

New insights in the pharmacological treatment of pain in dentistry.

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Pain may be described as a response to the sensitization of specific peripheral neurons, the nociceptors. These sensations are encoded by the peripheral nervous system (PNC) and central nervous system (SNC) by a conversion of all the information into frequency-modulated signals.

The comprehension of its pathophysiology permitted unveiling the protective role of this mechanism in maintaining body homeostasis and to protect against damaging stimuli.¹ Furthermore, most of the pain occurring in dentistry including dental pain, trigeminal neuralgia, temporomandibular joint pain, muscle pain and headache are associated to the structures that are covered by sensory innervation from the trigeminal nerve. Moreover, effective pain control stills remain a challenge to the dental practice, especially, when referring to post-operative conditions when the patient's cooperation and anxiety are limiting conditions.

Analgesia may be achieved with pharmacological interventions that include different analgesic therapies (*e.g.* non-steroidal anti-inflammatory drugs (NSAIDs), opioids, NMDA receptor blockers) and drugs regimens (*e.g.* preemptive, perioperative, postoperative analgesia). Depending on the nature and intensity of the expected pain different pharmacological protocols are recommended and in some situations, we may focus in initiating treatment before the signals are transmitted to the CNS, what is called preemptive analgesia.²

The idea is based on the concept that noxious signals should be blocked prior surgical assessment and this may prevent the input from the periphery and it continues throughout the healing process.

The pharmacological protocol may vary depending on the patients medical conditions and required treatment, but a classical intervention may include the use of a preoperative dose of a traditional NSAIDs *e.g.* ibuprofen, diclofenac, piroxicam, etc.

The oral administration of coxibs (*e.g.* celecoxib 100mg, 200mg or 400mg) may be effective in controlling pain in dentistry and in terms of pharmacokinetics it is a good option for the clinician due to its rapid absorption and only a 2 to 4 hours delay to achieve the maximum serum concentration.

Furthermore, the drug is well tolerated without food and has low occurrence of side effects, and does not interfere with platelet

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aggregation, preventing hemorrhagic episodes. Combining pre-incisional long-lasting local regional block anesthesia with 0.5% bupivacaine 1:200.000 epinephrine seems to decrease the perception of postoperative pain³ as a safe and valuable alternative to use as a coadjuvant to anti-inflammatory agents. Other protocols may consider nitrous oxide analgesia during surgeries or even the use of corticosteroids targeting

swelling and consequently avoiding postoperative pain.⁴

During the last years, preemptive analgesia has been questioned for its real efficacy,⁵ but what we should consider to establish certainty, and in developing individual protocols for our patients we do not discard the possibility of introducing multimodal analgesic techniques or even promote synergic antinociceptive drug effects.

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