PSYC301: Sensorimotor disorders

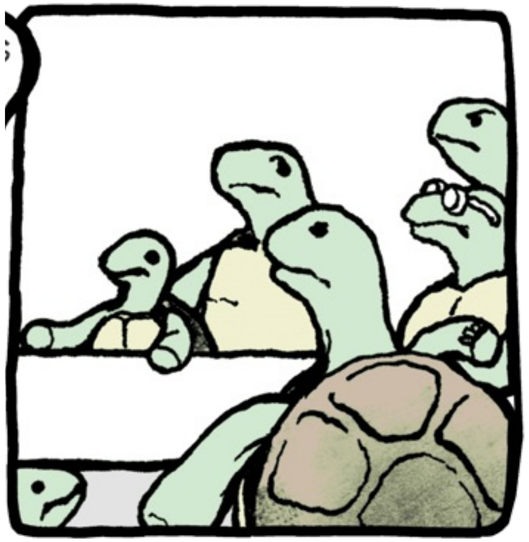
Jay Hosking, PhD

Lecture outline

Principles of sensorimotor organization

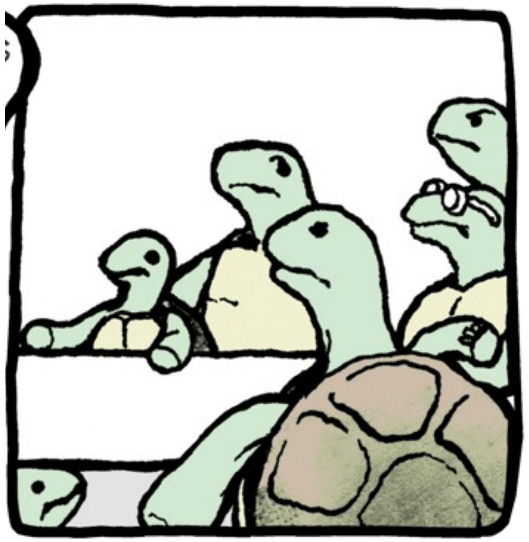
The standard motor hierarchy

Outside the standard motor hierarchy

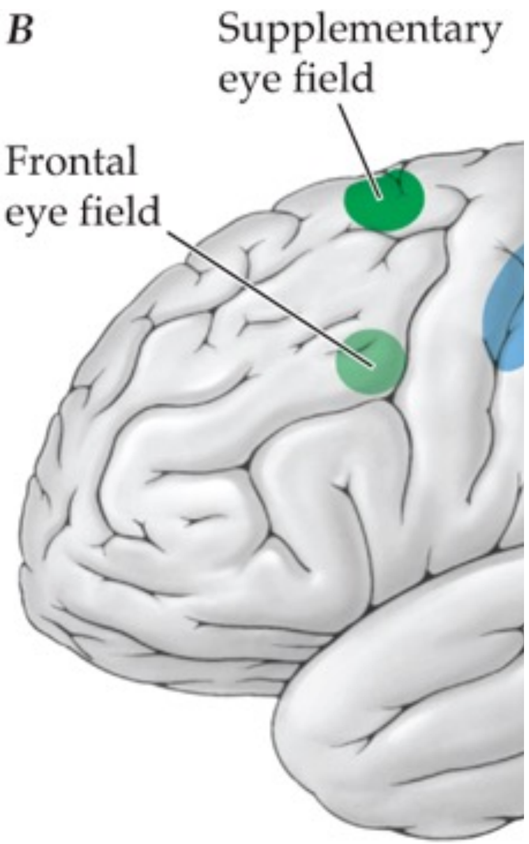


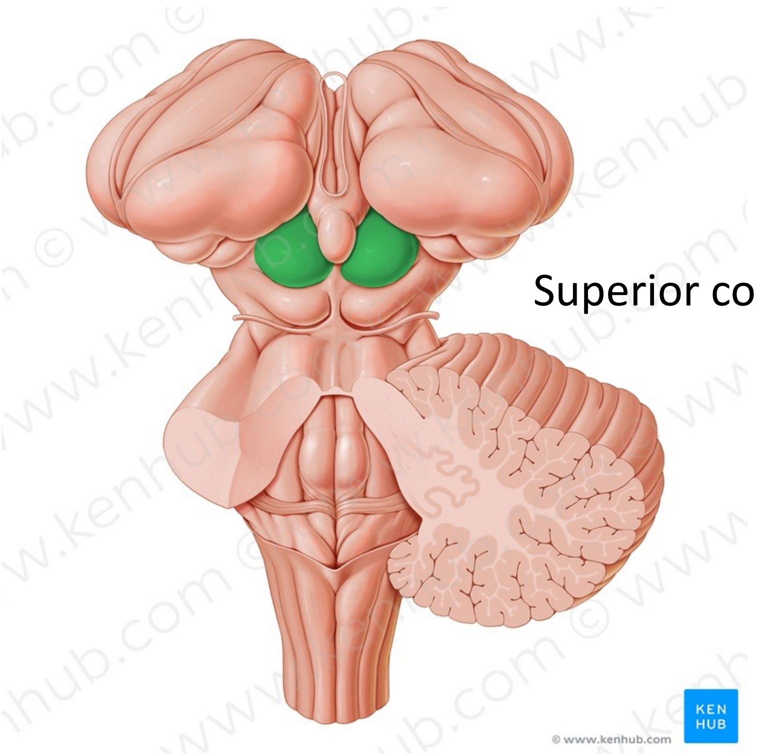
Learning objectives

1. Differentiate top-down vs. bottom-up processes in movement.
2. List and discuss the principles of sensorimotor function.
3. If we don’t need a brain to react to sensation and to elicit movement, then why have a brain at all?
4. Describe 2 major areas of sensorimotor association cortex and provide evidence of their functions. What happens when there is damage to the posterior parietal association cortex?
5. Describe the organization of the secondary and primary motor cortices and the current view of their functions. What happens when there is damage to the secondary or primary motor cortex?
6. Discuss the functions of the cerebellum. What happens when there is damage to the cerebellum?
7. Describe the two pathways of the basal ganglia. How do these pathways relate to Parkinson’s disease, Huntington’s disease, and psychostimulant use?
8. Read the associated paper by Buzsáki (2022). How does the inside-out perspective of the mind help us understand our motor control, and why we think of it as a “sensorimotor” system? What is corollary discharge, and what does it do? (This came up with the cerebellum, too.) What is meant by “perception is what we do”? And what does all this suggest about the famous science fiction “brain in a jar”, i.e. about potentially having sensation without any motor control?



Quick aside: different causes of movement



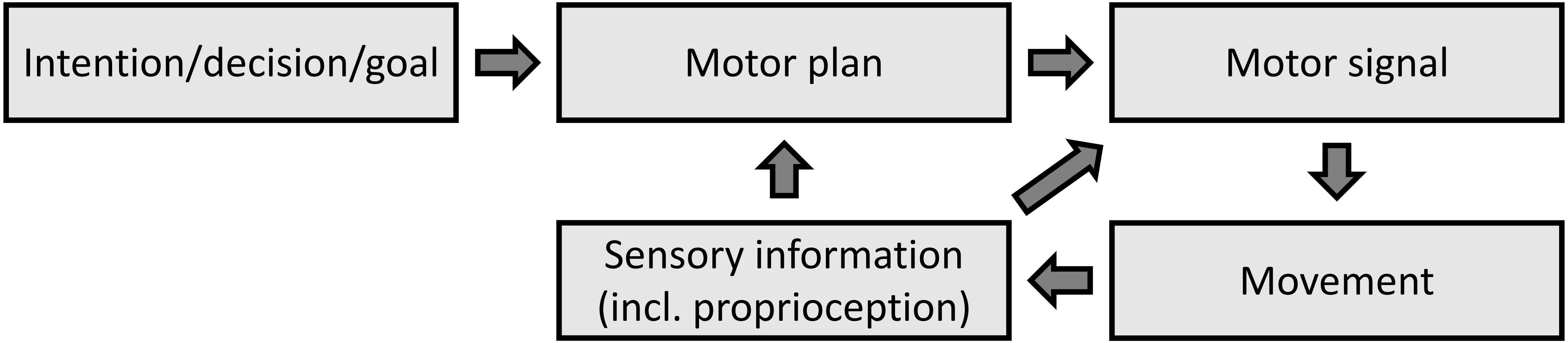


Superior colliculus (midbrain)

Top-down vs. Bottom-up

Theory

Movement: more than motor neurons



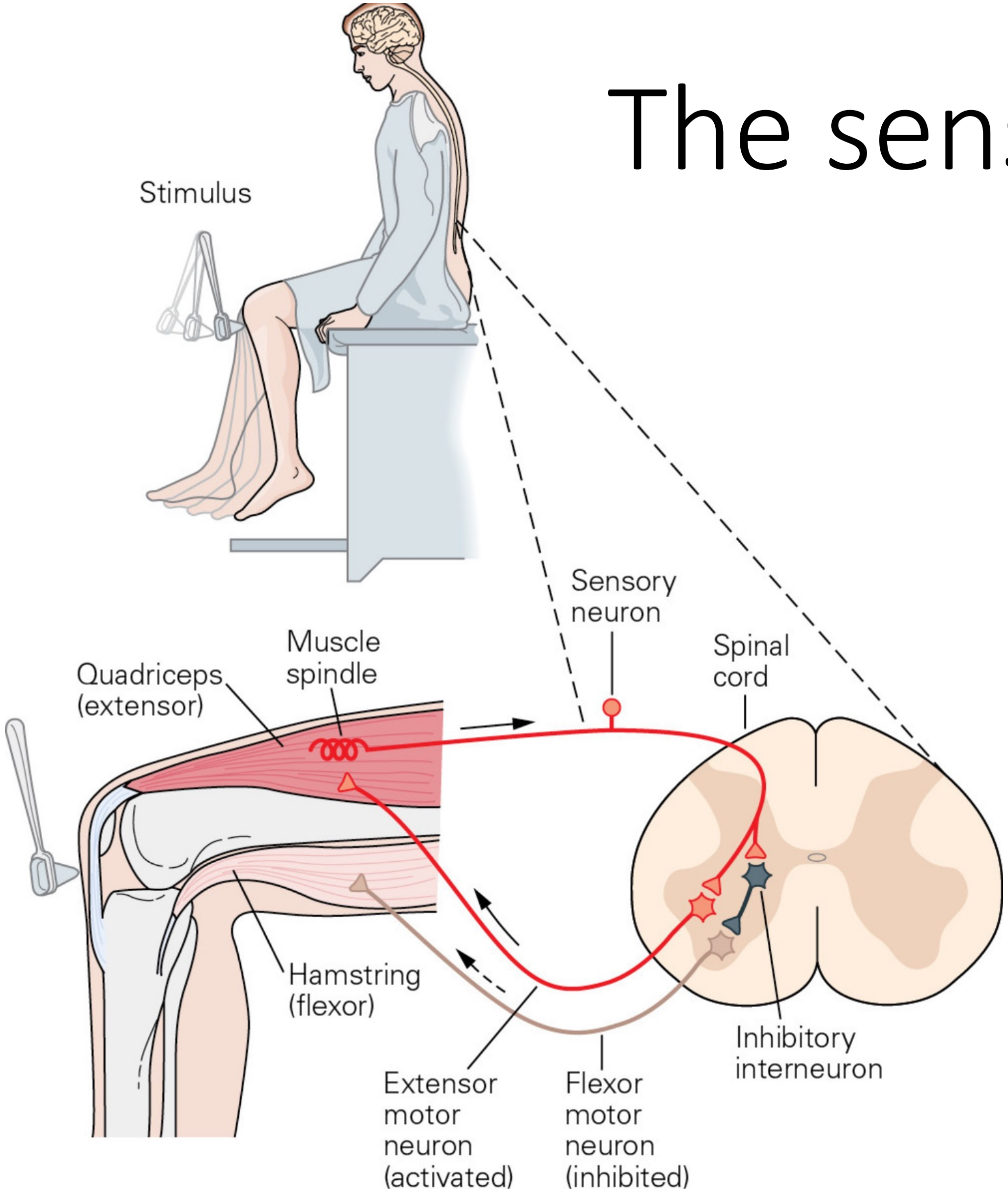
Case (study) in point: patient G.O. (Rothwell *et al*., 1982)

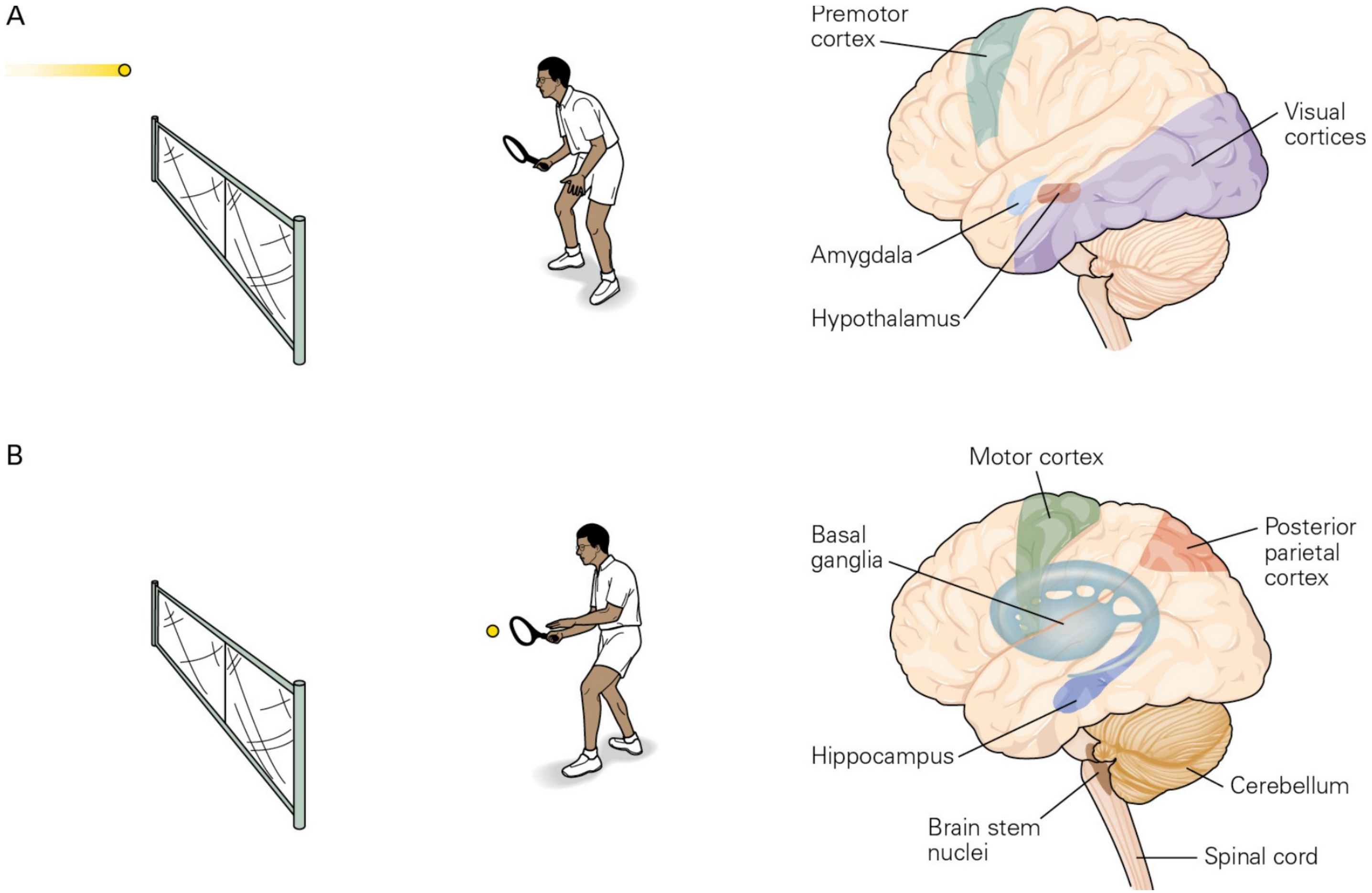
- Damage to somatosensory nerves of his arms
- Numerous problems with movement

Exception to this flowchart: ballistic responses

Theory

The sensorimotor system

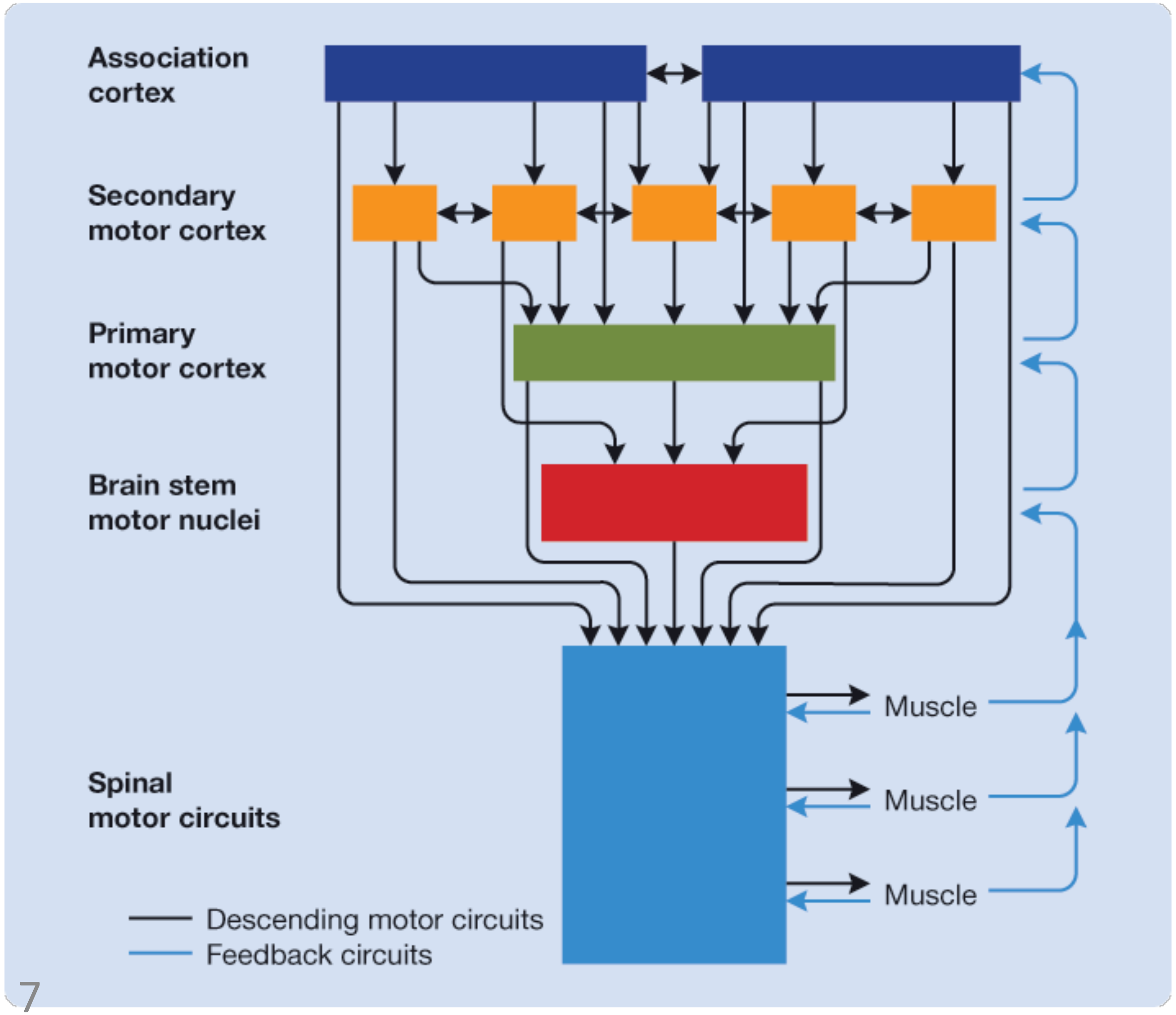


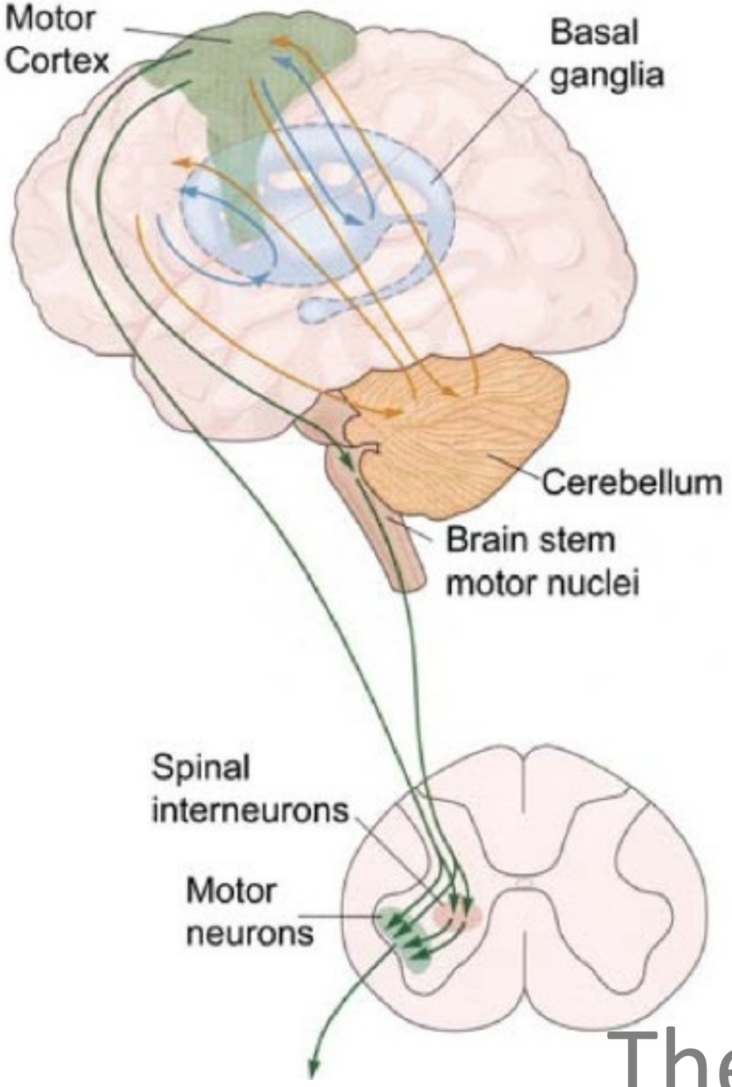


Sensation → movement → sensation → movement → ∞

Principles

Hierarchical control of movement





Theory

Hierarchical control of movement

Association cortex

Secondary motor cortex

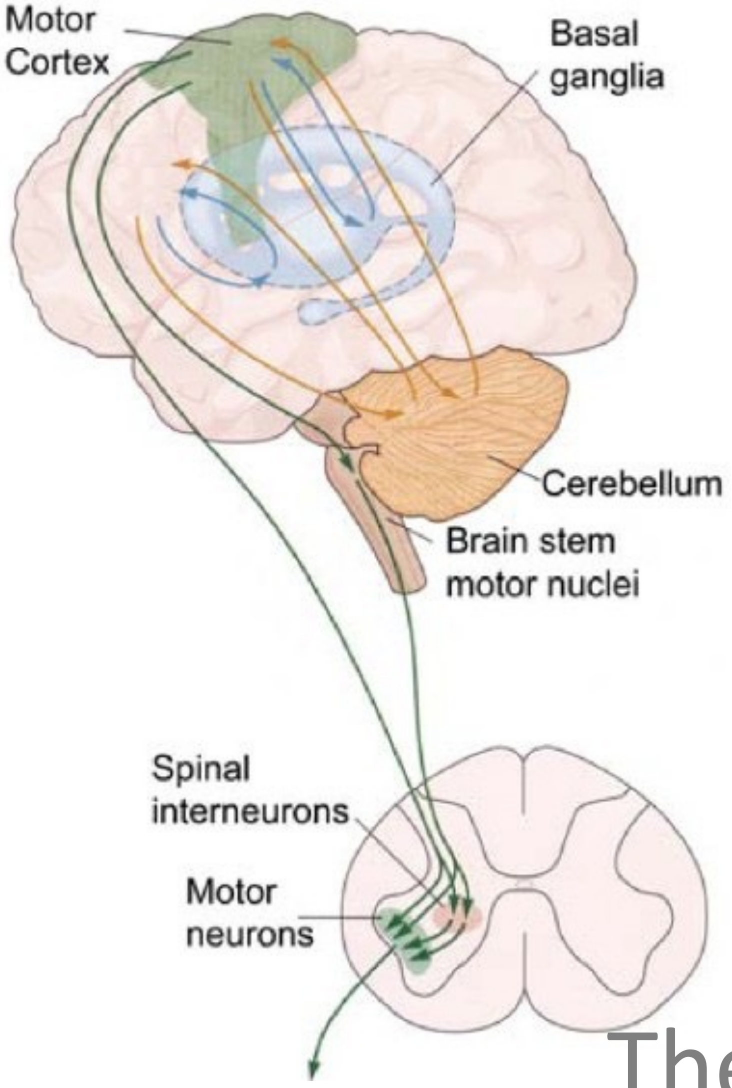
Primary motor cortex

Brainstem motor nuclei

Spinal motor circuits

Basal ganglia

Cerebellum

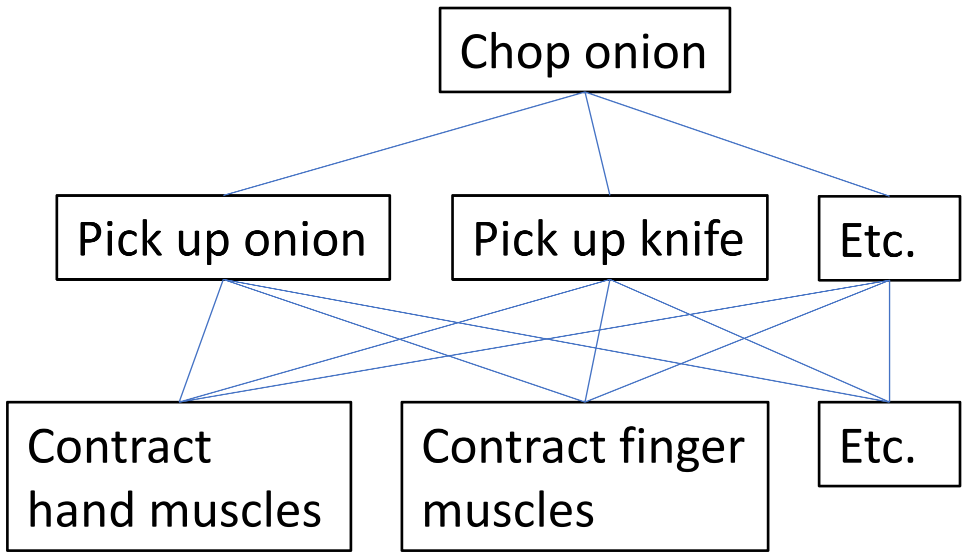


Theory

Central sensorimotor program theory

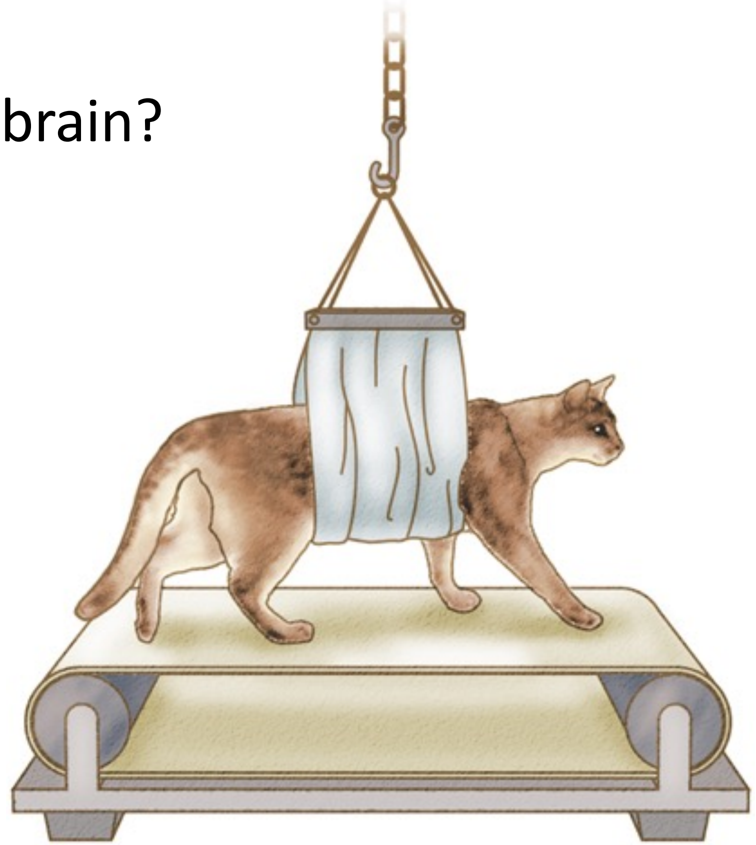
Three main assertions:

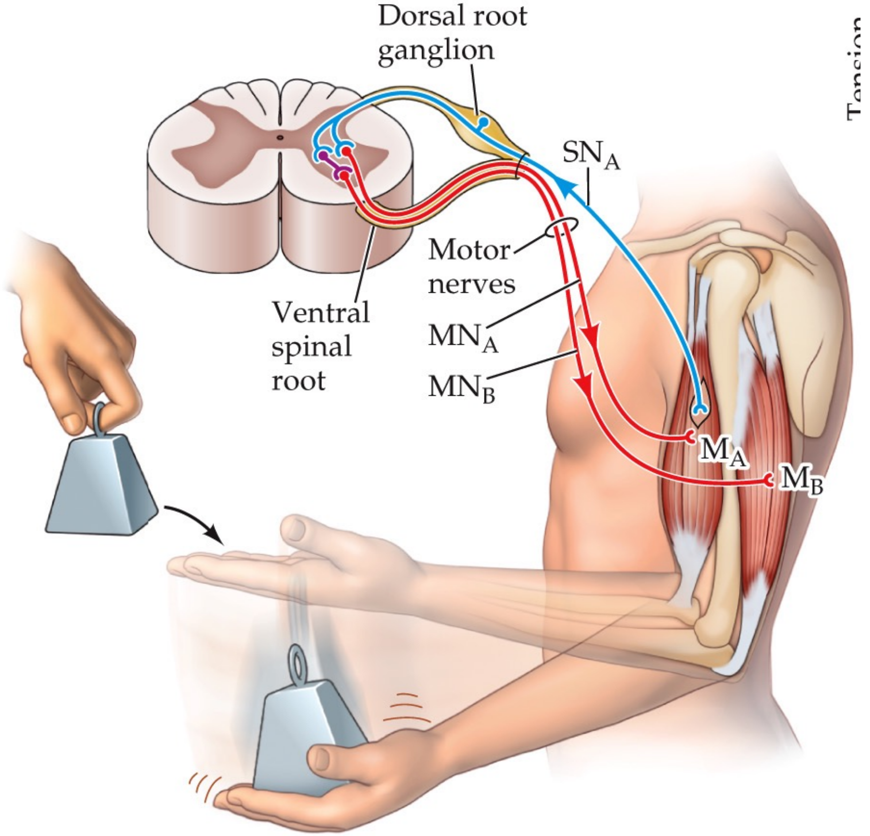
1. The lower levels of the sensorimotor system hierarchy possess “sensorimotor programs”, and those programs represent particular patterns of activity
2. A particular movement is produced by activating the appropriate combination of these sensorimotor programs
3. Once a particular level of the sensorimotor hierarchy is activated, it is capable of operating on the basis of sensory feedback without direct control by the higher levels



We don't need a brain to move

- Reflexes, e.g. stepping responses, limb approach or limb withdrawal to tactile stimuli, stretch reflex
- So why have a brain?





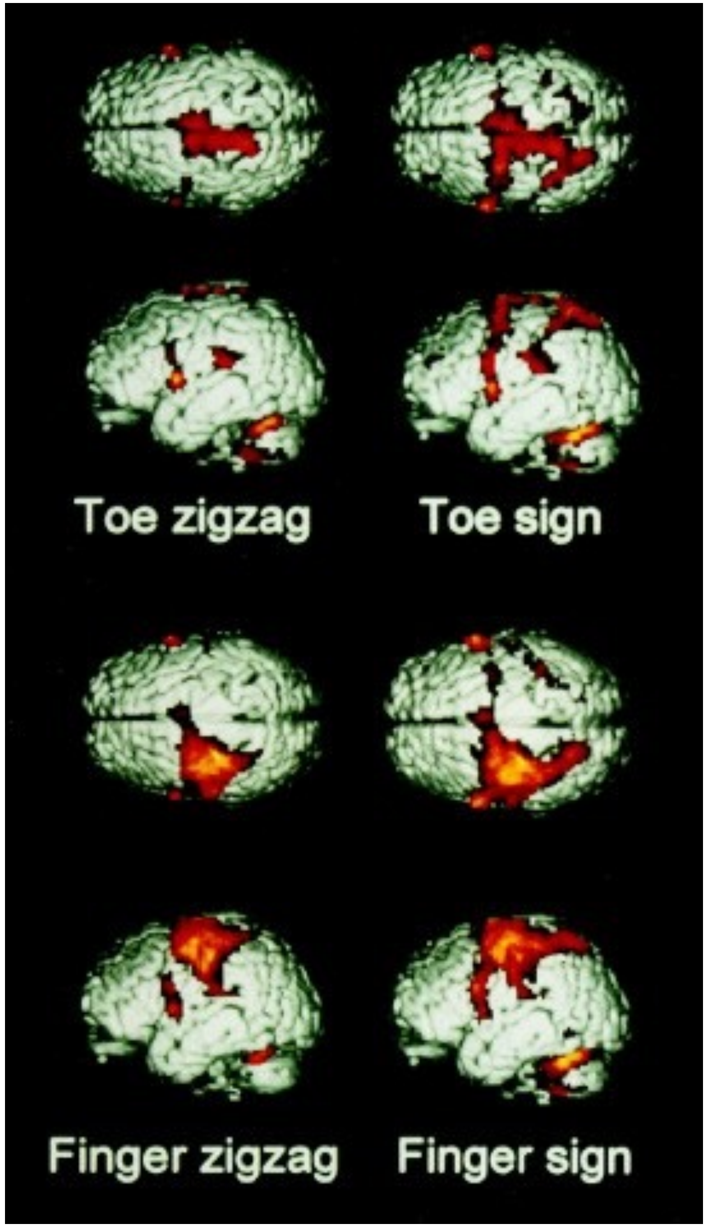
BEHAVIORAL NEUROSCIENCE 8e, Figure 11.11

Theory

Planning out movements

More than one way to carry out a movement
e.g. signing your name with your toe

Motor equivalence



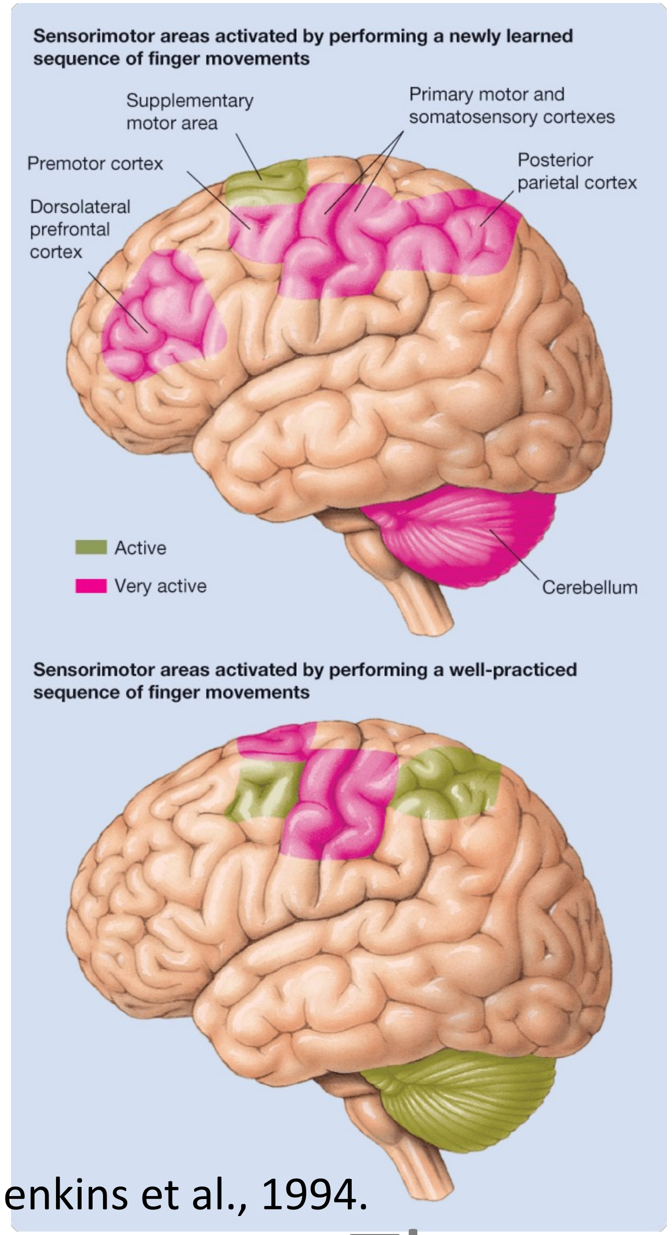
Rijntjes *et al*. 1999

Theory

Practice makes chunk-fect

Practice can create and/or modify sensorimotor programs. Most theories talk of two sorts of processes that influence the learning of sensorimotor programs:

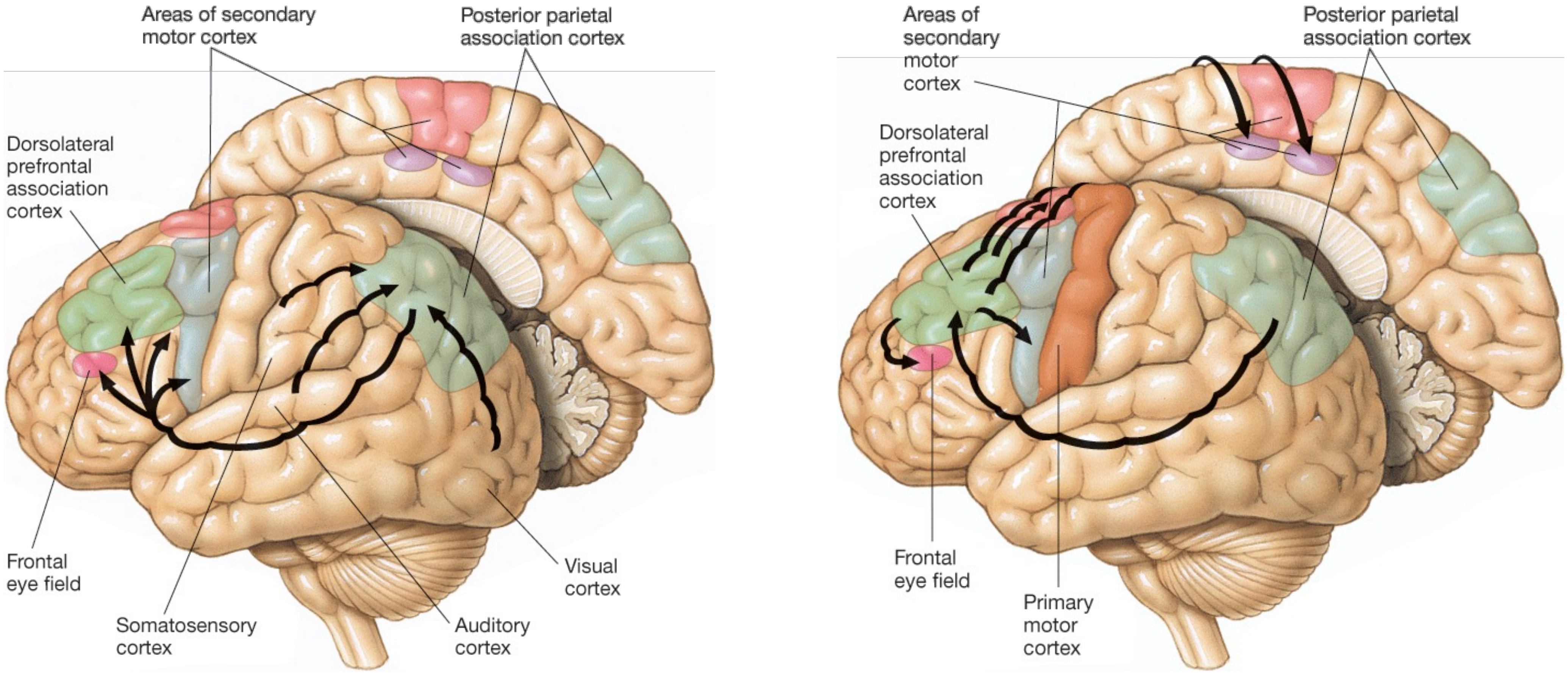
- Response chunking
- Shifting control to lower levels



Based on Jenkins et al., 1994.

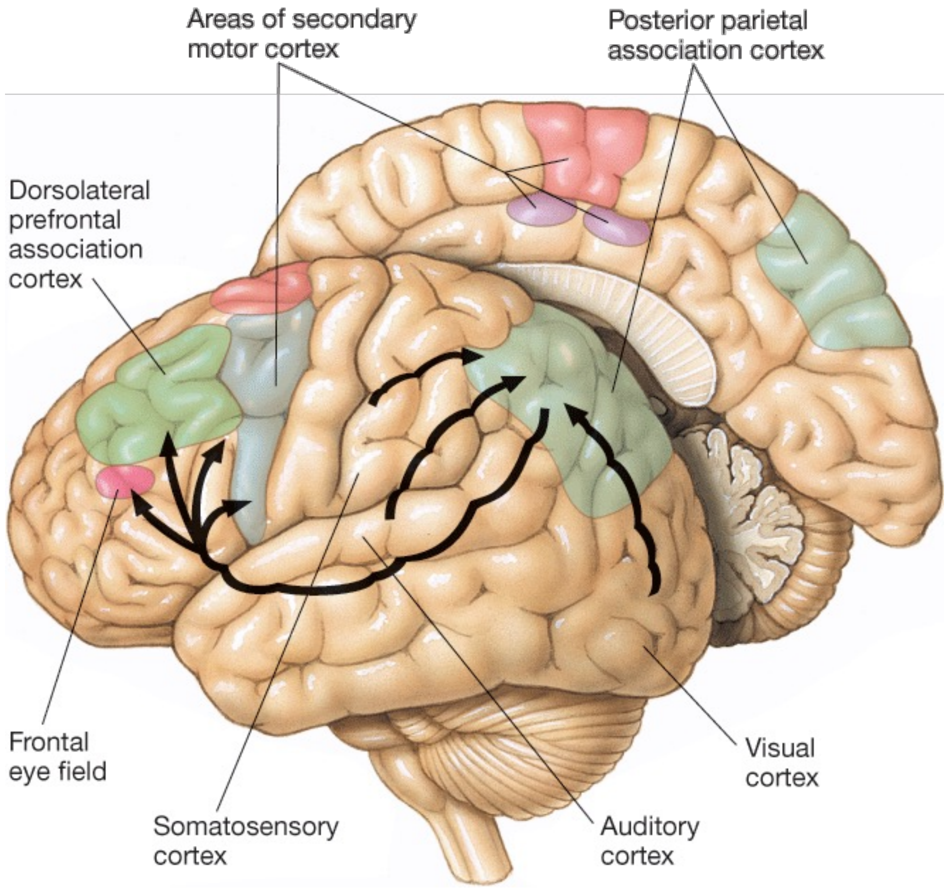
Theory

Sensorimotor association cortex



The standard hierarchy

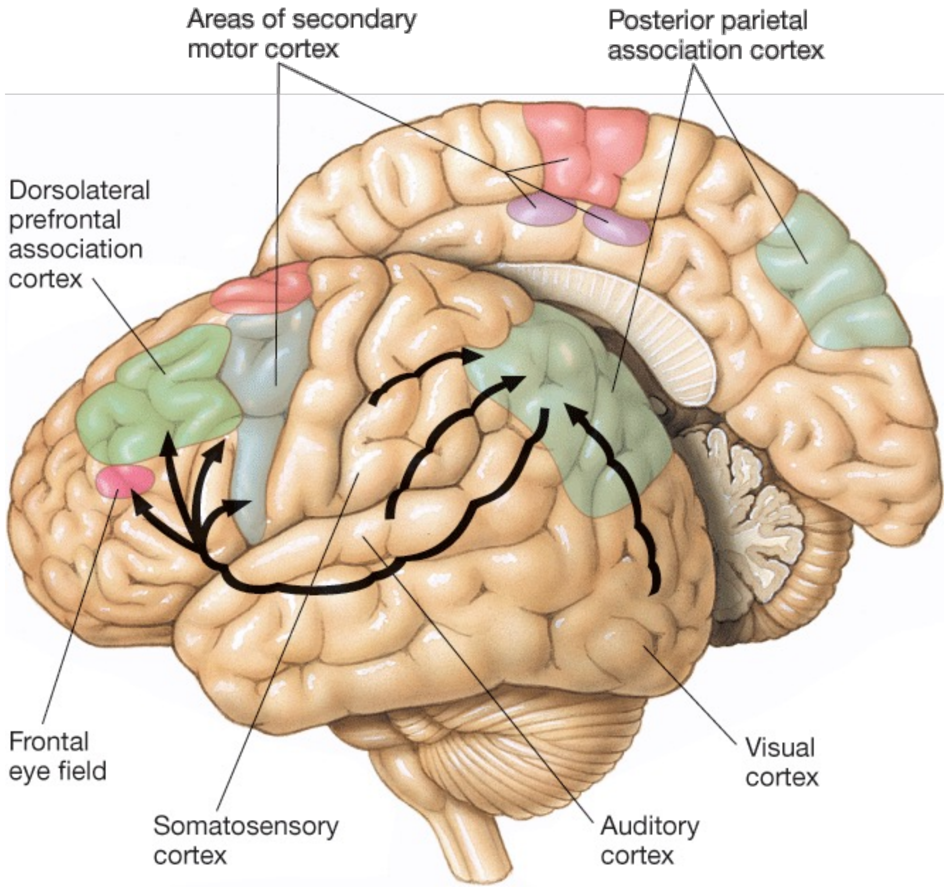
Posterior parietal cortex



- Provides information on where body parts are in relation to the external world
- Receives input from visual, auditory, and somatosensory systems
- Output goes to secondary motor cortex
- Stimulation of this area makes the subject feel they are performing an action

The standard hierarchy

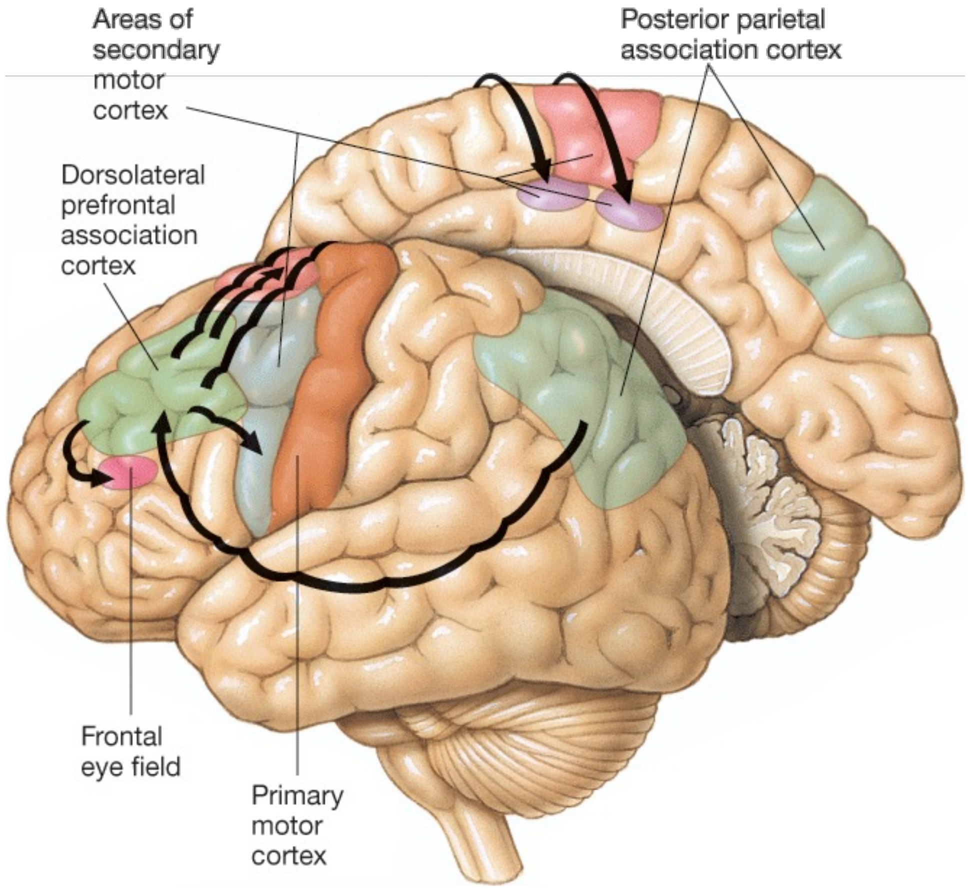
Posterior parietal cortex damage



- Apraxia (inability to perform movements on command)
  - Occurs when posterior parietal association cortex is lesioned
  - Associated with left hemisphere damage
  - Symptoms are bilateral
- Contralateral Neglect (fail to respond to visual, auditory, or somatosensory stimuli)
  - Produced by very large right parietal lesions
  - Individuals only attend to right side of body or items in environment
  - Individuals are capable of unconsciously sensing objects on the left

Dorsolateral prefrontal cortex

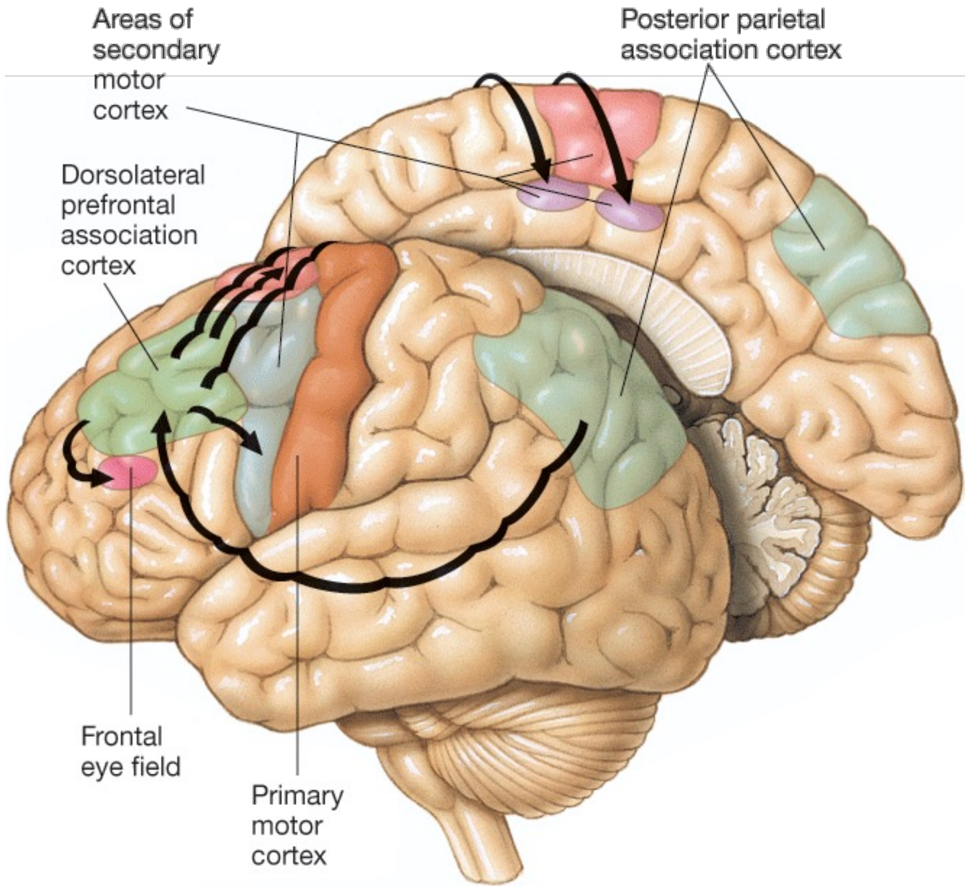
- Receives projections from posterior parietal cortex (and many other areas)
- Projects to secondary motor cortex, primary motor cortex, and frontal eye field
- Involved in assessments of external stimuli and our voluntary responses to them
- May work with posterior parietal cortex in decisions regarding voluntary response initiation



The standard hierarchy

Dorsolateral prefrontal cortex

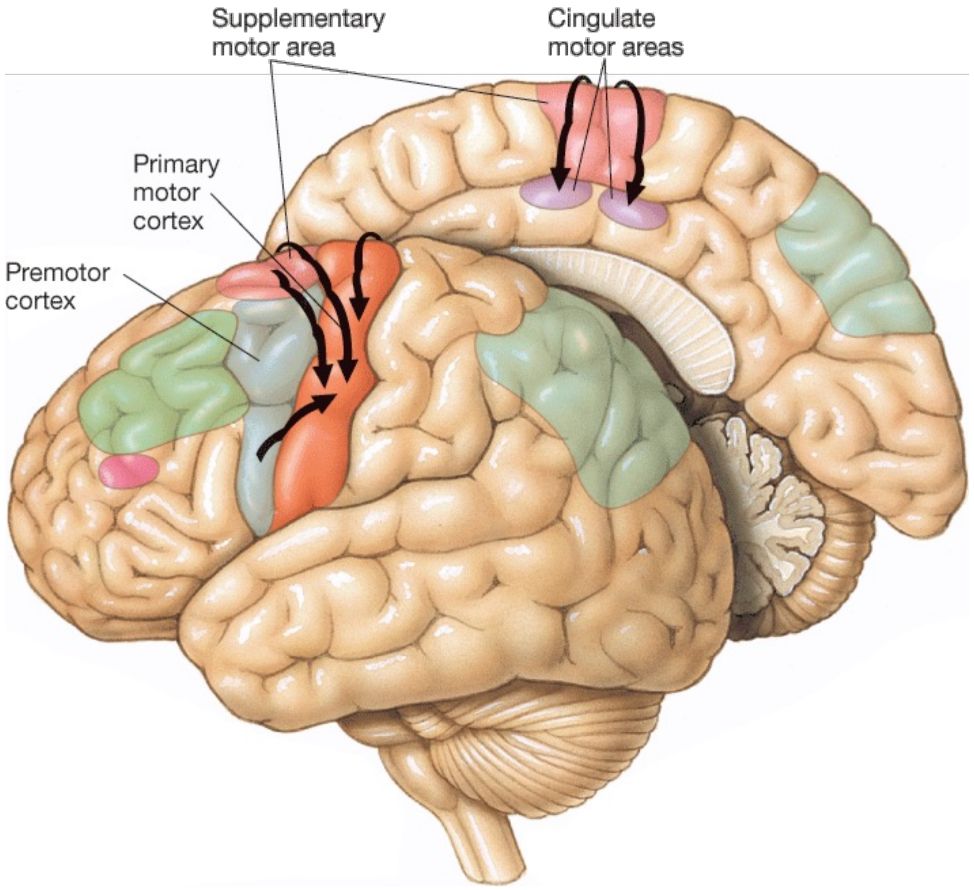
- dlPFC fires first in motor chain
- Decision making, voluntary movement
- But also critically involved in so many other functions (e.g. problem solving, math, working memory, learning, exec. functions)
- As such, damage here affects a number of sophisticated cognitive functions



The standard hierarchy

Secondary motor cortex

- Eight areas of secondary motor cortex
  - Two areas of premotor cortex
  - Three supplemental motor areas
  - Three cingulate motor areas
- Projects to primary motor cortex, each other, and brainstem
- Produce complex movements (before and during voluntary movements)
- Exact role of these areas is unclear
- SMA: planning, internally guided
- Premotor: externally guided
- Premotor areas encode spatial relations and program movements

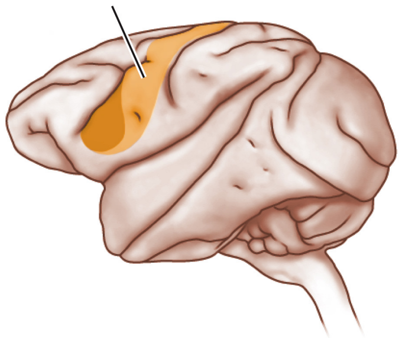


The standard hierarchy

Mirror Neurons

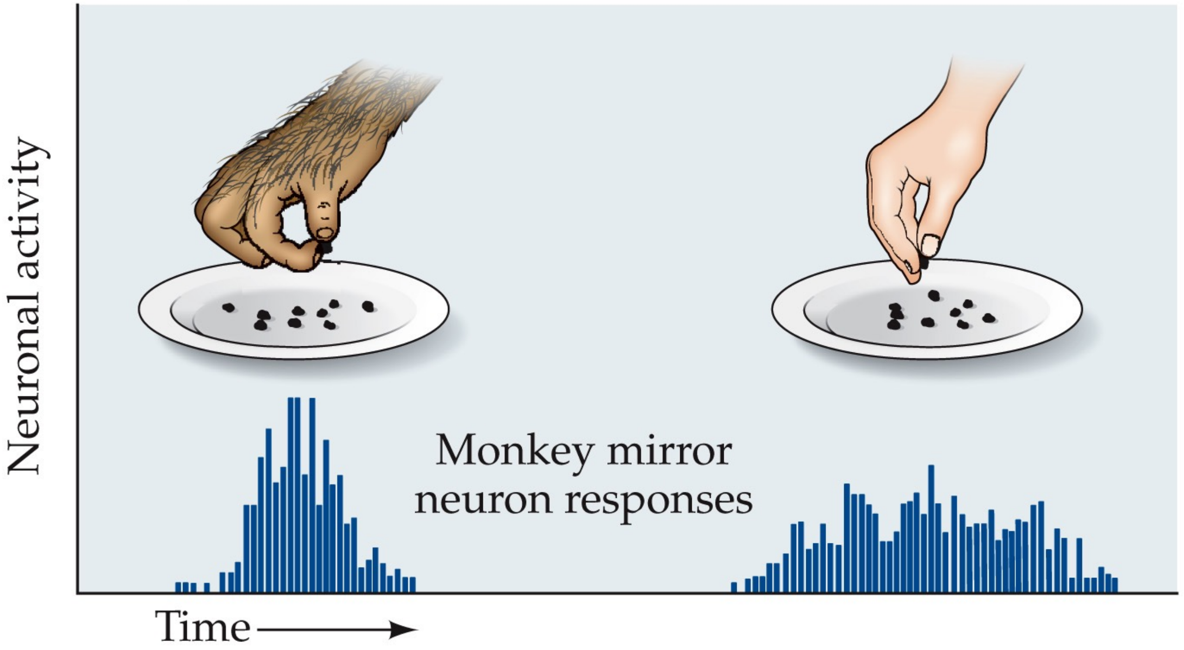
Likely the basis of mimicry, some learning
Perhaps related to theory of mind, empathy
Not just in the secondary motor cortex!
e.g. hippocampus (Mukamel *et al.* 2010)

(A)

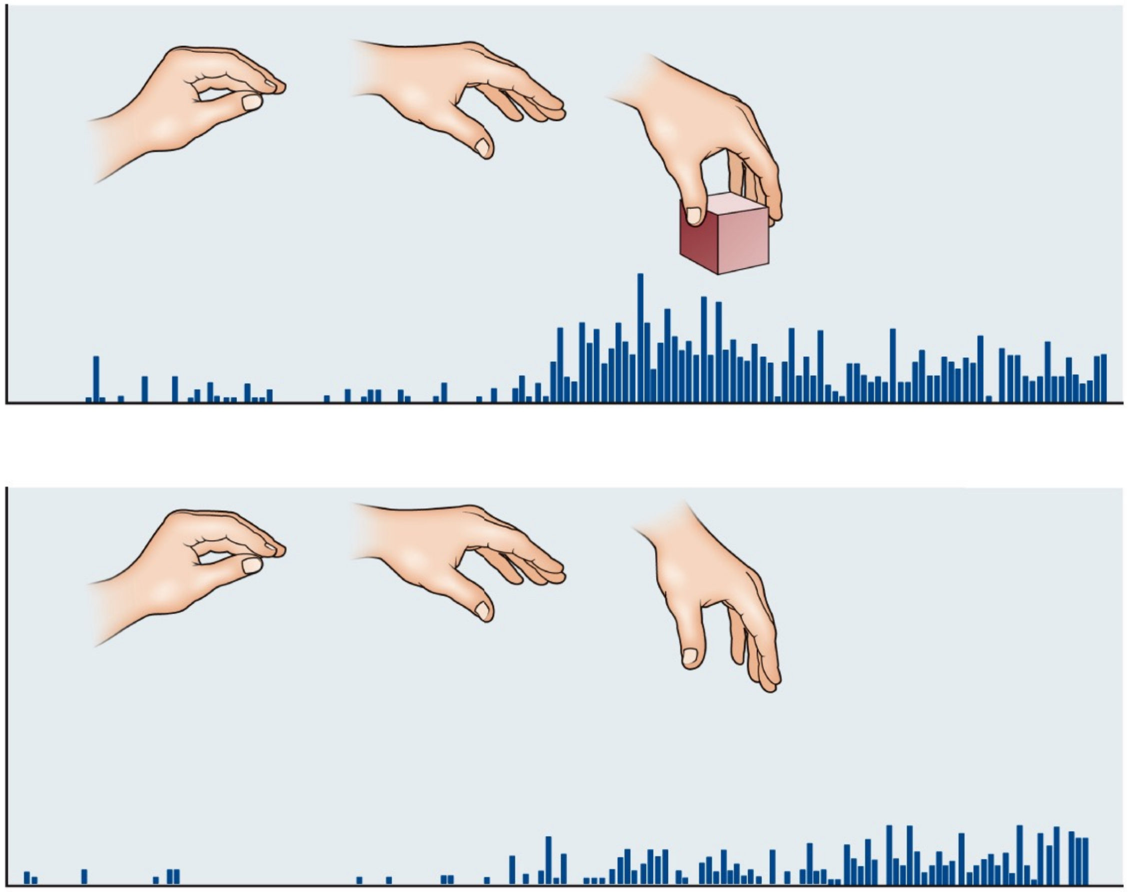


(B)

BEHAVIORAL NEUROSCIENCE 8e, Figure 11.19 (Part 1)



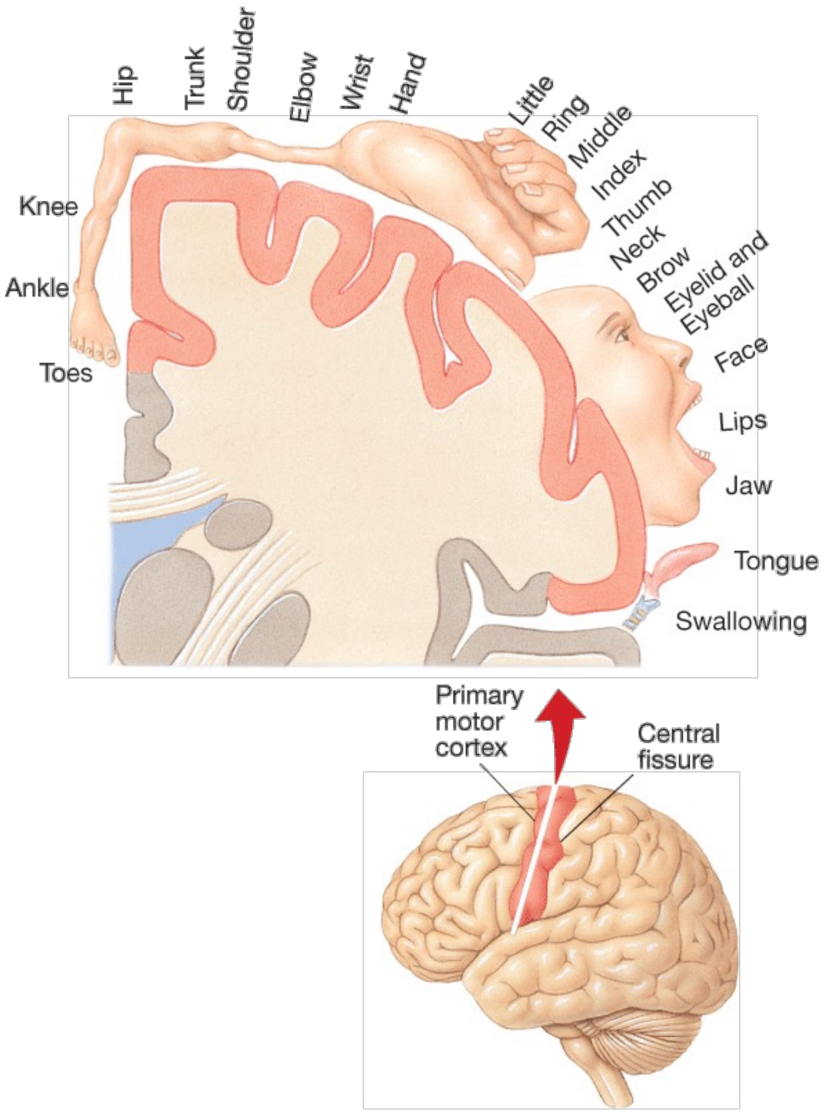
(C)



BEHAVIORAL NEUROSCIENCE 8e, Figure 11.19 (Part 3)

Primary motor cortex

- aka the precentral gyrus, M1
- Somatotopic organization: homunculus (Penfield)
- Receives feedback from muscle and joints
- Neurons code for preferred direction, not muscles per se
- Damage is not as disruptive as you might think
  - Independent movement
  - Astereognosia
  - Reduced speed/ accuracy/force
- Suggests SMA/premotor control



The standard hierarchy

Is that all there is?

- The theoretical association → secondary → primary → brain stem & spinal hierarchy cannot account for many notable motor disorders
- Cerebellar disorders?
- Alcohol's effects?
- Parkinson's disease?
- Huntington's disease?
- Stereotypy and impulsivity in psychostimulant addiction?
- Obsessive-compulsive disorder?
- Tourette's syndrome?

Other key regions

The cerebellum

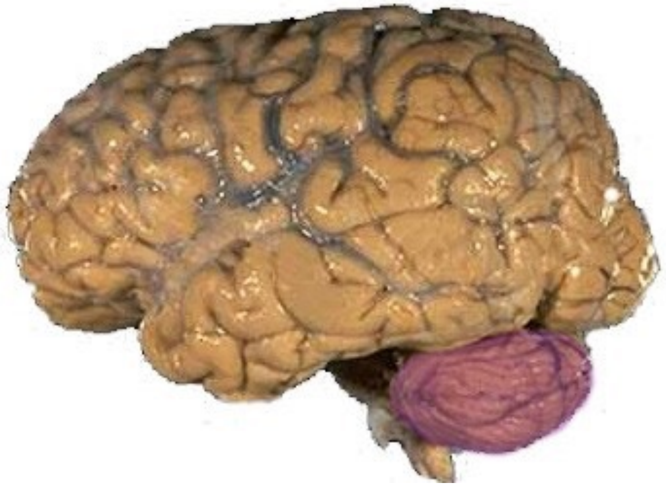
Receives inputs from:

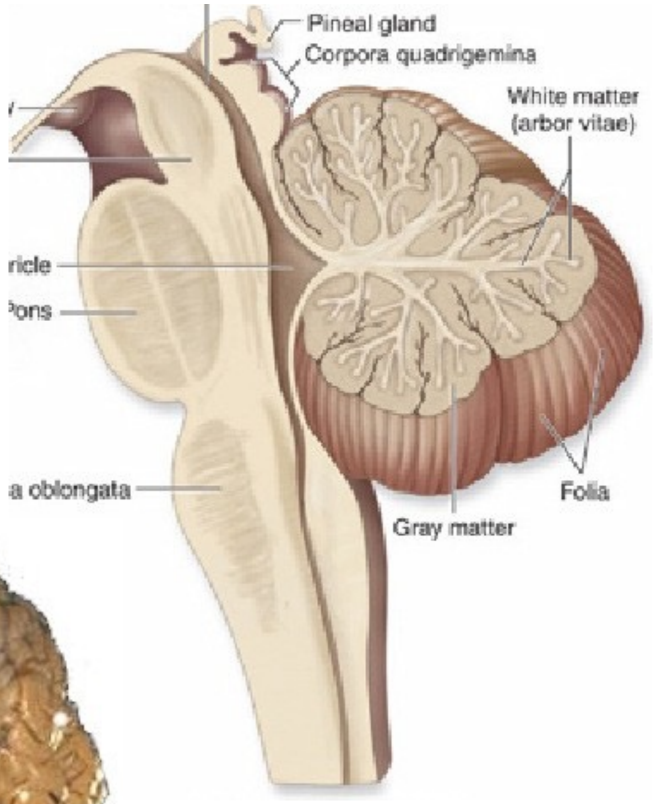
- Primary and secondary motor cortex
  - Corollary discharge
- Information about descending motor signals from the brain stem nuclei
- Sensory information, e.g. via the somatosensory and vestibular systems

In other words, it compares our intended movements to our actual movements, and then corrects our motor behaviour

It is also critical for timing and sequence (both motor and cognitive)

Note: ipsilateral control of body!



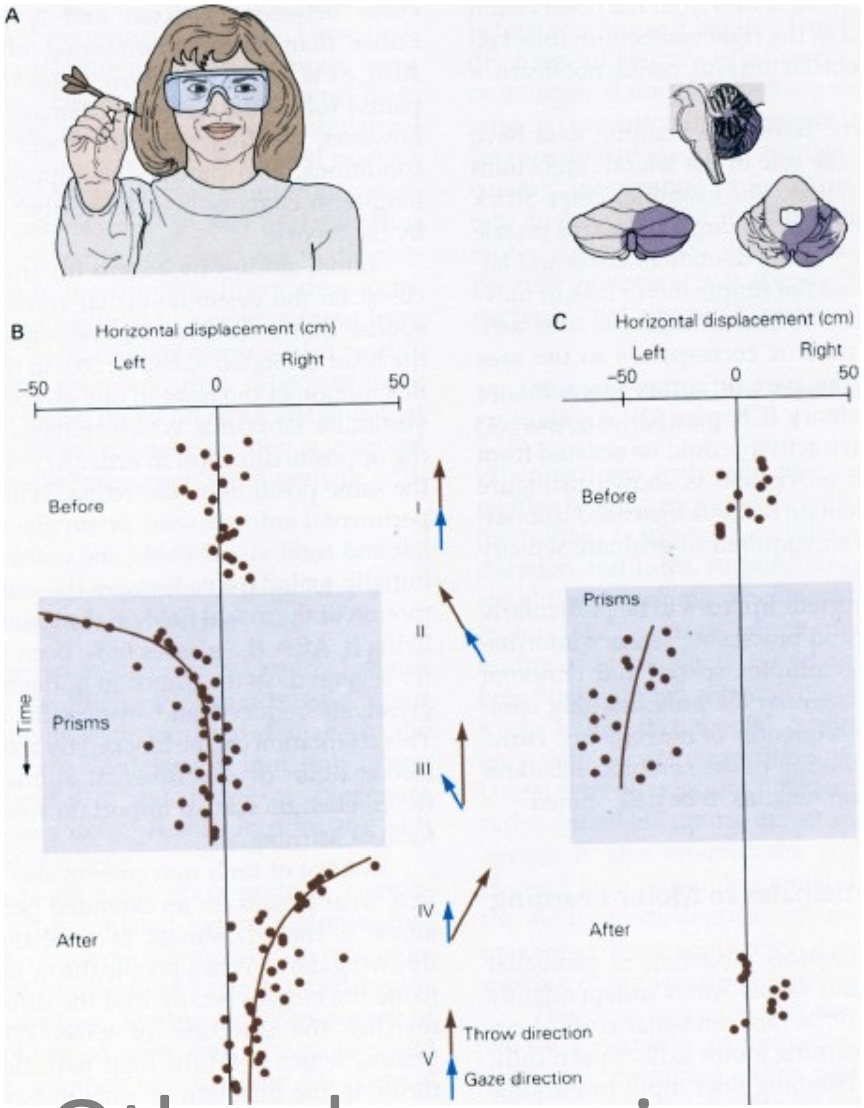


Other key regions

Damage to the cerebellum

Many possible effects:

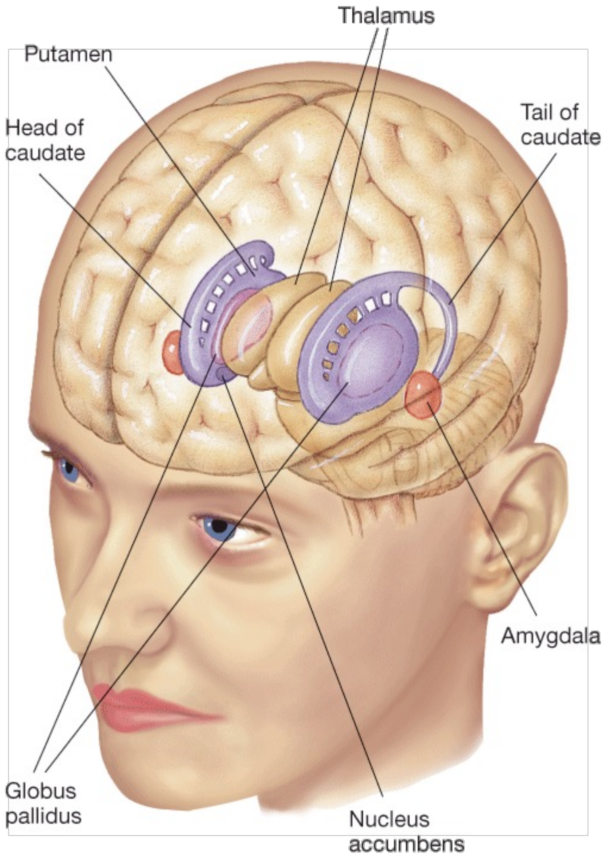
- loss of ability to precisely control the direction, force, velocity, and amplitude of movements
- loss of ability to adapt patterns of motor output to changing conditions
- difficulties in maintaining steady postures (e.g., standing)
- disturbances in balance, gait, and the control of eye movement
- impairments in the learning of new motor sequences
- impairments on measures of attention and executive control, procedural memory, working memory, language and visual-spatial processing (?????)

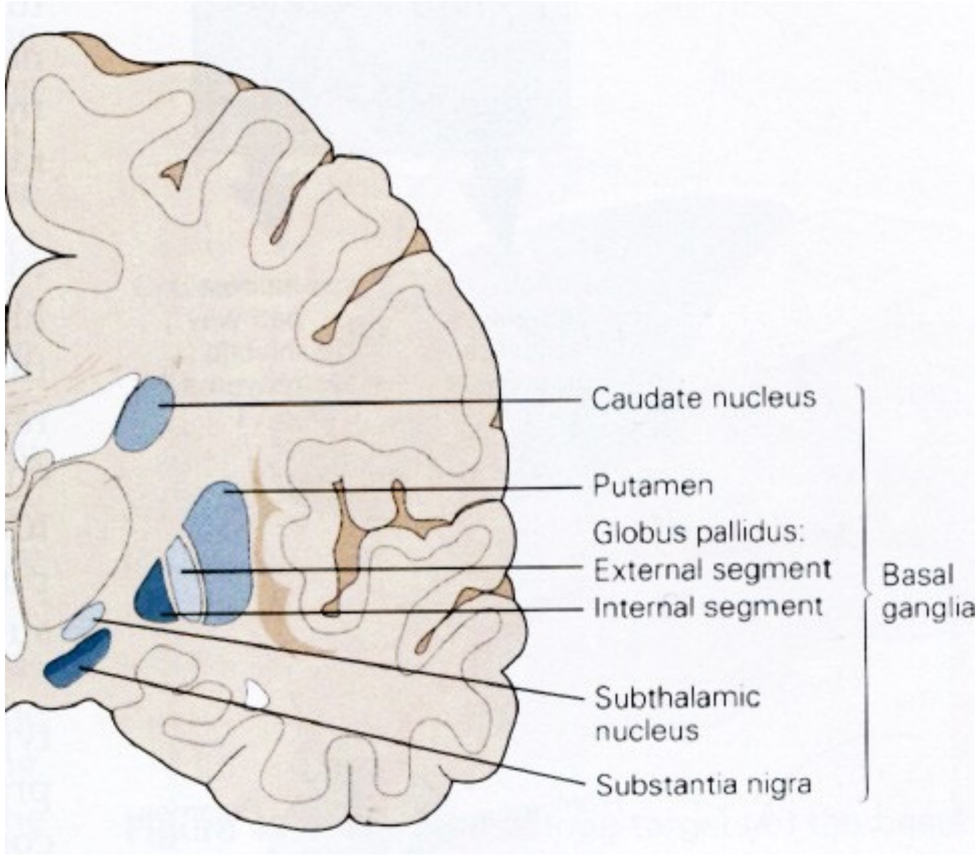


Other key regions

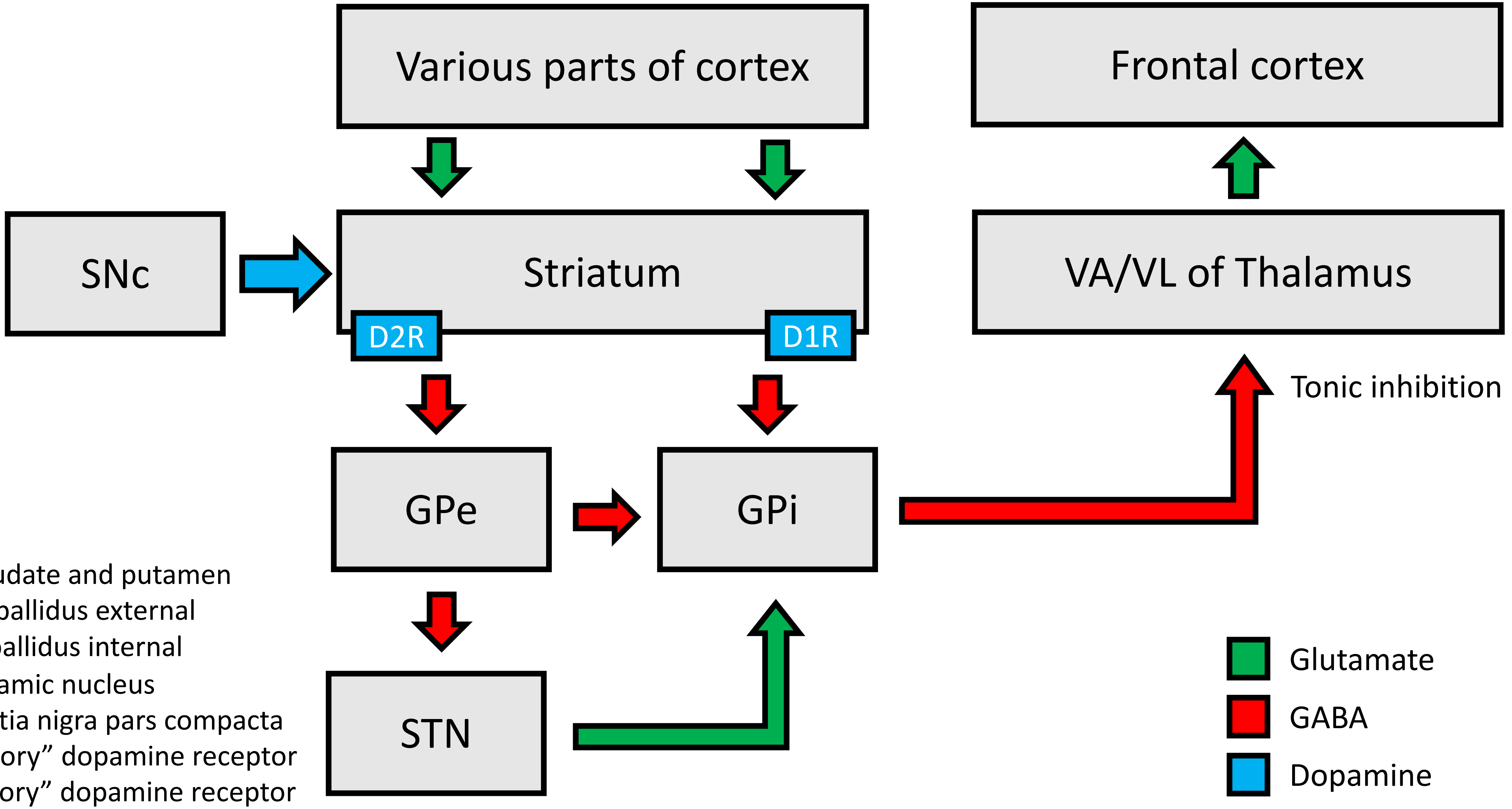
The basal ganglia

- Modulates motor output (classical view)
- Critical to habit formation
- Many cognitive roles
- Promotes skill learning



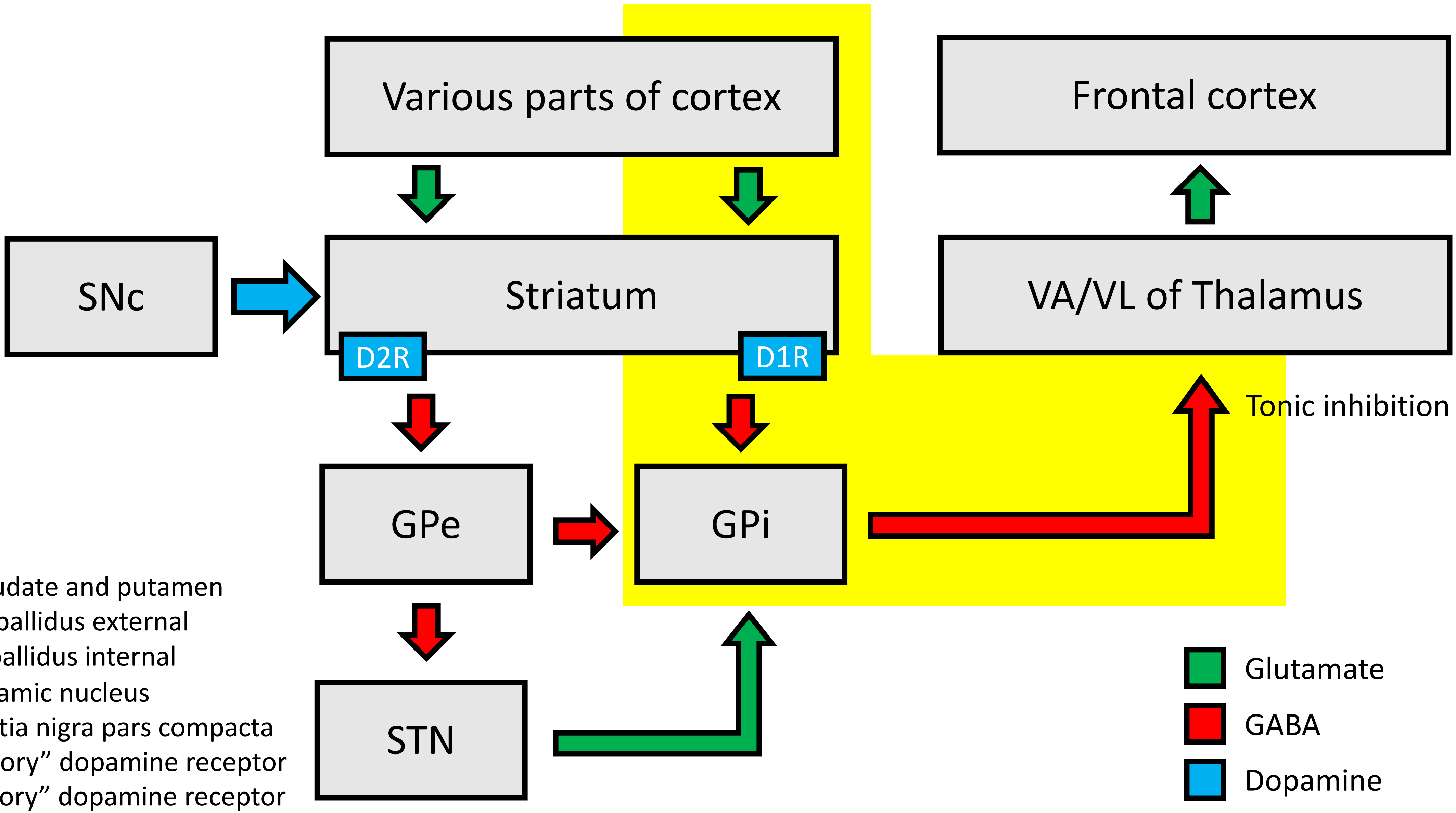


Two pathways of the basal ganglia



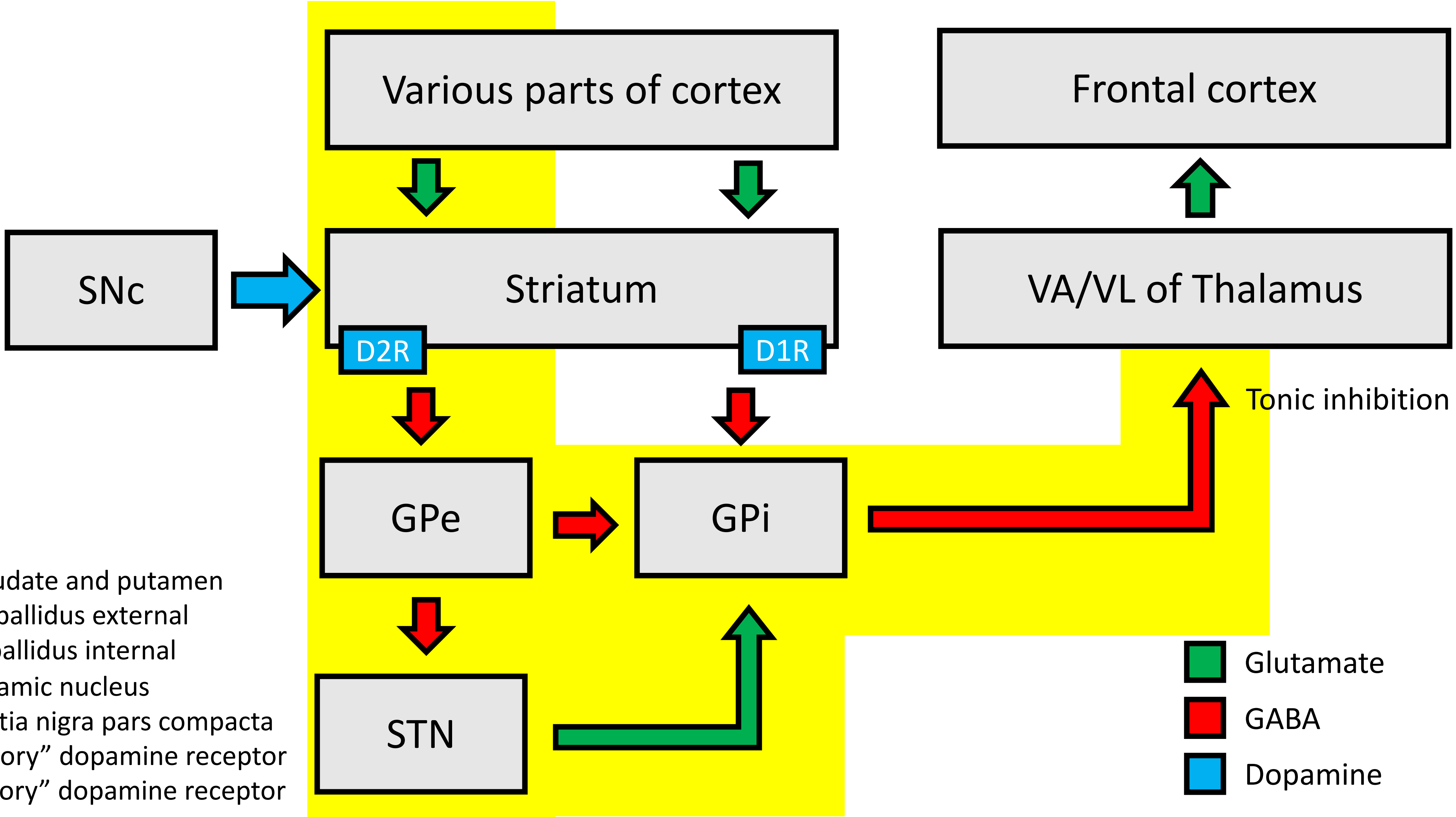
Striatum = caudate and putamen
GPe = globus pallidus external
GPi = globus pallidus internal
STN = subthalamic nucleus
SNc = substantia nigra pars compacta
D1R = “excitatory” dopamine receptor
D2R = “inhibitory” dopamine receptor

The direct pathway: "GO"



Striatum = caudate and putamen
GPe = globus pallidus external
GPi = globus pallidus internal
STN = subthalamic nucleus
SNc = substantia nigra pars compacta
D1R = "excitatory" dopamine receptor
D2R = "inhibitory" dopamine receptor

The indirect pathway: “STOP”

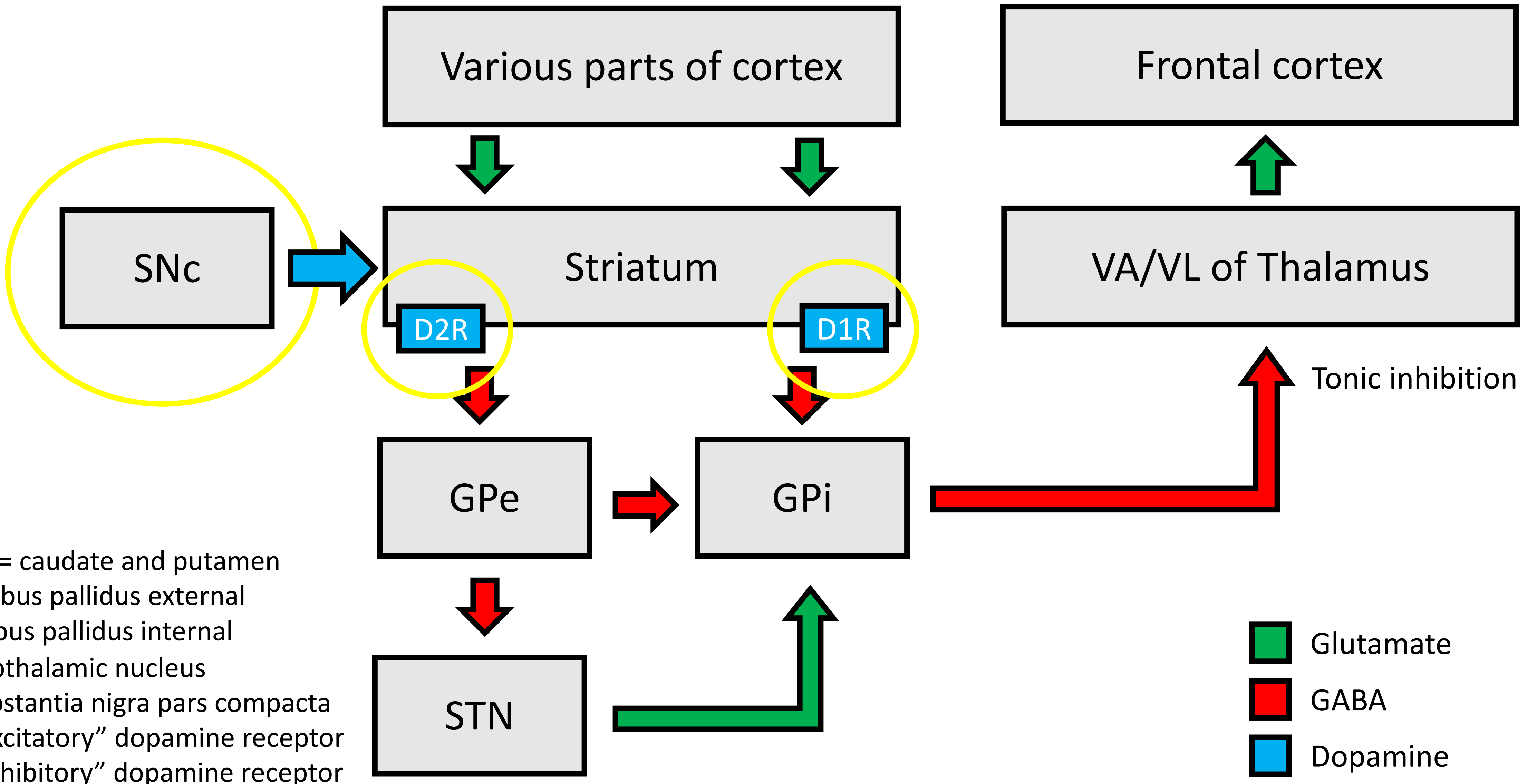


Striatum = caudate and putamen
GPe = globus pallidus external
GPi = globus pallidus internal
STN = subthalamic nucleus
SNc = substantia nigra pars compacta
D1R = “excitatory” dopamine receptor
D2R = “inhibitory” dopamine receptor

Two pathways of the basal ganglia

- Motor/premotor areas in frontal cortex are normally tonically inhibited
  - Result: no movement
- To activate motor cortex, we need to inhibit the inhibition: this is called **disinhibition**
  - Result: movement
- **Direct/“GO” pathway:** facilitates disinhibition of frontal cortex
- **Indirect/“STOP” pathway:** maintains the tonic inhibition of frontal cortex
- Whether or not movement occurs depends on balance of activity in these two pathways

Dopamine’s role in these pathways

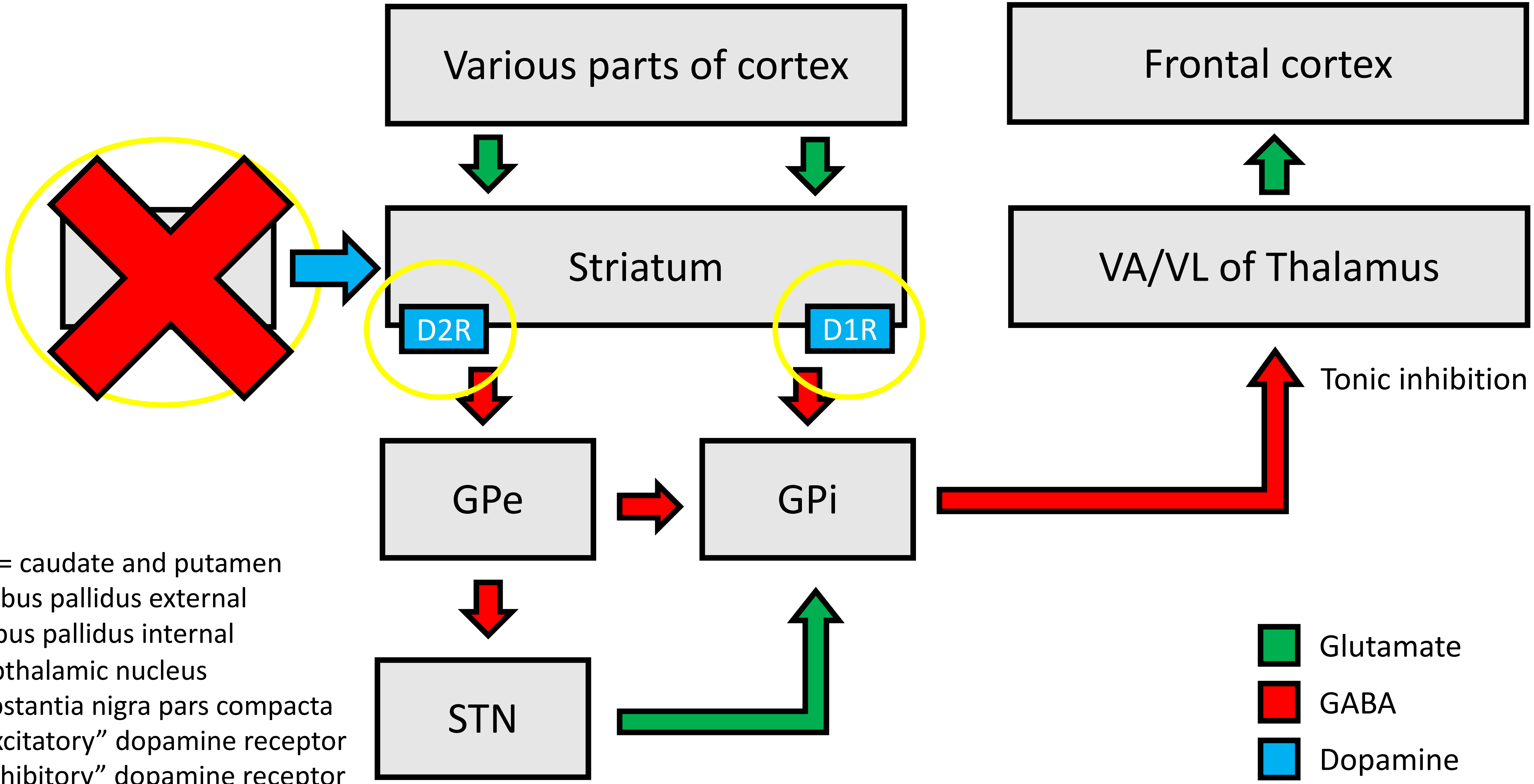


Striatum = caudate and putamen
GPe = globus pallidus external
GPi = globus pallidus internal
STN = subthalamic nucleus
SNc = substantia nigra pars compacta
D1R = “excitatory” dopamine receptor
D2R = “inhibitory” dopamine receptor

Dopamine’s role in movement

- Dopamine D1-family receptors have a positive modulatory role
- Dopamine D2-family receptors have a negative modulatory role
- SNc releases dopamine onto striatum
- D1R activity increases transmission in the direct/GO pathway
- D2R activity decreases transmission in the indirect/STOP pathway
- Net result: more go, less stop → movement

Parkinson’s disease



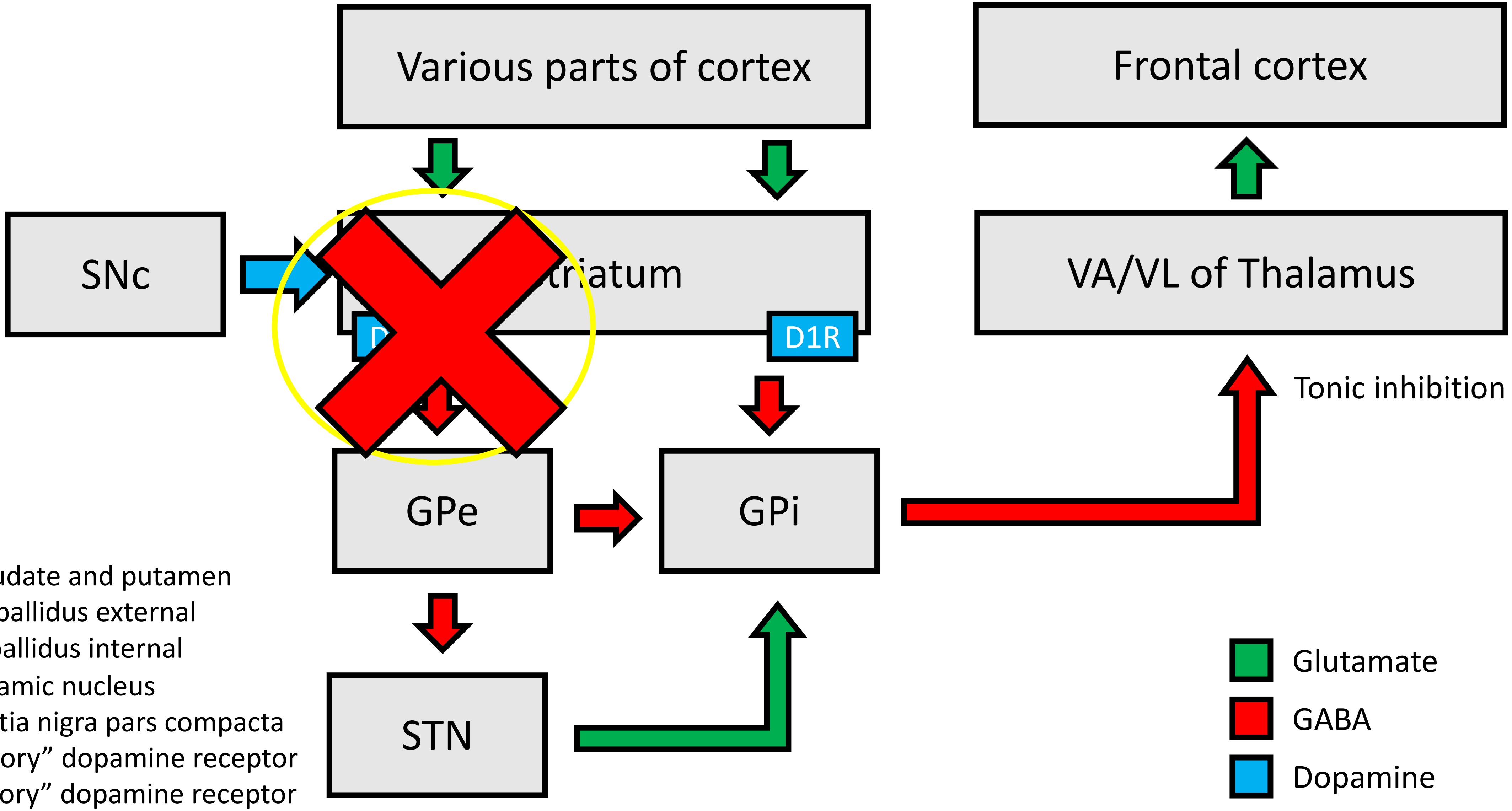
Striatum = caudate and putamen
GPe = globus pallidus external
GPi = globus pallidus internal
STN = subthalamic nucleus
SNc = substantia nigra pars compacta
D1R = “excitatory” dopamine receptor
D2R = “inhibitory” dopamine receptor

Parkinson’s disease

- In PD, most of the dopaminergic neurons of the SNc die
- As such, dopamine is not released onto the striatum
- This decreases transmission in the GO pathway
- This also increases transmission in the STOP pathway
- Net result: less go, more stop → diminished movement

- Treatment?
  - L-DOPA is the gold standard → effect?
  - Deep brain stimulation (DBS) of the STN → effect?

Huntington’s disease



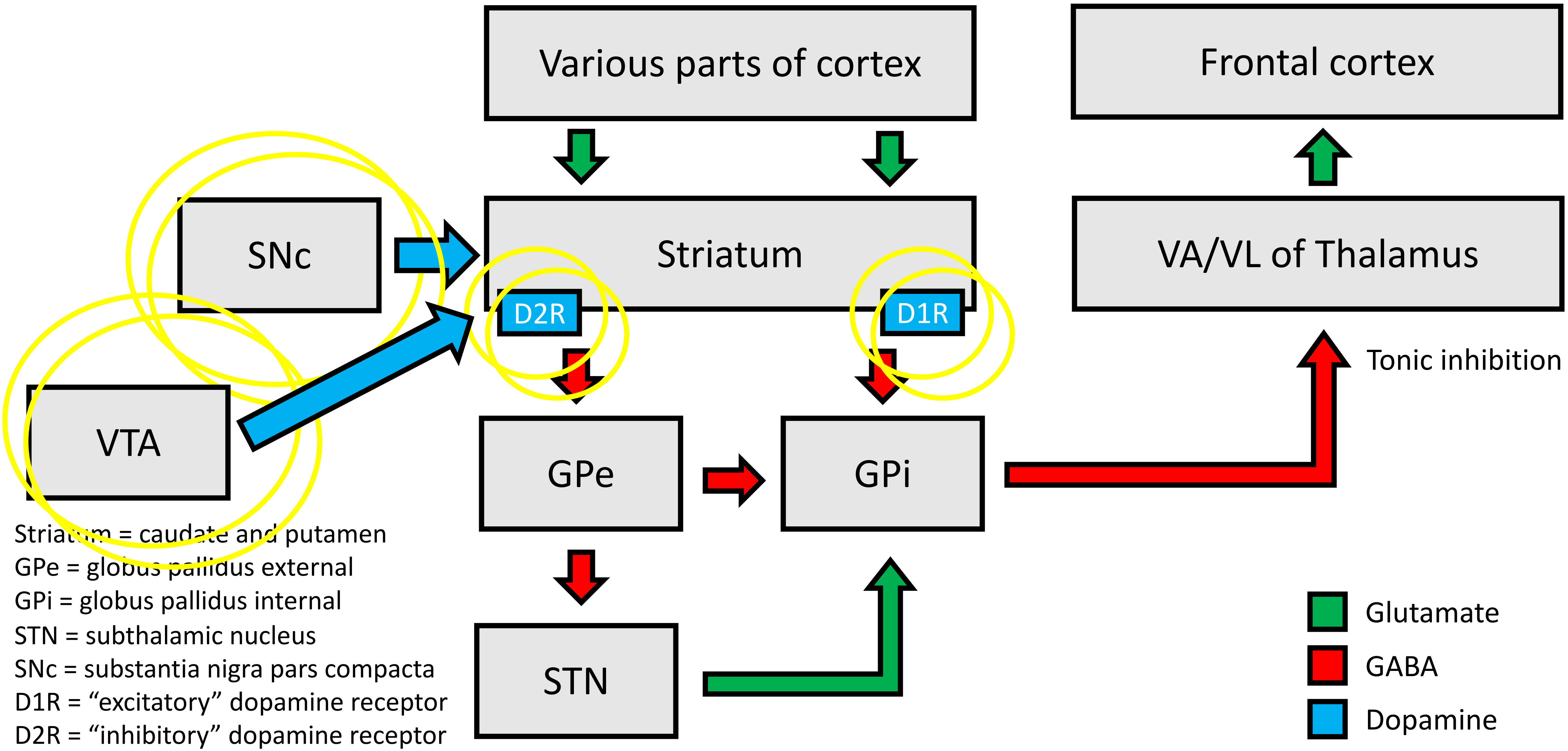
Striatum = caudate and putamen
GPe = globus pallidus external
GPi = globus pallidus internal
STN = subthalamic nucleus
SNc = substantia nigra pars compacta
D1R = “excitatory” dopamine receptor
D2R = “inhibitory” dopamine receptor

Huntington’s disease

- HD affects neurons across the brain, but especially the striatum
- In particular, the striatal neurons that project to GPe die (i.e. neurons in the indirect pathway)
- This decreases transmission in indirect/STOP pathway
- Net result: no stop → excessive movement

- Treatment?
  - Unfortunately very little

Increasing dopamine transmission



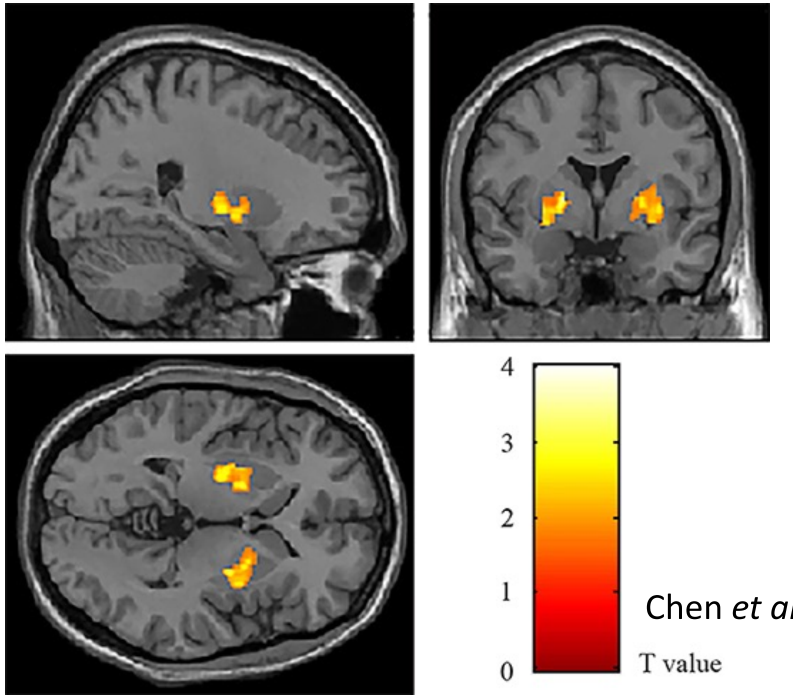
Striatum = caudate and putamen
GPe = globus pallidus external
GPi = globus pallidus internal
STN = subthalamic nucleus
SNc = substantia nigra pars compacta
D1R = “excitatory” dopamine receptor
D2R = “inhibitory” dopamine receptor
VTA = ventral tegmental area

Dopamine and reward

- All drugs of addiction directly or indirectly increase dopamine transmission from SNc and the other main dopaminergic region, the VTA
- While the pathways I've shown you are normally thought of for movement, they apply somewhat (and are analogous to other pathways) for reward and motivation
- **Thus, these drugs can shape our behaviours such that we seek out more of these drugs (e.g. addiction)**
- Psychostimulant drugs like cocaine or amphetamines increase (at lower doses) goal-directed behaviours and impulsivity, and (at higher doses) repetitive behaviours (called stereotypy or punding)
- Effect of prolonged use?
- Treatment?

Other basal ganglia disorders

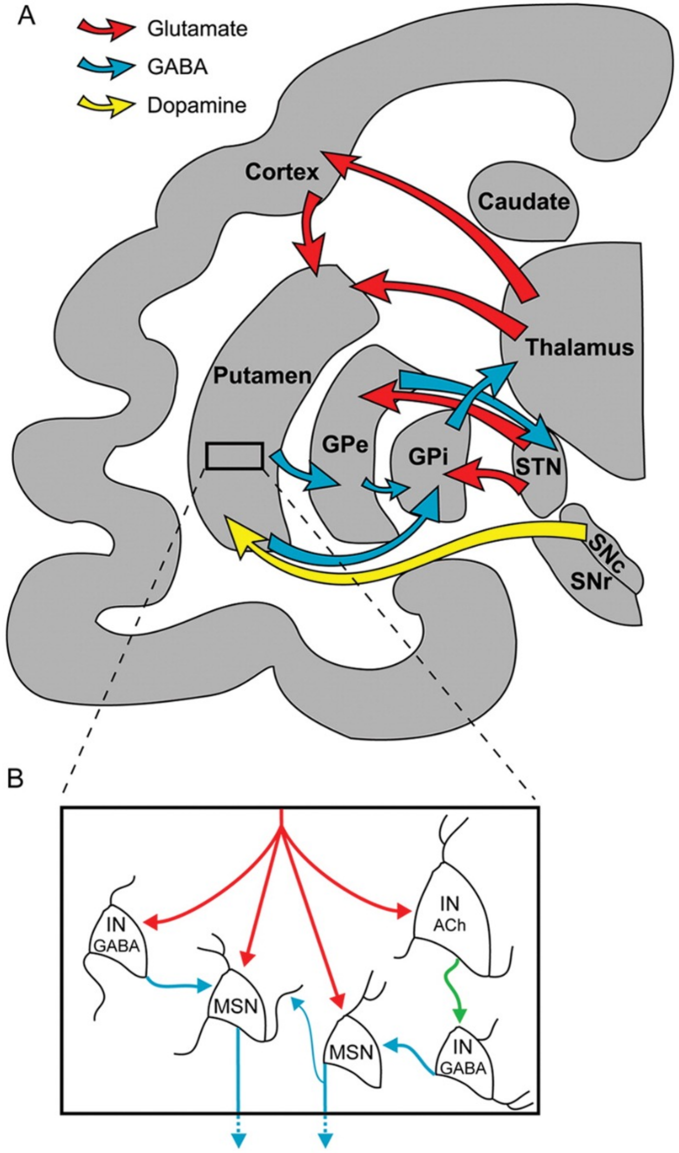
- Obsessive-compulsive disorder
    - Marked by intrusive thoughts, and behaviours to relieve distress of those thoughts
    - Brain data are somewhat heterogeneous/equivocal, but generally:
    - Increased size/activity in some basal ganglial structures (esp. caudate & putamen)
    - Decreased activity in some basal ganglial structures (e.g. globus pallidus)
    - Reduced D2R density in the basal ganglia
      → effect?



Chen *et al*. 2022

Other basal ganglia disorders

Bronfeld and Bar-Gad 2013



- Tourette's syndrome
  - A more severe type of tic disorder
  - Preceded by a premonitory urge, can be briefly suppressed
  - Often accompanied by OCD
  - Often (but not always) spontaneously resolves from childhood to adulthood
  - Neuroimaging suggests excess DA release in striatum → effect?
  - Animal models suggest disinhibition in striatum → effect?

Concluding thoughts on the motor system

- We don't have one motor system, but rather systems built on systems over evolutionary time, each adding nuance and sophistication
- The motor system is tightly coupled to, and relies upon, sensory inputs
- The motor system is more than just a motor system, and interacts with many aspects of cognition (e.g. decision making, attention, memory)
- The standard motor hierarchy helps us understand going from a vague notion to a concrete motor plan
- The cerebellum and basal ganglia help us understand how our motor plans are adjusted, learned, adapted upon
- Understanding the basal ganglia helps us understand "muscle memory", Parkinson's, Huntington's, addiction, and more