Interrelationship of inflammation and tooth mobility (trauma) in pathogenesis of periodontal disease

ALAN M. POLSON

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

Abstract. A series of studies has investigated interactions between periodontal trauma and marginal periodontitis in relation to the initiation, progression and treatment of periodontal disease. Lesions of trauma in the periodontal ligament consequent to either single or jiggling displacing forces result in morphologic alterations in the ligament and alveolar bone. These changes do not initiate the loss of connective tissue attachment characteristic of marginal periodontitis. Studies conducted in squirrel monkeys and beagle dogs in which jiggling forces have been produced subjacent to an established marginal periodontitis reported increased loss of alveolar bone, but the accelerated loss of attachment which occurred in the dog model did not occur in the monkey model. To clarify the relative importance of inflammation and tooth mobility in the treatment of advanced periodontal disease, periodontal response was evaluated after removing traumatic and/or inflammatory components. Elimination of trauma in the presence of existing marginal inflammation did not reduce tooth mobility or increase bone volume. Osseous regeneration and decreased tooth mobility occurred after resolving both components, however, similar findings occurred after resolving inflammation in the presence of continued tooth mobility. Resolution of marginal inflammation is of prime importance in the management of periodontal disease. After resolution of inflammation, bone regeneration may occur around mobile teeth and, furthermore, any residual tooth mobility does not result in increased loss of connective tissue attachment.

A series of investigations has been in progress designed to evaluate interactions between periodontal trauma and marginal periodontitis from the perspective of periodontal disease initiation, progression, and treatment (Polson et al. 1974, 1976a, b, 1979, Polson 1974, Meitner 1975, Kantor et al. 1976, Polson & Zander 1979a, b). The present paper reviews this series, and discusses possible clinical implications. The results from the investigations in relation to the questions posed are not final answers in themselves, but should be interpreted with respect to previous and current work done in this field by other investigators.

Traumatic lesions in the periodontium are the consequence of forces applied to the tooth which displace the tooth in its socket. The type of clinical force which produces this lesion can vary - occlusal forces, orthodontic forces, or a physical blow to the tooth. The most important thing to rea-
lize is that, irrespective of the nature of the displacing force, the histologic lesion which results in the periodontal ligament is similar. The presence or absence of such lesions, and their subsequent reactions, can be verified only by histologic examination.

The area of critical importance with regard to the initiation and progression of periodontal disease is the dento-gingival junction. In the normal periodontium, the apical cells of the junctional epithelium are located at the cemento-enamel junction, the area where the connective tissue attachment begins. In established periodontal disease, the termination of the junction epithelium is on the cementum surface, apical to the cemento-enamel junction, and the distance from the cemento-enamel junction to the apical cells of the junctional epithelium is referred to as the loss of connective tissue attachment. This loss of connective tissue attachment is accompanied by loss of crestal alveolar bone. When the possibility is considered of traumatic lesions as acting initiators of periodontitis, they must be evaluated with respect to the potential for producing the loss of attachment and loss of alveolar bone characteristic of marginal periodontitis.

It has been possible, using the squirrel monkey as an animal model, to develop controlled, reproducible methodologies for producing single and multiple (due to jiggling) traumatic lesions in the periodontal ligaments and evaluate the consequences of these lesions. A single, severe episode of trauma produced by mechanically displacing the tooth in one direction, resulted in compression of the coronal periodontal ligament (Polson & Zander 1979a). The compression produced ischemia, and the ischemia resulted in an area of necrosis characterized by acellularity and obliteration of the vasculature. Even though such a lesion may be situated in the most coronal portion of the periodontal ligament, the supracrestal tissues were always unaffected. As a consequence of the traumatic lesion, bone resorption occurred to widen the periodontal ligament space, and the acellular area revascularized and repopulated. After the stimulus for resorption had ceased, deposition of new bone began on the resorbed bone surfaces, signifying transition from a resorptive phase to a reparative phase. Bone formation continued until the original morphology of the alveolar bone had been restored. At this time, the periodontal ligament which had been acellular several weeks earlier as a result of the severe episode of trauma, now had normal orientation and cellularity. All these changes took place without any loss of connective tissue attachment to the root surface. Thus it appeared that a single, severe episode of periodontal ligament trauma initiated neither periodontal pocket formation nor produced an irreversible loss of coronal alveolar bone.

In a clinical situation, however, it is much more likely that the forces acting on a tooth will be alternating one direction and then another (jiggling forces) rather than in a single direction. In order to investigate the periodontal response in such a situation, the mandibular second and third bicuspid in the squirrel monkeys were jigged mesiodistally and the area between them studied (Polson et al. 1976a). The mesio-distal displacement was done by placing a separator between the third bicuspid and first molar which had the effect of displacing the teeth in a mesial direction. Forty-eight hours later, the separator was removed and a new one placed between the first and second bicuspid which had the effect of displacing the teeth distally. This sequence was repeated every 48 h. It should be noted that the contact area between the second and third bicuspid remained closed at all times, thereby preventing any damage due to food impaction and, therefore, the interproximal periodontium between these teeth (i.e. the
area under investigation) was subjected to only the alternating periodontal stresses.

After 2 weeks of the jiggling forces, no loss of connective tissue attachment had occurred. However, the appearance of the alveolar bone and individual periodontal ligaments differed considerably from the normal. The alveolar bone now consisted largely of marrow spaces which opened through widened channels into the adjacent periodontal ligaments. At the time the animals were killed, one of the ligaments was under compression, and the other under tension. The area under compression showed all the characteristic changes described earlier and, furthermore, extensive undermining (rear) resorption was taking place in the adjacent alveolar bone as evidenced by the presence of many multinucleated osteoclasts. In contrast to this, the periodontal ligament under tension was widened, highly cellular, and had dilated blood vessels.

After 10 weeks of the jiggling forces, there had still been no loss of connective tissue attachment. However, the interproximal periodontium differed in its appearance compared with the 2-week observations, and the normal specimens. The alveolar bone now had the appearance of a number of "islands" surrounded by a highly vascular and cellular connective tissue. Although these regions of bone appeared as discrete islands, they are, in fact, portions of bony trabeculae which course through the body of the alveolar process. Examination of the surfaces of these osseous islands showed that osteoclasts were few, indicating that resorption was no longer a dominant feature. The periodontal ligaments under compression did not show areas of acellularity, while the tension ligament was again widened and highly cellular. It is probable that the overall histological appearance at the 10-week time point represented one of adaptation to the traumatic forces applied to the teeth. Clinically the teeth were mobile in mesio-distal, bucco-lingual, and vertical directions.

In order to quantitate the osseous changes, the distance from the cemento-enamel junction to the crest of the bone was measured and, in addition, the percentage of bone in the coronal interproximal region was evaluated using a grid-point intersection system. These dimensions were compared with corresponding measurements from normal interproximal regions, and the results showed that, as a consequence of the repeated jiggling forces, there had been some loss in height of crestal alveolar bone and a considerable reduction in the overall volume of interproximal bone. In fact, the mineralized bone volume had been reduced by almost 40%.

When the findings from the results with jiggling forces were taken into consideration with those from a single, severe episode of trauma, it was apparent that neither single nor repeated episodes of trauma produced loss of connective tissue attachment. A jiggling trauma caused some loss in height of crestal bone, and a considerable loss in overall volume of alveolar bone. When the results of these investigations are compared to those of other workers in this field, the evidence is conclusive that traumatic lesions or their sequelae in the periodontal ligament will not produce periodontal pocketing.

In order to investigate the potential for reversibility of the periodontal ligament and alveolar bone changes subsequent to the jiggling forces, normal interproximal periodontia were subjected to 10 weeks of jiggling whereupon the jiggling was stopped, and the areas examined 10 weeks later (Polson et al. 1976b). A marked regeneration of alveolar bone occurred, and the interproximal area resembled that of a normal specimen. At close examination of the coronal bone, areas could be delineated
which corresponded to the osseous islands present in specimens after 10 weeks of jiggling. These had become surrounded by new bone which had been formed in an attempt to reconstitute the interproximal periodontium. The individual periodontal ligaments had regained a normal orientation and cellularity. Thus, it appeared that the adaptive changes which had taken place in the periodontal ligament were reversible, and that the interproximal bone loss due to the jiggling trauma alone was reversible.

The next question in the series of investigations considered the possible influence of periodontal trauma upon an existing marginal periodontitis. The definite possibility of an interaction had been alluded to first by Glickman (1963) when he hypothesized that, in certain situations, inflammation and trauma from occlusion could act as co-destructive factors in periodontitis. In order to describe this hypothesis, he divided the marginal periodontium into a Zone of Irritation and Zone of Co-destruction. The Zone of Irritation began with the gingival papilla and extended to the gingival-free fibers. Plaque and calculus on the tooth surface adjacent to the papilla caused an inflammatory response in the adjacent Zone of Irritation. Traumatic lesions in the periodontal ligament did not affect the Zone of Irritation. However, when inflammation had spread to involve the deeper supporting structures and gingivitis had become periodontitis, the inflammatory and traumatic lesions were adjacent to each other. As a consequence, an interaction occurred which produced an accelerated rate of periodontitis progression with formation of infrabony pockets and angular bony defects. Soon after the inception of this theory, several investigators tried to reproduce Glickman's findings (Comar et al. 1969, Carranza et al. 1971, Kenney 1971). Although all were unsuccessful, the studies were criticized for failing to meet the necessary co-destructive factor criteria. The inflammation was either only at a gingivitis stage, or it was not demonstrated at any stage of the investigations that the lesions of marginal periodontitis and trauma were adjacent to each other. The nature of marginal periodontitis, and the fact that traumatic lesions can be demonstrated only by histological evaluation, emphasized the need for controlled investigations using animal models to test the hypothesis.

A model for experimental marginal periodontitis had been defined in the squirrel monkey (Kennedy & Polson 1973). Periodontitis was induced by tying silk ligatures at the gingival margin to accumulate plaque and hold it in apposition to the gingival tissues. Several weeks later an established marginal periodontitis was present characterized by loss of connective tissue attachment, densely infiltrated supracrestal connective tissue, and loss of alveolar bone. The progression of the periodontitis with time had been documented by measuring the loss of attachment and the loss of alveolar bone in relation to a fixed point, the cemento-enamel junction. This animal model appeared suitable for investigations designed to evaluate the effect of traumatic episodes upon the progression of marginal periodontitis.

In the first of these investigations, a single, severe episode of trauma was produced subjacent to an established periodontitis (Polson 1974). Specimens obtained at the time of production of the traumatic lesion clearly showed an area of acellularity in the coronal periodontal ligament immediately subjacent to the inflammatory infiltrate associated with the periodontitis. Thus, the essential components for a co-destructive effect had been produced. Three weeks later, the acellular area had repopulated, bone resorption had increased the width of the ligament, and the presence of osteoid on this resorbed bone surface indi-
icated that repair had begun. New bone formation was more evident 8 weeks after
injury and at this time, the overall appearance of the periodontal ligament was simi-
lar to the ligament of a tooth (control) which had not received an injury but had
periodontitis for the same period of time. The histometric analysis showed that there
was no difference in loss of connective tissue attachment and loss of alveolar bone
between the groups, indicating that the presence of a single, severe episode of
trauma had not influenced the progression of periodontitis.

The effect of a jiggling trauma on marginal periodontitis was evaluated in an
experimental design which compared marginal periodontitis alone for 20 weeks with
marginal periodontitis for 20 weeks accompanied by a jiggling trauma for the last 10
weeks of this time (Meitner 1975). Radiographic comparison between the groups
showed that where the jiggling had been present in addition to the periodontitis,
there had been greater loss in height of the alveolar bone, the interproximal septum
was narrower and tapered toward the crest and the periodontal ligaments were wider.
Histologic and histometric analysis of the interproximal areas verified the greater loss
of alveolar bone. However, when the loss of connective tissue attachment dimensions
were compared between the groups, there was no difference in three out of four pairs
of surfaces examined, indicating that it was unlikely jiggling trauma superimposed on
the periodontitis had accelerated the loss of connective tissue attachment.

At the same time these investigations were being done in the squirrel monkey, the
same questions regarding possible co-
destructive factor effects were being in-
v estigated using the beagle dog as an ex-
perimental model (Lindhe & Svanberg
1974). The experimental tooth was the man-
dibular fourth premolar and trauma was
produced by means of a splint cemented to
the maxillary teeth. This cap splint had an
inclined plane which made primary oc-
clusal contact with the distal surface of the
fourth premolar. The direction of the in-
clined plane was such that essentially hor-
izontal forces were exerted on the tooth.
On the lingual side of the fourth premolar,
a lingual spring was fitted and attached to
a lingual bar. This spring was activated in
such a way that, as the teeth disoccluded,
the spring would return the mandibular
fourth premolar to its original position. Ex-
perimental periodontitis was produced on
the mesial aspect of the fourth premolar by
a combination of surgery and plaque reten-
tion. A localized, infrabony defect was pro-
duced surgically by removing the alveolar
bone adjacent to the mesial surface. A
notch was made on the tooth surface at the
bottom of the defect. This would serve to
act as a histologic marker for the analysis.
A copper band was fitted to enable epithelialization to occur, and after 3 weeks, it
was removed and replaced by a plaque-
retaining rubber band fitted at the cemento-
enamel junction. Four weeks later, the
jiggling was begun on the fourth premolar
on one side of the jaws while the contra-
 lateral, corresponding tooth served as its
control with marginal periodontitis alone.
The radiographic appearance at the end of
the 6 months of jiggling showed, when
 trauma was present, at dramatic difference
in the alveolar bone morphology at the
crestal and apical level. Histometric com-
parison of the location of the attachment
level between experimental and control
specimen showed that, whenever trauma
had been present, there was greater loss of
connective tissue attachment.

It was apparent that, from the investiga-
tions conducted in squirrel monkeys and
beagle dogs, trauma in association with
periodontitis increased the amount of alveo-
lar bone loss. However, the investigations
reported different findings with respect to loss of connective tissue attachment. The reasons for these differences may be speculated upon. For example, there were different animal models, different methods of inducing experimental periodontitis, and different methods for inducing jiggling forces. It is possible that, in the beagle dog studies, the forces were of greater magnitude and had a greater intrusive component. However, another consideration is that the periodontal defect present at the time the trauma was initiated was different in morphology. In the squirrel monkey, suprabony pockets were present, whereas in the beagle dog, infrabony pockets were present. In order to ascertain if it was the initial defect morphology which was the determining factor for the different results, an investigation was undertaken in squirrel monkeys to evaluate the effect of trauma upon existing infrabony pockets.

It had been known that, in the squirrel monkey model, if periodontitis was induced around an isolated tooth, periodontal destruction would occur localized only to the ligatured surface and an infrabony defect would result (Polson et al. 1974). In the study investigating the effect of trauma upon infrabony defects (Polson & Zander 1979b), localized defects were produced around the mandibular third bicuspid on one side of the jaw (control), while corresponding defects were induced around the corresponding tooth on the contralateral side followed by jiggling trauma (experimental). When corresponding histometric dimensions from experimental and control surfaces were compared statistically, there were no differences in loss of connective tissue attachment, but a greater loss of bone had occurred in specimens with the combination of periodontitis and trauma. In addition, there was a marked difference in osseous morphology between the groups. Infrabony pockets with angular bone loss were characteristic of the control specimens, however, the osseous changes which had occurred in the experimental specimens had eliminated the crestal angular bone loss morphology of the periodontal defects.

The findings from the latter investigation, when taken in combination with the findings from the previous investigations done in the different animal models (squirrel monkey and beagle dog), emphasize that trauma subjacent to periodontitis increases the amount of alveolar bone loss compared with periodontitis alone. However, the results from the two model systems differ with respect to effect on loss of connective tissue attachment, and further studies are needed in order to define the situations in which trauma may, or may not, act as a co-destructive factor in the presence of an ongoing, destructive periodontitis.

While the possibility of a co-destructive factor effect between periodontal trauma and marginal periodontitis is important from the perspective of pathogenesis of periodontal disease, knowledge of any interaction is even more important from a therapeutic standpoint. In advanced cases of periodontitis, the clinician is faced with the combined factors of marginal inflammation and tooth hypermobility. What is the relative importance of inflammation and trauma in the management of these situations? A search of the literature revealed a lack of scientific evidence to indicate the type or sequence of therapy. Consequently, it was decided to produce, in squirrel monkeys, an interproximal periodontium simultaneously reduced by the combined factors of inflammation (ligature-induced marginal periodontitis) and jiggling trauma, and then evaluate the periodontal response after selectively removing either traumatic, inflammatory, or both factors. In the following review of the studies investigating potential therapeutic responses after selectively removing inflammatory or traumatic factors,
whenever the term "reduced" periodontium is used, it refers to the periodontium reduced by the combined factors of experimental marginal periodontitis plus jiggling trauma.

In the first investigation, a reduced periodontium was produced and then only the jiggling trauma was discontinued (Polson et al. 1976b). On the clinical level, there was no decrease in tooth hypermobility even though active jiggling forces had been discontinued. Histological examination of the interproximal area 10 weeks after discontinuing trauma showed an identical appearance to the interproximal area of control specimens with respect to loss of connective tissue attachment, infiltrated supracrestal connective tissue, and alveolar bone morphology. No bone regeneration was apparent, and this observation was verified by the histometric analysis which showed no difference in the percentage of coronal alveolar bone between experimental and control groups. These findings indicated that either the increased bone loss due to trauma plus periodontitis was irreversible, or that inflammation in the supracrestal connective tissue might have been exerting an inhibitory influence upon a potential for bone regeneration following removal of the traumatic influence.

The latter possibility was evaluated by producing a reduced periodontium and then removing not only the traumatic factor but also resolving inflammation by removing the plaque-retaining ligature and maintaining an oral hygiene regime (Kantor et al. 1976). The regime consisted of mechanical toothbrushing three times a week. Two weeks after stopping jiggling and resolving inflammation, the most apical cells of the junctional epithelium were still located on the cementum, but the supracrestal tissue was less densely infiltrated with inflammatory cells. There was a marked difference in the appearance of the alveolar bone compared with control specimens (reduced interproximal periodontia). New bone formation was apparent, and this could be delineated from old bone by the presence of black "lead" lines. Lead acetate had been injected as a bone marker at the time the trauma was stopped and the oral hygiene begun. Although there was little new bone formation in a crestal location, much new bone was apparent in narrow spaces, and areas formerly occupied by widened periodontal ligaments. This bone appeared structurally different from the old bone, and had the characteristics of woven bone. Ten weeks after the combined factors were removed, long junctional epithelia were present on the root surfaces and the histometric analysis showed that there had been no coronal gain of lost connective tissue attachment. Inflammatory cells were sparse in the supracrestal area, and the latter connective tissue was well organized. The coronal alveolar bone had a heterogeneous appearance, with the old bone being lighter stained and lamellated compared with the basophilic appearance of the new bone. The histometric analysis showed that a significant amount of bone regeneration had occurred after resolving the inflammatory and traumatic components. However, although the amount of bone regeneration was significant, it had not increased the height of the alveolar bone.

If the increased bone loss due to trauma superimposed upon periodontitis were completely reversible, bone regeneration should occur to restore volume and height to a level comparable with periodontitis alone. Since this did not occur, it indicated that there may be some irreversible crestal alveolar bone loss due to the interaction between trauma and periodontitis or, alternatively, that the time period allowed in the experimental design was not sufficient to permit this crestal regeneration to occur.

The findings from the previous two in-
vestigations on the ability of a periodontium which has been reduced by co-destructive factors to repair, indicated that there is a potential for bone regeneration. Although it was necessary to remove both inflammatory and traumatic factors in order to realize this potential, it appeared that it was of prime importance to resolve the marginal inflammatory component, since it was the presence or absence of inflammation which dictated whether or not bone repair would occur after removing the traumatic influence.

As a result of the dominating influence which inflammation appeared to be having upon periodontal healing potential, an investigation was designed to evaluate the effect of resolution of inflammation in the presence of continued, active hypermobility (Polson et al. 1979). In experimental specimens, a reduced periodontium was produced whereupon the plaque retaining ligatures were removed and an oral hygiene regime begun. However, the active jiggling forces were continued. Clinically, although jiggling forces continued, a decrease in the degree of tooth hypermobility occurred during the remainder of the experimental period. The interproximal periodontium was examined 10 weeks after removing ligatures and initiating oral hygiene, and compared with a reduced periodontium in control specimens. There were no differences in the level of connective tissue attachment, or the location of the crestal alveolar bone between experimental and control specimens. However, there was a significantly greater percentage of bone in the coronal area of specimens in which the inflammation had been resolved but the active jiggling continued.

The findings from the latter series of investigations clarify the relative importance of inflammation and trauma in the management of advanced periodontal disease. The management of advanced disease with tooth hypermobility must be based upon resolution of marginal inflammation. Subsequent to the resolution of inflammation, continued tooth hypermobility does not result in further loss of connective tissue attachment or crestal alveolar bone. Furthermore, bone regeneration may occur in the presence of active, continued hypermobility after resolution of inflammation.

Zusammenfassung

Die Interaktionen zwischen der Entzündung und der Zahnbeweglichkeit (Trauma) bei der Pathogenese der Parodontalkrankheit

Résumé

Liaison entre l'inflammation et la mobilité dentaire (traumatisme) dans la pathogénie des parodontopathies

Une série de travaux ont étudié les interactions prenant place entre les traumatismes parodontaux et les parodontites marginales, par rapport au déclenchement des parodontopathies, à leur progression et à leur traitement. Les lésions traumatiques au niveau du desmodonte, à la suite de l'application de forces tendant à donner un déplacement unique ou un déplacement de va-et-vient, ont pour résultat des altérations morphologiques dans le desmodonte et dans l'os alvéolaire. Ces altérations ne déclenchent pas la perte de l'attachement de tissu conjonctif, caractéristique de la parodontite marginale. Des études effectuées sur l'écureuil, sur le singe et sur le chien briquet, avec production de forces de va-et-vient dans des cas où il existait une parodontite marginale, ont rendu compte d'une augmentation de la perte de l'os alvéolaire, mais la perte de l'attachement accélérée se produisant lorsqu'on utilise le chien ne s'est pas produite lorsqu'on utilisait le singe. Pour mettre en lumière l'importance relative de l'inflammation et de la mobilité dentaire dans le traitement de parodontopathies ayant atteint un stade avancé, la réaction du parodonte a été évaluée après élimination des facteurs traumatiques et/ou inflammatoires. L'élimination de l'action traumatisante lorsqu'il existait une inflammation marginale n'a pas diminué la mobilité dentaire, et n'a pas augmenté le volume osseux. Une régénération osseuse et une diminution de la mobilité dentaire se sont produites après l'élimination des deux facteurs à la fois; cependant, des résultats analogues ont été obtenus en éliminant l'inflammation, alors que la mobilité dentaire continuait à exister. L'élimination de l'inflammation marginale est d'importance capitale dans le traitement des cas de parodontopathies. Après l'élimination de l'inflammation, une régénération osseuse peut se produire autour de dents mobiles, et, de plus, la persistance d'une mobilité dentaire résiduelle ne provoque pas l'augmentation de la perte de l'attachement de tissu conjonctif.

Références


Address:

Alan M. Polson  
Department of Periodontology  
Eastman Dental Center  
625 Elmwood Avenue  
Rochester, New York 14620  
USA
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