The 2B-3D rule for implant planning, placement and restoration

1. What is biologic width?

Is there a golden rule for implant planning, placement and restoration as the Newton's laws of motion for force prediction? In order to answer this question, one needs to refer back to the biologic system which the implant site attempts to mimic.

In the human body, ectodermal tissue serves to protect against invasion from bacteria and other foreign materials. However, both teeth and dental implants must penetrate this defensive barrier. The natural seal that develops around both and protects the alveolar bone from infection and disease, is known as the biologic width.[1] Around natural teeth, the biologic width has been shown to consist of approximately 1mm sulcular depth, 1mm junctional epithelium, and 1mm connective tissue attachment (*Fig. 1*).[2,3]

To summarize then, the biologic width is equal to 3mm: 1mm sulcular depth, 1mm junctional epithelium and 1mm connective tissue attachment above the crestal bone. This is true on the broad facial surface. In the proximal papillae area, the correct biologic width increases to 4mm.[4,5] This can be measured on any tooth using the "sounding" technique.

This "sounding" technique of the crestal bone is not routinely practiced by most clinicians. However, for anterior esthetic cases where the margin is desired to remain subgingival, this "sounding" procedure will ensure its long term stability and esthetics.

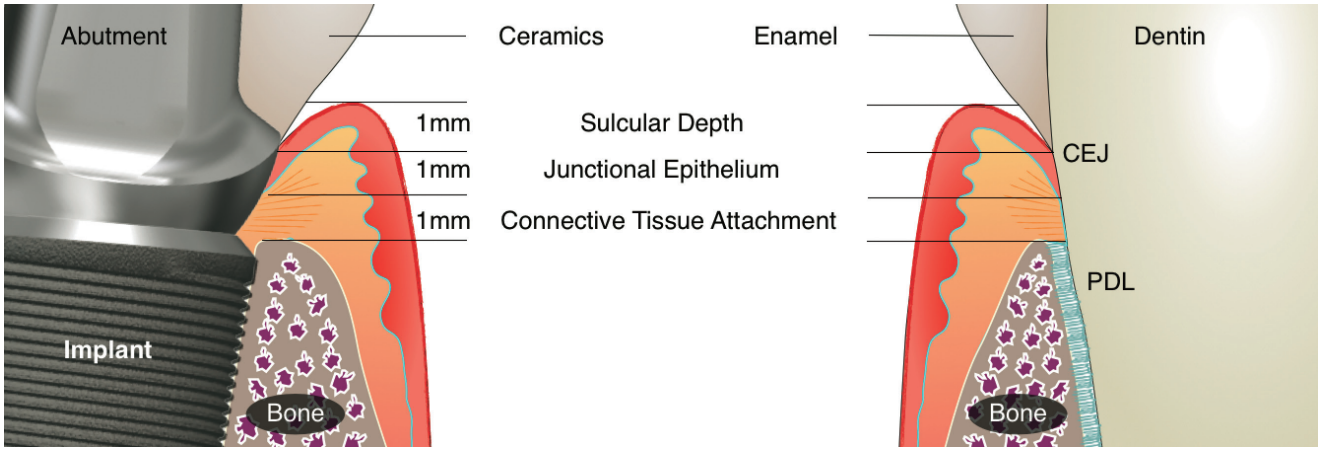

Fig. 1:

The biologic width is equal to 3mm: 1mm sulcular depth, 1mm juntional epithelium and 1mm connective tissue attachment above the crestal bone. As a general rule, the implant head should be placed 3mm apical to the future labial gingival margin position in order to allow development of the desired emergence profile, esthetics, and biologic width.

Chris Chang, DDS, PhD.
- *Founder, Beethoven Orthodontic Center*
- *Publisher, International Journal of Orthodontics & Implantology*

The "Sounding" Procedures:

First, anesthetize the area to be sounded. Second, use a narrow tipped periodontal probe, place it in the sulcus and lean it away from the tooth while keeping the tip against the enamel. Third, push through the attachment apparatus until the crest of bone is felt.[2,3] Finally, record three measurements per facial tooth surface.

One should be aware that the crest of bone follows the scallop of the cemento-enamal junction (*CEJ*) but DOES NOT always follow that of the gingival margin. Based on these measurements of the teeth to be restored (*proximals and center of facial*), one can predict how the tissue will respond post-cementation of the new prostheses.[3,4] The goal is to keep the prosthesis margin within the sulcular depth without interfering with the junctional epithelium and connective tissue attachment. [4,5]

2. Does an implant need this defense barrier-biologic width?

If a tooth needs a defense barrier to protect its supporting alveolar bone, it is reasonable to assume the same for an implant. Based on the study of Berglundh T, et al.,[6] the biologic width that develops around implants at the time of abutment connection has been shown to incorporate tissue zones of similar dimensions which is 1mm sulcular depth, 1mm junctional epithelium, and 1mm connective tissue attachment with insufficient principle fibers. This concept of biologic width around implants has been further investigated by Hermann JS, et al.[7] This group evaluated the impact of the position of the implant-abutment interface relative to the crestal bone and periimplant tissues. The investigation indicated that the biologic width around implants differed according to the depth and position of the interface. When the implant-abutment connection was placed at the gingival level, supracrestal to the alveolar bone (*i.e., as in a conventional single-stage implant placement*),[8] the biologic width was similar to that of natural dentition. When the interface was placed at a deeper level (*i.e., as in a standard submerged implant design*),[8] however, the biologic width increased accordingly. The primary difference was found in the depth of the junctional epithelium height, which extended just apical to the interface. The sulcus depth and connective tissue attachment width appeared stable regardless of the level of interface. It was, therefore, determined that implant placement with the implant-abutment interface placed supracrestal to the bone facilitated maintenance of the biologic width with minimal apical bone resorption.[9-11]

In the esthetic area, however, the prosthesis margin should always be placed subgingivally, regardless of whether the implant fixture is a one- or two-stage

design. As a general rule, the implant head should be placed 3mm apical to the future labial gingival margin position in order to allow development of the desired emergence profile and esthetics.[12-14] More importantly, this rule of 3mm depth from the future labial gingival margin is based on the biologic width which develops around the implant. With 3mm in depth from gingival margin, a defense barrier can form and further protect the alveolar bone around the implant which mimics natural dentition.[14]

3. Does buccal bone thickness affect biologic width?

The answer is YES. Buccal bone thickness and biologic width are inter-related. According to the long-term clinical study by Grunder U, et al.,[12-14] they concluded that to achieve a stable, optimal esthetic result with implants, given the anticipation of the circumferential bone resorption around the implant heads,[15] the thickness of the bone on the buccal side of an implant should be at least 2mm.[14] When the bone is found to be insufficient, a bone augmentation will be performed on the buccal side. For a papilla between two adjacent implants to be established, the inter-implant distance has to be more than 3mm. The study further suggests that additional bone on the buccal side of the papilla is required in order to prevent black triangle.[14] Grunder's conclusion[12-14] is based on the assumption of the inevitable occurrence of circumferential bone resorption around implant heads. Tarnow el al.,[15] proved that a certain amount of bone resorption occurred around implants as soon as the implant was placed. On average, the first bone to implant contact is about 1.5 to 2mm below the implant shoulder shortly after implant exposure.[16] This bone resorption occurs not only in a vertical but also in a horizontal direction.[14,16]

Can modern implant design[17,18] prevent circumferential bone resorption around implant heads? One promising solution involves the concept of platform switching.[18] This is based on the observation that, when the interface between the implant shoulder and abutment is moved horizontally away from the bone, bone resorption does not occur. This might be the result of distancing the contaminated microgap[19] away from the bone. To take a step further, the current morse taper design of abutment with a conical seal can dramatically reduce or eliminate this contaminated microgap.[20] Without the contaminated microgap, infection due to the pumping effect of the microgap and the consequential bone resorption can be avoided. Therefore, the crest bone can be preserved. In terms of bone preservation and preventing gingiva recession, abutment with the capacity of platform switching and a conical seal seems to be the answer. However, clinical observation shows that the labial gingiva recession will occur regardless the type of implants used if the buccal bone thickness is insufficient.[14] This begs another question: what make the existing buccal bone stable? For example, gingiva recession is rare in natural dentitions even when the buccal bone thickness is less than 1mm.[21] However, it is a common occurrence in implant sites where buccal bone is thin. Why? The reason may be due to the loss of supporting system or structure, i.e., periodontal ligament (*PDL*). Without PDL, the

buccal bone resorption will occur. This condition has been well documented by Araújo and Lindhe.[22] Beside the structure change, the nurturing system, i.e., vascular supply, is also modified.[23-25] Makigusa [24,25] had illustrated morphologic differences in the distribution of the vascular network around marginal gingiva between implant sites and natural dentitions as the following.

The origins of these blood supply routes[25] in marginal gingiva can be described as: (*1*) from the periodontal ligament to the connective tissue, (*2*) from the cancellous bone to the periodontal ligament and then to the connective tissue, (*3*) from the cortical bone directly to the connective tissue, (*4*) from the apical mucosa directly to the marginal gingiva (*Fig. 2*).

When implants replace lost teeth, and a new biologic width develops after connecting conventional two-stage implants to abutments, the overall blood supply to the gingival connective tissue is reduced, due to the lack of a periodontal ligament. Clinicians should take this into consideration when planning for implant placement, particularly in the esthetic zone, where buccal gingival tissue recession is common.[26,27] The reduction in blood supply, first after extraction and then after implant placement, may lead to this loss of soft-tissue volume and prompt implant and/or abutment exposure. Thus, evaluation of the patient's tissue biotype and bone thickness should be performed during treatment planning, with anticipations for the clinical outcome adjusted accordingly. The thicker the native hard and soft tissue are, the more robust the blood supply can be expected after implant placement, with enhanced expectations for esthetic success.

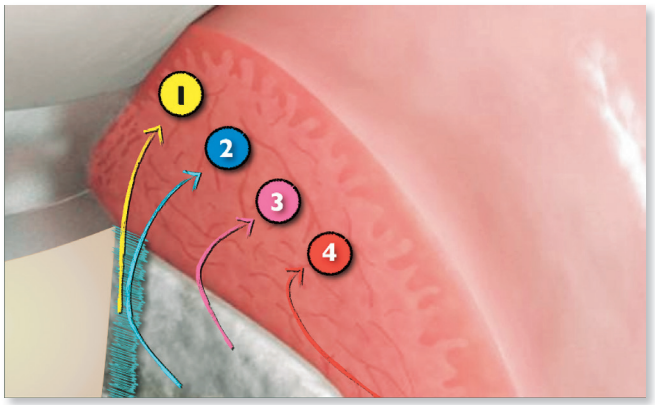

Fig. 2:

The origins of these blood supply routes in marginal gingiva are as follows:

(1) from the periodontal ligament to the connective tissue,

(2) from the cancellous bone to the periodontal ligament and then to the connective tissue,

(3) from the cortical bone directly to the connective tissue, and

(4) from the apical mucosa directly to the marginal gingiva.

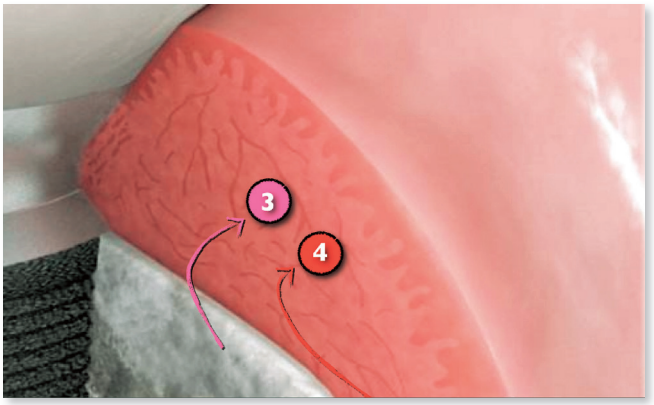

Fig. 3:

After losing the periodontal ligament, blood supply around dental implants is less than that around natural dentition due to the loss of first route and second route of blood supply. This may be the reason why Grunder consistently found that ***2mm of buccal bone thickness*** *could prevent gingiva recession. Because we can get a broader area of blood supply.*

After losing the periodontal ligament, blood supply around dental implants (*Fig. 3*) is reduced due to the loss of first and second route[25] as well as the result of a dynamic process of bone remodeling. After implant placement, the biologic width must be reestablished. As this occurs, circumferential bone loss typically takes place around the implant's coronal aspect up to the first implant thread. Also, resorption in a palatal direction following tooth loss leads to ridge thinning. The thin bone remaining on the facial aspect of the implant tends to be cortical, with significantly reduced vascularity. Furthermore, in a thin ridge, there is rapid drop off (*sloping*) of the buccal aspect of the crest, resulting in more of the blood supply being positioned apically, where the bone crest is wider and more cancellous. This may be the reason why Grunder[14] consistently asserts that 2mm buccal bone thickness proves to be advantageous for preventing gingiva recession for the broader area of blood supply.[25]

4. Conclusion: The 2B-3D rule

Based on the biologic evidence [28,29] discussed above, implants should be placed with 2mm buccal bone thickness and 3mm in depth from future prostheses margin to ensure the stability of implant restoration. In short, the author summarizes the above principle as the 2B-3D rule for ideal implant placement. What is the 2B-3D rule? 2mm of buccal bone thickness should be preserved before placing an implant 3mm in depth from the future prosthesis cervical margin. This 2B-3D rule is a practical guide, both for single implants or full mouth rehabilitation,[30] to achieve ideal implant positions. When these conditions could not be satisfied at the time of implant placement, bone augumentation, bone reduction, lingually positioning implant or smaller diameter of implant should be considered to ensure long-term stability of both hard and soft tissues.[14,30]

REFERENCES

1. Gargiulo AW, Wentz FM, Orban B. Dimensions and relations of the dentogingival junction in humans. J Periodontol 1961;32:261-267.
2. Kois JC. The restorative-periodontal interface: Biological parameters. Periodontol 2000 1996;11:29-38.
3. Van der Velden U. Regeneration of the interdental soft tissue following denudation procedure. J Clin Periodontol 1982;9:455–495.
4. Kan JYK, Rungcharassaeng K, Umezu K, Kois J. Dimensions of peri-implant mucosa: An evaluation of maxillary anterior single implants in humans. J Periodontol 2003;74:557–562.
5. Salama H, Salama MA, Garber D, Adar P. The interproximal height of bone: A guidepost to predictable aesthetic strategies and soft tissue contours in anterior tooth replacement. Pract Periodontics Aesthet Dent 1998;10:1131–1141.
6. Berglundh T, Lindhe J, Ericson et al. The soft tissue barrier at implants and teeth. J Clin Oral Implants Res 1991;2:81-90.
7. Hermann JS, Buser D, Schenik RK, Higginbottom FL, Cochran DL. Biologic width around titanium implants. A physiologically formed and stable dimension oven time. Clin Oral Implants Res 2000;11:1-11.
8. Ericsson I, Nilner K, Klinge B et al. Radiographical and histological characteristics of submerged and nonsubmerged titanium implants. An experimental study in the Labrador dog. Clin Oral Implants Res 1996;7:20-26.
9. Abrahamsson I, BerglundhT, Wennström J, Lindhe J. The peri-implant hard and soft tissues at different implant systems. Clin Oral Implants Res 1996;7:212–219.
10. Cochran DL, Hermann JS, Schenk RK, Higginbottom FL, Buser D. Biologic width around titanium implants. A histometric analysis of the implan-to-gingival junction around unloaded and loaded non-submerged implants in the canine mandible. J Periodontol 1997;68:186–198.
11. Abrahamsson I, Berglundh T, Glantz PO, Lindhe J. The mucosal attachment at different abutments. An experimental study in dogs. J Clin Periodontol 1998;25:721–727.

12. Grunder U, Spielman HP, Gaberthüel T. Implant-supported single tooth replacement in the aesthetic region: A complex challenge. Pract Periodontics Aesthet Dent 1996;8:835–842.
13. Grunder U. Stability of the mucosal topography around single tooth implants and adjacent teeth: 1-year results. Int J Periodontics Restorative Dent 200;20:11-17.
14. Grunder U, Gracis S, Capelli M. Influence of the 3-D Bone-to-Implant Relationship on Esthetics. Int J Periodontics Restorative Dent 2005;25:113-119.
15. Tarnow DP, Cho SC, Wallace SS. The effect of inter-implant distance on the height of interimplant bone crest. J Periodontol 2000;71:546-549.
16. Adell R, Lekholm U, Rockler B, Brånemark PI. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. Int J Oral Surg 1981;10:387-416.
17. Wöhrle PS. Nobel Perfect esthetic scalloped implant: Rationale for a new design. Clin Implant Dent Relat Res 2003; 5(suppl 1):64-73.
18. Lazzara RJ, Porter SS. Platform switching: a new concept in implant dentistry for controlling postrestorative crestal bone levels. Int J Periodontics Restorative Dent 2006;26:9-17.
19. Jansen VK, Conrads G, Richter EJ. Microbial leakage and marginal fit of the implant-abutment interface. Int J Oral Maxillofac Implants 1997;12:527-540.
20. Zipprich H, Weigl P, Lange B, Lauer HC. Micro-movements at the Implant-Abutment Interface: Measurement, Causes, and Consequences. Implantologie 2007;15(1):31-46.
21. Kan JKY, Roe P, Rungcharassaeng K, et al. Classification of sagittal root position in relation to the anterior maxillary osseous housing for immediate implant placement: a cone beam computed tomography study. Int J Oral Maxillofac Implants 2011;26(4):873-876.
22. Araújo MG, Lindhe J. Dimensional ridge alterations following tooth extraction. J Clin Periodontol 2005; 32:212-218.
23. Berglundh T, Lindhe J, Jonsson K et al. The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. J Clin Periodontal 1994; 21:189-193.
24. Makigusa K, Toda I, Suwa F. Microvasculature of the mandibular periosteum in the Japanese monkey. Japan Soc Periodontol 2001;43(3):227-239.
25. Makigusa K. Histologic comparison of biologic width around teeth versus implants: The effect on bone preservation. J Implant Reconstructive Dent 2009;1(1):20-24
26. Small PN, Tarnow DP. Gingival recession around implants: a 1-year longitudinal prospective study. Int J Oral Maxillofac Implants 2001;5(4):527-532.
27. Small PN, Tarnow DP, Cho SC. Gingival recession around wide-diameter versus standard-diameter implants: a 3- to 5-year longitudinal prospective study. Pract Proced Aesthet Dent 2001;13(2):143-146.
28. Esposito M, Ekestubbe A, Grondahl K. Radiological evaluation of marginal bone loss at tooth surfaces facing single Brånemark implants. Clin Oral Implants Res 1993;4:151–157.
29. Spray RJ, Black CG, Morris HF, Ochi S. The influence of bone thickness on facial marginal bone response: Stage 1 placement through stage 2 uncovering. Ann Periodontol 2000;5:119– 128.
30. Rojas-Vizcaya F. Rehabilitation of the maxillary arch with implant-supported fixed restorations guided by the most apical buccal bone level in the esthetic zone: A clinical report. J Prosthet Dent 2012;107:213-220.