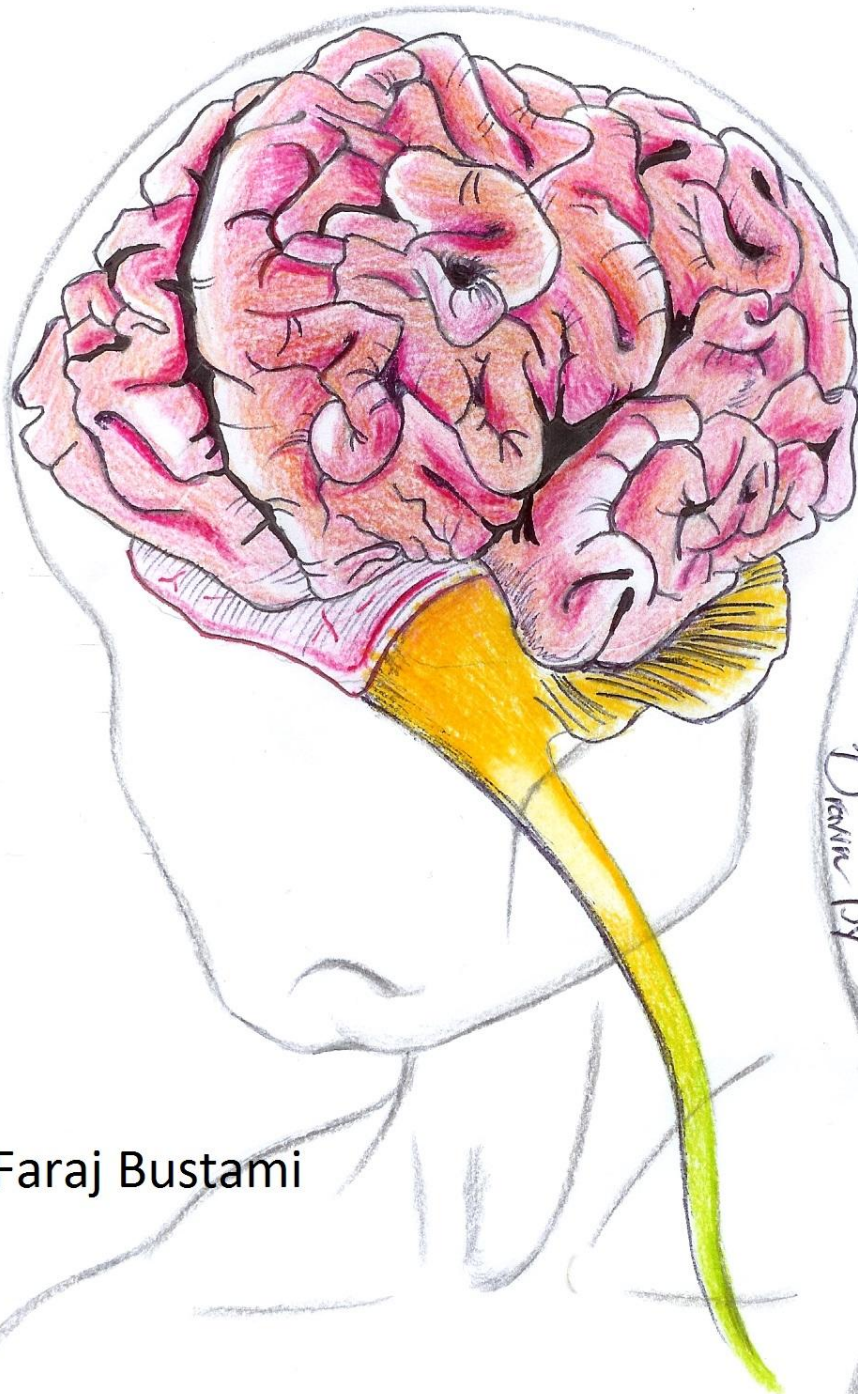


CENTRAL NERVOUS SYSTEM

- Handout
- Sheet
- Slide

- Anatomy
- Physiology
- Pathology
- Biochemistry
- Microbiology
- Pharmacology
- PBL



Drawn By Taria Bushraq...

Done By:

Dr. Name: Dr. Faraj Bustami

Lec #: 2

Neuro I
 2/2015

المجموعة الثانية (نظري)
 ا.د. طارق البساطي

of Sustani

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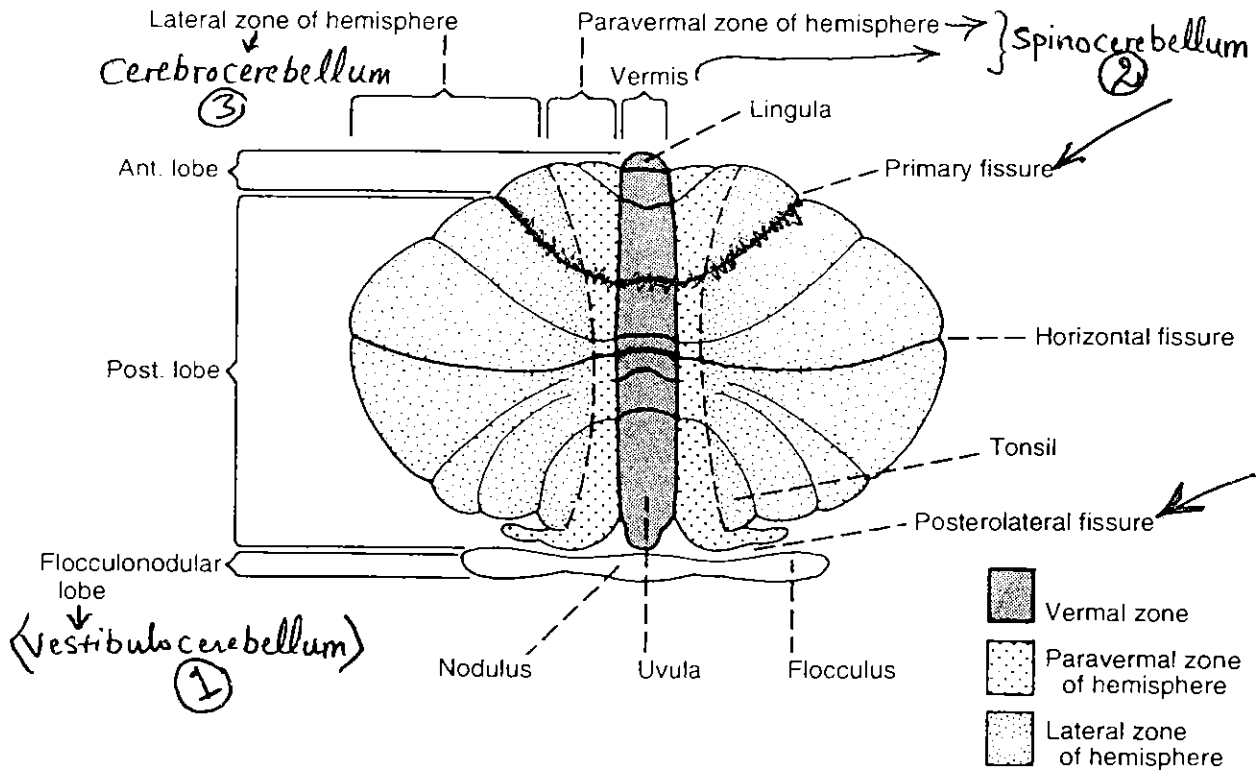


Figure 15.1. Schematic diagram of the fissures, lobules, and lobes of the cerebellum. Functional longitudinal zones of the cerebellum are associated with cerebellar nuclei. The vermal (median) zone projects to the fastigial nucleus; the paravermal (paramedian) zone projects to the interposed nucleus; the lateral zone projects to the dentate nucleus.

The LOBES and LOBULES of the CEREBELLUM

- The true division of the cerebellum is not into a median vermis and 2 cerebellar hemispheres but into lobes & lobules.
- Deep transverse fissures divide the cerebellum into different lobes and lobules. The fissures cut the vermis (transversely) and extend laterally into the hemispheres.

Therefore:

- Each lobe has a median part (in the vermis) and a pair of lateral extensions (in the hemispheres).
- A postero-lateral fissure separates the flocculo-nodular lobe (nodule in the middle and flocculus on either side) from the main part of the cerebellum.
- The most important fissure is the primary fissure which divides the main part of the cerebellum into anterior and posterior lobes.



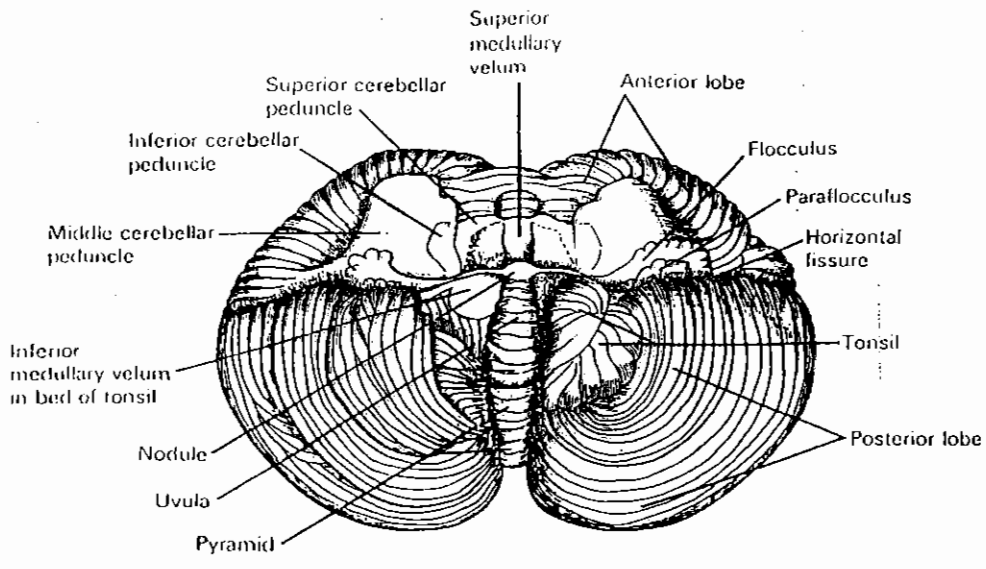


Fig. 7.2 Anteroventral surface of the cerebellum. The right tonsil of the cerebellum has been removed to show the inferior medullary velum.

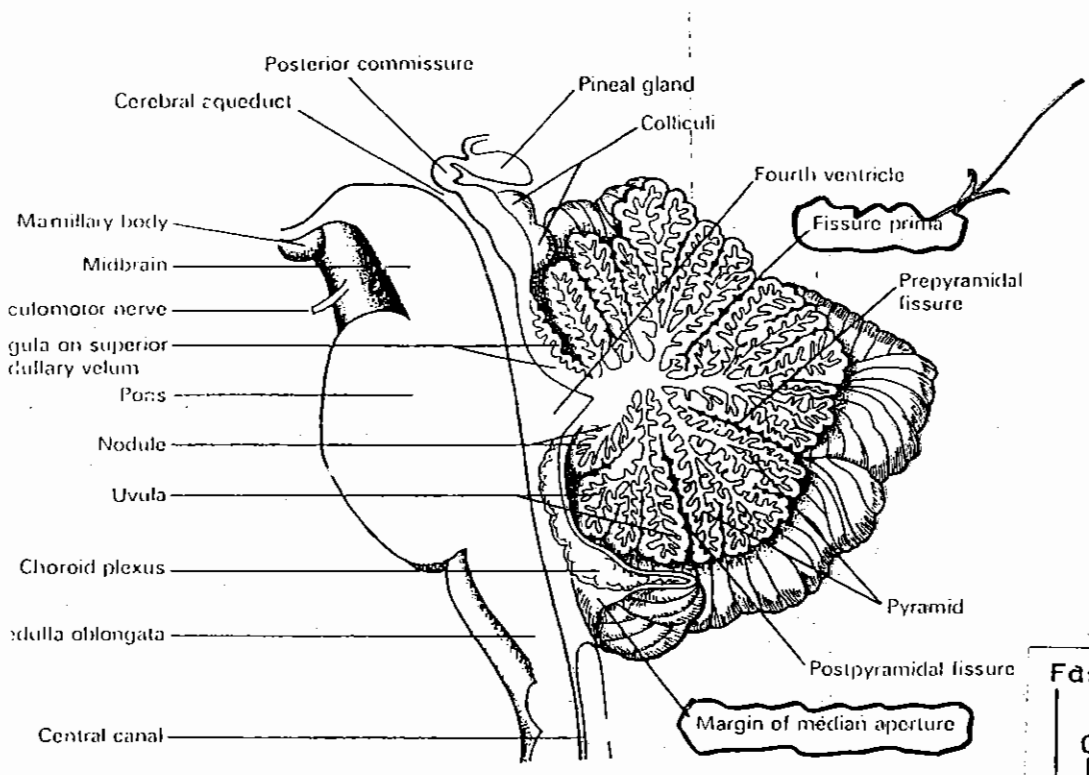
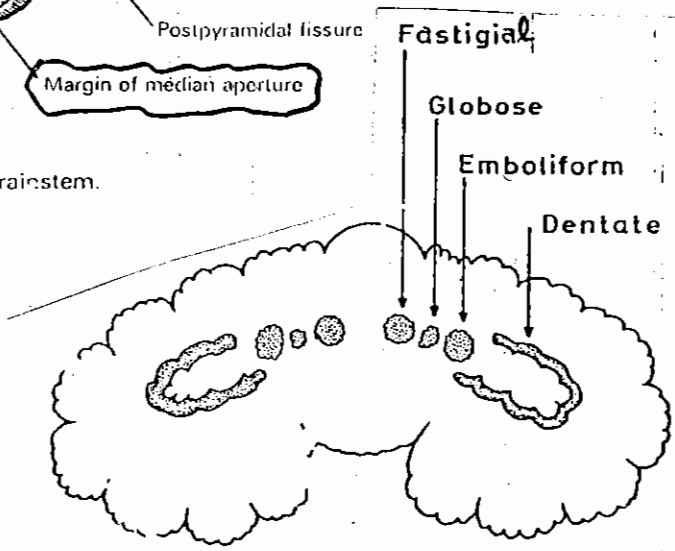


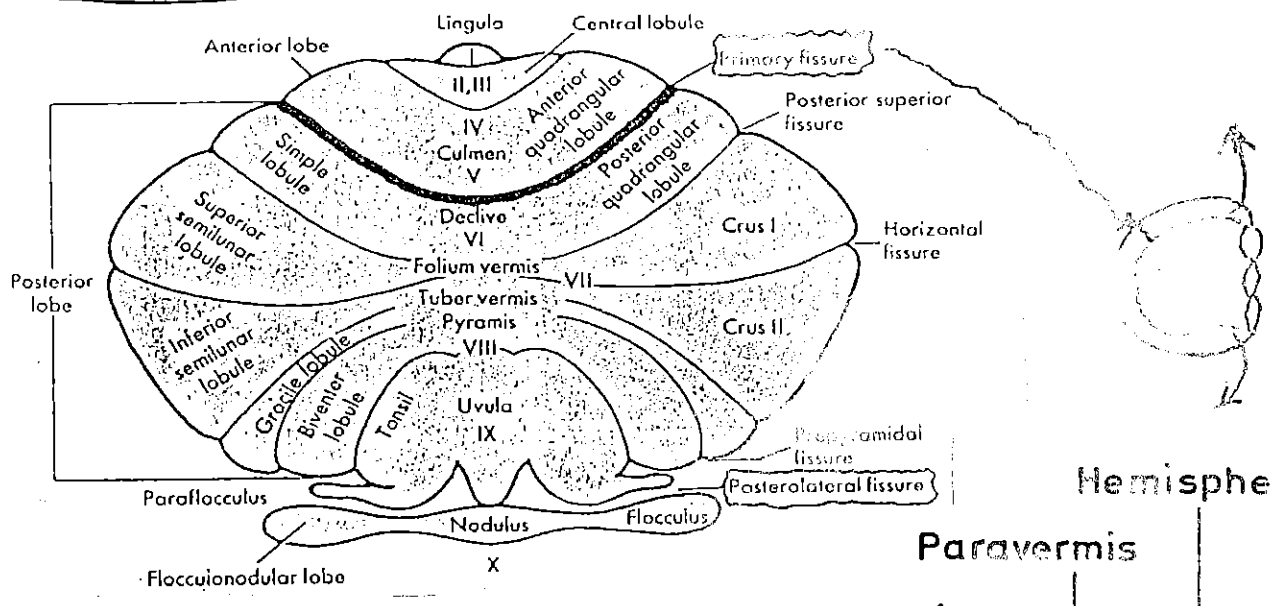
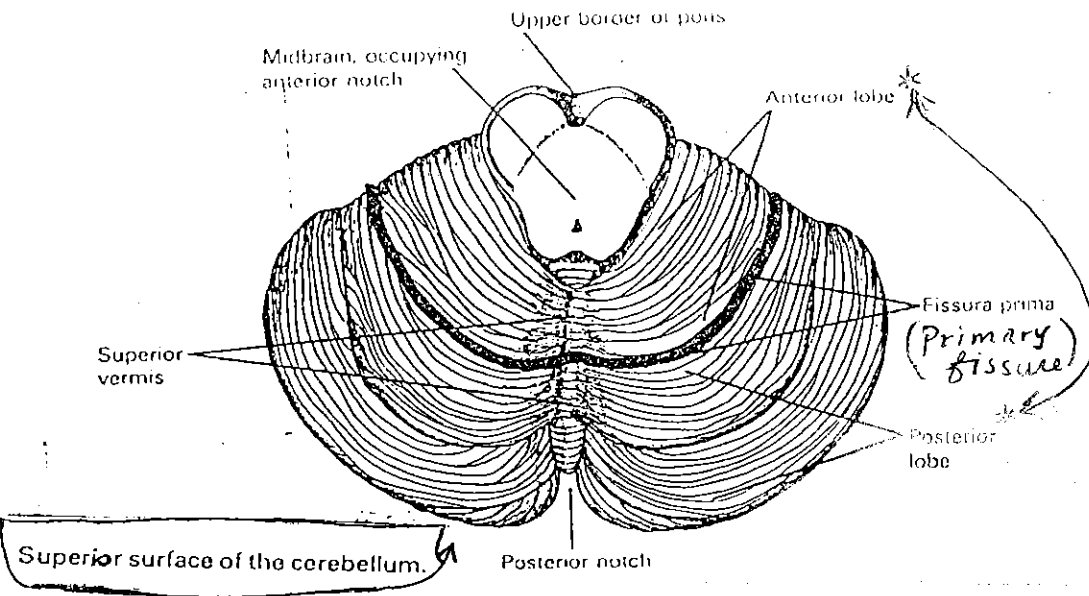
Fig. 7.3 Median sagittal section of the cerebellum and brainstem.

Buried in the white matter are the deep cerebellar nuclei. The dentate nucleus, shown in Fig. 14-10, is the most lateral of these. The other deep nuclei include the emboliform, globose, and fastigial nuclei. (In cats the emboliform and globose nuclei combine to form the interpositus nucleus.)



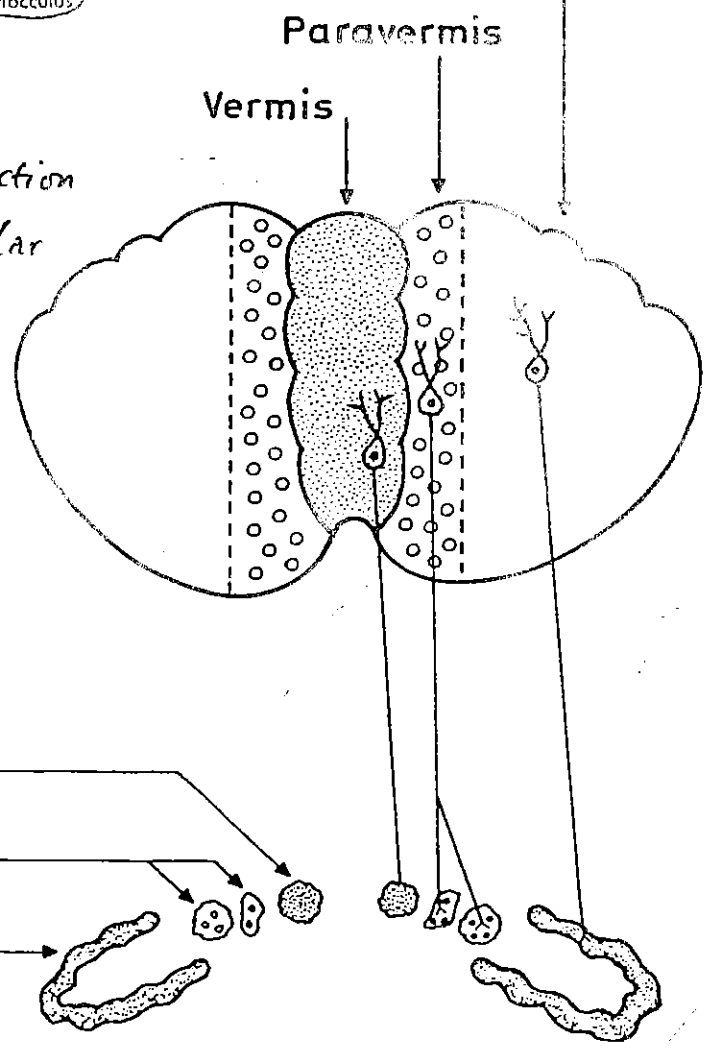
Schematic diagram showing the spatial arrangement of the deep cerebellar nuclei.

Botany

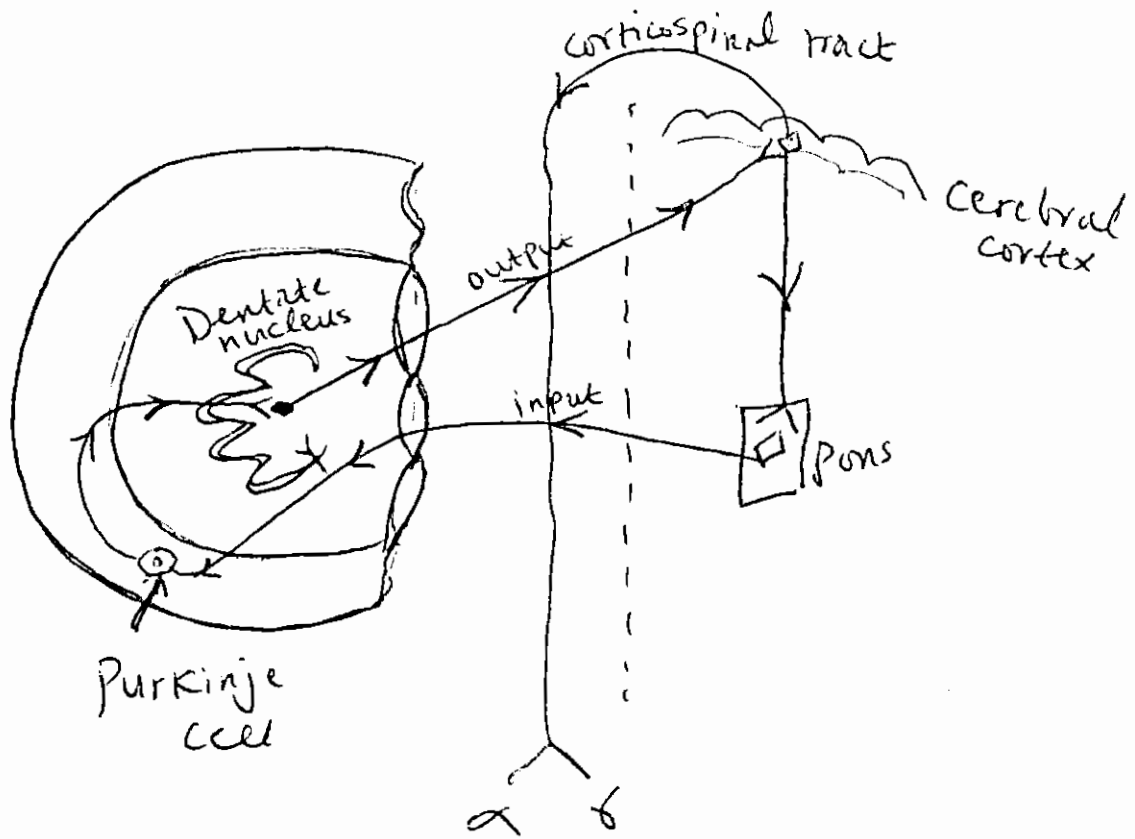
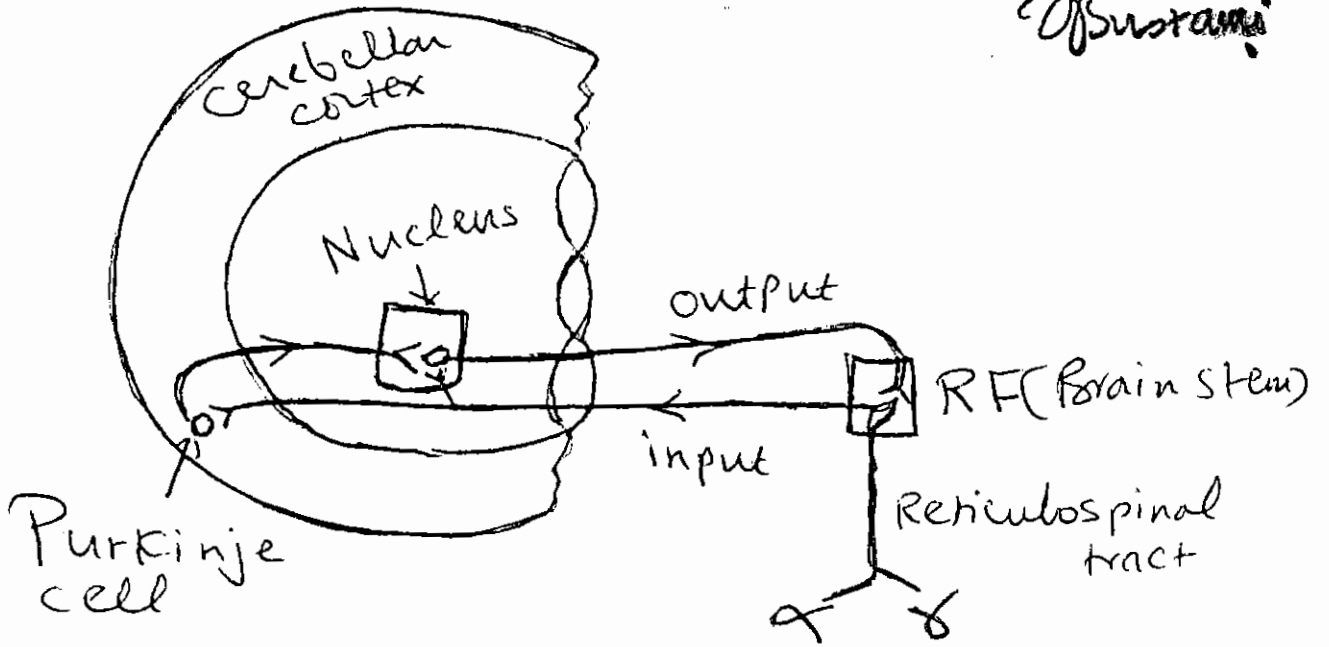


Hemisphere

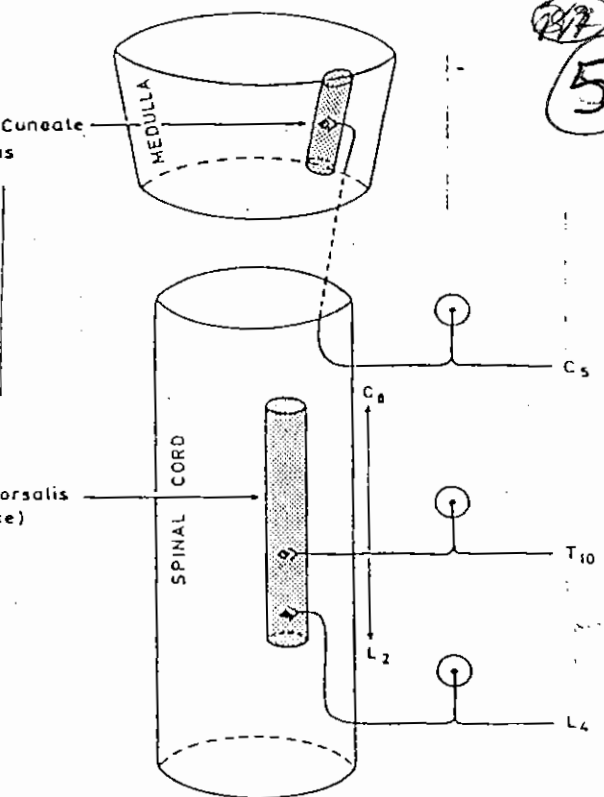
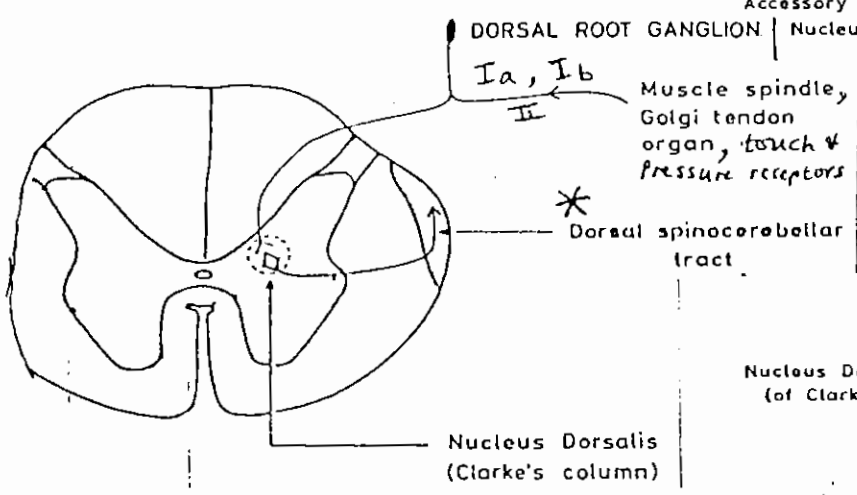
Diagram showing the projection of the different cerebellar zones into the deep cerebellar nuclei



of Buzsáki



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Spinocerebellar pathways (Fig. 9-12)

Figure 5.10. Schematic diagram of the spinal cord showing the homology of the accessory cuneate nucleus and the nucleus dorsalis of Clarke.

Pathway

1. Posterior (dorsal) Spinocerebellar tract

Primary afferents from

Ia, II, Ib from muscle spindle, Golgi tendon organ touch & pressure receptors (from skin) from Lower limb & Lower trunk

Transmission cells (nucleus)

Clarke's Column → their axons are the largest in the entire CNS → ascend ipsilaterally & enters inferior cerebellar peduncle

* Remember that the nucleus dorsalis (of Clarke) is limited to C₈-L₂ segments, then how afferents below L₂ reach this nucleus??

2. Cuneocerebellar tract

same afferents as the posterior sp. cerebellar tract but from UPPER LIMB

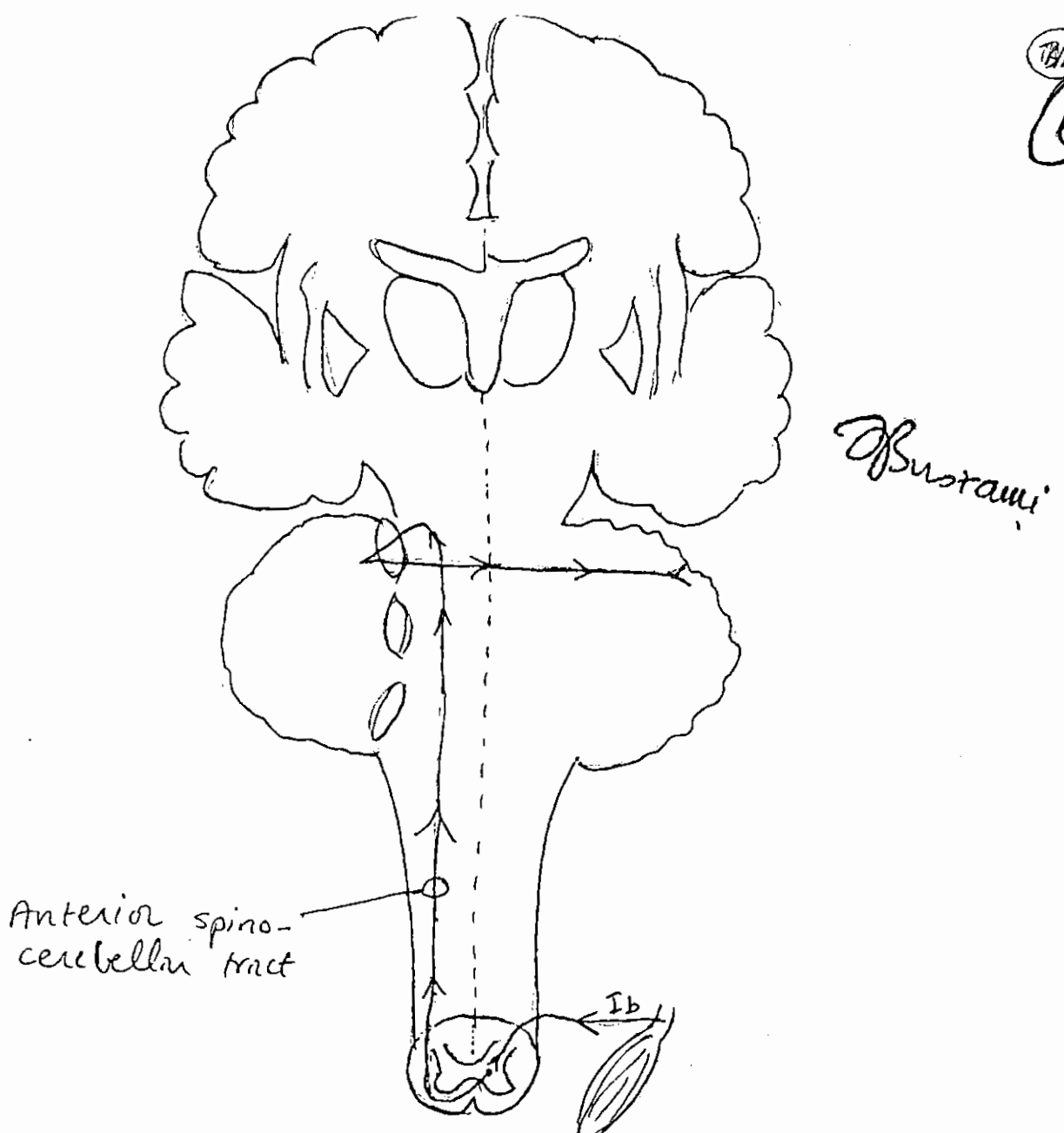
accessory cuneate nucleus (Rostro-lateral to cuneate nucleus in medulla) → axons ascend & enters ipsilateral inferior cerebellar peduncle

3. Anterior spinocerebellar tract

(Exclusively Ib afferent from Golgi tendon organ)

Along this pathway the cerebellum receive afferents from interneurons of spinal reflex arcs → Thus their function is to provide feedback to the cerebellum about on-going spinal reflexes
1 + 2 + 3 → Carry Unconscious Proprioception

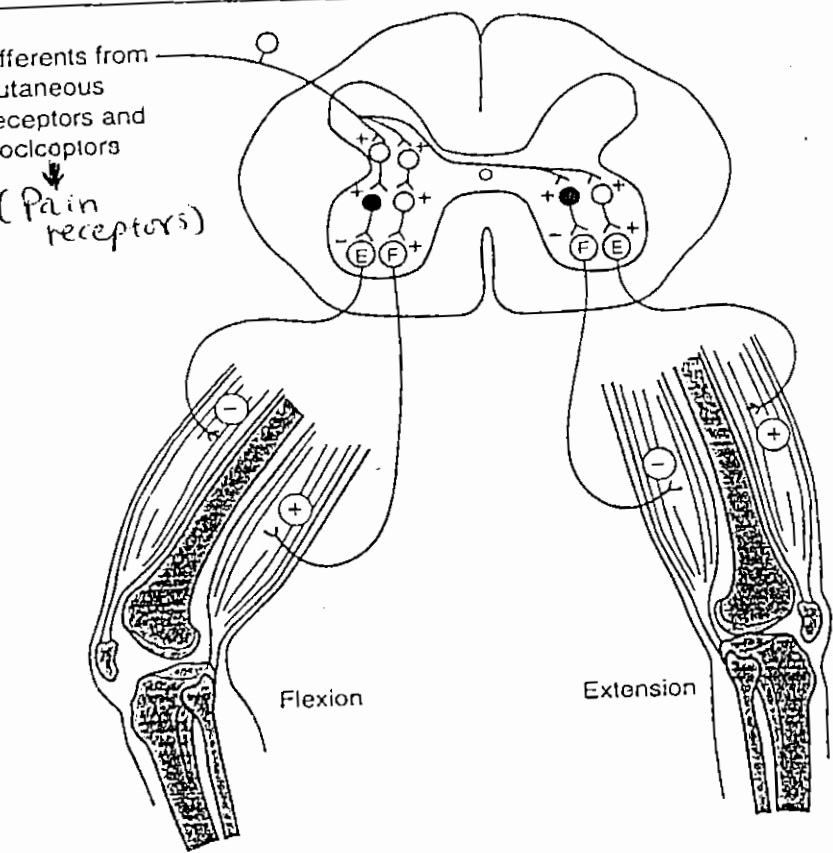
Laminae V to VII
↓
axons cross in sp. cord → ascend in brainstem → cross again & enters cerebellum along superior cerebellar peduncle



Notice that the anterior spinocerebellar tract undergoes double crossing, one in the spinal cord and another in the cerebellum-brainstem → This is in keeping with the known control by each cerebellum hemisphere of movements on its own side of the body (unlike the motor cortex which controls contralateral movement)

of the organization of the flexion-crossed extension reflex.

Bustami
 (BA)
 7

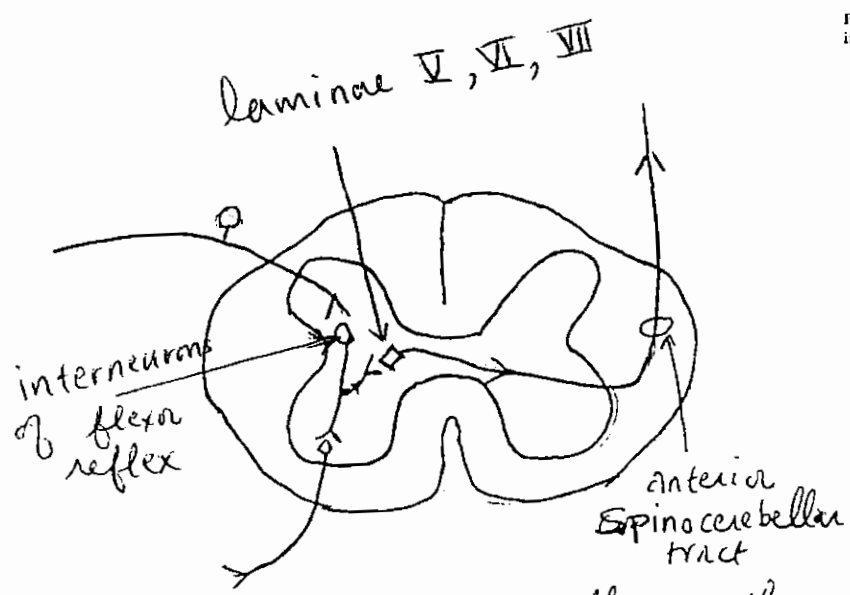


The flexion reflex is polysynaptic (Fig. 11-7). The afferent fibers enter the spinal cord and excite interneurons of the dorsal horn. The interneurons then act on alpha motor neurons through relay pathways involving other interneurons. The response is an excitation of alpha motor neurons to the flexor 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.

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.



Fig. 10-3 Flexor reflex with crossed extensor thrust. The black internuncial is inhibitory.

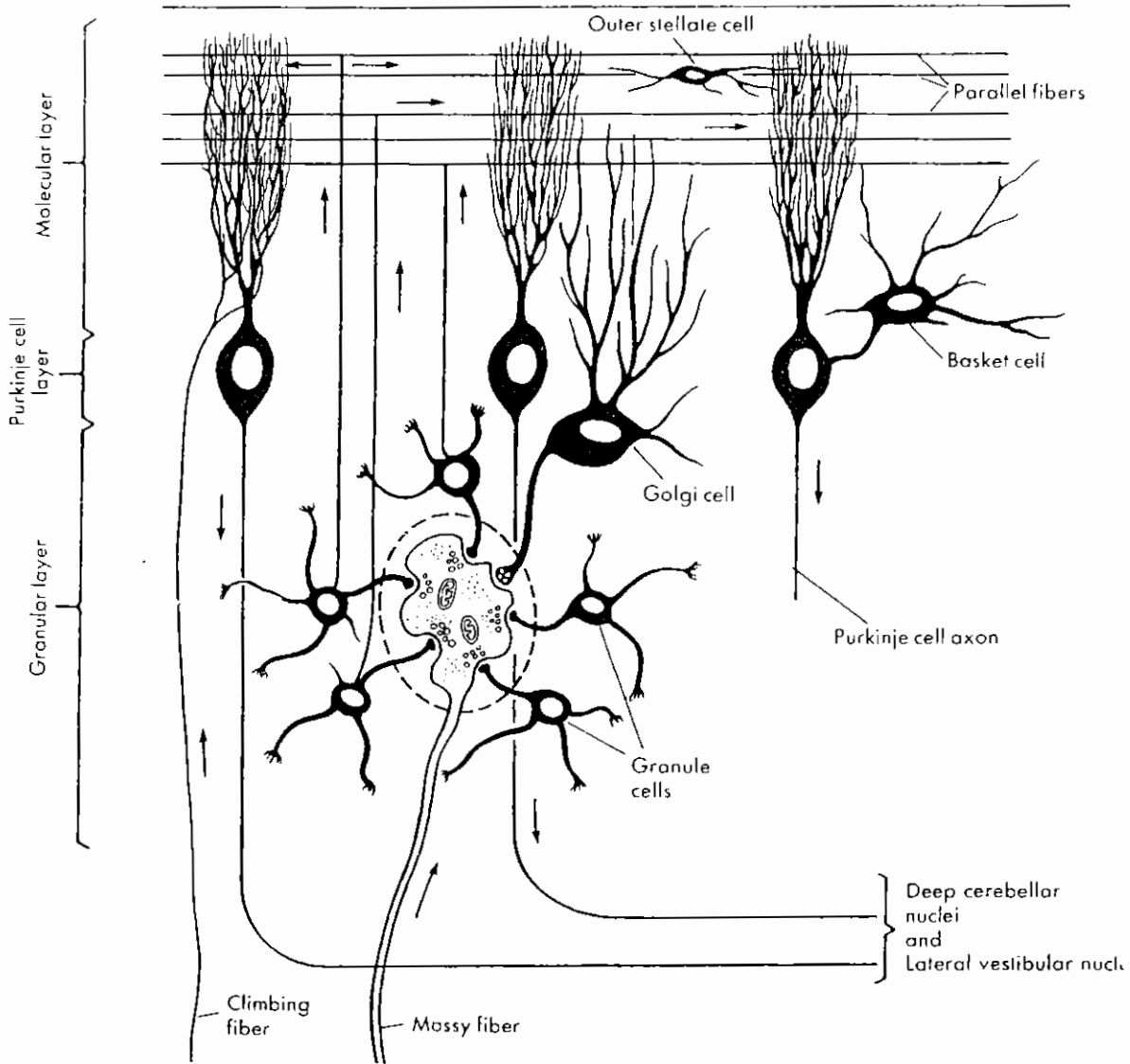


The transmission cells of the anterior spinocerebellar tract (in laminae V, VI, VII) receive afferents from interneurons of the flexor reflex → Their function is to provide feedback to the cerebellum about on-going spinal reflexes

Dr. Swarani

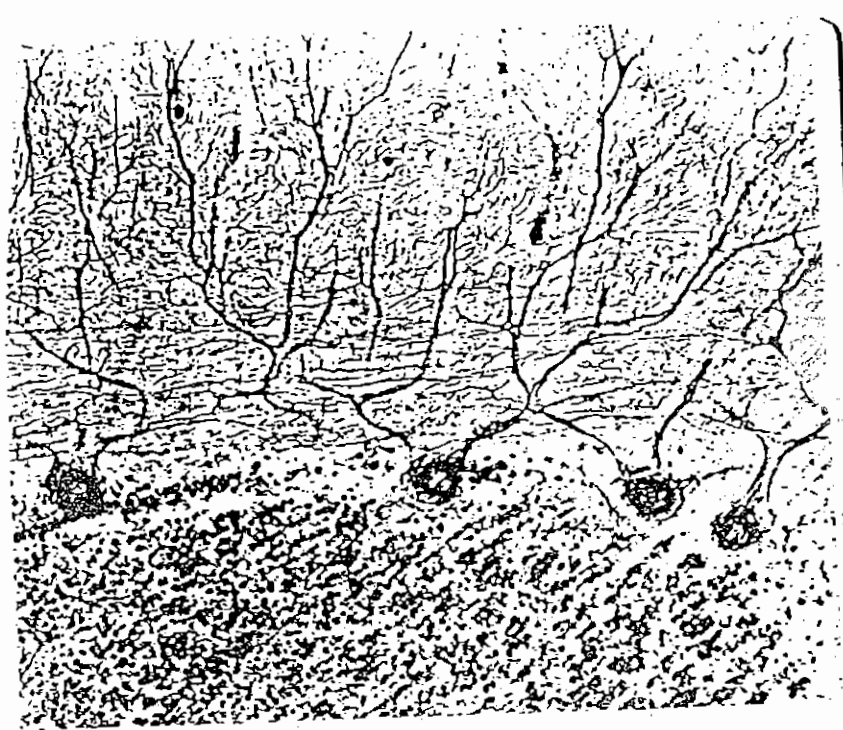


Dr. Swarani



Of Bostami

(BW) 9



2 types of neurons $\left\{ \begin{array}{l} \text{Stellate} \\ \text{\& basket cells} \end{array} \right.$

Molecular layer (outermost)

- dendrites of Purkinje and Golgi type II cells
- axons (T-shaped parallel fibres) of the granule cells

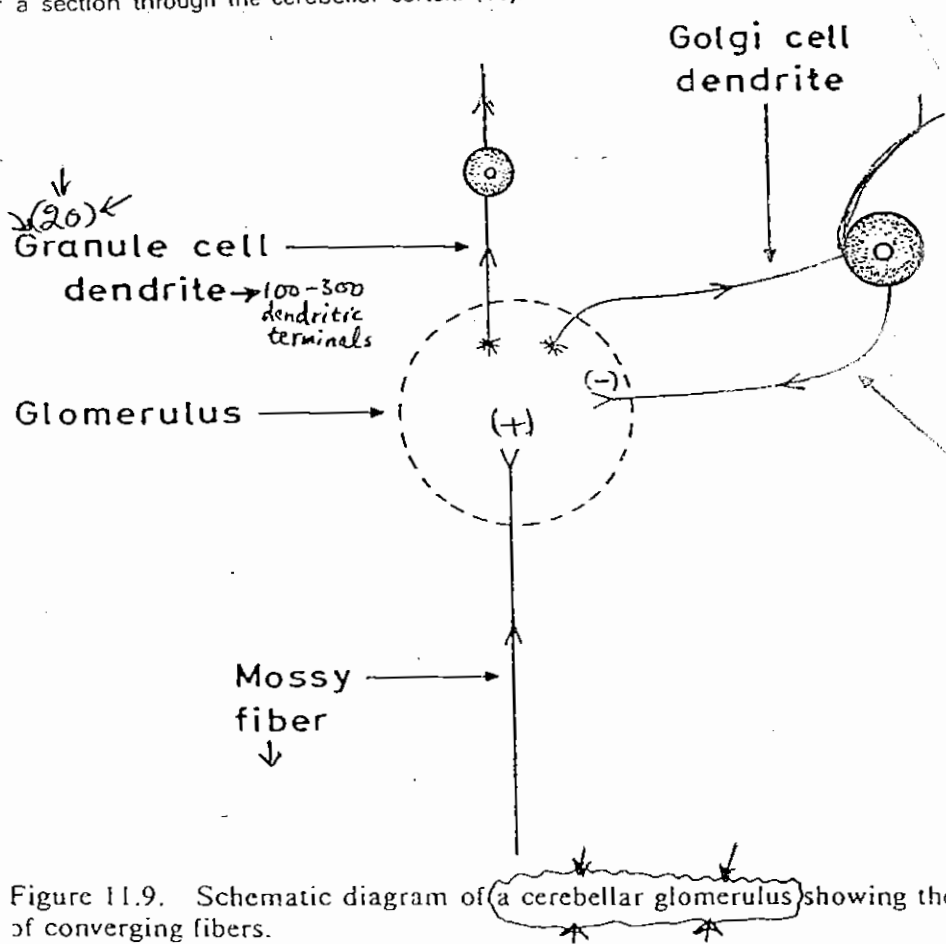
Purkinje cell layer (middle layer)

- their dendrites pass into the molecular layer
- their long axons synapse upon deep cerebellar nuclei (or vestibular nuclei)

Granular cell layer (innermost layer)

- numerous granule cells (neurons)
- Golgi type II cells (neurons)
- glomeruli*

* a section through the cerebellar cortex. (Cajal silver stain, x300.)

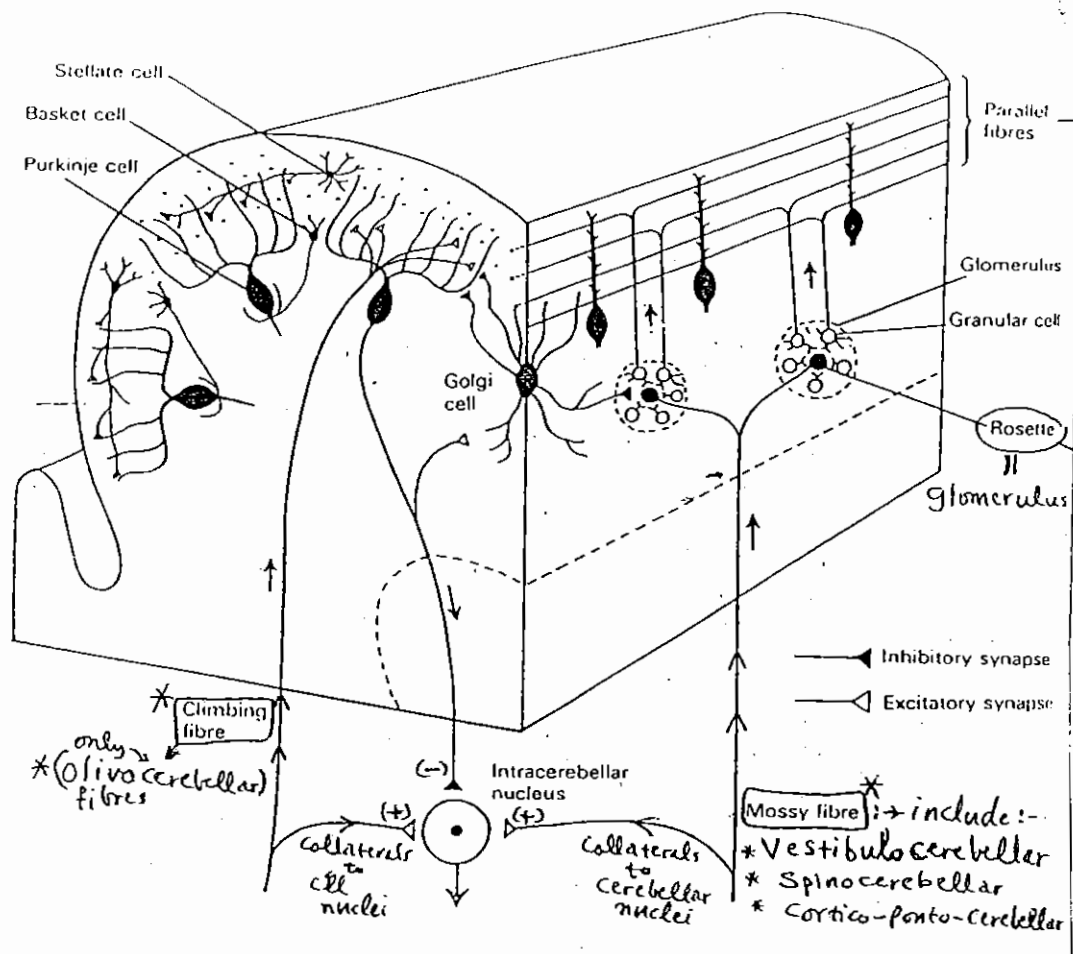


Complex synaptic nodules which contain AXONS of incoming MOSSY FIBERS (Vestibulo-cerebellar, spino cerebellar & costico pontocerebellar)

Golgi cell AXONS & DENDRITE axon of Golgi type II a

DENDRITES of granule cells

Figure 11.9. Schematic diagram of a cerebellar glomerulus showing the different sources of converging fibers.



Afferents (input) to the cerebellum terminate either in the granular cell layer (in the glomeruli as MOSSY FIBERS) or upon the dendrites of Purkinje cells as CLIMBING FIBERS.

Both inputs are excitatory.

Excited granule cell can excite Purkinje cells, basket and stellate cells and Golgi type II cell.

In turn the basket and stellate cells inhibit Purkinje cells.

the Golgi type II cells inhibit granule cells.

Fig. 7.7 Neuronal organization of the cerebellar cortex.

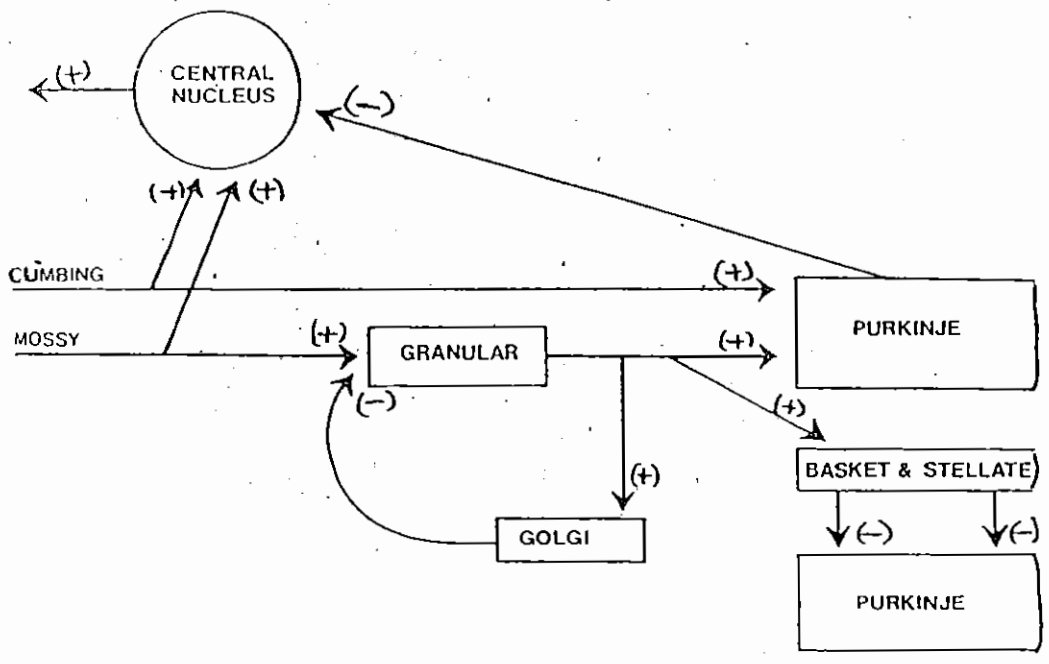


Fig. 7.8 Diagram of neuronal circuitry in the cerebellum: excitation is indicated in red (+) inhibition in black (-)

Finally the Purkinje cells (the bottleneck for all information coming out of the cerebellar cortex) are inhibitory to the deep cerebellar nuclei.

The cerebellar nuclei are subjected to initial excitation (by incoming climbing and mossy fibres) which is followed after few milliseconds by inhibition (from Purkinje cells).

The motor pathway (Motor cortex or brainstem nuclei) will receive an early excitation from cerebellar nuclei (which correct and modify the motor movement of the agonist), which is followed within a few milliseconds by excitation of the antagonists → To stop movement at the proper point → Damping

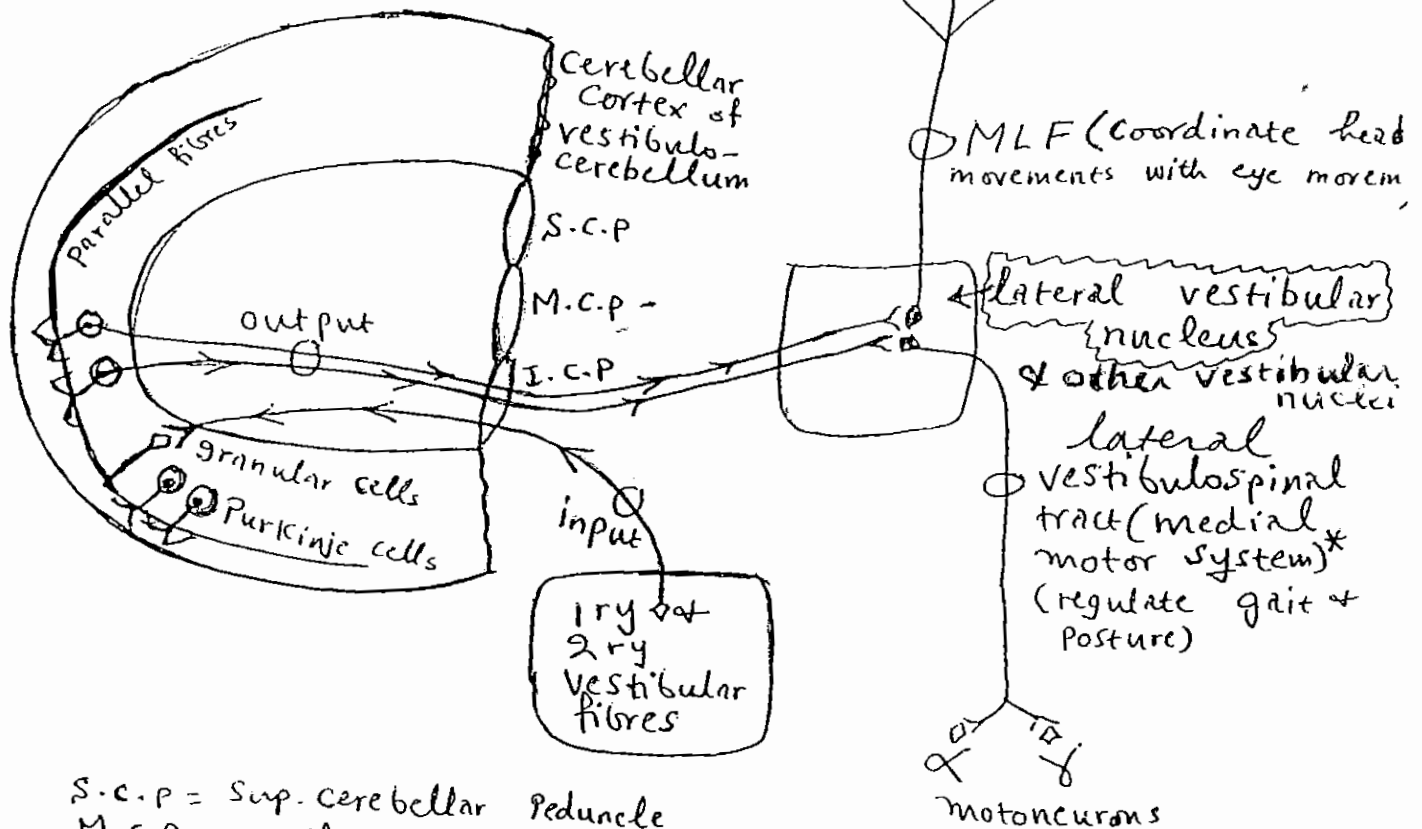
A balance between excitation & inhibition normally exists keeping an output from cell nuclei (cerebellum)

Connections of the vestibulo cerebellum (flocculonodular lobe)

Of Sustains

BBB

11



S.c.p = Sup. cerebellar peduncle

M.c.p = middle " "

I.c.p = Inferior " "

MLF = (medial longitudinal fasciculus)

Input to the vestibulo cerebellum (flocculonodular lobe)

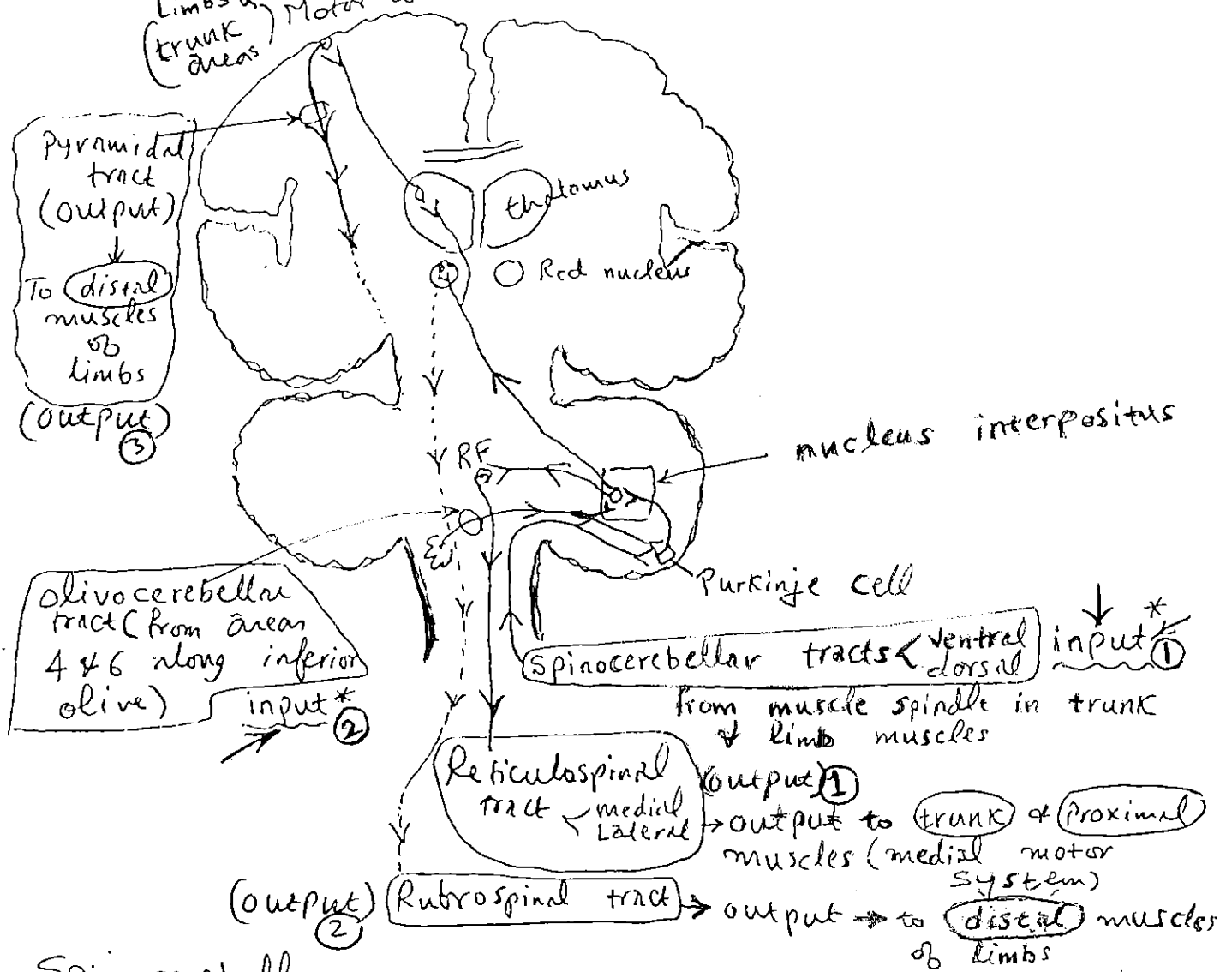
Primary (from labyrinth) and secondary (from vestibular nuclei) vestibular fibres → Enter the cerebellum (mostly through Inferior cerebellar peduncle) and end as mossy fibres of the flocculonodular lobe → The mossy fibres synapse with granule cells (within the glomeruli) which in turn synapse with the Purkinje cells in the molecular layer of the cerebellar cortex

Output of the vestibulo cerebellum

- The Purkinje cells of the vestibulo cerebellum project to the lateral vestibular nucleus (Exception to the rule) ???
- The lateral vestibular nucleus sends down the lateral vestibulospinal tract (part of medial motor system) which facilitates α & γ motoneurons that supply extensor muscles (gait & posture).

B Spino cerebellum

Limbs & trunk areas
Motor cortex



Spino cerebellum

input ← Spino cerebellar tracts ①
 ← Olivocerebellar = (cortico-olivo-cerebellar tract) ②

Output (from nucleus interpositus)

→ To RF (reticular formation) → Reticulospinal tracts
 = Red nucleus → Rubrospinal tract
 → To thalamus → motor cortex → Pyramidal tract
 area (4) (6)

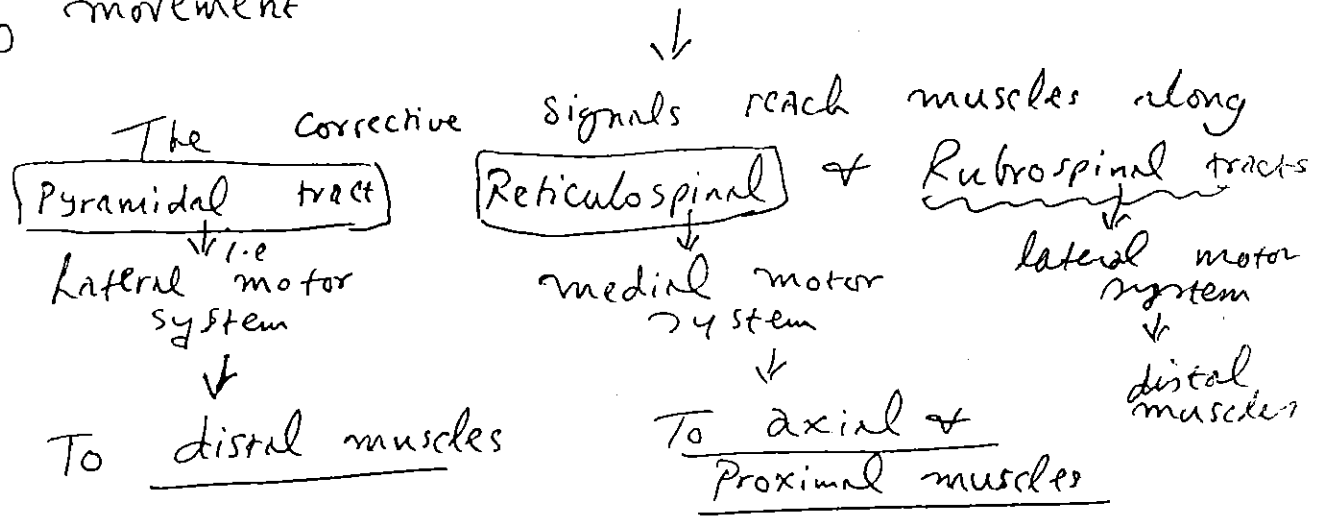
Spinocerebellum functions in the regulation of axial (trunk), proximal & distal muscles of Sustained

it controls SYNERGY (Coordination) among muscles (especially coordination between agonist and antagonist)

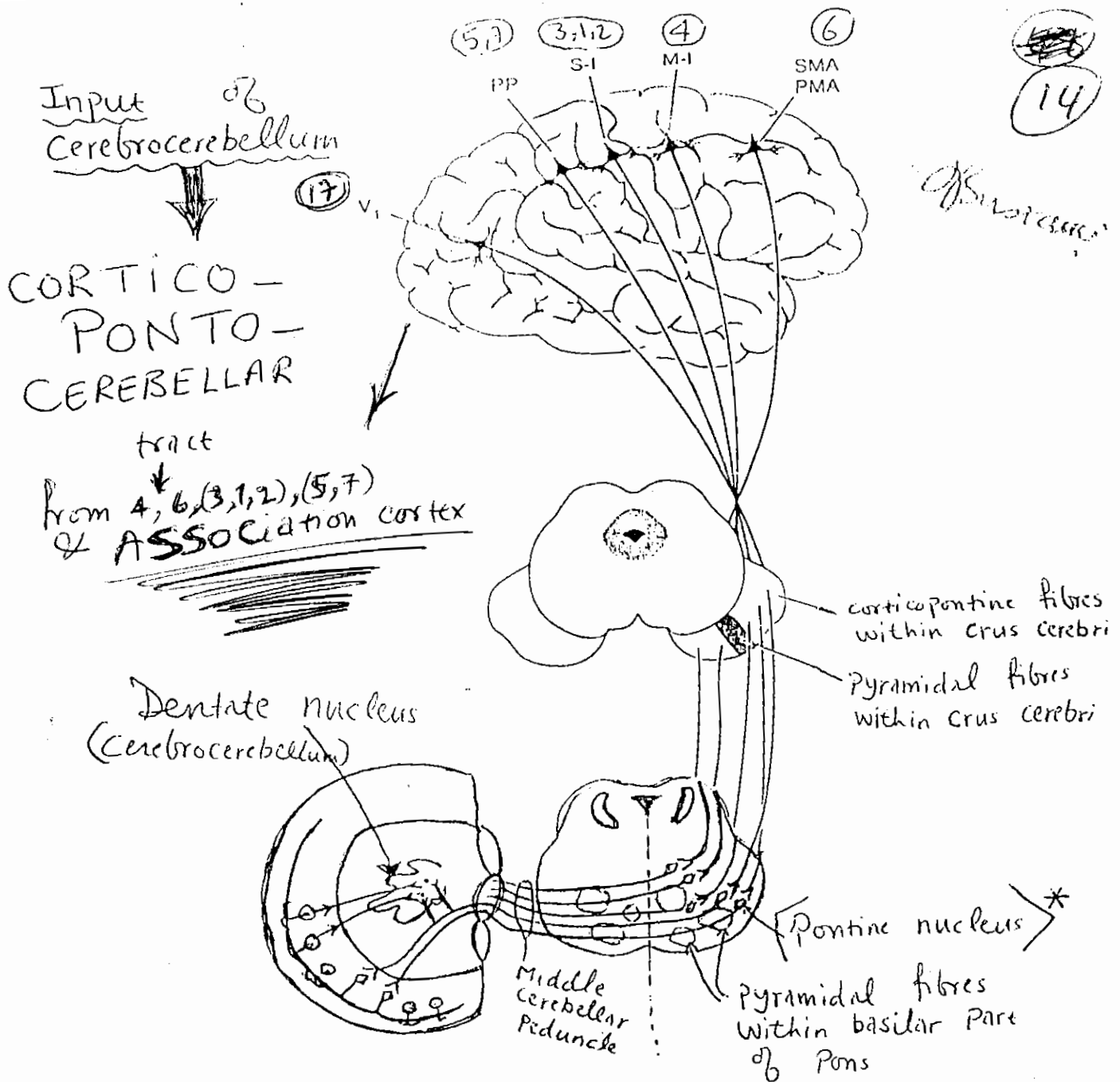
- ① it receives input from Receptors in muscles & joints about actual performance (movement)
- ② it receives input from cerebral cortex about intended performance (movement)

it compares the 2 inputs and when needed → sends Corrective signals that control RATE, RANGE, FORCE & DIRECTION

of movement



N.B The Spinocerebellum and vestibulo cerebellum affect the motor act while it is in progress i.e they affect actual movement



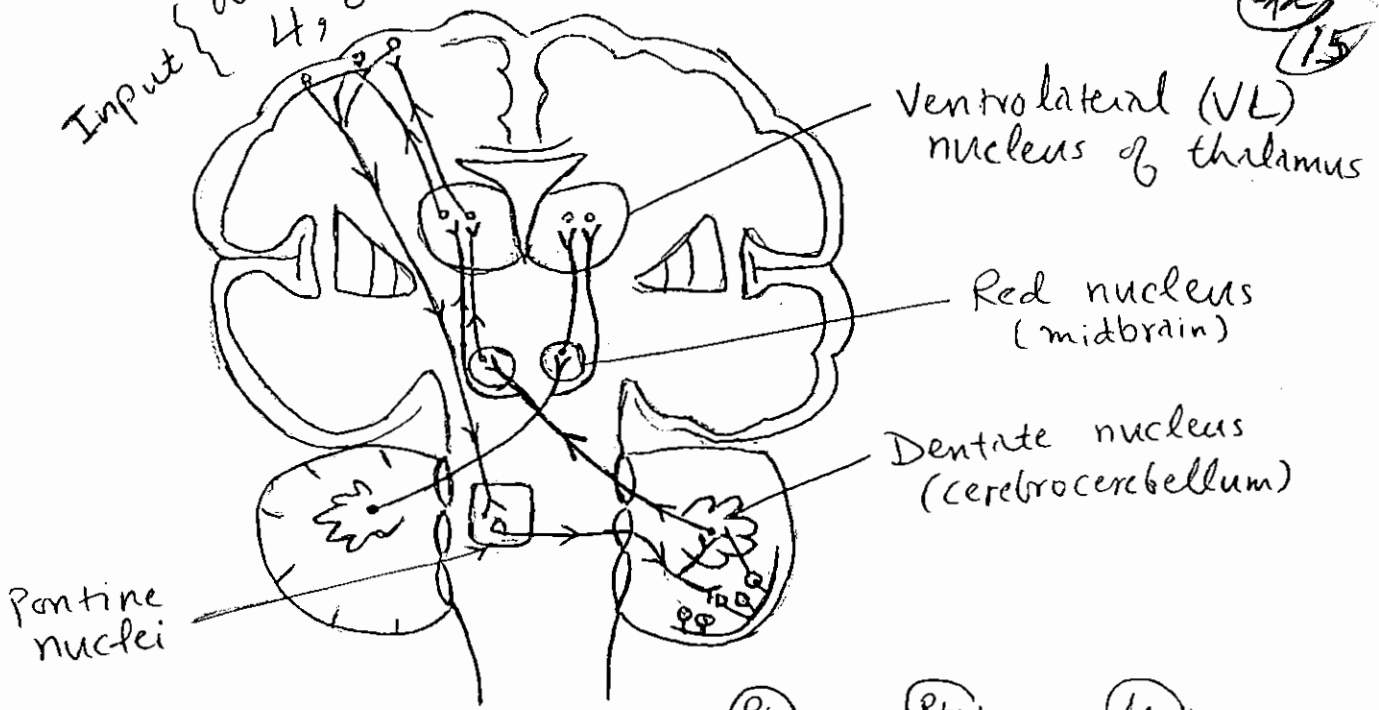
The (deep pontine nuclei) receive afferent fibers from most parts of the cerebral cortex. PMA, premotor area; SMA, supplementary motor area; M-I, primary motor cortex; S-I, primary somatosensory cortex; PP, posterior parietal cortex; -VI, primary visual cortex;

Notice → the Middle cerebellar Peduncle (M.C.P) is formed by the axons of Pontine nuclei (20 million axons) of the opposite side → enter the cerebellum and end as Mossy fibres of the cerebrocerebellum → (send collaterals to the Dentate nucleus and end by synapsing with granule cells which in turn synapse by the Purkinje cells) *

Input { areas 4, 6 } Association cortex

Bustani

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* Input of cerebrocerebellum $\text{Cortico}^{\text{Rt.}} \rightarrow \text{Ponto}^{\text{Rt.}} \rightarrow \text{Cerebellar tract}^{\text{Lt.}}$
 (synapse)

* Output of cerebrocerebellum
 $\text{Dentate}^{\text{Lt.}} \xrightarrow[\text{sup. cerebellar peduncles}]{\text{crossing of}} \text{Rubro}^{\text{Rt.}} \rightarrow \text{thalamo}^{\text{Rt.}} \rightarrow \text{Cortical}^{\text{Rt.}}$
 Dentate nucleus in cerebrocerebellum \downarrow some fibres synapse within the red nucleus, others, run around it forming capsule of red nucleus \downarrow VL nucleus of thalamus \downarrow areas 4, 6

* Functions of cerebrocerebellum
 \downarrow Regulation of ballistic movement
planning, initiation & timing of sequential movement

* The cerebrocerebellum receives information from primary motor area, premotor & supplementary motor areas (concerning the motor command that is about to be executed i.e. about intention movement) and from the sensory areas of the parietal lobe (about the postural state of the body) \rightarrow The output from the cerebrocerebellum feeds back to the red nucleus and the primary motor cortex where it can affect the motor command BEFORE THE EXECUTION OF THE COMMAND \rightarrow Thus the cerebrocerebellum is arranged to act like a FEED-FORWARD CONTROL SYSTEM (This is in contrast to the vestibular and spinal divisions of the cerebellum which act as typical feedback control systems \rightarrow The output from the vestibulocerebellum and spinocerebellum affect the motor act while it is in progress i.e. affect actual movement)

Of Sustains
~~50~~
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Lesions to the cerebellum DO NOT Produce Paralysis. Without the cerebellum, however, Precise, coordinated movements are impossible →

These observations suggest that the cerebellum acts as a REGULATORY SYSTEM that modifies motor acts initiated by other regions of the CNS

Deficits associated with lesions of the cerebellum can be grouped into 3 main classes:

Disturbances of $\left\{ \begin{array}{l} \text{Synergy} = \text{Asynergia} \\ \text{equilibrium} \\ \text{tone} \end{array} \right.$

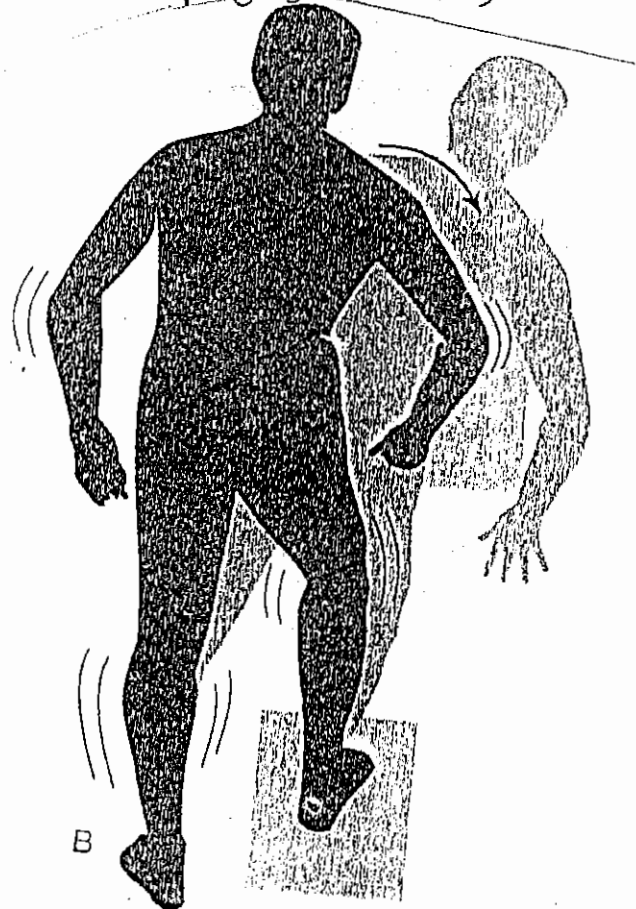
* Asynergia or impaired coordination results from errors in RATE, FORCE, RANGE & DIRECTION of movements. Asynergia can manifest itself in a number of ways

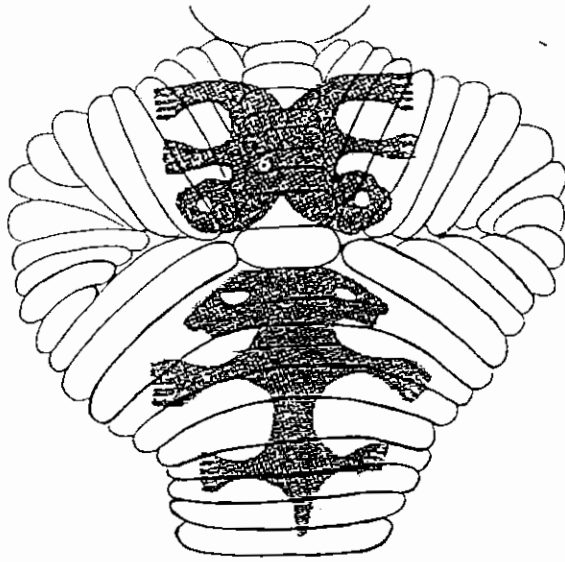
* Lesion of vestibulocerebellum → ① Disturbance of equilibrium (inability to maintain upright posture)

⊕ ② staggering ataxic gait with a tendency to fall toward the side of the lesion

⊕ ③ NYSTAGMUS (spontaneous oscillating eye movements in which the eyes move slowly in one direction and rapidly in the other (jerky eye movement when fixing the gaze on an object) -

- nystagmus reflects asynergia of the extrinsic eye muscles





Topographic representation of the body in the cerebellum

There are two separate representations. In the anterior lobe the body is represented upside down, while it is erect in the posterior lobe. The axial portions of the body lie in the vermis (Fig. 17.68) whereas the limb and facial regions lie in the intermediate zones. Obviously, the lateral zones would have no topographic representations of the body as these areas have different functions, i.e. planning and co-ordination of sequential patterns of muscular activity.

The Cerebellum is called the Silent area!!
 comment → Stimulation of cerebellum does not give rise to any Sensation & causes almost no motor movement



Reeling of trunk from side to side

Stands on wide base

Flocculonodular lobe syndrome: Truncal ataxia Standing on wide base and reeling from side to

Often
 The condition is seen in young children with → medulloblastoma arising in the roof of the 4th ventricle

In severe cases it is impossible for the patient to sit or stand without falling

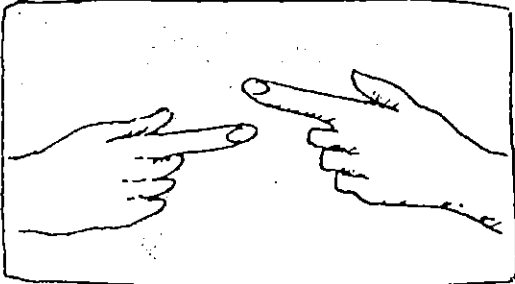
The patient has no control over the axial muscles & hence attempts to walk on a wide base with the trunk constantly reeling & swaying

Lesions of the spinocerebellum & cerebrocerebellum are difficult to differentiate → There is difficulty in initiation & execution & termination of movement.

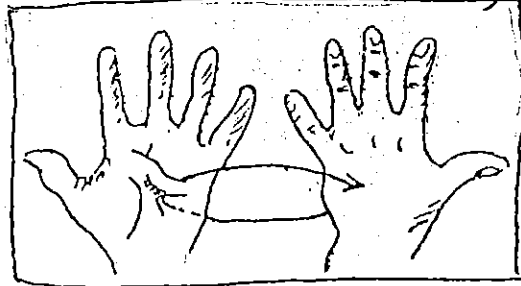
The clumsy movement is called **cerebellar ataxia**

18

(Signs of spinocerebellar and cerebrocerebellar injury)



Substani



Substani

DYSMETRIA → hand overshoots mark (errors in direction & force of movements).
 - limb overshoot → hypermetria
 - " under " → hypometria

DYSDIADOCHOKINESIA → difficulty in performing rapid alternating movements e.g. supination & pronation

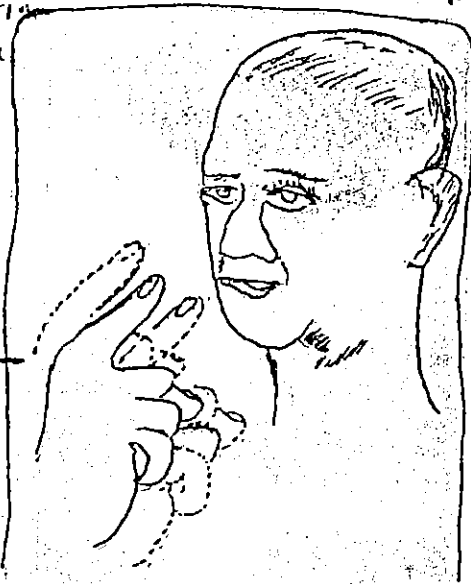
evident during purposeful movement

- worst at the end of movement
- * absent during rest

* Mostly follow a lesion involving the S.C.P

INTENTION TREMOR
 e.g. finger-nose test

* due to defective feedback control from cerebellum on cortically-initiated movement.



Substani 1985

Dysmetria & **Dysdiadochokinesia** ⇒ are the result of absence of proper TIMING in initiation and termination

of movements e.g. a delay in the termination of movements produced by a delay in the intervention of the antagonistic muscle to check the movements results in dysmetria - and overshooting. A delay in initiation of each successive movement will lead to dysdiadochokinesia

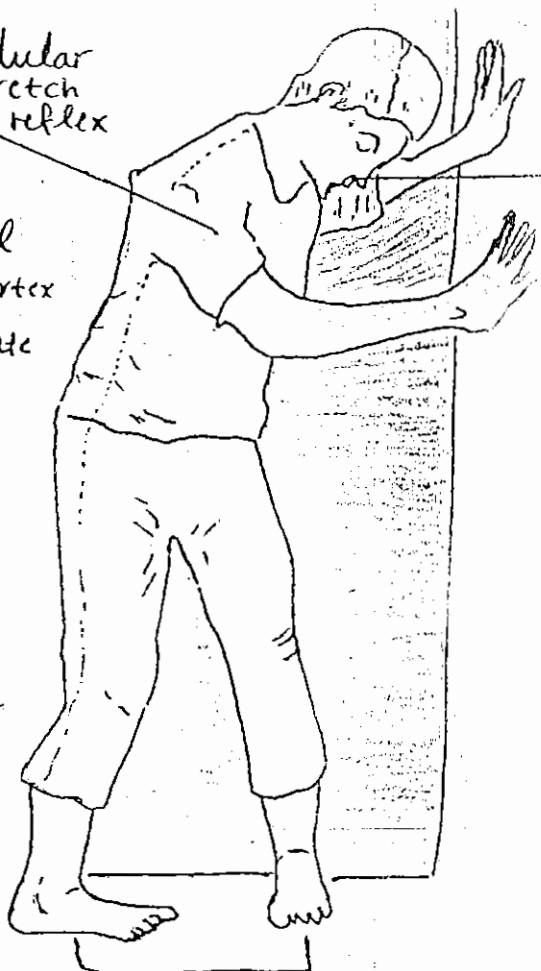
* Remember that intention tremor appears during movement as a result of cerebellar disease while rest tremor (Pill-rolling, Nodding) is the result of disease in the basal ganglia and related nuclei

HYPOTONIA ⊕ Pendular stretch reflex

insufficient signals are sent via the dentato thalamo cortical system to the motor cortex resulting in inappropriate control of γ -neurons via the descending motor pathways

A deficiency in tonic stretch reflex

hypotonia ⊕ Pendular jerk



SPEECH
↓
DYSARTHRIA

Speech is slurred & explosive with a telegram - staccato space (pauses in the wrong places)
* is the result of the incoordination of the muscles used in speaking. i.e. ataxia of respiratory & laryngeal muscles

GAIT → Wide-based & Unsteady (Sailor's walk)

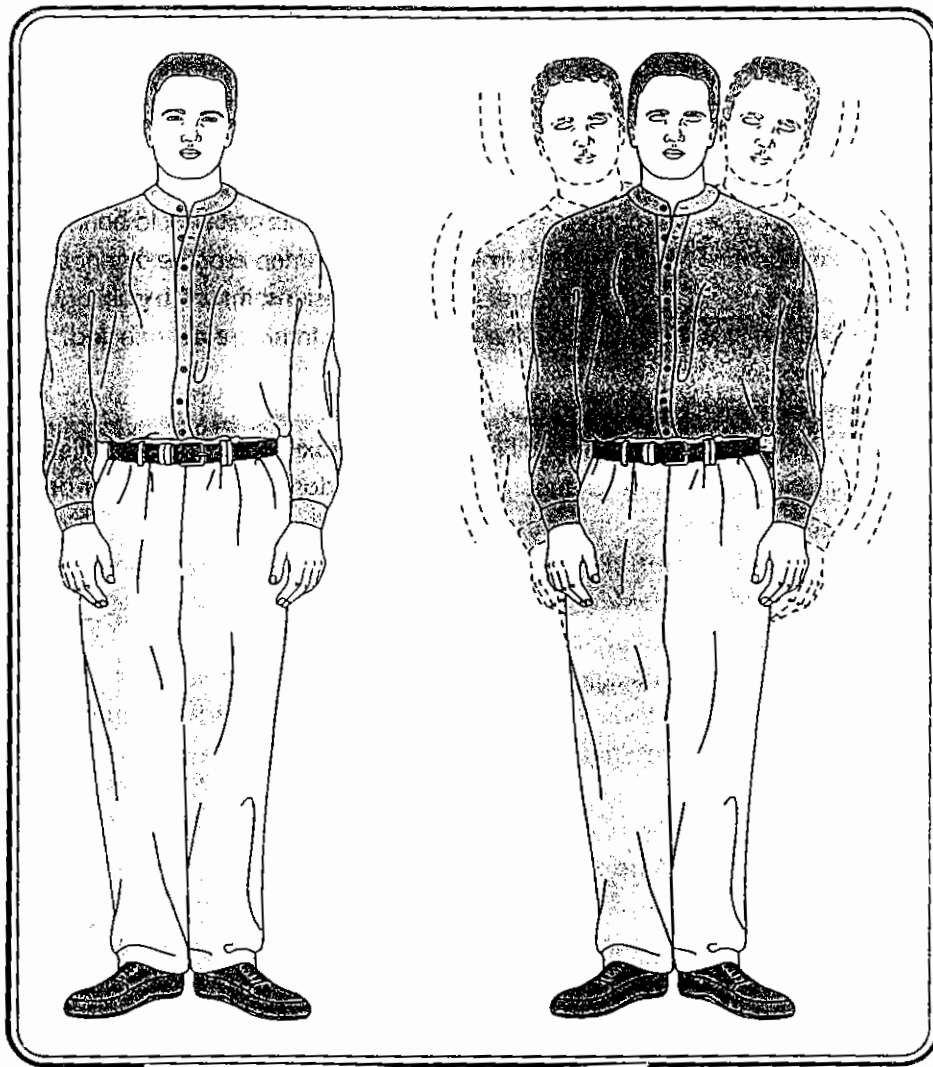
Pendular reflexes & hypotonia are related signs → they reflect REDUCED FACILITATION of γ -motoneurons → muscle tone is reduced because the decreased facilitation of the γ -motoneurons is reflected in a decreased facilitation of the α -motoneurons → with less resting muscle tone → less resistance to passive movement after the initial stretch reflex contraction i.e. the leg swings back & forth like a pendulum because of its own mass & not because of active muscle contraction

The term ataxia is used clinically for incoordination

of gait مشية غير مستقيمة which appears as Staggering مشية غير مستقيمة

(a) Sensory ataxia → caused by disease of the dorsal column of spinal cord. The patient staggers because he cannot perceive the position or movement of his legs, in other words he lacks conscious proprioception → to compensate he uses visual guidance

(b) Cerebellar ataxia → The patient staggers because the cerebellum is unable to coordinate the muscles of walking e.g. alcoholic intoxication. Visual guidance cannot compensate



~~Handwritten scribble~~
20

Romberg's test

To perform Romberg's test, ask the patient to stand with his or her feet together and assess his or her stability. Next, ask the patient to close his or her eyes, making sure that you will be able to support him or her if he or she falls (Fig. 12.8).

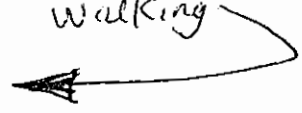
Dr. Surotami

Vestibular
cerebellar
Ataxia ← Sensory

↓ ↓ ↓ ↓
Patients with cerebellar or vestibular lesions are usually ataxic on a narrow base with their eyes open. Their ataxia might get marginally worse when the eyes are closed. Patients with proprioceptive sensory loss ↓
(patients with sensory ataxia → lesion in dorsal column - medial Lemniscus system)

might be slightly ataxic on a narrow base with their eyes open, but they fall when they close their eyes (positive Romberg's test).

Remember that in Sensory ataxia the patient staggers because he cannot perceive the position or movements of his legs (he lacks conscious proprioception). In cerebellar ataxia the patient staggers because the cerebellum is unable to coordinate the muscles of walking.
* He cannot compensate by visual guidance



Damping function of the cerebellum

22

↓
Essentially all voluntary movements being performed develop MOMENTUM (force keeping it moving) which would cause OVERSHOOTING of the movements beyond the intended point (i.e. pendular movement)

⇓
To overcome this → the cerebellum sends appropriate signals which stop the movement at the required point and prevent overshooting

↓
If the cerebellum is damaged → overshooting occurs → The cerebral cortex recognizes the overshooting and initiates a movement in the OPPOSITE direction to bring the moving part, for example the arm, to the intended point

⇓
But due to its momentum → the arm again overshoots and correcting signals are again sent from the cortex

↓
Thus the arm Oscillates beyond the point of intention several times before it settles on the intended point

⇓
This forms the basis of the kinetic or intention tremors of the ~~spinal~~^{spinal} cerebellar & cerebro-cerebellar syndromes

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Anterior lobe syndrome

Commonest cause → malnutrition accompanying

CHRONIC ALCOHOLISM

→ results in damaged to Purkinje neurons

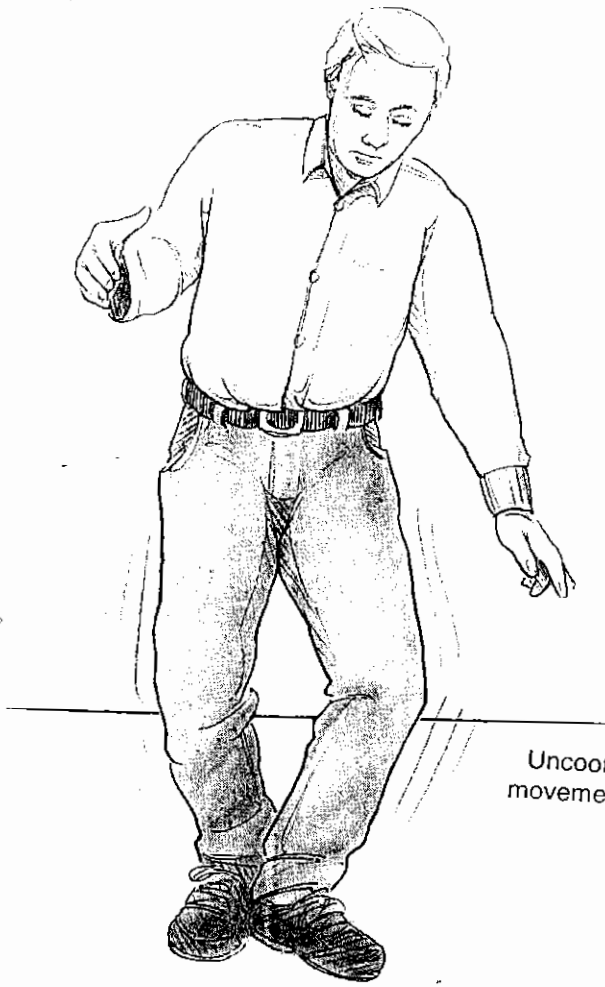
→ Patients suffer the loss of coordination chiefly in the lower limb

→ have marked gait instability

→ walks as if drunk

→ heel-shin test (sliding the heel of one foot smoothly down the shin of the other leg → extremely difficult)

→ If degeneration progresses posteriorly → upper limbs & speech may also be affected



Uncoordinated, clumsy movements of lower limbs

Anterior lobe syndrome: Gait ataxia. Clumsy movements of lower limbs.

Bustami

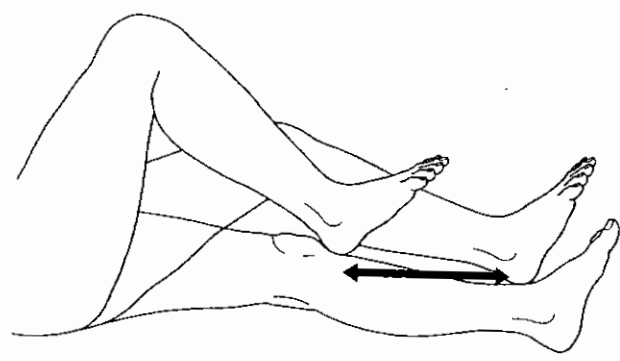


Fig. 8.50 Performing the heel-shin test with the right leg.

Chronic alcoholic

Marked gait instability

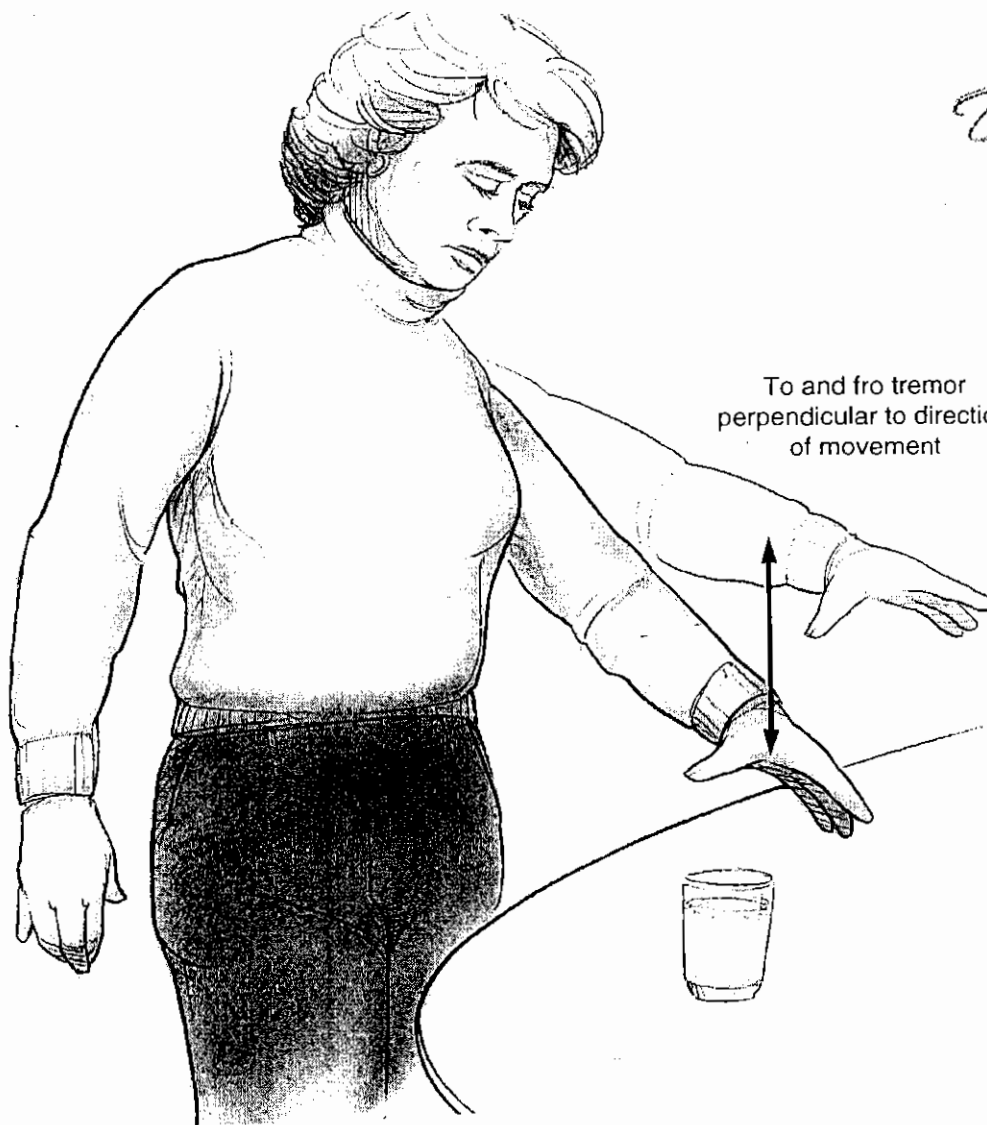
walks as if drunk

may show intention tremors

may show staccato speech

} late signs

Burhan



Posterior lobe syndrome (neocerebellar syndrome)

commonly results from CVA tumours, trauma or degenerative disease

manifested by loss of coordination of voluntary movements

→ A T A X I A ⊕ decreased muscle tone (in acute lesions)

- Rate Range Force direction of movements are ABNORMAL

The ataxic patient is unable to direct the limb to a target without its progression being interrupted by a swaying to & fro movement that is perpendicular to the movement → This is referred to as intention tremor (absent at rest)

- other manifestations of Post-lobe syndrome

- dysmetria
- dysidiadochokinesia
- speech disturbance