بسم الله الرحمن الرحيم

CNS-2: Physiology: **Vision**

"The eyes are the windows to the soul"

This is the **3rd** lecture in CNS-2 **Physiology**. In this lecture we will talk about one of our special senses, and that is **the Vision**. Hopefully this sheet will cover the **following:**

A: **Layers of the eye and some clinical applications.**

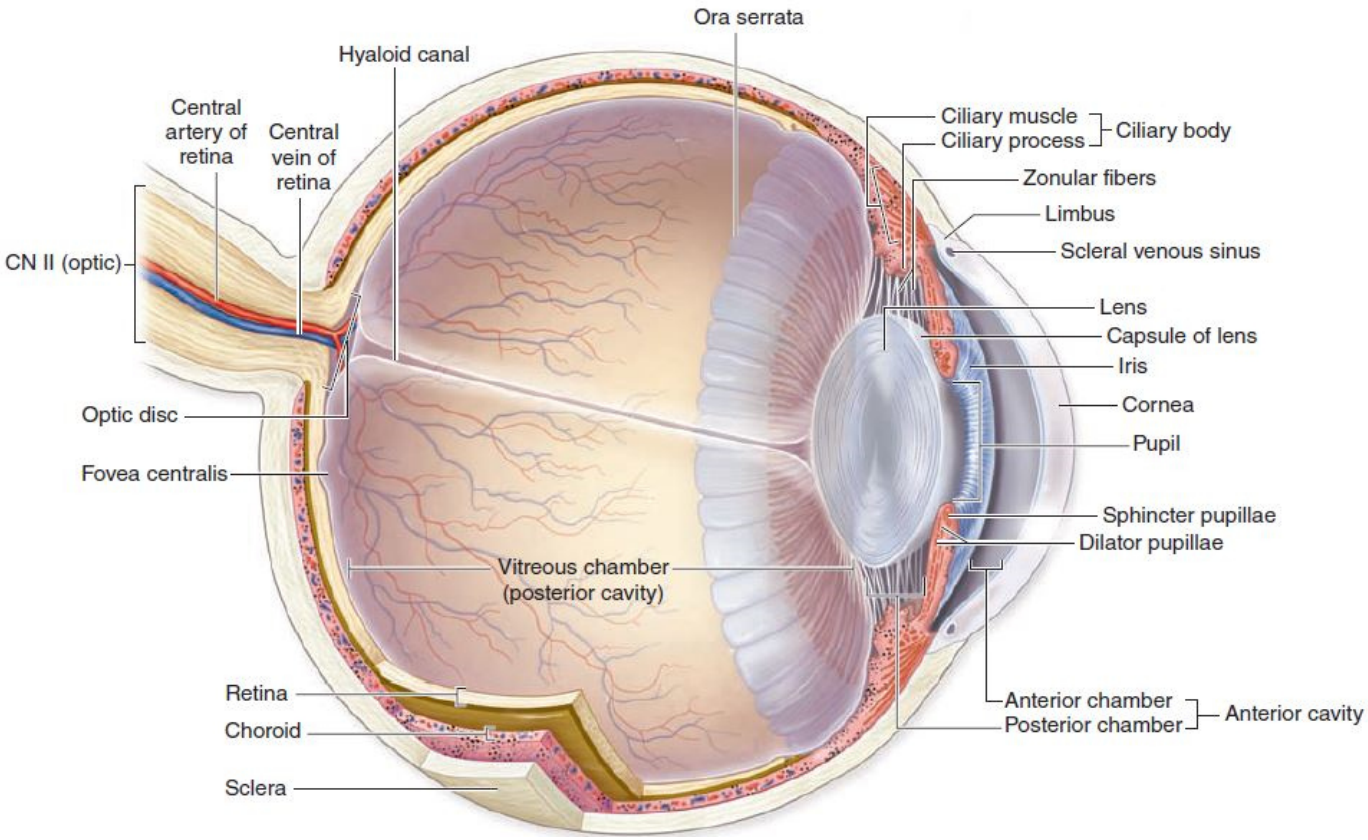
B: **Retina, Visual adaption, and ganglionic cells.**

C: **Visual pathways.**

D: **MRI**

A: Layers of the eye and some clinical applications:

The eye consists of **3 layers**: **Sclera**, **choroid**, and **Retina**. **Sclera** gives the eyes their shape, while **Choroid** is responsible for the blood supply; sensory processing and detection of light are the functions of the **Retina** of the eye. Anteriorly, we have the **Cornea** which is responsible for the passage & refraction of light in addition to its protection function. Behind the cornea we have the **Iris**; which is the colored part of the eye, its function is to determine the amount of the light that will pass through the eye by controlling the diameter and size of the pupil. The **Pupil** (which is found in the center of the iris) can be dilated or constricted by the effect of **muscles and both the sympathetic & parasympathetic** nervous systems. We also have muscles like lateral and medial recti muscles that move the eye to the desired direction.



There are **2 fluid** compartments in the eye: **vitreous humor** and **aqueous humor**; fluid is under continuous production, and obviously excessive production, or decrease in filtration, will lead to **increase in pressure**, this increase will lead to **death** of blood vessels & nerves (direct death of neurons) by compressing them, also it will **impede blood nourishment** to the retina leading eventually to what is known as **Glaucoma** المية الزرقاء**. Glaucoma** can be treated by drugs, however in some cases surgical intervention is required to correct the eye.

There is a **depressed** part in the retina called **Fovea Centralis (or macula),** in which high detailed vision is achieved. Almost only **cones** receptors are found in this part "with **almost** no rods", also cons are found in **high densities** and **small sizes**(smaller or as the same size of rods); this will make images in this part with **high pixels** (due to number of cons/area) and

more **detailed** and **accurate**. Other layers of the retina are shifted to the sides (not directly in front of the visual field) allowing the light to pass with **no interference** in other compartments of the retina.

Note: Moving the eyes horizontally (right to left) is actually for focusing the desired image (its light waves) on the macula.

Degeneration in the retina is more **targeted toward the cons** than the rods, this will lead to **Macular Degeneration;** usually it is **age related**, however it can be caused by diabetes or other diseases, also it can be **genetic** (i.e. **Juvenile type** macular degeneration **(Stargardt Disease)**.Such patients (macular degeneration pts.) will have normal vision but when image radiation hit on the macula there will be **blank area** in the formed picture, as the following:

Normal vision

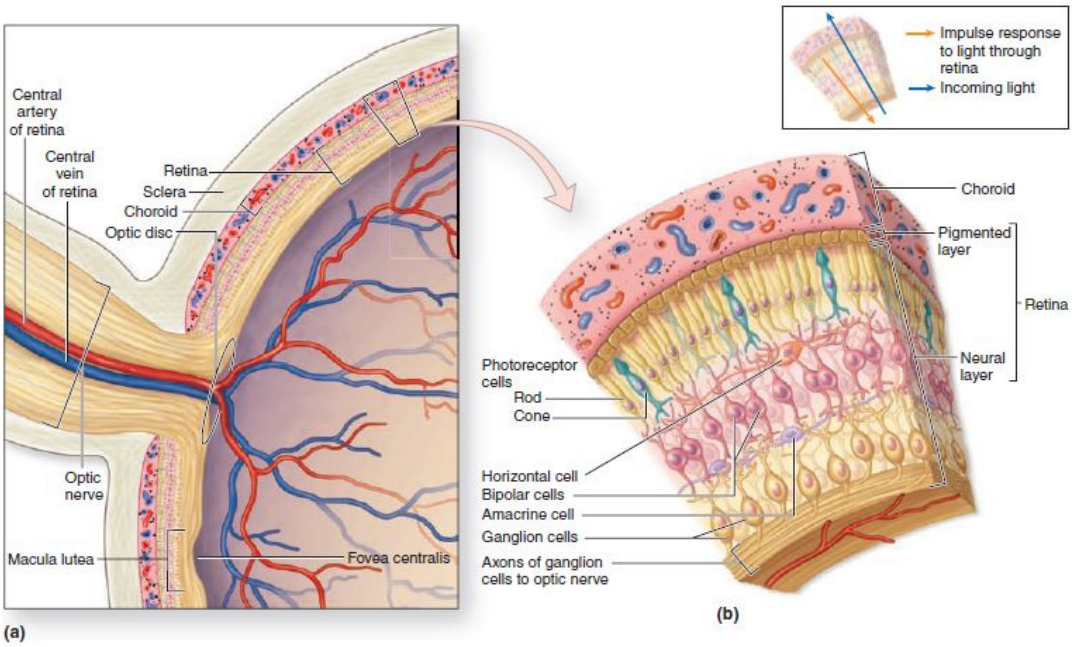
Macular Degeneration

This will culminate when macular degeneration patients look at **faces**; they will be able to see everything except faces (its sth called spotoma), usually this can be beaten by **specialized lenses** that will shift central radiations to the periphery to deliver it to other sites, or through **behavioral therapy** (by training those patients to look at shoulder area of human, leading to centering the shoulders on the macula, and consequently radiation from the face will be shifted to the periphery).

B: Retina, Visual adaption, and ganglionic cells:

We have many layers in the retina: the <u>first</u> one from the back is pigmented layer that will **absorb** the light and prevent **reflection** of it, so **light will contact with the receptors just once, not <u>twice</u>**. Then we have the **receptors layers** that will **detect** the information and pass it to **bipolar cells** that will pass it to **ganglion cell** from which axons will form **the optic nerve** that will enter the central nervous system.

2 types of cells found horizontally called **horizontal cell** and **Amacrine cell**-خلايا عديمة المحاور-they will do the processing of light signals at **the level of the retina**; before the passage of information to the ganglion. **Cons & rods** are the two types of photo receptors, **cons** are of 3 subtypes that are responsible for **colored vision**, while **rods** will only show **black/gray** color during night.



The retina is the thick layer of the eye inside the choroid. **(a)** The central retinal artery and vein pass through the optic nerve and enter the eye at the optic disc. These vessels initially lie between the vitreous body and the ILL of the retina, but their smaller lateral branches penetrate this layer and enter the retina, forming capillaries that extend as far as the INL. Nutrients and O_2 for the outer retinal layers diffuse from capillaries in the choroid. **(b)** The layers and major neurons of the retina and their general organization are shown schematically here and the Müller support cells are omitted.

In **fovea centralis (macula)** we **almost** exclusively have **cones** but when we move away from it cons will be reduced and rods will be increased until reaching the upper periphery where there is **almost** no cons.

Blood vessels & nerves enter and leave through an area called **optic disc** where there are no cons & rods (due to the absence of retina in this area), so it is referred to as the **blind spot**. We don't see blind spots for 3 reasons;

A) We have **two eyes** so each blind spot from an eye will **cancel** the other.

B) Due to **continuous movement of the eye**. This explains why there is no blind spot when you close one eye.

C) Brain **pattern**: when focusing on a point -when there is no eye movement- blind spots are **non-existent** because the brain will fill the image which its radiation reached the blind spot area and will fill it from its surroundings using brain pattern (it's like photo editing). Remember that "We see with our brains, not eyes ".

To test the brain pattern thing, do the following experiment:

1st: you will need a white paper, pen (or pencil), and ruler (optional)

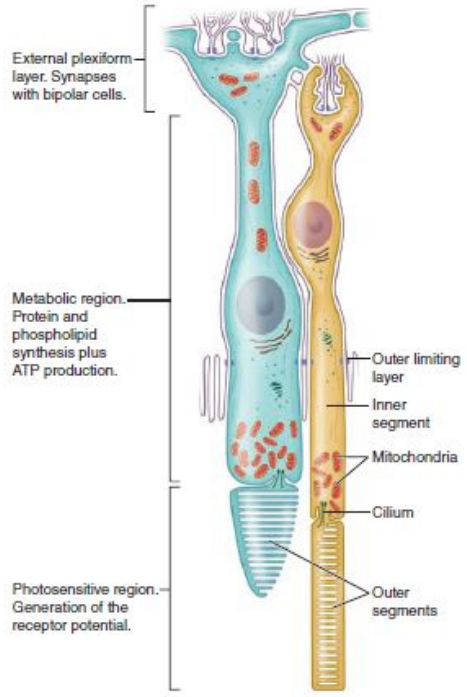
2nd: using your pen (or pencil: P), draw two points with a space between them of about 10-20 cm.

3rd: now with one eye closed, look at your masterpiece (the two points on paper), when you see the two points then both of them are on the retina.

4th: try focusing your opened eye on one point only and look closer, and closer … you will notice that the other point disappeared; because it has hit the blind spot , so the brain is not seeing it and there is a gap in the image , so the brain can't understand that there is another point so it will fill it with the background (white color) .

This experiment illustrates the concept of blind spot and brain pattern.

Photoreceptors: we have the **cons & rods** as photoreceptors. Rods have **rhodopsin**. Cons have **photopsin** which is of many types each of which will maximally absorb light, and produce maximum action potential depending on certain **wave length** that will divide waves into: 1)**high**, 2)**middle**, & 3)**low** frequencies. You can see more than one color due to the **overlap** between photopsins and the **percentage of activation** of each one of them. On the other side, Rods cover broad wave length.

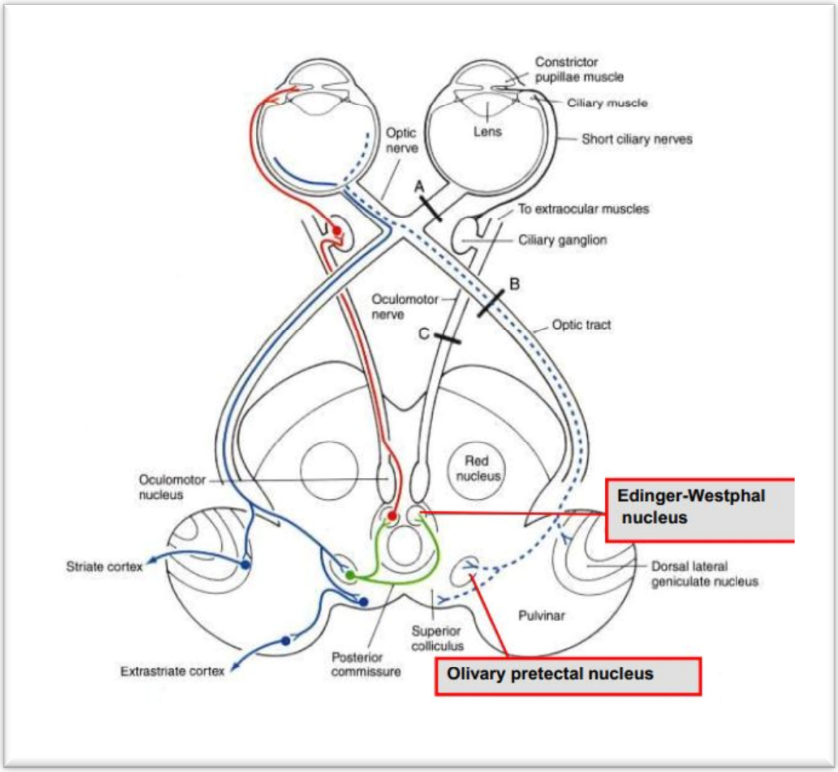


Rod cells (right) and cone cells (left) all have the general shapes and important cytoplasmic features shown diagrammatically here. The outer limiting layer is the line of adherent junctions that attach the rod and cone inner segments to the distal ends of the Müller glial cells (not shown). The membranous discs of the cone outer segments are continuous with the cell membrane; those of the rods are not.

Light detection: when there is light there will be a **decrease** in the release of **Glutamate**, to see clearly we need adaptation according to the light. **Adaptation** will occur at three levels:

1) **Pupil's size adjustment: very quick**, less than second, when we face any light there will be **constriction** of the pupil, while in the absence of light (in the Darkness) there will pupil **dilatation**. (This is achieved by sympathetic and parasympathetic).

- **Pupillary light reflex**: after light detection, impulses will travel through the optic nerve reaching the two sides of midbrain (Bilatellay) in the **tectum**, and each one of the optic nerves will innervate **Edinger-Wesphal Nucleus**, which is a **parasympathetic** nucleus; then it will send the impulses to the **occulomotor nerve** leading to pupils constriction, so each eye will send impulses to the two sides of midbrain, that will innervate EW nuclei leading to **pupil<u>s</u> constriction.**



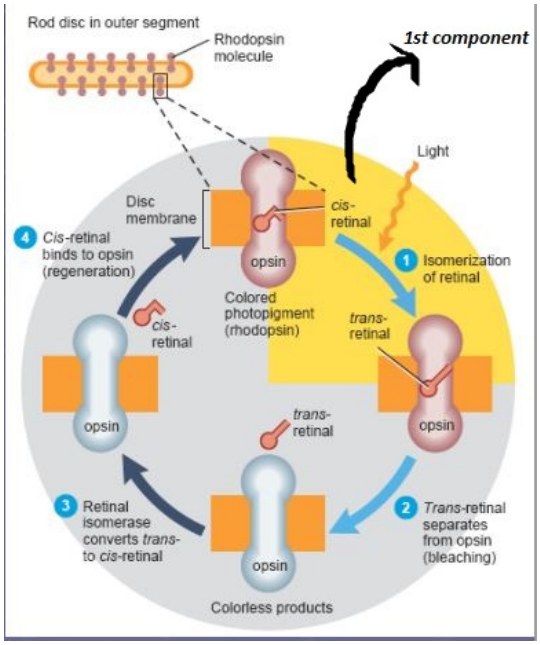
2) **Neuronal adaptation**: here the same neuron will have a **decreased** action potential or reduction in the receptors on it or maybe **channels**.

3) **Photoreceptor adaptation:** depending on **the amount of light**; photoreceptors and detection mechanisms will be adapted.
The light cycle has 2 components:
A) The component that will convert photo pigments from **11-Cis form** to the <u>active</u> **All-Trans form**.
B) The component that by **enzymes** will replace the active Trans form back to the **inactive Cis form**.

- If the **second component** was <u>more rapid</u> then the first component, then eventually and after a while the concentration of the compound that can absorb the light (initial compound) will **decrease**, leading **to decreased sensitivity to light.**
  ➔This is the case when the amount of light is increasing.

- If the **first component** was more rapid then the second component, then eventually and after a while the concentration of the compound that can absorb the light (initial compound) will **increase**, leading **to increased sensitivity to light.**
  ➔This is the case when the amount of light is decreasing.

- **Look at the following figure, the 1st component is the 1st quadrant of the cycle, while the second component is the last 3 quadrants of the cycle.**

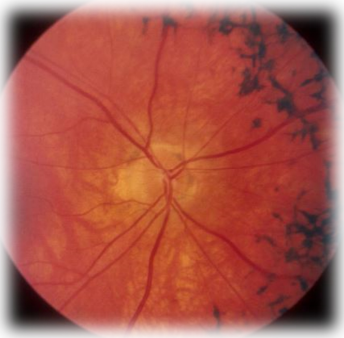


Note: differences between photoreceptors shall be considered as follow; the initial compound at the start level in the rods is more than it in cons, so at any circumstances the eye won't be able to see under a certain level; however the converting enzymes in the cons are faster than those in the rods. ➔ **Cons are faster in adaptation than rods, but the level of adaptation is lower so they won't be highly sensitive.** In other words; the rods have more proteins, so they adapt better, allowing us to see at lower light intensity.

Photoreceptor adaptation takes **long time** to occur, so it is **not** considered fast adaptation like pupils size & Neuronal adaptation. It's noteworthy that photoreceptor adaptation **depends** on **the initial compound** (which is the **11-Cis-retinal**

compound) that depends on **Vitamin A**, So if there were Vitamin A deficiency the initial compound will be reduced in rods (since it is more in it), this will affect vision in **low light conditions** leading to what's known as **Night Blindness. Vitamin A supplement**s are used in this condition to reverse this process and it usually require 1-2 weeks until night vision become better.
Note: check on the internet how night blindness patient see; because it won't be clear on papers and will consume a lot of unnecessary black ink.

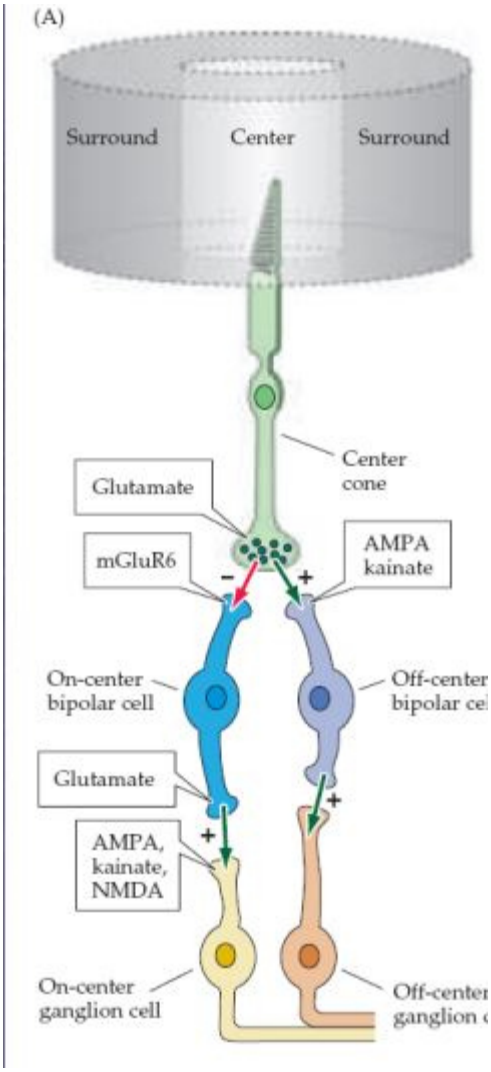
Similar to cons degeneration in macula, degeneration in the **periphery's rods** is also possible leading to **Retinitis pigmentosa,** it will start from the periphery and spread medially, patient will start to lose vision at the periphery & almost see things only centrally, and this state of vision is described as **tunnel vision;** In this case rods will disappear and will be replaced by pigmented cell layer hence the name (Retinitis pigmentosa).



Patients will perceive images as the following:

We have **visual processing** at the level of the **retina**, here we have more than one cell type, **ganglia are the only cells that can make action potential**, because their function is to send information (impulses) to the brain, while **other** cells are for detection and processing, that's why **processing happen through graded potential**, horizontal cells (that were mentioned before); both help in visual processing **at the level of the retina.**

When there is light, there will be **decrease in Glutamate release**, recall that the effect depends on **the receptor** not neurotransmitter. Two types of bipolar cells are present; one have **excitatory receptors** while the others have **inhibitory receptors**, so when Glutamate decreases; the cells that have **excitatory receptors will be inhibited**. On the other hand, the cells that have **inhibitory receptors will be**



activated; this will be translated to ganglion cell, so we'll have **on-center** & **off-center**.

Same principle apply for color vision, but remember that each type absorb a certain wavelength.

On-center will produce action potential when there is **central radiations** (i.e. coming from the center), and the opposite will occur if the light came from the periphery. **The more light is in the center the more firing of action potential from On-center bipolar cell**, however **if the light increases in the periphery there will be inhibition**, and **if the light wasn't on the field vision will be at spontaneous level;** The same principle applies for color vision.

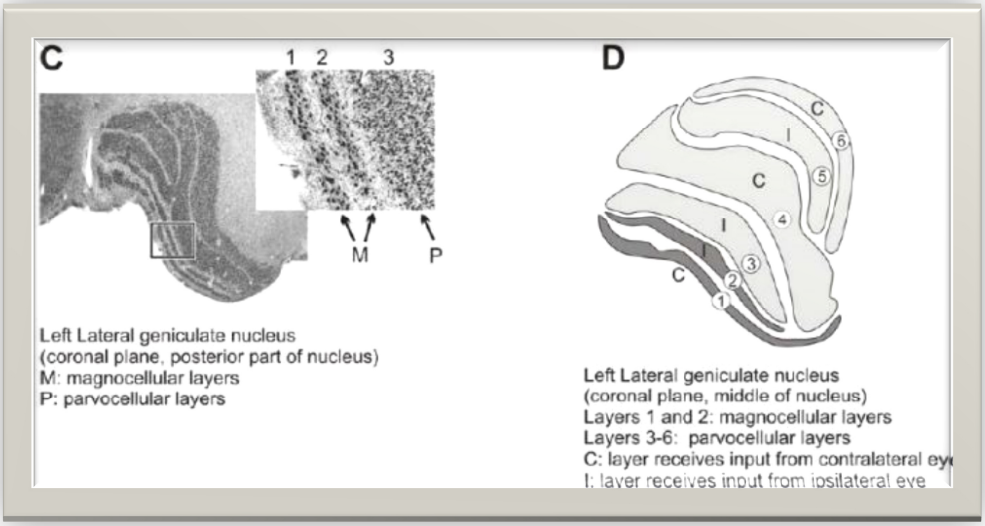
Determined by their **shape**, **type**, and **destination**; Ganglion cells are <u>mainly</u> of 3 subtypes:
1-**X ganglion:** is the **most important one**, it is **small** with **small receptive field** spreading on the retina, usually found more in **the central part** of the retina and **in the fovea centralis** and it contain **more cons** than rods, also it's with **better accuracy**; because it doesn't receive from wide field, and because it perceive details, it is **more related to the detailed vision**.

2-**Y ganglions:** are **large**, **widely spread** over the retina in the **periphery**, and with **higher receptive field** and it contains **more rods** than cons, it is concerned with **general shape** of the image than details, more involved in **motion,** also it receives more and more form **amacrine cells**(which is responsible for processing of moving images).

3-**W ganglion:** <u>similar to Y ganglion</u>, also it's found in the **periphery**, mainly it receives from **amacrine** cells than bipolar cells or rods, and also like Y it is involved in the moving images.

Main branch of optic nerve will go to the **optic tract**; from their main destination of optic information is **the thalamus**, especially **lateral geniculate body**.

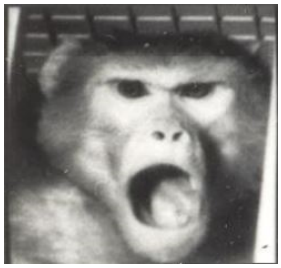
Lateral geniculate is divided into **six layers**; in 2 layers we have large cells called **Magnocellular layer (layers 1+2)**. While in the other 4 layers we have small cells called **Parvocellular layer (layers 3-6).**



The right lateral geniculate body will receive sensory input from **the left and right eye**, so **the 2 magnocellar will serve for both the ipsilateral eye and the contralateral eye (one for each eye)**. Also **parvocellular will serve for ipsi &contra-lateral eyes (2 parvocellular for each eye).**

X ganglion will send to the **parvocellular layer**, while the **Y Ganglion** will send to **magnocellular**. Then from the LGB visual information will ascend to **the cortex**. The signal from X and Y will remain separated till it reaches the cortex.

This how we see images by magnocellular and parvocellular:



Normal

Magnocellular only

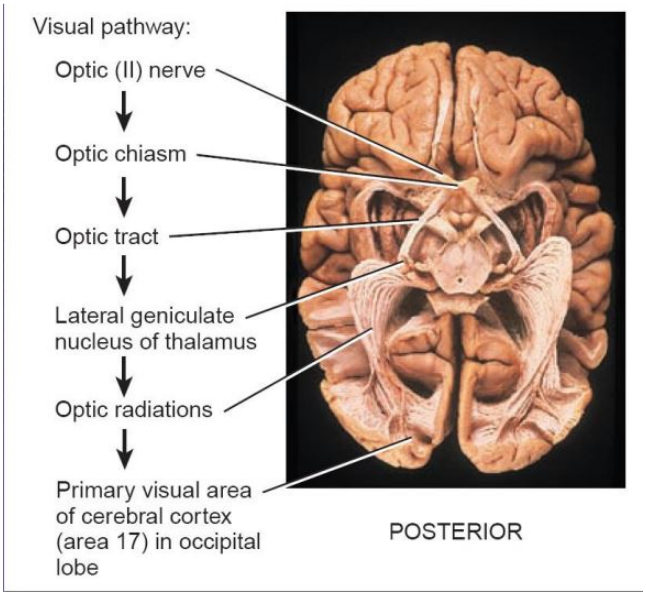
Parvocellular only

C: Visual pathways:

- **In this section Prof Loai mentioned the general vision pathway & some other pathways associated with its components.**

- **General Visual pathway**: impulses form **optic nerve (CNII)** will travel to **optic chiasm** and from here to the **optic tract** that ends in the **Lateral Geniculate nucleus of thalamus**, through **optic radiation** it will reach **primary visual area of cerebral cortex (Broadman area #17) in occipital lobe**.

- Remember that **nasal part of radiation crosses** to the other side in the optic chiasm, but **temporal part of radiation doesn't cross**.



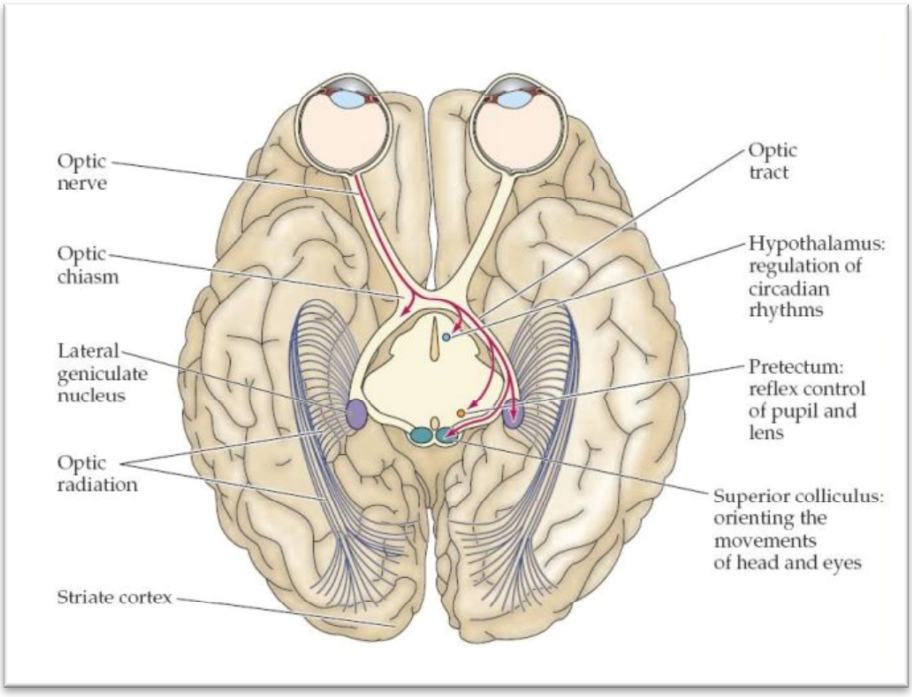
- Also note that the upper part of retina will go to the upper part of the visual area, but it receives the image from the lower part of the visual field.

- The temporal part of radiation is called **Meyer's loop** (its fibers are **not straight**; they curve and then get down. They are the higher part of optic radiation), it will go deep and anterior, it will reach almost to the end of temporal lobe until reaching the hippocampus, and any lesion to this area will damage the Meyer's loop that will lead to Upper **Quadrant anopia.**

- Radiation will come from both eyes, **nasal radiation** will **cross** while **the temporal won't cross**; through this the right visual field will go to the left & vice versa; the same principle applies in the retina.

- Damage in the optic radiation will lead to **hemianopina** in the **contralateral side.**

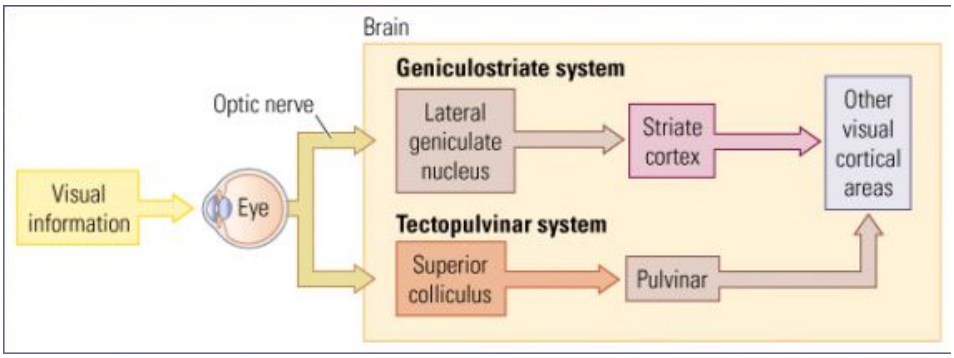
- **Other pathways**: a branch from the optic nerve will travel to the **LGB**, the optic nerve have **more than one target** after giving the optic chiasm:
- The **first target** is the **hypothalamus**, serving a function related to the **day-Night cycle (circadian rhythm)**, in this pathway there is a specific type of ganglion cells that will not receive from any receptor, this ganglion cell has **specific light detective proteins** called **Melanopsin,** and that's why they are called **Melanopsin ganglionic cell**, they will detect light and send the information to the hypothalamus. Its **noteworthy** that in cases of degeneration of rods or cons this pathway **will not be affected** and hence **its function won't be affected**, however **brain damage or retinal detachment** (loss of blood supply) will ultimately affect this pathway.



- The second target is the **mid-brain** , actually there are **two targets** in the midbrain ; the <u>first one</u> is to the **pre-tectum** and this is for **pupil reflex** and light reflex, the <u>second one</u> which is **more important** is to **the superior colliculus** (head movement and processing of visual information <u>motor-wise</u>) which is responsible for direct reflexes related to **head movement** and **eye movement** similar to the **tectospinal tract**, however it will yield more **processed information** that the cortex will harness to **direct** the direction of movement , it will ascend to the cortex through **pulvinar** in the thalamus then to **association cortex** of the vision(mainly **parietal lobe**)(since it's processed information) .

- The third target is to Lateral Geniculat nucleus (**LGN),** to striatal primary visual cortex.

- We have **two principle** branches of optic nerve pathway; one for movement and motion and depth, the other is mainly for information.



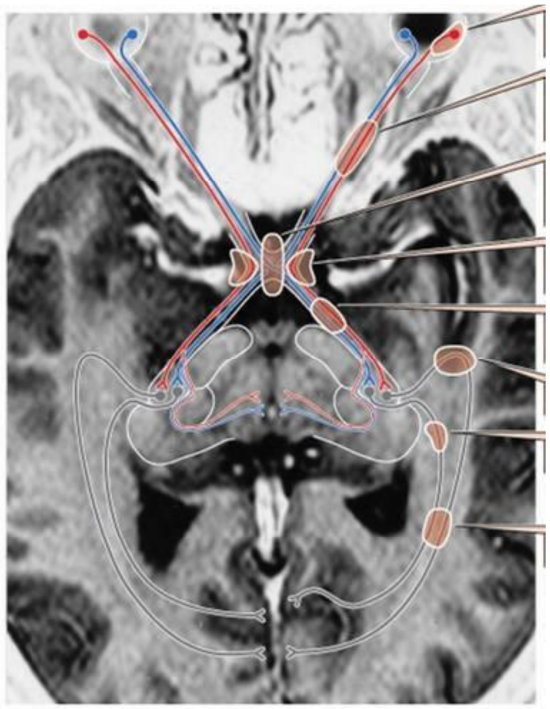
- **X ganglion impulses** will go exclusively to the lateral geniculate (**parvacellular layer**) and from there it will travel to **primary visual cortex** (broadman area**#17**) particularly **the 4th layer**(the destination of sensory information), through this pathway **colored and detailed vision is achieved** also information will travel in the **18th broadman area** (association area found in the **medial** and **ventral part** of the brain) after reaching area 17.This is called the **What pathway**, through which **analysis of images** and **pictures** take place (What do you see(shape) ? What's the color?).

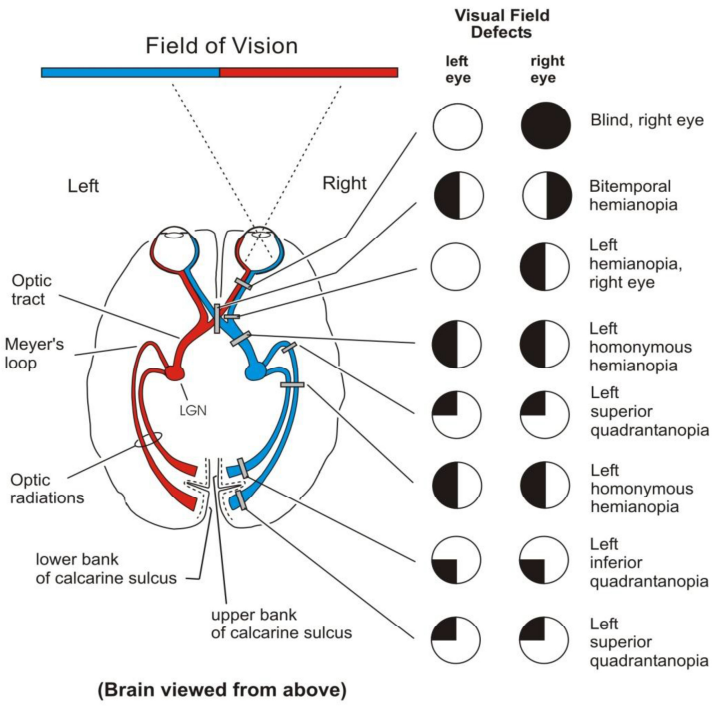
- **Y ganglion impulses** will travel mainly to the **magnocellular layer** of LGB, and from there to the **sensory radiation** to **broadman area#17 (primary visual cortex)** in cerebral cortex also in the **4th layer**, also it will travel to **association** areas (Broadman area**#18**) in **superior part of occipital lobe and posterior part of parietal lobe , and dorsal field** ; this is called **Where pathway**, it is related to **dynamic** and **motion** characteristics of objects and **spatial properties .**
  Also a little number of fibers travel through the superior colliculus.

- **W ganglion** & **small part of Y ganglion** will send impulses to the **superior colliculus**, and from there to **the pulvinar** and finally to **the association visual cortex** (also **layer 4)**.

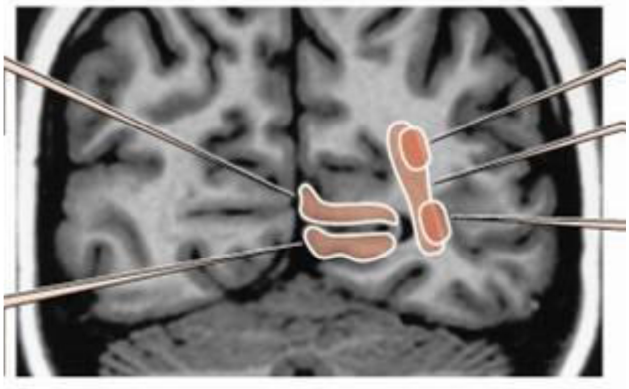
D: MRI:

In this MRI you can see damages to many areas
see the results of each damage from the picture next to it





In the coronal MRI we can see the Hippocampus and the damage will be in Meyer's loop, causing quadrantinopia (quadrant anopia).



- In the axial MRI in the level of temporal lobe we can see Meyer's loop, too.

Please refer to the slides

Special thanks to *Isam Bsisu*☺

أعتذر عن أي خطأ علمي أو لغوي.

زميلكم: علاء الدين دحبور