On Inconvenient Truths

This Journal's mandate is to publish papers on the management of patients' oral rehabilitative needs. It also seeks to provoke debate and challenge popular convictions, especially cherished and convenient ones whose genesis is anecdotal and those that lack scientific rigor. I also believe that convenient dogmas that have characterized our discipline's development now need to be recognized as inconvenient truths. This is one more way to help the discipline evolve and render our clinical decisions even more viable and relevant.

The concept of the inviolability of the so-called "biologic width" around teeth has lingered for far too long. It has expanded recently to even include implants, in spite of the obvious differences in the nature of the osseointegrated entity of an implant and the periodontal attachment of a tooth. Its implications are intimidating rather than logical, especially since its scientific validity is far from convincing—hence this effort to stimulate a "rethink" of the merits of periodontist-driven formulaic guides to the placement of crown margins.

The term "biologic width" presumes that the dimensions of the connective tissue barrier between sulci and bone around teeth or implants are specific, scientifically robust, and unchangeable. The associated dimensions, although acknowledged as averages, are quoted to the hundredths of a millimeter (2.04 mm), giving the impression that the range of variability is less than a tenth of a millimeter. Moreover, dentists are taught that "the science" on the subject is settled and that biologic width is indeed a reality.

The Science Behind Biologic Width

Data from the original paper by Gargiulo et al[1] was used as the basis for the introduction of the notion of biologic width. The reported findings were gleaned from 30 human cadaver jaws, with an age range of 19 to 50 years, and 287 teeth, of which 325 surfaces were examined histologically. The authors did not explain why only 28% of surfaces (325 of a possible 1,148) were examined or how these surfaces were selected. Tooth exposure was classified into four phases associated with active and passive eruption, and the periodontal status was not documented. The tissues were only described as being free of extensive pathology, and there was no assessment of the effect of specimen preparation.

The mean measurements of the epithelial and connective tissue attachments (EA and CTA)—combined into a biologic width—represented the means of several measurements for a given tooth surface: the means of all four tooth surfaces and all teeth in a given phase of eruption together with the means of all four eruption phases. In other words, these mean measurements of "biologic width" represented the means of the means of the means of the various measurements! Furthermore, EA ranged from 0.08 to 3.72 mm and CTA from 0.00 to 6.62 mm.

In a subsequent paper, Gargiulo et al[2] expressed concern that the mean values often quoted from the original autopsy study did not truly reflect the variability that exists in the dimensions of the dentogingival junction. They further stated that biologic width measurements obtained from the tissues of healthy periodontium should not be extrapolated for use in pathologic situations. It is of course recognized that a layer of connective tissue exists between the junctional epithelium and the alveolar bone crest around teeth and implants. It is also believed that these tissues provide a barrier against the penetration of bacteria into the underlying bone. However, the inconvenient truth is that neither these tissues' specific dimensions nor their time-dependent stability or behavioral response to diverse ecologic changes have been determined scientifically.

The Relationship Between Restoration Margins and Biologic Width

It has been known for over 50 years that plaque accumulation, facilitated by rough surfaces, is the cause for gingival inflammation around subgingivally placed restoration margins. As early as 1956, Waerhaug[3] demonstrated that roughening of tooth surfaces enhances plaque accumulation, while Löe[4] concluded that rough surfaces and ill-fitting margins, which facilitated plaque retention, explained the progressive destruction of the periodontium adjacent to fillings or crowns. Quite unsurprisingly, the elimination of plaque results in a healthy gingival response irrespective of margin form or position; however, the techniques, time, and dexterity involved in maintaining a plaque-free environment around deep, rough, and ill-fitting margins may preclude maintenance of long-term tissue health. Nonetheless, "violation" of some specific dimension of the biologic width or even allergic responses to materials continues to be cited as the causative factor for this poor gingival response, albeit without rigorous scientific evidence.

Silness[5] summarized the results of numerous studies and described the tooth transition zone roughness

resulting from several contributing factors, including the restorative material used, the prepared tooth surface, and the marginal discrepancy. He too claimed that it was this roughness that facilitated plaque retention and resulted in a poor tissue response. He also asserted that the deeper the margins, the more difficult it was to obtain a physiologic tooth-restoration transition zone. A more recent review by Padbury et al[6] focused on the interactions between the dentogingival complex and the margins of restorations. It was claimed that although there was confusion regarding the relevance of the "biologic width," many studies had resulted in general agreement that placement of restoration margins within the "biologic width" frequently led to gingivitis, attachment loss, and bone loss. However, it was conceded by these authors that recommendations regarding placement of margins in relation to the biologic width were based on "opinion articles."

It has also been reported that an individual's immune system[7,8] and hormone levels[9,10] modify the severity of the inflammatory response to bacterial plaque, and that specific bacteria in plaque can elicit differing degrees of inflammation.[11] It is likely that these factors would also influence the severity of the inflammatory response to a less-than-ideal tooth-restoration transition zone. They would also explain the varied responses observed clinically and the frequently repeated, if anecdotal, observation that the response is more severe in females.

Violation of the Biologic Width

It is the inadequacies of the tooth transition zone rather than depth of margin placement that cause tissue inflammation, and it is indeed possible to place restoration margins subgingivally (> 1 mm) without usurping the traditional notion of a biologic width. In other words, the biologic width can be physiologically disregarded or "disturbed." Moreover, extensive professional experience with dental implants has actually led to a consensus conference that, for esthetic reasons, deemed it appropriate to locate the implant-abutment junction submucosally up to several millimeters.[12] It was concluded that with appropriate oral hygiene, the intracrevicular position of the restoration did not appear to adversely affect peri-implant mucosal health or stability. This suggests that the commonly stated dimension associated with the presumed biologic width around implants can essentially be ignored. The lack of observed peri-implant mucosal pathology in these situations was attributed to the smooth implant component surfaces and the "rotation symmetric" design. This was contrasted with the scalloped cementoenamel junction of teeth. The apparent advantage of the implant-abutment rotation symmetric design is that a machine fit of flat surfaces with negligible marginal discrepancies is possible, even if minor rotational misplacements occur. It is also interesting to note that a study on 13 different implant-abutment combinations showed all systems had horizontal marginal discrepancies of < 10 µm.[13] Indeed, with modified surfaces of abutments and implant-abutment discrepancies of < 2 µm, bone formed above the implant-abutment junction in 40% of specimens.[14]

Therefore, it appears that with minimal implant-restoration transition roughness, peri-implant mucosal health can be maintained irrespective of the depth of margin placement. Transition roughness caused by misfit, excessive roughness of components, or presence of cement can logically result in peri-implant mucosal pathology. It seems reasonable to suggest that if the equivalent of a machined fit could be achieved at the tooth-restoration transition, a similar predictable physiologic response should ensue.

Restoration Margin Placement

The inconvenient truth is that poor restorative techniques, especially when coupled with inappropriate material choices, facilitate plaque accumulation around restoration margins. This causes gingival and periodontal pathology and associated unesthetic changes to any gingival profile, especially interproximally. One study showed up to 89% of tooth-supported fixed dental prosthesis (TFDP) impressions sent to dental laboratories had one or more detectable errors,[15] and this is just one of the many procedures involved in tooth-supported single crown and TFDP retainer fabrication. In another study, examination without magnification of bitewing radiographs showed that up to 75% of restoration margins had interproximal misfits.[16]

Marginal distortions of 10 to 50 µm were previously considered to have important biologic implications.[17] Currently, marginal misfits of 80 to 120 µm are considered the standard of clinical acceptability. Unfortunately, this derives from an assumption, once again without any scientific evaluation, that what occurs clinically with a given technique is biologically acceptable.[18] This "standard" has been applied to newer all-ceramic[19] and CAD/CAM[20] techniques, and these misfits are mostly "bogged-up" with resin cements. Complete removal of excess adhesive cement around subgingival margins is extremely difficult, if not impossible. Exceedingly rough transition zones will inevitably occur with resultant plaque accumulation and tissue inflammation.

The inherent risks of iatrogenic sequelae associated with current practice protocols would endorse the prudence of supragingival or minimally penetrating (< 0.5 mm) restorative margins. However, the inconvenient truth must be asserted that such advocacy must not be attributed to a pseudoscientific rationale of "violation" of specific dimensions of a so-called biologic width. Clinical and laboratory researchers must continue to look forward to the development of techniques and materials that inhibit plaque accumulation, promote epithelial adhesion and connective tissue attachment, as well as minimize changes to gingival profiles whenever subgingival restoration margins (tooth- or implant-associated) are placed.

A Suggested Name Change to "Biologic Barrier"

The term "biologic width" is regrettably perceived to bestow specific numeric dimensions to a unique and dynamic biologic entity. It must be recognized, however, that in any individual, EA and CTA "measurements" will change with time and in response to variations in the local and systemic environment. Given this vast variability, it is inappropriate and misleading to constrain discussion of margin placement to mathematically derived "averages," since another inconvenient truth is that reported averages tend first to become absolutes and, eventually, dogmas. These observations underscore the need for a name, or at least an emphasis, change—one that reflects the dynamic and mutable nature of the supra-alveolar connective tissue and one that does not imply that we dentists are confronted with an operating domain that is inviolable. We are, after all, dealing with a biologic barrier, no more and no less, and calling it so should help clarify one more of the inconvenient truths that we have inadvertently burdened ourselves with.

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References

1. Gargiulo AW, Wentz FM, Orban B. Dimensions and relations of the dentogingival junction in humans. J Periodontol 1961; 32:261–267
2. Gargiulo AW, Krajewski J, Gargiulo M. Defining biologic width in crown lengthening. CDS Rev 1995;88:20–23.
3. Waerhaug J. Effect of rough surfaces upon gingival tissue. J Dent Res 1956;35:323–325.
4. Löe H. Reactions of marginal periodontal tissues to restorative procedures. Int Dent J 1968;18:759–778.
5. Silness J. Placement of margins. In: Zarb GA, Bergmann B, Clayton JA, MacKay HF. Prosthodontic Treatment for Partially Edentulous Patients. St Louis: Mosby, 1978:333–344.
6. Padbury A Jr, Eber R, Wang HL. Interactions between the gingiva and the margin of restorations. J Clin Periodontol 2003;30: 379–385.
7. Okada M, Kobayashi M, Hino T, Kurihara H, Miura K. Clinical periodontal findings and microflora profiles in children with chronic neutropenia under supervised oral hygiene. J Periodontol 2001;72:945–952.
8. Genco RJ. Current view of risk factors for periodontal diseases. J Periodontal 1996;67(suppl):1041–1049.
9. Machtei EE, Mahler D, Sanduri H, Peled M. The effect of menstrual cycle on periodontal health. J Periodontol 2004;75: 408–412.
10. Carillo-de-Albornoz A, Figuero E, Herrera D, Bascones-Martínez A. Gingival changes during pregnancy: II. Influence of hormonal variations on the subgingival biofilm. J Clin Periodontol 2010;37:230–240.
11. Saito A, Inagaki S, Ishihara K. Differential ability of periodontopathic bacteria to modulate invasion of human gingival epithelail cells by Porphyromonas gingivalis. Microb Pathog 2009; 47:329–333.
12. Giannopoulou C, Bernard JP, Buser D, Carrel A, Belser UC Effect of intracrevicular restoration margins on peri-implant health: Clinical, biochemical, and microbiologic findings around esthetic implants up to 9 years. Int J Oral Maxillofac Implants 2003:18:173–181.
13. Jansen VK, Conrads G, Richter EJ. Microbial leakage and marginal fit of the implant-abutment interface. Int J Oral Maxillfac Implants 1997;12:527–540 [erratum 1997;12:709].
14. Welander M, Abrahamsson I, Berglundh T. Subcrestal placement of two-part implants. Clin Oral Implants Res 2009;20:226–231.
15. Samut N, Shohat M, Livny A, Weiss EI. A clinical evaluation of fixed partial denture impressions. J Prosthet Dent 2005;94: 112–117.
16. Björn AL, Björn H, Grkovic B. Marginal fit of restorations and its relation to periodontal bone levels 11. Crowns. Odontol Revy 1970;21:337–346.
17. Shillingberg HT, Hobo S, Whitsett LD, Jacobi R, Brackett SE. Fundamentals of Fixed Prosthodontics, ed 3. Chicago: Quintessence, 1997.
18. McLean JW, von Fraunhofer JA. The estimation of cement film thickness by an in vivo technique. Brit Dent J 1971;131:107–111.
19. Goldin EB, Boyd NW 3rd, Goldstein GR, Hittelman EL, Thompson VP. Marginal fit of leucite-glass pressable ceramic restorations and ceramic-pressed-to-metal restorations. J Prosthet Dent 2005;93:143–147.
20. Boening KW, Wolf BH, Schmidt AE, Kästner K, Walter MH. Clinical fit of Procera AllCeram crowns. J Prosthet Dent 2000;84: 419–424.