IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 16, Issue 9 Ver. IX (September. 2017), PP 61-70
www.iosrjournals.org

A Panoramic View of Junctional Epithelium And Biologic Width Around Teeth And Implant

*Dr. Jaimini Patel[1], Dr. Jasuma Rai[2],Dr. Deepak Dave[3],Dr. Nidhi Shah[4], Dr. Shraddha Shah[5]
[1,2,3,4,5,](Department of Periodontology/ K. M. Shah Dental College and Hospital/ Sumandeep Vidyapeeth, India)
Corresponding Author: *Dr. Jaimini Patel*

Abstract : *Junctional epithelium is the most dynamic feature of the periodontal tissues as it not only plays an important role in health but also displays various characteristic changes in disease. The biologic width around tooth and implants is also an important consideration from treatment point of view. In the following review we have discussed the importance of junctional epithelium and biologic width around teeth and implant and the factors that influence the peri-implant biologic width.*
Keywords : *Biologic Width, Junctional Epithelium, Implant*

Date of Submission: 29 -07-2017 Date of acceptance: 09-09-2017

I. Introduction

Teeth are trans-mucosal organs. This is a unique association in the human body where a hard tissue emerges through the soft tissue. Epithelia exhibit considerable differences in their histology, thickness and differentiation suitable for the functional demands of their location.[1] The gingival epithelium around a tooth is divided into three functional compartments– outer, sulcular, and junctional epithelium. The outer epithelium extends from the mucogingival junction to the gingival margin where crevicular/sulcular epithelium lines the sulcus. At the base of the sulcus connection between gingiva and tooth is mediated with junctional epithelium. It provides the attachment mechanism of the epithelium to the surface of tooth hard substance.[2]

II. Junctional Epithelium Around Tooth

The term junctional epithelium denotes the tissue that is affixed to the tooth on one side and to the oral sulcular epithelium and connective tissue on the other side. Junctional epithelium is the epithelium component of the dentogingival unit that is in contact with the tooth surface. Junctional epithelium forms the tissue attachment of the gingiva to the tooth structures. It differs from the gingival oral epithelium and sulcular epithelium in both its origin and its structure. Compared with other epithelia, junctional epithelial cells are interconnected by a few desmosomes only and occasionally by gap junctions. The fluid-filled intercellular spaces may vary in width, but are wider in comparison with the oral gingival or sulcular epithelium. These features account for the junctional epithelium's remarkable permeability.[1]

III. Mucosa And Junctional Epithelium Around Implants:

Peri-implant mucosa is composed of well-keratinized oral epithelium, sulcular epithelium, and junctional epithelium, as well as underlying connective tissue. In the implant site, the apical portion of the junctional epithelium is consistently separated from the alveolar bone by a zone of non-inflamed, collagen-rich but cell poor connective tissue. This zone, which is about 1-1.5 mm high, is continuous with the junctional epithelium and establishes together with the epithelium an implant-mucosa attachment that is about 3-4 mm high. In the collagen-rich zone, the fibers invest in the marginal bone and run a course more or less parallel to the implant surface. Between the implant surface and the epithelial cells are hemidesmosomes and basal lamina.[4] The soft-tissue interface is made up of the epithelium and the underlying connective tissue, which includes the biologic zone known as the "biologic width," referring to the height of the dentogingival attachment apparatus encircling the tooth. The term "biologic width" is based on the work of Gargiulo et al in 1991 who described the dimensions of the dentogingival junction in human cadavers.[5] The average dimension of 2.04 mm (1.07 mm + 0.97 mm) is comprised of supra-alveolar connective tissue and junctional epithelial attachment. Clinically, in a clinical sense, there must not be any encroachment within 2 mm of the bone that surrounds the tooth. That a similar relationship of bone to overlying soft tissues exists around implants and changes in this relationship may be one of the reasons for the early crestal bone loss.[3]

At the biochemical level as well, there are no differences between the peri-implant and periodontal soft tissue, even if some higher amounts of collagen type V and VI were noticed in peri-implant sof tissue. The vascular supply of the peri-implant gingival or alveolar mucosa is more limited than that around teeth. Indeed, because of the lack of a periodontal ligament, this vascular supply is often reduced because the principal proprioception in natural dentition comes from the periodontal ligament; its absence around the implants reduces the tactile sensitivity and reflex function.[3]

Coronally, close to the sulcus, the junctional epithelium (JE) is 15 to 30 cell layers wide and narrows towards the apical part of the tissue. The coronal two thirds of the JE is composed of two strata, the basal layer facing the connective tissue and the suprabasal layer facing the implant surface. The cells of the basal layer exhibit a cuboidal shape and form towards the connective tissue a basal lamina with desmosomes. The innermost suprabasal cells facing the implant or abutment surface are flat cells, oriented parallel to the surface. They are also called DAT cells (Directly Attached Cells). The apical one third of the JE is composed of only two-cell layers and ends at its apical termination in a one-cell layer of DAT cells. The proliferative capacity of the junctional epithelium leads to the rapid migration of the epithelial cells as soon as the fibrin clot/granulation tissue starts forming at the implant installation. Once the cells reach the implant surface, their attachment occurs rapidly through the basal lamina and the hemidesmosomes.[6]Another possible attachment modality by Kanwara et al in 1998 is an indirect epithelium-to-implant contact.[7] The peri-implant sulcus shares the structural, ultrastructural, and functional characteristics with the gingival tissues. Human studies have demonstrated that epithelium surrounding dental implants possesses similar patterns of differentiation and function to gingival tissues.[3]The presence of granulation tissue adhering to the surface of the transmucosal components is considered the principal factor that stops the epithelium from migrating down apically. Berglundh in 1991 speculated that the reason the epithelium does not migrate down apically is likely due to the interaction between the titanium and the soft tissue.[8]

Connective tissue adhesion of the healing wounds involves the following: formation and adhesion of the fibrin clot to the implant surface; adsorption of the fibrin clot to the implant surface; adsorption of the extracellular matrix (ECM) proteins and connective tissue cells to the implant surface; transformation of the clot into granulation tissue; and migration of epithelial cells on top of the fibrin clot/granulation tissue.[3] The connective tissue zone next to the implant surface is primarily divided into two parts. The first is a 50-µm inner zone that is rich in fibers; resembling scar tissue, several scattered fibroblasts in close contact with the titanium surface maintain the seal between the peri-implant bone and the oral environment.[9] The remaining part of the connective tissue comprises fibers running in different directions, along with the cellular elements and blood vessels.[11] Connective tissue cells and the collagen fiber bundles are separated from the TiO2 surface with a 20-nm-wide proteoglycan layer.[13]

IV. Biologic width

The physiological dento-gingival junction of natural teeth including the length of the epithelial attachment, the length of the connective tissue attachment and the depth of the sulcus is known as "Biologic Width". Biologic width is normally composed of 0.97mm junctional epithelium (JE) and 1.07mm connective tissue attachment (CTA). Accordingly, the biologic width is acknowledged 2.04 mm, reflecting the sum of the epithelial and connective tissue measurements with 0.69 for sulcus depth which makes it 2.73 mm.[5] In this review article we have writtenmentioned in detail about biologic width around implants as junctional epithelium is a part of biologic width and there are very few studies on junctional epithelium alone.

A. Biologic Width around teeth

In healthy periodontium at midfacial level, the sulcus depth is approximately 1 mm; proximally, when the interdental papilla fills the gingival embrasure, about 5 mm of soft tissue is present between the bone crest and the tip of interdental papilla. The 5 mm consist of 1 mm connective tissue, 1 mm epithelial attachment, and 3 mm of sulcular depth. The Biologic Width follows the architecture of the bone crest, which follows the scalloped shape of cement-enamel junction. The difference between the facial and proximal bone crest can range from 2.1 mm to 4.1 mm.[11] The complex is supracrestally located.

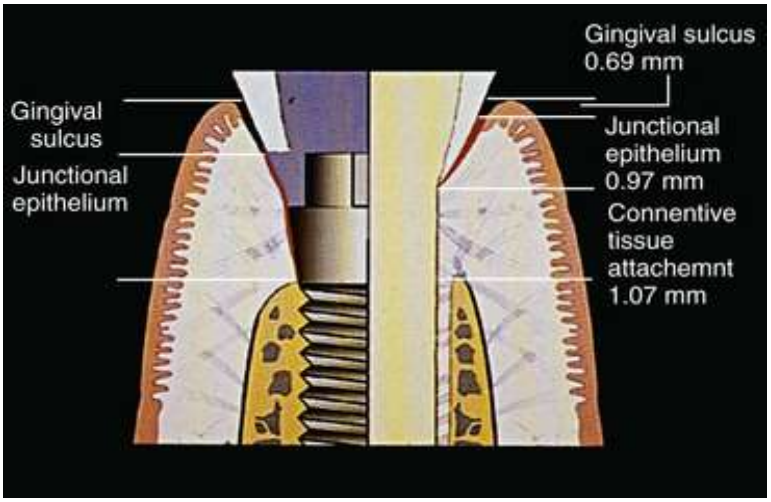

Fig 1. Biologic Width around Tooth and Implant

B. Structure of biologic width around implant

Glauser et al in 2005 in their study on one-piece mini implants calculated the mean dimensions as 4-4.5 mm.[12] Kan et al 2003 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.[13] Epithelium around two-piece implants was always located apical to the microgap.[14]

A biologic width dimension around two-piece implants is larger than that of one-piece implants and natural teeth. Presence of microgap and its location influences the marginal bone levels and the biologic width of the surrounding soft tissue. Hermann et al in 2001 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 for a short or long time. Cochran et al in 1997 observed the values for a biological width/seal around one-part implants before and after loading period of 12 months. Mean values for 3-month unloaded implants were 0.49 mm for sulcus depth (SD), 1.16 mm for the junctional epithelium attachment (JE), and 1.36 mm for the connective tissue attachment (CT). These dimensions differed from the 3-month loaded implants that showed 0.50 mm for SD, 1.44 mm for JE, and 1.01 mm for CT. After the 12-month loading period, these values were 0.16 mm for SD, 1.88 mm for JE, and 1.05 mm for CT. Connective tissue dimensions being more stable around one-piece implants and natural teeth relates 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. Biologic width once invaded around the implant undergoes similar structural and histologic changes as evident around the tooth, independent of tissue biotype (thick/thin).[14]

C. Comparison between Biologic Width around teeth and implants. Clinical relevance

These differences between biologic width around teeth and implant have great clinical impact in achieving aesthetic outcomes in implant restorations.

1. Structure – The connective tissue around teeth is cellular, rich in fibroblast and fibers are perpendicular to tooth. Around implants, the connective tissue has a paucity of cells and is composed primarily of dense collagen fibers, similar to scar tissue. The direction of fibers is parallel to the implant surface. The connective tissue adheres rather than attaches to the implant surface.[17]

2. Vascularity – The connective tissue is highly vascularized around teeth, but poorly vascularized around implants. The vascular supply around teeth is derived from the subperiosteal vessels lateral to the alveolar process and from the periodontal ligament. Peri-implant soft tissue is less vascularized. The blood supply, originates from terminal branches of larger vessels from the bone periosteum at the implant site, but the blood vessels from the periodontal ligament are missing. A zone of avascular connective tissue is directly adjacent to the implant surface.[18,19]

3. Localization: a striking difference - if around teeth the connective tissue fibers are inserted into the dentine coronal to the bone (supracrestal) and provide support for the soft tissues surrounding teeth. Biologic Width around implants forms apical to the bone crest (subcrestal). The depth is given by the final position of the remodeled bone: 2-3 mm apical to the implant abutment interface in two- piece implants.[19]

4. Circumferential morphology – comparing to the scalloped morphology of the Biologic Width around natural teeth, the Biologic Width around implants follows the shape of the implant platform. Usually, implant systems offer flat rotational platforms and, in aesthetic area, they are placed 3 mm - 4 mm sub gingivally.

D. Function of biologic width

Biologic width serves as protective mechanism for underlying bone. The function of junctional epithelium was investigated by Sanz in 1991 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. Chavrier in his histologic biopsy study on the connective tissue around implants revealed predominance of type 1 collagen fibers.[14] Research 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.[20, 21]

E. Biologic Width and implant design

Implant designs are evolving to maintain bone at predictable positions on the implant body. The alterations that impact the most tissue recession are located primarily in the cervical and collar area.[17] The shape and diameter of the platform are of great importance, as well as stable and simplified prosthetic choices in abutment design.

One piece implants - Least marginal bone loss occurred when the rough limit was placed at crestal level, and the polished collar was placed above the alveolar crest. One piece implant designs show a more closely mimicking the Biologic Width around natural teeth.[22] The Straumann Standard implants present a 2.8 mm polished collar, similar to the dimension of the Biologic Width. For more 1 mm apical positioning of implants in situations where mucosal conditions require shorter transmucosal dimensions. One-part implants seem to provide a more predictable surface for soft tissue attachment, since the implant/abutment connection is positioned more coronally.[24] Soft tissue attachment in two-part implants are dependent on the location of the restorative margin that must respect the dimensions necessary for the formation of a biological seal/width. If the required dimensions are not respected, an apical migration of the biological seal/width can be expected.

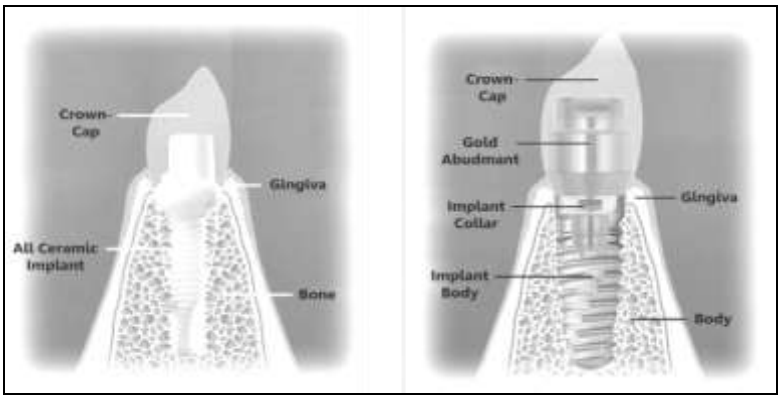

Fig 2 One Piece and Two piece implant

Wide diameter implants - Increased horizontal bone remodeling were found around wide diameter implants. Soft tissue recession around a wide diameter implant averaged 1.58 mm compared to 0.57 mm around a standard-diameter implant. While the wider diameter implant should provide an anatomically correct emergence profile, it may be more prudent to use standard-diameter implants in the aesthetic zone to avoid thinning the buccal cortical bone and excessive soft tissue recession.[17]

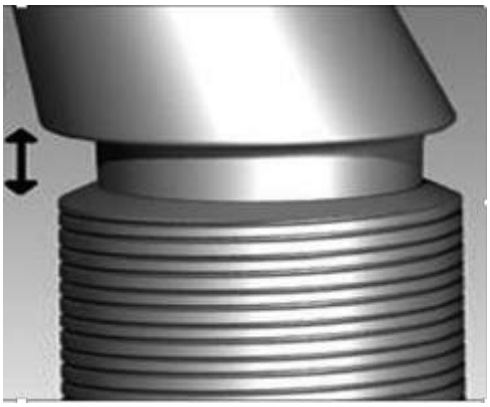

Fig 3 Platform Switch Implant

Platform switch implants - The use of prosthetic abutments with reduced width in relation to the implant diameter (platform switching), during the period of osseointegration, affects Biologic Width by altering the position of the microgap.[22] This seems to have a great potential to limit the crestal resorption. The reduction of the abutment of 0.45 mm on each side (5mm implant/4.1 mm abutment) was found to be sufficient to avoid peri-implant bone loss. The reported bone loss was 0.6 – 1.2 mm, comparing to 1.5 – 2 mm.[23]

Scalloped implants - A scalloped implant platform, that follows the osseous structure of the maxillary anterior teeth may conserve the tissue architecture by minimizing the proximal bone remodeling induced by the subosseous position of the implant head, thus improving the support of the papilla. They were developed specifically for the situations exhibiting three-dimensional ridge topography.[22]

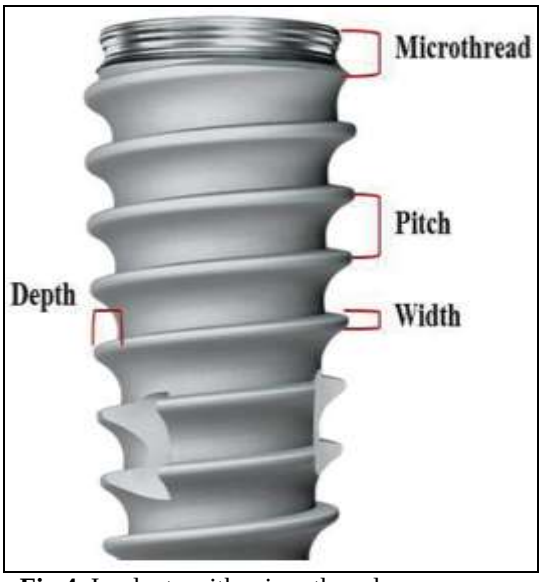

Fig 4. Implants with micro threads

Implants with microthreads reaching the col seem to prevent bone resorption from occuring.[17] Implant systems with restorative abutments positioned deep inside the implant allows the abutment–implant joint to remain stable, and allows the Biologic Width to form and remain on the side of the abutment, rather than the side of the implant.

F. Factors influencing peri-implant biologic width

a) Surface topography

Albrektsson and Wennerberg in 2004 have classified surface topography of implants into three general categories according to mean roughness (SA). The lowest degree of surface roughness is minimally rough with SA values of 0.5-1 μm. Moderately rough implants have SA of 1-2μm and rough ones have Sa greater than 2 μm. Buser et al. (1992) investigated the soft tissue dimensions around three different titanium surfaces, namely a rough surface, a sandblasted surface and a polished surface. There were no significant differences in terms of soft tissue responses among these three implant surfaces. The soft tissue barrier consisted of a sulcus with a non-keratinized sulcular epithelium, a junctional epithelium, and a supra crestal connective tissue with an area of dense circular fibers near the implant surface. In the inner zone of connective tissue, next to the titanium surface, circular fibers were found. In the outer layer, horizontal and vertical fibers were seen running from the periosteum and the alveolar crest towards the oral epithelium. According to the authors, the orientation of fibers was different in rough and smooth surfaces. The fibers forming on smooth surfaces were mostly parallel to the implant surface, while porous-coated surfaces promoted the formation of upright fibers.[26]

b) Implant and abutment materials

Multiple studies have documented the relationship between implant and abutment material composition and the nature of the resulting soft tissue attachment, which are summarized in (Table 1). Rompen et al in 2006 concluded that titanium was the only material that showed consistent soft tissue biocompatibility. Zirconium and aluminum oxide demonstrated favorable histological outcomes, whereas dental porcelain and gold were less biocompatible.[26]

Table 1. Relationship between implant and abutment materials

Study	Model	Implant/ abutment surface	Result
Welander et al (2008)[27]	Dogs	Ti, ZrO2, Au/Pt-alloy abutments	At Au/Pt-alloy abutment sites in comparison with Ti and ZrO2 : 1-apical migration of the barrier epithelium along with marginal bone loss occurred between the second to fifth months of healing. 2-the connective tissue zone (80 μm wide) contained less collagen and fewer fibroblasts and larger fractions of leukocytes. 3-Soft tissue healing appeared to be less stable.
Glauser et al (2005)[12]	Human	One-piece mini-implants made of CPT with either oxidized, acid-etched, or machined surfaces	A biologic width is approximately 4.0 to 4.5 mm, consisting of an epithelial and a supracrestal connective tissue barrier around the experimental one-piece mini-implants that was similar to that described in animal studies. The oxidized and acid-etched implants experienced less epithelial down-growth and longer connective tissue barriers than machined implants.
Kohal et al (2004)[28]	Monkeys	Zirconia and titanium abutments	9 months after implant placement, no significant differences between the responses to the two abutment materials.

c) Surgical protocol

A number of studies have examined the potential role of surgical protocol on peri-implant soft tissue healing. The effect of one- versus two-stage protocol on soft tissue healing of three different implant systems (Astra Tech Implants, Brånemark and Bonefit-ITI) was investigated and compared.[9] The histologic results demonstrated similar dimension and composition of epithelial and connective tissue components of biologic width with 1- or 2- stage procedures for all three implant systems. The current consensus appears to suggest that surgical protocol, especially one- versus two-stage procedures, has little effect on peri-implant soft tissue healing.[26]

d) Loading time

Cochran et al in 1997 stated that after 12 months of loading, biologic width around implants dimensionally resembles biologic width around teeth. In addition, the dimensions of its constituents appear to be independent of loading time.[29] Similar findings were confirmed by Hermann et al. in 2000 who compared non-loaded with loaded implants (ITI Implant System) and submerged with non-submerged healing at different time intervals.[30] Over a 15-month healing period, the total biologic width remained constant. According to the investigator no statistically significant differences were detected among groups during the study period. In a study by Bakaeen et al. in 2009 the dimensions of peri-implant soft tissues around immediately and early-loaded one-piece implants were compared with those of conventionally loaded one-piece implants. Forty-eight titanium sandblasted/acid-etched (SLA) implants were placed in four foxhounds. The implants were placed 3 months (group A), 21 days (group B), ten days (group C), and two days (group D) before restoration. Histometric analysis included dimensional measurements of the sulcus depth, junctional epithelium, the connective tissue seal, and gingival recession. There were no statistically significant differences among the four groups.[31] Finally, in a systematic review Glauser et al. in 2006 reported the occurrence of soft tissue healing comparable to that in conventionally loaded implants hence available evidence suggests that the loading time has little effect on the biologic width.[32]

e) Implant macro-design and microgap position

Weber et al. in 2002 compared the formation of the biological seal around one- and two-part implants and demonstrated that a biological seal is formed irrespective of the implant design. However, two-part implants resulted in a longer junctional epithelium attachment, which was located more apically. The distance from the crestal bone was also shorter in comparison to one-part implants. No differences were found in the length of connective tissue attachment. Findings of related studies are summarized in (Table 2) which seem to indicate that the dimensions and composition of the biologic width are not significantly influenced by the type of implant (i.e. one versus two-piece implants) or the surgical protocol (i.e. one- versus two-stage). Limited evidence suggests, however that more deeply placed implants lead to a longer biologic width.

Table 2. Relationship between one versus two-piece implants and biologic width

Study	Model	Implant/ abutment surface	Result
Judgar et al (2014)[33]	Human	Unloaded one- and two-piece implants	After 4 months of healing, marginal bone loss, gaps, and fibrous tissue were not detected around two types of implants. The biologic width dimension ranged between 2.55 ± 0.16 and 3.26 ± 0.15 to one- and two-piece implants, respectively. This difference was influenced by the connective tissue attachment, while the dimensions of sulcus depth and junctional epithelium were similar between two groups.
Canullo et al (2011)[34]	Human	Implants with matching and non-matching abutments ranging from 0.25 to 0.85mm	No significant differences between groups in inflamed connective tissue (ICT)], the microvascular density (MVD) and collagen content. Forty-eight months after restoration, platform switching and traditional platform implants had similar histological peri-implant soft tissue profiles.
Luongo et al (2008)[35]	Human	Implants with non-matched narrower abutments	The inflammatory connective tissue infiltrate initiated at the microgap located approximately 0.35 mm apical and coronal to the implant/abutment interface.

g) Mucosal thickness

When the connective tissue side of flaps around implants (Branemark System) was thinned to 2mm or less at the time of abutment connection, increased bone resorption was observed. The influence of soft tissue thickness on peri-implant bone remodeling has been investigated by correlating the abutment cuff height, as a surrogate measure for mucosal thickness, with peri-implant marginal bone loss. Results demonstrated increased marginal bone loss around implants with shorter abutments, reflecting thin mucosa, possibly attributed to the need for re-establishing biologic width.[26]

Platform switching has been proposed as a macro-design feature of implants to minimize peri-implant marginal bone loss. The outcome of a randomized trial has reported that platform switching decreases bone loss by 30%. However, platform switching appears to be effective only when adequate mucosal thickness (mucosa thicker than 4.22 mm) is present[36]. A promising concept evolving from the quality (thickness) and quantity (width) of keratinized mucosa is that the volume of the soft tissue over the crestal bone is the main factor establishing the biologic width and preventing crestal bone resorption around the teeth and implants.[37] The volume of the supracrestal gingival tissue was initially evaluated by sonar, followed by elevation of a mucoperiosteal flap and direct measurement. Results showed that the distance from the crestal alveolar bone margin to the crest of the gingival margin correlates positively with the increase in thickness and width of keratinized gingiva. A connective tissue circular ligament is present around the neck of implants.

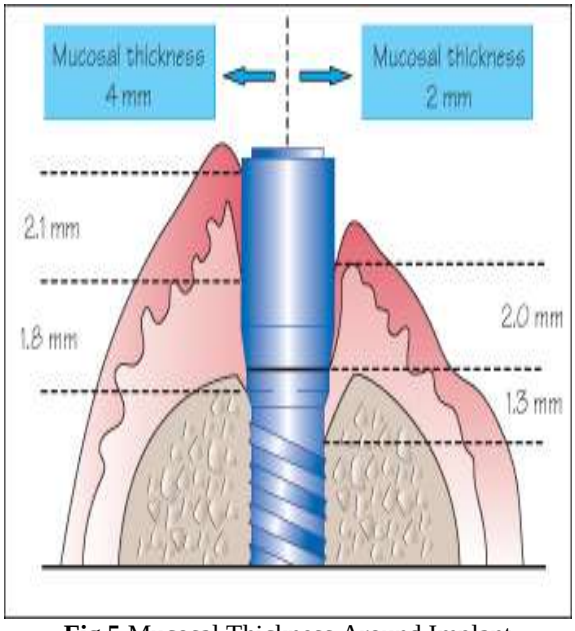

Fig 5 Mucosal Thickness Around Implant

h) Maxilla versus Mandible

Romanos et al. in 2010 conducted a study where a total of 12 implants were placed either in the maxilla or mandible of a patient with a history of smoking. Ten months post implant placement the patient died and the implants were removed en bloc and examined histologically. Distinct dimensional differences were noted. In the maxilla, the biologic width was approximately 6.5 mm versus 4.8 mm in the mandible. Importantly, in the maxilla the connective tissue component was significantly greater than in the mandible. However, no dimensional differences were found in junctional epithelial length for implants placed in either the maxilla or mandible.[38]

i) Flap vs. flapless techniques

Blanco et al. in 2010 examined whether inserting implants via flap or flapless techniques had an effect on the biologic width. In their study on five beagle dogs, four implants were placed in the mandible of each dog immediately following tooth extraction. Flaps were raised on one side (control) while no flaps were raised in the other (test). After 3 months of healing, the dogs were sacrificed. Histometric analysis revealed the followings: 1) Flapless surgery junctional epithelium: 2.54 mm buccal and 2.11 mm lingual; 2) Flapped surgery junctional epithelium: 2.59 mm buccal and 2.07 mm lingual; 3) Flapless surgery connective tissue: 0.68 mm buccal and 0.54 mm lingual; and 4) Flapped surgery connective tissue: 1.09 mm buccal and 0.91 mm lingual. None of the differences between the groups were statistically significant.

Flapless surgery, or limited flap designs (papilla preservation incisions) minimizes bone loss because does not interrupt the blood supply and consequently provides a better aesthetic outcome, with less papilla recession. Widely mobilized flap design generates more bone loss (1.2 mm) comparing to flapless technique. Tarnow reported 2.11 mm vertical bone loss with flap surgery versus 0.6 mm flapless. After full thickness flap procedures bone stabilizes more apically and Biologic Width forms further apically.

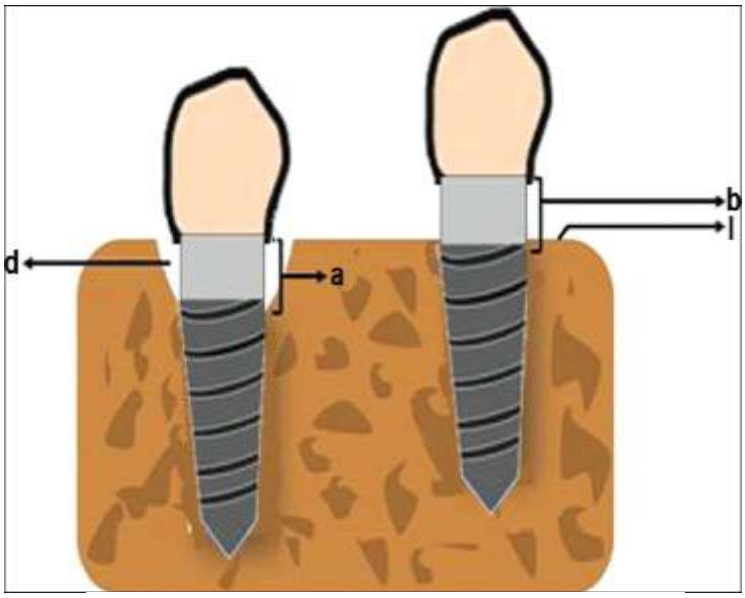

Fig 6. Submerged and non-submerged two-piece implants.

A- Submerged implant collar, B- Non-submerged implant collar, L- Bone level, D- Crestal bone loss.

j) Submerged / non submerged implants

No difference in soft tissue dimensions was found around bone level two–pieces implants that had been placed utilizing a submerged technique as opposed to placing them using a non submerged approach, but the dimension of Biologic Width was greater, and the position more apical than in one piece, non submerged implants with a rough – smooth border at the alveolar crest. In this second situation the smallest value of Biologic Width was measured: 2.84 mm, similar with that in natural teeth: 2.73 mm.[40]

V. Conclusion

The concept of the biologic width forms the basis of the successful peri-implant soft tissue integration around the implant. The peri-implant biologic width is similar to the biologic width around natural teeth and appears to serve similar protective barrier functions. Peri-implant biologic width is larger than the biologic width around teeth, primarily due to a longer junctional epithelium. Peri-implant soft tissue healing, including

establishment of a physiologic peri-implant biologic width, is important for long-term implant function. In order to establish a functional biologic soft tissue seal, a minimum dimension of biologic width is required. When this minimum dimension is absent, crestal bone resorption is likely to occur, to allow space for establishment of a biologic width. In this review article we have included human as well as animal studies in which evidence showed two part implant resulted in a longer junctional epithelium attachment which was located more apically. The distance from the crestal bone was also shorter in comparison to one part implant and loading time has little effect on the biologic width.

References

[1]. Available at: http://periobasics.com/the-dynamics-of-junctional-epithelium.html. [Cited on 2017 April 3].Nanci A ,Bosshardt D Structure of periodontal tissue in health and disease Perio 2000. 2006;40:1–28.
[2]. Dhir S, Lanka M, Kurtzman G, Vandana KL, Peri-Implant and Periodontal Tissues: A Review of Differences and Similarities 2013;34(7):1-12.
[3]. James RA, Schultz RL. Hemidesmosomes and the adhesion of the junctional epithelial prelimnary report. Oral Implantol. 1974;4(3):294-302.
[4]. Gargiulo AW, Wentz FM, Orban B. Dimensions and relations of the dentogingival Periodontol. 1961;32(3):261-267.
[5]. Listgarten MA, Lai CH. Ultrastructure of the intact interface between an endosseous and the host tissues. J Biol Buccale. 1975;3(1):13-28.
[6]. Kawahara H, Kawahara D, Mimura Y, et al. Morphologic studies on the biologic seal Report II. In vivo study on the defending mechanism of epithelial adhesion/attachment Oral Maxillofac Implants.1998;13(4):465-473.
[7]. Berglundh T, Lindhe J, Ericcson I, et al. The soft tissue barrier at implants and teeth. Res.1991;2(2):81-90.
[8]. Abrahamsson I, Berglundh T, Wennström J, Lindhe J. The peri-implant hard and soft tissue at different implant systems. A comparative study in the dog. Clin Oral Implants Res. 1996;7(3):212
[9]. Buser D, Weber HP. Soft tissue reactions to non-submerged unloaded titanium implants Periodontol. 1992;63(3):225-235.
[10]. Tarnow DP, Chu SJ The Interdental Papillae: Aesthetic Parameters Between teeth and Implants. In: Aesthetic restorative dentistry. Principles and Practice. Montage 2008 ;(13):367-383.
[11]. Glauser R, Schunbach P. Perimplant soft tissue barrier at experimental one piece mini-implant-a light microscopic and histometric review Clin Implant Dent Relat Res 2005;7:44-51.
[12]. Kan JY. Dimensions of the periimplant mucosa J Periodontol 2003;74:557-62.
[13]. Dhir S. Significance and clinical relevance of biologic width to implant dentistry. J Interdiscip Dentistry 2012;2:84-91
[14]. Berglundh T, Lindhe J. Dimension of the periimplant mucosa. Biological width revisited. J Clin Periodontol 1996; 23:971-3.
[15]. Cochran DL, Hermann JS, Schenk RK, Higginbottom FL, Buser D. Biologic width around titanium implants. A Histometric analysis of the implanto-gingival junction around unloaded and loaded nonsubmerged implants in the canine mandible. J Periodontol 1997; 68: 186-98.
[16]. Saadoun AP, Touati B Soft tissue recession around implants: is it still unavoidable Part II Pract Proced Aesthet Dent. 2007;19(1): A-H.
[17]. Berglundh T, Lindhe J, Jonsson K, Ericson I The topography of the vascular systems in the periodontal and periimplant tissues in the dog. J Clin Periodontol.21(3):189-193
[18]. Buser D, Weber HP, Donath K et al . Soft tissue reactions to non-submerged
[19]. unloadede titanium implants in beagle dogs. J Periodontol. 1992;63(3):225-235
[20]. Kawahara H, Hashimoto K. Morpholologic studies on the biologic seal of titanium dental implants Int J Oral Maxillofac Implants 1998;13:465-73.
[21]. Lindhe J. Experimental breakdown of periimplant and periodontal tissues Clin Oral Implants Res 1992;3:9-16.
[22]. Leblecioglu B, Rawal S, Mariotti A .A review of the functional and esthetic requirements for dental implants. JADA. 2007;138(3):321-329
[23]. Hermann F, Lerner H, Palti A. Factors influencing the preservation of the periimplant marginal bone. Implant Dent. 2007;16(2):165-175.
[24]. Cochran D. Implant therapy I. Ann Periodontol 1996; 1: 707-91.
[25]. Neiva R, Neiva K, Oh T Clinical and morphological aspects of the implant/soft tissue interface Int Chin J Dent 2002; 2: 151-161.
[26]. Esfahrood Z, Kadkhodazadeh M, Gholamin P, Amid R Biologic Width around Dental Implants: An Updated Review J Dent Mat Tech 2016;1-5
[27]. Welander M, Abrahamsson I, Berglundh T. The mucosal barrier at implant abutments of different materials. Clin Oral Implants Res 2008; 19:635- 641.
[28]. Kohal RJ, Weng D, Bachle M, Strub JR. Loaded custom-made zirconia and titanium implants show similar osseointegration: an animal experiment. J Periodontol 2004; 75:1262-1268.
[29]. Cochran DL, Hermann JS, Schenk RK, Higginbottom FL, Buser D. Biologic width around titanium implants. A Histometric analysis of the implanto-gingival junction around unloaded and loaded nonsubmerged implants in the canine mandible. J Periodontol 1997; 68:186-198.
[30]. Hermann JS, Buser D, Schenk RK, Higginbottom FL, Cochran DL. Biologic width around titanium implants. A physiologically formed and stable dimension over time. Clin Oral Implants Res 2000; 11:1-11.
[31]. Bakaeen L, Quinlan P, Schoolfield J, Cochran DL. The Biologic Width Around Titanium Implants: Histometric Analysis of the Implanto-gingival Junction Around Immediately and Early Loaded Implants. Int J Periodont Rest Dent 2009; 29:297–305.
[32]. Glauser R, Zembic A, Hammerle CHF. A systematic review of marginal soft tissue at implants subjected to immediate loading or immediate restoration. Clin Oral Implants Res 2006; 17; 82-92.
[33]. Judgar R, Giro G, Zenobio E, Coelho PG, Feres M, Rodrigues JA, et al. Biological Width around one-and two-piece implants retrieved from human jaws. Bio Med Res Int 2014; 23.
[34]. Canullo L, Pellegrini G, Allievi C, Trombelli L, Annibali S, Dellavia C. Soft tissues around long-term platform switching implant restorations: a histological human evaluation. Preliminary results. J Clin Periodontol 2011; 38: 86–94.
[35]. Luongo R, Traini T, Guidone PC, Bianco G, Cocchetto R, Celletti R. Hard and soft tissue responses to the platform-switching technique. Int J Periodont Res Dent 2008; 28:551-557.
[36]. Vandeweghe S, De Bruyn H. A within-implant comparison to evaluate the concept of platform switching: a randomised controlled trial. Eur J Oral Implant. 2012; 5:253-62.

[37]. Mendonça JAG. Analysis of a new methodology to evaluate marginal periodontium biological width (in Portuguese). [Thesis]. Bauru, SP: University of São Paulo; 2001. 202p.
[38]. Romanos GE, Traini T, Johansson CB, Piattelli A. Biologic width and morphologic characteristics of soft tissues around immediately loaded implants: studies performed on human autopsy specimens. J Periodontol 2010; 81:70-8.
[39]. Blanco J, Alves CC, Nuñez V, Aracil L, Muñoz F, Ramos I. Biological width following immediate implant placement in the dog: flap vs. flapless surgery. Clin Oral Implants Res 2010; 21:624-3
[40]. Joe Bloggs. The dimension of the biologic width related to aesthetic implant restorations (in Germany). [Thesis]. University of Muenster; 2012;1-31.

*Dr. Jaimini Patel. "A Panoramic View of Junctional Epithelium And Biologic Width Around Teeth And Implant." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) 16.9 (2017): 61-70