



**Physiology Team**



# Lecture : 3

## Physiology of Pain

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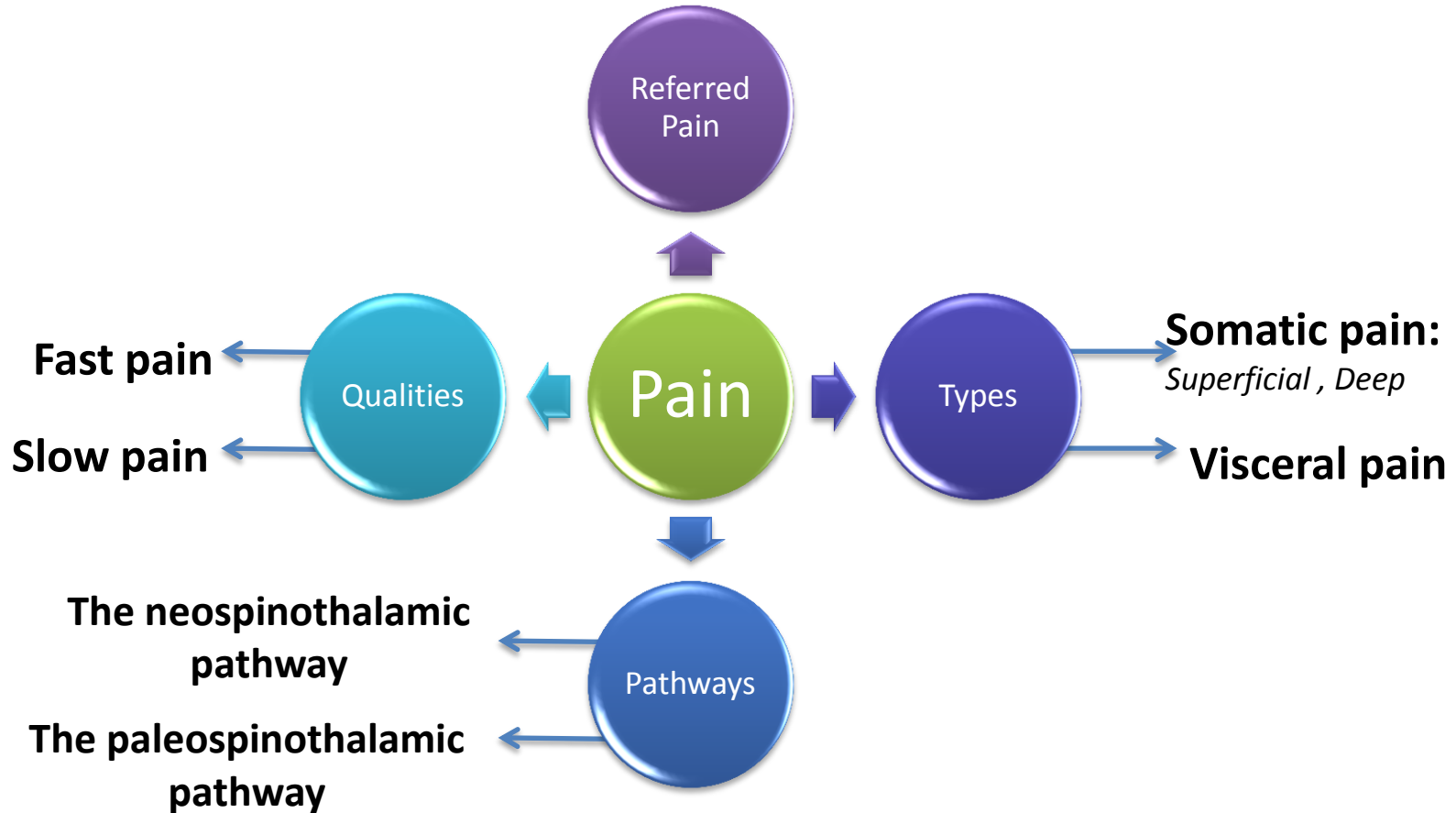
Thanks to Sara Al-haddab

# OBJECTIVES

**At the end of this lecture, student should be able to describe:**

- 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.

# MIND MAP



# Pain

**Pain** is unpleasant sensation and emotional experience associated with actual or potential tissue damage. Characterized by:

1. It has a protective function.
2. All pain receptors are free nerve endings of **unmyelinated C fibers** & small diameter **myelinated A delta ( $A^\delta$ ) fibers**.
3. Pain receptors are the **most widely distributed**.

## Classification of fibers:

- A: alpha, beta, gamma and delta.  
(High conduction velocity)
- B. (Low conduction velocity)
- C. (Low conduction velocity)

- Pain sensation can be produced by **various** types of stimuli i.e. mechanical, thermal & chemical, hence the existence of mechanoreceptors, thermoreceptors, & 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.

## Effects associated with pain sensation:

### **1- Motor reactions** (These may take the form of)

- Reflexes e.g. withdrawal reflex.
- Muscle rigidity (stiffness).

e.x: if the bone is broken , the muscles surrounding it will be stiffed

### **2- Autonomic reaction**

- Mild pain stimulates posterior hypothalamic N 'nucleus' → sympathetic changes e.g. tachycardia.
- Sever pain stimulates anterior hypothalamic N → 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<sup>+</sup>, Ach, proteolytic enzymes.
- Prostaglandins & interleukins lower threshold of pain receptors. تزيد من الاحساس بالألم

# Qualities of Pain

## *Fast pain (immediate, first)*

## *Slow pain (delayed or second)*

<b>Called</b>	Pricking, acute, sharp or electric pain	Burning, aching or chronic pain
<b>Occurs</b>	Skin	Skin, deep tissues & viscera
<b>Transmitted by</b>	Type A delta (A <sup>δ</sup> ) fibers	Type C fibers
<b>Conduction velocity</b>	3-30 m/s	< 2 m/s
<b>Percentage</b>	Account for 20% of nociceptors primary afferents	Account for 80% of nociceptors primary afferents
<b>Arise from</b>	All types of nociceptors	Polymodal nociceptors
<b>Appearance and duration</b>	It appears very <b>rapidly</b> within 0.1 sec., and lasts for <b>short time</b>	It appears <b>slowly</b> , after one sec. or more, and lasts for <b>longer duration</b>
<b>Localization</b>	Well localized	It is diffused ( <b>poorly localized</b> )
<b>Neurotransmitter</b>	Glutamate	Substance P.
<b>Example</b>	The type of sensation felt when skin is cut with a knife.	

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## Referred Pain:

- 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.

### Mechanism of referred pain:

#### **1- 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 misinterpreted by the brain as coming from skin.

#### **2- 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 “spinothalamic” 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.

## Examples of referred pain

Important!

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





# Types of Pain

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

Physiology Team		<b>1. Somatic Pain</b>		<b>2. Visceral Pain</b>
	<i>Superficial</i>	<i>Deep</i>	<i>Visceral</i>	
<i>Arises from</i>	Skin or other superficial structures.	Muscles, joints, periosteum, tendons & ligaments.	There are few pain receptors in most viscera. Some viscera are <b>pain insensitive</b> e.g. liver parenchyma, lung alveoli, brain tissue, visceral layer of peritoneum, pleura and pericardium.	
<i>Quality</i>	It occurs in <u>2 phase</u> of fast <b>pricking</b> followed by slow <b>burning</b> pain.	It is slow prolonged conducted by type <b>C fibers</b> .	It is slow pain conducted by <b>C fibers</b> . (pain arising from parietal peritoneum, pleura and pericardium is <u>sharp, pricking type</u> ).	
<i>Localization</i>	Well localized.	It is diffuse (poorly localized).	It is diffuse, the patient feels pain arising from inside but he cannot pinpoint it exactly.	
<i>Associations</i>	Associated with motor, autonomic, emotional reactions.	It can initiate <u>reflex</u> contraction of nearby muscles.	<ul style="list-style-type: none"> <li>• It is often associated with <b>autonomic reactions</b>.</li> <li>• It can be associated with rigidity of nearby muscles.</li> </ul>	
<i>Referred Pain</i>	<b>Cutaneous pain is <u>not</u> referred.</b>	It may be referred to other sites.	It may be referred to other sites.	
<i>Cause</i>	-----	It is caused by: trauma, bone fracture & inflammation, arthritis, muscle spasm & ischemia.	<ul style="list-style-type: none"> <li>• Distension of a hollow organs.</li> <li>• Inflammation of an organ.</li> <li>• Ischemia e.g. pain due to myocardial ischemia.</li> </ul>	

■ **Slides**

■ **Important**

■ **Doctor's Notes**

■ **Explanation**

■ **Boy's Slides**

# Pathway of Pain

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

	<i>The neospinothalamic pathway</i>	<i>The paleospinothalamic pathway</i>
	This transmits <b>fast pain</b> & thermoceptive sensation.	This transmit <b>slow pain</b> sensation & thermoceptive sensation.
<b>First order neurons</b>	Are mainly <b>A delta (A<sup>δ</sup>)</b> afferent nerves. They <u>ascend few segments</u> in Lissauer' tract & terminate at <b>lamina I &amp; V of dorsal horn</b> .	They are mainly type <b>C fibers</b> . They enter spinal cord via dorsal roots, <u>ascend a few segments</u> in Lissauer' tract & terminate at <b>substantia gelatinosa in laminae II &amp; III</b> of dorsal horn.
<b>Second order neurons</b>	These <u>constitute the tract</u> . They start at dorsal horn, <b>cross to opposite side</b> and ascend in lateral column of spinal cord. The fibers ascend in brain stem to terminate in <u>ventrobasal complex of thalamus</u> .	They start at <b>SGR</b> "substantia gelatinosa of rolando", <b>cross to opposite side</b> in front of central canal, ascend in lateral column of spinal cord & <u>terminate</u> at: <ul style="list-style-type: none"> <li>• <u>Reticular formation of brain stem.</u></li> <li>• <u>Intralaminar nuclei of thalamus.</u></li> <li>• <u>Hypothalamus &amp; adjacent region of basal brain.</u></li> </ul> Impulses arriving these regions have <b>strong</b> arousal effects and can be <b>perceived</b> .
<b>Third order neurons</b>	These start at thalamus & project to <b>somatosensory cortex</b> .	These start at thalamus, project to <b>all parts of cerebral cortex</b> .

# Role of cerebral cortex in pain perception

- ✧ Full perception of pain occurs when signals enter RF “Reticular Formation” 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.

# SUMMARY

- Pain is unpleasant sensation and emotional experience associated with actual or potential tissue damage.
- All pain receptors are free nerve endings of unmyelinated C fibers & small diameter myelinated A delta ( $A^{\delta}$ ) fibers.
- Effects associated with pain sensation:
  - 1- Motor reactions.
  - 2- Autonomic reactions.
  - 3- Emotional reactions.
- Qualities of pain:
  - 1- Fast pain.
  - 2- Slow pain.
- Referred pain is pain that is felt away from its original site.
- Mechanism of referred pain:
  - 1- Convergence theory.
  - 2- Facilitation theory.
- Types of pain:
  - Somatic pain (superficial & deep pain).
  - Visceral pain.
- Fast pain is localized better than slow pain .

# QUESTIONS

**1. Which pain is not referred:**

- a. Visceral.
- b. Deep.
- c. Superficial.

**2. The neurotransmitter for Fast pain is:**

- a. Glutamate.
- b. Substance P.

**3. The Appendix pain is referred to:**

- a. Testicles.
- b. Knee.
- c. Umbilicus

1. C  
2. A  
3. C

**THE END**

**If there are any Problems or Suggestions,  
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**THANK YOU**