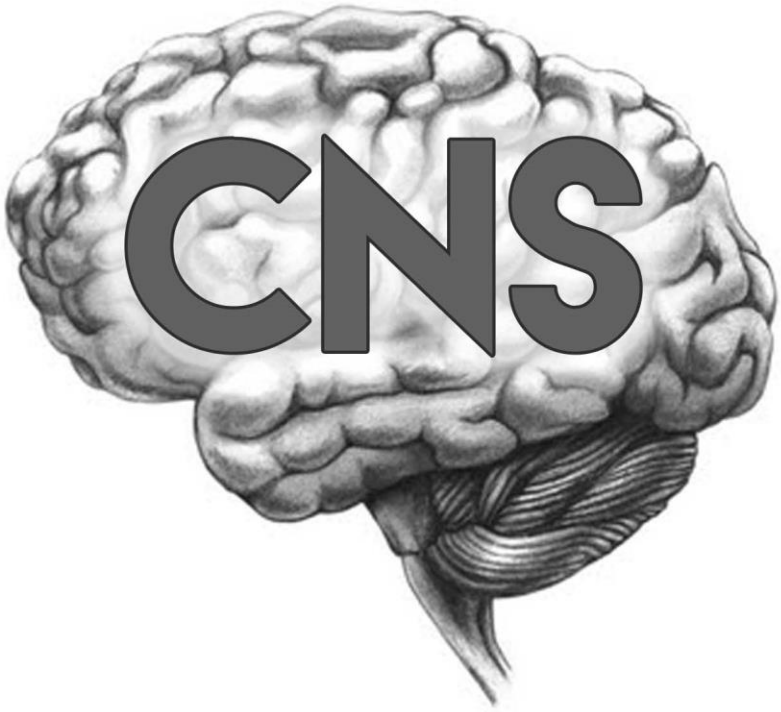


ANATOMY

☑ Sheet ○ Slide ○ Handout

Number

١٠

Subject

Basal Ganglia

Done By

Abdallah S. S.

Corrected by

.....

Doctor

Faraj Al-bustami

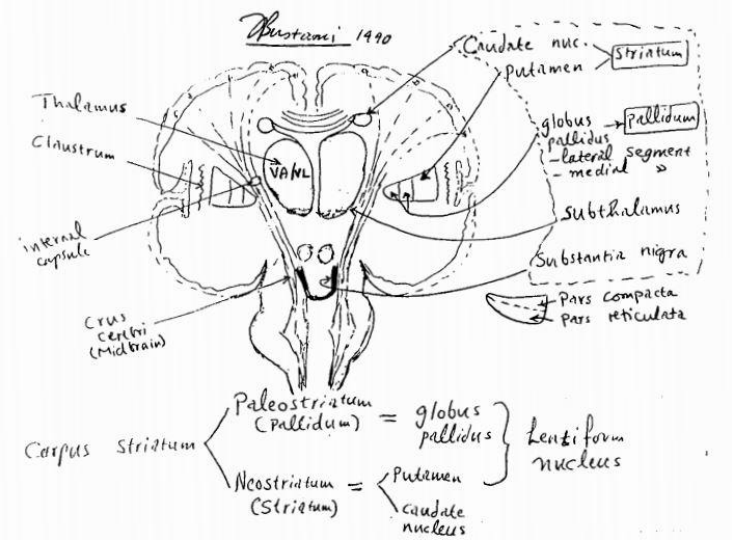
Date:

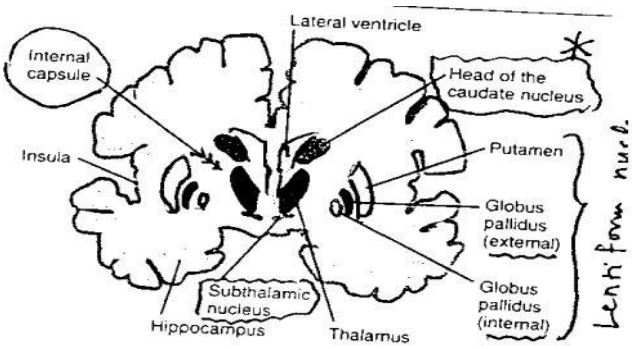
Price:

بسم الله الرحمن الرحيم

This sheet is written according to section ٢ recording. Hope you enjoy studying it.

See these two figures just to be familiar with basal ganglia structure:





- The basal ganglia and cerebellum control the commands and activity of the upper motor neurons
- Basal ganglia is mainly involved in initiation and control of movements; mostly for axial muscles which are responsible for the posture for a phasic movement as writing on a board.

☞ <u>How it works</u>? (see the figure)

- As a feedback Reciprocal (same as the cerebellum) .
- Doesn't deal with (α) and (γ) motor neurons.
- There's **NO** tract called cerebelluspinal tract.
- Also basal ganglia doesn't deal with (α) and (γ).
- Area ٦, lateral cerebellum and basal ganglia program the movement, but the only parts that descend are area ٤ and ٦.

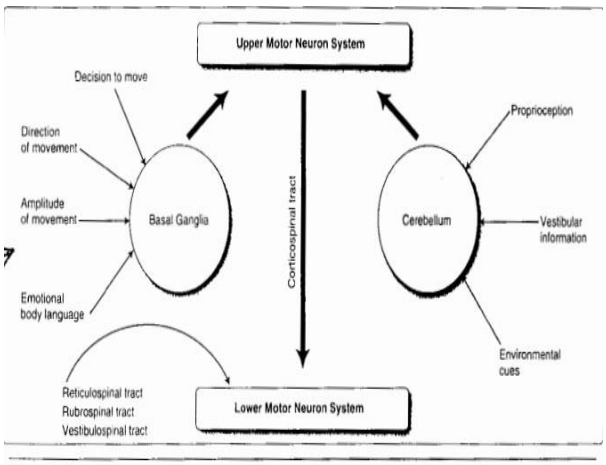
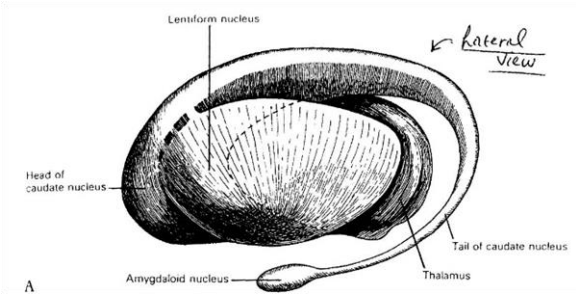


Figure 16.1
Conceptual overview of motor control.

ᘐ <u>Structure ;</u>

- Mainly consists of caudate and Lentiform (putamen and GP-I-medial & GP-E-lateral).
- Caudate is a comma shaped of a head, body & tail.
- Recall : caudate and putamen are separated anatomically , while connected functionally as striatum {entrance for all over cortex to basal ganglia} (aka input)
- While the output goes through GP-I segment to the thalamus to finally reaches the cortex.



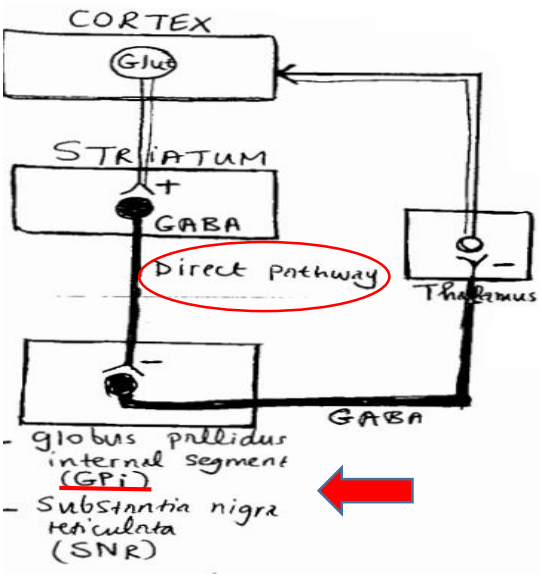
- There are two nuclei functionally connected to basal ganglia ; substantia nigra in midbrain and subthalamic nucleus in diencephalon . (outside basal ganglia)

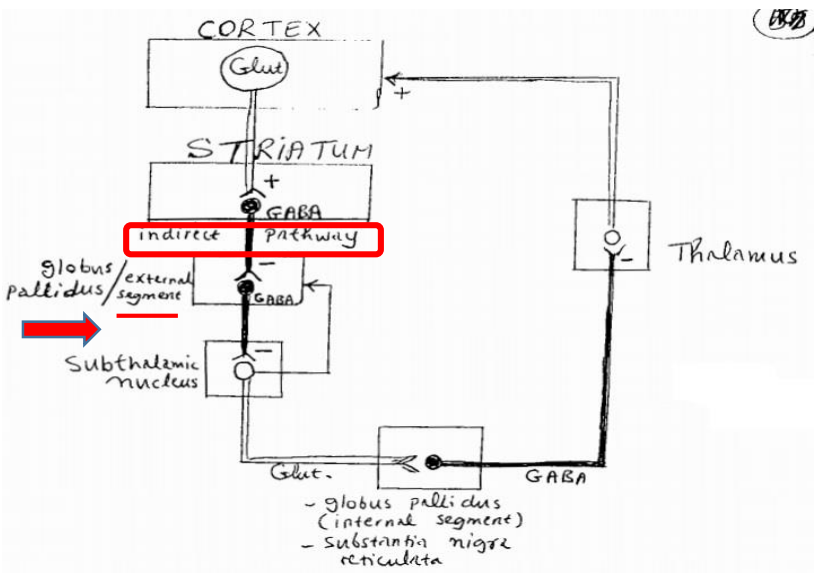
- Substantia nigra consists of two parts :
  - Dorsal part (pars compacta) ; supplying the striatum with dopamine.**(↓dopamine>Parkinson's D)**
  - The pars reticulate serves mainly as an output, conveying signals from the basal ganglia to numerous other brain structures.

<u>So the basal ganglia output goes through both GP-I and pars reticulate</u>

- Recall :
  - The spasticity of upper motor neurons is from disinhibition of pontine-reticulospinal tract.
  - Pontine tract descends mainly contralateral and passes segmental at the spinal cord, and this explains the spasticity and hyper-tonia.
- Basal ganglia regulates the aspects of movement; decision, direction and amplitude.
- E.g. patient with Parkinson's needs long time to decide to move …
- Motor expression of behavior is also a function of basal ganglia; e.g. if you visit the same patient, you notice that he has a **<u>mask face</u>** as he lost face expressions. (see page ٦)

- Now let's discuss the old functional circuits :
  - The input enters the striatum which composed of two types of cells ;
    - cholinergic(excitatory) : secretes ACh
    - GABAergic (inhibitory) : secrets GABA
  - Striatum here receives excitatory impulses from all over the cortex {sensory + motor + association} via glutamate (glutamatergic)
  - Then as the information integrates , striatum sends out GABAergic fibers to the GP.
  - GP in turn sends GABAergic projection to the thalamus that excites the cortex.
  - As you see the GP receives and sends inhibitory projections, so inhibition of inhibition will cause excitation to the thalamus (disinhibition). **Therefore, the cortex is excited to regulate the movements.**
  - If the thalamus is highly activated, we get undesirable movements as tremors. But, if it's inhibited, the cortex also will be inhibited and we get slowness in movement.
  - We have to pathways : Direct and Indirect (see the figures)





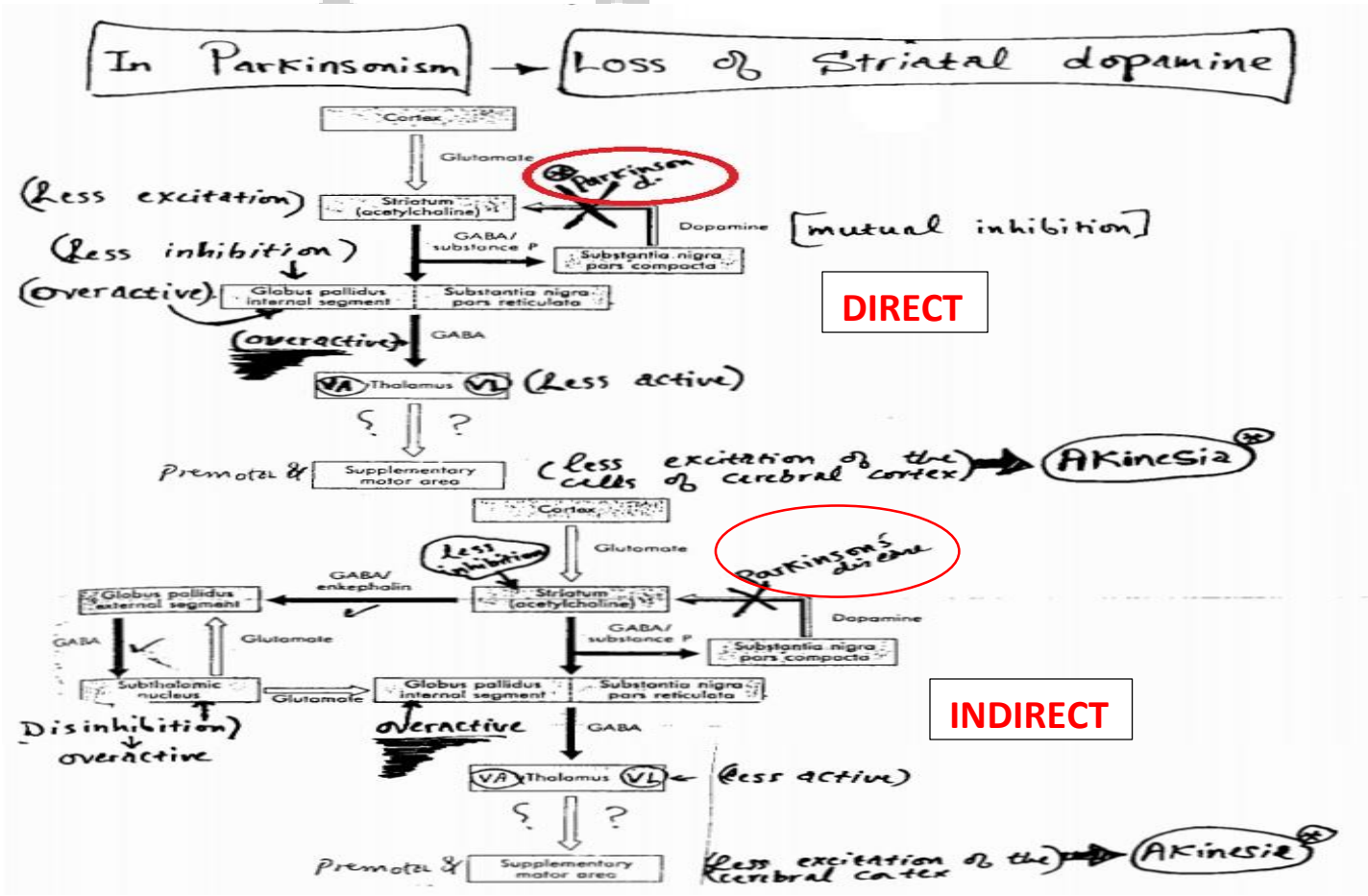
- Direct pathway causes excitation of the thalamus and cortex.
- For Indirect pathway an **inhibitory** projections exit the **GP-E to the subthalamic nucleus** that **excites** the GP-I …..
- Indirect pathway causes excitation of the subthalamus that activates GP-I which inhibits the thalamus and cortex.

ꕥ Direct pathway facilitates the desired movements , while the indirect pathway prevents undesirable movements.

ꕥ The striatum for both is GABAergic but different cells for each.

ꕥ **Parkinson's disease**

- LOSS OF STRIATAL DOPAMINE**.**
- Dopamine is excitatory for direct striatum cells and inhibitory for the indirect. That's receptor dependent. D_1 for direct, where D_2 for indirect.
- **Follow the figure , it's enough.**
- Results in disfacilitation (inhibition) of the cortex, and causes AKINESIA or hypokinesia (slowness in initiation, continuing and termination of the movement).
- **Either direct or indirect pathway, we end with overactive GP-I.**
- Akinesia is neither paralysis nor paresis. (i.e. ULN & LMN are intact.)
- Rest tremor is not always present.



ꕥ **Chorea : is a group of diseases characterized by rapid (dancelike) involuntary movements (DISKINESIA) largely restricted to muscles of distal extremities ;**

- May attacks children as a complication of **rheumatoid** fever of chronic tonsillitis.
- If in adults; called **Huntington** chorea, which is hereditary disease that presents at about age ٢٥th. It's also accompanied with dementia (i.e. cerebral cortex affected also).
- The lesion is in the indirect striatal cells (GABAergic neurons) that inhibit GP-E , so GP-E is disinhibited and results in excessive inhibition to subthalamic nucleus and reduce inhibition of thalamus to finally greater facilitation of cortex and ends with spontaneous & undesirable movements .

ꕥ Chorea: Hyperkinesia & hypo-tonia.

ꕥ Parkinson's D: Akinesia & hyper-tonia (rigidity / **bi**directional resistance). {no hyper-reflexia}
(Also bradykinesia which is hesitation to move)

- Previously, Parkinson's diseases was treated surgically by electrode to destroy GP-I.
- Subthalamic nucleus ;
  - ✓ Suppressed in chorea
  - ✓ Overactivated in Parkinson's
  - ✓ Treated by electrical stimuli in case of Parkinson's to regulate its impulses.

☞ **Overactive GP-I inhibits midbrain extrapyramidal area** , that in turn sends inhibitory fibers for pontine reticulospinal & rubrospinal tracts ; so,

- Disinhibition of (α) and **(γ)** motor neurons of pontine tract (recall : mostly contralateral)

→ as gamma represents stretch reflex , it represent the tone , so we get **hypertonia.** (Mainly **extensors**)

- Disinhibition of (α) and (γ) motor neurons of rubrospinal tract , and we get hypertonia (Mainly **flexors**)

☞ **Parkinson's D: → H.H. ; hypo-kinesis & hyper-tonia.**

ꕥ Patients with Parkinson's have **staring appearance**: Lack spontaneous eye movements accompanied with infrequent blinking. As FEF (broadman , area٨) is affected.

- **Recall:** spasticity is UMN lesion accompanied with hyper-reflexia and differs from rigidity.
- **Again:** Rest tremors are not always present, and affect muscles of fingers (pill-rolling). Patients try to hide this by any beneficial movement as it disappear unlike intention tremor of cerebellar disease.

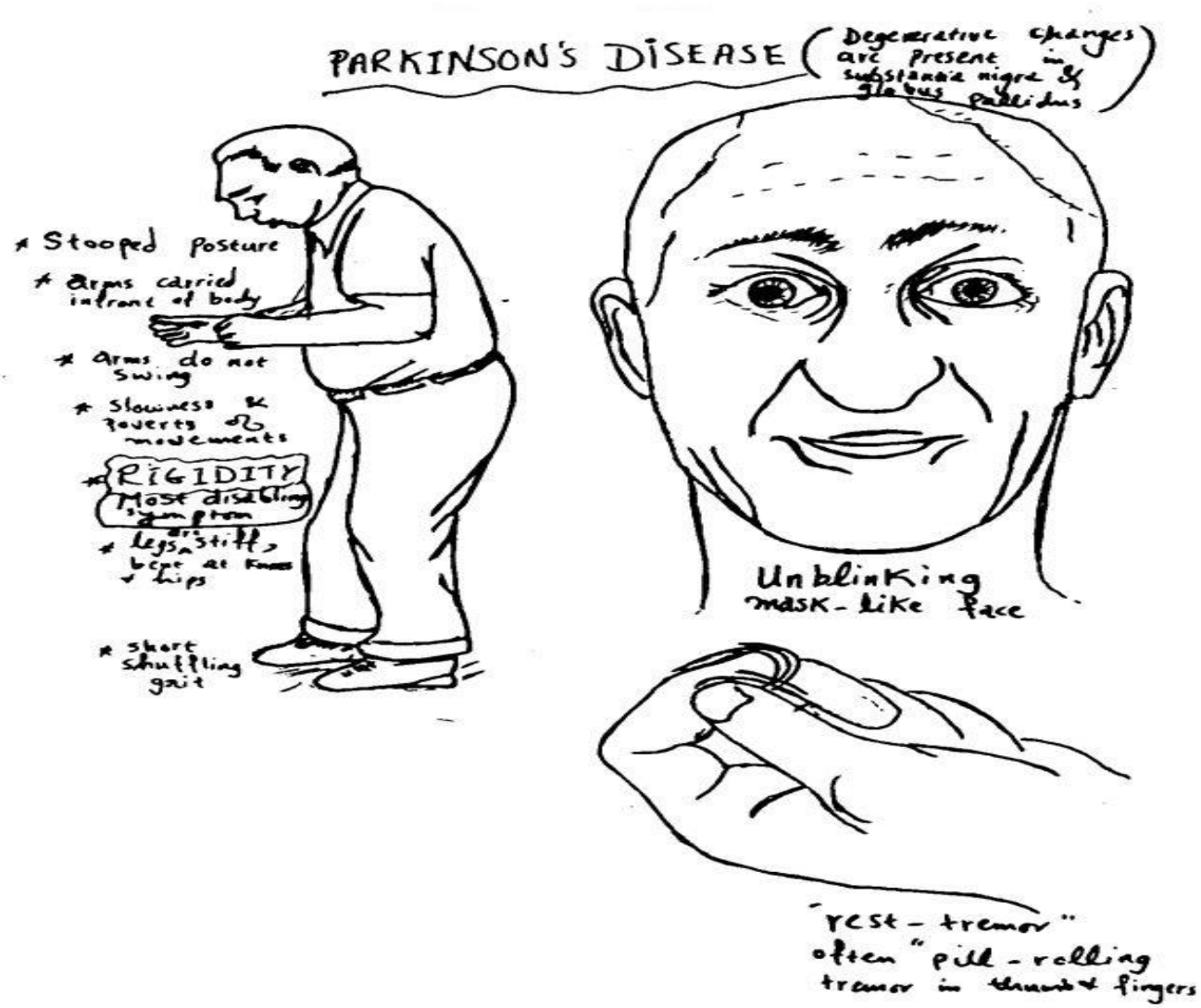
☞ Basal ganglia lesion → dyskinesia (hypo or hyper) اضطراب الحركة

☞ **Apraxia**: inability to understand, plan or execute a complex motor act. It follows a lesion to the **cerebral cortex** affects the ability to conceptualize the task. **It's not dyskinesia** .

- **A chemical change in Parkinson's is decrease in dopamine to ACH ration. (↓DA /↑ACh).**
- **Treat ↓DA by L-dopa , as dopamine can't penetrate BBB.**
- **Treat ↑ACh by anti-cholinergic drugs**

- **<u>Amantadine</u> can do both actions (↑DA & ↓ACh).**

- **L-dopa improves hypokinesia .**
- **The anti-cholinergic agents decrease the rest tremors**.



ᘐ The **relation between basal ganglia and the limbic system** :

- A part of Basal ganglia plays a role in the limbic system (emotions , motivations, affective behaviours & memories)
- The anterior part (head) of caudate incorporates with putamen to form **<u>nucleus accumbens that</u>** highly receives dopamine.
- The motor part of striatum (direct and indirect) is putamen while the caudate has a cognitive function (i.e. practiced movement is well learned).
- **To sum up: basal ganglia participates in emotions, motivations, affective aspects of behaviour, memories and cognition**.