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Understanding how Pain Affects the Human Body and how it Reacts

Shah NP*

Department of Anaesthesiologist, Baroda, Gujarat

Pain is a stressor that can endanger homoeostasis since it is induced by an unpleasant (noxious) input. Physiological changes occur as a result of the body's adaptive response to pain, which are beneficial and potentially life-saving in the early stages. If the adaptive reaction continues, it may have detrimental and life-threatening consequences. Pain is a potent protective factor since it is toxic; moreover, the inability to feel pain is linked to a shorter life expectancy. After an accident, pain motivates us to engage in behaviours that aid in the healing process, such as resting the pained body part. [1] The physiological response to pain, its therapeutic relevance, and its wide-ranging effects on the body are all discussed in this article. It also shows how nurses can assist their patients with appropriate pain management.

Pain can be transmitted

The transmission of pain, which encompasses four stages: transduction, transmission, perception, and modulation, is the first physiological change that occurs in the body after a pain input.

Transduction

The pain stimulus is converted into a nerve impulse during transduction. Nociceptors, which are receptors on the surface of nerve endings, respond to noxious stimuli that might be thermal (temperatures exceeding 40°C), mechanical (extreme pressure over a narrow region), or chemical (strong acid or alkali) [2].

When a stimulus interacts with receptors, chemical changes occur, leading the nerve to send an electrical signal (action potential). If the stimulus is strong enough, the sensory nerve fibre will create an action potential. A huge stimulation causes a higher frequency of action potentials, which results in more acute pain.

The stimulus causes chemical pain mediators such as prostaglandin, bradykinin, serotonin, substance P, and histamine to be released by the nerve and nearby mast cells, which: Activate more receptors on the nerve fibre, increasing the likelihood that the threshold for an action potential will be reached – this is known as primary sensitisation;

The inflammatory response, which is an important element of healing, involves causing changes in the walls of local blood vessels, increasing blood supply and allowing white cells to move into the extracellular fluid.

[3] Reduced sensitisation and activation of nerve terminals can help relieve pain; for example, non-steroidal anti-inflammatory medicines (NSAIDs) can block the formation of prostaglandin, one of the main sensitising mediators, while opioids make it more difficult for the nerve to create an action potential. Both NSAIDs and opioids must be used with caution.

Transmission

The nerve impulse travels in three phases from the transduction site to the brain during transmission: from nociceptors to spinal cord, spinal cord to brain stem, and brain stem to other areas of the brain.

The electrical signal travels along the nerve by sodium and potassium ions cycling between extracellular and intracellular fluid. Myelinated fibres transmit information the fastest: Because A-delta fibres are only weakly myelinated, they convey pain impulses faster than C fibres. The sharp feeling felt soon after an injury is transmitted via A-delta fibres. The 'second pain,' which is a duller, searing feeling, is transmitted by C fibres.

The signal must pass a small fluid-filled space called the synapse once it reaches the end of the long axon of the primary afferent fibre (PAF), which runs from the periphery to the spinal cord. [4] Neurotransmitters are released, which diffuse across the synapse and activate receptors on the next neuron in the chain (secondary neuron), as well as surrounding glial cells and interneurons. The production of enough neurotransmitters by a powerful pain signal activates the secondary neuron, and the signal then travels to the brain, where it stimulates cells in the brainstem, thalamus, and cortex.

The transmission of the pain signal can be slowed or interrupted by injecting an anticonvulsant such as gabapentin or pregabalin close to the nerve bundle. Although there is some evidence that these medications can help with neuropathic pain, there is growing concern that some people, particularly those with a history of opiate addiction, may become hooked to them.

Perception

Recognizing, defining, and responding to pain are all part of perception, which is when pain becomes a conscious experience. The cortex (location and motor reaction), limbic system (emotional response), and reticular system are all involved (arousal response). Distraction can be a useful technique for taking the mind off pain as part of a larger pain management strategy, and it has been shown to reduce the need for opioids in persons with severe trauma pain [5].

Modulation

Pain is reduced or increased by the body during modulation, the final stage of pain transmission, via descending (from brain to spinal cord) and ascending (from spinal cord to brain) mechanisms.

Pain signals activate the brainstem, which causes endogenous opioids (endorphin and encephalin), serotonin, noradrenaline, gamma-amino butyric acid (GABA), and neurotising to be released by descending nerve fibres. These chemical mediators bind to receptors on the PAF and secondary neuron, preventing neurotransmitters from being released and making it more difficult for the secondary neuron to generate an action potential. The brain activates this process, known

***Corresponding author:** Shah NP, Department of Anaesthesiologist, Baroda, Gujarat, Tel: 9898019910; E-mail: nehakinit@gmail.com

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as descending pain inhibition, when pain suppression is critical, such as when in danger.

Synthetic copies of chemicals produced by the body, such as the opiate morphine, the antidepressant duloxetine, or the anticonvulsant gabapentin, can be used to activate or increase descending pain inhibition [6]. Descending pain inhibition can also be activated by hypnosis (deep relaxation), which has been shown to be effective in the treatment of acute and chronic pain, as well as in the treatment of needle phobia. Acupuncture, exercise, and transcutaneous electrical nerve stimulation can all help to increase endogenous opioid production (TENS).

Ascending mechanisms can also be used to control pain. Touch or pressure activates A-beta fibres, which activate the same secondary neurons as C fibres. [7] The signal provided by a C fibre activating the secondary neuron indicates pain, whereas the signal created by an A beta fibre activating the secondary neuron represents touch. Only one of the two types of fibres, A beta and C, can activate the secondary neuron. The gate control theory states that when a large number of A beta fibres are stimulated, pain signalling is suppressed.

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