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Building effective strategies for the management of endodontic pain

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Recent advances in the understanding of pain physiology and pharmacology have provided dental practitioners with more reliable information upon which clinical decisions in the treatment of endodontic pain can be based. This article provides practical treatment strategies based upon current scientific evidence and uses information presented in the prior articles of this issue of *Endodontic Topics* to make treatment recommendations for given patient scenarios.

Managing pain can be one of the more challenging aspects of the clinical practice of endodontics, and one by which the skill of the clinician is often judged. Unfortunately, root canal therapy is still perceived by many as a standard against which painful experiences are judged. This is a two-edged sword. Because patients are expecting pain, it often makes their pain management more difficult. On the other hand, advances in local anesthesia and modern pharmacology allow dental practitioners to deal effectively with the patient experiencing odontogenic pain and, in most cases, exceed their expectations.

Endodontic pain management encompasses all aspects of treatment: preoperative pain control includes accurate diagnosis and anxiety reduction; intraoperative pain control revolves around effective local anesthetic and operative techniques; and postoperative pain management can involve a variety of pharmacologic agents. All of the above demand a thorough understanding of the pathophysiology of the pain system (1–11). This article reviews treatment strategies for managing acute odontogenic pain, emphasizing the importance of diagnosis, treatment and clinical and pharmacological therapeutic interventions.

Diagnosis of the endodontic pain patient

The initial phase of treating the endodontic pain patient is diagnosis. Diagnosis must be the starting point for pain treatment since many conditions can mimic odontogenic pain but do not necessarily require endodontic treatment (12–19). A classic example is the patient presenting with dull aching pain in the maxillary posterior teeth; obviously, the differential diagnosis must consider both sinusitis as well as odontogenic sources of pain. Thus, developing a differential diagnosis is an essential first step in effective pain management strategies. Table 1 provides an overview of major clinical conditions that can mimic odontogenic pain. Although the majority of patients who present with a complaint of tooth pain actually suffer from odontogenic pain, it is clear that this is not always the case. The astute clinician will consider these alternative pathoses given the presenting signs, symptoms and results from the clinical exam since, of course, the treatment strategies and prognoses depend upon the diagnosis.

Typically, the patient in need of endodontic evaluation is experiencing some sort of pain, has heard horror stories about ‘root canals’, and is in an anxious

state of mind. It is imperative therefore that the clinician should remain objective and perform the necessary diagnostic procedures in a methodical, consistent manner, so as not to be misled by the patient's misperceptions. The following represents a logical sequence to follow for any endodontic evaluation.

Diagnostic sequence

Establish chief complaint

Let the patient describe the chief complaint in their own words. By listening carefully without interrupting, you can gather important diagnostic information and take the first step in establishing rapport. If the patient sees that you are rushed, or jump to conclusions about their symptoms, they are likely to become more anxious, and consequently more difficult to treat (20). Many experienced clinicians summarize this advice by pointing out that the best diagnostic tool is their ears – patients often provide critical diagnostic information while they describe their chief complaint.

Table 1. Selected diagnoses that can mimic acute odontogenic pain

Odontogenic pain*
Dentinal 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..
Modified from: Seltzer & Hargreaves (18).

History of chief complaint

Once the patient has described their chief complaint, questions can then be directed to the nature of their discomfort that aid in the diagnosis:

When did the pain begin? Pain of pulpal etiology often has a rapid onset, and gets more intense and more localized. The patient who describes a dull discomfort that has been present for several months is not likely experiencing pain of pulpal origin.

Is the pain spontaneous? As described previously, spontaneous pain is a hallmark of hyperalgesia, and is often a sign of irreversible pulpal inflammation.

Does anything make it worse? Mechanical and thermal allodynia are to be expected in inflammatory pain conditions therefore exacerbation of the chief complaint by biting or exposure to varied temperatures are common findings. If the inflammation is limited to the pulpal tissues, it may be difficult for the patient to localize the offending tooth, due to the limited distribution of the discriminative touch receptors ('proprioceptors') in the pulp. It is imperative that the clinician reproduce the chief complaint. The ability to reproduce the chief complaint is a major finding in reducing the chance of misdiagnosis of odontogenic vs. non-odontogenic pain.

Medical and dental history

The need for a thorough medical history is obvious. The information obtained may alter the examination process (e.g. the need for S.B.E. prophylaxis), and/or influence the diagnosis (e.g. referred pain from carotid artery disease). Suffice it to say that a medical history must be obtained before *any* type of examination is performed. The dental history is also quite important and may offer clues not only to the etiology of the current problem, but also helps determine the patient's motivation to retain their teeth, which may impact on treatment planning decisions.

Clinical examination: Visual inspection

Examine the soft tissues for any sign of swelling, redness or sinus tracts that may be secondary to periapical pathosis; also look for other pathology that may be the cause of the chief complaint (e.g. an apthous ulcer that causes constant pain). Inspect the teeth carefully for evidence of caries, cracks, exposed dentin, defective restorations, etc. There ought to be a reasonable cause for pulpal disease. In the absence of an obvious etiologic agent, the clinician should consider a non-odontogenic source for the patient's discomfort.

Periodontal probing

It is not only important to assess the general periodontal health of the teeth in question, but to look for isolated defects as well. Probe the periodontal attachment by 'walking' the probe around the sulcus, so as not to miss an area of attachment loss that may be secondary to a vertical root fracture (21), or a sinus tract which may be draining through the sulcus.

Periapical tests

Exaggerated responses to percussion and/or palpation indicate inflammation or infection of the periradicular tissues. Start with control teeth to give the patient an idea of what to expect, then look for a significantly different response in the teeth in question. Palpate the soft tissues at the estimated level of the apices of the teeth being examined, both on the labial and lingual sides. Again, start with an area some distance from the suspected tooth or teeth, then slowly move the pressure of a gloved finger towards the target area.

It is important to note that there may be irritation of the periradicular tissues that is not secondary to pulpal pathosis. Traumatic occlusion, sinusitis, and periodontal disease are all examples of potential sources of periradicular symptoms.

If a patient's chief complaint is pain on chewing, it is advisable to attempt to recreate the complaint with a bitestick. There are several available that allow the clinician to localize the force of occlusion on isolated cusps (e.g. ToothSlooth™). A sharp, non-lingering pain on biting or release of biting pressure, in the absence of percussion and palpation tenderness, is a typical finding with a cracked tooth. If the pulp tests are normal, the treatment of choice is the placement of a casting that covers the cusps of the cracked tooth (without endodontic therapy) (22).

Pulp tests

In performing pulp tests, the clinician is attempting to determine if the pulp is vital or non-vital, and when vital, if the pulp is reversibly or irreversibly inflamed. Although clinical tests do not always correlate with pulpal histopathosis, when combined with history and radiographic examination, they provide a means for clinical decision-making.

Cold tests will allow for a definitive pulpal diagnosis most of the time (23). The test that recreates the patient's chief complaint should be employed. A cold stimulus can be applied with the use of carbon dioxide snow, or chlorofluorocarbons sprayed on a cotton pellet on the buccal or labial surface near (but not on) the gingiva of the tooth being tested. If using a saturated cotton pellet, beware of false positive results in teeth with extreme percussion/palpation sensitivity that are responding not to the temperature, but to the pressure of the pellet on the tooth. Heat can be easily applied to the buccal or labial surfaces by using a stick of impression compound or gutta percha stopping. Exaggerated, lingering responses to either hot or cold are generally indicative of irreversible pulpal inflammation, and further testing is not necessary. Vital pulps that are reversibly inflamed will give an exaggerated response to temperature stimulation (typically cold), but the pain will generally not linger, as compared to control teeth.

Electric pulp testing should be performed when the results of thermal testing are inconclusive. This is often the case in geriatric patients whose pulps have created a significant amount of reactionary dentin, and are unresponsive to temperature changes. If electric pulp testing cannot be performed due to the presence of a full coverage restoration, it may be necessary to cut a test cavity preparation. This is a class I cavity preparation made without anesthesia, which exposes dentin and stimulates afferent sensory fibers by hydrodynamics. If the pulp is necrotic, the operator can slowly advance the bur until the chamber is entered.

If the pulp is vital, the patient should experience a sharp pain before reaching the chamber. One could also stop after reaching dentin, and use an electric pulp tester with a micro tip to stimulate the dentin thus exposed. It should be emphasized that the level at which a pulp responds to an electric pulp tester is *relatively* meaningless. A positive response merely indicates the presence of A-delta nerve fibers, and cannot be used to determine the degree of inflammation (24).

Radiographic examination

It is tempting in many cases to make an endodontic diagnosis based solely on a radiograph. Radiographs are but *one piece* of the diagnostic puzzle! The information obtained must be correlated with clinical findings and histories to avoid jumping to the wrong conclusion. A minimal preoperative radiographic examination includes a parallel periapical radiograph, and it is often desirable to expose an angled (horizontal) periapical film as well in order to reconstruct the 3-dimensional nature of the teeth in question. If restorability is an issue, a bitewing radiograph is helpful to estimate the proximity of caries to furcation or crestal bone.

The *entire* radiograph should be carefully examined. The coronal tooth structure should be assessed for caries and/or restorations that may be causing pulpal pathosis. When determining the presence or absence of periapical pathology, it is often helpful to follow the periodontal ligament space and lamina dura beginning at the crestal bone, to the apices and back. Pulpal pathosis that extends through portals of exit will cause widening of the PDL space and breakdown of the lamina dura adjacent to the involved foramina. This is particularly challenging to differentiate from the norm in areas with superimposed radiolucency, such as the maxillary sinus, mental foramen, lingual salivary gland depression and inferior alveolar canal. In the case of the latter, the PDL space at the apices of mandibular second molars often *appears* widened in the normal condition.

Dual diagnosis

While endodontic pathosis begins with a diseased pulp, eventually the periradicular structures become involved via communications (i.e. apical foramina, lateral canals, etc.) between the pulp and periodontium. It is important, therefore, that a diagnosis be made of both the pulp and the periradicular tissues, as treatment decisions may be affected.

Non-odontogenic pain

Many excellent reviews are available on this topic (12–19, 25). Table 2 provides an overview of selected clinical features that should prompt immediate expansion of the differential diagnosis to include non-odontogenic pain conditions (18).

Table 2. Selected features of non-odontogenic dental pain

No apparent etiologic factors for odontogenic pain (no caries, leaky restorations, trauma, fracture, etc.)
Pain not consistently relieved by local anesthetic injection
Bilateral pain or multiple teeth are painful
Pain can be chronic and not responsive to dental treatment
Diagnosis-specific pain* qualities: burning, electrical shooting, stabbing, dull ache
Diagnosis-specific: pain concurrent with a headache
Diagnosis-specific: palpation of trigger points or muscles can increase pain
Diagnosis-specific: pain increased by emotional stress, physical exercise, head position, etc.

*'Diagnosis-specific' implies that these features are seen for some, but not all forms of non-odontogenic pain.
Modified from: Seltzer & Hargreaves, 2002 (18).

Table 3. Clinical cases

Case #1 – 46-year-old female

Chief complaint

'I have a constant ache in my upper back teeth on the left side'.

History of chief complaint

The pain started about 2 weeks ago. The pain is spontaneous and only increases when I bend over to tie my shoes. No change to temperature, some increase when I bite down. The pain is scored as a number '4' on a 0–10 pain scale.

Medical History

Non contributory.

Dental History:

No recent dental treatment in left maxillary or mandibular quadrants.

Clinical exam

Tooth #	Thermal test (ice)	Percussion	Tooth Slooth	Perio Pockets	Restorative
13	+	+	+	3–4 mm	sealed Cl II Amalgam
14	+	+	+	3–4 mm	sealed Cl II Amalgam
15	+	+	+	3–5 mm	sealed Cl II Amalgam

Testing of teeth #18–21 are within normal limits

Radiographic exam

No periradicular radiolucencies or caries noted on #12–15

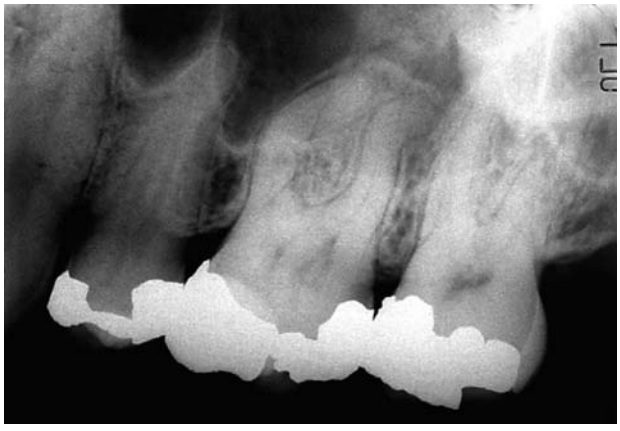
Diagnosis?

Pain treatment plan?

Endodontic treatment plan?

Restorative treatment plan?

See text for a discussion of this case.



Radiograph Case #1.

Treatment of the endodontic pain patient

As described in preceding review articles in this issue of *Endodontic Topics*, optimal pain management combines both pharmacological and non-pharmacological treatment strategies. For example, pulpotomy treatment has been reported to reduce pain symptoms in nearly 90% of patients 1 day after treatment (26). This finding has been confirmed by many other studies (Fig. 1). Indeed, both pulpotomy and pulpectomy are effective treatments for reducing postoperative pain regardless of whether any analgesics are prescribed. As described by Dr. Rosenberg, other non-pharmacological treatments (anxiety-reducing strategies, occlusal reduction, etc.) have also been shown to reduce patient apprehension and pain.

The biological reason why pulpotomies and pulpectomies reduce pain is based on reducing tissue levels of inflammatory mediators and the elevated interstitial tissue pressure that stimulate peripheral terminals of nociceptors (7, 27, 28). Thus, the effective debridement of the infected root canal system, combined, when indicated, with incision for

Table 3. continued

Case #2 – 35-year-old male

Chief complaint

'I have a severe toothache down here (points to tooth #30)'.

History of chief complaint

The pain started about 3 days ago. The pain is spontaneous and increases when I drink something cold or bite down. The pain is scored as an '8' on a 0–10 scale.

Medical history

Non contributory.

Dental history:

No recent dental treatment in right mandibular or maxillary quadrants.

Clinical exam

Tooth #	Thermal test (ice)	Percussion	Tooth Slooth	Perio Pockets	Restorative
28	+	–	–	3–5 mm	sealed Cl II Amalgam
29	+	–	–	2–3 mm	sealed Cl II amalgam
30	+ +Lingers	+	+	2–4 mm	caries under crown
31	++	–	–	3–4 mm	sealed crown

Testing of teeth #2–11 are within normal limits
(Note: ' ++' indicates severe response to this test)

Radiographic exam

No periradicular radiolucencies noted on #28–31. Caries evident on #30.

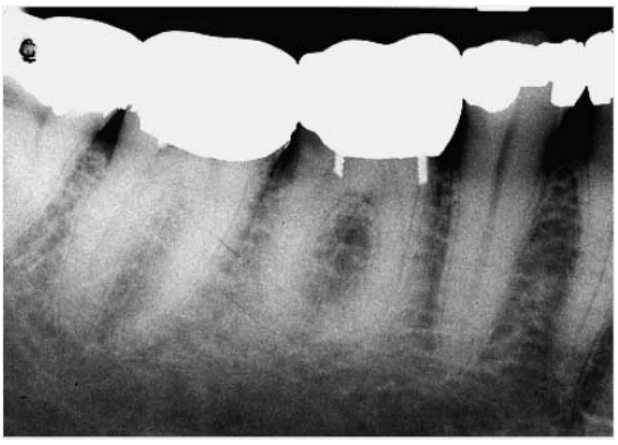
Diagnosis?

Pain treatment plan?

Endodontic treatment plan?

Restorative treatment plan?

See text for a discussion of this case.



Radiograph Case #2.

drainage and occlusal reduction, provides predictable pain reduction strategies in endodontic emergency patients (7, 27–29). Of course, if the tooth has a hopeless prognosis, then extraction will also reduce pain by reducing tissue levels of these factors. From this perspective, it can be concluded that treating the unscheduled emergency patient by the 'prescription pad' (i.e. by drugs alone) is not a definitive intervention. Instead, the pharmacological management of pain should be considered together with definitive dental treatment as a combined therapeutic approach for managing odontogenic pain. Dr. Rosenberg's paper provides an excellent review of modalities for the treatment of endodontic pain (including the use of antianxiety agents and techniques), as does Dr. Walton's paper on the treatment of the endodontic flare-up.

Pharmacologic strategies for postoperative endodontic pain

Several pharmacological strategies for pain control have emerged over the last 10 years and will be re-

Table 3. continued

Case #3 – 67-year-old female

Chief complaint

'I have a severe toothache down here (points to tooth #20)'.

History of chief complaint

The pain started about 5 weeks ago and has gradually increased. The pain is spontaneous and increases when I bite down. The pain is scored as an '6' on a 0–10 scale.

Medical history

Hypertension. Patient reports taking hydrochlorothiazide and captopril.

Dental history

No recent dental treatment in right mandibular or maxillary quadrants.

Clinical exam

Tooth #	Thermal test (ice)	Percussion	Tooth Slooth	Perio Pockets	Restorative
21	+	–	–	3–5 mm	sealed Cl II Amalgam
20	–	++	+	2–3 mm	open margin on crown
19	+	–	–	2–4 mm	sealed Cl I Amalgam

Testing of teeth #12–15 are within normal limits
(Note: '++' indicates severe response to this test)

Radiographic exam

No periradicular radiolucencies noted on #19, 21. Tooth #20 has 5 mm periradicular radiolucency.

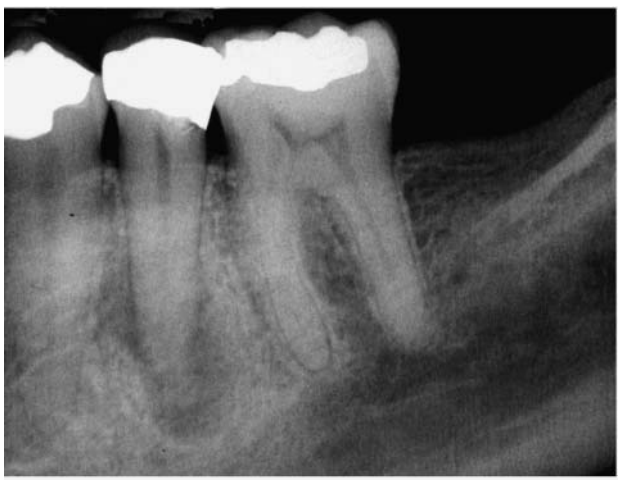
Diagnosis?

Pain treatment plan?

Endodontic treatment plan?

Restorative treatment plan?

See text for a discussion of this case.



Radiograph Case # 3.

viewed here. The reader is most certainly encouraged to read the companion papers in this journal for excellent specific reviews.

Non-narcotic analgesic dosing

The first strategy is to prescribe the right non-narcotic analgesics at the right dosages (30–34). Here we can make use of the systematic review of NSAIDs for treating endodontic pain, as described in the article by Dr. Holstein and colleagues. For example, pretreatment with either ibuprofen (800 mg) or flurbiprofen (100 mg) is effective for management of post-treatment pain (30–34). Some patients may not be able to tolerate NSAIDs. This might include patients with GI disorders (e.g. ulcers, ulcerative colitis), active asthmatics or hypertension (due to renal effects of NSAIDs as well as drug : drug interactions with many antihypertensive drugs). For those patients who cannot take NSAIDs, pretreatment with acetaminophen (1000 mg) is also effective for reducing post-treatment pain (33).

As described in the excellent review by Khan & Dionne, the mechanism of action of most NSAIDS is

Table 3. continued

Case #4 – 43-year-old female

Chief complaint

'I had my root canal treated yesterday (tooth #14) and now my pain is severe and constant'.

History of chief complaint

There was no preoperative pain. The non-surgical root canal procedure was completed yesterday morning, and the pain gradually became severe by last night
The pain is scored as an '9' on a 0–10 scale.

Medical history

Not contributory. Patient has been taking ibuprofen 600 mg every 6 h since yesterday morning.

Dental history

No recent dental treatment in left mandibular or maxillary quadrants, with the exception of completion of root canal treatment in tooth #14 yesterday. Today is an un-scheduled appointment initiated by the patient. The preoperative diagnosis of tooth #14 was pulpal necrosis with acute apical periodontitis.

Clinical exam

Tooth #	Thermal test (ice)	Percussion	Tooth Slooth	Perio Pockets	Restorative
12	+	–	–	3–5 mm	sealed Cl II Amalgam
13	+	–	–	2–3 mm	sealed crown
14	–	++	+	2–4 mm	sealed Cavit™
15	+	–	–	3–4 mm	sealed Cl III Amalgam

Testing of teeth #17–22 are within normal limits
No lymphadenopathy, elevated temperature or intra or extraoral swellings are noted.
(Note: '++' indicates severe response to this test)

Radiographic exam

No periradicular radiolucencies noted on #12,13,15. Tooth #14 has 7 mm periradicular radiolucency.

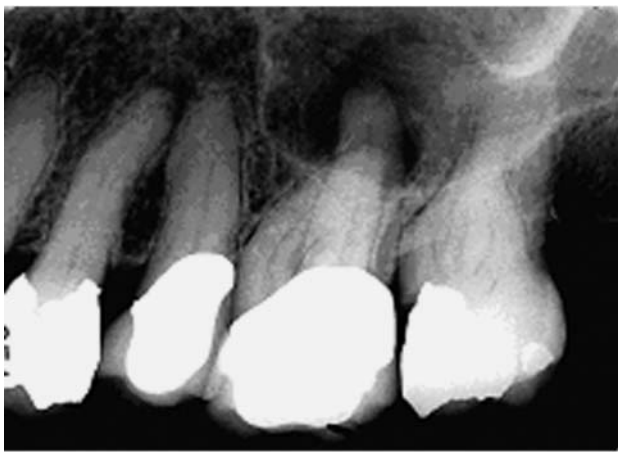
Diagnosis?

Pain treatment plan?

Endodontic treatment plan?

Restorative treatment plan?

See text for a discussion of this case.



Radiograph Case #4.

thought to involve the inhibition of the enzyme cyclooxygenase (COX) (31). The relative efficacy and safety of the COX-1 and COX-2 inhibitors are described in detail in their paper in this issue of *Endodontic Topics* and by others (35–38, 48). In addition, they described the recent discovery of the COX-3 enzyme that appears to be the CNS target for acetaminophen (39).

Pretreatment with NSAIDS for irreversible pulpitis should have the effect of reducing pulpal levels of the inflammatory mediator PGE_2. This would benefit in two ways. Firstly, decreasing pulpal nociceptor sensitization would mitigate an increase in resistance to local anesthetics (40). Secondly, it may diminish

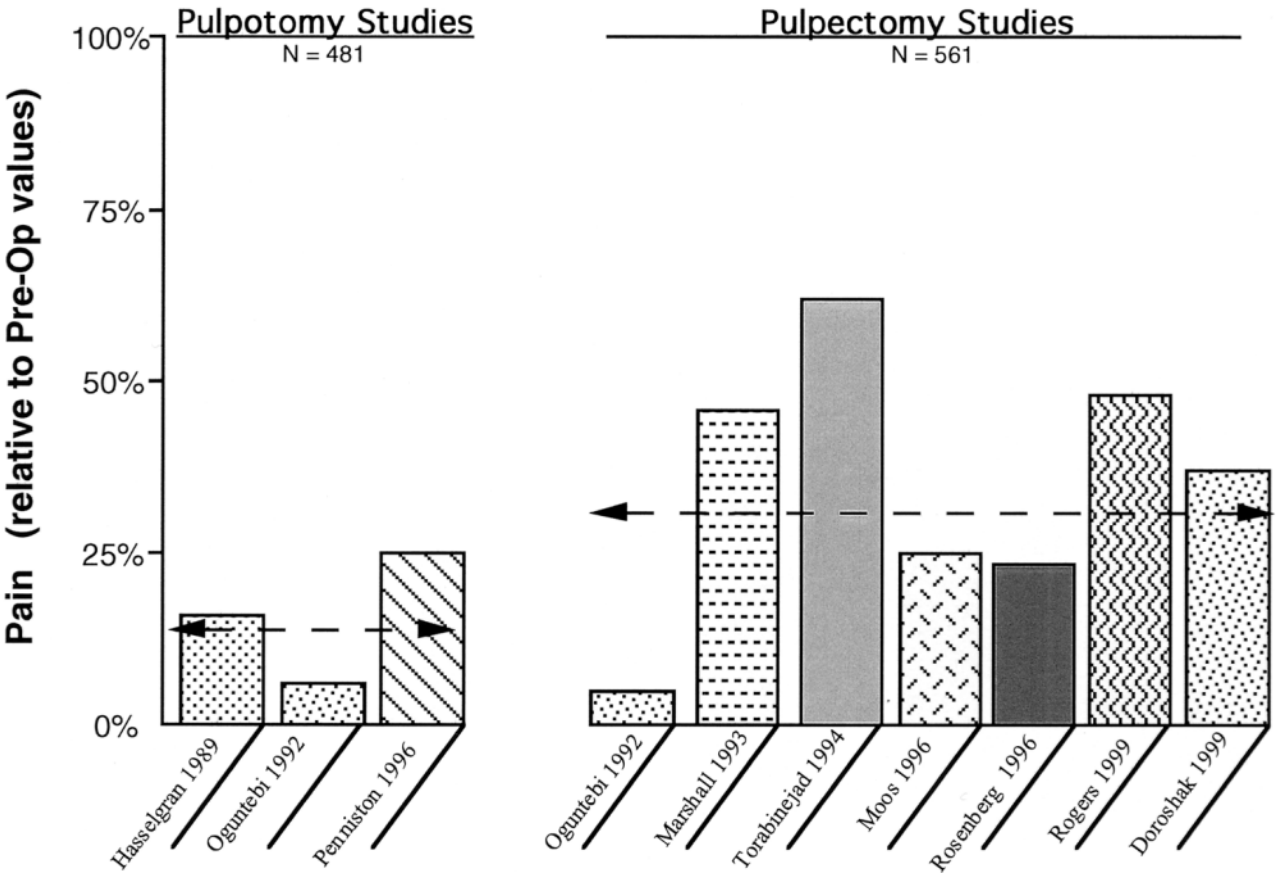


Fig. 1. Effects of definitive dental treatment on pain report in endodontic emergency patients. Preoperative pain was measured, the tooth was anesthetized, and treated with either a pulpotomy (left group of bars) or a pulpectomy (right group of bars), and pain was reassessed at a later time. Data are normalized across studies where '100%' represents the mean value of preoperative pain. Dashed horizontal lines represent that weighted mean postoperative pain (weighted by sample size for each study) for both pulpotomy (left horizontal line) and pulpectomy (right horizontal line) studies. From: Hargreaves KM & Baumgartner C. Endodontic therapeutics. In: Walton R, Torabinejad M, eds. *Principles and practice of endodontics.* Philadelphia: Saunders, 2002 533–544 (27).

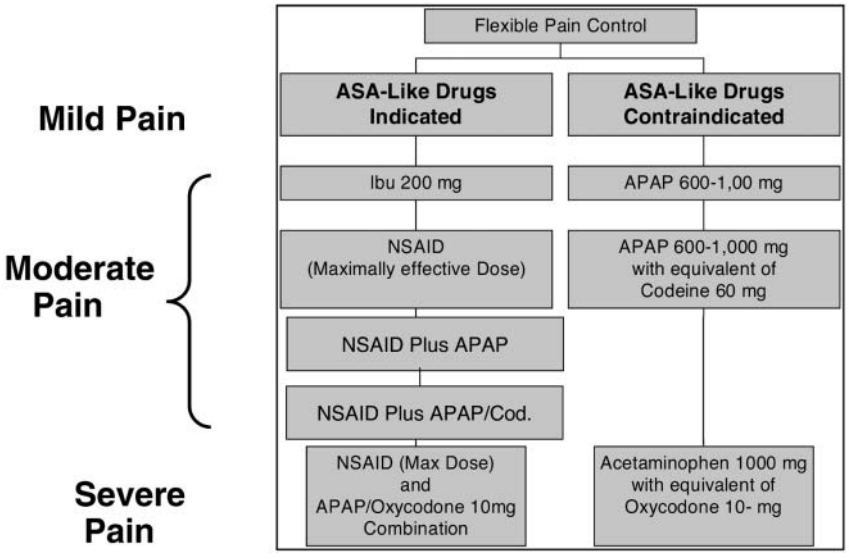


Fig. 2. Flexible prescription strategy for managing acute pain. The column on the left contains recommendations for patients who can take aspirin-like drugs, and the column on the right is for those patients in whom aspirin-like drugs are contraindicated. Each column of drugs is divided into three levels based on patient's report of pain magnitude. Modified from: Hargreaves KM & Seltzer S. Pharmacological control of dental pain. In: Hargreaves KM, Goodis HE, eds. *Seltzer and Bender's dental pulp*. Chicago: Quintessence, 2002: 205–226 (45).

a prostanoid-induced stimulation of TTX-resistant sodium channel activity; these channels also display relative resistance to lidocaine (41). Double blind clinical trials have shown that the injectable non-steroidal anti-inflammatory drug ketorolac tromethamine, when injected intraorally or intramuscularly, produces significant analgesia in patients with severe odontogenic pain prior to definitive treatment (42, 43). Although it has yet to be evaluated in endodontic pain patients, ibuprofen in a liquid gel formulation (e.g. Advil Liquid Gel®) may have similar effects.

Timing of drug administration

Although many clinicians develop a habit of writing analgesic prescriptions as take 'prn pain', several studies have argued that this is not the best way to have patients take their medication. If patients follow this advice and only take their analgesics 'as needed for pain', then there will be a delay (usually up to 1 h) after taking their medication when they are still experiencing pain. To avoid this problem, we suggest clinicians write prescriptions for patients to take their analgesics 'by the clock' (e.g. write the prescription 'q6h' or q8 h etc., depending on the formulation). Instructing patients to take their analgesics by the clock for the first few days provides a more consistent blood level of the drug and may contribute to more consistent pain relief.

Flexible prescription plan

The development of a flexible prescription plan has been proposed (7, 27) as a strategy to balance the patient's need for analgesics against the potential adverse side-effects of these drugs. Figure 2 represents such a strategy. This strategy consist of two parallel approaches (Fig. 2 is divided into two columns, the column on the left is for patients who can tolerate NSAIDs and the column on the right is for those who cannot take NSAIDs). Each column is divided into three sections (based on patients reporting pain as slight, moderate or severe) and recommended analgesics are listed. The objective of this flexible prescription plan is to obtain maximal analgesic benefits with minimal exposure to side-effects.

This strategy is a result of numerous randomized controlled clinical trials evaluating analgesic drugs (7, 27, 30–33, 44, 45). The first objective is to maximize the dose of the non-narcotic (e.g. NSAID or acetaminophen) before prescribing an analgesic containing a narcotic. The rationale is that a maximally effective dose of a non-narcotic generally provides greater analgesia and fewer side-effects than a combination drug containing both a non-narcotic analgesic with an opioid.

A small proportion of patients may still report pain after administration of NSAID alone. Two alternative approaches have been proposed for treating this subpopulation of patients (45). The first approach will coprescribe an NSAID together with acetaminophen. These two drugs show additive analgesia when taken together for treating dental pain (46, 47). The simultaneous administration of acetaminophen and NSAIDs appears to be well tolerated in most patients when given over a short period of time (46–50). A second general approach involves combining an NSAID with an opioid or with an acetaminophen/opioid combination. For example, the combination of flurbiprofen and tramadol appears to be one of the most efficacious means of treating postendodontic pain (see article by Holstein and colleagues in this issue of *Endodontic Topics*).

Long-acting anesthetics

As previously discussed, by blocking the activation of unmyelinated C nociceptors, we not only provide anesthesia for our patients but, by decreasing the potential for central sensitization, we can provide analgesia as well. In this respect, it is important to remember that the long-acting local anesthetics (i.e. bupivacaine, ropivacaine and the recently discontinued etidocaine) can provide an increased duration of post-treatment analgesia beyond the period of anesthesia (51, 52). The long-acting local anesthetics can provide a duration of analgesia up to 8–10 h following block injections, and may reduce pain report even 48 h later (51). Since endodontic treatment by itself (e.g. pulpotomy, pulpectomy), often provides substantial pain relief by 24 h (Fig. 1), the long-acting local anesthetics when given by block injection represent a logical means of initiating the postoperative pain management plan. The relative benefits of intraosseous injection of local anesthetics are reviewed in the companion article by Drs. Reader and Nusstein.

Case scenarios

The following cases represent common scenarios that will be used to emphasize points raised in this review and the preceding articles.

Case #1

This patient reports spontaneous pain in the maxillary posterior quadrant that is increased upon depression of the head. Several teeth present with mechanical allodynia and the pain is not localized to a single tooth. The clinician should consider reproducing the patient's chief complaint by the 'head dip' test (i.e. having the patient sit with both feet on the ground and bend their head forward to their knees). This test shows reasonable selectivity for sinusitis. If there is no history of sinus infection and a negative head dip test, then the patient should be reevaluated on a subsequent appointment or referred to a specialist who can conduct additional tests (e.g. lidocaine gel in the middle meatus has been reported to reduce pain due to sinusitis. There can be no definitive pain, endodontic or restorative treatment plan until the diagnosis is confirmed.

Case #2

The primary diagnosis for this case is irreversible pulpitis with an acute apical periodontitis. The pain treatment plan should consider:

- inferior alveolar nerve block injection followed by intraosseous injection of 3% mepivacaine at a site distal to #30;
- preoperative administration of an NSAID (e.g. ibuprofen 800 mg or flurbiprofen 100 mg) possibly with tramadol (50 mg) or acetaminophen (500 mg) augmentation for the next 2 days;
- occlusal reduction.

The endodontic treatment plan should consider modifying the access preparation by using a sharp #2 round bur to make the access preparation as a single channel (i.e. preparing a small cylinder-shaped access to the pulp). This gives faster access to the pulpal tissue and permits intrapulpal injection with resistance if required (remember that backpressure is the primary predictor of successful intrapulpal injections). Additional endodontic considerations should include a complete pulpectomy if time permits. The restorative treatment plan should consider a restoration that provides cuspal protection (see articles by Reader, Holstein and Rosenberg in this issue of *Endodontic Topics*).

Case #3

The primary dual diagnosis is tooth #20 necrotic pulp with acute apical abscess. The pain treatment plan should consider the following:

- Inferior alveolar nerve block injection. Drs. Reader and Nusstein recommend that teeth with necrotic pulps and periradicular radiolucencies not receive intraosseous injections. If incomplete anesthesia is observed, the clinician may wish to perform an intraosseous injection distal to #19; this should contribute to premolar anesthesia, although probably for a shorter period of time than that observed with intraosseous injection distal to tooth #20.
- Preoperative administration of acetaminophen 1000 mg (NSAIDs are relatively contraindicated in hypertensive patients taking captopril).
- The postoperative analgesic may include continued acetaminophen, alone or with an opioid.
- Occlusal reduction will probably not produce a significant reduction in pain.
- Antibiotics are not required in this case, nor are they likely to reduce postoperative pain.

The endodontic treatment plan should consider a complete pulpectomy procedure. The article by Reader & Nusstein argues against intracanal pulpal injections in necrotic pulp cases such as these due to concerns about injection of bacteria or byproducts into the periapical tissue. The restorative treatment plan should consider a restoration that provides cuspal protection (see articles by Reader, Holstein, and Rosenberg in this issue of *Endodontic Topics*).

Case #4

The pain diagnosis for this patient is a postendodontic flare-up. It is worth repeating vitality testing in these teeth since there have been case reports of postendodontic symptoms due to intact root canal systems that were not found during endodontic treatment. Note that this patient has several risk factors for developing a flare-up (female, necrotic pulp, acute apical peridontitis). The pain treatment plan should consider the fact that the patient did not respond to NSAIDs. This

suggests that the post-treatment pain is most likely due to non-prostaglandin mediators. The pain treatment plan may include:

- reassurance that, although rare, flare-ups do occur, are treatable and do not indicate that the root canal treatment has a poor prognosis;
- effective local anesthesia that in this case includes infiltration or block injection (e.g. PSA block injection with bupivacaine);
- steroid injection (e.g. dexamethasone 4–6 mg). Although there is a lack of controlled clinical trials on steroids for this subgroup of patients, it is likely that immune-mediated hypersensitivity reactions contribute to this condition and could be inhibited by steroid treatment;
- daily contact with the patient until symptoms subside.
- postoperative analgesics (e.g. flurbiprofen 100 mg tid with tramadol 50–100 mg q6 h).

The endodontic treatment plan would be to provide conservative care; the findings given in this scenario do not require removing the gutta percha for retreatment. The restorative treatment plan will include cast cuspal coverage of the tooth (see articles by Reader, Holstein and Rosenberg in this issue of *Endodontic Topics*).

Summary

Hopefully the reader has found this to be an informative review of the integration of clinical and pharmacological strategies for developing effective pain management plans for treating the endodontic pain patient. The importance of a proper diagnosis cannot be over-emphasized. Along with definitive therapy, it should reduce the need for controlled drugs with attendant side-effects. A flexible prescription plan has been presented, with appropriate pharmacological recommendations.

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