Overview

Movement control : M1 & Basal Ganglia

Reading – Purves Chapter 17 pp. 381 - 397; Chapter 18 pp. 407 - 417

Summary

Motor cortex

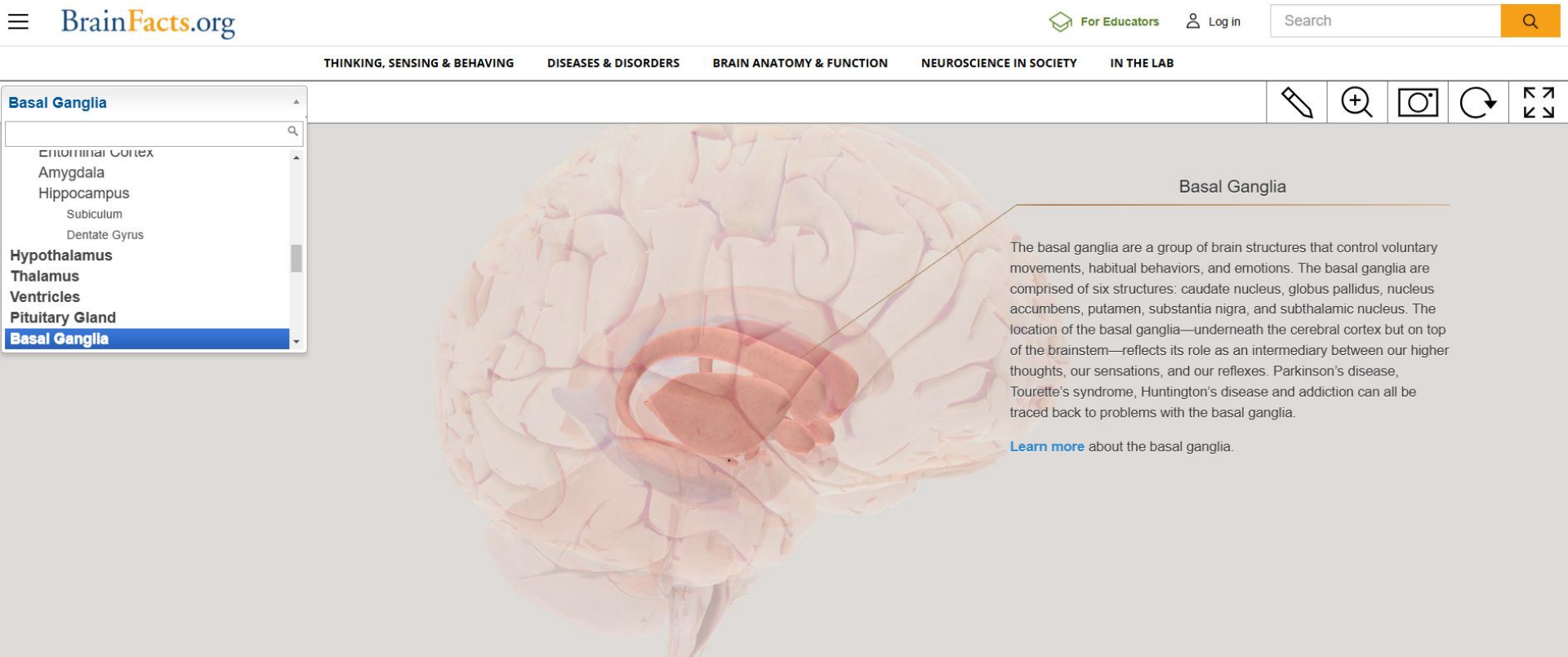
- Primary motor cortex (M1), output layer (L5), lateral corticospinal tract
- « Coding » of movement primitives in M1
- Principles and applications of a Brain-Machine Interface

Basal ganglia

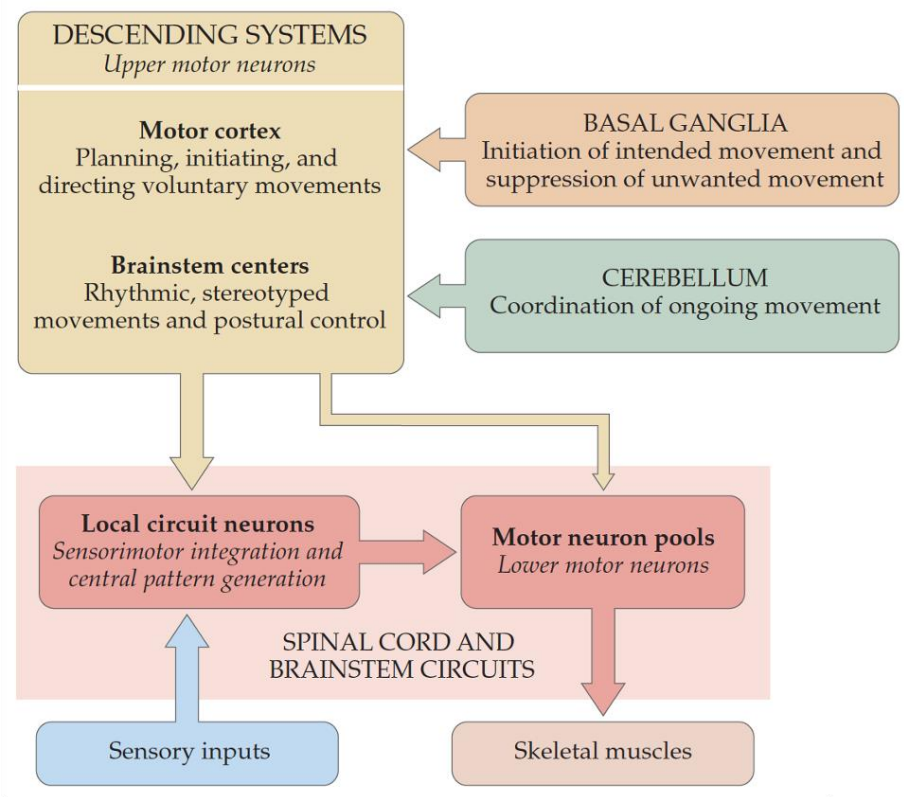
- Principal nuclei and their connectivity
- Medium spiny neurons (MSNs) in the striatum
- Direct and indirect pathways: modulation and role in action selection and movement
- Clinical cases of Parkinson’s and Hungtington’s disease

Convenient tool

Visualise the brain regions in 3D!!
https://www.brainfacts.org/3d-brain#intro=false&focus=Brain&zoom=false

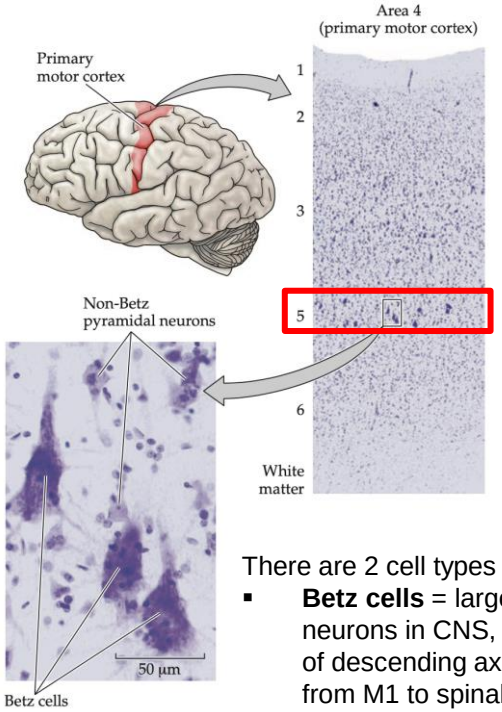


Principle brain areas involved in motor control



- ✓ **Spinal cord (and brainstem)**
  - signal relay
  - sensorimotor integration
  - CPG
- ✓ **Cerebellum**
  - error correction & coordination of ongoing movements
- ➢ **Descending control centers in the cerebral cortex**
  - voluntary movements planning
- ➢ **Basal ganglia**
  - initiation of intended movements
  - suppression of unwanted movements

M1 and lateral corticospinal tract



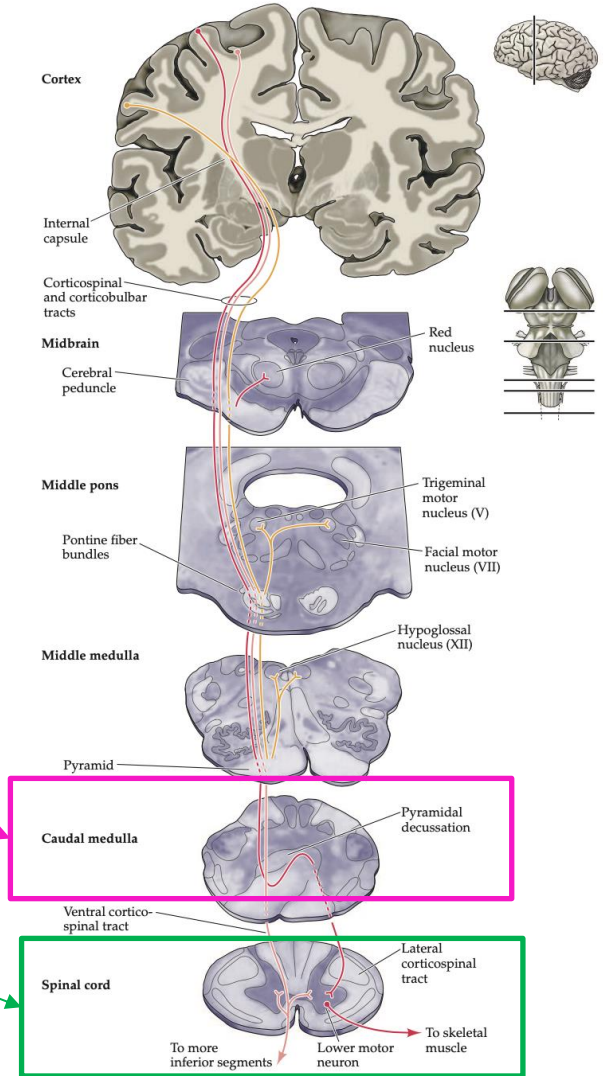
There are 2 cell types in L5:

- **Betz cells** = largest neurons in CNS, only 5% of descending axons from M1 to spinal cord
- **Non-Betz cells**

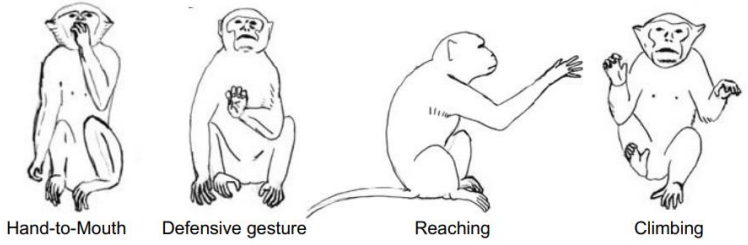
The pyramidal cells of the cortical layer **L5** are the upper motor neurons of M1.

«pyramidal decussation»: axons cross-over

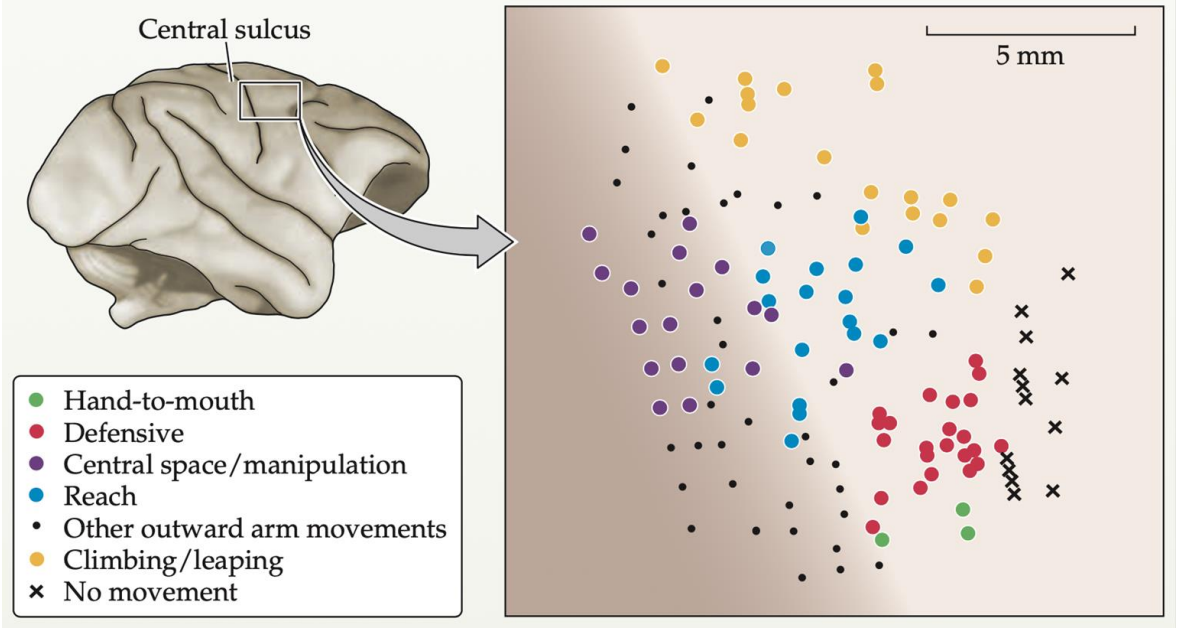
Some axons directly contact lower motor neurons, but most contact interneurons



“Coding” of movement primitives in M1



The functional maps in the primary motor and premotor cotex are **maps of movement**.



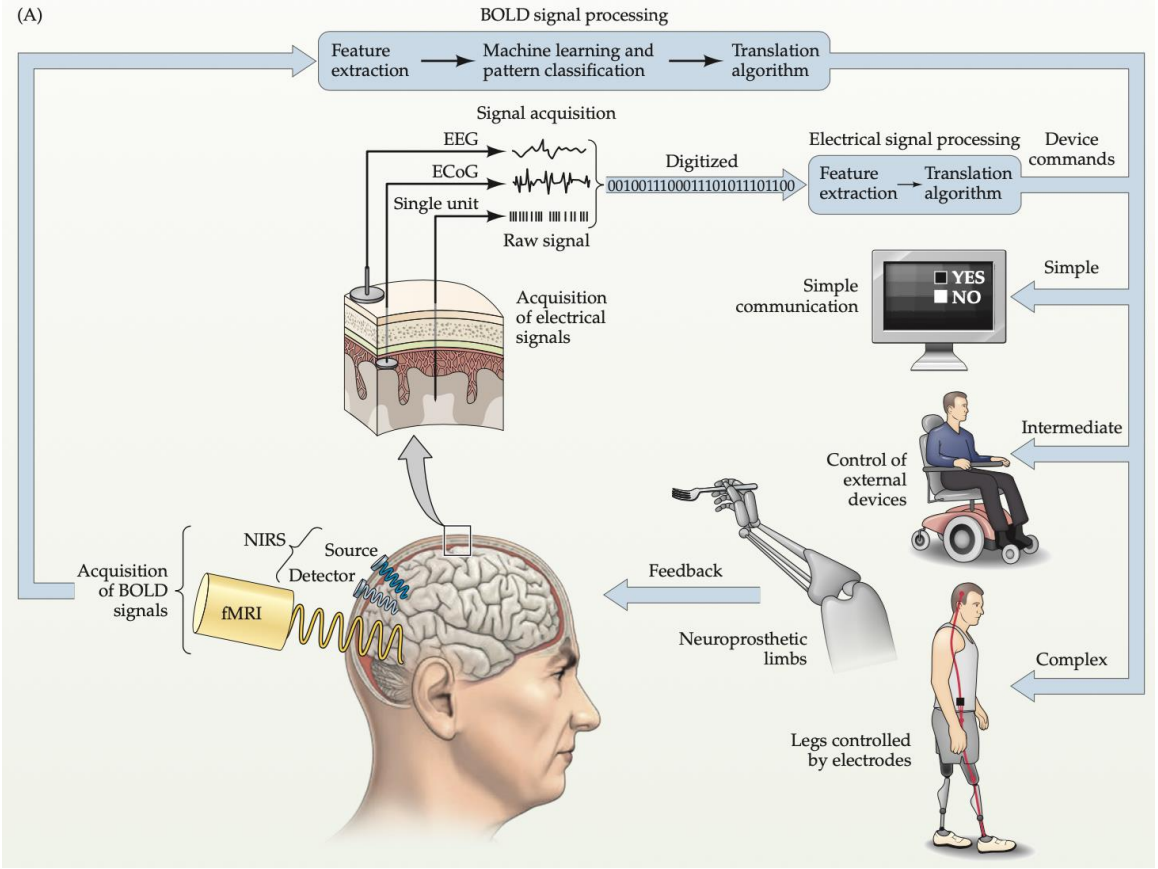
The topographic representations of movement in M1 are organized around **ethologically relevant categories of motor behavior.**

Brain-machine interfaces

«Devices that **record from the nervous system**, provide input directly to the nervous system or do both»

Use brain signals to control external objects

1. **Acquisition** of brain signals (*invasive/non-invasive*)
2. **Decoding** (*using ANNs*)
3. **Implement** signals
4. Generate sensory **feedback** to correct movement

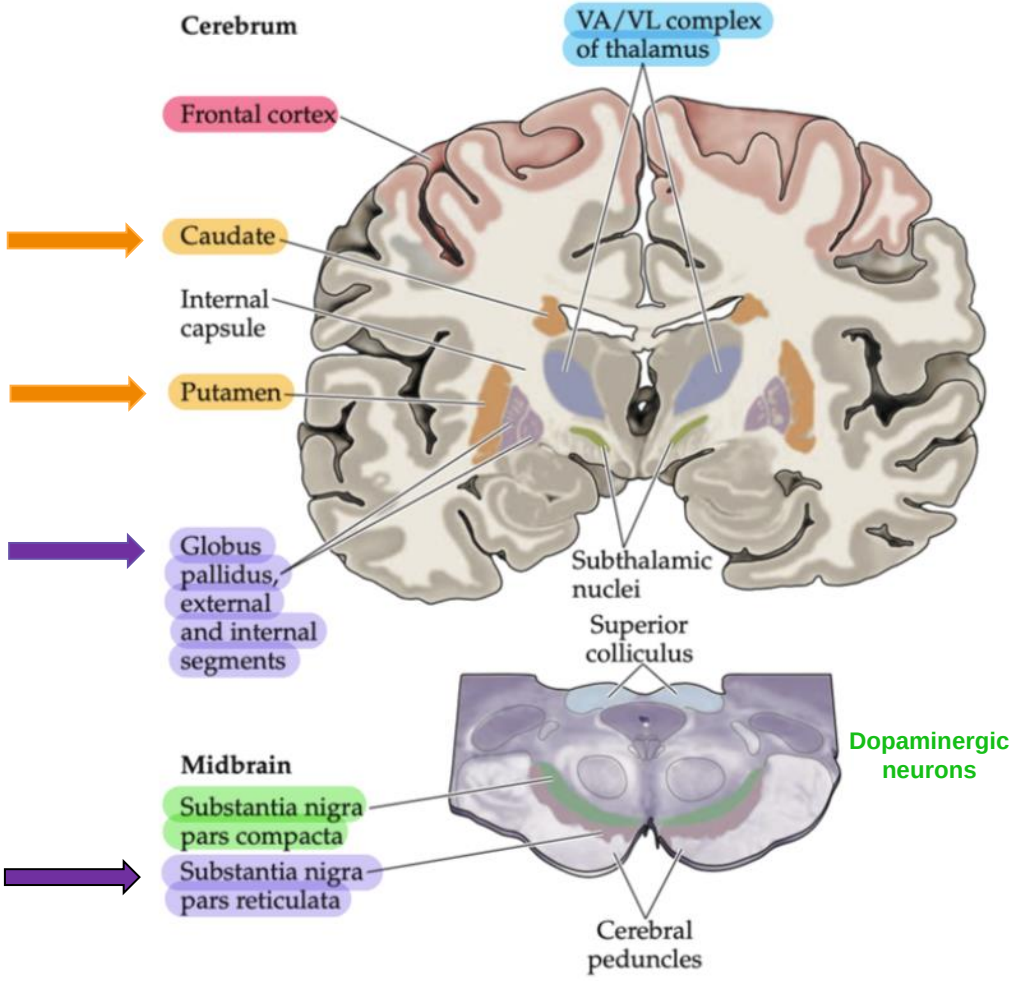


Basal ganglia (BG)

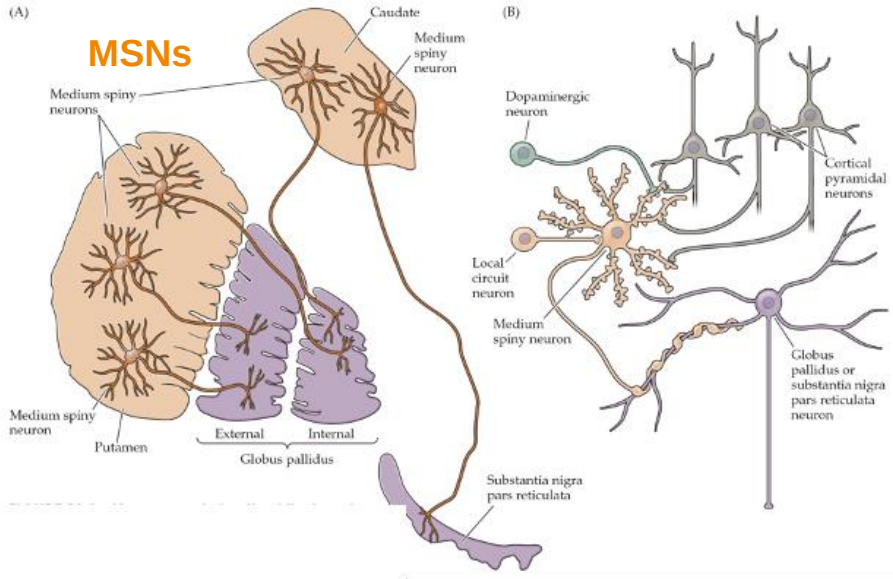
= striatum (caudate & putamen, input)
+ Gpe/Gpi (output)
+ SNpr (output)
+ SNpc
+ STN

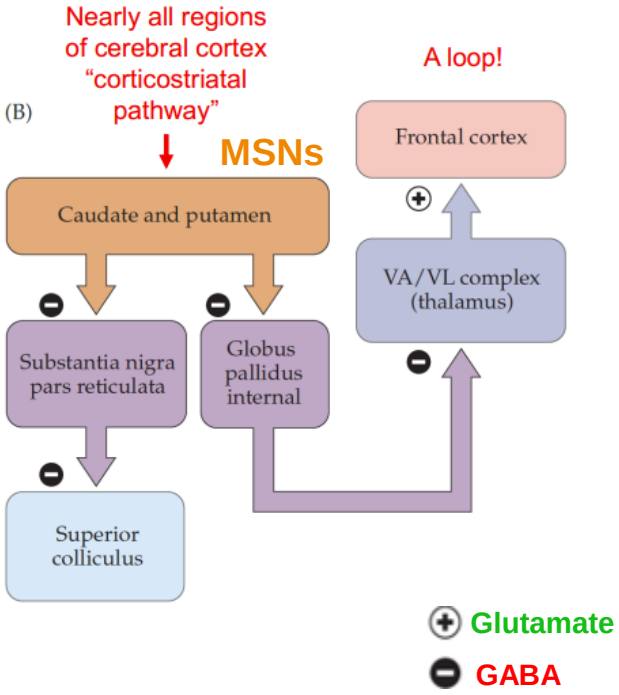
	From	To
Input	Sensory and associative cortices (*frontal/parietal lobe*)	Caudate + putamen = striatum
Output	Globus pallidus (GPe & GPi) Subtantia nigra pars reticulata (SNpr)	Motor cortex to drive movements

Neurons in the BG are **inhibitory** (*GABAergic*)



Connectivity of the basal ganglia

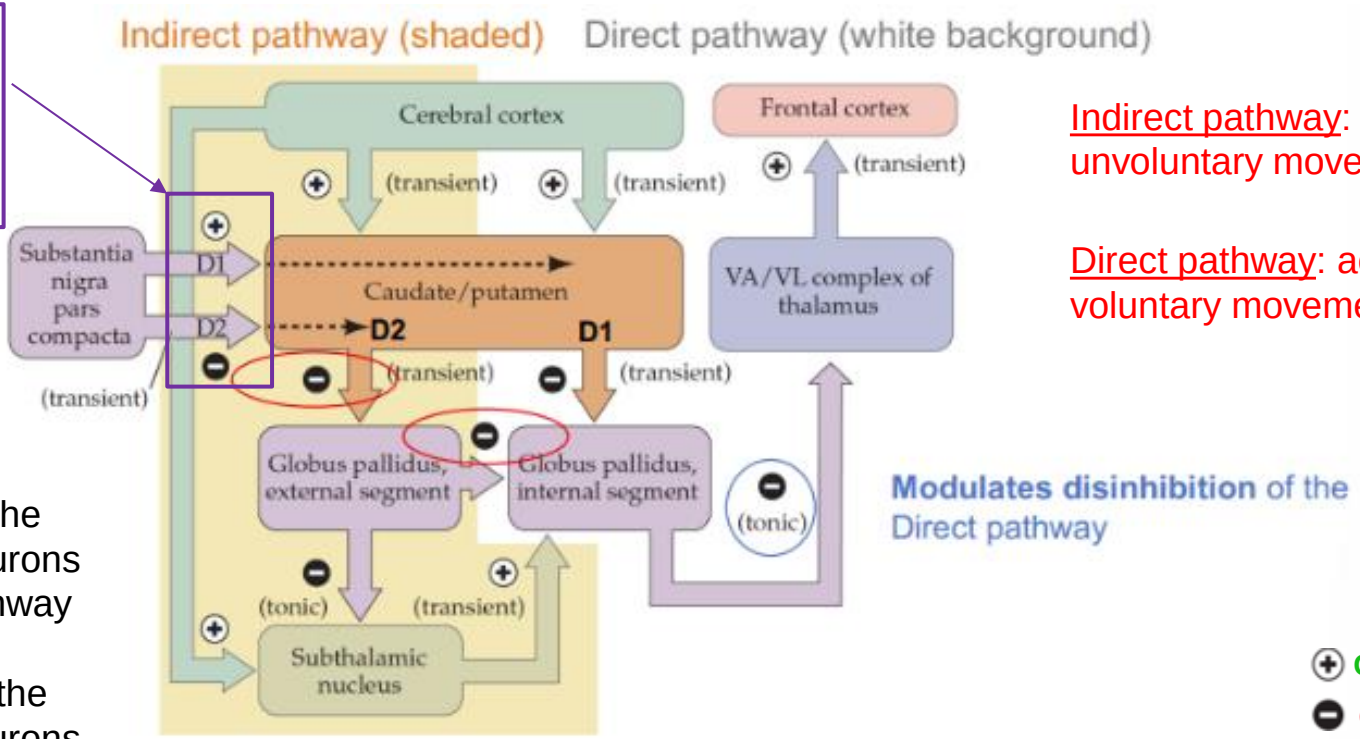




Direct and Indirect pathways

⊕ **Dopamine on D1 receptor**

⊖ **Dopamine on D2 receptor**



Indirect pathway: inhibits unvoluntary movements

Direct pathway: activates voluntary movements

D1 increases the excitability of neurons in the direct pathway

D2 decreases the excitability of neurons in the indirect pathway

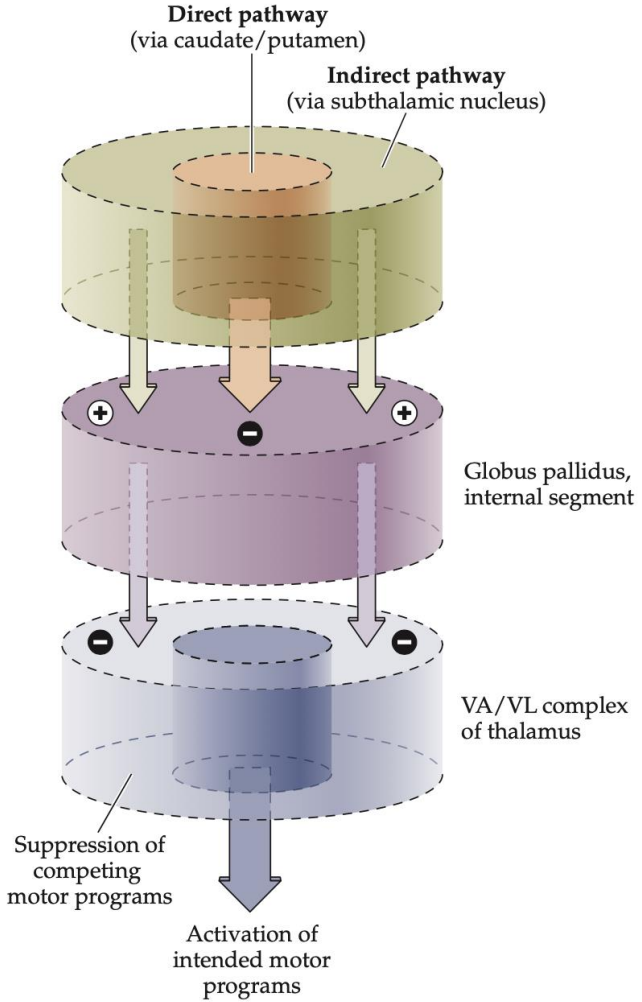
⊕ **Glutamate**

⊖ **GABA**

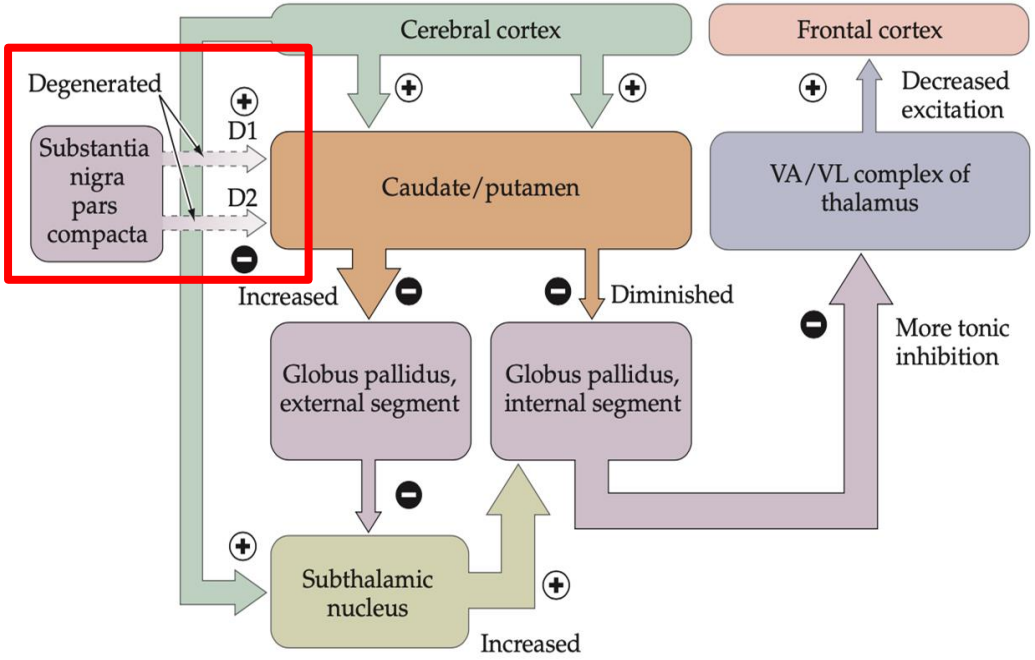
Modulation and action selection

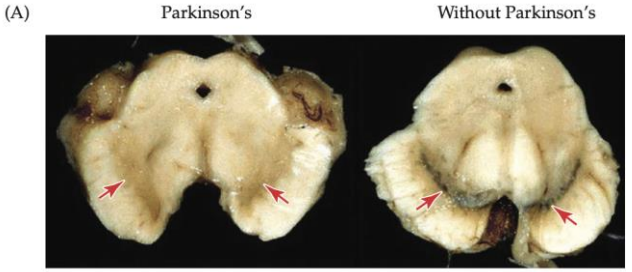
- **Direct** and **indirect** pathways compete
- **Center–surround functional organization**

- **Direct** pathway → activates / selects one behaviour
- **Indirect** pathway → suppresses all other behaviours



Parkinson's disease





Clinical features

- slow movements
- reduced facial expressions
- difficulties to initiate movements

Neuroanatomical observation

- Loss of pigmented SNpc dopamiergic neurons

Treatments

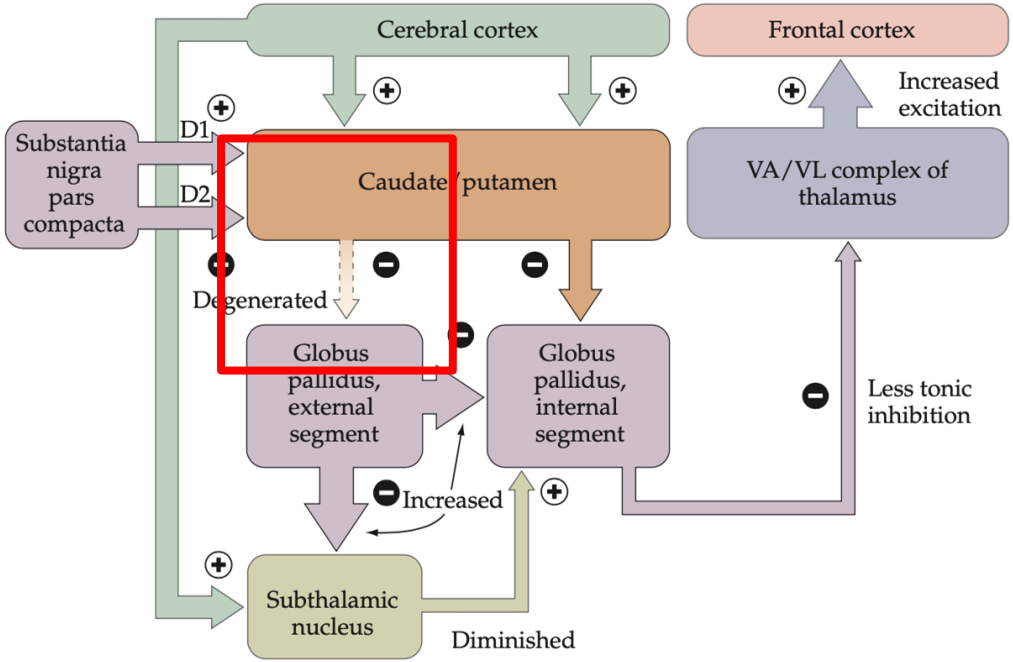
- Dopamine replacement therapies (e.g. L-DOPA)
- Deep brain stimulation of the subthalamic nucleus

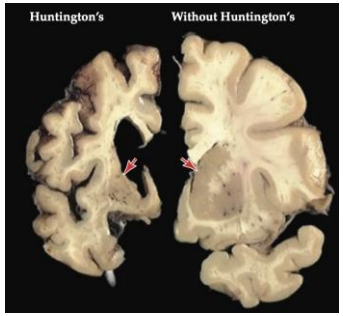
Hypokinetics

↓ activity of the thalamus/cortex

Video DBS before and after!!

Huntington's disease





Clinical features
- unwanted movements (chorea)
- muscle twitching (tics)

Neuroanatomical observation
- loss of striatal neurons of the indirect pathway

Treatments
- Antidopaminergic agents (*to decrease movement symptoms*)

Hyperkinetics
↑ activity of the thalamus/cortex

* A genetic disease (see here!)