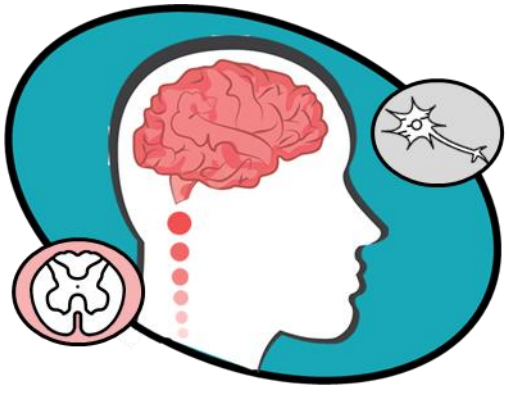

CNS

Physiology

Sheet Slide

Number

5

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CNS - Physiology

The Dr Started with the Diseases of the Vestibular System of the inner ear:

But Before that, we should understand the difference between Dizziness and Vertigo, Dizziness is a nonspecific, imprecise term of feeling weak,unsteady (Spatial disorientation) and loss of balance (Postural instability) and may be accompanied by nausea. Dizziness can be caused by Vestibular problems or other variety of problems.

However **Vertigo is specific for Vestibular Problems** that cause the patient to sense that he/she, or the environment around him/her is moving or spinning(Where there isn't real movement).

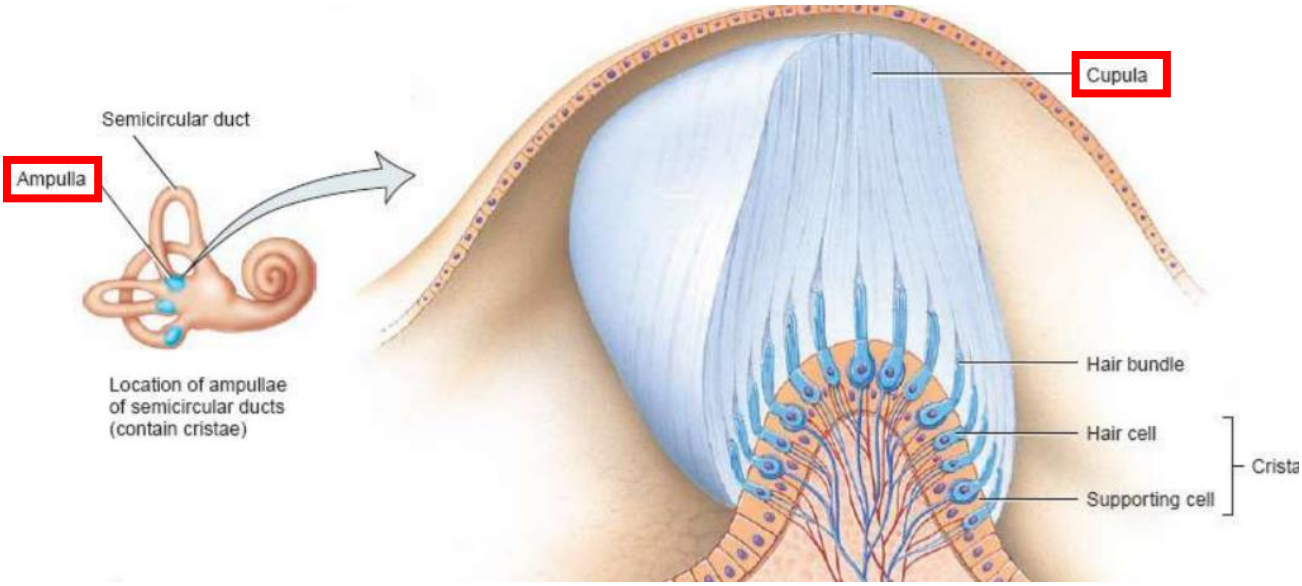
Additional Note: The Best way to imagine Vertigo is when we were children; we used to rotate around ourselves as fast as possible and stop suddenly, for a few moments the world seems to spin.

Benign Paroxysmal Positional Vertigo

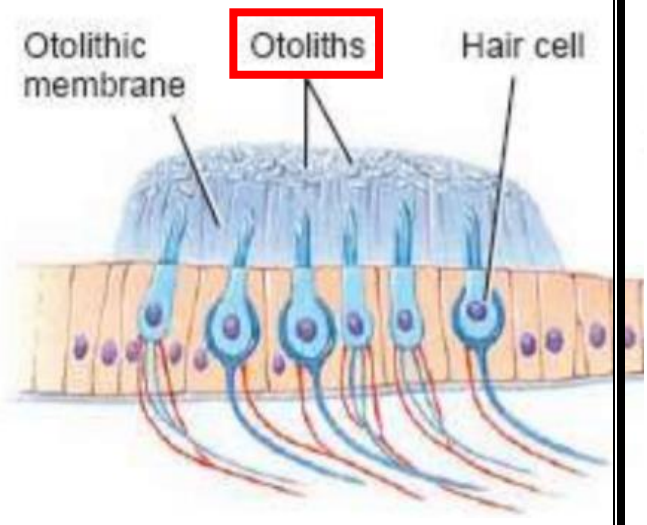
A common Vestibular Disorder (statistics say 20-22% of the population suffer from it). The pathophysiology is not well known, but a possible explanation suggests a defect in the Otoliths (Calcium crystals above the Otolithic membrane of the saccule and utricle) in which they fall out of place. Symptoms vary from Mild such as people who can't tolerate going to Amusement parks and they will throw up and suffer from head pain. Moderate cases include people who can't tolerate travelling or long driving journeys. Severe cases include episodes of vertigo after sudden movements and particular changes in body position such as getting up in the morning or turning over in bed which makes them fear such movements.

Notice that falling of the Otoliths has 2 consequences leading to this disease, firstly it decreases the sensitivity of the otilithic membrane to gravity making the movement of the otilithic membrane less than normal (as their goal was to make the otilithic membrane heavier and more sensitive to gravity) and one ear will differ from the other ; luckily ,

the brain can mostly adapt to this decrease in sensitivity and it won't be very severe. Secondly, if the Otilithic Crystal was Big enough, it can rotate in the vestibular system and may even close one or more of the semicircular canals! Preventing the movement of the fluid in the canals or the gelatinous membrane of the Cupula which is the cause of severe cases. (See below photos of the Otoliths and Cupula)



Luckily , Big Crystals of Otiliths dissolve after falling out from the otilithic membrane within 2 Weeks – 1 Month , that's why severe symptoms don't last forever , however there might be another incidence of a new large crystal falling out, creating another 2 Weeks- 1 Month of severe symptoms.

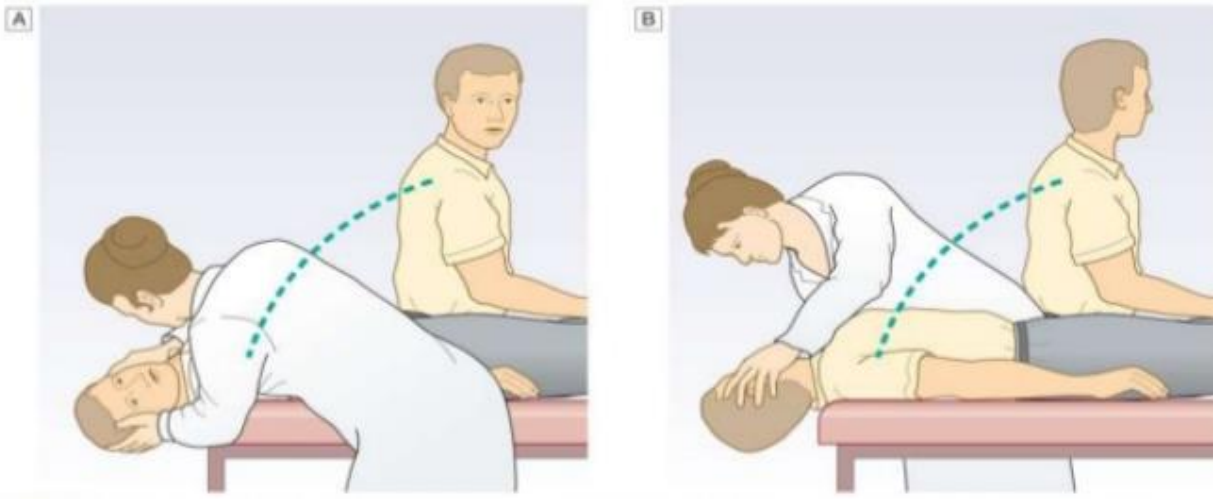


Dix-Hallpike test is the definitive diagnostic test for Benign Paroxysmal Positional Vertigo,And is also considered a method of ***Treatment***. The Procedure involves the following steps:

1. The patient sits on a bench relaxed
2. Turn the patient's head 45 Degrees to one side

3. Pull the patient suddenly to a Supine positionwith the head pointing 20 Degrees Posteriorly and Observe eyes for Nystagmus – And ask the patient if there are any rotational feelings
4. Pull the patient to a Sitting position again suddenly with head tilted in same way. Observe eyes for Nystagmus and ask the patient if there are any rotational feelings (A positive result includes a burst of Nystagmus)
5. Repeat same steps on the other side

Why is this procedure considered also a treatment? Because in severe cases, these steps can move the otilith crystal to the base of Inner Ear Labyrinth decreasing its effects on the semicircular canals. So we repeat these steps until the patient feels improvement.

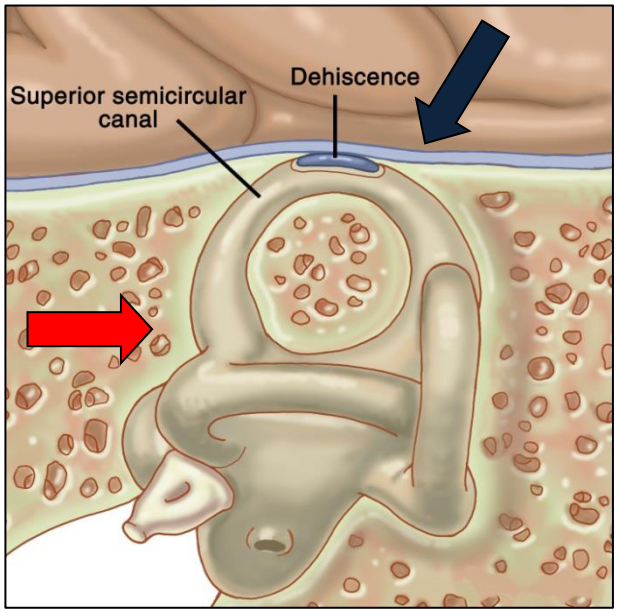


Vestibular Neuritis

A disorder caused by an infection to the vestibular branch (of vestibulocochlear nerve) or to the Vestibular ganglia which affects the conduction of information due to edema of the nerve or ganglion. (Mostly caused by an acute viral infection but can be any other infectious cause). Symptoms are severe vertigo and nystagmus but *usually there is no accompanying hearing loss (because the auditory - cochlear-part of the nerve is not affected) or CNS abnormalities.*

Semicircular Canal Dehiscence

We recall that the Inner Ear Labyrinth is the rigid, bony outer wall of the inner ear in the temporal bone which houses the vestibule, semicircular canals, and cochlea. Sometimes , Maldevelopment of the temporal bone above one of the semicircular canals (usually anterior/posterior) makes the canal only covered by the membranous layer without the rigid bony layer , and even maybe in more severe cases , the canal is totally open with no membranous or bony cover .This makes the fluid of the canals affected by the pressure of the extra-dural space or middle ear pressure. See picture below of a case of maldevelopment of the temporal bone above the superior semicircular canal with only a membrane coverage.

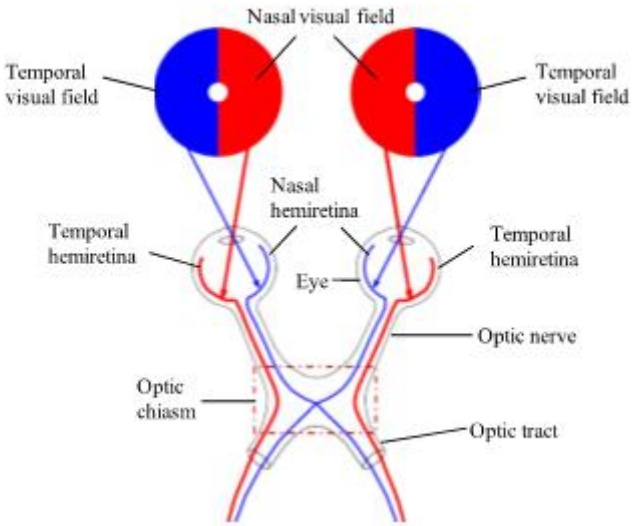


Symptoms include Veritgo, Nystagmus and Oscillopsia (a sense that objects are moving closer and further in the visual field). These symptoms are seen whenever there is a stimulus that affects the extradural pressure. (Red arrow resembles Middle ear pressure and Black arrow resembles extradural pressure).

One of the weirdest and most common stimuli is hearing loud noises (I think which increases pressure in the middle ear) causing vertigo and oscillopsia which is called the **Tullio Phenomenon**. Other stimuli involve increase in intracranial pressure (which increases pressure of the extra duralspace). Treatment is usually surgical by covering the opening dehiscence.

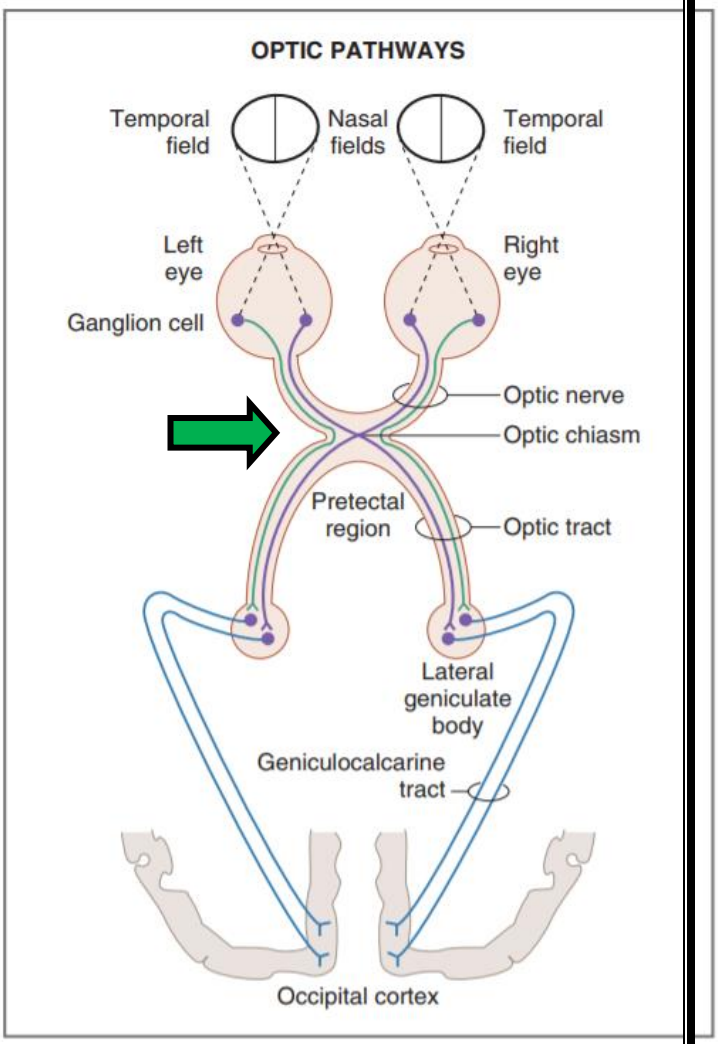
Deep Look:Visual Pathway

Let's start with some important basics: The Visual field is divided into temporal visual field (Blue) and nasal visual field (Red); also the retina itself is divided into Temporal Hemiretina(Red) and Nasal Hemiretina (Blue).



As seen in the photo,Notice that the **nasal hemiretina receives data from the temporal visual field (Contralateral) ,and the temporal hemiretina receives data from the nasal visual field (Contralateral)**.

After that , Axons from retinal ganglion cells form the optic nerves, which synapse in the lateral geniculate nucleus of the thalamus, and ascend to the visual cortex by the optic radiations geniculo-calcarine tract). (See photo)



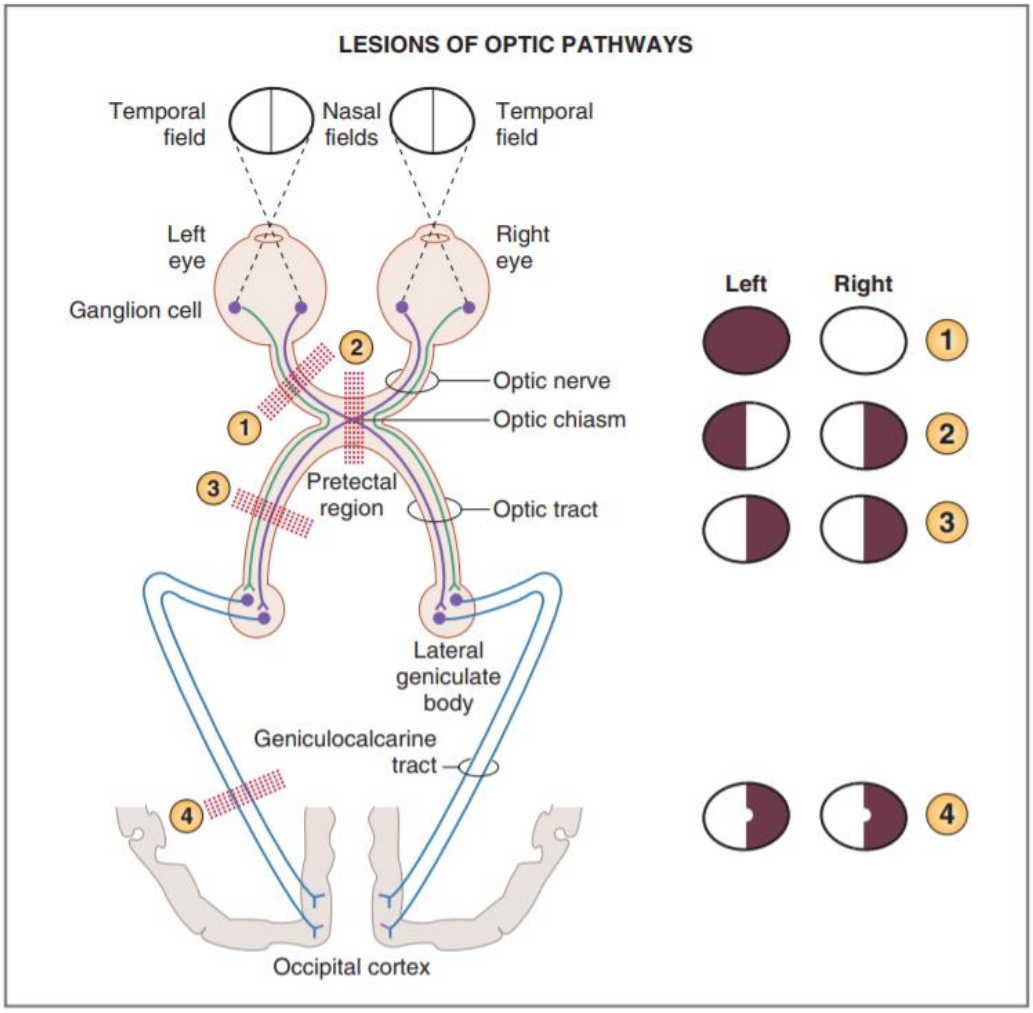
During this pathway, Nerve fibers from each nasal hemiretina cross at the optic chiasm and **ascend contralaterally**. Nerve fibers from each temporal hemiretina remain uncrossed and **ascend ipsilaterally**.(This is seen clearly at the green arrow) (Next lecture we will see that the crossed vs uncrossed fibers are not 50/50)

So we can conclude that fibers from the left nasal hemiretina and fibers from the right temporal hemiretina form the right optic tract and synapse on the right lateral geniculate nucleus. Conversely, fibers from the right nasal hemiretina and fibers from the left temporal hemiretina form the left optic tract and synapse on the left lateral geniculate nucleus.Fibers from the lateral geniculate body form the optic radiations (geniculocalcarine tract), which ascends to the primary visual cortex (area 17 on the medial surface of the occipital lobe, specifically on the supra and infra calcarine gyri. The newest name for this area is the ***striated cortex***).

After following all the fibers,***The Net Result of all of this is that the Right Visual Field in both eyes is represented on the left cortex and the Left Visual Field in both eyes is represented on the Right cortex***.

Deep Look: Lesions of the Visual Pathway

Lesions in the optic pathway cause deficits in vision, which can be predicted by tracing the pathway, as shown in the figure below:



Note: Hemianopia is the loss of vision in half the visual field of one or both eyes. If the loss occurs on the same side of the body or visual field as the lesion, it is called ipsilateral; if the loss occurs on the opposite side of the body or the visual field as the lesion, it is called contralateral.

The following lesions correspond to the circled numbers on the figure:

1. Optic nerve. Cutting the optic nerve causes blindness in the ipsilateral (same side) eye. Thus cutting the left optic nerve causes blindness in the left eye. All sensory information coming from that eye is lost because the cut occurs before any fibers cross at the optic chiasm. Thus patient can see *mostly* the whole visual field but by the right eye only.

2. Optic chiasm. Cutting the optic chiasm causes bitemporal (both temporal visual fields) hemianopia. Information from the temporal visual fields from both eyes is lost because data from the temporal visual fields is collected by the nasal hemiretina fibers which cross at the optic chiasm.

3. Optic tract. Cutting the optic tract causes contralateral hemianopia. We have described the contents of the optic tracts before, so for example cutting the left optic tract results in cutting the right nasal hemiretina fibers leading to loss of the temporal visual field from the right eye (crossed fibers) and also causes cutting the left temporal hemiretina fibers causing loss of the nasal visual field from the left eye (uncrossed). The net result in this example is loss of the right half of the visual field – Right Hemianopia (opposite side of the lesion which is in the left optic tract – thus considered contralateral hemianopia) While the left half of the visual field is preserved in both eyes (so patient still can see in both eyes but only the left half of the visual field).

Note: Also Destroying the Lateral Geniculate nucleus of the thalamus will cause the same effect of optic tract cutting - contralateral hemianopia – because the optic tract fibers end there.

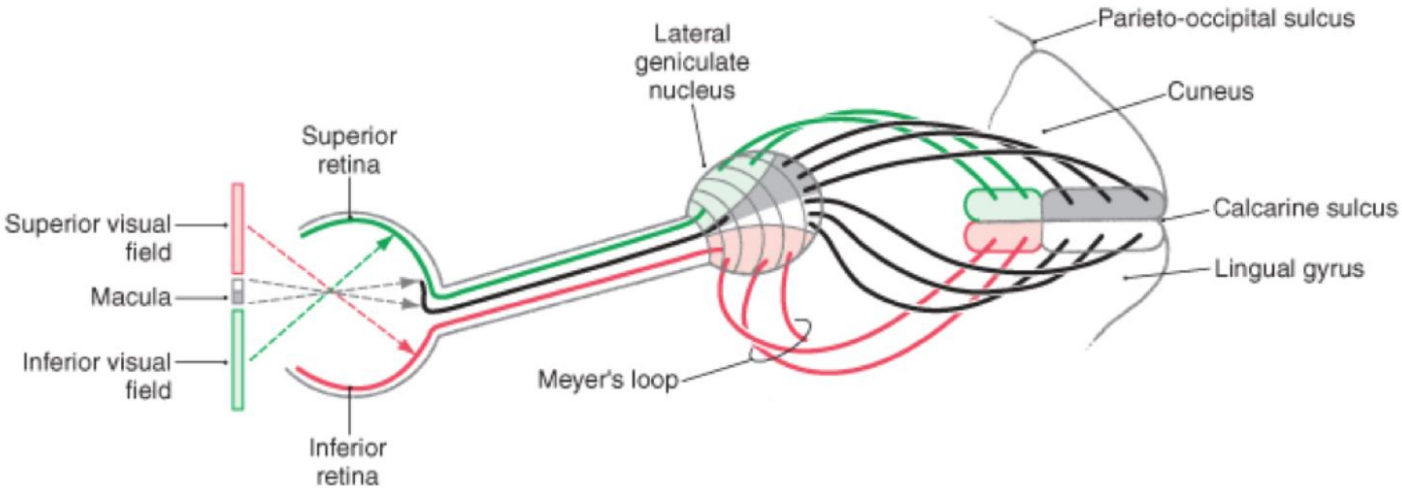
4. Optic Radiation (Geniculocalcarine tract). Cutting the geniculocalcarine tract causes contralateral hemianopia nearly same as number 3 but with macular sparing (the visual field from the macula is

intact). (Extra : Macular sparing occurs because this lesion does not destroy all neurons that represent the macula as they have a slightly different pathway)

5. Primary Visual Cortex: Destruction of the Primary Visual cortex causes contralateral hemianopia same as 3 and 4.

Deep Look: Vertical Cut in the Eye?!

It's very interesting to realize, that if we took a vertical cut in the retina dividing it into superior and inferior halves, due to the spherical shape of the eyes, the superior half of the retina will receive data from the inferior half of the visual field while the inferior half of the retina will receive data from the superior part of the visual field, (which is the same pattern we saw before in the nasal and temporal halves of the retina).



Notice how the superior retinal fibers (green) are separated from the inferior retinal fibers (red) all throughout the visual pathway, but will this create new variation in the defects we described before?

Well, in the optic nerve and optic tracts which are narrow, it's very unlikely that a cut or some kind of pressure will affect only the superior or inferior fibers, so usually the effect will be on all the fibers of the nerve and tract.

However, after the level of the Lateral Geniculate Nucleus , At the Optic Radiations (Level 4) the superior and inferior fibers are widely separated and don't move in a straight line , and at The Primary Visual Cortex (Level 5) also there is separation in which superior fibers end in the superior gyrus above the calcarinesulcus, while the inferior fibers end in the inferior gyrus below the calcarinesulcus.

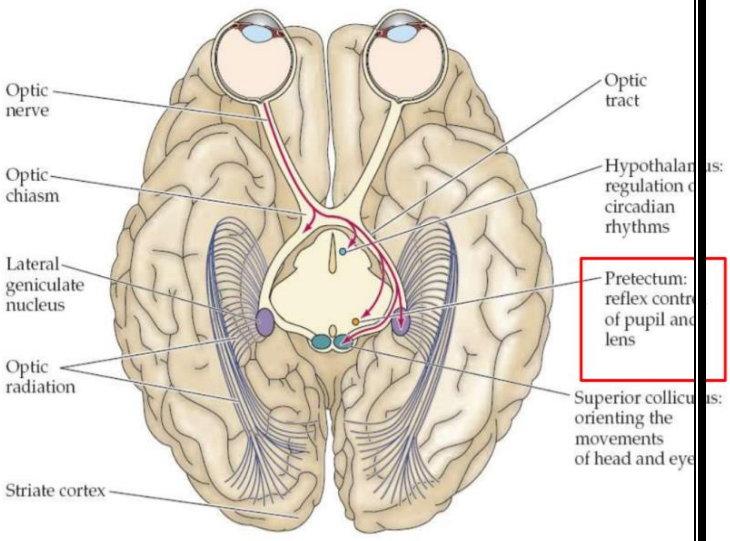
That's why, if there is case of destruction of inferior fibers of the right optic radiation or inferior gyrus below the calcarine sulcus of the right primary visual cortex,**the result will not be left hemianopia as we discussed before** because not all the fibers are affected as the inferior fibers are only destroyed, so the patient will only loose the upper left quadrant of visual field (Superior Left Quadrant Anopia)

Note: Notice how the inferior optic radiation fibers descend inferiorly and anteriorly (before they extend posteriorly to the inferior gyrus below the calcarine sulcus) this area is called Meyer's Loop (shown in picture Above) which is in the anterior part of the Temporal Lobe. So common causes of Superior Quadrant anopia are ischemia,stroke, and trauma to the anterior part of the temporal lobe)

- Refer to slide 13 in (slide-5) for a summary of all lesions, please note that the shaded areas represent the visual field and not parts of the eye.

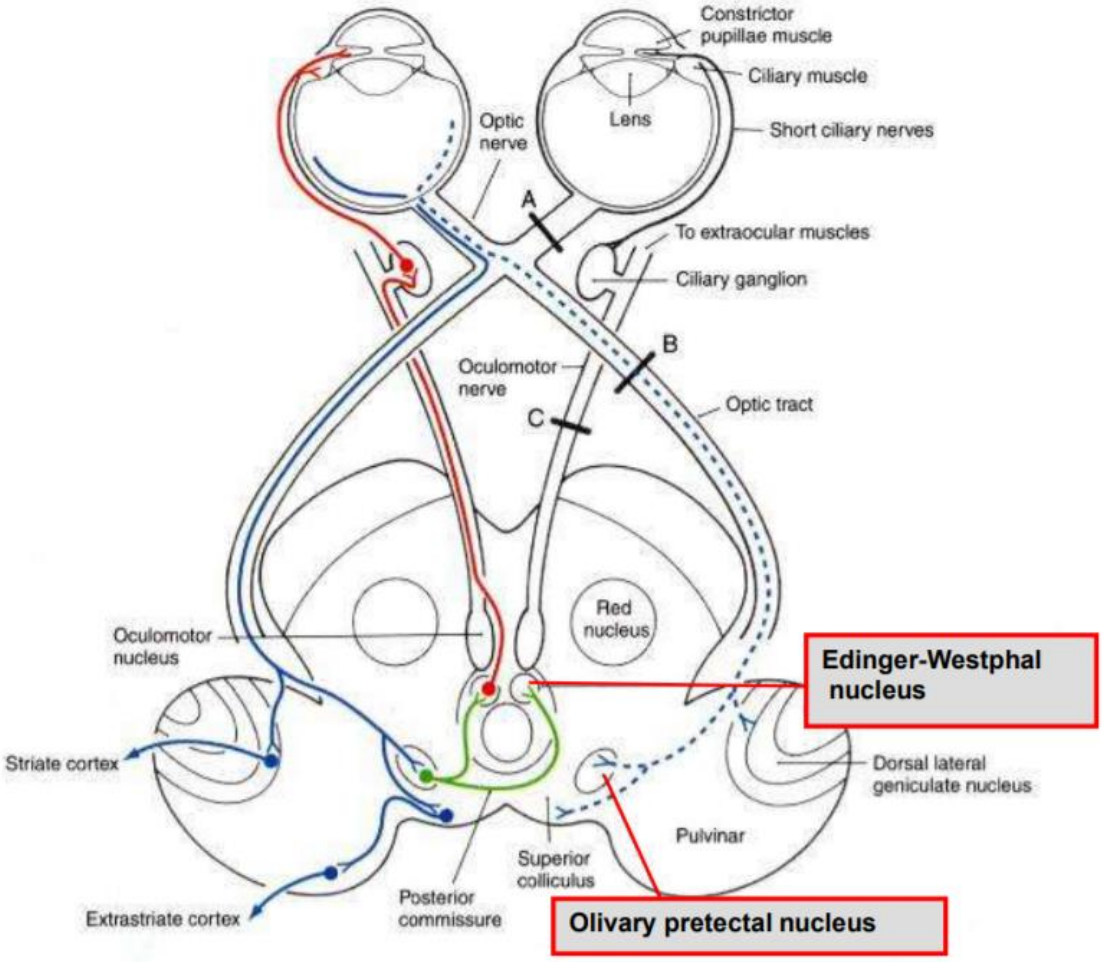
DeepLook:Pupillary Light Reflex

After finishing the general visual pathway,it's important to noticethat the destination of the fibers of the optic tract (as found in the online lecture video) is not only the Lateral Geniculate nucleus, there are other destinations including the Pretectum of Midbrain to control Pupil and Lens reflexes.



Now the whole reflex is described as the following:

1. The same optic nerve we described before (which contains the crossed and uncrossed fibers) sends fibers to the Olivary Pretectal Nucleus in the Pretectal Area.
2. From there , fibers are sent to BOTH the right and left Edinger-Westphal Nuclei.
3. From there fibers are sent to the Oculomotor nucleus (Parasympathatic Nucleus) and extending from it, the oculomotor nerve with the presynaptic neurons.
4. The Oculomotor presynaptic neurons synapse in the Ciliary ganglion and from there the postsynaptic neurons end in the constrictor papillae and ciliary muscles.



Deep Look:Pupillary Light Reflex Lesions

Going back to the photo before,let's describe the effect of lesions on the pupillary light reflex.

Lesion at A (OpticNerve): In this example it's the right optic nerve; If we inserted light into the right eye, There will not be any pupil constriction in both the right and left eyes. But if we inserted light into the left eye,there will be pupil constriction in both eyes due to crossing of fibers at the optic chiasm which will cause the signal to reach both right and left Olivary pretectal nuclei. (Right Olivary pretectal nucleus will receive the fibers of the left nasal hemiretina while the left Olivarypretectal nucleus will receive the left fibers of the left temporal hemiretina).

Lesion at B (OpticTract): In this example it's the right optic tract,; If we inserted light into the right eye, there will be pupillary constriction in both eyes because even if the right Olivarypretectal nucleus will not be activated at all by the cut right optic tract, the left olivarypretectal nucleus will be activated by the left optic tract (which contains the right nasal hemiretinalfibers). And as we mentioned before the left olivarypretectal nucleus will send fibers to Both the left and right Edinger-Westphal Nuclei causing motor signals to reach both eyes. And the same idea if we inserted the light into the left eye also there will be pupillary constriction in both eyes because the the left olivarypretectal nucleus will be activated by the left optic tract (which contains the left temporal hemiretinalfibers) and the left olivarypretectal nucleus will send fibers to Both the left and right Edinger-Westphal Nuclei causing motor signals to reach both eyes.

Lesion at C (OculomotorNerve): In this example it's the right Oculomotor Nerve; If we inserted light into the right eye, there will be pupillary constriction in the left eye only, because signals will reach both Olivarypretectal nuclei and both Edinger-Westphal Nuclei, but because the right Oculomotor Nerve is cut, there will not be any motor order of papillary constriction on the right side. And the same idea if we inserted light into the left eye, also there will be pupillary constriction in the left eye only for the same explanation.

Extra: Lesion at the optic chiasm (in other words destruction of crossed fibers) : light reflex will still be present; if we shine light on the right eye the signal is still being sent by the right temporal hemiretinal fibers (uncrossed) to the right Olivarypretectal nucleus which will send it to both Edinger-Westphal Nuclei , and same goes for the left eye .

Deep Look: Intensity of Pupillary Light Reflex Constriction

The intensity of pupillary constriction depends on the degree of activation of the Edinger-Westphal nucleus, which is activated by the olivary pretectal nucleus which is activated by the optic tract...and let's say I inserted the light into the right eye , the right temporal hemiretinal fibers will continue ipsilaterally in the right optic tract activating the right

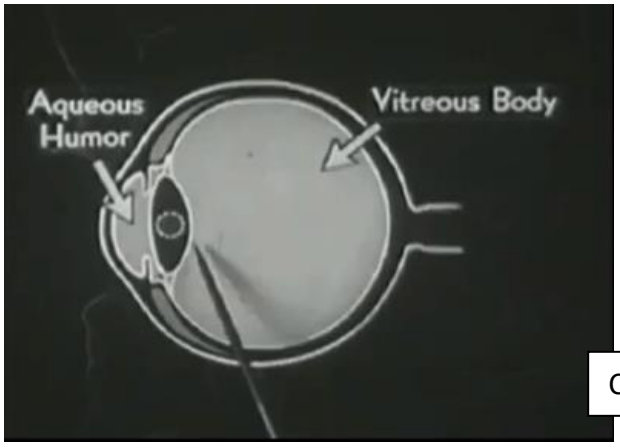
olivarypretectal nucleus activating both the right and left Edinger-Westphal nuclei and the right nasal hemiretinal fibers will continue contralaterally in the left optic tract activating the left olivarypretectal nucleus activating both the right and left Edinger-Westphal nuclei, **so at the end Both eyes will constrict in the same degree in normal conditions ! Because equal stimulation is reaching both Edinger-Westphal nuclei !** However , if a person has different degrees of constriction in which one eye constricts stronger and more powerfully than the other , this person has what is called ***RAPD (Relative Afferent Pupillary Defect) .This Defect is diagnosed by a test called swinging-flashlight test which will be explained next lecture.***

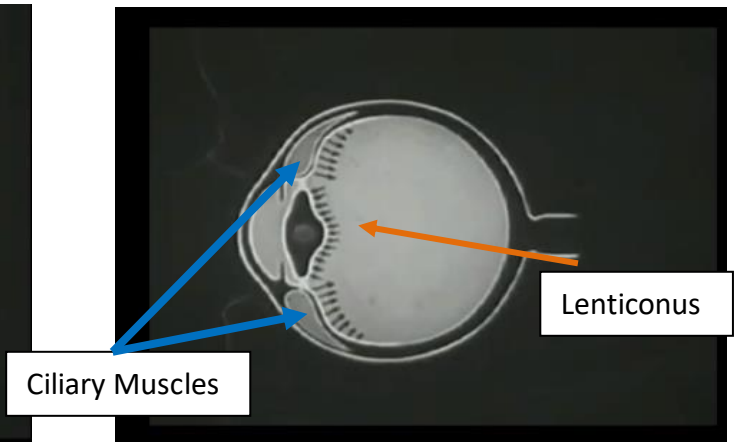
Note From the Slides: The Pupil constriction in the eye of the same side of insertion of light is called direct pupillary reflexwhile the pupil constriction in the eye of the opposite side of insertion of light is called the consensual pupillary reflex. (Which both should be equal normally)

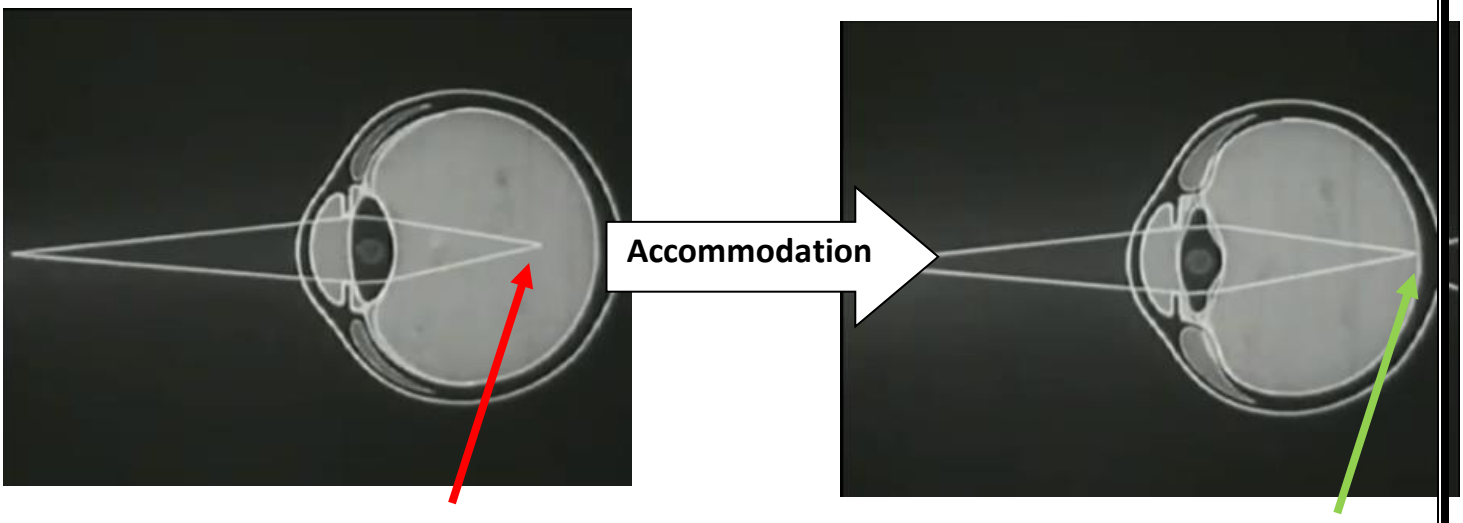
Deep Look:Near Reflex

When we look at a near object, the Near reflex is initiated, which consists of a triad:

1. **Lens Accommodation:** This phenomenon was explained in (Vision 1) Video online, but as a summary, it's a parasympathetic reflex including the activation of the Ciliary muscles (We mentioned before that the oculomotor nerve innervates also the Ciliary muscles in addition to the Constrictor papillae). This will lead to push the edges of the lens around the rigid spherical core of the vitruousbody, Thickening and rounding the lens in the middle creating what is called the Lenticonus . This will restore the sharp image on the retina.

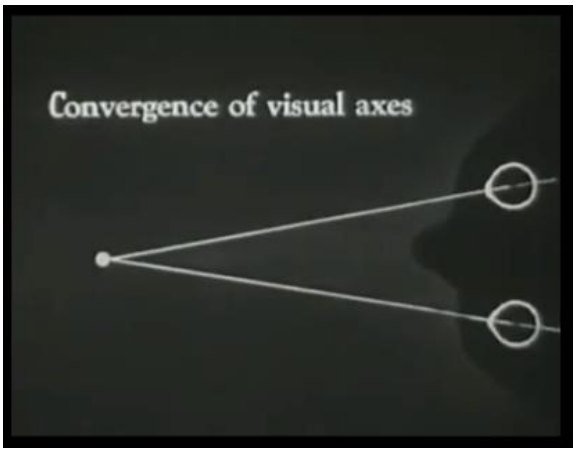






Blurred Image (Not focused on the retina)

Sharp Image (focused on the retina)



2. **Covergence of Visual Axes:** is when the eyes rotate towards each other(to the middle) in order to create a single image of a near object.

3. **Pupillary NearReflex:** Scientists found that pupil constriction does not only happen in response to light, it also happens in response of looking at a near object. The explanation for this is to focus the light entering the eye through the middle bulge (thickening) created by lens accommodation explained before,this gives the best *focal depth*.

   Note: *focal depth* is the distance in which an object stays within focus for a certain lens.

   Important Note: If you look at 2 objects (for example imagine putting your hand with the TV screen behind it, what decides where to focus on the hand or the TV screen?) The Decision is a conscious decision originating from the cortex. So this means that there are fibers that are descending from the cortex to synapse in the pretectum area (not in

the olivarypretectal nucleus) and continue to theEdinger-Westphal Nucleus.

So there are 2 reflexes to activate the Edinger-Westphal Nucleus and cause pupillary constriction , the light reflex and the near reflex , and they are independent in which if the right reflex is defected that doesn't mean that the near reflex should be defected and vice-versa . If a patient is having a big difference between the light reflex and near reflex – one is absent or poor and the other one is present – this case is called **Light-Near Dissociation** which will be explained next lecture.

For any Questions or further corrections feel free to contact

الحمد لله