Section 2

Nociception

"Pain refers to the subjective sensation that accompanies damage or near-damage to tissues. Pain can also occur in the absence of tissue damage if the systems of nociception are not functioning properly. Nociception is the physiological event that accompanies pain and refers to the system that carries signals of damage and pain from the tissues." [1]

Transmission of Nociception to the Central Nervous System

Nociceptive information reaches the central nervous system two ways via the neospinothalamic tract for "fast spontaneous pain"; and by way of the paleospinothalamic tract for "slow increasing pain" [2]

Neospinothalamic Tract

Fast spontaneous pain travels via type A-delta fibers to terminate on the dorsal horn of the spinal cord where they synapse with the dendrites of the neospinothalamic tract. The axons of these neurons travel up the spine to the brain and cross the midline through the anterior white commissure, passing upwards in the contralateral anterolateral columns. These fibers terminate on the ventrobasal complex of the thalamus and synapse with the dendrites of the somatosensory cortex. Fast pain is felt within a tenth of a second of application of the pain stimulus and is a sharp, acute, prickling pain felt in response to mechanical and thermal stimulation.[3]

Paleospinothalamic Tract

Slow pain is transmitted via slower type C fibers to laminae II and III of the dorsal horns, together known as the substantia gelatinosa. Impulses are then transmitted to nerve fibers that terminate in lamina V, also in the dorsal horn, synapsing with neurons that join fibers from the fast pathway, crossing to the opposite side via the anterior white commissure, and traveling upwards through the anterolateral pathway. These neurons terminate throughout the brain stem, with one tenth of the fibers stopping in the thalamus and the rest stopping in the medulla, pons and periaqueductal grey of the midbrain tectum. Slow pain is stimulated by chemical stimulation, is poorly localized and is described as an aching, throbbing or burning pain. [4]

A-Delta and C Fiber Synopsis

- "The thin (A-delta and C) peripheral sensory fibers carry information regarding the state of the body to the spinal cord. Some of these thin fibers do not differentiate noxious from non-noxious stimuli, while others, nociceptors, respond only to painfully intense stimuli."[5],[6]
- "Because the A-delta fiber is thicker than the C fiber and is thinly sheathed in an electrically insulating material (myelin), it carries its signal faster (5–30 m/s) than the unmyelinated C fiber (0.5–2 m/s). Pain evoked by the (faster) A-delta fibers is described as sharp and is felt first. This is followed by a duller pain, often described as burning, carried by the C fibers."[7],[8]

- "Spinal cord fibers dedicated to carrying A-delta fiber pain signals, and others that carry both A-delta and C fiber pain signals up the spinal cord to the thalamus in the brain have been identified. Other spinal cord fibers, known as wide dynamic range neurons, respond to A-delta and C fibers, but also to the large A-beta fibers that carry touch, pressure and vibration signals. Pain-related activity in the thalamus spreads to the insular cortex (thought to embody, among other things, the feeling that distinguishes pain from other homeostatic emotions such as itch and nausea) and anterior cingulate cortex (thought to embody, among other things, the motivational element of pain."[9]

Effects in the Central Nervous System

When nociceptors are stimulated they transmit signals through sensory neurons in the spinal cord. These neurons release the excitatory neurotransmitter glutamate at their synapses. If the signals are sent to the reticular formation and thalamus, the sensation of pain enters consciousness in a dull, poorly localized manner. From the thalamus, the signal can travel to the somatosensory cortex in the cerebrum when the pain is experienced as localized and having more specific qualities.
Nociception can also cause generalized autonomic responses before or without reaching consciousness to cause pallor, bradycardia, hypotension, lightheadedness, nausea and fainting.[10]

Nociceptors

Nociceptors are free nerve endings whose cell bodies are found outside of the spinal column in the dorsal root ganglia . Nociceptors can detect mechanical, thermal, and chemical stimuli, and are found in the skin and on internal structures such as the periosteum or joint surfaces.

The body's pain receptors are:

- **Mechanical**: capable of detecting a stretch gone too far.
- **Thermal**: receptors capable of detecting extreme heat or cold .
- **Chemical**: receptors that can detect body products released during trauma or inflammation. (Lactic acid, for example, causes muscle pain after heavy exercise.)

Deep internal surfaces are only weakly supplied with pain receptors and will propagate sensations of chronic, aching pain if tissue damage in these areas occurs. Nociceptors do not adapt to stimuli. In some conditions, excitation of pain fibers becomes greater as the pain stimulus continues, leading to hyperalgesia.

Noxious Stimulus

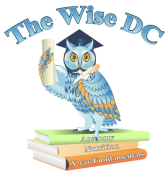
A noxious stimulus is one which is damaging to normal tissues.

Pain Threshold
Is the least stimulus intensity at which a subject perceives pain. Pain threshold is really the experience of the patient, whereas the intensity measured is an external event. In psychophysics, thresholds are defined as the

level at which 50% of stimuli are recognized. In that case, the pain threshold would be the level at which 50% of stimuli would be recognized as painful. The stimulus is not pain and cannot be a measure of pain.[11]

Peripheral Neuropathic Pain

Pain initiated or caused by a primary lesion or dysfunction in the peripheral nervous system.



[1] "Assessing Pain and Distress: A Veterinary Behaviorist's Perspective by Kathryn Bayne" in "Definition of Pain and Distress and Reporting Requirements for Laboratory Animals: Proceedings of the Workshop Held June 22, 2000 (2000)
[2] http://en.wikipedia.org/wiki/pain-and-nociception
[3] http://en.wikipedia.org/wiki/pain-and-nociception
[4] http://en.wikipedia.org/wiki/pain-and-nociception
[5] Shelemay KK, Coakley S. *Pain and its transformations: the interface of biology and culture*. Cambridge: Harvard University Press; 2007. ISBN 0-674-02456-7. Deconstructing pain: A deterministic dissection of the molecular basis of pain. p. 3.
[6] Craig AD. Pain mechanisms: labeled lines versus convergence in central processing. *Annu. Rev. Neurosci.*. 2003;26:1–30. doi:10.1146/annurev.neuro.26.041002.131022.
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