Review Article

Biological Width: The Periodontal Restorative Interlink: Short Discussion

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ABSTRACT

Biological width is the natural distance (combine heights) between the base of the healthy gingival sulcus OR epithelial attachment to the tooth and the height of the alveolar bone or connective tissue. For better description of relationship between the periodontal tissues and conservative procedures is to restore form, function, esthetics, and comfort for the dentition. Most of the dentists are aware of biological width, its maintenance and its importance during application of crown lengthening. Relevant publications regarding biologic width, its violation and management were identified up to August 2011 using manual and electronic database search in Medline, Embase, Directory of Open Access Journals, and Google Scholar.

Key words: Crown lengthening, biological width, gingival sulcus

INTRODUCTION

Biological width is the natural distance (combine heights) between the base of the healthy gingival sulcus OR epithelial attachment to the tooth and the height of the alveolar bone or connective tissue. The combined height or dimension widely known as biological width was initially described by Garguilo *et al.*[1] in a literature published in 1961. The dimensions were as follows:

- Attached connective tissue height - 1 mm (1.07 mm)
- Epithelial attachment - 1 mm (0.97 mm)
- Combined = 2 or 2.04 mm
- Sulcus Depth - 1 mm.

Anatomically the biological width follows the osseous scallop which is parallel to cementoenamel junction circumferentially. Clinical crown of the tooth is the distance from the gingival margin to incisal edge or occlusal surface of the tooth. This distance should be increased when:

- Margins of caries lesion are subgingivally
- Margins of the tooth crown fractures are subgingivally
- Tooth crown is too short for retention of restoration
- There is an excess of the gingiva and anatomical tooth crown is opened partially.

When this biologic width is violated by a restoration as a defense mechanism, inflammatory response accelerates bone loss to provide space for new connective tissue attachment, which results in increased pocket depth.[1] Therefore, impingement of a restoration on the biologic width will trigger loss of bone, connective tissue, and epithelial attachment.[2]

The significance of biological width is not only limited to restorative purposes but also it acts as a barrier and prevents penetration of micro-organisms in the periodontium.

Vacek *et al.*[3] reported in their study the revised range of this zone as 0.75-4.33 mm. It was observed that of all the tissue dimensions measured, the length of connective tissue attachment varied the least.

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The proceedings of the 3rd European workshop on periodontology and implant dentistry state that the function of peri-implant seal is to maintain homeostasis of the internal environment in response to challenges from the external environment.

HISTORICAL BACKGROUNDS

Garguilo *et al*.[1] did not use the term biological width in their published literature. D. Walter Cohen first coined the term biological width in the year 1962 from University of Pennsylvania.

In 1994, Vacek *et al*.[3] did details studies on cadaver on biological width to get the details clinical findings about the biological width and its dimensions. After his study, he concluded that biological width was almost similar in all the teeth in the same individual from anterior central incisors to posterior molars.

There are two aspects to the crown lengthening procedure: Esthetic and function. In both the cases goal of the surgical procedure is to re-establish the biological width apically while exposing more tooth structure. It is very well-known that biological width is defined as the sum of the junctional epithelium and the supracrestal connective tissue attachment.

Factors Affecting the Crestal Bone Loss[4,5]

Biologic width/seal

Biologic width forms within the first 6 weeks after the implant/abutment junction has been exposed to the oral cavity. It is a barrier against bacterial invasion and food ingress at the implant-tissue interface. The ultimate location of epithelial attachment following stage 2 surgery in part determines early postsurgical bone loss. Thus, implant bone loss is in part a process of establishing the biologic seal.

Surgical trauma

Surgical trauma due to heat generated during drilling elevation of the periosteal flap and excessive pressure at the crestal region during implant placement may contribute to implant bone loss during the healing period. Wilderman *et al*.[6] reported that bone loss due to periosteal elevation was restricted to the area just adjacent to the implant, even though a larger surface area of the bone was exposed during surgery. Early implant bone loss is in the form of horizontal saucerization. However, bone loss after osseous surgery in natural teeth is more vertical. Signs of bone loss from surgical trauma and periosteal reflection are not commonly observed at the implant stage 2 surgery in successfully osseointegrated implants. Thus, surgical trauma is unlikely to cause early crestal bone loss.

Microgap

In most of the two-stage implant systems, after abutment is connected, a microgap exists between the implant and the abutment at or below the alveolar crest. For all two-stage implants, the crestal bone levels are dependent on the location of the microgap ~2 mm below it. The countersinking below the crest is done to minimize the risk of implant interface movement during bone remodeling, to prevent implant exposure during healing, and also to enhance the emergence profile. Countersinking places the implant microgap below the crestal bone. The microgap-crestal bone level relationship was studied radiographically by Hermann *et al*.[7,8] who for the first time, demonstrated that the microgap between the implant/abutment has a direct effect on crestal bone loss, independent of surgical approaches. Epithelial proliferation to establish biologic width could be responsible for crestal bone loss found about 2 mm below the microgap.

Occlusal overload

Excessive stress on the immature implant-bone interface in the early stage of prosthesis in function is likely to cause crestal bone loss. Cortical bone is least resistant to shear force, which is significantly increased in bending overload. However, bone loss from occlusal overload is considered to be progressive rather than limited to the first year of loading.[9]

Crest module

The transosteal region of the implant receives crestal stresses after loading. The crest module design can transmit different types of forces onto the bone, which depends on its surface texture and shape. A polished collar and a straight crest module design transmit shear force, whereas a rough surface with an angled collar transmits beneficial compressive force to the bone.[10]

Evidence-based Review of the Biologic Width Around Implants

Important gray areas of concern

- What is the structure of the biologic width around implants?
- What is the function of the biologic width?
- What is the influence of the mucosal thickness on the biologic width?
- Does abutment connection/disconnection have influence on biologic width?
- What is the effect of macrostructure of the neck of the implant?

Structure of Biologic width Around Implant

Glauser *et al*.[11] in their study on one-piece mini implants calculated the mean dimensions as 4-4.5 mm. Kan *et al*.[12]

in a study on anterior implants after bone sounding on the specific sites calculated a mean dimension of 6.17 mm on mesial, 3.63 mm at midfacial, and 5.93 mm at distal sites of implants. Epithelium around two-piece implants was always located apical to the microgap.[13]

A biologic width dimension around two-piece implants is larger than that of one-piece implants and natural teeth. The presence of microgap and its location influences the marginal bone levels and the biologic width of the surrounding soft tissue. Hermann *et al*.[7] evaluated the changes over time and determined that the connective tissue around implants are more stable than the epithelial dimension, as evident around natural teeth. The biologic width did not vary significantly regardless of whether the implant was loaded (with restoration) for a short or long time. This suggests the formation of biologic width is a physiologic response in the oral cavity and is not dependent on the presence or absence of loading or the length of loading time.[14] Connective tissue dimensions being more stable around one-piece implants and natural teeth relate to the fact that once formed, they are predominated by protein collagen, and as collagen matures, more cross-linkages occur which stabilize this tissue and make it more resistant to dimensional change over time. Junctional epithelium, however, is constantly being challenged by microbial growth and pathologic microbial products. Biologic width once invaded around the implant undergoes similar structural and histologic changes as evident around the tooth, independent of tissue biotype (thick/thin).[15]

Function of Biologic Width

Biologic width serves as protective mechanism for underlying bone. The function of junctional epithelium was investigated by Sanz[9] in a comparative histologic study of healthy and infected implant sites, revealing high transmigration of inflammatory cells in sulcular epithelium of infected sites. A case control study showed significant increase of T-lymphocytes in sulcular epithelium in peri-implantitis human biopsies when compared with healthy peri-implant tissue.[16] Chavrier in his histologic biopsy study on the connective tissue around implants revealed predominance of type 1 collagen fiber.[17]

Some animal studies revealed migration of leukocytes through junctional epithelium toward bacterial plaque. Accumulation of these cells in the presence of infection may demonstrate the possible defense mechanism of biologic width.[17,18] The evidence of protective peri-implant seal abilities may be found in the peri-implantitis models in animal studies which confirm that combination of plaque accumulation and biologic width injury can result in crestal bone loss around implants.

DISCUSSION

The relationship between the health of the periodontium and the placement of the restorative margins are closely interlinked. Even though a great emphasis has been focused on the perio-restorative interface in restorative dentistry; many clinicians still fail to utilize the concept of biologic width in a clinical practice. The importance of biologic width to surgery relates to its reformation following surgical intervention. Research shows it will reform through coronal migration of the gingiva to recreate not just the biologic width, but also a sulcus of normal depth. This means if the surgery does not consider the dimensions of biologic width when placing the gingiva relative to the underlying bone, the gingival position won't be stable but instead will migrate in a coronal direction.

The primary significance of biologic width to the clinician is its importance relative to the position of restorative margins, and its impact on postsurgical tissue position. We know that if a restorative margin is placed too deep below tissue so that it invades the biologic width, two possible outcomes may occur. One, there may be bone resorption that recreates space for the biologic width to attach normally, this is the typical response seen in implants to allow the formation of a biologic width, the so-called funnel of bone loss to the first thread.

An accurate diagnostic and interdisciplinary approach is necessary for obtaining improved, conservative and predictable results in esthetically compromised areas, like the anterior maxillary dentition. Periodontal health is of paramount importance for all teeth, both sound and restored. Thus, this procedure, as an adjunct to a restorative treatment, can produce predictable results while ensuring good esthetics and maintaining periodontal health.[8]

There are two aspects to the crown lengthening procedure: Esthetic and functional. In both cases, the surgical procedure is aimed at re-establishing the biological width, apically while exposing more tooth structure. Biological width is the sum of the junctional epithelium and supracrestal connective tissue attachment. The average space occupied by the sum of the junctional epithelium and the supracrestal connective tissue fibers was found to be 2.04 mm. Violation of biological width has been associated with gingival inflammation, discomfort, gingival recession, alveolar bone loss, and pocket.[2]

To have a harmonious and successful long-term restoration, a 3 mm sound supracrestal tooth structure between bone and prosthetic margins, which allows for the reformation of the biological width plus sulcus depth is advocated. This can be achieved surgically by crown lengthening, orthodontically by forced tooth eruption or by a combination of both.[9]

The amount of tooth structure that is exposed above the osseous crest must be above 4 mm which is enough to provide for a stable dentogingival complex and biological width to permit proper tooth preparation and account for an adequate margin placement.[10] It was found that margins of fixed prosthesis significantly compromise the gingival health if placed below the gingival margin.[11]

After the procedure, it is customary to wait for 6-8 weeks before cementing the final restoration. This reduces chances of gingival recession after prosthetic crown insertion.[12] After a 2-3 weeks postsurgery period, temporary crowns may be used until there has been full healing and the gingival margin is in a stable position.[13]

Patients that require esthetic crown lengthening, however, frequently exhibit a high smile line. As a result, pressure is often placed on the restorative dentist to correct esthetic deficiencies as early as possible and maintain certain esthetic standards.

In a study, 66 anterior crowns with subgingival margins of varying depths were analyzed by Newcomb (1974) and compared them to uncrowned contra lateral controls. The results suggested that the severity of the gingival inflammation was likely to be directly related to how near the subgingival crown margin was placed to the epithelial attachment.

Günay *et al*. (2000)[17] showed how margins of restorations in area of gingival biological width cause pathology of periodontium. It was evaluated 116 restored and 82 healthy teeth of 41 patients. After 2 years results showed formation of periodontal pockets and increased index of gingival bleeding in the areas with distance less than 1mm from restoration margins to alveolar bone.

Before tooth restoration, the gingiva should be healthy without any sign of inflammation. Then, these rules could be followed:[16]

- If gingival sulcus is 1.5 mm or less, then, margins of restoration are prepared to 0.5 mm subgingivally;
- If gingival sulcus is 1.5 - 2 mm, then, margins of restoration are prepared to 0.7 mm subgingivally;
- If gingival sulcus more than 2 mm, especially in esthetical area from vestibular side then gingivectomy is recommended, and margins of restoration are prepared to 0.5 mm subgingivally.

The gingivectomy is recommended because the deeper gingival sulcus is less predictable in terms of stability of marginal gingiva and thus gingival retraction can can be more severe in gingivectomy procedure. Attempts to prepare margins of restoration as deep as possible subgingivally and thinking, if gingival retraction would happen, margins of restoration still stay subgingivally, could give contrary results - gingival retraction, open margins of restoration or periodontal pocket. It is known, that periodontioum could clean the area of restoration and step margin himself, when it is subgingivally not more than 0.7 mm.

CONCLUSION

After reviewing, these literatures finally concluded that biological width is a very important in not only restorative and endodontics procedures but for a health periodontium also which will keep the teeth health. Proper maintenance and cleaning of periodontium will help to preserve good biological width with preservation of a health periodontia and will also be helpful in removing the irritation or foreign substance which may damage the periodontia which can further lead to damage of biological width.

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