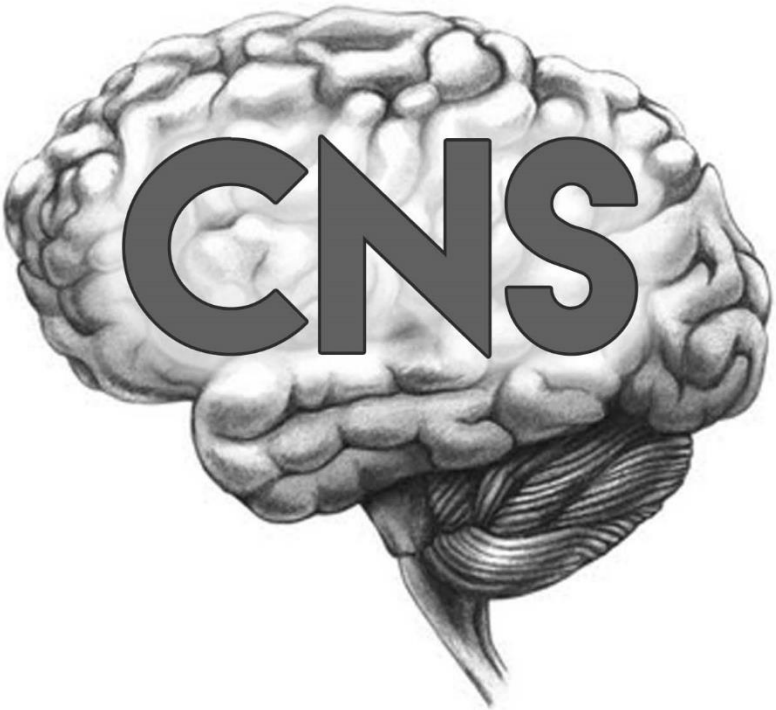

ANATOMY

☑ Sheet ○ Slide ○ Handout

Number

9

Subject

Cerebellum/Basal ganglia

Done By

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Date: 00/2/2017

This sheet was written according to the record of section 2.

The spinocerebellum and vestibulocerebellum regulate the movement after its occurrence, i.e. they influence actual movements. As for the cerebrocerebellum, it influences the idea and program of the move.

Cerebrocerebellum

This is the lateral part of the cerebellum.

Each part of the cerebellum has an input and an output.

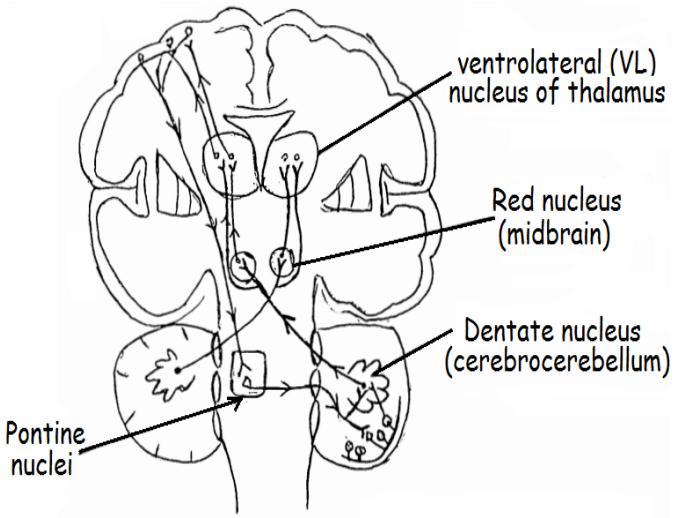
- **Input of the cerebrocerebellum**: cortico-ponto-cerebellar tract

- This tract starts from wide areas of the cerebral **cortex**, including sensory and motor areas, and most importantly from association cortices (meaning it brings information about the idea and program of the intended move).
- As it was explained before, certain parts of the cortex are concerned with the programming of movements; one example is area 6. So, it's not surprising that area 6 communicates with the cerebellum.
  Remember: To initiate a complex move you need: the lateral part of cerebellum, area 6, and the basal ganglia.
- This input synapses in **pons**, which means that fibers enter the cerebellum from the pons, forming the middle cerebellar peduncle (M.S.P).
  How was the middle cerebellar peduncle formed? From the axons of the pontine nuclei of the <u>opposite</u> side. This input enters the **cerebellum** with mossy fibers, synapse on granule cells, and then the granule cells synapse on Purkinje cells.
- As the fibers enter they excite the nucleus as usual. This input carries information about an intended movement, before its execution.

- Output: dentato-rubro-thalamo-cortical tract

The output is mainly from the **dentate nucleus**, and some from nucleus interpositus. Then it passes through the superior cerebellar peduncle (S.C.P) and goes to the **red nucleus**, and from there to the **thalamus** reaching the **cortex**. This tract sends its “reply” mainly to areas 6 and 4.

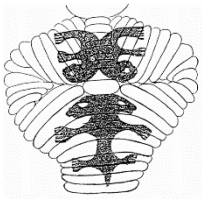
- This tract passes through the superior cerebellar peduncle, so any lesion in the S.C.P or part of the tract will cause the worst type of ataxia, why? Because it carries signals that correct the movement before its execution (it carries corrective signals). In contrast to the spinocerebellum which recognizes wrong movement then corrects it, but cerebrocerebellum makes the movement precise even before it is initiated (corrects it before it occurs).



- So what is the function of this part of the cerebellum?

1. **Planning the move** (we know that since it communicates with area 6).
2. **Initiation and timing** of the sequential movements. Typing is an example of this, where you need flexion that is followed by extension and then flexion again. Thus, it is important in the learning and storage of the sequential component of skilled movements.
3. **Regulates motor commands before the execution of movement** (feedforward), while the spinocerebellum and vestibulocerebellum act in a feedback mechanism.

How is the body represented in the cerebellum?



In the anterior lobe, the body is represented upside down. In the posterior lobe the body is erect. The central part, which is the vermis, represents axial muscles. As for the intermediate zone or paravermis, it represents the limbs and face.

What is represented in the lateral lobe? Nothing, as it is concerned with the planning and coordination of movements.

- The cerebellum is described as a **silent area**. If you put an electrode on it you wouldn't expect a movement in the hands or legs as you would expect in cerebral cortex stimulation. Also, the person won't feel a weird sensation, but if you put an electrode on a cortical sensory area the person will experience numbness and tingling.

That is all because the cerebellum doesn't communicate directly with the alpha and gamma. So, the cerebellum is called the silent area because its stimulation does not give rise to any sensation and causes almost no motor movement.

Lesions of the cerebellum

- Causes could include; CVA (cerebrovascular accident), tumor, trauma, inflammation.

- NO PARESIS OR PARALYSIS

- The effect is **ipsilateral**; lesion in the right cerebellar hemisphere will affect muscles on the right side of the body.

- Hypotonia: remember, hypotonia doesn't mean paresis or paralysis. A lesion in the cerebellum results in hypotonia in human beings, although it causes hypertonia in some animals.
- Disturbance of synergy: (coordination) the most important function of the cerebellum is the coordination between a muscle and its antagonist, for example; if you want to stimulate the biceps you have to inhibit the triceps. What is responsible for this? The pyramidal tract, under the control of cerebellum. Loss of this coordination or synergy is known as **asynergia**. Movement disorders also include errors in the range, force, rate, and direction of movements (those lead to asynergia).
- Loss of Equilibrium: inability to stand in an upright posture without support.

Vestibulocerebellar lesions

Anatomically, the vestibulocerebellum is the flucculonodular lobe, and phylogenetically it is called archicerebellum (the old cerebellum).

1. Disturbance of equilibrium; inability to maintain upright posture.

2. Staggering gait: tendency to fall towards the side of the lesion; if the lesion is on the right, the patient will fall on the right side because the cerebellum controls ipsilateral muscles.

3. Nystagmus: (jerky eye movement) the eyes go to one side slowly and then quickly move to the other side when trying to fix the gaze on a certain object. This is due to the loss of coordination between extraocular muscles, lateral rectus (which performs abduction, VI cranial nerve) and medial rectus (performs adduction, III cranial nerve).

- Lesion in vestibulocerebellum could be due to a tumor, called medulloblastoma that comes from the roof of the **fourth ventricle** which is close to the nuclei, and the most dangerous lesions are the ones affecting the nuclei of the cerebellum because the final output goes out from there. A patient with medullobastoma –usually a child- won't be able to stand without support, his trunk will be reeling from side to side, and he might also have nystagmus.

Spinocerebellar and cerebrocerebellar lesions

1. Disturbance in timing of initiation and termination of the movement. This includes:

- Dysmetria: the patient will either undershoot or overshoot (most commonly overshoot) when told to put two fingers tip to tip.
- Dysdiadochokinesia: inability to do two successive movements, for example; supination and pronation, or fast flexion and extension.

Again, dysmetria and dysdiadochokinesia reflect the absence of proper timing in initiation and termination of movement.

As you know, the antagonist muscle is the one that stops the move, so if there is a delay in activity of the antagonist, dysmetria occurs. While if there is a delay in the initiation of movement, dysdiadochokinesia occurs.

2. Intention tremor: reflects a disturbance in cerebellar functions. It is the opposite of resting tremor, which appears even when the person is not doing anything, and it reflects a disturbance in the basal ganglia (like in Parkinson disease). As for intention tremor, it is:

- Evident during purposeful movement
- Absent during rest
- Worst at the end of the purposeful movement
- Most commonly due to a lesion in the superior cerebellar peduncle because it corrects movements before their execution. (worst kind)

What causes this kind of tremor is that the normal control from the cerebral cortex to the cerebellum is lost here which disrupts cerebellar functions.

To test this, we ask the patient to put the tip of their finger on their nose and we will notice to and fro movement, until they finally touch their nose.

What is the cause of this to and fro movement?

- Loss of the damping function of cerebellum. What is damping movement?
  All of our voluntary movements carry momentum, think of it as extra force and excitation. This momentum is overcome by the cerebellum, as it sends appropriate signals which stop the movement at the required point and prevents overshooting.
- If the cerebellum is damaged, overshooting occurs. Then the cerebral cortex will recognize the overshooting (by seeing and feeling it) and initiate a movement in the opposite direction to bring the moving part – the finger – to the intended position. But, again and due to its momentum, the finger will oscillate beyond the intended point, and so on this will be repeated several times.
- This overshooting is called past pointing.

Again, in cerebellar diseases there is no paresis and no paralysis, but there's a worst effect; ataxia. Intention tremor is an example of it.

3. Hypotonia

Hypotonia appears mostly in the acute stage of cerebellar diseases, and it occurs in the following manner:

- Stretch reflex is responsible for the tone, and the gamma is especially important in this because it is stimulated faster and works for longer periods. If gamma is not working properly, the tone will either decrease (hypotonia) or disappear (atonia).
- Normally, there's a control from the cerebellum on the cerebral cortex. And there's control on the alpha and gamma from the cerebral cortex. So, if the cerebellum didn't function well, then the cortex won't drive the alpha and gamma properly, which results in an improper stretch reflex leading to a decrease in tone –**hypotonia** –, which might be represented as a **pendular jerk**.
- When you hit the patellar tendon of the patient for example, the normal reflex will be caused by the contraction of the muscle, and then it'll relax and return to its original position due to the tone. So, if the tone is absent the leg will move back and forth by its weight, like a pendulum.

4. Speech disturbance (dysarthria)

This is caused by the incoordination between the muscles of speech; respiratory, palate and tongue muscles.

The dysarthria caused by a cerebellar disease is called **staccato speech**; here the speech is slurred and explosive, also called telegram speech, as the patient will pause after every word or so.

5. Cerebellar ataxia

A movement disorder that occurs due to cerebellar diseases is called cerebellar ataxia, and it's due to the lack of coordination between agonist and antagonist muscles.

The term ataxia, clinically, is used to describe gait disturbances, which appear mostly as a staggering walk.

- There are two types of proprioception: one from muscles and joints and reaches the thalamus and cerebral cortex, and you feel it (like the position of your knee). This is called **conscious** proprioception.
- The second type is **unconscious** proprioception, we don't feel it, and the signal only reaches the cerebellum.
- The dorsal column system (posterior part of the white matter of the spinal cord) delivers conscious proprioception. A lesion in the dorsal column/medial lemniscus system which brings conscious proprioception leads to **sensory ataxia**. Those patients can't sense their joints and won't know, while they're walking for example, how high they lift their leg and will return it to the ground with great force, this is referred to as **stamping gait**.

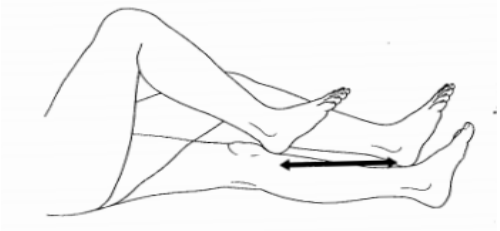
How to differentiate between sensory and cerebellar ataxia?

- Romberg's Test:
  Here we ask the patient to stand with his or her feet together and assess their stability.
  Then we ask the patient to close their eyes.
  - Patients with cerebellar lesion are ataxic in the same degree whether their eyes are closed or opened.
  - Patients with sensory ataxia are slightly ataxic on a narrow base when their eyes are open, but they fall when their eyes are closed (positive for the test). They compensate with vision.

Although some books say that it is difficult to differentiate between spinocerebellar (anterior lobe) and cerebrocerebellar lesions, some symptoms may be related to one type more than the other.

Anterior lobe lesion (syndrome):

The most common cause of lesions in the **spinocerebellum** is chronic alcoholism, which causes damage to Purkinje cells of the anterior lobe and the disturbance will be mostly in the **lower limb**. The patient has an ataxic gait, walks as if drunk. Malnutrition, which results from alcoholism is also thought to be related to this effect.

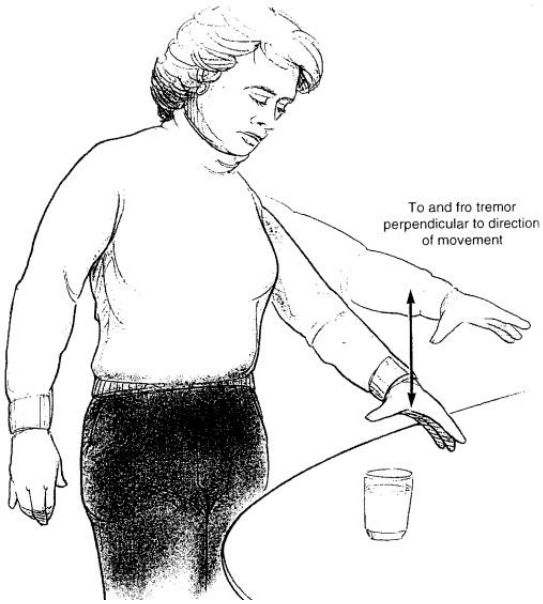


- This is tested by the **heel shin test**: we ask the patient to slide the heel of one foot smoothly down the shin of the other leg. In anterior lobe lesions the leg will move to the left and right, indicating a positive test.
- If the lesion grew bigger to include the posterior lobe, you will start seeing posterior lobe lesion symptoms: dysmetria, intention tremor, staccato speech, etc.

Posterior lobe lesion (syndrome):

The most affected here are the **upper limb** muscles. The cause can be CVA, inflammatory, tumor, degenerative diseases, or trauma, but it's commonly vascular (embolic, thrombotic or hemorrhagic). So here we will notice ataxia in the upper limb.

- This is tested by **intention tremor test**: Finger-nose test, or asking the patient to grab a glass of water (the hand will move up and down right before reaching the needed object). *Remember*: this is caused by loss of the damping function of the cerebellum.
- Again, if the lesion grew bigger more symptoms appear: dysmetria, staccato speech etc.



The Basal Ganglia

- The basal ganglia and cerebellum are important for the programs of movement.

- Basal ganglia participates in the initiation and control of movements. And so, a lesion here will cause no paresis or paralysis, but slowing in movement (initiation, continuation or termination). Also, most of its effects are on axial and proximal muscles, which are concerned with posture.

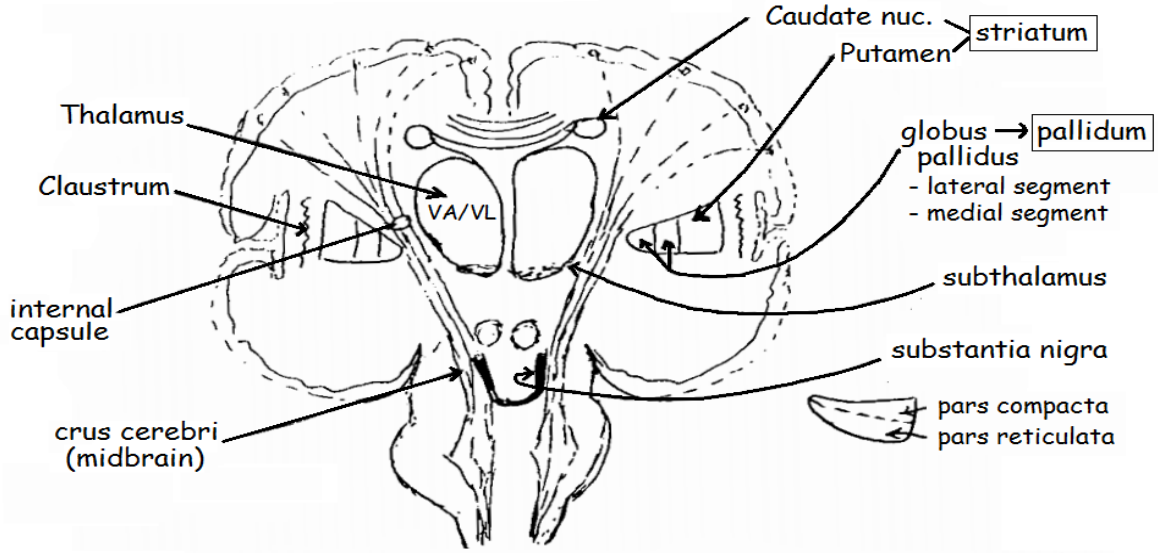
- From this we can conclude some of the signs of an advanced lesion in this area, for example those of Parkinson disease include: hesitation to move, slow movements, bent posture, and others. Again, no paresis and no paralysis.

- It works to some extent like the cerebellum, by a feedback mechanism: receives signals and replies to them. Receives signals from reticular formation and other tracts, then sends them till they reach the alpha and gamma (no direct contact). *Note*: the cerebellum and basal ganglia do not communicate directly with the alpha and gamma, but with upper systems/pathways.

- It has a reciprocal relation, meaning the basal ganglia receives input from cerebral cortex and replies to it.

Basal ganglia components

- **Corpus striatum**: (الجسم المخطط) this includes the caudate, putamen, and globus pallidus. It appears striated histologically in myelin preparations, masses of grey matter crossed by myelin fibers.
- **Amygdala**: part of the limbic system (emotion, motivation, behavior, memory).
- **Claustrum**: (الخط الرفيع) its functions and connections are not well understood.



Corpus striatum is described in two parts:

- **Paleostriatum/pallidum** (the relatively old part): it is the globus pallidus, which is part of lentiform and is medial to putamen. Globus pallidus has an external and an internal segment (lateral and medial) and both segments differ in their functions and connections.
- **Neostriatum/striatum**: it is the caudate and putamen, they are anatomically separated but related functionally. This is the entrance gate to the basal ganglia, meaning inputs (sensory, motor, association) from the cortex enter first through the striatum, and they are excitatory. The neurotransmitter here is **glutamate**, and thus those neurons are called glutamatergic.

- There are two nuclei that are outside the basal ganglia, but related to it functionally which are:

- **Subthalamic nucleus**, which is part of the diencephalon, anatomically.
- **Substansia nigra**, which is part of the mesencephalon, it has two parts:
  - Pars compacta: the posterior part, it synthesizes dopamine.
    A decrease in dopamine leads to Parkinson disease symptoms. *Note*: we don't give dopamine to those patients, since it doesn't cross the blood brain barrier (BBB), so we give L-dopa which crosses the BBB and is then converted to dopamine.
  - Pars reticulata: the anterior part, and has the same function as globus pallidus internal segment.

- The **cortico-strio-pallido-thalamo-cortical** loop: as mentioned earlier, the one that receives information and signals from the cortex is the striatum, then the way out from the basal ganglia is through globus pallidus internal segment. This signal then passes through part of the thalamus (VA and VL) and then to cortex.

- Motor commands come from the motor cortex, these commands are edited by the cerebellum and basal ganglia. The basal ganglia controls:

- The decision to move, for example if you ask a patient with basal ganglia disease to cross a line on the floor, they will take a very long time to perform that move, as they'll keep thinking and thinking since their decision machinery is disrupted.
- The motor expression of emotions (patient's face will be expressionless).
- Direction of movement.
- Amplitude of movement.

- Basal ganglia receives input from wide areas of the cortex (sensory, motor and association) through striatum, but it mainly replies to areas 4 and 6.

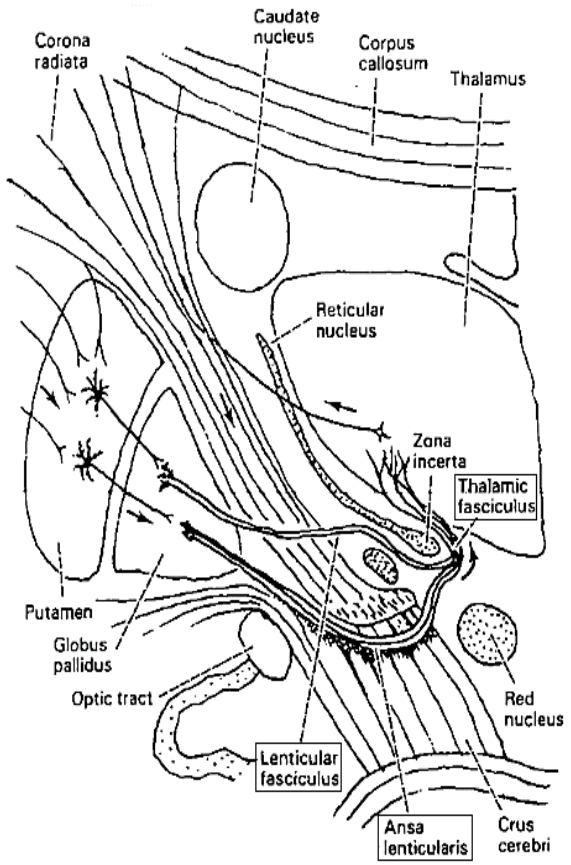
- The striatum contains two types of cells:

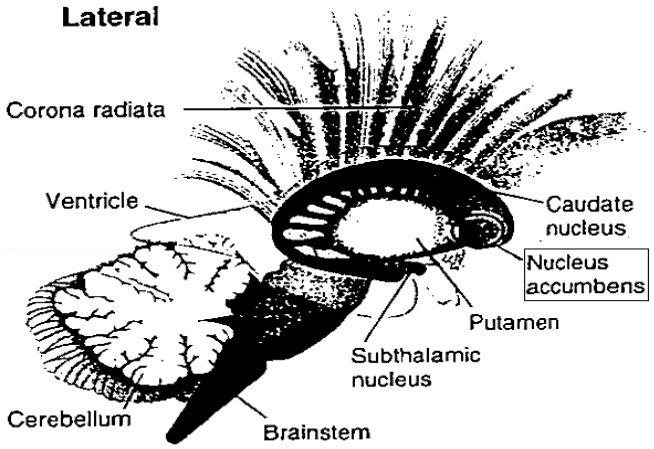
1. **Inhibitory, GABAergic**: the striatum sends signal to globus pallidus through GABAergic fibers, called striopallidal fibers. Then globus pallidus sends inhibitory fibers to part of the thalamus (VA and VL), which will send signals back to the cortex.
   The thalamus usually sends *thalamocortical fibers* activating the cortex, so if the thalamus is severely inhibited, the signals won't reach the cortex, and the movement will become slow. The opposite occurs when the thalamus is overactive, as the cortex will become overactive too, and there'll be more movement than needed.
2. **Excitatory, cholinergic**: discussed later.

- Between globus pallidus and thalamus are the **pallidothalamic fibers**, this tract is formed of two parts; lenticular fasiculus, and ansa reticularis. They run around the internal capsule and then connect together forming **thalamic fasiculus**.

- The anterior part of caudate and putamen fuse and form **nucleus accumbens**. It receives dopamine from pars compacta of substansia nigra. It plays a role in the limbic system.

- So, the basal ganglia doesn't only play a role in coordination of movements, but it is also involved in emotions, motivation, and memory (limbic system).





This sheet has been corrected and edited.