PHYSIOLOGY

SHEET NO. 5

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A quick recap for lecture 4:

Pain, temperature & crude touch and pressure are transmitted by the anterolateral spinothalamic pathway.

- 1st order neurons enter the dorsal horn and synapse with the nuclei of 2nd order neurons
- 2nd order neurons cross-over, anterior to the central canal and ascend in the anterior and lateral white columns **contralaterally**.

Pain has two types -> fast and slow.

The synaptic delay occurs with each synapsis. A greater number of synapsis makes the transmission *slow*, hence slow pain. The opposite happens with fast pain.

Fast pain	Slow pain
Stimulation of mechanical and thermal nociceptors	Stimulation of polymodal receptors
Transmitted by Aδ fibres	Transmitted by C fibres
Sharp, prickling, electric & acute	Dull, aching & chronic
Easily localised	Poorly localised

Neospinothalamic tract	Paleospinothalamic tract
Transmits fast pain [Aδ fibres]	Transmits slow pain [C fibres]
2nd order neurons ascend, 1. some terminate at the reticular substance, 2. most fibres continue to VPL in the thalamus and then 3rd order neurons to the cortex [for *localisation*]	C fibres terminate in Substantia Gelatinosa [laminae II & III] and synapse with 2nd order neurons [many synapses]. Some fibres continue to the thalamus, most of them terminate in 1. Reticular nuclei [awakening someone], 2. Tectal are of midbrain or 3. Periaqueductal grey region

Analgesia system of the brain and spinal cord [review]

The brain has the ability to suppress pain through endogenous analgesia using endogenous opiates [enkephalins, dynorphin & endorphin].
Route ->
Pain fibres end in **periaqueductal grey matter or periventricular nuclei** stimulate neurons that secret enkephalins -> synapse with **Raphe magnus** or **perigigantocellularis** nucleus -> 2nd order neurons descend [*ipsilaterally*] to the spinal cord and synapse pre-synaptically with pain-transmitting fibres [these fibers secrete serotonin and they synapse with fibres that secret opiates which inhibt the transmission of pain impulses by *lowering the secretion of substance P*]

- **Pain and tactile fibers**

The gate control hypothesis -> stimulation of large Aβ fibres depress or supress the trnsmission of pain [Lateral inhibition of pain] through a tonically active inhibitory neuron.

- **Visceral pain**

Pain from an internal organ felt at a distant area on the skin that is *derived from the same segemnt as the organ* [in the embryo].
Visceral organs are not represented in the cortex but the skin is. And the viscera has very few sensory fibres so localised damage may result in little or no pain.
Referred pain -> the cerebral cortex interprets pain from an internal organ as if it were coming from a somatic area because the neuron the carries pain from that organ synapses with the 2nd order neuron that has the same connection with fibres coming from the somatic area where tha pain is felt.

Causes of Visceral Pain

1. **Ischemia**: Decrease or low blood flow to the tissue → tissue damage → releases chemicals that excite the free nerve endings of pain receptors
2. chemical irritation from perforated peptic ulcer, or acids from the biliary system → irritates the viscera and peritoneum
3. Spasm of a hollow viscus → constriction or contraction of SI (intestinal colic) → mechanical stimulation of the free nerve endings as they press on the free nerve endings
4. Overdistension of a hollow viscus → increase the pressure on the walls of the organs.

We're now going to move on to the second sensation transmitted through the anterolateral spinal pathway: *Thermal Sensations*

- **Receptors**: Cold or warm
  - There are more cold receptors than warm receptors in any area
  - The density of cold receptors varies throughout the body, but they have the same distribution as tactile receptors
    - **Highest on the lips and fingertips**
    - **Lowest on the trunk**

§ Freezing cold and burning hot are the same sensation because of stimulation of pain receptors → they stimulate pain.

Stimulation of Thermal Receptors

- **Cold receptors**:
  - Respond from 7 to 44°C with a peak response at 25°C.
- **Warm receptors:**
  - Respond from 30 to 49°C with a peak response at 44°C, which can cause pain.
- The relative degree of stimulation of the receptors determines **temperature sensation.** It depends on the combination of stimulation of the four receptors.
- There are four different receptors:
  1. warm
  2. cold
  3. burning hot
  4. freezing cold.
- Thermal receptors adapt to the stimulus but not completely.

Mechanism of Stimulation

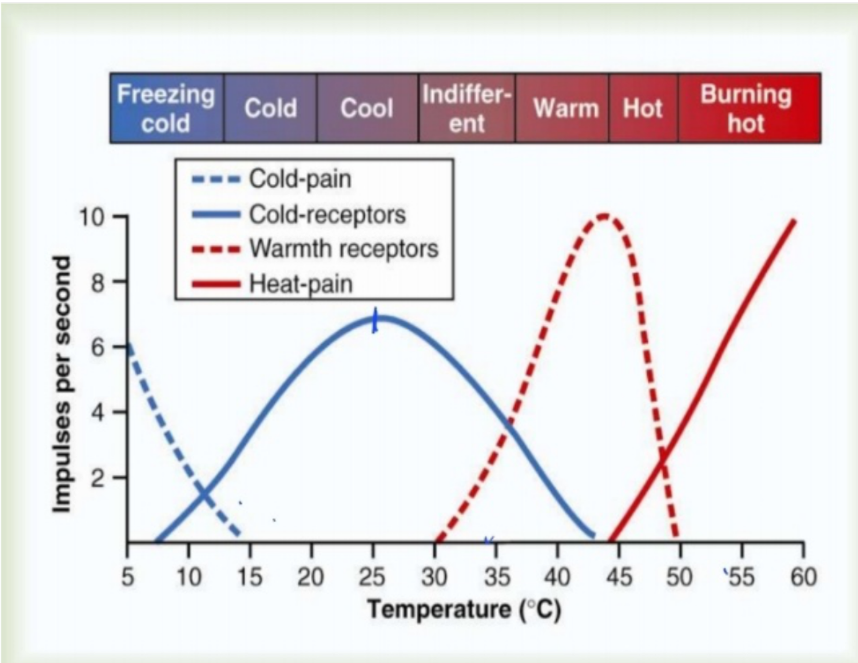
- Cold or warm temperature is thought to **change the metabolic rate of the receptor.**
  This changes the rate of **intracellular reactions**, by changing the **permeability of ions** in the receptors, changing the receptor potential and thus changing the **action potential** formed in the **afferent neuron connected to these receptors**.

- If you increase the temperature from 15 to 20 °C, the number of impulses increases to around the level of 25°C à The person feels too hot à After a while, there is a slight decrease in the number of impulses and the heat doesn't feel as intense or hot, reaching the level of feeling like it's 20°C.
  - Why does this happen? **Thermal adaptation** starts as fast adaptation then slow adaptation, but there is no complete adaptation to temperature.

- If you take a hot shower, in the beginning the water will feel too hot. Then the receptors adapt to the heat, making you withstand the heat. You then feel like the temperature of the water is somewhat lower than it had been when you first stood under the hot water.
- Same thing happens when you put your hand in cold water, it feels too cold at first but if you keep your hand in the cold water for a while the receptors adapt to the cold, but they do not adapt completely.

REMEMBER: Pain receptors, on the other hand ,, are non-adapting.

The figure further clarifies how we are able to discriminate between different temperatures. For example:



- A temperature of 32°C will stimulate the cold receptors to a certain extent, and it will stimulate heat receptors to a certain extent at a different rate.
- At a temp of 37°C, the rate of impulses differs for both warm and cold receptors.
  § At 52°C, the heat pain receptors are stimulated.
- Like we said, the relative degree of stimulation (combination of stimulation of the 4 different receptors mentioned above- Cold-pain, cold, warmth and heat pain receptors) determines the temperature sensation.

This is one of the theories revolving around how someone can discriminate different temperatures.

5 minutes break .. a new topic :)

Easing in:

you already know that we have general (pressure, touch, temperature, etc) and special (vision, audition, olfaction, etc.) types of sensation.

Receptors of special types of sensation are generally located in specific areas:

- vision—eyes (retina)
- hearing—ears
- taste—dorsal part of the tongue and pharynx
- smell—nose
- equilibrium—ears

Vision

Wandering down memory lane: a flashback to MSS

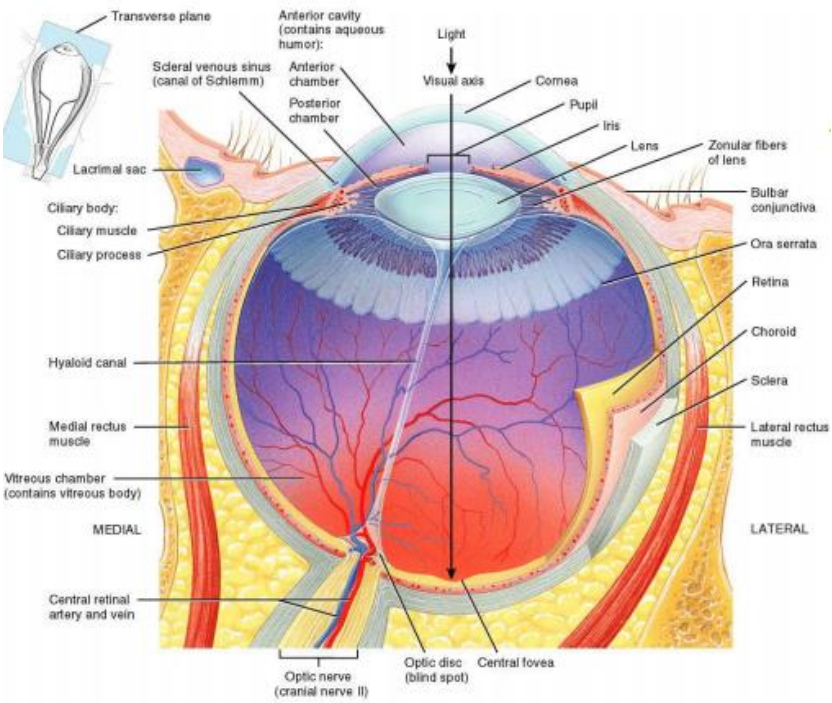
- We'll first start by going over the anatomy of the eye.

The eye consists of three layers:

- **the first and outermost layer is the sclera**, it surrounds 4/5 of the eyeball, as the last 1/5 is made up of **cornea** anteriorly.

› The sclera is made of very dense connective tissue.

› The cornea is a transparent, avascular part; its thickness is important to determine if one can undergo **laser surgery** or not since **it** involves changing of corneal thickness to correct the visual abnormality.



Interesting about the cornea: It's not exposed to the immune system! thus it doesn't have an antigenic activity, which facilitates corneal transplantation.

- **the middle layer is the choroid**, it's very vascular and continuous anteriorly with the **ciliary body**. A part of **it** is the ciliary muscle which continues anteriorly as the iris. And as you know the iris is the colored part of the eye and it has a hole called the **pupil**.

› Connecting the ciliary body and the lens' capsule are the suspensory ligaments, which through the work of the ciliary muscle change the shape of the lens.

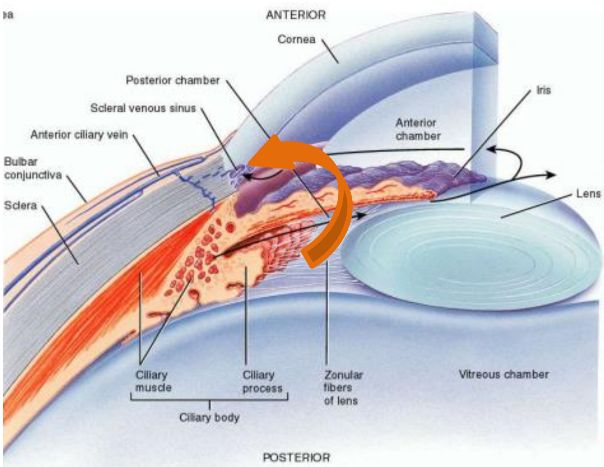
- The lens is made up of multiple layers of protein, and it's flexible; it has some fluidity that allows it to be pressed into a flatter shape (less convex) or into a fatter shape (more convex), depending on the capsule tightness around it.
- The lens also divides the eyeball into 2 cavities, one that lies between the lens and cornea called the **anterior cavity**, and one that extends behind to the retina called the **posterior cavity,** and the two are connected through the pupil**.**

- The anterior cavity: is further divided into 2 chambers, anterior to the iris, and posterior to the iris -between the lens and the iris-
- It contains a fluid called the aqueous humor which gives it great importance, since this fluid is the nutrition provider to the avascular cornea.

- The posterior cavity fluid is called the vitreous humor and it's derived from the embryonic mesenchyme.

Fluid System of the Eye

- The intraocular fluid keeps the eyeball round and distended.



As we mentioned we have 2 fluid chambers:

1. Aqueous humor which is a freely flowing fluid in front of the lens.

It's almost always in a constant dynamic amount, because it gets produced by the ciliary processes of the ciliary body at a rate of 2-3 microliters/min then it flows between the ligaments of the lens, through the pupil into the anterior chamber, goes between the cornea and the iris, through a meshwork of trabeculae to enter the canal of schlemm which empties into aqueous veins and then into extraocular veins.

2. Vitreous humor which is a gelatinous mass with little flow of fluid, derived from the embryonic mesenchyme (doesn't get renewed like the aqueous) lies behind the lens.

Intraocular Pressure

- Normally it is 15 mm Hg with a range of 12-20 mm Hg.
- The level of pressure is determined by the resistance to outflow of aqueous humor through the canal of Schlemm and the amount of its production.
- An increase in intraocular pressure caused by an increase in resistance to outflow of aqueous humor through the canal of schlemm can subsequently cause a form of damage to the optic nerve resulting in a condition called Glaucoma.
- And this can cause blindness due to compression of the axons of the optic nerve.

- **The inner most layer is the retina**, it's a very important layer as it's a neural layer that develops from ectoderm and contains the **visual receptors**, which can be divided into two types, rods and cones.
- In the central part of the retina (**macula lutea**), there's a slight depression called (**fovea**), which has the highest concentration of cones receptors that're important for sharp color vision and highest acuity of vision.
- The retina consists of six layers, only one of which contains light-sensitive photoreceptor cells (the outermost layer), and Light must pass through the overlying layers to reach them. However, in the central fovea, the inner layers of the retina are absent, so the light has an almost unrestricted passage to the cone cells. This meets its function as it is the point of sharpest vision.
- Another part of the retina is the **optic disc** (superomedial to the central fovea), where optic nerve fibers pass through along with retinal artery & veins. It is a blind spot (no rods and cones).

  Retinal vessels are the most superficial vessels in our body, and we can visualize them with ophthalmoscopy. We can examine them in cases like atherosclerosis.

- **The vitreous humor** is the pressing force that keeps the retina and choroid adherent together -it's important since the retina depends on the choroid for its nutrition-
- It may leak sometimes due to trauma, between the retina and choroid separating them, and causing what's known as **retinal detachment**.
- **Retinal detachment** causes the receptors on the retina to separate from the choroid which is what keeps them alive, and this separation eventually leads to neural death, ultimately **causing blindness**.
- Retinal detachment would actually be one of the very few ophthalmic emergencies. It has to be reattached in less than 48 hrs or the patient loses sight.

A visit to the physics world

Refractive index

- Speed of light is **greatest in air*** with a magnitude of nearly 300,000 km/sec. logically, light speed decreases when it passes through another transparent substance.

- When light travels **between two different media it refracts** (gets bended) due to this change of speed.

- **The refractive index** is the ratio of the speed of light in air to the speed of light in the substance.

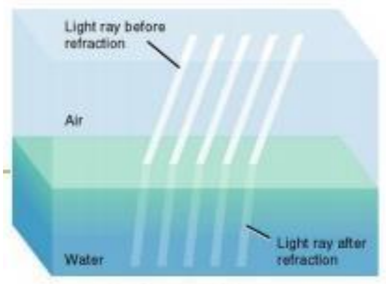
$$\frac{\text{speed of light in air } (300{,}000 Km/s)}{\text{speed of light in the substance}}$$

- **e.g.** speed of light in substance = 200,000 km/sec,
  R.I. = 300,000/200,000 = 1.5.

- Since the speed is fastest in air, this means the index would always be 1 or more

But what IS refraction?

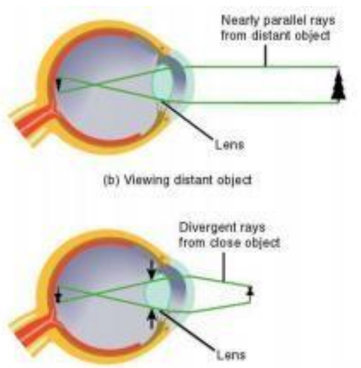
It's Bending of light rays through an angulated interface between two media with different refractive indices.



(a) Refraction of light rays

You can think of the refractive index as a mathematical representation of an intensive physical property of the substance (first year flashbacks 🟡), as it depends on its density. It means, 'how many times slower light will be in this substance compared to its speed in air' *for simplicity we said 'air', but the accurate calculations are compared to light's speed in 'space' or 'vacuum'

- The degree of refraction and the resulting degree of angulation of light increases as the difference in their R.I. increases.



(b) Viewing distant object
(c) Accommodation

- The features of the eye (like cornea, lens, humor) have different R.I. and thus they cause light rays to bend.
- This results in light rays eventually being focused on the retina whether the object is near ('point source') or far ('parallel lines'). *discussed soon