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Pain Terminology and Pain Pathways

Pain: "An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage."

International Association for the Study of Pain

Nociception: The activity in neural pathways that transmits or processes information about noxious events associated with tissue damage.

Pain: Pain is not the same as nociception. Pain is an individual and unique sensory experience combining nociceptive signals with inputs from cognitive, memory and emotional centers in the brain. In some clinical conditions nociception due to tissue damage can occur but the patient may not perceive it. Conversely the patient may report severe pain implying that there is tissue damage happening somewhere in the body, but a thorough search for that tissue damage fails to demonstrate it. In animal models, genetic influences can accounts for up to a 50-fold variability in pain sensitivity.

Suffering: The global impairment of quality of life due to a combination of pain, decreased function, and various psychosocial factors, such as cognitive status, emotional state and environmental factors.

Acute Pain: Pain that is of recent onset, usually caused by tissue damage and lasting from several seconds to several weeks. It may or may not be associated with overt pain behaviours (e.g.. grimacing, splinting, limping, sympathetic nervous system signs) depending on intensity, predictability and meaning of the pain. (ie. chest pain in a 50 y.o. male would likely have a different meaning than in a 24 y.o. female) It usually serves a biological function in protecting the organism from further tissue damage.

Chronic Pain: Pain persisting beyond the usual course of an acute illness or injury (usually beyond 3-6 months), associated with a pattern of recurrence over months or years or associated with a chronic, ongoing pathological process. It may be accompanied by affective (emotional) and vegetative (depressive) symptoms but the overt pain behaviours of acute pain are usually absent.

Baseline Pain: pain experienced for more than ½ the day

Breakthrough Pain: a transitory increase in pain to greater than moderate intensity that occurs on a baseline pain of moderate intensity or less. Clinically very relevant because it is very disturbing to the patient due to increased levels of psychological distress and significant effects on function.

Incident pain is a type of break through pain, which is made worse by movement.

Sensitization of Pain Pathways

Within minutes of the onset of tissue damage causing persistent pain, neurochemical and structural changes begin to take place in the body's pain pathways. In the transition from acute to chronic pain states, these changes can become "hard wired" into the system and result in an increase in the intensity of pain signals transmitted to the brain:

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Peripheral (Primary): Nociceptors are sensitized by the release of various chemical mediators at the site of tissue damage, causing a reduced response threshold for firing and an increased response magnitude to any noxious stimulation. In addition, these chemical mediators "awaken" a group of silent nociceptors which further increases the input of pain signals to the spinal cord.

Central (Secondary): Increased pain signals from peripheral nociceptors cause increased release of neurotransmitters such as Substance P, Glutamate, Aspartic Acid and Nitric Oxide in the spinal cord. This results in hyperexcitability of spinal neurons and increased pain signal transmission to the brain. Chronic pain signals may also change the balance of neurotransmitters in key areas of higher centers in the brain resulting in an increased perception of pain.

Neuronal plasticity: a chemical and physical "rewiring" of the central pain transmission system in the spinal cord via the sprouting of new sensory and sympathetic nerve terminals in response to continued firing of peripheral nociceptors and/or direct nerve damage. This change appears to involve local mediators such as Nerve Growth Factor (NGF), excitatory amino acids (EAA) such as glutamate and aspartic acid and the molecule nitric oxide (NO). The net effect of this rewiring is to decrease the threshold of the stimulus required to fire a pain neuron and to increase the intensity of pain signals sent to the brain.

Sensitization of CNS pain pathways can result in self-sustaining neuropathic pain with some or all of the following clinical phenomenon:

Hyperalgesia: an increased painful response to a mildly painful stimulus (i.e. pinch).
Widened Receptive Fields: hypersensitivity to painful or mechanical stimulation in a large, often non-dermatomal area surrounding a focus of chronic nociception.
Mechanical Allodynia: light touch of an affected area causes pain.
Wind-up: a repetitive, low intensity stimulus (i.e. repetitive pinprick) causes an incremental increase in the firing of pain neurons and rapidly increasing pain.
Spontaneous nociceptor or spinal cord pain neuron firing: (similar to an epileptic discharge in the brain)

Capsaicin-induced mechanical allodynia is an example of central sensitization and neuronal plasticity. Capsaicin directly stimulates C-fibers without causing tissue damage. Intradermal injection of a small amount on the back of the hand causes intense localized burning pain at the site of injection for about 20 minutes. After this time it causes a large surrounding zone of hyperalgesia and mechanical allodynia which can last for hours to days. This reaction can be decreased significantly by giving a systemic dose of an NMDA receptor blocking drug such as ketamine. This phenomenon helps explain the often non-anatomical distribution of neuropathic pain syndromes.

In research and clinically, neuropathic pain is often seen to have at least 3 components: a background, constant, often burning or prickly component, an intermittent, sharp, lancinating component and touch sensitivity of the skin in the involved area. These 3 manifestations may respond individually and differently to a given treatment - so called “differential sensitivity.” Thus one needs to ask about the different components of a patient’s pain when assessing treatment efficacy.

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Categories of Pain

A lesion anywhere in the nervous system that innervates a given part of the body can refer pain to that part of the body. It is reassuring to find "objective" organic lesions when trying to assess the relative contributions of body and mind to a patient's pain complaint. However, we cannot rely totally on the presence or absence of visible lesions because of the multidimensional nature of pain, the complexity of the wiring of the nervous system and the relatively crude tools we currently have to identify the precise location of lesions causing focal pain. A good example of this is the changing view of chronic whiplash pain due to the published research of Bogduk and colleagues regarding the role of the zygapophyseal joint. We do however have clinical experience that can help us to categorize and treat patients' pain even without knowing the precise pathophysiology.

1. Identification of pain syndromes: can guide us to appropriate investigation and specific curative treatments. Over 70 specific pain syndromes have been identified mostly in the cancer pain literature but also in the non-cancer pain literature.

2. Inferred pathophysiologies of pain: may allow us to select specific therapies that work better for some types of pain than for others.

a)Nociceptive pain: ongoing activation of nociceptive pathways by ongoing tissue damage

Somatic pain: . osteomyelitis, arthritic pain, and tumours

Visceral pain: chronic pancreatitis, endometriosis, Crohn's disease

b)Neuropathic pain: Pain initiated or caused by a primary lesion or dysfunction in the CNS pain transmission system

Peripheral

Mononeuropathies: carpal tunnel, herniated disk, postherpetic neuralgia

Polyneuropathies: diabetic, HIV neuropathy

Central

Deafferentation: phantom limb, post-stroke pain, nerve avulsions

Sympathetically Maintained Pain: causalgia, reflex sympathetic

c)Idiopathic pain: pain in the absence of demonstrable organic pathology or in excess of the degree of visible organic pathology (ie fibromyalgia)

d)Psychologically-mediated pain: pain which is a manifestation of a primary psychiatric disorder or is primarily influenced by psychologic processes. (i.e. somatization disorder).

Although most patients with chronic pain will have some psychological influences on their degree of suffering, true "psychogenic" pain (an older, imprecise term) is very rare and should be classified using the IASP or DSM IV criteria. Our ongoing discoveries regarding the complexities of the CNS pain system should make the clinician very careful in assigning this stigmatizing label to a patient with unexplained chronic non-cancer pain.