

## Literature Review Article

# Hypercementosis: a challenge for endodontic therapy

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## Abstract

**Introduction and objective:** The purpose of this study was to describe through literature review, the morphological characteristics of teeth with hypercementosis that are relevant to endodontic practice. **Literature review:** The pathologic deposition of cement increases proportionally as the patient's age increases. Genetic factors seem to be related to hypercementosis occurrence in young patients. Based on literature, it is possible to notice a lack of scientific studies which guide the endodontist for treating teeth with hypercementosis, since the cement deposition lead to the length increase of the cementum canal, and consequently, to the increase of the distance from CDC junction to apical root end. **Conclusion:** Thus, it is clinically relevant to correlate the morphology of teeth with hypercementosis with the specific aspects of endodontic therapy, aiming to establish the limits of root canal treatment in cases of hypercementosis.

## Introduction

Cementum is a mineralized conjunctive tissue, part of the insertion periodontium, which covers the root dentin in a firmly adhered way and, it is the interface between dentin and periodontal ligament. Cementum is a fine yellow layer with hardness inferior to dentin.

Cementum's main function is to protect the root and link it to bone through collagen fibers [22]. Chemically, cementum is constituted by approximately 50% of inorganic substances and 50% of water and organic material [29]. Similarly to bone tissue, its organic matrix is composed of mainly type 1 collagen and may undergo resorption and neoformation under pressure [28].

Excessive deposition of non-neoplastic cementum over root cementum alters root morphology, namely, hypercementosis. This cementum may be either hypocellular or cellular resembling bone (osteocementum). Generally, the cementum-like substance is deposited in concentric layers on either the entire root or to be limited to apical portion [28]. Radiographically, the exact amount of cementum thickness increase is difficult to assess, since cementum and dentin has similar radiodensity [10]. Additionally, the quantitative limits differing the physiological from the pathological cementum deposition – which characterizes the hypercementosis – are not measurable [31].

Clinically, hypercementosis may directly influence on root canal treatment, since the professional needs to know the most important anatomical references, during endodontic treatments, for determining endodontic treatment success: the cementum-dentin-canal (CDC) junction.

Based on the searched literature, we observed a lack of studies guiding the endodontic technique to be applied in teeth with hypercementosis, because cementum deposition leads to the increase of the cementum canal, and consequently, the increase of the distance from CDC junction to apical root end. Therefore, it is clinically important to correlate the morphological findings with the several specific aspects of endodontic therapy, aiming to establish the limits of root canal treatment in cases of hypercementosis.

## Literature review

### Hypercementosis: concept and etiopathogeny

Hypercementosis, so-called cementum hyperplasia, constitutes the excessive formation of this tissue and comprises either a limited point or the entire root surface [13].

Pinheiro *et al.* [31] emphasizes that to be characterized as hypercementosis, the cementum formation should be beyond the limit necessary to perform its normal functions, which results in both the abnormal thickening and root's macroscopic shape alterations.

Concerning to its etiopathogeny, in 1931, Gardner and Gardnes [16] discussed the hypothesis relation into hypercementosis aetiology. By analysing teeth with hypercementosis, the authors observed that this pathology occurred 4.2 times more frequently in teeth presenting necrosed pulp

than in teeth with vital pulp. However, it is difficult to determine whether the cementum deposition did not occur prior to pulp necrosis.

Thoma and Goldman [14] affirmed that secondary cementum deposition occurs through four different processes: tooth continuous eruption, presence of inflammatory reaction, formation of spicules, and root repair. The authors demonstrated that hypercementosis affects not only teeth already erupted and its occurrence cannot be only attributed to tooth function, because teeth without antagonists or not yet erupted can also present the pathology. According to the authors, irritating factors of low intensity seem to be the main cause of cellular activity at the periphery of the inflamed area, stimulating the cementum growth and, consequently, leading to hypercementosis.

Also, osteitis deformans (Paget's disease) was related to hypercementosis occurrence [11, 15, 37], in which there is fast resorption and extensive bone deposition. Among the characteristics of Paget's disease is the disappearance of lamina dura due to the amount of secondary cementum deposition on the tooth roots.

Azaz *et al.* [5] reported that cementum thickness increase in non-functional teeth is probably related to the age of the impacted tooth and that the increase of cementum apposition may happen due to impaired continue eruption force. In 1977, Azaz *et al.* [4] reinforced the idea that cementum apposition is a phenomenon caused by age, because increasingly occurs in older patients, even in impacted teeth.

According to Shafer *et al.* [37], cementum deposition may be due to a low-intensity inflammation, similarly to that occurring both in apical pericementitis and in pulp's chronic inflammatory process; or it can be still stimulated by the chronic inflammation of the periapex. Prabhakar *et al.* [32] still reported a clinical case in which the repetitive formation of dental abscesses would be responsible for the hypercementosis formation.

### Hypercementosis histopathology

Physiological cementum is a calcified tissue covering root surface with hardness inferior to dentin. Its composition is equally divided into organic material (50%) and inorganic material (50%), and it is the body's tissue with the highest percentage of fluoride. The cementum is part of the interface dentin/periodontal ligament, together with periodontal ligament's collagen fibers, so-called Sharpey's fibers, inserted into its most superficial layer [29].

McQuilen [26], the first author who microscopically described hypercementosis, reported a tissue similar to secondary dentin, with numerous lacunas and elongated canaliculi, either crooked or curved, as well as canals that enable the passage of blood vessels.

Menguini *et al.* [27] observed, through scanning electronic microscopy, teeth with hypercementosis and described a lamellar structure of the hyperplastic cementum with incremental lines either regular or irregular. They also found cementocytes, Tomes's granular layer, Sharpey's fibers, and small cementum particles. Hyperplastic cementum surface, as well as physiological, presented numerous mineralized globular formations, roughly hemispherical, corresponding to the insertion of Sharpey's fibers.

### Clinical implications of hypercementosis in Endodontics

By observing teeth with hypercementosis, Pinheiro *et al.* [31] reported that the dentinal canal is located more than 1 mm above the radiographic apex. Also, the authors highlighted that the endodontist may experience difficulty, in these cases, for reaching the adequate shaping and filling limit because this cementum may be not permeable to endodontic instruments. It is important highlighting that root canal shaping and filling below the adequate limit will cause the retention of either a contaminated area or inflamed tissue within root canal, without repair conditions, leading to endodontic treatment failure.

Coolidge [12] was perhaps the first author to draw attention for the role of pathological cementum deposition in Endodontics. He reports that cementum deposition would be related to root fracture occurrences, with cementum deposition because of either aggression to periodontium or functional trauma, and which would interfere in pulp removal and periapical inflammation treatment.

Barros [6] reported the hypercementosis relationship with the occurrence of a greater number of secondary canals, accessory canals, and apical deltas, as well as apical third constriction associated to change in the original path of the main canal. The author emphasized that these modifications in the internal morphology of the apical third of teeth presenting hypercementosis may not be seen radiographically, which may complicate endodontic treatment.

According to Pinheiro *et al.* [31], hypercementosis may represent additional sites for bacterial colonization during pulp contamination, contributing

to the occurrence of chronic apical periodontitis resistant to the endodontic treatment. Additionally, in hypercementosis, the foramen may be located at the root's lateral surface, leading to the development of endoperiodontal lesions [48].

### Apical root anatomy and its implications in endodontic treatment

Apical root anatomy comprises three different anatomical and histological limits: the apical constriction, CDC junction, and apical foramen. According to Kuttler [23], the apical constriction is generally localized at 0.5 to 1.5 mm short of the apical foramen. This area is still the most frequent landmark used by the dentist as the apical limit for performing root canal cleaning, shaping, and filling. CDC junction location, however, is very variable.

In most times, root canal is not apically opened in a single apical foramen, but in secondary and accessory canals, constituting the so-called apical delta; they are opened in small foramens, namely foramina, presenting diameters between 60 and 80  $\mu\text{m}$  [18].

In teeth without hypercementosis, starting from the apical constriction, root canal becomes wider as it is close to the apical foramen [43]. The shape of the space between the apical constriction and foramen, however, can be defined as conical or tapered, with the smallest diameter faced to the apical constriction. Due to the continuous deposition of cementum at the apical area, the mean distance between these two points increase, as the patient gets older [45].

### Philosophy of treatment in biopulpectomy and necropulpectomy cases

The main goal of endodontic treatment is the obtainment of favorable conditions to apical and periapical tissues' healing. The situations, in which dental pulp and root canal systems are found, would provide different conditions for tissue healing that should occur after the endodontic treatment, and root canal therapy must be initiated according to a correct pulp diagnosis.

In biopulpectomy cases sterile root canals are presented, and consequently, endodontic therapy should be based on root canal shaping (preparing it to proper filling), in the maintenance of the aseptic chain, and employment of biocompatible substance and materials, not resulting in aggression to pulp stump, therefore easing apical and periapical healing [20, 24].

However, in necropulpectomy cases, endodontic success depends mainly on elimination or maximum reduction of root canals' infection. Such goals are still more complex in cases of apical periodontitis with pulp necrosis, in which the infection is a longer duration process and the microorganisms and their sub-products have already been established in root canal systems, apical and periapical tissues [2, 25, 38].

Generally, teeth presenting root canals with pulp necrosis have some degree of apical inflammatory resorption of either the cementum or dentin [3, 17], which alters the normal apical anatomy, favoring bacterial colonization. Bacteria are located in apical ramifications, deltas or foramina, and accessory/secondary canals. Moreover, these resorption areas are very irregular and retentive, favoring bacterial biofilm formation, which is a medium for facilitating bacterial survival. In teeth with pulp necrosis, therefore, root canal cleaning is imperative, in its entire length, to maximize the removal of the infectious and necrotic content of root canal system.

### Root canal shaping and filling limits

The likelihood of aggression to the periapical tissue makes the endodontist limit root canal treatment to a working length always below the radiographic apex. Within this concept, cementum canal, as defended by some authors, should not be shape [19, 33, 34, 36].

One of the most discussed approaches during root canal treatment and filling is the establishment of apical limit. The exact point for cleaning, shaping, and filling of root canals is still very controversy [8, 30]. Apical constriction, placed from 0.5 to 2 mm below the radiographic apex is the main landmark used. However, clinical determination of root canal apical morphology is at least very difficult to be precisely perform, because the presence of the apical constriction seems to be more conceptual than clinically real [14]. Moreover, it is important highlighting that in cases of root resorptions and periapical pathologies, the occurrence of apical constriction is even rarer [39].

Wu *et al.* [47] emphasized the difficult in localizing the apical foramen and constriction, clinically; therefore, root apex would be the only trustable landmark during endodontic therapy. The authors recommended as the apical limit of the clinical procedures, measurements between 0 and 3 mm below the radiographic apex, depending on pulp diagnosis.

Apical pulp stump preservation is defended when its functional preservation is aimed at its anatomical site, after root canal shaping and filling of non vital teeth. Accordingly, Ingle *et al.* [21] stated that biologically, the ideal point would be the endodontic treatment terminus at sound dentin, to protect the pulp stump in cases of biopulpectomy and avoid postoperative discomfort resulting from over-obturation.

According to Leonardo [24], root canal instrumentation of teeth presenting pulp vitality should be limited to dentinal canal, radiographically placed approximately 1 to 2 mm below root apex, aiming not to traumatize the pulp stump, which would be essential for tissue repair in these cases. In necropulpectomy cases without periapical lesion, the author recommended that the working length be placed between 1 to 2 mm below the radiographic apex, since the cementum canal is still intact. In necropulpectomy cases with chronic periapical lesion, because the cementum canal was resorbed and dentin is not covered, working length should be closer to the apical portion, 1 mm below the radiographic apex.

### Apical patency

The concept of apical patency, i.e., the passage of patency files through root canal terminus, is controversial. Some endodontists report fear that, by exploring the apical foramen opening, they would cause debris extrusion for the periapical area, periapical inflammation and severe postoperative pain. However, it is known that, by only shaping root canal below the apical constriction, it is likely to increase the chances for persistent periapical infection [9].

Although the exact percentage of cases in which cementum canal contamination is not known, the infection occurrence at the most apical area of root canals system is very common in most of pulp necrosis cases [7, 46].

Such understanding has brought major changes to endodontic therapy and cementum canal should be cleaned during shaping, which means that in many cases, endodontic treatment should not be limited to only one point at 1 mm short of the radiographic apex, but it should be extended through root canal entire length, including both dentin and cementum canals [41]. Accordingly, in cases with periapical lesion, the recognition of microorganisms' presence within the cementum canal [7] and periapical lesions [46] contributes to disseminate the acceptance of the principle of apical foramen cleaning and debridement during



root canal shaping instead of instrumentation, because the filling limit must be kept below the radiographic apex.

Unfortunately, working length loss is very common in endodontic therapy, especially among less experienced professionals. Its main cause is the accumulation of dentin debris at apical portion [1, 41]. Therefore, apical patency importance is also mechanical and aimed to maintain working length.

It is observed that orthodox techniques have already established for several years a shorter instrumentation, preferably by an apical stop or at the apical constriction, without surpassing it. Most of the times, the constriction would be the main basis of apical limit determination. The periapex should be respected; however, this does not mean that apical foramen should not be cleaned [13].

To avoid mechanical and biological problems, it is necessary that root canal be cleaned in its entire extension, by removing an equal amount of dentin from the canal walls, at its three dimensions, following its own anatomical path. Also, the foramen should undergo patency during all root canal preparation. Moreover, apical patency will potentialize the action of the irrigants on bacteria within the canal and even at apical ramifications. In necrosed teeth with apical infection, bacteria may be located at apical foramina.

Simon [39], by referring to the preparation of necrosed teeth, states that to clean as best as possible and allow the correct penetration of sodium hypochlorite in all root canal systems, it is necessary to instrument up to the radiographic apex. However, it is known that in several circumstances, the radiographic apex is surpassed. Notwithstanding, if small instruments are used in tooth's real length, they do not cause greater clinical problems or flare-ups.

In pulp vitality cases, there is no infection in both dentin and cementum canals. Consequently, from a biological point of view, there is no need of disinfection procedures, which means that the foramen cleaning is not justified [19, 33, 34].

On the other hand, in cases presenting pulp necrosis the cleaning procedure is justified to create conditions for repair. Accordingly, in teeth with vital pulp, the removal of a healthy tissue to be replaced for a tissue in the same conditions does not seem consistent [42].

## Discussion

Literature displays studies on the features of hypercementosis, its frequency, and severity

degree. Notwithstanding, although mentioning its consequences, endodontic treatment difficulties or specificities of teeth presenting this alteration are rarely cited. Therefore, we intended to correlate the approach performed in usual endodontic treatment with the treatment to be executed in teeth with hypercementosis, always taking into account the biological aspects and the probable approaches for obtaining success in both procedures. With a frequency of about 4.9% in population [5], hypercementosis may be considered as a relatively common finding, and its influence on the treatment of teeth affected by this alteration should be known by the dentist.

Therefore, it is stated that, clinically, hypercementosis may directly influence on root canal treatment because the clinician needs to know the limits for root canal shaping and filling. Moreover, according to Siqueira *et al.* [40], the beginning of the cementum canal is the narrowest area of root canal and is divergent to tooth apex; even its clinical and radiographic determination is not viable because is highly inconstant regarding to tooth apex of several teeth.

On the other hand, the cementum presenting the function of dentin protection, even after the pulp death, can obliterate the apical foramens and therefore impede the passage of external irritating agents to the body. These alterations determine a change in the filling's apical limit and we must consider such factors which play a essential role in avoiding the presence of empty spaces, due to lack of material filling at root canal's apical portion. Through odontometry, it is possible to determine tooth's working length and provide less risk of chemical and mechanical aggressions, consequently obtaining a greater chance for repair. By following this theory, in hypercementosis cases, CDC junction is no more the main focus, and we should pay attention in the biological principles stating that the increased cementum canal could contain microorganism capable of perpetuating a chronic inflammatory reaction. In these cases, since the necrotic tissue is integrally removed, the periodontal ligament cells promoted their cure and healing.

Rosa-Neto [35] concluded that the apical limit should be at 1 mm short of the radiographic apex, which provides a better dentinal canal filling in teeth showing pulp necrosis and chronic periapical lesion. In cases of vital pulp, this limit also seems to be the most appropriate, because it will reduce the chances of inflamed tissue maintenance within root canal which would undergo necrosis and later contamination. Accordingly, root canal cleaning,

shaping and filling is recommended to be performed within the entire length up to apical foramen, under any pulp condition - with or without vitality, with or without contamination.

According to Siqueira Jr. *et al* [40], the lack of working length accuracy may lead to inappropriate cleaning and shaping, which eventually would favor to undesirable situations such as: symptomatic postoperative period, due to inflammation and/or infection in the pulp remnant at root canal's apical third.

Pinheiro *et al.* [31] stated that most of root apexes of teeth presenting mild and diffuse hypercementosis did not show irregularities and resorption, but they had a greater number of foramina. Moderate hypercementosis cases presented more irregular areas and also foramina presence. In severe hypercementosis, the authors found a decrease, and in sometimes, obliteration of root apex. Therefore, it is possible to indicate a conventional endodontic treatment in cases of mild and diffuse hypercementosis because the working length will not show many differences compared with normal teeth. However, to assure a correct root canal cleaning in cases of moderate and severe hypercementosis, the instrumentation should reach the apical foramen in cases presenting necrotic tissue at the apical portion.

Some authors support the idea that the cementum canal should be cleaned during instrumentation, conditioning the treatment extension to the entire root canal length [42, 42]. Accordingly, in cases showing periapical lesion, the acceptance of apical foramen's cleaning and debridement during canal's instrumentation, so-called apical patency, is well established. Nevertheless, in cases of pulp vitality, several authors recommended that the apical foramen area be kept intact. In hypercementosis cases, it is believed that this same approach must be followed, i.e., whenever possible the cases presenting pulp necrosis must have the entire root canal length cleaned; biopulpectomy cases cannot undergo apical patency.

Root canal treatment comprises several interlinked phases that should be strictly followed to obtain endodontic treatment success. By considering these steps, we should remember that root canal's filling is endodontic treatment main goal and must fill all empty spaces within root canal. If empty spaces are kept, the periapical exudate can infiltrate and compromise, physically and mechanically, the endodontic treatment outcome. Also, periapical infiltrate can result in interfaces between the filling material and root canal's walls, allowing that bacterial and their sub-products are accessible to

periapical tissues. The choice of filling technique by gutta-percha thermoplastification may provide a better filling of secondary/accessory canals and apical deltas [31], which is an interesting alternative also in cases of hypercementosis.

Both the presence of a greater number of foramina and apical foramen obliteration in hypercementosis cases warn for the need of further studies on the development of techniques, medicaments and products to allow the adequate root canal shaping and filling, aiming to a better repair and, if possible, to the biological sealing of root canal systems to assure a favourable prognosis of these cases.

## Conclusion

There is a lack of scientific-based studies guiding the root canal treatment in teeth with hypercementosis since this leads to the occurrence of cementum canal increase and increases the distance from the CDC junction to the apical vertex. The clinical implications of teeth with hypercementosis, in cases of biopulpectomy and necropulpectomy, lead to pay attention to its different type and severity. Therefore, the clinician should evaluate the necessity of adapting the usual endodontic therapy approach to assure all Endodontics principles and reach treatment success.

## References

1. Al-Omari MAO, Dummer PM. Canal blockage and debris extrusion with eight preparation techniques. *J Endod.* 1995 Mar;21(3):154-8.
2. Ando N, Hoshino E. Predominant obligate anaerobes invading the deep layers of root canal dentin. *Int Endod J.* 1990 Jan;23(1):20-7.
3. Andreasen JO. External resorption: its implication in dental traumatology paedodontics, periodontics, orthodontics and endodontics. *Int Endod J.* 1985 Apr;18(2):109-18.
4. Azaz B, Michaeli Y, Nitzan D. Aging of tissues of the roots of nonfunctional human teeth. *Oral Surg Oral Med Oral Pathol.* 1977 Apr;43(4):572-8.
5. Azaz B, Ulmansky M, Moshev R, Sela J. Correlation between age and thickness of cementum in impacted teeth. *Oral Surg Oral Med Oral Pathol.* 1974 Nov;38(5):691-4.

6. Barros LAP. Estudos macro e microscópico da morfologia do terço dentário apical na hipercementose: implicações clínicas e etiopatogênicas [tese]. Bauru: Universidade de São Paulo; 1999.
7. Baumgartner JC, Falkler Jr. WA. Bacteria in the apical 5 mm of infected root canals. *J Endod.* 1991 Aug;17(8):380-3.
8. Bergenholtz G, Spangberg L. Controversies in Endodontics. *Crit Rev Oral Biol Med.* 2004 Jan;15(2):99-114.
9. Buchanan LS. Management of the curved root canal. *J Calif Dent Assoc.* 1989 Apr;17(4):18-25.
10. Bush B, Matthee MJ. Radiological diagnosis IX. Hypercementosis. *J Dent Assoc S Afr.* 1985 Jan;40(1):23.
11. Colby RA, Kerr PA, Robinson HBG. Color atlas of oral pathology. 3. ed. Philadelphia: J. B. Lippincott; 1971.
12. Coolidge ED. The reaction of cementum in the presence of injury and infection. *J Am Dent Assoc.* 1931 Mar;18(3):499-525.
13. De Deus QD. Endodontia. 5. ed. Rio de Janeiro: Medsi; 1992.
14. Dummer PMH, McGinn JH, Rees DG. The position and topography of the apical canal constriction and apical foramen. *Int Endod J.* 1984 Oct;17(4):192-8.
15. Gardner AF. A survey of periapical pathology. *D Digest.* 1962;68:260-3.
16. Gardner BS, Goldstein H. The significance of hypercementosis. *Dent Cosmos.* 1931;73:1065-9.
17. Hammarstrom L, Lindskog S. General morphological aspects of resorption of teeth and alveolar bone. *Int Endod J.* 1985 Apr;18(2):93-108.
18. Hess JC, Culleras MJ, Lamiabile NA. A scanning electron microscopic investigation of principal and accessory foramina on the root surfaces of human teeth, thoughts about endodontic pathology and therapeutics. *J Endod.* 1983 Jul;9(7):275-81.
19. Holland R, Sant'anna-Júnior A, Souza V, Dezan-Junior E, Otoboni-Filho JA, Bernabé PFE et al. Influence of apical patency and filing material on healing process of dogs' teeth with pulp after root canal therapy. *Braz Dent J.* 2005 Jan-Apr;16(1):9-16.
20. Horsted-Bindslev P, Lovschall H. Treatment outcome of vital pulp treatment. *Endod Topics.* 2002 Jul;2 (1):24-34.
21. Ingle JI, Bakaland LK. Endodontia. 4. ed. Baltimore: Wiliam & Wilkins; 1994. 944 p.
22. Kronfeld R. Histopatologia dos dentes. 3. ed. Rio de Janeiro: Científica; 1955.
23. Kuttler Y. Microscopic investigation of root apexes. *J Am Dent Assoc.* 1955 May;50(5):544-52.
24. Leonardo MR. Endodontia, tratamento de canais radiculares. Princípios técnicos e biológicos. São Paulo: Artes Médicas; 2005.
25. Leonardo MR, Almeida WA, Ito IY, Silva LA. Radiographic and microbiologic evaluation of posttreatment apical and periapical repair of root canals of dog's teeth with experimentally induced chronic lesion. *Oral Surg Oral Med Oral Pathol.* 1994 Aug;78(2):232-8.
26. McQuillen JH. Exostosis. *Dent Cosmos.* 1860;1:428-32.
27. Menghini P, Piacentini C, Rysky C, Resta G, Sapelli PL. Contribution of light and scanning electron microcopy to the study hypercementosis. *G Stomatol Ortognatodonzia.* 1983 Jan-Mar;2(1):15-20.
28. Neville WB. Patologia oral e maxilofacial. 2. ed. Rio de Janeiro: Guanabara; 2004.
29. Orban B. Oral histology and embryology. 3. ed. St. Louis: Mosby; 1953.
30. Peters OA. Current challenges and concepts in the preparation of root canal systems: a review. *J Endod.* 2004 Aug;30(8):559-67.
31. Pinheiro BC, Novaes T, Capelozza ALA, Consolaro A. A scanning electron microscopic study of hypercementosis. *J Appl Oral Sci.* 2008 Nov-Dec;16(6):380-4.
32. Prabhakar AR, Reddy VV, Bassappa N. Duplication and dilacerations of a crown with hypercementosis of the root following trauma: a case report. *Quintessence Int.* 1998 Oct;29(10):655-7.
33. Ricucci D, Langeland K. Apical limit of root canal instrumentation and obturation. Part 2. A histologic study. *Int Endod J.* 1998 Nov;31(6):394-409.

34. Ricucci D. Apical limit of root canal instrumentation and obturation. Part 1. Literature review. *Int Endod J*. 1998 Nov;31(6):384-93.
35. Rosa-Neto JJ. **Estudos em microscopia eletrônica de varredura do ápice radicular e do limite de obturação em dentes portadores de lesão periapical crônica**. Araraquara, 1997 [tese]. Araraquara: Universidade Estadual Paulista; 1997.
36. Schaeffer MA, White RR, Walton RE. Determining the optimal obturation length: a meta-analysis of literature. *J Endod*. 2005 Apr;31(4):271-4.
37. Shafer WG, Hine MK, Levy BM. *A textbook of oral pathology*. 4. ed. Philadelphia: Saunders;1983.
38. Shovelton DS. The presence and distribution of microorganisms within non-vital teeth. *Br Dent J*. 1964 Aug;117(3):101-7.
39. Simon JHS. The apex: how critical is it? *Gen Dent*. 1994 Jul-Aug;42(4):330-4.
40. Siqueira Jr. JF, Lopes HP, Elias CN. Obturação do sistema de canais radiculares. In: Lopes HP, Siqueira Jr JF. *Endodontia, biologia e técnica*. Rio de Janeiro: Medsi; 1999. p. 451-84.
41. Souza RA. Limpeza de forame e sua relação com dor pós-operatória. *J Bras Endo Perio*. 2000 Oct-Dec;1(3):45-8.
42. Souza RA. The importance of apical patency and cleaning of the apical foramen on root canal preparation. *Braz Dent J*. 2006 Jan-Apr;17(1):6-9.
43. Taylor GN. *Techniche per la preparazione e l'otturazione intracanalare*. *Clin Odont N Am*. 1988 Jul;20(7):566-81.
44. Thoma KH, Goldman HM. *The pathology of dental cementum*. *J Am Dent Assoc*. 1939;26:1943-53.
45. Vertucci FJ. Root canal morphology and its relationship to endodontic procedures. *Endod Topics*. 2005 Mar;10(1):3-29.
46. Wayman BE, Murata SM, Almeida RJ, Fowler CB. *A bacteriological and histological evaluation of 58 periapical lesions*. *J Endod*. 1992 Apr;18(4):152-5.
47. Wu MK, Wesselink P, Walton R. Apical terminus location of root canal treatment procedures. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2000 Jan;89(1):99-103.
48. Zehnder M, Gold SI, Hasselgren G. Pathologic interactions in pulpal and periodontal tissues. *J Clin Periodontol*. 2002 Aug;29(8):663-71.