EDITORIAL

Dental factors in infective endocarditis

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SOMETIMES the well-being of patients is influenced profoundly by aspects of health care delivery that are so subtle as to nearly evade notice. An example is to be found in the issue of whether or not a causal relationship exists between repeat episodes of endocarditis and the failure to look for and eliminate dental sepsis during hospitalization for the initial episode. If this is a real issue, it is one that owes its existence to the historical accident whereby the fields of medicine and dentistry became separate realms. Most physicians receive little or no didactic exposure to oral diagnosis and principles of treatment while in medical school or during residency training. As a consequence, dental disease resides largely outside the pale of medicine, and its impact on overall health may occasionally be overlooked.

The reason patients with multiple episodes are alluded to above is that there seems to be a useful distinction to be made between patients who are at risk for endocarditis by virtue of having a turbulence-producing lesion and those who have already had an episode of endocarditis. Despite infective endocarditis being a rare disease even in the at-risk population, the proportion of patients with endocarditis who have subsequent episodes is reported in the literature to be about 5%. The literature suggests that an initial episode of endocarditis singles out the truly susceptible individual from the population of nominally at-risk patients.

An alternative hypothesis might be that patients with more than one episode are not necessarily more susceptible but that their treatment fails to eliminate a potent causative factor. These hypotheses are not mutually exclusive — in fact, they may be simultaneously operative in the same patient. Also, both lead to the same conclusion, as discussed below. In either case, the role of transient bacteremias in the pathogenesis of endocarditis is well established, and it has been amply demonstrated that professional manipulation of teeth and gums, routine home hygiene activities, and even the simple act of chewing all produce transient bacteremias, the extent of which reflect the individual’s underlying state of dental and periodontal health. We also know that the organisms most frequently cultured from the blood of patients with infective endocarditis are streptococcal species commonly found in the oral cavity.

Despite these considerations, at my hospital (Massachusetts General Hospital, Boston) no more than one-third of patients treated for endocarditis receive an oral surgical or dental evaluation, and it would appear that an even smaller proportion of those patients whose endocarditis is specifically of a nondental origin are ever seen for such an evaluation. Discussion with colleagues at other hospitals suggests that systematic screening for dental disease is an exception rather than the rule.

Given the prevalence of periodontal disease in the age group of the average patient with endocarditis, it seems highly likely that some patients successfully treated for endocarditis are being discharged from the hospital with significant untreated oral sources of bacteremia in the face of a demonstrated capacity to develop endocarditis.

Because of the difficulty inherent in prospective studies of endocarditis, there may never be a definitive answer to the question of whether or not neglected pathologic dental conditions actually contribute to the occurrence of repeat episodes of endocarditis. Nonetheless, there seems to be a logical basis for treating all endocarditis patients as members of a higher-risk subset of the population at risk. All such patients should be carefully screened for dental sepsis, even when the original causative organism has been identified as nondental in origin.

All patients being treated for endocarditis should receive a noninvasive oral examination in conjunction with a full-mouth series of periapical radiographs. Steps should also be taken to render such patients free of pathologically mobile teeth, and active periapical or periodontal disease should be treated either by extraction or by more conservative means consistent with the restorability of the dentition and the patient’s motivation to establish and maintain a satisfactory level of
dental health. The onus is upon the physician and dentist to satisfy themselves that a patient with an established history of dental neglect will, in fact, "turn over a new leaf." Otherwise, the patient may be done a disservice by the failure to remove such teeth as part of a strategy for long-term prophylaxis.

As to the dental needs of nominally at-risk patients with predisposing cardiac lesions but no prior history of endocarditis, given the indeterminate nature of actual susceptibility, it seems prudent to recommend that all such patients be maintained as free of dental disease as possible. However, there remains no authoritative basis for deciding how aggressive to be in the pursuit of this objective.

References