Osseointegration: Promise and Platitudes

This editorial is not an unbiased one. Our early acceptance of a prescient definition of osseointegration—a time-dependent healing process whereby clinically asymptomatic rigid fixation of alloplastic materials is achieved and maintained in bone during functional loading—acknowledged the promise of the induced interface as the sine qua non for all implant-dependent oral rehabilitative initiatives, and with our discipline as the major stakeholder. We therefore distance ourselves from colleagues who insist that the interface is periodontal ligament-like and equally vulnerable to its pathogenesis. The latter cohort of clinical educators undermines the technique’s extraordinary benefits through lecture title teasers such as “Peri-implant Mucositis: The Gateway to Peri-implantitis?” or assertions that, “In 5 years, implants of a specific macro- and microfinish will develop peri-implantitis.” It is clearly time for dentistry to start a serious debate on the topic of biologic failure of dental implants and deal with the above platitudes in a clinically rigorous context.

We suggest that a more meaningful conversation will ensue if both interfaces are viewed conceptually in the context of a host with an entity (tooth or implant) embedded in it. Modeling the two systems independent of worrying about how the two entities became embedded in bone in the first place would obviate the irrelevant and damaging biases conjoined with preosseointegration thinking. It is only at the macroscopic level that the osseointegrated implant–host interface and the tooth–host interface appear to be similar. If one imagined a tooth and an implant both as alloplastic inert objects, he should accept both interfaces with host osseous tissue and host mucosal tissue and that these two host interfaces are independent of each other except for the region where osseous and mucosal tissues interact. The nature of their interactions has been described from anatomical and histologic perspectives and shows that both mucosal interfaces may experience an inflammatory response as a result of bacterial challenge—a response that can be mitigated by a reduction in inflammation-eliciting bacteria below the tolerance threshold of the host’s immune system. However, there are significant dissimilarities at the interfacial osseous level for the very good reason that one results from evolutionary development and the other from an induced healing response. We believe that it is simplistic to presume that their associated time-dependent biologic and functional consequences and outcomes are identical.

Hence, there is a need for recognition of these differences and acceptance of an uncoupling of mucosal and osseous responses around an osseointegrated implant. If the mucosal responses are similar for the tooth and implant but the osseous responses are different, their response relationship must also be different. Profound concerns for how we think about diagnostic and therapeutic baselines and management of ideal and nonideal clinical presentation would then immediately come to mind: Should diagnostic thinking of marginal bone loss around the implant-host interface be predicated on our thinking of the tooth-host interface? And should treatment of marginal bone loss around the two interfaces be undertaken based on similar notions?

We readily admit that many questions remain unanswered in the field of implant therapy, especially our understanding of both the time-dependent qualitative and quantitative aspects of the resulting induced interfacial healing process. Osseointegration (OI) has been shown to retard residual ridge reduction while certainly not precluding different degrees of it. This has been a boon for prosthodontists who are very familiar with the otherwise inevitable, unpredictable, and diverse changes occurring in jawbone sites following tooth loss, hence our expectation that time-dependent, circumimplant marginal bone changes occur, albeit at a significantly reduced rate, when compared to what would happen if successful OI were not present. We regard such bone changes as inevitable and largely innocuous, as well as greatly influenced by site-specific morphologic considerations. Readers should also recall that robust long-term scientific investigations from different centers have already shown that these occurrences have proven to be of little concern for the vast majority of patients. Marginal bone loss around immobile and asymptomatic implants is not an automatic sign of osseous disease presence, even when threads are exposed and inflammatory gingivitis is present. It may be tempting for the dentist to employ a catchy term for such a clinical picture, but implying that this is a disease process that demands treatment intervention is intellectually and professionally indefensible. This would also presume comparable biologic outcomes for the evolved periodontal ligament and the healed response of OI, and even worse, it would subsume...
The reduction of bone around an implant into the pathogenesis of a periodontal disease. Such a narrative ignores the likelihood of a spectrum of healing responses with different time-dependent clinical marginal bone outcomes.

A broader context for the range of interfacial healing responses in OI is needed if we are to understand why osseoseparation (partial or complete osseointegration failure) may infrequently occur (see paper by Koka and Zarb in this issue, page 48). The topic demands its rightful place in dental curricula, especially at specialty training levels, since teeth and implants are simply different clinical entities and cannot be managed as such. Clinical educators should also avoid promulgating misleading analogies to periodontal disease pathogenesis. A day of reckoning for so-called “peri-implantitis” is well overdue, and it is time to expunge the term from routine use.

George A. Zarb and Sreenivas Koka

Sreenivas Koka is a professor of dentistry and chair of dental specialties at the Mayo Clinic, Rochester, Minnesota. Along with clinical education received from the University of Michigan, Dr Koka’s PhD dissertation (University of Nebraska) focused on bone physiology and osteoblast regulation. His clinical and scholarly passions stem from a full-time clinical practice and research into clinical outcomes, especially those related to understanding relationships between oral and systemic bone physiology. Dr Koka is a member of the editorial advisory board of the International Journal of Prosthodontics.

Dr Turul Altay, a Turkish prosthodontist who practices and teaches in Ankara, is an intrepid mountaineer. He sent his best wishes for a happy and healthy 2012 to this journal’s editorial board and international readership via this photograph that was taken during his recent expedition to Mount Blanc. It is a delightful reminder that the IJP makes for great reading even while pursuing nondental pastimes.