PHYSIOLOGY

SHEET NO. 8

WRITER : 018 sheets

CORRECTOR : Sanad Haddad

DOCTOR : Dr. Faisal Mohammad

Dark and light adaptation

	Dark adaptation	**Light adaptation**
Sensitivity of the retinal automatically adjusts to the light level	In dark conditions retinal is converted back to rhodopsin. (increasing formation of the pigment -> increasing the sensitivity)	In light conditions most of the rhodopsin has been reduced to retinal so the level of photosensitive chemicals is low. (decreasing the sensitivity)
Explanation	When you enter a dark area, the 1st pigment that starts to form is in the cones (they have fast response but low number). When cones finish the synthesis, rods start (they have slow response but high number) and the synthesis peaks after 30-40 minutes. Therefore, after 30-40 minutes you start seeing clearly.	When you enter a lighted area where the stimulus (light) is very strong, all the pigment is decomposed, decreasing the sensitivity of the retina starting with the cones (low number) then the rods (high number).
Opening and closing of the pupil also contributes to adaptation because it can adjust the amount entering the eye	Mydriasis	Miosis

[sensitivity is directly proportional to amount of pigment

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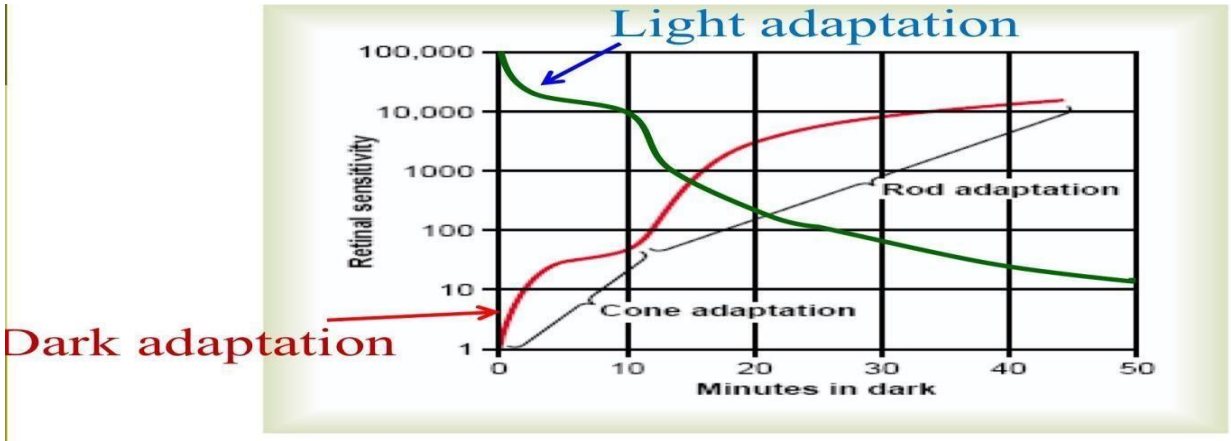


Figure 50–8

Dark adaptation, demonstrating the relation of cone adaptation to rod adaptation.

Notes on the figure:
In an area with a good amount of light, retina sensitivity is low because rhodopsin is depleted
When the person enters a dark area he cannot see well, the small amount of light does not reach the pigment, hyperpolarization will not take place.
Dark Adaptation
First part of dark adaptation is due to cones adaptation. Cones have faster formation and faster decay, in terms of 1 minute, sensitivity increases 20-50 times.
Second Part, rods start to form, they have slower formation but last longer. Around 20-30 min, sensitivity of retina increases because of rhodopsin reformation. That is why when you enter into a dark area, first 1-2 min you can see, but with time you start seeing better, until 20-30 min, you can see good (the pigment in rods has been formed)

Light Adaptation:
In a dark room: rhodopsin is increased, the sensitivity of retina is high. When you enter an area with lots of light, you cannot open your eyes properly. First thing stimulated is cones, decrease in amount of photopsin. Then rods stimulated

This sheet is written based on lecture 6 video and the live meeting.

Dark and Light Adaptation

Last lecture we talked about dark and light adaptation, let's have a quick recap:

- Light adaptation: if a person remains in light for long time → decomposition of large proportions of photochemicals in rods and cons → the sensitivity of the retina to light is reduced.
- Dark adaptation: If this person then enters a dark room, the retinal and opsins in the rods are converted back into light-sensitive pigments → the sensitivity of retina to light increases with time to several folds and the image becomes clearer.
  - ✓ Maximal sensitivity is reached in 20 min.
- Adaptation occurs four times more rapidly in cones than in rods, so increased pigment in cones produces slight dark adaptation in first 5 min.
- Increased rhodopsin in rods produces greater increase in sensitivity (they contribute for 100,000-fold increase in light sensitivity), and this is due to the convergence of large numbers of rods and the summation of signals.
- The sensitivity of retina is proportional to the logarithm of pigment concentration.
- The other part of adaptation involves changing the diameter of pupils as follow: in the dark they dilate, and in the light they constrict.
- Also the neuron contributes to adaptation (Corticofugal information that increases sensitivity)

The importance of dark and light adaptation

- ✓ The detection of images on the retina is a function of discriminating between darkand light spots. So, It is important for the sensitivity of the retina to be adjusted todetect the dark and light spots on the image.
- ✓ When you get exposed to the sun after leaving the movie theater, even the dark spots appear bright leaving little contrast. This poor vision remains until the retina has adapted sufficiently to light.
- ✓ Conversely, when you enter somewhere dark after being exposed to light, the light spots are not enough to register, because the sensitivity of retina is so slight.After dark adaptation, the light spots begin to register.

Color vision

Color vision is the result of activation of cones.

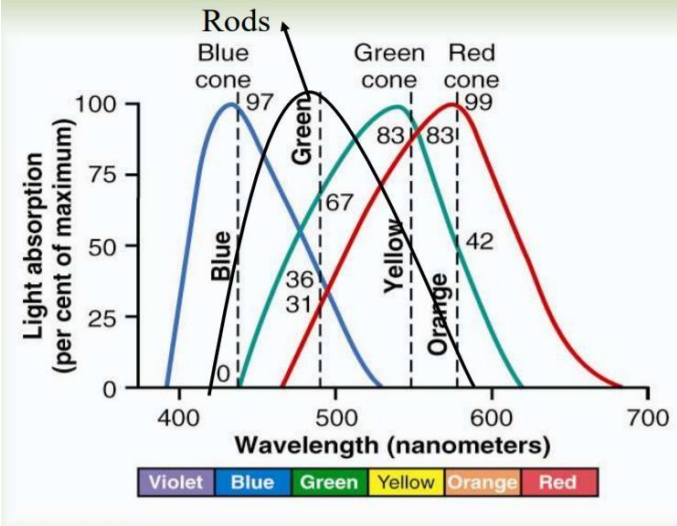
- 3 types of cones:

- blue cone
- green cone
- red cone

If deficiency of any of these cones, we have color blindness
Green and Red color blindness are sex-linked
Blue is autosomal, not that common. Green and Red blindness are more common
The wavelengths below blue (ultraviolet) and the wavelengths above red (infrared) are not visible for us.

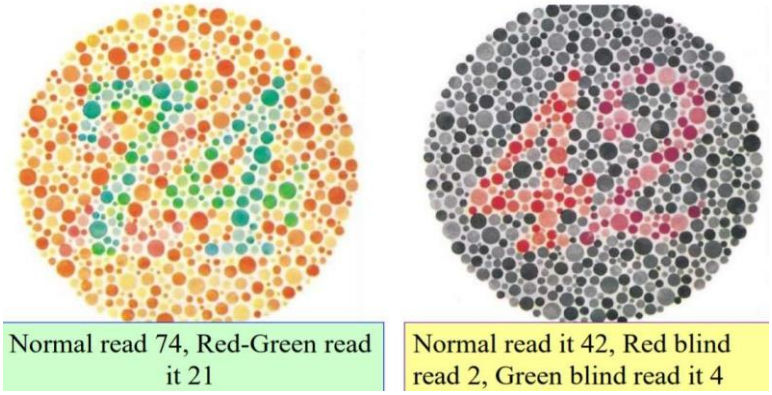
The pigment portion of the photosensitive molecule (11-cis retinal) is the same in the cones as in the rods. The protein portion is different for the pigment molecule in each of the cones, which makes each cone receptive to a particular wavelength of light.

- As you can see in the figure, blue cones absorb light at wavelength between 400-550 nm, green cones at 440-630 nm, and red cones at 470-680 nm.
- Rods have one range of wavelength, enabling you to see in black and white.
- Each color has specific combination of stimulation of these three cones. For example, when both the green cones and the red cones are stimulated in certain percentages, you will see yellow color.
- For example green color: stimulates red (at 31%), blue(36%), and green cone(76%). This combination is specific for this color.



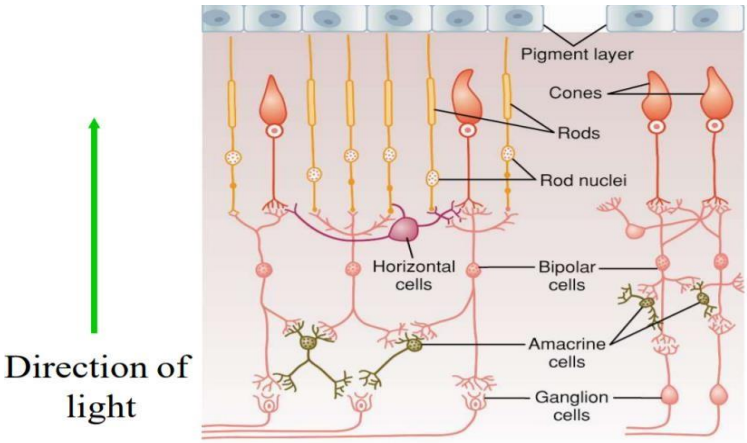
Color Blindness

- It's a genetic disorder passed along on the X chromosome. So it occurs almost exclusively in males.
- about 8% of women are color blindness carriers.
- It's due to lack of a particular type of cone.
- most color blindness results from lack of the red or green cones.
  lack of a red cone → **Protanope**.
  lack of a green cone → **Deuteranope**.
- Blue color blindness is an autosomal recessive gene (not X-linked) but it's rare.
- The test of color blindness is done using **Ishihara charts**, which are arranged with confusion of spots with several different colors. Below are two examples of these charts.



A color blind person, has a difficult ability to discriminate colors when they are close to each other. Colors are put in circles and form a number, so in the retina there could be discrimination between colors. A color blind person cannot discriminate.
If you ask a person to tell what a single color is on its own, the person can know the color in general or its shade, but finds it difficult to tell apart a certain color if many colors are together.

◆ *Neural function of the retina*

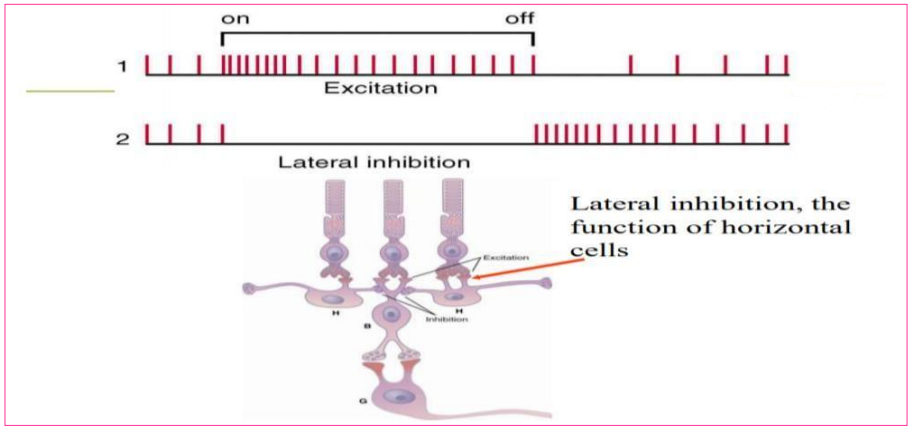


The different neuronal cell types are as follows:

1. The photoreceptors – the rods and cones- which synapse with bipolar cells and horizontal cells.
2. The horizontal cells, which connect laterally between the synaptic bodies of the rods and cones and with bipolar cells. The output of these cells is always inhibitory.

   The function of horizontal interneurons is lateral inhibition. When the light is focused on the retina, the visual pathway from the central area is excited, whereas the area on the sides is inhibited by these interneurons. This process is very

important for the sharpness of vision by transmitting contrast borders in thevisual image.



3. The bipolar cells, which synapse with ganglion cells and amacrine cells.
4. The amacrine cells between bipolar and ganglion cells, which also contribute to the lateral inhibition and the sharpness of vision. They are different than horizontal cells because they havemany types and can be stimulated by different kinds of visual stimuli.

5. The ganglion cells, which transmit output signals from the retina through the optic nerve into the brain.

- Blind spot is located about 15 degrees lateral to the central point of vision. no rods or cones in this area. it's called the optic disc, and it's the exit point for axons of the ganglion cells.

  Transmission of signals in retina is by electrotonic conduction
- Rods and cons and bipolar cells respond to light by generating **receptor potential**. This allows graded response that is proportional to light intensities. *

  The only cells which generate action potential are ganglion cells, and some amacrine cells.

  *When the receptor potential increases with the increase in light intensity, the number of action potentials generated in ganglion cells increases.

✍ Note: receptor potential (i.e. graded/ electrogenic potential): a non-propagated local potential, resulting from a local change in ionic conductance.
action potential: an 'all or none' propagated impulse.

Function of Amacrine Cells

- There are about 30 different types of amacrine cells, each one responds for one type of stimulation.
- Some are involved in the direct pathway from rods to bipolar to amacrine to ganglion cells.
- Some amacrine cells respond strongly to the onset of the visual signal, some to the extinguishment of the signal.
- Some respond to the movement of the light signal across the retina.
- Amacrine cells are a type of interneuron that aid in the beginning of visual signal analysis.

Note: the doctor didn't focus on the details, it's enough to know that there are many types of amacrine cells.

Three Types of Ganglion Cells

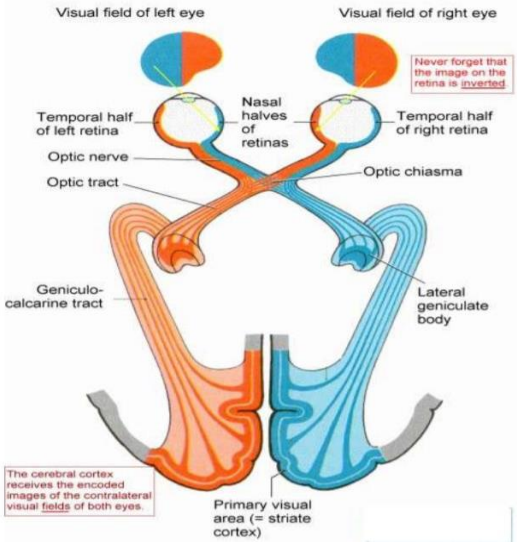
- W cells (40%): they receive most of their excitation from rod cells. They are sensitive to directional movement in the visual field.
- X cells (55%): they have small receptive field & discrete retinal locations. They might be responsible for the transmission of the visual image itself, always receive input from at least one cone, & may be responsible for color transmission.

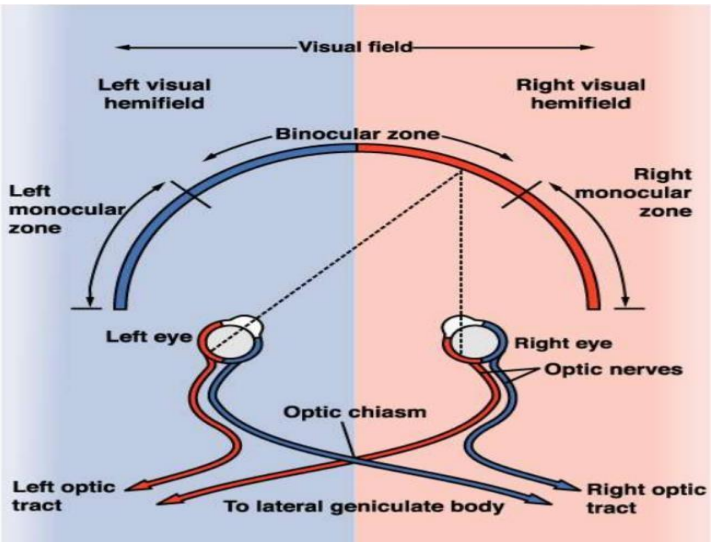
  Small receptive field → more sharpness of the image
- Y cells (5%): they have large receptive field and respond to instantaneous changes in the visual field.

Excitation of Ganglion Cells

- They are spontaneously active with continuous action potentials (they have basal rate of firing), and this is important for having positive and negative control.
- visual signals are superimposed on this background.
- many are excited by changes in light intensity.
- they respond to contrast borders, especially from the cons, this is the way the pattern of the scene is transmitted to the brain.

◆ *Fields of vision*





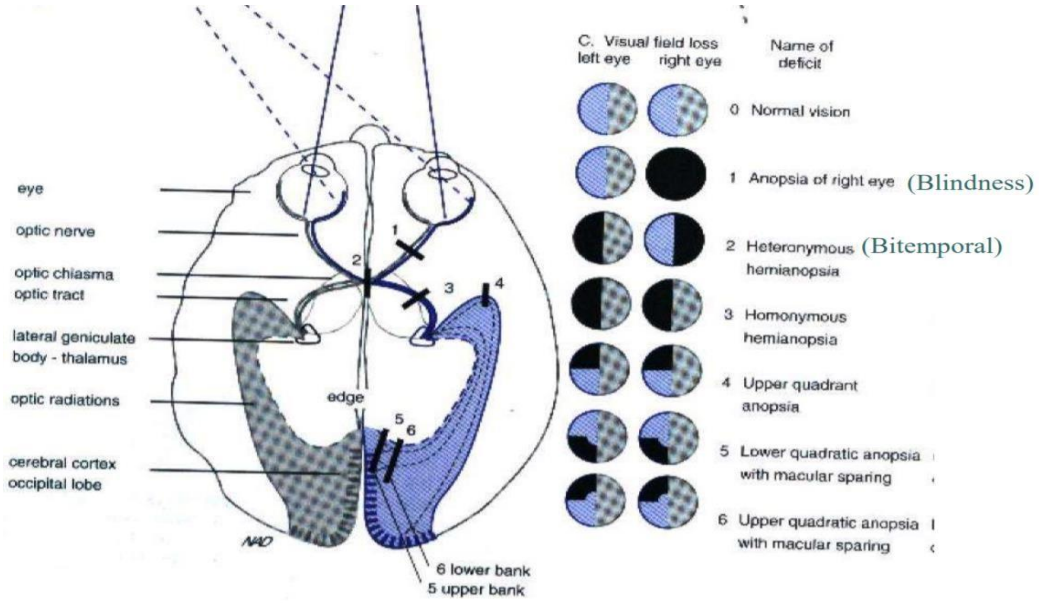
- The right and left half of each visual field are reversed in their projection onto the retina, meaning that the temporal (lateral) part of the retina looks at the medial halfof the visual field, and the nasal (medial) part of the retina looks at the lateral half ofthe visual field.
- Axons from ganglion cells in the nasal half of the retina cross the midline in the optic chiasm so that their information goes to the visual centers of the opposite side of the brain.
- Axons from the temporal half of the retina do not cross the midline and their information goes to the cerebral cortex of the same side.
- So eventually, the light from right side of visual field goes to the left visual cortex, and the light from left side of visual field goes to the right visual cortex.
- Optic chiasm is located above the pituitary gland. If there is a pituitary tumor, it may obstruct the optic chiasm, leading to loss of vision on the lateral aspects (fibers coming from nasal retina are affected), so this person can see just in the middle, which is called "**tunnel vision**", and this condition is called **bitemporal hemianopsia**.

◆ *Visual pathways to the brain*

- The visual nerve signals leave the retinas through the **optic nerves**.

- Optic nerve: axons of ganglion cells of the retina
- The fibers from the nasal halves of the retina cross to the opposite side in the **optic chiasm.**
- These fibers then join fibers from the opposite temporal retina to form the **optic tracts**. **(Right Optic Tract: fibers come from right and left eye, nasal from left and temporal from the right eye)** and vice versa.
- The fibers of each optic tract then synapse in the **dorsal lateral geniculate nucleus** of the thalamus.
- From there, they pass as **optic radiation** to the **primary visual cortex** in the occipital lobe.
- Visual fibers also pass to several areas of the brain:
  1. From the optic tracts to the **suprachiasmatic nucleus of the hypothalamus**. This pathway is important to control circadian rhythms that synchronize various physiological changes of the body with night and day. For example, thesecretion of hormones is not fixed throughout the day, it changes between dayand night "biological clock". For example: cortisol is high in the early morning
  2. **Pretectal nuclei** in the midbrain (Edinger-Westphal nucleus), which is important to activate pupillary light reflex and accommodation of the lens (willbe explained later in this sheet).
  3. **Superior colliculus** in the midbrain, to control rapid directional movements of both eyes
  4. **Ventral lateral geniculate**, to help control some of the body's behavioral functions.

◆ *Visual field defects*



Interruption of the visual pathway at different points leads to very specific visual field defects:

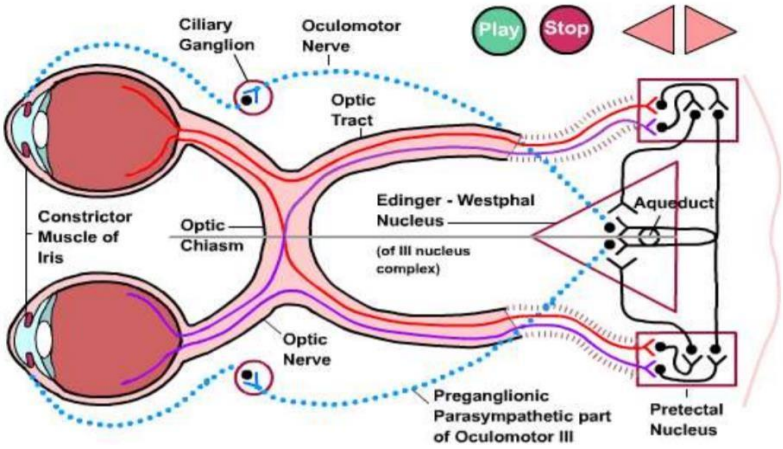
1. Damage to **one** of the optic nerves would lead to blindness of **one** eye **"anopsia".**
2. Damage to optic chiasm would lead to **bitemporal hemianopsia (heteronymous hemianopsia)** as explained before.
3. Damage to optic tract would lead to loss of vision from the opposite side of visual field. For example, damage to right optic tract would lead to loss of vision on the left side of both visual fields*. This is called **homonymous hemianopsia.**

   *remember that right optic tract receives fibers from temporal retina of the righteye, and nasal retina of the left eye, both of which receive light from left sides of visual fields (look at the figure).

4. damage of lower part of optic radiation would lead to **upper quadrant anopsia**.

5. damage to upper part of optic radiation would lead to **lower quadrant anopsia**, with macula lutea sparing. Macula lutea contribute to very large area of optic radiation, thus it's very hard for it to be affected.

◆ *Pupillary reflex pathway*



Pretectal nucleus (Edinger-Westphal nucleus) in the midbrain gives rise to parasympathetic fibers which exit with 3rd cranial nerve i.e. oculomotor nerve. These fibers have two effects: contraction of iris sphincter leading to constriction of pupils (meiosis), and contraction of ciliary muscles making the lens more spherical and leading to accommodation.

There are two pupillary reflex pathways:

1. **Direct pupillary reflex**
   When you flash a light on the right eye, visual signals proceed through the optic nerve and reach the pretectal nucleus. This causes stimulation to the right 3rd cranial nerve (ipsilateral), leading to the constriction of the right eye's pupil.
2. **Consensual (indirect) pupillary reflex**
   When you flash a light on the right eye, Visual signals from the right eye reach the pretectal nucleus and the fibers cross to the opposite side and activate the left 3rd cranial nerve leading to constriction of pupils of the left eye.

In conclusion, each afferent limb has two efferent limbs, one ipsilateral and one contralateral. So normally when you flash a light on one eye, the pupils of both eyes should constrict.

Clinical significance

Pupillary light reflex provides a useful diagnostic tool for testing the integrity of the sensory and motor functions of the eye. For instance, it's used to test for theoptic nerve injury, and oculomotor nerve damage.

Examples:

- ✓ If you flash a light on the right eye and you didn't see any response in the right eye pupil, but you saw a constriction of the left eye's pupil → this means that the right 3rd cranial nerve is damaged, so when the signal reaches the pretectal nucleus, it can't come back to the right eye.
- ✓ If you flash a light on the right eye and you didn't see a response in both eyes → this means that the right optic nerve is damaged, so the signal didn't reach the pretectal nucleus at all.

❖ Function of the dorsal lateral geniculate

It has two principal functions:

1. Relay of information to primary visual cortex (as we discussed earlier).

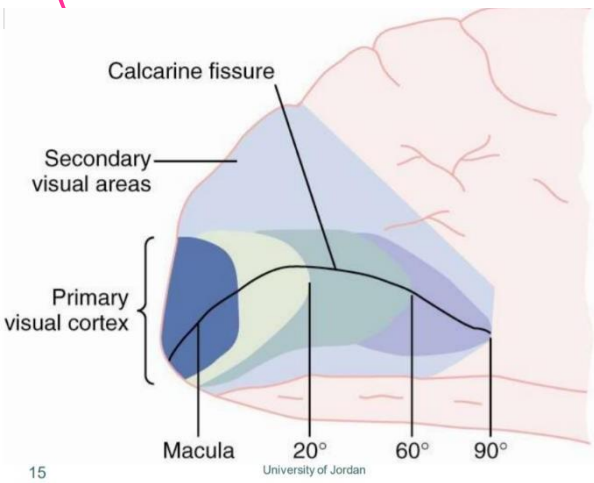
2. "Gate control" of information to primary visual cortex. That is, how much of the signal is allowed to pass to the cortex.
   LGN receives input from corticofugal fibers originating in the primary visual cortex and input from reticular areas of the midbrain. Both inputs are inhibitory and can turn off transmission of the signal in selected areas of the LGN. Both inhibitory inputs presumably control the visual input that can pass to the cortex.

   Note: corticofugal fibers are important to increase the sensitivity of somatic system as well as visual system.

❖ Primary visual cortex

- ✓ It is located in the occipital lobe in the calcarine fissure region.
- ✓ It has large representation for the macula lutea (region of highest visual acuity) because it is proportional to the number of photoreceptors in this area (remember that macula lutea has very large number of cones) - The same principle that applies to all sensory systems.
- ✓ It is a layered structure just like any cortical area (composed of six layers).
- ✓ It has columnar organization as well, and each column is responsible for one kind of visual sensations.
- ✓ It receives the primary visual input.

❖ Secondary visual cortex (visual association cortex)



- ✓ It's responsible for analyzing and interpreting the visual information.
- ✓ It's the area for 3-dimensional position, gross form, and motion.
- ✓ It's the area for color analysis.
- ✓ Having it damaged result in "**word blindness**". In this condition, the person can see normally but he cannot explain or give a meaning to what he sees. **Macula here has a big area of the cortex due to the great number of receptors (cones receptors)**.

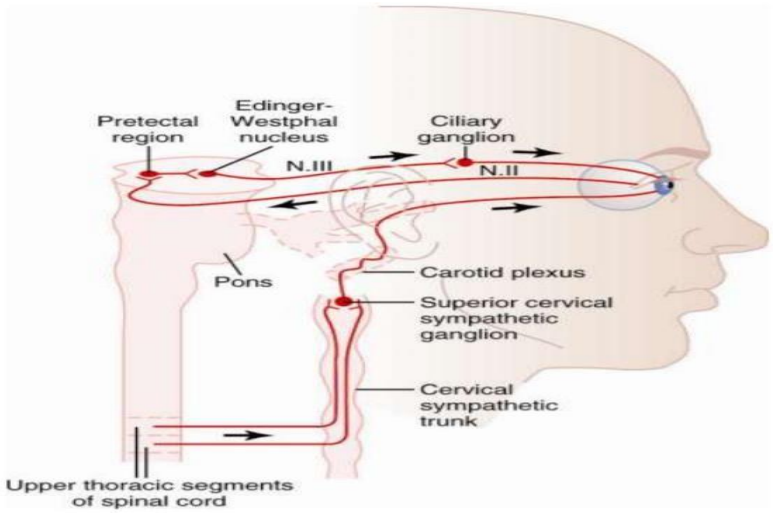
❖ Processing in the visual cortex

Separation of the signals from the two eyes is lost in the primary visual cortex. Signals from one eye enter the columns, alternating with signals from the other eye. This allows the cortex to decipher whether the two signals match, allowing you to see just one picture.

❖ The Autonomic Nerves to the Eyes

- the eye is innervated by both parasympathetic and sympathetic neurons.
- Parasympathetic fibers arise in the Edinger-Westphal nucleus, pass in the 3rd cranial nerve to the ciliary ganglion. Postganglionic fibers excite the ciliary muscle and sphincter of the iris.

- Sympathetic fibers originate in the intermediolateral horn cells of the superior cervical ganglion. Postganglionic fibers spread along the carotid artery and eventually innervate the radial fibers of the iris.



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❖ Control of Accommodation (Focusing the Eyes)

- It results from contraction or relaxation of the ciliary muscle.
- It's regulated by negative feedback mechanism that automatically adjusts the focal power of the lens for highest degree of visual acuity within about 1 sec. the exact mechanism is not known.

❖ Control of Pupillary Diameter

- miosis: decreasing of pupillary aperture due to stimulation of parasympathetic nerves that excite the pupillary sphincter muscle.
- mydriasis: dilation of pupillary aperture due to stimulation of sympathetic nerves that excite the radial fibers of the iris.

Good luck