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.3 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.
on the root. 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. 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. In general, an area of radicular dentine around the cervical area of the tooth may become 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 resorative 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. 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. The pulp plays no role in the aetiology of ICR and remains normal until the ICR becomes very advanced.

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 aetiologic (co)factor in ICR.

_Histology_

An interesting observation is that even in extensive lesions, the pulp is protected from the surrounding resorative 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 have been identified.
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 (∗) 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. 

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Fig. 5. Clinical classification of invasive cervical resorption. 
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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. 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. 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.

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. 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 discolouration 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-
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.\(^\text{13}\)

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.
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 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. 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 Torabinejad using mineral trioxide aggregate (MTA)
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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 (FU V), 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 FU Vs were scheduled. A radiograph taken at the 44-month FU V 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 FU V 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 curette. 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 Munce 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 Munce 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).

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
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/dental 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 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:

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 interfacing 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 dental 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 Munce 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 Munce 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. 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).

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, while 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 FL/UV radiograph demonstrated that the attempt to maintain the vitality of the tooth had not been successful (h).
The most recent FU V occurred after over eight years appropriate (Fig. 14d). A radiograph was taken from a distal off-angle view to ensure that post-operative integrity had been achieved (Fig. 14e).

After the preparation had been completed, a bonded smaller Munce burs (Fig. 14b). The process was repeated until all affected dentine had been removed.

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 5mm (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. 13j) 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.
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. 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’!”

_Acknowledgements_

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Iatrogenic errors before and after non-surgical root-canal treatment

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Several reports in the literature describe iatrogenic errors during root-canal treatment. The most common errors include perforations, ledging, transportation, zipping, overextension, file separation and underfilling. Little emphasis is placed on the preparation of a tooth before starting root-canal treatment, or on the finishing of the tooth after obturation of the root-canal system. On various online forums and in several clinical articles, beautifully executed root-canal treatments are shown with coronal restorations that are less than ideal. This is a serious problem, since it has been demonstrated that a successful outcome depends not only on adequate root-canal treatment, but also on adequate coronal restoration. In this article, I will elaborate on these aspects and present a case as an example.

As endodontists, we are specialised in the treatment of root-canal systems. However sometimes we focus on this only, forgetting that there is more to a tooth than a root. When a patient comes into our office, often he will have (a) symptomatic apical periodontitis. Whether the tooth has been treated before is somewhat irrelevant in the scope of this article. The first thing that we, as practitioners, should try to determine is the cause of the problem. The most cited causes are previous inadequate root-canal treatment, primary decay, recurring decay, worn restorations and poor restorations overall. If the tooth has not undergone root-canal treatment previously, then the cause of the problem is most likely one of the
coronal factors. It is important to address this. After all, what is the point of performing a beautiful root-canal treatment if the primary cause of the problem is not treated?

The best way to do this is by removing the old restoration completely, followed by full caries removal. This may sound logical, but it is not. There are certain disadvantages with this approach, and it is these disadvantages that guide many practitioners in their decision-making. Removing an existing restoration might result in the sacrifice of healthy tissue and it might make it more difficult to obtain proper isolation with a rubber dam. Another factor is time; removing an old restoration is time-consuming and even more so if a build-up is required before endodontic treatment. These are some reasons that many practitioners choose to leave the old restoration in place. This can compromise the treatment outcome and is a risk that can be avoided.

Fortunately, there are advantages too. By removing the old restoration and subsequently all the caries, the practitioner eliminates one of the major causes of failure and can assess immediately whether the tooth is restorable and thus avoid unnecessary treatment. Another advantage is that it is necessary to fabricate a completely new restoration afterwards, which avoids patching up of old restorations. Overall, the advantages are greater than the disadvantages and the only thing it requires from the practitioner is a change in behaviour and some perseverance.

_After root-canal treatment_

Once root-canal treatment has been completed, often we need to send the patient back to the referring dentist. In this case, an adequate temporary restoration must be placed. Typically, a temporary filling material like Cavit (3M ESPE) or a glass ionomer cement is used. A cotton pellet or some other form of space maintainer is generally placed underneath this temporary filling. This is done because the referring dentist then has easier access to the pulp chamber so that he can gain better retention when placing the permanent restoration. There are several disadvantages to this approach. Leaving space between the temporary restoration and the canal orifices puts the patient at risk of contamination. As practitioners we cannot guarantee that the patient will show up for the permanent restoration, sometimes the appointment is cancelled for a variety of reasons. Another risk is fracture of the restoration and/or tooth. If that happens the gutta-percha can be exposed to saliva, which too might lead to contamination. Ideally, however, the tooth should be restored immediately after the root-canal treatment has been carried out. This means that the endodontist places the permanent restoration.

Advantages with this approach are:

- It saves the patient a visit to his regular dentist.
- The tooth is already isolated, creating the ideal environment for a restoration.
- It saves the referring dentist time, which he can spend on other treatments.
- It offers the endodontist some variety in the treatments he performs, enabling him to broaden his skill set.

Again, this only requires a change in behaviour of the practitioner and some perseverance. It will also require that the referring dentist allow the endodontist to place the restoration. The endodontist will have to upgrade his skills, so that he can also create beautiful coronal restorations.

Following, is a case that illustrates the advantages and disadvantages of the above-mentioned approaches.
When I had just graduated as an endodontist, a 36-year-old male patient was referred because he was experiencing some mild pain in his left mandibular second molar. I was acting as a third-line practitioner in this case. Another endodontist did not wish to begin treatment and finally referred the patient to me.

The tooth was diagnosed as having symptomatic apical periodontitis and was previously treated inadequately, including a separated instrument in one of the mesial canals (Fig. 1).

In the first visit, I removed the gutta-percha from the mesiolingual canal, and cleaned and shaped it completely. The separated instrument was located in the mesiobuccal canal, but I could not remove it completely. I left the distal canal untouched. Calcium hydroxide was used as an inter-appointment dressing, and the tooth was restored with a cotton pellet and glass ionomer cement. An initial error was made by not removing the old restoration and caries completely.

One month later the patient returned in agony. When I re-opened the tooth, a great deal of pus and blood came out of the tooth. I then tried to bypass the remainder of the fragment in the mesiobuccal canal, but perforated the root with a 15.04 ProFile (DENSTPLY Maillefer; Fig. 2). I also retreated the distal canal in this session and fractured a small piece of a 25.06 ProFile in the apical part, but could bypass it. I then filled the canals again with calcium hydroxide and sealed the tooth with a glass ionomer filling.

One month later, I saw the patient again for the completion of the treatment. He no longer had any symptoms. I restored the perforation with grey MTA-Angelus (Fig. 3). I obturated the canals with gutta-percha and Topseal (DENSTPLY Maillefer) using warm vertical condensation. I sealed the cavity with Fuji IX A1 (GC) immediately on top of the gutta-percha (Fig. 4). I then referred the patient back to the dentist for a permanent restoration, with the explicit advice to have the distal restoration replaced too.

Nine months later the patient returned to my office for another tooth. I decided to take a follow-up radiograph of the left mandibular second molar to see if healing was favourable. The patient had not experienced any complaints since I completed the treatment and the radiograph showed a favourable apical outcome. However, the permanent restoration was less than ideal (Fig. 5). I had to refer the patient back to the dentist for a new restoration.

**Conclusion**

Looking back upon this case, I can conclude that I should have removed the old restoration and the caries at the start of the treatment. Positively, it was good that the glass ionomer filling was placed immediately above the canal orifices, preventing contamination via a leaky restoration. Ideally, I should have finished the restoration myself.

It required a change in my behaviour and some perseverance to begin to perform cases in accordance with the afore-mentioned approaches, as can be seen in Figures 6, 7 and 8.