Visceral Sensation & Referred Pain

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About viscera doesn't have much free new enologys the About viscera is the only sonsation that comes from the viscera is the Viscera clossoft have touch or passive receptor

- Free nerve endings in viscera are less than that in skin. However, pleura and peritoneum are rich in pain receptors. So sensitive to pain
- liver parenchyma & lung alveoli are devoid of free nerve endings.

 Sensory cortex is poorly aware by the visceral pain.
- The stimuli which cause severe cutaneous pain may even not cause any visceral pain e.g., cutting the viscera with a knife or cauterization of cervical erosion is not painful. On the other hand, some stimuli which cause visceral pain like bacterial toxins may not cause any cutaneous Pain.

 You of the criters of the characteristics of the viscers: referred to a other site (15 orgin is in a specific place cutaneously)

Visceral pain

شخرا السلايد اللي بعره

Common between deep

deep and slow pain aren't localized also

- Dull aching, not well localized it clossed reach a specific area in the samable sentary cortex
- It is transmitted by afferent sympathetic or parasympathetic nerves and sometimes by somatic afferent (3)

Visceral pain is produced by:

- overdistension of hollow organs (stomach). or the bladder
- Spasm of intestine or ureters.
- Toxins or chemicals in contact with mucosa.
- Ischemia.

in Portant for blood vissels

Traction on peritoneum or mesentery by a big tumor.

I this will cause ischemia to the organ

Visceral pain is usually accompanied by nausea, vomiting, bradycardia and shows phenomenon of referred pain as Sensory cortex is poorly aware of the visceral pain

Visceral Pain will give a Perasympethetic reaction (just like the deep Pain)

معے جدا جدا X the thoracic line - s from the end of eso phagus and tracter till the beginning of the stomache * the pelvic line -sat the level of iliac crest at the hip bone (in the intestine it takes the parimet two thirds of the colon) the last level of the section is under the line Hany structure between the 2 lines the afferent is Sympathetic 4 any structure above them (like esophylus)-sparasymp 4 any structure below them Locatur genitation) - sparasympth * the gallbladder (found in the gallbladder Possa in the liver), the iscanion of the gall bladder will cause the irratation of the central part of the diaphragm (Since it's booked above the liver. afferent -sphrenic Nerve c 3,4,5 (Somatic) afficient Jupain 1 21 21 15 is visceral prin 11 X

Referred pain

- It is pain which is felt in a site other than the diseased one that it originate from.
- It is pain which is felt in a (cutaneous) site rather than the (visceral) diseased one that it originates from.

Examples:

1) Anginal pain
(cardiac ischemia)
Retrosernum, medial side of left arm with little finger, jaw or root of the neck.

2) Gall bladder pain
Right shoulder.

3) Appendicular pain
Umbilicus & epigastrium.

4) Uterine & labor pain
The back.

5) Renal pain
Testis & loin. and angent shoulder.

Surface of the head.

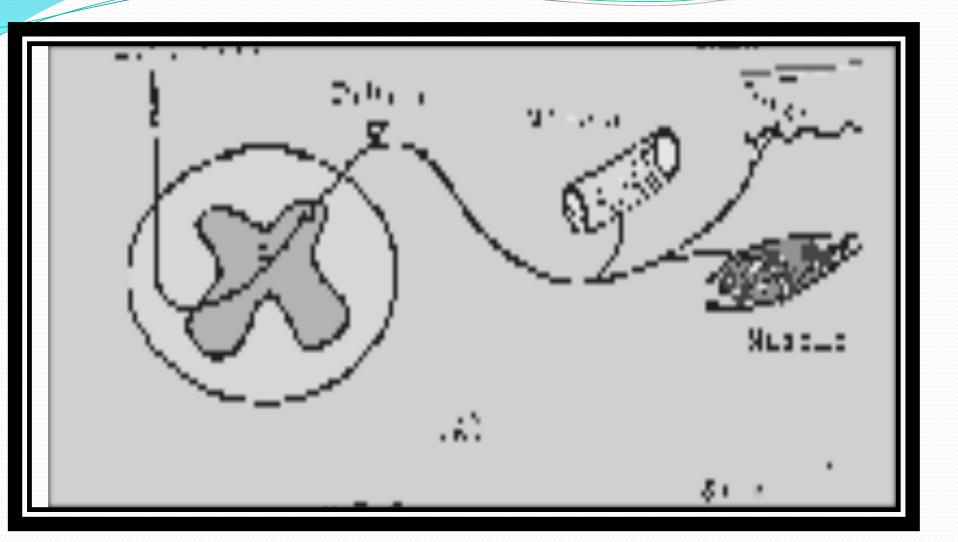
Mechanisms of referred pain

1. Branching dorsal root theory

- Pain from viscera enters the spinal cord in a certain dorsal root.
- Also pain from skin enters the spinal cord through the same dorsal root.

Because the **2 sites** have same embryological origin.

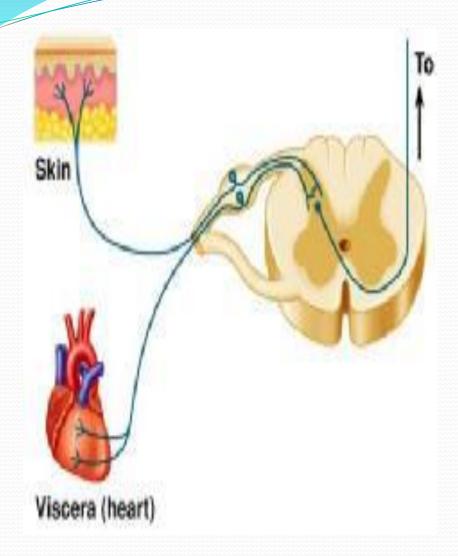
- The sensory cortex is adapted that if pain comes from this dorsal root it means that it comes from the skin not from viscera Because
- · a) Skin is usually exposed to trauma. Since it comes the extrior surface of the body
- b) Sensory cortex is poorly aware by the visceral pain.



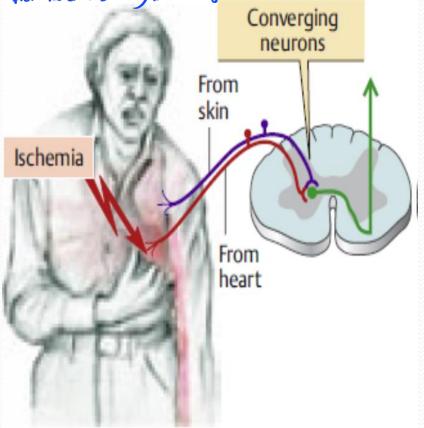
2 dorsal roots will go to the

ence -projection theory Similar to 2nd

- Pain from the viscera enters the spinal cord and converge on the dorsal horn cell.
- Sensations from certain area of the skin (that originate from the same embryonic segment as that viscera) enter spinal cord and converge on the same dorsal horn cell.
- Now pain sensation reach sensory cortex from this neuron, the cortex will project pain sensation as if it originate from the skin and not from the viscera because usually the skin is that organ which is always exposed to trauma and most pain that reach the cortex is coming from it.



If there was incheming in the heart the Pain Sentations are going to be transmitted by a classal horn Septerate from the classal horn that will transmit the Someation from the skin but they both will go to the Same classal cell



Pain control system 1- Analgesia system target slow Pain + (An = no & Algesia = pain) C fiber and substant P that is secreted (an = no & Algesia = pain) C fiber tenure tenurals of c libers

➤ It is a physiological system composed of group of neurons at different levels in CNS stimulate each other by chemical transmitters to minimize the pain.

This system is composed of: * He stimulation Keek on going until it centers the substance getatinose of colonali (which is the 2nd ocoder neuron of the slow Prin)

	Nucleus	Site	Transmitter released
1.	Peri ventricular nucleus	higher center of emotion there will be a colotest going to the HYDOTHA AMUS throtherms which will introduce the conditional function the analysistic system will then activate	β Endorphin will actions PAG
2.	Peri aqueductal gray matter (PAG)	Mid brain	Enkephalin Will activate Rmn
3.	Raphe magnus nucleus (RMN)	Mid line of upper medulla	Serotonin (5HT) Will activity PIC
4.	Pain inhibitory complex (PIC)	Dorsal horns of the spinal cord	Enkephalin
5.	Interneurons	Substantia Gelatinosa of Rolandi (SGR)	Enkephalin or GABA

Mechanism of analgesia cascade

Exposure to pain leads to:

- > Stimulation of peri ventricular nuclei of the hypothalamus release of β-endorphin.
- > Stimulation of the periaqueductal gray matter (PAG) \int release of **Enkephalin.**
- > Stimulation of raphe magnus nucleus (RMN) in medulla oblongata release of **Serotonin**.
- Stimulation of pain inhibitory complex (PIC) in posterior horns of the spinal cord.
- Stimulation of interneurons in SGR release of Enkephalin or GABA
- Enkephalin or GABA causes Closure of Ca++ channels of nerve terminals carrying pain sensations.

It is called **Presynaptic inhibition** which **P**revent release of substance **P** from nerve fibers that carry pain so, inhibit transmission of pain impulses (for PresynPatric to PastynPatric)

2- opiate system

It was discovered inside C.N.S and other many tissues in the body a certain type of receptors called <u>"opiate receptors"</u>, they are called so because they are **stimulated by opium and its derivatives.**

Also, inside the body it was discovered that **a group of chemical transmitters** can stimulate these **opiate receptors** and they are called <u>"Opioid peptides"</u> and they are widely distributed inside **C.N.S** and in **G.I.T**.

Combination of these <u>opioid peptides</u> with their <u>receptors</u> leads to marked <u>inhibition</u> of pain sensations by both pre and post synaptic inhibition.

The most important types of opioid peptides are: they stimulate coul other by caseades (locally)

- 1-Enkephalines 2-Endorphins as **B-Endorphins**
- 3 Dynorphins.
- -Opiate receptors are Mu-Delta -kappa Sigma $(\mu-\delta-\kappa-\theta)$.

Gate theory of pain inhibition

It is known that the **first gate** of pain sensation is the **S.G.R** in laminae II & III of dorsal horn cells. The pain impulses can be inhibited at this level before reaching the spinothalamic tract by many ways:

- A- By proprioceptive impulses that are carried by group "A" fibers from deep structures during rubbing the site of injury or inserting the specific needles of acupuncture.
- B- From the descending fibers that come from raphe magnus nucleus (RMN) in medulla oblongata (analgesic system) through releasing of Serotonin, these fibers cause inhibition to S.G.R through activating specific interneuron in the spinal cord to secrete GABA or Enkephalin causing presynaptic inhibition.
- C- Circulating opioids peptides like endorphins which are secreted from hypothalamus

