6. Motor System

Many of the fibres of the corticospinal tract are concerned with regulating the traffic to and from sensory nuclei, rather than in directly exciting motor neurones.

6.1 Spinal Cord Pathways

Motor Neurones

There are two classes of motor neurones - large (up to 70 microm) alpha cells with axons of 12-20 microm diameter that innervate skeletal extrafusal muscle fibres, and smaller diameter gamma cells with axons of 1-8 microm diameter that innervate the intrafusal fibres of the muscles spindles in skeletal muscles. The cell bodies of both classes of neurones are located in the ventral horns of the spinal cord and in analogous position in the brain-stem.

Alpha motor neurones are multipolar cells (many dendrites, one axon), their dendrites spreading from the ventral horn to the dorsal horn and even across the segment and also up and down a particular cord segment, thus giving many opportunities for synaptic contact. The cell bodies of motor neurones of trunk and proximal limb muscles lie in groups (*motor nuclei*) in the medial part of the ventral horn, the ventromedial group forming a long column running almost the entire length of the spinal cord (C1-L4). In the brain-stem the column becomes discontinuous but the cell bodies of the motor neurones of the cranial nerves XII, VI, IV and III can be found in an analogous position. In the cervical and lumbar enlargements of the cord the cell bodies of motor neurones of all distal limb muscles lie together in nuclei in the lateral part of the ventral horn. In general, the more lateral motor nuclei belong to more distal muscles. 50% of the vast dendritic tree of any motor neurone is covered with synaptic terminals originating from sources within and above the spinal cord and brain-stem. This extreme convergence occurs because motor neurones are the final common pathway from the CNS to muscle.

Gamma motor neurones are scattered amongst the alpha motor neurones of each motor nucleus in the medial and lateral columns of the ventral horn. Their axons pass out of the ventral roots with the larger diameter axons of the alpha motor neurones.

Interneurones

Lying dorsally at the base of the ventral horn and projecting to the motor nuclei are *excitatory* and *inhibitory* interneurones involved in the regulation of motor neurone discharge. Their topography is similar to that of motor neurones, the more medial interneurones projecting mainly upon the medial motor nuclei and the more lateral interneurones upon the lateral motor nuclei.

Organization of Function Within the Ventral Horn

The trunk muscles, together with the proximal limb muscles, are involved in functions such as balance, posture and walking which require body or whole limb movement. The muscles moving the fingers and wrists do not have postural functions but are involved in manipulatory movements. The motor neurones and interneurones in the ventral horn are

organized to carry out these diverse functions, the medial group being involved in posture and the lateral group directing manipulatory activity. The descending inputs to the ventral horn similarly divide into those primarily influencing the medial neurones and hence posture, and those influencing the lateral neurones and hence manipulatory activity.

Organization of Function Within a Motor Nucleus

The motor neurones of a group innervating a given muscle share much the same afferent input and therefore their excitability tends to change in much the same way at the same time. In general the smallest diameter motor neurones have the lowest threshold and the smallest motor unit. Their discharge therefore generates a small increment of tension. In graded contractions of a given muscle the recruitment order remains stable, each motor neurone firing in turn as its threshold is exceeded and increasing its firing rate as the excitation increases. Initially as each small motor neurone discharges there is a small increment of tension, larger increments being added later in the contraction when larger diameter cells are excited. With this mechanism it is possible to vary tension over a wide range with a relatively small number of motor units.

Organization Between Motor Nuclei

The information that reaches the spinal cord from the brain is coded in terms of movement not in terms of muscles. It is distributed to interneurones at the base of the ventral horn which in turn excite motor neurones in the motor nuclei for the movement. Complex connections exist, walking for instance being generated followed the delivery of a simple excitatory signal to the initiating interneurones. Swallowing is another example of a complex pattern of movements which may be begun with a simple afferent input provided by contact of food with the back of the pharynx. Normally, programmes are modified by incoming information from the periphery, superimposed on the basic pattern. For example, when walking the pattern is adapted to cope with irregularities on the surface.

Spinal Reflexes

In muscle there are mechanoreceptors providing information about muscle length and tension, involved in the subconscious control of posture and movement and also contributing to sensation. These are associated with specialized structures in muscle known as *muscle spindles* and also with receptors on tendons known as *Golgi tendon organs.* In addition there are, in muscle, receptors sensitive to severe pressure and stretch. These receptors, which give rise to cramping pain, are probably located in blood vessels and in connective tissue of the muscle and are sensitive to products released from muscle working without an adequate blood supply. As in the skin the different types of endings are supplied by nerve fibres of different diameter but they are usually referred to by the I, II, III, IV classification.

Muscle spindles. These structures are made up of two to twelve small-diameter striated muscle fibres termed *intrafusal* muscle fibres, the main muscle fibres known as *extrafusal* fibres. The whole bundle of intrafusal fibres is enveloped in a capsule and lies in parallel with the main muscle fibres and is attached to them at its ends. The middle third of the intrafusal fibre, known as the equatorial region, is non-striated and largely non-contractile. The outer

thirds, known as the polar regions, are striated and contractile. One muscle contains many muscle spindles.

There are two types of intrafusal muscle fibres: (a) *nuclear bag fibres*, commonly two per spindle, which are longer and thicker and have a group of nuclei clustered around the centre of the fibre; and (b) *nuclear chain fibres* which are shorter and thinner and have fewer nuclei arranged in a chain along the centre of the fibre; there are commonly four to five per spindle.

Group Ia fibres coil around the equatorial region of the nuclear bag and chain fibres to form *primary* receptor endings (*annulospiral endings*). Group II fibres (smaller and slower-conducting) end mainly on nuclear chain fibres to form *secondary* receptor endings (*flowerspray endings*).

The receptor endings of both group Ia and group II afferents are mechanoreceptors and they are stimulated when the equatorial region of the spindle is stretched, such as happens when the whole muscle is stretched, as this pulls on the ends of the spindle. This pattern of responses is known as *'in parallel'* behaviour as it is due to the fact that the spindles are in parallel with the extrafusal muscle fibres. The response from the spindle endings is reduced or silenced when stretch on the equatorial region of the spindle is removed, either by removing stretch from the whole muscle or by contracting the extrafusal muscle fibres, thereby slackening the intrafusal fibres.

Such a response to change in the length of the muscle is known as the static or length-sensitive response and is in some ways analogous to the position response of cutaneous mechanoreceptors. Both primary (ia) and secondary (II) endings give a static response. During static firing the frequency of impulses is proportional to the muscle length. The static response is probably associated with stretch of both bag and chain fibres.

Spindles also respond to the *rate of change* of length of the muscle. This is known as the *dynamic* or velocity-sensitive response. Only the primary (Ia) endings give a large dynamic response. The greater the velocity or rate of length increase, the higher the frequency. During release from muscle stretch group Ia fibres, both primary and secondary endings, commonly show a decrease in impulse frequency.

In addition to the two types of sensory ending, muscle spindles also have their own motor innervation. This innervation is supplied by *fusimotor* or gamma motor neurones, the extrafusal muscle fibres being supplied by extrafusal or alpha motor neurones. Stimulation of the fusimotor muscle fibres does not alter tension in the whole muscle but it produces contraction of the polar region of the intrafusal fibres. This, in turn, causes a stretch of the equatorial region of the intrafusal fibres and thus results in excitation of the primary and secondary afferent endings. The motor supply to the muscle spindles allows the spindle length to be regulated thus altering its sensitivity and allowing it to function over a wide range of muscle lengths.

Golgi tendon organs. These receptors occur on tendons usually near the muscle-tendon junction. Their afferents are Ib fibres which branch over several tendon fascicles, covered by

a capsule which, at its ends, becomes continuous with the connective tissue of the muscle or tendon.

A Golgi tendon organ responds to the tension in its muscle fascicle (and hence in the tendon) with an increase in frequency of impulses both when the muscle is stretched passively and when it contracts actively. This is known as *'in series'* behaviour as the tendon organ is on the tendon, in series with the muscle. The increase in response during extrafusal muscle contraction distinguishes the Golgi tendon organ from the muscle spindle receptors whose response is reduced.

The Golgi tendon organ responds to both the velocity of tension development and to constant or maintained tension. It has thus both dynamic and static response characteristics. It is less sensitive to passive muscle stretch than is the muscle spindle as passive stretch lengthens the muscle rather than the tendon. However, during active muscle contraction the response of the tendon organ is greater than for passive stretch of the same degree because active contraction causes greater stretch of the tendon. Thus Golgi tendon organs do not respond only at extreme tension as originally though but respond also during normal tension development.

Stretch (Myotatic) Reflexes

Muscle spindles are the starting point for the simplest of all reflexes found in mammals - the *stretch reflex.* When a muscle is stretched the elongation leads to a discharge in the muscle spindle afferents that is conducted into the spinal cord via the dorsal roots. Within the cord the sensory fibres branch and some terminals make *monosynaptic* excitatory contacts with *alpha motor neurones* to the extrafusal muscle fibres of the *same* muscle. Impulses generated in these motor neurones leave the cord in the ventral roots and conduct impulses back to the muscle causing it to contract in opposition to the change in length. These reflexes may be elicited by rapid or slow changes in muscle length and tend to maintain a constant muscle length with a change in load.

These reflexes persist after all cutaneous nerves have been cut and any tendon organs anaesthetized. They are specific to stretched muscle, hence the name *stretch* or *myotatic reflexes.* The degree of stretch need only be 1-2 mm and is normally the result of gravity. These reflexes have both phasic and tonic components, are of short latency, and, although the tonic component is well-nigh infatiguable, are very susceptible to inhibition. They are present to some extent in all muscles but are best seen in the antigravity muscles, which for most species means the extensors.

Primary endings respond both to the velocity of stretch and to maintained length, secondary endings only to the latter. The length of the spindle can be modified independently of that of the extrafusal fibres by impulses travelling along the gamma-efferent fibres. There are in fact two types of fusimotor fibres, static and dynamic. Stimulation of static fibres increases the static response of both groups Ia and group II afferents. Stimulation of the dynamic fibres increases both the dynamic and static responses of group Ia fibres. In addition there are two types of ending on the infrafusal fibres: (a) *plate endings,* on both nuclear bag and nuclear chain fibres at the ends or poles of the fibres, possibly associated primarily with

dynamic fusimotor fibres; and (b) *trail endings,* mainly on nuclear chain fibres just next to the equatorial region of the fibres and associated mainly with static fusimotor fibres.

Simple *phasic* stretch reflexes (tendon jerks) are evoked by a brief muscle stretch which excites the dynamically-sensitive spindle afferents. When a neurologist flexes and extends, for example, the patient's elbow, that is, when he alternately stretches the biceps and triceps muscles, he normally encounters a certain resistance. This resistance or *'tone'* is in fact reflex and not due to any elasticity of the muscles. This reflex is more complicated than a tendon jerk. An electromyogram shows a series of waves. The first, of short latency, is generated by the phasic action already described. The origin of later waves which provide the tonic-maintained aspects of this effect is not well understood. On the one hand it is claimed that there is a long-loop reflex with primary spindle activity routed to motor neurones by way of the motor cortex, with the additional 10-15 msec delay imposed by the cortical loop. On the other hand it is claimed that the later components result from spinal reflexes originating perhaps from spindle secondary endings, the delay being imposed by their smaller diameter afferent fibres and largely polysynaptic connections.

Flexor (Nociceptive) Reflexes

A typical flexor reflex is the withdrawal of a limb following painful or potentially painful stimulation. The receptors are skin nociceptors, the central connections polysynaptic and the efferent limb involves nuclei of most of the limb flexor muscles.

Reciprocal Innervation

Built into the circuitry of stretch and flexor reflexes is an inhibition of motor nuclei of muscles antagonistic to the movement. It is seen not only between the muscles at a joint but also between the two halves of the spinal cord. Thus an incoming nociceptive stimulus can excite flexor motor neurones and, while inhibiting the ipsilateral neurones of the antagonistic muscles, also excite the contralateral motor neurones of extensor muscles (*crossed extensor reflex*).

Recurrent Inhibition

In addition to the influence of the spinal mechanisms, alpha motor neurones are influenced directly by the activity of interneurones which they themselves activate. In this feedback circuit the interneurones, *Renshaw cells,* are inhibitory and are excited by transmitter released from collateral branches of the motor neurone. *Renshaw inhibition* appears to play an important role in dampening the activity of motor neurones; in particular, it appears to limit the discharge frequency of tonically active alpha motor neurones.

Regulation of Muscle Contraction

The properties of muscles are such that a constant signal will not always produce the same increase in tension or change in length. This variability arises because the contractile responses of muscles depend on their initial length and because muscles fatigue. To compensate for this behaviour and the varying loads imposed on muscles, they and their

nervous reflex loops are arranged as servo-mechanisms, that is, mechanisms that modify their output to adapt to varying loads. Such systems have distinct advantages as they can quickly and accurately follow command signals and automatically compensate for changes. Muscle spindles and Golgi tendon organs and the various neural pathways associated with these receptors are involved in such processes.

The myotatic (stretch) reflex pathway is involved in both the initiation of contractions and the control of muscle length. Contractions are initiated through this pathway when activity in gamma motor neurones causes muscle spindles to increase their rate of discharge. Thus both alpha and gamma motor neurones can be used to initiate muscle contraction. The pathway involving alpha fibres is faster, but the pathway involving gamma fibres probably gives more sensitivity of control and is uniformly effective over the whole range of muscle lengths.

The myotatic reflex pathway is also a feedback system in the control of muscle length. Stretching a muscle beyond its desired length activates the muscle spindles so that they produce action potentials at an increased rate. Their signals activate alpha motor neurones supplying that muscle, so that the muscle contracts until the desired length is restored. If a muscle contracts so that it is shorter than the desired length then the spindle receptors are silenced, activation of motor neurones is reduced, and the muscle relaxes.

As an example, consider the effects on the biceps muscle of placing an extra load on the hand when the forearm is outstretched. Before the load is placed on the hand the alpha and gamma motor neurones to the biceps discharge at a rate that is just sufficient to maintain the position of the limb. As the load is applied, the forearm drops and the biceps muscle is stretched. This lengthening is monitored by the muscle spindles and, after a brief delay due to conduction in the afferent fibres, the information is transmitted to the spinal cord. The biceps motor neurones almost immediately increase their rate of discharge and, after a further delay, this signal reaches the biceps muscle where a greater muscle force is developed. The force rapidly reaches a level greater than the disturbing force, so that the hand begins to move back towards its former position. However, as the muscle shortens, the signal from the muscle spindles is progressively reduced until the muscle produces a force no greater than the loading force and movement stops. The muscle does not return precisely to its former length because a small steady-state error is required to produce enough output from the spindles to overcome the extra load.

In some circumstances two opposing muscles can operate to fix the position of a limb. In this case the pathways producing reciprocal inhibition become active. For example, stretching the biceps muscle spindles produces inhibition (via an interneurone) of triceps motor neurones and, similarly, stretching triceps causes inhibition of biceps. Thus pairs of opposing muscles become organized into a single working unit. This unit may also be brought into play in normal limb movement when the contraction of antagonistic muscle will help to dampen movements and prevent overshoot.

In addition to the system regulating muscle length there is a negative feedback loop regulating muscle tension. This system depends on signals from the Golgi tendon organs of a muscle and leads to inhibition of the motor neurones that control that muscle. This inhibitory influence is present even when the muscles are contracting normally.

The role of this loop can best be considered when it operates in isolation from the length system so that the force generated by a muscle is maintained, despite changes in length or the development of fatigue. For example, if the muscle force is altered by fatigue, then inhibition of the motor neurones is decreased and muscle is excited more strongly. If the force becomes too great, for example as a result of a change in muscle length, the inhibitory effect will be increased and the force correspondingly reduced.

However, it should be noted that the above is an oversimplification. Both the muscle length and the muscle tension servo-mechanisms normally operate together and both employ the same alpha and gamma motor fibres. They are both arranged so that they reciprocally innervate antagonist muscles. Thus they allow muscles to exert a constant tension while changing length, and to quickly adapt to changing variations in load.

Autonomic Reflexes

Activation of nociceptors can give rise to reflexes which have their connections with autonomic preganglionic neurones in the intermediolateral column of the spinal cord. These neurones have axons which excite the cells of the adrenal medulla and the postganglionic neurones innervating sweat glands and smooth muscle of blood vessels.

Other autonomic reflexes bring about the emptying of the bladder and rectum. These hollow organs have stretch receptors in their walls which signal the wall tension and thus the organ volume. The signals are appreciated consciously as a sensation of fullness and they also activate reflex connections in the spinal cord. *Defaecation* is brought about by distension of the rectum as faeces pass into it from the colon. This causes the *anal sphincter* to open reflexly, and the diaphragm and muscles of the abdominal wall to contract with expulsion of the faecal mass.

The smooth muscle of the bladder wall (the *detrussor muscle*) and neck (the *internal sphincter*) is innervated by sympathetic fibres (inhibitory) from the lumbar segments of the spinal cord and by parasympathetic fibres (excitatory) from sacral segments 2 to 4. The *external sphincter* is a striated muscle with a somatic innervation. When bladder wall tension reaches a certain level the detrusor contracts reflexly and the internal sphincter relaxes. It should be noted, however, that the bladder wall can accommodate increasing volumes of urine with very little alteration in tension. Only when the volume reaches (usually) 300-400 mL is there an appreciable degree of discomfort associated with a steeper rise in tension and triggering of the *micturition reflex.* An indication that these emptying reflexes can be facilitated from the brain is that the bladder can be emptied at any volume. More often, however, these spinal reflexes are inhibited by cerebral activity until the sensation become insistent. The reflex activity is then augmented and the external sphincter relaxed. In the case of the bladder the flow of urine begins and is facilitated by ancillary reflexes from the urethra. These reflexes are stimulated by flow of urine and the efferent activity reinforces bladder muscle contraction and sphincter relaxation. Contraction of the abdominal wall and the pelvic floor also aid the complete emptying of the bladder.

Following transection of the spinal cord and the onset of spinal shock the bladder wall is inert and the sphincter closed, resulting in urinary retention with overflow. As the shock

wears off and detrusor muscle tone returns reflex emptying can be brought about by, for example, cutaneous stimulation, which may at the same time cause defaecation and other evidence of widespread autonomic activity (*'mass' reflex*).

6.2 Supraspinal Pathways to Motor Neurones

Through these pathways the brain evokes movements and their postural concomitants, by exciting directly or indirectly the motor neurones. We can distinguish four descending pathways extending the length of the spinal cord which have these functions. These are the cortico-, rubro-, lateral vestibulo- and reticulospinal tracts. A further two tracts extend only through the cervical region and are primarily concerned with the motor nuclei of neck muscles. These are the tecto- and medial vestibulospinal tracts.

Corticospinal Tract (Pyramidal Tract)

This tract is the most important pathway in motor control. It comprises axons of pyramidal cells in layers III and V of the premotor area, precentral gyrus and postcentral gyrus in almost equal proportions. The tract descends through the posterior limb of the internal capsule and the cerebral peduncles to pass through the pons and medulla. In the mid-brain and medulla, axons (bulbospinal fibres) leave the tract and pass to the cranial nerve motor nuclei. These are crossed pathways but no definite tracts can be seen as the axons pass across the brain-stem in small separate bundles. Axons also pass to the red nuclei and to the reticular nuclei, all of which are themselves the source of spinal pathways, sometimes spoken of as the *indirect* corticospinal pathway.

In the caudal medulla the pyramids are formed mainly by the decussation of the *direct* corticospinal fibres. The crossed fibres thereupon enter the lateral columns of the spinal cord forming the lateral corticospinal tract. A few fibres (15%) do not cross at this level and pass down ipsilaterally. They are though to cross in the spinal cord.

The fibres of the lateral corticospinal tract terminate on the lateral group of interneurones in the dorsal part of the ventral horn. In the lower cervical cord about 10% of the fibres also terminate directly on motor neurones, probably in motor nuclei which serve the finger and hand muscles.

Rubrospinal Tract

This tract arises from cells of the posterior part of the *red nucleus* in the mid-brain. The large diameter fibres cross immediately and run down the contralateral side of the brain-stem and spinal cord. Their terminals are distributed similarly to those of the corticospinal tract. The red nucleus receives input from the cerebral cortex and the dentate and intermediate nuclei of the cerebellum.

Lateral Vestibulospinal Tract

This tract arises from the lateral vestibular nucleus, is ipsilateral and terminates in the medial part of the base of the dorsal horn. A few fibres terminate on medial motor neurones.

The lateral vestibular nucleus receives inputs from the vestibular nerve and the fastigial nucleus of the cerebellum.

Reticulospinal Tracts

Two pathways exist. One originates in the pontine reticular system and runs ipsilaterally down the spinal cord. The other originates in the medullary reticular system and bifurcates to run down both sides of the cord. Both systems terminate on the medial group of interneurones and to a small extent on medial motor neurones. The reticular nuclei receive input from the cerebral cortex and the fastigial and intermediate nuclei of the cerebellum.

Tectospinal Tract

This tract originates in the mid-brain in the region of the superior colliculus. It is a crossed pathway and the fibres terminate on interneurones in the cervical cord. At its origin it receives input from the cerebral cortex, especially the occipital lobe, and from the superior colliculus. The pathway seems to generate head movements to help direct one's gaze at a particular point.

Medial Vestibulospinal Tract

The medial vestibulospinal tract arises mainly from the medial vestibular nucleus and is the caudal equivalent of the medial longitudinal fasciculus. The tract is uncrossed and terminates in relation to the same cells as the tectospinal tract. The function of the tract is to operate neck muscle in response to vestibular stimulation, so keeping the head stable during body movements.

There are pathways running in the ventral quadrant which originate first in the *raphe nuclei,* a narrow continuous collection of cells in the midline of the brain-stem which contain 5-hydroxytryptamine (5HT or serotonin) and secondly in the *locus caeruleus*, which is located near the floor of the rostral part of the fourth ventricle and the cells of which contain noradrenaline. These pathways appear to facilitate the discharge of motor neurones. Both serotonin and noradrenaline when injected intravenously increase the discharge of alpha and gamma motor neurones in experimental animals. Moreover section of the ventral quadrant of the cord above the level of the lumbar enlargement interrupts these pathways and abolishes the response of lumbosacral motor neurones to stimulation of the contralateral precentral motor cortex, despite the continued functioning of the lateral corticospinal tract which runs in the lateral quadrant. It seems likely then that these monoamine systems may set the background level of excitation of motor neurones in conformity with a person's emotional state.

6.3 Control of Posture

By *posture,* we mean the maintenance for a period of time of position (of head, limbs, trunk) in space, as a prelude or background to movement. Fundamentally, posture implies a particular distribution of muscle tone and the most basic of postural mechanisms is thus the *stretch reflex.*

When standing comfortably the body is in a position in which the weight is taken by bones and ligaments with little muscle activity. Attempts to displace the body from this position by gravity or acceleration are actively resisted. This resistance is the result of various postural reflexes acting via the eye, neck, trunk and proximal limb muscles, and which operate at an unconscious level. The pathways in brain-stem and spinal cord to the motor neurones of the postural (antigravity) muscles are the vestibulo- and reticulospinal tracts.

There are three basic classes of postural reflex: (i) reflexes responding to gravitational force, (ii) reflexes responding to linear acceleration and (iii) reflexes responding to angular acceleration.

Reflexes Evoked by Gravitational Force (Static Reflexes)

Stretch reflexes, requiring the nervous machinery of only one or two spinal cord segments, can be though of as units which can be combined into more and more complex patterns (static reflexes) and modulated in more and more ways as the involvement of the CNS increases. Thus a spinal animal cannot remain upright for any length of time although it will exhibit stepping motions; after upper medullary section an animal can stand for long periods but cannot right itself if laid on its side; while a mid-brain section in quadrupeds, although not in primates, will permit such righting responses.

Local Static Reflexes

These reflexes are confined to the stimulated limb and the most important is the *tonic stretch reflex,* the elements of which are described before and which is the basis of the maintenance of an upright posture. As an example, the muscles which extend the knee during standing are continuously stretched and therefore are in a state of reflex contraction.

Segmental Static Reflexes

These reflexes are evoked by, for example, nociceptor stimulation of one limb. The reflex response is seen in both limbs. For instance, flexion of a leg in response to a noxious stimulus to the foot is accompanied by extension of the opposite leg (the *crossed extensor reflex*). This response reflects the inbuilt circuitry of stretch and flexor reflexes (see reciprocal innervation before).

General Static Reflexes

In these reflexes the stimulus is local but the response includes many muscle groups. Many such reflexes involve stimulation, by changes in head position, of the abundant muscle spindles in the neck muscles. Such movements also produce stimulation of the labyrinths. These reflexes implicate both limb and eye muscles and are most easily illustrated in quadrupeds. After destruction of the labyrinths, *tonic neck reflexes* depending only on neck muscle proprioceptors can be elicited by, for example, turning the head to the right relative to the body. This will cause the right limb to extend and the left limb to flex. Turning to the left has the opposite effect. Dorsiflexion of the head will extend the forelimbs and flex the hind limbs, while ventroflexion has the converse effect. If the neck proprioceptors are eliminated, dorsiflexion will extend all four limbs and ventroflexion will flex them. These

later reflexes must therefore by labyrinthine in origin. Normally the two sorts work in a complementary fashion.

An example of the influence of neck movement on the eyes is the *doll's head phenomenon* seen in comatose patients whose brain-stem is still intact. In such patients rotation of the head to the left yields an eye movement to the right and vice versa. Dorsiflexion of the head produces eye movement downward and ventroflexion an upward movement. Presumably these reflexes normally contribute to the maintenance of visual fixation.

Righting Reflexes

When an animal laid on its side awakes from sleep, it stands up. It adopts this standing posture as a result of the operation of a series of righting reflexes which involve signals from skin receptors, the labyrinth, and muscle proprioceptors, and which also include visual reflexes dependent on the cerebral cortex.

Reflexes Evoked by Linear Acceleration

Receptors in the inner ear have been described. Fibres project from the utricle and saccule to the lateral vestibular nuclei which give rise to the vestibulospinal tracts. An example of this type of reflex is the *vestibular placing reaction.* This can be evoked by holding a blindfolded cat by its pelvis, head down, whereupon its forelegs extend. The response is lost if the utricle is destroyed.

If the blindfold is removed a cat will still extend its legs even if its utricles are destroyed - a *visual placing reaction.* In general, visual stimuli can substitute for labyrinthine in postural reflexes. Thus animals and men with bilateral labyrinthine damage have normal posture if they can use their eyes.

Reflexes Evoked by Angular Acceleration

Rotatory stimuli are detected by the receptors in the semicircular canals. The position of the cupulae, and thus stimulation of the receptors, depends upon angular acceleration with inertial lag of endolymph movement. The receptors project to the medial (and other) vestibular nuclei which give rise to the medial longitudinal fasciculi and the medial vestibulospinal tracts. The output side of the reflex response therefore involves movements of eye, neck and proximal arm muscles.

The situation is most easily analysed in relation to the horizontal canals. If a person is rotated to the *right,* movement of the endolymph and right cupola is to the left, i.e. *ampullopetal*, while that of the left cupola is *ampullofugal.* Appropriate signals are sent to the medial vestibular nuclei. The movement of the eyes is in the same direction as the endolymph flow, retaining the fixation point as the head moves. As the rotation continues the fixation point cannot be maintained and there is a quick flick of the eyes to the right to a new fixation point. The slow drift and quick flick will be repeated until there is no longer movement of the endolymph in the canal. The eye movements are called *vestibular nystagmus.* A

nystagmus is always named after the quick phase so, in the example, the nystagmus would be to the right.

If the head is rotating at constant velocity, there is no relative endolymph movement, the cupulae gradually take up the resting position and vestibular nystagmus ceases although reflex movement of the eyes (*optokinetic nystagmus*) continues depending on the visual input. On ceasing rotation, cupular movement is now in the opposite direction, returning to rest in 25-30 sec.

Ampullopetal movement, absolute or relative, of the endolymph in the right horizontal canal occurs (i) on moving the head to the right; (ii) on ceasing rapid rotation to the left; (iii) on syringing the right ear with warm water. Obviously the same effect is obtained by syringing the left ear with cold water and after destruction of the left labyrinth. The sensations which arise are those of movement to the right or of the external world to the left; thus the slow drift of the nystagmus and forced movements are to the left. These are commonly accompanied by vertigo and general autonomic disturbance. Nystagmus in the absence of appropriate stimuli may be a symptom of brain-stem, labyrinthine or cerebellar injury or disease.

Effects of Spinal and Brain-Stem Transection

The preceding sections outline the reflex mechanisms by which the motor neurones (both alpha and gamma) of antigravity muscles are excited, particularly via the vestibulospinal and reticulospinal paths. This excitation is an important determinant of the reflex resistance of a muscle to stretching (muscle tone). It is therefore understandable that a spinal transection which severs these connections causes a severe loss of tone and reflexes in segments below the section. Indeed, immediately following a spinal transection, for example in the cervical region below the level of the phrenic motor neurones, there ensues a state of profound torpor. The muscles lie inert, the strongest stimulation of a sensory nerve evokes no response, reflexes are unobtainable, the blood vessels dilate, the blood pressure falls, thermal sweating is absent, the bladder distends with urinary overflow and the viscera in general are quiescent. This condition is known as *spinal shock.* It is not due to the trauma nor to the low blood pressure but to interruption to the normal flow of impulses down the long descending tracts of the cord that impinge on interneurones and motor neurones so that their excitability is lowered. The lateral vestibular and pontine reticulospinal tracts are most important in this respect and spinal shock follows any section caudal to the lateral vestibular nucleus. The course of recovery varies with the species from a few minutes in frogs to some weeks in humans. There is a gradual increase in excitability. First flexor withdrawal reflexes return with extensor inhibition, then bladder and rectum empty reflexly, the blood pressure rises, and after some months (in man) extensor activity returns and tendon jerks can be elicited.

The recovery of excitability of these neurones which underlies the reflex recovery may be due to sprouting of the terminals of their remaining inputs. This may enable the input fibres to excite the motor neurones more readily. There may also be an increase in sensitivity of the motor neurones to the transmitter released by the remaining inputs, so that the same quantity of transmitter is now more effective. This would be akin to the post-denervation supersensitivity of skeletal muscle.

Brain-stem section just rostral to the vestibular nuclei causes the tone to be exaggerated instead of lost and was first described by Sherrington (1857-1952), a pioneer of neurophysiology. The exaggeration of tone is so extreme in antigravity muscles that a particular posture is maintained - *decerebrate rigidity.* Humans in this condition have arms and legs extended, the back arched and the head dorsiflexed. The feet are ventroflexed and the arms pointed. The wrists are flexed and the fingers little affected. The rigidity is due, essentially, to the removal of inhibitory influences with the maintenance of facilitatory influences, and can be made more extreme by making a more rostral transection, in the mid-brain, leaving the pontine reticular nuclei intact. Neurones in this nucleus, like the neurones of the lateral vestibular nucleus, excite the motor neurones of antigravity muscles.

Decorticate rigidity occurs when the postural mechanisms are damaged above the brain-stem. It differs from decerebrate rigidity in that it can be modified by reflex means and shows great variation in severity in different species. Cats, for instance, can walk about in a decorticate condition, but a man in this condition is unconscious, with legs extended and arms in a position determined by his head position (through neck reflexes). Thus if the head is turned to the right, the right arm is extended and the left arm flexed and if the rotation is to the left the opposite is seen.

6.5 Supraspinal Organization of Movement

The decision to move involves the cerebral cortex as a whole. The instruction is channelled through relatively direct connections to the basal ganglia and cerebellum. The instructions from these organs are then collected by the ventrolateral thalamic nuclei which relay to the motor cortex. The motor cortex is the source of that part of the corticospinal and corticobulbar tracts which relay to the spinal cord and brain-stem motor machinery and of the corticofugal pathways to the red and reticular nuclei which also relay to the spinal cord motor machinery.

At the spinal level there must be integration of these instructions with those that govern posture which are delivered via the medial pathway. The end result is alteration of firing rates of the appropriate neurones which leads to contraction or relaxation of muscles to produce the desired movement.

Role of Cerebral Cortex

That the cerebral cortex has a prominent role in movement has long been suggested on clinical grounds. Actions in the usual sense of the word, like the taking off of one's hat or the turning on of a light, are composed of a large number of individual movements. A person who suffers a left-sided parietal lobe lesion may be unable to carry out such an action on command, though quite capable of the individual movements which compose the action. This condition is termed *apraxia.*

More recently, electrical signs of cortical activity before movement have been recorded. This discovery was made by making subjects perform the same movement, i.e. the voluntary flexing of an index finger many times, and averaging the tiny signals recorded from the subject's scalp before and during the movement. In such an experiment when the electrodes are placed over the precentral gyrus a slow surface negative potential is recorded,

presumably indicating excitation of cortical neurones, which begins more than 0.5 sec before the movement and comes to a peak just after movement. This initial surface potential is termed a *readiness potential* and can be recorded bilaterally over the frontal and parietal lobes. Starting some 80 msec before movement there is a rapid increase in potential called the *motor potential* which signals activity of motor cortical neurones. It is recorded only unilaterally over the appropriate part of the motor cortex.

Basal Ganglia

The basal ganglia are found deep to the cerebral cortex in each hemisphere. They comprise the *claustrum,* the *putamen* and the *caudate nucleus* and, closest to the midline separated only by white matter from the thalamus, the *globus pallidus.* These four nuclear masses are collectively known as the *corpus striatum.* The caudate nucleus and the putamen together comprise the *neostriatum* and the globus pallidus the *paleostriatum.* The putamen and globus pallidus are together often referred to as the *lentiform nucleus* because of their combined shape. The white matter separating the thalamus and caudate nucleus from the putamen and globus pallidus is known as the *internal capsule.* All the axons passing to and fro between the motor and sensory areas of the cerebral cortex on the one hand, and the thalamus and brain-stem on the other, pass through this small region. Other smaller nuclei are for functional reasons usually considered along with the basal ganglia. The largest of these are the *subthalamic nucleus* and the *substantia nigra,* a darkly pigmented nucleus lying in the mid-brain. The function of the claustrum is unknown.

All components of the basal ganglia are richly interconnected. Little is known of these connections but some at least of the interneurones in the caudate and putamen use acetylcholine as their transmitter and the *striatonigral pathway* from the caudate and putamen to the substantia nigra is though to utilize gamma-amino butyric acid (GABA) as the transmitter. More importantly the *nigrostriatal pathway* from the substantia nigra to the caudate and putamen is *dopaminergic* and is disordered in Parkinson's disease. The subthalamic nucleus is known to be connected reciprocally with the substantia nigra and globus pallidus but the transmitters are unknown.

Afferent pathways. The caudate and putamen receive inputs from the cerebral cortex, the thalamus and the raphe nucleus. The cortical connection is massive, comes from layer V of the six-layered cortex and is distributed topographically. For instance, the frontal lobe is related to the head of the caudate, the parietal lobe to its body and the occipital lobe to its posterior part. The putamen receives a similar topographic input. Much detail is being added to these connections through the application of modern anatomical techniques. For example, it appears that cortical regions which are interconnected project also to the same part of the caudate nucleus. Thus one part of the caudate may be connected with parts of the parietal, temporal and occipital lobes. The thalamic projection to the neostriatum (caudate nucleus and putamen) is also topographically organized and comes from the intralaminar nuclei, though to be the target of the reticular activating system. The neostriatum also receives an extensive serotonergic projection from the dorsal raphe nucleus.

Efferent pathways. The only output from the neostriatum, other than its connections with the substantia nigra, is to the globus pallidus which is therefore the major projection

nucleus of the basal ganglia. Axons of pallidal cells terminate in the ventral thalamic nuclei (ventrolateral and ventroanterior) which in turn project to the motor cortex and frontal lobe. The circuit comprising the cortex - caudate nucleus and putamen - globus pallidus - thalamus - motor cortex, is thought to play an important part in the supraspinal control of movement.

Functions of The Basal Ganglia

Neurones in the striatum of experimental animals discharge in relation to movement of the *contralateral limb.* The basal ganglia of one side then, like each motor cortex, are related to the opposite side of the body. Further, these neurones discharge *before* movement and presumably these signals pass to the ventral thalamic nuclei which in turn send signals to excite motor cortex cells. It is known that the globus pallidus has an inhibitory effect on the ventral thalamus. The basal ganglia output then exerts its effect by selective inhibition of the ongoing activity of cells of the ventral thalamic nuclei. It is possible that the basal ganglia are concerned with the fixing of the body and proximal parts of limbs to provide a secure base for movements of the hands and feet. It is probable too that it is simplistic to think of the basal ganglia purely as concerned with motor functions. There is, for example, physiological and anatomical evidence of connections with the hypothalamus concerned with autonomic function.

The association of the basal ganglia with the control of movement was first proposed as the result of studies on patients with diseases of - or injuries to - this region. There are three main signs of such damage: (i) abnormal movements, (ii) abnormal muscle tone (*rigidity*) and (iii) *bradykinesia,* i.e. a slowness in initiating and changing movement. An example of bradykinesia is where a patient with Parkinson's disease when asked to walk away from the examiner is slow to get going, and when asked to come back has great difficulty in turning around.

Abnormal movements are of four basic types: (i) *tremor,* which is characteristically involuntary, occurs at rest, and disappears during movement; (ii) *athetosis* or slow writhing movements, which are particularly noted in patients with cerebral palsy whose basal ganglia were damaged at birth; (iii) *chorea* or involuntary jerky movements of the extremities and facial muscles; and (iv) *ballismus* or violent flailing movements usually of one limb, involving the proximal muscles. Ballismus is a characteristic sign of damage to the subthalamic nuclei.

The most common disorder of the basal ganglia is Parkinson's disease, in which patients have a resting tremor, rigidity and bradykinesia. These signs are though to be a consequence of the known loss of substantia nigra cells and thus of the dopaminergic pathway to the caudate-putamen complex. This view has led to treatment with a dopamine precursor, L-dopa, which crosses the blood-brain barrier and alleviates the symptoms. Surgery which interrupts the outflow of the basal ganglia (in globus pallidus or thalamus) can alleviate the tremor and rigidity, suggesting that these disorders arise from abnormal signals sent to the thalamus by the disordered basal ganglia. The bradykinesia is not attenuated, suggesting that it may be the primary deficit in the disease.

The rigidity in Parkinson's disease can be shown to arise from abnormal excitation of alpha and gamma motor neurones of both flexor and extensor muscles around a joint. The

abnormal degree of activity of both agonists gives rise to the rigidity. The excitation waxes and wanes with the tremor. Thus the rigidity also momentarily increases and weakens giving rise to its name *cog-wheel rigidity.*

Role of the Cerebellum

The gross anatomy of the cerebellum has already been discussed in Chapter 2. Its structure at the cellular level is fairly well understood, as are its neural circuits. It is thus particularly frustrating that an equivalent level of understanding of its function has so far not been achieved. All the lobes of the cerebellum have a three-layered cortex covering white matter in which are embedded the deep nuclei. Histologically the cortex displays a remarkable degree of uniformity.

Cerebellar Cortex

The three layers comprise: (i) a *molecular layer* containing a large number of axons which run along each folium parallel to its long axis, and which intersect Purkinje dendritic trees at right angles; (ii) a thin *Purkinje cell layer* containing the cell bodies of the large Purkinje cells; and (iii) a *granular layer* containing 20 billion granule cells and the elaborate synaptic complexes between incoming mossy fibre axons and granule cell dendrites.

There are five cell types - Purkinje cells, granule cells, Golgi cells, basket cells and stellate cells.

Purkinje cells are 15 million in number. Their dendritic trees pass up into the molecular layer to intersect with parallel fibres. Spines on their dendrites are the site of excitatory synapses between granule cell axons and Purkinje cell dendrites. The axons of the Purkinje cells pass to other cell types and out of the cortex to the deep cerebellar nuclei. Everywhere the discharge of the Purkinje cells has an *inhibitory* effect.

Granule cells are *excitatory* neurones which receive excitation from incoming fibres and send their axons up to the molecular layer to form the parallel fibres.

Golgi, basket and stellate cells are all *inhibitory* interneurones. *Golgi cells* have their cell bodies near the Purkinje cell layer. They inhibit granule cells and their dendrites receive excitation in both the molecular and granular layers. *Basket cells* have their cell bodies in the molecular layer close to the Purkinje cells. Their dendritic trees intersect the parallel fibres which excite them and their axons form a dense meshwork around the initial segment of each Purkinje cell. *Stellate cells* resemble basket cells but they lie more superficially in the molecular layer and their axons terminate on Purkinje cell dendrites.

Deep Intracerebellar Nuclei

These are four masses of grey matter embedded in the white matter of each half of the cerebellum. They lie in line from lateral to medial. The largest, the *dentate nucleus,* which has a convoluted bag-like shape, lies most laterally and the *fastigial nucleus,* a solid grey mass, lies most medially under the vermis forming part of the roof of the fourth ventricle.

Between the fastigial and dentate nuclei on each side are smaller masses of grey matter - the *globose* and *emboliform nuclei* in man, but which in experimental animals form one *nucleus interpositus.*

The computations performed in the cerebellar cortex influence movement and posture via these nuclei. These neurones receive excitatory impulses continuously from branches of all types of incoming afferent fibres on their way to the cerebellar cortex. Superimposed on this activity are periods of inhibition of varying length produced by the inhibitory output of the Purkinje cells related to the particular nucleus. The functioning unit in the cerebellum comprises a group of neurones in a deep intracerebellar nucleus and the neurones in a related region of the cerebellar cortex.

Table 6.1 summarizes these relationship and indicates the final destinations of the nuclear axons. The vestibular nuclei are included because, although they are not intracerebellar, they do receive direct projections from Purkinje cells.

Cerebellar Input-Output Relationships

Each half of the cerebellum receives information from skin, muscles and joints and from the vestibule and the visual and auditory systems on the same side of the body. In addition, the cerebellum samples motor commands from the cerebral and spinal levels.

Information from the *spinal cord* reach the cerebellum by many pathways. Of at least ten tracts four are sufficiently large and well-known to be described here. The *dorsal spinocerebellar tracts* in the dorsal quadrants of the spinal cord and the *ventral spinocerebellar tracts* in the lateral quadrants arise in the thoracolumbar segments of the cord. The *cuneocerebellar* and *rostrospinocerebellar tracts* are the rostral equivalents of these tracts. The information carried by the dorsal spinocerebellar and cuneocerebellar tracts is about muscle length and tension, joint position and skin deformation (*proprioception*). Each half of the cerebellum thus receives information during limb movement about the phase and strength of contraction of individual muscles, the joint angles and the time at which the limb extremity touches the ground. In contrast, the ventral spinocerebellar and rostrospinocerebellar tracts are thought to convey information mainly about the activity of the anterior horn cells in the execution of motor commands, such as the rhythm generation which underlies walking.

Information from the *cerebral cortex* is sent from many areas of the cerebral cortex, including the visual and auditory areas and the motor cortex. The pathways run with the corticospinal and corticobulbar fibres and terminate on the ipsilateral pontine nuclei. Collaterals from corticospinal and corticobulbar fibres also terminate here. These inputs are excitatory.

The *cerebellar peduncles* are composed of afferent and efferent fibres. The *superior peduncle* carries the efferent fibres from the dentate, globose and emboliform nuclei and the afferent fibres from the ventral spinocerebellar and rostrocerebellar tracts. The *middle peduncle* carries afferent fibres only from the contralateral pontine nuclei which are excited by corticofugal fibres. The large *inferior peduncle* carries efferent fibres from the fastigial nuclei and from Purkinje cells travelling to the vestibular nuclei. It also carries afferent fibres

of the dorsal spinocerebellar and cuneocerebellar tracts, and the afferent input from the lateral reticular nuclei and the contralateral inferior olivary nucleus, the neurones of which are excited by fibres from the spinal cord, brain-stem and cerebral cortex.

In the cerebellar cortex only two types of afferent fibres can be recognized - mossy fibres which excite the granule cells and climbing fibres which excite Purkinje cells. *Mossy fibres* have their largest source in the pontine nuclei. The *climbing fibres* are all axons of inferior olivary neurons.

Although the structure of the cerebellar cortex is uniform, three zones may be distinguished if their afferent and efferent connections are examined. These three zones - medial, intermediate and lateral - process information from the vestibular nuclei, the spinal cord and the cerebral cortex respectively. Information from the vestibular nuclei is also processed by the flocculonodular lobe of the cerebellum. The efferent and afferent connections of the cerebellar cortex are summarized in Table 6.1. The *vestibular-processing areas* consisting of the *posterior (flocculonodular) lobe* and the *medial zone (vermis),* are interconnected and are both supplied by and project to the vestibular nucleus. The *spinal-processing area* in the *intermediate zone* lies between the vermis and the lateral reaches of the hemispheres. It is supplied by the spinal cord in a somatotopic fashion and inputs from individual afferents (i.e. from muscle spindles) can be recorded in this zone. The intermediate zone projects to the globose and emboliform nuclei. The *cerebral cortical-processing area* in the *lateral zone* of the cerebellar hemispheres receives input from the cerebral cortex via the contralateral pontine nuclei and projects to the ipsilateral dentate nucleus.

Functions of the Cerebellum

The cerebellum, like the basal ganglia, has long been thought to be concerned with movement because disorders of movement are associated with structural changes in the organ. There are three disturbances that follow damage to particular parts of the cerebellum: (a) *disorders of balance and gait,* (b) *hypotonia* and (c) *incoordination (ataxia).* With incoordination the rate, range and direction of movement are disturbed. An intention tremor, due to a timing disorder of the relations between agonist and antagonist muscles in a movement, is often present. Signs of incoordination are also seen in eye movements (nystagmus) and in speech muscles, producing peculiar speech patterns (staccato or scanning speech).

The region of the cerebellum affected determines the nature of the defect. Impaired balance without incoordination or hypotonia is a sign of flocculonodular lobe damage, i.e., in medulloblastoma, a tumour of children. Lesions of the vermis and intermediate area, particularly in the anterior lobe, are a feature of alcoholic cerebellar degeneration and are associated with incoordination of proximal muscles resulting in disorders of gait and of limb movement. Disorders of the cerebellar hemispheres more laterally cause hypotonia, incoordination and intention tremor of the limbs, particularly of the distal rather than the proximal muscles. Cerebellar disorders are always on the same side as the lesion.

Role of the Ventrolatral Thalamic Nuclei

The ventral division of each thalamus consists of anterior, lateral and posterior nuclei. It is the lateral group which is concerned with motor function. It is well established that the neurones of the ventrolateral nucleus project to the ipsilateral precentral gyrus. The projecting neurones are in turn excited by axons from the contralateral deep cerebellar nuclei and also receive input from the basal ganglia. This input is topographically distributed.

There is thus the potential for complex integration of signals from the basal ganglia and cerebellum with signals from motor cortex representing recent motor commands. Some indication of the importance of this integration is given by the clinical finding that the tremor and involuntary movements of Parkinson's disease can be relieved by lesions of this region.

Motor Cortex

Stimulation of the cerebral hemisphere of man and experimental animals is known to evoke movements on the opposite side of the body. With reduced strength of stimulation or increased depth of anaesthesia the regions from which movement can be evoked narrow down to three, all in the vicinity of the central sulcus. They are known as motor I, II and III.

Motor responses to stimulation are found on either bank of the central sulcus and this area is often referred to as the sensorimotor cortex. The *precentral gyrus* or *motor I (MI)* is, however, thought to be the region most immediately concerned with the generation of movements. The movements evoked by stimulation of MI are always fractional, that is, only part of a full movement, indeed it is impossible to elicit complete or complex movement.

The leg movements are elicited from regions extending onto the medial surface of each hemisphere while movements of the face and scalp muscles are elicited most laterally. This somatotopic representation arises because each region is connected via the corticofugal tracts with the appropriate interneurones and motoneurones to produce muscle contraction of that part of the body. However the size of the representation in the precentral gyrus is proportional not to the density of receptors in the body region but to the number of different movements which the muscles in that region can make.

Motor I (MI) and the adjoining *premotor area* contain giant nerve cells (Betz cells) which together with smaller cells are the source of the corticobulbar and corticospinal tracts. These cells are known as *pyramidal cells* from their shape and they are organized in columns which are oriented perpendicular to the surface. Each column is about 1 mm in diameter and contains many hundreds of pyramidal cells. Each column contains neurones influencing muscles acting on a particular joint. Cortical neurones affecting a particular muscle will be found in several columns, each presumably controlling a different movement. Functionally there appear two types of pyramidal cells in MI. There are larger cells with fast conducting axons which discharge during movement and smaller cells with low conduction velocity which discharge continually though there is also a change during movement. One possibility is that the larger cells control the alpha motor neurones and the smaller affect the gamma motor neurones.

The source of the excitation of MI neurones is not only the input from the ventral lateral thalamic nucleus, but also input from other areas of cortex, notably the postcentral gyrus and the premotor cortex. Presumably part of the organization of muscles for movement is coded by the anatomy of the thalamocortical connections while the pattern of discharge is influenced by the pattern of thalamic input, determined in turn by cerebellar and basal ganglia input, together with input from other parts of the cortex.

Motor II (MII), also known as the *supplementary motor area,* has only recently been investigated. It is found on the medial surface of each hemisphere in the gyrus above the cingulate gyrus and in front of the area from which leg movements are obtained on stimulation. It is much smaller than MI, extending for only about 2 to 3 cm. It was identified first in man and, on stimulation, complex synergistic movements of the contralateral limbs and body, often with vocalization, were produced. These responses were quite unlike the localized contralateral contractions evoked by stimulation of MI. Following lesions of MII, patients grasp reflexly at anything put into their hand. Ablation of MII in monkeys brings about a disturbance of bimanual coordination tasks and recording from cells in MII shows that they discharge with a particular movement whether it is carried out by the right or left hand. A role for MII in the planning and programming of movements appears possible, for recent studies of regional blood flow in man have shown that when thinking about a motor task there is an increased blood flow in both MII areas but no increase in the contralateral MI unless the movement takes place.

The third motor region, *motor III (MIII),* which includes a region known as the frontal eye fields, is found in the premotor cortex in front of the precentral gyrus. Stimulation in the frontal eye field evokes movements of the eyes and sometimes turning of the head in the direction of movement. The commonest movement elicited is a conjugate deviation of the eyes to the contralateral side. Studies of neuronal activity in this region in unanaesthetized monkeys show that cells discharge during voluntary eye movements and destruction of the frontal eye fields prevents voluntary movement of the eyes though reflex movements are unaffected. Neurones of MIII contribute axons to the pyramidal tracts.

6.5 Lesions of the Motor Pathway

A complete transection of the spinal cord will cause permanent loss of all voluntary movement controlled by motor neurones caudal to the plane of section. People with a spinal cord transection above the lumbar but below the cervical enlargement are paralysed in both legs and are said to be *paraplegic.* A section above or in the cervical enlargement below C4 will give rise to paralysis of all four limbs. Such people are called *quadriplegics.* A transection above C4 leads immediately to death as a consequence of respiratory paralysis.

Paralysis or weakness of one side of the body (*hemiplegia*) commonly results from a sudden interruption of the blood supply to an area of the nervous system, causing disruption of the cortical motor pathways to the spinal cord. This is commonly referred to as a 'stroke'. Because of the *crossed* nature of this motor innervation, a hemiplegia signals a lesion of pathways on the opposite side of the brain.

After interruption of the pathways from motor cortex at any level in man there is a period of shock, similar to spinal shock, in which there are no detectable reflexes. It takes 2-4 weeks to recover from the loss of reflexes. Recovery from the paralysis takes much longer and is rarely complete. In particular finger and hand movements are seldom completely regained.

The signs commonly displayed by a patient with hemiplegia include:

1. Muscle weakness without wasting, particularly of distal muscle groups, i.e., finger muscles are more affected than shoulder. Also the arm muscles are commonly more affected than the leg muscles. Because of their bilateral innervation, the muscles which move the eyes, the muscles of the upper third of the face and the trunk muscles are spared in unilateral lesions.

2. Increased muscle tone. Resistance to movement typically increases as the movement continues - the so-called *'spastic' tone.*

3. Increased amplitude of reflexes such as the jaw, biceps, knee and ankle jerks. *Clonus,* i.e., repeated responses to a single stimulus, may be present.

4. Absence of superficial reflexes. The abdominal reflexes - a brisk contraction of the abdominal muscles following stroking of the overlying skin - are lost.

5. Extensor plantar response (*Babinski's sign*). Normally the big toe and all the other toes turn down when the lateral border of the foot is stroked. With interruption of the corticospinal pathway the big toe dorsiflexes instead. This is said to be the most important sign in clinical neurology.

This constellation of signs is known as the *upper motor neurone syndrome.*

Difficulties with speech with a right hemiplegia suggest a left-sided cortical lesion, while the presence of field defects and disorders of sensation may indicate a lesion in the internal capsule and a presence of a third nerve palsy, a mid-brain lesion.

The neurological basis of the signs of hemiplegia is to some extent obvious. For instance the loss of movement and its distribution reflect the interruption of the cortical output to motor neurones. The spastic tone and brisk tendon jerks indicate an increase in excitability of alpha- and gamma-motor neurones. This may be explained in terms of the imbalance of the influences playing on the motor neurones as the result of the loss not only of corticospinal but also of rubrospinal and medullary reticulospinal pathways, leaving the lateral vestibulospinal pathways little affected. There is then a loss of inhibitory influences producing a large increase in the force of the phasic stretch reflexes, especially in the antigravity muscles.

The abnormal reflexes can also be explained by the loss of cortical input. In babies of a few months old, Babinski's sign is present, but as myelination of nervous pathways proceeds and the corticospinal input takes control, the reflex disappears. Its return in hemiplegia is thus no surprise.