Influence of trauma from occlusion on progression of experimental periodontitis in the beagle dog

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Abstract. The experiments were performed in six beagle dogs fed a soft diet which allowed dental plaque formation. During a pre-experimental period of 7 weeks, periodontitis was induced by (1) surgically creating a bony pocket and (2) adapting a copper band to the exposed tooth surface. Two dogs were sacrificed at the end of this period and tissue sections were prepared for histological examination. In the remaining four dogs, trauma from occlusion was produced on the left mandibular fourth premolar by the installation of a cap splint and a bar device. The contralateral premolar served as a control.

At the start of, and at regular intervals during, an experimental period of 180 days, tooth mobility, gingival inflammation and plaque accumulation were assessed. After sacrifice, radiographs were taken of test and control tooth regions and histological sections analysed regarding the width of the marginal periodontal ligament space and the degree of apical downgrowth of the gingival pocket epithelium.

Only the test teeth showed a gradually increasing horizontal mobility, but gingival inflammation and Plaque Index scores were similar on test and control sides. Radiographs revealed (1) horizontal bone loss in both test and control areas, and (2) angular bone destruction only in test areas. Histological sections showed that the degree of apical proliferation of the pocket epithelium was more pronounced in test than in control regions.

Experiments carried out in different laboratories have demonstrated that it is possible in dogs and monkeys to induce a gingival inflammation which develops into periodontitis by allowing the accumulation of plaque and calculus (Saxe et al. 1967, Lindhe et al. 1973, Kennedy & Polson 1973). It has been suggested (for review see Posselt 1966, Glickman 1967) that trauma from occlusion in the presence of periodontal inflammation may be an important contributory factor in the pathogenesis of periodontal disease. Thus, occlusal forces may alter the path of spread of gingival inflammation and thereby facilitate direct penetration into the periodontal ligament (Macapanpan & Weinman 1954), enhancing angular bone resorption and infrabony pocket formation (Glickman & Smulow 1962, 1965).

In animals, periodontal tissue changes due to trauma from occlusion resemble those found in human autopsy material (for review see Posselt 1966). Objections have been raised, however, concerning the validity of conclusions made from autopsy
examinations and from experiments which do not consider jiggling forces in combination with a progressive periodontal lesion (Wentz et al. 1958, Ramfjord et al. 1966).

The aim of the present investigations was to assess what effect trauma from occlusion and permanent tooth hypermobility would have on the rate at which experimental periodontitis progresses in the beagle dog.

Material and Methods

Six beagle dogs, 2–3 years old and weighing around 12 kg, were used. Prior to the experiments the dogs were fed a diet favouring gross plaque formation on premolars and molars. At the beginning of the experiments all the dogs showed clinical signs of gingivitis in the premolar and molar regions. Throughout the study the animals were fed a diet allowing plaque and calculus formation (Hamp et al. 1973).

Periodontitis was induced according to a modification of the method described by Swenson (1947). On the mesial aspect of the fourth lower premolar on both sides (P4 and 4P), infrabony pockets were made with a slender diamond cylinder bur. A notch was made in the root at the level of the bottom of the surgically created pocket. This notch served as a landmark for loss of attachment measurements performed on histological preparations in the microscope (see below).

A copper band similar to that used by Swenson (1947) and Hurt (1963) was punched close to the tooth, with a tongue extending into and carefully adapted to the base of the notch in the tooth (Fig. 1). The band was then cemented to the tooth with copper cement*. Three weeks later the copper band was removed. To enhance formation of soft deposits a plaque retention band was placed around the tooth at the level of the cemento-enamel junction. The circumference of this rubber band** was equal to that of the tooth at the cemento-enamel junction.

Four weeks later, two of the dogs were sacrificed and sections of test and control teeth were prepared for histological examination, as described below. In four of the dogs, trauma from occlusion was produced by the installation of cap splints, as described by Svanberg & Lindhe (1973). On the left side of the maxilla the dogs were fitted with a cap splint with an ob-

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Fig. 2. Design of the experiment. Trauma from occlusion was introduced 49 days (21 + 28 days) after pocket surgery.

Fig. 2. Versuchsplanung. Die okklusale Traumatisierung begann 49 Tage (21 + 28 Tage) nach der chirurgischen Bildung einer Parodontaltasche.

Fig. 2. Plan de l'expérience. Le traumatisme occlusal fut introduit 49 jours (21 + 28 jours) après la création chirurgicale d'une poche parodontale.

The oblique plane which made primary contact with the distal surface of the left mandibular fourth premolar (P₄) when the mandible moved towards centric occlusion. The inclination of the oblique plane was such as to exert mainly horizontal forces upon the test tooth. During chewing movements P₄ was subjected to excessive horizontal forces and tilted in mesial direction.

On the left side of the mandible a spring* attached to a lingual bar was introduced through a channel in the crown of the test tooth. When not in occlusal contact, P₄ was tilted back to its original position by the spring appliance.

The mesial aspect of P₄ was designated the pressure side. In each dog the contra lateral tooth (i.e. 4P) served as a control. In order to attain equal plaque formation on both sides, the right lower jaw (control side) was fitted with a lingual bar device. However, a soft wire exerting no force upon 4P was introduced through the channel in the crown of the tooth.

The dogs were sacrificed 6 months after the installation of the cap splint and bar devices.

Immediately after installation of the cap splint and bar devices, i.e. on day TR (Fig. 2) and after 30 (TR + 30), 60 (TR + 60), 90 (TR + 90) and 180 (TR + 180) days, P₄ and 4P were studied for the following clinical features.

I. Tooth mobility. T₁₀₀ and T₅₀₀ were assessed in accordance with the method described by Mühlemann (1954).

II. Gingival inflammation. The state of the gingiva was assessed by measuring (in mm) the amount of gingival exudate obtainable on chromatography paper** strips inserted into the orifice of the mesial gingival pocket of P₄ and 4P (Löe & Holm-Pedersen 1965).

III. Plaque. The amount of plaque on the mesial surface of P₄ and 4P was estimated according to the plaque index system (Pl I) described by Silness & Löe (1964).

IV. Radiographic bone loss. On day TR + 180 the dogs were sacrificed with an overdose of Pentothal sodium® (Abbott, Belgium). The mandible was removed and bisected along the midline, and radiographs of the premolar region on both sides were taken on a skull.

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* Dentaurum Ltd., Germany, spring-hard wire 0.55 mm.
table. The central ray was directed perpendicular to the longitudinal axis of the teeth. The focus-to-film distance was 880 mm and roentgenography was performed at 4 kVp and 100 mAs with Kodak Ultra Speed film. The vertical distance between the notch made in the root surface and the alveolar bone was measured by the aid of a sliding caliper.

Histological Examination
Specimens containing the distal portion of P₃ (₃P), P₄ (₄P) and the mesial portion of the first molars (M₁ or 1M) were fixed in a buffered solution of 10% formalin in water, decalcified in a solution of equal amounts of 50% formic acid and 15% sodium formiate, dehydrated and embedded in paraffin. Mesiodistal sections were cut with the microtome (Leitz Wetzlar 1300) set at 7 μm, and stained with haematoxylin-eosin. From each biopsy specimen three sections 50 μm apart were used for determining the distance between the landmark in the root surface (i.e. the level of the bottom of the surgically created pocket) and the most apical cells of the pocket epithelium. A mean "loss of attachment" score for each tooth was calculated. The measurements were made in the light microscope (Leitz Ortoplan).

The periodontal ligament area (PLA) on the pressure side of P₄ and that on the corresponding side of ₄P were determined according to a technique recently described by Svanberg & Lindhe (1973). Statistical analyses of the data were performed according to Student's t-test.

Results

Tooth Mobility
On day zero, immediately before trauma from occlusion was produced, the mobility of the test and that of the control teeth were similar. Thus, the T₁₀₀ and T₅₀₀ values were 10.0 ± 1.7 and 16.5 ± 2.2 (test) and 9.2 ± 1.2 and 17.5 ± 3.7 (control). After installation of the cap splint and bar devices the mobility of the test teeth gradually increased (Fig. 3). T₁₀₀ and T₅₀₀ reached maximum values at the end of the 180 days of the experiment; T₁₀₀ = 49.0 ± 8.8; T₅₀₀ = 78.8 ± 18.0. The increase in mobility of the test teeth was significant (P < 0.001). In addition, all test teeth clinically exhibited a pronounced axial mobility. During the entire experiment the mobility of the control teeth remained practically unchanged.

Gingival Inflammation
The mean gingival exudate scores of the test and control teeth on day zero were 4.9 ± 0.9 and 3.1 ± 0.8 respectively, the difference not being significant. Neither in the test tooth areas nor in the control ones did any significant alteration of the gingival exudation occur during the experimental period (Table 1).

<table>
<thead>
<tr>
<th>Day</th>
<th>Test</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>4.9 ± 0.9</td>
<td>3.1 ± 0.8</td>
</tr>
<tr>
<td>30</td>
<td>4.5 ± 0.7</td>
<td>3.7 ± 0.9</td>
</tr>
<tr>
<td>60</td>
<td>3.3 ± 0.5</td>
<td>3.9 ± 0.9</td>
</tr>
<tr>
<td>90</td>
<td>4.0 ± 0.8</td>
<td>2.5 ± 0.6</td>
</tr>
<tr>
<td>180</td>
<td>3.2 ± 0.9</td>
<td>4.3 ± 0.6</td>
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Plaque Index
At all examinations, from day zero to day 180, the Plaque Index scores of both the test and the control teeth were 3, i.e. the gingival third of the tooth surfaces as well as the gingival pockets exhibited abundant soft deposits. At the examinations after 60, 90 and 180 days of the experiment, mineralized deposits were present on the test as well as on the control teeth.

Radiographic Bone Loss
At the end of the experiment radiographs of the regions of the control tooth in all four dogs revealed pronounced horizontal loss of marginal periodontal bone. Fig. 4 is a radiograph of the control side of dog II at the end of the experiment. The mean distance between the notch of the control teeth and the alveolar bone, as measured in the roentgenograms, was 2.1 ± 0.1 mm (Table 2).

Table 2. Distance between the notch in the root surface and the marginal alveolar bone level (control) or bottom of osseous pocket (test). The measurements were performed on radiographs (mm)

<table>
<thead>
<tr>
<th>Dog</th>
<th>Test</th>
<th>Tooth</th>
<th>Diff.</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>4.5</td>
<td>2.0</td>
<td>2.5</td>
</tr>
<tr>
<td>II</td>
<td>5.5</td>
<td>2.5</td>
<td>3.0</td>
</tr>
<tr>
<td>III</td>
<td>4.5</td>
<td>2.0</td>
<td>2.5</td>
</tr>
<tr>
<td>IV</td>
<td>5.0</td>
<td>2.0</td>
<td>3.0</td>
</tr>
<tr>
<td>x ± s.e.</td>
<td>4.9 ± 0.24</td>
<td>2.1 ± 0.12</td>
<td>2.7 ± 0.14</td>
</tr>
</tbody>
</table>
In the region of the test teeth there was not only a pronounced horizontal bone loss but also, on the pressure side, a marked cone-shaped widening of the periodontal ligament space. In addition, the periapical periodontal ligament space was markedly widened. Fig. 5 is a radiograph of the test side of dog II at the end of the experiment. Note the angular bony defect and the infrabony pocket on the pressure side, as well as the radiolucent zone around the apices. The mean distance between the notch of the test teeth and the marginal alveolar bone (i.e. the bottom of the mesial bony pocket) was 4.9 ± 0.2 mm (Table 2).

Histological Findings

Day 0. Tissue sections obtained from two dogs 7 weeks after the induction of experimental periodontitis (corresponding to day 0 of the trauma from occlusion experiment) revealed that the four epithelialized pockets (on the mesial aspects of $P_4$ and $P$ in each animal) extended to the level of or slightly apical to ($<100 \mu m$) the notch made in the cementum. The connective tissue subjacent to the pocket epithelium was infiltrated with round cells. In all four biopsy specimens the inflammatory cell infiltration comprised most of the supra alveolar connective tissue.

Day 180. Sections from the regions of the control teeth representing day 180 of the experiment consistently showed an apical downgrowth of epithelium from the notch in the root surface (= the bottom of the surgically created pocket). Adjacent to the gingival pocket were dilated vessels in a connective tissue heavily infiltrated with
leucocytes. Within the periodontal ligament leucocytes were seen only occasionally (Fig. 6a).

Sections of regions of the test teeth (Fig. 6b) showed more extensive proliferation of the pocket epithelium. The leucocyte infiltration area seemed to extend further apically than in corresponding sections of the regions of the control teeth. Markedly dilated vessels were seen within a supra-alveolar connective tissue which was heavily infiltrated with leucocytes. The periodontal ligament, however, was not infiltrated with inflammatory cells. On the bone surface of the alveolus, a few osteoclasts could be seen.

The marginal part of the periodontal ligament space on the pressure side of the test teeth showed an angular widening and, according to the planimetric data, the periodontal ligament area (PLA) was 3.3 (s.e. ± 0.60) times that of the controls. The bone surface of the alveolus was markedly undulated. The periodontal ligament appeared less dense than in the controls and wide communications between the periodontal space and the marrow spaces were present. The periapical periodontal ligament space of the test teeth was extremely wide and bordered by an alveolar bone, the surface of which was undulated. The tissue within the periapical portion of the periodontal ligament was characterized by the presence of a large number of small vessels and the absence of inflammatory cells.

The histologic measurements revealed that the distance between the notch in the root and the most apical cell of the pocket epithelium (“loss of attachment”) on the pressure side of the test teeth was 2.2 ±
Fig. 6. Sections from control and test teeth at the end of the experiment showing a more extensive apical proliferation of pocket epithelium on the pressure side of the test tooth (Fig. 6b) than on the control side (Fig. 6a). Haematoxylin-eosin, orig. magn. obj. X 1.

N: Notch in root surface (= bottom of surgical pocket).
P: Localization of the most apical cells of the pocket epithelium.
L: Loss of attachment (from induction of experimental periodontitis to the end of the trauma from occlusion experiment).

Fig. 6. Gewebsschnitte des traumatisierten Zahnes (Fig. 6b) und des Kontrollzahnes (Fig. 6a). Der traumatisierte Zahn zeigt im Vergleich mit dem Kontrollzahn (Fig. 6a) eine tiefer greifende apikale Proliferation des Taschenepithels auf der Druckseite (Fig. 6b). Hämatoxylin-Eosin. Orig. Vergr. 1 ×.

N: Kerbe in der Wurzeloberfläche = Fundus der chirurgisch gebildeten Parodontaltasche.
P: Apikalste Stelle der Taschenepithelzellen.
L: Gewebsloslösung (Attachment-Verlust) vom Beginn der Erzeugung der experimentellen Parodontitis bis zum Abschluss der künstlichen okklusalen Traumatisierung.

Fig. 6a, b. Coupes de dents traumatisées et contrôle à la fin de l'expérience montrant une prolifération apicale de l'épithélium du cul-de-sac plus étendue du côté traumatisé (Fig. 6b). Hématoxyline-eosine, mag. orig. × 1.

N: entaille dans la surface radiculaire (= fond du cul-de-sac chirurgical).
P: localisation des cellules les plus apicales de l'épithélium du cul-de-sac.
L: perte d'attachement (de l'induction de la parodontite expérimentale à la fin de l'expérience de traumatisme occlusal).
± 0.47 mm. The corresponding distance for the control side was 0.8 ± 0.13 mm. The difference is significant (P < 0.05).

Discussion

The present experiments have demonstrated that it is possible to produce pathological pocket formation in the beagle dog by (1) surgical creation of a local osseous defect, (2) prevention of initial reattachment, and (3) promotion of gross plaque formation by application of a non-elastic rubber band around the tooth at the level of the cemento-enamel junction.

Seven weeks after surgery the gingivae showed marked clinical signs of inflammation. Biopsy specimens from this period revealed the presence of an epithelialized gingival pocket extending down to the bottom of the surgical defect. The connective tissue subjacent to the epithelium was heavily infiltrated with leucocytes. These findings are in agreement with those published by Swenson (1947) and Ramfjord (1951). They used a similar method for producing a periodontal lesion and reported developing gingivitis and downgrowth of epithelium to the bottom of the surgical pocket.

The experiments also showed that the technique used can induce a rapidly progressing periodontal disease around a single tooth; the lesion included further apical shift of the pocket epithelium and extensive alveolar bone loss. Thus, about 8 months after the beginning of the experiment some 50% of the alveolar bone around the control teeth had been lost. This observation is largely in agreement with findings by Collings (1957) and Collings & Redden (1959). They created an initial bony lesion surgically, allowed gross plaque formation, and reported that a periodontal lesion resembling rapidly progressing periodontitis in humans could be produced in the dog. Similar findings have been reported by, e.g. Zander et al. (1971), Caton & Crigger (1972), and Kennedy & Polson (1973) from experiments in the monkey.

Immediately prior to the introduction of jiggling forces, i.e. after 7 weeks of the experiment, tooth mobility of the test teeth and control teeth was similar. It should be observed, however, that the T100 and T500 values obtained were about seven times as high as those of posterior premolars in dogs with non-inflammatory gingivae, as reported by Svanberg & Lindhe (1973). After the installation of cap splints the test teeth showed a very pronounced and gradually increasing mobility throughout the observation period. On “Day TR+180” the jiggling teeth were found to be extremely loose and mobile both transversely and longitudinally. In contrast, the mobility of the contralateral control teeth remained practically unchanged during the final 6 months of the experiment (Fig. 3).

The microscopic examination of sections from the pressure side of the test teeth obtained 6 months after the onset of “trauma from occlusion” revealed an extremely wide periodontal ligament space, bordered by an undulated alveolar bone surface and a cementum with resorption lacunae in some areas. The PLA scores showed that the width of the periodontal ligament space of the test teeth was about three times that of the controls. This finding is in close agreement with data presented by Wentz et al. (1958), who studied periodontal tissue reactions to the jiggling type of trauma in monkeys with normal gingiva. After 3- and 6-month experiments they observed a widening of the periodontal ligament space from, on the average, 0.19 mm (at start) to 0.65 mm. In the present material after 6 months' jiggling of the teeth, the periodontal tissues showed no marked signs of active resorption of bone or cementum, necrosis, vascular thrombosis or pronounced inflammatory cell
infiltration. This means that at this stage the test teeth and their periodontal tissues must have become almost but not entirely adapted to the altered occlusal function. Such a conclusion is in agreement with results published by Wentz et al. (1958) and Svanberg & Lindhe (1973). They reported initial tissue reactions, e.g. bone resorption and granulation tissue formation, following exposure of the teeth to jiggling forces. After some months of experiment, however, the periodontal tissues appeared normal except for a widening of the periodontal space.

The radiographs of the test tooth regions obtained at necropsy, i.e. after 6 months of jiggling and continued plaque formation, displayed not only advanced horizontal bone loss but also angular or crater-like osseous defects. The bony defects were most pronounced in the pressure areas. The defects in the bone resembled the lesion frequently described by Glickman and collaborators (for review see Glickman 1967) as the result of trauma from occlusion.

The radiographs also revealed obvious zones of bone resorption around the root apices; especially around those of the mesial roots. The microscopical analyses confirmed the x-ray findings, viz. markedly widened apical periodontal spaces. These spaces contained richly vascularized tissue, but no signs of vascular thrombosis, or inflammatory cell infiltration. The pulp tissue in the apical region did not harbour inflammatory cells. The cementum showed resorption lacunae and the bordering bone was undulated, both of which are indications of previous active bone resorption. The periapical alterations must therefore be regarded as adaption of the tissue to increased functional requirements.

Though the Plaque Index scores and the degree of gingival exudation of the test and control teeth were similar throughout the experimental period, the test teeth showed an apical shift of the pocket epithelium which was three times that of the controls. In dogs with no clinical inflammation of the gingiva or with well established gingivitis, trauma from occlusion by the same method as that used in this experiment did not result in an apical shift of the junctional epithelium (Svanberg 1974). In the study referred to, the most apical epithelial cells were always located at the cemento-enamel junction (CEJ), and the connective tissue subjacent to CEJ was consistently free from inflammatory cell infiltrates. Similar results have been obtained by Wentz et al. (1958) in monkeys with overt gingivitis. It may therefore be suggested that any effect of occlusal trauma on the attachment level requires the presence of a plaque-induced inflammatory cell lesion in the supra-alveolar connective tissue. In the present experiment, which allowed abundant plaque formation, deepening of the gingival pocket and loss of fibre attachment occurred at a faster rate on teeth subjected to jiggling tissue trauma. Hence it may be suggested that trauma from occlusion in dogs may accelerate progression of experimental periodontitis.

Zusammenfassung

Die vorliegende Arbeit wurde ausgeführt, um den Einfluss von traumatisierenden Kräften (Jiggling) auf das Fortschreiten einer vorbestandenen experimentellen marginalen Parodontitis festzustellen.


Radiologisch war auf der Seite des okklusalen Traumas, wie auch auf der Kontrollseite horizontaler Knochenschwund vorhanden. Vertikale Einbrüche konnten nur auf der Traumaseite festgestellt werden. Die Tiefenwucherung des Taschenepithels war auf der Testseite ausgeprägter.

Zusammenfassend schien die Progression der vorbestandenen marginalen Parodontitis durch die Traumatisierung, gemessen an der Zahnbeweglichkeitserhöhung, gefördert zu werden.

Résumé

Cette recherche fut entreprise pour analyser l'effet de forces occlusales traumatisantes de va-et-vient (jiggling) sur la progression de la parodontite marginale expérimentale. Six briquets reçurent une diète molle permettant la formation de plaque dentaire. Durant une période pré-expérimentale de 7 semaines, une parodontite marginale fut introduite par une méthode chirurgicale; à la fin de cette période deux des chiens furent sacrifiés en vue d'examens histologiques. Chez les quatre chiens restants le traumatisme occlusal fut produit sur la 4e prémolaire gauche inférieure (P4) par l'installation d'une attelle à bagues et d'un dispositif à barre. P4 servit de contrôle.

La mobilité dentaire, l'inflammation gingivale et l'accumulation de plaque furent évaluées au début et à intervalles réguliers durant une période expérimentale de 180 jours. Après sacrifice, on radiographia les dents testes et contrôles et on analysa les coupes histologiques quant à la largeur du desmodonte et au degré de prolifération apicale de l'épithélium du cul-de-sac.

Seule la dent test montra une mobilité horizontale augmentant graduellement et significativement tandis que l'inflammation gingivale et les résultats de l'index de plaque furent similaires pour les côtés test et contrôle. Les radiographies révélèrent 1) une perte osseuse horizontale dans le parodonte traumatisé et de contrôle; 2) une destruction osseuse verticale, du type triangulaire seulement dans le parodonte test. En plus, les coupes histologiques montrèrent une prolifération apicale de l'épithélium du cul-de-sac plus prononcée dans le parodonte test que dans le contrôle.

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