Stability of Diastemas Closure after Orthodontic Treatment

Marcos Jimmy Carruitero Honores

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/intechopen.82480

Abstract

The prevalence of diastemas varies greatly according to age and ethnic group. In permanent dentition, it varies from 1.7 to 38%. Its etiology is multifactorial. In the deciduous and mixed dentition phases, interincisal diastemas are considered normal. There are several approaches used in the treatment of anterosuperior diastemas, which vary according to the present etiologic factor. Orthodontic treatment also has the function of treating any other associated occlusal problem and helping in the elimination of parafunctional habits. Some authors agree that orthodontic closure of diastemas without subsequent surgery for removing the abnormal labial brake greatly increases the frequency of relapse in the postretention period, while others concluded that the fibrotomy of periodontal fibers together with the retainer had a positive effect on the stability of space closure. Buccal and lingual teeth inclinations have greater tendency to relapse, while mesial and distal movements, with a period of containment, are stable movements. Removable retainers are not considered a good choice. Depending on the type of initial malocclusion, the use of retainers throughout life is recommended. Fixed retention is often cited as the only satisfactory method to promote stability at the closure of previous diastemas.

Keywords: diastemas closure, stability, diastema, relapse, corrective orthodontics

1. Introduction

The presence of diastemas in the anterosuperior region used to be considered unpleasant, being one of the main reasons why patients seek orthodontic treatment. However, some studies report that the stability of the orthodontic closure of the diastemas, particularly of the median diastema in the permanent denture phase, is low. Long-term stability is one of the main objectives in orthodontic treatment; however, it is one of the most difficult to reach.
In the anterosuperior region, this stability is especially desired and verified by patients. Most of the publications on anterosuperior diastemas consist of clinical cases and reviews of the literature on the etiology of the problem and its possible treatments. But, there is a shortage in the literature of studies on the stability of the treatment of the upper interincisal subjects. The present chapter seeks to evaluate the recurrence of the upper interincisal diastemas, the factors associated to the relapse and stability, some treatment proposals, and considerations for retention.

2. Definition and epidemiology of diastemas

At the beginning of the last century, Angle [1] described median diastema as a common form of malocclusion where there is a space between the upper central incisors and, very rarely, between the lower central incisors. According to the author, the width of this space usually varies from 1 to 4 mm and always presents an unpleasant appearance, and may interfere with speech, depending on the width. Later, according to Bishara [2], diastemas were described as just clinically visible spaces between two or more adjacent teeth.

In publishing the Six Keys for normal occlusion, Andrews [3] stated in Key V that there must be fair points of contact throughout the dental arch, in the absence of dental size discrepancy. According to this information, diastemas are also considered a malocclusion by Andrews.

2.1. General prevalence

The prevalence of diastemas varies greatly according to age and ethnic group [4, 5]. In permanent dentures, the prevalence of diastemas varies from 1.7 to 38% [4].

2.2. Prevalence according to age

The literature demonstrates that the prevalence of diastemas decreases with age, mainly due to the development of occlusion. Richardson et al. [6] observed 5307 children between 6 and 14 years of age and, as a result, obtained a prevalence of 38% of children with median diastemas above 6 years of age, 56% at 8 years, and 18% at 14 years.

In a longitudinal study, Bergström et al. [7] aimed to observe the behavior of median diastemas in relation to the presence of the labial frenulum, in children of age 9 years on average, followed up after 2, 5, and 10 years. In the first two follow-ups, the group submitted to the removal of the labial frenulum presented a larger number of cases with closed diastemas than the control group. However, 10 years after the first consultation, there was no difference between groups. The authors found that the number of children with diastema declined with age and that there were a considerable number of individuals where diastemas closed from 14 to 19 years.

Steigman and Weissberg [8] found a prevalence of 50% in 1279 adolescents with spaced permanent dentures, ranging in age from 12 to 18 years. On the other hand, Steigman et al. [9]
found that the number of diastemas per individual decreased with age, but 79% of the pre-existing spaces remained, resulting in a percentage of 38% of young adults with spacing between the teeth.

2.3. Prevalence according to gender

Richardson et al. [6] observed a higher percentage of 6-year-old girls with median diastemas; however, at age 14, the opposite occurred. The authors believe that this is due to an earlier maturation of the girls in relation to the boys.

The findings of Steigman and Weissberg [8] corroborate those of the other authors, since they found dimorphism only in the subgroup between 14 and 16 years. There was no difference between genders in the 12- to 14-year-old and 16- to 18-year-old groups. Another study published by Steigman et al. [9] supported this finding, demonstrating that there was no difference between genders regarding the number of spaces in the arches in a sample of patients aged 16–22 years. It was also found that the number of diastemas decreased with age in both genders, but this occurred earlier in females than in males.

2.4. Prevalence according to race

In an epidemiological study on the prevalence of diastemas in white and black ethnic groups, Richardson et al. [6] observed, in a sample of children between 6 and 14 years of age, a higher frequency of diastemas in blacks at almost all ages. A prevalence of 23% of blacks at 14 years of age with diastema was found, differing from 14% of whites of this age with central interincisive spaces.

Similarly, Lavelle [4] found a higher incidence of central interincisive diastemas in melanodermas (5.5%) than in leucodermas (3.4%) and a lower incidence in xanthomas (1.7%). Likewise, McVay and Latta [5] found a very significant difference between the prevalence of blacks (29%) compared to the percentage of 20% of adults presenting this space in both the white and yellow race.

3. Diastemas and occlusion development

It is a consensus in the literature that, during the development of occlusion, in the deciduous and mixed dentition stages, the presence of diastemas is a characteristic of normality [2, 10–14]. Nevertheless, Richardson et al. [6] suggested that median diastemas are more than a phase of development eliminated over time, due to the high prevalence found in 18% of adolescents with medium diastemas. Edwards [15] stated that if the median space is greater than 2 mm, it is unlikely to close spontaneously. It was observed in a longitudinal study in adolescents that there was stability in most of the existing diastema, although some were eliminated even after the eruption of permanent canines [9].

3.1. Primary dentition

Authors have suggested that these spaces in the primary dentition are normal and would have the function of assisting in the eruption of permanent teeth [10, 16]. In describing the
biogenetic course of the deciduous dentition, Baume [11] classified the dental arches according to the spaces designated by him as type I, with anterior spaces, and type II, without such spaces. In this work, the author cites that one type cannot progress to the other.

Moyers [14] reported that there is a generalized spacing in the anterior region of the upper and lower arches in the deciduous dentition, which increases significantly after this dentition has been completed.

3.2. Mixed dentition

In the mixed dentition phase, the diastemas continue to appear as a physiological characteristic, mainly the diastema between the permanent maxillary incisors. The germs of these teeth remain separated within the maxilla, respecting the intermaxillary suture interposed between them [16].

Broadbent [12] published a study of 5000 individuals observed over 12 years. The author called the “ugly duckling stage” the period that ranges from the eruption of the upper incisors, around the age of 7 years, until approximately 10 years of age, when the upper canines erupt. At this stage, the lateral incisors remain with the converging roots until the maxilla size is sufficient to assume a more vertical position. With sufficient increase in the size of the subnasal area, and in the presence of normal growth, the canines move down, forward, and laterally to the lateral incisor roots. Correction of the ugly duckling stage will occur in the period between 8 and 12 years of age.

Burstone [13], observing the normal changes during development, identified that during the “ugly duckling phase” of Broadbent [12], protrusion of the crowns, overjet, and anterosuperior spaces occurs. He also reported that the upper central interincisive diastema would be closed with the eruption of the other permanent teeth, especially the canines, and that the overjet of the incisors would be corrected by the pressure of the labial musculature and the eruption of the other permanent teeth.

3.3. Permanent dentition

In the permanent dentition, the etiology of the diastemas is multifactorial [2, 16–19] and can be associated to the following factors: abnormal labial frenulum [1, 2, 16, 17, 20–22], microdontia, agenesis of maxillary incisors [2, 10, 16, 17, 20], dental discrepancy [2, 17, 23], shape of the anterior teeth (barrel) [10], brachyfacial pattern [17], a positive tooth-bone discrepancy [2, 16, 17], overbite, congenital anomalies such as soft tissue fissure, hybrid brake, or supernumerary teeth [2, 10, 16, 24], cysts and tumors [2, 24], periodontal disease [2, 25], macroglossia or neuromuscular imbalance of the tongue [10], acromegaly [26, 27], and orthodontic treatment [2, 20, 24].

The etiology of diastemas is also very well explored in a review article published by Bishara [2]. In this study, the authors divided diastemas into two categories, according to the etiology: (1) those not caused by orthodontic treatment, present before its accomplishment; and (2) those that appeared during or after orthodontic treatment. In the first category, the etiological factors mentioned are: physiological spaces in the deciduous dentition; developmental spaces in the mixed dentition phase, which are closed, according to the authors, after the canine
eruption; genetic factors such as large jaws and small teeth; tooth size, interarch discrepancy, agenesis, and micro-diseases; characteristics relating to ethnic groups; low insertion of the upper labial frenulum, preventing the mesial migration of the maxillary central incisors during canine eruption; dental rotations; supernumerary teeth, among which a classic example is the mesiodens; pathological conditions, such as proximal caries, periodontitis, cysts, and tumors; and, finally, the deleterious habits, exemplified by the lingual interposition and sucking lip. In the second category, the author refers to dental extractions and occasional dental size discrepancies, caused by extractions as diastematic agents during orthodontic treatment.

Steigman et al. [9] evaluated the stability of permanent denture spaces during adolescence in untreated patients, and also investigated the association of tooth spacing with tooth size and dental arch dimensions. They observed that women with spaced dentition had smaller dental widths and similar arch dimensions than those without dental spacing. In them, the spaces were equally distributed in both arches. On the other hand, in men, there was an equivalence of the dental dimensions, but the intercanine distances and superior interpremolars were greater in those with a spaced dentition. This is the reason why, in males, a greater number of spaces were found in the maxilla than in the mandible.

Oesterle and Shellhart [27] mentioned that the presence of generalized spaces in the dental arches may be the result of discrepancies between the size of the teeth and their respective apical bases, muscular imbalances, deleterious habits, and loss or absence of teeth. The large jaw and/or jaw combination with normal or slightly reduced teeth size is related to inherited characteristics, but may also be a sign of endocrine imbalances, which result in excess growth hormone, such as acromegaly.

Gass et al. [18], when evaluating the correlation between the heredity and the presence of the medium diastemas in leucodermas and melanodermas, found that the genetic expression was more significant for the whites than for the black ones, where the existence of an interincisive diastema is more related to environmental factors, such as excessive protrusion of the incisors, a predisposing periodontal tissue, habits, and absence of teeth. However, in relation to the stability of the treatment, an association between heredity and median diastema recurrence has been reported [28].

In the same year, Mondelli et al. [24], in a comprehensive review work describing the etiology and the various diastema treatments, add that there are diastemas of iatrogenic etiology. As examples, the authors cited the rapid expansion of the maxilla and a type of mechanics inadequate for diastema closure, where an elastic band is positioned on the central incisors, but slides in the cervical direction, causing periodontal damage and root approximation, with consequent divergence of the crowns, making the diastema even wider.

4. Treatment of anterosuperior diastemas

In the deciduous and mixed dentition phases, interincisive diastemas are considered normal [11–13, 16]. However, in the permanent dentures, diastema is frequently associated with several occlusal problems, which include missing teeth, dental anomalies, abnormal bone
structures, and excessive horizontal and/or vertical trespass [24]. It should be remembered that the diastema of racial and genetic etiologies are considered normal and the treatment will be dispensable, unless the patient considers the diastema an esthetic problem [24].

Patients’ perception of the need for treatment for anterosuperior diastemas is influenced by the epoch and culture in which they live [29]. There is also a great contribution of the media to the opinion of people. In the 1960s, Gardiner [20] referred to a famous movie actor who presented a medium diastema and suggested, between the lines, that this feature, when smooth, may be well accepted for those individuals with a rather pleasant facial appearance. However, this cannot be considered for the majority of the population. The author’s opinion was retracted through the following statement:

It was demonstrated [30] that when patients self-evaluate, they perceive a greater need for orthodontic treatment, when the problem is located in the anterior region, compromising aesthetics, as is the case of the anterosuperior diastema and anteroinferior crowding.

4.1. Treatment of diastemas in the mixed dentition

In the majority of cases, the central interincisive diastemas in the mixed dentition period are a temporary physiological feature of the “ugly duckling phase” [12, 13], which will be closed gradually with the eruption of the permanent lateral incisors and then with the eruption of the permanent canines. However, orthodontic interception is indicated in exceptional cases, where the diastema is preventing normal eruption of permanent teeth, stimulating the appearance or maintenance of deleterious habits, or compromising the child’s self-esteem [16].

4.2. Treatment of diastemas in permanent dentition

There are several approaches used in the treatment of anterosuperior diastema, which vary according to the present etiologic factor [2, 16, 17]. The success of such treatment will depend on the elimination of these factors [17].

4.2.1. Orthodontic treatment

The orthodontic approach can be performed with the following objective: close the diastema or redistribute the spaces for a posterior reanatomization of the anterior teeth. Orthodontic treatment also has the function of treating, if present, any other associated occlusal problem and helping in the elimination of parafunctional habits. In cases where there is discrepancy of dental size, orthodontic treatment alone is not able to offer the best results. Therefore, these diastemas must be closed by means of composites, facets, or prosthetic crowns. However, for a better esthetic result of these restorations, orthodontic movement is indicated to redistribute the spaces before the cosmetic procedure [31].

Proffit, in the new volume of his book [32], divides the protocol of treatment of anteroposterior diastema according to two basic groups: (1) incisors with diastema and vestibular inclination and (2) diastema in the upper midline. In the first group, the diastema is usually caused by deleterious habit, which must be removed before the space closes, which can only
be performed with a removable device, retracting the incisors. In the second group, the author recommends the closure of the space, followed by frenectomy, in case there is excess tissue pressed in the midline.

In cases where there is a deep overbite, it is interesting that it is corrected previously. This usually increases the horizontal overpass, making it possible to close spaces together with an anterior retraction [24].

4.2.2. Frenectomies

Labial frenulum is considered abnormal when it is enlarged and/or inserted near the gingival margin [15]. Some authors [16, 33] recommend frenectomy to be performed after orthodontic closure of space, since diastema closure and interdental papilla compression may act as a stimulus to promote atrophy of the fibrous tissue interposed between the incisors.

4.2.3. Restorative treatment

Peck and Peck [34] stated that teeth are, by nature, perfect structures. However, a tooth with altered anatomy can often form a malocclusion. For the authors, the orthodontist should increase their understanding of the limitations of orthodontic therapy, and know the value of procedures to change dental forms. Only in this way can treatment success be achieved.

Andrews [35] cited in his book “Straight Wire: Concept and Apparatus” that when there are spaces between teeth due to discrepancy of tooth size, where there are small teeth, orthodontic correction is contraindicated, and recommend the restoration of these teeth with composites or prosthetic crowns.

However, even if there is a discrepancy of dental size, orthodontic treatment may be an auxiliary tool to redistribute the spaces between the teeth before the restorative procedure. This allows the dentition in the anterior teeth to be performed according to the golden ratio, obtaining a better esthetic result [24, 31].

4.2.4. Other types of treatments

Bell [36] argued for the immediate closure of diastema by subapical and interdental osteotomy, justifying that there is a great unpredictability of diastole orthodontic closure stability and that this approach is difficult and takes a long time. In addition, the author believes that the alveolar bone is the major factor responsible for the difficulty in the orthodontic movement of the teeth and for the final stability, as opposed to the majority of authors who consider the labial frenulum and adjacent soft tissues as the main factor for relapse.

5. Stability of diastema treatment

Concern over the stability of results obtained with treatment has existed for more than a century [37].
Riedel [38], in reviewing the problem of containment in the literature, proposed nine theorems that should be considered for greater stability of treatment results. In the first, the author mentioned that orthodontically moved teeth tend to return to their original positions. In Theorem 3, it is reported that the etiological factors of malocclusion should be eliminated for greater stability. Theorem 6 states that the bone and adjacent tissues should have a time for reorganization around the new tooth position, so some type of holding device should be used. And finally, in Theorem 9, it is ensured that the higher the tooth movement, the less recurrence.

The work of Ormiston et al. [39] observed that the greater the severity of malocclusion, the greater the recurrence. This means that the greater the severity of malocclusion, the greater the movement required and the greater the relapse. In this way, they oppose Riedel’s Theorem 9 [38].

There are few scientific studies that evaluate the diastema recurrence after orthodontic closure [15, 28, 40, 41].

Orthodontic corrections in the growth and eruption phases of the teeth are considered more stable by some authors. According to Reitan [37], there will be little or no recurrence after orthodontic movement of an erupting tooth due to the fact that the supporting tissues are in a proliferation stage as a result of the eruption process. New fibers will be formed as the root develops, and these new fibers will help maintain the new tooth position.

Almeida et al. [16] have speculated that the closure of diastema in the mixed denture phase is more stable than if treated in the permanent denture. Therefore, it does not require definitive containment, as long as the habit is removed and, in case of interincisive gingival hypertrophy, surgical correction is performed before removal of the device.

Edwards [15] cited several factors responsible for the reopening of the diastema: incorrect axial inclination of the central incisor roots, tooth size discrepancies, deleterious habits, deleterious occlusal patterns, such as displacement from centric relation to maximal habitual intercuspation or other mandible position, which generate lateral forces on the central incisors, anatomy of the teeth (wider cervical region than the incisal region), and possibly some muscular imbalance in the oral cavity. But in addition to these, the labial frenulum, along with associated tissues, is cited as the most frequent etiological factor in relapsing interincisal diastemas.

The influence of muscular imbalances on the maintenance of bad dental positions varies according to three factors: duration, frequency, and intensity. Studies [10, 22] emphasized that when an etiological factor, such as atypical lingual pressure, cannot be eliminated, it is very difficult to keep the space closed, without the use of permanent retention, especially in adult patients. Attia [10] mentioned that in these patients, the chances of success of improving the lingual posture with only exercises are minimal and indicates the glossectomy.

Shashua and Ärtun [28] evaluated the proportion of diastema recurrence and the possible variables that may have contributed to this recurrence in a sample of 96 patients 4–9 years after the end of treatment. Diastemas varied from 0.5 to 5.6 mm in the pre-treatment stage. The following variables were analyzed: initial diastema size, tooth size discrepancy, overbite, upper incisor inclination, maxillary incisor mobility, labial frenulum, intermaxillary
septal cleft, root parallelism, anterior maxillary spacing, periodontal bone loss and heredi-
ity. Pre- and posttreatment data were obtained from the available documentation (models,
radiographs, photographs, and clinical records). Post-retention data were collected from a
follow-up visit of 37 patients (group A) and a telephone interview of 59 patients (group B).
The incidence was 49% in the total, and 46% in group A, considering the patients with the
diastema observed, or already portrayed by restoration or orthodontically, and still those who
make continuous use of the retention by perceiving a tendency to reopen of space. However,
in group A, the space in patients with recurrence ranged from 0.3 to 0.6 mm, but the mean was
0.1 mm. Logistic regression analysis revealed that the initial width of the diastema and the
family tendency were risk factors for relapse. Although patients with abnormal braking had
earlier initial diameters than those with a normal brake, no association was found between
recurrence and the presence of an abnormal or crevice brake in the intermaxillary bone crest.
The mobility of the upper incisors was the only parameter found in the postretention period
that could be associated with the reopening of the space.

Surbeck et al. [41] evaluated the influence of occlusal changes on the stability in the anterior
region of the maxilla. The sample was selected from study models for the posttreatment stage
and was divided into three groups: (1) with anterosuperior spaces in this phase; (2) with
crowding in the anterosuperior region; and (3) with perfect alignment. In group 1, it was
found that the presence of spaces before treatment, as well as at the end of the treatment,
increases the risk of increased spaces after treatment is finished. The authors suggest that the
contention strategy should be altered according to the presence of spaces before treatment
and their severity. Also, an association was observed between the increase of the spaces in the
post-containment period and the reduction of the intercanine distance during the treatment.
It was also found that reopening of spaces was associated with increased arch length and
intercanine and intermolar distances in the posttreatment period.

5.1. The upper lip frenulum and its relation to the recurrence of diastema

Some authors [15, 21] agree that orthodontic closure of diastemas caused by an abnormal
labial frenulum, without subsequent brake removal surgery, greatly increases the frequency
of relapse in the post-retention period.

Edwards [15] evaluated the relation of the abnormal labial frenulum with both the etiology of
diastema and the stability of its treatment. In this study, the author concluded that frenectomy
had a great contribution to increase the stability of the orthodontic treatment of the median
diastema and commented, citing that the upper labial frenulum is one of the most relevant
factors for the reopening of orthodontically closed diastema.

On the contrary, Shashua and Ärtun [28] did not consider the central labial cleft lip and fissure
in the central interincisive bone crest as risk factors for diastema recurrence, after evaluation
of diastema relapse 4–9 years after the end of treatment. The authors [28] observed that the
presence of the abnormal brake at the beginning of the treatment influenced the initial width
of diastema. However, they emphasized that this type of brake can remodel spontaneously
with the closing of the space.
5.2. Relation between periodontium and recurrence of diastema

It is believed that alveolar bone played the most important role in reopening orthodontically closed spaces [36]. However, several authors [15, 42] consider supra-alveolar gingival tissues as a primary factor in the relapse of dental positions. Transseptal fibers are part of the supra-alveolar fibers group of the periodontal ligament and are composed mainly of collagen fibers. They are firmly inserted into the cementum, close to the cementum-enamel junction, and are responsible for holding together the adjacent teeth. They do not have elastic fibers, but their elastic characteristic is due to their structural form of tiny springs, which form as the fibers mature [25]. It is believed that their role in medial diastema recurrence is due to this elastic property, pressing the incisors mesially and pulling them incisively distally to the initial position [25, 42].

When they examined microscopically the interdental papilla of patients with median diastema, Campbell, Moore, and Matthews [42] observed that the insertion fibers of the brake had continuity with the gingival fibers of that area. The authors also verified an excess of accumulated and compressed gingival tissue in this area after the orthodontic closure of the space, which would act to reopen the spaces. In conclusion, it was suggested that the fibrous network of interincisive supra-alveolar tissues may be one of the main factors responsible for median diastema recurrence.

Similar situation, where a compression of gingival tissues was observed in orthodontically closed dental extraction sites, had already been observed by Edwards [15] and Parker [43]. In the aforementioned study, Edwards [15] noticed that the transseptal fibers, especially those near the alveolar ridge, reorganized and appeared normal after 10–14 days with retention of space closure. The author suggested that this was caused by the heavy forces applied during the final closure of space and by the approximation of the roots (parallelism), causing ischemia at the site and subsequent destruction of the compressed transseptal fibers. These, in turn, would be completely replaced by new fibers. Ten Cate et al. [44] have mentioned that it would be possible to remodel the transseptal fibers by a process called fibroblastic activity, where the fibroblasts are able to synthesize and degrade the collagen simultaneously, controlling its remodeling in the periodontal ligament and in the deeper transseptal areas.

In addition, Parker’s findings [43] demonstrated that transseptal fibers within 60 days did not readapt to the new tooth position and were responsible for the reopening of the space. In this study, the effect of transseptal fibers on the stability of space closure was histologically evaluated, where the first permanent molar was extracted and the second premolar was retracted. In addition, the authors observed that the parallelism of the roots at the end of the closure increased stability and concluded that the fibrotomy of these fibers together with the containment had a positive effect on the stability of the retraction.

Bell [36] believed that the interdental septum was responsible for the instability of diastema closure and advocated subapical osteotomy in the space to be closed to facilitate movement and increase the stability of space closure.
5.3. Mesiodistal angulation of maxillary incisors

It has been mentioned in the literature that there is a greater stability of extraction space closure if the roots end up parallel, because when the roots also follow, the roots are also accompanied, a compression is generated in the place, causing ischemia and consequent destruction of the fibers transseptals. Subsequently, new fibers are formed and configured according to the new tooth position [33, 43].

Mulligan [45] stated that divergent roots are more conducive to keeping the space closed. He believes that when the roots are parallel, the vector of functional forces goes through the long axis of the tooth. However, when the roots are divergent, there is a space between the force vector and the tooth resistance center. This creates a moment that favors the approach of the crowns. The author also added that each patient has a moment of stability. And if within a period of up to 6 weeks without orthodontic treatment, the space remains closed, the ideal moment has been reached. But if there is a recurrence of the diastema, it would be necessary to further diverge the roots.

According to Reitan [37], the stability of the tooth position is greatly influenced by the direction in which the tooth was moved. The vestibular and lingual inclination of teeth has great chances of relapse. However, the mesial and distal movements, with a period of containment, are stable movements, except in cases of extrabuccal force for Class II correction.

Morais et al. reported no association between relapse of interincisor diastema and root parallelism. While midline diastema relapse occurred in 60% of the sample, lateral diastemas closure remained stable after treatment. Only initial diastema width and overjet relapse showed association with relapse of midline diastema [46].

5.4. Retention

In a study by Edwards [15], a recurrence rate of 88% was found in patients with abnormal labial frenulums who used retention for 16–22 months. In another similar group, but with a retention time of 8–10 months, this proportion was similar, 91% of cases with reopening of diastema. This may suggest that, in patients with abnormal labial frenulum, stability is not influenced by retention time, but rather by labial frenulum surgery.

Depending on the type of initial malocclusion, the use of retention throughout life is recommended [38].

Fixed retention is often cited as the only satisfactory method to promote stability at the closure of the previous diastema [32, 47]. However, the commonly available fixed retention present undesirable characteristics for long-term use, since they restrict access to gingival tissues, making their hygiene difficult. Currently, there is a type of fixed retention through small magnets attached to the lingual surface of the incisors, in previously diastematic areas [48].

Removable retention is not considered a good choice in cases of interincisive diastema because, soon after removal, the teeth already begin to move away [32, 45]. In addition, prolonged use of this retainer category would generate back-and-forth movements, which are potentially damaging.
6. Conclusions

The origin of the anterosuperior diastemas is multifactorial. In this sense, the clinician has several approaches for its treatment, which must be selected according to the etiological factor.

The etiology of the diastemas is associated to: abnormal labial frenulum microdontia, agenesis of maxillary incisors, dental discrepancy, shape of the anterior teeth, brachyfacial pattern, a positive tooth-bone discrepancy, supernumerary teeth, cysts, tumors, macroglossia, neuromuscular imbalance of the tongue, and orthodontic treatment.

Orthodontic treatment is a good alternative for diastemas closure, because it can also treat any other associated occlusal problem and help to eliminate parafunctional habits, if any.

When the width of the initial diastemas is very pronounced, the use of retainers can be considered throughout life. Fixed retention is often cited as the only satisfactory method to promote stability in the closure of previous anterosuperior diastemas, while removable retention is not considered a good option.

Acknowledgements

I would like to thank Juliana Fernandes de Morais for her previous contribution to this work.

Conflict of interest

The authors declare to have no conflicts of interest.

Author details

Marcos Jimmy Carruitero Honores
Address all correspondence to: m_carruitero@usp.br; m_carruitero@hotmail.com
Department of Orthodontics, Bauru Dental School, University of São Paulo, Brazil

References

[1] Angle EH. Treatment of Malocclusion of the Teeth. 7th ed. Philadelphia: S.S. White Company; 1907

[2] Bishara SE. Management of diastemas in orthodontics. American Journal of Orthodontics. 1972;61(1):55-63
[3] Andrews LF. The six keys to normal occlusion. American Journal of Orthodontics. 1972; 62(3):296-309

[4] Lavelle CL. Crowding and spacing within the human dental arch of different racial groups. Archives of Oral Biology. 1970; 15(11):1101-1103

[5] McVay TJ, Latta GH. Incidence of the maxillary midline diastema in adults. The Journal of Prosthetic Dentistry. 1984; 52(6):809-811

[6] Richardson ER, Malhotra SK, Henry M, Little RG, Coleman HT. Biracial study of the maxillary midline diastema. The Angle Orthodontist. 1973; 43(4):438-443

[7] Bergström K, Jensen R, Martensson B. The effect of superior labial frenectomy in cases with midline diastema. American Journal of Orthodontics. 1973; 63(6):633-638

[8] Steigman S, Weissberg Y. Spaced dentition. An epidemiologic study. The Angle Orthodontist. 1985; 55(2):167-176

[9] Steigman S, Gershkovitz E, Harari D. Characteristics and stability of spaced dentition. The Angle Orthodontist. 1985; 55(4):321-328

[10] Attia Y. Midline diastemas: Closure and stability. The Angle Orthodontist. 1993; 63(3):209-212

[11] Baume LJ. Physiological tooth migration and its significance for the development of occlusion. I. The biogenetic course of the deciduous dentition. Journal of Dental Research. 1950; 29(2):123-132

[12] Broadbent BH. Ontogenic development of occlusion. The Angle Orthodontist. 1941; 11(4):223-241

[13] Burstone CJ. Distinguish developing malocclusion from normal occlusion. Dental Clinics of North America. 1964; 479-491

[14] Moyers RE. Ortodontia. Rio de Janeiro: Guanabara Koogan; 1991

[15] Edwards JG. The diastema, the frenum, the frenectomy: A clinical study. American Journal of Orthodontics. 1977; 71(5):489-508

[16] Almeida RR, Garib DG, Almeida-Pedrin RR, Almeida MR, Pinzan A, Junqueira MHZ. Diastema interincisivos centrais superiores: Quando e como intervire? Revista Dental Press de Ortodontia e Ortopedia Facial. 2004; 9(3):137-156

[17] Baum AT. The midline diastema. Journal of Oral Medicine. 1966; 21(1):30-39

[18] Gass JR, Valiathan M, Tiwari HK, Hans MG, Elston RC. Familial correlations and heritability of maxillary midline diastema. American Journal of Orthodontics and Dentofacial Orthopedics. 2003; 123(1):35-39

[19] Goodman NR. Treatment of diastema: Not always frenectomy. Dental Survey. 1975; 51(4):28-29

[20] Gardiner JH. Midline spaces. Dental Practitioner. 1967; 17(8):287-298
[21] Graber TM. Ortodoncia: Teoria y Práctica. 3rd ed. México, DC: Interamericana; 1980

[22] Graber TM. The “three M’s”: Muscles, malformation and malocclusion. American Journal of Orthodontics. 1963;49(6):418-450

[23] Bolton WA. The clinical application of tooth-size analysis. American Journal of Orthodontics. 1962;48(7):504-529

[24] Mondelli J, Pereira MA, Mondelli RFL. Etiologia e tratamento dos diastemas dentários. Biodonto. 2003;1(3):11-111

[25] Stublely R. The influence of transseptal fibers on incisor position and diastema formation. American Journal of Orthodontics. 1976;70(6):645-662

[26] Brown EE. Interdental separation in subclinical acromegaly caused by sinusitis. American Journal of Orthodontics. 1953;39(12):932-941

[27] Oesterle LJ, Shellhart WC. Maxillary midline diastemas: A look at the causes. Journal of the American Dental Association. 1999;130(1):85-94

[28] Shashua D, Årtun J. Relapse after orthodontic correction of maxillary median diastema: A follow-up evaluation of consecutive cases. The Angle Orthodontist. 1999;69(3):257-263

[29] Offerman RE. A simple diastema closing device. Oral Health. 1986;76(9):21-22

[30] Peres KG, Traebert ESA, Marces A. Diferenças entre autopercepção e critérios normativos na identificação das oclusopatias. Revista de Saúde Pública. 2002;36(2):230-236

[31] Furuse AY, Franco EJ, Mondelli J. Esthetic and functional restoration for an anterior open occlusal relationship with multiple diastemata: A multidisciplinary approach. The Journal of Prosthetic Dentistry. 2008;99(2):91-94

[32] Proffit WR. Contenção. In: Proffit WR, Fields HW, Sarver DM, editors. Ortodontia Contemporânea. Rio de Janeiro: Elsevier Editora; 2008. pp. 575-588

[33] Edwards JG. The prevention of relapse in extraction cases. American Journal of Orthodontics. 1971;60(2):128-144

[34] Peck S, Peck H. Orthodontic aspects of dental anthropology. The Angle Orthodontist. 1975;45(2):95-102

[35] Andrews LF. Straight Wire. The Concept and Appliance. San Diego: L. A. Wells Co.; 1989

[36] Bell WH. Surgical-orthodontic treatment of interincisal diastemas. American Journal of Orthodontics. 1970;57(2):158-163

[37] Reitan K. Principles of retention and avoidance of posttreatment relapse. American Journal of Orthodontics. 1969;55(6):776-790

[38] Riedel RA. A review of the retention problem. The Angle Orthodontist. 1960;30:179-199

[39] Ormiston JP, Huang GJ, Little RM, Decker JD, Seuk GD. Retrospective analysis of long-term stable and unstable orthodontic treatment outcomes. American Journal of Orthodontics and Dentofacial Orthopedics. 2005;128(5):568-574
[40] Sullivan TC, Turpin DL, Artun J. A postretention study of patients presenting with a maxillary median diastema. The Angle Orthodontist. 1996;66(2):131-138

[41] Surbeck BT, Artun J, Hawkins NR, Leroux B. Associations between initial, posttreatment, and postretention alignment of maxillary anterior teeth. American Journal of Orthodontics and Dentofacial Orthopedics. 1998;113(2):186-195

[42] Campbell PM, Moore JW, Matthews JL. Orthodontically corrected midline diastemas. A histologic study and surgical procedure. American Journal of Orthodontics. 1975;67(2):139-158

[43] Parker GR. Transseptal fibers and relapse following bodily retraction of teeth: A histologic study. American Journal of Orthodontics. 1972;61(4):331-344

[44] Ten Cate AR, Deporter DA, Freeman E. The role of fibroblasts in the remodeling of periodontal ligament during physiologic tooth movement. American Journal of Orthodontics 1976;69(2):155-168

[45] Mulligan TF. Diastema closure and long-term stability. Journal of Clinical Orthodontics. 2003;37(10):560-574

[46] Morais JF, Freitas MR, Freitas KM, Janson G, Castello Branco N. Postretention stability after orthodontic closure of maxillary interincisor diastemas. Journal of Applied Oral Science. 2014;22(5):409-415

[47] Hunt NP. Hypodontia—Problems of permanent space closure. British Journal of Orthodontics. 1985;12(3):149-152

[48] Springate SD, Sandler PJ. Micro-magnetic retainers: An attractive solution to fixed retention. British Journal of Orthodontics. 1991;18(2):139-141
