CHAPTER II

LITERATURE REVIEW

The review is divided into six parts as follows:

I. Biology of tooth movement

II. Histological aspects of external apical root resorption

III. Classification and severity of root resorption

IV. Factors affecting external apical root resorption

a. Biological factors

b. Mechanical factors

V. Diagnostic aids for detection of root resorption

VI. Methods to accurately measure root length

I. Biology of tooth movement

Orthodontic tooth movement requires a healthy periodontal ligament (PDL) to remodel tooth and alveolar bone physiology. Forces applied to teeth are mediated through the PDL and result in remodeling of periodontal tissues. When force is transmitted to a tooth and then the tooth moves, the entire surface of the tooth socket is affected. The PDL and alveolar bone on the pressure side are compressed, resulting in bone resorption. On the opposite surface of the root, the movement stretches the ligament fibers, causing tension, which results in bone formation.[11] (Figure 2.1)

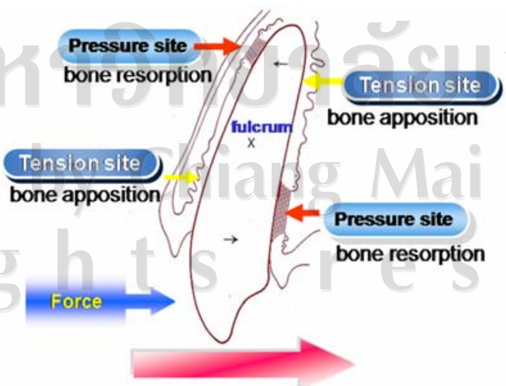

Figure 2.1 Graphical illustration of orthodontic tooth movement, where orthodontic force is applied (after Proffit and Fields[11])

Proffit and Fields[11] proposed that two theories, the bioelectric and the pressure/tension theories, may play a part in the biologic control of tooth movement.

The bioelectric theory relates tooth movement, at least in part, to changes in bone metabolism that are controlled by the electric signals produced when alveolar bone flexes and bends. Electric signals that might initiate tooth movement were initially thought to be piezoelectric. Piezoelectricity is a phenomenon observed in many crystalline materials, in which a deformation of crystal structure produces a flow of electric current as electrons are displaced from one part of the crystal lattice to another. When force is applied to bone or collagen, a flow of current is produced that quickly dies away. When the force is released, an opposite current flow is observed. The piezoelectric effect results from migration of electrons within the crystal lattice of bone.

The pressure/tension theory is the classic theory of tooth movement, which relies on chemical, rather than electric, signals as the stimulus for cellular differentiation and, ultimately, tooth movement. An alteration in blood flow within the PDL space is produced by the sustained pressure that causes the tooth to shift position within the PDL space, compressing the ligament in some areas while stretching it in others. Blood flow is decreased where the PDL is compressed, while it usually is maintained or increased where the PDL is under tension. If regions of the PDL are overstretched, blood flow may be decreased transiently. Alterations in blood flow quickly create changes in the chemical environment. For instance, oxygen levels certainly fall in the compressed area, but might increase on the tension side and the relative proportion of other levels of metabolic activity would also change in a matter of minutes. In essence, this view of tooth movement shows three stages: (1) alterations in blood flow associated with pressure within the PDL, (2) the formation and/or release of chemical messengers, and (3) activation of cells.

Figure 2.2 illustrate a typical tooth movement response after application of a moderate, continuous load (0.2-0.5 N, or about 20 to 50 g). Orthodontic tooth movement can be divided into three phases: the initial phase, lag phase and postlag phase (progressive tooth movement). The initial phase is characterized by the period of rapid tooth movement which largely represents displacement of the tooth in the periodontal space. The initial phase is followed immediately by the lag phase, in which the tooth either does not move or shows a relatively low rate of displacement. The lag phase is caused by hyalinization of the PDL in areas of excessive stress. Heavy force results in a larger area of hyalinized tissue. No tooth movement can occur until the area of hyalinization has been removed by cellular processes. The third phase of tooth movement is the postlag phase, or progressive tooth movement; it occurs when the rate of tooth movement either gradually or suddenly increases.[12]

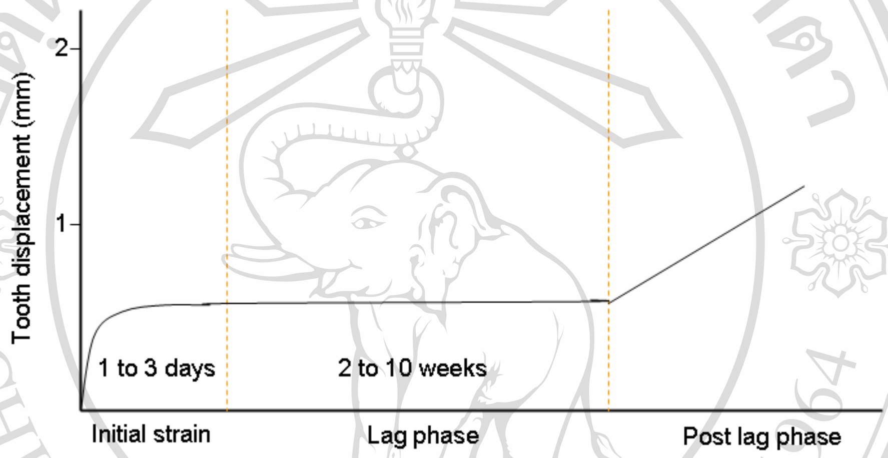

Figure 2.2 A typical tooth movement response after application of force (Redrawn from Robert[12])

II. Histological aspects of external apical root resorption

Orthodontically-induced EARR is a part of the elimination process of the hyalinized zone.[13-18] Macrophage-like cells are the first cells which are involved in this process of necrotic tissue removal. They are most probably activated by signals coming from sterile necrotic tissue, which is the result of orthodontic force application. During removal of the hyalinized zone, the nearby outer surface of the root, which consists of the cementoblast layer covering the cementoid, can be damaged,[19] exposing the underlying, highly dense, mineralized cementum.

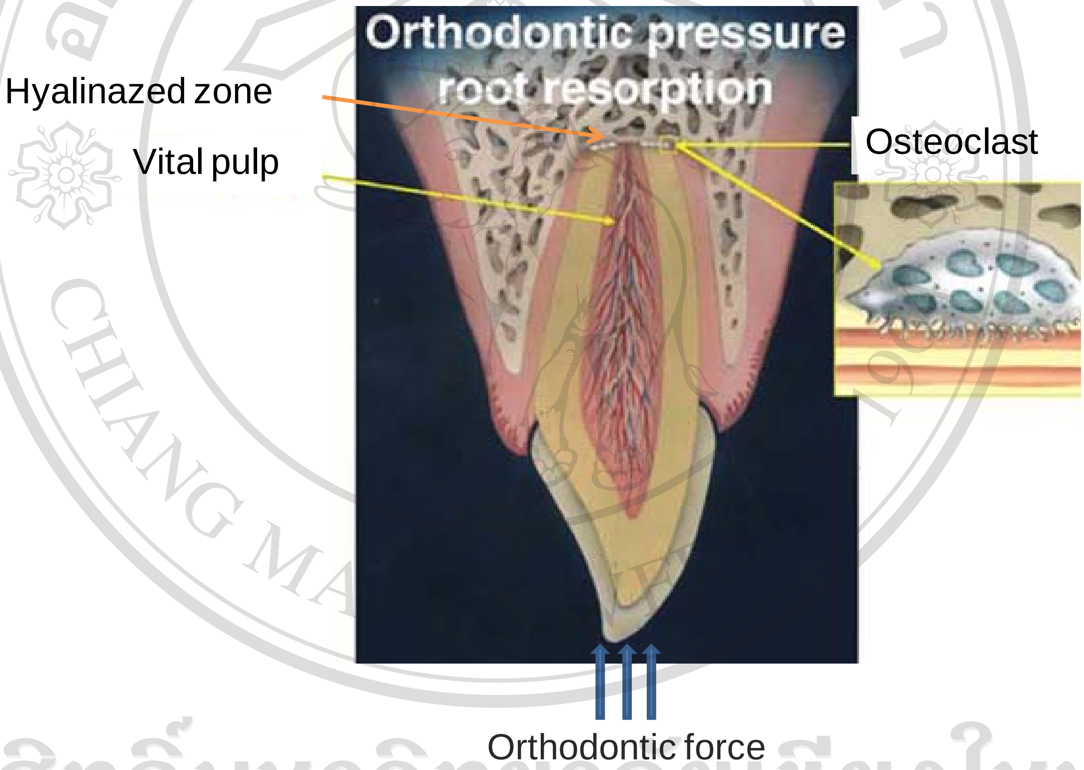

Figure 2.3 Graphical illustration of orthodontic pressure-induced EARR (after Fuss et al[1])

Figure 2.3 illustrates the excessive orthodontic pressure that results in a hyalinazed zone which, in turn, attracts macrophage-like cells. The macrophage-like cells later differentiate to osleoclasts and then resorb the root. According to Brudvik and Rygh,[20] the resorption process continues until no hyalinized tissue is present and/or the force level decreases. Resorption lacunae expand the involved root surfaces and thereby indirectly decrease the pressure exerted through force application. Thus, decompression allows the process to reverse and the cementum to be repaired. The repair process occurs by apposition of cementum-like tissue in the resorption lacunae. The small, shallow resorption lacunae may be completely filled with new cementum.

III. Classification and severity of root resorption

Andreasen[21] defined three EARR types: *surface resorption*, which is a self-limiting process, whereby small, thin, resorbed areas at the root surface, which are spontaneously repaired from adjacent intact parts of the periodontal ligament; *inflammatory resorption*, where inflammation from infected necrotic pulpal tissue extends through the dentinal tubules to the root surface, or where an infected leukocyte zone at the root surface causes resorption; and *replacement resorption*, where bone replaces the resorbed tooth material, leading to ankylosis.

EARR after orthodontic treatment is either surface resorption, or transient inflammatory resorption. Replacement resorption is rarely, if ever, seen after orthodontic treatment.

Brezniak and Wasserstein[22] classified the severity of EARR in three degrees:

1. Cemental or surface resorption with remodeling. In this process, only the outer cemental layers are resorbed and they are later fully regenerated or remodeled;
2. Dentinal resorption with repair (deep resorption). In this process, the cementum and the outer layers of the dentine are resorbed and usually repaired with cementum material. The final shape of the root after this resorption and formation process may or may not be identical to the original form;
3. Circumferential apical root resorption. In this process, full resorption of the hard tissue components of the root apex occurs, and root shortening is evident. (Figure 2.4)

Figure 2.4 Circumferential apical root resorption

IV. Factors affecting external apical root resorption

EARR is considered a multifactorial problem and has been related to factors associated with biological variation and with treatment modalities.[8]

There is no single explanation why teeth resorb to various degrees, but a number of factors taken together may explain why resorption takes place. Brezniak and Wasserstein[8, 9] reviewed and explained the factors which effect EARR. They described biologic factors, including individual susceptibility, genetics, systemic factors, nutrition, chronological age, dental age, sex, habits, tooth structure, previously traumatized teeth, endodontically treated teeth, presence of an impacted canine, alveolar bone density, types of malocclusion, overjet, overbite and specific tooth susceptibility to root resorption, and mechanical factors, including types of appliance, orthodontic movement type, orthodontic force, treatment duration, intermaxillary elastic used, performance of Lefort I osteotomy, and whether or not there is a history of extraction.

a. Biological factors

1. Individual susceptibility: Individual susceptibility is considered a major factor in determining root resorption potential, with or without orthodontic treatment.[23] In some individuals one or more teeth occasionally appear to be shortened by root resorption before any orthodontic treatment has started. Several causes of such root resorption, not related to orthodontics, have been observed. For instance, pressure from an erupting maxillary canine can cause root resorption on the distal surface of the adjacent lateral incisor. Damage to the periodontium after trauma can result in root resorption. The etiology of spontaneous resorption of lacunae is unknown.

Rygh[24] used light and electron microscopy to investigate 11 human premolar teeth, which were extracted after being moved buccally by fixed appliances for periods between two and 50 days. The forces used in the experiment were 70, 100, 120 and 240 grams. The root resorption process seemed to vary among people and within the same person at different times. Metabolic signals that generated changes in the relationship between osteoblastic and osteoclasic activity included hormones, body type, and metabolic rate. Rygh[24] suggested that it is reasonable to assume that disturbances or particularity in this interplay may explain the individual tendency to marked root resorption.

2. Genetics: Some studies strongly suggest a genetic component for shortened roots.[25, 26] In 2003 Al-Qawasmi[25] found that the *TNFRSF11A* locus, or another tightly linked gene, was associated with EARR. Moreover, a recent study by Hartsfield et al[26] suggested that decreased Interleukin 1 beta (IL-1ß) production in the case of IL-1ß (+3953) allele 1 may result in relatively reduced catabolic bone remodeling (resorption) at the cortical bone interface with the PDL, which may have resulted in prolonged stress concentrated in the root of the tooth, triggering a cascade of fatigue-related events, leading to root resorption.

3. Systemic factors: According to Becks,[27] endocrine problems, including hypothyroidism, hypopituitarism, hyperpituitarism, and other diseases, were related to root resorption. Loberg and Engstrom[28] and Prumpros et al[29] found that thyroxine can reduce EARR and is correlated with a change in the bone modeling process, especially as related to the resorption activity. Recently, Verna et al[30] investigated the effect of acute and chronic corticosteroid treatment on orthodontically induced EARR. Sixty four six-month-old male rats were divided into three groups; acute (n = 22), chronic (n = 23) and control groups (n = 19). The acute and chronic groups received corticosteroid treatment (8 mg/kg/day) for 3 and 7 weeks, respectively, whereas no pharmacological treatment was performed in the control group. Then the upper left first molar was moved mesially for 21 days in all three groups with 25 grams of force. The acute group showed significantly more EARR compared with either the control or the chronic group. They suggested that a careful monitoring of patients undergoing acute corticosteroid treatment should be performed, because they found a lack of balance between osteoblastic (inhibited by the drug) and the osteoclastic activities (enhanced or unchanged by drug administration) occurring in the initial phase of drug administration.

Allergic conditions have been reported that can increase the risk of EARR in orthodontically treated patients. In reviewing orthodontic patient records at the University of Oklahoma, Davidovitch et al[31] found higher excessive EARR during orthodontic treatment in patients who had experienced the incidence of asthma, allergies, and signs indicative of psychological stress, compared with a group of orthodontic patients who had completed orthodontic treatment without suffering these medical conditions.

4. Nutrition: Linge and Linge[5] suggested that nutritional imbalance was not a major factor in EARR during orthodontic treatment. However, in 1988, Engstrom et al[32] investigated the effects of orthodontic forces on the periodontal tissues in the normal and the hypocalcemic situation with secondary hyperparathyroidism in rats. Moderate orthodontic force was applied to upper incisors of normal and hypocalcemic rats in order to induce EARR. They showed that EARR was clearly related to the degradation process occurring near the hyaline zone in the hypocalcemic situation. Moreover, the increase in EARR was related to enhanced alveolar bone resorption. Late, in 1997, Bielaczyc and Golebiewska[33] studied EARR in 20 young Wistar rats. The results from the scanning electron microscope showed increased root resorption on the pressure side of the root after orthodontic tooth movement in rats fed a low-calcium, vitamin D-deficient diet.

5. Chronologic age: It can be explained that all tissues involved in the root resorption process show changes with increasing age. The periodontal ligament becomes less vascular and aplastic, the bone more dense, more avascular and more aplastic and the cementum wider. Besides that, both cell mobilization and conversion of collagen fibers are considerably slower in elderly individuals than in children and adolescents. These changes are reflected by a higher susceptibility to root resorption in children than in adults.[34] However, there are controversial reports in the literature. Some studies[35,36] have found no significant relationship between root resorption and chronological age.

Hendrix et al[35] determined the extent of EARR, after fixed appliance therapy, in the posterior part of the dentition. They measured tooth length on pre- and post-treatment panoramic radiographs of 153 patients who were treated with the standard edgewise appliance. Mean age at the beginning of treatment were 14.4 ± 2.8 years (range 10.8-24.4 years). They found that root shortening of posterior teeth during active orthodontic treatment was not dependent on age at the beginning of treatment.

Harris and Baker[36] studied EARR in 59 orthodontically treated patients (29 adolescents, 30 adults). The mean ages of adults (defined as being > 20 years at the beginning of treatment) and adolescents were 28 and 12 years, respectively. The adult and adolescent groups were matched for sex, malocclusion and treatment regimen. The root resorption measurement was performed on the central incisor, canine and first molar (distal root, the longer if there were two) teeth in the maxilla and the canine and first molar (distal root) in the mandible. The incisor and canine root measurements were taken from the standardized lateral cephalograms. Molar root lengths were measured from panoramic radiographs. At the start of treatment, adults had significantly shorter roots than did the adolescents. However, after treatment there was no difference in root length between both groups (average of 1.0 to 1.5 mm with the highest rates for the maxillary incisors). The authors concluded that the orthodontic treatment did not place adults at greater risk, but adult patients should be carefully evaluated before the start of orthodontic treatment. However, a study found that adult patients experienced more EARR than did children. Sameshima and Sinclair[3] investigated 868 patient records and divided age at the start of treatment into adults (age at start >16 years) and children. They compared mean amount of EARR of upper and lower anterior teeth between adults and children. Adults had significantly more EARR than children in the lower incisors and canines by as much as 0.8 mm. However, there was no statistical difference in root resorption in maxillary anterior teeth.

6. Dental age: Root development can be affected by tooth movement, resulting in dilacerations, decreased expected root length and increased root resorption. Hendrix et al[35] attemped to explain the differences by stage of root formation at onset of orthodontic treatment. Patients were divided into two groups according to their root formation. Group A consisted of patients with incomplete root formation, except for the first molars, at onset of orthodontic treatment. Group B consisted of patients where root formation was completed, with the exception of second and third molars. Root measurement was measured on panoramic radiographs. Post-treatment tooth lengths in Groups A and B were compared with pre-treatment tooth lengths in group B with a paired *t*-test. Mean root lengths in post-treatment Group A showed no significant difference from the mean root lengths in pre-treatment Group B. Differences between the mean pre-treatment and post-treatment root lengths in Group B were significant. Teeth with incomplete root formation at onset of orthodontic treatment showed root lengthening during active treatment, but did not reach their "normal" tooth length.

Mavragani et al,[37] investigated root lengthening during orthodontic treatment in relation to the developmental stage of the root. Root development was characterized as complete or incomplete, depending on whether or not the root apex was closed. The sample consisted of 80 patients with class II division 1 malocclusions, treated with extraction of at least two maxillary first premolar teeth and using the edgewise orthodontic technique. A control group of 66 untreated individuals matched by sex, pre- and post-treatment age with the experimental group was included. Crown and root lengths of maxillary incisors were measured on periapical radiographs before and after treatment. Root elongation during treatment did not differ from untreated teeth of similarly aged individuals. There was no significant difference in the extent of root lengthening between the root elongation during treatment and the normal root lengthening in aged-match untreated individuals.

7. Sex: Most studies have not found a consistent association between sex and orthodontically-induced EARR.[3,38] Sameshima and Sinclair[3] compared mean amount of EARR of upper and lower anterior teeth between 313 male and 555 female patients. They found no statistically significant difference in root resorption between male and female patients. Harris et al[38] studied 206 orthodontically treated patients, consisting of 84 boys and 122 girls. Mean age at the first examination was 14.1 for boys and 13.3 for girls. Measurements were made on maxillary central incisors, mandibular central incisors and left and right mandibular first molars. The maxillary and mandibular central incisors were measured from the pre- and post-treatment cephalometric radiographs. The mesial and distal roots of left and right lower first molars were measured on the pre- and post-treatment panoramic radiographs. Despite male-female differences in mean root lengths, there was no statistical difference between amounts of root length during treatment. However, some previous studies found differences in root resorption between sexes.[39,40] Baumrind et al[39] investigated a group of adult (aged more than 20 years) orthodontic patients. The mean EARR was 2.29 ± 0.35 mm in males and 1.09 ± 0.18 mm in females. Males had statistically greater prevalence of EARR than did females. In contrast, Kjar[40] investigated 70 girls and 37 boys who were treated by 35 orthodontists and found a greater prevalence of EARR in girls than in boys. Panoramic radiographs and additional dental films were analyzed.

8. Habits: Nail-biting[41] and tongue-thrusting habits associated with open bite, and increased tongue pressure[42] have been statistically related to increased root resorption. Odermick and Brattstrom[41] determined the incidence and intensity of nail-biting and its possible role in EARR during orthodontic treatment. The possible effect of nail-biting on EARR during orthodontic treatment was evaluated by radiographic examination of two groups, matched with regard to overjet, age, sex, duration and types of fixed orthodontic appliance treatment. One group consisted of 21 severe nail-biters and the other group, 21 patients without the habit. The apical root resorption was significantly increased in the severe nail-biters than in the patients without the habit both before and after orthodontic treatment.

Harris and Butler[42] studied 32 adolescents with the tongue-thrusting habit, leading to anterior open bites. Mean starting ages for experimental and control groups were early adolescence, (range from 10 to 32 years). Incisor root resorption was assessed from the pre- and post-treatment lateral cephalograms. The investigators found that the roots of permanent maxillary central incisors in patients who had tongue-hrusting with anterior open bite were significantly shorter and exhibited higher modal grades of EARR than those in the control group. They concluded that tongue-hrusting habits apply long term force to anterior teeth and enhance EARR.

9. Tooth structure: Deviating root form is more susceptible to EARR after orthodontic treatment.[3,4,7] Sameshima and Sinclair[3] examined records of 868 patients who were treated with full, fixed edgewise appliances. The EARR was assessed from first molar to first molar in both arches. Root shape was categorized into normal, blunted, pipette or bottle-shaped, pointed, dilacerated, and incomplete root shape. They found that dilacerated teeth (particularly maxillary lateral incisors) had the most root resorption, followed by bottle-root shaped and pointed root shaped teeth.

Mirabella and Artun[4] studied 343 adult records and periapical radiographs of maxillary anterior teeth. They first subjectively scored root shape as normal, eroded, pointed, bent or bottle shaped However, they did not classify the root shape when they analyzed the data. They concluded that the presence of long, narrow and atypical root shapes increased the risk of EARR.

Smale et al[7] examined periapical radiographs of maxillary incisors in 290 patients. They found that pointed or deviated root shapes were associated with increased EARR. Moreover, they found that wider central incisors roots and normal root form reduced the risk of EARR.

Moreover, Levander and Malmgren[43] studied 610 maxillary incisors in 153 patients (75 boys, 78 girls). The root form was classified as normal, short, blunt, apically bent, pipette-shaped. Intra-oral radiographs, before and after treatment, were evaluated. An index from 0 to 4 was used for the evaluation of the degree of EARR. The investigators found that the degree of EARR in teeth with blunt or pipette-shaped roots is significantly higher than it is in teeth with normal root form.

10. Previously traumatized teeth: Traumatized teeth can exhibit EARR without orthodontic treatment.[7] Many articles have reported that EARR can be induced in orthodontically-moved, traumatized teeth.[6, 10, 44, 45] Moreover, traumatized teeth with previously existing EARR are more sensitive to further loss of root material.[45]

Malmgren et al[45] studied the frequency and degree of EARR in traumatized incisors that have been orthodontically treated. The subjects were 27 patients (15 boys and 12 girls) with 55 traumatized incisors; 55 incisors without previous trauma served as controls. All the control patients were treated with extraction of four first premolars and a fixed appliance. The degree of EARR in traumatized teeth was compared to that in the uninjured control teeth in the same patient and in the patients without trauma. Either the intra-individual or the inter-individual comparisons support the finding that traumatized teeth have a greater tendency toward EARR than do uninjured teeth.

Drysadale et al,[46] in a review of the literature, suggested that it is important to know the specific type of dental injury in order to evaluate long term prognosis. Luxation or avulsion is particularly liable to damage the root surface cementum and predispose the root to resorbtion.

11. Endodontically treated teeth: A higher frequency and severity of EARR of endontically treated teeth during orthodontic treatment has been reported in an earlier study.[47] However, in 1990 Spurrier et al[48] studied 43 patients who had one or more endodontically treated incisors before orthodontic treatment and exhibited signs of EARR after treatment. In each patient the vital contra-lateral incisor served as a control. Vital incisors resorbed to a significantly greater degree than did endodontically treated incisors. Many later studies have suggested that endodontically treated teeth are more resistant to resorption because of increased dentin hardness and density.[4,49] However, Hamilton and Gutmann[49] suggested that minimal resorptive/remodeling change occurred apically in teeth that were being moved orthodontically and that were well cleaned, shaped and three-dimensionally obturated.

12. Presence of impacted canines: EARR of maxillary incisor roots resulting from pressure form an impacted canine is a well-recognized phenomenon.[50-52] Shellhart et al[50] reported advanced resorption of lateral incisors caused by bilaterally impacted maxillary canines. Sasakura et al[51] analyzed 23 cases with maxillary impacted canines. They found unusual EARR of 12 maxillary permanent central and 11 lateral incisors. The degree of EARR ranged from loss of the apical quarter to almost complete loss of root structure. This finding suggests that the pressure from a canine which persists in moving downward, despite the lack of space to permit normal eruption, can cause EARR of adjacent incisors, even in the absence of systemic factors. Recently, Milberg[52] reported that labial impaction of maxillary canines caused severe pressure EARR on the lateral aspects of the maxillary central incisors.

13. Alveolar bone density: Controversial reports on EARR and alveolar bone density appear in the literature. Thilander et al[23] reported that increased density of alveolar bone caused increased EARR during orthodontic treatment. However, Wainwright[53] reported that bone density affects the tooth movement rate but has no relation to the EARR. This is in agreement with Otis et al,[54] who suggested that the density and morphology of the dento-alveolar complex are not significant factors in the etiology of EARR. They examined the amount of alveolar bone around the root, the thickness of cortical bone, the density of the trabecular network and fractal dimensions on cephalometric radiographs of 22 patients with evidence of EARR on mandibular incisors.

14. Types of malocclusion: Skeletal discrepancy and dental malocclusions should be considered cautiously with EARR. Numerous factors affect the development and treatment of each malocclusion and EARR. Therefore, it is not surprising to find conflicting and controversial conclusions in recent and past studies.[4, 39, 55]

Mirabella and Artun[4] and Baumind et al[39] found no statistical difference in EARR regardless of the type of malocclusion. In contrast, Taner et al[55] who found that EARR was significantly higher in Class II malocclusion than in Class I malocclusion. They studied 27 patients with Class I and 27 with Class II malocclusions (16 girls and 11 boys), for whom first premolar extractions were planned. The average ages at start of treatment were 12.54±1.88 years for the Class I group and 13.61±2.51 years for the Class II division 1 group. The amount of EARR of central maxillary incisors was determined for each patient by subtracting the post-treatment tooth length from pre-treatment tooth length measured directly on cephalograms.

15. Overjet: Most studies have reported that overjet was associated with EARR. Linge and Linge[6] analyzed patient characteristics and clinical variables that may be associated with EARR in the maxillary incisor. EARR was measured in periapical radiographs from 485 treated patients aged 11.5 to 25 years at the beginning of treatment. Overjet was found to have significantly positive correlation to EARR. Sameshima and Sinclair[3] studied 868 patient records and their periapical radiographs. They found that overjet significantly contributed to EARR. They suggested that overjet often requires treatment with fixed appliances, with active torque, with rectangular arch wire and with the use of Class II elastics. They also found that overjet always results in trauma to incisor teeth. Furthermore, they suggested that the treatments used to correct large overjets were, themselves, risk factors for EARR. However, Linge and Linge[6] stated that some cases of overjet treated with activators were not found to result in EARR.

16. Overbite: Sameshima and Sinclair[3] found no correlation between the amount of overbite present at the beginning of treatment and the amount of EARR. Linge and Linge[6] found that overbite was not closely related to change in root length. But, these results are in contrast to those of Harris and Butler[42], who found that patients with open bite and tongue-thrusting experienced more EARR than did patients with normal bite or deep bite characteristics.

17. Specific tooth susceptibility to root resorption: A review by Brezniak and Wasserstein[8, 9] found that different teeth had different tendencies to resorb the root. All teeth examined after orthodontic treatment showed evidence of EARR. Most studies reported that maxillary teeth were more prone to resorption than were mandibular teeth. The most frequently affected teeth, in order of severity, were the maxillary laterals, maxillary centrals, mandibular incisors, distal root of mandibular first molars, mandibular second premolars, and maxillary second premolars. This was comparable to the findings of Sameshima and Sinclair[3], who found the worst resorption in maxillary lateral incisors, followed by maxillary central incisors, maxillary canines, mandibular canines, mandibular central incisors, and mandibular lateral incisors. Moreover, hypodontia or partial anodontia puts existing teeth at risk of root resorption.[40]

b. Meachanical factors

1. Types of appliance: Numerous studies comparing alternative treatment systems have been conducting over the years. Previously, few studies could demonstrate a clear advantage of one system over the others. Although several studies have been published in the last decade, it is still impossible to identify a system that can reduce or eliminate the orthodontically-induced root resorption phenomenon.[56]

Linge and Linge[5] compared average change of root length in 81 patients treated with removable appliances and 638 patients treated with fixed appliances. Fixed appliances caused significantly more EARR than removable appliances.

Brin et al[57] studied 138 children with Class II division 1 malocclusion. They compared EARR on three groups of patients treated as follows: Group 1, one phase with fixed appliances only; Group 2, one phase with headgear followed by one phase with fixed appliances; and Group 3, one phase with the bionator, a removable appliance for growth modification, followed by one phase with fixed appliances. These three groups were similar in age, sex, and malocclusion severity. They found, that early growth modification, using headgear or bionator, to reduce the severity of overjet in Class II malocclusion, can reduce EARR.

2. Orthodontic movement type: Intrusion is the most detrimental type of orthodontic movement in causing EARR.[58, 59] Recently, in an intra-individual study, Han et al[60] compared root resorption after application of continuous intrusive and extrusive forces. They assessed root resorption by using electron microscopy in nine patients whose maxillary first premolars were randomly intruded or extruded with a continuous force of 100 grams for eight weeks. They found that intrusion of teeth caused about four times more root resorption than did extrusion.

Parker and Harris[61] used stepwise multivariate linear regression analysis to determine which tooth movements and skeletodental relationships were the most predictive factors for the development of EARR. Their sample consisted of 110 adolescents with similar pretreatment malocclusions (Class I crowded or bimaxillary protrusive) and similar treatments (extraction of four first premolars) provided by experienced private practitioners. Incisor intrusion when applied together with increased lingual root torque was the strongest predictor of EARR. In contrast, distal bodily retraction, extrusion, or lingual crown tipping were not strongly correlated with EARR.

Tipping, torque, bodily movement and palatal expansion are also implicated in EARR. The risk of EARR due to bodily movement is less than that due to tipping movement because the stress distribution along the root during bodily movement is less than the stress concentration at the apex resulting from tipping.[9]

3. Orthodontic force: Harry and Sims,[62] using electron microscopy, found that increased stress causes increased root resorption. High levels of force tend to increase the damaged to the periodontal ligament, leading to more extensive orthodontically-induced root resorption.

Chan and Darendeliler[63] evaluated the effects of orthodontic force magnitude on root resorption craters, using volumetric measurements. They used a scanning electron microscope to investigate 36 extracted human premolar teeth to which were applied either light-force (25g) or heavy-force (225g) for 28 days before extraction. They found more resorption by volume in the heavy-force group than in the light-force group or in the controls. Although more resorption was recorded in the light-force group than in the control group, the difference in the amount of resorption between the light-force and control groups was not statistically significant. Therefore, they suggested that high-pressure zones might be more susceptible than light-pressure zones to external root resorption after 28 days of force application.

4. Treatment duration: There is controversy among reports regarding whether or not treatment duration is associated with EARR. Baumrind et al[39] and Levander et al [64] found treatment duration was an associated factor in the development of EARR after orthodontic treatment.

Baumrind et al[39] analyzed the relationship, in orthodontically treated adults, between upper central incisor displacement on lateral cehalograms and EARR measured on anterior periapical radiographs. They found the increased length of treatment time was positively associated with increased EARR.

Levander et al[64] studied 68 orthodontically treated patients with aplasia. The degree of EARR was assessed before and after treatment from intra-oral radiographs of maxillary incisors using a scale of 0-4. Total treatment time was divided into groups; 1 year, 2 years and more than 2 years. They found statistical differences between the groups. They concluded that the greater the total orthodontic treatment duration, the greater the EARR.

However, Linge and Linge[5] found no significant correlation between treatment duration and EARR in maxillary incisors. They stated that appliances may be present for long periods without creating pressure on the teeth. Therefore, treatment time has not been detected as a predictor of resorption.

5. Intermaxillary elastics: Some studies agreed that jiggling and movement caused by the application of intermaxillary elastics increased the risk of orthodontically-induced EARR.[4, 5]

Mirabella and Artun[4] found that the length of time that anterior elastics and Class II elastics were worn was associated with EARR in canines. They concluded that the use of elastic forces may increase the risk of EARR only on the teeth that support the elastics.

Linge and Linge[5] compared average change of root lengths of 201 patients with Class II elastics and 518 patients without Class II elastics. They found significantly more EARR on the side where elastics were used, and suggested that jiggling forces as a result of function combined with elastics are responsible for incisor EARR.

6. History of Extraction: Baumrind et al[39] found no difference in the extent of EARR in patients treated with or without extractions. They compared 38 non-extraction cases and 35 premolar extraction cases and found no statistical difference in the extent of EARR in patients treated with or without extractions.

However, Mohandesan et al[65] found significant more EARR in an extraction group than in a non-extraction group. They studied 151 maxillary incisor teeth of 40 patients, aged 12–22 years, with different malocclusions. The root resorption measurement was performed on periapical radiographs correcting for image distortion.

7. LeFort I osteotomy: Kaley and Phillip[66] concluded that LeFort I Osteotomy was a risk factor for EARR. This study used a case-control design; the characteristics of 21 patients with severe resorption were compared to those of randomly selected controls from the case series. Risk indicators for resorption, related to treatment procedures were the maxillary incisor roots when against the palatal cortical plate and maxillary surgery.

But in contrast, Mirabella and Artun[4] found no statistical correlation between root resorption and LeFort I Osteotomy. They explained that the root resorption may be due to the amount of tooth movement rather than pressure from the cortical plate.

Some previous studies found that periods of ischemia and hyperemia after LeFort I osteotomy which is likely reasons for observed pulpal changes in long term. Ramsay et al[67] analyze the effect of Le Fort I osteotomy on pulpal circulation. They used a laser Doppler flowmeter to measure pulpal blood flow of maxillary right and left central incisors and a randomly selected mandibular canine in 14 volunteers prior to surgery and at various intervals during the 6 months following surgery. Their result showed a significant reduction in vascular supply at the final observation. Beside that, Ellingsen and Artun[68] studied in 93 patients, 21.9 to 63.9 years of age (mean 38.5±9.4 years) who consented to participate in a follow-up study ranging from 4.7 to 15.3 years (mean 8.9±2.9years) after surgery. LeFort I osteotomy was performed on 42 patients and bilateral sagittal split osteotomies on 76 patients. They found periods of ischemia and hyperemia were the factors that caused the pulpal changes in the long term, after LeFort I osteotomy. However, these are difficult to explain any association between LeFort I osteotomy and EARR.

V. Diagnostic aids for detection of root resorption

There are many diagnostic aids for detection of EARR. Brezniak and Wasserstein[8] reviewed published research reports on EARR in their comprehensive review. The analysis of histology, scanning electron microscopy (SEM), radiographs, including periapical, panoramic and lateral cephalometric radiographs, have been used as diagnostic tools for detecting EARR. Later, more modern imaging technologies, such as computed tomography (CT), are currently used for research. Although there are many advantages of CT compared to conventional radiographs, CT is a costly procedure, both financially and in terms of radiation dose, and demands special equipment.

Recently, Mah and Prasad[69] compared the levels of Dentine Phosphoprotein (DPP) in the gingival crevicular fluid (GCF) in permanent central incisors of untreated subjects (control group), with the levels in primary second molars with half of the root resorbed (positive control group), and with those in permanent central incisors with mild root resorption of patients undergoing active orthodontic treatment (study group). They found that the study group showed elevated levels of DPP in the GCF relative to the control group but the elevation was less than that of the positive control group. They found that root resorption could be studied using the measurement of DPP in GCF as a biochemical assay.

However, although periapical radiographs are commonly used for detecting EARR, it is difficult to assess radiographically the amount of buccal and lingual root resorption. Despite its limitations, the periapical parelling radiographic technique provides the most favorable benefit to risk ratio in detecting and evaluating the degree of apical root material loss. It provides the most appropriate information with the least irradiation to the patients. Moreover, it provides less distortion and superimposition errors compared with the panoramic radiograph or the cephalogram.[70, 71]

Leach et al[70] have summarized the radiographic techniques commonly used in Orthodontics, with particular reference to measuring EARR. They illustrated the limitations of three commonly used radiographic views, the upper standard occlusal, dental panoramic and lateral cephalometric skull radiographs. They suggested that the paralleling periapical technique should be used when serial assessments of EARR are made.

VI. Methods to accurately measure root length

Brezniak et al[72, 73] compared three methods to accurately measure root length. (1) The root length changes were calculated simply by subtracting the measured radiographic post-treatment tooth length from the pre-treatment one length. (2) The rule-of- three formula was used to calculate the root length change. In this formula, it is assumed that during orthodontic treatment the crown length does not change. (3) Radiographic adjustment was used for crown length and root length in pre-treatment and post-treatment radiographs. The results revealed that the rule-of- three formula was the best method for EARR measurement on periapical radiographs. The angular changes between the tooth and film affect the measured tooth length. Therefore, the mid point between the mesial CEJ point and the distal CEJ point is the best reference point for measuring root length, especially when a correction factor is used, assuming that during orthodontic treatment the crown length does not change. Therefore, the ratio between the initial crown length (C1) and the final crown length (C2) determines the enlargement factor. If no change occurs in the root length during treatment, the ratio between the initial root length (R1) and the final root length (R2) should be equal to the C1/C2 ratio. If, during treatment, the root is shortened, the amount of EARR is R1-R2 (C1/C2).

This formula was first introduced by Linge and Linge,[5] and has later been used by Blake et al[74] and Mavragani et al[75]