PHYSIOLOGY OF BASAL GANGLIA & REGULATORY MECHANISMS

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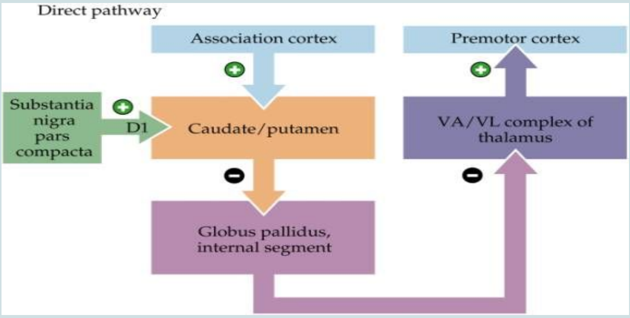
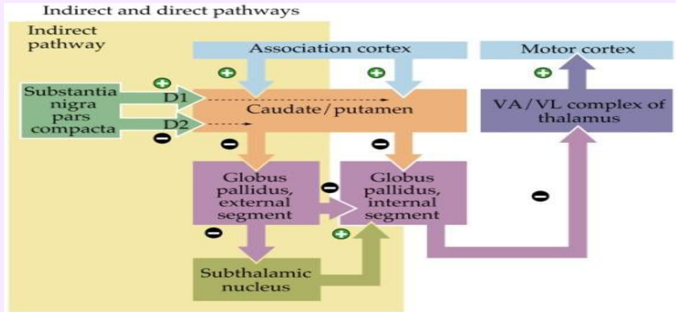
SUMMARY:

basal ganglia			
components	• Caudate nucleus • Lenticular nucleus (putamen + globus pallidus) • Associated structures: subthalamus + substantia nigra		
Functions	1. Control of movements → Planning and programming of movements	2. Cognition	
connections	main input	They come from the cerebral cortex (motor area) and projects to the neostriatum (caudate nucleus + putamen)	
	main output	Via the thalamus to the cerebral cortex (motor area)	
	between its parts	Motor loop (putamen circuit)	• Concerned with learned movement "it executes Learned Patterns of Motor Activity" • From: premotor area + supplementary motor + somatosensory cortex • To: primary motor cortex + premotor area + supplementary motor • Basal ganglia function in association with the corticospinal system to control complex patterns of motor activity • Examples: writing/cutting paper/shooting a basketball …etc
		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) • Cognitive control of motor activity is determined subconsciously & within seconds à patterns of movement will be used together to achieve a complex goal • From: associations area • To: prefrontal + premotor area + supplementary motor • Examples: A person seeing a lion
			• It is also concerned with changing the timing & Scaling the Intensity of Movements • Two important capabilities of the brain in controlling movement are: 1. To determine how rapidly the movement is to be performed 2. To control how large the movement will be
		Limbic loop	Involved in giving motor expression to emotions like, smiling, aggressive or submissive posture.
		Occulomotor loop	Concerned with voluntary eye movement [saccadic movement]

basal ganglia pathways	
Direct pathway	Indirect pathway

Direct pathway	Indirect pathway
● From cerebral cortex to striatum “glutamate” ● Excited striatum release GABA to GPi ● Inhibited GPi release ↓ GABA to the thalamus ● <u>Thalamus is disinhibited</u> “released from inhibition” so it release ↑ glutamate to cerebral cortex → facilitation of movement “↑ motor activity”	● From cerebral cortex to striatum “glutamate” ● Excited striatum release ↑ GABA to GPe ● Inhibited GPe release ↓ GABA to subthalamic nucleus ● <u>subthalamic nucleus is disinhibited</u> “released from inhibition” so it release ↑ glutamate to GPi ● Excited GPi release ↑ GABA to thalamus ● Thalamus is inhibited so it release ↓ glutamate to cerebral cortex → suppression of movement “↓ motor activity”

* Role of dopamine “Ach antagonizes it” - it is released from substantia nigra pars compacta and <u>it facilitates movements</u>

Dopamine bind to D1 receptor “stimulation”	Dopamine bind to D2 receptor “inhibition”
● From SNc to striatum “dopamine” ● Excited striatum release ↑ GABA to GPi ● Inhibited GPi release ↓ GABA to the thalamus ● <u>Thalamus is disinhibited</u> “released from inhibition” so it release ↑ glutamate to cerebral cortex → facilitation of movement “even more ↑ motor activity”	● From SNc to striatum “dopamine” ● Inhibited striatum release ↓ GABA to GPe ● Disnhibited GPe release ↑ GABA to subthalamic nucleus ● Inhibited subthalamic nucleus release ↓ glutamate to GPi ● Less excited GPi release less GABA to thalamus ● Thalamus release more glutamate to cerebral cortex → facilitation of movement “↑ motor activity”

- Activation of direct pathway facilitates movement & Activation of indirect pathway suppresses movement
- Direct output makes <u>focal inhibitory</u> contact on GPi/SNr
- Indirect output makes <u>diffuse, widespread excitatory</u> contact on GPi/SNr
- Co-activation of these pathways facilitates <u>action selection</u> through center-surround mechanism

<u>Why do we need action selection?</u>
Multiple stimuli in our environment often demand our attention/action. However, we’re often confined to make a single action to address these stimuli. Particularly where conflicting needs are present, action may require active inhibition

basal ganglia disorders

effects	● Movements (ataxia rate, range, force, direction) ● Speech □+ Posture □+ Gait +□ Mental activity

movement disorders

hyperkinetic	hypokinetic
<u>Chorea “Huntington/wilson’s disease/rheumatic fever”</u> ● Quick random movements “appendicular muscles” ● Atrophy of <u>striatum</u> <u>Athetosis “Huntington/wilson’s disease/rheumatic fever”</u> ● Slow movements “appendicular muscles” ● Diffuse hypermyelination of <u>corpus stiatum & thalamus</u>	<u>Parkinson’s disease</u> ● Pill rolling tremor of the fingers at rest ● Lead pipe rigidity ● Hypokinesia or akinesia ● Degenration of <u>Substantia Nigra “dopaminergic nigrostriatal□neurons”</u>
<u>Hemiballismus “hypertensive patients”</u> ● Wild flinging movements of half of the body ● Hemorrhagic destruction of contralateral <u>subthalamic n</u>	<u>Drug induced “MPTP”</u> ● The oxidant MPP+ is toxic to <u>Substantia Nigra</u>