Treatment of immature teeth with non-vital pulps and apical periodontitis

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Introduction

Teeth with immature root development, necrotic pulps, and apical periodontitis present multiple challenges for successful treatment (Fig. 1):

1. The infected root canal space cannot be cleaned and disinfected with the standard root canal protocol using an aggressive procedure with endodontic files.
2. After the disinfecting phase of treatment has been completed, filling the root canal is difficult because the open apex provides no barrier for containing the root filling material without impinging on periodontal tissues.
3. Even after successfully completing the endodontic procedure, the roots of these teeth are still thin and have a significant risk of subsequent fracture.

These problems can be managed using a disinfection protocol that minimizes root canal instrumentation, by stimulating the formation of a hard tissue barrier or providing an artificial apical barrier to allow for optimal filling of the canal, and by reinforcing the weakened root against fracture both during and after an apical barrier has been provided.

Traditional technique

Disinfection of the canal

Because in the vast majority of cases non-vital pulps are infected (1, 2), the first phase of treatment is to disinfect the root canal system to ensure periapical healing (2, 3). The canal length is estimated with a parallel pre-operative radiograph and, after access to the canals is made, a file is placed to this length. When the length has been confirmed radiographically, depending on the thickness of the remaining dentinal walls, either very light filing or no filing is performed with copious irrigation using 0.5% sodium hypochlorite (NaOCl) to remove necrotic pulp tissue (4, 5). A low concentration of NaOCl is used because of the increased danger of pushing sodium hypochlorite through the apex of immature teeth. The lower concentration of NaOCl is compensated for by the volume of irrigant used. An irrigation needle that can passively reach close to the apical length is useful for disinfecting the canals of these immature teeth. Newer irrigation protocols using devices such as EndoVac (6) or Ultrasound (7) may be useful in the canals of immature teeth. When the irrigant leaving the canal appears ‘clean’ of debris, intra-canal medication can then be placed to further disinfect the canal.

The canal is dried with paper points and a creamy mix of calcium hydroxide is spun into the canal with a lentulospiral instrument. The additional (to instrumentation and irrigation) disinfecting action of calcium hydroxide takes at least 1 week (8); the continuation of treatment can therefore take place any time after 1 week. Continuation of treatment should not be delayed more than 1 month since calcium hydroxide can be washed out by tissue fluids through the open apex, leaving the canal susceptible to re-infection.

Recently a new disinfection medicament has been used primarily when revascularization is attempted.
This medicament has been extensively studied by Hoshino et al. (9, 10). It is comprised of metronidazole, ciprofloxazone and minocycline in a saline or glycerin vehicle (Fig. 2). A recent study by Windley et al. (11) showed the effectiveness of this Tri Mix antibiotic mixture when used in immature infected dog teeth that had been irrigated only with sodium hypochlorite followed by placement of the medicament for 1 month.

![Fig. 1. The immature root with a necrotic pulp and apical periodontitis presents multiple challenges to successful treatment.](image)

**3Mix-MP**
- Antibiotics (3Mix)
  - Ciprofloxacin 200 mg
  - Metronidazole 500 mg
  - Minocycline 100 mg
- Carrier (MP)
  - Macrogol ointment
  - Propylene glycol

**Protocol for preparation**
- Antibiotics (3Mix) – be sure to not cross-contaminate
  - Remove sugar coating from tablets with surgical blade, crush individually in separate mortars
  - Open capsules, crush individually in separate mortars
  - Grind each antibiotic to a fine powder
  - Combine equal amounts of antibiotics (1:1:1) on mixing pad
- Carrier (MP)
  - Equal amounts of macrogol ointment and propylene glycol (1:1)
  - Using clean spatula, mix together on pad
  - Result should be opaque
- Separate out small portions of 3Mix and incorporate into MP using the following:
  - 1:5 (MP:3Mix) → creamy consistency
  - 1:7 (standard mix) → smears easily but does not crumble
  - If result is flaky or crumbly, then too much 3Mix has been incorporated

**Storage**
- Antibiotics must be kept separately in moisture-tight porcelain containers
- Macrogol ointment and propylene glycol must be stored separately
  - Discard if mixture is transparent (evidence of moisture contamination)

![Fig. 2. Composition and mixing instructions for the tri-antibiotic paste.](image)
Hard tissue apical barrier

Traditional method

The formation of a hard tissue barrier at the apex requires a similar environment to that which is required for hard tissue formation in vital pulp therapy, i.e. a mild inflammatory stimulus to initiate healing and a bacteria-free environment to ensure that the inflammation is not progressive.

As with vital pulp therapy, calcium hydroxide is used for this procedure (12–14). Pure calcium hydroxide powder is mixed with sterile saline (or anesthetic solution) to a thick (‘stiff’) consistency (Fig. 3). Ready-mixed commercial calcium hydroxide preparations are also acceptable. The calcium hydroxide is packed against the apical soft tissue with a plugger or thick paper point to initiate hard tissue formation. This step is followed by backfilling with calcium hydroxide to completely fill the canal, thus ensuring a bacteria-free canal with little chance of re-infection during the 6–18 months required for hard tissue formation at the apex. The calcium hydroxide is meticulously removed from the access cavity to the level of the root orifices and a well-sealing temporary filling is placed in the access cavity. A radiograph is taken and the canal should appear radiopaque, indicating that the entire canal has been filled with the calcium hydroxide (Fig. 4). Because calcium hydroxide washout is evaluated by its relative radiopacity in the canal, it is prudent to use a calcium hydroxide mixture without the addition of a radiopaque material such as barium sulfate. These additives do not wash out as readily as calcium hydroxide so that if they are present in the canal, evaluation of washout is not possible.

At 3-month intervals, a radiograph is exposed to evaluate whether a hard tissue barrier has formed and if the calcium hydroxide has washed out of the canal. This is determined to have occurred if the canal can again be seen radiographically. If calcium hydroxide washout is
When completion of a hard tissue barrier is indicated radiographically, the calcium hydroxide is washed out of the canal with sodium hypochlorite and a radiograph taken to evaluate the radiopacity of the apical stop. A file of a size that can easily reach the apex can be used to gently probe for a stop at the apex. When a hard tissue barrier appears radiographically and can be probed with an instrument, the canal is ready for filling. The hard tissue barrier that forms has been described as ‘Swiss cheese-like’ (Fig. 5). This is caused by the many soft tissue inclusions inside the hard tissue formed in response to the treatment. The result of this is that soft filling materials (e.g. sealers and softened gutta-percha) often pass through the apex, creating ‘apical puffs.’ Additionally, the hard tissue barrier will form at the site of healing of the periodontal granulation tissue. This site does not always conform to the radiographic apex of the tooth. Therefore, when that hard tissue is felt with a point or file, it may be short of the radiographic apex of the tooth. It is important not to force the file to the radiographic apex, thus destroying the apical barrier.

The traditional calcium hydroxide apexification technique has been extensively studied and has proven to have a very high success rate (16, 17). However, the technique has some disadvantages. The primary disadvantage is that it typically takes between 6 and 18 months for the hard tissue barrier to form. The patient needs to report every 3 months to evaluate whether the calcium hydroxide has washed out and/or the barrier is complete enough to provide a stop for a filling material. This requires patient compliance for up to six visits before the procedure is completed. In addition, it has been shown that the use of calcium hydroxide weakens the resistance of the dentin to fracture (18). Thus it happens that before the hard tissue barrier has formed, the patient may sustain another injury and fracture the root (Fig. 6).

**MTA barrier**

Mineral trioxide aggregate (MTA) (ProRoot-MTA, Dentsply, Tulsa, OK) has been used to create an immediate hard tissue barrier after disinfection of the root canal (Fig. 7). Calcium sulfate (or similar material) is pushed through the apex to provide a resorbable extra-radicular barrier against which to pack the MTA. The MTA is mixed and placed into the apical 3–4 mm

![Fig. 5. Histological appearance of a ‘Swiss cheese-like’ apical hard tissue barrier. Note the soft tissue inclusions inside the hard tissue.](image)

![Fig. 6. Root that suffered a horizontal root fracture during the long-term calcium hydroxide treatment. Courtesy of Dr. Jose Luis Mejia.](image)
of the canal in a manner similar to the placement of calcium hydroxide. A wet cotton pellet can be placed against the MTA and left for at least 6 h and then the entire canal filled with a root filling material or the filling can be placed immediately because the tissue fluids of the open apex will probably provide enough moisture to ensure that the MTA will set sufficiently. The cervical canal is then reinforced with composite resin to below the marginal bone level as described above (Fig. 7).

A number of case reports have been published using this MTA apical barrier technique (19, 20) and it has steadily gained popularity with clinicians. Presently no prospective long-term outcome study is available comparing its success rate to that of the traditional calcium hydroxide technique.

Because the apical diameter is larger than the coronal diameter of many of these canals, a softened filling technique is indicated in these teeth. Care must be taken to avoid excessive lateral force during filling due to the thin walls of the root.

The apexification procedure has become a predictably successful procedure (16, 17). However, the thin dentinal walls still present a clinical problem. Should
secondary injuries occur, teeth with thin dentinal root walls are more susceptible to fractures rendering them non-restorable. It has been reported that approximately 30% of these teeth will fracture during or after endodontic treatment (16) (Figs 6 and 8). Consequently, some clinicians have questioned the advisability of the apexification procedure and have opted for more radical treatment procedures including extraction followed by extensive restorative procedures such as dental implants. Recent studies have shown that intracoronal bonded restorations can strengthen endodontically treated teeth and increase their resistance to fracture (21, 22). Thus after root filling, the material should be removed to below the marginal bone level and a bonded resin filling placed (Fig. 7).

Routine recall evaluation should be performed to determine the success in the prevention or treatment of apical periodontitis. Restorative procedures should be assessed to ensure that they in no way promote root fractures or allow bacterial recontamination through microleakage.

Periapical healing and the formation of a hard tissue barrier predictably occurs with long-term calcium hydroxide treatment (79–96%) (14). However, long-term survival is jeopardized by the fracture potential of the thin dentinal walls of these teeth. It is expected that the newer techniques of internally strengthening the teeth described above will increase their long-term survivability.

New approach to treatment of non-vital pulps

Pulp revascularization

Revascularization of a necrotic pulp has been considered possible only after avulsion of an immature permanent tooth. Skoglund et al. (23) showed that pulp revascularization was possible in dog teeth and it took approximately 45 days (Fig. 9). An advantage of pulp revascularization is the possibility of further root development, thus reinforcing the dentinal walls and strengthening the root against fracture.

After re-implantation of an avulsed immature tooth, a unique set of circumstances exists that allows revascularization to take place. The young tooth has an open apex and a short root that allows new tissue to grow into the pulp space relatively quickly. The pulp is necrotic but usually not degenerated and infected so that it can act as a scaffold into which the new tissue can grow. It has been shown experimentally that the apical part of a pulp may remain vital and after re-implantation proliferate coronally, replacing the necrotized portion of the pulp (23–26). In addition, the fact that in most cases the crown of the tooth is intact and caries-free ensures that bacterial penetration into the pulp space through cracks (27) and defects will be a slow process. Thus the race between the new tissue formation and bacterial penetration of the pulp space favors the new tissue.

Revascularization of the pulp space in a tooth with necrotic infected pulp tissue and apical periodontitis has been thought to be impossible until recently. Nygaard-Østby and Hjortdal (28) successfully regenerated pulps after vital pulp removal in immature teeth but were unsuccessful when the pulp space was infected. However, if it were possible to create a similar environment as described above for the avulsed tooth, revascularization should occur. Thus, if the canal is effectively disinfected, a scaffold into which new tissue can grow is provided and the coronal access effectively sealed, revascularization should occur similarly to that in an avulsed immature tooth.

A recent case report by Banchs and Trope (29) indicates that the results in cases reported by others...
(25, 26) may be possible to replicate in a similar way to the unique circumstances of an avulsed tooth and obtain revascularization of the pulp in infected necrotic immature roots. The case (Fig. 10) describes the treatment of an immature mandibular second premolar with radiographic and clinical signs of apical periodontitis and the presence of a sinus tract. The canal was disinfected without mechanical instrumentation but with copious irrigation with 5.25% sodium hypochlorite and the use of a mixture of antibiotics described above (see Fig. 2).

A blood clot was produced to the level of the cementoenamel junction to provide a scaffold for the in-growth of new tissue, followed by a seal of MTA in the cervical area and a bonded resin coronal restoration above it. With clinical and radiographic evidence of healing as early as 22 days, the large radiolucency had disappeared within 2 months and at the 24-month recall it was obvious that the root walls were thick and the development of the root below the restoration was similar to the adjacent and contralateral teeth.

Our group has confirmed the potent antibacterial properties of the tri-antibiotic paste used in this case (11). A recent study on dogs to evaluate the potential for revascularization and the ability for a collagen-enhanced scaffold showed that the potential for revascularization does exist (Fig. 11). Additionally, this study appears to indicate that it is the blood clot with or without the addition of the collagen-enhanced scaffold that appears most important for the stimulation of the revascularization process (30). Further studies are underway to find other potential synthetic matrices that will act as more predictable scaffolds for new ingrowth of tissue than the blood clot used in these previous cases. A synthetic matrix may also allow easier and more predictable placement of the coronal seal than that provided by a relatively fresh blood clot. The procedure described in this section can be attempted in most cases and if, after 3 months, no signs of regeneration are present, the more traditional treatment methods can be initiated.

Regeneration versus revascularization

Cases such as those illustrated here have been described as examples of pulp regeneration and the beginning of stem cell technology in endodontics. It is important to distinguish between revascularization and pulp regeneration. Presently we can only say with certainty that the pulp space has returned to a vital state, but based on research in avulsed teeth and on a recent study on infected teeth, it is more likely that the tissue in the
pulp space is more similar to periodontal ligament than to pulp tissue (30). It appears that there is about a 30% chance of pulp tissue re-entering the pulp space (31). Future research will need to be done in order to stimulate pulp regeneration from the pluri-potential cells in the periapical region. It may also be a good idea to partially resect the pulp in an irreversible pulpotis case, and with the help of a synthetic scaffold it may be possible to re-grow the pulp rather than remove the entire pulp and replace it with a synthetic filling material.

Fig. 10. Immature tooth with a necrotic infected canal with apical periodontitis. The canal is disinfected by copious irrigation with sodium hypochlorite and tri-antibiotic paste. After 4 weeks the antibiotic is removed and a blood clot is created in the canal space. The access is filled with a mineral trioxide aggregate (MTA) base and a bonded resin is placed above it. At 7 months the patient is asymptomatic and the apex shows healing of the apical periodontitis and some closure of the apex. At 24 months apical healing is obvious and root wall thickening and root lengthening has occurred, indicating that the root canal has been revascularized with vital tissue.

Fig. 11. Revascularization of immature dog tooth with apical periodontitis. The tooth was devitalized and infected to produce apical periodontitis. After 3 months, revascularization has taken place.

References

9. Sato T, Hoshino E, Uematsu H, Noda T. In vitro antimicrobial susceptibility to combinations of drugs on bacteria from carious and endodontic lesions of human


