Preface: Implants Are Not the Same as Teeth

The imminence of the end of my tenure as Editor-in-Chief of the IJP demands additional acknowledgment of events and individuals who helped shape my life in dentistry. Study experiences at three dental schools—the Universities of Malta, Michigan, and Ohio State—provided me with invaluable foundations for pursuing clinical academia. My subsequent four decades at the University of Toronto ensured enriching contact with brilliant scholars from both basic and clinical sciences constituencies . . . plus the magic of sabbaticals. Sabbatical leave is inarguably an integral and essential part of a university career. It enriches intellectual growth by offering time and scope for consolidating a scientifically based clinical teaching strategy, while countering the risk of burnout (ever-present in a time-dependent context for clinical dentistry's teachers and practitioners). Above all, it reinforces a renewed commitment to the scientific method and the effort to get as close to it as humanly possible. This is particularly significant if Prosthodontics is to strengthen its biologic underpinnings rather than restrict itself to already impressive mechanical foundations. The experience often raises unanswered questions since it is a quest that underscores both the challenges and limitations of scientific progress as we continue to seek to promote clinical research embedded in care.

As a career academic, I took advantage of sabbatical leaves and was privileged to spend two of them in Göteborg, Sweden. The profound influence of Scandinavian clinical scholars (Neils Brill, Henry Beyron, Gunnar Carlsson, Jan Lindhe) combined with the revolutionary publications of Per-Ingvar Brånemark led me to spend my first sabbatical in the latter researcher's lab in 1983—an invaluable career experience, as it coincided with his introduction of the osseointegration (OI) technique and its eventual global impact on pre-prosthetic surgery and implant prosthodontics.[1] My second Göteborg sabbatical, in 1990, was spent in Tomas Albrektsson's research laboratory, where we engaged in fulsome debate on OI's verifiable facts and the loopholes and shortcomings that lingered in our practical and teaching applications of the technique. We sought to provoke in-depth debate regarding differences between evolutionary developmental aspects of a periodontal ligament and the induced healed interface between an implant and a selected host bone site that is the characterization of OI. We argued that diverse influences determined the long-term outcomes of the OI surgical and prosthodontic loading protocols in the context of the efficacy of the induced healing response, and that its likely time-determined dependence on systemic influences on bone behavior, needed further study. We sought to underscore the merit of this approach by challenging the popular conviction that marginal bone loss around implants resulted from a periodontitis-like disease that was conveniently, if misleadingly, labeled as a distinct disease: peri-implantitis. Our articulated conviction also emphasized the profession's failure to date to fully understand edentulous bone resorption behavior as an unpredictable and imperfectly understood (even if well-documented) morphologic sequel in bony sites that lack the organizational influence of a healthy periodontal ligament. The contributing roles of occlusal stresses, presence of chronic inflammation from overlying removable prostheses, or site specificity remain speculative considerations and have moreover been frequently overlooked when implants are located in host sites that demonstrate a clear discrepancy from the volumetric dimensions necessary for the implant.

The scientifically unresolved peri-implantitis debate has now raged on for several years. It regrettably continues to confuse many dentists and clinical educators, some of whom ominously claim that tsunamis of this presumed disease are now imminent. It has become increasingly clear that a broader approach—indeed, understanding—is needed to thoroughly differentiate long-term behavior of marginal bone around implants from that which occurs in periodontal disease.[2] Tomas Albrektsson has continued to champion the cause of a more robust, scientifically eclectic approach to better understand the profound differences between natural dentitions and implant-supported ones. He has continued to insist that the resulting ideologic divide seeking to explain marginal bone loss around implants should be a strong reminder that dental implant therapy is simply not reducible to tidy formulas or rigidly ordered credos and that it demands scrupulous observational skills that overcome the absence of hard scientific evidence to justify what might very well be unnecessary and misguided interventions.

I continue to regard his scholarly approach to this controversial topic as intellectually compelling; hence, my acceptance of his welcome invitation to co-author this, my penultimate IJP editorial. I am also delighted to include an introduction to Irena Sailer, who was recently selected by Quintessence Publishing to be this journal's next Editor-in-Chief. Her many academic accomplishments to date augur so very well for the IJP's ongoing commitment to advancing the discipline's scientific mission.

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Editor-in-Chief

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Implants Are Not the Same as Teeth

We have known since the 19th century that surgery must be done under aseptic conditions to ensure reliable clinical outcomes. However, although aseptic conditions do not prevail in the oral cavity, preprosthetic surgery for oral implants is generally associated with a very high rate of successful outcomes. While the natural dentition and implanted teeth root analogs known as implants are of course associated with different bony attachment mechanisms, they are considered identically vulnerable to bacterial attacks that result in inflammatory disorders accompanied by subsequent resorption of the surrounding marginal bone. However, there is one compelling difference between an implant and a tooth: The former is a foreign body that elicits an established immune response, one that is rarely accompanied by a plaque-induced loss of marginal bone. It is also obvious that implants must have a proper bacterial defense mechanism to enable them to survive in 95% or more of clinical situations, as has been documented over a decade of scrupulous follow-up documentation.

A recent paper[1] proposes the presence of a dual defense against bacteria when implants are placed in which inflammatory cells cooperate with immunologic cells, such as macrophages, to prevent adverse bacterial actions (Fig 1). Donath et al[2] had already reported the chronic nature of the circum-implant inflammatory state while emphasizing the activity of the immune defense throughout the lifetime of the implant, even if some evidence points to a time-dependent attenuation of the defense system. However, if implants are challenged by, for example, the presence of cement remnants in the surrounding soft tissues, sudden occlusal overload, or even pharmaceutical provocations, there is a clear reactivation of the inflammatory and immunologic defensive modes of action. At times, the challenge may be so substantial that the defense fails, and bacterial attacks against the implant will then be possible and even prevail. However, current research indicates that this defensive breakdown is indeed a rare occurrence, at about 1% for both oral and orthopedic implants.[1] Moreover, failure to revise these implants is associated with a higher risk for bacterial breakdown. The induction of osseointegration (OI) is an example of an immunologically determined bony demarcation from the implant, as originally pointed out by Donath et al[2] and best reflected in an updated OI definition: ". . .a foreign body reaction where interfacial bone is formed as a defense reaction to shield off the implant from the tissues."[3]

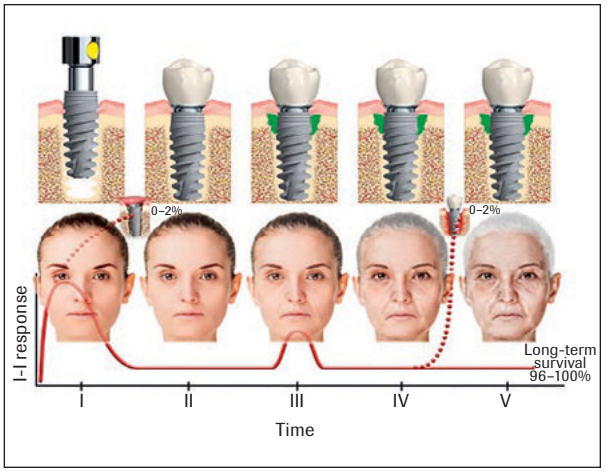

Fig 1 At implant placement, the inflammatory-immunologic (I-I) response is rapidly elevated (I) to protect against bacterial attack, with 0% to 2% of implants displaying primary failure for reasons that remain unknown. The I-I response is thereafter attenuated (II), but may be easily activated by provocations such as cement remnants in the soft tissues, occlusal overloading, or adverse pharmaceutical reactions (III). The time-dependent defense efficacy may fail in saving all implants, and the 10-year assessment evaluation may show that another 0% to 2% of implants have also failed (IV). However, scrupulously documented long-term survival rates of 96% to 100%, when carried out by well-trained clinicians using scientifically documented implant systems (V), offer robust reassurance for implant prosthodontic management of both partial and complete edentulism.

A growing body of evidence counters the simplistic explanation for marginal bone loss around oral implants as dependent on and preceded by plaque formation, with associated mucositis and eventual ongoing bacterial attacks. Plaque formation is commonly seen around parts of the oral implants that are not anchored to bone, and there is no evidence that this accumulation expands to other regions. Mucositis is nothing but the chronic inflammation that affects the soft tissues around an implant at the same time as it works as part of the bacterial defense. Marginal bone loss around oral implants is mainly related to treatment decisions and protocol complications rather than representing a form of disease. Moreover, marginal bone resorption usually ceases spontaneously or in response to different defense actions. For those of us who realize that implants are very different from teeth, these matters are easily understood and provoke an appropriate critical attitude toward those who have failed to appreciate the basic biologic principles that lead to successful osseointegrated implants.

We realize that time will regrettably elapse before the error of regarding tooth and implant as being similar bodies is rectified and that these examples

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of clinical nuisance are not automatically ominous conditions that must be treated, especially invasively. In fact, some clinicians have even presented drastic treatment approaches for what they perceive to be a disease by grinding down the entire superior portion of the implant—an extraordinary example of overtreatment that in fact may harm OI by spreading titanium particles into the tissues. The debate regarding therapeutic initiatives to arrest marginal bone loss will undoubtedly linger, and common clinical management techniques will go on being applied to inflammatory sequelae around teeth and remain an ongoing mindset for some colleagues, even though the alleged peri-implantitis disease can be so readily managed via minimal interventions.[1,4]

Many years ago, we adopted an educational objective of actively sharing the evidence-based concept of osseointegration with North American clinicians. We frequently heard the comment that OI's reported success was due to the fact that our treatment experiences were almost exclusively restricted to totally edentulous patients and that lateral therapeutic initiatives in partially edentulous patients would provoke bugs from nearby teeth to infect implant sites and lead to failure, although subsequent documented experiences proved otherwise. In fact, a recent clinical study in which the investigators examined patients with both teeth and implants in the same jaw verified that implants were not threatened by teeth bacteria. When implants showed marginal bone loss, teeth did not; and alternatively, when teeth lost bone, circum-implant bone was stable. Simultaneous bone loss was observed in only 3% of patients with teeth and implants,[5] underscoring the simple fact that implants and teeth are not the same.

There is now robust evidence favoring the positive effects of immunologically based defensive reactions that contribute to a preservation of the integrity of an implant's OI. The combined factor theory[1] summarizes the importance of treatment complications that may contribute to marginal bone loss, including implant hardware and surface, clinical handling, and individual patients' morphologic characteristics. A crucial consideration is that a combination of insults during the healing osseointegration stage (early occlusal overload and improper surgical protocol are the most likely harmful causes) will compromise the healing response of marginal bone. We cannot of course exclude the likelihood that bacteria can end up recruiting bone-resorbing cells in tandem with the aseptic loosening that can occur in earlier stages in the development of the induced OI interface. However, assigning an exclusive bacterial etiology for all forms of marginal bone loss around correctly placed implants is misleading. It is time for the dental profession to consider that so-called peri-implantitis is an operator-facilitated treatment outcome.

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