Visceral Sensation
&
Referred Pain

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About viscera

- Free nerve endings in viscera are less than that in skin. However, pleura and peritoneum are rich in pain receptors.
- 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.

Visceral pain

- Dull aching, not well localized
- It is transmitted by afferent sympathetic or parasympathetic nerves and sometimes by somatic afferent.

Visceral pain is produced by:

- overdistension of hollow organs (stomach).
- Spasm of intestine or ureters.
- Toxins or chemicals in contact with mucosa.
- Ischemia.
- Traction on peritoneum or mesentery by a big tumor.

- 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

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.
6) Headache	Surface of the head.

Mechanisms of referred pain

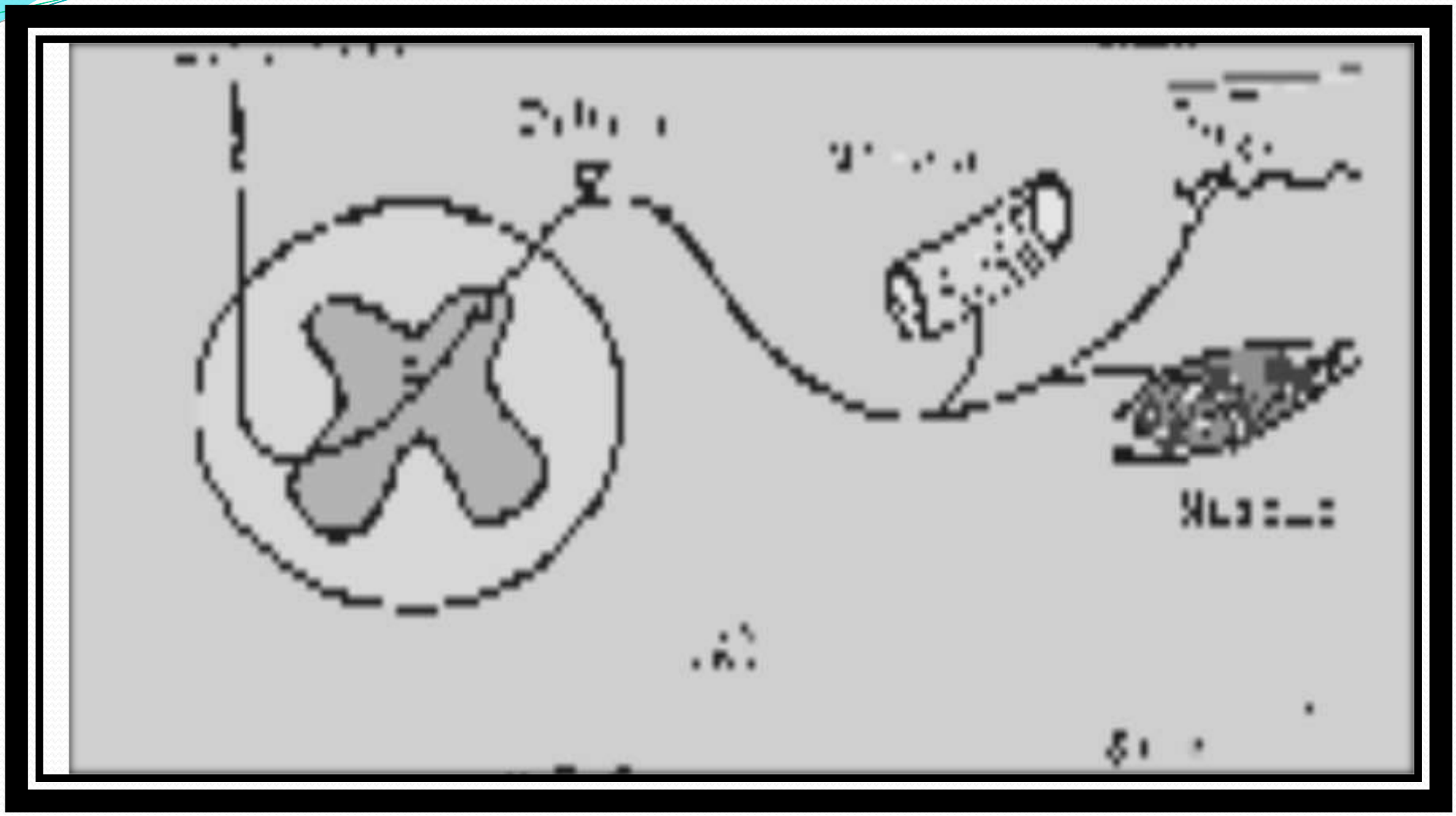
1. Branching dorsal root theory

- 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.

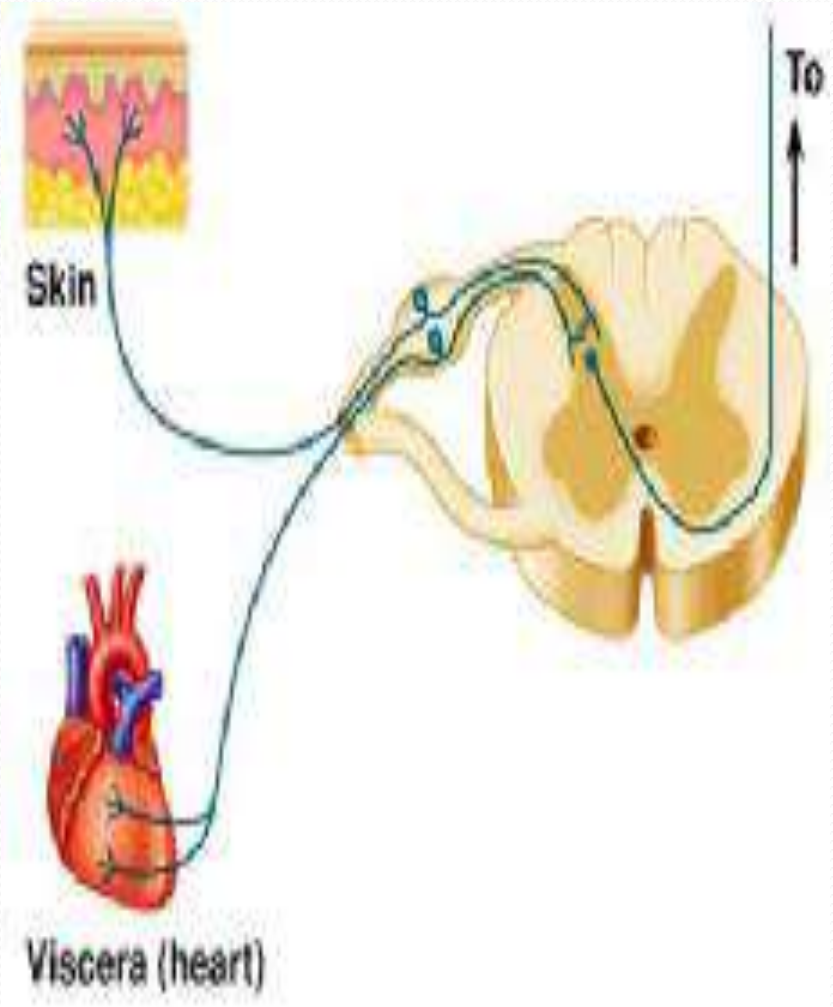
- The sensory cortex is adapted that if pain comes from this ***dorsal root*** it means that it comes from the skin not from viscera **Because**

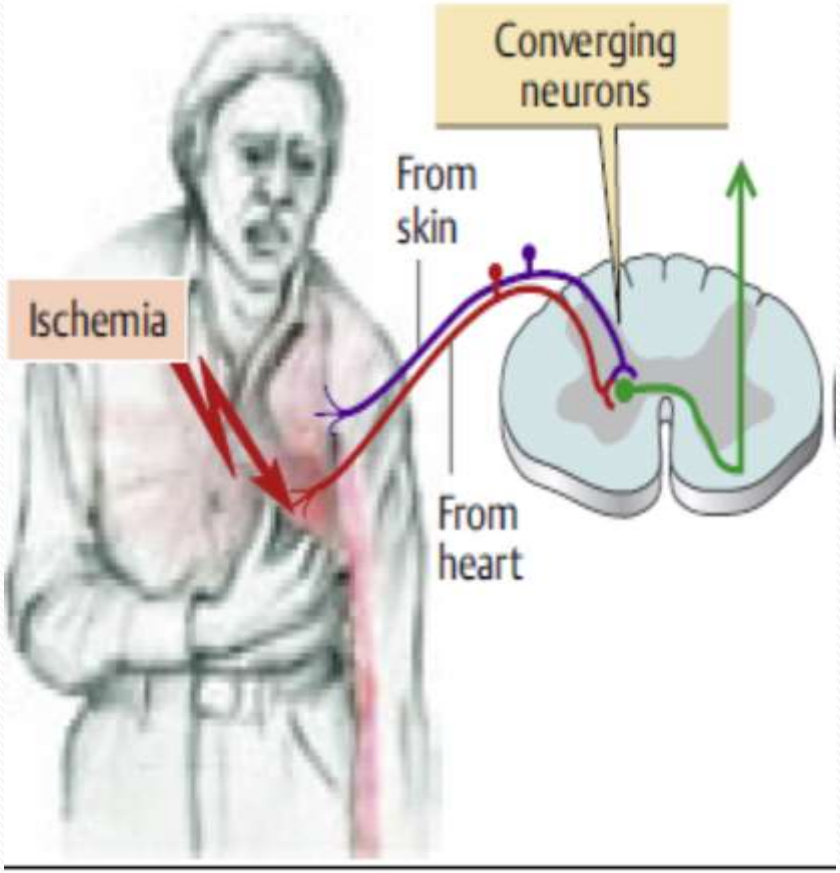
- a) Skin is usually exposed to trauma.
- b) Sensory cortex is poorly aware by the visceral pain.



2- Convergence -projection theory

- 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.


To
Skin
Viscera (heart)


Converging neurons
From skin
Ischemia
From heart

Pain control system
1- Analgesia system

- 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 :

Nucleus	Site	Transmitter released
1. Peri ventricular nucleus	Hypothalamus	β Endorphin
2. Peri aqueductal gray matter (PAG)	Mid brain	Enkephalin
3. Raphe magnus nucleus (RMN)	Mid line of upper medulla	Serotonin (5HT)
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) ➡ *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 **C**losure of **C**a++ 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.

2- opiate system

It was discovered inside C.N.S and other many tissues in the body a certain type of receptors called **"opiate receptors“ ,** 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 **"Opioid peptides"** and they are widely distributed inside **C.N.S** and in **G.I.T**.

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

The most important types of **opioid peptides** are:

1-Enkephalines 2-Endorphins as **B-Endorphins**

3 - Dynorphins.

-Opiate receptors are Mu- Delta -kappa – Sigma **(μ-δ-κ-θ).**

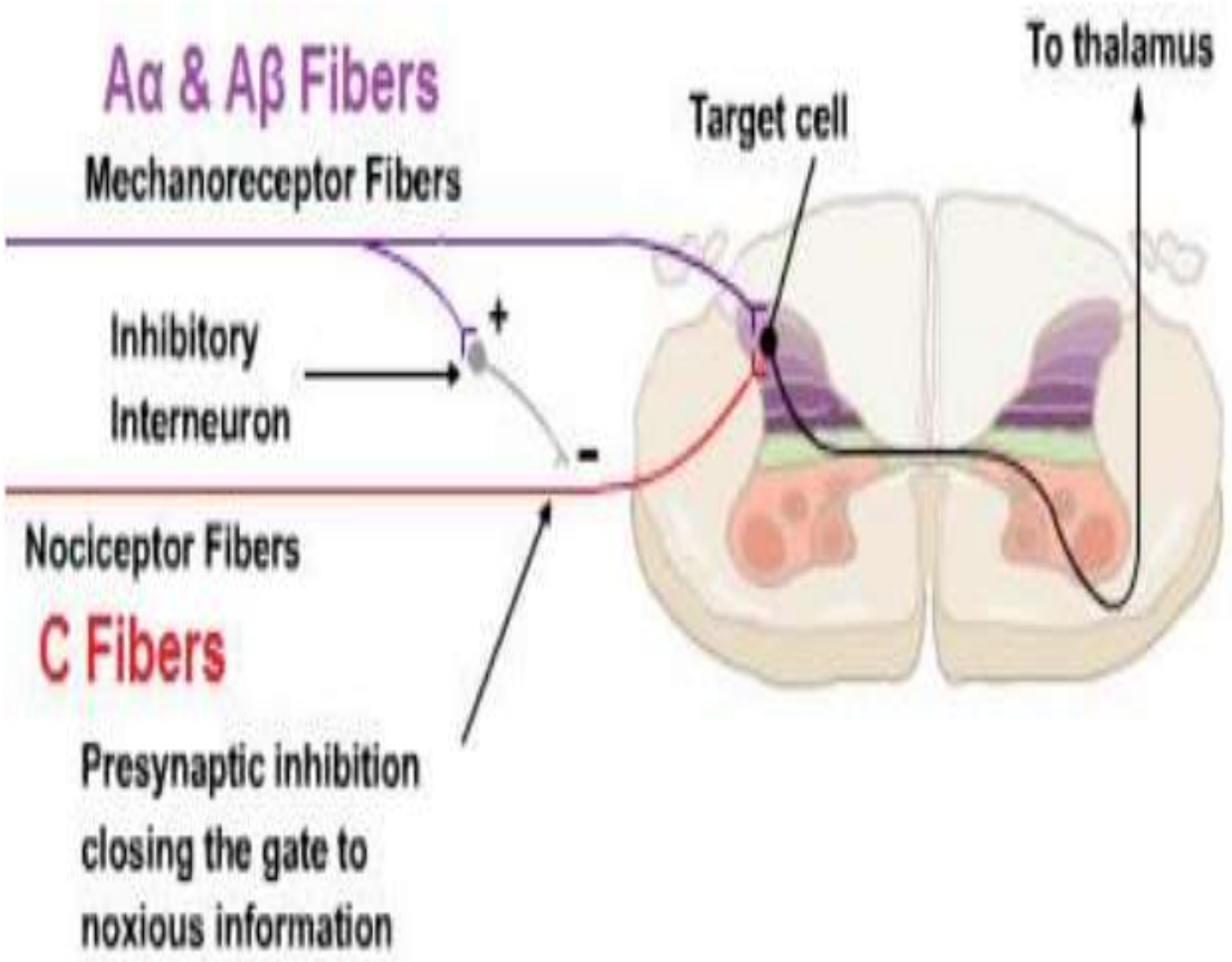
Gate theory of pain inhibition

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


Aα & Aβ Fibers
Mechanoreceptor Fibers
Target cell
To thalamus
Inhibitory
Interneuron
+
-
Nociceptor Fibers
C Fibers
Presynaptic inhibition
closing the gate to
noxious information

- Thank You