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Modeling of the buccal and lingual bone walls of fresh extraction sites following implant installation

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Key words: bone modeling, dehiscence, dogs, histometry immediate implants

Abstract

Objective: To determine whether the reduction of the alveolar ridge that occurs following tooth extraction and implant placement is influenced by the size of the hard tissue walls of the socket.

Material and methods: Six beagle dogs were used. The third premolar and first molar in both quadrants of the mandible were used. Mucoperiostal flaps were elevated and the distal roots were removed. Implants were installed in the fresh extraction socket in one side of the mandible. The flaps were replaced to allow a semi-submerged healing. The procedure was repeated in the contra later side of the mandible after 2 months. The animals were sacrificed 1 month after the final implant installation. The mandibles were dissected, and each implant site was removed and processed for ground sectioning.

Results: Marked hard tissue alterations occurred during healing following tooth extraction and implant installation in the socket. The marginal gap that was present between the implant and the walls of the socket at implantation disappeared as a result of bone fill and resorption of the bone crest. The modeling in the marginal defect region was accompanied by marked attenuation of the dimensions of both the delicate buccal and the wider lingual bone wall. Bone loss at molar sites was more pronounced than at the premolar locations.

Conclusion: Implant placement failed to preserve the hard tissue dimension of the ridge following tooth extraction. The buccal as well as the lingual bone walls were resorbed. At the buccal aspect, this resulted in some marginal loss of osseointegration.

Observations made on cadaver specimens [Rogers & Applebaum 1941] indicated that following tooth loss in the maxilla, the height of the ridge was reduced and its crest shifted palatally. Tyman & Tyman [1960], in a textbook chapter, stated that following the removal of teeth, the buccal alveolar bone plate resorbed much faster than the palatal plate. In the mandible, the authors claimed, the amount of bone resorption following tooth loss was rather similar in the buccal and lingual walls of the ridge. These claims, however, were not based on measurements but on anecdotal information.

Johnson [1963, 1969] monitored dimensional changes that occurred in 19 subjects (aged 14–45 years) scheduled for tooth extraction and complete denture therapy. Before and at various intervals after the removal of the teeth, cast models of the ridge were produced and scanned to disclose dimensional alterations. Johnson [1963] showed that following tooth extraction, (i) there was reduction of both the height (2.5–7 mm) and width (30–7 mm) of
the alveolar process, (ii) most change occurred in the first months, while (iii) minor additional diminution of the ridge continued over periods ranging between 10 and 20 weeks.

Pietrokovski & Massler (1967) examined plaster casts of the mandible and maxilla from 30 patients with complete natural dentition and measured the width of the various dentate segments. It was observed that the buccal and the lingual/palatal dimensions of the alveolar ridge of the right and left side of the jaws were almost identical. Subsequently, 149 dental casts were identified that had one tooth missing on one side of the jaw while the contra lateral tooth was still present. Measurement of amount of alveolar ridge resorption that had occurred after tooth extraction was obtained by a series of measurements in the edentulous and the opposite dentate regions. The authors concluded: ‘the amount of resorption was greater along the buccal surface than along the lingual or palatal surface in every specimen examined, although the absolute amounts and differences varied widely’.

Pietrokovski & Massler (1967), in addition, demonstrated that the amount of tissue attenuation was greater in the edentulous molar region than in the incisor and premolar regions of both the maxilla and the mandible. Schropp et al. (2003) studied changes of the alveolar ridge that took place following single-tooth extraction in 46 patients aged between 20 and 73 years. The alterations were studied in radiographs and on casts taken immediately after tooth extraction and at 3, 6 and 12 months of follow-up. The authors reported that following tooth extraction, the width of the ridge was reduced approximately 50% and that most change occurred during the first 3 months of healing. The change in the molar region was greater than in the premolar regions and more pronounced in the mandible than in the maxilla.

In this context, it must be realized that data describing ridge alterations in the studies referred were obtained from measurements that included both the hard and the soft tissues of the ridge. No attempt was made to separate the contribution of the
two components to the various dimensions assessed.

Paolantonio et al. (2001) included 48 patients in a study to determine the outcome of ‘implantation in fresh extraction sockets’. One implant was placed in an extraction socket and one implant in a fully healed ridge. The implants, together with surrounding tissues, were removed after 12 months and ground sections were prepared. The authors stated that when a ‘screw-type dental implant . . . is placed into a fresh extraction socket . . . the clinical outcome and degree of osseointegration does not differ from implants placed in healed, mature bone’. This study, however, did not report data for buccal–lingual sites and did not include information on whether loss of crestal bone height had occurred. Findings from experiments in humans and dogs, (Botticelli et al. 2003a, 2003b; Araujo et al. 2005) demonstrated that marked reduction of the height of the alveolar ridge consistently occurred following tooth extraction, and that implant installation in the fresh extraction socket did not interfere with the process of bone modeling. Further, in a clinical study comparing bone healing following implant placement immediately after tooth extraction or after 6–8 weeks, Covani et al. (2004) observed that marked reduction of the buccal–lingual width of the bone ridge had occurred 4–6 months after implant placement and independent of the timing of implant placement. The objective of the present experiment was to determine whether modeling of the alveolar ridge that occurs following tooth extraction and implant placement (i) was influenced by the size of the hard tissue walls of the socket, and (ii) would continue after the first 4 weeks of healing, i.e. once most of the effect of the surgical trauma was overcome.

Material and methods

The Ethics Committee for Animal Research at the University of Maringa, Brazil, approved the study protocol.

Six beagle dogs about 1-year-old were included in the experiment. The animals were fed a pellet diet and subjected to regular mechanical tooth and implant cleaning.

During surgical procedures, the dogs were anesthetized with intravenously administered Pentothal Natrium™ (30 mg/ml, Abbot Laboratories, Chicago, IL, USA). The third premolars and first molars in both quadrants of the mandible (3P3 and 1M1) were used as experimental teeth. The mesial root canals were reamed and filled with gutta-percha.

Mucoperiostal full-thickness flaps were elevated to disclose the buccal and lingual hard tissue wall of the ridge. The experimental teeth were hemi-sected and the distal roots were removed with the use of forceps. The buccal–lingual dimension of the entrance of the fresh extraction socket was measured using a sliding caliper.

Implants (Straumann® Standard Implant, 4.1 mm wide and 6 or 8 mm long; Straumann, Waldenburg, Switzerland) were installed in the fresh extraction sockets. The recipient sites were prepared for implant installation according to the guide-

### Table 1. Results of histometric measurements (mean and SD) describing the distance between the various landmarks

<table>
<thead>
<tr>
<th></th>
<th>SLA-C Buccal</th>
<th>SLA-C Lingual</th>
<th>SLA-B/I Buccal</th>
<th>SLA-B/I Lingual</th>
<th>C-B/I Buccal</th>
<th>C-B/I Lingual</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>4 weeks</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premolar</td>
<td>0.7 (0.6)</td>
<td>1.4 (0.2)</td>
<td>0.8 (0.7)</td>
<td>0.2 (0.1)</td>
<td>0.1 (0.4)</td>
<td>1.5 (0.6)</td>
</tr>
<tr>
<td>Molar</td>
<td>0.2 (0.9)</td>
<td>0.8 (0.9)</td>
<td>1.5 (0.3)</td>
<td>0.6 (0.5)</td>
<td>1.7 (1.5)</td>
<td>1.4 (1.7)</td>
</tr>
<tr>
<td><strong>12 weeks</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premolar</td>
<td>2.1 (0.5)</td>
<td>0.4 (0.3)</td>
<td>2.0 (0.5)</td>
<td>0.1 (0.2)</td>
<td>0.1 (0)</td>
<td>0.5 (0.3)</td>
</tr>
<tr>
<td>Molar</td>
<td>1 (0.7)</td>
<td>0 (0.9)</td>
<td>0.8 (0.8)</td>
<td>0.6 (0.6)</td>
<td>0.2 (0.3)</td>
<td>0.6 (0.6)</td>
</tr>
</tbody>
</table>

Negative values indicate that C or B/I were apical to SLA.

For abbreviations, see Fig. 2.
lines provided by the manufacturer. The marginal level of the modified, SLA-coated surface of the implant was following placement consistently located apical of the buccal bone crests (Fig. 1). Healing caps (Straumann® Dental Implant System, Waldenburg, Switzerland) were adjusted to the implants. The flaps and the wound margins were replaced and secured to allow a semi-submerged healing of the experimental sites. The sutures were removed after 2 weeks. The root extraction and implant installation procedure was first performed in the right side of the mandible. Two months later, an identical procedure was repeated in the left mandible. The animals were sacrificed 1 month after the second extraction and implantation procedure. The dogs were sacrificed with an overdose of Pentothal Natrium® (Abbott Laboratories, Chicago, IL, USA) and perfused, through the carotid arteries, with a fixative containing a mixture of 5% glutaraldehyde and 4% formaldehyde (Karnovsky 1965). The mandibles were dissected. Each implant site was removed using a diamond saw (Exact®, Apparatebau, Norderstedt, Hamburg, Germany). The biopsies were processed for ground sectioning according to the methods described by Donath & Breuner (1982) and Donath (1988). The samples were dehydrated in increasing grades of ethanol, infiltrated with methacrylate (Technovit® 7200 VLC-resin; Kulzer, Friedrichsdorf, Germany), polymerized and sectioned in the buccal–lingual plane using a cutting-grinding device (Exakt®, Apparatebau). From each biopsy unit, one buccal–lingual section representing the central area of the site was prepared. The sections were reduced to a thickness of about 20 μm by micro-grinding and polishing. The sections were stained in Ladewig’s fibrin stain (Donath 1993).

**Histological examination**

The examinations were made in a Leitz DM-RBE® microscope (Leica, Wetzlar, Germany).

In the sections, linear measurements (magnification × 16) were made between the following landmarks (Fig. 2):

- SLA: the marginal termination of the rough surface;
- C: the crest of the buccal or lingual bone wall; and
- B/I: the most coronal point of contact between bone and implant.

The width of the buccal and lingual bone walls was determined by measuring the distance between the buccal or lingual surface of the implant body and the outer surface of the hard tissue wall. The assessments were made at the SLA level and 1, 2 and 3 mm apical of SLA.

The mean values and standard deviation among animals were calculated for each variable and implant location.

**Results**

The mean buccal–lingual width of the entrance of the extraction socket of the premolar sites was 3.8 ± 0.3 mm, while
the corresponding dimension at the molar site was 5.8 ± 0.2 mm. Healing following tooth extraction and implant installation was uneventful. The gingiva in the premolar–molar regions as well as the peri-implant mucosa at clinical check-ups after the first two weeks was free from overt signs of inflammation (Fig. 3).

The thickness of the socket walls at various levels along the implant was determined in sections representing the central portion of the experimental site. Only sections in which the body of the implant was > 3.2 mm were used for the assessments. For this reason, one out of 30 sites had to be discarded.

**Histological and histometric observations**

**Peri-implant mucosa**

The mucosa at the implant sites was protected with a wide, well-keratinized oral epithelium that was continuous with a thin barrier epithelium that faced the implant surface. The dense connective tissue that resided between the two epithelial compartments was devoid of infiltrates of inflammatory cells.

**Implant sites after 4 weeks of healing (Table 1)**

At premolar sites (Fig. 4), the small marginal gap between the implant and the bone was occupied by various amounts of provisional connective tissue and newly formed woven bone (Fig. 5a, b). The crest of the lingual bone wall (Fig. 5a) was located about 1.4 mm coronal to the SLA border (Table 1) while the buccal crest (Fig. 5b) was consistently located at varying distance apical of this landmark (SLA-C: 0.7 ± 0.6 mm). No residual bone defect (C-B/I; Table 1) was seen at the buccal aspect but at the lingual aspect (Fig. 5) a 1.5 ± 0.6 mm deep angular hard tissue defect was present.

The center of the buccal and lingual bone walls was comprised of lamellar bone surrounded by newly formed bone. The number of bone multicellular units (BMUs) was larger in the lingual than in the buccal socket wall.

At the molar sites (Fig. 6), the tissue within the wide marginal gap was comprised of similar amounts of provisional connective tissue and newly formed bone (Fig. 7a, b). The depth of the residual hard tissue gap (Fig. 7a, b) at the buccal aspect was 1.7 ± 1.5 mm and 1.4 ± 1.7 mm at the lingual aspects (Table 2). The outer surface of both the buccal and lingual bone walls exhibited the

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**Table 2. Results of histometric measurements (mean and SD) describing the width of the buccal and lingual bone walls at 4 weeks and 12 weeks in premolar and molar sites**

<table>
<thead>
<tr>
<th>At SLA</th>
<th>At 1 mm</th>
<th>At 2 mm</th>
<th>At 3 mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buccal</td>
<td>Lingual</td>
<td>Buccal</td>
<td>Lingual</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>4 weeks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premolar</td>
<td>0 (0)</td>
<td>1.4 (0.8)</td>
<td>0.4 (0.3)</td>
</tr>
<tr>
<td>Molar</td>
<td>1.6 (1.3)</td>
<td>1.3 (0.5)</td>
<td>1.9 (0.8)</td>
</tr>
<tr>
<td><strong>12 weeks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premolar</td>
<td>0 (0)</td>
<td>1.1 (0.8)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Molar</td>
<td>0 (0)</td>
<td>0.7 (0.7)</td>
<td>0.9 (0.3)</td>
</tr>
</tbody>
</table>

For abbreviations, see Fig. 2.
presence of a large number of osteoclasts (Fig. 8a, b).

Implant sites after 12 weeks of healing (Table 1)
At the buccal aspect of the premolar sites, no residual hard tissue gap could be observed (Figs 9, 10). The crest (C) of the buccal bone wall as well as the level of bone-to-implant contact (B/I) was about 2 mm apical of the SLA border (SLA-C: $-2.1 \pm 0.5 \text{ mm}$, SLA-B/I: $-2 \pm 0.5 \text{ mm}$) (Fig. 10b). At the lingual aspect (Fig. 10a), a shallow ($0.5 \pm 0.3 \text{ mm}$ deep) marginal defect remained (C-B/I, Table 1).

At molar sites (Figs 11 and 12), the buccal bone crest was located $1 \pm 0.7 \text{ mm}$ apical of SLA while the marginal level of bone-to-implant contact was found on the average $0.8 \pm 0.8 \text{ mm}$ apical of the SLA border of the implant (SLA-B/I) (Fig. 12b). At the lingual aspect (Fig. 12a), a shallow ($0.6 \pm 0.6 \text{ mm}$ deep) angular defect (C-B/I) remained.

Width of the buccal and lingual bone walls (Table 2)
Four weeks of healing. In the premolar sites (Fig. 13, Table 2), no buccal bone was present at the SLA level, but the hard tissue wall was $0.4 \pm 0.3$, $0.8 \pm 0.2$, and $1 \pm 0.2 \text{ mm}$ wide at the reference levels 1, 2, and 3 mm apical of SLA. The corresponding dimensions for the lingual wall were $1.4 \pm 0.8 \text{ mm}$ at the level of SLA and $1.8 \pm 0.5$, $2.1 \pm 0.5$, and $2.3 \pm 0.5 \text{ mm}$ at more apically located levels.

In the molar sites (Fig. 14, Table 2), the overall buccal bone wall was thicker than its counterpart at the premolar sites. At the SLA level, the buccal bone was $1.6 \pm 1.3 \text{ mm}$ wide and increased in thickness ($1.9$, $2.2$, and $2.5 \text{ mm}$) the further apical of SLA the measurements were performed. The lingual bone of the molar site had a width that was similar to that of the premolar site.

Twelve weeks of healing. The measurements performed at the buccal aspect of the premolar sites (Fig. 13, Table 2) showed that no bone was present at the SLA and the 1 mm reference levels. At the 2 and 3 mm levels, the buccal bone wall was $0.3 \pm 0.3$ and $0.5 \pm 0.3 \text{ mm}$ wide. The lingual bone wall had a width that was similar to that observed at the 4-week interval.
In the molar sites (Fig. 14, Table 2), there was no bone present at the buccal SLA level, but at the 1, 2, and 3 mm reference levels, the buccal bone was 0.9 ± 0.3, 1.3 ± 1, and 1.7 ± 0.9 mm thick. The lingual bone wall was 0.7 ± 0.7 mm wide at the SLA level and 1.1 ± 0.8, 1.4 ± 0.6, and 1.7 ± 0.4 mm at the remaining levels.

Discussion

The present investigation demonstrated that marked hard tissue alterations occurred during healing following tooth extraction and implant installation in a fresh extraction socket. The marginal gap that was present between the implant and the walls of the socket at implantation disappeared as a result of bone fill and resorption of the crest regions of the hard tissue walls. The modeling in the marginal defect region was accompanied by marked attenuation of the dimensions of the buccal and lingual bone walls of the implant sites. Thus, implant placement evidently failed to preserve the hard tissue dimension of the ridge following tooth extraction.

Size of marginal defect

The extraction socket of the distal root of the mandibular third premolars and first molars was selected for implant installation as the size of the teeth and the dimensions of the ridge of the two sites are markedly different (Bartley et al. 1970). Implants of the same size [diameter 4.1 mm and length 8 mm] were placed in sockets of varying dimensions. As a result, the width and depth of the marginal defect (gap) between the implant and the walls of the socket were considerably larger in the molar than in the premolar locations [Fig. 1]. In the biopsy material, it was noted that the smaller (<0.3 mm wide) gaps at the premolar sites had been resolved already after 4 weeks of healing while the larger (1–1.3 mm) horizontal defects in the molar sites were completely resolved first in the 12-week specimens. These findings are in agreement with data presented by Botticelli et al. (2003a, 2003b) from experiments in the dog. They studied bone apposition in 1 mm wide marginal defects prepared around implants placed in a fully healed ridge. The defects were partially healed through appositional bone formation after 1 and 2 months but completely resolved with proper bone fill and bone-to-implant contact first after 4 months.

Crestal resorption

In the present experiment, it was observed that the process of bone apposition in the marginal gap region was accompanied by hard tissue alterations in the crestal regions of the buccal and lingual bone walls. In the premolar sites, these tissue alterations resulted in a marked reduction (>2 mm) of the height of the thin buccal crest and loss of bone-to-implant contact in the marginal portion of the implant. This finding is in agreement with data recently published by Botticelli et al. (2006). They studied healing of marginal defects that occurred at
implants placed in fresh extraction sockets in the premolar regions of the mandible of dogs. The authors reported that after 4 months, some bone fill had occurred in the more apical part of the marginal defects but also that this bone deposition was accompanied by marked loss of bone in the marginal segments of the socket.

The molar sites in the current study initially had a wider marginal defect than the premolar sites but a similar width of the buccal bone wall (Fig. 1). During the first 4 weeks of healing, the marginal gap was filled with newly formed bone and a provisional connective tissue that was replaced with woven bone in the later phase of healing. The width of the buccal wall in the molar sites was it hereby increased. Also, this wide hard tissue wall was, however, exposed to marked modeling and atenuation, although the ensuing diminution of its height had less effect on the level of bone-to-implant contact than was the case at the premolar sites (−0.8 vs. −2 mm). In other words, the wider the combined ‘defect and bone wall’ dimension was in the present study, the less the reduction of the bone-to-implant contact.

Reduction of the width of the socket walls

One important observation made in the present study was the marked reduction of the thickness of the buccal bone walls that occurred between 4 and 12 weeks (Table 2, Figs 13 and 14). It is obvious from the data reported that this decrease was more pronounced in the thicker buccal bone wall at molars than in the thinner wall at premolar sites. This finding is in agreement with data reported from measurements made on casts from edentulous regions in humans (Johnson 1963; Pietrokovski & Massler 1967; Schropp et al. 2003). In the studies referred to the amount of buccal resorption was much more pronounced than the resorption of the lingual aspect, and the resorption in the molar region was more substantial than that which occurred in the premolar region.

In the present analysis, it was noticed that the width of the lingual wall, at reference levels 1, 2, and 3 mm apical of SLA of the premolar but not at the molar sites, remained unchanged between 4 and 12 weeks. A further analysis of the histological sections revealed that new bone had formed on the outer aspects of the lingual wall of the premolar sites in this interval (Fig. 5a). Such incremental bone formation is normally the result of environmental influences such as load (Rubin et al. 1994). In the present experiment, the local environment, e.g. the markedly reduced dimension of the buccal bone wall may have stimulated bone formation at the lingual wall. It is, thus, suggested that the new bone that formed at the lingual surface compensated for bone loss that occurred at the buccal surface. Similar findings were
reported by Carmagnola et al. (1999) from experiments in dogs.

Position of implant in relation to bone walls

The shape (volume) of the alveolar process is determined by the form (size) of the teeth, their axis of eruption and inclination in occlusion [Schroeder 1986]. This means that at dentate sites, the size of the socket as well as its hard tissue walls may vary considerably. This fact must be considered when treatment planning calls for the placement of implants in fresh extraction sockets. Thus, the findings of the present experiment clearly showed that the thinner a bone wall of such a site and the closer to this wall the implant is placed, the higher the risk of compromised healing and occurrence of bone dehiscence.

References


