

27 BASAL GANGLIA

CNS



Sources:

- ✓ females slides
- ✓ Dr. najeeb videos

Physiology of basal ganglia and regulatory mechanisms

Objectives

- 1-appreciate different nuclei of basal ganglia
- 2-know different neurotransmitters that have a role in basal ganglia functions
- 3-appreciate general functions of basal ganglia
- 4-diagnose basal ganglia disorders

Terms

Striatum : **St**

Internal Globus Pallidus : **GPI**

external Globus Pallidus : **GPe**

Subthalamic Nucleus : **SThn**

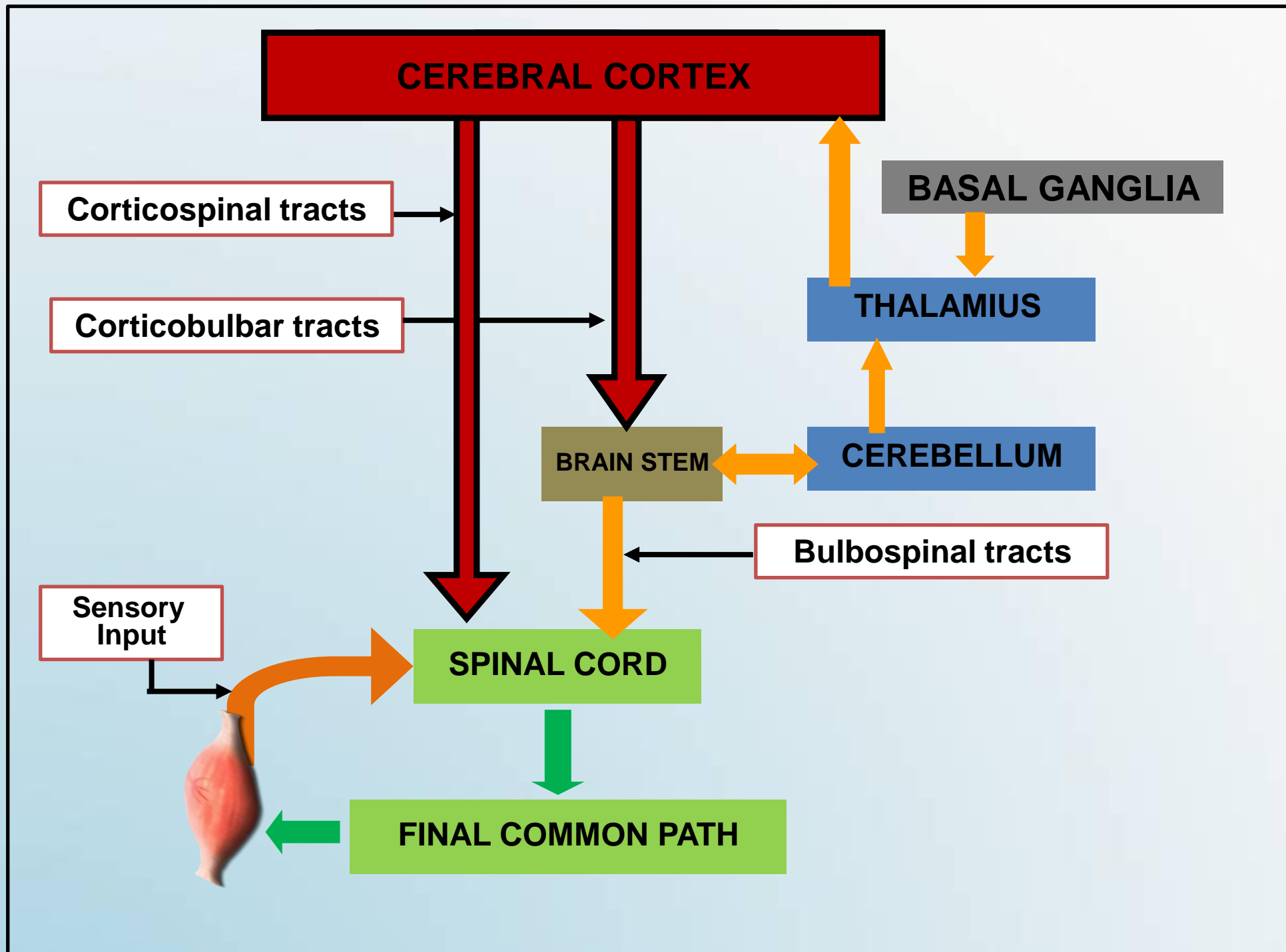
Pars compacta of Substantia Nigra : **SNPC**

Glutamine : **GUL**

D1 Dopamine receptor (excitatory) : **DA1+**

D2 Dopamine receptor (inhibitory) : **DA2-**

Overview Of Motor Activity Control



Not only the :
corticospinal tracts and corticobulbar tracts
Control the motor activity , It is control also by : **cerebellum and basal ganglia**

Cerebellum and basal ganglia receive information from cerebral cortex and send information to thalamus, which send it to cerebral cortex

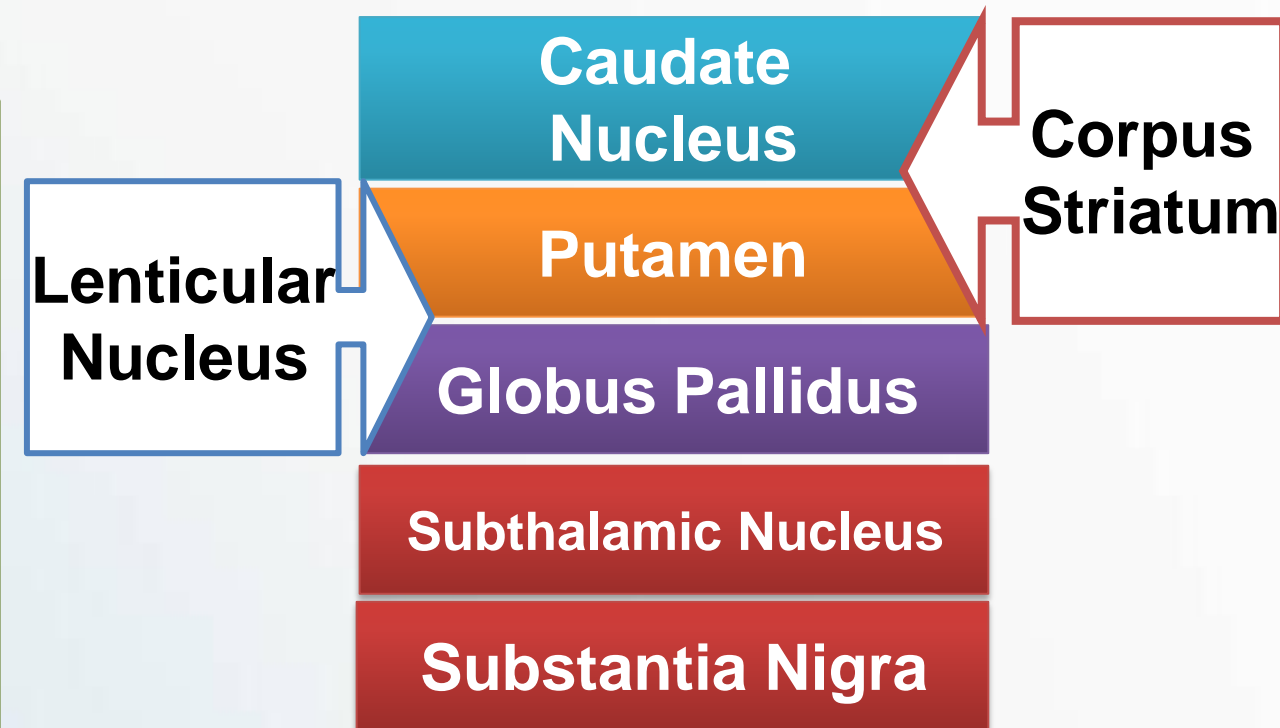
BASAL GANGLIA

Basal ganglia are subcortical nuclei of grey matter located in the interior part of cerebrum near about base

Components functional anatomy

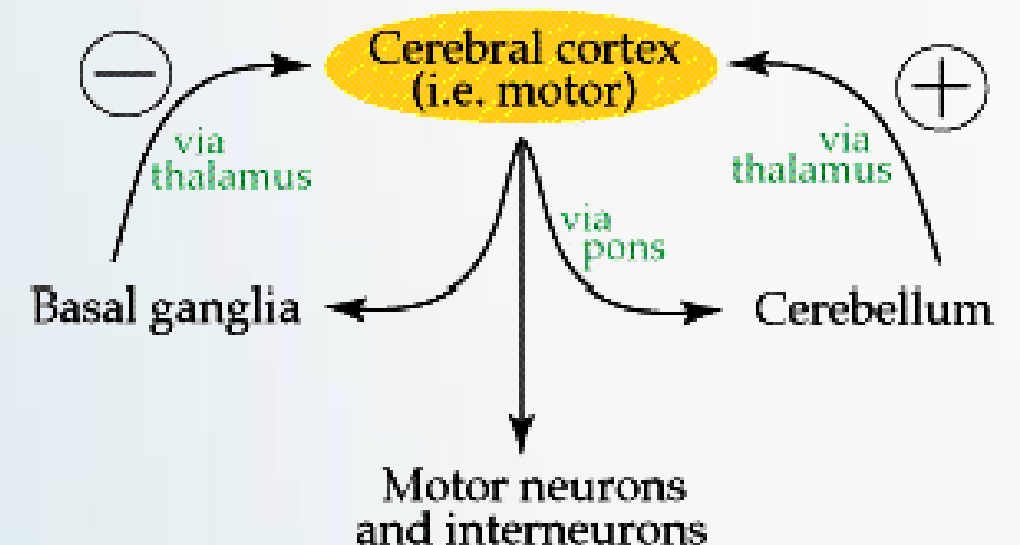
1. Caudate Nucleus
2. Putamen
3. Globus Pallidus
4. Subthalamic Nucleus
5. Substantia Nigra

Corpus Striatum = Caudate Nucleus + Putamen
Lenticular Nucleus = Putamen + Globus Pallidus



Connections

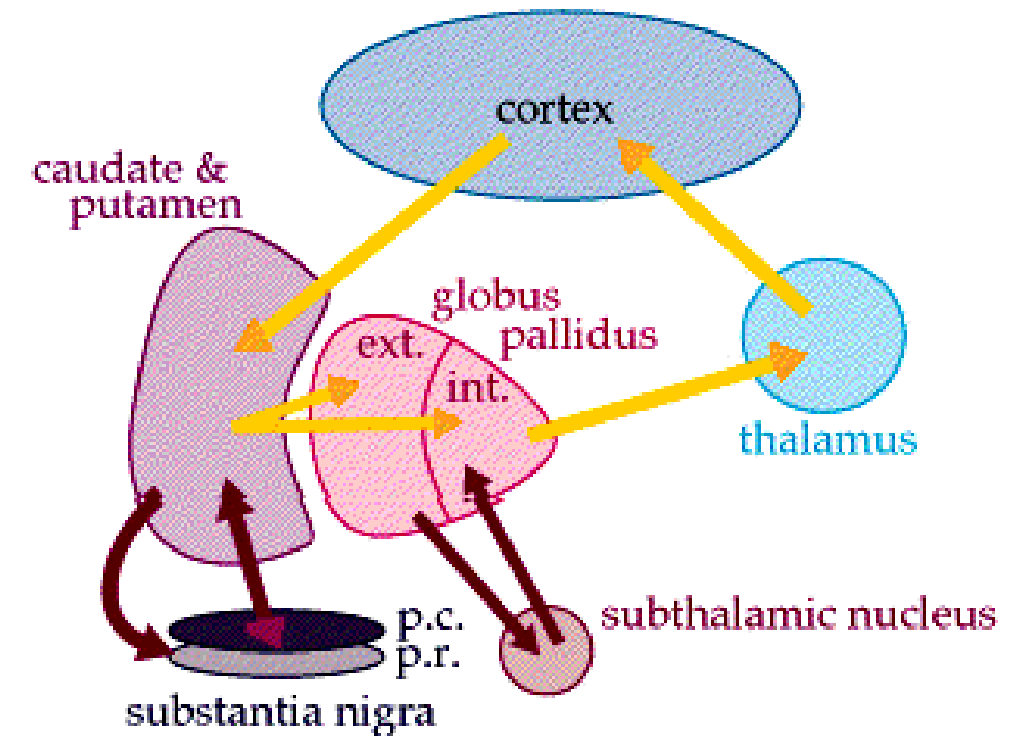
basal ganglia receive information from cerebral cortex and send information to cerebral cortex via thalamus.



Connections

3 Connections to remember

1. Main input to the basal ganglia
2. Main output from the basal ganglia
3. Connections between parts of basal ganglia



MAIN INPUT TO THE BASAL GANGLIA

The comes from the cerebral cortex (motor area) and projects to the **NEOSTRIATUM** (a term for the caudate nucleus and putamen)

THE MAIN OUTPUT

Is via the thalamus to the cerebral cortex (motor area)

Basic Circuits Of Basal Ganglia

1. **Motor loop (putamen circuit)** concerned with learned movement.
2. **Cognitive loop (Caudate circuit)** concerned with cognitive control of sequences of motor pattern. Basically it is concerned with motor intentions.
(Note: cognition means thinking process using sensory input with information already stored in memory.)
3. **Limbic loop** involved in giving motor expression to emotions like, smiling, aggressive or submissive posture.
4. **Occulomotor loop** concerned with voluntary eye movement [saccadic movement]

FUNCTIONS

1. Control of movements
2. Planning and programming of movements
3. Cognition

1. The Putamen Circuit (inhibitory)

The **information from : Premotor, Supplemental Motor and Somatosensory Cortex** go to **putamen** then to Globus Pallidus. Fibers from Globus Pallidus go to :
1. Sub thalamic Nucleus 2. Substantia Nigra 3. Thalamus
Fibers from Sub thalamic Nucleus and Substantia Nigra go to thalamus
• **All fibers from thalamus go to Primary Motor Cortex, Premotor and Supplemental Motor Areas**

Function :

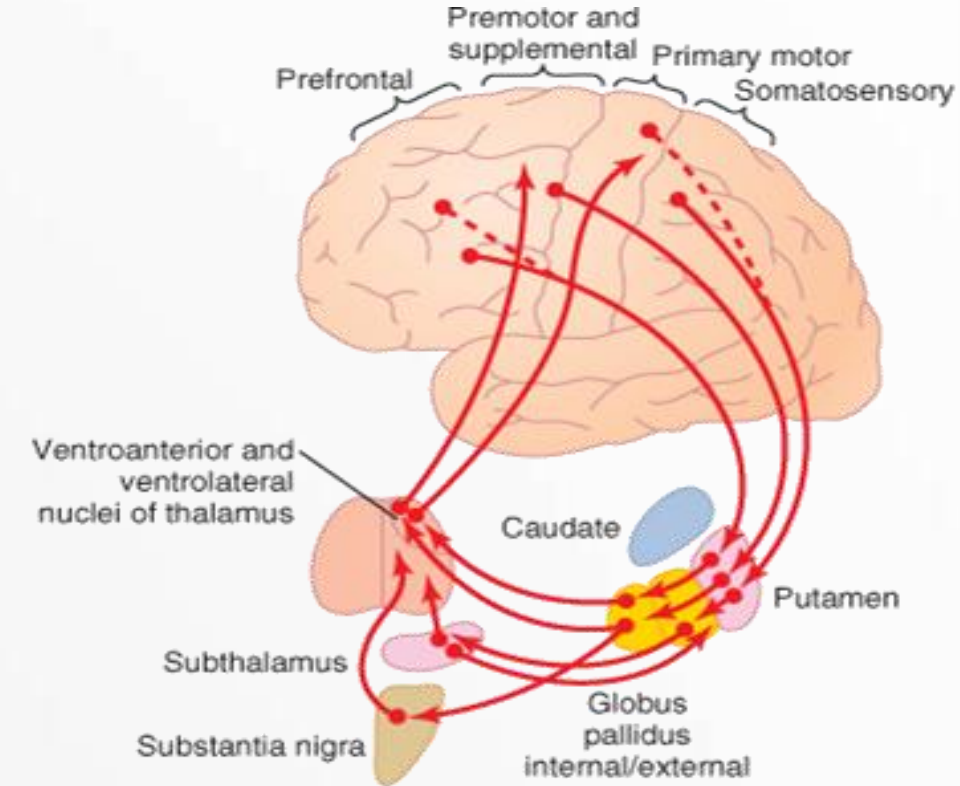
Executes complex learned pattern of motor activity



Putamen circuit in association with corticospinal system → control skilled movement

- **For example** : writing of letters of the alphabet, cutting paper with scissors, hammering nails, shooting a basketball through a hoop, passing a football, throwing a baseball, the movements of shoveling dirt, most aspects of vocalization, controlled movements of the eyes .

Most of these skilled movement performed **subconsciously**



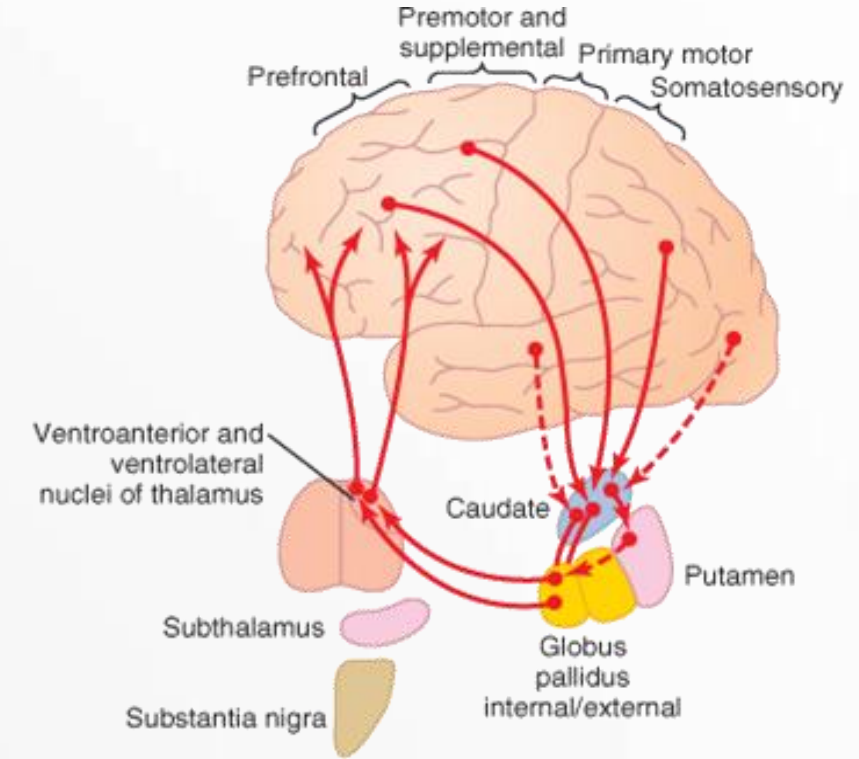
2. The Caudate Circuit (excitatory)

The **information from : Association**

Areas go to **Caudate** then to putamen or Internal Globus Pallidus. Fibers from putamen go to : Internal Globus Pallidus

All Fibers from Internal Globus Pallidus go to thalamus

All fibers from thalamus go to Prefrontal , Premotor and Supplemental Motor Areas



Function :

1- cognitive control of motor activity

(works quickly without thinking “**Subconsciously**”)

على سبيل المثال : لما تقابل أسد في غابة ما ، لحظيا وبلا وعي سوف تلتفت مبتعدا وتبدا تركض بلا هوادة
وممكن تتسلق شجرة لو لقيتها بطريقك ..

2- change the timing & scale the intensity of movement .

- Controlling any movement involved the following :

A- determine how rapidly the movement is to be performed .

B- determine how large the movement will be .

على سبيل المثال : لما تكتب حرف (أ) بسرعة او ببطئ او لما تكتبه بخط صغير او بخط كبير .. ما رح يتغير
شكل حرف (أ) ورح يكون شكل الحرف متشابه بالاربع حالات .

Basal Ganglial Pathways

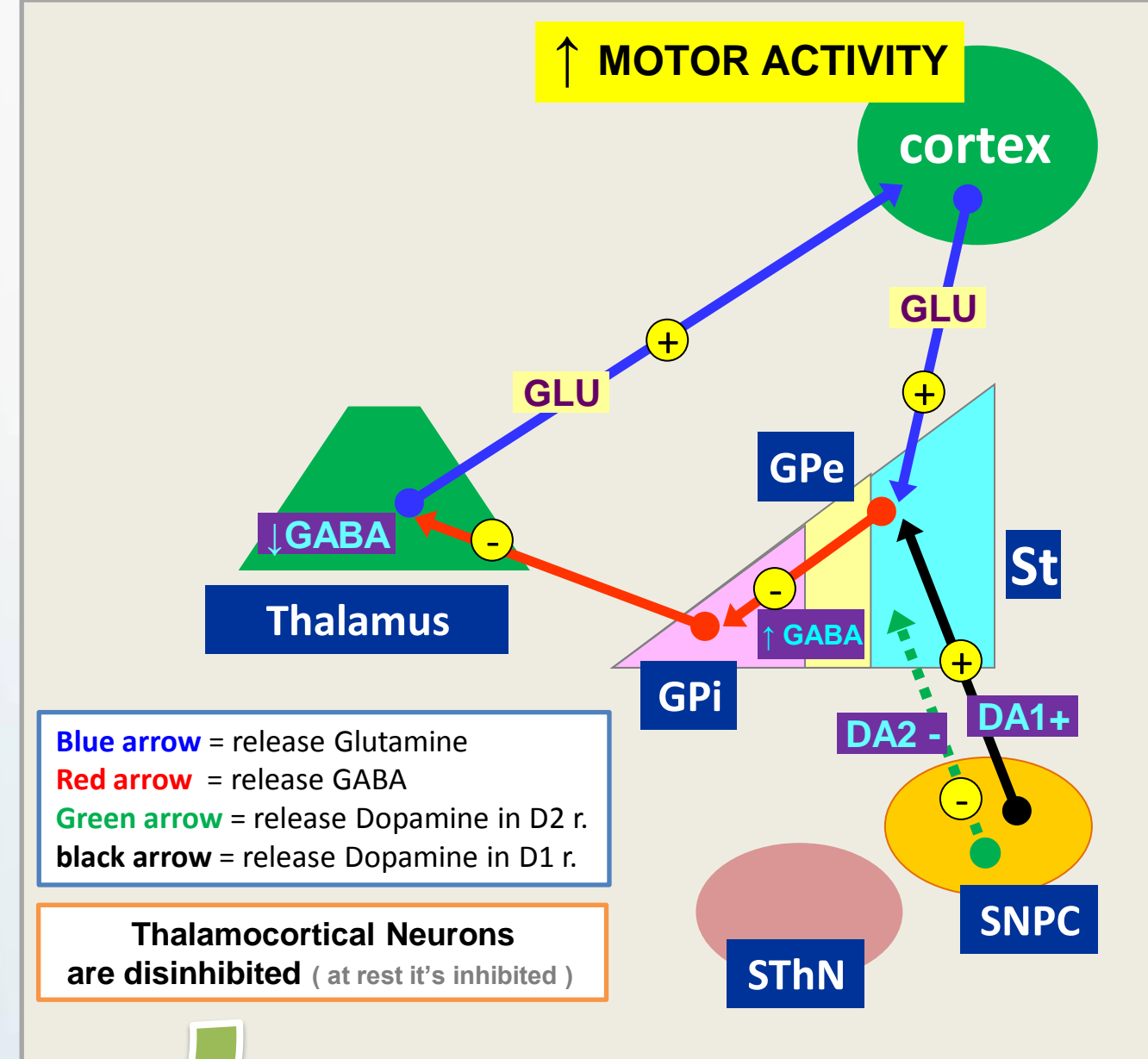
Components : to understand

1. Putamen
2. Internal Globus Pallidus
3. external Globus Pallidus
4. Thalamus
5. Subthalamic Nucleus
6. Substantia Nigra
7. Glutamine
8. GABA
9. Dopamine, D1 receptor (excitatory), D2 receptor (inhibitory)

1. Direct

Fibers :

1. excitatory Glutamine Fibers from cortex to Putamen (**Corticostriatum fibers**)
2. inhibitory GABA Fibers from Putamen to Internal Globus Pallidus
3. inhibitory GABA Fibers from Internal Globus to Thalamus
4. excitatory Glutamine Fibers from Thalamus to cortex (**Thalamocortical fibers**)



Do Motor Activity

Information send to **Putamen** from **cortex** by corticoStriatal fibers which **release the Glutamine** to excitatory the Fibers from Putamen to **Internal Globus Pallidus** to release **more GABA**. GABA is **inhibitory** the Fibers from Internal Globus to **Thalamus** to release **less GABA**. Decrease GABA in Thalamus causes **Stimulate** for Fibers from Thalamus to **cortex** (Thalamocortical Neurons) to release the **Glutamine** and excitatory the cortex → **↑ MOTOR ACTIVITY**

At Rest

Information **doesn't send to Putamen** from cortex → **no Glutamine** released in Putamen → Fibers from Putamen to Internal Globus Pallidus release **less GABA** → excitatory the Fibers from Internal Globus to Thalamus to release **more GABA**. increase GABA in Thalamus causes **inhibition** for Fibers from Thalamus to cortex → **doesn't** release the **Glutamine** → inhibition the cortex → **↓ MOTOR ACTIVITY**

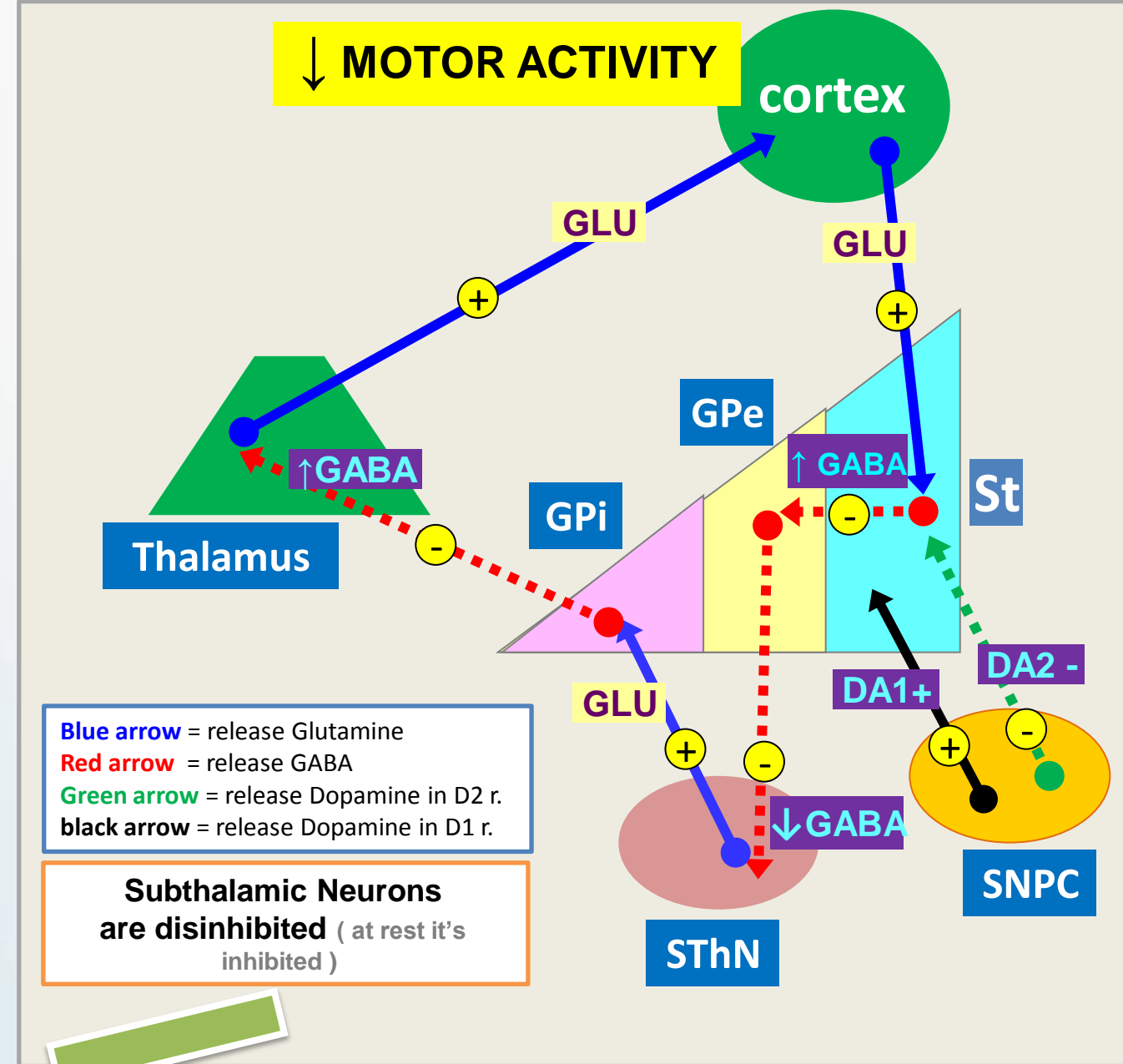
Basal Ganglial Pathways

2. Indirect

- to do motor activity, we need to excitatory some muscles or\and inhibitory some other muscles .
- Indirect Pathways include the Subthalamic Nucleus

Fibers :

1. excitatory Glutamine Fibers from cortex to Putamen (Corticostriatum fibers)
2. inhibitory GABA Fibers from Putamen to external Globus Pallidus
3. inhibitory GABA Fibers from external Globus Pallidus to Subthalamic Nucleus
4. excitatory Glutamine Fibers from Subthalamic Nucleus to Internal Globus Pallidus
5. inhibitory GABA Fibers from Internal Globus Pallidus to Thalamus
6. excitatory Glutamine Fibers from Thalamus to cortex (Thalamocortical fibers)



Information send to **Putamen** from **cortex** by corticoStriatal fibers which **release the Glutamine** to excitatory the Fibers from Putamen to **external Globus Pallidus** to **release more GABA**. GABA is **inhibitory** the Fibers from external Globus to **Subthalamic Nucleus** to release **less GABA**. Decrease GABA in Subthalamic Nucleus causes **Stimulate** for Fibers from Subthalamic Nucleus to **Internal Globus Pallidus** to release the **Glutamine in Gpi** and **Stimulate fibers** from Internal Globus Pallidus to **Thalamus** to release the **more GABA** in Thalamus. increase GABA in Thalamus causes **inhibition** for Fibers from Thalamus **to cortex** (Thalamocortical Neurons) → **doesn't** release the **Glutamine** → **inhibition** the cortex → ↓ **MOTOR ACTIVITY**

Basal Ganglial Pathways

Dopamine role

The dopamine neurons is in **Substantia Nigra**

Pars compacta of Substantia Nigra send 2 type of Dopamine fibers to **Putamen**, to effect the neurons in it.

	D1 receptor dopamine neurons	D2 receptor dopamine neurons
Action	Excitatory Putamen neurons → Release more GABA in Internal Globus Pallidus	Inhibitory Putamen neurons → Release less GABA in external Globus Pallidus
Receptors in Putamen neurons	D1 receptor	D2 receptor
substance release	Dopamine	Dopamine
The Pathways which work in it	Direct	indirect
their Effect in pathway	More ↑ MOTOR ACTIVITY	↑ MOTOR ACTIVITY

Metabolic characteristics

- High Oxygen consumption .
- High Copper content in **Wilson's disease** (Copper intoxication) Autosomal Recessive
- Copper binding protein **Ceruloplasmin** is low
- Lenticular degeneration occurs

Disorders of basal ganglia

- These disorders can involve : movements, speech, posture, gait, mental activity or others.

- The movements disorders are classified to :

1. Hyperkinetic such as : hemiballismus, huntington's disease & athetosis.
2. Hypokinetic such as : parkinson's disease, drug induced (neuroleptics, MPTP)

Some movement disorders in brief :

Movement Disorder	Features	Lesion
Chorea	Multifocal quick, random movements, usually most prominent in the appendicular muscles	Atrophy of the striatum. Huntington Chorea
Athetosis	Slow writhing movements, which are usually more severe in the appendicular muscles	Diffuse hypermyelination of corpus striatum and thalamus
Hemiballismus	Wild flinging movements of half of the body	Hemorrhagic destruction of contralateral subthalamic n. Hypertensive patients
Parkinsonism	Pill rolling tremor of the fingers at rest, lead pipe rigidity and akinesia	Degeneration of Substantia Nigra

Parkinson's disease

- Degeneration of 60-80 % of dopaminergic nigrostriatal neurons
- Loss of DA → loss of excitatory effect on direct & lose inhibition of indirect
- Overactive Gpi & STN → ↑ inhibition of motor thalamus

- 5 cardinal features :

- 1- tremor
- 2- rigidity
- 3- akinesia & bradykinesia
- 4- postural changes
- 5- speech changes

Effect of basal ganglia on motor activity are generally inhibitory

Huntington's disease

- Hereditary, autosomal dominant disease.
- It's also called "disease of caudate & putamen"
- Loss of GABA – Cholinergic neurons

-Symptoms & signs :

- 1- **Jerky** movement of hands toward end of reaching an object which is called "chorea" due to loss of GABAergic neurons.
- 3- slurred speech & incomprehensible.
- 4- progressive dementia .

Effects of Lesions of basal ganglia are contralateral.

Damage to basal ganglia does not cause paralysis

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CNS Block