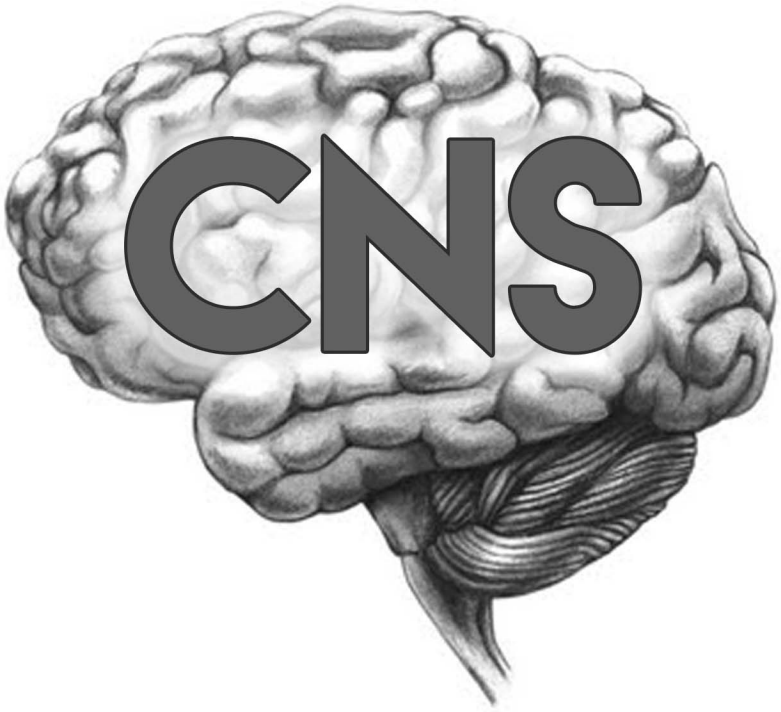


CNS

ANATOMY

☑ Sheet ○ Slide ○ Handout

Number

16

Subject

Done By

ريم الشياب

Corrected by

Heba al lala

Doctor

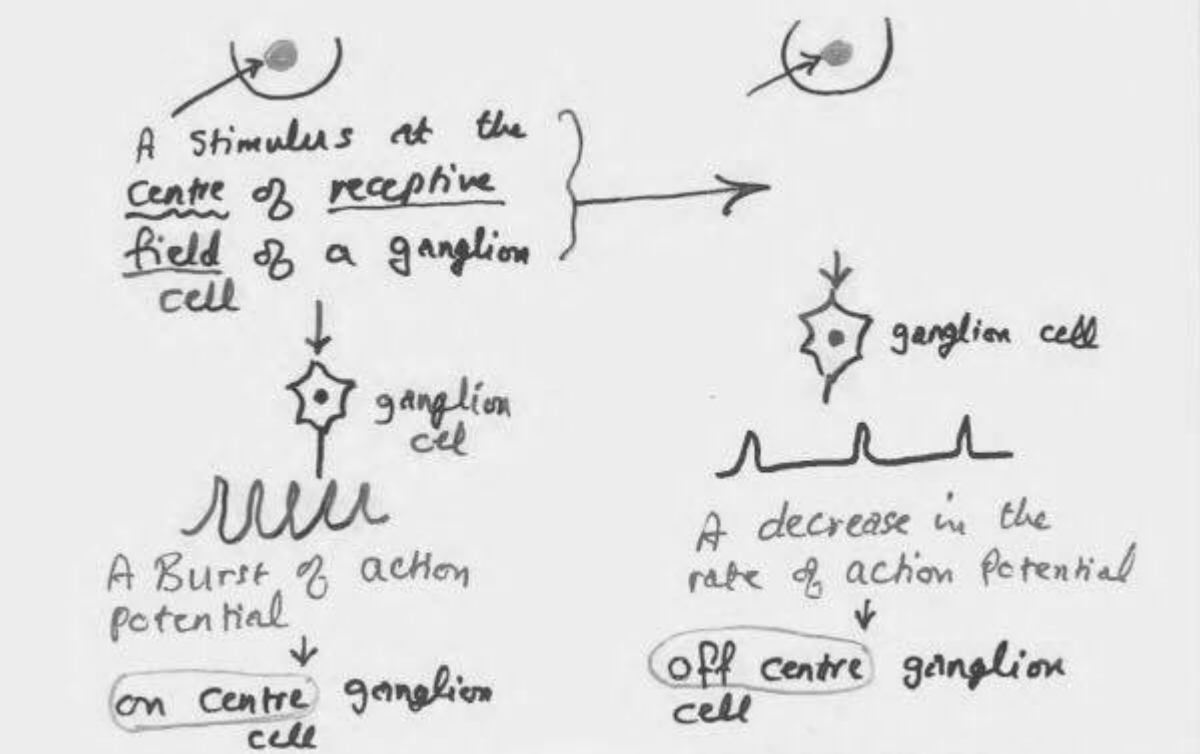
Faraj Al-bustami

Date: **00/00/2016**

Price:

بسم الله الرحمن الرحيم
وبه دوما نستعين

In the last lecture we talked about the retina...
The most important cell in the retina is the ganglion cell, which generates the action potential reaching the brain. Each ganglion cell has a circular receptive field and the receptor field is a group of photoreceptors (rods and cones), this circle is functionally divided into two parts: center + surround, the ganglion cell here is named as On center ganglion cell, why? Because if I put a spot of light on the center of the eye and an action potential was generated increasing the action potential in the ganglion cell then this cell is called on center ganglion cell (excited cell). On the other hand, we also have off center ganglion cell (inhibited cell), which means that the action potential here is decreasing in the ganglion cell when you put the light on the center of the eye.



Usually when we test the ganglion cells we find that most of the ganglion cells are of the **on-center off-surround** type OR **off-center on-surround**, what does this mean?
It means that most receptive fields combine excitation and inhibition, how?

1- if the spot of light on the center of the receptive field excites a ganglion cell, the same cell will be inhibited by light in the circular area surrounding the center. In this case the cell is On center/Off surround cell.
2- if the spot of light falling on the center of the cell is inhibitory while when falling on the surround it is excitatory, the cell is Off center/On surround cell.

What is the benefit of this?
Remember: we said that in order to strengthen a cell in its sensory pathway we have to inhibit the surrounding cells. Example: if I have 9 cells in row and there was a stimulus in the middle 3 cells, we need to inhibit the ones on the sides to have a sharp and strong output from the stimulated ones, and this is called **Lateral inhibition**. Here the same happened, to sharpen the impulse from the center we inhibit the surround, and vice versa.
This lateral inhibition process is one of the functions of the Horizontal cells, which receives impulses from a photoreceptor and inhibits the surrounding receptors. So in the case of the On center/Off surround cells, the horizontal cells block the output from the surrounding pathways when a contrasting illumination falls on the center.

We said that most of the sensory inputs from the retina are received by the thalamic center for vision; specifically, **the lateral geniculate body LGB**, this LGB is composed of 6 layers; in the first two layers the neurons are large and we call them Magnocellular layers (magno: large), and the other four layers (3,4,5,6) are called Parvocellular layers.

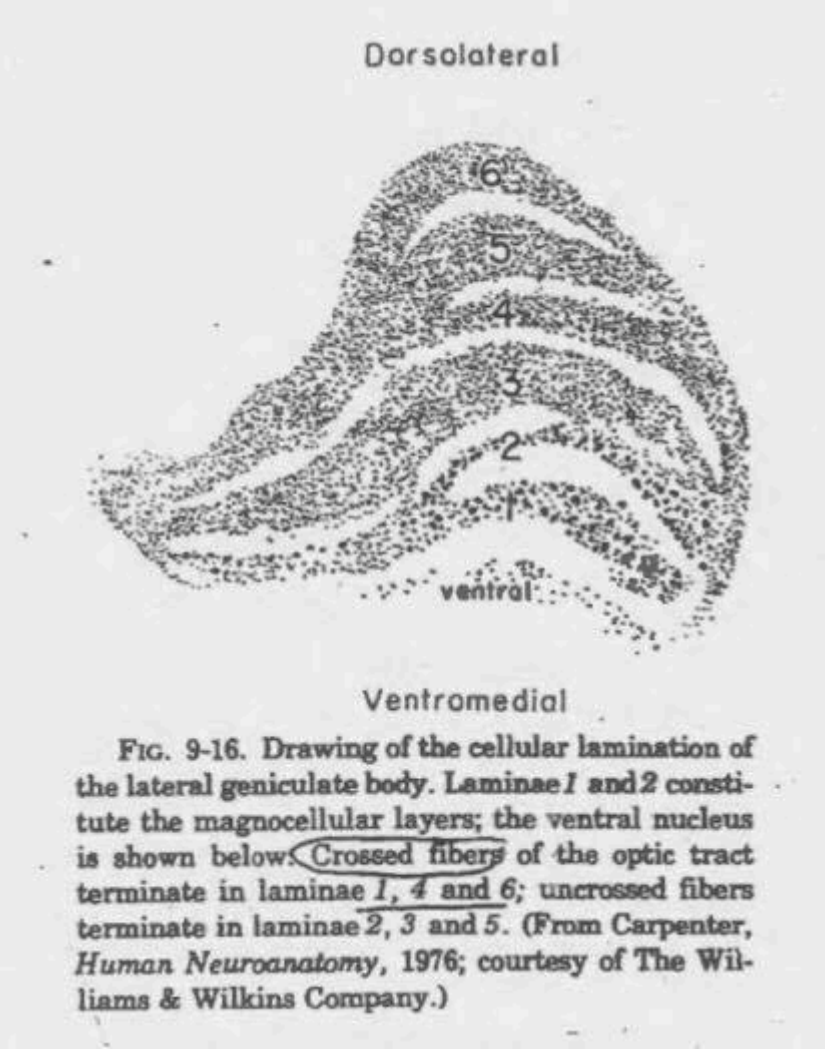


FIG. 9-16. Drawing of the cellular lamination of the lateral geniculate body. Laminae *1* and *2* constitute the magnocellular layers; the ventral nucleus is shown below. Crossed fibers of the optic tract terminate in laminae *1, 4* and *6;* uncrossed fibers terminate in laminae *2, 3* and *5*. (From Carpenter, *Human Neuroanatomy,* 1976; courtesy of The Williams & Wilkins Company.)

When looking to these layers we see that the cells of the thalamus made the optic tract which has temporal of the same side and nasal of the opposite side, the crossed fibers (nasal) come to the 1st,4th,6th layers and they cross going to the opposite side, and the ones that didn't cross (temporal) come to layers 2nd,3rd,5th of the same side, so the cells of the thalamus receive temporal of the same side and nasal of the opposite one. The nasal fibers CROSS and bring information from the contralateral side to layers 1,4,6, and the temporal didn't cross and take information to layers 2,3,5.

Now let's talk about the layers of the retina; there are two main types of ganglion cells in the retina: **large alpha** and **medium sized beta**.

Large alpha:

1- it's located in the peripheral part of the retina
2- it receives input mainly from the rods, also it has large receptive fields and thick rapidly conducting axons.

3- it respond mostly to moving stimuli.

(remember that the main cell is the ganglion cell which generates the action potential. هاي الجملة الدكتور عمل فيها اكثر من دخول مفاجئ في عدّة مواضع مختلفة خلال المحاضرة)

4- its old name is Y cell, but now we name the alpha M cell because it receives information from the magnocellular layer (first two layers).

5- works in the way we discussed; On or Off center/surround pattern.

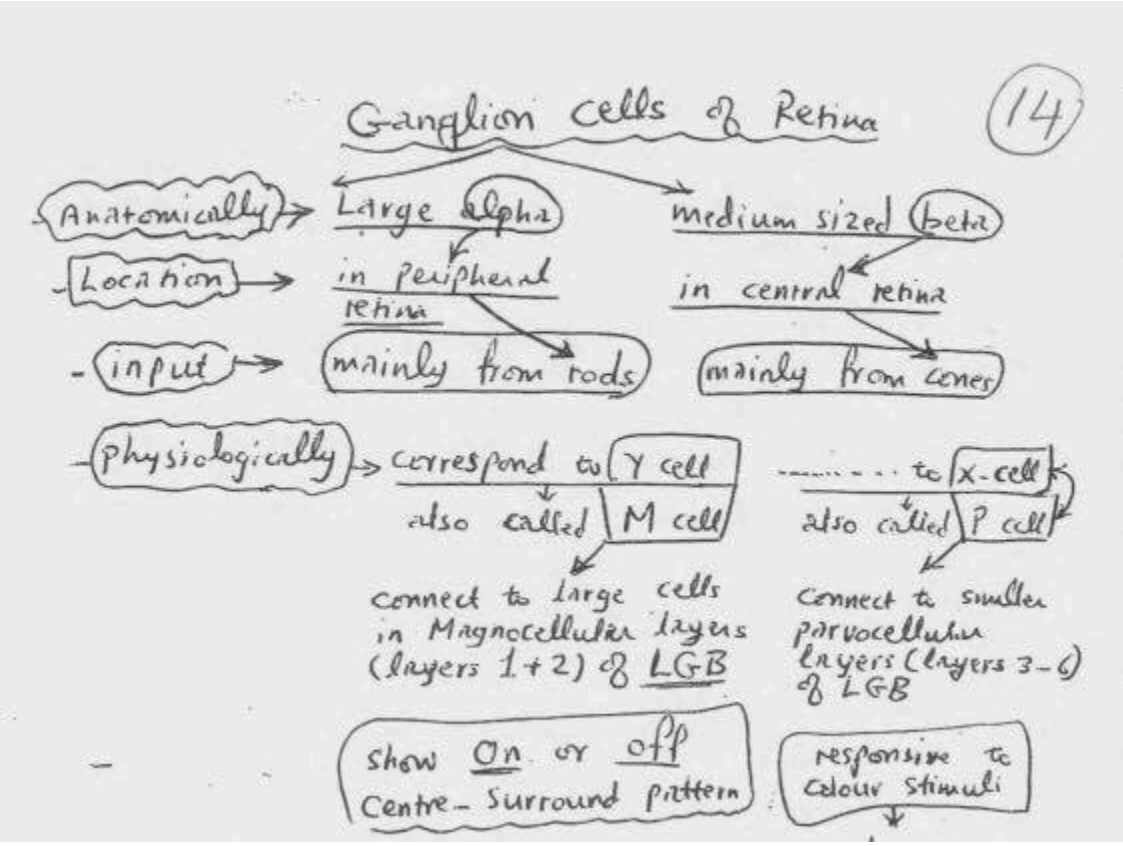
Medium sized beta:

1- it's located in the central part of the retina
2- it receives input mainly from the cons, and it has small receptive fields and slower-conducting axons.

2- it responds mostly to the stationary (fixed) stimuli.

3- its old name is X cell, but now we call it P cell because it receives information from the Parvocellular layer.

4- responds to the color stimuli more.



- The movements of the eye

The extraocular muscles are skeletal muscles innervated by somatic nerves (3,4,6). All extraocular muscles are supplied by the oculomotor nerve except the lateral rectus which is supplied by the abducent nerve and the superior oblique supplied by the trochlear nerve.

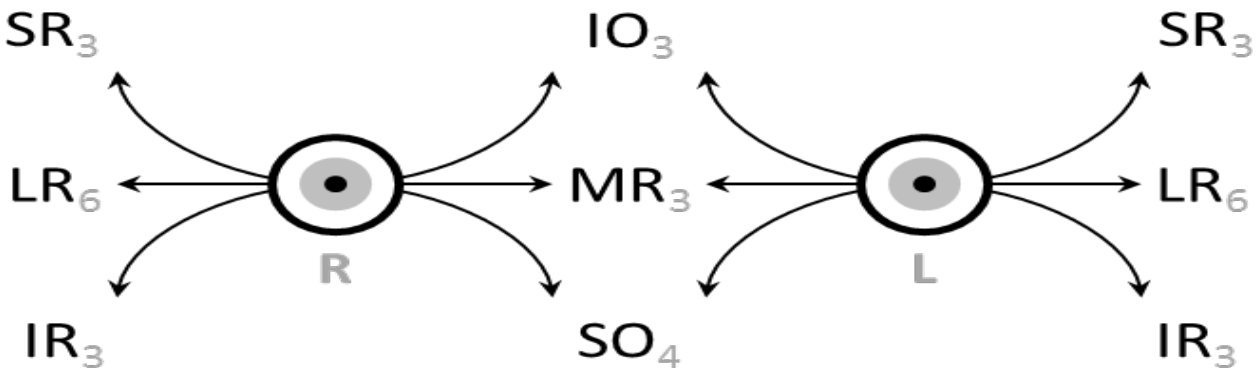
The movements of the eye are of three types:

1. **horizontal**; (abduction of one eye will cause adduction of the other, BOTH together) and if one of them didn't respond the patient will suffer from double vision/diplopia (عيونه بتزغلل), because the picture will not fall on the same area of the retina
(الصورة لن تقع على نفس البؤرة في الشبكية) .
So the movements of the eye must be conjugate, and harmony between them must be there.

2. **Vertical**; both eyes go upwards or downwards together.

3. **Convergence**; both eyes adduct like if you are looking to the tip of your nose.

If an eye was abducted and looking upwards the muscle responsible for this movement is the superior rectus muscle (innervated by the oculomotor) and the inferior rectus moves it downwards (innervated by the oculomotor), but if your eye was adducted the muscle which moves it upwards is the inferior oblique muscle innervated by the oculomotor, and the superior oblique moves it downwards innervated by the trochlear nerve.



- We will start talking about **the trochlear nerve**:

It innervates one single muscle; the superior oblique muscle, it brings your eye downwards when its adducted. It has special characteristics one of them that the nerve crosses after emerging from the nucleus before emerging from the brainstem. So if the nucleus was damaged the effect will be contralateral, whereas if the nerve itself was damaged the effect will be ipsilateral.
*Remember that the nucleus of the 3rd and 4th cranial nerves are found within the midbrain.
If the trochlear nerve which innervates the superior oblique will bring the eye downwards was injured the inferior oblique will move the eye upwards (remember that if a muscle was paralyzed the antagonist muscle will dominate). So when the patient is going downstairs the normal eye will move downwards medially, while the other with injured trochlear nerve will not, here double vision will occur.
For the patient to correct the double vision tilt his head away from the affected side.

Further, we have talked about the visual areas; primary visual area 17 and the association areas 18 and 19 where you recognize what you see, there is another association important area in the inferior temporal gyrus called the inferotemporal area, this area receives from areas 17,18,19 and from area 7, if a bilateral lesion happened to this area the patient will complain from visual agnosia where you see and don't recognize what you've seen.

- **The oculomotor nerve**; it has two parts:

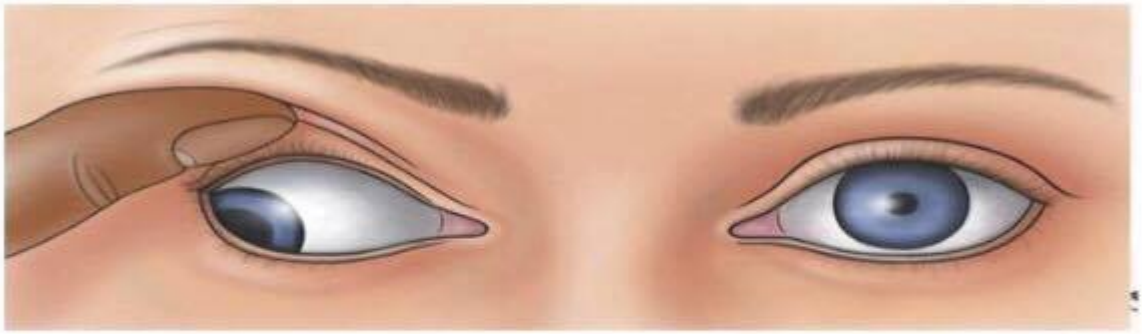
1- somatic to all extraocular muscles except lateral rectus and superior oblique.

2- parasympathetic for two smooth muscles inside the eye which are: the constrictor pupillae and the ciliary muscle. So if the oculomotor nerve was injured due to demyelinating disease (MS for example) both the somatic and parasympathetic parts will be affected causing:

- All extraocular muscles will be paralyzed except the superior oblique and the lateral rectus muscle, thus the patient will complain of downward and outward deviation of the eyeball.
- The skeletal part of the levator palpebrae superioris (رافعة الجفن) which elevates the upper eyelid and when its injured there will be MARKED ptosis and this sign is mostly specific for patients with oculomotor injury.

(this muscle has two parts: one is skeletal supplied by the 3rd cranial nerve and the other is smooth supplied by sympathetic innervation and if the sympathetic was cut in the head and neck, the patient will suffer from Horner's syndrome with mild ptosis).

- Markedly dilated pupil with no response to light or accommodation reflex.
- Diplopia



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

Left: Normal

Note: When the oculomotor nerve emerges from the midbrain it emerges between two arteries; the posterior cerebral and the superior cerebellar which are branches from the basilar artery, and when a nerve passes between two arteries in such a case its highly susceptible to be pressed by an aneurysm from one of the arteries. Example; if we found that a patient has an aneurysm in the posterior cerebral artery or in the superior cerebellar artery, which nerve is at risk of damage or compression?
It's the Oculomotor nerve.
We talked previously about **alternating hemiplegia** which is when a patient has an ipsilateral and contralateral presentations in different parts of the body, for example when a patient has hemiplegia on his left hand and leg and a damaged right oculomotor nerve. And always when we want to examine a patient with stroke we must examine his cranial nerves because the lesion may be in the brainstem, and when I see that the patient has hemiplegia on his left hand and leg and the right oculomotor nerve, then the lesion is in the right half of the midbrain (NOT in the internal capsule) because what deprived the nerve from its blood supply deprived the pyramidal and extrapyramidal too.

- **The abducent nerve**; it abducts the eyeball by the lateral rectus muscle, and when its paralyzed on one side the medial rectus will dominate one the same side bringing the eyeball to inside (adducting it), and the patient can't abduct the eye on the affected side while the other will move normally and the patient will suffer from double vision.

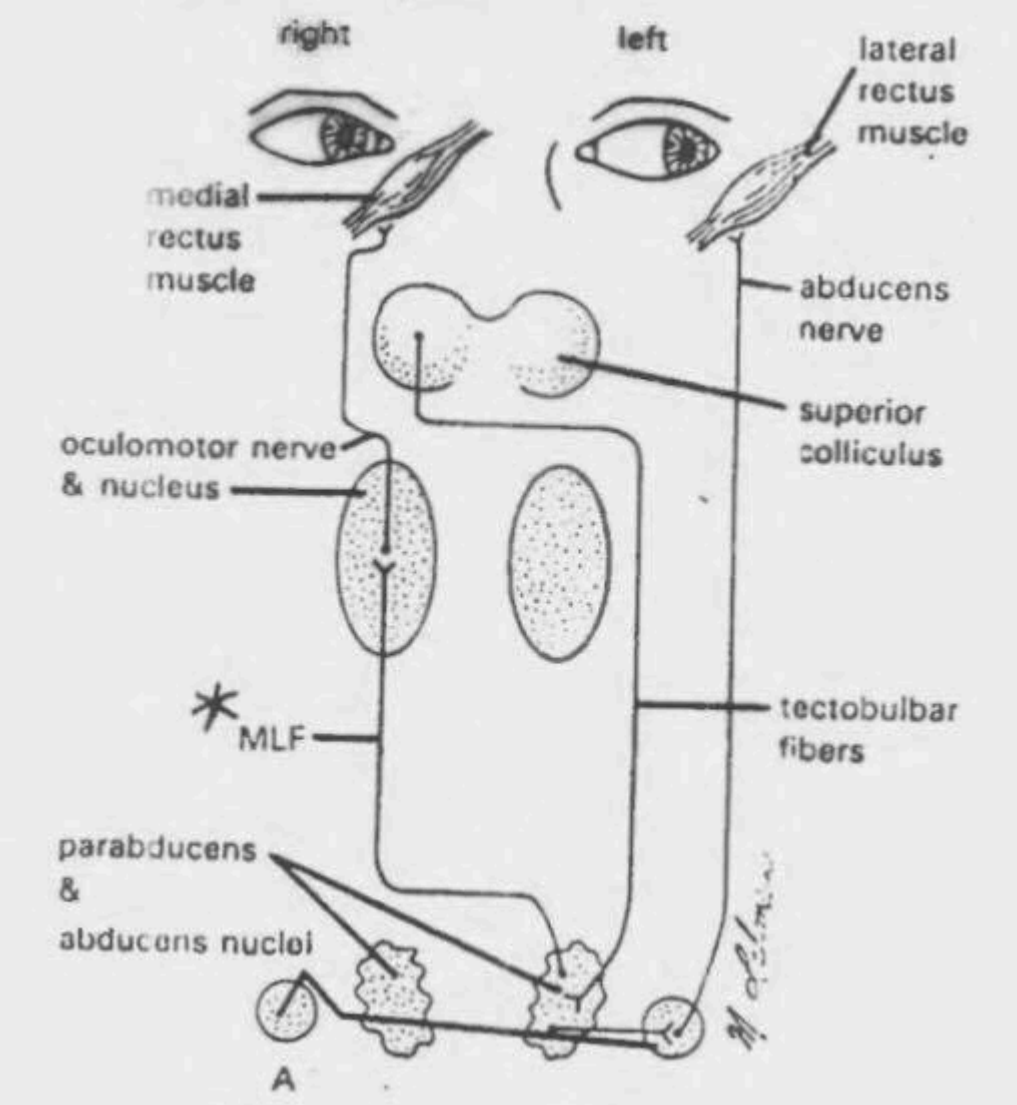
Remember that the oculomotor, trochlear and abducent nerves receive information from:
1- the frontal eye field (area 8): if it was stimulated on the right cerebral hemisphere the eyes go to the left, and if it was damaged on the right cerebral hemisphere the eyes come to the right as we said before.
2- the occipital eye field (areas 17,18,19).

The frontal eye field is responsible for fast eye movements that can be tested by asking the patient to look right and left rapidly and this movement is called Saccadic movement, while the occipital eye field is responsible for the pursuit movement (حركات التتابع) where I ask the patient to follow my finger to test it.

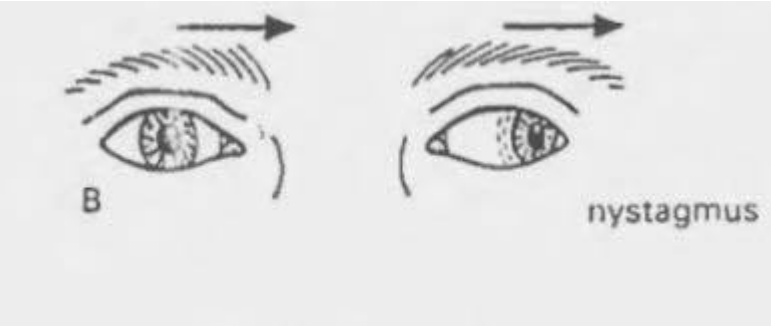
How do they affect the eye movements?
If I want for example to stimulate the frontal eye field on the left side, then the eyeballs will move to the right, and in order for this movement to occur I need to stimulate the right lateral rectus (right abducent in the midbrain) and the left medial rectus (left oculomotor in the pons).
And as we said too that the way that coordinates between the 3rd and 6th cranial nerves is the medial longitudinal fasciculus MLF. So a lesion of the MLF affecting the parabducent and oculomotor nuclei produces internuclear opthalmoplegia which means loss of coordination between the two eyes.
If we stimulate the left frontal eye field and the left occipital eye field the eyes will go to the right, how?
Information will descend from the frontal eye field and the occipital one to the superior colliculus on the same side, and from the superior colliculus through the tectobulbar tract which crosses the midline and descend from the midbrain to the pons specifically to the parabducent nucleus of the paramedian pontine of the reticular formation PPRF of the pons, this nucleus sends signals to the abducent nerve adjacent to it and the abducent nerve supplies the right lateral rectus muscle of the same side of the nuclei, and by this we've abducted the left eyeball.
Now to stimulate the part of the oculomotor that innervates the medial rectus, we have other fibers that emerge from the parabducent nucleus and go through the MLF to the oculomotor stimulating the left medial rectus.

Now by stimulation of those two muscles we completed the accurate needed movement.
The MLF coordinated the movements and it connected the abducent on the right and the oculomotor on the left, if it was injured we lose the coordination between eye movements causing double vision.

Now let's look at the picture below for a patient with a lesion of the right frontal eye field, if the right frontal eye field was injured the left is going to dominate and when the left is stimulated the eyes will go to the right, meaning that we used the right lateral rectus and the left medial rectus, then immediately signals will descend from the left eye field to the left superior colliculus which gives the left tectobulbar tract crossing the midline and descends to the right parabducent nucleus which stimulates the abducent nerve near to it innervating the lateral rectus and abducting the right eyeball, and through the MLF it ascends to the oculomotor stimulating the medial rectus adducting the left eyeball.



- Lesions of the MLF:
  If the MLF was injured, we call this case **MLF syndrome** or **internuclear ophthalmoplegia** (internuclear because we cut the way through parabducent nucleus and the oculomotor nucleus). If I suspected that the lesion is in the right MLF I ask the patient to look to the left, while if I suspect that the lesion is at the left MLF I ask him to look to the right.
  - Now if I had a patient with a lesion in the right MLF and I asked the patient to look to the left; the left eyeball will abduct normally by the left lateral rectus muscle, while the right eyeball will not adduct and will observe nystagmus in it trying to coordinate with the other normal eye to avoid double vision.



Looking to this figure:

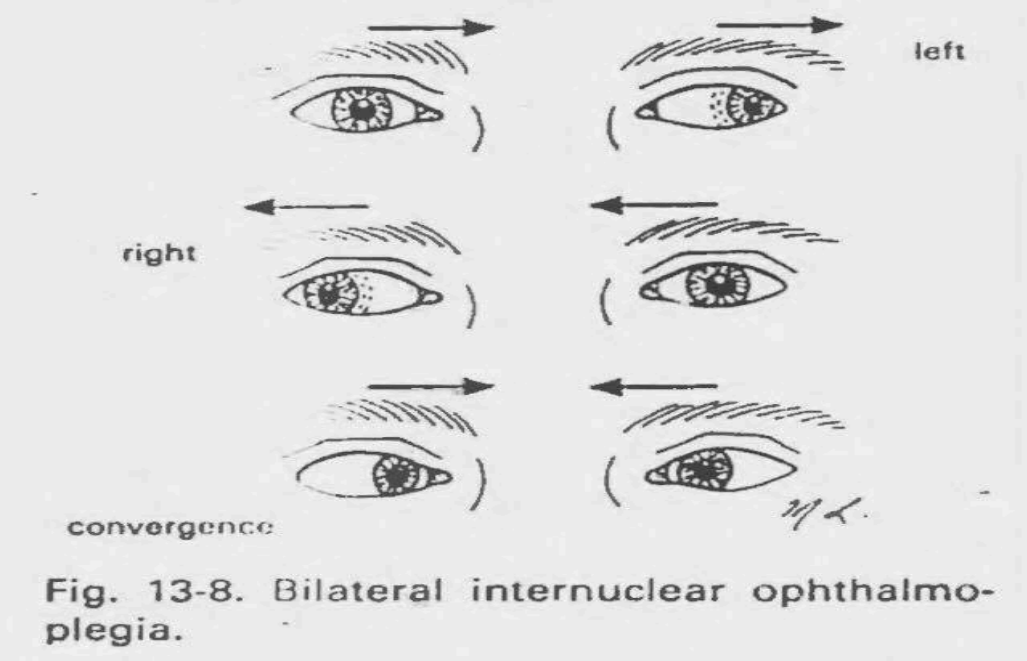


Fig. 13-8. Bilateral internuclear ophthalmoplegia.

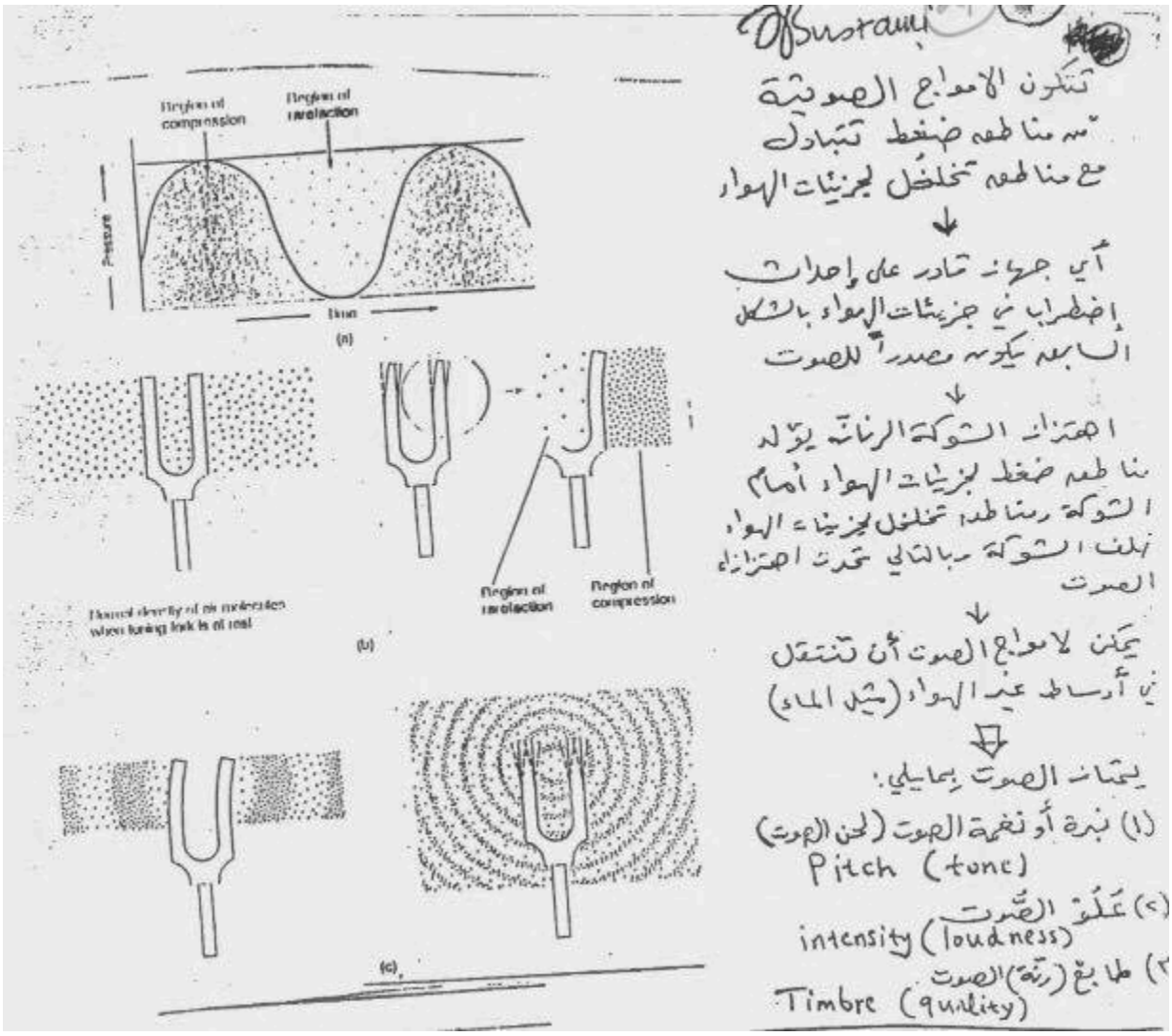
The lesion is in the MLF NOT in the medial rectus, and we test this by asking the patient to do convergence and he do it normally which means that the medial rectus muscle on both sides is working and the lesion is in the way between the abducent and the oculomotor, this case is called Bilateral MLF syndrome. Remember that we said MLF syndrome occurs in multiple sclerosis which occurs more in females, presentation mostly at the age 30-40 years when the patient come complaining of loss of vision or diplopia, we conclude that the damage occurred to the optic nerve.

The auditory pathway

Receptors of hearing and balance are in the INNER ear, we can find the cochlea القوقعة where receptors of hearing are found (in organ of corti especially hair cells), while the balance receptors are found in three semicircular canals in addition to a utricle and a saccule.
The cochlea is a spiral-shaped tube, making 2.5 turns around its axis, and it has both a basal turn and an apical turn, and inside the cochlea we find organ of corti where we find hair cells inside receiving audio, meaning that the function of these hair cells is converting sound waves to action potential.

How do sound wave form?
Firstly, sound waves are areas of compression alternating with areas of rarefaction, if I knock the tuning fork; compression will be in front of it and behind it will be rarefaction and by this alternation we will have sound waves.

How do sound wave travel?
Through air, water and bones of the skull.



What are the characteristics of sound?

1- pitch/tone: it depends on the frequency of the waves. The higher the frequency, the higher pitch.

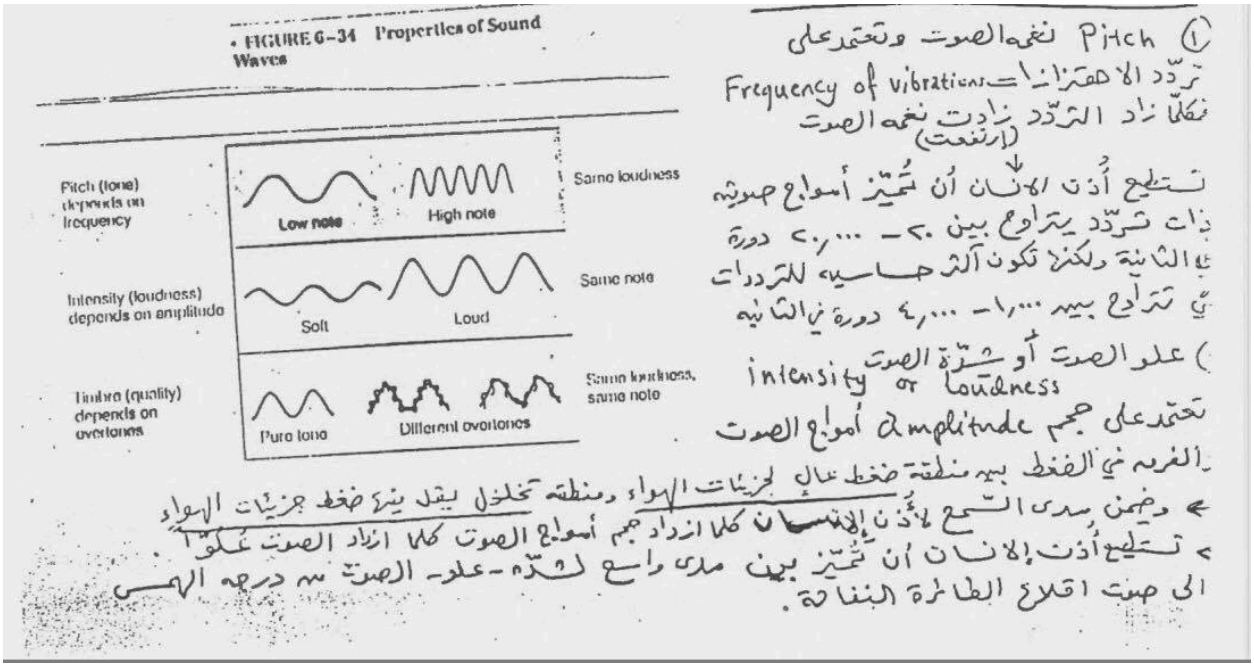
Females usually have higher pitch than males, except some doctors in our faculty: P

Human's ear responds to frequencies from 20 cycle/sec to 20,000 cycle/sec (from whisper to the take-off of the plane), the beneficial frequencies that our ears are most sensitive for them range from 1000-4000 cycle/second.

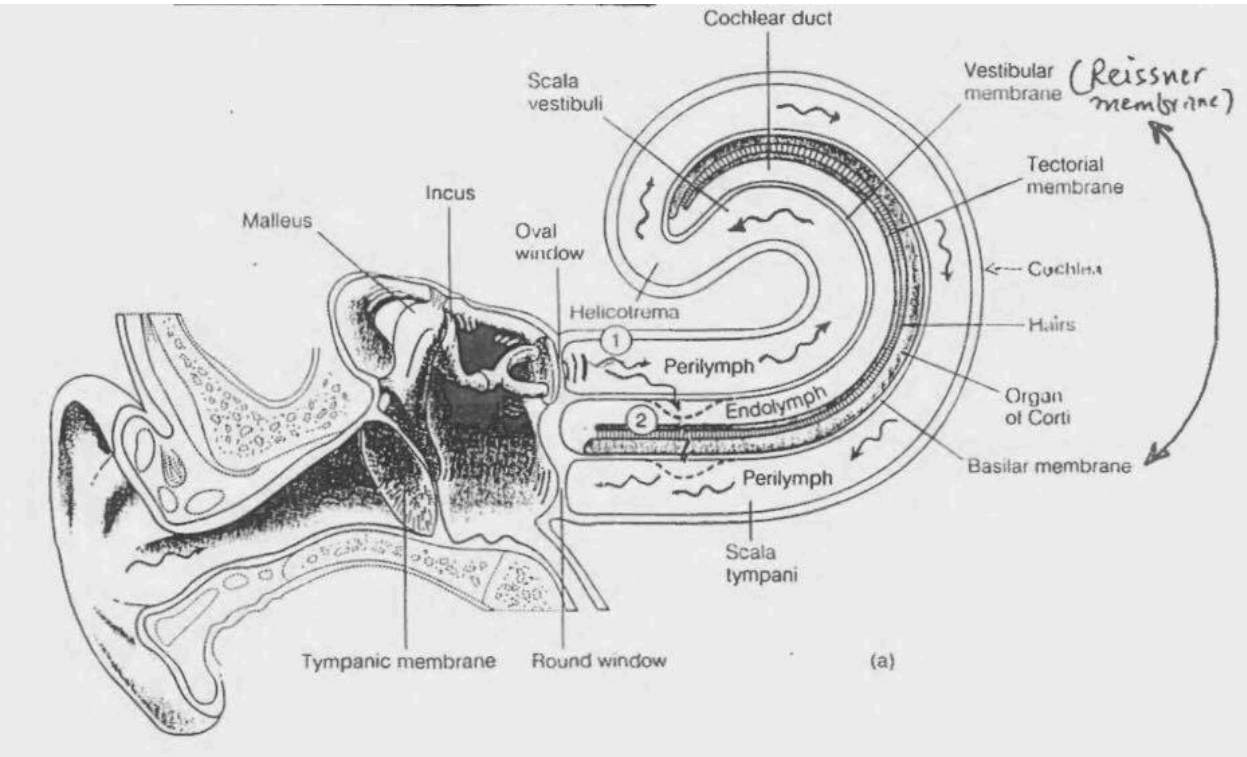
We measure the noise of voice by a unit called the Decible.

2- Intensity/loudness: depends on the amplitude of the waves. The higher the amplitude the higher the intensity.

3- Timbre/Quality: each person has a different quality that allows us to differentiate between different voices of people.



How does the voice travel?
By looking to the figure below we can see the external ear > ear drum > tympanic membrane > middle ear > internal/inner ear.
The sound waves travel as vibrations from the external ear to the ear drum, when the ear drum vibrates it causes the tiny bones of the middle ear to vibrate (malleus, incus, stapes)*, then the vibrations will travel to the membrane of the oval window then it will go to the cochlea, the cochlea is divided into three layers: the upper layer has perilymph called Scala vestibule, the middle layer contains endolymph and is called Scala media/Cochlear duct and here are the receptors, the inferior layer is called Scala tympani which has an opening at its end covered by a membrane called the round window.



- *those tiny bones have joints between them, if those joints undergo fibrosis they will stop moving in response to the vibration of the ear drum affecting the hearing process, this condition is called otosclerosis and it could be congenital or acquired.

  Now Look to the figure above again which is a section in the cochlea and notice the following:
  - The superior layer is **Scala vestibuli** containing perilymph which is rich in sodium like the extracellular fluid.
  - The middle layer is **Scala media/Cochlear duct** containing endolymph which is rich in potassium like the intracellular fluid, also here we have the receptors -organ of corti and hair cells -.
  - The inferior layer is **Scala tympani** containing perilymph too.
  - The roof of the middle layer is called Vestibular membrane or Reissner's membrane.
  - The ground of the middle layer is called Basilar membrane, and this membrane is where the hair cells are resting, those hair cells have sterocilia (hair) which are connected superiorly to the tectorial membrane.

- When the vibrations reach the Oval window, pressure waves will form compressing the perilymph, this fluid could move from the upper layer across the Helicotrema back to the inferior layer and cause vibrations in the Round window and this is useless! In order to have a beneficial movement, hair cells must be stimulated and this is done by vibrations in the basilar membrane, forcing the hair cells to move upwards and downwards, leading to the movement of sterocilia connected to the tectorial membrane to move forwards and backwards too away from the limbus, leading to the opening of ion channels (Na+, Ca++) in the roots of the hairs leading to depolarization. While when the hairs move towards the limbus they lead to the closure of the sodium and calcium channels leading to hyperpolarization. What causes the movement of the basilar membrane? When the fluid moves it compresses the Vestibular/Reissner's membrane >> this membrane compresses the endolymph >> leading finally to the compression of the basilar membrane >> vibration of the basilar membrane up and down.

To sum up: vibrations traveled from the external ear>> ear drum>> middle ear>> inner ear>> and finally it vibrates the basilar membrane. If the final step doesn't happen, the hair cells aren't going to be stimulated and no action potential formation will occur.

Up-and-down movement of the basilar membrane & tectorial membrane causes the stereocilia extending from the hair cells to bend BACK & FORTH

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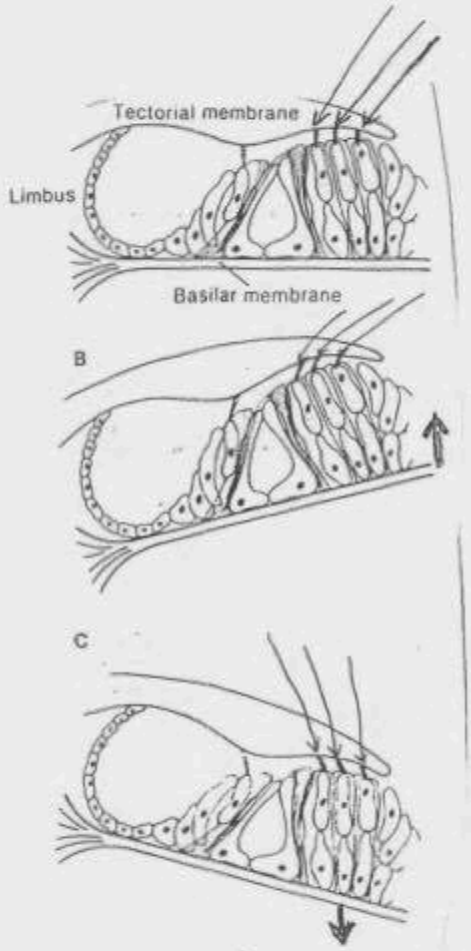


Depolarize

hyperpolarize

B - When the organ of corti moves upward (with the basilar membrane) → the stercocilia bend AWAY from the limbus & depolarize

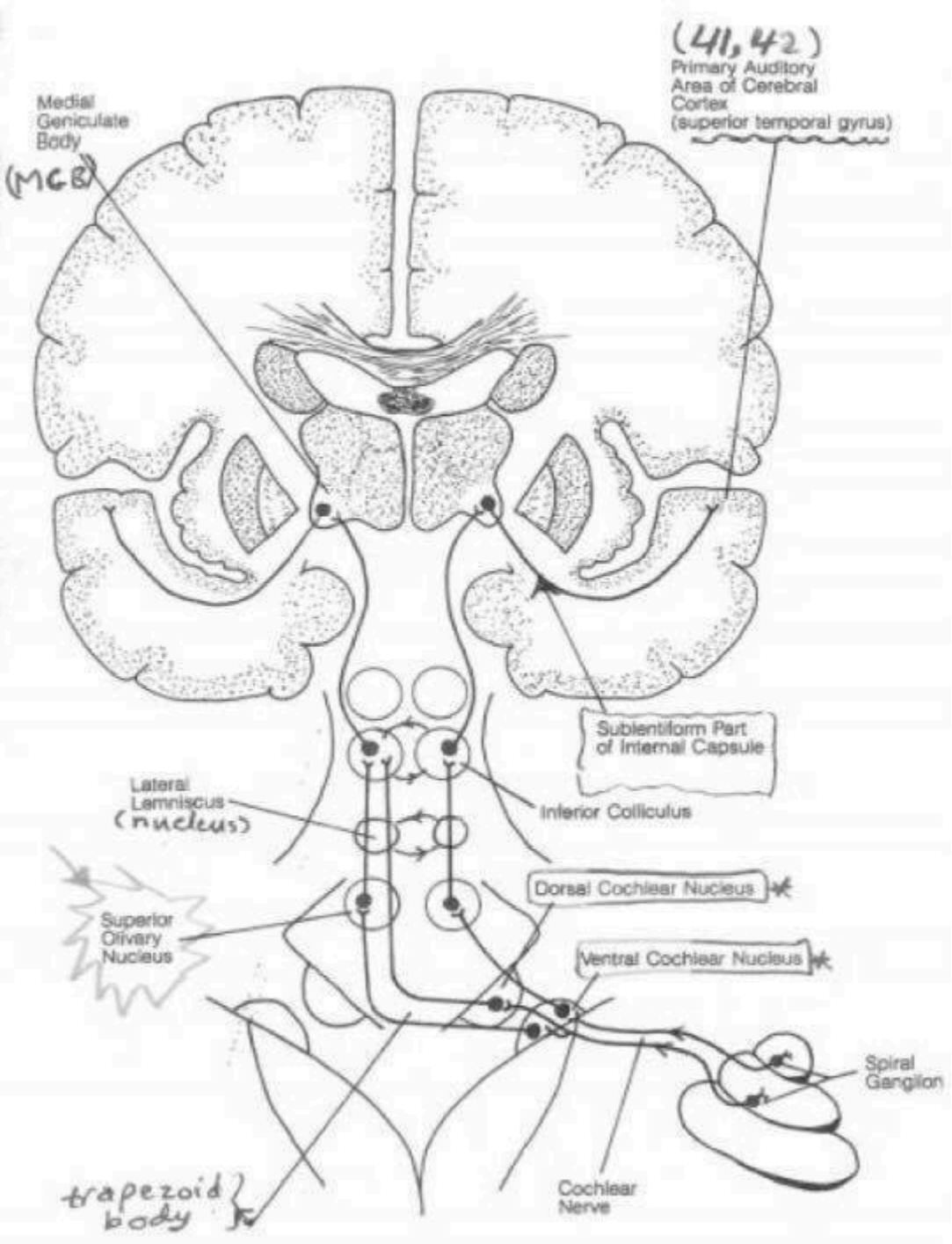
C - When the organ of corti moves downward the stercocilia bend toward the limbus & they hyperpolarize

Now let's look to the basilar membrane; its begging is near the oval window where its thin and stiff and it responds to high frequency sounds (high pitched) = 20,000 cycle/second, while its wide terminal is near the Helicotrema and this part responds to low frequency sounds (low pitched) = 20 cycle/second.
Looking again to the auditory pathway; any sensory pathway starts with receptors which are hair cells found in the organ of corti in the inner ear, especially in Sclala media of the Cochlea, and they rest on the basilar membrane, superior to them is the tectorial membrane.
Any receptor receives peripheral process from cells in the ganglion, so if we have a dorsal root ganglion (with NO synapse) its dendrites will go to the receptors and its axon goes to the spinal cord. Here the same rule applies, we have a **spiral ganglion** sending its peripheral processes to the receptors and its central process to the Pons towards two cochlear nuclei: dorsal cochlear nucleus and ventral cochlear nucleus. So the nuclei here represent second order neurons, while the first order neurons are found in the spiral root ganglion.
Also here at the level of the dorsal and ventral cochlear nuclei we can specify the pitch/frequency; the dorsal root ganglion receives from the basal turn of the cochlea (high frequency sound), while the ventral root ganglion receives from the apical part of the cochlea (low frequency sound).

The axons of the ventral and dorsal cochlear nuclei (2nd order neurons) ascend in an area of the pons called the tegmentum forming Acoustic striae (dorsal, intermediate and ventral), the ventral acoustic stria is called the trapezoid body. The dorsal and intermediate acoustic striae (axons of the 3rd order neurons) emerging from the dorsal cochlear nucleus crosses the midline aiding in the formation of the contralateral Lateral lemniscus.
The ventral acoustic stria (trapezoid body) synapses on two nuclei: 1- superior olive 2- nucleus of trapezoid body (3rd order neurons), axons emerge from the 3rd order neurons going mostly to the opposite side and minority of them to the same side, and this means that each lateral lemniscus receives from both sides mainly from the contralateral. So signals coming from the right ear reach both cerebral hemispheres mainly to the left.
Again, each lateral lemniscus receives from both ears mainly from the contralateral one.

The nucleus of the lateral lemniscus here is 3rd order neuron, its axons go to the inferior colliculus in the midbrain which is the 4th order neuron. Then from the inferior colliculus to the Medial Geniculate Body of the thalamus (MGB for hearing while LGB for vision), then from the thalamus to the primary auditory

cortex (area 41,42) through the Sub-lentiform part of the internal capsule ascending as the Auditory radiation.



The primary auditory cortex is found deep in the lateral fissure, specifically in the superior surface of the superior temporal gyrus and this area is known as the transverse gyri of Heschl; because the gyri are oriented horizontally not vertically, and each column receives different frequency; the posterior columns receive high frequency, while the anterior columns receive low frequency, and this leads us to the fact that each level of the auditory pathway differentiates the frequencies, ex: at the level of the basilar membrane, cochlear nuclei, and also the auditory cortex. This area receives the sounds and the association auditory (area 22) which is located on lateral surface of the superior temporal gyrus integrates the sounds into something that we can understand, also it integrates the coming sounds with auditory memory stores, that's why you can recognize voices of people that you heard years ago!

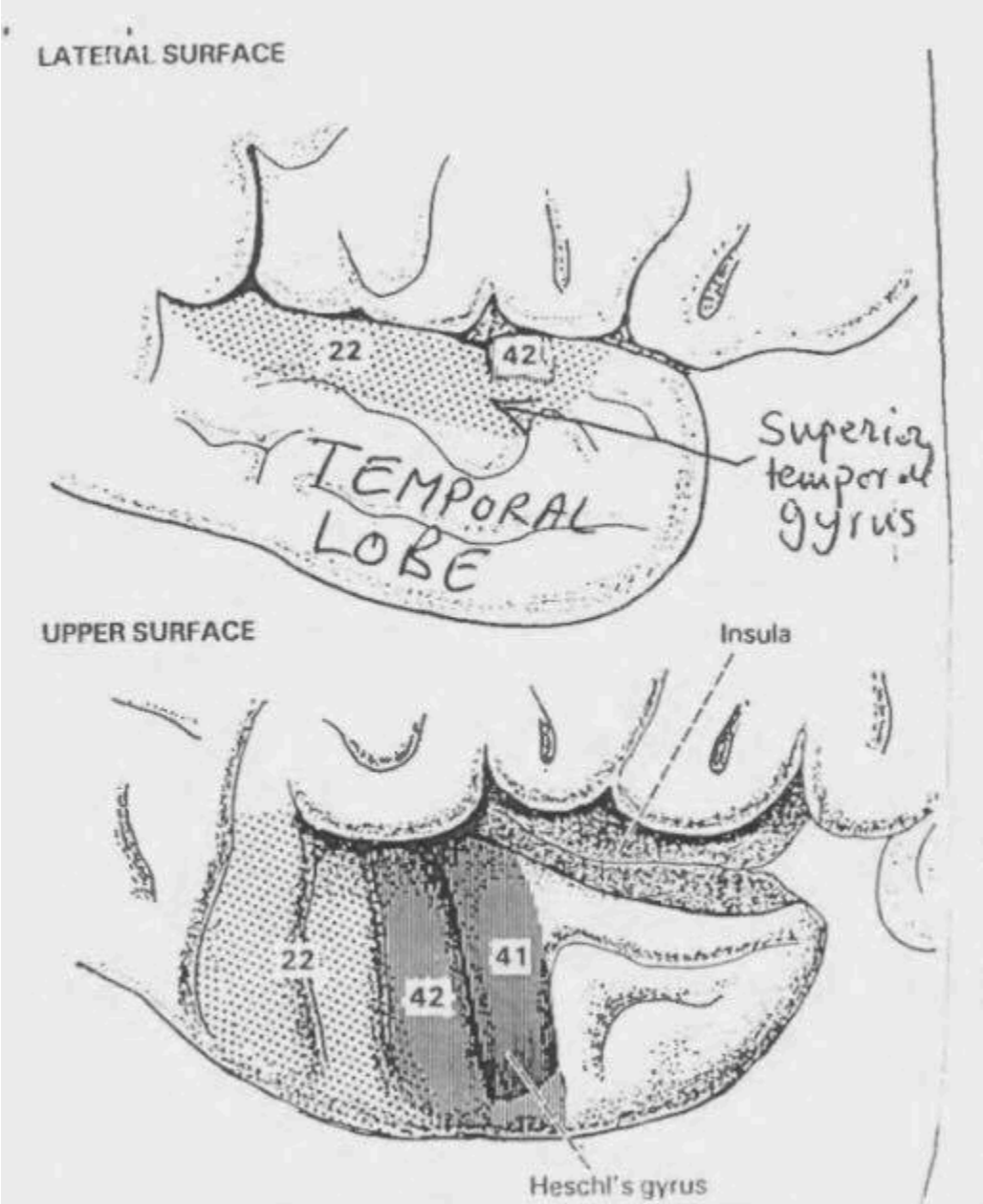


Fig. 30-9 Two views of the primary (41, 42) and secondary (22) auditory cortex.

Remember that we have both superior colliculi which aids in the visual pathway and if it was damaged we lose integration between movements of the eyes and **no** blindness will occur, and inferior colliculus which is an integral part of the auditory pathway and if it is damaged the hearing will be markedly affected.

- Lesions:

The association auditory cortex (Area 22) is divided into 6 areas; the most important one is the **wernix sensory speech area**, and if this area was injured the patient will suffer from sensory aphasia in which the patient can see and hear normally but he doesn't understand what he hear, and his speaking muscles are not paralyzed so he speaks fluently but his sentences and words are not understandable.

Later on we will talk about motor aphasia occurring when a lesion affects Broca's area in the frontal lobe, here the patient can understand what you say to him but he can't put words together to form a complete sentence, usually it is associated with depression.

When Unilateral Deafness occurs?
If the damage affects the receptors, the nerve or the cochlear nuclei. Everything after the cochlear nuclei (superior olive, lateral lemniscus, inferior colliculus, thalamus or the cortex) affects Bilaterally, and as we said the lateral lemniscus receives mostly from the contralateral ear and to a lesser extent from the ear of the same side, so it's lesion leads to Bilateral partial deafness (impaired hearing) mostly on the Contralateral ear.
How does Unilateral Deafness due to nerve or receptor damage occur?
By drugs that cause damage to hair cells, mainly antibiotics and especially Streptomycin which is used to treat Tuberculosis. If you give the patient a high dose for 1 week it will lead to deafness, and this case is called Nerve Deafness.
While if the external ear was obstructed by the wax secreted as a protective substance to our ears, sound waves will no longer enter through air from the external ear and it will start entering through the skull bones, and this will cause Conducting Deafness which can be treated and its less dangerous than Nerve deafness.

This is all what we have for today's lecture.
I tried to make it simple as possible as I can, sorry for any mistake.

" العينُ تُبصرُهُ، فالقلبُ يبتهجُ .. معَ البساطةِ فيهِ العلمُ يمتزجُ
مهما بلغْتَ بعلمِ الطبِّ مبلَغةً .. لم تفقَهِ الطبَّ إنْ لمْ يعطِهِ فرجُ "

ريم محمد الشيّاب