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Editorial

Endodontic Medicine

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In this volume we observe how dentistry is becoming more technical every day, and is achieving spectacular results in dental treatment. Similarly, we observe that dental research is highly active, and consider particularly worthy of note studies such as that on the new models of implants by the group of Dr. Kianor Shahmohammadi, or the eminently practical protocol proposed by the Greek group of Professor Zouloumi Lambros.

Without providing comment on the wealth of important articles from this excellent volume, as teachers of oral pathology we will comment specifically on the concept of Endodontic Medicine, an aspect that interrelates the degree of health of the teeth with the patient's systemic condition.

One of the groups that has carried out work in this area is the Sevillian group of Professor Juan José Segura Egea. This author specifies in his study [1]: [Adapted] Following the analogy for the hypothesis formulation described by Stuart Mill in his 2008 publication in the Journal of Endodontics [2], it is plausible to assume that chronic apical periodontitis is also associated with the same systemic alterations to which periodontal disease is associated. The "endodontic medicine" should, therefore, be developed following the path of "periodontal medicine": investigating the association between endodontic pathology and therapeutics and systemic pathologies. However, although studies have been published that relate endodontic pathology and therapeutics to diabetes mellitus [3-6], smoking [7], ischemic heart disease [8,9], hematological diseases [10,11], renal disease [12], or osteoporosis [13], the probable relationships between the state of systemic health and the chronic inflammatory pathology of endodontic origin have hardly been studied [14].

This aspect is justified if we think that, although there are differences between chronic periodontal inflammation and chronic apical periodontitis of endodontic origin, both share three aspects: i) both are chronic infections of the oral cavity, ii) the etiopathogenesis of both share a microbiota and are considered to be polymicrobial infections with a predominance of Gram negative anaerobic bacteria [15]; (iii) in their pathophysiology, the two entities involve an increase in the local levels of cytokines and mediators of inflammation (in the crevicular fluid in the case of periodontal disease and in periapical tissues in apical periodontitis). These mediators may have repercussions on the systemic levels [16-18].

Finally, we can conclude this very brief review by saying that the results of the studies carried out so far do not seem to be conclusive. Instead they indicate that the patients' periodical health may be directly related to their systemic health status, and that therefore it is important to conduct verified research on this subject.

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