

# *Special sense*

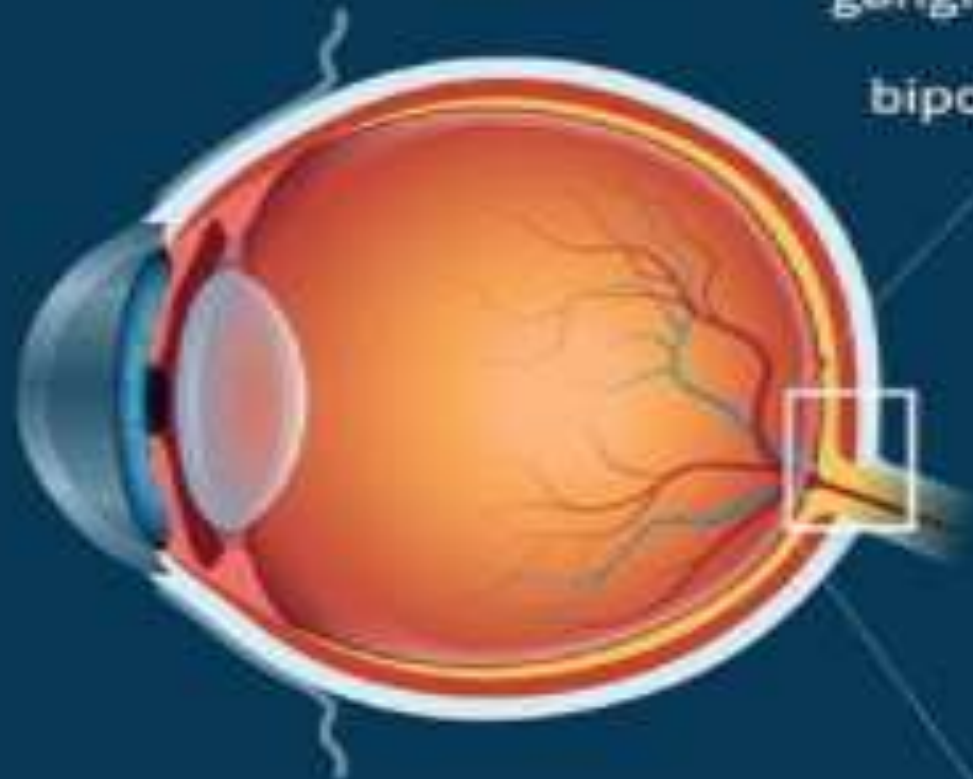
## *Vision*

*Lect. 3& 4*

*Prof . Dr. Huda Jabbar Al-bdery*

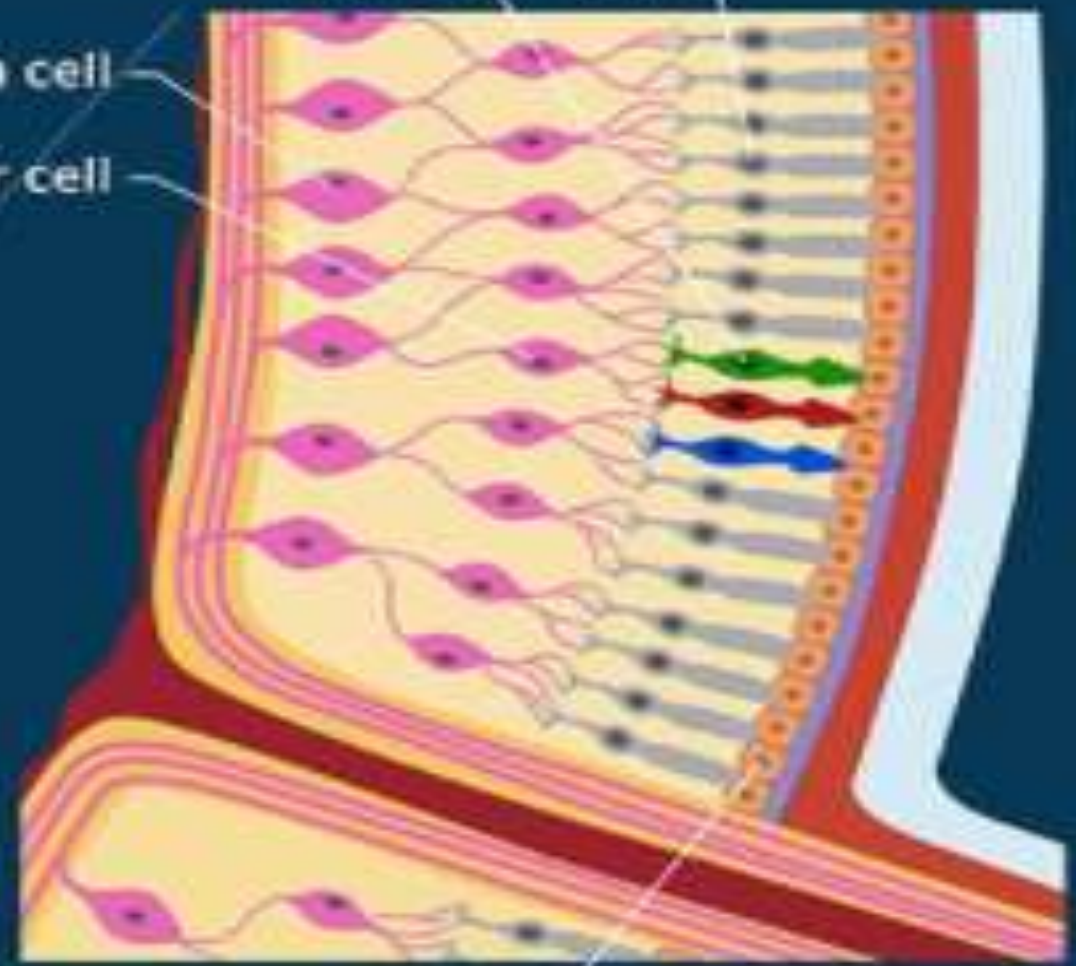


# Retina



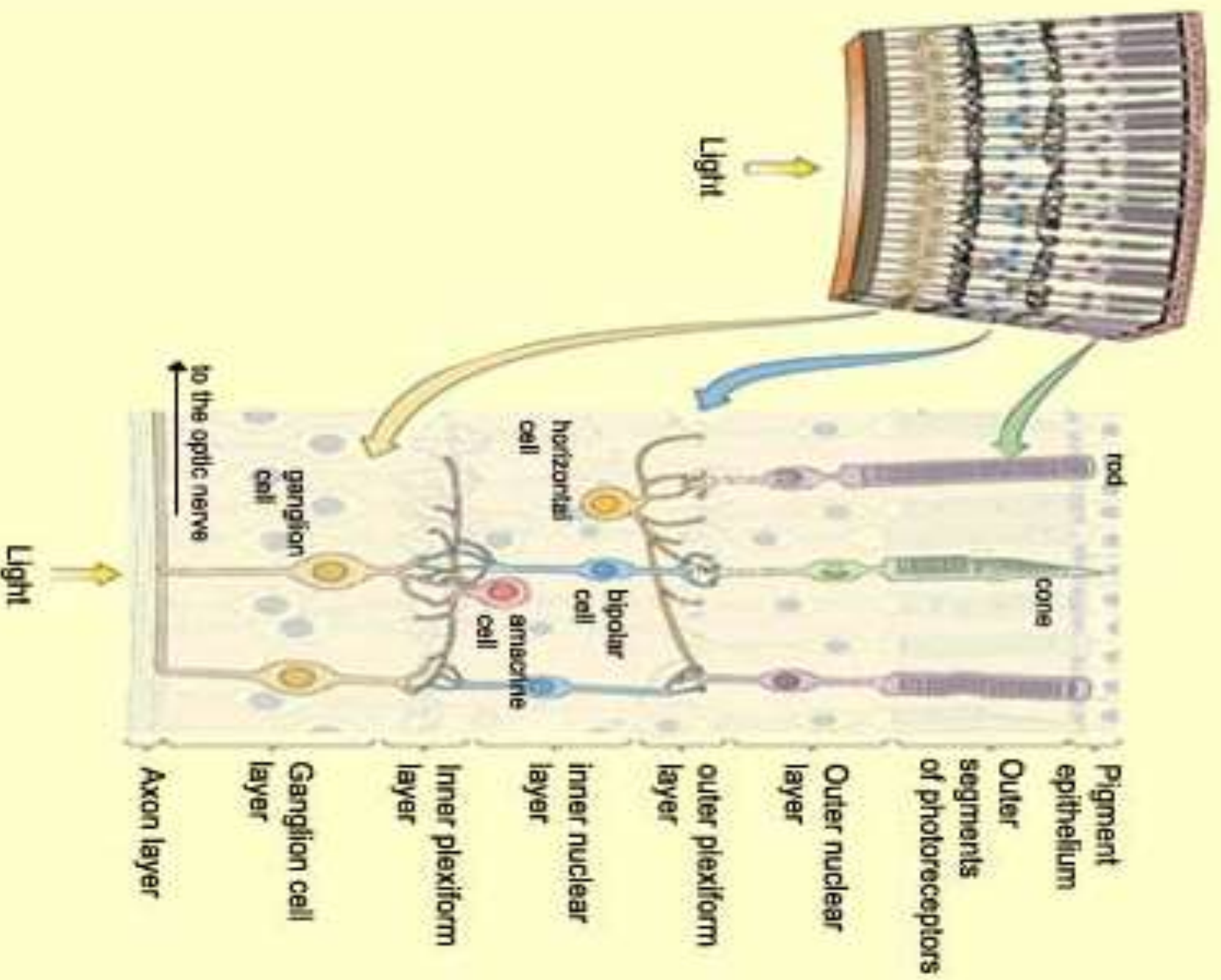
ganglion cell  
bipolar cell

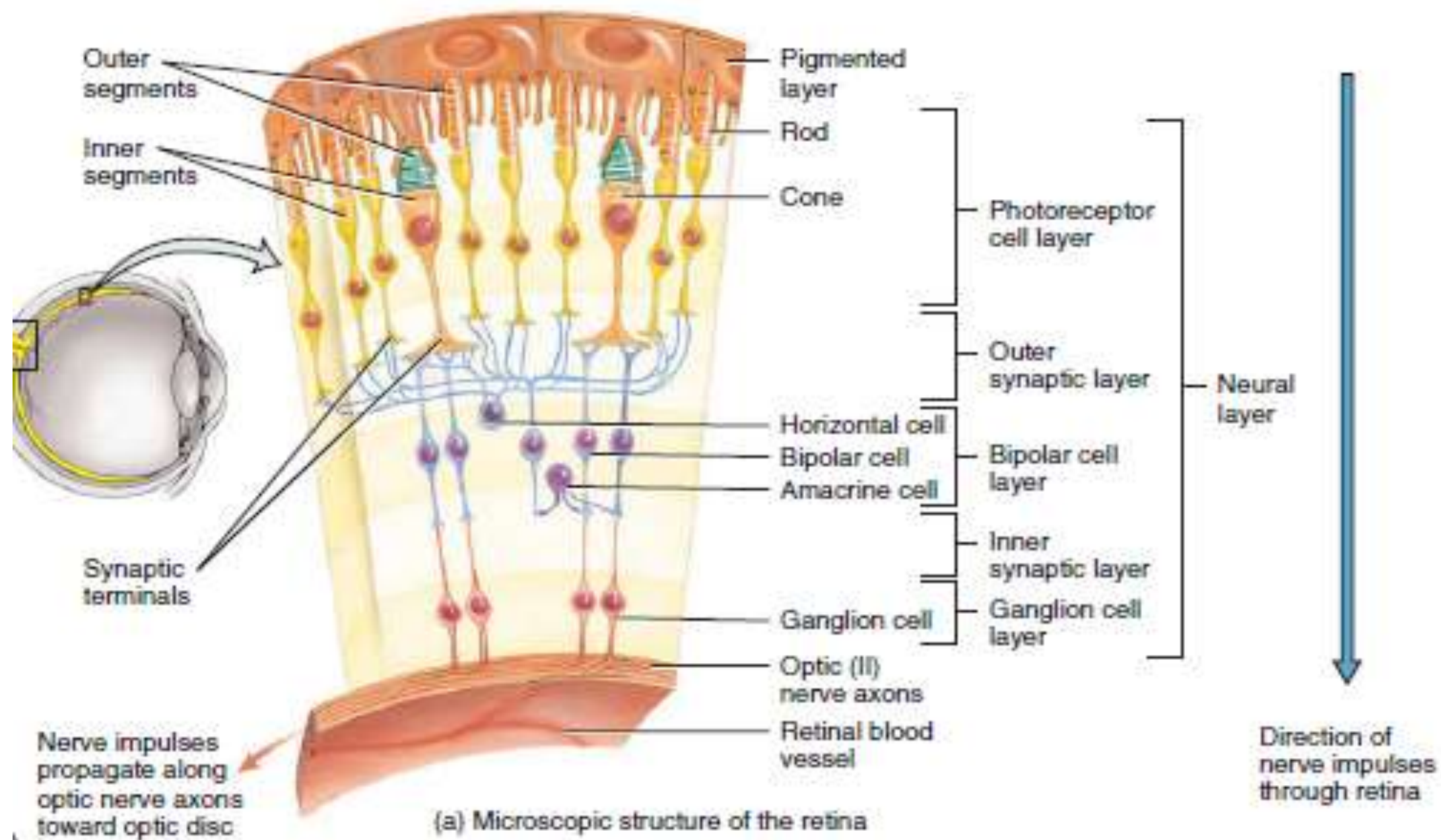
[photoreceptors]  
cone rod



retinal pigment epithelium (RPE)

Cross-section of the retina





# **Physiology of Vision**

## **Photoreceptors and Photopigments**

Rods and cones were named for the different appearance of the *outer segment*—the distal end next to the pigmented layer—of each of these types of photoreceptors. The outer segments of rods are cylindrical or rod-shaped; those of cones are tapered or cone-shaped. Transduction of light energy into a receptor potential occurs in the outer segment of both rods and cones. The photopigments are integral proteins in the plasma membrane of the outer segment. In cones the plasma membrane is folded back and forth in a pleated fashion; in rods the pleats pinch off from the plasma membrane to form discs. Photoreceptor outer segments are renewed at an astonishingly rapid pace.. The *inner segment* contains the cell nucleus, Golgi complex, and many mitochondria.

At its proximal end, the photoreceptor expands into bulblike synaptic terminals filled with synaptic vesicles.

The first step in visual transduction is absorption of light by a **photopigment**, a colored protein that undergoes structural changes when it absorbs light, in the outer segment of a photoreceptor.

Light absorption initiates the events that lead to the production of a receptor potential. The single type of photopigment in rods is **rhodopsin**. Three different **cone photopigments** are present in the retina, one in each of the three types of cones.

**All photopigments associated with vision contain two parts: a glycoprotein known as opsin and a derivative of vitamin A called retinal.**

## Rod

More **in number & most** are in peripheral part of retina

Important for retinal sensitivity

Specialized for night vision (scotopic vision )

Less----- edge detection  
acuity

color detection (shade of gray )

Contain rhodopsin

## Cone

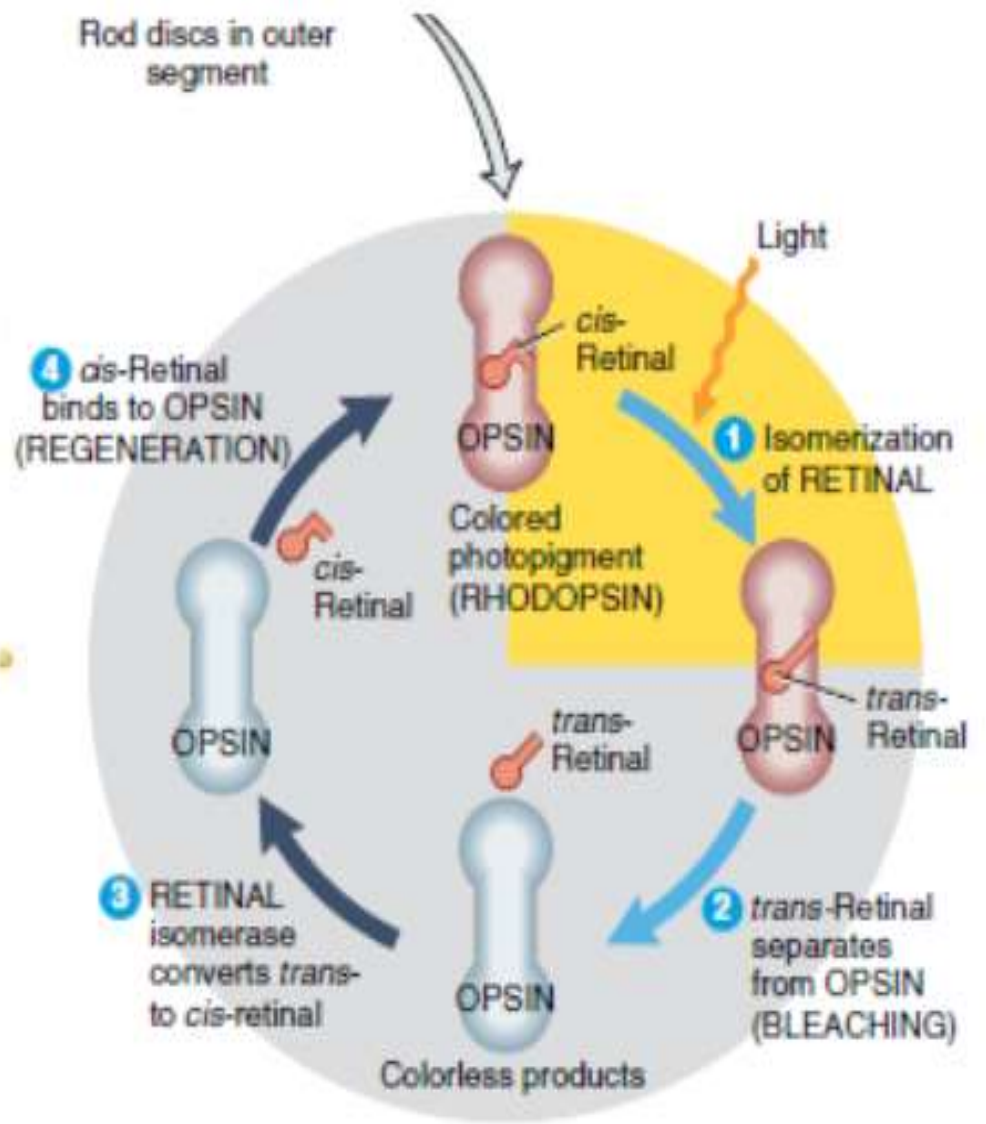
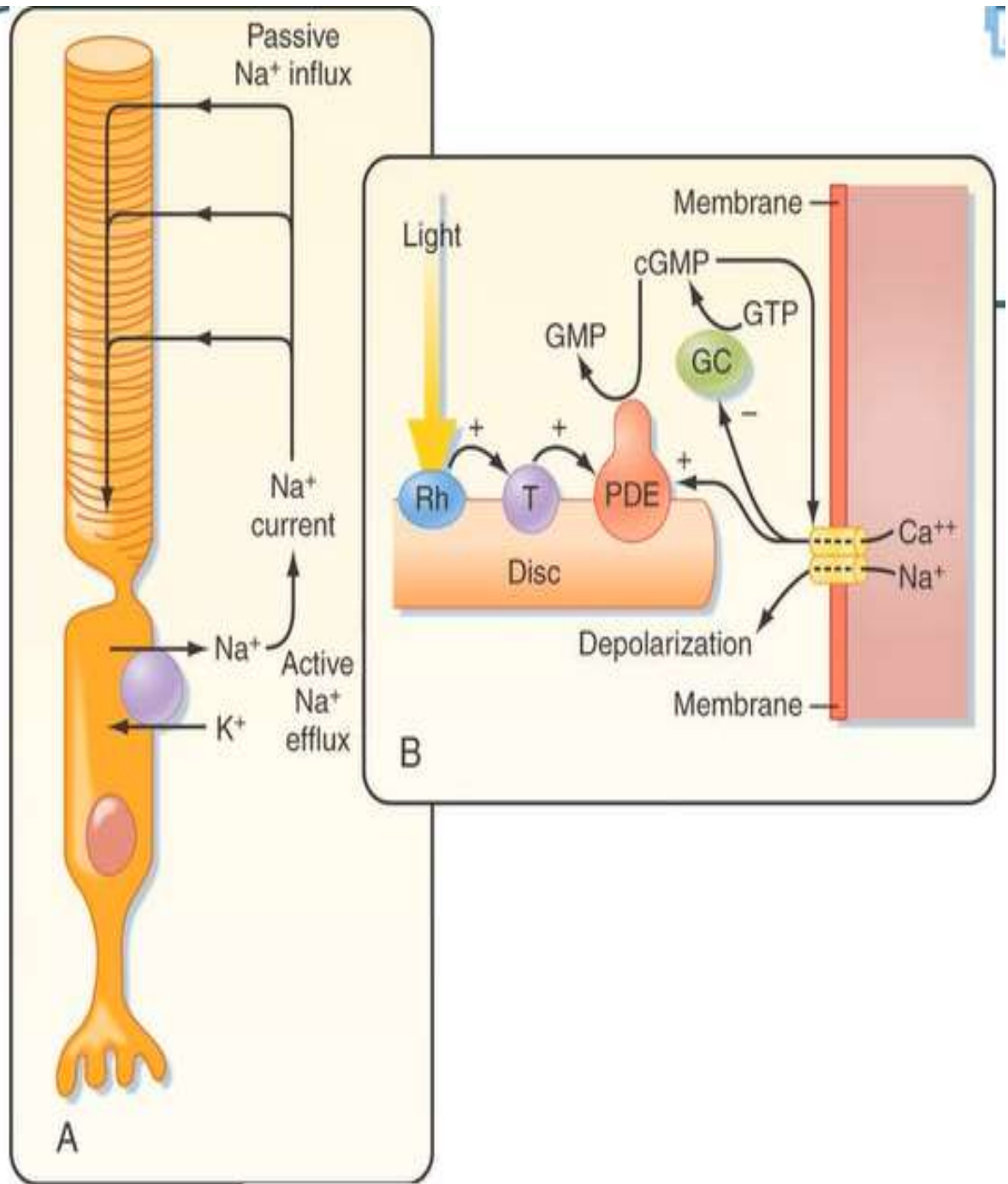
More in central part(fovea centralis)

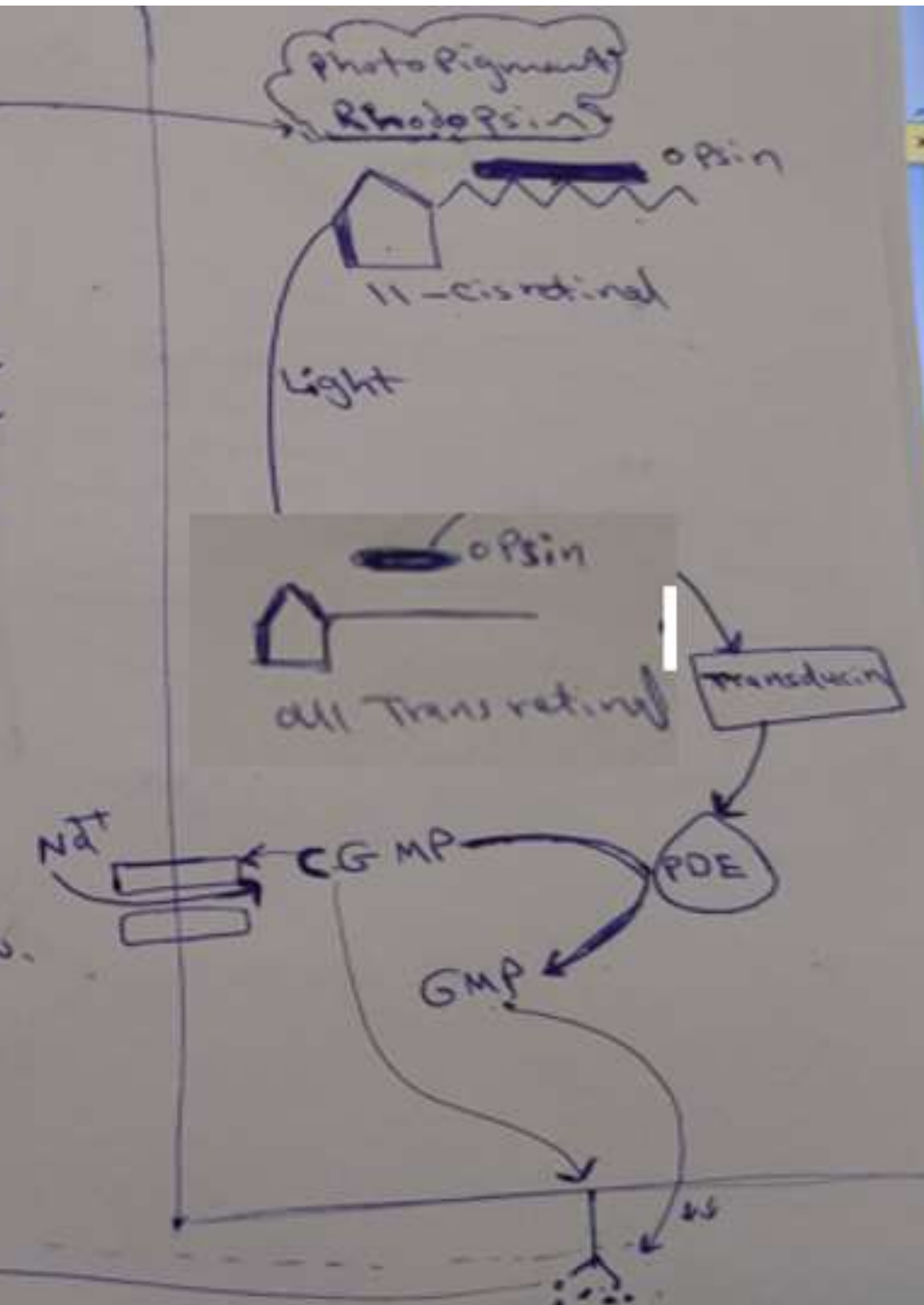
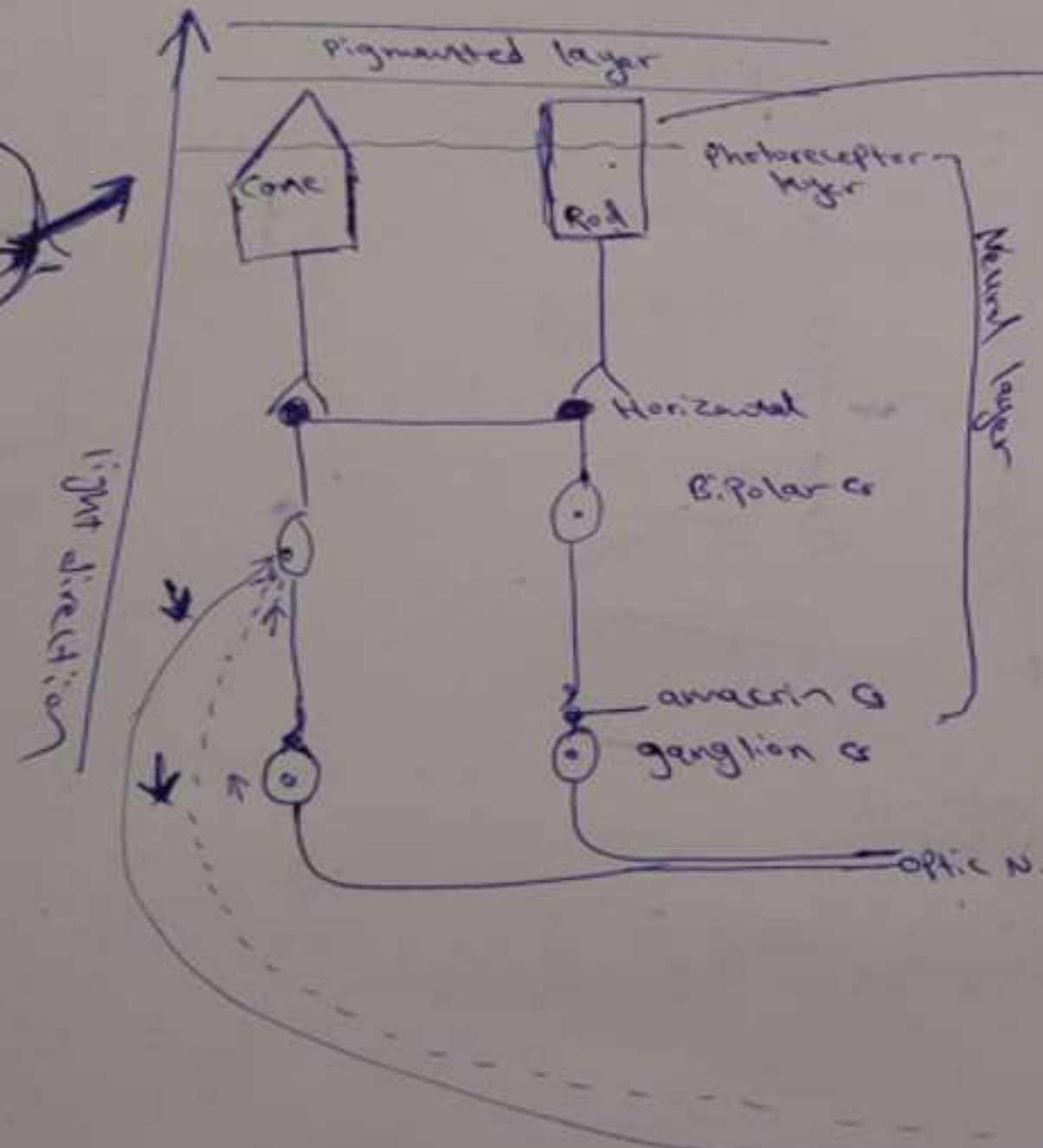
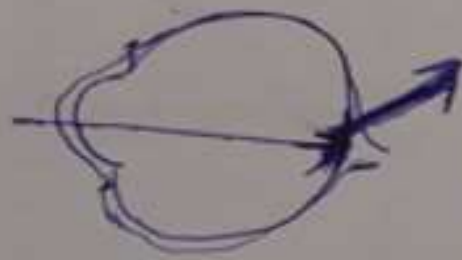
Visual acuity

Specialized for day vision (photopic vision )

High ----- edge detection  
acuity  
color detection

Contain different photopsine which response to different wave length of light







Below, we look at a tiny column of retina. The outer segment of the rod, closest to the back of the eye and farthest from the incoming light, is at the top.

**In the dark**      **In the light**

① cGMP-gated channels open, allowing cation influx. Photoreceptor depolarizes.

② Voltage-gated  $\text{Ca}^{2+}$  channels open in synaptic terminals.

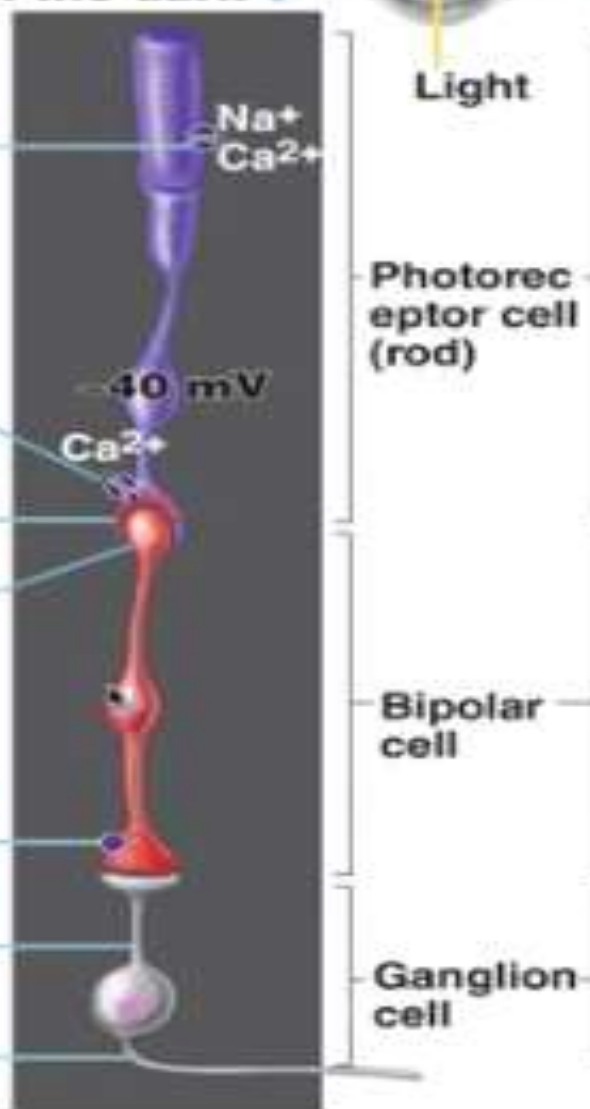
③ Neurotransmitter is released continuously.

④ Neurotransmitter causes IPSPs in bipolar cell. Hyperpolarization results.

⑤ Hyperpolarization closes voltage-gated  $\text{Ca}^{2+}$  channels, inhibiting neurotransmitter release.

⑥ No EPSPs occur in ganglion cell.

⑦ No action potentials occur along the optic nerve.



Light

Photoreceptor cell (rod)

Bipolar cell

Ganglion cell

Light

① cGMP-gated channels close, so cation influx stops. Photoreceptor hyperpolarizes.

② Voltage-gated  $\text{Ca}^{2+}$  channels close in synaptic terminals.

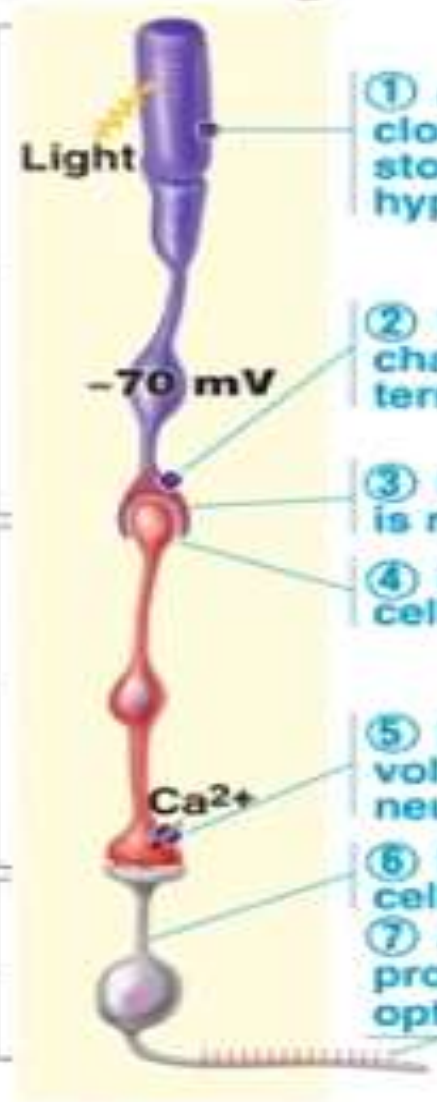
③ No neurotransmitter is released.

④ Lack of IPSPs in bipolar cell results in depolarization.

⑤ Depolarization opens voltage-gated  $\text{Ca}^{2+}$  channels; neurotransmitter is released.

⑥ EPSPs occur in ganglion cell.

⑦ Action potentials propagate along the optic nerve.



(Rhodopsin bleaching)



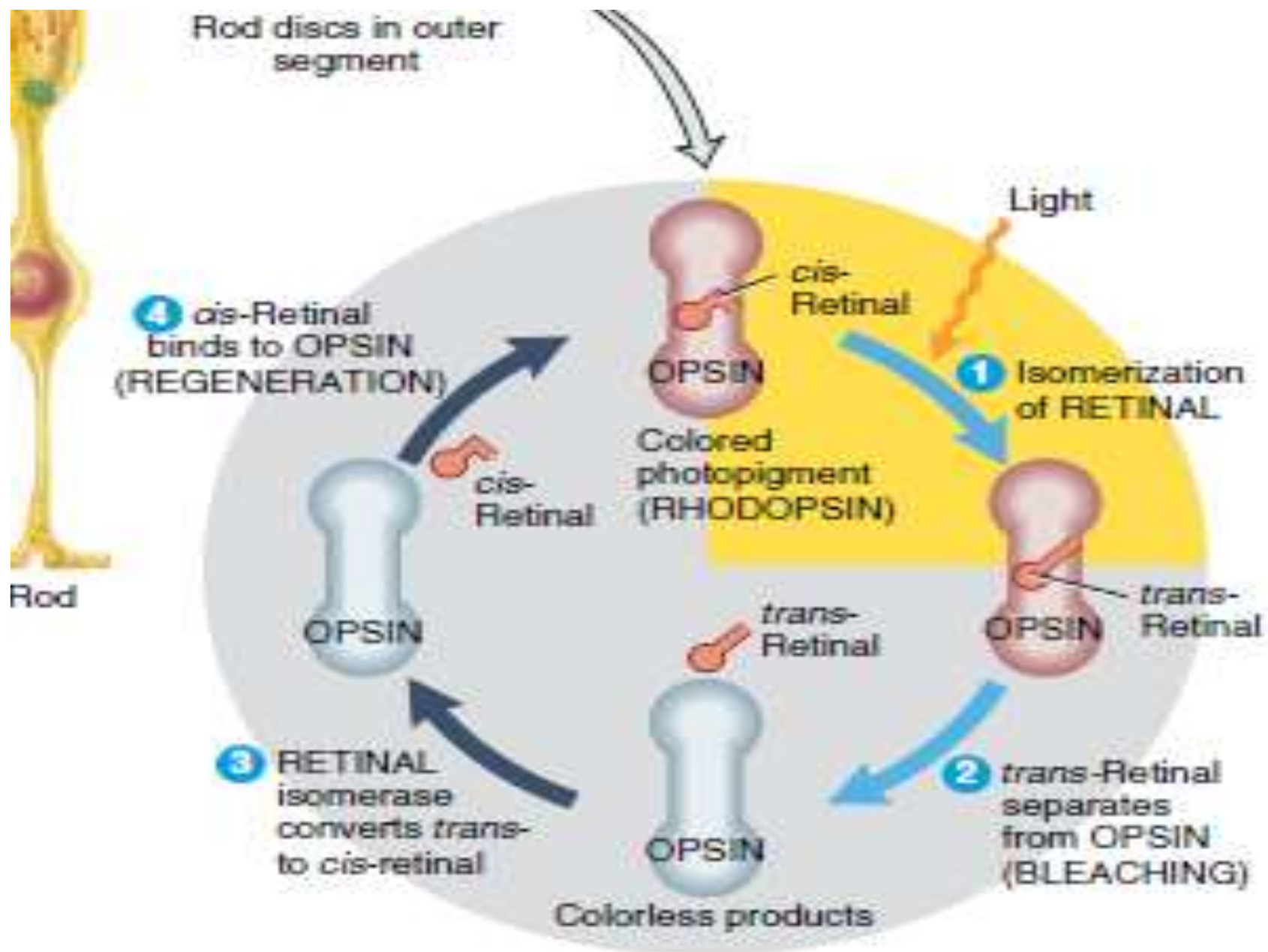
Retinal is the light-absorbing part of all visual photopigments

1- in darkness, retinal has a bent shape, called 11-*cis*-retinal, which fits snugly into the opsin portion of the photopigment. When 11-*cis*-retinal absorbs a photon of light, it straightens out to a shape called *trans*-retinal. This cis-to-trans conversion is called **isomerization** and is the first step in visual transduction. After retinal isomerizes, several unstable chemical intermediates form and disappear. These chemical changes lead to production of a receptor potential

2- in about a minute, *trans*-retinal completely separates from opsin. The final products look colorless, so this part of the cycle is termed **bleaching** of photopigment.

3- An enzyme called **retinal isomerase** converts *trans*-retinal back to *cis*-retinal.

The *cis*-retinal then can bind to opsin, reforming a functional photopigment. This resynthesis of a photopigment—is called **regeneration**..



The plasma membrane of a photoreceptor's outer segment contains chemically gated Na channels. Unlike other chemically gated channels that respond to extracellular chemical messengers, these channels respond to an internal second messenger, cyclic GMP, or cGMP (cyclic guanosine monophosphate). Binding of cGMP to these Na channels keeps them open. In the absence of light, the concentration of cGMP is high ). Light absorption leads to the breakdown of cGMP.) Therefore, the Na channels of a photoreceptor, unlike most receptors, are open in the absence of stimulation, that is, in the dark. The resultant passive inward Na leak, the so-called dark current, depolarizes the photoreceptor. The passive spread of this depolarization from the outer segment (where the Na channels are located) to the synaptic terminal (where the photoreceptor's neurotransmitter is stored) keeps the synaptic terminal's voltage-gated Ca channels open. Ca entry triggers the release by exocytosis of the neurotransmitter glutamate from the synaptic terminal while in the dark

**Photoreceptor Activity in the Light** On exposure to light, the concentration of cGMP is decreased through a series of biochemical steps triggered by photopigment activation. When 11-cis retinal absorbs light, it changes to the all-trans retinal conformation. This is the only light-dependent step in the entire process of phototransduction.

As a result of this change in shape, retinal no longer fits snugly in its binding site in opsin, causing opsin to also change its conformation, which activates the photopigment.

Photopigments are activated in response to light absorption by retinal. Rod and cone cells contain a G protein called transducin. The activated photopigment activates transducin, which in turn activates the intracellular enzyme phosphodiesterase. This enzyme degrades cGMP, thus decreasing the concentration of this second messenger in the photoreceptor.

During the light excitation process, the reduction in cGMP permits the chemically gated Na channels to close. This channel closure stops causing hyperpolarization. The hyperpolarization, passively spreads from the outer segment to the synaptic terminal of the photoreceptor. leads to closure of the voltage-gated Ca channels and a subsequent reduction in glutamate release from the synaptic terminal.

The hyperpolarizing potential and subsequent decrease in neurotransmitter release are graded according to the light intensity. The brighter the light is, the greater the hyperpolarizing response and the greater the reduction in glutamate release.

The retinal is converted back into its 11-cis form. In the dark, enzyme-mediated mechanisms rejoin opsin and this recycled retinal to restore the photopigment to its original inactive conformation

## ***Light and Dark Adaptation***

***When you emerge from dark surroundings (say, a tunnel) into the sunshine, light adaptation occurs—your visual system adjusts in seconds to the brighter environment by decreasing its sensitivity.***

***On the other hand, when you enter a darkened room such as a theater, your visual system undergoes dark adaptation—its sensitivity increases slowly over many minutes. The difference in the rates of bleaching and regeneration of the photopigments in the rods and cones accounts for some (but not all) of the sensitivity changes during light and dark adaptation.***

## Dark to light adaptation (S-cones)

① Pupil constricts in light  
Focus in macula (F.C.)

② Photopigment bleached

11-cis R  $\rightarrow$  all-trans R  
off  $\rightarrow$  on

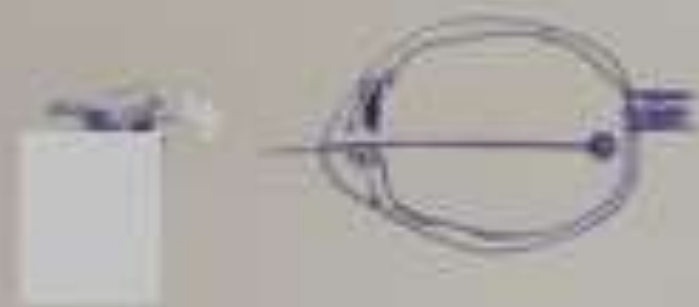
so Rod turned off

cone turned on

retinal sensitivity  $\downarrow$

visual acuity  $\uparrow$

color vision  $\uparrow$



## Light to dark adaptation

a pig is added  
to eye over time



$\rightarrow$  Rhodopsin (photopigment) (bleached)

Rod turned on

cone turned off

retinal sensitivity  $\uparrow$

visual acuity  $\downarrow$

color vision  $\downarrow$

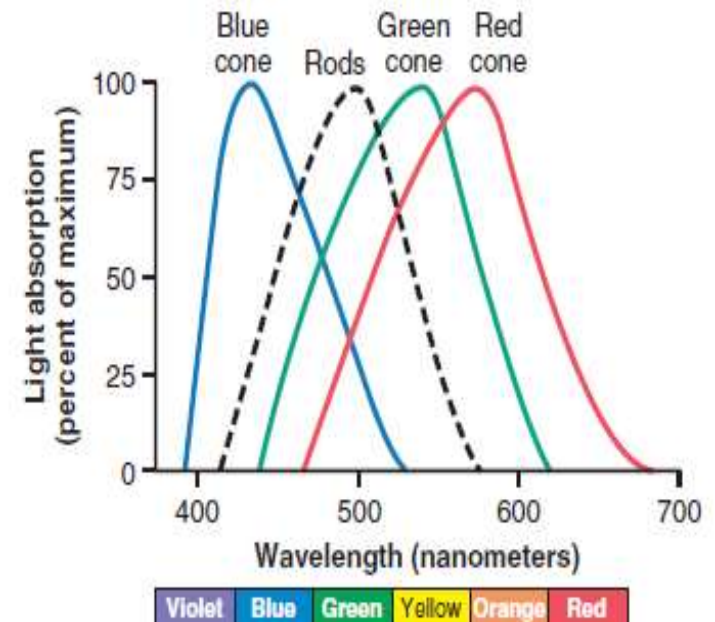


# Color vision

The *retinal* portion of all the visual pigments is exactly the same in the cones as in the rods. The color-sensitive pigments of the cones, therefore, are combinations of retinal and photopsins.

There are three types of color pigments in cones, but only one is present in each of the different cones, thus making the cones selectively sensitive to different colors: **blue**, **green**, or **red**. These color pigments are called, respectively, *blue-sensitive pigment* or called **S pigment**, *green-sensitive pigment* (**M pigment**), and *red-sensitive pigment* (**L pigment**). The absorption characteristics of the pigments in the three types of cones show peak absorbencies at light wavelengths of 445, 535, and 570 nanometers, respectively. These wavelengths are also the wavelengths for peak light sensitivity (spectral sensitivity) for each type of cone, which begins to explain how the retina differentiates the colors.

the absorption curve for the rhodopsin of the rods, with a peak at 505

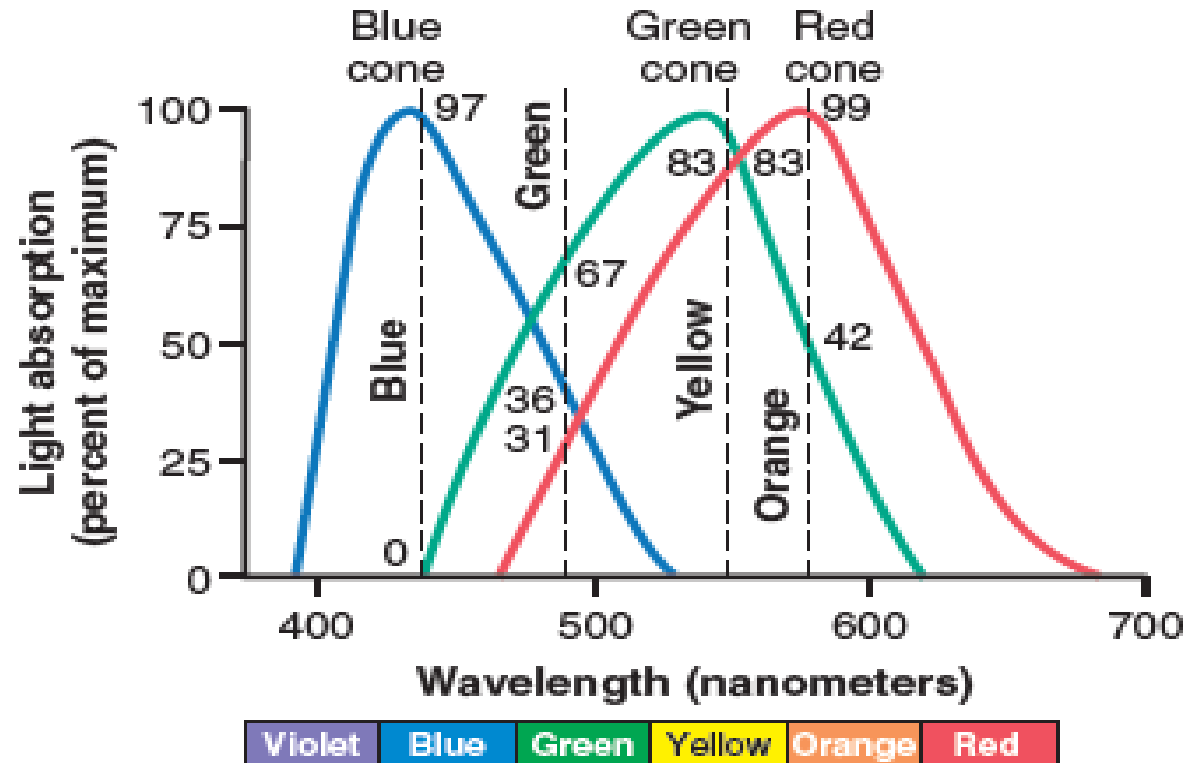


# Interpretation of Color in the Nervous System.

The orange monochromatic light with a wavelength of 580 nanometers stimulates the red cones to a value of about 99 (99 percent of the peak stimulation at optimum wavelength); it stimulates the green cones to a value of about 42, but the blue cones are not stimulated at all. Thus, the ratios of stimulation of the three types of cones in this instance are 99 : 42 : 0. The nervous system interprets this set of ratios as the sensation of orange.

Conversely, a monochromatic blue light with a wavelength of 450 nanometers stimulates the red cones to a stimulus value of 0, the green cones to a value of 0, and the blue cones to a value of 97.

This set of ratios—0 : 0 : 97—is interpreted by the nervous system as blue. Likewise, ratios of 83 : 83 : 0 are interpreted as yellow, and ratios of 31 : 67 : 36 are ir



- **Perception of White Light.** About equal stimulation of all the red, green, and blue cones gives one the sensation of seeing white. Yet, there is no single wavelength of light corresponding to white; instead, white is a combination of all the wavelengths of the spectrum. Furthermore, the perception of white can be achieved by stimulating the retina with a proper combination of only three chosen colors that stimulate the respective types of cones about equally.

## Color Blindness

Gene for blue sensitive S cone pigment is on chromosome 7. Gene for red & green sensitive cone pigment is on X chromosome. When a single group of color receptive cones is absent (due to absence of their gene) the person cannot see or distinguish some colors from others.

### Red-Green Color Blindness.

When a single group of color-receptive cones is missing from the eye, the person is unable to distinguish some colors from others.

Green, yellow, orange, and red colors, which are the colors between the wavelengths of 525 and 675 nanometers, are normally distinguished from one another by the red and green cones. If either of these two cones is missing, the person cannot use this mechanism for distinguishing these four colors; the person is especially unable to distinguish red from green and is therefore said to have *red-green color blindness*.

A person with loss of red cones is called a *protanope*; the overall visual spectrum is noticeably shortened at the long wavelength end because of a lack of the red cones. A color-blind person who lacks green cones is called a *deutanope*; this person has a perfectly normal visual spectral width because red cones are available to detect the long wavelength red color.

Red-green color blindness is a genetic disorder that occurs almost exclusively in males. That is, genes in the female X chromosome code for the respective cones. Yet color blindness almost never occurs in females because at least one of the two X chromosomes almost always has a normal gene for each type of cone. Because the male has only one X chromosome, a missing gene can lead to color blindness.

Because the X chromosome in the male is always inherited from the mother, never from the father, color blindness is passed from mother to son, and the mother is said to be a *color blindness carrier*; about 8 percent of all women are color blindness carriers.

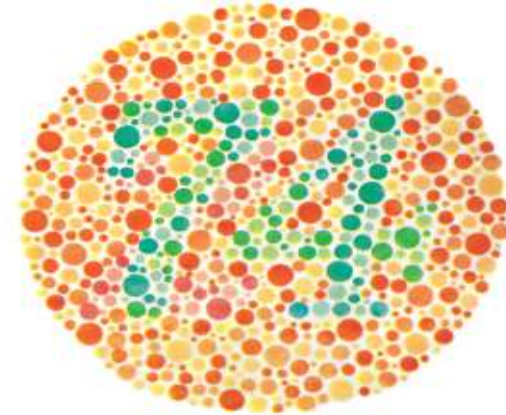
**Blue Weakness.** Only rarely are blue cones missing, although sometimes they are underrepresented, which is a genetically inherited condition giving rise to the phenomenon called *blue weakness*.

**Color Test Charts.** A rapid method for determining color blindness is based on the use of spot charts such .

These charts are arranged with a confusion of spots of several different colors.

a person with normal color vision reads “74,”

whereas a red-green color-blind person reads “21.”

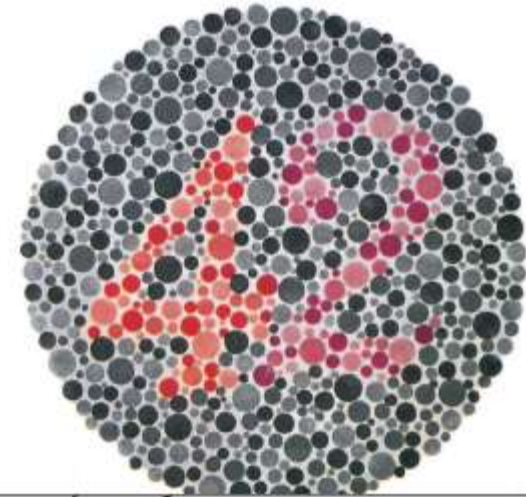


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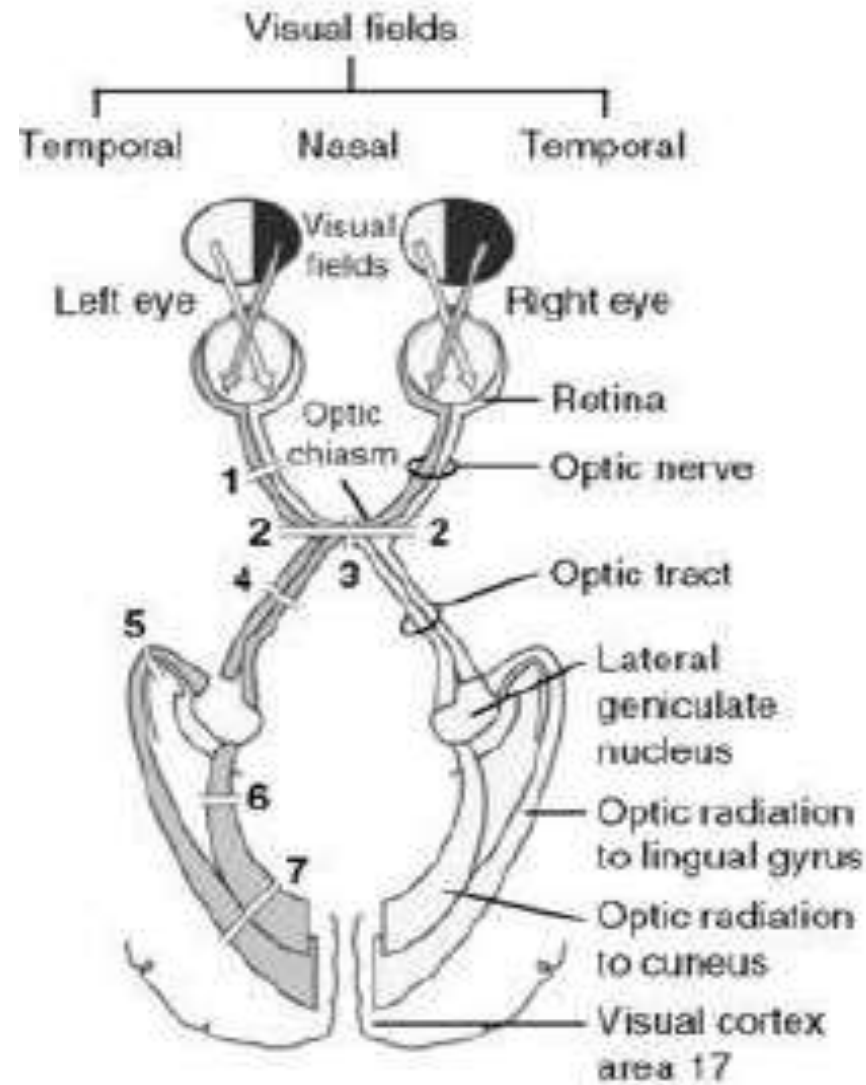
a person with normal color vision reads “42,”

whereas a red-blind person reads “2,”

and a green-blind person reads “4.”



# Visual pathway & defects



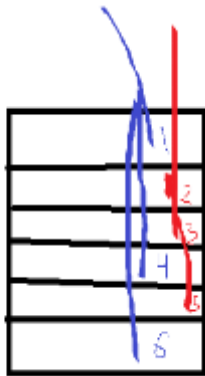
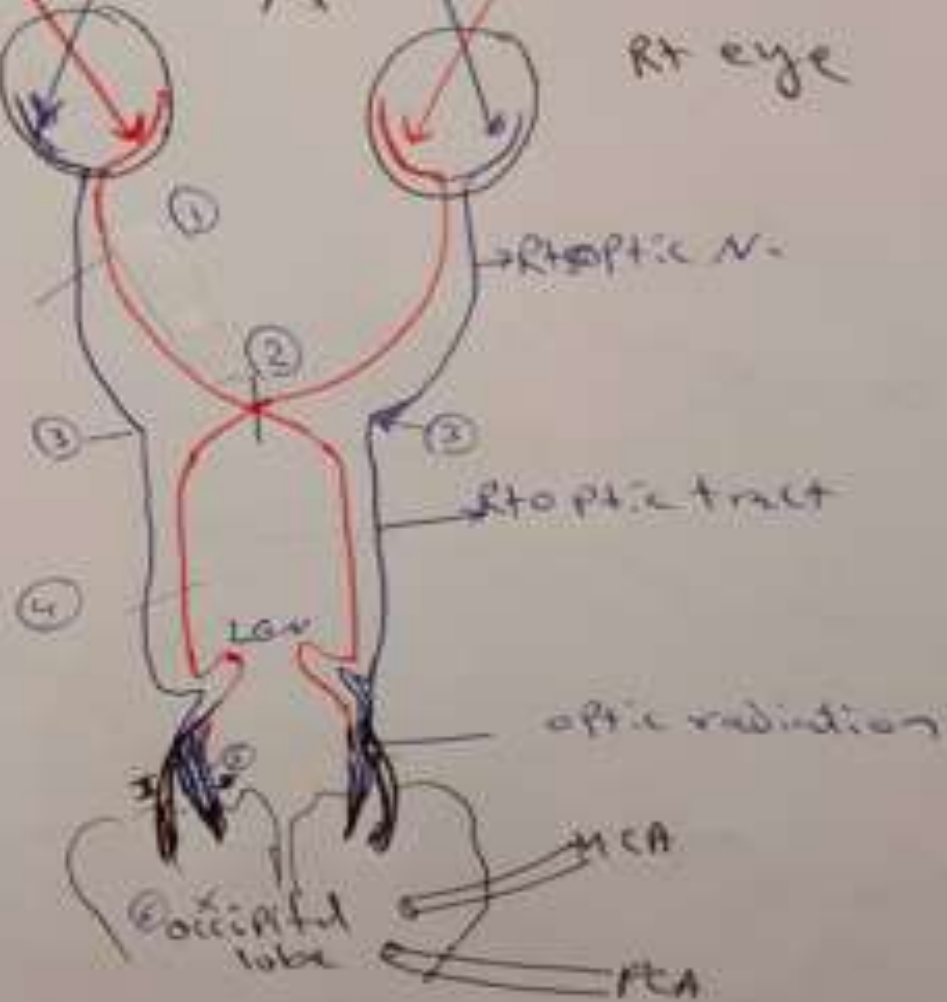
Lesion	Visual defect
1. Optic nerve	Ipsilateral blindness 
Optic chiasm 2. Bilateral lateral compression	Binasal hemianopia 
3. Midsagittal transection/pressure	Bitemporal hemianopia 
4. Optic tract (left)	Right homianopia 
Optic radiation (left) 5. Lower division	Right upper quadrantanopia 
6. Upper division	Right lower quadrantanopia 
7. Both divisions	Right hemianopia with macular sparing 

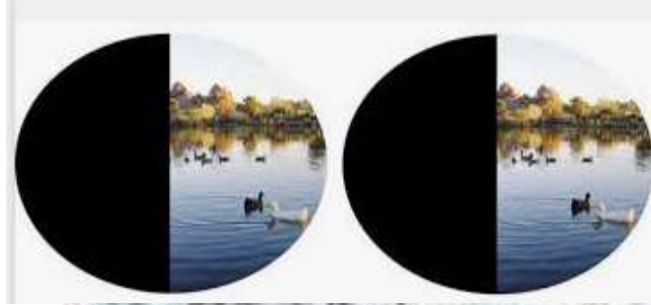
Lt v. Field

Rt v. Field

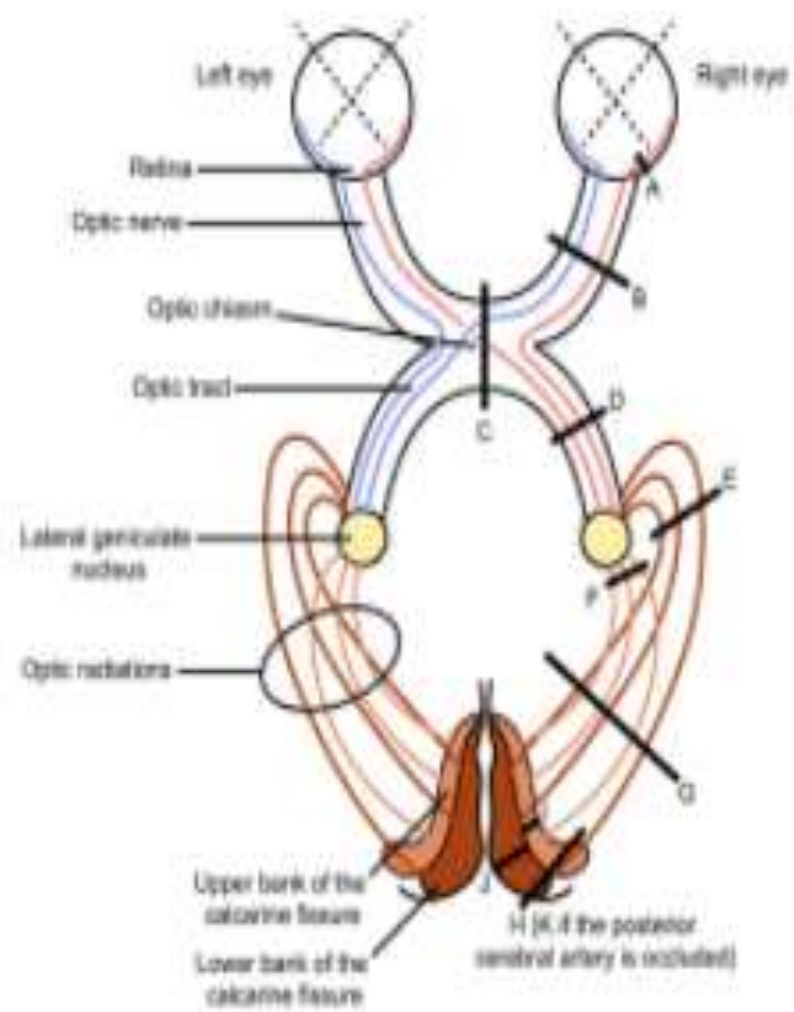


Rt eye





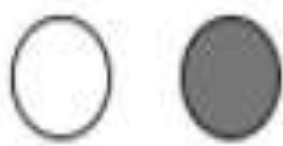




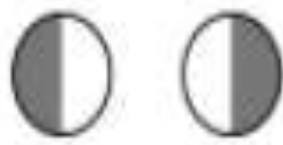
A) Central scotoma



B) Monocular vision loss



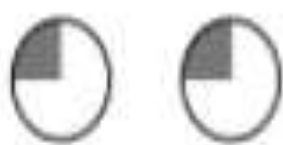
C) Bitemporal hemianopia



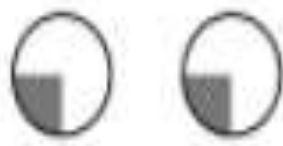
D, G, & H) Contralateral homonymous hemianopia



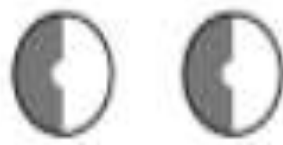
E & J) Contralateral superior quadrantanopia



F & I) Contralateral inferior quadrantanopia



K) Contralateral homonymous hemianopia with macular sparing



Thank

you.

