

Evaluation of Three Different Dental Implants in Ligature-Induced Peri-implantitis in the Beagle Dog. Part I. Clinical Evaluation

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The purpose of this study was to clinically evaluate experimental peri-implant breakdown. Hydroxyapatite-coated, titanium plasma-sprayed, and machined titanium-alloy surfaces were investigated. Eighty-four implants were placed in 14 beagle dogs. Pocket probing depths and clinical attachment level and mobility measurements were made. Dogs were sacrificed at 3 and 6 months. All experimental implants showed a significant loss in clinical attachment level ($P < .05$). Increased pocket probing depths for experimental implants occurred during the first 2 months, after which a plateau was reached. At the 3- and 6-month evaluation, pocket probing depths at experimental implants were significantly increased ($P < .05$). No differences among the three implant types were noted for clinical attachment levels and pocket probing depths. In general, greater mobility was found with the titanium-alloy implants than with hydroxyapatite-coated and titanium plasma-sprayed implants ($P < .025$). In addition, mobility measurements were significantly greater for experimental titanium-alloy implants during the first 3 months ($P < .05$). Clinical attachment level measurements were most sensitive to peri-implant status. All implants were equally susceptible to ligature-induced peri-implant breakdown. Consequently, meticulous oral hygiene and regular maintenance care are prerequisites for successful implantology.

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Key words: beagle dog, endosseous, hydroxyapatite, implant, machined titanium alloy, peri-implantitis, surface, titanium plasma sprayed

Since their introduction, the design of endosseous implants has evolved to include a wide variety of geometrics, substrate materials, and surface coatings. The predominant commercially available implant designs can be broadly categorized as follows: (1) uncoated, machined screw forms; (2) screw forms coated with hydroxyapatite or titanium; and (3) cylinder forms coated with hydroxyapatite or titanium. The published scientific evidence indicates that each can be used with predictable long-term success.

Reports of the effect of peri-implant inflammation on various implant types have caused considerable controversy about the influence of surface coatings on the loss of supporting bone around diseased implants. Furthermore, there has been some controversy in recent years regarding how to clinically describe peri-implant anatomy and pathology. For teeth, pocket probing depth (PPD) and clinical attachment level (CAL) have been accepted as sensitive parameters for clinically detecting periodontal status. Because of differences in probe-tip location, underestimation often occurs in healthy teeth, while overestimation is common at diseased sites exhibiting attachment loss.¹⁻⁵ In contrast, findings from probing at healthy implants and at peri-implant tissues exhibiting gingivitis usually are similar.⁶ This may be in part the result of a hemidesmosomal epithelial attachment found adjacent to machined titanium

surfaces.⁷⁻⁹ However, differences have been noted in cases of advanced peri-implant lesions, since a scarlike connective tissue contact forms on these surfaces.¹⁰⁻¹³

Mobility (MOB) is frequently used as a parameter to assess major attachment changes in teeth.¹⁴ Since osseointegrated endosseous implants exhibit a “functional ankylosis,”¹⁵ a more sensitive methodology must be applied to detect minor changes in implant integration over time.¹⁶ The dog model has been used to evaluate experimental periodontitis¹⁷⁻¹⁹ and peri-implantitis.^{11-13,20} However, no direct comparison among different implant types placed in the same host has been reported. Since the severity of peri-implant breakdown is closely associated with the quality and quantity of the bacterial attack, as well as the capacity of the host to respond to the bacterial challenge, it seems appropriate to compare different endosseous implants in the same animal.

The purpose of the present study was to clinically monitor and compare the progression of ligature-induced peri-implantitis around different types of endosseous implants with three different surface characteristics in the canine mandible.

Materials and Methods

Extractions and Implant Surgery. Animals were under the supervision of a veterinary team (Laboratory Animal Resources Department, University of Texas Health Science Center) throughout the study and were treated according to humane guidelines. The mandibular second, third, and fourth premolars of 14 healthy beagle dogs were extracted bilaterally. Three months later, implants with three different surfaces—a titanium-aluminum-vanadium alloy (Ti-6Al-4V) cylinder with a Calcite hydroxyapatite (HA) coating (Sulzer Calcitek, Carlsbad, CA), a Ti-6Al-4V cylinder with a commercially pure titanium plasma-spray (TPS) coating (APS Materials, Dayton, OH), and a Ti-6Al-4V screw with a machined titanium-alloy (Ti-A) surface finish (Sulzer Calcitek)—were placed on each side of the mandible at the bone crest level according to a standard protocol (Fig 1). The HA, TPS, and Ti-A test implants were placed in random anterior-posterior distribution (Fig 2). All implants were 10 mm in length and 4 mm in diameter. Three months following initial placement, second-stage surgery was performed to expose implants and connect transmucosal abutments. Three days later, a custom-made metallic superstructure was cemented (Ticonium, CMP Industries, Albany, NY) to adjacent teeth so as to protect the implants from functional and parafunctional loading during the experimental period (Figs 3 and 4). Oral hygiene, consisting of tooth brushing (CET, VRx Products, Harbour City, CA) and interproximal brushing and scaling with a graphite scaler (SteriOss, Yorba Linda, CA), was performed three times per week. No antimicrobial additives were used to prevent carryover effect from the control to the experimental side. When necessary, animals were sedated at 2-week intervals to ensure complete plaque and calculus removal.

Experimental Phase. Four weeks following second-stage surgery, baseline readings of PPD, CAL (Florida Probe, Gainesville, FL), and MOB (Periotest, Siemens, Bensheim, Germany) were made (Fig 1). Florida Probe measurements were obtained with a constant probing force of 0.2 N at six sites per implant (Figs 5a and 5b). To evaluate intra-examiner reliability, all CAL and PPD measurements were performed twice, and mobility measurements were recorded three times during the same session (Fig 6). The experimental side of each dog was chosen at random.

Peri-implant inflammation was induced using braided-cotton retraction cord (GingiBraid, VanR Dental Products, Oxnard, CA) without astringents on the experimental side of the mandible. Ligatures were placed subgingivally around the neck of the implants. Plaque-control efforts were discontinued on

the experimental side (Fig 4) of the mandible but were continued around control implants (Fig 3). If necessary, ligatures were replaced at the plaque-control appointments. PPD, CAL, and MOB readings were repeated monthly (Fig 1).

Sacrifice. Three months after initial ligature placement, six dogs were sacrificed. The remaining eight animals were sacrificed at 6 months.

Statistics. When control and experimental values were compared at the same time points and a normal distribution was present, the paired *t* test was used (experimental versus control implants for CAL, PPD, and MOB). For comparisons over time, analysis of variance (ANOVA) and a post-hoc analysis using a general linear model with least-mean squares were used. Differences among the implant types were assessed by *F* approximation for Friedman test. When multiple comparisons were made, the *P* value was adjusted accordingly. Correlations were examined for parametric data (CAL, PPD, MOB) using a Pearson correlation analysis. Fourteen dogs were in the 3-month evaluation, whereas eight dogs were sacrificed for the 6-month data collection.

Results

One implant site, involving an HA implant, developed a granulomatous infection during the early-healing phase, and the affected implant was subsequently lost. Two additional implants, one TPS and one Ti-A, were lost at second-stage surgery because of failure of tissue integration. They were removed with cotton pliers and reverse torque. The 81 remaining implants were deemed osseointegrated and considered clinically successful. Thirty-nine control implants demonstrated complication-free tissue integration (Fig 3). All 42 experimental implants showed significant plaque accumulation and typical signs of a peri-implant lesion (Fig 4).

Repeatability. A total of 11,160 measurements with the Florida Probe were taken for CAL and PPD readings by one examiner. When repeatability of the two measurements was analyzed, it was noted that 99% of all CAL and PPD readings were within a range of ± 0.7 mm; 95% of these measurements were within a range of ± 0.5 mm. Twenty percent of all CAL measurements were identical for the two probings, whereas 19% of the PPD measurements were identical.

Clinical Attachment Level. The 3- and 6-month evaluation of CAL for all three types of implants showed minimal attachment loss after the first month on the control side. Experimental implants, however, were associated with a continuous decrease in CAL (Figs 7 and 8). Differences in CAL loss between control and experimental implants were statistically significant ($P < .05$) from 2 and 3 months onward for all implant types (Tables 1 and 2). Over time, all experimental implants exhibited significant reductions in CAL when compared to baseline readings beginning at 3 and 5 months ($P < .0024$ for 3 months, $P < .0013$ for 6 months). *F* approximation for Friedman test did not detect any differences among the three implant types ($P > .025$) at either termination time point.

Pocket Probing Depth. For all control implants, PPD readings were stable throughout the study. Experimental implants showed an increase during the early phase with a plateau formation developing at 2 months (Figs 9 and 10). Average PPD readings ranged from 2.39 mm to 4.00 mm (Tables 1 and 2). For the 3-month data, differences in PPD readings between control and experimental implants became statistically significant at 2 and 3 months ($P < .05$). For the 6-month evaluation, significant differences for HA implants were noted for all time points ($P < .05$), whereas TPS implants revealed significant changes from 2 months onward ($P < .05$). Ti-A implants exhibited significant PPD increases at 2, 5, and 6 months ($P < .05$). When PPD changes were compared to baseline values, significant differences were

noted for HA and TPS implants, from 2 to 3 months onward, for both time intervals ($P < .0024$ for 3 months, $P < .0013$ for 6 months). These significant changes were not apparent at Ti-A implants. No statistically significant differences among the three implant types were noted by F approximation for Friedman test at either 3 or 6 months ($P > .025$).

Mobility. None of the 81 test implants were deemed clinically mobile by conventional examination technique at any point during the study.¹⁴ Although all implants demonstrated increases in MOB, experimental implants experienced a higher level of mobility, as determined by Periotest values (PTV), in both the 3- and 6-month data (Figs 11 and 12). Means ranged from -3.88 PTV to 0.62 PTV (Tables 1 and 2). Significant MOB differences between control and experimental HA and TPS implants were only detected twice ($P < .05$). However, for each time point of the 3-month evaluation, experimental Ti-A implants showed significantly increased MOB values compared to control Ti-A implants ($P < .05$). Comparing implant types with each other, MOB measurements were greater for both control and experimental Ti-A implants ($P < .025$). Also, a statistically significant increase in implant mobility was noted at the Ti-A implants when compared to the other two implants tested ($P < .025$).

Correlations. Correlation analyses revealed a high correlation of CAL with PPD readings for HA and TPS at 3 and 6 months. At 3 months, correlations from $r = .67$ to $r = .82$ ($P < .05$) were computed. At the 6-month evaluation, correlations ranging from $r = .71$ to $r = .86$ ($P < .05$) were found. No correlation between these two measurements was noted for Ti-A implants, except for the 3-month data at experimental implants. No consistent relation of MOB to either CAL or PPD readings was detected.

Discussion

In the present study, subgingival placement of cotton ligatures around dental implants and cessation of oral hygiene to accelerate plaque formation resulted in the anticipated clinical attachment loss, as well as increases in implant mobility. This seemed to represent a localized lesion arising in response to bacterial plaque accumulation, in a manner similar to that encountered in advanced periodontal disease.²¹ As a result, significant deterioration occurred as assessed by clinical parameters. When the three different implant types were compared for both the control and experimental groups, no clinically relevant differences among HA, TPS, or Ti-A could be found either in the performance of the control implants or in the response to inflammation. These findings suggest a similar susceptibility and response of the evaluated implant types to induced peri-implantitis.

When a detailed analysis of the results for each test was conducted, many observations could be made. All experimental implants showed significant loss of CAL and increased PPD. These increases were similar to results achieved by Schou et al,²² but of decreased magnitude relative to previous reports.^{23,24} Increasing PPD contributed to the initial loss of CAL, while gingival recession was responsible for continued attachment loss as seen in other studies.^{6,25}

At healthy sites, PPDs around Ti-A implants in this study were comparable to those found around successfully maintained, two-part titanium implants in beagle dogs.^{13,26,27} Similar findings of $PPD \leq 3$ mm at 80% of sites examined have been reported by Buser et al²⁸ in one-part titanium implants. For HA and threaded Ti-A implants, comparable results have been published.²⁹

The significance and applicability of methods to clinically evaluate and record peri-implant inflammation have been discussed.^{30,31} At teeth, the most common method used to detect clinical attachment levels is probing depth assessment. However, because of differences in probe-tip location,

minor underestimation occurs in clinically healthy conditions, whereas a slight overestimation takes place at diseased sites exhibiting attachment loss.¹⁻⁵ In healthy peri-implant sites, the probe tip has been shown to be located in the most coronal portion of the connective tissue, using a moderate probing force of 0.2 N, in nonsubmerged implants.⁶ In comparison, an excessive probing force of 0.5 N led to a probe tip located almost at the crest of the bone in submerged implants.¹³ This is likely the result of the structure of a scarlike connective tissue contact that has been shown to form adjacent to machined titanium surfaces in submerged and nonsubmerged implants.^{10,12} For this reason, in the present study a Florida Probe with a constant probing force of 0.2 N was used for all CAL and PPD measurements. In addition, the Florida Probe has been shown to have high reproducibility and small standard deviations in conjunction with a stent around teeth.³²⁻³⁷ These findings were substantiated in the present study on implants.

Mobility was slightly greater for Ti-A implants, both at control and experimental sites. Ericsson et al²⁷ recorded comparable MOB values. Two factors may be responsible for these differences: (1) different mechanisms of integration of the implant surface in bone, ie, osseointegration for the Ti-A and TPS surface and biointegration for the HA surface; and (2) different available surface areas. Although an increase of surface area is evident with a screw design as compared to a machined-cylinder implant, the surface roughness of cylindrical TPS and HA implants results in substantially greater surface areas than the machined-screw design.

The high correlation of CAL with PPD readings for HA and TPS implants indicates that decreased CAL is accompanied by increased PPD, as previously described.²⁴ The lack of correlation between these two measurements for Ti-A implants may be explained by the difference in implant design. It seems possible that because of the flare of the neck of the Ti-A implants and their screw design, probing parallel to the long axis of the implants may have been impossible, or at least impaired, until a substantial amount of tissue had been lost. The probe tip may have been hindered by the first thread of these implants, resulting in probing error. This, in turn, would have skewed the CAL and PPD measurements toward reduced readings.

When the three implants were compared to each other, no clinically relevant differences among HA, TPS, and Ti-A implants were found for CAL, PPD, and MOB measurements. This finding contradicts previously published anecdotal reports, where HA implants were associated with substantially more complications and failures resulting from peri-implantitis.^{38,39}

Conclusion

When analyzed by clinical parameters, all experimental implants were equally susceptible to ligature-induced peri-implantitis. Clinical attachment level measurements were the most sensitive parameter relative to peri-implant status. Mobility readings were greater in control and experimental Ti-A implants when compared to HA and TPS implants. Consequently, meticulous oral hygiene and regular maintenance care are prerequisites for successful implant treatment.

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FIGURES

Figure 1

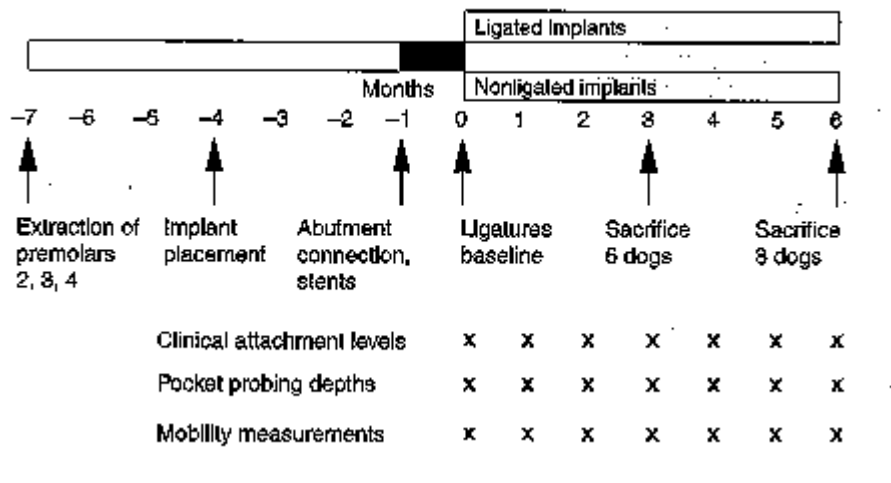


Fig. 1 Study design.

Figure 2



Fig. 2 Test implants with three different surfaces, shown left to right: titanium alloy (Ti-A), hydroxyapatite-coated (HA), and titanium plasma-sprayed (TPS).

Figure 3



Fig. 3 Control implants placed in canine mandible. Healthy peri-implant tissues can be seen around Ti-A, HA, and TPS implants. Note metal bar cemented on canine and first molar to protect implants from functional loading.

Figure 4



Fig. 4 Experimental implants placed on opposite side of canine mandible. Severe plaque accumulation/peri-implant inflammation is visible, with edema, erythema, and spontaneous bleeding around TPS, HA, and Ti-A implants. Note metal bar cemented on canine and first molar.

Figure 5a

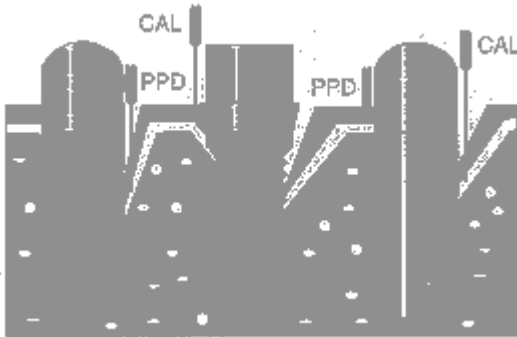


Fig. 5a Schematic of clinical attachment level (CAL) and pocket probing depth (PPD) measurements at different implant types. Note position of sleeve (reference point) of Florida Probe in relation to healing abutments.

Figure 5b

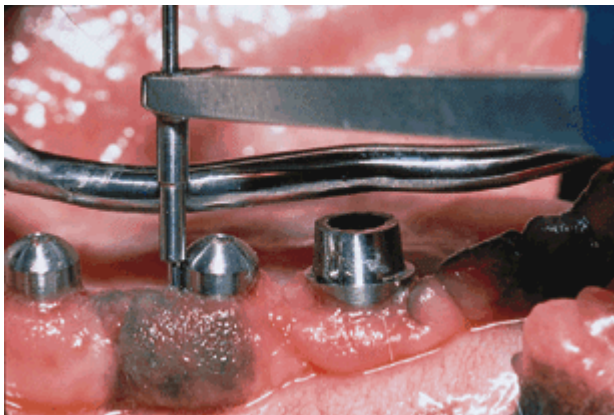


Fig. 5b Florida Probe in situ: clinical attachment level (CAL) measurement at TPS implant.

Figure 6



Fig. 6 Periotest measurement at tested implants while beagle dog is anesthetized.

Figure 7

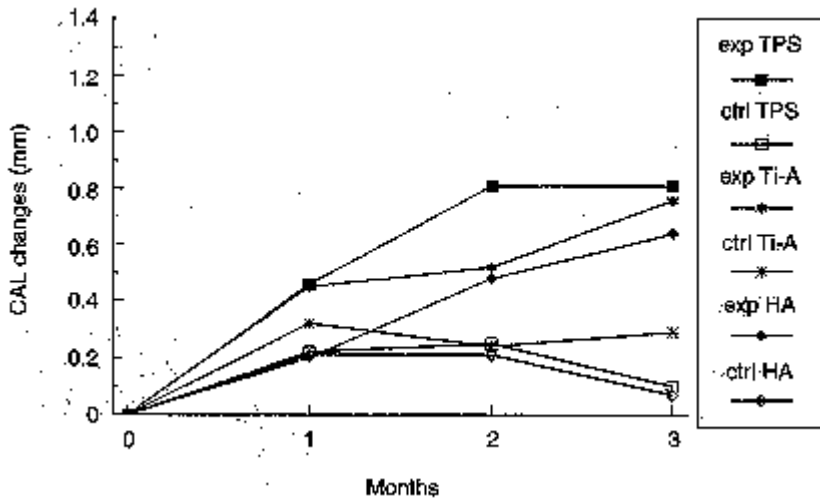


Fig. 7 Decrease in clinical attachment level (CAL) at 3 months (mean; n = 14). Consistent loss of CAL for all experimental implants. Note that after initial loss at 1 month, there is no further decrease in CAL for control implants.

Figure 8

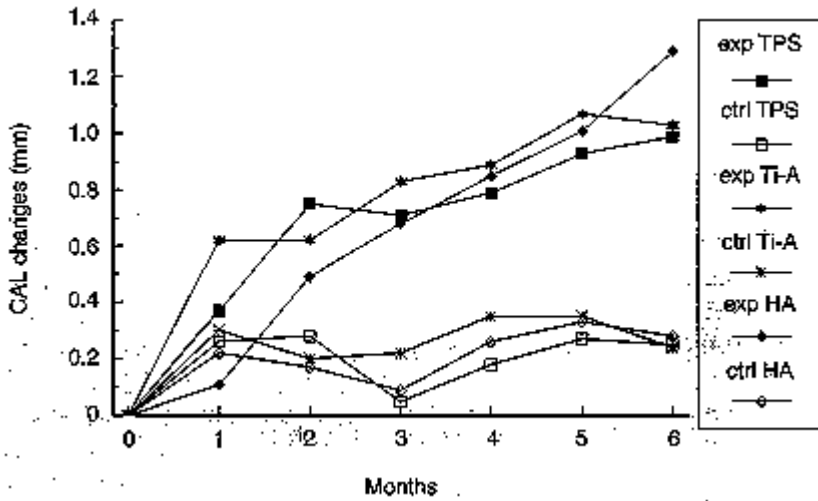


Fig. 8 Decrease in clinical attachment level (CAL) at 6 months (mean; n = 8). Continuous loss for experimental implants. Note plateau formation for CAL at control implants after initial loss at 1 month.

Figure 9

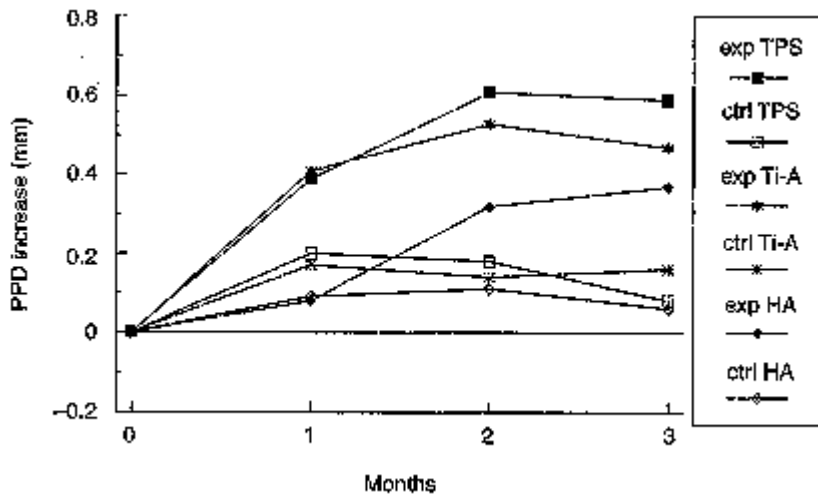


Fig. 9 Pocket probing depth (PPD) increase was apparent at experimental implants at 2 and 3 months (mean; n = 14).

Figure 10

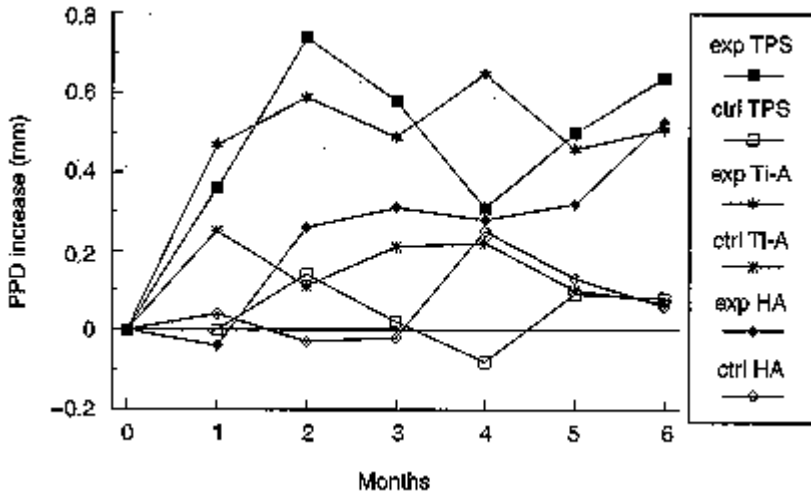


Fig. 10 Pocket probing depth (PPD) increase was evident at experimental implants for the first 2 months, with later plateau formation (mean; n = 8).

Figure 11

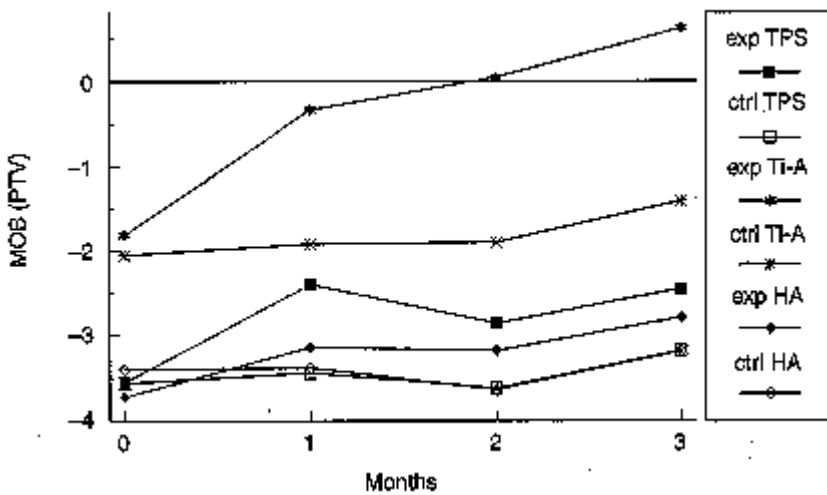


Fig. 11 Mobility (MOB) in creases were obvious only at experimental Ti-A implants (mean; n = 14). Note higher MOB for all Ti-A implants.

Figure 12

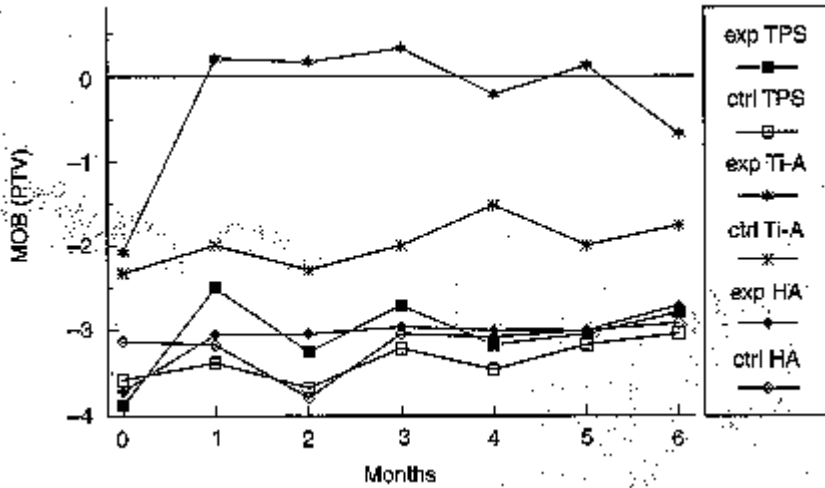


Fig. 12 Mobility (MOB) in creases were obvious only at experimental Ti-A implants (mean; n = 8). After initial changes, plateau is reached at 2 months. Note higher MOB for all Ti-A implants.

TABLES

Table 1

Table 1 Mean Values (\pm SD) for Clinical Measurements for Control and Experimental Groups (0 to 3 Months)

Month	CAL (mm)			PPD (mm)			MOB (PTV)		
	HA	TPS	Ti-A	HA	TPS	Ti-A	HA	TPS	Ti-A
Control (n = 14)									
Baseline				2.98 \pm 0.49	3.01 \pm 0.57	2.53 \pm 0.64	-3.40 \pm 0.97	-3.56 \pm 0.86	-2.05 \pm 1.24
1	0.21 \pm 0.19	0.22 \pm 0.27	0.32 \pm 0.38	3.07 \pm 0.52	3.21 \pm 0.74	2.70 \pm 0.43	-3.38 \pm 0.89	-3.44 \pm 0.74	-1.92 \pm 1.45
2	0.21 \pm 0.28	0.25 \pm 0.21	0.24 \pm 0.33	3.08 \pm 0.54	3.19 \pm 0.58	2.67 \pm 0.57	-3.64 \pm 1.03	-3.62 \pm 0.99	-1.90 \pm 1.48
3	0.07 \pm 0.29	0.10 \pm 0.34	0.29 \pm 0.29	3.03 \pm 0.51	3.09 \pm 0.58	2.70 \pm 0.50	-3.19 \pm 1.01	-3.18 \pm 0.89	-1.41 \pm 1.48
Experimental (n = 14)									
Baseline				3.28 \pm 0.45	3.02 \pm 0.43	2.51 \pm 0.34	-3.72 \pm 1.06	-3.55 \pm 1.05	-1.81 \pm 1.15
1	0.20 \pm 0.28	0.46 \pm 0.42	0.45 \pm 0.39	3.36 \pm 0.34	3.41 \pm 0.53	2.92 \pm 0.47	-3.14 \pm 0.78	-2.40 \pm 1.32*	-0.33 \pm 1.97*
2	0.48 \pm 0.30*	0.81 \pm 0.37*†	0.52 \pm 0.30*	3.60 \pm 0.64†	3.64 \pm 0.56*	3.03 \pm 0.34*	-3.18 \pm 1.17	-2.86 \pm 1.16	0.05 \pm 2.19*
3	0.64 \pm 0.37*†	0.81 \pm 0.33*†	0.76 \pm 0.46*†	3.65 \pm 0.56*†	3.61 \pm 0.56*†	2.98 \pm 0.35*	-2.79 \pm 1.17	-2.45 \pm 1.49	0.62 \pm 1.86*

*Difference between experimental and control groups ($P < .05$).
 †Difference versus baseline reading ($P < .0024$).

Table 2

Table 2 Mean Values (\pm SD) for Clinical Measurements for Control and Experimental Groups (0 to 6 Months)

Month	CAL (mm)			PPD (mm)			MOB (PTV)		
	HA	TPS	Ti-A	HA	TPS	Ti-A	HA	TPS	Ti-A
Control (n = 8)									
Baseline				2.94 \pm 0.49	2.97 \pm 0.69	2.44 \pm 0.54	-3.13 \pm 0.96	-3.58 \pm 0.97	-2.33 \pm 1.52
1	0.22 \pm 0.23	0.26 \pm 0.29	0.30 \pm 0.43	2.98 \pm 0.35	3.19 \pm 0.88	2.69 \pm 0.38	-3.27 \pm 0.85	-3.38 \pm 0.82	-2.00 \pm 1.39
2	0.17 \pm 0.33	0.28 \pm 0.16	0.20 \pm 0.41	2.92 \pm 0.47	3.12 \pm 0.71	2.55 \pm 0.37	-3.79 \pm 1.11	-3.67 \pm 0.98	-2.29 \pm 1.28
3	0.09 \pm 0.35	0.05 \pm 0.38	0.22 \pm 0.32	2.92 \pm 0.48	2.99 \pm 0.65	2.65 \pm 0.35	-3.04 \pm 1.18	-3.21 \pm 1.05	-2.00 \pm 1.40
4	0.26 \pm 0.29	0.18 \pm 0.28	0.35 \pm 0.31	3.19 \pm 0.42	2.89 \pm 0.60	2.66 \pm 0.42	-3.08 \pm 1.26	-3.46 \pm 1.25	-1.52 \pm 1.23
5	0.33 \pm 0.21	0.27 \pm 0.37	0.35 \pm 0.35	3.07 \pm 0.46	3.06 \pm 0.75	2.54 \pm 0.40	-3.00 \pm 1.08	-3.17 \pm 1.15	-2.00 \pm 1.78
6	0.28 \pm 0.30	0.25 \pm 0.37	0.24 \pm 0.28	3.01 \pm 0.43	3.06 \pm 0.77	2.52 \pm 0.31	-2.92 \pm 1.42	-3.04 \pm 1.31	-1.76 \pm 0.90
Experimental (n = 8)									
Baseline				3.47 \pm 0.35*	3.07 \pm 0.41	2.39 \pm 0.36	-3.71 \pm 1.37	-3.88 \pm 1.23	-2.08 \pm 1.19
1	0.11 \pm 0.32	0.37 \pm 0.52	0.62 \pm 0.41	3.44 \pm 0.28*	3.42 \pm 0.64	2.86 \pm 0.59	-3.05 \pm 0.95	-2.50 \pm 1.48	0.21 \pm 1.93*
2	0.49 \pm 0.32	0.75 \pm 0.38*	0.62 \pm 0.32*	3.73 \pm 0.73*†	3.80 \pm 0.63*†	2.98 \pm 0.38*	-3.04 \pm 1.20*	-3.25 \pm 0.66	0.17 \pm 2.42*
3	0.68 \pm 0.36*	0.71 \pm 0.34*†	0.83 \pm 0.38*†	3.78 \pm 0.60*†	3.64 \pm 0.56*†	2.88 \pm 0.35	-2.96 \pm 1.20	-2.71 \pm 1.60	0.33 \pm 1.35*
4	0.85 \pm 0.43*	0.79 \pm 0.25*†	0.89 \pm 0.33*†	3.76 \pm 0.56*	3.38 \pm 0.49*	3.03 \pm 0.67	-3.00 \pm 1.25	-3.17 \pm 1.11	-0.21 \pm 1.99
5	1.01 \pm 0.51*†	0.93 \pm 0.24*†	1.07 \pm 0.35*†	3.79 \pm 0.69*†	3.57 \pm 0.53*	2.84 \pm 0.42*	-3.00 \pm 1.17	-3.04 \pm 0.95	0.13 \pm 1.28*
6	1.29 \pm 0.59*†	0.99 \pm 0.28*†	1.03 \pm 0.45*†	4.00 \pm 0.77*†	3.70 \pm 0.60*†	2.90 \pm 0.46*	-2.71 \pm 1.40	-2.79 \pm 1.40	-0.67 \pm 1.08

*Difference between experimental and control groups ($P < .05$).
†Difference versus baseline reading ($P < .0013$).

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