

# Clinical strategies for managing endodontic pain

PAUL A. ROSENBERG

## Endodontic emergencies

Although many clinicians focus on drugs for pain management, this is only one aspect of the clinician's armamentarium for managing endodontic pain. Numerous studies indicate that various clinical treatments provide substantial benefit for relief of odontogenic pain. These clinical treatment options include pulpotomy, pulpectomy, incision and drainage, trephination, and occlusal adjustment for specific situations. Anxiety reduction is another important factor in reducing intraoperative and postoperative pain (1). Most often the decision, concerning how best to relieve pain, is made empirically based on a clinician's experience.

Attributing success or failure to a particular clinical technique or procedure is often problematic. It is not unusual for clinicians to review cases seemingly similar in nature that respond differently to the same clinical approach. In contrast, similar cases may respond well to different clinical approaches. What may seem to be a direct cause and effect clinical relationship may actually be a result of a variety of independent and dependent variables that were not recognized or recognized, but not fully appreciated. This article will review procedures used to treat endodontic emergencies in the context of relevant controlled clinical trials and their underlying biological principles.

## Diagnostic considerations

The initial challenge for the clinician is to understand the biological process resulting in pain. Among the diagnostic questions that must be resolved prior to treatment are:

- Is the pain of odontogenic or non-odontogenic origin?
- Is the tooth vital or non-vital?
- Is the pain due primarily to an inflammatory or infectious process?
- Is the pain of pulpal or periradicular origin or both?
- Is there a periodontal component?

Answers to these questions are elicited from a combination of the medical and dental histories as well as highly subjective clinical tests including thermal, electrical and percussion. From the results of these tests, radiographs and the history, the clinician determines which procedure or combination of procedures will most likely relieve the patient's pain.

## Factors affecting treatment

The patient's levels of anxiety and preoperative pain have been shown to influence levels of postoperative pain (1). Preoperative pain and anxiety are predictors of incomplete local anesthesia and postoperative pain. While nerve block injections are successful in 75–90% of patients with clinically normal teeth, local anesthetics are much less effective when administered to patients with inflamed tissue (2–8). Indeed, clinical studies have reported that a single inferior alveolar nerve block injection of local anesthetic (1.8 cc) is ineffective in 30–80% of patients with a diagnosis of irreversible pulpitis (9–12). As shown in Fig. 1, patients with irreversible pulpitis had an 8-fold higher failure rate of local anesthetic injections in comparison to normal control patients (12). Thus, local anesthetic failures can be anticipated in a substantial proportion of patients who seek relief of odontogenic

pain. Similar findings have been reported in children undergoing endodontic treatment, especially those who demonstrate anxiety (7). Understanding the biological basis of an anesthetic problem is an important step towards improved clinical outcomes. The patient who is treated despite inadequate dental anesthesia typically experiences increased anxiety, a reduced pain threshold and a less satisfactory postoperative result.

One of the most common problems relating to inadequate local anesthesia is the confusion concerning dental and soft tissue anesthesia. A positive lip sign (i.e. a 'numb lip') is not an accurate predictor of successful dental anesthesia. Instead, the persistence of the patient's chief complaint should be evaluated to determine the level of local anesthesia. Retesting thermally or with percussion is usually a more effective method of determining the level of dental anesthesia. It is often beneficial to have the patient scheduled for a local anesthetic 20–30 min prior to their regular appointment. A recent comprehensive review of the management of local anesthesia failures provides a summary of the biologic reasons for local anesthetic failures in endodontic pain patients and suggested supplemental strategies (12). It is only after the patient has satisfactory dental anesthesia that the appropriate procedure can be implemented.

## Pulpotomy

A pulpotomy is often performed in cases of acute pain of pulpal origin when there is insufficient time to do

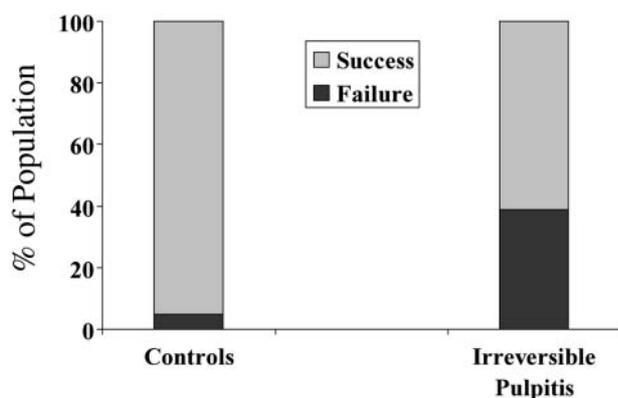


Fig. 1. Evaluation of the frequency of successful anesthesia in 25 patients with a clinically normal pulpal and periradicular diagnosis as compared to 25 patients with a diagnosis of irreversible pulpitis. Patients were injected with 1.8 cc 2% lidocaine with 1:100 000 epinephrine by inferior alveolar nerve block. From: Hargreaves KM & Keiser K. Local anesthetic failures in endodontics. *Endod Topics* 2002; 1: 26–39 (12).

a complete pulpectomy. The goal of the pulpotomy is to remove the coronal pulp tissue in the chamber without penetrating pulpal tissue in the root canal systems. The procedure should be done under rubber dam to prevent further microbiological contamination. After access is achieved, slow speed round burs are used to remove pulp tissue to the level of the canal orifice. Slow speed burs are used to prevent obliteration of the natural funnel at the mouth of a canal that makes initial penetration easier. High speed burs can easily destroy that anatomy. Bleeding is typically managed by a cotton pellet placed firmly against the coronal orifices. Questions often asked about this procedure include: how predictable is it compared to other emergency procedures and how important is a dressing?

The pulpotomy, including sealing of sedative and antibacterial dressings in the pulp chamber has been advocated in emergency situations for many years (13–15). Among the dressings suggested have been phenol, cresatin, and eugenol.

In a clinical study, 73 teeth with acute irreversible pulpitis received emergency pulpotomies (16). After removal of the coronal pulp tissue, a sterile cotton pellet or zinc oxide-eugenol cement was placed against the remaining tissue. The cotton pellet was dry or moistened with camphorated phenol, cresatin, eugenol, or isotonic saline. Accordingly, six different treatment groups were established. All teeth were sealed with zinc oxide-eugenol cement. Symptoms were recorded after the anesthetic effect was gone and at 1, 7 and 30 days after treatment. There was no difference in recorded symptoms among the six treatment groups. Thus, the use of various dressings was not found to contribute to the relief of pain. Instead, the removal of caries, pulpotomy, and sealing of the cavity was found to be a reliable means to relieve pain.

The biological basis for the high level of success associated with the pulpotomy procedure is probably associated with alteration of pulpal hemodynamics and interstitial fluid pressure. Neurogenic mediators play an important role in hemodynamic regulation of the pulp and indirectly control several specialized functions such as dentin formation, production of dentinal fluid and pain mechanisms (17). Two important components in pulpal inflammation are microcirculation and sensory nerve activity (18).

Sensory nerve activity and microcirculation in the

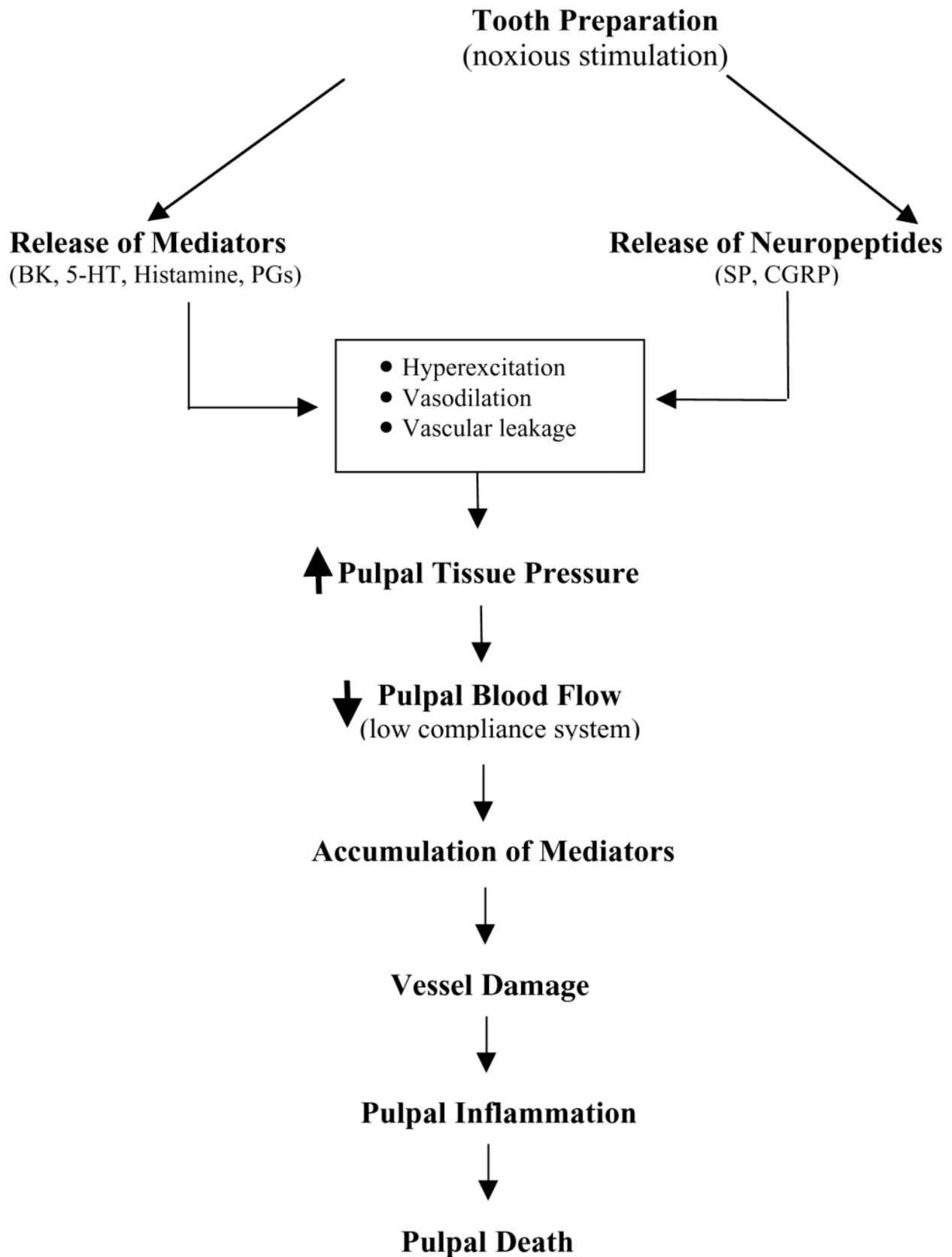


Fig. 2. Hypothetical model illustrating the role of the inflammatory process in the development of pulpal necrosis. Reproduced by permission from Kim S. Neurovascular interactions in the dental pulp in health and inflammation. *J Endod* 1990; 16: 48-53 (18).

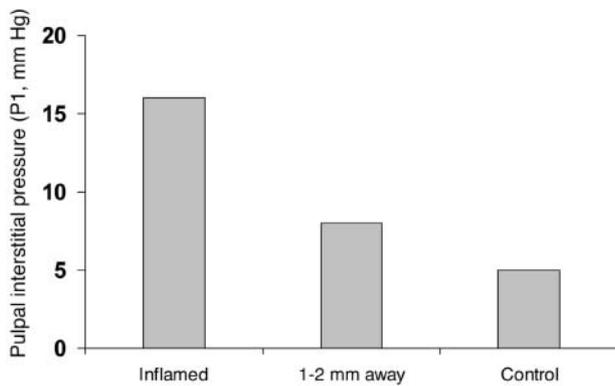


Fig. 3. Pulpal inflammation produces a restricted increase in interstitial pressure. The interstitial hydrostatic fluid pressures ( $P_1$ ) in dental pulp was measured by a server-controlled null micropuncture technique. The  $P_1$  was measured at the site of pulpal inflammation and at another site 1–2 mm away from the site of pulpal inflammation. The control  $P_1$  values were measured in a separate group of teeth. Redrawn from Tønder KJ & Kvinnsland I. Micropuncture measurements of interstitial fluid pressure in normal and inflamed dental pulp in cats. *J Endod* 1983; 9: 105–109 (26).

pulp have been studied by many investigators (19–23). Evidence suggests that neurogenic vasodilation is mediated not only by substance P but also by neurokinin A, neurokinin B and especially calcitonin gene-related peptide. An increase in pulpal blood flow by vasoactive substances, released either from sensory nerve endings or from other cellular components, may have profound effects on both circulatory and neural behavior. A hypothetical mechanism of pulpal necrosis has been suggested and is presented in Fig. 2.

Other studies have provided experimental support for this model. For example, there is a regional increase in pulpal interstitial pressure in response to inflammation (24, 25). It was demonstrated in cat's dental pulp that the interstitial pressure was twice as great at the site of pulpal inflammation as compared with a site only 1–3 mm away (26) (Fig. 3).

The results of this study are important because they demonstrate that the pulpal interstitial pressure response to pulpal inflammation is restricted to the site of injury and is not generalized throughout the pulp. The concept of a generalized increase in interstitial pressure during pulpal inflammation leading to a generalized collapse of venules and cessation of blood flow (the so-called 'pulpal strangulation' theory) is not supported by the results of the study. It seems that circulatory responses to pulpal inflammation are localized reactions to release of inflammatory mediators or other factors (27–30).

Regulation of pulpal blood flow during periods of health and inflammation seems to be dependent on the receptors expressed on endothelium and smooth muscle, as well as local tissue concentrations of inflammatory mediators and other substances (e.g. nor-epinephrine, CGRP, substance P, etc.) (Fig. 4). While the pulpal vascular system has features similar to those seen in other tissues, a significant difference is the local environment (i.e. the dental pulp exists in a low compliance environment). The pulpal vasculature responds as other tissues do to vasoconstrictors but it responds differently to vasodilators. The result is a

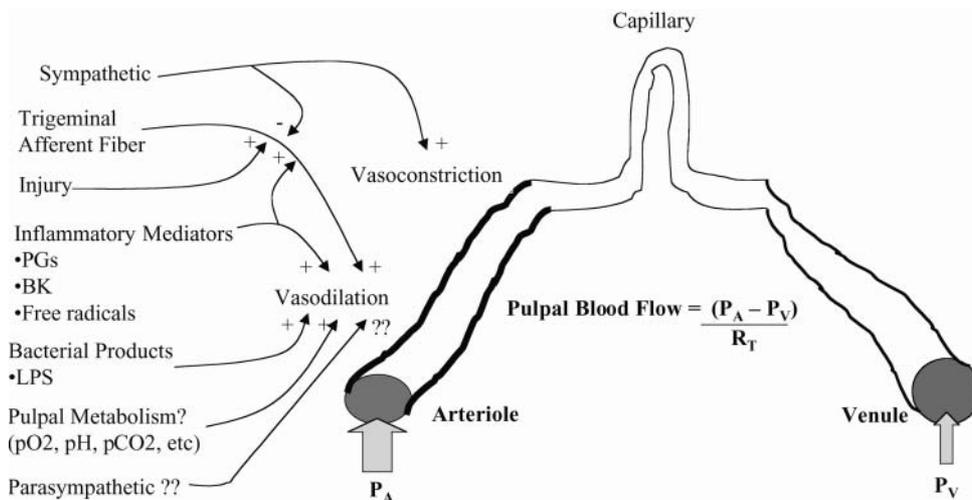


Fig. 4. Schematic illustration of the major mechanisms regulating pulpal blood flow. Reproduced by permission from Suda & Ikewa in *Seltzer and Bender's dental pulp*. Chicago: Quintessence, 2002: 133.

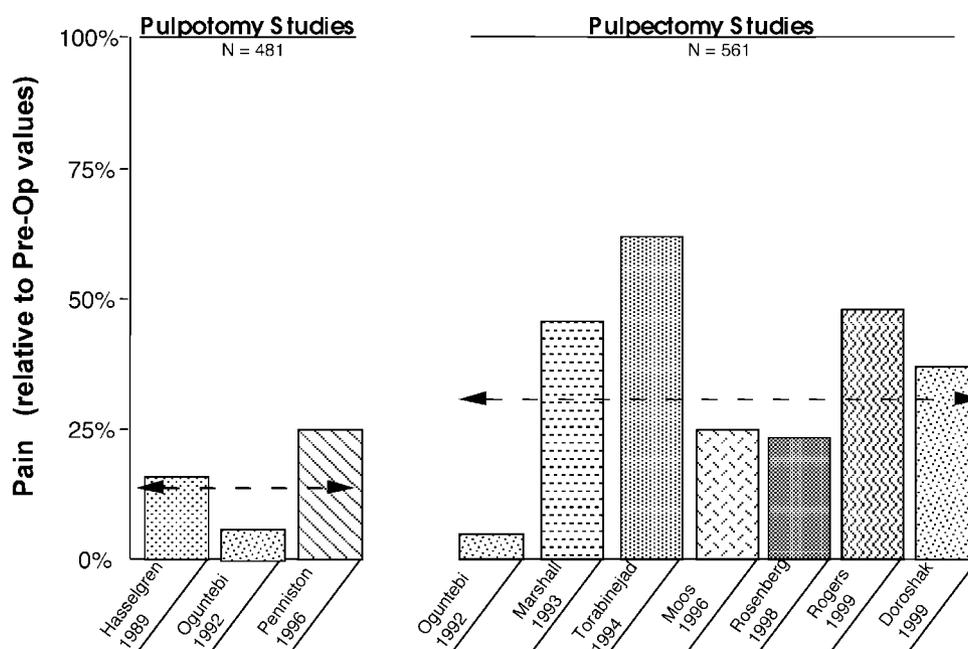


Fig. 5. Effects of pulpotomy or pulpectomy on endodontic-related pain. Pre-operative pain values are normalized to 100%. The two horizontal bars for pulpotomy and pulpectomy groups represent the sample size weighted mean reduction in pain. From: Hargreaves & Baumgartner, *Endodontic Therapeutics*. In: Walton R, Torabinejad M, eds. *Principles and practice of endodontics*, Ch 30, 3rd edn. Philadelphia: Saunders, 2002: 533–544.

sharp transient increase in pulpal blood flow followed by a sustained decrease. It has been theorized that this biphasic flow response to vasodilating agents, many of which are inflammatory mediators, may play an important role in the pathophysiology of the pulp during inflammation. Vasodilation in other tissues is a defensive reaction during inflammation, however, because of its secondary vasoconstriction the result in teeth may include pulpal necrosis (31).

Catecholamines (dopamine, epinephrine and nor-epinephrine) may also play an important role in the control of intrapulpal pressure during inflammation since studies have demonstrated that their levels are significantly greater in inflamed dental pulp when compared to uninflamed pulps (32, 33).

The success of a pulpotomy in relieving pain, particularly in the vital case, would seem to be due to a venting of the chamber with a concomitant reduction in local tissue pressure, inflammatory mediator concentrations and the severing of the terminal endings of nociceptive sensory neurons. Clinicians frequently note the dramatic effect of opening a chamber and observing the rapid relief that often follows. It seems reasonable to assume that these factors constitute the biological basis for its highly predictable effect of re-

ducing pain in patients with irreversible pulpitis. Furthermore, by avoiding the canal system, the clinician avoids performing a partial pulpectomy – which might traumatize already inflamed tissue. Partial pulpectomy may result in profuse hemorrhage due to the rupture of wide diameter vessels in the central part of the pulp. Less hemorrhage often results when the extirpation of the pulp is made to the apex of the tooth. A clinical study found a higher incidence of postoperative pain in cases where partial pulpectomy was performed (34). Thus, in treating patients with pain due to irreversible pulpitis, a pulpotomy procedure is preferable when time does not permit a complete pulpectomy (Fig. 5). A partial pulpectomy should be avoided in these cases.

## Pulpectomy

Since it is impossible for the clinician to precisely determine the apical extent of pulpal pathosis, a pulpectomy offers the advantage of complete removal of the pulp. However, it is possible that the pulpectomy itself can be the cause of increased post operative pain. This may happen when a pulpectomy is done without the benefit of an accurate canal length measure-

ment. The subsequent risk of leaving shredded, inflamed tissue in the canal or damaged periradicular tissue is adequate reason for the clinician to take the time to establish a measurement control (35).

Pulpectomy is the course of treatment often used in patients who present with symptoms of irreversible pulpitis, or pulp necrosis with or without swelling.

Questions that are associated with the pulpectomy procedure include: Is an intracanal dressing necessary after the procedure and if so which one? Despite an extensive body of literature focused on these questions, the answers remain controversial. At this time, the most widely used dressing is probably calcium hydroxide, which is an effective antibacterial agent but has not been shown to have any direct analgesic effect.

There is also a controversy concerning the use of an antibiotic to prevent postoperative pain following pulpectomy (for additional information, see the article by A. Fouad in this issue of *Endodontic Topics*). In a double blind, prospective study, administration of penicillin prophylactically was the same as a placebo treatment for reducing postoperative pain following root canal preparation (36). However, other studies have had results that differ (1, 37). In a prospective, double blind, clinical study the root canals of 588 consecutive endodontic patients with varying levels of pain were completely instrumented. The

study was conducted at 10 endodontic practices and four endodontic graduate programs (1). The participants were sequentially assigned to groups and were directed to use one of nine analgesics or antibiotics or a placebo. The amount of instrumentation during the emergency appointment and the type of medication(s) taken following the appointment were not standardized. Among all of the parameters studied, preoperative pain, apprehension and types of medication were found to be significant in determining post instrumentation pain. Other investigators have also found a relationship between the presence of preoperative pain and the incidence of postoperative pain (38–40). An association was also found between the intensity of pre- and postoperative pain. As the intensity of preoperative pain increased, the chances for more severe postoperative pain increased ( $P < 0.0001$ ). In addition, an association between the presence of apprehension before any treatment and postoperative pain was noted ( $P < 0.05$  37).

Despite conflicting results from clinical studies concerning the use of antibiotics to prevent postoperative pain, it is clear that such use opens the patient to risks that include the side-effects of nausea and diarrhea as well as the more significant risks of anaphalaxis and sensitization to antibiotics. A more predictable strategy for the prevention of root operative pain following pulpectomy would include the use of NSAID's preoperatively or immediately postoperatively (for further information, see the article by K. Keiser in this issue of *Endodontic Topics*). While there is not an extensive body of literature concerning the use of NSAID's prophylactically in endodontics, there are

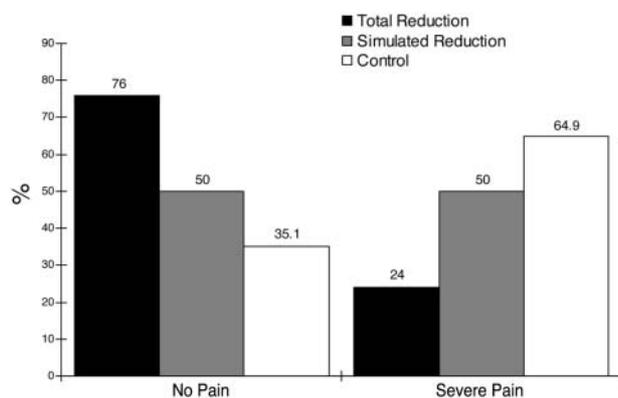


Fig. 6. Effect of occlusal reduction ('total reduction'), simulated reduction or no treatment on percentage of the patients who report either no postendodontic pain vs.% of patients who report moderate-to-severe postendodontic pain.  $N=117$  patients. The numbers above each bar represents the actual percentage values. From: Rosenberg et al. The effect of occlusal reduction on pain after endodontic instrumentation. *J Endod* 1998; 24: 492–496 (42). Data show that a definite relationship exists between occlusal reduction and a positive or negative pain response. ( $p > 0.01$ ).

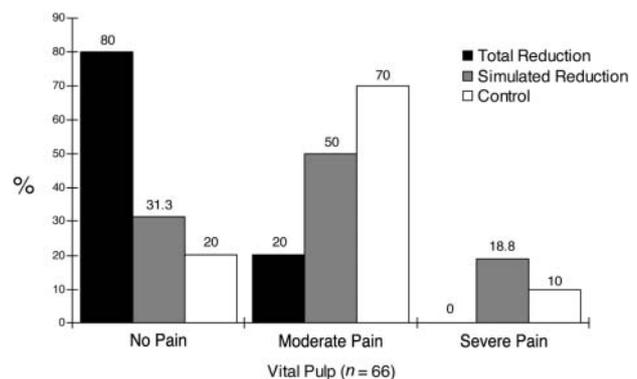


Fig. 7. Effect of occlusal reduction for the subset of patients from Fig. 6 who have vital pulps ( $N=66$ ). From: Rosenberg et al. The effect of occlusal reduction on pain after endodontic instrumentation. *J Endod* 1998; 24: 492–496 (42).

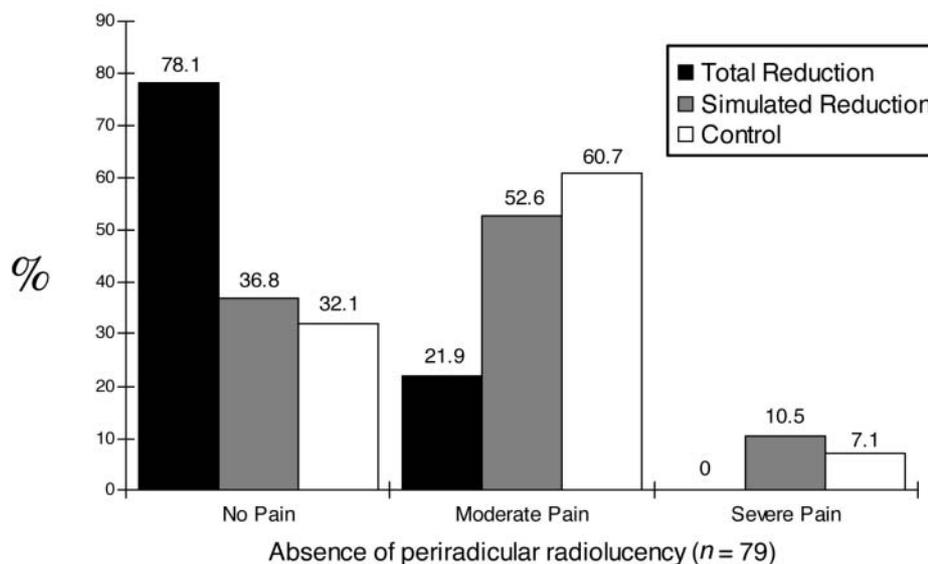


Fig. 8. Effect of occlusal reduction for the subset of patients from Fig. 6 who have no periradicular radiolucencies ( $N = 79$ ). From: Rosenberg et al. The effect of occlusal reduction on pain after endodontic instrumentation. *J Endod* 1998; 24: 492–496 (42).

generalizations that can be drawn from clinical research using other models (41).

As with any drug, the clinician must be aware of limitations and drug interactions as well as the patient's medical history before prescribing it. Numerous studies have found that the most consistent factor that predicts post endodontic pain is the presence of preoperative hyperalgesia (defined as preoperative pain or percussion sensitivity) (1, 38–41, 42).

Other factors were more variable in their predictive value of postoperative pain (38). For example, in a retrospective study, 1000 patients who had received non-surgical endodontic treatment and experienced no flare-ups (i.e. unscheduled patient return visits) were compared with the records of 1000 patients who experienced flare-ups after the cleansing and shaping of their necrotic root canals. The results showed that factors such as presence of preoperative pain, tooth type, sex, age, history of allergy and re-treatment were significantly predictive for the incidence of flare-up; intracanal medicaments, systemic disease, and establishment of patency of the apical foramen had no significant relationship to the incidence of flare-ups (38).

Following the pulpectomy it is best to close teeth in order to prevent contamination from the oral cav-

ity (43). Teeth left open to the environment are often involved in exacerbations during treatment (44).

If there is a flow of exudate from the canal following instrumentation and irrigation, it is best to wait to close the tooth until the flow stops. Infrequently, the flow will continue and, in those instances, a cotton pledget or porous material can be used as a barrier until the patient returns, preferably the next day. The goal is to close the tooth as soon as possible in order to prevent further bacterial penetration.

## Trephination

Trephination is the surgical perforation of the alveolar cortical plate over the root end of a tooth to release accumulated tissue exudate that is causing pain (45). The procedure has been recommended for patients with severe recalcitrant periradicular pain of endodontic origin. Those who are advocates of trephination do not agree in their choice of flap (or if a flap is necessary) as well as the instrument to be used to perforate through the cancellous bone toward the periradicular lesion. One report has suggested a small, vertical incision adjacent to the tooth in question. The mucosa is retracted with a tissue retractor, and a number six round bur is used to penetrate the cortical

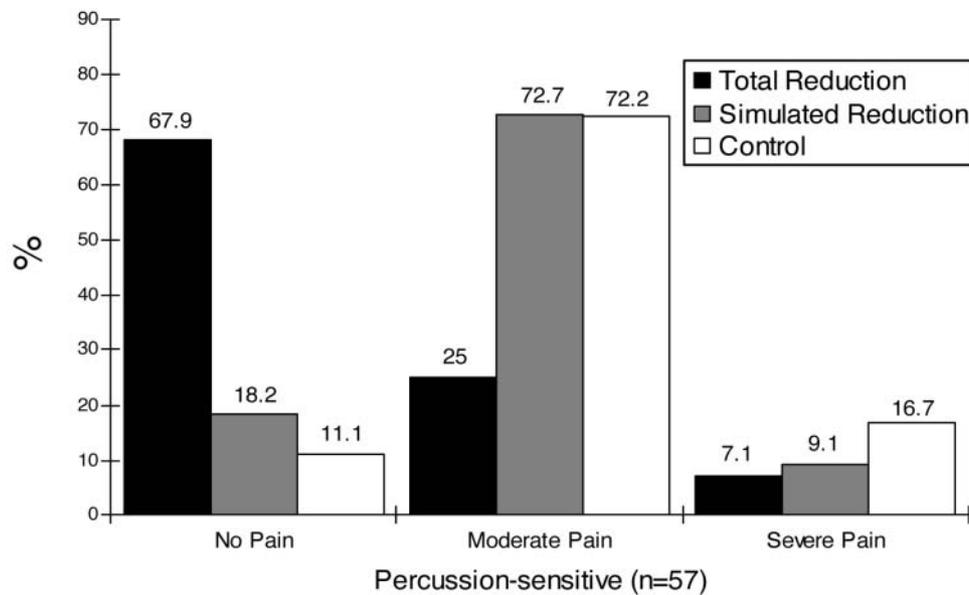


Fig. 9. Effect of occlusal reduction for the subset of patients from Fig. 6 who are percussion sensitive ( $N=57$ ). From: Rosenberg et al. The effect of occlusal reduction on pain after endodontic instrumentation. *J Endod* 1998; 24: 492–496 (42).

plate. An endodontic file has also been suggested to create a path through the cancellous bone toward the periradicular lesion, avoiding contact with the root structure or adjacent teeth (46). An engine-driven perforator has also been suggested as a means of entering the medullary bone without the need for an incision (47). Some clinicians disagree with that suggestion. The absence of a flap limits the visibility of the surgical site and increases the possibility of damaging the root of the tooth or adjacent teeth. This is especially likely in areas where roots are close approximation. It is presumed that if apical trephination is successful, its success is based on the establishment of drainage, relief of pressure and the removal of inflammatory mediators from the periradicular tissues. Among the problems associated with the procedure is the lack of accurate information about the precise location of the problem, especially in a multiradical tooth. Accessibility in the absence of a surgical flap makes the clinician vulnerable to operative errors. Biologically, the trephination procedure does not take into account the variety of conditions that can result in pain.

Trephination has become less frequent in its use over time. In 1977, a survey indicated that 16% of Diplomates of the American Board of Endodontics would perform trephination for necrotic teeth (14). However, in 1990 a similar survey showed that only 8% of Diplomates were using trephination (48).

Some clinicians have reported anecdotally that they treat intractable severe pain of endodontic origin, in the absence of acute infection, by obturating the canal(s) and then doing an immediate apicoectomy. The advantage of this approach is that following the obturation and apical surgery, no further procedure is necessary. Furthermore, the clinician has the advantage of good surgical visibility and access by using a conventional surgical flap.

A clinical study compared pulpectomy alone or pulpectomy with trephination for the reduction of post-treatment pain in patients presenting with acute periradicular pain of pulpal origin (48). Seventeen patients with pretreatment pain were studied. Eleven received a pulpectomy to the radiographically determined working length. Six patients received a pulpectomy and trephination. The trephination group reported significantly more postoperative pain intensity and unpleasantness and less pain relief at 4 h compared with the control group. Pulpectomy alone provided significantly better postoperative pain relief at 4 h compared with pulpectomy with trephination. At no time interval in the study (from 4 to 96 h) did the trephination group have less pain than the group without trephination. In two previous studies, however, trephination was shown to significantly decrease postoperative pain incidence when performed prophylactically along with root canal therapy (49, 50). One study was limited to asymptomatic, necrotic

anterior and premolar teeth. It was reported that no pain occurred in teeth that were prophylactically trephinated while 25% of those not trephinated had moderate to severe pain (50).

More recently, investigators have questioned the effect of trephination on postoperative pain and swelling in symptomatic necrotic teeth. Two studies examined the effectiveness of trephination in 50 emergency patients with symptomatic necrotic teeth. After endodontic treatment, patients randomly received either a trephination or mock trephination procedure. In neither study did the trephination procedure significantly reduce pain, percussion pain or swelling. One study did show a reduction in the use of acetaminophen with codeine overall for 7 days. Both research groups were unable to recommend trephination in symptomatic necrotic teeth with radiolucencies (51, 52). Thus, there are no consistent findings of benefit from trephination procedures.

## Incision and drainage

Pulpal necrosis may result in a periradicular abscess with swelling. The swelling may be seen at an emergency visit, as part of an interappointment flare-up,

or even as a post obturation complication. Swellings may be described as localized or diffuse and as fluctuant or hard. They may also extend laterally or vertically well beyond the involved tooth and can involve fascial spaces.

A serious diffuse swelling is characterized by its spread through adjacent soft tissues, dissecting tissue spaces along fascial planes. Such a swelling is called a cellulitis and because of the inability of the patient's defense mechanisms to localize the lesion, it has the potential to be dangerous. Patients with a cellulitis may actually have little or no intraoral swelling.

A well localized swelling does not have the same potential for serious complications that the cellulitis represents. A cellulitis must be most aggressively treated and monitored to reduce the possibility of serious sequelae. Cellulitis patients who do not seem to be responding to treatment, as evidenced by elevated body temperature, increased swelling, pain and malaise, are candidates for hospitalization. Particularly at risk are those patients who have compromising systemic conditions or are of advanced age and may not be able to care for themselves properly.

The goal of emergency treatment for patients with swelling is to achieve drainage (53). The object of

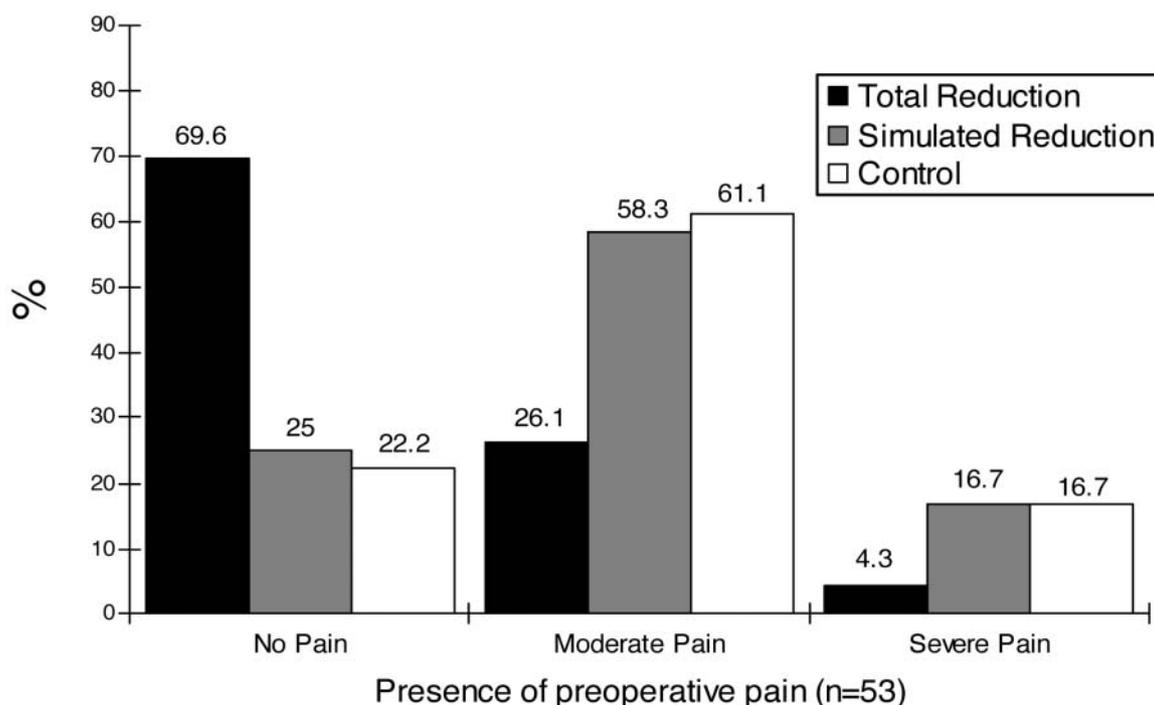


Fig. 10. Effect of occlusal reduction for the subset of patients from Fig. 6 who had preoperative pain (N = 53). From: Rosenberg et al. The effect of occlusal reduction on pain after endodontic instrumentation. *J Endod* 1998; 24: 492-496 (42).

drainage is to evacuate pus from the tissue spaces. In endodontic cases, drainage is best achieved through a combination of canal instrumentation and when there is a fluctuant swelling – incision and drainage. Antibiotics can be used to supplement the clinical procedures primarily in patients where there is poor drainage and large swellings. In patients with well-localized swellings where the canal can be instrumented and good drainage established with an incision and drainage, antibiotics are of only supplemental value.

Even in cases where an incision and drainage is to be implemented, the canal should be accessed, instrumented, irrigated, medicated and closed as soon as active drainage stops. In the rare case when drainage continues throughout the visit, the canal should be left open with a pledget of cotton or other porous material to permit continued flow while preventing the impaction of food and other debris. It is essential that such patients return for further treatment as soon as possible, preferably the next day. The sooner the canal can be instrumented and closed, the more rapid resolution of periradicular pathosis can be expected.

Conversely, the longer the canal is left open, the more healing is delayed as intra-canal bacterial penetration continues. There does not appear to be a strong biological basis for performing an incision and drainage procedure and yet allowing the canal to remain open. While incision and drainage usually provides symptomatic relief by evacuating pus and reducing pressure on distended tissues, healing can not take place until bacteria in the canal are eliminated and it is closed to the environment. Systemic antibiotics can be expected to be more effective once the canal has been debrided, medicated and closed. The use of antibiotics to treat swellings of endodontic origin is not recommended without concomitant canal instrumentation and when appropriate, incision and drainage (54).

In cases with well-localized fluctuant swellings after canal instrumentation and incision and drainage, the use of an antibiotic is usually unnecessary. A well-localized fluctuant soft tissue swelling is a candidate for incision and drainage. Profound anesthesia may be problematic at the surgical site but peripheral infiltration is helpful and usually permits tissue manipulation with minimum discomfort. The incision should be made at the site of greatest fluctuance and the clinician should then dissect gently to the bone overlying the root of the tooth causing the problem. The wound should be kept clean with hot saltwater rinses.

Intraoral heat application has been recommended and is presumed to increase dilation of small vessels, intensifying host defenses through increased vascular flow (53, 54).

There is disagreement among clinicians concerning the need for suturing a drain into the incision to maintain active drainage. While some clinicians routinely suture a drain in place, others depend on saline rinses to keep the incision open. Another controversy concerns the incision of hard swellings as well as indurated lesions. Some clinicians believe that the incision of a hard swelling represents little more than ‘bleeding a patient’, others believe that it can relieve pain from increasing tissue distention even if only hemorrhagic fluid is obtained. Unfortunately, there are few clinical trials evaluating these treatment options.

## **Occlusal reduction**

The value of reducing occlusion to prevent pain after endodontic instrumentation had been a source of controversy. In a review of the treatment of endodontic emergencies it had been recommended that ‘If a tooth responsible for an acute abscess is extremely painful on biting, occlusal contact should be reduced so that the tooth is reasonably comfortable in normal occlusion’ (55). Similarly a decade later it was recommended that ‘occlusal reduction will reduce symptoms regardless of cause’ (56). Other sources have continued to write anecdotally about theoretical benefits of occlusal reduction (57, 58).

A poll of Diplomats of the American Board of Endodontics in 1977 indicated that 80% of respondents adjusted occlusal contacts of teeth that had preoperative pain in both vital and non-vital cases (14). An update of that poll, over a decade later, determined that occlusal adjustment was performed in a higher percentage of cases with apical periodontitis than in those with no apical involvement (59). The condition in which most respondents adjusted the occlusion was when apical periodontitis existed without swelling. In vital cases without apical involvement, there was a decrease in the number of respondents who would adjust the occlusion (59).

However, some studies raised doubts about the actual value of occlusal reduction. One group studied 49 teeth by randomly assigning them to either an occlusal relief or mock-occlusal relief group after canal preparation. They found that preoperative pain levels in both

groups related to postoperative pain, but did not determine whether occlusal relief would reduce postoperative pain in patients with any other specific preoperative symptoms. It was concluded that 'the theory may be invalid that the prophylactic removal of occlusal contacts is a pain-preventive measure' (60).

In a similar study, 58 teeth were evaluated by removing occlusal contacts from alternate patients. In that study, subjects were asked to bite on a cotton tipped applicator twice a day for 6 days and to record whether or not this caused any discomfort. They concluded that 'no direct relationship exists between occlusal contact and patient comfort following the preparatory phase of endodontic therapy' (61).

The discrepancy between the clinical impressions of many endodontists and the findings of some investigators led to the following questions:

- Do all endodontic cases respond similarly to occlusal reduction?
- Can a reliable clinical profile be developed of patients most likely to benefit from occlusal reduction?

It was hypothesized that there may be certain preoperative conditions that are indicators of the need for occlusal reduction in the endodontic patient. Conditions evaluated included the presence or absence of pulp vitality, preoperative pain, percussion sensitivity, a periradicular radiolucency, a stoma, swelling and a history of bruxism.

A clinical study of 117 patients with posterior teeth in occlusion requiring endodontic treatment was initiated (42). The purpose of the study was to evaluate the effect of occlusal reduction on pain after endodontic instrumentation and to develop a statistically valid profile of patients most likely to benefit from occlusal reduction. Teeth excluded from the study were those lacking occlusal contacts or those with occlusal restorations that were to be retained (e.g. well-fitting cast restorations) and would not receive occlusal adjustment. Also excluded from the study were teeth with greater than class I mobility, pocket depths >5 mm, endodontic retreatments or patients who were taking antibiotics and/or pain-altering medications.

A series of preoperative conditions were recorded for each tooth included in the study:

- Pulp vitality
- Percussion sensitivity
- Periradicular radiolucency

- Preoperative pain
- Swelling
- Stoma
- History of bruxism

Teeth were randomly assigned to 1 of 3 occlusal treatment groups:

- *Total occlusal reduction*—occlusal contacts were reduced 0.5–1.0 mm in centric occlusion and all excursions.
- *Simulated occlusal reduction*—a non-functional cusp was reduced. Occlusion was left intact.
- *No treatment control group*—All occlusal surfaces were left untouched. Occlusion was verified after endodontic access and at the time of dismissal.

Each patient was given a questionnaire adapted from a previous study in which they were asked to select 1 of 3 specific statements that best described their pain experience during the 48 h immediately after canal instrumentation and occlusal adjustment.

The results were analyzed to determine whether or not the postoperative pain report was dependent on the experimental treatments (occlusal treatment groups). Groups of teeth exhibiting specific preoperative conditions were also analyzed. A statistically valid profile of patients most likely to benefit from occlusal reduction after endodontic instrumentation was developed (Figs 5, 6, 7, 8, 9, 10). The results indicate that occlusal reduction should aid in the reduction of post instrumentation pain in patients whose teeth exhibit preoperative pain, pulp vitality, percussion sensitivity and/or the absence of a periradicular radiolucency. While the presence of all four conditions is the strongest predictor, the presence of any one or more of the conditions it enough to indicate the need for occlusal reduction (Fig. 11).

Occlusal reduction when performed in appropriate cases is a highly predictable simple strategy for the prevention of postoperative pain and relief of pain due to endodontic emergencies.

There is a biologic rationale for the relief of pain provided by the previous techniques. Mechanical allodynia (i.e. sensitivity to percussion or biting forces) is due to tissue levels of factors that stimulate peripheral terminals of nociceptors. Occlusal adjustment reduces mechanical stimulation of sensitized nociceptors. Pulpotomy, pulpectomy, occlusal reduction and incision and drainage, when indicated, provide the clinician with highly predictable pain reduction strategies in endodontic emergencies.

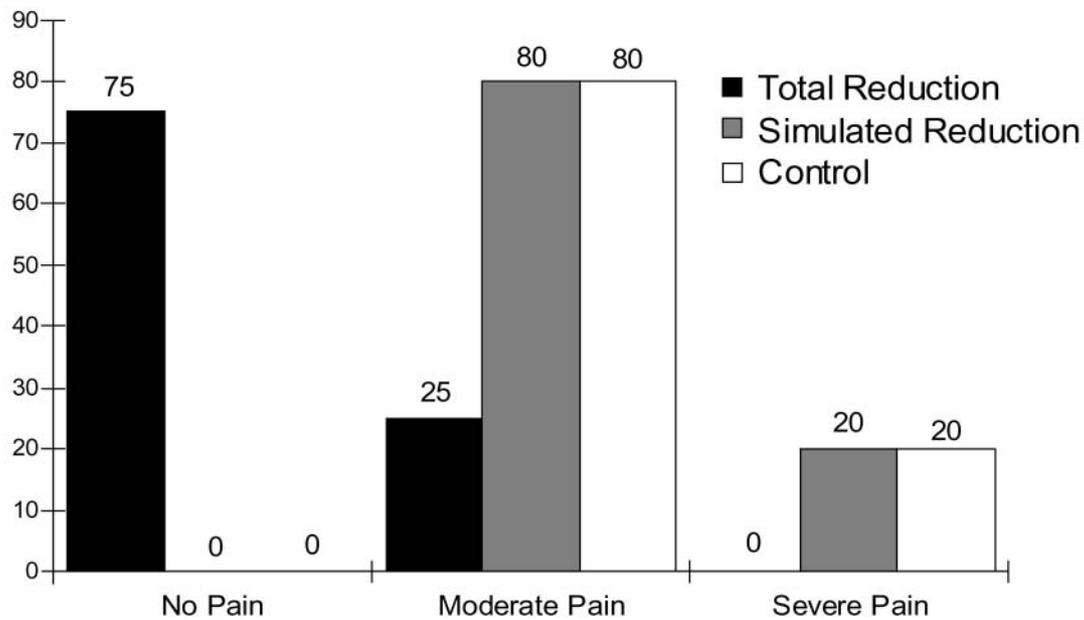


Fig. 11. Presence of all four significant preoperative conditions ( $n = 27$ ): vital pulp, no periradicular radiolucency, percussion sensitivity, and preoperative pain. From: Rosenberg et al. The effect of occlusal reduction on pain after endodontic instrumentation. *J Endod* 1998; 24: 492–496 (42).

## Anxiety and pain

Investigators have suggested a close relationship between pain and anxiety: the greater the anxiety, the more likely we are to interpret the sensation as pain. In a clinical study of children, it was found that anxiety is the strongest predictor of poor intraoperative pain control. Similarly, during heightened anxiety, the pain threshold is lowered for patients (62, 63). Highly fearful patients are more sensitive to pain in general and those who are dentally anxious are more sensitive to dental pain specifically. High levels of stress, anxiety or pessimism in preoperative patients predict poor outcomes in measures that range from speed of wound healing to duration of hospital stay (64). More than 200 studies indicate that pre-emptive, cognitive and behavioral interventions in unselected groups of patients decrease anxiety before and after surgery, reduce postoperative pain intensity and intake of analgesic drugs, improve treatment compliance, cardiovascular and respiratory indices, and accelerated recovery (65).

In an evaluation of pain thresholds it was determined that highly anxious patients had lower thresholds of pain when a tooth was electrically stimulated (66). In addition, evidence suggests that highly anxious patients tend toward lower general pain toler-

ance to a greater degree than normal controls (67, 68). It has also been shown that more highly anxious patients report greater pain during dental procedures than normal controls (1). Clinical dental studies have indicated a strong relationship between endodontic postoperative pain and anxiety as well as intraoperative pain and anxiety (7, 38).

Although pharmacological strategies to reduce anxiety are available, other non-pharmacologic approaches have been extensively evaluated (56). Behavioral techniques can be used to prevent excessively high levels of tension or anxiety from developing in response to potential dental stressors (69).

## Office tone

The tone of the office is established initially by the greeting of the receptionist and the décor. It is the first step in establishing a calm, welcoming environment. A receptionist who is harried, rushed and impersonal is sending the wrong message. The initial impression made on an anxious patient is an important ingredient in reducing or elevating stress. The level of stress translates directly to the patient's pain threshold.

## Information

Providing information about the procedure is an important step in preparing patients for endodontic treatments. Information about profound dental anesthesia and preventive pain strategies is an important anxiety reduction technique. Perhaps most importantly, the dentist should assure the patient that pain prevention is a primary concern.

Successful findings have come from studies in which combined procedural information and sensation information were provided to patients (procedural information alone has not been consistently effective). For example, significant changes in anxiety were found when cardiac catheterization patients were provided with a combination of sensory (what physical sensations might be expected) plus procedural (what steps were involved in the procedure) information (70).

In dental research with children aged 3–5, positive results were found when preparatory sensory information was combined with procedural information prior to restorations (71). In a study on adults, it was found that subjects given a running commentary on procedures and associated sensations rated themselves as less anxious and experiencing less pain than a normal control group (72). Collectively, these studies suggest that information about sensations when added to a description of procedures, appears to have a significant impact in reducing patient anxiety.

## Modeling

Modeling is a variant of information provision. Many studies have shown that observing a peer (either live or on a video) can be successful in reducing anxiety about dental treatment, especially for inexperienced children (73, 75). Allowing an anxious patient to observe, from a doorway, can be effective in building confidence and reducing anxiety.

## Distraction

Distraction has proved effective in dental situations as a means of preoccupation and anxiety reduction. Distraction is also credited with the ability to enable subjects to better cope with pain. Music with head-

phones and the patient controlling the selection as well as video games have been shown to be successful methods of reducing anxiety (62).

## Hypnosis

Hypnosis has a long history in clinical medicine with impressive documentation that indicates its effectiveness in alleviating pain (75). In an impressive dental study, the successful use of hypnosis was demonstrated in 99 of 100 patients involving extractions (76). Mediation-hypnosis has also been reported as the sole anesthesia in an endodontic case (77).

Root canal therapy is one of the most anxiety-inducing dental procedures (78). Research has shown that the amount of pain expected and experienced by dental patients is directly related to their anxiety (79). This section of our review has reviewed some of the possible approaches to anxiety reduction as a means of reducing intraoperative and postoperative pain. The particular approach used may be less important than the commitment of the clinician to find a way to integrate non-pharmacologic anxiety reduction strategies into their practice. Pharmacologic strategies can be used as a supplement when necessary.

## References

1. Torabinejad M, Cymerman JJ, Frankson M, Lemon RR, Maggio JD, Schilder H. Effectiveness of various medications on postoperative pain following complete instrumentation. *J Endod* 1994; **20**: 345–354.
2. Guglielmo A, Reader A, Nist R, Beck M, Weaver J. Anesthetic efficacy and heart rate effects of the supplemental intraosseous injection of 2% mepicaine with 1: 20,000 levonordefrin. *Oral Surg Oral Med Oral Path Oral Radiol Endod* 1999; **87**: 284–293.
3. Hargreaves KM. Neurochemical factors in injury and inflammation in orofacial tissues. In: Lavigne G, Lund J, Sessle B, Dubner R, eds. *Orofacial pain: basic sciences to clinical management*. Chicago: Quintessence Publications, 2001.
4. Jastak J, Yagiela J, Donaldson D. *Anesthesia of the oral cavity*. Philadelphia: Saunders, 1995: 1–339.
5. Malamed S. *Handbook of local anesthesia*, 3rd edn. St. Louis: Mosby, 1990: 1–332.
6. Miles M. The missed inferior alveolar block: a new look at an old problem. *Anesth Prog* 1984; **31**: 87–90.
7. Nakai Y, Milgram P, Mancl L, Coldwell SE, Domoto PK, Ramsay DS. Effectiveness of local anesthesia in pediatric dental practice. *J Am Dent Assoc* 2000; **131**: 1699–1705.

8. Walton R, Torabinejad M. Managing local anesthesia problems in the endodontic patient. *J Am Dent Assoc* 1992; **123**: 97–102.
9. Cohen HP, Cha BY, Spangberg LS. Endodontic anesthesia in mandibular molars: a clinical study. *J Endod* 1993; **19**: 370–373.
10. Nusstein J, Reader A, Nist R, Beck M, Meyers WJ. Anesthetic efficacy of the supplemental intraosseous injection of 2% lidocaine with 1: 100,000 epinephrine in irreversible pulpitis. *J Endod* 1998; **24**: 487–491.
11. Resiman D, Reader A, Nist R, Beck M, Weaver J. Anesthesia efficacy of the supplemental intraosseous injection of 3% mepivacaine in irreversible pulpitis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997; **84**: 676–682.
12. Hargreaves KM, Keiser K. Local anesthetic failures in endodontics. *Endod Topics* 2002; **1**: 26–39.
13. Dorn SO, Moodnik RM, Feldman MJ, Borden BG. Treatment of the endodontic emergency: a report based on a questionnaire. *J Endod* 1977; **3**: 94–100.
14. Weine FS. *Endodontic therapy*. St. Louis: CV Mosby, 1982.
15. Grossman LI, Oliet S, Del Rio CE. *Endodontic practice*. Philadelphia: Lea & Febiger, 1988.
16. Hasselgren G, Reit C. Emergency pulpotomy: pain relieving effect with and without the use of sedative dressings. *J Endod* 1989; **15**: 254–256.
17. Olgart LM, Edwall B, Gazelius B. Neurogenic mediators in control of blood flow. *J Endod* 1989; **15**: 409–412.
18. Kim S. Neurovascular interactions in the dental pulp in health and inflammation. *J Endod* 1990; **16**: 48–53.
19. Edwall L, Scott D Jr. Influence of changes in microcirculation on the excitability of the sensory unit in the tooth of the cat. *Acta Physiol Scand* 1971; **82**: 55–56.
20. Narhi MVO, Hirvonen TJ, Hakumaki MOK. Responses of intradental nerve fibers to stimulation of dentine and pulp. *Acta Physiol Scand* 1982; **115**: 173–178.
21. Tonder KH, Naess G. Nervous control of blood flow in the dental pulp in dogs. *Acta Physiol Scand* 1978; **104**: 13–23.
22. Kim S, Edwall L, Trowbridge H, Chien S. Effects of local anesthetics on pulpal blood flow in dogs. *J Dent Res* 1984; **63**: 650–652.
23. Kim S. Microcirculation of the dental pulp in health and disease. *J Endod* 1985; **11**: 465–471.
24. Stenvik A, Iverson J, Mjor IA. Tissue pressure and histology of normal and inflamed tooth pulps in macaque monkeys. *Arch Oral Biol* 1972 1973; **17**: 1501–1511.
25. Van Hassel HJ. Physiology of the human dental pulp. *Oral Surg Oral Med Oral Pathol Oral Radiol* 1971; **31**: 126–134.
26. Tønder KJ, Kvinnsland I. Micropuncture measurements of interstitial fluid pressure in normal and inflamed dental pulp in cats. *J Endod* 1983; **9**: 105–109.
27. Heyeraas KJ, Bergreen E. Interstitial fluid pressure in normal and inflamed pulp. *Crit Rev Oral Med* 1999; **10**: 328–336.
28. Heyeraas KJ. Pulpal hemodynamics and interstitial fluid pressure: Balance of transmicrovascular fluid transport. *J Endod* 1989; **15**: 468–472.
29. Heyeraas KJ. Pulpal microvascular and tissue pressure. *J Dent Res* 1985; **64** (Spec Issue): 585–589.
30. Kim S, Dorsher-Kim J. Hemodynamic regulation of the dental pulp. In: Inoki R, Kudo T, Olgart L, eds. *Dynamic aspects of dental pulp*. London: Chapman & Hall, 1990: 167–188.
31. Kim S, Dorsher-Kim J. Hemodynamic regulation of the dental pulp in a low compliance environment. *J Endod* 1989; **15**: 404–408.
32. Schachman MA, Rosenberg PA, Linke HAB. Quantitation of catecholamines in uninflamed human dental pulpal tissues by high performance liquid chromatography. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1995; **80**: 83–85.
33. Nup C, Rosenberg P, Linke HAB, Tordik P. Quantitation of catecholamines in inflamed human pulp by high performance liquid chromatography. *J Endod* 2001; **27**: 73–75.
34. Qguntebi BR, De Schepper EJ, Taylor TS, White CL, Pink FE. Post operative pain incidence related to the type of emergency treatment of symptomatic pulpitis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1992; **73**: 470–483.
35. Georgopoulou M, Anastassiadis P, Sykara S. Pain after chemico-mechanical preparation. *Int Endod J* 1986; **19**: 309–314.
36. Walton RE, Fouad A. Endodontic interappointment flare-ups. a prospective study of incidence and related factors. *J Endod* 1992; **18**: 172–177.
37. Morse DR, Furst ML, Belott RM, Lefkowitz RD, Spritzer IB, Sideman BH. Infections flare-ups and serious sequelae following endodontic treatment: a prospective randomized trial on the efficacy of antibiotic prophylaxis in cases of asymptomatic pulpal-periapical lesions. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1987; **64**: 96–109.
38. Torabinejad M, Kettering D, McCraw C, Cummings R, Dwyer TG, Tobias S. Factors associated with endodontic intrappointment emergencies of teeth with necrotic pulps. *J Endod* 1988; **14**: 261–266.
39. Genet JM, Wesslink PR, Thodencan Velzen SK. The incidence of preoperative and postoperative pain in endodontic therapy. *Int Endod J* 1986; **19**: 221–229.
40. Dionne RA. New approach to preventing and treating postoperative pain. *J Am Dent Assoc* 1992; **123**: 27–34.
41. Imura N, Zuolo ML. Factors associated with endodontic flare-ups. A prospective study. *Int Endod J* 1995; **28**: 261–265.
42. Rosenberg PA, Babick PJ, Schertzer L, Leung A. The effect of occlusal reduction on pain after endodontic instrumentation. *J Endod* 1998; **24**: 492–496.
43. Baumgartner JC. Treatment of infections and associated lesions of endodontic origin. *J Endod* 1991; **17**: 418.
44. Seltzer S, Naidorf IJ. Flare-ups in endodontics I. Etiological factors. *J Endod* 1985; **11**: 472–478.
45. American Association of Endodontists. *Glossary: contemporary terminology for endodontics*, 6th edn. Chicago: American Association of Endodontists, 1998.
46. Guthman J, Harrison JW. *Surgical endodontics*. Boston: Blackwell Scientific Publications, 1991.
47. Chestner SB, Selman AJ, Friedman J, Heyman RA. Apical fenestration solution to recalcitrant pain in root canal therapy. *J Am Dent Assoc* 1986; **77**: 846–848.

48. Moos HL, Amwell JD, Roahen JO. A comparison of pulpectomy alone versus pulpectomy with trephination for the relief of pain. *J Endod* 1966; **22**: 422-425.
49. Peters DD. Evaluation of prophylactic alveolar trephination to avoid pain. *J Endod* 1980; **6**: 518-526.
50. Elliot JA, Holcomb JB. Evaluation of a minimally traumatic alveolar trephination procedure to avoid pain. *J Endod* 1988; **14**: 405-407.
51. Houck V, Reader A, Beck M, Nist R, Weaver J. Effect of trephination on postoperative pain and swelling in symptomatic necrotic teeth. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2000; **90**: 507-513.
52. Nist E, Reader A, Beck M. Effect of apical trephination on postoperative pain and swelling in symptomatic necrotic teeth. *J Endod* 2001; **27**: 415-420.
53. Harrington GW, Natkin E. Midtreatment flare-ups. *Dent Clin North Am* 1992; **36**: 409-423.
54. Hutter JW. Facial space infections of odontogenic origin. *J Endod* 1991; **17**: 422.
55. Natkin E. Treatment of endodontic emergencies. *Dent Clin North Am* 1974; **18**: 243-255.
56. Antrim DD, Bakland LK, Parker MW. Treatment of endodontic urgent care cases. *Dent Clin North Am* 1986; **30**: 549-572.
57. Cunningham CJ, Mullaney TP. Pain control in endodontics. *Dent Clin North Am* 1992; **36**: 393-408.
58. Harrington GW, Natkin E. Midtreatment flare-ups. *Dent Clin North Am* 1992; **36**: 409.
59. Gatewood RS, Himel VT, Dorn SO. Treatment of the endodontic emergency: a decade later. *J Endod* 1990; **16**: 284-291.
60. Creech JL, Walton RE, Kaltenbach R. Effect of occlusal relief on endodontic pain. *J Am Dent Assoc* 1984; **109**: 64-67.
61. Jostes JL, Holland GR. The effect of occlusal reduction after canal preparation on patient comfort. *J Endod* 1984; **10**: 34-37.
62. Gatchel RJ. Managing Anxiety and Pain during dental treatment. *J Am Dent Assoc* 1992; **123**: 37-41.
63. Sokol D, Sokol S, Sokol C. A Review of non-intrusive therapies used to deal with anxiety and pain in the dental office. *J Am Dent Assoc* 1985; **110**: 217-222.
64. Gibson HB. *Psychology, pain and anesthesia*. London: Chapman and Hall, 1994.
65. Kiecolt-Glaser JK, Page GG, Marucha PT, MacCallum RC, Glaser R. Psychological influences on surgical recovery: perspectives from psychoneuroimmunology. *Am Psychol* 1998; **53**: 1209-1218.
66. Lauth H. Dental phobia. *Br J Psychiatry* 1971; **119**: 151-158.
67. Forgionne A, Clarke RE. Comments on an empirical study of dental fears. *J Dent Res* 1974; **53**: 496.
68. Shoben F, Borland L. An empirical study of the etiology of dental fears. *J Clin Psychol* 1954; **10**: 171-174.
69. Bernstein DA, Kleinknecht RA. Comparative evaluation of these social-learning approaches to the reduction of dental fear. In: Ingersoll B, McCutcheon W, eds. *Conference on Behavioral Dentistry: Clinical Research on Behavioral Dentistry*. Morgantown, WV, 1979: 37-49.
70. Kendall PC, Williams L, Peacheck TF, Graham LE, Shisslak C, Herzoff N. Cognitive-behavioral and patient education interventions in cardiac catheterization procedures: The Palo Alto medical psychology project. *J Consult Clin Psychol* 1979; **47**: 49-58.
71. Siegel LJ, Peterson L. Stress reduction in young dental patients through coping and sensory information. *J Consult Clin Psychol* 1980; **48**: 785-787.
72. Wardle J. Psychological management of anxiety and pain during dental treatment. *J Psychosom Res* 1983; **27**: 399-402.
73. Adelson D, Goldfried M. Modeling and the fearful child patient. *J Dent Child* 1970; **37**: 476-488.
74. Melamed BG, Weinstein D, Hawes R, Katin-Borland. Reduction of fear-related dental management problems with use of filmed modeling. *J Am Dent Assoc* 1975; **90**: 822-826.
75. Chaves JF, Barber TX. Cognitive strategies, experimenter modeling and expectation in the attention of pain. *J Abnorm Psychol* 1974; **83**: 316-363.
76. Russell MW. The management of dental pain: a review of possible alternatives. *Tex Dent J* 1980; **98**: 6-8.
77. Morse D, Wilko J. Non surgical endodontic therapy for a vital tooth with medication hypnosis as the sole anesthetic. Case Report. *Am J Clin Hypn* 1979; **21**: 258-262.
78. Eli I, Bartal Y, Fuss Z, Silberg A. Effect of intended treatment on anxiety and on reaction to electric pulp stimulation in dental patients. *J Endod* 1997; **23**: 694.
79. Klepac RK, Dowling J, Hauge G, McDonald M. Reports of pain after dental treatment electrical tooth stimulation and cutaneous shock. *J Am Dent Assoc* 1980; **100**: 692-695.