

Different manifestations of Class II Division 2 incisor retroclination and their association with dental anomalies

ABSTRACT

Objective: To investigate whether there is an association between dental development anomalies (DDAs) and the different manifestations of Class II Division 2 (CII/2) malocclusion incisor retroclination. This information may clarify whether the different CII/2 phenotypes, with regard to maxillary incisor retroclination, are a single clinical entity or etiologically different entities.

Design: Retrospective comparative study.

Setting: Private orthodontic practice in the regions of Lisbon and Porto, Portugal

Subjects and Methods: The sample comprised 115 CII/2 malocclusions distributed into 2 groups on the basis of incisor retroclination: Group I composed of 48 CII/2 with retroclination exclusively of both maxillary central incisors; Group II composed of 67 CII/2 with retroclination of all four maxillary incisors. Using the initial orthodontic records, it was determined for each patient the presence of the following DDAs: tooth impaction, tooth agenesis, maxillary lateral incisor microdontia, tooth transpositions and supernumerary teeth.

Results: 55.0% of patients were diagnosed with at least one of the DDAs studied. In the total sample the prevalence rates were determined as follows: 20.0% of palatal maxillary canine impaction, 27.4% of third molar agenesis, and 15.7% of maxillary lateral incisor microdontia. No patient exhibited any transpositions or supernumerary teeth. The distribution of the DDAs studied by groups revealed a strong association of palatal canine impaction, tooth agenesis and maxillary lateral incisor microdontia with Groups II but not with Group I.

Conclusion: The association of DDAs with CII/2 malocclusion is not common to all types of maxillary incisor retroclination, suggesting different etiologic bases among the different manifestations of CII/2 malocclusion incisor retroclination.

INTRODUCTION

Although a number of publications, including twin and triplet studies^{1, 2} and family *pedigrees*³⁻⁶, are indicative of a strong hereditary etiologic component in Class II Division 2 (CII/2) malocclusion, there is little knowledge about what is inherited, and, amongst the various morphological features found in this malocclusion, which ones have been strongly influenced by environment.

As progress is being made on the knowledge of genetic mechanisms controlling dentofacial development, some dental development anomalies (DDAs) have been related with mutations in certain genes that may be causing instability in dental development. The association of DDAs with different malocclusions can be important for a better understanding of the genesis of malocclusion, particularly those in which genetics plays a strong role. Whereas, Basdra et al.⁷ didn't find an association between malocclusions of Class III and Class II Division 1 with DDAs, in another study directed by the same author, a close association of CII/2 malocclusion with agenesis, peg-shaped lateral incisors, canine impactions and transpositions was found⁸.

The CII/2 malocclusion phenotype exhibits a combination of various features among which the retroclination of the maxillary incisors is clearly the most peculiar and the main distinctive sign of this singular malocclusion. This feature, however, doesn't always manifest itself in the same way there existing different forms of maxillary incisor retroclination described in the literature⁹⁻¹¹ (Figure 1 and 2). When considering the diversity of features associated to CII/2, particularly the different manifestations of incisor retroclination, we can speculate whether we are facing different clinical entities, or whether we are before different levels or even different manifestations of the same clinical entity. Assuming, as generally assumed by the scientific community, that the different types of incisor retroclination found in CII/2 malocclusion are the same clinical entity but without any studies having been carried out to confirm the possibility of a different aetiology in the various manifestations of this

malocclusion, it seems necessary to develop further studies that may bring some understanding as to whether the different expressions of CII/2 phenotype can be included in the same clinical entity.

The aim of the present research was to investigate if there is an association between DDAs and the different manifestations of maxillary incisor retroclination of CII/2 malocclusion. This information may clarify whether the different CII/2 phenotypes, with regard to incisor retroclination, are a single clinical entity or whether we are in the presence of etiologically different entities.

SUBJECTS AND METHODS

This retrospective study was approved by the Ethics Commission of the Faculty of Dental Medicine of the University of Porto, Portugal. The sample was collected from the private practice of the first two authors of this paper. From the consecutive analysis of the initial orthodontic records of 4364 patients seeking orthodontic treatment between 2002 and 2010, 215 CII/2 malocclusions were clinically diagnosed. These 215 patients, all non-syndromic Caucasians, were distributed into 2 groups on the basis of the type of the maxillary incisor retroclination, after having been applied the following inclusion criteria: (1) Class II molar relationship (distocclusion) on at least one side in centric occlusion; (2) no history of orthodontic treatment, maxillofacial or plastic surgery and trauma in the maxillary anterior teeth; (3) absence of prosthetic crowns or extensive restorations in the six maxillary anterior teeth; (4) the angle between the maxillary incisor long axis and the palatal plane less than or equal to 100°; (5) overbite equal to or greater than 50% and (6) previous eruption of the maxillary and mandibular second permanent molars. Subjects with agenesis of maxillary lateral incisors were excluded as they could not be included in the two groups to be studied. The total sample was thus made up of 115 subjects (69 females, 46 males) with a mean age of 22.5 years (SD, 9.0; range, 12-50 years) distributed into the two groups as follows:

Group I composed of 48 CII/2 malocclusions (29 females, 19 males) with retroclination exclusively of both maxillary central incisors, with a mean age of 22.1 years (SD, 9.0; range, 12-50 years);

Group II composed of 67 CII/2 (40 females, 27 males) with retroclination of all four maxillary incisors, with a mean age of 22.7 years (SD, 9.0; range, 12-43 years).

The initial orthodontic records (dental history, panoramic radiograph, cephalogram, study models, intraoral photographs and periapical radiographs when available) were used to evaluate the following DDAs:

Palatal maxillary canine impaction – As a consequence of the inclusion criteria adopted, most patients in this study were already past the eruption age of the maxillary canine. For the early diagnosis of impacted canines in subjects at the age of maxillary canine eruption we used the radiographic parameters proposed by Lindauer et al.¹². Thus, we considered an impacted canine when the cusp tip was located in sectors II, III or IV (Figure 3). In order to determine the bucco-palatal position of the impacted canine, we used periapical radiographs taken in two different incidence angles.

Impactions of other teeth – Using the initial radiographic data, besides palatal maxillary canine impaction all tooth impactions were recorded, except the impaction of third molars;

Third molar agenesis – To achieve a proper diagnosis, two critical questions were raised. First, it was necessary to determine the earliest age at which agenesis of third molars can be radiographically diagnosed. Although Garn and Lewis¹³ suggest that it is not possible to confirm third molar agenesis before the age of 14, in our study the chronological age was not used as an inclusion criterion, the option being to include subjects whose second molars were already erupted by the date of the first records. For all subjects under 14 included in the sample it was possible to confirm, by means of archived panoramic radiographs taken after the age of 14, that the diagnosis obtained from the initial radiographs was correct. Another critical

aspect that had to be taken into account was the risk of false diagnoses due to omissions or errors in the dental history of patients. Whenever the diagnostic procedures available were insufficient to remove any uncertainty about the cause of third molar absence, patients were contacted so that a rigorous questionnaire might be made for clarification;

Agenesis of other teeth – Using the dental history and the panoramic radiograph included in the initial orthodontic records, all dental agenesis were identified excluding third molar;

Maxillary lateral incisor microdontia– Alterations in the shape and size of maxillary lateral incisors were identified from study models. This category includes peg-shaped laterals that exhibit a conical shape with mesiodistal width greatest at the cervical margin^{14, 15} and small incisors when the maximum mesiodistal width was equal to or smaller than that of its mandibular counterpart^{14, 16, 17}.

Tooth transpositions – Despite the varied definitions found in the literature, in this study a tooth transposition was considered the positional interchange of two adjacent teeth, particularly of their roots, or the development or eruption of a tooth in the place normally occupied by a non-adjacent tooth¹⁸.

Supernumerary teeth – Supernumerary teeth were identified and registered from the plaster models and the initial panoramic radiograph of the subjects studied.

Statistical analysis

The statistical analysis was performed using IBM® SPSS version 20.0 (Statistical Package for Social Sciences) and R version 2.15.1. Given the nature of the variables involved, it was decided to use statistical tools based on the analysis most appropriate to the scales of measurement used. Thus, the analysis consisted of the prevalence study in which estimates were determined for all parameters evaluated, as well as interval estimates with 95% confidence, and analytical study of the data for qualitative variables, where the association between two variables was established using the chi-square test of independence (for 2x2

tables was used the exact test of Fisher). The decision rule consists of detecting statistically significant evidence for probability values (value of the proof test) less than 0.05.

RESULTS

Among the 115 patients participating in this research, it was not possible to confirm in six of them the existence of at least one of the DDAs studied, due to uncertainty regarding the cause of third molar absence. Of the remaining 109 patients, 60 (55.0%) were diagnosed with at least one dental development anomaly. A total of 28 impacted maxillary canines occurred in 23 (20.0%) out of the 115 subjects examined. Besides palatal maxillary canine impaction and third molar impaction, impaction of other four teeth was detected in three subjects (2.6%). It was not possible to confirm the cause for the absence of third molars in nine patients. Thus, 29 (27.4%) out of the 106 subjects whose records provided conclusive evidence displayed agenesis of at least one third molar, amounting to 69 teeth. Besides the third molar, the agenesis of other dental pieces occurred in 10 subjects, which corresponds to 8.7% of the CII/2 sample studied. The existence of at least one microdontic maxillary lateral incisor was recorded in 18 (15.7%) of the subjects studied making up a total of 28 maxillary lateral incisors with morphologic alterations. Out of the 115 subjects studied no one exhibited tooth transpositions or supernumerary teeth. A synthesis of the findings is provided in Table 1.

Table 2 provides the prevalence of the DDAs studied per group. From the distribution of the assessed DDAs into groups, it is worth mentioning the absence of maxillary canine impaction in Group I whilst a prevalence of 34.3% of this tooth anomaly was determined in Group II. High prevalence rate of third molar agenesis of 35.9% was found in Groups II, contrasting with 14.3% found in Group I. Likewise, the prevalence of lateral microdontia in Group I was only 4.2% whilst a prevalence rate of 23.9% was observed in Group II.

Chi-square tests were performed in order to evaluate whether there is an association of DDAs with the groups studied. Results confirmed a statistically significant association

between all variables studied and the group, except for the impaction of other teeth, and for the agenesis of other teeth. The statistically significant association implies that there is a strong association of canine impaction, total impaction, third molar agenesis, total agenesis and maxillary lateral incisor microdontia with Group II, and a poor association with Group I (Table 2).

The prevalence rates of at least one tooth anomaly studied were 23.3% and 75.8% for Groups I, and II respectively. In this case, there was also a statistically significant association between the presence of DDAs and the group, in the sense that there is a strong association between them and Group II (Table 2).

DISCUSSION

One hundred and fifteen patients with CII/2 malocclusion were examined with the purpose of determining the co-existence of DDAs, being the association with DDAs determined for the two groups based on the type of incisor retroclination in CII/2.

In the overall sample studied, 60 subjects (55.0%) exhibited at least one of the DDAs under research. These findings are practically identical to those found by Basdra et al.⁸, who, in a population of 267 CII/2, also diagnosed at least one dental anomaly in 56.6% of the subjects. The DDAs evaluated by these authors are similar to those of our study, except that they evaluate the maxillary palatal and buccal canine impaction and the maxillary lateral incisors agenesis, and do not evaluate either the presence of other tooth impactions besides the canine, or the agenesis of other teeth besides the maxillary lateral incisor and the third molar. However, Basdra et al.⁷, when studying the same DDAs, determined a prevalence of 25% and 15.4% of at least one dental anomaly for Class III and for Class II Division 1 malocclusion respectively. Furthermore, these authors found no statistically significant differences between Class III and Class II Division 1 samples regarding the presence of DDAs,

but there were statistically significant differences in comparison with the findings obtained in the CII/2 malocclusion study.

The prevalence per group of at least one of the DDAs assessed denotes particularly interesting findings. While for Group I the prevalence was 23.3%, for Group II a significantly higher prevalence of 75.8% was determined. The findings reveal that individuals carrying CII/2 malocclusion with retroclination of both maxillary central incisors present prevalence rates of at least one dental anomaly, very similar to those found by Basdra et al.⁷ for Class III and for Class II Division 1 malocclusion. Statistically significant differences regarding the presence of DDAs determined between Groups I and II show that the strong association of DDAs with CII/2 malocclusion can only be attributed to the CII/2 type with retroinclination of four or more maxillary anterior teeth and not to the type with retroclination of both central incisors only.

The literature data reveal a prevalence of canine impaction in the general population ranging between 0.8% and 2.8%¹⁹⁻²⁴. Much of canine impaction is palatal accounting for about 85 % of the overall canine impaction²⁵⁻²⁷. In the CII/2 sample, Basdra et al.⁸ determined a prevalence of buccal and palatal canine impaction of 33.5%, which proved to be significantly higher when compared either with study samples taken from the general population or with the research developed by the same author from Class III and Class II Division 1 malocclusion samples, where canine impaction prevalence rates of 9% and 3.3% respectively were determined⁷.

Distribution of impaction by groups revealed some interesting findings. The absence of maxillary palatal canine impaction found in Group I contrasts with the prevalence of 34.3% recorded in the group in which the four maxillary incisors appeared retroclined.

Were it not for the lack of consensus in this area, the canine impaction aetiology could probably explain these findings. There is sustainable evidence that the buccal and palatal eruption deviations of the maxillary canine have a different origin. Whilst buccal impaction has been attributed to a lack of space of the maxillary arch^{28, 29}, Jacoby²⁸ found that in 85% of the

palatally impacted canines the arch space was adequate. The debate over the origin of palatal canine impaction has focused on two theories. Given the high incidence of palatal canine impaction in subjects exhibiting maxillary lateral incisors agenesis and microdontia observed by Becker et al.^{14, 30}, they suggested that local factors like these may be the main etiologic agents responsible for the eruption deviation of that tooth. According to the so-called Guidance Theory, the maxillary lateral incisor root acts as a guide in the eruption of maxillary canines. Therefore, if the lateral incisor is missing or displays altered root morphology, the canine can develop an ectopic eruptive path³¹. The Genetic Theory postulated by Peck *et al.*³² considers genetic factors to be the primary etiologic agents of palatal deviation and consequently of canine impaction. According to this theory, the maxillary lateral incisor agenesis and palatal canine impaction share the same etiologic basis, whereas the defenders of the *Guidance Theory* consider DDAs of genetic origin like the maxillary lateral incisor agenesis or microdontia to create environmental conditions that favor eventual maxillary canine impaction. A first interpretation of the high prevalence of canine impaction in the CII/2 malocclusion group, with both maxillary lateral incisors retroclined, might provide a major support for the Guidance Theory. Retroclined lateral incisor crowns imply proclined roots and more mesially positioned apices, which means that the guidance effect, considered important by the defenders of this theory for the correct maxillary canine eruption, is lost. On the other hand, and supporting the Genetic Theory, the retroclination of the four maxillary incisors found in this type of CII/2 malocclusion might be understood as having the same genetic basis as palatal canine impaction. The high prevalence of DDAs in this type of CII/2, in contrast with the group in which incisor retroclination was limited to the centrals, supports this etiologic view. The high prevalence rate of DDAs in Group II might be interpreted as resulting from the high prevalence of canine impaction found in this group. However, even if palatal canine impaction is not considered, more than half of the subjects in this group (58.5%) exhibited at least one of the DDAs examined.

Apart from impaction of maxillary palatal canine and third molars, other four impacted teeth were found in three subjects, one patient in Group I and two patients in Group II. The reduced number of cases found does not allow conclusive findings regarding the association of impaction of other teeth and the two CII/2 groups analyzed.

Numerous prevalence studies about tooth agenesis can be found in the literature. Prevalence in the general population of at least one permanent tooth in Caucasian subjects, excluding third molars, ranges between 3.5% and 10.1%^{23, 33-41}. A prevalence of 8.7% of at least one permanent tooth agenesis excluding third molars was found in our CII/2 population. When third molars are considered, a 32.1% hypodontia prevalence rate was determined. Prevalence study estimates for agenesis of at least one third molar vary between 7% and 26%⁴²⁻⁴⁷. In our sample, a slightly higher prevalence was determined, more specifically 27.4%.

The prevalence rate of 35.9% for third molar agenesis and 40.6% for total agenesis determined in Group II contrast with the third molar agenesis rate of 14.3% and with the total agenesis rate of 19% found in Group I. The findings reveal a clear statistically significant association between tooth agenesis and the CII/2 malocclusion group with retroclination of all the maxillary incisors. This association does not occur with CII/2 with retroclination exclusively of the maxillary central incisors, whose values are within the average prevalence rates for tooth agenesis found in the general population.

Maxillary lateral incisor microdontia prevalence of 4.2% in Group I and 23.9% in Group II also shows poor association of this congenital anomaly with the group of CII/2 with retroclination exclusively of the maxillary central incisors in contrast with the strong association found in the group with retroclination of all the maxillary incisors. Reference values recorded in the literature regarding lateral incisor microdontia prevalence, in studies on the general population and even on orthodontic populations, vary between less than 1% and 5.8%^{15, 20, 48-50}. Comparable values were found for Class III and Class II Division 1 malocclusion, 3%

and 0.9% respectively⁷. These values coincide with the ones found in Group I but are significantly lower than those determined for Group II.

The association of tooth transpositions with CII/2 malocclusion reported by Basdra *et al.*⁸ could not be confirmed in this study. Nevertheless, the rarity of this anomaly and the size of the sample examined require a sensible interpretation of the results.

In line with Basdra *et al.*⁸ observations, no supernumerary tooth was diagnosed in our CII/2 sample. The findings are in accordance with other studies suggesting that supernumerary teeth are a hyperplastic anomaly apparently resulting from etiologic mechanisms different from the other dental anomalies examined and also from CII/2 malocclusion itself^{17, 50, 51}.

The global epidemiologic results obtained from this study, despite not being able to give an explanation for the various types of incisor retroclination, suggest that a better understanding is needed about the etiologic basis behind phenotypes that have been considered to be clinical entities with a common origin. The reciprocal association found among the various DDAs, as in the case of tooth agenesis and microdontia, with strong evidence of having a genetically based etiology, and their close association with CII/2 malocclusion with retroclination of the four maxillary incisors, suggests that the same genetic anomaly would likely be in the genesis of some DDAs as well as of the retroclination of all maxillary incisors. Therefore, a divergent genetic basis could account for the occlusal model characterized by retroclination exclusively of both maxillary centrals.

The clinical implications of these findings are unequivocally relevant. The close association of DDAs like tooth agenesis, palatal canine impaction and lateral incisor microdontia with CII/2 malocclusion phenotypes in which incisor retroclination involves all maxillary incisors, alerts clinicians to the need in these patients for an early diagnosis of these DDAs during the initial phases of mixed dentition. In the particular case of palatal canine impaction, early diagnosis is of paramount clinical importance, since the perception of the maxillary canine eruption deviation requires preventive therapeutic measures to be adopted

such as early extraction of deciduous canines⁵² and others, which may allow to recover the normal eruptive path of the permanent canine.

CONCLUSIONS

From the overall results obtained in this epidemiologic study it is possible to withdraw the following conclusions:

- The association of DDAs with CII/2 malocclusion is not common to all types of maxillary incisor retroclination.
- Maxillary palatal canine impaction, tooth agenesis and maxillary lateral incisor microdontia are DDAs closely associated with CII/2 phenotypes with retroclination of four maxillary incisors, but not with the manifestation with retroclination exclusively of the maxillary central incisors.
- CII/2 malocclusion phenotypes with retroclination exclusively of the maxillary central incisors seem to be an etiologically different entity from those in which retroclination involves all maxillary incisors.

REFERENCES

1. Markovic MD. At the crossroads of oral facial genetics. *Eur J Orthod* 1992; 14: 469-481.
2. Kloeppel W. Deckbiss bei Zwillingen. *Fortschr Kieferorthop* 1953; 14: 130-135.
3. Trauner R. Leitfaden der praktischen Kieferorthopädie. Berlin: Verlag Die Quintessenz, 1968. p.20-21.
4. Korkhaus G. Aetiologie der Zahnstellungs - und Kieferanomalien. *Fortschr Kieferorthop* 1931; 1: 136-154.
5. Rubbrecht O. Les variations maxilla-faciale sagittales et l'hérédité mendélienne. *Revue Belge Stomatol* 1930; 27: 1-24,61-91,119-153.
6. Korkhaus G. Investigations in to the inheritance of orthodontic malformations. *Dent Record* 1930; 50: 271-280.
7. Basdra EK, Kiokpasoglou MN, Komposch G. Congenital tooth anomalies and malocclusions: a genetic link? *Eur J Orthod* 2001; 23: 145-151.
8. Basdra EK, Kiokpasoglou M, Stellzig A. The Class II Division 2 craniofacial type is associated with numerous congenital tooth anomalies. *Eur J Orthod* 2000; 22: 529-535.
9. Canut JAB. *Ortodoncia Clinica*. Barcelona: Ediciones Científicas y Técnicas, S.A., 1992. p.427-441.
10. van der Linden FPGM. *Development of Dentition*. Chicago: Quintessence Publishing Co., 1983. p.93-103.

11. Korkhaus G, Bruhn C, Hofrath H. La Escuela Odontologica Alemana. Rio de Janeiro: Editorial Labor, 1939. p.557-592.
12. Lindauer SJ, Rubenstein LK, Hang WM, Andersen WC, Isaacson RJ. Canine impaction identified early with panoramic radiographs. *J Am Dent Assoc* 1992; 123: 91-92, 95-97.
13. Garn SM, Lewis AB. The relationship between third molar agenesis and reduction in tooth number. *Angle Orthod* 1962; 32: 14-18.
14. Becker A, Smith P, Behar R. The incidence of anomalous maxillary lateral incisors in relation to palatally-displaced cuspids. *Angle Orthod* 1981; 51: 24-29.
15. Bot PL, Salmon D. Congenital defects of the upper lateral incisors (ULI): condition and measurements of the other teeth, measurements of the superior arch, head and face. *Am J Phys Anthropol* 1977; 46: 231-243.
16. Shalish M, Peck S, Wasserstein A, Peck L. Increased occurrence of dental anomalies associated with infraocclusion of deciduous molars. *Angle Orthod* 2010; 80: 440-445.
17. Garib DG, Peck S, Gomes SC. Increased occurrence of dental anomalies associated with second-premolar agenesis. *Angle Orthod* 2009; 79: 436-441.
18. Peck L, Peck S, Attia Y. Maxillary canine-first premolar transposition, associated dental anomalies and genetic basis. *Angle Orthod* 1993; 63: 99-109; discussion 110.
19. Ericson S, Kurol J. Radiographic assessment of maxillary canine eruption in children with clinical signs of eruption disturbance. *Eur J Orthod* 1986; 8: 133-140.
20. Brin I, Becker A, Shalhav M. Position of the maxillary permanent canine in relation to anomalous or missing lateral incisors: a population study. *Eur J Orthod* 1986; 8: 12-16.
21. Grover PS, Lorton L. The incidence of unerupted permanent teeth and related clinical cases. *Oral Surg Oral Med Oral Pathol* 1985; 59: 420-425.
22. Shah RM, Boyd MA, Vakil TF. Studies of permanent tooth anomalies in 7,886 Canadian individuals. I: impacted teeth. *J Can Dent Assoc* 1978; 44: 262-264.
23. Thilander B, Myrberg N. The prevalence of malocclusion in Swedish schoolchildren. *Scand J Dent Res* 1973; 81: 12-21.
24. Dachi SF, Howell FV. A survey of 3, 874 routine full-month radiographs. II. A study of impacted teeth. *Oral Surg Oral Med Oral Pathol* 1961; 14: 1165-1169.
25. Stellzig A, Basdra EK, Komposch G. Zur Atiologie der Eckzahnverlagerung--eine Platzanalyse. *Fortschr Kieferorthop* 1994; 55: 97-103.
26. Ericson S, Kurol J. Radiographic examination of ectopically erupting maxillary canines. *Am J Orthod Dentofacial Orthop* 1987; 91: 483-492.
27. Rayne J. The unerupted maxillary canine. *Dent Pract Dent Rec* 1969; 19: 194-204.
28. Jacoby H. The etiology of maxillary canine impactions. *Am J Orthod* 1983; 84: 125-132.
29. Thilander B, Jakobsson SO. Local factors in impaction of maxillary canines. *Acta Odontol Scand* 1968; 26: 145-168.
30. Becker A, Zilberman Y, Tsur B. Root length of lateral incisors adjacent to palatally-displaced maxillary cuspids. *Angle Orthod* 1984; 54: 218-225.
31. Becker A. In defense of the guidance theory of palatal canine displacement. *Angle Orthod* 1995; 65: 95-98.
32. Peck S, Peck L, Kataja M. The palatally displaced canine as a dental anomaly of genetic origin. *Angle Orthod* 1994; 64: 249-256.
33. Aasheim B, Ogaard B. Hypodontia in 9-year-old Norwegians related to need of orthodontic treatment. *Scand J Dent Res* 1993; 101: 257-260.
34. Rolling S. Hypodontia of permanent teeth in Danish schoolchildren. *Scand J Dent Res* 1980; 88: 365-369.
35. Magnusson TE. Prevalence of hypodontia and malformations of permanent teeth in Iceland. *Community Dent Oral Epidemiol* 1977; 5: 173-178.
36. Bergstrom K. An orthopantomographic study of hypodontia, supernumeraries and other anomalies in school children between the ages of 8-9 years. An epidemiological study. *Swed Dent J* 1977; 1: 145-157.

37. Thompson GW, Popovich F. Probability of congenitally missing teeth: results in 1,191 children in the Burlington Growth centre in Toronto. *Community Dent Oral Epidemiol* 1974; 2: 26-32.
38. Brook AH. Dental anomalies of number, form and size: their prevalence in British schoolchildren. *J Int Assoc Dent Child* 1974; 5: 37-53.
39. Haavikko K. Hypodontia of permanent teeth. An orthopantomographic study. *Suom Hammaslaak Toim* 1971; 67: 219-225.
40. Egermark-Eriksson I, Lind V. Congenital numerical variation in the permanent dentition. D. Sex distribution of hypodontia and hyperodontia. *Odontol Revy* 1971; 22: 309-315.
41. Muller TP, Hill IN, Peterson AC, Blayney JR. A survey of congenitally missing permanent teeth. *J Am Dent Assoc* 1970; 81: 101-107.
42. Cardona L. Prevalencia del la agenesia del tercer molar. *Rev Esp Ortod* 1984; 14: 53-57.
43. Godt H, Greve R. Beitrag zur Unterzahl der Zahne einschliesslich der dritten Molaren. *Zahnarztl Prax* 1980; 31: 265-269.
44. Nanda RS. Agenesis of the third molar in man. *Am J Orthod* 1954; 40: 698-706.
45. Hellman M. Our third molar teeth; their eruption, presence and absence. *Dent Cosmos* 1936; 78: 750-762.
46. Banks HV. Incidence of Third Molar Development. *Angle Orthod* 1934; 4: 223-233.
47. Gorblirsch AW. A study of third molar. *J Am Dent Assoc* 1930; 17: 1848-1854.
48. Alvesalo L, Portin P. The inheritance pattern of missing, peg-shaped, and strongly mesio-distally reduced upper lateral incisors. *Acta Odontol Scand* 1969; 27: 563-575.
49. Meskin LH, Gorlin RJ. Agenesis and Peg-Shaped Permanent Maxillary Lateral Incisors. *J Dent Res* 1963; 42: 1476-1479.
50. Baccetti T. A controlled study of associated dental anomalies. *Angle Orthod* 1998; 68: 267-274.
51. Garib DG, Alencar BM, Lauris JR, Baccetti T. Agenesis of maxillary lateral incisors and associated dental anomalies. *Am J Orthod Dentofacial Orthop* 2010; 137: 732 e731-736; discussion 732-733.
52. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. *Eur J Orthod* 1988; 10: 283-295.

FIGURE LEGENDS

Figure 1. Class II Division 2 malocclusion with retroclination exclusively of the maxillary central incisors.



Figure 2. Class II Division 2 malocclusion with retroclination of all four maxillary incisors.



Figure 3. Sectors used in the Lindauer method.

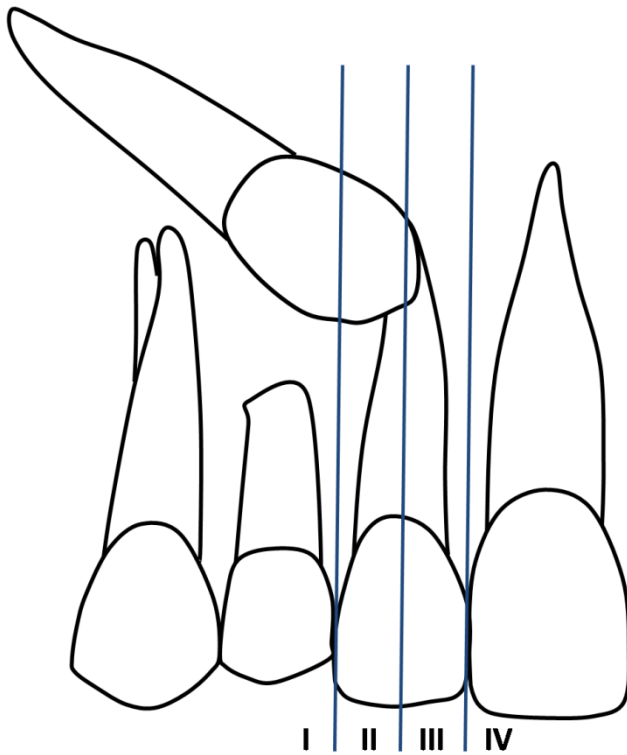


Table 1. Prevalence of dental anomalies in the CII/2 total sample with respective confidence intervals of 95%.

		N	Prevalence %	95% Confidence Interval
Palatal maxillary canine impaction	Absence	92	80.0%	[50.4%; 68.9%]
	13	8	7.0%	[3.3%; 13.7%]
	23	10	8.7%	[4.5%; 15.8%]
	Bilateral	5	4.3%	[1.6%; 10.3%]
	Total	115	100.0%	
Impaction of other teeth (Excl. 3rd molars)	Absence	112	97.4%	[92.3%; 99.3%]
	Presence	3	2.6%	[0.5%; 7.4%]
	Total	115	100.0%	
Third molar agenesis	Absence	77	72.6%	[63.0%; 80.6%]
	Presence	29	27.4%	[19.4%; 37.0%]
	Total	106	100.0%	
Agenesis of other teeth (Excl. 3rd molars)	Absence	105	91.3%	[84.2%; 95.5%]
	Presence	10	8.7%	[4.5%; 15.8%]
	Total	115	100.0%	
Total of agenesis	Absence	72	67.9%	[58.1%; 76.5%]
	Presence	34	32.1%	[23.5%; 41.9%]
	Total	106	100.0%	
Maxillary lateral incisor microdontia	Absence	97	84.3%	[76.1%; 90.2%]
	12	3	2.6%	[0.5%; 7.4%]
	22	5	4.3%	[1.4%; 9.9%]
	Bilateral	10	8.7%	[4.2%; 15.4%]
	Total	115	100.0%	
Presence of dental development anomalies	Absence	49	45.0%	[35.5%; 54.8%]
	Presence	60	55.0%	[45.2%; 64.5%]
	Total	109	100.0%	

Table 2. Prevalence of dental anomalies per group.

	Group I		Group II		P value*
	N (48)	%	N (67)	%	
Palatal maxillary canine impaction	0	0%	23	34.3%	<0,001
Impaction of other teeth (Excl. 3rd molars)	1	2.1%	2	3.0%	0.624
Total of impaction	1	2.1%	23	35.4%	<0.001
Third molar agenesis	6 (42)	14.3%	23 (64)	35.9%	0.012
Agenesis of other teeth (Excl.3rd molars)	2	4.2%	8	11.9%	0.130
Total of agenesis	8 (42)	19.0%	26 (64)	40.6%	0.016
Upper lateral incisor microdontia	2	4.2%	16	23.9%	0.003
Presence of dental development anomalies	10 (43)	23.3%	50 (66)	75.8%	<0.001

* Results from independence (Fisher exact test). Test the null hypothesis: There are no associations between the variables considered. Significant association is detected if p value is less than 0.05.

Values in brackets correspond to valid cases.