# The Influence of Soft Tissue Thickness on Crestal Bone Changes Around Implants: A 1-Year Prospective Controlled Clinical Trial

Tomas Linkevicius, DDS, Dip Pros, PhD¹/Peteris Apse, Prof, DDS, Dip Pros, MSc, Dr Habil Med²/ Simonas Grybauskas, DDS, MOS, MD, RCSEd, PhD³/Algirdas Puisys, DDS³

Purpose: The aim of this clinical trial was to evaluate the influence of gingival tissue thickness on crestal bone loss around dental implants after a 1-year follow-up. Materials and Methods: Forty-six implants (23 test and 23 control) were placed in 19 patients. The test implants were placed about 2 mm supracrestally, whereas the control implants were positioned at the bone level. Before implant placement, the tissue thickness at implant sites was measured with a periodontal probe. After healing, metal-ceramic cement-retained prostheses were constructed. According to tissue thickness, the test implants were divided into A (thin) and B (thick) groups. Intraoral radiographs were performed and crestal bone changes were measured at implant placement and after 1 year. Results: Mean bone loss around the test implants in group A (thin mucosa) was 1.61 ± 0.24 mm (SE; range, 0.9 to 3.3 mm) on the mesial and 1.28 ± 0.167 mm (range, 0.8 to 2.1 mm) on the distal. Mean bone loss in test group B (thick mucosa) implants was  $0.26 \pm 0.08$  mm (range, 0.2 to 0.9 mm) on the mesial aspect and  $0.09 \pm$ 0.05 mm (range, 0.2 to 0.6 mm) on the distal aspect. Mean bone loss around control implants was  $1.8 \pm$ 0.164 mm (range, 0.6 to 4.0 mm) and 1.87 ± 0.166 mm (range, 0.0 to 4.1 mm) on the mesial and distal aspects, respectively. Analysis of variance revealed a significant difference in terms of bone loss between test A (thin) and B (thick) groups on both the mesial and the distal. Conclusion: Initial gingival tissue thickness at the crest may be considered as a significant influence on marginal bone stability around implants. If the tissue thickness is 2.0 mm or less, crestal bone loss up to 1.45 mm may occur, despite a supracrestal position of the implant-abutment interface. Int J Oral Maxillofac Implants 2009;24:712-719

Key words: biologic width, crestal bone loss, dental implants, microgap, mucosal thickness

The concept of early crestal bone loss after prosthetic reconstruction of an implant was suggested by Albrektsson et al<sup>1</sup> more than two decades ago. Since then, many factors have been identified as possible reasons for this phenomenon. Overload,<sup>2</sup> the microgap at the implant-abutment interface,<sup>3</sup> a polished implant neck,<sup>4,5</sup> and others have been discussed extensively; however, the stability of the crestal bone remains controversial. Moreover, the influence of mucosal thickness and biologic width formation on crestal bone loss around implants has

been discussed only recently and has received little attention in comparison to other factors.<sup>6,7</sup>

It has been proposed that a minimum of 3 mm of peri-implant mucosa is required for a stable epithelial connective tissue attachment to form.<sup>8</sup> This soft tissue extension is usually referred to as the *biologic width* around implants, and it serves as a protective mechanism for the underlying bone.<sup>9</sup> Some have suggested that if a minimal dimension of gingival tissues is not available, bone loss may occur to ensure the proper development of biologic width.<sup>10</sup> These findings are consistent with prior tooth-related studies, which showed that the establishment of biologic width after tooth crown lengthening involved crestal bone loss.<sup>11</sup>

The transition of alveolar mucosa to peri-implant soft tissues after implant placement is a difficult and complex process. Berglundh et al<sup>12</sup> described the morphogenesis of the peri-implant mucosa and implied that the characteristics of gingival tissues may be important in this process. However, data regarding the relationship between mucosal thickness and marginal bone loss around implants are

**Correspondence to:** Dr Tomas Linkevicius, Institute of Odontology, Faculty of Medicine, Vilnius University, Zalgirio str. 115, LT-08217, Vilnius, Lithuania. Fax: +370-8-5-2728569. Email: linktomo@gmail.com

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<sup>&</sup>lt;sup>1</sup>Assistant Professor, Institute of Odontology, Faculty of Medicine, Vilnius University, Vilnius, Lithuania.

<sup>&</sup>lt;sup>2</sup>Professor, Department of Prosthodontics, Riga Stradina University, Riga, Latvia.

<sup>&</sup>lt;sup>3</sup>Private Practice, Vilnius Implantology Center, Vilnius, Lithuania.

sparse. In an animal experiment, Berglundh and Lindhe<sup>13</sup> reported that thin tissues can provoke crestal bone loss during formation of the peri-implant seal. Observations in another histologic study showed that implants surrounded by consistently thin mucosa had angular bone defects, while at implant sites with an even alveolar pattern, a wide mucosa biotype prevailed.<sup>14</sup> However, the evidence provided by well-designed animal studies is limited, which in turn reduces the generalizability of the aforementioned results to clinical practice.<sup>15</sup> In addition, clinical research regarding the effects of tissue thickness on bone stability around implants is lacking. Consequently, the question remains whether gingival tissue thickness plays a role in the etiology of early crestal bone loss.

The aim of this clinical trial was to test the influence of initial gingival tissue thickness on marginal bone loss around placed implants. A null hypothesis was formulated, with the authors anticipating no effect of gingival tissue thickness at the time of implant placement on crestal bone change around implants.

# **MATERIALS AND METHODS**

### **Patients**

Subjects were selected from partially edentulous patients who attended Vilnius Implantology Center (Vilnius, Lithuania) for implant treatment. Inclusion criteria were as follows: (1) presence of healed bone sites (at least 4 months after tooth extraction); (2) no bone augmentation procedures before or during implant placement; (3) edentulous gap large enough to accommodate at least two implants in any region of the mouth, with a minimum of 3 mm between implants; (4) no medical contraindications for implant surgery; (5) written informed consent provided for participation and permission to use the obtained data for research purposes.

A total of 22 patients participated in the study. Three subjects were excluded because the radiographic images of their implants were not sufficiently parallel to correctly calculate crestal bone changes. The final sample included 19 patients (11 men and 8 women) with an average age of 45.6 years (range, 23 to 71 years) at the beginning of the experiment.

# **Study Design**

A prospective controlled clinical trial was initiated. Two implants (test and control) were placed adjacent to each other in each patient. The test implant was placed 2 mm supracrestally, and the control implant was positioned at the crest (Fig 1). The patient's birth date was used to determine which implant would be

placed supracrestally. If a patient's birth year ended with an even number (eg, 1970), the first implant was designated as the test implant and positioned 2 mm above the bone crest. If the birth year was odd (eg, 1971), the first implant was placed at the crest and served as a control.

## **Implant Placement**

Placement of implants was planned after clinical and radiographic examination. Bone quantity was measured to ensure that an implant at least 9 mm in length could be placed without bone augmentation. Implants with an internal hex (Prodigy; BioHorizons) were placed in a single stage (nonsubmerged) by an experienced surgeon. All patients received a prophylactic dose of antibiotics (2 g amoxicillin, Ospamox; Biochemie) 1 hour prior to surgery. After the administration of 4% articaine solution (Ubistesin; 3M ESPE) for local anesthesia, a midcrestal incision on the center of the edentulous ridge was performed. The flap was raised in two stages:

- 1. A buccal flap was raised, and the mucosal thickness of the unseparated palatal-lingual flap was measured with a periodontal probe (Hu-Friedy) at the bone crest at the center of the future implant site (Fig 2).
- 2. A palatal-lingual flap was raised to expose the implant site.

The osteotomy site was measured to allow a minimum of 3 mm between the implants. Control implants were placed at the crest, and test implants were placed 2 mm above the bone level. Verification of the position of the implant was performed with a probe. After implant placement, healing abutments were connected, and 5/0 interrupted sutures (Polysorb; USS-DG) were placed to close the flaps. Immediately after suturing, radiographs were obtained using RVG Windows Trophy 5.0 (Trophy Radiologie) periapical films in high-resolution mode. Patients were instructed to rinse with 0.12% chlorhexidine digluconate (Fresenius Kabi Norge) solution twice a day for a week. For pain control, patients were prescribed 400 mg of ibuprofen (Ibumax; Vitabalans Oy) to be taken as needed. Patients were advised to minimize trauma at the site; no special diet was advocated. The sutures were removed 7 to 10 days after surgery. Patients were advised to clean healing abutments with an extra fine toothbrush.

## **Restorative Procedures**

Prosthetic procedures were initiated after 2 months of healing in the mandible and 4 months in the maxilla (Fig 3). Intraoral radiographs were obtained prior to



Fig 1 Positions of test (left) and control (right) implants.



Fig 2 Measurement of mucosal thickness at the time of implant placement with a 1-mm marked periodontal probe. The lingual flap has not yet been elevated to help ensure a more accurate measurement.



**Fig 3** Healed tissues around test (*left*) and control (*right*) implants. Note the differences in healing abutment emergence between the implants and the exposure of the polished implant neck around the test implant.

the first disconnection of healing abutments, at the time of framework fitting, and after prosthesis insertion. Impressions were made using an open-tray technique. If a fixed partial denture was constructed, impression transfers were splinted together with coldcured resin (Pattern Resin; GC). A polyvinylsiloxane putty (Flexitime; Heraeus Kulzer) and correction material were used for a one-step impression with the individual tray covered with adhesive. Porcelain-fusedto-metal fixed restorations were constructed and cemented with resin-modified glass-ionomer cement (Fuji Plus; GC) on modified standard abutments. The preparation line on the abutments was located no deeper than 0.5 mm below the mucosal margin. Prior to prosthesis cementation, the abutments were tightened to the implants using a torque wrench set to 30 N/cm<sup>2</sup>. Soft tissue probing was not performed to avoid disruption of soft tissues. All prosthetic treatment was performed by the same prosthodontist.

After cementation, radiographs were made to ensure abutment seating and check for residual cement. After prosthetic treatment, patients were instructed in cleaning implant-supported restorations.

# **Follow-up Examinations**

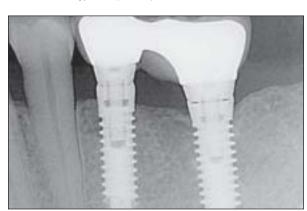
Patients were recalled 6 and 12 months after prosthetic treatment. At each visit, the restorations were evaluated for mobility, oral hygiene, peri-implant soft tissue conditions, and patient satisfaction. Intraoral radiographs were obtained to evaluate bone changes (Fig 4).

# **Radiographic Assessment and Measurements**

Intraoral radiographs were taken using a paralleling technique with a Rinn-type film holder in high-resolution mode. The standard setup for radiography was as follows: voltage, 70 kV; intensity of power, 4 mA; exposure time, standard program 7. A standardized setup was used for all radiographs and exposure time was specified manually depending on implant location, ranging from 0.110 to 0.189 seconds. This setup was used to ensure that the implant-abutment interface and the threads were clearly visible. Before measurement, the parallelism of all intraoral radiographs was evaluated. Therefore, radiographic images of three cases were excluded, as they were not considered sufficiently parallel for accurate calculation of bone



Fig 4 Radiographs of test implants. (Top left) Implants in thick tissue at baseline, (top right) implants in thick tissue at the 1-year follow-up, (bottom left) implants in thin tissue at baseline, and (bottom right) implants in thin tissue at the 1-year follow-up. Note the level of crestal bone (above) around the test implants in the thick tissue biotype and (bottom) around the thin tissues.







changes (Fig 5). All test implants (placed 2 mm supracrestally) were divided into two groups according to the thickness of the mucosa at the time of placement. Patients with gingival thickness of 2.0 mm or less were assigned to group A (thin mucosa; 9 patients), and patients whose gingival thickness was more than 2.5 mm were assigned to group B, which represented a thick mucosa (14 sites). The assignment to two groups was performed following the methodology of an animal experiment, which provided similar results. 12

Radiologic evaluation and measurements were performed by one of the examiners using the RVG Windows Trophy 5.0 software measurement program (Trophy Radiologie) with a magnification of  $6\times$ . Two images were selected to calculate the crestal bone changes: (1) after implant placement, and (2) 1 year

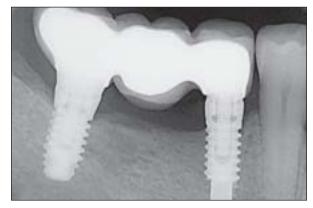


Fig 5 Excluded radiographic image. Note the lack of parallel visibility of implant threads.

after restoration. Before calculation of crestal bone changes, the RVG images were calibrated using the calibration program in the Trophy RVG software. The diameter of the implants was used for calibration as a reference point. The implant-abutment interface was chosen as a starting point for calculations because it was easily identified. The first measurement calculated the distance between the implant-abutment junction and crestal bone after implant placement on the distal and mesial aspects. The second measurement evaluated the same distance after 12 months of follow-up. The difference between these values showed the amount of bone loss. The measurements were repeated after 1 month.

# **Statistical Analysis**

Data were analyzed using SPSS version 15.0 for Windows (SPSS) statistical software. The single implant was treated as the statistical unit. Initially, each variable was assessed if parameters were normally distributed and parametric statistical tests could be applied. Because the variables appeared to be normally distributed, frequencies were calculated. The Pearson correlation coefficient was calculated to explore the direction and strength of the relationship between mesial and distal sites of the same implant. Next, a two-way analysis of variance (ANOVA) was conducted to assess mean differences within the groups. For comparison of continuous variables, means and standard errors of the means were calculated. Then, a paired t test analysis was conducted to assess mean differences between test and control groups. The mean differences were considered statistically significant at P = .05 with a confidence interval of 95%. To visualize the differences, 95% confidence intervals were plotted. Intraexaminer agreement was determined by a second measurement, which was performed after a 1-month interval. The mean difference between measurements was 0.1 ± 0.16 mm. All measurements were reproduced within a difference of  $\pm$  0.5 mm.

# **RESULTS**

A total of 46 implants (23 test and 23 control) were placed. Each pair of implants (test and control) was treated as a single case. The mandibular group consisted of 20 subjects (40 implants; 87% of the sample), and three patients received implants in the maxilla (6 implants; 13%). The implants were distributed in the different quadrants as follows: quadrant I, one patient (4.3%); quadrant II, two patients (8.7%); quadrant III, 11 patients (47.8%); and quadrant IV, nine patients (39.2%).

All 46 implants integrated successfully. Six single crowns (23.1%), 12 two-unit fixed partial dentures (46.2%), and eight three-unit (30.7%) fixed partial dentures were constructed. Overall, the implant success rate after 1 year of function in test and control groups was 100%. No prosthetic complications were recorded at follow-up visits.

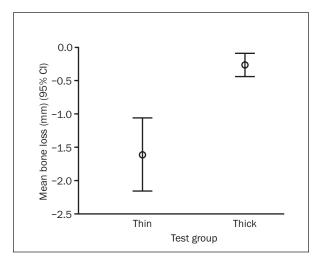
Mean ( $\pm$  standard error [SE]) bone loss around test implants in group A (thin mucosa) was 1.61  $\pm$  0.24 mm (range, 0.9 to 3.3 mm) on the mesial and 1.28  $\pm$  0.167 mm (range, 0.8 to 2.1 mm) on the distal. Therefore, the mean bone loss per implant in group A was 1.45  $\pm$  0.55 mm. The mean bone loss around test group B (thick mucosa) implants was 0.26  $\pm$  0.08 mm (range, 0.2 to 0.9 mm) on the mesial aspect and 0.09  $\pm$  0.05 mm (range, 0.2 to 0.6 mm) on the distal aspect, giving an overall mean of 0.17  $\pm$  0.19 mm bone loss per implant. The mean bone loss around control implants was 1.80  $\pm$  0.164 mm (range, 0.6 to 4.0) on the mesial and 1.87  $\pm$  0.166 mm (range, 0.0 to 4.1) on the distal, for an overall mean bone loss of 1.83  $\pm$  0.70 mm per implant.

The mean mucosa thickness around the group A implants was 1.95  $\pm$  0.3 mm; it was 3.32  $\pm$  0.76 mm around group B implants.

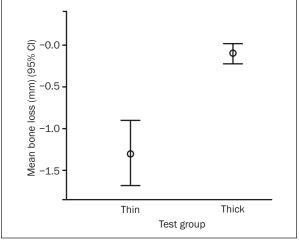
The Pearson correlation showed a significant positive relationship in the amount of bone loss between mesial and distal sites of control implants (r = 0.596; P= .003) and test implants (r = 0.853; P = .000) implants. Two-way ANOVA revealed a significant mean difference in terms of bone loss between test groups A and B on both the mesial ( $F_{[1,21]} = 38.7$ ; P =.001) and on the distal ( $F_{[1,21]} = 34.0$ ; P = .001). These differences are illustrated in Figs 6 and 7. The paired t test showed no significant difference between test group A (thin tissues) and the control group mesially (t[8] = .752; P = .474) and distally (t[8] = .859; P = .474).415). In contrast, there was a significant mean difference in crestal bone loss between test group B (thick tissues) and the control group both mesially (t[13] =8.624; P = .000) and distally (t[13] = 5.880; P = .000).

# **DISCUSSION**

The null hypothesis was rejected, since tissue thickness was shown to affect crestal bone stability around implants. The present study focused on the influence of gingival thickness at the time of surgery on crestal bone changes around nonsubmerged implants after 1 year of follow-up. The major finding was that positioning an implant 2 mm supracrestally did not prevent crestal bone loss if thin gingival tissues were present at the time of implant placement. All implants in test group A, with initially thin tissues,



**Fig 6** Mean bone loss around test implants on the mesial in the two groups (ANOVA,  $F_{1,21}$  = 38.7; P = .001).



**Fig 7** Mean bone loss around test implants on the distal in the two groups (ANOVA,  $F_{[1,21]}$  = 34.0; P = .001).

underwent additional bone loss on both the mesial and the distal. In contrast, implants in test group B, with a thick tissue pattern, had significantly less bone loss, compared to thin tissue test group A or the control group. In addition, there was no statistically significant difference between test implants with thin tissues and control implants. Bone loss around control implants was expected, as the placement of the microgap and the polished implant collar at the crestal level can cause marginal bone loss.

The decision to divide the test implants into two groups using the benchmark of 2.0 mm of gingival tissue thickness was based on the results of an animal study, which was the first attempt to analyze the influence of mucosal thickness on stability of bone. <sup>12</sup> In that experiment, the mucosal thickness in the test group was an average of about 2.0 mm; therefore this measurement was used as the means to distinguish between thin and thick mucosa.

An analysis of the literature on marginal bone loss helped the authors determine the study design (different apicocoronal implant positions). Two main factors responsible for early crestal bone loss were identified, namely the microgap and a polished implant collar. The microgap, if placed at the bone level or subcrestally, produces an infiltrate of inflammatory cells in the connective tissue at the implant-abutment connection. The inflammatory cells promote osteoclast formation, which results in alveolar bone loss.<sup>16</sup> Another factor associated with the microgap is the instability of the implant-abutment interface. It is suggested that micromovements of abutments can be linked to bone loss around implants.<sup>17</sup> The implant polished collar can stimulate crestal bone loss associated with a lack of loading; therefore it should be

positioned above the bone level. Because recent histologic<sup>18,19</sup> and clinical studies<sup>20–22</sup> have questioned the role of occlusion in the etiology of early crestal bone loss, occlusion was not taken into consideration in this study. The strength of the present study is in the measurement of gingival thickness at the crest just before implant placement. This allowed the authors to test the effect of gingival tissue thickness by isolating other factors as much as possible.

Radiographic measurements revealed variations in the extent of bone loss between mesial and distal sites around test and control implants. These differences can be explained by the fact that a flat alveolar ridge was not always available at the implantation site, such that some implants were placed on the ascending alveolar ridge. This resulted in different implant-abutment junction positions mesiodistally in relation to the bone level; however, the bone loss correlation between mesial and distal sites was shown to be significant.

The results of this clinical study are consistent with those of an animal study which showed the potential for thin tissues to cause crestal bone loss during the process of biologic width formation. In that experiment, during second-stage abutment placement surgery for test implants, the peri-implant mucosa was thinned to approximately 2 mm, whereas control implants had the healing abutments connected without alterations in tissue thickness. Histologic examination showed that for test implants, consistent bone resorption occurred after soft tissue healing, while the total extent of the biologic width was not statistically significantly different between test and control implants. The finding was explained on the basis of the assumption that the minimum dimension of

biologic width was not satisfied and bone resorption took place to allow a sufficient soft tissue attachment to form. However, the exact bone loss was not recorded in this animal experiment, making comparisons to the present trial difficult. It should be noted that Abrahamsson et al<sup>14</sup> expressed concern that implant sites with thin tissues were prone to develop angular defects around implants after healing.

The present findings contradict the assumptions that positioning of an implant-abutment junction above the bone level can prevent apical migration of bone.<sup>6,17,23</sup> The current study shows that stable crestal bone was maintained only at sites with thick tissue. These contradictory findings might have been obtained because of a lack of registration of initial mucosal thickness at a time of implant placement in other microgap studies.<sup>16,17,23–29</sup> If the mucosal factor had been considered, these studies may have been interpreted differently.

There have been few similar clinical studies. Kan et al<sup>30</sup> evaluated the difference between thick and thin biotypes of peri-implant mucosa by probing around restored implants in anterior arches. However, the primary width of the mucosa before implant placement was not recorded. In addition, bone loss and the position of the implant-abutment interface with respect to the bone crest were not reported. Cardaropoli et al<sup>7</sup> estimated gingival thickness before implant placement and calculated bone loss after 1 year of follow-up. However, the study design did not eliminate the influence of the microgap, as all implants were placed at the bone level; therefore, the results cannot be compared to the findings of the current study.

The process of biologic width formation around implants was described by Berglundh et al<sup>12</sup> in a dog study. The authors observed that the morphogenesis of peri-implant mucosa involved a loss of marginal bone. Two-piece implants (ITI Dental Implant System, Straumann) with a 2.8-mm polished neck were placed using a nonsubmerged technique, with the polished implant part and prosthetic abutment platform left above the bone crest. Dogs, which were used in the experiment, may have a thin mucosa type, as has been recorded in a number of prior studies. <sup>13,18</sup> In light of the results of the present experiment, it can be speculated that reduction of the marginal bone level occurred because of the thin mucosa biotype.

The present study has several limitations. The small sample size, especially in the test group with initially thin tissues (9 patients), could have negatively influenced the results. However, patient selection and implant placement were random; therefore, the number of patients in each test group could not be

increased or reduced by the researchers. In addition, the exclusion of some patients because of strict guidelines for radiographs also decreased the sample size. On the other hand, a number of earlier published and widely cited clinical trials used very similar<sup>30,31</sup> or even smaller sample sizes,<sup>6</sup> so it seems that sample size in the current experiment may be acceptable.

The radiographs were obtained using a parallel long-cone technique with film holders, and individual devices for the implants were not constructed. However, a similar approach was employed in prior prospective clinical studies<sup>31–33</sup>; therefore, the present technique can be considered adequate for bone loss measurements. In addition, Cameron et al<sup>34</sup> demonstrated that film position did not significantly influence the accuracy of measurements of the image if the tube head was maintained at less than 20 degrees from perpendicular to the long axis of the implant. Therefore, a careful examination of images for clear visibility of implant/abutment interface and threads was performed, and three patients were excluded from the present study.

Mucosal thickness was measured directly with periodontal probe. Compared to ultrasonic or radiographic measurement, this approach could be considered rather novel; however, it can be considered adequate for the assessment of tissue thickness. Lawson and Jones<sup>35</sup> have shown that direct visibility, which was achieved in the current study, is crucial for measurement precision. In addition, probing is considered a reliable procedure in evaluations of soft periodontal and peri-implant tissues.

# **CONCLUSION**

In spite of the aforementioned limitations, the study has significant theoretical and practical implications. The findings indicate that initially thin mucosal tissues can cause crestal bone loss after implant placement and 1 year in situ. If the initial tissue thickness is less than 2.5 mm, bone loss up to 1.45 mm can be expected within the first year of function. In thick tissues (2.5 mm or more), significant marginal bone recession could be avoided if the implant-abutment junction is positioned approximately 2 mm above the bone level; a negligible amount of bone loss (around 0.2 mm) would occur. Therefore, the authors recommend that supracrestal placement of implants be avoided if a thin mucosal biotype is present at an implant site. Furthermore, the measurement of gingival thickness should be mandatory in any evaluation of marginal bone loss. Finally, it is important to consider the thickening of thin mucosa before implant placement.

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