Question: **Define Pain, Describe the theories of pain, Explain the applications of Transcutaneous electrical nerve stimulation, Add a note on mechanism of pain relief.**

Define Pain

Pain is an unpleasant sensory and emotional experience associated with actual or threatened tissue damage.

Describe the theories of pain

Theoretical frameworks of pain explain the physiological basis of pain. A number of theories have been postulated to describe mechanisms underlying pain perception.

1. Intensive Theory (Erb, 1874)

This theory defines pain, not as a unique sensory experience but as an emotion that occurs when a stimulus is stronger than usual.

It says that stimulus intensity and central summation are critical determinants of pain. It was implied that the summation occurred in the dorsal horn cells.

There must be some form of summation that occurs for the sub-threshold stimuli to become unbearably painful.

2. Specificity Theory (Von Frey, 1895)

It holds that specific pain receptors transmit signals to a "pain center" in the brain that produces the perception of pain.

Von Frey (1895) argued that the body has a separate sensory system for perceiving pain.

This theory considers pain as an independent sensation with specialised peripheral sensory receptors [nociceptors], which respond to damage and send signals through pathways (along nerve fibres) in the nervous system to target centres in the brain. These brain centres process the signals to produce the experience of pain. Thus, it is based on the assumption that the free nerve endings are pain receptors.

3. Strong's Theory (Strong, 1895)

Strong investigated physical pain, particularly that felt through the skin. He proposed that pain was an experience based on both the noxious stimulus and the psychic reaction or displeasure provoked by the sensation. Strong concluded that pain is the sensation.

4. Pattern Theory

Goldschneider (1920) proposed that there is no separate system for perceiving pain, and the receptors for pain are shared with other senses, such as of touch.

This theory considers that peripheral sensory receptors, responding to touch, warmth and other non-damaging as well as to damaging stimuli, give rise to non-painful or painful experiences as a result of differences in the patterns [in time] of the signals sent through the nervous system. These patterns occur only with intense stimulation. Because strong and mild stimuli of the same sense modality produce different patterns of neural activity, being hit hard feels painful, but being caressed does not.

5. Central Summation Theory (Livingstone, 1943)

It proposed that the intense stimulation resulting from the nerve and tissue damage activates fibers that project to internuncial neuron pools within the spinal cord creating abnormal reverberating circuits with self-activating neurons. Prolonged abnormal activity bombards cells in the spinal cord, and information is projected to the brain for pain perception.

6. The Fourth Theory of Pain (Hardy, Wolff, and Goodell, 1940s)

It stated that pain was composed of two components: the perception of pain and the reaction one has towards it. The reaction was described as a complex physiopsychological

process involving cognition, past experience, culture and various psychological factors which influence pain perception.

7. Sensory Interaction Theory (Noordenbos, 1959)

It describes two systems involving transmission of pain: fast and slow system. The later presumed to conduct somatic and visceral afferents whereas the former was considered to inhibit transmission of the small fibers.

8. Gate Control Theory (Melzack and Wall, 1965)

According Melzack theory, pain stimulation is carried by small, slow fibers that enter the dorsal horn of the spinal cord; then other cells transmit the impulses from the spinal cord up to the brain. These fibers are called T-cells. The T-cells can be located in a specific area of the spinal cord, known as the substantial gelatinosa. These fibers can have an impact on the smaller fibers that carry the pain stimulation. In some cases they can inhibit the communication of stimulation, while in other cases they can allow stimulation to be communicated into the central nervous system. For example, large fibers can prohibit the impulses from the small fibers from ever communicating with the brain. In this way, the large fibers create a hypothetical "gate" that can open or close the system to pain stimulation. According to the theory, the gate can sometimes be overwhelmed by a large number of small activated fibers. In other words, the greater the level of pain stimulation, the less adequate the gate in blocking the communication of this information.

Explain the applications of Transcutaneous electrical nerve stimulation

Transcutaneous electrical nerve stimulation (TENS) is the application of low frequency current in the form of pulsed rectangular currents through surface electrodes on the patient's skin to reduce pain.

Methods of Application

Electrode placement: TENS electrode can be placed over

1. Area of greater intensity of pain.
2. Superficial nerve proximal to the site of pain.
3. To the appropriate dermatome.
4. To the nerve trunk trigger point.

A number of treatment methods may be used depending upon the severity of the problem.

1. TENS can be used for a single daily treatment of 40 minutes duration.
2. Portable TENS can be used continuously for 24 hours.
3. TENS can be used in night, e.g. for the treatment of phantom limb pain.

Add a note on mechanism of pain relief

Modulation of transmission of pain can be achieved by altering the excitability of pain pathway. The excitability of pain pathway can be altered by neurons (substantia gelatinosa) in the dorsal horn. The substantia gelatinosa (SG) cells have inhibitory influence on the T cells. This mechanism is called as presynaptic inhibition. Also, the nociceptive afferent sends collaterals to the substantia gelatinosa (SG) which inhibits the substantia gelatinosa cells when these nociceptive afferents are activated these causes inhibition of substantia gelatinosa (SG) cell activity which will further inhibit the mechanism of presynaptic inhibition thus allowing the nociceptive stimuli to reach the higher centers. Also, low threshold large diameter mechanosensitive afferent has excitatory influence on substantia gelatinosa (SG) cells. Their activation causes excitation of substantia gelatinosa (SG) activity which in result causes increased presynaptic inhibition blocking the transmission at T cells thus closes the gate for nociceptive stimuli to travel up to the higher center. This is the site where the pain gate operates. In addition to these input to SG cells from peripheral afferent there are descending influences on Transmission cells (T cells) which came principally from higher center such as periaqueduct gray matter PAG (midbrain) and raphe nucleus (medulla) these both have

excitatory influence on the substantia gelatinosa (SG) cells activity thus have ability to reduce pain transmission. These pathways are thought to exert their effect on Substantia gelatinosa (SG) cells by release of neurotransmitters such as noradrenaline and 5-hydroxy tryptamine.

During pain the inhibition on periaqueduct gray matter (PAG) and raphe nucleus (RN) is removed by influence of the limbic system thus allowing PAG and RN to exert its effect at substantia gelatinosa of dorsal horn of the spinal cord.

The TENS stimulates the large diameter myelinated fibers as these are highly sensitive to electrical stimulation and quickly conduct the electrical impulse to the spinal cord.

The A-delta and C-fibers are unable to pass the painful stimulus to spinal cord earlier than the large fibers.

This mechanism by which the nociceptor fibers are prevented from passing on their message to the spinal cord is called as presynaptic inhibition.