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Subgingival ultrasonic instrumentation of residual pockets irrigated with essential oils: a randomized controlled trial

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Abstract

Aim: To evaluate the clinical efficacy of subgingival ultrasonic instrumentation irrigated with essential oils (EOs) of residual periodontal pockets.

Material and methods: Sixty-four individuals with chronic periodontitis were invited to participate in this randomized, double-blind, parallel, and placebo-controlled clinical trial. All subjects received non-surgical periodontal therapy. After re-evaluation (baseline), residual pockets (pocket depth $\geqslant 5$ mm) received test (ultrasonic instrumentation irrigated with EOs) or control therapy (ultrasonic instrumentation irrigated with negative control). Probing pocket depth (PPD), gingival recession (R), clinical attachment level (CAL), bleeding on probing (BOP), and plaque were assessed at baseline and after 4, 12, and 24 weeks. Differences between groups and changes over the course of time were analysed according to a generalized linear model.

Results: There was a significant reduction in PPD and BOP, as well as a significant CAL gain in the two groups ($p < 0.001$). Nevertheless, there were no differences between the groups at any time of the study. When only initially deep pockets (PPD $\geqslant 7$ mm) were analysed, a significantly greater CAL gain ($p = 0.03$) and PPD reduction ($p = 0.01$) was observed in the test group.

Conclusion: The adjunctive use of EOs may promote significant CAL gain and PPD reduction in deep residual pockets.

Key words: essential oils; periodontal disease; periodontal pocket; ultrasonic instrumentation

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Dental biofilm is considered the main aetiologic factor of periodontal disease (Slots 1979, Socransky & Haffajee 2002). Longitudinal studies have demonstrated that periodontal disease can be successfully treated by means of mechanical removal of dental biofilm, dental calculus, and oral hygiene instruction (Claffey et al. 1988, van der Weijden & Timmerman 2002). Nevertheless, presence of residual pockets $\geqslant 5$ mm after treatment have been associated with greater risk for periodontal disease progression (Claffey & Egelberg 1995, Renvert & Persson 2002, Matuliene et al. 2008) and loss of the respective tooth (Matuliene et al. 2008), which would indicate the need for additional procedures to reduce residual pockets.

An approach to the treatment of residual pockets is the local application of antimicrobials. This application can be achieved with the use of ultrasonic instrumentation irrigated with antiseptics. Various antiseptics have been used as irrigating agents. Although chlorhexidine is considered the most efficient antimicrobial for supragingival plaque (P) control, its use as irrigant with subgingival ultrasonic instrumentation did not promote additional clinical benefits (Taggart et al. 1990, Reynolds et al. 1992, Guarnelli et al. 2008). Povidine iodine has shown good results as a substance for subgingival irrigation

Conflict of interest and source of funding statement

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(Rosling et al. 1986, 2001, Leonhardt et al. 2006, Ribeiro et al. 2006, Zanatta et al. 2006), but presents the risk of hypersensitivity reaction (Niedner 1997), and its prolonged use has been associated with thyroid dysfunction (Nobukuni et al. 1997) and stain formation on tooth surfaces and mucosa (Clark et al. 1989).

Essential oils (EOs) have shown evidence of efficacy and safety in gingivitis and supragingival plaque control (Overholser et al. 1990, Charles et al. 2004, Sharma et al. 2004, Stoeken et al. 2007). EOs have the capacity to rupture the cell wall of certain microorganisms and inhibit their enzymatic activity (Kubert et al. 1993). They may also extract endotoxins from Gram-negative pathogens (Fine et al. 1996a). In vitro and in vivo studies have shown the ability of EOs to penetrate the dental biofilm and exert a bactericide effect (Pan et al. 2000, Ouhayoun 2003). Moreover, a recent study (Fine et al. 2007) showed that EOs are capable of eliminating subgingival microorganisms, which further supports their potential to be tested as a subgingival irrigating agent.

Studies have evaluated the efficacy of EOs as a subgingival irrigation agent in the reduction of plaque, gingivitis, and the number of microorganisms (Ciancio et al. 1989, Fine et al. 1994, 1996b, Cortelli et al. 2009). However, in those studies, the EOs solution was either applied by means of home subgingival irrigation devices by the patients themselves, or by professional irrigation with syringes. No studies were found in the literature, evaluating the effects of ultrasonic subgingival instrumentation irrigated with EO in the clinical parameters of periodontal disease. Thus, the aim of the present study was to evaluate the clinical efficacy of subgingival ultrasonic instrumentation irrigated with EOs of residual pockets. The null hypothesis was that no difference in the outcome variables exists between patients treated with ultrasonic instrumentation irrigated with EOs or a negative control.

Material and Methods

Study design and casuistic

A randomized, double-blind, parallel, controlled clinical trial was conducted. The duration of the study was 6 months.

Individuals with chronic periodontitis, consecutively recruited from the Dental Clinics of University of São Paulo, were invited to participate in this study. To be eligible for the study, the volunteer had to present the following inclusion criteria: (1) chronic periodontitis according to Tonetti & Claffey (2005) (presence of proximal attachment loss of 5 mm or more in 30% or more of teeth present), (2) age equal to or over 35 years, (3) presence of at least 15 teeth in the oral cavity, (4) at least one site with probing pocket depth (PPD) $\geqslant$5 mm after the re-evaluation examination, and (5) consent to participating in the study. The exclusion criteria were as follows: (1) smoking, (2) treatment with antibiotics in the last 6 months, (3) any systemic alteration that might interfere in the prognosis of the periodontal disease (e.g., diabetes, HIV infection, etc.). The study protocol was approved by the institution's Ethics Committee. The trial was conducted at the Dental Clinics of University of São Paulo, São Paulo, Brazil.

The primary outcome for this study was clinical attachment level (CAL) gain after 24 weeks (CAL gain). Moreover, the following secondary outcomes were measured: plaque (P), bleeding on probing (BOP), pocket probing depth (PPD), and gingival recession (R). Plaque was assessed by percentage of sites with visible plaque, and BOP assessed by percentage of sites that bled after probing.

To calculate the sample size, it was considered that a difference of 1.0 mm in CAL between the groups would be clinically relevant. Using a power of 80% to detect this difference, a level of significance of 5%, a one-tailed test and a standard deviation of 1.5 mm in CAL, 28 subjects per group would be necessary. To compensate losses during the follow-up, 32 volunteers per group were recruited (64 individuals, 15% more than the calculated number). Sample size calculation was performed with the software STATA 11 (StataCorp LP, College Station, TX, USA).

The patients were randomly allocated to the test (ultrasonic subgingival instrumentation (Profi III Ceramic, Dabi Atlante, Ribeirão Preto, São Paulo, Brazil) irrigated with EOs (Listerine Cool Mint, Johnson & Johnson, Guarulhos, São Paulo, Brazil) containing 0.064% thymol, 0.092% eucalyptol, 0.06% methyl salicylate, 0.042% menthol, and 21.6% ethanol) or control group (ultrasonic subgingival instrumentation irrigated with a negative control: sorbitol solution 15%, ethanol 21%, sodium saccharin 0.05%, mint flavouring, green dye). The control solution had the same color, taste, and alcohol concentrations as the test solution.

An independent pharmacy (Farmácia ''Fórmula e Ação'', São Paulo, Brazil) produced the negative control and dispensed both the test and control mouthwashes in flasks identified only as ''Group A'' or ''Group B''. Thus the investigator responsible for the measurements, the investigator responsible for the treatment and the patients did not know whether they received the antiseptic or the control mouthwash. The pharmacy revealed this information at the end of the study.

A random sequence was generated by an independent statistician, by means of a computer software, in blocks of $n = 4$. The participants were included consecutively, and the sequence was concealed until their inclusion. The investigator responsible for the screening and inclusion of the individuals opened consecutively opaque envelopes that contained the information about the group to which the individual was allocated, according to the random sequence.

Clinical procedures

The participants in this study underwent a complete periodontal examination at the screening examination. The periodontal examination was performed in all the teeth, six sites per tooth, (mesio-buccal, buccal, disto-buccal, mesio-lingual, lingual, and disto-lingual). The following data were collected: plaque (P), BOP, pocket probing depth (PPD), R, and CAL. PPD and R were measured with a computerized probe (Florida Probe System, Florida Probe Corporation, Gainesville, FL, USA), and CAL was calculated as the sum of PPD and R. The data were measured by a single trained and calibrated examiner (C. C. B.). This examiner underwent an intra-examiner calibration before the study and during the course of the study (before the 3 and 6 months examinations). The calibration was performed in a total of 10 volunteers (four at the beginning of the study, three before the 3 months examination, and three before the 6 months examination). Calibration was performed for the parameters PPD and R, with an interval of 1 week between the examinations. The intra-class coefficients of correlation were calibrated to verify the reproducibility of the two examinations. There was

good correlation between the exams in the calibrations performed at the beginning and before the 3 and 6 months exams (ICC > 0.90).

After inclusion, all the volunteers received four to six sessions of non-surgical periodontal treatment (manual and ultrasonic supra and subgingival scaling and root planing, and oral hygiene instruction), performed by a periodontist (F. H.). No antimicrobial or anti-inflammatory therapy was prescribed during this phase.

Four weeks after the conclusion of periodontal treatment, the patients were recalled for re-evaluation (Segelnick & Weinberg 2006), in which a new complete periodontal examination was performed. This examination was considered the baseline examination. All the participants previously included maintained between 4 and 10 sites with PPD ⩾ 5 mm after non-surgical periodontal therapy; therefore, all continued in the study.

One week after the baseline examination, sites with pocket depth ⩾ 5 mm received subgingival ultrasonic instrumentation for 5 min., using EOs (test group) or control solution (control group) as the irrigation agent. In both groups the professional (H. S. F.) performed irrigation by moving the ultrasound tip slowly, vertically from the gingival margin to the apical extent of the pocket, and laterally in a sweeping motion. This procedure (ultrasonic instrumentation with irrigation agent for 5 min. per site) was repeated in the second and third weeks after re-evaluation, together with supragingival plaque control and calculus removal, performed with ultrasonic instrumentation and followed by prophylaxis with rubber cup.

Study subjects received periodontal examinations at baseline, and at 4, 12, and 24 weeks after baseline. At baseline, periodontal examination was performed in all the teeth, six sites per tooth, in order to identify residual pockets. After 4, 12, and 24 weeks, PPD, CAL, R, and BOP were measured only at the residual pockets sites (sites with PPD ⩾ 5 mm at baseline).

Adverse events

All the subjects who received at least one session of irrigation and who provided information about at least one follow-up session were included in the safety analysis and monitored for the incidence of adverse events. At each follow-up visit, the participants were asked about possible adverse events (such as a burning sensation on the tongue, sensitivity, aphtous lesions, etc.). The examiner also performed a complete oral examination to verify the presence of oral lesions.

Statistical analysis

The individual was the statistical unit. Thus, the means of all the sites were calculated for each individual, for all the outcome variables. Only the residual pockets sites (sites with PPD ⩾ 5 mm at baseline) were considered in the analysis.

The differences between the groups and the changes over the course of time were analysed according to a generalized linear model (repeated measures analysis of variance). Differences between the groups and study periods were verified by means of a multiple comparisons test (Newman–Keuls). An analysis of all the experimental sites was initially performed. After that, another analysis was performed, stratified by initial probing depth; that is, the sites were divided into initial PPD of 5–6.9 mm (moderate pockets) and sites with initial PPD ⩾ 7 mm (deep pockets). Also, an inter-group comparison of the changes in clinical variables between baseline and 24 weeks was performed with the Student's *t*-test.

Statistical analysis was performed per protocol, with the program SPSS for Windows (version 10.0). A level of significance α of 5% was used in all the statistical tests.

Results

Subjects were recruited from January to April 2008, and followed-up until October 2008. Figure 1 shows the flow of participants throughout the study. Sixty-four research subjects (22 men, 42 women, mean age 50.3 years) participated in this study and underwent an initial periodontal examination and subgingival irrigation according to the group to which he/she was allocated. The distribution of subjects according to sex and age is shown in Table 1.

Five research subjects (three from the control group and two from the test group) were lost in the follow-up period,

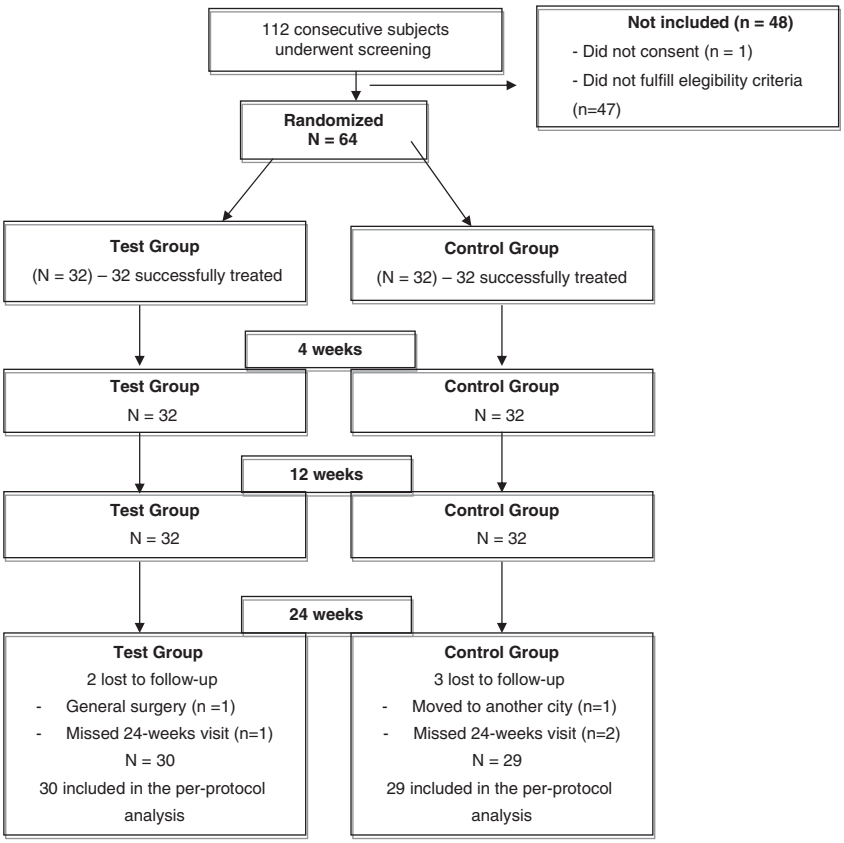

Fig. 1. Flow of participants.

between the 3 and 6 months examinations. Fifty-nine research subjects completed the study (30 in the test and 29 in the control group). A per-protocol analysis of the 59 subjects was conducted.

According to Table 2, there was no significant reduction in plaque in any of the groups. Also, there was no difference between the groups at any time of the study regarding plaque levels. A significant reduction in BOP was observed for the two groups, but there was no difference between the groups at any time of the study. A significant gain of 1.08 mm in CAL was verified in the test group between baseline and 24 weeks ($p<0.001$), while in the control group a significant gain of 0.94 mm was detected for the same period ($p<0.001$). Nevertheless, there was no difference between the groups at any experimental period regarding this outcome. A significant reduction of 1.55 mm between baseline and 24 weeks was observed in PPD in the test group ($p<0.001$), while in the control group a significant reduction of 1.18 mm was verified for the same period ($p<0.001$). There was no significant difference between the groups regarding PPD, at any experimental period of the study. In regard to R, the test group presented a significant increase of 0.47 mm and the control group a significant increase of 0.24 mm. Although this increase was significant ($p<0.001$), there was no difference between the groups at any time of the study. Also, there was no difference between groups regarding the difference between baseline and 24 weeks, for the variables plaque, BOP, CAL, PPD, and recession.

When only moderate pockets (initial PPD 5.0–6.9 mm) were analysed (Table 3), a significant gain in CAL was detected, being 0.98 mm ($p<0.001$) in the test group and 1.05 mm ($p<0.001$) in the control group. There was a significant reduction in PPD of 1.45 mm, after 6 months, in the test group ($p<0.001$) and 1.23 mm in the control ($p<0.001$). As regards recession, the test group presented a significant increase of 0.47 mm in R and the control group of 0.18 mm. There were no differences between the groups at any time of the study, regarding PPD, CAL, and R, when initially moderate pockets were analysed. No difference between groups regarding CAL gain, PPD reduction, and R increase was detected.

When only deep persisting pockets (initial PPD $\geqslant$ 7.0 mm) were considered (Table 4) (test group: 50 sites; control

Table 1. Distribution of subjects according to sex and age

	Test *N* (%)	Control *N* (%)	*p*
Sex			
Female	21 (65.6%)	21 (65.6%)	1.00
Male	11 (34.4%)	11 (34.4%)	
Age	54.31 ± 7.05	54.53 ± 8.92	0.91

Table 2. Mean, standard deviation, and comparison between groups according to the clinical parameters plaque (P), bleeding on probing (BOP), clinical attachment level (CAL), probing pocket depth (PPD), and gingival recession (R)

Variables	Group	Baseline	4 weeks	12 weeks	24 weeks	Difference baseline – 24 weeks	p^*
P	Test†	28.95 ± 28.47	20.55 ± 19.44	30.58 ± 27.41	20.32 ± 18.42	8.63 ± 7.57	0.28
Mean ± SD	Control‡	29.34 ± 26.18	28.12 ± 28.17	22.81 ± 28.62	26.12 ± 25.67	3.22 ± 5.29	
BOP	Test†	73.68 ± 23.57	41.14 ± 30.84§	45.90 ± 31.76§	31.78 ± 25.32§	41.90 ± 26.12	0.24
Mean ± SD	Control‡	80.00 ± 34.29	39.14 ± 24.11§	34.97 ± 26.06§	30.76 ± 28.54§	49.24 ± 28.19	
CAL	Test†	6.39 ± 0.79	5.33 ± 0.95§	5.08 ± 1.12§	5.31 ± 0.98§	1.08 ± 0.65	0.34
Mean ± SD	Control‡	6.73 ± 0.95	5.95 ± 1.11§	5.95 ± 1.11§	5.79 ± 1.27§	0.94 ± 0.63	
PPD	Test†	5.90 ± 0.62	4.42 ± 0.81§	4.29 ± 1.07§	4.35 ± 0.86§	1.55 ± 0.60	0.05
Mean ± SD	Control‡	5.95 ± 0.64	4.77 ± 0.80§	4.73 ± 0.93§	4.77 ± 0.94§	1.18 ± 0.65	
R	Test†	0.49 ± 0.72	0.91 ± 0.87§	0.79 ± 1.11§	0.96 ± 0.81§	− 0.47 ± 0.72	0.16
Mean ± SD	Control‡	0.78 ± 0.80	1.18 ± 0.91§	1.22 ± 1.07§	1.02 ± 0.77§	− 0.24 ± 0.69	

*Inter-group comparison regarding the difference between baseline and 24 weeks; *t*-test.
†Number of subjects = 30; number of sites = 152.
‡Number of subjects = 29; number of sites = 144.
§Intra-group significant difference in relation to baseline; Newman–Keuls test.

Table 3. Mean, standard deviation, and comparison between groups according to CAL (clinical attachment level), probing pocket depth (PPD), and gingival recession (R) in initially moderate pockets (5.0–6.9 mm)

Variables	Baseline	4 weeks	12 weeks	24 weeks	Difference baseline – 24 weeks	p^*
CAL (mean ± SD)						
Test†	5.94 ± 0.53	5.00 ± 0.80‡	4.76 ± 0.89‡	4.96 ± 0.99‡	0.98 ± 0.81	0.81
Control§	6.44 ± 0.73	5.76 ± 1.02‡	5.76 ± 1.00‡	5.39 ± 1.01‡	1.05 ± 0.66	
PPD (mean ± SD)						
Test†	5.47 ± 0.26	4.06 ± 0.61‡	3.92 ± 0.69‡	4.02 ± 0.79‡	1.45 ± 0.69	0.29
Control§	5.67 ± 0.37	4.56 ± 0.74‡	4.51 ± 0.87‡	4.44 ± 0.83‡	1.23 ± 0.71	
R (mean ± SD)						
Test†	0.47 ± 0.62	0.94 ± 0.86‡	0.84 ± 1.03‡	0.94 ± 0.78‡	− 0.47 ± 0.63	0.16
Control§	0.77 ± 0.81	1.20 ± 0.79‡	1.25 ± 1.03‡	0.95 ± 0.95‡	− 0.18 ± 0.58	

*Inter-group comparison regarding the difference between baseline and 24 weeks; *t*-test.
†Number of subjects = 28; number of sites = 102.
‡Intra-group significant difference in relation to baseline; Newman–Keuls test.
§Number of subjects = 27; number of sites = 100.

Table 4. Mean, standard deviation, and comparison between groups according to clinical attachment level (CAL), probing pocket depth (PPD), and gingival recession (R) in initially deep pockets (⩾7.0 mm)

Variables	Baseline	4 weeks	12 weeks	24 weeks	Difference baseline – 24 weeks	p^*
CAL (Mean ± SD)						
Test[†]	8.48 ± 0.71	7.12 ± 1.21[‡]	7.13 ± 1.76[‡]	6.92 ± 1.52[‡]	1.56 ± 1.35	0.03[§]
Control[¶]	8.95 ± 0.76	8.11 ± 1.16	7.60 ± 1.59[‡]	8.16 ± 0.90	0.79 ± 1.28	
PPD (Mean ± SD)						
Test[†]	7.93 ± 0.48	6.40 ± 0.83[‡]	6.48 ± 1.51[‡]	6.31 ± 1.33[‡]	1.62 ± 1.13	0.01[§]
Control[¶]	7.99 ± 0.51	6.47 ± 1.04[‡]	6.00 ± 1.71[‡]	7.02 ± 0.69[‡]	0.97 ± 1.11	
R (Mean ± SD)						
Test[†]	0.55 ± 0.63	0.72 ± 0.87	0.64 ± 1.03	0.61 ± 0.89	−0.06 ± 0.63	0.81
Control[¶]	0.96 ± 0.83	1.64 ± 0.93[‡]	1.60 ± 1.17[‡]	1.14 ± 0.98	−0.18 ± 0.83	

*Inter-group comparison regarding the difference between baseline and 24 weeks; *t*-test.
†$n = 12$; number of sites = 50.
‡Intra-group significant difference in relation to baseline; Newman–Keuls test.
§Inter-group significant difference regarding the difference between baseline and 24 weeks; *t*-test.
¶$n = 14$; number of sites = 44.

group: 44 sites), there was a significant gain of 1.56 mm in CAL for the test group ($p = 0.01$), between baseline and the 24 weeks measurements. In the control group there was a gain of 0.79 mm over the course of time, however, not significant. The test treatment promoted an additional 0.77 mm gain in initially deep pockets when compared with the control group. The difference in CAL gain between groups was significant ($p = 0.03$). There was a significant reduction in PPD of 1.62 mm for the test group ($p = 0.004$) after 6 months and 0.97 mm for the control group ($p = 0.01$). There was a significant difference of 0.65 mm ($p = 0.01$) between groups regarding PPD reduction. The test group presented an increase of 0.06 mm in R between the baseline and 24 weeks measurements, while the control group presented a reduction of 0.18 mm in gingival retraction. There was no difference between the groups during the study regarding this variable.

Adverse events occurred only during the ultrasonic instrumentation sessions. There was no report of adverse events in the follow-up sessions of 3 and 6 months. Two patients in the test group and two in the control group complained of dental sensitivity 1 week after ultrasonic instrumentation sessions. There was no association between group and the incidence of adverse events ($p = 1.00$).

Discussion

To the best of our knowledge, this was the first study that evaluated the efficacy of ultrasonic subgingival instrumentation irrigated with EOs in residual pockets. An improvement in the periodontal clinical parameters (PPD, CAL, and BOP) was observed in the two groups. Moreover, in pockets ⩾7 mm, there was an additional CAL gain and pocket depth reduction in the test group (irrigation with EOs) when compared with the control group. We used a parallel design instead of a split-mouth design due to the possibility of carry-across effect, that is, activity of the active agent (EOs) onto other sites (Quirynen et al. 2000).

The results of this study allow inferring that repeated ultrasonic instrumentation (three sessions) could be indicated for residual pockets (⩾5 mm), after non-surgical treatment. Ultrasonic instrumentation was capable of reducing moderate pockets with mean initial PPD between 5.47 mm (test) and 5.67 mm (control) to 4.00 mm and 4.44 mm, respectively (Table 3), which enables these sites to be maintained in a favorable situation, taking into consideration the risk of additional attachment loss presented by residual pockets ⩾5 mm (Claffey & Egelberg 1995, Renvert & Persson 2002, Matuliene et al. 2008).

When deep persisting pockets were analysed (initial PPD⩾7 mm), it was observed that ultrasonic subgingival instrumentation irrigated with EOs promoted a significantly greater 0.77 mm clinical attachment gain and a significantly greater PPD reduction of 0.65 mm, when compared with the control group. Deep pockets generally present a better response to mechanical treatment than moderate and shallow pockets. In a meta-analysis, Hung & Douglass (2002) showed that initially deep pockets presented a greater reduction in PPD and greater gain in CAL after scaling and root planing than moderate pockets. Although an additional gain in CAL was observed in the test group, there was no difference between the groups as regards PPD or any of the other secondary outcomes. Also, there was no difference between the groups with regard to any of the clinical parameters when pockets with initial PPD between 5 and 6.9 mm were analysed.

When analysing the results of the present study, it must be taken into consideration that the intervention (re-instrumentation with ultrasonics and irrigation with EOs) was performed after initial therapy. Interventions performed after initial therapy are supposed to result in less PPD reduction and CAL gain when compared with results of the initial phase of periodontal therapy, particularly due to the larger inflammatory infiltrate present at the gingival margin and soft tissue pocket wall before treatment. Wennström et al. (2005) performed re-instrumentation of residual pockets (sites with remaining PPD⩾5 mm) with ultrasonic or hand subgingival instrumentation, 3 months after initial therapy. The authors observed a further mean PPD reduction of 0.4 mm and mean CAL gain of 0.3 mm, 3 months after re-instrumentation, independent of the type of instrumentation. Salvi et al. (2002) randomized subjects to receive either locally delivered doxycycline, metronidazole gel or locally delivered chlorhexidine in residual pockets (PPD⩾5 mm). No re-instrumentation was performed. The authors reported mean PPD reduction varying from 0.25 to 0.33 mm, and mean CAL gain from 0.03 to 0.33 mm. Tomasi et al. (2008) treated residual pockets (PPD⩾5 mm after re-examina-

tion) with ultrasonic subgingival instrumentation with or without locally delivered doxycycline and observed mean PD reduction of 1.1 mm and mean CAL gain of 0.8 mm in the docycycline group. These values are comparable with the ones observed in the test group of the present study (1.55 mm mean PPD reduction and 1.08 mean CAL gain).

Many agents have been tested in subgingival irrigation procedures with varying results. Chlorhexidine is the most studied antimicrobial, but there is no evidence of efficacy after irrigation with this substance. One possible reason for this lack of effect could be the reaction with blood and proteins in the pocket fluids (Stanley et al. 1989). It is also possible that chlorhexidine would have to remain for a longer time within the pocket to exert an antimicrobial effect, since it takes 10 min. for a 0.5% chlorhexidine solution to eliminate *Porphyromonas gingivalis* after being mixed with serum (Oosterwaal et al. 1989). One possible explanation for the effect of EOs in deep pockets could be the lack of interaction with blood and fluid proteins, although there is no data to support it. Also, Fine et al. (2007) showed that a mouthwash containing EOs observed bactericidal activity against *P. gingivalis*, *Fusobacterium nucleatum* and *Veilonella* sp., which demonstrates the bactericidal potential of this antiseptic against subgingival bacteria.

Because this was the first study that evaluated the efficacy of ultrasonic instrumentation irrigated with EOs, the choice of irrigation time was based on studies with other active agents. The ultrasonic subgingival instrumentation time of 5 min. was chosen based on studies using agents such as chlorhexidine (Guarnelli et al. 2008), povidine iodine (Hoang et al. 2003), and tetracycline (Christersson et al. 1993).

With regard to the incidence of adverse events, only two volunteers in each group reported dental sensitivity. This event was probably associated with ultrasonic instrumentation, and not with the use of EOs, as it was reported 1 week after the instrumentation/irrigation sessions. There was no report of adverse events during follow-up of the participants at the 3- and 6-months visits. Moreover, the events occurred homogeneously in the two groups. Although the use of EOs has been associated with the complaint of burning sensation in the mouth (DePaola et al. 1989) when used in the form of a mouthrinse, no participant in the present study reported this sensation, probably because the solution of EOs was removed from the oral cavity with saliva ejectors during ultrasonic instrumentation.

In conclusion, the present study showed that ultrasonic instrumentation of residual pockets promoted a reduction in PPD and BOP and gain in CAL in the two groups. In pockets with initial PPD $\geqslant$ 7 mm, ultrasonic instrumentation irrigated with EOs may promote additional CAL gain and PPD reduction when compared with the control group. Further studies are necessary to verify the potential of this therapy when used during the initial stage of periodontal treatment, and whether the favorable effects observed could be achieved with fewer irrigation sessions.

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Clinical Relevance

Scientific rationale for the study: The presence of residual pockets $\geqslant 5$ mm has been associated with greater risk for periodontal disease progression and loss of the respective tooth, which would indicate the need for additional procedures to reduce residual pockets.

Principal findings: Ultrasonic instrumentation of residual pockets, irrigated with EOs, may promote additional CAL gain and PPD reduction in residual pockets with initial PPD $\geqslant 7$ mm. No benefits in moderate pockets were observed.

Practical implications: This experimental treatment may be used as an additional procedure to treatment and control of residual pockets.