Chronic Pain in Neurosurgery

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KEYWORDS

• Chronic pain • Neurosurgery • Multimodal analgesia • Spinal surgery • Craniotomy • Adjunctive analgesics • Opioid sparing

KEY POINTS

- Chronic pain is an experience resulting from a wide variety of derangements leading to the abnormal processing of pain.
- Providers caring for patients with chronic pain undergoing neurosurgery should use multimodal analgesia strategies to improve perioperative comfort and reduce the risk of postoperative sedation.
- There are few studies that have investigated specific anesthetic approaches for patients with chronic pain, and this requires a personalized approach to perioperative care.

INTRODUCTION

Providing safe and effective care for patients with chronic pain undergoing neurosurgery requires a carefully planned anesthetic strategy. If the anesthesiologist errs on the side of safety through conservative analgesic dosing, patients may emerge with harrowing pain, accompanied by unstable hemodynamics that may compromise the delicate operation. On the other hand, if the provider overdoses patients with opioids and other sedatives, patients may be too impaired to comply with the postoperative neurologic examination, may sustain respiratory failure, or the adrenergic response may be blunted and, thus, compromise perfusion to the spinal cord or brain.

Chronic pain has an estimated prevalence of 30% of the general population or 100 million people in the United States.[1,2] A proliferation of opioid prescribing has paralleled this epidemic as there has been a 4-fold increase in opioid prescriptions from 1999 to 2010.[3] A 2011 to 2012 survey reported 6.9% of adults taking an opioid in the last 30 days.[4] The implications of chronic opioid therapy (COT) and the pathophysiology of complex pain processing are separate but have overlapping clinical challenges. Pain is first and foremost an experience and should be understood in a biopsychosocial context that is influenced by musculoskeletal, nervous system,

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emotional, and environmental factors. Patients undergoing craniotomies for tumor staging or debulking may present with diffuse, severe cancer-related pain and patients with abnormal spine conditions may have associated chronic pain. Not surprisingly, there is a particularly high occurrence of chronic pain in patients undergoing lumbar and cervical surgery.[5]

There is an association with altered pain perception and patients living with chronic pain. Experimental human studies have measured lower pain thresholds with pressure,[6] cold,[7] and heat[8] stimuli in chronic pain conditions compared with healthy controls. Prediction models consistently document chronic or preexisting pain as an independent risk factor for poorly controlled postoperative pain.[9–11] Interestingly, Chapman and colleagues[12] demonstrated that patients with chronic pain on COT reported clinically significant greater pain levels up to 15 days postoperatively compared with patients with chronic pain off long-term opioids.

Investigations into models of pain continue to evolve as newer evidence elaborates on older theoretic mechanisms. A quantum leap in the field occurred in 1965, when Melzack and Wall[13] published the gate control theory of pain (**Fig. 1**A). Previously, pain was described as a unidirectional pathway that was first transduced by small nociceptive peripheral nerves (C- and A-delta fibers) that were transmitted to afferent pathways along the spinal thalamic tract in the spinal cord before synapsing on neurons in the brain where pain perception occurs. According to the gate control theory, pain is not an unimpeded pathway from periphery to brain but results from an interaction between ascending afferent input carried by small-diameter nerve fibers and descending, inhibitory input carried by larger-diameter nerve fibers. Specialized neurons in the brain and spinal cord create an elaborate neural network that exerts a tonic inhibitory effect on interneurons that synapse in the substantia gelatinosa of the dorsal horn. According to Melzack and Wall,[13] "the substantia gelatinosa acts as a gate control system that modulates the synaptic transmission of nerve impulses from peripheral fibers to central cells."[13]

Various pain pathologies may be explained by an abnormally opened gate, where chronic inflammation or other neurologic derangements may create an imbalance of excitatory and inhibitory signaling. A windup of the dorsal horn has been demonstrated in some neuropathic pain states, which may give rise to the clinical findings of hyperalgesia or allodynia, whereby patients show an exaggerated response to light noxious or non-noxious stimuli, respectively.[14]

Decades after the gate control theory was introduced, researchers, including Melzack, have shifted attention from the dorsal horn to the brain. A new model proposes the presence of a "pain neuromatrix", which describes sensory and cognitive processes imprinting a representation of the "body-self" through a widely distributed neural network (**Fig. 1**B).[15,16] In this view, chronic pain is the output of the pain neuromatrix whereby nociceptive and non-nociceptive input results in an amplified experience of hurting.[17] Supporting this theory, brain imaging studies have correlated structural and functional differences in the anterior cingulate, prefrontal cortex, insula, somatosensory cortex, and parahippocampal gyrus in patients with fibromyalgia and central sensitization.[18,19]

Anesthesiologists must understand that pharmacologic regimens, which predictably treat acute pain in most adults, may fail in certain patients with complex derangements of their pain processing network. This article reviews perioperative management principles for chronic pain and focuses on clinical evidence emerging from neurosurgical studies. It is hoped that the reader will enrich his or her understanding of chronic pain and identify a broader palette of strategies that may be used to personalize a multimodal plan for patients undergoing a spinal or cranial procedure.