

1. PURPOSE

The basal ganglia moderates the intensity of motor activity.

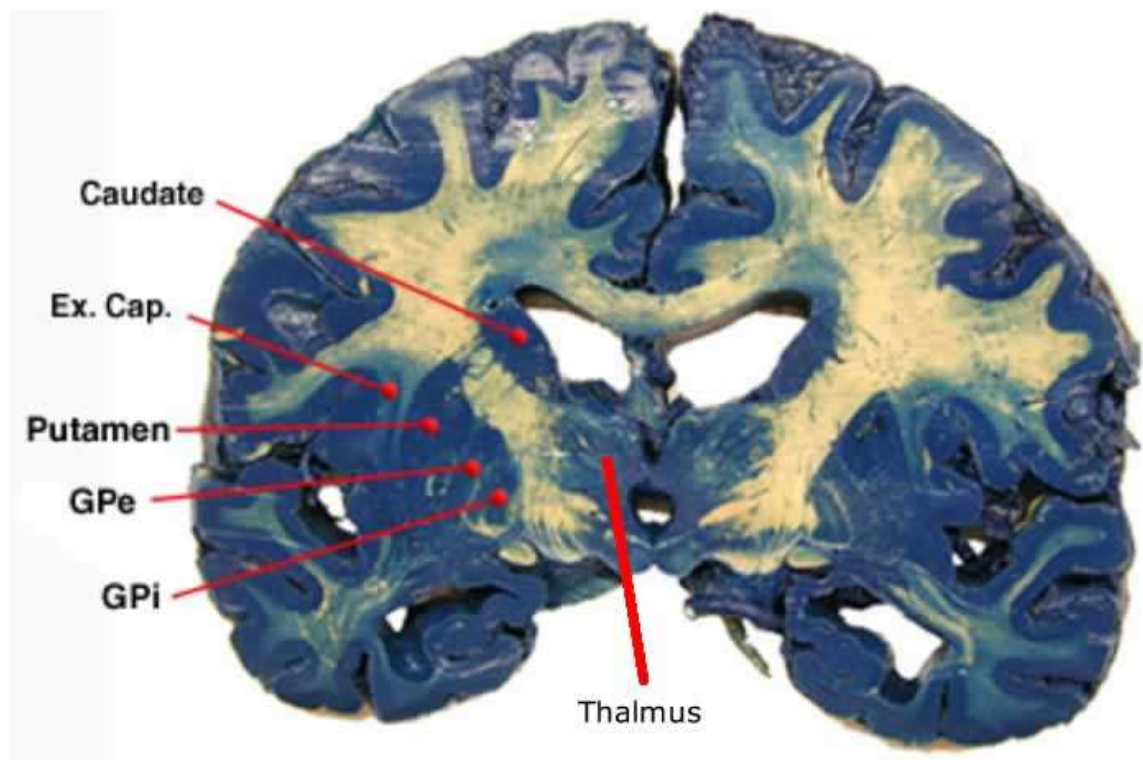
It does this by inhibiting the thalamus to a lesser or greater extent.

2. ANATOMY & NOMENCLATURE

The basal ganglia consists of a group of large gray matter masses located deep within the cerebral hemisphere. It includes the following distinct parts:

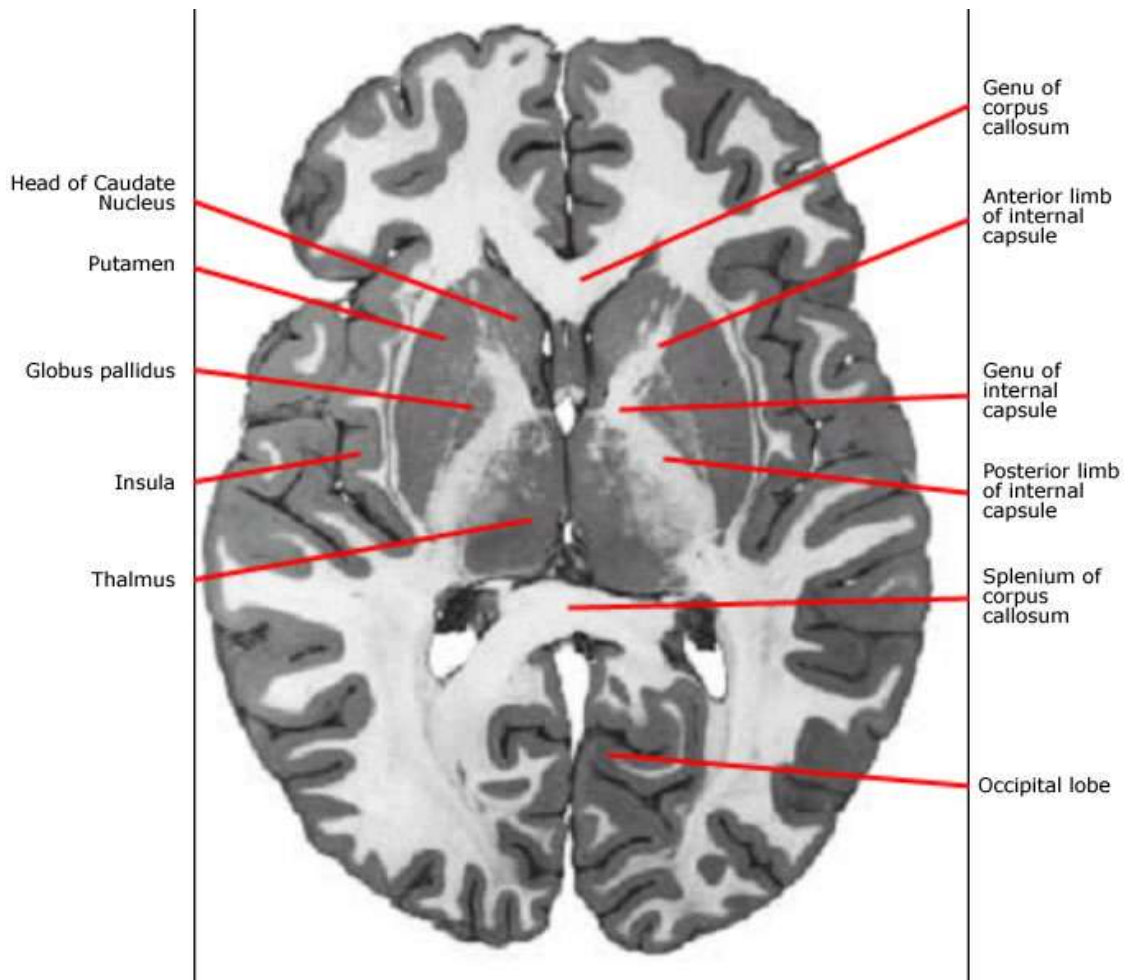
1. Caudate nucleus
2. Putamen
3. Globus pallidus [external] ~ abbreviated "GPe"
4. Globus pallidus [internal] ~ abbreviated "GPi"
5. Subthalamic nucleus ~ abbreviated "STN"
6. Substantia Nigra [pars compacta] ~ abbreviated "SNc"
7. Substantia Nigra [pars reticulata] ~ abbreviated "SNr"

Most of these structures show up clearly on scans -- we should be able to recognize the caudate, putamen, and globus pallidus on stained coronal scans.



("Ex. Cap" = External Capsule)

Also, we ought be able to recognize the caudate, putamen, and globus pallidus on stained horizontal views.



As a stupid memory thing, I use the phrase 'Catapult globe' to remind myself that if you look at either a coronal or horizontal scan, the caudate nucleus is at the top, followed by the putamen, and globus pallidus. This phrase helps me remember the striatum terminology as well (see below)

In addition to the aforementioned structures, here's a few important terms which refer to groupings of basal ganglia components:

1. Corpus Striatum = Caudate nucleus + Putamen + Globus pallidus
2. Striatum = Caudate nucleus + Putamen
3. Neostriatum = Caudate nucleus + Putamen
4. Paleostriatum = Globus pallidus
5. Lenticular nucleus = Putamen + Globus pallidus
 - a. This term is easy to remember, since if you look at the horizontal scan above, you can see that the putamen and globus pallidus are right next to each other, and look like a lens when together (Lentiform = 'lens-shaped')

3. FUNCTION

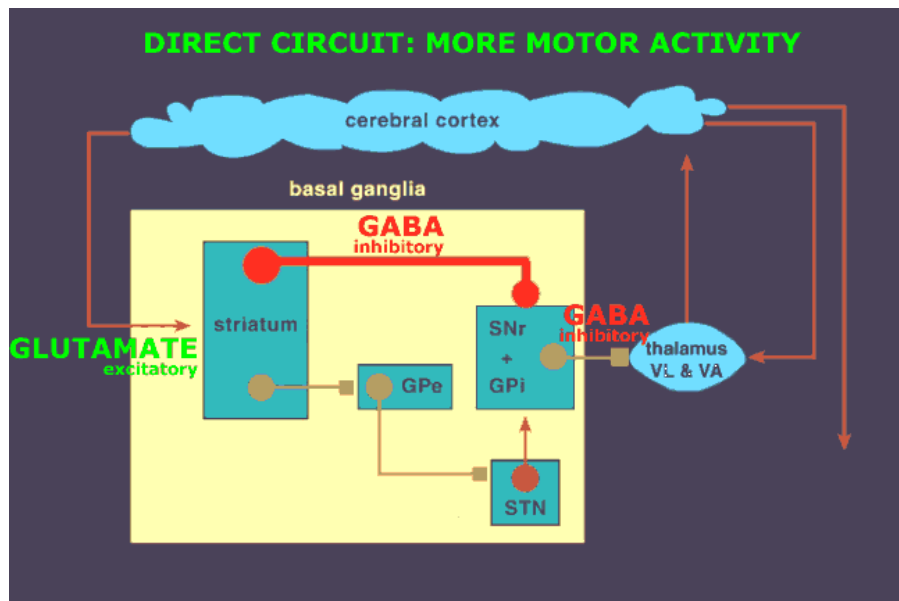
As noted before, the purpose of the basal ganglia is to "moderate the intensity of motor activity". This is done by 3 methods:

1. Activation of direct circuit, which increases motor activity
2. Activation of indirect circuit, which decreases motor activity
3. Stimulation of Substantia Nigra compacta, which increases motor activity

All three of these methods need a closer look.

3.1 THE DIRECT CIRCUIT

The 'direct circuit' is a pathway that the basal ganglia uses to increase motor activity. Here's the schematic:



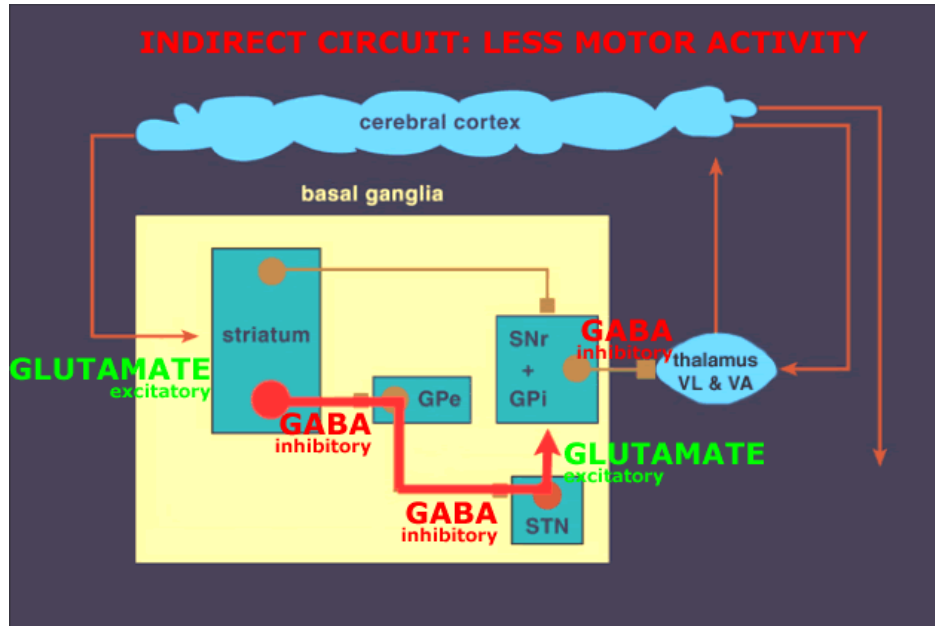
Here's how it works:

1. Cortex sends excitatory transmitter (glutamate) to striatum (i.e. the caudate and putamen)
2. Striatum sends GABA, an inhibitory transmitter, to slow activity in the Substantia Nigra reticulata (SNr) and internal Globus pallidus (GPi)
3. **Since they've been slowed down, the SNr and GPi produce less of their own neurotransmitter, GABA**
4. A smaller amount of GABA reaches the thalamus = the thalamus is less inhibited
5. Thalamus activity increases
6. Motor activity increases

This is really tricky stuff (at least for me) -- to put it another way, the direct circuit shows inhibition of inhibition of the thalamus, which causes excitation of the thalamus, which causes increased motor activity.

3.2 THE INDIRECT CIRCUIT

The 'indirect circuit' is a pathway that the basal ganglia uses to decrease motor activity. It's called the 'indirect circuit' since it has more steps than the 'direct circuit'. Here's the schematic:



Here's how it works:

1. Cortex sends excitatory transmitter (glutamate) to striatum (i.e. the caudate and putamen)
2. Striatum sends GABA, an inhibitory transmitter, to slow activity in the external Globus pallidus (GPe)
3. Since it has been slowed down, the GPe produce less of it's own neurotransmitter, GABA
4. A smaller amount of GABA reaches the Subthalamic nuclei (STN) = the subthalamic nuclei is less inhibited
5. The subthalamic nuclei releases more of it's own neurotransmitter (glutamate)
6. More glutamate (an excitatory transmitter) reaches the Substantia nigra reticulata (SNR) and internal Globus pallidus (GPi)
7. **Since they are swamped with excitatory transmitter, the SNR and GPi produce more of their own neurotransmitter, GABA**
8. A greater amount of GABA reaches the thalamus = the thalamus is more inhibited
9. Thalamus activity decreases
10. Motor activity decreases

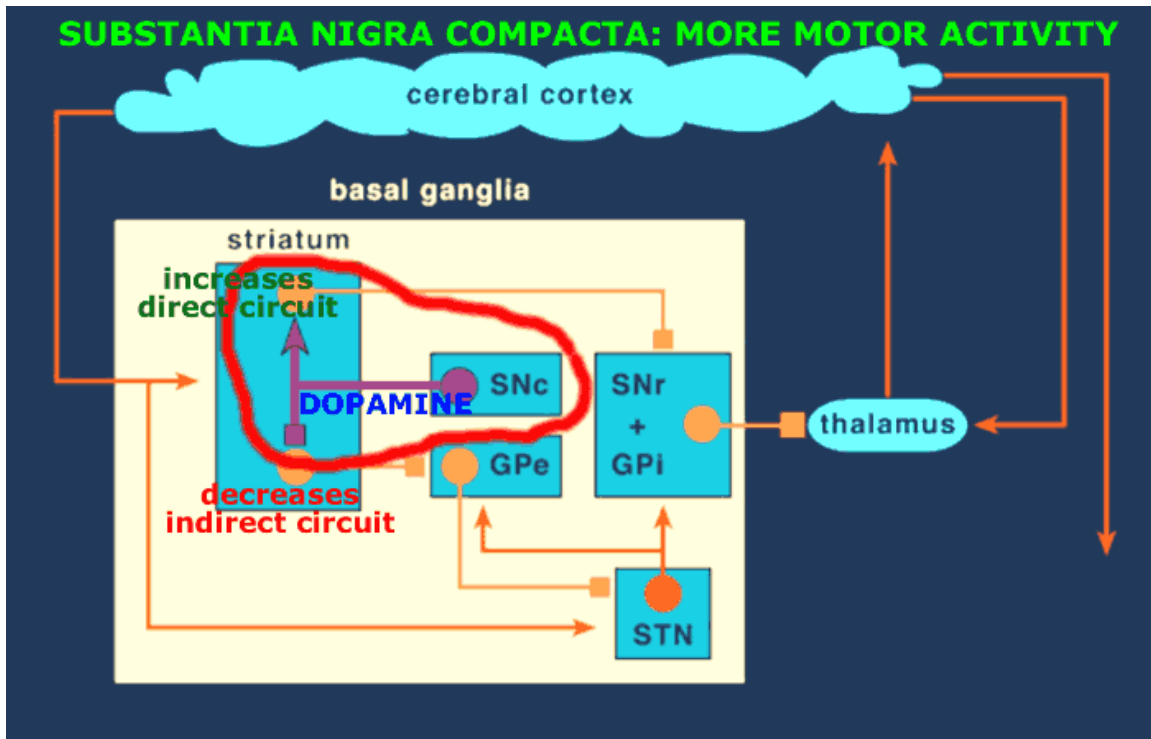
This is really complicated, but the take-home message is that the Substantia nigra reticulata (SNR) and internal Globus pallidus (GPi) are releasing more inhibitory transmitters, thus decreasing thalamus activity and motor activity.

3.3 THE SUBSTANTIA NIGRA COMPACTA

The substantia nigra compacta is used to increase motor activity.

However, unlike the previous two circuits, this pathway does NOT dump transmitter on the thalamus.

Here's a schematic:



Here's how it works:

1. Substantia nigra compacta (SNc) sends the transmitter dopamine to putamen
2. **Dopamine excites direct circuit**
3. **Dopamine inhibits indirect circuit**

This pathway has a double-whammy effect -- if you excite the direct circuit, you increase motor activity, and if you inhibit the indirect circuit, you also increase motor activity.

IMPORTANT: Parkinson's disease is closely tied to this pathway -- Parkinson's disease is characterized by dopamine deficiency, which is clinically manifested through slow movements and small movements (pill-rolling syndrome)

4 CLINICAL SIGNS

If there's a lesion in the nervous system, it can cause two kinds of problems with the motor system:

1. **Negative signs**, which is the loss of a function
2. **Positive signs**, which is the addition of new motor activities or responses, which are expressed due to a release from inhibition

Here's a chart of important negative and positive signs

NEGATIVE SIGNS (loss motor function)	POSITIVE SIGN (new motor function)
Akinesia = lack of movement	Lead-pipe / plastic rigidity = uniform resistance when a limb is passively stretched
Bradykinesia = slowness of movement	Cog-wheel rigidity = ratchet-like resistance when a limb is passively stretched
Masked facies = mask-like facial expression, infrequent blinking, "reptilian stare"	Athetosis = inability to maintain a fixed or sustained posture
Dystonia = involuntary muscle contractions (also a positive sign)	Dystonia = involuntary muscle contractions
	Chorea = involuntary arrhythmic movements, forceful, rapid, and jerky.
	Ballismus = violent flinging of the limbs

5 DISORDERS

Disorders with the basal ganglia can be lumped into two groups -- (1) hyperkinetic disorders, and (2) hypokinetic disorders.

Hyperkinetic disorders

- **Direct pathway active, indirect pathway inhibited**
- Thalamus = less inhibited, more motor activity
- Results in dyskinesias (uncontrolled movement), such as ballismus (violent flinging of limbs)

Hypokinetic disorders

- **Direct pathway inhibited, indirect pathway active**
- Thalamus = more inhibited, less motor activity
- Results in bradykinesia (slowness of movement) or akinesia (lack of movement)

6 OTHER DISORDERS

In addition to knowing the generalized categories of hyperkinetic / hypokinetic disorders, there's a few specific basal ganglia disorders that are important to know:

1. Parkinson's disease
 - a. **Hypokinetic disorder** (decreased muscle activity)
 - i. Tremor
 - ii. Rigidity
 - iii. Bradykinesia = slowness of movement
 - b. DOPAMINE deficiency
2. Huntington's disease
 - a. **Hyperkinetic disorder** (increased muscle activity)
 - i. Rigidity
 - ii. Chorea = involuntary arrhythmic movements, rapid and jerky
3. Tardive Dyskinesia
 - a. Movement disorder induced by narcolepsy
 - i. Narcolepsy is a sudden and uncontrollable attack of deep sleep
 - b. Dopamine transmission is affected, leads to **hypokinesia** (decreased muscle activity)
4. Wilson Disease
 - a. Hepalenticular degeneration (remember that the lenticular = Putamen and Globus pallidus)
 - b. Causes disorder of copper metabolism
 - i. Can see a copper-colored ring surrounding the cornea (in the eye)
5. Tourette Syndrome
 - a. **Hyperkinetic disorder** (increased muscle activity)
 - i. Motor tics (brief jerks to complex movements)
 - ii. Coprolalia (obscene, foul language bursts)
 - iii. Excessive throat clearing