ORIGINAL ARTICLE

Alkaline phosphatase activity in gingival crevicular fluid during human orthodontic tooth movement

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Bone remodeling that occurs during orthodontic tooth movement is a biologic process involving an acute inflammatory response in periodontal tissues. A sequence characterized by periods of activation, resorption, reversal, and formation has been recently described as occurring in both tension and compression tooth sites during orthodontic tooth movement. We used a longitudinal design to investigate alkaline phosphatase (ALP) activity in gingival crevicular fluid (GCF) to assess whether it can serve as a diagnostic aid in orthodontics. Sixteen patients (mean age, 15.5 years) participated in the study. The maxillary first molars under treatment served as the test teeth (TT) in each patient; in particular, 1 first molar was to be retracted and hence was considered the distalized molar (DM), whereas the contralateral molar (CM) was included in the fixed orthodontic appliance but was not subjected to the distal forces. The DM antagonist first molar (AM), free from any orthodontic appliance, was used as the baseline control. The GCF around the experimental teeth was harvested from mesial and distal tooth sites immediately before appliance activation, 1 hour after, and weekly over the following 4 weeks. The clinical gingival condition was evaluated at the baseline and at the end of the experimental term. ALP activity was determined spectrophotometrically at 30° C, and the results were expressed as total ALP activity (mUnits/sample). GCF ALP activity was significantly elevated in the DMs and the CMs as compared with the AMs at 1, 2, 3, and 4 weeks; conversely, in the AMs, GCF ALP activity remained at baseline levels throughout the experiment. Moreover, the enzyme activity in the DMs was significantly greater than in the CMs. In the DMs, a significantly greater ALP activity was observed in sites of tension compared with sites of compression. This difference was not seen with the CMs, in which the enzyme activity increased to the same extent in tension and compression sites. These results suggest that ALP activity in GCF reflects the biologic activity in the periodontium during orthodontic movement and therefore should be further investigated as a diagnostic tool for monitoring orthodontic tooth movement in clinical practice. (Am J Orthod Dentofacial Orthop 2002;122:548-56)

In orthodontics, mechanical stress appears to evoke biochemical and structural responses in a variety of cell types in vivo and in vitro.[1-8] The early phase of orthodontic tooth movement involves an acute inflammatory response, characterized by periodontal vasodilation and migration of leukocytes out of periodontal ligament capillaries.[1] The mechanism of bone resorption might also be related to the release of inflammatory mediators that can be detected in gingival crevicular fluid (GCF).[8] Moreover, orthodontic force involves an increased proliferation and differentiation of the cells of the periodontal ligament into osteoblasts.[2] GCF is an exudate, the constituents of which are derived from a variety of sources, including microbial dental plaque, host inflammatory cells, host tissue, and serum. In recent years, a number of GCF constituents have been shown to be diagnostic markers of active tissue destruction in periodontal diseases,[9,10] but only a few studies have focused on the GCF constituents involved in bone remodeling during orthodontic tooth movement. Uematsu et al[8] found that during orthodontic treatment, the levels of different inflammatory mediators in GCF, ie, interleukin 1β, interleukin 6, tumor necrosis factor-α, epidermal growth factor, and β_2 microglobulin,

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underwent significant elevations. Grieve et al[6] found similar results for prostaglandin E and interleukin 1β. Lowney et al[7] described an increase in tumor necrosis factor-α in GCF from teeth undergoing orthodontic force. Griffiths et al [11] reported an elevation of osteocalcin and Piridinium cross-links of bone collagen in GCF from orthodontically treated teeth. Bone turnover during orthodontic tooth movement has been described as a continual and balanced process characterized by bone deposition at sites of tension and bone resorption on the pressure sites.[12-15] Bone-forming cells have been shown to have alkaline phosphatase (ALP) activity,[16] and changes in this enzyme in serum and bone have been used as markers for bone metabolism in several diseases.[17,18] During orthodontic treatment, acid and alkaline phosphatase in human GCF have been correlated with the total appliance duration.[19] This study, however, was conducted on the basis of a cross-sectional design without controls. Insoft et al[19] also described the activity of both phosphatases on a longitudinal basis, but only for 3 cases. The present longitudinal study describes GCF ALP activity during orthodontic treatment in human subjects as it relates to the time of treatment and the type of stress exerted on the periodontium (tension or compression) by the tooth movement. Therefore it extends previous reports aimed at monitoring ALP activity temporally and spatially during orthodontic treatment in human subjects.

MATERIAL AND METHODS

Sixteen orthodontic patients, 10 females and 6 males (age range, 11-21 years; mean, 15.5 ± 3.5 years), were included in the study. The following inclusion criteria were observed: the need for fixed appliance therapy involving distal retraction of 1 maxillary first molar; good general health; no use of anti-inflammatory drugs during the month preceding the study[6,8]; probing depth values not exceeding 3 mm in the whole dentition; no radiographic evidence of periodontal bone loss; and a full-mouth plaque score and a full-mouth bleeding score less than or equal to 20%. Full-mouth plaque score and full-mouth bleeding score were recorded as the percentage of tooth surfaces with the presence of supragingival plaque or bleeding within 15 seconds after probing with a 20-g controlled force probe (Vivacare TPS Probe, Vivadent; Schaan, Lichtenstein).

During the 2 months preceding the baseline examination, all subjects received repeated oral hygiene instructions for the use of toothbrush, dental floss, and interdental brush. Before the study began, informed consent was obtained from the patients and from the parents of minor patients, and the protocol was reviewed and approved by the Ethical Committee of the G. D'Annunzio University Medical Faculty.

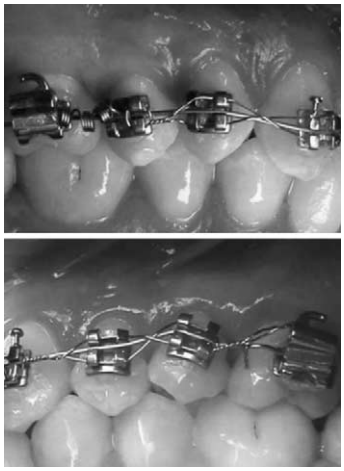

Fig 1. Orthodontic appliance. DM (*upper*) and CM (*lower*); see text for details.

In each patient, the maxillary first molars undergoing orthodontic treatment were used as the test teeth (TT). One of the TT molars, the distalized molar (DM) was to be treated for distal movement whereas its contralateral molar (CM) was not moved distally. The DM antagonist first molar (AM) was used as a control. Orthodontic brackets (MBT, 3M-Unitek; Monrovia, Calif) were placed on the buccal surfaces of the teeth in the maxillary arch, including incisors, canines, and premolars; tubes were bonded on both the DM and the CM. A bilateral 0.018-inch circular cross-sectional dimension nickel-titanium wire (MBT, 3M-Unitek) was used to activate the orthodontic appliance. In addition, teeth from the CM to the contralateral second premolar were laced together with a continuous stainless steel wire (0.010-inch) ligature according to Samuels et al.[20] At the same time, a nickel-titanium open coil spring (American Orthodontics; Sheboygan, Wis), exerting a constant force (250 g) over its range of activation, was included in the appliance to retract the DM.[21] The coil spring (Fig 1) was applied as previously reported[21] but with the following modifications: it was compressed between the second premolar bracket and the TT tube, and its length was set as twice the distance between the midbuccal axis of the second premolar and the mesial aspect of the DM tube. Furthermore, to avoid any unwanted dental displacements, the anterior teeth were rigidly splinted by direct proximal bonding with composite material. The entire orthodontic appliance and rigid splint were placed in a single clinical session. No orthodontic appliance was placed on the mandibular arch. At the mesial and distal aspects of the DM, CM,

and AM, GCF was collected for the ALP activity assay. In each sampling site, the presence or absence of dental plaque (PL), the probing depth (PD), and the presence or absence of bleeding on probing (BoP) were assessed as clinical monitoring. GCF was collected immediately before the appliance placement and activation, as described above, and at 1 hour and at 1, 2, 3, and 4 weeks after placement.

Clinical examination consisted of assessing the PL visually and assessing BoP within 15 seconds after probing with a 20-g controlled force probe and the PD in 6 sites per tooth (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, and distolingual/palatal). Clinical data were always collected by the same operator (D.D'A.). Contamination of the GCF samples was minimized by recording the plaque scores before carefully cleaning the tooth with cotton pellets, collecting GCF from the isolated area, and recording the PD and BoP as previously described by Griffiths et al.[11] These clinical parameters were assessed twice, at baseline (before the orthodontic appliance was placed) and on day 28.

Alginate impressions were taken for study models by the same investigator (G.P.) before orthodontic appliance placement and at the end of the experimental term. Measurements were made on stone models. The distances between the distal aspects of the canines, left or right, and the mesial aspects of each corresponding experimental tooth were recorded at baseline and on day 28; we used calipers capable of measuring to the nearest 0.05 mm. The total amount of displacement for each category was determined by subtracting the first recording from the second.

Each crevicular site included in the study was isolated with cotton rolls. Before the GCF collection, any supragingival plaque was removed with cotton pellets,[11] and a gentle air stream was directed toward the tooth surface for 5 seconds to dry the area. GCF was collected with no. 30 standardized sterile paper strips (Inline; Torino, Italy) inserted 1 mm into the gingival crevice and left in situ for 30 seconds. Care was taken to avoid mechanical injury. Immediately after collection, paper points were transferred to plastic vials. GCF total volume was determined for each sample as previously described.[22]

ALP activity was assayed spectrophotometrically[23] with a spectrophotometer at 405 nm (model 8453, Hewlett Packard; Waldgrohn, Germany). The cone sample was incubated at 30°C, with less than 0.05° C fluctuation, for 20 minutes in a substrate containing *p*-nitrophenyl phosphate (10 mmol/L), carbonate buffer (pH 10.2 $\pm$ 0.1 at 30°C), mannitol (200 mmol/L), and $MgCl_2$ (3 mmol/L), to a total volume of 1.0 mL. ALP hydrolyses *p*-nitrophenyl phosphate to *p*-nitrophenol and inorganic phosphate. The rate of increase in absorbance at 405 nm was monitored as the *p*-nitrophenol formed. We used 18.45 as the *p*-nitrophenol millimolar absorptivity and converted the absorbance into enzyme activity units (1 U = 1 μmol of *p*-nitrophenol released per minute at 30°C). Final results were reported as total ALP activity (mUnits/sample).

The overall percentages of tooth sites positive for plaque (%PL+) and bleeding on probing (%BoP+) and the mean PD were calculated from the %PL+, %BoP+, and mean PD of each DM, CM, and AM at baseline and on day 28. The %PL+ and %BoP+ were considered to be ordinal data; therefore, Friedman's test[24] was used to evaluate the statistical significance of the differences among the clinical data from the experimental categories at baseline and on day 28. When significant interactions were found, a Wilcoxon paired signed rank test[24] was performed. Changes in %PL+ and %BoP+ within the experimental groups were similarly tested by Wilcoxon paired signed rank test as a post hoc procedure. The statistical significance of the differences in PD of the experimental categories at baseline and on day 28 was evaluated with a 1-way repeated measures ANOVA[24]; when significant interactions were found, a Bonferroni-corrected paired Student *t* test[24] was performed for pair-wise comparisons. Changes in PD within the experimental groups were tested by paired Student *t* tests as a post hoc procedure. When appropriate, to statistically assess differences in clinical conditions between mesial and distal aspects of the same experimental tooth at the same experimental session, data obtained from the site corresponding to the GCF sampling area were tested. PL and BoP were processed as dichotomous data with a McNemar test, whereas the PD scores were processed with a paired Student *t* test. The measurements of GCF volume were recorded for the DM, CM, and AM categories at each sampling time and were expressed as a single score for each experimental group throughout the study; 1-way repeated measures ANOVA and Bonferroni-corrected paired Student *t* tests were used to examine the significance of differences in GCF volume among the experimental categories. The means and SDs of measurements for ALP activity values were calculated and arranged in a $2 \times 6 \times 3$ matrix, reflecting the sampling site (mesial or distal), the time points, and the 3 treatments. These 3 factors were used in a repeated measures 3-way ANOVA[25] to assess the data of GCF ALP activity. Furthermore, to test the simple main effect of each factor, 1-way repeated measures ANOVAs were performed to evaluate the significance of differences in ALP activity among the experimental

Table I. Clinical parameter changes in different experimental groups in relation to total appliance duration

Site	*Time*	*DMs*	*CMs*	*AMs*	*Difference among groups*
%PL+	Baseline	14.8 ± 10.3	15.8 ± 9.5	11.6 ± 11.7	NS
	28 d	28.1 ± 11.5*	28.1 ± 14.4*	13.7 ± 10.9	$P < .05$
	Wilcoxon test	$P < .01$	$P < .05$	NS	
%BoP+	Baseline	15.8 ± 9.5	15.8 ± 11.3	14.7 ± 11.9	NS
	28 d	27.1 ± 14.6*	27.1 ± 13.3*	12.7 ± 9.7	$P < .05$
	Wilcoxon test	$P < .05$	$P < .05$	NS	
PD (mm)	Baseline	1.7 ± 0.6	1.4 ± 0.7	1.6 ± 0.7	NS
	28 d	2.4 ± 0.9*	2.2 ± 0.8*	1.5 ± 0.7	$P < .01$
	Paired Student t test	$P < .05$	$P < .01$	NS	

Data presented as means ± SDs of total activity (n = 16). Results of pair-wise comparisons among groups at each location for each sampling time: *different from AMs.
NS, No statistically significant difference.

Table II. Clinical parameter scores of different experimental sites in DM and CM groups on day 28

Parameter	*Site*	*DMs*	*CMs*
PL+	Mesial	4	5
(No. positive sites)	Distal	8	7
	McNemar test	NS	NS
BoP+	Mesial	6	7
(No. positive sites)	Distal	5	8
	McNemar test	NS	NS
PD (mm)	Mesial	1.9 ± 0.9	2.3 ± 0.6
	Distal	2.2 ± 0.7	2.5 ± 0.8
	Paired Student t test	NS	NS

NS, No statistically significant difference.

groups at each time point and across times within each group in both mesial and distal sites. Bonferroni-corrected paired Student *t* tests were used as a pair-wise comparisons procedure when appropriate. The significance of differences in ALP activities between mesial and distal sites for the DM, CM, and AM categories at each time point were assessed with a paired Student *t* test as a post hoc procedure. A *P* value less than .05 was accepted as being statistically significant.

RESULTS

The DMs underwent a mean distal movement of 1.7 ± 0.3 mm. No clinically detectable movements in CMs and AMs were observed. As shown in Table I; the clinical parameters had similar scores in all 3 experimental groups at baseline, without any statistically significant differences. On day 28, all clinical parameters of the DMs and CMs were significantly worse than at the baseline; conversely, in the AMs, the parameters did not show significant changes. At this time in the experiment, the cross-sectional analysis also showed a significant difference among the clinical parameters from the 3 groups (Table I, final column). The pair-wise comparisons tests show that the significance is due to the differences in the clinical data from the AMs compared with those from the DMs and CMs. Within these DM and CM group parameters there were no statistically significant differences between the mesial and distal sites (Table II). The means and SDs of GCF volume in microliters for each experimental group were 0.15 ± 0.08 in the DMs, 0.15 ± 0.07 in the CMs, and 0.13 ± 0.07 in the AMs, with a significant difference among the groups (1-way ANOVA; $P < .01$). GCF volume was significantly greater in the DMs and CMs compared with the AMs ($P < .01$), but there was no statistical difference between GCF volumes from the DMs and the CMs ($P > .1$).

The 3-way repeated measures ANOVA reveals that the subjects demonstrated significant differences in GCF ALP activity levels among the time points (F-ratio = 13.23; $P < .01$), the treatments (F-ratio = 37.07; $P < .01$), and the sites (F-ratio = 11.46; $P = .01$). In addition, the interactions of treatments with times (F-ratio = 7.83; $P < .01$) and with sites (F-ratio = 6.43; $P = .02$) were also significant.

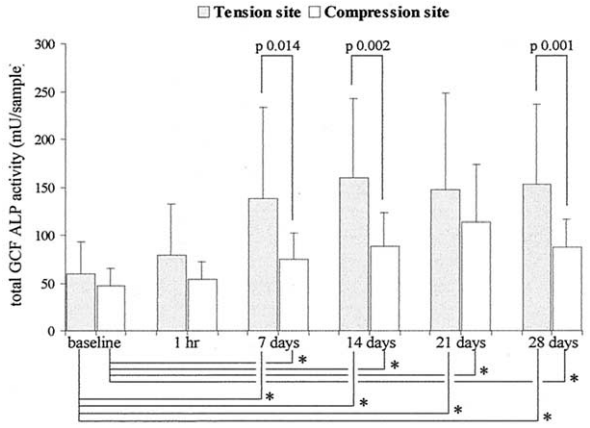

Fig 2. ALP activity in GCF from tension (mesial) and compression (distal) sites of DMs in relation to total appliance duration. Data presented as mean ± SD of total activity (n = 16). Level of signiifcance for pair-wise comparison at each sampling time compared with baseline: $*P < .05$.

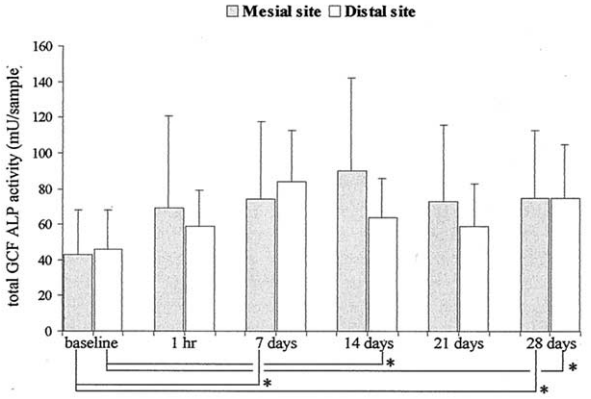

Fig 3. ALP activity in GCF from mesial and distal sites of CMs in relation to total appliance duration. Data presented as mean ± SD of total activity (n = 16). Level of significance for pair-wise comparison at each sampling time compared with baseline: $*P < .05$.

One-way ANOVA showed a statistically significant change in enzyme activity only in the DM and CM groups, in both mesial and distal sites, among the repeated samplings during the study period (Figs 2–4; Table III). Results of pair-wise comparisons show a significantly greater enzymatic activity in both mesial and distal sites from the DM group from day 7 to the end of the experiment, as compared with the baseline (Fig 2). Conversely, in the CM group, over the study period a significant statistical difference in ALP activities was seen on days 14 and 28 in mesial sites and on days 7 and 28 in distal ones, as compared with the baseline (Fig 3).

At baseline and at 1 hour, in both distal and mesial sites, ALP activity was similar among the 3 groups, without significant differences (1-way ANOVA). At each later sampling time, in mesial and distal sites, statistically significant differences among the groups were seen (1-way ANOVA). In mesial sites, pair-wise comparisons show an enzymatic activity significantly greater in the DMs than in the CMs and AMs from days 7 to 28. In the same sites, GCF ALP activity from the CMs was significantly greater than in the AMs from days 7 to 21. In distal sites, significant differences between the DMs and CMs were seen only on days 14 and 21, whereas the differences between the DMs and AMs were significant from days 7 to 28. In distal sites, statistically significant differences between the CMs and AMs were seen from day 7 to the end of the experiment. Statistically significant differences in GCF ALP activity between the mesial and distal sites were seen only in the DM group (Fig 2) on days 7, 14, and 28, being always greater in the tension sites (mesial).

DISCUSSION

The aim of this study was to investigate the GCF ALP activity longitudinally during orthodontic treatment in relation to the time of treatment and the type of stress exerted on the periodontium by the tooth movement. Our results show that GCF ALP activity was significantly different among the DMs, CMs, and AMs throughout the analysis; furthermore, the enzymatic activity was dependent on the orthodontic treatment phase. Bone remodeling produced as a consequence of orthodontic tooth movement has been described as a continuous phenomenon leading to bone resorption in pressure sites and bone deposition in tension sites.[12-15] However, subsequent studies[26,27] on rats showed that the remodeling process might be more complex; in fact, it has been histologically observed that bone deposition and resorption take place in tension and compression sites in the alveolar bone. In particular, it has been shown that in the early phases of bone remodeling, a resorption activity (3-5 days) is followed by its reversal (5-7 days) and, subsequently, by a late phase of bone deposition (7-14 days) in both tension and pressure sites of the alveolar wall. In the early phase of tooth movement, bone resorption is greater than bone deposition, but in a later phase, resorption and deposition can become synchronous. This might be due to the high acid phosphatase activity that has been observed in the early period of tooth movement; high levels of ALP activity have been described after 7 days, when bone deposition begins.[27] ALP is considered to be a marker

Table III. ALP changes in GCF of different groups in relation to total appliance duration

Site	*Time*	*DMs*	*CMs*	*AMs*	*ANOVA*
Mesial	Baseline	60 ± 33	43 ± 25	50 ± 24	NS
	1 h	79 ± 53	69 ± 52	46 ± 30	NS
	7 d	138 ± 95*†	74 ± 44*	45 ± 18	$P < .01$
	14 d	160 ± 82*†	90 ± 52*	50 ± 15	$P < .01$
	21 d	147 ± 101*†	73 ± 43*	37 ± 21	$P < .01$
	28 d	153 ± 84*†	75 ± 38	46 ± 23	$P < .01$
	ANOVA	$P < .01$, Note 1	$P < .05$, Note 2	NS	
Distal	Baseline	47 ± 19	46 ± 22	44 ± 33	NS
	1 h	54 ± 18	59 ± 20	47 ± 15	NS
	7 d	75 ± 27*	84 ± 29*	44 ± 21	$P < .01$
	14 d	88 ± 35*†	64 ± 22*	41 ± 25	$P < .01$
	21 d	114 ± 60*†	59 ± 24*	35 ± 14	$P < .01$
	28 d	87 ± 29*	75 ± 30*	45 ± 27	$P < .01$
	ANOVA	$P < .01$, Note 1	$P < .01$, Note 3	NS	

Data are presented as means ± SDs of total activity (mU/sample) (n = 16). Results of pair-wise comparisons among groups at each location for each sampling time: *different from AMs, † different from CMs. Notes on results of pair-wise comparisons among sampling times at each location for each group where ANOVA shows $P < .05$: 1, baseline vs 7, 14, 21, and 28; 2, baseline vs 14 and 28; and 3, baseline vs 7 and 28.
NS, No statistically significant difference.

of osteoblastic activity[28-30] because this enzyme is essential for bone deposition[29]; it hydrolyzes nonorganic pyrophosphate, which is a potent inhibitor of the mineralization process.[31] Different results have been reported in studies investigating ALP activity in the periodontium of teeth undergoing orthodontic treatment. Takimoto et al[32] reported an increased ALP activity in the periodontal ligament of rats; other authors have observed in rats that a high ALP activity occurs in tension sites whereas enzymatic activity decreases in pressure sites.[33,34] Conversely, in vitro studies on human periodontal ligament cells undergoing tensional stress show decreased ALP activity.[3] Other researchers[4,5] have observed both increases and decreases in ALP activity in animal periodontal ligament cells, depending on whether compressive forces were intermittent or continuous. Finally, it has been observed that ALP levels in rats remain elevated after force decay.[35] Despite these studies, a longitudinal investigation of ALP activity in human GCF is still needed. In the present human longitudinal study, we used a double test (DMs and CMs) for comparison with the control AM group. This allowed us to observe the GCF ALP activity behavior in different situations, such as when the teeth have an orthodontic appliance exerting a mechanical stress on the periodontium with (DMs) and without (CMs) a clinically detectable displacement and when the teeth do not undergo an intentional mechanical stress nor have an orthodontic appliance (AMs) that might interfere with the gingival condition. GCF is an inflammatory exudate found in the gingival crevice. The amount of such fluid, which increases with inflammation and capillary permeability, reflects the periodontal health.[36-39] Serum, gingival tissue through which the fluid passes, and bacteria in the tissue and the crevice can modify the GCF.[36-39] Our results show that GCF volumes are influenced by the presence of the orthodontic appliance independently of the existence of a clinically detectable dental movement. GCF volumes from DMs and CMs were not significantly different; on the contrary, both were higher than GCF volumes from the AMs, at a statistically significant level ($P < .01$). This observation is in accordance with previous studies that correlate the GCF volume with orthodontic treatment.[40] Indeed, the CMs showed increased gingival inflammation similar to the DMs (Table I), perhaps as a consequence of the orthodontic appliance's presence[41]; conversely, the gingival condition of the AM sites did not worsen. These data reinforce the view that GCF measurements represent a more sensitive and less subjective tool of assessing gingival inflammation.[11] GCF ALP activity is presented as the total activity per sample rather than the final concentration, because small errors in volume determination can lead to large errors in estimates of final concentrations if the total volumes collected are small[42]; moreover, an increase in GCF volume can significantly dilute its contents.[11] GCF ALP activity increases in the DMs, with respect to the baseline, are seen in tension and compression sites from day 7 to day 28 (Fig 2 and Table III). This finding might be explained by histomorphometric studies that have evaluated the bone cycle in humans[29,43,44]; the activation phase is sudden and is followed by osteoclastic resorp-

tion, which lasts for 10 days[29] or 3 weeks.[43,44] Subsequently, osteoblasts lay down the osteoid matrix, which fills the lacunae in 80 to 120 days.[29,43] This explains why ALP values increased for the entire length of the study. It is more difficult to explain increased ALP activity at 7 days; this is early for osteoblastic activation as described in the literature.[29,43,44] However, the bone cycle timing might be influenced by the magnitude of the force.[27] The force exerted by the coil spring might have affected the osteoclastic activation. Furthermore, increased dental plaque in the DMs and CMs, possibly caused by patients' difficulty when cleaning around appliances,[11,46] produced a clinically relevant gingival inflammation that might have contributed to the increased ALP activity in GCF.[19,45-48] Moreover, it must be noted that Insoft et al,[19] reporting on 3 patients who were longitudinally observed for 4 to 6 weeks, described the existence of an ALP activity peak at 7 days in 1 of them. Because no significant differences in clinical conditions were detected between mesial and distal sites in the DMs, the greater ALP activity values reported for the mesial (tension) sites as compared with the distal (compression) sites on days 7, 14, and 28 (Fig 2) might be considered a consequence of the prevalence of the bone deposition process over the resorption process.[29] Our data, which are consistent with those of other studies,[19,27,32,34] suggest that the increase in ALP activity in GCF might be related to dental site bone remodeling that is a consequence of the orthodontic forces. The ALP activity in the CMs was often significantly greater than in the AMs (Table III). This might depend on the existence of limited forces exerted by the wire, although no mesiodistal movement was detected in the CM group; several subclinical displacements and movements not measured, such as intrusion, extrusion, and rotation, could be present, leading to an increase in GCF ALP activity resulting from the periodontal ligament (PDL) alteration. It has been observed that the cells within the PDL can release ALP if mechanically stressed.[3-5] Considering that the clinical parameter scores of the CMs had worsened on day 28 compared with those at baseline (Table I), it is also probable that the enzymatic increase (Fig 3) was due to gingival inflammation, according to the correlation between GCF ALP activity and clinical conditions previously described.[19,45-48] On the basis of these observations, we hypothesize that the increased GCF ALP activity in the DMs and CMs was produced by a combination of gingival inflammation and mechanical stress; however, whereas the clinical parameter scores were similar, the clinically detectable movements in the DM group might have caused the greater enzymatic activity noted in this category as compared with the CMs. These results underline the properties of the GCF ALP activity in distinguishing between clinically moving and nonmoving teeth and show that this enzyme should be further studied as a diagnostic tool in orthodontics. Conversely, in the AMs, where no tooth movement occurred and no orthodontic appliances (that could interfere with gingival health) were placed, ALP activity remained stable throughout the study (Fig 4 and Table III). Our results suggest that ALP activity in GCF is affected by orthodontic forces that cause bone remodeling; activity values also might be influenced by factors other than mechanical stress, eg, gingival inflammation, as suggested by the results from the CMs. However, when gingival inflammation is kept under control, ALP activity in GCF can be considered a suitable indicator of the biologic effects produced by orthodontic treatment.

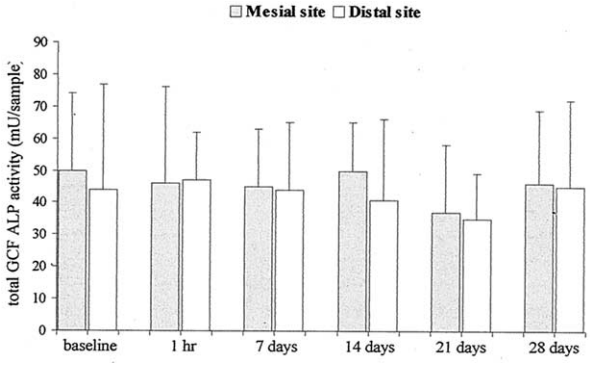

Fig 4. ALP activity in GCF from mesial and distal sites of AMs in relation to total appliance duration. Data presented as mean ± SD of total activity (n = 16).

CONCLUSIONS

ALP activity in GCF might be affected by forces deriving from the orthodontic treatment, and its levels are significantly greater in dental sites of tension than in sites of compression. Furthermore, increased ALP activity in teeth bearing orthodontic appliances might be due in part to gingival inflammation produced by plaque-retentive appliances, independently of clinically detectable dental displacements.

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