'A Requiem for the Periodontal Ligament' Revisited

The topic of loss of dental implant–harboring bone appears to be a never-ending story. Successful implant longevity was originally regarded as impossible, due to the inevitability of chronically inflamed host tissue responses, which threatened predictable anchorage. However, the advent of osseointegration protocols yielded such indisputably positive clinical long-term outcomes that a revised attitude to the technique quickly evolved. Colleagues in the periodontal discipline were quick to claim that published results depended on the different bacterial flora in treated edentulous patients. They readily opined that treatment of partially edentulous patients, particularly ones with a history of periodontal disease, would be vulnerable to periodontitis-like conditions and therefore likely to fail in great numbers. However, equally scrupulous documented clinical studies in such patients demonstrated comparable and even better outcomes than those available for the edentulous cohort—an observation that could not simply be ascribed to a shorter learning curve in the technique's application. It is therefore opportune for clinicians to debate the long-term effectiveness of implant placement efficacy in the context of the pathogenesis of the likely cause(s) of biologic failure.

In 1986, one of the authors of this commentary proposed criteria for successful implant treatment outcomes, which included the acceptance of an annual cervical bone loss of < 0.2 mm after the completion of the first year of treatment.[1] In an effort to provoke debate on the topic of treatment outcome determinants, the editorial "Osseointegration—A Requiem for the Periodontal Ligament" was published.[2] We underscored the fact that there is no reason to believe that bone tissue–anchored implants should behave entirely like periodontally anchored teeth, particularly in the way they fail biologically, given both their different genesis and resultant biology. Regrettably, the apparent logic of a different cause for implant failure seems to have fallen on deaf ears. In fact, we have recently seen an almost auction-like overbidding in reports of so-called peri-implantitis (PI) lesions around implants. This attitude is alarming since it fails to differentiate between primary PI as the reason for bone loss—likely to be irreversible and infrequently encountered—and the relatively benign gingival inflammation around an implant, or secondary PI—common, far from a cause for alarm, and accompanies circumimplant bone loss. Consider that one group of authors claims 6% of all implants are so diseased,[3] while another reports an incidence of 12%.[4] The first group then raised the bid, or rather widened the indications, to include 43% of all implants placed, followed by figures from the first group with 60% of all patients having at least one implant with peri-implantitis. All of this can of course be quite misleading, since it suggests an ominous prognosis for general implant outcomes; but this is clearly not the case. Numerous quiet roundtable discussions with clinical experts representing different disciplines seem to agree that it is in fact very uncommon for implant survival to be threatened by this sort of bone loss. Our impression is that most periodontists readily admit that circumimplant gingivitis is a very 'quiet' condition that is readily diagnosed and simply managed.

In scrutinizing the presented evidence for PI as the reason for whatever bone loss may be observed around implants (one commonly quoted definition of PI does include any bone loss, if only an accumulated amount of 0.1 mm between year 1 and year 20 of the implant's lifetime), we failed to find rigorous evidence. In fact, it seems like the most commonly cited evidence is based on placing ligatures around experimental implants and then studying the sequelae that inevitably follow this bold, if not somewhat extreme, invasive procedure. Chvartszaid et al[5] recently observed that "ligature studies do not accurately represent the etiology and progression of peri-implant bone destruction in humans, and their findings lack corroboration from human studies. As such, their scientific worth is questionable." Additional suggested evidence for PI is the finding of bone loss coupled to inflammation around retrieved oral implants; however, this observation in no way proves that the inflammation was the cause of the bone loss in the first place and that it may have actually been a secondary phenomenon. Yet, a third indication is the observation that patients with a previous history of periodontitis are likely to have an increased incidence of PI following implant treatment. This is far from rigorous evidence supporting the notion of an actual disease entity.

Consequently, we continue to find it scientifically difficult to embrace the notion of the near inevitability of PI as a disease entity. It is readily conceded that bone loss around implants does occur, and if not due to primary PI or normal remodeling of bone, it demands an answer to the question: Could there be other reasons for this bone loss? The most common alternative explanation in the literature is the notion of adverse loading or overloading. This hypothesis is reinforced by

observations made by prosthodontists that their first action when they see unacceptable bone loss around implants is to change fixed partial denture constructions to minimize the presumed strain around the implant with displayed bone loss. In fact, there are numerous anecdotal reports that in individual cases, such actions have proven successful with the bone resorption arrested. There are also a number of overload-confirming experimental studies[6–10] with most of the reported experimental data indicating that implant overload results in adverse bone loss or remodeling; and that it can be critically discussed in the same manner as we criticized ligature studies as supporting evidence for the PI theory. It must also be conceded that the applied loads in many experimental situations were far from being in the physiologic range, or that the animal models employed were often not even in the mouth but rather the dog or rabbit tibia.

We also hasten to assert that it is not the loading per se, but rather the strain in the bone around individual implants that matters. We also lack reliable clinical evidence that overloading alone must be the incriminating reason for every case of accidental bone loss around implants. Furthermore, the overloading theory is possible to debate within an orthopedic context. Orthopedic surgeons see accidental bone loss around hip and knee arthroplasties—in spite of the obvious lack of incriminating oral flora around hips and knees. We would suggest that few dentists would presume that such bone loss is due to a peri-implantitis type of reaction. The most common orthopedic explanation is that their implants suffer not from overload, but from stress shielding (ie, the implant takes the load and hence the bone is not needed according to Wolff's law). So, orthopedic surgeons believe in bone loss due to "underloading" of the bone rather than overloading of it, and therefore perhaps a term such as "adverse loading" could be agreed on by orthopedic surgeons and many prosthodontists as being the cause for the reported bone loss. However, we remain unconvinced of the evidence of adverse loading as the major incriminating factor for accidental bone loss as well. Having said this, too much strain around implants may indeed play a role in accidental bone loss. But another orthopedic explanation for bone loss is related to lack of blood supply to the bone due to injury from the associated implant surgery[11]—a theory that may be relevant to oral implants as well. But the observation of bone loss around implants is frequently reported and implant failures do occur both primarily as well as secondarily. We recently reported on the long-term fate of a specific implant system that was found with either implant failure or more than 3 mm of bone loss in one third of cases and at short follow-up times.[12–14] There is absolutely no reason to assume that these implant problems were in any way related to peri-implantitis. In our attempts to explain these implant problems, we incriminated not so much the implant per se, but rather an overtly aggressive treatment protocol that included grinding down the implant in situ and loading it directly. We coined the term "biologic challenge" for such tissue management mishaps.

The concept of such a biologic challenge is also an integral part of the theory of compromised healing/adaptation,[5] which can be applied to all sorts of adverse bone loss or failures irrespective of whether they are early or late occurrences. Thus, compromised healing may follow genetic disorders of the patient or poor bone quality for any other reason, including previous radiation or adversely traumatic surgical techniques. The sequelae of these various factors need not necessarily lead to implant failure, but if combined with implant loading the additional mobility of the implant in its site and the associated interfacial strain may combine to result in subsequent failure. Should the combined compromised/healing adaptation factors result in immediate failure, undue bone resorption may follow that will threaten the long-term survival of the implant. In this manner this new theory may explain early or late implant failures or early or late bone loss around an implant. As Chvartszaid et al[5] stated, "It is imperative that all possible factors that could influence the host-implant interface be viewed through a filter that asks the question: How will this factor influence the ability of the host, especially the response of the osseous tissues, to adapt to functional demands?"

A revisited requiem of the periodontal ligament demands a concluding observation on the saga of peri-implantitis. There is arguably no reason to doubt the occurrence of a secondary form of gingival inflammation around implants, which may even include minor degrees of time-dependent bone reduction. This is a local nuisance event and not a disease process. Like gingivitis, it is a relatively benign plaque-initiated response and in a long-term context, possibly modified by an initial minor disturbed healing/adaptation. It appears to respond readily to prudent and simple clinical interventions and should not be ascribed the misleading category of a disease process. However, the existence of PI as the single, primary

reason for bone and implant loss around implants is another matter altogether. This is a rare occurrence that is far more likely to result from additional insults to an initial compromise in the induced interfacial phenomenon of osseointegration. Its attempted management lacks rigorous protocols and is unlikely to result in a favorable, predictable outcome. The challenge clearly lies in the clinician's willingness (or lack thereof) to accept the fundamental differences between an evolved attachment mechanism for a tooth and an induced and controlled healing one for its analog.

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