Section 3

Pain Theories

Intensive Theory

In 1794 Erasmus Darwin postulated the idea in Plato's *Timaeus*, that pain is not a unique sensory modality, but an emotional state produced by stronger than normal stimuli such as intense light, pressure or temperature. [1],[2]

Following up on Darwin's findings, in 1874 Wilhelm Erb opined that pain can be generated by any sensory stimulus, provided it is intense enough, and his formulation of the hypothesis became known as the intensive theory. [3] Wilhelm Erb's intensive theory, that a pain signal can be generated by intense enough stimulation of *any* sensory receptor, has been soundly disproved.

Subsequent research in 1884 by Alfred Goldscheider confirmed the existence of distinct heat and cold sensors, by evoking heat and cold sensations using a fine needle to penetrate to and electrically stimulate different nerve trunks, bypassing their receptors. Though he failed to find specific pain sensitive spots on the skin, Goldscheider concluded in 1895 that the available evidence supported pain specificity.

Bernhard Naunyn performed pain experimentation in 1889 in which he rapidly (60–600 times/second) prodded the skin of tabes dorsalis patients, below their touch threshold (e.g., with a hair), and in 6–20 seconds produced unbearable pain. He obtained similar results using other stimuli including electricity to produce rapid, sub-threshold stimulation, and concluded pain is the product of summation.

" In 1894 Goldscheider extended the intensive theory, proposing that each tactile nerve fiber can evoke three distinct qualities of sensation – tickle, touch and pain – the quality depending on the intensity of stimulation; and extended Naunyn's summation idea, proposing that, over time, activity from peripheral fibers may accumulate in the dorsal horn of the spinal cord, and "spill over" from the peripheral fiber to a pain-signalling spinal cord fiber once a threshold of activity has been crossed.[4],[5]

"William Kenneth Livingston advanced a summation theory in 1943, proposing that high intensity signals, arriving at the spinal cord from damage to nerve or tissue, set up a reverberating, self-exciting loop of activity in a pool of interneurons, and once a threshold of activity is crossed, these interneurons then activate "transmission" cells which carry the signal to the brain's pain mechanism; that the reverberating interneuron activity also spreads to other spinal cord cells that trigger a sympathetic nervous system and somatic motor system response; and these responses, as well as fear and other emotions elicited by pain, feed into and perpetuate the reverberating interneuron activity. A similar proposal was made by RW Gerard in 1951, who proposed also that intense peripheral nerve signalling may cause temporary failure of inhibition in spinal cord neurons, allowing them to fire as synchronized pools, with signal volleys strong enough to activate the pain mechanism."[6]

The Specificity Theory Of Pain

In 1644, French philosopher, mathematician and scientist, René Descartes proposed a theory of pain that survived until the mid-1960s.Descartes postulated that the human body was a form of machine and therefore, could be studied like other mechanical entities.According to Descartes' classic thoughts regarding pain, if a

person cut their finger a half-inch in length, it would hurt twice as much as if they had cut it a quarter of an inch in length. In Descartes' model, pain traveled in a single direction. Under Descartes' influence, the scientific search for how pain was conducted culminated in what has come to be known as the specificity theory. This theory posits that pain is a simple system where an input of one kind travels along special nerves for that kind of input, terminating in specific areas of the brain which are receptive to that input. The input which went in was thought to be what was felt by the brain. This belief resulted in the use of various inappropriate kinds of therapy, including the cutting of nerve pathways to try and abolish chronic pain.[7]

"Descartes theory proposed that the intensity of pain is directly related to the amount of associated tissue injury. For instance, pricking one's finger with a needle would produce minimal pain, whereas cutting one's hand with a knife would cause more tissue injury and be more painful. This theory - the "specificity theory" - is generally accurate when applied to certain types of injuries and the acute pain associated with them. But chronic pain is often quite different, though no less severe, and a more extensive and up to date scientific understanding of pain is required to treat it. Unfortunately, many practicing doctors still try to extend the specificity theory to chronic pain cases. This approach is probably not valid when studying or treating chronic back pain. The theory assumes that if surgery or medication can eliminate the alleged "cause" of the pain, then the chronic pain will disappear. This is very often not true for chronic pain. If doctors continue to apply the specificity theory to a patient's chronic pain problem, the patient is at risk for receiving unnecessary and ineffective diagnostic procedures, drugs and surgical treatment as the search for the patient's "source of chronic back pain" presses on."[8]

Problems With The Specificity Theory And Chronic Pain

Up until the introduction of the gate control theory of pain in 1965, the specificity theory had been the dominant idea in the study of pain. One of the first doctors to question its validity was Dr. Henry Beecher. Dr. Beecher began his investigation into relationships between subjective psychological states and objective drug responses during his work with severely wounded soldiers in World War II.
Beecher's clinical observations proved that the specificity theory was inadequate to explain chronic pain. He observed that only one out of five soldiers carried into a combat hospital complained of enough pain to require morphine. When Dr. Beecher returned to his practice in the United States after the war, he noticed that trauma patients with wounds similar to those of the soldiers he had treated were much more likely to require morphine to control their pain. In fact, one out of three civilian patients required morphine for pain from these wounds. Dr. Beecher concluded that there was no direct relationship between the severity of the wound and the intensity of pain. Dr. Beecher believed the meaning attached to the injuries in the two groups explained the different levels of pain. To the soldier, the wound meant surviving the battlefield and returning home. Alternatively, the injured civilian often faced major surgery and a resulting loss of income, diminishment of activities, and many other negative consequences.[9]

Another finding that discredited the specificity theory was that of phantom limb pain. Patients who undergo the amputation of a limb may continue to report sensations or chronic pain that seems to come from the limb that has been amputated. This may include feeling that the limb is still there, or it may be a sensation of chronic pain. Clearly, these sensations cannot actually come from the limb since it has been removed. The specificity theory cannot account for these findings since there is no ongoing tissue injury in the amputated limb, which would mean that there should be no chronic pain.[10]

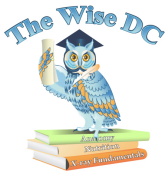
"The specificity theory cannot explain how hypnosis can be used for anesthesia during surgery. Under hypnosis, certain people can evidently undergo significant tissue damage from surgery without experiencing intense pain. This would support the notion that one's mental state or frame of mind can override the specificity theory. Similar examples of severe pain or chronic pain following relatively minor injuries can also be furnished." [11]

Upon final analysis, the specificity theory was clearly erroneous because it implied that if there was no specific, identifiable injury to account for the pain then it could not exist. Therefore, patients who did complain of pain in those circumstances were often mistakenly diagnosed as being mentally ill.

Peripheral Pattern Theory

In 1955, DC Sinclair and G Weddell proposed the "peripheral pattern theory". This theory ignored a large body of strong evidence for receptor fiber specificity and contended that all skin fiber endings (with the exception of those innervating hair cells) are identical, and that pain is produced by intense stimulation of these fibers.[12]

In 1953, Willem Noordenbos observed that a signal carried from the area of injury along large diameter "touch, pressure or vibration" fibers may inhibit the signal carried by the thinner "pain" fibers - the ratio of large fiber signal to thin fiber signal determining pain intensity; hence, we rub a smack. This observation was taken as a demonstration that pattern of stimulation (of large and thin fibers in this instance) modulates pain intensity.[13]



[1] Finger S. *Origins of neuroscience: a history of explorations into brain function*. USA: Oxford University Press; 2001.
[2] Wilhelm Erb
[3] Dallenbach KM. Pain: History and present status. *American Journal of Psychology*. July 1939;52:331–347.
[4] Dallenbach KM. Pain: History and present status. *American Journal of Psychology*. July 1939;52:331–347.
[5] Norrsell U, Finger S and Lajonchere C. Cutaneous sensory spots and the "law of specific nerve energies": history and development of ideas. *Brain Research Bulletin*. 1999;48(5):457-465.
[6] Bonica JJ. *The management of pain*. 2 ed. Vol. 1. London: Lea & Febiger; 1990. History of pain concepts and therapies. p. 7.
[7] www.spine-health.com
[8] www.spine-health.com
[9] Deardorff,William W. PhD, (March 11, 2003).Problems with the specificity theory and chronic pain
[10] Deardorff,William W. PhD, (March 11, 2003).Problems with the specificity theory and chronic pain
[11] Deardorff,William W. PhD, (March 11, 2003).Problems with the specificity theory and chronic pain.
[12] Bonica JJ. *The management of pain*. 2 ed. Vol. 1. London: Lea & Febiger; 1990. History of pain concepts and therapies. p. 7.
[13] Todd EM, Kucharski A. Pain: Historical Perspectives. In: Bajwa ZH, Warfield CA. *Principles and practice of pain medicine*. 2nd ed. New York: McGraw-Hill, Medical Publishing Division; 2004.