

## PHYSAOLOGY

SHEET NO. 16
WRITER: 18+19
CORRECTOR: Ahmad AL dabbagh
DOCTOR: Faisal Mohammad



Here we have the basal ganglia which is important in correction in the resting state, it inhibit the muscle tone and the damage to it will cause rigidity (clasp knife) (8:10-9:20)

From the cerebral cortex we will have afferent fibers going to the basal ganglia and the correction then will be made and received by the thalamus and from the thalamus to the cerebral cortex by efferent fibers

## Basal Ganglia and Motor Control

Basal ganglia are subcortical gray matter nuclei found deep in the brain. The correct name is **Basal nuclei** (not ganglia) because they are a collection of cell bodies and dendrites (gray matter) inside the CNS. But they got this name when they were discovered. It is important to know that basal ganglia do not send descending/ ascending fibers, they only have fibers in the brain (upper fibers).

Structure of Basal ganglia system:

consist of four nuclei:

- 1-Striatum: Caudate and Putamen
- 2-<mark>Globus Pallidus</mark>

3-Substantia Nigra

work as one unit but they are functionally different

## 4-<mark>Subthalamus</mark>

 - 3 + 4 are related functionally not structurally to the basal ganglia

- Lentiform (lenticular) nucleus: Putamen+ Globus Pallidus

- **Corpus striatum**: Striatum (Caudate and Putamen) + Globus Pallidus

-Caudate nucleus has a head, body and tail.

Globus Pallidus is divided into two part:
 Globus Pallidus Internus (GPi) and Globus
 Pallidus Externus (GPe)

- internal capsule: white matter structure between Putamen and Globus Pallidus in one side and Thalamus and Caudate in the other side. It consists of three parts and is V-shaped when cut horizontally, in a transverse plane: 1- the bend in the V is called the **Genu** (means elbow) and it contains the corticobulbar fibers going form the cortex to the nuclei of the cranial nerves 2- anterior limb (sensory fibers) 3- posterior limb (motor fibers)



Basal ganglia Afferents and Efferents:

The basal ganglia are the principle subcortical components of a family of parallel circuits linking the thalamus with the cerebral cortex.

To understand this topic easily, let us start by studying the two main circuits present in the basal ganglia system. Any circuit should start and end in the same area and this applies on these circuits also; they start and end in the cerebral cortex.

CC Caudate Globus Pallidus (internal/external)	Thalamus <u>VA and</u> <u>VL nuclei</u> CC
Caudate Circuit	Putamen Circuit
extends into all lobes of the cortex and receives	Receives mostly from premotor and
a large input from the association area of the	supplemental motor cortex [does not
cortex. [prefrontal, premotor and supplement,	receive from the association areas]
primary motor, parietal (somatosensory),	
temporal (auditory), occipital (visual)].	
To caudate nucleus	To putamen nucleus
Mostly projects to Globus Pallidus, no fibers to	some fibers project to Globus Pallidus then
subthalamus.	to the subthalamus and substantia nigra.
Goes back to the cerebral cortex through the	Goes back to the cerebral cortex through
thalamus, particularly to the premotor and	the thalamus, especially to the primary motor
supplementary motor areas.	area
What the caudate circuit does is integration of all the	What the putamen circuit does is receiving the
information from different lobes and association areas	plan from the caudate circuit and performing the
to conduct <b>a plan</b> about the motor movement we are	motor movements we must do.
going to respond with. That is, most motor actions	
occur as a result of a sequence of thoughts.	
Caudate circuit may play a role in <b>the cognitive</b>	
control of motor functions.	



**3** | Page

These two circuits can tell us some information about the basal ganglia afferents and efferents, but there is more.

Basal ganglia afferents:



Remember that: the centromedial nucleus of thalamus is sensory, while the VA and VL nuclei of the thalamus are motor. The centromedial nucleus and raphe magnus nucleus are related to pain pathways, which indicates that the basal ganglia have a role in cognitive function (emotional part of pain).

Basal Ganglia Efferents:





Motor Function of the Basal Ganglia:

control of **complex patterns** of motor activity: writing, using scissors, throwing balls, shoveling dirt, some aspects of vocalization.

The cortex has a discrete function, it causes contraction of simple muscle fibers. While the basal ganglia are not responsible for single movement, they take care of sequential contraction and relaxation of a group of muscles.

Function of the Basal Ganglia:

Not much is known about the specific functions of each of the structures.

Thought to function in timing and scaling of motion and in the initiation of motion.

Most information comes as a result of damage to these structures and the resulting clinical abnormalities.

Neurotransmitters in the Basal Ganglia:

Cerebral cortex  $\rightarrow$  Ach / Glutamate [excitatory]  $\rightarrow$  Putamen + Caudate

Putamen + Caudate  $\rightarrow$  **GABA** [inhibitory]  $\rightarrow$  Substantia nigra + globus pallidus

Substantia nigra  $\rightarrow$  dopamine [excitatory/inhibitory depends on con.]  $\rightarrow$  Putamen + Caudate



Please study the following picture carefully and notice what each structure sends as a neurotransmitter and the nature of the neurotransmitter (excitatory / inhibitory).





In addition to the functions we mentioned before, when the basal ganglia system is activated diffusely <u>it tends to inhibit muscle tone</u>. [how?] Globus pallidus is considered to have a <u>very high basal rate of firing</u>; it secretes GABA (inhibitory) continuously, leading to <u>the thalamus being inhibited most of the time</u>. If the thalamus is inhibited -> less excitation will reach the supplementary motor area in the cortex -> less excitation to the corticospinal tracts -> muscle tone is suppressed. corticospinal tract is always checked, we inhibit it to the extent that we have the normal muscle tone.

So, what should happen in order to perform an activity?

We stimulate the cortex -> cortex secretes glutamate (excitatory) to the striatum -> striatum secretes GABA (inhibitory), which means that the striatum is going to inhibit globus pallidus -> less GABA (inhibitory) will be secreted from the globus pallidus -> less inhibition on the thalamus; thalamus is active -> supplementary motor area in the cortex is active -> corticospinal tract is active.

Let us imagine someone has a disease in globus pallidus; globus pallidus cannot inhibit the thalamus, the thalamus is always active, the supplementary motor area is active, corticospinal tract is active = **rigidity** [increased muscle tone] of the flexors mainly.

Remember that decerebrate rigidity causes rigidity of the extensors.

Direct and Indirect Pathways of Basal Ganglia:

Direct pathway is basically the excitatory pathway that causes more activation of the muscles, we talked about it when we asked what should happen in order to have activity or movement.





Notice that substantia nigra sends excitatory dopamine to the direct pathway and inhibitory dopamine to the indirect pathway = **dopamine in general is inhibitory to the caudate and putamen nuclei**.

Please refer to the picture in page 5.

The motor control of basal ganglia is much more complex than what we studied, there are connections with the red nucleus, reticular formation, and cerebellum.

Lesions of Basal Ganglia:

All signs and symptoms of basal ganglia diseases are contralateral to the lesion, in contrast with cerebellar lesions which are ipsilateral [double crossing]

<b>Globus pallidus</b> Refer to the first paragraph in page 6 for the explanation	Athetosis – spontaneous writhing movements of the hand, arm, neck, face. [snake-like movements of the distal muscles] <u>https://youtu.be/8DLcS6fx WI</u>
Putamen	<b>Chorea</b> - involuntary flicking movements of the hands, face, and shoulders [acquired]
<b>Caudate nucleus and Putamen</b> loss of GABA containing neurons by globus pallidus and substantia nigra -> inhibition of the thalamus and supplementary motor area [hypotonia]	Huntington's Chorea [inherited]

Substantia Nigra Loss of dopaminergic input from substantia nigra to the caudate and putamen -> caudate and putamen are activated -> direct pathway is continued = increased tone [rigidity] + fine contractions [tremors]. Because of the rigidity, there is an inability to initiate the movements [akinesia], the patients can only do slow movements.	Parkinson's disease – rigidity, resting tremors [bill- rolling] and dys/akinesia. + slow speaking with the same sound tone, less facial expression.
Subthalamus	One side= <b>hemiballismus</b> , two sides = Balismus;
Less glutamate -> inhibition of globus pallidus ->	sudden flailing movements of the entire limb (group
activation of the thalamus and supplementary	of muscles/ grossly)
motor area [hyperkinetic]	<u>https://youtu.be/V6cxZa6gy6g</u>

Integration of Motor control

Spinal cord level	Preprogramming of patterns of movement of all muscles (i.e., withdrawal reflex, walking movement, etc.)
Brain stem level	Maintains equilibrium by adjusting axial tone (balance)
Cortical level	Issues commands to set the patterns available in the spinal cord into motion. Controls the intensity and modifies the timing.
Cerebellum	Function with all levels of control to adjust spinal cord motor activity, equilibrium, and planning of motor activity
Basal ganglia	Functions to assist the cortex in executing subconscious but learned patterns of movement, and to plan sequential patterns to accomplish a purposeful task