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Tissue modeling following implant placement in fresh extraction sockets

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Abstract

Objective: To study whether osseointegration once established following implant placement in a fresh extraction socket may be lost as a result of tissue modeling.
Material and methods: Seven beagle dogs were used. The third and fourth premolars in both quadrants of the mandible were used as experimental teeth. Buccal and lingual full-thickness flaps were elevated and distal roots were removed. Implants were installed in the fresh extraction socket. Semi-submerged healing of the implant sites was allowed. In five dogs, the experimental procedure was first performed in the right side of the mandible and 2 months later in the left mandible. These five animals were sacrificed 1 month after the final implant installation. In two dogs, the premolar sites on both sides of the mandible were treated in one surgical session and biopsies were obtained immediately after implant placement. All biopsies were processed for ground sectioning and stained.
Results: The void that existed between the implant and the socket walls at surgery was filled at 4 weeks with woven bone that made contact with the SLA surface. In this interval, (i) the buccal and lingual bone walls underwent marked surface resorption and (ii) the height of the thin buccal hard tissue wall was reduced. The process of healing continued, and the buccal bone crest shifted further in the apical direction. After 12 weeks, the buccal crest was located>2 mm apical of the marginal border of the SLA surface.
Conclusion: The bone-to-implant contact that was established during the early phase of socket healing following implant installation was in part lost when the buccal bone wall underwent continued resorption.

It is well established that tooth extraction will result in an apico-coronal as well as bucco-lingual (palatal) attenuation of the affected segment of the alveolar ridge (e.g. Pietrokovski & Massler 1967; Schropp et al. 2003; Araújo & Lindhe 2005; Araújo et al. 2005). Further, tissue alterations following tooth removal seem to result in more bone loss at the buccal than at the lingual/palatal aspects of the extraction site (Pietrokovski & Massler 1967; Botticelli et al. 2004; Araújo & Lindhe 2005). It was suggested that the thin buccal bone wall of the alveolar ridge, in the presence as well as in the absence of teeth, is more susceptible to surgical trauma and hence to resorption than its lingual counterpart (e.g. Wilderman et al. 1960; Wilderman 1963; Wood et al. 1972; Araújo et al. 2005). Moreover, the delicate marginal portion of the buccal bone wall frequently contains proportionally larger amounts of bundle bone than the lingual wall (Araújo et al. 2005). Bundle bone is a 'tooth-related' tissue (Schroeder 1986) that, following tooth loss, will model and eventually disappear (Araújo & Lindhe

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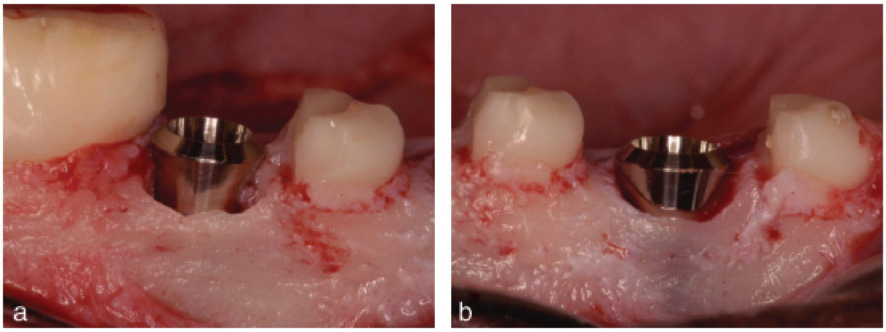

Fig. 1. Clinical photograph illustrating the position of the implants placed in the distal extraction socket of the mandibular third (a) and fourth (b) premolars. Note that the marginal level of the SLA surface of the implants is located below the buccal bone margin.

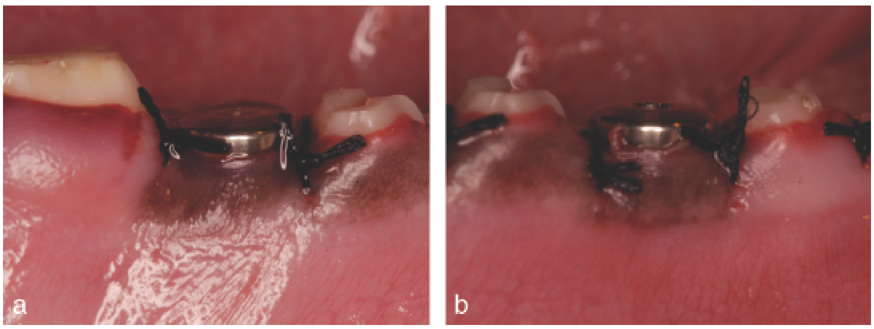

Fig. 2. Clinical photograph of the buccal aspect of the mandibular third (a) and fourth (b) premolars illustrating the mucosal flaps that had been replaced and secured with interrupted sutures.

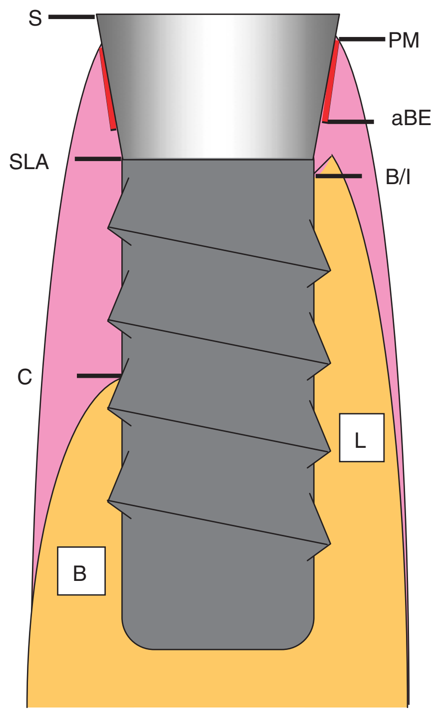

Fig. 3. Schematic drawing describing the different landmarks between which histometric measurements were performed. B, buccal; L, lingual; aBE, apical termination of the barrier epithelium; B/I, marginal level of bone-to-implant contact; C, marginal level of the bone crest; PM, margin of the peri-implant mucosa; S, implant shoulder; SLA, marginal level of the rough implant surface.

2005). This in turn may lead to a substantial reduction of the height of the buccal socket wall.

Paolantonio et al. (2001) suggested that implant placement in a fresh extraction socket may counteract this process of tissue modeling, and hence preserve dimensions of the alveolar ridge. Findings from experiments in dogs, however, failed to support this hypothesis (Araújo et al. 2005; Botticelli et al. 2005). Thus, it was demonstrated that (i) marked hard tissue resorption (in particular at the buccal aspect) inevitably occurred in the ridge following tooth extraction, (ii) implant installation apparently failed to interfere with the process that resulted in bone loss and (iii) the marginal portion of the implant after 3–4 months of healing was devoid of bone contact.

Botticelli et al. (2004) monitored hard tissue alterations that occurred following implant placement in fresh extraction sockets in humans. They measured the distance between the implant and the inner and outer surfaces of the buccal and lingual/palatal bone walls at implant installation and at re-entry after 4 months. The authors reported that during healing, the bone walls underwent marked 'horizontal resorption'. At the 4-month re-entry, it was also observed that there was a complete resolution of the large marginal defect that was present at the time of implant installation. Moreover, although the bone walls were substantially reduced in width, the rough surface of the implant was almost consistently covered with an albeit thin layer of bone.

Taken together, the observations made in the studies referred indicate that implant placement in a fresh extraction socket may result in an early hard tissue fill of the marginal defect. In a later phase of tissue remodeling following tooth extraction, however, this newly formed marginal bone may in part be lost.

The objective of the present experiment was to test the hypothesis that 'osseointegration' may be lost as a result of the physiological modeling that occurs following tooth extraction and implant installation.

Material and methods

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

Sample

Seven beagle dogs about 1-year old and weighing about 12–13 kg each were used. During surgical procedures, the animals were anesthetized with intravenously administered Pentothal Natrium® (30 mg/ml; Abbot Laboratories, Chicago, IL, USA). Throughout the experiment, the animals were (i) fed a pellet diet and (ii) subjected to mechanical tooth and implant cleaning once every second day with the use of a toothbrush and dentifrice.

Procedure

The third and fourth premolars in both quadrants of the mandible ($_3P_3$ and $_4P_4$) were used as experimental teeth. A rubber dam was placed around the four premolars, and the pulp tissue of the mesial roots was removed. The root canals were filled with gutta-percha and the coronal pulp chambers were sealed with light curing cement.

Sulcus incisions were placed along the buccal and lingual aspects of the premolars and 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 carefully removed with the use of forceps.

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 guidelines provided by the manufacturer. The implants were placed so that the marginal level of the SLA-coated surface was flush with or slightly apical of the buccal bone crest (Fig. 1). Healing caps (Straumann® Dental Implant System, Waldenburg, Switzerland) were adjusted to the implants. The mobilized buccal and lingual flaps were replaced to allow a semi-submerged healing of the implant sites. The wound margins were stabilized with interrupted sutures (Fig. 2).

Schedule

In five dogs, the experimental procedure was first performed in the right side of the mandible. Two months later, an identical procedure was repeated in the left mandible. These five animals were sacrificed 1 month after the final implant installation to provide specimens representing 4 and 12 weeks of healing. During the first week after surgery, the animals received Amoxicillin (500 mg, twice daily) via the systemic route.

In two dogs, the premolars on both sides of the mandible were treated in one surgical session. These two dogs were sacrificed within 2 h after implant installation.

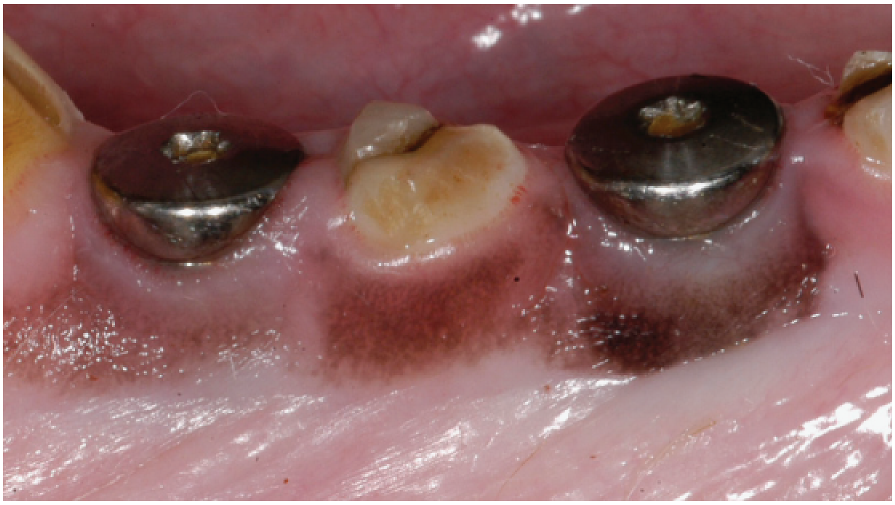

Fig. 4. Clinical photograph illustrating the implant sites after 12 weeks of healing. The peri-implant mucosa had normal texture and color and was free of signs of inflammation.

Biopsy

The dogs were sacrificed with an overdose of Pentothal Natrium® (Abbot 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 and placed in the fixative. Each implant site was removed using a diamond saw (Exact® Apparatebeau, 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 and infiltrated with methacrylate (Technovit® 7200 VLC-resin; Kulzer, Friedrichsdorf, Germany). Following embedding, the biopsies were polymerized and sectioned in the buccal–lingual plane using a cutting–grinding device (Exakt®, Apparatebau, Norderstedt, Germany). From each biopsy unit, two buccal–lingual sections representing the central area of the site were prepared. The sections

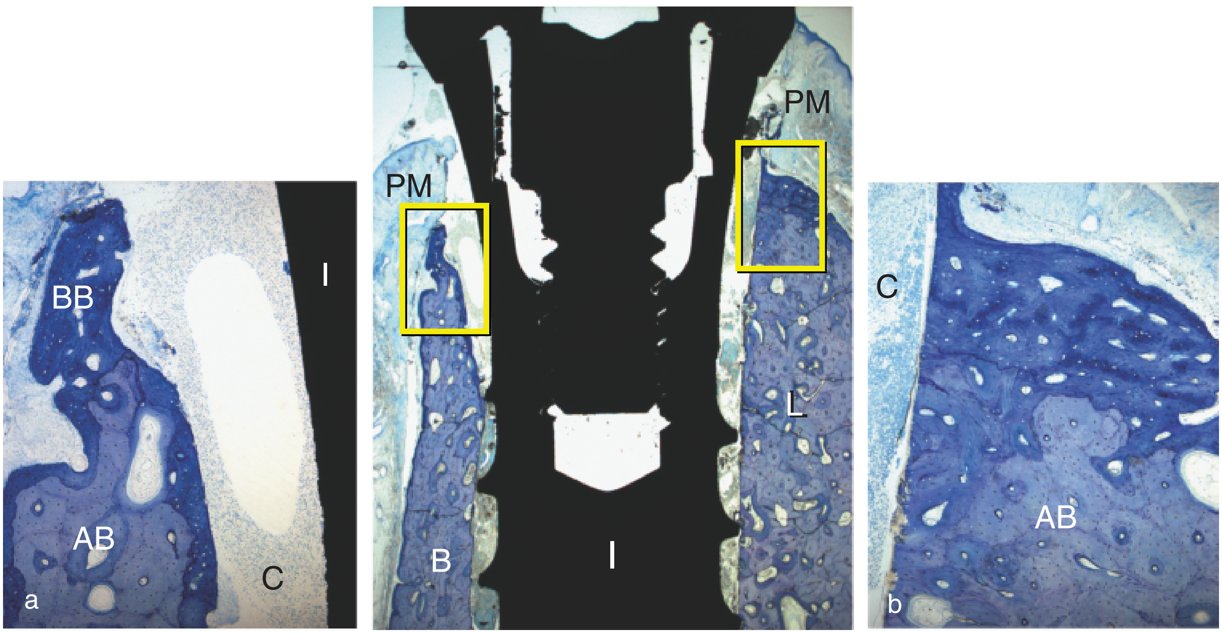

Fig. 5. Buccal–lingual section representing Day 0. B, buccal bone wall; I, implant; PM, peri-implant mucosa. Higher magnification of the outlined buccal (a) and lingual (b) crest areas. AB, alveolar bone; BB, bundle bone; C, coagulum. Toluidine blue staining; original magnifications × 1.6 and × 10.

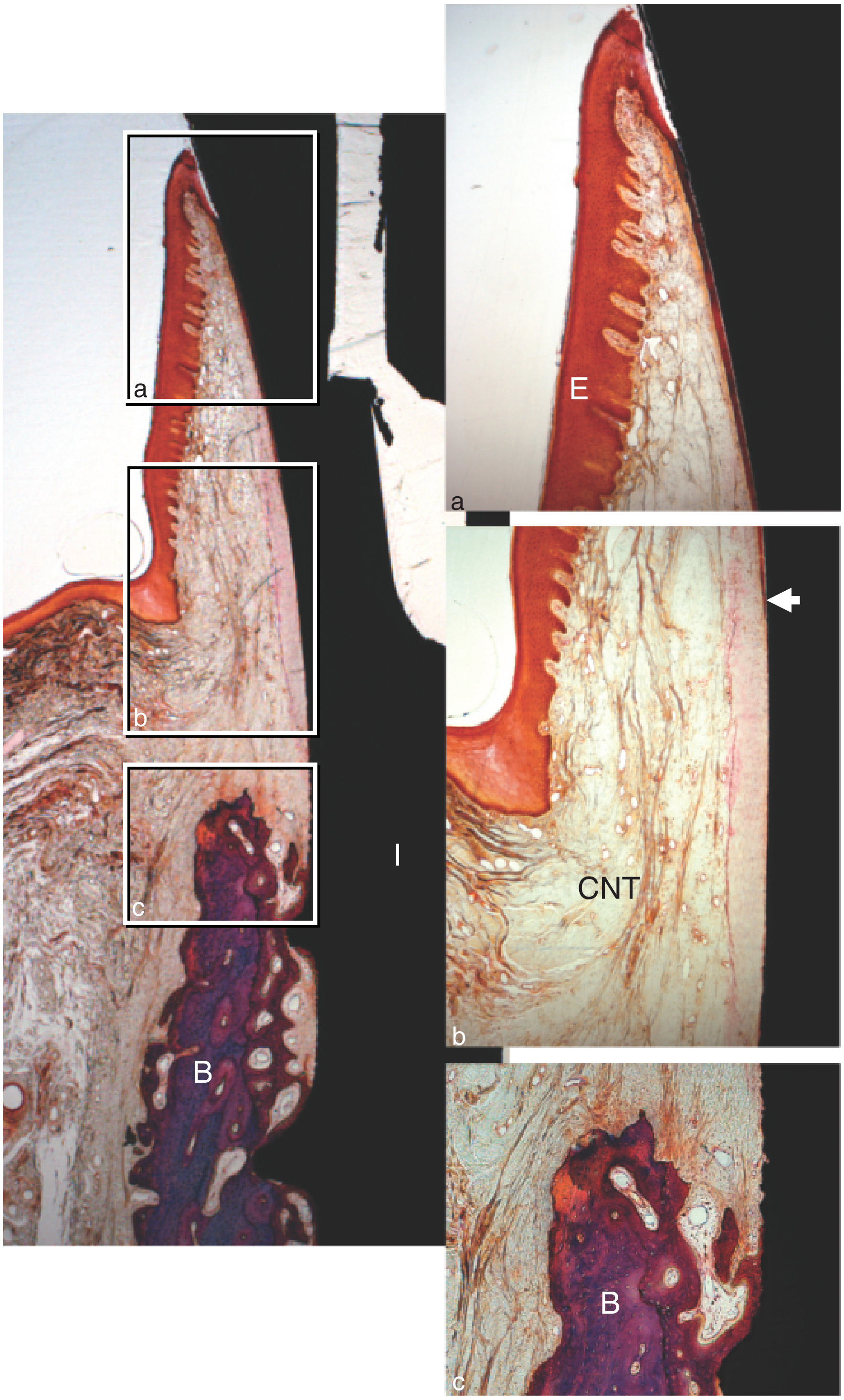

Fig. 6. Buccal–lingual section representing the buccal aspect of the implant after 4 weeks of healing. The insets show higher magnifications of the areas outlined. Note the presence of a thin barrier epithelium and that the connective tissue is devoid of infiltrates of inflammatory cells. The bone crest exhibits signs of new bone formation as well as resorption. I, implant; B, bone; CNT, connective tissue; E, oral epithelium; arrowhead, apical termination of the barrier epithelium. Ladewig's fibrin staining; original magnification × 2.5 and insets × 10.

were reduced to a thickness of about 20 μm by micro-grinding and polishing. The sections were stained in Toluidine blue or 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, the following landmarks were identified (Fig. 3):

S: the implant shoulder;
SLA: the marginal termination of the rough surface;
C: the crest of the buccal or lingual bone wall;
B/I: the most coronal point of contact between bone and implant;
PM: the margin of the peri-implant mucosa; and
aBE: the apical termination of the barrier epithelium.

Linear measurements (magnification × 16) were made to determine the distance between the landmarks. In sections representing Day 0, B/I and aBE were not identified.

Data analysis

The mean values and standard deviations among animals were calculated for each variable.

Results

Healing following tooth extraction was uneventful. The peri-implant mucosa at the 4- and 12-week intervals was free from clinical signs of inflammation (Fig. 4).

Histological observations

Day 0

The buccal bone wall of the socket (Fig. 5) was markedly thinner than the lingual wall. Bundle bone was present only in the marginal portion of the buccal and lingual walls. In other parts of the socket, the layer of bundle bone had obviously been removed in the preparation of the socket for implant installation.

The pitch of the implant made contact with the cortical bone in discrete regions in the middle and apical portions of the recipient site while in most areas a blood clot was seen to occupy the space between the metal body and the bone tissue. The crest (C) of the buccal as well as the lingual wall of the socket was located in a position coronal to the SLA border (SLA). The distance between the crest of the bone and the surface of the implant was similar at the buccal and lingual aspects of the site and varied between 0.4 and 0.2 mm.

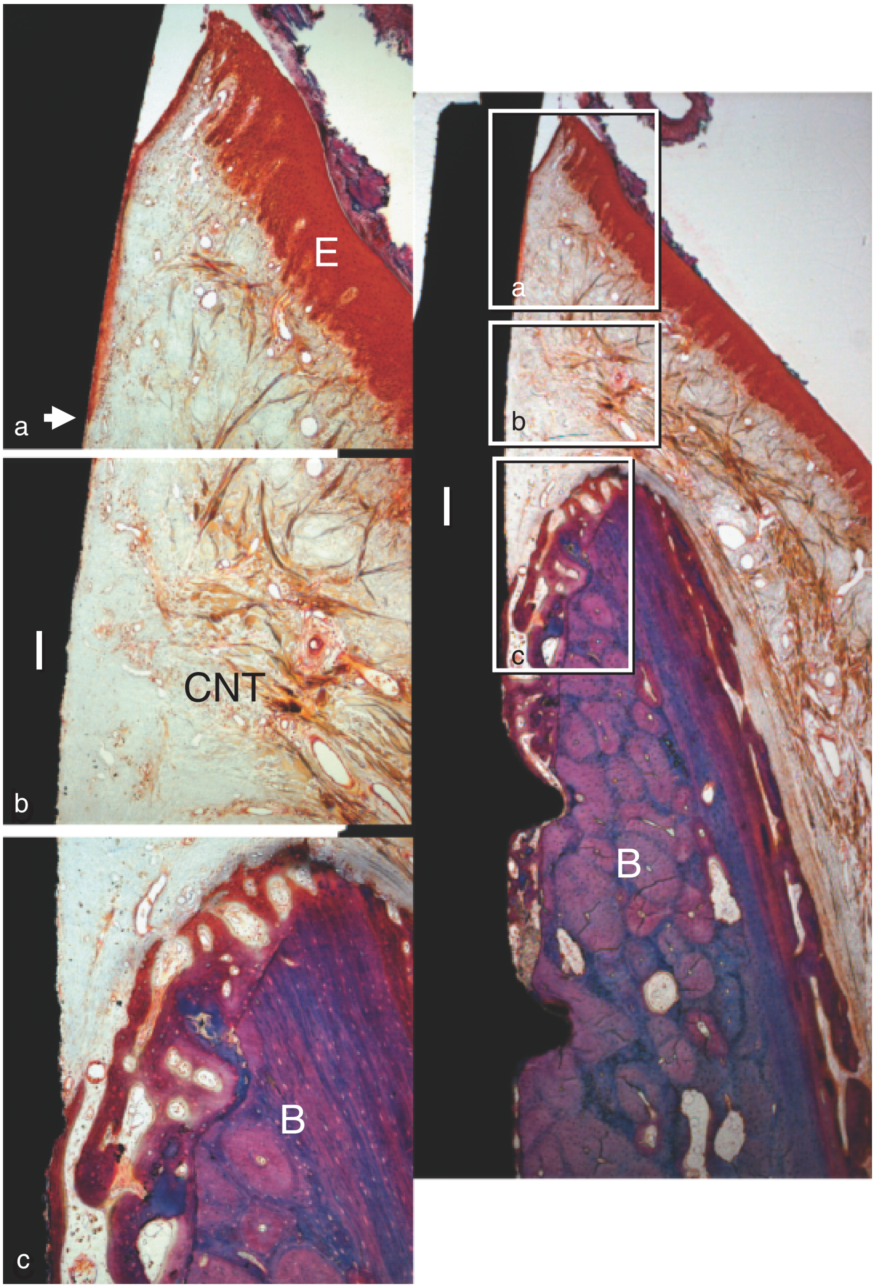

Fig. 7. Buccal–lingual section representing the lingual aspect of the implant after 4 weeks of healing. The insets (a–c) represent a higher magnification of the areas outlined. The thin barrier epithelium is in the apical direction continuous with a connective tissue devoid of infiltrates of inflammatory cells. The newly formed bone in the crest region is close to but not in contact with the smooth surface of the abutment portion of the implant. I, implant; B, bone; CNT, connective tissue; E, oral epithelium; arrowhead, apical termination of the barrier epithelium. Ladewig's fibrin staining; original magnification $\times$ 2.5 and insets $\times$ 10.

Four weeks

The peri-implant mucosa at 4 weeks (Fig. 6) was covered with a well-keratinized oral epithelium that was continuous with a short barrier epithelium that was facing the polished abutment portion of the implant. The connective tissue interposed between the apical cells of the barrier epithelium and the bone crest at both the buccal (Fig. 6) and the lingual (Fig. 7) aspects of the site was devoid of inflammatory cells but was comprised of mesenchymal cells, well-organized collagen fiber bundles and vascular structures.

The gap between the implant and the marginal bone that in the Day 0 specimens was filled with blood was occupied in this interval by provisional connective tissue and newly formed bone including woven bone, parallel-fibered and lamellar bone (Fig. 8). In more apical portions of the experimental sites, it was noted that the void between the implant and the socket walls was occupied by newly formed woven and lamellar bone and that the newly formed bone occurred around vascular structures (Fig. 9).

The center of the buccal and lingual bone walls was comprised of varying amounts of old lamellar bone that was surrounded by newly formed bone that was in contact with the implant surface. Large numbers of osteoclasts were found on the outer aspects of the bone crests, while the central portions of the walls contained large numbers of bone multicelluar units (BMUs) (Fig. 8). The number of BMUs was larger in the lingual than in the buccal socket wall. The crest of the lingual bone wall was close to the SLA border, while the buccal crest was consistently located at varying distance apical of this landmark.

Twelve weeks

The margin of the peri-implant mucosa (Figs 10 and 11) at both the buccal and lingual aspects of the implant site was located at or a short distance apical of the implant shoulder. The barrier epithelium at this interval was about 2 mm long and was in the apical direction continuous with a dense, apparently well-organized connective tissue virtually free of infiltrates of inflammatory cells. The zone of connective tissue attachment was considerably longer at the buccal than at the lingual aspect of the implant site.

The crest of the lingual bone wall was located close to the SLA border, while the buccal crest had a more apical location (Fig. 12). In several specimens, it was observed that islands or a continuous, thin layer of woven bone lined a portion of the implant surface coronal to the buccal bone crest. The contact region between the implant and the bone (Fig. 13) was characterized by the presence of primary osteons comprised of similar amounts of woven, parallel-fibered and lamellar bone.

Histometric observations

In biopsies representing Day 0, it was observed that the bone crest (C) was located on the average -0.4 ± 0.2 mm (buccal) and 1.1 ± 0.5 mm (lingual) coronal of SLA (Table 1). During the process of healing, the crest of the lingual bone wall

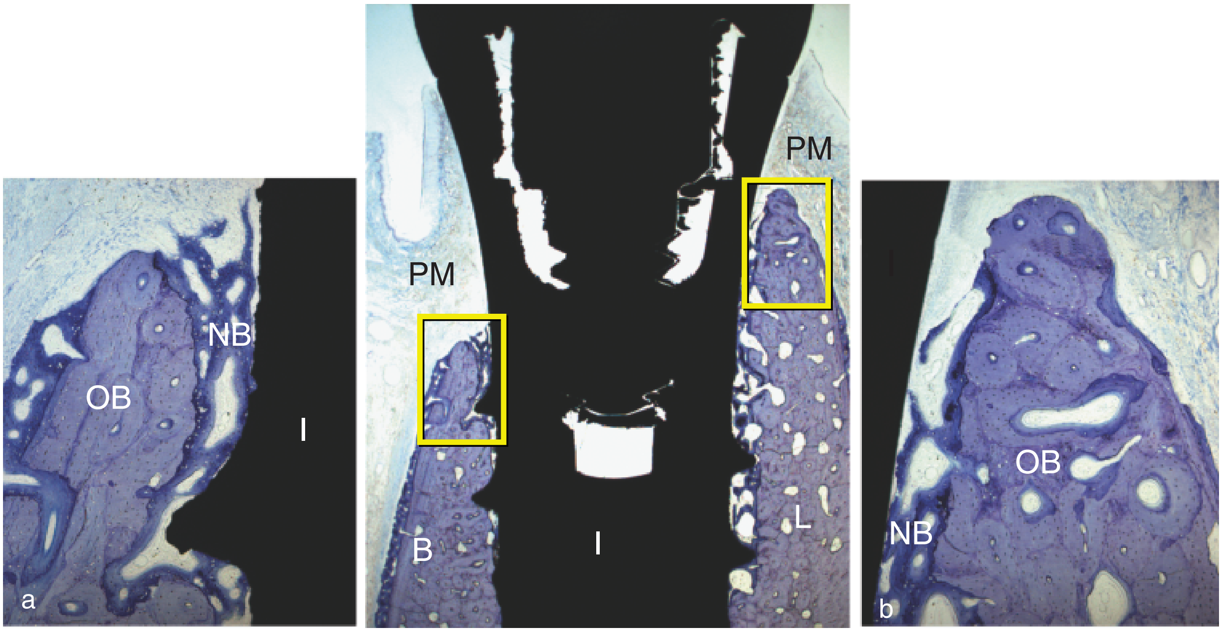

Fig. 8. Buccal–lingual section representing 4 weeks of healing. I, implant; PM, peri-implant mucosa. Higher magnification of outlined buccal (a) and lingual (b) crest regions. OB, old bone; NB, newly formed bone. Toluidine blue staining; original magnifications $\times$ 1.6 and $\times$ 10.

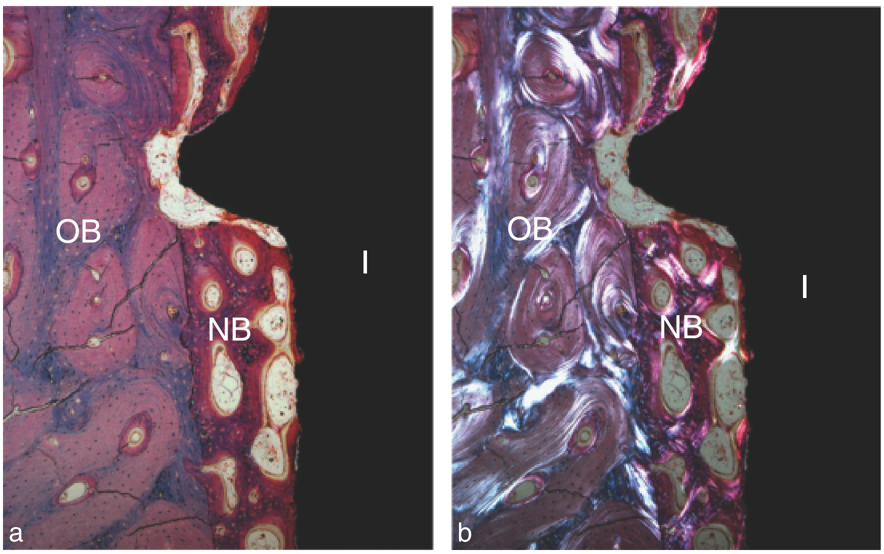

Fig. 9. Buccal–lingual section representing the buccal aspect of the implant after 4 weeks of healing; transmitted light (a) and polarized light (b). The newly formed bone is comprised of woven bone, parallel-fibered bone and also lamellar bone in discrete areas. I, implant; OB, old bone; NB, newly formed bone. Ladewig's fibrin staining; original magnification $\times$ 10.

***Table 1.* Histometric measurements of the peri-implant bone defect (mean (SD))**

	SLA-C		SLA-B/I	
	Buccal	Lingual	Buccal	Lingual
Day 0	0.4 (0.2)	1.1 (0.5)	–	–
4 weeks	− 0.7 (0.5)	1 (1)	− 0.8 (0.6)	− 0.4 (0.6)
12 weeks	− 2.1 (0.4)	0.4 (0.4)	− 2 (0.5)	− 0.1 (0.1)

Negative values indicate that C or B/I are apical to SLA.
For abbreviations, see Fig. 3.

remained comparatively unchanged (SLA-C lingual: at 4 weeks = 1 $\pm$ 1 mm, at 12 weeks 0.4 $\pm$ 0.4 mm), while a marked reduction of the height of the buccal bone wall obviously occurred (SLA-C buccal; at 4 weeks = − 0.7 $\pm$ 0.5 mm, at 12 weeks – 2.1 $\pm$ 0.4 mm). In other words, at the 12-week interval the buccal bone crest was located on the average 2.5 mm apical of its lingual counterpart. The marginal level of B/I after 4 weeks of healing was located 0.8 $\pm$ 0.6 mm (buccal) and 0.4 $\pm$ 0.6 mm (lingual) apical of SLA (SLA-B/I; Table 1). In sections representing 12 weeks, the corresponding dimensions were 2 $\pm$ 0.5 mm (buccal) and 0.1 $\pm$ 0.1 mm (lingual) below the SLA border.

The margin of the PM at the buccal side was in the Day 0 samples (Table 2) located 0.6 $\pm$ 0.2 mm apical of the implant shoulder (S) while at the lingual side, the corresponding distance was 0.6 $\pm$ 0.5 mm coronal to S. At the 4-week interval, PM was close to the S level at both buccal and lingual aspects. At 12 weeks, PM was located slightly below S (buccal 0.6 $\pm$ 0.3 mm and lingual 0.2 $\pm$ 0.3 mm).

The height of the PM-B/I at the buccal and lingual aspects of the implants varied in the 4-week sections between 3.3 mm (buccal) and 3.5 mm (lingual). The matching dimensions in the 12-week specimens were 4.2 $\pm$ 0.8 mm (bucal) and 2.7 $\pm$ 0.2 mm (lingual). The PM-aBE was

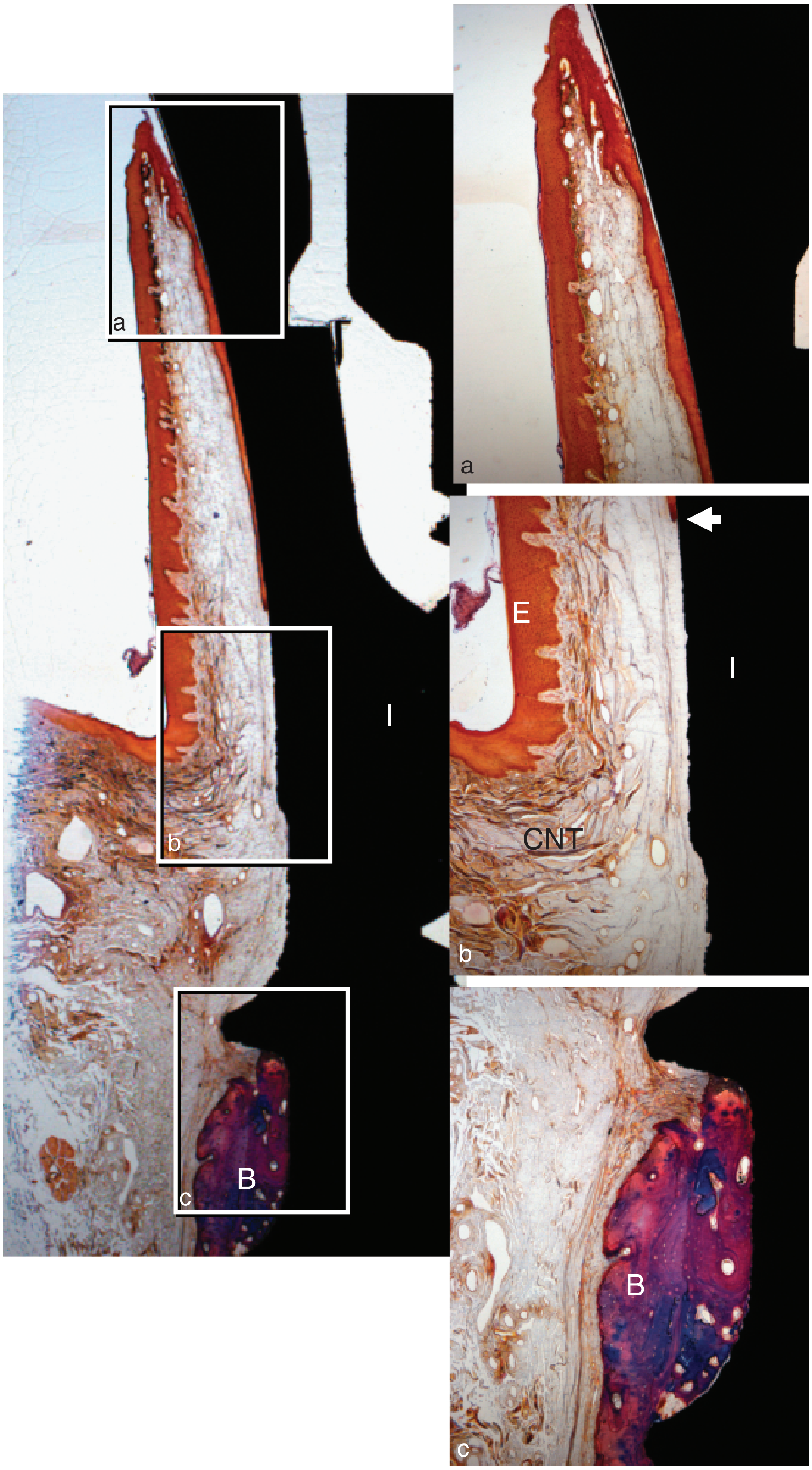

Fig. 10. Buccal–lingual section representing the buccal aspect of the implant after 12 weeks of healing. The insets represent a higher magnification of the outlined areas. The margin of the peri-implant mucosa is located slightly apical of the implant shoulder. The connective tissue lateral to as well as apical to the barrier epithelium is free of infiltrates of inflammatory cells. The surface of the bone crest exhibits signs of ongoing remodeling. I, implant; B, bone; CNT, connective tissue; E, oral epithelium; arrowhead indicates apical termination of the barrier epithelium. Ladewig's fibrin staining; original magnification $\times$ 2.5 and insets $\times$ 10.

in biopsies representing 12 weeks of healing was about 2-mm-long 2.2 $\pm$ 0.3 mm buccal and 2.1 $\pm$ 0.4 mm lingual. The apical termination of the barrier epithelium at 12 weeks was found to be closer to the B/I location at the lingual (0.6 $\pm$ 0.2 mm) than at the buccal (1.9 $\pm$ 0.6 mm) aspect of the implant.

Discussion

In the present experiment, it was observed that the void which at surgery occurred between the marginal portions of the implant and the walls of the fresh socket became filled with a coagulum. This coagulum after 4 weeks had been replaced with newly formed bone that, in the marginal gap region, also made contact with the rough surface of the implant. In this initial 4-week interval, (i) the buccal and lingual bone walls underwent pronounced surface resorption, (ii) the bundle bone in the marginal region was resorbed and (iii) the height of the thin buccal hard tissue wall was markedly reduced. Between 4 and 12 weeks, the process of healing continued, and the height of the buccal bone crest was further reduced.

The peri-implant mucosa

All implant sites monitored in the present experiment healed under non-submerged conditions. Hence, a portion of the abutment part of the one-piece implant was throughout the 12 weeks of monitoring exposed to the oral cavity and thus available for plaque formation. The plaque control program managed during the 3 months of study, however, effectively prevented plaque buildup and the development of peri-implant mucositis, i.e. conditions that may have interfered with wound healing including bone resorption and apposition (e.g. Polson et al. 1976; Schroeder et al. 1976; Berglundh et al. 2003). In other words, dimensional alterations of the soft and hard tissues that occurred around the implants in the current experiment were not influenced by plaque-associated inflammatory lesions in the mucosa.

Osseointegration

In specimens obtained about 2 h after tooth extraction and implant installation (Day 0), it was observed that a coagulum including

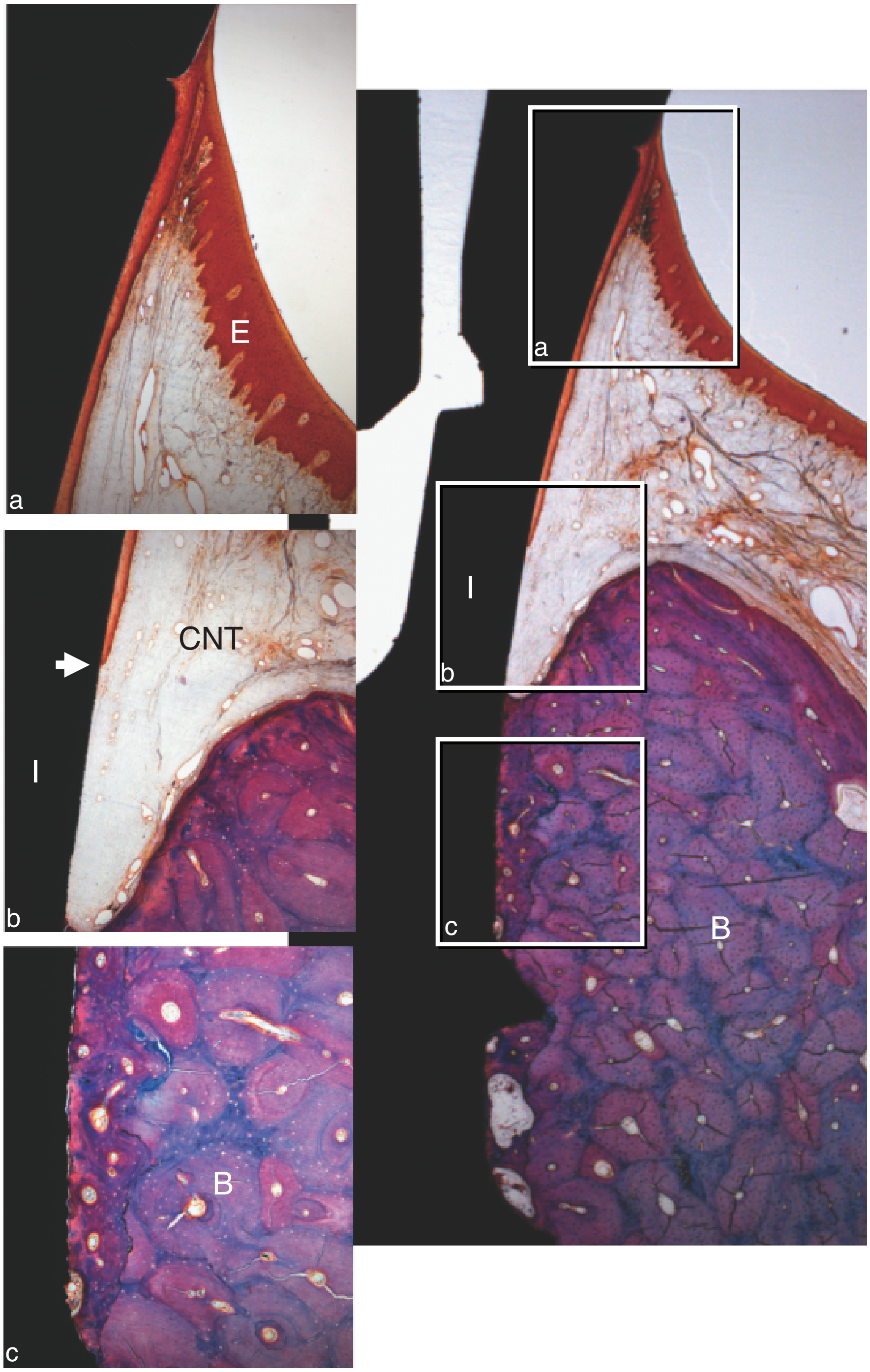

Fig. 11. Buccal–lingual section representing the lingual aspect of the implant after 12 weeks of healing. The insets (a–c) represent a higher magnification of the outlined areas. The margin of the peri-implant mucosa is located slightly apical of the implant shoulder. The connective tissue lateral to, as well as apical to the barrier epithelium is free of infiltrates of inflammatory cells. The surface of the bone crest exhibits signs of ongoing remodeling. I, implant; B, bone; CNT, connective tissue; E, oral epithelium; arrowhead, apical termination of the barrier epithelium. Ladewig's fibrin staining; original magnifications $\times$ 2.5 and $\times$ 10.

cells entrapped in a delicate fibrin network had formed in the marginal gap that occurred between the implant and the walls of the extraction socket. This coagulum was after 4 weeks replaced with newly formed, immature bone that was in apparent contact with the rough surface of the implant. This observation is in agreement with the findings previously reported (e.g. Sennerby et al. 1993; Berglundh et al. 2003; Botticelli et al. 2003; Abrahamsson et al. 2004). The present experiment, however, also documented that in the vicinity of the implant, the trabeculae of this newly formed woven bone were reinforced by layers of lamellar bone and parallel-fibered bone. This finding is in agreement with data by Johner (1972), who studied bone repair in small cortical bone defects in rabbits. He concluded that in such bone defects, mechanically stable conditions prevail and this allows the deposition of parallel-fibered bone and lamellar bone along with the woven bone.

An important observation made in the present study was that a continued resorption of the crest region occurred in both the buccal and lingual bone walls between 4 and 12 weeks. This progressive resorption of the comparatively thin buccal bone wall included the newly formed B/I ('osseointegration'). As a result, a bone dehiscence of >2 mm became established at the buccal aspect of the implant. The occurrence of such a bone dehiscence as a result of bone resorption following implant placement corroborates the findings previously reported from experiments in dogs (e.g. Araújo & Lindhe 2005; Araújo et al. 2005) and studies in humans (e.g. Spray et al. 2000; Cardaropoli et al. 2006).

Buccal and lingual bone walls

In the Day 0 samples of the present biopsy material, it was observed that the buccal bone wall of the implant site was markedly thinner than its lingual counterpart (Fig. 5). This is in agreement with findings previously reported for extraction sockets (e.g. Spray et al. 2000; Araújo & Lindhe 2005) and tooth sites (e.g. Wilderman et al. 1960; Araújo et al. 2005) in humans and dogs.

In biopsies representing 4 weeks of healing, the outer surfaces of the bone walls exhibited marked signs of ongoing resorption and remodeling. In addition, the height of the buccal bone wall was reduced about 1 mm while only minute height alterations occurred at the lingual aspect. There are reasons to suggest that, at least in part, this bone loss was the result of the surgical trauma that included flap elevation and detachment of the periosteum from the cortical bone plate. The degree of bone resorption that occurred during the first month in the present study corresponds to hard tissue changes observed following periodontal surgery as reported by e.g. Kohler & Ramfjord (1960), Wilderman et al. (1960) and Araújo et al.

Table 2. **Histometric measurements of the peri-implant soft tissue component (mean (SD))**

	PM-S*		PM-B/I		PM-aBE		ABE-B/I	
	Buccal	Lingual	Buccal	Lingual	Buccal	Lingual	Buccal	Lingual
Day 0	– 0.6 (0.2)	0.6 (0.5)	–	–	–	–	–	–
4 weeks	– 0.3 (0.3)	0.2 (0.3)	3.3 (0.6)	3.5 (0.3)	1.3 (0.5)	1.7 (0.6)	2 (0.5)	2 (0.9)
12 weeks	– 0.6 (0.3)	– 0.2 (0.3)	4.2 (0.8)	2.7 (0.2)	2.2 (0.3)	2.1 (0.4)	1.9 (0.6)	0.6 (0.2)

*Negative values indicate that PM is apical to S.
PM, margin of the peri-implant mucosa; S, implant shoulder; B/I, most coronal point of contact between bone and implant; aBE, apical termination of the barrier epithelium.

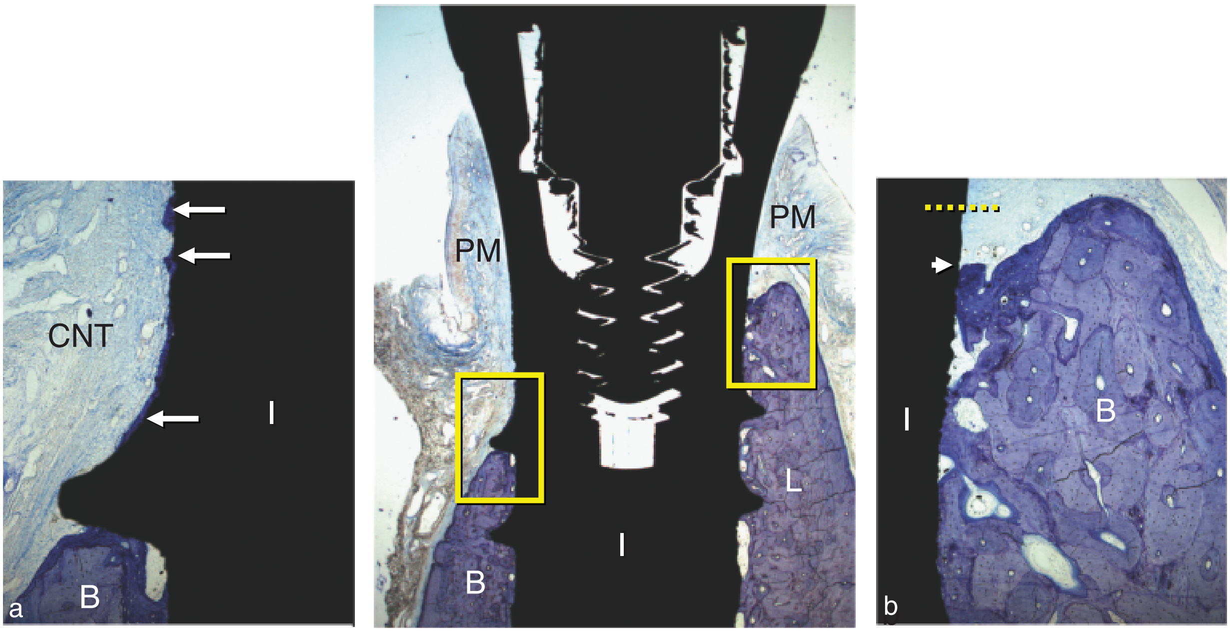

Fig. 12. Buccal–lingual section representing 12 weeks of healing. The insets represent a higher magnification of the outlined areas (a, buccal; b, lingual). At the buccal aspect of the implant (arrows), remnants of the newly formed bone can be observed. The bone-to-implant contact level at the lingual aspect is located close to the SLA level (dotted line; b). I, implant; B, bone; CNT, connective tissue; arrowhead, most coronal portion of the bone-to-implant contact. Toluidine blue staining; original magnification × 1.6 and insets × 10.

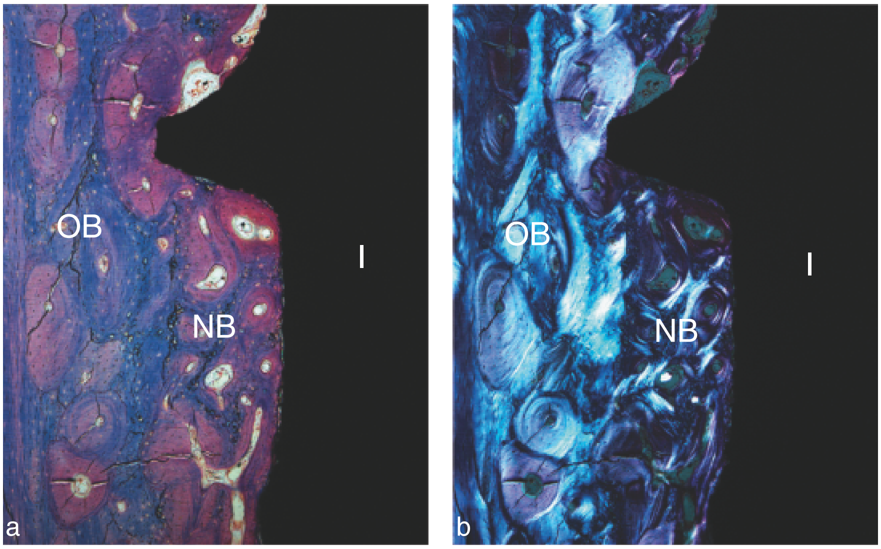

Fig. 13. Buccal–lingual section from the buccal aspect of the implant after 12 weeks of healing; transmitted light (a) and polarized light (b). The newly formed bone (NB) that is in contact with the implant is comprised of primary osteons including woven bone, parallel fibered bone and also in discrete areas lamellar bone. I, implant; NB, newly formed bone; OB, old bone. Ladewig's fibrin staining; original magnification × 10.

(2005). The fact that the buccal, but not the thicker lingual, bone wall was reduced in height is also in agreement with data showing that tooth sites with thin buccal bone tissue exhibit the most pronounced bone loss during healing following periodontal procedures that call for flap elevation (Wood et al. 1972; Yaffe et al. 1994; Araújo et al. 2005).

In the interval between 4 and 12 weeks, the dimensions of both the buccal and the lingual bone walls were further reduced. While the alterations of the lingual wall were minute, the buccal bone crest was reduced both in width and height. At the 12-week examination, the crest of the buccal bone was located about 2.5 mm apical of its lingual counterpart. This discrepancy between the buccal and lingual walls of the implant sites confirms findings previously reported from studies in humans (e.g. Pietrokovski & Massler 1967; Schropp et al. 2003; Botticelli et al. 2004) and from experiments in dogs (e.g. Araújo & Lindhe 2005; Araújo et al. 2005). It is suggested that tissue alterations that occurred between 4 and 12 weeks were related to the functional adaptation of the alveolar ridge that occurred after the loss of the teeth.

要旨

目的：新鮮抜歯窩に埋入したインプラントの骨性結合が、組織モデリングの結果、失われるかどうかを検討すること

材料と方法：７匹のビーグル犬を用いた。下顎両側の第３、第４前臼歯を実験に用いた。頬側と舌側で全層フラップを挙上し、遠心根を抜去し、インプラントを新鮮抜歯窩に埋入した。準埋入式の治癒を図った。５匹の犬では下顎右側で同処置を行い、2ヶ月後に下顎左側で行った。これら５匹の動物は最後のインプラント埋入後１ヶ月後に屠殺した。２匹の犬では、下顎両側前臼歯において１回の手術で処置を施し、インプラント埋入直後に組織生検を行った。全ての生検組織から、研磨切片を製作し、染色した。

結果：手術時にインプラントと抜歯窩骨壁の間に存在していた空隙は４週間後には網状骨によって埋まっており、SLA表面に接触していた。この間に(i) 頬側と舌側の骨壁は顕著な表面吸収を起こし、(ii)薄い頬側骨壁の高径は減少した。治癒の過程が進むにつれ、頬側の骨頂はさらに根尖側に移動した。１２週間後に頬側の骨頂はSLA表面の辺縁から２ｍｍ以上根尖側に位置していた。

結論：インプラント埋入後の抜歯窩において治癒の初期段階に確立された骨とインプラントの接触は、頬側骨壁の吸収が続くにつれて一部失われた。

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