The cerebellum

Note: this lecture was given during the physiology lab and its considered the last theory lecture in physiology the record for which can be found under the title "physiolab"

introduction

The cerebellum is the second motor regulator, "the first being the basal ganglia" and just like the basal ganglion it's not just a motor regulator its rather a regulator for the cerebral cortex and the sub-cortex as a whole and the best way to describe its function is by calling it the statutory auditor of the cortex for example if the cortex was to give an order "X" the cerebellum audits this order to make sure that it is **the motor intention** and that it shouldn't be "1\2X or 2X…etc".

A very easy example to understand the cerebellar function in motor regulation is trying to teach a child how to hold an egg, when the child tries to hold the egg he will either use too much pressure breaking the egg or too little dropping it.

what happens is that the cortex gives a **motor output** "X" and when the child holds the egg too hard breaking it the **sensation** he will get is that he used too much effort and here the cerebellum makes an adjustment that we need to reduce the motor output to "1\2X or 3\4X…etc" to hold the egg without breaking it, the child tries again and this time he drops it so the cerebellum makes another adjustment increasing the motor output, he tries a third time breaking It so the cerebellum reduces the motor output again and this goes on and on until finally the child learns the exact amount of pressure needed to hold the egg and the cerebellum records this amount so the next time the child tries he succeeds. Another example is basketball players getting better at shooting the ball into the basket with the more they practice.

Input and output of the cerebellum:

Input

1. **direct "non-proccesed"**: from the receptor to the cerebellum directly such as PCML modalities via the spino-cerebellar tract and proprioception via the vestibular tract "directly from the labyrinth to the cerebellum".
2. **semi-proccesed information:** these include a second order neuron before reaching the cerebellum such as PCML modalities via the cuneocerebellar tract and proprioception via the vestibular tract "from the vestibular nucleus in the brain stem to the cerebellum".
3. **proccesed information:** they come from the cortex and include PCML and somato-sensory from the primary somato-sensory cortex, visual/auditory* secondary and association cortex, prefrontal association cortex, posterior parietal association cortex and all the motor cortex "primary, secondary and association".

*If vision was to be received unprocessed it would be useless since the cortex is the one that does most of the visual processing and not the cerebellum for example if u look at a certain distance what helps u determine its 2 meters and not 1 is the cortex or if keys are thrown at you what helps u calculate the speed of the keys and where you should put your hands to catch them is also the cortex and not the cerebellum and the same applies to the auditory information and that's why the cerebellum receives these information after they get processed in the cerebral cortex.

Output

1. primary secondary and association motor cortex : it audits all types of motor output.
2. association cortex : it audits all types of cortical output.
3. extra pyramidal tracts : it audits unconscious movements.
4. thalamus motor nuclei and the hypothalamus: it audits the limbic system and the autonomic nervous system output.

motor functions of the cerebellum:

As mentioned previously the cerebellum audits the motor output whether it be conscious or unconscious, voluntary or involuntary and since the biggest aspect of involuntary movement is regulation of body balance the cerebellum contributes to **controlling the body's equilibrium**, the cerebellum also contributes to the

regulation of muscle tone and that's why a lesion in the cerebellum will cause hypotonia, finally the cerebellum contributes to the **coordination of movements** so a lesion in the cerebellum will cause ataxia "cerebellar ataxia"

Cerebellar ataxia: the main symptoms of which can be summarized in the following:

1. Disorders of equilibrium – patient can't stand:

If someone was tilted by 30 degrees and is about to fall the vestibulospinal system used to sense this deviation and it used to send sensory input so that the body can respond and fix this tilting before the person falls, and since the cerebellum is the memory storage house that stores information such as what exact motor output the body needs to exert incase its tilted by 30 degrees and which muscles should respond, if the cerebellum is damaged the body ends up exerting the wrong amount of motor output so the person ends up losing balance and falling.

2. Attactic gait – patient can't walk:

With each step we take there's shifting of body weight to the foot that's touching the ground and this happens through the vestibulospinal system under regulation of the cerebellum so if the cerebellum is the damaged the patient won't be able to shift the weight properly so as to avoid falling they will increase the distance between their legs while walking, the patient will also try to keep the tilting to a minimum so when these people walk they look like penguins.

3. Intention tremor – dynamic tremor (it is more expressed while moving and disappears while rest):

When you try to reach out for something the cerebellum gives you the exact amount of output needed to reach the object your going for however if the cerebellum was damaged the cortex will give multiple outputs all of which are wrong until eventually reaching the correct output and reaching the object thus resulting in a dynamic tremor.

4. Dysmetria (disturbed ability to gauge distances).

When the patients tries to give a certain output they will either give an exaggerated one or an underwhelmed one, for example when they try to throw the keys to someone they will either throw it too close or too far away.

5. Dysarthria:

Since the cerebellum regulates all kinds of voluntary movements including the corticobulbar and corticospinal tracts that go to the speaking muscles, ataxia patients will have some dysregulation in speaking*.

6. Nystagmus:

Caused by Problems in the regulation of eye movements.

7. Dysdiadochokinesia (Awkward performance of rapid alternating movements).

Functional division of the cerebellum:

There are 2 types of divisions for the cerebellum the first one is “anterior to posterior” dividing it into an anterior lobe, a posterior lobe and a flocculonodular lobe the second is “medial to lateral” dividing it into the vermis , the paravermis and the lateral cortices and according to these divisions we can divide the cerebellum into 9 zones each with its own function but since these zones overlap in their function greatly we usually study them as groups of zones for example:

1. the vermis of the anterior lobe is associated with the core midline muscles so if there is a problems in it we will notice midline muscle deficits such as eye problems, facial muscle problems, Dysarthria and **truncal ataxia**.

2.the paravermis of the lateral anterior lobe and the paravermis of the posterior lobe are mostly associated with the limbs so problems In them will present as **limbic ataxia including tremor and hypotonia** .

3. the lateral cortices are directly connected with the higher association cortex such as the prefrontal and the posterior parietal so damaging them will cause

A. problems in **the complex calculations of movements**
B. a more prominent appearance of **Dysdiadochokinesia** since the posterior parietal is involved in sequential processing of movement *

The limbic system

A group of subcortical nuclei that are highly connected with each other. In the past it was thought that they were one circuit doing the same function but now they are divided into multiple circuits the most important of which are:

1. hippocampal-diencephalic-retrosplenial network which connects the hippocampus with parts of the diencephalon “thalamus and hypothalamus” and finally with a cortex found in the posterior wallspelnium of corpus callosum.

2. temporal-amygdala-orbitofrontal network which connects the temporal cortex with the amygdala and finally with the lower part of the prefrontal cortex “orbitofrontal” cortex.

3. medial “default” network which connects most of the structures of the medial plane “ medial side of the cortex”

Function of the limbic system

First function: memory

Through the hippocampal-diencephalic-retrosplenial network.

We are exposed to a huge amount of sensations and information daily and we only form long term memories to a specific set of information that we deem important enough to be stored and so the function of this circuit is to determine which information should be stored and to physically form a long term potentiation **thus forming long term memories** this network also regulates the working memory “short term memory” determining which ones to keep active And so damage to this circuit will cause problems in memory both working and long term and is associated with **Alzheimer's disease, Amnesias and Korsakoff**

syndrome.

1. Alzheimer's disease: due to decrease in acetylcholine in this circuit which happens before it happens in other parts of the cortex and that's why memory problems is the first manifestations of this disease.

2. Amnesias: inability to form long term memories because of damage to this circuit, the patent would be able to access past memories but won't be able to form new ones and one of the most famous examples of this is the case of Henry Gustav Molaison (H.M.) a young male in the 1950s who had epilepsy in the temporal lobe and as a result had his temporal lobe removed on both sides including the hippocampus, he lived for 45 years after the surgery and he never formed a single new **declarative memory** but he was able to gain new **undeclarative** memories since they are unconscious.

Note: The main cause of H.M symptoms was the removal the hippocampus a part of the hippocampal-diencephalic-retrosplenial network.

Note: for the amnesia to happen the hippocampus needs to be damaged on both sides however since the hippocampus has a high demand for O2,blood supply and glucose a transient hypoxia even if it doesn't cause a complete stroke will damage the hippocampus and sometimes even a short severe episode of hypoglycemia will damage it

3. wernicke encephalopathy: acute thiamine deficiency will cause problems in the function of the hippocampal-diencephalic-retrosplenial network causing problems in the selection of memories "choosing which short memories to be active" so the symptoms will include: confusion, ataxia and ophthalmoparesis "weird movements of the eyes"

4. **Korsakoff Syndrome:** chronic thiamine deficiency will cause Neuronal degeneration in the mammillary body, dorsomedial thalamic n. & hippocampus. Degeneration or lesion in this pathway is often seen in chronic alcoholics as then will have degeneration especially in the mammillary bodies, and it will lead to both short- and long-term memory loss and it will also cause Confabulation " the production of fabricated, distorted, or misinterpreted memories" i.e: they will put together pieces of stories randomly because the selection of short and long term

memories are disrupted so when you ask them what did u do this morning they'll say I woke up than I had dinner with my family.

Second function: spatial orientation:

Through the hippocampal-diencephalic-retrosplenial

spatial orientation is a function done by a special type of cells known as place cells found in the hippocampus each place cell is associated with a certain location for example there's a place cell for your room a place cell for the roof of the house a place cell for the dark corner you go to cry in when the exam is the next day and u have so much left, place cells activate long and short term memories related to a certain location when you go to that location for example when you go to your room place cells activate memories related to your room.

temporal-amygdala-orbitofrontal network

the orbitofrontal is the lower part of the prefrontal cortex and as we took before the prefrontal is responsible for the selection and consequences of behavior and we also took a list of symptoms in case there was a damage in it such as stubbornness……etc.

this circuit is responsible for mediating all of the functions of the prefrontal cortex such as **Emotion processing** & **Motivation and behavioral selection** and that's why damage to this circuit will cause personality changes and other behavioral symptoms (e.g. aggression, disinhibition) such as Kluver-bucy syndrome, Psychopathy and Bipolar affective disorders.

in addition to all of this the amygdala has a role in **Multimodal "multisensory" integration** so it is connected to the higher sensation processing and higher meaning processing which includes the higher meaning of language and that's why this circuit is the one responsible for semantic processing "language processing".

"Semantic processing is the processing that occurs after we hear a word and encode its meaning." Wiki

A person who has a damage in wernicke's area will have semantic processing and will be able to understand “a little” what’s being said however if this circuit is damaged while keeping wernicke's area intact it won’t result in aphasia but it will result In a language deficit known as semantic deficits (semantic dementia)

Kluver-bucy syndrome:

is caused by a bilateral lesion in the amygdale, as we said before the amygdala has a role in Multimodal “multisensory” processing so a damage in the amygdala will lead to the loss of proper processing of sensation causing **Agnosia: visual, tactile & auditory** and since there’s no processing of sensation there will be no memory formation causing **Dementia** and in addition to that these patients will have a triad of symptoms consisting of **Hyperphagia** “over eating” Hyperorality “ insertion of inappropriate objects in the mouth” and Hypersexuality.

medial Default network:

connects many parts of the cortex found on the medial aspect such as the

1. Posterior cingulate cortex & precuneus
2. Medial prefrontal cortex
3. Angular gyrus.

We all have more than one personality and more than one way of thinking and this circuit is the one responsible for choosing which one we use as our default setting , for example if someone feels and thinks of himself as a “nerd” than his choices and actions would be based on that self image and this circuit would activate the areas of the cortex that would help processing for that purpose the same applies if someone has a self image as a “funny person” than this circuit would activate different areas in cortex for this different self image another example is that if someone wakes up and thinks im angry today im going to make decisions I wouldn’t normaly make because today I feel like im the angry guy the circuit would make changes and so on.

Damage to the default circuit is associated with some psychiatric disorders and some developmental disorders such as autism.