Dental Traumatology

Dental Traumatology 2012; **28**: 296–301; doi: 10.1111/j.1600-9657.2011.01089.x

The effect of frequency of calcium hydroxide dressing change and various pre- and inter-operative factors on the endodontic treatment of traumatized immature permanent incisors

Ghaeth H. Yassen[1,2], Judith Chin[1], Ahmed G. Mohammedsharif [2], Saif S. Alsoufy[2], Samer S. Othman[2], George Eckert[3]

[1]Indiana University School of Dentistry, Indianapolis, IN, USA; [2]University of Mosul, College of Dentistry, Mosul, Iraq; [3]Indiana University School of Medicine, Indianapolis, IN, USA

Correspondence to: Dr Ghaeth Yassen BDS, MSc, Indiana University School of Dentistry, 1121 W. Michigan Street, Indianapolis, IN 46202, USA
Tel.: +1-317-437-2240
Fax: +1-317-274-5425
e-mail: gyassen@iupui.edu

Accepted 23 October, 2011

Abstract – *Aim*: The objectives of this clinical study were as follows: (i) to determine the effect of frequency of calcium hydroxide [$Ca(OH)_2$] dressing change on the apical barrier formation in immature permanent incisors with necrotic pulps and (ii) to investigate the effect of various clinical factors before and during treatment that may be associated with the frequency of $Ca(OH)_2$ dressing changes. *Methods*: The study involved 21 healthy subjects, 8–12 years old. Twenty-three immature traumatized permanent maxillary central incisors were treated using $Ca(OH)_2$ powder mixed with barium sulfate and distilled water. The progress of barrier formation was reviewed after 6 months of first placement of $Ca(OH)_2$ and then every 3 months until the detection of an apical barrier. Clinical and radiographic evaluations were performed before and after treatment. Data were evaluated using a chi-square test. *Results*: Apical barrier formation was successful for all 23 teeth. Seventeen teeth (74%) needed only a single application of $Ca(OH)_2$, while six teeth (26%) required more than one application. The average time of apical barrier formation was 30 weeks, and the mean number of $Ca(OH)_2$ dressing changes was 1.3. A significant positive association was found between teeth that presented with displacement and the number of $Ca(OH)_2$ dressing changes ($P = 0.004$). *Conclusion*: An initial 6-month application of $Ca(OH)_2$ dressing followed by 3-month replacements (usually in teeth presenting with displacement and/or sinus tracts) may be successfully used in apexification treatment. This would assist in reducing the number of $Ca(OH)_2$ dressing changes, number of appointments, cost of treatment and radiation exposure.

Apexification is a method to induce a calcified barrier in a root with an open apex or the continued apical development of an incomplete root in teeth with a necrotic pulp (1). The use of calcium hydroxide [$Ca(OH)_2$] in apexification was first introduced by Kaiser (2) and Frank (3) in the 1960s. It appears that the high pH of non-setting $Ca(OH)_2$ is an important factor in stimulating the formation of mineralized and fibrous tissue in the apical part of the root canal (4). Furthermore, it has been demonstrated that apical barrier formation is more successful in the absence of microorganisms (5) and the antibacterial property of $Ca(OH)_2$ has been well established (6).

Some studies claim that certain factors may affect the speed of apical barrier formation, such as patient age, apical width, injury type, presence of an abscess, presence of an apical radiolucency at the start of treatment and inter-appointment symptoms (7–11). Controversy exists as to whether, or how often, the $Ca(OH)_2$ dressing should be changed. Chawla (12) found that a single application is sufficient to induce apical closure in 92.3% of treated teeth. Furthermore, Chosack et al. (13) in a study on monkeys' teeth found that after the initial root filling with $Ca(OH)_2$, there is no advantage to be gained by repeated root filling, either monthly or after 3 months. Mackie (14) suggested that the initial change be at 1 month and subsequent 3-month intervals. Ghose et al. (8) advocated changing at 1 month and again at 6–8 month intervals until apical barrier formation takes place. Other authors have suggested $Ca(OH)_2$ to be replaced only when symptoms develop or when the material appears to have washed out of the canal when viewed radiographically (10, 15).

Some clinical studies have suggested that the frequency of change of the $Ca(OH)_2$ dressing may increase the speed of apical bridge formation (7, 9, 16). On the other hand, a relatively recent study performed on 6-month-old dogs concluded that monthly renewal of $Ca(OH)_2$ paste significantly reduces the occurrence of apexification, and the replacement of $Ca(OH)_2$ paste was not necessary for apexification to occur (17).

The objectives of this clinical study were to determine the effect of frequency of change of $Ca(OH)_2$ dressing on apical barrier formation in immature permanent incisors with necrotic pulps. Additionally, we noted the effects of various clinical factors before and during treatment that may be associated with the frequency of $Ca(OH)_2$ dressing changes.

Methods

This clinical study was approved by the Research Ethics Committee of the College of Dentistry, Mosul University, Mosul, Iraq as well as the IUPUI/Clarian Institutional Review Board in Indianapolis, IN, USA (EX1004-37). The study was performed between January 2006 and July 2010. The study involved 22 healthy cooperative children, fifteen men and seven women, age 8–12 years. Each study subject visited the College of Dentistry's Pediatric Department at Mosul University in Iraq. To be included in the study, the subject had to be healthy without any systemic conditions, cooperative and had to have suffered loss of vitality following trauma to at least one permanent incisor resulting in incomplete or arrested root development with an open apex. Furthermore, a signed informed consent was obtained from each subject's parent or guardian. The exclusion criteria were as follows: medically compromised children, parents or guardians refusing to sign the informed consent, necrosis of permanent incisors caused by caries, any sign of active pathological root resorption, teeth with root fractures, non-cooperating children, difficulty with dental dam isolation or teeth with poor prognosis requiring immediate extraction.

Of the 22 subjects enrolled in our study, one male subject stopped attending after the initial application of $Ca(OH)_2$. Therefore, this study was completed with 21 subjects (14 men and seven women) with 23 maxillary central incisor teeth treated.

Comprehensive medical and dental records were taken for each child, and details of the dates along with the circumstances of the original injury were recorded. A clinical examination was carried out (inspection, palpation, percussion and mobility). Teeth were classified as displaced (luxated) or non-displaced. No teeth were avulsed among the study subjects. Radiological examination was completed using periapical radiographs. Vitality tests were also performed, using electrical and thermal stimulation. The degree of root development prior to treatment was assessed, according to Moorrees et al. (18).

The clinical treatment protocol was established based on clinical information from an initial pilot study. After local anesthetic was given, a dental dam was applied. A lingual access opening was made in the tooth sufficient to allow adequate debridement of the root canal, and then, the canal was widened cervically (when necessary) until the entrance to the canal was thoroughly visualized. An endodontic file was placed in the canal, and the working length was determined by a conventional periapical radiograph. Gentle debridement of the canal was performed using a size 25 K file (Dentsply, Maillefer, Ballaigues, Switzerland) to remove the remaining pulp tissue or necrotic debris, and the canal was irrigated with 0.5% sodium hypochlorite.

The canal was gently prepared using hand K files (Dentsply, Maillefer). The K file size used ranged from 60 to 120 depending on the width of the canal. Care was taken not to exceed working length to minimize damage to apical tissues. Filing was augmented with copious irrigation with 0.5% sodium hypochlorite. If purulent exudate was present, one or more further appointments were given until the acute signs/symptoms were controlled. Furthermore, oral antibiotics (amoxicillin) were also given during this time for all cases with purulent exudate. The number of the appointments from the commencement of the endodontic treatment until the application of $Ca(OH)_2$ paste ranged between two and four appointments. At each of these appointments, the canal was irrigated with 0.5% sodium hypochlorite, dried using paper points, a sterile cotton pellet was placed inside the chamber floor, and the access cavity was sealed with zinc oxide–eugenol IRM cement (Dentsply Caulk, Milford, DE, USA).

The $Ca(OH)_2$ dressing was prepared according to the manufacturers recommendations by mixing $Ca(OH)_2$ powder (Dentonics; Master-Dent, Monroe, NC, USA) with barium sulfate powder (Sigma-Aldrich, Gillingham, UK) in a ratio of 8:1 (19). The powder mixture was then mixed with distilled water (powder/liquid ratio of 3:4) (20). Placement of the $Ca(OH)_2$ dressing within the canal was performed after canal irrigation with sterile saline and dried using paper points. $Ca(OH)_2$ was then delivered into the canal using a Lentulo spiral rotary file (Dentsply, Maillefer) with sterile paper point condensation. A periapical radiograph was used to confirm correct placement of the paste. To facilitate treatment for future endodontic appointments, a sterile cotton pellet was placed on the chamber floor. The canal was sealed with glass ionomer cement (Ionofil, Voco, Germany).

The progress of barrier formation was examined 6 months after the first placement of $Ca(OH)_2$ and every 3 months thereafter. In case of any intra-appointment symptoms or the loss of the interim restoration, the parent and study subject were instructed to return to the clinic. At each follow-up appointment, clinical signs and symptoms were recorded, and the $Ca(OH)_2$ dressing was removed by flushing the canal with 5 ml of 0.5% sodium hypochlorite. If an apical barrier had not formed, another $Ca(OH)_2$ dressing was placed as described. This was repeated until the presence of an apical barrier or apical stop was found.

The positive clinical detection of an apical barrier was determined by gently probing the apical region with an endodontic file (11). The file was at least four sizes smaller than the largest file used during instrumentation.

Radiographic verification of an apical stop was considered positive when there was evidence of a normal periodontal space, a decrease in size of the periapical lesion compared with pre-operative radiographs and no evidence of inflammatory external resorption. The apical barrier was radiographically classified as being apically placed if it was within 1 mm of the apex and coronal to the apex in other cases (7). Apexification treatment was deemed to be successfully diagnosed if both tactile and radiograph assessment were positive. The root canal was then obturated using gutta-percha (Dentsply, Maillefer) and root canal sealer EWT (Dentsply, Maillefer), using either lateral condensation or a thermoplastic technique. After obturation, a minimum of a 3-month control period for each tooth was followed. Furthermore, if a radiolucency was present at the time of obturation, the subject was seen at subsequent 3-month intervals until the reduction or complete healing of the radiolucent lesion.

The timing of the visit on which the barrier was first detected was recorded, and the period from the application of $Ca(OH)_2$ dressing was calculated in weeks. The number of changes of $Ca(OH)_2$ dressing was also recorded. All the treatments were carried out by two experienced clinicians, and each subject was assigned randomly to one of the clinicians. The data were analyzed using SAS (SAS version 9.1; SAS Institute, Inc., Cary, NC, USA). An exact 95% confidence interval was calculated to estimate the percentage of subjects requiring a single $Ca(OH)_2$ dressing. A chi-square test was used to compare different clinical variables between teeth that required a single $Ca(OH)_2$ dressing to complete apexification treatment and those that required more than one. The difference was statistically significant if $P < 0.05$.

Results

The apexification treatment (apical barrier formation) was successful for all 23 teeth (100%). Examples of treatments and outcomes are depicted in Fig. 1a–f. Table 1 describes the different clinical variables that were measured in the study. The time from application of $Ca(OH)_2$ to barrier formation averaged 30 weeks with a SD of 6 weeks. Seventeen teeth required only a single $Ca(OH)_2$ dressing (74%), five teeth (22%) required two applications, while only one tooth (4%) required three applications. The average number of $Ca(OH)_2$ dressing replacements was 1.3. The range of follow-up period after apical barrier formation was 3 months to 3 years, and the average follow up was 13 months. Five cases were not followed up until the complete resolution of periapical radiolucency. No intra-appointment symptoms were reported in any subject during the period of $Ca(OH)_2$ paste application.

Table 2 details all the clinical variables that were measured with respect to the number of $Ca(OH)_2$ dressings needed until apical barrier formation was detected. A statistically significant positive association was found between displacement at time of trauma and number of $Ca(OH)_2$ dressing changes ($P = 0.004$). Furthermore, a trend of significant positive association was found between the presence of sinus tract at the beginning of the treatment and the number of $Ca(OH)_2$ dressing changes ($P = 0.057$). No significant difference was found between the two clinicians performing the treatment regarding the number of $Ca(OH)_2$ dressing changes ($P = 0.409$). In this study, no significant associations were found between the number of $Ca(OH)_2$ dressing changes required to complete treatment and

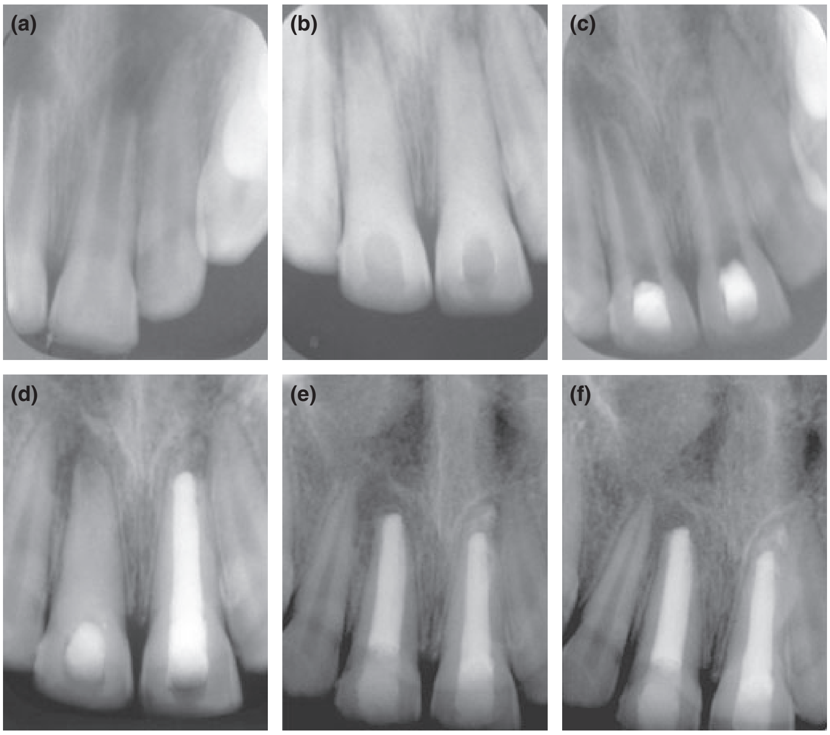

Fig. 1. Treatment and outcome for a subject requiring apexification. (a) A pre-operative radiograph showing two traumatized necrotic maxillary permanent incisors in a 12-year-old child. (b) The same teeth after application of calcium hydroxide dressing. (c) Twenty-six weeks after application of calcium hydroxide paste with apical barrier formation of tooth number 21. (d) Twenty-six weeks after application of calcium hydroxide paste, obturation of tooth number 21 and change of calcium hydroxide dressing for tooth number 11. (e) Thirteen weeks after application of the second calcium hydroxide dressing for tooth number 11, apical closure and obturation of tooth number 11. (f) Fourteen-week follow up after obturation of tooth number 11.

Table 1. Description of the clinical variables measured in the study

Clinical variable	Categories	*N* (%)
Age	8–9 years	4 (19)
	9–10 years	7 (33)
	10–11 years	6 (28)
	11–12 years	2 (10)
	12 years	2 (10)
Sex	Female	7 (33)
	Male	14 (67)
Displacement at time of trauma	No	15 (65)
	Yes	8 (35)
Degree of apical development	Converging/parallel	2 (9)
	Open	21 (91)
Time elapsed between the occurrence of trauma and start of treatment	<6 months	15 (65)
	6–12 months	5 (22)
	>12 months	3 (13)
Position of apical barrier	Apically placed	16 (70)
	Coronally placed	7 (30)
Operator	First operator	12 (52)
	Second operator	11 (48)
Number of calcium hydroxide dressing changes	1	17 (74)
	2	5 (22)
	3	1 (4)
Presence of radiolucency at start of treatment	No	11 (48)
	Yes	12 (52)
Presence of radiolucency after apical barrier detection	No	15 (65)
	Yes	8 (35)
Follow up until complete resolution of radiolucency	No	5 (22)
	Yes	3 (13)
Presence of sinus tract at beginning of treatment	No	15 (65)
	Yes	8 (35)
Presence of spontaneous pain at beginning of treatment	No	7 (30)
	Yes	16 (70)
Presence of pathological mobility at beginning of treatment	No	22 (96)
	Yes	1 (4)
Tenderness to percussion at beginning of treatment	No	9 (39)
	Yes	14 (61)

subjects' age ($P = 0.306$), gender ($P = 0.687$), degree of apical development ($P = 0.379$), position of apical barrier ($P = 0.226$), time elapsed between the occurrence of trauma and start of treatment ($P = 0.166$), presence of radiolucency at start of treatment ($P = 0.283$), presence of spontaneous pain at beginning of treatment ($P = 0.394$), presence of pathological mobility at beginning of treatment ($P = 0.544$) or tenderness to percussion at beginning of treatment ($P = 0.108$).

Discussion

The successful treatment of traumatized, immature permanent teeth with necrotic pulp presents multiple challenges. In addition to the traditional $Ca(OH)_2$ apexification technique, recently, the use of mineral trioxide aggregate (MTA) and pulp revascularization have been introduced (21, 22). However, at present, there is no prospective long-term outcome study that compares the success rate of the relatively new techniques with that of the traditional $Ca(OH)_2$ apexification (23). Cost and availability of MTA are also factors in some areas of the world. $Ca(OH)_2$ is readily available, easy to use and relatively inexpensive. Therefore, $Ca(OH)_2$ apexification is still a widely used clinical procedure (24).

The high success rate of $Ca(OH)_2$ apexification obtained in this study was similar to those previously reported in other studies ranging from 74% to 100% (7, 9–11, 25, 26). In the present study, the formed apical barrier was apically positioned (1 mm from the apex) in 16 treated teeth (70%); while it was coronally positioned (more than 1 mm from the apex) in seven treated teeth (30%). Furthermore, no significant relationship was found between the number of $Ca(OH)_2$ dressing changes and barrier position ($P = 0.226$). This finding agrees with Kinirons and colleagues (7), but contradicts Finucane & Kinirons (16), who found significant positive association between the frequency of $Ca(OH)_2$ changes and apically located barriers.

In this study, the average number of $Ca(OH)_2$ dressing changes was 1.3. This value is close to that reported by Chawla (12) which was 1.1. However, it is lower than those reported by Finucane & Kinirons (7), Walia et al. (25) and Dominguez Reyes et al. (26) which were 1.9, 1.8 and 3.3, respectively. The average time of apical barrier formation in this study was 30 weeks. In other studies, it ranged between 6.7 and 18.2 months (7, 9–12, 16, 20, 26). The relatively quick formation of apical root barrier in this study may be due to the minimal disturbance of the apical area by avoiding repeated instrumentation and dressing changes (27, 28). The monthly replacement of $Ca(OH)_2$ dressing in a dog apexification model has been shown to decrease the intensity of inflammation in the apical area but significantly delay apical barrier formation compared with single application of $Ca(OH)_2$ (17). On the other hand, studies found that the frequent replacement of $Ca(OH)_2$ paste may shorten the time of apical barrier formation (7, 9, 16).

Some concerns regarding the weakening effect of long-term $Ca(OH)_2$ dressing on an immature root have been reported. Recently, an *in vitro* study indicated that fracture strength of $Ca(OH)_2$-filled immature teeth was significantly decreased after 1 year compared with MTA-treated teeth (29). The relatively short duration of apical barrier formation obtained in this study (30 weeks $\pm$ 6) might decrease the proposed weakening effect of $Ca(OH)_2$ on immature root. $Ca(OH)_2$ apexification technique is associated with some disadvantages such as the long time required for the completion of treatment, the need for patient compliance, chance of coronal leakage and re-infection of the canal system after loss of temporary restorations during treatment and chance of unsuccessful treatment because of undiagnosed root fracture of the traumatized tooth. Additionally, the relatively thin tooth structure after apexification treatment of immature teeth increases the risk of root fracture or loss of final coronal restoration. In this study, high strength zinc oxide–eugenol cement was used in the initial appointments and high strength glass ionomer was used after $Ca(OH)_2$ dressing application to seal the canal to avoid any chance of coronal leakage and re-infection of the canal system. The initial 6-month follow up interval after the first application of $Ca(OH)_2$ followed by 3-month replacements (if needed) suggested in this study may assist in reducing the number of $Ca(OH)_2$

Table 2. Variables associated with number of $Ca(OH)_2$ dressing change required to complete the treatment

Clinical variable	Categories	One dressing *N* (%)	Two or more dressing *N* (%)	*P*-value
Gender	Female	4 (27)	3 (50)	0.306
	Male	11 (73)	3 (50)	
Age	8–10	7 (47)	4 (67)	0.687
	10–12	8 (53)	2 (33)	
Displacement at time of trauma	No	14 (82)	1 (17)	0.004*
	Yes	3 (18)	5 (83)	
Degree of apical development	Converging/parallel	2 (12)	0 (0)	0.379
	Open	15 (88)	6 (100)	
Time elapsed between the occurrence of trauma and start of treatment	<6 months	13 (76)	2 (33)	0.166
	6–12 months	2 (12)	3 (50)	
	>12 months	2 (12)	1 (17)	
Position of apical barrier	Apically placed	13 (76)	3 (50)	0.226
	Coronally placed	4 (24)	3 (50)	
Operator	First operator	8 (47)	4 (67)	0.409
	Second operator	9 (53)	2 (33)	
Presence of radiolucency at start of treatment	No	7 (41)	4 (67)	0.283
	Yes	10 (59)	2 (33)	
Presence of sinus tract at beginning of treatment	No	13 (76)	2 (33)	0.057
	Yes	4 (24)	4 (67)	
Presence of spontaneous pain at beginning of treatment	No	6 (35)	1 (17)	0.394
	Yes	11 (65)	5 (83)	
Presence of pathological mobility at beginning of treatment	No	16 (94)	6 (100)	0.544
	Yes	1 (6)	0 (0)	
Tenderness to percussion at beginning of treatment	No	5 (29)	4 (67)	0.108
	Yes	12 (71)	2 (33)	

*Significant difference; $P < 0.05$.

dressing changes and the number of appointments with subsequent reduction in cost of treatment and radiation exposure for the child.

Some studies have suggested that apexification treatment of displaced or avulsed teeth might be unsuccessful due to either extensive root resorption (9) or the penetration of the apex into the cortical plate and out of its normal physiologic position (30). Therefore, immature teeth with necrotic pulps included in this study were classified as displaced or non-displaced. The present study revealed that $Ca(OH)_2$ apexification treatment was successful among all displaced teeth. However, a significant positive association between tooth displacement at the time of trauma and number of $Ca(OH)_2$ replacements (longer time for barrier formation) was found in this study. This agrees with Finucane & Kinirons (7), who found that teeth with displacement injuries took significantly longer to form an apical bridge. Furthermore, a trend toward significant positive association was found in this study between the presence of a sinus tract at the beginning of treatment and number of $Ca(OH)_2$ dressing changes ($P = 0.057$). Mendoza et al. (31) found that immature teeth with necrotic pulps which presented with sinus tract required considerable increase in the treatment time (20 months).

In the present study, 12 teeth were presented with an apical radiolucency at start of treatment; five of them were not followed until the complete healing of the radiolucency. However, a decrease in size of the periapical lesion compared with pre-operative radiographs was observed in the five cases. Usually, apical barrier formation preceded the complete resolution of the radiolucency. Because it may be possible for an apical barrier to form with the persistence of a chronic bacterial infection, periodic recall examination should be reinforced to insure the complete healing of the periapical area (11). To the best of our knowledge, the present study is the first study to measure the effect of different operators on the outcome of $Ca(OH)_2$ apexification. However, no significant relationship was found ($P = 0.409$).

One limitation of the study is the small sample size; a larger sample size may have given a more significant result and a more accurate conclusion. Another limitation could be the gender distribution with more male participants than female. However, this may reflect the prevalence of dental trauma in the Iraqi children as reported by Noori & Al-Obaidi (32).

Conclusion

The clinical procedure suggested in this study reconfirms that $Ca(OH)_2$ apexification is predictable, highly successful and still an acceptable procedure in treating the traumatized immature permanent incisors. The average time of apical barrier formation was 30 weeks, and the mean number of $Ca(OH)_2$ dressing changes was 1.3. The procedure described may reduce the number of $Ca(OH)_2$ dressing changes, the number of appointments, cost of treatment and radiation exposure. Clinicians should expect that certain conditions might prolong the apexification treatment course such as displaced teeth or presence of a sinus tract at the beginning of treatment. In our study, other variables were not found to affect the treatment outcome significantly. These variables include the following: patient's age, degree of apical development, different operators' skill levels, time elapsed between the occurrence of trauma and the start of treatment, presence of periapical radiolucency, spontaneous pain, pathological mobility or tenderness to percussion.

References

1. American Association of Endodontists. Glossary of endodontic terms, 7th edn. Chicago: American Association of Endodontists; 2003.
2. Kaiser HJ. Management of wide open apex canals with calcium hydroxide. Presented at the 21st Annual Meeting of the American Association of Endodontists, Washington DC April 17 1964.
3. Frank AL. Therapy for the divergent pulpless tooth by continued apical formation. J Am Dent Assoc 1966;72:87–93.
4. Vojinovic O. Induction of apical formation in immature teeth by different endodontic methods of treatment. Experimental pathohistological study. J Oral Rehabil 1974;1:85–97.
5. Ham JW, Patterson SS, Mitchell DF. Induced apical closure of immature pulpless teeth in monkeys. Oral Surg Oral Med Oral Pathol 1972;33:438–49.
6. Kontakiotis E, Nakou M, Georgopoulou M. *In vitro* study of the indirect action of calcium hydroxide on the anaerobic flora of the root canal. Int Endod J 1995;28:285–9.
7. Finucane D, Kinirons MJ. Non-vital immature permanent incisors: factors that may influence treatment outcome. Endod Dent Traumatol 1999;15:273–7.
8. Ghose LJ, Baghdady VS, Hikmat YM. Apexification of immature apices of pulpless permanent anterior teeth with calcium hydroxide. J Endod 1987;13:285–90.
9. Mackie IC, Bentley EM, Worthington HV. The closure of open apices in non-vital immature incisor teeth. Br Dent J 1988;165:169–73.
10. Cvek M. Treatment of non-vital permanent incisors with calcium hydroxide. I. Follow-up of periapical repair and apical closure of immature roots. Odontol Revy 1972;23:27–44.
11. Kleier DJ, Barr ES. A study of endodontically apexified teeth. Endod Dent Traumatol 1991;7:112–7.
12. Chawla HS. Apical closure in a nonvital permanent tooth using one $Ca(OH)_2$ dressing. ASDC J Dent Child 1986;53:44–7.
13. Chosack A, Sela J, Cleaton-Jones P. A histological and quantitative histomorphometric study of apexification of non-vital permanent incisors of vervet monkeys after repeated root filling with a calcium hydroxide paste. Endod Dent Traumatol 1997;13:211–7.
14. Mackie IC. UK National Clinical Guidelines in Paediatric Dentistry. Management and root canal treatment of non-vital immature permanent incisor teeth. Faculty of Dental Surgery, Royal College of Surgeons. Int J Paediatr Dent 1998;8:289–93.
15. Feiglin B. Differences in apex formation during apexification with calcium hydroxide paste. Endod Dent Traumatol 1985;1:195–9.
16. Kinirons MJ, Srinivasan V, Welbury RR, Finucane D. A study in two centres of variations in the time of apical barrier detection and barrier position in nonvital immature permanent incisors. Int J Paediatr Dent 2001;11:447–51.
17. Felippe MC, Felippe WT, Marques MM, Antoniazzi JH. The effect of the renewal of calcium hydroxide paste on the apexification and periapical healing of teeth with incomplete root formation. Int Endod J 2005;38:436–42.
18. Moorrees CF, Fanning EA, Hunt EE Jr. Age varition of formation stage for ten permanent teeth. J Dent Res 1963;42:1490–502.
19. Marais JT. The use of calcium hydroxide as a dressing in root canal treatment. J Dent Assoc S Afr 1996;51:593–9.
20. Yates JA. Barrier formation time in non-vital teeth with open apices. Int Endod J 1988;21:313–9.
21. Erdem AP, Sepet E. Mineral trioxide aggregate for obturation of maxillary central incisors with necrotic pulp and open apices. Dent Traumatol 2008;24:e38–41.
22. Shah N, Logani A, Bhaskar U, Aggarwal V. Efficacy of revascularization to induce apexification/apexogensis in infected, nonvital, immature teeth: a pilot clinical study. J Endod 2008;34:919–25. Discussion 1157.
23. Trope M. Treatment of the immature tooth with a non-vital pulp and apical periodontitis. Dent Clin N Am 2010;54:313–24.
24. American Academy of Pediatric Dentistry. Guideline on pulp therapy for primary and immature permanent teeth. Reference Manual. Pediatr Dent 2011;33:194–201.
25. Walia T, Chawla HS, Gauba K. Management of wide open apices in non-vital permanent teeth with $Ca(OH)_2$ paste. J Clin Pediatr Dent 2000;25:51–6.
26. Dominguez Reyes A, Munoz Munoz L, Aznar Martin T. Study of calcium hydroxide apexification in 26 young permanent incisors. Dent Traumatol 2005;21:141–5.
27. Morse DR, O'Larnic J, Yesilsoy C. Apexification: review of the literature. Quintessence Int 1990;21:589–98.
28. Sheehy EC, Roberts GJ. Use of calcium hydroxide for apical barrier formation and healing in non-vital immature permanent teeth: a review. Br Dent J 1997;183:241–6.
29. Hatibovic-Kofman S, Raimundo L, Zheng L, Chong L, Friedman M, Andreasen JO. Fracture resistance and histological findings of immature teeth treated with mineral trioxide aggregate. Dent Traumatol 2008;24:272–6.
30. Webber RT. Apexogenesis versus apexification. Dent Clin N Am 1984;28:669–97.
31. Mendoza AM, Reina ES, García-Godoy F. Evolution of apical formation on immature necrotic permanent teeth. Am J Dent 2010;23:269–74.
32. Noori AJ, Al-Obaidi WA. Traumatic dental injuries among primary school children in Sulaimani city, Iraq. Dent Traumatol 2009;25:442–6.