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Is the risk of loss of pulp vitality increased by periodontitis and its treatment?



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KEY WORDS *dental pulp, endodontic disease, periodontal disease*

Anatomical links exist between the dental pulp and the periodontium and these include dentinal tubules, accessory canals and the apical foramina. The presence of these structures has led to the assumption that both periodontal disease and periodontal treatment can have a very negative effect on the dental pulp. Examination of the literature produces convincing evidence that the vital pulp is likely to be in danger only if periodontal disease is severe and extends to involve the apical tissues. Epidemiological research demonstrates that very few patients experience such severe disease and thus the effects of periodontal disease and its treatment on the dental pulp have been greatly exaggerated.

■ Introduction

It has been known for many years that there are anatomical links between the pulp and the periodontium. This has led to the persistent presumption that the mere presence of these communications must mean that disease on one side of the link is likely to spread via such communications to affect the tissues on the other side. This assumption has affected clinical practice in that dentists are taught to test the sensitivity of teeth with advanced periodontal disease, particularly if this includes furcation lesions, because of the concern that pulp health will have been compromised. It is important that this assumption is ques-

tioned by examination of the literature because if true, the logical extension would be to suggest that teeth with advanced periodontal disease could be electively root filled as a prophylactic procedure. This work would be undertaken when there was the minimum possible degree of endodontic damage that would in turn maximise the chance of root canal treatment success.

Another factor should also be considered. Morris et al¹ examined data from the most recent UK Adult Dental Health Survey, and found both that 55% of dentate adults had periodontal pockets exceeding 3.5 mm in depth, and that this percentage increased with age. If periodontal disease and periodontal



Fig 1 Branching accessory canals visible at the apex of a lower second premolar after completion of a root canal treatment.

treatment do indeed result in pulp damage it would be reasonable to expect to find many teeth that had lost vitality for which the main or only identifiable cause would be periodontal involvement.

This commentary explores the evidence linking both periodontal disease and periodontal treatment with pulp damage.

■ Communication between the periodontium and the pulp

There are three main anatomical communications between the pulp and the periodontal tissues: the dentinal tubules, lateral and accessory canals and the apical foramina. Literature on the potential role played by each of these in providing communication should be considered.

■ Dentinal tubules

Dentinal tubules are formed during tooth development by odontoblasts, which move in a pulpal direction while secreting dentine matrix. Root dentinal tubules thus run in a relatively straight course from the pulp to the external root surface, with the large opening being situated at the pulpal surface of the dentine and the smallest at the external root surface.

The root is normally covered in cementum, which means that the tubules are effectively sealed off from the periodontium. The cementum layer, however, is not always present. It may be simply absent in areas

with developmental grooves and may be lost in sites that have suffered periodontal disease or have undergone periodontal treatment. Experimental work using scanning electron microscopy showed that 18% of teeth overall, and 25% of anterior teeth in particular, display dentine exposure at the cemento-enamel junction². This figure is surprisingly high, and if lack of cement does predispose to pulpal damage one would expect to find loss of vitality in many teeth that could be attributed to this cause. However, there is no evidence to suggest that this is the case.

Dentinal tubules range from 1–3 µm in diameter³ but, as described by Michelich et al⁴, they contain the odontoblastic process, collagenous fibres and the sheet-like lamina limitans. The presence of these structures reduces the effective tubule radius to 5–40% of the anatomical radius, which means that there is less space in tubules than may be imagined. It is also known that the tubules contain a liquid similar to extra-cellular fluid⁵ and that the passage of organisms or toxins through the tubules from the outer root surface inwards toward the pulp would have to overcome the outward fluid flow. It is reasonable to suppose that this may be significant in reducing ingress of harmful organisms and products that could damage the pulp.

■ Accessory canals

It has been suggested that accessory canals are produced during tooth development because of the presence of persistent blood vessels during the apical growth of Hertwig's root sheath⁶. The prevalence of accessory canals has been reported to be between 23% and 76%⁷⁻⁹ and they may be present anywhere on the root, although they are most commonly situated in the apical third (Fig 1)¹⁰. Many authors have described the presence of accessory canals linking the pulp chamber and/or the main root canals to the furcation area of multi-rooted teeth (Figs 2a and 2b)^{6,7,11-15}. However, many different experiments, employing different techniques, have produced varying results. Interestingly, Vertucci and Anthony¹⁵ carried out a study using scanning electron microscopy and found that there was a high incidence of such openings on the periodontal surface of molar furcation areas, while Perlich et al¹⁴, using similar technology, found many fewer such openings on the floor



Fig 2a Accessory canal in the furcation area of an upper second molar (original magnification 6x).



Fig 2b Accessory canal in the furcation area of an upper second molar (original magnification 25x).



Fig 2c The apical foramen is visible on both the mesial and distal roots of a lower second molar (original magnification 20x).

of the pulp chamber. One suggested explanation for this finding is that the majority of these 'canals' are to be found only in the cementum layer covering the furcation and do not extend into the dentine proper and, in addition, they may not contain blood vessels but only connective tissue. The presence of patent canals running from the pulp chamber into the furcation area has been found to occur in as few as only 10% of all molars¹² and this again has significance for the assumed role of these structures in providing a pathway linking the pulp and the periodontium.

■ The apical foramen

Rotstein and Simon¹⁶ described the apical foramen as 'the principal and most direct route of communication between the pulp and periodontium', and it is logical to assume that such a communication pathway can work in both directions. Fig 3 is a photograph of apical foramina. It is certainly true that material from the pulp, including live bacteria and their toxic products, may escape from the apical foramen and evoke an inflammatory response in the adjacent periodontium. However, a classical histological study by Langeland et al¹⁷, which examined the effect of varying degrees of periodontal disease on the pulps of 60 extracted teeth, convincingly demonstrated that 'the pulp did not succumb as long as the main canal – the major pathway of circulation – was not involved. Pulpal ... disintegration apparently occurs only when all main apical foramina are involved by bacterial plaque.' This means that only very severe

periodontal destruction, which is in itself relatively unusual, and which extends to the apical regions of the teeth, could potentially secondarily affect the pulp. This finding has great clinical significance.

■ The role of bacteria

Periodontitis is generally accepted to be induced by a mixed anaerobic bacterial flora whose effects in terms of disease induction and progression are essentially determined by the host inflammatory and immune response mounted to counteract them^{18,19}. Research has demonstrated that many bacterial species isolated from endodontic infections may also be recovered from periodontal lesions^{20,21}. Somewhat confusingly, there is evidence to suggest that the flora associated with periodontal lesions is more complex²², while other work has found no significant differences²³. However, given the many and varied inherent problems associated with bacterial sampling and culture it is not unexpected that differing protocols and techniques produce differing results. Equally, similarities in bacterial populations from both endodontic and periodontal sources are not surprising given that the bacterial source for both pathologies is the same, namely the resident oral flora. It should also be appreciated that anaerobic conditions in both niches will favour colonisation by bacterial species able to thrive in both these environments. This means that the presence of such organisms in these locations does not necessarily imply participation in causation of the conditions, but



Fig 3a Radiograph showing an upper first molar tooth with severe bone loss and suggestion of furcation involvement. Clinical examination revealed this tooth to have 'through and through' furcation involvement from buccal, mesial and distal aspects yet the tooth responded positively to sensitivity testing, within normal limits, using ethyl chloride and an electric pulp tester.



Fig 3b Radiograph of an upper first molar tooth with moderate bone loss and suggestion of furcation involvement. Clinical examination revealed the tooth to have 'through and through' furcation involvement from buccal, mesial and distal aspects yet the tooth responded positively to sensitivity testing using both ethyl chloride and an electric pulp tester within normal limits.



Fig 3c Radiograph of a lower first molar displaying advanced bone loss. Clinical examination revealed the tooth to have 'through and through' furcation involvement from both buccal and lingual aspects yet the tooth responded positively, within normal limits, to sensitivity testing using ethyl chloride and an electric pulp tester.

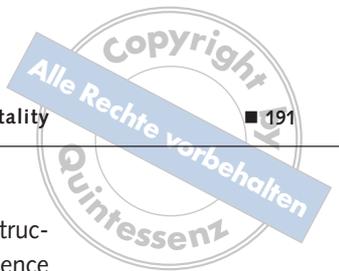
may simply reflect colonisation of an available niche. However, the presence of anatomical pathways, such as apical foramina, accessory canals and dentinal tubules, which connect the pulp and the periodontium, lends support to at least the possibility of cross-infection between the two.

■ Effects of periodontal disease and periodontal treatment on dental pulp

Histological techniques are often used to examine the dental pulp, and while useful it must be remembered that there are three associated inherent problems. Firstly, it is inevitable that the results produce a 'snapshot' of that pulp, frozen at one moment in time, and that this may not be representative of what has occurred in terms of pulp health in the past or would indeed occur in the future. The second is that a thin histological section only examines a tiny fragment of tissue, which again may or may not be representative of the whole. Thirdly, and perhaps most importantly, it may be difficult to achieve adequate fixation of the delicate pulp tissue and this can produce problems in differentiating between true pathology and mere artefact. This may complicate interpretation of the effects of many factors, including periodontal disease and treatment, on the health and integrity of the pulp. However, histology is the basis for much of the evidence found in the literature.

The evidence linking periodontal disease and periodontal treatment with pulpal disease is remarkably weak. A number of clinical and histological studies clearly demonstrate that it is very unusual for periodontal disease and/or therapy to result in pulp necrosis. These include the following studies.

- Histological examination of the pulps of molar roots, which had required resection due to severe periodontal destruction, but which demonstrated no inflammatory changes²⁴.
- Histological examination of the pulps of intact, caries-free teeth both with and without periodontal disease. Irrespective of the severity of periodontal disease present, all the pulps were histologically normal. Only in teeth with advanced caries or large restorations was there evidence of pulp damage²⁵.



- 100 patients with maxillary molar furcation involvement were studied for 5–24 years. Of the teeth 79% had < 50% bone support around one root pre-periodontal treatment. After therapy only 4% required root canal treatment and the authors believed pulpal damage to be due to caries or to have occurred below large restorations in each of these few cases²⁶.
- 52 patients with severe periodontal disease were followed over a 4–13-year period. Of 417 non-abutment teeth, 60% had lost more than two thirds of their bone support yet only 3% (14 teeth) required root canal treatment during the study time. In only 4 of these teeth was periodontal destruction the cause and in these cases it had extended to involve the root apices. In the same study 38 out of 255 abutment teeth required root canal treatment but periodontal disease was cited as the cause in only two teeth, and in these cases destruction had extended to involve the root apices²⁷.
- 571 teeth with advanced periodontal disease were studied for 5–14 years after they had undergone periodontal treatment. Only one tooth out of 571 required root canal treatment during the research period²⁸.

■ Conclusions

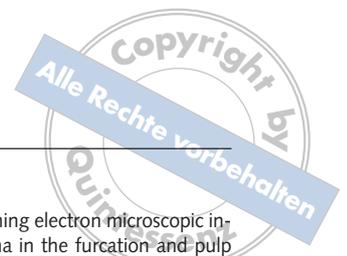
The most recent UK Adult Dental Health Survey was conducted in 1998 and demonstrated that over all age groups, 55% of dentate adults had pocketing greater than 3.5 mm and that this percentage increased with advancing age¹. This latter fact is not surprising given the greater time in older patients for ongoing insults from dental plaque to accumulate. Given the large number of teeth affected by periodontal disease, it would be logical to assume that if periodontal disease and/or periodontal treatment were indeed injurious to the pulp there would be large numbers of teeth that would lose vitality for which periodontal issues would be the obvious if not the only cause. The evidence from the literature shows convincingly that this is not so. Morris et al¹ reported that 5% of dentate adults over all age groups had pocketing greater than 5.5 mm and only 1% had pocketing in excess of 8.5 mm, although the respective figures for those aged 65 and

over were 15% and 4%. Severe periodontal destruction is thus relatively uncommon and the evidence from Langeland et al¹⁷ suggests strongly that only if destruction extends to affect the apical region is pulpal death likely to occur.

Figs 3a, 3b and 3c show examples of molar teeth with advanced periodontal disease sufficient to cause 'through and through' furcation involvements. All of these teeth responded positively within normal limits to sensitivity testing using both ethyl chloride and an electric pulp tester. This is in agreement with the experimental evidence quoted above.

It is also easy to assume that the presence of anatomical communications between pulp and periodontium mean that there are free open 'highways' available for bacteria and toxins to progress inward to the pulp. The literature demonstrates that these links may have partial obstructions, may not run the whole distance between periodontium and pulp and have an outward-moving fluid gradient. All these factors help explain why periodontal disease and periodontal treatment have apparently relatively little effect on the pulp.

Clinicians are charged with trying to ensure that clinical practice is evidence based and this is both important and necessary. It also means, however, that traditional methods and assumptions need to be challenged and re-examined and if necessary modified as required. Examination of the evidence suggests that the potential effects of periodontal disease and treatment on the pulpal health of teeth have been exaggerated. This does not mean that periodontal disease and treatment will never have endodontic effects but it does mean that the necessary periodontal insult would likely have to be so severe as to involve the root apex. The available epidemiological data imply that relatively few teeth will be so affected. There is no doubt that teeth that have periodontal destruction may lose vitality, but most of them will become non-vital because of other common causes including caries, presence of large restorations, and fractures. The responsibility of dentists to monitor vitality of teeth, including those with past or present periodontal disease, should not be abrogated and any loss of vitality detected should be appropriately managed. It seems clear, however, that the old perceptions of the threat from periodontal disease and treatment to the vital pulp need to be updated.



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