


Invasive cervical resorption (ICR): A description, diagnosis and discussion of optional management —A review of four long-term cases

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_Abstract

The external resorptive process of the permanent dentition referred to in this article has been given several different terms over the years, so therefore some confusion exists. Just a few popular labels are extra-canal invasive resorption (ECIR), invasive cervical resorption (ICR), external cervical resorption (ECR),

subepithelial external root resorption, and idiopathic external resorption. They all refer to a relatively uncommon form of dental resorption. If left undiagnosed, misdiagnosed, mistreated or untreated, it will usually be quite devastating for a tooth. An Australian dentist, Dr Geoffrey Heithersay, has contributed much to the literature regarding all facets of this type of dental resorption. His work has become the basis of research and treatment. With few changes over the past several years, the aetiology, predisposing factors, classification, clinical and radiological features, histopathology and the treatment of this resorptive process he described are still used in our practice today.^{1-4,6} For this reason, this article will adopt the same nomenclature used in his numerous publications: invasive cervical resorption (ICR).

I will present treatment of four cases—two Class 2 cases, one Class 3 and a Class 4—in an attempt to share some experiences, both good and bad, over the years when dealing with ICR. Hopefully, the following article will be successful in removing some barriers that may currently prevent the doctor from accepting the challenge presented by the next case of ICR.

_Aetiology of invasive cervical resorption

ICR is not a common occurrence, is insidious and often an aggressive form of external tooth resorption, and can occur in any tooth in the permanent dentition.⁴ External resorption can be divided into three broad groups: (a) trauma-induced tooth resorption; (b) infection-induced tooth resorption; and (c) hyperplastic invasive tooth resorption.⁵ ICR is one form of hyperplastic invasive tooth resorption.⁶ It results in the loss of cementum and dentine by an odontoclastic type of action.⁷ The ICR lesion begins just apical of the epithelial attachment of the gingiva at the cervical area of the tooth, but can be found anywhere

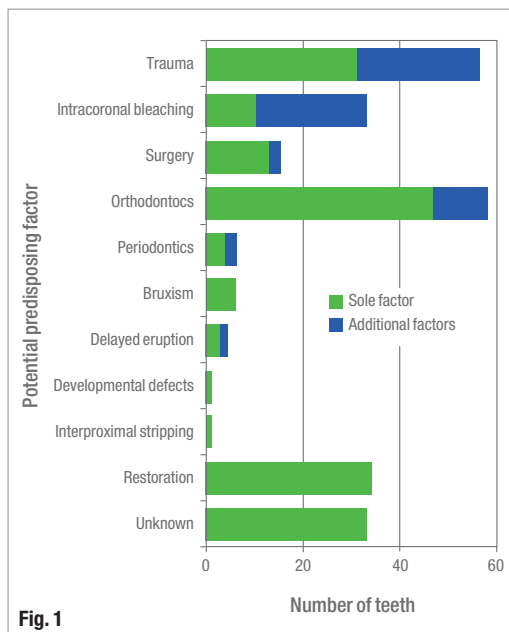
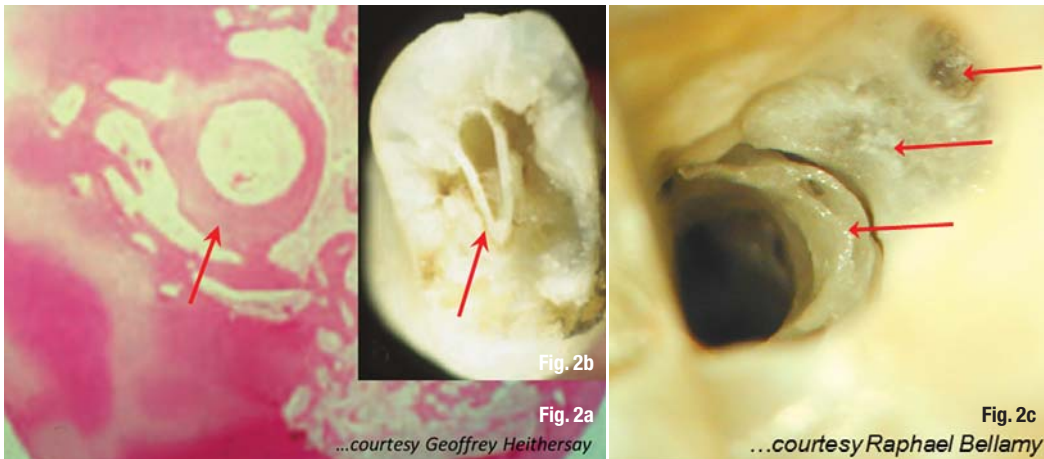


Fig. 1 Invasive cervical resorption: Distribution of potential predisposing factors for patients. (Reproduced with permission from Quintessence Publishing).²



Figs. 2a & b_ The pulp remains intact, encircled by a narrow band of dentine (red arrows). Histologically, the pulp remains intact and is protected from the extensive resorptive lesion by a narrow wall of dentine (a). A low powered photograph shows the walling off of the pulp by dentine, protecting it from the surrounding extensive resorptive process (b). (Slide adaptation reproduced with permission from Dr Geoffrey Heithersay.)

Fig. 2c_ High magnification of the distal orifice of a mandibular second molar being treated for ICR. The pulp remains intact encircled by a narrow band of dentine (bottom arrow). The affected dentine can be observed (middle arrow) and a possible distal penetration area (top arrow). (Slide adaptation reproduced with permission from Dr Raphael Bellamy.)

on the root.⁸ Owing to its location, the beginning lesion is difficult or almost impossible to recognise. The exact mechanism of ICR is still not clearly understood. Microscopic analysis of the cervical region of teeth has shown that there appear to be frequent gaps in the cementum in this area, leaving the underlying mineralised dentine exposed and vulnerable to osteoclastic root resorption.⁹ It is broadly accepted that either damage to or deficiency of the protective layer of cementum apical to the gingival epithelial attachment exposes the root surface to osteoclasts, which then resorbs the dentine.⁷ In general, an area of radicular dentine around the cervical area of the tooth may be devoid of the protective covering of cementum, exposing the root surface to colonisation by osteoclast-like cells, allowing the resorptive process to begin. Osteoclastic action in that area of the radicular dentine eventually results in a hyperplastic resorptive lesion containing fibro-osseous tissue. In order for dental resorption to occur, three conditions are necessary: a blood supply, breakdown or absence of the protective layer, and a stimulus. In the case of ICR, the external protective layer is the cementum, and the internal layer is the predentine of the pulp.

Several potential predisposing factors have been identified: trauma, intracoronal bleaching, surgery, orthodontics, periodontics, bruxism, delayed eruption, developmental defects, interproximal stripping and restoration. Heithersay studied a group of 222 patients with a total of 257 teeth with various degrees of invasive cervical resorption. From the subjects' dental histories, it was determined whether there was a sole predisposing factor, or a combination of factors. The results are shown diagrammatically in Figure 1.² The results indicated that a history of orthodontic treatment was the most common sole factor (found in 47 patients), while other factors, mainly trauma and/or bleaching, were present in an additional 11 subjects. Trauma was the second-most common sole factor, with 31 teeth. Intracoronal bleaching, combined with other factors, had the

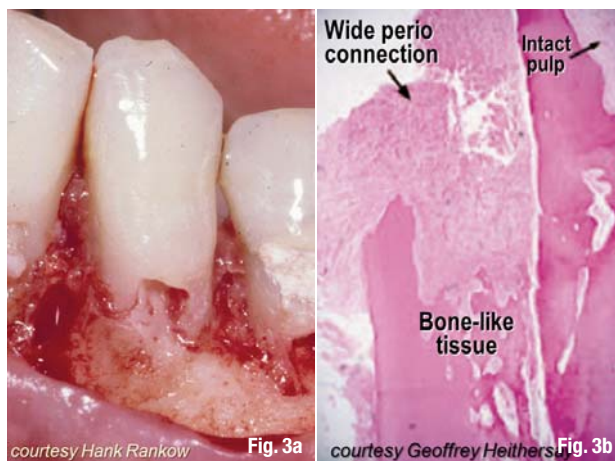
third-most affected teeth.² The pulp plays no role in the aetiology of ICR and remains normal until the ICR becomes very advanced.^{1,7,10}

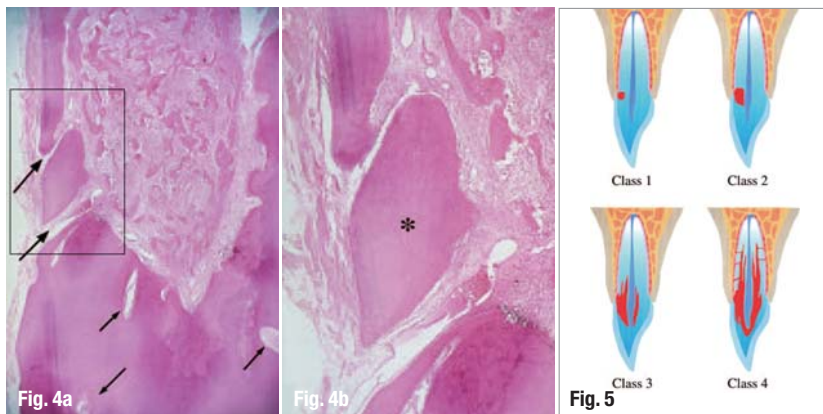
A recently published study has indicated there might be a connection between human and feline ICR. Four cases of multiple invasive cervical resorption (mICR) were presented. There was direct contact with cats in two cases, and indirect contact in the other two cases. Neutralised testing was done for feline herpes virus Type 1 (FeHV-1). Two of the cases were neutralised, and two were partly inhibited. The study indicates a possible transmission of FeHV-1 to humans and the possibility of its role as an aetiological (co)factor in ICR.¹¹

_Histology

An interesting observation is that even in extensive lesions, the pulp is protected from the surrounding resorptive process by a narrow band of dentine (Figs. 2a–c). In some cases of ICR, the clinical and histological views of the lesion substantiate that bone-like tissue has replaced the fibro-vascular tissue located within the resorptive cavity (Figs. 3a & b). In the larger Class 3 and Class 4 lesions, communication channels

Figs. 3a & b_ Both the clinical view (a) and histological view (b) show how the dentine has been extensively replaced by a bone-like tissue. A mass of fibro-vascular tissue infiltrated with inflammatory cells is evident, located within a large resorptive cavity that has a wide connection with the periodontal tissue (large arrow). A small section of intact pulp can be seen on the superior aspect of the section (small arrow). Haematoxylin-eosin stain; original magnification X 30. (Reproduced with permission from Quintessence Publishing and Dr Henry Rankow.)¹





Figs. 4a & b Histological appearance of an extensive ICR with radicular extensions. Masses of ectopic calcific tissue are evident both within the fibro-vascular tissue occupying the resorption cavity and on resorbed dentine surfaces. In addition, communication channels can be seen connecting with the periodontal ligament (large arrows). Other channels can be seen within the inferior aspect of the radicular dentine (small arrows). Haematoxylin-eosin stain; original magnification X 30. Higher magnification (b) shows communication channels from the periodontal ligament to the resorbing tissue. An island of hard tissue remains (*), consisting of an external surface of cementum and cementoid with some residual dentine, but the bulk has been replaced with a bone-like material with a canalicular structure.

Although some red blood cells are evident near the deeper channel, no inflammatory cells can be seen. Haematoxylin-eosin stain; original magnification X 50. (Reproduced with permission from Quintessence Publishing.)¹

Fig. 5 Clinical classification of invasive cervical resorption. (Reproduced with permission from Quintessence Publishing.)²

can be seen connecting with the periodontal ligament. Other channels can also occur within the internal aspect of the radicular dentine (Figs. 4a & b). The larger, more advanced lesions can be described as consisting of granulomatous bone-like fibro-osseous material with a canalicular structure that has extensions into the radicular dentine and periodontal tissue. Osteoclasts might be observed on the resorbing surface within the lacunae.² Over varying amounts of time, the lesion expands apically and coronally, encircling the pulpal tissue that is protected by a thin wall of predentine and dentine.

Clinical classification

Heithersay's clinical classification was developed as a guideline for treatment planning and comparative clinical research.² The classification is shown diagrammatically in Figure 5. The classification allows the operator to determine the probable extent of treatment more precisely. The more extensive the lesion, the more complex the treatment options become.

- _Class 1: Small invasive resorptive lesion with shallow penetration into dentine.
- _Class 2: Well-defined invasive resorptive lesion close to the coronal pulp chamber.
- _Class 3: Deeper invasion extending into the coronal third of radicular dentine.
- _Class 4: A large invasive lesion extending beyond the coronal third of the root.

Normally, a Class 1 lesion can be successfully treated without much difficulty. Class 2 lesions often require minor gingival flap surgery for retraction to achieve adequate access and removal of the affected dentine, and to restore the defect. Class 3 lesions usually involve a surgical approach and/or orthodontic extrusion. Class 1 and 2 lesions can be treated predictably, but the success rate in treating Class 3 and 4 lesions drops dramatically. Thus, in general, as the classification increases, the prognosis decreases.

Diagnosis

The earlier the diagnosis, the more predictable the outcome of treatment will be. Owing to the nature of the lesion, treatment based on an incorrect diagnosis will usually result in continued progression of the resorptive process and eventual loss of the tooth.

Unfortunately, the smaller Class 1 lesion is often not discovered owing to its location beneath the gingival attachment, but will usually show a small radiolucency on a radiograph. The dental examination may reveal a slight irregularity in the gingival contour, which will bleed upon probing.⁴ It is my experience that Class 1 lesions are seldom found during routine dental examinations at this early stage.

One of the problems with early diagnosis is that the lesion is asymptomatic and can remain so even in the more advanced stages. Pulp testing will be of no value because the pulp remains unaffected until late in the process. However, the larger Class 2 lesion can present with more obvious clinical signs. For example, a patient notices a pinkish area on an anterior tooth. The discoloration is the result of osteoclastic activity replacing the radicular structure of the tooth with reddish granulation tissue that shows through the more translucent enamel.

Radiographically, the smaller Class 1 lesion can be confused with a carious lesion, internal resorption or adumbration (cervical burn-out) of the radiograph. If the lesion is a Class 2, Class 3 or Class 4, bitewing radiographs will often present an atypical radiolucency and the examining dentist will be more inclined to believe that it is not just a carious lesion. If the lesion is on the proximal surface of the tooth, the outline of the pulp can usually be observed. The larger lesions can also be misdiagnosed as caries or internal resorption. The usual indication that the lesion is not carious is the irregularity of the radiolucency and/or the radiopaque outline of the protective predentine layer of the pulp (Figs. 6a & b). By utilising varying angulation of the radiographs, internal resorption can be ruled out. If the lesion is due to internal resorption, it will remain centered what the direction, or "off-angle" the radiograph is taken. However, if the lesion is one of ICR, Clark's Rule, or SLOB Rule, can be used to determine the location of the lesion (the most lingual object moves with the direction of the X-ray head). (Figs. 7a & b).

With the advent of Cone Beam Computed Tomography (CBCT), the clinician is given the opportunity to view teeth and anatomical entities in three dimensions. Compare with the typical periapical radi-



Figs. 6a & b The outline of the pulp can usually be observed radiographically (a). The bitewing X-ray (b) will show the ICR lesion and the predentine layer (red arrows). The predentine protective layer can exist even in advanced Class 4 lesions (red arrows).

Figs. 7a & b A definitive way to avoid a misdiagnosis of ICR is to take the X-ray from varying angles, including at a normal position (a). However, when the X-ray is taken from a different, more distal angle (b), the radiograph clearly demonstrates that the lesion is not internal resorption and is positioned to the lingual surface. The protective predentine layer surrounding the pulp is clearly visible.

ographs (Fig. 8a). Even if numerous angles, a complete view of the extent of the lesion cannot be established with any definitive accuracy. The extracted tooth #16 was a hopeless Class 4 lesion involving most of the cervical half of the lingual surface and extending into the area (Fig. 8b). Three planes of sections can be evaluated with CBCT: the frontal/coronal (X), sagittal (Y) and axial (Z; Fig. 8c). The X plane moves anterior \Leftrightarrow posterior (B \Leftrightarrow L in the anterior teeth and M \Leftrightarrow D in the posterior). The Y plane moves left \Leftrightarrow right (M \Leftrightarrow D in the anterior and B \Leftrightarrow L in the posterior). The Z plane moves coronal \Leftrightarrow apical for all teeth in the dental arch. Depending on the machine, up to 512 slices of the field of view can be visualised. The slice thickness is variable, again depending on the machine, from nearly 0.1 to several mm. However, generally speaking, the thinner the slice, the higher the spatial resolution.¹² When evaluating resorptive defects, higher resolution and 3-D images allow the experienced clinician to make a more definitive diagnosis and establish a confident and realistic plan for treatment, with a higher predictability of success.

In summary, the characteristic diagnostic signs that indicate that the lesion is a result of ICR are as follows:

- _The tooth is asymptomatic.
- _The pulp tests are within normal limits.
- _The ICR defect moves with varying X-ray angulations.
- _The protective pulpal wall is often intact and can be seen on the radiographs.
- _The portals of entry are near the osseous crest.
- _The portals of entry are difficult to locate clinically.¹³

I suggest that during the initial dental examination the patient be asked whether any of the three major predisposing factors have occurred in their dental history (bleaching, trauma or orthodontics). ICR can occur in any permanent tooth and once found in a patient, it is important to initiate regular follow-up visits to ensure no further lesions occur.

_Treatment

After the diagnosis of ICR has been confirmed, the treatment should be scheduled as soon as possible. If, for some reason this is not practical, the tooth should be monitored closely. The lesion can be very aggressive, so best not to wait for too long (Figs. 9a–c).

The Heithersay classification is of great help for advising the patient of the extent of treatment and gaining a better idea of the possible prognosis. The patient and doctor can decide on treatment together: (a) no treatment and extraction if the tooth becomes symptomatic; (b) extraction and possible replacement with an implant; or (c) to begin endodontic treatment in an attempt to eliminate the lesion and restore the tooth for as long as possible. In Class 1 and Class 2 cases, the patient must be advised that the treatment will probably be non-surgical but that the surgical approach may be necessary. In the more advanced Class 3 and Class 4 cases, the patient must be advised that both the non-surgical and surgical approaches will be necessary. Dental implants have become popular and, unfortunately, have led to a greater percentage of patients choosing the first two options.⁸ However, there are still enough pa-



Figs. 8a & b Pre-op PAX for extraction of tooth #16 (a). The extracted tooth (b).

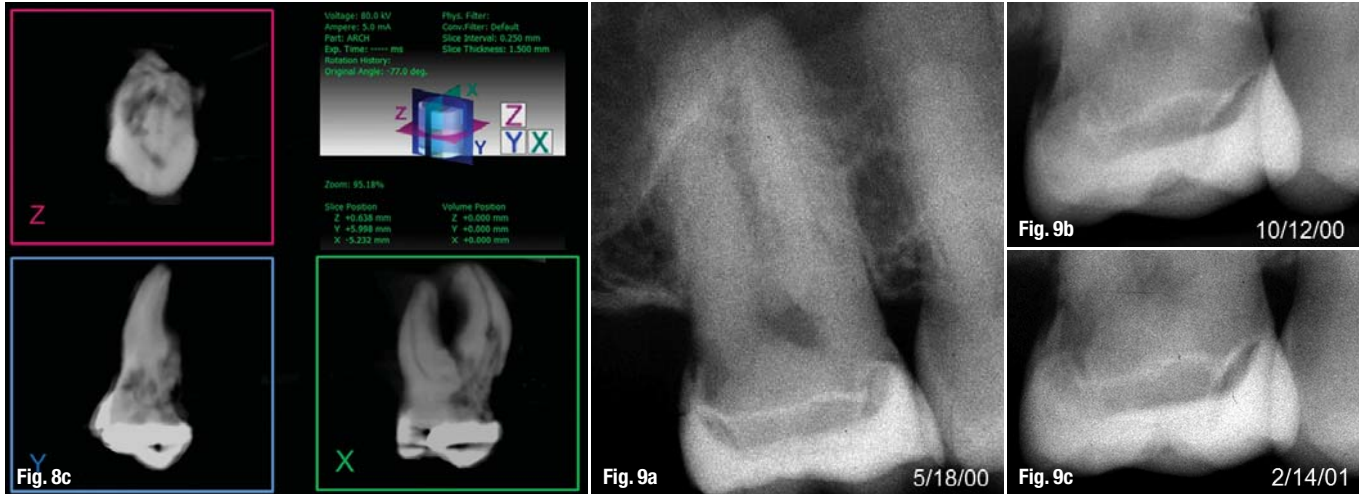


Fig. 8c_CBCT is essential to demonstrate the extent of the lesion and the amount of destruction that has occurred.

Figs. 9a–c_In a matter of just a few months, the lesion had advanced (a).

The diagnosis was made (b). The patient was seen again in five months and was scheduled for treatment (c).

tients who want to save their natural teeth, no matter what.

Heithersay developed what has become the standard guide for the treatment of ICR. Depending on the extent of the lesion, it is accessed either non-surgically or surgically. The granulation tissue is removed with either curettes or a round bur of varying sizes. During the removal of the bone-like tissue, 90% trichloroacetic acid (TCA) is applied with a small cotton pellet numerous times, with increasing pressure, to achieve coagulation necrosis. Using magnification, the fibro-osseous granulation tissue is removed until no communication channels are observed and the defect is lined with unaffected dentine, then restored with an appropriate restorative material. Endodontic treatment is performed when indicated. The aim of treatment is to eliminate all active resorbing tissue and restoration of the defect so the tooth can be maintained for as long as possible.⁴ It has been my experience that all Class 2 to 4 cases required endodontic treatment.

I wish to make something very clear. In the following cases, 90% TCA was not used. There was

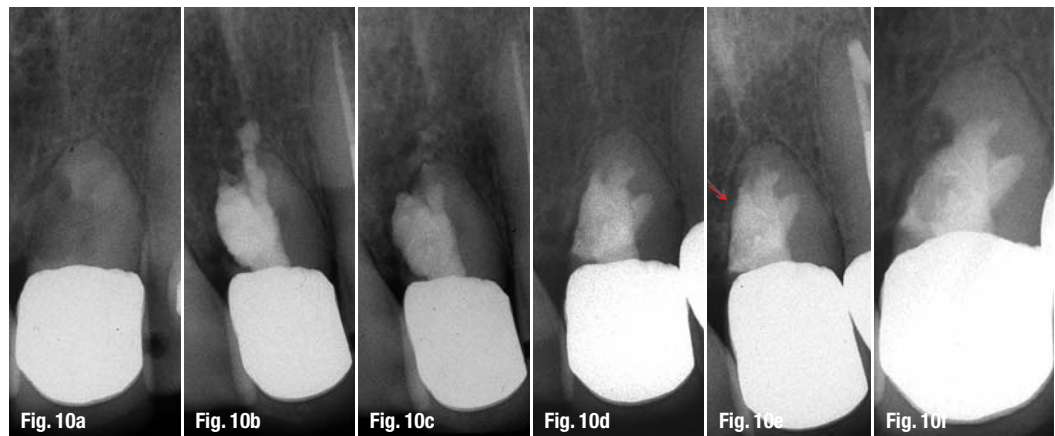
absolutely no disagreement about the use of TCA, but when the cases were treated, it was not available. The cases were treated with what was on hand. As a matter of convenience and necessity, Monsel's solution (MS), a 72% solution of ferric sulphate with sulphuric acid, was used. It had been used for many years as a coagulant when performing apical microsurgery. The use of MS to achieve coagulation necrosis when treating ICR over the years appeared to work well. As a result, the use of MS was continued.

_First patient

In 1993, a 62-year-old male patient presented for an evaluation of tooth #21 (Fig. 10a). His general dentist had recommended that the tooth to be extracted. At that time, a definitive protocol for the diagnosis and treatment of ICR had not been established. But the patient wanted us to do something to save the tooth. Sensing the sincerity of the patient, we agreed to attempt the salvation of the tooth, but informed him that we could not guarantee the outcome. At that time, there were some practising endodontists participating in clinical research for Dr Torabinjad using mineral trioxide aggregate (MTA)

Figs. 10a–c_Pre-operative X-ray (a). Immediate post-op MTA fill (b). Seven-month FUV (c).

Figs. 10d–f_Immediate post-op radiograph following microsurgical recontouring and enhancement of previous MTA fill (d). The 44-month FUV indicated an intact periodontal ligament (e; red arrow). Pre-op radiograph for extraction of tooth at 81 months (f). When comparing (f) with (e), there appears the probability of a continuation of the resorptive process (arrow).





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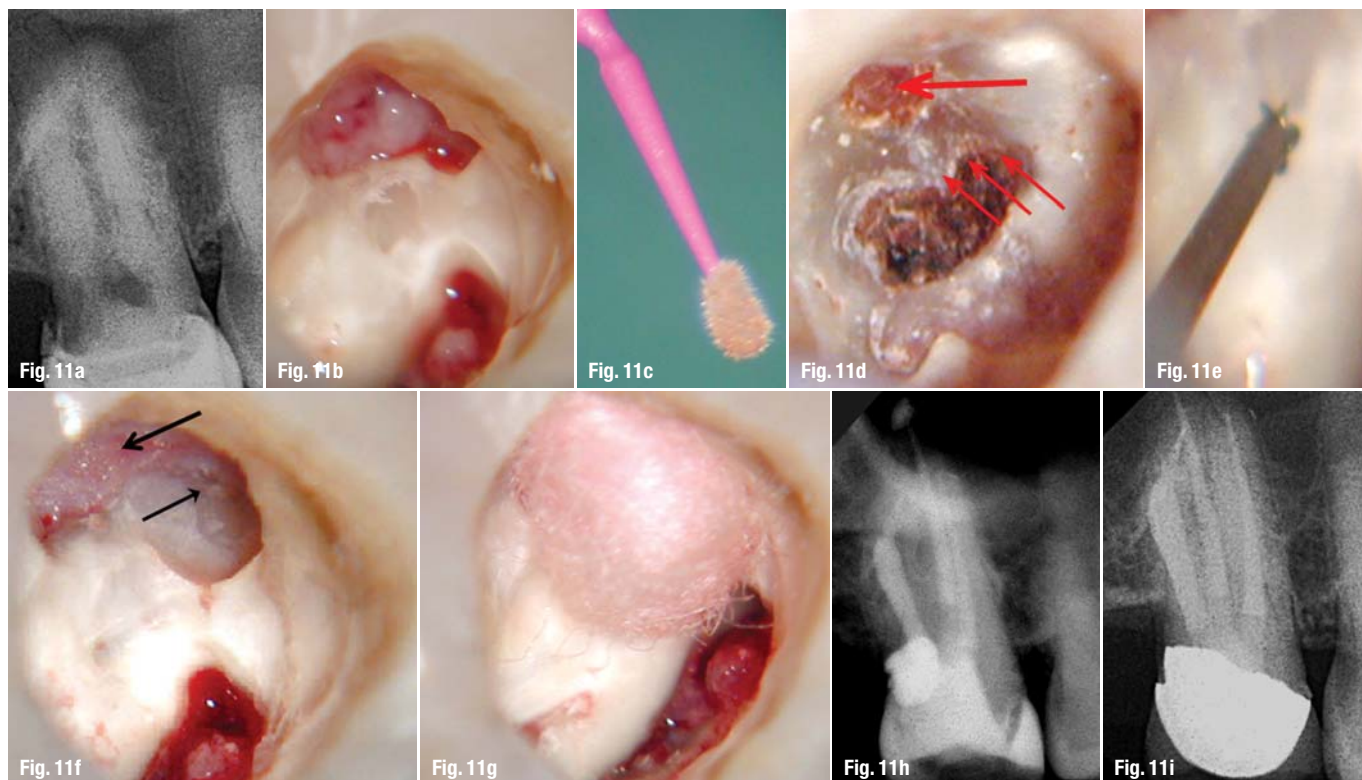
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Figs. 11a & b—The updated pre-op radiograph indicated an advanced Class 2 ICR (a). After gross removal of granulation tissue, the affected dentine could be seen (b).

Figs. 11c–f—A micro-brush was used to apply Monsel's Solution and brushed onto the floor of the defect (c). After application of the MS, the area was irrigated with NaCl, rinsed and dried for inspection of remaining affected dentine (d). Note the perforation in the distal surface of the tooth (large arrow), and remaining affected dentine (small arrows; f). A variety of Muncie burs are available for removal of the affected dentine.

Figs. 11f & g—Most of the lesion has been removed, resulting in a perforation of the distal wall of the chamber into the gingival sulcus (f; large arrow). A smaller Muncie bur will be necessary to remove slightly more of the affected dentine (small arrows). The epinephrine-soaked pellet was placed for an adequate time for haemostasis before bonding a glass ionomer repair (g).

Figs. 11h & i—The immediate post-op radiograph (h). The four-year FUV radiograph indicates a healed tooth (i).

in pulp capping, perforations and root-end filling.¹⁴ I had used MTA on just a few patients previously and had some confidence that it might serve as a last hope in this case. The MTA was an easy material to work with and required moisture to set completely. After the access was created, the obvious haemorrhaging was difficult to control whenever more granulation tissue was removed using curettes and a #6 round bur. Ferric subsulphate (MS) was repeatedly used for haemostasis, then irrigated with a 50% sodium hypochlorite solution (NaCl) and rinsed with sterile water, then gently air dried using the Stropko Irrigator (DCI). Haemostasis was achieved and vision was maintained while using an Aus-Jena surgical operating microscope (SOM) fitted with a co-observer tube for the assistant. After shaping to a #80 Kerr file at the terminus, and removing as much granulation tissue as possible, the canal system and defect were again copiously irrigated and dried as well as possible. Owing to the size of the apical opening, extra-large absorbent paper points (Kerr) were used to remove any remaining moisture and the entire case was obturated using MTA. The post-operative radiograph indicated that a significant amount of excess MTA was extruded (Fig. 10b). The patient was dismissed and reported no post-operative problems.

At the seven-month follow-up visit (FUV), the tooth #21 was totally asymptomatic, but I was concerned with the appearance of the very obvious overfill on the radiograph and wanted to eliminate

the excess MTA with a surgical approach. If the patient was seen by another dental office in the future, one could imagine someone saying, "Who in the heck did this to your tooth?" (Fig. 10c). On the appointed day, a full gingival flap was created and access to the area was achieved. In order to minimise the vibration that would be created when trimming the excess MTA from the root surface, a high-speed surgical handpiece with fibre optics (Impact Air 45 Star Dental), fitted with a surgical length, taper fissure #1171 bur (SS White), was used. After a satisfactory root profile had been established, a very small, inverted-cone, surgical length #330 bur (SS White) was used to prepare any of the lesion's periphery that was missed during the original non-surgical treatment. Once the necessary "troughing" had been completed, new MTA was added to the originally placed MTA for a more complete seal (Fig. 10d). Sutures were removed in a few days, and healing was uneventful. Regular FUVs were scheduled. A radiograph taken at the 44-month FUV was diagnosed as healing complete with an intact periodontal ligament (Fig. 10e).

About four years later, the patient returned with a three-unit fixed bridge replacement of tooth #21. The patient stated that tooth had become very loose and it was removed. The preoperative extraction radiograph was located (Fig. 10f). However, later comparison of the 44-month FUV to the pre-extraction radiograph indicated a possible continuation of the resorptive process—isn't it amazing what you can see when the light is just right?

_Second patient

A 64-year-old male patient presented for evaluation of tooth #17 because of the unusual appearance of the distal surface of the tooth. The previous clinical examinations and radiographs over the past ten months had diagnosed ICR (Figs. 11a–c). An updated radiograph was taken, all options were explained to the patient, and endodontic treatment was initiated (Fig. 11a).

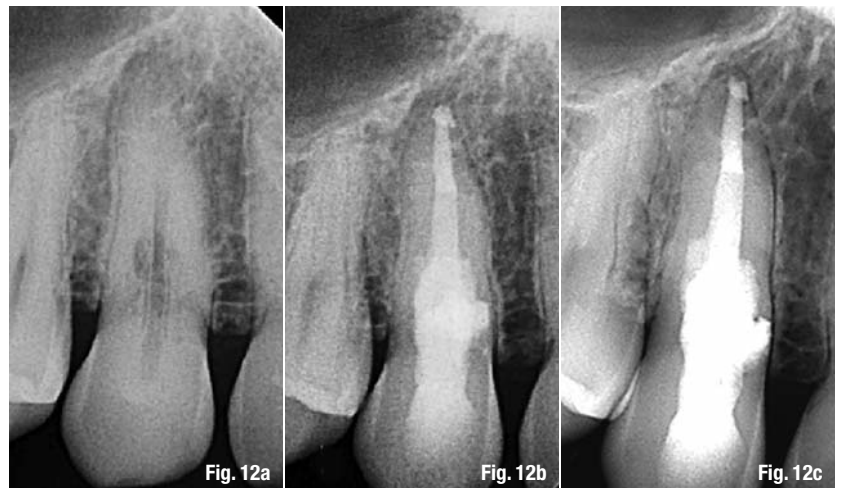
The tooth was accessed and a gross removal of fibrous granulation tissue was achieved using curettage. The chamber was copiously irrigated with NaCl, rinsed and dried gently. The ICR dentinal defect and granulation tissue were evaluated to obtain a better understanding of its position in relation to the distal wall of the access and to the pulp tissue (Fig. 11b). A micro-brush dipped in MS was applied to the involved area (Fig. 11c). The MS is used for coagulation necrosis and to display the affected dentine that needs removal. It is not necessary to use copious amounts when applying the MS. Instead it is best to rely more on a sequence of repeated brushing with MS, irrigation with NaCl, rinsing and gentle drying. Study under the SOM at varying magnifications for affected dentine (Fig. 11d). Then, if necessary, remove more of the affected dentine using varying sizes of Muncce burs (CJM Engineering; Fig. 11e). This process is repeated as necessary to achieve adequate vision. The floor of the access should be observed under the microscope at varying magnifications to determine whether any affected dentine remains. A celluloid strip was placed in the distal sulcus to act as a barrier to the flowable glass ionomer restoration (Fig. 11f). An epinephrine-soaked cotton pellet (EpiDry, Pascal) was also used to maintain haemostasis and enable the attempt at a non-surgical repair of the defect (Fig. 11g). The defect was etched and restored with a bonded glass ionomer, allowing the maintenance of sterility in the remaining chamber until the endodontic treatment had been completed. The pulp tissue was extirpated and canal system partially shaped. Enough calcium hydroxide (CaOH) was injected into the canals to cover the floor of the chamber, capped with a cotton pellet, and sealed with a bonded composite as a temporary restoration.

Two weeks later, the patient was scheduled to complete the endodontic treatment. During the process, a #6 file separated in the apical third of the distobuccal canal and had to be retrieved. At the final visit, the canal system was obturated using a Calamus (DENTSPLY Tulsa) for the injection of pre-warmed gutta-percha to the terminus. A bonded composite core was placed to seal the rest of the canal system and facilitate future restoration with a crown (Fig. 11h). The restorative dentist extended the distal margin of the full crown well apical to the distal defect for a good seal. The four-year FUV radiograph demonstrated complete healing (Fig. 11i).

_Third patient

A 47-year-old male patient presented for evaluation of "a small area of tingling, or numbness to the right of the nose". The initial radiograph was classic for ICR (Fig. 12a). All options were explained, and endodontic treatment was initiated.

The tooth was accessed, and as much fibrous granulation tissue was removed as possible. Monsel's solution was applied using a micro-brush to achieve coagulation necrosis. Then the chamber was irrigated with NaCl, rinsed with sterile saline, and gently dried using the Stropko Irrigator. The floor of the access was observed under the microscope at varying magnifications to determine whether any affected dentine was present. Any remaining affected dentine was efficiently removed with various Muncce burs. Then CaOH was sealed in with a bonded composite as a temporary restoration.



Two weeks later, the patient was seen in order to complete the non-surgical part of the treatment. The final shaping and cleaning was done, and the canal was filled to the terminus by injection of pre-warmed gutta-percha using a Calamus. A bonded FibreKor post (Pentron) with a bonded composite core (Core Paste, DenMat), was placed to seal the rest of the canal system and DenMat Marathon to repair the access opening. Then a simple flap was reflected to expose the lingual defect so it could be prepared and restored with bonded Geristore (DenMat; Fig. 12b). Healing was uneventful, and the numbing sensation beside the patient's nose was resolved. The radiograph at the four-year FUV showed uneventful healing (Fig. 12c).

Figs. 12a–c—The pre-op radiograph clearly demonstrates the appearance of the protective layer and the typical mottled radiolucent appearance of the resorptive lesion (a). The radiograph immediately after non-surgical obturation and surgical repair of the lesion (b). Normal healing is apparent in the four-year FUV radiograph (c).

_Fourth patient

This 64-year-old male patient was referred by his general dentist because of the unusual radiographic appearance of tooth #43 (Fig. 13a). Even though there were no symptoms present, the referring doctor was

Figs. 13a & b At the initial visit, the radiograph for tooth #43 in a 64-year-old male patient was taken at a normal angle (a). Taking a radiograph from a more distal angle demonstrated that the lesion was located to the lingual of the tooth. Both radiographs clearly show the protective dentinal wall surrounding the pulp (b).

concerned about the integrity of the tooth. Routine off-angled periapical radiographs were taken. The distal off-angled radiograph clearly indicated that the lesion was on the lingual surface of the tooth (Fig. 13b). Both radiographs clearly showed the thin predentine/dentinal wall protecting the pulp. His dental history revealed that he had had complete orthodontics during his early teens. In addition, the patient stated that tooth #43 had become misaligned about 20 years

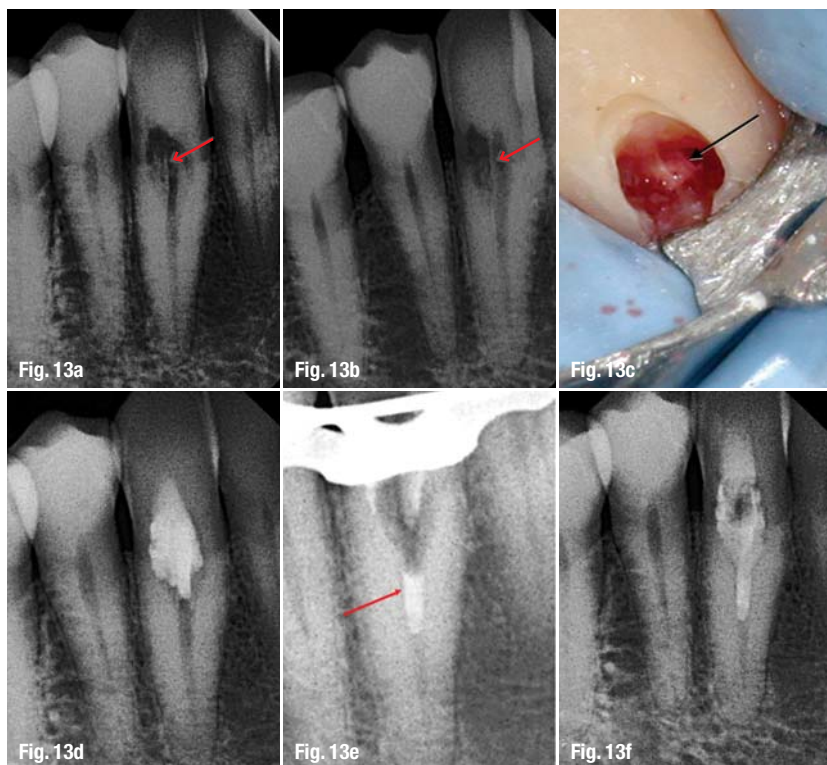
Microscopic description: Histological examination reveals multiple pieces of soft and hard tissue composed chiefly of inflamed granulation and fibrous connective tissues with bone and tooth structure. The granulation and fibrous tissues consist of interlacing bundles of dense to more delicate collagen fibres supporting varying numbers of fibroblasts, fibrocytes and small blood vessels. A mild infiltrate of chronic inflammatory cells, chiefly lymphocytes and plasma cells, is present within this tissue. Also prominent within our specimen are scattered trabeculae of bone containing osteocytes within lacunae, as well as fragments of dentinal tooth structure and calcified debris.

Diagnosis: Right posterior mandible, lingual aspect of tooth #43. Histological findings consistent with idiopathic external resorption.

Gradually, as more of the tissue was removed, the bleeding noticeably decreased, but haemostasis was not achieved. As an interim medication, a thick mixture of white MTA was firmly placed into the chamber, covered with a sterile cotton pellet and temporarily restored with a bonded composite (Fig. 13d). During the initial examination, pulp testing indicated a normal pulp, and I was wondering whether the vitality of the tooth could possibly be maintained at that point. All options, including the possible need for conventional root-canal treatment, were explained. Both the doctor and patient agreed to attempt to maintain the vitality of the tooth. The patient was rescheduled for a second visit in about two weeks.

During the second visit, the chamber was reopened, the MTA was eliminated and more granulation tissue was removed with small curettes. Under varying high magnifications of the microscope (Global Surgical Corporation), as much of the remaining affected dentine was removed with Muncce burs, and the remaining pulp tissue was identified. After irrigation with NaCl, an additional few millimetres of the pulp was removed and a pellet of grey MTA was placed into the canal using a medium Dovgan MTA Carrier (Quality Aspirators; Fig. 13e). CaOH was then placed, covered with a cotton pellet, and sealed in with a bonded composite temporary (Fig. 13f). To allow for a complete set of the MTA, the patient was scheduled two days later for a third appointment.

On this third visit, the CaOH was removed, and the floor of the defect was lightly brushed with Muncce burs of various sizes and studied under varying magnifications until no affected dentine was observed. The chamber was irrigated with NaCl, rinsed, gently dried and etched with 35% phosphoric acid gel (Ultra-etch, Ultradent). A bonded core was placed



Figs. 13c & d The initial access demonstrated the unusual texture of the fibrous bone-like granulation tissue in the coronal area (c; arrow). After removal of most of the granulation tissue, white MTA was placed into the chamber and a temporary placed (d).

Figs. 13e & f At the second visit, in an attempt to maintain the vitality of the pulp, MTA was placed into the canal (e; arrow). CaOH was placed as an inter-appointment medication and to allow the MTA to achieve a complete set (f).

Figs. 13g & h After a few days, the chamber was irrigated and a bonded core was placed and was finished with a bonded composite (DenMat; g). The two-month FUV radiograph demonstrated that the attempt to maintain the vitality of the tooth had not been successful (h).

ago. In order to correct the misalignment of the tooth, the doctor reduced the tooth on each side and repositioned it with a removable appliance. Clinical examination was essentially within normal limits, except that a 4–6–4 mm periodontal probing of the lingual tissue resulted in moderate bleeding. All teeth in the posterior quadrants had been restored with full porcelain coverage, and the occlusion was a normal Class 1 molar relationship. All pulp tests were within normal limits. The diagnosis was clearly a Class 3 ICR. All things were explained to the patient, and we agreed to be as conservative as possible during treatment. At the time, I had no idea what a learning experience this case would be.

At the first visit, the initial access confirmed the diagnosis. The granulation tissue consisted of granules of bone-like haemorrhagic tissue (Fig. 13c). The pulpal wall was very thin, and pieces would come out with the granulation tissue. During the curettage, the fibrous tissue resembled a "crumbling sponge made of bone that was soaked with blood". Pieces of tissue were sent to an oral pathology laboratory for the following definitive diagnosis:



and access restored with a bonded composite (Fig. 13g). An FUV was scheduled for two months later.

At the two-month FUV, the periapical radiograph revealed that the response to treatment was not as expected. The ICR lesion had significantly progressed in that short time (Fig. 13h). All options were discussed with the patient, but there was no doubt that conventional endodontic treatment, followed by surgical repair of the lesion would be necessary.

During this visit, adequate access was created to remove the previously placed MTA. Using Gates Glidden burs and a #4 round bur, more affected dentine was removed in the coronal aspect of the canal. The canal system was then shaped and cleaned to the terminus, and CaOH was sealed in with a bonded composite temporary (Fig. 13i). After about ten days, the canal system was obturated by the injection of pre-warmed gutta-percha to the terminus with a Calamus. Then, most of the core was removed and a post space created. A fibre post with a composite core was bonded in, and the access opening filled with Geristore to prepare the tooth for the surgical repair of the ICR lesion (Fig. 13j).

A sulcular flap was reflected enough to adequately access the ICR lesion. The lesion was at the lingual crestal bone and had been slightly stained from the previous use of Monsel's Solution (Fig. 14a). After gross removal of the remaining granulation tissue and affected dentine, more solution was applied with a micro-brush to achieve coagulation necrosis and the stained affected dentine was removed using smaller Munce burs (Fig. 14b). The process was repeated until all affected dentine had been removed. After the preparation had been completed, a bonded Geristore was placed (Fig. 14c). A radiograph from a normal view indicated that the restoration was appropriate (Fig. 14d). A radiograph was taken from a distal off-angle view to ensure that post-operative integrity had been achieved (Fig. 14e).

The patient was followed at regular intervals. The most recent FUV occurred after over eight years

post-operatively. The patient remained asymptomatic from the beginning to the end of the entire treatment process. The recent buccal view does show a slight grey shadow in the cervical half of the tooth as a result of the earlier use of the MTA when trying to maintain the vitality of the pulp (Fig. 14f). Apart from normal staining, the lingual view was within normal limits and the periodontal probing was still 5 mm (Fig. 14g). The post-operative radiographs indicate complete healing with good integrity of the fill (Figs. 14h & i). Note that the excess filling material from the original obturation (Fig. 13i) was resolved.

Discussion

Unfortunately, ICR is normally not detected in its early stages and/or is often misdiagnosed. By the time it is discovered, the resorptive process is advanced enough to be at least a Class 2 or worse. Fortunately, ICR is not a very common occurrence in an endodontic practice, though it can be quite demanding of our time. Some Class 2 ICR cases and all Class 3 and Class 4 cases, with rare exception, will involve conventional endodontic treatment.

Figs. 13i & j The MTA was removed, the canal shaped, and CaOH sealed with a bonded temporary (i). The canal was obturated with gutta-percha and a bonded fibre post with a composite core was placed (j).

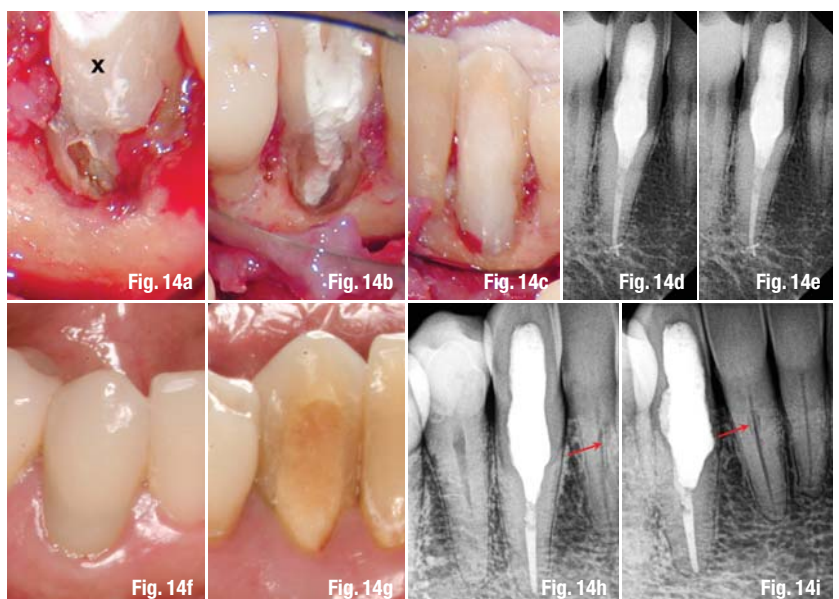
Figs. 14a & b After a surgical flap had been raised, the ICR lesion was observed at the lingual crest of bone. "X" is the glass ionomer placed at the pre-surgical appointment (a).

After each application of Monsel's solution, the granulation tissue was removed with various sizes of Munce burs. The apical portion of the preparation is shown (b).

Figs. 14c-e A bonded glass ionomer was placed and cured (c). Immediate post-op radiograph (d). The radiograph from a distal off-angle shows integrity of the post and core (e).

Figs. 14f & g The eight-year FUV indicates healthy tissue on both the buccal (f) and the lingual views (g). Note the slight grey shadow left as the result of using grey MTA at the beginning of the non-surgical treatment.

Figs. 14h & i Radiographs taken at a normal angle (h) and at a mesial off-angle (i) also indicate complete healing.



The diagnosis of ICR is made more precise with currently available radiographic technology. Digital radiographs and CBCT have set a new standard of clinical management, allowing more predictable results with less stress. The 3-D view presented by CBCT removes many of the unknowns from the diagnosis.

In today's world, the use of a surgical operating microscope (SOM) is essential to enable the operator to overcome the difficulty of treating ICR cases. The variable magnification and superior lighting of the SOM give the operator the enhanced vision necessary to treat ICR cases with less stress and a higher probability of success. Having a dental assistant involved, using a co-observer tube during any dental procedure, is an incredible help because now he or she is able to see what you see at the time you see it and better anticipate what is needed next.

In all cases presented, Monsels solution (MS) was used successfully for coagulation necrosis. Based on an early report, I used it routinely during microsurgery for crypt management.¹⁵ As a result, when the first case of ICR presented for treatment, 90% TCA acid was not a familiar alternative protocol. Having never used TCA, I can offer no comparison or comment. The original protocol for the clinical management of ICR using 90% TCA, suggested by Heithersay in 1999, is still the most popular and well documented. There are various techniques for restoring a tooth with ICR, as previously described in the literature, that are different from what is presented in this publication. However, the real purpose in the treatment of ICR was, and still is, to eliminate as much of the affected dentine as possible. If this is not achieved, the process will progress and be a disaster for the tooth.

While MTA was extensively used in the first case presented (Figs. 13a–g), I do not intend to suggest the use of it as a material for the repair of ICR defects. I did that case almost 20 years ago. Today the materials of choice would be bonded glass ionomers or composites for their strength and adhesiveness. MTA is currently used as a pulp capping material, for perforation repairs or as a restorative material for the repair of a radicular defect that is apical to the osseous crest.

It is important to remember that unless the challenge to treat a seemingly hopeless or extremely difficult case is accepted, the opportunity to learn what can be accomplished is lost. Experience has shown that in such cases there have been more pleasant and favourable surprises than unpleasant results. As William F. O'Brien said, "It is better to try and fail, than to not try at all." Hindsight is always 20-20, and it is one of the best teaching tools we always have at our disposal. The important thing is to learn from our mistakes and those of others.

If a tooth can be saved for only a few years, the rapid advancement of technology will permit a significantly better treatment in the future. So, if an opportunity is presented to save the tooth, then why not? If the question remains, the words of Dr Herbert Schilder are pertinent, "Make yourself the patient, and you have the answer!" The important consideration is what is in the best interest of the patient. Remember, an implant can always be done, and should be the last resort.

In conclusion, the quote from Dr Henry Rankow gives the best explanation of the predicament presented for the clinical management of this lesion, "ICR is an 'outside-in' problem that is very difficult to treat 'inside-out'!"

_about the author

roots



John J. Stropko received his DDS from Indiana University in 1964. For 24 years, he practised restorative dentistry. In 1989, he received a certificate for endodontics from Boston University and has recently retired from the private practice of endodontics in Scottsdale, Arizona. Stropko is an internationally recognised authority on micro-endodontics and has performed numerous live micro-endodontic and microsurgical demonstrations. He has been a visiting clinical instructor at the Pacific Endodontic Research Foundation, an adjunct assistant professor at Boston University, an assistant professor of graduate clinical endodontics at Loma Linda University, and a member of the endodontic faculty at the Scottsdale Center for Dentistry in Scottsdale, as an instructor of microsurgery; and is a co-founder of Clinical Endodontic Seminars. His research on in vivo root-canal morphology has been published in the *Journal of Endodontics*. He is the inventor of the Stropko Irrigator, has published in several journals and books, and is an internationally known speaker. Stropko and his wife currently reside in Prescott, Arizona.

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