A HISTOLOGICAL EXAMINATION OF HUMAN TOOTH MOVEMENT PERIODONTAL FIBER SYSTEMS

Bruce D. Fiske, D.M.D.

This paper submitted in partial fulfillment of the requirements for a Certificate in Orthodontics, University of Oregon Dental School.

June 1974

ACKNOWLEDGMENTS

I would like to express my thanks to Dr. Douglas L. Buck for his guidance throughout the course of this project, to Dr. Ralph G. Merrill who performed the surgical procedures, to Dr. Robin W. Matthews and the anatomy tissue laboratory staff for histological preparations and advice, and to Ms. Roberta J. Beckman for typing this manuscript.

TABLE OF CONTENTS

INTRODUCTION

Orthodontic tooth movement has been studied extensively for many years, however, the exact biological mechanisms of tooth movement are still unclear. Nonetheless, it is a biologic fact that a force placed on a tooth does produce movement. In an attempt to explain this phenomenon, various theories of tooth movement have been proposed.[4,5,7,62] Formerly, major research emphasis has been on the reaction of bone to orthodontic forces. This emphasis has by-passed many questions concerning the reaction of the soft tissue of the periodontium to orthodontic forces, which remain a fruitful area for histologic research.

The purpose of this paper is to emphasize the relationships that exist between orthodontic tooth movement and noncalcified connective tissue of the periodontium, both during and after active tooth movement. Emphasis will be placed on particular fiber systems active in the periodontium, but especially on oxytalan fibers, their origin, formation, distribution and possible role in certain types of orthodontic relapse. A histological

examination of these fiber systems and their interrelationships in post 14 and 21 day human tooth movement material will be examined.

REVIEW OF THE LITERATURE

The importance of connective tissue fibers in maintaining tooth position is well described by G.V. Black, who said:

> The influence of the bone forming the alveolar process has been much overrated in its importance in maintaining the teeth in their positions. Hard and rigid as the bones of the skeletons seem in the dried state, bone is a very plastic tissue during life, and is subject to more or less constant resorptive and rebuilding processes. It will not stand against a connective tissue constancy of stress. The connective tissue group, other than active muscles, has a great function in directing the building of the body, holding organs in their places in health, and bringing them back to place when the correction of conditions will allow them freedom of action... There is no place in the human body where there are as fine examples of this play at control of form by the non-muscular connective tissue as in the gingivae, or so much harm from its influence when the conditions have given them a wrong direction.[8]

In orthodontics, fascination with treatments designed to increase arch dimensions to accommodate all teeth ebbed as tissue-induced relapse in these cases was recognized as a serious consequence. E.H. Angle's[1] non-extraction "bone growing" treatments were eventually replaced by the extraction philosophies of Case,[15] Tweed,[79] and others.[37,51] It was recognized that good occlusion, arch form, and muscle balance alone could not ensure a stable orthodontic result. These observations, as reported by Reidel,[56] gradually became precepts of retention: 1) teeth

moved orthodontically have a tendency to return to their former position, 2) arch form, especially in the mandible, cannot be permanently altered by treatment, 3) bone and adjacent tissues must be allowed time to reorganize around the newly positioned teeth, and 4) the farther teeth are moved, the less likelihood of relapse (presumably because tooth-soft tissue attachments have been severed by excessive stress).

These "laws" evolved from a recognition that the soft tissue influences of the lips, tongue, and more importantly, the periodontium had to be manipulated for a long-term stable result. In order for control of some of these detrimental influences, theories of histological processes occuring during tooth movement became well publicized. Based on an understanding of tooth movement histology, various retention philosophies and procedures to minimize relapse became known.

Although various theories of tooth movement have been proposed,[4,5,6] the oldest, and most generally accepted, is the pressure-tension theory. The evolution of this theory began in the late 1800's, but Sanstedt[66,67] was the first to publish information describing it as a histologic phenomenon. Utilizing jack-screws on a labial arch, he tipped maxillary

canines of a dog lingually. Histologically, he noticed bone resorption on the pressure side and deposition on the tension side. Of particular interest is his observation that heavy pressures caused compression of the "peridental" membrane and subsequent necrosis. Before tooth movement could recommence, this material had to be removed via resorption from alveolar marrow spaces. This process was described by him as "undermining resorption."

Oppenheim,[53] in 1911, reported that when deciduous teeth of baboons were moved, resorption occurred on the pressure side; and, on the tension side, bone was deposited along stretched tissue bundles in the direction of pull. He theorized that bone reacted to pressure by architectural transformation and that deposition preponderated over resorption. He also noted that heavy pressures caused vessel thrombosis and subsequent injury to the "periosteum," resulting in no new bone formation. Therefore, light forces were advocated.

In order to try to quantify forces that would not be injurious to the periodontium, Schwarz,[68] in a dog study, found that a force of

20-26 gm./cm^2 of root surface (equal to capillary blood pressure in the periodontal ligament) caused no injury to these tissues. Forces above this level, he postulated, would cause capillary compression and would subsequently "suffocate and strangulate" the periodontal ligament. Tissue necrosis would then result, causing bone resorption to cease, as resorbing cells were not active in this damaged tissue. Undermining resorption from marrow spaces was then necessary to remove this necrotic debris so that frontal bone resorption (and tooth movement) could begin again.

Human tooth movement investigation was first reported in this country by Herzberg in 1932,[44] who described bone formation in the periodontal tissue of an orthodontically-tipped maxillary bicuspid. His results were similar histologically to those of previous investigators who had experimented in animals. This led him to the conclusions that alveolar bone of humans reacted similarly to alveolar bone of animals.

Information from these and other early tooth movement studies became the basis of understanding the reaction of both hard and soft tissues to tooth movement. The best explanation of what happens to

these tissues occurring during pressure-tension tooth movement has been by Kaare Reitan,[60] a prolific worker in tooth movement histology. He reported that the process of tooth movement is related to a series of tissue factors, the most important of which are: 1) the formation of cell-free or hyalinized areas, and 2) reaction of fibrous tissue.[59]

The first tissue factor, the formation of cell-free areas, occurs when forces applied to a tooth produce a gradual compression of the periodontal ligament and, eventually, one or more areas of hyalinization. Reitan[62] ascribed the formation of this hyalinized area to both anatomical and mechanical factors. An example of an anatomical consideration is the form and outline of the bone surface. That is, if there are open clefts and spaces in the bone, hyalinization, if present at all, will be of shorter duration than when dense bone, with few marrow spaces, is encountered. The direction of tooth movement is the best example of a mechanical consideration. Tipping tooth movement usually causes a hyalinized area near the alveolar crest and, if the force is excessive, another hyalinized area may form apically.[62] In bodily movement, any

cell-free areas formed are usually located nearer the middle third of the root.[62] Rotation usually causes two areas of hyalinization, one buccal and one lingual, unless the root is perfectly round.[59,72]

The terms "hyalinization" and "cell-free," although usually used interchangeably in tooth movement literature, may be different histologic entities.

Hyalinization is a gross pathological term and refers to a tissue area which is clear and glass-like in appearance, thereby implying loss or destruction of all formed elements of the tissue.[11] Presently, there is still controversy whether hyalinized areas are totally cell-free areas of homogeneous amorphous material, or rather, still contain fiber bundles in regular arrangement. Differences in the appearance and interpretation of the contents of hyalinized areas may be partially due to research methodology.

Under the light microscope, the first changes in hyalinization occur in cell nuclei as some become pyknotic while others disappear altogether. Then cell outlines become indistinct, and the compressed

collagenous fibers gradually unite into a more or less cell-free mass. It is felt that the uniform hyaline appearance of the compressed zone is caused primarily by certain changes in the ground substance which tend to mask the collagen fibrils. Reitan[62] noted that the collagen fibers themselves appeared to be relatively unaltered.

Kvam, however, in a light microscope and autoradiographic study on rats[45] and, subsequently, in a scanning electron microscope study of orthodontically-positioned human premolar teeth,[46] found that hyalinized areas consisted of homogeneous and coalesced fiber bundles where no structural arrangement remained.

Rygh, in both animal tooth movement studies[64,65] and a human study[63] did not totally agree with Kvam's findings. Using a transmission electron microscope, he found that, although various stages of cellular necrosis had occurred, fibrils in typical hyalinized areas had retained their forms and cross banding. He suggested, as did Reitan, the glass-like appearance is related to compression of fibers and changes of the surrounding elements, but not loss of fibrillar structural arrangement.[64]

Regardless of the exact nature of the cellular contents as seen under the electron microscope, it is true that bone resorption cannot occur along the pressure side until the hyalinized or cell-free areas, and tissue remnants therein, have been eliminated by undermining resorption. The term "lag phase" is applied to this static tooth movement period during the process of undermining resorption. The purpose of light forces is to minimize the lag phase by keeping hyalinized areas small in size and few in number so that they may be readily eliminated by undermining resorption. Initial cell-free areas are eliminated as undermining resorption breaks through from alveolar marrow spaces, resulting in a cellularly repopulated widened periodontal ligament space. With the use of proper mechanics, further localized concentration of forces, which can cause hyalinization and concomitant lag phases, are thought to be avoided.

Hyalinization, therefore, can be reduced or even prevented by proper force magnitude and distribution over the root surface area after initial tooth movement. Tipping tooth movements, with force concentrations (and cell-free area formation) at the alveolar crest, are

avoided. Bodily tooth movement, by most effectively accomplishing good force distribution over the root surface, minimizes hyalinization, thereby increasing the efficiency of tooth movement.

The second tissue factor mentioned by Reitan is the influence of the stretched and displaced fibrous tissue on the tension side. Under proper tensile force of a continuous nature, these stretched fibers have been shown to induce bone spicule formation. Osteoid is formed quite rapidly on the tension side, especially if pressure side undermining resorption has been completed. Calcification of this osteoid helps to re-establish normal periodontal width. However, in all tooth movement, especially the tipping and rotation types, stretched periodontal fibers not only have been shown to induce osteoid and subsequent bone formation, but also, on completion of orthodontic movement, tissue-induced orthodontic relapse. It is the presence of this undesirable tissue factor, especially active as a tensile relapse mechanism, that has prompted much research to try to prevent its formation and control its effects. Many procedures, including prolonged post-treatment retention, treatment over-correction

and various surgical methods, have been advocated to eliminate this post-treatment influence.

Skogsburg,[73] in 1932, postulating that "tissue tensions" initiated the relapse of orthodontically positioned teeth, devised a "septotomy" procedure to reduce these relapse tendencies. He surgically divided the alveolar process into separate segments so that each part could heal independently of each other "to make a new coherent whole where the factors tending toward a relapse, even if not quite annihilated, would yet be very largely reduced." Skogsburg felt that bone still had "tensions" in it even after the resorption and deposition processes of tooth movement had taken place. His surgical procedure was a clinical success in preventing some forms of relapse, but at that time the reasons for his success were unclear.

The traumatic rotation of teeth was reported as a means of correcting malposed teeth by Hallett.[40] Using an extraction forcep, he forcibly rotated teeth, thereby severing the attached fibers of the periodontium. After correct positioning, the tooth was stabilized for

2 to 3 weeks. Although this clinical procedure was not without serious complications (i.e., ankylosis, root resorption and pulpal necrosis in 5 of 23 cases), most of the "treated" teeth did show remarkable resistance to rotational relapse. Although the reason for his success was not clear to Hallett, his observation that surgical disruption of connective tissue attachment markedly reduced rotational relapse was valid.

Thompson[76-78] attributed the stability of orthodontic treatment to three factors: 1) the process of bone remodelling--the reaction of the periodontal ligament to pressures and tensions, 2) proper muscle balance of the lips, tongue, and cheeks, and 3) the influence of connective tissues superior to alveolar bone, i.e., the transeptal and gingival fibers. His work was concerned with the influence of the supra-alveolar connective tissue fibers on orthodontic retention. In a study utilizing monkeys,[78] he completely removed all soft tissues around orthodontically rotated teeth. After a retention period of three weeks, the retention devices were removed. The amount of rotational

relapse of the experimental teeth was then compared with the control teeth similarly treated but not gingivectomized. His results indicated that the experimental teeth experienced about four times less relapse than did the controls, thereby confirming the importance of supra-crestal fibers as a factor in determining post-treatment stability.

Thompson, reflecting on Skogsburg's work, reasoned that the success of the septotomy procedure was not due to a relief of "bone tension," but rather due to an effective neutralization of supra-alveolar connective tissue fibers severed in an attempt to reach the deeper alveolar structures of the periodontium. In fact, Thompson felt that almost any form of relapse in an orthodontically treated case could be explained theoretically in terms of the response of connective tissue fibers.

In 1969, Brain[10] performed an experiment to determine the effect of gingival surgery on retention of rotated teeth. He found that the transection of the free gingival fibers almost eliminated relapse compared with the contra-lateral non-surgical control teeth (5 animals;

experimental results--4 animals 0^{o}, 1 animal 1^{o} rotational relapse; control results--5 to 34^{o} relapse). He indicated that free gingival fibers were responsible for rotational relapse. The cause of this relapse was that rotational tooth movement caused a displacement of gingiva, which in turn placed tension on gingival fibers. These stretched fibers, which are also attached to root cementum, contracted upon removal of the rotational force, thereby causing re-rotation.

The studies cited above indicated that various methods of severing connective tissue attachments to orthodontically positioned teeth were successful, to varying degrees, in preventing relapse. Common to all procedures was the severence of some or all of the fibers of the periodontium. Surgical procedures, therefore, appeared to have value in preventing relapse, certainly of the rotational type. More information was needed, however, to ensure that simpler, less traumatic methods might not be equally effective. Information was also necessary on exact tissue mechanisms involved in this type of relapse process. Were particular fiber groups of the periodontium responsible for relapse, or could it be

the result of the single or combined action of some of the formed fibrillar components in these tissues?

The fibers of the periodontium are composed of gingival fibers and fibers of the periodontal ligament proper. The gingival fibers are arranged in three groups: 1) the gingivodental, extending from the crest and outer surface of the marginal gingiva to the cementum, 2) the circular group, and 3) the transeptal group, horizontal bundles extending between the cementum of approximating teeth.

The most important element of the periodontal ligament are the collagenous principal fibers, which are arranged in bundles and follow a wavy course. Terminal portions of these fibers inserting into cementum and bone are called Sharpey's fibers. The principal fibers of the periodontal ligament include the alveolar crest fibers, which are fibers extending obliquely from cementum just beneath the epithelial attachment to the alveolar crest, and the horizontal, oblique and apical groups, which describe the direction or position that these fibers extend from root cementum to alveolar bone.[34]

The tissue reaction of these soft tissue fibers to orthodontic tooth movement and various surgical control procedures varies, dependent on the relationship of the fiber groups to bone. As will be seen, the classification terms "supra-alveolar" and "infra-alveolar" are more meaningful when discussing this reaction.

Reitan,[61] in a histological study of retention in dogs, found that some gingival fibers adjacent to rotated teeth remained stretched and displaced even after a retention period of 232 days. He attributed relapse of rotated teeth after retention to the contraction of these displaced gingival and supra-alveolar fibers. In marked contrast, however, the infra-alveolar fibers of the periodontal ligament were not seen to be displaced and stretched, but had become re-arranged quite rapidly, most within 28 days. The difference between the reaction of the two fiber groups was due primarily to the influence of bone. Initially, stretched infra-alveolar fibers induced bone spicule formation. As bone deposition took place, a gradual reduction of periodontal width, and consequently fiber stretch and displacement, also occurred. In fact, tissue

re-arrangement in these infra-alveolar areas was seen to be so complete that histologically, it appeared that no tooth movement had occurred at all. It appeared from this study that infrabony fibers of the periodontium were not the cause of long-term rotational orthodontic relapse, since customary retention procedures allowed enough time for adequate bone and tissue re-organization to recreate a "normal" periodontal ligament. Supra-alveolar fibers, on the other hand, with no bone remodeling available to effectively shorten stretched and displaced fibers, remained potential sources of relapse for long periods of time.[58]

Edwards[21] in a study of rotational relapse confirmed Reitan's findings by noting histologically that five months after orthodontic rotation, the tissue still appeared taut and oriented in the direction of rotation. The tissue had not become re-organized in accommodation to this new position.

In contrast to rotational tissue retention problems, the types of tissue-induced relapse tendencies associated with retention of extraction space closures were quite different in that tooth movement in this area placed compressive forces upon the gingival and transeptal soft

tissue. Erikson, et al.[22] observed histologically that transeptal connective tissue fibers were compressed and coiled in the area of extraction space closures. This was due, they indicated, to a slow metabolic re-organization rate of these fibers. According to their observation, they hypothesized that relapse of extraction space closures may be due to the rebound effect of the compressed, coiled, unresorbed transeptal connective tissue fibers.

Atherton and Kerr[2,3] described these effects of orthodontic tooth movement on the gingiva. They observed that a tooth does not move through tissues, as it does bone, but instead, causes a tissue pile-up on the pressure side. On the tension side, however, they described the presence of a triangular "red patch" of new tissue, formed as enamel epithelium became detached from the rapidly moving tooth. This study pointed out that, unlike bone which is capable of rapid re-orientation and re-organization, pressure causes gingival deformation and accummulation.

The findings of Edwards,[19] in a study designed to determine the cause of extraction space relapse, were in agreement with those of

Atherton and Kerr. He also noted that in extraction space closure, teeth seemed to push the gingival tissue ahead. These distorted, compressed and piled up tissues could then become potential sources of relapse. He found that surgical removal of the excess tissue in the extraction space prior to debanding, successfully eliminated or markedly reduced relapse of this type.

As indicated in the above studies, it appeared that the causative tissue factors in various types of orthodontic relapse were the presence of compressed, elongated, or in some way distorted supra-alveolar tissues. What became obvious was that to prevent this form of relapse, tissue of the periodontium had to become accommodated in some manner to the new position of the tooth. Reitan's[57,58] and Edward's[21] observations indicated that, although short periods of retention would allow infra-alveolar tissue-bone re-organization to take place, extremely long-term and impractical retention periods were indicated in the hopes that sufficient time would be available for the metabolic re-organization of the supra-alveolar tissues.

How long it took for this re-organization to occur had not been agreed upon, but basically depended on the formative and degenerative metabolic activity of collagen. Of the four fibrillar components of the tissues of the periodontium (collagen, reticular fibers, elastic fibers and oxytalan fibers), collagen is by far the major tissue component.

The importance of collagen is that it has the ability to resist a variety of internal and external challenges, i.e., mechanical stress and stain. The physical and chemical properties of the collagen molecule aids in understanding this reaction to stress. Collagen is a three-stranded molecule approximately cylindrical in shape with the three strands winding on each other with a 100A^{o} repeat of the order. It is about 3,000A^{o} long and 14A^{o} in diameter with a molecular weight of 300,000.[54] Individual collagen fibrils are composed of specific aggregates of collagen molecules with their axes overlapping regularly to produce the repeat order of about 700A^{o}, seen as regular banding under the electron microscope. The molecules themselves can form fibrils easily, but these are not stable until interchain covalent cross-linkage occurs which induces maturation

of the collagen.

By denaturing collagen it has been determined that the molecule is composed of three types of chains, alpha (α), beta (β), and gamma (γ). Young collagen protofibrils are formed by the joining of two kinds of alpha chains a_1 and a_2 by non-covalent bonds. With aging, and covalent bond formation, denaturing reveals the presence of dimers of alpha chains designated as beta chains and trimers of alpha chains called gamma chains. These dimers and trimers represent stronger electrochemical forces that require more forceful measures to break these bonds. Covalent inter- and intra-molecular cross-linkages, which produced these dimers, impart important properties to tissue, such as increased tensile strength and a decrease in extractable collagen in the denaturing process, thereby indicating an increase in stability.[81]

Both Piez[54] and Neuberger[52] report that mature collagen turnover rates are very low. Therefore, it would appear that prolonged post-treatment distortion of collagen rich tissues was due to a high percentage of the covalent-bonded, stable, and metabolically-inactive collagen. Theoretically,

rapid collagen turnover should occur in young individuals with immature (i.e., non-covalent bonded) collagen.

The influence of tooth movement on collagen formation has been studied in research animals by the administration of radioactive proline, an amino acid present in high concentrations in collagen. Autoradiographic analysis of sequentially sacrified animals has, by determining sites of proline uptake, given temporal evidence as to the amount and position of newly formed collagen.

Stallard[74] demonstrated this process in rats. He injected radioactive proline which later became apparent as hydroxyproline in the collagen of the periodontal ligament. He found that, not only were new collagen fibrils formed, but also that they were being added to existing collagen bundles.

Baumrind and Buck,[6] using a model system based on Macapanpan, Weinmann and Brodie's study,[48] injected rats undergoing molar separation with labeled proline and other metabolites at 6-hour intervals from 6 to 72 hours. They found that instead of an expected increase of collagen production on the tension side, and decreased activity on the

pressure side, a general decrease in metabolic activity was found.

Crumley,[18] in a similar study, found that collagen production in the periodontal ligament of tooth movement experimental rats was very similar to that of the control animals. He postulated that collagen was being formed at a rate very near its potential capacity.

These animal studies seem to indicate that, at least in early tooth movement, there is no change in the rate of new collagen formation. This information, clinically applied, seemed to indicate that the formation of new connective tissue around newly positioned teeth may not occur.

A high rate collagen degradation, which is caused by the presence of certain enzymes called collagenases, acting on the intra- and inter-molecular cross-linkages between collagen molecules and other tissue components, theoretically could act as a tissue releasing mechanism. However, under normal homeostatic conditions, collagen is highly resistant to the degenerative influence of the tryptic type of enzymes found in tissue fluids, although inflammation seems to cause collagen

degradation by a direct result of microbial collagenase production and the products of polymorphonuclear leukocytic lysis and degranulation, tooth movement per se has not been shown to cause an increase in collagen degradation.[81]

In a classic study designed to measure the elasticity of the fibers of the periodontal ligament, Urban, Beisler and Skillen[80] placed separators of various widths between the teeth of a dog. The separation achieved was retained for 30 minutes prior to animal sacrifice. Although separation ranges of .75 mm. to 2.0 mm. were performed, histological examination revealed that extensive tearing of the fibers of the periodontal ligament occurred at the lowest separation (.75 mm.). They said "such a tearing of the periodontium under the circumstances, clearly indicates the non-elastic quality of the fibers which constitute the membrane." However, in this experiment, forces were applied over a 10-minute time span. It appears that longer time intervals would have allowed more tissue accommodation to occur without periodontal damage.

As indicated in the above studies, the slow (or absent) rate of

collagen formation and degradation during tooth movement and its relative metabolic inactivity thereafter demonstrates its potential as a source for orthodontic relapse. However, the apparent inelasticity of collagen should limit the distance over which rapid tooth movement could occur without fiber rupture. The ability of teeth to be moved (and relapse) over distances in excess of collagen elasticity might indicate either that past collagen research was inaccurate or other relapse mechanisms are in effect.

Another possible explanation for the ability of a tooth to move beyond the apparent tensile limitations of the attached collagen bundles is that, instead of the principle fiber bundles of the periodontal ligament space stretching continuously from the cementum to bone, the individual fiber bundles do not reach across the entire space. Instead, it has been suggested that the fibers meet midway between cementum and bone and are spliced together in a zone referred to as an "intermediate plexus."[70] This arrangement would explain the facility of tooth movement to occur without requiring the formation of new connective tissue fibers and their

attachment to cementum and bone. The metabolic stability noted during tooth movement could be due to the presence of this non-metabolically active slippage mechanism.

However, Melcher and Eastoe[50] doubt the existence of the intermediate plexus by suggesting that it may be an artifact, resulting from the plane in which the principal fibers are sectioned.

In addition, Zwarych and Quigley[82] found evidence which also tended to disprove the existence of the plexus. They traced single fibers of the periodontal ligament completely from cementum to bone. They do suggest that fibers may pass directly from many small bundles on the tooth side, join with other bundles and end in a few large bundles on the alveolar side. This interconnection of fiber bundles, although not an intermediate plexus in the true sense, probably does allow some fiber adjustment to occur.

Reitan[62] does not discount the possibility of an intermediate plexus existing in the form of a mid-periodontal ligament proliferative zone. In an early study,[60] he noted a definite increase in cellular

elements of the periodontal ligament. He proposed that connective tissue cells in this area produce new fibrils which aid in elongation of the periodontal fibers, thus allowing further tooth movement.

Although the theoretical presence of an intermediate plexus would allow greater range of tooth movement in a specific time period, its potential importance in tissue-induced relapse is unknown at this time.

Another possible explanation for tissue recoil or relapse in excess of the elastic range of collagen is the presence of elastic fibers in the periodontium. Elastic fibers are highly resistant to deformation and return to their original length and width after cessation of a distorting force. They impart a diffuse elasticity to any tissue in which they branch.[41] However, although elastic fibers are present in the periodontal ligament, they are few in number and are mainly situated perivascularly[13,49,71] and not arranged in a manner to support the teeth during mastication.[25] Their importance in the relapse mechanism is unlikely.

However, in 1958, a new and previously unknown fiber, similar

histochemically to elastic fibers and apparently supportive in nature was observed in the peridontium of man by Fullmer and Lillie[33] who utilized a new histochemical procedure. Sections of formalin or alcohol fixed tissues were oxidized with peracetic acid and then stained with aldehyde fuchsin; many fibers that were unreactive without oxidation became visible. These fibers so stained were called oxytalan because of their resistance to acid hydrolysis (Greek: acid; to endure). Of interest is that these fibers were seen in increased numbers and density in areas of stress.[24,25,28,30] As would be expected then, an increase in oxytalan fibers was subsequently observed adjacent to orthodontically positioned teeth.[9,19,21]

Most of the research concerning oxytalan fibers has been that of Fullmer.[24-33] He demonstrated its existence as a fiber separate from collagen, elastic and reticular fibers by differential histo-chemical and enzymatic digestion techniques,[24,26,27,30,32] and by descriptive optical properties.[26,27]

His findings that oxytalan fibers were a distinct new fiber of the periodontium were later confirmed by other investigators.[35,47,55]

Although different from any other fiber type previously described, it has been reported that oxytalan fibers bear many similarities to elastic tissue: 1) Although there is a size difference between elastic and oxytalan fibers, both are homogeneous in appearance and have no periodicity as seen under the electron microscope.[14] 2) In animals, elastic fibers are seen in the transeptal region of the periodontium with a morphology analogous to oxytalan fibers in man. 3) On the basis of various histochemical methods,pre-elastic tissue and oxytalan fibers cannot be distinguished.[24] 4) Three of the elastic strains also stain oxytalan fibers in man.[27] However, Rannie, who confirmed the presence of oxytalan fibers in man, feels that there is no justification to believe that because oxytalan fibers respond (after oxidation) to stains which are usually considered specific for elastic tissue, that they are related to that tissue.[55]

Loe and Nuki[47] suggest that oxytalan fibers are not elastic in nature since they are not composed of elastin; their course in the periodontal ligament does not conform to any known component of the attached apparatus.

They offer the unsubstantiated opinion that oxytalan fibers are of neural origin.

Harris and Griffin,[42] using various histochemical and enzymatic digestion procedures on material from the developing human periodontium, reported that electron microscopy revealed a similarity in the reaction of the reticulum of ground substance microfibrils and oxytalan fibrils. They postulated that oxytalan fibrils could form as a result of end-to-end linkage, and later aggregation, of these ground substance microfibrils.

Selvig,[69] in an electron microscope study of human teeth with adhering periodontal tissue, noted that the unbanded fibrils previously described as oxytalan[14] were extensions of cross-banded matrix fibrils. Selvig proposed that "oxytalan" fibers represented partially decomposed collagen fibrils.

Therefore, controversy exists over the true nature of the oxytalan fiber. Histochemical procedures combined with light microscopy seems to indicate a difference between oxytalan fibers and other fiber types. In contrast, however, the use of electron microscopy reveals them to be

unbanded and similar to degenerating collagen or ground substance protein-polysaccharide complexes.

These fibers are classically described as being as much as 2 mm. in length and are round, elliptical, or flattened in cross-section, varying from 0.5 to 3 μ in diameter. They are anchored in either cementum or bone of the periodontal ligament but are not usually traceable from one to the other. A far greater proportion of oxytalan fibers insert into cementum than into alveolar bone. Generally oxytalan fibers follow the path of the principal fibers of the periodontium. In the area of the cemento-enamel junction, they either curve upward with the gingival fibers or join with the transeptal group. The largest and most numerous oxytalan fibers are found transeptally. In the middle and apical region of the root, they are smaller and less numerous.[27]

Simpson[71] noted that oxytalan fibers, arising at different levels of the root, follow different paths. This could have the effect of increasing tooth stability, especially with regard to rotation. He suggested that another purpose of these fibers might be to "transfix"

the collagen bundles. There seemed to be three types of oxytalan fibers according to location: one group interlaced various structures in the periodontal membrane and outlined vascular spaces, another group seemed to reinforce collagen bundles, and a third group was a fine fibrillar network seen near the cemental surface. This last group appeared to be the termination of many of the larger anchoring type fibers.

Rannie[55] postulated that because oxytalan fibers were embedded in bone and cementum and are woven through collagen fibers, they might have some anchoring effect perhaps to prevent overstretching of the tissue, thereby preventing ischemia due to obliteration of the vascular channels.

Carmichael,[13] in a histologic study of the distribution and "connexion" of the oxytalan fiber in the lower jaw of the mouse, demonstrated that the majority of oxytalan fibers ran between periodontal vessels and cementum, thus giving evidence to Rannie's hypothesis. Carmichael noted that the orientation of most of the oxytalan fibers was obliquely across the periodontal membrane, frequently perpendicular to the bundles of collagen. He also suggested that these fibers may be concerned with the

stability and patency of the periodontal vessel under pressure.

For some unexplained reason, although basically similar in arrangement to oxytalan fibers in adult teeth, oxytalan fibers in the periodontal membrane of deciduous teeth are oriented more apico-occlusally and frequently do not display an apical or occlusal attachment. They have been found to be fewer in number in the furcation areas of molars and are more prominent in a fibrillar network near cementum.[35]

Besides periodontal connective tissue, oxytalan fibers have been revealed to be present in certain pathological states, such as sclerosing hemangiomas,[75] ameloblastomas,[23] dental granulomas and cysts[29] and in some periodontal ligaments of people afflicted with scleroderma.[31]

The development of oxytalan fibers, as determined by chronological staining techniques in embryos and fetuses, seems to be from a mass of mucopolysaccharides between bundles of collagen.[30,42] They form in two locations: 1) adjacent and peripheral to the outer enamel epithelium (these fibers become part of the periodontal ligament and/or gingiva), and 2) the oral mucosa apical to the developing teeth (these become fibers of the gingiva).

When first seen, oxytalan fibers are 0.5 μ in diameter but increase in length and diameter when functional demands are placed on the teeth. The largest oxytalan fibers observed are in areas of stress in adults.[30]

This correlation between stress and oxytalan fibers caused some investigators to propose that oxytalan fibers might be the "elastic" type of tissue constituent responsible for tissue-induced orthodontic relapse.

Edwards[21] in 1968, in a study on rotational tooth movement and relapse in dogs, sought to examine the changes, if any, in the structure or orientation of this possible elastic tissue component (oxytalan) and subsequent relapse. Although a definite relationship could not be established between oxytalan fibers and relapse potential, several important observations were made. Oxytalan fibers were more numerous, particularly in supra-crestal areas, in the periodontiums of rotated teeth than in those of control teeth. Periods of retention did not eliminate this preponderance of oxytalan in the gingival tissue of rotated teeth. In serial sections of the entire length of the root, the

oxytalan fibers were more numerous in the buccal and lingual thirds of the periodontium than in the middle third. It is these buccal and lingual areas of the attachment apparatus which Reitan believes are subjected to the most tension during rotation. Surgical severance of these tissues was advocated as a means of controlling this relapse.

In a subsequent study designed to learn more about the cause of relapse in extraction sites, Edwards[19] noted that extraction space collagen was not piled up and coiled, as reported by Erikson,[22] but was "surprisingly normal." Edwards theorized that perhaps there was some mechanism which might re-organize transeptal tissues following orthodontic treatment. As before, oxytalan fibers were seen in increased numbers in gingival tissue adjacent to the experimental teeth. Normally, these fibers are relatively scarce. As in his rotational study,[20] surgical removal of piled up gingival tissue in the extraction space was advocated as the most practical preventive measure to eliminate extraction site opening.

Boese,[9] in a study of orthodontic rotation in animals, sought to

determine the influence of supra-alveolar fibers, in general, and oxytalan fibers, in particular, on rotational relapse. He confirmed Reitan's findings that two types of relapse mechanisms were active. In the first four weeks, he reported that relapse was due to the rebound effect of the stretched principal fibers of the periodontium (prior to the relaxing tissue effect of osseus recontouring). After eight weeks, relapse was caused primarily by supra-alveolar fibers. He noted that the number of oxytalan fibers and the amount of collagen in these tissues appeared to be increased by orthodontic tooth rotation. He attributed the second type of relapse to this proliferative phase of the supra-alveolar tissue. In contrast to Reitan's proposal that over-rotation and prolonged retention were the methods of choice for the prevention of rotational relapse, Boese found these procedures ineffective. Observing the histological differences of tissues adjacent to a) non-rotated teeth, b) rotated experimental control teeth not gingivectomized, and c) rotated gingivectomized teeth, he concluded that surgical gingival stripping procedures increased rotational stability six times that of experimental control rotated teeth.

The role of oxytalan fibers in this relapse mechanism could not be determined directly. But again, as in Edwards' studies, a correlation between relapse potential and oxytalan fiber build-up and configuration suggested the possibility that this "elastic" tissue had rebound potential.

In normal non-rotated teeth, oxytalan fibers were seen to follow a straight course, not spanning the transeptal area or the periodontal space. After traveling various distances from the cementum, they arborized and appeared to terminate in the collagen bundles.

In rotated control teeth (not gingivectomized), there was a marked increase in the number of oxytalan fibers, particularly in the transeptal area. Collagen also was seen to be present in increased numbers traveling with oxytalan in dense bands between the rotated and adjacent teeth.

In rotated experimental teeth (gingivectomized), oxytalan fibers in the transeptal region appeared in numbers greater than in control non-rotated teeth, but less than in rotated experimental control teeth. Compared to both the control non-rotated teeth and the non-surgical rotated teeth, the gingivectomized areas showed fewer bands of collagen

connecting the rotated and adjacent teeth. Of importance is the observation that in the transeptal area oxytalan fibers did not completely span the transeptal area and appeared wavy without tension, in contrast to the dense bands seen in rotated control teeth.

Boese, although not demonstrating specific characteristics or components of supra-alveolar tissue responsible for relapse, suggests that the higher concentration of oxytalan fibers seen combined with slower cemental remodeling compared to bone may prolong orthodontically induced tissue deformities.

Although oxytalan fibers,present in increased numbers and size in force distorted tissue, are suspect as a potential source for tissue rebound, this article and past research has not indicted them as the fibrillar tissue component responsible for relapse.

In summary, the pressure-tension concept of tooth movement of Sanstedt,[66,67] Oppenheim,[53] Herzberg,[44] Schwarz[68] and others,[62] demonstrated the plasticity of bone in tooth movement. According primarily to Reitan's work,[52,62] supra-alveolar tissue remained distorted

compared to that of osseus recontoured infra-alveolar tissue. Various procedures were advocated, both surgical[9,10,19-21,40,73,76-78] and non-surgical,[58-62] in an attempt to neutralize the tissue rebound effect of supra-alveolar fibers.

Inherent collagen, elastic and oxytalan tissue components, were examined in light of the ability of each to cause rotational and extraction site relapse.

Although collagen was determined to be relatively inelastic and slow metabolically, tooth movement beyond its elastic limits could be explained by the presence of an intermediate plexus or a proliferation of new fibers. However, relapse due to inelastic collagen should be self-limited. True elastic fibers are not present in large enough amounts in the periodontal tissues to cause relapse. Oxytalan, an elastic-type fiber, was consistently demonstrated in higher amounts in tissues under stress and with the greatest relapse potential. Although one particular fiber type probably is not solely responsible for tissue orthodontic relapse, oxytalan fibers must be considered potential sources.

Perhaps relapse problems of this nature in orthodontics could be better handled if the relapse role of oxytalan fibers were fully understood and their formation could be, in some way, controlled.

The purpose of this paper is to examine histologically the relationships between some of the fiber types present in the periodontium in histologic sections of human teeth which have undergone orthodontic tooth movement.

MATERIALS AND METHODS

Histological specimens from two patients who had undergone 14 days of tooth movement were combined with four patients from a former 21-day human tooth movement study.[39] All patients required extraction of maxillary first premolars as part of orthodontic therapy. The six patients included five males and one female, ages 12 to 15 with a mean age of 13.8.

Prior to extraction, the maxillary first premolars were tipped orthodontically by the method of Griffith and Mills and others.[16,17,36,46,60] The lingual appliance used was stabilized by cemented first molar bands to which was soldered a double helix finger spring configuration fabricated from .016 regular orthodontic wire (Unitek) (Fig. 1). In all patients, the tooth most unencumbered by occlusal and proximal contacts was selected for tooth movement and called experimental; its contralateral counterpart became the control. The appliance was calibrated by a dead weight loading device to deliver 70 $\pm$ 7 grams over a 3-4 mm. working range.

Small indentations were placed in the lingual cusp tips of both maxillary first premolars with a dental handpiece using a ¼-inch round bur. Measurements from cusp tip to cusp tip were obtained with a standard boley gauge and recorded.

The appliances were activated to deliver the required force and seated in the mouth. The patients undergoing 14 days of tooth movement were observed on the 7th day and on the day of surgical removal, the 14th. In the other four patients, the appliances were observed on the 7th, 14th, and on the day of surgical removal, the 21st. At the last observation period interpremolar measurements were again obtained from the same indentation points on the premolar cusp tips in order to determine the amounts of tooth movement which had taken place.

At the end of each observation period, the maxillary premolars were removed surgically along with approximately 7 mm. vertical height of attached buccal alveolar bone. The tissue was rinsed in running water and placed in a neutral buffered 10% formalin solution. Twenty-four hours later the crowns of the teeth were removed by a high speed dental

handpiece with a water spray-cooled 701 bur. The specimens were then fixed for approximately 10 days. Decalcification with Kristensen's decalcification solution (sodium formate and formic acid) took from 16 to 18 days. In the 14-day sample, the tissues were embedded in paraffin and 7 micron frontal sections were cut with a clinical microtome. The 12 micron frontal sections from the 21-day sample had been obtained by microtome sectioning of carbon dioxide frozen specimens. These sections had been stored in water prior to the present study. All sections were mounted on glass slides and three sections of each specimen were stained with hematoxylin and eosin, Wilder's reticular stain, Mallory's connective tissue stain, Verhoeff's elastic tissue stain and the peracetic acid-aldehyde fuchsin stain for oxytalan fibers. The oxidation step prior to all stains for oxytalan fibers were done either with peracetic acid or with 15% potassium monopersulfate. For contrast, Verhoeff's, von Gieson's and light green stains were used as alternative counterstains to the aldehyde fuchsin.

Each slide was examined under a light microscope and photomicrographs were made on a Zeiss photoscope.

HISTOLOGICAL OBSERVATIONS

The total sample for this study consisted of 12 maxillary first premolar teeth with attached alveolar bone, a control and an experimental from six patients. In two of these patients, the duration of orthodontic movement was 14 days; in the remaining four, the teeth were moved for 21 days. The range of tooth movement, obtained as indicated by interpremolar measurements, was between .5 and 1.5 mm.

The results below, unless specified, were observed on the alveolar (pressure) side of the periodontal ligament of the first premolars.

The histological specimens from the two patients of the 14-day sample were disappointing. In only two of the four specimens was alveolar bone in its normal relationship in the periodontal ligament. In the other specimens, the bone had been displaced at some point in the histological process prior to paraffin embedding. Comparisons between all control and experimental models in this sample, therefore, could not be done. Control and experimental sections from one individual were artifact-free and were

could be seen to be taking place.

Mallory's stain was unremarkable in both the control and experimental sections. While of value in clearly observing the attachment of Sharpey's fibers to bone, the collagen arrangement in the mid-periodontal ligament area was indistinct.

An apparent difference was observed between control and experimental sections stained with Wilder's reticular stain. Control specimens appeared to contain fewer of these darkly stained fibers than were present in the periodontal pressure areas of the experimental teeth. The arrangement and distribution of these fibers were similar to that of the collagenous bundles. In narrowed areas of the periodontal ligament, most fibers ran parallel to the root surface, while in thicker areas, the fiber configuration became more haphazard. In one specimen, active frontal resorption was evident on the lingual surface of the buccal root tip. In this area, reticular stained fibers were demonstrated in especially thick arrangements.

Oxytalan fibers were observed in both the control and experimental sections. In the control sections, they were commonly few in number,

fine in appearance, and in the thinner areas of the periodontal ligament, traveled next to and parallel with the cemental root surface. In the thicker areas, they were not regularly arranged and were generally more delicate in appearance. The fibers seem to originate from the cemental surface of the root and, after traveling a short distance outward, turned and,after joining with other fibers,continued in a parallel direction to the root surface. Oxytalan fibers were also present in the walls of larger vessels in the periodontal ligament.

In the experimental section, the oxytalan fibers did not appear markedly different from that of the control. The arrangement of the fibers was similar to that of the non-orthodontic specimens and the size of the fibers was not markedly different. The oxytalan fibers generally did not seem to run in the same direction as either the collagen fibers or reticular stained fibers. They usually were observed oriented at an angle to these fibers.

Stains for elastic fibers (Verhoeffs and the oxytalan control stains) were inconclusive. None could be demonstrated, except occasionally in

the wall of a larger vessel of the periodontium.

The 21-day control group stained with hematoxylin and eosin was demonstrative of the periodontium at rest. The width of the periodontal ligament in most specimens was approximately 400 microns. The collagen fibers appeared to be densely packed, yet wavy and traveled obliquely apically from bone to tooth except adjacent to neurovascular bundles where the fibers seemed somewhat disarranged. Large patent vessels were commonly present, especially in the occlusal one-half of the periodontal ligament. Alveolar bone marrow spaces generally were without many demonstrable cells. Although small localized areas of osteoblastic and osteoclastic activity were in evidence, alveolar bone itself appeared to be relatively inactive as no large areas of osteoid formation or undermining resorption were apparent.

Hematoxylin and eosin sections of the 21-day experimental group revealed the classic picture of periodontal re-organization associated with prior tooth movement. Occasional cell-free areas, with associated compression of the collagen fibrils, were evident. Numerous multinucleated

giant cells were in evidence in the periodontal ligament space. Proliferating fibroblasts were quite obvious as was the presence of many small capillaries.

Evidence of previous undermining resorptive activity was noticeable as localized areas of markedly increased periodontal ligament width. In these areas, the periodontal tissue seemed to bulge inward into an opening in the adjacent alveolar bone. In this experimental group, the collagen fibers did not seem to be as densely packed and as regularly arranged as in the control group. Collagen re-organization, as evidenced by smaller, less dense fibers with little apparent arrangement, was obvious in most non-cell free areas.

The collagen staining ability of Mallory's connective tissue stain did not yield new information. The collagen fibrillar arrangement could also be quite adequately observed in hematoxylin and eosin-stained sections. Although cell-free areas were few in the experimental samples, in those present, all inherent compressed collagen stained blue.

Wilder's reticular stain was useful in collagen contrast comparisons with the other stains. Collagen fibers, appearing as dark thick bundles,

could be traced quite easily from both tooth and bone into the substance of the periodontal ligament (Fig. 2). Although fiber bundles could be plainly seen and traced, in no instances could they be seen to extend directly from cementum to bone.

The stains for oxytalan fibers were quite revealing. These fibers varied markedly in both length, presumably due to sectioning, and diameter (Fig. 3). Many fibers, small in diameter and wavy in appearance seem to eminate from the cementum as fine fibrils. As they continued into the periodontal ligament proper, they joined with additional fibers, appearing as darker, thicker wavy bands (Fig. 4). The fibers generally traveled parallel to each other in the long axis of the tooth. The termination of many of these fibers, if not joining with one another, was not easily determined. Few fibers were seen to be attached or closely adjacent to alveolar bone. The majority of oxytalan fibers, therefore, were observed on the cemental side of the periodontal ligament. Compared with collagen fibers, the arrangement and distribution of oxytalan fibers was usually different. In most instances, oxytalan

fibers were seen to be at an angle to the general pattern and direction of most of the collagen fibers. Yet in some sections oxytalan fibers could be seen travelling parallel to, and interspersed with, collagen. Oxytalan fibers were especially numerous in the walls of the large blood vessels of the periodontal ligament.

Where sections included the cemento-enamel junction and tissues of the gingiva, oxytalan fibers were observed to be extremely numerous. Most fibers in this area were observed to travel subjacent to the basement membrane of the epithelium lining the gingival crevice, arcing upward in parallel fibers into the lamina propria of the gingival tissue proper. Other oxytalan fibers in this area, not arising subepithelially, were as numerous, but were not as regularly arranged.

Although separation artifacts between alveolar bone and cementum made many intrapair comparisons difficult, it appeared that not only were oxytalan fibers more prevalent in these experimental sections, they also were of greater diameter.

Verhoeff's elastic tissue stain and the oxytalan control sections

(stained exactly as the other oxytalan stains but not oxidized with either peracetic acid of monopersulfate, were not of value in demonstrating this type of fiber.

DISCUSSION

In previous tooth movement studies,[16,17,36,38,46,60] the lingual appliance was suitable as a source of force for tipping tooth movement, but there was a tendency for the active finger spring to slip subgingivally. For this reason, this type of appliance is best suited to studies of less than 30 days duration. Perhaps fixed banded appliances would be more advantageous in longer studies.

To ensure that the laboratory calculated force is delivered to the periodontal tissues on the pressure side, occlusal and proximal contact relationship should be absent or at lease minimal. In this study the tooth most unencumbered by such occlusal and proximal contacts was chosen to be moved experimentally. This probably was not totally suitable, as variations in force delivery undoubtedly occurred dependent on remaining tooth contacts.

Surgical removal of the orthodontically tipped teeth and attached alveolar bone was done quite uneventfully. Healing has been without

complications in the complete series of human tooth movement patients.[16,17,36,38] All patients are, or have been, successfully treated orthodontically with no untoward effects.

Histological preparation of the tissue specimens was accomplished without excessive separation between alveolar bone and root surface in six of eight patients in the 21-day sample. In the 14-day sample, bone unaccountably was dislodged in two of four samples, rendering them almost useless for research material.

Frontal sections were used for study as histological examination of these sections was determined to be most preferable for study of the fibrillar elements of the periodontium and their configurations. Conversely, the study of cell-free or hyalinized areas may be best accomplished utilizing cross-sectioned material. If frontal sections are to be used to study pressure areas of the periodontium, they must include the greatest height of the mesio-distal tooth contour, which is the probable site of the greatest concentration of pressure and, therefore, cell-free area formation. Sections cut mesially or distally to this height of contour undoubtedly would not include cell-free areas.

In fact, it is probable that periodontal width may not even be markedly reduced in these areas. In the frontal sections of this study many periodontal ligament spaces of experimental samples were not severely reduced in width. This could be due to sectioning technique or, more likely, is the result of an increase in periodontal ligament width as periodontium re-organization occurred during the 21 days of tooth movement. Possibly due to the above reasons, cell-free areas were not a significant finding in this study.

The histological findings with Wilder's reticular stain were of interest because it appeared that a greater concentration of reticular stained fibers were present in areas where active osteoclastic activity (and possibly increased collagen metabolism) had occurred. There is still controversy as to the true nature of reticular fibers. However, it is possible that these reticular fibers, observed in areas of active metabolism, are miniature immature collagen fibrils. The morphology and distribution of these fibers is similar to that of collagen, thereby lending some validity to this hypothesis. It is also possible that the

histochemical reactions of this more immature, and therefore chemically active reticular-type collagen, might differ from the more stable, less reactive mature collagen. Reticular stained fibers were quite remarkable in their clear appearance and distribution and could be traced quite easily into the body of the periodontal ligament. But unlike Zwarych and Quigley's[82] apparent negation of the existence of the intermediate plexus by their observations of fibers extending directly from cementum to bone, none were seen to do so in these samples.

The existence of the intermediate plexus remains probable, but not scientifically demonstrable, most likely due to the difficulty in achieving tissue sectioning through the periodontal ligament in a plane parallel to the anchoring infrabony fibers.

Oxytalan fibers were seen quite readily with all stains utilizing the oxidization step as performed according to Rannie.[55] In most sections, there was little difference in the number and diameter of oxytalan fibers in the experimental and the control specimens. However, there was an increase in both size and number of these fibers in one of the 14-day

control and experimental samples. Others[9,19-21,24,25,28,30] have reported that a direct relationship between stress and the numbers and size of oxytalan fibers does exist. In general, this study did not confirm that observation. The fibers were seen to exist in the three arrangements mentioned by Simpson.[71] Most of the fibers eminating from the cementum and travelling relatively parallel to the root surface could be labelled as tooth anchoring fibers. Fibers observed with little arrangement, seemingly at angles to the general pattern of collagen bundles, might indeed be some sort of collagen-anchoring fibril. A different type was seen in the connective tissue stroma surrounding the largest blood vessels. No mention was made of gingival oxytalan fibers by Simpson. In this study they appeared to be in the greatest concentrations.

It has been suggested that oxytalan fibers may not be a new fiber type as Fullmer has described but rather a partially decomposed collagen fiber or a type of unbanded elastic type fiber. Loe and Nuki[47] have even suggested that these fibers are nerves, but nothing was observed

which would reinforce this opinion. The possibility that oxytalan fibers are decomposing collagen fibers is unlikely as the distribution and general arrangement of oxytalan fibers is usually not similar to that of collagen. In addition, collagen fibers are regularly observed to become anchored in alveolar bone. Oxytalan fibrils do not appear to do this with much regularity.

The theory that oxytalan fibers may be elastic in nature may have some validity. As reported in the review of literature, the usual stains for elastic fibers, subsequently oxidized with either peracetic acid or permonosulfate, allow this new fiber configuration to be expressed. This does suggest a histo-chemical similarity. In addition, elastic fibers observed under the electron microscope are noted to be unbanded. Oxytalan fibers have been similarly described. The arrangement of oxytalan fibers perivascularly in the periodontal ligament is clearly reminiscent of elastic fiber configurations.

Oxytalan fibers have been seen by others as eminating from the root and, after traveling a certain distance through the substance of the

periodontal ligament, inserting into alveolar bone. In this study, it was interesting that fibers could be plainly seen near the root cementum and in the mid-periodontal ligament area, but very few were observed adjacent to alveolar bone. One hypothesis of oxytalan fiber function is that they are an elastic type of periodontal supportive fiber thought to be a significant factor in certain types of orthodontic relapse. In order for this tissue-induced relapse to occur, it is probable that a sizeable force must be applied to the tooth. The observation that few oxytalan fibers attach directly to bone seems to be inconsistent with this hypothesis.

For intra-alveolar oxytalan fibers to be an important factor in short-term relapse, one would expect: 1) a larger than normal number of these fibers, 2) an increase in fiber bundle size and arrangement, and 3) more fibrillar attachment directly to alveolar bone.

In contrast, supra-alveolar oxytalan fibers, seen in those few sections which included some attached gingiva, were of greatest in number and density. In these gingival areas, it is probable that they could quite easily exert a formidable post-treatment relapse force. The clinical success

of Edwards,[19-21] Boese,[9] Brain,[10] and others may be due, not only to severance of stretched and displaced collagen fibers, but also to the elimination of large numbers of oxytalan fibers. If these fibers, especially prominent supra-alveolarly, are indeed of elastic nature, their surgical neutralization could certainly ensure the clinical success of the various tissue detachment types of relapse preventive procedures.

The role of collagen in the relapse mechanism is probably that of an inhibitor of short-range tooth movements. Removal of stress applied to these metabolically inactive fibers within their elastic limit may result in tissue relapse.

Long-range tooth movements have been reported[56] to result in better long-term stability. It is possible that collagen fibrils rupture as their elastic limit is exceeded, or the concept of an intermediate plexus is valid. Perhaps metabolic activity in mid-periodontal ligament has not been adequately determined by the proline labeling technique and, in fact, is more active than has been apparent. These possibilities would seemingly explain the

movement of a tooth over long distances without massive relapse subsequently occurring.

Mature collagen, extending directly from cementum to alveolar bone infra-alveolarly and supra-alveolarly connecting tooth to tooth, must be self-limited in its ability to stretch (and relapse). Collagen, therefore, is probably more important in infra-alveolar tissue relapse mechanisms, whereas the elastic-type oxytalan fiber, if indeed it is a separate fiber type interlaced with collagen, may be an additional important factor in long-term supra-alveolar tissue-induced relapse.

Further research is necessary to determine the "normal" variations in oxytalan fiber numbers and size, expecially in gingival tissue. Methods of quantifying an increase or decrease in those fibers should be formulated. Only then can oxytalan fibers be accurately described as present in increased numbers. Histochemical and electron microscopy studies must first be able to authenicate the existence of oxytalan fibers and differentiate them from elastic fibers. Additional research in these areas should be rewarding in helping to reduce or eliminate

periodontal tissue influences on orthodontically-positioned teeth.

SUMMARY

The purpose of this study was to provide more information on the role of the fibrillar components of the periodontium in tissue-induced orthodontic relapse. Of interest was the theory that "elastic" oxytalan fibers of the periodontium were important as possible causes of tooth position relapse, and a general review of the orthodontic tooth movement literature was presented.

The material for this study consisted of 12 orthodontic patients requiring extraction of maxillary first premolars as a part of treatment. A force of 70 ± 7 grams was applied to one of these premolars for 14 days in two patients and 21 days in six patients of a former study. The contralateral premolar was used as a non-orthodontic control.

Histological frontal sections were obtained from the teeth and vertically attached buccal alveolar bone. These sections were stained with hematoxylin and eosin, Wilder's reticular stain, Mallory's connective tissue stain, Verhoeff's elastic stain, and variations of the peracetic

acid-aldehyde fuchsin stain for oxytalan fibers. Eight of 12 samples were suitable for study. The slides were observed under a light microscope. The conclusions were:

1. Frontal sections were preferable for study of the fibrillar composition of the periodontal ligament.

2. Cell-free areas were rare in these specimens either due to normal periodontal re-organization occurring as a part of tooth movement, or histological sectioning in a plane distant from the greatest height to mesial-distal tooth contour, the most probable site of cell-free area formation.

3. With the exception of one 14-day tooth movement patient, tissue specimens were not of sufficient quality to confirm the findings of Fullmer and others who found that oxytalan fibers were increased in areas of stress, i.e. in association with orthodontic tooth movement.

4. Oxytalan fibers were seen in three general locations--the first eminated from root cementum and, after travelling a short distance into the periodontal ligament, joined with other similar fibers. The function

of this group was thought to aid in anchoring the tooth to alveolar bone. A second group was observed, more variable in arrangement than the first and at angles to the general orientation of the collagen bundles. This group has been described as a collagen supportive role. The third group was seen in the walls of the larger vessels of the periodontium. A relationship to vascular patency and support is suspected.

5. In a few sections which included gingival tissue, darkly-stained oxytalan fibers were seen in increasing size and large concentrations. In contrast, the size and numbers of oxytalan fibers in most areas of the infra-alveolar periodontal ligament were unremarkable.

6. Reticular-stained fibers were observed to be in increasing numbers in areas of new bone formation.

BIBLIOGRAPHY

1. Angle, E.H.: Bone growing. Dental Cosmos 52:261, 1910.

2. Atherton, J.D.: Gingival response to orthodontic tooth movement. Am. J. Orthodont. 58:179, 1970.

3. Atherton, J.D. and Kerr, N.W.: Effect of orthodontic tooth movement upon the gingivae. Brit. Dent. Journal 124:555, 1968.

4. Bassett, C.A.L. and Becker, R.O.: Generation of electric potentials by bone in response to mechanical stress. Science 137:1063, 1962.

5. Baumrind, S.: A reconsideration of the propriety of the "pressure-tension" hypothesis. Am. J. Orthodont. 55:12, 1969.

6. Baumrind, S. and Buck, D.L.: Rate changes in cell replication and protein synthesis in the periodontal ligament incident to tooth movement. Am. J. Orthodont. 57:109, 1970.

7. Bien, S.M.: Hydrodynamic damping of tooth movement. J.D. Res. 45:907, 1965.

8. Black, G.V.: Operative dentistry, Vol. 4. Chicago, Medico-Dental Publishing Co., 1938, p. 19.

9. Boese, L.R.: Increased stability of orthodontically rotated teeth following gingivectomy in Macaca Nemestrina. Am. J. Orthodont. 56:273, 1969.

10. Brain, W.E.: The effect of surgical transsection of free gingival fibers on the regression of orthodontically rotated teeth in the dog. Am. J. Orthodont. 55:50, 1969.

11. Buck, D.L. and Church, D.H.: A histologic study of human tooth movement. Am. J. Orthodont. 62:507, 1972.

12. Burstone, C.J. and Groves, M.H.: Threshold and optimum values for maxillary anterior tooth movement. J.D. Res. 39:695, 1961.

13. Carmichael, G.G.: Observations with the light microscope on the distribution and connexions of the oxytalan fibre of the lower jaw of the mouse. Archs. Oral Biol. 13:765, 1968.

14. Carmichael, G.G. and Fullmer, H.M.: The fine structure of the oxytalan fiber. J. Cell Biol. 28:33, 1966.

15. Case, C.S.: Dental Orthopedia. Chicago, C.S. Case and Co., 1921.

16. Christensen, Dean A.: Early tissue changes during human tooth movement. Certificate thesis, University of Oregon Dental School, June 1972.

17. Church, Dean H.: Histologic changes occurring during human tooth movement. Certificate thesis, University of Oregon Dental School, June 1971.

18. Crumley, P.J.: Collagen formation in the normal and stressed periodontium. Periodontics 2:53, 1964.

19. Edwards, J.G.: The prevention of relapse in extraction cases. Am. J. Orthodont. 60:128, 1971.

20. Edwards, J.G.: A surgical procedure to eliminate rotational relapse. Am. J. Orthodont. 57:35, 1970.

21. Edwards, J.G.: A study of the periodontium during orthodontic rotation of teeth. Am. J. Orthodont. 54:441, 1968.

22. Erikson, B.E.,et al.: Orthodontics and transeptal fibers. AJO and OS 31:1, 1945.

23. Fisher, A.K., et al.: Oxytalan fibers in ameloblastomas. OS, OM and OP 15:246, 1962.

24. Fullmer, H.M.: A comparative histochemical study of elastic pre-elastic and oxytalan fibers. J. Histochem. and Cytochem. 8:290, 1960.

25. Fullmer, H.M.: A critique of normal connective tissues of the periodontium and some alterations with periodontal disease. J.D. Res. 41:223, 1962.

26. Fullmer, H.M.: Differential staining of connective tissue fibers in areas of stress. Science 127:1240, 1958.

27. Fullmer, H.M.: Histochemical studies of the periodontium. J.D. Res. 45:469, 1966.

28. Fullmer, H.M.: A histochemical study of periodontal disease in the maxillary alveolar processes of 135 autopsies. J. Perio. 32:206, 1961.

29. Fullmer, H.M.: Observations on the development of oxytalan fibers in dental granulomas and radicular cysts. Arch. Path. 70;59, 1960.

30. Fullmer, H.M.: Observations on the development of oxytalan fibers in the periodontium of man. J.D. Res. 38:150, 1959.

31. Fullmer, H.M.: Periodontal membrane affected by scleroderma. Arch. Path. 73:184, 1962.

32. Fullmer, H.M. and Lillie, R.D.: The peracetic acid-aldehyde fuchsin stain. J. Histochem. and Cytochem. 6:391, 1958.

33. Fullmer, H.M. and Lillie, R.D.: The oxytalan fiber: a previously undescribed connective tissue fiber. J. Histochem. and Cytochem. 6:425, 1958.

34. Glickman, I.: Clinical periodontology, ed. 4 Philadelphia, W.B. Saunders Co., 1972.

35. Goggins, J.F.: The distribution of oxytalan fibers in periodontal ligaments of deciduous teeth. Periodontics 4:182, 1966.

36. Goya, H.H.: Tissue changes incident to twenty-one days of tooth movement. Certificate thesis, University of Oregon Dental School, June 1972.

37. Grieve, G.W.: Some theories obstructing the progress of the science of orthodontics. Int. J. Ortho. 18:5, 1932.

38. Griffin, C.J. and Harris, R.: The fine structure of the developing human periodontium. Archs Oral Biol. 12:971, 1967.

39. Griffith, D.S. and Mills, M.J.: Confirmation of lipids in human tooth movement. Certificate thesis, University of Oregon Dental School, June 1973.

40. Hallett, G.E.M.: Immediate torsion - a preliminary report on 23 cases. Dent. Pract. 7:108, 1956 and 7:126, 1957.

41. Ham, A.W. and Leeson, T.S.: Histology, ed. 4, Philadelphia, J.B. Lippincott, 1961.

42. Harris, R. and Griffin, C.J.: The protein-polysaccharide complex of the human developing periodontium. Archs Oral Biol. 12:1107, 1967.

43. Hasegawa, J.: Oxytalan fibers of the dermal-epidermal junction. Arch. Dermat. 82:250, 1960.

44. Herzberg, B.L.: Bone changes incident to orthodontic tooth movement in man. J.A.D.A. 19:1777, 1932.

45. Kvam, E.: A study of the cell-free zone following experimental tooth movement in the rat. Trans. Eur. Orth. Soc. 419, 1969.

46. Kvam, E.: Organic tissue characteristics on the pressure side of human premolars following tooth movement. Angle Orthod. 43:18, 1973.

47. Loe, H. and Nuki, K.: Observations on the peracetic acid-aldehyde fuchsin (oxytalan) positive tissue elements in the periodontium. Acta Odonta. Scan. 22:579, 1964.

48. Macapanpan, L.C., Weinmann, J.P., and Brodie, A.G.: Early tissue changes following tooth movement in rats. Angle Orthod. 24:79, 1954.

49. Maximow, A.A. and Bloom, W.: A textbook of histology. Philadelphia, W.B. Saunders and Co., 1957.

50. Melcher, A.H. and Eastoe, J.G.: "Connective tissues" in Biology of the periodontium. Melcher and Brown, London, Academic Press, 1969, p. 228.

51. Nance, H.N.: The limitations of orthodontic treatment II.Diagnosis and treatment in the permanent dentition. A.J.O. and O.S. 33:253, 1947.

52. Neuberger, A., Perrone, J.C., and Slack, H.C.: The relative inertia of tendon collagen in the rat. J. Biol. Chem. 49:119, 1951.

53. Oppenheim, A.: Tissue changes, particularly of the bone, incident to tooth movement. Amer. Orthod. 3:57, 113, Oct. 1911, Nov. 1912.

54. Piez, Karl A.: Maturation of collagen. J. Dent. Res. 45:463, 1966.

55. Rannie, I.: Observations on the oxytalan fibre of the periodontal membrane. Trans Eur. Ortho. Soc. 1, 1963.

56. Reidel, R.A.: A review of the retention problem. Angle Orthod. 30:179, 1960.

57. Reitan, K.: Clinical and histologic observations on tooth movement during and after orthodontic treatment. Am. J. Orthodont. 53:721, 1967.

58. Reitan, K.: Principles of retention and avoidance of post-treatment relapse. Am. J. Orthodont. 55:776, 1969.

59. Reitan, K.: Tissue behavior during orthodontic tooth movement. Am. J. Orthodont. 46:881, 1960.

60. Reitan, K.: The initial tissue reaction incident to orthodontic tooth movement. Acta Odont. Scan. Sup. 6, 1951.

61. Reitan, K.: Tissue rearrangement during retention of orthodontically rotated teeth. Angle Orthodont. 29:105, 1959.

62. Reitan, K.: "Biomechanical principles and reactions" in Current orthodontic concepts and techniques, Vol. I, Graber, T.M., Philadelphia, W.B. Saunders Co., 1969.

63. Rygh, P.: Ultrastructural changes in pressure zones of human periodontium incident to orthodontic tooth movement. Acta Odont. Scand. 31:109, 1973.

64. Rygh, P.: Ultrastructural changes of the periodontal fibers and their attachment in rat molar periodontium incident to orthodontic tooth movement. Scand. J. Dent. Res. 81:467, 1973.

65. Rygh, P.: Ultrastructural cellular reactions in pressure zones of rat molar periodontium incident to orthodontic tooth movement. Acta. Odont. Scand. 30:575, 1972.

66. Sandstedt, C.: Nagra bidrag tiu tandregleringens teori. Stockholm, 1901 (as reported by Reitan, 1951).

67. Sandstedt, C.: Einige beitrago zur theorie der zahnregulierung. Nordish Tandlakare Tidsrift, No. 4, 1904 and Nos. 1 and 2, 1905 (as reported by Schwarz, 1932).

68. Schwarz, A.M.: Tissue changes incident to orthodontic tooth movement. Int. J. O. & O.S. 18:331, 1932.

69. Selig, K.A.: Nonbanded fibrils of collagenous nature in human periodontal connective tissue. J. Periodont. Res. 3:169, 1968.

70. Sicher, H.: (quoting Sicher, H.: Ztschr Stomatol 21:590, 1923) Changing concepts of supporting dental structures. O.S., O.M. & O.P. 12:31, 1959.

71. Simpson, H.E.: A three-dimensional approach to the microscopy of the periodontal membrane. Proc. R. Soc. Med. 60:537, 1967.

72. Skillen, W.G. and Reitan, K.: Tissue changes following rotation of teeth in the dog. Angle Orthod. 10:140, 1940.

73. Skogsburg, C.: The use of septotomy (surgical treatment) in connection with orthodontic treatment, and the value of this method as proof of Walicoff's theory of tension of the bone after regulation of the teeth. Int. J. of O., O.S. 18:1044, 1932.

74. Stallard, R.E.: The utilization of H^3 Proline by the connective tissues of the periodontium. Periodontics. 1:158, 1963.

75. Tedeschi, C.G. and Sommers, S.C.: Oxytalan fibers in sclerosing hemangiomas. Arch. Dermat. 84:128, 1961.

76. Thompson, H.E.: Orthodontic relapses in a study of connective tissue fibers. Am. J. Orthodont. 5:93, 1959.

77. Thompson, H.E.: Preliminary macroscopic observations concerning the potentiality of supra-alveolar collagenous fibers in orthodontics. Am. J. Orthodont. 44:485, 1958.

78. Thompson, H.E.: The role of connective tissue fibers in retention of orthodontic tooth movement. Dental Survey 34:342, 1958.

79. Tweed, C.W.: Indications for extraction of teeth in orthodontic procedure. Am. J. Orthodont. 30:405, 1944.

80. Urban, L.B., Beisler, E.H., and Skillen, W.G.: Tissue disturbances caused by mechanical separation of the teeth of the dog. J.A.D.A. 18:1943, 1931.

81. Weissman, A.M.: A review of literature: Collagen: It's physical characteristics and degradation. J. Periodontology-Periodontics 40:611, 1969.

82. Zwarych, P.D. and Quigley, M.B.: The intermediate plexus of the periodontal ligament; history and former observations. J.D. Res. 44:381, 1965.

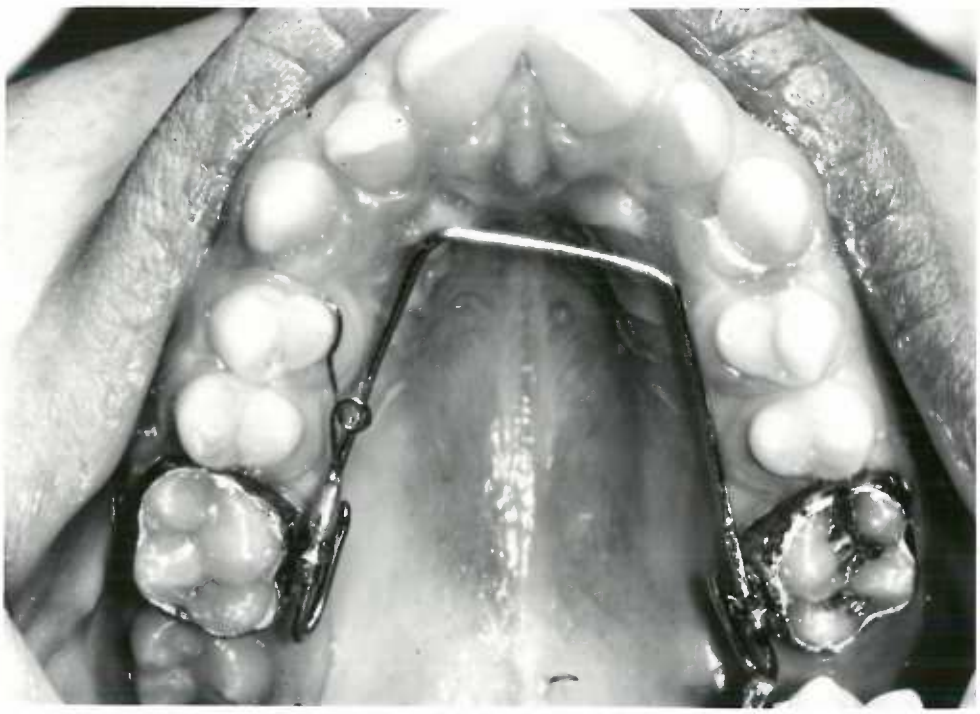

Fig. 1 Representative tooth movement appliance

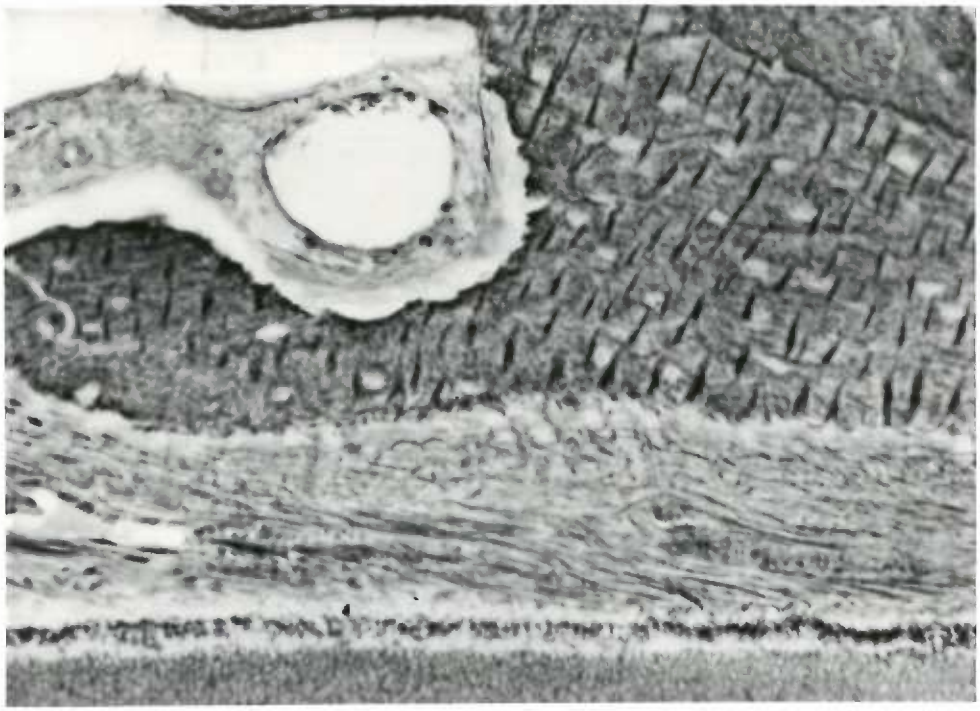

Fig. 2 Wilder's recticulum stain. Note orientation of stained periodontal fibers from root cementum into the periodontal ligament. Sharpey's fibers seen entering bone.

X64

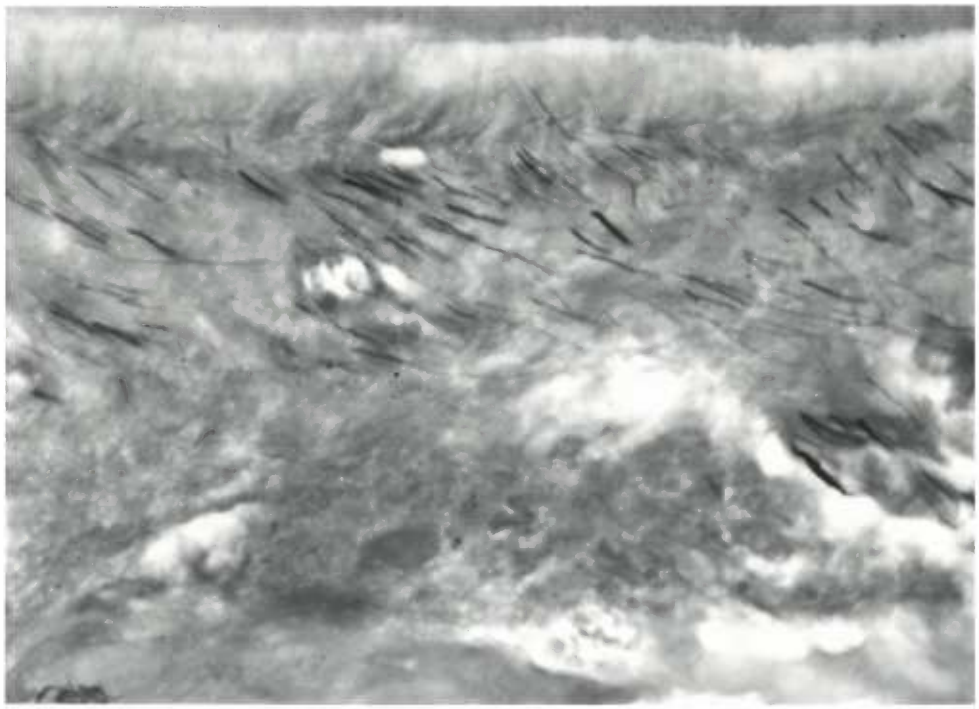

Fig. 3 Oxytalan fibers. Note variation in diameter. Root surface at top.

X40

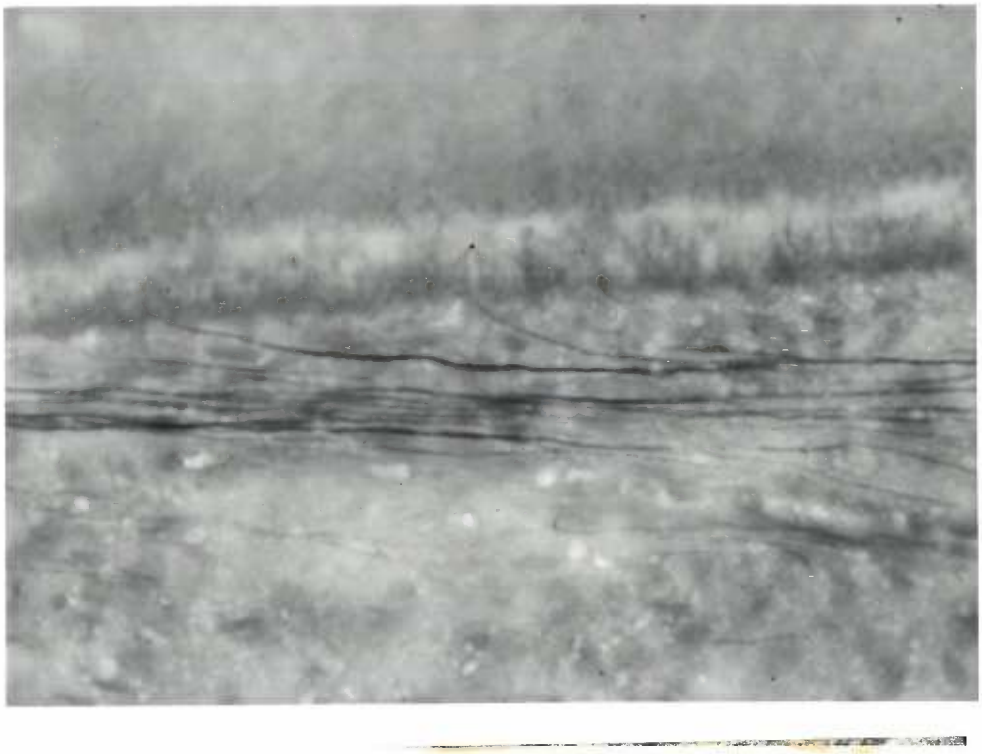

Fig. 4 Oxytalan fibers originating from root cementum at top, joining with other fibers in mid-periodontal ligament.

X160