

Race, Ethnicity, Gender, and Age as Dimensions of Pain

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The Experience of Pain

As every doctor knows, the experience of pain is exceedingly complex. It strikes a patient as the final common pathway that originates in a complexity of anatomical, physiological, psychological, and sociological causes, so that a particular level of suffering often does not correspond in any straightforward way or any specific level of physiologic-anatomic pathology as such.

In what follows, I advocate that for success in treatment, a clinical diagnosis of pain needs to expand the concept of a pathologic lesion to include psychological; emotional; intellectual; cultural; and societal components. To do so is to acknowledge that even when an anatomic-physiological disorder cannot be cured, and the associated pain remains intractable, psychosocial aspects of the lesion can be addressed, often with considerable success, so that suffering can be lessened or even eliminated, and patients can return to the normal activities of daily living in spite of an otherwise untreatable lesion. Chronic pain is not just a directly mediated sensation. It is a complexly created experience of suffering.

Pain and the avoidance of pain are ubiquitous in human experience. Pain is both a physical sensation of discomfort and an emotional experience of suffering. Every human activity must take account of the need to avoid or minimize that sensation of discomfort, distress, or agony we call pain. For most people, avoiding or minimizing pain takes place outside of conscious awareness. But everyone also experiences times when pain commands attention.

People who are in pain usually experience it as an acute but time-limited problem, even if it is severe. They take steps to diminish and remove the noxious sensation. Little do they care for the sage observation that acute pain serves a good purpose because it warns the victim to identify the source, seek care and eliminate it. Rather, when it has passed, those healed of acute pain return to their lives, often with scars (psychological or physical), but free at least of the dominating, often overwhelming awareness that a part of the body hurts.

Chronic pain is another matter. No longer can one speak of the benefit of pain as a protective mechanism. When it is bad, it nags and grinds away relentlessly, stealing attention from the joys of life, diverting energy from work and social life, until for those who suffer the most, only the experience of pain persists as the overwhelming reality of existence.

Because pain is ubiquitous and obnoxious, healers and sufferers alike have struggled for millennia to gain understanding and control. It has not been easy. As poet Emily Dickinson reminds us, the experience is difficult to analyze while it is felt, and equally difficult to remember after it has eased. Throughout most of modern history, scientific efforts to understand and control pain have directed attention to underlying anatomy and physiology. Only in recent decades have behavioral scientists, especially psychologists and anthropologists, made significant progress in directing scientific attention to nonbiologic factors in the experience.

The Anatomy and Physiology of Pain

Turning first to biologic mechanisms, much can now be said about mechanisms of pain. In our time, research in this area is on a fast track, as the remarkable capabilities of scientific technology yield new understandings of biochemical processes at an astonishing rate. While one can still summarize pain mechanisms in rather straightforward ways, it must be acknowledged that the more we learn, the more we realize that the process is extraordinarily complex. As one result, the long search for a pain center in the brain had to be abandoned (Talbot, et al., 1991). No such center exists. Instead, complex neural networks interact peripherally, as well as centrally, at all levels of the nervous system and in all directions. Dr. Clifford Woolf, of Harvard Medical School, finds it useful to distinguish two kinds of pain that only roughly conform to the distinction between acute and chronic pain.

The first type of pain, physiologic, results when injurious or potentially injurious stimuli trigger high-threshold nociceptors. These include small diameter, myelinated, fast-conducting A-delta fibers located in peripheral nerves and dorsal nerve roots. A-delta fibers identify well-localized, sharp pain sensations. Nociceptors also include C-fibers, the large, unmyelinated, slow-conducting fibers that transmit dull, burning, poorly localized pain sensations. These fibers, located in peripheral nerves and dorsal nerve roots (with a few in ventral nerve roots), are the most numerous of the fibers that transmit pain.

Yet another kind of nociceptive neuron, the A-beta, is composed of large, myelinated fibers that conduct much faster than C-fibers. Located in peripheral nerves and dorsal spinal nerve roots, their exact function in pain transmission is still not completely understood. It appears that they function above all to modulate pain transmission by other neurons. This occurs in part to the extent that they release endorphin, which exerts an analgesic effect. In addition, in the *substantia gelatinosa* of the dorsal horn, A-beta fibers, as well as A-delta fibers, stimulate interneurons, which produce presynaptic blocking of C-fibers. These A-fibers also produce postsynaptic inhibition of neurons projecting to higher centers in the central nervous system. The latter inhibition is enhanced by descending signals from the nucleus *raphe magnus* of the brain that travel along the serotonergic descending inhibitory pathway of the dorsolateral funiculus, modulated along the way by endorphins secreted in the periaqueductal gray area of the midbrain. This blocking of C-fibers by A-fibers, plus descending neurons at the level of the dorsal horn, has been described as the "gate control" theory by Melzack and Wall (Melzack and Wall, 1982). Physiologic pain serves as a protective mechanism, since it triggers flexion and escape reflexes.

The second kind of pain, allodynia (also known as neurogenic inflammation), occurs after injury. Rang, Bevan and Dray identified hyperalgesia mediated by nerves that normally did not result in pain, but became sensitized for pain transmission when bathed in biochemicals released in their vicinity (Rang, et al., 1991). In the injured area of primary hyperalgesia, tissue damage activates the sympathetic nervous system and local inflammatory responses. Mast cells and neutrophils release histamine and leucotriene B-4 in the area. In addition, peptides synthesized in the dorsal root ganglia are transported peripherally to the terminals of the sensory fibers, where they are released into the local environment. These include substance P; calcitonin gene-related peptide (CGRP); vasoactive intestinal polypeptide (VIP); and somatostatin. These peptides enhance pain sensation by means of direct effects on endothelium; epithelium; smooth muscle; and the immune system. They also transport centrally to the dorsal horn, contributing to enlargement of receptive fields, increased spontaneous activity, and lowered thresholds to mechanical stimulation. Other chemical irritants that contribute to allodynia include lactic acid; potassium ions; prostaglandin E-2; glycosaminoglycans; 5-hydroxytryptamine (serotonin); and bradykinin. Out of this potpourri of stimulating and modulating chemicals, noxious sensations eventually ascend the contralateral

spinothalamic tract to the thalamus and the cerebral cortex, where they cause pain and suffering (Mapp and Kidd, 1994).

In noninjured areas, pain may result from so-called secondary hyperalgesia. Often this occurs at some distance from the wound itself. The A-beta afferent nerves, which normally signal touch and vibration, become pain mediators, apparently as a response to sensitization in the dorsal horn of the spinal cord. Relying on a rat model, Thompson, King and Woolf concluded that this so-called mechanical allodynia required prior sensitization by A-delta and C-fibers (Thompson, et al., 1990). By means of the temporal summation of slow synaptic potentials, prior sensitization produces a prolonged depolarization of neurons of the dorsal horn. It appears that this, in turn, activates post-synaptic amino acid receptors, particularly N-methyl D-Aspartate (NMDA) receptors by glutamate and neurokinin receptors by tachykinins and CGRP. This period of prolonged depolarization also permits A-beta fibers to activate dorsal horn neurons that normally would not be responsive to their input.

Secondary hyperalgesia lasting more than a few minutes results from further metabolic changes. Prolonged depolarization, for example, leads to changes in intracellular calcium, which in turn causes protein kinase C to activate, leading to phosphorylation of the NMDA receptors. Still further in this chain reaction, the magnesium blockade of the NMDA receptors is additionally reduced, resulting in additional slow depolaration. Prolonged depolarization can also alter the gene expression of dynorphin and other proteins. These feed-forward mechanisms maintain pain. Chong argues that even this allodynia has a protective function, however, since it encourages protection of the injured area to permit healing (Chong, 1993). What is still unclear is why chronic pain occurs in some cases, since the usual outcome for prolonged allodynia is eventually for this chain reaction with feedback and feedforward loops to quiet down and return to normal. Chronic pain is often found where no nerve damage can be identified.

Pain and Human Biological Diversity

Human responses to pain are highly variable, as we shall see. That variability, however, does not appear to reflect populational differences in the anatomy and physiology of pain in terms of race or gender. It is often assumed that blacks, whites, Asians, Melanesians, Polynesians, and Amerindians constitute large, geographic races. Such assumptions are scientifically unjustified (Montagu, 1964). The misleading concept of large-scale geographic races as constituting biologic units or entities arises out of the simplistic assumption that populations with the same skin color (together with hair form, shape of the nose, and a few other physically noticeable traits) represent biological categories of people (shared genotypes). Such groups are mistakenly thought to be genetically uniform within each so-called race and its associated geographic area, and clearly differentiated from other races in much of their DNA (genome). In fact, other traits such as blood antigens, pulmonary adaptations to high-altitude hypoxia, or stature vary independently of skin color as clines that cut across so-called racial boundaries. The concept of race, then, is scientifically and socially ill-conceived, and not likely to serve well to discriminate biological variation on a populational level (Anderson, 1995). To the extent that efforts have been made to demonstrate differences in the anatomy and physiology of pain related to race, all have failed.

As an illustration, one large study enrolled 40,000 research subjects to test for tolerance of deep pain resulting from pressure on the Achilles tendon. That study appeared to demonstrate that whites had the highest tolerance, Asians the lowest, with blacks intermediate. However, this was not in actuality a study of pain perception (and hence of pain biology), but of the ability to tolerate the pain. How much pain one can tolerate is better understood as a psychologically and culturally influenced response. What one person finds intolerable, another finds merely noticeable, even though they may well be experiencing the same sensation in physiologic terms (Woodrow, 1972). It

is reasonable to assume that races do not differ in the way pain mechanisms work (Anderson and Anderson, 1994).

The same applies to gender differences. Although males and females may differ in how they respond to pain, it has never been shown that these differences reflect differences in biologic mechanisms. Rather, they appear to reflect cultural expectations and psychological predispositions.

Age may constitute the one important exception to the generalization that the physiologic capacity to sense pain is shared equally in all human populations. It is possible that the still immature nervous system of infants includes immaturity of nociception, but little can be said about that at this time. Certainly, it was an error to assume that infants did not experience pain on circumcision (Walco, et al., 1994). There is also evidence, much of it anecdotal, that pain sensation declines somewhat with aging (Sherman and Robillard, 1964).

Pain and Ethnicity

If one accepts that the mechanisms of physiologic pain and allodynia are the same for all populations, then one is left with culture as the major basis for explaining differences in pain experiences. Certainly, differences in the pain experience can be quite dramatic. A team of medical anthropologists at Harvard University makes this point in the following paragraph describing diversity in the categories, idioms, and modes of pain experience:

Professor Emiko Ohnuki-Tierney, anthropologist at the University of Wisconsin at Madison, for example, describes complaints among the Sakhalin Ainu culture of Japan as including "bear headaches" that "sound" like the heavy steps of a bear; "deer headaches" that feel like the much lighter sounds of running deer; and "woodpecker headaches" that feel like a woodpecker pounding into the trunk of a tree. Ots describes a common experience of headache among Chinese as characterized by a painful dizziness or vertigo - a complaint that is an embodiment of the traditional Chinese medical category of imbalance as the proximate cause of ill health. Researchers V. Abad and E. Boyce report that Latinos in North America distinguish *dolor de cabeza* (headache) and *dolor de cerebro* (brainache) as two distinctive experiences and disorders. ... O. Ebigbo indicates that Nigerians complain of a wide range of specific pains... "it seems as if pepper were put into my head," "things like ants keep on creeping in various parts of my brain," or "by merely touching parts of my brain it hurts" (Kleinman, et al., 1992: 1-2).

We have long known that the subjective experience of pain is powerfully contoured by culture. Mark Zborowski, an anthropologist, demonstrated this between 1951 and 1954, in a study of responses to pain among men in a VA hospital in New York (Zborowski 1969). He found that "old Americans" (OA) accepted pain without complaint. (OAs were defined as having lived in the new world for so many generations that they no longer had any ties with their countries of origin. Irish Americans in his study also accepted pain without complaint. The two groups differed greatly in how they experienced the meaning of pain, however.

To understand the pain behavior of the OA, one needs to distinguish private from public pain. In private, an OA left alone may collapse into tears, but never in public. The OA tended, therefore, to withdraw in the face of strong pain. In the hands of doctors and nurses, however, to admit to pain was permitted because the professional situation transformed complaints into purposeful discussion. Possessing a mechanistic attitude to the body and its function, the OA had considerable faith in the abilities of doctors, and tended therefore to be fairly optimistic about ultimate outcomes.

Irish-American patients, equally uncomplaining when in pain, differed from OAs in how they felt

about their illnesses. They lacked the optimism of OAs. In public, an Irish patient masked pain; but this was also done when talking to medical practitioners. This patient articulated pain concerns very ineptly, mumbling and bumbling along. What a surprise, since under other circumstances the Irish can be expected to display notable loquacious skill. The Irish-American felt helpless, guilty about becoming ill, and very pessimistic about the future.

Zborowski also described two kinds of patients who were very different from the stoic OA and Irish. He demonstrated that Italian-American and Jewish-American veterans tended to display highly emotional responses to pain: They groaned, cried and complained; they lay in wait to provide to any and all a description in redundant detail of how they suffered. They shared a cultural trait that permitted vivid expressions of pain, yet their pain behavior rested on very different emotional foundations.

The Jewish patient tended to experience a future-oriented anxiety. Pain was taken as a frightening warning of ultimate possible doom. This patient needed reassurance from the doctor, who found it almost impossible to evade listening to a recital of complaints that seemed endless. But this patient, skeptical toward the doctor, was reluctant to take prescribed medications. He worried that the pills might only provide temporary improvement at the cost of disguising symptoms in a way that could mislead physicians. He feared becoming addicted to pain-killing drugs. Experiencing pain and suffering, it was the suffering that overwhelmed him. Jewish pain, as identified in this study, tended to be associated with powerful existential concerns and with ultimate escatological issues.

The Italian-American was equally vocal when in pain. In place of skepticism, however, this patient showed great trust in doctors and hospitals. In place of a Jewish future orientation, the Italian experienced a present-oriented apprehension. The focus was on pain as such. In complete trust, the Italian accepted, even begged for strong analgesics to quiet the pain. As soon as the pain was gone, this patient became calm, full of smiles, and almost forgetful of the illness.

The work of Zborowski was carried out half a century ago. One would set different standards for this kind of research now. His characterizations resemble stereotypes rather than documented generalizations. Today we would ask for more precision about the nature and quality of the pain. Clarification of ethnicity would be required, since some of these veterans were immigrants while others were third-generation Americans. It would be necessary to search for intra-ethnic variation to break away from the old assumption of cultural uniformity and behavioral predictability within an ethnic group, the error of essentialism. It would be important to control for socioeconomic status, education, age, and other possible sources of variance that might be confused with ethnicity. Not the least, such a study should include women.

In the last few years another anthropologist, Maryann Bates, has done these necessary things. In collaboration with a number of colleagues, she has carried out several projects of quantitative questionnaire-based research in the Pain Control Center at the University of Massachusetts Medical Center. In one of these projects, six ethnic/cultural groups in a patient population characterized by severe chronic pain were selected for study. There were 372 subjects. The first three groups, it should be noted, were younger generations of three of those studied by Zborowski.

Old Americans (100)

Irish (60)

Italians (50)

Hispanics (44)

French Canadians (90)

Polish (28)

Bates, Edwards and Anderson confirm that how people respond to pain is shaped by ethnic heritage. Because the neurophysiology of pain appears to be the same in all racial groups, they are left to conclude that "the association between pain intensity and ethnic identity suggests that experiences, beliefs, attitudes and meanings derived from growing up with these social communities may affect one's reported perception of pain intensity." Consistent with this observation, they found that Hispanics and Italians reported high pain intensity; OAs and Irish subjects were intermediate, while Poles and French Canadians reported the lowest pain intensity scores (Bates, Edwards, and Anderson, 1993: 106).

And what of stereotypes about men and women in pain? Are women the weaker sex who cannot take as much pain as a man? Or are they the stoic sex who suffer more and complain less? It is important to realize that within ethnic categories, Bates and associates found no variation in this study of reported pain intensity based on gender. It is equally important to realize that one cannot reach valid conclusions based on one or a few research reports.

The fact is, men and women may vary, but again, based on cultural rather than biological variables. On the one hand, one study demonstrated that women showed less tolerance for experimental pain than did men (Woodrow, et al., 1972). On the other hand, Bates, et al., in recent work in Puerto Rico, found that men there cope less well with pain than do women. The difference appears to be related to those in male and female cultures. Men suffer depression and a loss of self-esteem, particularly when they are unable to work or be physically active because of their pain. Several Puerto Rican chronic pain subjects attempted suicide or thought about it. Women, suffering the same amount of pain, handled it more successfully. Bates and her fellow investigators conclude that gender role expectations set by ethnic heritage make it more difficult for men than for women to cope successfully with severe chronic pain in Puerto Rican cultures (Bates, et al., 1994).

Based upon research on the body in pain as shaped by different cultures, a caution is in order:

"We stress that ethnic [and gender] stereotyping is as dangerous as inattention to cultural variables. ... There is significant intragroup variation in our study, and others have found insignificant intragroup variation in other populations of pain sufferers. Clinicians must acknowledge that not all patients are alike, that pain does not have the same meaning and significance for different patients, and that patients may exhibit different coping strategies and different responses to pain and to treatments provided. If clinicians are to treat multiethnic [and gender-differentiated] chronic pain sufferers successfully, they must be able to unlock the 'illness reality' of each particular patient, and thereby provide more humane and personal care and treatment." (Bates and Edwards, 1992: 80.)

Culture shapes the experience of pain, but people within a society do not experience their culture in a uniform way. They differ in personality, temperament, life experiences, and social involvements. Nonetheless, one can generalize on a level that allows for variation within the generalization and exceptions to it. To better gauge pain, one must take into account the cultural values, attitudes, and practices (praxis) that tend to be shared as common cultural traits.

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SEPTEMBER 2001