Forced eruption and implant site development: Soft tissue response

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The increased use of implants in orthodontics has stimulated interest in augmenting bone in patients who have deficient alveolar ridges that preclude ideal implant placement. A nonsurgical technique for increasing the amount of available bone for implant site development and fixture placement is orthodontic extrusion, or forced eruption. The concept of a tooth moving coronally by orthodontic means and the clinical alterations in the soft tissue architecture of the periodontium demonstrated during orthodontic extrusive movement of periodontally compromised teeth have demonstrated a direct relationship of pocket depth reduction, accompanied with an immature appearing tissue, "the red patch." This article will describe the periodontium during eruptive tooth movement. (Am J Orthod Dentofac Orthop 1997;112:596-606.)

A basic tenet of osteophysiology is that the fibers of the periodontal membrane are secured to the bone by the formation of new bone around the ends of the fibers. Therefore, when tension is applied to the periodontal ligament, periodontal fiber bundles are elongated, and osteoblasts are induced to deposit new bone in the areas of the alveolus where periodontal attachment exists.

The alteration of the hard tissue and soft tissue components by forced eruption is based on sound orthodontic and osteophysiologic principles.[1,2] Brown[3] suggested that there is a potential for reduction in pocket depth, increase in attachment apparatus and change in the architecture of both hard and soft tissues of the periodontium. Ingber[4,5] showed that teeth could be erupted for the purpose of lengthening the clinical crown, altering the gingival margins, and leveling the osseous defects. He noted that as teeth were extruded by using light forces, both the bone and gingiva migrated coronally.[4,5]

The contemporary use of forced eruption has been to improve the three-dimensional topography of the recipient implant site before extraction. Subsequently, implant placement would follow the stabilization period. Noting that hopeless teeth are not useless teeth, Salama and Salama[6] showed that the attachment apparatus can be used to develop the bone and gingiva in an incisal or occlusal direction for the development of a potential implant site.

Recently, Mantzikos and Shamus[7] have reported on the use of forced eruption in augmenting bone in patients who have deficient alveolar ridges that preclude ideal implant placement. Consequently, implant site development guided by forced eruption enhances two specific areas: the hard tissue component and the soft tissue component.

Both of the areas are essential to the success of an implant restoration. The volume of the osseous structure must allow for implant placement in an ideal situation for the restoration, whereas the anatomic topography of the soft tissue must mimic that of the adjacent soft tissue architecture.[8,9]

The initial effect of orthodontic tooth movement on the gingiva has been studied. Reitan[10,11] demonstrated that eruptive tooth movement results in a stretching of the gingival and periodontal fibers, which produces a coronal shift of bone and gingiva. Elongation of the tooth in its alveolus causes a stretching of the gingival and periodontal ligament fibers. This results in a coronal shift of the bone at the base of the defect as the tooth moves occlusally.[10,11] Kokich[12] has demonstrated the relationship between tooth position and gingival esthetics, delineating when tooth movement or periodontal surgery is most appropriate to improve anterior dental esthetics.

Furthermore, previously published works have been directed mainly to the gingival response of orthodontic tooth movement associated with retraction mechanics.[13,14] The effect is typically seen when a maxillary canine is retracted. Clinically, the gingiva on the mesial aspect of the tooth appears to be left behind, as the tooth moves away from it. Thus the epithelial attachment peels off the tooth surface and a small red triangular patch appears adjacent to the mesial aspect on the surface of the tooth. The extent to which the red patch appears varies with each person.

In the past, less emphasis has been placed on the

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Table I. Measurements of pocket depth in relation to coronal-apical measurement of "red patch"

Patient	*A* Initial facial depth of periodontal defect (mm)*	*B* Posttreatment depth of periodontal defect (mm)*	*A*-B* total reduction in depth of periodontal defect (mm)*	*Coronal-apical measurement of "Red patch" (mm)†*
A	9.2	3.6	5.6	4.7
B	8.7	3.5	5.2	4.4
C	8.0	2.6	5.4	4.7
D	8.9	3.8	5.1	4.6
E	9.0	3.7	5.3	4.1

*A** Measurement made from center of clinical gingival margin facially to the depth of pocket (Before forced eruption).
*B** Measurement at completion of forced eruption.
†Coronal-apical measurement of "red patch."
Clinical measurement in the reported series of periodontally compromised anteriors (maxillary central incisors) established that tissue pocket depth can be reduced by an average 5.32 mm in forced eruption cases with infrabony defects whose mean depth was 8.76 mm.

anatomic changes occurring with the soft tissue when a periodontally compromised tooth is moved coronally by orthodontic means. The purpose of this article is to describe in greater detail the clinical alterations in the soft tissue architecture of the periodontium demonstrated during eruptive tooth movement of periodontally compromised teeth.

MATERIAL AND METHODS

Treatment Protocol

The main source of material for this study has been provided by the observation of patients who were undergoing orthodontic extrusion, or forced eruption for implant site development. However, because the changes may be seen with photographic evidence, five patients were selected for a more detailed study. In each case, the orthodontic treatment initially involved orthodontic extrusion of the maxillary central incisors by fixed appliances.

Orthodontic treatment was initiated in the maxillary arch with bonding of 0.022-inch pretorqued and preangulated edgewise brackets from maxillary right to maxillary left canine and an 0.0175 wildcat arch wire placed. The brackets on the maxillary central incisors were located further apically at the location of the cementoenamel junction, to provide an extrusive component.

The patients were seen every 2 weeks to reduce the incisal surface of tooth being extruded, control inflammation, and monitor progress. The probing depth was measured at the midfacial line of the tooth. The diameter of the periodontal probe was 0.7 mm and applied with a force of approximately 20 gm.

After 16 weeks of orthodontic extrusion accompanied with incisal equilibration, the central incisor brackets were rebonded further apically on the root surface. The remaining brackets maintained their original positions and an 0.016 stainless steel arch wire was placed. Additional extrusion was accomplished with simultaneous occlusal reduction. Usually, root canal therapy is performed on these significantly extruded and incisally reduced teeth.

During the course of orthodontic treatment, the patients were placed on 0.12% chlorhexidine gluconate mouthrinse, Peridex (Proctor and Gamble), and strict oral hygiene maintenance that included scaling and polishing every 6 weeks.

OBSERVATIONS AND RESULTS

It is possible by close observation of the treatment in association with biweekly visits of the patient, to describe four stages of tooth movement in relation to the gingiva. It is proposed to describe these stages in general and then to comment on the individual variations. It should be mentioned, however, that the changes to be described can be seen only when a periodontally compromised tooth is orthodontically extruded.

Data recorded at the beginning of treatment (A*) are presented with the measurement made from the facial center of the clinical gingival margin to the depth of the pocket depth before forced eruption and the measurement recorded at the completion of forced eruption (B*). The significance of the difference (A* – B*) that occurred and its relation to the everted sulcular epithelium (Table I).

Stage I. Before Forced Eruption

The gingiva were normally pale pink in color and the papilla in the tooth embrasures were blunted. The labial aspect of the papilla lacked the usual gingival contour, mainly because of the compromised periodontal condition of the maxillary anterior teeth (Fig. 1, *A*).

A maxillary anterior periapical radiograph indicated that both maxillary central incisors had verti-

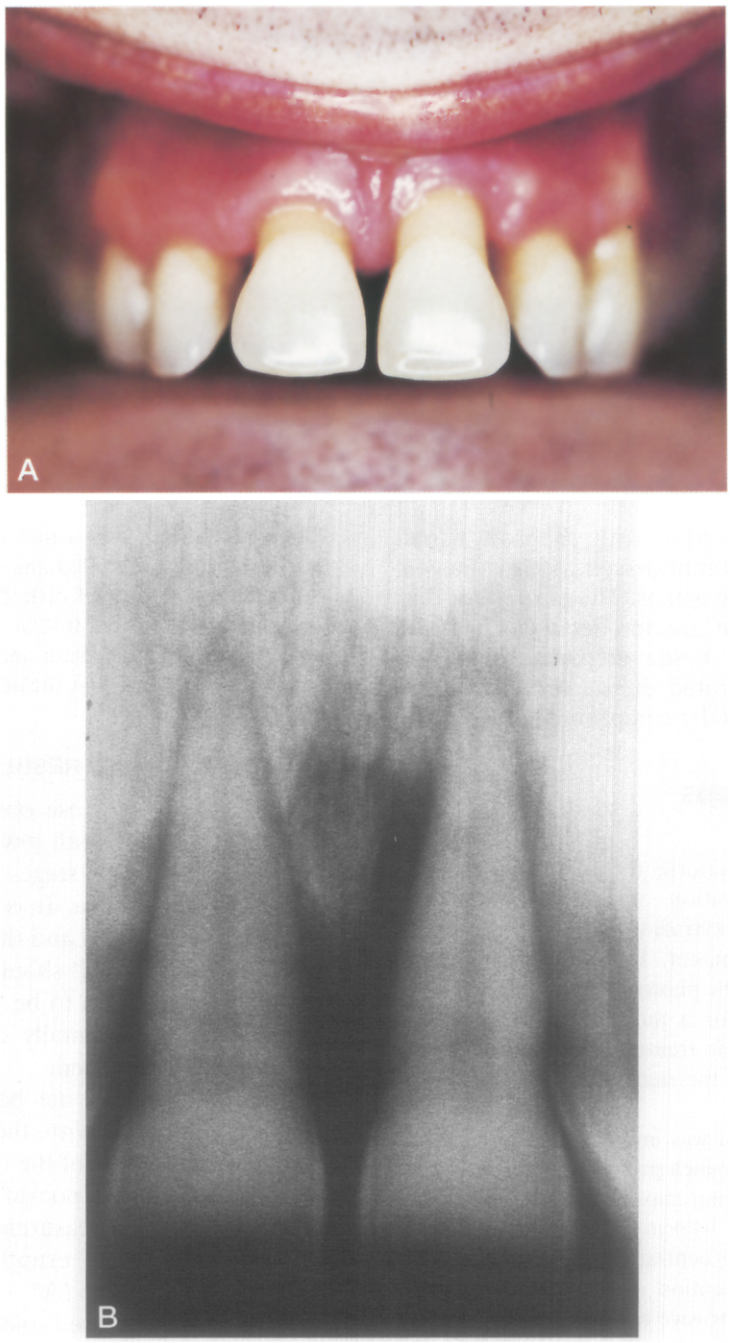

Fig. 1. A, Pretreatment frontal view of maxillary right and left central incisors. Labial aspect of papilla lacked usual gingival contour and exhibited 4 to 6 mm of gingival recession. **B,** Vertical alveolar defects and horizontal bone loss of greater than half root length along the mesial and distal surfaces of maxillary central incisors (periapical radiograph).

cal alveolar defects and horizontal bone loss of greater than half the root length along the mesial and distal surfaces (Fig. 1, *B*).

It is imperative that periodontal inflammation be brought under control before orthodontic forced eruption. However, as shown in the literature by Vanarsdall and Van Venrooy,[15] even in the presence of inflammation, orthodontic extrusion affects supporting tissues in a way that is unique from other types of tooth movement.

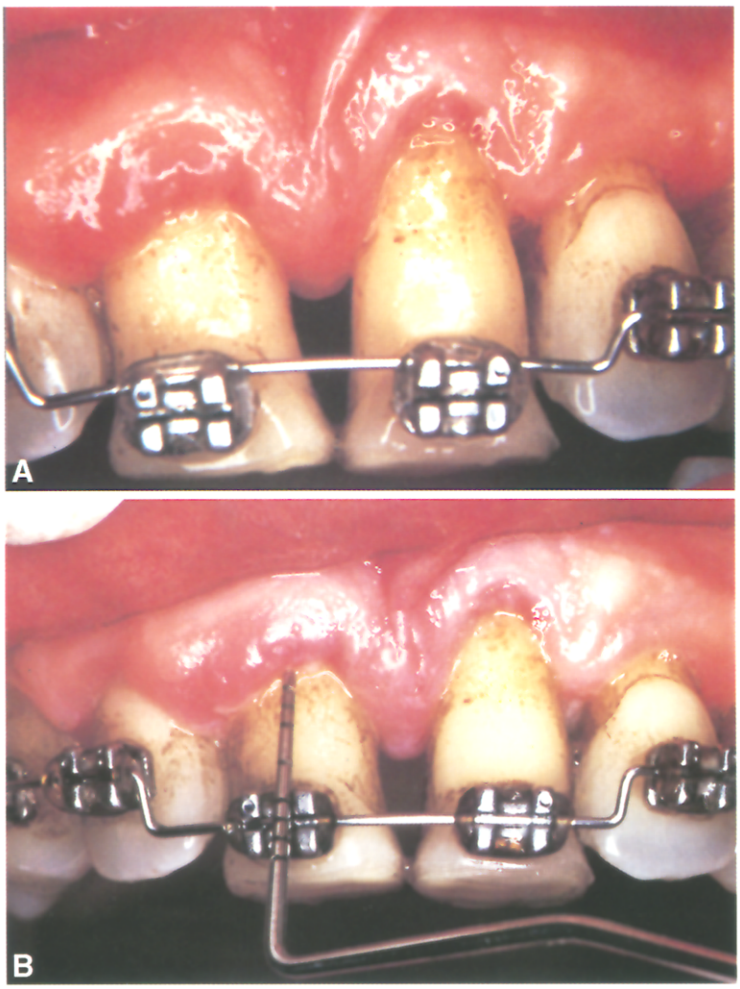

Fig. 2. A, Frontal view with 0.018-inch stainless steel arch wire placed with step-down bends; mild chlorhexidine staining; initial redness at gingival margin of maxillary right central incisor; incisal equilibration completed. **B,** Periodontal probe pointing to line demarcating typical triangular boundary of initially everted sulcular epithelium.

Initial pocket depth measurement. Initial measurement from the facial center of the clinical gingival margin to the depth of the periodontally compromised pocket before forced eruption. The mean starting pocket depth was 8.76 mm, with individual variation from 8.0 to 9.2 mm.

Stage II. Gingival Collar Redness

During the orthodontic extrusion of the maxillary central incisors, a redness on the facial aspect of the gingival margin is seen. The teeth that are erupted in the presence of soft tissue pocket depth appear to move coronally for a considerable distance before the gingival margin follows. The lapse in time associated with the appearance of the redness is in direct relation to the pocket depth on the maxillary right and left central incisors. Consequently, the greater pocket depth exhibits the red collar later in the orthodontic extrusion procedure. (Fig. 2, *A* and *B*).

Stage III. The Red Patch

As continued orthodontic extrusion occurs, there is a potential for reduction in pocket depth and a change in the architecture of the soft tissue component. Usually, root canal therapy is performed on these significantly extruded and incisally reduced teeth. Therefore orthodontic extrusive remodeling whereby a hopeless tooth was extruded over the course of several weeks and then stabilized produces this appearance. Concurrently, the gingiva appears to be induced to peel away from the tooth,

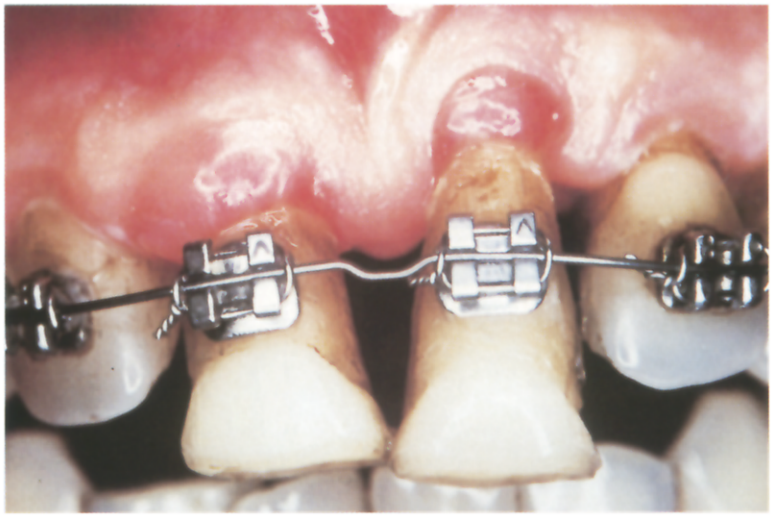

Fig. 3. Frontal view showing keratinized gingiva of maxillary right central incisor and progressive redness of gingival margin of maxillary left central incisor. Bracket placement on cementum of maxillary right and left central incisor. Gingiva is induced to peel away from tooth, pocket depth is reduced and immature appearing tissue, "red patch," appears coronal to original gingival margin.

the pocket depth is reduced and an immature appearing tissue, "the red patch," appears coronal to the original gingival margin. It is recognized that the increase in the soft tissue component is directly due to the eversion of the pocket lining (Fig. 3).

Final pocket depth measurement. The final measurement recorded from the facial center of the clinical gingival margin to the depth of the orthodontically extruded tooth. The mean final pocket depth was 3.44 mm, with a range from 2.6 to 3.8 mm.

Reduction of pocket depth. As a result of orthodontic extrusion, or forced eruption, the mean pocket depth reduced was 5.32 mm, with an individual variation of 5.1 to 5.6 mm.

Stage IV. Keratinization

The patch is no longer sunk beneath the general level of the gingiva. This epithelium lacks a keratinous covering of the oral epithelium and is only a few cells thick. It has been described as "degenerate," whereas the oral epithelium is a proliferative and maturing tissue. It would be anticipated that the sulcular epithelium, when peeled off the tooth surface, would be bright red, because it is so thin and that, for same reason, would lie below the level of the gingiva.

This nonkeratinized tissue remains erythematous for 28 to 42 days until keratinization occurs, and it assumes the keratinized appearance characteristic of the normal surrounding gingiva (Fig. 4, *A*).

At this time, it is recognized radiographically that the maxillary central incisors are no longer within the confines of the alveolus, such that, the remaining extrusive component generates an actual orthodontic extraction (Fig. 4, *B*).

Coronal-apical distance of "red patch." Clinical appearance of red patch at the completion of orthodontic extrusion exhibited a mean of 4.5 mm, with a individual variation of 4.1 to 4.7 mm.

Observations on several hundred patients show wide variations in the clinical appearance of the gingiva in relation to orthodontic extrusion. These variations appear to be modifications of the basic changes described in Stages I to IV, rather than a real difference (Fig. 5).

The changes described are most clearly seen in a patient in whom the treatment plan necessitates the orthodontic extrusion to the point of extraction of a maxillary anterior incisor. In this way, the gingiva appears induced to peel away from the tooth, revealing the "red patch." If tooth movement is slow, this appearance of the red patch is modified. The patch is no longer sunk beneath the general level of the gingiva and is the same color. It can usually be distinguished, however, by the line demarcating its typical triangular boundary (Fig. 6).

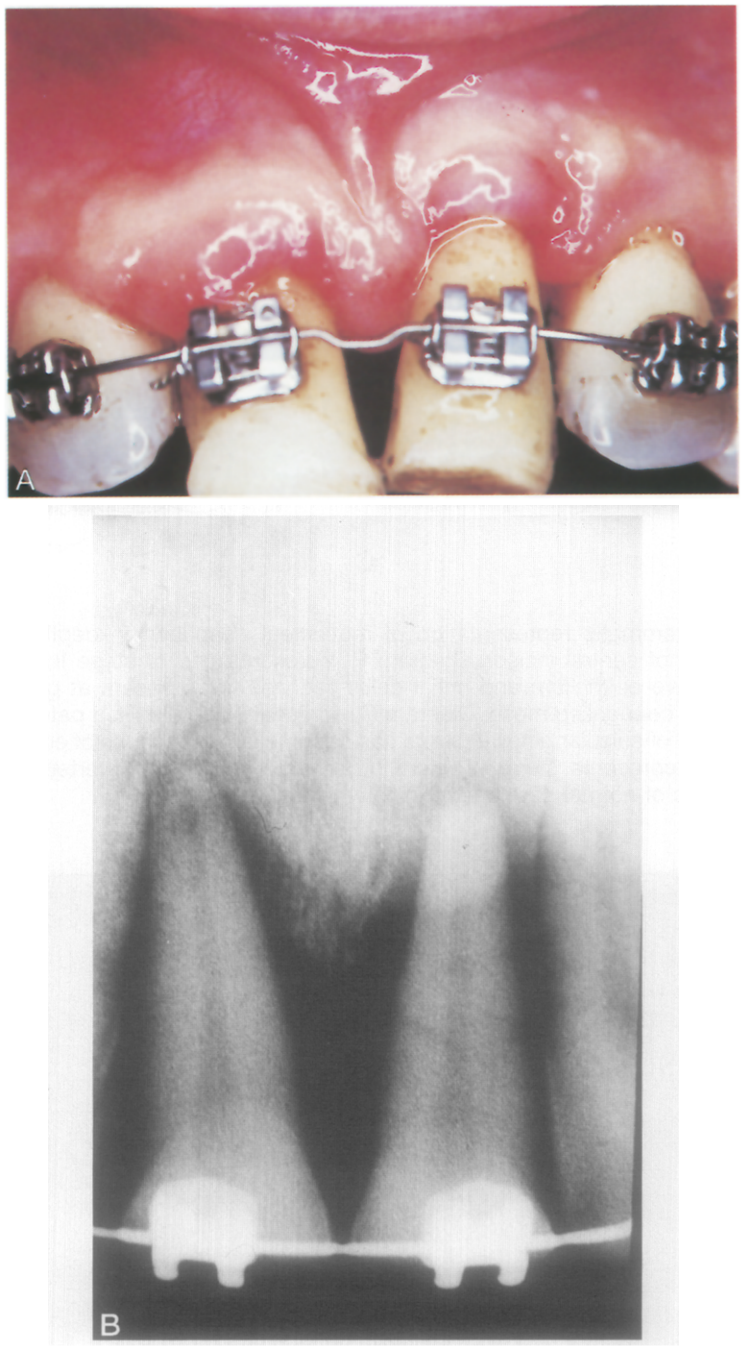

Fig. 4. A, Keratinized sulcular epithelium exhibited on maxillary right and left central incisors. Nonkeratinized tissue remains erythematous for 28 to 42 days until keratinization occurs and it assumes keratinized appearance, characteristic of normal surrounding gingiva. **B,** Radiographically recognized that maxillary central incisors are no longer within confines of alveolus and vertical bone improvement, relative to lateral incisors (periapical radiograph).

DISCUSSION

The orifice of the gingival sulcus is bound by the tooth surface on one side and sulcular epithelium on the other, the bottom of the sulcus is frequently surrounded by epithelium on all sides. This is due to the persistence of junctional epithelial cells on the tooth surface coronal to the sulcus bottom. The soft tissue wall of the gingival sulcus is lined coronally with nonkeratinizing epithelium, the sulcular epithelium. The apical part of the soft tissue wall and the

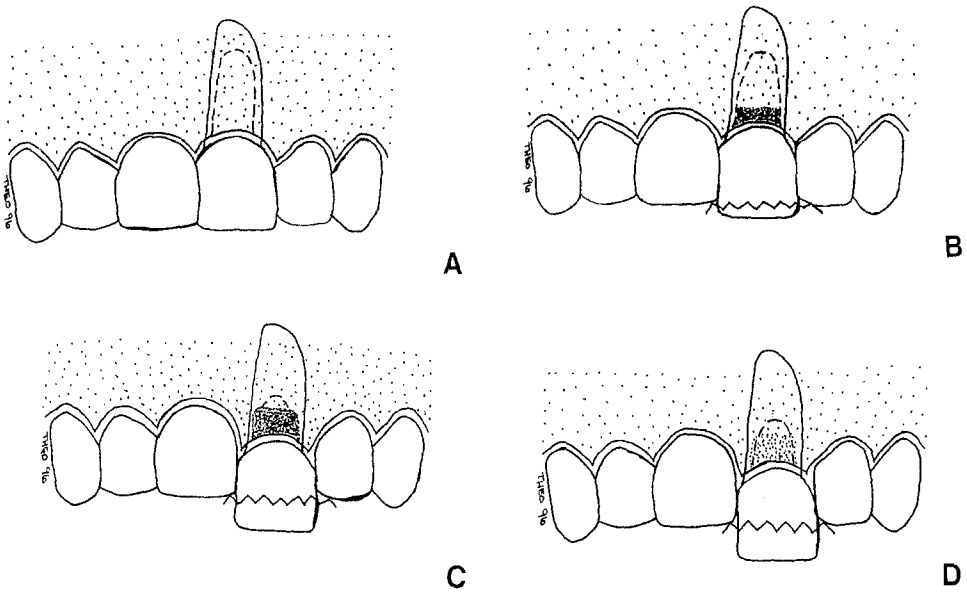

Fig. 5. Diagrammatic representation of movement described in depth represented on labial aspect of central incisor. Diagram B represents end of stage II, where tooth has begun to leave epithelium and initial collar redness first appears at gingival margin as illustrated by condensed mesh. Diagram C represents stage III. Red patch is well defined. Facial aspect of sulcular gingival tissue has become larger because of epithelium everting. Diagram D represents stage IV. Keratinization of red patch. Everted tissue assumes characteristic of normal surrounding gingiva.

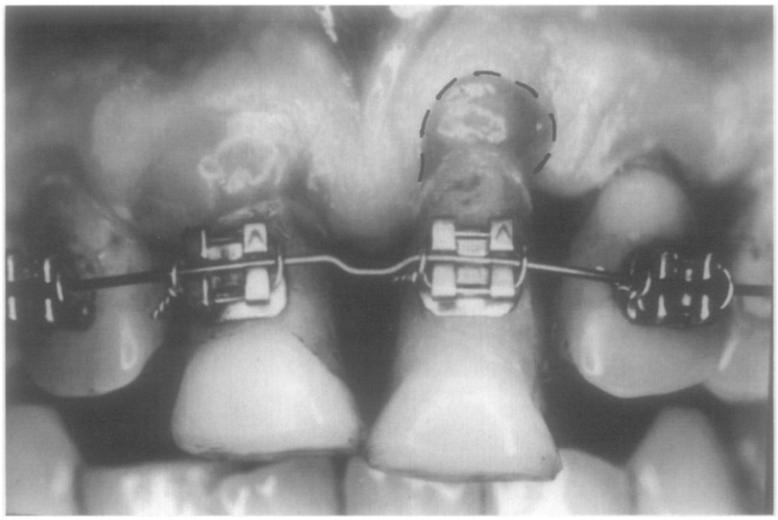

Fig. 6. Frontal view of maxillary incisors exhibiting line demarcating typical triangular boundary of everted sulcular epithelium before keratinization.

bottom of the sulcus is maintained by the coronal surface of the junctional epithelium. The junctional epithelium consists of a thin layer of epithelium that joins the gingival connective tissue to the tooth surface by the basal lamina and hemidesmosomes. This epithelium extends from the bottom of the gingival sulcus to the apical border of the enamel under normal conditions. The sulcular epithelium is continuous with the junctional epithelium that abuts on the tooth surface. Consequently, the junctional

epithelium, in a broad sense, provides a seal at the base of the sulcus against the penetration of chemical and bacterial substances (Fig. 7).

The gingival epithelium that covers the free and attached gingiva is normally of the keratinizing type. The epithelium of the gingival sulcus, as with that which appears because of an eversion of the pocket lining, is not keratinized. The attachment or junctional epithelium, like the sulcular epithelium, is thin and nonkeratinized. Histochemically, unlike gingival epithelium and sulcular epithelium, normal junctional epithelium does not show any tendency toward keratinization.

To understand more fully the attachment of the gingiva to the tooth, we must appreciate the mechanism of the changing position of the gingiva to the tooth during eruption and during its recession and pocket formation.

Initially, the surface of the enamel, after its formation and calcification are completed, is covered by the reduced enamel epithelium. As an erupting tooth nears the oral epithelium and penetrates the tissue, the reduced enamel epithelium surrounding the enamel and the oral epithelium fuse at the incisal edge of the tooth (Fig. 8). When the tip of the tooth emerges from the membrane, the reduced enamel epithelium continues to be attached to almost the entire enamel surface coronal to the junctional epithelium (Fig. 9, *A* and *B*).

As the epithelium separates from the tooth surface during pocket formation or in recession, the apical end of the attachment epithelium (reduced enamel epithelium) gradually moves onto the cementum. The bottom of the sulcus, which was first located on enamel, moves progressively toward the cementoenamel junction and apically beyond that point. Thus the attachment may move from the enamel to the cementum.

In periodontal disease, the deepening of the gingival sulcus occurs as a result of migration of the junctional epithelium apically and its separation from the tooth surface (Fig. 10). Pocket formation initially starts as an inflammatory change in the connective tissue wall of the gingival sulcus caused by bacterial plaque. The cellular and fluid inflammatory exudate causes degeneration of the surrounding tissue, including the gingival fibers. In association with the inflammation, as the pocket depth is increased, the coronal portion of the junctional epithelium detaches from the tooth surface so that the sulcus bottom shifts apically and the oral sulcular epithelium occupies a gradually increasing portion of the sulcular lining.

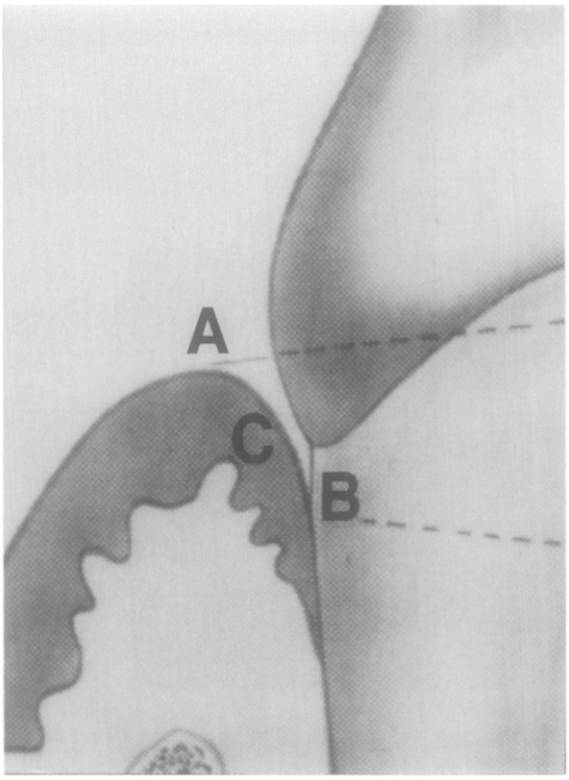

Fig. 7. Dentogingival junction is composed of gingival sulcus, which is 0.5 mm in depth between tooth and healthy gingiva (*C*); this lined by unkeratinized epithelium (*A*). At base of gingival sulcus, there is junctional epithelium (*B*).

When a periodontally compromised tooth is moved during orthodontic extrusion, changes may be observed in the surrounding gingiva. The teeth that are erupted in the presence of soft tissue pocket depth appear to move coronally for a considerable distance before the gingival margin follows. Concurrently, the pocket depth is reduced and an immature appearing tissue, "the red patch," appears coronal to the original gingival margin. It is recognized that the increase in the soft tissue component is directly due to an eversion of the pocket lining. Specifically, the appearance of the "red patch," that is sulcular epithelium, has peeled off the tooth surface directly, because of an eversion of the pocket lining. Initially, this epithelium lacks a keratinous covering of the oral epithelium and is only a few cells thick. It is anticipated that the sulcular epithelium, when peeled off the tooth surface, would be bright red, because it is so thin and, for the same reason, would lie below the general level of the gingiva. If tooth

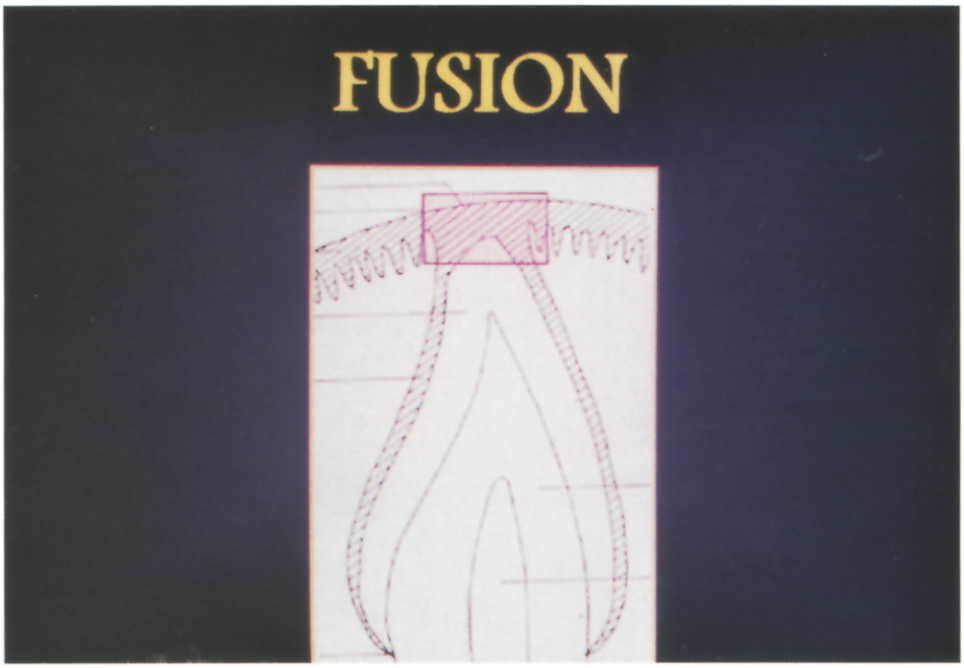

Fig. 8. Fusion: Reduced enamel epithelium fuses with oral epithelium.

movement is slow, however, the proliferative nature of oral epithelium is such that the newly introduced sulcular epithelium characteristically displays keratohyaline granules that are associated with keratin formation, and the color and texture difference of the red patch.

The nonkeratinized tissue remains erythematous for 28 to 42 days until keratinization occurs and it assumes the appearance of normal gingiva.[16] Keratinization results from redifferentiation of implanted prickle cells to basal cells with an inward to outward migratory cell pattern. The keratinizing event appears to be genetically determined by epithelial quality and modified by local and somatic environment.

The mechanotherapy used resulted in a light continuous force, producing a precisely regulated crown and root movement, which altered the position of the crown in a vertical plane. Clinical evidence presented suggests that the principle fibers of the gingiva attached to the tooth are displaced in the direction of tooth movement. The histopathology of the periodontal pocket suggests that the diseased gingival pocket wall is produced by dissolution of the connective tissue fibers formally attached to the root surface.

As previously mentioned, there is no contraindication to treatment of adult patients who have periodontal disease, as long as the acute inflammatory process has been brought under control. Consequently, it is imperative that all pathoses be under control before all orthodontic treatment. The periodontal situation must receive major attention in planning and executing orthodontic treatment for adults. It is of great clinical benefit if a chemical agent could be used during the active phase of orthodontic treatment to reduce the bacterial plaque accumulation, thereby improving the gingival condition and possibly reducing an acute inflammatory process. As a result, the patients were placed on 0.12% chlorhexidine gluconate (CH) mouthrinse, Peridex, during the course of orthodontic treatment.

The staining seen with the use of chlorhexidine gluconate is significant to a mild to moderate degree, and it was removed with a dental prophylaxis. The taste may be bitter to some, but the advantages of this agent far outweigh the disadvantages and make it an acceptable chemical agent for reducing gingival inflammation and plaque accumulation.[17,18]

In summation, the reduction in soft tissue pocket

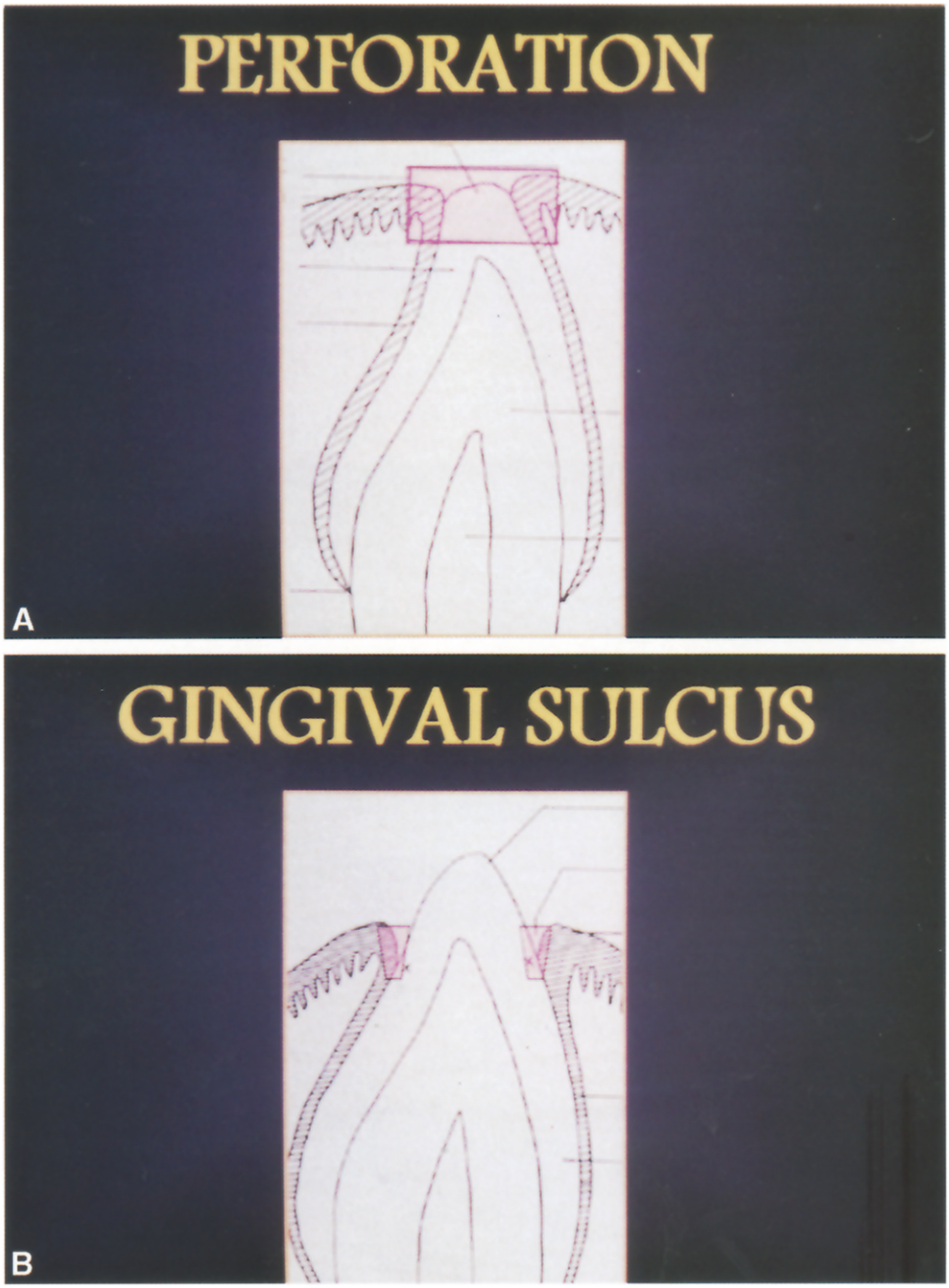

Fig. 9. A, Penetration: Tooth emerges through perforation in fused epithelium. **B,** Gingival sulcus is early stage of tooth eruption.

depth occurred as a result of coronal displacement of the attached fiber bundles apical to the epithelial attachment, causing a substantial portion of the pocket wall to become everted. The direct relationship was identified with a mean pocket depth reduction of 5.32 mm accompanied with a coronal-apical distance of the "red patch" at 4.5 mm. This directly explains the leveling of the soft tissue defect and the coronal positioning of the new gingival margin. The enhanced anatomic topography and increased volume of soft tissue develop the emergence profile for the restoration. Subsequently, the osseous support allows for improvement of the three-dimensional configuration of the soft tissue.

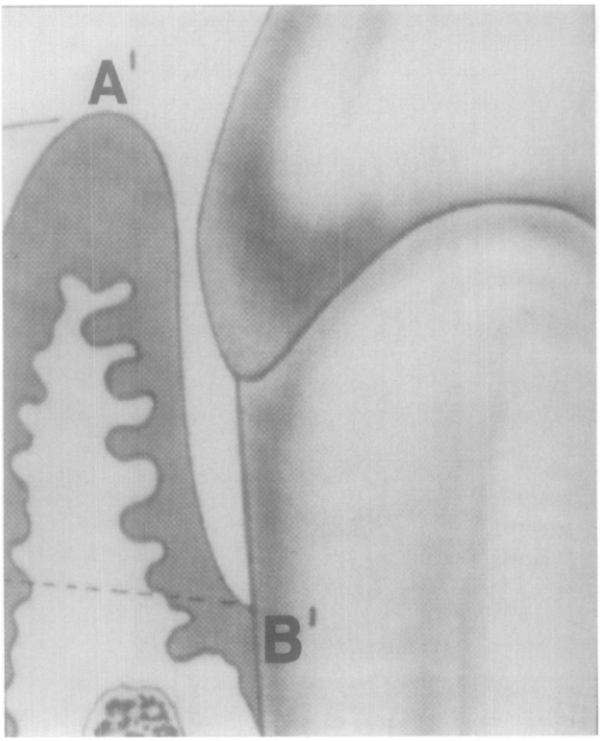

Fig. 10. Representation of pocket formation: Normal gingival sulcus to periodontal pocket as result of migration of junctional epithelium apically and its separation from tooth surface.

CONCLUSION

Forced eruption and implant site development is a nonsurgical technique for improving the three-dimensional topography of the implant recipient site before extraction. Therefore the concept of a periodontally compromised tooth moving coronally by orthodontic means, and the clinical alterations in the soft tissue architecture reveals a red patch appearance in the wake of orthodontic extrusion.

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