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The Nature and Science of Pain

Key Points

- Pain is a subjective experience influenced by physical (nociceptive and neuropathic), psychological and environmental factors.
- Pain can be acute, sub-acute, recurrent, or chronic.
- Left untreated or under-treated, acute pain can become chronic.
- Chronic pain can become a disease in its own right.

What is pain?^{1,2}

The International Association for the Study of Pain describes pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage".

Pain is the result of complex brain processes, which are influenced by overlapping physical (nociceptive and neuropathic), psychological, and environmental factors.

All pain is an individual human experience that is entirely subjective and can only truly be appreciated by the person experiencing the pain.

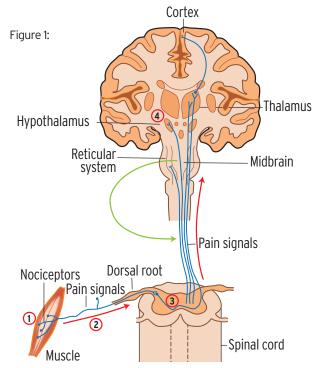
A person's attitudes, beliefs and personality can strongly affect their pain experience.

Acute pain is pain that lasts for a short time and occurs following surgery or trauma or other condition. It acts as a warning to the body to seek help.

Sub-acute pain is pain that is progressing towards chronic pain, but this progression may be prevented. This is known as the transition phase.

Recurrent pain is pain that occurs on a cyclical basis, such as migraine or pelvic pain.

Chronic pain is pain that lasts beyond the time expected for healing following surgery or trauma or



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other condition. It is often associated with an increased pain experience, not just in the area of injury, but also in surrounding tissue or nerves. It can also exist without a clear reason at all.

What is the mechanism for nociceptive pain?²

Nociceptors are specialised nerve endings that are activated by a biological stimulus of sufficient intensity to threaten tissue damage. The cell bodies of these nociceptors are located outside the spinal cord in the dorsal root ganglia and extend to the periphery.

Activation of nociceptors triggers incoming impulses that travel to the spinal cord almost exclusively through the dorsal root, and synapse in the dorsal horn of the spinal cord, where they project to higher levels such as the thalamus, hypothalamus, reticular system and cortex of the brain. (See Figure 1)

The higher-level sites bring about the adverse emotional feelings (thalamus and limbic system), alterations in sleep patterns (reticular system and hypothalamus) and stress responses (hypothalamus) that pain may evoke.

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What is the mechanism for neuropathic pain?²

Neuropathic pain is initiated or caused by injury to neural tissue in the central nervous system (CNS) or peripheral nervous system (PNS).

Neuropathic pain can sometimes be difficult to identify because it can be present in an area that has no feeling, and it can cover an unusual area. Patients with neuropathic pain often complain not only of spontaneous pain, but also of pain from stimuli that are not normally painful, such as a breeze or light touch.

Figure 2 lists the main types of neuropathic pain and their probable causes.

Figure 2: Types of neuropathic pain and their probable causes	
Туре	Cause
Trigeminal neuralgia	Compressin of trigeminal ganglion or its branches
Postherpetic neuralgia	Shingles
Complex regional pain syndrome	Trauma/infection/surgery/ inflammation
Diabetic neuropathy	Persistent hyperglycemia (diabetes)
Central pain	Trauma to the spinal cord
	Stroke
Phantom pain	Amputation
Postincisional pain	Surgery

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What is the Gate Theory?^{3,4}

The Gate Theory, developed in 1965, revealed that the PNS and CNS are more than just a collection of cables passively collecting nociceptive information.

Rather, the nervous system is dynamic, and its structure and function are shaped and reshaped by activity within it. At each level it continually amplifies or inhibits the signals that the brain ultimately interprets as pain.

Importantly, while all pain messages reach the spinal cord, their entry to the brain can be determined by other nerve messages – powerful transmitters that are able to decrease or increase the passage of "painful messages" on their way to the brain.

In the case of chronic pain, the "gate" is left open, allowing for pain messages to persist, and even routine daily activities will result in the release of chemicals in the spinal cord that increase noxious traffic reaching the brain.

At the level of the brain, similar but more complex mechanisms input into a pathway down to the spinal cord, known as "descending modulation". This provides further "fine tuning" of the ascending pathway.

Can acute pain become chronic?^{2,5,6}

The human brain boasts a remarkable ability to change and effectively "rewire" itself, a concept known as "neuroplasticity".

This is particularly important when it comes to pain. Acute pain that is left untreated or under-treated can lead to neuroplastic changes within the nervous system at peripheral, spinal cord and brain levels.

After tissue or nerve damage, peripheral nociceptors become sensitised to noxious stimuli, and this peripheral sensitisation along with persistent stimulation in pain fibres can elicit functional, chemical and anatomic reorganisation in spinal cord neurons.

The resulting pain "memory" leads to "pain sensitisation", where pain signals are transmitted unnecessarily – long after the original source of pain (surgery, trauma or other condition) has healed – and pain is now chronic.

Is chronic pain a separate disease entity? ^{2,6,7}

Pain has often been regarded merely as a symptom that serves as a passive warning signal of an underlying disease process.

However, accumulating evidence shows that chronic pain has a distinct pathology that can worsen over time, it can be present without an obvious cause or source, and can result in severe physical, psychological and environmental changes in the individual – constituting a disease entity in its own right.

The physical aspects of chronic pain are largely a complex range of abnormalities in the CNS, with the end result a highly sensitised state of the nervous system, predisposing to chronic pain. It is likely that psychological and environmental factors also influence functioning of the CNS.

The concept of chronic pain as a disease was initially proposed in 2004, and has been adopted by National Pain Summits in Australia (2010) and Canada (2010), and by the US Institute of Medicine in 2011. It is also a central understanding of Australia's National Pain Strategy.

References

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