Continuation to Lesions of the Spinal Cord

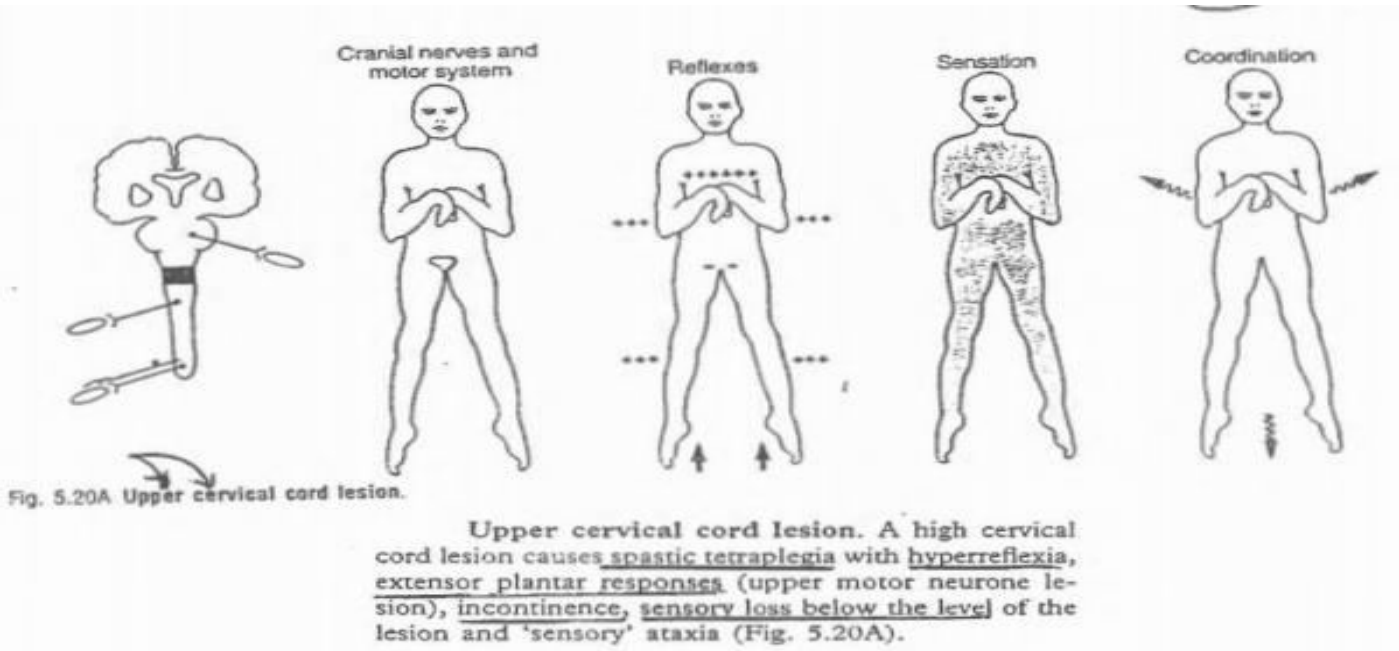


Fig. 5.20A Upper cervical cord lesion.

Upper cervical cord lesion. A high cervical cord lesion causes spastic tetraplegia with hyperreflexia, extensor plantar responses (upper motor neurone lesion), incontinence, sensory loss below the level of the lesion and 'sensory' ataxia (Fig. 5.20A).

If we were looking at a lesion at the upper cervical region, a complete section of the spinal cord, what are the changes that can be seen? (motor and sensory)

1) Motor: Paralysis of the muscles of upper and lower limb (quadriplegia) below level of the lesion, since the pyramidal and extrapyramidal tracts reaching alpha and gamma motor neurons will be disrupted.

Is it an upper or lower motor neuron lesion?

Since we have not reached the alpha and gamma neurons, it is a upper motor neuron lesion, and all signs of UMN injury could be seen; such as hyperreflexia (exaggerated deep tendon reflex), positive babinski sign, spasticity of upper limb flexors and lower limb extensors (anti-gravity muscles)

2) Sensation: complete sensory loss below the level of the lesion, that includes pain, touch, temperature, proprioception, vibration etc...

Will the patient exhibit ataxia? And if so what type is it?

Yes, but we cannot examine if it is cerebellar ataxia since obviously his muscles are paralyzed, we can however touch his arm or extend his elbow and check if he has conscious awareness of whether his elbow is flexed or extended, the patient cannot tell and so we conclude he has sensory ataxia due to loss of sensation from joints or muscles.

By observing all those symptoms we can decide it is an upper cervical lesion.

Lower cervical lesion

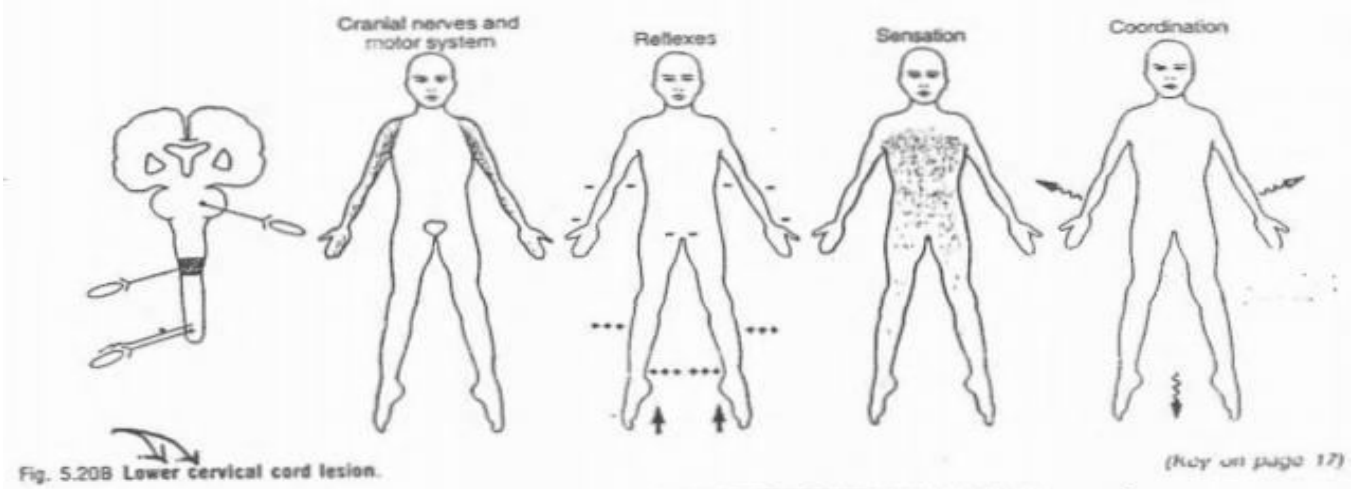


Fig. 5.20B **Lower cervical cord lesion.**

(Key on page 17)

Lower cervical cord lesion. A lower cervical cord lesion causes weakness, wasting and fasciculation of muscles, and areflexia of the upper limbs (lower motor neurone lesion). In addition, there is spastic paraparesis, hyperreflexia and extensor plantar responses (upper motor neurone lesion) in the lower limbs, incontinence, sensory loss below the level of the lesion and 'sensory' ataxia (Fig. 5.20B).

The lesion could be in C6,7,8 and T1, what do those segments remind you of?

Brachial plexus (c5 also)

So what are the expected outcomes?

Since brachial plexus is injured then the muscles of the upper limb are paralyzed, spastic or flaccid paralysis?

Flaccid ,since there would be a LOWER motor neuron lesion at the level of the injured segment (atrophy of upper limbs with time)

We would also see hypo-reflexia which is another sign of LMN lesion, the alpha and gamma would be destroyed in those segments, clinically speaking more than one segment should be effected, since for example if a bullet caused this injury, it rarely just hits one segment only

The pyramidal and extrapyramidal tracts cannot reach the lumbo-sacral region and are disrupted, thus we will have UPPER motor neuron lesion BELOW the level of the injury... such signs would be spastic paralysis, hyperreflexia, positive babinski, extensor plantar response etc... and of course sensory ataxia since we lost all sensation below the lesion.

Pay attention to the two types of paralysis and their causes (spastic and flaccid)

What about a lesion at the level of the thoracic spinal cord?

Lesions of spinal cord (*continued*)

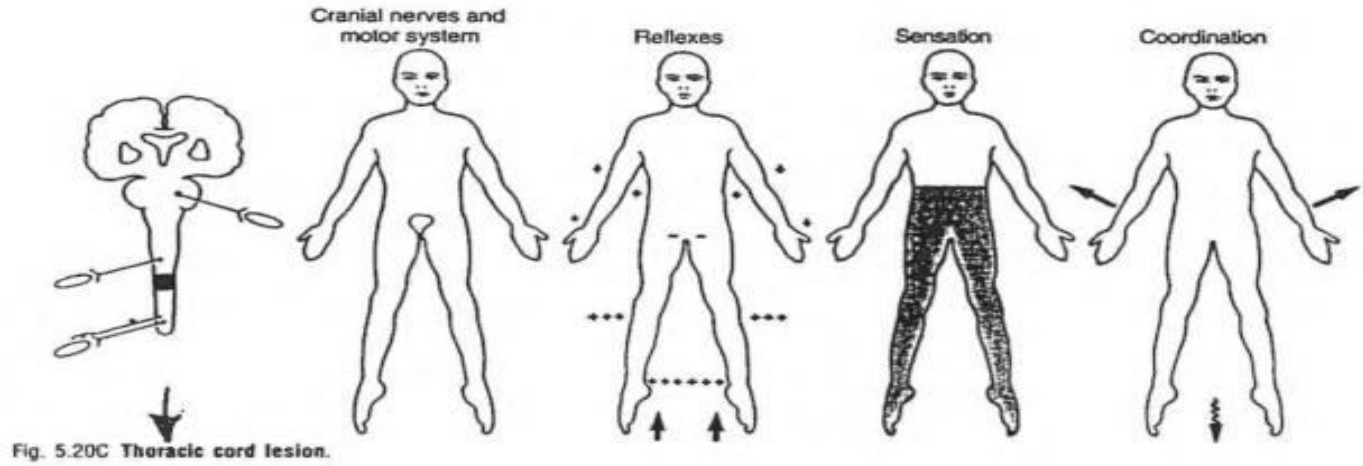


Fig. 5.20C **Thoracic cord lesion.**

Thoracic cord lesion. A thoracic cord lesion causes a spastic paraparesis, hyperreflexia and extensor plantar responses (upper motor neurone lesion), incontinence, sensory loss below the level of the lesion and 'sensory' ataxia (Fig. 5.20C).

We will only see symptoms below the level of the lesion (upper limbs not affected)

Motor and sensory loss in the lower limbs

We will have normal reflexes in the upper limb

This lesion is an upper motor neuron lesion, the alpha and gamma of the lower limb are deprived from the pyramidal and extrapyramidal tracts.

A reminder: we would have spastic paralysis but why?

The **pontine-reticulospinal tract** increases the muscle tone while the **medullary reticulospinal tract** inhibits it, since those tracts are disrupted and there was an increase in tone, we can conclude that the inhibitory effect of the medullary tract was greater than the excitatory effect of the pontine tract (on the extensors MAINLY and flexors of lower limb).

So again with the absence of the medullary tract there is an INCREASE in tone.

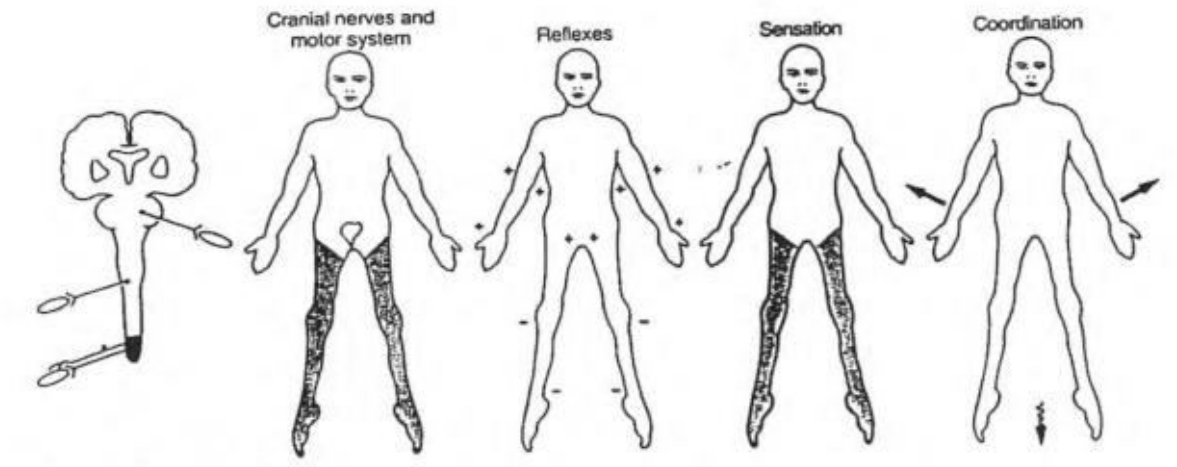


Fig. 5.20D **Lumbar cord lesion.**

↳ destroying lumbar and Sacral segments of sp cord

Lumbar cord lesion. A lumbar cord lesion causes weakness, wasting and fasciculation of muscles, and areflexia of the lower limbs (lower motor neurone lesion), incontinence, sensory loss below the level of the lesion and 'sensory' ataxia (Fig. 5.20D).

Lastly, **a lumbosacral lesion**

The alpha and gamma neurons of the lower limb are destroyed, so we would have the typical signs of the lower motor neuron lesion; flaccid paralysis, atrophy with time, **a**reflexia/hyporeflexia. And the upper limb would be normal. Also there is a loss of sensation of the lower limbs with sensory ataxia.

Accuracy of sensation

The accuracy of sensation depends on two factors:

1- Density of receptors.

2- The concept of lateral inhibition.

If you pointed a pencil on your skin, the area right beneath the tip of the pencil will receive maximum stimulation, and the surrounding area will be stimulated less, we need to inhibit the transmission of surrounding impulses ,so that we can tell exactly from where the sensation is coming (localization of stimulus).

The sensory pathway starts at the tip of pencil, if impulses are sent from more than one place the brain won't be able to determine the exact place of the stimulus.

How can we stimulate only the central part?

Through the sensory pathway leaving the cerebral cortex, it sends collaterals through interneurons that inhibit other pathways leaving the surrounding areas, and this lateral inhibition occurs in the CENTRAL nervous system (not peripheral). This phenomenon can mostly be seen with touch sensation and in the retina.

The retina can be stimulated by a tiny spot of light better than by a large spotlight, i.e the tiny spot of light is detected better and responds with an action potential more than when a large amount of light is displayed in front of the retina, but why is that?

The tiny spot of light can cause lateral inhibition as it more effectively alerts a small area in the retina, however a large light alerts a huge area and there won't be any lateral inhibition

End of sensory pathways 3:)

Anatomy of the Eye

As we took in MSS, the eyeball is made up of 3 layers:

1) Outer fibrous layer: in front 1/6 **cornea** transparent normally, 5/6 **sclera** dense fibrous tissue.

The cornea forms 1/6th of this layer, it is transparent and lies in front, behind it and forming the remaining 5/6th is dense fibrous tissue called the **sclera**.

2) The second layer is **muscular**.

3) Third layer and most inner: nervous tissue (**Retina**).

If we were looking at a patient's eye, what can we see?

The cornea and the **CONJUNCTIVA**

And what the heck is that?

It is a mucous membrane that lines the sclera and the cornea, it connects the eyeball with the eye lid and it has two parts; bulbar and palpebral. Bulbar that covers the sclera and palpebral that covers the eyelid.

At the junction of the cornea and sclera there is a **canal of schlemm** (sinus venosus sclerae)

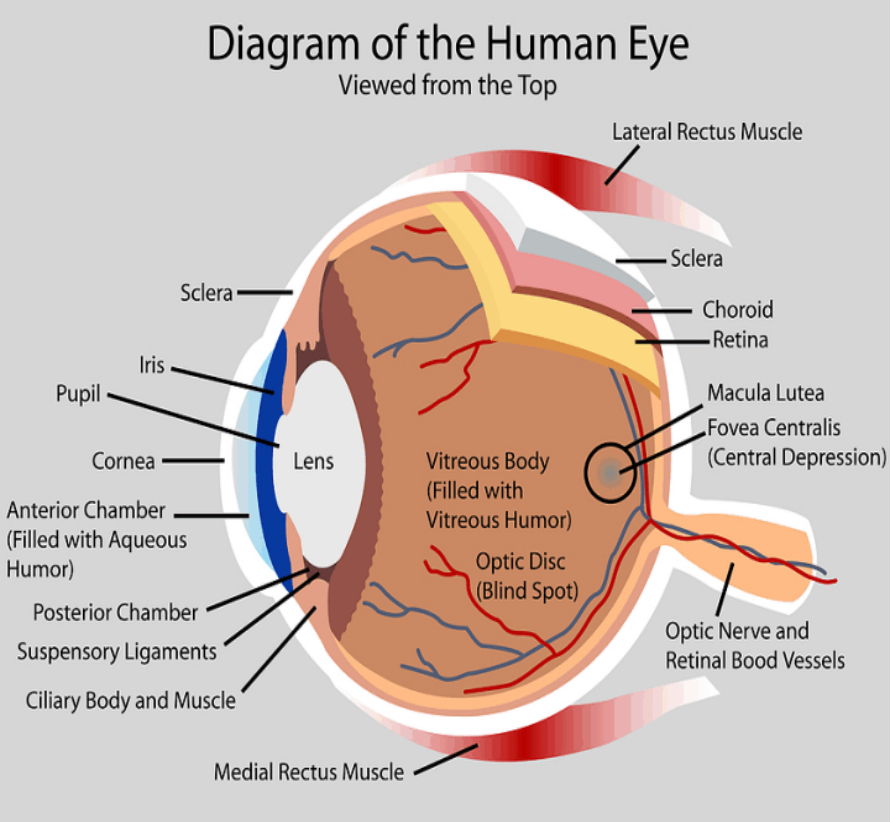
It receives and drains aqueous humor, a fluid in the eye found in the anterior and posterior compartment of the eye.

Moving on to the middle layer, it is muscular and filled with blood vessels, the iris lies in front and consists of two parts, an upper and lower part, between them lies the pupil.

The iris differs from a person to another in colour. *It is the colored part that we see around the pupil when we gaze into someone's eyes*

Shout-out to the green-eyed people in our batch, you may constitute only 2% of the world, but you sure as heck constitute 100% of my world :3

The pupil can be dilated or constricted by the smooth muscles of the iris under the effect of sympathetic or parasympathetic stimulation, the muscles are called **constrictor pupillae** (under the effect of parasympathetic) and **dilator pupillae** (under the effect of sympathetic)**.**



Behind the iris we can find a **ciliary body** which is a circular ring of smooth muscles surrounding the iris, they control the thickness of the lens which will be explained later on. Projecting from this ciliary body are **ciliary processes** which secret aqueous humor, this fluid moves from the posterior chamber into the anterior chamber, how?

Well firstly, the space between the cornea and iris is the anterior chamber, and the space behind the iris, extending till the lens is the posterior chamber, the aqueous humor is released by the ciliary process into the posterior chamber, connecting the two chambers is the pupil's opening which allows the fluid to move into the anterior chamber.

If for some reason there was pressure on the Cannal of Schlemm, there wouldn't be anymore drainage, this will result in an increase in intraocular pressure, scientifically termed; **Glaucoma** "المياه الزرقاء".

Glaucoma could hurt the retina of the eye if left untreated. When we look at the iris of a patient, we give him an eye-drop that dilates the pupil, when this happens pressure is exerted on the iris, which is close to the canal of schlemm and hence would exert pressure on this canal, disturbing the drainage. What's the clinical significance of this? Never give a patient suspected with glaucoma an eye-drop that dilates the pupil, worsening of glaucoma could hurt the retina and blind the patient.

The last part of the middle layer is the **choroid**, it is loose areolar tissue filled with blood vessels.

Moving on to the inner layer; the **Retina** is made up of two parts, an outer pigmented layer and an inner nervous.

Looking at the posterior part of the retina, there is a site where the optic nerve leaves the retina, a major neuron in the retina is the ganlion cell, its axon demerges forming the **Optic nerve**, and its cell body lies in the ganglion cell layer.

At the site where the nerve leaves, lies the **Optic disc**, also known as the blind spot since there are no receptors (no 'rods or cones'). Lateral to this we can find the **Macula lutea**, and in its center: **Fovea centralis**, this is the area of most accurate vision, it only contains cones which are responsible for colour vision as well as visual details.

Normally the eye-lens is biconvex, thick and elastic, with old age, the lens starts losing its elasticity, i.e the ability to accommodate to near objects, this condition is called **presbyopia**, the lens would be thin instead of thick and has lost its elasticity.

The lens is also normally transparent; however in a condition called **cataracts**" المياه البيضاء", vision is affected as glucose precipitates in the lens, making it no longer transparent. This condition is benign and we can simply remove the lens and replace it with an artificial one.

So **accommodation** is the ability to look at and recognize near objects, but how exactly does it occur?

In stages, and the most important one is that the thickness of the lens needs to increase, but how?

Well firstly we should know that the lens is attached to ciliary muscles (in the ciliary body) through suspensory ligaments that hold the lens with the ciliary body.

The ciliary muscle is a ring, it is normally relaxed (like when you are looking at the board), when the muscle is relaxed, the suspensory ligaments are tensed, the lens is flat (thin) and weak, there isn't any parasympathatic stimulation to it at that moment.

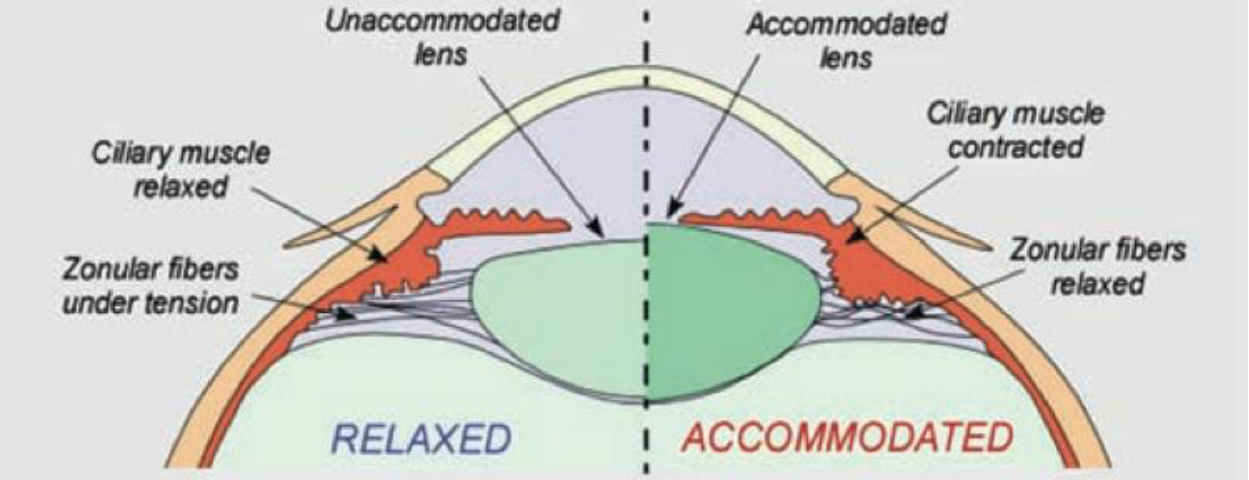
When you are reading this sheet, accommodation is taking place:

1) Parasympathatic innervation stimualtes two muscles i) a muscle in the iris, constrictor pupillae (its function discussed later on) and ii) ciliary muscle

2) As ciliary muscle contracts, its diameter decreases.

3) Less tension on ligament.

4) Lens becomes thicker and stronger >> better vision.



Moving on to the retina:

As we said earlier we have two layers, the first is the outer pigmented layer which is attached to the choroid. We may hear the term "**retinal detachment**" انفصال الشبكية , which is when the retina detaches from the choroid, and since the choroid is the blood supply to the retina, ischemia could take place and we may end up with blindness.

Let's have a look at the layers and receptors of the retina:

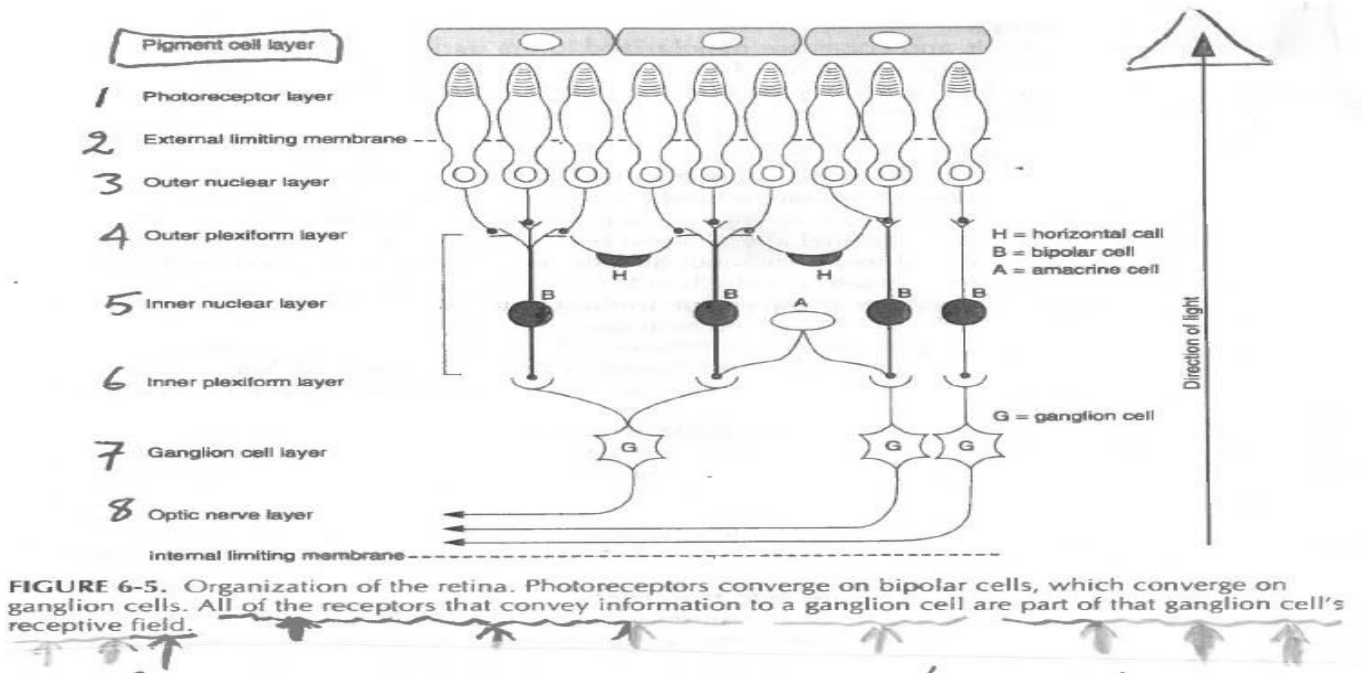


FIGURE 6-5. Organization of the retina. Photoreceptors converge on bipolar cells, which converge on ganglion cells. All of the receptors that convey information to a ganglion cell are part of that ganglion cell's receptive field.

Notice that the receptors lie in the outermost layer not in the innermost, what does that mean?

In order for light to reach the rods it has to pass all layers of retina. (except in the fovea, where the receptors are exposed and light reaches the receptors directly)

1-First layer and outermost: photoreceptors (where light reaches) cones + rods.

2- External limiting membrane which is not important.

3- Outer nuclear membrane, here the nuclei of the rods and cones receptors lie.

4- Outer plexiform, this is the outer synaptic layer where synapses between photoreceptors with bipolar cells and horizontal cells take place, the most important synapse is the synapse between photoreceptors and bipolar cells.

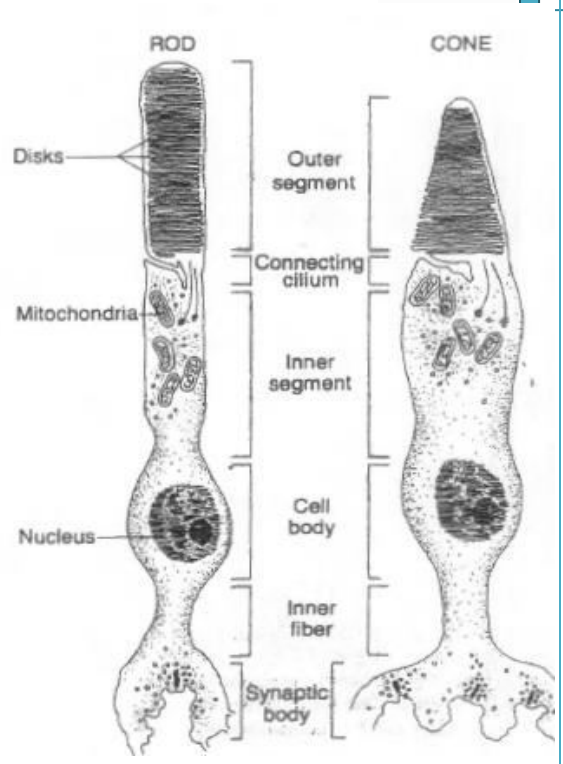
5- Inner nuclear layer, here the nuclei of bipolar cells and horizontal cells lie , there are also the nuclei of amacrine cells whose functions aren't clear

6- Inner plexiform; Inner synaptic layer between ganglion cell layer (which is the most important) and bipolar cells

7- Ganglion cell layer: Major neurons lie here: photoreceptors > bipolar cells > ganglion cells

8- Optic nerve layer: the axons of ganglion cells leave this layer and form the optic nerve.

And finally the internal limiting membrane.



Notice the photoreceptors (Rod and Cone) in the picture, both have an outer segment formed of disks.

These disks contain photo-pigments which react to light (Rhodopsin).

Rhodopsin is composed of:

1) A protein called Opsin (more important), which determines the color of pigment.

- The amino acid composition of Opsin determines the wavelength of light absorbed by the photopigment .

-Rods contain a single type of Opsin, Cones contain 3 types of Opsins (absorb blue, green, or red depending on the portion of the visual spectrum they absorb best).

2) A light absorbing analogue of vitamin A (also called 11-cis retinal).

On the other hand, the inner segment contains mitochondria, nucleus, and synaptic vessels.

Once the photoreceptors are activated, they will release transmitters affecting Bipolar Cells, the Bipolar Cells then affect the Ganglion Cells.

(Pathway: photoreceptors→bipolar cells→ ganglion cells)

A Comparison Between Rods and Cones:

Rods	Cones
-More sensitive to light -Responsible for night vision -Less visual detail -More pigment in outer segment	-Less sensitive to light -Responsible for day vision -More visual detail -Less pigment in outer segment -3 types of photopigments (absorb red, green, or blue) - Fovea Centralis (central pit of compacted cones in the eye)

Response of photoreceptors:

Rods and Cones are depolarized (activated) in the dark, and hyperpolarized (inhibited) in the light. These are the only receptors in our body that respond to an adequate stimulus by hyperpolarization.
Dark "on pathway": The resting membrane potential is low ~ -40 mv. It is a result of the high Na^+ conductance of the outer segment, allowing a continuous release of a synaptic transmitter.
-Depolarization always needs an inflow of sodium via Na^+ channels located in the outer segment. Na^+ is transported out of the outer segment via Na-K Pumps.
-Opening of these Na^+ channels requires the presence of cGMP, which activate the channels directly.
-ATP is provided by the mitochondria located in the inner segment to maintain the activity of the Na-K pump.
-The released transmitters act on the Bipolar cells.

Bipolar cells have 2 types of receptors reacting to transmitters. One causes depolarization upon reaction (depolarizing), the other causes hyperpolarization upon reaction (hyperpolarizing).And that depends on its receptor and neurotransmitter released from it .The depolarizing →causes activation of the ganglion cell . The hyperpolarizing →causes NO activation of the ganglion cell.
activation of the ganglion cell→Action potential.

Depolarizing receptors activate the Ganglion cells (the only cells that can produce an AP, the rest can only generate a partial depolarization or graded potential). There is a basic level of AP in the Ganglion cell.

Light "off pathway": photopigments react upon exposure to light, causing the hydrolysis of cGMP, closing the Na channels, leading to hyperpolarization.
Some Ganglion cells are activated and others are inhibited.
The membrane potential is decreased.

This prevents the release of transmitters (absence of transmitters).

So, Bipolar cells that were depolarized in the presence of transmitters (dark) become relatively hyperpolarized when light excites the receptors.

already mentioned above The Fovea Centralis: the visual center of the eye & the area of the highest resolution, here the receptors are exposed and light reaches them directly.

It's a yellowish depressed region located in the center of the retina (macula lutea).

#In the dark the photoreceptors will be depolarized, and will release neurotransmitters to bipolar cells and these bipolar cell are either depolarizing or hyperpolarizing. In the light the photoreceptors will be hyperpolarized and no neurotransmitters will be released to bipolar cells and the depolarizing bipolar cells in the dark will be hyperpolarized in the light and the hyperpolarizing bipolar cells in the dark will be depolarized in light.

The Visual Pathway

The Retina has 2 halves: Temporal (outer) and Nasal (inner)

The field of vision (what you see when you look straight forward),The field of vision has a right and left half.

The temporal half receives input from the left half (opposite to each other), and the nasal half receives input from the right half (opposite to each other)

So: Light falls on the Rods & Cones of the retina (1st order neurons) triggering a photochemical reaction, initiating nerve impulses > Bipolar cells (2nd order neurons) > Ganglion cells (3rd order neurons) > axons converge forming the Optic Nerve which pierces the sclera > Optic Chiasm (only the Nasal halves of retina decussate) > Optic Tract > Thalamic Center of Vision (lateral geniculate body-containing the lateral geniculate nucleus).

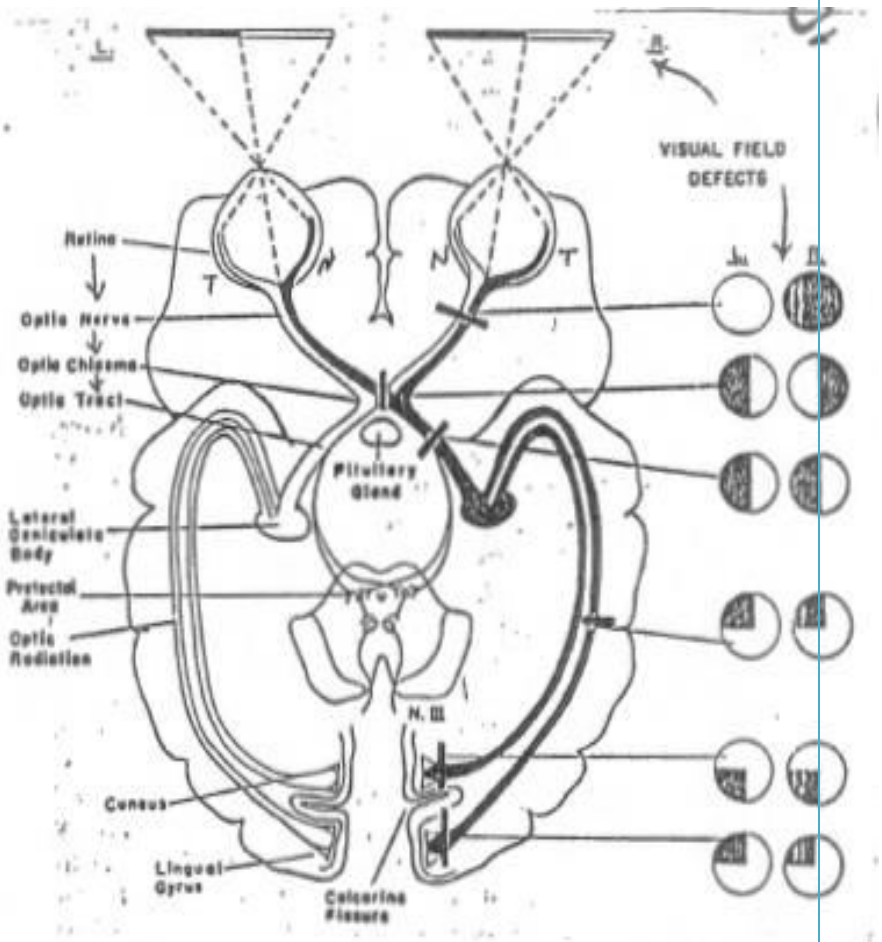


FIGURE 38. The visual pathway. On the right are maps of the visual fields with areas of blindness darkened to show the effects of injuries in various locations.

*Every sensation has a nucleus
*Center for hearing: Thalamic Center of Hearing (medial geniculate body)
In Optic Chiasm ,only the Nasal halves (nerve fibers of the nasal half) of retina decussate, the temporal halves do not decussate.

-When going back from the thalamus to the cortex, everything should pass through the internal capsule. Cells of the Geniculate Bodies give rise to fibers which form the **Geniculo-Calcarine Tract** a.k.a The **Optic Radiation** which ends on the visual cortex (area 17) on either side of the calcarine fissure within the occipital cortex.
The **Optic Radiation** pass through the retrolentiform part of internal capsule.

This sheet is dedicated to Bana Mikhi, Yara Bahar, Dina Murad, Saba Barham, Shatha Khader, Asma Jisrawi, Leen Younis, Dania 7abebti Tobasy, Marah Abu Rumman and basically everyone in lab 8 <3
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#لتسهيل_حياة_طالب_الطب