Pain

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Opinion

Pain acts as a protective mechanism of the body, by forcing the person to react so that it is removed from the stimulus. It is important not only for cases where there is marked tissue damage, but also for everyday simple activities. Thus, when a person sits on the hips for a long time, it is possible to damage the tissues due to the inhibition of the skin's blood supply to the places where the skin is compressed by body weight. When the skin starts to ache because of ischemia, the person completely unconsciously changes position. However, when the sensation of pain is lost, as is the case with spinal cord injury, the person cannot feel the pain and thus does not change position. This condition leads to ulcers in the area where the pressure is applied very quickly [1-3]. The feeling of pain is caused by the irritation of the nerve receptors in the skin and deep tissues. There are two different types of pain: rapid pain and slow pain (rapid and slow pain). Rapid pain occurs within 0.1 second after the application of alogen stimuli, while slow pain begins to be felt after a second or more. Subsequently, the intensity increases slowly for many seconds, and in many cases even for several minutes. Rapid pain is also described as acute pain, cramping pain, electrical pain, etc. This type of pain can be felt by inserting a needle into the skin, either by knife-fitting the skin and by the effect of electrical discharge on the skin. Rapid pain is not felt by most of the body's deeper tissues [4,5]. Slow pain is characterized as caustic pain, shallow pain, pulse pain, chronic pain, etc. This type of pain is usually associated with tissue damage. It can become excruciating and can lead to long unbearable hopeless anxiety. It can come from both the skin and any deep tissue or organ. The ways to treat these two types of pain are different. The centripetal fibers that lead to rapid pain are thin, type AD-fibers, while the fibers for slow pain are type C and starches. The feeling of pain they transmit is characterized as "slow", prolonged, blunt and diffuse pain. All pain receptors are free nerve endings. They abound in the superficial layers of the skin, as well as in some internal tissues, such as the periosteum, arterial walls, articular surfaces, as well as the sickle and skull dome. Most other deep tissues are not richly equipped with nerve endings of pain [6,7].

However, any extensive tissue damage may cause pain of a slow type from these areas. The pain receptors are triggered by mechanical, thermal and chemical stimuli. Although most pain nerve fibers are stimulated by multiple stimuli, some fibers respond more to excessive mechanical stretching, others to excessively high or low temperature, while others react more strongly to specific chemicals within the tissues [8]. These nerve endings are respectively classified as mechanical, thermal and chemical pain receptors. Rapid pain is released by mechanical and thermal receptors, while slow pain can be released by all three types of pain receptors. Some of the chemicals that stimulate the chemical receptors of pain are bradykinin, serotonin, histamine, potassium ions, acids, acetylcholine, and proteolytic enzymes. In addition, prostaglandins enhance the sensitivity of nerve endings to pain, but do not cause immediate stimulation. These chemicals are especially important in relieving the slow excruciating pain that occurs after tissue damage. Although all pain nerve endings are free nerve endings, two separate nerve pathways are used to treat these nerve impulses to the central nervous system. These two neural pathways correspond to the two types of pain, namely one pathway for rapid, acute pain and another pathway for slow, chronic pain. 'double' pain: a rapid-onset pain, followed by about a second of slow, burning pain [9]. Rapid pain informs the individual about the effect of the harmful factor, so this type of pain plays an important role in the person's immediate reaction and removal from the stimulus. In addition, the slow burning sensation tends to become more and more painful over time. Eventually, this feeling becomes unbearable constant pain.

After entering the spinal cord with the posterior nerve roots, the nerve fibers of the pain rise or fall for one to three neurotomies in the Lissauer Street, which is just behind the posterior horn, and then make synapses with neurons of the hind horns. But even at this point, there are two systems for processing pain signals along the way to the

brain. fibers have "no", while the Cines lack it because they carry a variety of stimuli. However, CNS pain tolerance also determines the requirements for analgesics and not the "threshold" of peripheral nerves [10]. The AD and C fibers are embedded in the gray matter of the posterior spinal cord. The A-d fibers end up in zone 1, while the C fibers in zone 2 of Rexed (gelatinous substance). They then connect through the ascending threshold to the spinal cord. The first and basic treatment of painful stimuli is done in the spinal cord through or with the help of inhibitory neurons or suppressor neurons of the cation formation.

This mechanism is part of the "pain gate" as proposed by Melzack, et al. [6]. A small number of fibers of the neo-thalamic pathway end up in the reticular areas of the brainstem, but most fibers reach the optic cavity and end up in the ventricular complex, along the anterior path of the hind limbs. Also, a small number of these fibers end up in the back cores of the optical chamber. From these areas the signals are transmitted to other areas at the base of the brain as well as to the body's aesthetic state of the cortex.

References

1. Millan MJ (2002) Descending control of pain. Prog Neurobiol 66(6): 355-474.

2. Meyer RA, Ringkamp M, Campbell JN (2006) Peripheral neural mechanisms of nociception. In: McMahon SB, Koltzenburg M, et al. (Eds.), Wall and Melzack's Textbook of pain. 5th (Edn.), Philadelphia: Elsevier Limited, pp: 3-34.

3. Guilbaud G, Besson JM (1997) Physiology of the pain circuit. In: Brasseur L, Chauvin M, Guilbaud G, et al. (Eds.), Douleurs. Paris: Maloine, pp: 7-22.

4. Levine J, Taiwo Y (1994) Inflammatory pain. In: Wall PD, Melzack R, et al. (Eds.), Texbook of pain. New York: Chruchill Livingston, pp: 45-56.

5. Byers MR, Bonica JJ (2001) Peripheral pain mechanisms and nociceptor plasticity. In: Loeser JD, et al. (Eds.), Management of pain. New York: Lippincott Williams & Wilkins, pp: 26-72.

6. Melzack R, Wall PD (1965) Pain mechanisms: a new theory. Science 150(3699): 971-979.

7. Price DD (1999) Primary afferent and dorsal horn mechanisms of pain. In: Price DD, et al. (Eds.), Psychological mechanisms of pain and analgesia. New York: Raven Press, pp: 71-96.

8. Treede RD, Meyer RA, Campbell JN (1998) Myelinated mechanically insensitive afferents from monkey hairy skin: heat-response properties. J Neurophysiol 80(3): 1082-1093.

9. Stander S, Steinhoff M, Schmelz M, Weisshaar E, Metze D, et al. (2003) Neurophysiology of pruritus: cutaneous elicitation of itch. Arch Dermatol 139(11): 1463-1470.

10. Olausson H, Lamarre Y, Backlund H, Morin C, Starck G, et al. (2002) Unmyelinated tactile afferents signal touch and project to insular cortex. Nat Neurosci 5(9): 900-904.