

# Internal inflammatory root resorption: the unknown resorption of the tooth

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Internal inflammatory root resorption is a relatively rare resorption that begins in the root canal and destroys surrounding dental hard tissues. Odontoclastic multinuclear cells are responsible for the resorption, which can grow to perforate the root if untreated. The initiating factor in internal root resorption is thought to be trauma or chronic pulpal inflammation, but other etiological factors have also been suggested. Active, expanding resorption requires vital pulp tissue and continuous microbiological irritation, likely from the necrotic coronal part of the root canal. In its classical form, internal root resorption is easy to diagnose. However, in many instances advanced diagnostic methods may be required for a definitive diagnosis. Internal root resorption is usually symptom free, but in cases of perforation, a sinus tract usually forms. The prognosis for treatment of small lesions of internal root resorption is very good. If, however, the tooth structure is greatly weakened and perforation has occurred, the prognosis is poor and tooth extraction must be considered. Sodium hypochlorite, ultrasonic instrumentation and calcium hydroxide are the cornerstones of treatment of internal inflammatory root resorption. Mineral trioxide aggregate is being increasingly used as a root canal filling material, particularly in cases of perforation.

## Introduction

Internal (inflammatory) root resorption can be characterized both as a well-known and poorly known disease entity destroying the dental hard tissue (1–3). It is well known in the sense that most dentists recognize the diagnosis ‘internal resorption.’ However, internal inflammatory root resorption is rare, its etiology and pathogenesis are only partially understood, and there is considerable confusion between internal and cervical invasive resorption, which is often incorrectly diagnosed as internal resorption. Unlike cervical resorption and external inflammatory root resorption, internal inflammatory root resorption will stop ‘by itself’ if the whole root canal pulp tissue becomes necrotic because of advancing root canal infection. When correctly diagnosed, the treatment of internal inflammatory root resorption is relatively simple, with good or even excellent prognosis. However, in cases where the

resorption has perforated the root, the tooth structure may have become too weak, and elimination of infection can also be more difficult.

## Physiological resorption

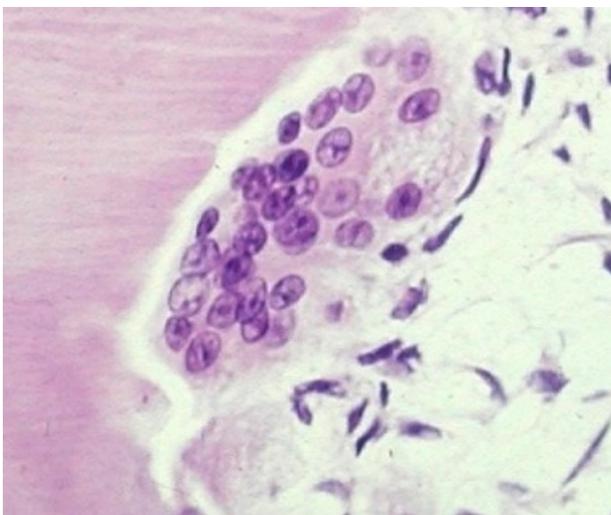
Resorption is an important part of a multitude of physiological and pathological processes in the human body. Resorption can affect hard tissues such as bone and dental hard tissues (4), but it can also involve soft tissue and foreign material such as necrotic pulp tissue or materials used in pulp capping or root filling extruded through the apical foramen (5–7). A well-known example of physiological hard tissue resorption is resorption of bone by osteoclastic activity, known as bone turnover. Parathyroid hormone (PTH), secreted by the parathyroid glands, increases the amount of calcium in the blood by various methods, one of which

involves release of calcium from bones (8). Pathological overproduction of PTH, hyperparathyroidism, will result in imbalance in the physiological bone resorption – apposition cycle, and can cause radiolucent hyperparathyroidism lesions in the jaws [see, e.g. (9–12)]. PTH and PTH-related protein (PTHrP) induce spontaneous osteoclast formation and are required for tooth eruption (13, 14).

Resorption of primary teeth is another relatively well-characterized example of physiological resorption (15, 16). Pressure from the permanent teeth is the driving force for resorption of the roots of primary teeth. A complex network of events on a cellular level including several activating and inhibiting cytokines and other compounds is required to direct the resorption to the primary tooth and the bone while protecting the permanent developing tooth from resorption. It is important to note that there is no infectious (microbiological) component in the various types of physiological resorption.

## Cells resorbing hard tissues

Osteoclasts are multinuclear cells responsible for resorption of bone, while odontoclasts are corresponding cells resorbing dental hard tissues (17–19). The multinuclear cells are formed by fusion of mononuclear cells (Fig. 1). Microscopic studies of odontoclasts using three-dimensional reconstruction have shown that



**Fig. 1.** A histological specimen showing an odontoclast cell with a high number of nuclei next to a resorbed dentin surface. Hematoxylin eosin staining. Courtesy of Dr. P.-L. Lukinmaa.

several mononuclear odontoclast precursor cells may undergo fusion simultaneously with each other and with multinuclear cells (20). Mononuclear odontoclasts can also actively resorb dental hard tissue, although during progressive resorption most cells have several nuclei (20). Actively resorbing mononuclear osteoclasts have also been reported (21). Domon et al. (22) showed that in deciduous teeth undergoing resorption, the mean number of nuclei per odontoclast was 5.3, and only 2.9% of the resorbing cells were mononuclear. Comparative studies on cell ultrastructure have shown that odontoclasts resorbing dentin or cementum are similar to those resorbing enamel (23). Close similarity to bone osteoclasts was also documented. A study of key enzymes in the resorptive process, acid phosphatase, cathepsin K, and matrix metalloproteinase-9 in osteoclasts and odontoclasts during physiological root resorption in human deciduous teeth found that there were no differences in the expression of these molecules between the two cells (18). Based on available knowledge about osteoclasts and odontoclasts, there does not seem to be any difference between these cells other than their site of action in the body; they share a common mechanism in cellular resorption of bone and teeth (18).

## Regulation of osteoclast and odontoclast activity

Osteoclasts do not have a receptor for direct binding of PTH, therefore the stimulation of osteoclasts by PTH is indirect. PTH or PTHrP bind to osteoblasts, bone forming cells, which increases their expression of ‘Receptor Activator of Nuclear Factor  $\kappa$  B Ligand,’ (RANKL), which can bind to the RANK receptor of osteoclast precursor cells, the latter become active osteoclasts through cell fusion (14). Osteoprotegerin (OPG) is a glycoprotein that is a secreted member of the tumor necrosis factor (TNF) receptor superfamily and has a variety of biological functions including the regulation of bone turnover. OPG is a potent inhibitor of osteoclastic bone resorption by competitively inhibiting the association of the OPG ligand with the RANK receptor on osteoclasts and osteoclast precursors (24–26). Fukushima et al. (27) reported that periodontal ligament cells express RANKL during physiological root resorption of primary teeth but decrease OPG expression. Expression of RANKL participates

in odontoclastogenesis and activates physiological root resorption.

It has recently been shown that TNF- $\alpha$  contributes to the development of osteoclasts and multinuclear cells with dentin resorbing capacity. Komine et al. (25) demonstrated that human TNF- $\alpha$  markedly stimulated the formation of mononuclear preosteoclast-like cells (POC) in the presence of conditioned medium of osteoblastic cells. TNF- $\alpha$  also aided differentiation of hematopoietic progenitor cells into POCs. The POC induced by human TNF- $\alpha$  formed multinuclear cells, which showed dentine-resorbing activity after co-culture with primary osteoblasts. Extremely low levels of TNF- $\alpha$  increased the level of mRNA for calcitonin receptor and cathepsin-K of the POC (25). The effects of both RANKL and TNF- $\alpha$  on osteoclast development are inhibited by OPG.

Studies on the role of macrophage colony stimulating factor (M-CSF) in osteoclast activation have been to some extent contradictory. It has been postulated that M-CSF is not involved in the activation of osteoclasts by osteoblasts (28). However, in another study it was shown that M-CSF and soluble RANKL produced by osteoblasts were essential for osteoclast precursor cell formation and for osteoclast formation (29). Interestingly, fibroblast growth factor 2 (FGF-2) has been shown to have a dualistic effect on osteoclast differentiation/activation (30). In the co-culture system of osteoblasts and bone marrow cells, FGF-2 stimulated osteoclast formation from the latter. The effect of FGF-2 on osteoclast formation is inhibited by OPG and, interestingly, also by cyclo-oxygenase 2 (COX-2) inhibitor, which indicates that FGF-2 stimulates osteoclasts partially by prostaglandin production. However, FGF-2 also exhibits a direct inhibitory action on osteoclast precursors by counteracting M-CSF signaling (30). Prostaglandin E2 upregulates the production of RANKL messenger RNA in osteoblasts, thereby stimulating osteoclast activation (28).

A number of other substances have also been shown to regulate osteoclast activity. Activators/stimulators of multinuclear resorbing cells include PTH, PTHrP, interleukin (IL)-1, IL-6 and IL-11, platelet-derived growth factor (PDGF), 1,25 hydroxy vitamin D3, glucocorticoids and substance P (31–43), while calcitonin, estrogen, interferon, IL-4, IL-8, IL-10, IL-18 and corticosteroids are involved in the inhibition of osteoclast/odontoclast cells (44–51).

Inflammation caused by a microbial infection is regarded as a major factor in several types of progressive resorptions. However, the role of individual microorganisms has been poorly studied. Choi et al. (52) reported that bacterial sonicate from three potential periodontal pathogens, *Treponema denticola*, *Treponema socranskii* and *Porphyromonas gingivalis*, induced osteoclast formation in the co-culture system of mouse calvaria-derived osteoblasts and bone marrow cells. The sonicates increased the expression of RANKL and prostaglandin E2, and decreased the expression of OPG in osteoblasts. The addition of OPG completely suppressed the osteoclastogenesis that was stimulated by the bacterial sonicates.

Increased RANKL production and stimulation of osteoclastogenesis have also recently been demonstrated by incubating PDL cells with whole cells of *Prevotella intermedia* or lipopolysaccharide from *P. nigrescens* (53, 54). The above bacterial genera (*Treponema*, *Porphyromonas* and *Prevotella*) have differences in their surface antigens (including lipopolysaccharide structure, LPS); therefore it can be speculated that a wide variety of bacteria have the capacity to stimulate differentiation and activation of multinuclear resorbing cells. Increased RANKL production has also been shown with stimulation by a Gram-positive bacterial species, *Streptococcus pyogenes* (55, 56). Interestingly, it was recently shown that surface associated (capsular) material from *Staphylococcus aureus*, another Gram-positive species, stimulates osteoclast differentiation by a RANKL-independent mechanism (57).

Nair et al. (58) reported that cell surface-associated proteins (SAP) from *S. aureus* are potent stimulators of bone resorption. They demonstrated that SAP are powerful stimulators of bone resorption in the murine calvarial bone resorption assay. Their results indicated that bone resorption was due to proteins and not the result of contamination with lipoteichoic acid or muramyl dipeptide from the peptidoglycan. The *S. aureus* SAP effect was completely blocked by high concentrations of a neutralizing monoclonal antibody to murine TNF. The role in periapical bone resorption of LPS from Gram-negative bacteria isolated from endodontic infections has been shown (59, 60). However, many endodontic infections with a periapical lesion are dominated by Gram-positive bacteria and in some only Gram-positive species are found. Therefore, it is possible that they possess antigenic structures

which can stimulate hard tissue resorption by mechanisms similar to LPS. Lipoteichoic acid, a biologically active cell surface component found exclusively on Gram-positive bacteria, has been shown to cause alveolar bone resorption (61, 62).

The cellular activation mechanisms in internal inflammatory root resorption are not known. However, a constitutive expression of OPG, RANKL, and M-CSF mRNA has been reported in mouse odontoblast and pulp cell lines (63). Interestingly, in a co-culture system, the dental cells were inhibitory to osteoclast formation from spleen and bone marrow precursors, despite their production of osteoclast stimulatory factors RANKL and M-CSF. The authors suggested that activation of resorbing cells is dependent on a balance between the stimulating and inhibitory factors (OPG) (63). Corresponding studies on periodontal ligament and gingival fibroblasts have also indicated the presence of inhibitory mechanisms that limit osteoclast differentiation or their resorbing ability (64). Cell culture supernatants have revealed low levels of RANKL (osteoclast activator) and high levels of OPG (inhibitor). The addition of M-CSF and RANKL to the co-culture resulted in increased resorbing activity by the multinuclear cells, indicating that lack of resorption by these cells in co-culture with periodontal and gingival fibroblasts was caused by inhibition rather than lack of resorbing potential of the multinuclear cells.

Much of the present knowledge of dentin resorption is from studies of resorbing roots of shedding primary teeth. Multinuclear odontoclast cells involved in internal inflammatory root resorption are thought to be identical to bone resorbing osteoclasts. Several mediators produced by a number of cells, also present in the pulp, can participate in the development of multinuclear cells from precursors and stimulation/inhibition of dentin resorption. Key events in the initiation of internal inflammatory root resorption, however, are not yet fully known.

## Etiology and pathogenesis of internal inflammatory resorption

The rare occurrence of internal root resorption is probably the main reason for its etiology being poorly understood. It is generally assumed that damage to the organic sheath, predentin and odontoblast cells covering mineralized dentine inside the root canal must

occur to expose the mineralized tissue to pulpal cells with resorbing potential. However, it is not known with certainty what kind of trauma or other event may be required to produce the damage needed to initiate resorption. The predisposing factors to internal root resorption as suggested in the literature include trauma, pulpitis, pulpotomy, cracked tooth, tooth transplantation, restorative procedures, invagination, orthodontic treatment and even a Herpes zoster viral infection (2, 65–67).

In an interesting study of the etiology and pathogenesis of internal inflammatory root resorption, Wedenberg & Lindskog (68) stimulated development of internal resorption lesions in monkey teeth, which were then extracted after varying observation periods and examined using a variety of microscopic and radiographic methods. The root canals of monkey incisors with vital pulps were accessed and injected with Freund's complete adjuvant (a non-specific stimulator of the immune response) and either sealed aseptically or left open to the oral cavity. The authors reported that colonization of the dentin wall by macrophage-like cells was observed in both experimental groups. Interestingly, the colonization was transient in the sealed teeth with no pulpal infection, whereas the unsealed teeth whose pulps became infected by the oral bacteria demonstrated a more extensive and prolonged colonization of the dentin surface by the resorbing cells. Spreading of the macrophages/multinuclear giant cells could be seen only in areas where dentin was denuded. According to the study, this occurred either by mineralization of predentin in the affected area or by degeneration of the odontoblasts and predentin layer. In the sealed teeth with no pulpal infection, the number of macrophage-like cells declined 6–10 weeks after the initiation of the experiment and an increase in the number of fibroblast-like cells as well as hard tissue barrier formation were observed. However, in the open teeth with infected coronal pulps, no reduction in the number of cells with resorbing potential could be detected. Brown and Brenn staining of the histological sections showed that most of the dentinal tubules in the area were invaded by bacteria of different morphotypes (68). The authors concluded that for internal root resorption to occur, mineralized dentin must be exposed. Moreover, they suggested that internal root resorption can be divided into two different types: *transient resorption* and *progressive internal root resorption*. The first one would thus be analogous with transient

external surface resorption of the root and is self-limiting while the latter is stimulated by the presence of bacteria and will continue to expand.

Sahara et al. (23) studied the resorption by odontoclasts of a superficial non-mineralized layer of predentin prior to the shedding of human deciduous teeth by light and electron microscopy. They found multinucleate cells on the predentin surface of the coronal dentine between the degenerated odontoblasts and resorption lacunae in the non-mineralized predentin. In some other areas in the same study, the multinuclear cells 'simultaneously resorbed both non-mineralized and calcospherite-mineralized matrix in the predentin.' The authors suggested that multinucleate odontoclasts can resorb the non-mineralized predentin matrix *in vivo*, probably in the same way that they resorb the demineralized organic matrix in the resorption zone underlying their ruffled border (23). In another study of shedding primary teeth, it was found that as long as the roots were actively resorbed by odontoclasts, the pulpal tissue did not undergo any major structural changes. When root resorption was nearly finished, increased numbers of inflammatory cells were detected in the pulp (17). At this stage, odontoblasts began to degenerate, multinucleate odontoclasts appeared on the dentin surface, and resorption proceeded from the predentin to the dentin. The odontoclastic activity was first demonstrated at the cervical area of the crown pulp, but eventually the resorption of dentin spread coronally toward the pulp horns.

The uncommon occurrence of dentin resorption can be explained by the dominance of osteoclast/odontoclast inhibitory substances such as OPG over activators such as RANKL. However, it has also been suggested that dentin contains a non-collagenous compound/component which may function as a resorption inhibitor in dentin. Wedenberg & Lindskog (69) studied the ability of stimulated and unstimulated peritoneal macrophages to spread *in vitro* on different inorganic and organic components of dental tissues in order to establish morphologic evidence of a presence of a resorption inhibitor in dentin. Macrophages attached and spread on enamel, dentin, and collagen-coated coverslips; however, the same cells attached but did not spread when incubated on predentin or demineralized dentin. The authors concluded that the resistance to resorption of predentin and dentin may be caused by an organic, non-collagenous component of the tissue. Subsequent experiments indicated that when

demineralized dentin and predentin were treated with guanidium hydrochloride, macrophage spreading could readily be detected (70). In another series of experiments, osteoclasts were isolated from neonatal rats and seeded onto pieces of fully mineralized dentin, demineralized dentin, and predentin with or without prior extraction with guanidium hydrochloride. Osteoclasts (odontoclasts) colonized and resorbed fully mineralized dentin, whereas clastic cells were not observed on unextracted demineralized dentin and predentin. However, after guanidium hydrochloride extraction, osteoclasts also attached and spread on demineralized dentin and predentin (71).

Damage to the cells of the odontoblastic layer may occur not just as a consequence of trauma but also because of inflammation as a reaction of the pulp connective tissue to infection approaching either through dentin (caries) or from the more coronal pulp. Odontoblast cell death is frequently seen in areas of microabscesses in the peripheral pulp tissue in teeth with a deep caries lesion. The fact that internal inflammatory root resorptions are also found in the middle and apical parts of the roots of mandibular premolars and molars (Fig. 2), which are well protected against trauma, may be regarded as an indication that pulpal inflammation can initiate internal inflammatory



**Fig. 2.** Internal inflammatory root resorption with an asymmetric shape in the lower second mandibular premolar.

root resorption. This could occur if odontoblasts are killed at the advancing front of inflammation but the pulp still retains vitality in the area. Cells with resorbing capability may become activated and gain contact with exposed predentin/dentin. As mentioned earlier in this article, several bacterial species have been shown to increase RANKL expression and osteoclast activation (52–54).

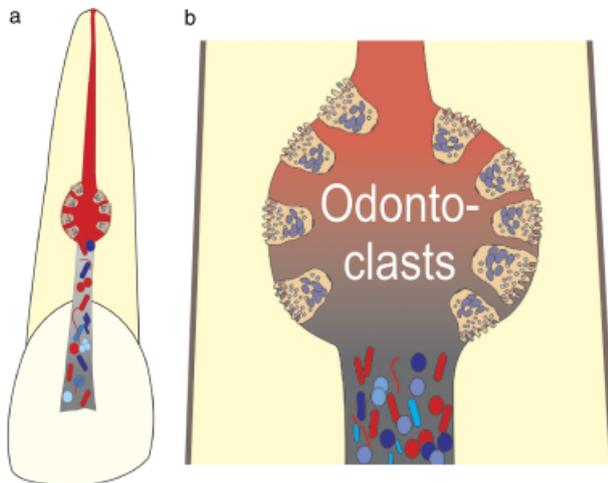
It may be of interest that internal inflammatory root resorption in its most classical form spreads symmetrically in all directions into the dentin surrounding the pulp. The initiation of internal root resorption throughout the full circle of dentin at certain depths in the coronal-apical direction can perhaps be explained either by a slowly advancing pulpitis (inflammation) or by mechanical trauma. However, the fact remains that internal inflammatory root resorption is rare and the amount of information on it is scarce. Thus, the key events in the initiation of internal inflammatory root resorption remain largely unknown.

Irrespective of the possible initiating factor (trauma, inflammation or some other reason), there is a general agreement that the progress of internal root resorption is dependent on two things: the pulp tissue at the resorption area must be vital, and the pulp coronal to the resorption must be partially or completely necrotic, allowing bacterial infection and microbial antigens to enter the root canal (Fig. 3). Microbial stimulus is necessary for the continuation of internal inflammatory

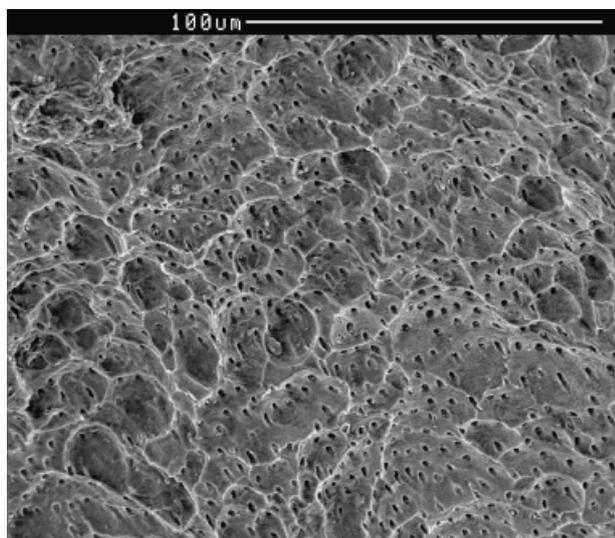
root resorption. Otherwise, limited internal surface resorption might be the only consequence of the initial phase. The microbiology of internal inflammatory root resorption has not been studied. However, as several recent studies have shown that both Gram-negative and Gram-positive bacteria as well as spirochetes have the potential to stimulate RANKL expression and osteoclast activation, it can be speculated that there may not be specific species which are more likely than others to be involved in the initiation and pathogenesis of internal inflammatory root resorption.

### Histological features of internal inflammatory resorption

Reports of the histological and microanatomical features of internal tooth resorption are based on examination of extracted teeth with ‘naturally occurring’ internal resorptions and on experimentally induced resorptions in animal models (68, 72, 73). Allen & Gutmann (72) described highly vascularized pulpal connective tissue infiltrated by lymphocytes and plasma cells as well as ‘resorptive bays.’ Wedenberg & Zetterqvist (73) examined 13 primary and permanent teeth extracted because of internal resorption. The authors reported that the progress of the resorption was faster in primary teeth, but that there were no other differences between the two groups of teeth. The pulp tissue next to the resorption showed hyperemia and varying degrees of inflammation and infiltration of lymphocytes, macrophages, and neutrophilic leukocytes. Bacteria were detected histologically only in the teeth undergoing rapidly progressing resorption. The bacteria were located either in the dentinal tubules or in the necrotic part of the coronal root canal. Interestingly, the authors reported osteoid or cementum-like tissue in some areas of the pulpal wall as well as small calcifications in the pulp tissue. Odontoblast cells were not detected in the resorption area in any of the teeth, and predentin was also absent in most areas. Neutrophils and macrophages were seen attached to the mineralized dentin surface, and numerous odontoclastic cells (see Fig. 1) were present in resorption lacunae (73). Scanning electron microscopy (SEM) of the samples revealed large odontoclastic cells, ca. 50  $\mu\text{m}$  in size and with a ruffled border directed against the dentin surface. Figure 4 shows an area of dentin resorption by odontoclastic cells.



**Fig. 3.** (a) A schematic drawing showing the pathogenesis of internal inflammatory root resorption. The canal coronal to the resorption is necrotic and invaded by microbes. The resorption cavity contains highly vascularized resorption tissue with multinuclear odontoclast cells. (b) A close-up image of the resorption.



**Fig. 4.** A scanning electron micrograph of the dentin surface treated with hypochlorite (to remove organic debris) after resorption. The micrograph shows resorbed dentin with numerous shallow resorption lacunae, which indicate the site of odontoclast activity. The small openings are dentinal tubules.

A histological variation of the common type of internal root resorption has also been suggested in the literature. Ne et al. (74) described root canal replacement resorption (metaplastic resorption) which involved resorption of dentin around the root canal and subsequent deposition of osteoid or cementoid hard tissue. The authors reported that root canal replacement resorption can occur where there is chronic inflammation next to an area where damage to odontoblast cells and predentin has exposed the mineralized dentin. However, it may be difficult to detect the difference between revascularization and subsequent formation of osteoid tissue from root canal replacement resorption.

### **Epidemiology: prevalence of internal inflammatory resorption**

Epidemiological data on internal inflammatory root resorption is scarce or completely lacking. Internal resorption is so uncommon that it is difficult to gather reliable data about its prevalence. It is also unknown whether there is any geographical, age- or sex-related differences in the occurrence of internal root resorption. In a study of ca. 1000 teeth, Thoma (75) reported internal root resorption in only one tooth. Cabrini et al.

(76) reported internal root resorption in eight out of 28 teeth (28%) where pulpotomy in the coronal pulp and capping with calcium hydroxide (covered by zinc oxide eugenol) had been performed. The teeth were extracted 49–320 days after the endodontic procedures and subjected to a histological study. Ahlberg et al. (77) reported a long-term evaluation of autotransplanted maxillary canines with an average follow-up time of 6 years. Of the 33 teeth included in the study, 17 (55%) developed internal resorption. Interestingly, no internal resorption was detected during the first year after autotransplantation, and the majority of resorptions appeared 3 years or more after the procedure. The teeth were endodontically treated after the resorption was diagnosed.

Many cases that have been referred to endodontic specialists or university clinics as internal root resorptions have turned out to be external cervical resorptions. It should also be kept in mind that the diagnosis of internal root resorption is mainly based on radiographs, which means that a considerable amount of root canal wall dentin must be resorbed to be reliably detected in the radiograph. In cases where the root canal infection proceeds rapidly through the root canal, resulting in necrosis of the whole pulp, the resorption (if present) stops at an early stage and would remain undetected both clinically and radiographically.

Based on the limited studies and clinical experience of the authors, we suggest a prevalence of between 0.01% and 1% (patients affected) for internal inflammatory root resorption. Although this estimate is quite rough and may be wrong, it is unlikely that it will be replaced in the near future with a more correct figure that would be based on an epidemiological study. Typically, only one tooth per patient is affected by internal root resorption. However, occasionally two adjacent teeth have internal root resorption, with trauma being the likely initiating factor in such cases.

### **Clinical and radiological features of internal inflammatory resorption**

The clinical characteristics of internal root resorption are dependent on the development and location of the resorption. Most teeth with internal root resorption are symptom-free. However, when the resorption is actively progressing, the tooth is at least partially vital and may present symptoms typical of pulpitis. If the

resorption occurs in or near the crown, it may in advanced cases show as a pinkish or reddish color through the crown if only a thin layer of enamel is left to cover the resorption (Figs 5–7). The red color is caused by the highly vascularized connective tissue adjacent to the resorbing cells. In internal resorption, the color is typically located centrally, whereas in cervical resorption the color ('pink spot') may also be mesially or distally located (Fig. 8). Teeth with untreated internal



**Fig. 5.** Pink/dark red spot in the middle of the cervical area of the crown of a maxillary central incisor with internal inflammatory root canal resorption in the crown area.



**Fig. 7.** A photograph of a lower first right molar with internal resorption which has perforated the crown in the distobuccal area at the cemento-enamel border. Highly vascularized granulation tissue can be seen through the perforation. A pink color through the enamel can be seen in a large area coronally and laterally to the perforation.



**Fig. 8.** A mesial pink spot in the cervical area of the crown of a maxillary canine with cervical resorption. Courtesy of Dr. S. Heistein.



**Fig. 6.** (a) A patient with large internal inflammatory root resorptions in both maxillary central incisors. Tooth #11 has a pink discoloration indicating that the pulp is vital, whereas tooth #21 has a dark discoloration, an indication of pulp necrosis. (b) The teeth seen from the palatal side. The strong discolorations are clearly visible. (c) A radiograph of the teeth reveals wide destruction of dentin and enamel caused by internal inflammatory root resorption. Courtesy of Dr. M. Ree.

resorptions in the coronal area often turn gray/dark gray if the pulp becomes necrotic.

Internal inflammatory root resorptions continue to expand until either endodontic treatment is started or the pulp becomes necrotic. Eventually the resorption will perforate the root unless it is stopped by one of the above-mentioned events (Figs 7 and 9–11). Perforation of the root is usually followed by the development of a sinus tract, which confirms the presence of an infection in the root canal. After the perforation, the continuation of the resorption may no longer be dependent on the presence of vital pulp tissue because

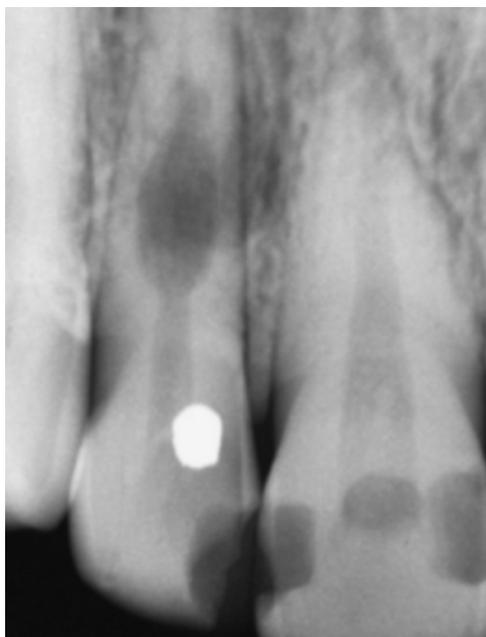


Fig. 9. A maxillary lateral incisor with internal inflammatory root resorption, which has perforated mesially in mid root. Inflammatory changes in the bone can be seen at the site of the perforation.



Fig. 10. A maxillary lateral incisor with internal inflammatory root resorption. A fistulograph taken with a gutta-percha point inserted into the sinus tract indicates a perforation on the buccal surface of the middle third of the root.

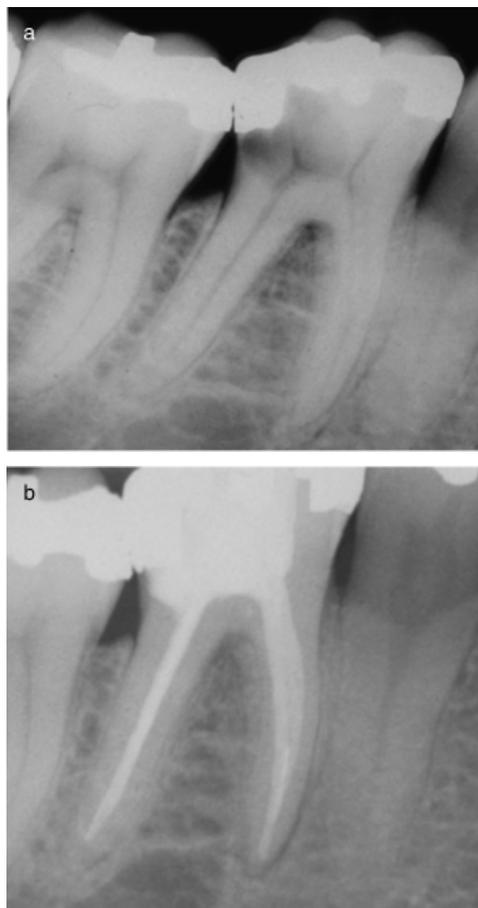


Fig. 11. (a) A radiograph of internal inflammatory resorption in the coronal pulp area (tooth from Fig. 7). The resorption is affecting a relatively large area around one pulp horn while the other areas remain unaffected. (b) The same tooth after root canal treatment and restoration of the resorption area.

the resorbing cells may now obtain nutrients from tissues surrounding the tooth. After perforation, the control of infection is more difficult than in an unperforated root canal. The tooth structure is also weaker in teeth with perforation as a result of loss of hard tissue. In addition to the sinus tract, swelling may be present, while most patients complain of only mild or no pain.

With advancing infection, the entire pulp becomes necrotic and internal root resorption ceases because the resorbing cells are cut off from the circulation and nutrients, unless root perforation has occurred before the development of total necrosis. Pulpal necrosis can therefore be regarded as an effective protection against spreading of the resorption. The consequence of pulp necrosis is, as usual, apical periodontitis. There is no data indicating that the (previous) resorption has any

effect on the pathogenesis or symptoms of apical periodontitis.

In its most classical appearance, internal inflammatory root resorption is relatively easy to identify radiographically and the correct diagnosis can be made. The resorption is seen as a radiolucent, round and symmetrical widening of the root canal space (Fig. 10). At the area of the resorption, the original canal shape can no longer be observed. However, not all internal root resorptions show similar progression, and oval as well as asymmetrically shaped internal root resorptions can be found (see Figs 2 and 9). In the coronal pulp/crown area, internal resorption can be symmetrical in teeth with one root canal and a narrow pulp chamber where pulp horns are situated close to each other. However, in molar teeth with several roots and a wide pulp chamber, internal resorption may begin at one part of the chamber and spread locally into the surrounding dentin (Fig. 11). In such cases, it may be difficult to make the diagnosis between internal and external cervical resorption until the resorptive area is accessed directly, cleaned and carefully studied under a surgical microscope during endodontic treatment. However, cervical resorptions in the crown area often have a more irregular outline and contain randomly shaped thin opaque lines which are not seen in lesions of internal resorption (Fig. 12).

### Diagnosis and differential diagnosis of internal inflammatory resorption

Internal inflammatory root resorption is usually first detected radiographically. Many lesions are found accidentally during routine check-up radiographs, as teeth with internal resorption are typically symptom-free. As indicated earlier, diagnosis of symmetrical, round or oval lesions in the root canal can easily be done. For more irregularly shaped resorptions, the key diagnostic feature is the disappearance of the original canal shape in the area of the resorption.

The diagnostic challenge with internal root resorption is external cervical resorption when it projects over the root canal on a radiograph. Cervical root resorption is known for its inability to penetrate into the root canal with vital pulp tissue. Micro computed tomography (CT) scans of teeth with cervical resorption show a zone of 0.1–0.3 mm dentin which separates the (external) cervical resorption from the pulp; this zone

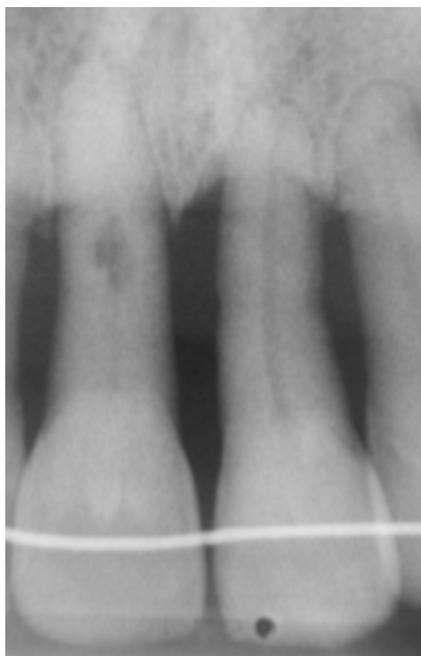


**Fig. 12.** Cervical resorption spread all over the cervical area of the tooth, including parts of the crown and the most coronal part of the root. Comparison with Fig. 11 where internal resorption is seen in same area shows that the cervical resorption has a more mosaic-like internal structure and the opaque lines separating the process from the pulp are prominent.

being ca. 10–20 times wider than the thickness of the predentin layer (78–80). The thin layer of dentin thus includes enough mineralized tissue to make it visible on the radiographs as an opaque line next to the root canal. This opaque line is a reliable differential diagnostic sign of cervical root resorption (Figs 11 and 12). If the cervical resorption projects on top of the root canal, the original shape of the canal can in many cases be seen through the resorption (Fig. 13).

Internal and cervical resorption can also both occur in the crown area. Although the point of invasion on the root surface of cervical resorption can sometimes be difficult or impossible to detect on the radiograph, the presence or absence of the opaque lines surrounding the pulp is still a useful indicator to differentiate between the two types of resorption (Figs 11 and 12).

Recent evolution of radiographic techniques has begun to have an impact on the diagnosis of tooth resorptions, including internal root resorption. Assessment of the resorptive lesions by three-dimensional imaging using various modifications of the CT techniques will greatly facilitate differential diagnosis and help to determine the location, dimensions, spreading, and possible site(s) of perforation in much



**Fig. 13.** A cervical resorption lesion in the middle third of the root of a maxillary central incisor resembles internal resorption. However, the shadow of the root canal can be seen, although weakly, through the resorption. The resorption is located at the marginal bone level, which is typical of cervical resorption. The diagnosis of cervical resorption was verified during a flap operation.



**Fig. 14.** A computed tomography scan of a vital lower first molar with cervical resorption. The resorption can be seen distally to the pulp chamber at the level of marginal bone. The limitations of the resolution make it difficult to determine whether the resorption has perforated into the pulp.

greater detail than has been previously possible (Figs 14 and 15) (81–85). Reduced radiation dosage, improved resolution, and better tolerance of disturbances caused by metal structures such as posts and metal filling materials already help to make better recommendations and decisions of optimal treatment choices and to minimize the loss of dental hard tissue.

Cervical or root surface caries seldom create a diagnostic problem even in cases where the radiolucent carious lesion projects on top of the root canal. Figure 16 shows two molars, one with a cervical resorption and the other with a caries lesion.

Color changes that are clinically visible are present only in a minority of cases of internal and cervical resorptions. The color change related to internal resorption can be pink, red, dark red, gray or even dark gray depending on the size of the resorption and the vitality status of the pulp. As soon as the coronal pulp becomes necrotic, it is likely that the original pink/reddish color will gradually change to a dark red/dark gray (Figs 5–7). Cervical resorption is independent of the pulp. Therefore, it is less likely that a pink

spot which is detected in the crown area in some cases of cervical resorption will turn dark as in internal resorption. Another characteristic that may be an indication of the origin of the resorption is the location of the spot: a color change from internal inflammatory resorption is typically seen in the middle of the tooth in the mesio-distal direction (except in multirrooted teeth), whereas a color change from the cervical resorption can be located mesially, centrally, or distally (Figs 5–8).

Clinical examination by probing may be useful in the differential diagnosis between root surface caries and cervical resorption: the former is practically always accessible by probing while cervical resorption is usually not because it starts apical to the junctional epithelium. However, in cases of wide-spreading cervical resorption (types 3 and 4), one can sometimes probe into the resorptive lesion when the patient is anesthetized. Even in these situations, the caries lesion usually feels softer (sticky) while the resorption feels more like normal dentin. Only in extremely rare cases can internal inflammatory root resorption be clinically probed. A prerequisite for this is that the resorption has

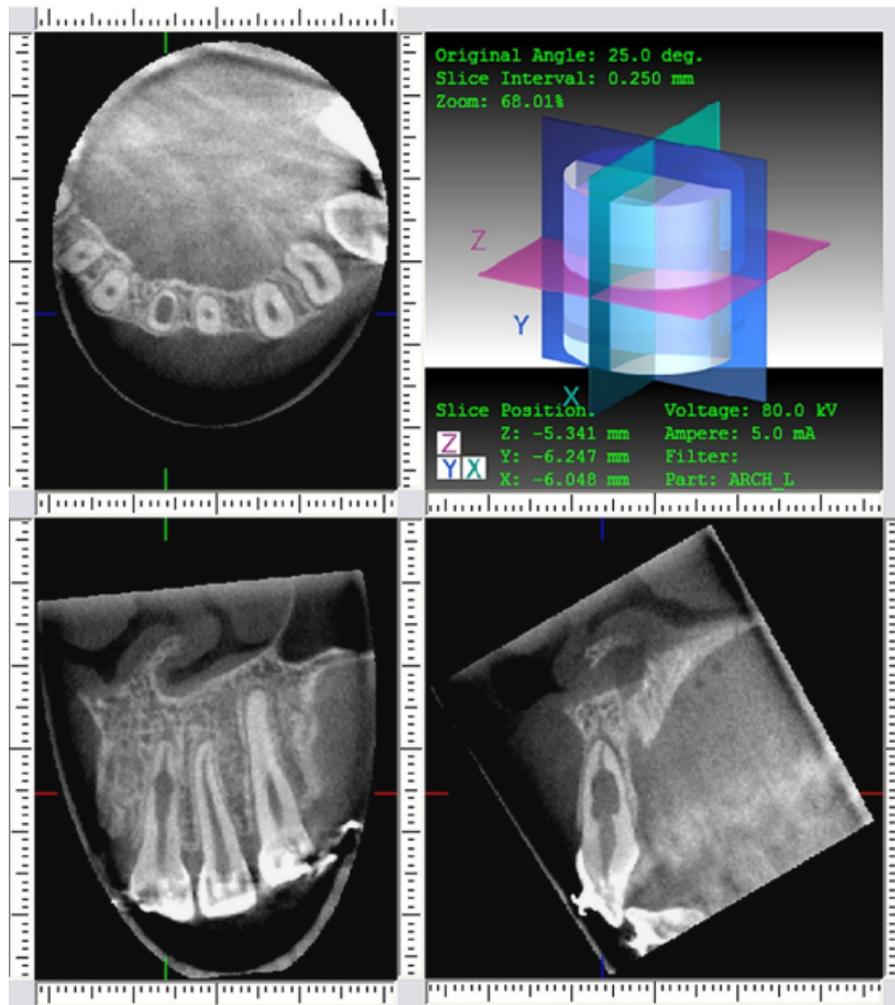


Fig. 15. A cone-beam volumetric tomography (CBVT) image of an internal inflammatory root resorption in a maxillary central incisor. The technique allows observation of the dimensions of the lesion in axial, sagittal, and coronal planes. Courtesy of Drs. T. Cotton, T. Geisler, D. Holden, S. Schwartz, and W. Schindler. (Another section of the same tooth was originally published in *J Endod* 2007; 33: 1121–1132.)

perforated the root or the crown at the level of or coronally to the marginal bone. In such situations, the probe will easily penetrate deep into the resorption because of the typical shape and type of spreading of internal resorptions, whereas in cervical resorption the depth of probe penetration is quite limited.

## Treatment of internal inflammatory resorption

### Instrumentation of teeth with internal resorption

Instrumentation and cleaning of the root canal space of teeth with internal resorption faces a few challenges

different from those of normal endodontic treatment. In cases where the resorption is active, there is typically brisk bleeding from the pulp tissue, which may make it difficult to locate the root canal openings. However, as soon as the apical pulp tissue has been cut off and removed using large enough instruments in the apical canal, the bleeding stops or is greatly reduced, allowing better visibility into the work area. Irrigation by concentrated sodium hypochlorite will in most cases help to reduce the bleeding. Sometimes it is preferable to pack calcium hydroxide into the pulp chamber and the canal(s) and seal the tooth with a temporary filling. A few days later, bleeding of the soft tissue is no longer a problem because calcium hydroxide effectively necrotizes the granulation tissue. For teeth where the resorption has perforated the root, placement of



**Fig. 16.** A radiograph of two molars, one with a cervical resorption and one with a caries lesion. Adhering to the diagnostic criteria of each disease, these two conditions can usually be reliably identified as not being internal inflammatory root resorption.

calcium hydroxide is recommended to necrotize the resorptive tissue and to stop the bleeding.

Although the majority of the literature on internal inflammatory root resorption is case reports, there is no generally accepted protocol for the chemomechanical instrumentation of the root canal system in these cases. However, it is obvious that a great emphasis must be placed on the chemical dissolution of the vital and necrotic pulp tissue. Therefore, irrigation with sodium hypochlorite is an important part of the treatment of teeth with internal resorption. Small perforations do not seem to require abandonment of the use of hypochlorite; on the contrary, hypochlorite will help to control bleeding from the perforation and disinfect and clean the area as experienced with accidental perforation complications. However, with large perforations, low-concentration hypochlorite solutions should be used and other irrigants such as chlorhexidine should be considered.

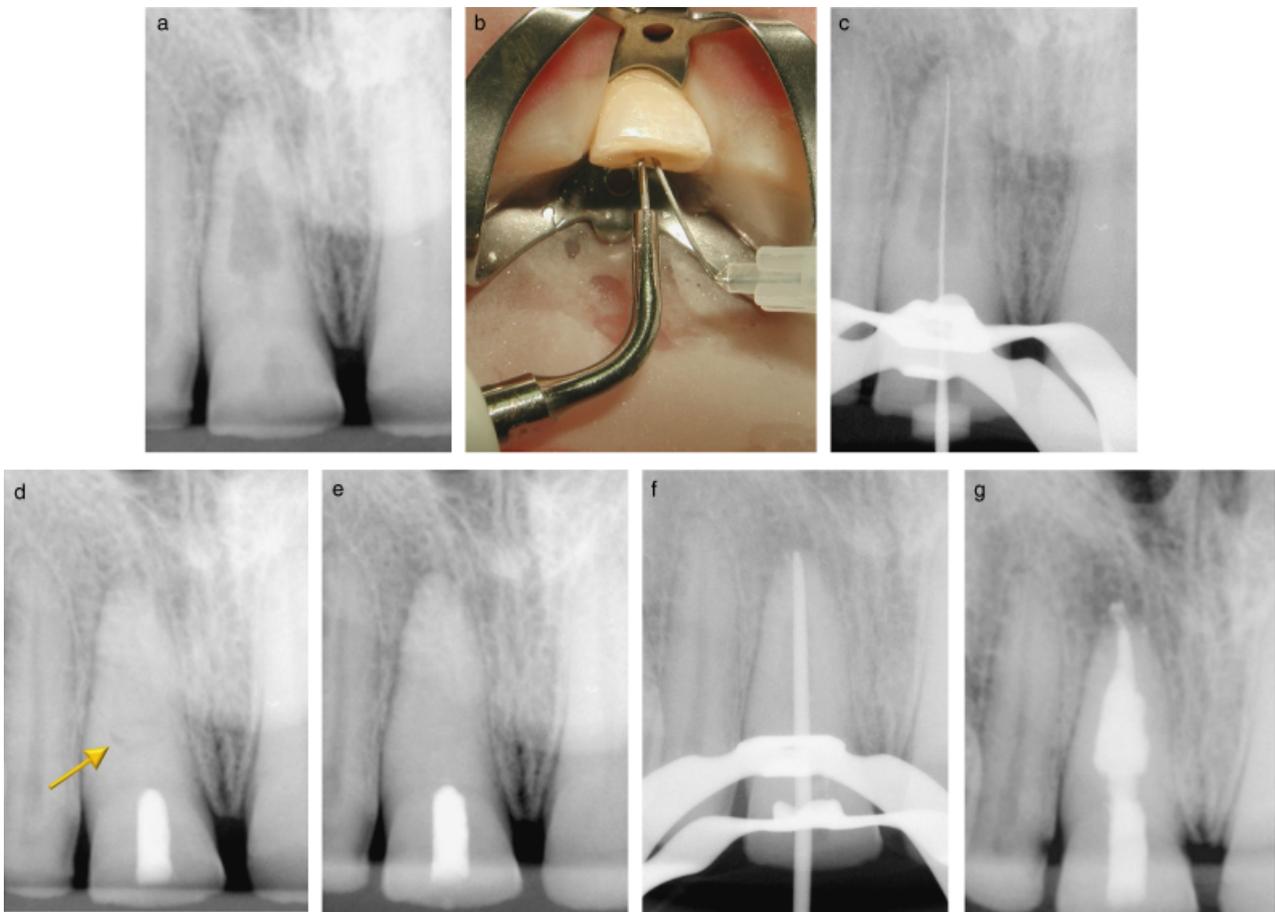
The shape of a resorbed root canal prevents instrument access to all areas of the canal. Creating a straight line access to the resorption cannot be done in many cases because it would weaken the tooth structure too much. This is one reason why the use of ultrasound has been advocated for the treatment of internal resorptions (86). Ultrasound can facilitate the penetration of an irrigant to all areas of the root canal system and break loose necrotic tissue in the canal (Fig. 17). In order to better reach the most distant areas of the resorption, hand instruments are often bent at 1–4 mm from the tip

to help to gain contact with the walls of the resorption cavity and help to remove all soft tissue. Although use of hypochlorite and ultrasound are mainly responsible for cleaning of the most challenging areas, the importance of careful mechanical cleaning should not be underestimated.

### **Use of intracanal medicament in the treatment of internal inflammatory resorption**

Intracanal interappointment medicaments are used in endodontic treatments mainly to maximize the effect of disinfection procedures (87). In the treatment of internal resorption, the use of calcium hydroxide also has two other important goals: to control bleeding, and to necrotize residual pulp tissue and to make the necrotic tissue more soluble to sodium hypochlorite (Fig. 17). Because of the limited access by instruments to all areas of the resorption cavity, chemical means are needed to completely clean the canal. Studies on the effectiveness of sodium hypochlorite and calcium hydroxide to remove the resorptive and other tissues from the root canal indicate that they have an additive or even synergistic effect (88–93). In cases where the resorption has not perforated, it is usually enough to use calcium hydroxide paste in the canal once from 1 to 2 weeks. This allows removal of the residual tissue at the next appointment by irrigation and instrumentation. Ultrasound is recommended both to facilitate tissue removal and for cleaning the canal from all calcium hydroxide before permanent root filling.

In perforated internal resorptions, calcium hydroxide treatment has been carried out for extended time periods for up to 1 year to secure complete healing of the site of perforation (2, 94). There are no comparative studies on the long-term prognosis of perforated internal resorptions treated with either short-term or long-term calcium hydroxide treatment. However, it is possible that the new material, mineral trioxide aggregate (MTA), could change the recommended treatment protocol for internal resorption. A number of recent studies, including two meta-analyses, have shown that MTA is superior to formocresol in pulpotomies of primary molars (95–97). A notable difference between the two materials was the absence of resorption complications following treatment in the MTA groups. The excellent performance of MTA



**Fig. 17.** (a) Internal inflammatory root resorption in the middle root of a patient with dentinogenesis imperfecta. The root canal is necrotic and a small apical lesion can be seen in the radiograph. (b) Simultaneous use of ultrasound and irrigation with sodium hypochlorite in a tooth with internal inflammatory root resorption. (c) Working length radiograph of the tooth. (d) Calcium hydroxide was packed into the root canal and resorption. The arrow points to an area which was not completely filled with calcium hydroxide, possibly because of residual tissue left in the resorption. (e) At the second appointment, chemomechanical instrumentation including irrigation with sodium hypochlorite and use of ultrasound were repeated. Packing of calcium hydroxide no longer shows empty spaces in the resorption area. (f) A master cone radiograph of the tooth at the end of the third appointment. (g) A final radiograph showing the root canal and the internal resorption filled with gutta-percha and sealer using warm vertical condensation.

as a retrograde filling material is well recognized and filling the complete root canal of immature permanent incisors with MTA has been reported (98). Recently, filling of the internal resorption cavity with MTA in a primary molar was reported (99). Although not supported yet by long-term results from clinical studies, it is possible that the treatment of perforated internal resorptions in the future will consist of a thorough chemomechanical cleaning and disinfection of the root canal and resorption area including the perforation site, followed by a short-term calcium hydroxide treatment. At the second appointment, in the absence of any clinical symptoms, the resorption cavity will be filled with MTA.

### Permanent filling of the root canal and the internal resorption

There is no generally accepted consensus on the materials and techniques that should be given priority when teeth with internal resorptions are permanently filled. However, case reports and clinical experience indicate that root filling methods using warm gutta-percha are generally preferred over other techniques (Figs 17–20) (86, 100–103). However, in cases where the resorption has perforated, MTA should be considered instead of gutta-percha because of its antimicrobial properties and better seal. MTA is also very well tolerated by the tissues. MTA carriers,

ultrasound, inverted paper points used as pluggers, and radiographic control of the MTA filling at the early phase of condensation are all crucial factors for success and to ensure a high quality filling (Fig. 21). In teeth with a large resorption cavity in the coronal third of the root canal, use of composite materials should be considered in order to strengthen the tooth and to make it more resistant to tooth fracture (103).

## Prognosis of the treatment of internal inflammatory resorption

In light of the rare occurrence of internal inflammatory root resorption, it is not surprising that there are very few studies on the outcome and prognosis of the treatment of the resorption. Caliskan & Turkun (100) reported on the success of endodontic treatment of teeth with internal root resorption. Twenty-eight teeth

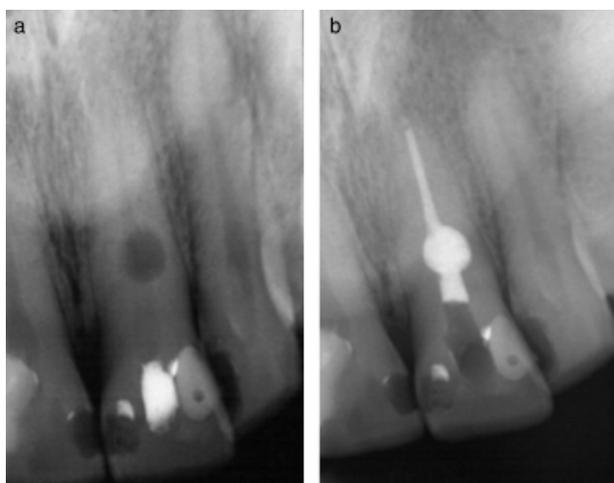


Fig. 18. (a) A maxillary central incisor with internal inflammatory root resorption. (b) The tooth after root canal treatment. Following treatment with calcium hydroxide, the canal and the resorption were filled with a warm gutta-percha technique using a MacSpadden compactor.



Fig. 19. Maxillary central incisor with internal inflammatory root resorption after treatment. Courtesy of Dr. G. Bergenholtz.

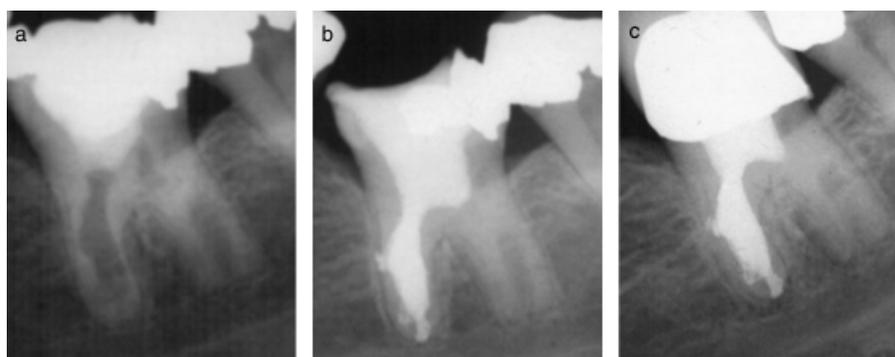
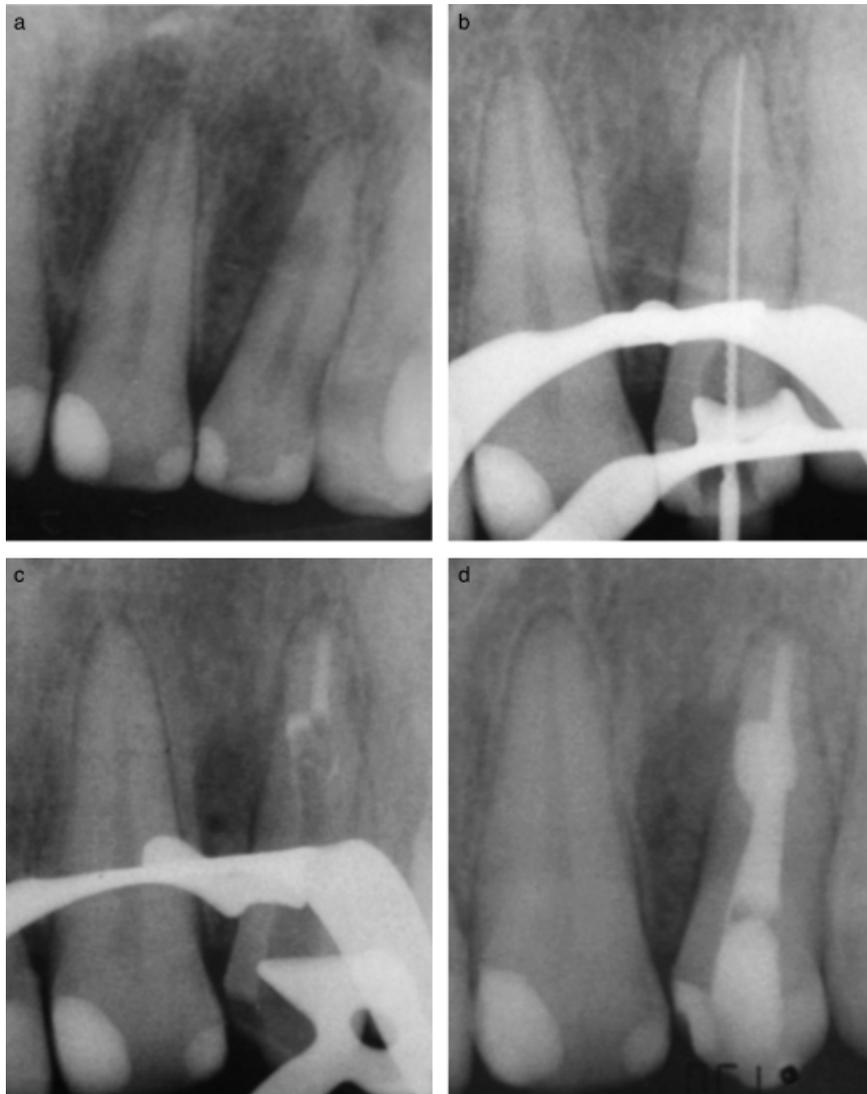


Fig. 20. (a) Mandibular molar with internal root resorption extending all the way from the coronal to the apical part of the mesial root. (b) The mesial root was cleaned, disinfected with irrigants and calcium hydroxide, and filled with warm vertical condensation. Two areas of root perforation were detected when the root was filled. The distal root canal was separated from the rest of the root canal space by a thick dentin bridge and deemed to be vital with no apparent resorption. (c) The same tooth 2 years after the endodontic treatment of the resorption was completed.



**Fig. 21.** (a) A maxillary lateral incisor with a perforated internal inflammatory root resorption. (b) Endodontic treatment commenced and the canal was filled with calcium hydroxide after chemomechanical preparation and irrigation with copious amounts of sodium hypochlorite. (c) Root filling with MTA of the most apical canal was placed at the second appointment. The quality (porosities) of the MTA filling must be controlled early with a radiograph before adding more material. (d) Finished root filling with MTA apically and in the lesion, and gutta-percha in the coronal canal. Courtesy of Dr. A. Senjabi.

were referred for treatment because of internal resorption, and 20 of them were treated by conservative endodontic treatment. Sixteen of the teeth had non-perforated internal inflammatory root resorption and responded well to the treatment. However, teeth with a perforation that were treated by long-term calcium hydroxide treatment showed a favorable result in only one of the four cases. The three failed cases were then treated surgically, resulting in success in two of the three cases. In the remaining case, the tooth was lost

because of extensive loss of marginal bone and increased tooth mobility.

Although the lack of follow-up studies on the long-term prognosis of the treatment of teeth with internal root resorption is obvious, there is a general consensus based on clinical experience and case reports that the prognosis of the treatment is fairly good or even excellent for cases which have not perforated and where the tooth has not been weakened too much by the loss of tooth structure (86, 100–103).

## Conclusion

Internal inflammatory resorption is an uncommon resorption of the tooth which starts from the root canal and destroys the surrounding tooth structure. Odontoclast cells which are responsible for the resorption are structurally and functionally similar to bone osteoclasts. The diagnosis of internal resorption is in many cases simple, but may in some situations require use of advanced diagnostic techniques such as CT scanning. The prognosis of the treatment of internal tooth resorption is good unless the tooth has been weakened too much by the resorption. With proper treatment and use of modern endodontic techniques and materials, the prognosis of even perforated cases is fairly good.

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