

## **GUEST EDITORIAL**

## Apical periodontitis: A very prevalent problem

Apical periodontitis, an inflammatory process around the apex of a tooth root, is primarily a sequel to microbial infection of the pulp space of teeth and is a remarkably widespread problem.1 The prevalence of apical periodontitis increases with age: by 50 years of age, 1 in 2 individuals will experience the disease.2 In individuals over 60 years old, the prevalence of apical periodontitis rises to 62%.2 The clinical management of apical periodontitis involves infection control by root canal treatment, which is the only viable alternative to the unsatisfactory option of tooth loss. On the basis of an average number of 2.2 root fillings per adult,3 it can be estimated from census data that there are 25 million root filled teeth in Australia and more than 420 million root filled teeth in the US. In the year 1990, an estimated 14 million root canal treatments were performed in the US alone.4

The magnitude of the problem does not seem to have been fully appreciated, and therefore apical periodontitis has not attracted the attention deserved by such a common disease. Another reason that apical periodontitis may not have received a greater degree of interest is that the success rate of treatment has generally been regarded as high, of the order of 87%.2 This figure applies to endodontic treatment done in specialist practice where a higher expertise is likely to result in a better technical standard of treatment, whereas the success rate in general practice is of the order of 72%.2 However, when the failure rate is measured relative to the prevalence of root canal treatment, the full dimension of the problem becomes apparent. When a conservative failure rate of 13% for root canal treatment in the average population is assumed, it can be estimated that there are 3.3 million failed root canal treatments in Australia and 54 million in the US; taking the greater failure rate for treatment in general practice, the figures could be as high as 7 million and 117 million, respectively. When these numbers are multiplied by the cost of endodontic retreatment and crown or restoration replacement, the cumulative economic impact is in the order of billions of dollars.

In recent years there has been a trend toward focusing on the technical aspects of mechanical instrumentation of the root canal as a measure of success of endodontic treatment. Although the mechanical elements of treatment undoubtedly deserve attention, a critical evaluation of relevant factors involved in the etiology of endodontic failure is more likely to yield meaningful information and, ultimately, rational treatment solutions. Sound clinical treatment must be based on a clear understanding of the causative factors in the disease process, so that intelligent and logical solutions can be applied to address the problem. For apical periodontitis, the essential role of microbial infection is well recognized as the critical etiological factor. Therefore, endodontic treatment is fundamentally the clinical management of a microbiological problem.

It is logical, then, that the principal treatment objective is elimination of bacteria and exclusion of further infection from the root canal. A clean, infection-free canal can best be accomplished by mechanical instrumentation in the presence of antibacterial irrigation followed by an antimicrobial intracanal dressing, which should in most cases provide predictable elimination of bacteria.

Although the method of root filling with guttapercha was first described more than 135 years ago,<sup>5</sup> it is astonishing that relatively modest progress has been made in defining the relative contribution of the root filling in the overall antimicrobial approach to root canal treatment. The idea that fluids percolating into an empty "dead" space might break down and be the cause of apical periodontitis originated as early as the 1930s,<sup>6</sup> but vestiges of the concept linger to the present day despite evidence demonstrating the fallacy of the idea.<sup>7,8</sup> The hypothesis that the "dead" space was a prime cause of apical periodontitis was propagated further during the mid-1950s and 1960s when the "Washington study," taken together with an

apical leakage study,<sup>9,10</sup> purported to confirm that most failures are *caused* by poor root canal obturation. A critical flaw in the hypothesis was that it failed to take into account the essential role of bacteria in the pathogenesis of apical periodontitis.

If the canal is free of infection at the time of obturation, there is a higher chance of success. 11,12 Advanced bacteriological techniques have recently been used to show that when bacteria persist in the canal at the time of root filling, there is a higher risk that the treatment will fail. 13 How high this risk will be depends on the quality of the root filling. In some of these cases the bacteria will die from the agents used in the root filling, and in other cases they will be entombed or lose access to nutritional material and die. But in all cases where viable bacteria remain in the root canal, there is a constant risk that they may continue to maintain a periapical inflammation.

During the latter part of the 20th century, a large number of journal publications have been devoted to variations of root filling methods with proponents for different techniques, but this has not led to a more sophisticated scientific approach to root canal treatment. That is not to say that a well-obturated root canal is inconsequential—it is undoubtedly an important component of good clinical treatment—but it ought to be viewed in relation to how it supplements the overall antimicrobial strategy. Recent studies have shown that obturation of infected canals may result in periapical tissue healing in a proportion of cases, but that optimal success is achieved when the canal has been rendered free of recoverable bacteria before root filling.<sup>13-15</sup>

It is clear from this discussion that there are broad health, social, and economic consequences of failure of endodontic treatment and potentially significant benefits individually and collectively if it were possible to reduce the proportion of endodontic failures. More research activity should be directed to where it can be of most benefit, which means a greater focus on the primary problem—why treatment fails and what can be done to address it. Although much more knowledge is required, there is already a substantial

source of information on improved treatment procedures. This information needs to be applied in the educational process and implemented at all levels.

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## REFERENCES

- Figdor D. Microbial aetiology of endodontic treatment failure and pathogenic properties of selected species. Umeå University Odontological Dissertations No. 79. Umeå (Sweden): Umeå University: 2002.
- Eriksen HM. Epidemiology of apical periodontitis. In: D Ørstavik, TR Pitt Ford, editors. Essential Endodontology. Prevention and treatment of apical periodontitis. London: Blackwell Science; 1998. p. 179-91.
- Eriksen HM. Endodontology—epidemiologic considerations. Endod Dent Traumatol 1991;7:189-95.
- 4. American Dental Association. 1990 Survey of Dental Services Rendered. Chicago: American Dental Association; 1994.
- Cruse WP, Bellizzi R. A historic review of endodontics, 1689-1963, part 1. J Endod 1980;6:495-9.
- Rickert UG, Dixon CM. The controlling of root surgery. Transactions of the Eighth International Dental Congress, 1931;15.
- Torneck CD. Reaction of rat connective tissue to polythylene tube implants. Part I. Oral Surg Oral Med Oral Pathol 1966;21:379-87.
- Torneck CD. Reaction of rat connective tissue to polyethylene tube implants. Part II. Oral Surg Oral Med Oral Pathol 1967; 24:674-83.
- Ingle JI, Glick DH. Modern endodontic therapy. In: JI Ingle editor. Endodontics. 1st ed. Philadelphia:Lea & Febiger;1965. p. 25-77.
- Dow PR, Ingle JI. Isotope determination of root canal failure. Oral Surg Oral Med Oral Pathol 1955;8;1100-4.
- Engstrom B, Hård af Segerstad L, Ramström G, Frostell G. Correlation of positive cultures with the prognosis for root canal treatment. Odont Revy 1964;15:257-70.
- Heling B, Shapira J. Roentgenologic and clinical evaluation of endodontically treated teeth, with or without negative culture. Ouintessence Int 1978; 9:79-84.
- Sjögren U, Figdor D, Persson S, Sundqvist G. Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis. Int Endod J 1997; 30:297-306.
- Katebzadeh N, Hupp J, Trope M. Histological periapical repair after obturation of infected root canals in dogs. J Endod 1999; 25:364-8.
- Katebzadeh N, Sigurdsson A, Trope M. Radiographic evaluation of periapical healing after obturation of infected root canals: an in vivo study. Int Endod J 2000;33:60-6.