Medical Neuroscience | Tutorial Notes

Modulation of Movement by the Basal Ganglia—Normal and Abnormal Movement

MAP TO NEUROSCIENCE CORE CONCEPTS[1]

NCC1. The brain is the body's most complex organ.

NCC3. Genetically determined circuits are the foundation of the nervous system.

NCC4. Life experiences change the nervous system.

LEARNING OBJECTIVES

After study of the assigned learning materials, the student will:

1. Discuss the critical role of dopamine in facilitating the function of basal ganglia circuitry.
2. Explain hypokinetic movement disorders in terms of the function of basal ganglia circuitry.
3. Explain hyperkinetic movement disorders in terms of the function of basal ganglia circuitry.

TUTORIAL OUTLINE

I. Basal ganglia function
   - A. normal functions
     1. general properties
        - a. neurons in the striatum fire action potentials before and during the onset of movement
        - b. suggests a role for basal ganglia in initiation of movement, not the ongoing coordination of movements (role played by cerebellum)
     2. for both the dorsal motor and ventral limbic streams, the balance of activation in the direct and indirect pathways serves to **initiate** the selected behavioral program and **suppress** non-synergistic programs
     3. conceptually, this is another example of "center" (initiation of intended behavior) "surround" (suppression of unintended behavior) antagonism (consider **Figure 18.8**[2])
   - B. abnormal function in disease states
     1. Parkinson's disease

[1] Visit **BrainFacts.org** for *Neuroscience Core Concepts* (©2012 Society for Neuroscience) that offer fundamental principles about the brain and nervous system, the most complex living structure known in the universe.

[2] Figure references to Purves et al., *Neuroscience, 5th Ed.*, Sinauer Assoc., Inc., 2012. [click here]

a. symptoms: akinesia (lack of voluntary movement) or bradykinesia (extreme slowness of voluntary movement), reduced facial expressions, postural rigidity, resting tremor (e.g., "pill-rolling" tremor) in arms/hands, shuffling ("festinating") gait, difficulty initiating and terminating movements, cognitive changes (dementia)

b. onset: typically after the fifth decade of life

c. pathology: loss of (>80%) dopaminergic neurons in the substantia nigra, pars compacta

(i) by "natural" (genetic/epigenetic) causes; etiology is unknown

(ii) by abuse of MPTP (synthetic heroin)

d. consequences for basal ganglia function (see **Figure 18.10A**)

(i) decreased output from caudate/putamen to internal segment of the globus pallidus

(ii) excessive tonic inhibition of ventral anterior/ventral lateral complex of the thalamus

(iii) decrease activation of motor cortex via ventral anterior/ventral lateral complex

2. Huntington's chorea (autosomal dominant; onset between 30-50 years)

a. symptoms: involuntary choreic ("dance-like") movements: may be jerky, hyperkinetic, and/or dystonic; severe dementia then ensues

b. onset: usually in the 4^{th} or 5^{th} decades of life, with an average life expectancy of about 15 years thereafter

c. pathology

(i) widespread degeneration of cerebral cortex and striatum

(ii) especially affected are striatal neurons that project to the external segment of the globus pallidus (indirect pathway)

d. consequences for basal ganglia function (see **Figure 18.10B**)

(i) decreased output from caudate/putamen to external segment of the globus pallidus

(ii) increased tonic inhibition of subthalamic nucleus

(iii) decreased drive of internal segment of globus pallidus via subthalamic nucleus

(iv) decreased tonic inhibition of ventral anterior/ventral lateral complex of the thalamus

(v) increased activation of motor cortex and via ventral anterior/ventral lateral complex

STUDY QUESTION

In idiopathic **Parkinsonism**, there is pathological difficulty initiating movement. Which of the following statements provides the best explanation of this difficulty?

A. There is too much activity in the external segment of the globus pallidus and, therefore, insufficient inhibition of the ventral anterior/ventral lateral complex of the thalamus.
B. There is too much activity in the internal segment of the globus pallidus and, therefore, too much inhibition of the ventral anterior/ventral lateral complex of the thalamus.
C. There is insufficient activity in the subthalamic nucleus and, therefore, insufficient output from the internal segment of the globus pallidus.
D. The subthalamic nucleus is overactive, which results in the excessive disinhibition of the ventral anterior/ventral lateral complex of the thalamus.
E. The substantia nigra pars compacta is overactive, which results in the excessive disinhibition of the ventral anterior/ventral lateral complex of the thalamus.