REVIEW ARTICLE

Peri-implant Tissue Measurement Terminologies in Health and Disease: A Critical Insight

Anil Akansha[1], Kharidhi L Vandana[2]

Abstract

The implantology field has been a center of interest for several clinicians, teachers, and students globally. Amidst these fast-moving tissues, the terminologies for peri-implant measurements and the standard concept of measurement guidelines remain obscure and compromised. Unfortunately, the pioneering implantologists have not made an adequate attempt to address the existing deficiencies in guidelines, terminologies, and measurements pertaining to peri-implant tissues in health and disease. There is a lack of consistency across definitions of peri-implant osteitis in the literature, and the diagnostic criteria are not clear. Most of the published strategies for peri-implant osteitis therapy are mainly based on treatments used for teeth with periodontitis. The required platform to diagnose, classify, treat and comprehensive terminologies are the need of the hour in the implant related world. Hence, an attempt is made in this paper to briefly address the peri-implant-related clinical measurements, peri-implant disease classification, and its treatment strategies.

Keywords: Peri-implant measurements, Peri-implant osteitis, Peri-implant osteitis therapy, Periodontitis.

CODS Journal of Dentistry (2018): 10.5005/jp-journals-10063-0037

Introduction

Implantology is growing tremendously in clinical practice and in the academic front. Various systems and techniques have been researched for better implant bone stability to benefit the patient at a faster pace. The implantology field has been a center of interest for several clinicians, teachers, and students globally. Amidst these fast-moving issues, the terminologies for peri-implant measurements and the standard concept of measurement guidelines remain obscure and compromised, especially the classification of peri-implant disease and its treatment strategies. Unfortunately, the pioneering implantologists have not made an adequate attempt to address the existing deficiencies in guidelines, terminologies, and measurements pertaining to peri-implant tissues in health and disease.

Hence, an attempt is made in this paper to briefly address the peri-implant-related clinical measurements, disease classification, and treatment strategies.

The peri-implant measurement nomenclature can be initiated with reference to the periodontal measurements. Some of the important terminologies derived from natural tooth, i.e., periodontal measurements are:

- Probing pocket depth (PPD) referred to as peri-implant probing depth (PIPD).[1]
- Clinical attachment level (CAL) can be referred to as peri-implant bone level (PIBL).
- The term peri-implantitis disease can be replaced by peri-implant osteitis.[2,3]
- There is a lack of any standardized classification to differentiate the various degrees of peri-implantitis, which has led to the confusion in interpreting the results of studies evaluating the prevalence, treatment, and outcome of therapy. The classification based on CIST is modified and presented in Table 1.[3,4,5–32]
- The features of PPD and PIPD are presented in Table 1.

[1,2]Department of Periodontics, College of Dental Sciences, Davangere, Karnataka, India

Corresponding Author: Kharidhi L Vandana, Department of Periodontics, College of Dental Sciences, Davangere, Karnataka, India, Phone: +91 9448393364, e-mail: vanrajs@gmail.com

How to cite this article: Akansha A, Vandana KL. Peri-implant Tissue Measurement Terminologies in Health and Disease: A Critical Insight. CODS J Dent 2018;10(2):43–49.

Source of support: Nil

Conflict of interest: None

Limitations of Probing Pocket Depth and Peri-implant Probing Depth

The gingival margin tissue[26] of natural tooth and mucosal margin tissue[33] has a tendency to recede due to bone loss. Therefore, they both are not useful as dependable measurements to appreciate changes from baseline to postoperatively. Hence, there is a need for dependable measurement which utilizes a fixed landmark on tooth [cementoenamel junction (CEJ)] and implant (implant shoulder for 1-stage nonsubmerge implant or its suprastructure) to the base of the probable depth which is CAL in natural tooth and peri-implant bone attachment level (PIBL) in implants.

Peri-implant Bone Attachment Level

- The implant counterpart of CAL in normal tooth can be named as PIBL.
- So, instead of CAL in natural tooth, the term peri-implant bone attachment level (PIBL) is appropriate. This can be measured from a fixed landmark point on the implant (e.g., implant shoulder for 1-stage nonsubmerged implant systems) or its suprastructure[1] to the bone level. As of now, there is a lack of

Table 1: Features of probing pocket depth vs peri-implant probing depth

Criteria	*Sulcus depth/probing pocket depth*	*Peri-implant sulcus depth/peri-implant probing depth*
Definition	The distance from the soft tissue (gingival or alveolar mucosa) margin to the tip of the periodontal probe during usual periodontal diagnostic probing[5]	It is the distance from mucosal marginal position to peri-implant sulcus/pocket depth[3]
	The distance from the gingival margin to the location of the periodontal probe tip inserted for gentle probing at the attachment[6]	
	The probing depth is the distance between the gingival margin and the depth of the probe tip penetration into the pocket[7]	
Instruments used	Metallic instruments	It is possible to use the same type of metal probe around the implant; however, specifically designed titanium probes are manufactured
	Williams probe, UNC-15 probe, Michigan o probe, etc.[8]	Softer flexible plastic probes can be used to prevent the damage to the implant surface[9]
		(Fig. 1)[9]
Probing force	A light probing force is used (0.2–0.3 N) so that the tip of the probe will stop coronal to the bone level, at the apical extension of the barrier epithelium[10]	A light probing force is used (0.2–0.3 N) so that the tip of the probe will stop coronal to the bone level, at the apical extension of the barrier epithelium[11]
	The probing force of 0.75 N has been found to be well tolerated and accurate[12]	Peri-implant probing is more sensitive to force variation than periodontal probing[13]
	Curve analysis of depth force patterns showed that a change in probing force had more impact on the depth reading in the peri-implant than in the periodontal situation[14]	
Probing depth (Fig. 2)[15]	Clinical sulcus depth of <3 mm and true pocket depth of >3 mm are definitive of health and disease status of periodontium	A light probing force is used (0.2–0.3 N) so that the tip of the probe will stop coronal to the bone level, at the apical extension of the barrier epithelium[11]
		The baseline PIPD varies depending on the level of implant placement
		The changes in the PIPD need to be always compared with baseline sulcus depth for ascertaining disease state[11]
		Change in probing parameters over time is more important than initial findings, i.e., there is no normal sulcus depth around implant and it varies; hence a baseline probing should be done once the final restoration has been installed[3]
		A casual mention on implant sulcus depth is said to be around 2.5 mm to 4 (average)[13]
		Clinical probing depth is higher around implants vs teeth[16]
		Probe tip penetrates closer to the bone level unlike natural tooth. This occurs even when the tissues are healthy, because of the lack of connective tissue fiber bundle embedded in the implant surface, which does not prevent the penetration of the tip[17]
Fiber arrangement	The dentogingival collagen fibers are firmly inserted into the cementum and the bone and in a perpendicular or oblique direction, thus serving as a barrier to the epithelial migration and the impending bacterial invasion[18]	Fibers run a parallel course to the implant surfaces as observed by some investigators, and in some, fibers found to be running in different directions.[19] However, perpendicular orientation of the fiber was also found in implants with porous surface[20]
	Thus establishing a probing depth around the teeth	The connective tissue adhesion with implants has a poor mechanical resistance as compared to the natural tooth.[21] This, combined with reduced cellularity and vascularity in the peri-implant connective tissue, may make them more susceptible to disease initiation and progression[22]
		Thus explaining the deeper penetration of probe tip
		Peri-implant crevice is surgically created and is not developed as it is for natural tooth[23]

Contd…

Contd…

Criteria	*Sulcus depth/probing pocket depth*	*Peri-implant sulcus depth/peri-implant probing depth*
Role of occlusal force in pocket formation	There appears to be a resilient connection between bone, periodontal ligament, and cementum around the tooth.[24] This explains the gradual dissipation and distribution of occlusal forces via periodontal fibers to bone	A rigid connection appears in the form of functional ankylosis/osseointegration due to the lack of periodontal ligament around implants, leading to direct transmission of load to bone-implant interface[24] Hence the occurrence rate of bone loss is more with implant
Histology	Higher proportion of lymphocytes, macrophages, and PMNs	Lower proportion of lymphocytes, macrophages and PMNs. Hence, implants render a weak biological barrier to prevent the apical migration of inflammatory cells compared to teeth[25]
	Junctional epithelium attaches to the enamel surface via internal basal lamina and desmosomes along the entire length of junctional epithelium[19]	Hence, probability of early occurrence of disease as well as increase in probing depth around implant is higher The attachment of peri-implant epithelium to the implant surface is confined to the apical region[19]
Landmarks	Three landmarks: • Gingival margin[26] • Cement–enamel junction • Base of the sulcus/pocket • The probing pocket depth is read out in relation to the gingival margin using the markings of the periodontal probe[10]	Three landmarks: • Mucosal marginal position • As CEJ is missing, fixed reference point on implant (e.g., implant shoulder for 1 stage nonsubmerge implant) or its suprastructure • Base of implant probing depth[1] • The PIPD is read out in relation to the mucosal margin position[3]
Gingival thickness	Thicker biotype is usually associated with pocket formation and thinner biotype is generally accompanied by recession[26]	PIPD seems to be related to thickness and type of mucosa circumscribing the abutment. Alveolar mucosa is generally associated with deeper pocket, whereas keratinized collar is usually accompanied with shallower depths[27]
Influencing factors:	• Root morphology[28] • Shape of the crown[29] • Anatomic features like concave surfaces, anomalies, shape of cervical third, and position of furcation[30]	• Abutment height • Depth of the fixture countersinking at stage 1 surgery • Amount of the tissue thinning at the stage 2 surgical procedure[23] • Surface texture irregularities • Shape of the implant • Configuration of the restoration[31]
Instruments used for treatment	Metallic supra and subgingival scalers and curettes Ultrasonic and sonic instruments[32]	Plastic scaler tips (Implacare H6/H7 © 2015 Hu-Friedy Mfg. Co., LLC, USA), titanium alloy curettes (Maxil®, Chicago, USA), plastic modified ultrasonic point (SofTip implant insert by Dentsply, USA), and air polishing systems (Air-N-Go® Satelec, Acteon, USA) were used[3]

specific nomenclature for several of implant-related clinical measurements.

- As with the natural tooth, the CAL is measurable due to the presence of the connective tissue between tooth and bone. However, this connective tissue is missing in relation to the peri-implant area as the peri-implant bone hugs the implant screw.[34]
- Histologic study in human biopsy specimens showed that the inflammatory infiltrate in peri-implantitis lesions is in direct contact with the alveolar bone and extends into marrow spaces. This differs from the periodontal lesion, in which the inflammatory infiltrate is separated from the bone by approx. 1 mm of noninflamed connective tissue.[35]

Conflicts on Classification

Most of the published strategies for peri-implantitis therapy are mainly based on the treatments used for teeth with periodontitis.

- The extant treatment strategies for peri-implant diseases are based on the Cumulative Interceptive Supportive Therapy (CIST) protocol. The major drawback of this proposal is dependence

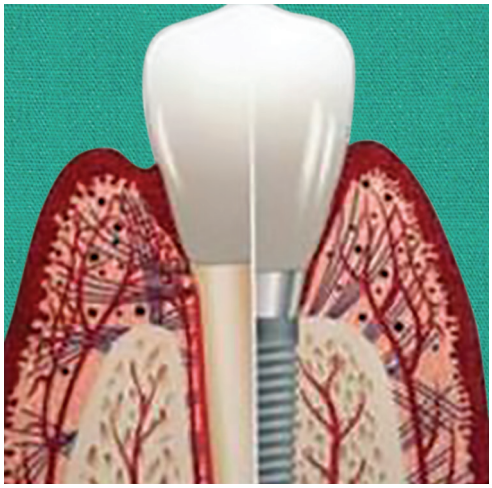

Fig. 1: Peri-implant vs natural teeth in health

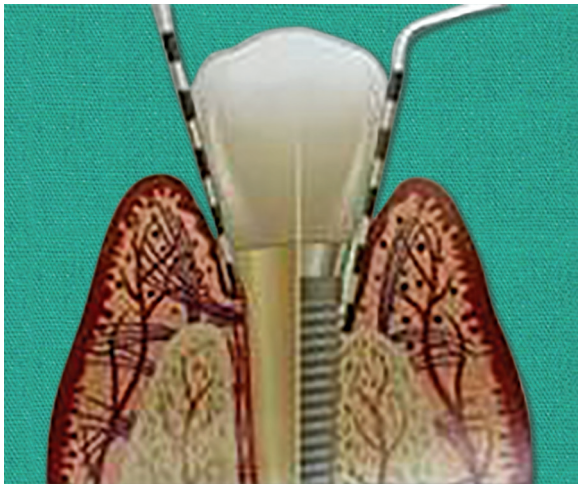

Fig. 2: CAL in peri-implant area vs natural teeth

on the implant pocket depth, which is found to be variable even in healthy status.

- There is a lack of any standardized classification to differentiate the various degrees of peri-implantitis, which has led to confusion in interpreting the results of studies evaluating the prevalence, treatment, and outcomes of therapy.
- Froum and Rosen[4] proposed a classification for peri-implantitis based on the severity of the disease. A combination of bleeding on probing and/or suppuration, probing depth, and extent of radiographic bone loss around the implant were used to classify the severity of peri-implantitis into early, moderate, and advanced categories. Again, this classification considered pocket depth as a clinical parameter, and hence a modified treatment strategy[3] based on the clinical, radiologic, and diagnostic criteria and the prognosis has been introduced for the first time (Table 2). The use of greater than or less than 2 mm of PIPD is eliminated as there is no specific normal PIPD/sulcus depth, which depends on the level of implant placement.[3]

Controversial Nomenclature

The nomenclature "peri-implantitis" appears to be a misnomer: the addition of the suffix "*itis*" is used to signify inflammation of living tissues, e.g., *mucos* "*itis*"; the addition of "*itis*" to a nonliving implant remains highly questionable scientifically. The natural bone that integrates around the implant, peri-implant bone, is inflamed during the disease process. The obvious sign of osteitis is loss of bone appreciated radiographically. Hence, the term "peri-implant osteitis" is recommended instead of "peri-implantitis".[3]

Treatment Strategies

The treatment of peri-implant infections comprises conservative (nonsurgical) and surgical approaches as suggested by Vandana (2015) and is presented in Table 3.
Primary goals of the treatment:

- Elimination of peri-implant mucosal inflammation.
- Cessation of peri-implant disease progression.
- Maintenance of functionality of implant with healthy peri-implant tissues.
- Regeneration of lost peri-implant tissues.
- Restoring peri-implant esthetics such as treatment of mucosal recession, inadequate width, and thickness of peri-implant mucosa.

Other terminologies which are of limited mention in literature and with minimum explanation:

- Early peri-implantitis, defined as the presence of an inflammatory lesion of infective etiology when osseointegration is being established and the implant is mechanically stable.
- Chronic implantitis, the slowly progressive form of the disease.
- Aggressive form of peri-implantitis, a rapidly progressive form of the disease.

- Necrotizing form of peri-implantitis.
- Early implantitis.
- Retrograde implantitis (Meffert,1996): refers to the presence of a lesion at the apex of the implant. A condition known as retrograde peri-implantitis may also be associated with implant failure.

Retrograde implant failure may be due to bone microfractures caused by premature implant loading or overloading, other trauma, or occlusal factors. Implant failures from retrograde peri-implantitis are characterized by periapical radiographic bone loss without, at least initially, gingival inflammation. The distinction between implant failure due to infection with periodontal pathogens (infective failure) and implant failure associated with retrograde peri-implantitis (traumatic failure) is also reflected in the microflora.[3]

Conclusion

There is a lack of consistency across definitions of peri-implant osteitis in the literature, and the diagnostic criteria are not clear. Most of the published strategies for peri-implant osteitis therapy are mainly based on treatments used for teeth with periodontitis. The required platform to diagnose, classify, treat and comprehensive terminologies are the need of the hour in the implant related world. This attempt made by the authors to present these issues is first of its kind and may benefit the implantologists across the globe.

Table 2: Classification of peri-implant disease based on the clinical, radiological, and diagnostic criteria and prognosis (adapted from Vandana)[3]

Diagnosis	*Classification*	*Treatment*	*Prognosis*
Healthy peri-implant tissues	Grade 0 (healthy peri-implant mucosa) Peri-implant mucosa pink and firm	• Continue oral hygiene instructions • Revaluation • Periodic maintenance	Excellent
Peri-implant mucositis (PIM)	Grade I (PIM) • Inflamed, enlarged and soft edematous peri-implant mucosa • Bleeding on probing (BOP) (diagnostic sign) • No bone loss	• Nonsurgical therapy • Patient education and motivation • Institution of plaque control measures • Management of risk factors for peri-implant disease. • Peri-implant scaling • Systemic antimicrobials • Peri-implant local delivery of drugs • Occlusal therapy wherever it is indicated • Revaluation • Periodic maintenance	Good
Peri-implantitis (PI) Osteitis (mild)	Grade II (mild) • Inflamed enlarged edematous peri-implant mucosa • BOP • Suppuration • Increase in PIPD from baseline • Clinical attachment loss—recession may be seen • Radiographic bone loss—25% of implant length	• Nonsurgical therapy • Reinforcing oral hygiene instructions • Surface decontamination • Peri-implant surgery • Resective or regenerative peri-implant surgery • Peri-implant esthetic surgery if indicated, e.g., treatment of mucosal recession • Revaluation • Periodic maintenance	Fair

PI (moderate)	Grade III (moderate)
	• Inflamed, edematous, enlarged peri-implant mucosa
	• BOP
	• Suppuration
	• Increase in PIPD from baseline clinical attachment loss—recession may be seen
	• Radiographic bone loss—25% to 50% of implant length
		• Nonsurgical therapy
		• Surface decontamination
		• Peri-implant surgery (depending on osseous defect morphology)
		• Class I (horizontal bone loss)
		• Class II (vertical bone loss)
		  • IIa—three wall defect (regenerative peri-implant surgery)
		  • IIb—two wall defect (regenerative osseous surgery)
		  • IIc—one wall defect (respective osseous surgery)
		  • Resective peri-implant surgery
		  • Osteoplasty
		  • Ostectomy (one wall defect)
		  • Regenerative peri-implant surgery (two wall and three wall defects)
		• Guided bone regeneration
		  • Osseous grafts and substitutes
		  • Osseous grafts and substitutes and membranes
		• Peri-implant esthetic surgery
			Fair to poor

Contd…

Contd…

Diagnosis	*Classification*	*Treatment*	*Prognosis*
		• Enhancement of keratinized width and thickness • Treatment of recession • Frenectomy/frenotomy • Vestibuloplasty • Ridge augmentation • Revaluation • Periodic maintenance	
PI (severe)	Grade IV (severe) • Features of PIM • Suppuration • Radiographically more than 50–75% bone loss of implant length	• Nonsurgical treatment • Possible surgical treatment or explanation of the implant	Questionable to hopeless

Table 3: Classification of treatment strategies as suggested by Vandana[3]

Emergency therapy	Treatment of emergency: • Drainage of peri-implant abscess • Systemic antimicrobials and anti-inflammatory agents
Nonsurgical therapy	Nonsurgical therapy: • Patient education and motivation • Institution of plaque control measures • Management of risk factors for peri-implant diseases, peri-implant scaling (manual peri-implant scaling instruments, e.g., plastic/Teflon coated/titanium alloys or curettes or power-driven peri-implant scaling instrument: e.g., plastic ultrasonic points) • Systemic antimicrobials • Peri-implant local delivery of drugs • Occlusal therapy when indicated • Revaluation • Periodic maintenance
Surgical therapy	• Implant surface decontamination (physical-implantoplasty)/chemical/laser/PDT) • peri-implant surgery (depending on the osseous defect morphology) • Resective peri-implant surgery • Osteoplasty: bulbous bony contour • Ostectomy (one wall defect) • Regenerative peri-implant surgery (two wall and three wall defects) • Guided bone regeneration • Osseous grafts and substitutes • Osseous grafts and substitutes and membranes • Peri-implant esthetic surgeries • Enhancement of keratinized width and thickness • Treatment of recession • Frenectomy/frenotomy • Vestibuloplasty • Ridge augmentation

Future directions:
Use of PRF, PRP and growth factors for regenerative periodontal therapy
Use of ozone and probiotics as a part of nonsurgical peri-implant therapy, PDT, photodynamic therapy

References

1. Salvi GE, Lang NP. Diagnostic parameters for monitoring peri-implant conditions. Int J Oral Maxillofac Implants 2004;19(Suppl):116–127.
2. Antolín AB, Pascua García MT, Nasimi A. Infections in implantology: from prophylaxis to treatment. Med Oral Patol Oral Cir Bucal 2007;12:E323–E330.
3. Vandana KL, Dalvi P, Nagpal D. Management of periimplant infections. J Int Clin Dent Res Organ 2015;7:160–179. DOI: 10.4103/2231-0754.172931.
4. Forum SJ, Rosen PS. A proposed classification for peri-implantitis. Int J Periodontics Restorative Dent 2012;32:533–540.
5. Glossary of periodontal terms, 4th ed. 2001.
6. Wilkins EM. Clinical practice of the dental hygienist, 10th ed., Lippincott Wiliams and Wilkins. p. 213.
7. Hermann F, Lerner H, Palti A. Factors influencing the preservation of the periimplant marginal bone. Implant Dent 2007;16(2):165–175. DOI: 10.1097/ID.0b013e318065aa81.
8. Newman MG, Takei H, Klokkevold PR, et al. Scaling and root planing. In: Pattison AM, Pattison GL. Clinical Periodontology, 10th ed., ch. 51. Elsevier's Saunders. p. 750.
9. Renevert S, Giovannoli JL. Diagnosis. In: Renevert S, Giovannoli JL. Peri-Implantitis, ch. 2 France: Quintessesnce International; 2012. p. 22.
10. Eicholz Peter, T-S Kim. Reproducibility and validity of the assessment of clinical furcation parameters as related to different Probes. J Periodontol 1998;69:328–336. DOI: 10.1902/jop.1998.69.3.328.
11. Peri-implant mucositis and peri-implantitis: a current understanding of their diagnoses and clinical implications. J Periodontol 2013;84:436–443. DOI: 10.1902/jop.2013.134001.
12. Newman MG, Takei H, Klokkevold PR, et al., In: Diagnosis Kokkevold PR, Cochran DL. Clinical Periodontology, 10th ed., ch. 74. Elsevier's Saunders. p. 552.
13. Dhir S, Mahesh L, Kurtzman GM, et al. Peri-implant and periodontal tissues: A review of differences and similarities. Compend Contin Educ Dent 2013;34:e69–e75.
14. Mombelli A, Mühle T, Brägger U, et al. Comparison of periodontal and peri-implant probing by depth-force pattern analysis. Clin Oral Implants Res 1997;8(6):448–454. DOI: 10.1034/j.1600-0501.1997.080602.x.
15. Renevert S, Giovannoli JL. Diagnosis. In: Renevert S, Giovannoli JL. Peri-Implantitis, ch. 2 France: Quintessesnce International; 2012. p. 23.
16. Mombelli A, Lang NP. Clinical parameters for evaluation of dental implants. Periodontol 2000 1994;4:81–86. DOI: 10.1111/j.1600-0757.1994.tb00008.x.
17. Newman MG, Takei H, Klokkevold PR, et al. Clinical aspects and evaluation of the implant patient Kokkevold PerryR, Cochran DavidL. Clinical Periodontology, 11th ed., ch. 74. Elsevier's Saunders. p. 1084.
18. Stern IB. Current concepts of the dentogingival junction: The epithelial and connective tissue attachments to the tooth. J Periodontol 1981;52(9):465–476. DOI: 10.1902/jop.1981.52.9.465.
19. Ericcsson I, Lindhe J. Probing depth at implants and teeth. An experimental study in the dog. J Clin Periodontol 1993;20(9):623–627. DOI: 10.1111/j.1600-051X.1993.tb00706.x.
20. Ximénez-Fyvie LA, Haffajee AD, Socransky SS. Comparison of the microbiota of supra- and subgingival plaque in health and periodontitis. J Clin Periodontol 2000;27(9):648–657. DOI: 10.1034/j.1600-051x.2000.027009648.x.
21. Hermann JS, Buser D, Schenk RK, et al. Biologic width around one- and two-piece titanium implants. Clin Oral Implants Res 2001;12(6): 559–571. DOI: 10.1034/j.1600-0501.2001.120603.x.
22. Ivanovski Saso, Lee Ryan. Comparison of peri-implant and periodontal marginal soft tissues in health and disease. Periodontol 2000 2017; 1–15.
23. Buser D, Weber HP. Soft tissue reactions to non-submerged unloaded titanium implants in beagle dogs. J Periodontol 1992;63(3):225–235. DOI: 10.1902/jop.1992.63.3.225.
24. Jacobs R, van Steenberghe D. Role of periodontal ligament receptors in the tactile function of teeth: A review. J Periodontal Res 1994;29(3):153–167. DOI: 10.1111/j.1600-0765.1994.tb01208.x.
25. Ericsson I, Berglundh T, Marinello C, et al. Long-standing plaque and gingivitis at implants and teeth in the dog. Clin Oral Implants Res 1992;3(3):99–103. DOI: 10.1034/j.1600-0501.1992.030301.x.
26. Vandana KL, Gupta I. The relation of gingival thickness to dynamics of gingival margin position pre- and post-surgically. J Indian Soc Periodontol 2016;20(2):167–173. DOI: 10.4103/0972-124X.175173.
27. Atassi F. Periimplant probing: Positives and negatives. Implant Dent 2002;11(4):356–362. DOI: 10.1097/00008505-200211040-00015.
28. Armitage GracyC. Manual periodontal probing in supportive periodontal treatment. Perio 2000 1996;12:33–39. DOI: 10.1111/j.1600-0757.1996.tb00078.x.
29. Renvert S, Badersten A, Nilveus R, et al. Healing after treatment of periodontal intraosseous defects. I. Comparitive study of clinical methods. J Clin Periodontol 1981;8:387–399. DOI: 10.1111/j.1600-051X.1981.tb00888.x.
30. Wilkins EM. Clinical practice of the dental hygienist, 10th ed., Lippincott Wiliams and Wilkins. p. 234.
31. Bauman GR, Mills M, Rapley JW, et al. Clinical parameters of evaluation during implant maintenance. Int J Oral Maxillofac Implants 1992;7(2):220–227.
32. Newman MichaelG, Takei Henry, Klokkevold PerryR, et al. Sacling and root planing Pattison AnnaM, Pattison GardonL. Clinical Periodontology, 10th ed., ch. 51. Elsevier's Saunders. pp. 750–763.
33. Vroom M, Timmerman M. Clinical attachment level as a measure to detect changes in clinical peri-implant condition: A 12-year longitudinal study. Clin Oral Impl Res 2014;25(Suppl):10.
34. Renevert S, Giovannoli JL. Pathogenesis Renevert S, Giovannoli JL. Peri-Implantitis, ch. 1 France: Quintessesnce International; 2012. p. 5.
35. Renevert S, Giovannoli JL. Pathogenesis Renevert S, Giovannoli JL. Peri-Implantitis, ch. 1 France: Quintessesnce International; 2012. p. 9.