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Review Article

THE HOT TOOTH: BEATING THE ANESTHETIC BARRIER IN ENDODONTIC EMERGENCIES

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ABSTRACT

The "hot tooth," a tooth with symptomatic irreversible pulpitis that does not respond to traditional local anesthetic techniques, is one of the most challenging clinical scenarios in endodontics. This phenomenon is based on inflammation-induced peripheral sensitization, altered sodium channel expression, and tissue acidosis, resulting in reduced efficacy of the standard inferior alveolar nerve block (IANB) in mandibular teeth. This manuscript covers the current evidence on ten themes: definition and diagnostic criteria for the hot tooth; etiology and neurobiological basis; clinical features and differential diagnosis; challenges with local anesthesia; supplemental anesthetic strategies; pharmacological premedication; endodontic treatment approaches, including pulpotomy versus root canal therapy; postoperative pain management; psychological considerations; and evidence-based clinical recommendations. Recent advances in vital pulp therapy, bioceramic materials, and computer-controlled anesthesia delivery systems have expanded the clinician's armamentarium. A systematic stepwise protocol with combined use of these modalities provides the best chance for a patient-centered pain-free endodontic treatment.

Keywords: Hot Tooth, Irreversible Pulpitis, Inferior Alveolar Nerve Block, Supplemental Anaesthesia.

INTRODUCTION

The most frequent cause of urgent dental care is pulpal pain, and the diagnosis is always symptomatic irreversible pulpitis. There is a subset of patients in this group that every endodontist learns about by experience long before they can fully explain it, known as the "hot tooth." This condition is frequently called "hot tooth," a tooth with irreversible pulpitis and spontaneous moderate-to-severe pain that doesn't respond to conventional local anesthesia. An inferior alveolar nerve block or infiltration fails or wears off mid-procedure, leaving the patient and clinician trapped in a cycle of pain. Failure rates for inferior alveolar nerve blocks in teeth with irreversible pulpitis reach as high as 80 percent, compared to 15 percent in healthy pulp tissue (Nusstein, *et al.*1998). The hot tooth's behaviour can be understood by looking beyond the syringe and into the biology of the pulp. The dental pulp is a highly vascularized, densely innervated connective tissue confined within a rigid, nonexpansile chamber of dentin. This confinement is the root of the problem. When caries, trauma, or a failed restoration allow bacterial by-products and inflammatory mediators to reach the pulp, the tissue reacts like any other connective tissue: capillaries dilate, fluid leaks into the interstitium, and local pressure increases. The pulp's inability to swell outward causes the thin-walled venules to collapse, slowing drainage and intensifying congestion, creating a self-reinforcing loop of ischemia and inflammation. The superficial A-delta fibers, responsible for the sharp, well-localized pain of reversible pulpitis, are supplemented by the slower, unmyelinated C-fibers that convey the dull, throbbing, often unrelenting pain characteristic of acute irreversible pulpitis as the process intensifies. The recruitment of activity in C-fibers and a cascade of locally released prostaglandins,

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bradykinin, and substance P contribute to this phenomenon (Kim, *et al.* 1972).

It is a very inflammatory environment that is largely responsible for compromising the anesthetic itself. Various theories about why this occurs have existed over the years, though none alone are wholly adequate to explain the phenomena but rather contribute to the explanation: a relatively slight decrease in the pH level of tissue that results in a higher ratio of charged anesthetic molecules ,changes in the resting potential and excitability threshold in the nerve fiber in question, beyond simply the pulpal nerve; central sensitization, in which a constant stream of noxious stimuli causes second order trigeminal nucleus neurons to become sensitized to even the smallest amount of peripheral stimuli; and, possibly the most convincing explanation on the molecular biology level, the presence of a higher density of sodium channels in the nociceptive fibers which are to tetrodotoxin (which is how lidocaine acts) as well as being enhanced by inflammation products such as prostaglandin E2. Fear and negative experiences with dental injections further lower pain thresholds, confirming the patient's belief that the anesthetic never works. The hot tooth is not a failure of technique but a clinical expression of a coherent pathophysiology: an inflamed pulp generating rising intrapulpal pressure, a sensitized peripheral and central nervous system, and an anesthetic working against altered pH, channel kinetics, and neuronal thresholds (Renton, *et al.* 2011).

The thing that distinguishes a systematic approach to management from reactive troubleshooting at the chair is the recognition of this chain of pathophysiology. This manuscript traces the path from the inflamed pulp through the pain circuitry to differential diagnosis and premedication strategies that can tip the scales in favour of the practitioner. The process develops from the diseased pulp, continuing through pain perception pathways, differential diagnoses, and pre-medication strategies, anesthesia techniques, and adjuncts documented in the literature to help the practitioner regain control shows in (Hargreaves & Keiser, 2002) (Figure 1 & 2).

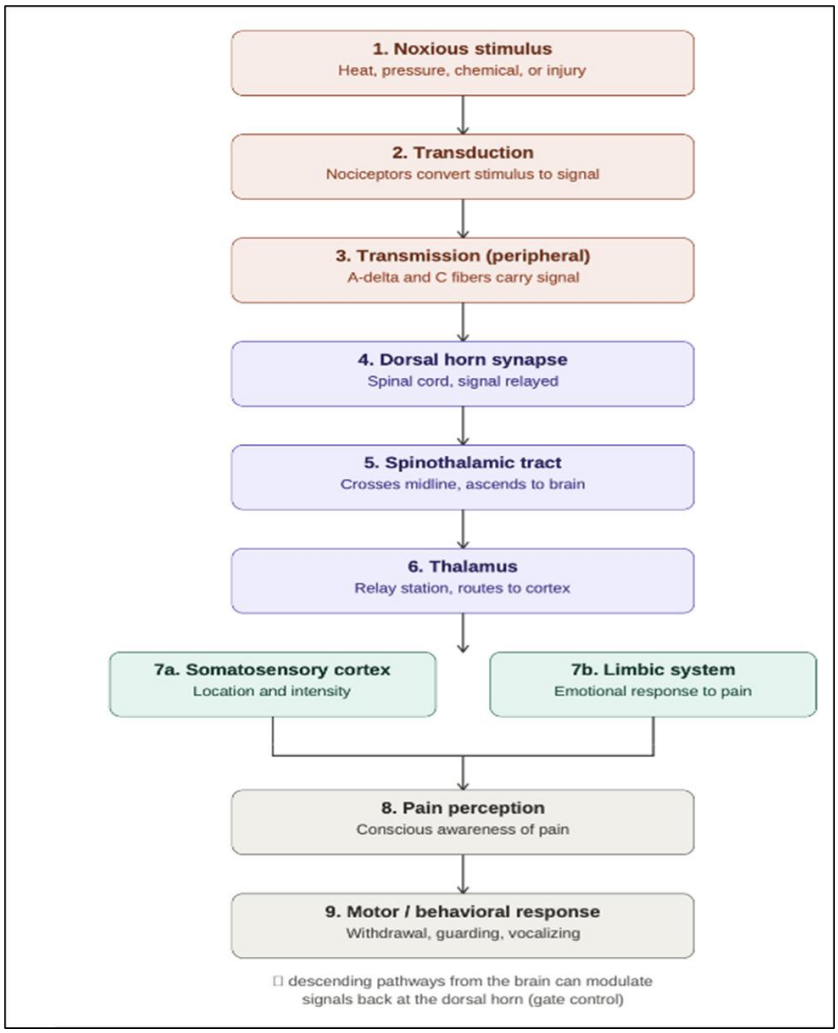

Figure 1. The Pain Pathway from noxious stimulus to conscious perception and response

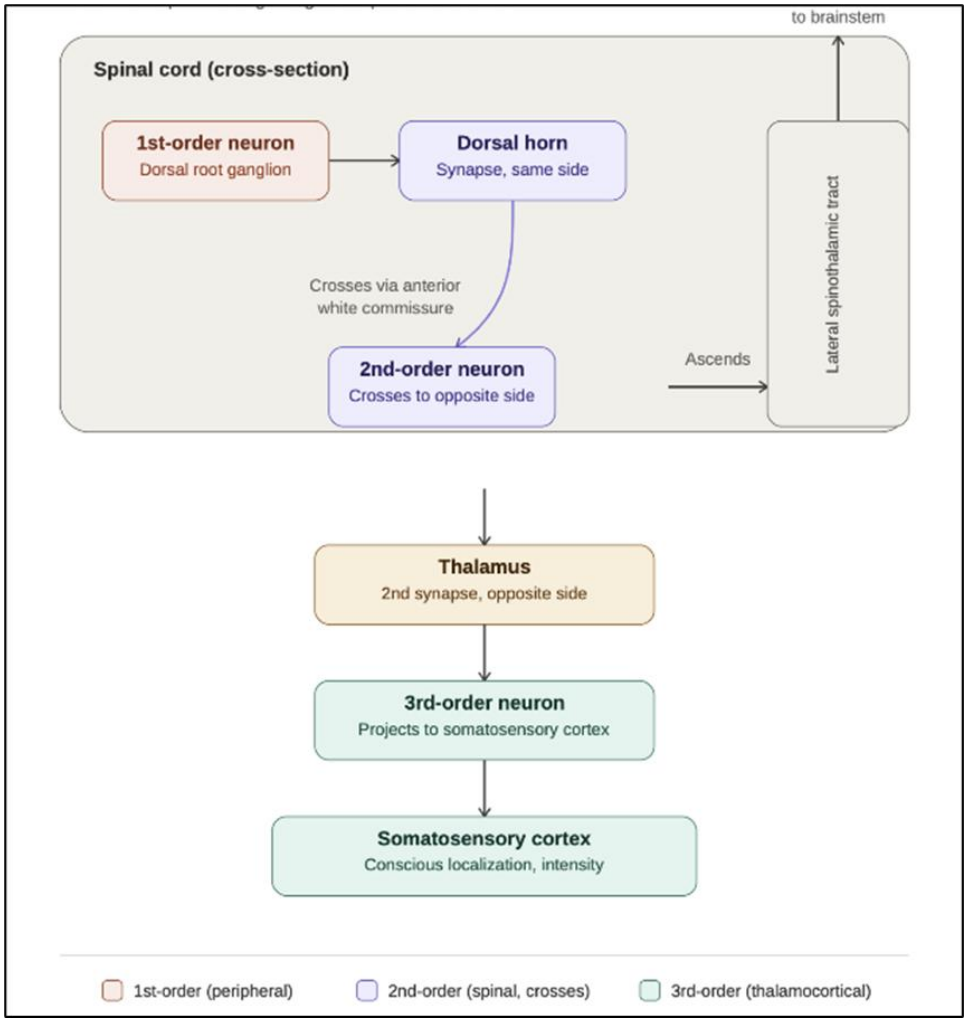

Figure 2 Spinothalamic Tract Pathway -pain temperature signalling from spinal cord to cortex.

HOT TOOTH: DIAGNOSIS & MANAGEMENT

What is Hot Tooth?

The colloquial term "hot tooth" has been a part of the endodontic clinical vocabulary for decades, but its definition has varied considerably as research has advanced. Currently, the hot tooth is a tooth with symptomatic irreversible pulpitis (SIP), in which the pulp is vital but irreversibly inflamed, producing moderate to severe, often spontaneous pain and, importantly, not responding to profound local anesthesia by conventional means. The American Association of Endodontists (AAE) says that irreversible pulpitis is a clinical diagnosis based on subjective and objective findings indicating that the vital, inflamed pulp is unable to heal. The hallmark of a hot tooth is not the severity of pain but the challenge it presents to the anesthesiologist, that is, a tooth that cannot be made pain-free by a standard inferior alveolar nerve block (IANB) administration alone (Gerald, 2010). The clinical problem is most noticeable in mandibular posterior teeth, especially the first and second molars, although maxillary teeth are also included in the term. Studies have shown repeatedly the failure rate of IANB in patients with symptomatic irreversible pulpitis is from 44% to 81% versus approximately 15% in teeth with normal pulp status. This dramatic difference highlights the unique neurobiological milieu engendered by pulpal inflammation. (Gerald, 2010).

Clinical Diagnosis Framework

Accurate diagnosis is the cornerstone of hot tooth management. The diagnosis is established by correlating the subjective patient history with objective clinical and radiographic tests and integrating them into a definitive pulpal and periapical diagnosis. (Duncan, 2023).

Subjective Evaluation

Hot tooth patient often has a history of long-lasting lingering pain to thermal stimuli, especially hot beverages, for 30 seconds or longer after the stimulus has been removed. The pain may be spontaneous and throbbing in nature and aggravated by postural changes such as lying supine or bending forwards. Night pain waking a patient from sleep is a strong clinical sign of advanced pulpal inflammation. Localization can be difficult, and referred pain to neighboring teeth, the ear, or the temporal region

can necessitate a systematic evaluation of each tooth suspected individually. Pain Characteristics: Symptomatic irreversible pulpitis is usually poorly localized in its early stages because of the sparse distribution of A-delta fibers in the pulp. As the disease progresses, C-fiber nociceptors, which mediate slow, burning, diffuse pain, become the dominant signalers. This neurophysiological change explains the classic dull, throbbing characteristic described by patients with advanced pulpitis and the difficulty in localizing the offending tooth accurately in the absence of objective testing. (Rodd & Renton, 2000).

Objective Clinical Examination

Cold testing (ethyl chloride, Endo-Ice): A response for more than 10 seconds after removal of the stimulus is diagnostic of symptomatic irreversible pulpitis. Heat testing: Interestingly, the classical hallmark of the hot tooth is relief of pain on the application of cold and exacerbation with heat, reflecting the heightened sensitization of pulpal nociceptors. Electric pulp testing (EPT): A measure of pulp sensory nerve fiber integrity. Low threshold values for hyperresponsiveness supports irreversible pulpitis. Percussion: Tenderness to percussion indicates periapical involvement and may suggest a transition to apical periodontitis. Palpation testing: Tenderness on palpation over the apex of the mucosa indicates periradicular inflammation. Periodontal probing: Excludes periodontal-endodontic lesions as a contributing aetiology (Nusstein, *et al.* 1998 & Gearld, 2010).

Evaluation radiographique

The periapical radiographs still remain the baseline imaging modality. Findings may range from a normal periapical appearance (in early irreversible pulpitis) to widening of the periodontal ligament space or early periapical rarefaction in more advanced cases. CBCT indications include inconclusive conventional radiographs, especially in the case of multirooted teeth, complex root morphology, or suspicion of additional canals. It is important to remember that radiographic findings alone are not sufficient for the diagnosis of irreversible pulpitis, and the clinical examination is paramount (Duncan, *et al.* 2023).

Differential Diagnosis

The presentation of the hot tooth can be mimicked by a number of conditions that need to be differentiated: Reversible pulpitis: Pain is short-lived and subsides within seconds of removal of stimulus. No spontaneous attacks. Treatment involves the removal of caries and restoration. Cracked tooth syndrome: History of sharp pain with biting or release of occlusal pressure on certain cusps. May coexist with irreversible pulpitis requiring endodontic treatment. Dentinal hypersensitivity: local, sharp, short duration response to cold, air or tactual stimuli; no spontaneity. Symptomatic apical periodontitis: Constant, well-localized pain on biting, percussion-positive; pulp may be vital or necrotic Atypical odontalgia/persistent dentoalveolar pain: Chronic pain without a discernable odontogenic cause; unresponsive to endodontic therapy. Temporomandibular joint (TMJ) disorders and myofascial pain: May refer pain to posterior teeth mimicking pulpal pathology (Balivada & Chandra, 2023).

The Diagnostic Dilemma of Teeth with Multiple Symptoms

If a patient presents with pain from multiple quadrants or multiple teeth with positive findings, individual teeth must be isolated with topical anesthetic or selective nerve blocks before definitive treatment can be rendered. The clinician should only treat the tooth with the most compelling combination of history, clinical, and radiographic evidence at any one appointment. Residual symptoms should be reassessed at follow-up (Nusstein & Reader, 2010).

MANAGING THE HOT TOOTH IN ENDODONTICS

Overview of Management Paradigm

Successful management of the hot tooth involves a structured, stepwise approach that involves three interrelated objectives: (1) achieving profound pulpal anesthesia, (2) performing definitive endodontic therapy, and (3) providing adequate postoperative pain control. If any of these pillars are not addressed, patient comfort, treatment quality, and therapeutic outcome are compromised. The management protocol should be modified according to the severity of the symptoms, the specific tooth involved, the medical history, the psychological state of the patient, and the available resources (Cohen & Hargreaves, 2021).

Preoperative Assessment and Counselling of the Patient

It is both an ethical imperative and a clinical benefit to have a thorough discussion preoperatively. Patients in pain at the dental office are frequently anxious, and clear communication about the possibility of anesthetic challenges greatly reduces fear and increases cooperation. The clinician should inform the patient that complete pain control may require multiple injections and the use of supplemental techniques. Setting realistic expectations helps to reduce the patient's distress when the initial anaesthesia is inadequate and improves their willingness to accept additional interventions (Cohen & Hargreaves, 2021).

Anaesthesia Depth Assessment Before Access Preparation

Prior to starting access cavity preparation, it is essential to verify the adequacy of pulpal anesthesia with objective signs. Numbness of the lip and tongue (for mandibular blocks) confirms that the nerve block was deposited but does not ensure pulpal anesthesia. The clinician should, if tolerated, perform a tactile stimulation of the marginal gingiva with a sharp explorer and an abrupt cold application near the gingival margin. Absence of response indicates adequate pulpal anesthesia, although intrapulpal

sensitivity may develop on entry (Cohen & Hargreaves, 2021).

Intraoperative Pain Management Plan

A pragmatic intraoperative algorithm is as follows: The primary IANB is delivered with an adequate volume (1.5-1.8 mL) and confirmed with lip numbness. If the patient reports pain with the entry of the access, the clinician stops and provides supplemental anesthesia instead of proceeding under pain. Section 5 covers the hierarchy of supplemental techniques in detail, which is buccal infiltration with articaine, intraosseous (IO) injection, intraligamentary (PDL) injection, and last, intrapulpal injection as a last resort. The highest success rates (close to 90%) have been reported in hot tooth cases with the combination of a primary IANB and intraosseous injection (Fowler *et al.* 2016 & Elnaghy *et al.* 2023).

Preparation of access cavity and biomechanical treatment

Once adequate anesthesia is obtained, the access cavity is prepared according to standard endodontic protocols. Immediate pulpotomy (removal of coronal pulp tissue) prior to instrumentation of the radicular portion of severely inflamed pulps results in rapid pain relief and facilitates further treatment in a more comfortable manner. Heavy irrigation with 2.5 to 5.25 percent sodium hypochlorite has a double action, that of an antiseptic and that of dissolving inflamed organic debris. The electronic apex locator with radiographic confirmation of the findings allows you to determine the working length accurately. The continuous, smooth and proficient instrumentation reduces the iatrogenic pressure changes within the canal system, which could aggravate the postoperative discomfort (Cohen & Hargreaves, 2021).

One-Visit vs. Several-Visit Root Canal Treatment

Contemporary evidence supports single-visit root canal treatment (SV-RCT) for most cases of symptomatic irreversible pulpitis without periapical pathology, as it reduces the number of appointments, eliminates interappointment pain episodes, and decreases the risk of coronal microleakage through the temporary restoration (Cohen & Hargreaves, 2021). Multi-visit approaches remain appropriate when treatment time is limited, access is complex, or when the clinician prefers to defer obturation to confirm symptom resolution. The use of calcium hydroxide as an interim dressing has antimicrobial benefits but does not predictably reduce postoperative pain compared with single-visit approaches in cases of irreversible pulpitis (Fowler, *et al.* 2016)

HOT TOOTH: PROBLEMS WITH LOCAL ANAESTHESIA AND PAIN CONTROL

The Context of the Anesthesia Problem

The problem with anesthetizing a hot tooth is that so many neurobiological, pharmacological, anatomical, and psychological factors come together. It is important to understand each dimension so that rational supplemental strategies can be adopted rather than repeating failed techniques with the same agents and approaches (Hargreaves & Keiser, 2002).

Neurobiological Mechanism of Anesthesia Failure

Sodium Channels Resistant to Tetrodotoxin

The main molecular basis for the failure of hot tooth anesthesia is the upregulation of tetrodotoxin-resistant (TTX-R) voltage-gated sodium channels (Nav1.8 and Nav1.9) in inflamed pulpal and periapical nociceptors. In normal healthy pulp tissue the majority of sodium channels are of the tetrodotoxin-sensitive (TTX-S) type and are efficiently blocked by local anesthetics like lidocaine. However, in the inflamed pulp of a hot tooth, the expression of TTX-R channels increases about twofold, and these channels are about four times more resistant to conventional local anesthetic agents than are their TTX-S counterparts. The presence of TTX-R channels decreases the activation threshold of the nociceptors, increases the influx of sodium ions across the nerve membrane, and maintains the generation of action potentials despite the presence of standard anaesthetic concentrations (Roy & Narahashi, 2002). This peripheral sensitisation explains why an IANB that results in complete lip numbness (indicating blockade of the inferior alveolar nerve trunk) can still fail to abolish intrapulpal pain.

Decreased Ionisation and Tissue Acidosis

The inflamed pulpal environment is defined by a significant reduction in tissue pH due to the accumulation of acidic metabolic products of inflammatory cells, bacteria, and necrotic tissue. Local anesthetic agents (generally weak bases with pKa values close to physiologic pH) must be in the uncharged, lipid-soluble base form to cross the nerve membrane and reach the sodium channel binding site. In an acidic environment, the equilibrium shifts to the charged, ionized form (BH^+), which does not effectively penetrate the lipophilic nerve membrane. This pH-dependent pharmacokinetic impairment significantly decreases the fraction of local anesthetic that is bioavailable and therefore reduces its clinical efficacy, regardless of the accuracy of the injection technique (Parirokh, & Abbott, 2014).

Inflammatory mediators and central sensitization

Peripheral sensitization is the process of sensitizing peripheral nociceptors and decreasing their excitation thresholds by inflammatory mediators such as prostaglandins (especially PGE2), bradykinin, histamine, substance P, calcitonin gene-related peptide (CGRP), and nerve growth factor (NGF). With persistent and intense nociceptive input, as in symptomatic irreversible pulpitis, central sensitization of dorsal horn neurons (or their trigeminal equivalent) may occur, further amplifying the pain signals beyond that which can be controlled with peripheral anesthetic blockade. Furthermore, PGE_2 directly

activates TTX-R sodium channels through cAMP-dependent phosphorylation, which establishes a bidirectional loop facilitating inflammation and anaesthetic resistance (Roy & Narahashi, 2002).

Anatomical aspects

Several unique anatomic factors affect the mandibular IANB. The course of the inferior alveolar nerve may be atypical in some persons, and the geometry of the pterygomandibular space is highly variable. Branches of the mylohyoid nerve, long buccal nerve, and contralateral inferior alveolar nerve can provide persistent sensory innervation to mandibular teeth despite technically accurate IANB. Specifically, the mylohyoid nerve has been implicated in innervating the mesial roots of mandibular first molars through an accessory pathway that accounts for the persistent sensitivity often observed even in the presence of documented IANB success (Parirokh & Abbott, 2014).

Pain amplification by psychological means

Dental anxiety is a strong modulator of pain perception. Dental phobic patients have lower pain thresholds and show increased central sensitization (i.e., the same nociceptive input leads to a more intense pain experience). The fear of pain associated with dental procedures activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to increased levels of cortisol and catecholamines (Parirokh & Abbott, 2014). These hormonal changes result in heightened sympathetic nervous system activity and reduced effectiveness of local anesthetics. Addressing psychological barriers through empathic communication, relaxation techniques, and, if indicated, nitrous oxide sedation or oral anxiolytics is an important aspect of hot tooth management.

Clinical Decision Points and Indications of Anaesthesia Failure

The clinician should recognize signs of inadequate pulpal anesthesia during treatment: Patient complains of pain on first access cavity preparation with bur in low speed. Sharp pain when entering pulp chamber. Flinching or hand signals during instrumentation of coronal pulp. Residual sensitivity on cold testing post IANB confirmation. At each of these decision points the clinician must pause, reassess, and apply appropriate supplemental anesthesia. Not only is it unethical to go ahead under pain, but it is also counterproductive, as it increases anxiety and muscle tension and decreases cooperation of the patient in following steps of treatment (Nusstein & Reader, 2010)

HOT TOOTH: CLINICAL PRESENTATION, ANAESTHESIA AND MANAGEMENT

Clinical manifestations

The clinical picture of a hot tooth is recognizable but must be confirmed systematically: Spontaneous, unprovoked pain of moderate to severe intensity (VAS > 54/170 on a Heft-Parker Visual Analogue Scale, which is commonly used in endodontic research) Prolonged thermal sensitivity—pain lasting more than 10-30 seconds after the cold stimulus has been removed. Pain aggravated by heat: paradoxical relief by cold application (a classical but not universal feature). Pain that may wake you up or get worse when lying flat. Possible radiation to same ear, temple or neck. Electric pulp testing revealed positive response at a lower threshold than contralateral control teeth (Balivada & Chandra, 2023)

Clinical Characteristics to Distinguish Hot Tooth from Necrotic Pulp

It is important to be able to differentiate between a vitally inflamed pulp (hot tooth) and a necrotic pulp with periapical involvement because the treatment protocols are quite different (Cohen & Hargreaves, 2021). The main distinguishing features are as follows: Vital (hot) pulp: Positive to cold and electric pulp testing. May have positive percussion in advanced cases with periapical involvement. No sinus tract. Necrotic pulp: No response to cold and EPT. A sinus tract may be present. Radiographic periapical rarefaction is usually present. Positive percussion due to periapical inflammation. Calcified pulp: May cause nonresponsive or false negative EPT results in the absence of symptoms CBCT helps to confirm the calcification.

Primary Anaesthesia: Block of the Inferior Alveolar Nerve

The IANB is the gold standard for anesthesia of mandibular teeth. The classic Halstead technique deposits LA at the mandibular foramen, targeting the inferior alveolar and lingual nerves (Nusstein, *et al.* 1998) The key technical factors for maximizing success are Correct landmark identification: The injection should be parallel to the occlusal plane of the mandibular molars and within 1 cm of the level of mandibular molars in adults. Aspiration prior to injection: essential to prevent intravascular injection. Slow deposition rate: 1.5-1.8 mL deposited over at least 60 seconds to reduce patient discomfort and allow diffusion. Waiting time: Allow time for complete diffusion of anesthetic. Minimum of 10-15 minutes before starting treatment. Agent of choice: 2% lidocaine with epinephrine 1:80,000 or 1:100,000 is the gold standard. Articaine 4% with epinephrine may be more effective for supplemental buccal infiltration but has not been shown to improve the success of primary IANB (Ritchie & Greene, 1985)

Handling of IANB Failure

Subjective numbness of the lip/tongue is mandatory to confirm successful IANB but is insufficient to confirm pulpal anesthesia in cases of a hot tooth. If the patient has a confirmed lip numbness but still complains of pain, the clinician should not repeat the same IANB with the same agent. Rather, the stepwise supplemental protocol described in Section 5 should be initiated. Repeated failed IANB without modification only increases patient anxiety and delays effective treatment (Nusstein & Reader, 2010)

Hot Tooth and Maxillary Teeth

The hot tooth phenomenon is mostly discussed in relation to mandibular teeth, but maxillary teeth with symptomatic irreversible pulpitis can also be a challenge for anesthesia. The maxillary pulps are also relevant in the local tissue acidosis and TTX-R channel upregulation. Standard buccal infiltration anesthesia for maxillary teeth usually has a higher success rate than IANB because the maxillary alveolar bone is more porous, allowing better anesthetic diffusion. However, palatal root anaesthesia may still be inadequate. Additional palatinal infiltration or intraosseous injection may be needed in case of a persistent hot tooth presentation of maxillary molars. Studies comparing 4% articaine and 2% lidocaine for buccal infiltration of maxillary teeth with irreversible pulpitis suggest that articaine may provide marginally superior pulpal anesthesia due to its greater lipid solubility and bone-penetrating ability (Kolay & Kirici, 2025).

Hot Tooth: A Full Review of Supplemental Anaesthesia

Additional Anaesthesia: Justification and Timing

Supplemental anesthesia is the key to overcoming local anesthetic failure in the hot tooth. Supplemental techniques need to be considered an expected and preemptively designed part of the anesthetic protocol in any tooth diagnosed with symptomatic irreversible pulpitis rather than a rescue measure used as a last resort. The order of additional modalities is guided by data on efficacy, availability, ease of administration, patient comfort, and risk-benefit considerations (Nusstein & Reader, 2010).

Articaine infiltration (buccal)

Following failed IANB, articaine (4% with 1:100,000 epinephrine) has become the agent of choice for supplemental buccal infiltration of mandibular molars. The chemical structure of articaine is unique in that it has a thiophene ring instead of a benzene ring, which makes it more lipid-soluble and able to penetrate membranes. It is also subject to ester hydrolysis in the blood, resulting in a shorter systemic half-life (~20 minutes) and a better safety profile than amide anesthetics. In a prospective randomized clinical trial in 2022, supplemental buccal infiltration of 1.7 mL of 4% articaine after failed IANB in mandibular molars with irreversible pulpitis was successful in 58-64% of cases, representing a clinically meaningful improvement over repeat IANB alone. The injection is given buccally at the level of the apex of the molar roots with a 30-gauge short needle and usually takes 3-5 minutes to take effect (Matthews, *et al.* 2009).

Intraosseous Anaesthesia

Intraosseous (IO) anesthesia is the administration of local anesthetic directly into the cancellous bone adjacent to the target tooth. It allows for rapid (onset within 30-60 seconds) and reliable pulpal anesthesia by bypassing the limitations of the inferior alveolar nerve block and the pH barrier of inflamed soft tissue. The Stabident system and the X-Tip system are the most commonly used delivery systems. A retrospective study in 2024 comparing articaine buccal infiltration and lidocaine intraosseous anesthesia in mandibular molars reported that 100% of patients in the IO group did not require any additional anesthesia as compared to 70.8% in the buccal infiltration group ($p<0.001$). The IO technique is composed of perforation of the cortical plate with a perforator or slow-speed bur followed by insertion of the injection needle into the cancellous space. The main adverse effect of IO anesthesia is transient tachycardia secondary to absorption of epinephrine. The QRS complex is usually not affected; however, those patients with cardiac compromise may need epinephrine-free solutions or changed concentrations (Nakamura, *et al.* 2024).

Computerized delivery systems (e.g., QuickSleeper5, Wand/CompuDent) combine pressure-controlled injection with IO or PDL techniques and provide programmable delivery rates that may reduce discomfort and improve reproducibility of technique. These devices are particularly useful in anxious patients or those with a fear of needles (Parirokh & Abbott, 2014).

Intra-Ligamentous (Periodontal Ligament) Injection

The periodontal ligament (PDL) injection deposits local anesthetic in the PDL space next to the root, where it spreads into the alveolar bone and reaches the apical neurovascular bundle. As an adjunctive technique, success rates of 55-68% have been reported in mandibular molars with irreversible pulpitis. The injection is given at 30 degrees to the long axis of the tooth, mesial and distal. A high-pressure syringe (e.g., Ligmaject) is used for the injection. The rapid onset (within 30 seconds) and short duration (15-20 minutes) may allow enough time for access cavity preparation and initial pulpectomy to be completed. The technique is only effective when there is significant tissue resistance. No resistance means the placement is incorrect. Mild soreness after surgery is common and usually resolves in 24 to 48 hours (Parirokh & Abbott, 2014).

Intra-pulpal Anaesthesia

Intrapulpal anesthesia is considered the ultimate adjunctive technique, used when all other measures have failed to provide adequate pulpal anesthesia and the pulp chamber has been partially accessed. The mechanism is largely pressure-dependent: direct injection of local anesthetic (with or without vasoconstrictor) under back-pressure into the pulp chamber achieves immediate and absolute pulpal anesthesia, so that coronal pulpotomy and initial canal instrumentation can usually be completed. The injection is painful and should be warned to the patient. The numbness after the injection is intense but relatively short, about 5-10 minutes, and should be used efficiently. Intrapulpal injection, if well done, should be regarded as a permanent rather than a temporary measure (Reader, *et al.* 1998).

Comparative Analysis and Algorithm for Clinical Decision

Clinical Pearl: Recommended sequential supplemental protocol for a hot mandibular molar: (1) Primary IANB with 1.8 mL 2% lidocaine + confirm lip numbness Wait 10-15 min. (2) If inadequate: supplemental buccal infiltration with 1.7 mL 4% articaine (3) If still not enough: intraosseous injection (preferred) or PDL injection. (4) As a last resort, intrapulpal injection under back pressure after access to the pulp has been obtained (Duncan, *et al.* 2023).

HOT TOOTH: MODERN CONCEPTS IN ENDODONTIC PAIN MANAGEMENT

Pre-surgical pharmacological strategies

There is increasing evidence in favor of the use of pharmacological premedication 30-120 minutes prior to endodontic treatment to improve anesthetic success and reduce intra- and postoperative pain in patients with symptomatic irreversible pulpitis (Duncan, *et al.* 2023).

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

NSAIDs work by inhibiting cyclooxygenase (COX) enzymes, decreasing prostaglandin E2 (PGE2) synthesis, and reducing PGE2-induced activation of TTX-R sodium channels via cAMP-dependent pathways. NSAIDs reduce peripheral sensitization and partially restore tissue pH by reducing the inflammatory milieu before treatment, improving the efficacy of local anesthetics. Preoperative NSAIDs, e.g., ibuprofen (400-800 mg) 30-60 minutes preoperatively, have the strongest evidence base of improving the success of IANB in patients with irreversible pulpitis. Ketorolac, diclofenac potassium, naproxen sodium, etoricoxib, and meloxicam have been studied, and results are variable. A 2024 systematic review found that premedication with anti-inflammatory agents (both NSAIDs and corticosteroids) significantly improves the efficacy of IANB in symptomatic irreversible pulpitis (Ianiro, et.al). Corticosteroids enhance anaesthetic efficacy by inhibiting the cyclooxygenase and lipoxygenase cascade and the production of prostaglandins and leukotrienes. Acetaminophen (paracetamol) is an alternative for patient's intolerant to NSAID, but evidence is less compelling for improving IANB success than for ibuprofen (Elnaghy, *et al.* 2023).

Corticosteroids

Dexamethasone, a potent synthetic glucocorticoid, has been well studied as a premedication agent in tooth management. Dexamethasone is administered orally (0.5-8 mg) 60-120 minutes before IANB and blocks inflammatory prostaglandin and leukotriene synthesis, thereby decreasing sensitization of pulpal nociceptors and tissue edema. A systematic review with meta-analysis and trial sequential analysis of randomized clinical trials published in 2024 showed that oral corticosteroid premedication improves the anesthetic efficacy of the IANB compared to placebo. Several routes of administration have been used, including oral and intraligamentary administration with local anaesthetic solution. Reported success rates with dexamethasone premedication (54-60%) are much greater than placebo (21-32%). Other improvements were noted with prednisolone and ketorolac. Short-term use of a single preoperative dose of corticosteroids has few adverse effects in otherwise healthy patients (Elnaghy, *et al.* 2023).

Combination Premedication Strategies

Theoretically, the combination of an NSAID with a corticosteroid (eg, ibuprofen 400 mg + dexamethasone 0.5 mg) targets both cyclooxygenase (COX)-dependent and -independent inflammatory pathways with additive antiinflammatory effects. A prospective randomized controlled trial on combined oral premedication showed that the combination of dexamethasone and ibuprofen improved IANB success rates in mandibular molars with symptomatic irreversible pulpitis compared with each individual agent. However, the risk of additive gastrointestinal side effects should be considered in patients with peptic ulcer disease or renal impairment, and a gastroprotective agent may be indicated (Elnaghy, *et al.* 2023).

Additives to Local Anaesthetics

Clonidine added to lidocaine: An alpha-2 adrenergic agonist that prolongs the duration and improves the quality of IANB. Few clinical studies in hot tooth situations, but promising mechanistic rationale. Sodium bicarbonate buffering (alkalinization) Increasing the pH of the local anaesthetic cartridge before administration increases the percentage in the uncharged base form. Commercial alkalinizing devices are available and early data suggests a modest improvement in onset and efficacy. LOCAL ANAESTHETIC WARMING Warming the anaesthetic cartridge to body temperature (37°C) minimises injection discomfort and may slightly enhance diffusion kinetics. Nitrous oxide (N2O) sedation: Reduces anxiety, increases pain threshold, and increases the effectiveness of local anesthesia by increasing the pain tolerance threshold (Hargreaves, et al. 2002).

Control of post-operative pain

Pain after endodontic treatment of a hot tooth is a normal and expected phenomenon and is a reflection of post-instrumentation periapical inflammation. Evidence-based prescription of postoperative analgesics: The single most effective analgesic for post-endodontic pain is still ibuprofen 400-600 mg every 6-8 hours for 3-5 days. The maximum daily dose for a healthy adult is 3200 mg. Combination of ibuprofen + acetaminophen: Superior pain control than either agent alone due to complementary mechanisms (peripheral COX inhibition + central analgesic effect). Opioids are not recommended as first-line agents for post-endodontic pain because of limited efficacy advantage over NSAIDs, risk of dependence and adverse effect profile. Intracanal corticosteroid (e.g. Ledermix) in single dose: Placed in the canal at the end of the

appointment for local anti-inflammatory effect and to reduce the post-instrumentation flare-ups. Occlusal reduction: Selective reduction of the occlusal contact of the treated tooth reliably reduces postoperative pain. It is a cost-free, underutilised intervention (Cohen & Hargreaves, 2021).

HOT TOOTH: AETIOLOGY, DIAGNOSIS AND CLINICAL MANAGEMENT

Reason for Irreversible Pulpitis

The disease that causes the hot tooth, irreversible pulpitis, is caused by a prolonged injury to the pulp that is beyond the ability of the dental pulp to repair itself. The most frequent etiologic factors are (Cohen & Hargreaves, 2021): Deep dentinal caries: The predominant aetiology. Bacterial toxins, acids and by-products diffuse through the dentinal tubules and trigger an inflammatory cascade in the pulp. The deeper and faster the progression of a carious lesion, the greater the risk of irreversible injury to the pulp. Traumatic dental injuries: Mechanical disruption, vascular compromise, and microbial contamination can cause irreversible pulpitis from luxation injuries, crown fractures with pulp exposure, and avulsion/replantation. Iatrogenic injury Excessive heat generation during cavity preparation without adequate water cooling; aggressive pin placement; galvanic shock from dissimilar metals; chemical injury from cavity liners or bonding agents can induce pulpal inflammation. Marginal microleakage: Bacteria can gradually penetrate through defective or ageing restorations, challenging the pulpal defence mechanisms until they are defeated. Periodontal disease and retrograde pulpitis. Advanced periodontal disease with exposure of lateral canals or the apical foramen can result in bacterial colonisation of the pulp from the periodontium. Cracked tooth syndrome: Cracks that extend into the pulp open dentinal tubules, allow bacterial entry and cause cyclic hydraulic pressure trauma with occlusal loading.

Histopathological Correlates

Histologically, the inflamed pulp in irreversible pulpitis shows a spectrum of changes from focal chronic inflammation (Cohen & Hargreaves), with lymphocyte and macrophage infiltration in the early stages to areas of frank liquefaction necrosis, abscess formation, and nociceptor sensitization in advanced disease. The transition from reversible to irreversible pulpitis is not a histological threshold, but rather a clinical determination that symptoms such as spontaneous pain and prolonged thermal sensitivity indicate the point of no return for pulp healing irrespective of the exact histological stage. This highlights the importance of clinical diagnosis over histological correlation in endodontic practice.

Molecular mediators of pulpal pain

Prostaglandins, especially PGE2, have direct actions on nociceptor ion channels like Nav1.8 and Nav1.9, decreasing their firing threshold through protein kinase A (PKA) and protein kinase C (PKC) phosphorylation cascades. Bradykinin activates the BK1 and BK2 receptors on nociceptors, leading to increased sodium channel conductance and release of substance P. Calcitonin gene-related peptide (CGRP) and substance P induce neurogenic vasodilation, which increases pulpal blood flow and edema in an environment previously pressure-constrained, further amplifying nociceptive signaling. Inflammatory cells release nerve growth factor (NGF), which induces phenotypic switching of A-delta fibers to C-fiber-like behavior, explaining the shift from sharp, localizable pain to dull, diffuse, spontaneous pain with the progression of irreversible pulpitis (Roy & Narahashi, 2002)

HOT TOOTH: TIPS FOR A SUCCESSFUL ROOT CANAL TREATMENT

Planning for Integrated Treatment

Successful treatment of the hot tooth is not limited to the intraoperative session. It starts with an accurate diagnosis preoperatively, involves meticulous execution intraoperatively, and extends to adequate pain management and timely restoration postoperatively. Each step is a contributor to the overall therapeutic outcome (Nusstein & Reader, 2010).

Vital Pulp Therapy: An Emerging Alternative

There has been a major shift in the treating teeth with irreversible pulpitis in upcoming years, with a more conservative approach being adopted due to advances in bioactive materials such as mineral trioxide aggregate (MTA) and hydraulic calcium silicate-based bioceramics. Full pulpotomy (complete removal of coronal pulp tissue while preserving healthy radicular pulp) has been established as a clinically validated alternative to root canal treatment in selected cases of permanent teeth with symptomatic irreversible pulpitis in mature teeth (Shoba, *et al.* 2023).

Taha, Abuzaid, and Khader's (2023) randomized controlled trial found that mature teeth with irreversible pulpitis and carious pulp exposure had comparable radiographicand clinical success rates at 12 months after full pulpotomy and root canal therapy, with the pulpotomy group reporting higher patient-reported quality of life scores. A 2024 randomized controlled experiment comparing root canal treatment and complete pulpotomy in mature molar teeth revealed similar outcomes. Clinical rates of success for pulpotomy in mature permanent teeth with incurable pulpitis ranged from 81.2 to 98.19%, according to a comprehensive review and meta-analysis published in 2024. Additionally, a 2024 cost-effectiveness analysis found that pulpotomy is a useful and cost-effective substitute for root canal therapy, particularly in circumstances where complete endodontic facilities might not be easily accessible. Important considerations in the selection of pulpotomy cases are: (1) no periapical pathology; (2) the ability to attain adequate hemostasis following pulp amputation, confirming radicular pulp

vitality; (3) the availability of a bioceramic capping material; and (4) the patient's consent for long-term radiographic follow-up. (Taha, *et al.* 2023)

Photobiomodulation as an Adjunct Therapy

Low-level laser therapy (LLLT/photobiomodulation) has been explored as an adjunct to local anaesthesia in hot tooth cases. Proposed mechanisms include modulation of mitochondrial cytochrome c oxidase activity, reduction in the synthesis of inflammatory mediators and direct inhibitory effects on C-fiber nociceptors. There have been studies investigating the use of photobiomodulation prior to administration of IANB with modest improvement in IANB success rates reported, however the evidence base is limited and methodologically heterogeneous (Rueda-Ibarra, *et al.* 2023). Photobiomodulation is not yet a first-line recommendation, but holds promise as a non-pharmacological adjunct for patients in whom pharmacological premedication is contraindicated.

Cryosurgery

Cryoanalgesia (topical application of ice or refrigerant-cooled devices to the buccal mucosa overlying the mandibular molar region) has been suggested to decrease firing frequency of nociceptors and increase IANB success through temperature-dependent depression of sodium channel conductance. Current clinical evidence, however, is not sufficient to establish a definitive benefit and should not be considered a substitute for proven supplemental techniques ((Rueda-Ibarra, *et al.* 2023).

Correct Irrigation and Disinfection

The inflamed state of a hot tooth provides an opportunity for bacteria released from a carious lesion into the periapical tissues during access preparation, which may increase postoperative pain. The application of complete irrigation protocols consisting of 2.5-5.25% NaOCl, 17% EDTA, and chlorhexidine (0.2% or 2%) in the correct order, in addition to passive ultrasonic irrigation (PUI) or sonic activation, guarantees the maximum effectiveness of disinfection and decreases the bacterial load that maintains periapical inflammation. Excess irrigation also clears inflammatory exudate from the pulp chamber, relieving pressure that could lead to postoperative pain (Cohen & Hargreaves, 2021).

HOT TOOTH: EVIDENCE-BASED STRATEGIES FOR ANAESTHESIA AND THERAPY

Evidence Status of Systematic Reviews and Meta-Analyses

Over the last decade, the evidence base for hot tooth management has grown significantly. Below is a summary of key findings at the systematic review level (Shoba, *et al.* 2023). IANB failure in irreversible pulpitis: Consistent across several studies; failure rates of 44-81% vs ~15% in normal pulp (Shoba, *et al.* 2023). Meta-analyses confirm significantly greater anesthetic success with articaine supplemental buccal infiltration than with repeat IANB alone. Articaine 4% was superior to lidocaine 2% for buccal infiltration (Kolay, *et al.* 2025). Intraosseous Anesthesia: Highest rates of supplemental success (up to 90-100%) when used after a failed IANB. Preferred supplemental technique recommended in mandibular molars (Nakamura, *et al.* 2024). NSAID premedication: Ibuprofen (400-800 mg) has been shown to significant increase in the rate of success of IANB. The combination of ibuprofen and dexamethasone has an additive benefit (Elnaghy, *et al.* 2023). Corticosteroid premedication. A 2026 systematic review with meta-analysis showed that oral corticosteroid premedication improves IANB anesthesia effectiveness in mandibular teeth with irreversible pulpitis compared to placebo (Suresh, *et al.* 2026). Pulpotomy versus root canal treatment: Growing RCT evidence shows similar results in carefully selected cases, with improved patient-reported quality of life and reduced cost for pulpotomy (Taha, *et al.* 2023)

Evidence Gaps and Areas for Future Research

Combined multiple pharmacological agents with supplemental techniques and optimal pre-treatment protocols. Long-term radiographic outcomes of pulpotomy vs. root canal treatment in mature mandibular molars: a follow-up > 3 years. Role of CBCT in accurate characterisation of pulpal and periapical status of hot tooth. Photobiomodulation, cryotherapy and ultrasound-enhanced drug delivery: utility in management of hot tooth. Patient-specific genetic polymorphisms in ion channel expression and NSAID metabolism and their impact on anaesthetic outcomes (Balivada & Chandra, 2023).

Clinical Practice Guideline

The European Society of Endodontology (ESE) S3-level Clinical Practice Guideline (2023) and the American Association of Endodontists (AAE) guidelines for the management of symptomatic pulpal disease. Key recommendations related to the hot tooth include confirmation of clinical diagnosis with systematic testing prior to the initiation of endodontic treatment; use of evidence-based supplemental anesthetic techniques when primary anesthesia is inadequate; consideration of vital pulp therapy where appropriate; and adequate postoperative analgesia with NSAIDs as first-line agents. (Gerald, *et al.* 2021).

HOT TOOTH IN ENDODONTIC PRACTICE: DIAGNOSIS, ANAESTHESIA AND MANAGEMENT - AN INTEGRATED PERSPECTIVE

Clinician Knowledge, Attitudes and Practices: Evidence from a Survey

A 2023 Knowledge, Attitude, and Practices (KAP) survey was conducted among general dentists and endodontists that found significant gaps in the clinical management of the hot tooth. The survey showed that infiltration and IANB were the most known techniques (96.6% and 98.5%), and

intraosseous injection was the least known technique, with only 6.8% of the participants. The study also found that adjunctive injections such as intraosseous and intraligamental techniques are underutilized despite the strong evidence on their efficacy, and the benefits of preanesthetic medication are not being fully realized by clinicians. The findings were corroborated by a 2023-2024 survey conducted by Gandhara University, Pakistan, which indicated that although most practitioners were cognizant of TTX-R channel resistance to local anesthetics and could diagnose irreversible pulpitis, their confidence in managing difficult anesthesia cases was poor, particularly among general dentists. Specialist training and continuing education in hot tooth protocols seem to be essential for closing this knowledge-to-practice gap (Khan, *et al.* 2023).

Patient-Centered and Psychological Issues

Patient-centered care in the setting of the hot tooth requires an awareness of the psychological toll of acute dental pain. Patients with a hot tooth are usually in great distress—not only from the physical pain but also from the anxiety, fear of treatment, sleep deprivation, and functional disability associated with advanced pulpitis. A short but caring visit that recognizes what the patient has gone through, establishes a relationship, describes the treatment plan in plain language, and establishes realistic expectations for pain control significantly improves the therapeutic alliance (Nusstein & Reader, 2010).

Dental anxiety should be formally assessed with validated tools (e.g. Modified Dental Anxiety Scale, MDAS) and managed proactively. In patients with high anxiety, pre-medication with oral benzodiazepines (triazolam 0.25 mg) 1 hour before the appointment, under adequate conditions of informed consent and monitoring, significantly reduces anxiety and improves the effectiveness of anesthesia. Inhalation sedation with nitrous oxide and oxygen (30-50% N2O) is a safe, reversible and widely used anxiolytic adjunct in endodontic practice (Parirokh & Abbott, 2014).

Proposed Integrated Clinical Protocol of the Hot Tooth

The following evidence-based stepwise protocol encompasses all the discussed elements involved in the management of a hot tooth in a mandibular molar (Duncan, *et al.* 2023). Step 1 - Accurate Diagnosis: Conduct systematic pulpal and periapical tests. Confirm diagnosis of irreversible symptomatic pulpitis. Take a periapical radiograph (CBCT if indicated). Differentiate from other diagnoses. Step 2 - Communication with Patient and Assessment of Anxiety: Discuss the hot tooth phenomenon, realistic anesthesia expectations, and the possibility of supplemental injections. Assess for level of anxiety and consider prescription of an anxiolytic. Step 3 — Premedication with drugs: Pre-medicate 45-60 minutes before the appointment with 400-600 mg ibuprofen. In severe cases consider adding dexamethasone 0.5-4 mg. NSAID alternative: prescribe acetaminophen in those with contraindications. Step 4 - Primary Anaesthesia: Perform typical IANB with 1.5-1.8 mL of 2% lidocaine with 1:100,000 epinephrine Check for numb lips and tongue. Leave for 10 to 15 minutes before proceeding. Step 5. Supplemental anaesthesia (as needed): Give additional buccal infiltration with 1.7 mL of 4% articaine (1:100,000 epi). If still not enough: intraosseous injection (preferred) or PDL injection. Do not give an intrapulpal injection until you have partial access to the pulp chamber. Step 6 — Root Canal Treatment: Access cavity preparation → pulpotomy of inflamed coronal tissue → determination of working length → biomechanical preparation → thorough irrigation → obturation (single visit if appropriate) OR interim dressing with calcium hydroxide if multi-visit. Step 7 — Aftercare: Reduction of the treated tooth occlusally. Prescribe ibuprofen 400 mg (alternating with acetaminophen 500 mg every 3 hours as appropriate). Interappointment dressing with intracanal corticosteroid. Postoperative instructions written clear 24-48 hr follow up.

The Future of Hot Tooth Management

Emerging technologies and pharmacological agents are poised to revolutionize hot tooth management further. Selective Nav1.8 channel blockers are a promising class of targeted analgesic/anesthetic agents that may specifically target the TTX-R channel up-regulation responsible for anesthesia failure without the systemic cardiovascular and central nervous system effects of conventional local anesthetics. Computer-controlled local anesthetic delivery systems with real-time pressure feedback and guided intraosseous approach technologies are making supplemental anesthesia techniques more precise and better tolerated by patients. Advances in regenerative endodontics and bioactive material science are constantly refining the evidence base for vital pulp therapy, expanding the range of viable treatment options for mature teeth with irreversible pulpitis beyond conventional root canal treatment (Roy & Narahashi, 1992). The advent of artificial intelligence-based diagnostic tools that integrate clinical history, laboratory data, and imaging findings is starting to aid clinicians in the accurate diagnosis of hot teeth and in treatment planning. With the advent of personalized medicine in dentistry, genotyping for ion channel polymorphisms and metabolic enzyme variants may allow for individualized pharmacological strategies to optimize anesthetic efficacy in individual patients (Asgary, *et al.* 2023).

CONCLUSION

The hot tooth is one of the most challenging clinical scenarios in modern endodontics, with a complex interaction of neurosensory dysregulation, pharmacological constraints, anatomical variations, and the psychological aspects of the patient. Accurate diagnosis, pharmacological premedication with NSAIDs and/or corticosteroids, a primary IANB administered with optimal technique, and a predetermined cascade of supplemental anesthesia with intraosseous injection as the preferred second-line modality can provide a structured, evidence-based approach to profound pulpal anesthesia in the presence of irreversible pulpitis. The growing body of evidence supporting vital pulp therapy, particularly full pulpotomy with bioceramic

materials, provides a less invasive and potentially less expensive alternative to root canal therapy in well-selected cases, reflecting the overall trend towards tissue-preserving and biologically driven endodontics. Effective postoperative pain management with NSAIDs, occlusal adjustment, and patient-centered communication complete the therapeutic cycle. Bridging the gap between the best available evidence and clinical practice requires focused continuing education, access to adjunctive anaesthesia equipment and materials, and development of a systematic, patient-centered mindset that sees the hot tooth not as an insurmountable obstacle, but rather as a predictable clinical challenge that can be systematically solved.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest

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Not applicable

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The authors declare that no AI and related tools are used to write the scientific content of this manuscript.

DATA AVAILABILITY

Data will be available on request

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