HOT TOOTH: A REVIEW

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ABSTRACT

The successful management of pain have been one of the cornerstones of endodontic practice and dentistry worldwide since time immemorial. Achieving profound pulpal anesthesia not only helps patients overcome their fears and displeasures towards dentistry but also favours the dentists who will be less stressed worrying about the patient's reaction or sudden movement during the treatment procedure. But, achieving adequate anesthesia in patients with a hot tooth can be a challenge. This article describes the hot tooth, reasons for anesthetic failure and some of its management strategies.

Key Words: Anesthesia; hot tooth; supplemental injection; irreversible pulpitis

INTRODUCTION

The successful use of local anesthesia has changed the emotional appeal of clinical dentistry from being a painful and dreadful experience to being a much less painful and pleasant experience. Moreover, optimum pain management results in building up trust and facilitates the entire procedure (Walton et al., 2008). However, local anesthetics commonly fail in endodontic patients with a hot tooth.[1]

To begin with, it is necessary to define what a ''hot'' tooth really is. In endodontic terms, hot tooth certainly does not mean a tooth of extreme attractiveness or even a tooth that is undergoing an exothermic reaction in which its temperature is well above the normal body temperature. But, it generally refers to a pulp that has been diagnosed with irreversible pulpitis (Symptomatic Irreversible Pulpitis) with spontaneous, moderate-to-severe pain. A classic example of one type of hot tooth is a patient who is sitting in the waiting room, sipping on a large glass of ice water to help control the pain.[1]

REASONS FOR ANAESTHETIC FAILURE IN PATIENTS WITH A HOT TOOTH.

Anesthetic failures after inferior alveolar nerve block (IANB) have been reported to be between 44% and 81%.[2,3,4,5]In cases of irreversible pulpitis, the rate of success is reported to be as low as 20%.[6]Similarly, the failure rate of a maxillary infiltration injection is as high as 30% in teeth with irreversible pulpitis.[7]

A number of explanations have been proposed for this-

1) Conventional anesthetic techniques do not always provide profound pulpal anesthesia, and patients with pre-existing hyperalgesia may be

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unable to tolerate any noxious input.[8]

2) Inflamed tissue has a lower pH, which reduces the amount of the base form of anesthetic that penetrates the nerve membrane. Consequently, less of the ionized form is available in the nerve to achieve anesthesia. This explanation however does not account for the mandibular molar with pulpitis that is not readily blocked by an inferior alveolar injection administered at some distance from the area of inflammation.[1,8]

3) Another theory is that, the nerves arising from the inflamed tissue have an altered resting potentials and reduced thresholds of excitability [8,9,10] because of which, the prevention of transmission of nerve impulses by the anesthetic agents is impeded.[9,11]

4) Another factor might be the tetrodotoxin resistant (TTX-R) sodium channels. These channels are relatively resistant to local anesthetics, [12,13] are sensitized by prostaglandins[14] and are increased in inflamed dental pulp.[15,16] They are four times as resistant to blockade by lidocaine and their expression is doubled in the presence of prostaglandins E2 (PGE2).[17] The sensitization of these channels by prostaglandins suggests that, rapid-acting non steroidal anti-inflammatory drugs (NSAIDs) may be useful in pretreatment to enhance the efficacy of local anesthetics in patients with odontogenic pain .[6,18]

5) Sensitization of TTX-R channels by prostaglandins also lowers the activation threshold of voltage-gated sodium channels (VGSCs) and hence increases the amount of sodium ions that flow through the channel.[14,18]

6) Activation of nociceptors in the presence of inflammation is one of the strongest theories explaining the reduced efficacy of anesthesia.[19,20] Inflammatory mediators reduce the stimulation threshold in nociceptor neurons to a level at which the slightest stimulators induce a severe neurogenic response (Goodis et al., 2006). This inflammatory process occurs as a result of the production of prostaglandins (PGs) as the end point product of the metabolism of arachidonic acid through the cyclooxygenase pathway (COX). Prostaglandins (PGs) then result in increased sensation of pain by increasing the sensitivity of the nerve endings to bradykinin and histamine (Dray, 1995).

7) Finally, patients in pain often are apprehensive, which lowers the pain threshold.[8]

MANAGEMENT STRATEGIES IN PATIENTS WITH A HOT TOOTH.

When irreversible pulpitis is a factor, the teeth that are most difficult to anesthetize are the mandibular molars, followed by the mandibular premolars, the maxillary molars, the premolars and the mandibular

anteriors. The fewest problems arise in the maxillary anterior teeth.[8]

PRE-MEDICATION

In symptomatic teeth with irreversible pulpitis , one theory regarding the high rate of local anesthetic failure is the prostaglandin induced sensitization of peripheral nociceptors.[19,20,21] Peripheral terminals of nociceptors express receptors that can detect chemical and physical stimuli which results in the activation of various ion channels. Inflammatory mediators such as prostaglandins produce their effects by binding to various protein receptors. Hence, interventions that decrease the overall concentration of prostaglandins, leads to reduced activation of these receptors.[20,22,23]Therefore, it appears logical that if the production of prostaglandins is interrupted, it may increase the efficacy of local anesthetics.[24]

A meta analysis on the effect of pre-medication with NSAID's on the success of IANB showed that, dosages of 600-800 mg of ibuprofen , 75mg indomethacin ,8mg lornoxicam and 50 mg of diclofenac potassium significantly increased the success rate of IANB ,whereas other NSAID's such as ketorolac ,a combination of ibuprofen and acetaminophen, as well as acetaminophen alone had no significant effect on the success of anaesthesia compared to placebo.[25]

However, controversy exists for the use of analgesics prior to IANB. Several researchers including Aggarwal et al. (2009), Oleson et al. (2010) and Simpson et al. (2011) reported an insignificant improvement in the success of IANB accompanied by premedication with analgesics in treating irreversible pulpitis.

MANDIBULAR TEETH [8]

In a patient with hot tooth, local anesthesia produced the classic soft-tissue signs and relieved the painful symptoms, but pain frequently resulted when the access opening was begun or the pulp was accessed. Hence, after administering conventional anesthesia and observing the signs of soft-tissue anesthesia (which is required for a successful supplemental injection), the clinician administers a supplemental *intraosseous injection* (IO). 1.8 ml of 3% mepivacaine plain (e.g., 3% Carbocaine) has been recommended for this procedure. There are several IO systems available in the market, including the Stabident system (Fairfax Dental Inc, Wimbledon, UK), X-Tip system (Dentsply, York, PA, USA), the IntraFlow handpiece (Pro-Dex Inc, Santa Ana, CA, USA) and the comfort control syringe (Dentsply, York, PA, USA).

After anesthesia has been given, the rubber dam is placed, and the access preparation is slowly begun. If pain occurs, the rubber dam is removed and another cartridge of 3% mepivacaine administered, which should then be successful. Again, the clinician should make sure that lip numbness has developed from the IAN block and that the anesthetic solution is deposited into the medullary bone. If the patient still has pain, an *intrapulpal injection* is given.

For mandibular teeth with irreversible pulpitis, the supplemental intraosseous injection has evolved into a routine second injection, which is made before the clinician places a rubber dam and begins the access opening preparation.

Other supplemental injections:

- *Buccal infiltration (BI)* with 4% articaine with 1:100,000 epinephrine . Its administration after IANB increased pulpal anesthetic success by 17%–36%.[26,27]Articaine has been found to be 3.8 times more effective for infiltration than lidocaine.[28]

- *Intraligamentary (Periodontal Ligament) Injection* with 2% lidocaine with 1:100,000 epinephrine.[1] Cohen and colleagues[2] reported that, the supplemental PDL injections were successful 74% of the time, whereas reinjection boosted success to 96%.

PDL injections are usually given using either a standard dental anesthetic syringe, a high-pressure syringe or the Wand.[1]

MAXILLARY MOLAR AND PREMOLAR TEETH

The initial anesthetic dose of 2% lidocaine with 1:100,000 epinephrine is doubled (to 3.6 ml) for the buccal infiltration.[8,29] Administration of an intraosseous injection before proceeding with access may prove helpful in anesthetizing these teeth. If the patient experiences pain during the later stages of instrumentation, an additional infiltration injection is necessary. Occasionally, pain is experienced in the palatal canal of molars. Infiltration over the palatal apex with 0.5 ml of anesthetic solution enhances pulpal anesthesia in these cases.[8,30]

MAXILLARY ANTERIOR TEETH

Articaine with 1:100,000 epinephrine may be used initially for labial infiltration.[8,31] Although supplemental anesthesia is not often necessary, the intraosseous injection should be successful when given. The duration of anesthesia may be less than 1 hour. An additional infiltration injection may be necessary if the patient experiences pain during the later stages of instrumentation.[8]

SYMPTOMATIC TEETH WITH TOTAL PULP NECROSIS AND PERIRADICULAR ADIOLUCENCIES [8]

For mandibular teeth, the IAN block (and long buccal injection) are given in all situations. For maxillary teeth with no swelling, anesthesia is administered with conventional infiltration or a block. If soft-tissue swelling is present (i.e cellulitis or abscess) infiltration should be done on either side of the swelling, or a block should be administered (second division nerve block, PSA nerve block or infraorbital nerve block). These provides some degree of bone and soft-tissue anesthesia. After signs of anesthesia are observed, the rubber dam is placed, and the access is begun slowly.

Occasionally, the conventional injections do not provide profound anesthesia particularly in the maxillary teeth. In these cases, careful consideration of a supplemental injection consisting of an additional palatal infiltration injection may be helpful.

PAIN REDUCTION IN IRREVERSIBLE PULPITIS WHEN

ENDODONTIC TREATMENT IS IMPOSSIBLE

Endodontic debridement (pulpectomy or pulpotomy) most predictably relieves the pain of irreversible pulpitis.[32] When debridement is not possible clinicians may prescribe strong analgesics and penicillin in an attempt to relieve the pain. Intra osseous injection of Depo-Medrol has been found to reduce the pain to manageable levels for up to 7 days before the patient received endodontic treatment, supporting this as a method for controlling a patient's pain until definitive endodontic treatment can be performed.[8]

CONCLUSIONS:

Providing profound anesthesia is the ultimate goal of every dental practitioner. However, the clinician who treats patients diagnosed with a hot tooth (irreversible pulpitis) often finds achieving adequate pulpal anesthesia to be a challenge. Hence, the clinician should have fall back strategies to attain good pulpal anesthesia when failure of the traditional techniques are encountered. This will boost the confidence of the clinician to impart and provide a relatively pain free treatment for the patients having a hot tooth.

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