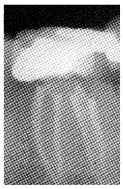

CLINICAL ARTICLE

Prognosis of large cyst-like periapical lesions following nonsurgical root canal treatment: a clinical review

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Abstract

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Aim To evaluate clinically and radiographically the long-term clinical outcome of nonsurgical root canal treatment using calcium hydroxide in teeth with large cyst-like periapical lesions.

Summary A total of 42 mature anterior teeth with large periapical lesions ranging in size from 7 to 18 mm in diameter were included in this study. Fluid samples from the lesions contained cholesterol crystals, which were identified by light microscopy. After adequate draining, the teeth were treated endodontically using calcium hydroxide as the intracanal medicament. All cases were followed up for a period of 2–10 years. Complete healing was observed in 73.8% and incomplete healing in a further 9.5% of cases.

Key learning points

- Root canal treatment using calcium hydroxide as an antibacterial dressing was successful in healing large cyst-like periapical lesions.
- This study suggests that the size of a periapical lesion is not a major determining factor in the decision to perform conventional root canal treatment or surgical removal of the lesion. Even large cyst-like periapical lesions containing cholesterol crystals can heal following nonsurgical root canal treatment.

Keywords: calcium hydroxide, cholesterol crystal, cyst, endodontic therapy, periapical healing.

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Introduction

Most periapical lesions (>90%) can be classified as dental granuloma, radicular cyst or abscess (Bhaskar 1966, Lalonde & Luebke 1968). It is generally accepted that periapical

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lesions cannot be differentially diagnosed as either radicular cysts or apical granulomas based on radiographic evidence alone (Bhaskar 1966, Lalonde 1970), although there is a trend towards increased incidence of cysts among larger lesions (Lalonde 1970, Natkin *et al.* 1984).

The epithelial lining of radicular cysts is thought to arise from epithelial cell rests of Malassez in the periodontal ligament and is believed to proliferate as a result of inflammation, usually following death of the pulp. The incidence of radicular cysts is reported to be highest among patients in their third decade of life and greater among men than women (Bhaskar 1966, Shear 1992). They have particularly high incidence in the maxillary anterior region, presumably as a result of trauma (Shear 1992).

There are two distinct categories of radicular cysts namely, those containing cavities completely enclosed by epithelial lining, true cysts, and those containing epithelial-lined cavities that are open to the root canals (Simon 1980, Nair *et al.* 1996). The later was originally described as a 'bay cyst' (Simon 1980), and has been newly designated as a 'periapical pocket cyst' (Nair *et al.* 1996).

There has also been a prolonged debate about the management of large cystic lesions, with some maintaining that true cysts can only be successfully treated by surgical means (Natkin *et al.* 1984, Nair *et al.* 1993). However, endodontists believe that a large majority of cysts will heal after nonsurgical root canal treatment (Bhaskar 1972, Morse *et al.* 1975). This notion rests solely on deductive logic. Success rates of up to 85% have been reported after endodontic treatment of teeth with periapical lesions (Sjögren *et al.* 1990, Çalışkan & Şen 1996). As it has been claimed that more than 40% of periapical lesions are radicular cysts, many of these lesions must be responding to root canal treatment (Lalonde & Luebke 1968, Bhaskar 1972).

Although reports have described the possible healing mechanism of periapical cystic lesions when treated by nonsurgical means (Bender 1972, Bhaskar 1972) there is only one prospective clinical study that investigated the healing response of cystic lesions (as determined by electrophoretic analysis of withdrawn fluid) after nonsurgical root canal treatment (Morse *et al.* 1975).

The purpose of the present study was to evaluate the long-term outcome of nonsurgical root canal treatment using calcium hydroxide in teeth with large cyst-like periapical lesions.

Materials and methods

The material consisted of 70 teeth with large periapical lesions in 50 patients referred for specialist endodontic treatment at the Department of Endodontics, Ege University between 1991 and 2001. Of these, 42 mature permanent anterior teeth with large periapical lesions in 32 adults patients (17 men, 15 female) were selected for this study because their fluid samples from the lesions revealed the presence of cholesterol crystals following microscopic examination. The aetiology of apical periodontitis, and the localization of the teeth were recorded. The patient's medical histories were all noncontributory. No previous endodontic therapy had been performed on any of the teeth. Periapical radiographs demonstrated large radiolucent lesions with well-defined margins around the apices of all the teeth. The initial diameter of the periapical radiolucent lesions varied from 7 to 18 mm. Twenty-six teeth had signs and symptoms with pain, tenderness to percussion, localized intraoral swelling and/or facial swelling and mobility while the remaining teeth were asymptomatic. All the cases were treated by one operator using a standardized technique.

After access cavity preparation a clear, straw-coloured fluid exuded from the canals of all but six teeth. These six were trephinated, using size 15 K-files beyond the apical

foramen in an attempt to secure drainage into the canal as it had not occurred spontaneously. Aspiration of this fluid from inside root canal was made directly into a syringe with a 20 or 22-gauge needle. Drainage of the lesion took from 4 days to a month. In most cases drainage was performed daily on two to four subsequent appointments until discharge through the canal ceased. The access cavities were sealed with zinc oxide-eugenol cement (Austenal, Harrow, UK) after drainage. Long-term drainage of this type was performed from 15 days to 1 month in six cases because of lasting and substantial discharging exudate from the canals. After daily draining within the first week, these six cases were examined weekly and in each visit the canals were debrided, dried and sterile paper points were placed and then the access cavities were sealed with zinc oxide-eugenol cement. Calcium hydroxide was not applied to the canal until active drainage ceased. No antibiotics were prescribed except in six cases with extraoral swelling.

When the pain and the swelling subsided and drainage ceased, the root canals were finally instrumented by a conventional step-back technique using K-type files, and copious irrigation with 5.25% sodium hypochlorite under rubber dam isolation. After drying with sterile paper points, calcium hydroxide paste (calcium hydroxide and barium sulphate powder in a ratio of 8 : 1 mixed with glycerine as a vehicle) was applied to the canal with spiral filler. The paste was further condensed using sterile cotton pellets before sealing the coronal access. Calcium hydroxide paste was unintentionally extruded into the periapical lesions of six teeth. The calcium hydroxide dressing was changed twice at 3-week intervals and maintained for 3 months.

After the removal of the dressing by irrigation with 5.25% NaOCl and reaming motion with a K-type file, the root canals were obturated with Diaket (Espe GmbH, Seefeld, Germany) and gutta-percha (Hygenic, Akron, OH, USA) using a cold lateral condensation technique. Following root canal obturation, the teeth were permanently restored. The patients were generally recalled at 3-month intervals, during the first year and subsequently at 6- or 12-month intervals aiming for a 2–10-year postoperative follow-up period to assess the stage of periapical healing. All radiographs were obtained by a long-cone technique with standardized exposure times and manually processed by the same operator. The largest diameter of the lesions was measured on each radiograph by a ruler. The treatment was considered to be successful (complete healing) when the tooth was found to be clinically symptomless and the radiograph showed complete disappearance of the preexisting radiolucency. The cases showing a decrease in size of the periapical radiolucency or presenting a scar tissue were placed in the incomplete healing category. If expansion or no change in size of the preexisting lesion was observed, the treatment was recorded as a failure. These criteria are modifications of evaluation procedures used by Strindberg (1956) and Rud *et al.* (1972). The radiographs were analysed separately by two trained independent observers using a light-box with variable illumination and a magnification viewer according to the method described by Halse & Molven (1986). The two observers calibrated their criteria before analysis, only a few cases required subsequent joint evaluation to reach consensus.

Statistical analysis

Interobserver agreement was evaluated by Kappa test and the correlation between initial size of the lesions and outcome of treatment was analysed statistically by chi-square test.

Results

Trauma was considered the most common aetiological factor in pulp death and the development of periapical lesions (22 teeth) followed by either caries or defective

restorations. All but 12 teeth were maxillary anterior teeth (71.4%, 30 teeth). Pain was eliminated in 1–3 days in 20 of 26 symptomatic teeth while intra and/or extraoral swelling subsided in 6–12 days. There was high agreement between observers relating to evaluation of the outcome of treatment ($k = 0.800$; $P = 0.00$). Complete healing was observed in 31 teeth (73.8%) and incomplete healing in a further four (9.5%). Failure was observed in seven teeth (16.7%). All successful healing occurred within 8–24 months of treatment (Table 1). Lesions of 7–10 mm diameter (24 teeth) healed in 21 cases (87.5%) consisting of 19 complete healing cases (79.2%) and two healing incompletely (8.3%). The length of time required for healing in these cases ranged from 8 to 14 months.

Lesions with a diameter ranging from 11 to 18 mm (18 teeth) healed in 14 cases (77.8%), including 12 (66.7%) cases of complete healing and two (11.1%) of incomplete healing. The period for complete healing in these cases ranged from 9 to 24 months (Figs 1 and 2). Healing of periapical lesions around the partially resorbed calcium hydroxide paste had occurred in 15–24 months in five of six accidentally overfilled cases (Figs 3 and 4). No positive relationship was observed between the treatment outcome and the size of the periapical lesions ($P = 0.643$).

Root canal treatment failed in seven teeth. Possible causes of therapy failure included: overfilling (one case), occlusal trauma and advanced periodontal disease (two cases) and further traumatic injury (one case).

Discussion

The definitive diagnosis of the type of periapical lesion can only be made by a histological examination. However, a preliminary clinical diagnosis of a periapical cyst is reasonable if

Table 1 Distribution of teeth according to the initial size of periapical lesion and failure rate

	Success		Incomplete		Failure	
Initial size of lesion (mm)	*n*	%	*n*	%	*n*	%
7–10	19	79.2	2	8.3	3	12.5
11–18	12	66.7	2	11.1	4	22.2
Total	31	73.8	4	9.5	7	16.7

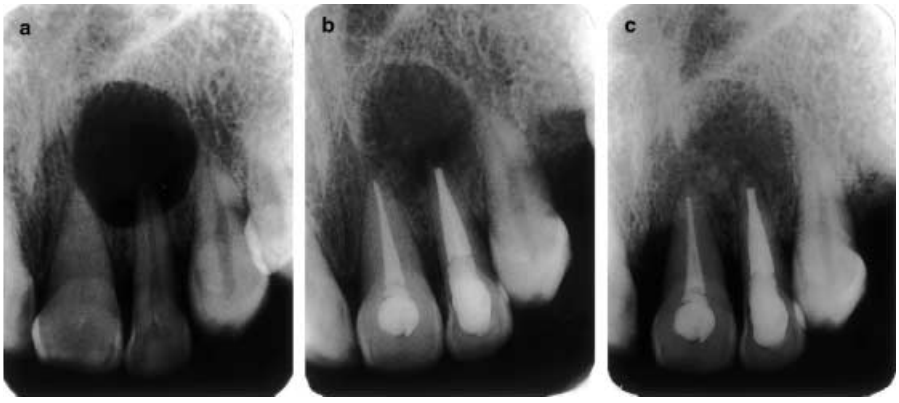

Figure 1 (a) Preoperative periapical radiograph showing a large well-circumscribed radiolucent lesion involving apices of maxillary left central and lateral incisors. (b) Fourteen months post-treatment radiograph showing reduction of periapical radiolucency. (c) Follow-up 4 years after completion of endodontic treatment. Periapical radiograph showing periapical repair. Periodontal treatment of the marginal periodontal lesion is continuing.

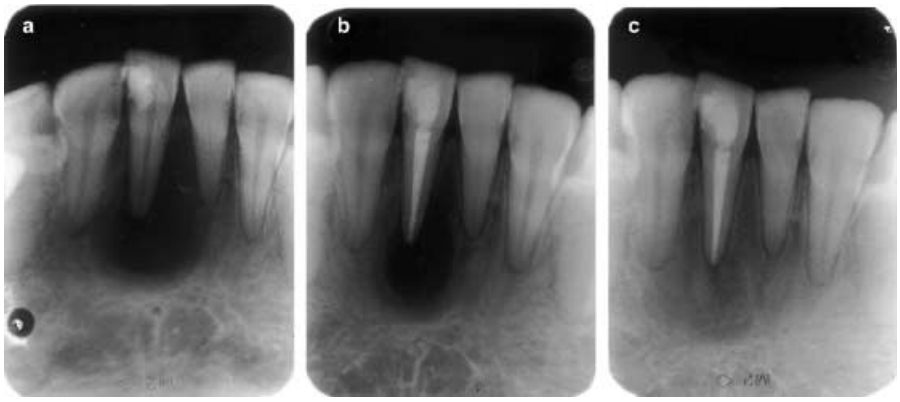

Figure 2 (a) Preoperative radiograph of mandibular right central incisor with large periapical lesion. Note the calcification of root canal of vital left central incisor due to trauma. (b) Six months post-treatment radiograph showing reduction of the periapical radiolucency. (c) Resolution of the radiolucent lesion 5 years after completion of endodontic treatment.

Figure 3 (a) Preoperative periapical radiograph demonstrating a large periradicular lesion with osseous extension and radio-opaque border involving apex of maxillary right lateral incisor. (b) Palatal swelling (abscess) in right lateral and central area. (c) Three-year follow-up radiograph showing healing of the lesion around the partially resorbed paste.

all of the following conditions exist: (i) the periapical lesion involves one or more teeth with necrotic pulps; (ii) the lesion is greater than 200 mm^2 in size, (iii) a straw-coloured fluid is produced upon aspiration or on drainage through an access; and (iv) the fluid contains cholesterol crystals (Eversole 1984). Additionally, the incidence of cysts has been shown to be 60–67% in lesions with a diameter of 10–20 mm (Lalonde 1970, Morse *et al.* 1973). Cholesterol crystals are encountered in 29–43% of dental cysts (Shear 1963, Browne 1971). They are more frequently encountered in radicular cysts than in apical granulomata. Crystals are identifiable under a microscope (Shear 1963, Browne 1971, Çalışkan & Türkün 1997). In the present study, aspiration of the fluid sample from inside the canal itself was simple to carry out, as most of the teeth had adequate drainage through the root canal. Radiographs revealed that the involved teeth had large periapical lesions with uniformly dense radiolucencies and well-defined margins around the apices. Upon observing a radiographically large periapical lesion and a straw-coloured fluid containing cholesterol crystals, a presumptive diagnosis of a periapical cyst can be made. This assumption is also supported by others (Loushine *et al.* 1991, Al-Kandari *et al.* 1994, Rees 1997, Çalışkan & Türkün 1997). The treatment options available to manage large cysts range from nonsurgical root canal treatment and/or apical surgery to extraction. In some instances,

Figure 4 (a) Periapical radiograph showing well-defined large radiolucent lesion associated with maxillary left lateral incisor, apical root resorption is evident. Radiograph taken immediately after the accidentally placement of calcium hydroxide paste beyond the apex into the lesion. (b) Follow-up 10 years after completion of endodontic treatment. Healing of the periapical lesion around partially resorbed paste is evident.

nonsurgical treatment may be ineffective or difficult; those cases may be treated by surgery. The decompression technique, which involves tubing, has been used widely with success (Loushine *et al.* 1991, Rees 1997).

Drainage is important in the conservative management of large periapical lesions. When direct and immediate drainage is procured from localized swellings or abscesses or cysts, the symptoms are reduced. Systemic antibiotics are not indicated (Dumsha & Gutmann 1988). According to Bender (1972) drainage is necessary before fibroplasia and cell migration can occur. The need for drainage is supported by histological studies (Fujii & Machida 1991). In the present study, the use of antibiotics was limited to six of 26 symptomatic patients and drainage procedures were performed for 15 days to 1 month due to persistent exudation.

The exact mechanism by which periapical cysts heal is not clearly understood. According to Simon (1980) and Nair *et al.* (1996) as the lumen of a 'bay' or 'pocket' cyst is open to the root canal it is likely to heal after conventional root canal treatment due to the removal of intracanal irritants. In contrast, the tissue dynamics of a true cyst are self-sustaining by virtue of its independence of the presence or absence of irritants in the root canal. True cysts, particularly large ones, containing cholesterol crystals are less likely to be resolved by conventional root canal treatment (Nair *et al.* 1993). Because it is clinically and radiographically impossible to differentiate a bay cyst from a true cyst, as it is likewise between a cyst and granuloma (Wood 1984), judicious treatment planning should favour a conservative approach to treatment; this approach is supported by many authors (Lalonde & Luebke 1968, Bhaskar 1972). It is claimed that nonsurgical management should in the same way lead to the local or generalized destruction of the epithelial lining of true cysts. Bhaskar (1972) suggested that if instruments are extended 1 mm beyond the apical foramen, the inflammatory reaction that develops destroys the cyst lining and converts the lesion into a granuloma. Once the causative factors are eliminated the granuloma heals spontaneously. Bender (1972) added that penetration of the apical area to the centre of the radiolucency may help in resolution by establishing drainage and relieving pressure. However, the added trauma of the minimal over-instrumentation may enhance epithelial

proliferation and cystic expansion, not resolution (Bender 1972, Seltzer 1988). Seltzer (1988) suggested that over-instrumentation allowed drainage of the cystic fluid, which then allowed degeneration of the epithelial cells by strangulation because fibroblastic and collagen proliferation squeezed the capillary supply to the cystic lining. The over-instrumentation technique was based on the assumption that the periapical lesion could be a cyst. Although the reasons these techniques might work are only theoretical, clinical success was claimed (Esposito 1990).

Packing the canal system with calcium hydroxide or the placement of calcium hydroxide into the periapical cyst is considered an alternative to over-instrumentation (Simon 1980, Webber 1983). Placement of the root canal paste beyond the apex is not indicated according to contemporary endodontic principles. However, it may be accidentally extruded during filling procedures.

The exact mechanism of action of calcium hydroxide is still speculative. Ghose *et al.* (1987) has advocated that direct contact between the calcium hydroxide and the periapical tissue was necessarily beneficial for osseoinductive reasons. It is suggested that if the calcium hydroxide is confined to the root canal, it is possible that the inflammation created by the diffusion of the calcium hydroxide through the apical foramen may be sufficient to cause break-up of the cystic epithelial lining, thereby allowing a connective tissue invagination into the lesion with ultimate healing (Webber 1983, Sahli 1988). Moreover, Souza *et al.* (1989) suggested that the action of calcium hydroxide beyond the apex may be fourfold: (i) anti-inflammatory activity; (ii) neutralization of acid products; (iii) activation of the alkaline phosphatase, (iv) antibacterial action.

It is apparent that its use beyond the apex intentionally or accidentally has been associated with the successful nonsurgical management of many cases of large periapical lesions (Webber 1983, Sahli 1988, Souza *et al.* 1989, Çalışkan & Şen 1996). Çalışkan & Şen (1996) suggested that teeth with periapical lesions, in which calcium hydroxide paste was extruded, did not show a different healing pattern from the ones treated conventionally. Although extrusion of calcium hydroxide was avoided in the present study, it did occur in six cases and five of these healed successfully.

Matsumato *et al.* (1987) has demonstrated that the prognosis for the treatment of large periapical lesions is not as good as that of small lesions. In contrast Strindberg (1956) and Sjögren *et al.* (1990) found no significant differences in healing frequency between lesions initially larger than 5 mm and those smaller than 5 mm. They have also stressed the importance of a long observation time for treated teeth with periapical lesions. In this study, follow-up examination ranged from 2 to 10 years. Approximately 70% of all healed cases were apparent within 2 years of treatment. Follow-up of 2–4 years was carried out in 16 cases, 10 cases were followed for 5–7 years and the remaining six cases were followed for 7–10 years. The failure incidence of teeth with periapical lesions from 11 to 18 mm in diameter was marginally higher than teeth with periapical lesions of 7–10 mm diameter.

Root canal treatment including calcium hydroxide indicator resulted in 73.8% complete healing of large periapical lesions, which was in accordance with previously reported results (Sjögren *et al.* 1990, Çalışkan & Şen 1996). Endodontic failure occurred in seven teeth. Scientific evidence indicates that microbial factors, comprising intraradicular and/or extraradicular infection, and intrinsic or extrinsic nonmicrobial factors may be associated with the unsatisfactory outcome of even well-treated cases (Siqueira 2001). It has been demonstrated that cholesterol crystals can be an intrinsic aetiological factor in nonresolving chronic inflammation (Nair *et al.* 1993). These crystals evoke a foreign body reaction with an accumulation of giant cells (Shear 1963, Browne 1971). Shah (1988) suggested that some individual immunological response variation may more influence the final outcome of the endodontic treatment.

Conclusions

The favourable results of this long-term study demonstrate that nonsurgical root canal treatment using calcium hydroxide in teeth with cyst-like large periapical lesions containing cholesterol crystal can be an alternative treatment to surgical therapy.

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References

Al-Kandari AM, Al-Ououd OA, Gnansekhar JD (1994) Healing of large periapical lesions following nonsurgical endodontic therapy: case reports. *Quintessence International* **25**, 115–9.

Bender IB (1972) A commentary on General Bhaskar's hypothesis. *Oral Surgery, Oral Medicine and Oral Pathology* **34**, 469–76.

Bhaskar SN (1966) Periapical lesion-types, incidence and clinical features. *Oral Surgery, Oral Medicine and Oral Pathology* **21**, 657–71.

Bhaskar SN (1972) Nonsurgical resolution of radiculer cysts. *Oral Surgery, Oral Medicine and Oral Pathology* **34**, 458–68.

Browne RM (1971) The origin of cholesterol in odontogenic cysts in man. *Archives in Oral Biology* **16**, 107–13.

Çalışkan MK, Şen BH (1996) Endodontic treatment of teeth with apical periodontitis using calcium hydroxide: a long-term study. *Endodontics and Dental Traumatology* **12**, 215–21.

Çalışkan MK, Türkün M (1997) Periapical repair and apical closure of a pulpless tooth using calcium hydroxide. *Oral Surgery, Oral Medicine and Oral Pathology* **84**, 683–7.

Dumsha TC, Gutmann JL (1988) Problems in managing endodontic emergencies. In: Gutmann IL, Dumsha TC, Lovdahl PE, eds. *Problem Solving in Endodontics*. Chicago, IL: Year Book Medical Publishers Inc., pp. 123–37.

Esposito JV (1990) Apical violation in periapical area cases-blasphemy or therapy? *Dental Clinics of North America* **94**, 171–8.

Eversole RL (1984) *Clinical Outline of Oral Pathology: Diagnosis and Treatment*, 2nd edn. Philadelphia, PA: Lea & Febiger, pp. 203–59.

Fujii H, Machida Y (1991) Histologic study of therapy for infected nonvital permanent teeth with incompletely formed apices. *Bulletin of the Tokyo Dental College* **32**, 35–45.

Ghose LJ, Baghdady VS, Hikmat BYM (1987) Apexification of immature apices of pulpless permanent anterior teeth with calcium hydroxide. *Journal of Endodontics* **13**, 285–90.

Halse A, Molven O (1986) A strategy for diagnosis of periapical pathosis. *Journal of Endodontics* **12**, 534–8.

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

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

Loushine RJ, Weller RN, Bellizzi R, Kulild JC (1991) A 2-day decompression: a case report of a maxillary first molar. *Journal of Endodontics* **17**, 85–7.

Matsumato T, Nagai T, Ida K et al. (1987) Factors affecting successful prognosis of root canal treatment. *Journal of Endodontics* **13**, 239–42.

Morse DR, Patnik IW, Schacterlie GR (1973) Electrophoretic differentiation of radicular cysts and granulomas. *Oral Surgery, Oral Medicine and Oral Pathology* **35**, 249–64.

Morse DR, Wolfson E, Schacterlie GR (1975) Nonsurgical repair of electrophoretically diagnosed radicular cysts. *Journal of Endodontics* **1**, 158–63.

Nair PNR, Sjögren U, Schumacher E, Sundqvist G (1993) Radicular cysts affecting a root-filled human tooth: a long-term post-treatment follow-up. *International Endodontic Journal* **26**, 225–33.

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

Natkin E, Oswald RJ, Carnes LI (1984) The relationship of lesion size to diagnosis, incidence and treatment of periapical cysts and granulomas. *Oral Surgery, Oral Medicine and Oral Pathology* **57**, 82–94.

Rees JS (1997) Conservative management of a large maxillary cyst. *International Endodontic Journal* **30**, 64–7.

Rud J, Andreasen JO, Jensen JE (1972) Radiographic criteria for the assessment of healing after endodontic surgery. *International Journal Oral Surgery* **1**, 195–214.

Sahli CC (1988) L'hydroxyde de calcium dans le traitment endodontique des grandles lesions periapicales. *Revue Francaise d'Endodontie* **7**, 45–51.

Seltzer S (1988) *Endodontology-Biologic Consideration in Endodontic Procedures*. 2nd Edn. Philadelphia, PA, USA: Lea and Febiger, pp. 412–28.

Shah N (1988) Nonsurgical management of periapical lesions: a prospective study. *Oral Surgery, Oral Medicine and Oral Pathology* **66**, 365–71.

Shear M (1963) Cholesterol in dental cysts. *Oral Surgery, Oral Medicine and Oral Pathology* **16**, 1465–73.

Shear M (1992) *Cysts of the Oral Regions*, 3rd edn. Oxford, UK: Wright, pp. 136–70.

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

Siqueira JF Jr (2001) Aetiology of root canal treatment failure: why well-treated teeth can fail. *International Endodontic Journal* **34**, 1–10.

Sjögren U, Hagglund B, Sundqvist G, Wing K (1990) Factors affecting the long-term results of endodontic treatment. *Journal of Endodontics* **16**, 31–7.

Souza V, Bernabe PFE, Holland R, Nery MJ, Mello W, Otoboni Filho JA (1989) Tratamento nao curugico de dentis com lesos periapicais. *Revista Brasileira de Odontologia* **46**, 36–46.

Strindberg LZ (1956) The dependence of the result of pulp therapy on certain factors. An analytic study based on radiographic and clinical follow-up examinations. *Acta Odontologica Scandinavian* **14** (Suppl. 21), 1–175.

Webber RT (1983) Traumatic injuries and the expanded endodontic role of calcium hydroxide. In: Gerstein CH, ed. *Techniques in Clinical Endodontics*. Philadelphia, PA: WB Saunders, pp. 238–9.

Wood NK (1984) Periapical lesions. *Dental Clinics of North America* **28**, 725–66.

