

PAIN

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PAIN

- It is mainly a protective mechanism of the body, as it is not a pure sensation but a response to tissue injury.
- Response may be
 - Motor – e.g. withdrawal
 - Emotional – e.g. anxiety, crying, depression
 - Autonomic reaction e.g. tachycardia, rise in B.P., sweating,

Classification of pain

- FAST OR SLOW
- SUPERFICIAL OR DEEP
- VISCERAL
- REFERRED

Classification of pain

1. Fast pain
 - It is felt within 0.1 sec. after stimulation.
 - e.g. pricking, cut with knife.
2. slow pain
 - Felt in 1 sec. or more following painful stimulus.
 - It is associated with tissue damage & can be referred to as burning pain, aching pain or chronic pain

▲ TABLE 6-2
Characteristics of Pain

<i>Fast Pain</i>	<i>Slow Pain</i>
Occurs upon stimulation of mechanical and thermal nociceptors	Occurs upon stimulation of polymodal nociceptors
Carried by small myelinated A-delta fibers	Carried by small unmyelinated C fibers
Produces sharp, prickling sensation	Produces dull, aching, burning sensation
Easily localized	Poorly localized
Occurs first	Occurs second; persists for longer time; more unpleasant

Pain receptors

- Free nerve endings (Nociceptors)
- Pain receptors do not adapt at all or very slowly.
- They are found in largest no. & density in skin, periosteum joint surface, arterial wall & duramater.
- pain receptors are activated by 3 types of stimuli;
 1. Mechanical – they elicit fast pain.
 2. Thermal - they elicit also fast pain.
 3. Chemical - they produce slow pain.

Chemical agents that produce pain

- Bradykinin, serotonin, Histamin, K^+ ion, Acids, acetyl choline, & proteolytic enzymes.
- Prostaglandins & substance – P enhance the sensitivity of pain receptors.

Pain Pathways

FAST PAIN

- Transmitted by $A\delta$ (delta) fibers in the peripheral nerves
- Characteristics of $A\delta$ fibers
 - Myelinated -
 - Diameter fine 2 - 5 μm
 - 12 - 30 m/sec. conduction velocity
 - Terminated at I and V layer
 - Fast pain, rapid, pricking and well localized
 - Neurotransmitter - Glutamate
 - 20% pain conduction

Pain Pathways

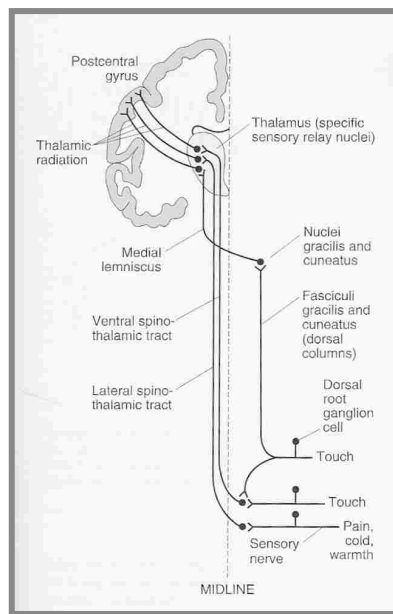
- **SLOW PAIN**
- Chronic type of pain, transmitted by C fibers

Characteristics of C fibers

- Non-Myelinated
- Diameter 0.4 – 1.2 μm
- conduction velocity 0.5 - 2 m/s
- Terminate in layer II and III of dorsal horn (substantia gelatinosa)
- Slow, diffuse, dull, aching
- Neurotransmitter - P-Substance
- 80% of pain conduction

Pain Pathways

- Axons from dorsal root ganglia of a particular spinal segment enter the dorsal horn & end there. The second order neuron start here.
- Cross to the opposite side & ascend in white matter as lateral spinothalamic tract.
- Most of them project to thalamus VPL (ventroposterolateral) nucleus.
- Then thalamic neuron project to S-I (primary somatosensory cortex)



Pain Pathways

- Most of the slow pain fibers project to reticular formation & then proceed to thalamus (posterior nuclei) .
- Reticular system project to all parts of brain but specially to cerebral cortex therefore they cause arousal from sleep.

Dual Pathways for Transmission of Pain Signals into the Central Nervous System

Neospinothalamic Tract
Paleospinothalamic Tract

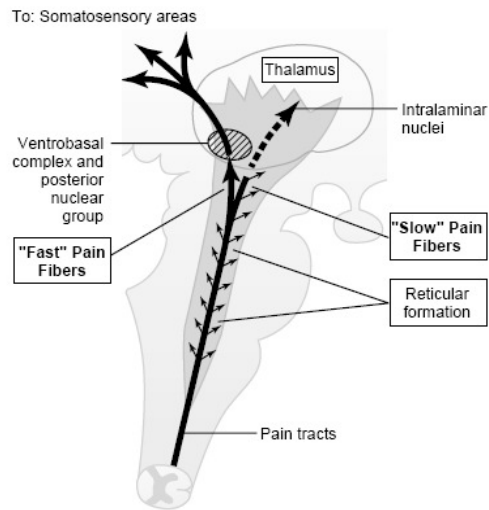


Figure 48-3

Transmission of pain signals into the brain stem, thalamus, and cerebral cortex by way of the *fast pricking pain pathway* and the *slow burning pain pathway*.

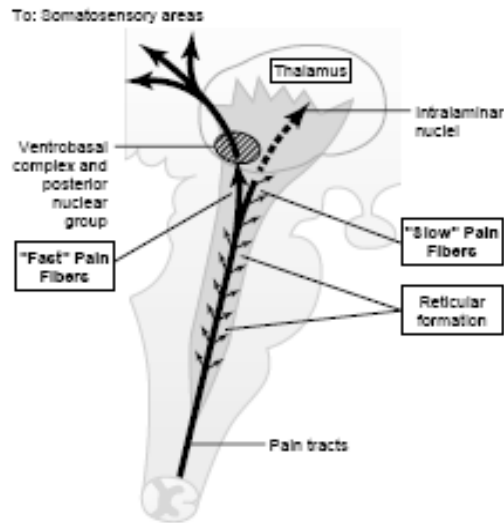


Figure 48-3

Transmission of pain signals into the brain stem, thalamus, and cerebral cortex by way of the *fast pricking pain pathway* and the *slow burning pain pathway*.

Applied

1. What will happen if sensory area SI is removed.

Ans. persons ability to interpret the quality of pain & precise location of pain will be affected.

2. Why patient with chronic pain syndrome have difficulty in sleeping?

Ans. Paleospinothalamic pathway as it sends information to reticular formation and thalamic nuclei which are part of brain activating / alerting system, therefore it may explain why chronic pain syndrome causes difficulty in sleep.

REFERRED PAIN

Branches of visceral pain fibers synapse in the spinal cord on the same second-order neurons (1 and 2) that receive pain signals from the skin

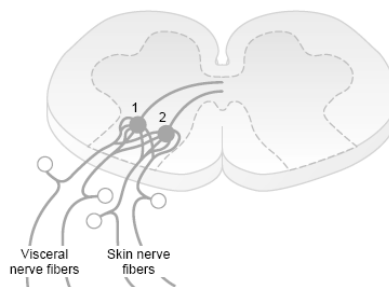


Figure 48-5

Mechanism of referred pain and referred hyperalgesia.

REFERRED PAIN

**Localization of Visceral Pain—
“Visceral” and the
“Parietal” Pain
Transmission
Pathways**

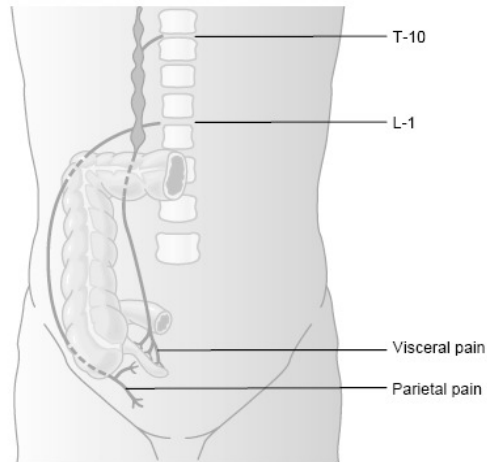


Figure 48-7

Visceral and parietal transmission of pain signals from the appendix.

REFERRED PAIN

When visceral pain is referred to the surface of the body, the person generally localizes it in the dermatomal segment from which the visceral organ originated in the embryo, not necessarily where the visceral organ now lies.

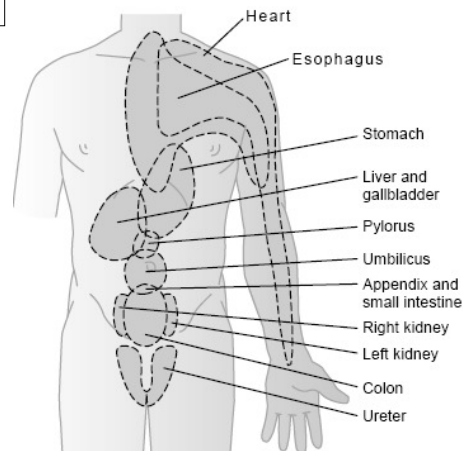


Figure 48-6

Surface areas of referred pain from different visceral organs.

Visceral Pain

- It is produced by stimulation of pain receptors in viscera.
- Pain receptors in viscera are sparsely distributed.
- Afferent from viscera reach CNS via sympathetic & parasympathetic pathway.
- Visceral travel along the same pathway as somatic sensation i.e. Spinothalamic tract.

CHARACTERISTICS OF VISCERAL PAIN

- Poorly localized
- Associated with nausea and autonomic disturbance
- Often referred to another part of the body
- Cutting, crushing are not painful when applied to viscera
- Pain is caused by distension, ischemia and inflammation

Referred Pain

- Pain that is not felt in the diseased structure itself, but at another place in the body far away from the site of origin.
- Visceral and deep somatic pain are often referred, but superficial pain is not.
- Mechanism of referred pain
 - Dermatomal rule
 - Convergence of peripheral & visceral pain on the same second order neuron that project to brain

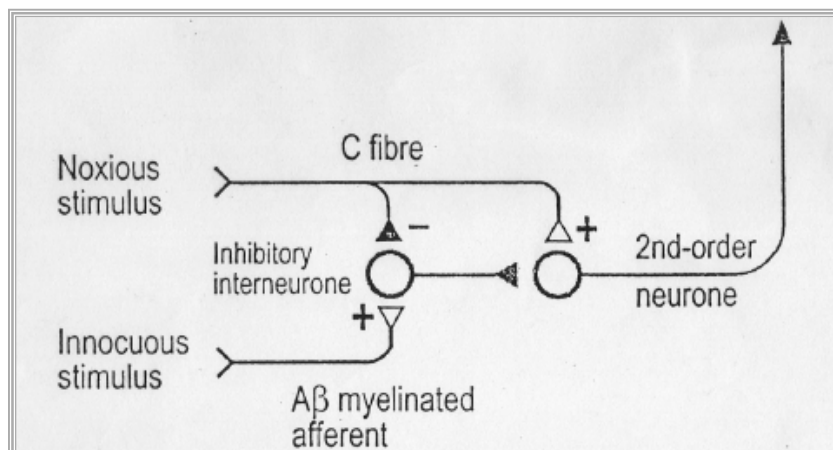
IMPORTANT EXAMPLES OF REFERRED PAIN

<u>ORGAN</u>	<u>SITE OF ACTION</u>
• HEART	• PRECORDIUM, INNER ASPECT OF LEFT ARM, EPIGASTRIUM
• APPENDIX	• UMBILICUS
• SMALL INTESTINE	• UMBILICUS
• PLEURA	• ABDOMEN
• TONGUE	• EAR
• TEETH	• HEAD
• UTERUS	• LOW BACK RADIATING TO LOWER ABDOMEN

GATE CONTROL THEORY OF PAIN

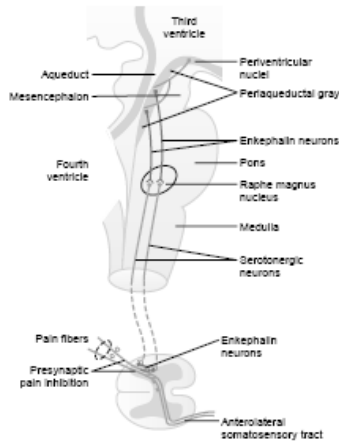
- The dorsal horn of spinal cord , form the gate through which pain impulses must pass in order to reach the brain.
- Impulses coming along the C fibers cause the release of substance P & open the “gate” while impulses coming along the large diameter A β fibers close the “gate”.
- The “gate” is also under control of higher centers by means of analgesic system of corticospinal & reticulospinal fibers.

GATE CONTROL THEORY OF PAIN



ENDOGENOUS ANALGESIC SYSTEM

- Enkephalin secreting neurons



Pain Suppression (“Analgesia”) System in the Brain and Spinal Cord

**Brain’s Opiate System
Endorphins
and Enkephalins**

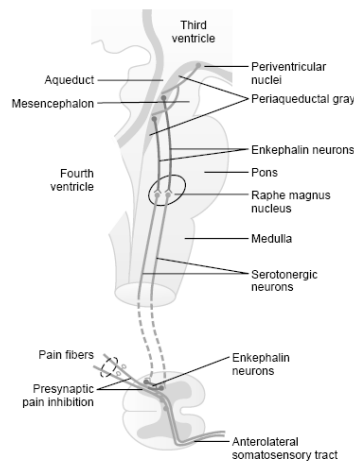


Figure 48-4

Analgesia system of the brain and spinal cord, showing (1) inhibition of incoming pain signals at the cord level and (2) presence of enkephalin-secreting neurons that suppress pain signals in both the cord and the brain stem.

FACIAL PAIN

- Sensation from face & head are carried by trigeminal nerve.
- Fibers of trigeminal nerve join the anterolateral system in the brainstem.

TERMS FREQUENTLY USED

- **Hyperalgesia: Excessive Pain**
- **Allodynia: Pain caused by any other sensation**
e.g. touch will cause pain.
- **Muscular Pain: Less blood flow in the muscles (ischemia).**
- **Stress analgesia: Mild degree of pain is not felt if the other part of the body has excessive pain.**
- **Causalgia: Burning pain.**

TERMS FREQUENTLY USED

Thalamic Syndrome

.Obstruction of the thalamogeniculate branch of the posterior cerebral artery Affects posterior thalamic nuclei.

. Prolonged severe pain.