The Prosthetic–Biologic Connection and Its Influence on Peri-implant Health: An Overview of the Current Evidence

Muhammad H. A. Saleh, BDS, MS, MSD[1,2]/Matthew Galli, DDS[1]/Rafael Siqueira, DDS, MS, PhD[1]/ Maria Vera, DDS[1]/Hom-Lay Wang, DDS, MSD, PhD[1]/Andrea Ravida, DDS, MS[1]

Prosthetic design is a critical step in implant treatment planning that must synchronize with implant positioning to promote a state of peri-implant health. Improperly designed prostheses may not only hinder patient (or professional) hygiene measures but also impact the ability of clinicians to examine the peri-implant supporting tissues for diagnostic purposes. The purpose of this review was to discuss the current state of the evidence surrounding prosthetic factors associated with peri-implant diseases. Following the chronologic order of implant treatment, key prosthetic variables were discussed in relation to peri-implant disease pathogenesis. Specific concepts including the impact of implant spatial positioning, abutment height, residual cement, and implant splinting were found to be associated with peri-implant disease pathogenesis. Excessive occlusal forces were found to play a role in susceptibility to prosthetic complications with limited evidence to suggest a role in peri-implant disease progression. An intimate prosthetic-biologic connection exists, which must be respected to promote an environment for long-term peri-implant stability and health. *Int J Oral Maxillofac Implants 2022;37:690–699. doi: 10.11607/jomi.9523*

Keywords: dental implants, dental prosthesis designs, etiology, peri-implantitis

The overarching objective of contemporary implant-supported prosthetic rehabilitation is long-term clinical success with minimal complications. Implant complications can be classified as either biologic or biomechanical (ie, prosthetic) in nature. Among these, some of the most frequent and challenging to manage include peri-implant mucositis and peri-implantitis.[1] The mean prevalence of peri-implantitis has been estimated to be 45% at the patient level and 25% at the tooth level.[2] Commonly, peri-implantitis can lead to peri-implant soft and hard tissue deficiencies and ultimately implant failure.[3]

The 2017 World Workshop stated that there is robust evidence pointing toward a positive relationship between peri-implantitis and a previous history of periodontitis, poor plaque control, and a lack of regular maintenance visits.[4] Overall, the risk factors/indicators with the strongest level of evidence are those pointing toward a biofilm-based etiology for peri-implantitis. Biofilm accumulation and the ability to access dental implants and their restorations for microbial decontamination are directly related to prosthetic design and restorative characteristics. It is a common observation that peri-implant bone remodeling generally begins after delivery of the prosthesis or healing abutment and seldom occurs before this point. Interestingly, up to date, no literature has summarized the current state of the evidence regarding the prosthetic-biologic connection in the context of peri-implant disease pathogenesis. Hence, the aim of this article was to discuss key prosthetic factors that may have an influence on peri-implant bone loss (disease) susceptibility following the chronologic order of treatment, starting from treatment planning considerations all the way up to occlusion.

RELATIONSHIP BETWEEN IMPLANT POSITIONING AND ACCESS FOR MONITORING AND HYGIENE

Treatment planning philosophies regarding implant positioning have evolved significantly over time, with the weight shifting from placement based on bone availability toward prosthetically driven implant therapy.[5] Biologically driven implant therapy was also described.[6] Ultimately, the ideal implant position must facilitate fabrication of a functional, esthetic, and cleanable prosthesis. In addition, the three-dimensional implant position and the definitive crown contours are crucial factors

[1]Department of Periodontics and Oral Medicine, University of Michigan School of Dentistry, Ann Arbor, Michigan, USA.
[2]Department of Periodontics, University of Louisville School of Dentistry, Louisville, Kentucky, USA.

The first two authors contributed equally to this work.

Correspondence to: Dr Hom-Lay Wang, Department of Periodontics and Oral Medicine, University of Michigan School of Dentistry, 1011 N University Ave, Ann Arbor, MI 48109-1078, USA. Email: homlay@umich.edu

Submitted June 10, 2021; accepted February 22, 2022.

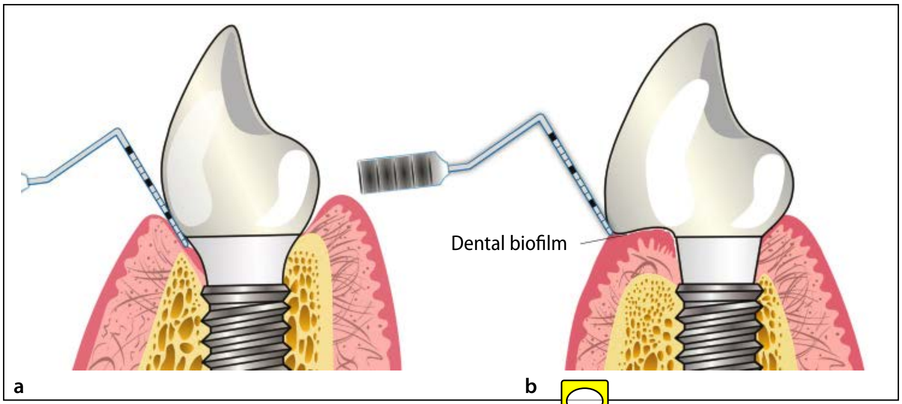


Fig 1 *(a)* Ideal crown contours should enable access for oral hygiene measures, maintenance therapy, and monitoring of peri-implant tissue health. *(b)* Bulky crown contours not only hinder proper hygiene but also mask early disease detection due to limited access for probing.

contributing to the maintenance of peri-implant tissue health.

Effect of Buccolingual Implant Position on Peri-implant Health

Buccolingual positioning may directly influence prosthetic design and peri-implant tissue responses. "Too buccal" placement can predispose toward increased peri-implant bone remodeling as well as a higher susceptibility to development of hard and soft tissue deficiencies and esthetic concerns.[7] Approximately 2 mm of buccal bone relative to the implant platform is recommended to preclude mucosal recession.[8] On the other hand, the often recommended "too lingual" placement may result in an overcontoured restoration facilitating food debris accumulation and hindering access for hygiene and peri-implant probing (Fig 1).[9,10]

Effect of Vertical Implant Position on Peri-implant Health

In regard to how vertical positioning impacts prosthetic design, "too shallow" implant placement does not allow establishment of adequate peri-implant supracrestal tissue height, frequently resulting in overcontoured restorations (due to a lack of apicocoronal distance between the implant platform and the gingival margin), implant thread exposure, and esthetic concerns. Conversely, if the implant is placed slightly deeper, the restorative dentist has more vertical height to develop a prosthetic profile with a more acute emergence angle (Fig 2a). More acute angles generally facilitate access for hygiene and maintenance.[11] On the other hand, deeper implant placement may also lead to a higher propensity for deeper probing depths and may potentially create a niche for pathogenic bacterial accumulation.[12,13] A recent systematic review demonstrated that subcrestal implant positioning does not create significant differences in marginal bone loss (MBL) as long as the implants are not placed too deep (> 3 mm subcrestally).[14]

Effect of Mesiodistal Implant Position on Peri-implant Health

In respect to horizontal positioning, off-center placement especially in the posterior area affects the restorative emergence profile and has a significant influence on access for hygiene.[15] A recent in vitro study advocated for a modified prosthetic design for cases where the horizontal implant position is offset in the mesiodistal dimension to facilitate biofilm removal.[16] At posterior sites, this alternative design consists of a slim premolar-like crown in conjunction with a cantilevered pontic. This may facilitate access for oral hygiene through interproximal embrasures (Fig 2b). Off-center implant positioning may be considered in cases where the mesiodistal restorative space limits placement of two adjacent implant-supported crowns but is too large for a single conventional restoration. Further clinical trials should assess the validity of this approach.

EFFECT OF IMPLANT DIAMETER ON RESTORATION DIMENSIONS

Clinicians are encouraged to match the implant platform with the desired restorative dimensions when biologically feasible. This may limit several restorative complexities. Matching wider crowns with a wider implant platform (and vice versa) results in a more biologically maintainable restoration and simplifies examination via peri-implant probing.[17] A large mismatch between the crown and the implant body may lead to overhanging contours and an obtuse emergence angle, with all the associated negative biologic consequences (Fig 2c).

The vertical and horizontal position of the implant must synchronize with other key prosthetic and biologic factors to set the stage for success. For example, an obtuse emergence angle would be aggravated by a narrow-diameter implant if placed in a shallow position. It may be safer in this situation to place implants with narrower platforms slightly deeper (Fig 2d).

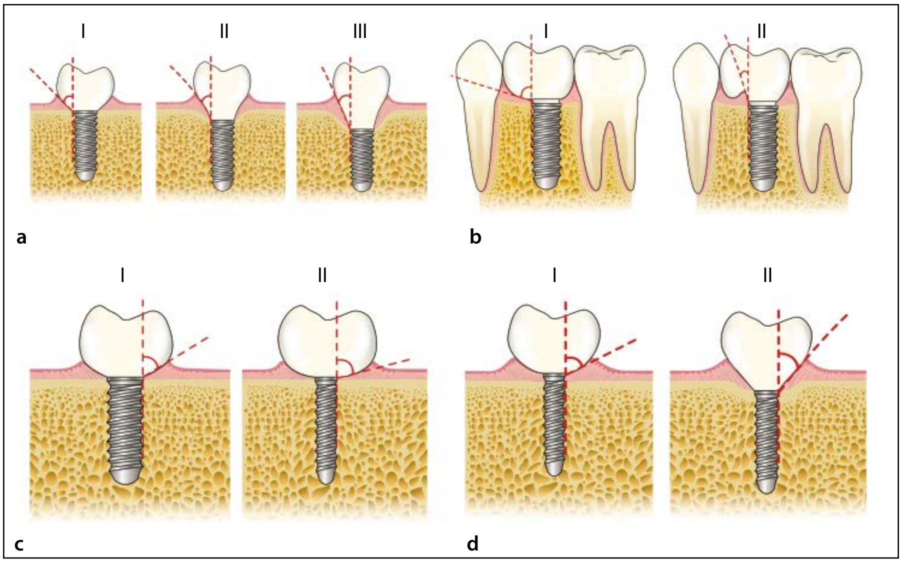


Fig 2 The effect of implant and/or crown position on the emergence angle. *(a)* The effect of apicocoronal implant position on the emergence angle. *(b)* I) The effect of horizontal implant position (horizontal offset) on the emergence angle. II) Suggested crown design to overcome a compromised implant position.[16] *(c)* The effect of implant diamaeter to crown size ratio on emergence angle. This is more accentuated when narrow-diameter implants are placed in posterior sites. *(d)* The illustrated situations in Figs 2a to 2c rarely occur independently. I) Shallower implant placement can amplify the problematic situation in Fig 2c. II) Deeper implant placement can alleviate the problematic situation in Fig 2c.

It is also worth mentioning that multiple studies revealed that tissue-level implants may not be as affected by changes in emergence angle as bone-level implants.[11,18] In the case of tissue-level implants, this finding may be due to the contribution of a machined collar, which contributes to an increased distance between the crown-abutment margin and the alveolar crest. Increased abutment height may be considered to compensate for this if a bone-level system is used.[19,20]

IMPACT OF ABUTMENT HEIGHT ON MARGINAL BONE LEVELS

The periodontal literature has historically reported that a minimum distance of approximately 2 mm from the restorative margin to the alveolar crest is indispensable for adequate formation of the supracrestal tissue attachment around teeth and maintenance of a healthy periodontium.[21,22] It would seem logical to extend equivalent expectations toward implant restorations (Fig 3). However, several key differences between the peri-implant and the periodontal apparatuses make drawing parallels between the two fairly nebulous.[23–27] The last decade was marked by a great interest in understanding whether abutment height may play a role in influencing MBL. Appropriate selection of abutment height is essential, allowing placement of the crown margin in a position that favors adequate formation of supracrestal tissue adhesion (STAd) or supracrestal tissue height[25] and minimizes MBL.[28] Many recent clinical studies have demonstrated a higher magnitude of peri-implant MBL when short abutments are used compared to longer ones.[19,20,29–32]

Abutment height is often selected considering that the prosthetic margin should be placed at or slightly below the level of the peri-implant mucosa[33] to support a cleanable and esthetic prosthetic design. It has been suggested that in cases of thick vertical mucosa (> 2 mm), abutment selection should consider establishing an adequate STAd (2 to 4 mm) to minimize the risk of MBL (Fig 4a). For equicrestal placement, a long abutment (> 2 mm) in the presence of thin vertical mucosa (< 2 mm) is contraindicated from an esthetic perspective due to the resulting supragingival position of the abutment-prosthetic margin, atlhough minimal MBL will be expected (Fig 4b). Subcrestal placement in conjunction with a longer abutment may be implemented in this scenario. When thick vertical mucosa is present, equicrestal placement with a long abutment is preferred to minimized the extent of MBL, as if a short abutment is utilized, more MBL may be anticipated (Fig 4c). On the other hand, when thin mucosal height is present, the selection of a short abutment maximizes esthetics while compromising sufficient biologic dimensions for STAd formation. This potentially leads to greater MBL (Fig 4d). However, Linkevicius et al demonstrated that significantly greater MBL occurred when vertical mucosal thickness/supracrestal tissue height was ≤ 2 mm.[34–36] Based upon this concept, some authors recommended soft tissue grafting procedures for augmenting vertical mucosal height at sites with a thin phenotype when shallow placement is necessary, although evidence in support of this is limited.[37,38] Such procedures may permit selection of a longer abutment.[37,39]

Vervaeke demonstrated that planning implant vertical positioning (ie, subcrestal or equicrestal) based on soft tissue thickness was highly successful in avoiding

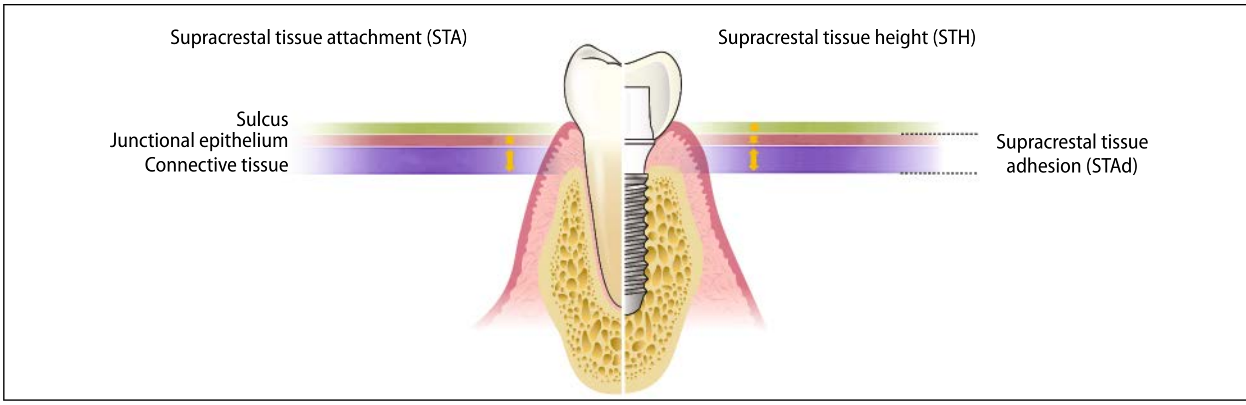
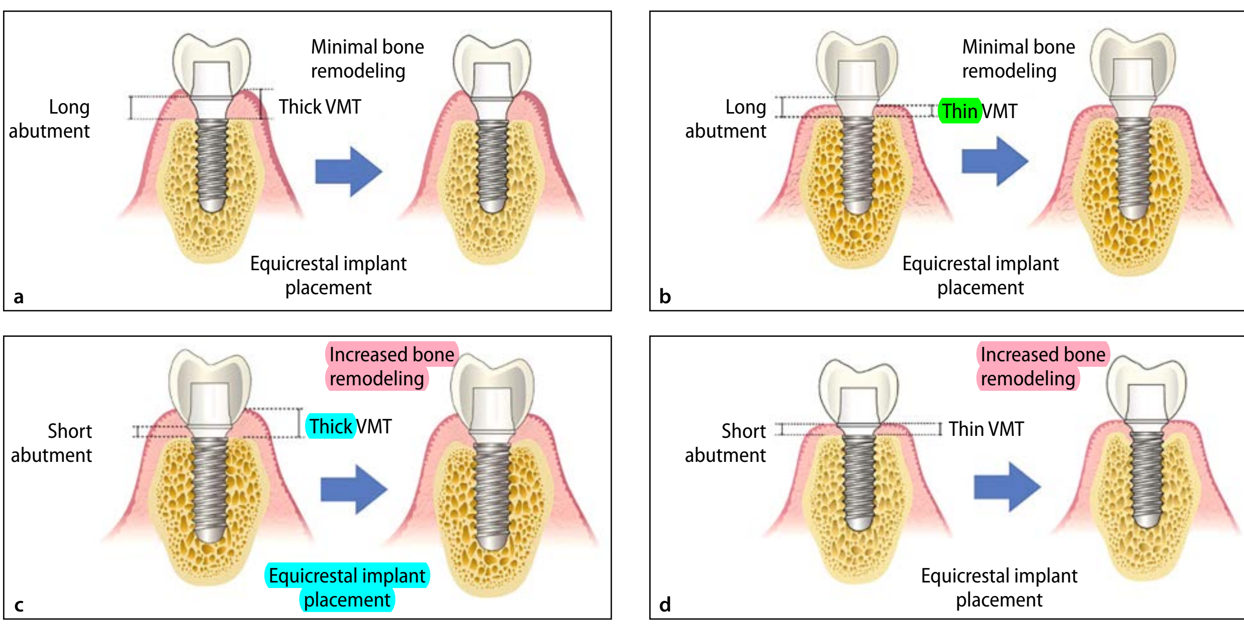


Fig 3 Illustration of the biologic structures around teeth and implants. Supracrestal tissue attachment (STA) describes the periodontal attachment apparatus for natural dentition because it possesses perpendicular fibers that attach to the tooth structure. Two terms will be used to describe the peri-implant supporting tissues. Supracrestal tissue height (STH), also known as vertical mucosal thickness, consists of sulcular depth, junctional epithelium, and connective tissue adhesion (STAd). The second term is supracrestal tissue adhesion (STAd), which specifically refers to junctional epithelium and connective tissue adhesion. It is important to note that around implants, the term *adhesion* is more accurate than *attachment*, as fibers do not attach to the implant surface, but instead adhere due to their parallel orientation.



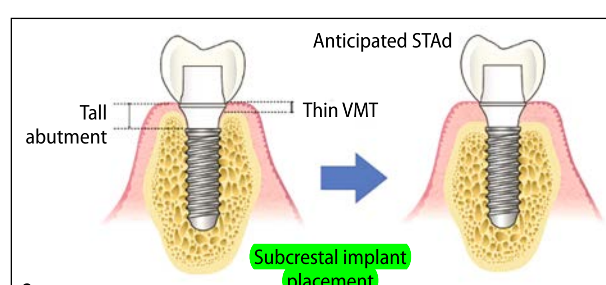


Fig 4 *(a)* Abutment height selection should be based on both apicocoronal implant positioning and mucosal thickness. Various articles have defined thin (≤ 2 mm) and thick (> 2 mm) vertical mucosal thickness (VMT),[32,36] as well as tall (≥ 2 mm) and short abutment heights (< 2 mm).[29,32,41] Scenarios a–d consider a clinical scenario involving equicrestal implant placement. a) In the presence of a thick VMT, a tall abutment height favors establishment of an ideal supracrestal tissue height (2 to 4 mm). In this case, minimal bone remodeling is expected. *(b)* The selection of a tall abutment in the presence of thin VMT has also been associated with reduced bone remodeling in the literature.[32] However, it should be noted that this scenario is not commonly implemented in clinical practice due to esthetic consequences associated with the supragingival position of the abutment-prosthetic margin. *(c)* Choice of a short abutment height in the presence of a tall VMT is a poor clinical choice and will likely result in increased bone remodeling. *(d)* The selection of a short abutment when thin VMT is present promotes esthetics but often leads to increased bone remodeling as a compensatory reaction. (e) In cases of thin VMT (such as in D), subcrestal placement may be utilized with bone-level systems to anticipate bone remodeling during establishment of STAd. This can potentiall reduce the risk of implant thread exposure.

implant surface exposure.[40] A similar concept was reported in a study by de Siqueira et al, where implants placed subcrestally with longer abutments (≥ 2.5 mm) did not exhibit thread exposure after 5 years of follow-up (Fig 4e).[31] Subcrestally placed implants facilitate adequate distance for establishment of an ideal STAd and may be associated with a reduced risk for thread exposure. This concept is valid for implants with abutment-fixture connections characterized by minimal micromovement. If an implant does not allow such features, MBL is expected to happen apical to the implant platform regardless of vertical implant position. The abutment height concept can be seen as the building block for analyzing outcomes of clinical studies reporting MBL. It should be noted that a key limitation of several studies on this topic was the absence of accurate soft tissue measurements.[33,41]

Challenging the relationship between vertical mucosal thickness/supracrestal tissue height and marginal bone loss, Spinato and coworkers showed in a randomized clinical trial that implants restored with short abutments (1 mm) consistently demonstrated twice the bone loss of identical implants restored with long abutments (3 mm), irrespective of vertical mucosal thickness (groups with < 2 mm or > 2 mm).[32] Clinically, the utilization of a long abutment (> 2 mm) may not be feasible if the implant is placed equicrestally in areas with thin vertical mucosal thickness due to the esthetic compromise. This would necessitate a more obtuse emergence profile and possibly expose the abutment surface above the mucosal margin.

Although the aforementioned evidence revealed the role of abutment height and supracrestal tissue height on MBL, long-term data on the effectiveness of this approach in reducing the risk of peri-implantitis is scarce. One consideration is that the deeper the position of the crown-abutment margin, the greater the prevalence of undetected cement.[42] The authors reported that the greatest quantity of cement remnants was found when margins were positioned 2 to 3 mm subgingivally. Consequently, the balance between vertical implant positioning and abutment height must be considered to minimize the risk for retained cement after crown delivery.

BIOLOGIC RESPONSE OF PERI-IMPLANT TISSUES TO SUBGINGIVAL CEMENT

It has been well documented that subgingival cement remnants can contribute to the incidence of peri-implant diseases.[43] Cement acts as a nidus for bacterial colonization, leading to inflammation in the peri-implant supporting tissues and in some cases to bone loss.[44] The rough cement surface enhances biofilm adherence and impedes cleanability of the implant surface.[45]

The biologic mechanisms by which the retention of cement leads to peri-implant diseases remain unclear. Two possible explanations are that residual cement can cause mechanical irritation[45] as well as induce a foreign body reaction in the peri-implant tissues.[46] The mechanical irritation hypothesis resembles the scientifically validated concept of invasion of the STAd by deep restorative margins. Future studies in this area are needed to validate these theories. Interestingly, the theory that initiation of peri-implantitis is primarily due to a foreign body reaction stimulated by both the surgical implant placement procedure and subsequent prosthetic rehabilitation, and not a biofilm-mediated inflammatory process, was presented by Albrektsson et al.[47] During crown delivery, excess cement can potentially travel apically into the sulcus, following the direction of least resistance due to hydraulic pressure.[48] Subgingivally retained cement around the natural dentition is an uncommon finding; this may be due to the fact that peri-implant probing depths are on average deeper than those around teeth, and key differences exist in the anatomy and physiology of the peri-implant and periodontal supporting tissues.[43]

A distinction should be made between prospective articles reporting the prevalence of peri-implantitis around implants restored with cement- versus screw-retained crowns, and retrospective/cross-sectional studies investigating the proportion of diseased implants often displaying excess cement.[49,50] This is important because in many studies cement-retained restorations were not found to be at a higher risk for peri-implantitis compared to screw-retained prostheses.[1,51] This means that using a cement-retained prosthesis is not a risk factor, but its undetected invasion of the peri-implant sulcus promotes a chronic inflammatory response. Additionally, some studies analyzed excess cement by radiographic examination,[52–54] which is an unreliable method. Indeed, two-dimensional radiographs do not allow detection of buccal and lingual excess cement and have limited capacity in the detection of interproximally retained cement.[55]

There is limited evidence to support the notion that different cement properties may play a role in the biologic consequences of peri-implant subgingival retention. Indeed, methacrylate-based cements favor plaque formation, while zinc oxide–eugenol cements can dissolve over time and exhibit antimicrobial properties.[49] Once subgingivally retained cement has set, it is difficult to remove. No difference in utilizing metal and plastic instruments has been seen in terms of cement removal efficiency, and both instruments may potentially cause damage to the implant surface

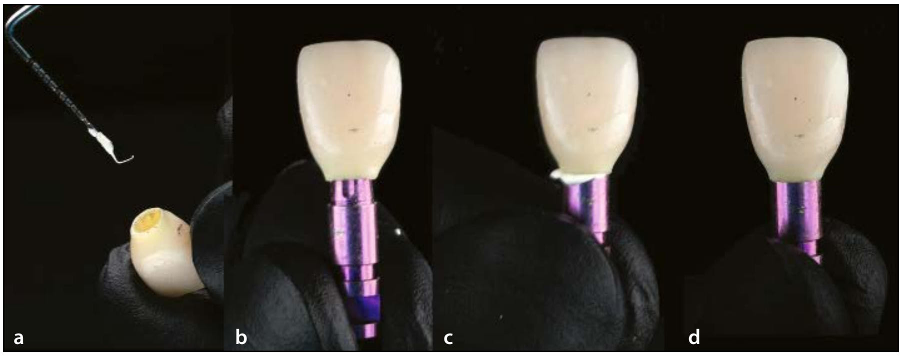


Fig 5 Clinical user-friendly and effective cementation technique that involves placing luting agent on the intaglio surface of the restoration *(a)* and seating the crown on an implant analog prior to cementation in the mouth *(b)*. The excess cement can be cleaned extraorally around the analog, resulting in the crown containing only a thin layer of cement limited to the intaglio surface prior to intraoral delivery *(c and d)*.

if mishandled.[56] In addition, use of plastic instruments for implant debridement may result in plastic particle remnants.[57]

In 2002, Dumbrigue et al discussed two different approaches to control the amount of cement inside the crown prior to cementation.[58] The first approach involves placing luting agent only on the occlusal portion of the intaglio surface of the restoration to avoid flow of the cement subgingivally. The second involves an extraoral trial cementation, during which the crown is seated on an implant analog and then removed immediately. Excess cement at this point is wiped off, resulting in the crown containing only a thin layer of cement limited to the intaglio surface prior to intraoral delivery (Fig 5). Flossing using a crisscross technique has shown effectiveness in reducing retained cement during implant crown delivery.[59] At this moment, none of the approaches and instruments can guarantee perfect cement removal; hence, strategies aimed at preventing accumulation of subgingival cement instead of those designed to remove it are preferred. Combined screw- and cement-retained restorations can be used to avoid subgingival cement accumulation as the cementation process takes place outside the oral cavity.[60] However, these restorations still require a screw access hole at the surface of the restoration, and thus may be associated with a higher risk of prosthetic complications such as porcelain fracture compared to traditional cemented restorations.[61]

TO SPLINT OR NOT TO SPLINT?

Splinting implant-supported prostheses is not always a straightforward decision and is a common source of debate among clinicians. On the one hand, it allows for a more even distribution of occlusal forces, leading to fewer prosthetic complications; on the other, maintenance of proper hygiene is more complex.[62] Indeed, Serino and Ström reported that 4% of patients with prosthetic designs allowing adequate access for hygiene developed peri-implantitis, whereas patients without proper access developed peri-implantitis at 48% of sites. Interestingly, the majority of patients who presented with peri-implantitis exhibited a good level of plaque control for their remaining natural dentition.[63] A long-term randomized clinical trial comparing three non-splinted implants versus three splinted implants reported no incidence of peri-implantitis during the follow-up period.[64] On the other hand, in articles with a different methodology (retrospective and cross-sectional) that might better resemble daily clinical practice, a higher prevalence of peri-implantitis has been found for splinted implants, especially at the central implant, where access for hygiene is usually the most difficult.[18,65]

One of the important challenges for splinting multiple implants involves achieving a passively fitting superstructure. Increased plaque accumulation has been associated with clinical scenarios characterized by misfits between the implant and the overlying prosthesis.[66] Misfits are also accompanied by a higher degree of stress at the bone-implant-prosthetic interfaces, which can lead to technical (ie, screw loosening or prosthetic fracture) and biologic consequences (ie, MBL).[66] Clinical studies reported excellent outcomes when frameworks are cemented to prefabricated cylinders, as they exhibit precise adaptation to abutments or the implant platform, minimizing deformation of prosthetic materials inherent during laboratory steps.[67,68] Today, with the introduction of digital technologies for impression making and prosthetic fabrication, the risk of misfit is expected to be reduced. Dental implant restorations fabricated digitally have been shown to be more precise than those produced by a casting technique.[69,70] A recent clinical study on splinted restorations supported by two implants revealed that restorations fabricated digitally had a better fit on the control cast. However, no difference in passive fit was found intraorally.[71] Due to the diversity of influencing factors and lack of reliable measures to evaluate the impact of misfit on biologic complications, there is no clear consensus on the "amount of fit" that is needed to prevent biofilm

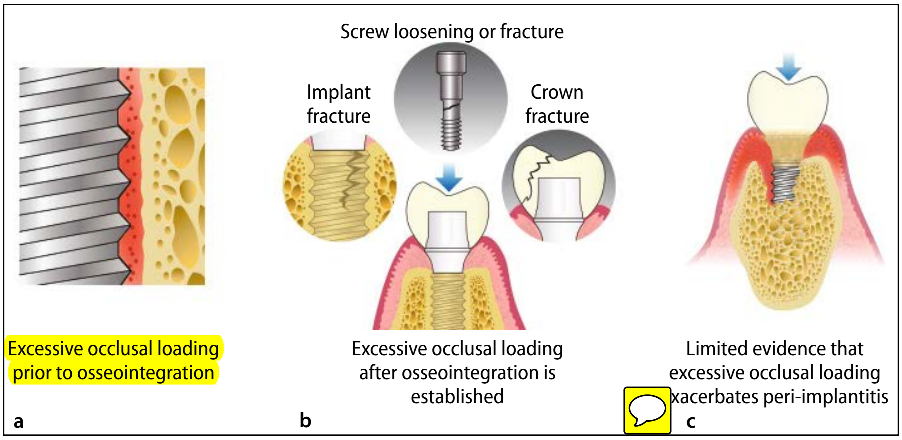


Fig 6 *(a)* Fibrous encapsulation due to excessive occlusal loading prior to establishment of osseointegration. *(b)* After osseointegration is established, prosthetic complications and peri-implant bone densification are possible responses to high occlusal forces in the absence of plaque-induced disease. *(c)* There is inconclusive and limited evidence supporting the notion that excessive occlusal forces exacerbate peri-implantitis.

accumulation. The decision to splint or not to splint should take into account the need for an even distribution of occlusal forces to deliver a well-fitted restoration with a cleanable design.

ARE OCCLUSAL FORCES RELATED TO PERI-IMPLANT DISEASE PATHOGENESIS?

The potential influence of excessive occlusal forces on periodontitis and peri-implantitis is a controversial subject without clear evidence to support definitive conclusions. The 2017 World Workshop concluded that the evidence clearly demonstrates that occlusal trauma does not initiate periodontal disease, and that although a number of animal and clinical studies show an association between occlusal trauma and progression of periodontitis, the overall quality of this evidence is weak.[72] The biomechanical responses of teeth and implants to occlusal forces exhibit distinct differences due to how they are connected to the surrounding bone.[73]

Before exploring the potential association between excessive occlusal forces and peri-implant disease pathogenesis, it is important to first understand how occlusal forces play a role in peri-implant bone remodeling in the absence of plaque-induced inflammatory disease. Establishment of osseointegration begins 2 to 3 weeks after implant placement, during which time there is a transition from primary (mechanical) stability toward secondary (biologic) stability as bone remodeling occurs.[74] During initial healing, excessive micromovement due to either a lack of mechanical stability or the influence of occlusal forces may result in fibrous encapsulation and failure to achieve osseointegration (Fig 6a).[75,76] After secondary stability (osseointegration) is established, orthodontic-like forces have been demonstrated to directly impact peri-implant bone turnover and cellular activity,[77] resulting in augmented peri-implant bone density near areas of moderate strain, whereas extreme values of either low or high strain resulted in reduced bone density.[78] In an animal study, Lima and coworkers reported significantly increased radiographic and histologic bone density in response to excessive occlusal forces relative to unloaded implants and those with stable occlusal contacts.[79] The same study also reported a higher incidence of screw loosening and abutment fracture in response to excessive nonaxial occlusal contacts, indicating that prosthetic complications are more likely in this scenario (Fig 6b). Studies investigating functional loading of osseointegrated implants with physiologic forces have corroborated these results, demonstrating increased bone-to-implant contact relative to unloaded controls.[80]

If repetitive biomechanical forces are increased up to within a certain range, then this may result in net bone apposition as an adaptive response, enabling the bone to better deal with the increased stress.[81] In rare cases of excessive occlusal load in a predominantly lateral direction, complete loss of osseointegration of dental implants has been reported, as in two animal studies conducted by Isidor, characterized clinically by mobility and peri-implant radiolucency,[82] and histologically by fibrous encapsulation.[83] In another study, excessive occlusal forces acting on osseointegrated dental implants with supraocclusal restorative contacts resulted in no significant differences in terms of bleeding on probing or pocket depth measurements, histologic bone-to-implant contact, marginal bone levels, or bone density relative to unloaded controls in the presence of adequate plaque control.[84] Other work on static lateral forces has suggested that implant surface modifications may modulate stress transfer to the surrounding bone, with rough surfaces exhibiting a more favorable peri-implant bone reaction than machined surfaces in terms of bone-to-implant contact, bone density, and the propensity to develop vertical bony defects.[85]

Implant length may also play a role in the response of peri-implant bone to functional loading. Human clinical studies have demonstrated increased peri-implant bone density for short (6 mm) relative to long implants (10 mm) after 3 to 5 years of functional loading.[86,87]

Due to the ethical issues associated with conducting prospective human clinical studies analyzing the effects of occlusal trauma on plaque-associated peri-implant disease, the majority of relevant findings are based on animal studies or cross-sectional/retrospective analyses.[4] Kozlovsky et al conducted a split-mouth study in beagle dogs with ligature-induced peri-implantitis on one side versus regular oral hygiene on the other.[88] Different abutment heights were used to create two loading conditions: supraocclusion and infraocclusion (unloaded). Thus, four clinical scenarios were present in each dog: unloaded inflamed, loaded inflamed, unloaded uninflamed, and loaded uninflamed. In the absence of inflammation, excessive loading significantly increased bone-to-implant contact in the unloaded uninflamed and loaded uninflamed groups, whereas in the presence of inflammation, overloading had no such effect, and an increased tendency for bone loss resulting in thread exposure was reported. Since crestal bone changes were not measured, it is difficult to extrapolate the data based on these results.

Dalago et al conducted a cross-sectional study in humans and found a significantly elevated risk for peri-implantitis by 2.4-fold if wear facets were found on implant-supported restorations.[89] Retrospective analysis of 332 implants placed in 56 patients with at least one implant diagnosed with peri-implantitis revealed a 15× higher prevalence of prosthetic complications such as abutment fracture, loss of retention, and chipping at sites with peri-implantitis compared to healthy sites.

Together, these findings suggest that in cases of progressive peri-implant MBL, the etiology is primarily microbial in nature with limited evidence to support the notion that occlusal forces act as a modifying factor (Fig 6c), although prosthetic complications are more likely when implants are subjected to excessive occlusal forces.

LIMITATIONS

It is important to note the limitations of this narrative review. Firstly, it would be ideal to conduct a systematic review and/or meta-analysis to eliminate potential bias or personal opinions; however, there is currently an insufficient number of randomized controlled trials available to allow for meaningful statistical analyses. Hence, one of the main goals of this manuscript is to stimulate more insightful research on the linkages between peri-implant health and prosthetic factors. Secondly, we did not address the potential complications related to implant-abutment mismatch, fixture/abutment fatigue fracture, or zirconia implant-related complications because we do not have adequate information to provide a meaningful assessment on these topics. Future studies in these topics are encouraged.

CONCLUSIONS

Current evidence shows that the prosthesis is significantly intertwined with the biologic response of the peri-implant supporting tissues. Each step of the restorative process is important in determining long-term prosthetic and biologic outcomes. These factors do not act in isolation but instead interact to directly influence biologic outcomes. Careful analysis and treatment planning are key to fabricating a prosthesis that is harmonious with a state of peri-implant health, allowing clinicians to take advantage of the prosthetic-biologic connection to decrease the risk for peri-implant diseases.

ACKNOWLEDGMENTS

ORCID for Dr Hom-Lay Wang: 0000-0003-4238-1799. The authors report no conflicts of interest.

REFERENCES

1. Derks J, Schaller D, Håkansson J, Wennström JL, Tomasi C, Berglundh T. Effectiveness of implant therapy analyzed in a Swedish population: Prevalence of peri-implantitis. J Dent Res 2016;95:43–49.
2. Derks J, Tomasi C. Peri-implant health and disease. A systematic review of current epidemiology. J Clin Periodontol 2015;42(suppl 16):s158–s171.
3. Berglundh T, Armitage G, Araujo MG, et al. Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Clin Periodontol 2018;45(suppl 20):s286–s291.
4. Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. J Periodontol 2018;89(suppl 1):s267–s290.
5. Garber DA. The esthetic dental implant: Letting restoration be the guide. J Am Dent Assoc 1995;126:319–325.
6. Greenwell H, Wang HL, Kornman KS, Tonetti MS. Biologically guided implant therapy: A diagnostic and therapeutic strategy of conservation and preservation based on periodontal staging and grading. J Periodontol 2019;90:441–444.
7. Sanz-Martín I, Regidor E, Navarro J, Sanz-Sánchez I, Sanz M, Ortiz-Vigón A. Factors associated with the presence of peri-implant buccal soft tissue dehiscences: A case-control study [epub ahead of print 24 Jan 2020]. J Periodontol 2020;91:1003–1010.
8. Spray JR, 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.
9. Hämmerle CHF, Tarnow D. The etiology of hard- and soft-tissue deficiencies at dental implants: A narrative review. J Clin Periodontol 2018;89(suppl 1):s291–s303.
10. Zucchelli G, Tavelli L, Stefanini M, et al. Classification of facial peri-implant soft tissue dehiscence/deficiencies at single implant sites in the esthetic zone. J Periodontol 2019;90:1116–1124.
11. Katafuchi M, Weinstein BF, Leroux BG, Chen YW, Daubert DM. Restoration contour is a risk indicator for peri-implantitis: A cross-sectional radiographic analysis. J Clin Periodontol 2018;45:225–232.

12. Cho-Yan Lee J, Mattheos N, Nixon KC, Ivanovski S. Residual periodontal pockets are a risk indicator for peri-implantitis in patients treated for periodontitis. Clin Oral Implants Res 2012;23:325–333.
13. Pjetursson BE, Helbling C, Weber HP, et al. Peri-implantitis susceptibility as it relates to periodontal therapy and supportive care. Clin Oral Implants Res 2012;23:888–894.
14. Saleh MHA, Ravidà A, Suárez-López Del Amo F, Lin GH, Asa'ad F, Wang HL. The effect of implant-abutment junction position on crestal bone loss: A systematic review and meta-analysis. Clin Implant Dent Relat Res 2018;20:617–633.
15. Monje A, Pons R, Insua A, Nart J, Wang HL, Schwarz F. Morphology and severity of peri-implantitis bone defects. Clin Implant Dent Relat Res 2019;21:635–643.
16. Tuna T, Kuhlmann L, Bishti S, Sirazitdinova E, Deserno T, Wolfart S. Removal of simulated biofilm at different implant crown designs with interproximal oral hygiene aids: An in vitro study. Clin Oral Implants Res 2019;30:627–636.
17. O'Mahony A, MacNeill SR, Cobb CM. Design features that may influence bacterial plaque retention: A retrospective analysis of failed implants. Quintessence Int 2000;31:249–256.
18. Yi Y, Koo KT, Schwarz F, Ben Amara H, Heo SJ. Association of prosthetic features and peri-implantitis: A cross-sectional study. J Clin Periodontol 2020;47:392–403.
19. Pico A, Martín-Lancharro P, Caneiro L, Nóvoa L, Batalla P, Blanco J. Influence of abutment height and implant depth position on interproximal peri-implant bone in sites with thin mucosa: A 1-year randomized clinical trial. Clin Oral Implants Res 2019;30:595–602.
20. Blanco J, Pico A, Caneiro L, Nóvoa L, Batalla P, Martín-Lancharro P. Effect of abutment height on interproximal implant bone level in the early healing: A randomized clinical trial. Clin Oral Implants Res 2018;29:108–117.
21. Vacek JS, Gher ME, Assad DA, Richardson AC, Giambarresi LI. The dimensions of the human dentogingival junction. Int J Periodontics Restorative Dent 1994;14:154–165.
22. Jepsen S, Caton JG, Albandar JM, et al. Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Periodontol 2018;89(suppl 1):s237–s248.
23. Oh TJ, Yoon J, Misch CE, Wang HL. The causes of early implant bone loss: Myth or science? J Periodontol 2002;73:322–333.
24. Tatarakis N, Bashutski J, Wang HL, Oh TJ. Early implant bone loss: Preventable or inevitable? Implant Dent 2012;21:379–386.
25. Avila-Ortiz G, Gonzalez-Martin O, Couso-Queiruga E, Wang HL. The peri-implant phenotype. J Periodontol 2020;91:283–288.
26. Judgar R, Giro G, Zenobio E, et al. Biological width around one- and two-piece implants retrieved from human jaws. Biomed Res Int 2014;2014:850120.
27. Tomasi C, Tessarolo F, Caola I, Wennström J, Nollo G, Berglundh T. Morphogenesis of peri-implant mucosa revisited: An experimental study in humans. Clin Oral Implants Res 2014;25:997–1003.
28. Berglundh T, Lindhe J. Dimension of the periimplant mucosa. Biological width revisited. J Clin Periodontol 1996;23:971–973.
29. Galindo-Moreno P, León-Cano A, Ortega-Oller I, et al. Prosthetic abutment height is a key factor in peri-implant marginal bone loss. J Dent Res 2014;93(7 suppl):80s–85s.
30. Borges T, Leitão B, Pereira M, Carvalho Á, Galindo-Moreno P. Influence of the abutment height and connection timing in early peri-implant marginal bone changes: A prospective randomized clinical trial. Clin Oral Implants Res 2018;29:907–914.
31. de Siqueira RAC, Savaget Gonçalves R Junior, Dos Santos PGF, de Mattias Sartori IA, Wang HL, Fontão FNGK. Effect of different implant placement depths on crestal bone levels and soft tissue behavior: A 5-year randomized clinical trial. Clin Oral Implants Res 2020;31:282–293.
32. Spinato S, Stacchi C, Lombardi T, Bernardello F, Messina M, Zaffe D. Biological width establishment around dental implants is influenced by abutment height irrespective of vertical mucosal thickness: A cluster randomized controlled trial. Clin Oral Implants Res 2019;30:649–659.
33. Vervaeke S, Dierens M, Besseler J, De Bruyn H. The influence of initial soft tissue thickness on peri-implant bone remodeling. Clin Implant Dent Relat Res 2014;16:238–247.
34. Linkevicius T, Apse P, Grybauskas S, Puisys A. The influence of soft tissue thickness on crestal bone changes around implants: A 1-year prospective controlled clinical trial. Int J Oral Maxillofac Implants 2009;24:712–719.
35. Linkevicius T, Apse P, Grybauskas S, Puisys A. Influence of thin mucosal tissues on crestal bone stability around implants with platform switching: A 1-year pilot study. J Oral Maxillofac Surg 2010;68:2272–2277.
36. Linkevicius T, Puisys A, Steigmann M, Vindasiute E, Linkeviciene L. Influence of vertical soft tissue thickness on crestal bone changes around implants with platform switching: A comparative clinical study. Clin Implant Dent Relat Res 2015;17:1228–1236.
37. Puisys A, Linkevicius T. The influence of mucosal tissue thickening on crestal bone stability around bone-level implants. A prospective controlled clinical trial. Clin Oral Implants Res 2015;26:123–129.
38. Zucchelli G, Mazzotti C, Bentivogli V, Mounssif I, Marzadori M, Monaco C. The connective tissue platform technique for soft tissue augmentation. Int J Periodontics Restorative Dent 2012;32:665–675.
39. Puisys A, Vindasiute E, Linkevciene L, Linkevicius T. The use of acellular dermal matrix membrane for vertical soft tissue augmentation during submerged implant placement: A case series. Clin Oral Implants Res 2015;26:465–470.
40. Vervaeke S, Matthys C, Nassar R, Christiaens V, Cosyn J, De Bruyn H. Adapting the vertical position of implants with a conical connection in relation to soft tissue thickness prevents early implant surface exposure: A 2-year prospective intra-subject comparison. J Clin Periodontol 2018;45:605–612.
41. Galindo-Moreno P, León-Cano A, Monje A, Ortega-Oller I, O'Valle F, Catena A. Abutment height influences the effect of platform switching on peri-implant marginal bone loss. Clin Oral Implants Res 2016;27:167–173.
42. Linkevicius T, Vindasiute E, Puisys A, Peciuliene V. The influence of margin location on the amount of undetected cement excess after delivery of cement-retained implant restorations. Clin Oral Implants Res 2011;22:1379–1384.
43. Gerber JA, Tan WC, Balmer TE, Salvi GE, Lang NP. Bleeding on probing and pocket probing depth in relation to probing pressure and mucosal health around oral implants. Clin Oral Implants Res 2009;20:75–78.
44. Staubli N, Walter C, Schmidt JC, Weiger R, Zitzmann NU. Excess cement and the risk of peri-implant disease—A systematic review. Clin Oral Implants Res 2017;28:1278–1290.
45. Wilson TG, Jr., Valderrama P, Rodrigues DB. The case for routine maintenance of dental implants. J Periodontol 2014;85:657–660.
46. Wilson TG Jr, Valderrama P, Burbano M, et al. Foreign bodies associated with peri-implantitis human biopsies. J Periodontol 2015;86:9–15.
47. Albrektsson T, Dahlin C, Jemt T, Sennerby L, Turri A, Wennerberg A. Is marginal bone loss around oral implants the result of a provoked foreign body reaction? Clin Implant Dent Relat Res 2014;16:155–165.
48. Patel D, Invest JC, Tredwin CJ, Setchell DJ, Moles DR. An analysis of the effect of a vent hole on excess cement expressed at the crown-abutment margin for cement-retained implant crowns. J Prosthodont 2009;18:54–59.
49. Korsch M, Obst U, Walther W. Cement-associated peri-implantitis: A retrospective clinical observational study of fixed implant-supported restorations using a methacrylate cement. Clin Oral Implants Res 2014;25:797–802.
50. Korsch M, Walther W. Peri-implantitis associated with type of cement: A retrospective analysis of different types of cement and their clinical correlation to the peri-implant tissue. Clin Implant Dent Relat Res 2015;17(suppl 2):e434–e443.
51. Kotsakis GA, Zhang L, Gaillard P, Raedel M, Walter MH, Konstantinidis IK. Investigation of the association between cement retention and prevalent peri-implant diseases: A cross-sectional study. J Periodontol 2016;87:212–220.
52. Ferreiroa A, Peñarrocha-Diago M, Pradíes G, Sola-Ruiz MF, Agustín-Panadero R. Cemented and screw-retained implant-supported single-tooth restorations in the molar mandibular region: A retrospective comparison study after an observation period of 1 to 4 years. J Clin Exp Dent 2015;7:e89–e94.

53. Gapski R, Neugeboren N, Pomeranz AZ, Reissner MW. Endosseous implant failure influenced by crown cementation: A clinical case report. Int J Oral Maxillofac Implants 2008;23:943–946.
54. Tomson PL, Butterworth CJ, Walmsley AD. Management of peri-implant bone loss using guided bone regeneration: A clinical report. J Prosthet Dent 2004;92:12–16.
55. Linkevicius T, Puisys A, Vindasiute E, Linkeviciene L, Apse P. Does residual cement around implant-supported restorations cause peri-implant disease? A retrospective case analysis. Clin Oral Implants Res 2013;24:1179–1184.
56. Agar JR, Cameron SM, Hughbanks JC, Parker MH. Cement removal from restorations luted to titanium abutments with simulated subgingival margins. J Prosthet Dent 1997;78:43–47.
57. Sirinirund B, Garaicoa-Pazmino C, Wang HL. Effects of mechanical instrumentation with commercially available instruments used in supportive peri-implant therapy: An in vitro study. Int J Oral Maxillofac Implants 2019;34:1370–1378.
58. Dumbrigue HB, Abanomi AA, Cheng LL. Techniques to minimize excess luting agent in cement-retained implant restorations. J Prosthet Dent 2002;87:112–114.
59. Ferreira CF, Shafter M, Jain V, Wicks RA, Linder E, da Silva Ledo CA. Evaluation of effectiveness of cement removal from implant-retained crowns using a proposed "circular crisscross" flossing technique. J Oral Implantol 2018;44:177–183.
60. Preiskel HW, Tsolka P. Cement- and screw-retained implant-supported prostheses: Up to 10 years of follow-up of a new design. Int J Oral Maxillofac Implants 2004;19:87–91.
61. Al-Omari WM, Shadid R, Abu-Naba'a L, El Masoud B. Porcelain fracture resistance of screw-retained, cement-retained, and screw-cement-retained implant-supported metal ceramic posterior crowns. J Prosthodont 2010;19:263–273.
62. Ravidà A, Saleh MHA, Muriel MC, Maska B, Wang HL. Biological and technical complications of splinted or nonsplinted dental implants: A decision tree for selection. Implant Dent 2018;27:89–94.
63. Serino G, Ström C. Peri-implantitis in partially edentulous patients: Association with inadequate plaque control. Clin Oral Implants Res 2009;20:169–174.
64. Vigolo P, Mutinelli S, Zaccaria M, Stellini E. Clinical evaluation of marginal bone level change around multiple adjacent implants restored with splinted and nonsplinted restorations: A 10-year randomized controlled trial. Int J Oral Maxillofac Implants 2015;30:411–418.
65. Ravidà A, Tattan M, Askar H, Barootchi S, Tavelli L, Wang HL. Comparison of three different types of implant-supported fixed dental prostheses: A long-term retrospective study of clinical outcomes and cost-effectiveness. Clin Oral Implants Res 2019;30:295–305.
66. Katsoulis J, Takeichi T, Sol Gaviria A, Peter L, Katsoulis K. Misfit of implant prostheses and its impact on clinical outcomes. Definition, assessment and a systematic review of the literature. Eur J Oral Implantol 2017;10(suppl 1):121–138.
67. Able FB, de Mattias Sartori IA, Thomé G, Moreira Melo AC. Retrospective, cross-sectional study on immediately loaded implant-supported mandibular fixed complete-arch prostheses fabricated with the passive fit cementation technique. J Prosthet Dent 2018;119:60–66.
68. Borges AF, Dias Pereira LA, Thomé G, Melo AC, de Mattias Sartori IA. Prostheses removal for suture removal after immediate load: Success of implants. Clin Implant Dent Relat Res 2010;12:244–248.
69. de Araújo GM, de França DG, Silva Neto JP, Barbosa GA. Passivity of conventional and CAD/CAM fabricated implant frameworks. Braz Dent J 2015;26:277–283.
70. de França DG, Morais MH, das Neves FD, Carreiro AF, Barbosa GA. Precision fit of screw-retained implant-supported fixed dental prostheses fabricated by CAD/CAM, copy-milling, and conventional methods. Int J Oral Maxillofac Implants 2017;32:507–513.
71. Rutkunas V, Larsson C, Vult von Steyern P, Mangano F, Gedrimiene A. Clinical and laboratory passive fit assessment of implant-supported zirconia restorations fabricated using conventional and digital workflow. Clin Implant Dent Relat Res 2020;22:237–245.
72. Fan J, Caton JG. Occlusal trauma and excessive occlusal forces: Narrative review, case definitions, and diagnostic considerations. J Periodontol 2018;89(suppl 1):s214–s222.
73. Kim Y, Oh TJ, Misch CE, Wang HL. Occlusal considerations in implant therapy: Clinical guidelines with biomechanical rationale. Clin Oral Implants Res 2005;16:26–35.
74. Raghavendra S, Wood MC, Taylor TD. Early wound healing around endosseous implants: A review of the literature. Int J Oral Maxillofac Implants 2005;20:425–431.
75. Monje A, Ravidà A, Wang HL, Helms JA, Brunski JB. Relationship between primary/mechanical and secondary/biological implant stability. Int J Oral Maxillofac Implants 2019;34:s7–s23.
76. Wazen RM, Currey JA, Guo H, Brunski JB, Helms JA, Nanci A. Micromotion-induced strain fields influence early stages of repair at bone-implant interfaces. Acta Biomater 2013;9:6663–6674.
77. Gotfredsen K, Berglundh T, Lindhe J. Bone reactions adjacent to titanium implants subjected to static load. A study in the dog (I). Clin Oral Implants Res 2001;12:1–8.
78. Melsen B, Lang NP. Biological reactions of alveolar bone to orthodontic loading of oral implants. Clin Oral Implants Res 2001;12:144–152.
79. Lima LA, Bosshardt DD, Chambrone L, Araújo MG, Lang NP. Excessive occlusal load on chemically modified and moderately rough titanium implants restored with cantilever reconstructions. An experimental study in dogs. Clin Oral Implants Res 2019;30:1142–1154.
80. Berglundh T, Abrahamsson I, Lindhe J. Bone reactions to longstanding functional load at implants: An experimental study in dogs. J Clin Periodontol 2005;32:925–932.
81. Teichtahl AJ, Wluka AE, Wijethilake P, Wang Y, Ghasem-Zadeh A, Cicuttini FM. Wolff's law in action: A mechanism for early knee osteoarthritis. Arthritis Res Ther 2015;17:207.
82. Isidor F. Loss of osseointegration caused by occlusal load of oral implants. A clinical and radiographic study in monkeys. Clin Oral Implants Res 1996;7:143–152.
83. Isidor F. Clinical probing and radiographic assessment in relation to the histologic bone level at oral implants in monkeys. Clin Oral Implants Res 1997;8:255–264.
84. Heitz-Mayfield LJ, Schmid B, Weigel C, et al. Does excessive occlusal load affect osseointegration? An experimental study in the dog. Clin Oral Implants Res 2004;15:259–268.
85. Gotfredsen K, Berglundh T, Lindhe J. Bone reactions adjacent to titanium implants with different surface characteristics subjected to static load. A study in the dog (II). Clin Oral Implants Res 2001;12:196–201.
86. Sahrmann P, Schoen P, Naenni N, Jung R, Attin T, Schmidlin PR. Peri-implant bone density around implants of different lengths: A 3-year follow-up of a randomized clinical trial. J Clin Periodontol 2017;44:762–768.
87. Sluka B, Naenni N, Jung RE, Attin T, Schmidlin PR, Sahrmann P. Changes of radiopacity around implants of different lengths: Five-year follow-up data of a randomized clinical trial. Clin Oral Implants Res 2020;31:488–494.
88. Kozlovsky A, Tal H, Laufer BZ, et al. Impact of implant overloading on the peri-implant bone in inflamed and non-inflamed peri-implant mucosa. Clin Oral Implants Res 2007;18:601–610.
89. Dalago HR, Schuldt Filho G, Rodrigues MA, Renvert S, Bianchini MA. Risk indicators for peri-implantitis. A cross-sectional study with 916 implants. Clin Oral Implants Res 2017;28:144–150.