

Management of maxillary midline diastema with emphasis on etiology

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The importance of the presence of a maxillary midline diastema resides in its position and the concern it causes to patients. This specific diastema has been attributed to genetic and environmental factors, even though it is often a normal feature of growth, especially in primary and mixed dentition.

The need for treatment is mainly attributed to esthetic and psychological reasons, rather than functional ones. Although it is often the case, treatment plans should not be selected empirically but rather should be based on adequate scientific documentation. Possible therapeutic approaches include orthodontics, restorative dentistry, surgery and various combinations of the above. The ideal treatment should seek to manage not only the diastema in question but also the cause behind it. Irrespective of the treatment alternative selected, permanent retention of stable results should be considered as a treatment objective.

The aim of this paper is to underscore the main etiological factors for the presence of a maxillary midline diastema and to illustrate the clinical and laboratory examinations required to recognize these factors. Furthermore, alternative treatment options are discussed depending on the etiology of the problem.

Keywords: maxillary midline diastema, dental spacing, etiology, treatment, relapse

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INTRODUCTION

The maxillary midline diastema is, indisputably, one of the dentoalveolar disorders that cause special concern to parents and patients, specifically given its position. Among five-year old children, during the period of primary dentition, maxillary midline diastema appears in 97% of cases¹ and, along with primate spaces, predicts a future development of the mixed and permanent dentition without crowding.² The presence of maxillary midline diastema is a normal characteristic in the development of the stomatognathic system in the mixed dentition period, especially in the initial phase of the eruption of permanent maxillary central incisors (“ugly duckling” stage).²⁻⁶ During the early mixed dentition this condition appears in 48.8% of children⁷ and decreases with age.^{3,5,7,8} In adults, the prevalence of maxillary

midline diastema ranges from 1.6% to 25.4%.^{5,9-13}

Most literature references to date promote the concept that maxillary midline diastema is of multifactorial etiology, considering only environmental factors.^{1,4,6,12,14,15} On the other hand, there are well documented studies that support the concept of a possible genetic predisposition for the maxillary midline diastema.^{12,16-18} Recently, Gass *et al.* (2003)¹⁸ suggested that the midline diastema is probably inherited by an autosomal dominant mode of inheritance.

Moyers (1988)¹⁹ studied 82 patients that presented maxillary midline diastema and reported the following causes: a) imperfect fusion at midline of premaxilla (32.9%), b) enlarged or malposed upper labial frenum (24.4%), c) midline diastema as part of normal growth (23.2%), d) congenitally missing lateral incisors (11%), e) supernumerary teeth at the midline (3.7%), f) unusually small teeth (2.4%), and g) combination of imperfect fusion and congenitally missing lateral incisors (2.4%). Other causes for the development of the maxillary midline diastema referred in literature involve: a) rotated teeth,⁴ b) parafunctional oral habits, such as thumb/finger sucking or abnormal tongue posture,^{1,4,20} c) orthodontic treatment, as in cases of rapid palatal expansion or false teeth movement,⁴ d) increased anterior overbite,^{1,21} e) distal or labial inclination of maxillary central incisors,^{1,4} f) generalized spacing,¹ and g) pathologic teeth migration due to periodontal disease.^{22,23}

Selecting the most appropriate treatment for the maxillary midline diastema is not always an easy decision, given that it presupposes having a sound diagnosis as well as

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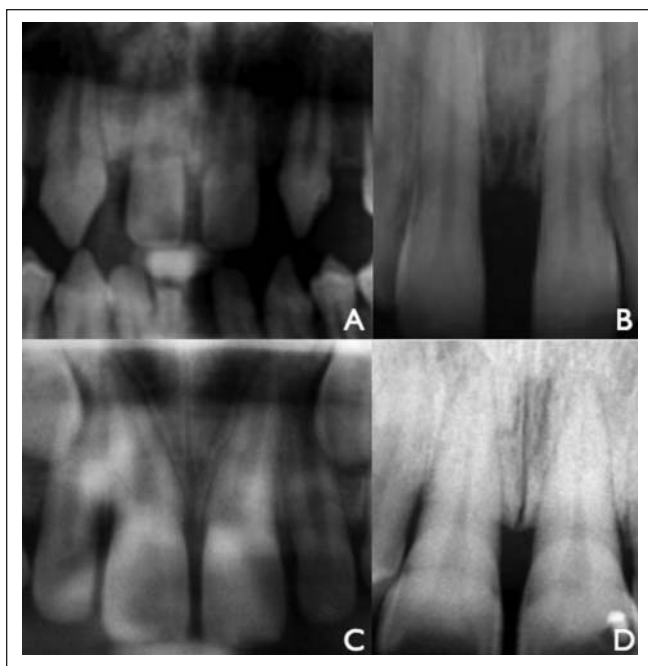


Figure 1. A—A normal V-shaped bone structure slightly bisected by the intermaxillary suture. The diastema is due to congenital absence of lateral incisors B—Abnormal, W-shaped, alveolar process between the maxillary central incisors. C—Abnormal suture wider than normal. D—Abnormal, spade-shaped, alveolar process between the maxillary central incisors.

recognizing and managing the cause of the problem. In addition, permanent retention of the result must be considered as part of the treatment.

MANAGEMENT OF THE CAUSE

The most common causes, referred to in relevant literature, for maxillary midline diastema and the possible treatment approaches in response to the etiology of this problem, are discussed below.

Imperfect fusion at midline of premaxilla (Fig 1, B–D).^{3,19,20,24} The existing gap is occupied by connective and epithelial tissue, while fibers of the maxillary labial frenum or gingival fibers (especially interdental) are often inserted and attached in that site. Normally, the interdental (transeptal) gingival fibers, along with other types of gingival fibers,

have the functional role of maintaining teeth in their position.^{20,25-28} The disturbance of the gingival fiber system can lead to a distal movement of the two maxillary central incisors, sometimes accompanied by lateral incisor rotation and ectopic eruption of the canines.²⁰ It must be mentioned that Popovich *et al.* (1977a, b)^{7,29} stated that an incomplete fusion of the intermaxillary suture is only a primary cause for the development of a diastema when other predisposing factors are also present. Additionally, Sullivan *et al.* (1996)³⁰ and Shashua and Artun (1999)¹⁷ did not confirm a relation between the diastema relapse and the presence of an imperfect fusion at the midline of the premaxilla.

The final diagnosis of an imperfect fusion must be based on a radiograph (Fig 1). It is important that the central ray be perpendicular to the alveolar process for a correct diagnosis.¹⁹ The alveolar process between the maxillary central incisors normally appears in the radiograph as a V-shaped structure, slightly bisected by the intermaxillary suture (Fig 1, A). The suture is considered abnormal when the radiograph shows a W-shaped bone between the maxillary central incisors (in cases where the bisection of the bone is relatively deeper) (Fig 1, B); or a suture that is wider than normal (approximately 2 mm) (Fig 1, C); or a circumscribed irregular ovoid (sometimes spade-shaped) alveolar process in this region (Fig 1, D). The W-shaped, and the spade-shaped alveolar processes are usually accompanied by a hypertrophic/inferiorly attached frenum.^{7,29}

Treatment is based on surgical excision of the fibers attached to the residual suture by proceeding with an osteotomy along the intermaxillary suture.³¹ This intervention must be performed after the orthodontic closure of the diastema, in order for tissue healing and fiber remodelling to occur in the new position of the teeth.^{20,32} In cases where relapse occurs, the same procedure must be repeated. Moreover, when the imperfect fusion makes the diastema closure impossible, the surgery must be performed before the closure of the diastema.³¹

Hypertrophic or malposed upper labial frenum (Fig 2).^{3,26,27,33,34} Normally, the frenum does not follow the growth of the alveolar process occurring during teeth eruption.^{14,35} This has as a result the appearance of a more nasally positioned frenum in accordance with age, while, practically, it



Figure 2. A—Diagnosis of an abnormal labial frenum by observation alone. B—Diagnosis of an abnormal labial frenum by stretching the upper lip and observing the ischemia caused to the interdental papilla. C—Diagnosis of an abnormal labial frenum by observation of an unusually wide frenum, with no apparent zone of attached gingiva along the midline.



Figure 3. Management of the maxillary midline diastema caused by an abnormal labial frenum. A—Result of the orthodontic treatment. B—Surgical intervention. C—Retention of the orthodontic appliances during the healing phase.

remains more or less in the same position.³⁶

There is remarkable consensus among scientists concerning the existence of a cause-effect relationship between the presence of hypertrophic or malposed maxillary labial frenum and the maxillary midline diastema. Shashua and Artun (1999)¹⁷ found that there is a correlation between the width of the diastema and the presence of an abnormal frenum.

Numerous authors supported that the maxillary labial frenum is associated with the pathogenicity of the maxillary midline diastema.^{3,14,27,33-35,37-39} The frenum fibers can sometimes be attached to the periosteum and the connective tissue of the residual intermaxillary suture, that might be present,^{3,14,35} while others may simply interrupt the continuity, particularly, of the interdental gingival fibers.^{20,24,38,40} In such cases, the frenum attachment does not “migrate nasally” or changes minimally with age²⁵ and it should not be expected that the midline diastema will spontaneously close with the eruption of the maxillary lateral incisors and canines, as it usually happens.^{1-3,19,21} It seems that the deeper within the tissues and the closer to the incisive papilla the frenum is attached, the more likely it is for it to cause a diastema.³⁶

On the other hand, Popovich *et al.* (1977a,b)^{7,29} argued that in cases with diastema, the hypertrophic frenum continues to develop more coronally as the alveolar process grows with teeth eruption, because the dentition exercises minimal or no pressure on the frenum. Tait (1924)⁴¹ supported that the frenum has no effect on the maxillary incisors. Ceremelo (1953)⁴² concluded that the presence of the frenum is not related with the presence or the width of the midline diastema. Finally, Bergstrom *et al.* (1973)⁴³ noted that the probability of long-term spontaneous diastema closure in patients with an abnormal frenum is the same, regardless of whether or not the frenum had been surgically excised. Consequently, further research is needed to determine the cause-effect relationship between the abnormal labial frenum and the maxillary midline diastema.

Considering diagnosis, sometimes, when frenum fibers are inserted quite deeply, the presence of an abnormal labial frenum can be diagnosed by observation alone (Fig 2A) or by stretching the upper lip and observing the ischemia caused to the interdental papilla (Fig 2B).^{19,27,37} Miller (1985)⁴⁴ recommended that the frenum should be character-

ized as pathologic when it is unusually wide or there is no apparent zone of attached gingiva along the midline (Fig 2C) or the interdental papilla shifts when the frenum is extended. However, evaluating the frenum (normal or pathologic) is sometimes rather difficult, especially in borderline cases.^{17,25,43} All clinical data should be assessed in relation to patient’s age, as well as to other parameters relevant to the problem.^{26,27}

The management of this specific condition initially involves the orthodontic closure of the diastema. Then, the frenum must be surgically removed^{13,26,44} and the orthodontic appliances must be retained in place during healing (Fig 3, A-C). By choosing this approach, the tissue expected to form in the new position will help retain the result of treatment.^{2,45} Nevertheless, occasionally, when the frenum is particularly hypertrophic and inhibits the orthodontic closure of the diastema, it is necessary to surgically reposition the frenum nasally before the end of orthodontic treatment.⁴⁵ The above options must be performed only when the diastema persists after the eruption of permanent canines, as, in most cases, the eruption of the canines leads to spontaneous closure of the maxillary midline diastema.^{2,27,35,46} Finally, it must be noted, that sometimes the pressure which is induced to the frenum fibers during the orthodontic approximation of the maxillary central incisors, may cause avascular necrosis, along with frenum and gingival fibers remodelling, making the surgical intervention useless.^{19,25}

Diastema as part of normal development. Permanent maxillary central incisors can normally erupt with a diastema that will be reduced in size with the eruption of the lateral incisors and will completely disappear with the eruption of the canines.^{1-3,19} This happens because each permanent incisor and canine is 2-3 mm wider than its primary predecessor. Therefore, the maxillary midline diastema is frequently, not only physiologic, but necessary.² If there is no pathological condition related to these specific teeth or major deviations from normal teeth size, spontaneous closure of the diastema should be considered certain in most cases.^{3,5,8,27} If, however, this does not happen, then intervention by the dentist may be necessary.

Congenitally missing lateral incisors.¹⁹ In these cases, the maxillary central incisors tend to occupy the existing space and move distally resulting in local spacing in the



Figure 4. Management of the maxillary midline diastema, caused by congenitally missing lateral incisors, including orthodontic closure of the diastema and creating the appropriate space for the placement of a Maryland-type fixed partial denture.

maxillary anterior region. Early diagnosis of the congenital absence, based on a radiograph examination, is a matter of great importance for the progress of these cases.

The diastemas due to congenital absence of lateral incisors can be treated orthodontically with closure of the diastema and proper guidance of the canines to the position of the missing lateral incisors and of the posterior teeth mesially. In such cases, it is obligatory to achieve an Angle II occlusal relation. Selective grinding of the incisive and palatal canine cusps and of the palatal cusps of the first premolars and restorations with resin composite must be performed in order to transform canines and first premolars into lateral incisors and canines, respectively. This is essential for satisfying patient's esthetic requirements, as well as proper function of the stomatognathic system.⁴⁷⁻⁴⁹ In certain cases, it might be necessary to place a crown on the canine, in order to look more similar, in shape and color, to the lateral incisor.⁵⁰ Alternative treatment options for the maxillary midline diastema caused by congenitally missing lateral incisors are to close the diastema and create the appropriate space for placing tooth-supported restorations (Fig 4)⁵¹ or single-tooth implants.⁵² The last options are of particular significance in cases of unilateral tooth absence, mainly because of the difficulties faced during orthodontic treatment, when trying to achieve dental arch symmetry. The selection of the appropriate treatment option in cases with congenitally missing lateral incisors, depends on the present malocclusion, on the anterior teeth relationship, on the

specific needs concerning the available space, on the condition of the adjacent teeth, on the tooth-size relationship and on the size and shape of the canine.⁵⁰⁻⁵²

Supernumerary teeth at the midline.^{53,54} A mesiodens is usually interposed between the roots of the maxillary central incisors and does not allow them to move to the midline and close the diastema. Tay *et al.* (1984)⁵⁵ stated that when supernumerary teeth are normally orientated, they are more likely to cause a delayed eruption of permanent teeth, while when they are inverted, they usually cause bodily displacement of the permanent incisors, torsion and midline diastema. Diagnosis is exclusively based on the radiographic examination, unless the mesiodens has erupted. Treatment involves the removal of the supernumerary tooth as soon as diagnosed, without causing injury to the adjacent teeth.⁵⁶ Often, timely removal of the impediment allows central incisors' eruption forces to close the diastema, without any further intervention.⁵⁷ If this does not occur, then the diastema is usually corrected by using orthodontic forces.

Small teeth.^{1,4,15,58} Approximately 5% of the population have some degree of disproportion among the size of individual teeth.² The small size or conical shape of the crown of upper lateral incisors is the commonest tooth size abnormality. Sometimes, this abnormality is responsible for the development of a local diastema between the maxillary central incisors, since the space created mesially to the lateral incisors allows the central incisors to move distally. A well-known method for the diagnosis of size disproportions

between maxillary and mandibular teeth is the Bolton's analysis. Another diagnostic method for teeth-size abnormalities is the diagnostic cast setup, where dental casts are mounted on articulators.¹

The treatment of this condition depends on the age when the problem is revealed, on the size and shape of the crown and root of the tooth involved, on the position and extension of the problem and on the occlusal relationship. If the root provides appropriate periodontal support, then, after closing the diastema and properly managing the available space, a crown is placed on the abnormal tooth or the tooth is restored with resin composite. If the root is not satisfactory, and the problem becomes apparent before the canines erupt, the case may be treated as if the maxillary lateral incisors were congenitally missing. However, if the diagnosis is made after the eruption of the canines, it is preferable to select a restorative treatment plan and use a fixed partial denture for splinting the teeth, rather than orthodontic treatment.⁵⁹

RETENTION OF THE RESULT

Relapse of the maxillary midline diastema appears, according to Sullivan *et al.* (1996),³⁰ in almost 34% of cases, while, according to Shashua and Artun (1999)¹⁷ this rate rises to 50%. The reason for relapse is the placement of teeth in a position where no equilibrium exists with their functional environment.^{60,61} In most of these cases, the factor disturbing this equilibrium is still present after treatment.^{4,15,62}

Shashua and Artun (1999)¹⁷ concluded that the most important risk factors for relapse are the increased pretreatment width of the midline diastema, the presence of a family member with a similar condition, and the presence of more than one diastemas in the maxillary anterior region. However, according to Sullivan *et al.* (1996)³⁰, no pre-treatment predictors of relapse can be established.

As a general rule, treatment is unlikely to produce assured and stable results, thus the use of permanent retention for a considerable period of time or even for life, is essential in almost every case.^{45,61,63} The most appropriate method for achieving long-term retention after orthodontic treatment is through the use of palatally bonded multi-stranded stainless steel wire retainers, which allow teeth to maintain their physiologic mobility and are easy to fabricate.^{45,64} In cases where the retainer interferes in functional movements of the mandible, it can be bonded cervically or within a shallow rim constructed in the enamel of teeth.⁴⁵

DISCUSSION

In a recent study conducted by Kokich *et al.* (2006),⁶⁵ laypeople did not rate a midline diastema as unattractive until distance between the contacts of the central incisors was 2.0 mm. The above study, also concluded that relapse of a diastema after orthodontic treatment that is less than 1 mm, will not be rated as unattractive by the orthodontic patient. However, according to Bernabe and Flores-Mir (2007),⁶⁶ maxillary anterior dentoalveolar spacing had the most negative influence on self-perceived dental appearance among young adults. Consequently, the decision regarding any

intervention applied to a maxillary midline diastema is not easy. A variety of factors, including the width and the cause of the diastema, and the possible treatment options for each case, should be critically evaluated by the patients, their parents, and the dentist in order to determine the need for treatment, and select the appropriate treatment plan.

When a treatment approach of a maxillary midline diastema is to be implemented, the first and probably most important stage of treatment is the diagnosis of the cause of the problem. The dentist should evaluate several parameters to reach a sound diagnosis. These are the patient's age and normal development, any malocclusion present, teeth size, relations with adjacent teeth, antagonist teeth and their osseous base, the presence of diastemas in other arch segments and, finally, the presence of a concomitant pathology. When the diagnosis is established, the appropriate therapy should include management of the causative factors, along with the diastema correction and the permanent retention of the result.^{17,30,45} This is the only way to fully satisfy the patient's needs and maintain long-term stable results.

As a general guideline, only maxillary midline diastemas exceeding 2 mm are unlikely to close spontaneously following the eruption of permanent lateral incisors and canines, while an initial diastema less than 2 mm hardly ever remains.^{2,25} Therefore, the treatment of the maxillary midline diastema is usually postponed until the eruption of the permanent canines, but it may start earlier, depending on the cause of the diastema or in cases with a relatively large diastema.^{6,67} The treatment of the maxillary midline diastema may start before the eruption of permanent canines in cases where the diastema is due to congenitally missing lateral incisors, the presence of a mesiodens, odontoma or other pathology in the midline, or small teeth. Main indications for early closure of a maxillary midline diastema, i.e. during the stage of mixed dentition, are: a) an urgent aesthetic demand by the patient and b) a central incisor position that inhibits the eruption of the lateral incisors or canines, since the lateral incisors might have been displaced into the space where canines normally erupt (Fig 5).² Retaining the result of treatment is a particularly difficult issue, especially if lateral incisors and canines have not yet erupted.

Concerning biomechanics, elastic bands should never be used around teeth to close a maxillary midline diastema, because of the likelihood to slide apically and destroy the periodontal ligament and occasionally leading to loss of the maxillary central incisors.⁶⁸ Usually, in cases with a diastema that exceeds 2 mm, tipping movements are not sufficient and it is necessary to move teeth bodily and treat by using fixed orthodontic appliances. Prognosis in such cases is better when only mesiodistal and not palatal repositioning is required.² It should always be kept in mind that when tipping takes place, it usually results in diastema relapse and, therefore, it is indispensable to apply permanent retention.^{26,44}

In some cases, closure of a maxillary midline diastema or other diastemas in the maxillary anterior region may be achieved with minimal preparation veneers⁶⁹ or through teeth restorations with composite resin.⁷⁰ However, the long-term



Figure 5. Early orthodontic treatment of a large maxillary midline diastema.

prognosis of these therapeutic approaches must be further investigated.⁷¹ In particular, the cases where these options can be performed are when: a) the patient does not want to undergo orthodontic treatment, b) there are other aesthetic problems present as well (e.g. *amelogenesis imperfecta* or discoloration), and c) treatment requires combined orthodontic and restorative treatment, in cases with a very large diastema.⁷²

With regards to the patient's acceptance of the treatment plan, according to Almog *et al.* (2004)⁷³ a total of 87,5% of their subjects stated that, compared to other consultation methods, they prefer the method of computer-imaging simulation, because it helps them understand the proposed treatment plan for closing diastemas in the maxillary anterior region.

CONCLUSIONS

1. Maxillary midline diastema is a common occurrence, especially before the eruption of permanent canines.
2. Its presence has been attributed by most authors to environmental factors, but according to recent studies, genetic predisposition seems to have great importance.
3. The cause-effect relationship between the abnormal labial frenum or the imperfect fusion at midline of pre-maxilla and the maxillary midline diastema needs to be further investigated.
4. The clinician should evaluate various parameters, including patient's age and stage of growth, in order to define the final diagnosis.

5. Before the adoption of any treatment plan, it is crucial to precisely determine if there is "actual" need for treatment.
6. Treatment timing primarily depends on the cause of the problem and must always be individualized.
7. In most cases, treatment requires a combined approach, performed after the eruption of all permanent teeth.
8. The ideal option is the one that addresses the diastema, along with the cause of the problem.
9. In almost all cases, permanent retention of the result of treatment is inevitable for satisfying long-term results.

REFERENCES

1. Oesterle LJ, Shellhart WC. Maxillary midline diastemas: a look at the causes. *J Am Dent Assoc*, 130: 85–94, 1999.
2. Proffit W, Fields H. *Contemporary Orthodontics*. 3rd ed. Mosby, St. Louis, 77, 87, 170, 429–30, 463–65, 2000.
3. Gardiner JH. Midline spaces. *Dent Pract Dent Rec* 17: 287–298, 1967.
4. Bishara SE. Management of diastemas in orthodontics. *Am J Orthod*, 61: 55–63, 1972.
5. Richardson ER, Malhotra S, Henry M, Little RG, Coleman H. Biracial study of the maxillary midline diastema. *Angle Orthod*, 43: 438–443, 1973.
6. Huang WJ, Creath CJ. The midline diastema: a review of its etiology and treatment. *Pediatr Dent*, 17: 171–179, 1995.
7. Popovich F, Thompson GW, Main PA. The maxillary interincisal diastema and its relationship to the superior labial frenum and intermaxillary suture. *Angle Orthod*, 47: 265–271, 1977a.
8. Weyman J. The incidence of median diastema during the eruption of the permanent teeth. *Dent Pract Dent Rec*, 17: 276–278, 1967.

9. Lavelle CL. The distribution of diastemas in different human population samples. *Scand J Dent Res*, 78: 530–534, 1970.
10. McVay TJ, Latta GH Jr. Incidence of the maxillary midline diastemas in adults. *J Prosthet Dent*, 52: 809–811, 1984.
11. Steigman S, Weissberg Y. Spaced dentition. An epidemiologic study. *Angle Orthod*, 55: 167–176, 1985.
12. Nainar SM, Gnanasundaram N. Incidence and etiology of midline diastema in a population in south India (Madras). *Angle Orthod*, 59: 277–282, 1989.
13. Brunelle JA, Bhat M, Lipton JA. Prevalence and distribution of selected occlusal characteristics in the US population, 1988–1991. *J Dent Res*, 75: 706–713, 1996.
14. Baum AT. The midline diastema. *J Oral Med*, 21: 30–39, 1966.
15. Becker A. The median diastema. *Dent Clin North Am*, 22: 685–710, 1978.
16. Schmitt E, Gillenwater JY, Kelly TE. An autosomal dominant syndrome of radial hypoplasia, triphalangeal thumbs, hypospadias, and maxillary diastema. *Am J Med Genet*, 13: 63–69, 1982.
17. Shashua D, Artun J. Relapse after orthodontic correction of maxillary median diastema: a follow-up evaluation of consecutive cases. *Angle Orthod*, 69: 257–263, 1999.
18. Gass JR, Valiathan M, Tiwari HK, Hans MG, Elston RC. Familial correlations and heritability of maxillary midline diastema. *Am J Orthod Dentofac Orthop*, 123: 35–39, 2003.
19. Moyers R. *Handbook of Orthodontics*. 4th ed. Year Book Medical Publishers, Chicago, USA, 348–360, 1988.
20. Stublely R. The influence of transseptal fibers on incisor position and diastema formation. *Am J Orthod*, 70: 645–662, 1976.
21. Popovich F, Thompson GW. Maxillary diastema: indications for treatment. *Am J Orthod*, 75: 399–404, 1979.
22. Rahilly G, Crocker C. Pathological migration: an unusual cause of midline diastema. *Dent Update*, 30: 547–549, 2003.
23. Cirelli JA, Cirelli CC, Holzhausen M, Martins LP, Brandao CH. Combined periodontal, orthodontic, and restorative treatment of pathologic migration of anterior teeth: a case report. *Int J Periodontics Restorative Dent*, 26: 501–506, 2006.
24. Ferguson MW, Rix C. Pathogenesis of abnormal midline spacing of human central incisors. A histological study of the involvement of the labial frenum. *Br Dent J*, 154: 212–218, 1983.
25. Edwards JG. The diastema, the frenum, the frenectomy: a clinical study. *Am J Orthod*, 71: 489–508, 1977.
26. Edwards JG. Soft-tissue surgery to alleviate orthodontic relapse. *Dent Clin North Am*, 37: 205–225, 1993.
27. Lindsey D. The upper mid-line space and its relation to the labial frenum in children and in adults. A statistical evaluation. *Br Dent J*, 143: 327–332, 1977.
28. Konstantinidis A. *Periodontology*. Vol. 1. Konstantinidis A, Thessaloniki, 77, 2003.
29. Popovich F, Thompson GW, Main PA. Persisting maxillary diastema: differential diagnosis and treatment. *Dent J*, 43: 330–333, 1977b.
30. Sullivan TC, Turpin DL, Artun J. A postretention study of patients presenting with maxillary median diastema. *Angle Orthod*, 66: 131–138, 1996.
31. Kraut R, Payne J. Osteotomy of intermaxillary suture for closure of median diastema. *J Am Dent Assoc*, 107: 760–761, 1983.
32. Proffit W, Fields H. *Contemporary Orthodontics*. 2nd ed. Mosby-Year Book, St. Louis; 128–129, 486, 1993.
33. Angle EH. *Treatment of malocclusion of the teeth*, 7th ed. S.S. White dental manufacturing. Co, Philadelphia; 167, 1907.
34. Sicher H. *Oral Anatomy*. 2nd ed. CV Mosby Company, St. Louis; 73–75, 1952.
35. Dewel BF. The labial frenum, midline diastema, and palatine papilla: a clinical analysis. *Dent Clin North Am*, 175–184, 1966.
36. Diaz-Pizan ME, Lagravere MO, Villena R. Midline diastema and frenum morphology in the primary dentition. *J Dent Child*, 73: 11–14, 2006.
37. Adams CP. The relation of spacing of the upper central incisors to abnormal labial frenum and other features of the dento-facial complex. *Dent Pract Dent Rec*, 74: 72–86, 1954.
38. Campbell PM, Moore JW, Matthews JL. Orthodontically corrected midline diastemas. A histologic study and surgical procedure. *Am J Orthod*, 67: 139–158, 1975.
39. Henry SW, Levin MP, Tsaknis PJ. Histologic features of the superior labial frenum. *J Periodontol*, 47: 25–28, 1976.
40. Scott JH, Symons NB. *Introduction to dental anatomy*. 7th ed. Churchill Livingstone, Edinburgh; 264, 1974.
41. Tait CW. The median frenum of the upper lip and its influence on the spacing of the upper central incisor teeth. *Dent Cosmos*, 76: 991–992, 1924.
42. Ceremelo PJ. The superior labial frenum and the midline diastema and their relation to growth and development of the oral structures. *Am J Orthod*, 39: 120–139, 1953.
43. Bergstrom K, Jensen R, Martensson B. The effect of superior labial frenectomy in cases with midline diastema. *Am J Orthod*, 63: 633–638, 1973.
44. Miller PD Jr. The frenectomy combined with a laterally positioned pedicle graft. Functional and esthetic considerations. *J Periodontol*, 56: 102–106, 1985.
45. Zachrisson BU. *Orthodontics and periodontics*. In: Lindhe J, 3rd ed. *Clinical periodontology and implant dentistry*. Munksgaard, Copenhagen, 741–793, 1997.
46. Finn SB. *Clinical Pedodontics*. WB Saunders, Philadelphia; 416–418, 1971.
47. Miller WB, McLendon WJ, Hines FB 3rd. Two treatment approaches for missing or peg-shaped maxillary lateral incisors: a case study on identical twins. *Am J Orthod Dentofac Orthop*, 92: 249–256, 1987.
48. Thordarson A, Zachrisson BU, Mjor IA. Remodelling of canines to the shape of lateral incisors by grinding: a long-term clinical and radiographic evaluation. *Am J Orthod Dentofac Orthop*, 100: 123–132, 1991.
49. Millar BJ, Taylor NG. Lateral thinking: the management of missing maxillary lateral incisors. *Br Dent J*, 179: 99–106, 1995.
50. Kokich VO Jr, Kinzer GA. Managing congenitally missing lateral incisors. Part I: Canine substitution. *J Esthet Restor Dent*, 17: 5–10, 2005.
51. Kinzer GA, Kokich VO Jr. Managing congenitally missing lateral incisors. Part II: tooth-supported restorations. *J Esthet Restor Dent*, 17: 76–84, 2005a.
52. Kinzer GA, Kokich VO Jr. Managing congenitally missing lateral incisors. Part III: single-tooth implants. *J Esthet Restor Dent*, 17: 202–210, 2005b.
53. Mason C, Rule DC. Midline supernumeraries: a family affair. *Dent Update*, 22: 34–35, 1995.
54. Tyrologou S, Koch G, Kuroi J. Location, complications and treatment of mesiodentes—a retrospective study in children. *Swed Dent J*, 29: 1–9, 2005.
55. Tay F, Pang A, Yuen S. Unerupted maxillary anterior supernumerary teeth: report of 204 cases. *J Dent Child*, 51: 289–294, 1984.
56. Mucedero M, Ballanti F, De Toffol L. Supernumerary teeth and mental retardation: the importance of early surgical intervention. *Eur J Paediatr Dent*, 7: 45–49, 2006.
57. Fernandez Montenegro P, Valmaseda Castellon E, Berini Aytés L, Gay Escoda C. Retrospective study of 145 supernumerary teeth. *Med Oral Patol Oral Cir Bucal*, 11: E339–344, 2006.
58. Ooshima T, Ishida R, Mishima K, Sobue S. The prevalence of developmental anomalies of teeth and their association with tooth size in the primary and permanent dentitions of 1650 Japanese children. *Int J Paediatr Dent*, 6: 87–94, 1996.
59. Kokich VG, Spear FM. Guidelines for managing the orthodontic-restorative patient. *Semin Orthod*, 3: 3–201, 1997.
60. Proffit WR. Equilibrium theory revisited: Factors influencing position of the teeth. *Angle Orthod*, 48: 175–186, 1978.

61. Zachrisson BU. Use of self-ligating brackets, superelastic wires, expansion/proclination, and permanent retention—a word of caution. *World J Orthod*, 7: 198–206, 2006.
62. Mulligan TF. Diastema closure and long-term stability. *J Clin Orthod*, 37: 560–574, 2003.
63. Lang G, Alfter G, Goz G, Lang GH. Retention and stability—taking various treatment parameters into account. *J Orofac Orthop*, 63: 26–41, 2002.
64. Karaman AI, Polat O, Buyukyilmaz T. A practical method of fabricating a lingual retainer. *Am J Orthod Dentofac Orthop*, 124: 327–330, 2003.
65. Kokich VO, Kokich VG, Kiyak HA. Perceptions of dental professionals and laypersons to altered dental esthetics: asymmetric and symmetric situations. *Am J Orthod Dentofac Orthop*, 130: 141–151, 2006.
66. Bernabe E, Flores-Mir C. Influence of anterior occlusal characteristics on self-perceived dental appearance in young adults. *Angle Orthod*, 77: 831–836, 2007.
67. Campbell A, Kindelan J. Maxillary midline diastema: a case report involving a combined orthodontic/maxillofacial approach. *J Orthod*, 33: 22–27, 2006.
68. Redlich M, Galun EA, Zilberman Y. Orthodontic-prosthetic treatment to replace maxillary incisors exfoliated because of improper use of orthodontic elastics: a case report. *Quintessence Int*, 28: 241–244, 1997.
69. Nazarian A. Closing the gap with minimal preparation veneers. *Dent Today*, 25: 70–71, 2006.
70. Willhite C. Diastema closure with freehand composite: controlling emergence contour. *Quintessence Int*, 36: 138–140, 2005.
71. Peumans M, Van Meerbeek B, Lambrechts P, Vanherle G. The 5-year clinical performance of direct composite additions to correct tooth form and position. I. Esthetic qualities. *Clin Oral Investig*, 1: 12–18, 1997.
72. Beasley WK, Maskeroni AJ, Moon MG, Keating GV, Maxwell AW. The orthodontic and restorative treatment of a large diastema: a case report. *Gen Dent*, 52: 37–41, 2004.
73. Almog D, Sanchez Marin C, Proskin HM, Cohen MJ, Kyrkanides S, Malmstrom H. The effect of esthetic consultation methods on acceptance of diastema-closure treatment plan: a pilot study. *J Am Dent Assoc*, 135: 875–881, 2004.