fibres on ONE side (usually called upper motor neuron lesion or stroke)

↓

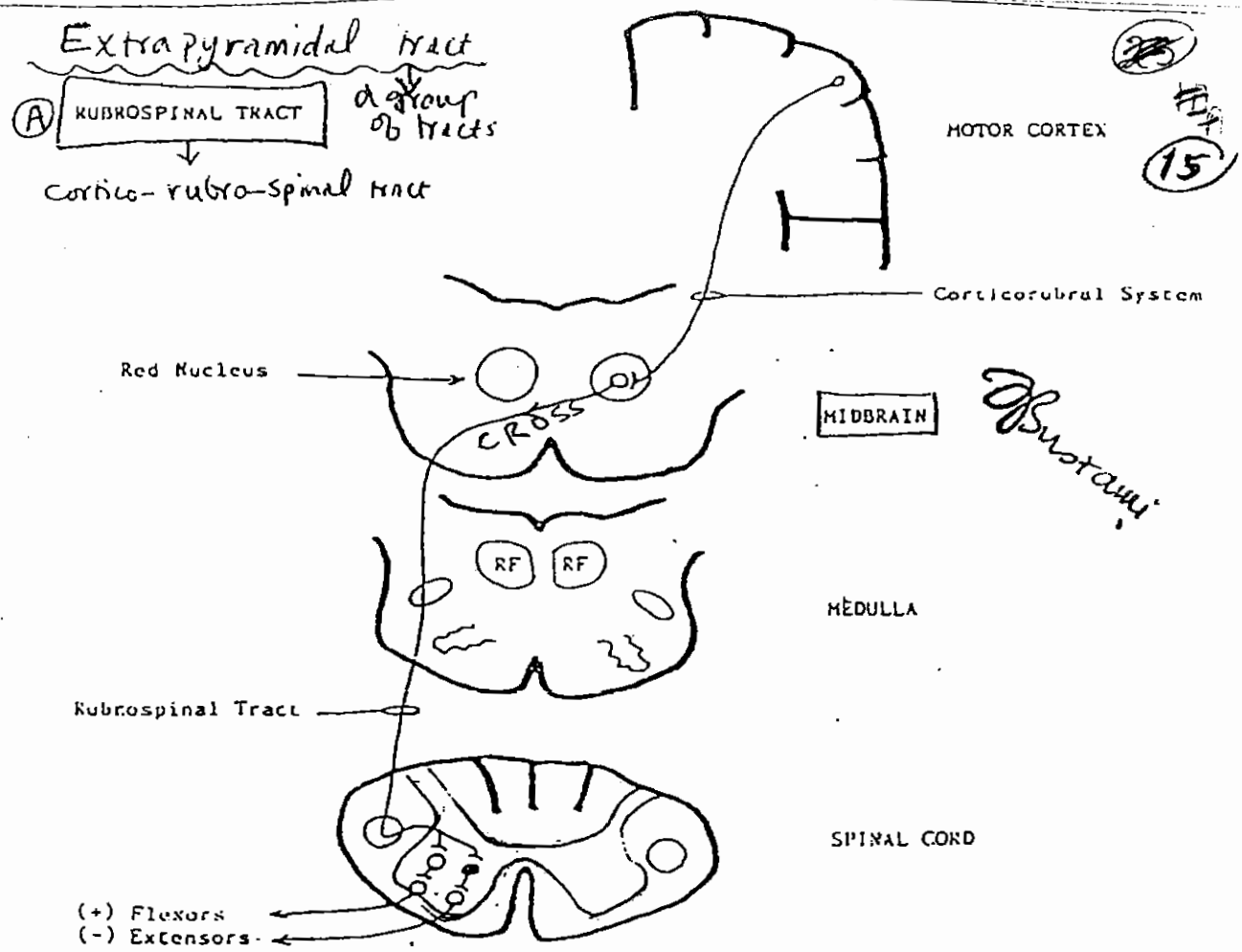
Weakness (Paresis) of lower facial muscles (peri-oral muscles) CONTRALATERAL to the side of lesion (the upper facial muscles are much less affected ?? → Remember they receive bilateral cortico-bulbar fibres.

- Remember → muscles of Pharynx & Larynx receive bilateral corticobulbar fibres → significance ??



In a case of STROKE when the lesion involves the Left corticobulbar fibres → The patient will show weakness in the contralateral lower half of his face (perioral muscles) & the upper half will show less weakness WHY ?

→ Look to the nucleus of facial nerve i.e (Motor nucleus) → its upper part (which supplies the upper face) receives both ipsilateral & contralateral corticobulbar fibres while its lower part (which supplies the lower face) receives ONLY contralateral corticobulbar fibres → which part of the nucleus will suffer more ? ? ?



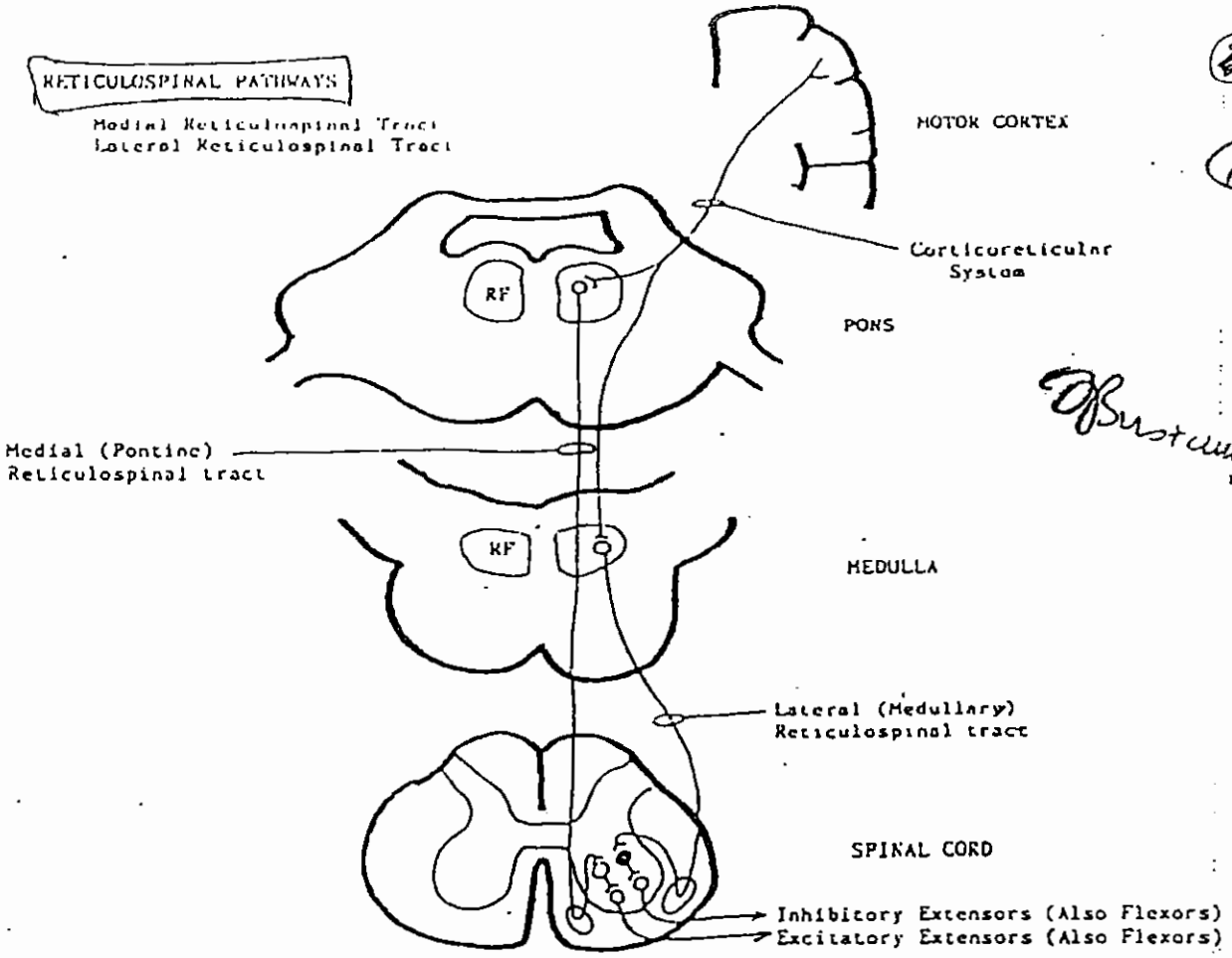
A. Rubrospinal Tract

This tract originates in the red nucleus. Fibers project to interneurons in the lateral region of the spinal cord. Stimulation of the red nucleus causes facilitation of flexors (+++) and inhibition of extensors (---).

Notice — rubrospinal tract is really cortico-rubro-spinal tract (i.e indirect corticospinal tract)

the rubrospinal tract CROSSES in the ventral tegmental decussation of midbrain & continues in the lateral funiculus of spinal cord VERY CLOSE to the LATERAL CORTICO-SPINAL TRACT → terminate on the same laminae as the lateral corticospinal tract i.e laminae IV to VIII

Rubrospinal ⊕ lateral corticospinal tracts form the LATERAL MOTOR SYSTEM (influence α and γ motoneurons present at the LATERAL part of ventral horn which supply DISTAL FLEXOR MUSCLES)



B. Pontine (medial) Reticulospinal Tract

This tract originates from cells in the nucleus pontis caudalis and nucleus reticularis pontis oralis located in the medial two thirds of the pons (Pontine reticular formation). Fibers project to the ventromedial spinal cord where they have a general excitatory effect* on both extensor and flexor motoneurons, although maximal excitation is on the extensors.

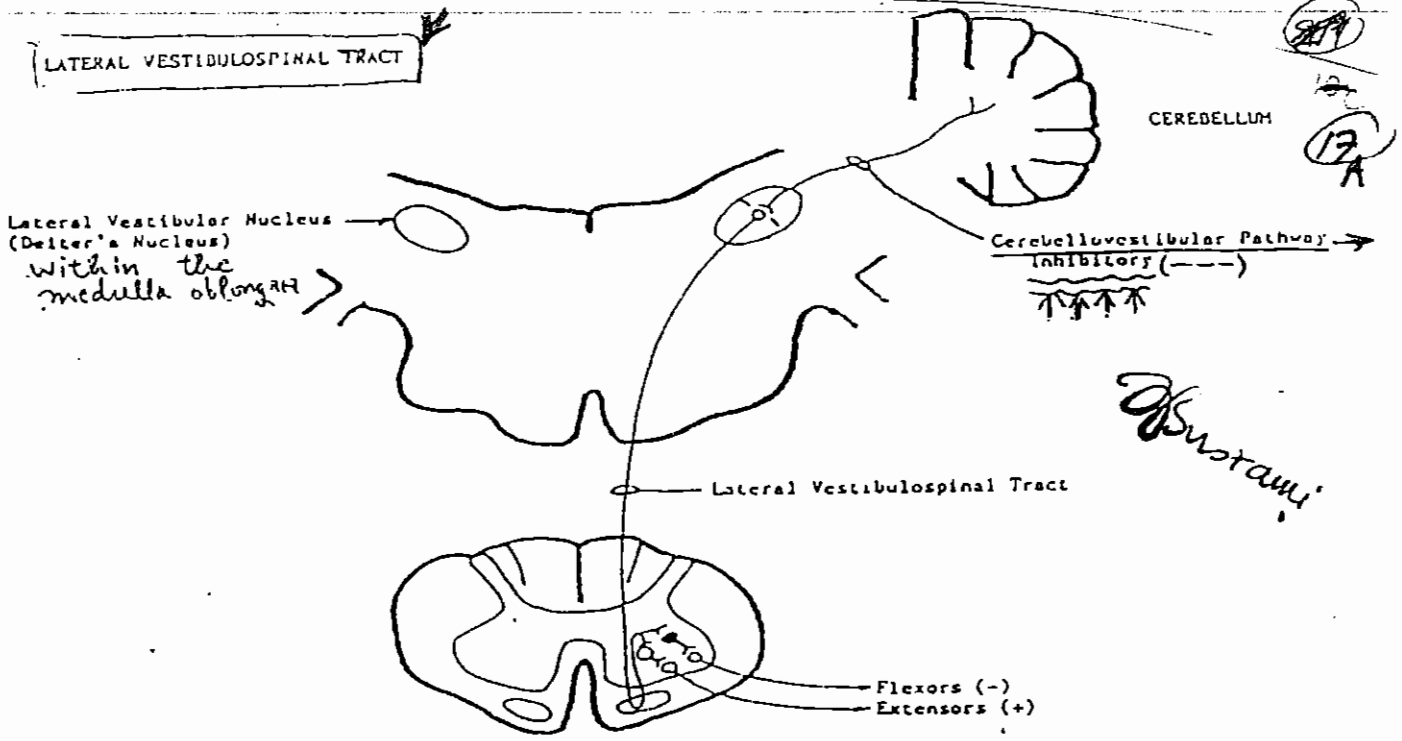
C. Medullary (lateral) Reticulospinal Tract

Cells originate in the medullary reticular formation (nucleus reticularis gigantocellularis) and terminate on spinal cord interneurons in the intermediate gray. The medullary reticulospinal tract has the opposite effect of the Pontine reticulospinal tract, in that it has a general inhibitory effect* on motoneurons with a stronger inhibition on extensors.

Notice → both Reticulospinal tracts form part of the MEDIAL MOTOR SYSTEM → they synapse with α and γ motor neurons present at the MEDIAL part of ventral horn which supply Axial (trunk) & Proximal muscles (of limbs)

both reticulosp. tracts synapse on (inter-neurons) within laminae VII & VIII

the reticulospinal tracts are the Principle Supraspinal pathways that control POSTURE such as sitting & standing and automatic movements such as walking & running



D. Lateral Vestibulospinal Tract

Cells originate in the lateral vestibular nucleus (Deiters' nucleus) and project to ipsilateral motoneurons and interneurons. Stimulation of cells in Deiters' nucleus produces a powerful excitation of extensors and inhibition of flexors It plays an important role in the control of antigravity muscles and the maintenance of posture.

Notice → Both lateral vestibulospinal tract ⊕ Pontine(medial) reticulospinal tract are Excitatory to EXTENSOR muscles while the medullary(lateral) reticulospinal tract is inhibitory to extensors

→ Cells in the lateral vestibular nucleus (Deiter's nucleus) are normally inhibited by projections from the cerebellum → Removal of cerebellum (in experimental animals) → increases the activity of these cells → increase tone in extensor muscles

E. Medial vestibulospinal tract: descends as a component of medial longitudinal fasciculus (MLF) and is excitatory to flexor motor neurons

Notice → Both vestibulospinal tracts → lateral / medial } form part of the MEDIAL MOTOR SYSTEM

F. Tectospinal Tract

Cells of origin are in the superior colliculus. Fibers project to the cervical spinal cord where they control neck muscles involved in head movement.

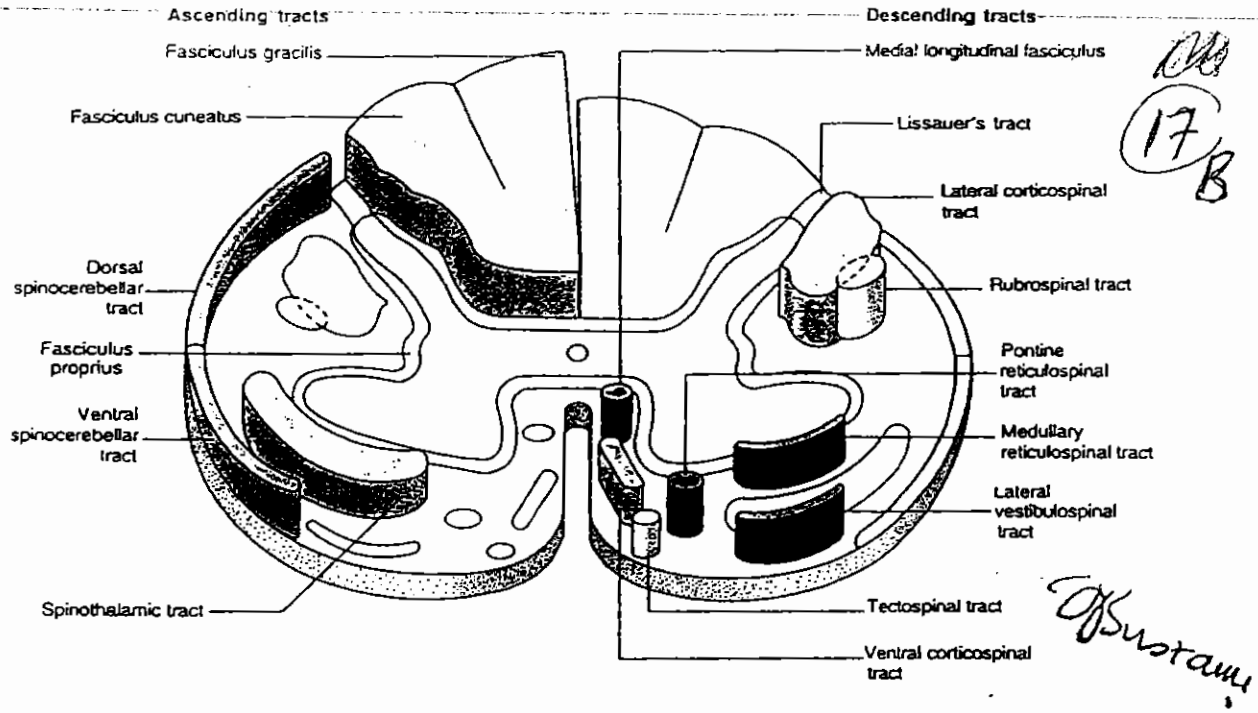


Fig. 5.13 Ascending and descending tracts of the spinal cord.
All ascending and descending tracts are present bilaterally. In this figure, ascending tracts are emphasised on the left side and descending tracts are emphasised on the right side. In addition, the locations of Lissauer's tract and the fasciculus proprius (which contain both ascending and descending fibres) are shown.

Descending motor Pathways ⇒ classified according to their site of termination in the spinal cord

Lateral System

Ends on α-motoneurons in the lateral part of lamina IX OR on the interneurons that project to these neurons.

⇩

Controls muscles of the distal and part of the proximal limbs

⇩

These muscles subserve fine movements used in manipulation and other precise actions especially of the digits

⇩

Includes 2 pathways from the brain to the spinal cord → the lateral corticospinal tract and the rubrospinal tract

In addition, the part of the corticobulbar tract that controls the lower face and tongue can be considered part of the lateral system.

Medial System

Ends on motoneurons in the medial part of lamina IX or on interneurons that project to these neurons

⇩

controls axial and girdle muscles as well as most cranial nerve nuclei

⇩

The muscles of the body regulated by the medial system contribute to Posture, Balance & locomotion. Those in the head are involved in such activities as closure of the eyelids, chewing, swallowing & phonation

⇩

includes ventral corticospinal tract, Much of the corticobulbar tract & several pathways descending from brainstem

*tectospinal tract & medial vestibulospinal *Pontine & medullary reticulospinal

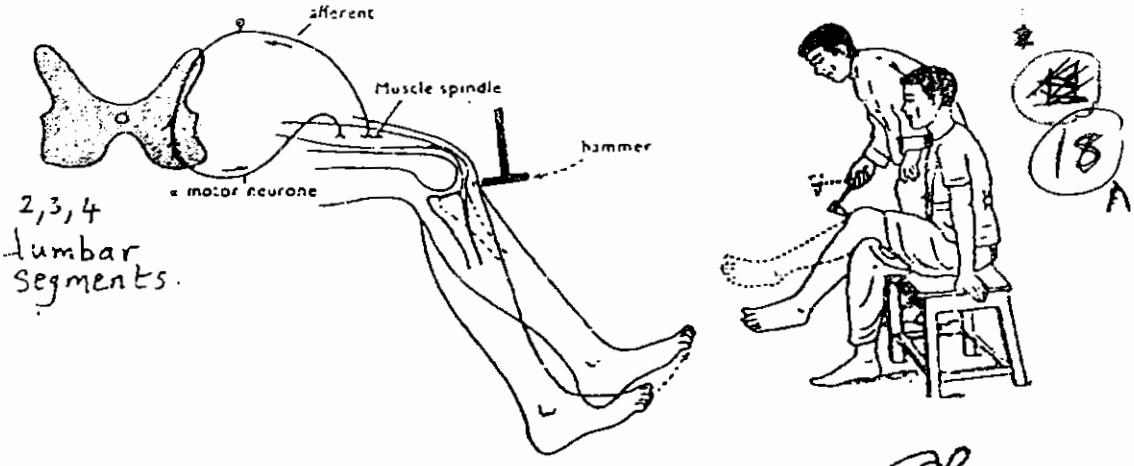


Figure 33 : The knee Jerk.

THE STRETCH REFLEX AND SKELETAL MUSCLE TONE

What is the stretch reflex ? :

When a skeletal muscle is passively stretched, it contracts reflexly. This response of a skeletal muscle to stretch, is known as the stretch reflex.

Nervous pathway of the stretch reflex :

The stretch reflex is the **only monosynaptic reflex** in the body. The **stimulus** that starts this reflex is passive stretching of skeletal muscles. This results in stimulation of specialised **stretch receptors** located in the fleshy part of the muscles known as the **muscle spindles** (see below), which discharge impulses in **afferent fibres.** These are thick myelinated, rapidly conducting (Ia afferent) nerve fibres, which end **directly** (i.e. without intervening interneurons) on large A.H.C. (= the **alpha motor neurons**) that supply the stretched muscle. These neurons constitute the **centre of the reflex,** from which **efferent fibres arise.** These, like afferent fibres, are thick myelinated (about 16 microns in diameter), rapidly conducting (group A alpha) nerve fibres that supply the skeletal muscle resulting in its contraction → (Ia afferent)

THE MUSCLE SPINDLES (intrafusal fibres)

Structure :

These are capsulated fusiform stretch receptors present in the fleshy parts of skeletal muscles ~~parallel to their fibres.~~ Each spindle is few millimeters in length, and is formed of 4-10 small muscle fibres called **intrafusal fibres** which are enclosed in a connective tissue capsule. The spindles are attached either to the tendon of the muscle or to the sides of ordinary muscle fibres, which are called the **extrafusal fibres.** The intrafusal fibres are smaller and less developed than the extrafusal fibres, and each consists of a **central non-contractile part called the receptor area,** and a **peripheral contractile part.**

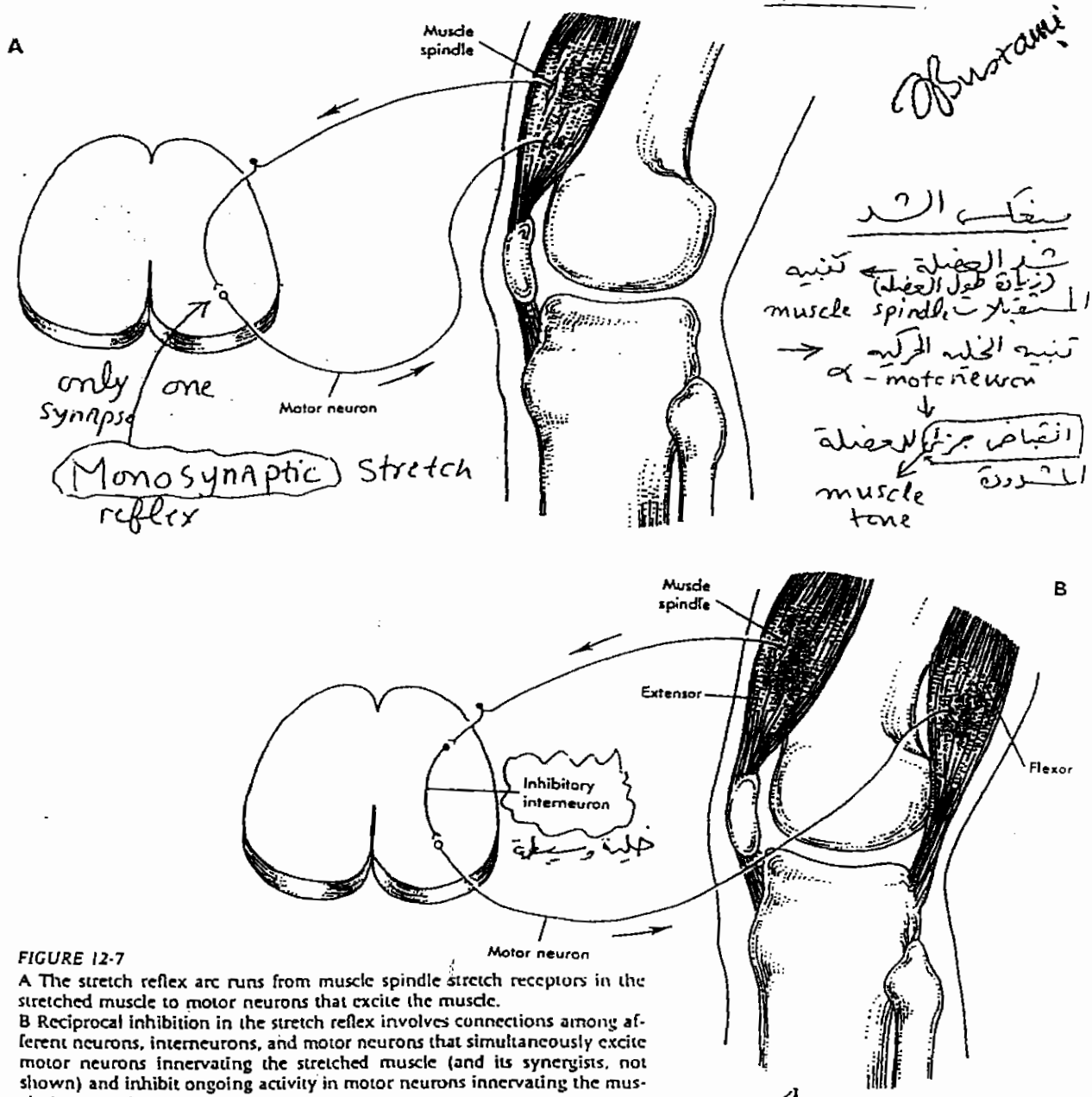


FIGURE 12-7
A The stretch reflex arc runs from muscle spindle stretch receptors in the stretched muscle to motor neurons that excite the muscle.
B Reciprocal inhibition in the stretch reflex involves connections among afferent neurons, interneurons, and motor neurons that simultaneously excite motor neurons innervating the stretched muscle (and its synergists, not shown) and inhibit ongoing activity in motor neurons innervating the muscles' antagonists.

monosynaptic reflex يؤدي الى انقباض العضلي
(disynaptic reflex) وإرتخاء العضلة المضادة

There are 2 types of intrafusal muscle fibres : (19)

1. **Nuclear bag fibres :** These have many nuclei, which are grouped together forming a dilated bag in the central part of the receptor area.

2. **Nuclear chain fibres :** These have a smaller number of nuclei, forming a chain throughout the receptor area. These fibres are thinner and shorter than the nuclear bag fibres, and their ends are connected to the sides of these fibres.

Innervation :

(1) Afferent fibres :

These arise from 2 types of sensory nerve endings in the muscle spindles, which are stimulated by stretch of the central receptor area :

a) **Primary or annulospiral endings :** These encircle the central parts of the receptor areas of both the nuclear bag and nuclear chain

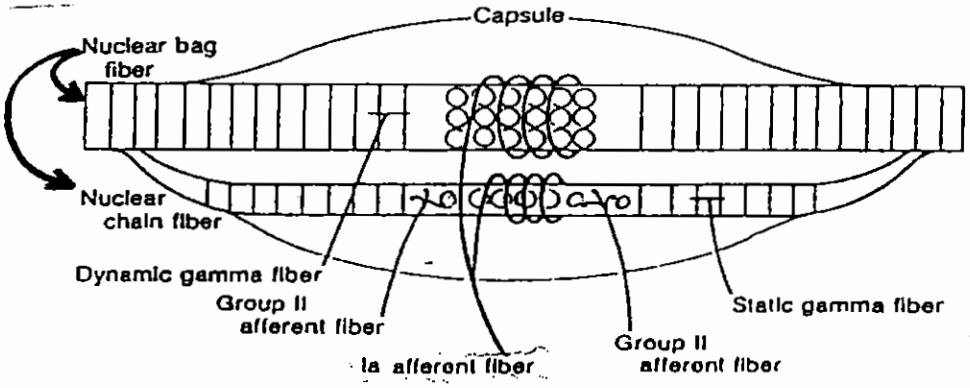


Figure 1-52. Diagram of an intrafusal muscle fiber, showing its nuclear bag and nuclear chain fibers. The afferent innervation (Ia and II fibers) and efferent innervation (gamma dynamic and gamma static fibers) of the intrafusal muscle fiber also are illustrated.

intrafusal fibres, and give rise to thick (about 16 microns in diameter) myelinated group A (rapidly-conducting) afferent fibres. (Ia)

b) **Secondary or flower spray endings :** These lie on both sides of the primary endings and encircle the peripheral parts of the receptor areas of **only the nuclear chain fibres.** They give rise to thinner (about 8 microns in diameter) myelinated group B (less rapidly conducting) afferent fibres. (II)

(2) Efferent fibres : THE GAMMA EFFERENT FIBRES :

The peripheral contractile parts of the intrafusal fibres of the muscle spindles are supplied by thin motor nerves about 4 microns in diameter called gamma efferent fibres. These nerves are the axons of small A.H.C. called the **gamma motor** neurons, and constitute about 30% of the efferent nerves that leave the spinal cord in the ventral roots.

There are 2 types of these gamma efferent fibres :

a) **Dynamic fibres (gamma-d fibres)** : These supply the nuclear bag intrafusal fibres.

b) **Static fibres (gamma-s fibres)** : These supply the nuclear chain intrafusal fibres.

When the gamma efferent fibres are stimulated, the peripheral parts of the intrafusal fibres contract, leading to stretch of the central

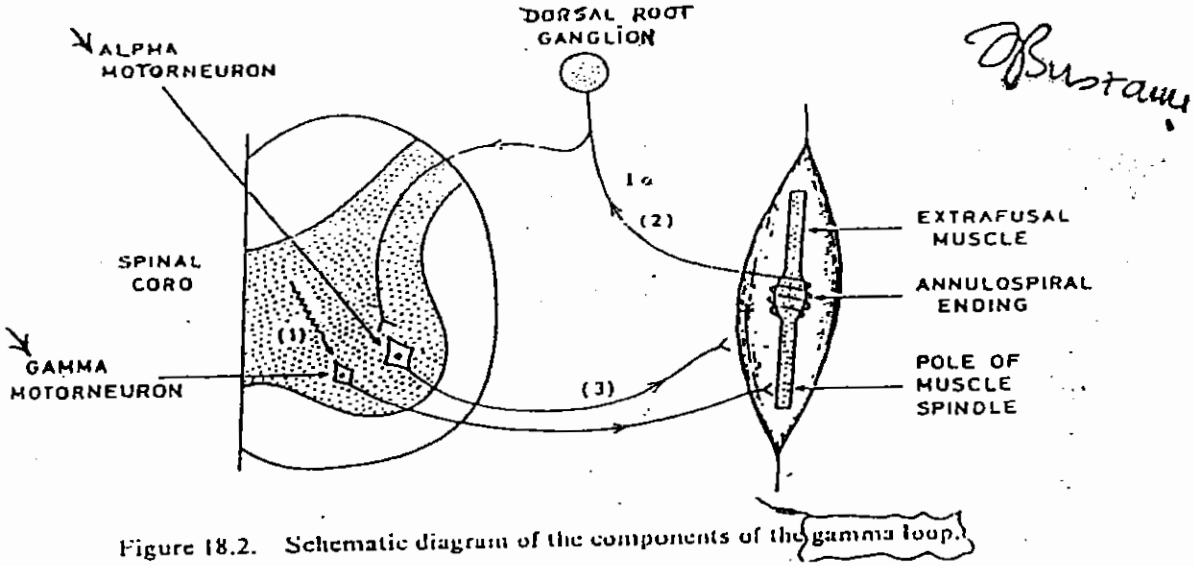
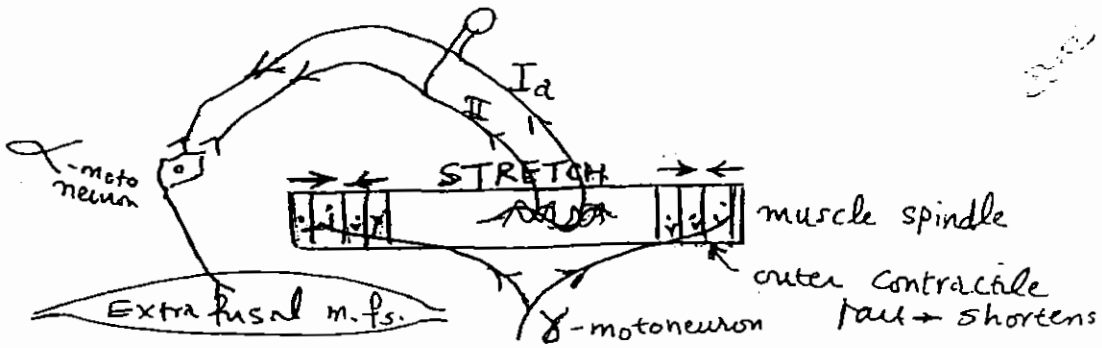


Figure 18.2. Schematic diagram of the components of the gamma loop.



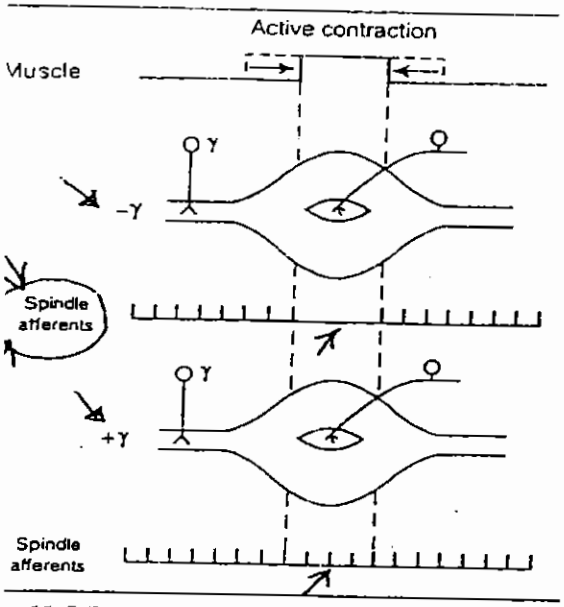
receptor area, thus the primary and secondary endings are stimulated and discharge impulses in their afferent nerves, which produce reflex contraction of the extrafusal muscle fibres.

Methods of stimulating the muscle spindles :

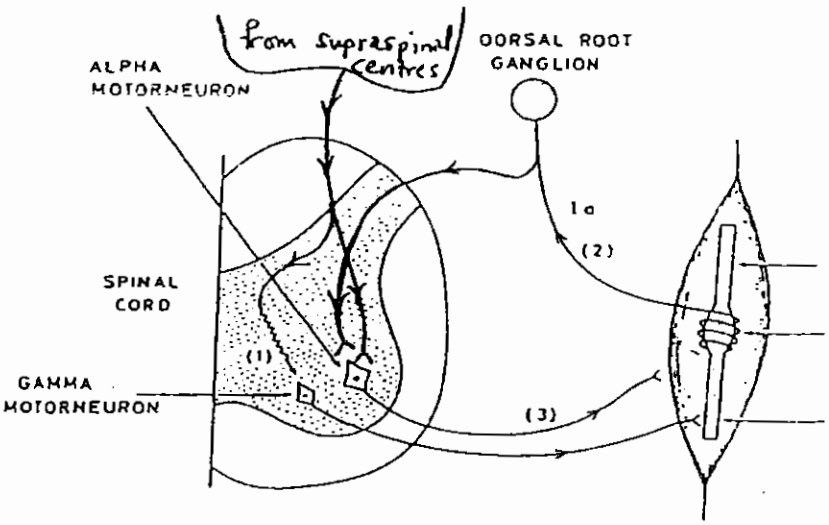
1) Stretching of the whole muscle.

2) Stimulation of the gamma efferent fibres (as described above).

The muscle spindles are **silent** i.e. not stimulated during active contraction of the muscles (which releases the stretch of the spindles), provided they are not stretched by gamma efferent fibre activity. On the other hand, they are **maximally stimulated** when the muscle is stretched and the intrafusal muscle fibres are contracted through stimulation of the gamma efferent fibres.



g. 11-5. Response of muscle afferents to muscle con-
ction without and with activation of gamma motor neu-
ns. During active contraction, discharge in afferent
ers ceases in the absence of gamma activation because
: spindle becomes unloaded as extrafusal muscles
orten. Activation of gamma motor neurons prevents un-
ding of the spindle, and the discharge in the afferents
m the spindle is maintained.



The alpha motor neurons that innervate the extrafusal muscle fibres are stimulated by 2 ways :

1. Directly by descending impulses from supraspinal centres
2. Indirectly (reflexly) by afferent impulses discharged from the muscle spindles along (Ia) & (II) fibres

Alpha-gamma linkage (coactivation of alpha and gamma motor neurons) :

It seems that during active muscle contraction, the muscle spindles are not completely silent. There is evidence that impulses from supraspinal centres stimulate both the alpha and gamma motor neurons, leading to contraction of both the extrafusal and intrafusal muscle fibres at the same time. This has been called the **alpha-gamma linkage**, through which the muscle spindles continue discharging throughout contraction, thus remaining capable of reflexly adjusting the alpha motor neuron discharge, in spite of the change that occurs in the length of the muscle (due to contraction).

Oversensitivity of the gamma system may lead to hypertonia

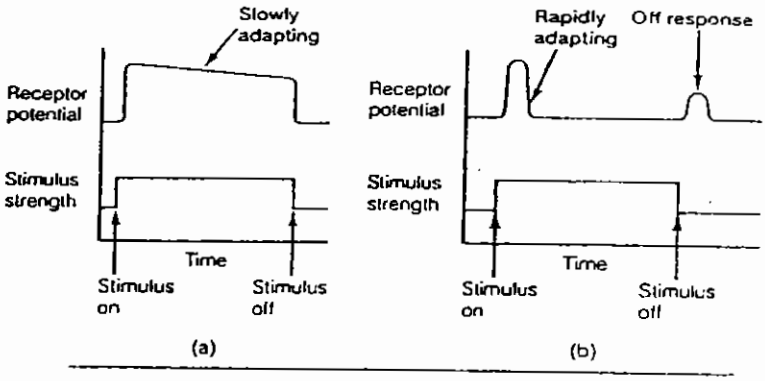
Activation of gamma neurons alone can produce a reflex contraction of the muscle. Since gamma motor neurons are smaller than alpha motor neurons, they have a lower threshold for excitability than the alpha motor neurons. are more easily excited, and have higher tonic discharge rates. Therefore, tonic discharge of the gamma motor neurons may be responsible in large part for maintenance of muscle tone. Overactivity of the gamma system may lead to hypertonia.

Function of the muscle spindles :

The muscle spindles and their reflex connections constitute a feedback mechanism which maintains the length of muscles constant e.g. if a muscle is stretched, its spindles discharge leading to reflex contraction, so the muscle will be shortened. On the other hand, if the muscle is shortened, the discharge from its spindles decreases helping its relaxation, so the muscle will be lengthened.

Response of muscle spindles to stretch :

When the muscle spindles are stretched, both the primary (annulospiral) and secondary (flower spray) endings will be stimulated, but the pattern of response of each is different as follows :



• FIGURE 6–4 Tonic and Phasic Receptors (a) Tonic receptor. This receptor type does not adapt at all or adapts slowly to a sustained stimulus and thus provides continuous information about the stimulus. (b) Phasic receptor. This receptor type adapts rapidly to a sustained stimulus and frequently exhibits an off response when the stimulus is removed. Thus, the receptor signals changes in stimulus intensity rather than relaying status quo information.

1) Dynamic response of the primary endings :

The primary endings are rapidly-adapting receptors, so when the muscle spindle is stretched, the rate of discharge of impulses from these receptors initially increases, but it rapidly declines to the original level when the stretching force is maintained and the length of the muscle stops to increase.

Since the primary endings (Ia) are stimulated only during the stretching movement (i.e. during actual increase in the length of the muscle), their response has been called the **dynamic response,** which informs the nervous system about the rate of change in the length of the stretched muscle.

This dynamic response is the result of stretching of the **nuclear bag** intrafusal fibres, from which the primary endings arise. Therefore, this response can be increased by stimulating the gmma-d (dynamic) fibres, which supply the nuclear bag fibres.

2) Static response of the secondary endings :

The secondary endings are slowly-adapting (= tonic) receptors, so when the muscle spindle is stretched, the number of impulses discharged from these endings increases in proportion to the degree of stretch. But when the stretching force is maintained, these receptors (unlike

the primary endings) continue to discharge at a fast rate for a long period of time, as long as the muscle stretch is maintained. Therefore, this response has been called the **static response,** which continuously informs the nervous system about the length of the stretched muscle.

This static response is the result of stretching of the **nuclear chain intrafusal** fibres, from which the secondary endings arise. Therefore, this response can be increased by stimulating the gamma-s (static) fibres, which supply the nuclear chain fibres (figure 30).

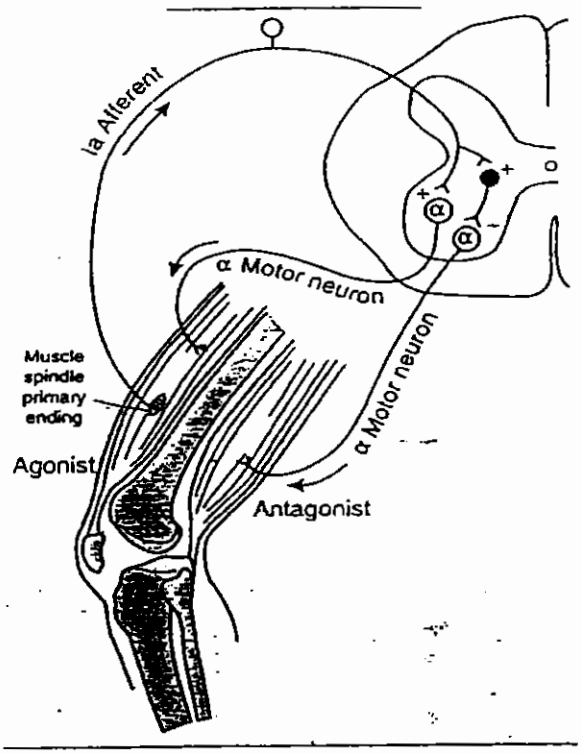


Fig. 11-4. The elements of the monosynaptic stretch reflex, including reciprocal inhibition.

TYPES OF STRETCH REFLEX :

Depending on the dynamic and static responses of the muscle spindles to stretch (see above), the stretch reflex **has dynamic and static components,** thus it can be divided into the following 2 types :

(1) Dynamic stretch reflex :

This occurs when a muscle is **suddenly stretched.** This increases the discharge **from the primary endings** which leads to reflex contraction of the stretched muscle. However, such discharge of impulses rapidly declines (due to adaptation) leading to rapid relaxation of the muscle. Therefore, the dynamic stretch reflex leads to both rapid contraction and rapid relaxation of the muscle, and this is the basis of the **tendon jerks** (see later).

(2) Static stretch reflex :

This occurs on **maintained stretch** of the muscle (during which the dynamic response disappears). This increases the discharge from the **secondary endings** which leads to reflex contraction of the stretched muscle. Such contraction continues as long as the muscle is stretched (due to slow adaptation of the secondary endings). Therefore, the static stretch reflex leads to continuous muscle contraction, as long as its stretch is maintained, and this is the basis of the **skeletal muscle tone** (see below).

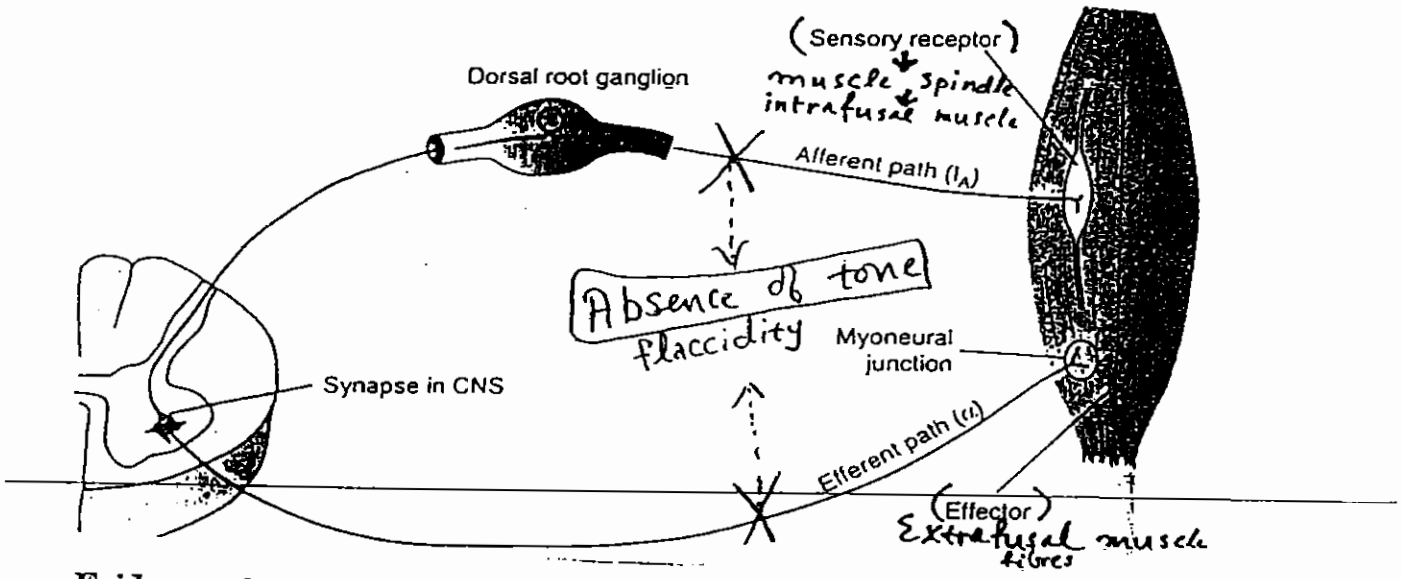
SKELETAL MUSCLE TONE :

Definition : It is a continuous reflex sub-tetanic (i.e. partial) contraction of skeletal muscles during rest. It is produced through the stretch reflex (as described below), so it is a neurogenic property.

Plain muscles also have tone, but in this case, it is due to a myogenic property i.e. it is produced as a result of inherent properties in the plain muscles themselves, and not as a result of nervous reflexes.

Mechanism :

During rest, the skeletal muscles are usually **shorter** than the distance between their origin and insertion, so they are continuously subjected to stretch. This stimulates the muscle spindles which send impulses, mostly from the **secondary endings** (see above), resulting in reflex partial contraction of these muscles. Since during rest this partial contraction is a continuous process, it has been called the **muscle tone**.



Evidence of the reflex nature of muscle tone :

Cutting the afferent or efferent nerves of a certain muscle, leads to **atonia** (= loss of tone) in this muscle, which will accordingly become **flaccid** i.e. completely relaxed.

Distribution of muscle tone :

Tone is present in all skeletal muscles of the body, but it is more marked in the **antigravity muscles,** because they are the most stretched muscles in the body, by the effect of gravity. These muscles are the extensors of the lower limbs, flexors of the upper limb, extensors of the back and neck, elevators of the lower jaw (the mandible), and the anterior abdominal wall muscles.

Functions of the muscle tone :

1. It maintains the erect (standing) posture against the force of gravity.
2. It helps both venous return and lymph flow from the lower parts of the body against the effect of gravity. This effect is known as the **muscle pump** (refer to circulation).

Inverse Myotatic Reflex (Fig. 18.3)

Severe tension in a muscle produced by stretch or contraction will stimulate nerve endings in its tendon (Golgi tendon organ). Impulses from Golgi tendon organs travel via Ib nerve fibers. In the spinal cord, they project upon inhibitory neurons, which in turn will inhibit alpha motor neurons supplying the muscle under tension (homonymous motor

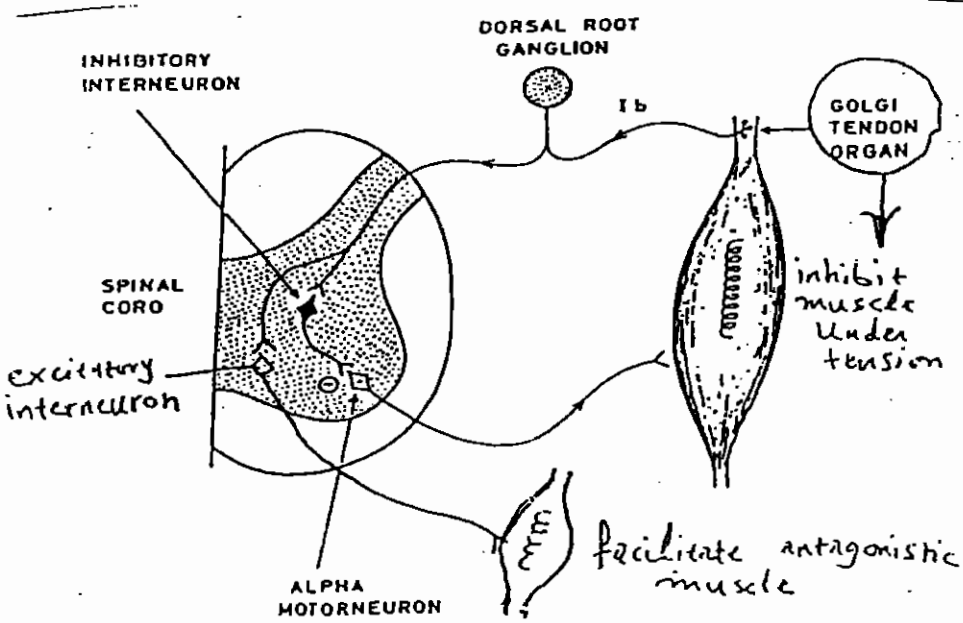


Figure 18.3. Schematic diagram of the components of the inverse myotatic reflex.

neurons). The result is relaxation of the muscle (lengthening reaction, autogenic inhibition). At the same time, the Ib activity will facilitate motor neurons that supply the antagonistic muscle. This is a protective mechanism to prevent tearing of the muscle under great tension. This reflex also underlies the mechanism of the "clasp knife" phenomenon noted in spastic muscles. In such situations, passive stretching of the spastic muscle will be met with great resistance up to a point, after which the muscle gives way suddenly. The phenomenon has been termed "clasp knife" by Sherrington because of its similarity to the action of a jack-knife.

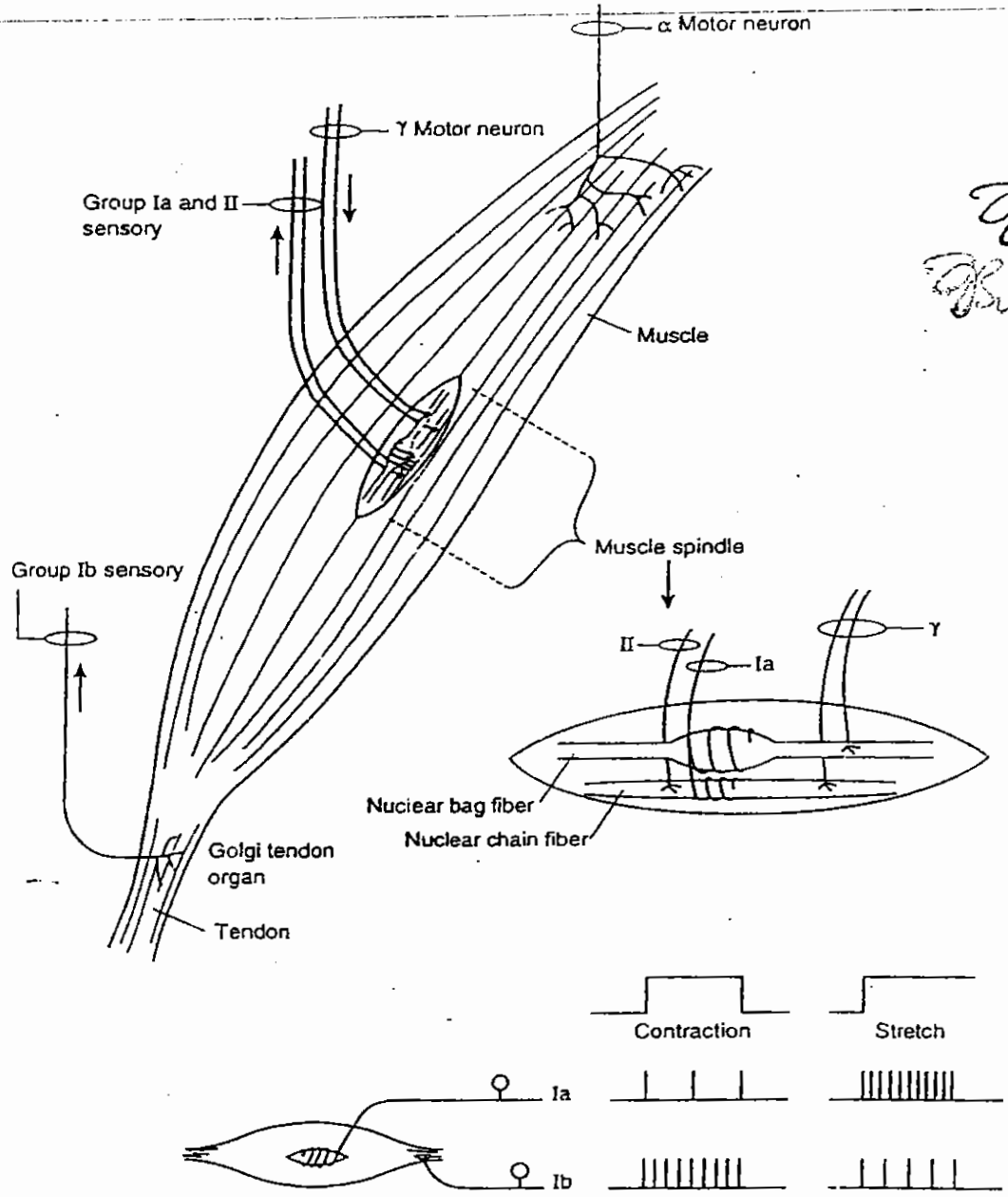
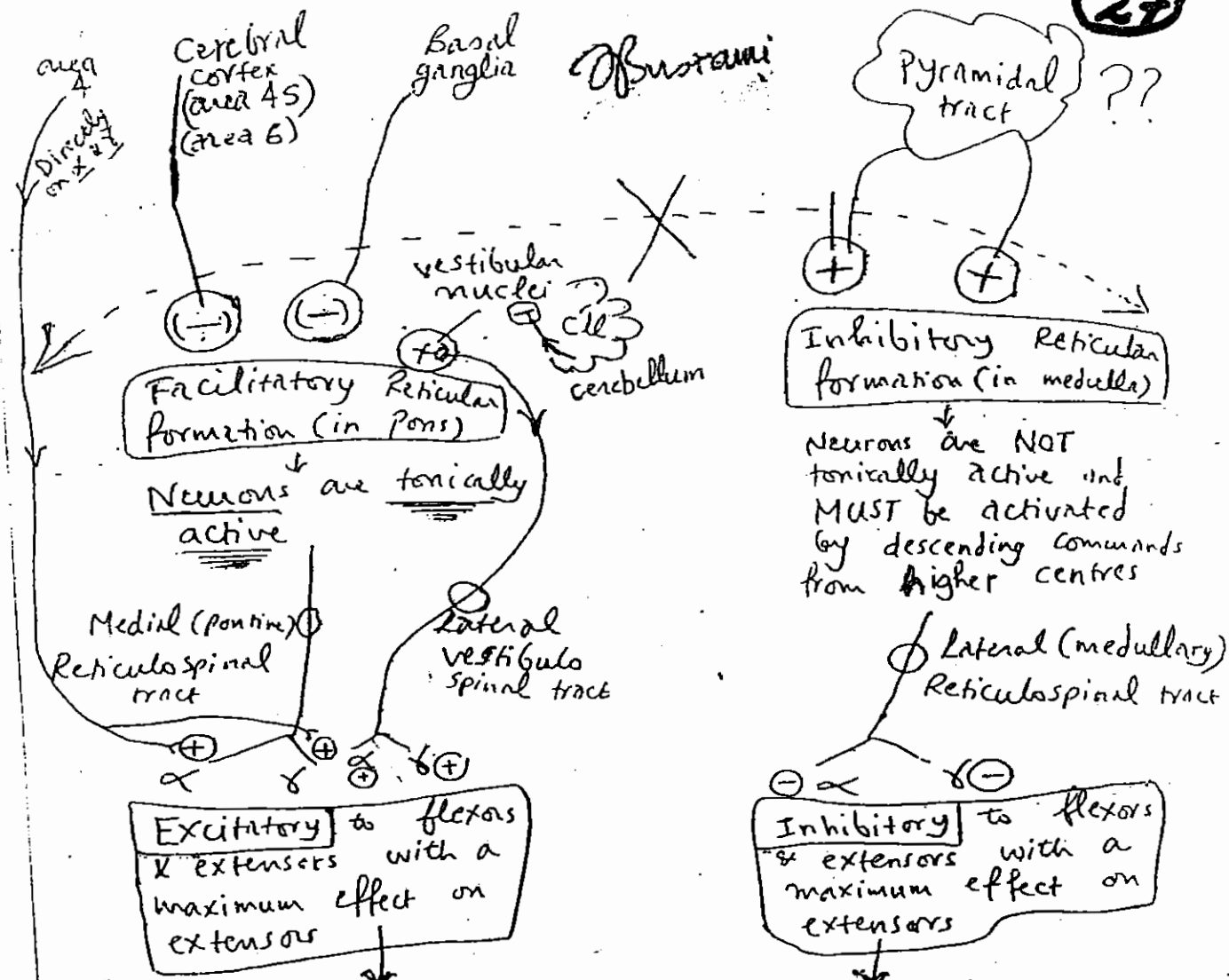


Fig. 11-3. Localization of muscle spindles and Golgi tendon organs in relation to extrafusal muscles. Enlarged view of the muscle spindle illustrates the two types of intrafusal muscle fibers and their innervation. Responses of muscle spindle and Golgi tendon organ to muscle stretch and contraction are illustrated at the lower right part of the figure.

Make use of this diagram to explain one of the upper motor neuron lesion signs → CLONUS ?



In the normal state there is a balance between the facilitatory and inhibitory descending influences ensuring a proper activation of stretch reflex → proper activation of antigravity muscles and regulation of postural tone

Experimental decerebrate rigidity is produced by transection of the brainstem above vestibular nuclei at the boundary between midbrain and Pons

Release of brainstem mechanisms from control by higher centres → excessive contraction of antigravity muscles (spasticity)

Is it alpha or gamma rigidity (spasticity) ??

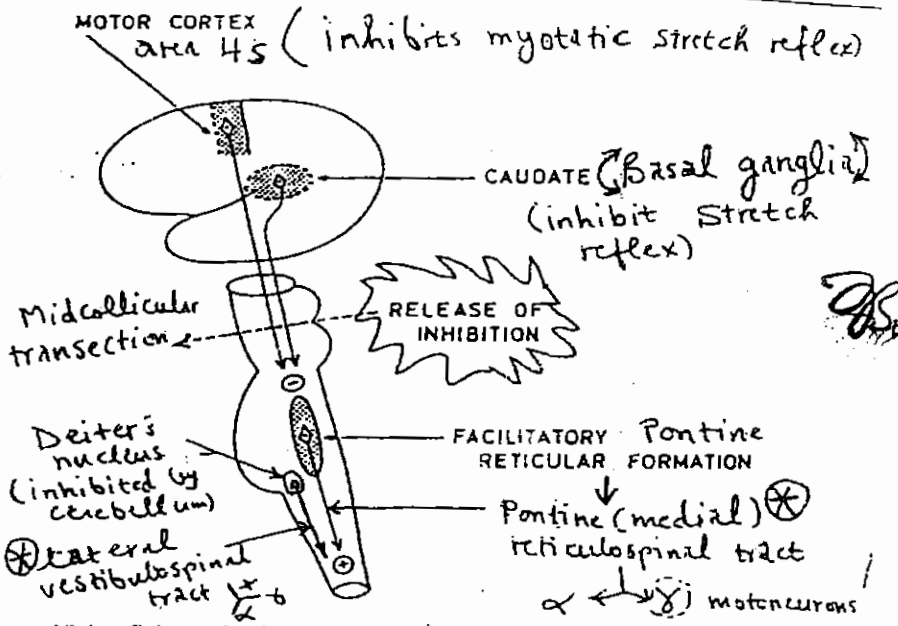


Figure 18.6. Schematic diagram showing the mechanism of decerebrate rigidity.

In the normal state there is a balance between facilitatory (medial reticulospinal tract & lateral vestibulospinal tract) and inhibitory (lateral or medullary reticulospinal tract) descending tracts ⇒ Proper activation of γ-motoneuron ⇒ Proper activation of antigravity muscles and regulation of Postural tone by the myotatic reflex

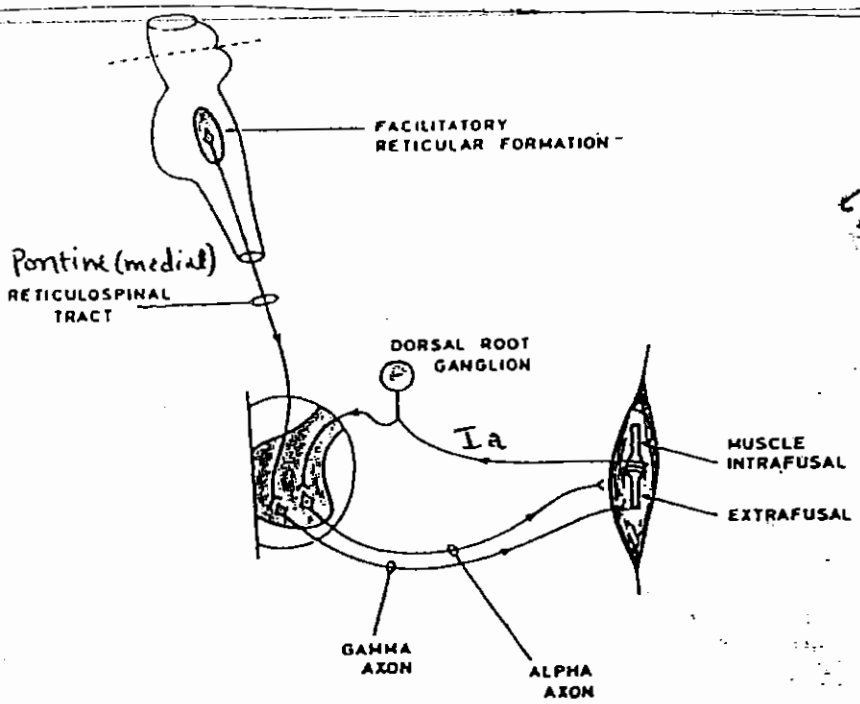
↓

The release of brainstem mechanisms from control by the higher centres results in increased γ efferent discharge → hyperactive stretch reflex → excessive contraction of antigravity muscles producing various forms of extensor rigidity & Spasticity, a condition that can be experimentally demonstrated in the decerebrate animal

B. Decerebrate Rigidity (Mid-Collicular Transection)

Two brainstem centers that are very important to the maintenance of muscle tone in antigravity muscles (primarily extensors) are the pontine reticular formation (medial reticulospinal tract), and Deiter's nucleus (lateral vestibulospinal tract). Both centers have an excitatory influence on extensors. Stimulation of cells in the pontine reticular formation has a very powerful excitatory effect on extensors, but its activity is normally modulated *(inhibited) by central (cortical) projections. If the Brain Stem is cut above the level of the pontine reticular formation (mid collicular), the inhibitory influence is removed and there is an exaggerated activation of muscle tone in extensors (antigravity muscles). This produces a rigid posture which is referred to as decerebrate rigidity. In humans arms and legs are extended, back is arched, head dorsiflexed, and feet ventroflexed (curling of toes lifts against gravity). This stiff posture does not permit joints to bend and the body is capable of standing upright. This is very different from spinal transection, where extensor muscle tone is abolished and the body becomes limp.

DBustami

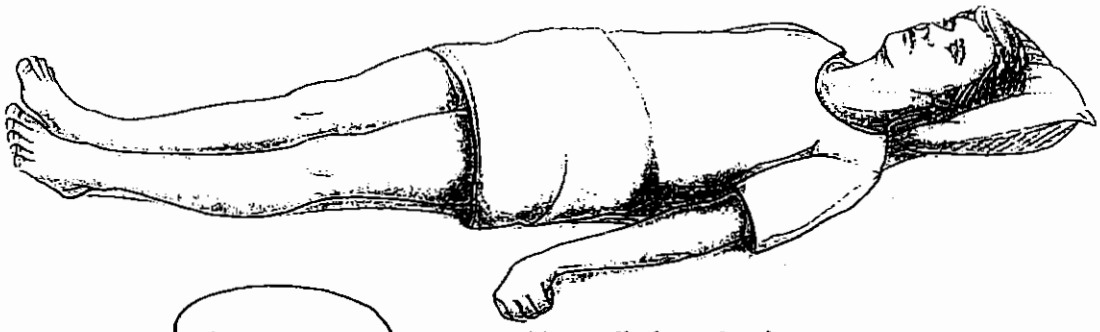


1. Gamma rigidity

Cutting the dorsal roots abolishes decerebrate rigidity. Cutting the dorsal roots interrupts Ia spindle afferents that act to excite homonymous motoneurons via the myotatic stretch reflex. Since Ia afferents signal spindle activity, this demonstrates that the decerebrate rigidity was primarily due to the hypersensitivity of muscle spindles resulting from descending excitation of gamma motoneurons. Removal of the Ia spindle afferents abolishes the rigidity. Therefore, decerebrate rigidity is considered primarily a gamma rigidity.

2. Alpha rigidity

A selective increase in alpha motoneuron activity can produce what is referred to as alpha rigidity. This can be demonstrated after reversing decerebrate rigidity caused by gamma excitability (cutting the dorsal roots) and increasing the excitation of alpha motoneurons. Since cells in the lateral vestibular nucleus (Deiters' nucleus) are normally inhibited by projections from the cerebellum, removal of cerebellar projections increases the activity of these cells. The result is an increase in descending excitation of extensors and rigidity is restored by alpha motoneurons (gammas may fire too, but they are ineffective since the dorsal roots have been cut).



A. Decerebrate; upper and lower limbs extend

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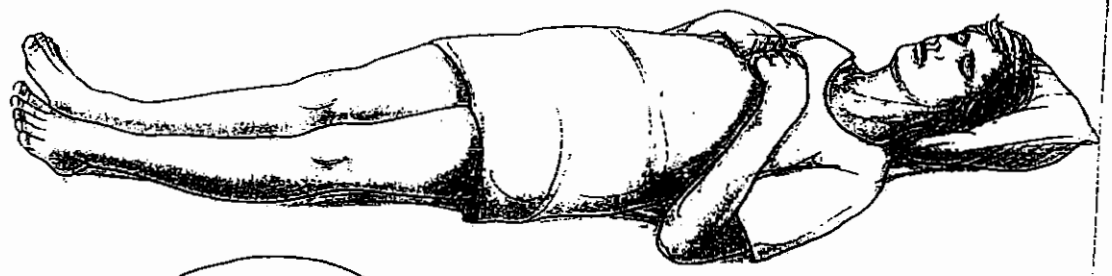
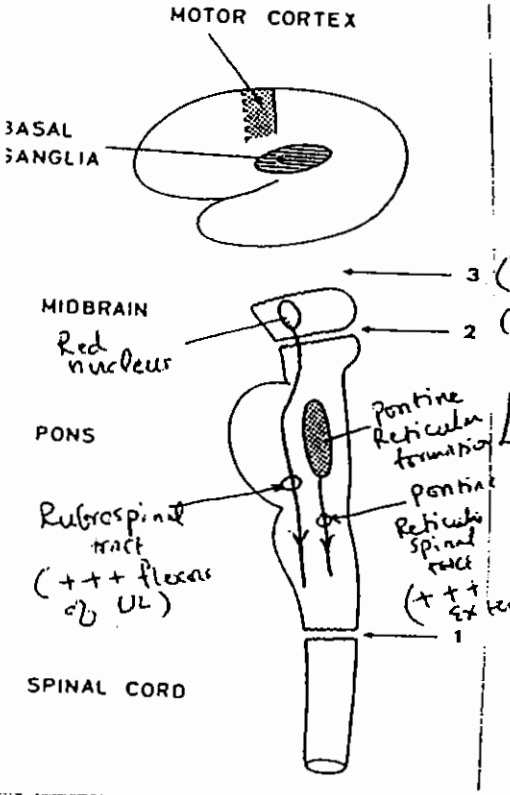


B. Decorticate upper limbs flex, lower limbs extend

FIGURE 7–4. Abnormal posturing in comatose state. A. Decerebrate (upper and lower limbs extend). B. Decorticate (upper limbs flex, lower limbs extend).



A. Decerebrate & B. Decorticate → Both are in comatose state → what happens if both receive a painful stimulus ?

↓

In Decerebrate patient → Extension of both UL & LL

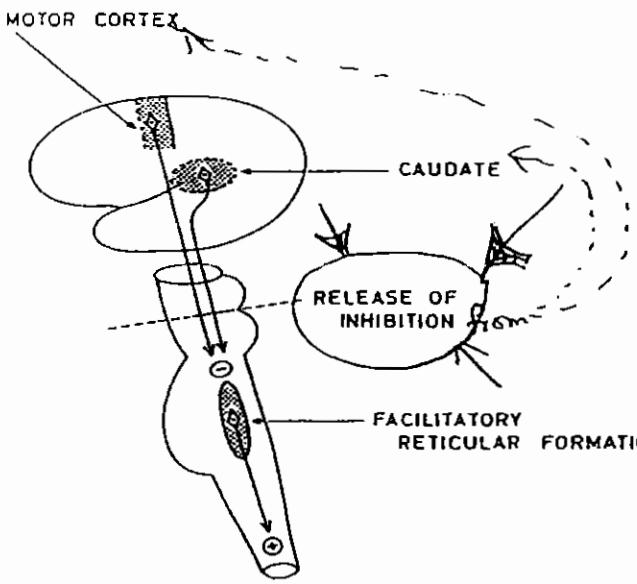
In Decorticate patient → lower limb extend but the upper limbs flex

[1] Comment ? ? ? ?

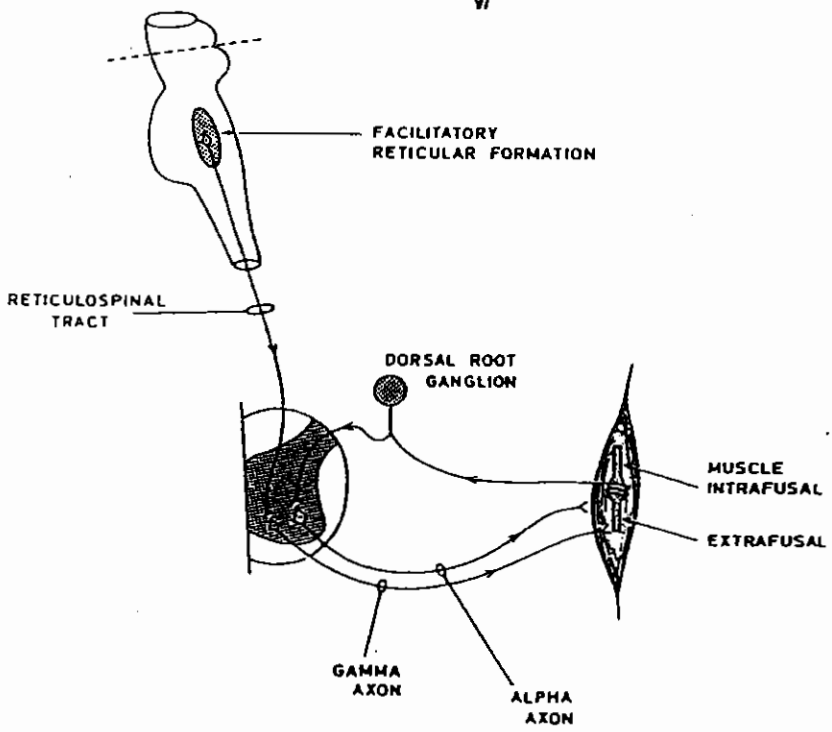
[2] Decorticate posture indicates a higher or more rostral level of brainstem impairment than decerebrate posture ✓

In a comatose patient whose condition alters from decerebrate to decorticate posture → the prognosis is (better / worse) ? ?

Comment !!



Remember : →
the antigravity muscles in the cat are the forelimbs & hindlimbs
↓
In human they are flexors of UL & Extensors of LL



Mechanism

Figure 18.7. Schematic diagram showing the mechanism of decerebrate rigidity.

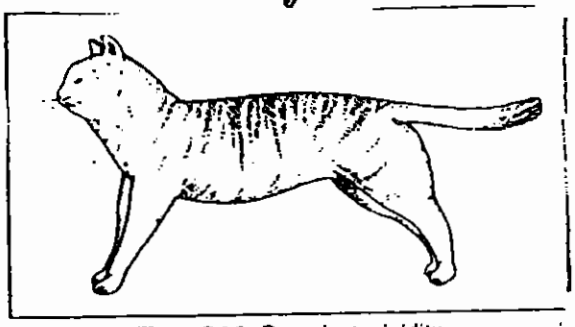
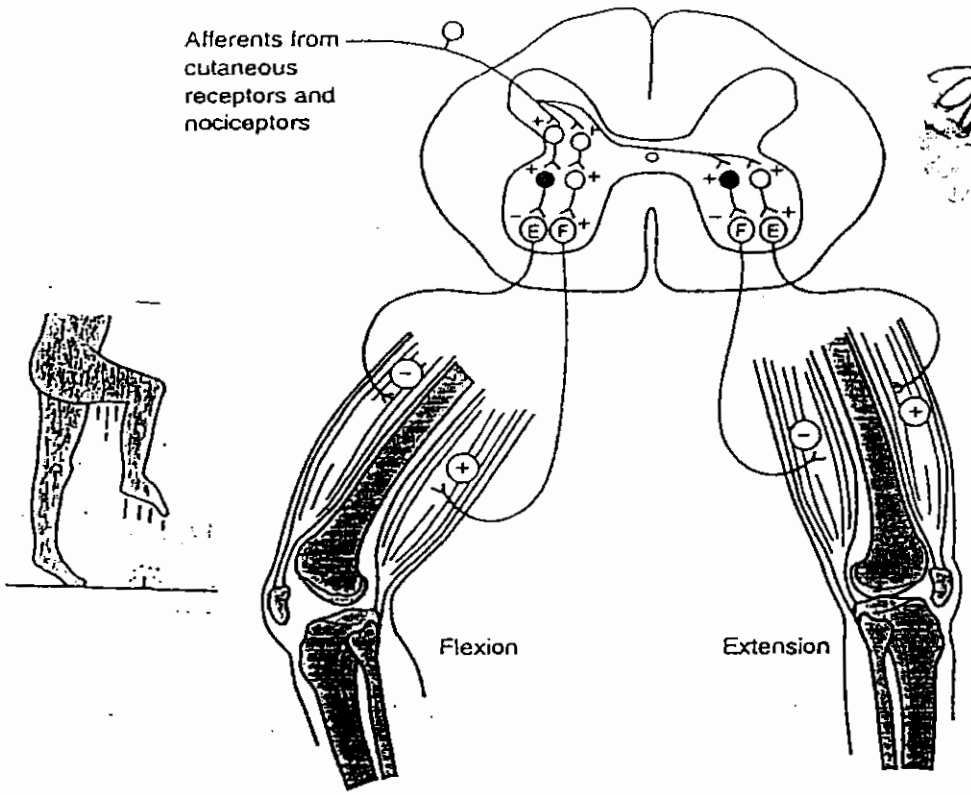


Fig. 10C.2.5. Decerebrate rigidity

Fig. 11-7. Schematic of the organization of the flexion–crossed extension reflex.



Flexion Reflex

Flexion reflexes are important in a number of behavioral patterns; e.g., flexion of limbs is part of the activity involved in walking. One of the most obvious functions of the flexion reflex is withdrawal of a limb from painful, noxious stimuli. Hence the flexion reflex is frequently called the **withdrawal reflex.** Also, since flexion of the limb ipsilateral to the stimulus is usually accompanied by an extension of the contralateral limb(s), this reflex is also referred to as the **flexion-crossed extension reflex.**

The flexion reflex is polysynaptic (Fig. 11-7). The afferent fibers enter the spinal cord and excite interneurons of the dorsal horn. The interneurons then act on alpha motor neurons through relay pathways involving other interneurons. The response is an excitation of alpha motor neurons to the flexor muscles and inhibition of alpha motor neurons to the extensor muscle of the stimulated limb (ipsilateral). In addition, this is frequently accompanied by excitation of alpha motor neurons to extensor muscles and inhibition of flexors to the contralateral muscle. This behavior is the appropriate response to painful stimuli; for example, if a person steps on a sharp object, the injured foot is withdrawn (flexion), while the other limb of the pair is extended, thereby providing support for the body and preventing the person from toppling.

The flexion reflex can be initiated by activity in afferent fibers from a variety of sensory receptor organs. These sensory receptors may be in the skin, in muscle, and in joints and involve afferent fibers II, III, and IV; collectively, these are called **flexor reflex afferents** (FRA). The degree of flexion response can vary from a flexor twitch in response to relatively innocuous stimulation to a complete withdrawal of the limb from a noxious stimulus. A very strong stimulus to the FRA fibers results in activity of all four limbs. This response is mediated via intersegmental connections and is sometimes referred to as **irradiation of the stimulus;** the stronger the stimulus, the more extensive is the reflex reaction.

Bustami Bustami

Sequence of events in a voluntary movement (Knee flexion)

↓

(1) Activation of Ia interneurons → inhibits ANTAgonist α MN

↓

(2) Activation of Agonist α MN & γ MN

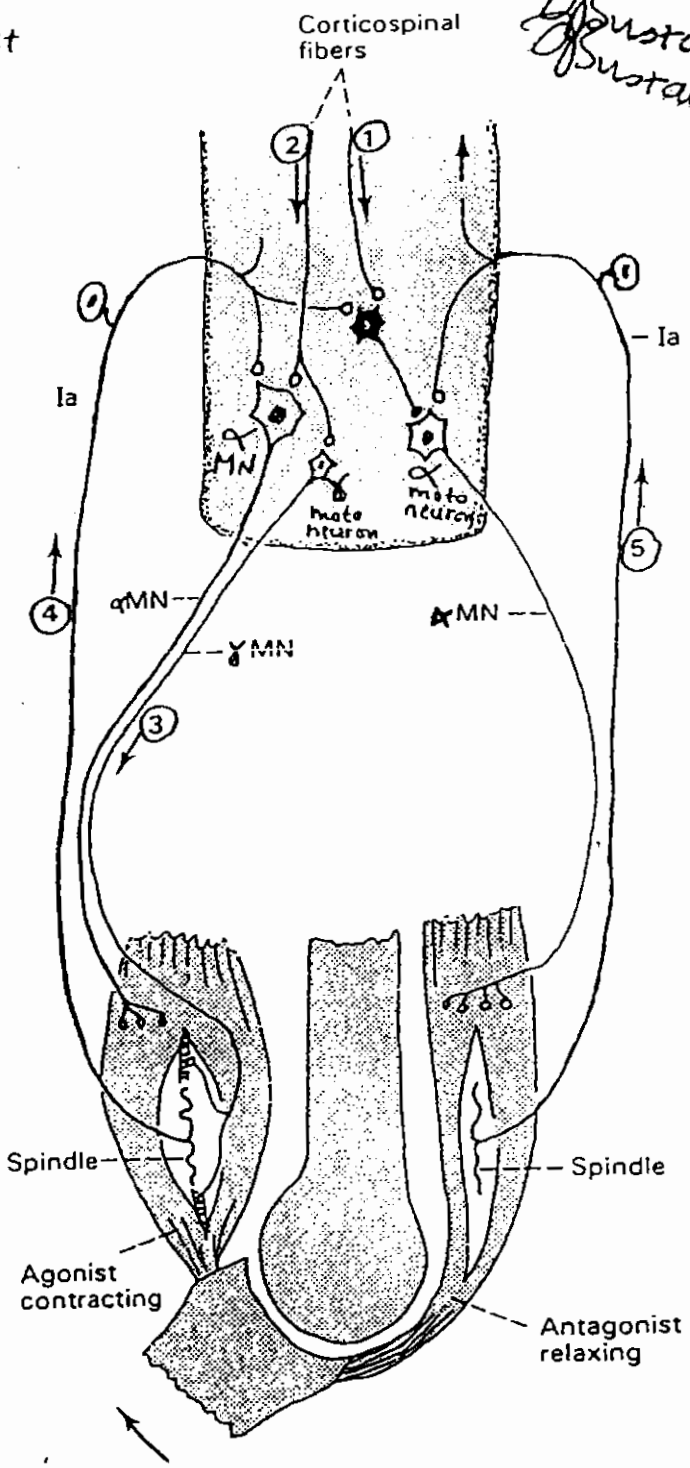
↓

(3) Contraction of extra- and intrafusal muscle fibres

↓

(4) (feedback) from contracting spindle increases α MN excitation and Ia inhibition

(5) Antagonist Ia fibre finds its homonymous α MN Refractory but it transmits to higher centres (arrow)



Remember (1) (γMN), (Ia) and (αMN) constitute the (gamma loop)

(2) In voluntary movements αMNs and γMNs are recruited (stimulated) together → This is known as α-γ coactivation or linkage

(3) (The γMNs REINFORCE α-excitation through what is known as the gamma loop)

IN THE EXECUTION OF VOLUNTARY MOTOR ACTIVITY → THE DESCENDING INFLUENCES FROM THE CORTEX and SUBCORTICAL STRUCTURES VIA THE TWO PATHWAYS (PYRAMIDAL, EXTRAPYRAMIDAL) ⇒ MOST LIKELY ACT SIMULTANEOUSLY on Alpha and gamma motor neurons of the spinal cord. Alpha activation, however, predominates in the case of rigid movements whereas gamma activation predominates in connection with slow, graduated movements

↓

* Selective lesions in the two pathways (Pyramidal and extrapyramidal) are difficult to produce and BOTH are usually affected TOGETHER to varying degrees.

↓

Motor deficits Produced by lesions of the motor cortex or its outflow (descending tracts) depends on the extent to which Pyramidal and extrapyramidal tracts are affected

↓

1- Interruption of the corticospinal tract by Unilateral Section of the Pyramid ⇒ Paresis (weakness) ⊕ hypotonia of the distal muscles of the limbs on the contralateral side (These findings are consistent with a loss of the descending facilitatory effects on the spinal α and γ motoneurons)

2- Experimentally produced localized lesions of Cortical Primary motor area (area 4) resemble the effects of Pyramidotomy i.e Distal Paresis (weakness) or paralysis (loss of function) and hypotonia ⟶ when the lesion is increased to include premotor area 6 (in which proximal muscles are represented) ⟶ the loss of control over brainstem centres generates a state of ≤ spasticity that overshadows the hypotonia due to area 4 lesion.

3- Lesions at the level of the internal capsule (a common lesion in cases of Stroke and could be the result of haemorrhage or thrombosis or embolism of the blood vessels of internal capsule) → results in interruption of corticospinal fibres (Pyramidal) as well as Projections to the brainstem (extrapyramidal) →

The Extrapyramidal effects OVERSHADOW the Pyramidal effects → In addition to Paresis or Paralysis of the contralateral half of the body → hemiparesis or hemiplegia ← the predominant Signs of such a lesion are:

a- Spasticity → increased muscle tone particularly affecting antigravity muscles (flexors of upper limb and extensors of lower limb)

b- HYPERREFLEXIA → Exaggerated muscle stretch reflex (MSR) (used to be called deep tendon reflexes or JERKS)

c- Clonus → rhythmic contractions of muscles when they are subjected to Sudden sustained stretch

d- Positive Babinski's sign
e- absent { abdominal reflex / Cremasteric reflex } } Reversal of certain FRA-Driven reflexes

Spasticity, hyperreflexia & clonus → Reflect RELEASE of the brainstem centres from descending controls (i.e brainstem centres are released from inhibition) → imbalance between facilitatory and inhibitory effects on stretch reflex → increased γ efferent discharge → hyperactive Stretch reflex → Hyperreflexia
↘ excessive contraction of antigravity muscles (spasticity)

What are the signs of upper motor neuron lesion (UMNL)? → (Paresis) or (Paralysis) - - - - - - - → distal muscles (hand)
⊕ Signs a, b, c, d, e

Spasticity and Rigidity

Obrotami

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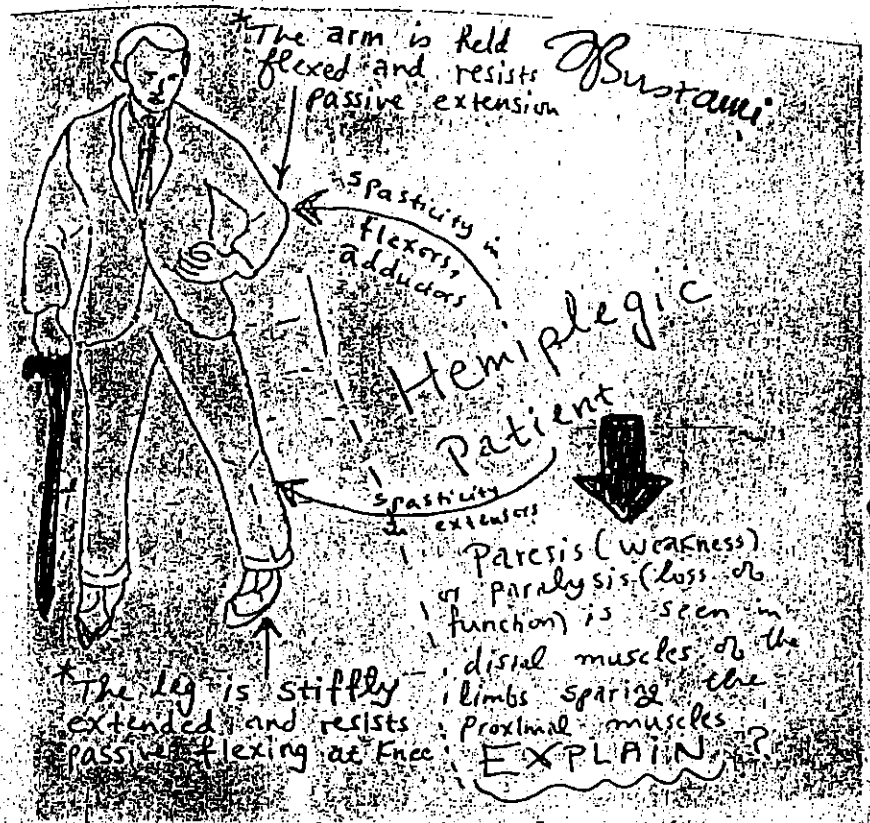
* Both are fundamentally the same phenomenon in that they reflect RELEASE of the brainstem mechanisms from descending controls resulting in IMBALANCE between the facilitatory and inhibitory influences on the spinal motor circuits (esp. stretch reflex)
* How you examine for spasticity? → increased resistance of spastic muscles to PASSIVE movement that is usually greater in antigravity muscles (unidirectional) → a clasp-knife reflex is observed.



Obrotami

This patient has an upper motor neuron lesion (UMNL) known as stroke or cerebro-vascular accident (CVA)

↓

The lesion which is either haemorrhage or thrombotic vessel or embolism affecting this Right internal capsule (So the lesion will affect both pyramidal & extrapyr. tracts) → He suffers Spastic weakness or paralysis of this left upper and lower limbs (Hemiparesis or Hemiplegia)

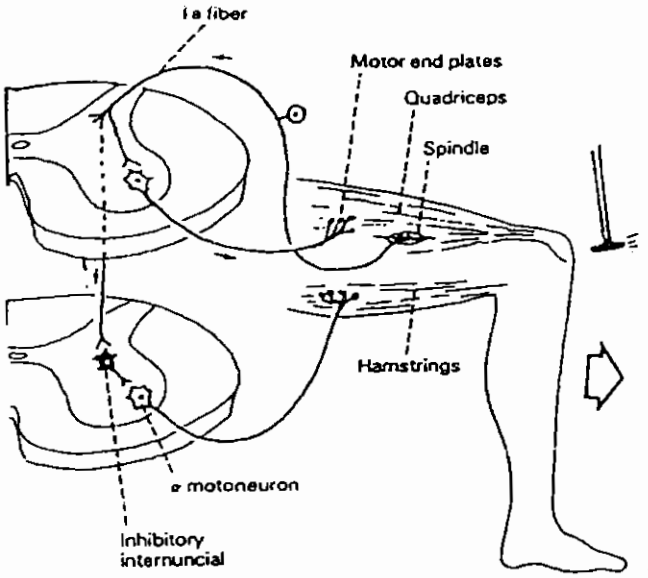
Remember → Spasticity (of upper motor neuron lesion) is accompanied by Hyperreflexia & clonus, Why??

→ the underlying mechanism is the same

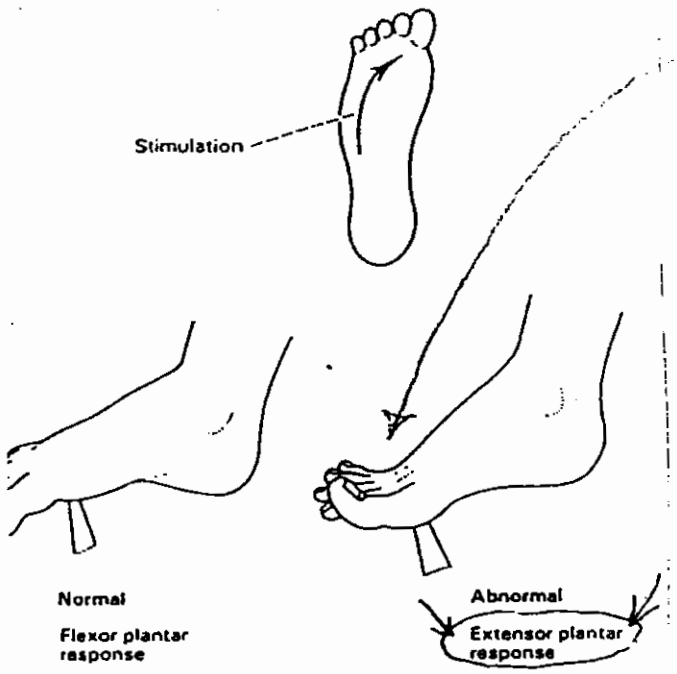
RELEASE of - - - - - - - -

Four Primary Reflexes

Reflex	Roots Needed for Reflex	Muscle Carrying out the Relex
Ankle jerk	S1	Gastrocnemius
Knee jerk	L2, L3, L4	Quadriceps
Biceps	C5, C6	Biceps
Triceps	C7, C8	Triceps



ig. 4-9 The knee jerk.



g. 11-11 Plantar reflex, showing the Babinski sign – an extensor ntar response to a stimulus applied to the sole.

I HYPERREFLEXIA signifies an upper motor neuron lesion along the neuraxis from cortex to lateral columns of the spinal cord.

Exaggerated tendon reflexes. These are seen on the affected side, exemplified by the knee and ankle jerks, and are due to the release of the stretch reflex from inhibition by higher centres

4 *Clonus.* This is the occurrence of rhythmic contractions of muscles when they are subjected to sudden sustained stretch, e.g. ankle clonus. The precise cause of clonus is not known. This phenomenon is associated with increased gamma efferent discharge, occurring as a result of the release of the stretch reflex from inhibition.

II Positive Babinski's Sign
||
[up-going toes] → Extensor plantar response

the plantar reflex becomes extensor, known as a positive *Babinski's sign* (i.e. scratching the outer aspect of the sole by a blunt object results in dorsiflexion of the big toe and fanning of the other four toes). The abnormal response is thought to be a primitive reflex that reappeares following injury of the pyramidal fibres.

The Babinski's sign is considered physiological during the first year of life, due to immaturity of the pyramidal tract, and in adults during sleep, deep anaesthesia or coma, due to the depressed activity of the motor cortex. *

Remember → If the hyperactive reflexes truely reflect upper motor neuron lesion → the toes should also be abnormal i.e up-going toes → Positive Babinski's Sign

The clinical term rigidity is one of the major signs of Parkinson's disease which affects the functions of the basal ganglia. In contrast to spasticity this type of rigidity has the following characteristics:

(a) increased resistance to passive movement is bidirectional (i.e both flexors and extensors are affected) ✓Bustami

(b) patients with Parkinson's disease do not have a hyperactive muscle stretch reflex (deep tendon reflexes) ✓Bustami

Remember → If the upper motor neuron lesion (UMNL) occurs above the level of motor (pyramidal) decussation i.e in the motor cortex or internal capsule or brainstem → the paresis or paralysis will be CONTRALATERAL to the side of the lesion

However if the lesion is below the level of decussation → paresis or paralysis will be ipsilateral to the side of lesion (i.e within the lateral funiculus of white matter of spinal cord)

	Lower Motor Neuron	Upper Motor Neuron
1	Flaccid weakness or paralysis	Spastic weakness
2	Decreased or absent MSR	Increased MSR with or without clonus
3	Signs of muscle denervation; fasciculations, fibrillations, profound atrophy	No signs of muscle denervation
4	Muscles affected singly or in small groups innervated by a common nerve or spinal root	Muscles affected in large groups, organized by quadrants or halves of the body

The term lower motor neuron (LMN) is used to designate the ventral horn cells of the spinal cord (α & γ MNs) which innervate skeletal muscles of body (+) the motor neurons of the brainstem which innervate muscles (facial muscles, tongue muscles, muscles of pharynx & larynx) supplied by certain cranial nerves

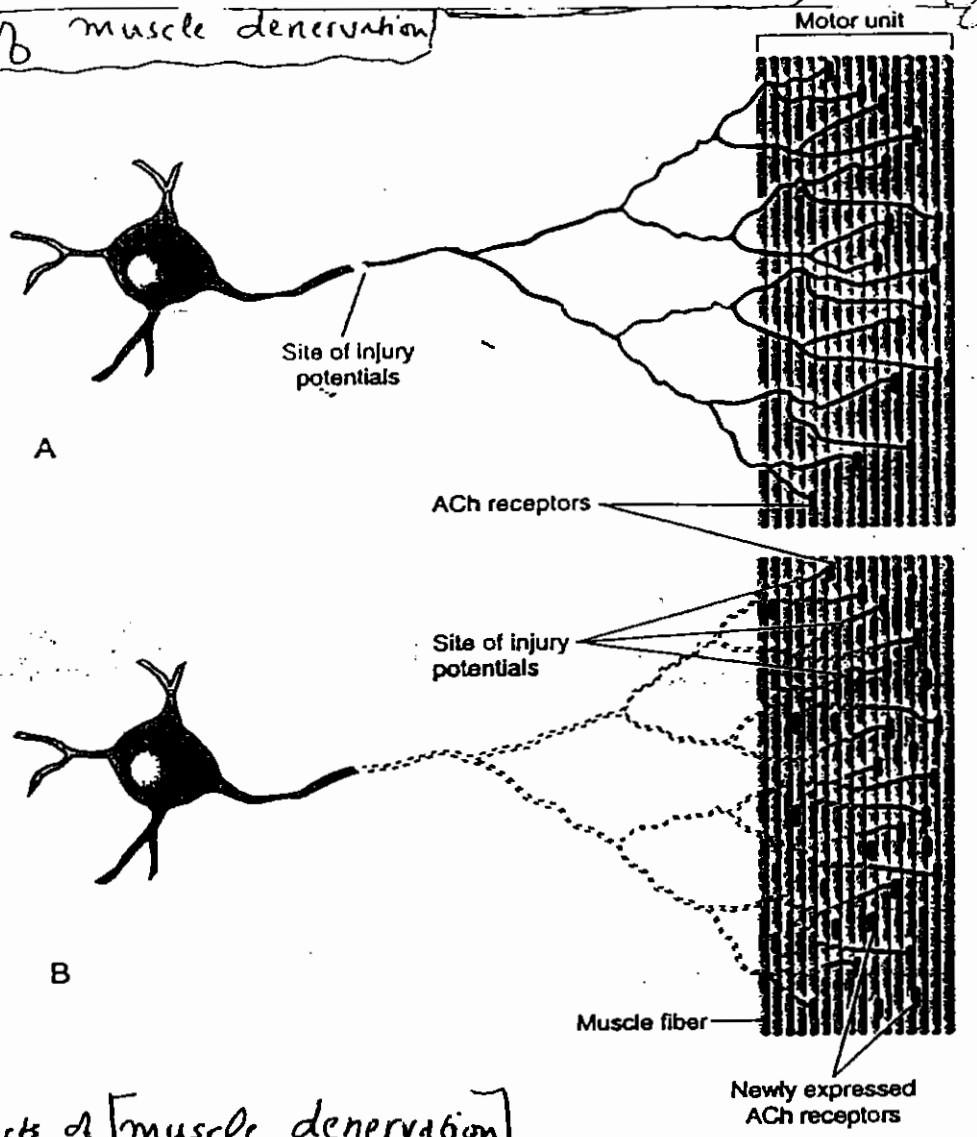
Destruction of lower motor neurons (LMNs) or their axons (e.g by poliomyelitis) → Loss of voluntary & reflex response of muscles supplied by these neurons

Signs of Lower Motor Neuron Lesion (LMNL)

1. Paresis or Paralysis } → Flaccid paralysis
2. Decreased muscle tone (flaccidity) }
3. Decreased or absent muscle stretch reflexes (MSR)
4. Fasciculation → fibrillation (spontaneous activity of muscle fibres at rest)
5. Marked denervation atrophy

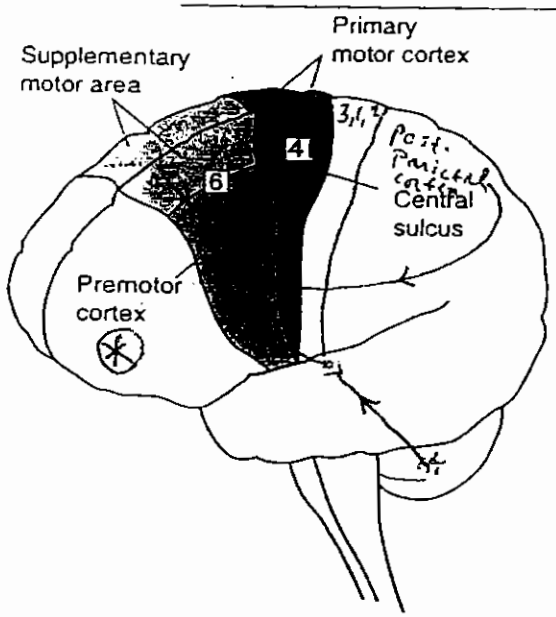
All these signs occur ipsilateral to the spinal cord lesion → in the muscle or muscles supplied by the spinal cord segment involved (38)

N.B Fasciculations are the earliest objective sign of muscle denervation



Effects of [muscle denervation]

Fasciculations and fibrillations are caused by injury potentials generated at the site of injury to a motor neuron axon. **A.** Injury potentials cause all of the elements of the motor unit to contract simultaneously, producing a coordinated twitch (fasciculation) that is visible on the surface of the body. **B.** As the distal axon degenerates, the distal branches disconnect, and each has its own site where injury potentials are generated. Because the individual muscle fibers no longer contract as a unit, the twitches (fibrillations) are uncoordinated among the individual muscle fibers and not visible on the surface. In addition, as a consequence of denervation, the muscle fibers express numerous ACh receptors that make the muscle fibers hypersensitive to circulating ACh.

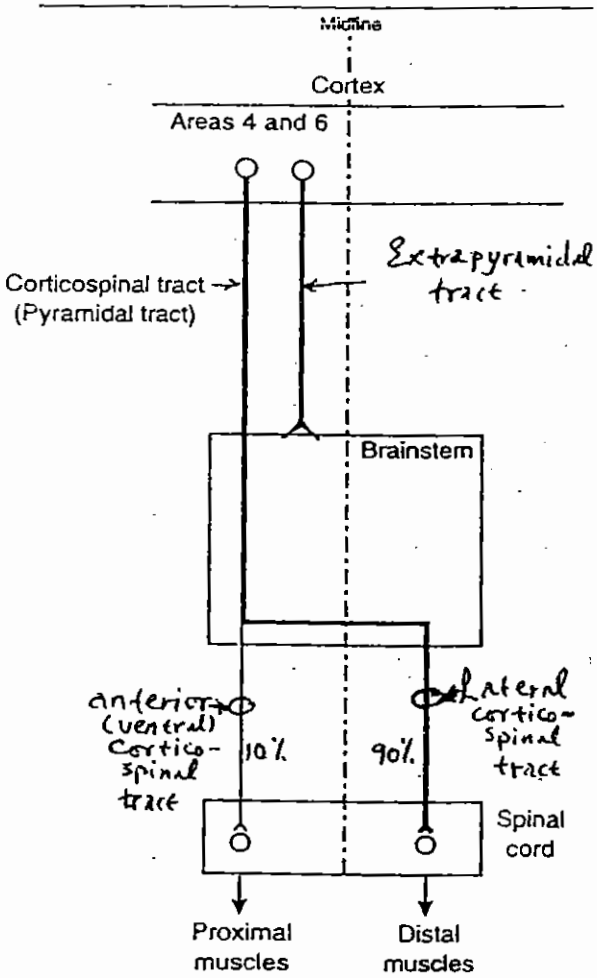


Premotor cortex ⊛

The premotor cortex receives its main inputs from the posterior parietal cortex, the cerebellum (via the ventrolateral thalamus) and the supplementary motor area. The main outputs project to the motor cortex, the brainstem and the spinal cord via the ventral corticospinal tract.

As with the supplementary motor area, the premotor cortex shows neural activity beginning well before movement onset. The premotor cortex appears to be involved in postural preparation for the coming movement, as indicated by its input to the anterior corticospinal tract.

Lesion : appearance of grasp Response ??



Remember that the Premotor cortex controls Proximal & axial muscles (through anterior (ventral) corticospinal tract as well as extrapyramidal tracts)

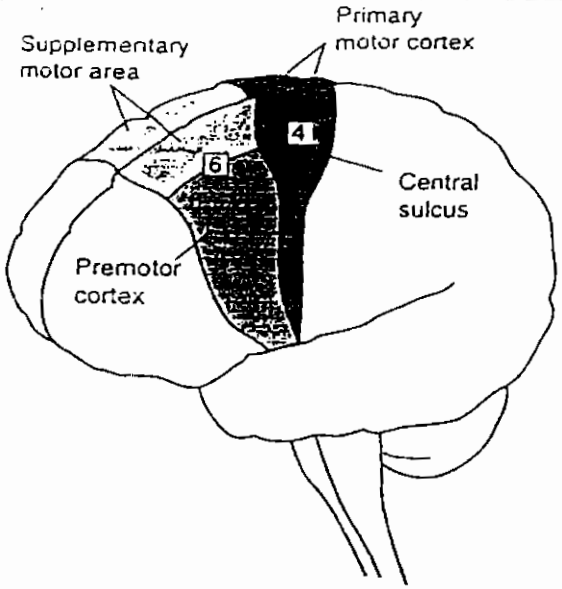
Remember that a lesion limited to area 4 → flaccid paralysis while a lesion affecting both areas 4 & 6 → Spastic paralysis ?? (Loss of control over brainstem inhibitory reticular formation .....)

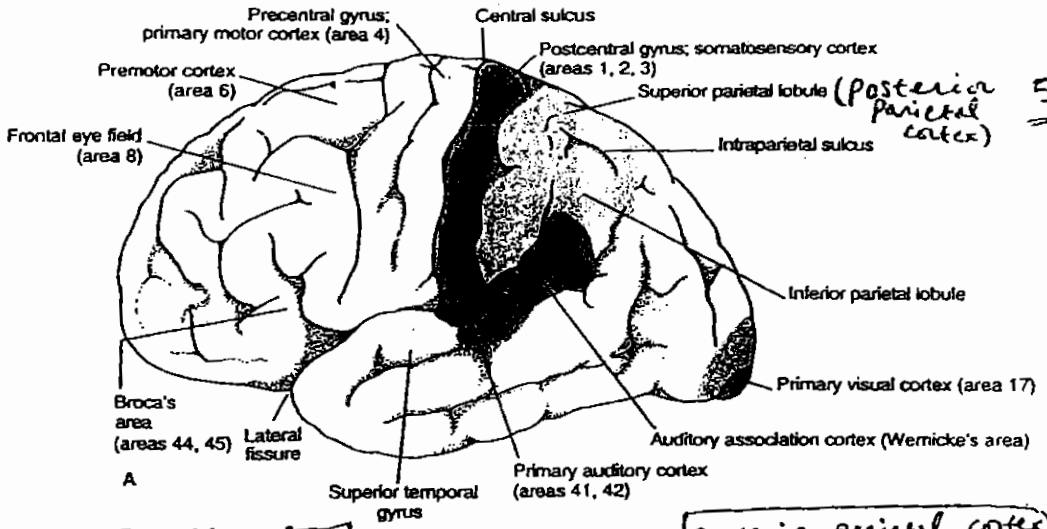
Posterior parietal cortex (5,7)

The posterior parietal cortex lies posterior to the somatic sensory cortex (Fig. 3.7.3A). The inputs to the posterior parietal cortex come from sensory areas of cerebral cortex carrying visual, auditory, cutaneous & proprioceptive informations as well as inputs from the motor areas → i.e in this region of brain Sensorimotor information is brought together to generate the conscious map of the body & of the body's position in space.

Lesion of Posterior parietal cortex → ⓐ inability to direct attention to sensory stimuli ⓑ the condition of hemi-neglect where the patient ignores & even denies the existence of one side of body ⓒ failure of movement planning → inability to relate the position of objects in space to that of the body

Fig. 12-4. Motor areas of the cerebral cortex.





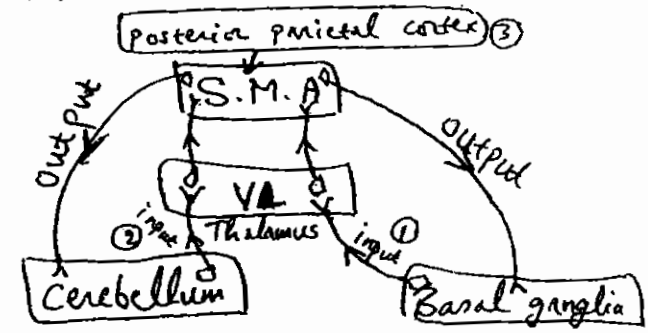
Supplementary motor area

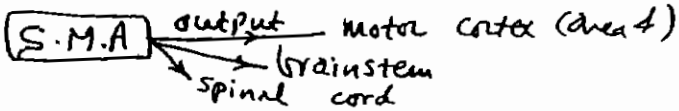
In order to produce complex movements, there must be a motor plan that specifies the sequence and extent of the muscle contractions needed to execute the movement itself and to effect the necessary postural adjustments associated with the movement; for example, compensating for a change in the position of the centre of gravity.

The output of the motor cortex activates specific muscles but does not of itself produce complex motor behaviour. This appears to be the role of the supplementary motor area and the premotor cortex (Fig. 3.7.3A).

The supplementary motor area receives inputs from the basal ganglia and the cerebellum (via the ventrolateral nucleus of the thalamus) and from the posterior parietal cortex. It also has outputs going to both the basal ganglia and the cerebellum as well as to the motor cortex and brainstem and a minor component direct to the spinal cord via the corticospinal tract. The input–output loops with the basal ganglia and the cerebellum indicate a role in movement programming.

Electrical stimulation of the supplementary motor area often produces complex, bilateral movements; and measurements of cortical blood flow reveal that the area is active during movements involving extensive coordination, particularly of both hands, but not during simple flexion/extension movements of single joints.





Lesions of the supplementary motor area result, for example, in the inability to orient the hand correctly when reaching for a target or to coordinate the hands during bimanual tasks.

40

Grasp Response (Reflex) *

Grasp Reflex. Stroke the patient's palm so he grasps your index finger between his thumb and index finger (Fig. 11-35). When the grasp reflex is present, he cannot release the fingers when he tries. This is a normal response in young infants; later in life, lesions of the premotor cortex may uncover the reflex as a pathologic finding.

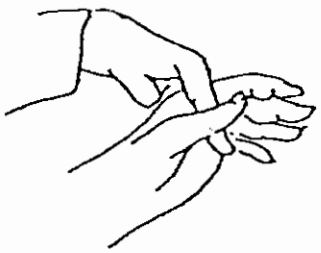


Fig. 11–35. Grasp reflex. *With your index finger between his thumb and index finger, stroke the patient's palm as he grasps your finger. In lesions of the premotor cortex, he may be unable to release his grasp.*

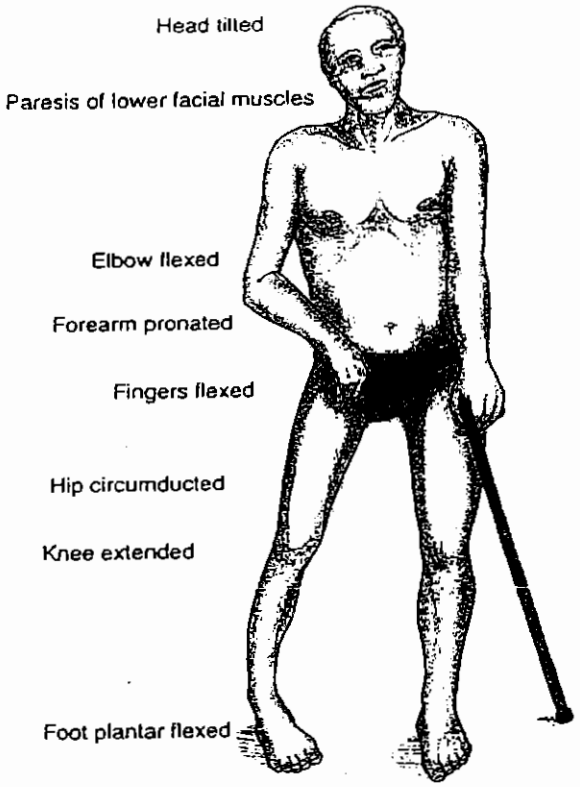


FIGURE 6–5. Right spastic hemiplegic. Gait resulting from left capsular lesion.

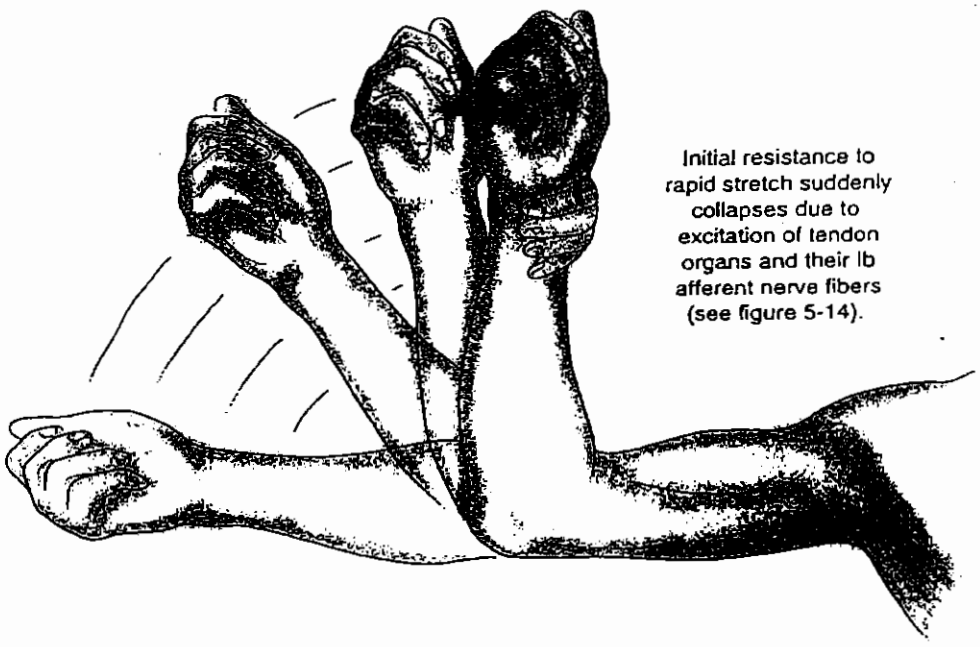


FIGURE 6–6. The clasp-knife response.

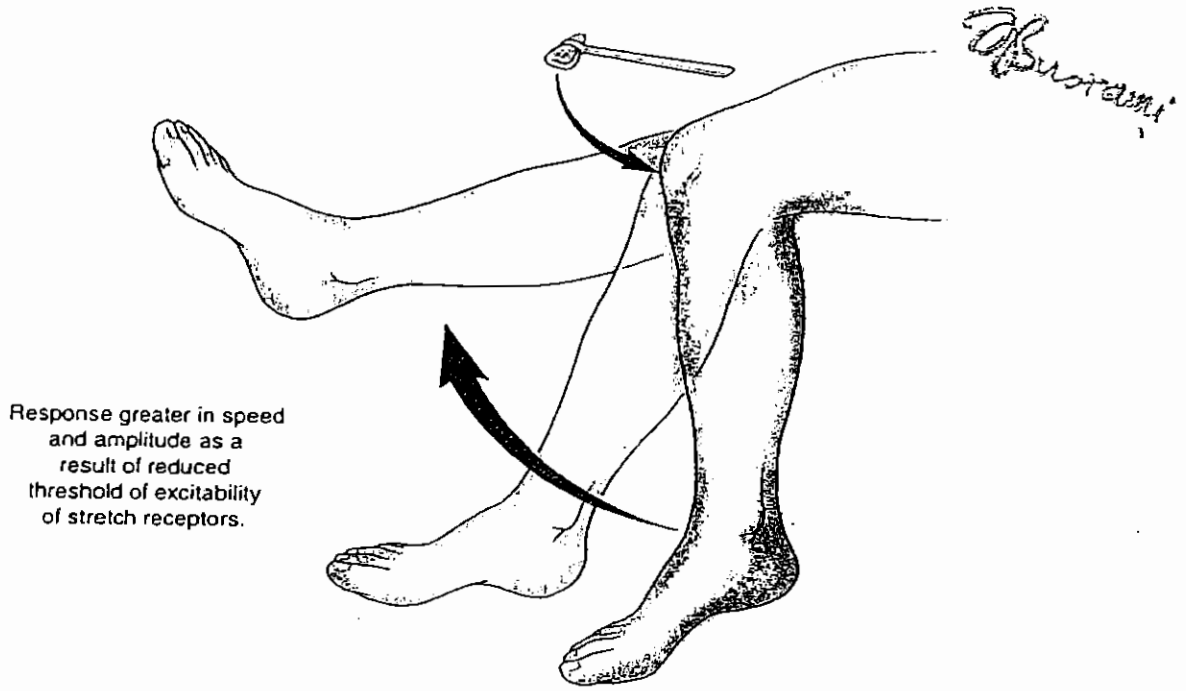


FIGURE 6-7. Exaggerated patellar reflex.

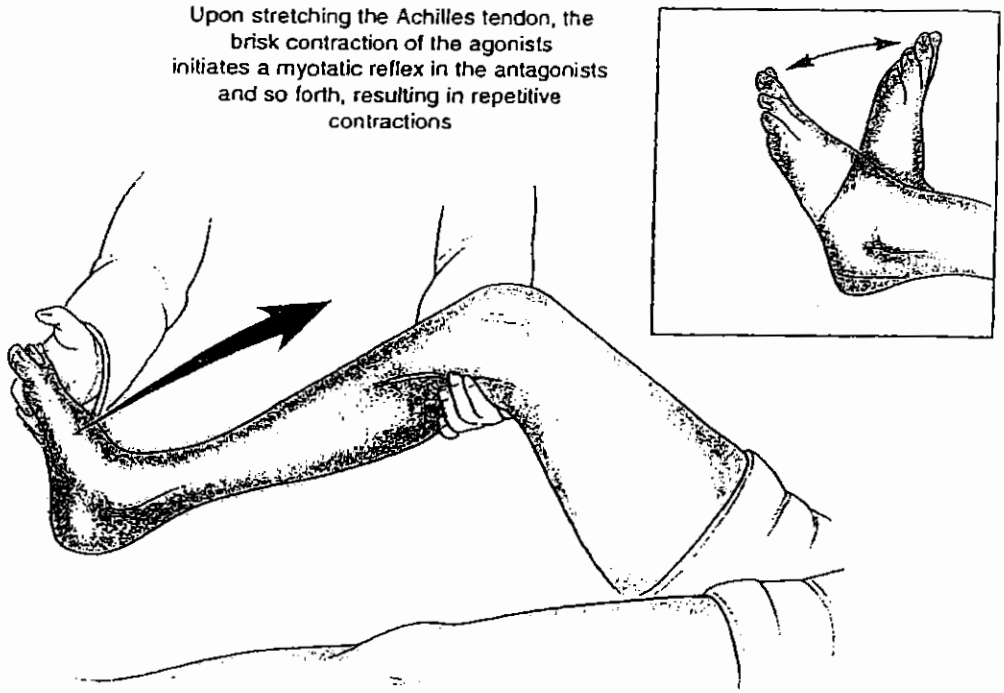


FIGURE 6-8. Clonus.

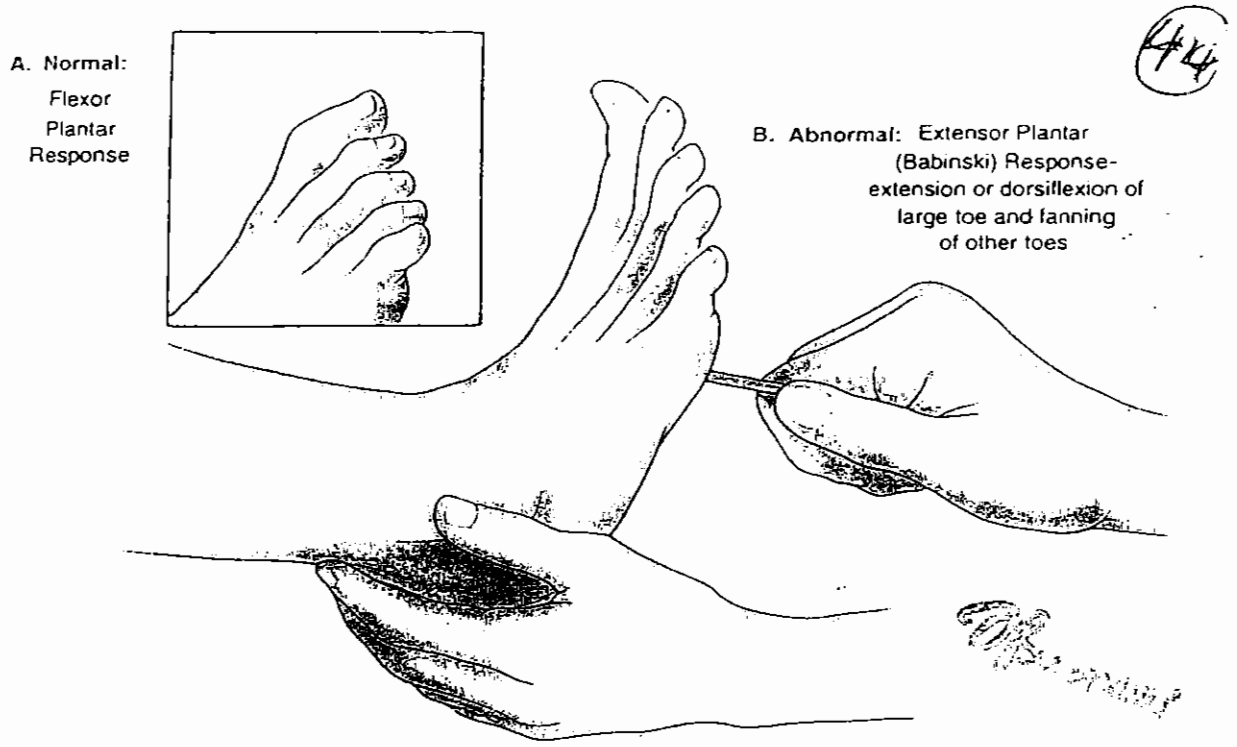


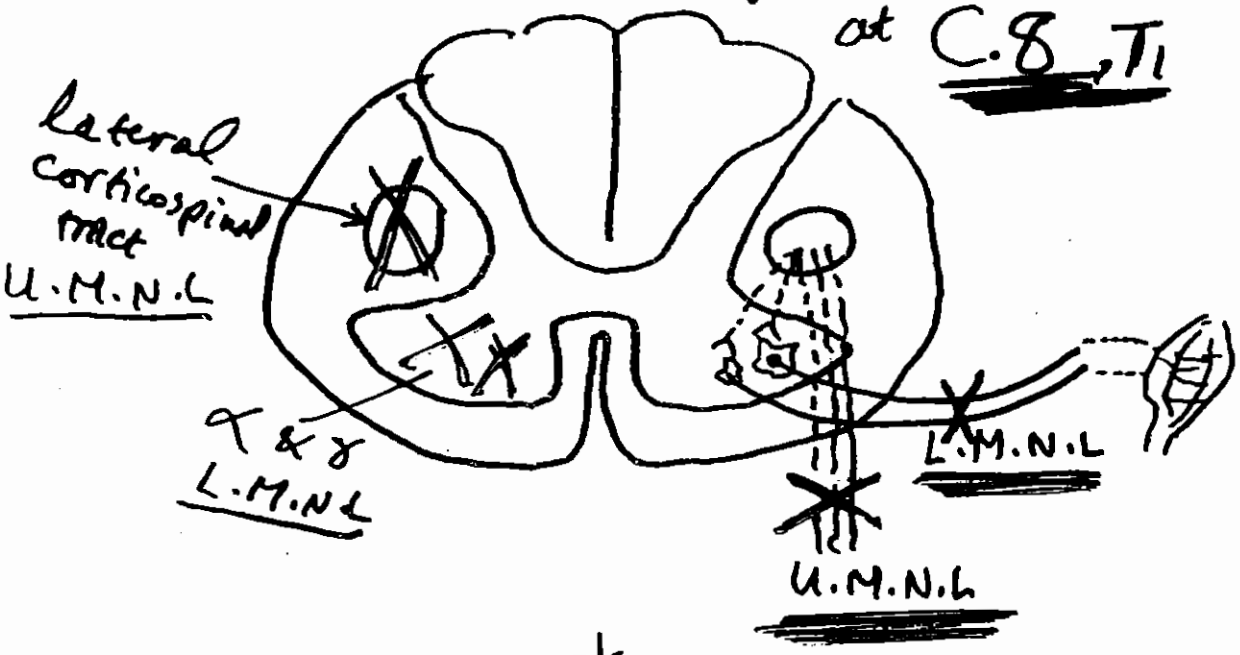
FIGURE 6–9. Plantar responses. A. Normal flexor. B. Abnormal extensor or Babinski.

MOTOR NEURON DISEASE

DBustami

Degeneration of both upper & lower motoneurons

e.g Lesion at lower cervical region of sp. cord at C.8, T1



* L.M.N.L at upper limb ⟶ Paralysis & ATROPHY of small muscles of hand

* U.M.N.L at lower limb ⟶ Paralysis ⊕ Hyperreflexia ⊕ clonus ⊕ +ve Babinski ....