

MODULE 2. The Role of the Cerebellum and the Basal Ganglia

Unit 2.1 Cerebellum

2.1.1 Structure of the Cerebellum

The **cerebellum** is the largest organ in the encephalon, and is located behind the protrusion and the medulla oblongata, in the posterior cranial fossa.

The cerebellum is divided into two hemispheres, separated by the **cerebellar vermis** (this is an important structure, located in the middle area of the posterior and anterior lobe). The vermis is the end of the road for many somatic signals from various parts of the body. It also plays a very important role in posture management in subconscious processes.

The cerebellum can also be divided into three lobes, namely: **the flocculonodular lobe, anterior lobe and posterior lobe**, also known as the middle lobe.

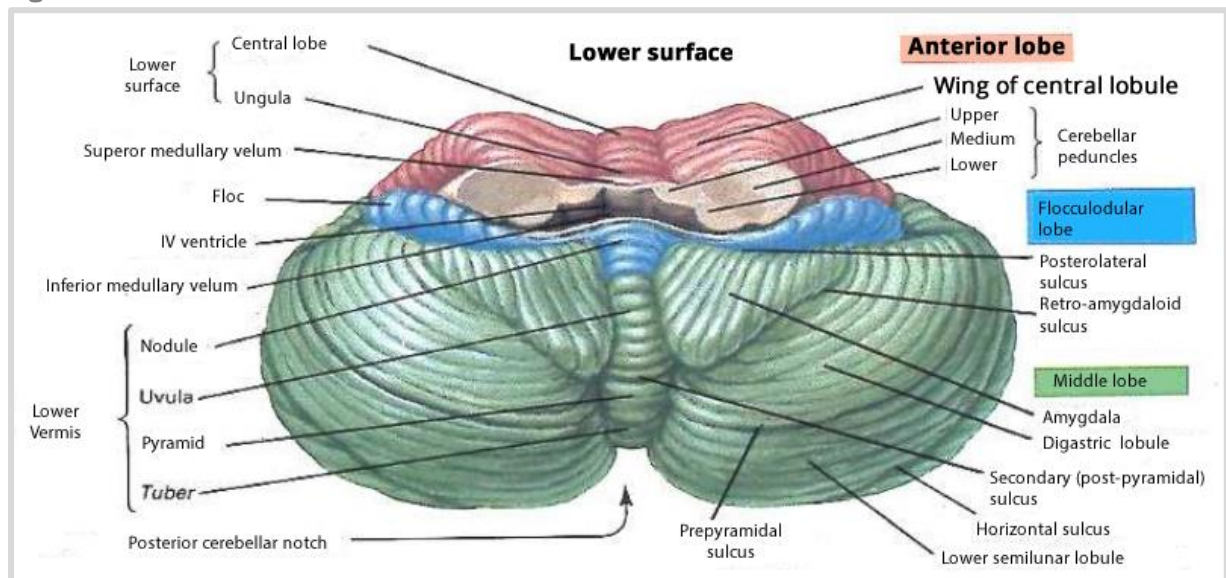
- **Posterior lobe:** This is the largest part of the cerebellum, and is located between the primary and uvula nodular fissures. It is made up of two cerebellar hemispheres. It is also closely related to the cerebral cortex and is involved in the planning and initiation of movements. This lobe "ensures the automatic organization of voluntary motor functions" (Rigal, 1987. p.77).
- **Flocculonodular lobe:** This is located behind the uvula nodular fissure and is part of the archicerebellum, which is closely related to the vestibular system, making it intimately tied to equilibrium. "It contributes to equilibrium through its relationship with the vestibular nuclei which administer activity and axial and proximal muscles" (Rigal, 1987. p. 77).
- **Anterior lobe:** "This can be seen over the top surface of the cerebellum and is separated from the posterior lobe by a v-shaped fissure known as the primary fissure" (Snell, 1999. p. 218). It has reciprocal connections with the spinal cord and is involved with muscle tone. According to Rigal (1987), this lobe controls the postural tone of limbs and during locomotion.



Its structure is made up of white matter on the inside, although small clusters of gray matter, referred to as **intracerebellar nuclei**, can be found, and grey matter on the edge, referred to as the **cerebellar cortex**, which is made up of three layers:

- 1) Molecular layer: contains two types of neurons, namely: **external stellate cells** and **internal basket cells**.
- 2) Purkinje cells: Large Golgi type I cells which are contained in a single layer. The dendrites in these cells head towards the molecular layer where they branch off extensively. An axon extends from the base of these cells, heading towards the deepest part of the cerebellum, bypassing the granular layer and finally entering the white matter. There, it acquires a layer of myelin to continue its path until synapsing in other nervous cells of the intracerebellar nuclei (clusters of grey matter, housed in white matter). There are four nuclei: **dentate nucleus, emboliform nucleus, globose nucleus** and **fastigial (or roof) nucleus**.
- 3) Granular layer: made up of small cells whose axons head towards the molecular layer and bifurcate into a "T" shape (parallel fibers) (Snell, 1999).

Figure 1: The cerebellum



Source: [Untitled image on the cerebellum]- (n.d.). Retrieved from <https://goo.gl/RZPw1E>

According to Snell (1999), the anatomic location of the various cerebellar nuclei is:

- **Dentate nucleus:** this is the largest of the cerebellar nuclei. It resembles a wrinkled bag with an orifice towards the inside.
- **Emboliform nucleus:** this is located inside the dentate nucleus and is an oval shape.
- **Globose nucleus:** this is located within the emboliform nucleus and is made up of a group of roundish cells.
- **Fastigial nucleus:** this is located near the middle line of the vermis, near the roof of the fourth ventricle.

Large amounts of white matter are found in the cerebellar hemispheres, and small amounts in the vermis. Said matter is made up of **intrinsic fibers, afferent fibers and efferent fibers**. **Intrinsic fibers** never leave the cerebellum. These are responsible for connecting the cerebellar cortex with the vermis and, on other occasions, connecting both cerebellar hemispheres. **Afferent fibers** make up most of the white matter and head towards the cerebral cortex, entering the cerebellum through the middle and inferior cerebellar peduncles. **Efferent fibers** are the exit paths from the cerebellum. The route begins through the axons of the Purkinje cells which enter the intracerebellar nuclei to synapse, while another group of axons leaves the cerebellum without synapsing in the intracerebellar nuclei.

"The fibers of the dentate, emboliform and globose nuclei leave the cerebellum through the **superior cerebellar peduncle**. The fibers of the fastigial nucleus do so through the **inferior cerebellar peduncle**" (Snell, 1999. p. 223). The superior peduncles connect the cerebellum to the mesencephalon, the middle peduncles connect it to the protrusion and the inferior peduncles connect it to the medulla oblongata.

2.1.2 The Role of the Cerebellum in Motor Refinement

The **cerebellum** is a CNS organ that constantly receives information from various parts of the body. The motor program begins its journey from area 4 up to the medulla, the re-afferent information (feedback) climbs through the spinal cord, diverts through the inferior olive, enters the cerebellum, and in its cortex occurs the act of comparing what we meant to do (efferent value) with what we are actually doing (afferent value). The role of the cerebellum, then, is to compare the two values and detect the differences; this is why it can intervene in order to ensure that what is effectively happening matches what is really happening. In other words, it coordinates and attempts to make what is happening match what we had previously planned as closely as possible.

The cerebellum has different pathways: it may directly intervene in the corticospinal tract so that the actions are corrected, it can intervene in area 6 to adjust the motor program and, if necessary, it can change the motor program. Changing the pre-established program in the superior areas of the cortex takes more time than adjusting it and, were it necessary to make a change, we would have a delay, which, in a way, justifies the movements of hesitation or deterrence. The cerebellum can work on two levels: **adjusting or changing the program**. The cerebellum is indispensable for coordination.

Through its multiple paths of afference and efference, the cerebellum contributes to the regulation and control of the motor act, though most of its actions are not conscious. The **cerebellar hemispheres** anticipate movement and "prepare" the muscles that will be involved, while the **intermediate zones** will influence the execution of the movement, establishing the parameters for the levels of strength, direction, speed and deceleration needed to accomplish it.



The cerebellum is widely related to the gamma motor neurons, which pre-activate the neuromuscular junctions and predispose the musculature for action. Once movement is initiated, this organ is in charge of comparing the action being executed to an "ideal" model of the action, and if it detects an error, it may adjust or change the motor program. It is possible that the cerebellum may anticipate erroneous actions, but it will always be easier to anticipate actions that have already taken place than errors produced during new actions.

The cerebellum's role of comparing between what is planned in superior areas and what is executed in practice is possible because it receives a copy of the central motor or spinal efferences and the proprioceptive reafferences, simultaneously reaching the alpha motor neurons, which regulate muscle power, and the gamma motor neurons, which adjust the muscle receptors for position and speed, and play a vital role in controlling the development of slow or fast motor actions. In the case of the fast, well-learned movements, the intermediate cerebellum will ensure anticipatory error control and will suppress these before they happen (Rigal, 1987).

Following Guyton's (2006) thoughts, the motor cortex sends many more stimuli than necessary for performing a specific movement, which is why one of the cerebellum's actions is the following: once it receives the afference of the activation model that will be performed, it sends inhibiting signals to the motor cortex so that the action is as close as possible to that programmed. The cerebellum analyzes the intensity of the force and speed of the muscle action to reach a specific objective automatically, and instantly sends inhibiting signals to the agonist muscles and excitatory signals to the antagonist muscles.

The cerebellum has several functions that make it an indispensable organ for efficient motor control:

- The first of these functions to highlight is its **buffering capacity**: many of the body's movements are performed in a pendular way. Like any pendulum, the movement begins with a specific strength that is capable of overcoming the initial purpose and, as the pendulum swings, one force or another will stop the initial movement. In the case of the cerebellum, it is in charge of inhibiting certain muscles and activating others, in order to stop the initial movement at the desired moment.
- Another important function is its **capacity to predict**: as the cerebral cortex has to deal with more complex processes, the cerebellum is in charge of receiving the reafferences coming from different receptors that allow us to understand the position of our body or limbs in a specific space. Through these proprioceptive signals, the probable evolution of a movement in a specific time frame can be calculated. While this is not a purely motor function, it will influence most motor actions (very important in sports) and is involved with the cerebellum's ability to detect, through changes in the visual field, the speed at which an object is moving (Di Santo, 2015).



2.1.3 Correcting and Changing the Motor Program

Programming involves integrating two neuromotor dimensions:

- One is **anatomical** (which we call the footprint or engram), which is invariant; this is what you retain from movements.
- While the other is **parameterization** or, in other words, the possibility of unlimited variability, meaning that the protagonists of the movement may have absolute versatility. Versatility is the consequence of the invariability that is no longer an action, but something that is in the memory.

These two dimensions constitute **the act of programming**.

- 1) **Programming** is choosing who the soldiers are, the appropriate protagonists for performing the engram according to the demands of the situation. Obviously, the selection of protagonists depends on which will be more efficient in solving the problem of adaptation to its environment.
- 2) **Parameterization** is a decisional action which allows the brain to choose the most suitable protagonists.

However, putting the program together does not mean it will be carried out. So, where does neuromotor programming take place?

Ultimately, in two areas:

- The most activity, when you put the movement together, plan it and have yet to execute it, is in **area 6 (premotor cortex)**. This is where motor engrams are stored.
- The role of other very important areas is also being discovered, such as the **supplementary motor area** and the **pre-supplementary motor area**, which are responsible for activating the program.

Based on this, we can say that the area which puts the program together is not the same as the area which decides to execute it. Of course, experience in neuromotor programming will make this go quicker. As more experience is gained in the movement, the more complex programming action will request help from the SMA.

The IMC, the pre-motor area and the supplementary motor area is a possible but not obligatory sequence. Why not obligatory? Because a program can definitely trigger an action without the involvement of intention; in other words, it depends on the immediate response of a stimulus without you initiating it, independently of any outside stimulus.

If there is an immediate answer to a stimulus, we don't have any mediation from the SMA or the pre-SMA, but we always have the mediation of the premotor cortex; however, if you stand in front of a goalkeeper to take a penalty shot, you will definitely have the mediation of the supplementary motor area without an answer to a stimulus, such as the referee blowing his whistle.

What are the parameters? To understand this, we must understand that muscle is not part of the motor footprint. It would be a tremendous disadvantage if it were. Observe



everything that a subsystem parameterizes and directs: try to time all possible speeds in milliseconds for every one of the millions of times a soccer ball has been kicked. Imagine that all the possible speeds and their protagonists remain in the memory. How long would it take your brain to find that in order to kick a ball in one second? That's why it would not be an advantage for everything to belong to the engram.

Another component to consider is the recruitment of motor units, in other words, how many muscle fiber I'll recruit. For example, if every time I throw a basketball I have to activate a specific number of bicep fibers, it will become impossible to find the right parameter for each situation.

Unstable training works along these principles. The micromovements produced by the instability of the device will adjust the program parameterization. There may come a time in which you decide to change the program because you're about to fall, and in order not to fall you must perform another action, which would involve changing. While this lasts, there is a permanent recalibration of the program parameterization on the base of the proprioceptive information you're receiving; a permanent calibration and modulation of the optimal protagonists for the deployment of the engram. If you continue with the same protagonists as you're falling, and you don't use others, you're going to end up falling down. The cerebellum may act towards the objective of not falling and thus activate new programs. The cerebellum can correct the engram's parameters, which would take around 80 milliseconds, while changing it altogether may take between 400-600 milliseconds. So, is it always easier to try to adjust the same engram? What is the best choice? This shows the mistake that so many people make: repeatedly training their worst versions.

Before deciding to execute, the brain already knows what you're going to do, as it's the first area to receive the information. The basal ganglia also receives a copy of the program, and these activate the gamma motor system to generate a support tone for the action that is going to be performed. The muscle tone is programmed by the basal ganglia (Di Santo, 2015).

The rationale behind the "fake-out" movement in sport is based on the correction and change times of the motor program. When we fake out, we are forcefully changing the motor program, which is a lot slower than its correction. Changing it takes over 400 milliseconds, while correcting it would only take about 80 milliseconds or less. We must therefore train the speed of correction and change of the motor programs (Di Santo, 2015).

2.1.4 Alterations of Movement due to Cerebellum Injuries

In accordance with the functions of the cerebellum, the destruction of small parts would not significantly impact the subject's life, as long as they perform slow movements. When faced with a cerebellar cortex injury, other nervous structures could also perform some of its functions. However, cerebellar disorders typically alter the levels of strength, distance and speed of muscle actions, which may distort the voluntary motor act, both in



tonic and phasic activity. There are several conditions produced by a dysfunction in the cerebellar nuclei, such as the dentate nucleus, roof or interpositum, such as:

- **Dysmetria:** Also known as ataxia. As mentioned, one of the main functions of the cerebellum is to determine how far a movement should go. If the functions of the spinocerebellar bundles are affected by a specific injury, the feedback from various parts of the body will be distorted, meaning that the information returned will not be enough to generate precise and controlled movements.
One of the types of dysmetria is called hypermetria. This is a condition in which the afflicted person is unable to control their movements and, generally, goes past the intended point. This happens when the motor cortex sends the signals to perform a specific action, for example, reaching for an object with a hand, and the agonist muscles are activated but the cerebellum is unable to generate the brake or inhibitor for the movement, resulting in the subject lunging at the object they were attempting to reach (Guyton, 2006).
- **Dysarthria:** among its functions, the cerebellum is in charge of controlling the succession of movements, and this includes speech, which involves facial muscles, the larynx and tongue structures. Sufferers from this condition have difficulties speaking; they cannot control the intensity of the volume or duration of their speech. A person with dysarthria may be unintelligible when communicating, since there will be volume fluctuations in their speech and some sentences may be very fast while others are slow.
- **Adiadochokinesia** is a lack of progression in movements. If the cerebellum helps us predict the position of our body segments, a dysfunction may result in us not registering certain segments at certain times. This may cause movements to start too slowly or too late, stripping them of fluidity and progressivity.
- **Intention tremors:** Without the help of the cerebellum, muscles activate with no buffering (which is one of the cerebellum's functions). This condition occurs when there are injuries in the cerebellar peduncle or the dentate nucleus, which play an important part in the feedback with the motor cortex.
- **Cerebellar nystagmus:** much like intention tremors, in this condition there is a lack of buffering in the movement of the eyeballs. This condition is caused by injuries in the flocculonodular lobe and is noticeable when trying to focus the gaze on an object in our peripheral field of vision. When unfocusing, there are quick and oscillating eye movements.
- **Rebound:** this condition means that the cerebellum is unable to produce the activation of the antagonist muscles of a specific action. This is because, when faced with a cerebellar injury, the brain cannot efficiently communicate with the spinal cord to unlock the myotatic reflex that stops or interrupts a movement that occurs unexpectedly.
- **Hypotonia:** Injuries in the dentate nuclei provoke a decrease in the peripheral muscle tone next to where the cerebellar dysfunction is located (Guyton, 2006).

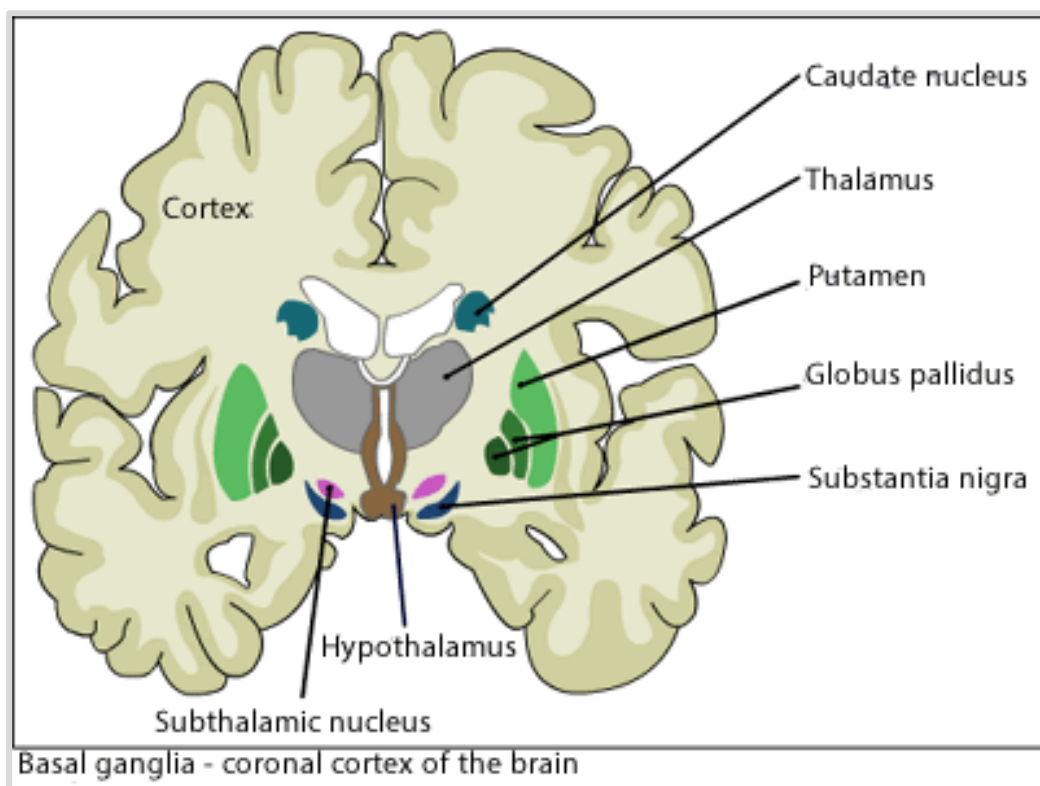


Unit 2.2 Basal Ganglia

2.2.1 Structure of the Basal Ganglia

The **Basal Ganglia** are clusters of neuronal structures located in the base of the brain (Rigal, 1987). These ganglia are comprised of the **dorsal striatum** (caudate nucleus and putamen), the **ventral striatum** (nucleus accumbens), the **globus pallidus**, the **subthalamic nucleus** and the **substantia nigra**. The red nucleus and the reticular formation are closely related to the ganglia. These are connected to various organs in the nervous system, and their function is centered around motor function regulation.

Figure 2: Basal Ganglia



Source: Tezanos, n.d., <https://goo.gl/WXU5kn>

- **Caudate nucleus:** This is indirectly involved in the modulation of movement. It signals the frontal lobe when something is not going well and something must be done about it.
- **Putamen:** This is in charge of all voluntary movements of precision. It also has an important role in operating conditioning.
- **Striate body:** This regulates instinctive behavior, muscle tone, character and sexual conduct. Inhibits the activity of the cerebral cortex and receives impulses from the thalamus.
- **Globus pallidus:** This transmits information from the putamen and the caudate nucleus to the thalamus.
- **Subthalamic nucleus:** This receives the afferences from the caudate nucleus and the putamen, participates in the motor control regulation and is associated with involuntary movement.
- **Substantia nigra:** This is a micro-regulator of the striate body through its neurotransmitter (dopamine) (Asociación Educar, 2015).
While basal ganglia are closely related to motor functions, they are not directly connected to the motor neurons of the spinal cord. Instead they receive the afferent information from the cerebral cortex and send information back to the cortex through afferent pathways. Prior to this, these efferences relay through the thalamus.

2.2.2 Connection between the Basal Ganglia and other Nervous Structures

The basal ganglia contain a huge number of nervous connections to the motor area of the cerebral cortex; in particular, many of these connections are between the primary motor cortex association area and the striate body.

The **striatum** is the functional receptive structure of the gangliar system. It receives afferences from several structures which are internal and external to the ganglia.

With regards to the structures it connects to, we can say that:

- It receives afferences from the cerebral cortex (frontal cortex, parietal, occipital and temporal) as well as the substantia nigra.
- The ventral part of the striatum receives information from the cingulate cortex, which is related to motivation.
- The thalamus sends afferences to the striatum "from the interlaminary nuclei to the caudate nucleus and from the central medial nucleus to the putamen" (Rigal, 1987). A large number of nerve pathways pass from the pre-motor area of the primary motor cortex to the striatum. In turn, the striatum sends afferences to the globus pallidus, subthalamus and substantia nigra, from which groups of nerve fibers go towards the thalamus and the cerebral cortex (motor areas). In this way, nerve connections are established between cortical motor areas and basal ganglia, by which a feedback circuit is formed, helping motor control (Guyton, 2006).

There are also nerve connections between the motor cortex centers with protrusion and cerebellum. When the signals return from the cerebellum, they pass through the

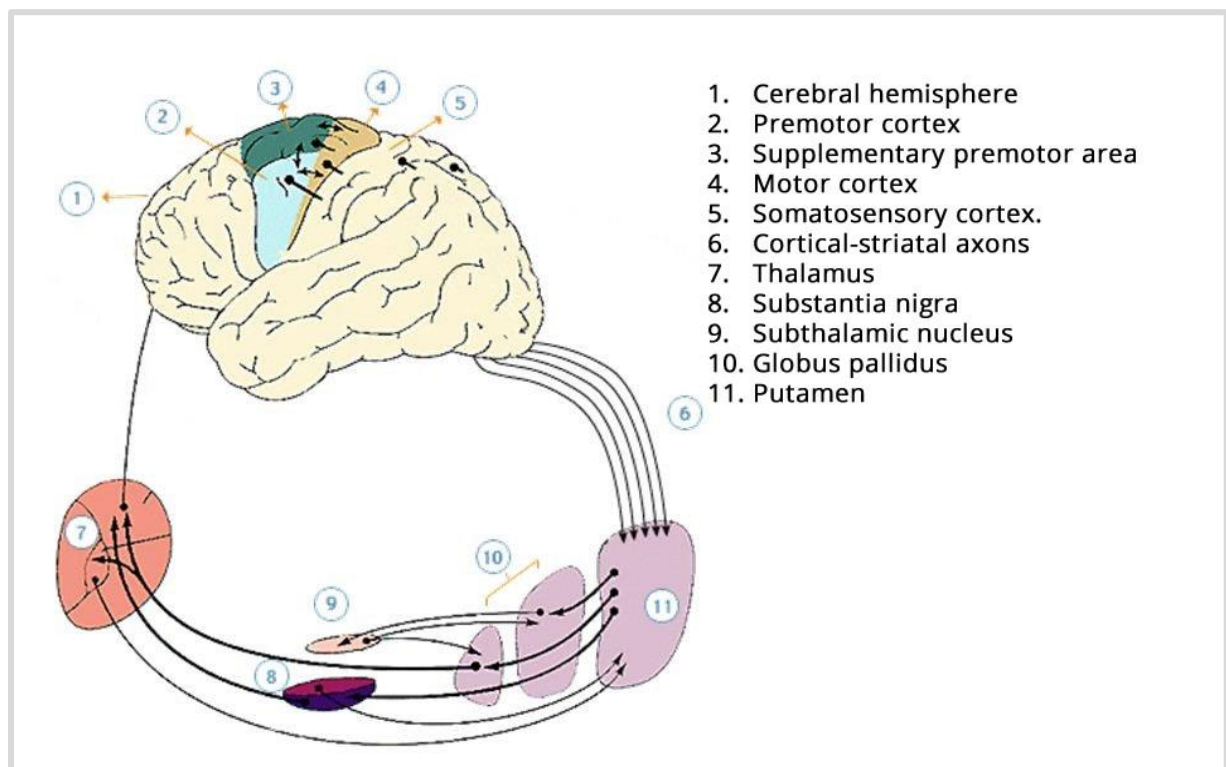


thalamus, which is related to the ganglia. This allows for connections between gangliar feedback and cerebral feedback.

The basal ganglia are also connected to each other. In this circuit, it is clear that nerve signals are sent down from the striatum, headed towards the substantia nigra (through the globus pallidus). Immediately afterwards, the signal returns to the striatum. In this circuit, the descending pathway secretes the neurotransmitter GABA, and the ascending pathway secretes dopamine.

The globus pallidus is connected to the encephalic stem. It sends numerous nerve fibers towards the red nucleus and the inferior olivary nucleus. Although the cerebral cortex has a direct connection with these brain stem nuclei, these connections converge in the same place as the basal ganglia.

Figure 3: Feedback circuits from the Basal Ganglia and the cerebral cortex



Source: Kandel, (1997)

2.2.3 Assemblage of Movements in the Charge of the Basal Ganglia (Automatism)

The striatum is the entry point for information originating from the cerebral cortex towards the basal ganglia. The afferences will arrive in different sectors of the striatum, depending on the cortical sector from which they originated. For example: the motor cortex sends afferences to the putamen for it to act on movement regulation;

meanwhile, the caudate receives information on cognitive processes and ocular movement.

The signals coming from the motor cortex towards the striate body belong to the cortico striatal pathway and originate from the motor cortex, premotor cortex and supplementary motor area.

Once the information enters the basal ganglia, it is directed to the thalamus through the substantia nigra and the inner face of the globus pallidus, through two different pathways. These two pathways are referred to as the **direct pathway** and the **indirect pathway**.

- **Direct pathway:** the caudate nucleus and putamen (striated nucleus) have inhibiting functions over the internal globus pallidus and the substantia nigra. In turn, the substantia nigra and the internal globus pallidus connect with the thalamic nuclei, and also have inhibiting functions. We can therefore see that when striatum activity increases, the activity of the thalamus nuclei increases as well, since the striatum would be inhibiting the inhibition caused by the substantia nigra and the internal globus pallidus on the thalamus.

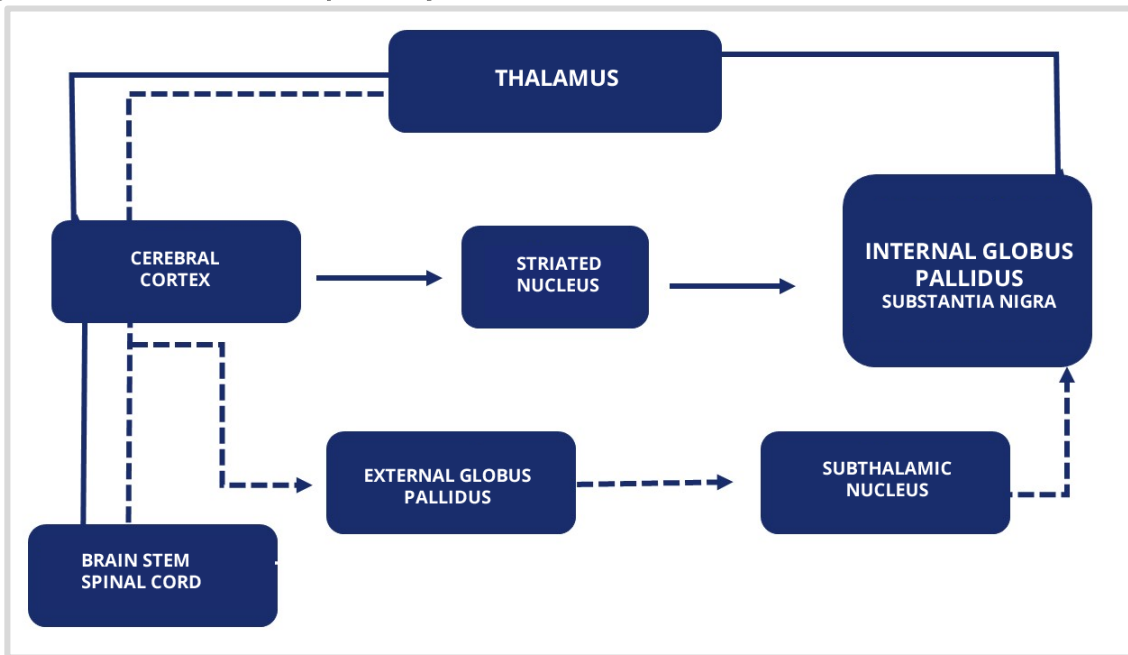
When the inhibition of the thalamus nuclei is inhibited, this stimulates the supplementary motor area (SMA), which sends the order to generate a movement to the primary motor cortex (PMC), which will synapse with the motor neurons of the spinal cord.

- **Indirect pathway:** some of the neurons of the striate body have a connection to the external globus pallidus (with inhibiting functions) which in turn communicates with the subthalamic nucleus and inhibits it. The subthalamic nucleus connects and activates the internal globus pallidus and the substantia nigra. Through the activation of the indirect pathway, the striatum inhibits the external globus pallidus, which uninhibits the subthalamic nucleus and causes the activation of the internal globus pallidus and the substantia nigra (thus inhibiting the thalamic nuclei and, as a consequence, the motor cortex) (Di Santo, 2015).

The objective of the direct pathway is to activate voluntary movements, while the indirect pathway seeks to inhibit the start of principal involuntary movement activities (parasitic movement). The normal development of movement and posture in general will depend on the existing relationship between these pathways. A nucleus that does not act optimally may influence the quality of movements and, in this way, generate different alterations.



Figure 4: Direct and indirect pathways



Source: Prepared by the author.

2.2.4 Alterations of Movement due to Pathologies (Parkinson's)

As mentioned, the basal ganglia have several functions, among them: controlling involuntary or subconscious (stereotyped) body movements. An injury in this region would therefore cause the subject to employ the superior cortical centers to supplement their lack of use.

Injuries in the basal ganglia area may occur due to carbon monoxide poisoning, drug overdose, infections, head trauma, lead poisoning, manganese or other heavy metals, tumors and strokes, among others (Guyton, 2006).

Through the study of cadavers of people who, in their life, displayed basal ganglia injuries, different conditions have been identified, such as: **Chorea, Hemiballism, Athetosis, Parkinson's.**

- The word chorea means "dance" in Greek and is used in this pathology because it is characterized by constant, random movement with no motor control, which may affect one or several body segments.

This condition occurs after injury to the caudate nucleus, the putamen, and produces a significant decrease in the secretion of the neurotransmitter GABA; thus the inhibiting GABA signals (directed from the putamen and caudate nucleus) cannot keep the globus pallidus and substantia nigra inhibited.

The spasmodic movements decrease with sleep and increase when the person with this condition feels stress or emotions, or when the body receives large amounts of sensitive information.

- Hemiballism means "half ballistic", and is referring to the ample, powerful movements that occur in a single hemibody. This causes violent movements in large areas of the body, which may cause a person to fall down if they are standing. It only affects one hemibody and is caused by an injury to the subthalamus of the contralateral hemibody.
- Athetosis is produced by an injury in the globus pallidus (outer face), which influences the feedback circuits between the ganglia, thalamus and cortex.

Under normal conditions, these circuits allow the execution of fine movements in a fluid way, since they connect and interrelate agonist and antagonist groups; however, when these circuits are interrupted, this generates abnormal movements due to the deviation of the path of the "normal" nervous impulse.

- Parkinson's disease is a condition caused by the death of substantia nigra neurons, in charge of producing dopamine, which is directed towards the putamen and the caudate nucleus, and whose main function is the proper control of movements. When the dopamine levels decrease, the information in the basal ganglia circuit is altered and this results in tremors, stiffness, slow movements and postural instability, among other symptoms.

The main symptoms of this condition are:

- Stiffness in the musculature of large areas of the body or isolated areas.
- Tremors in hands, legs, arms, jaw and face.
- Difficulty initiating movements, also known as akinesia (Guyton, 2006. Rigal, 1987).



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