Several longitudinal studies have reported high survival and success rates for dental implants (see reference 1 for review). Nevertheless, dental implant failures due to peri-implant infection have also been reported.2 Animal studies have shown that peri-implantitis is a condition characterized by soft tissue inflammation, bleeding on probing, and suppuration, presenting with clinical attachment loss and vertical bone loss.3-5 These animal investigations reported that a bacterial shift was correlated with clinical attachment loss and peri-implant bone loss.6,7 Tillmanns et al.8 evaluated the peri-implantitis around different implant surfaces and concluded that all implants were equally susceptible to peri-implant breakdown. Hanish et al.8 monitored the peri-implant tissue breakdown in hydroxyapatite (HA)-coated implants and reported 2.7 mm of attachment loss. The average measured to attachment loss in commercially pure titanium (CP Ti) was 3.7 mm.9 Although the increase of probing depth and clinical attachment loss often was associated with the detection of Porphyromonas gingivalis, Prevotella intermedia, Tannerella forsythensis, Fusobacterium nucleatum, and, less frequently, Actinobacillus actinomycetemcomitans, the relationship between different dental implant surfaces and bacterial biofilm in peri-implantitis development is unclear. The different surfaces could influence the bacterial adsorption.11 Physical and chemical factors can affect the attachment of biofilms to a hard surface. The rough-

Progression of Experimental Chronic Peri-Implantitis in Dogs: Clinical and Radiographic Evaluation

Marilia Compagnoni Martins,* Jamil Awad Shibli,† Ricardo Samih Georges Abi-Rached,* and Elcio Marcantonio Jr.*

Background: The aim of this study was to evaluate the progression of experimental peri-implantitis in dogs using implants with different surface coatings.

Methods: Thirty-six dental implants with four different surface coatings, commercially pure titanium (CP Ti), titanium plasma-sprayed (TPS), hydroxyapatite (HA), and acid-etched (AE), were placed in six mongrel dogs. Five months after implantation, peri-implantitis was induced by cotton ligatures to facilitate plaque accumulation for 60 days. After 60 days, the ligatures were removed and supragingival plaque control was initiated for 12 months. Probing depth (PD), clinical attachment level (CAL), vertical bone level (VBL), horizontal bone level (HBL), and mobility were obtained at baseline, and 20, 40, 60 (acute phase), and 425 days (chronic phase) after ligature removal.

Results: PD and CAL changed around all implant surfaces after ligature placement (P < 0.0001). However, the means of PD and CAL were not statistically significant among the different surfaces (P > 0.05). The range of CAL variation, calculated between baseline and 60 days (acute phase) and between 60 and 425 days (chronic phase), decreased (P < 0.05). Bone loss increased during the entire experiment (P < 0.0001). The HA surface showed the greatest bone loss measurement (5.06 ± 0.38 mm) and the TPS showed the smallest bone loss (4.27 ± 0.62 mm). However, statistical significance was not assessed for different coatings (P > 0.05).

Conclusions: The clinical data at the initial phase showed rapid and severe peri-implant tissue breakdown. However, removal of ligatures did not convert the acute destructive peri-implant phase to a non-aggressive lesion and the progression of peri-implantitis was observed at chronic phase. The experimental peri-implantitis in dogs may be a useful model to evaluate the progression of peri-implantitis. J Periodontol 2005;76:1367-1373.

KEY WORDS
Animal studies; dental implants/adverse effects; dental implants/complications; follow-up studies; peri-implant diseases/prevention and control.

* Department of Periodontology, Dental School of Araraquara, State University of São Paulo (UNESP), Araraquara, São Paulo, Brazil.
† Department of Periodontology, Dental Research Division, Guarulhos University, Guarulhos, São Paulo, Brazil.
ness of the surface can increase surface area and hence increase the bacterial colonization. Roughness also provides protection from shear forces and increases the difficulty of cleaning methods. Quirynen et al.\textsuperscript{12,13} have shown that supragingival plaque formation, after initial bacterial colonization, was faster on a rough surface. The initial colonization of an intraoral hard surface starts from surface irregularities such as cracks, grooves, or abrasion defects and subsequently spreads out from these areas as a relatively even monolayer of cells. The roughness of different dental implant surfaces can work like grooves for initial periodontal pathogen adhesion.

Therefore, the purpose of this investigation was to evaluate the clinical and radiographic changes in the experimental chronic peri-implantitis around different dental implant surfaces in dogs.

**MATERIALS AND METHODS**

**Animals**

The outline of the study is presented in Figure 1. Six adult, systemically healthy, male mongrel dogs, 2 years of age and an average weight of 18 kg, were used. Animal selection, management, and surgical protocol followed routines approved for this study by the Institutional Animal Care and Use Committee.

All surgical and clinical procedures were performed under general anesthesia accomplished by 0.05 mg/kg of subcutaneous preanesthesia sedation (atropine sulfate) and intravenous injection of chlorpromazine and thiopental. Oral prophylaxis was performed within 2 weeks before teeth extraction. After that, all mandibular premolars were extracted, creating an edentulous ridge. Both the mandibular quadrants and the alveoli were allowed to heal for a period of 3 months. The upper premolars were also extracted to avoid occlusion trauma interference. During the healing period, bacterial biofilm control was instituted by means of scrubbing 0.12% chlorhexidine daily and scaling and root planing once a month until the placement of cotton ligatures.

**Implant Surfaces**

Thirty-six dental implants representing four different surfaces were inserted in canine jaws so that each dental implant surface was represented at least once in each animal: nine commercially pure titanium implants (cpTi),\textsuperscript{‡} nine titanium plasma-sprayed (TPS),\textsuperscript{§} nine hydroxyapatite (HA),\textsuperscript{∥} and nine hybrid surfaces (machined titanium in the first three threads and acid-etched [AE] in the other threads).\textsuperscript{¶} All implants were 10 mm in length and 3.75 mm in diameter (except TPS, which had a 4.1 mm diameter).

**Implant Surgery and Experimental Chronic Peri-Implantitis**

The dental implants were placed after a full-thickness flap under aseptic surgical conditions as previously described.\textsuperscript{10,14-16} In brief, all implants were randomly placed at bone level and flaps were sutured with single interrupted sutures, submerging all dental implants. Three months after dental implant insertion, healing abutment connections were installed. After 45 days of a plaque control program and the healing of the soft tissue, cotton floss ligatures were placed around the dental implants. The ligatures were checked twice a week, tying further ligatures at 20-day intervals for a period of 60 days. After 60 days, the ligatures were removed. A 12-month experimental chronic peri-implantitis was initiated with a supragingival plaque control by means of scrubbing daily with 0.12% chlorhexidine and scaling the abutment surface once a month.

**Clinical Evaluation**

Clinical parameters were recorded at baseline and 20, 40, 60, and 425 days after ligature tissue breakdown. A single precalibrated examiner carried out the clinical exams. The probing depth (PD) and clinical attachment level (CAL) were registered using a force-controlled calibrated periodontal probe\textsuperscript{#} with a constant probing force of 0.20 N and a probe-tip diameter of 0.4 mm. Data were recorded at disto-buccal, mid-buccal, mesio-buccal, mesio-lingual, mid-lingual, and disto-lingual aspects of each dental implant. PD and the distance between the gingival margin (GM) and the fixed point in the abutment surface (FP) were recorded. CAL was then calculated according the formula: PD + (GM-FP). All measurements were performed in the same position and same place with the

---

\textsuperscript{‡} 3i Implant Innovations, Inc., Palm Beach Gardens, FL.

\textsuperscript{§} Esthetic plus ITI Dental Implant System, Straumann AG, Waldenburg, Switzerland.

\textsuperscript{∥} Calcitek, Centerpulse Dental, Carlsbad, CA.

\textsuperscript{¶} Osseotite, 3i Implant Innovations, Inc.

\textsuperscript{#} Florida Probe, Computerized Probe Inc., Gainesville, FL.
aid of a dot marked in the abutment at baseline.

**Mobility**
Implant mobility (IM) was evaluated with an electronic instrument** by means of the scores. The implants were tapped with the instrument rod perpendicular to the longitudinal axis of the implants. The handlepiece of the instrument was held parallel to the floor at a distance of about 2.0 mm from the abutment surface. The spot chosen for tapping was at the buccal aspect of the abutment, and the spot was marked with a dot so the measurement was always performed at the same place. The instrument was calibrated before every measurement of IM and all measurements were performed by the same investigator. The IM mean variations were assessed at intra-surfaces to avoid variation among the different implant systems used in this study.

**Radiographic Analysis**
Standardized periapical radiographs were taken with a digital image system†† in order to measure the relative vertical peri-implant bone loss (VBL) and horizontal bone loss (HBL). A film holder system was affixed to a silicone bite block made of polyvinyl siloxane putty impression material used to standardize the placement of the sensor in relationship to the implants and the x-ray source. Images were obtained at baseline and 60 and 425 days after ligature placement.

A dental x-ray machine equipped with a 35 cm cone was used to expose the periapical intraoral sensor. Exposure parameters were 70 kilovolt (peak), 15 mA, and 1/4 second at a focus-to-sensor distance of 37 cm. The linear distance between the fixed point in abutment and the first visible bone-to-implant contact was determined mesially and distally using digital images of the implant to determine VBL. The HBL was measured between a fixed point in the implant shoulder and the crestal bone margins in the horizontal aspect. The mesial and distal values were averaged to obtain a mean implant value for both radiographic recordings. Two examiners made all measurements independently. If there was a discrepancy of 0.5 mm or less, the mean value of the two measurements was used. In situations with greater discrepancies, the images were analyzed again and discussed until a consensus was reached.

### Statistical Analysis
The clinical and radiographical data were compared by mean paired *t* test (2-tailed). All tests were stratified according to dog (unit of analysis, *N* = 6). Level of significance was set at 0.05.

### RESULTS
None of the 36 implants were lost during the acute phase of experimental peri-implantitis (initial 60 days). At 425 days, 17 dental implants were lost due to bone loss and mobility. The distribution of implants after chronic peri-implantitis phase was: four dogs retained at least one osseointegrated cpTi implant, five dogs presented six implants with a TPS surface, three dogs each retained an HA-coated surface implant, and five dogs each retained an AE implant.

### Probing Depth
The means of buccal and approximal PD are presented in Tables 1 and 2, respectively. After ligature placement, the means of both buccal and proximal sites increased statistically for all surfaces (*P* < 0.05).

---

**Table 1.**
Mean ± SD of Probing Depth at Buccal Sites (mm)

<table>
<thead>
<tr>
<th>Surface</th>
<th>Baseline</th>
<th>20 Days</th>
<th>40 Days</th>
<th>60 Days</th>
<th>425 Days</th>
<th>Change: Baseline to 425 Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>cpTi</td>
<td>1.47 ± 0.80</td>
<td>3.43 ± 0.80*</td>
<td>4.84 ± 1.37*</td>
<td>4.85 ± 0.82*</td>
<td>3.80 ± 1.30*</td>
<td>2.32 ± 0.63*</td>
</tr>
<tr>
<td>TPS</td>
<td>1.33 ± 0.30</td>
<td>3.14 ± 0.61*</td>
<td>3.42 ± 0.61*</td>
<td>4.58 ± 0.86*</td>
<td>4.40 ± 1.14*</td>
<td>3.06 ± 0.48*</td>
</tr>
<tr>
<td>HA</td>
<td>1.41 ± 0.37</td>
<td>3.62 ± 0.48*</td>
<td>4.70 ± 0.39*</td>
<td>5.16 ± 0.37*</td>
<td>5.66 ± 0.76*</td>
<td>4.25 ± 0.36*</td>
</tr>
<tr>
<td>Acid</td>
<td>1.25 ± 0.41</td>
<td>3.87 ± 0.62*</td>
<td>4.99 ± 0.55*</td>
<td>5.45 ± 0.67*</td>
<td>5.75 ± 1.50*</td>
<td>4.50 ± 0.63*</td>
</tr>
</tbody>
</table>

* Statistically different from baseline, *P* < 0.05.

**Table 2.**
Mean ± SD of Probing Depth at Proximal Sites (mm)

<table>
<thead>
<tr>
<th>Surface</th>
<th>Baseline</th>
<th>20 Days</th>
<th>40 Days</th>
<th>60 Days</th>
<th>425 Days</th>
<th>Change: Baseline to 425 Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>cpTi</td>
<td>2.18 ± 0.87</td>
<td>4.53 ± 0.86*</td>
<td>5.32 ± 0.98*</td>
<td>5.87 ± 0.97*</td>
<td>4.30 ± 1.03*</td>
<td>2.11 ± 0.57*</td>
</tr>
<tr>
<td>TPS</td>
<td>2.03 ± 0.68</td>
<td>3.58 ± 0.94*</td>
<td>3.95 ± 0.61*</td>
<td>5.01 ± 0.47*</td>
<td>5.06 ± 2.70*</td>
<td>3.03 ± 1.13*</td>
</tr>
<tr>
<td>HA</td>
<td>2.27 ± 0.83</td>
<td>4.77 ± 0.98*</td>
<td>5.25 ± 0.57*</td>
<td>5.66 ± 0.20*</td>
<td>5.66 ± 0.57*</td>
<td>3.39 ± 0.54*</td>
</tr>
<tr>
<td>Acid</td>
<td>2.27 ± 0.97</td>
<td>4.77 ± 0.77*</td>
<td>5.56 ± 0.32*</td>
<td>5.68 ± 0.79*</td>
<td>6.25 ± 0.64*</td>
<td>3.98 ± 0.55*</td>
</tr>
</tbody>
</table>

* Statistically different from baseline, *P* < 0.05.
Clinical Attachment Level
Clinical attachment loss at buccal and approximal sites (Tables 3 and 4) changed around all dental implant surfaces, not only after ligature-induced tissue breakdown, but also after ligature removal (or chronic phase) \((P < 0.0001)\). However, the means of buccal and proximal CAL were not statistically significant among the different implant surfaces \((P > 0.05)\).

When the range of CAL progression was calculated between baseline and 60 days (acute phase) and between 60 and 425 days (chronic phase), a decrease in the means of CAL was observed for both buccal and proximal sites (Figs. 2A and 2B).

Radiographic Bone Loss
At baseline, no implant surface exhibited peri-implant radiolucencies. The means of VBL and HBL for all dental implant surfaces are presented in Tables 5 and 6, respectively. The HA-coated surface showed the greatest bone loss measurement \((5.06 \pm 0.38 \text{ mm})\) and the TPS surface showed the smallest bone loss measurement \((4.27 \pm 0.62 \text{ mm})\). However, there was no statistical significance for the different surfaces \((P > 0.05)\). The VBL increased statistically in both the acute and chronic phases \((P < 0.0001)\).

The progression of vertical bone loss between acute and chronic phase decreased significantly for all implant surfaces (Fig. 2C). The HBL increased during the first 60 days relative to ligature placement. At 425 days the means of HBL decreased for all surfaces; however, a statistical significance was observed over time \((P < 0.05)\). In addition, the different dental implant surface was not found to be significant \((P > 0.05)\).

Mobility Assessment
The abutments were checked before each evaluation in order to avoid false results with regard to mobility. None of the implants exhibited mobility on manual examination. Table 7 shows the time course of IM scores. After

Table 3.
Mean ± SD of Clinical Attachment Level at Buccal Sites (mm)

<table>
<thead>
<tr>
<th>Surface</th>
<th>Baseline</th>
<th>20 Days</th>
<th>40 Days</th>
<th>60 Days</th>
<th>425 Days</th>
<th>Change: Baseline to 425 Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>cpTi</td>
<td>8.18 ± 0.74</td>
<td>11.29 ± 0.80*</td>
<td>12.18 ± 0.93*</td>
<td>12.75 ± 0.82*</td>
<td>14.90 ± 1.04*</td>
<td>6.71 ± 0.55</td>
</tr>
<tr>
<td>TPS</td>
<td>7.68 ± 0.95</td>
<td>9.82 ± 0.70*</td>
<td>10.67 ± 0.98*</td>
<td>11.93 ± 0.77*</td>
<td>14.75 ± 1.04*</td>
<td>7.07 ± 0.63</td>
</tr>
<tr>
<td>HA</td>
<td>7.81 ± 0.62</td>
<td>10.16 ± 1.06*</td>
<td>11.14 ± 0.92*</td>
<td>11.95 ± 0.95*</td>
<td>15.06 ± 0.11*</td>
<td>7.25 ± 0.37</td>
</tr>
<tr>
<td>Acid</td>
<td>8.08 ± 0.68</td>
<td>10.95 ± 0.52*</td>
<td>12.16 ± 0.73*</td>
<td>13.04 ± 0.91*</td>
<td>15.50 ± 0.38*</td>
<td>7.41 ± 0.38</td>
</tr>
</tbody>
</table>

* Statistically different from baseline, \(P < 0.0001\).

Table 4.
Mean ± SD of Clinical Attachment Level at Proximal Sites (mm)

<table>
<thead>
<tr>
<th>Surface</th>
<th>Baseline</th>
<th>20 Days</th>
<th>40 Days</th>
<th>60 Days</th>
<th>425 Days</th>
<th>Change: Baseline to 425 Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>cpTi</td>
<td>8.37 ± 0.54</td>
<td>11.35 ± 1.10*</td>
<td>11.97 ± 0.93*</td>
<td>13.25 ± 1.19*</td>
<td>14.30 ± 1.43*</td>
<td>5.92 ± 0.63</td>
</tr>
<tr>
<td>TPS</td>
<td>7.77 ± 0.88</td>
<td>9.66 ± 0.68*</td>
<td>10.18 ± 0.40*</td>
<td>11.57 ± 0.42*</td>
<td>13.75 ± 1.44*</td>
<td>5.97 ± 0.72</td>
</tr>
<tr>
<td>HA</td>
<td>7.64 ± 0.61</td>
<td>10.48 ± 0.68*</td>
<td>11.81 ± 0.39*</td>
<td>12.55 ± 0.45*</td>
<td>13.86 ± 1.02*</td>
<td>6.22 ± 0.53</td>
</tr>
<tr>
<td>Acid</td>
<td>8.39 ± 1.00</td>
<td>11.42 ± 0.63*</td>
<td>11.70 ± 0.62*</td>
<td>12.66 ± 0.54*</td>
<td>14.27 ± 0.53*</td>
<td>5.87 ± 0.55</td>
</tr>
</tbody>
</table>

* Statistically different from baseline, \(P < 0.0001\).

**Figure 2.**
Mean and standard deviation of CAL variation at buccal (A) and proximal (B) sites and VBL (C) between the acute and chronic phase. All measurements decrease at the chronic phase \((P < 0.05)\).
ligature-induced tissue breakdown, these scores increase over time ($P<0.05$), although differences among the surfaces were not observed ($P>0.05$).

**DISCUSSION**

The aim of this study was to evaluate the chronic peri-implant breakdown around different implant surfaces in a canine model. Several studies evaluated the experimental peri-implantitis around machine, TPS, and HA-coated surfaces using ligatures for plaque accumulation.\(^3\)\(^-\)\(^10\) In addition, placement of cotton\(^6\)\(^-\)\(^8\) or silk ligatures\(^5\)\(^,\)\(^17\)\(^,\)\(^18\) could induce a foreign body reaction different from peri-implant diseases. The trauma due to ligature placement has been implicated as a cause of periodontal breakdown in experimental periodontitis.\(^19\)\(^-\)\(^21\)

In this investigation, tying further ligatures at 20-day intervals for a period of 60 days accelerated the loss of peri-implant apparatus. The peri-implant bone loss was followed by a bacterial shift 60 days following ligature placement (acute phase).\(^10\) After that, the ligatures were removed and a supragingival plaque control was performed for 12 months (chronic phase). However, the removal of ligatures did not convert the acute destructive peri-implant phase in a non-aggressive lesion as previously reported.\(^8\)\(^,\)\(^22\)\(^,\)\(^23\) We can speculate that the ligature removal at 20-day intervals for 60 days may perpetuate the peri-implant lesion.

More than 66% of HA-coated implants were lost after chronic peri-implantitis. HA resorption is most likely caused by low pH\(^24\)\(^,\)\(^25\) and phagocytosis mediated by osteoclast-like cells, monocytes, and fibroblasts.\(^26\) Gineste et al.\(^28\) reported a resorption rate of more than 50% of HA coatings in dogs after a 1-year implantation period. We can speculate that the HA resorption must be higher with the presence of periodontal pathogens.\(^29\) However, the power of our results was limited due to HA-coated implants after the acute phase.

The higher mean of vertical bone loss, in conjunction with the increase in mobility (micromotion) observed for the HA surface in this study, could be additional factors for HA dissolution.\(^30\)

The course of PD at the acute phase was characterized by a constant increase in both buccal and proximal sites. However, at the chronic phase, different characteristics were observed among the surfaces. Overall, the cpTi surface showed a decrease of probing depth followed by TPS at buccal sites. HA and AE

<table>
<thead>
<tr>
<th>Surface</th>
<th>Baseline</th>
<th>20 Days</th>
<th>40 Days</th>
<th>60 Days</th>
<th>425 Days</th>
<th>Change: Baseline to 425 Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>cpTi</td>
<td>2.32 ± 0.53</td>
<td>4.12 ± 0.72*</td>
<td>5.20 ± 0.71*</td>
<td>6.32 ± 0.33*</td>
<td>6.92 ± 0.22*</td>
<td>4.60 ± 0.25*</td>
</tr>
<tr>
<td>TPS</td>
<td>2.50 ± 0.61</td>
<td>3.85 ± 0.95*</td>
<td>4.61 ± 0.90*</td>
<td>6.00 ± 0.70*</td>
<td>6.77 ± 1.38*</td>
<td>4.27 ± 0.62*</td>
</tr>
<tr>
<td>HA</td>
<td>2.01 ± 0.46</td>
<td>3.62 ± 0.29*</td>
<td>4.65 ± 0.84*</td>
<td>6.22 ± 0.50*</td>
<td>7.07 ± 0.72*</td>
<td>5.06 ± 0.38*</td>
</tr>
<tr>
<td>Acid</td>
<td>2.36 ± 0.54</td>
<td>3.64 ± 0.17*</td>
<td>5.19 ± 0.51*</td>
<td>6.06 ± 0.27*</td>
<td>6.80 ± 0.80*</td>
<td>4.44 ± 0.42*</td>
</tr>
</tbody>
</table>

* Statistically different from baseline, $P<0.0001$.

**Table 5.**

**Mean ± SD of Horizontal Bone Loss (mm)**

<table>
<thead>
<tr>
<th>Surface</th>
<th>Baseline</th>
<th>20 Days</th>
<th>40 Days</th>
<th>60 Days</th>
<th>425 Days</th>
<th>Change: Baseline to 425 Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>cpTi</td>
<td>0.66 ± 0.41</td>
<td>2.03 ± 0.77*</td>
<td>2.86 ± 0.64*</td>
<td>3.31 ± 0.44*</td>
<td>2.52 ± 1.75*</td>
<td>1.85 ± 0.73</td>
</tr>
<tr>
<td>TPS</td>
<td>0.68 ± 0.34</td>
<td>1.60 ± 0.53*</td>
<td>2.98 ± 0.84*</td>
<td>3.53 ± 0.58*</td>
<td>2.47 ± 1.82*</td>
<td>1.79 ± 0.74</td>
</tr>
<tr>
<td>HA</td>
<td>0.63 ± 0.36</td>
<td>2.18 ± 0.68*</td>
<td>3.20 ± 0.61*</td>
<td>3.90 ± 0.27*</td>
<td>1.63 ± 2.82*</td>
<td>1.00 ± 1.09</td>
</tr>
<tr>
<td>Acid</td>
<td>0.57 ± 0.37</td>
<td>1.61 ± 0.69*</td>
<td>2.76 ± 0.68*</td>
<td>3.85 ± 1.00*</td>
<td>3.12 ± 1.00*</td>
<td>2.55 ± 0.43</td>
</tr>
</tbody>
</table>

* Statistically different from baseline, $P<0.05$.

**Table 6.**

**Mean ± SD of Vertical Bone Loss (mm)**

<table>
<thead>
<tr>
<th>Surface</th>
<th>Baseline</th>
<th>20 Days</th>
<th>40 Days</th>
<th>60 Days</th>
<th>425 Days</th>
<th>IM Variation</th>
</tr>
</thead>
<tbody>
<tr>
<td>cpTi</td>
<td>−1.50 ± 2.34</td>
<td>−0.50 ± 1.22*</td>
<td>1.50 ± 3.39*</td>
<td>2.33 ± 2.25*</td>
<td>5.80 ± 1.92*</td>
<td>7.30 ± 1.31</td>
</tr>
<tr>
<td>TPS</td>
<td>−2.16 ± 2.48</td>
<td>−1.66 ± 2.25*</td>
<td>−2.16 ± 2.22*</td>
<td>−0.50 ± 2.50*</td>
<td>4.00 ± 1.41*</td>
<td>6.16 ± 1.20</td>
</tr>
<tr>
<td>HA</td>
<td>−2.66 ± 2.25</td>
<td>−1.16 ± 1.83*</td>
<td>−0.50 ± 1.64*</td>
<td>1.66 ± 2.06*</td>
<td>6.00 ± 2.64*</td>
<td>8.66 ± 1.67</td>
</tr>
<tr>
<td>Acid</td>
<td>−1.66 ± 2.65</td>
<td>−1.00 ± 1.78*</td>
<td>1.00 ± 1.78*</td>
<td>2.33 ± 1.63*</td>
<td>6.80 ± 2.48*</td>
<td>8.46 ± 1.56</td>
</tr>
</tbody>
</table>

* Statistically different from baseline, $P<0.05$. 

**Table 7.**

**Mean ± SD of Implant Mobility**
surfaces showed an increase of probing depth, but not in the same proportion that was observed at the acute phase. After conducting a detailed analysis of each surface, many observations can be made. The CAL was always higher at buccal sites than proximal sites. These observations were similar to results achieved by Schou et al.31 We observed that increase of probing depth contributed to initial attachment loss, while peri-implant mucosal recession was responsible for continued attachment loss, in agreement with Lang et al.32 and Tillmanns et al.3 Although our study design does not permit a direct conclusion, the different marginal connective tissue fiber configurations associated with bone availability may explain the higher range of proximal CAL. Previous studies9,31 have demonstrated many features in common for initial peri-implant/periodontal breakdown. In addition, Schou et al.31 reported that the differences between teeth and implants may not only be due to different configurations of tissues but also to the necessity of ligature removal.

Bone loss increased over time, but at the chronic phase the proportion of VBL decreased statistically compared to the initial phase. The vertical bone loss was initially lowest at the HA surface at baseline and highest at the end of the experiment. In contrast to VBL, the horizontal bone loss decreased over time and the bone defect became horizontal instead of the characteristic saucerization. In a human study, Tarnow et al.33 showed that there was a lateral component of bone loss once the biological width formed. In addition, the parallel collagen fiber orientation observed around dental implants can influence this bone resorption mechanism in the horizontal aspect.

Implant surface characteristics may influence the bone response during the healing and long-term period.34 The TPS showed the lowest range of VBL over time, followed by cpTi and AE. However, a statistical difference was not observed. The radiographic data reflected the association of factors such as bone remodeling on the coronal aspect, bone loss due to ligature trauma, and bone loss induced by bacterial products. The cpTi presented the lowest VBL progression at the chronic phase. The stylus profilometry of TPS and HA surface (undercuts) and acid surface (micropits) could have retained more bacterial biofilm and consequently more periodontal pathogens, suggesting that cpTi surface may function better during maintenance after peri-implantitis infection. However, we cannot understand clearly the features observed in the acid surface. The acid surface used in this study is hybrid, with a cpTi surface in the first three screws and acid treatment in the remaining screws. We could speculate that after the acid treatment, the oxide formation differs from that shown in cpTi surface, altering the surface affinity for lipopolysaccharide.35

The IM variation was lowest for the TPS surface; however, mobility as assessed by electronic device did not show a statistical difference among the surfaces. The microstructure and greater diameter of implant used in this study (4.1 mm for TPS surface and 3.75 mm for the other three surfaces) can explain and validate these data.

In conclusion, the clinical data observed at the initial phase showed a rapid and severe peri-implant tissue breakdown. After ligature removal, progressive peri-implantitis occurred and the HA-coated surface appeared to be more susceptible to tissue breakdown. However, these results should be considered with caution due to the sample size evaluated; further investigations must be conducted. Finally, this study indicates that experimental chronic peri-implantitis in dogs may be a useful model to evaluate the etiology, progression, and treatment of peri-implant diseases.

ACKNOWLEDGMENTS

This study was supported by grants 98/10100-0, 99/03026-1, and 00/02433-1 from FAPESP (Fundação de Amparo a Pesquisa do Estado de São Paulo, Brazil). The authors thank Drs. Carlos Nassar, Patricia Nassar, Rodrigo Rego, Solange A. Vergani, and Susana d’Avila, Araraquara Dental School, State University of Sao Paulo (UNESP), Araraquara, SP, Brazil, for invaluable assistance in surgical and clinical procedures.

REFERENCES


Correspondence: Prof. Elcio Marcantonio Jr., Department of Periodontology, Dental School of Araraquara, State University of São Paulo (UNESP), R. Huamita 1680. Araraquara-SP, Brazil. Fax: 55-16-3301-6364; e-mail: elciojr@foar.unesp.br.

Accepted for publication January 13, 2005.