Pain: Cognitive and Contextual Influences

Recent pain research has been greatly influenced by the gate control model of pain presented in 1967 by psychologist Ronald Melzack and neuroscientist Patrick Wall. They suggested that both physiological and clinical data, as well as everyday experience, run counter to the classical view that pain simply arises from overstimulation of the somatosensory system. Anatomical, physiological, and psychological evidence point to a complex interaction of both peripheral and central information in responding to noxious stimuli.

Melzack and Wall also noted that the amount of pain after an injury is greatly influenced by contextual factors. An athlete, soldier, or worker may suffer a severe wound yet not report pain until long after the event, likely because the individual's attention was focused upon some vital task when the injury occurred.

The gate control theory, emphasizing parallel ascending and descending effects within the nervous system, suggested that pain is not a single sensation. Rather, it has several distinct dimensions that Melzack and Kenneth Casey called (1) the sensory-discriminative system, (2) the motivational-affective system, and (3) the cognitive-evaluative system. Put simply, the first deals with identifying the location and intensity of the pain (how it feels), the second with our emotional response to that sensation (how it makes us feel), and the third with our interpretation and response to that event (how we think about it and act).

Consequently, pain research and management must deal with sensory, emotional, and cognitive mechanisms. This is reflected in pain measurement techniques, in functional imaging studies, in medical interventions, and in psychological approaches to pain reduction. This entry discusses some of the techniques used to measure pain, behavioral measures that illustrate how pain is influenced by cognition, and how pain is influenced by context effects.

Pain Measurement

Melzack and Warren Torgerson studied the basic dimensions of pain through an analysis of the English language. They came up with a list of 102 adjectives commonly used to describe elements of the pain experience. A group of subjects were asked to place these terms into categories, yielding three major classes. The classes were words that described the sensory qualities of the experience (such as their temporal, spatial, and thermal characteristics), the affective qualities (words such as *tiring, frightful,* or *wretched*), and evaluative ones that descried the overall character of the pain experience (such as *annoying, miserable,* or *unbearable*).

These terms were scaled for their intensity or severity, and an instrument called the McGill Pain Questionnaire (MPQ) was developed to measure the overall pain impression as well as its distinct components. The MPQ (now translated into more than 20 languages) has become widely used both for pain assessment (measuring, for example, changes in score after treatment) as well as diagnosis (because certain terms are used much more often by individuals suffering from one pain syndrome or another).

Words, of course, are only one means by which people can describe their level of pain or suffering. Numbers are another method; marks along a line (the visual analog scale) are a third. Richard Gracely devised several scales in which individuals were asked to report their pain intensity and distress separately. Although it is generally the case that increases in intensity are accompanied by increases in unpleasantness, that is not always so. Researchers sometimes remind people being asked to rate the two components that soft music that they dislike is likely to be markedly more unpleasant than loud music performed by a favorite band.

Groups of dental patients were administered diazepam, a tranquilizing antianxiety drug, and asked to report the intensity or unpleasantness of a noxious stimulus. The drug lowered only the affect or distress rating, leaving the intensity rating untouched. The dimensions did not change in unison. This is somewhat reminiscent of the descriptions given by psychiatric patients who had undergone prefrontal lobotomies. Those who had a long-standing pain problem reported that the "little pain" (the sensation) was still there but the "big pain" (the distress and suffering) had been attenuated or eliminated.

Functional Neuroimaging of Pain

Given the subjective nature of pain, it is impossible to obtain a truly "objective" measure of the experience. There is no "pain thermometer." In the final analysis, it is the individual's report of his or her experience that determines the evaluation of symptoms and response to them. Such reports are cortically determined. Although there are fascinating issues related to the receptors, nerve fibers, spinal pathways, and subcortical regions that convey signals generated by noxious inputs, it is the cortical regions that mediate, either within a complex regional neural matrix or through descending influences, such critical variables as context, expectations, pain beliefs, attention, and coping mechanisms in developing the pain percept.

Recent technical advances have permitted us to combine psychophysical and neuroimaging techniques in order to investigate the neural correlates of pain, making it possible to see how emotional and cognitive factors influence neural representations of the pain experience. Not surprisingly, a large number of brain regions have been implicated in the experience of even acute pain (chronic pain being even more complex). These include the primary and secondary somatosensory cortices as well as the insular, anterior cingulate, and prefrontal cortices (accompanied, sometimes, by motor cortices, the posterior parietal cortex, and the posterior cingulate), plus such subcortical regions as the thalamus, amygdala, hippocampus, cerebellum, and medulla.

Although the specifics change somewhat from study to study, at least partially due to methodological differences, there is general agreement that the sensory-discriminative aspects of pain perception, such as judgments about intensity, duration, quality, and location, are subserved within the SI and SII regions of the somatosensory cortex, and the motivational-affective and cognitive-evaluative aspects of pain tend to reflect activity in the insular cortex (IC), anterior cingulate cortex (ACC), and prefrontal cortex (PFC).

It is in these latter areas that unpleasantness, suffering, and evaluation of the pain experience are mediated. The IC is seen as important in emotional responses to noxious input, and the ACC is considered to play a critical role in both emotion and cognition, particularly with respect to anticipation and expectation of pain and the interplay of attention and response planning. It is here where activity is reduced by hypnotic suggestions for the reduction of pain.

The PFC may act as a sort of supervisory attention system or "pain control center," integrating emotion and such cognitive components as perceived control over a painful event. Interestingly, not all studies of brain activation by noxious signals show activity in the PFC in healthy subjects, although chronic pain conditions are often associated with increases there, as well as in the somatosensory cortex and the ACC.

Activation of these regions does not necessarily require noxious stimulation. A series of recent studies have shown that observing films showing another individual receiving painful stimuli may trigger a sort of empathic reaction, causing activity in the ACC and IC not unlike that created by painful stimulation itself. In another study, when subjects observed pain from the faces of chronic pain patients, activations in the IC, ACC, and parietal lobe in the observer's brain correlated with their estimates of the intensity of observed pain. Even imagining pain through hypnotic induction activates the thalamus, ACC, IC, PFC, and the parietal cortex.

Other cognitive tasks can modulate the cortical response to noxious inputs. The anticipation of pain, perhaps by creating hypervigilance to impending threat, increases activity in the emotion-processing network consisting of the IC, ACC, and amygdala. Just the task of providing a discomfort rating increases brain activity in the cortical pain matrix. Such effects are bidirectional. Many of these regions show decreases in activity when subjects receive placebo analgesia. Particularly noteworthy is the finding that these changes in neural response occur during the stimulus itself, rather than somewhat later when pain reports are made.

Behavioral Studies of Pain and Cognition

Cognitive effects can modulate pain in two directions. That is, such factors as anticipation of pain, attention, anxiety, and maladaptive thoughts increase pain and suffering, yet distraction, hypnotic suggestion, placebo administration, cognitive therapies, and induction of positive mood states can reduce both experimentally induced pain and reports of pain and distress created by chronic pain syndromes.

In laboratory studies, cognitive strategies such as engaging in mental imagery, interpreting the stimulation as something else, or diverting attention to environmental cues have often been shown to enhance pain threshold or tolerance or reduce pain ratings to a constant stimulus. The literature in this field is murky, likely due to large differences in pain induction methods (generally various forms of intense heat, cold, pressure, or electrical pulses), instructions, content, and nature of the coping strategies used and measures taken. There are differences even in the general nature of attentional control; distraction can diminish pain, but so too can deliberate focus upon the part of the body receiving stimulation, in which subjects are told to attend to the information coming from the site while ignoring the emotional aspects (redefinition).

It is not only the case that attention affects pain; the reverse is also true. Pain often serves as a warning of impending danger, and neglect of this signal is highly maladaptive. In the laboratory, pain has been seen to significantly disrupt performance on various attentionally demanding tasks involving perception, learning, memory, vigilance, and movement. Likewise, pain patients often show impairment in everyday cognitive activities (things such as recall of people, places, and activities or the use of spoken and written language), a phenomenon some fibromyalgia patients label "fibrofog."

Cognitive response styles characterize how individuals deal with potentially threatening situations. Some people are prone to use adaptive thought patterns (coping). Others use maladaptive coping styles (catastrophizing) in which they expect negative outcomes, show high levels of fear or hypervigilance to threat, misinterpret events, and are impaired in their ability to divert attention away from their pain. Psychological tools (the Coping Strategies Questionnaire or the Pain Catastrophizing Scale) can measure the tendency to engage in adaptive or maladaptive thought patterns; others (the Pennebaker Inventory of Limbic Languidness and the Modified Somatic Perception Questionnaire) assess attention to physical sensations and symptoms.

Contextual Effects in Pain Judgments

Pain judgments are not made in a vacuum. There is considerable evidence that descriptions and ratings of pain are influenced by contextual information about the environment, circumstances, and anticipation of the noxious stimulus. Dental and medical students are advised to notify patients just before administration of a procedure (such as an injection) is likely to become painful, so that the patient is able to prepare for the event. Even knowledge that one can influence the course of the clinician's action, through finger signs or grunts, helps patients to attenuate both negative affect and pain.

Gary Rollman found that pain ratings are influenced by other stimuli in the presentation set. A stimulus will be judged as less painful when presented in the same session as a stronger one than it is when paired with a weaker stimulus. This adaptation level effect, which is similar to ones found in other modalities, highlights the fact that pain judgments are relative rather than absolute.

In the case of pain patients, judgments regarding the painfulness of experimentally induced discomfort are often lower than those provided by individuals who are pain free. Endogenous pain serves as an anchor or comparison point by which newly added stimuli are judged. This adaptation level model is also applicable to at least some instances of a phenomenon known as diffuse noxious inhibitory controls (DNIC), in which strong, tonic pain at one part of the body reduces the response to a phasic pain stimulus presented elsewhere. Seen in both humans and lower animals, the DNIC effect is at least partly mediated by endogenous opiates, but cognitive comparisons are also fundamental factors.

Mental states induced by placebo instructions are also capable of releasing the body's endogenous opiates, such as endorphin. Thoughts and expectations have physiological effects; placebos are not biologically inert. For example, dental patients who are given placebos show reductions in pain ratings to induced stimuli, which are reversed by the administration of naloxone, an opiate antagonist drug that blocks the receptor sites where endorphins bind. Moreover, neuroimaging studies have revealed that decreases in pain ratings after administration of a placebo are accompanied by decreases in activity in the ACC, IC, and PFC.

Reflections

The literature makes clear that pain is not simply an overstimulation of the central nervous system. The richness of the pain experience, the multiplicity of neural sites that respond to noxious signals, and the amplification or diminution of pain created by emotions, thoughts, and expectations make clear that pain is essentially constructed by a cascade of peripheral and central events involving ascending and descending neural networks. This distributed pain network evaluates incoming information within both a bottom-up and a top-down context determined by memories of previous events, current emotional states, pathology, genetics, and many cognitive variables. There has been considerable success in utilizing cognitive-behavior therapies, which address thoughts, beliefs, appraisals, attitudes, and coping strategies, to ameliorate both acute and chronic pain states. While cognitions can enhance pain and suffering, they can also be utilized to diminish those distressing conditions.

Gary B. Rollman

See also Attention: Cognitive Influences; Brain Imaging; Emotional Influences on Perception; Multimodal Interactions: Pain–Touch; Pain: Assessment and Measurement; Pain: Neuromatrix Theory; Pain: Physiological Mechanisms; Pain: Placebo Effects

Further Readings

Apkarian, A. V., Bushnell, M. C., Treede, R. D., & Zubieta, J. K. (2005). Human brain mechanisms of pain perception and regulation in health and disease. *European Journal of Pain, 9,* 463–484.

Eccleston, C., & Crombez, G. (1999). Pain demands attention: A cognitive-affective model of the interruptive function of pain. *Psychological Bulletin, 125,* 356–366.

Lautenbacher, S., Prager, M., & Rollman, G. B. (2007). Pain additivity, diffuse noxious inhibitory controls, and attention: A functional measurement analysis. *Somatosensory & Motor Research, 24,* 189–201.

Rollman, G. B. (1992). Cognitive effects in pain and pain judgments. In D. Algom (Ed.), *Psychophysical approaches to cognition. Advances in psychology* (Vol. 92, pp. 517–574). Amsterdam: North-Holland.

Tracey, I., & Mantyh, P. W. (2007). The cerebral signature for pain perception and its modulation. *Neuron, 55,* 377–391.

Wiech, K., Ploner, M., & Tracey, I. (2008). Neurocognitive aspects of pain perception. *Trends in Cognitive Sciences, 12,* 306–313.

volume 2

encyclopedia of **perception**

e. bruce goldstein
university of pittsburgh
university of arizona

SAGE
Los Angeles | London | New Delhi
Singapore | Washington DC

A SAGE Reference Publication