

Influence of the Size of the Microgap on Crestal Bone Levels in Non-Submerged Dental Implants: A Radiographic Study in the Canine Mandible

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Background: Accumulating evidence suggests that alveolar crestal bone resorption occurs as a result of the microgap that is present between the implant–abutment interface in dental implants. The objective of this longitudinal radiographic study was to determine whether the size of the interface or the microgap between the implant and abutment influences the amount of crestal bone loss in unloaded non-submerged implants.

Methods: Sixty titanium implants having sandblasted with large grit, acid-etched (SLA) endosseous surfaces were placed in edentulous mandibular areas of 5 American fox hounds. Implant groups A, B, and C had a microgap between the implant–abutment connection of <10 μm , 50 μm , or 100 μm , respectively, as did groups D, E, and F, respectively. Abutments were either welded (1-piece) in groups A, B, and C or non-welded (2-piece screwed) in D, E, and F. All abutment interfaces were placed 1 mm above the alveolar crest. Radiographic assessment was undertaken to evaluate peri-implant crestal bone levels at baseline and at 1, 2, and 3 months after implant placement whereupon all animals were sacrificed.

Results: The size of the microgap at the abutment/implant interface had no significant effect upon crestal bone loss. At 1 month, most implants developed crestal bone loss compared with baseline levels. However, during this early healing period, the non-welded group (D, E, and F) showed significantly greater crestal bone loss from baseline to one month ($P < 0.04$) and 2 months ($P < 0.02$) compared with the welded group (A, B, and C). No significant differences were observed between these 2 groups at 3 months ($P > 0.70$).

Conclusions: Crestal bone loss was an early manifestation of wound healing occurring after 1 month of implant placement. However, the size of the microgap at the implant-abutment interface had no significant effect upon crestal bone resorption. Thus, 2-piece non-welded implants showed significantly greater crestal bone loss compared with 1-piece welded implants after 1 and 2 months suggesting that the stability of the implant/abutment interface may have an important early role to play in determining crestal bone levels. At 3 months, this influence followed a similar trend but was not observed to be statistically significant. This finding implies that implant configurations incorporating interfaces will be associated with biological changes regardless of interface size and that mobility between components may have an early influence on wound healing around the implant. *J Periodontol* 2002;73:1111-1117.

KEY WORDS

Dental abutments; dental implants, endosseous; alveolar bone loss/etiology; follow-up studies; wound healing.

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Criteria for implant success lie not only with the establishment of direct bone to implant contact¹ but also with the development of peri-implant hard and soft tissue margins with the coronal portion of the implant.^{2,3} Evidence suggests that the peri-implant tissues comprising the biological width around 1-piece dental implants are similar to that found in the natural dentition.⁴ This is important as bacteria found in the implant sulcus are similar to that found around a natural tooth.⁵ When natural teeth or implants develop periodontitis or peri-implantitis, respectively, a similar shift in microflora from aerobic to anaerobic microorganisms occurs.⁶ A protective barrier created by the coronal soft tissue connection around implants may, in the presence of good plaque control, act to prevent pocket formation and colonization of pathogenic bacteria leading to bone loss adjacent to the implant. Thus, alveolar bone is critical to the establishment of the biological width. Therefore, if crestal bone resorption occurs around the implant, this may result in a more apical position of the gingival margin.^{4,7,8} The development and the location of the biological width relative to the implant and the alveolar crest become all the more important when minimal alveolar bone is available to house the implant due to anatomical restrictions arising from structures such as the sinus floor, inferior alveolar canal, and the mental foramen or when the implant is placed in areas of esthetic concern. For example, subsequent crestal bone loss around implants after placement may produce unfavorable crown:implant ratios or expose metal collars.

Crestal bone resorption around 2-piece implants in humans has been reported to be inevitable with expected loss of 1.5 mm in the first year of loading⁹ and 0.1 mm in subsequent years.¹⁰ Various hypotheses for these crestal bone changes have been postulated, including the insult from the surgical procedure upon the periosteum and bone,¹¹ the size of the microgap between the implant–abutment connection,¹² bacterial colonization of the implant sulcus,⁶ development of the biological width,³ and biomechanical factors relating to mechanical loading resulting in force transfer at the implant to crestal bone tissue interface.¹³

Histometric studies indicate that different implant designs impact the dimensions of the biological width including the level of crestal bone around the implant.^{3,4} In particular, differences in the biological width have been reported between 1- and 2-piece implant designs.¹⁴ These 2 designs are sometimes referred to as submerged and non-submerged implants respectively. However, submerging or non-submerging 2-piece implants do not result in changes specifically at the crestal bone level.^{4,7} Rather, a fundamental difference between the 1- or 2-piece implants is the location of the abutment interface (i.e., the abutment connection with either an internal connection in

the one piece implant or the external connection such as the hex butt joint in the 2-piece implant) relative to the alveolar crestal bone.

Studies demonstrated that 2-piece implants uncovered after 3 months with subsequent abutment connection and creation of the interface or microgap developed significant alveolar crestal bone loss.^{4,7} When the implant interface was placed far enough above the crest, no bone loss occurred. However, the greatest crestal bone loss occurred with 2-piece implants when the interface was located below the crest rather than at or above the crestal bone level.^{4,7} Thus the presence and location of the abutment interface or microgap changes the dimensions of the peri-implant tissues in relation to any crestal bone resorption. Recent data also demonstrate that the observed osseous changes influence the location of the gingival margin and the dimensions of the biologic width.^{3,15}

The potential consequences of crestal bone resorption include an increase of the crown-implant ratio, biological width changes, and increased bacterial colonization subgingivally¹⁶ which could result in further alveolar bone loss. Moreover, since the microgap influences the level of crestal bone, it is possible that the size of the microgap and subsequent bacterial invasion between implant and abutment may exert a profound effect upon crestal bone levels. Additional evidence also suggests that the movement that occurs between the implant components during function as well as the movement created experimentally during tightening and loosening of abutments may also initiate alveolar crestal bone resorption.^{4,7,8} These forces on experimental implants may play a role in the development of crestal bone loss that appears to mimic the effects of micromovement and occlusal loading.

This study presents longitudinal radiographic data of a histometric study¹² that evaluated the effect of the microgap between implant and abutment on the crestal bone levels from non-decalcified tissue sections using an established dog model. The purpose of this longitudinal radiographic study was to determine whether the size of the interface or microgap between implant and abutment influences the rate and amount of crestal bone loss in unloaded 2-piece implants. This study also evaluated the effect of the forces on crestal bone levels attributable to the type of abutment connection as defined by 2-piece implants with welded abutments to the implant and 2-piece non-welded abutments to implants that received monthly abutment tightening and loosening.

MATERIALS AND METHODS

This study was approved by the Institutional Animal Care and Use Committee of the University of Texas Health Science Center at San Antonio. Five quaran-

tined laboratory bred, 2-year-old male American fox hounds weighing 30 to 35 kg were used.

Experimental Protocol

Six different cylindrical grade IV, commercially-pure, experimental titanium implants^{||} (A-F) were placed in dog mandibles. Each implant was 9 mm long including the abutment with an outer diameter of 4.1 mm (including threads) and 3.5 mm inner diameter. The surface of the implant apico-coronally was comprised of a rough SLA surface that extended 5 mm from the apex to the alveolar crest with a 1 mm smooth machined titanium surface at the coronal supraalveolar portion. The 3.5 mm long abutment was a smooth machined surface titanium cylinder. Implant groups A, B, and C had the abutments spot welded in 4 places to the implant while groups D, E, and F had abutments screwed in place, representing the non-welded 2-piece implants. Gaps of <10 μm , 50 μm , and 100 μm at the implant-abutment interface were created for the different implants. Implants were paired with respect to the gap created, termed the microgap, at the implant-abutment interface as follows: A and D: <10 μm ; B and E: 50 μm , and C and F: 100 μm . Thus groups A, B and C were the same as groups D, E, and F, respectively, except that the abutments were screwed into the latter group at the time of surgery. All implants had the microgap placed 1 mm above the alveolar crest.

Extractions

The bilateral lower 4 mandibular premolars (P_1 - P_4) and the first molar (M_1) were carefully extracted after root sectioning to prevent root fracture under general anesthesia after localized infiltration of 2% lidocaine HCl with epinephrine 1:100,000 around the teeth. Sockets were sutured to obtain primary wound healing. Animals received 4% thiopental-Na solution i.v. (0.4 mg/kg bw) for premedication, intubated and medicated with 1.5% to 2% isoflurane and monitored with an electrocardiogram during surgery. On the day of surgery, animals were given 20 mg nalbuphine subcutaneously (sc) and subsequently 10 mg/ml BID, 3 ml benzathine penicillin (150,000 IU) combined with procaine penicillin G (150,000 IU) sc SID every 7 to 10 days. Sutures were removed 7 to 10 days during brief anesthesia.

Implant Surgery

After 6 months healing after the extractions, animals were anesthetized and a mid-crestal incision with mucoperiosteal reflection to expose the alveolar crest was made. The crestal bone was flattened to achieve a plane that was 90° to the future implant long axis with a large bur under copious irrigation. Randomly assigned 6 implants (A-F) were placed on each side of the mandible following osteotomy site preparation.

An O-ring with a height of 1 mm aided the placement of the interface 1 mm above the crest of the flattened ridge. The abutments for the non-welded (D-F) implants were screwed into place. Tension-free mucoperiosteal flaps were sutured in place. Finally, silicone impressions were taken in custom made trays to fabricate individual stents to ensure standardized radiographic analysis.¹⁷ Dogs were fed a soft diet and mechanical and chemical plaque control with 0.2% chlorhexidine gel was carried out using a soft toothbrush and soft sponge.

Evaluation Period

At 7 to 10 days after surgery, baseline standardized periapical radiographs were taken while the dogs were under general anesthesia. Exposure parameters were 70 kVp, 15 mA at 0.25 seconds with a focal distance of 37 cm. Processing was done manually and carried out according to the manufacturer's recommendations. Standardized radiographs were taken at monthly intervals until 3 months.¹⁷ Abutment loosening and retightening of implants D, E, and F was performed at 4, 8, and 10 weeks after surgery. All animals were sacrificed at 3 months with an overdose of intravenous pentobarbital sodium (0.2 ml = 65 mg/kg bw). Block dissection of mandibles containing the implants was obtained and immersed in 4% formaldehyde combined with 1% CaCl_2 for histological analysis as previously reported.¹²

Radiographs were digitized by converting them to 640 × 480 pixels using a calibrated video camera and a 50 mm lens. Prior to capturing the image, transillumination was adjusted to best visualize the bony margin of the most coronal area adjacent to the implant. Linear measurement from the top of the implant to the first bone implant contact (Top:fBIC) was obtained both mesially and distally using a software program to analyze each calibrated image. If Top:fBIC was observed both mesially and distally at all time points, the implant was included in the analysis and the mesial and distal values were averaged resulting in a single Top:fBIC value per implant and time point. A difference in the score of Top:fBIC at 1, 2, and 3 months minus baseline Top:fBIC was then calculated, representing crestal bone loss after implant placement. The crestal bone loss measures were then analyzed using a mixed-model analysis of variance (ANOVA) to check if implant types differed in a consistent fashion for each dog. The mixed-model ANOVA tested the main effects of welded/non-welded, microgap (interface) size and follow-up month as well as all interactions among these 3 effects, with all results adjusted for any dog effect. If any of the F-tests were significant ($P < 0.05$), then relevant pairwise comparisons, using unpaired Student *t*

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tests adjusted for any dog effect were performed to identify differences across implant types (A-F).

RESULTS

Clinical and Radiographic Assessment

All 60 implants were successfully placed and all were evaluated monthly radiographically in a standardized fashion for the duration of the study. All implants maintained clinical stability throughout the course of the study. Light microscopic evaluation of the bone to implant contact indicated hard tissue integration was successful.¹²

Radiographic Data

Implants were analyzed monthly by measuring the mesial and distal surfaces of the 60 implant sites up to 3 months after placement (Figs. 1 and 2). The edentulous ridge, which followed the natural slope of the arch that had been leveled prior to implant placement, occasionally resulted in a slight differential in bone level on the mesial and distal surfaces of the implant. Several radiographs failed to show the most distal surface of the implants due to the difficulty of obtaining access. Measurements were taken from the top of the implant to the fBIC as it was often impossible to see the interface radiographically. Since the top of the implant to the microgap was 3.5 mm long, values were

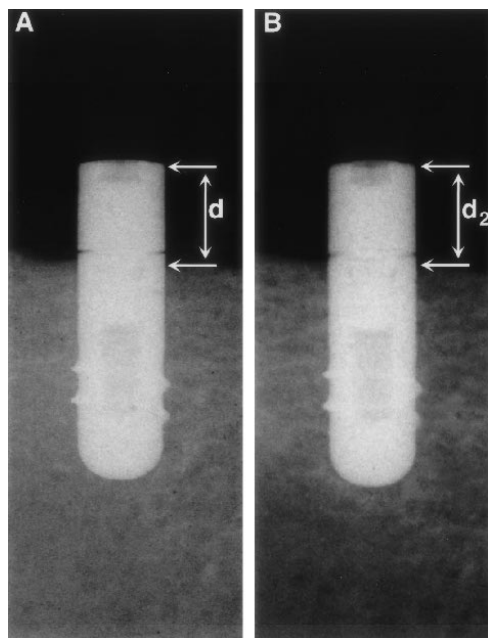


Figure 1.

Standardized periapical radiographs taken at baseline (A) and at 3 months (B) after implant placement of a welded implant design. Vertical linear measurements were made between the top of the abutment and the first bone-to-implant contact. The change in bone level was determined by subtracting the distance in the follow-up radiographs (d_2) from the distance in the baseline radiographs (d).

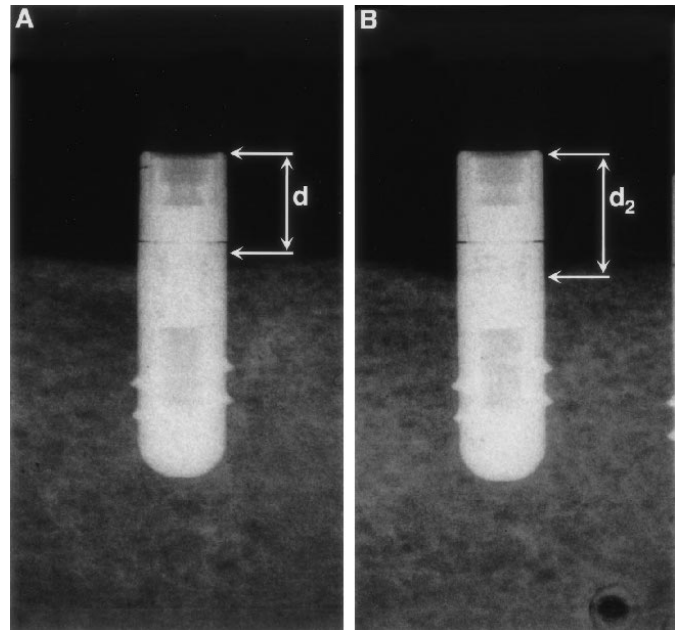


Figure 2.

Standardized periapical radiographs similar to Figure 1 of a non-welded implant.

determined by calculating the microgap (MG) to fBIC by subtracting 3.5 mm.

One implant (B) was excluded because of questionable values at 2 months due to a vertical radiolucent defect affecting 50% of the mesial aspect of the implant surface. At all other time points (baseline and 1 and 3 months) the crestal bone levels around this implant were similar to other type B implants. The reason for this unusual value at 2 months could have been due to a radiolucent artifact or an acute infection which recovered a month later. Two other implants (B and C) were not assessed as the implants most likely had been subjected to early occlusal forces resulting in the microgap becoming submerged at the 1-month radiographic evaluation. Two additional implants (A and F) were excluded due to incomplete mesial and distal data.

The results of the mixed-model ANOVA indicated that the main effect of welded/non-welded was significant ($P < 0.001$), while the main effects of microgap size ($P > 0.70$) and follow-up month ($P > 0.70$) as well as all interactions ($P > 0.35$) were non-significant, indicating that differences in crestal bone loss were observed only for welded versus non-welded regardless of microgap size or follow-up month (Tables 1A and 1B). These results indicate that the combination of the welded implant types (A-C) had a significantly lower mean crestal bone loss as compared to the combination of the non-welded implant types (D-F). Specifically, this result was true for 1 ($P < 0.04$) and 2 months ($P < 0.02$), but not 3 months ($P > 0.70$) (Table 1B). The

Table IA.

Distance Between the Top of the Implant (TOP) to the First Bone Implant Contact (fBIC) for Welded (A, B, C) and Non-Welded (D, E, F) Implants

Implant	N Implants	Baseline		1 Month		2 Months		3 Months	
		Mean ± SD	SD	Mean ± SD	SD	Mean ± SD	SD	Mean ± SD	SD
Welded	26	3.73	0.66	4.59	0.34	4.58	0.51	4.61	0.53
Non-welded	29	3.90	0.55	5.01	0.46	5.04	0.50	4.87	0.48

Table IB.

Crestal Bone Loss for Welded (A, B, C) and Non-Welded (D, E, F) Implants

Implant	N Implants	1 Month		2 Months		3 Months	
		Mean ± SD	SD	Mean ± SD	SD	Mean ± SD	SD
Welded	26	0.86	0.52	0.85	0.57	0.88	0.58
Non-welded	29	1.11	0.58	1.14	0.71	0.97	0.58
		<i>P</i> <0.04*		<i>P</i> <0.02*		<i>P</i> >0.70	

**P* value was significant if *P* <0.05.

mean crestal bone loss was consistently higher for non-welded implant types compared to welded implant types at each follow-up month, but the mean crestal bone loss for non-welded implant types decreased by more than 0.1 mm from 2 to 3 months, while the corresponding means for welded implant types were relatively unchanged.

DISCUSSION

The purpose of this radiographic study was 2-fold: first, to determine whether the size of the microgap of either 10 µm or 50 µm, or 100 µm between the implant-abutment interface influences the amount and rate of crestal bone resorption and second, whether welded versus a non-welded implant-abutment connection with monthly loosening and tightening of the abutment may also contribute to bone resorption. At baseline, each implant microgap was placed at the same level of 1 mm above the alveolar crestal bone.

All implants developed crestal bone loss at 1 month compared with baseline levels. However, during the early healing period, the non-welded group (D, E, and F) showed significantly greater crestal bone loss from baseline to 1 month (*P* <0.04) and 2 months (*P* <0.02) compared with the welded group (A, B, and C). No significant differences were observed between these 2 groups at 3 months. Thus, 2-piece implants (non-welded) showed significantly greater crestal bone loss compared with 1-piece implants (welded) after 1 and

2 months suggesting that the stability of the implant/abutment interface may play a key role in preventing early crestal bone resorption.

The bone loss that was evident after 1 month in the 1-piece welded group (A, B, and C) generally remained stable up to the 3-month evaluation. In contrast, the 2-piece, non-welded group, while losing significantly more bone loss at 1 and 2 months compared to the welded group, tended to regain bone height around the implants during the 2- to 3-month period. Hence, by 3 months no differences were observed between the 2 groups. It would appear that the welded group establishes its bone remodeling equilibrium at an earlier stage compared with the non-welded group suggesting that the stability of the connection is critical during the early healing period. This is not surprising as other studies report that significant crestal bone loss occurs following abutment loosening and tightening.^{4,7,8} Radiographic results evaluating 1- and 2-piece implants using a submerged and non-submerged approach in a canine model also showed no differences in the peri-implant bone levels at the end of the healing period.¹⁸ In that study, the microgap of approximately 10 µm on the 2-piece implant was placed 0.5 mm coronal to the crestal bone (and was 0.5 mm coronal to the rough/smooth border). A temporal pattern of bone loss varied with submerged and non-submerged implants with the greatest amount of bone loss occurring in the 2-piece submerged implant immediately after implant placement while at 3 months no differences were observed between implant types.

In the present study, greater cortical bone loss followed the surgical placement of the implants in the non-welded group and this difference was detectable radiographically. Subsequently, remodeling in the non-welded group appeared to have obliterated any statistical differences between the groups. However, the histological findings¹² showed that crestal bone loss was greater in the non-welded group compared with the welded group indicating that significant differences in crestal bone levels remained between the 2 groups at 3 months. The radiographic findings would suggest that crestal bone loss in the welded group was substantial during early healing, but that this bone loss was not different from the non-welded group at 3

months. Therefore, the limitation in the sensitivity of the radiographic technique may have precluded the detection of differences between the implants at 3 months. The radiographic technique used in this study was the same as used in previous studies.^{4,17} Recently, it has been shown that standardized periapical radiography using customized fixation stents and rigid connection to the long cone device compared favorably with histological data evaluating mesial and distal sites using the same dog model as in the present study to within a range of 0.2 mm.¹⁷ However, others report that periapical radiography may underestimate the results of histometric analyses.¹⁹ Recently, it has been shown that linear radiographic measurements underestimated bone fill by approximately 1 mm in furcation defects compared with re-entry data.²⁰

In the present study, it is conceivable that differences between radiological and histological measurements occurred as a result of the superimposition of the buccal and lingual walls upon the radiograph. This will occur despite operator and radiographic standardization. The amount of bone loss will alter the density and thus may increase the likelihood of error in interpreting the actual crestal bone loss to the fBIC. It is interesting to note that the earlier study using the same radiographic techniques reported by Hermann et al. utilized not only linear measurements of standardized periapical radiographs but also evaluated bone density changes using computer assisted densitometric image analysis.⁴ This may have highlighted more accurately the true bone loss minimizing the error from the superimposition of buccal and lingual bone walls and thus matched the histological findings more accurately.^{4,7} Cortical mineralization occurring in the superimposed buccal and lingual plates is likely to further obscure the actual osseous changes around the implants.

An objective of this study was to determine whether the size of the microgap of either 10 μm , 50 μm , or 100 μm between the implant-abutment interface influences the amount and rate of crestal bone resorption. The present study shows that the size of the microgap at the abutment/implant interface had no significant effect upon crestal bone loss as the implants with the 10 μm , 50 μm , and 100 μm microgap (corresponding to implants A and D, B and E, and C and F, respectively) showed similar crestal bone levels. The amount of bone loss observed in this study was consistent with an earlier study⁴ where one type of implant (E) was similar to that observed here. This confirmation and consistency lends further support that the interface plays a critical role in the crestal bone level around implants. The observations regarding the influence of the microgap and an interface between the abutment and the implant highlight the need for human clinical studies to confirm these findings and determine their clinical relevance not only in relationship to hard and

soft tissue relationships, but also to the inflammatory status.¹⁸

In conclusion, this study supports previous histological results that 2-piece implants with a microgap located 1 mm above the alveolar crest have a significant effect in promoting crestal bone resorption. However, this study further points out that the actual size of the microgap (10 μm , 50 μm , or 100 μm) between the implant-abutment interface did not significantly affect the observed crestal bone resorption. This suggests that manufacturing small interfaces between components will have limited effect on the biological consequences that result from the presence of the interface. Additionally, the data suggest that the stability of the interface may have a significant influence on the early biological events occurring adjacent to the interface but not at later times. Thus, the 2-piece implants (non-welded) showed significantly greater crestal bone loss compared to 1-piece implants (welded) after 1 month suggesting that the stability of the implant/abutment interface may have an important role to play in determining early crestal bone levels. The most important finding in this study implies that implant configurations incorporating an interface will be associated with biological changes regardless of interface size.

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REFERENCES

1. Albrektsson T, Zarb G, Worthington P, Eriksson AR. The long-term efficacy of currently used dental implants: A review and proposed criteria of success. *Int J Oral Maxillofac Implants* 1986;1:11-25.
2. Schroeder A, van der Zypen E, Stich H, Sutter F. The reactions of bone, connective tissue, and epithelium to endosteal implants with titanium-sprayed surfaces. *J Maxillofac Surg* 1981;9:15-25.
3. 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.
4. Hermann JS, Cochran DL, Nummikoski PV, Buser D. Crestal bone changes around titanium implants. A radiographic evaluation of unloaded nonsubmerged and submerged implants in the canine mandible. *J Periodontol* 1997;68:1117-1130.
5. Rams TE, Roberts TW, Tatum H Jr, Keyes PH. The subgingival microbial flora associated with human dental implants. *J Prosthet Dent* 1984;51:529-534.
6. Mombelli A, van Oosten MA, Schürch E Jr., Lang NP. The microbiota associated with successful or failing osseointegrated titanium implants. *Oral Microbiol Immunol* 1987;2:145-151.
7. Hermann JS, Buser D, Schenk RK, Cochran DL. Crestal

- bone changes around titanium implants. A histometric evaluation of unloaded non-submerged and submerged implants in the canine mandible. *J Periodontol* 2000;71:1412-1424.
8. Abrahamsson I, Berglundh T, Lindhe J. The mucosal barrier following abutment dis/reconnection. An experimental study in dogs. *J Clin Periodontol* 1997;24:568-572.
 9. Bidez MW, Misch CE. Issues in bone mechanics related to oral implants. *Implant Dent* 1992;1:289-294.
 10. Adell R, Lekholm U, Rockler B, Brånemark P-I. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. *Int J Oral Surg* 1981;10:387-416.
 11. Gómez-Róman G. Influence of flap design on peri-implant interproximal crestal bone loss around single-tooth implants. *Int J Oral Maxillofac Implants* 2001;16:61-67.
 12. Hermann JS, Schoolfield JD, Buser D, Schenk RK, Cochran DL. Influence of the size of the microgap on crestal bone changes around titanium implants: A histometric evaluation of unloaded non-submerged implants in the canine mandible. *J Periodontol* 2001;72:1372-1383.
 13. Rangert B, Jemt T, Jorneus L. Forces and moments on Brånemark implants. *Int J Oral Maxillofac Implants* 1989;4:241-247.
 14. Hermann JS, Buser D, Schenk RK, Schoolfield JD, Cochran DL. Biologic width around one- and two-piece titanium implants. A histometric evaluation of unloaded nonsubmerged and submerged implants in the canine mandible. *Clin Oral Implants Res* 2001;12:559-571.
 15. 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.
 16. Tsai N, McManus LM, Oates TW, Hermann JS, Cochran DL. An evaluation of inflammation associated with the implant/abutment interface. *J Dent Res* 2000;79(Spec. Issue):168(Abstr. 197).
 17. Hermann JS, Schoolfield JD, Nummikoski PV, Buser D, Schenk RK, Cochran DL. Crestal bone changes around titanium implants: A methodologic study comparing linear radiographic with histometric measurements. *Int J Oral Maxillofac Implants* 2001;16:475-485.
 18. Fiorellini JP, Buser D, Paquette DW, Williams RC, Haghghi D, Weber HP. A radiographic evaluation of bone healing around submerged and non-submerged dental implants in beagle dogs. *J Periodontol* 1999;70:248-254.
 19. Gotfredsen K, Rostrup E, Hjørting-Hansen E, Stoltze K, Budtz-Jørgensen E. Histological and histomorphometrical evaluation of tissue reactions adjacent to endosteal implants in monkeys. *Clin Oral Implants Res* 1991;2:30-37.
 20. Toback GA, Brunsvold MA, Nummikoski PV, Masters LB, Mellonig JT, Cochran DL. The accuracy of radiographic methods in assessing the outcome of periodontal regenerative therapy. *J Periodontol* 1999;70:1479-1489.

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