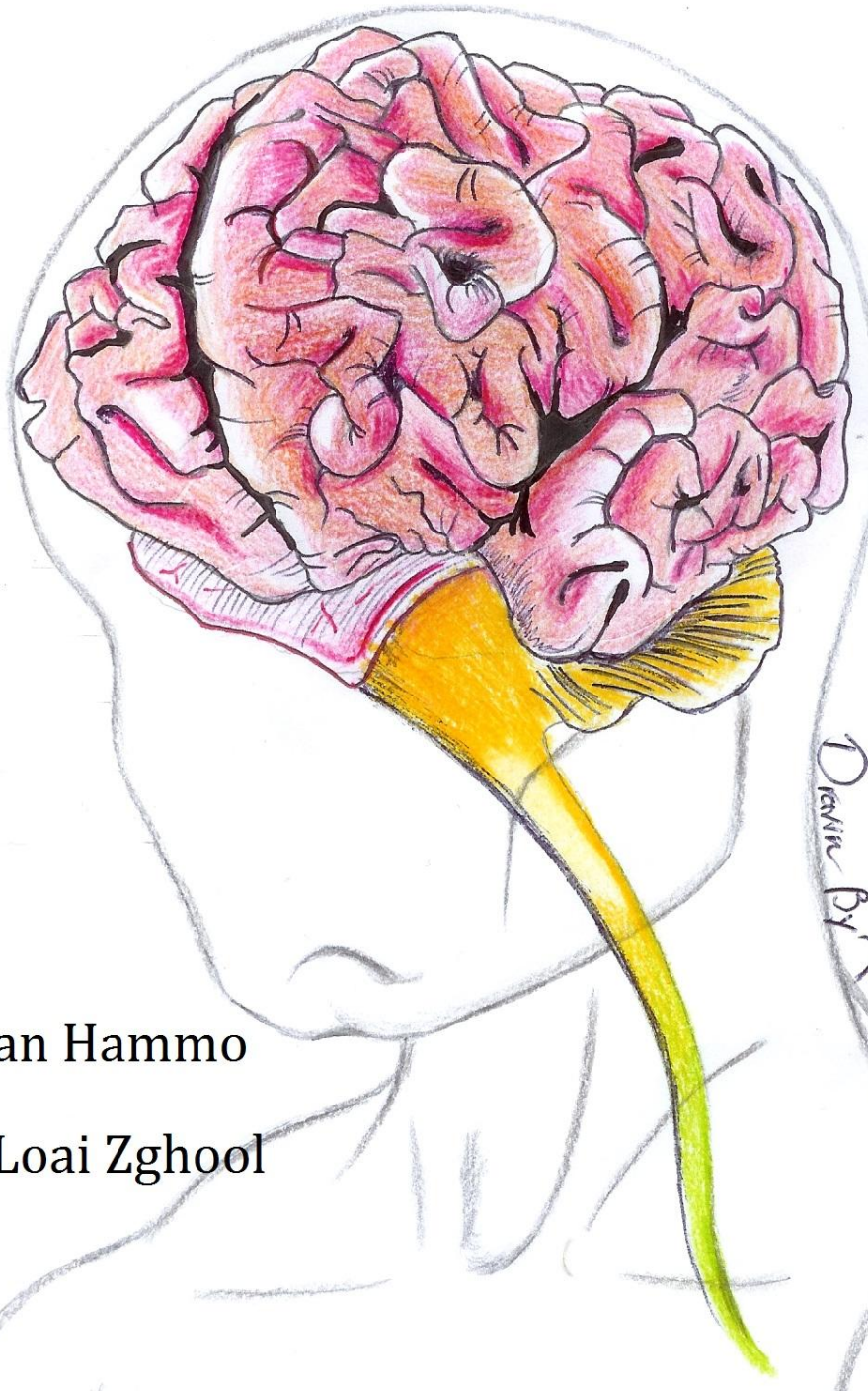
CENTRAL NERVOUS SYSTEM

- ○ Handout
- ● Sheet
- ○ Slide

- ○ Anatomy
- ● Physiology
- ○ Pathology
- ○ Biochemistry
- ○ Microbiology
- ○ Pharmacology
- ○ PBL



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بسم الله الرحمن الرحيم

The Gustatory and Visual Sensations

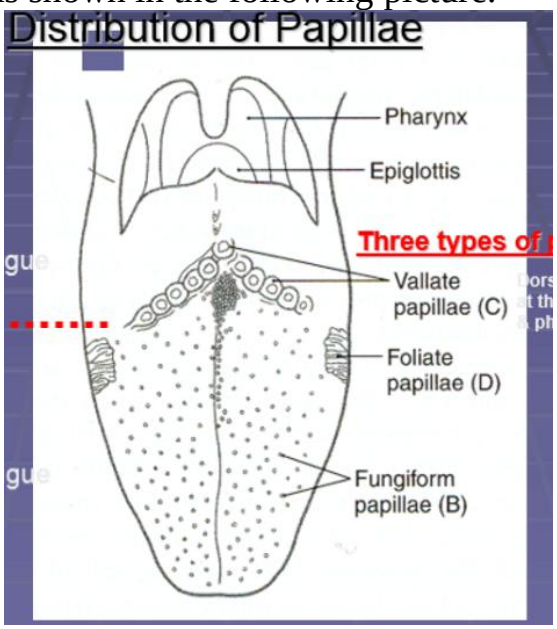
Gustatory Sensation (Taste)

Last time we stopped at the mechanisms of taste sensation. Taste sensation starts at the taste receptors, which can be found lingually and extralingually, like in the soft palate. However, the main taste receptors are those that are found on the tongue.

We have different types of receptors to differentiate between different types of taste. Most taste receptors of the tongue are found in special structures called papillae. (Note that the types of papillae and their shapes are not required in this physiology course).

There are 5 types of taste receptors that distinguish the 5 types of taste sensations, or basic taste modalities. They are: bitter, sweet, sour, salty, and umami. Umami is a Japanese word meaning delicious. It distinguishes savory tastes of meats, proteins, and especially foods that contain monoamine sodium glutamate.

They are distributed as shown in the following picture.



Please keep in mind that all parts of the tongue have all types of these taste

receptors.
*The mechanism of action and activation of these receptors is not required.

The sensation is detected in the receptor by certain mechanisms (not required). The receptor will then translate this chemical signal into an electrical signal and conduct it to the CNS through three cranial nerves: 7 (facial), 9 (glossopharangeal), and 10 (vagus). The facial nerve is the most important of the three, as it delivers sensation from the anterior two thirds of the tongue and the soft palate. It delivers these sensations to the brain stem to enter a special tract called the solitary tract, which will take the sensation rostrally to a special nucleus called the solitary nucleus (gustatory) in the upper pons.

The afferent fibers of these cranial nerves will synapse with the dendrites of the 2nd order neurons found in **the solitary nucleus**, and the axons of these fibers will continue on to the thalamus. After synapsing in the thalamus, the impulse will be taken to the primary taste cortices, found in **the postcentral gyrus** and insular lobe.

Disorders of taste will result in ageusia (complete loss of taste) or hypoageusia. This is mainly a result of the inability of the chemicals *to reach* the receptors, due to a lack of solvent (saliva). This lack of solvent can be caused by certain types of cancer, chemotherapy, or radiation induced. It is also seen in DM and Alzheimer's patients.

To compare and contrast with smell. We have 5 taste receptors, but no glomerulus formation like in the olfactory pathways. Therefore, there is no convergence of taste sensations like in olfaction, and ultimately we can only detect 5 tastes. Yet many different flavors are detected. How? These different flavors are detected by the *coordination* of olfaction and taste. Flavor is a complex sensation caused by the integration of many types of modalities (mainly taste and olfaction). Olfaction is a much bigger player in the sensation/perception of flavor since it works by 2nd messenger systems, while taste sensations work by direct opening of ion channels. Evidence of this is seen when you lose your ability to smell in cases of stuffy nose or common cold. Here, although nothing happened to your taste receptors, you lose the ability to detect flavor of foods.

The integration between smell and taste does not happen directly between their respective cortices, but instead through a specialized association cortex that takes from many sensations. These sensations include mainly smell, taste, and also a bit from proprioception, ALS (to give us pain of spicy foods), and

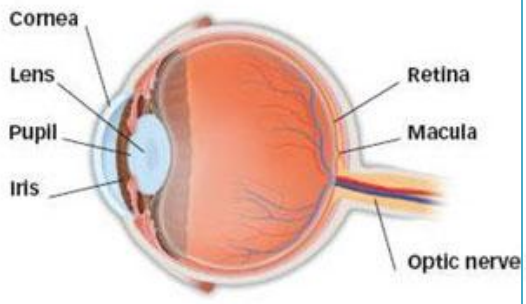
visual sensations. They all go to the medial orbitofrontal cortex, which is the association cortex where flavor is determined. This is why you may not enjoy the flavor of tasty food that looks bad (evidence that it's not just about the taste but many other associations).

Disorders from book; Dysagueia (Distortion in the perception of taste) One of the most frequent causes of taste disturbances is drug use.
Meningioglioma.

Visual System.

Anatomy of the Eye.

There are 3 layers surrounding the eye: the sclera, choroid, and retina. The **sclera** is the hard outer shell, and makes up the shape of the eyeball. It is continued in front by the translucent cornea. The **choroid** layer underneath it contains the blood and nerve supply. The **retina** contains the pigments and the photoreceptors, and it is the most important layer in this physiology course.



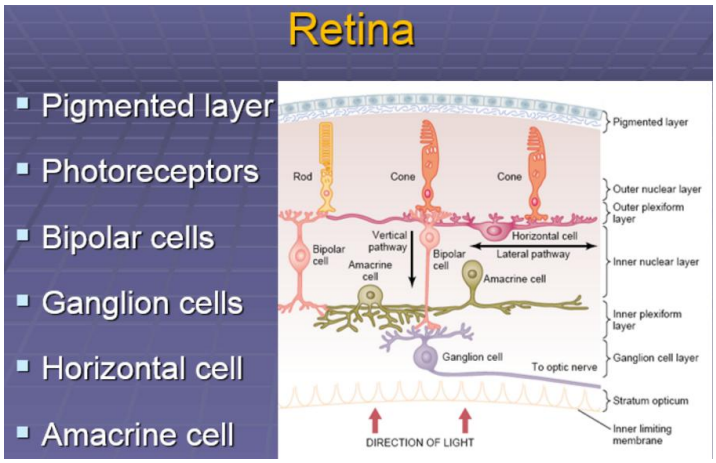
Anteriorly, we have the concave cornea. Its main function is to refract light waves انكسار الضوء and reassemble them. Behind the cornea is the lens, whose function is to help the cornea. Most of the refraction of light occurs here, as well as the accommodation reflex, which we will learn about in upcoming lectures inshallah. The lens is usually elastic, and this helps keep it round. It is attached to a muscle (ciliary muscle, by means of suspensory ligaments) which, when contracted, will result in the thickening of the lens. This parasympathetic reflex is needed for accommodating vision to focus on nearby objects.

Between the cornea and the lens is the pigmented iris, which gives the different colors of the eye from green to blue to gray. However, the main function of the iris is to control the size of the pupil and consequently control the amount of light that enters the eye. It dilates in the dark to allow as much light as possible to enter, and constricts in bright light to prevent too much light entering and damaging the retina.

The extraocular muscles facilitate the many different eyeball movements, and they are:
-The medial rectus: adduction of eye, innervated by occulomotor (III)
-The lateral rectus: abduction of eye, innervated by abducent (VI)
-The superior rectus: elevation of eye, innervated by occulomotor (III)
-The inferior rectus: depression of eye, innervated by occulomotor (III)
-The superior oblique: depression of eye, innervated by trochlear (IV)
-The inferior oblique: elevation of eye, innervated by occulomotor (III)

The eye contains aqueous humour fluid and vitreous humour semi-solid. Anterior to the lens is the aqueous humor and posterior to the lens is the vitreous humor.
*Their composition and cycle is not required.
What you need to know is that there is always continuous production of fluid and continuous filtration and exudation of it. This happens in the corner of the eye in an area called the **canal of Schlemm**. If the production increases or the filtration decreases, the amount of fluid in the eye will increase. This will result in rising intraocular pressure. This increased pressure will compress the photoreceptors which will eventually stop working. It will also compress the small vessels, cutting off blood supply and ending in death of these photoreceptors. This condition is known as **glaucoma**. المياه الزرقاء
Treatment of glaucoma includes drugs that reduce intraocular pressure, and if that doesn't work surgical methods may need to be considered. *Keep in mind that cataracts الماء البيضاء is the change of lens into a little bit less translucent (clouding), affecting the entry of light.

The retina is the most important part of the eye. It contains the photoreceptors that will absorb the light. These receptors are distributed all over the eye.
This picture shows the direction of light as it passes through the different layers in the retina.



Light will pass through all of the layers of the retina before actually reaching the receptors, the rods and cones. It will first pass through the ganglion cells of the first layer. These ganglion cells produce the action potential (they are the only cells in retina that produce an AP) and transmit these signals to the CNS through the optic nerve, which is actually the aggregation of the axons of these ganglion cells.
So light is **not absorbed** by these ganglion cells. It merely passes through them on its way to the outermost photoreceptors.

The second cell is Bipolar cell. It has 3 ends; dendrites and axons. It can do some processing, but it doesn't have photoreceptors activity.

After the bipolar cells come the photoreceptor layer that contain the rods and cones. These are the cells that will absorb the light and translate it into **electrical information**. Light will cause **hyperpolarization** of these photoreceptors by causing a conformational change in **rhodposin**, the protein in the photoreceptors. This will lead to the breaking down of cyclic GMP (cGMP). The depletion of cGMP will close the Na+ ion channel gates, and will decrease the membrane potential from -40mV to a much lower membrane potential, resulting in hyperpolarization. This is the ONLY receptor in the human body that reacts to a stimulus (light) by hyperpoloarization. When the receptor as depolarized (as it normally was during the dark and absence of light stimulus) it released large quantities of the neurotransmitter glutamate. Now that the receptor has become hyperpolarized, it will no longer release glutamate.

The last layer of the retina after the photoreceptors is the pigmented cell layer. The

pigmented cell layer is basically epithelium that contains variable amounts of the pigment melanin, the same pigment found in skin. Its function is to absorb any extra light that hasn't been absorbed by the photoreceptors. If the layer after the photoreceptors did not absorb this extra light, it would bounce off the retina and become scattered inside the eye, distorting vision.

Amacrine and horizontal cells process light.

Photoreceptors:

The retina has two types of photoreceptors, cones and rods.

Besides the fact that cones are smaller than rods and have less outer segments than them, the main differences between them are:

1- They contain different types of proteins. There are three different types of cones, each containing a unique protein to absorb a specific spectrum of light and thus a specific color. Color is for cones. There is only one type of rod cells, and they have only one type of protein. Therefore, it will only detect black, white, and grayscale, while the combinatory absorbed spectrum of cones will allow us to see an entire palette of colors.
Color---> Cones

2- Location of the photoreceptors. The center of the retina is called the macula lutea. It contains mainly cones, with the majority localized in one area called the fovea. This makes sense, because what I want to see in my direct line of sight should be as detailed and colored as possible.
On the other hand, the more peripheral you go, the more rods you will find proportionately, along with a subsequent decrease in the number of cones.

3- The amount of photoreceptor protein in rods is higher than in cones. Therefore, the ability to detect light, or sensitivity to light, in rods is much higher than cones. And because rods are highly sensitive to light, they can detect a light stimulus at a lower threshold. I.e., not much stimulus is needed to activate the rods due to their very high sensitivity, and so in low amounts of light (ex. nighttime or in the dark) the rods will be activated rather than the cones.

This will allow you in the dark to see the shape and figure of an object in front of you, but you will not be able to see the colors of this object, since not enough stimulus is present to activate the high threshold of cones.

The Retina.

The retina covers the entire eye and is the innermost layer. It has two specialized points.

The first of them is the entry point of blood supply. This area also acts as the exit point of the optic nerve. Anatomically, it is the optic disc, and physiologically this area is called the blind spot. It is called as such because no image is detected here. There are no photoreceptors here, only nerve axons forming the optic nerve.

A little ways lateral to the blind spot is the macula lutea (or fovea centralis). This area has most of the retina's cones. It exclusively contains cones, i.e it has no rods. This area contains the fovea, which transmits the highest definition image detected by the eye (detailed). Asem is amazing.

Therefore, when we want to see something with the highest clarification possible, we turn our eyes to focus the image onto our fovea. Anything not focused onto our fovea will be detected by our peripheral retina, but won't give as accurate an image as it would have were it detected by the fovea.

The fovea contains only cones, and a high number of them are densely packed together in this small area. Also, here, the bipolar cells and ganglion cells are displaced laterally a bit to allow for uninterrupted passage of light directly to the photoreceptors. These characteristics of the fovea help in providing the best image at this site.

Disorders affecting the retina

Some disorders will lead to death of certain areas of the retina, and are called macular degeneration diseases. Some if these diseases are the result of normal aging, but others may happen in younger ages (Juvenile), which may be referred to as "**Stargardt**

disease". These disorders affect **cones** more than rods, and mainly affect the macula.

In cases where the disease affects the macula, it will be destroyed. However, the periphery will still intact. This will lead to an image that is viewable from the periphery yet hollow in the center, I.e. the macular area. Behavior treatment; these patients tend to look from the angle of the shoulders at an object to contain it within their peripheral vision.

Facial agnosia, when you look at people's faces but can't recognize its details.

Another disorder that could lead to death of cells in the retina is the detachment of the retina from the choroid layer, thus cutting off blood supply and nutrients from the retinal cells and subsequently leading to their death.

Photoreception:

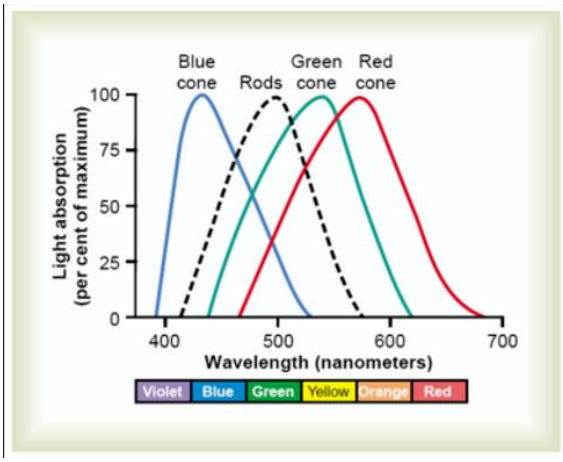
Remember we have only one type of rod and only one type of protein in these rods. Therefore there is no combination of detection between receptors (like the combination seen in olfaction). It will only allow us to see white, black and gray scale in between. This is because it doesn't work as on/off. As the intensity of light increases to a certain wavelength, we will have maximum hyperpolarization and maximum difference in action potential frequency. At other wavelengths, the amount of hyperpolarization will be less and therefore

the frequency of action potentials will decrease. This is seen in the curve.
At 500nm for rods there is a maximum light absorption frequency, and as you increase or decrease the wavelength past this maximum, the frequency will decrease.

There are 3 different types of cones, each with their own unique protein. The absorbance of light wavelengths of each differs. The low wavelength cone absorber absorbs blue light at a maximum intensity of 420 nm. The middle green and highest red also have similar curves but with maximum intensities at higher wavelengths. Due to interaction between the absorbance and combination of frequency of the three types of cones we can see all colors. Ex. To detect the color green, which has a wavelength of about 500 nm, blue cones will absorb 30% of the light, green cones 75% of the light, and red cones 30%. The absorbance of each shows the intensity of the stimulus in each receptor. Each receptor will then react according to how intensely it was stimulated, and will send that amount of signal to ganglion cells to send to the optic nerve for the color to be pictured.

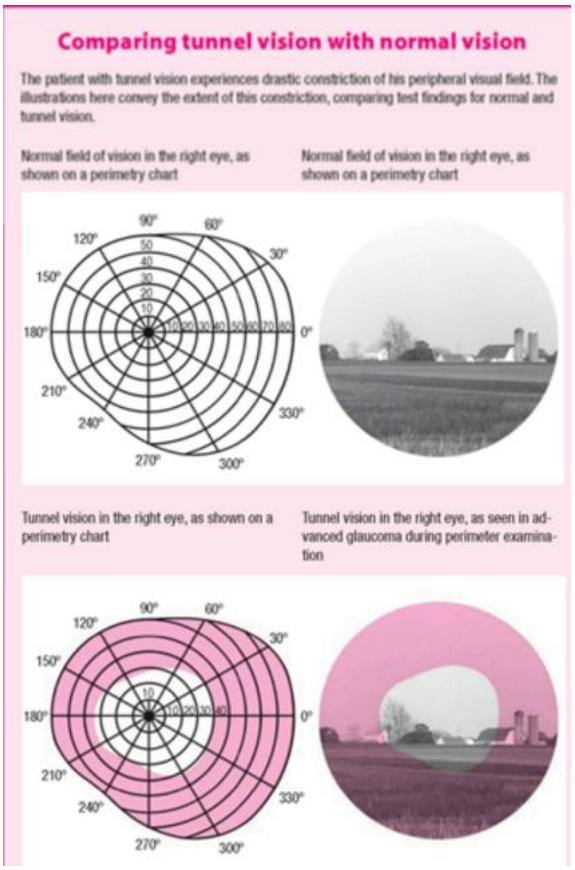
Light activates rhodopsin complex in rods, (photopsin in cones) and this leads to degradation of cGMP. The ion channel closes and no entry of sodium, this leads to hyperpolarization, and finally cessation of glutamate release.
Note: in the dark, there was continuous glutamate release because Na+ ion channels were open, along with open Ca+2 ion channels, depolarizing the membrane.

The main protein of rods is rhodopsin, which is made of vitamin A. People who are deficient in vitamin A will have less rhodopsin available to absorb light. Sensitivity to the stimulus is highly determined by concentration of the photo-absorbing protein. So deficiency in vitamin A will lead to less rhodopsin, which will result in less sensitivity to light. The



remaining rods will have a higher threshold and will require a greater light stimulus which cannot be provided at night. These people will therefore develop night-blindness. To treat them, give them vitamin A (carrots). In a month or two they will recover.

Light adapatation



The amount of light that enters the eye is very important; very high light causes problems, very low light, we won't be able to see. Therefore we have adapted some light reflexes, like the pupillary reflex (to be taught after the mid i.e not included), in which light will cause papillary constriction and dark will cause papillary dilation.

Another reflex is photoreceptor adaptation which depends on the amount of protein available. Receptor will adapt to the amount of light; if it was targeted by a large amount of light, activity will decrease. This explains why when you move from dark to light, you first don't see then start seeing gradually.

As the concentration of protein increases, the adaptation increases. Cones have a small amount of photo absorbing protein, and therefore, although their adaptation is faster, quality of adaptation and sensitivity is less. But because rods have a larger amount of protein, and although adaptation takes a longer time, in the end their sensitivity and adaptation will be greater and this will allow them to see at a lower threshold.

Last of the retinal disorders, degeneration and death of rods. The rods die, and the melanin filled pigmented cells will replace them in the periphery. This will affect the periphery of the retina, and peripheral vision will decrease. This is called **retinitis pigmentosa**. The center is seen but periphery is not, this is called tunnel vision.

I advise you to study this topic well from Dr. Faraj's handout :P
The pages required from the book from chapter 20 are pages 267-272 until receptive fields.
This sheet is dedicated to Ali Tamimi since no one was taking physio :p
Also dedicated to Mo'nes Badaineh for sitting next to me while I typed this up
And of course Mohammad Abu Alia for correcting this mess
Other props go to Baha Shraideh who I finally understand what writing a sheet for dr Loay means :P and Mamoun Suleiman rafeeq al sa3a 7 <3

Good luck