

Pain management

→ Pain is an unpleasant sensory & emotional experience associated with actual or potential tissue damage. or prescribed in terms of such damage.

Etiology: Etiology of Pain may not always be identifiable.

- 1) Musculoskeleton causes. eg: osteoarthritis, R.A.
- 2) Neurologic causes.
- 3) Causes of Headache.

Pathophysiology of Pain

→ A complex array of neural networks in the Brain that are acted on by afferent stimuli to produce the experience we know as pain.

- 1) Nociceptive pain.
- 2) Pathophysiologic Pain.
Neuropathic & Functional Pain.

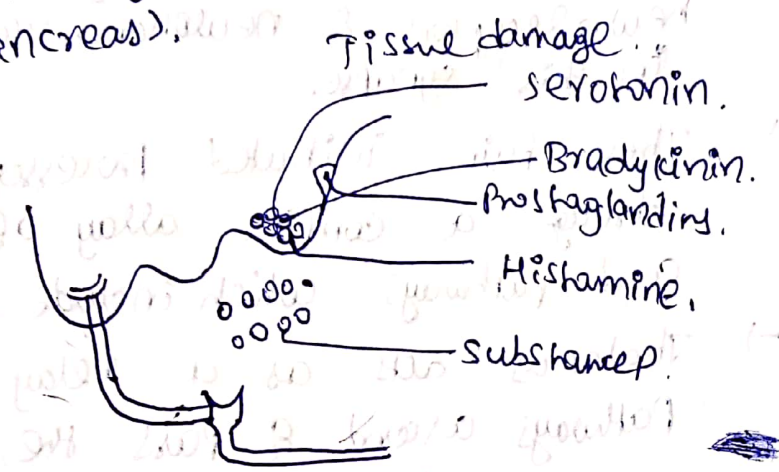
→ Nociception in terms of

- 1) Transduction
- 2) Transmission
- 3) Perception
- 4) Modulation.

Nociceptive pain is considered as protective & the physiologic. typically classified as either flom.

Somatic (arising from skin, bone, joint, muscle) ②
 Visceral (Internal organs such as large intestine (I) Pancreas).

1) Transduction:



Stimulation of free nerve endings (Nociceptors).

Found in both somatic & visceral structures

Nociceptors activated & sensitized by mechanical, thermal & chemical impulses.

activation of Bradykinin, Nerve growth substance P, serotonin.

leads to action potential (change in electrical potential associated with the passage of an impulse along the membrane of a muscle)

Transmitted through (I) along afferent nerve fibres to the spinal cord.

2) Transmission:

Nociceptive transmission takes place in the A-δ & C-afferent nerve fibres.

send impulses very fast

myelinated A-δ fibres. Evokes sharp localized pain.

unmyelinated fibres - aching, poorly localized pain.

Afferent nociceptive pain fibres synapse in various layers of the spinal cord, dorsal horn, releasing a variety of neurotransmitters including, glutamate, Aspartate & Substance P.

②
→ Complex array of events that influence pain can be explained in part by the interactions b/w. neuroreceptors & neurotransmitters that take place in this synapse.

→ These pain initiated processes reach the brain through a complex array of ascending spinal cord pathways. which include spinothalamic tract.

→ Thalamus acts as a relay station as these pathways ascend & pass the impulses to central structures where pain can be processed further.

Perception:

At this point in transmission pain is thought to become a conscious experience that take place in higher cortical structures.

Cognitive & behavioural functions can modify pain.

↓
Relaxation, distraction, meditation & guided mental imagery may strongly influence pain perception.

→ In contrast change in our neurobiochemical make up that results in states such as depression or anxiety may worsen pain.

Modulation: Body modulates pain through a number of complex processes.

Endogenous opiate system: consists of neurotransmitters.

Eg: Enkephalins, dynorphins & β -endorphins.

Receptors Eg: (M, S, ϵ , κ) found throughout the CNS & peripheral nervous system.

④ Enkephalins - Bind to opiate receptors & Release controlled levels of pain.

→ Regulating nociception in the body.

Dynorphins - members of opioid peptide family & Predominantly (preferentially) bind to kappa receptors.

→ ↑ sed levels - Block the release of glutamate.

Blocking - Enhance dopamine signaling.

→ transmits electrical signals within the nervous system ↓ reduce of depressive symptoms associated w stress

β-Endorphins - Utilized in the body to reduce stress & maintain homeostasis.

→ interact with the opiate receptors in the brain to reduce our perception of pain & act similarly to drugs such as morphine & codeine.

→ activation of the opiate receptors by the body's endorphins does not lead to addiction & dependence.

→ Stress & Pain are the two most common factors leading to the release of endorphins.

→ CNS also contains a highly organised descending system for the control of pain transmission.

Important neurotransmitters here include.

→ Endogenous opioids.

→ Serotonin [Regulate anxiety, happiness & mood in brain] → Bowel movements in GI tract.

→ Norepinephrine [acts as neurotransmitter & hormone] → Emotions, sleeping, dreaming, learning

↳ from the sympathetic nervous system.

→ γ-GABA - inhibitory neurotransmitter.

→ Released as a hormone into the blood, where it causes blood vessels to contract & heart rate to increase.

→ when GABA release it suppresses the activity of the neurons, motor control, vision & many cortical functions.

Inhibitory Neurotransmitters

→ GABA (major)

Glycine

Excitatory neurotransmitters

L-glutamate (major)

Serotonin

Epinephrine

Norepinephrine

~~Acetylcholine~~

Nitric oxide

Dopamine acts as a both inhibitory & excitatory neurotransmitter

Acetylcholine same as dopamine can be both excitatory & inhibitory neurotransmitter.

Adaptive Inflammation:

→ Inflammation pain can be thought of as the body's shifting from preventing tissue damage to the promotion of healing. (Eg. Surgical wounds, traumatic injury)

→ Inflammatory pain is also associated with an increase in the excitability or responsiveness of neurons within the CNS.

Neuropathic & Functional Pain:

→ Disengaged from noxious stimuli & healing & often is described in terms of chronic pain.

→ Neuropathic pain - result of nerve damage

Eg: diabetic neuropathy

Functional pain - abnormal operation of the nervous system

Eg: fibromyalgia, irritable bowel syndrome

→ mechanism responsible for neuropathic & functional pain may be the nervous systems endogenous dynamic nature.

→ Nerve damage (&) certain disease states may evoke changes seen in inflammatory pain.

→ Ectopic excitability (in an abnormal place (&) position).

→ Enhanced sensory transmission

→ nerve structure reorganisation.

→ loss of modulatory pain inhibition.

Classification of Pain:

1) Acute Pain:

Acute pain can be a useful physiologic process.

→ Acute pain is usually nociceptive in nature with common causes including acute illness, trauma, labour & medical procedure.

2) Chronic Pain:

→ Pain persists for months (&) years leading to a chronic pain.

→ This type of pain can be nociceptive / neuropathic / functional / mixed.

→ Chronic pain can be classified either being associated with cancer or from non cancer etiologies.

3) Cancer pain: Pain associated with life threatening condition after called malignant pain (&) simply cancer pain. This is caused by the disease itself treatment (&) diagnostic procedure.

Characteristics of Acute & Chronic Pain:

(8)

Characteristics	Acute Pain	Chronic Pain
1) Relief of Pain	Highly desirable	Highly desirable
2) Dependence & tolerance of medication	unusual	Common.
3) Psychological component	usually not present	After a major problem.
4) Environmental/family issues	Small	Significant.
5) Insomnia.	unusual	Common.
6) Depression	uncommon.	Common.

Clinical Presentation:

Signs & symptoms

acute pain	Chronic pain
Obvious distress.	Anger
Tingling	fear.
Anxiety	Hypotension.
depression	Tachycardia
Fatigue.	Pallor.
Radiating pain.	
	Hypertension
	Tachycardia
	Shock
	Pallor
	Tingling.

Laboratory test:

- > Pain is always subjective.
- > There is no specific lab test for pain.
- > Based on pt-history and patient description.

management of Pain:

⑧

Non Pharmacological:

- Application of heat (&) cold, massage & Exercise.
- Cognitive & Behaviour and social aspects of Pain are well established.

Pharmacological:

Non opioid agents: