J Clin Periodontol 2011; 38: 65–73 doi: 10.1111/j.1600-051X.2010.01646.x

Journal of Clinical Periodontology

Treatment of peri-implantitis using an Er:YAG laser or an air-abrasive device: a randomized clinical trial

Stefan Renvert[1,2,3], Christel Lindahl[1,4], Ann-Marie Roos Jansåker[4] and G. Rutger Persson[1,5,6,7]

[1]Department of Oral Sciences, School of Health and Society, Kristianstad University, Kristianstad, Sweden; [2]School of Dental Sciences, Trinity College, Dublin, Ireland; [3]Blekinge Institute of Technology, Karlskrona, Sweden; [4]Department of Periodontology, Public Dental Health Service, Kristianstad, Sweden; [5]Department of Periodontology, University of Bern, Berne, Switzerland; [6]Department of Oral Medicine, University of Washington, Seattle, WA, USA; [7]Department of Periodontics, University of Washington, Seattle, WA, USA

Renvert S, Lindahl C, Roos Jansåker A-M, Persson GR. Treatment of peri-implantitis using Er:YAG laser or an air-abrasive device: a randomized clinical trial. J Clin Periodontol 2011; 38: 65–73. doi: 10.1111/j.1600-051X.2010.01646.x

Abstract

Background: Non-surgical peri-implantitis therapies appear to be ineffective. Limited data suggest that ER:YAG laser therapy improves clinical conditions. The present study aimed at comparing the treatment effects between air-abrasive (AM) and Er:YAG laser (LM) mono-therapy in cases with severe peri-implantitis.

Materials and methods: Twenty-one subjects in each group were randomly assigned to one time intervention by an air-abrasive device or an Er:YAG laser. Clinical data were collected before treatment and at 6 months. Data analysis was performed using repeat univariate analysis of variance controlling for subject factors.

Results: No baseline subject characteristic differences were found. Bleeding on probing and suppuration decreased in both the groups ($p<0.001$). The mean probing depth (PPD) reductions in the AM and LM groups were 0.9 mm (SD 0.8) and 0.8 mm (SD $\pm$ 0.5), with mean bone-level changes (loss) of -0.1 mm (SD $\pm$ 0.8) and -0.3 mm (SD $\pm$ 0.9), respectively (NS). A positive treatment outcome, PPD reduction $\geqslant$0.5 mm and gain or no loss of bone were found in 47% and 44% in the AM and LM groups, respectively.

Conclusions: The clinical treatment results were limited and similar between the two methods compared with those in cases with severe peri-implantitis.

Key words: air-abrasive; bone loss; intervention; laser; non-surgical; peri-implantitis

Accepted for publication 11 October 2010.

Over the last decades, dental implants have become a commonly used treatment alternative to other dental procedures. The prognosis of implant therapy in dentistry is perceived to be very good. The survival rates of dental implants after 10 years in function are in the range of 95% (Roos-Jansåker et al. 2006a). Nevertheless, infections adjacent to implants occur. The term peri-implant mucositis was proposed for reversible inflammation of the soft tissues surrounding implants, and if such an inflammation is combined with loss of bone, it is referred to as peri-implantitis (Albrektsson & Isidor 1994, Lindhe & Meyle 2008). Peri-implantitis, if not successfully treated, may lead to complete disintegration and implant loss (Esposito et al. 1999, Quirynen et al. 2002, Leonhardt et al. 2003). Data suggest that the prevalence of peri-implantitis is in the range of 16–25% (Fransson et al. 2005, Roos-Jansaker et al. 2006b, Koldsland et al. 2010). With an increasing population with dental implants, the prevalence of implant-related infections would most likely increase and cause major challenges to therapy.

The primary aetiology of implant mucositis and peri-implantitis is considered to be bacterial infections. After installation in the oral cavity, bacterial colonization occurs rapidly on oral implant surfaces (Quirynen et al. 2006, Fürst et al. 2007, Salvi et al. 2008), and the development of a tightly fixed layer of plaque binds to the implant surface as a biofilm (Lamont & Jenkinson 2000).

The goal in non-surgical therapy of peri-implant mucositis and peri-implantitis is to eliminate or significantly reduce the amounts of oral pathogens in the pockets around implants to a level

Conflict of interest and sources of funding statement

None of the authors have a conflict of interest. All authors met the authorship requirements listed by the ICJME guidelines.

The study was sponsored by Electric Medical Systems (EMS, Nyon, Switzerland), by KAVO (Biberach, Germany) and by Philips Oral Healthcare (Snoqualmie, WA, USA).

that allows healing and re-establishment of a clinically healthy condition. Using conventional means of therapy, eradication of pathogens by mechanical means on implant surfaces with threads and often with rough surface structures is difficult (Persson et al. 2010). Treatment models, such as scaling and root planning, effectively used to treat teeth with periodontitis, cannot be used in the same way on rough threaded implant surfaces. The implant rough surface structure also provides the bacteria with ''protected areas'' inaccessible to conventional mechanical removal. Treatment attempts have been made with an adjunct use of local antibiotics (i.e. Mombelli et al. 2001, Porras et al. 2002, Renvert et al. 2004, 2006, Jorgensen et al. 2004, Persson et al. 2006, Salvi et al. 2007). The adjunct use of local antibiotics to mechanical therapy has been shown to reduce bleeding on probing (BOP) and probing pocket depth (PPDs) in cases with peri-implantitis (Renvert et al. 2008). Another treatment model that may offer an advantage over traditional mechanical treatment includes the use of laser therapy. Data have shown that treatments with Er:YAG lasers have a bactericidal effect (Kreisler et al. 2002). Er:YAG laser treatment can debride the implant surface effectively and safely (Takasaki et al. 2007). Slightly better clinical results have been reported by Er:YAG laser treatment as compared with traditional non-surgical mechanical debridement (Schwarz et al. 2006a, b). In a recent consensus paper on the treatment of peri-implantitis, the authors concluded: ''In peri-implantitis lesions, non-surgical therapy was not found to be effective'' but that ''minor beneficial effects of laser therapy on peri-implantitis have been shown'' (Lindhe & Meyle 2008).

For several years, an air-abrasive method for the removal of bacterial plaque on tooth surfaces has been in use (Weaks et al. 1984, Berkstein et al. 1987, Horning et al. 1987, Petersilka et al. 2003). This method has also been used in the treatment of peri-implantitis, demonstrating no relevant adverse effects (Duarte et al. 2009). Until recently, air-polishing devices have used a slurry of water and sodium bicarbonate ($NaHCO_3$) and pressurized air/water. A less abrasive method using an amino acid glycine has been proven to be effective in removing bacterial biofilm structures in deep periodontal pockets. According to the manufacturer, a specially designed small, flexible and thin disposable plastic nozzle is placed in the infected pocket and the glycine powder in a compressed air flow removes biofilm structures under irrigation. Recent data have suggested that this treatment is safe by not causing emphysema, and provides clinical results comparable to those obtained by sub-gingival debridement of teeth using hand instruments (Moëne et al. 2010). The reasons for a low risk of emphysema may be the specially designed instrument tips, and the reduced flow and pressure in comparison with the previous methods used for supra-gingival polishing will cause less trauma to the tissues.

In vitro data have also demonstrated that the use of an air-abrasive device may change the surface characteristics of titanium implant surfaces. This may especially be the case when sodium bicarbonate particles are being used while the use of a glycine-based powder does not seem to cause titanium implant surface changes (Schwarz et al. 2009).

The aim of the present study was to assess the clinical outcomes following treatment with either a non-surgical debridement using an air-abrasive device or an Er:YAG laser in subjects with implants and a diagnosis of peri-implantitis.

Materials and methods

Study design

The design was a single masked, randomized 6-month clinical intervention trial including two study groups with a diagnosis of peri-implantitis.

Study population

The Ethics Committee of Lund University, Sweden, approved the study. Written consent was obtained from all enrolled subjects. The CONSORT guidelines for clinical trials were followed (Fig. 1). Subjects were enrolled if they presented with at least one dental implant with bone loss >3 mm at implants identified on intra-oral radiographs (Fig. 2), and having a PPD$\geqslant$ 5 mm with bleeding, and/or pus on probing as assessed by a 0.2 N probing force. Subjects may have had more than one implant, meeting the inclusion criteria.

The study was conducted between October 2007 and September 2009, and was performed at the Specialty Clinic for Periodontology, Region Skåne, Kristianstad, Sweden. The following criteria were used to exclude subjects from entry into the study: (I) poorly controlled diabetes mellitus (HbA1c >7.0), (II) use of anti-inflammatory prescription medications, or antibiotics within the preceding 3 months or during the study, (III) use of medications known to have an effect on gingival growth and (IV) subjects requiring prophylactic antibiotics.

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 randomized allocation was performed using a computer software program (SPSS Inc., Chicago, IL, USA). A clinician not involved with the study sequenced the study subjects to the therapy allocated. When performing their study tasks, the study examiner (M. N.) and the therapist

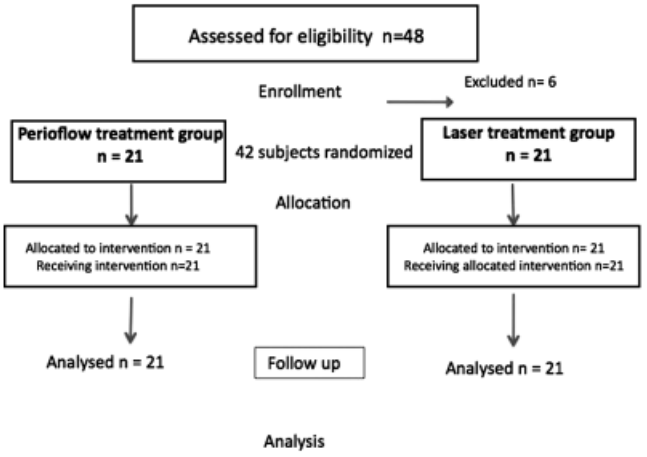

Fig. 1. Consort flow chart.

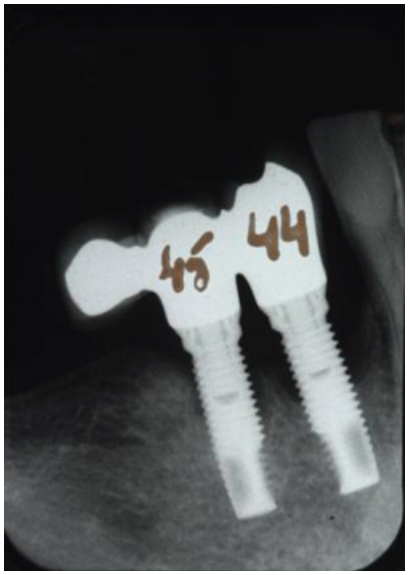

Fig. 2. Intra-oral radiograph of a dental implant with bone loss >3 mm.

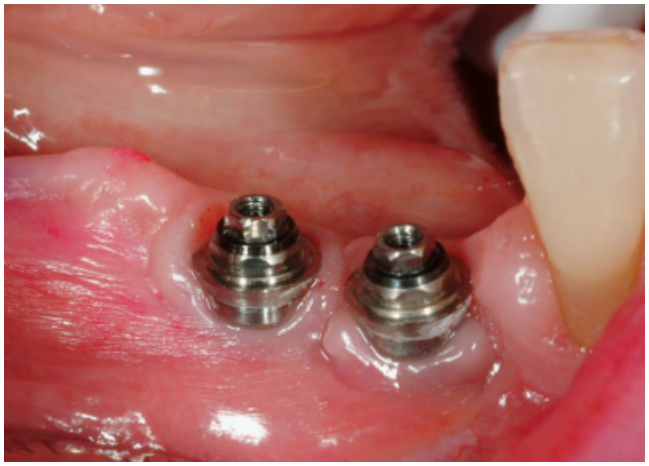

Fig. 3. Image representing implants to be measured after the removal of the implant superstructure.

(C. L.) were not jointly present with the study subjects. Study subjects were instructed not to discuss therapy with the study examiner. The study examiner was unaware of study treatment allocations, and performed all clinical measurements. The clinician performing treatments had >10 years of clinical experience in the mechanical treatment of implants with a diagnosis of peri-implantitis.

Clinical measurements and procedures

The PPD and bleeding on probing (BOP) measurements were performed using a plastic probe with a standardized probing force of 0.2 N (Click-Probe, KerrHawe SA, Bioggio Switzerland). All clinical measurements were obtained after removing the supra structure. The following clinical assessments were performed at baseline and at month 6: (I) full-mouth plaque score (FMPS) 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), (II) local plaque score defined as the presence of dental plaque along the mucosal margin at four sites of the treated implants, recorded after the use of a disclosing dye, (III) PPD at four sites per implant, (IV) full-mouth bleeding scores, bleeding appearing after PPD measurements of probing depth and expressed as a percentage of examined sites (four sites per tooth and the implant), (V) presence/absence of BOP at the implant (four sites/implant) and (VI) the amounts of bleeding at the implant sites was graded as follows: (0) no bleeding, (1) point of bleeding, (2) line of bleeding and (3) drop of bleeding.

Intra-oral standardized radiographs of sites of interest were obtained at baseline and at 6 months. Eggen holders were used for standardization purposes. Radiographs were analysed by one of the study investigators (G. R. P.) masked to study assignment, assessing digital images using the ImageJ software program 1:43r (National Institute of Health, Bethesda, MA, USA). For each image, the distance between three threads with a known thread distance was used for image calibration to compensate for image distortion. Thus, the distance between a known reference point at the implant to the deepest point of the bone lesion was defined in mm values.

Analysis of bone height assessments from 30 randomly selected digitized images identified Crohnbach's $\alpha = 0.96$ with an intra-class correlation of 0.96 (95% CI: 0.91–0.98, $p < 0.001$).

Treatment procedures

Before the treatments, the supra-structures were removed and the baseline measurements were performed (Fig. 3). In order to avoid bias, a dental hygienist who was not involved in the registrations performed the treatments (C. L.). After treatment, the supra-structures were remounted. Implants in the air-abrasive group were treated using the PERIO-FLOW® device (Fig. 4). The nozzle was placed in the pocket and mesially, lingually, distally and buccally, and used for approximately 15 s in each position and circumferentially in the pocket around the implant (Fig. 5). Careful attempts were made to cover the full circumference of the implant. The PERIOFLOW® device utilizes a 25 μm hydrophobic powder and a flexible tip allowing access to periodontal and implant pockets and with less biokinetic pressure than the original device for supra-gingival polishing.

Implants in the laser group were treated using an Er:YAG laser (Key Laser 3 Perio, KaVo, Biberach, Germany) (Fig. 6) at an energy level of 100 mJ/pulse and 10 Hz (12.7 J/cm^2) using a cone-shaped sapphire tip. The instrument tip was used in a parallel mode using a semicircular motion around the circumferential pocket area of the implant.

Routine local anaesthesia was used as needed. At all study time points, all subjects received individualized oral hygiene instructions. Each subject also received a sonic toothbrush (FlexCare+rechargeable sonic toothbrush, Philips Oral Healthcare, Snoqualmie, WA, USA). The study subjects were carefully instructed in the use of the toothbrush. They were supplied with new brush heads after 3 months.

Statistical methods

If a PPD difference in the change of PPD between methods of 1 mm is to be

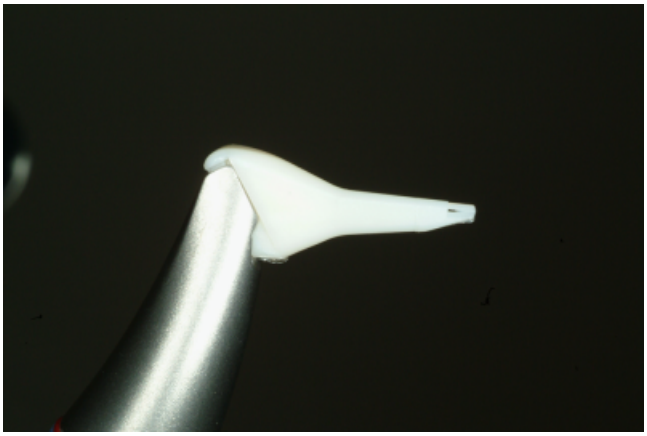

Fig. 4. The PERIO-FLOW® device.

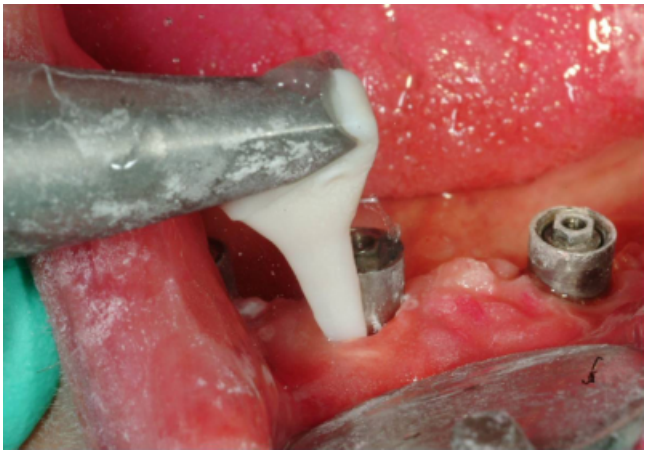

Fig. 5. Use of the air-abrasive device at an infected site with the supra-structure removed.

detected at $\alpha = 0.05$ and a power of $\beta = 0.2$, the appropriate number of subjects per group would be around 20. Hence, the inclusion of 42 subjects in the study would yield the necessary statistical power. The data were analysed using repeated univariate analysis of variance adjusting for the subject factor and the number of implants treated in each subject. The data were also analysed using independent *t*-tests for continuous variables with a normal distribution (equal variance not assumed) PPD, bone level changes and using Mann–Whitney *U*-tests for non-parametric data (BOP, PI, suppuration) and by χ^2 analysis. A statistical software package (SPSS PASW, Statistics 18.0 for MAC, SPSS Inc.) was used for the statistical analysis. Statistical differences were defined by a p value <0.05. PPD change was defined as the primary outcome measure. The secondary outcome measure was a change in bone height.

Results

All subjects completed the study, and no implants were lost. The mean age of the subjects was 68.5 (SD ± 6.4) in the laser group and 68.9 (SD +12.5) in the air-abrasive group, with no group difference ($p = 0.91$). Statistical analysis also failed to demonstrate study group gender differences ($p = 0.34$), differences in smoke years ($p = 0.58$) or differences in the medications used ($p = 0.17$). Furthermore, statistical analysis also failed to demonstrate differences in the number of implants in the upper ($p = 0.54$) or the lower jaw ($p = 0.19$) between study groups.

In the laser group, 21 subjects had a total of 55 implants (machined surface: 41, medium rough surface:14). Each subject in this group had, on average, 2.6 implants (SD ± 0.2, range: 1–8 implants) with a diagnosis of peri-implantitis. In the air-abrasive group, 21 subjects had a total of 45 implants (machined surface: 29, medium rough surface: 16). Each subject in this group had, on average, 2.0 implants (SD ± 0.2, range: 1–5 implants) with a diagnosis of peri-implantitis. Subjects in the laser group had more implants (mean difference: 0.6, SE ± 0.2, 95% CI: 0.1–0.2, $p<0.05$).

Analysis by independent *t*-tests (equal variance not assumed) failed to demonstrate differences in the treatment outcomes (PPD change and bone height change) as a result of implant surface characteristics within each group.

Evidence of inflammation defined by assessment of bleeding, presence of visible plaque or suppuration

At the subject level, statistical analysis failed to demonstrate baseline study group differences in the gingival index scores ($p = 0.67$) and plaque scores ($p = 0.55$). Consistently, statistical analysis failed to demonstrate study group differences in the gingival index and plaque scores at the 6-week control visit, or at 3- and 6-month controls. Statistical analysis also failed to demonstrate group differences in changes of BOP scores between baseline and after 6 months by study group interventions ($p = 0.79$).

At the implant level, the distributions of BOP at baseline and month 6 in the two study groups are presented (Fig. 7). At baseline, a point of bleeding was found at 5.1% of all implant surfaces, a line of bleeding at 37.8% and a drop of bleeding at 57.1% of the sites. Statistical analysis failed to demonstrate baseline differences by BOP between different implant surfaces (four surfaces per tooth) ($p = 0.76$). At month 6, no evidence of bleeding was found at 30.9% of the implants treated in the laser group and at 25.0% of the implants treated in the air-abrasive group. The decrease in BOP was significant in both study groups ($p<0.001$). Statistical analysis failed to demonstrate differences in changes of BOP by study intervention groups ($p = 0.22$).

The proportions of implants with visible plaque at baseline 2, 6, 12 weeks and at 6 months after treatment for the two study groups are presented (Fig. 8). At baseline, the presence of visible plaque was found at 35% of implants in the laser group and at 21.7% of implants in the air-abrasive group ($p<0.01$). At 2, 6 and 12 weeks after treatment, statistical analysis (Mann–

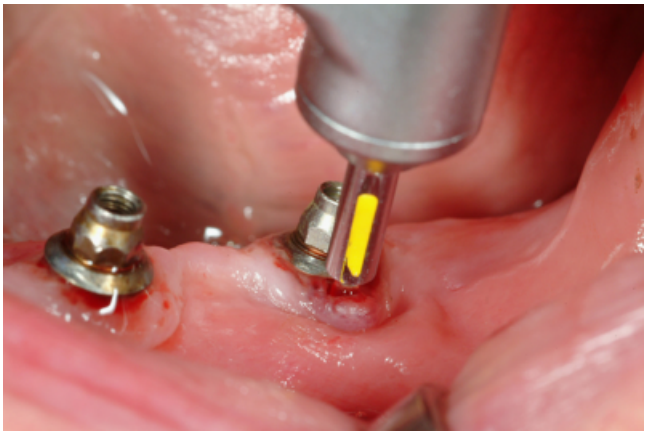

Fig. 6. Use of the Er:YAG laser at an infected site with the supra-structure removed.

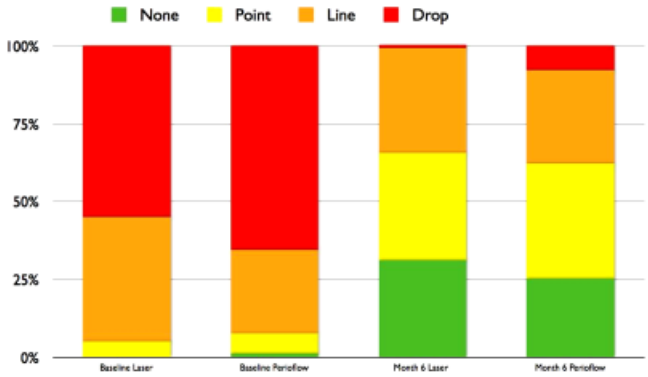

Fig. 7. The distributions of bleeding on probing (no bleeding, point of bleeding, line of bleeding and drop of bleeding) at baseline and month 6 in the two study groups.

Whitney *U*-tests) failed to demonstrate differences in visible plaque between treatment groups. At month 6, however, less plaque was found at implants treated in the air-abrasive group ($p < 0.05$).

At baseline, 30.9% (17/55) of implants in the laser group presented with suppuration while in the air-abrasive group, 31.1% (14/45) of implants presented with suppuration. At 6 months after treatment, 10.9% (6/55) of sites in the laser-treated group presented with suppuration. In the air-abrasive treatment group, 11.1% (5/45) of sites presented with suppuration. Statistical analysis (Mann–Whitney *U*-test) failed to demonstrate baseline differences by treatment group assignment with regard to the presence/absence of suppuration ($p = 0.63$). The decrease in suppuration was significant in both treatment groups ($p < 0.001$), and with no group differences in the change (decrease) of suppuration between baseline and at 6 months after treatment ($p = 0.42$). Nevertheless, and among the sites that presented with suppuration at baseline in the laser-treated group, 37.9% also presented with suppuration at the examination at 6 months. Furthermore, among the sites that presented with suppuration at baseline in the air-abrasive treatment group, 46.0% also presented with suppuration at the examination at the study endpoint at 6 months.

Assessments of PPDs

The proportional changes in PPDs between baseline and month 6 defined at the implant level are presented (Table 1). The changes in PPD between baseline and month 6 in the two treatment groups at the subject level are illustrated in a box-plot diagram (Fig. 9). Repeat univariate analysis of variance adjusting for subject factors and the number of implants treated in each subject failed to demonstrate differences in changes of PPD by study group assignment ($p = 0.76$). At the implant level, the PPD change (reduction) between baseline and 6 months in the laser-treated group was 0.8 mm (SD $\pm$ 0.5), whereas the PPD change (reduction) in the air-abrasive-treated group 0.9 mm (SD $\pm$ 0.8). Statistical analysis failed to demonstrate differences in changes of PPD by study group intervention ($p = 0.55$).

Thus, at the subject level, 25% of the subjects in the laser group had an average PPD reduction $\geqslant 1.0$ mm whereas 38% of the subjects in the air-abrasive group had an average PPD reduction $\geqslant 1.0$ mm.

Radiographic assessments

Analysis by repeat univariate analysis of variance at the subject level adjusting for subjects factors and number of implants treated in each subject failed to demonstrate differences in alveolar bone changes between baseline and 6 months as an effect of intervention at the mesial ($p = 0.46$) and distal implant sites ($p = 0.83$). When the individual implant was used as the unit of observation, statistical analysis (independent *t*-test) also failed to demonstrate intervention group differences in changes of bone height for mesial ($p = 0.35$), and distal ($p = 0.55$) aspects, or combined mean values between mesial and distal bone changes ($p = 0.42$). The average change in the bone level was a loss of 0.3 mm (SD +0.9) for the laser group and a loss of 0.1 mm (SD $\pm$ 0.8) bone height for the air-abrasive group.

The proportional changes in bone height levels between baseline and month 6 assessed from radiographs and defined at the implant level are presented (Table 2). The changes in bone height levels between baseline and month 6 at the subject level in the two treatment groups are illustrated in a box-plot diagram (Fig. 10).

Combined outcome assessment

Defining a positive outcome of therapy as a subject-based reduction in PPD $\geqslant 0.5$ mm and gain or no further loss of bone as successful and all other treatment results as clinically not satisfactory, statistical analysis failed to demonstrate treatment group differences at the subject level ($p = 0.84$). When the implant level was used to assess outcome, the laser treatment resulted in improved conditions at 44% of the implants. Treatment with the air-abrasive device resulted in improved conditions at 47% of the implants. However,

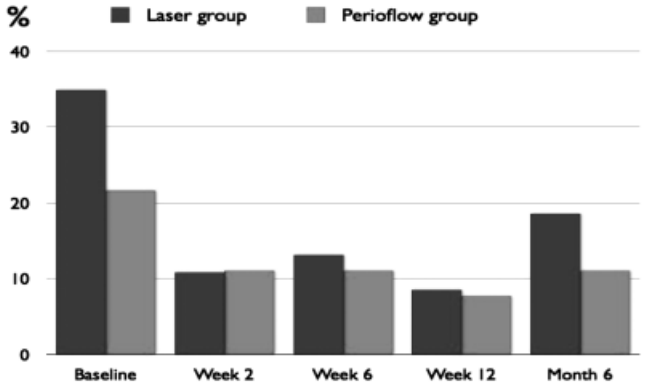

Fig. 8. Proportions of implants with a visible presence of plaque at baseline, 2, 6 and 12 weeks and 6 months after treatment in the two study groups.

Table 1. Proportional changes in probing pocket depth between baseline and month 6 defined at the implant level (mean value of four sites/implant)

Probing depth changes	Laser treatment (%)	Air-abrasive treatment (%)
Decrease (mm)		
⩾4	0.0	0.0
3.1–4.0	1.9	2.2
2.1–3.0	4.2	7.9
1.1–2.0	12.1	14.0
0.1–1.0	37.4	29.2
Unchanged (mm)		
0.0	35.0	35.4
Increase (mm)		
0.1–1.0	7.9	7.9
1.1–2.0	1.4	1.7
2.1–3.0	0.0	1.7
3.1–4.0	0.0	0.0

if defining a positive outcome as having PPD⩾5 mm, with BOP and suppuration at baseline but no PPD⩾5 mm, no BOP and no suppuration at 6 months, none of the cases in either group obtained this level of treatment outcome.

Discussion

In a recent Cochrane systematic review, the authors concluded that there is very little reliable evidence suggesting as to which could be the most effective interventions for peri-implantitis (Esposito et al. 2008). We have demonstrated previously that there are no differences in treatment outcomes in the treatment of non-surgical debridement of implants with a diagnosis of peri-implantitis comparing hand instrumentation or debridement with an ultrasonic device, and none of these treatment modalities resolved the clinical conditions (Renvert et al. 2009). The consensus report by the European Workshop on Periodontology 2008 agreed that in peri-implantitis lesions, non-surgical therapy was not found to be effective, whereas minor effects of laser therapy of peri-implantitis have been shown (Lindhe & Meyle 2008). Given the fact that non-surgical mechanical debridement of peri-implantitis has been found to be ineffective, we did not use this as a control treatment modality.

Treatment of peri-implantitis using Er:YAG laser therapy has been investigated (Schwarz 2006a, b). Thus, treatment with an Er:YAG laser appears to result in a more effective reduction in bleeding around implants than non-surgical debridement with hand instruments and sub-gingival application of chlorhexidine (Schwarz et al. 2006a). In order to treat the implants with the laser or the air-abrasive device in the present study, the superstructures were removed, allowing the best access possible to the implant surfaces. Thus, the results of the present study are limited to implants where the superstructures can be removed. All implants are not screw retained. It might be difficult to gain access with the laser tip or the air-abrasive nozzle if implant super-structures are not removed. Removing and repositioning of superstructures add to the clinical time necessary to provide care, thereby increasing the treatment expenses.

In the present study, no differences in the reduction of BOP 6 months after treatment were found between laser treatment and treatment with the air-abrasive device. Although oral hygiene improved greatly and absence of plaque at implants treated was found at many implants, a large proportion of the implants continued to have BOP at the month 6 post-treatment assessments. In the present study, BOP was graded to discriminate the severity of inflammation. It could be argued that when measuring around an implant, it is easy to traumatize the tissues, resulting in a dot of bleeding. This may not represent an actual inflammatory response. If a line or a drop was considered as representative of inflammation, approximately 30% of implants in the laser- and air-abrasive-treated groups presented with bleeding, which was consistent with other data (Schwarz et al. 2006a).

In cases with periodontitis, good plaque control has been identified in many periodontal studies as a prerequisite for good treatment results (i.e. Axelsson et al. 2004). Good plaque control is also a prerequisite for success in implant maintenance therapy (Roccuzzo et al. 2010). In a previous study (Renvert et al. 2009) assessing the efficacy of non-surgical mechanical debridement of implants with a diagnosis of peri-implantitis oral hygiene, although significantly improved in comparison with baseline, remained with insufficient plaque index scores. In that study, following careful instructions, manual toothbrushes were used as the method of oral hygiene. In the present study, subjects were instructed in the use of a sonic toothbrush (Philips Oral Healthcare). The use of a sonic toothbrush to maintain oral hygiene around teeth appears to have clinical benefits in comparison with the use of manual toothbrushes (O'Beirne et al. 1996, Zimmer et al. 2002). Different from the study by Renvert et al. (2009) although treated by the same clinician (C. L.), the subjects in the present study obtained a very good level of plaque control at their implants. In

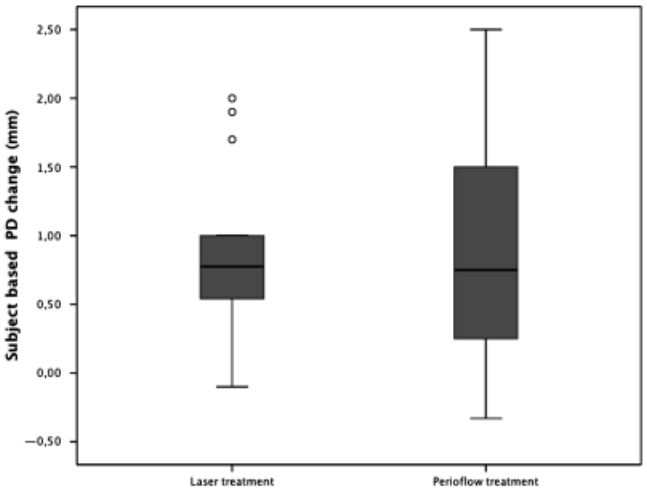

Fig. 9. The changes in the probing pocket depth values (mm) between baseline and month 6 at the subject level in the two treatment groups are illustrated in a box-plot diagram identifying median, 25th and 75th percentiles.

Table 2. Proportional changes in bone levels between baseline and month six defined at the implant level based on the mean value of mesial and distal bone height changes

Radiographic changes in bone levels	Laser treatment (%)	Air-abrasive treatment (%)
Decrease in bone level (gain) (mm)		
0.1–1.0	35.4	29.3
1.1–2.0	4.2	12.2
Unchanged (mm)		
0.0	2.1	2.4
Increase in bone level (loss) (mm)		
2.1–3.0	6.3	0.0
1.1–2.0	12.5	17.1
0.1–1.0	39.5	39.0

spite of this, many of the study subjects in both groups still had clinical evidence of peri-implantitis and significant levels of clinically visible inflammation expressed both as BOP and suppuration, indicating that both treatment modalities are insufficient to treat deep peri-implantitis defects.

BOP appears to be a common finding around oral implants (Bonde et al. 2010, Koldsland et al. 2010). The width of keratinized mucosa around implants may or may not explain plaque accumulation and bleeding (Kim et al. 2009, Schrott et al. 2009). One of the primary problems in assessing the extent of inflammation around implants is the design of the supra-structures. Thus, it is often difficult to gain adequate access to the implant surfaces for clinical measurements. In the present study, all supra-structures were removed before measurements and clinical procedures providing the best access possible. In the present study, we also applied two methods for the assessment of tissue inflammation: (I) the conventional bleeding index (bleeding or no bleeding) and (II) an index suggested for assessments around implants (Mombelli et al. 1987, Schrott et al. 2009). There appears to be a need to develop specific methods for the assessment of inflammation around titanium dental implants.

The scientific literature on the use of radiographs to assess alveolar bone conditions around dental implants is extensive. Early evaluations of interventions and follow-up are also common using 3-, 6-, 9- or 12-month follow-up periods (Huynh-Ba et al. 2008, Bergkvist et al. 2009, Kronstrom et al. 2010). A 6-month period to assess bone changes by analysis of the intra-oral radiographs may be considered as a short evaluation period.

Approximately 50% of subjects in both the groups showed improved clinical conditions as an effect of the study intervention. One explanation why the two treatment modalities had similar and limited success might be the fact that due to the severity of disease, the instrumentation was unable to have an impact on the microbiota around the implants. Further studies are needed to assess the impact of laser and air-abrasive treatments of cases with peri-implant mucositis and peri-implantitis, and whether there are threshold values beyond which non-surgical intervention may not be possible.

Another explanation may be that both treatment modalities resulted in tissue trauma resulting in progressive bone loss around the implants. The settings for the Er:YAG laser in the present study were, however, below the defined risk values reported recently (Stubinger et al. 2010). None of the subjects in the laser study group presented with serious adverse events following therapy. Likewise, and consistent with studies using the air-abrasive device in periodontal pockets around teeth (Moëne et al. 2010), no serious adverse events were identified in subjects treated with the air-abrasive device.

A common problem in clinical dental research is the availability of study subjects who meet the defined inclusion criteria. This problem also exists in studies related to the treatment of peri-implantitis. Furthermore, the criteria for clinically relevant treatment outcome in the management of peri-implantitis vary considerably between available studies. In the present study, we considered potential errors in measurements of probing pocket depth and what might be a reasonable clinical improvement. Given the time-consuming and expensive removal of implant prosthesis before treatment with either of the two devices, we decided that for one method to be superior to the other, a 1 mm difference in the clinical change of PPD would be required.

In conclusion, the results of therapy of subjects with peri-implantitis after 6 months are similar between treatments using an Er:YAG laser or by the air-abrasive PERIO-FLOW® for debridement of implants diagnosed with severe peri-implantitis. Both methods resulted in a reduction of PPD, the frequency of suppuration and bleeding at implants with a diagnosis of peri-implantitis. The overall clinical improvement was limited.

Acknowledgements

We appreciate the financial support and equipment provided by Electric Medical Systems (EMS, Nyon, Switzerland), by

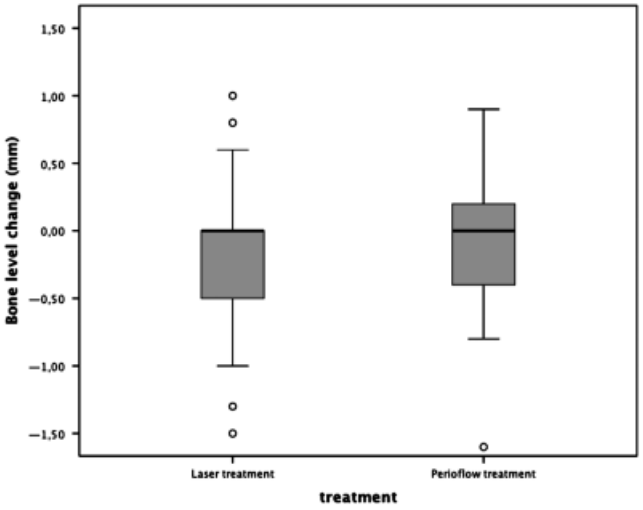

Fig. 10. The changes in bone levels between baseline and month 6 in the two treatment groups at the subject level are illustrated in a box-plot diagram identifying median, 25th, 75th percentiles and outlier values = ○.

KAVO, Biberach, Germany) and by Philips Oral Healthcare, Snoqualmie, WA, USA). We also appreciate work performed by Ms. Margareta Nilsson and Ms. Pernilla Karlgren-Andersson in the study.

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.

Axelsson, P., Nyström, B. & Lindhe, J. (2004) The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults. Results after 30 years of maintenance. *Journal of Clinical Periodontology* **31**, 749–757.

Bergkvist, G., Nilner, K., Sahlholm, S., Karlsson, U. & Lindh, C. (2009) Immediate loading of implants in the edentulous maxilla: use of an interim fixed prosthesis followed by a permanent fixed prosthesis: a 32-month prospective radiological and clinical study. *Clinical Implant Dentistry and Related Research* **11**, 1–10.

Berkstein, S., Reiff, R. L., McKinney, J. F. & Killoy, W. J. (1987) Supragingival root surface removal during maintenance procedures utilizing an air-powder abrasive system or hand scaling. An in vitro study. *Journal of Periodontology* **58**, 327–330.

Bonde, M. J., Stokholm, R., Isidor, F. & Schou, S. (2010) Outcome of implant-supported single-tooth replacements performed by dental students. A 10-year clinical and radiographic retrospective study. *European Journal of Oral Implantology* **3**, 37–46.

Duarte, P. M., de Mendonça, A. C., Máximo, M. B., Santos, V. R., Bastos, M. F. & Nociti, F. H. (2009) Effect of anti-infective mechanical therapy on clinical parameters and cytokine levels in human peri-implant diseases. *Journal of Periodontology* **80**, 234–243.

Esposito, M., Grusovin, M. G., Coulthard, P. & Worthington, H. V. (2008) The efficacy of interventions to treat peri-implantitis: a Cochrane systematic review of randomised controlled clinical trials. *European Journal of Oral Implantology* **1**, 111–125.

Esposito, M., Hirsch, J., Lekholm, U. & Thomsen, P. (1999) Differential diagnosis and treatment strategies for biologic complications and failing oral implants: a review of the literature. *International Journal of Oral Maxillofacial Implants* **14**, 473–490.

Fransson, C., Lekholm, U., Jemt, T. & Berglundh, T. (2005) Prevalence of subjects with progressive bone loss at implants. *Clinical Oral Implants Research* **16**, 440–446.

Fürst, M., Salvi G, E., Lang, N. P. & Persson, G. R. (2007) Bacterial colonization immediately after installation on oral titanium implants. *Clinical Oral Implants Research* **18**, 501–508.

Horning, G. M., Cobb, C. M. & Killoy, W. J. (1987) Effect of an air-powder abrasive system on root surfaces in periodontal surgery. *Journal of Clinical Periodontology* **14**, 213–220.

Huynh-Ba, G., Friedberg, J. R., Vogiatzi, D. & Ioannidou, E. (2008) Implant failure predictors in the posterior maxilla: a retrospective study of 273 consecutive implants. *Journal of Periodontology* **79**, 2256–2261.

Jorgensen, M. G., Safarian, A., Daneshmand, N., Keim, R. J. & Slots, J. (2004) Initial antimicrobial effect of controlled-release doxycycline in subgingival sites. *Journal of Periodontal Research* **39**, 315–319.

Kim, B. S., Kim, Y. K., Yun, P. Y., Yi, Y. J., Lee, H. J., Kim, S. G. & Son, J. S. (2009) Evaluation of peri-implant tissue response according to the presence of keratinized mucosa. *Oral Surgery Oral Medicine Oral Pathology Oral Radiology and Endodontics* **107**, 24–28.

Koldsland, O. C., Scheie, A. A. & Aass, A. M. (2010) Prevalence of peri-implantitis related to severity of the disease with different degrees of bone loss. *Journal of Periodontology* **81**, 231–238.

Kreisler, M., Kohnen, W., Marinello, C., Götz, H., Duschner, H., Jansen, B. & d'Hoedt, B. (2002) Bactericidal effect of the Er: YAG laser on dental implant surfaces: an in vitro study. *Journal of Periodontology* **73**, 1292–1298.

Kronstrom, M., Davis, B., Loney, R., Gerrow, J. & Hollender, L. (2010) A prospective randomized study on the immediate loading of mandibular overdentures supported by one or two implants: a 12-month follow-up report. *International Journal of Oral Maxillofacial Implants* **25**, 181–188.

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

Leonhardt, Å., Dahlén, G. & Renvert, S. (2003) Five year clinical, microbiological, and radiological outcome following treatment of peri-implantitis in man. *Journal of Periodontology* **74**, 1415–1422.

Lindhe, J. & Meyle, J.Group D of European Workshop on Periodontology. (2008) Peri-implant diseases: consensus report of the Sixth European Workshop on Periodontology. *Journal of Clinical Periodontology* **35** (Suppl.), 282–285.

Moëne, R., Décaillet, F., Andersen, E. & Mombelli, A. (2010) Subgingival plaque removal using a new air-polishing device. *Journal of Periodontology* **81**, 79–88.

Mombelli, A., Feloutzis, A., Brägger, U. & Lang, N. P. (2001) Treatment of peri-implantitis by local delivery of tetracycline. Clinical, microbiological and radiological results. *Clinical Oral Implants Research* **12**, 287–294.

Mombelli, A., van Oosten, M. A., Schurch, E. Jr & Land, N. P. (1987) The microbiota associated with successful or failing osseointegrated titanium implants. *Oral Microbiology and Immunology* **2**, 145–151.

O'Beirne, G., Johnson, R. H., Persson, G. R. & Spektor, M. D. (1996) Efficacy of a sonic toothbrush on inflammation and probing depth in adult periodontitis. *Journal of Periodontolology* **67**, 900–908.

Persson, G. R., Samuelsson, E., Lindahl, C. & Renvert, S. (2010) Mechanical non-surgical treatment of peri-implantitis: a single-blinded randomized longitudinal clinical study. II. Microbiological results. *Journal of Clinical Periodontology* **37**, 563–573.

Persson, G.R, Salvi, G. E., Heitz-Mayfield, L. J. & Lang, N. P. (2006) Antimicrobial therapy using a local drug delivery system (Arestin) in the treatment of peri-implantitis. I: microbiological outcomes. *Clinical Oral Implants Research* **17**, 386–393.

Petersilka, G. J., Tunkel, J., Barakos, K., Heinecke, A., Häberlein, I. & Flemmig, T. F. (2003) Subgingival plaque removal at interdental sites using a low-abrasive air polishing powder. *Journal of Periodontology* **74**, 307–311.

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.

Quirynen, M., De Soete, M. & van Steenberghe, D. (2002) Infectious risks for oral implants: a review of the literature. *Clinical Oral Implants Research* **13**, 1–19.

Quirynen, M., Vogels, R., Peeters, W., van Steenberghe, D., Naert, I. & Haffajee, A. (2006) Dynamics of initial subgingival colonization of 'pristine' peri-implant pockets. *Clinical Oral Implants Research* **17**, 25–37.

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**, 362–369.

Renvert, S., Lessem, J., Lindahl, C. & Svensson, M. (2004) Treatment of Incipient peri-implant infections using topical minocycline microspheres versus topical chlorhexidine gel as an adjunct to mechanical debridement. *Journal of the International Academy of Periodontology* **6**, 154–159.

Renvert, S., Roos-Jansåker, A. M. & Claffey, N. (2008) Non-surgical treatment of peri-implant mucositis and peri-implantitis: a literature review.

Journal of Clinical Periodontology **35** (Suppl.), 305–315.

Renvert, S., Samuelsson, E., Lindahl, C. & Persson, G. R. (2009) Mechanical non-surgical treatment of peri-implantitis: a double-blind randomized longitudinal clinical study. I: clinical results. *Journal of Clinical Periodontology* **36**, 604–609.

Roccuzzo, M., De Angelis, N., Bonino, L. & Aglietta, M. (2010) Ten-year results of a three-arm prospective cohort study on implants in periodontally compromised patients. Part 1: implant loss and radiographic bone loss. *Clinical Oral Implants Research* **21**, 490–496.

Roos-Jansåker, A. M., Lindahl, C., Renvert, H. & Renvert, S. (2006a) Nine- to fourteen-year follow-up of implant treatment. Part I: implant loss and associations to various factors. *Journal of Clinical Periodontology* **33**, 283–289.

Roos-Jansaker, A. M., Lindahl, C., Renvert, H. & Renvert, S. (2006b) Nine- to fourteen-year follow-up of implant treatment. Part II: presence of peri-implant lesions. *Journal of Clinical Periodontology* **33**, 290–295.

Salvi, G. E., Fürst, M. M., Lang, N. P. & Persson, G. R. (2008) One-year bacterial colonization patterns of *Staphylococcus aureus* and other bacteria at implants and adjacent teeth. *Clinical Oral Implants Research* **19**, 242–248.

Salvi, G. E., Persson, G. R., Heitz-Mayfield, L. J., Frei, M. & Lang, N. P. (2007) Adjunctive local antibiotic therapy in the treatment of peri-implantitis II: clinical and radiographic outcomes. *Clinical Oral Implants Research* **18**, 281–285.

Schrott, A. R., Jimenez, M., Hwang, J. W., Fiorellini, J. & Weber, H. P. (2009) Five-year evaluation of the influence of keratinized mucosa on peri-implant soft-tissue health and stability around implants supporting full-arch mandibular fixed prostheses. *Clinical Oral Implants Research* **20**, 1170–1177.

Schwarz, F., Bieling, K., Bonsmann, M., Latz, T. & Becker, J. (2006a) Nonsurgical treatment of moderate and advanced periimplantitis lesions: a controlled clinical study. *Clinical Oral Investigations* **10**, 279–288.

Schwarz, F., Bieling, K., Nuesry, E., Sculean, A. & Becker, J. (2006b) Clinical and histological healing pattern of peri-implantitis lesions following non-surgical treatment with an Er: YAG laser. *Lasers Surgical Medicine* **38**, 663–671.

Schwarz, F., Ferrari, D., Popovski, K., Hartig, B. & Becker, J. J. (2009) Influence of different air-abrasive powders on cell viability at biologically contaminated titanium dental implants surfaces. *Journal of Biomedical Materials Research. Part B, Applied Biomaterials* **88**, 83–91.

Stubinger, S., Etter, C., Miskiewicz, M., Homann, F., Saldamli, B., Wieland, M. & Sader, R. (2010) Surface alterations of polished and sandblasted and acid-etched titanium implants after Er: YAG, carbon dioxide, and diode laser irradiation. *International Journal of Oral Maxillofacial Implants* **25**, 104–111.

Takasaki, A. A., Aoki, A., Mizutani, K., Kikuchi, S., Oda, S. & Ishikawa, I. (2007) Er: YAG laser therapy for peri-implant infection: a histological study. *Lasers in Medical Science* **22**, 143–157.

Weaks, L. M., Lescher, N. B., Barnes, C. M. & Holroyd, S. V. (1984) Clinical evaluation of the Prophy-Jet as an instrument for routine removal of tooth stain and plaque. *Journal of Periodontology* **55**, 486–488.

Zimmer, S., Nezhat, V., Bizhang, M., Seemann, R. & Barthel, C. (2002) Clinical efficacy of a new sonic/ultrasonic toothbrush. *Journal of Clinical Periodontology* **29**, 496–500.

Address:
Stefan Renvert
Department of Oral Sciences
School of Health and Society
Kristianstad University
Kristianstad
Sweden
E-mail: stefan.renvert@hkr.se

Clinical relevance

Scientific rationale for the study: Presently, limited evidence exists on the efficacy of treatment interventions following non-surgical treatment of peri-implantitis.

Principal findings: The overall clinical improvement was limited. Similar treatment results were obtained for the implants treated by the air-abrasive PERIO-FLOW® or with the Er:YAG Key 3 laser. Both methods resulted in a reduction of probing pocket depth, the frequency of suppuration and bleeding.

Clinical implications: Clinical conditions at advanced peri-implantitis lesions improve following laser or air-abrasive treatments. Neither laser nor air-abrasive treatments predictably resolve advanced cases of peri-implantitis.