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Maxillary Lateral Incisor Agenesis (MLIA)

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1. Introduction

In the permanent dentition, the type of congenital missing teeth varies from author to author and the demographic and geographic profiles. In Europeans, the mandibular second premolar is the most frequently absent tooth, followed by the maxillary lateral incisor and the maxillary second premolars (Bergstrom, 1977; Rolling, 1980; O’Dowling & McNamara, 1990; Aasheim & Øgaard, 1993; Nordgarden et al., 2002). In the Malaysian (Nick-Hussein & Majid, 1996), Israeli (Chosack et al., 1975) and American populations (Muller et al., 1970), the most frequently missing teeth are the maxillary lateral incisors. Focusing on MLIA the prevalence varies between 0.8 and 4.25% (Horowitz, 1966; Muller et al., 1970; Thilander & Myrberg, 1973; Magnusson, 1977; Rolling, 1980; Aasheim & Øgaard, 1993; Johannsdottir et al., 1997; Tavajohi-Kermani et al., 2002). In the Portuguese population a prevalence of 1.3 per cent was estimated, with a slightly higher frequency in females and also a unilateral occurrence more common than the bilateral occurrence (Pinho et al., 2005). Unilateral agenesis is often associated with dysmorphia or microdontia of the corresponding contralateral tooth (Pinho et al., 2009). This discovery led to the presumption that maxillary lateral incisors microdontia may represent a different expression of these molecular changes that lead to a defective development of the maxillary lateral incisors and it should therefore be considered with particular emphasis in the clinical diagnosis or in family history, allowing us to suspect of tooth agenesis (Pinho et al., 2010a). Hypodontia in the temporary dentition is a rare occurrence (0.4–0.9 per cent) and, when present, occurs more frequently in the incisor region, generally including an upper lateral incisor or a lower central or a lateral incisor (Ravn, 1971; Bennett & Ronk, 1980; Järvinen & Lehtinen, 1981; Johannsdottir et al., 1997; Pinho et al., 2005). When hypodontia occurs in the temporary dentition, most authors report 100 per cent absence of the permanent successor (Ravn, 1971; Bennett & Ronk, 1980; Järvinen & Lehtinen, 1981; Johannsdottir et al., 1997; Pinho et al., 2005) (Figure 1).

Fig. 1. Temporary MLIA in a 3-year-old child and panoramic x-ray showing the temporary and permanent MLIA as well as of one permanent lower incisor.
There is also a strong association between double teeth formation (fusion) in the temporary teeth and hypodontia in the permanent dentition (Ravn, 1971; Nick-Hussein & Majid, 1996). When a mandibular temporary lateral incisor and a canine are fused, agenesis of the permanent mandibular lateral incisor is a constant fact (Ravn, 1971; Johannsdottir et al., 1997). In cases where the fusion occurs only in incisors, they are rarely changed at the permanent dentition (Ravn, 1971). However, in Figure 2 we can see a case with temporary mandibular left central fusion and permanent lateral incisor agenesis on the same side. Cases of germinations at the temporary dentition are usually followed by normal permanent dentition (Ravn, 1971).

Fig. 2. Double tooth by fusion in temporary mandibular left central and lateral incisors in a 3-year-old child; Panoramic x-ray showing agenesis in a permanent incisor on the same side of the double teeth.

2. Etiology

Several etiological factors have been suggested for the development failure of the permanent tooth germ, thus leading to its absence, such as: physical obstruction, dental lamina rupture, limitation of space or functional anomalies. In spite of recent progress, the etiopathogenesis of hypodontia remains largely unknown (Kuchler et al., 2010; Vastardis, 2000; Kapadia et al., 2007). There is evidence that congenital tooth absence can be the result of environmental or hereditary causes, or even of their interaction (Schalk-van der Weide & Bosman, 1996; Swinnen et al., 2008).

The development of human dentition in terms of structure and organization is under genetic control and involves several factors, therefore it is logical to assume that mutations in some genes encoding these factors may affect the normal development of teeth and, eventually, may cause their absence.

Non-syndromic hypodontia is more common than the syndromic type. The evidence of a genetic cause for non-syndromic hypodontia came from the identification of significant family aggregation of MLIA and suggest microdontia of maxillary lateral incisors as part of the same phenotype, segregating as an autosomal dominant trait with incomplete penetrance (Pinho et al., 2010a); however, modes of transmission linked to X-chromosome and of polygenic or multifactorial type have also been proposed (Chosack et al., 1975).

Candidate genes in early steps of tooth development regulation (MSX1, PAX9, AXIN2, TGFa, Activin-b A, LEFI, RUNX2, BMP4, MMP1, MMP20), have been screened for putative mutations in affected families (Kuchler et al., 2010; Lin et al., 2008; Tummers & Theisleff, 2009). Some mutations associated with tooth agenesis have been identified in humans at the MSX1 (Vastardis et al., 1996; Lidrali et al., 1998; Lidral & Reising, 2002; Mostowska et al., 2006) and PAX9 (Schuffenhauer et al., 1999; Stockton et al., 2000; Nieminen et al., 2001; Das et al., 2002; Pereira et al., 2006; Hansen et al., 2007; Tallon-Walton et al., 2007; Zhao et al., 2007; Guala et al., 2008) genes. Nevertheless, these genes might be fundamentally implicated in the
odontogenesis of posterior teeth (Pinho et al. 2010b). Lammi et al., (2004), reported that a nonsense mutation in the AXIN2, an essential component of the WNT/b-catenin pathway, caused familial oligodontia with a severe phenotype. In addition to oligodontia, those authors also found that a mutation in AXIN2 predispose the individual to colorectal cancer. Considering the discrepancy between the high prevalence rate of tooth agenesis and the relatively small number of reported causative mutations in the PAX9, MSX1, and AXIN2 genes, the genetic contribution to hypodontia/oligodontia seems quite heterogeneous (Gerits et al., 2006). Environmental and epigenetic factors as well as genes regulating odontogenesis require further in vivo and in vitro investigation in order to better explain the phenotypic heterogeneity and to increase our knowledge about the odontogenic process (Swinnen et al., 2008).

In spite of recent developments, data regarding the genes responsible for the less severe forms of hypodontia are still scarce and controversial (Kuchler et al., 2010). A study of familiar aggregation in a Portuguese population of sixty-two probands with MLIA and 142 first-degree relatives showed that the relative risk (RR) for a first-degree relative of an individual with MLIA to have the same type of agenesis was 15 times higher when compared with a relative of an individual without that agenesis. Published results support a significant familial aggregation of MLIA and show that MLIA almost never segregates with other forms of agenesis, and suggest that microdontia of maxillary lateral incisors is part of the same phenotype (Pinho et al. 2010a). However previous search for mutations in the PAX9 and MSX1 genes, and their potential association with the MLIA phenotype in 12 Portuguese families, didn’t show a clear association between those genes and the MLIA phenotype (Pinho et al., 2010b).

3. Craniofacial repercussions

There is no consensus on whether the changes that may occur during maxillary development are correlated or not with dental agenesis. However, some authors described a possible correlation (Wisth et al., 1974; Woodworth et al., 1985; Pinho et al., 2011a). According to Pinho et al. (2011a) MLIA is associated with an upper maxilla shortening, and also a negatively conditioned anterio-superior facial height dimension. Woodworth et al. (1985) showed that the decrease in maxillary length in individuals with MLIA is more frequently associated with skeletal Class III. However, others concluded that dental agenesis of few teeth, have little or no effect on craniofacial structure, as there is a higher prevalence ratio of skeletal Class I in patients with agenesis (Dermaut et al., 1986; Yuksel & Ucem, 1997; Pinho et al., 2011a).

Patients with severe congenital teeth absence have unique dental and skeletal patterns (Ben-Bassat and Brin 2009) probably caused by reduced occlusal support (Nodal et al., 1994). Dentofacial development in individuals with severe hypodontia, may be due to skeletal and functional compensation rather than being motivated by a different growth pattern (Ogaard & Krogstad, 1995).

4. Clinical manifestations

Early diagnosis of dental anomalies are essential when evaluating the pediatric patient and for treatment planning (Pilo et al., 1987). There are some direct and indirect clinical signs that can allow us to suspect of tooth agenesis. Among individuals with missing teeth, those who most frequently request
treatment are those with missing maxillary anterior teeth, namely the lateral incisors, probably for esthetic reasons. Hypodontia / oligodontia can be directly or indirectly supposed (Fig 3 and 4). The persistence of a temporary lateral incisor in the arch, beyond the expected date of eruption of its successor (Baccetti, 1998; Taylor, 1998), and / or asymmetric loss of temporary teeth (Millar & Taylor, 1995; Peck et al., 1996), dental midline shift in those who had unilateral agenesis on the same side of the agenesis and a molar Class II relation, which can be translated as a dental compensation towards the mesial sectors to camouflage the MLIA, are all indirect examples of this (Pinho et al., 2005, 2009, 2011b).

Fig 3. a, b) Temporary left MLIA in a 3-year-old child with a dental midline shift to the same side. (c) Panoramic x-ray showing bilateral permanent MLIA; d, e) Her sister with 2 years of age with no eruption of bilateral temporary lateral incisor; f) and only at 4 years of age the left one erupted; g) Panoramic x-ray showing bilateral permanent MLIA.
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Fig. 4. a, b) Left MLIA in a 13-year-old girl with a dental midline shift to the same side; c, d) Bilateral MLIA in a 13-year-old male with centered dental midline; e) Persistence of a maxillary temporary lateral incisor in a 18 years old girl with bilateral MLIA; f) MLIA with a Class II molar and canine relation.

In multiple congenital absences, you can also find other signs: attrition of the correspondent temporary persistent tooth, atypical tooth migration, ankylosis, infra-occlusion of temporary molars, supra-eruption of the permanent teeth, contralateral microdontia and diastemas (Bergendal et al., 1996, Dhanrajani, 2002) (Figure 5).

Fig. 5. a) Multiple congenital absences, with atypical tooth migration, ankylosis, infra-occlusion of temporary molars, and supra-eruption of the permanent teeth; b) Panoramic x-ray.
A radiographic examination should be used to complement the study of the patient, allowing confirmation of the number of absences and their location. Taking a panoramic radiography for routine was defended (Pilo et al., 1987) as a methodology to adopt for individuals who are less than 8 years old, whenever an incisor is missing in the arcade. This would facilitate an early diagnosis of tooth agenesis (Hobkirk et al., 1994; Bergendal et al., 1996).

According to Garn & Lewis (1970), crown size reduction associated with congenitally missing teeth is more significant in multiple agenesis than in cases of third molar agenesis, and occurs more frequently in women. Schalk-van der Weide et al. (1994) observed that patients with oligodontia (more than 6 teeth agenesis) had a reduction on both mesio-distal and labio-lingual dimensions of the tooth crowns (Figure 6). The reduction in size of some teeth in relatives may be an important factor for the determination of familiar occurrence of missing teeth (Schalk-van der Weide & Bosman, 1996). Yaqoob et al. (2011) stated that isolated bilateral absence of maxillary lateral incisors is associated with reduced mesiodistal tooth widths of both maxillary and mandibular anterior segments.

Some authors stated that permanent tooth agenesis, maxillary lateral incisor microodontia, palatally displaced canines, and distoangulation of mandibular second premolars are frequently associated with maxillary lateral incisor agenesis, providing additional evidence of genetic interrelationship as cause for these dental anomalies (Garib et al., 2010). For others, the factors involved in third molar agenesis and that of other teeth are probably the same (Baum & Cohen, 1971).

Fig. 6. a) Multiple diastemas associated with generalized microdontia and multiple dental agenesis in a 12 years old female; b) Generalized microdontia confirmed by models measurements c) Panoramic x-ray confirming the congenital absence of 18, 16, 12, 22, 26, 28, 38, 35 and 48 teeth.

In addition, some authors (Becker et al., 1981; Peck et al., 1996; Pirinen et al., 1996; Baccetti, 1998; Peck et al., 2002), referred that in early detection, we must take also into account the importance of the lateral incisor in the eruption of the canine tooth. The frequency of paradoxical bad positioning of the canine in cases of agenesis and / or lateral incisors microdontia demonstrates the importance of these teeth in guiding the way to canine eruption (Figure 7).

Other authors (Zirberman et al., 1990; Peck et al., 1996; Pirinen et al., 1996; Peck et al., 2002) suggested that these situations can be caused by the same genetic factor. Despite this strong evidence, there are also those who disagree (Brenchley & Oliver 1997; Pinho et al., 2005, 2009) and do not associate an ectopic canine with microdontia or with agenesis of maxillary lateral incisor but with malocclusions of Class II Div 2, due to the typical displacement of the labiopalatine adjacent lateral incisor (Millar & Taylor 1995).
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The simultaneous occurrence of agenesis and supernumerary teeth is uncommon (Ranta & Tulensalo 1988; Zhu et al., 1996; Zhu et al., 1996; Pinho et al., 2005; Anthonappa et al., 2008) and it is more frequent in the permanent than in the primary dentition (Ranta & Tulensalo, 1988; Pinho et al., 2009) (Figure 8).

In some syndromes there are typical patterns of hypodontia, while in others the congenital reduction in teeth number is described as sporadic. Anodontia (congenital absence of all teeth) is rare (Burzynski & Escobar, 1983) and is often associated with ectodermal dysplasia (Marques & Till, 1994). Hypohidrotic ectodermal dysplasia is the most common form of ectodermic dysplasia in humans and is estimated to affect at least 1 in 17,000 people worldwide (ghr.nlm.nih.gov/hypohidrotic-ectodermal-dysplasia). Most people with hypohidrotic ectodermal dysplasia have hypohidrosis, hypotrichosis and absent or malformed teeth (Marques & Till 1994; Itthagarun & King 1997; Kobielak et al., 2001) (Figure 9). There are other syndromes like Rieger’s syndrome where hypodontia is also a main feature (Prabhu et al., 1997). For Schalk-Van Der Weide et al. (1994), patients with oligodontia/I (isolated) showed a low degree association of extra-oral signs with combinations of just one or two ectodermic anomalies. On the contrary, patients with oligodontia/S (Syndrome) show a strong tendency to present a combination of three or more ectodermic anomalies.
5. Treatment

Of all missing teeth, those that most often motivate treatment request, probably for esthetic reasons, are the early absent maxillary teeth, namely the lateral incisors. There are clinical situations in which the residual spaces are minimal and the patients feel fully satisfied with their esthetic appearance (Figure 10). Sometimes, though not completely satisfied, they are not motivated or economically capable for orthodontic treatment (Figure 11 and 12).

Clinical case 1 (Fig 10a-e)

Fig. 10. a) Canine mesialized with unsightly color; b) Canine color corresponding to an A4 (range of Vita®); c) canines bleaching; d) Final aesthetic result after nighttime use of bleach; e) color change to an A1.
Clinical case 2 (Fig 11a-d and Fig 12a-d)

Fig. 11. Slight diastemas, unilateral agenesis of UR2 (12) with the UR3 (13) totally mesialized and the persistence of the temporary URC (53) distally positioned in relation to the corresponding permanent tooth. UL2(22) with conical appearance.

Fig. 12. Improvement of the UL2 (22), and the temporary UR3 (53) shape by an adhesive composite restoration in combination with cusp reshaping and adhesive composite restoration of the UR3 (13) mesial face.

If uni- or bilateral agenesis of the maxillary lateral incisors leads to situations that are esthetically unpleasant or unacceptable, therapeutic options should be orthodontic space closure or opening. The choice between these two types of treatment should not be made empirically. In most instances, the presence or absence of major occlusion problems serves as the primary criterion for either space closure or space opening.

There are several factors, and sometimes several interrelated factors, that limit different treatment options. Before starting any treatment the professional is obliged to inform patients of the various options, their clinical implications, advantages and disadvantages, and what should be the best plan for their particular case.

Several factors such as molar ratio, degree of incisors protrusion, facial patterns, skeletal arches interrelation, dental arch configuration, dental inclination, tooth shape, incisal contact, gingival margins contour, black triangles smile line, lip shape, and esthetic results should be considered in therapeutic options (Pinho & Neves 2001, 2003; Park et al., 2010). Also, we have to take in consideration the position of the lip attachment at the nasolabial junction that has a profound effect on the esthetics of the profile (Park et al., 2010).

5.1 Bilateral maxillary lateral incisors agenesis

5.1.1 Closure of the space

Closing the space means definitive treatment with orthodontic mesialization of the canine, replacing the missing lateral incisor, thus closing the anterior diastema. For many authors this is the treatment of choice since it is able to get a good esthetic (Millar & Taylor 1995; Pinho, 2003; Park et al., 2010). When the solution is space closure, to obtain an optimal
esthetic and functional result it must be assumed that the canines will be modified, so to look and function as lateral incisors. The canine tooth requires, in cases associated with orthodontic mesialization, reduction of the mesial and distal face, as well as cutting of the cusp tip and palate face, and remodeling of the convexities in contact areas in order to create vertical interproximal areas, which are more agreeable with the contact points morphology of the lateral incisors and does not induce occlusal interferences (Bowden & Harrison, 1994; Millar & Taylor, 1995; Miller, 1995).

5.1.1.1 Indications

Some consideration factors, when closing the space, must be followed: Negative maxillary tooth disharmony (DDM), with crowding in the anterior area (Bowden and Harrison 1994); Class I with crowding, where extractions at the lower arch are indicated (Millar and Taylor 1995; Rosa & Zachrisson, 2001); Mesialized canine and easily modified with an acceptable color, compatible with the adjacent teeth (Millar & Taylor, 1995); the yellowish color of the canines can be improved using the technique of selective vital teeth bleaching (Pinho, 2003); Value of incisors relation with Class II division 1 (Bowden & Harrison 1994); Malocclusion that do not require extraction of mandibular teeth, with respect to Class II canine and molar (Pinho, 2003; Park et al., 2010).

5.1.1.2 Advantages

The benefits obtained by the closure of spaces corresponding to the missing lateral incisors are the following (Millar & Taylor, 1995): Solving of any previous crowding; avoiding artificial teeth; limiting treatment to orthodontics, decreasing costs by eliminating prosthetic treatment.

5.1.1.3 Disadvantages

The main disadvantage is the loss of the canine Class I. Some authors (Rosa & Zachrisson, 2001) stated that no difference existed in adequacy of the occlusal function between canine-protected and group function (with remodeling procedures and occlusal adjustment, the group function is satisfactory). So, for these authors the elimination of the prosthetic solution makes this alternative the first option whenever possible.

Clinical case 3 (Figure 13 and 14)

Bilateral MLIA, with space closure in a 23 years old male.
Diagnosis: Bilateral agenesis of UR2 (12) and UL2 (22); canine and molar Class I relation on the right side, canine and molar Class II relation on the left side; bi-protrusion with pro-inclined incisors in a hyperdivergent biotype with labial protrusion; overjet and overbite were 0 mm and -1 mm (open bite) respectively; the patient used a removable prosthesis with microdontic lateral incisors due to minimal space in the arcade. Due to esthetic factors, like bilabial protrusion and a closed naso-labial angle, space closure was the best option combined with extraction of the two mandibular first premolars (Figure 13).
After treatment, improvement of the incisors position and labial aesthetics can be observed. The harmony between the gingival margin of the central incisors, canines and first premolars was good, despite the mesio-distal canine dimension. Shorter canine gingival margin in relation to both central incisors, and first premolars more buccally positioned to bring them to a canine appearance (Figure 14).
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Fig. 13. Pre-treatment documentation, and panoramic x-ray at the middle of the treatment.

Fig. 14. Post-orthodontic treatment documentation.

Clinical case 4 (Figure 15-17)

Fig. 15. Pre-treatment intra-and extra-oral photos.
Clinical case 4: Bilateral MLIA, with space closure in a 16 years old female.
Diagnosis: Bilateral agenesis of UR2 (12) and UL2 (22), convex facial profile with an increased lower facial third, lip incompetence and high smile line; mesialization of both canines, persistence of the deciduous canines, right-sided Class II molar relation and a left-sided Class I relation, with a 4.6-mm overbite and a 11.6-mm overjet, highly pronounced Spee curve, due to the extrusion of lower incisors; 4mm left deviation of the upper dental midline and right 1mm deviation of the lower dental midline; despite these deviations, no facial asymmetry was detected (Figure 15).

The treatment aims were maintenance of the deciduous canine at the molar and canine Class I side and extraction the deciduous canine at the molar and canine Class II side. These steps aimed to allow the overjet and upper midline correction by retrusion of the anterior sector and deviation of the upper midline to the right. Improvement of the overbite and crowding, by intrusion and protrusion of the lower incisor group, and the maintenance of enough bone space at the level of the deciduous left upper canine in order to place an implant later.

After treatment we can observe improvement of the incisors position, as well as a more aesthetic labial position. Four months after orthodontic treatment ending, canines were extracted and implants were placed.
remodeled, by proximal aspects rectification and vestibular aspect modification and remodeling with a composite resin (final documentation, a-f). Eight years after, the results remained stable (Figure 17).

5.1.1.4 Interceptive treatment

Interceptive extractions of the deciduous upper lateral incisors and canine should be considered, while they are relatively high in the alveolus, in order to promote mesialized canine eruption and therefore spontaneous closure of the residual space caused by the absence of lateral incisors. Also, second deciduous molars interceptive extraction promote mesial migration of posterior teeth, causing an eruption of the permanent molars in a molar Class II relation, thus reducing the need for further treatment (Bowden & Harrison, 1994; Millar & Taylor, 1995). It is important to consider any coexisting malocclusion, a factor that may influence the decision to extract or not teeth. For example, in a nine years-old patient with a Class I (or Class III) relation, the extraction of the temporary maxillary canine and lateral incisor may allow an advance of the posterior teeth, whenever combined with the temporary loss of the second molars to help this advancement. However, in severe Class II, extractions should be done with additional caution because, they can curb the anterosuperior sector growth, or can facilitate the advancement of posterior sector making it impossible to fix Class II (bloking central incisor retrusion) (Millar & Taylor, 1995).

Clinical case 5 (Figure 18-23)

Fig. 18. Extra- and intra-oral photos; lateral telerradiography and panoramic x-ray, before interceptive treatment.
Fig. 19. Oclusal and smile photos during the use of removable appliance in interceptive treatment.

Fig. 20. Extra- and intra-oral photos; lateral telerradiography and panoramic x-ray, after interceptive treatment.

Fig. 21. Intra-oral photos, during straight wire technique treatment, with an overlay activated to improve the left posterior cross-bite.

Clinical case 5:
Unilateral MLIA, interceptive treatment, space closure with symmetry in a 7 years old female, at the beginning of the treatment.
Diagnosis: Unilateral agenesis of UR2 (12) with included microdontic UL2 (22) and retained temporary UL2 (62); bilateral Class II molar relationship, pro-maxilla, increased overjet, labial incompetence, protruded UL1 (21) with lower lip interposition; right 5 mm deviation of the upper dental midline and centered lower dental midline; crossbite of the UL6 (26) and LL6 (36) (Figure 18).
Interceptive treatment: extraction of the included microdontic UL2 (22), the retained temporary UL2 (62), the temporary UR3 (53) and UL3 (63) when the patient was 8 years old. A removable appliance was placed for 1 year in order to improve the UL1 (21) position (Figure 19).

The extraction program led to a mesialized canine eruption and hence a spontaneous closure of residual space occurred and the upper dental midline was improved as well as the overjet (Figure 20).

Two years and six months after the use of the removable appliance ended, a bilateral fixed appliance allowed the improvement of the incisors position and of the dental intermaxillary relation; at the first stage a Straight-Wire technique (Figure 21) was used and, followed by a second stage with Multiloop Edgewise Arch-Wire (Figure 22) at the lower arch to finish the case with stable sagittal and vertical occlusion relationships between maxillary and mandibular arches.

The harmony between the gingival margin of the central incisors, canines and first premolars was good, despite the mesio-distal canine dimension. The facial profile improved and in spite of lateral incisor space closure, a stable dental articulation was accomplished with balanced occlusion with molar and canine bilateral Class II relation. Both midlines arches were aligned with the facial midline, and a correct overjet and overbite relationship were obtained (Figure 23).
5.1.2 Opening or maintaining space

The placement of an intraosseous implant in the edentulous area, followed by placement of a crown should be the first therapeutic option in cases of orthodontic space opening in post growth patients (Bowden & Harrison, 1994; Thilander et al., 1994; Small, 1996; Pinho & Neves, 2001; Zarone et al., 2006). Diagnosis and treatment of growing children with missing lateral incisors can be a problem because implants cannot be placed until facial growth is complete (Kokich, 2002, 2005). Females mature faster than males, and their adolescent growth spurt occurs sooner.

Space will be determined ultimately by the occlusion. Canines should be placed in a position that allows canine discursion along the central incisors in a position that will provide optimal esthetics. Remaining space should be ideal for lateral incisor restoration, generally with 5 to 7 mm (Kokich, 2005). For Bergendal et al., (1996) minimum interdental space required for an implant is 6 mm mesio-distal and 5 mm in a buccolingual direction. However there are biologic limits of the bone dimension around implants that influence soft tissue esthetics. From the mesiodistal view, the distance between an implant and a tooth should not be less than 1.5mm, and if this minimum distance is not maintained, the attachment on the tooth side will undergo resorption, causing reduction or loss of the interproximal papilla (Esposito et al., 1993). From the buccolingual view, a 1.5mm-wide crater will also appear on the buccal side of the implant head (Spray et al., 2000).

In MLIA this ideal amount of bone is not available in the majority of cases, with the consequence of a high risk for soft tissue recession after an implant placement and prosthetic restoration. So, in these situations with a very esthetically demanding case, bone augmentation procedure will be needed (Grunder et al., 2005). An x-ray examination, using periapical and panoramic techniques and, if necessary, computed tomography, gives us information that may be necessary for the proper assessment of the receptor site for the implant.

Autotransplantation is a viable option for treating missing teeth when a donor tooth is available (Bae et al., 2010).

5.1.2.1 Indications (Bowden & Harrison, 1994; Millar & Taylor, 1995, Pinho, 2001, 2004)

A canine that is difficult to remodel esthetically, as it presents a negative color to the bleaching technique or an unfavorable morphology; Concave profile; Widespread microdontia; Positive DDM, with multiple diastemas; Some cases of Class III malocclusion; Insignificant malocclusion, with Class I, without associated anomalies; Good intercuspidation; Some cases of cleft palate.

This option is recommended when a molar and canine Class I relation is the goal, with canine-protection as the basis of laterality.

Because some restorative options are possible, it is important to perform a clinical examination to decide which treatment plan best suit each patient. The solution with an implant is a viable option for replacing congenitally missing lateral incisors and should be seen as a definitive treatment plan, provided that all permanent teeth have erupted and skeletal growth is completed (Thilander et al., 1994; Small, 1996).

5.1.2.2 Advantages and disadvantages

The intercuspidation is maintained within the neutrality, preserving both canine and molar Class I relation. However, it is necessary to have different prosthetic solutions (Pinho & Neves, 2001).
5.1.2.3 Early treatment

Early diagnosis and the effective clinical management of hypodontia are important because the condition can lead to esthetic, physiologic, and functional problems (Tunc et al., 2011). In order to maintain various treatment options in the future, some authors defended that early orthodontic intervention may eliminate some of the periodontal and restorative problems that could arise in these patients as adults (Kokich 2005). For example, as a result of extracting the maxillary primary lateral incisor and guiding the eruption of the permanent canine into the lateral incisor space, an excellent implant site can be developed in the mixed dentition. In contrast, other authors stated that deciduous teeth should be retained if present, in order to preserve alveolar bone after tooth extraction, since alveolar bone resorbs rapidly after tooth extraction (Bowden & Harrison 1994).

Clinical case 6 (Figure 24-27)

Bilateral MLIA, with space opening in a 14 years old male.

Diagnosis): Bilateral agenesis of UR2 (12) and UL2 (22) and bilateral total mesialization of canines; concave soft-tissue profile, right molar Class I and left molar Class II relation (Figure 24).

Treatment goals involved regaining space for implant placement at the areas corresponding to UR2 (12) and UL2 (22). At the end of the treatment a bilateral molar Class I relationships was obtained. At the first treatment stage a Straight-Wire technique was used for twelve months, to allow alignment and levelling and, at the second stage, an occlusal plane modification and mandible repositioning was achieved with Multiloop Edgewise Arch-Wire (MEAW), used for six months. Different MEAW activation on both sides and short Class I vertical elastics in the anterior area on the right side and Class II on the left side were important to apply different orthodontic forces (Figure 25). This procedure made it possible to finish the case with stable sagittal and vertical occlusion relationships between maxillary and mandibular arches (Figure 26). A removable retainer has been used with a denture tooth to replace bilateral MLI until complete vertical and horizontal growth of the jaws (Figure 27).

Fig. 24. Pre-treatment intra- and extra-oral photos (with cephalometric tracing superimposed) and panoramic x-ray.
Fig. 25. Intra-oral photos with Multiloop Edgewise Arch Wire (MEAW) in the lower arch.

Fig. 26. Intra- and extra-oral photos (with cephalometric tracing superimposed) and panoramic x-ray after the orthodontic treatment.

Fig. 27. Removable retainer with denture teeth to replace bilateral MLIA.

**Clinical case 7 (Figure 28 and 29)**

Bilateral MLIA, space opening in a 14 years old female with implants placement when the patient was 18 years old.

Diagnosis: Diastemas in the maxillary anterior region and partial bilateral mesialization of canines, due to bilateral agenesis of UR2 (12) and UL2 (22); concave soft-tissue profile with an open nasolabial angle, lip competence and a normal smile line, in relation to the facial midline, upper dental midline deviated to the left and centered mandibular dental midline; LL5 (35) agenesis with the persistence of the correspondent temporary; bilateral molar and canine Class II relationship (Figure 28).

Treatment involved space recovery for implant placement at the areas with congenital absences of UR2 (12) and UL2 (22), when the patient was 18 years old. Bilateral molar and canine Class I relationship obtained due to a lateral and posterior teeth distalization and mesial reposition of the mandible (Figure 29).
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Fig. 28. Pre- treatment intra- and extra-oral photos (with cephalometric tracing superimposed) and panoramic x-ray.

Fig. 29. Intra- and extra-oral photos and panoramic x-ray after orthodontic treatment.

5.2 Unilateral maxillary lateral incisors agenesis
When the lateral incisor microdontia is marked and/or root malformation is associated, it is preferable that the incisor extraction and the canine mesialization happens simultaneously, thus creating a symmetrical situation with preservation of the midline (Bowden & Harrison, 1994). If the option is to maintain the tooth, even if microdontic, we create mesial and distal spaces to the tooth to allow for later restoration by composite resin full ceramic crown, if at the root level no significant changes are seen.

If the lateral incisor is present in the arcade and is normal in size, the treatment option will depend on what has been mentioned, mainly the initial molar relations and the maxillary symmetry.

Not all solutions in unilateral agenesis should be based on predefined criteria, and an adequate clinical sense must always be present at the time of decision. When the occlusion is
not appropriate to carry out the treatment plan with the opening or closure of spaces, some secondary criteria may help our choice. The position of the lip during smiling should be assessed, since the difference of color among teeth, lip contour, canine and adjacent teeth, as well as the difference in height between gingival margins will be less evident in patients with a low smile line. However, in patients with a high smile line, differences will be more detectable. These are the cases where there is always more difficulty, whether the option is the replacement with prosthesis or the space closure.

The harmony between the gingival margin of the front six maxillary teeth plays an important role in the aesthetic appearance (Kokich, 1996). The gingival contour depends on various factors like the periodontal biotype and the tooth shape. The gingival margin should follow the contour of the line-cementum junction, while the apex of the interproximal papilla must be situated midway between the incisal edge and the cervical margin of each anterior tooth, filling all the interproximal space. There are two acceptable standards for the height of the gingival margin of the six anterior-superior teeth: Standard Class 1, in which the gingival margins of the two central incisors (ICS) and upper canines (CS) must be within the same level and the lateral incisors (ILS) is positioned 1-2 mm more incisal; Standard Class II, in which the ICS, ILS and CS are at the same height (Chiche, 1994).

Clinical case 8 (Figure 30-32)

Fig. 30. Pre-treatment intra-and extra-oral photos and panoramic x-ray.

Fig. 31. Intra-oral photos during and at the end of the orthodontic treatment.

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Clinical case 8: Symmetric space opening, in a 23 years old female.
Diagnosis: Unilateral agenesis of the UR2 (12) with a conical contralateral UL2 (22) and a mesialized UR3; molar and canine Class II relation on the right side and a molar and canine Class I on the left side; no dental crowding in the mandibular arch and some mesial and distal spaces related to the LR3 (43). Overjet and overbite were approximately 0mm. Panoramic x-ray revealed a mesialized root of the UR3 (13) (Figure 30).
The main factor that influenced the decision for the opening of space preserving symmetry was the high smile-line and thin lips that the patient presented.
The treatment was carried out in order to create symmetry, opening of space for placing an implant to replace tooth agenesis. The right molar Class I relation was obtained due to distalization of all 1st quadrant teeth and mesial movements of the posterior-inferior sector to close the existing mesial and distal diastema at the LR3 (43). After opening the space a temporary prosthesis tooth was placed to improve the aesthetics. In order to improve the symmetry of the smile a gingival recontouring and a crown was performed on the UL2 (22) (Figure 31). Two years after the treatment the occlusion remained stable (Figure 32).

Clinical case 9: (Figure 33-35)
Symmetric space opening and lower incisor extraction, in a 12 years old female.
Diagnosis: Unilateral agenesis of the UR2 (12) with a microdontic contralateral UL2 (22); bilateral molar and canine Class I relation; severe dental crowding of the mandibular incisors; normal overjet and overbite, labial incompetence; crossed UL5 (25), UL6 (16), LL5 (35) and LL6 (36). Panoramic x-ray confirmed mesial position of the UR3 (13) (Figure 33). The main factor that influenced the decision to symmetrically open the space and extraction of LL1 (31) was the severe dental crowding of the mandibular incisors associated with a fine gingival biotype at the level of LL1 (31) and LR2 (42), labial incompetence and the existence of Bolton discrepancy due to the UL2 (22) microdontia (Figure 34). The treatment was carried out in order to create enough space to allow the placement of an implant with the same dimension of the prosthetic tooth corresponding to the microdontic lateral incisor. At the end, left side bite was uncrossed (Figure 34). Six years after the treatment the occlusion remained stable (Figure 35).
Fig. 33. Pre-treatment intra-and extra-oral photos and panoramic x-ray.

Fig. 34. Intra-oral photos during orthodontic treatment after the lower incisor extraction and at the end of the treatment.

Fig. 35. Intra-and extra-oral photos, 6 years after orthodontic treatment; panoramic x-ray.
Clinical case 10 (Figure 36-39)

Symmetric space opening, in a 28 years old female.

Diagnosis: Unilateral agenesis of UR2 (12) with the UR3 (13) totally mesialized and persistence of the temporary URC (53) with distal positioned corresponding permanent tooth; contralateral UL2 (22) with conical appearance and a large periapical lesion; high vertical inclination of the incisors with an hypodivergent biotype; incomplete molar and canine Class II relation on both sides, except at the UR3 (13) where it is complete; overjet and overbite were 4.5mm and 8mm, respectively (Figure 36).

Due to esthetic factors originated by the smile line, as well as the periapical lesion of UL2 (22), the option was to extract UL2 (22) as well as the URC (53), to close the space with symmetry, aligning the permanent canines in order to replace the lateral incisors (Figure 37).

After treatment an improvement of the torque and the vertical position of upper incisors, was achieved. The option for leaving the upper canines slightly tilted towards a mesial position was to allow greater stability at the contact points with the central incisors, thereby preventing space reopening, taking into account the existing hypodivergent facial pattern (Figure 38). The improvement of the canines color and shape was possible though a bleaching of the canines and a combination of cupid reshaping and adhesive composite restoration of the mesial face (Figure 39).

Fig. 36. Pre- treatment intra-and extra-oral photos; lateral telerradiography and panoramic x-ray.

Fig. 37. Intra-oral photos, during orthodontic treatment
Clinical case 11 (Figure 40-44)

Asymmetric space closure, in a 12 years old female.
Diagnosis: Unilateral agenesis of the UR2 (12) with the UR3 (13) not totally mesialized; the contralateral UL2 (22) presented conical appearance; overjet increased due to high pro-inclination of the upper incisors; molar and canine Class II relation on the right side, being incomplete at the molar and full at the canine; on the left side a molar Class I and a canine Class II, due to a distal space at the UL3 (23); protruded upper lip with a closed nasolabial angle tendency. Panoramic x-ray revealed a considerable space between the root of UR1 (11) and the UR3 (13) (Figure 40).

The main factor that influenced the decision to close the space with asymmetry was the lower smile-line without visibility of the gingival margin during a forced smile.

The treatment was carried out in order to close space only on the right side with asymmetry, aligning the UR3 (13) with cupid reshaping to replace the UR2 (12). On the contralateral side the right molar Class I relation was maintained and the conical UL2 (22) was restored by adhesive resin composite that was done before the orthodontic treatment to facilitate the brackets bonding and then improved at the end of treatment. At the first stage a Straight-Wire technique was used (Figure 41) and then at the second stage a Multiloop Edgewise Arch-Wire technique was also used in order to improve occlusal relations between maxillary and mandibular arches (Figure 42).
After treatment, improvement of the incisors and anterior upper teeth gingival margin positions, as well as an esthetics labial position, instead of dental asymmetry can be observed (Figure 43).

Fig. 40. Pre-treatment intra-and extra-oral photo; lateral telerradiography and panoramic x-ray.

In order to improve the symmetry of the smile a gingival recontouring was performed on the maxillary left lateral incisor, and resin composite reconstructions were done on the mesial face of UR3 (13) and UL2 (22). On the right side, lateral mandibular movements were guided by the first premolar, and in the left side by the canine. In the protrusion movement there is a disocclusion of the posterior teeth (Figure 44).

Fig. 41. Intra oral photos, during straight wire technique treatment.
Fig. 42. Intra oral photos, during Multiloop Edgewise Arch Wire (MEAW).

Fig. 43. Intra-and extra-oral photos after orthodontic treatment; and after UR3 (13) and UL2 (22) composite resin remodelation; lateral telerradiography and panoramic x-ray.

Fig. 44. Lateral and protrusive mandibular movements.

6. Final considerations

Orthodontists frequently face patients with maxillary lateral incisor agenesis (Pinho et al., 2005). Results obtained from Portuguese data showed that the risk for a first-degree relative
of an individual with MLIA to have the same kind of agenesis is about 15 times higher than that of the general population (Pinho et al., 2010a). So, it is thus probable that different genes are involved in different phenotypes (Pinho et al., 2010a, 2010b).

In the diagnosis of agenesis of maxillary lateral incisors it is necessary to perform a good clinical examination and subsequent radiographic confirmation in order to observe not only the absence itself but also all the anomalies that may be associated (Pinho et al., 2005, 2009, 2011a, 2011b).

In the treatment plan for agenesis of the lateral incisors and the consequent choice for orthodontic therapy with closure or opening of the space we have to take into account esthetic, skeletal, periodontal and functional factors (Pinho et al., 2001, 2003, 2004). Questionable situations should be based on predefined criteria, taking into account the clinical sense at the time of decision. In unilateral agenesis, the main factor that influences the decision to close the space with asymmetry is the position of the lip during forced smile.

7. References

Aasheim, B. & Øgaard B. (1993). Hypodontia in 9-year-old Norwegians related to need of orthodontic treatment. Scandinavian Journal of Dental Research, 101(5): 257-260.

Anthonappa, R. P.; Lee, C. K. et al. (2008). Hypohyperdontia: literature review and report of seven cases. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology & Endodontics, 106(5): e24-30.

Baccetti, T. (1998). A controlled study of associated dental anomalies. Angle Orthodontist 68(3): 267-274.

Bae, J. H.; Choi, Y. H. et al. (2010). Autotransplantation of teeth with complete root formation: a case series. Journal of Endodontics, 36(8): 1422-1426.

Baum, B. J. & Cohen, M. M. (1971). Agenesis and tooth size in the permanent dentition. Angle Orthodontist, 41(2): 100-2.

Becker, A.; Smith, P. et al. (1981). The incidence of anomalous maxillary lateral incisors in relation to palatally-displaced cuspids. Angle Orthodontist, 51(1): 24-29.

Ben-Bassat, Y. & I. Brin (2009). Skeletal and dental patterns in patients with severe congenital absence of teeth. American Journal of Orthodontics and Dentofacial Orthopedics, 135(3): 349-356.

Bennett, C. G.; & Ronk S. L. (1980). Congenitally missing primary teeth: report of case. ASDC Journal of Dentistry for Children, 47(5): 346-348.

Bergendal, B., Bergendal, T. et al. (1996). A multidisciplinary approach to oral rehabilitation with osseointegrated implants in children and adolescents with multiple aplasia. European Journal of Orthodontics, 18(2): 119-129.

Bergstrom, K. (1977). An orthopantomographic study of hypodontia, supernumeraries and other anomalies in school children between the ages of 8-9 years. An epidemiological study. Swedish Dental Journal, 1(4): 145-157.

Bowden, D. E. & Harrison J. E. (1994). Missing anterior teeth: treatment options and their orthodontic implications. Dental Update, 21(10): 428-434.

Brenchley, Z. & Oliver R. G. (1997). Morphology of anterior teeth associated with displaced canines. British journal of orthodontics, 24(1): 41-5.
Burzynski, N. J. & Escobar V. H. (1983). Classification and genetics of numeric anomalies of dentition. *Birth Defects*, 19(1): 95-106.

Chiche, G. P., A. (1994). Esthetics of anterior fixed prosthetics. Chicago: *Quintessence Books*, 13-32.

Chosack, A.; Eidelman E. et al. (1975). Hypodontia: a polygenic trait--a family study among Israeli Jews. *Journal of Dental Research*, 54(1): 16-19.

Das, P.; Stockton, D. W. et al. (2002). Haploinsufficiency of PAX9 is associated with autosomal dominant hypodontia. *Human Genetics*, 110(4): 371-376.

Dermaut, L. R.; Goeffers, K. R. et al. (1986). Prevalence of tooth agenesis correlated with jaw relationship and dental crowding. *American Journal of Orthodontics and Dentofacial Orthopedics*, 90(3): 204-210.

Dhanrajani, P. J. (2002). Hypodontia: etiology, clinical features, and management. *Quintessence International*, 33(4): 294-302.

Esposito, M.; Ekestubbe A., et al. (1993). Radiological evaluation of marginal bone loss at tooth surfaces facing single Branemark implants. *Clinical Oral Implants Research*, 4(3): 151-157.

Garib, D. G.; Alencar, B. M. et al. (2010). Agenesis of maxillary lateral incisors and associated dental anomalies. *American Journal of Orthodontics and Dentofacial Orthopedics*, 137(6): 732 e1-6; 732-733.

Garn, S. M. & Lewis, A. B. (1970). The gradient and the pattern of crown-size reduction in simple hypodontia. *Angle Orthodontist*, 40(1): 51-8.

Gerits, A.; Nieminen, P. et al. (2006). Exclusion of coding region mutations in MSX1, PAX9 and AXIN2 in eight patients with severe oligodontia phenotype. *Orthodontic Craniofacial Research*, 9(3): 129-136.

Grunder, U.; Gracis, S., et al. (2005). Influence of the 3-D bone-to-implant relationship on esthetics. *International Journal of Periodontics & Restorative Dentistry*, 25(2): 113-119.

Guala, A.; Falco, V., et al. (2008). Deletion of PAX9 and oligodontia: a third family and review of the literature. *International Journal of Paediatric Dentistry*.

Hansen, L.; Kreiborg, S. et al. (2007). A novel nonsense mutation in PAX9 is associated with marked variability in number of missing teeth. *European Journal of Oral Sciences*, 115(4): 330-333.

Hobkirk, J. A.; Goodman, J. R., et al. (1994). Presenting complaints and findings in a group of patients attending a hypodontia clinic. *British Dental Journal*, 177(9): 337-339.

Horowitz, J. M. (1966). Aplasia and malocclusion: a survey and appraisal. *American Journal of Orthodontics*, 52(6): 440-453.

Itthagarun, A. and N. M. King (1997). Ectodermal dysplasia: a review and case report. *Quintessence International*, 28(9): 595-602.

Järvinen, S. & Lehtinen L. (1981). Supernumerary and congenitally missing primary teeth in Finnish children. An epidemiologic study. *Acta Odontologica Scandinavica*, 39(2): 83-86.

Johannsdottir, B.; Wisth, P. J. et al. (1997). Prevalence of malocclusion in 6-year-old Icelandic children. *Acta Odontologica Scandinavica*, 55(6): 398-402.

Kapadia, H.; Mues, G. et al. (2007). Genes affecting tooth morphogenesis. *Orthodontic Craniofacial Research*, 10(4): 237-44.
Kobielak, K.; Kobielak, A. et al. (2001). Mutations in the EDA gene in three unrelated families reveal no apparent correlation between phenotype and genotype in the patients with an X-linked anhidrotic ectodermal dysplasia. *American Journal of Medical Genetics, 100*(3): 191-197.

Kokich, V. (2005). Early Management of Congenitally Missing Teeth. *Seminars in Orthodontics* 11: 146-151.

Kokich, V. G. (1996). Esthetics: the orthodontic-periodontic restorative connection. *Seminars in Orthodontics*, 2(1): 21-30.

Kokich, V. O. Jr. (2002). Congenitally missing teeth: orthodontic management in the adolescent patient. *American Journal of Orthodontics and Dentofacial Orthopedics*, 121(6): 594-5.

Kuchler, E. C.; Menezes, R. et al (2010). MMP1 and MMP20 contribute to tooth agenesis in humans. *Archives of Oral Biology, [Epub ahead of print]*

Lammi, L., S. Arte, et al. (2004). Mutations in AXIN2 cause familial tooth agenesis and predispose to colorectal cancer. *American Journal of Human Genetics*, 74(5): 1043-1050.

Lidral, A. C. & Reising, B. C. (2002). The role of MSX1 in human tooth agenesis. *Journal of Dental Research*, 81(4): 274-8.

Lidral, A. C.; Romitti, P. A. et al. (1998). Association of MSX1 and TGFB3 with nonsyndromic clefting in humans. *American Journal of Human Genetics*, 63(2): 557-568.

Lin, Y.; Cheng, Y. S. et al. (2008). FGFR2 in the dental epithelium is essential for development and maintenance of the maxillary cervical loop, a stem cell niche in mouse incisors. *Development Dynamics*, 238(2): 324-330.

Magnusson, T. E. (1977). Prevalence of hypodontia and malformations of permanent teeth in Iceland. *Community Dentistry and Oral Epidemiology*, 5(4): 173-178.

Marques, A. P. F. & Till, M. J. (1994). Ectodermal Dysplasia: treatment considerations and case reports. *Revista Portuguesa Cirurgia Maxilofacial*, 35(1/2): 17-22.

Millar, B. J. & Taylor, N. G. (1995). Lateral thinking: the management of missing upper lateral incisors. *British Dental Journal*, 179(3): 99-106.

Miller, T. E. (1995). Implications of congenitally missing teeth: orthodontic and restorative procedures in the adult patient. *Journal of Prosthetic Dentistry*, 73(2): 115-122.

Mostowska, A.; Biedziak, B. et al. (2006). A novel c.581C>T transition localized in a highly conserved homeobox sequence of MSX1: is it responsible for oligodontia? *Journal of Applied Genetics*, 47(2): 159-164.

Muller, T. P.; Hill, I. N. et al. (1970). A survey of congenitally missing permanent teeth. *Journal of the American Dental Association*, 81(1): 101-107.

Nick-Hussein, N. N. & Majid, Z. A. (1996). Dental anomalies in the primary dentition: Distribution and correlation with the permanent dentition. *Journal of Clinical Pediatric Dentistry*, 21(1): 15-19.

Nieminen, P.; Arte, S. et al. (2001). Identification of a nonsense mutation in the PAX9 gene in molar oligodontia. *European Journal of Human Genetics*, 9(10): 743-746.
Nodal, M.; Kjær, I. et al. (1994). Craniofacial morphology in patients with multiple congenitally missing permanent teeth. European Journal of Orthodontics, 16(2): 104-109.

Nordgarden, H.; Jensen, J. L. et al. (2002). Reported prevalence of congenitally missing teeth in two Norwegian counties. Community. Dental Health, 19(4): 258-261.

O’Dowling, I. B. & McNamara, T. G. (1990). Congenital absence of permanent teeth among Irish school-children. Journal of the Irish Dental Association, 36(4): 136-138.

Ogaard, B. & Krogstad O. (1995). Craniofacial structure and soft tissue profile in patients with severe hypodontia. American Journal of Orthodontics and Dentofacial Orthopedics, 108(5): 472-477.

Park, J. H.; Okadakage, S. et al. (2010). Orthodontic treatment of a congenitally missing maxillary lateral incisor. Journal of Esthetic and Restorative Dentistry, 22(5): 297-312.

Peck, S.; Peck, L. et al. (1996). Prevalence of tooth agenesis and peg-shaped maxillary lateral incisor associated with palatally displaced canine (PDC) anomaly. American Journal of Orthodontics and Dentofacial Orthopedics, 110(4): 441-443.

Peck, S.; Peck, L. et al. (2002). Concomitant occurrence of canine malposition and tooth agenesis: evidence of orofacial genetic fields. American Journal of Orthodontics and Dentofacial Orthopedics, 122(6): 657-660.

Pereira, T. V.; Salzano, F. M. et al. (2006). Natural selection and molecular evolution in primate PAX9 gene, a major determinant of tooth development. Proceeding of the National Academy of Science of the USA, 103(15): 5676-5681.

Pilo, R.; Kaffe, I. et al. (1987). Diagnosis of developmental dental anomalies using panoramic radiographs. ASDC Journal Dentistry for Children, 54(4): 267-272.

Pinho, T. (2003). Agenesis of upper lateral incisors- case study: orthodontic and restorative procedures. Gnathos, 2(2): 35-42.

Pinho, T. (2004). Skeletal Class III in a case with upper lateral incisors agenesis. Gnathos, 5: 33-41.

Pinho T. & Lemos C. Dental repercussions of maxillary lateral incisor agenesis. European Journal of Orthodontics 2011; doi: 10.1093/ejo/cjr084.

Pinho, T.; Maciel, P. et al. (2010a). Familial aggregation of maxillary lateral incisor agenesis. Journal of Dental Research, 89(6): 621-625.

Pinho, T.; Maciel, P. et al. (2009). Developmental disturbances associated with agenesis of the permanent maxillary lateral incisor. British Dental Journal, 207(12): E25.

Pinho, T. & Neves, M. (2001). Tratamento da ausência congênita de incisivos maxílares quando a opção é manter ou abrir o espaço. Dental Sapiens, 1(1): 7-16.

Pinho, T.; Pollmann, C. et al. (2011b). Craniofacial Repercussions in Maxillary Lateral Incisors Agenesis. International Orthodontics, 9:274-285.

Pinho, T.; Silva-Fernandes, A. et al. (2010a). Mutational analysis of MSX1 and PAX9 genes in Portuguese families with maxillary lateral incisor agenesis. European Journal of Orthodontics, 32(5): 582-588.

Pinho, T.; Tavares, P. et al. (2005). Developmental absence of maxillary upper lateral incisors in the Portuguese population. European Journal of Orthodontics, 27(5): 443-449.

Pirinen, S.; Arte, S. et al. (1996). Palatal displacement of canine is genetic and related to congenital absence of teeth. Journal of Dental Research, 75(10): 1742-1746.
Prabhu, N. T.; John, R. et al. (1997). Rieger's syndrome: a case report. *Quintessence International*, 28(11): 749-52.

Ranta, R. & Tulensalo, T. (1988). Symmetry and combinations of hypodontia in non-cleft and cleft palate children. *Scandinavian Journal of Dental Research*, 96(1): 1-8.

Ravn, J. J. (1971). Aplasia, supernumerary teeth and fused teeth in the primary dentition. An epidemiologic study. *Scandinavian Journal of Dental Research*, 79(1): 1-6.

Rolling, S. (1980). Hypodontia of permanent teeth in Danish schoolchildren. *Scandinavian Journal of Dental Research*, 88(5): 365-369.

Rosa, M. & Zachrisson B. U. (2001). Integrating esthetic dentistry and space closure in patients with missing maxillary lateral incisors. *Journal of Clinic Orthodontic*, 35(4): 221-234.

Schalk-van der Weide, Y.; Beemer, F. A. et al. (1994). Symptomatology of patients with oligodontia. *Journal of Oral Rehabilitation*, 21(3): 247-261.

Schalk-Van Der Weide, Y.; Beemer, F. A. et al. (1994). Symptomatology of patients with oligodontia. *Journal of Oral Rehabilitation*, 21: 247-261.

Schalk-van der Weide, Y. & Bosman F. (1996). Tooth size in relatives of individuals with oligodontia. *Archives of Oral Biology*, 41(5): 469-472.

Schalk-van der Weide, Y.; Steen, W. H. et al. (1994). Reductions in size and left-right asymmetry of teeth in human oligodontia. *Archives of Oral Biology*, 39(11): 935-939.

Schalk-van der Weide, Y.; W. H. Steen, et al. (1992). Distribution of missing teeth and tooth morphology in patients with oligodontia. *ASDC Journal of Dentistry for Children*, 59(2): 133-140.

Schuffenhauer, S.; Leifheit, H. J. et al. (1999). De novo deletion (14)(q11.2q13) including PAX9: clinical and molecular findings. *Journal of Medical Genetics*, 36(3): 233-236.

Small, B. (1996). Esthetic management of congenitally missing lateral incisors with single-tooth implants: A case report. *Quintessence International*, 27(9): 585-590.

Spray, J. R.; Black, C. G. et al. (2000). The influence of bone thickness on facial marginal bone response: stage 1 placement through stage 2 uncovering. *Annals of Periodontology*, 5(1): 119-228.

Stockton, D. W.; Das, P. et al. (2000). Mutation of PAX9 is associated with oligodontia. *Nature Genetics*, 24(1): 18-19.

Swinnen, S.; Bailleul-Forestier, I. et al. (2008). Investigating the etiology of multiple tooth agenesis in three sisters with severe oligodontia. *Orthodontic Craniofacial Research*, 11(1): 24-31.

Tallon-Walton, V.; Manzanares-Cespedes, M. C. et al. (2007). Identification of a novel mutation in the PAX9 gene in a family affected by oligodontia and other dental anomalies. *European Journal of Oral Sciences*, 115(6): 427-432.

Tavajohi-Kermani, H.; Kapur, R. et al. (2002). Tooth agenesis and craniofacial morphology in an orthodontic population. *American Journal of Orthodontics and Dentofacial Orthopedics*, 122(1): 39-47.

Taylor, R. W. (1998). Eruptive abnormalities in orthodontic treatment. *Seminars in Orthodontics*, 4(2): 79-86.

Thilander, B. & Myrberg N. (1973). The prevalence of malocclusion in Swedish schoolchildren. *Scandinavian Journal of Dental Research*, 81(1): 12-21.
Thilander, B.; Odman, J. et al. (1994). Osseointegrated implants in adolescents. An alternative in replacing missing teeth? *European Journal of Orthodontics*, 16(2): 84-95.

Tummers, M. & Thesleff I. (2009). The importance of signal pathway modulation in all aspects of tooth development. *Journal of Experimental Zoology Part B: Molecular and Developmental Evolution*.

Tunc, E. S.; Bayrak, S. et al. (2011). Dental development in children with mild-to-moderate hypodontia. *American Journal of Orthodontics and Dentofacial Orthopedics*, 139(3): 334-338.

Vastardis, H. (2000). The genetics of human tooth agenesis: new discoveries for understanding dental anomalies. *American Journal of Orthodontics and Dentofacial Orthopedics*, 117(6): 650-656.

Vastardis, H.; Karimbux, N. et al. (1996). A human MSX1 homeodomain missense mutation causes selective tooth agenesis. *Nature Genetics*, 13(4): 417-421.

Wisth, P. J.; Thunold, K. et al. (1974). The craniofacial morphology of individuals with hypodontia. *Acta Odontologica Scandinav*, 32(4): 281-290.

Woodworth, D. A.; Sinclair, P. M. et al. (1985). Bilateral congenital absence of maxillary lateral incisors: a craniofacial and dental cast analysis. *American Journal of Orthodontics*, 87(4): 280-293.

Yaqoob, O.; Dibiase, A. T. et al. (2011) Relationship between bilateral congenital absence of maxillary lateral incisors and anterior tooth width. *American Journal of Orthodontics and Dentofacial Orthopedics*, 139(3): e229-233.

Yuksel, S. & Ucem T. (1997). The effect of tooth agenesis on dentofacial structures. *European Journal of Orthodontics*, 19(1): 71-78.

Zarone, F.; Sorrentino, R. et al. (2006). Prosthetic treatment of maxillary lateral incisor agenesis with osseointegrated implants: a 24-39-month prospective clinical study. *Clinical Oral Implants Research*, 17(1): 94-101.

Zhao, J.; Hu, Q. et al. (2007). A novel missense mutation in the paired domain of human PAX9 causes oligodontia. *American Journal of Medical Genetics*, 143A(21): 2592-2597.

Zhu, J. F.; Crevoisier, R. et al. (1996). Congenitally missing permanent lateral incisors in conjunction with a supernumerary tooth: case report. *Pediatric Dentistry*, 18(1): 64-66.

Zhu, J. F.; Marcushamer, M. et al. (1996). Supernumerary and congenitally absent teeth: a literature review. *Journal of Clinical Pediatric Dentistry*, 20(2):87-95.

Zirberman, Y.; Cohen, B. et al. (1990). Familial trends in palatal canines, anomalous lateral incisors, and related phenomena. *European Journal of Orthodontics*, 12: 135-139.
Orthodontics is a fast developing science as well as the field of medicine in general. The attempt of this book is to propose new possibilities and new ways of thinking about Orthodontics beside the ones presented in established and outstanding publications available elsewhere. Some of the presented chapters transmit basic information, other clinical experiences and further offer even a window to the future. In the hands of the reader this book could provide an useful tool for the exploration of the application of information, knowledge and belief to some orthodontic topics and questions.

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