

Physiology of Pain

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Learning Objectives

- Pain receptors (nociceptors)
- Effects associated with pain sensation
- Mechanism of stimulation of pain receptors
- Qualities of pain
- Types of pain
 - •Somatic pain (superficial & deep pain).
 - •Visceral pain.
- Referred pain
- Pathway of pain
 - The neospinothalamic pathway
 - The paleospinothalamic pathway
- Role of cerebral cortex in pain perception



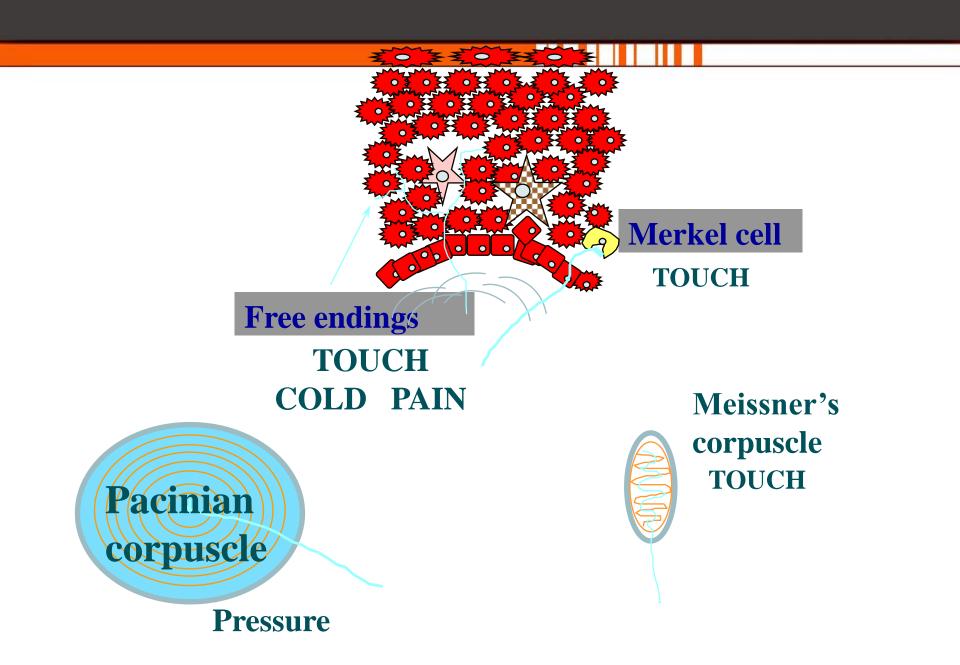
Pain is unpleasant sensation and emotional experience associated with actual or potential tissue damage.

Pain is characterized by the following:

OIt has a protective function.

 All pain receptors are free nerve endings of unmyelinated C fibers & small diameter myelinated Aδ fibers.

• Pain receptors are the most widely distributed.



- Pain sensation can be produced by various types of stimuli i.e. mechanical, thermal & chemical, hence the existence of mechanoceptors, thermoceptors, & polymodal pain receptors (nociceptors).
- Pain receptors adapt very little, if not at all.

- Localization of pain stimuli is less exact than that of other modalities.
- Pain receptors are high threshold receptors i.e. painful stimuli must be strong & noxious to produce tissue damage.
- Pain is perceived at both the cortical & thalamic levels.



1- Motor reactions

These may take the form of:-

* Reflexes e.g. withdrawal reflex.

* Muscle rigidity (stiffness).

2- Autonomic reactions

- * Mild pain stimulates post. hypothalamic $N \rightarrow$ sympathetic changes e.g. tachycardia.
- * Sever pain stimulates ant. hypothalamic $N \rightarrow$ parasympathetic changes e.g. bradycardia.
- 3- Emotional reactions as anxiety,

crying.....etc.

Mechanism of stimulation of pain receptors (nociceptors)

- Pain receptors are depolarized either directly or through the production of pain producing substances that are produced from damaged tissues as a result of inflammation (also called inflammatory mediators) e.g. bradykinin, serotonin, histamine, interleukins, substance P, K⁺, Ach, proteolytic enzymes.
- Prostaglandins & interleukins lower threshold of pain receptors.



I. <u>Fast pain</u> (immediate, first)

- It is also called pricking, acute, sharp or electric pain.
- It occurs mainly in skin by mechanical or thermal stimuli.
- It is transmitted via type Aδ fibers, conduction velocity 3-30m/s, account for 20% of nociceptors primary afferents, arise from all types of nociceptors.

- It appears very rapidly within 0.1 sec., and lasts for short time.
- It is usually well localized.
- The neurotransmitter is glutamate.
- e.g. The type of sensation felt when skin is cut with a knife.

II. <u>Slow pain</u> (delayed or second)

- It is also called **burning**, aching or chronic pain.
- It occurs in skin, deep tissues & viscera.
- It is transmitted via type C fibers, conduction velocity < 2m/s, account for 80% of nociceptors primary afferents, arise from polymodal nociceptors.

- It appears *slowly*, after one sec. or more, and lasts for longer duration.
- The neurotransmitter is substance P.
- It is diffused (poorly localized).



Pain can be classified according to the site of stimulation into:-

1. Somatic pain (superficial & deep pain).

2. Visceral pain.



- It arises from skin or other superficial structures.
- It occurs in 2 phase of *fast pricking* followed by *slow burning* pain.
- It can be well localized.
- It may be associated with motor, autonomic, emotional reactions.



- It originates from muscles, joints, periosteum, tendons & ligaments
- It is slow prolonged conducted by type C fibers.
- It is **diffuse** (i.e. poorly localized).

- It can initiate reflex contraction of nearby muscles.
- It may be *referred* to other sites.
- It is caused by: trauma, bone fracture & inflammation, arthritis, muscle spasm & ischemia.

Visceral pain

> There are few pain receptors in most viscera

Some viscera are pain insensitive e.g. liver parenchyma, lung alveoli, brain tissue, visceral layer of peritoneum, pleura and pericardium.

Characters of visceral pain

- It is *slow* pain conducted by *C fibers* (pain arising from parietal peritoneum, pleura and pericardium is sharp, pricking type).
- It is *diffuse*, the patient feels pain arising from inside but he cannot pinpoint it exactly.
- It is often associated with *autonomic reactions*.
- It can be associated with *rigidity* of nearby muscles.
- It may be *referred* to other sites.

Causes of visceral pain

To Distension of a hollow organs

Inflammation of an organ.

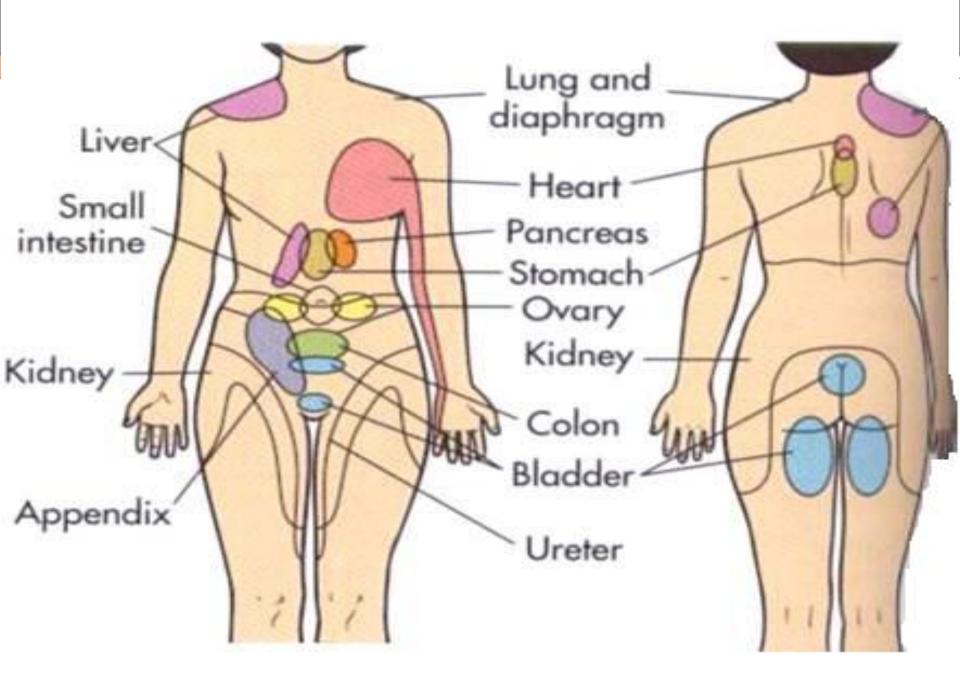
Ischemia e.g. pain due to myocardial ischemia.



- This is pain that is felt away from its original site.
- It is most frequent with visceral pain & deep somatic pain but cutaneous pain is not referred.
- Pain is referred according to dermatomal rule.

Examples of referred pain:

- *Cardiac pain* is referred to left shoulder & inner side of left arm.
- Pain of *appendicitis* is referred to umbilical region.
- Pain from the *ureter* is referred to testicular region.

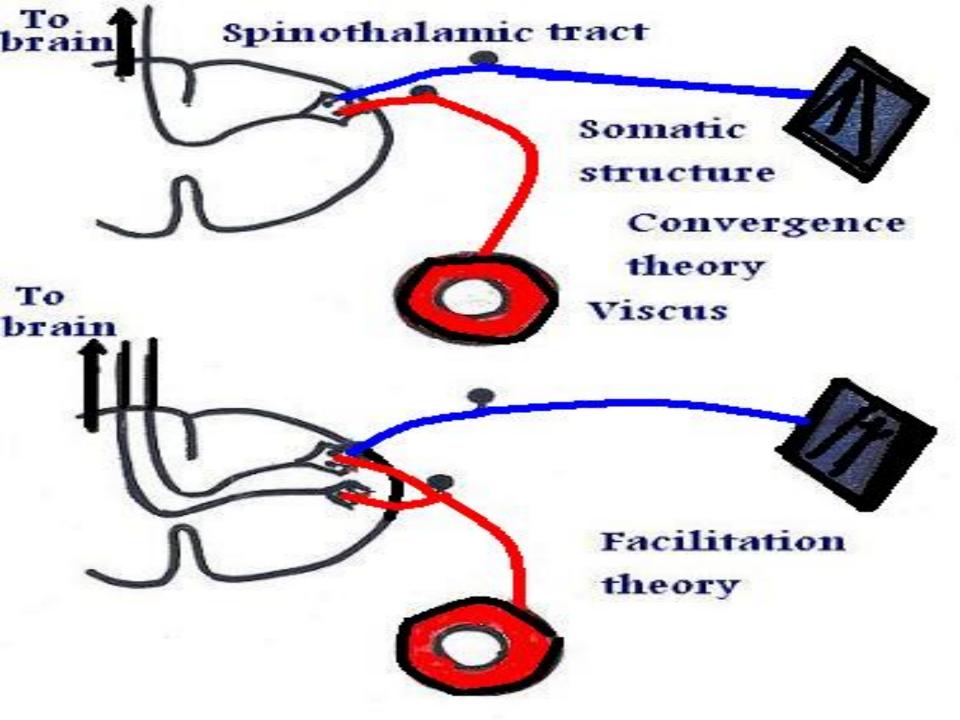


Organ	Site of referred pain
Meninges	Back of head &neck
Heart	Central chest, left arm
Diaphragm	Shoulder tip
Esophagus	Behind sternum
Stomach, duodenum	Epigastrium
Kidney	Loin
Ureter	Testicles
Trigone of bladder	Tip of penis
Hip	Knee
Appendix	Umbilicus
Uterus	Low back

Mechanism of referred pain

Convergence theory

- Afferent nerves from somatic structure & viscera that develop from same embryonic segment converge on same spinothalamic tract.
- Since brain is accustomed to receiving impulses from skin than viscera, so pain impulses carried to cortex along spinothalamic neurons shared by afferents from skin & other from diseased viscus are *misinterprited* by the brain as coming from skin.



Facilitation theory

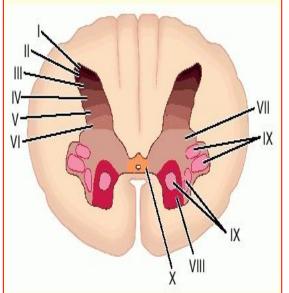
Pain fibers from skin are always carrying impulses, but they are not enough to produce pain. Impulses from diseased viscus pass through afferents which give collaterals to ST neurons receiving pain fibers from skin. As a result, ST neurons' excitability is raised (they are facilitated) to reach a threshold level. The signals reaching the brain are projected to skin area and pain is felt in skin dermatome

Pathway of Pain

Pain sensation is carried by **lateral spinothalamic tracts** which includes 2 separate pathways:-

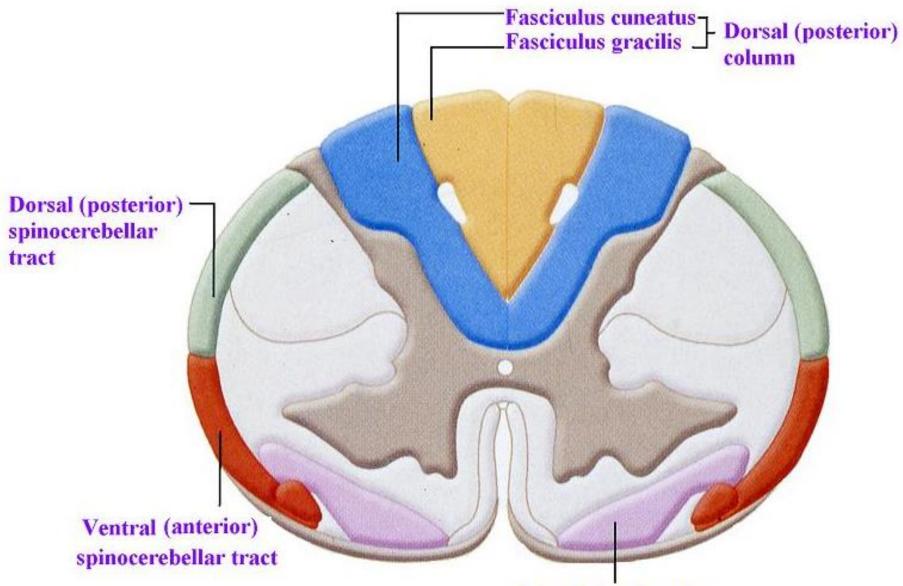
A) <u>The neospinothalamic pathway</u>:

This transmits fast pain & thermoceptive sensation.



○ *First order neurons*

Are mainly $A\delta$ afferent nerves. They ascend few segments in Lissauer' tract & terminate at lamina I & V of D. horn.



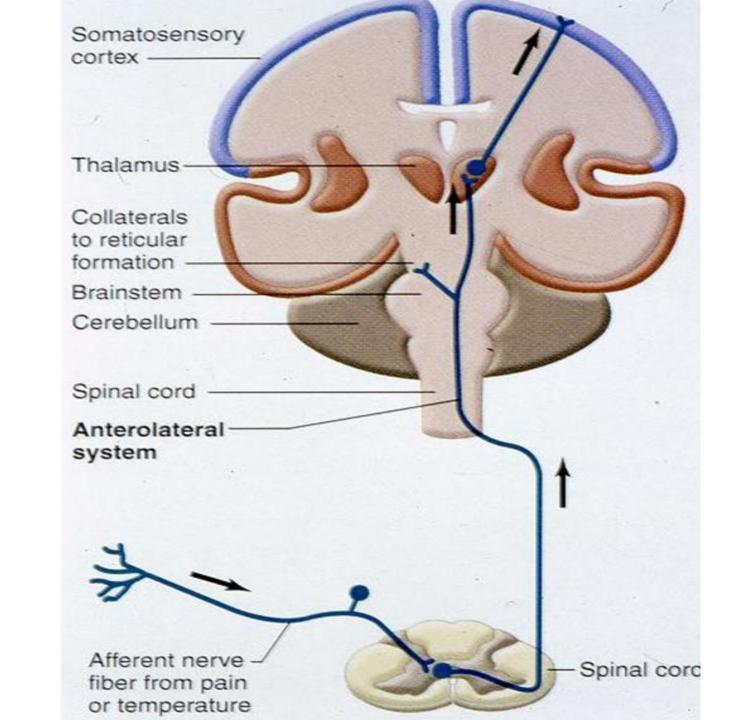
Anterolateral system

• Second order neurons

These constitute the tract. They start at dorsal horn, cross to opposite side and ascend in lateral column of spinal cord. The fibers ascend in brain stem to terminate in ventrobasal complex of thalamus.

• *Third order neurons*

These start at thalamus & project to somatosensory cortex.



B) <u>The paleospinothalamic pathway</u>:

This transmit slow pain sensation & thermoceptive sensation.

• First order neurons

They are mainly **type C fibers**. They enter spinal cord via dorsal roots, ascend a few segments in Lissauer' tract & terminate at substantia gelatinosa in laminae II & III of dorsal horn.

Second order neurons

They start at SGR, cross to opposite side in front of central canal, ascend in lateral column of SC & terminate at:-

- Reticular formation of brain stem.
- Intralaminar nuclei of thalamus.
- Hypothalamus & adjacent region of basal brain.

Impulses arriving these regions have strong arousal effects and can be perceived.

O*Third order neurons*

- These start at thalamus,
- Project to all parts of cerebral cortex.

Role of cerebral cortex in pain perception

- Full perception of pain occurs when signals enter RF of brain stem, thalamus & basal regions.
- Somatosensory cortex plays important role in topognosis i.e. localization & interpretation of pain quality.
- Fast pain is localized better than slow pain because signals carried in neospinothalamic tract reach somatosensory cortex, while a small propotion of paleospinothalamic pathway reach there.

