Lack of Significance of Increased Tooth Mobility in Experimental Periodontitis*

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This experiment was carried out in order to study the rate of progression of experimentally produced periodontal tissue breakdown in teeth which prior to the placement of cotton floss ligatures either had a normal or a permanently increased tooth mobility. Six beagle dogs were used. Trauma from occlusion of the jiggling type was produced on test teeth using a model previously described. Four months later experimental periodontal tissue breakdown was induced around both control and test teeth by placing cotton floss ligatures around the necks of the teeth. The ligatures which were exchanged once every 4 weeks during a 4-month period were replaced at the level of the gingival margin. Tooth mobility measurements and radiographic examinations were performed on Days 0, 30, 60, 90, 120, 160, 240 and 300. One dog was killed on Day 120 and the remaining 5 dogs on Day 300. Biopsies of the test and control teeth were fixed in formalin, decalcified, embedded in paraffin and mesiodistal sections cut with the microtome set at 4 µm. The results demonstrated that the degree of periodontal breakdown, initiated and maintained by ligature placement and plaque accumulation, was similar around teeth with a wide periodontal ligament space and in teeth with a normal width of the periodontium. In other words, progression of the plaque-associated lesions appeared to be unrelated to the width of the periodontal ligament space, i.e., to the degree of horizontal tooth mobility.

Data previously reported from experiments in beagle dogs have demonstrated that alterations in tooth mobility produced by jiggling forces involve one phase of developing (progressive) followed by one phase of established (permanent) tooth hypermobility.1-5 In teeth with normal height of the supporting tissues such mobility changes are associated with loss of alveolar bone resulting in a widening of the periodontal ligament space but not with loss of connective tissue attachment.6-9 Such a reduction of the height of the attachment apparatus is accompanied by a gradual, apical displacement of the fulcrum for the movement of the crown of the tooth. This in turn, results in a progressive increase of the horizontal tooth mobility.10 In the same animal model, the rate of periodontal breakdown may be enhanced in teeth on which jiggling trauma is superimposed on the experimentally induced periodontal disease.11-13 Findings presented from clinical trials14 and animal experiments10 indicate that healing following treatment of periodontitis seems to be more favorable in teeth with normal mobility than in teeth with a permanently increased mobility. The above data suggest that increased tooth mobility influences not only the progression of plaque-associated periodontal disease but also healing following treatment. Such an assumption, however, appears to be in variance with data reported by Meitner15 from experiments in the monkey and trials in humans by Rosling et al.16 and Polson & Heijl.17

The present experiment in the beagle dog was carried out in order to study the rate of progression of experimentally produced periodontal breakdown in teeth which, prior to the placement of cotton floss ligatures, either had a normal or a permanently increased tooth mobility.

MATERIALS AND METHODS

The outline of the experiment is delineated in Figure 1. Six inbred beagle dogs were used. They were 2 years old and had been inoculated against distemper, canine hepatitis and Parvo virus enteritis. During a preparatory period of 6 weeks, the animals were subjected to a careful plaque control program consisting of scaling, polishing with rubber cup and pumice once weekly and

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Figure 1. Outline of the experiment. On Day 0 trauma from occlusion was inflicted on P4 (test). On Day 120 periodontal breakdown was induced by ligatures placed around P4 (control) and P4 (test) to act as a plaque collector between Day 120 and Day 240. Tooth mobility measurements and radiographic examinations were carried out on Days 0, 30, 60, 90, 120, 160, 240 and 300. One dog was killed on Day 120 and the remaining five on Day 300.

tooth brushing twice daily. A clinical and radiographical examination on Day 0 revealed that the gingiva in the lower premolar region of all dogs was healthy and that the supporting tissues in this part of the dentition had a normal height. In addition, the mobility of $\text{P}_4$ (control tooth) and $\text{P}_4$ (test tooth) was measured using the Periodontometer described by Mühlemann.\textsuperscript{15} $T_{500}$ values were recorded.

On experimental Day 0 (Fig. 1) trauma from occlusion of the jigging type was inflicted on $\text{P}_4$ (test) by the installation of a cap splint cemented to the canine and the premolars in the left (test) side of the maxilla. The cap splint was designed with an oblique plane\textsuperscript{1} which made “primary” contact with $\text{P}_4$. Following the installation of the cap splint, the incisors did not reach contact in occlusion. This meant that the mandible was moved towards centric occlusion. $\text{P}_4$ was subjected to an excessive force and tilted in a mesiobuccal direction. A lingual bar was fitted to the canine and the first molar of the left mandibular jaw. A spring (hard wire: $\varnothing = 0.6$ mm, Remanite\textsuperscript{1}, Dentaurum Ltd.) was attached to the bar and introduced through a channel—prepared in a buccal-lingual direction—in the crown of $\text{P}_4$. Each time the animal discolocated, the spring pulled $\text{P}_4$ back to its original position. Tooth mobility measurements and radiographic examinations were repeated on experimental Days 30, 60, 90, 120, 160, 240 and 300. From experimental Day 90 and onward, in each dog the deflection of the crown of $\text{P}_4$ allowed the incisors to reach contact in occlusion.

Throughout the entire period of observation, the animals were fed a diet which permitted gross accumulation of dental plaque and calculus.\textsuperscript{19,20} Between Day 0 and Day 120, plaque control measures were performed twice daily while during the remaining part of the experiment, i.e., between Day 120 and Day 300, the dogs were allowed to accumulate plaque and calculus.

Subsequent to the clinical examination on experimental Day 120 a phase of periodontal tissue breakdown was induced around $\text{P}_4$ (control) and $\text{P}_4$ (test) by placing cotton floss ligatures around the necks of the teeth. The ligatures which were exchanged once every 4 weeks during a 4-month period were replaced at the level of the residual gingival margin. This method of producing breakdown of the supporting tissues of premolars was originally described in the monkey model by Kennedy & Polson\textsuperscript{21} but has been modified for use in dogs by Ericsson et al.\textsuperscript{6} and Lindhe & Ericsson.\textsuperscript{7} The plaque-collecting, cotton floss ligatures were removed on Day 240.

One dog was killed on Day 120 and the remaining five dogs on Day 300. The mandibles were dissected and divided along the midline. Specimens containing the distal portion of the third premolar, the fourth premolar and the mesial portion of the first molar ($\text{I}_4$, $\text{M}_3$, $\text{M}_2$ (control) and $\text{I}_4$, $\text{M}_3$, $\text{M}_2$ (test)]) were dissected, fixed in formalin, decalcified and embedded in paraffin. Mesio-distal sections were cut at 4 $\mu$m and stained in hematoxylin-eosin. From each biopsy five sections, 20 $\mu$m apart, were magnified and their images depicted onto white paper. In the drawings the following linear distances on the mesial surface of the mesial root and on the distal surface of the distal root of $\text{P}_4$ (control) and $\text{P}_4$ (test) were measured:

1. The width of the marginal periodontal ligament (1) at the level of the bone crest (BC) and (2) at a point located midway between BC and apex.
2. The cementoenamel junction (CEJ) to the most apical cells of the dentogingival epithelium (JE), CEJ-JE.
3. CEJ to the apex of the root, CEJ-apex. The loss of connective tissue attachment was expressed as the quotient (CEJ-JE/CEJ-apex) $\times 100$ ($\%$).

In addition, the apical position of the infiltrated gingival connective tissue (ICT) in relation to the apical termination of the dentogingival epithelium was determined. Differences between the test and control teeth regard-
ing the various parameters were analyzed using Student’s paired t test.

RESULTS

Clinical and Radiographical Observations. At the start of the experiment all six dogs were free from signs of gingival inflammation in the premolar regions of the mandible. The condition of the gingival units remained unchanged until experimental Day 120, when the cotton floss ligatures were applied around the test and control teeth and the plaque accumulation period was initiated (Fig. 1). Clinical examination and photographs obtained on experimental days 160, 240 and 300 revealed that the gingiva of the test and control teeth was red and edematous. On Day 300 heavy amounts of plaque were present on all test and control teeth and, furthermore, all gingival units studied displayed pronounced signs of inflammation as well as some recession of the gingival margin (Fig. 2).

The tooth mobility data (T500) are reported in Figure 3. The bars reveal that the test and control teeth on Day 0 had a T500 value of, on the average, 5.3 mm/100. The installation of the cap splint and bar devices in the left jaws on Day 0 resulted in an increased horizontal tooth mobility of P4 (test). During the first 3 months of experimentation this mobility gradually increased, i.e., was progressive (Day 0: 5.3 mm/100 ± 0.5 [SE]; Day 90: 13.8 mm/100 ± 4.1 [SE]). Between Day 90 and 120 the T500 value, however, did not further increase. The mobility of the control teeth remained unchanged between Day 0 and Day 120. Following ligature application on Day 120, the T500 values again gradually increased, not only in the test but also in the control teeth. On Day 240 the mean T500 value of the test teeth was 40.6 mm/100 ± 4.4 (SE). The corresponding value from the control teeth was 25.6 mm/100 ± 6.0 (SE). This difference of the T500 values was statistically significant (P < 0.01) while the increase in mobility between Day 120 and Day 240 was similar in the test and control tooth regions. Sixty days after ligature removal the tooth mobility of the test and the control teeth were 39.2 mm/100 ± 4.2 (SE) and 24.7 mm/100 ± 4.5 (SE), respectively (P < 0.01).

The radiographs obtained on experimental Day 120 revealed that the supporting apparatus of the test and control teeth had a normal height but also that the periodontal ligament spaces of the test teeth (P4) were markedly widened (Fig. 4). The radiographic examination of P and P4 performed at the end of the experiment not only showed that about 25% of the supporting alveolar bone was lost in both tooth regions, but also that in all test teeth the periodontal ligament spaces were markedly widened (Fig. 5).

Histometric assessments. The analysis of the histologic sections from the dog killed on experimental Day 120 disclosed that the apical termination of the junctional epithelium of the test (P4) as well as of the control tooth (4P) was located at the cementoenamel junction (CEJ).

The results of the histometric assessments made in sections representing Day 300 are presented in Table 1. The average width of the marginal periodontal ligament at the level of the bone crest was in the test teeth 642.5 μm ± 73 (SE) and in the control teeth 187.5 μm ± 23 (SE). This difference was statistically significant (P < 0.001). At a level located midway between the bone crest and the apex of the root, the average width of the periodontal ligament in the test tooth regions was 247.8 μm ± 27 (SE). The corresponding value in the control tooth regions was 138.8 μm ± 21 (SE) (P < 0.01).

In all the test and control biopsies, an infiltrated gingival connective tissue portion (ICT) was present along the entire length of the dentogingival epithelium (Figs. 6 and 7). In eight out of the 10 biopsies of both the test and control teeth, the ICT portion appeared to be in contact with the bone crest.

DISCUSSION

The results of the experiment demonstrated that the degree of periodontal breakdown, initiated and maintained by ligature placement and plaque accumulation, was similar in teeth with a widened periodontal ligament space and in teeth with a normal width of the periodontium. In other words, progression of the plaque-associated lesion in the attachment apparatus appeared to be unrelated to the width of the periodontal ligament space.
Figure 3. Alterations of the average tooth mobility (T$_{500}$) of the test and control teeth calculated from measurements with the Periodontometer on Days 0, 30, 60, 90, 120, 160, 240 and 300 (mean ± SE).

In the experiment the test teeth were exposed to trauma from occlusion (jiggling) as well as “experimental periodontitis,” while in the controls jiggling was avoided. Trauma from occlusion was produced using a technique originally described by Svanberg & Lindhe. Thus, following the installation of a cap splint and a bar device on Day 0 (Fig. 1), during an initial 60- to 90-day period, the test teeth were exposed to forces which gradually increased the “horizontal” mobility of the teeth. This mobility increase (progressive mobility) was the result of a gradual widening of the periodontal ligament space without an accompanying reduction of the height of the supporting alveolar bone. Between Day 90 and Day 120, the mobility of the test teeth did not further increase but remained 2 to 3 times higher than that of the controls (Fig. 3). In all respects the
alterations of the mobility of the test teeth observed between Day 0 and Day 120 corresponded to the mobility data reported by Svanberg & Lindhe.\textsuperscript{1} A histologic analysis of biopsy material obtained from one dog on Day 120 revealed that in both the test and control tooth specimens, the apical cells of the junctional epithelium were located at the cementoenamel junction. This observation, which is in agreement with data previously reported,\textsuperscript{1,5} demonstrates that jiggling per se fails to interfere with the original height of the connective tissue attachment.

Following the installation of the ligatures (on Day

Table 1

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<thead>
<tr>
<th>Test</th>
<th>Control</th>
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<td></td>
<td>X</td>
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<tr>
<td>Width of the marginal periodontal ligament (\textmu m)</td>
<td>642.5</td>
</tr>
<tr>
<td>at the bone crest (BC)</td>
<td>247.8</td>
</tr>
<tr>
<td>midway between BC and apex</td>
<td>25.5</td>
</tr>
<tr>
<td>CEJ-JE \times 100 (%)</td>
<td>27.3</td>
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The average loss of connective tissue attachment (percentage of root length) X = mean value; SE = standard error.
\textsuperscript{+} NS, not significant.

Figure 7. A photomicrograph of a control tooth (P4) illustrating the infiltrated gingival connective tissue portion (arrows) present along the entire length of the dentogingival epithelium.

120; Fig. 1) and the termination of the plaque control program, both groups of teeth (test and controls) showed a gradual increase in the horizontal tooth mobility (Fig. 3). This mobility increase, which was similar in the test (26.9 mm/100) and control teeth (20.5 mm/100), appeared to be progressive throughout the entire phase of experimental periodontitis. A histologic analysis obtained from biopsies sampled at the end of the 120 days of experimental periodontitis revealed that around 25% of the height of the attachment apparatus in both test and control teeth had been destroyed (Table 1). This degree of attachment loss is comparable to data previously reported from similar experiments\textsuperscript{7} and indicates that the progressive increase of the T\textsubscript{500} values in the two groups of teeth (between Day 120 and Day 240) was mainly the result of an apical displacement of the alveolar bone margin and a corresponding apical shift of the fulcrum for the movement of the crown of the teeth (Fig. 3) in the tooth mobility measurements (for review see Lindhe et al.\textsuperscript{22}). Thus, even if the mobility of the test teeth at the end of the experiment was significantly higher than that of the controls, the degree of periodontal tissue destruction observed in the two categories of teeth was the same. This indicates that in a model experiment of the kind reported here, experimental periodontitis progresses at a rate which is independent of the mobility status of the teeth.

The results from this experiment are interesting in relation to data reported from one clinical trial\textsuperscript{14} and
one animal experiment. Fleszar et al.14 analyzed whether an increased tooth mobility influenced healing following treatment of periodontal disease in humans. In this study tooth mobility data were assessed prior to treatment and were subsequently related to changes in the attachment levels over an 8-year period of maintenance. Fleszar et al.14 stated “Pockets of clinically mobile teeth do not respond as well to periodontal treatment as do those of firm teeth exhibiting the same disease severity.” Similar findings were reported by Lindhe & Ericsson10 from an experiment involving five dogs. Periodontal breakdown was first produced around contralateral lower premolars by ligature placement and jiggling trauma using the model described by Lindhe & Svanberg.11 Subsequently, the periodontal lesions were treated with a surgical approach and from some teeth the jiggling trauma was also eliminated. During healing the dogs were placed on a careful plaque control regimen. Three months after active treatment biopsies were obtained from the teeth and analyzed in histologic sections. The results indicated that the teeth from which the trauma was removed seemed to have more favorable healing, i.e., gain of connective tissue attachment, than the contralateral, hypermobile teeth. Based on the findings from the present experiment and data reported by Fleszar et al.14 and Lindhe & Ericsson,10 it seems reasonable to suggest that while permanently increased (but not progressive) mobility of the teeth evidently has no influence on the development of periodontitis, healing following surgical treatment of periodontal disease may be more advantageous in nonmobile than in mobile teeth.

REFERENCES


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