

Pathologic interactions in pulpal and periodontal tissues

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Abstract

Both endodontic and periodontal disease are caused by a mixed anaerobic infection. The pathways for the spread of bacteria between pulpal and periodontal tissues have been discussed with controversy. This article is an attempt to provide a rational approach to the perio-endo/oro-perio questions based on a review of the relevant literature. In the light of evidence, clinical concepts for the diagnosis and treatment of lesions involving both periodontal and pulpal tissues are discussed.

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A lesion involving both periodontal and pulpal tissues can be of primary endodontic, primary periodontal or stem from separate origins (arising from both the endodontic lesion and the marginal periodontal lesion have developed independently). Different authors have created varying nomenclatures for these pathologies, based on either aetiological or clinical criteria, or a combination of these (Olthoff & Pittsack 1964; Nason et al. 1972; Chakraborty 1985). The clinical aspects of perio-endo lesions have been discussed in depth (Hann 1977; Harrington 1979; Garbisa 1984; Mong 1999). However, scientific support for the current understanding of the matter is sparse. Much of what is in the literature consists of anecdotal reports. There is general agreement today that the vast majority of pulpal and periodontal lesions are a result of bacterial infection. Under which conditions and especially in which direction spread of the disease occurs in the pulpo-periodontal continuum remains a matter of controversy.

This article attempts to provide a rational approach to the perio-endo-question based on a review of the relevant literature. Special attention is given to the natural and man-made path com-

muting the involved tissues and the communication of microorganisms and their products within these pathways. Clinical considerations are discussed for each type of lesion, using the classification of Nason et al. (1972), separating lesions involving both periodontal and pulpal tissues into the following groups:

- primary endodontic lesions with secondary periodontal involvement,
- primary periodontal lesions with secondary endodontic involvement, and
- true combined lesions.

Microbiology of endodontic and periodontal lesions

In a letter to the Royal Society of London in 1683, Antoni van Leeuwenhoek, the father of microbiology, described the 'animalcules' contained in dental plaque and tartar and produced the first drawing of bacteria (Ishida 1988). The presence of bacteria in necrotic human pulp tissue was first described by Miller (1904). It was not until the groundbreaking experiments in animal models by Ogata et al. (1977) and Kakehashi et al. (1987) that micro-

organisms were established as the main cause of periodontal and pulpal disease. The concepts obtained from the previously cited animal studies were later confirmed in man (Olin et al. 1985; Sundqvist 1976). Periodontal disease is now thought by most researchers to be caused by a mixed anaerobic infection, mediated by a complex interplay with local and host factors (for review, see Löwrich 1999, Page 1999). Similarly, endodontic infection of necrotic pulp tissue is of an anaerobic nature (Sundqvist 1976). An exception to this rule seems to be the microaerophilic *A. pyrophilum* complex, which has been associated with aggressive periodontitis (Nyman & Socransky 1977). Most of the species that have been found in infected root canals can also be present in the periodontal pocket (Mason 1987; Sundqvist 1994). However, *Porphyromonas endodontalis* seems to be very rare in oral infections other than those of endodontic origin (Van Winkelhoff et al. 1988). Overall, the root canal flora does not appear to be as complex as the periodontal flora of adjacent pockets (Karlsson et al. 1997). However, it is an inherent problem in bacterial sampling of periodontal pockets that bacteria from more shallow levels of the site are har-

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