Understanding and Managing Patients with Chronic Pain

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• Analgesics

Dentistry has long been associated with the management of patients suffering from pain. Indeed, many of the advancements in early forms of anesthesia were discovered by and used by dentists in an effort to control the inevitable pain associated with dental procedures, predominantly dental extractions. Pain and anxiety control have remained a staple of dental education at the predoctoral and postdoctoral levels. Specifically, the specialty of oral and maxillofacial surgery has had at its core the foundations of anesthesia and pain and anxiety control. The specialty has continued to research and use various treatment strategies in the management of patients' pain. However, along with daily successes, every practitioner must remember that pain is diverse and complex. Research and treatment experience are expanding our knowledge of the mechanisms producing the experience of pain, and have revealed clinically significant differences between acute pain at one end of the pain spectrum and chronic pain at the other. Because most oral and maxillofacial surgeons manage acute pain and anxiety as part of the fabric of daily practice, clinicians must not bias diagnostic and management strategies in terms of acute pain paradigms. For chronic pain, as with all other conditions to be managed, a thorough understanding of pathophysiology is the foundation needed for effective management.

Previous editions of the *Oral and Maxillofacial Surgery Clinics of North America* have covered the diagnosis and management of chronic facial pain extensively. In fact, the *Clinics* were an important reference source used while researching for this article. The reader is well advised to refer to these reference sources to compile a complete database on this topic.[1–12] Rather than restating this material, the goal of this article is to refamiliarize the reader with clinical pearls helpful in the management of patients with chronic pain conditions. The authors also hope to highlight the

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interplay of chronic pain and psychology as it relates to the oral and maxillofacial surgery patient. To that end, this article outlines and reviews the neurophysiology of pain, the definitions of pain, conditions encountered by the oral and maxillofacial surgeon that produce chronic pain, the psychological impact and comorbidities associated with patients experiencing chronic pain conditions, and concepts of multimodal treatment for patients experiencing chronic pain conditions.

PAIN THEORIES

Pain is a ubiquitous experience and a necessary function in human existence. Attempts to explain pain have been recorded throughout history and across many cultures. Early explanations were based on observations and superstitions. One of the first recorded theories that attempted to explain pain through scientific thought was the specificity theory.

Specificity Theory

In the 1600s, Descartes[13] proposed the specificity theory of pain. The theory postulated that a specific set of cutaneous receptors delivers signals from the periphery to the brain. When stimulated, these receptors—cold, warm, pain, and touch—signaled the brain with their specific messages. This theory presumed that receptors are differentiated and specialized and that there is a one-to-one relationship between the intensity of the particular stimulus and the experience of the particular sensation.

Pattern Theory

Another early theory, the pattern theory, proposed that receptors were undifferentiated, and that a specific pattern of stimulation was transmitted from the periphery directly to the brain. It was this "pattern" of stimulation that the brain would decipher as pain, pressure, cold, hot, and so forth. which explained the experience of pain.[13]

Gate Control Theory

Although commendable for their attempts to explain pain transmission scientifically, the specificity and pattern theories could not account for many conditions seen in clinical practice. In 1965, Melzack and Wall[13] proposed the gate control theory of pain, which proposes the existence of a "gating" mechanism between the sensory stimuli in the periphery and the processing of the stimuli within the central nervous system. The "gate" was thought to be in the substantia gelatinosa layer in the dorsal horn of the spinal cord, where the various afferent (sensory) fibers terminate. This mechanism was thought to modulate nociceptive input entering the central nervous system. The theory proposes that nociceptive and non-nociceptive input from the periphery is transmitted to the substantia gelatinosa where the signals compete with each other. The non-nociceptive input activates interneuronal systems that inhibit transmission of the nociceptive signals to higher levels in the nervous system.

Additional modulation is then provided through downward signals from the central nervous system (CNS). The brain processes nociceptive input and influences further transmission by "signaling" down to the "gate." This process provides an opportunity for a patient to influence the pain response by altering these downward signals. In short, this is a neuroanatomical explanation for the "mind-body" connection. Although current pain theory differs somewhat from the ideas of Melzack and Wall, it is their work that established a strong foundation for our current understanding of how pain is perceived.

THE NEUROPHYSIOLOGY OF PAIN

Pain is initiated by the excitation of nociceptors, receptors that respond to noxious stimuli. Nociceptors are free nerve endings found in abundance in the skin, periosteum, arterial walls, joint surfaces, falx cerebri, and the tentorium cerebelli.[14] Deep structures are much more sparsely innervated. The impulses from the stimulation of these receptors are carried to the CNS in one of two ways: fast transmission of fast pain and slower transmission of slow pain. In fast pain, impulses are carried on larger diameter, myelinated peripheral nerves called Aδ fibers at about 6 to 30 m/s; this produces a pain response in approximately 0.1 second. Fast pain is usually described as sharp, pricking, or electrical.[14]

Slow pain impulses are carried from the periphery on smaller, unmyelinated fibers known as type C fibers. The speed of transmission ranges from 0.5 to 2 m/s. In these nerves, the pain response occurs in approximately 1 second or greater and can be more sustained. The clinical quality of this pain has often been referred to as burning, aching, throbbing, or nauseating.[14]

Excitation of nociceptors is produced by mechanical, thermal, and chemical stimulation. Chemical stimulation is related to substances such as bradykinin, histamine, serotonin (5-hydroxytryptamine; 5-HT), proteolytic enzymes, acids, potassium ions, and acetylcholine. In the periphery, other substances, namely substance P and prostaglandins, sensitize the receptors