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Review Article

Descending Motor Pathways

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ABSTRACT

The brain is the origin of the descending motor pathways or tracts. These are responsible for the origination or modification of motor activity. This action is performed by motor signals, which transmitted from the brain and target lower motor neurons.

Motor control is a chief task of the central nervous system (CNS). It can be recognized as the initiation of signals to coordinate muscle contraction of the body and head, aiming to keep a posture or to make a movement (transition between two postures). A large amount of the CNS is included in the process of motor control.

The term "motor neuron, also known as motoneurons" may refer to single or one type of neurons responsible for the movement. However, this is not the truth. Actually, the "motor neuron" describe two categories of neurons, the "upper and lower motor neurons". They are significantly different in their origins, synapses, pathways, neurotransmitters and developed lesions due to their affection. Motor neurons are essential constituents of two different circuits "the upper and lower motor circuits". These circuits control both voluntary and involuntary movements. This was achieved by the connection of higher centers with muscles and glands

Descending tracts of the motor system are originating mainly from two parts, the cortex and the brainstem. Tracts originating from the cortex control the voluntary movements and adjust posture relating to voluntary movement. Furthermore, the descending pathways are subdivided into (pathways) describing their start and termination. These are (1) corticospinal and corticobulbar, (2) cortico-reticulo-spinal, (3) cortico-rubro-spinal (4) cortico-tecto-spinal, (5) vestibulo-spinal and (6) raphe–spinal and ceruleus–spinal tracts (aminergic pathways)

Pyramidal tracts, originating in the cortex and carry motor fibres to the spinal cord and brain stem. It is subdivided into corticospinal and Corticobulbar tracts. The extrapyramidal tracts originate in the brainstem. Then carry motor projections to the spinal cord. These tracts are primarily concerned with the control of the involuntary and automatic innervations for all musculature (e.g., muscle tone, balance, posture and locomotion).

Keywords: Motor Neuron; Tracts: Motor Cortex; Pyramidal; Extrapyramidal.

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INTRODUCTION

The brain is the origin of the descending motor pathways or tracts. These are responsible for the origination or modification of motor activity. This action is performed by motor signals, which transmitted from the brain and target lower motor neurons **(1).**

Movements are the chief way by which subjects Interact with the surrounding world. Most activities (e.g., running, eating, talking, writing, and reading) eventually include motor acts. Motor control is a chief task of the central nervous system (CNS). It can be recognized as the initiation of signals to coordinate muscle contraction of the body and head, aiming to keep a posture or to make a movement (transition between two postures). A large amount of the CNS is included in the process of motor control **(2).**

The term "motor neuron, also known as motoneurons" may refer to single or one type of neurons responsible for the movement. However, this is not the truth. Actually, the "motor neuron" describe two categories of neurons, the "upper and lower motor neurons". They are significantly different in their origins, synapses, pathways, neuro-transmitters and developed lesions due to their affection **(3).**

Motor neurons are essential constituents of two different circuits "the upper and lower motor circuits". These circuits control both voluntary and involuntary movements. This was achieved by the connection of higher centers with muscles and glands **(4).** For example, glutamate is the main neurotransmitter in upper motor neurons, while acetylcholine is the main transmitter in lower motor neurons **(5).**

In addition, the upper Motor Neurons starting in the cerebral cortex and descending down to the brain stem or spinal cord, while lower motor neurons starting in the spinal cord and extends to innervate glands and muscles all over the body. The understanding and differentiation upper from lower motor neurons and their tracts is crucial for the diagnosis of neuronal injuries and localization of the lesions (figure 1) **(4).**

The commands produced by the brain to muscles are transmitted by motor neurons through neuronal fibres known as "descending fibers" or pathways. Thus, descending fibers describes fibers travel from the brain to the spinal cord. These pathways originating mainly from the cortex and many sites from the brainstem. The main functions of these pathways include movement control, muscle tone and posture, modification of the spinal reflexes, and controlling transmission of afferent data to the higher centers. In addition, these pathways mediate the control over the autonomic neurons in the spinal cord **(6).**

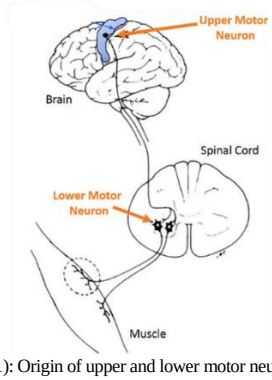

Figure (1): Origin of upper and lower motor neurons **(7).**

The cell bodies of upper motor neurons (known as Betz cells) (figure 2) are located in the primary motor cerebral cortex (mainly in precentral gyrus). It is deeply situated in the layer 5 of the cortex, with long apical dendrites extending upwards into the upper layers of the cortex. The signal originating from these cells is the first step for initiation of movement. Then transmitted down through descending tracts to the targeted muscles **(8,9).**

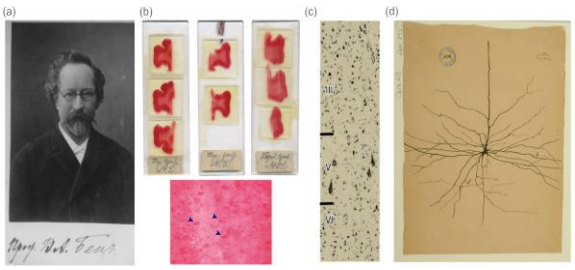

Figure (2): Betz cells as in the original preparations by Betz, Brodmann, and Ramòn y Cajal. (a) Portrait of Vladimir Betz with his Cyrillic name (signature) in his self-published Atlas of the Human Brain (published 1879). (b) Betz's original slide preparations stained with his new carmine method. Arrowheads designate the "Riesenpyramiden." (c) Brodmann's original micrograph portraying the diverse layers of the area 4 (primary motor cortex); note area 4-defining Betz cells in layer Vb. (d) Original sketch by Ramòny Cajal of a giant pyramid of the motor cortex **(8).**

Classification of descending motor pathways

The descending motor pathways were subdivided into pyramidal and extra-pyramidal tracts. This terminology showed a clinical differentiation between pyramidal and extrapyramidal tract diseases **(10).**

Another way to classify the motor pathways is based on their end in the cord and their function in controlling movements and posture. For example, the lateral pathways end in the lateral portions of the gray matter in the spinal cord. These pathways can stimulate motor neurons straight, irrespective of the fact that, the interneurons are the main target of these pathways. They controls the reflex arcs controlling fine movements of the distal ends of the limbs and activating supporting muscles in the proximal ends of the limbs. On the other side, the medial pathways ends in the medial ventral horns of the cord, on the medial group of interneurons, which connect bilaterally with motor neurons controlling the axial muscles of the balance and posture

control. In addition, these pathways contributing in the control of some proximal limb muscles [2]. Furthermore, the descending pathways are subdivided into (pathways) describing their start and termination. These are (1) corticospinal and corticobulbar, (2) cortico-reticulo-spinal, (3) cortico-rubro-spinal (4) cortico-tecto-spinal, (5) vestibulo-spinal and (6) raphe–spinal and ceruleus–spinal tracts (aminergic pathways) (Figure 3) [11].

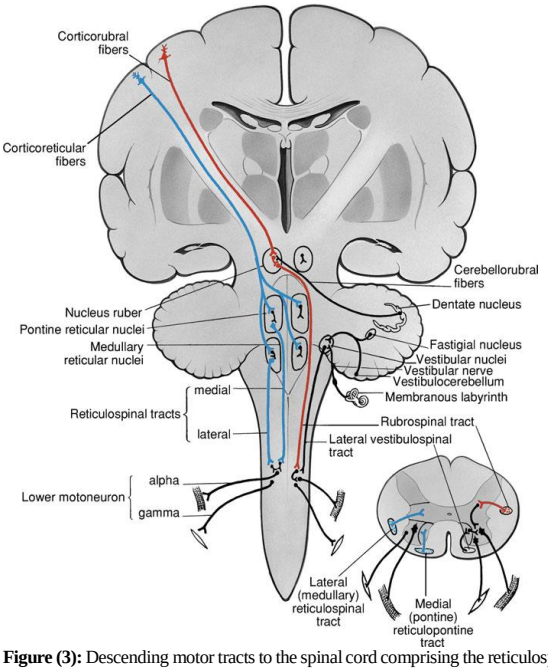

Figure (3): Descending motor tracts to the spinal cord comprising the reticulospinal tracts (corticoreticulospinal pathways), rubrospinal tracts (corticorubrospinal tracts), and vestibulospinal pathways. The corticoreticular fibers end bilaterally but with a slight contralateral preponderance [11].

Motor cortex: the origin of descending tracts

The cerebral cortex areas can be categorized into three main areas from the functional point of view, the motor, the sensory and the association areas. As their name refers to, the motor areas are concerned with the activities of motor control, the sensory areas responsible for receiving sensory information, while association zones are responsible for associative, cognitive and integrative functions [12, 13].

Precentral gyrus (motor area 4) primary motor area

The basic understanding and data about the primary motor area was described by Walter Penfield. This was completed by electrical stimulation of the cerebrum surface. This was performed under local anesthesia to permit observation of the response to the stimulation. This led to the detection of the fact that, the muscle movements were directly stimulated from the precentral gyrus. However, it is now known that the primary motor cortex collects data from many zones to aid in the planning of movements. The main output stimulates the spinal cord neurons to activate contraction of the skeletal muscle. The muscles of the fine and agile movements (e.g., fingers and lower face muscles) received the greatest amount of cortical space. On the other side, the "power muscles" performing coarser movements (e.g., the muscles of the back and buttock), are represented by the much less space on the motor cortex. The precentral gyrus is located on the lateral surface of both frontal lobes of the cortex, anterior to the central sulcus. Then, it runs parallel to the central sulcus and extends to the precentral sulcus [14]. The primary motor cortex is situated within the precentral gyrus. It is concerned with controlling the voluntary movements. Several motor tracts originate within it (e.g., corticospinal, corticobulbar, and cortico-rubrospinal tracts) all start within the precentral gyrus) [6, 15]. The primary motor area showed the lowest threshold for stimulating the muscle contraction on the contralateral side [16].

The primary motor area is also termed as "Brodmann area 4" and it represented the primary motor cortex of the brain in humans. The margins of the area are the precentral sulcus anteriorly, medial longitudinal fissure medially, central sulcus posteriorly, and lateral sulcus laterally [17]. It has the design of a "homunculus", as the legs and trunk fold over the midline, while the arms and hands are situated on the lateral surface. The topographical map of the contralateral half of the body is presented in an inverted pattern in the primary motor area. It is mainly concerned with motor function. However, it plays a role in sensory perceptions. Lesions of this area may lead to paralysis and reduced somatic sensation [18].

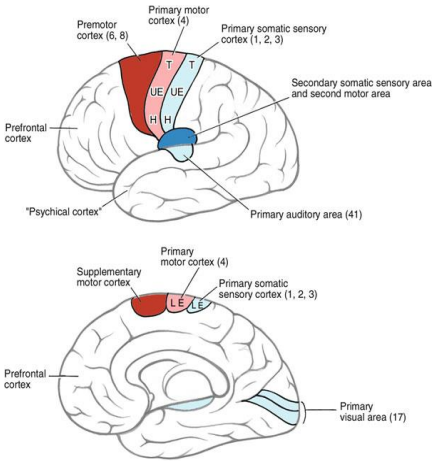

Figure (4): The different functional areas of the cortex. The body parts are represented on the primary motor and somatic sensory cortices comprises the head (H), upper extremity (UE), trunk (T), and lower extremity (LE). Numbers represent areas according to the classification of Brodmann (14).

The mapping of body areas in the motor area 4 (figure 5) is related to precision and skill of movements it controls rather than the physical size of the region. For example, face and hand muscles occupy large areas of the primary motor cortex while the muscles of thigh occupies a small area. The head is represented in the most lateral and inferior area, while the hand is represented in the central part. Furthermore, the leg and foots are represented in the medial surface of the paracentral lobule [19].

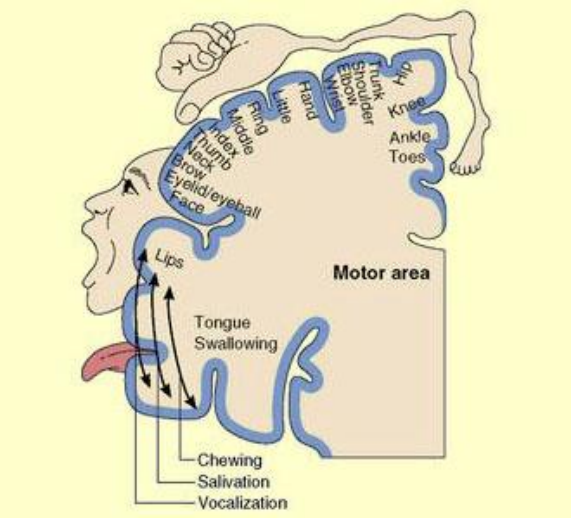

Figure (5): The mapping of body areas in the motor area 4 [20].

The stimulation of primary motor area led to distinct repetitive movement of focal nature. This reflected the stimulated area (e.g., repetitive jerks of the thumb or of the foot). Sometimes, a march of focal movements may occur (e.g., the movement is started in the thumb for example and then spread to hand, arm, face and legs, etc). When injured, an upper motor neuron weakness led to a contralateral monoplegia or hemiparesis [21]**.** The stimulation of the giant pyramidal cells of the primary motor area produces movements in definite directions (e.g., flexion, extension, abduction, and adduction). These movements are due to muscle contraction at a specific joint. The primary motor area also receives data from somatosensory cortex (of the postcentral gyrus and the ventral lateral thalamic nucleus). This data may permit the motor cortex response to peripheral stimuli in what is called "long loop reflexes" [8].

The precentral gyrus received its blood supply from the anterior cerebral and the middle cerebral arteries. Both are branches from the internal carotid artery. The lateral surface of the precentral gyrus is supplied by the superior division of the middle cerebral artery. The medial aspect of the precentral gyrus is supplied by the anterior cerebral artery. The majority of the surface of the cerebral cortex, as well as the precentral gyrus are drained by the superficial cerebral veins. Then, the veins are drained into the superior sagittal sinus [22].

In cases of stroke, the precentral gyrus may be affected. Symptoms could help in localization of the site of the lesion. For example, if the superior division of the middle cerebral artery was affected, it will affect the more lateral aspects of the precentral gyrus, which appeared clinically in the form of weakness in the contralateral face and arm, with signs of upper motor neuron disease. If the middle cerebral artery affected, it led to inclusion of other areas besides the precentral gyrus. This is also clinical presented with contralateral hemisensory loss of the face and arm. If the dominant hemisphere affected, Broca's aphasia may be detected. A stoke of anterior cerebral artery will led to affection of the more medial precentral gyrus aspects. Clinically, it is presented as a weakness of the contralateral led with signs of motor neuron involvement. A stroke of the anterior cerebral artery may be presented with a hemisensory loss of the contralateral leg [23].

Betz Cells

Betz cells, (also recognized as "the pyramidal cells of Betz"), are giant pyramidal neurons found within the 5th layer of the grey matter in the primary motor cortex. These cells are the largest in the CNS, sometimes reaching 100 μm in diameter. Betz cells are upper motor neurons, their axons extended down to the spinal cord through the corticospinal tract. In humans, these cells synapse directly with anterior horn cells of the spinal cord, which in turn synapse with their target muscles [8].

Layers (lamination) of the cerebral cortex

On microscopic examination, the following horizontal cellular layers (Laminae) are detected

1. *Layer – I (Molecular (Plexiform) layer)*. It is consisted of the apical dendritic tufts of pyramidal neurons, horizontally organized axons, Cajal-Retzius cells and glial cells.
2. *Layer- II (External granular layer)*: consisted mainly of granular cells and small pyramidal neurons.
3. *Layer –III (External pyramidal layer)*: consisted of small and medium-sized pyramidal neurons
4. *Internal granular layer IV*: consisted of and contains small round granular and small pyramidal neurons
5. *Internal pyramidal layer V*: contains the largest pyramidal neurons. Axons of these cells leave the cortex to reach subcortical zones
6. *Multiform (Polymorphous) layer VI:* consisted mainly of excitatory cells and inhibitory interneurons [24,25]

The Premotor area (BA6)

It is located anteriorly to the primary motor area, occupying the posterior part of the superior, middle, and inferior frontal gyri. It extends on the medial surface of the cortical hemisphere [26]**.**

The cells of Betz do not present in this zone. However, this area is the chief cortical origin of the extrapyramidal tracts**.** It receives sensory signals and stores the past data. On the basis of previous studies, it programs planned motor movements (i.e., activities). Thus, it has a significant role in

voluntary skilled movements [27]. Area 6 (the premotor area) contains a second motor map. It appears to be accountable for the programming the intended movements and controlling movements in progress. It is divisible into two areas (i.e., a dorsal and a ventral areas). The dorsal area is responsible for the movements started by the subject, while the ventral area is responsible for the control of movements due to external stimuli[28]. The stimulation of this area led to specific movement patterns. For example, tonic rotation of the head, eyes, and trunk to the contralateral side. This is associated with tonic abduction of the arm at the shoulder joint and flexion of the arm at the elbow joint [29]. The lesion of this area is presented by a) limb kinetic apraxia, clumsiness of movement without weakness (b) Severe spasticity with mild hemiparesis, (c) Appearance of released reflexes [30].

The supplementary motor area (SMA, MsII)

It lies on the medial surface of the cerebral hemisphere, in front of the paracentral lobule [16]. However, it sends extensions to Broadmann's areas 4, 6 and 8 on to the medial cortical surface. The body is represented in the supplementary motor area from anterior to posterior in craniocaudal order [27]. The supplementary motor area is responsible for control of complex tasks movements which need retrieval of memory and temporal organization of consecutive movements [31]. The lesions of this area led to a transient release of "primitive" reflexes mediated by the primary motor cortex (e.g., instinctive grasp) [28].

The Presupplementary motor area (Pre SMA)

It situates anterior to the supplementary motor zone on the medial cortical surface [28].

The frontal eye field

The frontal eye field (FEF, BA8) is situated near the junction of the precentral and middle frontal sulci. The FEF is concerned with the voluntary scanning movements of the eye (Conjugate eye movement) and moving eyes to the opposite side [16]. The FEF is connected to the superior colliculus, substantia nigra and the dentate nucleus of the cerebellum through the thalamo-cortical pathways. It receives data (input) from the parietal, temporal, and occipital lobe visual area, in addition to the middle temporal area [32]. The FEF comprises parts of areas 6, 8, and 9. Stimulation of FEF causes movement of both eyes to the opposite side (what is called conjugate movements). In addition pupillary dilatation and head movements may also occur. FEF is connected to the cerebral cortex of the occipital lobe (lobe concerned with vision). It is also had thalamic connection (medial dorsal nucleus). The FEF and the motor speech area (of Broca) are parts of the premotor area. Lesions led to a transient paralysis of the voluntary conjugate gaze to the contralateral visual field [33].

Broca's motor speech area

Broca's motor speech area 44 (BA44) is situated at the pars triangularis, and Broca's motor speech area 45 (BA45) is positioned at pars opercularis in the inferior frontal gyrus on the dominant hemisphere [27]. The posterior and middle superior temporal gyri including association areas, as well as posterior parietal and middle temporal cortices sends impulses and fibres to the Broca's speech areas [34]. At the same time, this area sends impulses to the SMA, FEF and the dorsal premotor cortex [35]. This area controls the expressive speech and gets out the data of words through connection with the primary motor area. Thus, motor aphasia developed as a result of lesions to this area, with specific characterization of the inability to express speech [19]. These effects are associated with the lesion of dominant hemisphere [33]. This language defect is associated with a contralateral hemiparesis [29]. Lesions of varies areas of the cerebral cortex and their effects are summarized in the next table [33].

Table (1): Lesions of Various Areas in the Cerebral Cortex (Brodmann no. in Brackets) and Their Effects [33].

Site of lesion	Features of the lesions
Area 4, the primary motor area	***Epileptic fits***–Due to irritative lesion ***Hemiplegia*** (contralateral flaccid paralysis)
Area 6, premotor area	***Apraxia*** (clinically manifested by difficulty in performance of the skilled movements)
Area 8, FEF	Contralateral loss of the voluntary ***conjugate movements of the*** and the eye deviates to the side of lesion. However, pursuit movements on both sides are normal, as they are controlled by the occipital lobe.
Areas 44, 45, Broca's motor speech area	***Expressive/motor aphasia***–Difficulty in spoken speech or writing (***agraphia***). Non-fluent speech and telegraphic language. Keywords spoken are normal
Supplementary motor area (M II)	Non-permanent loss of movement, with bilateral ***flexor hypotonia***

Upper motor neurons (UMNs)

The pyramidal tract is the primary tract concerned with carriage of signals for voluntary movement. It is not a single tract, but subdivided into pathways, cortico-spinal and cortico-bulbar tracts. The UMN integrates both the excitatory and inhibitory cortical signals and translate these into one signal initiating or inhibiting voluntary movement. UMN also under regulation by the thalamo-cortical and callosal projection neurons. The exact mechanism of this regulation is not yet fully understood. However, it is believed that, the majority of excitatory signals to these neurons are originating from neurons in the second, third and fifth layers of the motor cortex [36]. The axons of the UMN travel down and pass through the posterior limb of the internal capsule. Then, the axons continue through the cerebral peduncles in the midbrain, longitudinal pontine fibers, and finally the medullary pyramids, where 90% of the fibers decussate and

continue down the spinal cord as the lateral CTS (LCST) on the contralateral side. The LCST is the largest descending tract and is located in the lateral funiculus. In the anterior horn of the spinal cord, the LCST directly synapse onto the lower motor neuron (LMN) [37].

The pyramidal tract small fibers, which do not decussate are continued as the anterior cortico-spinal tract. This tract is located close to the anterior median fissure. It is concerned with the movement and controlling of axial and proximal limb muscles, which contributes to the posture. This tract crosses at the spinal level, where it is innervated [36]. The CTS controls many functions and the anatomical data supports this multifunctional view. The CST originates from a wide variety of cortical areas, each concerned with specific function (e.g., the primary motor cortex (M1), the ventral and dorsal premotor cortical areas, supplementary motor area (SMA), and cingulate motor areas [38].

The CST is vital for the expression and control of the precise and voluntary movements. It originates from lamina V of the cortex and three specific sites of the cortex. About 30% arise from the primary motor cortex (M1, area 4) in the precentral gyrus. Less than 5% of the axons of this particular area belong to the cells of Betz and this region is involved in the real execution of a movement. Another 30% of the fibers originates from the premotor cortex (PMC) and SMA located on the lateral and medial surfaces of the cortical area 6, respectively. These areas, especially the SMA, are involved in the process of planning and start of motor movements. The remaining 40% of these fibers arise from the primary somatosensory cortex (S1, areas 3, 1, and 2) in the post-central gyrus and with area 5 and area 7 of the posterior aspect of the parietal cortex. These regions constitute a connection through association fibers with areas 4 and 6. In addition, these regions provide cortical representation of space. This representation is crucial for eye movements and precision grasping movements (1). In short, the pyramidal tract starts from all 4 neocortical lobes [33].

The majority of cells bodies of the pyramidal tract UMNs are located in the precentral motor cortex (Brodmann area 4) and the premotor area (Brodmann area 6). Cell bodies are also located in the SMA, primary somatosensory cortex, and the superior parietal lobe [39].

The motor output from the cerebral cortex is transmitted to the brain stem and spinal cord and the main function is to control the muscles through motor neurons. Betz cells, the main and largest cells in the origin of pyramidal tract are large neurons, which synapse with LMN cells in the brain stem or in the spinal cord. Thus, two tracts are formed by the axons of Betz cells (the cortico-bulbar and the cortico-spinal tracts). The name of both tracts is related to the origin in the cortex and their end, either the brain stem (bulb) or the spinal cord, respectively [40].

The tract descends through corona radiata followed by the posterior limb of the internal capsule. It continues in the middle three-fifths of the cerebral peduncle (crus cerebri) in the midbrain and then to the basilar part of the pons (called pontine enlargement). In the pons, the fibers are divided into smaller axon bundles and then grouped together to form the pyramids of the medulla. Then, descending through the spinal cord [1].

Although, most axons of the CST arise in the motor cortex, some fibers from the CTS originate in the sensory cerebral cortex, and end in the dorsal column nuclei. This may be associated with the modification of their function, and acting to filter incoming sensory stimuli [41].

UMNs are first-order neurons. They are responsible for transmission of the electrical impulses to initiate, control and coordinate different movements. The pyramidal tract is the major UMN tract initiating the voluntary movement. The pyramidal tract offers a direct pathway between cortex and spinal cord. However, extra-pyramidal tracts provide indirect pathways for movement coordination. The pyramidal tract splits into the CST and the cortico-bulbar (CBT). CST fibers synapse with spinal nerves while CBT synapse with cranial nerves [42].

The CBT also includes fibres from distinct regions from across the cerebral cortex (not limited to the frontal lobes) [43]. The main function of the tracts descending from the cerebral cortex is to control LMNs (either anterior horn cells of the spinal cord or motor nuclei of the cranial nerves in the brainstem). These descending pathways innervates the limbs and the trunk. Beyond their main function of controlling LMNs, the descending pathways have a number of other functions that are of utmost importance for control of movement (e.g., provide gating for spinal reflexes; responsible for the descending Effects on afferent (ascending, sensory) systems; and perform a trophic, or nutritive, function for the neuron groups they come in contact with [44].

The head is presented in the precentral gyrus (area 4), in a lower down fashion. This is followed by neck, upper limb, trunk and lower limb (inverted motor homunculus). However, the region beyond the knee and perineum are represented on the medial surface of cerebral hemisphere in the anterior part of the para-central lobule [33]. Then, fibres pathways through corona radiata to reach the internal **capsule (lie in the posterior** limb). However, the genu of the internal capsule included fibers of the cortico-nuclear fibres to the head region [44].

As the fibres for head are represented in the most anterior and the lower limb are most posterior, the inferior-to-superior representation in the motor cerebral cortex have become anterior-to-posterior. The fibres have rotated a 90°, and by the time these fibers have reached the internal capsule [33].

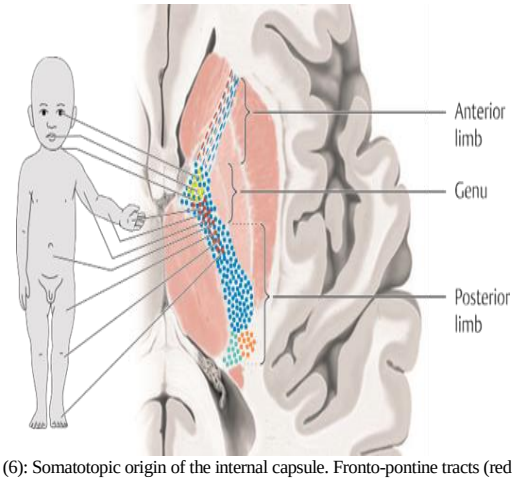

Figure (6): Somatotopic origin of the internal capsule. Fronto-pontine tracts (red dashes); anterior thalamic peduncles (blue dashes); cortico-nuclear fibers (yellow dots); cortico-spinal (red dots); posterior thalamic peduncle (blue dots); temoralontine tracts (orange dots) (45).

Internal capsule is supplied by terminal arteries. When stroke occurs, the severity depends on the lesion site (location) in the internal capsule. Common clinical presentations are contralateral hemiplegia, sensory deficits and contralateral hemianopsia [46]. After passage in the internal capsule, fibres enter the crus cerebri in the midbrain and occupy middle two thirds of the crus. Frontopontine fibers in their way to cerebellum occupy the medial one sixth, while parietooccipital and temporopontine fibers occupy the lateral one sixth. Fibers of the head and fibers for the lower limb are the most medial and posterior in the crus cerebri, respectively. Thus, fibers rotate a further 90º to reach the midbrain by the time. Then, fibers descend on the ventral part of the pons and get scattered by different pontine nuclei and transversely running pontocerebellar fibres. By time, these fibers reach the medulla and regrouped to enter the pyramids in the upper aspect of the medulla. The majority of fibers (about 80-90%) cross to the opposite side near the lower end of the medulla forming the decussation of the pyramids and fibers of the upper limb cross at a slightly higher levels than the fibers of the lower limb [33].

The fibres crossed to the opposite side in the medulla then enter the lateral funiculus of the spinal cord and complete their course as the lateral CST. The fibres of this tract end in grey matter at different levels of the spinal cord. Most of them end by synapsing with neurons in the bases of the dorsal and ventral grey columns and impulses are transmitted to the ventral horn cells. However, some fibers end directly on the ventral horn cells. The lateral CST mainly controls the movement of the distal muscle of the limb [47]. The CTS fibers are arranged from medial to lateral starting by cervical, as the most medial, then thoracic, lumbar and sacral. The non-decussating fibers enter the spinal cord through the anterior funiculus forming the anterior CST. When reaching the appropriate level in the spinal cord, the fibers cross to the contralateral side (through the anterior white commissure) to attach the grey matter on the contralateral side of the cord. They terminating in a manner similar to the fibers of the lateral CST. However, some fibers terminate on the same side also. The anterior CTSs are concerned with the movement of the trunk and proximal (postural) muscles of the limb. About 25% of corticospinal fibers are concerned with the movement control of the upper limb, 20% for the trunk and 25% of the lower limb [33].

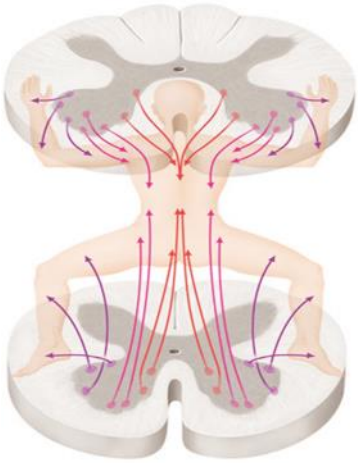

Figure (7) Somatotopic organization of nuclear columns of the anterior horns (45).

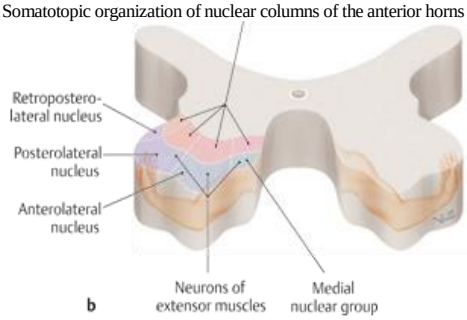

Figure (8): Enlargement of the cervical cord showing the medial-to-lateral and anterior-to-posterior organization of motor nuclei [45].

The pyramidal tract had approximately one million fibers. These fibers originating from area 4 (31%), the premotor cortex of area 6 (29%) and 40% from areas 3, 1, 2, 5, and 7 of the parietal lobe. The number of fibers with large diameter (9–22 μ) is about 30,000 and roughly equal the number of the giant cells of the pyramidal tract. Area 4 represent the main origin of these large fibers. Areas give origin to the pyramidal tract also give rise to other descending motor pathways (e.g., corticoreticular, cortico-rubral, corticostriate and cortico-thalamic tracts) [29]. Descending motor pathways can also categorized in medial and lateral descending motor systems. The medial motor system consisted of four medial systems. These systems are anterior CTS, the vestibulospinal, the reticulospinal, and the tectospinal tracts. These tracts are mainly concerned with controlling the proximal axial and girdle muscles responsible for postural tone, balance, orienting movements of the head and neck, and automatic gait-related movements. The medial motor systems descend ipsilaterally or bilaterally. Some extend only to the upper few cervical segments. The medial motor systems end on interneurons project to both sides of the cord. These are responsible for the control of movements involving multiple bilateral spinal segments. Thus, unilateral lesions of the

medial motor systems led to no obvious deficits. In contrast, lesions of the lateral CST led to dramatic deficits. The rubrospinal tract is small, and its clinical importance in humans is uncertain. However, it may share in taking over functions after corticospinal injury. It may also play a role in flexor posturing of the upper limbs, which is typically seen with lesions above the level of the red nuclei, in which the rubrospinal tract is spared [(48)].

Lateral CST

After decussation, the fibers of lateral CST descends down the spinal cord in the lateral funiculus. The lateral funiculus is organized with the fibers supplying the sacral region, which are the most lateral, followed by the thoracic, lumbar, and then cervical fibers traveling more towards the midline. These fibers synapse directly and indirectly, on alpha and gamma LMNs, especially those associated with more distal muscles [(15)]. At the level of the pyramid, 90% of the tract fibers decussate and descend through the posterior portion of the contralateral lateral funiculus of the cord as the lateral CST. The fibers then descend through all levels of the spinal cord, with the axons eventually terminate on interneurons of the lateral aspect of laminae IV–VII [(49)].

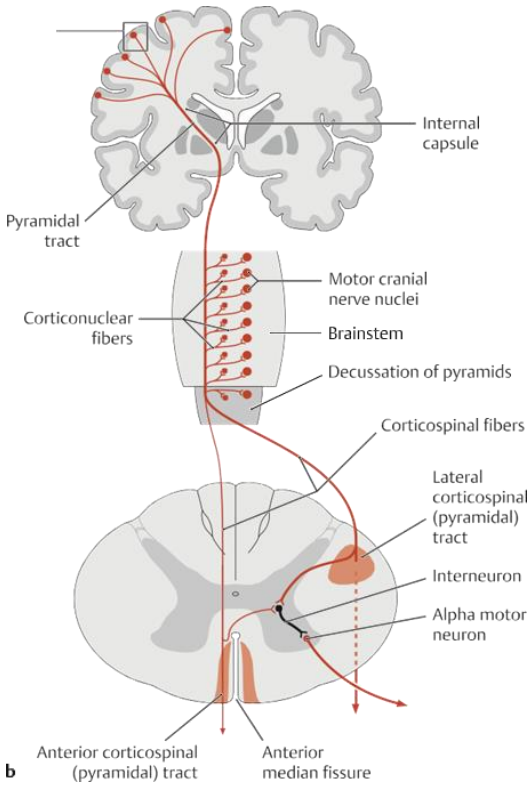

Figure (9): Course of the CST (45).

Ventral CST

At the pyramidal decussation, approximately 10% of the axons of the CST do not decussate and descent down on the ipsilateral side. Then, fibers enter the spinal cord through the ventral aspect and are known as the anterior CST. However, most of them crossed to the contralateral side as fibers descend. The decussation occur mostly through the anterior white commissure, then synapsing with interneurons [(36)]. Axons of the anterior CST usually synapse on the lower motor neurons supplying more medial muscles [(15, 48)].

Movements of the trunk include both sides of the body. Thus, fibers of the anterior CST are not entirely contralateral. Some collateral fibers project into the ipsilateral ventral horn to control synergistic muscles on this side of the body, or to inhibit antagonistic muscles within the ventral horn. The CTS coordinates the postural muscles in broad movements of the body as they affect both sides of the body. These coordinating fibers of the anterior CST are often treated as bilateral, as they are ipsilateral and contralateral at the same time [(40)]. Below L2, the only innervations to LMNs are from the lateral CST and the myotomes are related to the lower limb (no more trunk) [(39,50)].

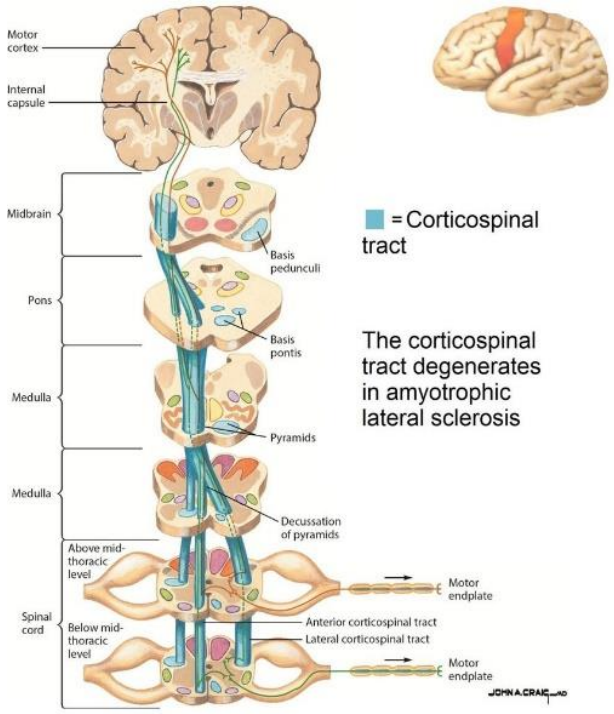

Figure (10): the CST.

The Corticobulbar Tract (CBT)

The CBT is a similar tract to the CST, except instead of supplying the muscles of the body, it innervates the head and face muscles. This tract mainly initiated in the lateral aspect of the primary motor cortex, within the precentral gyrus (PCG). Then, it travels through the genu of the internal capsule, cerebral peduncle, basis pontis, and the medullary pyramids on the ipsilateral side. Fibers then synapse on the motor nuclei of cranial nerves, in direct and indirect pattern through interneurons [(15)].

The CST sends direct fibers bilaterally to the hypoglossal, facial, and trigeminal nerve's motor nuclei. The facial nerve motor nucleus nerve is a special case in this tract. The part supplies muscles around the mouth and lower face receives only contralateral fibers from the CBT. On the other side, part that supplies the forehead and upper face muscles receives bilateral fibers originating from both sides [(15)].

The CBT fibers arise partially from the area 4 just above the lateral fissure of lateral surface of the cerebral hemisphere. This area is related to the head of the motor homunculus. The axons of CBT accompany those of the CTS until the level of the pons. Then, the axons start to travel dorsally through the brainstem tegmentum before attaining the motor nuclei of the trigeminal, facial, glossopharyngeal, vagus, spinal accessory and hypoglossal nerves. The CTB fibers are situated in the genu of the internal capsule and are the most medial of all cortically arising UMNs in the cerebral peduncle. This tract supplies the cranial nerve LMNs bilaterally and for most cases the muscles are unable to be contracted voluntarily on one side only. The facial (below the eye) and hypoglossal nuclei receive the majority of their supply from the contralateral cortex. Generally, not considered part of the CBT system would be the motor nerve supply to the oculomotor, trochlear and abducent motor nuclei. They instead receive direct supply from area 8 (frontal eye fields). Area 8 input is not direct but instead passes through gaze centers located in the reticular formation [(1)].

Only 50% of the CBT fibers decussate, in contrast to those of the CST where most of fibers decussate at the level of the internal capsule. Cranial nerve nuclei supplying the skeletal muscle thereby receive bilateral first-order neuron innervation (i.e. from both hemispheres) [(43)].

Fibers of the CTB converge predominantly in the genu (but may also lodge in the most anterior part of the posterior limb) of internal capsule from which they descend together with CST fibers. An additional constituent of the CBT is a subset of cortical fibers that supply the brainstem nuclei, and then exert descending influences on the neurons of the spinal cord. These descending fibers end in the reticular formation, the superior colllculus, and the red nucleus. These connections coordinate the cortical and brainstem motor systems [(44)].

Descending pathways function (single or multiple?):

A single pathway, from the neuroanatomical point of view, can carry out many and different functions. The CST offers an excellent example. It is included in the following functions (a) descending control of afferent nociceptive inputs; (b) actions on spinal reflexes (e.g., selection, gating, and gain control); (c) excitation or inhibition of motoneurons; (d) autonomic control; (e) long-term plasticity of the spinal cord circuits and 6) trophic roles [(19)].

The UMNs extend downward as the pyramidal tract, where it obtains the blood supply from the lenticulostriate arteries. When tract reaches the brainstem, its main blood supply is provided by the paramedian branches of the basilar artery. In the region of the caudal medulla, the anterior spinal artery provides the most of blood supply. This artery continues to supply the lateral and anterior CST and the anterior horn cells (AHCs) in the spinal cord [(19)].

Lower motor neurons (LMNs)

The Function of LMNs

The LMN is vital for transmitting signals from the ULM to the effector muscle, enabling movement. There are three main types of LMNs:

1. Somatic Motor Neurons
2. Special Visceral Efferent (Branchial) Motor Neurons
3. General Visceral Motor Neurons [(5)]

Somatic Motor Neurons (SMNs)

SMNs are situated in the brainstem and can be categorized into three classes: alpha, beta, and gamma. LMNs are categorized on the basis of size and targets as follows: (1) alpha motor neurons (AMNs) have large cell bodies and axons supplying the skeletal muscle and are the commonest type of the LMN; (2) gamma motor neurons (GMNs) have smaller diameter of the axon fiber and supply specialized structures known as muscle spindles which help in the control of muscle stretch reflexes; and (3) beta motor neurons (BMNs) are less abundant and supply muscle fibers inside and outside muscle spindles [(48)].

Alpha Motor Neurons (AMNs): AMNs supplies extrafusal muscle fibers. They are the principle drivers of the skeletal muscle contraction. The large cell bodies of AMNs may be found in the brainstem or the spinal cord. These cells bodies in the spinal cord are located in the anterior horns. This is why they are referred to as anterior horn cells (AHCs). Each AHC sends out a single axon that supplies multiple muscle fibers within a single muscle. The muscle fibers supplied by AMNs are nearly identical in their properties. This permits the control and synchronous movement of the motor unit when the LMN is depolarized [(51)].

Beta Motor Neurons (BMNs): BMNs are not well understood, but they are known to supply both extrafusal and intrafusal muscle fibers [(5)]

Gamma Motor Neurons (GMNs): GMNs supply muscle spindles and regulate their sensitivity. These neurons essentially respond to the stretching of the muscle spindle. Although they are called "motor neurons," GMNs do not

directly produce motor functions. Instead, they are activated alongside AMNs to fine-tune muscle contractions by a process known as alpha-gamma co-activation. Disruptions in alpha or gamma motor neurons can lead to problems with muscle tone [52]

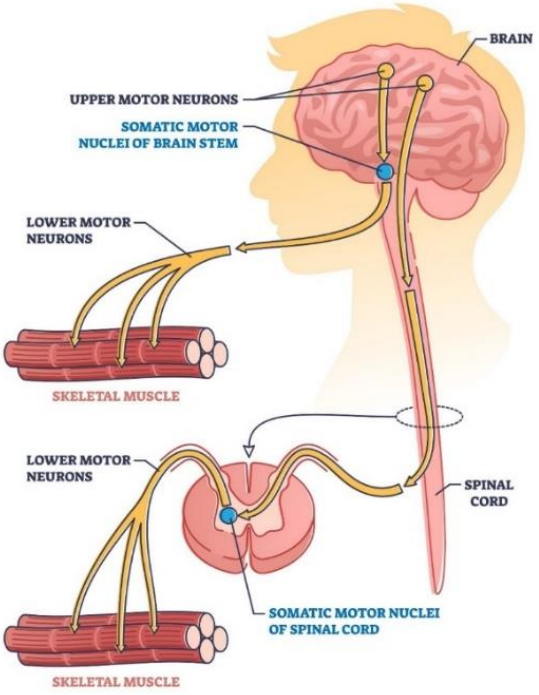

Figure (11): Somatic nervous system [53]

The motor unit—

The motor unit describes the motor neuron, together with its axon and all the muscle fibres it innervates, including the neuro-muscular junction (NMJ). There is a somatotopic organization in the AHCs in the ventral grey horn of the spinal cord. Neurons regulating the neck muscles, are situated in mesial columns; neurons controlling proximal muscles are located in the mid-region; and neurons regulating the musculature of the distal aspect of the limbs are situated in laterally placed columns [30].

In addition, a motor unit can be recognized as the single AMN and the muscle fibers that it supplies. The muscle fibers of a single motor unit will be found scattered throughout the muscle. Fine precision movements in small hand and extra-ocular muscles are possible as they will have motor units occupying only a few fibers of a muscle. A large bulky muscle (e.g., the gastrocnemius) will have a motor unit that supplies hundreds of muscle fibers. The type A-alpha myelinated nerve fibers arising from the ventral horns of the cord or motor nuclei of cranial nerves will pass in a skeletal muscle and give many branches. A single branch will end on a muscle fiber at a site called the neuromuscular junction (NMJ or motor end plate) [1].

Axons of motor neurons connected to the muscle fibers at a NMJ. This is a specialized synaptic formation where multiple axon terminals synapse with the sarcolemma of the muscle fibers. The neurotransmitter in this structure is the acetylcholine which secreted by the synaptic end bulbs of the motor neurons. Then, it binds to receptors on the sarcolemma to exert its function [40].

Anterior horn: The motor neurons in the anterior horn usually project to the muscles at the level of their exit of the spinal cord. AMNs are controlled by Renshaw cells. In the cervical and lumbar enlargements, the anterior horn is very large (hence their name), as both upper and lower limbs are innervated from this region. In thoracic levels, the anterior horn is relatively small, as it innervates only muscles of the axial Skelton. The AHC neurons are arranged in a somatotopic pattern, meaning that they correspond both anatomically and functionally with the organs they innervate. For example, neurons innervating the flexors are more posterior, and those innervating the extensors are more anterior. Distal groups of muscles have more lateral neurons in the anterior horn. On the other side, proximal and axial (trunk) muscle motor neurons are medial [44].

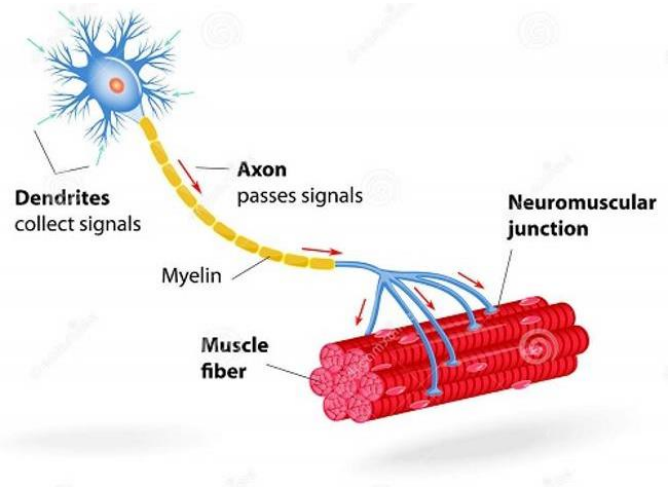

Figure (12): Motor neuron [53]

Laminas VIII and IX

Lamina VIII and IX are found in the spinal cord, and specifically in the ventral gray matter. Neurons receive descending motor supply from the cerebral cortex and the brainstem. Alpha and motor neurons are the abundant type of cells innervating the skeletal muscles (54). Somatotopic pattern is present where neurons supplying the extensors are ventral to those innervating the flexor muscles, and neurons supplying the axial muscles are medial to those supplying muscles of the distal extremities [55].

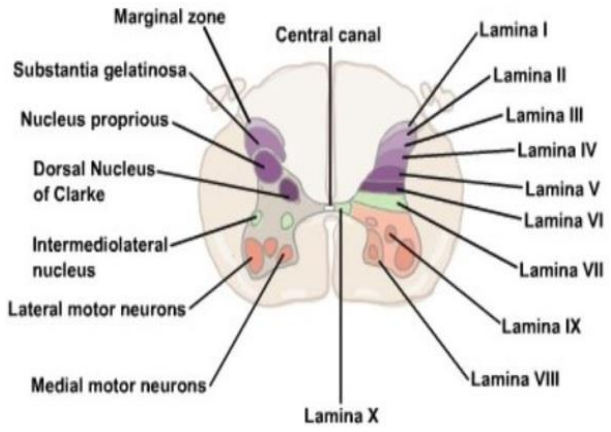

Figure (13): Laminas VIII and IX [56].

Lower Motor Neurons (LMNs) and the Somatic Reflex Arc

LMNs are vital in the somatic reflex arc (SRA). When muscle spindles sense a sudden stretch, they send a signal through the afferents. These fibers connect directly to the AMN in a monosynaptic reflex arc, or may connect to interneurons, which then linked to the AMN in a polysynaptic reflex arc. The LMN stimulates the effector muscle, enabling a quick response of the muscle. This reflex arc permits immediate interpretation and response to the stimulus via the spinal cord, skipping the brain with subsequent faster muscular response [57].

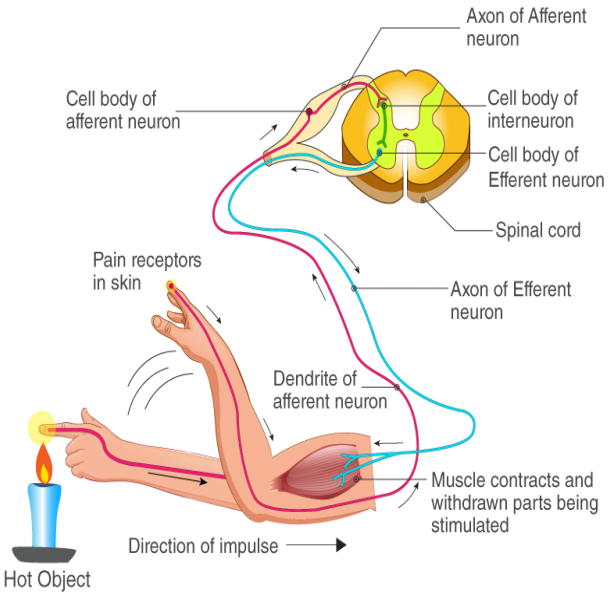

Figure (14): Reflex action [56].

Branchial Motor Neurons (BMNs)

Branchial motor neurons (BMNs) are concerned for nerve supply of the head and neck muscles that originate from the branchial arches. These neurons are located in the brainstem. Together with sensory neurons, they form the nuclei of cranial nerves V, VII, IX, X, and XI [5]

Visceral Motor Neurons (VMNs) and the Autonomic Nervous System (ANS)

Sympathetic fibers arising from centers for autonomic control in the hypothalamus. However, parasympathetic fibers arising from several brainstem nuclei. VMNs exerts a vital role in the sympathetic and parasympathetic functions of the ANS. In the sympathetic nervous system, central motor neurons are situated the segment of the spinal cord from T1 to L2. These neurons are found in the intermediolateral (IML) nucleus and have three distinct tracts: The first two tracts extend to the prevertebral and paravertebral ganglia. From these ganglia, peripheral neurons supply various anatomic structures, (e.g., heart, colon, intestines, kidneys and lungs). The third tract supplies the catecholamine-secreting chromaffin cells of the adrenal medulla [58].

Through these pathways (tracts), VMNs in the sympathetic division assist in the "fight-or-flight" response. On the extreme side, in the parasympathetic nervous system, VMNs share to the construction of cranial nerves III, VII, IX, and X. In addition to the brainstem, these neurons are also found in the spinal cord at the levels of S2 to S4. Similar to the sympathetic part, these motor neurons directly supply the ganglia in the heart, pancreas, lungs, and kidneys. In short, both divisions of the autonomic nervous system (ANS) include LMNs that differ from somatic motor neurons (SMNs), as they do not directly supply effector muscles but instead innervate ganglia [5].

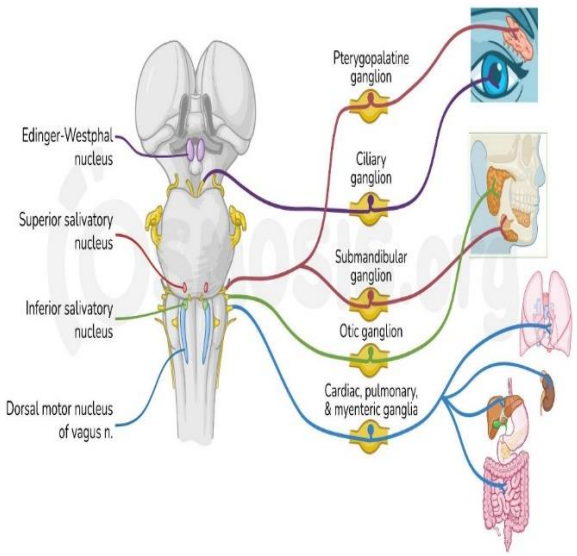

Figure (15): Parasympathetic cranial nerves nuclei and ganglion [58].

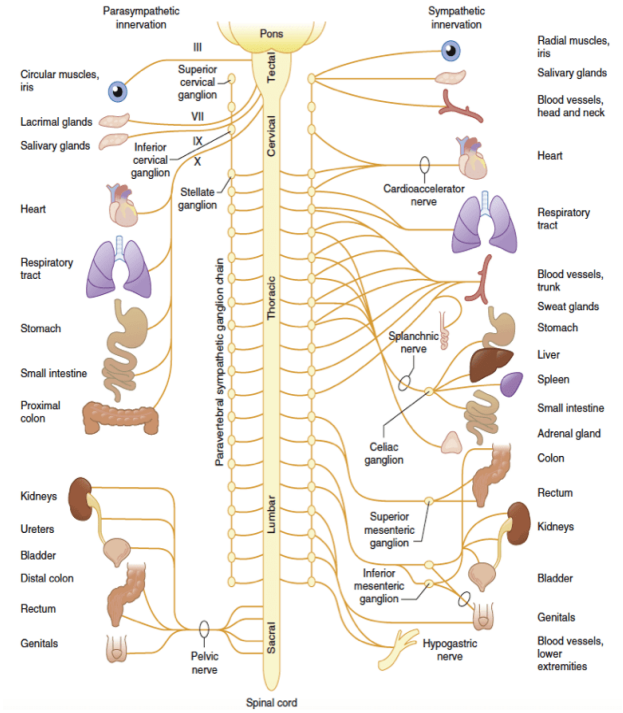

Figure (16): Sympathetic and parasympathetic innervations [58].

Upper and Lower Motor Neuron Syndromes

Lower motor neuron syndrome (LMNS) is the result of an injury to the anterior horn cells (AHCs) or the peripheral nerve. Diseases affecting the neuromuscular junction (NMJ) or the muscle itself can mimic LMN lesion, making it important to be considered in the differential diagnosis. Patients with LMN lesion, similar to those with an UMN lesion, will present with weakness. However, LMN specific manifestations include hyporeflexia, flaccid paralysis, muscle twitches and atrophy (muscle wasting) [59].

Motor Neuron Diseases

Motor neuron diseases are presented by different forms, amyotrophic lateral sclerosis (ALS) is the commonest. ALS is a unique condition as it shows signs of both UMN and LMN involvement. Patients in typical cases present with weakness, spastic paralysis & hyperreflexia in lower limbs, flaccid paralysis and hyporeflexia in upper limbs and muscle twitches affecting tongue and extremities. No sensory loss is associated with ALS. The disease progression is associated with serious complications. These complications include difficult speaking (Dysarthria), Difficult swallowing (Dysphagia), severe weakness and difficult breathing (dyspnea). Patients with ALS usually had an estimated median survival between 2 and 4 years. The commonest causes of death is the respiratory failure due to muscle weakness [60].

The clinical Significance

UMN and LMN lesions led to very different clinical manifestations. An UMN lesion is recognized as the lesion anywhere in the pathway from the cerebral cortex to the CST. This lesion manifested by hyperreflexia, spasticity, and a positive Babinski reflex (i.e., an upward response of the big toe when the plantar surface of the foot is stroked, with fanning out of the other toes). On the other hand, LMN lesions describes lesions anywhere on the pathway from the AHCs of the spinal cord, peripheral nerve, NMJ, or muscle. This lesion manifested clinically by hyporeflexia, flaccid paralysis, and muscle atrophy. Understanding the anatomy of the motor neurons is very important to localization of the lesion when faced with a patient with a weakness due to a motor neuron injury. Focusing mainly on the lateral CST, it is important keep in mind the decussation of this pathway at the level of pyramids. This decussation means that an UMN lesion above the medulla will cause clinical manifestations on the contralateral side of the body. However, a lesion to the lateral CTS after decussation will present on the ipsilateral side of the body [37].

Examples of lesions led to UMN symptoms are strokes, traumatic brain injury (TBI), spinal cord injury, ALS, primary lateral sclerosis (PLS), multiple sclerosis (MS), or anoxic brain injury. Positive and negative features of the UMN disease are present. For example, the positive features include hyperreflexia, spasticity, and a positive Babinski reflex. However, negative features include impairment of the motor control, easy fatigability, weakness and loss of dexterity [47].

Patient with lower motor neurolon lesion (LMN) lesion will manifested with weakness, hyporeflexia, flaccid paralysis, fasciculations, and atrophy [37]. Table (2) summarize the differences between Upper and lower motor neuron diseases [60].

Amyotrophic lateral sclerosis (ALS).

This disease is unique in that it is clinically presented with both upper and lower motor neuron manifestations. The patient typically had weakness, spastic paralysis and hyperreflexia in the lower limbs and flaccid paralysis and hyporeflexia in the upper limbs. The patient may also had fasciculations in the tongue and limbs. However, sensory loss is absent. ALS had a progressive course and ultimately, severe complications are developed (e.g., dysarthria, dysphagia, severe weakness, and dyspnea), and patient usually dead within 2-4 years due to respiratory failure **(60)**.

Table (2): Differences between Upper Motor Neuron (UMN) Lesion and Lower Motor Neuron (LMN) Lesion [60].

Feature	Upper motor neuron lesion	Lower motor neuron lesion
Paralysis	Groups of muscles of one or more limbs paralysed	Individual muscles paralysed
Muscle tone	Spasticity/rigidity (spastic paralysis	Flaccid (flaccid paralysis)
Deep tendon reflexes	Exaggerated	Absent
Superficial reflexes like abdominal and cremasteric reflexes	Absent	Absent
Plantar response	Extensor (Babinski sign positive	No response
Muscle atrophy	May not be marked (may occur late and will be due to disuse	Will be early and severe and due to denervation
Fasciculations and fibrillations	Do not occur	Are common

Spinal muscular atrophy (SMD)

One group of genetic disorders that led to LMN disease is spinal muscular atrophy (SMA), which clinically presented with different forms. However all of them are due to degeneration of the motor nuclei in the brainstem and AHCs of the spinal cord. One specific type of SMA is spinobulbar muscular atrophy. These diseases are usually expressed in adulthood (between 30 and 50 years). First presenting manifestations include tremor, lower limb weakness, and oro-lingual fasciculations. It is also a progressive disease with development of limb atrophy, bulbar, and facial muscles [61].

Poliomyelitis

Poliomyelitis is categorized as LMN disorder due to infection by the poliovirus. The virus led to destruction of the AHCs. As a consequence, affected individuals had weakness and other manifestation of LMN damage (e.g., flaccid paralysis essentially of the lower limbs). This paralysis is usually asymmetric. Unfortunately, this paralysis may extend up to include the respiratory muscles [61].

Cranial nerves

Most cranial nerves are supplied by UMNs bilaterally. However, cranial nerves VII and XII are exceptions, as they supplied only by unilateral input from the contralateral side of the brain. Specifically, damage to the CST and/or facial nerve led to a distinctive presentation depending on whether the damage in the upper vs. LMN. The forehead is dually supplied by CBT from each side of the brain, while the rest of the face below the forehead is supplied essentially by the LMN of CN VII. An UMN lesion of the facial nerve can arise anywhere in the CBT rostral to the facial motor nucleus on the pons. If an UMN lesion occurs, the forehead will be spared due to its dual supply. However, LMN lesion of CN VII led to flaccid paralysis of the entire ipsilateral side of the face [62].

Cortico-mesencephalic system

The fibers controlling eye movements arise from the FEF in the caudal part of the middle frontal gyrus and the adjacent inferior frontal gyrus (area 8). At the levels of the upper midbrain, fibers to cranial nerves III and IV leave the cerebral peduncles and descend through the tegmentum, usually in the medial lemniscus (corticomesencephalic tract). Fibers to cranial nerve VI also descend in the same pathway to the parabducens and parapontine reticular nuclei. Voluntary movements of the muscles associated with cranial nerves V, VII, and IX, X, XI, and XII are via the corticonuclear/bulbar pathway [63].

Corticonuclear/corticobulbar system—

This system distributes to the motor nuclei of the cranial nerves V, VII, IX, X, XI, and XII. It offers voluntary and involuntary control of the muscles and glands supplied by these nerves. Previously, this tract was called corticobulbar as the medulla and pons comprising these nuclei are collectively called the bulb. However, the term corticonuclear is preferred because it describes the key role of the cranial nerve nuclei in movement. These fibers are found anterior to the corticospinal fibers in the genu of the internal capsule and medial to the CTS in the cerebral peduncle, pons, and medullary pyramids. The fibers innervating the cranial nerves have the following origin of the cortex:

(a) Muscles of facial expression (motor nerve VII), mastication (motor nerve V), and deglutition (ambiguous nuclei of nerves IX and X) arise from pyramidal cells in the inferior part of the precentral gyrus, area 4.

(b) Muscles in the larynx are controlled from the inferior frontal gyrus and the frontal operculum (the posterior part of the pars triangularis, area 44). It seems that many corticonuclear axons end on interneurons and not directly on the motor neuron of the cranial nerve.

(c) The cortical innervation of the cranial nerves is bilateral, with the exception of the lower facial muscles, which are innervated by the contralateral cortex. Thus, unilateral lesions in the corticonuclear system led to weakness and not paralysis. Paralysis results only from bilateral involvement in the corticonuclear system [64].

Ocular movements are controlled by supra- and infra-nuclear pathways. The FEF area (area 8) with output to the paramedian pontine reticular formation and medial longitudinal fasciculus in the brainstem that controls the third, fourth and sixth nuclei pass through the individual cranial nerves and the NMJ to the orbital muscles. Four cortical areas which are connected to each other are responsible for generation of the saccadic (rapid) movement to the contralateral side. These areas are the FEF area (area 8),

supplementary eye field (SEF) area which is part of the supplementary motor area (SMA), the dorsolateral prefrontal area and the parietal posterior eye field area. Fibres from these areas descend to the pontine paramedian reticular formation (PPRF which predominantly responsible for activation of the sixth nerve nucleus) and the medial longitudinal fasciculus (MLF which mainly activates the third and fourth nerve nuclei). Lesions of the supranuclear tracts or brainstem led to disturbances of gaze (gaze palsies including bilateral eyes). Whereas infranuclear lesions of nuclei in brainstem or cranial nerves or NM junction or ocular muscle cause individual or multiple ocular muscle weakness [(64)].

Oculomotor Nerve

The oculomotor nerve (CN III) offers innervation to five of the seven extra-ocular muscles that control upper eye lid and eye movements. The functional components of this nerve are GSE, GSA and GVE. The GSE cell bodies arise from the oculomotor nucleus located in the midbrain [(49)].

Oculomotor Nerve Palsies

Damage to the oculomotor nerve affects the ipsilateral eye. There is a "down and out" appearance when a person looks straight ahead. The unopposed action of the lateral rectus muscle (LRM) forces the eye to deviate laterally (i.e., lateral strabismus) and because the superior oblique muscle (SOM) still functions to depress the eyeball, this forces the "down and out" appearance. Levator palpebrae superioris function is lost as well, leading to drop of the upper eyelid (ptosis). Any lesion of the somatic motor fibers would be referred to as external ophthalmoplegia and includes the loss of most extraocular muscle functions [(49)].

Trochlear Nerve

The trochlear nerve (CN IV) offers motor innervation to just the superior oblique muscle (SOM) of the eye. The SOM depresses, abducts and medially rotates the eye. GSE and GSA are the functional components of this nerve. The cell bodies of the motor fibers are in the trochlear nucleus which itself is located near the midline in the tegmentum of the caudal midbrain [(49)]. Paralysis of SOM results in vertically separated diplopia especially in inferior gaze. For example, reading and climbing downstairs, and there is usually head tilt away from the side of paralysis [(30)].

Trigeminal Nerve

Its smaller motor root carries SVE branchiomotor fibers that are responsible for supplying the muscles of mastication, tensor tympani, tensor veli palatini, mylohyoid and anterior belly of digastric muscles. It joins the mandibular nerve (CN V3) just outside the skull. The motor nucleus is found in the mid-pons region of the brainstem and anterolateral to the edge of the fourth ventricle [(49)].

Damage of the Trigeminal Nerve

The damage of trigeminal nerve injury (e.g. traumatic, infectious, aneurysmal, or cancerous) may lead to a loss of general sensation to the face or mucous membranes of the nasal and oral cavities, flaccid paralysis of the muscles of mastication, deviation of the jaw to the weak side, partial deafness to low-pitched sounds (hypacusis) due to paralysis of the tensor tympani muscle, and loss of the afferent limb of the corneal reflex [(49)].

Abducent Nerve

The abducent nerve (CN VI) is concerned with supplying only one of the extraocular muscles, the lateral rectus muscle (LRM). The LRM abducts the eye. The functional components of this nerve are GSE and GSA. The abducent nucleus contains motor nerve cell bodies that have axons course ventrally through the pontine tegmentum before exiting at the pontomedullary junction (~70%) and supply the ipsilateral lateral rectus or interneurons (~30%) that decussate and project via the medial longitudinal fasciculus (MLF) to the contralateral oculomotor motor nucleus. Then, neurons go on to supply the medial rectus. This means that the abducent motor nucleus negotiates horizontal eye movements and permits for the two muscles to move in unison [(1)].

Lesion of the Abducent Nerve or Nucleus

A lesion of the abducent nerve distal to the motor nucleus leads to paralysis of the lateral rectus and forces the eye to deviate towards the midline because of the unopposed action of the medial rectus. This type of paralysis leads in a medial strabismus and the individual has horizontal diplopia (double vision). Diplopia can be reduced if the subject turns their head towards the affected side when looking forward. If the lesion is located at the abducent motor nucleus, the deficiency is similar to damage including the individual nerve, but in addition there is the inability to offer excitatory input to the contralateral oculomotor nucleus and its neurons concerned with innervation of the medial rectus. This is called lateral gaze paralysis [(49)]. Clinical features of sixth nerve palsy if the injury in the brainstem may include the fifth, seventh and eighth cranial nerves, as well as the pyramidal tract or the cerebellum.

(a) Raymond's syndrome: sixth nerve palsy and contralateral hemiplegia.

(b) Millard-Gubler syndrome: sixth nerve palsy, ipsilateral seventh nerve palsy and contralateral hemiplegia.

(c) Foville's syndrome: sixth nerve palsy, horizontal conjugate palsy, ipsilateral fifth, seventh, eighth cranial nerve

palsy and ipsilateral Horner's syndrome [30].

Clinical Interpretation of Ocular Movement Abnormality

(a) Sixth nerve palsy (LR6)

• Horizontally separated diplopia that worsened on abduction of the eye.

• The eye abduction is restricted.

(b) Fourth nerve palsy (SO4)

• Vertically separated diplopia worsens on looking down.

• Reduction of the inferior movement in adducted position and impaired in torsion.

(c) Third nerve palsy

- Vertically separated diplopia, ptosis, mydriasis, eyeball placed down and out with difficult adduction, elevation and depression of eyeball.
- Pupillary fibres are superficially located in the third nerve and hence extrinsic compression, for example, by posterior communicating artery (PCOM), aneurysm leads to mydriasis by including the peripherally placed pupillary fibres whereas diabetic ischemic neuropathy which is non-compressive with inclusion of the central fibres of the third nerve due to ischemia will spare the pupil [30].

Facial Nerve

The facial nerve (CN VII) is concerned with the second pharyngeal arch of development and it comprises two initial roots, a facial nerve proper (or motor root) and an intermediate nerve (or sensory root). SVE: the cell bodies arise from the facial nucleus located in the caudal pons. These fibers loop around the ipsilateral abducent nucleus before ongoing through the motor root. Prior to leaving the stylomastoid foramen the SVE fibers supply the stapedius muscle of the middle ear. After exiting through the foramen, the SVE fibers are concerned with nerve supply of the muscles of facial expression, scalp and auricular region, the stylohyoid and posterior belly of digastric muscles [66].

The facial nucleus

It obtains corticonuclear fibres for volitional control. Neurons that supply muscles of the scalp and upper face have bilateral corticonuclear fibres, while those innervating lower facial musculature receive a predominately contralateral nerve supply. Clinically, upper and lower motor neuron lesions of the facial nerve can be differentiated because the former leads to paralysis confined to the contralateral lower face (supranuclear facial palsy), whilst the latter leads to a complete ipsilateral paralysis (Bell's palsy) [66].

Facial Nerve Damage

• Central (supranuclear) paralysis: includes a loss or lesion of the upper motor neurons. It clinically manifests by paralysis of the contralateral facial expression muscles in the lower half of the face while the contralateral forehead and eyelid region remain intact. The peripheral (infranuclear) paralysis includes a loss or lesion of the LMNs. It presents with a compete paralysis of the ipsilateral facial muscles of expression. This is representative of Bell's palsy [1].

The nucleus ambiguus

The nucleus ambiguus is best described as a group of large motor neurons located in the reticular formation of the medulla oblongata (MO) in the brainstem. It is a shared cranial nerve nucleus of the glossopharyngeal nerve (CN IX), and vagus nerve (CN X) the cranial root of accessory nerve (CN XI) is now also considered as (displaced) fibers of CN X originating from the caudal nucleus ambiguus to travel some distance with those of the (spinal root of) CN XI before joining the main CN X (67). The name 'ambiguus' arising from its difficult-to-find position and variation in different species. The nucleus ambiguus offers fibers that supply the somatic muscles of the pharynx, larynx, and soft palate. Additionally, the nucleus ambiguus has a role in parasympathetic cardiac inhibition through CN X. Unilateral injury of the nucleus ambiguus may lead to the development of dysphagia and hoarseness. These characteristics are classic in lateral medullary syndrome (Wallenberg syndrome). The nucleus obtains bilateral (but mostly contralateral) UMN afferents via corticonuclear fibers. This nucleus gives origin to the branchial efferent motor fibers of the CN X supplying the laryngeal and pharyngeal muscles, axons from the nucleus ambiguus passing in the CN IX supply stylopharyngeus muscle [68].

Glossopharyngeal Nerve

The glossopharyngeal nerve is the 9th cranial nerve (CN IX). It is one of the cranial nerves that has sensory, motor, and parasympathetic functions. It arises from the medulla oblongata and ends in the pharynx. It is associated with the third pharyngeal arch of development and has the functional components SVE, GSA, GVE, GVA, and SVA [69].Special visceral efferent fibers (branchial motor) are the main motor fibers of the glossopharyngeal nerve and provide motor supply to the stylopharyngeus muscle. This muscle is concerned with the elevation of the larynx and pharynx, especially during speaking and swallowing [70].

Trauma to the base of the skull may lead to a compromise of structures passing through the jugular foramina, which can lead to hemorrhage or air embolism due to injury of the jugular vein [71]. The injury of the three cranial nerves IX, X, or XI, leading to different issues. The damage of the cranial nerve IX may lead to impairment of the taste in the posterior one-third of the tongue, as well as dysphagia. Cranial nerve X damage can lead to a myriad of dysfunctions (for example, dysphagia, speaking issues, gastrointestinal and cardiac issues). Cranial nerve XI damage essentially leads to muscular pain in the shoulder, and neck, as well as weakness of the sternocleidomastoid and trapezius muscles [72].

Glossopharyngeal Neuralgia

Specific lesions including the glossopharyngeal nerve are rare. However, when occurs, the lesion is most likely affecting multiple cranial nerves such as the vagus and spinal accessory nerves all at once (jugular foramen syndrome) [49].

Glossopharyngeal nerve lesions result in the following:

Impaired swallowing: Impairment of the taste sensation over posterior one-third tongue. Impairment of the light touch and pain sensation over the pharynx, tonsillar region, soft palate and middle ear, posterior wall of external auditory canal and posterior part of tympanic membrane. Absent gag reflex—on ipsilateral stimulation which is preserved on contralateral stimulation [64].

Vagus Nerve

The vagus nerve (CN X) is linked to fourth and sixth pharyngeal arches of development. It has the functional components SVE, GSA, GVE, GVA, and SVA. The cell bodies of SVE arise from the nucleus ambiguus. Then pass through the superior laryngeal, external laryngeal, and recurrent laryngeal nerves to supply the laryngeal muscles. They pass through the pharyngeal branches of the pharyngeal plexus to supply the soft palate (except tensor veli palatini) and pharyngeal muscles (except stylopharyngeus) [49].

Lesion of the vagus nerve

Like the glossopharyngeal nerve, isolated vagus nerve lesions are rare. If the recurrent laryngeal nerve is the site of the lesion, this could lead to difficult speaking (dysphonia) or hoarseness because of the paralysis of the vocal cords. Lesions including the pharyngeal nerve branches may lead to difficult swallowing (dysphagia). If the soft palate or anterior pharynx is stimulated and a lesion of the vagus nerve is suspected, the uvula and palate will deviate away from the lesion [49]. Also Vagus nerve lesions will lead to absent gag reflex—on ipsilateral and contralateral stimulation, abnormal oesophageal motility, and gastric acid secretion, emptying of the gall bladder and abnormalities of heart rate and other autonomic dysfunctions [64].

Accessory nerve:

The cranial accessory is made from the nucleus ambiguous and join the vagus nerve. It offers motor control to the muscles of the soft palate, larynx and pharynx. The spinal accessory nerve (CN XI) offers motor supply to both the sternocleidomastoid and trapezius muscles. The functional components of this nerve are GSE. The cell bodies of the motor fibers are organized in the spinal accessory nucleus, from the posterolateral aspect of the ventral horns of cervical spinal cord levels C1-C5 (or C6) [68].

Lesions of the Spinal Accessory Nerve: On its pathway to innervate trapezius muscle, lesions of CN XI will lead to shoulder dropping due to weakness and atrophy of the trapezius muscle. Impaired function of the sternocleidomastoid muscle would lead to a deficiency of rotary movements of the neck towards the opposite side against a resistance [49].

Hypoglossal Nerve

This is the twelfth nerve that originates from hypoglossal nucleus in the medulla. CN XII is provides pure motor supply of tongue muscles. The hypoglossal nerve innervates all extrinsic and intrinsic tongue muscles except for palatoglossus (innervated by vagus nerve) [30].

The functional components of this nerve are GSE and GSA. The GSE motor fibers have their cell bodies in the hypoglossal nucleus. The nucleus appears as a column found anterior to the floor of the fourth ventricle besides the midline of the medulla. This forms a triangular elevation described as the hypoglossal trigone [49].

Lesions of the Hypoglossal Nerve: A hypoglossal motor nucleus is supplied by UMNs associated with CBT fibers that decussate and target the contralateral hypoglossal nucleus. The LMN then targets the ipsilateral tongue musculature except for the palatoglossus muscle. An UMNL found before the decussation leads the tongue to deviate away from the side of the lesion. A LMN lesion after the decussation leads the tongue to deviate to the same side of the lesion. When protruding the tongue, its apex diverges toward the paralyzed side due to the unopposed action of the genioglossus muscle on the normal side of the tongue. A lesion to the UMNs for CN XII will lead to spastic paralysis of the contralateral genioglossus. This will lead to the deviation of the tongue to the contralateral side [50].

Medial (basal) medullary syndrome

It usually includes the pyramid, part or all of the medial lemniscus, and nerve XII. If it is unilateral, it is also termed

"alternating hypoglossal hemiplegia". The term denotes the finding that the cranial nerve weakness is on the same side as the lesion, but the body paralysis is on the opposite side. Larger lesions can lead to bilateral defects. The included area is supplied by the anterior spinal artery or by medial branches of the vertebral artery [41].

Basal pontine syndromes

It can include both the CST and a cranial nerve (VI, VII, or V) in the affected region, depending on the extent and level of the lesion. The syndrome is termed alternating abducens (VI), facial (V), or trigeminal hemiplegia (V). If the injury is large, it may embrace the medial lemniscus. The vascular supply comes from the perforators, or pontine branches, of the anterior inferior cerebellar artery [41].

Extrapyramidal tracts

The extrapyramidal system (EPS) is described as an anatomical concept first introduced by Johann Prus in 1898 when he exposed that the disturbance in pyramidal pathways failed to prevent epileptic motor activity. Prus hypothesized that, "apart from pyramidal tracts, there must be alternative tracts, called the "extrapyramidal tracts," that "brought epileptic activity" from the cortex to the spinal cord [73].

The EPS provides an important function in maintaining posture and regulating involuntary motor actions. In particular, the EPS offers:

- Adjustment of the postural tone
- Preparation of predisposing tonic approaches for involuntary movements
- Provide actions to make voluntary movements more natural and correct
- Higher control of the automatic regulations of tone and movements
- Control of the reflexes that accompany reactions.
- Control of the originally voluntary movements but then become automatic through exercise and learning (e.g., in writing)
- Inhibition of involuntary movements (hyper-kinesias), which are chiefly obvious in extrapyramidal diseases [74].

To summarize, we could say that, the EPS controls the automatic activities but also affects voluntary movements through a tonic function. These control mechanisms include the treating of centers in multiple brain areas, such as parts of the cortex, cerebellum, thalamus, reticular substance, and several basal ganglia. The term basal ganglia denotes a group of subcortical nuclei. Among these nuclei, the caudate and putamen nuclei "together create the neostriatum", plus the substantia nigra (SN), red nucleus (RN), and the subthalamic nucleus of Luys constitute the nuclei of the EPS. Many subcortical tracts are starting from all these centers, stem out and end in the spinal cord. However, the majority of pathways travel through the basal ganglia. Anatomically, the EPS can be described as a set of nuclei and fiber pathways that receive projections from the cortex and send projections to brainstem and spinal cord and, functionally, provides a complex motor-modulation actions [74]. The extrapyramidal tracts include the reticulospinal, vestibulospinal, rubrospinal, and tectospinal tracts [75].

Reticulospinal Tract

The reticulospinal tract center on the complicated network of neurons and circuits called the reticular formation. Reticular formation provide various functions that include the regulation of consciousness, location of the centers responsible cardiovascular and respiratory systems, and organization of the eye movement [49]. This tract transmits motor signals from the reticular formation. The medial reticulospinal tract starts in the pontine reticular formation and descends to the ventromedial spinal cord via the ipsilateral anterior funiculus, which encloses alpha and gamma motor neurons of the extensor muscles. The ascending spinothalamic pathways also stimuate the medial reticulospinal tract. The lateral reticulospinal tract starts in the medullary reticular formation and descends to motor neurons in the spinal cord via the bilateral lateral funiculus [76]. Axons of the lateral and medial reticulospinal tracts end at the medial parts of the ventral horn gray matter. At this site, they have a primary actions on the local circuits that coordinate movement of both axial and proximal limbs but they are largely inhibitory to alpha, and gamma neurons in the spinal cord [49]. Reticulospinal tract regulates activities of muscle group that activate primitive motor performances such as the direction of the body away from to toward a stimulus and motor behaviors that do not need dexterity. It also integrates distal muscle with proximal muscle actions and starting changes in muscle tone related to voluntary limb movements. Reticulospinal tract also provide control over the process of breathing and which is one of its important functions of the motor nuclei in the reticular formation. Topically active neurons in the medulla descends to neurons in the spinal cord that will activate the skeletal muscles included in respiration. Voluntary control of these neurons included in respiration occurs through cortical projections [44].

Vestibulospinal Tract

The medial Vestibulospinal tract starts in the medial vestibular nuclei "known as Schwalbe's nucleus", in the medulla and ends in the motor neurons of the limb motor. They are concerned with provide innervation for muscles of the upper half of the body, especially that of the neck and

forelimbs. The lateral Vestibulospinal tract starts in the lateral vestibular nuclei "also known as Deiter's nucleus" of the pons, and ipsilateral descends down to the Rexed's laminae VII and VIII. These laminae include premotor interneurons and other motor neurons of the alpha and gamma types that are concerned with innervation the extensor muscles that oppose gravity as well as inhibiting the flexor muscles. The vestibulospinal tract provides a crucial role in maintaining an erect posture [(77)]. This tract connects the brain stem vestibular nuclei with the spinal cord. This permits modulation of the posture, movement, and balance on the basis of equilibrium information offered by the vestibular system [(40)]. The vestibular nuclei apply a strong excitatory actions upon the antigravity muscles through the medial and lateral vestibulospinal pathways. The antigravity muscles include the epaxial muscles of the vertebral column and the extensor muscles of the lower limbs [(41)].

Rubrospinal tract

The cortico-rubrospinal pathway is a motor tract that helps the regulation of flexor muscle movements of the upper and lower extremities. This system controls spinal cord LMNs indirectly [(15)]. It starts from the red nucleus of the midbrain tegmentum. It crosses the midline in ventral tegmental decussation of the caudal midbrain. The tract constitutes a contralateral tract in the dorsolateral part of the lateral funiculus and located in the ventrolateral part of the spinal cord. The rubrospinal tract is mainly concerned with transmission of signals into the red nucleus from the motor cortex and cerebellum to the spinal cord and ventral horn lamina V, VI, and VII [(78)].

In these laminae, the rubrospinal tract synapses with alpha and gamma neurons that stimulate the flexor muscles. This tract concerned with the maintenance of muscle tone and in the control of rudimentary motor skills that are refined by corticospinal control. The rubrospinal tract controls the movement of the hand and fingers in addition to flexor muscles [(79)]. The origin, localization, termination and functions of rubrospinal connections are poorly understood in humans, and the tract seems to be rudimentary [(41)].

Tectospinal Tract

The tectospinal tract originates from the superior colliculus in midbrain and receives stimuli from the retina and cortical visual association areas, then it directed ventromedial to the periaqueductal gray (PAG) matter, and ends in the contralateral anterior gray horn lamina VI, VII, and VIII of cervical and upper thoracic segments of the spinal cord. It provides an important function in the orientation of the head, neck, eyes, and upper extremities in response to sudden movement, loud noises, and bright lights [(80)]. It is crucial for postural movements driven by the superior colliculus [(40)]. Fibers of the tract descends only to the upper cervical cord segments, terminating in laminae VI–VIII. They make polysynaptic connections with motor neurones innervating muscles in the neck, facilitating innervation of the contralateral muscles and inhibiting innervation of ipsilateral ones [(41)].

Solitariospinal tract

The solitariospinal tract is a small group of fibers arises from neurons in the ventrolateral part of the medullary nucleus solitarius. Fibers then descend in the ventral funiculus and ventral part of the lateral funiculus of the cord, to end on the phrenic motor neurons supplying the diaphragm and thoracic motor neurones innervating intercostal muscles [(67)].

Olivospinal Tract

The tract and its existence have been questioned for some time. If present, it may function by controlling the reflex movements and facilitating muscle tone [(49)].

Medial longitudinal fasciculus

This originating from other nuclei of the vestibule "called as medial vestibulospinal tract". It projects to cervical part of spinal cord for reflex control of neck muscles to co-ordinate with the movement of the eye [(33)]. It is an important tract concerned with the control of gaze and head movements. The medial longitudinal fasciculus starts in the vestibular nuclei and carries vestibular influences downward. In the pons, the tract sends projections rostrally from the vestibular nuclei to the abducens, trochlear, and oculomotor nuclei and from the lateral gaze center in the pons to the oculomotor nuclei [(41)].

Decorticate rigidity

Damage to the cerebral cortex or the tracts descending from it lead to decorticate rigidity. This is manifested by flexor rigidity of upper limb and extensor rigidity in lower limb. The flexion of the upper limbs is attributed to excitatory action of rubrospinal on flexor muscles and by the lateral reticulospinal tract from medulla (both favor flexors). The extension of lower extremities is attributed to intact medial reticulospinal tract from pons and vestibulospinal tract. Widespread hemorrhage in internal capsule may lead to hemiplegia and unilateral decorticate rigidity [(33)].

Other motor control tracts

The Dentatothalamic Tract

The dentatothalamic tract starts in the dentate nucleus and ends by synapsing with cells in the contralateral ventrolateral nucleus of the thalamus. The axons of the thalamic neurons then ascend through the internal capsule to reach the primary motor area of the cortex. This pathway permits the dentate

nucleus to affect the motor activity on the same side of the body by acting on the motor neurones located in the opposite cortex [(81)].

Role of the Basal Ganglia

The basal ganglia affect the motor areas of the cerebral cortex. Thus, the basal ganglia have an important effect on the lateral corticospinal system of motor pathways. Such an influence is consistent with some of the movement disorders observed in diseases of the basal ganglia. The basal ganglia also control the medial motor pathways, because diseases of the basal ganglia can also affect the posture and tone of proximal muscles [(2)].

Corticopontine fibers

Corticopontine fibers arising from all areas of the cortex, i.e. frontal, parietal, temporal and occipital lobes. The largest arise from the frontal lobe. The purpose of the corticopontine fibers is a line of communication with the opposite cerebellum to permit for the coordination of motor functions, as it ends in the deeper pontine nuclei. The corticopontine fibers project initially from the cortex (frontal lobe) to end in the pontine nuclei. Then, fibers project through the middle cerebellar peduncle to the contralateral cerebellum via the pontocerebellar fibers. This tract from cortex to pons to cerebellum is important for the cerebellar function and integrity [(82)].

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