Determinants controlling iatrogenic external root resorption and repair during and after palatal expansion

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With the rejection[1,2] of the old prevailing concept[3,4] that systemic factors, such as endocrine imbalances (hypothyroidism) and metabolic disorders of bone (osteoporosis) are solely responsible for orthodontically induced external root resorption (ERR), extensive research has elucidated the mechanism of iatrogenic ERR resorption. A brief review of current research shows the complexity of the iatrogenic reaction.

Reitan[5] has shown that ERR is weakly correlated to force magnitude (25g to 240g) and closely related to the type of tooth movement, specifically intrusion and tipping. Subsequent studies[6-9] have characterized ERR as a time-related response. Two time-related features have been observed: ERR is initiated 14 to 20 days after force onset,[6,8,10] and the process of ERR continues even during extended retention periods of up to 1 year.[7,9,11]

On the cellular level, Andreasen[12] studied ERR in replanted teeth and concluded that the resorption cycle is hastened at ankylosis sites. Thus, ERR will begin when the integrity of the cell line populating the periodontal ligament-cementum interface is interrupted. The yielding of the periodontal ligament is a two-step process: an initial morphometric change — compression — followed by biochemical changes. The histochemical changes seen in the periodontal ligament include: fiber coalescence; hyalinization;[13] degradation of connective tissue matrices by both an extracellular pathway (metaloendoproteinases e.g. collagenase) and an intracellular phagocytic pathway (lysosomal enzymes);[14] and demineralization of Sharpey's fibers.[15] An odontoclastic process follows these changes. The ERR sequence of reduced periodontal ligament width, followed by ankylosis and then scavenging of root hard tissue was also confirmed in the study

Abstract

The mechanisms controlling iatrogenic external root resorption (ERR) and repair were studied on 8 *Macaca fascicularis* monkeys. The animals were divided into short-term and long-term groups, and were treated with jackscrew, magnetic and sham palatal expansion appliances. Scanning electron microscopy morphometric analysis found major evidence of ERR in the tooth-borne jackscrew appliance, in the long-term group, in the maxillary premolars, on the buccal and furcation root surfaces, on the mesiobuccal root, and in the apical zone. Correspondingly, the ERR mechanism is controlled by the impulse ($F \cdot \Delta t$) and the critical barrier of the periodontal ligament as primary determinants and by the environmental density as a secondary determinant. ERR is initially regulated by the force component of the impulse and, with increased duration, by the time component of the impulse. The impairment/repair dynamics were found to be regulated by three principles: ERR level of irreversibility, delayed resorption response and jiggling.

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Key Words

Root resorption • Palatal expansion • Magnets

of Wesselink et al.[16] The systemic administration to rats of 1-hydroxyethylidene-1, 1-bisphosphonate (HEBP —a drug which prevents tissue mineralization) caused a reduction in the periodontal ligament width from 70 μm to 16 μm, presumably by suppressing fibroblast activity.

The environment surrounding the dental apparatus also merits consideration. It has been found that ERR prevalence and magnitude are directly correlated with the mineralized content of the environment surrounding the mobile tooth. The effect of increased mineralized content is demonstrated by the increase in frequency (2x) and severity (8x) of apical root resorption of upper and lower incisors during stage II of Begg treatment, due to approximation of the apices to the labial compact cortical bone.[17] Likewise, resorption of the distobuccal root of the upper first molar occurred when the distalization movement impinged upon the eruption of the upper second molar.[18] The effect of decreased mineralized content was demonstrated by Follin et al.[19] They found that when a dog's lower premolar was bodily mesialized into an extraction space, its mesial root experienced three-fold less ERR than its distal root. The same reduction in ERR (although temporary) was demonstrated when tooth movement in lactating rats fed a calcium-deficient diet was studied.[20]

The objectives of the current study were: (1) To study the mechanism controlling iatrogenic ERR by defining ERR determinants and analyzing their interaction; and (2) To investigate the mechanism regulating the impairment/repair dynamics of ERR.

Materials and methods

Eight *Macaca fascicularis* monkeys aged 3 to 3½ years (comparable to a human age of 14 to 18 years) were divided into short-term and long-term groups. The four animals in each group received the following palatal expansion appliances (for details see Vardimon et al.[21,22]).

IS Indirect Screw — a tooth-borne acrylic plate containing an expansion screw. This appliance, which is bonded to the teeth bilaterally from the canine to the first molar, transmitted high forces ($\bar{F} = 2035$ g) indirectly to the midpalatal suture via abutment teeth.

IM Indirect Magnets — the active unit consisted of two rare earth permanent magnets, $SmCo_5$ (Incor 21 — IG Technologies, Inc., Valparaiso, Ind.) in the short-term group and $Nd_2Fe_{14}B$ (Crumax 35 — Colt Industries Crucible Magnetic Division, Elisabethtown, Ky.) in the long-term group. The magnets were positioned in a repelling configuration. The appliance was bonded to the abutment teeth (3 to 6) and transmitted low forces ($\bar{F}_{Sm} = 258$ g, $\bar{F}_{Nd} = 360$ g) indirectly to the mid-palatal suture.

DM Direct Magnets — the magnetic unit was exactly the same as the IM appliance, however, it applied low force directly to the hard palate by means of four endosseous pins. The plate was fixated to the palatal shelves instead of being bonded to the teeth via occlusal cover.

IC Indirect Control, sham appliance — the control animal received a non-active upper indirect sham acrylic appliance bonded to the upper dental arch with occlusal cover.

The short-term group was sacrificed upon completion of the active palatal expansion (PE) treatment phase, when a full posterior crossbite was achieved (**IS** = 33 days, **IM** = 135 days, **DM** = 95 days). In the long-term group the active treatment phase (**IS** = 28 days, **IM** = 216 days, **DM** = 140 days) was followed by four months of retention with the PE appliances remaining in an inactive form, plus two months of relapse with the PE appliances removed from the mouth.

Upon sacrifice, one maxillary quadrant was processed for histological evaluation. The results will be reported in a separate article. The other upper quadrant was assayed with scanning electron microscopy. The pulp chambers of six maxillary teeth (2 to 7) were exposed and the pulps gently removed with a cotton pellet. The remaining non-calcified tissues (gingiva, periodontal ligament and remaining pulp canal contents) were removed by treating the sample with Biz bleach detergent (Procter & Gamble, Cincinnati, Ohio) at 80°C for 24 hours with two to three water rinse changes. An iatrogenic resorption artifact was avoided by removing the distal interdental septum and the palatal alveolar wall prior to releasing each tooth from its alveolar socket. This tooth-by-tooth dissection was conducted in a posteroanterior direction, starting with the third molar. Bone detachment was enhanced by the early deproteinized treatment with the Biz detergent. Residual periodontal ligament and pulp canal tissues were degraded by treating the teeth for 20 to 40 minutes in 5% sodium hypochlorite, followed by a thorough rinsing in distilled water. The specimens were then dehydrated in an ascending graded series of ethyl alcohols (20%, 50%, 75%, 95%, 100% and 100%) for 30 minutes in each concentration. The ethanol was then substituted by ascending concentrations of amyl acetate (50%, 75%, 100% and 100%). The specimens were critical-point dried

in CO_2 using Bomar SPC-1500 dryer (Bomar Co., Tacoma, Wash.). To provide standardized spatial orientation of the specimens during SEM morphometric examinations, each aluminum stub bearing a tooth at the occlusal table was engraved around its circumference at 90° intervals. The tooth was coated with gold by sputter coating (Technics, Alexandria, Va.) and examined in Cambridge Steroscan 250 MK 2 SEM (Cambridge Instruments Inc., Monsey, NY), using an accelerating voltage of 20 kV.

Each of the four root surfaces was divided into three root zones (apical, mid and cervical). Each root zone was scanned by means of SEM at magnifications of 20x, 60x and 250x, and scored for ERR magnitude. ERR magnitude was assigned as: 0 — no ERR; 1 — cemental and superficial dentinal ERR; 2 — augmented dentinal ERR, where the total unresorbed surface area exceeded the total resorbed surface area; and 3 — severe dentinal scavenging, where the total resorbed area was greater than the unresorbed area. All teeth were examined twice by the same examiner. The raw ERR scores were converted into an ERR index using the formula:

$$\sum_{i=1}^{n} \frac{100 \cdot X_i}{3n}$$

where X_i is a score for a given zone and n is the number of scored zones. The ERR index strictly refers to a loss of root surface substance and not to a loss in total tooth structure. Means and standard deviations were calculated and compared for all teeth, individual teeth, single roots (mesiobuccal, distobuccal, palatal) of multirooted teeth (4 to 7, macaque premolars are three-rooted teeth), and each root surface (buccal, mesial, distal, palatal, furcation) by root zone (apical, mid, cervical). These comparisons were also evaluated by type of PE appliance (**IS, IM, DM, IC**) and for treatment duration (short-, long-term). Due to sample size limitations, no attempt was made to determine significance levels; thus, the results should be considered, at best, a conceivable trend.

Results

Teeth

Multirooted teeth were substantially more susceptible to ERR than single rooted teeth (Figures 1 and 2A). The mean total ERR indices were, in descending order: $\overline{X}_4 = 22.5 \pm 18.9$; $\overline{X}_5 = 22.1 \pm 12.4$; $\overline{X}_6 = 21.1 \pm 11.3$; $\overline{X}_7 = 14.4 \pm 4.0$; $\overline{X}_3 = 10.6 \pm 3.8$; and $\overline{X}_2 = 5.1 \pm 3.8$. The three multirooted teeth demonstrated ERR effects of essentially equal magnitude due to a common tooth/appliance attachment in all indirect PE appliances. However, the canine unexpectedly exhibited a characteristic resistance to ERR assault, despite the fact that the tooth was linked to all indirect PE appliances. The second molar, which was not bonded to any PE appliance, displayed an ERR index greater than the canine (Figure 1).

Surfaces

The buccal root surface (Figures 1A and 3A; $\overline{X}_B = 26.2 \pm 19.2$) and the furcation surface (Figures 1E and 4; $\overline{X}_F = 20.2 \pm 18.3$) showed distinguishable ERR destruction. The other three surfaces were considerably less affected and declined in the following order: palatal surface (Figure 1B; $\overline{X}_P = 15.2 \pm 18.2$), mesial surface (Figure 1C; $\overline{X}_M = 11.0 \pm 13.1$) and distal surface (Figure 1D; $\overline{X}_D = 10.7 \pm 11.6$).

Single roots of multirooted teeth

The mesiobuccal (MB) root always exhibited the highest ERR assault followed by the distobuccal (DB) and the palatal (P) roots for the buccal surface (Figures 2A and 5A) and the palatal and distobuccal roots for the palatal surface (Figure 5B). Correspondingly, the ERR indices for the buccal surfaces were: $\overline{X}_{MB} = 41.6 \pm 28.3$; $\overline{X}_{DB} = 34.5 \pm 26.0$; and $\overline{X}_P = 29.3 \pm 20.2$. The ERR indices for the palatal surfaces were: $\overline{X}_{MB} = 21.6 \pm 23.7$; $\overline{X}_P = 15.3 \pm 21.0$; and $\overline{X}_{DB} = 13.6 \pm 15.0$.

Root zones

The apical root zone was the most disturbed vertical root region followed by the midroot and cervical root zones. ERR indices for the buccal side of the vertical zones (Figure 6A) were: $\overline{X}_{APICAL} = 36.9 \pm 22.8$; $\overline{X}_{MIDROOT} = 30.1 \pm 21.0$; and $\overline{X}_{CORONAL} = 26.9 \pm 24.3$. ERR indices for the palatal sides of the vertical zones (Figure 6B) were: $\overline{X}_{APICAL} = 22.5 \pm 23.6$; $\overline{X}_{MIDROOT} = 15.5 \pm 20.1$; and $\overline{X}_{CORONAL} = 10.5 \pm 15.2$.

Type of PE appliance

The three PE appliances differed from each other in force threshold and/or point of force application. Comparison of the indirect screw and indirect magnetic appliances demonstrated a change in ERR when the force threshold acts as the variable factor and the point of force application acts as the non-variable factor. There was only a moderate reduction in the ERR index in the direction of tooth movement (i.e. bucco-palatal) with a decline in the applied force (Figures 1A and B; 5 and 6). High forces of $\bar{F}_{IS} = 2035$ g resulted in a mean bucco-palatal ERR index of $\overline{X}_S = 35.9 \pm 23.1$ whereas low forces of $\bar{F}_{IM} = 309$ g resulted in a mean bucco-palatal ERR index of $\overline{X}_{IM} = 29.5 \pm 21.5$.

Comparison of the indirect and direct magnetic appliances showed a change in ERR when

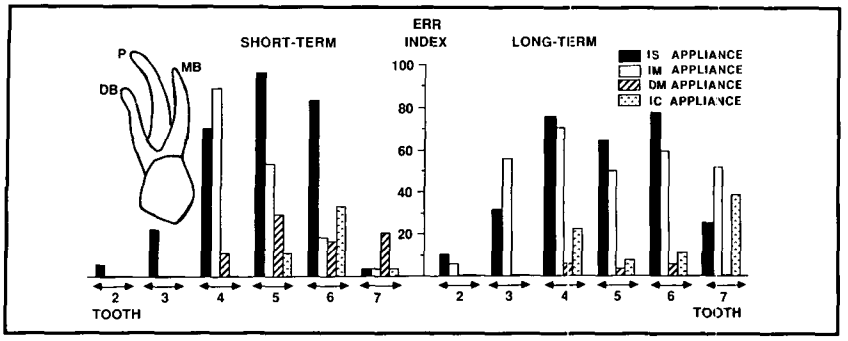
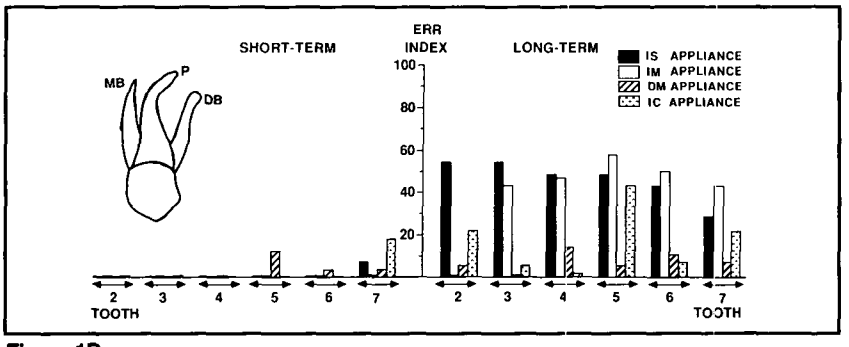


Figure 1A



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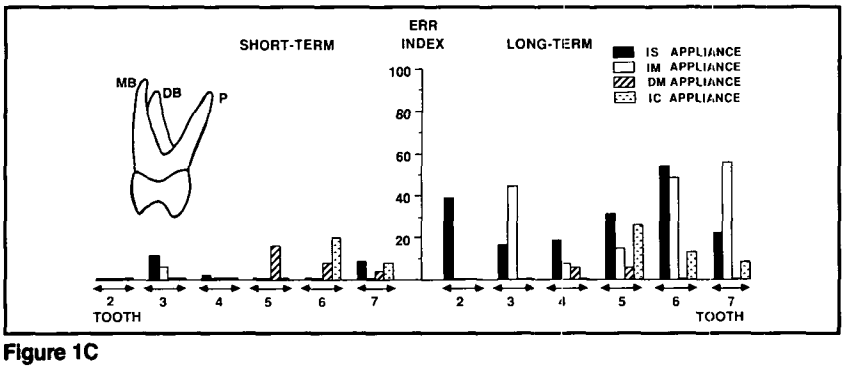


Figure 1C

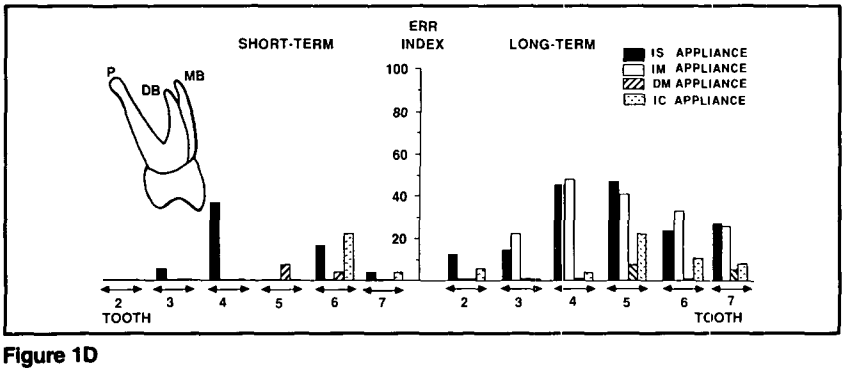
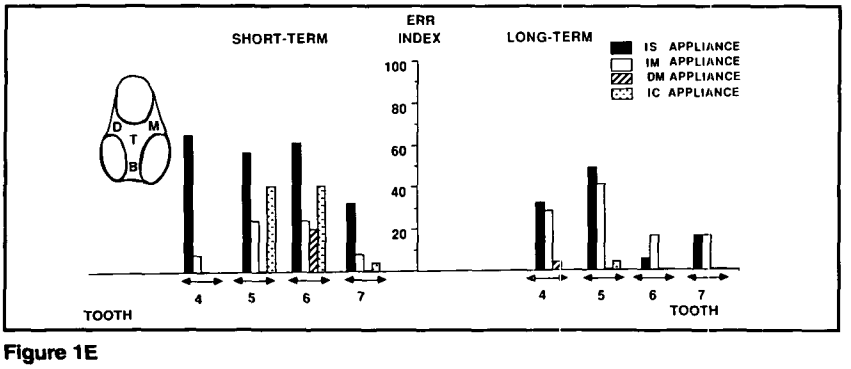


Figure 1D



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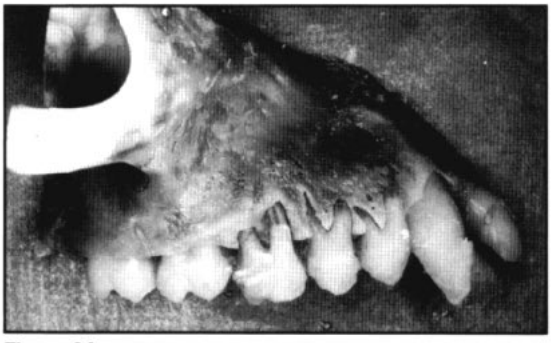

Figure 2A

Figure 2B

Figure 1A-E
External root resorption per tooth for six maxillary teeth (2-7) per palatal expansion appliance for the short- and long-term groups. A. Buccal root surface — highest affected root surface; ERR indices (IS and IM of the short-term group) virtually did not dissipate in the long-term group; multirooted teeth (4-6) were significantly more affected than the canine. B. Palatal root surface. C. Mesial root surface. D. Distal root surface. These three root surfaces demonstrated increased ERR indices in the long-term group due to jiggling. E. Furcation root surface — the second highest affected root surface due to the intrusive force component.

Figure 2A-B
Maxillary quadrant (after bleaching) of the long-term subject receiving indirect magnetic palatal expansion appliance (360 g; 216 d expansion + 120 d retention + 61 d relapse). A. Buccal view — multirooted teeth (4 to 6) linked to the appliance were highly affected by bone recession, root dehiscence, and ERR. More precisely, the mesiobuccal root, due to its anatomical proximity to the buccal cortical plate, showed the greatest ERR impairment. The canine, as a single rooted tooth linked to the appliance, was affected by severe bone recession but demonstrated a high resistance to ERR sequelae. B. Occlusal view — jiggling effect (i.e. tooth oscillating along the line of movement) was characteristic of subjects of the long-term group. The jiggling which affected the mesial, distal and palatal root surfaces was distinguished by an increase in osteoclastic activity along the corresponding alveolar bone (arrows) which presumably triggered an ERR response whenever the critical barrier (λ) was surpassed.

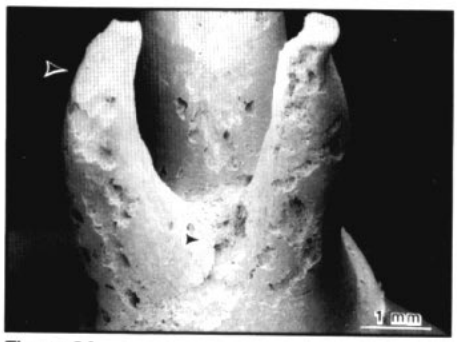

Figure 3A

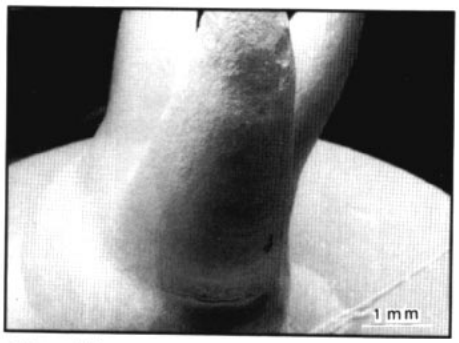

Figure 3B

Figure 3A-D
Buccal (A), palatal (B), mesial (C) and distal (D) root surfaces of the maxillary left second premolar subjected to short-term, indirect screw palatal expansion appliance (2035 g, 35 d). ERR is confined to the buccal (compression) surface. Areas along the convergence of the intrusive-translatory force vectors were most severely affected. This applies to the buccal apical zone of the mesiobuccal and distobuccal roots (particularly the mesiobuccal root, upper arrow-A) and the buccal furcation area (lower arrow-A) (magnification 18x).

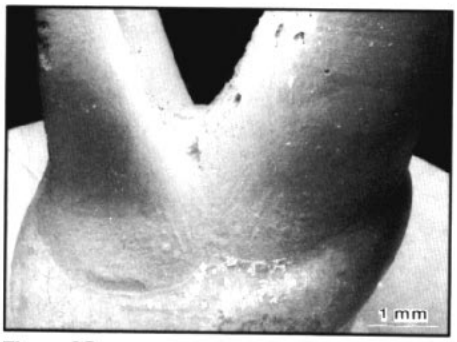

Figure 3C

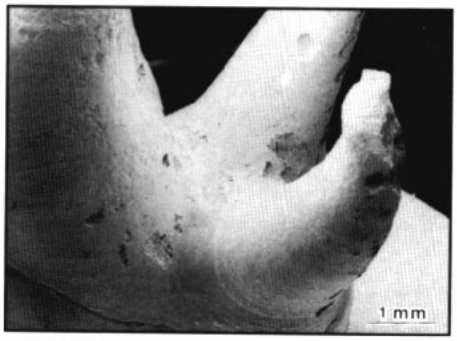

Figure 3D

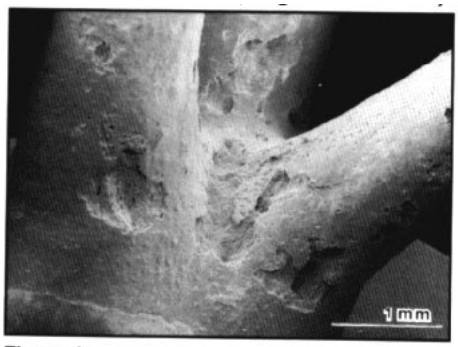

Figure 4

Figure 4
ERR propagated at the furcation surface from the buccal furcation area towards the trifurcation area. This corresponds to the applied intrusive — translatory force vector (magnification 28x).

the point of force application acts as the variable factor while the force threshold factor remains unchanged. There was a dramatic decrease in ERR index in the direction of movement (Figures 1A and B, 5 and 6) when the teeth did not serve as force transmitters. Correspondingly, an equal amount of force ($\bar{F}_{IM,DM}$ = 309 g) resulted in a mean buccopalatal ERR index of $\overline{X}_{IM}$ = 29.5 ± 21.5 for the indirect magnetic appliance and an ERR index of $\overline{X}_{DM}$ = 6.6 ± 4.8 in the direct magnetic appliance.

Short-term vs. long-term

An increase in the buccal ERR indices from the short-term group to the long-term group was recorded for both indirect appliances (Table I; Figures 1A and B, and 5; $\overline{X}_{IS,\,SHORT}$ = 46.9 ± 41.2 vs. $\overline{X}_{IS,\,LONG}$ = 47.8 ± 27.9; and $\overline{X}_{IM,\,SHORT}$ = 27.5 ± 36.4 vs. $\overline{X}_{IM,\,LONG}$ = 48.9 ± 22.4). In contrast, the direct magnetic subjects showed a recovery in the buccal ERR index from a short-term level of $\overline{X}_{DM,\,SHORT}$ = 13.0 ± 11.7 to a long-term level of $\overline{X}_{DM,\,LONG}$ = 2.5 ± 2.8 (Table I; Figures 1A and B, 5 and 6), suggesting that only low ERR impairments (cementum defects) are repairable. The fluctuation of ERR indices on the palatal surface (Table I; Figure 1B) between the short- and long-term groups demonstrates the direct relationship between the compressed and impaired root surfaces. None of the appliances showed any damage to the palatal root surface by the end of the active treatment phase ($\overline{X}_{IS,\,SHORT}$ = 1.2 ± 3.0; $\overline{X}_{IM,\,SHORT}$ = 0.0; $\overline{X}_{DM,\,SHORT}$ = 3.4 ± 5.0). However, during the relapse phase, with no appliance in place, teeth rebounded and compressed against the palatal cortical plate, resulting in a sharp increase in the ERR indices, particularly in the indirect appliances ($\overline{X}_{IS,\,LONG}$ = 47.5 ± 9.7; $\overline{X}_{IM,\,LONG}$ = 41.4 ± 21.0; $\overline{X}_{DM,\,LONG}$ = 7.4 ± 5.1).

Discussion

The preponderance of evidence in the current study suggests that under normal microenvironmental conditions (i.e. balanced endocrine system, no inflammatory processes and hence no immunodeficiency, no previous trauma, and no parafunctional activity, etc.), an iatrogenic (orthodontic) ERR results when a given impulse ($F \cdot \Delta t$) is applied to a root surface. Initially, a force, which largely determines the impulse, will cause an instantaneous compression of the periodontal ligament, most likely exceeding a relative critical barrier (λ). If λ stands for the relative reduction in periodontal ligament width for a given root site, then we hypothesize that at values exceeding λ, the periodontal ligament will fail to operate as a physio-immune system protecting the root surface from clastic assault. The onset of the ERR sequelae is determined by the environmental density, which is considered a secondary factor. The following discussion elucidates the three ERR determinants: impulse, critical barrier, and environmental density.

Impulse

The current study indicates that ERR is a function of impulse (momentum), i.e. the product of the average force and the time during which it acts [ERR = f $({}^{t2}\int_{t1} F dt)$]. The interplay between the two components of the impulse

Table I
External root resorption index per palatal expansion appliance per root surface for the short- and long-term groups.

Time	Short-term				Long-term			
Appliance	IS	IM	DM	IC	IS	IM	DM	IC
Root Surface								
Buccal (teeth 2-7)	46.9±41.2	27.5±36.4	13.0±11.7	9.6±14.0	47.8±27.9	48.9±22.4	2.5±2.8	13.3±15.0
Palatal (teeth 2-7)	1.2±3.0	0.0	3.4±5.0	3.7±8.3	47.5±9.5	41.4±21.0	7.4±5.1	17.3±15.8
Mesial (teeth 2-7)	3.7±4.7	0.9±2.3	4.3±5.9	5.2±8.1	30.3±14.2	28.4±23.7	1.9±2.9	7.9±10.4
Distal (teeth 2-7)	10.5±14.4	0.0	1.9±3.1	5.2±9.6	29.0±15.2	28.4±16.8	2.2±3.4	8.5±7.7
Furcation (teeth 4-7)	55.2±15.0	16.7±9.6	5.2±10.4	29.2±21.7	26.4±19.4	26.1±19.4	1.1±2.1	1.4±2.4

suggests that during initial, short time periods, magnitude of force is the predominant factor dictating ERR intensity and is most likely related to ERR in a logarithmic function. However, with longer treatment, time becomes the deciding factor. Thus, although the **IS** appliance exerts eight times more force than the **IM** appliance, the **IS** buccal ERR index in the short-term group is only 1.7 times greater than that of the **IM** (Table I). However, by taking treatment duration into account, an equivalent ratio arises between ERR indices and impulses for the two indirect appliances in the short-term group, that is:

$$\frac{ERR_{IS,SHORT,BUCCAL}}{ERR_{IM,SHORT,BUCCAL}} \simeq \frac{Impulse_{IS,SHORT}}{Impulse_{IM,SHORT}} = \frac{46.9}{27.5} \simeq \frac{2035g \times 33d}{258g \times 135d}$$

Long-term ERR effects are controlled by impairment/repair dynamics. Since severe ERR damage is irreversible (at least for the observed 6 month post-treatment period), both the **IS** and the **IM** subjects in the long-term group showed basically the same level of ERR (47.8 ± 27.9 vs. 48.9 ± 22.4). The impulse-related response of ERR is very similar to that of an inflammatory process. While an acute inflammatory process is determined by an external factor like bacterial invasion (force in short-term ERR), a chronic inflammatory process (long-term ERR) is determined by the efficiency (time factor) of the body or dental immuno-defense system.[23]

Critical barrier λ

In all probability, no root resorption will be initiated unless a narrowing of the periodontal ligament width, referred to as the periodontal ligament critical barrier (λ), is exceeded. The amount of the narrowing, not the residual periodontal ligament width, is the determinate measure for ERR onset. However, there is no data regarding the value of λ at which osteoclastic activity turns into osteo-odontoclastic activity. Göz et al.[24] found a maximal reduction in periodontal ligament breadth from 78.4 μm to 11 μm when dog premolars were loaded for 7 hours under a dynamic force of 20 N, a stage where periodontal ligament vascular impairments were observed. However, like Coolidge,[25] they also noted that the normal periodontal ligament contour in the medio-lateral plane is shaped like an hour-glass, with the narrowest width located one-eighth of the root length apical to the midroot.[26]

Thus, the location of the narrowest periodontal ligament width along the root axis corresponds to the position of the calculated center of rotation when a tipping force system is acting on a tooth.[27] That is, during tipping movement, when the center of rotation is located apical to the center of resistance,[27] no change in periodontal ligament width is expected at the narrowest width. Conversely, substantial changes are expected in root extremities. For this reason, when strictly tipping movement occurs, more marginal root areas are prone to ERR assault.[7,11,19] In contrast, in the current study, the almost uniformly distributed ERR sites along the three root zones, with a slight amplification of the apical zone (Figure 6), suggests more con-

trolled bodily tooth movement. This finding is in agreement with studies by Follin[19] and Kvam.[8] Thus, the association of ERR with a specific type of tooth movement[28] corresponds to surpassing the critical barrier at diverse root surface areas.

The λ-related response of the odontoclastic process is presumably related to the initial synergistic mechanism between the osteoclastic cell line and the osteoblastic cell line. This was hypothesized[29] on the grounds that parathyroid receptors have been found on osteoblasts but not on osteoclasts,[30] and that only osteoblasts are capable of synthesizing both procollagenase and collagenase enzymes.[31,32] Thus, a rise in the number of osteoclast-mediated PTH, and the prerequisite degradation of mineralized cementum matrix collagen prior to scavenging of mineralized cementum matrix by osteoclast progenitor cells (odontoclasts), might well be regulated by osteoblastic activity. However, it is most unlikely that cementoblasts play a roll as "helper cells" in cementum resorption, since their response to the parathyroid hormone differs from that of osteoblasts.[33] On the contrary, it is more plausible that cementum resorption is inhibited by the activity of cementoblast-like cells and Sharpey's fibers.[34] The presumably coupling mechanism (odontoclasts/osteoblasts) might well explain the morphological requirement of the alveolar socket to approximate the cementum root surface to incite degradation of precementum (nonmineralized cementum matrix). Once the resorption process has penetrated the resistant precementum and cementum layers, it will proceed rapidly, radiating peripherally in the dentine tissue,[6] irrespective of λ values. Thus, the direct relationship between λ and ERR does not hold true when a prolonged time factor is introduced. The immuno-physiological reaction to hyperfunction conditioning is increased periodontal ligament width (+ 50%),[25] while decreased width is a response to hypofunction.[25,35] A change of periodontal ligament breadth exceeding the value of λ should be considered a malfunction (pathology) of the ligament's normal physiological defense system.

Environmental density

By definition, environmental density relates to the density (physical property) of any environment opposing tooth movement. This includes bone (compact vs. cancellous), tooth (e.g. supernumerary, ectopic), or neoplasms (e.g. cysts, osteoscleroses, tumors).

The present study, supported by previous reports,[19,20] found bone density to be only a secondary ERR predisposing factor. This inference is

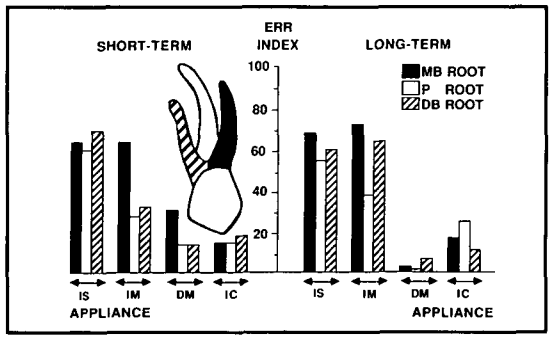

Figure 5A

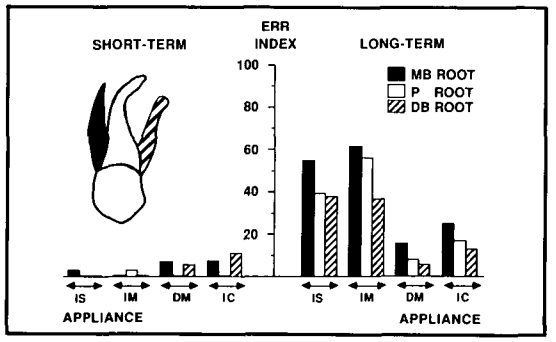

Figure 5B

Figure 5A-B
External root resorption per root of maxillary multirooted teeth (4-7) per palatal expansion appliance for the short- and long-term groups. A. Buccal root surface, B. Palatal root surface — the mesiobuccal root was the most ERR affected root for both sides.

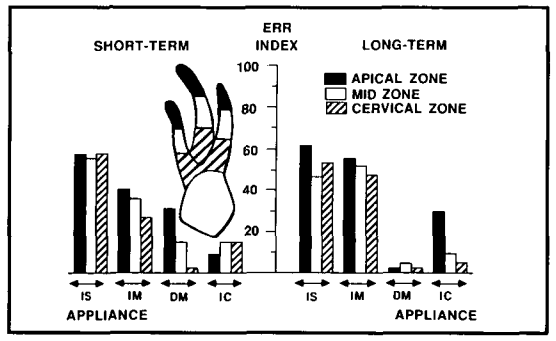

Figure 6A

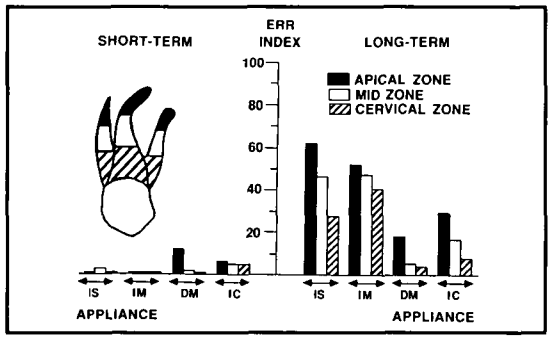

Figure 6B

Figure 6A-B
External root resorption per root zone of maxillary multirooted teeth (4-7) per palatal expansion appliance for the short- and long-term groups. A. Buccal root surface, B. Palatal root surface — the apical root third was the most ERR impaired vertical root zone for both sides.

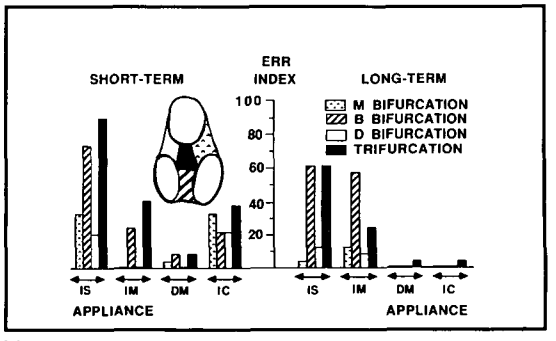

Figure 7

Figure 7
External root resorption per furcation area of maxillary multirooted teeth (4-7) per palatal expansion appliance for the short- and long-term groups. The buccal furcation and trifurcation areas were mostly affected by ERR assault.

based on three findings: (A) the mesiobuccal root of multirooted teeth, being positioned in closest proximity to the buccal cortical plate[36] (Figures 2A and 5), showed 12% and 49% increases in ERR assault as compared to the distobuccal and palatal roots, respectively. (B) The single-rooted canine, despite abutting all indirect PE appliances, exhibited 120% less ERR than all anchored multirooted teeth (Figures 1A-D; 2A). By reason of the tipping movement, the canine root moved away from the dense laminated bone, whereas multirooted teeth translated bodily into the buccal cortical plate. (C) An additional observation seems to refute the concept of bone density. Roots located near the dense buccal cortical plate failed to demonstrate greater ERR indices than roots near the less dense palatal cortical plate (e.g. $ERR_{IS,\ SHORT,\ BUCCAL} = 46.9 \pm 41.2$ vs. $ERR_{IS,LONG\ PALATAL} = 47.5 \pm 9.7$) (Table I; Figures 1A and B). However, the unexpectedly high palatal ERR score of the long-term group is attributed to both the jiggling effect, which affected not only the palatal root surface (Table I; Figures 1B and 2B) but also the mesial and distal root surfaces (Table I; Figures 1C and D), and the extended time factor (see repair section).

The question of whether environmental density is a primary or secondary cause of iatrogenic ERR has no simple answer. Follin et al.,[19] in a study of dogs, found that when a lower premolar was bodily mesialized into an extraction space, its mesial root experienced threefold less ERR than its distal root. However, they also noticed that for the same environmental condition (extraction space), the dispersion and magnitude of ERR varied when tipping or bodily movements were introduced (i.e. the critical barrier factor). Goldie et al.[20] documented that tooth movement in lactating rats fed on a calcium deficient diet resulted in less ERR than in control animals. However, ERR response was time-related and was reversed by day 14. That is, in the calcium-deficient animals, a time lag was required to reach λ values, but when the critical barrier was achieved, the treated animals showed even greater ERR than their control counterparts. Some delay in ERR response, but not elimination, was also evidenced when HEBP, a mineralization blocking agent, was administered to rats in conjunction with periodontal ligament cryoprobe treatment.[16] Thus it can be concluded that environmental density is secondary to primary factors like critical barrier and impulse.

Repair

The comparison of the short-term and long-term groups elucidates the impairment/repair mechanism. Impairment/repair dynamics are most likely regulated by three principles: ERR level of irreversibility, delayed resorption response and jiggling.

The first principle, ERR level of irreversibility, refers to resorption damage at a level above which no repair will take place. Most likely, ERR scores above 2 fall into this category. That is, all cementum and superficial dentinal resorption sites with an ERR score of 1 will undergo repair; augmented dentinal resorption patches with an ERR score of 2 will undergo repair unless there is continuing damage due to a delayed resorption response and/or jiggling degrades the dentinal resorbed patches into an ERR score of 3; and finally, resorbed sites with an ERR score of 3 are irreparable. ERR level of irreversibility, is demonstrated by the consistency in buccal ERR scores between the short-term and long-term groups for the IS and IM subjects (Table I; Figures 1A and E) (e.g. $ERR_{IS,\ SHORT,\ BUCCAL} = 46.9 \pm 41.2$ vs. $ERR_{IS,\ LONG,\ BUCCAL} = 47.8 \pm 27.9$). In contrast, the DM subjects, with low ERR scores by the end of the active phase, showed almost a complete recovery by the end of the retention phase, as follows: $ERR_{DM,\ SHORT,\ BUCCAL} = 13.0 \pm 11.7$ vs. $ERR_{DM,\ LONG,\ BUCCAL} = 2.5 \pm 2.8$.

The second principle of impairment/repair dynamics, the delayed resorption response (initiation and decay) is responsible for the lag in the repair response. The resorption process starts at day 10-20 from force onset[6-9] and continues for several weeks after force elimination.[10,11,28] Most likely, increases in the time and severity of the active resorption phase will extend the decay phase. Histological studies have demonstrated that while repair in the form of cellular cementum progressed from the centers of the patches outward, resorption continued to radiate and expand at the periphery of the patches.[7,9] Moreover, while cervical zones undergo healing, apical zones might still undergo resorption assault.[7] Additionally, the deficiency of Sharpey's fibers in cellular cementum makes repaired and apical cementum more vulnerable to resorption.[15] Recently, King et al.[23] demonstrated a systemic depression in the titer of dentinal antibodies which lasted as long as denuded dentin was undercovered by secondary cementum (or recovered to baseline values when the affected tooth was extracted). The delayed resorption decay is implied by the poor recovery response (actually no recovery) associated with the IM subjects (Table I; Figures 1A and E). The drastic continuous rise in ERR score from short- to long-term subject ($ERR_{IM,\ SHORT,\ BUCCAL} = 27.5 \pm 36.4$ vs. $ERR_{IM,\ LONG,\ BUCCAL} = 48.9 \pm 22.4$) is the result of ex-

tended active treatment time ($IM_{SHORT} = 135$ d, $IM_{LONG} = 216$ d).

The third principle, the jiggling resorption, is a resorption related to oscillating tooth movement. Jiggling resorption might be due to a delayed auto-immune response,[23] and/or a boost in odontoclasts (Figure 2B) by reason of recurrent bone/tooth contact ($>\lambda$), and/or motility of existing osteoclasts (odontoclasts). The latter is well demonstrated by a 400% increase in ERR indices for both mesial and distal root surfaces from short-to long-term groups (Table I; Figures 1C and D) (e.g. $ERR_{IM, SHORT, MESIAL} = 0.9 \pm 2.3$ vs. $ERR_{IM, LONG, MESIAL} = 28.4 \pm 23.7$). This change is to be explained by the migration of osteoclasts (odontoclasts) from the previous compression side (buccal) to the new compression side (palatal), along the mesial and distal root surfaces. Likewise, resorption along the mesiobuccal and distobuccal aspects of the roots was also found by Timms and Moss[11] in a long-term PE study subsequent to the relapse phase. Clearly, the iatrogenic potential of rapid palatal expansion should be considered clinically.

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