Trauma and progression of marginal periodontitis in squirrel monkeys

IV. Reversibility of bone loss due to trauma alone and trauma superimposed upon periodontitis

A. M. POLSON, S. W. MEITNER, AND H. A. ZANDER

Department of Periodontology, Eastman Dental Center
Rochester, New York, U. S. A.

Previous studies have shown that repeated trauma to the interproximal periodontium induced by jiggling of teeth resulted in alveolar bone loss and, when combined with periodontitis, increased the amount of bone loss compared with periodontitis alone. This study was designed to answer the question on the reversibility of such bone loss. Eight squirrel monkeys were divided into two equal groups. In four animals, the mandibular second and third bicuspids were jiggled mesio-distally for ten weeks. On one side a marginal periodontitis was induced at the same time. In the other four animals the same procedures were instituted except these animals were killed ten weeks after jiggling had been stopped. Step-s serial histological sections of the coronal interproximal area were analyzed. Loss in height of alveolar bone and percentage of bone occupying the coronal area were measured histometrically. When jiggling was stopped, a significant reversibility of bone loss occurred. However, no such reversal was seen in the presence of periodontitis. It was postulated that presence of an existing marginal inflammation inhibited the potential for bone regeneration.

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**Introduction**

Repetitive trauma to the periodontium incident to orthodontic tooth movement and occlusal disharmonies results in increased tooth mobility. Histologic examination of such specimens shows that trauma alone does not produce periodontal pocketing as judged by apical migration of the junctional epithelium along the root surface due to loss of connective tissue attachment (Waerhaug 1955, Wentz, Jarabak & Orban 1958, Svanberg 1974, Polson, Meitner & Zander 1976). Although trauma does not produce any loss of connective tissue attachment, it has a significant effect upon the alveolar bone. Interproximal alveolar bone adapts to repetitive trauma by becoming less dense and losing crestal height (Polson et al. 1976).

When a jiggling type of trauma was produced subjacent to experimental marginal periodontitis in squirrel monkeys, the trauma had no effect on connective tissue attachment (Meitner 1975). However, the amount of interproximal bone loss which occurred with periodontitis was increased by repetitive trauma. The purpose of this investigation was to see whether the bone loss due to trauma alone, and the increased bone loss
due to repeated trauma superimposed upon marginal periodontitis, are reversible when the trauma is discontinued.

**Materials and Methods**

The mandibular second and third bicuspid of eight young adult squirrel monkeys were jiggled mesio-distally by a method previously described (Polson et al. 1976). At the same time as jiggling was begun, marginal periodontitis was induced on one side of the mandible using the method described by Kennedy & Polson (1973).

After ten weeks of jiggling, four of the eight animals were killed. At this time, the jiggling was stopped in the four remaining animals, and these animals were killed ten weeks later. The mobility of the bicuspid was assessed (Polson et al. 1976) immediately prior to the start of the investigation, and at ten week intervals thereafter.

Immediately after death of the animal, the mandible was dissected out and placed in 10 percent neutral buffered formalin fixative. Subsequent histological preparation and sectioning of the interproximal area between the second and third bicuspid was as reported earlier (Polson 1974).

The coronal interproximal area was examined histologically. The distance from the crest of the alveolar bone to the cemento-enamel junctions and the percentage of bone in the coronal periodontium was measured histometrically (Meitner 1975).

**Results**

**Clinical**

The bicuspid which had been subjected to ten weeks of trauma alone were mobile in mesio-distal and bucco-lingual directions. The gingiva had minimal inflammation and was essentially the same as before the jiggling. After jiggling had been stopped for ten weeks, the most striking clinical altera-

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**Table 1**

Location of crestal alveolar bone (CEJ-A) (Mean ± Standard Error, Microns)

<table>
<thead>
<tr>
<th>Surface</th>
<th>10 weeks N = 4</th>
<th>10 weeks after cessation of jiggling N = 4</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal second bicuspid</td>
<td>458.6 ± 68.8</td>
<td>378.4 ± 41.6</td>
<td>319.5 ± 23.9*</td>
</tr>
<tr>
<td>Mesial third bicuspid</td>
<td>388.5 ± 63.5</td>
<td>317.4 ± 46.3</td>
<td>256.3 ± 13.8*</td>
</tr>
<tr>
<td>Periodontitis alone</td>
<td></td>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>Distal second bicuspid</td>
<td>1228.3 ± 175.6</td>
<td>1229.0 ± 123.8</td>
<td>960.4 ± 53.4**</td>
</tr>
<tr>
<td>Mesial third bicuspid</td>
<td>1045.7 ± 110.7</td>
<td>1042.3 ± 38.1</td>
<td>933.7 ± 35.6**</td>
</tr>
</tbody>
</table>

* Values from Polson et al. 1976.
** Values from Meitner, 1975.
tion was the absence of detectable mobility of the bicusps. There was, however, no difference in the clinical appearance of the gingival tissues.

After ten weeks of jiggling in combination with marginal periodontitis, the bicusps were extremely mobile and could be displaced mesio-distally, bucco-lingually and vertically. The marginal tissues around this interproximal area were very inflamed, enlarged, and tended to bleed spontaneously. The mobility of these bicusps was just as pronounced ten weeks after the jiggling had been discontinued. The teeth were splayed in different directions, the contacts were open, and the marginal tissues continued to show severe inflammation.

Histologic and Histometric
The repeated trauma produced by alternate mesio-distal jiggling of the bicusps for ten weeks did not produce any loss of connective tissue attachment and the alveolar bone consisted of a number of islands surrounded by a loosely arranged highly cellular and vascular connective tissue (Figs. 1, 2). There had been a loss in height of the alveolar bone (Table 1), and a considerable reduction in the percentage of osseous tissue between the jiggled bicusps (Table 2).

Table 2
Percentage of osseous tissue in coronal interproximal area
(Mean ± Standard Error)

<table>
<thead>
<tr>
<th>Time</th>
<th>10 weeks</th>
<th>10 weeks after cessation of jiggling</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jiggling alone</td>
<td>30.6 ± 1.46</td>
<td>42.1 ± 1.54</td>
<td>5.43</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Periodontitis plus jiggling</td>
<td>16.8 ± 1.9</td>
<td>15.1 ± 0.64</td>
<td>1.33</td>
<td>N. S.</td>
</tr>
</tbody>
</table>

* Values from Polson et al. 1976.
** Values from Meitner, 1976.
rower periodontal ligament adjacent to the third bicuspid was due to the mesial displacement of this tooth at the time of animal sacrifice. The interproximal alveolar septum was thin, and its crest was a greater distance from the most apical cell of the junctional epithelium than is seen normally.

The interproximal areas of specimens obtained ten weeks after discontinuing trauma in the presence of existing marginal periodontitis appeared essentially the same as those obtained at the moment the trauma was stopped (Fig. 7). The histometric measurements of the location of the alveolar crest, and the percentage of interproximal bone from these specimens in which trauma had been discontinued, were virtually identical with those obtained from specimens after ten weeks of periodontitis plus active trauma (Tables 1 and 2).

Discussion

In those interproximal areas which had been subjected to trauma alone, extensive bone regeneration occurred during the ten weeks after cessation of the trauma. The ability of the supporting alveolar bone to remodel under the influence of traumatic forces to the teeth has been well documented (Gottlieb & Orban 1931, Wentz, Jarabak & Orban 1958, Waerhaug & Hansen 1966, Svanberg...
& Lindhe 1973, Polson et al. 1976). The degree to which these changes are reversible after discontinuation of the trauma has not previously been established and quantitated. It would appear from the histologic observations of this study that alveolar bone which has undergone marked adaptive alterations in response to environmental forces retains tremendous innate potential for regeneration and return to normal morphology. The alveolar bone morphology in these specimens closely resembled the normal (Polson et al. 1976). However, the quantitative histometric measurements showed that the coronal regeneration of bone and the overall percentage of interproximal bone had not reached the levels normally present.

This means either that more than ten weeks are necessary for complete regeneration after traumatic changes of this magnitude, or that there may be a small degree of irreversibility in the bone loss. The amount by which the regenerated alveolar bone dimensions differed from the normal values is probably not of any clinical significance since the loose teeth tightened to the point where there was no longer any discernible mobility.

In marked contrast to these findings, when trauma was discontinued in the presence of marginal periodontitis, the histologic and histometric results showed that there was no regeneration of alveolar bone. When the dimensions of the alveolar bone...
were compared with those in periodontitis alone for the same period of time, the alveolar crest was found to be more apical (Table 1) and there was 50 percent less bone in the periodontitis specimens which had received initial trauma followed by a period without trauma (Table 2). In view of the pattern of bone loss and regeneration with trauma alone, it could be expected that, if marginal periodontitis and trauma were unrelated factors, bone regeneration should have taken place to the level found with periodontitis alone for the same period of time. However, not only did this not happen, but no evidence of bone regeneration was seen.

In marginal periodontitis the junctional epithelium is always located apical to the cemento-enamel junction indicating loss of connective tissue attachment to the root surface. There is a concomitant loss of crestal alveolar bone which apparently maintains the original distance between the epithelial cells and bone. Perhaps, therefore, the apical proliferation of the junctional epithelium in the periodontitis specimens which had received trauma acted as an inhibitory factor to coronal bone regeneration. There is evidence, however, which makes this explanation unlikely.

When Meitner (1975), using this same animal model system, produced a jiggling
casts serious doubt on the existence of an inhibitory effect of junctional epithelium upon coronal regeneration of alveolar bone has recently been reported by Caton & Zander (1976). After curettage of an experimentally produced infrabony pocket in a rhesus monkey, coronal regeneration of bone occurred without any new connective tissue attachment. This resulted in the crest of the bone being a considerable distance corona] to the apical portion of the junctional epithelium. This finding emphasizes the fact that it is unlikely that the location of the junctional epithelium was the critical factor in preventing the coronal regeneration of bone after the cessation of trauma in the presence of periodontitis.

Secondary occlusal trauma has been described as "the effect on already reduced or weakened periodontal structures of occlusal forces which may or may not be abnormal but are excessive for reduced or weakened supporting structures" (Ramfjord & Ash 1968). It is possible, perhaps, that the occlusal forces in our animal model when superimposed upon periodontal structures damaged and reduced by marginal periodontitis and trauma, were enough to maintain the degree of tooth displacement and thereby prevent bone regeneration. If this were true, then immobilization of the teeth by splinting after withdrawal of the trauma would be necessary to maximize the healing potential. There are two reasons why this is probably not the case. Firstly, the degree of tooth displacement produced by alternating the wedge position was considerable and greater than that which can be induced by occlusal factors. Secondly, at the end of the phase of active trauma, the teeth which received only trauma were almost as mobile as those subjected to the combination of periodontitis and trauma. However, these teeth became firm and bone regeneration occurred in the weeks subsequent to removal of trauma. It is probable, therefore, that

Fig. 7. Interproximal area between second (2) and third (3) bicusps ten weeks after jiggling had been discontinued in the presence of existing marginal inflammation. H & E stain. Orig. Mag. × 16.

type of trauma adjacent to existing marginal periodontitis, he found increased loss of alveolar bone, but no increased loss of connective tissue attachment compared with periodontitis without the trauma. Therefore, there was a greater distance than was usual between the junctional epithelium and the crestal bone. This was analogous to the situation in our study at the moment trauma was discontinued in the presence of periodontitis. Consequently, there was an area of connective tissue into which bone could have regenerated without encroaching upon this apparent minimum dimension between epithelium and bone.

Another strong piece of evidence which
secondary occlusal trauma was not the factor responsible for preventing bone regeneration after withdrawal of the jiggling forces in the presence of marginal periodontitis.

Gingival inflammation is characterized histologically by a cell-rich inflammatory exudate in the connective tissue subjacent to the epithelium which lines the gingival sulcus and forms the junctional epithelium (Oliver, Holm-Pedersen & Loe 1969, Schroeder 1970). The inflammatory response is the host reaction to the presence of bacterial plaque in the neighborhood of the gingival sulcus. From the earliest stages of periodontitis, this cellular exudate spreads into the coronal alveolar bone and bone resorption takes place within the marrow spaces (Weinman 1941, Goldman 1957, Akiyoshi & Mori 1967). In the specimens in which trauma was discontinued in the presence of periodontitis, the coronal alveolar bone was similarly involved due to the existing marginal periodontitis. Perhaps the presence of this inflammation inhibited the potential for bone regeneration. This possibility has been alluded to by Glickman in discussing the management of a periodontium which is suffering from both inflammation and trauma from occlusion (Glickman 1971). He stated that, if the occlusal factors are eliminated first, “the inflammation-induced degeneration may interfere with the periodontal repair that is the goal of the occlusal adjustment.” There is no scientific evidence to substantiate this statement. However, with the aid of our animal model in which we are able to control the factors of marginal periodontitis and jiggling trauma, both singly and in combination, it will be possible to provide the biologic information which is necessary to test this hypothesis.

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Address:
Department of Periodontology
Eastman Dental Center
800 Main Street E.
Rochester, New York 14603
U. S. A.
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