Apical root resorption: classification, diagnostic approach, therapeutic management

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Abstract

In clinical practice, cases of root resorption in one or more teeth are often encountered. In primary teeth, this condition is not clinically significant; however, in permanent teeth, it is an abnormality. This descriptive review will address all parameters related to the pathological condition of apex root resorption, including its etiology, prevalence, diagnosis, clinical significance, therapeutic approach, and classifications proposed by various authors over time. These classifications are linked to the etiology, location, and histological features of apex root resorption. In contrast, therapeutic management is associated only with the cause of its onset. This study aims to provide clinicians with a tool to distinguish among various pathological entities and manage them effectively. Accurate diagnosis and proper prognosis are crucial for guiding the practitioner toward personalized management and appropriate treatment.

Keywords: Apical resorption, Inflammatory resorption

Introduction

Apical root resorption in permanent teeth is a pathological condition characterized by the loss of root substance as a result of the action of osteoclasts and immune cells, in response to factors such as bacterial infection, trauma, and chemical or physical agents that irritate or injure the periodontal ligament and the dental pulp (1,2,3). In contrast, in primary teeth, it is a desirable physiological process that allows the atraumatic eruption of their permanent successors. The morphology of the apical root area is decisive for determining the biologically acceptable point at which the working length and the obturation of the root canal should be established. According to the literature, the point at which chemomechanical preparation should be completed is the apical constriction, which terminates at the apical foramen, whose position does not always coincide with the anatomical apex (4).

Deviations in the morphology of the apical region have great clinical significance because they affect the ability to achieve a hermetic seal. It is evident that, in cases where apical resorption is also present, the morphology of the apex resulting from the resorptive processes negatively influences the clinician's ability to obturate the root canal. Resorption may destroy the apical constriction and compromise the retention of obturation materials and irrigating solutions within the root canal. Therefore, for treatment purposes, the clinician must be aware of and able to manage such conditions. The categories of resorption indicate its etiology, location, and morphology. The

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most widely used classification of apical resorption is Andreasen's. Each type of resorption has its own clinical and histopathological characteristics, as well as characteristic causative factors and management approaches, in addition to its frequency of occurrence—elements that will be analysed in the present article.

Classification and etiology of apical resorption

Apical resorption constitutes a pathological process of degradation of the mineralized dental tissues, which is activated following injury or inflammation of the periodontal ligament and the protective layer of cementoblasts and the epithelial rests of Malassez (5,6). When these tissues become irritated, a chemotactic process leads to the accumulation of osteoclasts, which initiate resorption. Osteoclasts are located between the periodontal ligament and the cementum, residing in Howship's lacunae (6). Although multiple classifications have been proposed, Andreasen was the first to introduce a clear functional framework, distinguishing resorption into internal (inflammatory and replacement) and external (surface, inflammatory, and replacement) categories (1).

Subsequent authors, such as Gartner et al. (7), Tronstad (2), and Trope (8), maintained the same basic distinction, while modifying specific subcategories based on location or etiology. Heithersay (9), Patel (10), and Darcey & Qualtrough (11) expanded this perspective by adding forms such as cervical or transient resorption.

Finally, Kanas and Kanas (12) proposed the most comprehensive and contemporary classification, incorporating parameters of dental and non-dental etiology (traumatic, inflammatory, idiopathic, orthodontic, or systemic).

Despite the evolution of classification systems, Andreasen's approach (1) remains the most widely used, as it establishes the fundamental distinction between internal and external resorption, upon which subsequent frameworks are based. External inflammatory resorption comprises the following subcategories: cervical resorption, with or without pulpal involvement, and apical resorption, which is primarily associated with pulpal infection (13, 14, 15).

However, apical resorption may also be associated with non-microbial causes, such as orthodontic tooth movement, in which aseptic inflammation is observed. Additionally, certain types of dental trauma, such as avulsion and intrusion, also appear to contribute to resorption of the apical portion, as the violent injury to the periodontal ligament can induce external replacement resorption.

Prevalence of different types of resorptions

Bates first described the concept of apical resorption in 1856, associating it with trauma to the periodontal membrane (16). Ketcham (1927, 1929) studied 500 patients after orthodontic treatment and found resorption in 21%, while in untreated individuals the rate was 1% (17, 18). Rudolph (1936) reported a prevalence of up to 74%, which in a later study of his reached 100%. In a control sample without orthodontic treatment, he found only 5% (19, 20). In 1947, at the University of Illinois, histological analysis of 261 teeth showed resorption in 90.5% of cases (21). Massler and Malonea (1954), in a sample of 708 individuals, recorded 86.4% with resorption, 1.6% with no lesions at all, and 81.2% idiopathic cases (22).

Sex-related differences have been inconsistently reported: some studies found a higher prevalence in females (23), while others reported no statistically significant association with gender (24). The prevalence increases with age (1, 2). Resorption is most frequently located at the apex (22) and in maxillary teeth (25), although opposing findings have also been reported (26). Molars are most affected (27), while other studies report central incisors due to their susceptibility to trauma (18, 28, 29). External apical resorption accounts for 95.2% of cases, whereas internal resorption accounts for 4.8%. The external inflammatory form appears in 58.04%, with pulpal infection being the main cause (71.2%) (30), although others do not confirm this association (31).

According to the University of Illinois, inflammation does not always cause resorption, as no defects were observed in the cervical third despite the presence of periodontal inflammation. External inflammatory resorption is also associated with trauma, occurring in 18% of cases after partial avulsion and in 30% after replantation (1, 30). Replacement resorption was found mostly in molars (0–15.04%) (30), and more frequently after complete avulsion (87.2%), intrusion (57.1%), lateral displacement (35.5%), or vertical displacement (4.8%) (1). The type of resorption is related to the type of tooth: in second molars, replacement resorption is more prevalent, whereas in first molars, external inflammatory resorption predominates (30). External cervical resorption is recorded at 22.12%, while periodontal-related resorption is at 3.9%, increasing with reduced bone support, deeper pockets, and greater tooth loss. Idiopathic resorption occurs mainly in young individuals (mean age 23 years) and accounts for 4.42% of cases, mostly in maxillary canines (26).

However, the limitations of these studies must be noted. Few studies analyse the prevalence of each type of resorption separately, while most rely on panoramic radiographs, which underestimate lesion extent. In studies using CBCT or histological analysis, the reported rates are significantly higher (32). Moreover, there is no universally accepted severity scale or standardized criteria for what constitutes pathological resorption, making comparisons between older and more recent data difficult. Finally, classification and etiological associations vary among authors, leading to variable and often non-comparable results (33, 34).

Diagnosis

The diagnosis of resorption is usually an incidental radiographic finding and is difficult, as the affected teeth are often asymptomatic (35). The most common diagnostic method is panoramic radiography, either alone or in combination with periapical imaging, primarily to overcome diagnostic difficulties in the

anterior region (36). In their study, Sameshima and Asgarifar, comparing panoramic radiographs with periapical images as a diagnostic tool, found that panoramic imaging overestimates resorption and root shortening by more than 20% (37). However, imaging with the paralleling technique, as reported by Remington et al., cannot accurately measure root lengths (38). Furthermore, although it has high diagnostic value, it is geometrically inaccurate (39).

To investigate the morphology and extent of resorption on all root surfaces, the MBD method proposed by Gartner et al. is often used, which involves taking two radiographs—one perpendicular to the root and the other with mesial angulation (7). The different imaging angles help identify the surfaces involved and allow proper classification of the lesion. Nevertheless, in their study, Creanga et al. observed slightly better diagnostic outcomes (40) using only perpendicular projections compared with angled projections. The available literature, however, is limited, as all studies comparing two-dimensional with three-dimensional imaging treat periapical radiography as a single modality regardless of the angle at which it is taken (40).

The above study agrees that the most reliable diagnostic method for resorption is three-dimensional imaging. CBCT imaging is a more sensitive technique, although its reported sensitivity is 69% (41).

The specificity of the method using different angulations of conventional periapical radiographs and CBCT was found to be above 79%, with that of CBCT reaching 91% (40).

Patel et al. support the use of cone-beam computed tomography, as it appears to have greater diagnostic value—something that is now also recommended by the European Society of Endodontology (32).

Chapnick reports that small apical resorptive defects are more difficult to diagnose (42). For this reason, Heo proposes digital subtraction radiography for the detection of small resorptions (43), although Levander demonstrated that digital techniques were comparable to conventional film-based methods (44). In their study, Chan and Darendeliler found that after 28 days of orthodontic force application, the diagnostic value of CBCT and periapical radiographs was comparable. However, they report that after a longer period, when resorptive areas become larger, periapical radiographs may be insufficient for evaluating resorption (45).

In the studies by Darcey and Qualtrough (11), the clinical and radiographic characteristics of the various types of resorptions are described. The presentation of signs and symptoms must be framed within an accepted classification system. The classification we will use is that of Darcey and Qualtrough, one of the most contemporary and closely aligned with that of Andreasen, which is the most widely used and generally accepted, an advantage when synthesizing the literature. Specifically, they describe internal replacement and internal inflammatory resorption, which may progress and lead to root perforation. They then distinguish the external resorptions caused by periodontal damage, subdivided into surface, inflammatory, replacement (which leads to ankylosis), cervical, and apical resorption. For each type of resorption, the authors provide radiographic diagnostic features.

Regarding external apical resorption, in two-dimensional imaging, it appears as shortening and rounding of the apex with well-defined margins and the presence or absence of a periapical lesion, depending on whether an inflammatory cause is present. The root canal remains clearly visible and continuous. In three-dimensional imaging (CBCT), the authors describe asymmetry at the apex, conical loss of tooth structure, or a crater-like defect, with or without bone involvement, depending on the presence of an inflammatory etiology. When the cause is orthodontic treatment, multiple teeth are symmetrically affected.

Management of apical resorption

Non-inflammatory apical resorption

It is mainly caused by orthodontic treatment (3, 38, 46). If resorption is diagnosed during treatment, the magnitude of the applied forces is reduced, although this does not guarantee cessation of the resorption process; alternatively, orthodontic treatment is discontinued for approximately 3–6 months, allowing osteoclastic activity to subside (47, 48). If the diagnosis is made after completion of orthodontic treatment, regular follow-up and radiographic evaluation every 6–12 months are recommended (49) to confirm that resorption has not continued. Endodontic treatment is not required unless pulp necrosis occurs. Overall, the prognosis is excellent, especially when the resorption is small (<2 mm) (2, 33). (Figure 1)

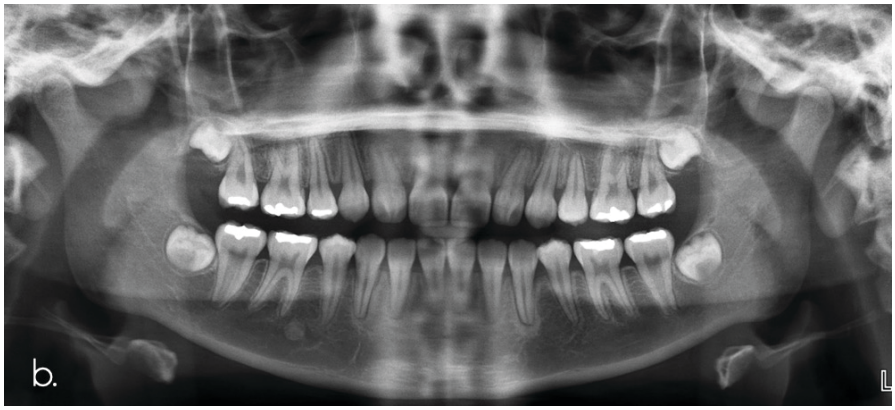


Figure 1. Case of orthodontic apical resorption. a) OPT during treatment, evidencing apical resorption mainly in the maxillary lateral incisors. b) OPT after orthodontic therapy with increased resorption in the maxillary lateral incisors.

10.59987/ads/2026.1. 152-158

External inflammatory apical resorption

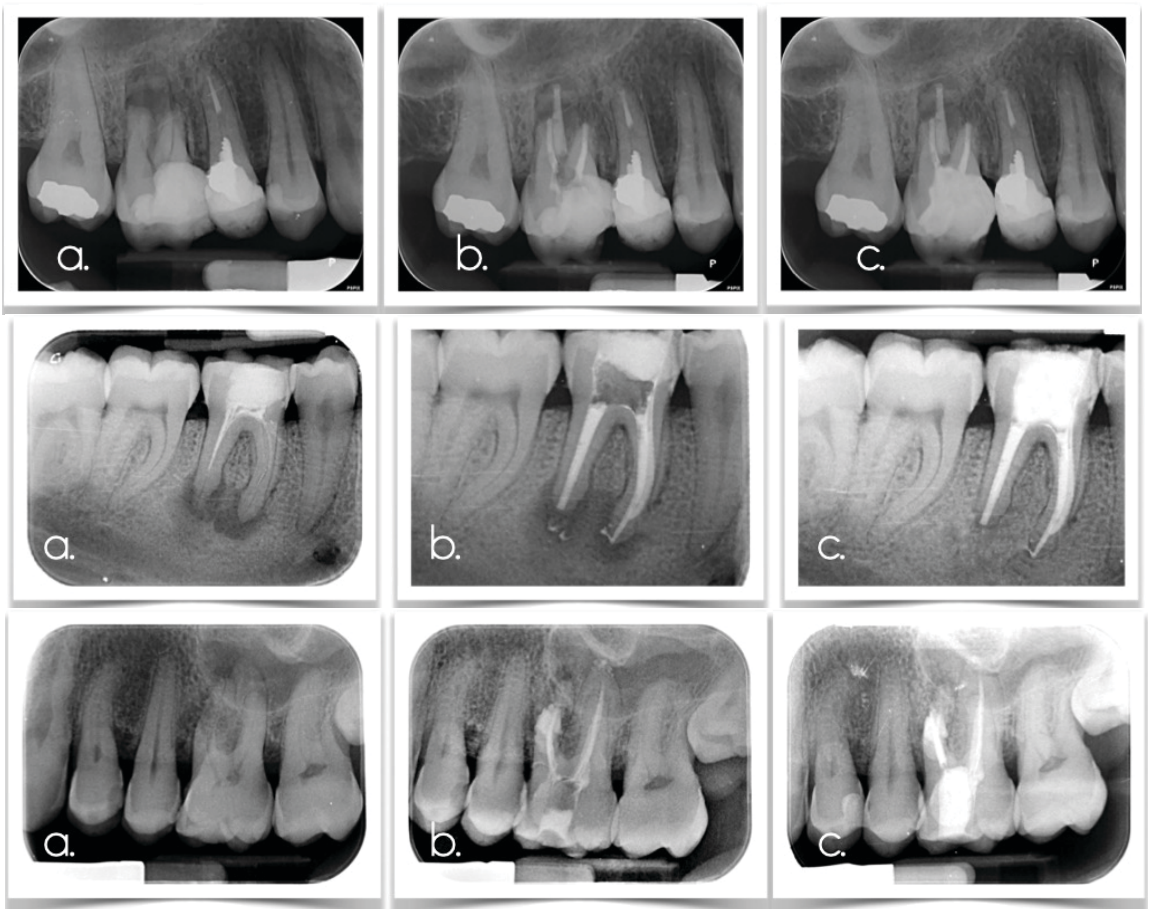
It is caused by a microbial stimulus, namely, pulpal infection. The goal is to eliminate this microbial irritation. In permanent teeth with a closed apex, treatment includes endodontic therapy with chemomechanical preparation using sodium hypochlorite and EDTA, followed by placement of calcium hydroxide paste inside the canal for 2–3 weeks (14, 48, 50). Final root canal treatment is performed when radiographic signs of inflammation subside.

The management of necrotic permanent teeth with an open apex represents one of the greatest challenges in endodontic therapy due to incomplete root development and increased fragility of the canal walls (14). Regenerative endodontics has emerged as an innovative treatment approach, aiming to revascularize and regenerate pulp tissue (50), allowing continued root development and strengthening the tooth's structural integrity (51). Its success depends on strict case-selection criteria, such as patient age (6–17 years), presence of an open apex (>1.1 mm), the ability to induce bleeding, and the absence of systemic factors that impair healing (52).

When these criteria are not met, or canal disinfection is not achievable, apexification using calcium hydroxide, MTA, or bioceramic materials remains the preferred option, offering high success rates (>90%) without promoting further root development (53, 54).

Additionally, biological techniques such as PRP (Platelet-Rich Plasma) and PRF (Platelet-Rich Fibrin) have shown promising results, as their growth factors enhance tissue regeneration and restoration of apical morphology (55, 56). According to Peter E. Murray (2022), PRP/PRF techniques outperform simple blood-clot revascularization (BCR) in achieving apical closure, periapical healing, and root elongation. However, their outcomes remain unpredictable, and for this reason, they are not widely adopted in the scientific community (57).

Therefore, the selection of the appropriate treatment must be based on differential diagnostic criteria that take into account: root maturity, pulp status, patient health and cooperation, and the clinician's capabilities. In addition, several studies have examined pharmaceutical agents such as doxycycline, acetazolamide (58), ascorbic acid, and alendronate for the prevention or management of inflammatory resorption (59, 60, 61), but not for replacement resorption. Ledermix, which contains corticosteroids and antibiotics, has been shown to prevent inflammatory resorption, while Emdogain (62, 63) may contribute to periodontal healing, although its efficacy against ankylosis remains controversial. It should be noted that most of these studies involve replanted teeth and were conducted on animal models, limiting the generalizability of the results to humans (60, 62, 64). (Figures 2-4)



Figures 2-4. Cases of Inflammatory apical resorption. a) Initial X-ray with evident reduction of the apical length combined with periapical lesion. b) After endodontic therapy. c) Follow-up evidencing periapical healing and stabilization of resorbed root length.

Apical resorption of traumatic etiology

According to Andreasen (1), following lateral luxation injury or avulsion, 78% of teeth will develop replacement resorption and become ankylosed. This occurs mainly in fully formed apices (33). It is not related to microorganisms within the pulp and is a hormone-dependent process. In such cases, the lesion cannot be halted. When dentin loss exceeds 20%, ankylosis develops. The prognosis is poor, and the tooth will eventually be lost over time.

The term transient apical resorption also appears in the literature. This entity is observed in 23% of cases at 3 months and in 86% at 1 year after trauma. Radiographically, it appears as root shortening or blunting and is often misdiagnosed by clinicians as inflammatory resorption. In reality, it is an organismic healing process; therefore, periodic monitoring is recommended, especially if the patient exhibits no other symptoms. (Figure 5)

Discussion

Apical root resorption is a multifactorial process influenced by mechanical, microbial, and traumatic stimuli. The literature review highlights considerable discrepancies in reported prevalence, largely due to heterogeneity in study design and imaging modalities. Older studies based on panoramic radiographs underestimate the extent of lesions, whereas CBCT and histological analyses reveal substantially higher rates, underscoring the need for standardized diagnostic criteria.

Although Andreasen's classification remains the most widely accepted, it does not fully encompass the spectrum of clinical presentations. More recent systems aim to integrate etiological and morphological parameters, yet universal adoption remains limited, limiting the comparability of findings across studies. Therapeutically, regenerative endodontics and adjunctive pharmacological agents show promise, but their predictability and long-term outcomes remain uncertain, restricting routine clinical use. Overall, early diagnosis and accurate etiological assessment remain the most decisive factors for successful management.

Conclusions

Apical resorption is caused by various factors, such as bacterial infection, orthodontic treatment, trauma, and chemical agents, affecting the periodontal ligament and the pulp. Several categories of resorption exist, distinguished according to etiology and location.

The prevalence of resorption varies by population and clinical conditions, with external inflammatory resorption being the most common form.

Diagnosis is primarily achieved through radiographic methods, with CBCT providing the highest diagnostic accuracy, particularly for small lesions.

Depending on the type and cause of resorption, treatment may include conservative endodontic procedures, localized removal of resorptive defects with specialized materials (e.g., glass ionomers or bioceramics), or tooth extraction (65).

Accurate classification of resorption is crucial for distinguishing between different pathological conditions and selecting the appropriate treatment.

Regenerative endodontics using stem cells, growth factors, and scaffolds (PRP, PRF) may enhance healing and regeneration of the apical area, providing an alternative approach for necrotic teeth with immature apices.

Future Directions

1. Unified Classification System
2. Targeted Biomaterials and Pharmacologic Agents
3. Genetic and Systemic Risk Factors
4. Artificial Intelligence in Detection

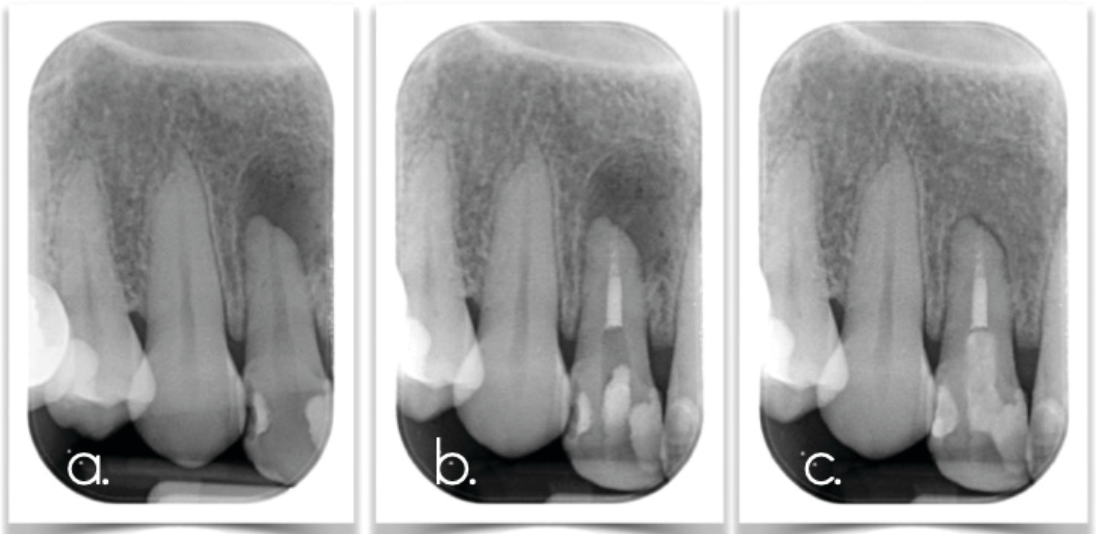


Figure 5. Case of apical resorption after trauma. a) Initial X-ray with evident reduction of the apical length combined with peri-apical lesion in the lateral incisor and apical resorption asymptomatic in the canine. b) After endodontic therapy. c) Follow-up evidencing periapical healing and stabilization of resorbed root length.

10.59987/ads/2026.1. 152-158

References

1. Andreasen, J. O. External root resorption: its implication in dental traumatology, paedodontics, periodontics, orthodontics and endodontics. Int Endod J 1985, (2), 109-18. DOI: 10.1111/j.1365-2591.1985.tb00427.x.
2. Tronstad, L. Root resorption--etiology, terminology and clinical manifestations. Endod Dent Traumatol 1988, 4(6), 241-52. DOI: 10.1111/j.1600-9657.1988.tb00642.x.
3. BREZNIAK, N.; WASSERSTEIN, A. Orthodontically induced inflammatory root resorption. Part I: The basic science aspects. The Angle orthodontist 2002, 72(2), 175-9. DOI: 10.1043/0003-3219(2002)072<0175:OIIRRP>2.0.CO;2.
4. KUTTLER, Y. Microscopic investigation of root apexes. J Am Dent Assoc 1955, 50(5), 544-52. DOI: 10.14219/jada.archive.1955.0099.
5. Hammarstrom, L.; Lindskog, S. General morphological aspects of resorption of teeth and alveolar bone. Int Endod J 1985, 18(2), 93-108. DOI: 10.1111/j.1365-2591.1985.tb00426.x.
6. Lindskog, S.; Hammarström, L. Evidence in favor of an anti-invasion factor in cementum or periodontal membrane of human teeth. Scand J Dent Res 1980, 88(2), 161-3. DOI: 10.1111/j.1600-0722.1980.tb01209.x.
7. Gartner, A. H.; Mack, T.; Somerlott, R. G.; Walsh, L. C. Differential diagnosis of internal and external root resorption. J Endod 1976, 2(11), 329-34. DOI: 10.1016/S0099-2399(76)80071-4.
8. TROPE, M. Root resorption due to Dental Trauma. Endodontic Topics 2002, 1(1), 79-100. DOI: 10.1034/j.1601-1546.2002.10106.x.
9. Heithersay, G. S. Invasive cervical resorption following trauma. Aust Endod J 1999, 25(2), 79-85. DOI: 10.1111/j.1747-4477.1999.tb00094.x.
10. Patel, S. New dimensions in endodontic imaging: Part 2. Cone beam computed tomography. Int Endod J 2009, 42(6), 463-75. DOI: 10.1111/j.1365-2591.2008.01531.x.
11. Darcey, J.; Qualtrough, A. Resorption: part 1. Pathology, classification and aetiology. Br Dent J 2013, 214(9), 439-51. DOI: 10.1038/sj.bdj.2013.431.
12. Kanas, R. J.; Kanas, S. J. Dental root resorption: a review of the literature and a proposed new classification. Compend Contin Educ Dent 2011, 32(3), e38-52. DOI: not available.
13. Langeland, K. Tissue response to dental caries. Endod Dent Traumatol 1987, 3(4),149-71. DOI: 10.1111/j.1600-9657.1987.tb00619.x.
14. Nair, P. N. Pathogenesis of apical periodontitis and the causes of endodontic failures. Crit Rev Oral Biol Med 2004, 15(6), 348-81. DOI: 10.1177/154411130401500604.
15. Simon, J. H.; Glick, D. H.; Frank, A. L. The relationship of endodontic-periodontic lesions. J Periodontol 1972, 43(4), 202-8. DOI: 10.1902/jop.1972.43.4.202.
16. Bates, S. Absorption of the alveolar process. American Journal of Dental Science 1856, 2(6), 380-384. DOI: not available.
17. KETCHAM, A. H. A Progress Report of an Investigation of Apical Root Resorption of Vital Permanent Teeth. International Journal of Orthodontia, Oral Surgery and Radiography 1929, 15, 310-328. DOI: 10.1016/S0099-6963(29)90554-8.
18. KETCHAM, A. H. A Preliminary Report of an Investigation of Apical Root Resorption of Permanent Teeth. International Journal of Orthodontia, Oral Surgery and Radiography 1927, 13, 97-127. DOI: 10.1016/S0099-6963(27)90316-0.
19. RUDOLPH, C. E. A Comparative Study of Root Resorption in Permanent Teeth. The Journal of the American Dental Association (1922) 1936, 23, 822-826. DOI: 10.14219/jada.archive.1939.0381.
20. RUDOLPH, C. E. An Evaluation of Root Resorption Occurring During Orthodontic Treatment. Journal of Dental Research 1940, 19: 367-371. DOI: 10.1177/00220345400190040301.
21. HENRY, J. L.; WEINMANN, J. P. The pattern of resorption and repair of human cementum. J Am Dent Assoc 1951, 42(3), 270-90. DOI: 10.14219/jada.archive.1951.0045.
22. MASSLER, M.; MALONE, A. J. Root resorption in human permanent teeth, a roentgenographic study. American Journal of Orthodontics 1954, 40, 619-633. DOI: 10.1016/0002-9416(54)90070-6.
23. VANJA, O. G.; SLAVOLJUB, Z. Frequency of the external resorptions of tooth roots. Srpski arhiv za celokupno lekarstvo 2004, 132(5-6), 152-6. DOI: 10.2298/sarh0406152o.
24. Newman, W. G. Possible etiologic factors in external root resorption. Am J Orthod 1975, 67(5), 522-39. DOI: 10.1016/0002-9416(75)90298-5.
25. Kaley, J.; Phillips, C. Factors related to root resorption in edgewise practice. Angle Orthod 1991, 61(2), 125-32. DOI: 10.1043/0003-3219(1991)061<0125:FRTRRI>2.0.CO;2.
26. Vanzant, H. C. Root resorption during orthodontic treatment. American Journal of Orthodontics, 1954. 40(6), 457. DOI: not available.
27. Mirabella, A. D.; Artun, J. Prevalence and severity of apical root resorption of maxillary anterior teeth in adult orthodontic patients. Eur J Orthod 1995, 17(2), 93-9. DOI: 10.1093/ejo/17.2.93.
28. Sameshima, G. T.; Sinclair, P. M. Predicting and preventing root resorption: Part I. Diagnostic factors. Am J Orthod Dentofacial Orthop 2001, 119(5), 505-10. DOI: 10.1067/mod.2001.113409.
29. Brezniak, N.; Wasserstein, A. Root resorption after orthodontic treatment: Part 1. Literature review. Am J Orthod Dentofacial Orthop 1993, 103(1), 62-6. DOI: 10.1016/0889-5406(93)70106-X.
30. BĂNICĂA, C.; MARINESCU, I. R.; GHEORGHE, D. N.; TRUSCĂ, A. G.; DRĂGHICI, E. C.; MERCUT, V.; POPESCU, S. M. ROOT RESORPTION PREVALENCE IN ADULTS FROM DOLJ COUNTY, ROMANIA – A RADIOLOGICAL EVIDENCE. Romanian Journal of Oral Rehabilitation 2028, 10: 170-179. DOI: not available.
31. VIER, F. V.; FIGUEIREDO, J. A. P. Internal apical resorption and its significance in endodontics. International Endodontic Journal 2004, 37(11), 300–308. DOI: 10.1111/j.1365-2591.2004.00830.x.
32. Patel, S.; Dawood, A.; Wilson, R.; Horner, K.; Mannocci, F. The detection and management of root resorption lesions using intraoral radiography and cone beam computed tomography - an in vivo investigation. Int Endod J 2009, 42(9), 831-8. DOI: 10.1111/j.1365-2591.2009.01592.x.
33. Fuss, Z.; Tsesis, I.; Lin, S. Root resorption--diagnosis, classification and treatment choices based on stimulation factors. Dent Traumatol 2003, 19(4), 175-82. DOI: 10.1034/j.1600-9657.2003.00192.x.
34. Gunraj, M. N. Dental root resorption. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999, 88(6), 647-53. DOI: 10.1016/s1079-2104(99)70002-8.
35. Brezniak, N.; Wasserstein, A. Root resorption after orthodontic treatment: Part 2. Literature review. Am J Orthod Dentofacial Orthop 1993, 103(2), 138-46. DOI: 10.1016/S0889-5406(05)81763-9.
36. Soğur, E.; Baksi, B. G.; Gröndahl, H. G. Imaging of root canal fillings: a comparison of subjective image quality between limited cone-beam CT, storage phosphor and film radiography. Int Endod J 2007, 40(3), 179-85. DOI: 10.1111/j.1365-2591.2007.01204.x.
37. Sameshima, G. T.; Asgarifar, K. O. Assessment of root resorption and root shape: periapical vs panoramic films. Angle Orthod 2001, 71(3), 185-9. DOI: 10.1043/0003-3219(2001)071<0185:AORRAR>2.0.CO;2.
38. Remington, D. N.; Joondeph, D. R.; Artun, J.; Riedel, R. A.; Chapko, M. K. Long-term evaluation of root resorption occurring during orthodontic treatment. Am J Orthod Dentofacial Orthop 1989, 96(1), 43-6. DOI: 10.1016/0889-5406(89)90227-8.
39. Leach, H. A.; Ireland, A. J.; Whaites, E. J. Radiographic diagnosis of root resorption in relation to orthodontics. Br Dent J 2001, 190(1), 16-22. DOI: 10.1038/sj.bdj.4800870.
40. Creanga, A. G.; Geha, H.; Sankar, V.; Teixeira, F. B.; McMahan, C. A.; Noujeim, M. Accuracy of digital periapical radiography and cone-beam computed tomography in detecting external root resorption. Imaging Sci Dent 2015, 45(3), 153-8. DOI: 10.5624/isd. 2015.45.3.153.
41. Liedke, G. S.; Silveira, H. E.; Silveira, H. L.; Dutra, V.; & Figueiredo, J. A. Influence of cone beam computed tomography on the diagnosis of simulated external root resorption. Journal of Endodontics 2009, 35(11), 1530-

1533. DOI: 10.1016/j.joen.2009.08.001.
42. Chapnick, L. External root resorption: an experimental radiographic evaluation. Oral Surg Oral Med Oral Pathol 1989, 67(5), 578-82. DOI: 10.1016/0030-4220(89)90276
43. Heo, M. S.; Lee, S. S.; Lee, K. H.; Choi, H. M.; Choi, S. C.; Park, T. W. Quantitative analysis of apical root resorption by means of digital subtraction radiography. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001, 91(3), 369-73. DOI: 10.1067/moe. 2001.113592.
44. Levander, E.; Bajka, R.; Malmgren, O. Early radiographic diagnosis of apical root resorption during orthodontic treatment: a study of maxillary incisors. Eur J Orthod 1998, 20(1), 57-63. DOI: 10.1093/ejo/20.1.57.
45. Chan, E. K.; Darendeliler, M. A. Exploring the third dimension in root resorption. Orthod Craniofac Res 2004, 7(2), 64-70. DOI: 10.1111/j.1601-6343.2004.00280.x.
46. Zahed Zahedani, S.; Oshagh, M.; Momeni Danaei, Sh.; Roeinpeikar, S. A Comparison of Pical Root Resorption in Incisors after Fixed Orthodontic Treatment with Standard Edgewise and Straight Wire (MBT) Method. J Dent (Shiraz) 2013, 14(3), 103-10. DOI: not available.
47. Mundy, C. R.; Altman, A. J.; Gondek, M. D.; Bandelin, J. G. Direct resorption of bone by human monocytes. Science 1977, 196 (4294), 1109-11. DOI: 10.1126/science. 16343.
48. LAUX, M.; ABBOTT, P. V.; PAJAROLA, G.; NAIR, P. N. Apical inflammatory root resorption: a correlative radiographic and histological assessment. International endodontic journal 2000, 33(6), 483-93. DOI: 10.1046/j.1365-2591.2000.00338.x.
49. Sameshima, G. T.; Sinclair, P. M. Predicting and preventing root resorption: Part II. Treatment factors. Am J Orthod Dentofacial Orthop 2001, 119(5), 511-5. DOI: 10.1067/mod.2001.113410.
50. SELTZER, S.; BENDER, I. B.; ZIONTZ, M. The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp. Oral Surg Oral Med Oral Pathol 1963, 16:846-71 contd. doi: 10.1016/0030-4220(63)90323-2.
51. Banchs, F.; Trope, M. Revascularization of immature permanent teeth with apical periodontitis: new treatment protocol? J Endod 2004, 30(4), 196-200. DOI: 10.1097/00004770-200404000-00003.
52. GEISLER, T. M. Clinical considerations for regenerative endodontic procedures. American Association of Endodontists 2021, 56(3), 603-26 DOI:10.1016/j.cden.2012.05.010.
53. CVEK, M. Prognosis of luxated non-vital maxillary incisors treated with calcium hydroxide and filled with gutta-percha. Endodontics & dental traumatology 1992, 8(2), 45-55. DOI: 10.1111/j.1600-9657.1992.tb00228.x.
54. Torabinejad, M.; Chivian, N. Clinical applications of mineral trioxide aggregate. J Endod 1999, 25(3), 197-205. DOI: 10.1016/S0099-2399(99)80142-3.
55. MURRAY, P.E.; GARCIA-GODOY, F.; HARGREAVES, K. M. Regenerative endodontics: a review of current status and a call for action. Journal of Endodontics 2007, 33(4), 377-90. DOI: 10.1016/j.joen.2006.09.013.
56. Jadhav, G. R.; Shah, N.; Logani, A. Platelet-rich plasma supplemented revascularization of an immature tooth associated with a periapical lesion in a 40-year-old man. Case Rep Dent 2014, 2014:479584. DOI: 10.1155/2014/479584.
57. Rebimbas Guerreiro, S.; Marto, C. M.; Paula, A.; Pereira, J. R. A.; Carrilho, E.; Marques-Ferreira. M.; Vicente Paulo, S. Platelet-Rich Plasma and Platelet-Rich Fibrin in Endodontics: A Scoping Review. Int J Mol Sci 2025, 26(12), 5479. DOI: 10.3390/ijms26125479.
58. Mori, G. G.; Garcia, R. B.; Gomes de Moraes, I. Morphometric and microscopic evaluation of the effect of solution of acetazolamide as an intracanal therapeutic agent in late reimplanted rat teeth. Dent Traumatol 2006, 22(1), 36-40. DOI: 10.1111/j.1600-9657.2006.00416.x.
59. Sato, M.; Grasser, W.; Endo, N.; Akins, R.; Simmons, H.; Thompson, D. D.; Golub, E.; Rodan, G. A. Bisphosphonate action. Alendronate localization in rat bone and effects on osteoclast ultrastructure. J Clin Invest 1991, 88(6), 2095-105. doi: 10.1172/JCI115539.
60. LEVIN, M. D.; JONG, G. The Use of CBCT in the Diagnosis and Management of Root Resorption. 3D Imaging in Endodontics 2016, 131-143. DOI:10.1007/978-3-319-31466-2_7.
61. Komatsu, K.; Shimada, A.; Shibata, T.; Shimoda, S.; Oida, S.; Kawasaki, K.; Nifuji, A. Long-term effects of local pretreatment with alendronate on healing of replanted rat teeth. J Periodontal Res 2008, 43(2), 194-200. DOI: 10.1111/j.1600-0765.2007.01012.x.
62. Bryson, E. C.; Levin, L.; Banchs, F.; Abbott, P. V.; Trope, M. Effect of immediate intracanal placement of Ledermix Paste(R) on healing of replanted dog teeth after extended dry times. Dent Traumatol 2002, 18(6), 316-21. DOI: 10.1034/j.1600-9657.2002.00142.x.
63. Kenny, D. J.; Barrett, E. J.; Johnston, D. H.; Sigal, M. J.; Tenenbaum, H. C. Clinical management of avulsed permanent incisors using Emdogain: initial report of an investigation. J Can Dent Assoc 2000, 66(1), 21. DOI: not available.
64. Andreasen, J. O. Periodontal healing after replantation and autotransplantation of incisors in monkeys. Int J Oral Surg 1981, 10(1), 54-61. DOI: 10.1016/s0300-9785(81)80008-7.
65. Mincik, J.; Urban, D.; Timkova, S. Clinical Management of Two Root Resorption Cases in Endodontic Practice. Case Rep Dent 2016, 2016:9075363. DOI: 10.1155/2016/9075363.

10.59987/ads/2026.1. 152-158