

BASAL GANGLIA

I. OVERVIEW

- a. Function
 - i. Modulate cerebral cortex outputs -suppress unnecessary actions
 - ii. No direct innervation → lesions never produce paralysis, only imbalance
 - iii. Comparison to cerebellum:
 - 1. Cerebellum receives input from only motor centers, output to both motor cortices and all subcortical centers
 - 2. Basal ganglia receive input from entire cortex, output only to motor cx
- b. Deep telencephalic nuclei (noncortical gray matter)
 - i. Amygdala – see limbic system
 - ii. **Caudate and putamen** = “striatum”
 - 1. Developmentally identical; cannot be distinguished in lower mammals
 - 2. In primates, separated by internal capsule
 - iii. **Globus pallidus** = “pallidum”
 - iv. Claustrum – no known function
- c. Associated nuclei
 - i. Diencephalon: ventral anterior thalamic nucleus, ventral lateral thalamic nuc, central median nuc, **subthalamic nuc**
 - ii. Mesencephalon: **substantia nigra**
 - iii. NOT red nucleus, reticular formation

II. CONNECTIONS

- a. Caudate and putamen
 - i. Caudate = projections mostly from prefrontal, temporal, parietal assoc areas
 - ii. Putamen = from motor, somatosensory cortices
 - iii. Inhibitory to Globus pallidus (GABAergic)
 - *Ventral striatum (nucleus accumbens – rec orbitofrontal) to ventral pallidum
- b. Globus pallidus
 - i. External segment (GPe)– inhibitory (GABA) to Subthalamic nucleus
 - ii. Internal segment (GPi) – inhibitory (GABA) to VL/VA, CM of thalamus (balances excitatory input from dentate nucleus of cerebellum)
 - iii. **Substantia nigra pars reticulata** – part of internal segment – same connections
 - iv. 2 pathway to thalamus: via **ansa lenticularis** (around cerebral peduncle) or **lenticular fasciculus** (through internal capsule)
 - *ventral pallidum to MD of thalamus
- c. Subthalamic nucleus
 - i. Excitatory to internal globus pallidus (GPi) (inhibits thalamus → cortex)
 - ii. Receives strong excitatory projection from motor cx (net inhibition of cortex)
- d. **Substantia nigra pars compacta** – DA efferents to caudate/putamen
 - i. D1 = excitatory, D2 = inhibitory
 - ii. Projects to pallidum, VL/VA = indirect/direct influence
- e. Afferents from thalamus to Cortex
 - i. VL → primary motor, premotor
 - ii. VA → motor cortices; diffuse to prefrontal, parietal, temporal, occipital cortices

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- iii. CM → somatosensory cortex; diffuse to cerebral cortex
 - *MD → prefrontal, limbic cortices
 - f. Other descending pallidal efferents
 - i. small projections to midbrain reticular form. → modulate ReST (muscle tone)
 - ii. small projections to tectum → tectospinal system (orienting)
 - iii. small proj to habenula → limbic system (species-preserving behaviors)
- III. PATHWAYS (SUMMARY OF CONNECTIONS)
- a. Direct pathway
 - i. Cortex (+) → caudate/putamen (-) → GPi/SNpr (-) → VA/VL (+) → Cortex
 - ii. Net excitatory effect on cortex (2 inhibitions cancel - disinhibition)
 - b. Indirect pathway
 - i. Cortex (+) → caud/put (-) → **GPe (-) → STN (+) → GPi (-) → VA/VL (+) → Cortex**
 - ii. Net inhibitory effect on cortex
- IV. CLINICAL CONSIDERATIONS
- a. Damage to basal ganglia – classical symptoms:
 - i. Increased motor tone – rigidity
 - ii. Dyskinesias – tremors, other involuntary movement
 - iii. Bradykinesia/Akinesia – slowing/ almost absent voluntary movement
 - b. Hemiballismus
 - i. Damage to subthalamic nucleus or its connections to GPi (unilateral)
 - ii. Involuntary, irregular flinging of contralateral extremity (usually upper)
 - c. Huntington's disease
 - i. Early degeneration of the striatum
 - ii. Involuntary movements "choreiform" - writhing
 - d. Parkinson's disease
 - i. Damage to SNpc (depletion of DA in striatum)
 - ii. Rigidity and tremor, brady/akinesia
 - iii. Reflects disruption of cortical - basal ganglia - cortical loop:
 - 1. Direct pathway: loss of DA1 → decrease inhibition of GPi... less net excitation of cortex
 - 2. Indirect pathway: loss of DA2 → more inhibition → less net excitation of cortex
 - 3. Result is akinesia
 - iv. Imbalance of excitatory (D1) and inhibitory (D2) effects of pathway → treat by restoring balance:
 - 1. increasing efficacy of dopamine release (administer L-DOPA)
 - 2. lesions to block GPi/STN activity
 - 3. transplant dopaminergic neurons
- *Pyramidal and Extrapyramidal syndromes → implies anatomical-functional relationships that don't reflect actual organization (damage is caused by damage to motor cortex, not pyramids or pyramidal tract)