Chapter 5

Pain Management

James R. Miner and John Burton

Perspective

Pain-related complaints represent as many as 70% of presenting complaints for patients in the emergency department (ED) setting.[1] Uncontrolled pain should be considered a medical emergency. The estimated degree of pain experienced by a patient should play a role in the determination of a patient's overall acuity and urgency for therapy. Pain intensities, from both provider- and patient-derived scales, should be obtained and recorded for patients as frequently as one obtains or patient indicator. Although pain can be present in a wide variety of physical and psychosocial situations, it is almost always present in the context of tissue injury. Pain can therefore be assumed to be present in patients with physically apparent disease or injury, even in those who cannot effectively communicate their conditions. Important terms relating to analgesic practices are listed in Box 5-1.

A wide variety of options are available for the treatment of pain. Despite having effective treatments available for both acute and chronic pain therapy, the treatment of pain can be difficult and is often one of the most challenging and frustrating aspects of the practice of emergency medicine.[2]

Patients' perceptions of their ED care are highly influenced by pain treatment. Satisfaction with emergency care often depends on the techniques and timeliness of analgesia as well as the discharge plans for pain relief.[3–5] In every interaction with a patient in pain, a balance should be achieved between relief of patient suffering and the diagnosis and treatment of the underlying medical condition.

A growing body of evidence supports the importance of pain management as a central aspect of disease treatment. Unrelieved pain is associated with a variety of potentially negative physiologic outcomes, including increases in sympathetic outflow, peripheral vascular resistance, myocardial oxygen consumption, and the production of carbon dioxide (CO$_2$). Other adverse effects of unrelieved pain appear to include hypercoagulability, decreases in gastric motility, and immune dysfunction impairment.

Poorly treated acute pain can promote the development of chronic pain syndromes and cognitive symptoms, as well as increase the need for pain management during any recovery period.[6–8] Pain during serial medical procedures may increase if successful analgesia was not provided during initial procedures.[9] It is also likely that a patient's experience of pain increases the ability to perceive pain from similar stimuli in the future.[10]

As an affirmation of the recognized importance of pain management in health care, The Joint Commission (TJC) requires hospitals to develop quality improvement efforts related to acute pain management in addition to comprehensive programs for the measurement, documentation, and treatment of pain. Improvements in pain management are occurring as a result of an enhanced interest in pain research, education, and regulatory efforts.[11,12]

Pathophysiology

Pain can be generally described as nociceptive or neuropathic. Nociceptive pain results from the activation of sensory neurons that signal pain (nociceptors) in response to noxious stimuli. Neuropathic pain results from altered processing changes in the central nervous system (CNS). Neuropathic pain is usually described as burning, tingling, or shooting sensations and includes neuropathies and deafferentation. Both nociceptive and neuropathic types of pain involve peripheral and central sensitization with complex series of mediators to sensitize peripheral nociceptors and perpetuate (balance) signals shown in Figure 5-1. At each level in the physiologic process of pain production or transmission, interventions and therapeutic opportunities should be considered to alter the process and ultimately improve the patient's pain experience.

Pain Conduction Pathways

Pain perception can be divided into three separate processes (see Fig. 5-1): pain detection (transduction), pain transmission (conduction), and pain appreciation (perception). The transduction of painful sensory input is initiated by the activation of nociceptors, with subsequent depolarization of their axons. The axons then relay information (afferent input) to their cell bodies located in the dorsal root ganglion, lateral to the spinal cord.[13] Central duration of these first-order neurons synapse in the dorsal horn, where sensory input is modulated. These sensory signals travel through the CNS (spinothalamic tracts and projection columns) to synapse in the reticular system and the thalamus. Thalamic and reticular signals are then projected to the cerebral cortex (Fig. 5-2).

Pain Detection

The somatosensory system is responsible for the detection of pain as well as tactile, proprioceptive, and thermal sensations. Receptors responsible for the detection of pain are termed nociceptors. Nociceptors include sensory nerves that are capable of detecting mechanical, thermal, or chemical stimulation. Several different subtypes of nociceptors are present in cutaneous tissues, including mechanonociceptors, polymodal nociceptors (PMNs), and a variety of chemonociceptors.[14] Most nociceptive input is derived from inflammatory mediators through PMNs in response to chemical, thermal, and mechanical stimuli.

The threshold of activation of a nociceptor can be modulated, increased or decreased, by a variety of chemical mediators including prostaglandins, cyclic adenosine monophosphate, endothelins,