

Hot Tooth and Its Management

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ABSTRACT

Managing a "hot tooth" — a tooth with acute, often intense pain due to inflammation or infection of the pulp — remains one of the most challenging situations in dental practice. The primary goal is to relieve the patient's pain while managing the underlying pathology effectively to prevent recurrence. This paper discusses the etiology of hot teeth, highlighting the role of pulpitis and periapical inflammation, and reviews evidencebased approaches to treatment. Techniques like local anesthesia optimization, adjunctive pharmacological support, and single-visit versus multiple-visit root canal treatment are explored. Additionally, newer approaches involving supplemental anesthesia techniques (e.g., intraosseous injections, PDL injections) and various pharmacologic aids, such as NSAIDs and corticosteroids, are evaluated for efficacy in managing persistent pain. By combining clinical judgment with advances in anesthesia and pain management, practitioners can achieve more predictable and effective outcomes for patients suffering from hot teeth. This review aims to provide clinicians with practical strategies to improve patient comfort, procedural efficiency, and overall treatment success in managing hot teeth.

I. INTRODUCTION

Teeth are usually described as "hot" when the pulp is diagnosed with irreversible pulpitis and is undergoing spontaneous, moderate to severe discomfort. One typical instance of a hot tooth is a patient who is waiting in the waiting area and consuming a large glass of ice water to ease the ache. According to endodontic terminology, a "hot tooth" is unquestionably not a tooth that is overly beautiful or even one which is experiencing an exothermic reaction, in which case its temperature is significantly higher than the average body temperature. But usually, it refers to a pulp that has been diagnosed with either irreversible pulpitis with symptoms or irreversible pulpitis with moderate to severe pain that occurs on its own. [1]

Prior to beginning endodontic therapy, it is imperative that a tooth with an irreversible pulpitis diagnosis and moderate to severe pain have sufficient pulpal anesthetic. The endodontic therapy must always start with administering anesthetic for that tooth. To get a decent anesthetic, one must be well-versed in both the process and the correct anatomy. Pain can sometimes cause a patient to react badly to treatment even after receiving the appropriate anesthetic. This condition is commonly referred to as "Hot Tooth" in the field of endodontics. The clinician will always find it difficult to treat such a problem. [2]

HOW TO IDENTIFY HOT TOOTH?

The most typical places for both primary and permanent teeth to occur are: Locations of recent or flawed repairs sites affected by recent trauma It is more difficult to anesthetize mandibular molars. Patients who have had discomfort for multiple days or who are anxious about receiving dental treatment typically need a more strategic approach. [3]

STRATEGIES FOR PREVENTING PAIN

Preemptive methods utilizing clinical methodologies, suitable local anesthetic, preemptive analgesia, and anxiety reduction techniques should be the foundation of pain preventive efforts. Painless endodontic treatment still depends on an effective anesthetic. It will also make the surgery easier for the operator to do while also keeping the patient comfortable.

It is far more difficult to achieve complete anesthesia in mandibular posterior teeth with irreversible pulpitis than in teeth with a normal, non-inflamed pulp. [4] [5] [6]

PREDISPOSING FACTORS FOR PAIN

Even when the patient appears to have enough anesthetic, there are a number of



recognized characteristics that predispose the patient to pain.

Genetics, psychological health, ethnicity, gender, age, and environment are some of these variables.

GENETICS AND GENDER

Certain patients may be more susceptible to pain, sluggish healing, and the development of abscesses due to genetics.It's clear that different genetic variants influence how people experience and respond to pain.

An increasing body of research suggests that men and women respond to pain differently inboth in clinical and experimental contexts. Many causes have offered for these findings, including hormonally and genetically driven sex differences in brain neurochemistry, which put women at a significantly higher risk for a wide range of clinical pain problems. [7]

PATIENT'S EDUCATION

In order to minimize anxiety, the patient should be knowledgeable about the treatment and have a clear mental picture of the processes involved. This will help to allay any fears of the unknown. [3]

MANAGEMENT OF ANXIOUS PATIENT

a) Scheduling brief morning visits following a hearty breakfast.

b) Premedication the night before bedtime and 90 minutes prior to the surgery with 1 mg of lorazepam (after confirming that it won't interfere with other medications).

c) You must be accompanied by a friend or relative and refrain from driving.

d) The waiting area is really tiny.

e) Duration: only as long as the patient is comfortable with ensuring the patient feels taken care of.

To make sure the anesthetic is working, a little test cavity might be created prior to starting the access preparation process.

To obtain a sound anesthesia, further anesthetic or additional injections are required. [3]

PREVIOUS DIFFICULTY WITH ANESTHESIA

According to reports, patients who have previously struggled with anesthesia are more likely to have a failure anesthetic. [8] Typically, these patients will say, "I always need more injections to get my teeth benumbed." One way to identify these people is to simply inquire if they have ever experienced trouble becoming numb. In the event that the operator has encountered these situations, they should promptly schedule an additional anesthetic on top of the initial one.

PSYCHOLOGICAL MANAGEMENT OF PAIN

Anxiety related to dentistry, fear of pain, expectation and anticipation of pain, unpleasant dental experiences in the past, attitudes toward the dentist, and attitudes toward the delivery of dental care in general are psychological factors that frequently contribute to pain. One very effective noninvasive method for treating acute dental pain is psychological intervention, which can take many different forms, such as sensory information, perceived control, positive dental experiences, or distraction techniques. [9]

In the meantime, pre-procedural sensory explanations about the procedure and predicted sensations, like a rotor noise, might be used as distraction techniques. Other examples of these might be visually stimulating stimuli, music, etc. The ability to halt the operator by raising their arms, which is a pause signal, allows patients to feel in control of the dentist's actions. Better cooperation can result from giving patients a positive dental experience and boosting the dentist's confidence by talking about the advantages of introduction to management. [10]

Wardle came to the conclusion that giving sensation information was the most successful psychological management strategy after looking into the effects of several methods on anxiety and discomfort during dental treatment. Patients receiving endodontic treatment reported less discomfort in a study by Logan et al. [11]

PREMEDICATION

Sodium channels that are resistant to LA and tetrodotoxin may be more prevalent in inflammatory pulps. Inflammatory pulps also have significantly higher levels of prostaglandins, which can affect tetrodotoxin-resistant receptors and reduce nerve responses to anesthetic medications.

Therefore, it seems that premedication corticosteroids and nonsteroidal with antiinflammatory drugs (NSAIDs) will improve anesthetic efficacy. However, there is disagreement among researchers regarding the influence of premedication anesthesia's on efficacy. Pretreatment with specific NSAIDs, however, may increase the efficacy of anesthesia for treating permanent pulpitis if the patient does not voluntarily suffer pain. An inferior alveolar nerve block (IANB) injection preceded by corticosteroid premedication before anesthesia demonstrated a noticeably greater success rate. [12] An



investigation of alprazolam premedication did not increase the IANB's success. [13]

WHY DO LOCAL ANESTHETICS FAIL IN A HOT TOOTH?

Anatomical reasons

The mandibular molars dense cortical bone prevents anesthetic penetration, whereas the maxilla's weaker bones allow diffusion. Failure to achieve anesthesia has been linked to cross innervations and accessory innervation from the contralateral inferior alveolar nerve (IAN). The effectiveness of local anesthesia is also influenced by foramina and tooth position. [14]

Inflammation and Tissue pH

Because inflammatory tissue has a lower pH, less base anesthetic enters the neuron membrane. Consequently, the nerve can achieve anesthesia with a reduced amount of the ionized form present. Tissue acidosis is unlikely to have an impact on block injections, as ion trapping is only feasible with infiltration injections. [15]

Effect of Inflammation on Blood Flow

The physiology of nearby tissues is also impacted by inflammation. Because inflammatory mediators produce peripheral vasodilation, local anesthetics are less concentrated at the site of action, their systemic absorption is negatively impacted, and the increased buffering capacity of inflamed tissues expedites the anesthetics disposal. [15]

Tetrodotoxin-resistant Channels (TTXr)

On C-fibers, there is a unique class of sodium channels called tetrodotoxin channels. Prostaglandin expression causes these C-fibers in the sodium channels to change from TTX sensitive to TTX resistant channels during inflammation. Inflamed tooth pulp has double the expression of TTX channels, which makes them local anesthetics resistant. This also enhances the influx of Na ions across nerve membranes by lowering the activation threshold of voltage-gated sodium channels (VGSCs) and prevents anesthesia from working. Tetrodotoxin-resistant channels are a reasonable explanation for the ineffectiveness of local anesthetics. [15]

Activation of Nociceptors

When there is inflammation, neuronal stimulation threshold is lowered by inflammatory mediators. Prostaglandins (PGs) are the end products of inflammation that are produced by the cyclooxygenase pathway. By making nerve ends

more sensitive to inflammatory mediators like bradykinin and histamine, prostaglandins heighten the experience of pain. [15]

MANAGEMENT OF HOT TOOTH

Alteration in Anesthetic Solution:

Dextamethasone is added to lidocaine (Twin mix): For SIP patients, the glucocorticoid dexamethasone successfully reduces pain. In addition to increasing sensory blocking, the lidocaine and dexamethasone combination also increases free base concentration and pH in the local anesthetic solution. There is less pain or burning sensation at the injection site due to the higher pH. [16]

Addition of mannitol to lidocaine:

Mannitol was added to lidocaine, which had the dual effects of expanding the perineurial membrane and making lipophilic substances like lidocaine more soluble. It also had the additional impact of preventing or reducing the propagation of action potentials in particular neurons. [17]

Clonidine as a vasoconstrictor alternative to epinephrine:

Clonidine is an alpha-2 adrenoceptor agonist that acts both centrally and peripherally and is used to treat hypertension. When compared to local anesthetics that contain epinephrine, clonidine directly inhibits C-fibers and A-delta fibers, vascular uptake is reduced in local anesthetics and increasing threshold of pain through peripheral blood vessel vasoconstriction. Because of its fewer cardiotoxic side effects, clonidine is regarded as a safe alternative to epinephrine as a vasoconstrictor. [18] [19]

Addition of magnesium sulfate:

One of the causes of anesthetic failure is the overexpression of N-methyl-Daspartate (NMDA) receptors through central sensitization. A noncompetitive NMDA receptor antagonist, magnesium sulfate inhibits nociceptor-induced central sensitization by interfering with NMDA receptors. [20]

Addition of sodium bicarbonate to local anesthesia:

By raising the dissociation rate and uncharged base form, alkalinizing local anesthetics can reduce discomfort. Buffered LA lessens the discomfort of injections, hastens the onset of anesthesia, and raises the success rate of anesthesia. [21]

Prewarming or preheating the anesthetic solution:



When compared to conventional LA, prewarming or preheating the anesthetic solution to 37–42°C has decreased intraoperative pain and discomfort. [22]

Cryotherapy

One of the well-established methods in dentistry for postoperative care and pain management is cryotherapy. When cold is applied to tissues, the temperature drops and blood vessels constrict.

The combined effect of cryotherapy and reduced release of chemical mediators involved in pain transmission results in a delay of brain signals. By decreasing the conduction velocity of pain signals and the activation threshold of nociceptors, cryotherapy also produces a local anesthetic effect. [23]

SUPPLEMENTAL TECHNIQUE:

ANESTHETIC

Intraseptal technique:

By directly injecting anesthetic solution through the crestal alveolar bone into the interdental septum, intraseptal anesthesia eliminates the requirement for a perforating instrument and simplifies the procedure by using an anesthetic needle to puncture the cortical plate. [24]

Intraosseous (IO) anesthetic technique:

This method uses a port or perforating system to deliver local anesthetic directly into the porous cancellous bone surrounding a tooth is known as the intraosseous (IO) anesthetic technique. Compared to IANB, the intraosseous anesthetic method had a better success rate, reaching up to 90% when used as supplemental anesthesia. [25]

Infilltration:

Research indicates that the length of pulpal anesthesia is significantly longer. The best way to guarantee deep local anesthesia is with additional intraligamentary or intraosseous injections. By using the intraosseous technique, analgesic medication can be applied straight to the cancellous bone surrounding the tooth apices. It has a quick onset and has proven to be a very effective supplemental analgesic for hot teeth. [26]

Intraligamental:

Analgesics are deposited intraligamentally, or directly into the space between the periodontal ligaments. The needle is placed in contact with the tooth and into the distal and mesial gingival sulcus. The crestal and root alveolar bones are where the needle is positioned for maximal penetration, supported by the fingers. For thirty seconds, the syringe handle is gradually compressed. For this technique to be effective, backpressure must be developed, and soft tissue blanching would indicate that the treatment is working. [26]

Intrapulpal:

A significant disadvantage of intrapulpal injection is the requirement to insert a needle into an extremely sensitive and inflammatory pulp. This means that the action might hurt. Analgesic issues might have arisen before the pulp was exposed, which is another requirement for administering the injection. Under severe backpressure, the injection must be administered. Anatomical limitations, such as dense bone or accessory innervation (mylohyoid nerve branch), may be observed in a very small number of cases and subsequently managed. Remarkably, bupivacaine was discovered to be more effective than lidocaine at blocking TTXr channels, suggesting that it might be the preferred anesthetic for treating "hot tooth." [26]

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