The influence of trauma from occlusion on reduced but healthy periodontal tissues in dogs

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Abstract. The experiments were performed in five dogs fed a soft diet which allowed dental plaque accumulation. Experimental periodontal breakdown was introduced on Day 0. After 180 days trauma from occlusion was produced in 4P and 5P, i.e., the mandibular fourth premolars, in the manner described by Svanberg & Lindhe (1973). On Day 280 the periodontal pockets around 4P and 5P were eliminated. A notch was prepared in the root at the level of the bottom of the surgically eradicated pocket. In addition, the occlusal trauma in the 4P region was deleted. From Day 280 to Day 370 the teeth of the animals were brushed twice a day. The animals were then sacrificed, radiographs of the premolar regions were taken, and tissue sections comprising 3P, 4P, 5P (and 3P, 4P, 5P) were produced and subjected to microscopic analysis. The results indicate that jiggling type occlusal trauma and tooth hypermobility are not factors which detrimentally affect healing following periodontal surgery.

Results from analyses of human autopsy material have suggested that trauma from occlusion is a co-destructive factor in periodontitis (for review see Svanberg 1974a). Thus trauma produced by occlusal forces may alter the pattern of spread of the plaque-induced inflammation, thereby producing angular osseous defects and infrabony pockets (Glickman 1967). Experiments in monkeys and dogs with normal gingiva or overt gingivitis have revealed that trauma from occlusion of the jiggling type induces a series of adaptive alterations within the periodontal tissues (Wentz et al. 1958, Svanberg & Lindhe 1973, Svanberg 1974b). Under certain experimental conditions, jiggling forces produce one phase of increasing tooth mobility (the traumatic phase) and one phase of permanently increased tooth mobility (the post-traumatic phase). The traumatic phase is characterized by an osteoclastic alveolar bone resorption, gradually increasing width of the periodontal membrane, and by a periodontal ligament tissue in which there is an increased number of vessels exhibiting enhanced permeability to plasma and leukocytes. During the post-traumatic phase the vascularity (and permeability) of the enlarged periodontal membrane tissue is normal. Furthermore, there is no obvious retention of leukocytes within the periodontal ligament, nor are there signs of increased osteoclastic activity. During the post-traumatic phase the periodontal tissues become adapted to the altered functional demands (Svanberg & Lindhe 1973, Svanberg 1974b).
Lindhe & Svanberg (1974) have presented data indicating that the jiggling type occlusal trauma, if occurring concomitantly with an ongoing experimentally induced process of periodontal tissue breakdown, may cause: (1) an increased rate of apical downgrowth of pocket epithelium, and (2) infrabony pocket formation. In some aspects the results of this study support findings reported from examinations of human autopsy material (for review see Svanberg & Lindhe 1973). Thus, for example, Glickman & Smulow (1965, 1967) stated that the periodontal tissues at the pressure side of teeth subjected to periodontitis and trauma from occlusion could be consistently characterized by the presence of angular bony defects and infrabony pockets. This would imply (Glickman 1967) that, when the chronic inflammatory lesion produced by the bacterial plaque reaches the major supporting tissues, trauma from occlusion may become a co-destructive factor in periodontitis.

The aim of the present investigation was to study what remaining influence trauma from occlusion may have on the process of periodontal breakdown once the causative factors of infectious origin have been eliminated.

Material and Methods

The experiments were performed in five dogs (Beagles), which at the start of the study were 10–12 months old. During a pre-experimental period of several weeks, the teeth of the dogs were first carefully scaled and polished with rubber cups and pumice, and then subjected twice a day to meticulous toothbrushing. At the initiation of the experiment (Day 0), none of the dogs exhibited clinical signs of gingivitis. Throughout the study the animals were fed a diet which allows gross plaque formation (Hamp et al. 1973).

On Days 0, 180, 280 and 370 (Fig. 1) the periodontal tissues of the $P_1$- and $P_4$-regions were studied using the following criteria:

1: Gingival inflammation. The state of the gingiva was assessed according to the criteria of the Gingival Index system (Löe & Silness 1963). Separate scorings were made for the mesial, distal, buccal and lingual aspects of the gingiva, and a mean score for the tooth was then calculated. In addition, clinical colour photographs were taken.

2: Plaque. The amounts of plaque on the mesial, distal, buccal and lingual surfaces of the two teeth were estimated according to the Plaque Index system (Silness & Löe 1964). A mean score for each tooth was calculated.

3: Tooth mobility. T$_{500}$-values for $P_1$ and
were assessed in accordance with a method described by Mühlemann (1954).

4: Alveolar bone topography. Standardized roentgenograms of the marginal alveolar bone were produced in accordance with a modification of a method described by Eggen (1969) and Lindhe et al. (1973).

Immediately following the clinical examination on Day 0, inflammation was induced in the periodontal tissues around $\text{P}_4$ and $\text{P}_4$, according to a technique described by Ericsson et al. (1975). A scalpel was introduced 2 to 3 mm into the periodontal ligament around the entire root surface of the two teeth. Narrow, infrabony pockets of standardized depth (1 mm) were prepared on the mesial and distal aspects of $\text{P}_4$ and $\text{P}_4$. Approximately 1 mm of the marginal alveolar bone was then removed from the buccal and lingual areas of the same teeth. A copper band was cemented to $\text{P}_4$ and $\text{P}_4$ in order to prevent reattachment of the periodontal tissues. The copper bands were removed 21 days later, and a ligature of cotton floss was placed around the teeth at the level of the cemento-enamel junction.

Six months later (Fig. 1), trauma from occlusion was produced by the installation of cap splints and bar devices as described by Svanberg & Lindhe (1973). On both sides of the maxilla the animals were supplied with cap splints fitted with oblique planes which made primary contacts with the two lower fourth premolars (P and P4) before centric occlusion was established. Following installation of the cap splints, the incisors did not make contact in occlusion. Thus, when the mandible, after having made primary contact in the posterior premolar region, was moved further towards the centric occlusion, $\text{P}_4$ and $\text{P}_4$ were subjected to excessive horizontal forces and tilted in a mesial-buccal direction. On both sides of the mandible, the canines (C and C1) and the first molars (M and M1) were fitted with crowns and connected with a lingual bar. A spring (Dentaurum Ltd®, Germany: Spring-hard wire diam, 0.55 mm) was attached to each of the lingual bars, and was also introduced through channels (running in a buccal-lingual direction) in the crowns of $\text{P}_4$ and $\text{P}_4$. When the animal disoccluded the teeth, the springs pulled $\text{P}_4$ and $\text{P}_4$ back to their original positions. The masticatory movements resulted in jiggling forces acting upon the lower fourth premolars. It should be stressed that: (1) the anatomy of the temporomandibular joint of the dogs permits the animal to exercise hinge-axis movements only, and (2) the oblique planes of the cap splints were adjusted in such a way that they made simultaneous and even contact with $\text{P}_4$ and $\text{P}_4$.

On Day 280, when a severe periodontal lesion had been established (Ericsson et al. 1975), the oblique plane of the cap splint on the right side of the maxilla was removed. The premolars and first molars on both sides of the mandible (M, P, P, M and P, P, P, M) were then scaled and polished with rubber cups and pumice. The chronically inflamed periodontal tissues around $\text{P}_4$ and $\text{P}_4$ were eliminated using a surgical approach originally described by Widman (1918), the so-called modified Widman flap procedure (Ramfjord & Nissle 1974). During surgery a notch was carefully prepared in the root surface at the level of the bottom of the clinically detectable infrabony pocket. This notch served as a reference point for measurements to be made later on histological sections in the microscope. The flaps were sutured. The sutures were removed 2 weeks later.

From Day 280 to the termination of the experiment on Day 370 (Fig. 1) the teeth of the dogs were subjected to careful mechanical tooth cleaning twice a day. Particular care was taken to clean the interradicular area of $\text{P}_4$ and $\text{P}_4$. 
At the end of the experiment the dogs were sacrificed with an overdose of Pentothal (Pentothalsodium®, Abbott), the mandibles dissected and divided along the midline. Radiographs of the premolar region on both sides were taken on a skull-table.

Specimens containing the distal portion of the third premolar, the fourth premolar and the mesial portion of the first molar, were harvested, fixed in formalin, decalcified in a solution of equal volumes of 50% formic acid and 15% sodium fomate; they were then dehydrated and embedded in paraffin. Mesio-distal sections were cut with the microtome set at 7 μm and were stained in hematoxylin-eosin. From each biopsy five sections, 20 μm apart, were used for determining the following distances (Fig. 2) on the mesial aspect of $P_3$ and $P_4$:

1) The apical border of the notch (A) to the most apical cells of the junctional epithelium (JE);
2) The marginal bone crest (BC) to JE;
3) A to BC;
4) The length of the junctional epithelium (EA).

Using microscopic projection, the sections were magnified ($\times 98.4$) and their images depicted on a white paper screen. The size of the periodontal ligament area (PLA) on the mesial side of $P_3$ and $P_4$ was determined by the use of a planimeter (9527–12 Ingut® Polar Compensating Planimeter, Ingut Ltd., Sweden), as described in detail by Svanberg & Lindhe (1973). The total vascular cross section area (VCA) within the PLA was also assessed planimetrically. In addition, the value of VCA/PLA $\times 100$ for each specimen was calculated. The number of osteoclasts present on the alveolar bone surface bordering the PLA was determined.

**Results**

**Gingival Conditions and Amount of Plaque**

At the start of the study, the gingivae around the test and control teeth were normal and no plaque could be detected on the gingival third of the tooth surfaces (Table 1). Following termination of the
Table 1. The average values of GI, PII and tooth mobility (T₅₀₀) of the test and control teeth on Day 0, 180, 280 and 370.

Die durchschnittlichen Werte von GI, PII und Zahnmobilität (T₅₀₀) der Testzähne und der Kontrollzähne an den Tagen 0, 180, 280 und 370.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>GI</th>
<th>PII</th>
<th>T₅₀₀ (1/100 mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Days</td>
<td>Test</td>
<td>Control</td>
<td>Test</td>
</tr>
<tr>
<td>0</td>
<td>0.0</td>
<td>0.0</td>
<td>3.4</td>
</tr>
<tr>
<td>180</td>
<td>2.8</td>
<td>2.8</td>
<td>2.4</td>
</tr>
<tr>
<td>280</td>
<td>3.0</td>
<td>3.0</td>
<td>2.4</td>
</tr>
<tr>
<td>370</td>
<td>0.2</td>
<td>0.2</td>
<td>4.2</td>
</tr>
</tbody>
</table>

C = P < 0.001.

Tooth cleaning procedures and the application of cotton floss ligatures, large amounts of plaque rapidly accumulated in the posterior premolar regions. The gingival condition gradually deteriorated. On Days 180 and 280 the gingivae around 4P and P₄ exhibited clinical signs of severe chronic inflammation. Following scaling, periodontal pocket elimination and the reinstitution of daily tooth cleanings on Day 280, the clinical symptoms of gingivitis almost entirely disappeared in a very short period of time. Thus, on Day 370, only one dog exhibited minute signs of gingivitis in the 4P and P₄ region; a mean GI of 0.2 (test and control) was calculated.

Tooth Mobility

Immediately before the induction of periodontal inflammation, the T₅₀₀ values (Table 1) of the test and control teeth were 3.4 (± 0.5) and 3.1 (± 0.3). After 6 months of experimental periodontitis, the T₅₀₀ values exhibited a small but significant increase from 3.4 to 9.6 (test) and from 3.1 to 9.9 (control). After the installation of the cap splints and the bar devices there was a very pronounced increase of the mobility of 4P and P₄ (Fig. 3). The T₅₀₀ values calculated from measurements on Day 280 were 25.7
INFLUENCE OF TRAUMA FROM OCCLUSION ON HEALTHY PERIODONTIUM

The removal of the oblique plane on Day 280 resulted in a reduction of tooth mobility in the control teeth. On Day 370 the average mobility value of the control teeth did not significantly differ from the corresponding value on Day 0. In the test teeth, however, there was a further increase of the mobility towards the end of the study. At the termination of the experiment the mean $T_{900}$ value of the control teeth was 5.4 as compared with 42.4 for the test teeth.

Fig. 4. Radiograph from the $P_4$ region obtained after 180 days of experimental periodontal breakdown. Note the marked "horizontal" alveolar bone loss.

Röntgenaufnahme vom $P_4$ Bereich nach 180 Tagen mit experimentellem parodontalen Abbau. Man bemerke den ausgesprochenen "horizontalen" Knochenverlust.

Radiographie de la région $P_4$ après 180 jours de destruction parodontale expérimentale. Noter la résorption osseuse "horizontale" marquée.

Alveolar Bone Topography

After 6 months of experimental periodontitis, the level of the marginal alveolar bone had been moved around 4 mm apically. The marginal crest of the alveolar bone had an uneven and indistinct appearance (Fig. 4). From roentgenographs obtained on Day 280, it could be seen that the jiggling forces had produced a marked
cone-shaped widening of the marginal periodontal ligament space; this was especially pronounced around the mesial root. In addition, the periapical ligament space was also somewhat widened (Fig. 5). The removal of the oblique plane from the cap splint (i.e. the elimination of jiggling forces) and the treatment of the periodontal inflammation resulted in alterations in the periodontal tissues which, in roentgenographs, were characterized by the reestablishment of a narrow periodontal ligament space and the presence of a distinct and radiopaque marginal termination of the alveolar bone (Fig. 6). On radiographs obtained from the test tooth regions at the end of the study, the marginal termination of the alveolar bone exhibited an even and rather distinct outline (Fig. 7). The perio-

Fig. 6. Radiograph of a control tooth region obtained at the end of the experiment. Note the distinctly outlined marginal alveolar bone crest and the narrow periodontal ligament space.

Fig. 7. Radiograph of a test tooth region obtained at the end of the experiment. Note the distinctly outlined marginal alveolar bone crest but also the widened periodontal ligament space; this was especially pronounced at the mesial root (arrows).
dental ligament space, however, still appeared markedly widened, not only in the marginal but also in the apical regions.

Histological Findings

The gingival tissues of four out of five dogs had a rather uniform appearance. The oral epithelium was well keratinized and the junctional epithelium was smooth without signs of rete peg formations. The connective tissue was almost devoid of inflammatory cells but characterized by the presence of a dense collagenous network. In one dog the very marginal part of the gingiva (on test as well as control regions) contained a small zone of round cell accumulations. In no section did the periodontal ligament tissue harbour infiltrates of leucocytes. The periodontal ligament tissue on the pressure side of the test teeth harboured a large number of small-sized (<10 μm in diameter) vascular units. In the control teeth, the vasculature of the same region was mainly composed of larger vascular units.

The most apical cells of the junctional epithelium were, in the control as well as in the test teeth, always located at a level marginal to the apical border of the notch (A) (Fig. 8). The dentin surface of the part of the notch which made contact with the junctional epithelium did not display signs of cementum formation. The part of the notch, however, which faced connective tissues was always lined with newly formed cellular cementum (Fig. 8). Connective tis-
Table 2. The average distance (in units) between 1) the apical border of the notch (A) and the apical cells of the junctional epithelium (JE), 2) the bone crest (BC) to the junctional epithelium (JE), 3) the apical border of the notch (A) to the bone crest (BC) and 4) the average length of the epithelial attachment (EA) on the mesial surface of the teeth.

<table>
<thead>
<tr>
<th>Test</th>
<th>Control</th>
<th>Level of significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>A-JE</td>
<td>441 ± 75.0</td>
<td>774 ± 63.5</td>
</tr>
<tr>
<td>BC-JE</td>
<td>474 ± 64.1</td>
<td>422 ± 56.0</td>
</tr>
<tr>
<td>A-BC</td>
<td>-36 ± 85.3</td>
<td>351 ± 62.1</td>
</tr>
<tr>
<td>EA</td>
<td>1880 ± 130</td>
<td>1550 ± 110</td>
</tr>
</tbody>
</table>

Sue fibres seemed to terminate in this cementum.

The average distance (Fig. 2) between the apical border of the notch (A) and the most apical cells of the junctional epithelium (JE) was 441 μm in the test teeth and 774 μm in the controls (Table 2). This difference is statistically significant at the 1% level.

In both the test and control teeth the average distance between the bone crest (BC) and the apical border of the dento-gingival epithelium (JE) was around 440 μm.

In the control teeth the bone crest (BC) was consistently located on a level marginal to the apical border of the notch. In the test teeth, however, the bone crest in three dogs out of five was located at a level apical to the notch. The difference between the test and control teeth regarding the location of the bone crest (BC) in relation to the notch (A) was statistically significant (P < 0.01).

The results from the determinations of the periodontal ligament area (PLA), the vascular cross-section area (VCA) and the number of osteoclasts are given in Table 3. On the average, the PLA as well as the VCA of the test teeth was twice the size of the controls. The vascular cross-section area per periodontal ligament area, however, was similar in test and control teeth (17% and 14%, respectively). There was a significantly larger number of osteoclasts present on the alveolar bone surface bordering the PLA of the test teeth than in the corresponding area of the control teeth (8.25 and 0.13).

Discussion

In the present study periodontal disease was induced around single posterior premolars by allowing plaque to accumulate around cotton floss ligatures placed around the neck of the teeth. Earlier studies in the dog (Lindhe & Svanberg 1974, Ericsson et al. 1975) have shown that 6 months of plaque accumulation of this kind will result in a periodontal lesion characterized by “horizontal” alveolar bone loss, apical downgrowth of the dento-gingival epithelium and suprabony pocket formation. If under otherwise identical experimental conditions trauma from occlusion of the jiggling type is introduced as a co-factor, the rate of progression of periodontal breakdown seems to increase and angular osseous defects combined with infrabony pockets will result (Svanberg & Lindhe 1974, Lindhe & Svanberg 1974).
Table 3. The average periodontal ligament area (PLA), vascular cross-section area (VCA) and number of osteoclasts on the alveolar bone surface bordering PLA of the test and control teeth.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Test</th>
<th>s.e.</th>
<th>Control</th>
<th>s.e.</th>
<th>Level of significance</th>
<th>Test/Control</th>
<th>s.e.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Periodontal ligament area (PLA)</td>
<td>767.3</td>
<td>87</td>
<td>386.5</td>
<td>25</td>
<td><em>P &lt; 0.01</em></td>
<td>2.0</td>
<td>0.3</td>
</tr>
<tr>
<td>Vascular cross-section area (VCA)</td>
<td>129.4</td>
<td>18</td>
<td>57.2</td>
<td>6</td>
<td><em>P &lt; 0.001</em></td>
<td>2.3</td>
<td>0.4</td>
</tr>
<tr>
<td>VCA/PLA × 100 (%)</td>
<td>17</td>
<td></td>
<td>14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of osteoclasts</td>
<td>8.25</td>
<td>0.8</td>
<td>0.13</td>
<td>0.09</td>
<td><em>P &lt; 0.001</em></td>
<td></td>
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</tr>
</tbody>
</table>

In order to evaluate further the relative importance of plaque and the "co-destructive factor" in periodontal disease, a study was performed in dogs with experimentally induced periodontal breakdown. Plaque was removed and pathologically deepened pockets were surgically eliminated, but jiggling trauma was allowed to persist during healing. The results demonstrated that, provided plaque and inflamed periodontal tissues were removed and a proper oral hygiene regimen was established, healing also occurred in cases where jiggling forces were acting on hypermobile teeth. This indicates that microbial plaque is the main causative factor not only in gingivitis and early periodontitis (Lindhe et al. 1973) but also in the progressive lesion where trauma from occlusion may act as a "co-destructive" component. It should be observed that healing after periodontal treatment occurred when the test teeth were still in the so-called traumatic phase (Svanberg & Lindhe 1973), i.e. exhibited increasing tooth mobility, increased osteoclastic activity and a large number of small-sized vessels within the periodontal ligament of the zone of "co-destruction". This implies that, in the periodontal ligament area, tissue alterations caused by trauma from occlusion do not interfere with the regeneration and the reorganisation of the epithelial and connective tissue components of the gingiva. This interpretation is supported by observations reported by Waerhaug (1955). He studied the pathogenesis of pocket formation in traumatic occlusion in dogs and stated: "The area of principal fibers between the alveolar crest and the deepest line of the epithelial cuff represents a safety zone. This band of tissue follows the movements of the tooth and will not be injured by the same trauma that causes injury to the periodontal membrane within the alveolus." As a matter of fact, in the present study, the distance between the apical cells of the junctional epithelium and the alveolar crest (BC to JE; Table 2), i.e. the "safety zone", was the same in the test and non-jiggled control teeth.

In the present experiment, healing of the periodontal tissues was documented clinically as well as by microscopic analyses of biopsy preparations. The clinical data
showed that at the termination of the experiment: (1) the gingiva around the test teeth was normal (GI = 0 in four dogs; GI = 1 in one dog), and (2) the marginal termination of the alveolar bone in the roentgenographs exhibited an even and distinct outline. In the histological sections (except in one dog which showed clinical as well as histological signs of overt gingivitis), there were no signs of round-cell infiltration in the supraalveolar connective tissue or in the periodontal ligament in either the test or control tooth regions. The junctional epithelium was smooth and without rete pegs. Collagen fibres were inserted in the newly formed cementum apical to the junctional epithelium. In this respect the results of this study corroborate data presented by Glickman et al. (1966). They studied the effect of occlusal forces on healing following muco-gingival surgery and reported that trauma from occlusion "did not influence the healing of the gingiva in terms of the formation of a normal gingival sulcus and zone of attached gingiva."

The findings of the present experiments also support observations made in clinical studies reported by Nyman et al. (1975) and Rosling et al. (1975). They showed that once plaque and chronically inflamed periodontal tissues had been removed and provided the dentitions were kept up to a proper standard of oral hygiene, normal healing of periodontal tissues always occurred, even around teeth which were subjected to jiggling forces.

The histometric analysis of the periodontal tissues revealed various differences between the test and control teeth. Thus, in the test teeth (1) the size of the periodontal ligament area, as well as the vascular cross-section area, was twice as large and (2) the number of osteoclasts was several times as high as in the control teeth. Similar findings were reported by Glickman et al. (1966). Three months after periodontal surgery, the periodontal tissues of dog teeth subjected to occlusal trauma were characterized by a widened periodontal ligament with increased vascularity. Glickman et al. (1966) also noted that the gingival segment of the alveolar bone in the pressure zone was thinner due to increased osteoclastic activity. In the present material a similar thinning of the bone was noted in the jiggled teeth. In the most coronal part of the marginal bone this thinning resulted in a reduction of the height of a thin alveolar bone crest. Therefore in the control dogs the bone crest was always located at a level marginal to the apical border of the notch, whereas in the test dogs the bone crest was located at or slightly apical to the notch. The difference in location of the bone crest between the test and control teeth may, therefore, be regarded as a consequence of different functional demands. Since, however, the apical cells of the junctional epithelium in the control teeth were located more coronally than in the test teeth, it may be argued that in the control teeth, but not in the jiggled test teeth, a reattachment resulted following periodontal treatment. The design of the present experiment does not allow a detailed analysis of this problem.

**Zusammenfassung**

Die Einwirkung traumatisierender Ocklusion auf reduziertes aber gesundes parodontales Gewebe bei Hunden

Vom Tage 280 bis zum Tage 370 wurden die Zahne der Versuchstiere zweimal taglich gereinigt. Dann wurden die Tiere geopfert. Der Bereich der Prämolaren wurde mit Röntgenaufnahmen untersucht, Gewebeschnitte der Region P3 P4 M3 (sowie P3 P4 M4) hergestellt und mikroskopisch analysiert.

Die Resultate zeigen, dass okklusales Trauma mit Vor- und Zurückverschiebung der Krone (Schaukeltyp) sowie Hypermobilität von Zähnen keinen Anlass zu Heilungsstörungen nach parodontaler Chirurgie geben.

Résumé

Influence des traumatismes occlusaux sur les tissus parodontaux affaiblis, mais non pathologiques, chez le chien

Ces expériences ont été effectuées sur cinq chiens recevant une nourriture molle qui permettait l'accumulation de la plaque dentaire. Une destruction expérimentale du parodonte a été introduite au jour 0. 180 jours plus tard, un traumatisme occlusal a été produit au niveau de P3 et P4, c.-à-d. au niveau des quatrièmes prémolaires inférieures, suivant la méthode décrite par Svanberg & Lindhe (1973). Au jour 280, on a procédé à l’éradiication chirurgicale des culs-de-sac existant autour de P3 et P4. Une rainure devant servir de repère a été faite dans la racine au niveau du fond du cul-de-sac. De plus, dans la région de P3, le traumatisme occlusal a été supprimé. Du jour 280 au jour 370, les dents des animaux ont été brossées deux fois par jour. Les animaux ont ensuite été sacrifiés, les régions des prémolaires ont été radiographiées, et des coupes comprenant P3 P4 M3 (et P3 P4 M4) ont été préparées et étudiées au microscope. Il ressort des résultats de cette étude que le traumatisme occlusal de type "jiggling" (va-et-vient) et l'exagération de la mobilité dentaire ne sont pas des facteurs agissant au detriment de la cicatrisation après les interventions de chirurgie parodontale.

References


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