
Influence of apical foramen widening and sealer on the healing of chronic periapical lesions induced in dogs' teeth

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Objective. The aim of this study was to evaluate the influence of apical foramen widening on the healing of chronic periapical lesions in dogs' teeth after root canal filling with Sealer 26 or Endomethasone.

Study design. Forty root canals of dogs' teeth were used. After pulp extirpation, the canals were exposed to the oral cavity for 180 days for induction of periapical lesions, and then instrumented up to a size 55 K-file at the apical cemental barrier. In 20 roots, the cemental canal was penetrated and widened up to a size 25 K-file; in the other 20 roots, the cemental canal was preserved (no apical foramen widening). All canals received a calcium hydroxide intracanal dressing for 21 days and were filled with gutta-percha and 1 of the 2 sealers: group 1: Sealer 26/apical foramen widening; group 2: Sealer 26/no apical foramen widening; group 3: Endomethasone/apical foramen widening; group 4: Endomethasone/no apical foramen widening. The animals were killed after 180 days, and serial histologic sections from the roots were prepared for histomorphologic analysis. Scores were assigned according to preestablished histomorphologic parameters and analyzed statistically by Kruskal-Wallis and Mann-Whitney *U* tests.

Results. Regarding new cementum formation, repair of cementum and bone resorption areas, presence of microorganisms, inflammatory cell infiltrate and periodontal ligament conditions, significantly better periapical healing was obtained when foramen widening was done and Sealer 26 was used.

Conclusion. Apical foramen widening and calcium hydroxide-containing sealer were more favorable to the healing of chronic periapical lesions. (*Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2010;109:932-940)

The main goal of root canal therapy is the proper cleaning and shaping of the root canal system, as well as filling it with proper material, such as gutta-percha and sealers. Ideally, the sealers should have adequate physical, chemical, and biologic properties.¹ During filling, sealers may come in direct contact with the periodontal tissues through apical foramen and accessory communications. In such conditions, they could delay wound healing, which justifies the importance of studying the tissue reaction before clinical use.

Root canal sealers have several bases: zinc oxide–eugenol, epoxy resin, glass ionomer, or calcium hydroxide. The presence or release of substances may

generate different reactions when in contact with the tissues. The reaction varies according to the substance, the amount released, and the resorption speed and is closely related to the width of the apical foramen.

The passive cleaning of the cemental canal with flexible files without widening the apical constriction has been specifically recommended for the endodontic treatment of teeth with necrotic pulps.² This procedure has become widespread, under the denomination of apical patency, with the aim of avoiding contaminated dentin chips, pulpal remnants, and microorganisms interfering in the healing process after endodontic treatment. However, the findings of an earlier study in dogs' teeth with periapical lesion showed that the best result was obtained when the cemental canal and the apical foramen were widened more than the patency instrument.³

Calcium hydroxide– or calcium oxide–containing cements have been suggested as filling materials because of their ability to dissociate into calcium and hydroxyl ions, resulting in a higher pH in the adjacent medium and inducing mineralized tissue formation.⁴⁻⁸ Sealer 26 (S26) (Dentsply, Rio de Janeiro, Brazil) is an epoxy resin–based endodontic sealer containing cal-

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cium hydroxide. Figueiredo et al.⁹ and Sunzel et al.¹⁰ reported that Sealer 26 caused only slight tissue reactions when evaluated over longer periods. The initial irritating effect of Sealer 26 is due to various substances present in its formulation. Sealer 26 is in fact modified AH-26 in which the silver in the original formula is replaced with calcium hydroxide.¹¹

Endomethasone is zinc oxide–eugenol based, consisting of paraformaldehyde and antiinflammatory steroids (corticosteroids). Paraformaldehyde does not impair biologic compatibility even though it is released after hardening of the sealer.¹² Ørstavik and Hongalo¹³ studied the mutagenic activity of isolated formaldehyde and of formaldehyde-releasing endodontic sealers, such as Endomethasone and AH-26. Only Endomethasone showed no mutagenic activity. The favorable tissue response to Endomethasone may be related to the corticosteroids, whose activity delays fibrogenesis.¹⁴ Bernáth and Szabó¹⁵ reported that Endomethasone did not cause an inflammatory reaction in 6 of 9 obturated root canals in monkeys.

The aim of the present study was to evaluate the influence of apical foramen widening on the healing of chronic periapical lesions in dogs' teeth after root canal filling with Sealer 26 or Endomethasone.

MATERIAL AND METHODS

Forty root canals of maxillary central and intermediate incisors, maxillary second and third premolars, and mandibular third and fourth premolars from 2 adult mongrel dogs aged ~1 year were used. The research protocol was reviewed and approved by the Research Ethics Committee of the University of Marília, Brazil, in compliance with the ethical guidelines for animal experimentation.

The animals were anesthetized with an intramuscular injection of a combination of xylazine (Coopazine; Coopers do Brasil, São Paulo, Brazil; 0.05 mL/kg body weight) and tiletamine hydrochloride:zolazepam hydrochloride (Zoletil-50; Virbac do Brasil, São Paulo, Brazil; 0.2 mL/kg body weight).

The first phase of the study was the induction of periapical lesions. For this purpose, the access cavities were prepared with a #1090 cylindrical diamond bur (KG Sorensen, Barueri, Brazil), the pulp tissue was extirpated with a size 15 K-file (Dentsply/Maillefer Instruments, Ballaigues, Switzerland) and the root canals exposed to the oral cavity for 180 days. After this period, rubber cup/pumice prophylaxis was performed, a rubber dam was placed, the tooth crowns were cleaned with a polyvinylpyrrolidone-iodine solution (Asteriodine-Aster, Sorocaba, Brazil), and the root canals were instrumented up to a size 55 K-file (Dentsply/Maillefer Instruments) using the crown-down tech-

nique at the limit of the cementodentinal junction (CDJ) that was determined by tactile sense. Throughout the preparation, the root canals were irrigated with 2.5% sodium hypochlorite (NaOCl; CicloFarma Indústria Farmacêutica, Serrana, Brazil) at each change of file. In 20 root canals, the cemental canal was penetrated with a size 15 K-file (Dentsply/Maillefer Instruments) to obtain a main apical foramen. This length was determined by radiography, and the cemental canal and the foramen were widened up to a size 25 K-file (apical foramen widening). In the other 20 root canals, the apical cemental barrier was preserved (no apical foramen widening).

Thereafter, all 40 canals were irrigated with 2.5% NaOCl, dried, and flooded with 17% EDTA solution for 3 minutes for removal of smear layer. After a final flush with 2.5% NaOCl, the canals were dried and filled with a paste prepared with calcium hydroxide and saline, and the access cavities were sealed with gutta-percha (SS White Artigos Dentários, Rio de Janeiro, Brazil) and zinc oxide–eugenol cement (SS White Artigos Dentários).

After 21 days, the intracanal dressing was removed with a K-file and saline irrigation, and the root canals were dried with sterile paper points and immediately filled at CDJ limit by lateral condensation of gutta-percha points and either a calcium hydroxide–containing sealer (Sealer 26; Dentsply Indústria e Comércio, Petrópolis, Brazil) or a zinc oxide–eugenol–containing sealer (Endomethasone; Septodont, Saint-Maur-des-Fossés, France). Four groups of 10 specimens each were formed, as follows: group 1: Sealer 26/apical foramen widening; group 2: Sealer 26/no apical foramen widening; group 3: Endomethasone/apical foramen widening; group 4: Endomethasone/no apical foramen widening.

Root canal filling was completed with the insertion of accessory gutta-percha points and active lateral condensation with finger spreaders (Dentsply/Maillefer Instruments). Radiographs were taken to evaluate the quality of root filling regarding homogeneity and apical extension. Material excess was removed with a heated plugger at the coronal level, and the remaining filling material was vertically condensed to obtain well compacted fillings. The pulp chambers were cleaned and the access cavities sealed with intermediate restorative material (IRM; Dentsply Indústria e Comércio) and silver amalgam (SS White Artigos Dentários).

The subjects were killed by anesthetic overdose 180 days after treatment. The maxillas and mandibles containing the root-filled teeth were removed, fixed in 10% neutral-buffered formalin, and decalcified in formic acid–sodium citrate solution. Segments of the jaws, each containing 1 root, were embedded in paraffin and

prepared for histologic examination in the usual manner. The blocks were cut serially and 6- μm -thick longitudinal sections were obtained, stained with hematoxylin and eosin (HE) and Brown and Brenn techniques, and examined under light microscopy by a skilled examiner blinded to the groups. The following histomorphologic parameters were examined:¹⁶ thickness and extension of newly formed cementum, closure of the apical opening of accessory canals and apical foramen of the main root canal with newly formed cementum, apical cementum and bone resorption, presence of microorganisms, intensity and extension of acute and chronic inflammatory infiltrate and thickness and organization of the apical periodontal ligament (PDL). Each parameter was scored from 1 to 4, with 1 being the best result and 4 the worst, according to previously established criteria¹¹ (Table I). Data were analyzed statistically by Kruskal-Wallis and Mann-Whitney *U* nonparametric tests for 5% significance level.

RESULTS

Group 1: Sealer 26/apical foramen widening (n = 10)

All specimens presented newly formed apical cementum with thickness ranging from 2 to 200 μm . Closure of the apical opening of all accessory canals in the apical delta occurred in 4 specimens (Fig. 1, *a*) and most accessory canals in 3 specimens. The other 3 specimens presented closure of the apical opening of few accessory canals. Complete closure of the apical foramen of the main canal was observed in 6 specimens (Fig. 1, *a* and *b*), 1 specimen presented partial closure, and in 3 specimens the apical foramen was patent. The areas of apical cemental resorption were nonresorbed or totally repaired in 6 specimens (Fig. 1, *a* and *b*), partially repaired in 3 specimens, and nonrepaired in 1 specimen. Five specimens showed bone tissue with no resorption areas, and the other 5 presented inactive or partially repaired resorption areas. The Brown and Brenn staining revealed bacteria inside dentinal tubules and cementoplasts in 5 specimens. A severe neutrophil infiltrate was observed in 4 specimens. Chronic inflammatory cells were observed in all specimens, being of mild, moderate, and severe intensity in 2 (Fig. 1, *a*), 4, and 4 specimens, respectively. The apical PDL thickness was up to 300 μm in 9 specimens and >400 μm in 1 specimen. The PDL was well organized in 1 specimen, partially organized in 8, and totally disorganized in the entire apical region in 1 specimen.

Group 2: Sealer 26/no apical foramen widening (n = 10)

All specimens presented newly formed apical cementum with thickness ranging from 19 to 150 μm .

Table I. Histomorphologic parameters and scores

<i>Thickness of newly formed cementum</i>	
1.	$\geq 60 \mu\text{m}$
2.	20-59 μm
3.	1-19 μm
4.	Absence of newly formed cement
<i>Extension of newly formed cementum</i>	
1.	Deposited in all resorption areas or over preexisting cementum
2.	Deposited in more than one-third of the resorption areas
3.	Deposited in up than one-third of the resorption areas
4.	Not deposited in the resorption areas
<i>Closure of the apical opening of accessory canals</i>	
1.	Closure of all accessory canals
2.	Closure of most accessory canals
3.	Closure of few accessory canals
4.	No closure of accessory canals
<i>Closure of the apical foramen of the main root canal</i>	
1.	Complete closure
2.	Partial closure
3.	Deposition only on the main canal walls
4.	No closure
<i>Areas of apical cementum resorption</i>	
1.	Absent or totally repaired
2.	Partially repaired
3.	Nonrepaired
4.	Active resorption areas
<i>Areas of bone tissue resorption</i>	
1.	Absent or totally repaired
2.	Inactive or partially repaired
3.	Few active resorption areas
4.	Several active resorption areas
<i>Microorganisms</i>	
1.	Absent
2.	Present
<i>Intensity of inflammatory cell infiltrate*</i>	
1.	Absent or very few cells
2.	Mild: <10 cells
3.	Moderate: 10-25 cells
4.	Severe: >25 cells
<i>Extension of inflammatory cell infiltrate*</i>	
1.	Absent
2.	Mild: only close to the foramen
3.	Moderate: in part of the apical PDL thickness
4.	Severe: in the entire apical PDL thickness
<i>Apical PDL thickness†</i>	
1.	$\leq 200 \mu\text{m}$
2.	201-300 μm
3.	301-400 μm
4.	>400 μm
<i>Apical PDL organization‡</i>	
1.	Well organized in all 4 parts
2.	Well organized in 3 parts
3.	Well organized in 1 or 2 parts
4.	Totally disorganized

PDL, Periodontal ligament.

*Acute and chronic processes were evaluated in different areas ($\times 400$ magnification).

†Average of measurements taken in 5 different areas.

‡The apical third of the root was divided into 4 parts of similar dimensions.

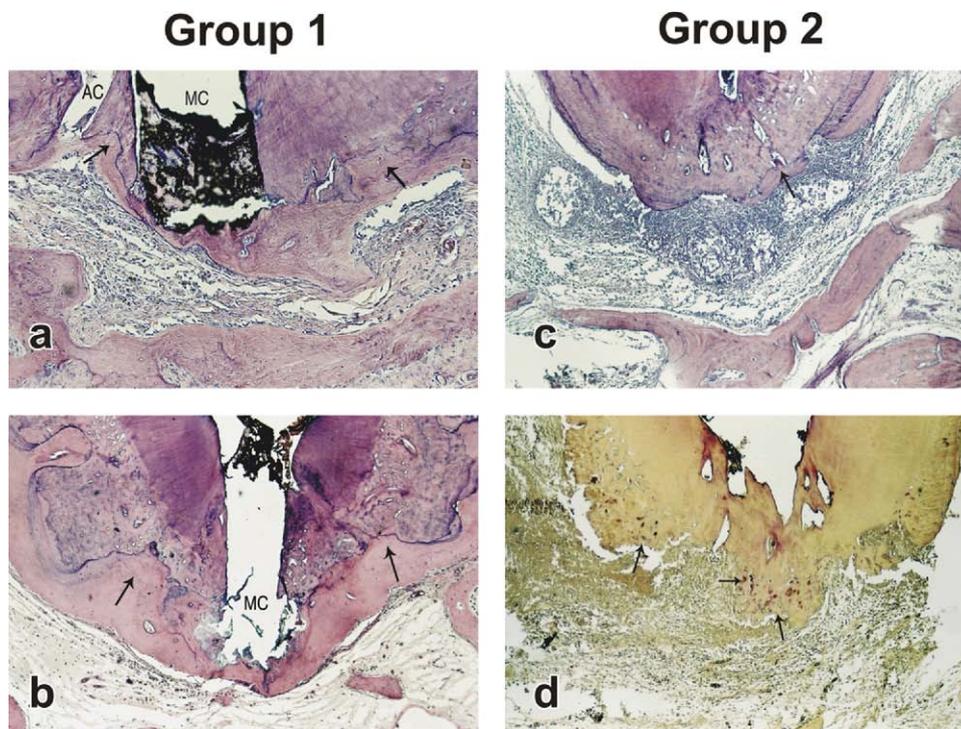


Fig. 1. **a**, Sealer 26/apical foramen widening. Complete closure of the apical foramen of the main canal (MC) and apical opening of one accessory canal (AC) by deposition of newly formed cementum, totally repaired areas of apical cemental resorption (*arrows*), and mild inflammatory infiltrate. Hematoxylin and eosin (HE), original magnification $\times 100$. **b**, Sealer 26/apical foramen widening. Complete closure of the apical foramen of the main canal (MC), totally repaired areas of apical cemental resorption (*arrows*), and mild inflammatory infiltrate. HE, original magnification $\times 40$. **c**, Sealer 26/no apical foramen widening. Closure of the apical opening of one accessory canal, partially repaired areas of apical cemental resorption (*arrow*) and severe inflammatory infiltrate. HE, original magnification $\times 40$. **d**, Sealer 26/no apical foramen widening. Nonrepaired areas of apical cemental resorption and microorganisms (*arrows*). Brown and Breen, original magnification $\times 40$.

Closure of the apical opening of all accessory canals in the apical delta occurred in 2 specimens, most accessory canals in 4 specimens, and few accessory canals in 3 specimens (Fig. 1, c). In 1 specimen, all apical ramifications were patent. The areas of apical cemental resorption were nonresorbed or totally repaired in 4 specimens, partially repaired in 2 specimens (Fig. 1, c), and nonrepaired in 3 specimens. One specimen presented active apical cementum resorption. Three specimens showed bone tissue with completely repaired resorption areas, 6 specimens showed partially repaired resorption areas, and 1 specimen presented few nonrepaired active resorption areas. The Brown and Brenn staining revealed bacteria inside the dentinal tubules and cementoplasts in 7 specimens (Fig. 1, d). A severe acute inflammatory infiltrate was observed in 5 specimens, and the other 5 specimens did not exhibit neutrophils. A severe chronic inflammatory infiltrate was observed in 8 specimens and was absent in the other 2 specimens. The apical PDL thickness was 200 μm in 1 specimen and ranged from 450 to 800 μm in the other

9 specimens. The PDL was well organized in the entire apical portion in 1 specimen, well organized in three-fourths of the apical third in 2 specimens, and totally disorganized in 7 specimens.

Group 3: Endomethasone/apical foramen widening (n = 10)

All specimens presented newly formed apical cementum with thickness ranging from 15 to 75 μm . Closure of the apical opening of all accessory canals in the apical delta occurred in 1 specimen, most accessory canals in 5 specimens, and few accessory canals in 1 specimen. In 3 specimens, the apical openings of all ramifications were patent. Partial closure of the apical foramen of the main canal was observed in 3 specimens, in 4 specimens deposition of newly formed cementum occurred only on the lateral walls of the cemental canal (Fig. 2, a), and in the other 3 specimens the apical foramen of the main canal was patent (Fig. 2, b). The areas of apical cemental resorption were totally repaired in 3 specimens (Fig. 2, a), partially repaired in

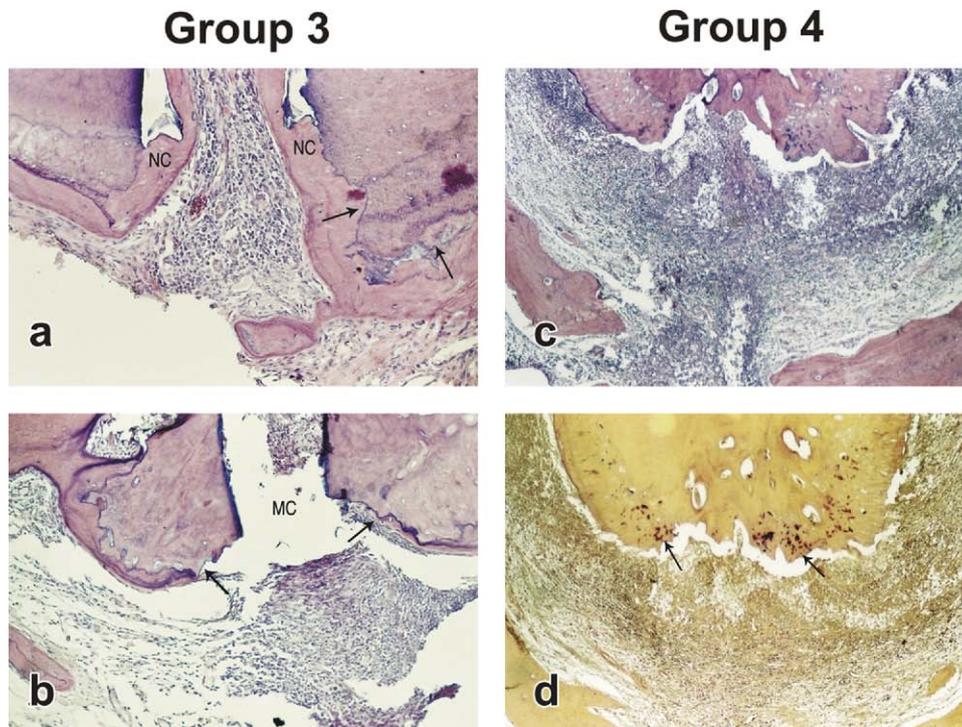


Fig. 2. **a**, Endomethasone/apical foramen widening. Deposition of newly formed cementum (NC) on the lateral walls of the main canal, totally repaired areas of apical cementum resorption (*arrows*), and severe inflammatory infiltrate in the ingrown periodontal tissue. Hematoxylin and eosin (HE), original magnification $\times 100$. **b**, Endomethasone/apical foramen widening. No closure of the apical foramen of the main canal (MC), partially repaired areas of cementum resorption (*arrows*), and severe inflammatory infiltrate in the widened apical periodontal ligament (PDL) space. HE, original magnification $\times 100$. **c**, Endomethasone/no apical foramen widening. Nonrepaired areas of apical cementum resorption and severe inflammatory infiltrate in the widened apical PDL space. HE, original magnification $\times 40$. **d**, Endomethasone/no apical foramen widening. Same specimen presented in panel *c*. Nonrepaired areas of apical cementum resorption showing cementoplasts with microorganisms (*arrows*). Brown and Brenn, original magnification $\times 40$.

5 specimens (Fig. 2, *b*), and nonrepaired in 2 specimens. Three specimens showed bone tissue with non-resorbed or completely repaired resorption areas, and the other specimens exhibited inactive or partially repaired bone resorption areas. The Brown and Brenn staining revealed bacteria in only 2 specimens, mainly inside cementoplasts. Neutrophil infiltrates with different intensities and extensions were observed in 8 specimens, but chronic inflammatory cell infiltrates with different intensities and extensions were found in all specimens. The apical PDL thickness was up to 200 μm in 2 specimens, between 301 and 400 μm in 6 specimens and >401 μm in 2 specimens. The PDL was well organized in the entire apical portion in 1 specimen, well organized in one-half of the apical third in 5 specimens, and totally disorganized in 4 specimens.

Group 4: Endomethasone/no apical foramen widening (n = 10)

Two specimens did not present new cementum formation in the apical region. In the other 8 specimens,

the thickness of the newly formed cementum ranged from 15 to 60 μm . Closure of the apical opening of most accessory canals in the apical occurred in 4 specimens and of few accessory canals in 2 specimens. In 4 specimens, the apical openings of all ramifications were patent. The areas of apical cemental resorption were partially repaired in 6 specimens (Fig. 2, *b*) and non-repaired in 4 specimens (Fig. 2, *c*). Regarding the bone tissue in the periapical region, 9 specimens showed inactive or partially repaired resorption areas and 1 specimen exhibited few active resorption areas. The Brown and Brenn staining showed bacteria inside the dentinal tubules and mainly cementoplasts in 8 specimens (Figs. 2, *d*). Acute inflammatory infiltrate was absent in 1 specimen. In the other 9 specimens, the acute inflammatory infiltrate was present with moderate and severe intensity and extension in 4 and 5 specimens, respectively. All specimens were infiltrated by chronic inflammatory cells with moderate and severe intensity and extension in 4 and 6 specimens, respectively. The apical PDL thickness was <200 μm in 1

Table II. Distribution of specimens according to the scores attributed to the histomorphologic parameters in each group and group comparison

Histomorphologic parameters*	Sealer 26 (n = 20)					Endomethasone (n = 20)														
	Group 1 (n = 10): apical foramen widening					Group 2 (n = 10): no apical foramen widening					Group 3 (n = 10): apical foramen widening					Group 4 (n = 10): no apical foramen widening				
	1	2	3	4	S†	1	2	3	4	S†	1	2	3	4	S†	1	2	3	4	S†
Newly formed cementum																				
Thickness	3	3	4	—		5	4	1	—		3	5	2	—		1	3	4	2	
Extension	8	2	—	—		4	4	2	—		4	6	—	—		—	6	—	4	
Closure of the apical opening of accessory canals	4	3	3	—		2	4	3	1		1	5	1	3		—	4	2	4	
Closure of the apical foramen of the main root canal	6	1	—	3		—	—	—	—		—	3	4	3		—	—	—	—	
Areas of apical cementum resorption	6	3	1	—		4	2	3	1		3	5	2	—		—	6	4	—	
Areas of bone tissue resorption	5	5	—	—		3	6	1	—		3	7	—	—		—	9	1	—	
Microorganisms	5	—	—	5		3	—	—	7		8	—	—	2		2	—	—	8	
Inflammatory infiltrate																				
Acute—intensity	6	—	—	4		5	—	—	5		2	3	2	3		1	—	4	5	
Acute—extension	6	3	1	—		5	—	—	5		2	3	3	2		1	—	2	7	
Chronic—intensity	—	2	4	4		2	—	—	8		—	1	4	5		—	—	4	6	
Chronic—extension	—	3	4	3		2	—	—	8		—	1	5	4		—	—	3	7	
Apical periodontal ligament:																				
Thickness	4	5	—	1		1	—	—	9		2	—	6	2		1	2	2	5	
Organization	1	6	2	1		1	2	—	7		1	—	5	4		—	2	1	7	
Group comparison					a					b					b					c

*See Table I for explanation of scores.

†Significance: Different letters indicate statistically significant difference at 5%.

specimen and ranged from 260 to 750 μm in the other 9 specimens. The PDL was partially organized in the entire apical portion in 3 specimens and totally disorganized in 7 specimens.

Statistical analysis

The distribution of specimens according to the scores attributed to the histomorphologic parameters in each group (individual values and mean scores), as well as the statistical comparison of groups, is summarized in Table II. The histomorphologic parameter “closure of the apical foramen of the main root canal” was excluded from the groups with no apical patency because the maintenance of the cemental barrier in these groups prevents the identification of a main apical foramen. This exclusion was also considered in the statistical analysis.

The statistical analysis applied to the scores of both studied variables (apical foramen widening and type of sealer) showed that regarding new cementum formation, repair of cementum and bone resorption areas, presence of microorganisms, inflammatory cell infiltrate, and periodontal ligament conditions, significantly better healing of the chronic periapical lesions was observed when apical foramen widening was done (P = .05) and Sealer 26 was used (P = .05; Table II).

DISCUSSION

The findings of the present study showed that 6 months after the endodontic treatment, apical foramen widening and the type of endodontic sealer influenced the outcome of the healing of induced chronic periapical lesions in dogs’ teeth. The need of performing apical patency during endodontic treatment of root canals with necrotic and contaminated pulps remains an issue of discussion.¹⁷⁻¹⁹ For some authors, the debris compacted in the apical end of the root canal should be removed mechanically,^{20,21} whereas other authors believe that the debris can be removed with abundant irrigation and infection eliminated with the use of an antimicrobial intracanal medication.^{22,23} The results of an in vivo histologic study involving apical and periapical tissues following root canal therapy after different observation periods showed that the most favorable histologic conditions were obtained when the instrumentation and obturation remained at or short of the apical constriction. According to those authors, instrumentation and filling beyond the apical constriction results in a severe periapical inflammatory reaction, thus making it deleterious to the healing process.²⁴ The results of the present study showed that for both sealers, the apical patency by widening of the apical foramen favored healing. The divergence of results can be at-

tributed to the different protocols used in both studies, mainly the apical limit of obturation. It has been demonstrated that in teeth with chronic periapical lesions, microorganisms are present in apical delta ramifications, dentinal tubules, and cementoplasts.²⁵⁻²⁷ Therefore, performing apical widening of the cemental canal and apical foramen certainly contributed to obtaining better results, because it removed a greater amount of contaminated cementum and promoted a more favorable condition for the healing.

Although an increasing number of researchers have indicated the completion of the endodontic treatment in a single session, our option for using of a calcium hydroxide dressing was based on earlier histologic studies which showed better periapical healing when an intracanal dressing was used.^{25,26,28} The action of the calcium hydroxide dressing is believed to be related to its capacity to increase the pH of the root segment to a level that is incompatible with the survival of microorganisms.²⁹ It has been demonstrated that maximum alkalization of dentin occurs within 3-4 weeks after root canal filling with a calcium hydroxide paste.^{30,31} It is assumed that maximum alkalization might have been reached in all groups.

Regarding the type of endodontic sealer alone, Sealer 26 was superior to Endomethasone. This result is most likely due to the maintenance of an alkaline pH in the canals for a longer time as a result of the association of a calcium hydroxide dressing and a calcium hydroxide-based sealer.^{32,33} This hypothesis could also explain the similar results obtained for groups Sealer 26/no apical foramen widening and Endomethasone/apical foramen widening. It may be assumed that the lack of apical widening might have been compensated by the maintenance of an alkaline pH in the periapical region for an extended period induced by the calcium hydroxide-containing sealer. The good results obtained in the present study confirm the synergism between the calcium hydroxide-based intracanal dressing and root canal sealer, as demonstrated in a previous study (25).

Among the histomorphologic parameters evaluated in this study, the inflammatory infiltrate, new cementum formation, and apical PDL conditions are the most frequently used to evaluate the progression of periapical lesion healing.

Regarding the inflammatory infiltrate, considering all 4 experimental groups, chronic inflammation was present in 38 specimens (95%) and acute inflammation was observed in 26 specimens (65%). According to Orstavik and Mjör,³⁴ the presence of inflammation 6 months after the endodontic treatment may be related more to the persistence of the infection than to the toxicity of the root canal filling materials. In the present study, in all treated specimens, bacteria were identified

in 22 specimens (55%), with higher incidence in the groups in which apical foramen widening was not performed and acute inflammation was present. For this reason, although the Brown and Brenn staining may present limitations in the identification of microorganisms,³⁵ it is possible to assume that, 6 months after treatment, the relationship between inflammation and infection always applies to the acute inflammation. On the other hand, the presence of chronic inflammation with different intensities in 95% of the specimens also indicates that factors others than infection might have contributed, such as sealer biocompatibility and the presence of necrotic pulp tissue remnants in the apical ramifications which were not involved in the widening of the apical foramen.

Another event that also characterizes the progression of periapical lesion healing is the deposition of new apical cementum, mainly to repair areas of resorption resulting from apical periodontitis. In the present study, widening of the apical foramen allied to the use of a calcium hydroxide-based sealer produced the best results. It is likely that the release of calcium ions started with the dressing continued with Sealer 26,³³ thus maintaining the stimulus to new cementum formation for a longer time. This condition would also explain the occurrence of healing of all resorption areas observed in 50% of the root canals (with and without apical foramen widening) that were filled with Sealer 26, and in only 15% of the root canals (with and without apical foramen widening) that were filled with Endomethasone.

Other parameters that characterize the progression of the healing of a periapical lesion are the reduction of its size due to new bone and cementum formation and the organization of the apical PDL. It has been demonstrated that the thickness of the apical PDL in teeth of dogs aged 1-2 years ranges from ~100 to ~200 μm .^{36,37} Earlier studies using the same methodology as that of the present study for induction of periapical lesions showed that, after 6 months of exposure of the root canals of dogs' teeth to the oral cavity, the apical PDL thickness was $>800 \mu\text{m}$.^{25,26,38} In the present study, however, in all specimens of the 4 experimental groups, the apical PDL thickness was below this value, which suggests an ongoing healing process, though at different progression rates. It demonstrated that both studied variables (apical foramen widening and type of sealer) had a significant influence on the outcomes, with better results for the group in which apical foramen widening was done and the root canals were filled with Sealer 26. In this group, the apical PDL thickness had returned to normality in 40% of the specimens, which also determined a better organization. Therefore, based on the analysis of the studied histomorphologic

parameters that characterize the progression of the healing of chronic periapical lesions, it may be concluded that apical widening of the apical foramen and the use of a calcium hydroxide-containing sealer (Sealer 26) influenced positively the treatment outcomes. The association of both conditions (group 1) provided the most advanced periapical healing of chronic periapical lesions within the experimental period.

UNCITED REFERENCE

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