SPRINGHOUSE GUIDE

Expert Pain Management

Foreword by Carol A. Warfield, MD Harvard Medical School

Expert Pain Management

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Preface

Throughout the ages, the fear of suffering unrelieved pain has been a very real concern that crosses all socioeconomic, political, and cultural lines. Just as pain has existed since the beginning of time, so have attempts at treating it. Early humans often attributed pain to demons and therefore attempted to ward them off with charms, amulets, rattles, and incantations. Eventually, sorceresses and shamans, seen as pain exorcists, became an integral part of human culture. Shrines such as the pyramids were built to appease the gods, and priests used charms and sacrificial offerings as a treatment for pain.

References to pain and pain-relieving techniques abound throughout recorded history. Pain relief was depicted on Babylonian clay tables, Egyptian papyri, and the artifacts of ancient Persia and Troy. About 4,600 years ago, the Chinese first described Yin and Yang, two opposing yet unifying forces in the body, an imbalance of which was thought to be the source of pain. The Egyptians recorded the use of opium for pain relief as early as 1550 BC.

Opium and its active ingredient, morphine, were widely used for many years until eventually the pendulum swung toward the contemporary concern with addiction. Today the widespread fear of drug abuse and addiction has resulted in inadequate and inappropriate treatment of pain. Although recent medical research has provided tremendous technological advances in pain relief, the wealth of knowledge and techniques available are widely underutilized by the health care community.

Inadequate pain management was thought to be such a public health problem that a few years ago, the Agency for Health Care Policy and Research (a division of the U.S. Department of Health and Human Services) convened panels of scientists and health care specialists to compose guidelines for pain treatment. Despite the official recognition of untreated pain as a major public health issue, however, formal medical education and training in pain management lags far behind other topics.

Today pain medicine is a board-certifiable subspecialty. Important breakthroughs in our understanding of pain mechanisms have spurred the development of improved pain management techniques, including

intraspinal and transdermal opioid delivery systems, patient-controlled analgesia, and implantable spinal cord stimulators. Using this new knowledge, researchers have also produced several new medications involving nerve transmission of pain.

Perhaps one of the most important advances in pain management has been the development of a multidisciplinary approach. Several years ago the pain therapy prescribed for a patient was primarily determined by a particular practitioner's specialty. For example, if a patient had back pain, an acupuncturist would recommend acupuncture, a physiatrist would recommend physical therapy, a chiropractor would recommend manipulation, an internist would recommend analgesics, an orthopedic surgeon would recommend surgery, a psychiatrist would recommend psychotherapy, and so on. The advent of the multidisciplinary pain clinic brought together the expertise of numerous specialties and drew attention to the importance of the pain management team.

Today this team is composed not only of physicians specializing in pain management, but also nurses, physical therapists, physician assistants, paramedics, pharmacists, psychologists, and other health care practitioners. Each has a unique role in managing pain. In fact, the pain management physician is seldom the first practitioner to assess the patient in pain. More commonly, paramedical personnel must make important assessments and decisions in the triage of such patients. Without this team approach, modern-day pain therapy would be ineffective.

One of the most important requirements of this team approach to pain management is that all of us as colleagues in various health care fields have a solid knowledge of the current pain treatment techniques as well as an understanding of their physiologic and pharmacologic bases. This shared foundation of basic knowledge encourages each of us to draw upon the expertise of our pain management colleagues and thus provide each patient the optimal course of treatment for pain.

Expert Pain Management provides the reader with crucial information about the pathophysiology and mechanisms of pain perception as well as an introduction to the gamut of pain therapies available and the indications for choosing each. It also provides an invaluable clinical guide to treatment of common pain problems. I hope that this text inspires not only implementation of improved pain management techniques, but also a reordering of patient-care priorities such that relief of the patient's pain becomes a primary measure of our effectiveness as health care professionals.

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Contents

of anesthesia is sufficient during a surgical procedure. Nociceptive reflexes are protective withdrawal (motor) reflexes that are organized at the level of the spinal cord. For example, if a person touches an unexpectedly hot surface with his hand, the result will be reflex withdrawal followed by conscious appreciation that he has just burned himself. The conscious appreciation of a peripheral event as painful requires supraspinal integration of the information in several areas of the brain and, most importantly, interpretation of the event. This supraspinal integration and interpretation are what make pain the unique experience it is.

A subtle but important distinction exists between nociception and the experience of pain. Nociception and pain are not synonyms. The term *nociception* refers to the neural events and reflex responses produced by a noxious stimulus. The term *pain* refers to an unpleasant sensory and emotional experience associated with actual or potential tissue injury or described in terms of such injury. Pain is always subjective. Accordingly, nociception can occur in the absence of the perception of pain, and pain can arise in the absence of nociception. For example, among persons with spinal cord injury, such as paraplegics or quadriplegics, noxious stimuli applied below the level of spinal cord injury can evoke nociceptive withdrawal reflexes because the peripheral nociceptors and spinal reflex circuitry are intact. However, because the information cannot be transmitted above the spinal injury to the brain, such a patient does not perceive noxious stimuli as painful. An example of pain in the absence of peripheral nociceptive input is central poststroke pain syndrome, a condition in which typically deep, aching or burning pain develops in the area of sensory loss or neurologic disability contralateral to the side of the cerebral infarct. Also, a number of pain syndromes show no evident pathophysiology, yet the pain associated with these syndromes can be debilitating.

Pain sensory channel

Sensations arising from skin, joints, muscles, and viscera may be thought of in terms of sensory channels. The term *sensory channel* means the peripheral receptors and nerves, the neurons in the spinal cord upon which the peripheral nerves terminate and transfer their message, the ascending tracts in the spinal cord that carry the information supraspinally, and the sites in the brain where integration and interpretation of input occur. For example, the temperature sensory channel is associated with separate cold and heat peripheral receptors and central pathways. Activation of a sensory channel provides information about the location, onset, intensity, and duration of the stimulus. The sensory channel for pain is generally considered to be com-

posed of two parts: a sensory-discriminative component and a motivational-affective component.

Sensory-discriminative component

The sensory-discriminative component of pain is directly linked to the noxious stimulus and generally refers to pain as a physical sensation. That is, a noxious stimulus activates nociceptors and initiates a series of neural events that ultimately reaches the cortex of the brain to allow a person to determine the location of the stimulus as well as its intensity and duration. Personal experience reveals the importance of the ability to determine easily the location and characteristics of pain; for example, a person does not normally wonder whether a pain is in the left or right hand or even, given a painful finger, which of two adjacent fingers is the correct location. This exquisite ability to characterize and locate the site of pain is best developed in the skin but relatively poorly developed in deeper tissues such as the viscera.

Motivational-affective component

The motivational-affective component of pain includes the nature and intensity of emotional responses that make pain personal and unique for each person. The sites in the brain that contribute to the motivational-affective component and the spinal pathways that convey nociceptive information to these brain sites are different from those associated with the sensory-discriminative component of pain. The motivational-affective component of pain is considered to be served by older, relatively indirect neural pathways that are conserved phylogenetically; that is, they are common to all vertebrates. Whereas the sensory-discriminative component of pain is organized to give information about the location, the time, and the duration of the nociceptive event, the motivational-affective component of pain emotionally colors a person's response to the nociceptive input.

Overlying the sensory-discriminative and motivational-affective components of pain are learned cultural and cognitive contributions to the interpretation of and concern about pain. The cognitive contributions include attention, anxiety, anticipation, and past experiences. For example, if past experience with a potentially painful procedure has been bad, then anxiety about the procedure and anticipation that it will be painful will influence a person's interpretation of and response to any nociception that arises during the procedure. Similarly, attention to or distraction from a potentially painful procedure can influence a person's response. Thus, cognitive contributions can significantly modulate the response and reaction to a painful stimulus.

Pain classifications

Pain can be conveniently classified according to its duration or source. Classification according to duration is important because pain associated with tissue injury sets into motion changes that contribute to altered sensations. Classification according to source is important because the mechanisms of pain associated with superficial structures are different from pain mechanisms associated with the viscera.

Classification according to duration

Pain is most commonly described in terms of its duration as acute, prolonged, or chronic and in terms of its source as somatic or visceral. Most of the above material relates to acute pain. Acute pain has a short duration, is protective, and is not associated with significant tissue injury. Acute pain often evokes a withdrawal reflex but fails to evoke or evokes modest autonomic reflexes and emotional reactions. Venipuncture and paper cuts are examples of acute pain. Both the sensory-discriminative and the motivational-affective components of pain are involved, but past experience instructs that the consequences of the event are not serious.

Chronic pain, in many ways the polar opposite of acute pain, can be of unlimited duration, is not protective, and is associated with significant tissue damage. Chronic pain is typically defined as pain lasting longer than six months. It usually arises from nerve damage, including brain damage, tumor growth, or inexplicable, abnormal responses by the central nervous system to tissue injury. Chronic pain often exists in the absence of obvious pathology long after the initial injury has apparently been repaired or surgery has corrected the damage. Obviously, the motivational-affective and cognitive contributions to chronic pain are very significant. In addition to the personal suffering and debilitation caused by chronic pain, tremendous economic and societal consequences are associated with the millions of chronic-pain sufferers in the U.S. Chronic pain serves no useful purpose and can, if severe and intractable, lead to depression and sometimes suicide. (See *Treatment of chronic pain* for additional comments on the difficulties of treating chronic pain.)

Perhaps the most common pain is described as prolonged, meaning that it lasts days to weeks. Prolonged pain is always associated with tissue injury and inflammation, such as sunburn, sprains, or surgery. One consequence of tissue injury and inflammation is the development of an exaggerated response to a normally painful stimulus, a phenomenon termed hyperalgesia. For example, a pinprick may cause the experience of severe pain in an inflamed finger. In hyperalgesia, chemicals released or synthesized at the site of injury

Treatment of chronic pain

Chronic pain is the most difficult type of pain to treat. Even ancient cultures resorted to surgical procedures such as drilling holes in the skull to let the evil spirits escape. Modern surgical procedures such as cutting the ascending spinothalamic tract (cordotomy) are sometimes used to treat chronic pain unresponsive to less invasive therapy. Unfortunately, although the pain disappears immediately following surgery, it often reappears over the following 6 to 12 months. Possible reasons for the reappearance of pain offer interesting insights into the mechanisms underlying nociception. One possible explanation for the reappearance of pain following cordotomy is that alternative, parallel nociceptive pain pathways are engaged following surgery. Alternatively, information in nonnociceptive pathways may be used to estimate noxious stimuli. These hypotheses underscore the robustness of the nociceptive system.

increase the sensitivity of nociceptors. Because of the increased activity of nociceptors, central neurons upon which nociceptors terminate also undergo a change and they become more easily excited. Thus, prolonged pain is characterized by a change in the function and behavior of almost all elements that comprise the sensory-discriminative component of pain. Importantly, these changes are normal and reversible. It follows then that the neural components of the sensory channel for pain are not static and immutable but exhibit remarkable flexibility and plasticity. Prolonged pain, like acute pain, serves an important protective function. The tenderness and increased sensitivity of tissue surrounding the site of an injury help to protect the site and prevent further damage.

Classification according to source

Sensory input from the body (soma) to the central nervous system has traditionally been referred to as somatosensory input. However, based on the current understanding of pain mechanisms as well as on the observation that the qualities of pain vary according to the site of pain origin, it is more appropriate to refer to pain as either somatic or visceral, depending on the origin of the pain. If somatic pain arises from skin, it is called superficial pain. If it arises from muscle, joints, or connective tissue, it is called deep pain. Visceral pain, which arises from internal organs, is different in significant ways from somatic pain and is considered a separate quality of pain.

Somatic pain

The skin is continuously exposed to the external environment and thus to a wide variety of stimuli. Consciousness of itch, touch, tem-

perature, pain, and the like are distinguishable from one another and are generally easy to localize. The skin is densely innervated by a wide variety of specialized sensory receptors, including nociceptors, which give a person the ability to distinguish light touch from noxious pinch and to localize the movement of even a single hair on the back of the hand. Superficial pain is thus best characterized by its ability to be easily and accurately localized. Because the skin is a protective barrier against the environment, noxious stimuli applied to skin typically evoke protective withdrawal reflexes.

In the case of deep somatic tissues (muscles, joints, connective tissues), the prominent sensation that reaches consciousness is pain. Most sensory nerve activity arising from muscles and joints does not reach consciousness, but joint sprains and deep muscle bruises can be very painful. Damage to a joint or muscle does not generally evoke the same type of withdrawal reflex associated with noxious stimulation of the skin, but pain from joints and muscles serves a similar protective function. Joints and muscles are innervated by nociceptors, and just as is true for skin, pain from these deeper structures is generally well localized. A person rarely attributes the source of muscle or joint pain to the wrong muscle or joint. Because pain from joints and muscles is commonly associated with tissue injury and inflammation, the sensitivity of nociceptors innervating the injured tissue increases, and the sensory channel for pain undergoes a reversible plastic change. One consequence of such plasticity is referral of pain to the overlying skin. Careful sensory examination of the overlying skin will reveal that its sensitivity to stimulation has increased and it has become hyperalgesic. In this example of an injured joint or muscle, referred pain and hyperalgesia help to protect the deeper structures from further damage.

Visceral pain

Visceral pain is a separate quality of pain that is in most ways unlike pain in skin, muscles, or joints. Visceral pain is diffuse and difficult to localize and is typically referred to other deep visceral or nonvisceral structures and to the skin. The complexity of visceral pain is illustrated by the pain associated with insufficient oxygen supply to the heart, angina pectoris. Anginal pain is typically referred to the skin and muscles of the upper left chest, shoulder, and arm. A similar pattern of referred pain is produced by obstruction of the gall bladder or by pain arising from the esophagus. Esophageal or gallbladder pain is typically confused with anginal pain. Thus, visceral pain is often not correctly localized to the site of its cause.

Whether the viscera are innervated by nociceptors has long been argued and remains uncertain at present, but it is apparent that the sensory-discriminative component of visceral pain is poorly developed with respect to localization of the source. Because a person usually has

little or no previous experience with visceral pain and cannot easily assess the significance of the pain, the motivational-affective component of pain arising from internal organs is significant. Many people experience acute visceral pain on occasion that passes without concern. The common response to repetitive or aching visceral pain, on the other hand, is fear of a serious disorder or disease. Indeed, among the most intense chronic pains are those associated with tumor growth that distends and distorts a viscus.

Not easily placed in the categories described above is the pain arising from veins and the pain referred to as migraine headache. Veins, but not arteries, appear to be innervated by sensory nerve endings that function like nociceptors when a vein is distended or exposed to a cold infusion or a solution with low pH. Accordingly, intravenous drug administration or infusions can be unintentionally painful. Migraine headache has long been considered to be a disorder of the brain and meningeal vasculature. Recently, however, it has been established that many headaches are neurogenic in that the neural innervation of the meningeal vasculature and sinuses initiates the events that produce headache. Indeed, headache resembles visceral pain in many ways in that it is diffuse in character, difficult to localize, and referred to other structures, such as the eyes, teeth, or head and neck muscles.

Four neural stages of nociception

Functionally, nociception can be divided into four stages:

- transduction—the conversion of stimulus energy into neural activity
- central processing and abstraction—the processing of nociceptive neural signals by the central nervous system to extract relevant information
- modulation—the adaptation of nociceptive activity to changes in the environment and needs of the individual
- development and plasticity—long-lasting or permanent changes in the neural mechanisms that mediate nociception in response to development, experience, and injury.

Each of these stages will be considered in detail below.

Transduction

The first stage in nociception is the reception of a nociceptive stimulus by a primary afferent neuron, which consists of a specialized receptor that transduces somatic or visceral stimuli, an axon that conveys the electrical information toward the spinal cord or cranial nuclei, and the