

## CHAPTER 10

### THE CEREBELLUM

Anatomically, the cerebellum (CB) consists of 3 lobes separated by 2 deep transverse fissures (a) A small anterior lobe (b) A large posterior lobe (c) A *flocculonodular lobe* (located posteroinferiorly). The anterior and posterior lobes on either side constitute 2 *large hemispheres*, which are separated by a narrow band called the *vermis* (figure 59 A).

3 pairs of peduncles connect the cerebellum to the brain stem (the superior peduncle to the midbrain, the middle peduncle to the pons, and the inferior peduncle to the medulla oblongata)

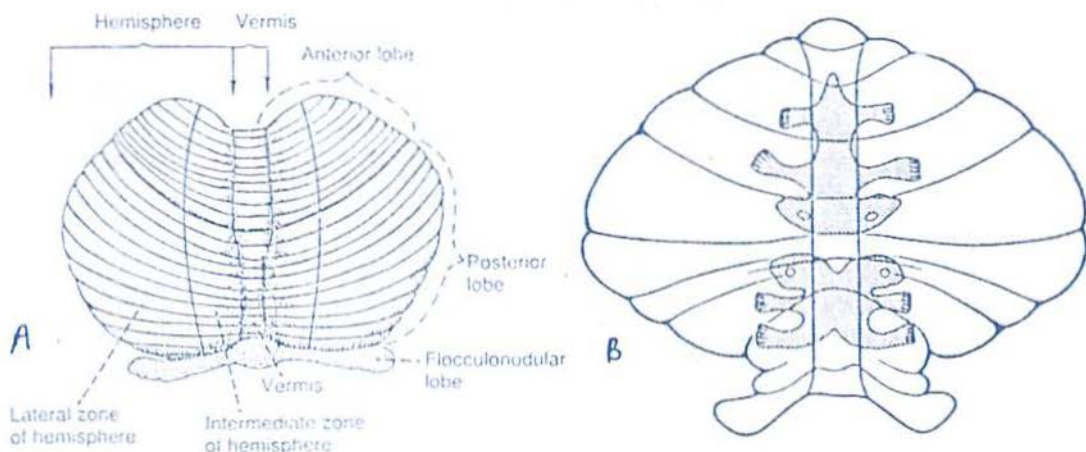
#### FUNCTIONAL (PHYSIOLOGICAL) DIVISIONS OF THE CB

From the functional point of view, the anterior and posterior lobes are organized along their *longitudinal axes*, and the CB is divided into 3 parts :

(1) **Vestibulocerebellum (= archicerebellum)** : This is the oldest part of the CB, and it consists mainly of the *flocculonodular lobe*.

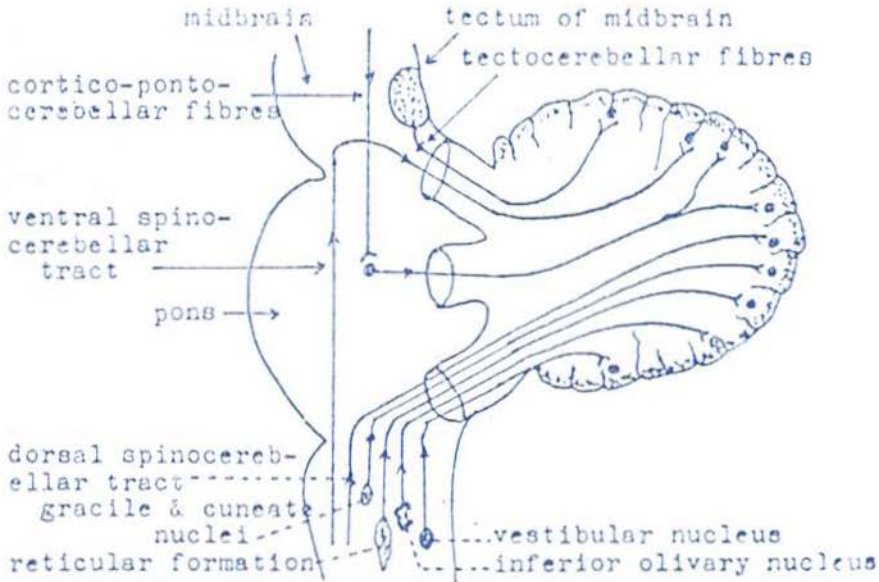
(2) **Spinocerebellum (=Intermediate or paleo-cerebellum)** : This consists of the intermediate zones of the 2 hemispheres and most of the vermis.

(3) **Cerebrocerebellum (= Lateral or neo-cerebellum)** : This is the newest part of the CB. It consists of the large lateral zones of the 2 hemispheres.



**Figure 59** : (A) Functional parts of the CB (B) Topographical representation.

The various parts of the body are *topographically represented* in the CB. The axial parts of the body lie in the vermal part, while the limbs and facial regions lie in the intermediate zones. Also, the body is represented upright in the posterior lobe and upside down in the anterior lobe (figure 59 B).



**Figure 60** : Afferent (input) nerve fibres to the cerebellum.

## CONNECTIONS OF THE CEREBELLUM

The CB has an external layer of gray matter (= *cerebellar cortex*), and an inner layer of white matter. In the latter, there are 3 deep nuclei (a) *Dentate nucleus* laterally (b) *Fastigial nucleus* medially (c) *Interpositus nucleus* (formed of the *globose* and *emboliform nuclei*) between the other 2 nuclei.

Both the afferent and efferent connections of the CB pass through the 3 *cerebellar peduncles* (= *CPs*). The afferent nerve fibres relay first at the *cerebellar cortex*, then the latter discharges to the *deep nuclei* from which the efferent nerve fibres originate and leave the CB (figure 63).

### AFFERENT (INPUT) NERVE FIBRES TO THE CB

The CB receives both *sensory and motor informations* as follows (figure 60)

(A) Through the superior CP, the CB receives fibres from :

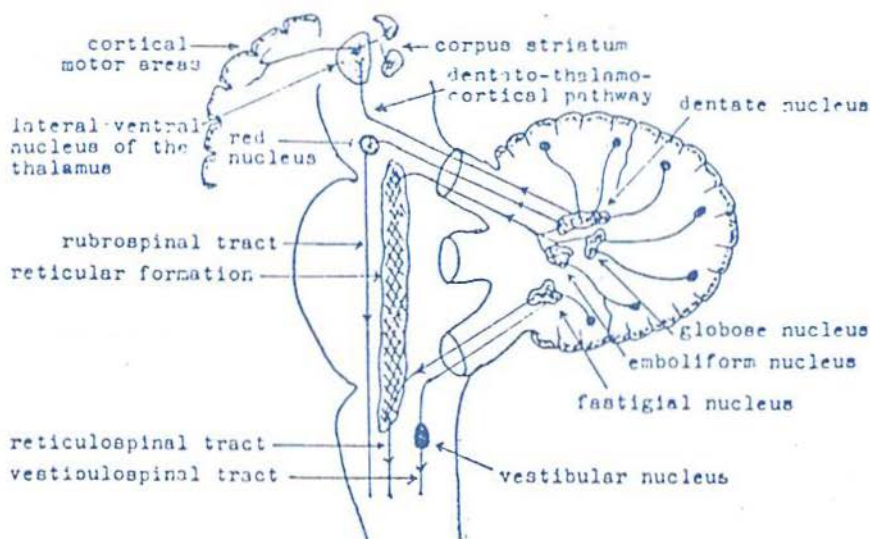
1. The *tectum of the midbrain* (= the superior and inferior colliculi), which transmits visual and auditory signals to the CB.
2. The *ventral spinocerebellar tract*, which terminates in the *spinocerebellum* and informs about the signals that reach the spinal motor neurons from the cortical motor areas (= *efferece copy*, page 110).

(B) Through the middle CP, the CB receives few fibres from the reticular formation, but mainly fibres from the *contralateral motor areas of the cerebral cortex* via the *cortico-ponto-cerebellar pathway* (figure 62).

(C) Through the inferior CP, the CB receives fibres from :

1. The *inferior olivary nucleus* (to all parts of the CB).

2. *The vestibular apparatus* (both directly and via the vestibular nuclei) mainly to the flocculonodular lobe.
3. *The reticular formation* (mainly to the vermis)
4. *The dorsal spinocerebellar tract*, which transmits signals from *proprioceptors* that inform the CB about performance of movements (page 110).
5. *The gracile and cuneate nuclei* : The fibres that arise from these nuclei are called *external arcuate fibres* (page 25) and they also transmit proprioceptive signals to the CB.



**Figure 61** : Efferent (output) nerve fibres from the cerebellum.

### **EFFERENT (OUTPUT) NERVE FIBRES FROM THE CB**

There are 3 efferent pathways from the 3 parts of the CB (figure 61) :

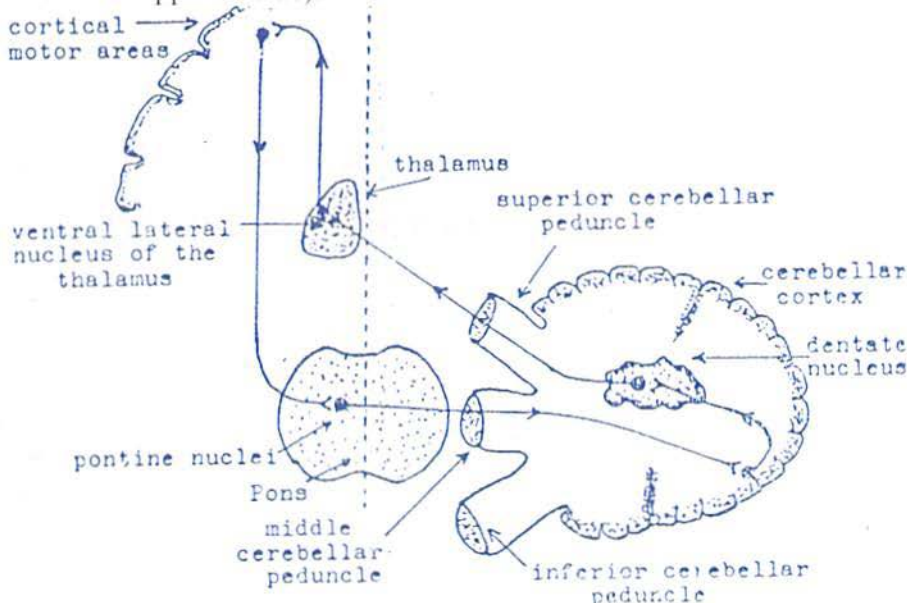
(A) **From the vestibulocerebellum** : Fibres from this part relay at the *fastigial nucleus*, from which efferent fibres arise and pass through the *inferior CP* to the *vestibular nuclei and reticular formation* (then to the spinal motor neurons via the *vestibulospinal and reticulospinal tracts*).

(B) **From the spinocerebellum** : Fibres from this part relay at the *nucleus interpositus*, from which efferent fibres arise and pass through the *superior CP* to (1) *The opposite ventrolateral thalamic nucleus*, then to the *opposite cortical motor areas* (2) *The red nucleus*, then to the spinal cord via the rubrospinal tract (3) *The reticular formation* in the upper part of the brain stem, then to the spinal cord via the reticulospinal tract.

(C) **From the cerebrocerebellum** : Fibres from this part relay at the *dentate nucleus*, from which efferent fibres arise and pass through the *superior CP* to the *opposite ventrolateral thalamic nucleus*, then to the opposite cortical motor areas (= *cerebello-dentato-thalamo-cortical pathway*).

\*\* Each cerebellar hemisphere is connected to the **contralateral cerebral cortex** by both afferent and efferent fibres, which constitute a **neuronal circuit** that starts and ends in the cerebral cortex. This circuit is called the **cortico-ponto-cerebello-dentato-thalamo-cortical circuit** (figure 62).

\*\* Since each cerebellar hemisphere controls the **contralateral cortical motor areas**, then it is clear that the cerebellum exerts its effects mostly **on the same side of the body** (because almost all fibres of the pyramidal tract cross to the opposite side).



**Figure 62 :** The cortico-ponto-cerebello-dentato-thalamo-cortical circuit.

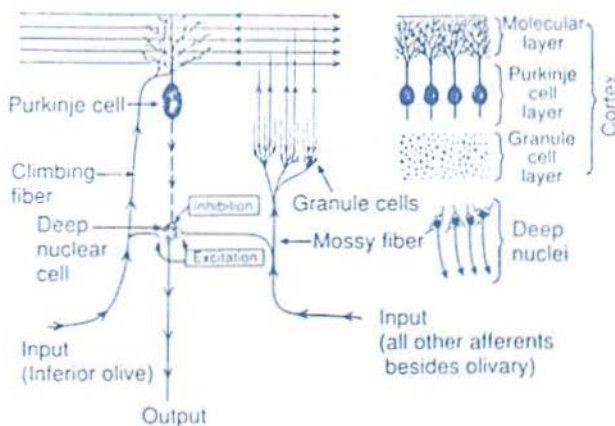
## STRUCTURE OF THE CEREBELLAR CORTEX AND ITS NEURONAL CIRCUITS

The cerebellar cortex (figure 63) is formed of 3 layers (1) **Molecular layer** that contains *parallel interconnecting fibres* as well as 2 types of cells called *basket and stellate cells* (not shown in figure 63) (2) **Purkinje cell layer (PCs)**, the axons of which are *the only fibres that leave the cerebellar cortex* (3) **Granule cell layer** that contains *granular cells*.

The afferent fibres entering the CB are divided into 2 groups :

(A) **Climbing fibres :** These are afferent fibres from the *inferior olivary nucleus*. They excite both the *deep nuclear cells (DNCs)* as well as the PCs.

(B) **Mossy fibres :** These include *all other afferent fibres* that enter the CB + some fibres from the inferior olivary nucleus. They excite both the DNCs and the *granule cells*, the axons of which then *excite the PCs as well as the basket & stellate cells*. The latter 2 cells cause *lateral inhibition of the adjacent PCs* which *sharpens the output signals from the CB* (page 50)



**Figure 63** : The cerebellar cortex and the climbing and mossy fibre circuits.

### FEATURES OF THE CEREBELLAR NEURONAL CIRCUITS

(1) The inhibitory neurons in the CB release **GABA** while the excitatory neurons release **glutamate**.

(2) *The PCs continuously fire inhibitory signals to the DNCs.* However, the excitatory effect of the climbing and mossy fibres on the DNCs normally predominates, so during rest the *DNCs continuously fire excitatory signals.*

(3) The circuits contain *no reverberators*, so normally there is *no afterdischarge and the effects of cerebellar stimulation are transient.*

(4) The mossy fibre circuit *sharpens the input signals to the CB through lateral inhibition of the adjacent PCs* (see above).

### Functions of the mossy fibre circuit

This circuit helps **precise execution of voluntary movements** as follows : A *copy* of the signals discharged from the cortex to perform a certain movement is conducted to the CB via the pontine mossy fibres (the *cortico-ponto-cerebellar pathway*). These signals stimulate the DNCs, which discharge excitatory "**turn on**" signals that help *initiation of the movement*. At the end of the planned movement, the PCs would have been excited by the granule cells, so they send *inhibitory "turn off" signals* to the DNCs, which thus stop discharging. This mechanism has been called **negative feed-forward inhibition**, and it leads to relaxation of the muscles (which helps *termination of the movement without overshooting or oscillation*).

### Functions of the climbing fibre circuit

This circuit is important for *learning the CB to perform new patterns of movement*. The *inferior olivary nucleus* receives informations about (1) the

intended movement (from the motor cortex) (2) the performed movement (from the contracting muscles). It *compares both informations*, and if there was **mismatch** (which usually occurs when a new pattern of movement is performed for the first time), its firing rate is modified leading to a *change in the sensitivity of the PCs*. If the new movement is repeated over a period of time, such change in the sensitivity of the PCs (plus other possible cerebellar processes) will learn the CB to perform such movement coordinately.

## FUNCTIONS OF THE CEREBELLUM

The cerebellum is concerned with *subconscious control of motor activity*. Its functions as well as the involved parts include the following :

### (A) CONTROL OF EQUILIBRIUM AND POSTURAL MOVEMENTS

This is the function of the **vestibulocerebellum**. It receives information from the *vestibular apparatus*, then through the *fastigial nucleus*, it discharges to the brain stem, and through the vestibulospinal and reticulospinal tracts it controls equilibrium and postural movements by affecting the activity of the *axial muscles* (= trunk and girdle muscles).

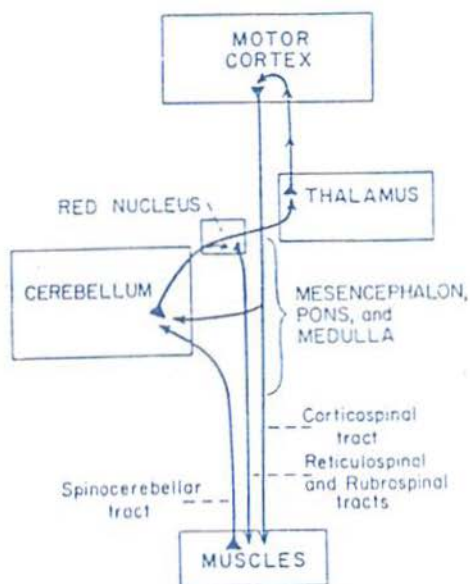
**Trunk ataxia** : This results from lesions of the vestibulocerebellum. It is characterized by equilibrium disturbances (the patient sways on standing, he cannot maintain the erect posture and walks by a staggering or drunken gait)

### (B) CONTROL OF THE STRETCH REFLEX

The *cerebrocerebellum* exerts a *facilitatory effect* on the stretch reflex and increases the muscle tone (page 71), while the *spinocerebellum* exerts an *inhibitory effect* (page 72). However, normally *the facilitatory effect predominates* so *cerebellar disease often results in hypotonia* (see below).

### THE CEREBELLAR STRETCH REFLEX

Impulses from the muscle spindles are transmitted to the cerebrocerebellum (via the *spinocerebellar tracts*) which responds by discharging signals via the descending extrapyramidal tracts (specially the reticulospinal tracts) which stimulate the *spinal motor neurons* resulting in muscle contraction. Such *cerebellar stretch reflex* is *more complex* than the spinal stretch reflex and *its feedback time is longer*. It is important for (a) Many postural adjustments (b) The *damping function* of the CB (page 110) specially when carrying heavy loads (which is sometimes called the *load reflex*).



**Figure 64 :** The comparing function for control of voluntary movements.

### (C) CONTROL OF VOLUNTARY MOVEMENTS

#### (1) ROLE OF THE SPINOCEREBELLUM

(1) **Comparing function :** When a movement is performed, the spinocerebellum receives 2 informations (figure 64) (a) *Signals from the motor cortex* (via the cortico-ponto-cerebellar pathway) that inform about the intended plan of movement (= *afference copy*) (b) *Feedback signals from the periphery* via the spinocerebellar tracts. The ventral tract informs about the cortical signals that reach the spinal motor neurons (= *afference copy*, page 105) while the dorsal tract informs about performance of movements (page 106).

The spinocerebellum *compares all these informations*, and if there is an error in performance, it sends *signals from the nucleus interpositus* to the *motor cortex, red nucleus and reticular formation*. These areas send corrective signals that control the activity of the spinal motor neurons, resulting in adjustment of the performance to match the intention, which leads to coordinated movements specially of the hands and fingers. Such function is *aided by the climbing fibre circuit* (page 108).

(2) **Damping function :** Almost all movements are *pendular* (due to momentum) so they have a tendency to overshoot. However, the spinocerebellum prevents this by *subconscious signals* that stop the movement at the intended point. Such damping effect is produced by contraction of the antagonistic muscles through the *cerebellar stretch reflex* (page 109).

If the CB was damaged, the *cerebral cortex can consciously recognize the overshoot* and it then initiates a movement in the reverse direction by contraction of the antagonistic muscles. However, this movement also overshoots, and is corrected again by overshooting cortical signals. This process is repeated, so the arm oscillates back and forth for several cycles before it finally settles at the intended point (= *kinetic, intention or action tremor*).

(3) **Coordination of ballistic movements** : Ballistic movements are those which occur very rapidly (e.g. the fingers during typing, and the eyes during reading) and for this reason, the above comparing coordinating function cannot operate. They are *pre-planned* to go specific distances then stop, and this is also coordinated by the spinocerebellum aided by the *turn on / turn off signals of the mossy fibre circuit* (page 108).

## (2) ROLE OF THE CEREBROCEREBELLUM

(1) **Planning of movements** : The cerebrocerebellum is informed about the desired movement *before it starts* (via the cortico-ponto-cerebellar pathway). The basal ganglia receive a similar information (page 100) and both provide the plan of execution of the movement.

(2) **Prediction of movements** : The cerebrocerebellum *predicts the next movement at the same time a present movement is occurring*. This function is necessary for smooth transition from one movement to the next (thus joining the sequential movements and preventing decomposition).

(3) **Timing of movements** : The cerebrocerebellum also provides appropriate timing for each succeeding movement. This function *determines when the next movement should begin*, so its absence causes the succeeding movement to begin too early or too late, resulting in incoordination of the movement (specially rapid movements e.g. writing, running and talking).

## Extramotor predictive function of the cerebrocerebellum

The cerebrocerebellum can predict events *other than movements*, specially the rates of progression of auditory and visual stimuli e.g. a person can predict from a changing visual scene how rapidly he is approaching an object.

## THE NEOCEREBELLAR SYNDROME

The manifestations of neocerebellar disease are **ipsilateral**, and include :

(A) **Hypotonia** : This is due to loss of the facilitatory effect on the stretch effect, and is associated with a *pendular knee jerk* (page 69).

(B) **Asthenia** (= **muscle weakness**) : This is due to difficulty in initiation of muscle contraction caused by loss of the function of the mossy fibre circuit (page 108).



(C) **Motor ataxia** : This is incoordination of voluntary movements, specially the rapid movements. Its manifestations include the following :

(1) **Dysmetria** : This is inability to control the distance of a motor act, which may either overshoot the intended point (= *hypermetria* or *pastpointing*) or stops short before it (= *hypometria*).

(2) **Kinetic (intention or terminal) tremor** : This is an oscillatory movement that appears on performing movements (specially at their end) but is absent at rest. It is due to *cortical correction of the overshoot* (page 111).

(3) **Rebound phenomenon** : This is overshooting of a limb when a resistance to its movement is suddenly removed. It is well demonstrated by the *arm pulling test* (figure 65 a), in which the patient may hit his face by his forearm after release of the resistance that is exerted by the examiner.

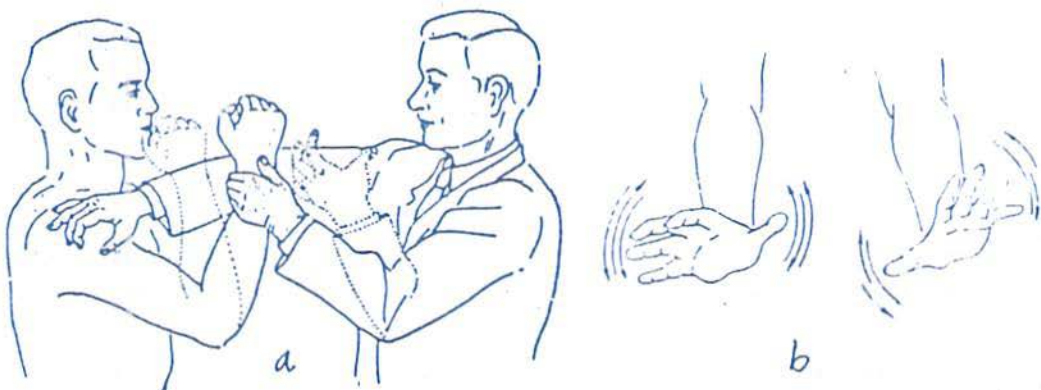
(4) **Asynergia** : This is loss of the harmony between the various groups of muscles involved in performance of voluntary movements, which results in decreased ability to perform smooth and coordinated movements.

(5) **Failure of progression of movements** : This is manifested by (a) *Adiadochokinesia* i.e. inability to perform rapidly alternating opposite movements e.g. repeated pronation and supination of the hands (figure 65 b) (b) *Decomposition (= fragmentation) of movements* i.e. performing the movement in steps and not as a continuous act.

(6) **Dysarthria** : This is difficulty in producing clear speech. The syllables may be too long or too short (resulting in jumbled vocalization), and speech may also become *staccato* or *scanning* (i.e. cut off into separate syllables).

(7) **Nystagmus** : This is a tremor of the eyeballs that occurs on fixing the eye at an object placed at one side of the head (page 144).

(8) **Staggering (or drunken) gait** : The patient walks unsteadily and on a wide base (= *zigzag-like gait*) in a drunken (swaying) manner, and tends to fall on the diseased side.



**Figure 65** : (a) Arm pulling test. (b) Test for adiadochokinesia.

### TYPES AND CAUSES OF TREMORS

1. **Static tremor** : This occurs in *Parkinsonism*. It is present *during rest* and is associated with *hypertonia*.
2. **Kinetic tremor** : This occurs in *neocerebellar disease*. It is specially present at the *end of movements* and is associated with *hypotonia*.
3. **Fine tremor** (occurs in *hyperthyroidism*).
4. **Flapping tremor** (occurs in *liver failure*).
5. **Senile tremor** (occurs in *old age*).
6. **Emotional and psychological tremors**.
7. **Toxic tremors** (e.g. that occurring in *chronic alcohol drinkers*).
8. **Familial and hereditary tremors**.

### ATAXIA

This means *incoordination of voluntary movements*, and it is either *sensory or motor (sometimes mixed in the thalamic syndrome, page 90)*.

**(A) Sensory ataxia** : This occurs as a result of lesions (or diseases) of the proprioceptive sensory pathways, commonly in the dorsal column of the spinal cord (= the gracile and cuneate tracts). It specially occurs in (1) *Tabes dorsalis* (page 31) (2) *Subacute combined degeneration* (page 33) (3) *Polyneuritis* (page 32) (4) *Thalamic syndrome* (page 90) (5) *Chronic alcoholism* (6) *Friedreich's ataxia* (a hereditary disease that causes degeneration of the dorsal and lateral columns of the spinal cord).

The **manifestations (features) of sensory ataxia include** (a) Loss of dorsal column sensations (page 24) (b) *+ve Romberg's sign* (page 32) (c) Inability to walk in the dark (d) Difficulty in walking at day light. There is a *stamping gait* (= *high steppage gait* with a slap when the foot reaches the floor), and the patient walks at a broad base and *always looks at his feet*.

*The following table shows the differences between sensory and motor ataxia*

	SENSORY ATAXIA	MOTOR ATAXIA
<b>Most common cause</b>	Tabes dorsalis	Neocerebellar disease
<b>Gait</b>	High steppage (stamping gait)	Staggering (drunken gait)
<b>Romberg's sign</b>	Positive	Negative
<b>Effect of vision</b>	Corrected by vision	Not affected by vision
<b>Deep sensations</b>	Impaired or lost	Normal
<b>Tremors</b>	Absent	Kinetic tremors present
<b>Nystagmus</b>	Absent	Present
<b>Speech</b>	Normal	Scanning or staccato