# Prevalence of different periapical lesions associated with human teeth and their correlation with the presence and extension of apical external root resorption

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## Abstract

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**Aim** The aim of this study was to determine the prevalence of various periapical pathologies and their association with the presence and extent of apical external inflammatory root resorption in human teeth.

**Methodology** One hundred and four root apices from extracted teeth with periapical lesions were examined. Semi-serial sections of soft tissue lesions were stained with HE. The lesions were classified as noncystic or cystic, each with different degrees of acute inflammation: 0, 1, 2 and 3, increasing in severity. The root apices were analysed by SEM. External root resorption was classified according to site, as periforaminal or foraminal, and the extension of the resorbed area graded in increasing area as 0, 1, 2 or 3.

**Results** Cysts accounted for 24.5% of the samples, 84% of which were associated with marked inflammation. The most prevalent diagnosis was noncystic periapical abscess with varying degrees of severity (63.7%). Periapical granuloma was not a frequent finding. SEM analysis showed that 42.2% of the root apices had periforaminal resorption extending over 50% of their circumference. When the foraminal resorption was evaluated, 28.7% had resorption affecting >50% of the periphery. Only 8.9% of the samples showed no periforaminal or foraminal resorption.

**Conclusions** In the sample of extracted teeth investigated, 24.5% of the periapical lesions were cysts. Most periapical lesions (84.3%) displayed acute inflammation, whether cystic or not. Periforaminal resorption was present in 87.3% of the cases, and foraminal resorption in 83.2%. Periforaminal and foraminal resorptions were independent entities. There was no association between external root resorption and the nature of the periapical lesions.

**Keywords:** apical root resorption, diagnosis, periapical pathology, SEM.

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## Introduction

Following pulp necrosis, the root-canal system encourages the colonization and proliferation of microbes (Estrela & Figueiredo 1999). The low intensity, chronic stimulus provided by bacteria and their products allows for the maintenance of inflammation in the periapical region (Yanagisawa 1980, Lin *et al.* 1984). Chronic periapical lesions bearing a proliferative character, represented by granulomas and periapical cysts, are the result of this process (Leonardo *et al.* 1998). The slow growth of these lesions results in bone resorption that is visible radiographically.

The precise nature of such lesions can only be determined histologically (Linenberg *et al.* 1964). However, true prevalence of each pathological conditon is unclear. Cystic lesions have been reported to account for between 3.2% (Nair 1987) and 54% (Priebe *et al.* 1954) of apical

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lesions, and granulomas for between 45% (Lalonde & Luebke 1968) and 96.8% (Nair 1987). Some of this may be explained by differences in sample source and histological methods.

A number of chemical mediators of inflammation, including the cytokynes IL-1α, IL-1β, TNFα, prostaglandins and LPS, seem to be related to the pathogenesis of periapical lesions (Schein & Schilder 1975, Schonfeld *et al.* 1982, Burchett *et al.* 1988, Wang & Stashenko 1993a,b). These substances may stimulate root resorption in the same way that they stimulate bone resorption (Hammarström & Lindkog 1992).

Although radiographic examination is an important resource in clinical diagnosis, it is rarely helpful in the diagnosis of small areas of external root resorption associated with teeth having apical periodontitis (Bhaskar & Rappaport 1971, Ferlini Filho 1999, Laux *et al.* 2000).

Irregular resorbed areas are frequently situated in sites that are not within the reach of root-canal instruments or medication and may act as niches for extraradicular bacterial colonization (Tronstad *et al.* 1990, Lomçali *et al.* 1996), besides causing technical problems for root-canal treatment (Delzangles 1989, Malueg *et al.* 1996).

There are few reports correlating periapical lesions with external inflammatory root resorption (Delzangles 1989, Bohne 1990, Vier & Figueiredo 2000). The aim of this study was to evaluate the prevalence of different periapical lesions as well as to evaluate the presence and extension of apical external resorption and its association with different categories of disease.

## Materials and methods

The sample comprised 113 extracted human teeth with visible periapical lesions. These extracted teeth were obtained from the Public Dental Services in the state of Rio Grande do Sul, Brazil, where endodontic or restorative treatment is not provided. Some of the teeth were symptomatic, others were removed because the crown was completely compromised by dental caries. Care was taken to include only teeth with whole and intact periapical lesions.

The specimens were stored at room temperature in a solution of formaldehyde (10%, w/v), and radiographed to exclude previous root-canal treatment or incomplete root formation. Fifteen teeth were discarded, leaving a final sample of 98 teeth, amongst which six presented two lesions on separate roots. A total of 104 apical lesions and 104 root apices were analysed.

The periapical lesions were gently removed from the root apices and labelled before processing. Semi-serial sections (0.5 μ at 0.5-mm intervals) of soft periapical lesions were stained with HE. The periapical lesions were classified as:

Noncystic lesions

- *Periapical granuloma*: Lesions predominantly infiltrated by lymphocytes, plasma cells and macrophages, with or without epithelial remnants (Nair *et al.* 1996), and covered by a capsule of collagen fibres. In these lesions, neutrophils were sparse forming no abscess microcavities or concentrated infiltrates.
- *Periapical abscess*: Lesions with a distinct collection of neutrophils in the interior of a previously existent granuloma (Nair *et al.* 1996). These were further categorized as 1 = abscess cavity occupying up to one-third; 2 = 1/3–2/3; 3 ≥ 2/3 of the total area of the visualized lesion in the histological sections.

Cystic lesions

- *Periapical cyst*: Lesions with a layer of stratified squamous epithelium along a surface of sufficient quantity of conjunctive tissue to indicate a delineated cavity and surrounded by a slight fibrous capsule (Patterson *et al.* 1964, Lalonde & Luebke 1968, Lalonde 1970, Nobuhara & Del Rio 1993, White *et al.* 1994).
- *Abscessed periapical cyst:* Cysts containing pus-filled cavities were classed as 1, 2 or 3 as above.

The histological sections were analysed by two blinded and previously calibrated observers, using 32, 100 and 400× magnification (Zeiss microscope, Thornwood, USA).

The diagnosis of each lesion was determined by considering all the histological sections. The presence of epithelium delineating a pathological cavity in one or more sections of a lesion characterized it as a periapical cyst. Classification of abscess severity was based on the section showing its largest dimension. Thereafter, the periapical lesions were grouped in two subgroups:

- noncystic lesions: A1 – with absence or with a small abscess (periapical granuloma and abscess degree 1); A2 – with advanced abscess (periapical abscess degrees 2 and 3).
- cystic lesions: B1 – with absence or with a small abscess (periapical cyst and abscessed cyst degree 1); B2 – with advanced abscess (abscessed cysts degree 2 and 3).

The apical portion of the root was cut perpendicular to the long axis with carborundum disc to favour placement on stubs. The root apices were submerged in a solution of 2.5% sodium hypochlorite for 3 h, dehydrated in an ascending sequence of alcohols (70, 90 and 99.96%, for 5 h in each), attached to the stubs with the apex upwards, and sputtered (Balzers, Liechtensten) with gold palladium, to a thickness of 150 Å. Scanning electron

microscopy was conducted using a Philips XL 20 (Eindhmoven, Netherlands) microscope, operating at 15 Kv. The areas that surrounded the apical foramina of the apices were imaged at 100×. The apical foramen occupied the central region of the video screen and was totally surrounded by the root. If a root presented more than one apical foramen, more than one image was obtained, so as to guarantee the analyses of all the foramina. SEM images were analysed separately by two blinded and previously calibrated observers.

The dental apices were classified depending on the presence or absence of external apical resorption, as well as to its extent:

*Periforaminal resorption*: Periforaminal resorption was defined as the area of resorption not comprising the outline of the foramen, but the surrounding area. The degrees of severity 0–3 were employed when there was absence of resorption, resorption of up to 1/4, from 1/4 to 1/2 and in more than 1/2 of the area that surrounded or circumscribed the apical foramen, respectively.

*Foraminal resorption*: Defined as the resorption within the outline or the perimeter of the foramen. The degrees of severity 0–3 were employed when there was absence of resorption, resorption of up to 1/4, from 1/4 to 1/2 and in more than 1/2 of the area of the outline or the perimeter of the apical foramen, respectively.

Whenever a tooth presented with two or more apical foramina or had fused roots, the degree of final resorption measured was the foramen where the resorption was most severe.

Kappa coefficient was employed to evaluate the degree of agreement amongst the examiners, both in diagnosing the periapical lesions and in the presence and extension of periforaminal and foraminal external resorption.

The ANOVA statistical test was used to evaluate the correlation between the histopathological diagnosis of the periapical lesions and the presence and extension of the external apical root resorption, followed by the Duncan test of multiple comparisons. Due to the ordinal nature of the data, the Kruskal–Wallis ANOVA was also used. The frequencies observed in the groups were compared using the Chi-square test.

## Results

### Histopathology

The results are presented in Tables 1–5. Analysis was completed in a total of 102 specimens, as two were lost during preparation. The degree of agreement amongst the observers concerning the diagnosis of the

**Table 1** Prevalence of different periapical lesions

| Periapical lesion | f | % |
|---|---|---|
| Noncystic lesions with absence or with a small degree of abscess | 12 | 11.8 |
| Noncystic lesions with high degree of abscess | 65 | 63.7 |
| Cystic lesions with absence or with a minimum degree of abscess | 4 | 3.9 |
| Cystic lesions with high degree of abscess | 21 | 20.6 |
| Total | 102 | 100.0 |

**Table 2** Periapical lesions of minor and major severity of abscess

| Type of lesion | f | % |
|---|---|---|
| A1 + B1 | 16 | 15.7 |
| A2 + B2 | 86 | 84.3 |
| Total | 102 | 100.0 |

A1–noncystic lesions with absence or with a small degree of abscess.
A2–noncystic lesions with high degree of abscess.
B1–cystic lesions with absence or with a minimum degree of abscess.
B2–cystic lesions with high degree of abscess.

**Table 3** Extension degrees of periforaminal and foraminal resorption of the dental apexes

| | Resorption | | | |
|---|---|---|---|---|
| | Periforaminal (PR) | | Foraminal (FR) | |
| Degree of resorption | f | % | f | % |
| 0 | 13 | 12.7 | 17 | 16.8 |
| 1 | 15 | 14.7 | 25 | 24.8 |
| 2 | 31 | 30.4 | 30 | 29.7 |
| 3 | 42 | 42.2 | 29 | 28.7 |
| Total | 102 | 100.0 | 101* | 100.0 |

*In one specimen, it was not possible to perform a classification of the foraminal resorption.

**Table 4** Distribution of degrees of periforaminal and foraminal resorption combined

| | Foraminal (FR) | | | | |
|---|---|---|---|---|---|
| Periforaminal (PR) | 0 | 1 | 2 | 3 | Total |
| 0 | 9 | 2 | 1 | 1 | 13 |
| 1 | 3 | 5 | 5 | 2 | 15 |
| 2 | 3 | 10 | 15 | 3 | 31 |
| 3 | 2 | 8 | 9 | 23 | 42 |
| Total | 17 | 25 | 30 | 29 | 101 |

**Table 5** Median (p25–75) of the degrees of periforaminal and foraminal resorption according to the different periapical lesions

| | | Resorption | | | |
|---|---|---|---|---|---|
| | | Periforaminal (PR) | | Foraminal (FR) | |
| Periapical Lesion | *n* | md | p25–p75 | md | p25–p75 |
| A1 | 12 | 2.0 | 2.0–3.0 | 2.0 | 1.5–2.0 |
| A2 | 65* | 2.0 | 1.0–3.0 | 2.0 | 1.0–3.0 |
| B1 | 4 | 2.0 | 0.5–3.0 | 1.0 | 0.5–2.0 |
| B2 | 21 | 2.0 | 1.0–2.0 | 1.0 | 0.0–2.0 |

Periforaminal: $P = 0.227$; foraminal: $P = 0.163$.
A1–noncystic lesions with absence or with a small degree of abscess.
A2–noncystic lesions with high degree of abscess.
B1–cystic lesions with absence or with a minimum degree of abscess.
B2–cystic lesions with high degree of abscess.
*In one specimen, it was not possible to perform a classification of the foraminal resorption.

periapical lesions gave a Kappa coefficient of 0.96 (IC 95% = 0.82–1.0).

Periapical granuloma was not a common finding. A1 lesions (granuloma and periapical abscess score 1) comprised only 11.8% of the sample (Table 1). The most prevalent histological diagnosis was the lesions classified as A2 (noncystic periapical abscess scores 2 and 3) (Fig. 1), which comprised 63.7% of the sample (Table 1).

Periapical cysts represented 24.5% of the sample. Just four (3.9%) periapical cysts presented with no or small abscess cavities (Fig. 2). Amongst the 25 cysts analysed, 21 (84%) had large abscess cavities. The abscessed areas were associated with an epithelial component resulting in disorganization and discontinuity. The greater the severity of the abscess, the bigger the epithelial disintegration observed. In other cases, the cavities of the

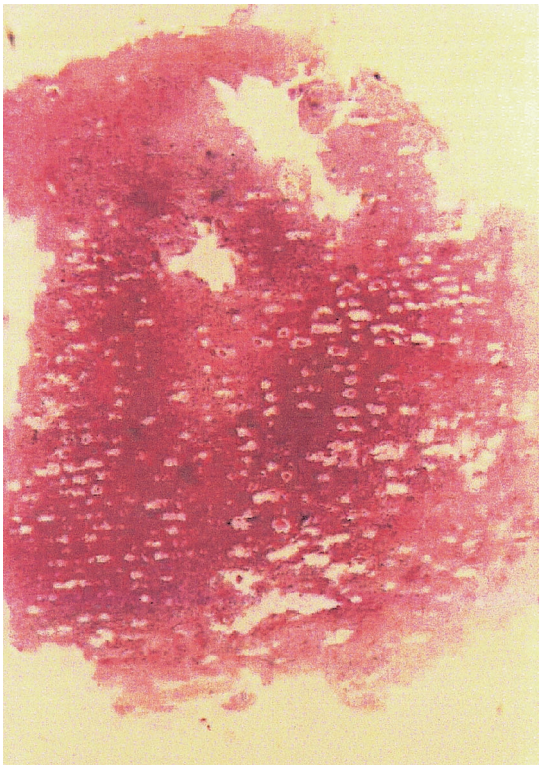

**Figure 1** Periapical abscess degree 3 (14×).

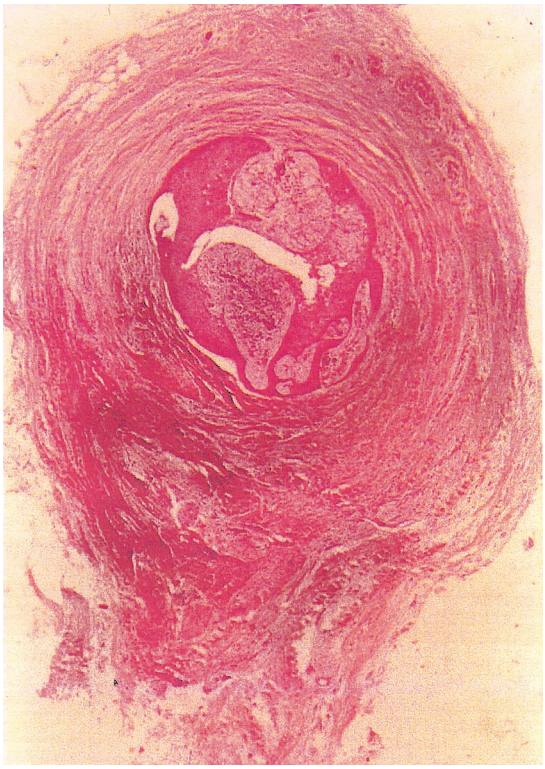

**Figure 2** Periapical cyst (cystic lesion with the absence of abscess microcavities) (32×).

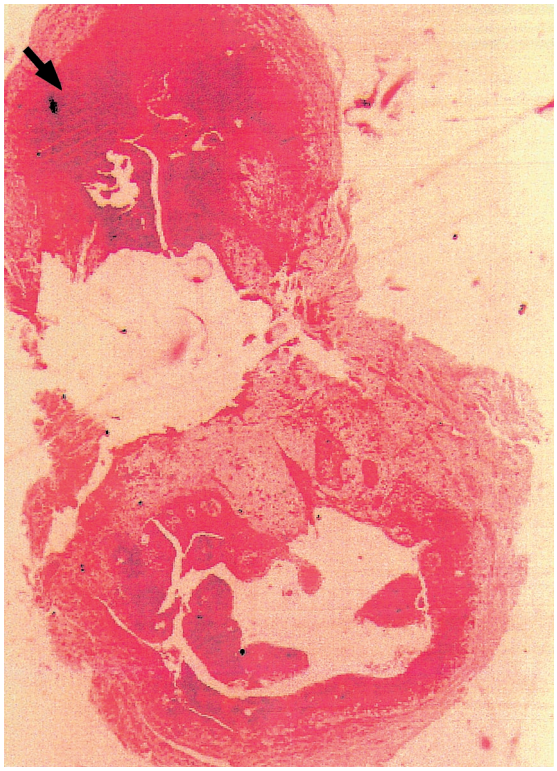

**Figure 3** Abscessed cyst degree 3, with abscessed area adjacent to the epithelial component (14×).

abscesses were situated mainly on the connective tissue adjacent to the epithelial layer (Fig. 3).

There were cases where focal areas of neutrophils were absent within the cyst epithelium in one histological section, but the following section often revealed a defined abscess in that site. Disregarding the cystic characteristic of the lesions and by just analysing the presence of an abscess, we were able to observe that 86 lesions (84.3%) had abscess cavities that occupied a large area. Just 16 lesions (15.7%) were free from or contained only microcavities of acute inflammatory cells (Table 2).

### Resorption

Only 12.7% of the samples were free of periforaminal resorption and 16.8% showed integrity of the foraminal surroundings (Table 3). From all the samples, 72.6% had periforaminal resorption, reaching more than 1/4th of the area around the foramen (Fig. 4). Moreover, in 42.2% of these cases, the resorption included more than half of this area (Fig. 5).When foraminal resorption occurred, 58.4% of the samples were affected in more than 1/4 of their perimeter (Fig. 6), and 28.7% in more than half (Fig. 7). Just 8.9% of the samples had no resorption (Table 4).

From Table 4, it is clear that the pattern of periforaminal resorption did not depend on the pattern of foraminal resorption or vice-versa. An apex may present with substantial periforaminal resorption, without alteration to the foramen. The apical foramen may be resorbed, even if the zone that surrounds it had an intact cementum structure. However, the resorption could occur both in a periforaminal and foraminal location and to different degrees.

By associating the presence of periforaminal and foraminal resorption, we were able to perceive that 49.5% of the apices had resorption in more than 1/4th of the

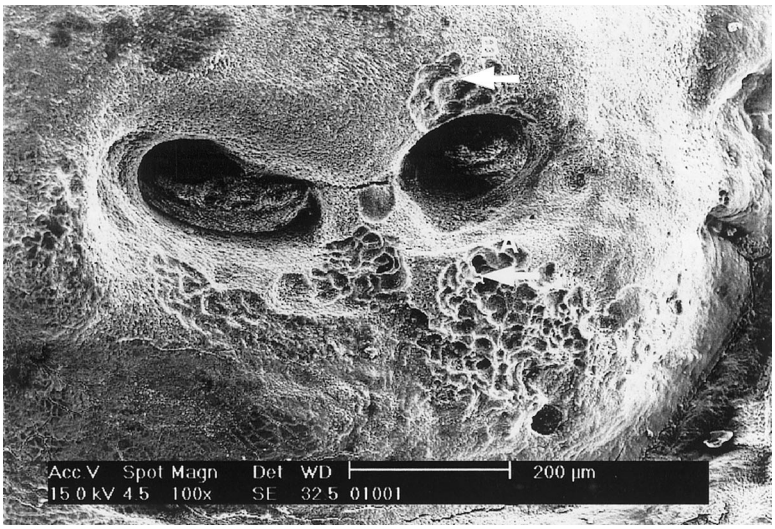


**Figure 4** Apex with periforaminal resorption involving 1/4–1/2 of the area. Pronounced areas of lacunae (A). Interlacunae crests were not much evident (B) (PR: 2; FR: 2; lesion: B2) (100×).

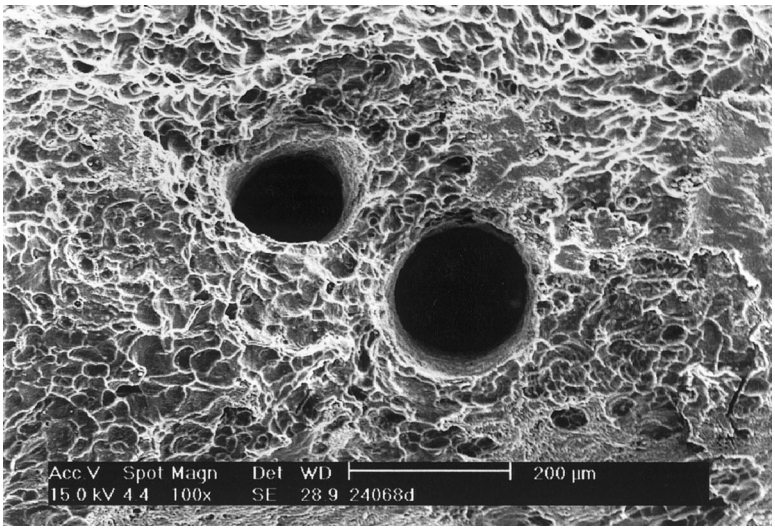


**Figure 5** Apex with periforaminal resorption involving more than half of the radicular surface examined. The resorption displayed a honeycomb aspect (PR: 3; FR: 3; lesão: A2) (100×).

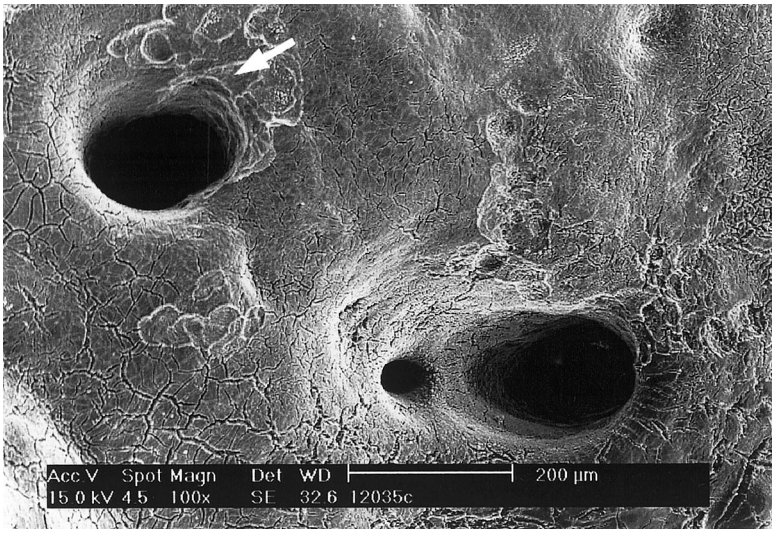


**Figure 6** Apex with foraminal resorption involving 1/4–1/2 of the perimeter with a not well defined, or even absent outline of the interlacunae crests (PR: 1; FR: 2; lesion: B2) (100×).

examined area, and in more than half in 22.8% of cases (Table 4)(Fig. 7). The lacunae of resorption were similar to the Howship type, showing a circular shape of different sizes. The combination of various lacunae resembled the characteristic aspect of honeycombs (Fig. 5). Generally, the areas of resorption were superimposed, projecting pronounced margins (Fig. 4). However, in some cases, due to the fusion of the lacuna, or due to its shallowness, the interlacunae crests became less obvious or even absent (Figs 4 and 6).

The resorbed areas resulted in the presence of one or two isolated lacunae, surrounded by an integral cementum surface or by a set of interconnected gaps that were occasionally related to other resorption zones. Sometimes, the apex had fused areas of resorption, and was totally deprived of intact cementum (Fig. 5). There were cases with evidence of exposed dental tubules (Fig. 8), demonstrating sites of increased resorption.

The foraminal resorption viewed by SEM followed two peculiar characteristics. In some apices, there was deformation of the original foramen outline, due to deep resorption, though in very specific places (Fig. 9). In others, although the apical foramen had not suffered visible morphological alterations, it was possible to

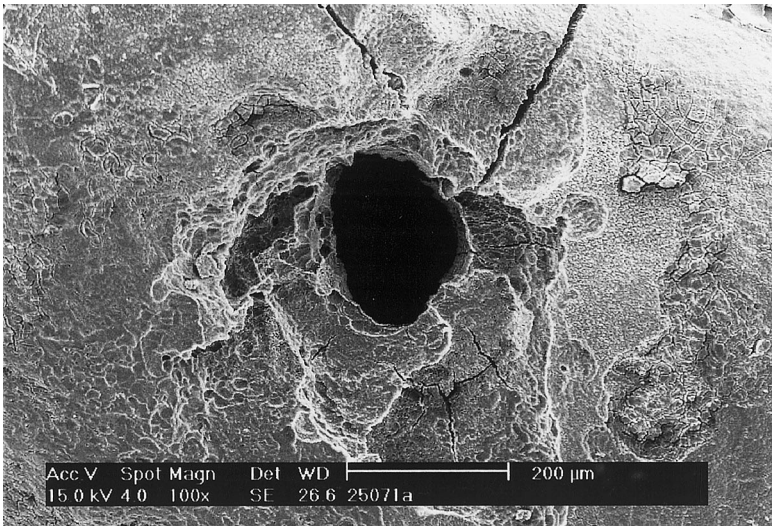


**Figure 7** Apex with periforaminal and foraminal resorption involving more than half of the radicular surface examined (PR: 3; FR: 3; lesion: A2) (100×).

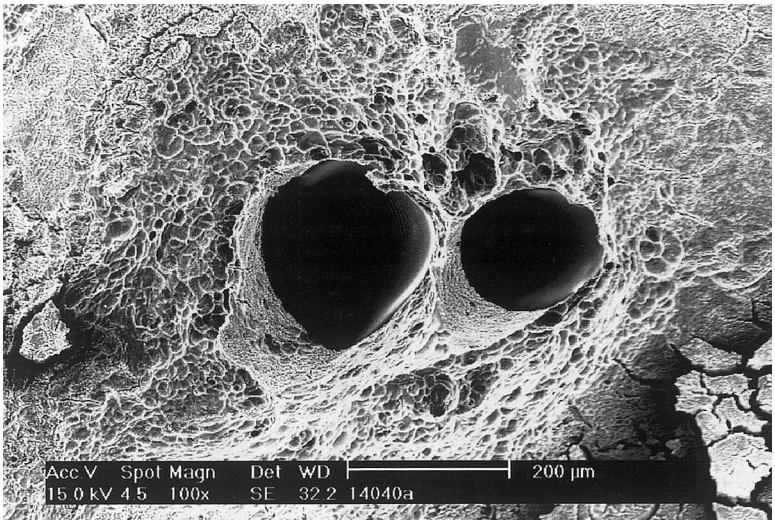


**Figure 8** Apex with extensive periforaminal and foraminal resorption exposing dentinal tubules (PR: 3; FR: 2; lesion: A2) (100×).

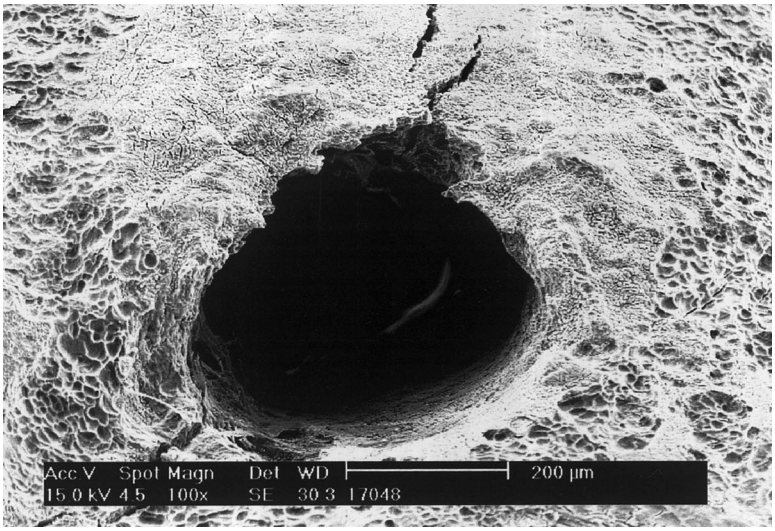


**Figure 9** Apex with foraminal resorption with morphological alterations of the apical contour (PR: 3; FR: 3; lesion: A2) (100×).

observe shallow resorption gaps, surrounding the majority of its outline (Figs 5 and 8).

According to Table 5, we can verify that there was no statistically meaningful difference between the type of periapical lesion and the degree of periforaminal and foraminal resorption at the root apex.

## Discussion

Many teeth with apical periodontitis are believed to show some degree of external periapical resorption, but few studies have related resorption with the nature of the apical lesion (Delzangles 1989, Bohne 1990, Vier & Figueiredo 2000). This study sought to address this issue by correlating periapical pathology with the presence and extension of periforaminal and foraminal resorption.

The presence of periapical pathosis was determined by direct visualization of root apices (Linenberg *et al.* 1964, Simon 1980, Garrocho & Antonio Neto 1984, Nair 1987, Bohne 1990, Nair *et al.* 1996). Radiographic examination was not conducted prior to tooth extraction. The presence of a periapical radiolucency does not necessarily indicate periapical inflammation, other causes may include apical scar tissue (Nair *et al.* 1999). Conversely, a periapical inflammatory reaction of endodontic origin may exist without being visible radiographically (Bender & Seltzer 1961).

Semi-serial sections were performed on the specimens, since abscess cavities or even the epithelial lumen of a cystic cavity may be away from the centre of the specimen or from the place elected for randomized sections (Garrocho & Antonio Neto 1984), a fact which could make it difficult to reach the final diagnosis. The most accurate analysis is achieved from serial sectioning of intact lesions.

We classified abscesses as lesions without epithelium in the body of the lesion and with a massive accumulation of neutrophils. The lesions diagnosed as periapical granulomas exhibited neutrophils, though not forming distinct arrangements or in a degenerating state. We classified cysts according to Patterson *et al.* (1964), Lalonde & Luebke (1968), Lalonde (1970), Nobuhara & del Rio (1993) and White *et al.* (1994), because when cysts become infected the epithelium may not be complete or intact, but disintegrated in some areas.

After a thorough and individual analysis of each histological section, we agreed with Linenberg *et al.* (1964), who demonstrated transitional stages between one type of lesion and another when analysing various histological sections of the same lesion.

Differences in histological criteria create difficulty in comparing similar studies (Langeland *et al.* 1977, Spatafore *et al.* 1990, Nobuhara & del Rio 1993). Our study, for example, demonstrated an unusually low prevalence of periapical granuloma and high prevalence of noncystic abscesses. This may be due to the sample, which consisted of teeth extracted in the Public Oral Services in poor areas in southern Brazil, where the presence of symptoms is one of the main reasons for dental visits.

The prevalence of 24.5% of cystic lesions is in accord with previous reports (Linenberg *et al.* 1964, Langeland *et al.* 1977, Nobuhara & del Rio 1993, White *et al.* 1994). However, when evaluating other studies with similar methodology, i.e. lesions associated with the apices of extracted teeth, the results were higher than the 17 and 15% of the cysts diagnosed by Simon (1980) and Nair *et al.* (1996) respectively. The results were closer to the 28% reported by Linenberg *et al.* (1964), whose samples included material curetted from extraction sockets and lesions associated with extracted teeth.

This study also evaluated the extension of resorption over the apical root surface. The results are difficult to correlate with many others who simply reported the presence of resorption on the root surface (Simon *et al.* 1981, Hess *et al.* 1983, Laurent-Maquin *et al.* 1986, Delzangles 1989, Bohne 1990, Lomçali *et al.* 1996, Bonifácio *et al.* 2000).

From the methodology employed, 8.9% of specimens had no periforaminal or foraminal resorption, despite pulp necrosis and periapical lesions. These results are in agreement with those of Henry & Weinmann (1951) and Ferlini Filho (1999) who reported that 10% of their specimens had no resorption.

Apical periodontitis is amongst the causes of progressive inflammatory resorption cited by Soares & Goldberg (2001). Nevertheless, it is impossible to determine the influence of other factors that might potentiate resorption such as occlusal trauma. We had no access to detailed medical or clinical data to control for such parameters.

The apical resorption observed in the present study did not differ amongst the various periapical pathological conditions, since all of them exhibited a common feature, i.e. the presence of inflammation. This is an important aspect to consider in the histological description of these cystic and noncystic lesions, independent of the degree of abscess. Both constitute a continuous and variable aspect of the same phenomenon, inflammation. The physical presence of a periapical lesion could promote root resorption in addition to bone lysis, as it makes room for its organization and growth, independently of

its histological classification. Thus, the results of the present study differ from those of Delzangles (1989) and Vier & Figueiredo (2000), who included a small number of cystic samples and used less refined histological sectioning.

Normally, the failure of root-canal treatment is related to the persistence of infection in the root-canal system (Sjögren *et al.* 1997, Sundqvist *et al.* 1998). The presence of root resorption in teeth with periapical lesions is important for infection control since these areas are niches for bacteria. Moreover, the apical limit of instrumentation may be altered in teeth with widely resorbed apices, since the cementum–dentine junction at the constriction can be missing (Delzangles 1989, Malueg *et al.* 1996). In such circumstances, sealing the canal may be difficult and overfilling is likely.

Bacteria may also be found in the external surface of the root, forming a periapical biofilm (Leonardo & Silva 1998), thus confirming the importance of appreciating the existence of periforaminal root resorption. No bacterial biofilms were, however, demonstrated in this study.

## Conclusions

On the basis of our study of extracted human teeth, we conclude that:

- Cystic lesions accounted for 24.5% of chronic periapical lesions;
- The majority of chronic periapical lesions (84.3%), whether cystic (20.6%) or noncystic (63.7%), had large collections of acute inflammatory cells;
- Periforaminal and foraminal resorptions were present in 87.3 and 83.2% of roots associated with periapical lesions;
- The pattern of periforaminal resorption was independent of the pattern of foraminal resorption;
- There was no correlation between the histopathological diagnosis of the periapical lesion and the presence and extension of apical external root resorption.

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## References

Bender I, Seltzer S (1961) Roentgenographic and direct observation of experimental lesions in bone: I. *Journal of the American Dental Association* **62**, 152–60.

Bhaskar S, Rappaport H (1971) Histologic evaluation of endodontic procedures in dogs. *Oral Surgery, Oral Medicine and Oral Pathology* **31**, 526–35.

Bohne W (1990) Light and ultrastructural studies of human chronic periapical lesions. *Journal of Oral Pathology and Medicine* **19**, 215–20.

Bonifácio K, Leonardo M, Rossi M, Silva L, Ito I (2000) MEV– avaliação do ápice root de dentes de humanos com e sem vitalidade pulpar. (Abstract A217) *Pesquisa Odontontológica Brasileira* **14** (Suppl.), 87.

Burchett S, Weaver W, Westall J, Larsen A, Kronheim S, Wilson C (1988) Regulation of tumor necrosis factor/cachectin and IL-1 secretion in human mononuclear phagocytes. *Journal of Immunology* **140**, 3473–81.

Delzangles B (1989) Scanning electron microscopic study of apical and intracanal resorption. *Journal of Endodontics* **15**, 281–5.

Estrela C, Figueiredo J (1999*) Endodontia: princípios biológicos e mecânicos*, 1st edn. São Paulo, SP, Brazil: Artes Médicas, pp. 191–245.

Ferlini Filho J (1999) Estudo radiográfico e microscópico das reabsorções radiculares na presença de periodontites apicais crônicas (microscopia ótica e eletrônica de varredura). Thesis. São Paulo, Brazil: Universidade de São Paulo.

Garrocho A, Antonio Neto M (1984) Da relação entre a cavidade do cisto radículo dentário com o forame apical. *Arquivos Do Centro de Estudos Da Faculdade de Odontologia Da UFMG* **21**, 95–100.

Hammarström L, Lindkog S (1992) Factors regulating and modifying dental root resorption. *Proceedings of the Finnland Dental Society* **88** (Suppl. 1), 115–23.

Henry J, Weinmann J (1951) The pattern of resorption and repair of human cementum. *Journal of the American Dental Association* **42**, 270–90.

Hess J, Culieras M, Lamiable N (1983) A scanning electron microscopic investigation of principal and accessory foramina on the root surfaces of human teeth: thoughts about endodontic pathology and therapeutics. *Journal of Endodontics* **9**, 275–81.

Lalonde E (1970) A new rationale for the management of periapical granulomas and cysts: an evaluation of histhopathological and radiographic findings. *Journal of the American Dental Association* **80**, 1056–9.

Lalonde E, Luebke R (1968) The frequency and distribution of periapical cysts and granulomas. *Oral Surgery, Oral Medicine, Oral Pathology* **25**, 861–8.

Langeland K, Block R, Grossman L (1977) A histopathologic and histobacteriologic study of 35 periapical endodontic surgical specimens. *Journal of Endodontics* **3**, 8–23.

Laurent-Maquin D, Blocquaux Verchere M, Bouthors S (1986) Modifications morphologiques radiculaires de dents atteintes d'une lésion périapicale. Étude au microscope électronique à balayage. *Revue d'Odonto-Stomatologie* **15**, 379–84.

Laux M, Abbott P, Pajarola G, Nair P (2000) Apical inflammatory root resorption: a correlative radiographic and histological assessment. *International Endodontic Journal* **33**, 483–93.

Leonardo M, Leal J, Lia R, Martins J (1998) Filosofia do tratamento de canais radiculares Necropulpectomia: Conceituação. In: Leonardo M, Leal J, eds. *Endodontia: Tratamento de Canais Radiculares*, 3rd edn. São Paulo, Brazil: Panamericana, pp. 127–58.

Leonardo M, Silva L (1998) Medicação tópica entre sessões, 'curativo de demora' em biopulpectomia e necropulpectomia I e II. In: Leonardo M, Leal J, eds. *Endodontia: Tratamento de Canais Radiculares*, 3rd edn. São Paulo, Brazil: Panamericana, pp. 491–534.

Lin L, Shovlin F, Skribner J, Langeland K (1984) Pulp biopsies from the teeth associated with periapical radiolucency. *Journal of Endodontics* **10**, 436–48.

Linenberg W, Waldron C, Delaune G Jr (1964) A clinical roentgenographic and histhopathologic evaluation of periapical lesions. *Oral Surgery, Oral Medicine and Oral Pathology* **17**, 467–72.

Lomçali G, Sen B, Çançaya H (1996) Scanning electron microscopic observations of apical root surfaces of teeth with apical periodontitis. *Endodontics and Dental Traumatolology* **12**, 70–6.

Malueg L, Wilcox L, Johnson W (1996) Examination of external apical root resorption with scanning electron microscopy. *Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology, Endodontics* **82**, 89–93.

Nair P (1987) Light and electron microscopic studies of root canal flora and periapical lesions. *Journal of Endodontics* **13**, 29–39.

Nair P, Pajarola G, Schroeder H (1996) Types and incidence of human periapical lesions obtained with extracted teeth. *Oral Surgery, Oral Medicine and Oral Pathology* **81**, 93–102.

Nair P, Sjögren U, Figdor D, Sundqvist G (1999) Persistent periapical radiolucencies of root-filled human teeth, failed endodontic treatments, and periapical scars. *Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology, Endodontics* **87**, 617–27.

Nobuhara W, Del Rio C (1993) Incidence of periroot pathoses in endodontic treatment failures. *Journal of Endodontics* **19**, 315–8.

Patterson S, Shafer W, Healey H (1964) Periapical lesions associated with endodontically treated teeth. *Journal of the American Dental Association* **68**, 191–4.

Priebe W, Lazansky J, Wuehrmann A (1954) The value of the roentgenographic film in the differential diagnosis of periapical lesions. *Oral Surgery, Oral Medicine and Oral Pathology* **7**, 979–83.

Schein B, Schilder H (1975) Endotoxin content in endodontically involved teeth. *Journal of Endodontics* **1**, 19–21.

Schonfeld S, Greening A, Glick D, Frank A, Simon J, Herles S (1982) Endotoxic activity in periapical lesions. *Oral Surgery, Oral Medicine and Oral Pathology* **53**, 82–7.

Simon J (1980) Incidence of periapical cysts in relation to the root canal. *Journal of Endodontics* **6**, 845–8.

Simon J, Yonemoto G, Bakland L (1981) Comparison of cellular cementum in normal and diseased teeth – a scanning electron microscopic study. *Journal of Endodontics* **7**, 370–5.

Sjögren U, Figdor D, Persson S, Sundqvist G (1997) Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis. *International Endodontic Journal* **30**, 297–306.

Soares I, Goldberg F (2001) *Endodontia: Técnica E Fundamentos*, 1st edn. Porto Alegre, Rio Grande do Sul, Brazil: Artes Médicas Sul. pp. 339–68.

Spatafore C, Griffin J, Keyes G, Wearden S, Skidmore A (1990) Periapical biopsy report: an analysis over a 10-years period. *Journal of Endodontics* **16**, 239–41.

Sundqvist G, Figdor D, Persson S, Sjögren U (1998) Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative re-treatment. *Oral Surgery, Oral Medicine and Oral Pathology* **85**, 86–93.

Tronstad L, Barnett J, Cervone F (1990) Periapical bacterial plaque in teeth refractory to endodontic treatment. *Endodontics and Dental Traumatolology* **6**, 73–7.

Vier F, Figueiredo J (2000) Morphologic analysis of apical resorption on human teeth with periapical lesions. *Endodontics Clinical Practice, Education and Research* **2**(3). On-line publication of the University of São Paulo: http://www.usp.br

Wang C, Stashenko P (1993a) Characterization of bone-resorbing activity in human periapical lesions. *Journal of Endodontics* **19**, 107–11.

Wang C, Stashenko P (1993b) The role of interleukin–1 alpha in the pathogenesis of periapical bone destruction in a rat model system. *Oral Microbiology and Immunology* **8**, 50–6.

White S, Sapp P, Seto B, Mankovich N (1994) Absence of radiometric differentiation between periapical cysts and granulomas. *Oral Surgery, Oral Medicine and Oral Pathology* **78**, 650–4.

Yanagisawa S (1980) Pathologic study of periapical lesions 1. Periapical granulomas: clinical, histopathologic and immunohistopathologic studies. *Journal of Oral Pathology* **9**, 288–300.