CHAPTER

4

PAIN RESPONSIVENESS

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Given the universal nature of pain, it is surprising how difficult its definition can be. A moment's reflection may, however, bring the difficulty to the fore. Can you define pain without invoking synonyms such as "hurt" or variants such as "painful?"

Further reflection may raise additional problems. Is pain a sensation, a perception, an emotion, or a thought? How should it be compared with the other sensory experiences described in this volume? Does it belong in a unique category or is it part of a continuum with pressure or heat or cold?

Individuals faced with the task of dealing with pain, whether as researchers or clinicians, need to consider these philosophical dilemmas, but they also need to get on with the task of quantifying pain, attempting to alleviate it, and addressing the efficacy of their treatments. Fascinating challenges confront them in each of these endeavors.

While there is no universally accepted definition of pain, there is, in fact, an "official" one, presented by the Subcommittee on Taxonomy of the International Association for the Study of Pain (Merskey, 1986a). Their definition states that pain is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."

Note, first, that the definition emphasizes two components of pain: the sensory and the emotional. While the two are often linked, evidence from laboratory and clinical studies suggests that they can be distinguished and, often, treated separately.

Note, also, that pain is not necessarily linked to tissue damage. In fact,

pain can arise in the absence of any evident physical injury and, in other instances, can persist long after the healing of a wound.

The difficulty in evaluating pain is well summarized by the subcommittee's note that "pain is always subjective." While insurance companies, attorneys, or worker's compensation boards may understandably wish for a "pain thermometer" to distinguish between "real" pain and "unreal" pain motivated by compensation claims (Mendelson, 1984), such a device will never be developed. Individual pain experiences are inextricably linked to early life events, social and cultural conditioning, and the behaviors of role models (Craig, 1986). Pain judgments are often relative, not absolute (Rollman, 1979). As the subcommittee aptly stated, if individuals "regard their experience as pain, it should be accepted as pain."

THEORIES OF PAIN

The classical view of pain, best exemplified by the description offered by Descartes in the 17th century but carried over to more recent anatomical and physiological thinking, sees the pain system as involving a direct path from the skin to the brain. Injury at the periphery is signaled to a central pain monitor. In Descartes's model, the message is likened to a tug on a rope that rings a bell in a church steeple; the more contemporary version of this model speaks of barrages of neural impulses arising from the site of injury and conveyed directly through the central nervous system to a cortical pain center. Both approaches emphasize the notion of *specificity:* specific peripheral receptors, pathways, brain centers, and sensations. Such theories, while attractive, are clearly wrong in the light of present-day knowledge of anatomy, physiology, and clinical data.

An alternative view of the mechanisms underlying pain led to the development of pattern theory. In its extreme form (e.g., Nafe, 1929), it suggested that an individual fiber "could at one time contribute towards the experience of a sensation of touch, and at another towards the experience of pain, cold, or warmth" (Sinclair, 1955). The emphasis was on the temporal and spatial components of the peripheral neural activity—factors such as frequency of action potentials, duration of activity, and the number of responding fibers.

Both pattern theory and specificity theory were challenged by anatomical and physiological data. Specificity theory is shown to be wrong by the lack of receptors, nerve fibers, spinal tracts, or brain areas whose activity invariably gives rise to reports of pain and by the failure of neurosurgical or pharmacological interventions at any of these putative pain units to eliminate pain reliably. Pattern theory is an oversimplification. Nerve endings are not equally sensitive to all cutaneous modalities. As we'll see, evidence shows that there is a high degree of specialization within the peripheral

somatosensory system, such that many nerve fibers respond only to very intense stimuli and are uninfluenced by light touch or moderate heat or cold.

An adequate pain theory needs to consider anatomical and physiological knowledge, clinical data on the causes and treatment of pain, and the influence of psychological factors on pain behaviors. Specificity theory and pattern theory dealt somewhat poorly with the first, very poorly with the second, and wholly ignored the third. A revolution in pain research and treatment was begun in 1965 by the publication of an article entitled "Pain Mechanisms: A New Theory," written by an experimental psychologist, Ronald Melzack, and a neurophysiologist, Patrick Wall. The two, who were then colleagues at the Massachusetts Institute of Technology, introduced an integrative theory, which, as we'll see, was far-reaching in its impact on pain research and management.

THE ANATOMY AND PHYSIOLOGY OF PAIN: ASCENDING SYSTEMS

Three regions require examination: the peripheral receptors and nerve fibers, the ascending tracts within the spinal cord, and the subcortical and cortical areas of the brain (see Fig. 4.1).

Little is known about receptor cells in the skin that might respond to noxious inputs (and thus would be called nociceptors). Von Frey (1895) had suggested that the free nerve endings serve this purpose, while the encapsulated endings such as Pacinian corpuscles, Ruffini cylinders, and Krause end bulbs mediate other somatosensory experiences such as pressure, warmth, and cold. Although von Frey's assignment of specific receptors to specific sensations is certainly wrong, the attention of physiological investigations of the periphery has largely been devoted to studying the afferent nerve fibers rather than the receptors.

Examination of these fibers reveals a wide range of diameters as well as the presence or absence of a myelin sheath. This discussion will emphasize those fibers that convey sensory information, but it should be noted that many of the fibers are sympathetic axons that regulate autonomic functions or motor axons that influence muscles.

The sensory fibers are divided into three distinct groups: the myelinated A-beta and A-delta fibers and the unmyelinated C-fibers. The A-beta fibers, with a diameter of 5 to 20 microns (a micron is one-thousandth of a millimeter, or about one-twenty-five-thousandth of an inch) are relatively large, rapidly conducting, and maximally responsive to weak mechanical stimulation. The A-delta fibers have an intermediate size (1–5 microns), a moderate conduction velocity, and respond best to intense pressure or heat, although some also respond to cold and irritating chemicals. Because of their high threshold

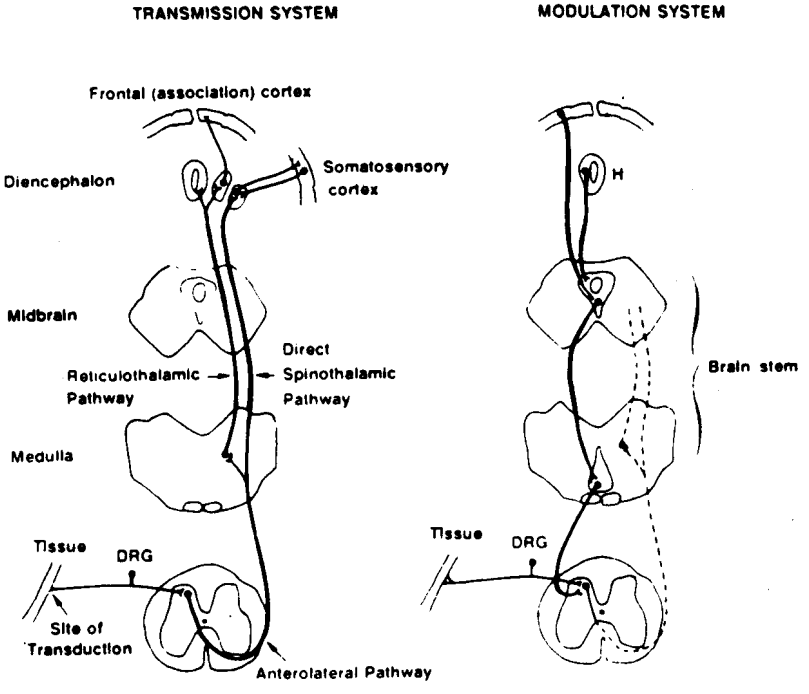

FIG. 4.1. A schematic diagram of the major ascending and descending systems involved in the transmission and modulation of nociceptive information. DRG-dorsal root ganglion; H-hypothalamus (from M. Osterweis, A. Kleinman, & D. Mechanic (Eds.), *Pain and Disability. Clinical, Behavioral, and Public Policy Perspectives;* copyright, 1987, by the National Academy of Sciences, National Academy Press, Washington, DC; reprinted by permission).

and the finding that stimulation of the A-delta fibers often gives rise to a sharp, pricking pain, these afferents have been labeled "myelinated nociceptors."

The narrow diameter (.3 to 2 microns), unmyelinated C-fibers are very slowly conducting. They, too, respond best to noxious levels of stimulation in their rather small receptive fields. Interestingly, they are the most common fiber in most peripheral nerves and, generally, respond only to intense stimulation.

The nature of the stimulus is less important than its intensity: high levels of pressure, heat, cold, or chemicals (such as bradykinin or the prostaglandins that are released from damaged tissue) will induce bursts of activity in these fibers, causing them to be labeled "unmyelinated nociceptors" or "C polymodal nociceptors." Their action seems to be associated with delayed but prolonged experiences of dull or burning pain. In fact, observers presented with repetitive thermal or electrical pulses report two pains: The first is sharp and the second, which may occur as much as a second later, is described as burning or throbbing (Price, Hu, Dubner, & Gracely, 1977). The differen-

tial response time is in keeping with the idea that the first is subserved by the rapidly conducting A-delta fibers, while the second is due to activity in the slowly conducting C-fibers. So, too, is the finding that first pain can be abolished by a pressure block of the myelinated fibers (Price et al., 1977), while the second pain is eliminated by selective blocking of unmyelinated fibers using local anesthetics.

The pain transmission process only begins at the periphery. The capacity to stimulate large and small fibers selectively, to record their electrical activity in human subjects (Torebjörk & Hallin, 1973) and to correlate neural information with subjective reports of quality and intensity provides the opportunity for very elegant psychophysical investigations. Nonetheless, the more central spinal cord and brain mechanisms modulate the activity arising in the sensory fibers and provide the locus for psychological factors to interact with first-order physiological variables mediated by the primary afferents.

The second-order neural pathways are found in the spinal cord. Here, the simplicity of the peripheral coding mechanism gives way to a system of enormous complexity. The spinal cord receives inputs from both nociceptive and nonnociceptive afferents, receptive fields for spinal neurons are often much larger than for nerve fibers, and excitatory influences are balanced by inhibitory ones that arise from both afferent activity and descending influences from the brain stem and cortex (Fields, 1987).

The examination of spinal influences on nociception is focused on the dorsal horn at the rear of the cord. Examination of a cross-section of this gray matter reveals a series of 10 layers or laminae. The A-delta fibers project to laminae I and V, the C-fibers end principally in lamina II, while the larger A-beta fibers terminate in lamina III and deeper.

Nociceptive projection neurons that pass the message to higher brain centers are identified by their ability to respond maximally to noxious stimuli, by their projection to areas known to be involved in pain processing, by the generation of pain experiences when they are activated electrically, and by the reduction of pain when their activity is reduced (Fields, 1987). Such neurons are widely distributed within the spinal cord.

About a quarter of the spinal neurons respond only to noxious stimuli. These analogues of the peripheral nociceptors (which, in fact, project directly to them) are labeled "nociceptive-specific" neurons. They are outnumbered, however, by neurons that receive input from both low-threshold mechanoreceptors and high-threshold nociceptors. Such "wide dynamic range" neurons, which tend to have large receptive fields, respond weakly to brushing, pressure, and mild pinch but vigorously to strong pressure or pinch.

It may seem surprising to have two distinct classes of spinal cord neurons involved in relaying information about pain. It may also seem surprising that

the second class, the wide dynamic range neurons, are activated by both noxious and innocuous stimuli. Consider, however, that pain can be produced by a wide variety of stimuli—intense heat or cold, strong pressure or pinch, electrical pulses, certain classes of chemicals. The resulting pain sensations are not identical—they differ in quality, intensity, location, and duration. A straight-through pain system that signaled simply the absence or presence of pain would not provide the coding mechanisms necessary for such a complex range of experiences.

Intense levels of heat or pressure applied to the same area of the skin will each excite wide dynamic range neurons and some common nociceptive-specific units. However, the heat will selectively excite some nociceptive-specific neurons while intense mechanical stimulation will excite others. As well, a pinch or prick will excite low-threshold mechanoreceptive neurons. Consequently, different patterns of stimulation, involving wide dynamic range neurons, two types of nociceptive-specific neurons, and low-threshold neurons can underlie the capacity to distinguish between different noxious inputs (Price, 1988). Furthermore, the nociceptive-specific neurons, with their small receptive fields, may play a particular role in conveying information about the site of stimulation, whereas the wide dynamic range neurons, particularly given their overlapping receptive fields, may better signal the intensity of the stimulus applied to the skin.

The neural processing of pain does not, of course, end at the spinal level. As we'll see shortly, the situation at the cord is even more complex than already mentioned, but it is necessary to note that spinal neurons project, via a number of ascending pathways, such as the spinothalamic and spinoreticular tracts, to a variety of sites in the brain stem (particularly the reticular formation of the medulla), the midbrain, and the medial and lateral thalamus (see Fig. 4.1).

Cells in the reticular formation may contribute in large part to aversive drive and resulting escape behaviors. Those in the midbrain seem to trigger emotional reactions such as fear, aversion, and other negative affects. The neurons in the lateral and medial thalamus receive input from the nociceptive specific and wide dynamic range neurons of the spinal dorsal horn and pass these messages, in turn, to the somatosensory cortex or the association areas of the frontal cortex. Many of the former appear to be involved in the localization of pain and discrimination of its sensory intensity. Neurons in the frontal lobes appear to subserve emotions brought on by the pain and motivations to escape or avoid it.

THE GATE CONTROL THEORY OF PAIN

Much of the physiological data mentioned in the preceding sections was unknown 25 years ago, when Melzack and Wall (1965) proposed their new theory of pain. Nonetheless, enough had been determined to provide the foun-

dation for a radically different approach and Melzack and Wall were prescient in their ability to anticipate some major new developments.

In formulating their theory, Melzack and Wall considered the data on physiological specialization, clinical information about prolonged pain syndromes that far outlast tissue damage, the difficulty of treating pain with pharmacological or surgical procedures designed to interrupt afferent pathways, and the counterintuitive aspects of many successful pain treatments (Melzack & Wall, 1988). One example of the last is the manner in which we often deal with a cut or bruise: We rub the injured area, hold it under cold water, or apply a warm dressing. These tactile or thermal stimuli markedly increase the total afferent barrage, yet they reduce or eliminate pain rather than rendering it even more severe.

Melzack and Wall (1965) considered the psychology of pain as well as its physiology. They had to consider a host of anecdotal and clinical data: individuals severely injured in military battle, accidents, or athletic competition frequently fail to complain of pain until many hours later; initiation ceremonies, tribal rituals, and religious observances often involve trauma, but the individual appears to be in a state of ecstasy rather than discomfort; individuals of different cultural background often show widely divergent pain behaviors; early experience and the behavior of family role models appear to shape responses to noxious events; expected painful procedures (such as an inoculation) seem less aversive than unexpected ones; noninvasive therapies, such as hypnosis, often seem to ameliorate pain; pain is amplified by anxiety and attenuated by relaxation or distraction; personality, coping behaviors, and knowledge about the source of the pain markedly affect pain complaints; about one-third of patients report sizable decreases in their levels of discomfort after being given a placebo (Melzack & Wall, 1988).

Clearly, it was wrong to think of pain as simply a sensation arising from overstimulation of pain fibers and consequent stimulation of a cortical pain center. At the least, one had to invoke processes of perception, emotion, evaluation, and reaction. These affective and cognitive mechanisms didn't seem to exist as independent processes that followed pain as a sensory event; rather, they appeared to interact with sensory mechanisms from the inception of the transmission process.

Melzack and Wall's theory made much of this interaction, suggesting inhibitory relations at numerous levels of the nervous system. The initial version of the "gate control theory" is presented in Fig. 4.2. Briefly, it proposed that noxious stimuli activate small-diameter (S) A-delta and C-fibers that project to transmission cells (T) in the spinal cord and, from there, to an "action system" locally and in the brain. As noted earlier, the cord also receives input from large-diameter (L) myelinated A-beta fibers that are excited by low levels of mechanical stimulation.

These excitatory connections are muted by a complex interplay of inhibi-

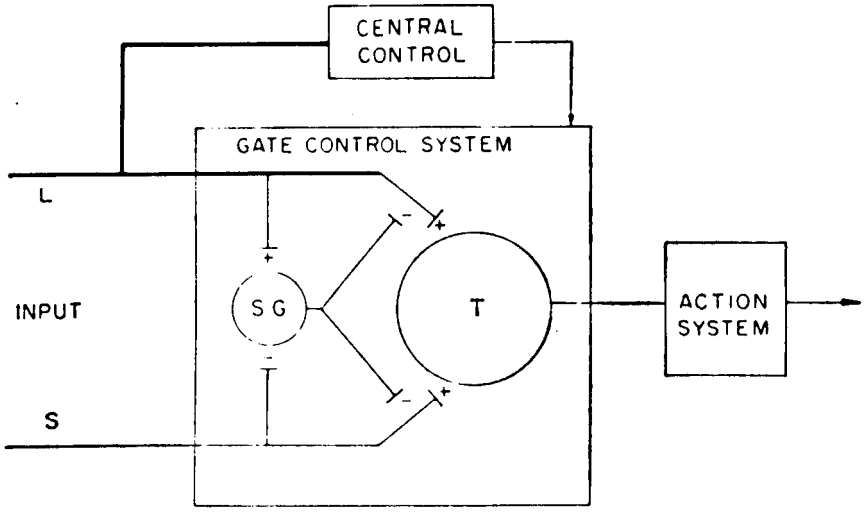

FIG. 4.2. A schematic representation of the original gate control model of pain. L and S represent the large- and small-diameter fibers, respectively, which project to transmission cells (T) in the spinal cord. The fibers also project to the substantia gelatinosa (SG) cells, modifying, by means of excitatory (+) and inhibitory (−) actions, its influence on the transmission cells. Further regulation from central areas is also shown (from R. Melzack, and P. D. Wall (1965), "Pain Mechanisms: A New Theory," *Science, 150*, 971–978; copyright, 1965, by the American Association for the Advancement of Science; reprinted by permission).

tory influences mediated through the substantia gelatinosa (SG) (jelly-like substance), a region of interconnecting neurons in laminae I and II of the dorsal horn. In the initial version of the theory, Melzack and Wall (1965) proposed that the SG has an inhibitory influence on the T-cells and that it, in turn, was subject to an excitatory influence from the L-fibers and an inhibitory one from the S-fibers. Thus, noxious inputs would make a powerful contribution to T-cell activity; small fiber activity would directly excite the T-cells and would inhibit the inhibitory influence of the substantia gelatinosa (a process called "disinhibition").

Activity in large fibers, however, brought on by lower levels of input, would excite the substantia gelatinosa neurons and, consequently, exert a strong inhibitory effect on the T-cell. This would account, at least in part, for the pain-alleviating effects of massage, rubbing, acupuncture, and other forms of peripheral stimulation, although a second mechanism will be described shortly.

An important component of the gate control theory was the notion of descending influences on T-cell activity in the spinal cord that arise at "central control" areas of the brain stem, midbrain, or cortex. This activity, which

is inhibitory, can diminish or block the transmission of pain information in the dorsal horn.

A rather dramatic notion was being proposed: the passage of information from the cells of the spinal cord could be likened to the passage of an object through a gate; an open gate allows the pain message to flow uninterrupted, whereas a gate that is partly or completely closed (due to inhibitory effects from large fiber activity from the periphery or descending influences from the central brain regions) moderates the spinal activity and reduces the afferent barrage (and the pain).

Subsequent anatomical and physiological knowledge about the substantia gelatinosa has led to a modification of the gate control theory, although the basic idea of inhibitory influences from both peripheral and central impulses remains. The revised model (Melzack & Wall, 1988), shown in Fig. 4.3, provides for multiple excitatory influences arising from small fiber input, showing both direct effects on T-cells and indirect ones from interneurons in the SG. The large fibers, as before, contribute directly to T-cell activity (combining with small fiber input to produce wide dynamic range cells, as opposed to nociceptive specific cells that receive input from only S-fibers). However, the L-fibers also inhibit the T-cells through the substantia gelatinosa.

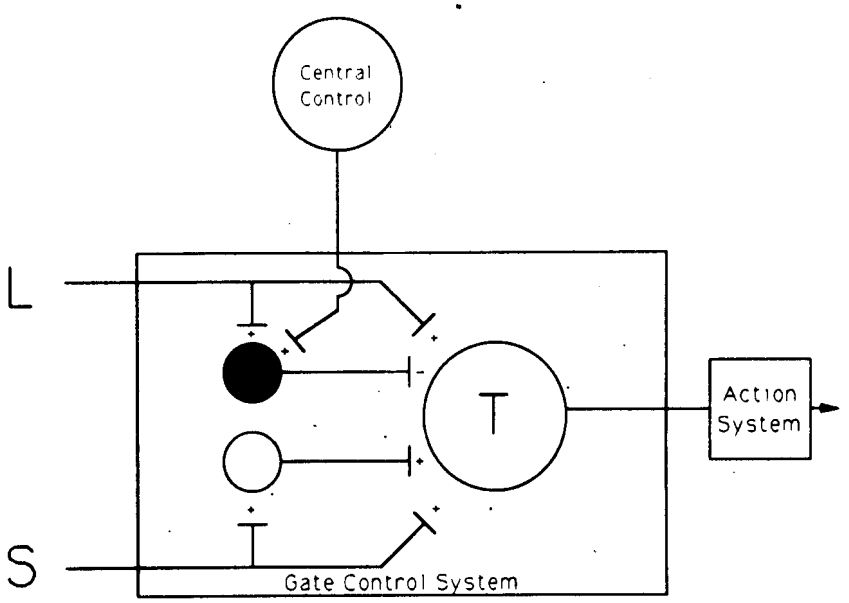

FIG. 4.3 A schematic representation of Melzack and Wall's (1988) revised gate control model, showing cells in the substantia gelatinosa, which exert inhibitory (solid circle) and excitatory (open circle) influences on dorsal horn transmission cells (T) after activation of large (L) and small (S) diameter afferents. The modulating influence of higher nervous system activity is depicted by the input from central control areas.

In a manner rather more complex than earlier thought, powerful inhibitory effects from the brain stem also act to inhibit T-cell activity, through their facilitation of inhibitory interneurons. These descending effects provide for both inhibition of pain by psychological factors and for a negative feedback loop in which some noxious inputs activate brain stem structures that exert an inhibitory effect on continued transmission through the spinal cord. The curious phenomenon of counterirritation, in which intense levels of heat, cold, pressure, or electrical stimulation can help reduce pain may be due to activity in fast-conducting afferent pathways influencing, in turn, activity in inhibitory efferent ones.

DESCENDING INHIBITORY SYSTEMS

In the late 1960s, experiments conducted in two laboratories (Mayer, Wolfe, Akil, Carder, & Liebeskind, 1971; Reynolds, 1969) demonstrated powerful analgesic effects produced by midbrain stimulation in the rat. Electrical pulses applied to the periaqueductal gray (PAG) area yielded analgesia that was long-lasting, applicable over a wide range of pain-inducing situations, and, ultimately, generalizable to humans (Meyerson, 1983; Young, 1989). This "stimulation-produced analgesia" appears to be mediated by a descending circuit that projects to the nucleus raphe magnus of the medulla and then to nociceptive neurons in various laminae of the dorsal horn of the spinal cord. Fig. 4.1 shows inputs from the frontal association cortex and the hypothalamus impinging upon cells in the midbrain which, in turn, send fibers to the medulla and then the cord. Inhibition of spinal pain-transmission cells by this system modifies the pain experience.

This descending system has been studied extensively in recent years because it has become clear that it is intimately linked to the neural substrates of opiate analgesia. The resulting emphasis on neurochemistry of pain modulation has produced an enormously fertile area for research on the interactions among structure, function, chemical transmitters, and behavior.

Stimulation produced analgesia (SPA) can be mimicked by microinjections of morphine into the periaqueductal gray (Bennett & Mayer, 1979). The PAG has a high density of opiate receptors. Tolerance effects observed with morphine occur as well with SPA. In fact, there is also a cross-tolerance effect, so that continued administration of morphine reduces the effectiveness of midbrain stimulation and vice versa. Naloxone, a morphine antagonist that binds to opiate sites and blocks the morphine molecule, also reduces the effectiveness of SPA (Watkins & Mayer, 1982).

The identification of opiate receptors raised a provocative question: Did Mother Nature provide such sites in the hope that someday individuals would harvest poppy plants, extract the milky fluid from the unripe seed pods,

process it, and inject it into pain sufferers? That seemed highly unlikely. More likely was the thought that the presence of such receptors indicates that there are also endogenous opiates—morphine-like chemicals that naturally occur within the body. Verification of the existence of such neurochemicals occurred in the late 1970s and they were labeled endorphins ("endogenous morphine") and enkephalins ("within the brain"). Numerous variants of these polypeptides have been discovered: several types of enkephalins, larger molecules called dynorphins, and the complex beta-endorphins. A whole witch's caldron of other neurotransmitters is known to influence pain transmission (Yaksh & Aimone, 1989) and enormous efforts are under way to create synthetic molecules that provide the analgesic effects of the opiate drugs but not the major side-effects: nausea, constipation, respiratory depression, tolerance, and dependence.

The endogenous opiates play an important role in the pain experience. Nonetheless, it is clear that a separate pain suppression system exists as well. For example, certain forms of stress (e.g., intermittent foot shock or immobilization) produce a marked increase in pain tolerance in animals ("stress-induced analgesia") (Lewis, Cannon, & Liebeskind, 1980), which can be reversed with the morphine antagonist naloxone. Other forms of stress (brief continuous foot shock, centrifugal rotation, cold-water swims) also produce analgesia, but these effects are uninfluenced by naloxone, although they are attenuated by drugs which block other neurotransmitters such as serotonin (Coderre & Rollman, 1984).

In humans, certain forms of intense, low-frequency electrical stimulation applied through the skin to peripheral nerves (acupuncture-like transcutaneous electrical nerve stimulation—TENS) produce analgesic effects which are naloxone-reversible; other parameters (high frequency) of TENS also have analgesic effects, but naloxone does not interfere with these (Sjolund & Eriksson, 1979). Appropriately, only the low-frequency TENS produced an elevation of beta-endorphins measured in human cerebrospinal fluid (CSF).

Measurement of endogenous opiates in CSF and in blood plasma has become a fascinating, if controversial, area. Controversy focuses on a number of issues: the utility of such peripheral information, especially when obtained from blood fractions, and the ethics and risks of tapping into the spinal cord for CSF (Sternbach, 1979).

Subjects with high levels of endorphins showed greater tolerance to experimentally induced pain than those with low endorphin levels (von Knorring, Almay, Johansson, & Terenius, 1978). In a related study, surgical patients with low preoperative levels of endorphins required more opiate analgesia postoperatively to relieve their pain than did patients with higher endorphin levels (Tamsen, Sakurada, Wahlstrom, Terenius, & Hartvig, 1982). Finally, a small group of chronic-pain patients with clear neurological signs of organic lesions showed lower levels of endorphins than patients in which no

nerve damage could be found (Almay, Johansson, von Knorring, Terenius, & Wahlstrom, 1978). This raises the intriguing question of whether these individuals developed a chronic syndrome because they had low levels of endorphins prior to the onset of their disorder, rendering them less able to recover, or because the nerve damage, and resulting pain, puts unusual demands on their endogenous opiates, reducing the capacity of the endorphin system. Some evidence for the latter view comes from the finding that there is a negative correlation between the endorphin levels and the duration of the pain syndrome.

There is an intriguing counterposition to the situation of chronic-pain patients with low endorphin levels. Some individuals are congenitally insensitive to pain; they do not feel it. Lest you consider them lucky, reflect on the ambivalent situation in which we are placed by pain; it is one of the scourges of humankind, yet it serves to warn us of mild damage so that we can withdraw or seek attention before it becomes more severe, it forces us to rest so that the body can recuperate, it helps us learn to avoid future dangers. Congenitally insensitive persons are unable to utilize the beneficial aspects of pain and often experience serious burns, cuts, and bruises as well as severe damage to the joints and bones that lead to tragically brief lives (Melzack & Wall, 1988). It's likely that several different mechanisms underlie this disorder, but consider two for the moment: The affected individuals may have a neurological deficit in which nociceptive transmission is attenuated in some manner or they may have an overabundance of endorphins, producing an effect something like a constant infusion of morphine.

Some recent findings support both of these concepts. One case study discovered an absence of A-delta fibers in the roots feeding the dorsal horn and a reduced size of the major ascending tract that carries small fiber afferents to the brain stem (Swanson, Buchan, & Alvord, 1965). A second study was conducted on the foreskin of a 4-year-old boy who showed no sensitivity to noxious or thermal stimuli and had many scars from self-damage, particularly on the tongue. It, too, revealed a marked deficit in the number of small myelinated fibers (Bischoff, 1979). As well, there was an almost total absence of C-fibers. Microscopic analysis of the tissue, however, revealed Pacinian corpuscles, Meissner's corpuscles, and large myelinated A-fibers. Not surprisingly, reaction to tactile stimuli was clearly present.

An alternative account of the factors underlying at least some cases of congenital pain insensitivity comes from the investigation of Dehen, Willer, and Cambier (1979). Their patient was a 34-year-old woman who was brought to their attention when she suffered a third-degree burn of the hand after placing it on a radiator. She showed "no behavioral reactions to pain," no matter how intense the noxious stimulus or where it was applied. She was, however, normal in her capacity to judge warm from cold, know the position of her joints or identify objects by touch. In her case, unlike the patients cited earlier, nerve biopsy was normal.

The investigators studied a nociceptive flexion reflex of the lower limb by placing electrodes over the sural nerve at the heel and measuring a long latency reflex at the thigh that involves narrow diameter fibers and typically arises at about the threshold for pain (Willer, 1977). The threshold for normal subjects was about 10 mA; that for their patient was 45 mA. However, after injection of naloxone, the opiate antagonist that blocks the sites at which the endorphins bind, her nociceptive reflex threshold fell to about 16 mA (whereas naloxone had no effect on the controls). Interestingly, the woman still did not describe the shock as "painful;" rather, she said it gave rise to a sensation of "warmth." Nonetheless, the data suggest that the patient had an endorphin system that was "permanently hyperactive" and produced a tonic inhibition via the descending system described earlier. Only when the naloxone temporarily blocked this inhibition was the woman able to demonstrate a normal nociceptive reflex and at least some sensation, albeit still far from painful, from the electrical pulse train applied to her foot.

CHARACTERISTICS OF PAIN

It won't be a revelation to note that not all pains are the same. Pain experiences differ in quality, intensity, location, extent, and duration. Some pains are cramping; others seem stabbing, burning, or pounding. Some are mild or moderate; others are excruciating. Pains due to burns are enormously different than those arising from headaches or back injuries or stomach aches. A pinprick is localized to a small spot on the finger, whereas the inflammation of the nerves produced by the virus herpes zoster (shingles) leaves some people with a postherpetic pain that not only covers large body parts, but creates an extensive area that is also exquisitely sensitive to mild tactile or thermal stimuli. This effect, called hyperesthesia, is accompanied by alterations of summation that are akin to the auditory phenomenon of recruitment: a warm stimulus applied to the skin may have no effect for a while, but then a sudden, intense pain, explosive in character, renders the stimulus unbearable (Noordenbos, 1959).

The duration of pain is of key importance in regard to both its experience and its psychological effect. Three particular categories deserve attention: transient, acute, and chronic pain.

Transient pain is usually sudden and brief—a pinprick, a jab from an elbow, a spill from a coffee cup. It is the closest that pain comes to being purely a sensation. A fleeting experience of discomfort leads to escape behavior and, if the individual is fortunate, the matter is over.

More severe injury causes greater tissue damage and, with that, the release of chemicals such as prostaglandins and bradykinin, which sensitize nearby nerve endings. The pain experience now is considerably prolonged, often

variable in intensity, and likely to trigger a series of emotions and thoughts that accompany the sensory experience. Such pain is called acute.

Usually, the recovery process is straightforward and, as the injury heals, acute pain diminishes and then disappears. Nonetheless, even when the cause of the pain is quite evident, there is at least some concern about the duration and extent of limited activity. When the cause is not immediately evident, as is frequently the case for pain arising within the body, the concern is considerably greater. Does the pain signal a serious disease? Is it going to last a long time? Might it become increasingly severe? Anxiety, often of severe magnitude, accompanies the pain. Moreover, the anxiety may amplify the pain experience. Assurance from a physician that the cause of the problem is a minor matter often causes the anxiety to fade quickly—and with that, also the pain.

Regretfully, sometimes pain persists, often well past the time of tissue healing. Acute pain, when it lasts longer than 6 months, becomes chronic. More than simple duration distinguishes acute from chronic pain. Psychological factors become inextricably woven with organic ones, in regard to both the reaction to the discomfort and in its becoming a chronic problem in the first place. A key characteristic of the chronic-pain patients is depression (Merskey, 1986b). Not unexpectedly, the individual is deeply distressed by the many months the pain has lasted, by the inability of a series of physicians to provide a quick fix, by the ineffectiveness of many analgesic drugs to control the situation, and by the dramatic changes in the patient's life that are brought about by the pain. These effects are seen in restrictions on the patient's daily activities, changed relationship with family members and friends, reduced income, and increased medical expenses. As Sternbach (1989) notes, "whereas acute pain may promote survival, chronic pain is usually destructive physically, psychologically, and socially."

Psychological methods are often used to help in both the diagnosis and treatment of chronic pain. Psychological tests such as the Minnesota Multiphasic Personality Inventory (MMPI) are employed to identify personality disturbances that, depending on the view of the investigator, are predisposing factors in the onset of the chronic-pain disorder or consequences of it. That is, a personality disturbance may cause an individual to assume the role of a chronic-pain patient when faced with a disease or injury that would quickly heal in another individual. On the other hand, a "normal" personality may undergo major alterations as a consequence of prolonged and unremitting pain.

MMPI profiles of chronic pain patients often show elevations on three primary scales: depression, hysteria, and hypochondriasis. This "neurotic triad" or "conversion-V profile" (based upon a common pattern of response) is then assumed to represent a personality disturbance (before or after the pain, although the return toward normal levels after successful treatment suggests the latter is more likely).

Another account, however, seems even more plausible. The original MMPI was established as a test of psychopathology and its norms are based on psychiatric patients. Its use for medical patients is a common but irregular practice. If individuals taking the MMPI answer "True" to items such as "I feel weak all over much of the time" or "My sleep is fitful and disturbed" or "False" to the questions "I have few or no pains" or "I am about as able to work as I ever was," the scoring procedure adds points to all three scales: hypochondriasis, depression, and hysteria. Thus two problems emerge. First, common symptoms of pain disorders inevitably boost the scales that are used to diagnose a neurotic disorder. Second, because individual items contribute to the scores on several scales, the scales are not independent. As Smythe (1984) observed, "The MMPI is not an appropriate scale for use in patients with organic diseases causing pain or disability." While there may be a psychological component in chronic-pain disorders, personality tests with such built-in biases must be interpreted with extreme caution.

Chronic-pain disorders are often unresponsive to pharmacological, surgical, and other medical interventions. In recent years, such patients have been aided by a wide range of psychological treatments: cognitive–behavioral therapy (Turk, Meichenbaum, & Genest, 1983), behavioral therapy (Fordyce, 1976), hypnosis (Hilgard & Hilgard, 1975), relaxation and biofeedback (Jessup, 1989), psychotherapy (Merskey, 1986b), and family therapy (Roy, 1986). Not all patients are helped; those who fail with one may respond to another. The therapeutic approaches tend to share some common features, such as providing a rationale for the patient's pain, communicating a message of hope and optimism, tailoring the approach to the individual, and relying heavily on the patient's participation in his or her own treatment (Turk & Holzman, 1986). Nonetheless, they differ widely in both philosophy and technique. A major challenge to psychological research is to identify the individual factors that will optimize the patient–therapy link, so that customization rather than happenstance characterizes the treatment provided.

PAIN ASSESSMENT

The definition presented at the outset of this chapter indicated that pain is "an unpleasant sensory and emotional experience." Both introspection and experimental data suggest that the two are quite different: Pain as a sensation involves concepts of intensity, quality, duration, location, and area, while pain as an emotional reaction involves considerations of unpleasantness, motivational drive toward escape, and cognitive interpretation of the situation in regard to previous experiences, knowledge about the current status, and concern about future outcomes.

Melzack and Casey noted that:

> To consider only the sensory features of pain and ignore its motivational and affective properties, is to look at only part of the problem, and not even the most important part of that. Even the concept of pain as a perception, with full recognition of past experience, attention, and other cognitive determinants of sensory quality and intensity, still neglects the crucial motivational dimension. (1968, p. 423)

While some earlier notions had differentiated between "pain sensation" and "reactions to pain," the latter was generally seen as a secondary consideration, which arose after the pain sensation had occurred. Clinical evidence challenged that conception. Beecher's (1959) report that soldiers badly wounded on the battlefield "entirely denied pain from their extensive wounds or had so little that they did not want any medication to relieve it" suggested that pain could be blocked by cognitive activities.

Patients given prefrontal lobotomies (Barber, 1959) as a last-ditch attempt to treat intractable pain, still describe a sensory component of their internal pain, but they don't show facial or bodily reactions, appear not to be anxious, and don't ask for medication. Sometimes they suggest the "little pain" is still there but the "big pain" is gone. Again, the data point to a complex interaction between motivational and emotional factors and sensory ones.

Melzack and Casey (1968) proposed a new conceptual model that had three highly interdependent components of the pain experience: (1) the sensory–discriminative system, (2) the motivational–affective system, and (3) the cognitive–evaluative system.

The proposed scope of activities mediated by these systems fits well with the known function of the principal neural loci reviewed earlier. The sensory–discriminative dimension seems particularly linked to activity projected via the neospinothalamic system from the spinal cord to the lateral thalamus and then to the somatosensory cortex. The motivational–affective dimension, underlying the unpleasant feelings and escape behaviors associated with noxious stimuli, activates neurons in the brain stem reticular formation, medial thalamus, limbic forebrain structures, and frontal cortex. The cognitive–evaluative dimension, subserving the interpretation of the pain experience, is tied to the frontal cortex and other association areas.

Although there are numerous points of interconnection between these systems, and consequent opportunity for interactions, critical questions still remain about the extent to which the systems should be considered part of a parallel processing network, as Melzack and Casey (1968) proposed, or a sequential processing network, in which pain sensations and arousal are activated in parallel but emotional responses and cognitive appraisals interact at higher levels to determine the pain expression (Price, 1988).

While there is disagreement about the particular role of the various ascending and descending pathways and about the precise nature of the spinal gating mechanism, it is clear that the gate control theory and the tridimen-

sional conceptual model played an enormously important role in introducing psychology to a field that had earlier been almost exclusively the domain of neurology, physiology, and pharmacology. By emphasizing the complexity of the pain experience, the crucial role of emotion and thought, and the possibility of modulating the experience through manipulation of affect and cognition, Melzack and his colleagues established an enormously fruitful research area.

More importantly, they provided a rationale for the psychological treatments mentioned earlier. Melzack and Casey showed both sensitivity and foresight when they wrote,

> The therapeutic implications of the model should be obvious; but because of the historical emphasis on the sensory dimension of pain, they are not obvious at all. The surgical and pharmacological attack on pain might well profit by redirecting thinking toward the neglected . . . contributions of motivational and cognitive processes. Pain can be treated not only by trying to cut down the sensory input by anesthetic block, surgical intervention, and the like, but also by influencing the motivational–affective and cognitive factors as well. Relaxants, tranquilizers, sedatives, suggestion, placebos, and hypnosis are known to influence pain, but the historical emphasis on sensory mechanisms has made these forms of therapy suspect, seemingly fraudulent, almost a sideshow in the mainstream of pain treatment. Yet, if we can recover from historical accident, these methods deserve more attention than they have received. (1968, p. 435)

The foregoing makes it clear that the assessment of pain requires attention to three dimensions: sensory, affective, and cognitive. Interestingly, attention in both clinical and laboratory settings has focused almost entirely on the first two, although some recent studies have considered the nature of the interactions between multiple sources of pain. The adaptation level and hypervigilance models (Rollman, 1979, 1983; Scudds, Rollman, Harth, & McCain, 1987) focus on the modulation of judgments of experimentally induced pain induced by intense pain states elsewhere in the body; the functional model (Algom, Raphaeli, & Cohen-Raz, 1986) addresses the summation between more nearly equal pain states. Inhibition is frequently seen in the former condition; summation in the latter.

For approaches that emphasize the sensory and affective components, there is controversy about the relative emphasis that ought to be paid to each member of the pair and the philosophical and methodological orientation of the assessment process. Wall (1979), for example, distinguishes between two types of sensory experience: one, evoked by external events, such as seeing and hearing and a second, produced by internal events, such as hunger and thirst. Based on a number of criteria (e.g., ability to describe and localize the stimulus, effects of distraction and suggestion, association with emotion-

al response and predictable behavior), he suggests that pain has more in common with the second class. Moreover, the imprecise link between tissue damage and pain intensity as well as the inability to utilize pain information as a warning, since damage has already occurred, suggests to Wall that pain signals a body state (as do hunger and thirst) that, in this instance, leads to escape, treatment, and recovery.

Wall claims that pain and its associated emotion are "two faces of the same coin" and that attempts to split the experience into two components is an artifact of training subjects. While claims that sensory and affective components of pain can be independently assessed are unwarranted, two approaches to the assessment process demonstrate that distinctions between sensation and affect are possible, even with naïve observers.

The first example comes from the clinical domain. Clinical pain assessment often involves simple questions: Is the pain mild, moderate or severe?" or "where would you rank the pain on a five-point scale?" Such approaches have the benefit of speed and simplicity, but they lack quantitative rigor (being, at best, ordinal scales) and they fail to capture the complexity of the pain experience.

Melzack and Torgerson (1971) reflected on the fact that patients generally talk about their pain using adjectives—a "splitting" headache, a "shooting" pain in the knee, a "cramping" stomach ache. After gathering more than 200 pain-related adjectives, they categorized the words into 3 major classes and 16 subclasses. Later, they added 4 supplementary subclasses. The words in the first class described sensory qualities of pain: temporal characteristics (flickering, pulsing, pounding), spatial characteristics (jumping, shooting), punctate pressure (pricking, drilling), thermal (hot, burning), and so on. The second class, which had 5 categories, compared with 10 in the first one, brought together affective terms relating to such emotions as tension (tiring, exhausting), fear (frightful, terrifying), or punishment (cruel, vicious). The third category, evaluative, had but a single subclass, containing words such as annoying, troublesome, miserable, and unbearable.

Melzack and Torgerson then asked various groups to rank order the words within a subclass in terms of severity. Quite reliably, individuals established a hierarchy, such that "stinging," for example, was more severe than "smarting," which was, in turn, more severe than "itchy" and "tingling." The fourth category had miscellaneous terms that were largely sensory in nature.

From this multidimensional scaling, Melzack (1975) established the McGill Pain Questionnaire (MPQ; Fig. 4.4), which has been widely adopted in pain clinics throughout the world. Patients are asked to examine each subclass, decide if one of the terms is descriptive of their pain, and, if so, to check the appropriate one.

Up to 20 words could be selected, although patients generally choose far fewer. Melzack devised a Pain Rating Index (PRI), based upon the summed

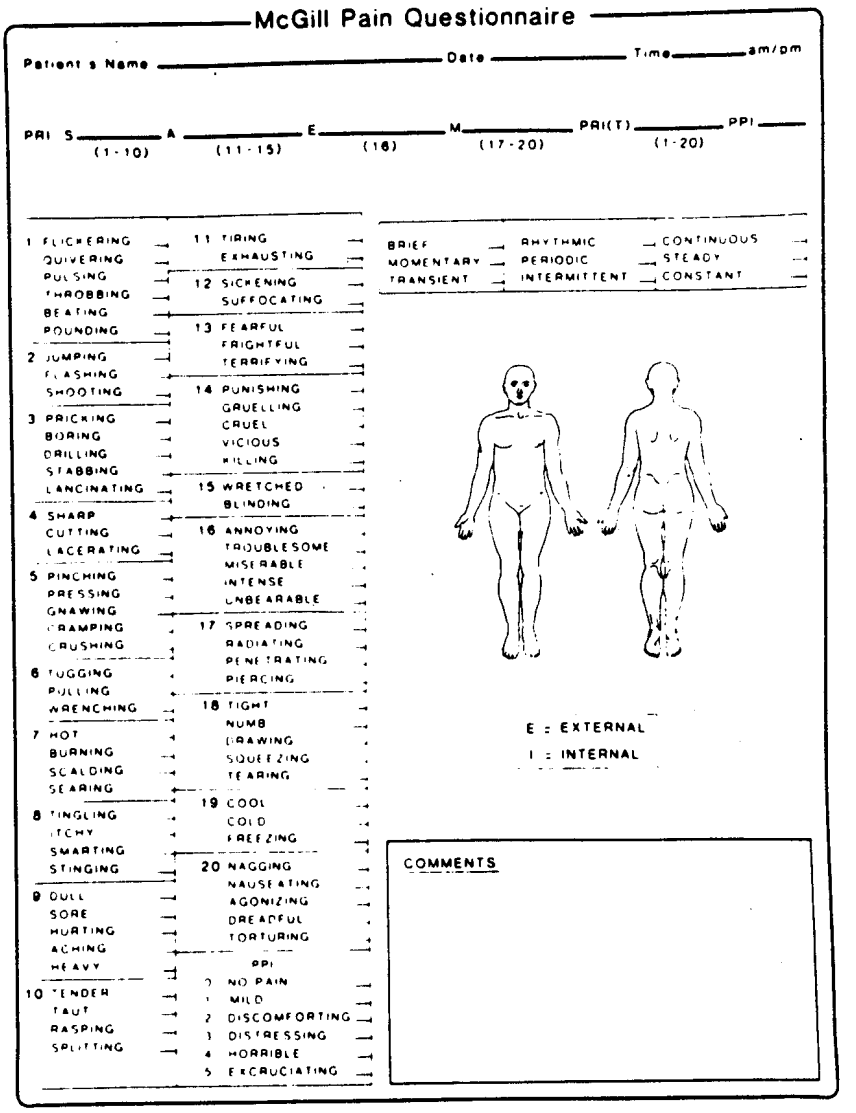

FIG. 4.4 The McGill Pain Questionnaire. The verbal descriptors fall into four categories: 1–10 are sensory, 11–15 are affective, 16 is evaluative, and 17–20 are miscellaneous. The Pain Rating Index (PRI) is the sum of the rank values of the words selected in each category. The Present Pain Intensity (PPI) is based on a six-point category scale (from R. Melzack (1983), The McGill Pain Questionnaire; in R. Melzack (Ed.), *Pain Measurement and Assessment* (pp. 41–47); New York: Raven Press; reprinted by permission).

rank order of the words chosen; "pulsing" in category 1 provides three points, since it is ranked third, while "flickering" provides one point. The potential range, then, of the PRI is from 0 to 77.

There are problems with this scheme: There is a preponderance of sensory items, some subclasses have more words than others, the intervals between words are uneven. Furthermore, some patients lack the linguistic skills to complete the form. Nonetheless, the MPQ has proven to be a robust instrument for assessing a wide range of clinical syndromes. It has acceptable reliability and validity (Reading, 1983), it is moderately sensitive to analgesic procedures, its emphasis on three dimensions of pain is generally supported by factor-analytical studies (Prieto & Geisinger, 1983). A brief form has recently been published (Melzack, 1987). The high intercorrelations among the sensory, affective, and evaluative subscales have led to some concern about the discriminant validity of the MPQ (Melzack, 1985; Turk, Rudy, & Salovey, 1985). Although different pain syndromes tend to produce different profiles on the MPQ (higher in sensory scales for some, higher in affective for others), evidence is still sparse about the capacity of the MPQ to identify alterations in one component of pain after a specific therapeutic intervention (e.g., a reduction in the affective score) while leaving the other main component (in this case, the sensory) relatively unaffected.

Data suggestive of such selective modulation of pain have appeared in several studies involving other approaches which attempt to assess directly the sensory and affective components of pain. Price, McGrath, Rafii, and Buckingham (1983) had pain patients and healthy volunteers describe the intensity and affective magnitude of a series of noxious heat pulses applied to the forearm through a contact thermode. The responses were made on printed visual analog scales (VAS), which were 15-cm lines anchored by the terms "no sensation" and "the most intense sensation imaginable" in the case of the sensory judgment and "not bad at all" and "the most intense bad feeling possible for me" in the case of the description of emotion.

The two components did not increase at the same rate when the temperature was increased from 43° to 51°C. The slope of the power function linking sensation intensity to temperature was 2.1, while the slope of the function between affective magnitude and temperature was nearly twice as large (3.8). These data suggest that the unpleasantness of a noxious stimulus increases much more rapidly than the intensity of the pain. Acupuncture treatment of chronic-back-pain patients gradually reduced both the sensory and affective ratings over a 4-month period; there is a suggestion that the affective component changed more than the sensory.

In a subsequent study, Price, von der Gruen, Miller, Rafii, and Price (1985) reported a reduction in affect but not intensity following low doses of morphine, but changes in both following higher doses. The synthetic narcotic fentanyl also reduced both components (Price, Harkins, Rafii, & Price, 1986).

However, when it was used to treat low-back pain, fentanyl caused a significantly larger decrease in VAS affective responses (dropping by 65%) than sensory ones (a decline of 51%).

These data don't show that the two components, sensory and affective, are independent. In fact, it would be difficult to imagine that the emotional component of the pain experience is unrelated to the sensory intensity of the pain. The findings do suggest, however, that analgesic treatments, whether they be pharmacological, surgical, or psychological, might alter sensation and affect differentially. A tranquilizer, for example, could reduce the emotional reaction to discomfort while leaving the intensity of the sensation unchanged (Gracely, McGrath, & Dubner, 1978). An assessment tool that focused only on intensity would mistakenly conclude that the patient's condition was unimproved.

As noted earlier, pain patients are faced with a disease or injury that has both sensory and emotional consequences. Price, Harkins, and Baker examined whether

> Affective VAS ratings of clinical pain will be higher in patients whose pain is likely to be associated with a serious threat to health or life in comparison to patients whose pain is likely to be less threatening . . . for comparable levels of pain sensation intensity. (1987)

Their results indicate that affective VAS ratings were, as expected, generally greater than sensory ratings for patients suffering from back pain, the burning causalgia pain that follows nerve injury, and cancer pain. Those suffering from the dental myofascial pain dysfunction (MPD) syndrome had no difference between the two ratings. Women undergoing labor, which is an acute pain with a very positive outcome, rated the emotional component as significantly *less* intense than the sensory. The steep increase in affective response to experimentally induced pain in volunteer subjects, compared with the sensory response, was not replicated.

Pain assessment is a complex but rewarding area of research. Rigorous psychophysical procedures (Chapman et al., 1985; Gracely, 1989; Rollman, 1989) can be utilized to assist in diagnosis and to evaluate treatment. The last 25 years have seen an explosive interest in pain research and treatment, in the establishment of international research societies and journals, in the proliferation of pain clinics, and in the understanding of the physiological and psychological factors that contribute to the experience of pain and to its alleviation. There is no cause for complacency about the status of pain control, but there is much cause for hope.

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The Psychology of Touch

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