Section 4

The Gate Control Theory Of Pain

Ronald Melzack and Patrick Wall introduced the gate control theory of pain in 1965.The gate control theory is based on several propositions:

- The transmission of impulses from the body into the central nervous system is “gated” (altered, changed, modulated) in the spinal cord.
- Gating is affected by the degree of activity in the large diameter and the small diameter nerve fibers. Impulses along the larger fibers tends to block pain transmission (close the gates) and more activity in the smaller fibers tends to facilitate transmission (open the gates).
- This gating mechanism in the spinal cord is affected by descending impulses from the brain.
- Large fibers may activate specific cognitive processes in the brain, which then influence the gate by descending impulse transmission.
- When the pain output to the brain reaches a certain level it activates the action system and the individual in pain takes an action to alleviate the pain.[1]

Melzack and Wall theorized that the perception of physical pain is not a direct result of activation of pain receptor neurons, but instead is modulated by the interaction between different neurons. Melzack utilized these scientific and clinical observations to explain how pain is multidimensional and not just something that happens to the body, as Descartes’ mechanistic view of pain suggested. Melzack and Wall further proposed that pain is ‘gated’ or modulated by past experiences. They suggested that once the noxious agent that triggered the pain had ‘opened the pain gate’; other factors may contribute to the perpetuation of pain. Although their work drew on Descartes’ theory, they went further to claim that the brain and central nervous system played integral and active parts in receiving, modulating and transmitting pain impulses. As a result of their research, a bodily function for pain was proposed and chemical and surgical treatment methodologies were developed to help alleviate, or even completely remove patient complaints of pain.[2]

The Physiology of the Gate Control Theory

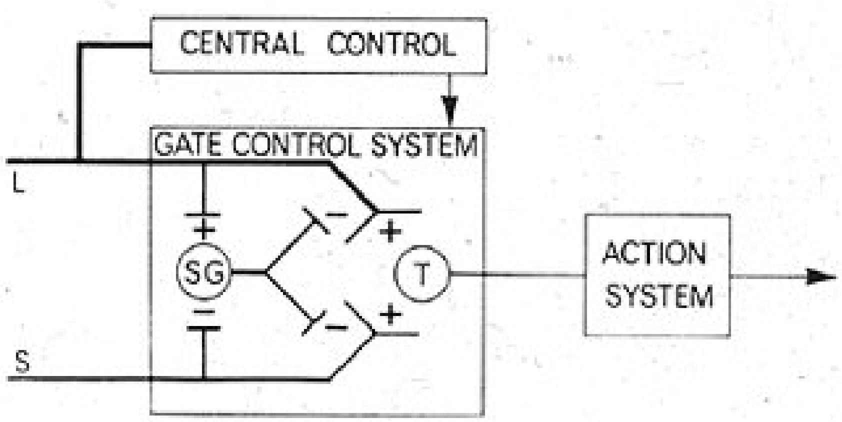
Afferent pain-receptive nerves are responsible for bringing pain signals to the brain. These nerves are comprised of two kinds of fibers. The first fiber is a fast, thick, and myelinated "A-delta" fiber that carries messages quickly with intense pain. The second is a small, unmyelinated, slow "C" fiber that carries the longer-term throbbing and chronic pain signals. Large-diameter A-beta fibers are nonnociceptive (do not transmit pain stimuli) and inhibit the effects of firing by A-delta and C fibers. The peripheral nervous system has centers which regulate pain stimuli. Areas in the dorsal horn of the spinal cord that are involved in receiving pain stimuli from A-delta and C fibers, called laminae, also receive input from A-beta fibers. The nonnociceptive fibers indirectly inhibit the effects of the pain fibers, 'closing a gate' to the transmission of their stimuli. In other parts of the laminae, pain fibers also inhibit the effects of nonnociceptive fibers, 'opening the gate'.[3]

An inhibitory connection may exist with A-beta and C fibers, which may form a synapse on the same projection neuron. The same neurons may also form synapses with an inhibitory interneuron that also synapses on the projection neuron, reducing the chance that the latter will fire and transmit pain stimuli to the brain. The C fiber's synapse would inhibit the inhibitory interneuron, indirectly increasing the projection neuron's chance of firing. The A-beta fiber, on the other hand, forms an excitatory connection with the inhibitory interneuron, thus decreasing the projection neuron's chance of firing (like the C fiber, the A-beta fiber also has an excitatory connection on the projection neuron itself). Therefore, depending on the relative rates of firing of C and A-beta fibers, the firing of the nonnociceptive fiber may inhibit the firing of the projection neuron and the transmission of pain stimuli.[4]

The gate control theory explains how stimulus that activates only nonnociceptive nerves can inhibit pain. The pain seems to be lessened when the area is rubbed because activation of nonnociceptive fibers inhibits the firing of nociceptive ones in the laminae. In transcutaneous electrical stimulation (TENS), nonnociceptive fibers are selectively stimulated with electrodes in order to produce this effect and thereby lessen pain.[5]

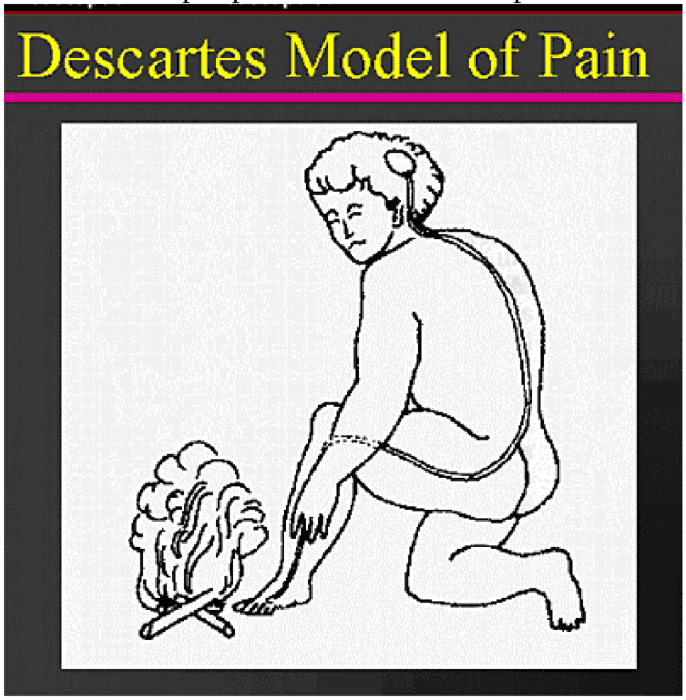
One area of the brain involved in the reduction of pain sensation is the periaqueductal gray matter that surrounds the third ventricle and the cerebral aqueduct of the ventricular system. Stimulation of this area produces analgesia by activating descending pathways that directly and indirectly inhibit nociceptors in the laminae of the spinal cord. It also activates opioid receptors, which are contained in parts of the spinal cord.[6]

Afferent pathways interfere with each other constructively, so that the brain can control the degree of pain that is perceived, based on which pain stimuli are to be ignored to pursue potential gains. The brain determines which stimuli are best to ignore over time. Thus, the brain controls the perception of pain directly, and can be trained to turn off or lessen pain signals. This understanding led Melzack to point out that pain is in the brain and he opined that the brain should be the focus of the future of pain research.



The Gate Control Model. Large-diameter afferent fibers (L) stimulate the substantia gelatinosa (SG) and the transmission cells (T) in lamina 4. The SG cells reduce the membrane potential of afferent terminals, thus producing presynaptic inhibition. Small-diameter afferent fibers (S) also stimulate the transmission cells but inhibit SG cells and thus turn off the existing presynaptic inhibition.[7]

Melzack illustrated his theory with Descartes' well-known picture "Boy with Foot in Fire" seen below. The treatment of pain by surgical transection of peripheral nerves and/or spinal cord was derived from this theory.



Descartes' *Boy with Foot in Fire*.

According to Melzack and Wall's gate control theory, large fiber afferents modulate the pain transmission of the small nociceptive fibers through a gateway. The substantia gelatinosa in the dorsal horn of the spinal cord was the proposed location of the gate that modulated the synaptic transmission of nerve impulses from peripheral fibers to central cells (refer to the figure below). This theory predated the advent of transcutaneous electrical nerve stimulation and spinal cord stimulation to modulate the transmission of pain in the spinal cord. Other methods of modulation include psychology and hypnosis. [8]

Melzack's Neuromatrix Theory

In the 1990s, Melzack introduced a theory involving the integration of three separate pathways in the brain, with a concept called the "neurosignature". He proposed that a large number of interconnected neurons, called a "neuromatrix", exist in everyone. This neuromatrix analyzes the sensory information and gives perception of sensation. The neurosignature then comes into play and tells the brain that the perceptions of sensation are from the "self", in other words the neurosignature tells the brain that your arm is YOUR arm and not someone else's. Although his work is continually updated and revised, it continues to influence the research and management of pain.[9]

Melzack's neuromatrix theory of pain posits that pain is a multidimensional experience produced by characteristic "neurosignature" patterns of nerve impulses that are generated by a widely distributed neural network -- the "body-self neuromatrix" -- in the brain. It is a parallel and serial process that can be thought of as an expansion of the central control processes in the original gate control theory. The central control processes would encompass cognitive-evaluative, motivational-affective, and sensory-discriminative systems. [10]

Multiple factors act on the neuromatrix and contribute to the output "neurosignature." These factors include sensory inputs; visual and other sensory inputs that influence cognitive interpretation; phasic and tonic cognitive and emotional inputs; intrinsic neural inhibitory modulation; and the activity of the stress-regulation system (endocrine, autonomic, immune, and opioid systems). Pain may be triggered by sensory inputs, but may also be generated independently of them. Therefore, pain could be produced by the output of a widely distributed neural network in the brain, rather than directly by nociceptive stimuli.[11]

An example Dr. Melzack gave of the central process of pain is phantom limb pain in patients with spinal cord transection. In this scenario, the pathway for pain transmission has been severed, yet the patient feels pain in the insensate limb. He also presented the case of a woman who had the congenital absence of both legs and an arm, and felt phantom limb pain in the missing limbs. Phantom limb pain is commonly experienced by quadriplegics who have lost large portions of the spinal cord and cannot possibly be receiving any messages from it. This phenomenon is explained by Melzack's notion of the body-self neuro-matrix.. The sensation of a foot that is burning, for instance, has become separated from the foot that is still attached to a useless leg dangling into space - the disconnection actually frees the neurological part of the matrix to "invent" a body. Different parts of the brain then perceive the whole body in different ways, and these are more free to clash if real signals from a real body are not disciplining them all with a common external experience.[12]

At present, pain theorists continue to explore the genetic, endocrine, and immune systems, all of which may contribute to the neuromatrix. Pain theorists have offered explanations of pain that entail both physiological and psychological components. The neuromatrix theory is still evolving and the brain functions and mechanisms in this theory need to continue to be further explored and scrutinized. [13]

Pain Theory Experimentation

Research records indicate that pain experimentation was performed on dogs that were raised in confined environments. When the dogs were released, they were excited, constantly ran around, and required several attempts to learn to avoid pain. When a pain stimulus such as a pinch was introduced, the animals did not take action to avoid the stimulus immediately. This finding seemed to demonstrate that pain is understood and avoided only by experience .Consequently, these experimental observations led to the thought that aversion to pain is not inherent or automatic, and the organism has no way to know what will cause repeated pain without a repeated experience.[14]

Advantages of the Gate Control Theory

Prior to Melzack and Wall's gate control theory, neurochemistry theorists had not taken into account the brain in the discussion of pain. Based upon the classic theory of pain proposed by René Descartes, pain was thought to be simply a direct response to a stimulus. This one- way 'alarm system' pain-pleasure theory was incapable of offering a scientific explanation for phantom limb pain where the pain signal is in fact impossible to receive in the absence of a neurological pain pathway. Conversely, the gate control theory in general, and the neuromatrix theory in particular, provide an explanation for the phantom limb pain phenomenon. Unlike the specificity theory of pain, the gate control theory allows for the dynamic role of the brain in the pain process. Psychological aspects of pain are now seen as integral parts of the process and not just reactions to pain. This has opened the way for psychological approaches to be used for pain treatment and management. Simplistic treatments such as cutting nerve pathways (which often made the pain worse) have been replaced by more realistic and efficacious treatment methodologies.

Consequences of Melzack's Work

In his paper The Tragedy of Needless Pain, Melzack further asserts that "pain is a fundamental human experience, and requires an integrative understanding of that whole experience, and every choice we have made, that has formed our own "gates". He frames the choice to deal with pain or ignore it as moral: if the brain *can* control pain, we who know that *must* make use of that capacity, and in turn take control of pain on a species level - only by doing so can we achieve control of the larger causes of all of the pain that humans cause each other by carelessness, hatred, and failures of empathy - which might extend beyond humans."[15]

Melzack's work has led to advancements in the assessment of pain. Melzack was a professor at McGill University and thus the name of his McGill Pain Questionnaire. According to Melzack pain is an entirely personal experience, and therefore it is difficult to accurately measure. Melzack's McGill Pain Questionnaire asks a number of directed questions to assess and categorize the experience of pain. Melzack collected approximately 200 words used pain by his patients to describe pain. A "burning pain" for instance, can be described as "hot" or even "searing". "Throbbing" becomes "palpitating". Some describe sensory aspects of pain, and others, such as "excruciating", describe emotional experiences. More intense pain generally requires more words to describe. A patient, in the questionnaire, picks one of 20 words to describe their pain, and assign it a point on a scale. Melzack's work was the first to actually qualify and define types of pain - something impossible prior to the gate control theory. An advantage of this was to make it easier to determine the difference between organic pain and non-organic pain - the latter being entirely treatable by psychological means.[16]

[1] www.emotionalprocessing.org.uk
[2] www.emotionalprocessing.org.uk
[3] Melzack R. Evolution of pain theories. Program and abstracts of the 21st Annual Scientific Meeting of the American Pain Society; March 14-17, 2002; Baltimore, Maryland. Abstract 102.
[4] Melzack R. Evolution of pain theories. Program and abstracts of the 21st Annual Scientific Meeting of the American Pain Society; March 14-17, 2002; Baltimore, Maryland. Abstract 102.
[5] Melzack R. Evolution of pain theories. Program and abstracts of the 21st Annual Scientific Meeting of the American Pain Society; March 14-17, 2002; Baltimore, Maryland. Abstract 102.
[6] Melzack R. Evolution of pain theories. Program and abstracts of the 21st Annual Scientific Meeting of the American Pain Society; March 14-17, 2002; Baltimore, Maryland. Abstract 102.
[7] Melzack R, Wall P. Pain mechanisms: a new theory. Science. 1965;150:975. 1965 American Association for the Advancement of Science, www.sciencemag.org
[8] Melzack R. Evolution of pain theories. Program and abstracts of the 21st Annual Scientific Meeting of the American Pain Society; March 14-17, 2002; Baltimore, Maryland. Abstract 102.
[9] www.emotionalprocessing.org.uk
[10] Melzack R. Evolution of pain theories. Program and abstracts of the 21st Annual Scientific Meeting of the American Pain Society; March 14-17, 2002; Baltimore, Maryland. Abstract 102
[11] Melzack R. Evolution of pain theories. Program and abstracts of the 21st Annual Scientific Meeting of the American Pain Society; March 14-17, 2002; Baltimore, Maryland. Abstract 102
[12] Melzack R. Evolution of pain theories. Program and abstracts of the 21st Annual Scientific Meeting of the American Pain Society; March 14-17, 2002; Baltimore, Maryland. Abstract 102
[13] Melzack R. Pain and the neuromatrix in the brain. J Dent Educ. 2001;65:1378-1382
[14] www.en.wikipedia.org/wiki/gate-control-theory
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[16] www.en.wikipedia.org/wiki/gate-control-theory