

Spontaneous Early Exposure of Submerged Endosseous Implants Resulting in Crestal Bone Loss: A Clinical Evaluation Between Stage I and Stage II Surgery

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Spontaneous early exposure of submerged implants during the osseointegration healing phase may be a harmful factor that results in early crestal bone loss around the implants. The objective of this study was to assess the effect of spontaneous early exposure on crestal bone loss around submerged implants, with special attention given to the relationship between the degree of exposure and the amount of peri-implant bone loss. Crestal bone level relative to the shoulder of the implant was measured at the time of placement and at the time of exposure 4 to 5 months later. During the period between stage I and stage II surgery, implant sites were observed, and each implant site in which spontaneous early exposure was detected was recorded. Perforations were classified according to the degree of implant exposure from Class 0 (no perforation) to Class IV (complete exposure). Measurements from 206 implants in 64 patients produced 85 groups valid for statistical comparison; each of these contained at least 2 lesions of different types. There was a statistically significant difference between bone loss associated with intact mucosa (Class 0) and Class I, Class II, and Class III lesions, and between Class I and II lesions. There were no significant differences between Class I and III and between Class II and III. In Class II and III lesions, there was more bone loss associated with the buccal aspect of the implants. Of the 115 perforated sites, 10 were associated with bone loss exceeding 2 mm, 2 presented 3 to 4 mm bone loss, 1 showed more than 4 mm, and 1 displayed more than 5 mm. In view of the clinical implications that spontaneous early exposure may have on the success of osseointegration, prematurely partially exposed implants should be exposed as soon as possible after the perforation is observed. (INT J ORAL MAXILLOFAC IMPLANTS 2001;16:514-521)

Key words: bone loss, dental implants, early implant exposure, gingival perforation

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At the implant-bone interface after successful placement of endosseous dental implants. This biodynamic process, referred to as *osseointegration*, provides the final anchorage of the implant in the bone.¹ During the osseointegration healing phase of submerged implants, complete mucosal coverage and isolation of the implant from the oral cavity avoids trauma and infection and establishes favorable conditions for osseointegration.^{1,2}

Although there is general agreement of high rates of successful implant integration to bone, crestal bone loss during the submerged stage may occur that is clinically evident at the time of implant exposure. Early bone loss may occur because of surgical complications, improper fit, micromovements

of the implant, reduced bone quality, and harmful habits of the patients, especially smoking.^{3,4}

Spontaneous early exposure may be an additional harmful factor resulting in early crestal bone loss around submerged implants. Adell and associates⁵ suggested that any communication with the oral cavity seen during the first 6 weeks postoperatively should be treated by excision of the perforated site, flap mobilization, re-suturing, and appropriate adjustment of the denture. Block and Kent,⁶ who investigated factors that compromise endosseous implant healing, found that spontaneous early exposure appeared to be associated with an increased incidence of crestal bone loss. In a recent biometric study of 275 implants, patients with 1 or more exposed sites demonstrated a probability of bone loss that was 3.9 times greater than in patients with nonexposed sites.⁷

Spontaneous early perforations have been classified according to the degree of implant exposure, from Class 0 (no perforation) to Class IV (complete exposure).⁸ Intact and perforated oral mucosa covering dental implants has also been studied histologically.^{9,10} It is logical to suggest that in Class 0 and I, in which there is no direct communication between the implant and the oral environment, less peri-implant bone loss will occur between stage I and stage II surgery, compared with implants presenting Class II, III, or IV exposures. Additionally, Class IV lesions, in which the implant is completely exposed, will result in less peri-implant bone loss compared to Class II and III lesions.

The purpose of this study was to assess the effect of spontaneous early exposure on early crestal bone loss around submerged endosseous implants, focusing on the relationship between the degree of exposure and the amount of peri-implant bone loss.

MATERIALS AND METHODS

Patients who participated in this study were treated in the Department of Periodontology, Tel Aviv University School of Dental Medicine, as well as in 5 different clinics, which adopted the following protocol.

Presurgical Inclusion/Exclusion Criteria

1. Patients were generally healthy, not on regular medication, and did not take any medication 2 months prior to implant placement.
2. Patients were nonsmokers.
3. Extractions were not performed at the implant sites at least 1 year prior to implant placement.
4. Patients wearing conventional removable dentures presented no signs of trauma to the mucosa immediately prior to participation in the study.

Surgical Procedures

A conservative 2-stage surgical procedure was adopted.⁸ Threaded implants, 3.8 mm in diameter (Steri-Oss, Nobel Biocare, Yorba Linda, CA), were placed with the implant shoulder level with the surrounding bony crest. When there were crestal irregularities, bone was leveled to the implant shoulder using hand chisels.

After implant placement, the crestal bone level relative to the shoulder of each implant was measured to the nearest 0.5 mm using a hand periodontal probe marked from 1 to 10 mm. Measurements were taken at the midbuccal (B), midlingual/palatal (L), midmesial (M), and middistal (D) aspects of each implant and recorded. Implants were sealed with appropriate flat cover screws.

Flap incisions were closed in an attempt to achieve complete closure, applying simple interrupted and/or simple mattress lock sutures, using 3-0 silk suture material. Flaps were released before wound closure to avoid tension where necessary.

Postoperative Care

Temporary fixed and removable restorations, as well as a 2-week special soft diet, were designed to eliminate or reduce trauma to the surgical sites. Patients were instructed to rinse their mouth with 0.2% chlorhexidine solution 3 times daily for 1 minute during the first 2 weeks after surgery. Sutures were removed 10 to 14 days after implant placement.

Follow-up Procedures

Patients were followed weekly for the first 2 months postsurgery. After 3 weeks, the implant sites were gently cleaned and examined by air steam and gentle probing with a thick, blunt periodontal probe. Each implant site at which exposure was observed was recorded. When exposure was detected or observed, the patient was instructed to clean the exposed site by gently rubbing the mucosa with gauze soaked in 0.2% chlorhexidine solution twice daily. At 6 to 8 weeks postsurgery, the mucosa covering implants was clinically classified according to Tal⁸:

- Class 0: Mucosal covering over the implant is intact.
- Class I: A breach in the mucosa covering the implant is observed. Oral implant communication can be detected with a periodontal probe, but the implant surface cannot be seen (Figs 1a and 1b).
- Class II: Mucosa above the cover screw is fenestrated, and the cover screw is visible. The borders of the perforation aperture do not reach or overlap the borders of the cover screw at any point (Fig 2).



Fig 1a Class I perforations 3 weeks postsurgery.



Fig 1b Oral cavity-implant communication was detected by probing, but the implant surface is not visible (original magnification $\times 2$).



Fig 2 Class II perforation that was initially misidentified as Class I; however, the implant surface was visible during air blowing (original magnification $\times 2$).



Fig 3 Three implants showing different conditions: *Class 0 (intact mucosa); **Class III exposure: the borders of the perforation aperture partly overlap the buccal aspect of the cover screw border; ***Class IV exposure: the cover screw is completely exposed.

Class III: Cover screw is visible. In some parts, the borders of the perforation aperture overlap the borders of the cover screw (Fig 3).
Class IV: Cover screw is completely exposed (Fig 3).

Before stage II surgery, 4 to 5 months after implant placement, the classifications were reconfirmed. At this stage, only patients who had 2 or more implants with different classifications (Class 0 to IV) were approved for inclusion in the study. Of

the 462 patients, 67 met this criterion. Of these, 3 patients in whom lesions had changed and thus scored a different classification were omitted from the study. In the remaining 64 patients, all implants were exposed. For the purpose of implant exposure, the mucosa was incised and reflected as previously described.^{9,10} Remnants of soft tissue were removed from the bony crest surrounding the implants, and measurements of the crestal bone level relative to the implant shoulders were repeated in a manner

Table 1 Mean Bone Loss (in mm) Around Implants According to Degree of Perforation

	Type of lesion					Total (n = 206)
	0 (n = 91)	I (n = 35)	II (n = 52)	III (n = 23)	IV (n = 5)	
Mean I	0.062	0.032	0.059	-0.043	0.00	-0.02
Mean II	-0.061	-0.371	-0.797	-0.819	-0.375	-0.48
Amount of change	0.123	0.403	0.856	0.776	0.375	0.46

Mean I = Mean bone level at Stage I surgery; Mean II = mean bone level at Stage II surgery.

Table 2 Statistical Comparison of Mean Bone Loss (in mm) Between Different Lesions

	0 vs. I (n = 19)	0 vs. II (n = 28)	0 vs. III (n = 9)	I vs. II (n = 18)	I vs. III (n = 4)	II vs. III (n = 7)
Bone loss 1	0.0691	0.1004	0.1065	0.4097	1.1250	0.7433
Bone loss 2	0.3092	0.8763	1.1250	1.0278	1.3438	0.2864
Difference	-0.2401	-0.7759	-1.0185	-1.6181	-0.2185	0.4569
SD	0.484	0.377	0.003	0.621	0.780	0.548
P	.044	.000	.002	.001	.614	.069

Bone loss 1 = bone loss associated with the first lesion; Bone loss 2 = bone loss associated with the second lesion; Difference = difference in bone loss between the first and second lesion.

similar to that performed at the time of implant placement. Data were collected for all implants and tabulated on a patient-by-patient basis.

Since the sampling unit was the patient, measurements associated with all implants involved with the same type of lesion (Class 0, I, II, III, IV) were averaged in each patient. Also, measurements were grouped in each lesion according to surfaces (B, L, M, D).

Because of the selection criteria, statistical analysis was carried out between different lesions in patients who presented at least 2 different lesions. Although outcome data were available for 206 implants, demographic data were summarized for 64 patients. The difference in mean bone loss according to lesions was examined using the paired *t* test. For each lesion type, analysis of variance with repeated measures was used to test the difference of mean bone loss according to surfaces (B, L, M, D). To further assess the clinical implications of bone loss associated with individual implants, the frequencies of the most severe bone loss measured on individual implants were calculated.

RESULTS

Data were available from 64 patients in whom 206 implants were placed. The mucosa covering 91 implants remained intact (Class 0), 35 sites were classified as Class I, 52 as Class II, 23 as Class III, and only 5 as Class IV. Table 1 presents the mean bone loss around implants according to the degree of perforation. When all implants were combined, mean bone loss was less than 0.5 mm. Bone loss under intact mucosa averaged 0.12 mm; it increased to 0.40 mm, 0.86 mm, 0.78 mm, and 0.38 mm under Class I, II, III, and IV perforations, respectively.

Only 85 groups were valid for statistical comparison of bone loss between lesions. Each of these contained 2 lesions of different types (Table 2). There was a statistically significant difference between bone loss associated with intact mucosa and Class I ($P < .05$), Class II ($P < .001$), and Class III lesions ($P < .001$), and also between Class I and Class II lesions ($P < .01$). There was no statistically significant difference between Class I and Class III lesions and between Class II and Class III lesions.

Table 3 Mean Bone Levels (in mm) at Stage I and Stage II Surgery According to Different Surfaces

Site	Type of lesion and stage of surgery									
	Class 0 (n = 91)		Class I (n = 35)		Class II (n = 52)		Class III (n = 23)		Class IV (n = 5)	
	I	II	I	II	I	II	I	II	I	II
Buccal	-0.023	-0.135	-0.024	-0.364	0.005	-1.110	-0.348	-1.446	0.000	-0.500
Mesial	0.203	0.061	0.164	-0.278	0.143	-0.641	0.337	-0.681	0.000	-0.400
Lingual	0.040	-0.100	-0.0429	-0.386	0.095	-0.665	-0.163	-0.909	0.000	-0.300
Distal	0.050	-0.08	0.079	-0.436	0.321	-0.867	0.098	-0.583	0.200	-0.200
<i>P</i> (I vs II)	.003	.0002	.0001	.0001	—*					
<i>P</i> (BMLD)	.795	.233	.003	.0009	—*					

*No statistical analysis performed.

I = Mean bone level at Stage I surgery; II = mean bone level at Stage II surgery.

Too few Class IV lesions were available for statistical analysis.

Comparison of bone loss according to the different surfaces (B, L, M, D) using analysis of variance with repeated measures (Table 3) has shown that in Class 0 sites and Class I lesions, there was no statistically significant difference between bone loss associated with different surfaces. In Class II and Class III lesions, there was more mean bone loss associated with the buccal aspects compared with other surfaces, and the difference was statistically significant ($P < .01$). There were insufficient lesions in the Class IV group for statistical analysis.

Since averaging the measurements of all implants under each classification in each patient may mask the clinical impact of bone loss associated with individual implants on the success/failure of osseointegration (Figs 4a and 4b), bone loss associated with individual implants and with individual surfaces was also examined and analyzed. Table 4 presents the frequencies of mean bone loss according to lesion types in all 206 implants. The frequency of the more severe average bone loss increased from Class 0 to Class I and from Class I to Class II and III. However, only a few implants were affected by an average bone loss greater than 2 mm (1 in Class II and 3 in Class III lesions).

Further investigation of individual surfaces and recording only the surface associated with the most severe bone loss in each implant showed that 10 of 206 implants (4.8%) were associated with bone loss of 2 mm or more. Since all belonged to Class I, II, and III groups, 10 of 115 sites (8.7%) with perfo-

rated mucosa were associated with bone loss of 2 mm or more, 2 implants presented with 3 to 4 mm of bone loss, 1 implant showed more than 4 mm of loss, and 1 displayed more than 5 mm of bone loss. Bone loss of more than 4 mm was from the Class II group.

DISCUSSION

This study demonstrates a statistically significant difference between bone loss associated with implants covered by intact mucosa and that associated with implants that presented spontaneous early exposures between the time of implant placement and the time of implant exposure 4 to 5 months later. To understand the clinical significance of this premature bone loss, it is important to compare it with the final bone level that is established around functionally loaded implants following exposure.

Submerged dental implants integrate with the surrounding bone during the time between placement (stage I surgery) and the time of intentional exposure (stage II surgery). During this period, the level of crestal bone associated with the implant is expected to remain unchanged.¹¹ It has been reported that following exposure and abutment connection, healing results in bone resorption and soft tissue recession. The dimensions of the mucosal/implant attachment studied in the dog comprised junctional epithelium (approximately 2 mm high) and a connective tissue zone (measuring approximately 1 mm or more).¹¹ This attachment

Fig 4a (Left) Favorable peri-implant bone loss. A mean bone loss of 1.75 mm is composed of 1, 2, 2, and 2 mm bone loss at the different aspects of the implant (buccal, lingual, mesial, distal).

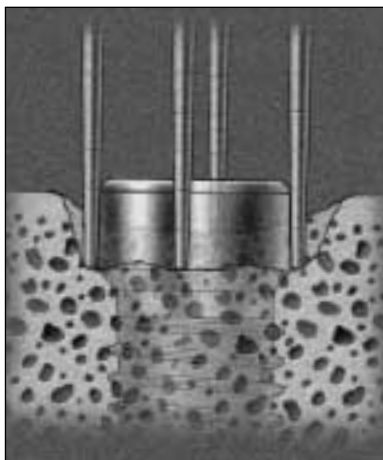


Fig 4b (Right) Unfavorable peri-implant bone loss. A mean bone loss of 1.75 mm is composed of 0, 0, 5, and 2 mm bone loss relating to the different aspects of the implant (buccal, lingual, mesial, distal).

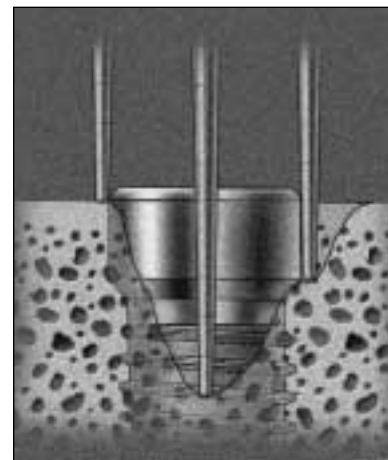


Table 4 Distribution of Frequencies of Mean Bone Loss Measurements According to Types of Lesions

Bone loss (mm)	Class 0 (n = 91) (n and %)	Class I (n = 35) (n and %)	Class II (n = 52) (n and %)	Class III (n = 23) (n and %)	Class IV (n = 5) (n and %)
≥ 0	62 (68.1%)	19 (54.3%)	7 (13.5%)	6 (25.0%)	3 (60.0%)
0–0.99	27 (29.7%)	7 (20.0%)	16 (30.8%)	7 (30.0%)	1 (20.0%)
1–1.99	2 (2.2%)	9 (25.7%)	28 (53.8%)	7 (30.0%)	1 (20.0%)
2–2.99	0 (0%)	0 (0%)	1 (1.9%)	2 (10.0%)	0 (0%)
3–3.99	0 (0%)	0 (0%)	0 (0%)	1 (5.0%)	0 (0%)
≥ 4	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)

(%) = Percentage of implants associated with maximal bone loss measurement.

may protect the zone of osseointegration from factors released from plaque and from the oral cavity, similar to the dentogingival junction, referred to as the “biologic width” associated with healthy natural teeth.¹² Depending on the thickness of mucosa, bone loss, which is associated with establishment of the peri-implant mucosa following exposure, reaches an average of 1.3 mm.¹³

The development of a peri-implant soft tissue zone, analogous to the biologic width associated with natural teeth, has been further investigated and verified.^{14–18} According to these studies, it develops around implants of all shapes following exposure (stage II) surgery, as well as around nonsubmerged, 1-stage implants. This phenomenon is unrelated to function and occurs whether the implant is loaded or not.

When all 91 Class 0 implants were combined, the mean change in bone level was 0.12 mm; 62 (68.1%) presented no change or slight bone growth, 27 displayed (29.7%) less than 1 mm of bone loss, and only 2 (2.2%) showed 1 to 1.99 mm of bone

loss (Table 4). This observation was further confirmed by examining bone loss associated with individual surfaces. Only 6 (6.6%) surfaces associated with implants covered by intact mucosa showed 1 to 1.99 mm of loss, and none exceeded 2 mm. Therefore, it appears that submerged implants covered by intact mucosa do not show bone loss exceeding the final dimensions of the expected soft tissue biologic width.

More crestal bone loss was associated with Class I lesions. The average bone loss around 35 Class I implants was 0.4 mm; the difference between Class 0 and Class I implants in 19 patients with both types of sites was statistically significant. Although mean bone loss increased in Class II and Class III lesions (0.86 mm and 0.78 mm, respectively), the difference was statistically significant between Class 0 and Class I, II, and III, and also between Class I and Class II, but not between Class I and Class III or between Class II and Class III. These observations may be explained by the histologic and morphologic differences between the different mucosal

lesions. Recently it has been shown that Class I lesions, ie, those with minimal mucosal perforations but no visible communication to the implant surface, present chronic inflammatory infiltrate surrounding a cyst-like structure, with a thin layer of connective tissue forming the "cystic" wall proximal to the implant cover screw.¹⁰ In the present study, most Class I specimens showed the epithelial invagination and inflammatory infiltrate to be limited to the area above the implant cover screw. In contrast, in Class II and Class III specimens, in which there was direct contact between the implant and the oral cavity, the epithelial invagination and inflammatory infiltrate spread beyond the borders of the implant shoulder. Therefore, an adverse effect of the lesion on the surrounding crestal bone could be expected. The pathologic structure of the perforated lesions could also act as a plaque-retentive site, thereby increasing bone loss. Plaque accumulation around implants generally results in peri-implant mucositis. It has been shown that de novo plaque formation results in the establishment of inflammatory cell infiltrates in dogs^{19,20} as well as in human volunteers.²¹ In the dog model after 3 months, experimentally induced inflammation expanded and progressed apically.²⁰

The observation that more bone loss was associated with the buccal aspects compared to the other surfaces ($P < .01$; Table 3) is noteworthy. While perforations (Class II and Class III) contributed to early bone loss in all aspects, there were also additional factors. A major factor contributing to early bone loss or gain is facial bone thickness. A significantly greater amount of facial bone loss has been measured between stage I and II surgery when the facial bone thickness was less than 1.8 mm.²² Therefore, bone volume and implant position are significant contributing factors to bone loss during the early stage of healing after implant placement. Interimplant distance also influences the height of interimplant bone crest. Increased crestal bone loss has been associated with less than 3 mm distance between implants once the implant is exposed.²³ Bone quality, an additional factor that was not examined in the present study, could also contribute to early bone loss.

In view of the clinical implications that spontaneous premature fenestrations may have on the success of osseointegration, and based on available data that crestal bone resorption up to 2 mm is expected and considered "normal" following abutment connection, it was decided to focus on the frequency of implants presenting with bone loss of 2 mm or more. Only 4 implants (1 from a Class II site and 3 from Class III sites) showed mean bone loss exceed-

ing 2 mm. However, when only the most severe measurement related to each implant was considered, 10 of 111 implants (8.7%) presented bone loss of 2 mm or more on at least one of their surfaces; 2 of these showed 3 to 3.99 mm of bone loss, 1 showed 4 to 4.99 mm of loss, and 1 showed more than 5 mm of loss. The more severe cases were in the Class II group of lesions, followed by Class I and Class III. Implants that were covered by intact mucosa and implants that were completely exposed (Class IV) did not present bone loss exceeding 1.99 mm on any of their surfaces. This last observation is in agreement with recent reports suggesting that unloaded nonsubmerged implants present no significant difference from submerged implants with respect to osseointegration.^{17,18}

CONCLUSION

Within the limitations of this study, it is suggested that to avoid peri-implant bone loss associated with 2-stage surgical procedures, premature partially exposed implants should be completely exposed as soon as possible after the perforation is observed.

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REFERENCES

1. Albrektsson T. Bone tissue response. In: Brånemark P-I, Zarb GA, Albrektsson T (eds). *Tissue-Integrated Prostheses: Osseointegration in Clinical Dentistry*. Chicago: Quintessence, 1985:129-143.
2. Adell R, Lekholm U, Brånemark P-I. Surgical procedures. In: Brånemark P-I, Zarb GA, Albrektsson T (eds). *Tissue-Integrated Prosthesis: Surgical Procedures*. Chicago: Quintessence, 1985:223-225.
3. Zoldos J, Kent JN. Healing of endosseous implants. In: Block MS, Kent JN (eds). *Endosseous Implants for Maxillofacial Reconstruction*. Philadelphia: Saunders, 1995:40-69.
4. Smith RA. Long-term complications of osseointegrated implants. In: Kaplan LB, Pogrel MA, Perrott DH (eds). *Complications in Oral and Maxillofacial Surgery*. Philadelphia: Saunders, 1997:319-358.
5. Adell R, Lekholm U, Rockler B, Brånemark P-I. A 15-year study of osseointegrated implants in the treatment of edentulous jaws. *J Oral Surg* 1981;10:387-415.
6. Block MS, Kent JN. Factors associated with soft- and hard-tissue compromise of endosseous implants. *J Oral Maxillofac Surg* 1990;48:1153-1160.

7. Toljanic JA, Banakis ML, Willes LAK, Graham L. Soft tissue exposure of endosseous implants between stage I and stage II surgery as a potential indicator of early crestal bone loss. *Int J Oral Maxillofac Implants* 1999;14:436-441.
8. Tal H. Spontaneous early exposure of submerged implants: I. Classification and clinical observations. *J Periodontol* 1999;70:213-219.
9. Tal H, Dayan D. Spontaneous early exposure of submerged implants: II. Histopathology and histomorphometry of non-perforated mucosa covering submerged healed implants. *J Periodontol* 2000;71:1224-1230.
10. Tal H, Dayan D. Spontaneous early exposure of submerged implants: III. Histopathology of perforated mucosa covering submerged implants. *J Periodontol* 2000;71:1231-1235.
11. Berglundh T, Lindhe J. Dimension of the peri-implant mucosa. Biological width revisited. *J Clin Periodontol* 1996; 23:971-973.
12. Gargiulo AN, Wentz FM, Orban B. Dimension and relations of the dentogingival junction in humans. *J Periodontol* 1961;32:261-267.
13. Berglundh T, Lindhe J, Ericsson I, Marinella CP, Liljenberg B, Thomsen P. The soft tissue barrier at implants and teeth. *Clin Oral Implants Res* 1991;2:81-90.
14. Buser D, Weber HP, Donath K, Fiorellini JP, Paquette DW, Williams RC. Soft tissue reactions to non-submerged unloaded titanium implants in beagle dogs. *J Periodontol* 1992;63:226-236.
15. Abrahamsson I, Berglundh T, Wennstrom J, Lindhe J. The peri-implant hard and soft tissues at different implant systems. *Clin Oral Implants Res* 1996;7:212-219.
16. Abrahamsson I, Berglundh T, Lindhe J. The mucosal barrier following abutment disconnection. An experimental study in dogs. *J Clin Periodontol* 1997;24:568-572.
17. 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 non-submerged implants in the canine mandible. *J Periodontol* 1997;68:186-198.
18. Hermann JS, Cochran DL, Nummikoski PV, Buser D. Crestal bone changes around titanium implants. A radiographic evaluation of unloaded non-submerged and submerged implants in the canine mandible. *J Periodontol* 1997;68:1117-1130.
19. Berglundh T, Lindhe J, Marinello C, Ericsson I, Liljenberg B. Soft tissue reaction to de novo plaque formation on implants and teeth. An experimental study in the dog. *Clin Oral Implants Res* 1992;3:1-8.
20. Ericsson I, Berglundh T, Marinello CP, Liljenberg B, Lindhe J. Long-standing plaque and gingivitis at implants and teeth in the dog. *Clin Oral Implants Res* 1992;3:99-103.
21. Pontoriero R, Tonelli MP, Cornevale G, Mobelli A, Nyman S, Lang NP. Experimentally induced peri-implant mucositis. A clinical study in humans. *Clin Oral Implants Res* 1994;5: 254-259.
22. 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;5:120-128.
23. Tarnow DP, Cho SC, Wallace SS. The effect of inter-implant distance on the height of inter-implant bone crest. *J Periodontol* 2000;71:546-549.