Incidence of Postoperative Pain After Intracanal Procedures Based on an Antimicrobial Strategy

José F. Siqueira, Jr., DDS, MSc, PhD, Isabela N. Rôças, DDS, Amauri Favieri, DDS, MSc, Andréia G. Machado, DDS, Sérgio M. Gahyva, DDS, MSc, Julio C. M. Oliveira, DDS, MSc, and Ernani C. Abad, DDS

The purpose of this prospective study was to evaluate the incidence of postoperative pain after intracanal procedures based on an antimicrobial strategy. Data were examined from 627 teeth that had necrotic pulps or required retreatment. Information was obtained for each patient treated with regard to presence of preoperative pain. Occurrence of periradicular bone destruction detected by radiographs was also recorded. The operators consisted of undergraduate students, who were in their first year of clinical training. Root canals were instrumented and then medicated with a calcium hydroxide/camphorated paramonochlorophenol paste. No systemic medication was prescribed. Approximately 1 week after the initial appointment, patients were asked about the occurrence of postoperative pain and the level of discomfort was rated as no pain, mild pain, moderate pain, or severe pain. Data were statistically analyzed using the Chi-square test. Mild pain occurred in 10% of the cases, moderate in 3.3%, and severe (flare-up) in 1.9%. Postoperative pain was significantly associated with the treatment of previously symptomatic teeth without periradicular lesions (p < 0.01). No other correlations were detected between the occurrence of postoperative discomfort and other clinical conditions. There was also no difference regarding the incidence of postoperative pain between treatment and retreatment (p > 0.01). The intracanal procedures used in this study to control root canal infections showed a small incidence of postoperative pain, particularly flare-ups, even performed by inexperienced undergraduate dental students.

Postoperative pain after endodontic procedures is an undesirable occurrence for both patients and clinicians. Certain factors may predispose to the development of postoperative pain, such as preoperative pain and retreatment (1–3). The causes of postoperative pain include mechanical, chemical, and/or microbial injury to the pulpal or periradicular tissues. In fact, it has been suggested that microbial injury is probably the major and the most common cause of postoperative pain, and some Gram-negative anaerobic bacteria may play an important role in the development of symptoms (4, 5).

Acute periradicular inflammation is the most common cause of postoperative pain. It can develop as a result of any type of insult from the root canal system. In reality, regardless of the type of injury, the intensity of the periradicular inflammatory response is directly proportional to the intensity of the tissue injury. When periradicular tissues are injured, a myriad of chemical substances are released or activated, which will mediate the events of inflammation, such as vasodilation, increase in vascular permeability, and chemotaxis to inflammatory cells. The chemical mediators of inflammation include histamine, arachidonic acid metabolites, cytokines, neuropeptides, lysosomal enzymes, nitric oxide, oxygen-derived free radicals, and plasma-derived factors (complement, kinin, and clotting systems) (5). Most of these mediators have been detected in periradicular lesions (6). Although some mediators can cause pain by direct effects on sensory nerve fibers, the major inflammatory event responsible for periradicular pain is the increase in vascular permeability and the consequent edema, which lead to the compression of nerve fibers.

The occurrence of mild pain is relatively common even when the treatment has followed the highest standards and should be expected and anticipated by patients. However, a flare-up, characterized by the development of severe pain and/or swelling after intracanal procedures, is an unusual occurrence. Studies have reported varying frequencies of flare-ups ranging from 1.4% to 16% (1–3, 7–11).

Because of the microbial etiology of the periradicular diseases, endodontic therapy should be based on antimicrobial strategies (5). In addition, if microorganisms are the commonest causative factors of postoperative pain, a lower incidence of pain might be expected after the accomplishment of intracanal procedures based on antimicrobial strategies. Therefore, the purpose of this prospective study was to evaluate the incidence of postoperative pain after intracanal procedures based on an antimicrobial strategy.

TABLE 1. Occurrence of postoperative pain in different clinical conditions

Clinical Condition	n	Pain			
		Absent	Mild	Moderate	Severe
Treatment	499	423 (84.8%)	50 (10%)	16 (3.2%)	10 (2%)
Asymptomatic teeth	139	128 (92.1%)	8 (5.7%)	3 (2.2%)	0
Symptomatic teeth	53	38 (71.7%)	10 (18.9%)	3 (5.7%)	2 (3.8%)
Asymptomatic teeth/periradicular lesion	240	205 (85.4%)	23 (9.6%)	7 (2.9%)	5 (2.1%)
Symptomatic teeth/periradicular lesion	67	52 (77.6%)	9 (13.4%)	3 (4.5%)	3 (4.5%)
Retreatment	128	109 (85.1%)	12 (9.4%)	5 (3.9%)	2 (1.6%)
Asymptomatic teeth	65	57 (87.7%)	6 (9.2%)	1 (1.5%)	1 (1.5%)
Asymptomatic teeth/periradicular lesion	55	47 (85.4%)	3 (5.5%)	4 (7.3%)	1 (1.8%)
Symptomatic teeth/periradicular lesion	8	5 (62.5%)	3 (37.5%)	0	0
Total	627	532 (84.8%)	62 (10%)	21 (3.3%)	12 (1.9%)

MATERIALS AND METHODS

Data were examined from 627 teeth from 602 patients that had been referred for root canal treatment to the undergraduate Clinic of Endodontics, Estácio de Sá University, Rio de Janeiro, Brazil, over a 3-yr period. The selected teeth had a necrotic pulp or need for retreatment. Information was obtained for each patient treated with regard to the presence of preoperative pain. Occurrence of periradicular bone destruction radiographically detected was also recorded. Patients' age ranged from 18 to 75 yr. The operators consisted of undergraduate students who were in their first year of the endodontic program.

Each patient was anesthetized with local anesthetic solutions (long-acting local anesthetics were not used). The rubber dam was placed, the operative field decontaminated with 2.5% sodium hypochlorite (NaOCl), and conventional straight-line access preparations were performed. Chemomechanical preparation was completed using the same technique for all teeth—the alternated rotation motion technique, as described by Siqueira (5). Briefly, the coronal two-thirds of the root canals were enlarged with Gates Glidden burs (sizes varying depending on the root anatomy). In curved canals, initial passive step-back instrumentation was performed to facilitate the use of Gates Glidden burs as well as to direct their cutting action to the anticurvature dentinal walls. Working length was established 1 mm short of the root apex and the patency length coincided with the radiographic root end. Apical preparation was completed to the working length with either hand stainless steel files (in straight roots) or nickel-titanium files (in curved roots), always using a back-and-forth rotation motion. Master apical files ranged from #35 to #60, depending on both root anatomy and initial diameter of the root canal. Apical patency was confirmed with a small file throughout the procedures after each larger size file. Preparation was completed using step-back of 1-mm increments. Irrigation was always performed using 2.5% NaOCl solution.

In retreatment cases, after removal of the previous root canal filling using Gates Glidden burs, hand files, and eucalyptol, root canal preparations were completed as described above.

The root canals were irrigated with 17% EDTA followed by 2.5% NaOCl to remove smear layer and then medicated with a calcium hydroxide/camphorated paramonochlorophenol (CPMC)/glycerin paste in a creamy consistency. The paste was prepared by using equal volumes of CPMC and glycerin and applied into the canal using hand endodontic files. Although no systemic medication was prescribed, the patients were instructed to take mild analgesics if they experienced pain.

Approximately 1 week after the initial appointment, the patients returned for obturation of their root canals. All cases were obturated with gutta-percha cones and Sealer 26 sealer (Dentsply, Petrópolis, RJ, Brazil) using the lateral condensation technique. In the beginning of this appointment, patients were asked about the occurrence of postoperative pain. The level of discomfort was rated as follows: no pain; mild pain, which was recognizable but not discomforting; moderate pain, which was discomforting but bearable (analgesics, if used, were effective in relieving pain); severe pain, which was difficult to bear (analgesics, if used, were ineffective in relieving pain). Cases with severe postoperative pain and/or the occurrence of swelling were classified as flare-ups and treated accordingly.

The overall incidence of postoperative discomfort was recorded and expressed as a percentage of the total number of teeth evaluated. Incidence of postoperative pain was also calculated for each studied variable. Data were statistically analyzed using the Chi-square test.

RESULTS

Some level of postoperative pain occurred in 15.2% of the cases. Mild pain occurred in 10% of the cases, moderate in 3.3%, and severe (flare-up) in 1.9%. There was an absence of any level of discomfort in 84.8% of the all cases (84.8% of the treated teeth and 85.1% of the retreated teeth). Postoperative pain was significantly associated with previously symptomatic teeth without periradicular lesions ($p < 0.01$). No other correlations were detected between the occurrence of postoperative discomfort and other clinical conditions. There was also no difference regarding the incidence of postoperative pain between treatment and retreatment ($p > 0.01$).

The overall incidence of flare-ups was relatively low (1.9% of the cases). Severe pain occurred in 2% of the treatment cases and 1.6% of the retreatment cases. The occurrence of flare-up was only positively associated with the treatment of previously symptomatic teeth without periradicular lesions ($p < 0.05$). Again, there was no significant difference regarding the occurrence of flare-ups when comparing treatment cases with retreatment cases ($p > 0.01$).

Of the previously symptomatic teeth, the treatment was effective in completely eliminating pain in 71.7% of the teeth without associated periradicular lesions, in 77.6% of the teeth with associated lesions, and in 62.5% of the retreatment cases. Data are summarized in Table 1.

DISCUSSION

An acute inflammatory response develops in the periradicular tissue as a result of additional insults from the root canal system, which can be of mechanical, chemical, or microbial origin. Mechanical and chemical injuries are usually associated with iatrogenic factors, such as overinstrumentation, apical extrusion of irrigants or medications, perforations, and so on. Microbial injury to the periradicular tissues is probably the commonest cause of flare-ups. Although microbial insult can be coupled with iatrogenic factors, it can sometimes occur even when the root canal procedures have been judicious and careful. Apical extrusion of contaminated debris to the periradicular tissues is one of the principal causes of postoperative pain. Forcing microorganisms and their products into the periradicular tissues can generate an acute inflammatory response, whose intensity will depend on the number and the virulence of extruded microorganisms (5). Theoretically, the presence of some bacteria commonly associated with clinical symptoms, such as *Porphyromonas* spp., *Prevotella* spp., and *Fusobacterium nucleatum* in asymptomatic teeth may predispose to postoperative pain provided they are apically extruded or allowed to overgrow (4, 5). Incomplete instrumentation may disrupt the equilibrium of the endodontic microbiota, favoring the overgrowth of some microbial species, which can cause exacerbations. In addition, introduction of new microorganisms into the root canal system during treatment, as a result of breakage of the aseptic chain, may be another cause of postoperative pain of microbial origin (5).

All instrumentation techniques are reported to cause apical extrusion of debris, even when the file action is maintained short of the apical terminus (12, 13). The difference is that some techniques extrude more debris than others do. Crown-down techniques, such as the alternated rotation motion technique used in this study, have been demonstrated to extrude a lesser amount of debris (12, 13). Because the amount of extruded debris may influence the response of the periradicular tissues, crown-down instrumentation has theoretically less probability to induce flare-ups.

During chemomechanical procedures, the apical patency of the root canal was maintained by using small instruments that moved passively through the terminus of the root canal without binding or enlarging the apical foramen. Although the concept of apical patency is a controversial issue in endodontics (14), this procedure did not seem to influence the occurrence of postoperative pain, even when used by inexperienced practitioners as it was in this study, based on the very small incidence of flare-ups observed. More importantly, the clinician should be aware of the risks of using large instruments at the patency length, because this procedure can result in severe periradicular injury, cause lack of an apical stop, and extrude a large amount of infected debris, which predispose to the occurrence of postoperative discomfort and/or jeopardize the outcome of the endodontic therapy (15).

Chemomechanical procedures were always completed in one visit. Although we did not compare the incidence of postoperative pain after partial versus complete preparation, maximum removal of irritants from the root canal system could have significantly contributed to the low incidence of flare-ups observed. In addition, the treatment was effective in completely eliminating pain in most of the previously symptomatic teeth, which can also be explained by the fact that maximum removal of irritants was performed in the emergency visit.

The incidence of postoperative pain and particularly of flare-ups was positively associated with the treatment of previously symptomatic teeth without periradicular lesions. Studies have shown that the presence of preoperative pain can significantly increase the probability of postoperative pain (1–3). There are conflicting results with regard to the influence of periradicular bone destruction on the incidence of postoperative pain. We found previously symptomatic teeth without radiolucent areas were more susceptible to flare-ups. Our results are diametrically opposite to those of Morse et al. (2) and Imura and Zuolo (3) but are in agreement with the study by Torabinejad et al. (1). Higher incidence of postoperative pain in teeth without periradicular lesions might be attributed to a lack of space for pressure release when periradicular bone resorption is absent.

Some studies have reported a significantly higher incidence of flare-ups in teeth that needed retreatment (1, 2). However, we found no correlation between the incidence of postoperative pain in cases of treatment or retreatment. Walton and Fouad (9) have also found no significant difference in flare-up incidence between treatment and retreatment cases. The reasons for these differences are not apparent. However, unlike most of the previous studies, treatment procedures were standardized in this investigation. Whether this was the cause of differences is difficult to determine.

There was an absence of any level of discomfort in 84.8% of the all cases. The overall incidence of postoperative pain and particularly of flare-ups was low. This incidence was compatible with most data from the literature and can be considered relatively unexpected when one takes into account that therapy was performed by inexperienced undergraduate students in their first year of clinical endodontic training. Such results with dental student patients are similar to those reported by Walton and Fouad (9).

Although we did not sample root canals before treatment, the pathologic conditions of both pulpal and periradicular tissues suggested that most of the treated cases were infected before treatment. Thus, treatment procedures were based on an antimicrobial strategy to control the root canal infection. Endodontic infections are polymicrobial, and no medication is effective against all the bacteria found in infected root canals. Combination of two medications may produce additive or synergistic effects. Evidences suggest that the association of calcium hydroxide with CPMC has a broader antibacterial spectrum (eliminating microorganisms that are resistant to calcium hydroxide), a higher radius of antibacterial action (eliminating microorganisms located in regions more distant from the vicinity where the paste was applied), and will kill bacteria faster than mixtures of calcium hydroxide with inert vehicles (16). Although CPMC has strong cytotoxic activity (17), studies have reported a favorable tissue response to a calcium hydroxide/CPMC mixture (18, 19). This association probably owes its biocompatibility to the slow release of PMC from the paste, to the denaturing effect of calcium hydroxide on connective tissue, which may prevent further periradicular tissue penetration by CPMC reducing its toxicity, and to the fact that the effect on periradicular tissues is probably associated with the antimicrobial effect of the paste, which allows natural healing to occur without persistent infectious irritation (16). If the wound area is free of bacteria when the transitory chemical irritation occurs, there is no reason to believe that tissue repair would not take place as the initial chemical irritant decreases in intensity.

The use of an antimicrobial intracanal dressing is a valuable tool to control endodontic infection. Whereas some investigators have reported that intracanal medications had no influence on the incidence of postoperative pain (1, 8), Harrison et al. (20) have shown that the use of an antimicrobial intracanal medication and sodium hypochlorite irrigation could prevent postoperative pain. There-

fore, the use of an antimicrobial strategy during the endodontic therapy can significantly remove microorganisms from the root canal and theoretically prevent postoperative pain, provided antimicrobial substances are not highly cytotoxic and do not extrude to the periradicular tissues. Although we did not compare different irrigants and intracanal medications, the low incidence of postoperative pain, particularly flare-ups, reported by this study can perhaps be explained by the used antimicrobial therapy.

Drs. Siqueira, Rôças, Favieri, Machado, Gahyva, Oliveira, and Abad are professors of Endodontics, Department of Dentistry, Estácio de Sá University, Rio de Janeiro, RJ, Brazil. Address requests for reprints to José F. Siqueira Jr., R. Herotides de Oliveira 61/601, Icaraí, Niterói, RJ, Brazil 24230-230.

References

1. Torabinejad M, Kettering JD, McGraw JC, Cummings RR, Dwyer TG, Tobias TS. Factors associated with endodontic interappointment emergencies of teeth with necrotic pulps. J Endodon 1988;14:261–6.
2. Morse DR, Koren LZ, Esposito JV, Goldberg JM, Sinai IH, Furst ML. Asymptomatic teeth with necrotic pulps and associated periapical radiolucencies: relationship of flare-ups to endodontic instrumentation, antibiotic usage and stress in three separate practices at three different time periods. Parts 1–5. Int J Psychosom 1986;33:5–87.
3. Imura N, Zuolo ML. Factors associated with endodontic flare-ups: a prospective study. Int Endod J 1995;28:261–5.
4. Seltzer S, Naidorf IJ. Flare-ups in endodontics: I. Etiological factors. J Endodon 1985;11:472–8.
5. Siqueira JF Jr. Tratamento das infecções endodônticas. Rio de Janeiro, Brazil: Medsi, 1997.
6. Torabinejad M. Mediators of acute and chronic periradicular lesions. Oral Surg Oral Med Oral Pathol 1994;78:511–21.
7. Barnett F, Tronstad L. The incidence of flare-ups following endodontic treatment. J Dent Res 1989;68(special issue):1253.
8. Trope M. Relationship of intracanal medicaments to endodontic flare-ups. Endod Dent Traumatol 1990;6:226–9.
9. Walton R, Fouad A. Endodontic interappointment flare-ups: a prospective study of incidence and related factors. J Endodon 1992;18:172–7.
10. Harrington GW, Natkin E. Midtreatment flare-ups. Dent Clin North Am 1992;36:409–23.
11. Kvist T, Reit C. Postoperative discomfort associated with surgical and nonsurgical endodontic retreatment. Endod Dent Traumatol 2000;16:71–4.
12. Al-Omari MA, Dummer PMH. Canal blockage and debris extrusion with eight preparation techniques. J Endodon 1995;21:154–8.
13. Favieri A, Gahyva SM, Siqueira JF Jr. Extrusão apical de detritos durante instrumentação com instrumentos manuais e acionados a motor. J Bras Endodon 2000;1:60–4.
14. Wu MK, Wesselink PR, Walton RE. Apical terminus location of root canal treatment procedures. Oral Surg Oral Med Oral Pathol 2000;89:99–103.
15. Siqueira JF Jr. Aetiology of the endodontic failure: why well-treated teeth can fail. Int Endod J 2001;34:1–10.
16. Siqueira JF Jr, Lopes HP. Mechanisms of antimicrobial activity of calcium hydroxide: a critical review. Int Endod J 1999;32:361–9.
17. Spangberg L, Rutberg M, Rydinge E. Biologic effects of endodontic antimicrobial agents. J Endodon 1979;5:166–75.
18. Torneck CD, Smith JS, Grindall P. Biologic effects of endodontic procedures on developing incisor teeth. IV. Effect of débridement procedures and calcium hydroxide-camphorated parachlorophenol paste in the treatment of experimentally induced pulp and periapical disease. Oral Surg Oral Med Oral Pathol 1973;35:541–54.
19. Holland R, Souza V, Nery MJ, Mello W, Bernabé PFE, Otoboni Filho JÁ. A histological study of the effect of calcium hydroxide in the treatment of pulpless teeth of dogs. J Br Endod Soc 1979;12:15–23.
20. Harrison JW, Baumgartner JC, Zielke DR. Analysis of interappointment pain associated with the combined use of endodontic irrigants and medicaments. J Endodon 1981;7:272–9.