A postretention study of patients presenting with a maxillary median diastema

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Information on stability following maxillary midline diastema closure is limited[1-7] and focuses on stability following a posttreatment surgical adjunct, such as frenectomy. However, anecdotal reports and at least one study[1] indicate that relapse may be a major problem, especially following closure of diastemas that persist after the eruption of permanent canines. To minimize relapse, permanent retention in the form of bonded wires, restorative options, and even small powerful magnets bonded to the mesiopalatal surfaces of central incisors has been proposed.[8-12]

Varying incidence of maxillary midline diastema in the permanent dentition, from 1.6% to 25.4%, has been reported. This wide variation is probably due to the operational definition of "diastema," inclusion criteria, and the type of population studied.[13-23] However, investigators agree on the potential for closure with eruption of the permanent maxillary canines.[13-17,24] Some have suggested that occlusal factors such as deep overbite, displaced teeth, and maxillary tooth structure deficiencies may be associated with a persistent diastema.[7,20] An association may also exist between generalized spacing and the presence of a median diastema.[23,25] Statements pertaining to "abnormal" frenum and alveolar cleft as an etiologic factor for persistent diastema are mainly speculative in nature, or based on case reports.[4-6,26-29,31] However, Edwards[1] found a strong correlation between an abnormal frenum and a vertical osseous cleft, and the presence of a median diastema. Popovich,[25] on the other

Abstract

The purpose of this study was to assess postretention stability of maxillary midline diastema closure, to search for predictors of relapse, and to test for associations between relapse and other postretention changes. The sample consisted of 35 patients with pretreatment diastemas ranging from 0.9 mm to 3.0 mm (mean 1.4, SD = 0.5) following eruption of the maxillary canines. Data were gathered from treatment charts, study models, periapical radiographs, and cephalograms taken pretreatment, posttreatment, and 1 to 26 years postretention (mean 11.4, SD = 6.4). Measurable diastema relapse was observed in only 12 cases. The majority of the relapse was 0.6 mm or less, and maximum relapse was 3.0 mm. Abnormal frenums and/or intermaxillary osseous clefts did not appear to be risk factors for relapse, and no pretreatment predictors of relapse could be established. The only posttreatment change associated with diastema relapse was proclination of the maxillary incisors ($p<0.01$).

Key Words

Diastema • Stability • Abnormal frenum

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Table 1
Variables measured on study models and radiographs made before treatment (T1), after treatment (T2), and a mean period of 11.4 years postretention (T3), collected from records of 35 patients presenting with a maxillary median diastema

Variable	Time periods measured
* Diastema width (mm)	T1, T2, T3
* Overbite (mm)	T1, T2, T3
* Overjet (mm)	T1, T2, T3
Bolton index	T1, T2
Incisal to gingival width ratio of max. central incisor	T1, T2
* Generalized maxillary anterior spacing (yes/no)	T1, T3
* Maxillary incisor to SN (degrees)	T1, T2, T3
Root parallelism of max. central incisors (parallel, converge, diverge)	T1, T2, T3
* Maxillary labial frenum (normal/abnormal)	T1, T2
* Intermaxillary osseous cleft (present/absent)	T1, T2
Adjunctive surgical procedures (yes/no)	T2
Treatment time (years)	T2
Retention time (years)	T3
Postretention time (years)	T3

* Variables included in regression analysis

hand, found no such relationship. Also, a longitudinal evaluation of a group of 9-year-olds with abnormal frenums revealed no differences in spontaneous diastema closure between subgroups with and without frenectomy.[30]

Few studies have looked at the stability of diastema closure, and information is limited regarding diastema relapse as a truly multifactorial event. The purpose of this study was to assess a sample at least 1 year out of retention in order to evaluate the frequency and amount of relapse, to identify predictors for relapse, and to search for any associations between relapse and postretention changes.

Materials and methods

Sample

Study models, charts, cephalograms, and periapical radiographs made before treatment (T1), after treatment (T2), and a minimum of 1 year postretention (T3) of adolescent patients treated by faculty members and/or graduate students in the orthodontic department at the University of Washington were examined. Sample criteria were limited to patients presenting with a maxillary midline diastema greater than or equal to 0.9 mm after the maxillary permanent canines were at least half erupted. Occlusal status at T3 or cephalometric characteristics were not considered in the sample selection. Patients with prior orthodontic treatment to close the diastema, midline pathology, mesiodens, missing anterior teeth, generalized microdontia, severe periodontal disease, and postorthodontic restoration of maxillary anterior teeth resulting in an increase in mesiodistal width were excluded. A total of 35 patients aged 9.1 to 15.4 years at T1 (mean 12.8 years, SD = 1.4 years), with a diastema of 0.9 to 3.5 mm at T1 (mean 1.4 mm, SD = 0.5 mm), treated for 1.2 to 4.4 years (mean 2.3 years, SD = 0.8 years), and who were 1.0 to 26.0 years out of retention (mean 11.4 years, SD = 6.4 years) were included in the study.

Records were analyzed in a random fashion using numerical codes for identification. All quantitative measurements were done by the principal investigator. Subjective determinations were made by the principal investigator and two University of Washington orthodontic faculty members. All three members of the panel were required to be in agreement on each subjective determination. The variables measured are presented in Table 1.

Examination of study models

Maxillary midline diastemas were measured in the following manner: Impression putty was injected between and around the central incisors. After setting, the impression of the diastema was measured with stainless steel calipers (Iwanson, Sweden) at the narrowest portion, to the nearest 0.1 mm. Overbite was measured as the amount of vertical overlap of the maxillary central incisors over the mandibular central incisors, and overjet as the distance along the occlusal plane from the labial surface of the mandibular central incisors to the labial aspect of the mesial maxillary central incisal edges. The right and left measurements were averaged in situations with discrepancy. The measurements were made with a Union Broach stainless steel endodontic ruler to the nearest 0.5 mm. Tooth width was measured as the distance between the mesial and distal anatomic contact point of maxillary and mandibular incisors and canines, to the nearest 0.5 mm using a stainless steel bow divider. The width of the maxillary central incisors was also measured at the level of the interdental papillae. Generalized anterior spacing was judged subjec-

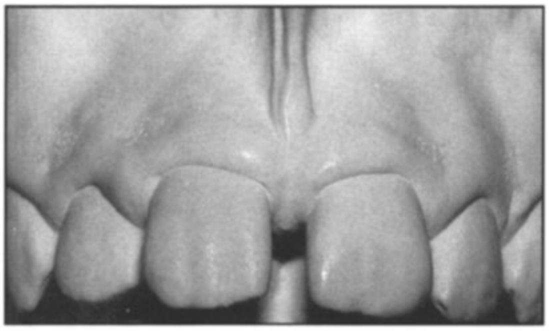

Figure 1A

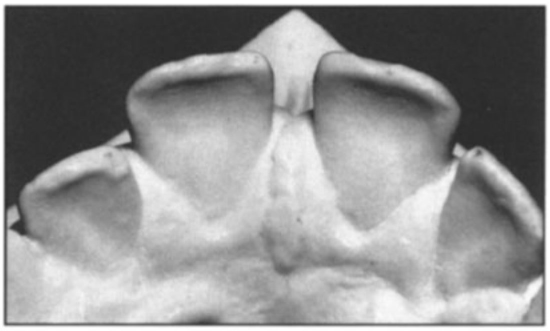

Figure 1B

Figure 1C

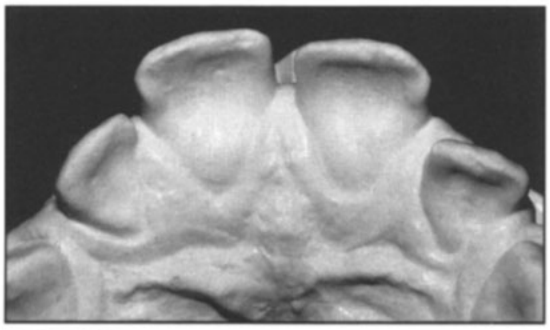

Figure 1D

Figure 1A-D
Study model analysis of normal versus abnormal frenum. Note the presence of a prominent incisive papilla and apparent continuity with the abnormal frenum, as compared with the normal frenum.
A-B: Thick, abnormal frenum inserting low on the alveolus, with prominent incisive papilla.
C-D: Thin, normal frenum inserting well above the alveolar crest, with lack of prominent incisive papilla.

Figure 2A

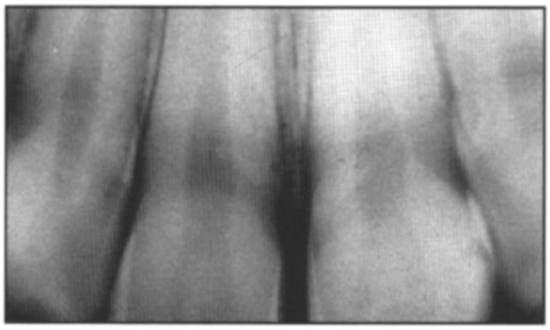

Figure 2B

Figure 2
Radiographic interpretation of presence or absence of osseous cleft between maxillary central incisors.
A: Cleft–definite V-shaped notch in alveolar bone.
B: No cleft–some linear radiolucency but no definite notch.

tively as present or absent, evaluating each proximal contact from the mesial of the maxillary right canine to the mesial of the maxillary left canine. Frenum type was judged subjectively as "normal" or "abnormal" from study models taken at T1 and T2. An "abnormal" score was given to a frenum that exhibited excessive thickness, an alveolar attatchment between the maxillary central incisors, and apparent continuity with a large incisive papilla (Figure 1A-B). The T1 and T2 models were scored at the same time, and if one model showed signs of abrasion or distortion of the frenum, both frenums were scored according to the most accurate model. Borderline cases were scored as "normal." One patient with an "abnormal" frenum at T1 had a frenectomy at T2. For this patient the T1 frenum score was adjusted to "normal" prior to being entered into the regression model.

Radiographic examination

Maxillary incisor inclination was measured in angular relation to sella-nasion line (SN) on cephalograms. An intermaxillary osseous cleft was scored subjectively as present or absent based on periapical radiographs taken at T1 and T2. A cleft was scored if a V-shaped radiolucency was present in the crestal bone between the maxillary central incisors, extending from the intermaxillary suture (Figure 2A). A parallel radiolucency was considered a continuation of the intermaxillary suture and not scored as a cleft (Figure 2B). The T1 and T2 radiographs were scored simultaneously to avoid errors due to differences in radiographic projection. A cleft that was evident at one time period was scored as present at the other time period, even if the radiographic presence of the cleft was questionable at that time period. Borderline cases were scored as "no cleft." Maxillary central incisor root parallelism was judged from periapical or pan-

Figure 3
Examples of cases with postretention re-opening of median diastema.
A. 0.1 mm relapse
B. 0.6 mm relapse
C. 1.3 mm relapse
D. 3.0 mm relapse

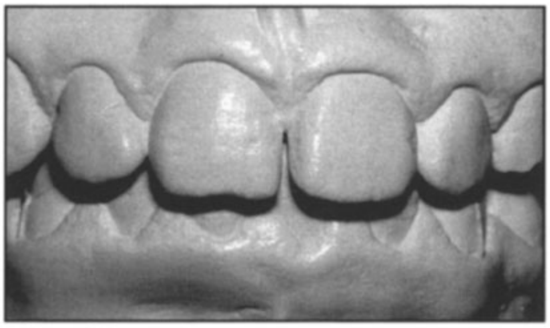

Figure 3A

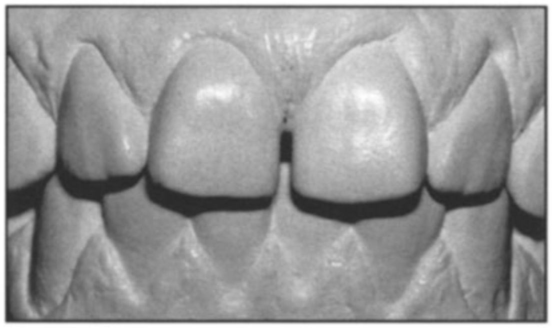

Figure 3B

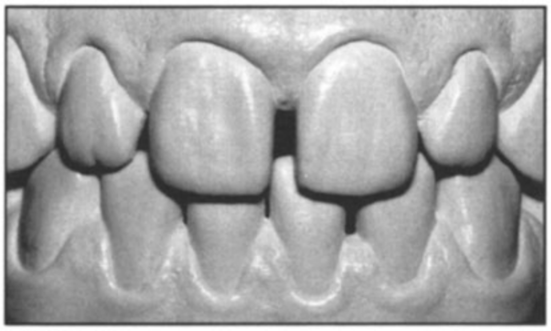

Figure 3C

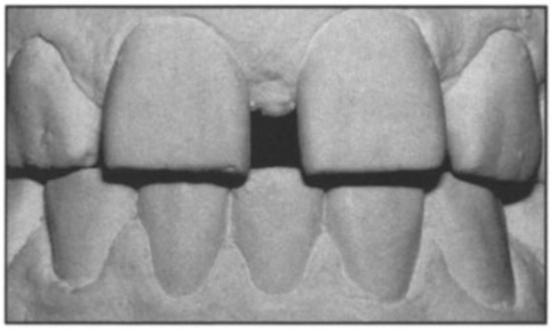

Figure 3D

oramic radiographs taken at T1, T2, and T3. The roots were scored subjectively as parallel, convergent, or divergent at each time period.

Examination of charts

Treatment and retention time, and time out of retention were determined from notes in the charts. Performance of surgical procedures at T2 was based on notes and correspondence in the charts.

Error of the method

The reproducibility of the linear and angular measurements was assessed by statistically analyzing the difference between double measurements taken at least 1 week apart on records of one-third of the sample, selected at random. The measurement error was calculated from the equation:

$$S_x = \text{square root of } \Sigma D^2/2N$$

where D is the difference between duplicated measurements and N is the number of double measurements.[32] The errors for the study model measurements were 0.27 mm for overbite, 0.30 mm for overjet, and 0.13 mm for diastema width. The error for maxillary incisor inclination to SN was 1.62 degrees.

The reproducibility of the subjective scorings was determined by re-evaluating one-third of the sample 1 week after the first scoring. No differences were found between the first and second scoring.

Data analysis

Means and standard deviations were calculated for each of the parameters at the different time intervals. In addition, changes in measurements from T1 to T2, and T2 to T3 were calculated. Bolton index was based on the tooth width measurements and determined according to a revised Bolton analysis chart. A maxillary deficiency index above 1.65 (the standard deviation of the Bolton study[33]) was recorded as "significant." In addition, an index of incisal to gingival width was calculated for the maxillary central incisors. Finally, variables recorded from the charts, study models, and radiographs (Table 1) were entered into a stepwise backward multiple regression model. With this procedure, variables were successively eliminated from the model if their effects were not significant at level 0.15.[34] This approach was used to investigate associations between the independent variables and relapse of median diastema.

Results

Changes from T2 to T3

The amount of postretention relapse of maxillary median diastema was small (Table 2). Twelve patients (34%) had measurable relapse. Relapse of 0.1 to 0.3 mm was seen in 6 subjects (17%, Figure 3A), 0.4 to 0.6 mm in 3 individuals (9%, Figure 3B), 1.0 to 1.6 mm in 2 others (6%, Figure 3C), and 1 patient had relapse of 3 mm (Figure 3D). Mean relapse was found to be 0.3 mm, while mode and median were each 0.0 mm. Of the 13 subjects judged to have an abnormal frenum at T1, 3 had measurable relapse of median diastema. Of the 14 patients with an

Table 2
Mean measurements of study models and cephalograms made before treatment (T1), after treatment (T2), and a mean period of 11.4 years postretention (T3) in 35 patients presenting with maxillary median diastema

	T1		T2		T3	
	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
Diastema width (mm)	1.4	0.5	0.0	0.1	0.3	0.6
Overbite (mm)	3.9	1.2	2.4	0.9	3.3	1.3
Overjet (mm)	8.7	3.1	2.6	0.7	3.0	0.8
Maxillary incisor to SN (degrees)	112.1	8.4	98.2	7.5	99.1	7.5

intermaxillary osseous cleft at T1, 4 had measurable relapse, and of the 7 subjects judged to have a combination of abnormal frenum and osseous cleft, only 1 showed relapse of median diastema.

Changes in median diastema from T2 to T3 versus T1 variables

Bolton index was found to be significant in only 2 subjects, maxillary central incisor roots were judged to be parallel in all but 2 patients, and incisal to gingival crown width was found to be approximately 1:1 in all patients. Accordingly, these variables were eliminated from the regression model. Frenectomy was also excluded from the model because only 5 patients had frenectomy performed posttreatment, 2 of which were combined with circumferential supracrestal fiberotomy of the maxillary incisors. Of these 5 subjects, 4 were judged to have normal frenums at T1. Additionally, 8 cases had missing data in that category. Of the 7 remaining pretreatment variables (Table 1), none were found to be associated with diastema relapse, including initial diastema width.

Change in median diastema from T2 to T3 versus other postretention changes

The dichotomous variables (Table 1) showed no changes from T2 to T3 and were eliminated from the model. Of the remaining variables, only change in maxillary incisor inclination was found to be associated with diastema relapse (regression coefficient 0.04, standardized regression coefficient 0.51, standard error of regression coefficient 0.01, $p<0.01$). As the incisors proclined, the magnitude of diastema relapse increased.

Discussion

The results suggest that approximately two-thirds of the diastema closures are stable postretention, and that relapse of more than 0.6 mm is rare. However, our sample may not be representative of the population presenting with a median diastema and therefore may not be suitable for generalizations. Patients who consider a median diastema unesthetic may seek retreatment if relapse occurs, and comply with permanent retention measures to avoid repeated relapse. Also, patients considered at risk due to repeated space reopening during active treatment, or who show signs of relapse early in retention may be followed more closely and may also be more likely to accept permanent retention measures. Neither category would meet our inclusion criteria. Another possible bias is that patients with a favorable long-term outcome may be more likely to participate in a follow-up examination than patients with severe relapse. Accordingly, our sample may have an overrepresentation of patients who are indifferent to relapse, as well as patients whose relapse was too small to be considered significant. However, regardless of these shortcomings, the results may be interpreted as a contradiction to the general clinical impression that maxillary midline diastemas show a high rate of relapse postretention.

With differing results, Edwards[1] reported relapse of 0.5 mm or more in 84% of a sample of 162 patients during the first 3 months postretention. As many as 33 patients relapsed more than 1.5 mm. However, Edwards' sample differed significantly from ours in severity of pretreatment diastema. He included only subjects with diastemas wider than 2 mm, and the mean of his sample was 3.2 mm. Our inclusion criteria was only 0.9 mm, with a mean of 1.4 mm. Furthermore, Edwards observed relapse immediately postretention, while the follow-up period in our sample was on average 11.4 years postretention. It cannot be ruled out that immediate postretention relapse of median diastema decreases over time in a fashion similar to that documented for extraction sites.[35,36]

The only postretention change that demonstrated an association with diastema relapse in our sample was maxillary incisor inclination, with increased tendency for relapse as the incisors proclined. However, with the exception of one patient, the space opening was limited to the area between the central incisors. In other words, generalized anterior spacing did not tend to recur. Incisor inclination may be associated with relapse of overjet.[37] However, we could not demonstrate a similar association between diastema relapse and change in overjet. Of the 12 cases that relapsed, 5 showed an increase in maxillary incisor proclination. In these patients overjet either decreased or remained unchanged. Possible explanations for this unusual result may be a concommitant increase in mandibular incisor proclination, as may be seen with a forward tongue posture, or a relative increase in mandibular prognathism, neither of which were measured in our sample.

Some investigators have suggested that an increase in overbite may contribute to median diastema.[20,38] We could not confirm that hypothesis. However, our sample may not be considered suitable to test such associations, as the mean postretention change in overbite was only 0.9 mm (Table 2). Among the 12 cases that relapsed, 8 showed either an increase in overbite of only 0.5 mm, no change in overbite, or a decrease in overbite postretention.

No pretreatment variable in our study could be considered a risk factor for diastema relapse. We could not confirm the freqently suggested hypothesis[1,6] of an association between the presence of a maxillary midline alveolar bony cleft or "notch" and diastema relapse. Neither could we confirm Edwards' findings of a high frequency of relapse in cases judged to have abnormal frenums. However, it should be stressed that Edwards evaluated frenum appearance from intraoral photographs, while we used study models.

Frenectomy was confirmed in only 1 of 10 stable cases among the 13 judged to have abnormal frenums in our sample. Only 2 of the 8 cases with missing data on frenectomy belonged to that category of stable cases. Such data suggest limited significance of frenectomy as a measure to enhance postretention stability of cases with relatively small pretreatment distemas. Edwards concluded a dramatic reduction in relapse of median diastema closure following retreatment and frenectomy.[1] However, no long-term examination was performed, and any effect of the retreatment itself on the outcome was not controlled for. Miller's[2] finding of only 3 subjects with postretention relapse following frenectomy and orthodontic closure of median diastema in 27 patients is also difficult to interpret, since no control group was included. His results may simply support our finding that the incidence of relapse is low. In addition, the indications for frenectomy procedures seem unclear. In our sample, 4 of the 5 patients who had the procedure performed were judged to have normal frenums prior to frenectomy.

Our method of assessing frenum anatomy from the study models may be considered questionable. A frenum on a study model may easily be distorted during the fabrication process, and even abraded from the finished model. Accordingly, we may have underscored the presence of abnormal frenum in our sample. However, a thick abnormal frenum is more likely to be accurately reproduced than one that is thin and normal. Previous investigators[1,30] have also pointed out the difficulties associated with an objective evaluation of the frenum, even under good clinical conditions. The validity of our scorings of frenum anatomy as well as appearance of the intermaxillary suture may have improved by evaluating pre- and posttreatment records simultaneously. However, the scoring of borderline structures as "normal" may have tended to underestimate the possible effects of these variables on diastema relapse. Another problem is that the retrospective nature of this study precluded collection of information on occlusal function. Heavy function in the anterior segment and fremitus may cause a diastema to reopen.

Conclusions

The results suggest that the postretention stability of orthodontic closure of a diastema may be better than previously reported. However, our sample may have an overrepresentation of patients who were indifferent to the relapse of median diastema, or patients in whom the relapse was too small to be considered significant. For these reasons our data may not allow generalizations regarding frequency and severity of relapse.

The presence of an abnormal frenum or an intermaxillary osseous cleft appears to be of minor significance for long-term stability in cases with a relatively small diastema pretreatment, and no predictors for relapse could be established. The only posttreatment change associated with diastema relapse was an increase in maxillary incisor proclination.

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References

1. Edwards JG. The diastema, the frenum, the frenectomy: a clinical study. Am J Orthod 1977;71(5):489-508.
2. Miller PD. The frenectomy combined with a laterally positioned pedicle graft. J. Periodontol 1984;56(2):102-106.
3. Thompson HE. Orthodontic relapses analyzed in a study of connective tissue fibers. Am J Orthod 1959;45:93-109.
4. Gillies H. Prosthetic appliances as an aid to surgery in facial restorations. Brit Dent J 1935;59:361.
5. Stubley R. The influence of transseptal fibers on incisor position and diastema formation. Am J Orthod 1976;70(6):645-662.
6. Kraut RA, Payne J. Osteotomy of intermaxillary suture for closure of median diastema. J Am Dent Assoc 1983;107:760-761.
7. Bishara SE. Management of diastemas in orthodontics. Am J Orthod 1972;61(1):55-63.
8. Chan KC, Andreasen GF. Conservative retention for spaced maxillary central incisors. Am J Orthod 1975;67(3):324-329.
9. Zachrisson BU. Clinical experience with direct-bonded othodontic retainers. Am J Orthod 1977;71:440-448.
10. Springate SD, Sandler PJ. Micromagnetic retainers: an attractive solution to fixed retention. Br J Orthod 1991;18(2):139-141.
11. Wright G, Shinn D. Treatment of a median diastema with permanent retention. Dent Practit 1971;22(3):81-84.
12. Reid J, Stirrups DR. A new solution to a difficult problem of orthodontic retention. Br J Orthod 1987;14:281-283.
13. Taylor JE. Clinical observation relating to the normal and abnormal frenum labii superioris. Am J Orthod Oral Surg 1939;25:646.
14. Gardiner JH. Midline spaces. Dent Practit 1967;17(8):287-297.
15. Jakobsson SO. Diastema mediale i overkaken; en frekvensundersokning. Sven Tandlak Tidskr 1962;55:549-562.
16. Backlund E. Diastema mediale superioris, frekvens och etiologi (a longitudinal study of 300 9-12 years old). Svendsk Tadlak 1964;T57:273.
17. Weyman J. The incidence of median diastemata during the eruption of the permanent teeth. Dent Practit 1967;17(8):276-278.
18. Richardson ER, Malhotra SK, Henry M, Little RG, Coleman HT. Biracial study of the maxillary midline diastema. Am J Orthod 1973;43(4):438-443.
19. McVay TJ, Latta GH. Incidence of the maxillary midline diastema in adults. J Prosthet Dent 1984;52(6):809-810.
20. Becker A. The median diastema: a review of its aetiology. Israel J Dent Med 1977;26(1):21-27.
21. Lavelle C. The distribution of diastemas in different human population samples. Scand J Dent Res 1970;78:530-534.
22. Steigman S, Weisberg Y. Spaced dentition, an epidemiologic study. Angle Orthod 1985;55:167-176.
23. Nainar SMH, Gnanasundarum N. Incidence and etiology of midline diastema in a population in South India (Madras). Angle Orthod 1988;59(4):277-282.
24. Broadbent BH. Ontogenic development of occlusion. Angle Orthod 1941;11:223.
25. Popovich F, Thompson GW, Main PA. Persisting maxillary diastema: differential diagnosis and treatment. J Canad Dent Assn 1977;7:330-333.
26. Angle EH. Treatment of malocclusion of the teeth. Ed. 7: Philadelphia, S.S. White Co., 1907.
27. Adams CP. The relation of spacing of the upper central incisors to abnormal fraenum labii and other features of the dentofacial complex. Dent Rec 1954;74:72-86.
28. Dewel BF. The normal and abnormal labial frenum: clinical differentiation. J Am Dent Assoc 1946;33(318).
29. Ferguson MWJ, Colin R. Pathogenesis of abnormal midline spacing of human central incisors. Br Dent J 1983;154:212-218.
30. Bergstrom K, Jensen R, Martensson B. The effect of superior labial frenectomy in cases with midline diastema. Am J Orthod 1973;63(6):633-638.
31. Baum AT. The midline diastema. J Oral Med 1966;21(1):30-39.
32. Dahlberg G. Statistical methods for medical and biological students. London: George Allen and Unwin Ltd. 1940:122-132.
33. Bolton WA. Disharmony in tooth size and its relation to the analysis and treatment of malocclusion. Am J Orthod 1958;28:113-130.
34. Neter J, Wasserman W, Kutner M. Applied linear statistical models. Boston: Richard D. Irwin Inc., 1990.
35. Wisth PJ, Oftedal B. Residual extraction sites after orthodontic treatment. Part II. At the end of retention. Eur J Orthod 1982;4:99-104.
36. Oftedal B, Wisth PJ. Residual extraction sites after orthodontic treatment. Part III. Changes in the postretention period. Eur J Orthod 1982;4:203-210.
37. Fiddler BJ, Årtun J, Joondeph DR, Little RM. Long-term stability of Angle Class II, division 1 malocclusions with successful occlusal results at the end of active treatment. Am J Orthod Dentofac Orthop 1995;107:276-285.
38. Southard KA, Southard TE. Conservative management of anterior spacing and deep bite: a case report. Quint Int 1990;21(10):807-811.