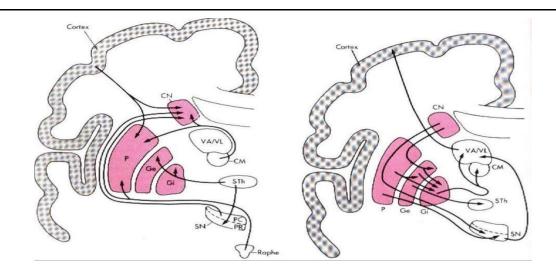


PHYSAOLOGY

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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.

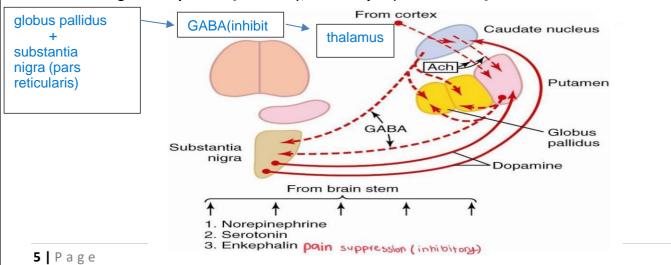
Most information comes as a result of damage to these structures and the resulting clinical abnormalities.

Neurotransmitters in the Basal Ganglia:

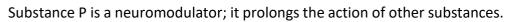
Cerebral cortex \rightarrow Ach / Glutamate [excitatory] \rightarrow Putamen + Caudate

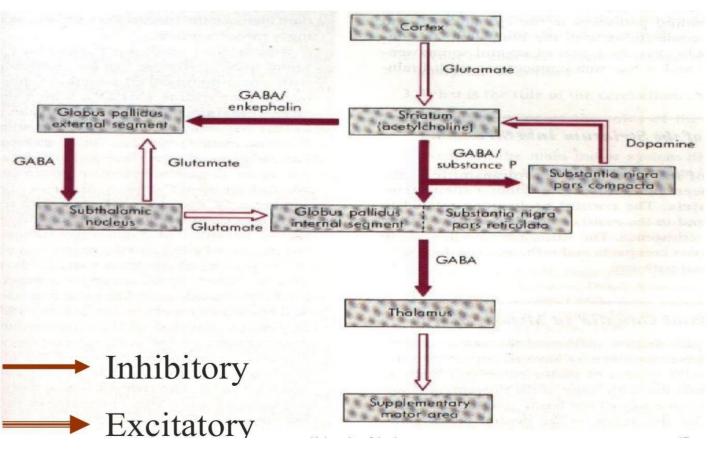
Putamen + Caudate \rightarrow **GABA** [inhibitory] \rightarrow Substantia nigra + globus pallidus

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?

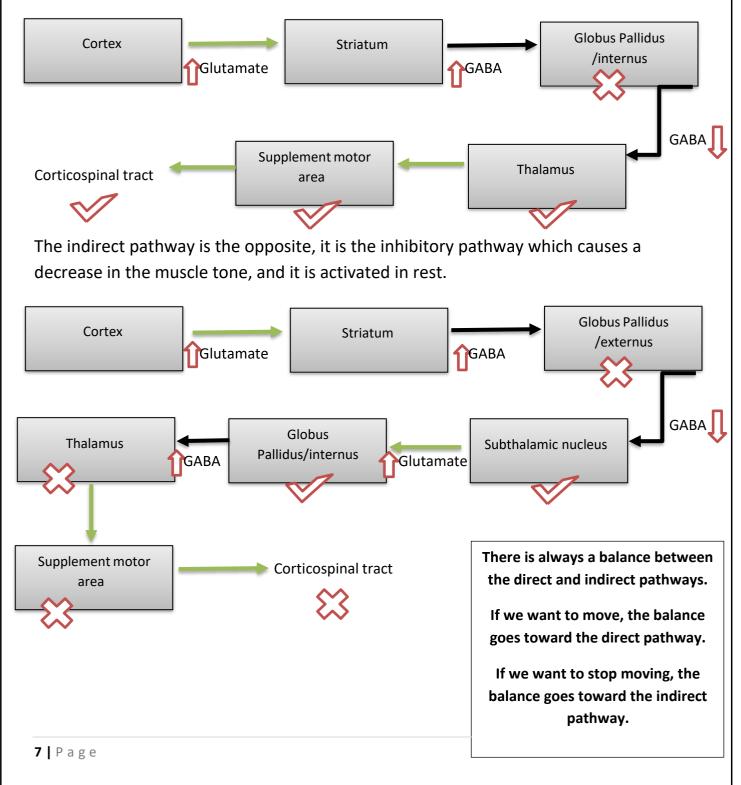
We stimulate the cortex -> cortex secretes glutamate (excitatory) to the striatum -> striatum secretes GABA (inhibitory), which means that the striatum is going to inhibit globus pallidus -> less GABA (inhibitory) will be secreted from the globus pallidus -> less inhibition on the thalamus; thalamus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

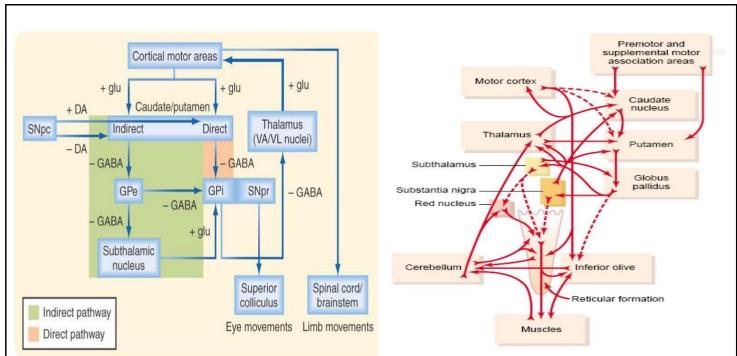
Let us imagine someone has a disease in globus pallidus; globus pallidus cannot inhibit the thalamus, the thalamus is always active, the supplementary motor area is active, corticospinal tract is active = **rigidity** [increased muscle tone] of the flexors mainly.

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]

Globus pallidus 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	Chorea - involuntary flicking movements of the hands, face, and shoulders [acquired]
Caudate nucleus and Putamen 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= hemiballismus , 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