On Manufactured Diseases, Healthy Mouths, and Infected Minds

Our profession takes pride in its successful merger of clinical skills and academic excellence, together with the realization that diagnostic and treatment concepts are not necessarily static ones. Some of our dearly held convictions have successfully withstood the test of time, while others have fallen into disrepute. We no longer replace every missing tooth, treat temporomandibular disorders with occlusal rehabilitations, argue over the "best" occlusal scheme, or ignore the biologic cost of prosthodontic interventions on the oral ecology. We also make a conscious effort to identify gaps in our knowledge and address them through objective analyses and, where possible, research initiatives. We remain scrupulous in our efforts to address our patients' concerns while ensuring that we neither promote nor condone overtreatment. The same occurs even more frequently in medicine, where a far larger spectrum of intervention considerations often demand renewed assessments. A particularly recent example is the realization that the standard removal of cancerous lymph nodes for breast cancer in certain women will no longer be required. This research will inevitably change medical practice and have profound implications for patients.

The successful incorporation of osseointegrated implants into clinical practice has already brought immense benefits to many patients worldwide. A great deal is now known about the biology of osseointegration and its clinical yield, with evolving biotechnologies catalyzing new knowledge for managing anatomical challenges, increasing patient expectations with respect to cost and an enhanced appearance, expanding therapeutic indications, and even refining long-term outcome results. But no intervention is entirely free of complications, and this too must be readily acknowledged. A particular ecologic concern is whether implant prosthodontic treatments induce clinically relevant disease states in the oral cavity. In fact, several recent investigators have suggested that a substantial percentage of treated patients suffer from diseases and conditions affecting their osseointegrated implants that may, in turn, require invasive and costly interventions to address. Given the enormous body of knowledge that already clearly demonstrates excellent long-term clinical outcomes, a number of questions have to be posed: Are there really diseases and conditions that constitute a credible health threat, or has an overt sense of caution suddenly generated a severe case of "cold feet" and displaced sound clinical reasoning? Are patients treated with osseointegrated implants really vulnerable to serious disease processes, or are we succumbing to the temptation to welcome a new member to the "manufactured diseases" club?

The most debatable implant-related diseases in question are arguably so-called "peri-implantitis" and "peri-mucositis." The latter is a version of gingivitis seen around implants or implant-anchored prostheses and is an inflammatory response to plaque. Peri-implantitis, on the other hand, is popularly defined as inflammation in the tissues around the implant combined with loss of supporting bone. The hypothesis for its occurrence is that bacterial deposits in a susceptible host will elicit and propagate bone loss, eventually leading to implant loss. Hence, the reasoning goes, careful screening of candidates for implant therapy, thorough monitoring of patients with osseointegrated implants, and detailed intervention protocols for suspected cases of breakdown are strongly advocated. We contend that this is an overt "periodontal ligament–centric" approach that regrettably presumes the following: *(1)* that the tissues around osseointegrated implants are automatically vulnerable to bacterially induced disease processes and conditions and *(2)* that bacterially induced peri-implantitis is really a precursor or predictor of implant failure.

It is therefore ironic that a therapy that has helped so many patients (including those suffering from severe periodontal disease, which may have accounted for tooth loss in the first place) is now regarded with suspicion as being susceptible to the very same pathologic agents as periodontal disease per se. This is, of course, not a new supposition; it was raised early on when osseointegration was first introduced and when implants were making a lateral move from fully edentulous to partially edentulous applications. Clinical evidence from human trials has subsequently shown the suspicion to be groundless and of minor cause for concern. Similarly, anxiety that implants would experience significantly higher failure rates in patients previously treated for periodontal disease has also been largely discounted.

The existence of occasional marginal bone loss around osseointegrated implants cannot be denied and remains a clinical challenge. Both its cause and management are based on sheer speculation at this stage of our knowledge of the specific role that the induced healing phenomenon plays in the long-term maintenance of the osseointegrated response. Consequently, almost nothing is known about the best maintenance protocol for implants in terms of either the frequency or the parameters to be monitored.

The frequent observation of self-limiting postsurgical bone remodeling confounds the easy distinction between peri-mucositis and peri-implantitis. Inconsistent definitions of so-called peri-implant diseases and disease severity hamper interpretation of prevalence data and intervention studies. And, while good oral hygiene practices are routinely promoted, and the relationship between plaque deposits and mucosal inflammation is self-evident, any connection between bacterial deposits and clinically relevant bone loss is yet to be compellingly demonstrated in long-term human clinical trials. Glaring differences exist between peri-implant and periodontal physiology, although some underlying disease risk factors, such as smoking, appear to be shared between diseases affecting attachment loss around teeth and crestal bone loss around implants. The key consideration that this journal has recurrently underscored remains a very compelling one: There is a very profound difference between the two interfaces upon which teeth and osseointegrated implants depend for their continued integrity. The periodontal ligament is the result of an evolutionary phenomenon, while the interfacial osteogenesis that occurs in osseointegration is the result of a controlled and induced healing response. Insisting that the pathogeneses of the periodontal ligament's disease processes are identical to what causes infrequent, if diverse, amounts of marginal bone loss around osseointegrated implants is probably specious and misleading.

Many questions remain unanswered in the field of implant therapy, with marginal bone behavior in the context of long-term loading a particularly interesting one. Fortunately, however, attempts at rigorous and repeated long-term scientific investigations have clearly shown that so-called peri-implantitis is of little concern for the vast majority of patients. Assertions for its occurrence as deserving of disease status, together with the ensuing need for costly and invasive interventions, are unwarranted and misguided. A disease continues to be defined as an abnormal condition of the body or mind that causes discomfort or dysfunction, and we do not believe that an infrequent and ill-defined change in marginal bone height around implants qualifies for such a label. There is clearly a need for better science and more common sense to be devoted to this intriguing, if clinically peripheral, topic. In the meantime, we must continue to make sure that the diseases we treat are in our patients' mouths and not in the minds of overzealous colleagues.

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Both Drs Koka and Chvartszaid are on the IJP's editorial team—the latter as a reviewer and the former as a member of the advisory board. Dr Chvartszaid is qualified in both the specialities of prosthodontics and periodontics at the University of Toronto. Dr Koka is professor and head of dentistry at the Mayo Clinic in Rochester, Minnesota. He is qualified in prosthodontics and his PhD dissertation focused on bone physiology.

Clarification

I would like to apologize to those authors whose work I inadvertently failed to acknowledge in my recently published paper "Evaluation of Different Esthetic Smile Criteria," which appeared in issue 1 of 2011 on pages 64 to 70. My oversight was clearly due to my extensive familiarity with their publications, which are quite similar to my own work. As a result, I regrettably failed to distinguish between my own descriptions and the already published ones. I extend my sincere apologies to the authors, plus an expression of gratitude to them for their published works as listed below.

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Respectfully submitted,
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