Dentistry and Oral Health

Chapter 1

Tooth Resorption in Various Clinical Situations

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Abstract

Nowadays the root resorptions are studied in depth with the aim to preserve the dental structures. Resorption procedure can be inflammatory or replaceable, caused by trauma, inducing in this way the tooth movement, periapical diseases, or discolored teeth whitening. The root resorptions are clinically asymptomatic, not inducing pulp, periapical or periodontal changes. The resorption mechanism occurs when structures as cementoblasts and epithelial cell rests of Malassez (ERM), which protect teeth from bone remodeling, are eliminated. There are two main forms of root resorptions; external root resorption (ERR) and internal root resorption (IRR). The most complicated situation, IRR, involves the pulp disease, characterizing by dentin loss as a result of clastic cells action stimulating the pulp inflammation. The IRR treatment bases in removing the granulation tissue and blood supply of the clastic cells. The other situation, ERR, is an iatrogenic consequence of orthodontic treatment, but it can be encountered even in the absence of orthodontic appliances. Orthodontic therapy generally causes root shortening and disrupts the integrity of dental arch. It is very important to know the root resorption risk factors and interfering to reduce this kind of situation. In Orthodontics, root resorption is related to fixed appliance duration, tooth structure, type of orthodontic forces or tooth movements, etc.

Keywords: Root resorption; Internal and External resorptions; Endodontic Treatments; Surgical Techniques; Orthodontic forces; Tooth movements.

1. Introduction

The classification of tooth resorption is divided according to the inflammatory or by replacement mechanisms. The first one [1], proceeds by inflammatory mediators that stimulate bone multicellular units (BMU), where clastic cells gradually resorb the dentin surface, where the latter is free of cementoblasts and odontoblasts, eliminated as a result of the resorption procedure. Cementoblasts cover the root surface, and the Sharpey collagen fibers are inserted among them. Teeth are very close to the bone and separated by the periodontal ligament, which has a thickness ranging from 0.2 to 0.4 mm. Bone is constantly under remodeling thanks to the stimulation of local and systemic factors; depending on the receptors that osteoblasts and macrophages have on their membranes managing in this manner the osteoclast activities. The mineralized root surface will be exposed if any factor will be active in the place where cementoblasts are found and that removes them from the surface; the bone cells, which are very close, will promote root resorption, even temporary. Mostly, root resorption has local causes that eliminate cementoblasts from root surface, and no systemic cause known about this effect. This situation is observed more in orthodontic movements during activation periods. It is very important to identify and eliminate the resorption causes, stopping in this manner their progression. The second one [1], proceeds by systemic and local bone tissue mediators that regulate the remodeling or circulation process. This situation happens as a result of epithelial cell rests of Malassez (ERM) death leading to dental ankyloses. Naturally, each new bone layer deposited on the alveolar periodontal surface is closer to the tooth, at a mean thickness of 0.25 mm, where dental ankylosis can happen. Cementoblasts and osteoblasts interact here forming areas of fused cement and bone. However, that does not occur due to the presence of ERM, a net made up of cords that are 20 cells long and 4 to 8 cells wide and that form a structure in the periodontal ligament around the tooth root. Dental ankylosis happens almost when ERM are eliminated, usually by dental trauma, which may range from light trauma, such as concussion, to more severe trauma, such as avulsion. Bone remodeling affects mineralized dental tissues, which are gradually resorbed and replaced with bone, which explains the replacement resorption. This phenomenon can be seen even in that clinical case, where tooth remains unerupted for long time, severe periodontal ligament atrophy may facilitate its ankylosis development. Dental resorption is a condition related to a physiologic or a pathologic process leading in dentin, cementum, and/or bone loss [2]. This condition can happen after mechanical, chemical or thermal injuries. In physiologic resorption, dental pulp remains vital, where this situation is not pathologic (**Figure 1 a&b**); this mechanism occurs in deciduous teeth [3]. It is a discontinuous procedure, where have been found multinucleate odontoclasts (tooth resorbing cells) [4].

(a) (b)

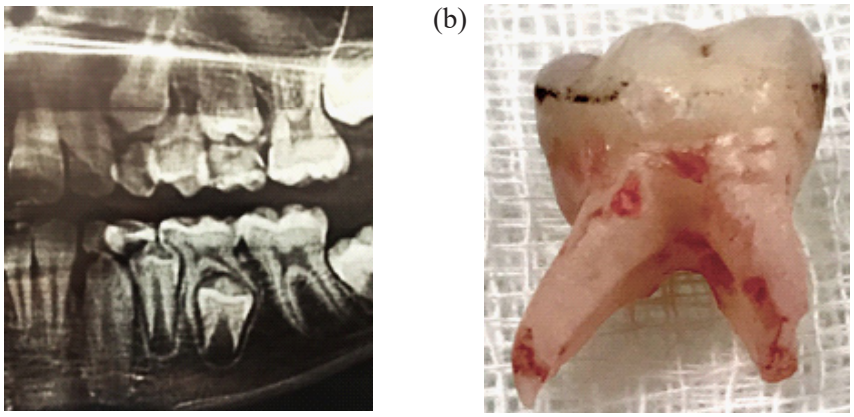

Figure 1: **(a)** Dental physiologic resorption in a 10 years old boy; **(b)** Extracted inferior deciduous molar with apical physiologic resorption.

In generally, tooth resorption can be classified as external root resorptions (ERR) or internal root resorptions (IRR).

IRR concerns in permanent teeth, as an inflammatory process including the pulp space bringing dentin loss and possible cementum invasion [1, 5]. The most difficult thing in these lesions is their undertaken diagnosis, where conventional X-ray is often inadequate. It has been demonstrated that in early stages, when the lesions are small, internal root radiolucencies are not so well detectable on radiographs **(Figure 2)**, because of this 2-dimensional (OPT) method limitations [5]. Over the time, the diagnostic technology has evolved, spotting the cone beam computerized tomography (CBCT) as a powerful tool **(Figure 3)**, which allows an earlier and more accurate diagnosis of dental resorptions [6].

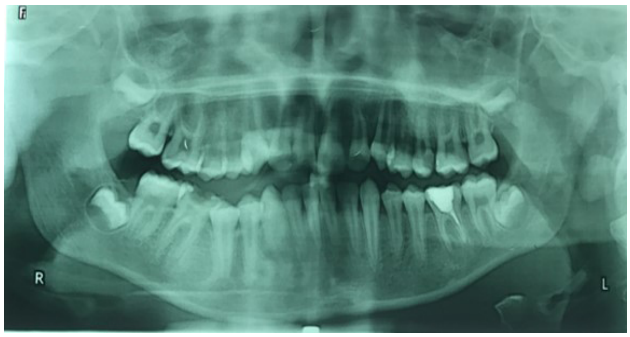

Figure 2: Two-dimensional radiograph with limitations

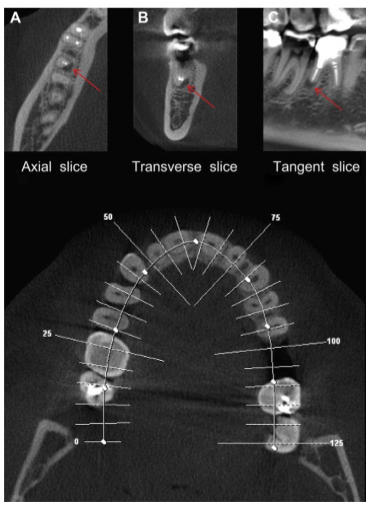

Figure 3: CBCT tool for an accurate diagnosis of apical resorption (Estrela et al. 2009)

These lesions have the necessity to be treated with materials inducing healing and remineralization, preserving in this way the teeth [7, 8].

The other concerned situation, ERR, is a posttraumatic reaction, in some cases as a consequence of orthodontic movement, orthognathic surgery, periodontal treatment, or devital teeth whitening, etc. It is noted that, immunopathological response can play a principal a role in the resorption etiopathogenesis [9].

1.1. General considerations of ERR

ERR is classified according to clinical and histopathological characteristics as superficial external root resorption, inflammatory external root resorption and resorption by replacement, subdivided furthermore into cervical or apical forms [9]. This clinical framework is usually diagnosed by X-rays [10], if there isn't any pain, percussion sensitivity or mobility absence. Its radiographic view represents a radiolucent area with irregular margins along the root length localized in different places.

1.2. Resorption mechanisms

According to Júnior CP [11], demonstrated that resorption of mineralized dental tissues occurs when absorptive cells gain access to mineralized tissues, after partial removal of cementoblast layer and cementoid tissue. The resorption was associated with the damage or partial layer destruction, which covered the precementum. Damaging this layer, by a local physical agent, odontoclasts come to the play interfering in tooth mineralized tissues. It initiates with odontoclasts together with macrophages, formatting a bone remodeling unit, managed by osteoblasts, which have receptors for resorption mediators. Generally, it occurs in the nearest site to a hyalinized area of the periodontal ligament (sterile necrotic zone) with compression of it and its components, reducing the nutritional support and formation of a degenerated and acellular zone (hyalinized area), especially during orthodontic treatment [11]. There are three stages of these hyalinized areas: degeneration, elimination of destroyed tissues and repair [12]. During the destroyed tissues phase, is a direct relationship with the root resorption process, periodontal membrane is narrowed and osteoclast activity removes bone tissue, with the objective to reduce the pressure and allowing vascularization, and after this, a cellular activity happens in the area that eliminates the destroyed tissues and enables repair [11].

Hard tissues resorption process happens as a result of interactions between clast cells with local regulatory factors, cytokines, inflammatory cells, and systemic factors, mostly represented by hormones, where sexual steroids and parathormone play an essential role. It has been shown that bone remodeling is controlled by parathormone, calcitonin, vitamin D and sexual steroids [12]. Resorption occurs in response to mechanical or chemical cells stimulation in periodontal ligament and characterized by prostaglandin (E-1) synthesis, together

with an increase in cyclic adenosine monophosphate. The parathyroid hormones, calcitonin, neurotransmitters and cytokines or monokines regulate this process [13], while local factors synthesize the cells including the insulin-like growth factors (IGF I and II), transforming growth factor beta (TGF-β), fibroblast growth factors, platelet derived growth factor (PDGF), bone morphogenetic proteins, cytokines (interleukins IL-1 and IL-6), tumor necrosis factor (TNF), colony stimulating factors (CSF) and products of arachidonic acid such as the prostaglandins [12].

There is no evidence about a genetic predisposition among patients who have tooth resorptions. The cells responsible for the resorption, enzymes released or mediators that are synthesized and released are all determined by genes that control cell functions, but they are not caused by a specific gene for dental resorptions [14].

1.3. Causes of root resorptions

In cases of inflammatory root resorptions, the causes remove cementoblasts from the surface, as described below:

- *Chronic periapical lesions:* toxic microbial products, resulting from their metabolism, are released in the periapical environment or reaching the apical root surface through dentinal tubules.

- *Dental trauma:* may break vessels and may put the tooth in contact with the alveolar bone surface, such as in accidents, during leisure activities, car crashes, violence or other incidents. Cases of trauma due to the action of laryngoscopes over teeth during intubation for general anesthesia.

- *Long periods of occlusal trauma*: may lead to the cementoblasts death inducing inflammatory root resorptions.

- *Unerupted teeth:* may compress blood vessels of neighboring teeth when they come closer with the help of eruptive forces. In situation, where is a tooth unerupted for a long time, excessive periodontal ligament atrophy may create a dental ankylosis and as a consequent the replacement resorption.

- *Orthodontic forces:* may close the blood vessels lumen, and nutrition cannot reach them. From excessive forces can occur the cementoblasts elimination from the root surface via compression.

In cases of replacement resorption, the causative factors remove the ERM from the periodontal ligament. The main and practically single cause that eliminates this ligament component is dental trauma, which may range from concussion, in its milder form, to avulsion

and replantation, in its more severe forms. Commonly, inflammatory or replacement root resorptions do not cause pain no matter how close to the pulp they are. In case of a painful sensation in teeth with any type of these resorptions, another cause should look for another pain explanation, because root resorption is asymptomatic and silent biological process

In IRR cases, root resorption does not induce pulp necrosis even it is very close to the pulp, or in the pulp structure. Tooth resorption does not release products that are toxic to cells, even when the mineralized tissues resorption has the only purpose of deconstructing them with the aim to recycle their mineral and non-mineral contents, which are reused as ions, amino acids and peptides.

2. Prognosis

The most important therapeutic principle about inflammatory root resorptions is the removal of causes. Especially, when inflammation and cell stress are eliminated from the resorption area, bone remodeling units and their osteoclasts are demobilized and leave the root surface; consequently, mediators disappear. In that area, pH goes back to neutral, and new cementoblasts are formed and colonize the root surface another time in few days. After all, new cement is formed, with collagen fibers reinsertion into the new cementoblast layer.

In the case of bacterial contamination via root canal, adequate endodontic treatment removes the cause, and inflammatory resorption is repaired. When the cause is an orthodontic force, it is necessary the appliance deactivation and forces dissipation in order to stop this kind of process. In the other part, if the possible cause is eliminated and inflammatory root resorption isn’t stopped, perhaps the true cause has not been eliminated.

Replacement resorption leads to dental ankylosis and, after it is established, the process cannot be stopped. At the moment that ankylosis is discovered before it progresses into replacement resorption, luxation followed by extrusion, in most of the cases can restore the periodontal ligament or the bone-tooth connection. We can say that, inflammatory root resorption can be controlled and treated, because of its good prognosis; meanwhile in the replacement resorption, having a poor prognosis, the tooth can be lose, sooner or later.

2.1. Pathogenesis and histology

IRR is a pathologic phenomenon characterized by the dentin loss as a result of clastic cells action, occurring in pulp inflammation condition, where the clastic cells are brought through blood supply in the pulp chamber. Odontoclasts are morphologically analogous to osteoclasts and have similar enzymatic properties and resorption patterns. They are smaller in size and form than osteoclasts [15]. Odontoclasts have a ruffled border, containing fewer nuclei than osteoclasts, and have smaller or no clear zone. Both of cells have intense tartrate-

resistant acid phosphatase activity. Most of odontoclasts that form lacunae on dentin are multinucleated, having 10 or fewer nuclei. Oligonuclear odontoclasts (cells with fewer than five nuclei) resorb more dentin per nucleus than do cells with a higher number of nuclei [16]. Dental resorption requires injury and stimulation phases, where injury is related to the non-mineralized tissues covering the internal surface of root canal, predentin and odontoblasts layer. The main stimulation factor for IRR is the infection. In the early stage of resorption, tooth is not symptomatic. The resorbing cells origin is pulpal, coming from the apical vital pulp part [17].

3. IRR

3.1. IRR etiology

Different etiologic factors have been proposed for the predentin loss and trauma seems to be the most advocated. In a study including 27 patients, trauma is the most common etiological factor (43%), followed by carious lesions (25%) [18]. Pulp infection through bacteria causes the pulp chamber walls colonization by macrophage-like cells. The attachment and spreading of such cells is the primary prerequisite for initiation of root resorption [19]. From these data, trauma and pulpal infection are principal contributory factors in the IRR initiation [20].

3.2. IRR prevalence

Generally, IRR is considered rare, but its frequency is not well known. The diffusion of IRR has been estimated to be between 0.01% and 55%, depending on pulp inflammation in teeth affected by pulpitis and pulp necrosis [21]. The complete progression of pulp necrosis stops the resorption growth [22].

3.3. IRR clinic and radiographic diagnosis

IRR, being asymptomatic is usually evident through full oral radiograph. In some cases, pain depends on pulp condition or root perforation resulting in a periodontal lesion [18]. A clinical aspect of pink spot can be observed, when IRR is localized in the coronal part, where this color is related to the highly vascularized connective tissue adjacent resorbing cells. When the pulp comes in necrosis, this color turns grey/dark grey [23].Root perforation is usually followed by the development of a sinus tract, which confirms the presence of an infection of root canal, mostly by Gram-negative, strict anaerobes species [24].

The IRR x-ray appearance is characterized by an oval shape enlargement within the pulp chamber or the root canal. Except this type of radiograph, CBCT has been successfully used to evaluate the true nature and severity of resorption lesions, where in this manner the clinician can manage better the defect [25]. CBCT provides a 3-dimensional appreciation of the resorption lesion with axial, coronal, parasagittal views of the anatomy. In cross-sectional

views, the size and the location of resorption are clearly determined with high sensitivity and an excellent specificity [6].

CBCT gives information about the following:

- Location, size, and shape of the lesion,

- Presence of root perforations,

- Root wall thickness,

- Presence of an apical bone lesion,

- Localization of anatomical structures: maxillary sinus, mental foramen, and inferior alveolar nerve.

All of the above points validate the differential diagnosis by ERR and allow the prognosis evaluation of the tooth if the lesion is variable in treatment.

3.4. IRR therapy

Taking the right decision about the resorption therapy, different key points have to be considered during clinical examination and CBCT:

- monitoring infectious signs and symptoms present or absent,

- orthograde root canal treatment in: complete root canal filling with gutta-percha on non-perforated lesions; combined gutta-percha in root canal and mineral trioxide aggregate (MTA) filling in perforation area; complete filling with a bioactive material (MTA or Biodentine) on apical perforated lesions located in a short root length,

- Retrograde apical treatment,

- Extraction and implants insertion: this non-conservative treatment is indicated if the tooth is too weak and not resistant to be treated or maintained,

- Root canal treatment remains the treatment of choice of IRR removing the granulation tissue and blood supply of the clastic cells.

IRR is a situation that represents difficulties in instrumentation and filling. In these cases, the access cavity preparation must be as conservative as possible to preserve tooth structure and avoid further weakness of resorbed tooth. Several times, direct mechanical instrumentations are impossible to be used, because of the shape of resorptive defects [20]. The apex locator application to measure the working-length is not allowed in case of resorptive perforation. A great importance must be emphasized on chemical dissolution of vital and necrotic pulp tissue

via sodium hypochlorite, where can be used the ultrasonic devices to activate and penetrate the irrigant to all the areas of root canal system [21]. Non-traumatic plastic tips of EndoActivator are particularly indicated to achieve a complete chemomechanical debridement of root canal. The clinical use of calcium hydroxide as an interappointment medication, is necessary for its disinfection effects, helps to control the bleeding, and necrotizes residual pulp tissue. About the final/definitive root canal filling, the material is better to be flowable to seal the resorptive defect. Beside this property, for perforated root wall(s), MTA is the material of choice to seal the perforation, for its biocompatible, bioactive, and well tolerated by periradicular tissues [26]. If MTA starts to harden during its elaboration, the working time can be modulated by adding water.

3.5. IRR surgical treatment

Surgical treatment should be performed, when it is not possible to get access to the lesion through the root canal, after the orthograde treatment (or retreatment) performed, firstly the coronal part of canal has to be filled. This approach allows getting direct access to the lesion and doing a mechanical cleaning of the resorbed defect. The principal guidelines of endodontic surgery procedures must be respected [27]. After performing the local anesthesia, a mucoperiosteal flap is applied; meanwhile the cortical bone is removed to provide access to the root area. The soft tissue lesion is curetted and the intraradicular dentin cavity is prepared, on its external surface a filling material is necessary to be placed and smoothed. In the end of this step, the flap is repositioned and sutured.

4. Orthodontic consequences

The inflammatory root resorption is one of the most complication induced by orthodontic treatment, diagnosed even in patients that haven’t undergone orthodontic treatment. This kind of root resorption differs from other resorptions [28, 29], characterized by its sterile condition, local inflammatory process and has all characteristic of inflammatory symptoms [30]. Root shortening may be induced by these etiological factors, such as trauma, infectious inflammation of periapical tissues or periodontal diseases [28]. Mostly, in asymptomatic cases we can observe the root resorption process until destruction of solid dental structure only during radiological examination [30, 31]. Root resorption is considered clinically important when 1-2 mm (1/4) of the root length is lost [32] **(Figure 4).**

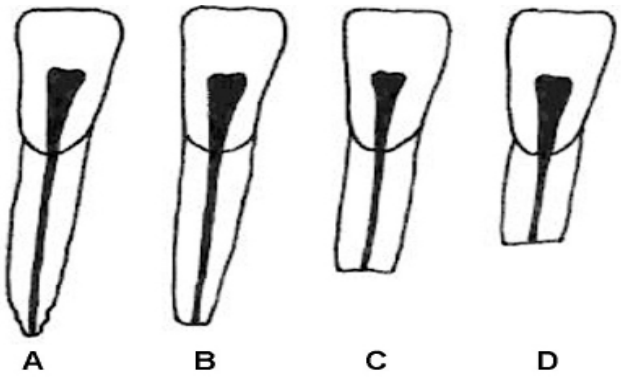

Figure 4: The root resorption degrees:
A. Irregular root contour
B. Apical root resorption is less than 2 mm
C. Apical root resorption is from 2 mm to 1/3 of initial root length
D. Apical root resorption is more than 1/3 of initial root length

Severe root resorption during orthodontic treatment (more than 1⁄4 of the root length, >5 mm) occurs very rarely, just in 1-5 % of patients [33]. When are better-known the risk factors about root resorption could be a great help for orthodontist to assess a patient upon planning orthodontic treatment and to choose the best method for treatment.

4.1. Root resorption degrees

There are three severity degrees of root resorption as follow:

1. Cementum or surface resorption, when outer cementum layer is resorbed, which regenerates or remodels later; this process is similar to trabecular bone remodeling [29].

2. Dentin resorption with repair, when cementum and outer dentin layer are resorbed; resorption is irreversible because only cementum regenerates [29].

3. Surrounding apical root resorption, when hard apical root tissues fully resorbed and root shortening are observed. In cases where root apical tissues under cementum are lost, root tissues do not regenerate. Repair of outer surface occurs in the cementum layer [29].

4.2. Root resorption mechanism

According to Brudvik and Rygh, inflammatory root resorption induced by orthodontic treatment is a part of hyaline zone elimination [29]. Occurrence of root resorption can be induced by strong forces through orthodontic treatment and hyalinisation of periodontal ligaments by increased activity of cementoclasts and osteoclasts [31]. During tooth movement, areas of compression (where osteoclasts are in action inducing bone resorption) and areas of tension (where osteoblasts are active inducing bone deposition) are formed. In this way, the tooth moves towards the side of bone resorption. An imbalance between bone resorption and deposition, losing protective characteristics of cementum may contribute to the cementoclasts/ osteoclasts resorbing root areas [32]. When it is formed the hyaline zone, tooth movement

will stop. At the moment of periodontal ligament regeneration, hyaline zone is removed by mononucleus cells similar to macrophages, and by multinucleus gigantic cells the tooth starts to move again.

During removal of hyaline zone an outer tooth root surface consists in a damage of cementoblasts layer, exposing the underlying highly dense mineralized cementum. It is possible that a force during orthodontic treatment may directly damage outer root surface. Tooth root surface under the hyaline zone can be resorbed just after few days, when in the periphery a repair process is happening. On the literature databases, it can be stated that the resorption process is completed after removal of the hyaline zone, and/or when orthodontic force decreases [29, 32].

4.3. Risk factors

There are several risk factors that can initiate and induce root resorption during orthodontic treatment. All these factors can be distributed to biological, mechanical and combined biological and mechanical factors and other circumstances.

4.3.1. Biological factors

Individual susceptibility is a main factor determining root resorption, which can manifest in both deciduous and permanent teeth [34]. Among orthodontic patients susceptible to root resorption, this may be a consequence of a systemic or innate predisposition to the appearance of resorption. It is assumed that in this situation, severe root resorption may occur without any evident reason [30].

Genetics: Predisposition to root resorption may be autosomal dominant, autosomal recessive, or hereditary determined by a few genes. It is supposed that, genetic predisposition is very important for root resorption occurrence [34]. Genetic factors account for at least 50 % of the variation in root resorption [30].

Systemic factors: Owman-Moll and Kurol have established that allergic patients are in the group of increased risk for root resorption [28]. It was established that lack of estrogens may induce quick orthodontic tooth movement, and calcitonin inhibits activity of odontoclasts [30]. Conditions like astma also appear to indicate a greater risk for a large amount of apical root resorption [35].

Nutrition: Several authors have shown that root resorption occurred even in animals lacking calcium and vitamin D in their foods [34].

Chronological age: Periodontal membrane becomes narrower and less vascularized, aplastic; meanwhile alveolar bone occurs denser, less vascularized and aplastic, and cementum becomes

wider with age. Through these changes adults show higher susceptibility to root resorption [34]. According to some authors [31, 36], when the patient is older than 11 years, risk for root resorption increases.

Dental age: Rosenberg has stated that teeth with incomplete root formation undergo less root resorption than those with completely formed roots, where incompletely formed roots reach their normal root length [34]. Brezniak declared that if teeth roots are not completely formed in the beginning of orthodontic treatment, they further develop during treatment, but remain shorter [28]. It has been established that ortodontically treated teeth lose averagely 0,5 mm of the root length [34].

Sex: No significant relationship between sex and root resorption has found by performed studies [34].

Ethnic group: Root resorption more rarely occurs in Asians than in white, Caucasian or Hispanic patients [30, 37].

Before orthodontic treatment: Root resorption that existed prior to orthodontic treatment increases risk for root resorption during orthodontic treatment [34].

Habits: Such as bruxism, nail biting, tongue thrust associated with open bite and increased tongue pressures are related to increased root resorption [30, 37].

Anomalies of position and number of teeth: Apparently, hypodontia increases risk of root resorption; impacted teeth may also induce occurrence of root resorption [28]. Third molars are the most commonly impacted teeth, which may cause root resorption of the second molar [38]. Maxillary impacted canine can induce root resorption of the incisors and first premolars [39] **(Figure 5)**. It is recommended annual palpation of the canine regions, dental radiographs before 10 years of age and early extraction of deciduous canines [40].

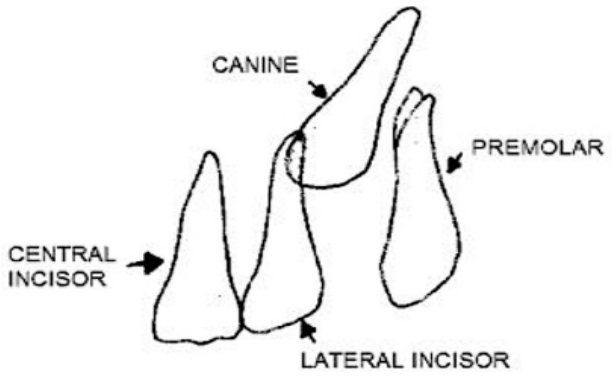

Figure 5: Erupting canine teeth may induce root resorption of the lateral incisors and first premolars

Tooth structure: Root resorption most often occurs in the apical part of the root; forces there are concentrated at the root apex, because orthodontic tooth movement is never entirely translatory and the fulcrum is usually occlusal to the apical part of the root. Periodontal ligaments are

situated in different directions of tooth root apical parts; the apical third of the root is covered with cellular cementum, whereas the coronary third is covered by non-cellular cementum. Active cellular cementum depends on blood circulation; thus periapical cementum is more friable and easily injured in trauma cases [30]. Levander and Malmgren divide root forms to normal, short, blunt, dilacerated and pipette-shaped [33] **(Figure 6).**

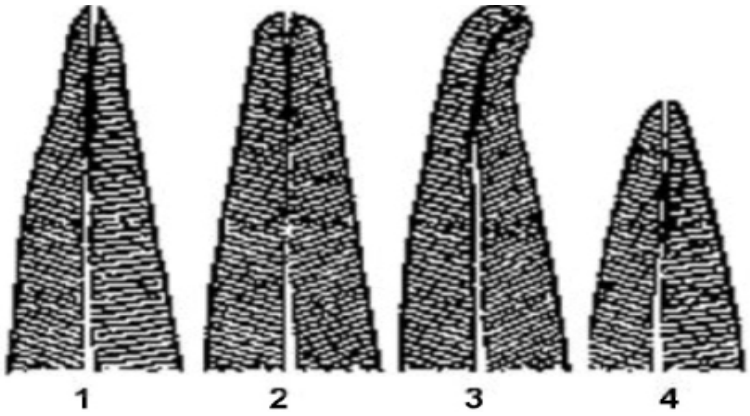

Figure 6: Root forms: **(1)** – pipette-shaped root, **(2)** blunt root, **(3)** dilacerated root, **(4)** short root

Most authors have shown that roots with abnormal shape have a higher susceptibility to root resorption [41]. According to the data of Sameshima and Sinclaire, normal and blunt tooth roots are resorbed less [33].

Pipette-shaped roots are the most susceptible form to root resorption [34]. Short roots have a greater risk for root resorption than average of long roots [31]. It has found that small roots resorb almost twice more than other root forms [33]. There are controversial data about initial length of tooth root and the amount of tooth root resorption. There is an opinion that longer roots are more likely to be resorbed than shorter ones because longer roots are displaced farther for equal torque [36]. Tooth with longer roots need stronger forces to be moved and that the actual displacement of the root apex is greater during tipping or torqueing movements [30, 42]. It was established that a normal root form of central incisors and wide roots are preventive factors of these teeth roots, decreasing risk of root resorption [43]. Slight increased root resorption is characteristic for the tooth with narrower roots [42].

Dental trauma may cause root resorption to the teeth without orthodontic treatment. Orthodontically moved traumatized teeth with previous root resorption are more sensitive to further loss of root material [34]. The teeth can be treated orthodontically three months after the tooth transplantation or replantation. According to the research data, a completely assimilated transplanted tooth reacts to orthodontic force as a normal tooth [28]. More often root resorption is characteristic during orthodontic treatment, when the tooth is endodontically treated before. The hypothesis was raised that endodontically treated teeth are more resistant to root resorption because of increased hardness and density of dentin [33, 34]. Qualitative endodontic treatment of teeth is very important. When the root canal filling reaches the root apex, resorption doesn't start [33]. Alveolar bone density on root resorption is assessed controversially. A part of the

studies has established that the denser is alveolar bone, the more root resorption occurs during orthodontic treatment.

Strong continuous force affecting alveolar bone of less density causes the same root resorption as a mild continuous force affecting alveolar bone of higher density [34, 35]. Bone density determines tooth movement rate but has no relation to the extent of root resorption [34]. Cementum is harder than alveolar bone and more mineralized, more fibers of periodontal ligaments are inserted into cementum than in alveolar bone, thus osteoclasts have less possibility to injure the cementum layer and inducing root resorption [44]. Correlation between malocclusion and tooth root resorption is assessed No orthodontic malocclusion is immune to root resorption [28]. Upon studying tooth root resorption occurring during treatment of Angle II class orthodontic malocclusion, it was established severe (2 mm) maxillary incisor root resorption in 12.4% of children [45]. According to the research data of Kaley and Phillips, the patients with Angle III class orthodontic malocclusion experience increased root resorption [36]. Relationship between the change in overjet and severity of root resorption was observed. The greater the overjet during the orthodontic treatment, the greater the root resorption for maxillary anterior teeth, because greater tooth movement is necessary in order to decrease overjet [36, 37]. Malocclusion in vertical plane do not influence occurrence of increased root resorption. Increased overbite may correlate with more root resorption of maxillary lateral incisors [37]. It was established that the deeper is overbite, the greater is root resorption of a maxillary permanent first molar distal root and maxillary incisor [30].

Tooth weakness to root resorption: Some teeth are more susceptible to root resorption, other less. Maxillary teeth are more sensitive to root resorption than the mandibular teeth [31, 34, 37] and anterior teeth are more susceptible to root resorption relative to posterior teeth [46, 47]. Other researches have shown that root resorption is more common in mandibular incisors [34]. The most resorbed tooth in the lower arch is the canine, followed by lateral and central incisors [37]. Root resorption of molars and premolars is very low (less than 1mm) [34, 37, 48].

The most resorbed teeth are the maxillary incisors and canines, mandibular incisors, mandibular and maxillary second premolars [34, 37, 43, 44, 48].

4.3.2. Mechanical factors

Orthodontic appliances: Stuteville has stated that forces caused by removable appliances are more harmful for the roots [34]. Brin has studied influence of treatment methods of class Angle II orthodontic malocclusion on teeth roots. The results showed that root resorption was diagnosed more rarely in children who had undergone 2-phase orthodontic treatment, firstly with functional removable appliance and later with fixed appliance, than in children, who had undergone orthodontic treatment with fixed orthodontic appliances only [45].

While assessing the influence of metal and aesthetic brackets on root resorption, it was diagnosed more often in patients treated with aesthetic brackets. This is because treatment with aesthetic brackets lasts longer [33]. Application of an additional upper utility arch for intrusion of maxillary incisors induces root resorption of maxillary central incisors more often than by treating with straight arch [49].

Intermaxillary elastics: Greater root resorption is found on the side of tooth, where elastics are used. Use of Class III elastics increases root resorption of first mandibular molars distal root [34]. Some researchers have established that use of intermaxillary Class II elastics and type of orthodontic arches do not have any influence on occurrence of root resorption [50].

Influence of tooth extraction: Controversially this situation is valued on root resorption. McFadden and VonderAhe failed to find any differences between root resorption in patients treated with or without extraction [7]. Higher root resorption rates (0.43 mm) were established in patients with several extracted teeth than in those, who had not undergone tooth extraction (0.31 mm) [31]. Root resorption develops more often after extraction of four first premolars if compared to the patients with non-extracted teeth or with extracted of just maxillary first premolars [34].

Type of orthodontic tooth movement: Any orthodontic tooth movement may induce root resorption. Most often root resorption is shown after orthodontic intrusion (anchoring of a tooth into an alveolar bone). According to Reitan, the force that distributes along the root during bodily movement is less, which concentrates at the root apex resulting from tipping. Bodily movement induces less risk for root resorption than tooth tipping [34]. Root resorption occurs in cervical and apical part of the root during tipping movement. Middle part of the root is resorbing during bodily tooth movement; this happens because of the shape of periodontal space, which is the thinnest in the middle part of the root [51]. Comparing root resorption after application of the same magnitude continuous intrusion and extrusion forces it is demonstrated that teeth intrusion causes four times more root resorption than extrusion. Deep and extensive resorption areas, situated near the root apex foramen can be observed in the apical part of the intruded tooth root. A superficial and limited resorption cavity around the root apex foramen is characteristic for extruded teeth [52]. Teeth rotation causes only minor injuries of periodontal tissues especially in single-root teeth. Resorption areas during the tooth rotation appear in the medial root third. Horizontal section of the root shows how prominent root zones might generate pressure areas when single-root tooth rotation is performing. The resorbed areas are consistently located at the boundaries between the buccal and distal surfaces as well as lingual and mesial root surfaces [53].

Orthodontic force leads to micro trauma of periodontal ligaments and activation of inflammation related cells [30]. According to some researches there was no root resorption

difference detected while using low and high forces (50 and 200 g) [28]. However Harry and Sims have shown that distribution of resorbed lacunae is directly related to the force magnitude, resorbed lacunae develops more quickly in case of higher forces [34]. According to Schwartz, forces increasing 20-26 g/cm^2, cause periodontal ischemia, which may lead to root resorption [34]. When orthodontic force decreases to less than 20-26 g/cm^2, tooth root resorption stops [51]. Optimal force for orthodontic tooth movement but not causing root resorption should be 7-26 g/cm^2 on root surface area [54]. It was established that intermittent force causes root resorption more rarely than the continuous force; this because the intermittent force protects from formation of hyalinized areas or it allows reorganization of hyalinized periodontal ligaments and restoration of blood circulation at the time, when forces are not active. Continuous force leaves no time to repair of damaged blood vessels and other periodontal tissues and this may lead to higher level of root resorption [51].

4.3.3. Combined biological and mechanical factors

Orthodontic treatment duration: It is considered an important factor that may cause root resorption [54]. Many studies have shown that severity of root resorption is related to duration of orthodontic treatment. Levander and Malmgren have investigated that root resorption after 6 to 9 months of orthodontic treatment was detected in 34% of teeth, meanwhile in the end of orthodontic treatment that lasted 19 months, root resorption increased up to 56% [34, 55]. Goldin has stated that the amount of root resorption during orthodontic treatment is 0.9 mm per year [34]. Results of other studies have shown that root resorption may begin in the early stage of orthodontic treatment; it is especially characteristic to teeth with long, narrow and deviated roots [43].

Patients, whose orthodontic treatment with fixed appliances lasts longer, experience significantly more grade 2 root resorption. Average treatment length for patients without root resorption is 1.5 years and for the patients with severe root resorption – 2.3 years **(Figure 7)** [48].

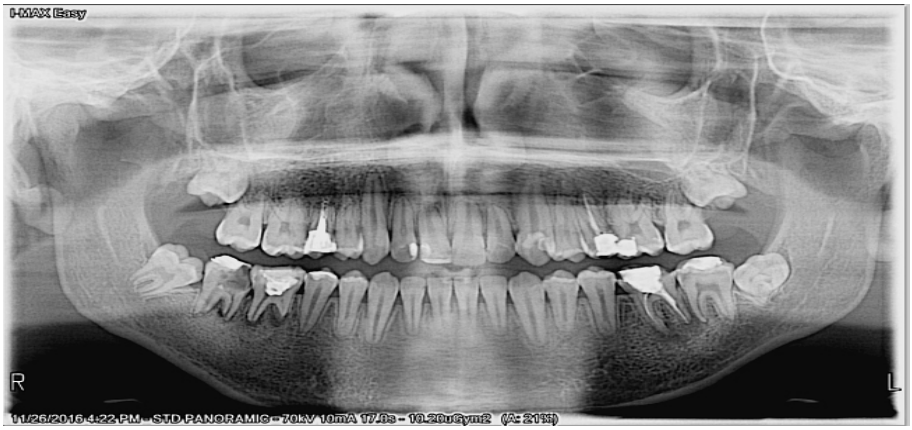

Figure 7: Severe root resorptions in a patient boy after 4 years of fixed orthodontic treatment

Several contemporary studies have found no relation between the length of orthodontic treatment and root resorption [56].

Root resorption detected radiographically during orthodontic treatment: Minor root resorption or an irregular root contour detected 6-9 months from the beginning of orthodontic treatment shows that there is a high risk to further root resorption. If root resorption fails to occur after 6-9 months of orthodontic treatment then no severe root resorption will be in the end of the treatment [34].

Root resorption after removal of appliances: Root resorption associated with orthodontic treatment will stop after completion of active orthodontic treatment. Active root resorption lasts approximately about a week after orthodontic appliance removal. Subsequently, cementum repair lasts 5-6 weeks after removal of orthodontic appliance. Root resorption after removal of orthodontic appliances is mostly related to such causes as occlusal trauma, active retainers or others [34].

4.3.4. Tooth vitality

Tooth vitality and color don't change even at extensive root resorption. Orthodontic movement may cause pulp blood flow disturbances, vacuolization and, in rare cases, pulp necrosis, however it doesn't relate to root resorption [34].

4.3.5. Alveolar bone loss and tooth stability

Marginal bone loss is more harmful than the equivalent amount of root length loss because of root resorption. Results indicate that 4 mm of root resorption translate into 20% total attachment loss and 3 mm apical root loss equals only 1 mm crestal bone loss [57]. Bone loss leads to decrease the stability of a tooth, because major part of periodontal fibers is the crestal area if compared to the surface of root apex. About 0.2-0.5 mm of alveolar bone height is lost during orthodontic treatment [34].

5. Conclusions

- Inflammatory IRR is a particular category of pulp disease, which can be diagnosed by clinical and radiographic examination of teeth in daily practice.

- Today, the diagnosis of IRR is significantly improved by three-dimensional imaging, as CBCT resulted in an improved management of resorptive defects and a better outcome of conservative therapy of teeth with internal resorption.

- Modern endodontic techniques including optical aids, ultrasonic improvement of chemical debridement, and thermoplastic filling techniques should be used during IRR teeth-treatment.

-Alternative materials such as calcium silicate cements offer new opportunities for the rehabilitation of resorbed teeth.

- There is no evidence that root resorption is linked to systemic factors.

- In the future, it would be great and necessary to discover the factors that inhibit the immunopathologic response, without change of oral physiology.

- The early diagnosis can help us to prevent and control dental resorption without tooth loss and safe orthodontic movements/treatments.

- To avoid severe root resorptions, is important to do radiographic controls to all orthodontic patients after 6-9-12 months of orthodontic treatment.

- If before the orthodontic treatment will be evident radiographically a minor root resorption or an irregular tooth root contour, shows that there is a high risk for further root resorption over time/months.

- To not cause root resorption, it is documented that the optimal force for orthodontic tooth movement should be 7-26 g/cm^2 on root surface area.

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