9.14 class #30: Corpus striatum.

Readings:

Nauta & Feiertag, Extrapyramidal System, pp. 97 -101.

Brodal, Per ,"Chapter 13, The basal ganglia, *The Central Nervous System: Structure and Function, edition 2.* , 1998,pp. 371 – 391.

Mesulam, M.-Marsel .,"Chapter 1, Behavioral neuroanatomy: Large-scale networks, association cortex, frontal syndromes, the limbic system, and hemispheric specializations" *Principles of Behavioral Neurology,* Philadelphia, F.A. Davis Company2001, (pp. 1-120). For this class, study (Basal ganglia and cerebellum)pp. 66-71.

Questions:

1. What is meant by the statement that the major output of the extrapyramidal system is the pyramidal system? What other output does the striatum have?

2. What is the "ansa lenticularis"?

3. What are the major satellites of the striatum? How are they connected to the striatum?

4. What is the limbic striatum? What structures does it include, and how does it differ from the non-limbic striatum?

5. Chemoarchitectural studies have given two very different kinds of information about the caudate and putamen: they have revealed a pattern in overall structural organization, and they have showed cell-type differentiation. What is the pattern, and give examples of cell types according to chemical content.

6. Contrast different routes for sensory information to travel from primary sensory cortical areas, e.g., visual cortex, to motor output systems. Use information from previous 9.14 sessions as well as the new information. You should be able to describe at least two or three.

7. Contrast, for the most strictly sensori-motor components of the caudate-putamen, the pathways to motor cortex and the pathways to the superior colliculus.

8. What is the "double inhibition" of pathways through the striatum? What neurotransmitter is involved?

9. Understanding striatal function has been made especially complicated by the discovery of multiple parallel pathways, with possibly opposing effects on movement. Give an example.

10. The corpus striatum is called a cross-roads of limbic and non-limbic systems. What does this mean?

11. Why is it that basal ganglia disease causes hyperkinesias (too much movement)? Give examples of such hyperkinesias, and describe a possible mechanism underlying the disorder.

Supplementary readings:

Sawle, G.V. ," Living neurochemistry: the dopaminergic system", *Seminars in the Neurosciences,*1995,7: 173 - 177.

Johnston, J. G. ," The development of striatal compartmentalization: the role of mitotic and postmitotic events", *The Basal Ganglia III,* G. Bernardi et al. (Eds.), Plenum Press, pp. 13 - 20.

Calabresi, P., Pisani, A., Mercuri, N.B. and Bernardi, G. ," The corticostriatal projection: from synaptic plasticity to dysfunctions of the basal ganglia", *Trends in Neuroscience,* 1996, 19*:* 19 - 24.

Progress in Brain Research, vol .xxx, 1999: