Immature Teeth With Periradicular Periodontitis or Abscess Undergoing Apexogenesis: A Paradigm Shift

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Abstract

Four clinical cases of immature teeth that developed periradicular periodontitis or abscess underwent a conservative treatment approach, i.e. without canal instrumentation. Instead, only copious 2.5% NaOCl irrigation was performed. All cases presented herein developed mature apices after 7 months to 5 years after the initial treatment without complications, although narrowing canal space was observed. Our clinical observations support a shifting paradigm toward a conservative approach by providing a favorable environment for tissue regeneration. The mechanism of this continued development and formation of the root end is discussed. *(J Endod 2006;32: 1205–1213)*

Key Words

Apexification, apexogenesis, calcium hydroxyde, immature teeth, NaOCl, open apex

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Given the right condition, many tissues are programmed for self-regeneration to restore the lost part. Pulp tissue in immature teeth with open apices has a rich blood supply and contains a structure at developing stage that is more potent to regenerate in response to damage. The general consensus for clinical treatment of immature teeth with vital pulps is to preserve remaining normal vital tissue to allow continued physiological development and complete formation of the root end–apexogenesis. Whereas for those teeth with nonvital pulps, it is to clean and fill the canals with $Ca(OH)_2$, the most commonly used material, to induce the formation of a calcified barrier at the open apex–apexification (1, 2). Teeth after successful apexogenesis develop a normal thickness of dentin and root length. In contrast, those receiving apexification normally gain only an apical hard tissue bridge, not dentin, because of the loss of vital pulp tissues, odontoblasts, and Hertwig epithelial root sheath needed for the complete root development. However, this paradigm has been challenged by recent reports showing convincingly that immature teeth clinically diagnosed with nonvital pulp and periradicular periodontitis or abscess can undergo apexogenesis (3, 4). These reports stimulated a new perspective as to how we treat these cases. It has been advocated that immature teeth should be treated as conservatively as practical to allow any possible apexogenesis to occur (5). Teeth inherit thin and weak roots after successful apexification that are susceptible to fracture. Shifting apexification to apexogenesis even for nonvital pulps with periradicular periodontitis or abscess is a clinically beneficial approach for patients if we gathered more clinical experience to help predict the treatment outcome.

Most interesting aspect of the reported cases (3, 4) is that those teeth showing continual maturation of root and apex had developed extensive periradicular lesions with sinus tract formation before the treatment; a condition normally resulting from total necrosis and infection of the pulp. Herein we further demonstrate four cases of immature teeth with periradicular periodontitis or abscess treated with conservative approaches that led to continued development of normal apical morphology. The possible mechanism underlying this clinical observation is discussed with published information regarding pulp healing after traumatic or experimentally induced injuries.

Case Report

Case 1

A 10-year-old Asian girl, referred by a pediatric dentist, after presenting an asymptomatic tooth #20 with a sinus tract on buccal gingiva (Fig. 1*A*). The general health history was noncontributory and periodontal probing was within normal limit. There was a fractured central cusp (dens evaginatus) that was likely the cause of the pathosis. The radiographic finding revealed periradicular radiolucency approximately 5×4 mm with a gutta-percha point tracing the sinus tract to tooth #20 (Fig. 1*B*). There was no percussion discomfort. Pulp vitality test was not performed as it gives unreliable results from immature teeth. Upon accessing the tooth without anesthesia, viable tissue with hemorrhage was observed. The clinical diagnosis was partial pulp necrosis with chronic periradicular abscess. The pulp chamber was irrigated with approximately 20 ml of 2.5% NaOCl (Clorox, Oakland, CA), carefully dried with paper points and filled with $Ca(OH)_2$ paste (powder mixed with saline, Merck, Frankfurt, Germany). Tooth was sealed with Caviton (GC, Aichi, Japan) and IRM (Caulk Dentsply, Milford, DE). Two weeks later, patient claimed that tooth was sensitive for 1 day after the first visit and remained asymptomatic since. There was no hemorrhage upon re-entry and the same procedure was performed as in the previous appointment. Three months after the initial

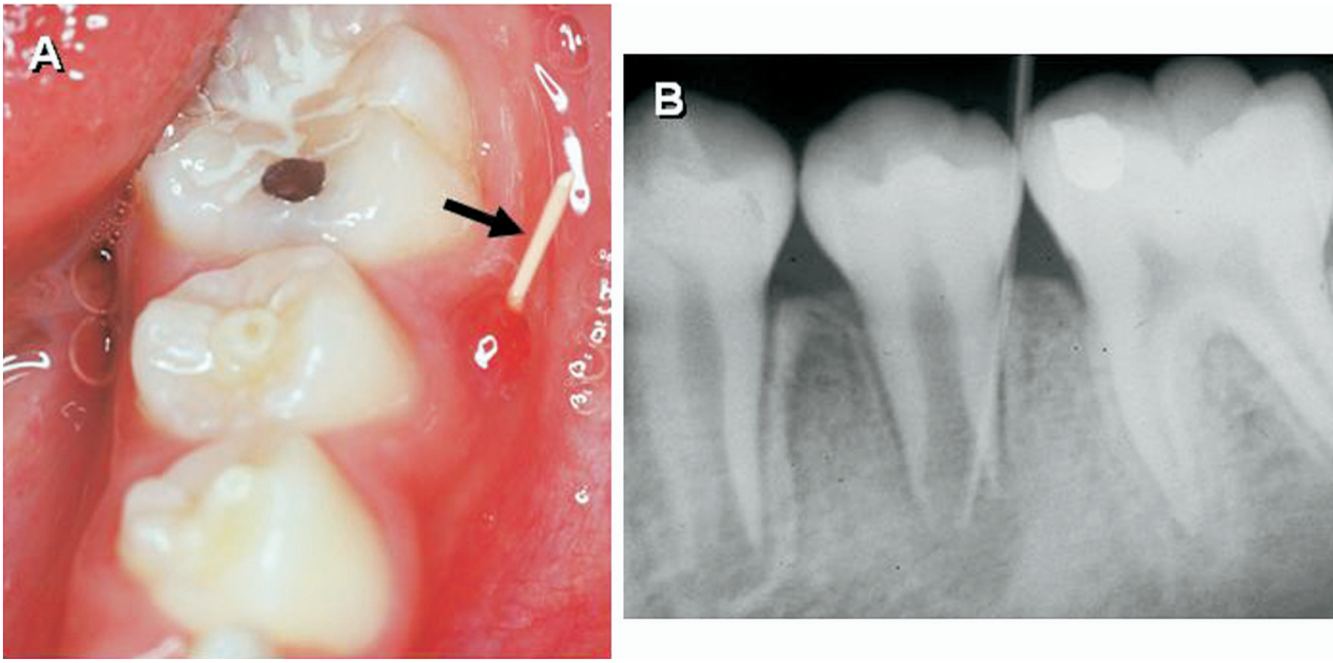

Figure 1. Clinical photograph and periapical radiograph of case 1. *A*, Photograph showing a sinus tract at the alveolar mucosa between teeth #20 and #19 and insertion of a gutta-percha point into the sinus tract (arrow). The fractured central cusp is evident. *B*, Radiograph showing a radiolucent lesion at the periapical area of tooth #20 with a wide open apex.

treatment, patient presented asymptomatic. Hard barrier was detected after removal of $Ca(OH)_2$ paste. A new mixture of $Ca(OH)_2$ was placed and the access closed with Caviton/IRM. Seven months after the first visit, the tooth remained asymptomatic but still showed an open apex (Fig. 2*A*). The periapical radiograph made 11 months after the initial treatment (Fig. 2*B*) demonstrated a more narrowed root canal compared to that shown in Fig. 2*A*. Therefore, the IRM and part of the Caviton were removed and replaced with amalgam. At 20 months after the initial treatment, the radiographic examination showed healing of the periapical bone and more reduction of the root canal space (Fig. 2*C*). At 35 months after the initial treatment, the radiographic examination revealed a marked reduction of the root canal space and maturation of the root apex (Fig. 2*D*).

Case 2

A 10-year-old Asian boy complained of toothache in the mandibular posterior region 4 days before the appointment and the condition gradually became worse with the development of swelling on the right cheek 2 days before. According to the patient's statement, ice-packing was helpful in pain relief. The patient's past medical history was unremarkable. Intraoral examination revealed a swelling at the buccal gingiva and alveolar mucosa between teeth #28 and #29 and central cusp fracture of tooth #29 (Fig. 3*A*). Tooth #29 showed a grade I mobility, a negative response to cold test and a sensitivity to palpation and percussion. Radiographic findings showed immature root with open apex. Extensive radiolucency was observed in the periradicular region extending coronally on the mesial aspect of the root (Fig. 3*B*). The clinical diagnosis was pulp necrosis with acute periradicular abscess. Without anesthesia, the tooth was accessed. Patient felt mild pain upon reaching the pulp chamber and copious hemorrhage was noted (Fig. 3*C*). As performed in case 1, pulp chamber was irrigated with NaOCl, dried, $Ca(OH)_2$ paste (Merck) placed into the pulp chamber and the canal, and the access sealed with glass-ionomer cement. At the 4th week after the initial visit and treatment, the tooth was asymptomatic and the soft tissue had healed. Hemorrhage from the canal was observed upon accessing. The canal was again irrigated, $Ca(OH)_2$ placed, and the access sealed as before. Periapical radiograph made after the second treatment showed partially healed periradicular bone with an open apex of tooth #29 (Fig. 4*A*). At 8 weeks the patient returned asymptomatic. After accessing, calcified barrier was detected by probing with a periodontal probe. $Ca(OH)_2$ was refreshed and the tooth sealed. At 7 months the patient returned with no symptoms. Radiographic examination revealed nearly complete maturation of the root apex and healing of the periradicular bone except minor radiolucency around the apical bone, likely the result of healed bone with less trabecular density. There was a significant increase of hard tissue thickness of the root with reduced canal space. The coronal third of the canal appeared to be filled with calcified tissue (Fig. 4*B*). The tooth was then sealed with Caviton/Ketac-silver. Patient did not return for subsequent follow-ups.

Case 3

A 10-year-old Asian girl suffered from an acute toothache and was referred by a prosthodontist. The periapical radiograph brought by the patient showed a mesially tilted tooth #20 with an open apex (Fig. 5*A*). An emergency treatment, formocresol pulpotomy, was performed 9 days before at the referring clinic. Initial periapical radiograph made after the emergency treatment revealed a radiolucent lesion ($\sim 3 \times 4$ mm) at the periradicular area of tooth #20 with an open apex (Fig. 5*B*). Clinical diagnosis was chronic periradicular periodontitis of tooth #20 with possible partial necrosis of the pulp. Central cusp fracture was suspected as the cause of pulp infection. After accessing, no hemorrhage was observed. The canal was irrigated with 2.5% NaOCl, medicated with $Ca(OH)_2$ (Merck), and sealed with IRM. Periapical radiograph taken after the initial treatment showed an open-apex tooth #20 with a radiolucent lesion at the periradicular area of tooth #20 (Fig. 6*A*). One month later, the periapical radiograph made before treatment showed a calcified barrier in the middle portion of the root canal (Fig. 6*B*). A firm mid-root stop was noted by probing. Fresh $Ca(OH)_2$ was

Figure 2. Follow-up periapical radiographs of case 1. *A*, Radiograph made 7 months after the initial treatment showing an asymptomatic tooth #20 with an open apex. *B*, Eleven months after the initial treatment demonstrating a slightly narrowed root canal. *C*, Twenty months after the initial treatment showing healing of the periapical bone and more reduction of the root canal space. *D*, Thirty-five months after the initial treatment revealing a market reduction of the root canal space and maturation of the root apex.

placed and the tooth was temporized with IRM. Another month later and subsequently every 2 to 3 months, $Ca(OH)_2$ was refreshed and access sealed with Caviton/glass-ionomer cement. The periapical radiograph made 4 months after the initial treatment showed healing of the periapical bone (Fig. 6*C*) and that taken 10 months after the initial treatment demonstrated a gradual hard-tissue deposition on the canal wall and reduction of root canal space at the apical half of the root (Fig. 6*D*). No endodontic procedure was made at this point (six treatments) onward. At the 11th month after the initial treatment, patient complained of sensitivity of the tooth while undertaking orthodontic treatment. Periapical radiograph made at that time showed a matured apex of tooth #20 with a well-formed lamina dura (Fig. 7*A*). Further hard-tissue deposition of canal wall and reduction of the root canal space at the apical half of the root was observed 13.5 months after the initial treatment (Fig. 7*B*). At 18.5 months after the initial treatment, there was radiographic evidence of further deposition of calcified tissue at the apical half of the root canal (Fig. 7*C*), thus the coronal half of the root canal and the coronal pulp chamber were filled with amalgam (Fig. 7*D*). At the 34th month after the initial treatment, the tooth was asymptomatic with complete root formation and an almost completely calcified apical half of the root canal (Fig. 7*E*). Four and a half years after the initial treatment, the tooth remained asymptomatic but the apical half of the root canal was completely calcified (Fig. 7*F*).

Case 4

A 9-year-old Asian boy had a toothache in #29 for 5 days and went for an emergency treatment, after which the tooth was left open. Patient's mother was told that an endodontic treatment was needed. At the first endodontic appointment, periapical radiograph was made and revealed an open apex of tooth #29 without noticeable periradicular radiolucency (Fig. 8*A*). The tooth, without spontaneous pain, was sensitive to percussion and the clinical diagnosis was pulp necrosis with chronic periradicular pathosis. The patient was apprehensive, therefore, the test results may not reflect the actual tissue response. Without anesthesia, the rubber dam was placed and the canal irrigated with 40 ml 2.5% NaOCl. No instrumentation was performed, the canal was filled with $Ca(OH)_2$, and the access sealed with IRM. Two weeks later, the same procedures were performed. A visit 5-weeks after the first endodontic treatment, the same procedures were performed, and a firm stop at mid-root was detected. At 5 months, the same procedures were performed, and the periapical radiograph showed progressing apical maturation of tooth #29 with an extension of the root length (Fig. 8*B*). At 11, 17, 24 (Fig. 8*C*), and 32 months after the initial treatment, same procedures were performed at each visit. Three years after the first treatment, root formation was complete. The canal space was filled with amalgam to the point of the calcified bridge (Fig. 8*D*). At 5 years there

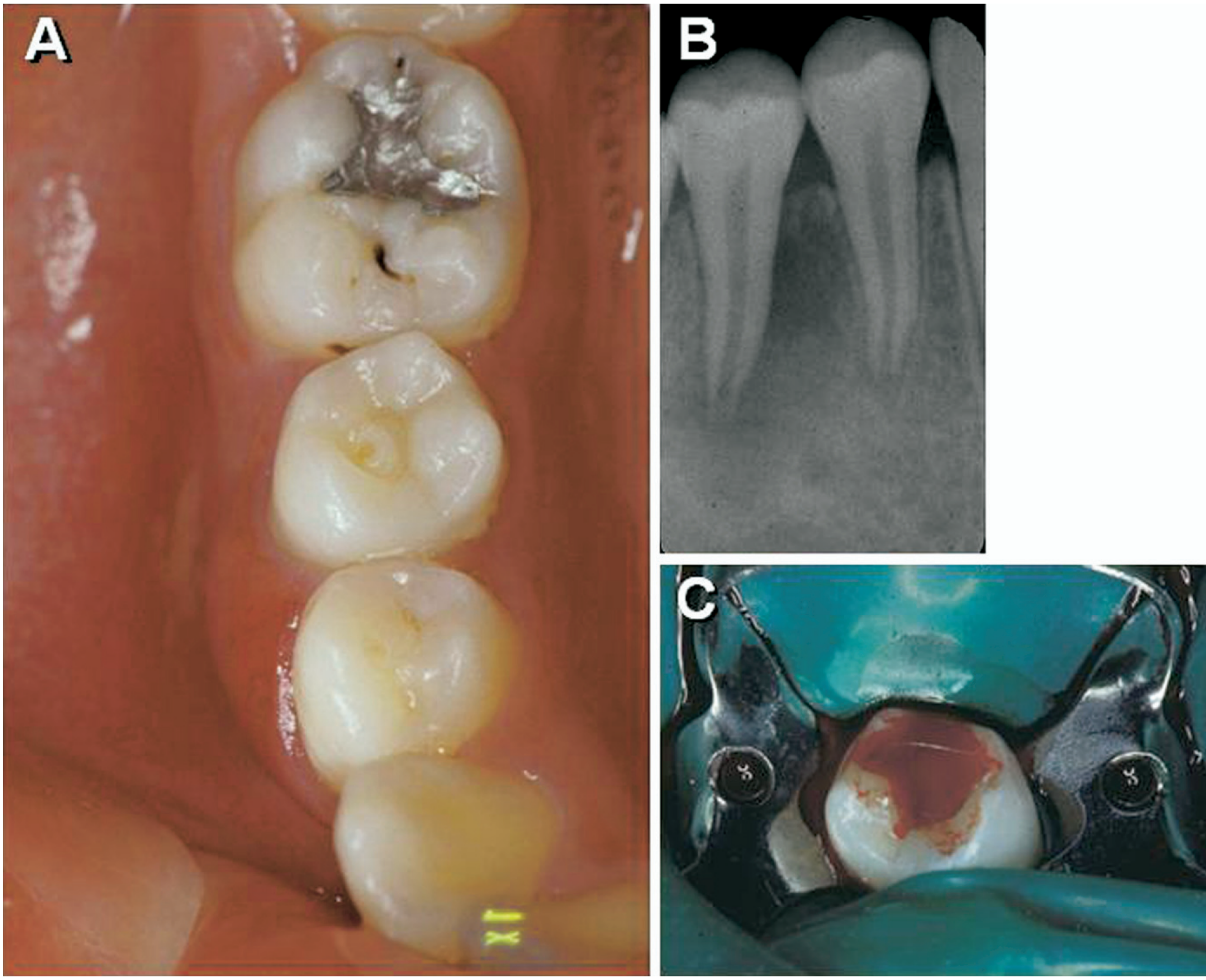

Figure 3. Clinical photographs and periapical radiograph of case 2. *A*, Photograph revealing a swelling at the buccal gingiva and alveolar mucosa between teeth #28 and #29 and central cusp fracture of tooth #29. *B*, Radiograph showing an immature root of tooth #29 with an open apex and an extensive radiolucency at the periapical and mesial regions of the root of tooth #29. *C*, Photograph demonstrating copious hemorrhage upon accessing the pulp chamber.

were no symptoms, but evidence of severe narrowing of canal space (Fig. 8*E*). Intraoral examination revealed a sound tooth #29 without discoloration (Fig. 8*F*).

Discussion

Here we reported four clinical cases, all of which have been conventionally considered as indications for apexification treatment. Instead of utilizing endodontic files to clean and shape the infected and partially or totally necrotic pulp, these cases were treated with the most conservative approach, namely no instrumentation of the canal besides irrigation with 2.5% NaOCl. The treatment periods of these cases span between 1988 and 2000.

Iwaya et al. (3) reported an unconventional treatment of an immature permanent tooth (mandibular 2nd premolar) with periradicular abscess and a sinus tract. The tooth was instrumented but not to its full length because the patient felt the insertion of a smooth broach. In the first visit the tooth was left open and at the following visits the canal was only irrigated with NaOCl and hydrogen peroxide without any instrumentation. Antimicrobial agents (metronidazol and ciprofloxacin) were placed in the canal and at the next visit vital tissue was visualized 5 mm apical to the canal orifice. Thirty-five months later the root apex formation was complete along with thickened root structure as the result of significantly reduced canal size compared to the adjacent normal first premolar. The tooth responded to electrical pulp test. Later, Banchs and Trope (4) reported a case of similar condition but with more extensive periradicular bone loss. They applied a similar treatment procedure except some hemorrhage was induced to allow blood clot formation in the canal. A remarkable result comparable to the report by Iwaya et al. was also achieved. They also noted the positive response to cold test at the recall. In both reports, the authors consider that there was a regain of vascularization in the canal tissue, possibly pulp tissue. Although Iwaya et al. applied $Ca(OH)_2$ at the 6th visit, Banchs and Trope emphasized not to use $Ca(OH)_2$ to preserve any remaining viable pulp tissue and Hertwig epithelial root sheath. Our clinical observations support this notion as demonstrated by cases 3 (Figs. 5–7) and 4 (Fig. 8) in which placement of $Ca(OH)_2$ far down into canals may have at least prevented the deposition of hard-tissue formation in the coronal half of the canals.

In the traditional apexification treatment, apical barrier is established by the formation of cementum-like tissue of various thicknesses.

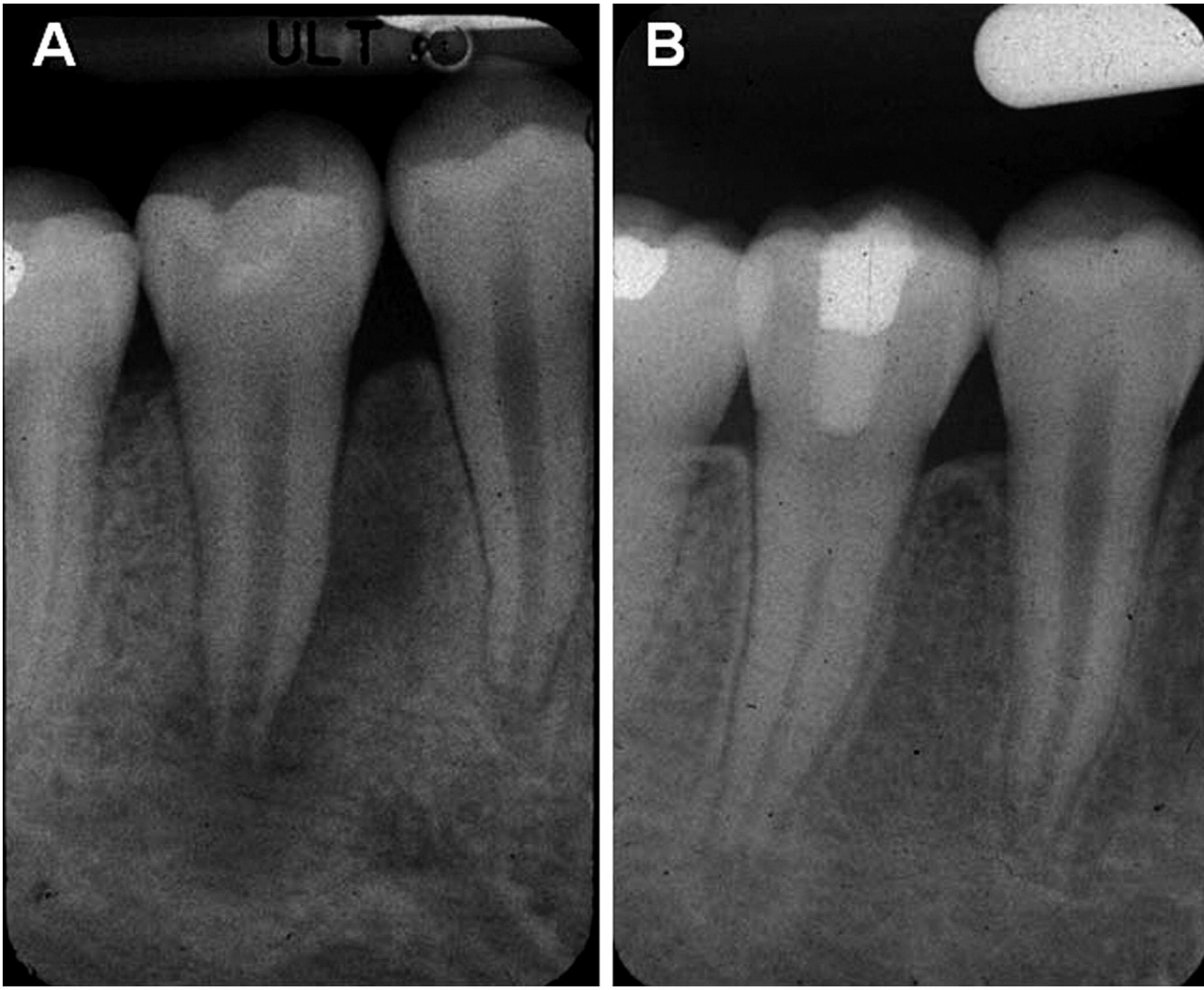

Figure 4. Periapical radiographs of case 2. *A*, After the second treatment showing partially healed periradicular bone and tooth #29 with an open apex. *B*, Seven months after the initial treatment showing complete maturation of the root apex, healing of the periradicular bone, a significant increase of the calcified tissue in the root, a significant decrease of root canal space, and the calcified coronal third of the root canal.

A recent review by Rafter (1) summarizes the histological characteristics reported in the literature. The hard tissue barrier has been described as a cap, bridge, or ingrown wedge that may be composed of cementum, dentin, bone, or so-called osteodentin that can deposit on the inner walls of the canal (6–11). Cementum formation can proceed from the periphery of the apex towards the center in decreasing concentric circles. In contrast to apexogenesis, apexification treatment does not generally lead to an additional formation of root dentin or an extension of root length. The undesirable outcome is a weak root susceptible to fracture. Filling the canal from mid-root to coronal third with resin bonding to strengthen the root has been advocated after the completion of apexification (12–14). Conservative approach to reserve any remaining vital pulp tissue may provide hope for a better outcome, although the control of root canal infection may be a difficult issue. The duration of the infection, the involved microbial species, the host immunity, and the size of the open apex all may theoretically play a role in the outcome of this conservative treatment approach.

In the present report, with an utmost conservative approach, all cases showed noticeable apical maturation with increased root length but a significant narrowing of canal space. The question is, whether the thickened root was formed by pulp tissue from the remaining vital pulp tissue at the apical region that was resistant to infection, capable of regenerating the pulp tissue in the canal space and making new dentin; or the thickened root was formed by periodontal ligament (PDL) tissue, which grew into the root canal from the apical foramen and deposited the cementum onto the inner surface of the root dentin.

Although lacking histological data from human specimens, one may speculate based on animal studies. Reports by Nevins et al. (15, 16) using rhesus monkeys demonstrated that after total pulp tissue removal in immature teeth, either treated with $Ca(OH)_2$ or collagen gel, there was cementum tissue formation at the apex and in the canal. PDL-like tissue can also be found in canal treated with collagen gel. PDL and cementum tissues formed in the canal space can be verified histologically by the presence of Sharpey's fibers (Nevins, unpublished data). Studies by Ellis et al. (17) and Hitchcock et al., (18) revealed that when blood supply is cut off in the middle of the root in monkeys, the canal space is eventually replaced by cementum and PDL along the inner canal dentinal wall, accompanied by some bone tissues. These tissues may

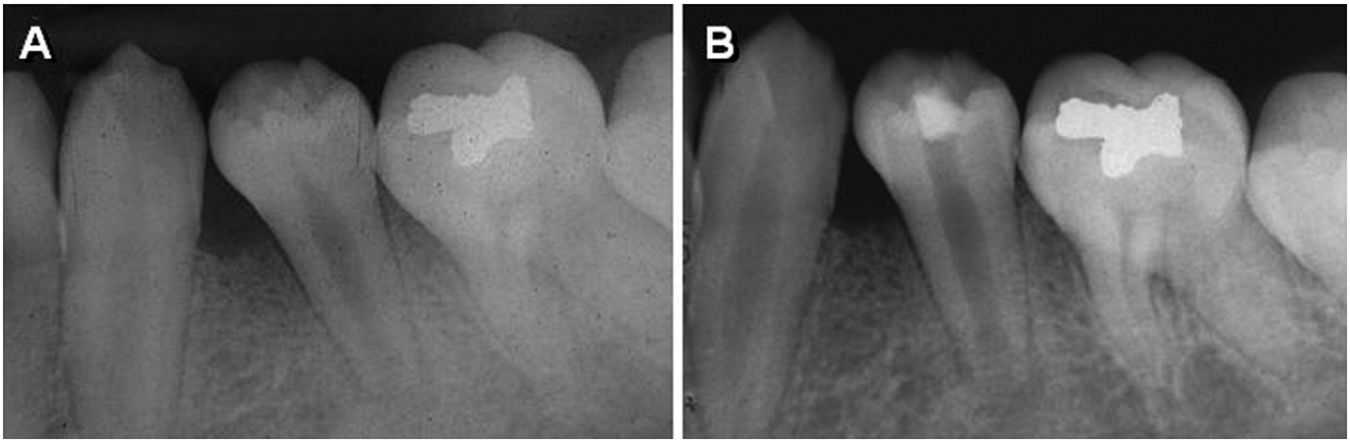

Figure 5. Periapical radiographs of case 3. *A*, Radiograph brought by the patient from the referring clinic showing a mesially tilted tooth #20 with an open apex. *B*, Radiograph made after the formocresol pulpotomy revealing a radiolucent lesion at the periapical area of tooth #20 with an open apex.

extend into pulp chamber. Other animal studies focusing on the changes in pulp tissue after replantation showed that various hard tissues including dentin, cementum, and bone may form in pulp space depending on the level of pulp recovery (11, 15, 19–22). Therefore, if one assumes the total loss of pulp tissue but remaining in a sterile condition, the outcome is the ingrowth of periodontal tissues. Evidence of stem cells in PDL has recently been shown and the formation of new cementum is characterized by the presence of Sharpey's fibers (23). This possibility may explain the increased thickness of the canal wall and the severe shrinkage of canal space. This deposition of cementum or bone in the canal may gradually and eventually obliterate the space as demonstrated in all four cases presented in the present report and those

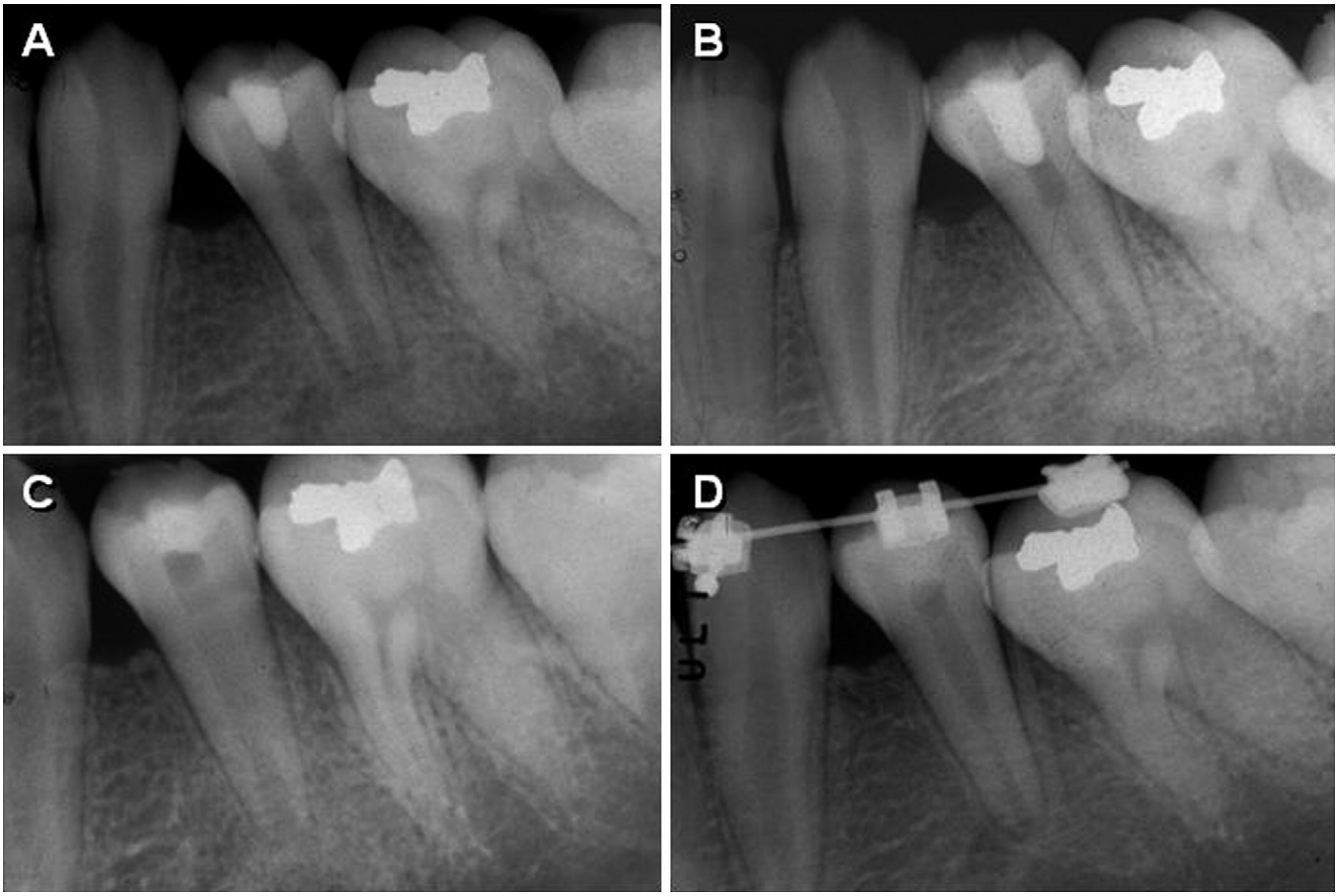

Figure 6. Periapical radiographs of case 3. *A*, Radiograph taken right after the initial endodontic treatment of tooth #20. *B*, Radiograph made before the second treatment one month after initial treatment demonstrating a calcified barrier in the middle portion of the root canal. *C*, Radiograph made before the fourth treatment 4 months after initial treatment showing a healing of the periapical bone. *D*, Radiograph made before the sixth treatment 10 months after initial treatment demonstrating a gradual deposition of calcified structure of the root and reduction of the root canal space at the apical half of the root.

Figure 7. Follow-up periapical radiographs of case 3 after the completion of six treatments. *A*, Eleven months after the initial treatment showing a mature apex of tooth #20 with a well-formed lamina dura. *B*, Thirteen and a half months after the initial treatment demonstrating further deposition of hard tissue in the root and reduction of canal space at the apical half of the root. *C*, Before amalgam filling 18.5 months after the initial treatment exhibiting evidence of deposition of more calcified material at the apical half of the root canal. *D*, After amalgam filling 18.5 months after the initial treatment. *E*, Thirty-four months after the initial treatment showing complete root formation and an almost completely calcified apical half of the root canal. *F*, Fifty-four months after the initial treatment revealing a completely calcified apical half of the root canal.

reported by Iwaya et al. (3) and Banchs and Trope (4). Tsukamoto-Tanaka et al. (24) observed hard tissue formation in rat dental pulp during healing process after replantation. Using histochemical and immunocytochemical approach, they identified specific cells and their activities in the pulp and periodontal tissues. Tertiary dentin formation was observed by newly differentiated odontoblasts, whereas osteoclasts associated with bone matrix in pulp were observed in cases where tertiary dentin could not be recognized.

The fate of human pulp tissue after dental traumas has been observed in clinical radiographs. Andreasen et al. (25, 26) and Kling et al. (27) showed excellent radiographic images of the ingrowth of bone and PDL (next to the inner dentinal wall) into the canal space with arrested root formation after replantation of avulsed maxillary incisors. Some cases demonstrated partial formation of root accompanied with ingrowth of bone and PDL into the canal space. In their reports, when teeth continued to develop roots to their completion, normally associ-

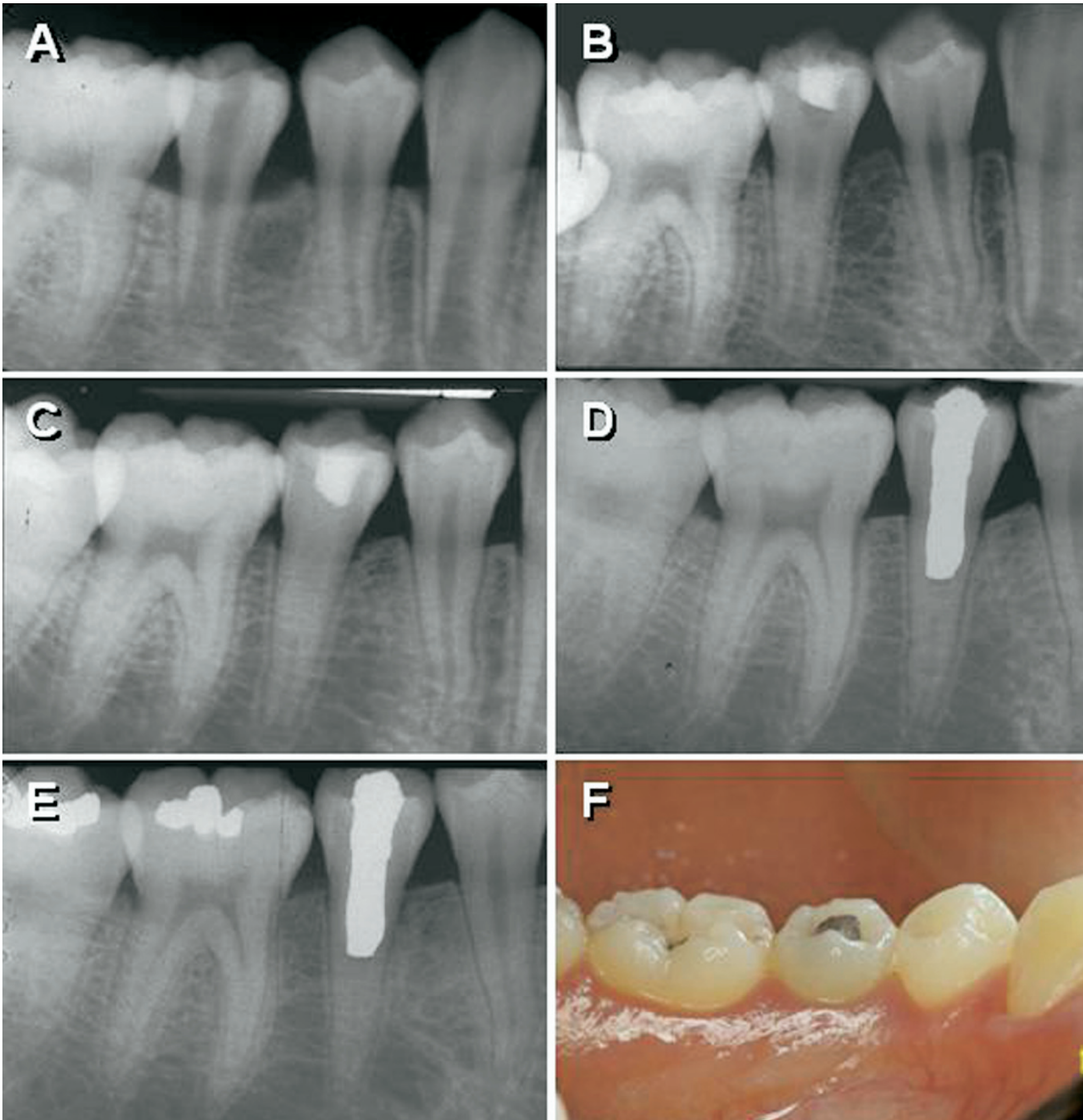

Figure 8. Periapical radiographs and clinical photograph of case 4. *A*, Radiograph made before the first treatment revealing an open-apex of tooth #29 without noticeable periradicular radiolucency. *B*, Five months after the initial treatment showing more apical maturation of tooth #29 with an extension of the root length. *C*, Twenty-four months after the initial treatment exhibiting a nearly complete root end formation. *D*, Three years after the first treatment demonstrating a complete root formation. The root canal was filled with amalgam to the point of the calcified bridge. *E*, Five years after the first treatment revealing evidence of severe narrowing of the canal space. *F*, Clinical photograph taken 5 years after the first treatment showing a sound tooth #29 without discoloration.

ated with severe narrowing of canal space, the authors considered pulp survival after the replantation.

Saad (28) reported a clinical case of a maxillary incisor with nonvital pulp and with periradicular pathosis that underwent apexogenesis after canal cleaning and shaping with NaOCl and instruments and filled with $Ca(OH)_2$, although the canal appeared obliterated at 2.5 years after the treatment. Lieberman and Trowbridge (10) observed a clinical case of apical bridge formation and was able to perform histological examinations after extraction. Although the authors stated the presence of atubular dentin and cementum in the calcific barrier, their data showed that the apical barrier is basically made of cementum that not only forms a bridge but also extends coronally along the dentinal wall. Sharpey's fibers, not mentioned by the authors, appear to form between the canal cementum and the soft tissue. Taken together, if pulp tissue is totally lost, the canal space may be occupied by cementum, PDL, and bone. In this situation, it is difficult to identify clinically via radiographs because the canal space may well be PDL tissue and the thickened root structure be cementum.

On the other hand, if there was survived pulp tissue despite the development of periradicular abscess because of the rich blood supply through the wide open apex, apexogenesis could occur. Even in mature teeth, there may be remaining vital pulp tissues when a periradicular lesion is developed (29, 30). Dental pulp stem cells have been identified to exist in permanent teeth (31–33). In the case of a developing tooth, the dental papilla at the apex may contain more stem cells than a mature tooth and therefore possesses a greater potential to rebuild the lost pulp tissue and continue the root maturation. If this is the case, using revascularization to describe this phenomenon does not encompass the scope of regeneration of pulp tissue in which genuine pulp containing functional odontoblasts is capable of laying down new dentin to complete the root development. Pulp stem cells seeded onto existing dentin may differentiate into odontoblasts and may deposit new dentin (34, 35) Or, the survived pulp tissue fragments may form dentin in the center of the pulp and mixed with ingrown cementum (11, 19). In conclusion, whereas scientifically it is interesting to know the histological feature of the tissues involved in the formation of the root after treatment, clinically our case reports shown here along with those by others strongly suggest a paradigm shift in the clinical management of this type of cases, i.e. provide a favorable condition to allow natural tissue regeneration rather than replacement with artificial materials. Furthermore, a conservative treatment for mature teeth under this circumstance may even be a possibility in the future with advanced tissue engineering technologies (35–37). Both basic and clinical research toward reaching this goal is needed.

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