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Mechanical non-surgical treatment of peri-implantitis: a double-blind randomized longitudinal clinical study. I: clinical results

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Abstract

Background: Peri-implantitis is a frequent finding in patients with dental implants. The present study compared two non-surgical mechanical debridement methods of peri-implantitis.

Material and Methods: Thirty-seven subjects (mean age 61.5; S.D ± 12.4), with one implant each, demonstrating peri-implantitis were randomized, and those treated either with titanium hand-instruments or with an ultrasonic device were enrolled. Data were obtained before treatment, and at 1, 3, and 6 months. Parametric and non-parametric statistics were used.

Results: Thirty-one subjects completed the study. The mean bone loss at implants in both groups was 1.5 mm (SD ± 1.2 mm). No group differences for plaque or gingival indices were found at any time point. Baseline and 6-month mean probing pocket depths (PPD) at implants were 5.1 and 4.9 mm ($p = 0.30$) in both groups. Plaque scores at treated implants decreased from 73% to 53% ($p < 0.01$). Bleeding scores also decreased ($p < 0.01$), with no group differences. No differences in the total bacterial counts were found over time. Higher total bacterial counts were found immediately after treatment ($p < 0.01$) and at 1 week for ultrasonic-treated implants ($p < 0.05$).

Conclusions: No group differences were found in the treatment outcomes. While plaque and bleeding scores improved, no effects on PPD were identified.

Key words: mechanical; non-surgical therapy; peri-implantitis; ultrasonic; ultrasonic

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During the most recent decades, implant dentistry has become an effective method to re-establish aesthetics and chewing function following tooth loss. Although in most cases dental implants, as a tooth replacement device, have a good prognosis, complications do occur. Biological complications are referred to as peri-implant mucositis or peri-implantitis (Albrektsson & Isidor 1994). Peri-implant infections have been associated with biofilm development (Costerton et al. 1999, Lamont & Jenkinson 2000). As a consequence, the elimination of the biofilm seems to be essential in the management and control of peri-implant infections. Therapies proposed for the management of peri-implant infections, however, appear to be largely based on the evidence available from the treatment of periodontitis. The screw-shaped designs of dental implants, combined with various degrees of surface modifications allowing for an enhanced osseointegration, may also enhance biofilm formation, and thereby increase the risk for inflammation. Most publications on treatment of peri-implant lesions in humans report individual cases treated by combined procedures, aimed at reducing the bacterial load within the peri-implant pocket

Conflict of interest and source of funding statement

None of the authors have a conflict of interests.
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(for a review, see Renvert et al. 2008a, b). Implant surface debridement constitutes the basic element for treatment of peri-implant mucositis and peri-implantitis.

There are few studies that have assessed the efficacy of non-surgical treatment of infections around dental implants. In cases of peri-implantitis, the use of mechanical debridement alone has been demonstrated to be ineffective whereas some positive effects have been demonstrated using a special ultrasonic treatment device (Karring et al. 2005). Mechanical debridement as well as mechanical debridement supplemented with chlorhexidine can be beneficial to patients with peri-implant mucositis (Porras et al. 2002). Data also suggest that a high recall frequency with mechanical debridement can reduce the extent of inflammation and reduce probing pocket depths (PPD) and bacterial loads (Strooker et al. 1998). In non-surgical treatment of peri-implantitis comparing debridement with plastic curettes+chlorhexidine with YRG laser treatment, a significantly greater reduction in bleeding on probing (BOP) was found following the use of the yttrium, aluminium and garnet laser treatment (Schwarz et al. 2005). In a recent review on non-surgical therapy of peri-implantitis (Renvert et al. 2008a, b), the authors concluded that mechanical non-surgical therapy could be effective in the treatment of peri-implant mucositis lesions whereas in peri-implantitis lesions non-surgical therapy was not found to be effective. The data available to support these conclusions were, however, scarce. Therefore, there is a need for randomized clinical trials to determine whether mechanical debridement of the implant surface in areas with peri-implantitis is efficacious.

The purpose of the present study was to compare two mechanical debridement methods with specially designed devices in the non-surgical treatment of peri-implantitis.

Material and Methods

The Ethics Committee of Lund University, Sweden, approved the study. Written consent was obtained from all subjects enrolled. The CONSORT guidelines for clinical trials were followed. Subjects were enrolled, if they presented with one dental implant with bone loss <2.5 mm identified on intra-oral radiographs and having a PPD $\geqslant 4$ mm with bleeding, and/or pus on probing using a 0.2 N probing force. Subjects may have had >1 implant but only one implant in each subject met the inclusion criteria and only these implants were studied.

The study was conducted between March 2007 and June 2008. The following criteria were used to exclude subjects from entering the study: (I) poorly controlled diabetes mellitus, (II) use of anti-inflammatory prescription medications, or antibiotics within the preceding 3 months or during the study, and (III) bone loss >2.5 mm in comparison with findings from radiographs taken immediately following placement of the implant supra-structure.

Before enrolment in the study, any periodontal lesions at remaining teeth had been treated. Subjects were randomly assigned to one of the two treatment regimens. The allocation was carried out using a computer software program (SPSS Inc.) for the randomization. The study examiner and the therapist were not jointly present with the study subject when performing their study tasks. Study subjects were instructed not to discuss the therapy with the study examiner. The study examiner was unaware of study treatment allocation and performed all clinical measurements, and collected samples for microbiological analysis. The clinician performing treatment had >7 years of clinical experience in the mechanical treatment of implants with a diagnosis of peri-implantitis.

Clinical measurements and procedures

At all study time points, the PPD and BOP measurements were performed using a plastic probe with a standardized probing force of 0.2 N (Hawe Click-Probe, Hawe Neos Dental, Switzerland). The following clinical assessments were performed at baseline, at month 1, at month 3 and at month 6: (I) presence/absence of hyperplasia, (II) full-mouth plaque score recorded as the presence of dental plaque along the gingival/mucosal margin following the use of disclosing dye and expressed as a percentage of examined sites within each subject (four sites per tooth and implant), (III) local plaque score defined as the presence of dental plaque along the mucosal margin at four sites of the treated implant, recorded after the use of a disclosing dye, (IV) PPD at the worst site of implant, (V) mean PPD based on scores from four sites per implant, (VI) presence /absence of BOP at the implant (four sites/implant) and (VII) bleeding appearing after PPD measurements of probing depth and expressed as a percentage of examined sites (four sites per tooth and the implant). The amount of bleeding at the implant site was graded as follows: (1) no bleeding, (2) point of bleeding, (3) line of bleeding, and (4) drop in bleeding.

After removal of supra-gingival plaque with sterile cotton pellets, microbiological samples were taken before initial clinical measurements and 30 min. after treatment, 1 week after treatment, 1, 3 and 6 months after treatment. Two sterile paper points (Dentsply Maillefer size 55, Ballaigues, Switzerland) were inserted submucosally until resistance was felt and left in place for 20 s. Then, the samples were placed in dry Eppendorf tubes (1.5 ml natural flat cap microcentrifuge tubes, Starlab, Ahrensburg, Germany). The samples were frozen at -80°C. Within 4 weeks, the samples were sent to the microbiology laboratory at the University of Bern, Switzerland. At the laboratory, the samples were analysed by the checkerboard DNA–DNA hybridization technique for the total bacterial load of 40 different bacteria. Details of the microbiological findings including assessments of the presence of 74 different species will be presented elsewhere. The total bacterial load among species assayed was only calculated among subjects who completed the study.

Treatment procedures

Implants in group 1 were treated with mechanical debridement using titanium curettes (Deppeler SA, Rolle, Switzerland) and were polished with rubber cups and polishing paste. Implants in group 2 were treated with mechanical debridement using an ultrasonic device (the Vector system®, Dürr Dental AG, Bietigheim-Bissingen, Germany) with a specially designed tip for the treatment of infections around implants (LM Instruments Oy, Parainen, Finland). All implants were polished with rubber cups and polishing paste. If needed, routine local anaesthesia was used. All subjects received oral hygiene instructions on an individual basis and at all study time points.

Statistical methods

Power calculation: In the absence of reliable data on changes in clinical mea-

sures with regard to non-surgical mechanical therapy, we assumed a mean difference of 0.6 mm between groups. This resulted in a sample size estimation of 18 individuals in each group.

The data were analysed using Kruskal–Wallis ANOVA and GLM ANOVA for dichotomous /non-parametric or parametric data as appropriate. The data were analysed by independent *t*-tests for continuous variables with a normal distribution (equal variance not assumed) and by the Mann–Whitney *U*-tests for non-parametric data using a statistical software package 16.0 for MAC (SPSS Inc., Chicago, Il, USA). Statistical difference was defined by a p value <0.05. Primary outcome measures were the changes in PPD and reduction in BOP and the secondary outcome variable was changes in the total bacterial load.

Results

A total of 37 subjects were enrolled. Two subjects in group 1 discontinued the study and four subjects in group 2 discontinued the study (Fig. 1). Thus, complete data were available for 17 subjects in group 1 and 14 in group 2. The mean age of the subjects in group 1 was 62.7 years (SD $\pm$ 12.1) and 60.3 (SD $\pm$ 12.9) in group 2. Seven subjects in each group were women and a total of five subjects (15.6%), three in group 1 and two in group 2, reported a smoking habit. None of the smokers changed their habit during the 6-month trial, or had changed their smoking habits within the preceding 6 months.

Clinical findings

Subjects who declined to return for the follow-up visits did not provide reasons as to why they did not return. No adverse events were reported by the subjects participating in the study. At baseline, the mean amount of bone loss around the implants including only subjects who completed the study was 1.5 mm (SD $\pm$ 1.2) in both groups. None of the implants demonstrated bone loss $\geqslant$2.5 mm. At the different time points, group characteristic clinical data are presented (Table 1). Probing depths at the worst site were 4 mm in five of the cases, 5 mm in 19 of the cases and 6 mm in seven of the cases. The average PPD at the four sites per implant varied between 2.8 and 5.5 mm. The distribution of implants was as follows: Nobel Biocare implants ($n = 24$) Astra implants ($n = 6$), and one implant that could not be identified. Nine of the subjects were edentulous. The average number of remaining teeth was 15.1 (SD $\pm$ 10.6). Among the 31 subjects, a total of 30 tooth-sites (0.02%) presented with a PPD$\geqslant$5 mm.

GLM univariate analysis of variance failed to demonstrate differences in PPDs from the worst site, mean PPD at four sites per implant. Further analysis by an

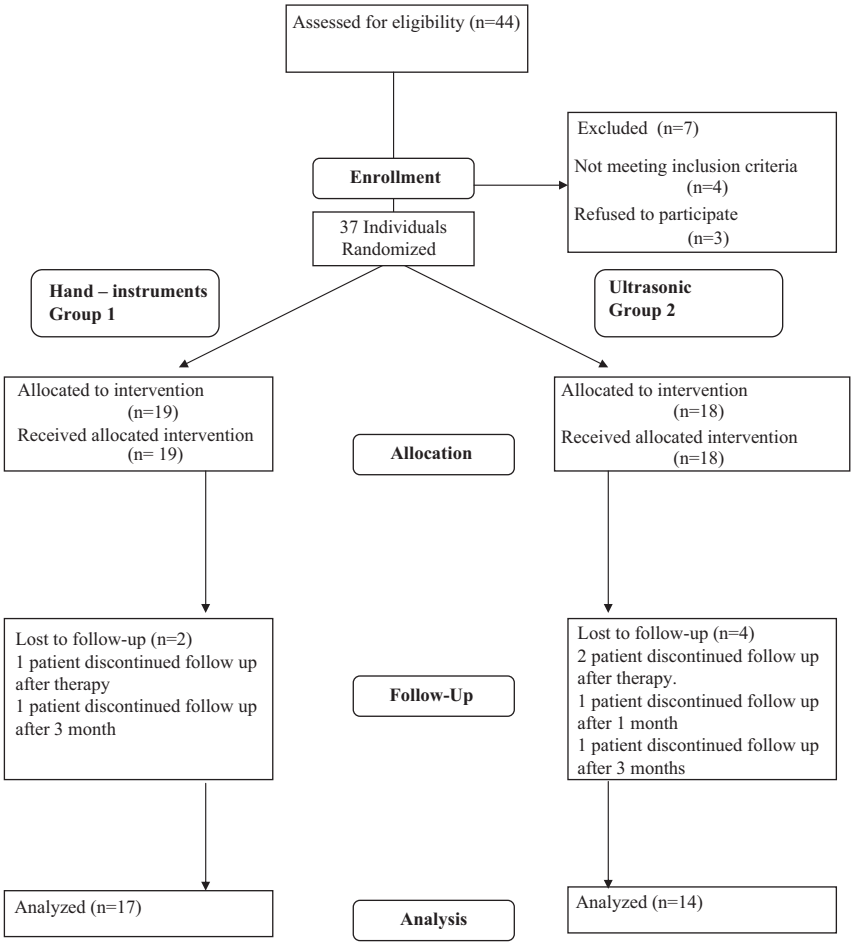

Fig. 1. A consort E-flowchart of the enrolment, allocation, follow-up, and analysis.

Table 1. Clinical characteristics in the two treatment groups over time are presented

Study group	Variable	Baseline		1 month		3 months		6 months	
		mean	SD	mean	SD	mean	SD	mean	SD
Hand instruments	PPD at worst site	5.1	0.6	5.1	0.7	5.0	0.7	4.9	0.8
($N = 17$)	Mean PPD at implant	4.0	0.8	3.9	0.7	4.0	0.8	4.0	0.8
	Graded bleeding at worst site	2.2	0.7	1.9	0.9	1.9	0.9	1.8	1.0
	Mean bleeding at implant	1.7	0.9	1.5	0.8	1.4	0.9	1.4	1.0
	Full mouth mean BOP	32.6	22.2	29.4	23.0	33.7	23.5	34.3	28.2
	Mean PI at implant	91.2	15.1	67.2	17.2	75.0	32.9	62.5	36.5
	Full mouth mean PI	78.0	19.8	55.4	21.9	56.9	29.5	54.9	29.5
Ultrasound	PPD at worst site	5.2	0.7	5.1	0.6	4.9	0.9	4.9	0.9
($N = 14$)	Mean PPD at implant	4.3	0.6	4.2	0.6	4.1	0.6	3.9	0.8
	Graded bleeding at worst site	2.3	0.5	1.6	1.1	1.8	1.0	1.6	0.8
	Mean bleeding at implant	1.7	0.6	1.1	0.7	1.2	0.7	1.2	0.7
	Full mouth mean BOP	35.4	23.3	25.1	22.8	26.6	26.7	28.7	26.4
	Mean PI at implant	82.1	30.1	53.6	36.5	63.4	35.5	57.5	37.5
	Full mouth mean PI	68.2	22.2	48.9	25.4	48.4	21.8	51.3	23.9

PPD, probing pocket depth; BOP, bleeding on probing; PI, plaque index; SD, standard deviation.

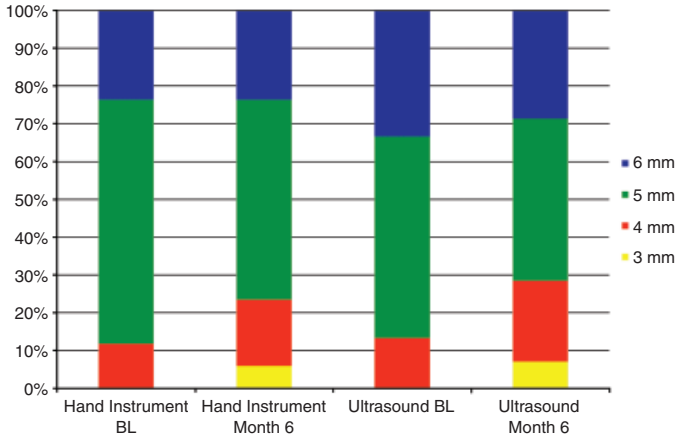

Fig. 2. Distribution of probing pocket depths at baseline and at month 6 at the worst implant sites treated either with hand instruments or with ultrasonic devices.

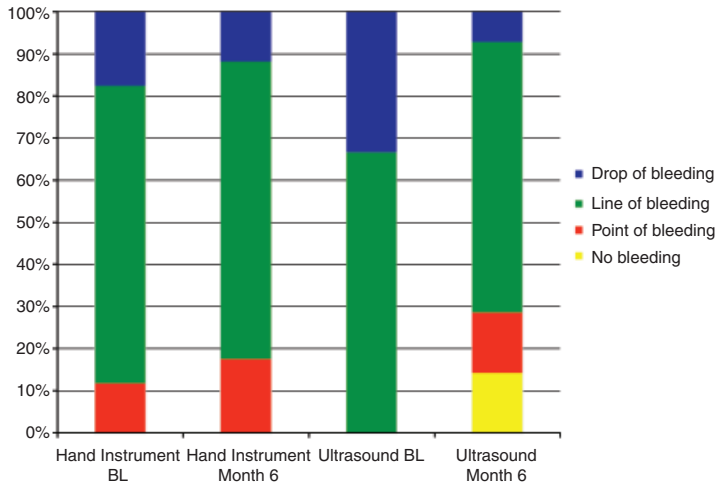

Fig. 3. Distribution of bleeding scores at baseline and at month 6 at implants treated with hand instruments or ultrasonic devices.

independent *t*-test (equal variance not assumed) failed to demonstrate group differences by PPD at any time point (*p*-values varying between 0.85 and 0.62). Over time, analysis by Kruskal–Wallis ANOVA failed to demonstrate differences in BOP and plaque scores between study groups. At the different time points, data analysis by the Mann–Whitney *U*-tests also failed to demonstrate group differences. *p*-values varied between $p = 0.14$ (mean bleeding score at implant month 1) and 0.97 (PPD at the worst site of implants and at month 6). The distributions of PPD and graded bleeding scores at the worst site of implants are presented in Figs. 2 and 3, respectively. The range of the mean probing depth (four sites at the implants) varied between 2.8 and 5.5 mm. In other words, the probing depths were unchanged in comparison with the baseline measurements. At the worst implant sites, nine implants had a PPD of 4 mm, 16 implants had a PPD of 5 mm and six implants had a PPD of 6 mm.

While no differences were found between study groups, the data from the two groups were merged. Further analysis comparing baseline data with the results at 6 months demonstrated that oral hygiene had improved significantly from a mean plaque index (PI) score of 73% at baseline to 53% at month 6 (mean difference: 20.2%, SE ± 6.3, 95% CI: 7.0–32.7, $p < 0.01$). The improved oral hygiene was also reflected by a decrease in PI at the study implants (mean difference: 27.2%, SE ± 7.9, 95% CI: 11.3–43.1, $p < 0.001$). At month 6, the graded bleeding index at the worst site of each implant studied was significantly lower ($p < 0.01$). At the four sites of study implants, the mean BOP score was also reduced ($p = 0.026$). Statistical analysis failed to demonstrate changes in PPD values between baseline and month 6 ($p = 0.30$).

Microbiological findings

At baseline, statistical analysis by the Mann–Whitney *U*-test failed to demonstrate group differences in the total bacterial load ($p = 0.58$). Immediately following treatment, however, statistically and significantly lower total bacterial counts were found in the group treated with hand instruments ($p < 0.01$). At week 1, a statistically significant difference was found between treatment groups, and with higher bacterial counts in the group treated with ultrasonic devices ($p < 0.05$). The distributions of total bacterial counts are presented in a boxplot diagram illustrating a decrease in median counts at week 1, but only in the group treated with hand instruments (Fig. 4).

Intent-to-treat analysis

The baseline assessments for those subjects who never returned to follow-up visits were carried forward and included in the 1-, 3- and 6-month clinical data set. In this intent-to-treat analysis, the mean PPD at the deepest implant site in the group treated with hand instruments varied between 5.2, 5.2, 5.2 and 5.1 mm at the baseline, 1-, 3- and 6-month measurements, respectively. In the group receiving treatment with the ultrasonic device, the corresponding mean PPD values were 5.2, 5.2, 5.1 and 5.1 mm, respectively. Similar lack of changes was seen for the implant mean PPD value changes at sites treated with hand instruments varying from baseline to month 6 between 4.1, 4.0, 4.0 and 4.1 mm, respectively. The corresponding PPD mean values in the ultrasonic-treated group were 4.2, 4.3, 4.2 and 4.1 mm, respectively. Statistical analysis by Kruskal–Wallis ANOVA and by repeated Mann–Whitney *U*-tests, or by repeated independent *t*-tests (equal variance not assumed) processed both for within groups or between groups failed to demonstrate differences in values for the implant site with the deepest probing, or for the implant overall mean PPD values (*p*-values varied between 0.60 and 0.95). Analysis of the extent of inflammation by bleeding index also consistently failed to demon-

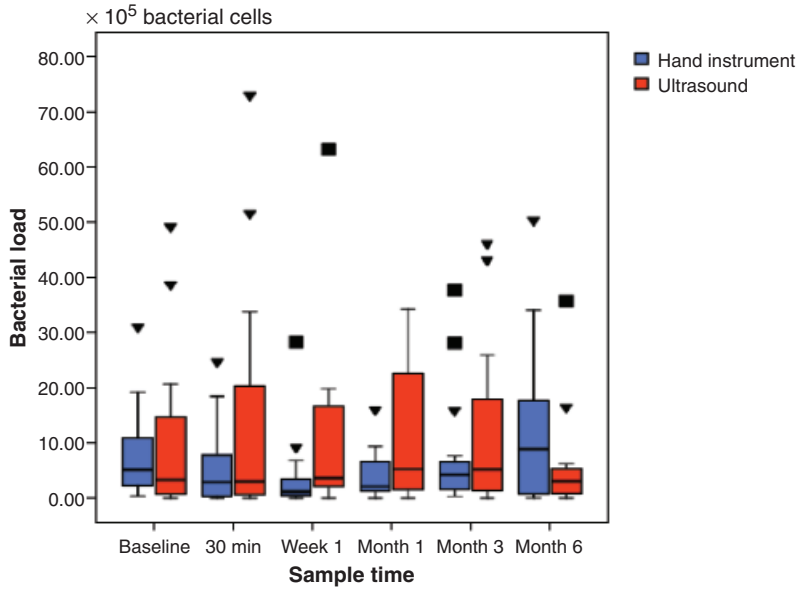

Fig. 4. Boxplot diagram illustrates the distribution of total bacterial load at implants treated with hand instruments or ultrasonic devices from baseline through month 6.

strate differences between groups or over time changes.

In the intent-to-treat analysis of the microbiological data based on the sum of bacterial load for the species included, the latest available values were carried forward. In the hand instrument-treated group, statistical analysis by Kruskal–Wallis ANOVA failed to demonstrate changes over time ($p = 0.78$). Likewise, in the ultrasound-treated group, no differences were found over time in bacterial sum ($p = 0.97$). In addition, statistical analysis failed to demonstrate differences with regard to the effect of different treatment modalities at the different time points.

Discussion

Knowledge of the prevalence of implant mucositis and peri-implantitis is limited. However, in a recent review it was reported that the prevalence of implant mucositis is >60% and that the prevalence of peri-implantitis may vary between 28% and 56% (Zitzmann). As a consequence, adequate intervention methods for the treatment of implant mucositis and peri-implantitis need to be explored and evaluated.

A 12% difference in BOP scores between groups and improvements in PPD within the range of measurement errors is clinically irrelevant. Our findings that the interventions did not result in clinically relevant improvements are consistent with a previous report (Karring et al. 2005). Other data suggest that non-surgical debridement with adjunct chlorhexidine or antibiotics can result in clinically relevant improvements of peri-implantitis (Renvert et al. 2006, 2008a, b, Máximo et al. 2009). The data from the present study demonstrated that the non-surgical intervention reduced bleeding scores at the study implants but with no impact on PPD.

We calculated the statistical power for the present study such that 18 subjects were needed in each treatment arm. After the initial treatment session, six subjects did not return and were therefore excluded from the data analysis. In the intent-to-treat analysis carrying forward the latest known values for subjects who never completed the study also failed to demonstrate clinical differences between treatment groups or within groups over time. This was perhaps anticipated, given the lack of differences demonstrated in the analysis that excluded these subjects. It might have been anticipated that immediately after the treatment and up to 3-month data should show positive clinical effects. This intent-to-treat analysis also failed to demonstrate clinically positive effects of therapy in cases with early peri-implantitis.

Whether the six subjects who did not fulfil the study were included or not, the analysis did not change our conclusion that neither treatment modality had an impact on the total bacterial load of species included in the analysis. This further enforces our perception that not even shortly after treatment can a relevant reduction in bacterial counts be obtained by any of these two treatment modalities

Implant designs and implant surface structures hamper the ability to mechanically disturb and remove the biofilm. In addition, the design of supra-structures and the typical anatomy of implant lesions further limit the ability to debride the infected regions. This was illustrated by the inability to reduce the bacterial load. In the present study, we used specifically designed instruments (curettes and ultrasonic tips) aimed at the mechanical treatment of implant infections. It should also be highlighted that the therapist performing the clinical interventions was highly experienced and was given no time restrictions in terms of treatments. Because of the fact that only a few subjects were smokers, we could not assess the potential impact of smoking as a factor in the treatment outcomes.

Because of the design of prosthetic construction on implants, assessment of bleeding at implant sites is difficult. Another complicating factor when assessing bleeding at implant sites may be the absence of attached soft tissues. In an attempt to discriminate between possible traumatic bleeding from bleeding as an expression of inflammation, we used a modified bleeding index. The criteria used were related to the characteristics of the bleeding following probing in the following manner: (1) no bleeding, (2) point of bleeding, (3) line of bleeding, and (4) drop in bleeding. Figure 3 illustrates that it may be possible to distinguish between different bleeding characteristics at implant sites both before and following therapy.

Oral hygiene improved over time both at implant sites and as assessed from full-mouth scores. Nevertheless, the overall oral hygiene remained poor and in spite of the fact that oral hygiene measures were emphasized at each visit. This lack of good oral hygiene may partly explain why the interventions were ineffective. The impact on the subgingival microbiota assessed as the total bacterial load at a time point 30 min. after instrumentation was limited in both treatment arms. This further demonstrated the inability of both methods in removing subgingival bacteria. The reduction in bacterial counts at week 1 observed in the hand instrument-treated

group is difficult to explain. It is possible that hand instruments may be more aggressive to surrounding soft tissues, inducing a host immune response of limited duration that might explain this finding. It is also possible that the ultrasonic therapy had resulted in dislodging of bacteria in a suspension that was easily collected with the paper points. Other data suggest that, over time, it is not possible to eliminate or reduce the counts of key pathogen at periodontal sites in subjects with persistent deep probing pocket depths (McColl et al. 2006).

There are few intervention studies that have assessed the effects of mechanical therapy on the microbiota at implant lesions (Renvert et al. 2008a, b). Although the total bacterial load was not affected, changes may have occurred in the composition of the biofilm at the implants treated. It would be of importance to assess microbial changes over time allowing the development of adjunct antimicrobial therapies.

Furthermore, a better understanding of local inflammatory responses during therapy could also provide information that could possibly allow the development of anti-inflammatory adjunct treatment methods. Whether surgical intervention with or without adjunct antimicrobial and/or anti-inflammatory drugs would allow effective control and management of implant lesions remains to be studied. The small sample size and the dropout ratios must be considered in the interpretation of the results of this study. In spite of these limitations, the clinical and microbiological changes reported following non-surgical mechanical debridement suggest that a non-surgical treatment of early cases of peri-implantitis is not clinically relevant.

Conclusions

- We failed to demonstrate differences in treatment outcomes between the two treatment methods studied.
- We failed to demonstrate clinically relevant changes within groups over a follow-up period of 6 months.
- We failed to demonstrate that the treatment provided changed the total bacterial load.
- We found that oral hygiene and bleeding scores at implant sites remained poor.

References

Albrektsson, T. & Isidor, F. (1994) Consensus report of session IV. In: Lang, N. P. & Karring, T. (eds). *Proceedings of the First European Workshop on Periodontology*, pp. 365–369. London: Quintessence.

Costerton, J. W., Stewart, P. S. & Greenberg, E. P. (1999) Bacterial biofilms: a common cause of persistent infections. *Science* **284**, 1318–1322.

Karring, E. S., Stavropoulos, A., Ellegaard, B. & Karring, T. (2005) Treatment of peri-implantitis by the Vectors system. A pilot study. *Clinical Oral Implants Research* **16**, 288–293.

Lamont, R. J. & Jenkinson, H. F. (2000) Subgingival colonization by *Porphyromonas gingivalis*. *Oral Microbiology and Immunology* **15**, 341–349.

Máximo, M. B., de Mendonça, A. C., Santos, R. V., Figueiredo, L. C., Feres, M. & Duarte, P. M. (2009) Short-term clinical and microbiological evaluations of peri-implant diseases before and after mechanical anti-infective therapies. *Clinical Oral Implants Research* **20**, 99–108.

McColl, E., Patel, K., Dahlen, G., Tonetti, M., Graziani, F., Suvan, J. & Laurell, L. (2006) Supportive periodontal therapy using mechanical instrumentation or 2% minocycline gel: a 12 month randomized, controlled, single masked pilot study. *Journal of Clinical Periodontology* **33**, 141–150.

Porras, R., Anderson, G. B., Caffesse, R., Narendran, S. & Trejo, P. M. (2002) Clinical response to 2 different therapeutic regimens to treat peri-implant mucositis. *Journal of Periodontology* **73**, 1118–1125.

Renvert, S., Lessem, J., Dahlén, G., Lindahl, C. & Svensson, M. (2006) Topical minocycline microspheres versus topical chlorhexidine gel as an adjunct to mechanical debridement of incipient peri-implant infections: a randomized clinical trial. *Journal of Clinical Periodontology* **33**, 362–329.

Renvert, S., Lessem, J., Dahlén, G., Renvert, H. & Lindahl, C. (2008a) Mechanical and repeated antimicrobial therapy using a local drug delivery system in the treatment of peri-implantitis: a randomized clinical trial. *Journal of Periodontology* **79**, 836–844.

Renvert, S., Roos-Jansåker, A-M. & Claffey, N. (2008b) Non-surgical treatment of peri-implant mucositis and peri-implantitis: a literature review. *Journal of Clinical Periodontology* **35** (Suppl. 8), 305–315.

Schwarz, F., Sculean, A., Rothamel, D., Schwenzer, K., Georg, T. & Becker, J. (2005) Clinical evaluation of an Er:YAG laser for nonsurgical treatment of peri-implantitis: a pilot study. *Clinical Oral Implants Research* **16**, 44–52.

Strooker, H., Rohn, S. & Van Winkelhoff, A. J. (1998) Clinical and microbiologic effects of chemical versus mechanical cleansing in professional supportive implant therapy. *International Journal of Oral Maxillofacial Implants* **13**, 845–850.

Zitzmann, N. U. & Berglundh, T. (2008) Definition and prevalence of peri-implant diseases. *Journal of Clinical Periodontology* **35** (Suppl. 8), 286–291.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. CONSORT Statement 2001.

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Clinical Relevance

Scientific rationale for the study: Peri-implantitis is a common clinical condition.
Principal findings: The extent of bleeding at implant sites was reduced, and plaque levels decreased but probing pocket depths and bacterial load did not differ between pre-treatment and 6-month results or between treatment modalities.
Practical implications: Mechanical debridement with hand instruments or ultrasound devices of dental implants with a diagnosis of peri-implantitis does not result in clinical or microbiological changes. Therefore, these methods alone are insufficient in the management of peri-implantitis.