The Place of Sedation in Dentistry: Controlling Acute Pain by Local Anesthesia Is Not the End of the Story

Local anesthesia is effective, and dentists nowadays are well-trained for controlling the patient's acute pain. In most cases, however, practitioners provide few approaches to suppress the stress response or limit its consequences. The current general thinking is that the practitioners' goal should be to give the best possible care, from a technical point of view, while controlling patients' pain. Although most dental care users are satisfied with this approach, there is a significant percentage of the population who still consider dental care to be the most stressful event of all health-care treatments.[1] Therefore, the common belief that dental treatment–induced fear is an old, outmoded problem is wrong.

Basic sciences have described in detail the deep relationships that closely link the physiology of nociceptive pain with the physiological response to acute stress. Chapman et al have recently described these relationships from the perspective of reciprocal interactions of the neural, endocrine, and immune systems.[2] The role of each of these three systems is well known. The nervous system detects threats and actual injuries and induces protective motor reactions. The endocrine system induces a stress response through the hypothalamic-pituitary-adrenocortical (HPA) axis and the sympathoadrenomedullary axis. The release of catecholamines by sympathetic efferent endings participates in this response to stress. The immune system detects other levels of threat before addressing them by the inflammatory response and wound healing. Chapman et al[2] have pointed out the deep interdependence between these three systems. For example, it is well known that neural structures initiate hormonal responses such as β-endorphin or adrenocorticotropin hormone from the same pituitary cells and that these same substances act as neuromediators in the central nervous system. Other examples are stress-induced analgesia and hyperalgesia, the mechanisms of which probably rely on the descending pain-control systems and the sympathetic system which participates directly in both the response to nociceptive stimulation and the stress response. Shared mechanisms exist at the peripheral and central nervous system levels, with interactions between the different ligands and receptors that make possible an adapted response to any stressor including nociceptive pain.

The causal or synergic relationship between pain and the stress response is also well established. Many pain conditions are considered stress-related syndromes, including fibromyalgia, irritable bowel syndrome, chronic headaches, dysmenorrhea, and temporomandibular disorders.[3] In several of these conditions, patients show a reduced threshold to noxious stimuli and a heightened sensitivity to stress. The prevalence of post-traumatic stress disorder was shown to be considerably higher in orofacial pain and fibromyalgia patients than in the general population.[4,5] Early life stress has been suggested as a major risk factor for triggering post-traumatic stress disorder because of the acquired maladaptive HPA axis response to stress, which can be "memorized" in the HPA axis as long-term changes. For example, Essex et al have shown that exposure to early maternal stress, which induces higher baseline cortisol levels in children, may sensitize their pituitary-adrenal responses to subsequent stress exposure.[6] In later life, these children may react to stressful life events with increased circulating cortisol. Subsequently, these patients with early or/and chronic life difficulties may display impaired behavioral and pain responses to ordinary late-life stressful events.

In contrast, the dental literature is limited in the clinical interactions between nociceptive pain and responses to acute stress. As a result, the clinical consequences of these interactions have been underestimated. In persons showing a certain level of anxiety, the exposure to dental care procedures, especially when repeated, constitutes an acute stress stimulus which may aggravate anxiety and render analgesic procedures more difficult. Similarly, an aggravated anxiety level has been proposed to explain the increased rate of anesthesia failure in cases of acute irreversible pulpitis.[7] In addition, stress experience may act like an acute pain experience and induce a long-term hyperalgesia.[8] Consequently, the concept of preventive analgesia, which is based on the control of the effects of a conditioning nociceptive stimulus, should probably be extended to clinical cases in which an acute stress stimulus is the conditioning stimulus. Sedation would be the natural agent to carry out this preventive analgesia which could be effective not only on pain sensitization, as suggested by animal studies, but also on long-term anxiety levels.[9]

In general surgery, regional anesthesia and sedation are routinely applied together to control the consequences of stress. In dentistry, sedation is considered more as an alternative to general anesthesia to facilitate dental treatment, and its positive impact on physiological consequences of pain and stress are underestimated. After several decades of great improvement in dental analgesia, it is time now to switch the emphasis toward the control by sedation of the consequences of dental stress.

Alain Woda, DDS, PhD
Associate Editor

Martine Hennequin, PhD, HDR
Université d'Auvergne

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