THE “MYSTERIOUS BASAL GANGLIA”
(DAVID MARSDEN): WHY? NAMES!

Globus pallidus = pale globe, internal (Gpi) and external (Gpe) aka “pallidum” (slang)

Putamen = shell

Globus pallidus + Putamen = “Lenticular nucleus” (lens-shaped)

Caudate = tail-like

head, body, & tail (!) of caudate

Globus pallidus + Putamen + Caudate = “Striatum” (striped by internal capsule)

Accumbens = “leaning” up against the septum, a part of head of caudate and putamen

Substantia nigra = “black stuff”

Subthalamic nucleus (STN)

Amygdala = almond shaped, now considered part of the limbic system.
THE MYSTERIOUS BASAL GANGLIA (C.D. MARSDEN)

MOST OF WHAT WE THINK WE KNOW ABOUT THE BASAL GANGLIA BEGAN WITH (AND STILL CONSISTS OF) KNOWLEDGE OF SIGNS AND SYMPTOMS OF BG DISEASES: FOR EXAMPLE, THE “RESTING” FIXED FLEXED POSTURE OF PARKINSON’S DISEASE.

THIS HAS LED TO EFFECTIVE TREATMENTS, BUT WE STILL HAVE MANY QUESTIONS ABOUT THEIR NORMAL FUNCTIONS AND WHY THE TREATMENTS WORK.
OLD STORY: BASAL GANGLIA EXCITE TARGETS AND INITIATE POSTURE AND MOVEMENT (Denny-Brown, Mehler); CEREBELLUM MODIFIES P+M AFTER INITIATION (BY MOTOR CORTEX: Sherrington, Ruch).
NEW STORY: BASAL GANGLIA INHIBIT THEIR DOWNSTREAM TARGETS  (Anderson; Penney and Young)
• THE BRAKE MODEL: BASAL GANGLIA INHIBIT THOSE MOVEMENTS WE DON’T WANT TO MAKE, AND ALLOWS/SELECTS THOSE MOVEMENTS WE WANT TO MAKE.

• SOME BASAL GANGLIA DISEASES FEATURE THE UNWANTED INHIBITION OF ALL MOVEMENTS (PARKINSON’S); OTHERS FEATURE THE UNWANTED RELEASE OF MOVEMENTS (HUNTINGTON’S)
Dyad: BG “brake” model

You have parked your car on a hill facing up the incline. Now you want to start up and drive off.

• Q1: When do you start the engine and press on the gas pedal accelerator?
• Q2: When you release the parking brake?
• Q3: Is the accelerator depressed and the brake ever on at the same time?
SIGNS OF DISEASE OF BASAL GANGLIA OCCUR AT “REST”:
1) COCONTRACTION
RIGIDITY OF AGONISTS AND ANTAGONISTS,
2) ABNORMAL POSTURES
AND
3) INVOLUNTARY
MOVEMENTS (TREMOR, CHOREA, ATHETOSIS).

NOTE: THE ABNORMAL POSTURES AND INVOLUNTARY MOVEMENTS LOOK LIKE EXAGGERATIONS OF THE NORMAL.

Figure 3.
Figure 4. Patterns of involuntary movements showing frequency, relative amplitude, and regularity.
Cerebral Palsy with athetosis = changing posture

Dystonia Musculorum Deformans with athetosis

Huntingdon’s Disease with chorea = dance
NEW STORY: BASAL GANGLIA DISCHARGE *AFTER* MOVEMENT (EMG) HAS BEGUN (Anderson; Mink)

THE CEREBELLUM DISCHARGES *BEFORE* MOVEMENT (EMG) HAS BEGUN (DENTATE LEADS MOTOR CORTEX WHICH LEADS EMG (Thach). DENTATE COOLING DELAYS MOTOR CORTEX AND MOVEMENT (Brooks et al.).
Inactivation of Gpi impairs turning off the antagonist muscles.
NEW HYPOTHESIS:
The basal ganglia help to *SELECT* the movement that we *WANT* to make by *INHIBITING* those movements and postures that we *DON’T WANT* to make and by *DISINHIBITING* those movements we *DO WANT* to make.

Otherwise, there might be conflict between wanted and unwanted movements and postures (as in diseases of the basal ganglia).

For example, to *EXTEND* the arm in reaching, we have to *INHIBIT* the postural reflexes in the arm, which would otherwise keep the arm *FLEXED*. 
Proposed mechanism for INHIBITION/SELECTION (Mink)

Activity of Gpi Target Neurons

Figure 2. Diagram of effects of neurons in Gpi and posture generators in thalamus and PPN.
NORMAL......

Figure 1
BAD NEWS:
In normal subjects, lesion of STN causes *Chorea* = incessant involuntary “dancing” movements.

GOOD NEWS:
In Parkinson patients, STN lesion or stimulation removes rigidity and restores normal movement.

HOW????????$$$$$$$

Figure 1.
• Some Basal Ganglia Paradoxes
  • The motor deficits of Parkinson's Disease are similar to those following damage of the GPI basal ganglia output cells. Yet, oddly, one of the first effective surgical treatments consisted of ablating a medial portion of GPI to relieve the symptoms and signs of Parkinson’s Disease (Cooper)!
  • Another ablative treatment of PD is to lesion STN. Yet in normals, lesion of STN causes TOO MUCH movement: wild flinging movements (hemibalismus).
  • To complicate the issue further, another treatment of PD is to implant chronic electrodes to electrically stimulate STN! Why should STN stimulation have the same result as ablation? Who knows?
• As for the chemistry, PD is caused by a deficiency of dopamine secretion onto putamen and caudate by dying SNpc cells. Restoration of dopamine production by supplying the precursor, L-Dopa, relieves the symptoms of PD, but given in excessive amounts, may cause involuntary movements resembling hemiballismus.
• Giving drugs that block dopamine receptors (for schizophrenia) may cause a motor disability indistinguishable from PD.
• In sum, an important principle in the function of the basal ganglia appears to be the maintenance of the proper balance of inhibition and disinhibition of the downstream motor targets, as formulated in the brake-selector hypothesis.
Basal Ganglia in Mental Control?

Anatomy: basal ganglia project to many regions of frontal cortex.

Diseases with unwanted mental activity include:
- Schizophrenia
- Tourette’s Syndrome
- OCD

Drugs (phenothiazines, haloperidol) may eliminate unwanted mental activity, but may cause a movement disorder that resembles Parkinson’s Disease.

PD drugs (levodopa) helps movement but impairs thinking.

STN stimulation for Parkinson’s Disease helps movement but impairs thinking.
BASIC FUNCTIONS OF THE BASAL GANGLIA

• MOTOR BRAKES: BASAL GANGLIA INHIBIT THOSE MOVEMENTS WE DON’T WANT TO MAKE, AND ALLOWS/SELECTS THOSE MOVEMENTS WE DO WANT TO MAKE.

• SOME BASAL GANGLIA DISEASES FEATURE THE UNWANTED INHIBITION OF ALL MOVEMENTS (PARKINSON’S); OTHERS FEATURE THE UNWANTED RELEASE OF MOVEMENTS (HUNTINGTON’S)