



# NEW ADVANCES IN THE MANAGEMENT OF ENDODONTIC PAIN EMERGENCIES

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## ABSTRACT

The development of more effective strategies for managing endodontic pain emergencies draws from the research of both clinicians and basic scientists. This review explores evidence-based approaches for managing endodontic pain emergencies and newly emerging pain management strategies based upon molecular, cellular and physiologic research into pain mechanisms.

The successful management of the endodontic emergency patient represents a challenging test of clinical acumen, requiring skills in diagnosis, endodontic treatment, and clinical pharmacology. Although this scenario is often perceived as stressful to both the patient and clinician, we believe that skills leading to the effective and efficient management of these cases provide a high point of satisfaction to many practitioners. This review will focus on current evidence-based reviews and basic research fronts likely to provide effective strategies for managing endodontic pain patients. One overall strategy that has gained rather broad acceptance is the use of a structured mnemonic approach for evaluation and treatment of these patients. This “3D” approach consists of Diagnosis, Definitive dental treatment and Drugs.

Diagnosis is clearly a critical first step in evaluating any pain patient (Table 1). Several factors must be kept in mind when examining the emergency pain patient.<sup>1</sup> First, the patient’s chief complaint should be noted. Important characteristics of the pain (location, spontaneity, effect of temperature or chewing, pain quality, etc.) should be elicited from the patient. These features are important not only in establishing the

differential diagnosis, but also in confirming the suspected tooth. For example, a patient with a chief complaint of severe lingering pain to cold would be expected to report these same symptoms when the suspected tooth is selectively stimulated with cold. In some cases, this may require rubber dam isolation of individual teeth in order to selectively stimulate the suspected tooth. Although selective testing often consumes additional examination time, the failure to reproduce the patient’s chief complaint is a risk factor for a mis-diagnosis and should prompt the prudent practitioner to consider alternative diagnoses. The clinical examination should evaluate for potential etiologic factors of odontogenic pain (eg. caries, open margins of

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Table 1

DIAGNOSTIC STEPS IN EVALUATING THE ACUTE ODONTOGENIC PAIN PATIENT
Establish the Chief Complaint Patient's words History of CC (When did it start? Stop? What makes it worse? Better? Location?) Nature of pain (dull, throbbing, sharp, shooting, burning, etc)
Review Medical and Dental History
Clinical Examination Visual inspection Periodontal probing Periapical percussion/palpation Pulpal tests
Radiographic exam
Differential Diagnosis Endodontic diagnosis as a dual diagnosis

restorations, crown-root fractures) as well as potential pathophysiologic sequelae of pulpal or periradicular disease (eg. sinus tract, intra/extraoral swelling, lymphadenopathy). Clinical examinations should include assessment of pulpal responsiveness (typically using cold or electrical stimuli), mechanical allodynia (eg. percussion or palpation of teeth and soft tissues), periodontal pocketing. The radiographic exam should include evaluation of both suspected teeth (coronal examinations focusing on potentially open margins, pulp stones, etc, and radicular examinations focusing on loss of the lamina dura, periradicular radiolucencies or opacities, resorptive processes) as well as examination of other visible structures. It is the total analysis of these data that form the basis for establishing the differential diagnosis. In rare cases, interventional tests (eg. anesthetic injection, test cavity) may be indicated to verify the differential diagnosis. However, most clinicians would agree that the practitioner's ears (eg. skills in listening and soliciting a history) often provide the most important diagnostic information.

The development of a differential diagnosis must include consideration of both odontogenic and non-odontogenic forms of pain (**Table 2**). Although space limitations preclude a comprehensive review on this topic, readers can refer to several recent and comprehensive reviews.<sup>2-7</sup> These reviews provide important and practical information on differential diagnosis and management of a wide range of non-odontogenic pain disorders that have been reported to mimic odontalgia.

The rationale for developing a differential diagnosis is obvious, but its application is challenging. The vast majority of dental pain emergencies consist of disorders with relatively little diagnostic challenge (eg. pulpal necrosis with acute apical abscess). This dominant pattern of odontogenic pain disorders may lead to clinical shortcuts in which a differential diagnosis is never established. Under these conditions, the patient presenting with a rare but very real form of non-odontogenic pain may not receive appropriate therapy due to a misdiagnosis. Thus, the challenge is to maintain vigilance in evaluating patients and developing differential diagnoses.

Table 2

SELECTED DIAGNOSES THAT CAN MIMIC ACUTE ODONTOGENIC PAIN
Diagnosis
Odontogenic Pain* Dental Hypersensitivity Reversible pulpitis Irreversible pulpitis Acute apical periodontitis Acute apical abscess
Non-Odontogenic pain - Musculoskeletal Myofascial pain - TMD Bruxism
Non-Odontogenic pain - Neuropathic Trigeminal neuralgia Atypical odontalgia Glossopharyngeal neuralgia
Non-Odontogenic pain - Neurovascular Migraine Cluster headaches
Non-Odontogenic pain - Inflammatory Allergic sinusitis Bacterial sinusitis
Non-Odontogenic pain - Systemic disorders Cardiac pain Herpes zoster Sickle cell anemia Neoplastic disease
Non-Odontogenic pain - Psychogenic origin Munchausen's Syndrome
*Odontogenic pain may arise from the suspected tooth, or the pain may be referred from another tooth.

The second "D" in the "3D" algorithm is for definitive dental treatment. Too often the busy practitioner favors simple pharmacotherapy over definitive dental treatment as an efficient means of managing the unscheduled emergency. However, numerous studies have established clearly that the delivery of appropriate dental procedures offers significant and substantial reductions in



Table 3

**COMPARATIVE EFFICACY OF ORAL ANALGESICS\***

Analgesic	% Patients with 50% Pain Relief **	Sample Size (N)
Ibuprofen 800 mg	100%	76
Ibuprofen 600 mg	79%	203
Acetaminophen 650 mg + Oxycodone 10 mg	66%	315
Acetaminophen 1,000 mg + Codeine 60 mg	57%	197
Ibuprofen 400 mg	56%	4,703
Morphine 10 mg IM injection	50%	946
Acetaminophen 1,000 mg	46%	2,759
Ibuprofen 200 mg	45%	1,414
Acetaminophen 600/650 mg	38%	1,886
Tramadol 100 mg	30%	882
Codeine 60 mg	15%	1,305
Placebo	18%	>10,000

\* Data from: The Oxford League Table of Analgesic Efficacy (URL: <http://www.jr2.ox.ac.uk/bandolier/booth/painpag/Acutrev/Analgesics/lftab.html>)

\*\* Percentage of patients with moderate to severe pain who report at least 50 percent pain relief at four hours to six hours after taking medication. Data are from randomized, double-blind, placebo-controlled analgesic clinical trials.

odontogenic pain due to acute inflammatory processes.

Pulpotomies can be considered for treating cases of irreversible pulpitis even when the pain is severe in magnitude. The procedure consists of anesthesia, rubber dam isolation, access, removal of pulp chamber contents with a sharp spoon, and avoiding penetration of the root canal systems. In addition to its technical simplicity, pulpotomies have been shown to result in a significant and substantial reduction in pain by as little as 24 hours after the procedure.<sup>8-10</sup> Thus, pulpotomies represent a viable approach for managing even moderate to severe odontogenic pain due to irreversible pulpitis.

Pulpectomies include debridement of the root canal systems and are typically performed on necrotic cases or in those vital cases where sufficient time is available for this longer clinical procedure.

The pathophysiology of pain associated with pulpal necrosis is more complex than pain associated with irreversible pulpitis and therefore the mechanisms for the pain relief differ between pulpectomy and pulpotomy. Pulpectomies only indirectly influence periradicular nociceptors by their removal of irritants from necrotic root canal systems. In contrast, pulpotomies directly reduce pulpal nociceptor activity via the axotomy that occurs during removal of dental pulp in the pulp chamber space. Although the mechanisms for pulpectomy-induced pain relief is more complex, clinical trials often demonstrate a significant and substantial reduction in pain by 24 hours to 36 hours after pulpectomy treatment.<sup>9,11-16</sup>

Other dental procedures have been shown to reduce odontogenic pain. For example, occlusal adjustment reduces post-endodontic pain, particularly in patients presenting with pre-operative

pain, percussion sensitivity or vital teeth.<sup>14</sup> An incision for drainage procedure is indicated for management of swellings due to an infectious process, but is useful both for control of infection and the related pain.

If the tooth has a hopeless prognosis, extraction of painful but non-restorable teeth reliably reduces pain. In one study, patients with moderate to severe pre-operative pain reported pain as none to mild by two to three days after dental extraction.<sup>17</sup> Of course, the purpose of endodontics is to save teeth, and as described above, endodontic treatment significantly reduces odontogenic pain.<sup>8-16</sup>

The third “D” in the “3D” algorithm is for drugs. Non-steroidal anti-inflammatory drugs (NSAIDs) have been demonstrated to be effective in treating odontogenic pain. Randomized placebo-controlled studies in endodontic pain

patients have reported significant analgesic benefit from ibuprofen (400 mg to 600 mg), flurbiprofen (50 mg to 100 mg) and ketorolac (30 mg to 60 mg), with comparatively little benefit from etodolac (400 mg).<sup>10,11,15,16,18-20</sup> More recent studies have indicated that the COX-2 isozyme is upregulated in inflamed human dental pulp and that rofecoxib (50 mg) significantly reduced post-endodontic pain compared to placebo.<sup>20,21</sup> However, the analgesic benefit of rofecoxib 50 mg was similar to that observed in the first six hours following administration of ibuprofen 600 mg. Thus, the Coxib class of NSAIDs appear effective in treating odontogenic pain, but not superior to the mixed COX1-2 inhibitors such as ibuprofen. For patients with contraindications for NSAIDs, acetaminophen (650 mg to 1000 mg) alone or with an opioid appear effective for treating post-endodontic pain.<sup>11</sup> A very useful Web site for comparing analgesic effectiveness for acute inflammatory pain is the Oxford League Table of Analgesic Efficacy.<sup>22</sup> This group uses evidence-based analyses to develop comparison among analgesics for patients with moderate to severe pain. A summary of these data are presented in **Table 3** and are the results of a large and ongoing meta-analysis of analgesic clinical trials across multiple acute pain conditions. A meta-analysis of clinical trials evaluating oral NSAIDs for post-endodontic pain has been published recently with similar results.<sup>23</sup>

Recent studies indicate that NSAIDs such as ibuprofen appear to interfere with the anti-platelet effects of aspirin.<sup>24</sup> In this study, the administration of rofecoxib or acetaminophen had no effects on the anti-platelet effects of aspirin. Moreover, a retrospective study of more than 7,000 post-myocardial infarction patients taking low-dose aspirin therapy over several years found a significantly elevated hazard ratio for cardiac death in patients also taking NSAIDs.<sup>25</sup> This finding has led to the

suggestion that patients taking “baby” aspirin should not take certain NSAIDs under chronic conditions. It is not yet known whether a short-term course of NSAID treatment imposes a medically significant risk for cardiac death in certain patients. However, these data are consistent with the consideration of alternative analgesics such as acetaminophen combinations or COX2 inhibitors in patients taking low-dose aspirin for medical indications.

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NSAIDs are effective for treating the majority of endodontic pain patients who can tolerate this class of analgesics. However, a small subset of patients may still experience significant pain even after consuming maximal dosages of NSAIDs. Therefore, under certain conditions, the co-administration of NSAIDs with other analgesics may be indicated for short-term (one- to two-day) treatment of severe pain. For example, a randomized placebo-controlled clinical trial in post-endodontic patients demonstrates that the opioid tramadol (Ultram) significantly enhanced NSAID analgesia.<sup>16</sup> In addition, randomized placebo-controlled clinical trials demonstrate that acetaminophen (650 mg to 1,000 mg) significantly enhances NSAID analgesia in both oral surgery and post-endodontic pain trials.<sup>26,27</sup> Given the substantial reduction in pain that occurs typically with pulpectomy or pulpoto-

my procedures, NSAID therapy alone is sufficient for the majority of patients who can tolerate this class of analgesics. However, opioids or acetaminophen co-treatment may serve an important adjunctive role when combined with NSAIDs role in managing severe odontalgia. When indicated, these adjunctive therapeutics are usually provided for one to two days after treatment due to the substantial reduction in pain that occurs due to the definitive dental treatment.

Many practitioners report difficulty in obtaining effective local anesthesia when treating the patient with severe odontalgia. Proposed mechanisms for local anesthesia failure have been reviewed recently and include upregulation of sodium channels resistant to local anesthetics (eg. NaV1.8, NaV1.9), central sensitization.<sup>28</sup> Clinical trials in endodontic patients indicate clearly that a positive “lip sign” does not predict effective pulpal anesthesia (see 28), and thus clinicians should not interpret a lip sign as a marker for effective pulpal anesthesia. Although some textbooks claim that lip sign predicts pulpal anesthesia, this is not supported by clinical research and recent basic research indicates that this disparity is likely due to the fact that unmyelinated nociceptors are more resistant to lidocaine as compared to the A delta fibers that mediate touch or proprioception.<sup>29</sup>

One effective strategy for enhancing anesthesia in odontalgia patients is the use of the intraosseous route of injection.<sup>30</sup> Clinical trials in odontalgia patients demonstrate that intraosseous anesthetic injection significantly enhances anesthesia due to inferior alveolar nerve block.<sup>31</sup> The use of vasoconstrictor-containing local anesthetics is associated with a transient tachycardia after intraosseous injection.<sup>32</sup> The use of 3% mepivacaine without vasoconstrictor avoids tachycardia but has a shorter duration of action.<sup>32</sup> Thus, the intraosseous route of injection represents an evidence-based adjunct for the difficult



to anesthetize mandibular tooth.

Many randomized, double-blind, placebo-controlled clinical trials have evaluated the effectiveness of antibiotics for reducing endodontic related pain. Most, but not all have failed to detect any difference from placebo for post-endodontic pain, swelling, flare-up or analgesic consumption.<sup>17,33-36</sup> Thus, the preponderance of evidence does not support a beneficial role of antibiotics for odontogenic pain. This is clearly an area of continuing interest and continued research in this area is warranted.

While this represents the current state of the art in pain control, ongoing basic research offers the potential for new approaches for pain control.

## Summary

Taken together, the "3D" mnemonic of Diagnosis, Definitive Dental Treatment and Drugs represents an effective, efficient, and evidence-based approach for managing the endodontic pain patient. This review has highlighted the continued development of more effective and predictable methods for pain control as a process that combines advancements in both the basic science and clinical arenas. This is most certainly an ongoing process, and emphasizes the need for life-long learning as epitomized by the evidence-based approach to therapeutics. It is hoped the reader has found this material both stimulating and clinically relevant, with the ultimate effect of improved care for patients in pain. **CDA**

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