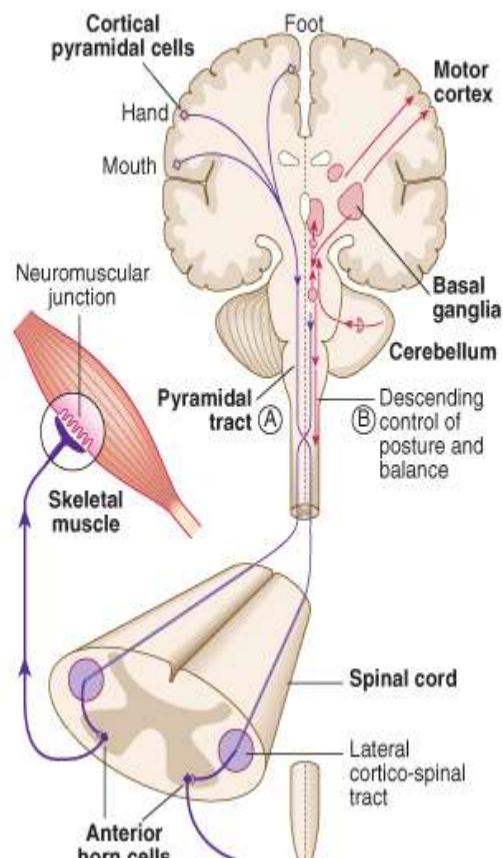


Motor Control Systems

CORTICOSPINAL OR PYRAMIDAL SYSTEM

The corticospinal tracts originate in neurones of the fifth cortical layer and terminate at motor nuclei of cranial nerves or at the lower border of L1 in spinal cord [anterior horn cells]. The nerve pathways of particular clinical significance) congregate in the internal capsule and cross in the medulla (decussation of the pyramids), passing to the contralateral halves of the spinal cord as the lateral corticospinal tracts. This is the *pyramidal system*, disease of which causes upper motor neuron (UMN) lesions. This system enables *purposive, skilled, strong* and *organized* movement to take place. Defective function is recognized by a distinct pattern of loss of skilled voluntary movement, spasticity and reflex change. This is seen, for example, in a hemiparesis or hemiplegic

THE MOTOR SYSTEM



1-pyramidal drift

Normally, the outstretched upper limbs are held symmetrically, even when the eyes are closed. With a pyramidal (UMN) lesion, when both upper limbs are held outstretched, palms upmost, the affected limb drifts downwards and medially. The forearm tends to pronate and the fingers flex slightly. This sign is often first to occur, sometimes before weakness or reflex changes become obvious

2-Weakness and loss of skilled movement

unilateral pyramidal (UMN) lesion above the decussating in the medulla (e.g. an internal capsule infarct) causes weakness of the opposite limbs, i.e. contra lateral hemi paresis.. In the upper limb flexors remain stronger than extensors, while in the lower limb extensors remain stronger than flexors. In addition to weakness, there is loss of skilled movement. For example, fine finger and toe control diminishes. Muscle wasting (except from disuse) is not a feature of pyramidal lesions. Muscles remain normally excitable electrically.

3-Increase in tone [spasticity]

acute lesion of one pyramidal tract (e.g. the internal capsule stroke) causes initially **flaccid paralysis**, and loss of tendon reflexes.

Increase in tone follows within several days owing to loss of the inhibitory effect of the corticospinal pathways and an increase in spinal reflex activity. The pattern is characterized by *changing* resistance to passive movement - the sudden clasp-knife effect. The relevant tendon reflexes become exaggerated and clonus evident.

4-Changes in superficial reflexes

The normal flexor plantar response becomes extensor (a positive Babinski). The stimulus should be unpleasant (an orange-stick is the correct instrument). An extensor plantar is certain when dorsiflexion of the great toe is accompanied by fanning (abduction) of adjacent toes. Abdominal (and cremasteric reflexes) are abolished on the affected side.

EXTRAPYRAMIDAL SYSTEM

The extrapyramidal system facilitates fast, fluid movements that the corticospinal system has generated. Defective function is recognized usually by slowness (bradykinesia), stiffness (rigidity) and/or disorders of movement (rest tremor, chorea and other dyskinesias). Frequently, one sign (e.g. stiffness, tremor or chorea) will predominate.

The extrapyramidal system is a general term without an absolute definition for motor structures of the basal ganglia, i.e. corpus striatum (caudate nucleus + globus pallidus + putamen), subthalamic nucleus, substantia nigra and parts of the thalamus.

In basal ganglia/extrapyramidal disorders, either or both of two features become apparent in the limbs and axial muscles:

reduction in speed, known as bradykinesia (slow movement) or akinesia (no movement), with muscle rigidity

involuntary movements (e.g. tremor, chorea, hemiballismus, athetosis, dystonia).

Lesions of the extrapyramidal system produce an increase in tone, which is not an exaggerated response to stretch but is continuous throughout the range of movement at any speed of stretch ('lead pipe' rigidity). Involuntary movements are also a feature of extrapyramidal lesions

tremor combined with rigidity produces typical 'cogwheel' rigidity.

Rapid movements are slowed and clumsy (bradykinesia).

Extrapyramidal lesions also cause postural instability, precipitating falls.

Chorea, athetosis, ballism and dystonia

Jerky, small-amplitude, purposeless involuntary movements are termed 'chorea' (the Greek for 'dance').; they suggest disease in the caudate nucleus

More dramatic ballistic movements of the limbs usually occur unilaterally (hemiballismus) in vascular lesions of the subthalamic structures.

Slower writhing movements of the limbs are called athetosis. These are often combined with chorea (and have a similar list of causes) and are then termed 'choreo-athetoid' **movements.**

CEREBELLUM

The *cerebellum* and its connections have a role in coordinating *smooth* movement initiated by the corticospinal system, and in the regulation of balance.

Cerebellar lesions

Expanding mass lesions within the cerebellum

1. obstruct the aqueduct to cause hydrocephalus, with severe pressure headaches, vomiting and papilloedema.
2. Coning of the cerebellar tonsils) through the foramen magnum and respiratory arrest occur, often within hours. Rarely,
3. tonic seizures (sudden attacks of limb stiffness) occur.

Lateral cerebellar lobes lesions

- 1-causes disruption of the normal sequence of movements (dyssynergia) on the same side
- 2-The outstretched arm is held still in the early stages of a cerebellar lesion (cf. the drift of a pyramidal lesion) but there is rebound upward overshoot when the limb is pressed downwards and released.
- 3-Gait becomes broad and ataxic; the patient falters towards the lesion, tremor and ataxia.
- 4-Movement is imprecise in direction, in force and in distance (dysmetria). Rapid alternating movements (tapping, clapping or rotary hand movements) are clumsy and disorganized (dysdiadochokinesis). Intention tremor (action tremor, with past-pointing) is seen. Speed of movement is preserved, cf. extrapyramidal disease
- 5-Nystagmus: Coarse horizontal nystagmus develops with lateral cerebellar lobe lesions - the fast component towards the lesion.
- 6-Dysarthria: A halting, jerking dysarthria occurs - the scanning speech of cerebellar lesions (usually bilateral).

Other signs

Titubation - rhythmic head tremor in either to and fro ('yes-yes') movements or rotary ('no-no') movements - also occurs,

Hypotonia (floppy limbs) and depression of reflexes (and slow, pendular reflexes) are also sometimes seen with cerebellar disease

Midline cerebellar lesions

Midline cerebellar vermis lesions have a dramatic effect on trunk and axial musculature-difficulty standing and sitting unsupported, with a rolling, broad, ataxic gait (truncal ataxia).

The LMN is the motor pathway from anterior horn cell (or cranial nerve nucleus) via peripheral nerve to motor endplate

Signs of a lower motor neurone lesion

1) Weakness

2) Wasting

3) Hypotonia

4) Reflex loss

5) Fasciculation

long term effects:

6) Contractures of muscle

7) Trophic changes in skin and nails

Signs of lower motor neurone lesion

These are seen in voluntary muscles, which depend upon an intact nerve supply to produce movement *and* for metabolic integrity. Signs follow rapidly if the LMN is interrupted (). Muscle wasting appears within 3 weeks. Fasciculation (visible twitching) occurs - due to contractions of denervated single motor units. Fibrillation potentials are seen when denervated muscle is sampled electrically

Thank you,,,