Orthodontic Root Resorption Studied by Electron Microscopy

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INTRODUCTION

Root resorption and root shortening may be undesired consequences of orthodontic treatment. DeShields[1] found apical root resorptions large enough to be registered in 51 of 52 treated Class II, Div. 1 cases, and Massler and Malone[2] reported a considerable increase of root resorption in the apical area after orthodontic treatment (from 0.4% to 14.2%). In 1972 Kvam[3], employing scanning electron microscopy, found small marginal root resorptions on the pressure side in all 40 premolars after 5 days of experimental force. After 25 days resorption, lacunae with involvement of dentin were seen in all experimental teeth.

Hyalinization of the periodontal membrane has been reported by several researchers[3,16] in connection with orthodontically induced resorptions. Recently, Reitan[4] in an extensive study found that apical root resorption tends to start adjacent to a hyalinized zone and is more likely to occur in cases where the compression is strong and of some duration.

Some authors have, however, expressed doubt with regard to the combination of hyalinization and root resorption.[5] If the periodontal membrane is acellular, where are the cells participating in the resorption processes? Recently, the nature and extent of the fine structural alterations of the vascular system, the connective tissue cells, and the fibers during hyalinization of the periodontal ligament have been studied by the author.[6-9] In these experimental studies root resorption was frequently observed adjacent to persisting hyalinized zones and in areas where the hyalinized tissues had been eliminated and the periodontal ligament re-established.

Some of the findings related to the degree of breakdown of the fibrous structures[9] and to the process of removal of hyalinized tissue[10] seem to yield new insight into the relationship between degeneration and repair of connective tissue and root resorption. These findings were based on experimental tooth movement in rats.

The purpose of the present investigation was, by including human material where premolars had been moved by forces of a magnitude used in clinical practice, 1) to further evaluate certain aspects of the mechanisms of elimination and repair following elimination of hyalinized tissue and 2) to study at the ultrastructural level the processes of orthodontically induced cementum and dentin resorptions. Transmission electron microscopy does not appear to have been used before in such studies.

REVIEW OF LITERATURE

Nonorthodontic root resorption may be seen in many circumstances, physiological as well as pathological. A brief survey of some of the conditions necessary for the breakdown of cementum and dentin may yield some clues for a better understanding of the mechanisms related to these processes.

Although tooth structures generally show considerable resistance against resorption, it is more a question of degree rather than immunity. Whereas the roots of the permanent teeth are very resistant, the roots of the deciduous teeth are readily resorbed as part of the physiological process of tooth loss.

Resorption of all hard tissues is seen

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in the path of eruption of the permanent teeth provided a layer of connective tissue is interposed. If the connective tissue layer is lacking, the direct pressure between the hard structures will only cause mechanical displacement without any resorption taking place. Impacted teeth or permanent teeth with abnormal paths of eruption may cause pathological resorptions of other permanent teeth. Frequently, the maxillary laterals may be rapidly damaged by root resorptions caused by deviation of the erupting canines. Local pathological processes involving the jawbone may cause root resorptions. Thus, in deciduous teeth, the process seems to be accelerated by pulpitis while reduced or prevented by necrosis and periapical inflammation. According to Bouyssou, Lepp and Zerosi[11] acute apical inflammation will not produce root resorptions. On the rare occasions when resorption does occur, it usually takes place some distance from the focus of inflammation. In chronic apical inflammatory processes, however, resorptions develop fairly frequently.

When the apex is separated from the granulation tissue by the formation of an abscess, no resorption takes place, but a passive chemical corrosion in the purulent exudate produces a root resorption.

There is no general agreement about cementum resorptions in the marginal areas in connection with periodontal inflammation and pocket formation. Harvey and Zander[12] found a higher frequency of cementum resorptions (10%) in cases with periodontitis and ascribed this to changes in the cementum. Henry and Weinmann,[13] however, found little or no resorption caused by marginal periodontitis. Chronic pulp infections may cause internal resorption, and open, chronic pulpitis with the development of a pulp polyp may often result in considerable and rapid resorption of enamel and dentin. It has been assumed that the development of odontoclasts is caused by the same stimuli that cause circulatory disturbances. Tooth resorption in the gingival margin area may also be caused by changes in the gingiva, frequently in connection with posttraumatic hyperplastic gingivitis or in cases of epulis tumors.[14] Histologically these resorptions appear to be caused by proliferating inflamed gingival connective tissue.

Impacted teeth may undergo peripheral resorptions by the proliferation of a cystic membrane or the pericoronal connective tissue follicle. It is worth noting that in such cases the dentin and enamel are resorbed, but the pulp tissue surrounded by a zone corresponding with the predentin is spared or left intact for a considerable period of time. The susceptibility to root resorptions in connection with the development of various diseases of bone and tumors of the jaws is very variable. According to Bouyssou et al.,[11] benign mesenchymal tumors may cause root resorption, but the highest frequency is found in connection with giant-cell tumors. Malignant tumors of the epithelial type tend to displace teeth rather than resorb. Malignant tumors of mesenchymal origin may, however, cause considerable tooth resorptions. These are the most vascularized, as well as the most destructive, with the greatest tendency for the formation of metastases. The presence of tumor cells of a giant-cell type similar to osteoclasts may explain this particular tumor characteristic.

Trauma of the periodontal ligament of mechanical, chemical or thermal origin may result in resorption of tooth substance and, in many cases, with subsequent ankylosis.[15] External root resorptions take place in connection with various experimental traumas and overloading of teeth[16,17] and in cases of traumatic occlusion.[18] External resorp-

tions may also be the almost inevitable sequela after replants and transplants of teeth both in man and animals.[19] It does, however, appear normal that all teeth show some degree of root resorption throughout life. Massler and Malone[2] found apical root resorption in one or more teeth in all of the 708 patients in a group survey. These resorptions were described as idiopathic, because no detectable roentgenologic changes could explain their presence. A similar investigation based on histologic observations by Henry and Weinmann[13] does give some clues. In a postmortem study of permanent teeth in 15 dentitions, 90% showed areas of resorption indicated by resorption lines and reversal lines, and in 10% of these active resorption was taking place. Local traumatic changes such as compression, crushing, and necrosis of the periodontal ligament occurred invariably where active resorption of the alveolar bone and cementum was taking place. These authors concluded that in normal patients, generally, the most important cause of cementum resorption is the minor local traumas. Thus every individual has a certain resorption potential, but a high degree of root resorption can be expected in approximately 10% of the population. This individual tendency to marked root resorption was reported in 1939 by Becks in connection with hormonal disturbances and systemic diseases.[20]

To summarize, some particular features appear in common for various situations leading to root resorption: 1) increase of pressure (tooth eruption, tumors), 2) tissue damage in the periodontal membrane (mechanical, chemical, thermal), 3) increased blood supply (hyperemia connected with certain types of inflammation, hypertrophy, epulis), 4) infection and 5) individual predisposition (systemic diseases, endocrine disturbances).

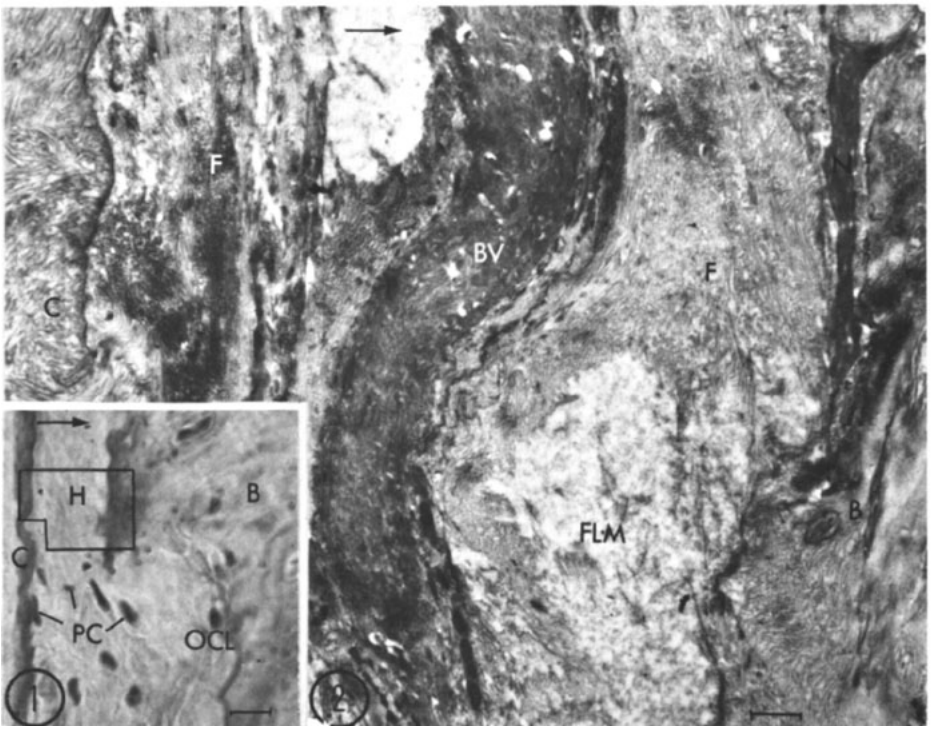
Material and Methods

Experimental tooth movement was carried out in 67 Wistar rats of both sexes. In the animals one maxillary first molar was moved buccally with a fixed appliance, as previously reported.[6] The appliance was activated to provide a constant force of 5, 10 or 25 grams. The experimental periods were 30 min., 2, 6, 12, 24, 48, and 60 hours and 5, 14 and 28 days. The removal of hyalinized tissues was studied in the specimens where force had been applied for 48 hours to 14 days. The human material consisted of 11 premolar teeth that were moved buccally by means of fixed appliances for periods between 2 and 50 days. The experimental forces were 70, 100, 120 and 240 grams. At the end of the experimental periods specimens containing the experimental tooth, adjacent periodontal ligament, and alveolar bone were processed for light and electron microscopy.[21]

Results

Light microscopy

The light microscopic findings were in agreement with previous reports of orthodontic pressure zones in rats by Kvam[22] and in human experimental material by Reitan.[23-26] The following observations are of interest in the present context. In the rat material, local areas appeared to be cell-free and had a homogeneous appearance after 6-12 hours, while extensive hyalinization was seen after 2-3 days (Figs. 1, 2). The width of the compressed periodontal ligament had been reduced to 15-40 μm and could readily be distinguished from the adjacent periodontal tissues which were rich in invading pioneer cells and blood vessels. This zone of reorganization was wider than the periodontal ligament in the control specimens, and osteoclasts were frequently observed adjacent to the alveolar bone (Fig. 1). In humans the width of the compressed



Figs. 1-2 Pressure zone in the periodontal ligament of the rat. Force 10 gr. Duration 60 hours. B, alveolar bone. C, cementum. Arrow, direction of force. Fig. 1 Pioneer cells invading hyalinized tissue (H). OCL, osteoclast. × 500, bar = 10 μm. Fig. 2 Electron micrograph of boxed area of Fig. 1. Compressed fibrils (F), blood vessel (BV), flocculent material (FLM) and nuclear remnants (N). × 6000. Bar = 1 μm.

hyalinized periodontal ligament after 21 days was 70 μm and the adjacent reparative zone varied between 250 and 350 μm.

In some specimens, both in the rat and the human material, multinuclear large cells were observed near the cementum surface at some distance from the hyalinized tissues, particularly in the specimens with longer experimental periods (Fig. 3). It may be of interest that some of these cells were not situated in contact with, but at some distance from, the cementum surface. When serially sectioned, it appeared that the multinuclear large cells belonged to a group of cells that were actually resorbing tooth substance, both cementum and dentin (Figs. 4, 5). These large cells were generally not the first pioneer cells to invade the hyalinized tissue.

Resorption lacunae were frequently bordered by active resorbing osteoclasts. The resorption of the cementum appeared as a resorption from the rear. The cementum, which on the periodontal side appeared to be functioning with a normal, or possibly reconstructed periodontal ligament, was resorbed by cells working from behind and from the periphery. The periodontal ligament, adjacent to the cementum being resorbed from the rear, was rich in blood vessels and cells and revealed bundles of fibrils inserting almost perpendicularly into the root surface (Figs. 4, 5). The blood vessels in the resorption lacunae seemed to be in close contact with the adjacent odontoclasts.

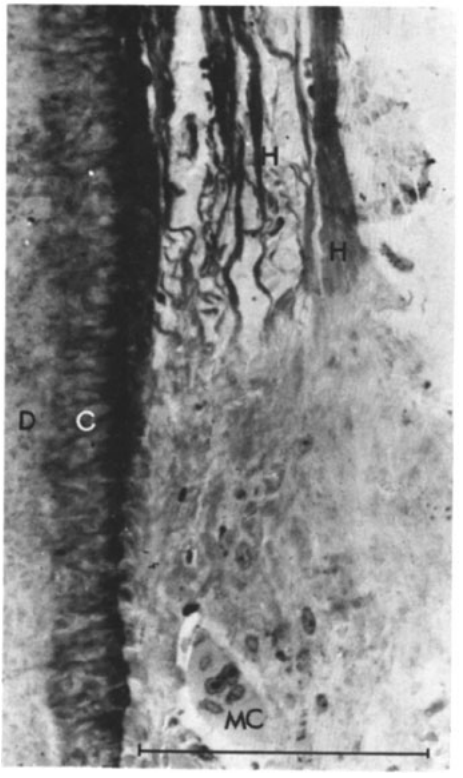


Fig. 3 Pressure zone in the marginal area of first premolar. Force 100 gr. Duration 50 days. C, cementum. D, dentin. Multinuclear cell (MC) near the cementum surface at some distance from hyalinized fibrils (H). × 400, bar = 100 μm.

Electron microscopy

The degenerative processes of vascular, cellular, and fibrous structures during hyalinization have been described.[6-9] Nuclear remnants occluded blood vessels with contents and compressed fibrils remained together with more amorphous filamentous material after compression for 3-4 days in rats and 21 days in humans. At low magnification the blood vessels appeared as dark strands (Fig. 2).

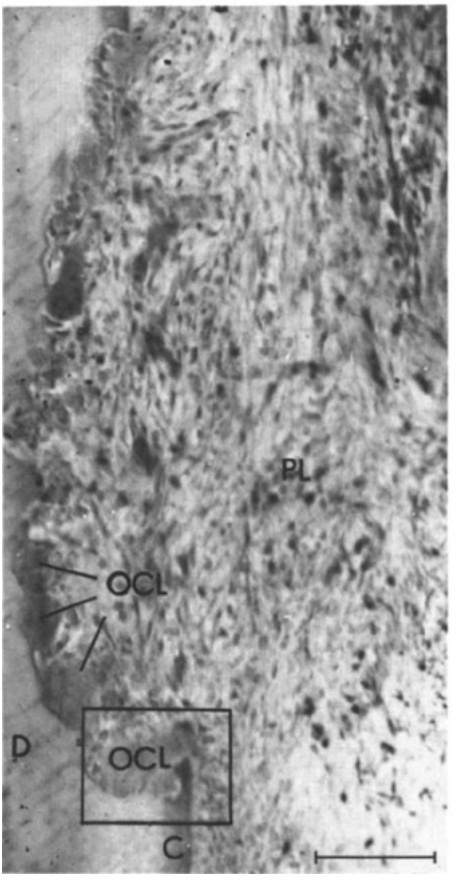


Fig. 4 Deeper section of same specimen as Fig. 3 revealing resorption lacuna bordered by odontoclasts (OCL). × 140. Bar = 100 μm.

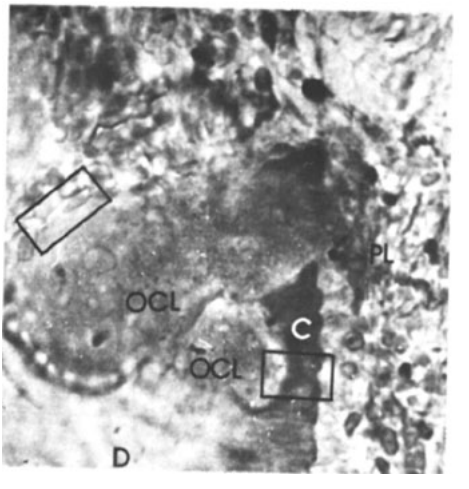


Fig. 5 Higher magnification of boxed area in Fig. 4. Odontoclasts (OCL) resorbing dentin (D) and cementum (C) from the rear. PL, periodontal ligament. × 700.

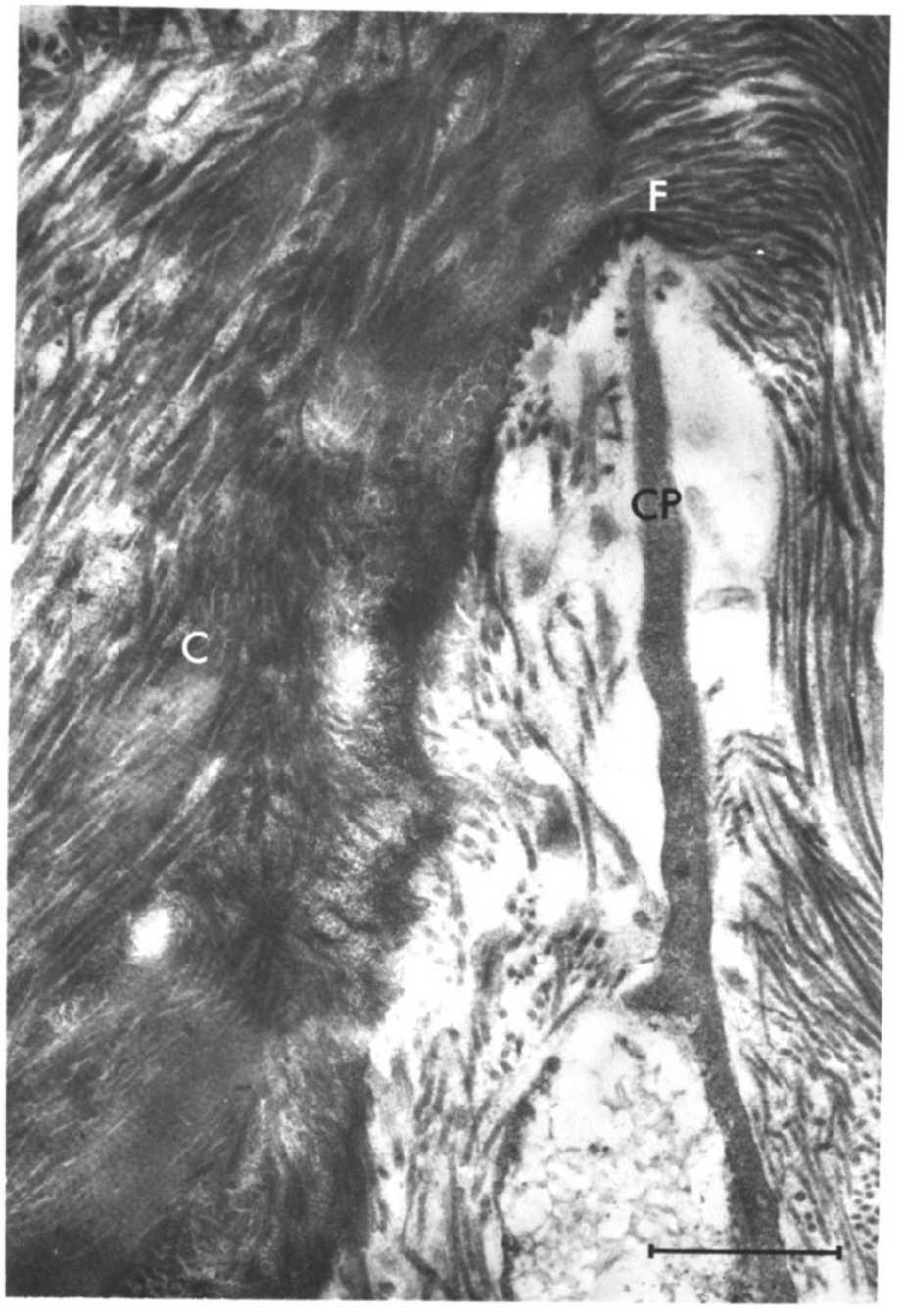


Fig. 6 Electron micrograph from pressure zone of human periodontal ligament. Force 10 gr. Duration 21 days. Projecting cell process (CP) of pioneer cell ascending along the cementum surface (C) into hyalinized area seems to cut off hyalinized fibrils (F). × 25,000. Bar = 1 μm.

Elimination of hyalinized tissue

Previous studies in rats indicated that the hyalinized structures disappeared concomitantly with an invasion of cells and blood vessels from the neighboring periodontal ligament and that the removal of collagen, cell remnants, and degraded vascular elements were mediated by various forms of cellular activity.[10] In the human material breakdown of the collagen fibrils in front of and around the processes of the invading cells was observed. As a rule, the cell processes were surrounded by a light zone containing collagen fibrils and a flocculent material. On the cementum side the projecting cytoplasmic process of the first invading cell seemed to cut off the hyalinized fibrils which appeared to be in a process of degeneration, leaving a naked tooth surface (Fig. 6).

In the rat material only spots of fibrous material were discerned in scattered areas both on the cementum and on the alveolar bone side in specimens where the invading capillaries occupied the entire space between the tooth and the alveolar bone (Fig. 7).

Root resorption

The ultrastructural study of the resorption process was concentrated on the relationship between 1) odontoclasts and adjacent blood vessels, 2) odontoclasts and cementum, and 3) cementum under rear resorption and inserting fibrils on the periodontal tooth surface (Fig. 5).

The odontoclasts revealed an intimate relationship with the capillaries on the nonresorbing surface. The impression was gained that only a thin membrane separated the lumen of the blood vessels and the cell cytoplasm. From the membrane, thin processes projected into the vessel lumen (Fig. 8). This surface of the odontoclast was very different from the side where the resorption took place (Fig. 9).

The study of a cementum projection being resorbed from the rear (Fig. 5) elucidates some of the finer details of

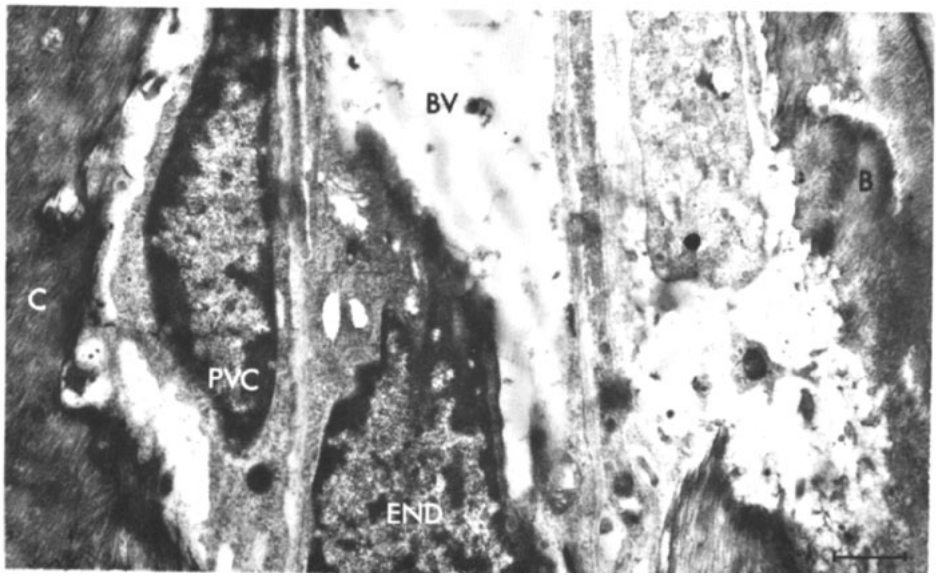


Fig. 7 Blood vessel (BV) invading a hyalinized zone of rat periodontal ligament occupying the entire space between alveolar bone (B) and cementum (C). PVC, perivascular cell, END, endothelial cell. Force 10 gr. Duration 5 days. × 9000. Bar = 1 μm.

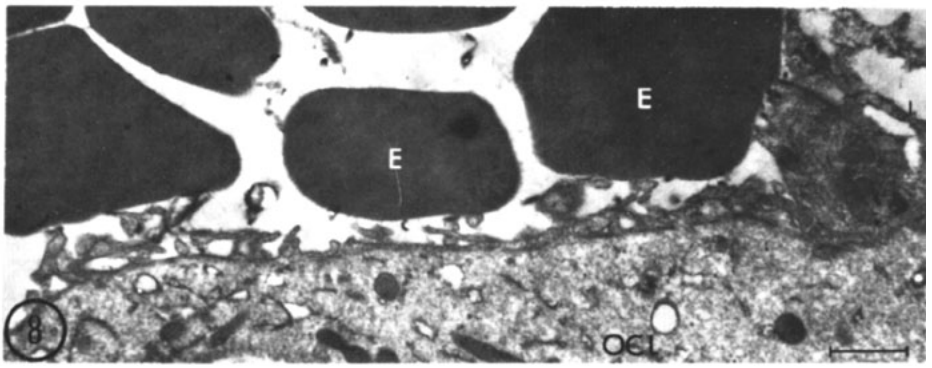


Fig. 8 Electron micrograph of boxed area (left) of Fig. 5 showing intimate relationship between odontoclast (OCL) and capillary with erythrocyte (E). × 9600. Bar = 1 μm.

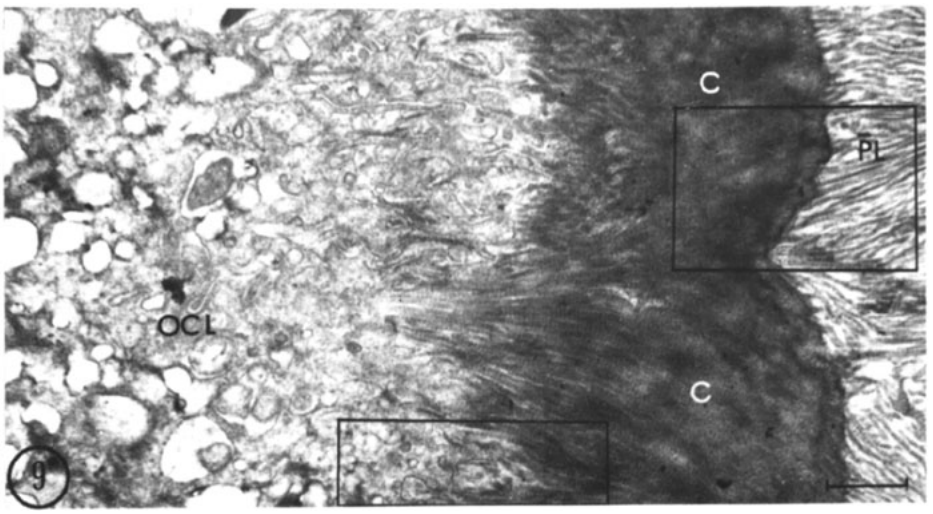


Fig. 9 Electron micrograph of boxed area (right) of Fig. 5 showing odontoclast (OCL) resorbing cementum (C) from the rear, while fibrils of the periodontal ligament (PL) insert on the root surface. × 10,000. Bar = 1 μm.

the relationship between odontoclasts and cementum during the breakdown (Figs. 9, 10).

The odontoclasts showed numerous mitochondria and vacuoles. The part of the cells adjacent to cementum revealed a complicated system of folds and deep penetrating clefts corresponding to the ruffled border of osteoclasts (Fig. 9). The cross-striations of the cementum fibrils were clearly discernible. At higher magnification (Fig. 11) it appeared that the striation pattern of collagen fibrils of the cementum was less clearly seen in the deep clefts between the cytoplasmic projections than in the cementum more distant from the resorbing cell.

The periodontal surface of the undermined cementum revealed incremental lines and intact fibrils inserting into the cementum from the ligament (Fig. 9). At higher magnification the cross-striation of the fibrils inside the cementum and in the periodontal ligament can be clearly seen (Fig. 10).

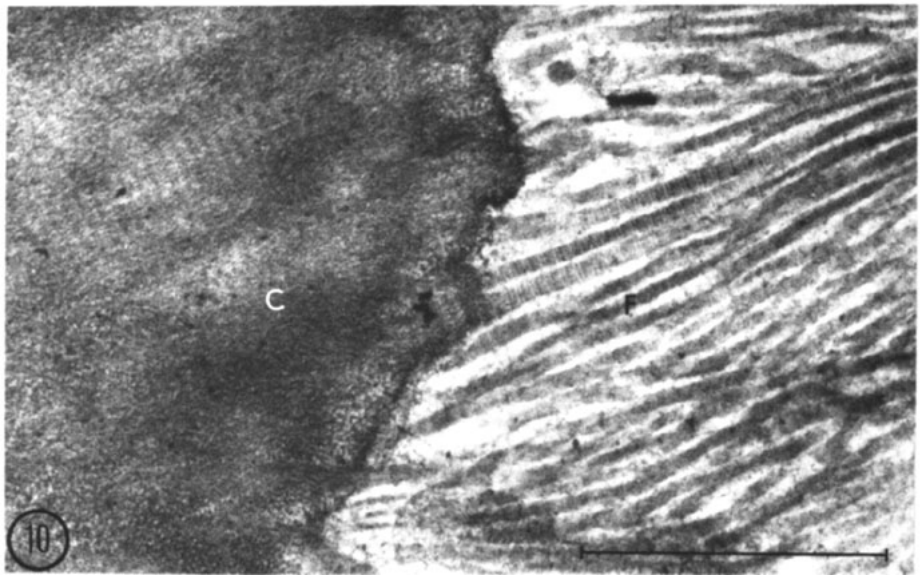


Fig. 10 Higher magnification of boxed area (right) of Fig. 9 showing fibrils (F) with cross striation pattern inserting into cementum (C) × 40,000. Bar = 1 μm.

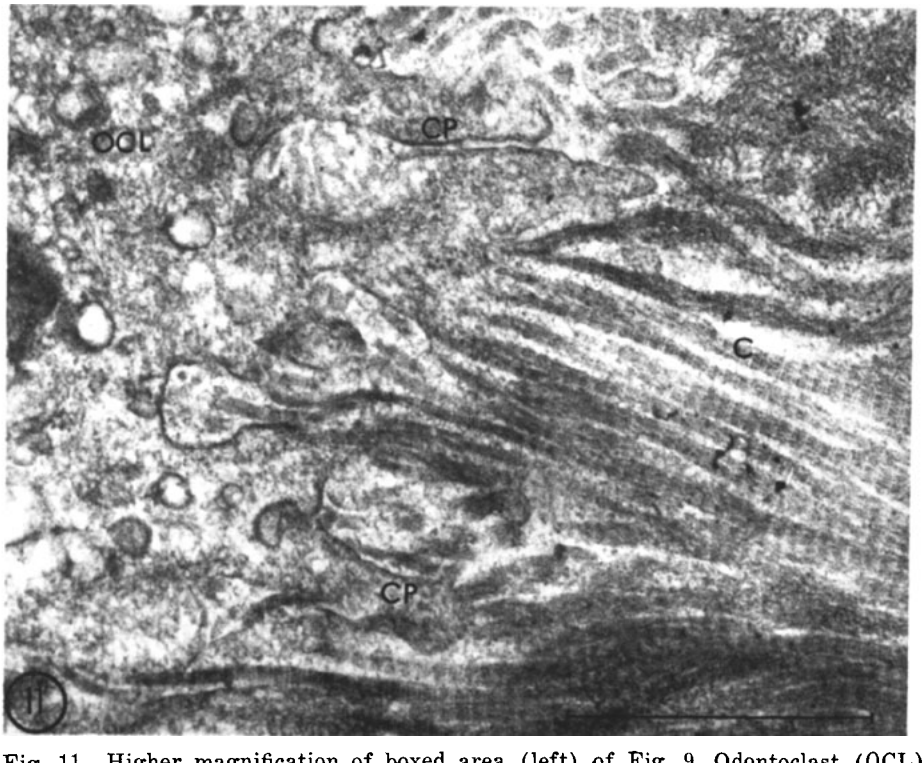


Fig. 11 Higher magnification of boxed area (left) of Fig. 9. Odontoclast (OCL) resorbing cementum (C). Note that cross striation of the cementum fibrils is less clearly discerned in clefts between odontoclasts' cellular projections (CP). × 40,000. Bar = 1 μm.

Discussion

The present light microscopic findings support previous observations by Stuteville,[27] Gottlieb,[28], Kvam[3], and Reitan[4,17,23,26] that, simultaneously with the removal of hyalinized tissue, resorption of the cementum may take place. The observation that root resorption occurs around the hyalinized tissue and is mediated by cells from the adjacent healthy periodontal ligament seems to explain why resorption seemingly occurs behind compressed, acellular parts of the ligament.

The study corroborates previous findings by Reitan that root resorption occurs where the compression is strong and of some duration. There is a clear relationship with regard to time when root resorption ensues following the elimination of hyalinized tissue. The elimination of hyalinized tissue in the human periodontal ligament is usually complete after 20-25 days, but the degree of root resorption was increased after this time. This study clearly demonstrated that the resorptive processes continue after the hyalinized tissue has been eliminated. It was also clearly shown that the resorption of cementum occurs as an undermining process whereby odontoclasts attack the cementum from behind in the resorption lacunae in the dentin. This indicates that the surface of the cementum is more resistent to resorption than dentin and that deeper parts of root cementum are more readily attacked than the periodontal surface. The initial resorption must, however, be a break through the outer cementum layer. This poses an important question. What causes the initial cementum resorption? Before this can be answered, it is also necessary to evaluate another problem: Why is it primarily bone, and not cementum, that is resorbed when a tooth is moved in the jaw or in marginal periodontitis?

Barriers against root resorption

The greater resistance of cementum has been attributed to several factors. In many ways cementum is similar to bony tissue, the cellular cementum to a greater degree than the acellular. There are differences, however. The chemical composition of cementum is similar to that of bony tissue, apart from the fluorine content which is considerably higher than in bone, particularly in the outer layers of the cementum. Also anatomical differences exist. The bony tissue has an ample blood supply whereas the cementum is completely void of vascular tissue. The anastomoses and the canaliculi within the cementum and between cementocytes and the periodontal ligament are inadequate to ensure the vitality of the cementocytes in the deeper layers of cementum. The hard tissues of the teeth are permanent depositories of mineral salts, as contrasted to the role of the bony system as a mineral reservoir for the whole organism. This circumstance is of great importance for the resorption mechanisms in bone and cementum.

Two breakdown mechanisms are active in bone: 1) osteolysis and 2) osteoclastic resorption. With regard to the cementum Furseth's[29] findings indicate that odontoclastic resorption is the active mechanism in human teeth, and that this mechanism is triggered by local stimuli.

The activity in the periodontal ligament is greater close to the alveolar bone than close to the cementum.[30] When teeth are exposed to pressure or tension, the greatest response is seen in the part of the periodontal ligament closest to the bony tissue. Thus, a particular feature is the continuous deposition of cementum throughout life, while the alveolar bone is constantly being remodelled. On the alveolar bone surface, as on all bone surfaces, the osteo-

clasts are part of the normal cell environment, but this is not the case on the cemental side. The high rate of turnover on the bone side results in the tissue being newer and more immature than adjacent to the cementum. In this context the collagen is of particular interest. Thus, the cementum is surrounded by older and more mature collagen which is more resistant to the actual chemical changes than the bone. Several researchers consider this to be the decisive factor in determining the greater resistance of cementum to resorption.[31,32] Correspondingly, in periodontal disease the periodontal fibers at some distance from the cementum surface are the first to be broken down.[32]

The unmineralized precementum layer, or cementoid, covering the cementum has also been attributed special importance as a resorption-resistant "coating." The precementum zone is 3-5 μm thick in acellular cementum, and somewhat thicker in cellular cementum. Since the precementum is continuously deposited, an uncalcified precementum layer will always be present on the root surface.

In an experimental study where human premolars were intruded for 4-35 days, Stenvik and Mjør[33] reported apical root resorptions in 60% of the teeth. It was clearly shown that the odontoclasts did not resorb unmineralized predentin. The precementum was, however, partly resorbed. In recent experiments on human premolars Reitan[4] demonstrated that the presence of a cementoid or predentin layer on the root delays the resorption process. The verified poorer ability of the odontoclasts to remove the unmineralized dentin matrix is interesting, because these findings seem analogous to previous observations indicating that the osteoclasts can only with great difficulty resorb unmineralized osteoid tissue.[34,35]

It has therefore been assumed that the initiation of resorption depends upon barrier alterations. Thus, osteoclasts always appear on a raw bone surface unprotected by a barrier.[36] The rapid appearance of osteoclasts on any raw bone surface indicates rapid induction by metabolic breakdown products.[36]

There are observations which give a measure of the resistance of osteoid in orthodontic tooth movement. In 1962 Reitan carried out experimental tooth movement in humans, first for 8 days in a buccal direction, and thereafter a reversed movement. This caused the formation of osteoid on the primary tension side. When the tension side became a pressure side, the osteoid showed a clear resistance to direct resorption, but only for a brief period of approximately three days. With the present knowledge of the resorption process the barrier concept is meaningful also with regard to tooth resorption.

There seem to be two barriers of particular interest in the subsequent discussion of the orthodontically induced root resorption: 1) the unmineralized cementoid, and 2) the more mature collagen surrounding the tooth root and not the alveolar bone.

Before discussing whether such barriers are altered during hyalinization, an evaluation is required of the degree to which the microenvironment in and around the hyalinized zone may promote resorption in general.

Microenvironment in the periodontal ligament during compression

The cell activity is directed and controlled by the area or microenvironment where the cell is living. The interplay between metabolic, circulatory, and mechanical factors influences the cell modulation and cell activity.

Metabolic signals generating changes in the relationship between osteoblast and osteoclast activity include the complicated interplay of hormones,

body type, and metabolic rate which may modify the individual cell metabolism and thereby the individual reaction pattern to disease, trauma, and aging.[40] It is reasonable to assume that disturbances or peculiarities in this interplay may explain the individual tendency to marked root resorption. Recent investigations strongly suggest a genetic component for shortened roots.[41] Local activation or differentiation of cells in alveolar bone or tooth cementum may, on the other hand, be caused by locally produced inductors in connection with circulatory conditions or changes in the forces affecting the teeth.

Circulatory changes influence cell metabolism to a large degree. In a localized area the bone resorption and bone formation are dependent on the degree and quality of the regional hyperemia.[37] In active hyperemia the blood flow is rapid, the lymphatic flow is reduced, and the loss of oxygen, proteins and vitamins is minimal. Active hyperemia with high oxygen pressure supports and induces osteoclastic activity, whereas passive, edematous hyperemia, yielding a high protein content in the tissue fluid, induces osteoblastic activity.

In vitro experiments have suggested that local resorption of alveolar bone requires one or more co-stimulating factors and increased oxygen pressure. Several local stimulating factors have been recognized, for example, vitamins A and D.[37] Dead bone, old bone, and raw surfaces of bone provoke osteoclastic resorption. It has been assumed that breakdown products from the bone matrix initiate osteoclastic activity.[36]

Mechanical factors have a strong impact on the bone structure. Thus, immobilization leads to a new balance between bone formation and bone destruction with the latter being the dominant factor.

Piezo-electricity is produced when crystalline materials are deformed by pressure on the crystals. The alveolar bone is thin, stands free, and may be bent during orthodontic tooth movement. Zengo, Pawluk and Bassett[38] found that the bone on the pressure side was deformed and took on a convex shape, while on the tension side the periodontal fibers bent the alveolar bone in such a way that it became concave. It was further discovered that the alveolar wall was deformed against the tooth with every systolic contraction and sprang back with every diastole. The hemodynamic forces presumably cause polarization, not only in the deformation of teeth and supporting structures, but also with the formation of so-called streaming potentials, electrical charges arising from the blood flow through the blood vessels.

An increased pressure and the abovementioned signals, positively affecting osteoclast development, are relevant in all orthodontic pressure zones with direct bone resorption. What then distinguishes the hyalinized zone and its surroundings from the compressed periodontal ligament where direct bone resorption occurs?

Microenvironment of the hyalinized zone and adjacent periodontal ligament

The present study showed that, due to intolerable pressure, the hyalinized zone or focus is characterized by cessation of circulation and degenerative changes of the periodontal structures, while the degree of damage to the adjacent cementum and alveolar bone is not readily evaluated. The adjacent undamaged periodontal ligament in the border areas is characterized by active hyperemia. The width of the ligament is considerably wider than in control specimens, both in rat and human material. Thus, it was clearly established that the *circulatory* conditions are well-

suited for the development of hard-tissue resorbing cells.

Zengo, Pawluk and Bassett have suggested that, when the circulation in the periodontal ligament is reduced or ceases on the pressure side, this leads to considerable changes in regional streaming potentials. These potentials would arise in addition to the regular piezo-electric potentials. It seems tempting to the author to relate these findings to the observations indicating that root resorptions are more frequent close to blood vessels, in canals in the root surface, and in the root pulp.

Norton hypothesized that, if excessive forces are applied, the deformation potential can exceed its limit and become zero. One could thereby pass the deflection tolerance of alveolar bone and enter the range where the tooth starts to deform.[39] This would establish the electrically positive environment which could initiate root resorption.

As far as is known, no attempts have been made to study the bioelectric potentials arising from hyalinization. There is no basis yet for claiming that special electric polarity in and around the hyaline zone should promote the susceptibility to resorption of cementum. The present study indicates that during hyalinization the tissue damage in the periodontal ligament alters the biochemical environment. It has been demonstrated that the process of hyalinization may be divided into three phases: degeneration, elimination, and re-establishment. There is reason to believe that the initial breakthrough of cementum is connected with the elimination of the hyalinized parts of the periodontal ligament.

In addition to the favorable environment for the development of the hard tissue resorbing cells found in all pressure zones in the periodontal ligament during orthodontic tooth movement, there are certain border areas of the hyalinized zones which are exposed to maximum, tolerable mechanical influence and a local breakdown of tissue with waste products. The conditions in the hyalinized area seem to a remarkable extent to correspond with the particular features stated for the other forms of root resorption, and coincide with the view that a local trauma in the periodontal ligament is the main cause also for the nonorthodontically induced root resorptions.

The mechanism of elimination of degraded tissue seems to be influenced by the composition and shape of the material to be removed. Foreign-body giant cell aggregates seemed to develop and mediate the removal of certain blood components in the rat material.

Irving and Handelman[42] claim that various forms of foreign-body giant cells are closely related with regard to enzymatic action. If certain metabolites or their ratios are deciding factors in the induction to varied cell modulation and activity, it seems feasible that biologically active substances are released by destruction of tissue within the hyalinized zone. These may affect the development of various cells around the hyalinized zone including giant cells with the ability to resorb cementum. The destruction of tissue in the hyalinized zone may possibly be influenced by the amount of pressure being exerted. It seems that a large initial force with the development of a fairly widespread hyalinized zone may cause greater amounts of blood components to be trapped in the central parts of the pressure zone. These matters need further study.

The degree of damage to the outer layers of cementum during orthodontic hyalinization has yet to be determined, but it has been shown that the continuous cementum deposition has ceased.[9]

Regarding the barriers of the root surface, this study on human material corroborated the author's previous observations in rats that the hyalinized tissue, including cementoid, collagen fibrils and osteoid, is removed before and during the re-establishment of the periodontal ligament.[10] This means that both the cementoid layer and the more mature collagen adjacent to the cementum disappear after hyalinization, at least in some areas. A barrier alteration has taken place. Certain limited areas on the root surface are then comparable to the previously mentioned raw bone surface.[36] There is reason to believe that these areas are less well-protected against attacks of resorption from giant cells which are easily formed in the border areas. It is also conceivable that raw tooth surfaces actively induce odontoclast formation.

The present observations seem to render a basis for the hypothesis that the development of local root resorption in connection with hyalinization occurs in two stages. (1) Because the barriers on the root surface are eliminated after hyalinization, an initial resorption of cementum may easily take place in the strongly resorption-promoting environment around a hyalinized zone. A small breakthrough may be adequate to start the process. (2) When a resorption lacuna has been formed, the ensuing events will depend upon whether or not application of force is continued. If it is, root resorption will continue. The resorption lacuna seems a favorable, protected environment for the resorbing cells during the application of a force of a magnitude used in clinical orthodontic treatment. The present study clearly shows that, once a resorption lacuna is established, the cementum on the edges of the lacuna is resorbed from the rear, even when the cementum has a normal periodontal surface.

If the application of force is absent or is below a certain level, repair will commence in the resorption lacunae with the deposition of cementum.

An hypothesis of this kind poses many questions. It does not verify whether the precementum or the mature collagen, or a combination, is the most important barrier. It is at the present time not possible to establish to what degree breakdown products from hyalinized tissue have any importance as a signal for the development of resorbing cells.

The importance of the bioelectric signals needs further clarification, because they may yield information regarding the importance of force application and contribute to a better understanding of the influence of circulation of fluids on the phenomenon of resorption.

The cause of the greater frequency of resorptions taking place in the apical area is also unknown. Is it due to the similarity between cellular cementum and bone, or is the blood supply particularly favorable?

This view of the resorption mechanism coincides well with the clinical observations that the root resorptions cease if the orthodontic treatment is interrupted or stopped. The repair taking place will cause new deposits of precementum on the root surface which may possibly act as a new barrier.

This supports Reitan's recommendation for a rest period if a strong tendency for root resorption seems present.[43] One might consider going further by including rest periods throughout the course of orthodontic treatment, particularly in those stages where one might expect resorption to take place.

Summary

The present study supports previous findings that root resorption takes

place simultaneously with and after the elimination of hyalinized tissue.

The cementoid layer and the more mature periodontal collagen fibers adjacent to cementum are possible barriers preventing root resorption. The microenvironment around hyalinized tissue is favorable for the induction of hard-tissue resorbing cells.

The findings show that the elimination of hyalinized tissue leads to the removal of the cementoid and the mature collagen thus leaving a raw cemental surface without a barrier. It is therefore hypothesized that such an area is readily attacked by odontoclasts. Once resorption lacunae are established, the cementum is resorbed from the rear as an undermining process. By continued orthodontic force application the resorption process will proceed even after all hyalinized tissue is eliminated. If the orthodontic force is discontinued or falls under a certain level, the resorption lacunae are repaired.

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