conditions are characterized by ***hypertonia mainly in the antigravity muscles***, so the increased resistance to passive movements is ***uni-directional*** (i.e. it occurs during either flexion or extension of the examined limb). It is ***velocity-dependent*** (i.e. it increases with more rapid movements of the limb), of the ***clasp-knife type*** (page 65), and is accompanied by ***exaggerated tendon jerks and frequently also clonus*** (page 69).

Alpha rigidity (= rigidity clinically) refers to muscle stiffness that occurs as a result of ***enhanced activity of the alpha motor neurons*** e.g. in ***Parkinsonism***. These conditions are characterized by ***hypertonia in all muscles,*** so the increased resistance to passive movements is ***bi-directional*** . ***It is not velocity-dependent,*** not necessarily accompanied by hyper-reflexia, and is felt throughout the whole length of the movement (= ***lead-pipe rigidity***) or it may be interrupted (= ***cogwheel***) rigidity (page 102).

The following table differentiates both types of rigidity :

	Gamma rigidity	Alpha rigidity
Cause	Increased gamma discharge	Increased alpha discharge
Muscles affected	Antigravity muscles	All muscles
Resistance to movement	Uni-directional	Bi-directional
Type of rigidity	Clasp-knife	Lead-pipe or cogwheel
Effect of velocity	Increases with velocity	Not velocity-dependent
Tendon jerks	Exaggerated and clonus may also be present	Not necessarily exaggerated
Common diseases	Upper motor neuron lesion and decerebrate rigidity	Parkinsonism

THE DESCENDING (MOTOR) TRACTS

The descending tracts can be classified into 2 systems known as the ***pyramidal and extrapyramidal systems***. The neurons of these tracts are called ***upper motor neurons,*** and they terminate at the spinal motor neurons which are called the ***lower motor neurons***.

THE PYRAMIDAL SYSTEM

This consists mainly of the ***corticospinal and corticobulbar tracts***.

(1) THE CORTICOSPINAL TRACT (= PYRAMIDAL TRACT)

This tract performs the following functions mainly in the ***opposite side*** :

From the various nuclei in the brain stem, the various extrapyramidal tracts arise and descend in the spinal cord, where they terminate at the spinal motor neurons. Some of the cortically-originating extrapyramidal fibres project ***directly to the red nucleus and reticular formation*** in the brain stem.

PROPERTIES OF THE EXTRAPYRAMIDAL SYSTEM

(1) Its origin is both ***cortical and extracortical***.
(2) Its ***cortical origin is wide***.
(3) Its pathway is ***multineural*** (i.e. contains many synapses).
(4) Its tracts ***do not occupy the medullary pyramids***.
(5) About ***50 % of its fibres cross to the opposite side***.
(6) It functions during ***the first year of life***.
(7) *Some of its tracts are facilitatory* and *some are inhibitory, but the* ***inhibitory tracts predominate***.
(8) Its tracts terminate ***only at the spinal motor neurons*** (*no extrapyramidal tracts terminate at the cranial nerve nuclei*).

The following table summarizes the differences between the pyramidal and extrapyramidal systems :

	PYRAMIDAL SYSTEM	EXTRAPYRAMIDAL SYSTEM
Origin	Cortical only	Cortical (much wider) and extracortical
Tract	Mononeuronal	Multineuronal
Pathway	Direct activation pathway	Indirect activation pathway
Crossing	About 90%	About 50 %
Termination	Cranial nerve nuclei and alpha neurons in the spinal cord	Alpha and gamma neurons in the spinal cord only (not at the cranial nuclei)
Location	Medullary pyramids, and in lateral column of spinal cord mainly	Outside the medullary pyramids, and in lateral and ventral columns of spinal cord
Time of function	Only after the first year of life	During and after the first year of life
Function	Initiates fine skilled voluntary movements and increases the muscle tone	Initiates gross and associated movements, decreases the muscle tone and controls autonomic functions

** **Tlie head muscles are controlled by pyramidal fibres only through the corticobulbar tract**. On the other hand, *the body muscles are controlled*

(1) In the midbrain, the pyramidal fibres occupy the ***middle 3/5 of the cerebral peduncles*** (= ***basis pedunculi or crus cerebri***).

(2) In the pons, the fibres occupy the ***basis pontis***, and are divided into ***bundles*** by the ***transverse pontine fibres***.

(3) In thje upper part of the medulla, the bundles collect in the anterior part forming ***the pyramid***. In the lower medulla, about 80 % of the fibres cross to the opposite side (in the ***motor decussation***) and descend in the lateral column of spinal cord as the **lateral corticospinal tract**, while the uncrossed fibres descend directly in the anterior column of the spinal cord as the **ventral corticospinal tract**. Most fibres of the latter tract gradually cross in the cervical and upper thoracic spinal segments to the opposite side, but ***some fibres terminate at the same side***. A few fibres from the ipsilateral cortex also descend directly, constituting an ***ipsilateral corticospinal tract*** which joins the lateral tract crossing from the opposite side (figure 46).

The uncrossed fibres from the ventral and ipsilateral tracts constitute about 10 % of the total fibres of the pyramidal tract, and they *stimulate the* ***motor neurons that supply the respiratory and abdominal muscles***. Therefore, these muscles receive a ***bilateral upper motor neuron supply,*** and accordingly they are ***not paralyzed in cases of hemiplegia***.

The medial and lateral motor systems

The medial motor system includes *the ventral corticospinal tract and the vestibulospinal and reticulospinal tracts*. These tracts terminate at the ***medial portions of the anterior horns,*** and they control the ***muscles of the trunk*** (= ***axial muscles***) ***and proximal portions of the limbs***. This system is concerned with **(a)** Stabilization of the pelvic and shoulder girdles to maintain posture and equilibrium **(b)** Production of a postural background for performance of fine movements **(c)** Control of gross and automatic movements

The lateral motor system includes the *lateral corticospinal tract and the rubrospinal tract*. These tracts terminate at the ***lateral portions of the anterior horns***, and they control the ***distal muscles of the limbs*** which perform fine discrete (= skilled) movements specially in the fingers and hands.

(2) THE C0RT1COBULBAR TRACT

This tract *originates as the corticospinal tract* and both descend together in the corona radiata, then it occupies ***the genu of the internal capsule***. Its ***fibres cross in the brain stem and terminate at the nuclei of all cranial nerves*** on the opposite side ***except the 1st,2nd and 8th cranial nerve nuclei***. The fibres of this tract constitute ***upper motor neurons***, while the ***cranial nerves themselves are lower motor neurons***

CHAPTER 6

COMMON LESIONS IN THE CNS

(1) UPPER and LOWER MOTOR NEURON LESIONS

An UMNL results from damage of the cortical motor areas or anywhere along the course of their descending tracts, commonly in the ***internal capsule*** due to cerebral hemorrhage or thrombosis. On the other hand, a LMNL results from either damage of the spinal (or cranial) motor neurons by disease (commonly ***poliomyelitis***) or injury of the motor nerves by trauma or disease (e.g. ***polyneuropathy***).

DIFFERENCES BETWEEN UMNL and LMNL

Although both lesions result in paralysis of skeletal muscles, yet each has characteristic manifestations.

(1) Extent of paralysis (widespread in UMNL and localized in LMNL).

(2) Site of paralysis : This is always at the same side in LMNL but it may be on either side in case of UMNL e.g. a hemisection of the spinal cord at the cervical region leads to ipsilateral hemiplegia, while a lesion in the internal capsule leads to contralateral hemiplegia (page 83).

(3) Recovery : UMNL does not recover because the upper motor neurons ***cannot regenerate due to absence of neurolemma***. Conversely, LMNL can recover if it is due to injury of the motor nerves (because these nerves ***can regenerate due to presence of neurolemma***), but they cannot recover if the motor nerve cells themselves are damaged (e.g. in poliomyelitis).

(4) Muscle tone : In LMNL, there is ***hypotonia or atonia*** (i.e. muscle flaccidity) due to interruption of the efferent limb of the stretch reflex.

On the other hand, in UMNL there is ***hypertonia*** i.e. muscle spasticity (= ***decorticate rigidity***) mainly in the ***antigravity muscles***. It is a type of ***gamma rigidity*** that shows the ***lengthening (clasp-knife) reaction*** (page 65). It occurs as a ***release phenomenon*** due to *damage of the extrapyramidal fibres with the pyramidal fibres while passing together in the internal capsule* (page 76), which results in ***reversed supraspinal balance on the gamma motor neurons from inhibition to excitation*** (page 72).

(5) Tendon jerks : These are lost in LMNL and exaggerated in UMNL due to the same causes of hypertonia (see above). Also, ***clonus*** (page 69) ***often appears in UMNL,*** and is the most diagnostic feature of this lesion.

(6) Superficial reflexes :

(a) The *abdominal and cremasteric reflexes are lost in both lesions* due to loss of pyramidal facilitation in UMNL (page 74) and interruption of their efferent limbs in LMNL.

(b) The plantar reflex is lost in LMNL, but is modified in UMNLs and the response becomes ***dorsiflexion of the big toe and fanning of the other toes*** (probably due to interruption of the pyramidal and extrapyramidal fibres respectively). Such response is known as the ***positive Babinski's sign or the plantar extensor reflex*** (figure 49). ***However, this sign may be present in some normal individuals*** (see below).

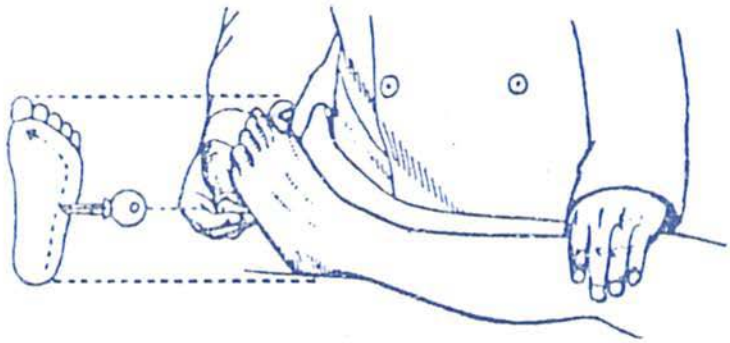

Figure 49 : A positive Babinski's sign.

(7) Tonic neck reflexes : In UMNL, turning the head towards the hemiplegic side leads to extension and abduction of the paralyzed arm, while turning the head towards the healthy side leads to more flexion and adduction of the paralyzed arm (the latter position is the posture usually acquired by the paralyzed upper limb).

(8) Muscle status : Muscle wasting (= atrophy) occurs rapidly and markedly ***in LMNL*** due to degeneration of the motor nerves that supply the muscles. On the other hand, the paralyzed muscles in UMNL are atrophied only after relatively long periods due to disuse (= ***disuse atrophy***).

(9) Response to electric stimulation : Normally, stimulation of skeletal muscles by Faradic (alternating) currents produces tetanus while their stimulation by Galvanic (direct) currents produces contraction only at the make (= closing) and break (= opening) of the circuit, whether the cathodal or the anodal electrode was used for stimulation. Therefore, in the latter condition, there are ***4 contraction states*** **(1) CCC** (= cathodal closing contraction) **(2) COC** (= cathodal opening contraction) **(3) ACC** (= anodal closing contraction) **(4) AOC** (= anodal opening contraction), and the strength of contraction was found to be as follows :

CCC > ACC > AOC > COC

In UMNL, the paralyzed muscles respond normally to both currents and their chronaxies are also normal. On the other hand, ***the response in case of LMNL is altered, and is called the reaction of degeneration*** (see next).

REACTION OF DEGENERATION (RD)

This is an abnormal response of skeletal muscles to electric stimuli that ***occurs in LMNL only***. It is characterized by the following :

(1) The chronaxie of the paralyzed muscles is prolonged.

(2) There is no response to Faradic currents.

(3) There is sluggish (= weak) response to Galvanic currents.

(4) The **ACC becomes stronger than the CCC** (due to unknown causes).

** The RD is one of the manifestations of muscle denervation. The **reaction of denervation** is the diagnostic feature of LMNL, and is characterized by RD, muscle fasciculation and fibrillation (refer to nerve and muscle), as well as by loss of the Hoffmann's reflex (page 63).

** The Babinski's response is *a part of the flexor withdrawal reflex*, and is ***normally inhibited by the pyramidal tracts*** (so it becomes positive in UMNL) However, these tracts *may be non-functioning in some normal subjects, leading to a* ***+ve Babinski s sign in absence of UMNL*** This occurs in :

(1) Infants below one year of age (because the pyramidal tracts are unmyelinated and not completely developed).

(2) Adults during sleep, coma and anesthesia (because the high cortical centres are depressed in these conditions).

The following table shows the differences between UMNL and LMNL :

	UMNL	LMNL
Extent of paralysis	Widespread	Localized
Site of paralysis	Commonly contralateral	Only ipsilateral
Recovery	No recovery	Occurs if AHC are intact
Muscle tone	Hypertonia	Hypotonia or atonia
Tendon jerks	Exaggerated with clonus	Lost
Superficial reflexes	Lost + Babinski's sign	Lost
Tonic neck reflexes	Present	Absent
Muscle status	Normal (disuse atrophy in long-standing cases)	Rapid atrophy
Electric stimulation	Normal response	Reaction of degeneration

(2) EFFECTS OF LESIONS OF THE PYRAMIDAL TRACT AT VARIOUS LEVELS

Lesions of the pyramidal tract cause paralysis of the ***UMNL type below the level of the lesion***. However, the side affected and the extent of paralysis differ according to the site of the lesion as follows :

(1) IN AREA 4 : This usually leads to restricted paralysis in the opposite side e.g. ***monoplegia*** (= paralysis of one limb) because area 4 is widespread and is rarely damaged completely.

(2) IN THE CORONA RADIATA : This leads to ***contralateral monoplegia or hemiplegia*** (= complete paralysis of one half of the body), depending on the extent of the lesion.

(3) IN THE INTERNAL CAPSULE : This leads to ***contralateral hemiplegia*** in most cases because *almost all descending fibres are injured.*

(4) IN THE BRAIN STEM : This leads to ***contralateral hemiplegia plus ipsilateral paralysis of the cranial nerves of the IMNL type*** (due to damage of the nuclei of these nerves by the lesion). This condition is known as **crossed hemiplegia** and the nerve (or nerves) affected differ as follows :

(a) If the lesion was in the ***midbrain***,-*the* 3rd and 4th cranial nerves are affected (= ***Weber's syndrome***).

(b) If the lesion was in *the* ***pons***, the 5th, 6th, 7th and 8th cranial nerves are affected (= ***Millard-Gubler syndrome***).

(c) If the lesion was in the ***medulla oblongata***, the 9th, 10th, 11th and 12th cranial nerves are affected.

Bilateral lesions in the brain stem are rare and lead to ***quadriplegia*** (= *paralysis of the 4 limbs*) + ***bilateral paralysis of the cranial nerses***

(5) IN THE SPINAL CORD :

(a) Bilateral lesions : In the upper cervical region, such lesions are fatal due to interruption of the respiratory tracts. However, in the ***lower cervical region*** (***above the brachial plexus***), ***they lead to quadriplegia***, and in the ***midthoracic region*** (***above the lumbosacral plexus***) , ***they lead to paraplegia*** (= paralysis of both lower limbs).

(b) Unilateral lesions : In the cervical region, such lesions lead to ***ipsilateral hemiplegia*** while in the midthoracic region they lead to ***ipsilateral monoplegia*** (paralysis in the ipsilateral lower limb only).

(3) COMPLETE TRANSECTION OF THE SPINAL CORD

This is fatal if it was above the origin of the phrenic nerve (i.e. above the 3rd cervical segment) e.g. ***in hanging*** due to paralysis of the respiratory muscles. However, at lower levels, patients pass in **3 stages** (a) ***Spinal shock*** (b) ***Recovery of spinal reflex activity*** (c) ***Failure of spinal reflex activity***.

(A) STAGE OF SPINAL SHOCK

The manifestations of this stage include the following :

(1) Loss of all sensations below the level of the lesion.

(2) Quadriplegia or paraplegia (depending on the level of the lesion).

(3) V.D. below the level of the lesion due to interruption of the descending

fibres from the vasomotor centre (may lead to ***hypotension*** in severe cases).
(4) Bilateral miosis *if the lesion was in the lower cervical region*, due to interruption of the descending pupillodilator fibres.
(5) Loss of spinal reflex activity below the level of the lesion *because the spinal centres at this part become functionless*. This causes **loss of :**

a- The withdrawal reflex and other superficial reflexes.

b- The stretch reflex and tendon jerks, leading to flaccidity of the paralyzed muscles (i.e. ***flaccid paralysis***).

c- The erection reflex, as well as the defecation and micturition reflexes, leading to feces and urine incontinence, and dribbling of urine (a condition known as ***retention with overflow***).

CAUSE (MECHANISM) OF SPINAL SHOCK

Spinal shock is due to ***sudden withdrawal of supraspinal facilitation on the spinal alpha motor neurons*** (page 74). It is ***not a hypotensive shock*** (as proved by presence of normal spinal reflexes above the level of the lesion), and is ***not a traumatic shock*** (as proved by the fact that a *second transection made below the first transection does not lead to a second spinal shock*).

DURATION OF THE SPINAL SHOCK

This varies directly with the ***degree of encephalization*** (= degree of development of the cerebral cortex). Accordingly, the lower the degree of encephalization, the shorter the duration of the spinal shock will be (e.g. it lasts only a few minutes in frogs and rats, 1-2 hours in dogs and cats, and several days in monkeys). On the other hand, in man (who is maximally encephalized), it lasts ***2-6 weeks***.

COMPLICATIONS OF SPINAL SHOCK

(1) Hypotension (specially in cases of high-level spinal cord lesions).

(2) Excessive protein catabolism (due to lack of movement) causing muscle wasting and bone dissolution.

(3) Ischemia of the areas compressed against bed (upper back, gluteal region and heels) so these areas are liable to ulceration (= ***decubitus ulcers or bed sores***) which heal poorly due to protein depletion.

(4) Urinary tract infection due to urine retention and stasis.

(5) Fall of the body temperature (due to reduction of the metabolic rate after loss of the muscle tone).

** Rapid recovery of the spinal reflex activity can be achieved by **(1)** ***Antibiotics*** to prevent infection in the urinary tract or bed sores **(2)** Drugs that ***stimulate the spinal centres*** **(3)** ***Bladder catheterization*** to prevent urine stasis **(4)** ***Prevention of bedsores*** by cleaning the skin with antiseptics and frequent changing of the patient's position. **(5)** ***Adequate nutrition***.

(B) STAGE OF RECOVERY OF SPINAL REFLEX ACTIVITY

After the spinal shock, the spinal centres recover gradually as follows :

(1) The flexor (withdrawal) reflex and Babinski's sign are usually the first responses to reappear, followed by the extensor reflexes e.g. the knee jerk.

(2) The static stretch reflex (muscle tone) recovers resulting in ***spastic paralysis***. It appears first in the flexor muscles (resulting in ***paraplegia in flexion***) then in the extensor muscles a few months later (resulting in ***paraplegia in extension***) accompanied by the ***positive supporting reaction*** .

(3) The activity of the ***spinal vasomotor centres*** (the lateral horn cells) is restored, leading to V.C., so the arterial blood pressure rises.

(4) Reflex micturition and defecation return, but such acts are ***automatic as in infants*** (due to absence of voluntary control).

(5) Sexual reflexes recover (e.g. stimulation of the external genital organs in males leads to erection).

(6) Appearance of the MASS REFLEX : This is a hyper-reactive spinal reflex response that ***appears after a few months***. Mild noxious stimuli *applied to the skin below the level of the lesion result in widespread effects* including **(a)** *Exaggerated withdrawal of the stimulated part* **(b)** *Urination and defecation* **(c)** *V.C. and pallor with profuse sweating* **(d)** *Rise of the arterial blood pressure.* It is due to hyperexcitability of the spinal centres accompanied by ***irradiation of signals*** in the spinal cord (see below).

** Patients are trained to induce urination or defecation through producing ***intentional mass reflexes*** (by striking or pinching the skin of the thigh).

** Spinal recovery and the mass reflex may be a ***release phenomenon*** (i.e. due to release of the spinal neurons from the supraspinal gamma inhibitory control). However, it has also been attributed to ***denervation hypersensitivity*** of the spinal neurons as well as to growth of ***new collaterals*** that constitute additional excitatory endings on the spinal neurons.

(C) STAGE OF FAILURE OF SPINAL REFLEX ACTIVITY

This is a ***terminal*** (i.e. ***premortal***) ***stage*** that usually results from bad management during the recovery stage. It is often associated with ***general toxemia*** due to infection of the bed sores or the urinary tract (and the latter frequently terminates by ***uremia***).

The spinal centres below the level of the lesion become depressed again resulting in the following symptoms :

(1) Loss of the muscle tone and tendon jerks, followed by loss of the mass reflex and the withdrawal reflex then the Babinski's sign. Thus, the muscles become flaccid and the body temperature falls.

(2) Loss of the defecation and micturition reflexes resulting in constipation and ***urine retention with overflow***.

(3) ***Hypotension*** (due to depression of the spinal V.C. centres).

(4) HEMISECTION OF THE SPINAL CORD (= THE BROWN-SEQUARD SYNDROME)

This lesion interrupts the ascending and descending tracts on the affected side, which results in the following symptoms :

(A) ABOVE THE LEVEL OF THE LESION

Cutaneous hyperesthesia (= increased sensibility) in the ***ipsilateral dermatomes*** due to irritation of the nerve roots by the trauma.

(B) AT THE LEVEL OF THE LESION

The following effects occur ***on the same side*** :

(1) Loss of all sensations in the areas innervated by the afferent nerves that enter the damaged segments.

(2) Paralysis of the muscles supplied by the efferent nerves that arise from the damaged segments. This is a ***LMNL*** due to damage of the spinal motor neurons (i.e. the anterior horn cells).

(3) Loss of the reflexes that involve the damaged segments.

(C) BELOW THE LEVEL OF THE LESION

(1) ***UMNL in the same side*** that causes hemiplegia or paraplegia (depending on the level of the lesion) due to interruption of the pyramidal tract.

(2) ***V.D. in the same side*** due to interruption of the vasomotor tract

(3) ***Ipsilateral miosis*** (in cases of *cervical lesions only*).

(4) Sensory disturbances :

(a) Ipsilateral loss of fine tactile, proprioceptive, kinesthetic, pressure and vibration sensations, due to *interruption of the dorsal column tracts*.

(b) Contralateral loss of pain and temperature sensations due to interruption of the lateral spinothalamic tract that crosses from the opposite side.

** Crude touch is little affected at both sides because it is transmitted partly by the ventral spinothalamic tracts and partly by the dorsal column tracts.

(5) QUADRANT LESIONS OF THE SPINAL CORD

(A) Anterior quadrant lesions : These lead to **(a)** Contralateral loss of pain and temperature *below the level of the lesion* due to interruption of the lateral spinothalamic tract that crosses from the opposite side **(b)** Ipsilateral LMNL paralysis of the muscles as well as loss of the reflexes *at the level of the lesion* due to damage of the spinal motor neurons.

(B) Posterior quadrant lesions : These lead to **(a)** ***Ipsilateral UMNL*** *below the level of the lesion* which causes hemiplegia or paraplegia (depending on the level of the lesion) due to interruption of the pyramidal tract **(b)** ***Ipsilateral loss of all sensations transmitted by the dorsal column tracts*** *below the level of the lesion* **(c)** ***Ipsilateral loss of sensations and reflexes*** from areas *at the level of the lesion* due to damage of the posterior nerve roots.