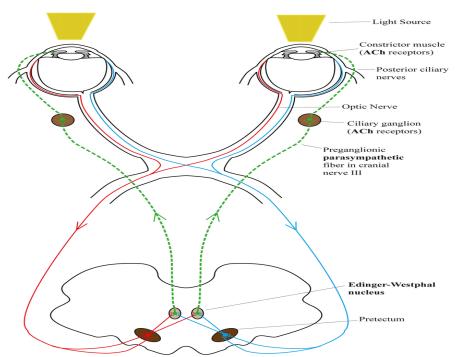


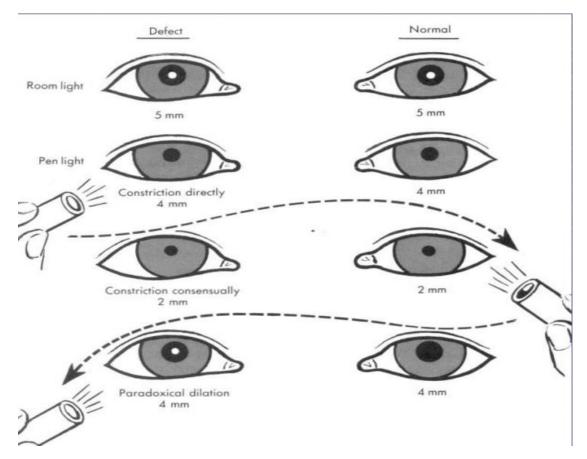
The doctor started with a short conclusion about the last lecture which was about the pupil reflex and he explained the medical terminology related to the reflexes such as direct reflex (constriction in the eye where the light was shined into) and consensual reflex (constriction in the other eye where no light was shined into) and that due to the fact that there's an unequal crossing of fibers a disease called RAPD (Relative afferent pupillary defect) might develop.

For example the right eye, the fibers in it will cross to the contralateral optic tract and some of them will remain ipsilateral, and these ipsilateral fibers will unite with the ones crossing from the left eye, so when the light is shined into any of those eyes, a 100% stimulus will reach the fibers and the same amount of constriction will occur in both eyes. However, when one of the tracts is damaged, the other optic tract will contain 60% of the fibers of the other eye (Crossed fibers) and 40% coming from the same eye (Uncrossed fibers), so if the light is shined into eye where the fibers are not crossing 40% of the normal constriction will occur, and if it's shined into the other eye, 60% of the normal stimulation will occur, so if we presume that the eye with 40% constriction is (X), the eye where 60% constriction occurs is considered (1.5X)



And since it's difficult for doctors to notice the degree of constriction in such subtle amounts, we use the swinging light test to diagnose RAPD.

You start the examination by ask the patient to look directly forward and shine the light into the right eye, you will notice constriction, and then shine it into the left side and you'll also notice constriction, then you'll start swinging the light into both patient's eyes back and forth. In a normal case because there is no damage, while swinging the light back and forth, the same amount of constriction will persist. However, in a RAPD patient, while swinging the light, whenever it is directed into the eye with less crossing (40%) you'll notice a slight dilation of the pupils, and we will also notice which eye is the one with less crossing and determine it as the eye with RAPD.



In this picture, we notice that while swinging the light, there is a slight dilation in the both eyes whenever the light is shined into the left eye, so we say that the defect is in the left eye.

Normal Pupillary Response OD OS OD OS OD OS Relative Afferent Pupillary Defect in the Left Eye OD OS OD OS OD OS

In this picture, we can see two different responses, One where both eyes are normal, and one where one eye is not functioning properly (RAPD).

The normal eyes example: Wherever the light is shining, the constriction remains the same (no difference in constriction intensity).

The RAPD case: As you can see, when we shine the light into the right eye (Left from your perspective) a stronger degree of constriction can be noticed than when we shine it into the left eye (Right from your perspective), that means the RIGHT optic tract is the one damaged.

There are many causes which lead to RAPD:

- Optic tract lesion

- Glaucoma (Increased intra-ocular pressure) might also cause it, because the increased pressure will compress the retina causing degeneration and the ganglionic cells will decrease causing the same effect.

- Cataracts also cause this disease since they decrease the amount of light entering the eye causing the same effect.

- The most common causes of rapid usually occur before the optic chiasm such as: demyelination, ischemic retina, retinal detachment, optic nerve injuries, and diabetic neuropathy.

- Unilateral Mid-brain lesions will also cause RAPD.

The doctor also spoke about the 2 pupillary methods of constriction:

1 – The pupil's near reflex

2 – The pupil's light reflex

4 | Page

An example to further explain the test:

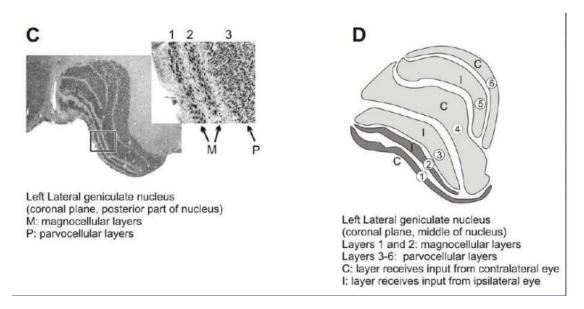
And both of these reflexes will go towards the pretectum region to eventually reach the Edinger-Westphal nucleus. The near reflex descends from the cortex posteriorly and the light reflex moves from anterior to posterior, because of that the near reflex area in the pretectum lies more posteriorly.

So in case of a mid-brain damage that originates from back to front in the area of the pretectum, the near reflex will be damaged and the light reflex will not. There are many causes which result in this type of defect (Near-light dissociation), the most common being Dorsal midbrain syndrome (Parinaud's Syndrome), and others such as: Stroke, Meningitis, Tumour, Neurosyphilis, Diabetic neuropathy, and Demyelination (MS).

There are different shapes of ganglionic cells in the eyes, which lead to different modalities of vision, and the most important of ganglionic cells are the ones that go to the optic nerve and the thalamus and go through the optic tract, which were the X and Y shaped ganglionic cells.

X represents the detailed vision while the Y deals with movement. Now we use both eyes to see, but there's an area in between which both eyes see the same thing, and the representation of that area is different in each eye, so there are 2 important things to preserve which are, which eye is viewing the object in that area, and is it an X modality or Y modality, that's why when we arrive at the thalamus, at the geniculate nucleus, the information from the right eye synapses at a different place than the left eye, the right thalamus receives information from both eyes and the same happens at the left part of the thalamus. So, information from the X ganglion stays separate from the Y ganglion. Therefore, if we look at the geniculate nucleus, it contains 6 layers, the first 2 layers are called magnocellular (Because they're large in size), and the rest are called parvocellular. Y-cells go to the Magnocellular cells, and X-cells go to the parvocellular. And magnocellular layers have one for each eye, the same is found in the parvocellular layers. (Don't memorize which layer is contra/ipsilateral).

Note: X-type ganglionic cells give detailed vision and colour processing. More representative and is found more abundant in the fovia centralis.



So, information from both type of cells, X and Y, will be preserved up until we reach the cortex, specifically the 4th layer, so both will arrive at the same layer. However, at each point of the space there will be 1 column from each eye like the auditory pathway. So different shaped neurons will come from the magnocellular and the parvocellular.

Each will be processed differently, so the visual information processing will go in different pathways, one of them is called "What pathway" which is in the inferior-medial surface of the cortex where processing of shapes, textures and colours occur. And the other pathway which is the "Where pathway" which happens in the dorsal aspect of the cortex, which will give us information about the location and position of the object in regards of oneself, the other thing that it gives is that if the object is moving and the speed it is moving at.

For cortex to be able to process information it needs memories and it compares the things to the memories it has, through sequence processing starting with shape, then color then texture for example. So while the cortex is stimulating all the memories we have for this specific object, it is also inhibiting all the memories of similar objects which are not related to it, for example, seeing some guy with a beard, the memories that might fit this description are stimulated more than others, this is what called "Stimulus to noise ratio", the more the things are related then the stimulus to ratio will decrease and we will learn in the future, that a major part of processing is dedicated to inhibiting more than stimulating, because inhibition is more important.

The cortex is the outermost 3-5 mm layer of the brain and there we have different shapes neuron with different characteristics depending on which layer of the cortex, physiologically speaking we have 3 types of cortex:

1- **Primary cortical area**: Is connected to outer brain information, and receives information from outside the brain, which is called Sensory cortical area such as the visual cortex, auditory cortex, primary somatosensory cortex.

So this area receives information, processes it a little and directs it towards the secondary areas, any issue in the primary sensory cortexes will cause loss of sensation (blindness, deafness, loss of somatic sensation)

2- Secondary cortical area: the area responsible to receive information from the primary area and its function is to process this information, and the more complex the sensation is, the larger the secondary cortical area of that sensation is. (the smallest secondary cortex being the taste area, and the largest being the visual area), This area will compare the received information to the old information we have to understand what current type of sensation is occurring.

In case of defect in the secondary areas, we will lose the ability to distinguish between sensations, for example being able to smell something but not knowing what it is. (Called Agnosia: Being able to sense but not differentiate what a person is sensing)

The most famous type of agnosia is the one that occurs in the visual area which causes the inability to differentiate between people from their faces, instead they will attempt to recognize people from other type of information such as hair, clothes, expressions. (Facial blindness/ Prosopagnosia) 3- Association cortex: Now we already have the sensation processed separately, but still we need to combine all processed sensations to become a complex piece of information to get the true required meaning of all this processed information, such as the example mentioned in the online videos which is Flavor. 2/3 up to ³/₄ of the brain is dedicated to association cortex.

So after processing in the association complex, I received the sensation, processed it, explained it and extracted the information, then It's redirected into a higher level processing cortex, it helped me decided what I want to do with this information, it's embedded into my motivation, my intentions, now it is time to either issue an order or not, such as voluntarily movement. So even voluntary motor movements, the go through primary (Pre-central gyrus), secondary (Pre-motor cortexes) and association complex to produce movement. And damage to the primary area will cause paralysis, the damage to the secondary area causes Apraxia.

Keep in mind each cortical area process different information at the same time, the secondary area on the right will process something while the left secondary area will process something else, or else it would be an awful waste of resources.