

BASAL GANGLIA

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BASAL GANGLIA

PHYSIOLOGICAL
ANATOMY

FUNCTIONS

DISORDERS

- Components
- Connections

Parkinsons disease
Chorea & Athetosis
Huntingtons disease
Wilson's disease
Kernicterus

➤ Basal Ganglia

is a group of nuclei (mass of *Grey Matter*) in the forebrain and upper part of brainstem that have **motor function** of great importance.

BASAL GANGLIA

CAUDATE

SUBSTANTIA
NIGRA

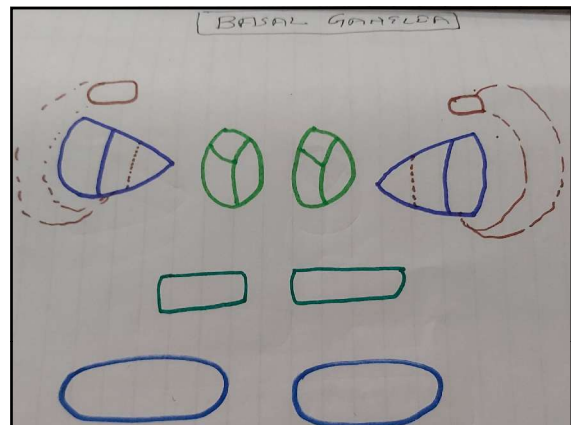
PUTAMEN

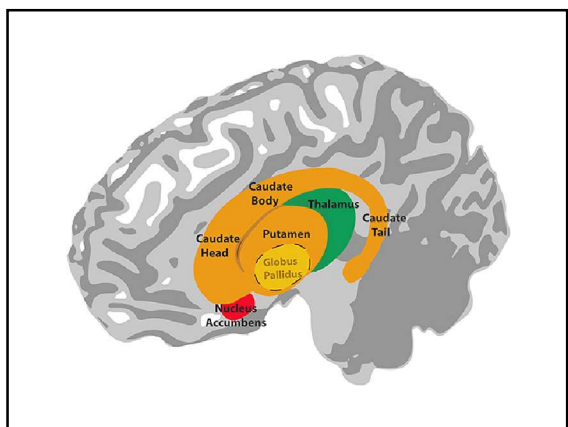
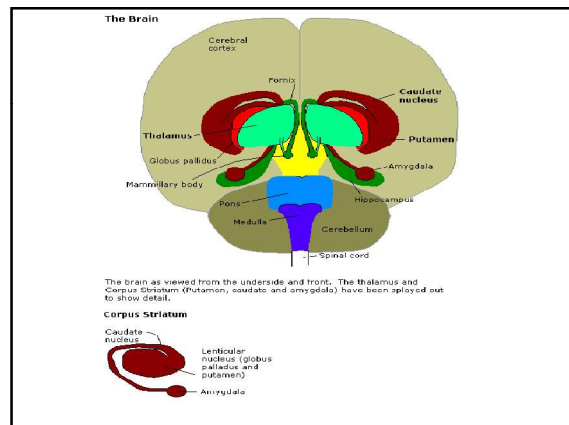
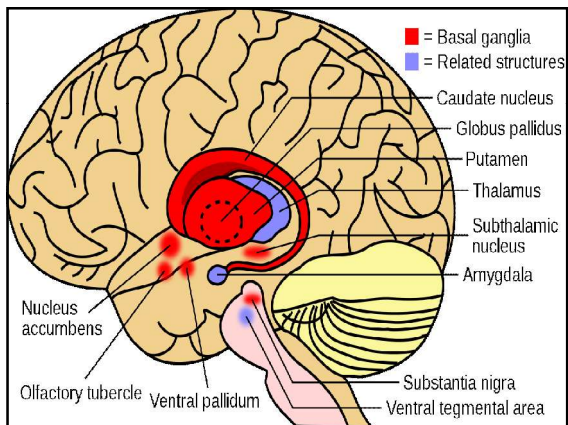
SUBTHALAMUS

GLOBUS
PALLIDUS

• WHAT IS

- Corpus Striatum ?
- Striatum ?
- Lentiform Nuclei ?
- Neostriatum ?
- Paleostriatum ?





CORPUS STRIATUM.

- Subcortical masses of grey matter situated in white core
- Divided into 2 parts by internal capsule.
 - **Caudate nucleus**
 - **Lenticular nucleus.**
 - Putamen
 - Globus pallidus.

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Salient Features of Nuclei of Corpus Striatum.

- Caudate nucleus.
- Highly curved, comma shaped band of grey matter.
- Consists of Head, body & Tail
- Separated from lenticular nucleus by internal capsule

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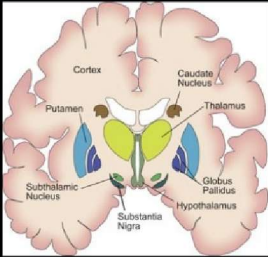
Lenticular Nucleus.

- Biconvex lens.
- Triangular in both coronal & horizontal sections.
- Divided into 2 parts by external lamina of white matter.

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Putamen

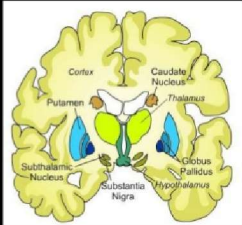
- Outer part of lenticular nucleus.
- Dark in colour
- Quadrangular in shape.



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Globus Pallidus.

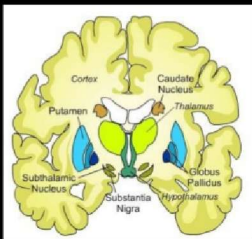
- Inner smaller part.
- Paler.
- Divided into 2 parts
 - External segment.
 - Internal segment.



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Sub Thalamic Nucleus.

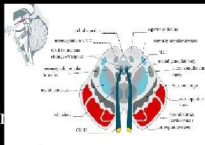
- Biconvex mass of grey matter lateral to red nucleus & dorsal to substantia nigra.
- Separated from thalamus by Zona inserta.



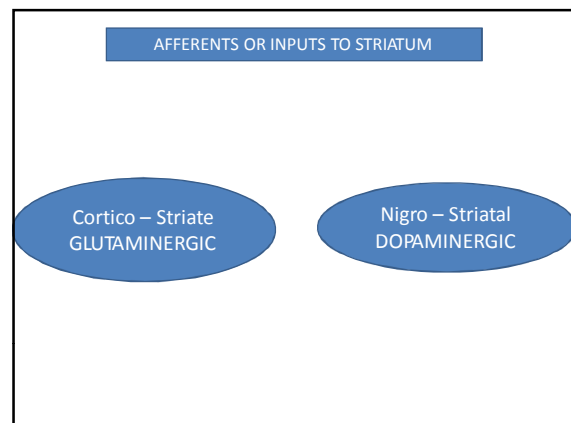
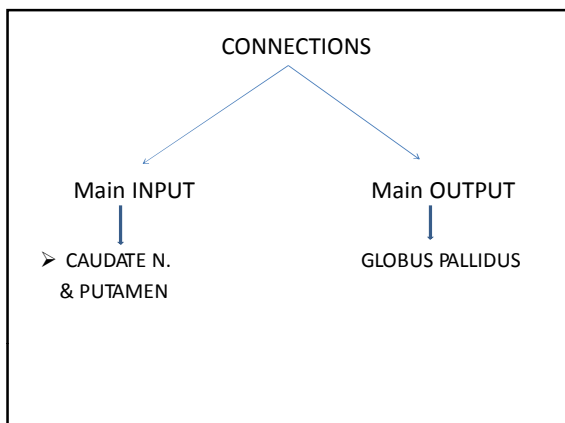
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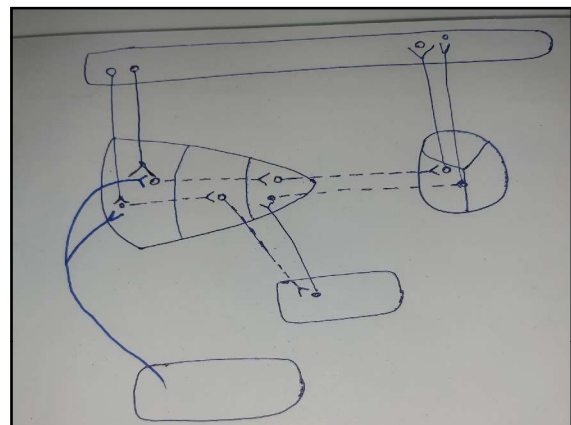
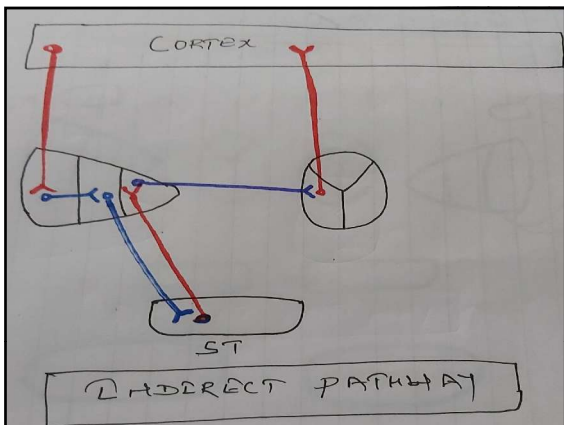
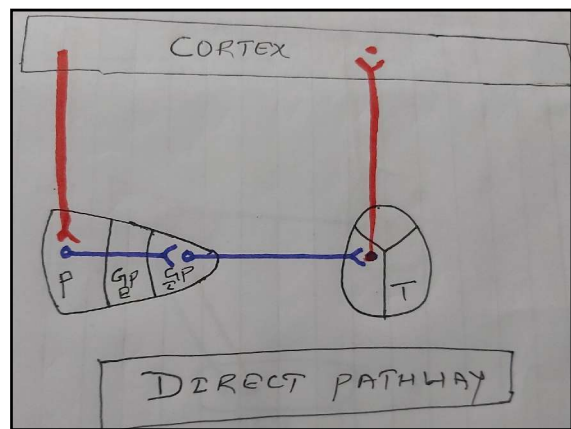
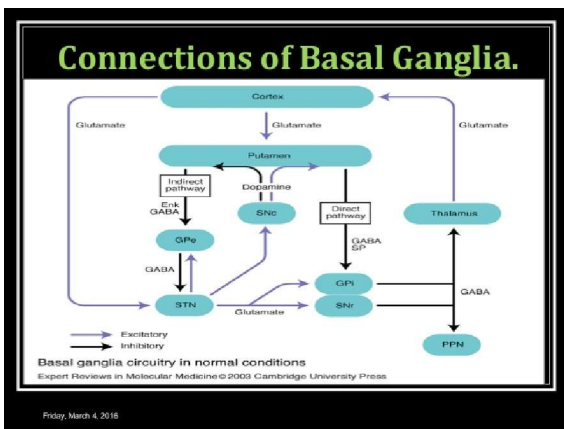
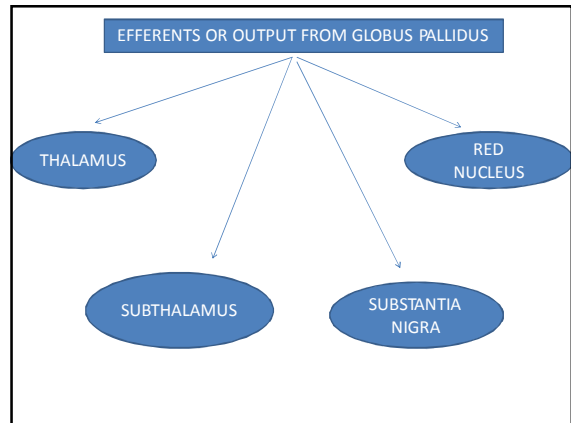
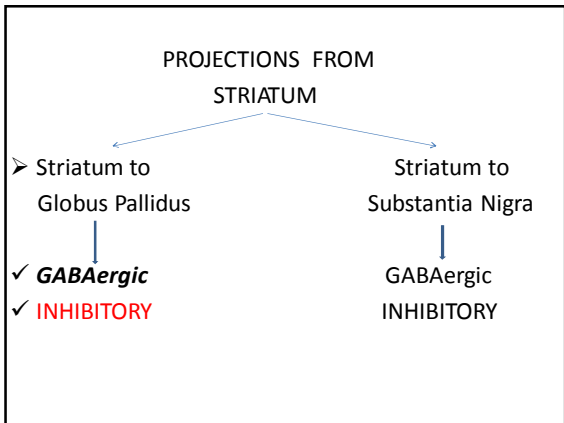
Substantia Nigra.

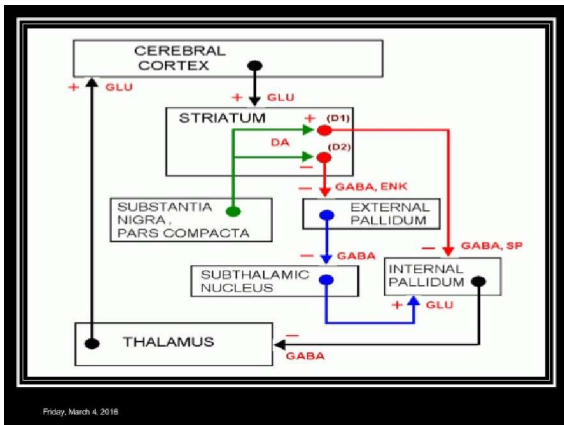
- Made up of small unpigmented & large pigmented nerve cells.
- Contains neuromelanin.
- Divided into 2 parts
- **Pars compacta** – contains dopaminergic (75%) & cholinergic (25%) neurons.
- **Pars reticularis** contains GABAergic neurons.



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Functions of basal ganglia.

- Control of voluntary motor activity.
- Control of reflex muscular activity.
- Control of muscle tone.
- Role in arousal mechanism.

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Control of voluntary motor activity.

- Cognitive control of motor activity.
- Neural discharge in Basal Ganglia begins well before the movement begins.



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Control of voluntary motor activity.

- Most of the motor actions occur as a consequence of thought process in mind.
- So basal ganglia is involved in planning & programming of movements.
- It is executed through functional neuronal circuits.

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Timing & scaling of intensity of movements.



- How rapidly & how much large the movement should be.

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Subconscious execution of some movements.

- Swinging of arm while walking.
- Crude movements of facial expressions with emotions.
- Movements of limbs while swimming.
- Importance – cortex can be free to plan its actions.

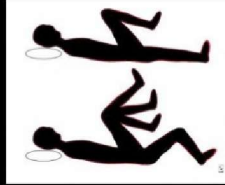
Pathway- Putamen feedback circuits.



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Control of muscle tone.

- **Substantia Nigra** of Basal Ganglia control **γ motor neuron** which maintain muscle tone.
- **Pathway** - cortical inhibitory area- striatum-pallidum-substantia nigra-reticular formation- spinal cord.
- **Lesion** - Lead pipe type Rigidity in Parkinsonism.



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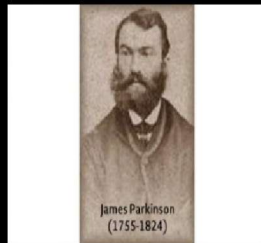
Disorders of Basal Ganglia.

- Parkinson's disease.
- Chorea.
- Athetosis.
- Huntington's disease.
- Hemiballism.
- Wilson's disease.

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Parkinson's disease

- Paralysis agitans or shaking palsy.
- Described by **James Parkinson in 1817.**



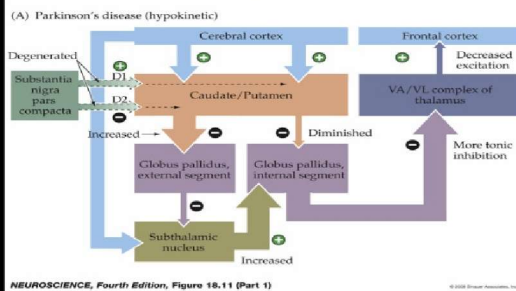
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Aetiopathogenesis.

- Primary idiopathic condition.
- In elderly due to **Idiopathic degeneration of Nigrostriatal system of Dopaminergic Neurons.**
- Secondary causes- Parkinsonism nigra.
- Viral Encephalitis.
- Cerebral Arteriosclerosis.
- **Drugs** - Phenothiazines.
- Experimentally by injecting MPTP (methyl-phenyl-tetrahydropyridine)

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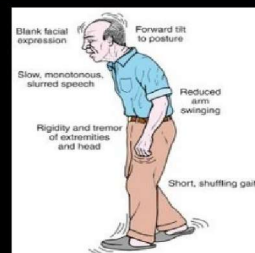
Pathogenesis.



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Clinical features. Akinesia or Hypokinesia.

- Unable to initiate voluntary movements or decreased movements.
- **Causes** - Due to Hypertonicity of Muscle.



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Clinical features

- C/F - **Bradykinesia**, **mask like face**, prolonged reaction time,
- Absent associated movements.
- **Shuffling or Festinant type gait.**
- Retropulsion.



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Rigidity

- Increased tone in the muscle.
- Cause - increase discharge of γ Efferents to muscle spindle.
- **Mechanism** - striatum under influence of both Ach(excitatory) & Dopa (Inhibitory)
- Degeneration of neurons of SN - less Dopa & more Ach activity - hyperkinetic features.
- C/F - lead pipe & cog-wheel type rigidity.
- Hypertone in protagonists & antagonists muscle.
- Statue like appearance.
- **Posture** - flexion attitude.

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Tremors

- Involuntary rhythmic oscillatory movements of distal parts of limb & head.
- Resting tremors.
- Absent at sleep & increased by stress & excitement.
- 4-6 times/sec.
- Frill rolling movements.
- Neural mechanism.
- Due to pacemaker activity in nucleus ventralis intermedius of thalamus.

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Treatment.

- **L-Dopa**
- As it crosses BBB.
- With Carbidopa as it prevents its conversion to dopamine in liver.
- Low dose - reduces rigidity & high dose reduces tremors.
- Surgical Destruction.
- Of Globus Pallidus or VLN of thalamus.

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Chorea.

- Rapid, jerky involuntary movements (dancing)
- Due to damage to caudate N.
- Seen in children as a complication of Rheumatic fever.



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Athetosis.

- Slow, rhythmic, twisting, worm like, confluent writhing movements of the extremities
- Mainly fingers & wrist.
- Due to damage to Putamen.
- Seen in children after birth injuries.



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Huntington's disease.

- Genetic disease.
- Trinucleotide repeat expansion.
- Autosomal Dominant disorder.
- 30-50 yrs of age.
- Abnormal gene at short arm of chr-4.
- Lesion – damage to GABAergic & cholinergic neurons of striatum to pallidum.
- Hyperkinetic features.
- C/F – hyperkinetic choreiform movements, Slurred speech, dementia, jerky trajectory of hand.

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Cause – damage of subthamic Nucleus commonly haemorrhage.

Hemiballism.

- So reduce output from GPiSNpc to thalamus.
- Disinhibition of thalamic output – hyperkinetic
- C/F – spontaneous attack of flail-like, intense violent movements of whole



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Wilson's disease.

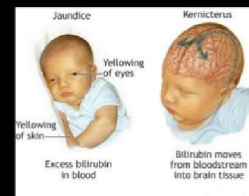
- Hepatolenticular degeneration by Cu toxicity due to impaired biliary excretion of Cu.
- Changes more marked in lenticular nucleus mainly Putamen.
- C/F – Parkinsonism, Akinesia, muscle rigidity & tremors.
- Ceruloplasmin level low & Cu content of SN high.**



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Kernicterus.

- In Hemolytic diseases of Newborn due to Rh antibodies -- **Raised indirect bilirubin** – crosses BBB – damages Globus Pallidus.
- C/F – Rigidity, Chorea, Athetosis & Mental Deficiency.



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