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Tooth mobility and resolution of experimental periodontitis

An experimental study in the dog

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Abstract. The aim of the present experiment was to study alterations in the mobility of teeth that occurred during resolution of experimentally induced periodontitis lesions in the dog. 5, 1-year-old, beagle dogs were used in the study. The left and right 4th, 3rd, and 2nd mandibular premolars ($_4P_4$, $_3P_3$, $_2P_2$) served as experimental teeth. Periodontal tissue breakdown was initiated by placing plaque-collecting cotton-floss ligatures around the neck of the experimental teeth. The ligatures were replaced to the level of the receding gingival margin 1 × every month. On Day 120, the ligatures were removed and debridement was performed. A groove, parallel to the long axis of the mesial root, was prepared in the mesio-buccal surface of the crowns of $_2P$ and P_2. Guided by the groove and with a probing force of 0.5 N, a probe was inserted into the buccal gingival pocket of the *mesial* root and was attached to the buccal surface. Biopsies including both the mesial and distal root of $_2P$ and P_2 and the surrounding hard and soft tissues were harvested. The biopsy procedure was repeated in a similar manner 15 days (i.e. Day 135) and 3 months (i.e. Day 225) after ligature removal in the 4th ($_4P_4$) and 3rd ($_3P_3$) premolar regions. After fixation, decalcification and sectioning, the biopsy material was exposed to histometric and morphometric measurements. Assessment of the mobility of the experimental teeth was performed on Days 120, 135 and 225 using the Periotest system. The amount of remaining bone at the experimental teeth was evaluated in radiographs obtained in a standardized manner. The findings of the present experiment disclosed that in dogs allowed to form plaque, the placement of cotton-floss ligatures at the neck of mandibular premolars initiated a process that resulted in (i) the formation of an inflammatory lesion which extended deep into the supracrestal connective tissue; (ii) extensive loss of alveolar bone; (iii) markedly increased tooth mobility. It was also observed that, within a 4-month period the removal of the ligature and, as a consequence, a substantial portion of the subgingival microbiota, reduced the size and the apical extension of the inflammatory lesion in the supracrestal connective tissue. The alterations in the soft supracrestal tissue were accompanied by a marked decrease in the mobility of the experimental teeth and a reduced probing pocket depth. It was suggested that the reduced penetration of the probe was the result of the change in the size and position of the infiltrate as well as of a reduced mobility of the experimental teeth.

Key words: dog; experimental periodontitis; probing pocket depth; resolution; tooth mobility.

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The biological significance of tooth mobility as a means of assessing tooth movements was analyzed by Mühlemann and co-workers in the 1950s (e.g., Mühlemann 1951, 1954, 1960, Mühlemann & Zander 1954, Fehr & Mühlemann 1956). A device, a "Periodontometer" was designed that enabled detailed measurements of the displacement of a tooth that occurred after standardized force application. Mühlemann (1954) distinguished between "initial tooth mobility" (ITM) and "secondary tooth mobility" (STM). "Initial tooth mobility" represents the displacement of a tooth that occurs following the application of a small force (100 pounds) to its crown. This movement is, according to Mühlemann & Zander (1954), the consequence of an intraalveolar displacement of the root and "a functional reorientation of the periodontal membrane fibers". "Secondary tooth mobility" results when

larger forces are applied to the crown and occurs after the periodontium of the "pressure side" has been "distorted and compressed" (Mühlemann & Zander 1954). Based on results from a long series of experiments, Mühlemann (1960) concluded that "secondary tooth mobility" is larger in children than in adults, increases during pregnancy and varies between different groups of teeth and during the course of the day.

Data from clinical and animal research have revealed that the height of the alveolar bone and the width of the periodontal ligament space are factors that influence both ITM and STM (for review, see Svanberg (1974), Lindhe et al. (1989), Lindhe & Nyman (1989)). It has also been reported that conventional treatment of plaque associated periodontal disease results in (i) reduction/elimination of the gingival lesion without accompanying marked alterations of the height of the connective tissue attachment and the supporting alveolar bone (e.g., Lindhe & Nyman 1975, Lindhe & Ericsson 1976, Caton et al. 1978) and (ii) obvious reduction of the initially increased mobility of the treated teeth (Goldberg 1962, Persson 1980, 1981a, b). In other words, not only the quality and quantity of periodontal ligament structures, but also the condition of the supraalveolar connective tissue are factors that seem to affect tooth mobility (for review, see Persson (1978)).

The aim of the present experiment was to study tooth mobility and some periodontal tissue alterations that occurred during resolution of experimental periodontitis lesion in the dog.

Material and Methods

5, 1-year-old, beagle dogs were used in the present investigation. The dogs were fed a diet which allowed gross plaque accumulation (Hamp et al. 1972). The left and right 4th, 3rd, and 2nd mandibular premolars ($_4P_4$, $_3P_3$, $_2P_2$) served as experimental teeth.

Experimental procedure

On Day 0, the plaque control program was terminated. Experimental periodontal tissue breakdown was initiated in accordance with a method described by Ericsson et al. (1975) and Lindhe & Ericsson (1978), i.e., by placing plaque collecting cotton floss ligatures around the neck of the experimental teeth ($_4P_4$, $_3P_3$, $_2P_2$). The ligatures were replaced at the level of the receding gingival margin once every month.

On Day 120, the ligatures were permanently removed and supragingival debridement was performed. A groove, parallel to the long axis of the mesial root, was prepared in the mesio-buccal surface of the crowns of $_2P$ and P_2. Guided by the groove and with a probing force of 0.5 *N* (Electronic Periodontal Probe®, model 200, Vine Valley Research, Middlesex, N.Y., USA) a probe (ø = 0.5 mm) was inserted into the buccal gingival pocket of the *mesial* root according to the technique described by Van der Velden (1981). The probe was attached to the buccal surface of the tooth by the use of a light curing composite filling material. Biopsies including both the mesial and distal root of $_2P$ and P_2 and the surrounding hard and soft tissues were harvested according to the technique described by Schroeder et al. (1973).

Probe placement and biopsy of the 4th ($_4P_4$) and 3rd ($_3P_3$) premolars were performed in a similar manner 15 days (i.e., Day 135) and 3 months (i.e., Day 225) after ligature removal.

Tooth mobility

Assessment of the mobility of the experimental teeth was performed on Days 120, 135 and 225 using the Periotest system (Periotest®, Siemens AG, Bensheim, Germany) described by Schulte (1986). The Periotest® determines the damping effect of the periodontium and the Periotest Value (PV-score) is calculated by assessing the deceleration of the electronically controlled rod of the instrument when it percusses the tooth crown.

Radiographic bone loss

Intraoral radiographs of all the experimental teeth were obtained in a standardized manner on Days 0, 120, 135 and 225 (Eggen 1969, Lindhe et al. 1973). The radiographs were evaluated regarding the amount of remaining alveolar bone. The measurements made in the radiographs were performed by the use of a digitizer (CalComp 91365, Digitizer Product Division, Scottsdale, AZ, USA) and a computer program (Status XR, AEC, Sweden) based on AutoCAD (Autodesk Inc, USA). The linear distances between the bone crest (BC) and the apex (A) of the root (BC-A) and between the cemento-enamel junction (CEJ) and the apex (CEJ-A = root lenght) were measured at the mesial surface of the mesial root and at the distal surface of the distal root. The mean height of the remaining supporting alveolar bone was calculated for each experimental tooth and expressed as % of the root length.

Histological procedures

The biopsies were placed in a fixative (4% formaldehyde) and decalcified in EDTA. Following decalcification each biopsy was divided into 2 portions, one including the *mesial* root (with the probe) and one including the *distal* root and adjacent soft and hard tissues.

The *mesial* portion (root) was, following removal of the probe, embedded in paraffin. Bucco-lingual sections parallel to the long axis of the root were cut with the microtome set at 3 μm. The sections were stained in hematoxylin-eosin. From each biopsy, 5 sections, representing the site of probing, were selected for *histometric measurements.*

The *distal* portion (root) was embedded in plastic resin (Polarbed®, Biorad House, Hemel Hemstead, Hertfordshire, UK). Bucco-lingual sections parallel to the long axis of the root were cut with the microtome set at 3 μm. The sections were stained in PAS and toluidine blue (Schroeder 1969). From each portion, representing the central area of the root, 5 sections were selected for *histometric* and *morphometric* determinations.

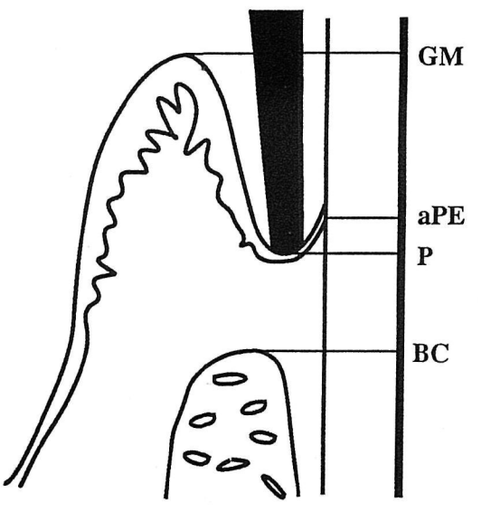

Fig. 1. Schematic drawing illustrating the landmarks for the histometric measurements made in the mesial root (paraffin embedded) sections. GM: gingival margin; aPE: apical cells of the pocket epithelium; BC: marginal portion of the supporting alveolar bone; P: most apical level of the probe tip.

listometric measurements

n the sections representing the mesial oot (paraffin sections), the following andmarks were identified (Fig. 1): (i) gingival margin (GM), (ii) apical cells of the pocket epithelium (aPE), (iii) marginal portion of the supporting alveolar bone (BC) and (iv) most apical level of the probe tip (P). The following linear distances were measured:

(1) GM-P
(2) aPe-P
(3) P-BC

In the distal sections, the following landmarks were identified (Fig. 2): (i) gingival margin (GM), (ii) apical cells of the pocket epithelium (aPE), (iii) marginal portion of the supporting alveolar bone (BC) and (iv) apical level of the inflammatory cell infiltrate (aICT). The following linear distances were measured:

(1) GM-aPE
(2) aPE-BC
(3) GM-aICT
(4) aICT-BC

In addition, the width of the periodontal ligament was assessed by determining the distance between the root surface and the inner bone wall of the alveolus at (i) the level of BC (wPDL/BC) and (ii) 1 mm apical to BC (wPDL/BC+1).

Morphometric measurements

The morphometric measurements carried out in the present study included level 1 and level 4 (Schroeder & Münzel-Pedrazzoli 1973) determinations and were made in a Wild® sampling microscope. At level 1 (mang. × 200; lattice P42; Weibel 1969) the analysis included the assessment of the volumes occupied by oral epithelium (OE), pocket epithelium (PE), non-infiltrated connective tissue (NCT) and infiltrated connective tissue (ICT) of the marginal gingiva. The level 4 analysis (magn. × 1000; lattice containing 100 light points) was performed in the non-infiltrated supracrestal connective tissue (N-SCT) and in the infiltrated connective tissue (ICT) (Fig. 2). In the supracrestal connective tissue compartment, the volume fractions occupied by collagen (Co), vascular structures (V), fibroblasts (Fi), leukocytes (Leu) and residual tissues (R) were determined. The ICT analysis included, in addition, assessment of macrophages (Mac), lymphocytes (Ly), plasma cells (Pc), and polymorphonuclear granulocytes (PMN).

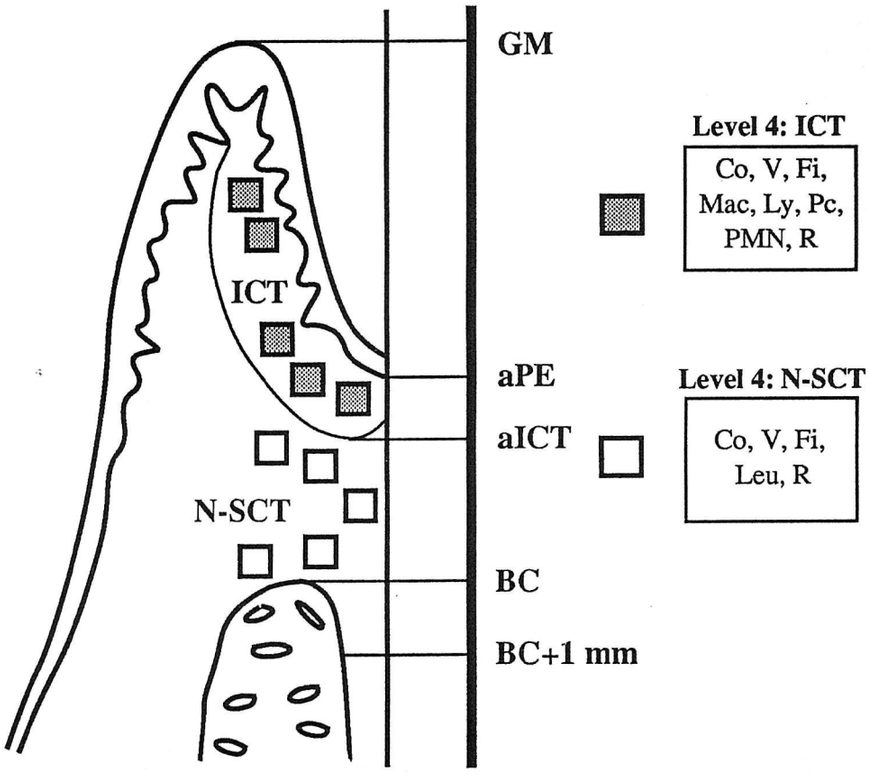

Fig. 2. Schematic drawing illustrating the landmarks for the histometric measurements made in the distal root (plastic embedded) sections and the areas in which the level 4 analysis have been performed. GM: gingival margin; aPE: apical cells of the pocket epithelium; aICT: apical level of the inflammatory cell infiltrate; BC: marginal portion of the supporting alveolar bone; BC+1 mm: level 1 mm apical to BC; ICT: infiltrated connective tissue; N-SCT: non-infiltrated supracrestal connective tissue; CO: collagen; V: vascular structures; Fi: fibroblasts; Mac: macrophages; Ly: lymphocytes; Pc: plasma cells; PMN: polymorphonuclear granulocytes; R: residual tissues.

Statistical analysis

The average value for each group of teeth was calculated and the results, from the different groups or time intervals, compared by use of the Student *t*-test for paired samples. A *p*-value of < 0.05 was considered to indicate a statistically significant difference.

Results

The clinical and radiographic examination performed at the start of the experiment, i.e., on Day 0, revealed that all the experimental units had a normal height of the supporting alveolar bone. On Day 120, i.e., after 4 months of experimental periodontal tissue breakdown, heavy amounts of plaque and calculus covered most of the exposed surfaces of the experimental teeth. In addition, the margin of the gingiva had markedly receded. Fifteen days following ligature removal, i.e., on Day 135, accumulations of plaque were still present at the remaining experimental teeth ($_4P_4$, $_3P_3$) but the gingival inflammation appeared less pronounced. At the end of the study, i.e., on Day 225, the clinical signs of gingival inflammation had further decreased but could still be noted in the 3rd premolar region ($_3P_3$).

Tooth mobility

The Periotest Values (PV-scores) are presented in Fig. 3. On Day 120, the PV-score, expressed as the mean of all measurements, was found to be 26.2 ± 2.3. The corresponding figure representing Day 135 was 27.1 ± 0.6 while, at the end of the study (i.e., Day 225), the PV-score was significantly smaller (16.0 ± 4.3). PV-scores of the teeth that were retained throughout the entire experiment ($_3P$ and P_3), were 26.1 (Day 120), 26.7 (Day 135) and 16.0 (Day 225).

Radiographic bone level

The mean amount of alveolar bone at the start of the experiment (Day 0) was $90.0 \pm 4.0\%$ and on Days 120, 135 and 225, $62.5 \pm 3.4\%$, $63.9 \pm 4.4\%$ and $59.8 \pm 5.2\%$, respectively (Fig. 4). The corresponding figures for $_3P_3$ were 90.0% (Day 0), 60.1% (Day 120), 61.4%

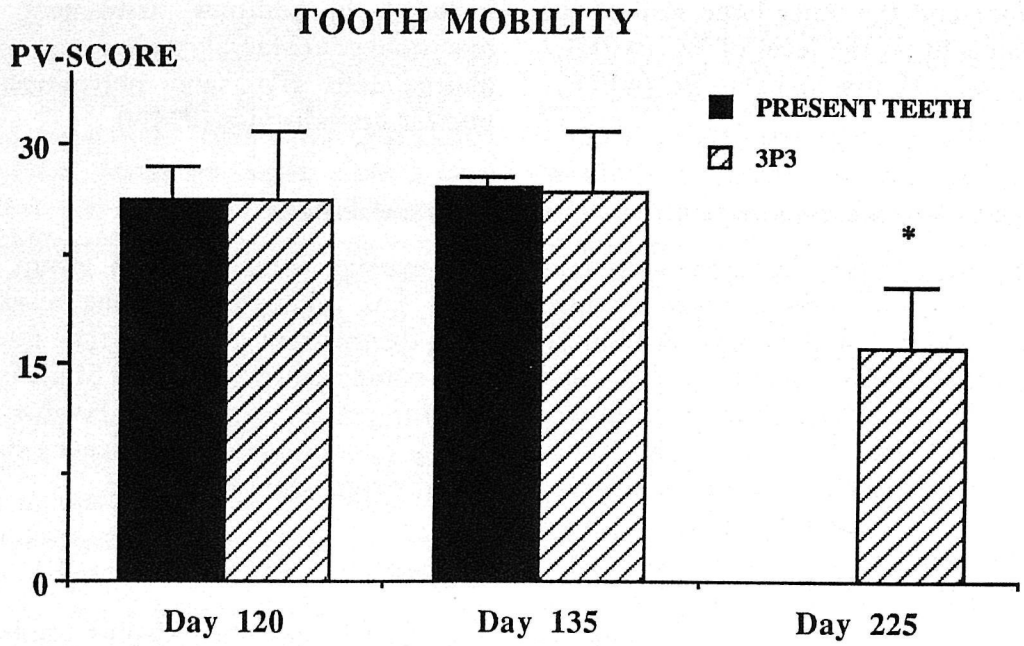

Fig. 3. Results (Periotest Value-PV score) from mobility measurements made at the various observation intervals.
* Statistically significant difference.

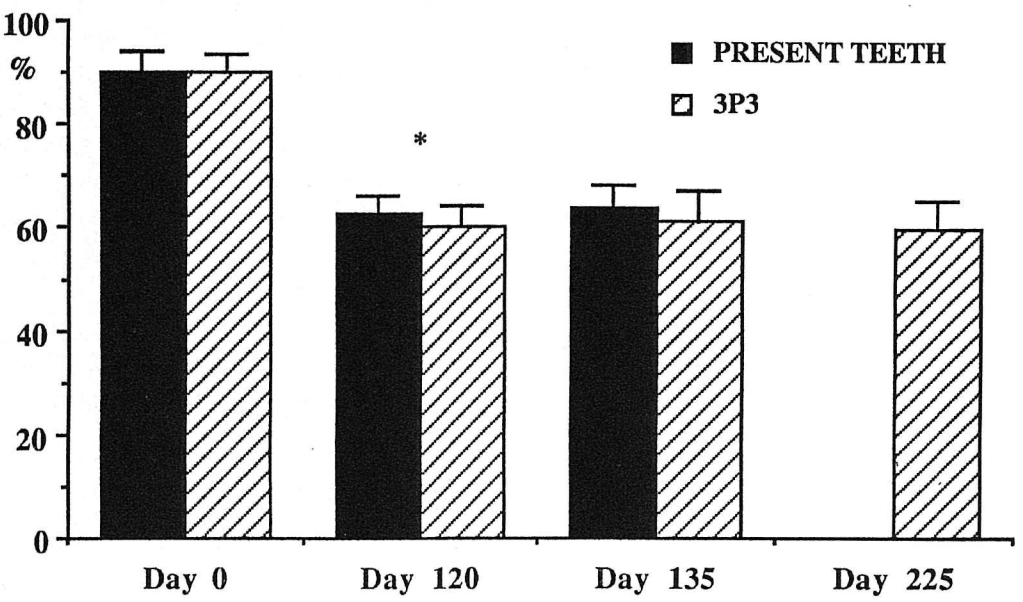

Fig. 4. % of remaining supporting alveolar bone present at the experimental teeth on Days 0, 120, 135 and 225.
* Statistically significant difference.

Table 1. Results from the histometric measurements made in the paraffin embedded sections

	Histometry: mesial root						
	Day 120 ($_2P_2$)		Day 135 ($_4P_4$)			Day 225 ($_3P_3$)	
(mm)	*X*	(SD)	*X*	(SD)		*X*	(SD)
GM–P	1.1	(0.4)	1.2	(0.3)	*	0.8	(0.2)
aPE-P	0.8	(0.2)	0.5	(0.3)	*	0.2	(0.4)
P-BC	0.4	(0.2)	0.6	(0.5)		0.5	(0.4)

Mean value (*X*) and standard deviation (SD). GM-P: distance between the gingival margin and the tip of the probe; aPE-P: distance between the apical cells of the pocket epithelium and the tip of the probe; P-BC: distance between the tip of the probe and the bone crest.
* Statistically significant difference.

(Day 135) and 59.8% (Day 225). Between Days 0 and 120 there was a significant reduction of the height of the alveolar bone, while between Days 120 and 225 the level of the alveolar bone remained unchanged.

Histometric measurements

The results from the histometric assessments are reported in Tables 1, 2.

In the sections representing the mesial root (Table 1) of the experimental teeth the various landmarks (GM, BC and P) could readily be identified. In all sections the tip of the probe was found to be located apical of the termination of the pocket epithelium (aPE). In the sections representing Day 120 ($_2P_2$), the distance between the tip of the probe and the gingival margin (GM-P) was found to be, as an average, 1.1 mm. A further analysis of the specimens revealed that the probe tip inded 0.8 mm apical to aPE (aPE-P) and 0.4 mm coronal to the bone crest (P-BC). The corresponding figures representing Day 135 ($_4P_4$) and Day 225 ($_3P_3$) were 1.2 mm and 0.8 mm (GM-P), 0.5 mm and 0.2 mm (aPE-P) and 0.6 mm and 0.5 mm (P-BC). The distances GM-P and aPE-P were, on Day 225, significantly shorter than the corresponding dimensions on Day 135.

The measurements carried out in the plastic embedded sections (Table 2) revealed that the height of the free gingival unit (GM-aPE) varied between 1.4 mm and 1.6 mm. The distance aPE-BC was, at the various observation intervals, found to vary between 1.2 mm (Day 120) and 1.0 mm (Day 225). The apical extension of the inflammatory cell infiltrate (GM-aICT) was significantly reduced from 1.8 mm (Day 120) to 0.8 (Day 225), while the distance aICT-BC significantly increased from 0.8 mm (Day 120) to 1.9 mm (Day 225). The width of the periodontal ligament, at the level of the bone crest (wPDL/BC), varied between 0.33 mm (Day 120) to 0.24 (Day 225) while wPDL/BC+1 varied between 0.26 mm (Day 120) and 0.21 mm (Day 225).

Morphometric measurements

The results of the morphometric analysis at level 1 are reported in Fig. 5. The free gingival unit representing Day 120 was comprised of about 45% epithelium (OE: 21%; PE: 24%) and 55% connective tissue (NCT: 26%; ICT: 29%). The corresponding figures were 48% epithel-

ble 2. Results from the histometric measurements made in the plastic embedded sections presenting the distal root of the experimental teeth

	Histometry: distal root						
	Day 120 ($_2P_2$)		Day 135 ($_4P_4$)			Day 225 ($_3P_3$)	
(mm)	*X*	(SD)	*X*	(SD)		*X*	(SD)
GM-aPE	1.4	(0.5)	1.4	(0.2)		1.6	(0.3)
aPE-BC	1.2	(0.3)	1.1	(0.2)		1.0	(0.3)
GM-aICT	1.8	(0.5)	1.6	(0.3)	*	0.8	(0.1)
aICT-BC	0.8	(0.2)	0.9	(0.3)	*	1.9	(0.4)
wPDL (BC)	0.33	(0.08)	0.27	(0.08)		0.24	(0.06)
wPDL (BC+1)	0.26	(0.03)	0.21	(0.05)		0.21	(0.06)

Mean value (*X*) and standard deviation (SD). GM-aPE: distance between the gingival margin and the apical cells of the pocket epithelium; aPE-BC: distance between the apical cells of the pocket epithelium and the bone crest; GM-aICT: distance between the gingival margin and the apical level of the inflammatory infiltrate; aICT-BC: distance between the apical level of the inflammatory infiltrate and the bone crest; wPDL/BC, wPDL/BC+1: width of the periodontal ligament space at the bone crest and 1 mm apical.
* Statistically significant difference.

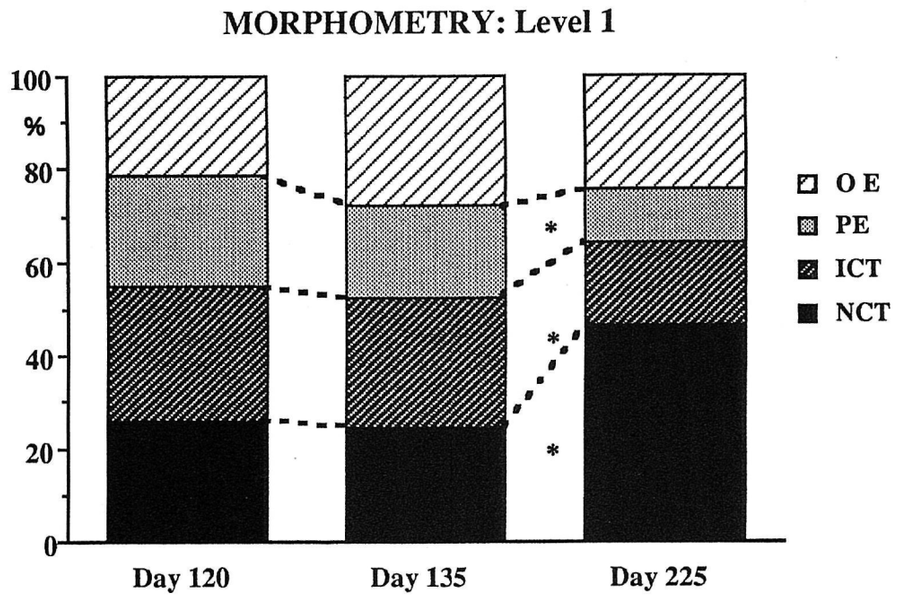

Fig. 5. Composition of the free gingival unit presented as the % of oral epithelium (OE), pocket epithelium (PE), non infiltrated connective tissue (NCT) and infiltrated connective tissue (ICT) at the different observation intervals.
* Statistically significant difference.

ium (OE: 28%; PE: 20%) and 52% connective tissue (NCT: 24%; ICT: 28%) on Day 135 and 35% epithelium (OE: 24%; PE: 11%) and 64% connective tissue (NCT: 46%; ICT: 18%) on Day 225. The gingival units representing Day 225 had significantly smaller proportions of ICT and PE and a larger relative volume of NCT than the corresponding units representing Days 120 and 135. The results from the level 4 assessment of the non-infiltrated supracrestal connective tissue (N-SCT) are reported in Table 3. On Day 120, the N-SCT was comprised of 55% collagen (Co), 9% vascular structures (V), 6% fibroblasts (Fi), 0.3% leukocytes (Leu) and 29% residual tissues (R). On Day 135, the corresponding figures were (56% Co; 11% V, 6% Fi; 0.2% Leu; 26% R). The N-SCT representing Day 225 contained a statistically significant larger volume of collagen (62% Co) but a smaller volume of fibroblasts (4% Fi) and residual tissues (25% R). No leukocytes (Leu) could be identified in the specimens representing Day 225.

The analysis of the data representing the level 4 measurements of the ICT (Fig. 6) revealed that the amount of collagen (Co) increased significantly during the observation period (Day 120: 32%; Day 225: 45%), while there was a significant reduction in the amount of fibroblasts (Fi). The relative amount of inflammatory cells (Mac+Ly+Pc+PMN) in the ICT did not vary between the examination intervals.

Discussion

The present investigation demonstrated that in dogs allowed to form plaque, the placement of cotton floss ligatures at the neck of mandibular premolars initiated a process that resulted in (i) the formation of an inflammatory lesion which extended deep into the supracrestal connective tissue, (ii) extensive loss of alveolar bone, and (iii) markedly increased tooth mobility. These findings confirm data from similar experiments in the rat (Rovin et al. 1966), in the ferret (Fischer & Klinge 1994), in the monkey (e.g., Kennedy & Polson 1973, Polson & Zander 1983, Holt et al. 1988) and in the dog (e.g., Ericsson et al. 1975, 1993, Lindhe & Ericsson 1978) demonstrating that periodontal tissue breakdown in this model is dependent on (i) the mechanical irritation provided by the ligature and (ii) the establishment of a subgingival plaque.

The present findings also revealed that the removal of the ligature and, as a consequence, a substantial portion of the subgingival microbiota, and a thorough supragingival debridement within a 4 month period reduced the size and the apical extension of the inflammatory lesion in the supracrestal connective tissue.

This observation agrees with data previously reported (Lindhe & Ericsson 1978) showing that the ICT within the interdental papilla expressed as percentage of the gingival tissue (i) decreased significantly during a 6 month period following ligature removal in the beagle dog model, and (ii) that this decrease was enhanced if the ligature removal was accompanied by supragingival tooth cleaning measures.

The present findings further demonstrated that the resolution of a gingival lesion which follows antimicrobial therapy is a process that requires several months of healing. Thus, during the first 2 weeks after ligature removal, the size (Fig. 5) as well as the position (Table 2) of the inflammatory lesion changed only to a limited extent while during the subsequent months marked improvements occurred. This observation is in agreement with Lindhe et al. (1978) from a study in the beagle dog in which the structural alterations that characterized the healing gingiva were examined. The authors reported that even if the

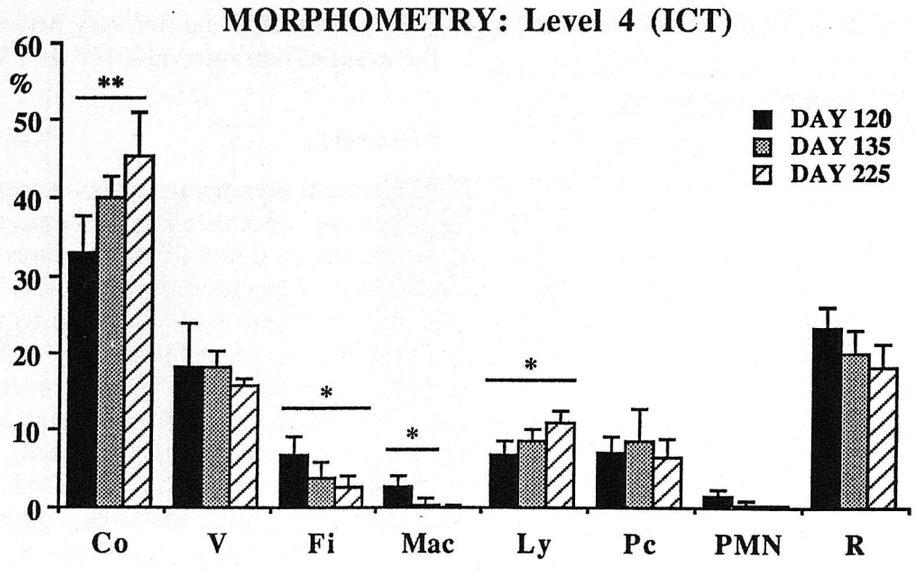

Fig. 6. Composition of the infiltrated connective tissue (ICT) presented as the % of collagen (Co), vascular structures (V), fibroblasts (Fi), macrophages (Mac), lymphocytes (Ly), plasma cells (Pc), polymorphonucleated granulocytes (PMN) and residual tissues (R) at the different observation intervals.
* Statistically significant difference.

Table 3. Composition (level 4 measurements) of the non-infiltrated supracrestal connective tissue (N-SCT) presented as the % of collagen (Co), vascular structures (V), fibroblasts (Fi), leukocytes (Leu) and residual tissues (R) at the different experimental time intervals; mean value (X) and standard deviation (SD)

	Morphometry: level 4 (N-SCT)							
	Day 120 ($_2P_2$)			Day 135 ($_4P_4$)			Day 225 ($_3P_3$)	
%	X	(SD)		X	(SD)		X	(SD)
Co	55.2	(2.9)		56.1	(3.9)	*	62.3	(1.9)
V	8.7	(1.2)	*	11.2	(1.6)		8.1	(2.5)
Fi	6.3	(0.3)		6.2	(1.5)	*	4.4	(0.8)
Leu	0.3	(0.1)		0.2	(0.1)	*	0.0	(0.0)
	↑			*			↑	
R	29.5	(2.1)		26.4	(2.7)		25.2	(1.3)
	↑			*			↑	

* Statistically significant difference.

resolution to the gingival lesions had begun already a few says after subgingival scaling, a progressive reduction of the size of the infiltrate occurred during the entire 6 week period of supragingival plaque control. The present data underline that the process of healing continues also beyond the 6 week interval, a conclusion which is supported by data derived from treatment studies in humans (e.g., Badersten et al. 1984a, b, Listgarten et al. 1978).

From the histometric measurements performed in the present study, it was noted that concomitant with the reduction of the size of the ICT, there was a coronal displacement of the apical border of the inflammatory lesion. As a consequence the height of the non-infiltrated portion of the supracrestal connective tissue was increased, although the position of the marginal bone level and the size of the periodontal ligament remained largely unaltered. The alterations in the soft supracrestal tissue that occurred following ligature removal were accompanied by a marked decrease in the mobility of the experimental teeth. These observations are in agreement with data from previous clinical studies reporting on an association between the resolution of an inflammatory lesion in the gingival tissue and a decrease in the mobility of the associated teeth (e.g., Fehr & Mühlemann 1956, Wüst et al. 1960, Rateitschak 1963, Lindhe & Nyman 1975). Fehr & Mühlemann (1956) in a clinical trial studied the effect of subgingival scaling. They reported that 4 months after initial therapy there was 20% decrease of the mobility of the teeth treated. Wüst et al (1960) noted that scaling combined with selective grinding resulted in a 25% mobility decrease that was maintained for at least 2 years. Rateitschak (1963) studied changes in tooth mobility in 80 patients subjected to various modalities of periodontal therapy. He observed that teeth which initially exhibited markedly increased mobility had their STM-value (Mühlemann 1954) significantly reduced following a treatment program which included "curettage, functional equilibration and gingivectomy". Lindhe & Nyman (1975) studied healing following surgical periodontal therapy in 75 patients with advanced periodontal disease. Following active therapy, all patients were placed in a maintenance care program and were monitored for 5 years. From the clinical and radiographical examination performed at the end of the 5 years, it was noted that although no alteration of the alveolar bone margin had occurred, the number of teeth still showing increased mobility had been reduced from 931 to 422. Persson (1980) studied the tooth mobility in 8 subjects with moderately advanced periodontal disease who were treated with non-surgical therapy. He reported that "during the course of the investigation, a gradual decrease of tooth mobility was observed". In similar studies, Persson (1981a, b) monitored tooth mobility alterations in subjects with moderate to advanced periodontitis, who were exposed to pocket elimination procedures: gingivectomy and flap surgery. In these trials Persson detected that in the majority of the teeth treated the mobility was (i) enhanced immediately after therapy but (ii) was subsequently reduced to levels below baseline values. On the other hand, it has also been reported that physiological tooth mobility is "not increased by gingivitis, and correspondingly not influenced by local treatment" (Donzé et al. 1973). Based on the findings from the present study and the clinical trials referred to, it seems justified to conclude that at sites with periodontal tissue destruction the condition of the supraalveolar tissue, e.g., presence and size of an inflammatory lesion, amount of collagen in the supracrestal connective tissue etc. plays an important role for the mobility characteristics of the associated teeth.

At the end of the experimental period, the periodontal tissues exhibited an increased resistance to probing. This was illustrated by the findings from the

istometric measurements which disclosed reduced values of GM-P and PE-P (Table 1) and an increased length of the distance between the tip of the probe and the bone crest (P-BC; Table). This observation is in strict agreement with previous findings demonstrating that there is a close relationship between the degree of inflammation in the gingiva and the histological probing depth (Armitage et al. 1977, Jansen et al. 1981, Robinson & Vitek 1979, Magnusson & Listgarten 1980). On the other hand, in the present material, not only was the gingival lesion reduced in size and position but the mobility of the experimental teeth was also decreased. This is also a factor which must be considered when the results from the probing depth measurements are evaluated. Neiderud et al. (1992) recently reported from a Beagle dog study that non-inflammatory tissue alterations that occur at mobile teeth may reduce the resistance offered by the periodontal tissues to probing. Thus, the reduced penetration of the probe that occurred at the end of the present experiment may be the net effect of (i) changes in size and position of the gingival infiltrate, and (ii) reduced mobility of the experimental teeth.

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Zusammenfassung

Zahnlockerung und Rückbildung experimenteller Parodontitis. Eine Experimentalstudie am Hund

Mit der vorliegenden Arbeit wurde beabsichtigt, Veränderungen der Zahnmobilität zu studieren, die am Hund während der Rückbildung experimentell induzierter Parodontitisläsionen vorkamen. Die Versuche wurden an 5 einjährigen Beaglehunden vorgenommen. Zu Versuchszähnen wurden die linken und rechten 4., 3. und 2. Unterkieferprömolaren ($_4P_4$, $_3P_3$, $_2P_2$) bestimmt. Durch Anbringen plaquesammelnder Ligaturen aus Zahnseide um den Zahnhals der Versuchszähne, wurde ein Abbau parodontaler Gewebe eingeleitet. Einmal im Monat wurden die Ligaturen erneuert und in Höhe der retrahierenden Ebene des gingivalen Randes angebracht. Am Versuchstag 120 wurden Ligaturen und oberflächliche Nekrosen entfernt. In die mesiobukkale Oberfläche der Zahnkronen $_2P$ und P_2 wurde, parallel zur Längsachse der mesialen Wurzel, eine Rille präpariert. Geführt durch die Rille, wurde eine Sonde mit der Druckkraft von 0.5 *N* in die bukkale Zahnfleischtasche der mesialen Wurzel eingebracht und an die bukkale Zahnoberfläche angelegt. Biopsien, die die mesiale und distale Wurzel des $_2P$ und P_2, wie auch die umgebenden Hart- und Weichgewebe enthielten, wurden entnommen. Die Entnahme wurde 15 Tage nach der Ligaturentfernung (d.h. am Tage 135) und 3 Monate danach (d.h. am Tage 225) in den Regionen der 4. and 3. Prämolaren ($_4P_4$ und $_3P_3$) in ähnlicher Form wiederholt. Nach Fixation, Dekalzifizierung und der Anfertigung von Schnittpräparaten wurde das Biopsiematerial histometrisch und morphometrisch vermessen. Die Beweglichkeit der Versuchszähne wurde an den Versuchstagen 120, 135 und 225 mit dem Periotest System beurteilt. Die Menge verbliebener Knochenstütze der Versuchszähne wurde mit Hilfe standardisierter Röntgenaufnahmen bewertet. Die Resultate des vorliegenden Versuchs zeigten, daß bei Hunden mit ungehinderter Plaquebildung, durch Anbringen von Zahnseideligaturen um die Zahnhälse ein Prozess eingeleitet wurde, der (i) eine tief in das supraalveoläre Bindegewebe eindringende, entzündliche Läsion verursachte sowie (ii) extensiven Verlust von alveolärem Knochen und (iii) deutlich erhöhte Zahnlockerung zur Folge hatte. Weiterhin wurde beobachtet, daß durch die Entfernung der Ligatur und als Folge der Entfernung einer bedeutenden Menge subgingivaler Mikrobiota, in dem supraalveolären Bindegewebe innerhalb von 4 Monaten eine Reduktion des Umfangs und der apikale Ausbreitung der entzündlichen Läsion stattfand. Die Veränderungen in dem supraalveolären Gewebe gingen mit einer deutlichen Herabsetzung der Beweglichkeit der Versuchszähne und einer Verringerung der sondierten Taschentiefe einher. Es scheint, daß die reduzierte Sondenpenetration das Ergebnis einer Veränderung der Größe und der Lage des Infiltrates, wie auch der herabgesetzten Mobilität der Versuchszähne war.

Résumé

Mobilité dentaire et guérison de la parodontite expérimentale. Une étude expérimentale chez le chien

Le but de cette étude a été d'analyser les altérations de la mobilité dentaire qui survenaient au cours de la guérison de la parodontite induite expérimentalement chez le chien. Cinq chiens briquet âgés d'un an ont participé à cette étude. Les deuxièmes, troisièmes et quatrièmes prémolaires inférieures ($_2P_2$, $_3P_3$, $_4P_4$) ont servi de dents expérimentales. La destruction du tissu parodontal a été induite en placant autour du collet de ces dents des ligatures en coton accumulant la plaque dentaire. Une fois par mois, les ligatures ont été replacées au niveau de la récession de la gencive marginale . Au jour 120, les ligatures ont été ôtées et un lissage radiculaire a été effectué. Une encoche placée parallèlement à l'axe longitudinal de la racine mésiale a été taillée le long de la surface mésio-vestibulaire des couronnes des $_2P$ et P_2. Guidée par l'encoche et avec une force de sondage de 0.5 *N* une sonde a été insérée dans la poche gingivale vestibulaire de la racine mésiale puis attachée à la surface vestibulaire. Des biopsies comprenant tant les racines mésiales et distales des $_2P$ et P_2 que les tissus mous et durs avoisinants ont été prélevées. Ce procédé de biopsie a été répété 15 jours (J 135) et 3 mois (J 225) après l'enlèvement de la ligature au niveau des troisièmes ($_3P_3$) et quatrièmes ($_4P_4$) prémolaires. Après fixation, décalcification et découpage, le matériel de biopsie a été utilisé pour des mesures histométriques et morphométriques. L'enregistrement de la mobilité des dents expérimentales a été effectué aux jours 120, 135 et 225 en utilisant le système du Périotest. La quantité d'os entourant les dents expérimentales a été évaluée à partir de radiographies standards. Les résultats de l'expérience ont montré que chez les chiens où la plaque dentaire peut s'accumuler, le placement de ligature en coton autour de prémolaires mandibulaires démarre un processus qui résulte en (1) la formation d'une lésion inflammatoire qui descend profondément dans le tissu conjonctif sus-crestal, (2) une abondante perte d'os alvéolaire et (3) une augmentation marquée de la mobilité dentaire. Il a été également observé que l'enlèvement de la ligature et, par voie de conséquence, la disparition d'une importante portion de la flore microbienne, réduit l'étendue et l'extension apicale de la lésion inflammatoire dans le tissu conjonctif sus-crestal, dans les 4 mois. Les altérations dans le tissu mou sus-crestal étaient accompagnées d'une diminution marquée de la mobilité des dents expérimentales et d'une réduction de la profondeur des poches au sondage. La réduction de la pénétration de la sonde était sans doute tant le résultat de la variation de l'étendue et de la position de l'infiltrat que celui de la diminution de la mobilité des dents expérimentales.

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