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 labeled several different names over the years; 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, idiopathic external resorption, etc. 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. Australian dentist 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 etiology, predisposing factors, classification, clinical and radiologic features, histopathology and the treatment of this resorptive process he described are still used without much change in our practice today.[1–4,6] In respect, this article will use the same nomenclature appearing in his numerous publications: invasive cervical resorption (ICR).

The author 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, accumulated over the years when dealing with ICR. Hopefully this article will be successful in removing some barriers that may currently prevent the dentist from taking on the challenge presented by the next case of ICR.

_Etiology of invasive cervical resorption

Not a common occurrence, ICR is an insidious and often 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: 1) trauma-induced tooth resorption; 2) infection-induced tooth resorption; and 3) hyperplastic invasive tooth resorption.[5] Invasive cervical resorption 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 to the epithelial attachment of the gingiva at the cervical of the tooth, but can be found anywhere on the root.[8] Due to its location, the beginning lesion is difficult or almost impossible to recognize. The exact mechanism of ICR is still not clearly understood. Microscopic analysis of the cervi-
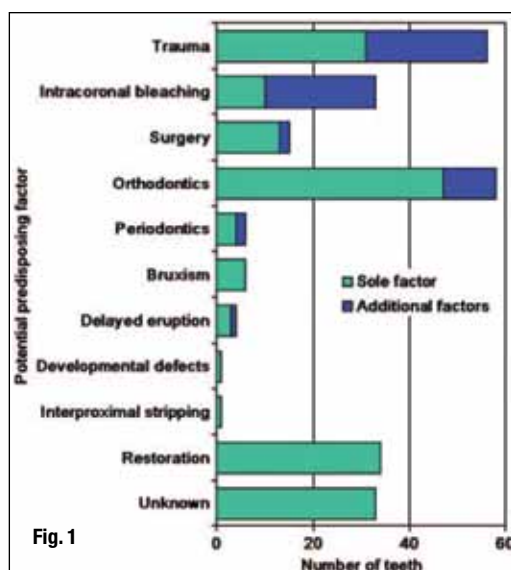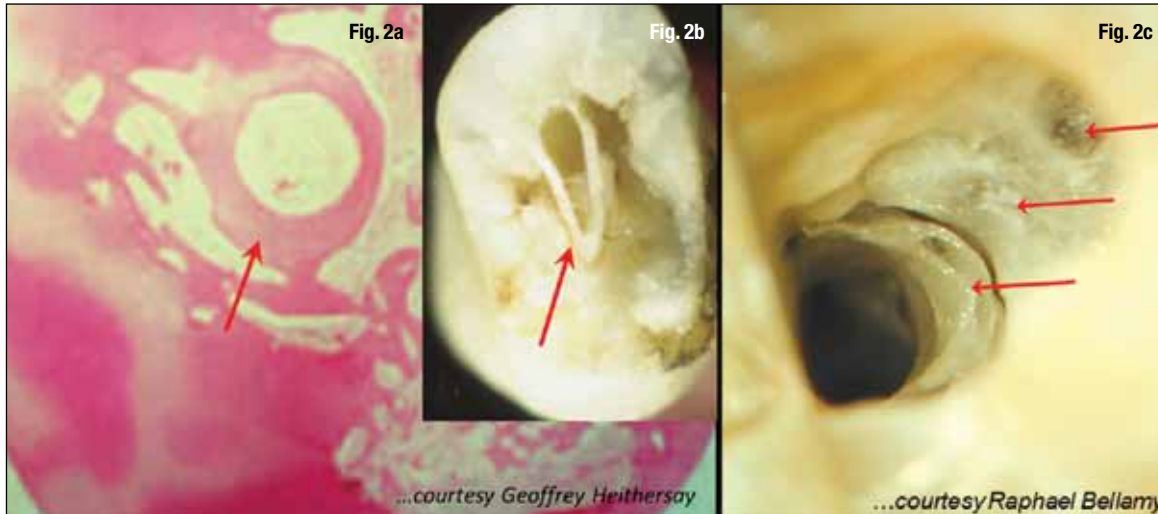

Fig. 1_Invasive cervical resorption: Distribution of potential predisposing factors for patients.[2] (Reproduced with permission from Quintessence Publishing.)

Fig. 2a

Fig. 2b

Fig. 2c

...courtesy Geoffrey Heithersay

...courtesy Raphael Bellamy

cal region of teeth has shown that there appears to be frequent gaps in the cementum in this area, leaving the underlying mineralized dentin 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 dentin.[7] In general, an area of radicular dentin around the cervical area of the tooth may be devoid of the protective covering of cementum, exposing the root surface to be colonized by osteoclast-like cells, allowing the resorptive process to begin. Osteoclastic action on that area of the radicular dentin eventually results in a hyperplastic resorptive lesion containing fibro-osseous tissue. In order for dental resorption to occur, three conditions have to be present: 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 predentin of the pulp.

Several potential predisposing factors have been identified. They are: trauma, intracoronal bleaching, surgery, orthodontics, periodontics, bruxism, delayed eruption, developmental defects, interproximal stripping and restoration. Heithersay used 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. Intra-coronal bleaching, combined with other factors, had the third most affected teeth.[2] The pulp plays no role in the etiology of ICR and remains normal until the ICR becomes very advanced.[1,7,10]

An interesting study was recently published indicating there might be a connection between human and feline ICR. Four cases of were presented of multiple invasive cervical resorption (mICR). There was direct contact with cats in two cases, and indirect contact in the other two cases. Neutralized testing was done for feline herpes virus type 1 (FeHV-1). Two of the cases were neutralized, and two were partly inhibited. The study indicated a possible transmission of FeHV-1 to humans and the possibility of a role as an etiologic (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 dentin (Figs. 2a–c). In some cases of ICR, the clinical and histological views of the lesion substantiate that bonelike tissue has replaced the fibro-vascular tissue located within the resorptive cavity (Figs. 3a,b). In the larger Class 3 and Class 4 lesions, communicating channels can be seen communicating with the periodontal ligament. Other channels can also occur within the internal aspect of the radicular dentin (Figs. 4a,b). The larger, more advanced lesions can be described as consisting of granulomatous bonelike fibro-osseous material with a canalicular structure that has extensions into the radicular dentin 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 predentin and dentin.

_Clinical classification

Heithersay's clinical classification was developed as a guideline for treatment planning and for comparative clinical research.[2] The classification is shown diagrammatically in Figure 5. The classifica-

Figs. 2a, b_The pulp remains intact, encircled by a narrow band of dentin (red arrows). Histologically, the pulp remains intact and is protected from the extensive resorptive lesion by a narrow wall of dentin (a). A low powered photograph shows the walling off of the pulp by dentin, 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 lower second molar being treated for ICR. The pulp remains intact, encircled by a narrow band of dentin (bottom arrow). The involved dentin (middle arrow) and a possible distal penetration area (top arrow) can be observed. (Slide adaptation reproduced with permission from Dr. Raphael Bellamy.)

Figs. 3a,b_Both the clinical picture (a) and histological view (b) show how the dentin has been extensively replaced by a bonelike tissue. A mass of fibrovascular tissue infiltrated with inflammatory cells is located within a large resorptive cavity that has a wide connection with the periodontal tissue (large arrow). The dentin has been extensively replaced by bonelike tissue. A small section of intact pulp can be seen on the superior aspect of the section (small arrow). Hematoxylin-eosin stain; original magnification X 30.[1] (Reproduced with permission from Quintessence Publishing and Dr. Henry Rankow.)

Figs. 4a,b_Histologic appearance of an extensive invasive cervical resorption with radicular extensions. Masses of ectopic calcific tissue are evident both within the fibrovascular tissue occupying the resorption cavity and on resorbed dentin surfaces. In addition, communicating 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). Hematoxylin–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; some residual dentine; but the bulk has been replaced with a bonelike material with canalicular structure. Although some red blood cells are evident near the deeper channel no inflammatory cells can be seen. Hematoxylin-eosin stain; original magnification X 50.[1] (Reproduced with permission from Quintessence Publishing.)

Fig. 3a   Fig. 3b
courtesy Hank Rankow   courtesy Geoffrey Heathersay

Fig. 4a   Fig. 4b
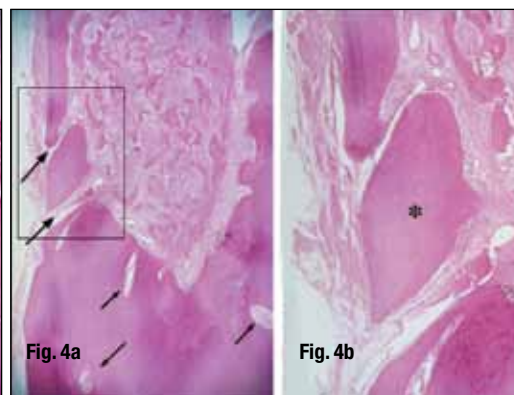
tion allows the operator to more precisely determine the probable extent of treatment. The more extensive the lesion, the more complex the treatment options become.

Class 1: Small invasive resorptive lesion with shallow penetration into dentin.

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. But Class 2 lesions often require minor gingival flap surgery for retraction to achieve adequate access, removal of the affected dentin and restoration of 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. So, in general, as the classification increases, the prognosis decreases.

_Diagnosis

The earlier the diagnosis, the more predictable the outcome of treatment will be. Due 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 due to its location beneath the gingival attachment; but it 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 the author's experience that Class 1 lesions, at this early stage, are seldom found during routine dental examinations.

One of the problems with early diagnosis is 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. On the other hand, 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 is showing through the more translucent enamel.

Radiographically, the smaller Class 1 lesion can be confused with a carious lesion, internal resorption or adumbration (cervical burnout) 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 isn't just a carious lesion. If the lesion is on the proximal 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 predentin layer of the pulp (Figs. 6a,b). By utilizing varying angulation of the radiographs, internal resorption can be ruled out. If the lesion is internal resorption, it will remain centered whatever 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 radiographs (Fig. 8a). Even if numerous angles are taken, a complete view of the extent of the lesion cannot be established with any definitive accuracy. The extracted tooth #3 was a hopeless Class 4 lesion involving most of the cervical half of the lingual 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"–frontal/coronal plane moves anterior–posterior (B–L in the anterior teeth and M–D in the posterior). The "Y"-sagittal plane moves left–right (M–D in the anterior and B–L in the posterior). The "Z"-axial plane moves coronal–apical for all teeth in the dental arch. Depending on the machine, up to 512 slices of the field of view

Fig. 5 | Fig. 6a | Fig. 6b _... courtesy Joe Dovgan_ | Fig. 7a | Fig. 7b

(FOV) can be visualized. 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 spacial resolution.[12] When evaluating resorptive defects, higher resolution and three-dimensional images allow the experienced clinician to make a more definitive diagnosis, a confident and realistic plan for treatment, with a higher predictability of success.

So, in a nutshell, the characteristic diagnostic signs that indicate the lesion is a result of ICR are as follows: 1) The tooth is asymptomatic; 2) The pulp tests are within normal limits; 3) The ICR defect moves with varying X-ray angulations; 4) The protective pulpal wall is often intact and can be seen on the radiographs; 5) The portals of entry are near the osseous crest; and 6) The portals of entry are hard to locate clinically.[13]

During the initial dental examination, the author suggests the patients should be asked if any of the three major predisposing factors have occurred in their past dental history (bleaching, trauma or orthodontics). And, as a side note, "Do you have any cats?" ICR can occur in any permanent tooth and once found in a patient, it is important to initiate regular follow-up visits to be sure 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 and watch" for too long a time, never more than two to three months (Figs. 9a–c).

The Heithersay Classification is of great help to advise the patient of the extent of treatment and get a better idea of the possible prognosis. The patient and doctor together can decide: 1) No treatment and extraction if the tooth becomes symptomatic; 2) Extraction and possible replacement with an implant; or 3) 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 is advised the treatment will probably be nonsurgical, but the surgical approach may be necessary. In the more advanced Class 3, and Class 4 cases, the patient is advised that both the non-surgical and surgical approach will be necessary. Dental implants have become popular, and unfortunately, have led to a greater percentage of patients choosing options one and two.[8] However, there are still enough patients 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 nonsurgically, or surgically. The granulation tissue is removed either with curettes, or a round bur of varying sizes. During the removal of the bonelike tissue, 90 percent trichloroacetic acid (TCA) is applied with a small cotton pellet numerous times, with increasing pressures, 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 dentin, then restored with an appropriate restorative material. Endodontic treatment is performed when indicated. The basic aim of treatment is to eliminate all active resorbing tissue and restoration of the defect so the tooth can be maintained as long as possible.[4] It has been the author's experience that all cases of Class 2–4 required endodontic treatment.

The author wishes to make something very clear. In the following cases, the 90 percent TCA was not used. There was absolutely no disagreement about the use of TCA, but at the time the following 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 percent solution of ferric sulfate with sulfuric acid, was used. It had been used for many years as a coagulant while performing apical microsurgery. The use of MS to achieve coagulation necrosis when treating ICR over the years appeared to work just fine. As a result, the use of use of MS was continued.

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

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

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 the lesion is not internal resorption and is positioned to the lingual. The protective predentin layer surrounding the pulp is clearly visible.

Fig. 8a

Fig. 8b

Fig. 8c

Fig 9b
10/12/00

Fig. 9a
5/18/00

Fig. 9c
2/14/01
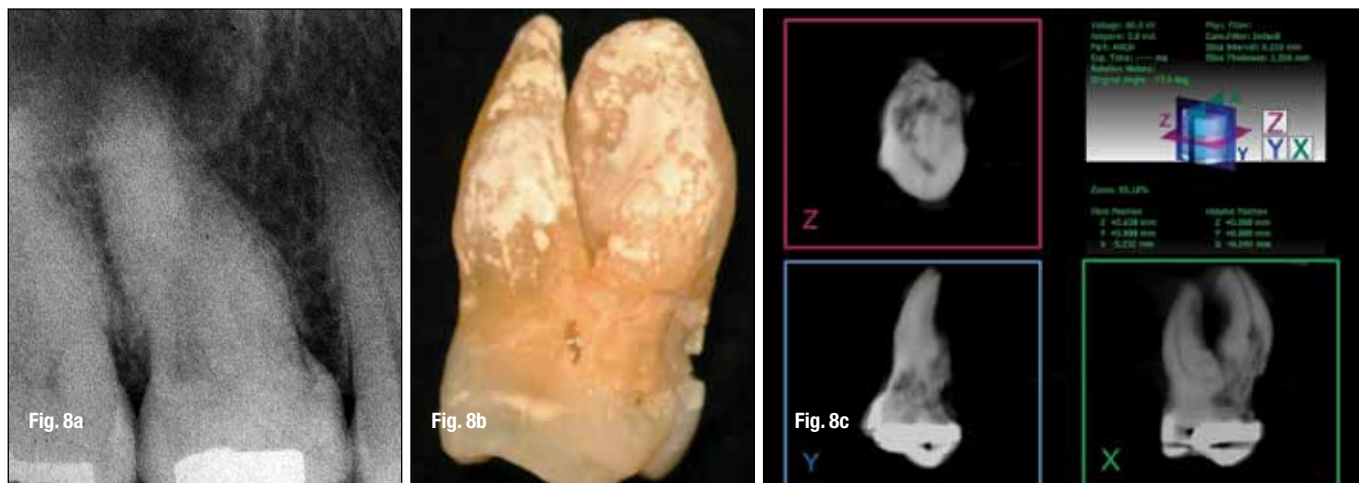
Figs. 8a,b_Pre-op PAX for extraction tooth #3 (a). The extracted tooth (b).

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

Figs. 9a–c_In a matter of just a few months, the lesion has advanced (a). The diagnosis was made (b). The patient was seen again in five months and was scheduled for treatment (c).

_First patient

In 1993, a 62-year-old male presented for an evaluation of tooth #9 (Fig.10a). His general dentist had recommended the tooth 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, "no-matter-what!" Sensing the sincerity of the patient, we agreed to attempt the salvation of the tooth, with "no guarantees." At that time, there were some practicing endodontists participating in clinical research for Dr. Torabinijad using MTA in pulp capping, perforations and root-end fills.[14] The author had used MTA on just a few patients previously and had some confidence it might serve as a "last hope" in this case. The MTA was an easy material to work with and required moisture to reach a complete set. After the access was made, the obvious hemorrhaging was difficult to control whenever more granulation tissue was removed using curettes and a #6 round bur. Ferric subsulfate (Monsel's Solution) was repeatedly used for hemostasis, then irrigated with 2.6 percent sodium hypochlorite solution (NaCl) and rinsed with sterile water, then gently air dried using the Stropko Irrigator (DCI, Newberg, Ore., USA). Hemostasis was achieved and vision was maintained while using an Aus-Jena (East Germany) surgical operating microscope (SOM) fitted with a co-observer tube for the assistant. After shaping to a size 80 Kerr file at the terminus, and removing as much granulation tissue

as possible, the canal system and defect was again copiously irrigated and dried as well as possible. Due 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 a significant amount of excess MTA was extruded (Fig.10b). The patient was dismissed and reported no post-op problems. At the seven-month follow-up visit (FUV), the tooth #9 was totally asymptomatic, but the author 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 made and access to the area was achieved. To minimize the vibration that would be created when trimming the excess MTA from the root surface, a high speed surgical handpiece with fiber-optics (Impact Air 45 Star Dental, Lancaster, Pa., USA), fitted with a surgical length taper fissure #1171 bur (SS White) was used.

After a satisfactory root profile was 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. When the necessary "troughing" was 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 FUV were scheduled. A radiograph taken at the 44-month FUV was diagnosed as "healing complete with an intact PDL" (Fig. 10e, arrow.) About four years later, the patient returned with a three-unit fixed bridge replacing tooth #9. The patient stated that "about a year ago the tooth got real wobbly" and it was removed. The pre-op extraction radiograph was located (Fig. 10f). However, later comparison of the 44-month FUV to the pre-extrac-
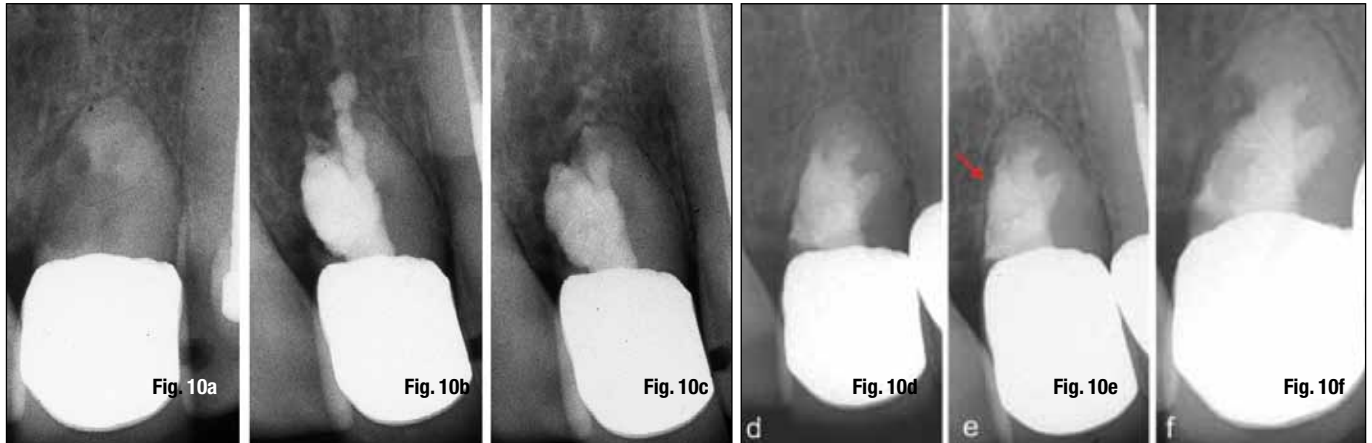
Fig. 10a Fig. 10b Fig. 10c Fig. 10d Fig. 10e Fig. 10f

tion radiograph indicates a possible continuation of the resorptive process (arrow). Isn't it amazing what you can see when the light is just right?
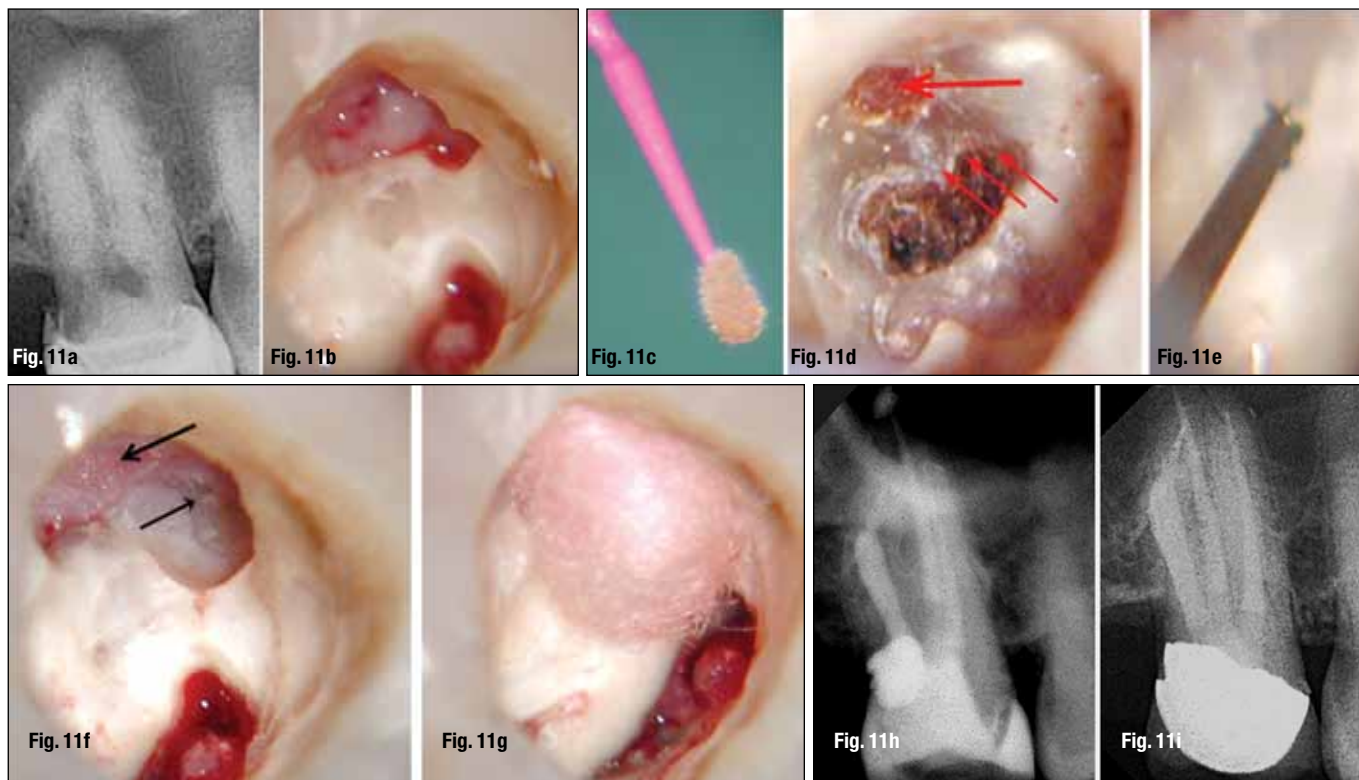
_Second patient

A 64-year-old male presented for evaluation of tooth #2 because of the unusual appearance on the distal of the tooth. The previous clinical examinations and radiographs over the past 10 months had previously been diagnosed as ICR (Figs. 9a–c). An updated radiograph was taken, all options were explained to the patient, and endodontic treatment was initiated (Fig. 9a). The tooth was accessed and using curettage, a gross removal of fibrous granulation tissue was achieved. The chamber was copiously irrigated with NaCl, rinsed and dried gently. The ICR dentinal defect and granulation tissue were evaluated to get a better concept of its position in relation to the distal wall of the access and to the pulp tissue (Fig. 9b). A small micro brush dipped in Monsel's Solution (MS) is applied to the involved area (Fig. 9c). The MS is used for coagulation necrosis and to display the affected dentin that needs removal. It is not necessary to use copious amounts when applying the MS (Fig. 11d). Instead it is best to rely more on a sequence of repeated brushing with MS, irrigation with NaCl, rinsing and gentle drying. Study with varying powers of the SOM for involved dentin. Then, if necessary, remove more of the affected dentin using varying sizes of Munce burs (CJM Engineering, Santa Barbara, Calif.) (Fig. 11e). This process is repeated as necessary to achieve adequate vision. The floor of the access should be observed, using varying powers of the microscope, to see if any affected dentin remains. A celluloid strip was placed in the distal sulcus to act as a barrier for the flowable glass ionomer restoration (Fig. 11f). An epinephrine-soaked cotton pellet (EpiDry by Pascal) was also used to maintain hemostasis and enable the attempt of a nonsurgical 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 was completed. The pulp tissue was extirpated and canal system partially shaped. Enough 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 distal buccal canal and had to be retrieved. At the final visit, the canal system was obturated using the 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 demonstrates complete healing (Fig. 11i).

_Third patient

A 47-year-old male 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 a Stropko Irrigator. The floor of the access was observed using varying powers of the microscope to identify any remaining affected dentin, which was efficiently removed with various Munce burs. Then calcium hydroxide (CaOH) was sealed in with a bonded composite as a temporary restoration. Two weeks later, the patient returned for completion of the nonsurgical 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 (Dent-

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

Figs. 10d–f_Immediate post-op following microsurgical recontouring and enhancement of previous MTA fill (d). Forty-four months FUV, indicated an intact PDL (red arrow) (e). Eighty-one months, pre-op radiograph for extraction of tooth (f). When comparing (f) with (e), there now appears to be a probability of the continuation of the resorptive process.

Fig. 11a

Fig. 11b

Fig. 11c

Fig. 11d

Fig. 11e

Fig. 11f

Fig. 11g

Fig. 11h

Fig. 11i

Figs. 11a,b_ The updated pre-op radiograph indicated an advanced class 2 ICR (a). After removal of granulation tissue, the involved dentin is seen (b).

Figs. 11c–f_A micro-brush is used to apply Monsel's solution and brushed into the floor of the defect (c). After application of the MS, the area is irrigated with NaCl, rinsed, and dried for inspection of remaining affected dentin (d). Note the perforation in the distal of the tooth (large arrow), and remaining involved dentil (small arrows) (f).

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 (large arrow) (f). A smaller Munce bur will be necessary to remove slightly more of involved dentin (small arrow). The epinephrine-soaked pellet was placed for an adequate time for hemostasis 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).

sply Tulsa). 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 was used 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 Gerestore (DenMat) (Fig.12b). Healing was uneventful, and the "numbing" sensation beside the patient's nose did resolve. The radiograph at the four-year FUV showed uneventful healing (Fig. 12c).

_Fourth patient

This 64-year-old male was referred by his general dentist because of the "unusual radiographic appearance" of tooth #27 (Fig. 13a). Even though there were no symptoms present, the referring doctor was concerned about the integrity of the tooth. Routine off-angled periapical radiographs were taken. The distal off-angled radiograph clearly indicated the lesion was on the lingual of the tooth (Fig. 13b). Both radiographs clearly showed the thin predentin-dentinal wall protecting the pulp. The past dental history revealed that the patient had complete orthodontics during his early teens. He said that tooth #27 "came out of alignment" about 20 years ago. To correct the misalignment, the doctor "sliced some off of each side of the tooth" and repositioned it with a removable appliance. Clinical examination was essentially within normal limits, except 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 invasive cervical resorption. All things were explained to the patient, and we agreed to be as conservative as possible during treatment. At the time, the author 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 bonelike hemorrhagic 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 path lab for the following definitive diagnosis:

Microscopic description: *Histologic examination reveals multiple pieces of soft and hard tissue composed chiefly of inflamed granulation and fibrous connective tissues with bone and tooth structure. The fibrous and granulation tissues consist of interlacing bundles of dense to more delicate collagen fibers 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.*
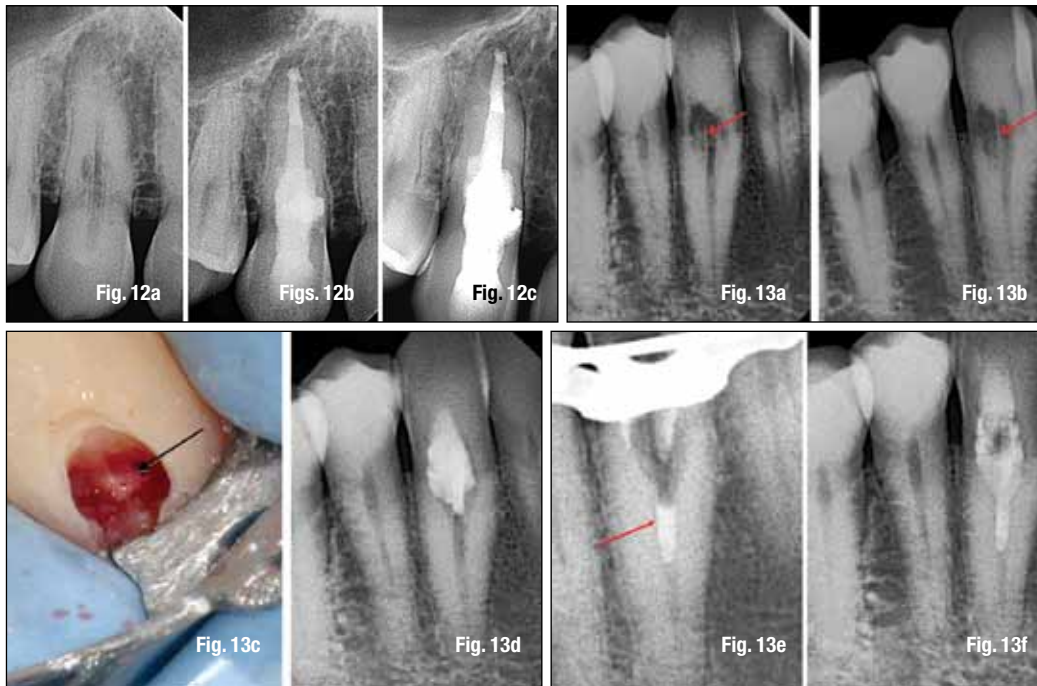
Figs. 12a–c_Initial radiograph clearly demonstrates the appearance of the protective layer and the typical mottled radiolucent appearance of the resorptive lesion (a). Immediate post-operative radiograph after nonsurgical obturation and surgical repair of the lesion (b). Normal healing is apparent in the four-year FUV radiograph (c).

Figs. 13a,b_At the initial visit, the radiograph for tooth #27, on a 64-year-old male, was taken at a normal angle (a). Taking a radiograph from a more distal angle demonstrated the lesion is located to the lingual of the tooth (b). Both radiographs clearly show the protective dentinal wall surrounding the pulp (arrows).

Figs. 13c,d_The initial access demonstrated the unusual texture of the fibrous bonelike granulation tissue in the coronal area (arrow) (c). After the removal of most of the granulation tissue, white MTA was placed into the chamber and a temporary placed (d).
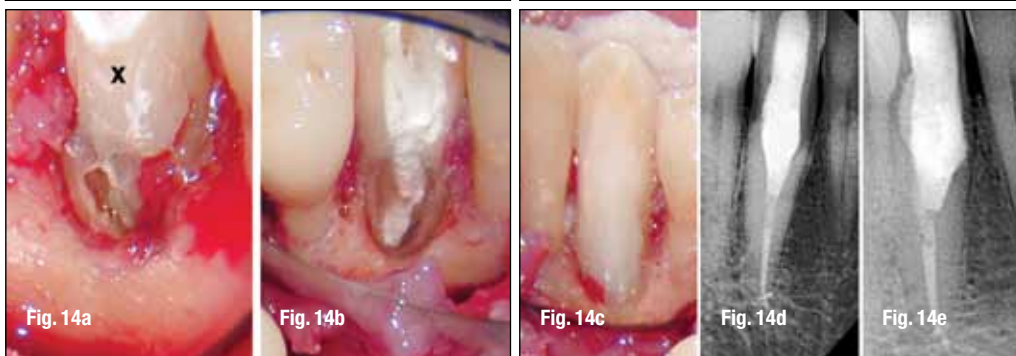
Figs. 13e,f_At the second visit, in an attempt to maintain the vitality of the pulp, MTA was placed into the canal (arrow) (e). Calcium hydroxide (CaOH) was placed as an inter-appointment medication and to allow the MTA to achieve a complete set (f).

Diagnosis: *Right posterior mandible, lingual aspect of tooth No. 27, Histologic findings consistent with idiopathic external resorption.*

Gradually, as more of the tissue was removed, the bleeding noticeably decreased, but hemostasis 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 now the author was wondering if the vitality of the tooth could possibly be maintained. 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. Using varying high powers of the operating microscope (Global Surgical Corp., St. Louis, Mo., USA), as much of the remaining affected dentin as possible was removed with Munce burs, and the remaining pulp tissue was identified. After irrigation with NaCl, an additional few millimeters of the pulp were removed and a pellet of gray MTA was placed into the canal using a medium Dovgan MTA carrier (Quality Aspirators, Duncanville, Texas, USA) (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 rescheduled 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 various sizes of Munce Discovery Burs and studied under varying magnifications until no affected dentin was observed. The chamber was irrigated with NaCl, rinsed, gently dried and etched with 35 percent phosphoric acid gel (Ultra-etch by Ultradent). A bonded core was placed, and access restored with a bonded composite (Fig. 13g). The patient was placed on a two-month schedule for an FUV.

At the two month FUV, the periapical radiograph revealed 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, an adequate access was created to remove the previously placed MTA. Using Gates Glidden burs and a #4 round bur, more involved dentin 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 10 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 fiber post with a composite core was bonded in; and the access opening filled with Gerestore 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 dentin, more solution was applied

with a micro brush to achieve coagulation necrosis; and using smaller Munce burs, the stained affected dentin was removed (Fig 14b). The process was repeated until all affected dentin was removed. After the preparation was completed, a bonded Gerestore was placed (Fig. 14c). A radiograph from a normal view indicated the restoration was appropriate (Fig. 14d). A distal off-angle view was taken to be sure the post-op integrity was achieved (Fig. 14e).

The patient was followed at regular intervals. The most recent FUV was more than eight years post-op. The patient remained asymptomatic from the beginning to the end of the entire treatment process. The recent buccal view does show a slight gray shadow in the cervical half of the tooth as a result of earlier use of the MTA when trying to maintain the vitality of the pulp (Fig. 14f). Apart from normal staining, the lingual view is within normal limits and the periodontal probing is still a 5 mm (Fig. 14g). The post-op radiographs indicate complete healing with good integrity of the fill (Figs. 14h,i). Note: the excess filling material from the original obturation (Fig. 13j) has been resolved.

_Discussion

Unfortunately, invasive cervical resorption 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.

The diagnosis of ICR is made more precise with currently available radiographic technology. Digital radiographs and cone beam computerized tomography (CBCT) have set a new standard of clinical management, allowing more predictable results with less stress. The three dimensional view presented by CBCT, removes a lot of the "what-ifs" 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 gives 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 the assistant is able to "see what you see at the same time you see it" and better anticipate what is needed next.

In all cases presented, Monsel's solution (MS) was used successfully for coagulation necrosis. Based on an early report, the author used it routinely during microsurgery for crypt management.[15] As a result, when the first case of ICR presented for treatment, 90 percent TCA acid was not a familiar alternative protocol. Having never used TCA, the author can offer no comparison or comment. The original protocol for clinical management of ICR using 90 percent TCA, suggested by Heithersay in 1999, is still the most popular and well documented. There are various techniques to restore a tooth with ICR, as previously described in the literature that are different from what is presented in this article. However, the real purpose in the treatment of ICR was, and still is, to eliminate as much of the involved dentin as humanly

Fig. 14f Fig. 14g Fig. 14h Fig. 14i
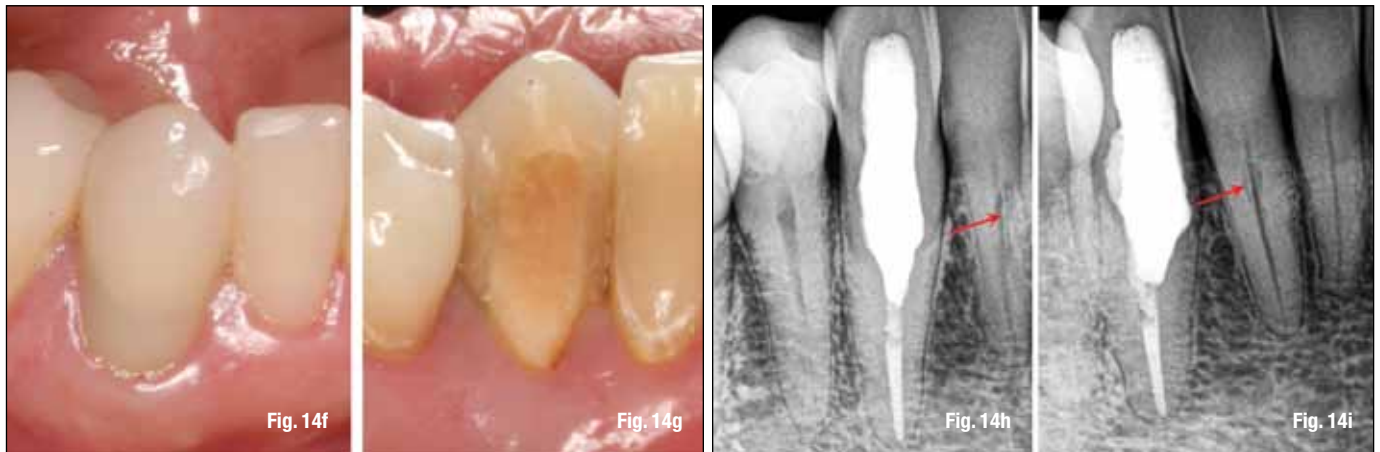
possible. If is not achieved, the process will progress and be a disaster for the tooth.

Since mineral trioxide aggregate (MTA) was extensively used in the first case presented (Figs. 10a–f.), the author does not intend to suggest the use of it as a material for the repair of ICR defects. The author 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, 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 is accepted to treat a seemingly hopeless or extremely difficult case, the opportunity to learn what can be accomplished is lost. Experience has shown that in those "basket" cases there have been more pleasant and favorable 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 still 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'!"

_Acknowledgements

Note: A complete list if references is available from the publisher.

_about the author   roots

John J. Stropko received his DDS from Indiana University in 1964. For 24 years he practiced 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, Ariz. Stropko is an internationally recognized authority on microendodontics and has performed numerous live microendodontic and microsurgical demonstrations. He has been a visiting clinical instructor at the Pacific Endodontic Research Foundation (PERF); an adjunct assistant professor at Boston University; an assistant professor of graduate clinical endodontics at Loma Linda University; a member of the endodontic faculty at the Scottsdale Center for Dentistry in Scottsdale Ariz., 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 texts, and is an internationally known speaker. Stropko and his wife, Barbara, currently reside in Prescott, Ariz. You may contact him at *docstropko@gmail.com*.

Figs. 14f,g_The eight-year FUV indicates healthy tissue on both the buccal (f), and the lingual views (g). Note the slight gray shadow left as the result of using gray MTA at the beginning of the nonsurgical treatment.

Figs. 14h, i_Radiographs taken at the normal angle (h) and taken at a mesial off-angle (i) also indicate complete healing of tooth #27. However, note the arrows pointing to a previously unnoticed ICR lesion. The various off-angles of these radiographs indicate the lesion is on the buccal surface. This should serve as a good example about the "tunnel vision" we sometimes catch ourselves doing!