Guest Editorial: Focal Infection Revisited-the Dentist as Physician

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Dentists can undervalue themselves. Sometimes we do not take cognizance of our significance as members of the medical profession. We are responsible for dealing with the most widespread of all human diseases, chronic inflammatory periodontal diseases and dental caries. Do our medical colleagues recognize this? Do we?

While not as attention-grabbing as some of the infections of our time, we appear to be in danger ourselves of forgetting that the mouth constitutes a major portal of entry for a wide range of micro-organisms, whether inherently oral commensals or direct pathogens, the effects of which may be merely troublesome, as with the common oral infectious diseases, or more serious. There is an analogy with oral neoplasia which, though it may be as prevalent as cervical cancer, does not attract anything like the attention of the latter. One wonders why. Ever since the original concept of focal infection led to an excess of extractions over 70 years ago, the theory has been in relative disrepute. And yet to ignore focal infection is to refuse to recognize an abundant literature, all of medical significance.

It was W.D. Miller (1890) who, as with so many other aspects of oral microbiology, drew attention to the importance of mouth bacteria in systemic disease. Since his description, every major body system has been listed as a target for infections from oral foci, mainly pulpal and periodontal in origin. From the mouth, oral organisms have been traced to the sinuses, including the cranial blood sinuses, the brain and peripheral nerves, the heart and other elements of the cardiovascular system, the mediastinum, the lungs, and the eyes. The consequences have often been fatal—mediastinitis, endocarditis, myocarditis, brain abscesses. There is far more to consider than bacterial endocarditis as a consequence of bacteremia of oral origin. There is also some evidence that microbial fractions, rather than whole cells, may play a part, perhaps in some form of immune complex, in systemic forms of inflammation not overtly infectious in nature—for example, some forms of arthritis.

Perhaps the age of antibiotics has made us complacent. It is possible that we may also become complacent about the common dental infections that are the source of much of our livelihood. Some would say that dental caries is a diminishing problem, even though it remains a widespread disease, is far from disappearing, and has periapical sequelae of considerable importance with regard to more serious oral and systemic infections. Similarly, the term "periodontal disease" is used as if it were a single entity, whereas the periodontium is subject to a range of disease as wide as that of any other tissue or body system. In the context of focal infection, inflammatory periodontal diseases comprise a group which, though often similar in pathology, has a host of microbial etiological factors which may enter the local tissues, or spread systemically—for example, through fascial planes or *via* the bloodstream.

How often do we say that bacteremia is a frequent occurrence, in health, in gingivitis, in periodontitis, provoked even by such minimalist factors as chewing or toothbrushing? Yet how seldom do we appreciate the possible systemic consequences of that bacteremia? One problem is that inflammatory periodontal diseases and caries are so common, that sequelae are much less frequent, and their possible systemic significance is easily overlooked. A further difficulty is that the consequences of bacteremia are so unpredictable. To date, there seems to be no clear correlation between the fact or level of a bacteremia and the incidence of a linked systemic disease, even for the favorite, endocarditis. Yet, which dentist would refuse antibiotic prophylaxis to a patient at risk to bacteremia? Then there are the further complications of increasing resistance to antibiotics, the expanding range of microbial species that may be involved, and the rising numbers of patients with immunosuppression of drug or other origin.

We cannot say that the age of dental surgery is passing and that the age of dental medicine is about to begin. But we must be aware that it is becoming increasingly unacceptable to rely upon empiricism in dental treatment, or always to expect the appliance or the complicated procedure to be the apotheosis of dental practice. The age of diagnosis is here. For dental caries and, especially, for chronic inflammatory periodontal diseases, we now depend increasingly upon accurate definition of the etiological factors, key host-response elements, and the precise disease classification for each individual patient. We may expect a much clearer understanding to follow of the relationship between the oral foci of these infections and their near and distant body targets. We may also expect our colleagues in other fields of medicine to turn to us for assistance in preventing the often serious consequences to other tissues and organs of oral focal infection, in understanding inflammatory processes in their fields of study, and in exploring possible links between oral micro-organisms and inflammatory systemic disorders not heretofore considered infective in nature.

If our role is to be increasingly that of oral physician, then our skills in oral diagnosis will have to increase. We will have to be more precise about the evidence for oral sources of systemic infection, so that we do not revert to that orgy of extractions that many years ago discredited focal infection. The mouth may be more boring, in the public mind and in the health departments of government, than other body orifices, but its medical significance has been underestimated for too long. It is time to re-examine the role of focal oral infection in systemic disease.

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