Endodontic infections and apical periodontitis. An analysis of microbial factors prior, during and after endodontic treatment

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Chapter 2

THE FATE AND ROLE OF BACTERIA LEFT IN ROOT DENTINAL TUBULES

Abstract. - In this article the question of the relevance, and consequences, of bacteria remaining in the tubules of root dentine after cleaning and shaping of the root canal space is addressed. A second aim is to discuss the necessity of clinical measures taken to eradicate those bacteria that are presumed to survive there. The available clinical and experimental evidence supports the use of antibacterial dressings in cases where the root canal space remains temporarily unobturated after removal of necrotic and infected pulp tissue. There is no evidence, however, that special measures should be taken to kill the bacteria in the dentinal tubules. Should time permit, a sound obturation technique immediately following the cleaning, shaping and disinfection phases, allows the remaining bacteria in the tubules to be either inactivated or prevented from repopulating the (former) canal space. In the vast majority of cases, those bacteria appear not to jeopardize the successful outcome of root canal treatment.

INTRODUCTION

The essential role of bacteria in the initiation, propagation and persistence of apical periodontitis has been established (Kakehashi et al. 1965, Möller 1966, Sundqvist 1979, Sundqvist et al. 1979). Endodontic therapy is aimed at the elimination of bacteria from the infected root canal and at the prevention of reinfection. This is accomplished by a thorough chemomechanical cleaning of the root canal followed by a complete filling of the canal space. Cleaning, shaping and irrigating greatly reduce the cultivable numbers of bacteria.

However, a number of studies have shown that it is impossible to achieve a sterile root canal space in all cases even by thorough cleaning, shaping and irrigating with disinfectants or antiseptics after one visit (Byström and Sundqvist 1981, Byström, Claesson and Sundqvist 1985). Therefore, concern exists as to the fate and consequences of microorganisms left in the canal. It has been shown that they may multiply rapidly in two to four days (to almost the original numbers) in cases where the canal is not filled or not dressed with a disinfectant between visits (Byström & Sundqvist 1981).

In clinical practice the remaining bacteria can be prevented from repopulating the root canal space by enclosing an interappointment dressing like calcium hydroxide in the canal (Byström, Claesson and Sundqvist 1985, Chong & PittFord 1992). Some authors consider therefore a two-visit root canal treatment mandatory in case of root canal infection (Heithersay 1990, Tronstad 1991). Another approach has been to allow the remaining microorganisms no nutrition or room to multiply by direct and complete filling of the prepared and disinfected canal space, as practiced in single-visit endodontics (Soltanoff 1978, Oliet 1983). In orthograde surgical endodontics, the canal is cleaned and obturated in one visit, neglecting the remaining microorganisms (Kouhry et al. 1987, Grung et al. 1990). Independent of the use and type of interappointment dressing, 70-95% success rates are claimed with two-visit endodontic treatments, as well as with single-visit endodontic treatments and with orthograde surgical endodontics (Soltanoff 1978, Oliet 1983, Pekruhn 1986, Khoury et al. 1987). The published success rates appear to be mainly determined by the criteria used for defining success, by the time between treatment and evaluation, and by the pretreatment status of the tooth and periapex (Sjögren et al. 1990).

Often, the causes of failure of root canal treatment are obvious such as: unfilled canals, incomplete root canal preparation and filling, vertical root fractures, external root resorption (Vire 1991). In addition, some failures occur even after seemingly perfect root canal treatments. It may be significant that a perfect appearance of a root canal filling on the radiograph has limited predictive value about its sealing quality (Kersten et al. 1987).
However, even when we the root canal treatment is assumal to be perfect, some cases fail and one can only speculate on the reasons for these failures, unless a peri-apical biopsy reveals a cause like a self-sustaining cyst, a cholesteatoma or a foreign body reaction. Bacteria remaining in the apical part of the root canal have been suggested as other causes of failure, despite the fact that no bacteria could be detected in the canal prior to filling and after the use of an interappointment dressing (Nair et al. 1990, Sjögren et al. 1990). Also, periradicular infection per se (Tronstad et al. 1987, Barnett et al. 1990) causes failures. Recently, concern has been expressed about microorganisms and their products remaining in the root dentinal tubules. This initiated evaluation of measures to kill bacteria left in the dentine (Safavi et al. 1990, Ørstavik and Haapasalo 1990, Horiba et al. 1990).

In this review article the question of the relevance and consequences of bacteria, or bacterial products, remaining in the tubules of root dentine is addressed. A second aim is to discuss the clinical necessity of special measures taken to eradicate them.

THE PRESENCE OF BACTERIA AND THEIR PRODUCTS IN ROOT DENTINE IN VIVO

Ando & Hoshino (1990) reported the presence of viable bacteria $\left(10^2 \text{ per mg}^{-1}\right)$ in dentine samples collected from areas at a distance of 0.5-2 mm from the canal-dentine boundary of infected teeth with decayed clinical crowns. In histologic sections of heavily infected teeth, Armitage et al. (1983) found bacteria in the dentinal tubules up till halfway the cemento-dentinal junction.

Horiba et al. (1990) reported the presence of endotoxin within the dentinal wall of infected root canals. The endotoxin content was significantly higher in samples taken from the area of 300 $\mu$m adjacent to the pulpal surface, than in samples from areas at a distance of 301 - 800 $\mu$m.

In 27% of periodontally diseased caries-free teeth, bacteria ($>100$ colony forming units/mg dentin) were found in dentine samples adjacent to the pulp; however, no mention is made about the rate of migration of microorganisms through dentine (Adriaens et al. 1988). Although this suggests a potential danger for the vital pulp, it disintegrates only if the main apical foramina are colonized by dental plaque from the periodontal process to involve the root canal (Langeland 1974). No information is available about the colonization by bacteria of dentinal tubules in periodontally diseased teeth without a vital pulp.
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External inflammatory root resorption

After traumatic injuries to the teeth the periodontal ligament is often damaged, followed by necrosis of connective tissue at the root surface. Phagocytosis then causes resorption of cementum, resulting in areas of root surface denuded from cementum (Andreasen 1981). If, in these cases, the pulp is necrotic and bacterial products from within the infected dentine reach the resorptive lacunae on the root surface through the dentinal tubules, it may sustain inflammatory resorption of the root (Tronstad 1988).

The presence of bacteria in root dentine in vitro

Numerous investigators have, for various research reasons, inoculated the pulpal side of root dentine with bacteria. These studies showed that, in vitro, bacteria penetrate into the tubules and remain viable there for periods of up to 10 days (Akpata and Blechman 1982, Haapasalo and Ørstavik 1987).

It appears that several factors influence the depth of penetration into the tubules. The presence of a smear layer prevents complete penetration, while etching of the dentine before exposure to the bacteria results in deeper penetration (Safavi et al. 1989). According to Akpata and Blechman (1982), the greater the number of bacteria and the more rapid their multiplication rate, the deeper the penetration will be. They found that extension of the exposure time of the dentine to bacteria leads to an increase in the number of infected tubules and also to an increase in penetration depth. These observations were confirmed by Ørstavik and Haapasalo (1990). In their study Enterococcus faecalis and Streptococcus sanguis penetrated the dentinal tubules within 2-3 weeks to a depth of 300-400 µm while Pseudomonas aeruginosa was only scarcely found in the scanning electron microscopic sections, even after 4 weeks of incubation. Perez et al. (1993) found a mean penetration depth of 479 µm for Streptococcus sanguis after 28 days of incubation, with an extreme value of 737 µm.

If bacteria are inoculated from the coronal part of the tooth there are more tubules infected in the cervical area of the dentine than in the middle or apical part (Akpata and Blechman 1982). Bacterial penetration from the pulpal side is deeper when the cementum is removed from the root surface (Haapasalo and Ørstavik 1987, Adriaens et al. 1988). If cementum is not present on the root surface, bacteria are able to colonize the tubules from the periodontal side. The speed of penetration from this side is lower than that from the pulpal side. It slows down when the bacteria progress towards the middle portion of the root, presumably because the nutrition...
source (pulp or periodontium) is situated further away (Haapasalo and Ørstadtevik 1987; Adriaens et al. 1988).

Conclusions from this research may be that one important factor for invasion of dentinal tubules by bacteria is the availability of a nutrient source. Ingrowth or progress of bacteria can be delayed or prevented by the presence of a smear layer and an intact root cementum and periodontal ligament (Safavi et al. 1989).

THE FATE OF BACTERIA LEFT IN DENTINAL TUBULES AFTER ROOT CANAL TREATMENT

Although, in vitro, most bacteria in the dentinal tubules died within 24 hours after removal of the nutrient medium (Ørstadtevik and Haapasalo 1990), concern has been expressed about their survival after root canal therapy since in vivo conditions may permit nutrient supply to bacteria in dentinal tubules, (Ørstadtevik and Haapasalo 1990).

Therefore, the penetration of various antiseptics into the dentinal tubules has been evaluated. According to Safavi et al. (1990), iodine potassium-iodide has “long distance” bactericidal effects in vitro and is more potent than calcium hydroxide, and Ørstadtevik and Haapasalo (1990) found it to be more potent than sodium hypochlorite. Its antimicrobial efficacy, however, has been shown to be of very short duration (Ørstadtevik and Haapasalo 1990). Its depth of disinfecting activity in the tubules reached up to 1000 μm within 5 minutes, whereas this was 300 μm for NaOCl and Hbitane (chlorhexidine). Gutierrez et al. (1990) found that, in histological sections of roots irrigated with NaOCl and hydrogen peroxide, cuboidal sodium chloride crystals were present, together with bacteria within the tubules, up to 220 μm from the main root canal. Vahdaty et al. (1993) found that Chlorhexidine and NaOCl were equally effective antibacterial agents at similar concentrations against E. faecalis present in dentinal tubules.

To prevent bacteria from repopulating the root canal after completion of the root canal instrumentation, interappointment dressings have been recommended. It is hoped that these will kill the microorganisms in the tubules. Camphorated p-monochlorophenol (CMCP) appeared to have a stronger bactericidal effect in the dentinal tubules than calcium hydroxide (Haapasalo and Ørstadtevik 1987). However, CMCP used as an interappointment dressing has a short half-life and almost 95% of the CMCP inside the pulp chamber is lost within 24 hours (Messer and Chen 1984). Recently, it has been shown that a sustained-release device containing chlorhexidine gluconate reduces the number of bacteria in the dentinal tubules and prevents their regrowth whereas calcium hydroxide does not (Cervone et al. 1990). The bactericidal effects of calcium hydroxide on microorganisms in dentinal tubules appeared less efficient than that of CMCP in vitro (Haapasalo and
Ørstavik 1987). However, Byström et al. (1985) found Calasept (calcium hydroxide) dressings to be most effective in interappointment treatment of infected root canals. Application of calcium hydroxide for 1 month resulted in negative cultures of the canal contents in 97% of the cases. The favourable effect of calcium hydroxide is due possibly to its low solubility which makes it act as a slow release-system within the root canals. By its release of hydroxyl ions it kills or inactivates bacteria. Its physical obturation of the canal space may effectively block accumulation of exudate, thereby reducing the potential of nutrient support for the bacteria and eliminating the space for bacteria to multiply.

**LONG-TERM FAILURE BECAUSE OF BACTERIA LEFT IN THE TUBULES**

It may be asked if bacteria, remaining in tubules do, in fact, lead to failure or unsuccessful repair of the periapex. This has not been investigated *in sensu stricto*. After proper cleaning, shaping and obturation of the canal system the numbers of bacteria are greatly reduced compared to initial numbers. Even if all bacteria originally present in the tubules would remain viable at the end of the clinical session, their numbers are certainly quite small, again compared with the initial number in the root canal system that caused (periapical) pathology (Byström and Sundqvist 1981).

It is conceivable that those bacteria either do not survive the treatment, are inactivated subsequently or remain in insufficient numbers to sustain or cause pathology. This is, of course, supported by the notion that a high percentage of "properly treated" cases are successful. This is clearly not due to the almost unobtainable sterility of the treated dentine-pulp complex, but rather to the low numbers of remaining bacteria just before obturation. Calcium hydroxide greatly helps reducing or inactivating bacteria (when used as an interappointment dressing). This is because of its disinfecting capacity as well as its physical obturation of the canal system, withholding bacteria from their sources of nutrients and limiting the space for multiplication.

If the latter is appropriate, obturation with gutta percha and sealer during the first appointment, after chemo-mechanical cleaning and disinfection with NaOCl, also deprives the remaining microorganisms from their nutrition and leaves them no space to multiply to sufficient numbers to cause or maintain disease.
**External inflammatory root resorption**

Removal of the irritants from the root canal by complete debridement and obturation stops the inflammatory resorptive process in most cases (Cvek 1973). However, the possibility exists that bacteria in the tubules survive the treatment and get nutrition from tissue fluid entering the tubules from the periodontal side where the tubules are patent owing to resorption. Prolonged calcium hydroxide treatment is recommended in cases of trauma to prevent resorptive processes (Camp 1983). In cases where inflammatory resorption is evident calcium hydroxide is believed to kill bacteria in the tubules and provide an alkaline environment stimulating repair processes (Tronstad 1988).

Conversely, Hammerström et al. (1986) and Lengheden et al. (1990, 1991) have warned that the use of calcium hydroxide in root canals of teeth with damaged root surfaces may result in ankylosis because of the necrotizing effect of calcium hydroxide on the repopulating cells. There appeared to be no difference in the occurrence of root resorption whether (2 weeks after pulp infection) the root canals were immediately obturated with gutta percha and sealer or a calcium hydroxide paste was applied (Dumsha and Hovland 1993). These results, together with those of Cvek (1973) suggest that bacteria in tubules, in cases of inflammatory root resorption, probably do not survive after root canal treatment, or where they do survive, their influence on repair processes is not noticeable.

**Conclusions**

- Failure of root canal therapy seems to be unrelated to the relative small numbers of bacteria left in the dentinal tubules after proper root canal preparation and obturation. Rather, partly or improperly filled canals give way for regrowth or reinfection leading to failures.
- There is not enough evidence to support the clinical application of special measures to eradicate the bacteria left in the dentinal tubules.
- Further research is necessary to give an answer to the question: do bacteria survive in dentinal tubules or not and if they survive do they grow to sufficient, pathologically significant, numbers?
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