



Physiology of Basal Ganglia & Regulatory Mechanisms





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Recommended Videos!



Please check out this link before viewing the file to know if there are any additions/changes or corrections. The same link will be used for all of our work Physiology Edit

giving motor learned movment. Limbic loop motor loop expression inhibitory voluntary eye CIRCUITS Huntington's Disease movement motor intentions. Caudate Nucleus Occulomotor loop Hyperkinetic Cognitive loop Athetosis putamen Corpus striatum MOVEMENTS Basal exitiatory Lenticular Nuclei Hemiballismus Nucleus DISORDERS Globus pallidus Hypokinetic Subthalamic Nucleus SPEECH basal ganglia High oxgen Parkinson's Metabolic Substantia Nigra characteristics Control of movements POSTURE High Copper content FUNCTIONS Planning and programming of movements Cognition

Mind Map

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Basal Ganglia

♦Basal ganglia are subcorticle nuclei of grey matter located in the interior part of cerebrum near about base.

♦ Component Of Basal Ganglia:

Corpus striatum	Caudate Nucleus + Lenticular Lenticular nucleus " putamen + Globus pallidus "	Head of Tholomus
Striatum	Caudate Nucleus + Putamen	Globus palidus Lateral
Palostriatum	Globus Pallidus	Putamen
Subthalamic nucleus	Nucleus found below hypothalamus	Optic tract
Substantia nigra	Found in midbrain at all level.	Amygdoloid nucleus nucleus

\diamond Connections Basal of Ganglia :

• Main input to the basal ganglia:

The comes from the cerebral cortex (motor area) and projects to the

Neostriatum (a term for the caudate nucleus and putamen)

- Main output to the basal ganglia:
- Is via the thalamus to the cerebral cortex (motor area)
- Connections between parts of basal ganglia

WHICH WILL BE DISCUSSED NEXT



Basic Circuits of Basal Ganglia

- 1. Motor loop (Putamen circuit): concerned with learned movement.
- The input from: Premotor, supplementary motor and somatosensory cortex
- The output from: Primary motor cortex, premotor and supplementary motor areas.
- Putamen circuit give feedback to thalamus then they will go to primary motor cortex, premotor , and supplementary.

2. Cognitive¹ loop (Caudate circuit): concerned with cognitive control of sequences of motor pattern. Basically it is concerned with motor intentions. The cognitive loop Likened memory with movement.

- The input from: Association area
- The out put to : Prefrontal , Premotor and Supplementary Motor.
- If there damge to caudate circuit pathway they will be either: bradykinetic (slow movement) or akinetic (no movement).

- 3. Limbic loop : involved in giving motor expression to emotions like, smiling, aggressive or submissive posture.
- Occulomotor loop : concerned with voluntary eye movement [saccadic movement]

Brain steam is the one who control occulomotor loop

But all motor loop and all cognitive loop and part of limbic loop is concerned with basal ganglia

1: Cognition means the thinking process of acquiring knowledge and understanding through thought, experience, and the senses.

The Putamen Circuit

• Executes Learned Patterns of Motor Activity

- Basal ganglia function in association with the corticospinal system to control complex patterns of motor activity.
- Examples are:
 - ✓Writing of letters of the alphabet.
 - ✓Cutting paper with scissors.
 - ✓Hammering nails.
 - ✓ Shooting a basketball through a hoop.
 - \checkmark Passing a football.
 - ✓Throwing a baseball.
 - ✓The movements of shoveling dirt.
 - ✓Most aspects of vocalization.
 - ✓ Controlled movements of the eyes.
 - Virtually any other of our skilled movements, most of them performed subconsciously.





GODFY CONNECTION Think of Putaman like patman, we know that patman is a skilled and well taught super hero.. SO putaman circuits concerned with skilled learned movement..



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The Caudate Circuit

Cognitive Control of Sequences of Motor Patterns

Cognition means the thinking processes of the brain, using both sensory input to the brain plus information already stored in memory. Thoughts are generated in the mind by a process called cognitive control of motor activity.

Example: A person seeing a lion approach and then responding instantaneously and automatically by (1) turning away from the lion, (2) beginning to run, and (3) even attempting to climb a tree.

Thus, cognitive control of motor activity determines subconsciously, and within seconds, which patterns of movement will be used together to achieve a complex goal

Change the Timing and to Scale the Intensity of Movements

Two important capabilities of the brain in controlling movement are:

- To determine how rapidly the movement is to be performed and
- To control how large the movement will be.

Example: A person may write the letter "a" slowly or rapidly. Also, he or she may write a small "a" on a piece of paper or a large "a" on a chalkboard. Regardless of the choice, the proportional characteristics of the letter remain nearly the same



GODFY CONNECTION Cudate N looks like a rat (Head & tail) we know that rats when they see a piece of cheese they will think and plan a sequence of moves in order to get it. SO Caudate circuit concerned with cognitive control of

motor patterns..



Basal Ganglia Pathways Direct & Indirect

Direct Pathway "Excitatory Pathway"

1)Motor cortex send Excitatory (**^**Glutamate) input to St

2) St send inhibitory (**^**GABA) input to **Gpi**.

(3)Gpi sends less inhibitory (↓GABA) input to Thalamus.

Thalamus send excitatory(**†**Glutamate) input back to the motor cortex

5 The output of direct pathway is Excitatory = Increases motor activity.

Substantia Nigra and Dopamine involvement:

6 SNPC make connection with striatum by release DA at **DA receptor 1** which is excitatory.

St: Striatum Gpi: Globus Pallidus Internal DA: Dopamine SNPC: Substantia Nigra Pars Compacta SNPR: Substantia Nigra Pars Reticulata



Pathway

Direct

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Inhibitory NT here is : GABA Excitatory NT here is : Glutamate

Basal Ganglia Pathways Direct & Indirect

Indirect Pathway "Inhibitory Pathway"

(1) Motor cortex send Excitatory (**^**Glutamate) input to St.

2) St sends inhibitory (↑GABA) input to Gpe.

(3) Then Gpe sends less inhibitory (\checkmark GABA) input to STN.

4 STN project excitatory (**^**Glutamate) input to Gpi.

5 Gpi send inhibitory (↑GABA) input to thalamus.

6 The thalamus then send less excitatory (♥Glutamate) input back to motor cortex.

7 The output of indirect pathway is inhibitory = decreases muotor activity.

Substantia Nigra and Dopamine involvement:

8)SNPC make connection with striatum by release DA at **DA receptor 2** which is inhibitory.



very helpful

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Gpe: Globus Pallidus External STN: Subthalami Nucleus

Function 00 **Metabolic**

Metabolic Characteristics:

- High Oxygen consumption.
- High Copper content.

Wilson's disease (Copper intoxication):

Autosomal Recessive

Copper binding protein Ceruloplasmin is low

Lenticular degeneration occurs.

MORE ABOUT WILSON'S DISEASE

Wilson's disease, also known as hepatolenticular degeneration, is a rare genetic disorder caused by the buildup of copper. Small amounts of copper exist in the body and are released through the urine. In Wilson's disease, the copper cannot leave the body so it accumulates in certain organs.

Neurological symptoms result from copper buildup

- Memory impairment
- Gait disturbance
- Vision impairment
- Psychosis
- Tremor



\diamond Functions of Basal Ganglia :

- Control of movements
- Planning and programming of movements
 - Cognition

Basal Ganglia Disorders

Will affect:

Movements

(ataxia rate, range, force, direction)

(In parkinson's disease the hand writing will be Difficult and accessory movement is decrease And difficult to initiation and stop of movement)



Movement Disorder

Chorea

Huntington Chorea

Features

Multiple quick, random

movements, usually

most prominent in the

appendicular muscles.

(Michael Jackson's dance)

Basal Ganglia Disorders

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Lesion

Atrophy of the

striatum

Diffuse

hypermyelination of

corpus striatum and

thalamus

Hemorrhagic

destruction of

contralateral

subthalamic n.

Hypertensive

patients

Degenration of

Substantia Nigra

Parkinson's Disease

Degeneration of dopaminergic nigrostriatal neurons (60-80 %).

Loss of DA \rightarrow Lose excitatory effect on direct, lose inhibition of indirect Overactive GPi and overactive STN \rightarrow increased inhibition of motor thalamus

\diamond Cause of parkison's could be by toxin like :

- Phenthiazines (tranquilizers drugs) .
- Methyl-Phenyl-Tetrahydro-Pyridine (MPTP). The oxidant MPP+ is toxic to SN.
- \diamond Features:
- oTremor
- Rigidity
- o Akinesia & Bradykinesia
- Postural Changes
- Speech Changes



Huntingtons Disease

Hereditory, autosomal dominant. **Causes:** The loss of GABAergic neurons at caudate & putamen, which will leads to <u>chorea.</u>

\diamond Features :

- $\circ\;\;$ Jerky movement of hands toward end of reaching an object.
 - \circ Chorea
 - \circ $\;$ Slurred speech and incomprehensive.
 - Progressive Dementia

MCQS

1- Which one is correct about metabolic characteristic ?

A. High o2 consumption

- B. High copper content in wilsons disease
- C. Copper binging protein ceruloplasmin is high
- D. Lenticular degeneration occurs

2- Which one is the cause of wilsons disease?

A. High copper

B. Low copper

C. High o2

D. Low o2

3- Which part of basal ganglia if effected in case of Parkinsonism ?

A. Striatum

B. Substantia nigra

C. Subthalamic neuron

D. Corpus striatum

4- Cognitive associated with what ?

A. Emotions

B. Voluntary eye movement

- C. Motor intention
- D. Learn movement

5- Which one of this part is not involve in basal ganglia ?

A. Thalamus B. Subthalamus C. Lenticular nucleus D. Corpus straitum

6- The main input to basal ganglia ?

- A. Substantia nigra
- B. Cerebral cortex
- C. Thalamus
- D. Globus pallidus

7- The main output to basal ganglia ?

A. Substantia nigra B. Cerebral cortex C. Thalamus D. Globus pallidus

8- Which one of these neurotransmitter is consider with inhibition ?

A. Glutamate B. Dopamin 1 C. Dopamin 2

D. Acetylcholin

1- List four disease that will happen in case of any part of basal ganglia is effected?

1-Chorea

2-Athetosis

3- Hemiballismus

4- Parkinsonism

2- Function of basal ganglia?

1-Control of movements2-Planning and programming of movements3-Cognition

3- Describe the direct pathway of excitation ?

Information send to **putamen** from **cortex** by corticostriatal fiber which release the glutamine to excitatory the fiber from putamen to **internal globus pallidus** to release more GABA. GABA in inhibitory the fiber from from internal globus to **thalamus** to release less GABA. Decrease GABA in thalamus cause stimulate for fibers from thalamus to **cortex** (thalamocortical neuron) to release the glutamine and excitatory the cortex -> increase motor activity

4- Describe the indirect pathway of inhibition ?

Information send to **putamen** from **cortex** by corticostriatal fiber which release the glutamine to excitatory the fibers from putamen to external

globus pallidus to release more GABA. GABA in inhibitory the fibers from external globus to **subthalamic nucleus** to release less GABA.

Decrease GABA in subthalamic nucleus cause stimulate for fibers from subthalamic nucleus to **internal globus pallidus** to release the glutamine in Gpi and stimulate fibers from internal globus pallidus to thalamus to release more GABA in thalamus .

Increase GABA in thalamus cause inhibition for fibers from thalamus **to cortex** (thalamocortical neuron) --> doesn't release the glutamine \rightarrow inhibition the cortex \rightarrow decrease motor activity

$\diamond \quad \textbf{Basal ganglia}$

is groups of subcortical neurons the effects of basal ganglia on motor activity are generally inhibitory

\diamond Function of basal ganglia :

starting ,stopping motor functions , inhibiting unwanted movement. It changes the timing and scales the intensity of movements

\diamond Basal ganglia circuit :

- 1. Putamen circuit is inhibitory "indirect connect to cortecx "
- 2. Caudate circuit is excitatory "direct connect to cortecx "

\diamond Lesions of the basal ganglia

produce effects on contra lateral side of the body Damage to basal ganglia does not cause paralysis. It's only results in abnormal movements

♦ Basal Ganglia Disorders:

Disorder	Features	Etiology
Wilson's disease	Lenticular degeneration occurs > Neurological symptoms	Metabolic: Copper intoxication
Chorea (Huntingtons)	Multiple quick, random movements.	Atrophy of the striatum .
Athetosis	Slow writhing movements.	Hypermyelination of corpus striatum and thalamus.
Hemiballismus	Wild flinging movements of half of the body.	Hemorrhagic destruction of contralateral subthalamic n.
Parkinsonism	Pill rolling tremor of the fingers at rest, lead pipe rigidity and akinesia.	Degenration of Substantia Nigra.

THANK YOU FOR CHECKING OUR WORK! BEST OF LUCK

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اللهم إني أستودعك ما قرأت و ما حفظت و ما تعلمت، فرده عند حاجتي إليه، إنك على كل شيء قدير، حسبنا الله و نعم الوكيل ...

