Lesions of Visual pathway

Salam everyone ☺ this sheet was written according to section 2 record, some parts are rearranged …

In this lecture we will talk about the lesions affecting different parts of the visual pathway.

We've talked about the lesion of optic nerve and that it causes blindness (loss of vision) in the corresponding eye, also we said that the commonest pathology affecting the optic nerve by the process of demyelination is multiple **sclerosis (MS).** Patients of MS have several signs and symptoms including problems (weakness) in vision.

Optic tract:

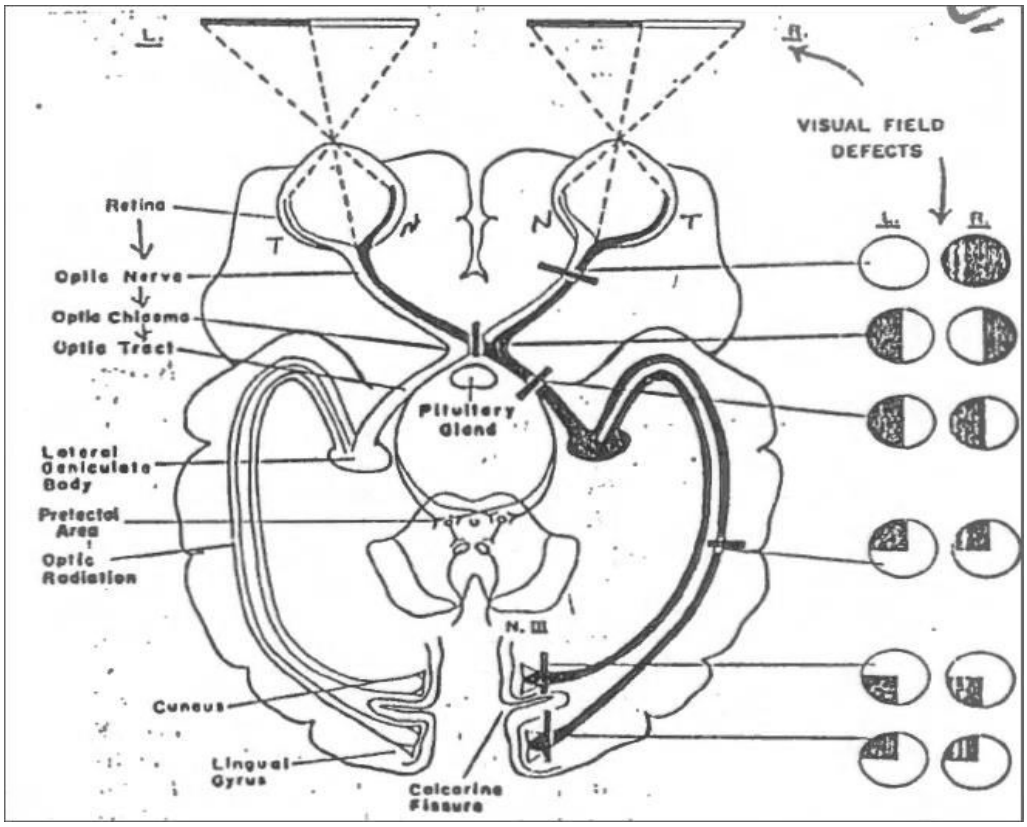
The Optic tract contains two types of nerve fibers :

1) fibers coming from the **temporal** half of the same side (since these fibers don't cross or decussate). 2) fibers coming from the **nasal** half of the opposite side. **So optic tract receives fibers from both eyes** .

Fibers of the Optic tract synapse in the thalamus (in the **lateral geniculate body or nucleus (LGB)**) and from **LGB** the **optic radiation** leaves and passes through **retrolentiform** part of internal capsule and ends at the **visual cortex** .

Optic radiation can be so called **geniculo-calcarine tract** since it extends from geniculate body to calcarine cortex (visual cortex around calcarine fissure).

{Remember that **Retina** has two halves : temporal(outer half) and nasal (inner half)} ; the right half of the right eye is the temporal and the left half of the right eye is the nasal while the left eye is the opposite ,so If we interrupt the optic radiation each eye will be affected (temporal of one eye and nasal of the other one).



We describe the effect of interruption on the **visual field** حقل الرؤية (the **visual field** is what you see when you look straight forward on the external world).

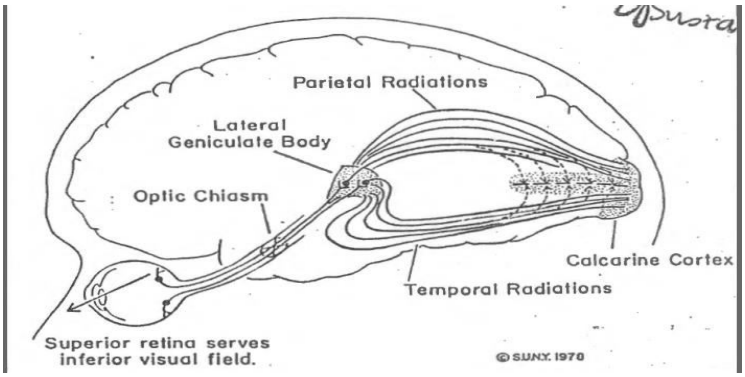
The right side of the retina ------> the left visual field

The left side of the retina -----> the right visual field

So if the lesion is in the optic tract at right i.e. fibers coming from **temporal** half at **right** eye and **nasal** half at **left** eye the effect will be on **left** half of each field (the patient can't see the left half of each field).

Again, the lesion is at the fibers coming from right side of each eye(right half of right eye is temporal while right half of left eye is nasal) the result will be loss of vision in left half of each field . this case is called (**contralateral homonymous <u>bitemporal</u> hemianopia**) . { contralateral :because the effect is on the opposite side , homonymous متجانس , **hemi**anopia : lost vision in **half** of the field }.

Same symptoms (contralateral homonymous hemianopia) will take place if the lesion was at **lateral geniculate body**(LGB) or at the **whole optic radiation** or at the whole **visual cortex** .



It is difficult to have a lesion affecting the whole optic radiation because optic radiation is formed of extensive fibers and not of a small bundle . the optic radiation runs partially in the **parietal lobe** and partially in **temporal lobe** (so it runs in a wide area) but both(parietal and temporal radiations) will pass through the **retrolentiform** part of internal capsule . (look at the figure) and see the geniculate body of thalamus , cortex, upper part of optic radiation(parietal radiation) ,and temporal radiation . Notice that it is rare to have a lesion that can interrupt all these parts, it can affect either the lower part or the upper part.

Upper fibers of optic radiation that run in the parietal lobe will end in the **cuneus**(part of visual cortex **above** the calcarine fissure), lower part (the temporal) will end in the **lingual gyrus** (part of visual cortex **below** the calcarine fissure) .

The **upper** fibers of optic radiation receive impulse (visual impulse) from the **upper** part of retina but from **lower** part of the field. *Again, the upper part of optic radiation and upper part of visual cortex receive visual impulses from upper part of retina and lower part of the field.* ☺

If we interrupt the whole optic radiation there will be **homonymous hemianopia** , vision of half of the field will be lost, but if we interrupt the upper or lower fibers (i.e. half of the fibers of the optic radiation) we will lose **half of the half** = one quadrant . so for example if the lower part(**temporal**) was interrupted ,what will happen?

Temporal fibers receive from **lower** part of retina and **upper** part of the field ,so there will be **contralateral superior homonymous quadrantinopia.** The same will happen if we interrupt the lower part of visual cortex (lingual gyrus).

Now , If the **cuneus**(upper part of visual cortex) or the upper part of optic radiation was interrupted the result is **contralateral inferior homonymous quadrantinopia** .

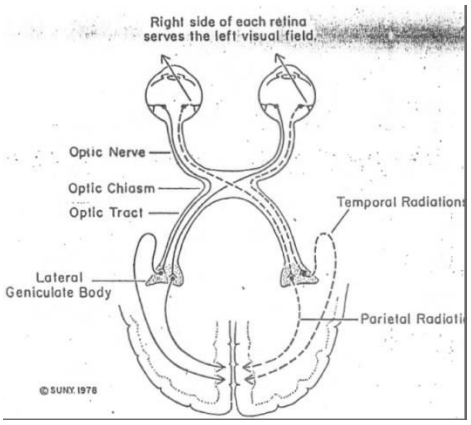
Soooo if we interrupt the optic tract or the whole optic radiation or the whole visual cortex we will lose half of the field (hemianopia) but if we interrupt half of the radiation (upper or lower) or half of the cortex we will lose **half of the half** of the field (quadrantanopia) .

✍. is the patient of hemianopia or quadrantanopia aware of his/her condition??? Well, sometimes a patient of hemianopia might live for 20 years and won't be aware of the condition and then he/she discover the condition by accurate examination and testing.

Optic chiasma:

optic chiasm is the X-shaped structure formed at the point below the brain where the two optic nerves cross over each other. it is superior to the pituitary gland , so if there is a tumor (a big one) in the pituitary gland it will interrupt the middle of the chiasma (or at the periphery but we will take it at the middle) , so if we interrupt the optic chiasm in the middle the decussating fibers will be interrupted , which fibers? The **nasal** fibers ☺.

Nasal fibers in the right eye are at the left and receive from right field , while nasal fibers of the left eye are at the right side and receive from the left field. 😅



So when the nasal fibers are Interrupted , the **right** eye will not be able to receive from the **right** field and the **left** eye will not receive from the **left** field , soooo we call it here

(**contralateral heteronymous hemianopia**), heteronomous because we lost vision at right side in one eye and left side in the other eye because the lesion is in nasal fibers (the crossed fibers) .

✎. Not all fibers of optic tract end in the thalamus(in **LGB** ; lateral geniculate body),some of the fibers leave the optic tract and go to (the **pretectum** in the midbrain) . these fibers of the optic tract which end in the midbrain and not in the thalamus serve the function of **Light Reflex** ; when you direct a light toward the eyes , both pupils will be constricted – it will be discussed soon☺.

If the optic tract was interrupted at its beginning , the fibers will not reach the midbrain , if the tract was interrupted at its termination (i.e. the optic radiation was interrupted) the light reflex will be intact.

*Homonymous hemianopia (loss of vision in half of the field) + loss of light reflex ------> a lesion in the **beginning** of the optic tract.*

*Homonymous hemianopia + intact light reflex ------ > a lesion in the **termination** of optic tract or at **optic radiation**.*

The visual cortex :

*****area 17 (primary visual area) at the **right** side will receive visual impulses from **right** side of each retina that receive from **left** side of each field.

*Area 18 and 19 (the secondary or association visual cortex) their function is the recognition of what you see. These areas are called "**occipital eye field** " while **area 8** is called **frontal eye field**.

{Occipital and frontal eye fields are connected by superior longitudinal fasciculus and both of them play a role in eye movements}.

area 18 and 19 play a role in **conjugate movements of the eye** (if you adduct one eye by the 3rd nerve you should abduct the other by the 6th nerve) . if you stimulate area 18 and 19 the patient will feel hallucination of light and vision (وميض ضوء) , while the **destruction** of these areas will cause "**visual agnosia**" ; a condition in which you can't recognize and describe what you see ☹ .

*There is another area thought to be the highest visual association cortex which is the **infratemporal area** ; a part of inferior temporal gyrus , it receives impulses from area 18 and 19 through a bundle of fibers called **inferior longitudinal fasciculus** (a type of association fibers) , if this bundle was injured bilaterally that will cause **"visual agnosia"** too. the patient can't recognize or identify what he /she sees. Also this area receives from area 7, (**Area 5 and 7** are important to recognize what you feel in the **somatic-sensory cortex)** . so it receives from area 7 and most importantly from 18 and 19 ,the **infratemporal** area which is the **highest association area**.

Now, how is the field of vision represented in The visual cortex around the calcarine fissure in the occipital cortex ?

the Center of visual field (the point of fixation) is **macula** and the **fovea centralis** that is located in the center of macula is the most accurate site of vision.

macular vision (vision of center of the field) is represented in the most **posterior** part of visual **cortex** , the rest of the field is represented anteriorly. *so the peripheral retinal fibers occupy the anterior part of the cortex while macular fibers occupy the posterior part of the cortex.*

It is said that a patient with **homonymous hemianopia** can still see the center of the field , **macular vision** is intact ,why is that?

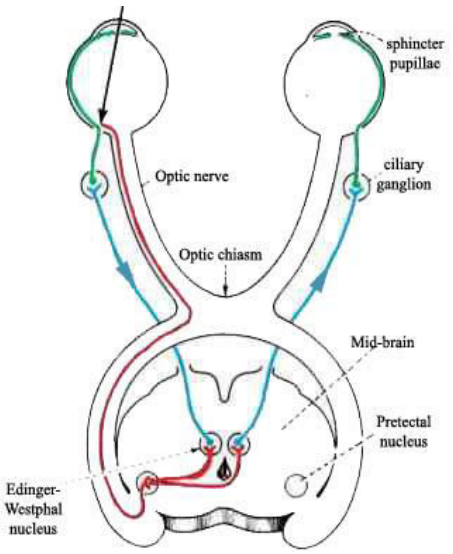
Because this part of **visual cortex** receives double blood supply so if there was a lesion in the "**posterior** cerebral artery" this area will

not die like the surrounding areas because the **middle** cerebral artery will compensate and supply it . So if you have a patient with homonymous hemianopia and has central vision (can see center of the field) but can't see the periphery ,this is called " **macular sparing** " . (**Macular sparing** is when the center of the visual field is unaffected in an otherwise hemianopic defect).

. Sooo { even though one optic tract has been completely interrupted ,vision is preserved in a small area at the fixation center ;the area of macula . } ☺☺

The light reflex:

any reflex as we know is composed of afferent limb ,center and efferent limb. the afferent of this reflex is the **optic** nerve (the sequence : retina --- > optic nerve ---- > optic chiasma ---- > optic tract ;some of its fibers leave to the pretectal nucleus in the midbrain) , the **pretectal** nucleus is the **center** of the reflex ,some fibers leave the pretectal nucleus in the midbrain, these fibers are called **posterior commissural fibers** and they end bilaterally on "edinger westphal nucleus " on both sides.



'**edinger-westphal nucleus EW**' has preganglionic parasympathetic fibers , and we know that the effect of parasympathetic on the eye is on the ciliary muscle and constrictor pupilae.

From edenger-westphal nucleus arising parasympathetic fibers , and these parasympathetic fibers before reaching the constrictor pupillae muscle in the eye ,they will synapse in a **ciliary ganglion** .

Always remember the rule: autonomic fibers(sympathetic or parasympathetic) before reaching the effector organ must pass through parasympathetic ganglia .

At the beginning of the light reflex, a light is directed toward the eye ---> retina is stimulated---- > nerve impulse is formed (visual impulse)---- >the impulse moves through optic nerve ---- > optic chiasma ----- >optic tract ---- > pretectal nucleus of midbrain ---- > fibers coming out from pretectal nucleus will go to (**edinger westphal nucleus**)---> coming out from the EW nucleus parasympathetic fibers to the same side and opposite side ---- > they enter and synapse with the ciliary ganglia before reaching the eye and stimulate the constrictor pupillae (sphincter pupillae)muscle.

Again, in this reflex: the afferent is the **optic nerve** , the efferent is **oculomotor** (the parasympathetic fibers are part of the oculomotor) and the center is **pretectal nucleus** ☺ ☺ ☺ .

Normally when we expose one eye to the light and the other eye is hidden or covered - for example if the left is covered and the light is directed toward the right eye- there will be pupillary constriction at the **right and left** eyes , the response of the right eye (that was exposed to light) is called **direct light reflex** , while the response of the left eye (that was covered) is called **consensual or indirect light reflex**.

Now, What happens if we interrupt the afferent limb?

If the afferent (optic) was interrupted by MS we will lose the direct and indirect reflexes , there won't be constriction in **both** eyes, because if the **afferent** is interrupted we will never have a response.

But If we interrupt the oculomotor in the eye that is exposed to light we will lose the direct light reflex , but we will not lose the consensual (indirect)reflex .

So if there was no constriction in the right eye after being exposed to light we should think of (lesions in optic or oculomotor) , if the left eye responds to light (i.e. it was constricted) that means the lesion is in **the right oculomotor** (since we lost the direct light reflex only and not both light reflexes).

Light reflex is very important clinically because it tells whether the optic and oculomotor are intact or not .

*Light reflex is different than the **accommodation reflex** (which functions to recognize and see near objects like reading a book)and it has a different pathway. For example the patient of syphilis will have what is called **(Argyll Robertson pupil**) in which the response of light reflex will be lost , but the pupil will respond to the accommodation reflex. So the "Argyll Robertson pupil" confirms that **the pathway of light reflex is different than the accommodation reflex**.

The accommodation reflex:

To be able to see near objects, many changes will take place, the most important three are :

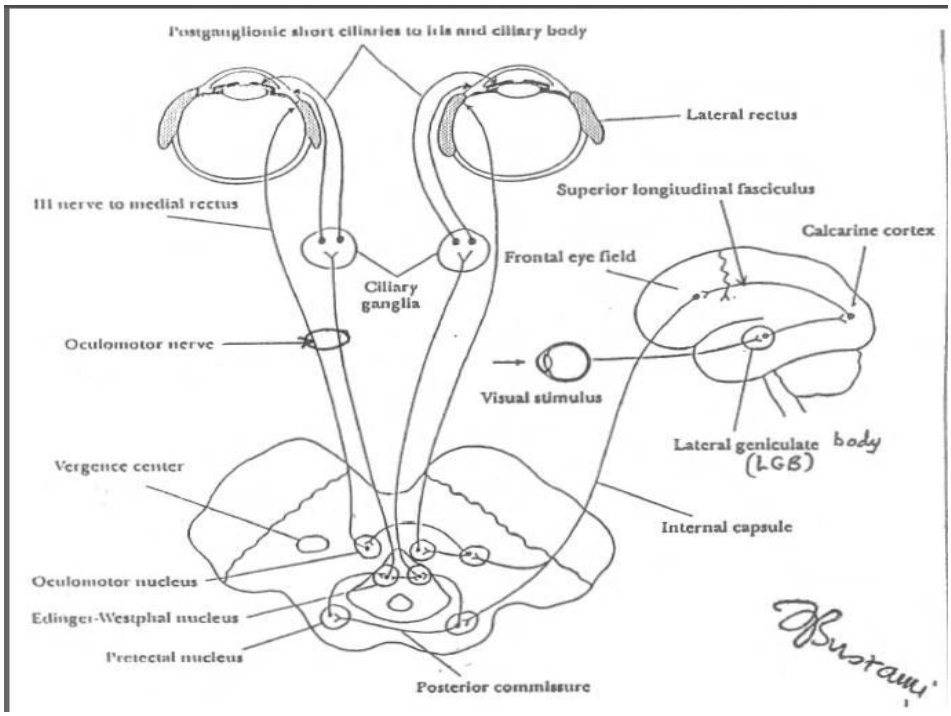
1. increasing power of the lens by increasing the thickness (when the ciliary muscle contracts its diameter will decrease ,relaxing the tension in the suspensory ligaments ,as a result the lens becomes thicker).

Ciliary muscle is stimulated by parasympathetic , Remember : parasympathetic can stimulate two muscles in the eye : ciliary and constrictor pupillae muscle. ☺

2. convergence of the eyes: adduction of both eyes to see near objects .

3. constriction of the pupil.

constriction of the pupil in light reflex was **protective** ,to prevent the light from entering and damaging the retina , but in the accommodation reflex the constriction helps to regulate the **depth of focus** (حتى تقع الصورة على بؤرة محدودة بحيث يُمنع الضوء من التشتت الذي يسبب عدم وضوح في الرؤية), so the constriction limits the light to a certain focus on the retina which is the **macula** (that has the most accurate vision). If light enters and the pupil wasn't constricted it will be distracted and distributed.



The accommodation reflex

the light reaches the eye(the usual sequence : retina, optic nerve, optic chiasma, optic tract , lateral geniculate body in the thalamus) , the optic radiation comes out from the thalamus, optic radiation will reach the calcarine cortex(area17) and then to 18 and 19**(the occipital visual field)**, the occipital visual field will send signals to the frontal visual field through association fibers called (**superior longitudinal fasciculus**) , so we reach the frontal eye field , fibers coming down from the **frontal eye field** will run in the internal capsule , these fibers will go to **the pretectal nucleus** and **vergence center** (both are in the midbrain) , fibers coming out from the pretectal nucleus will go to the **EW** nucleus (like in light reflex) then parasympathetic from this nucleus will form part of the oculomotor , these parasympathetic will not reach the constrictor pupillae muscle directly but will pass through the ciliary ganglion.

So the fibers of **EW** nucleus when entering the eye will cause : constriction of the pupil and stimulation of ciliary muscle, so the parasympathetic

causes the **two changes**(constriction of the pupil and contraction of ciliary muscle that increases the thickness of the lens thus increasing its power).

The third change is **convergence** (adduction of both eyes by stimulating medial rectus on both sides) this is achieved by the **Vergence center** that affects the oculomotor nucleus in the midbrain on this side and the other . fibers coming from the oculomotor nucleus will enter and stimulate the medial rectus on both sides, when both eyes are adducted the image will be on the same area (focus) of the retina .

Accommodation reflex at old age will not be working properly because the lens will lose its flexibility and this is called (**presbyopia** ; inability to see near objects).

the constriction of the pupil is common between light reflex and accommodation reflex, but constriction of the pupil in light reflex is **protective** , while in the accommodation reflex it helps to limit the light on a certain area (focus) in the retina so that it won't to be distributed. So although the constriction of pupil is common in these two reflexes, they have **different pathways** (the accommodation reflex is long and we used the occipital and frontal eye field and other parts) . the **argyll rebertson pupil** (which we see in syphilis when the disease reaches the nervous system) confirms this fact…'again ☺' .

Eye movements :

We already talked about one type of eye movements which is **convergence** . now, the rest of eye movements are called **conjugate movements** : conjugate vertical (elevation or depression of **both** eyes) and conjugate horizontal *(important)* (for example : you adduct one eye and abduct the other one) .

The nucleus of oculomotor (the 3rd) is located in the midbrain, nucleus of the sixth is located in the pons , so there is a long distance between them and there must be some kind of coordination between them.

✎.The muscles that **elevate** your eye are **superior rectus** and **inferior oblique** and both are innervated by **oculomotor** (its nucleus is in midbrain).

✎. the muscles that **depress** your eye are the **inferior rectus** (innervated by **oculomotor**) and the **superior oblique** (innervated by **trochlear** the 4th nerve).

you have to know that when the eye is abducted (away from the nose ☺) it will be elevated by superior rectus and depressed by inferior rectus , but when the eye is adducted (close to the nose ☺) it will be elevated by inferior oblique and suppressed by superior oblique.

✎.The muscle that **abduct** your eye is **lateral rectus** (innervated by **abducent** the 6th nerve) and the muscle that **adduct** your eye is **the medial rectus** (innervated by the **oculomotor** the 3rd nerve). ☺

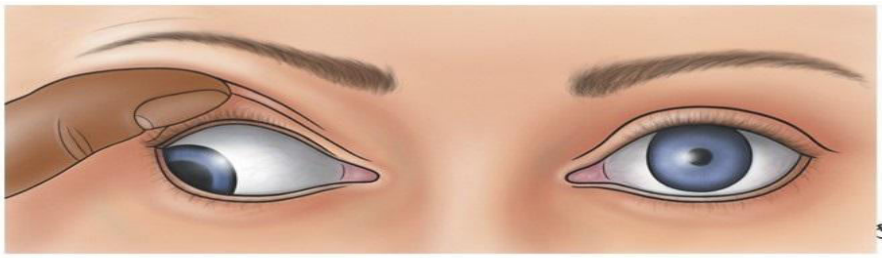
✎.There is a muscle called (**levator palpebrae superioris** رافعة الجفن) that elevate the eyelid. the major part of this muscle is skeletal innervated by **oculomotor**, and a small part of this muscle is smooth and innervated by **sympathetic** .

if we interrupt the sympathetic supply, the result is **Horner syndrome** : small part of the muscle will be paralyzed (little ptosis or mild ptosis) . but if we interrupt the oculomotor the major part of the muscle will be paralyzed (marked ptosis) so the eyelid will cover the eye*(important)* .

Sooo Oculomotor nerve contains somatic fibers that supply all extrinsic muscles of the eye (five muscles out of seven : superior rectus, inferior rectus, medial rectus , inferior oblique and levator palpibrae) and leaves two muscles (superior oblique innervated by trochlear , and lateral rectus innervated by abducent) ☺ .and parasympathetic fibers to ciliary and constrictor pupillae muscles.

so when the oculomotor is interrupted (by multiple sclerosis for example) all extrinsic muscles of the eye will be paralyzed ☹ except lateral rectus (will bring the eye laterally) and superior oblique (will bring the eye downward)

so the eye will be deviated downward and outward which is called **external squint** (الحَوَل الوحشي) , the eyelid is covering the eye, and the pupil is dilated . why is the pupil dilated ? because the oculomotor nerve has somatic fibers supplying five muscles and parasympathetic fibers to the constrictor pupillae , so when the oculomotor is interrupted there will be no parasympathetic , thus the sympathetic will become in charge and cause dilation in the pupil) .



Right eye: Downward and outward gaze, dilated pupil, eyelid manually elevated due to ptosis

Left: Normal

When you raise the eyelid that is covering the eye ***(marked ptosis)*** you will see a fully dilated pupil that doesn't respond to light reflex nor to accommodation reflex (because the main nerve in these reflexes is the oculomotor), and that is a very important sign .

A lesion in oculomotor will cause 4 changes that start with a **D**:

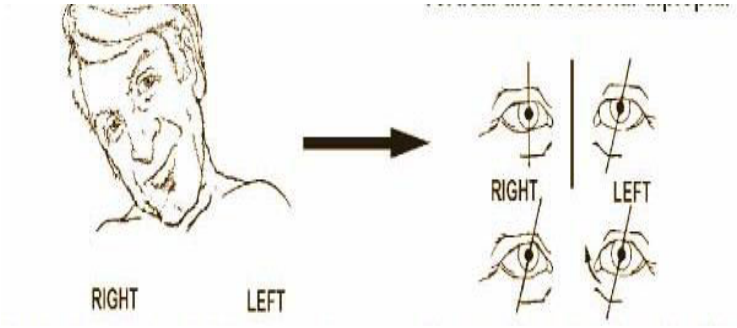
1) **D**rop of the upper eyelid 2)**D**ownward and outward deviation of eye ball.
3)**D**ilation of the pupil . 4)**D**ouble vision(because we lost conjugate movement.)

*A lesion in the **abducent** nerve : the abducent nerve cause abduction of the eye, when the abducent is interrupted the **lateral rectus** will be paralyzed☹, the eye will be adducted by medial rectus ( على مبدأ غاب القط العب يا فار) and the result is **internal squint** , whenever a muscle is paralyzed the antagonist

will take over, so if you asked a patient with a lesion in the right abducent to look laterally the eye will be looking forward or in the adduction position .

*A lesion in the **trochlear** nerve: the trochlear nerve supplies one muscle (**superior oblique**) that **depresses** the eye . *the trochlear is the only nerve that decussate **before** emerging from the **dosal** surface of the midbrain.* if it was interrupted before decusation the effect is **contralateral** which is rare to happen , but if it was interrupted after emerging the effect is **ipsilateral** . when the trochlear is interrupted the superior oblique will be paralysed so the inferior oblique will move the eye upward ( على نفس المبدأ) , the patient will **try to look downward** (when going down stairs or reading) but his/her eye will look upward ! more over, this patient will have **double vision or diplopia** (اذا لم تتحرك العينان معًا فلن تندمج الصورة على الشبكية) .

*the patient will have double vision when he tries to use the superior oblique , so the patient will tilt his/her head to make both eyes at the same level so that the image will be on the same area(focus) at the retina , so if the left superior rectus was paralyzed the patient will tilt his/her head to the right to raise the left eye and make them both at one level . look at the figure :



The end ☺

Written by : Afnan Abu Qaddoum ☺ .

This sheet is dedicated to : sondos dehidi , tasneem suhail, yasmeen khater,noor abu gunaim and tamara ayasra ❀✿❀.