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Flare-ups in Endodontics: II. Therapeutic Measures

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Various treatment regimens for the relief of pain during endodontic therapy, including relief of occlusion, pre-medication, establishment of drainage, and intracanal and systemic medications are presented. In addition, the rationale for the use of placebos is discussed.

Se presentan varios regimenes de tratamiento para el alivio del dolor durante la terapia endodóntica, inclu-yendo el alivio de la oclusión, premedicación, estable-cimiento de un drenaje y medicaciones sistemáticas y dentro del conducto. Además, es discutido el uso racional de placebos.

TREATMENT OF PAIN DURING ENDODONTIC THERAPY

Because the etiological factors often cannot be precisely determined, many treatments have been empirically advocated for the prevention or alleviation of symptoms during root canal therapy. These include: relief of occlusion; premedication of the pulp chamber or root canal; the establishment of drainage through the root canal, or by the excision of the overlying tissues; and various medications applied to the root canal or administered systemically. No specific treatment is universally accepted. Each treatment has it advocates, but many of the regimens may be successful because of the placebo effect.

The following discussion of each of these treatment regimens is based on the available evidence which has been published.

Relief of Occlusion

Occlusal relief prior to endodontics has been advocated by Cohen (1) for the prevention of postoperative endodontic pain. Other endodontists (2–5) have recommended occlusal relief prior to endodontic therapy only in teeth with painful periapical symptoms. Many endodontists reduce the occlusion of teeth undergoing endodontic therapy when painful symptoms develop (6). However, in a controlled study on 49 patients, Creech et al. (7) found that relieving the occlusion was no more effective than mock-occlusal relief for relieving postoperative, spontaneous pain and duration of pain. However, their study did not determine whether occlusal relief would help patients with several preoperative pain.

Premedication of the Pulp Chamber or Root Canal at the First Appointment

The concept of medicating the pulp chamber or root canal to reduce the possibility of flare-ups due to the forcing of infected debris into the periapical tissues appears to be desirable. However, the benefits seem to be more imaginary than realistic. The incidence of painful exacerbations in patients whose root canals were premedicated prior to instrumentation has not been found to be different than in those whose root canals were completely instrumented (8, 9).

Establishment of Drainage

Inflammatory edema is induced by chemical mediators, especially the leukotrienes (LT's) and the vasodilator prostatglandins (PG's) E_2 and I_2; suppuration usually results from infections. In the presence of suppuration, drainage of exudate is the most effective method for reducing pain and swelling. The relief is frequently dramatic. Drainage is most simply accomplished by removing the temporary dressing from the root canal or by removing the temporary filling in the access opening. In most instances, the accumulated exudate will surge from the root canal, affording immediate relief. However, upon occasion, no exudate will emerge; it may be blocked by packed dentinal shavings in the apical third of the root canal.

Passing a root canal instrument, such as a file or reamer, through the caked material may help to establish the flow of exudate. In exceptional cases, the exudate is either absent or cannot be evacuated through the root canal. Surgical intervention is then necessary. The removal of the alveolar bone over the apex of the tooth root (creation of an artifical sinus tract), or a soft tissue incision when swelling has occurred (10) usually affords relief. The root canal can then be resealed, usually without further discomfort to the patient.

After the exudation is reduced, the access opening to the root canal can be temporarily closed again. Many endodontists prefer to leave the root canal open until symptoms have subsided. In our experience and those of Weine et al. (11) and August (12), this exposure to the oral flora serves no useful purpose and may actually cause subsequent flare-ups when additional treatment is undertaken. Exposure of the root canal to salivary products logarithmically increases bacterial growth, introduces new microorganisms, activates the alternate complement pathway, and may enhance bradykinin production, all leading to the exacerbation of pain.

Intracranial Medicaments

Among the medicaments claimed to afford relief from, or to prevent, painful exacerbations during root canal therapy are antimicrobial agents, irrigating solutions, sulfa compounds, and corticosteroids.

ANTIMICROBIAL AGENTS

Most intracanal medicaments are used primarily for their antimicrobial action. Since microorganisms are responsible for exacerbating inflammation, it would appear that the intracanal placement of root canal antiseptics and germicides should at least indirectly reduce posttreatment pain. Such does not appear to be the case in most instances.

The anodyne properties of formocresol, cresatin, eugenol, camphorated monochlorphenol, and iodine-potassium iodide have been studied (13, 14). None appeared to be particularly effective, nor was there any significant relationship between interappointment pain and the type of therapy used (15).

IRRIGATING SOLUTIONS

Our clinical experiences have indicated that the type of irrigating solution used makes little difference in the incidence of postoperative discomfort, providing that the irrigating solution is not forced beyond the foramen of the tooth. However, Harrison et al. (16) found that there was a higher incidence and degree of pain in patients whose canals were either not irrigated or irrigated with saline solution, compared with those irrigated with 5.25% sodium hypochlorite and 3% H_2O_2. An added benefit of such irrigation, even at 0.5% sodium hypochlorite (17), is its antibacterial activity, including that against Gram-negative anaerobes which produce endotoxin (18, 19).

Buttler and Crawford (18) found that, in vivo, small amounts of endotoxin were detoxified by 2.6% solutions of NaOCl. However, large amounts of endotoxin were not. On the other hand, formocresol, 5.25% NaOCl, and 3% H_2O_2 have not been found to increase the incidence of interappointment pain (15, 20–22). Since the induction of pain in endodontic therapy is multifactorial, it is difficult to attribute a lower pain incidence specifically to the use of any particular irrigant.

SULFA COMPOUNDS

When placed in the root canal, sulfa compounds have been reported to dramatically reduce the incidence of pain during endodontics. Nygaard-Östby (23) and Frank et al. (24) have reported impressive results with these drugs. On the other hand, our studies have shown that the sulfonamides yielded no better results than placebos (25).

CORTICOSTEROIDS

The anti-inflammatory activity of corticosteroids is based partly on their ability to retard lysosomal release from cells by inhibiting fusion of lysosomes with their target membranes (26). In addition, corticosteroids inhibit the liberation of free arachidonic acid from the phospholipids of the cell membrane by phospholipases. On the other hand, aspirin and other nonsteroidal anti-inflammatory drugs block the enzymatic conversion of arachidonic acid to the PG's by inhibiting the cyclo-oxygenase responsible for oxygenation (27). The formation of LT's is apparently unaffected by the nonsteroidal anti-inflammatory agents. However, steroids not only prevent the formation of PG's and thromboxanes but also LT's and other oxygenated derivatives. It would thus appear that the therapeutic effects of steroids, which are not shared by aspirin-like drugs, are due not only to the inhibition of PG formation but also the inhibition of LT formation.

Cortisone also appears to have the ability to obtund pain, possibly because of its effect on stabilizing membranes. Although the exact mechanism is not known, the hormone may cause hyperpolarization of the nerves in the inflamed area or it may enhance the production of cyclic AMP, which in turn, activates a protein kinase. The kinase may cause phosphorylation of a protein constituent of the nerve cell membrane, leading to a change in membrane permeability (28). Increase of cyclic AMP causes a hyperpolarization that reduces transmission of nerve impulses.

A number of investigators have reported that corticosteroids placed into the root canal control pain successfully (29–33).

The disadvantage of using corticosteroids in endodontic therapy derive from their effects on inflammatory cells. Although the density of the inflammatory infiltrate in the periodontal ligament may be reduced by corticosteroids (34), they interfere with phagocytosis and protein synthesis. As a result, infections may become rampant and repair may be impaired or delayed.

SYSTEMIC DRUGS

Antibiotics

Antibiotics have been used, both locally and systemically, in anticipation of or for the relief of pain during endodontic therapy. Whether the pain-reducing effects of antibiotics are imagined or real remains to be documented. The systemic use of antibiotics should be restrained generally but appears to have some value when the patient exhibits signs of systemic involvement, such as cellulitis, fever, malaise, and toxemia. Even then, the choice of antibiotic is frequently empiric. The overuse of antibiotics risks the induction of hypersensitivity or anaphylactic reactions, systemic side effects, and the development of resistant strains of microorganisms.

Group A streptococci (*Streptococcus pyogenes*), which are responsible for many human infections, have a number of characteristics that exhibit antigenic variation of virulence. These include the hyaluronic capsule, the streptococcal chemotactic factor, and the M proteins. The last, M proteins, are responsible for the ability of these streptococci to resist phagocytosis by polys. Strains of new M proteins have arisen in populations experiencing frequent infections (35).

Most of the studies of the past few years, dealing with the sensitivity of microorganisms to various antibiotics, must now be discarded.

The use of the most popular antibiotic, penicillin, is based on the predominance of penicillin-sensitive microorganisms reportedly found in infected root canals.

Although most strains of bacteria found in endodontic infections are susceptible to penicillin, some, such as the anaerobic peptostreptococci, *Bacteroides fragilis*, are resistant (36–38). Despite many new antibiotics, bacteria have responded by the rapid evo-

lution of genetic variants which are resistant to all antibiotics. It seems that, in time, totally resistant bacteria emerge and, in many cases, predominate. Increasing numbers of strains of pathogens, such as *Streptococcus viridans* and *Staphylococcus aureus*, originally susceptible to penicillin (39), are becoming resistant (38, 40). Resistance is transferred from organism to organism by packages of genes, called plasmids (41). Such transference may occur both within and across species lines by conjugation (42). Many of the genes specifying antibiotic resistance are found on movable elements of DNA called transposons (43). Penicillin-sensitive organisms, such as *Bacteroides melaninogenicus*, may produce *β-lactamase* (a penicillinase) which renders them penicillin-resistant (44, 45). Such resistant strains may then protect other pathogens that would normally be susceptible to penicillin.

There appears to be a trend toward an increase in the number of anaerobic dental infections (46, 47). In such cases, some antibiotics, such as clindamycin or tinidazole, may be effective, but the organisms may be resistant to erythromycin, demeclocycline, or doxycycline (45). In a few cases of cellulitis induced by mixed anaerobic and facultative streptococcal root canal infections, Matusow and Goodall (48) obtained good resolution by root canal treatment and by using erythromycin.

The rational selection of an appropriate antibiotic to control root canal infections should depend on culturing and sensitivity testing. However, there are no significant studies which show that any specific antibiotic is capable of reducing or eliminating painful exacerbations during endodontic therapy.

Corticosteroids

Systemic corticosteroids have been successfully used to reduce pain and swelling mainly in oral surgical procedures (49–53). In a controlled study, Marshall and Walton (54) found that intramuscular injection of 4 mg of dexamethasone significantly reduced both the incidence and severity of pain 4 h after single-appointment endodontic therapy. After 24 h, pain incidence was still less than in the controls, but the results were not statistically significant.

Tryptophan

Tryptophan is an essential amino acid. When ingested, a small amount is carried past the blood-brain barrier into the brain. There it is utilized by certain brain neurons for conversion into serotonin (5-hydroxytrypt-amine). Centrally, serotonin plays a role in various behavioral responses, including elevation of pain threshold. Shpeen et al. (55), in a controlled study, reported that when 3 g of tryptophan were given daily to 25 patients, there was a significant reduction in postendodontic treatment pain after 24 h, compared with a control group.

ANALGESICS

Nonnarcotoc Analgesics

Nonnarcotic analgesics relieve pain without altering consciousness. They are relatively ineffective against severe pain; however, they can control most pain of dental origin.

The nonnarcotic analgesics are a heterogeneous group of synthetic organic compounds. They may act at the receptor site, controlling the cause of the pain; at the cord, affecting the transmission of pain impulses, or, at a central level, altering the perception of pain.

The analgesics that act at receptor sites presumably reduce the output of impulses from the receptors, but they may also counteract the chemical mediators produced as a result of the inflammatory response. In that case, the analgesic effect reduces the firing of nerve impulses. One of the substances implicated as a pain mediator is bradykinin. The thromboxanes and PG's E_2 and F_2 appear to exacerbate the pain induced by bradykinin (56).

Among the analgesics that act primarily on the pain perception threshold are salicylates (aspirin); combinations of aspirin, phenacetin, and caffeine; acetophenetidin (Phenacetin); acetaminophen (Liquiprin, Tempra, Tylenol, and Valadol); and propoxyphene (Darvon).

Nonsteriodal Anti-Inflammatory Agents

The anti-inflammatory and the analgesic activities of nonsteroidal anti-inflammatory agents (NSAIA's) are primarily due to their inhibition of PG production by inactivating the enzyme, cyclooxygenase (57). They may also inhibit phosphodiesterase, leading to increased cyclic AMP production (58).

The drug of choice for mild to moderate pain has always been aspirin. It is the most efficient nonnarcotic analgesic. It is also antipyretic, has a peripheral anti-inflammatory effect, and appears to be antagonistic to the action of bradykinin (59). The analgesic and anti-inflammatory actions of aspirin are based on its inhibition of cyclooxygenase. Aspirin has also been shown to cause hyperpolarization of the neural cell membrane due to an increase in permeability of the potassium ion and a decrease in that of the chloride ion (60).

Analgesics may also abolish pain awareness by acting on the dorsal horn cells and the reticular formation. Although aspirin does not appear to affect reticular pathways, a small portion (about 10% of the plasma level) has been detected in the brain (61). Nevertheless, the hypothalamus appears to be the primary site for the action of aspirin in the central nervous system (62).

Aspirin should not be used for patients who are prone to gastrointestinal stress or those who are allergic to it. In those cases, acetaminophen is a better substitute than phenacetin since it is somewhat less toxic. Both acetaminophen and phenacetin have analgesic and antipyretic effects similar to those of aspirin. However, they have only weak anti-inflammatory effects. In some cases, combinations of aspirin with phenacetin and caffeine appear to increase analgesic effects. However, none of the mixtures, including the traditional aspirin-phenacetin-caffeine combination, have been shown to have greater advantages than aspirin alone (62).

More recently, other nonsteroidal anti-inflammatory agents have been reported to be more effective than aspirin, with less side effects (63).

The early NSAIA's were phenylbutazone and indomethacin. Because of their toxicity, many newer compounds have subsequently been developed and marketed. These include tolmectin (Tolectin), ibuprofen (Motrin), naproxen (Naprosyn), and fenoprofen (Nalfon), among many others. All have been reported to be superior to aspirin as analgesics, at least following oral surgical procedures (63).

Reports of the efficacy of NSAIA's for relieving postendodontic pain are meager. In one study, empirin with codeine #3, Synalgos-DC, and Motrin were found to be equally effective for the relief of

postendodontic pain after 3 h (64). There were no reports after longer periods. In an unpublished study, Sas et al. found that the ibuprofen was no better than placebo in relieving pain following endodontic treatment. A possible reason may be that the formation of LT's and possibly other chemical mediators is not blocked by currently available NSAIA's.

Pentazocine (Talwin) belongs to a class of compounds called benzomorphans; however, it is inappropriate to group this drug with morphine and the other opiates. When given orally in doses of 50 mg, it is a less effective analgesic than 650 mg of aspirin (65). In addition, adverse side effects, such as drowsiness, dizziness, nausea, vomiting, sweating, and constipation, may be induced. Furthermore, it has been reported that administration of this drug caused hallucinations (66, 67).

When combined with 650 mg of aspirin (Talwin compound), the quantity of pentazocine can be reduced to 25 mg. Such mixtures have been reported to have analgesic effects superior to aspirin alone (65).

Failure to control pain with nonnarcotic analgesics may require the use of narcotic analgesics.

Narcotic Analgesics

Narcotic analgesics are most commonly prescribed for relief of severe pain. Most of the more potent analgesics (morphine, codeine, meperidine, pentazocine, and percodan) are primarily narcotics. They react with neurons in the brain stem, spinal cord, thalamus, and cerebral cortex (67), although the exact site of the action is unknown. However, the opiate receptors for enkephalins and endorphins have been found in the brain and hypophysis, particularly in the limbic system and the periaqueductal gray matter. The attachment of an opiate to one of these sites triggers a series of biochemical steps that are not yet completely understood. Simon (69) has theorized that the binding of opiates to receptors causes the release of sodium, which then enhances analgesia. Also, the endorphins may inhibit the production of cyclic AMP, which might counteract the pain-enhancing properties of the PG's.

The narcotic analgesics act primarily by controlling the reaction to pain. Sharp, localized pain is poorly relieved by the opiates, which effectively relieve duller, more chronic, and less severe pain. However, they are capable of raising the pain reaction threshold by causing relaxation, apathy, and freedom from anxiety. The control over the effects of stimuli and the euphoria that opiates produce are blocked by naloxone (70).

The commonly used narcotic analgesics consist of morphine, codeine, meperidine, and propoxyphene.

Morphine is the drug of first choice for severe pain (*Reference Handbook of the Medical Letter on Drugs and Therapeutics*, 1975). Previously, morphine was not found to be effective when administered orally. However, an effective oral morphine has now been formulated (Roxanol). Alternative drugs can be used when oral routes of drug administration are desired. These include narcotic analgesics (codeine, hydrocodone, oxycodone, and pholcodine), or combinations of narcotic analgesics and other drugs. Most of these opiate drugs produce approximately the same incidence and degree of unwanted side effects in equianalgesic doses (71). However, the availability of a wide range of semisynthetic and synthetic surrogates provides for therapeutic flexibility.

The optimum, recommended dosage for each analgesic affords the maximum pain-relieving action. Increasing the amount does not raise the pain threshold significantly; rather, it prolongs the effect only slightly and accentuates side effects. The most effective way of dealing with pain is the repeated administration of one agent at regular intervals to keep the threshold high (72).

Although narcotics are effective for control of pain in pathosis, Beecher (73) has found it impossible to demonstrate any dependable relationship between pain threshold and narcotic dosage. For example, only a few patients experience high-intensity pain during their early postoperative period. However, since anxiety and reactivity to pain may be at a high pitch during the early postoperative period, narcotics and sedatives tend to be given too freely. Dodson and Bennett (74) found that patients could be kept free of suffering after operation if there were nurses available with sterile hypodermics of saline to provide relief of pain upon request. These patients were as comfortable as those receiving opiates. Also, instructions and assurances by the doctor reduced the number of times postsurgical patients requested pain-relieving drugs (75).

There is no specific analgesic that is preferentially effective for the pain induced during root canal therapy.

Since pain consists of the actual perception of the painful stimuli and their psychic modifications, the drugs prescribed for the relief of pain during endodontic therapy may alter either of these components. Thus, the prescription of an analgesic should not be made randomly.

The patient's previous experience with analgesics frequently affects the potency of the drug. For example, codeine should not be prescribed if it was not effective previously or had produced undesirable side effects, such as nausea or intestinal upset. Frequently, patients will report earlier satisfaction with less powerful analgesics or even with placebos. Consequently, it is wise to ask the patient what medications have been found in the past to be effective for pain relief. The same medications should then be prescribed, even if the patient's confidence in the drug is not shared by the dentist. Only if the medication is medically contraindicated should the dentist disregard the patient's preference.

Placebos

Relief of pain need not be related to the amount of analgesic administered. In fact, pain may be relieved by administration of placebos.

Placebos are pharmacologically inert substances that nonetheless have a therapeutic effect. They act by alleviating anxiety and are fairly effective in a high percentage of cases. As analgesics, they mimic the action of active drugs. In 15 studies, Beecher (76) has shown that about 35% of more than 1,000 patients reported relief of pain from severe postoperative wounds after receiving a placebo. Placebos have also been reported to relieve headaches in 52% of patients (77) and pain from angina pectoris in 38% of patients (78, 79).

Administration of placebos have been shown to have other beneficial effects. Burger (80), in a review of the placebo effect, has reported that saline injections reduced asthma attacks in one-third of asthmatic patients. Sugar blood levels have been controlled by the use of placebos in 62% of patients with diabetes. Ninety-two percent of patients with peptic ulcers have reported improved symptoms following placebo usage. Symptoms have also been improved in 80% of patients with rheumatoid and degenerative forms of arthritis.

In addition, toothaches have been cured by placebos in a remarkably high percentage of cases. Shapiro (81) comprehensively reviewed the history of the effects of placebos. He revealed that, in

1794, Dr. R. Gerbi, an Italian professor, published a manuscript in which a miraculous, year-long cure for toothache was described. A worm species, *Curculio antiodontalgious*, was crushed between the thumb and forefingers of the right hand. The patient was then instructed to touch the affected part of the tooth with his fingers. As determined by an investigatory commission, 431 of 629 (69%) toothaches were stopped immediately. This discovery was later advanced by Dr. Carradori, court physician at Weimar, who substituted the more pleasant ladybird in the prescription, and an official commission confirmed the immediate relief of toothache in 65 of 70% of the cases.

Soon afterwards, an English paper was published in which the following prescription for toothache was reported: "fill your mouth with milk and shake it until it becomes butter," in this way at least three out of four toothaches cease immediately and without fail (82).

But by far the most dramatic cure for a toothache was reported in the June 30, 1977 issue of *Moneys-worth* (p. 7): "Man Takes Shot for Tooth Pain"—Montevideo, Uruguay—To end the tormenting pain of toothache, an Uruguayan farmer shot away the tooth with a .22 caliber pistol. Hospital officials in Salto, 300 miles northwest of Montevideo, said Ernesto Erosa, 29, was recovering from the gunshot that not only demolished his tooth, but also his gums, his lower lip, and jaw."

Modern versions of toothache remedies take the form of "toothache drops," which at least contain a topical anesthetic, chlorobutanol.

The observed effects of drug administration are a combination of the pharmacological and the placebo (83). Frequently, the placebo effects are based on the patient's comprehension of, and emotional response to, drug administration (84). Improvement in psychiatric patients has even been noted to occur even when the patients knew they were receiving placebos (85).

Stangely, placebos are 10 times more effective in relieving pain of pathological origin than they are in relieving contrived pain (86). Their greater effectiveness is based on their ability to control the anxiety present in a diseased state, which is more intense than the anxiety in an experimental situation.

The therapist himself exerts a potent placebo effect (80). His conviction that an analgesic is effective is communicated to the anxious patient's expectations of relief (77). Moreover, the instructions or suggestions given the patient are powerful aids in the control of pain. In addition, patients who receive information about possibly unpleasant consequences of impending surgical procedures are less likely to overreact to postsurgical pain (87). Simple admission to a modern hospital has a dramatic placebo effect on some patients, and the performance of a series of diagnostic tests may also, at times, alleviate pain. A placebo does not have to be a medication. It can be a person, a procedure, a place, or ritual.

When a therapist has confidence in a particular drug or treatment regimen it is frequently successful; the enthusiasm of the practitioner is transferred to the patient (77). A pill is a potent symbol of the therapist; it supports the patient in the doctor's absence. It reinforces the patient's desire to get better, may fulfill a need to be punished, or may even satisfy a need to feel dependent. Strangely, a pill may be effective even though the patient knows it is a placebo (85). Furthermore, placebos are capable of enhancing endorphin production with resultant pain relief (88).

As Dinnerstein et al. (84) have noted, some of the effects of drug administration are based on the patient's comprehension of the expected effects and the instructions and suggestions given by the therapist. A sympathetic and professional attitude on the part of the dentist can provide a most important therapeutic benefit.

TREATMENT OF PAIN AFTER COMPLETION OF ENDODONTIC THERAPY

Following the completion of endodontic therapy, patients occasionally complain of pain, especially on biting and chewing. The incidence of such pain after root canal filling is small (89), and the number of treatment visits apparently makes little difference (90, 91).

The reported incidence of postoperative pain following single- or multiple-visit endodontic treatments varies considerably. In some studies, single-visit procedures produced less pain (92). In others, the incidence was the same (93–95).

In a few studies, single-visit procedures produced a much higher incidence of posttreatment pain (96–98).

There appears to be a tendency toward increased incidences of postoperative discomfort when pain is present preoperatively (22, 54, 93). Endodontic treatment of posterior teeth also seems to produce more postoperative discomfort (93, 96), especially after single-visit procedures (92). Fox et al. (99) found that 90% of the teeth treated endodontically in one visit were free of spontaneous pain after 24 h, whereas 82% had little or no pain on pressure. Pressure pain usually lasts longer than spontaneous pain. These painful episodes are usually caused by the pressures inherent in the insertion of the root canal filling materials or by the chemical irritation from the ingredients of the root canal cements or pastes. Overextending the root canal filling material beyond the foramen increases the incidence of subsequent pain (22). The ensuing periapical inflammation results in the firing of proprioceptive nerve fibers in the periodontal ligament. As a rule, these effects are short-lived and abate within a 24- to 48-h period. No treatment is usually necessary. The persistence of pain on biting, especially when accompanied by swelling, is an indication that a severe, acute inflammation has developed. Such cases usually require surgical intervention, such as periapical curettage. Persistent sensitivity or pain for longer periods may indicate failure of resolution of the inflammation in the periradicular tissues. In rare cases, inflamed but viable pulp tissue may be left in the root canal and may receive nutrition from accessory canals in the area. Retreatment of the root canal is then indicated.

Another possible cause for persistent pain after root canal therapy is a fracture of the crown or root. Cracks in the dentin may result from excessive pressure during the insertion of the root canal filling material or from masticatory pressures. These teeth eventually require extraction.

In rare cases, paresthesia and pain of the lower jaw has been induced by overinstrumentation or overextension of root canal filling material (100, 101), especially with paraformaldehyde preparations (102, 103). Although the paresthesias are usually temporary, a few have been reported to persist for longer than 1 yr, especially after periapical surgery (104, 105).

Acupuncture

Acupuncture has been used for dental analgesia with good success in a number of dental procedures (107, 108). With respect to endodontics, Gross and Morse (109) found that the depth of induced analgesia was not enough to permit pain-free pulp extirpation in most cases; supplementary injections of local anesthetics were needed. They concluded that acupuncture was not practical as a routine procedure for clinical endodontics. In another study (110), acupuncture was reported to be effective (at the 90%+ level) for the relief of "tooth-related pain." In this group were an

unspecified number of patients who complained of intense postoperative endodontic pain; their pain was reduced to tolerable levels within 15 to 20 min. This relief lasted indefinitely in almost 50% of the cases.

The effects of acupuncture are possibly due to the central release of endorphins (111–113).

At the present time acupuncture is not used to treat endodontic flare-ups, possibly because most endodontists are not familiar with the technique. Furthermore, as with many pain control techniques, the popularity of acupuncture has waned.

Explanations and Instructions

Detailed explanations of the procedures, the expected benefits, and the possible pain responses help to allay the patients' anxiety and apprehension and reduce tension (114, 115). Patients were more willing to endure pain if it was predicted. Janis and Mann (87) have shown that such explanations enable patients to cope better with operations and reduce the number of analgesics. Patients prefer to know what will happen and that knowledge reduces the impact of aversive stimuli (116). Furthermore, there is an increasing body of evidence to show that stress can suppress the immune system, rendering organisms more vulnerable to certain diseases and neoplasias. Stress reduces the level of circulating antibodies and suppresses the reactivity of lymphocytes to mitogenic and antigenic stimulation. The activity of natural killer lymphocytes is also suppressed in humans who cope poorly with the stresses of life changes. Such suppression may be mediated by endogenous opioid peptides (117).

Specific instructions relating to therapeutic regimens, such as application of ice, exact timing for ingestion of analgesics, and possible alterations in the character of pain also result in an elevation of pain threshold (118).

An infrequent unexpected anxiety may be induced by predictions of pain and swelling that fail to materialize, but such anxieties can usually be resolved by reassurances.

SUMMARY

A number of factors responsible for pain and swelling during and after endodontic therapy have been presented. In addition, the currently available treatment modalities for such flare-ups have been discussed.

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