A sella turcica bridge in subjects with dental anomalies

Rosalia Leonardi*, Ersilia Barbato**, Maurizio Vichi*** and Mario Caltabiano*
*Department of Orthodontics, Dental College, University of Catania and Departments of **Orthodontics and ***Statistics, Probability, and Applied Statistics, University La Sapienza, Rome, Italy

SUMMARY Calcification of the interclinoid ligament (ICL) of the sella turcica, or sella turcica bridging, has been associated with severe craniofacial deviations. Despite no comprehensive study on the sella turcica bridge, a relationship with tooth and eruption disturbances has been reported. In order to investigate whether congenital absence of the second mandibular premolar, or the presence of a palatally displaced canine (PDC), is associated with sella bridging, a retrospective study was performed.

Lateral cephalometric radiographs from 20 males and 14 females, aged between 8 and 16 years, with a PDC and second mandibular premolar aplasia were reviewed and compared with a control group. A standardized scoring scale was established to quantify the extent of a sella turcica bridge from each radiograph (no calcification, partially calcified, and completely calcified).

The prevalence of complete calcification of the ICL in adolescents with dental anomalies was equal to 17.6 per cent, while an incidence 9.9 per cent was found in the control group. A partially calcified sella turcica was observed in 58.8 per cent of adolescents with dental anomalies compared with 33.7 per cent in the control group. The association between the degree of calcification of the ICL and the presence of dental anomalies in the studied adolescents was statistically significant according to chi-square statistics ($P = 0.004$). This was confirmed by Fisher's exact test ($P = 0.003$).

According to these findings, the prevalence of a sella turcica bridge in adolescents with dental anomalies is increased, while age and gender do not greatly influence ossification of the ICL. The very early appearance during development of a sella turcica bridge should alert clinicians to possible tooth anomalies in life later.

Introduction

Cephalometric films and tracings are mainly used for the evaluation of skeletal and dental patterns as a basis for predicting facial growth. However, in addition, they contain other diagnostic information concerning the skull, face, and upper cervical spine. A number of studies have described skeletal abnormalities and normal variants on cephalometric radiographs and some of these have dealt with the calcification of the interclinoid ligament (ICL) of the sella turcica (Bisk and Lee, 1976; Kantor and Norton, 1987; Tetradis and Kantor, 1999).

In healthy subjects, the occurrence of sella bridging ranges from 3.8 to 13 per cent (Cederberg *et al.*, 2003; Axelsson *et al.*, 2004a,b; Jones *et al.*, 2005).

A clear tendency towards a greater frequency of sella turcica bridge has been described in patients with severe craniofacial deviations (Becktor *et al.*, 2000). In that study, 177 lateral cephalometric radiographs were reviewed and a sella turcica bridge was observed in 18.6 per cent of subjects. In patients treated by combined surgical orthodontics, the incidence of bridging was reported to be 16.7 per cent, but only 7.3 per cent in the group treated orthodontically (Jones *et al.*, 2005).

However, more frequently, an altered morphology of the sella turcica seems to be caused by congenital malformations. In fact, sella bridging has been reported as a radiographic feature of basal cell carcinoma (Gorlin–Goltz) syndrome, along with calcification of the falx cerebri (Kimonis *et al.*, 1997).

An altered sella turcica morphology, or bridging of the sella, is also present in some patients with other disorders and syndromes (Kjær *et al.*, 1998, 1999, 2001; Russell and Kjær, 1999). In these subjects, the altered structure of the sella turcica has been related to the syndrome.

Formation and development of the sella turcica and teeth share, in common, the involvement of neural crest cells. In fact, the anterior part of the sella turcica is believed to develop mainly from neural crest cells, and dental epithelial progenitor cells differentiate through sequential and reciprocal interaction with neural crest-derived mesenchyme (Miletich and Sharpe, 2004; Morotomi *et al.*, 2005). Notwithstanding this developmental relationship, no systematic study has been undertaken to examine the presence of any association between a sella turcica bridge and dental anomalies. The only previous study (Tetradis and Kantor, 1999) was limited to describing the prevalence of dental anomalies and skull calcification and normal variants seen on cephalometric radiographs without presenting any correlations between them.

Accordingly, this retrospective study was performed to evaluate the prevalence of a sella turcica bridge in young subjects with congenital dental anomalies (Pirinen *et al.*, 1996;

Arte *et al.*, 2001; Peck *et al.*, 2002), such as a palatally displaced canine (PDC) or missing mandibular second premolar.

Subjects and methods

Study population

The treatment records of 34 Caucasian subjects (20 males and 14 females), aged between 8 and 16 years, with a PDC or missing mandibular second premolar were selected from a large number of patients seen during a 24-month period in the Department of Orthodontics, University of Catania. For inclusion in the study, the case records were required to include good quality lateral cephalometric radiographs and dental pantomograms. The exclusion criteria were the presence of a cleft lip and palate or other craniofacial anomaly or syndrome, trauma and multi-reagent chemotherapy, and subjects with severe craniofacial deviations who had combined surgical-orthodontic treatment (Becktor *et al.*, 2000; Jones *et al.*, 2005). The PDC group comprised 18 subjects (11 males and 7 females), and the missing mandibular second premolar group 16 consecutively treated subjects (9 males and 7 females).

The diagnosis was made on the basis of clinical examination and diagnostic radiographs, according to established standardized techniques (Seward, 1963; Hunter, 1981; Becker, 1998).

Control group

A power analysis suggested that a sample size of 135 patients was required to significantly evaluate differences in the calcification of the sella turcica between subjects and a control group. Thus, the control group consisted of 135 subjects (81 males and 54 females). This group represented randomly selected Caucasians treated at the University Orthodontic Clinic, during the previous 3 years. The exclusion criteria were the same as for the study population.

Since age has been identified as a risk factor for calcification of the sella turcica (Cederberg *et al.*, 2003), and the dissimilar age distribution in the sample with respect to the population could distort the prevalence of calcification, the sample was post-stratified according to age distribution of the population of Catania (Table 1). The demographics statistics of the population, at 1 January 2001, was obtained from the National Institute of Statistics (http:www.istat.it), and used to determine the weights of each stratum. The post-stratification thus helped to improve the estimator of prevalence by taking advantage of the correct utilization of appropriate sources of information.

Cephalometric analyses

In order to quantify the extent of a sella turcica bridge from each profile radiograph, the length and diameter were calculated (Axelsson *et al.*, 2004a,b). Briefly, the sella turcica was measured by tracing the contour of the pituitary fossa from the tip of the dorsum sellae to the tuberculum sella. The length of the sella turcica was measured as the distance from the tuberculum sella to the tip of the dorsum sellae; this straight line corresponded to the position of diaphragma sellae. The antero-posterior greatest diameter of the sella turcica was measured from the tuberculum sella to the most posterior point on the inner wall of the pituitary fossa.

The cephalometric radiographs were traced and measured manually by one observer (RL), in a dark room. The tracings were made on ultraphan transparent acetate sheets (20.3 × 25.4 cm with a thickness of 0.003 inches), on a luminator using a Pentel 0.5-mm lead pencil.

A standardized scoring scale was established which consisted of comparing measurements of the sella turcica length and diameter. If the length of the sella turcica was greater than or equal to three-fourths of the diameter, the sella was scored as Class I (no calcification); if less than or equal to three-quarters (ICL partially calcified) as Class II; and Class III for a radiographically visible diaphragma sella (ICL completely calcified).

Method error

Duplicate tracings of 20 films were made on two separate occasions by the same author with a 2-week interval between tracings, and the random error was assessed as described by Dahlberg (1940) and Houston (1983). The difference between the first and second measurements of the 20 radiographs was not significant.

The measurement errors ranged from 0.12 to 0.18 mm for both length and diameter and were not significant. On this basis, it was considered that the experimental error was unlikely to bias the accuracy of the sella measurements.

Table 1 Distribution and standard deviation (SD) by age and gender of the study population.

Age (years)	N	Mean	SD	Median	Female %	Male %
8–10	45	9.0	0.826	9	51.1 (23)	48.9 (22)
11–13	44	12.0	0.835	12	43.2 (19)	56.8 (25)
14–16	46	15.0	0.815	15	26.1 (12)	73.9 (34)
Total	135	12.0	2.621	12	40.0 (54)	60.0 (81)

Statistical analyses

Statistical comparisons included independent chi-square tests, Spearman's correlations, and ordinal logistic regression in order to evaluate any dependence between the three classes of degree of calcification, and gender, age, and dental anomalies.

Results

Data on the age and gender distribution of the study sample are reported in Table 2.

Chi-square statistics were computed to evaluate whether the conditional distribution of the degree of calcification for males was similar to that for females. The test was not significant ($P = 0.076$) suggesting no dependence between the degree of calcification and gender (data not shown).

Spearman's rank correlation between age in the Classes and the degree of calcification was significant ($P = 0.003$) with a weak positive correlation $\rho = 0.255$, suggesting a slightly increased degree of ICL calcification for increased age. The distribution of the age in Classes and the degree of calcification of the adolescents are shown in Table 3.

The distribution of the degree of calcification for adolescents with and without dental anomalies is shown in Table 3. The prevalence of complete calcification in adolescents without anomalies was 9.9 per cent, while for those with a dental anomaly it was 17.6 per cent. In adolescents without anomalies, the prevalence of a partially calcified sella turcica ($>3/4$) was 33.7 per cent and increased to 58.8 per cent for adolescents with dental anomalies (Table 3).

Chi-square statistics confirmed that there was a significant association between ICL degree of calcification and the presence of dental anomalies ($P = 0.004$). Sixteen per cent of cells had expected values of less than 5; therefore, Fisher's exact test was used to confirm the chi-square test.

This statistic was also significant ($P = 0.003$) and equal to 11.462, thus verifying the hypothesis that calcification of sella turcica bridging is associated with dental anomalies.

For the adolescents with dental anomalies (34 subjects), the prevalence of those with a PDC that had a partially calcified sella turcica (Figure 1) increased to 77.8 per cent (Table 4), while for those with missing second premolars, it was lower (37.5 per cent), although higher in adolescents with no dental anomalies (33.7 per cent). Overall, a sella turcica bridge (Figure 2) was observed in 16.7 per cent of subjects with a PDC and in 18.7 per cent of patients with missing second premolars.

The result of ordinal logistic regression (PoLytomous Universal Model) where the degree of calcification is predicted by gender, age, and dental anomalies is shown in the tables. Significant chi-square statistics equal to 21.12 ($P = 0.0$) indicate that the model is a significant improvement over the baseline intercept-only model. It can be observed that the significant parameters for age ($P = 0.004$) and dental anomalies ($P = 0.008$) confirm that these two predictors significantly explain the degree of calcification of the sella turcica (Table 5).

Discussion

Calcification of diaphragma sellae, which radiologically has been described as 'roofing' or 'bridging' of the sella, in the absence of clinical signs or symptoms, is considered a normal variant of the sella turcica (Kantor and Norton, 1987), although many pathological processes can be associated with this calcification.

As far as aetiology is concerned, it has been suggested that an ICL is laid down in cartilage at an early stage of development and then ossifies in very early childhood. This ossification can be due to the complex embryology of the

Table 2 Degree of calcification of the sella turcica by age (chi-square = 14.625, $P = 0.005$).

(*n*) % within age	8–10	11–13	14–16	Total
Sella < 3/4 calcified	(28) 62.2%	(25) 56.8%	(12) 26.1%	(65) 48.1%
3/4 ≤ sella < 1 calcified	(12) 26.7%	(16) 36.4%	(26) 56.5%	(54) 40.0%
Complete calcification	(5) 11.1%	(3) 6.8%	(8) 17.4%	(16) 11.9%
Total	(45) 100.0%	(44) 100.0%	(46) 100.0%	(135) 100.0%

Table 3 Degree of calcification of the sella turcica in adolescents with and without dental anomalies (chi-square = 11.034, $P = 0.004$).

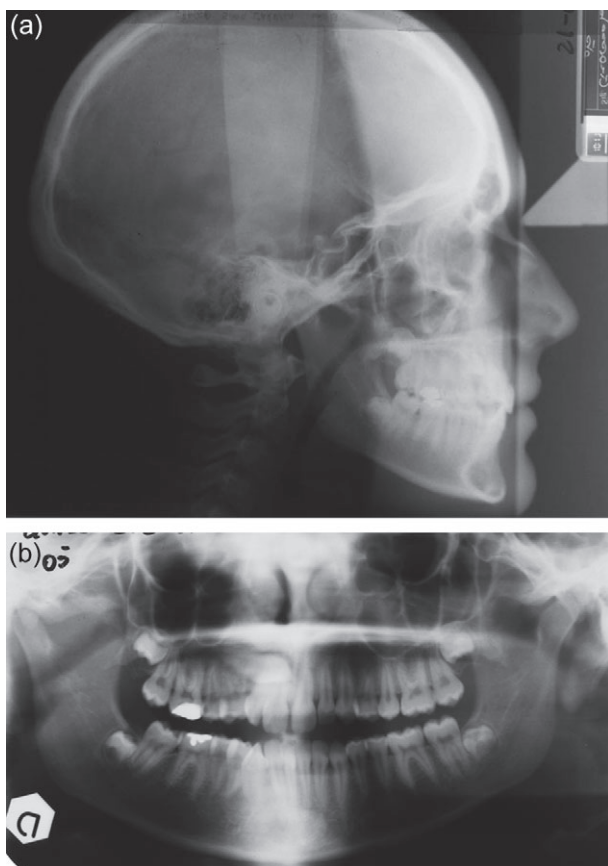| (*n*) % within anomalies	Adolescents without dental anomalies	Adolescents with dental anomalies	Total
Sella < 3/4 calcified	(57) 56.4%	(8) 23.5%	(65) 48.1%
3/4 ≤ sella < 1 calcified	(34) 33.7%	(20) 58.8%	(54) 40.0%
Complete calcification	(10) 9.9%	(6) 17.6%	(16) 11.9%
Total	(101) 100.0%	(34) 100.0%	(135) 100.0%

Figure 1 Lateral cephalometric radiograph (a) and dental pantomogram (b) of a patient, with a palatally displaced canine. The sella turcica is partially calcified.
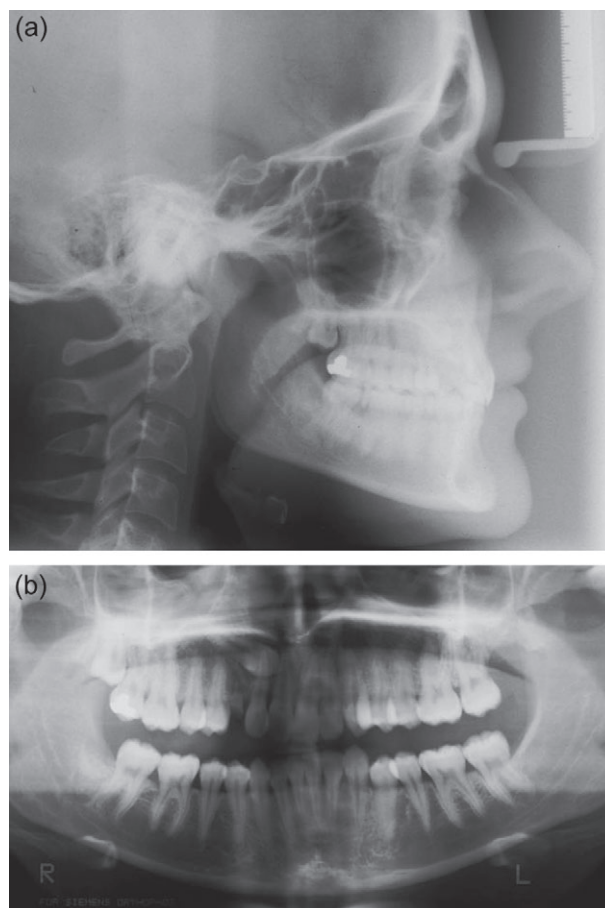

Figure 2 Lateral cephalometric radiograph (a) and dental pantomogram (b) of a patient, with a palatally displaced canine. The sella turcica is completely calcified.

sphenoid bone (Hochstetter, 1940; Kier, 1966; Lang, 1977; Inoue *et al.*, 1990; Becktor *et al.*, 2000). According to this theory, a sella turcica bridge should be considered a developmental anomaly. Moreover, as the area anterior to the sella turcica in the early embryonic period develops predominantly from neural crest cells, any structural deviations in the anterior wall are believed to be related to specific deviations in the facial skeleton (Kjær *et al.*, 1998).

In this study, the prevalence of a sella turcica bridge was investigated in a group of patients with tooth abnormalities (i.e. the presence of a PDC or congenital absence of the mandibular the second premolar) and compared with normal subjects.

Table 4 Degree of calcification according to dental anomalies (chi-square = 6.553, P = 0.041).

(n) % within sella, % within anomalies	Palatal displaced canine	Aplasia	Total
Sella < 3/4 calcified	(1) 5.6%	(7) 43.8%	(8) 21.2%
3/4 ≤ sella < 1 calcified	(14) 77.8%	(6) 37.5%	(20) 60.6%
Complete calcification	(3) 16.7%	(3) 18.7%	(6) 18.2%
Total	(18) 100.0%	(16) 100.0%	(34) 100.0%

Among tooth anomalies, a PDC and second premolar hypodontia were chosen as they have been reported as belonging to the same spectrum of dental abnormalities (Pirinen *et al.*, 1996; Baccetti, 1998; Peck *et al.*, 2002) which is genetically determinated and phenotypically heterogenous (Pirinen *et al.*, 1996; Arte *et al.*, 2001).

The data from this study suggest an incidence of 9.9 per cent for complete calcification for the ICL in the control sample. This result is consistent with the reported radiographic data of a sella turcica bridge in normal subjects (Becktor *et al.*, 2000; Cederberg *et al.*, 2003). On the other hand, patients with tooth abnormalities showed a frequency of 17.6 per cent for a sella turcica bridge (16.7 per cent in patients with a PDC and 18.7 per cent in subjects with second premolar agenesia) which is similar to that for subjects with severe craniofacial deviations (Becktor *et al.*, 2000). The presence of a partially calcified sella was also increased in patients with dental abnormalities (60.6 per cent) versus control group (33.7 per cent).

Molecular studies of odontogenesis, using the mouse tooth as a model, have shown that tooth development is under strict genetic control, which determines tooth position,

Table 5 Nominal logistic regression: parameter estimates.

		Estimated coefficient	Standard error	Wald statistics[a]	df	Significance	95% confidence interval	
							Lower bound	Upper bound
Threshold	(Sella = <3/4)	1.387	0.956	2.104	1	0.147	−0.487	3.261
	(Sella = <1)	3.751	1.005	13.921	1	0.000	1.781	5.722
Location	Age	0.201	0.070	8.244	1	0.004	0.064	0.338
	(Gender = male)	−0.404	0.366	1.215	1	0.270	−1.122	0.314
	(Gender = female)	0[b]	—	—	—	—	—	—
	(Anomalies = no)	−1.053	0.400	6.948	1	0.008	−1.837	−0.270
	(Anomomalies = yes)	0[b]	—	—	—	—	—	—

df, degrees of freedom; Link function: Logit.

[a]The ratio of the estimated coefficient to its standard error, squared, equals the Wald statistic.

[b]This parameter is set to zero because it is redundant.

number, size, and shape (Thesleff, 1998; Peters and Balling, 1999; Vastardis, 2000). Although tooth agenesis is occasionally caused by environmental factors, such as trauma of the dental region or by multi-reagent chemotherapy or radiotherapy (Näsman *et al.*, 1997), the majority of cases of hypodontia and oligodontia are due to genetic factors. Mutations of several genes are associated with syndromic tooth agenesis. To date, the familial and sporadic forms of tooth agenesis have been associated with mutations in MSX1 and PAX9 (Mostowska *et al.*, 2003). Interestingly, MSX1- and PAX9-deficient mice exhibit several other craniofacial abnormalities (Satokata and Maas, 1994; Peters *et al.*, 1998).

As far as a PDC is concerned, there are many theories as to why canine impaction occurs, but they can be separated into two categories: guidance and genetics. Abnormal tooth eruption, abnormal eruption rate, and delayed resorption of primary teeth as possible guidance factors have been cited (Bishara *et al.*, 1976). Evidence for a genetic aetiology has many forms, among which is an association between canine impactions and other dental anomalies. Baccetti (1998) and Peck *et al.* (2002) reported that 33 per cent of patients with impacted canines had other congenitally missing teeth.

Furthermore, it has been claimed that a PDC and second premolar aplasia appear to be different manifestation of one syndrome with incomplete penetrance and variable expressivity (Pirinen *et al.*, 1996; Baccetti, 1998). According to the present findings, the hypotheses that some tooth anomalies are genetically linked and related to each other should be broadened, i.e. other signs in the craniofacial skeleton may also be present in these patients.

No association between tooth anomalies and other signs in the craniofacial skeleton in non-syndromic patients, except for that found in this investigation, has been previously reported. If in humans, as in mice with tooth agenesis (Satokata and Maas, 1994; Peters *et al.*, 1998), there may be other craniofacial abnormalities that deserve further studies.

Last, but not least, is the clinical importance of the present findings. For many orthodontists being able to determine whether canine impaction will occur is a dilemma and the timing for preventive treatment of impacted canine is of paramount importance for a successful treatment outcome.

Conclusions

There are several implications of this newly interrelated sella turcica bridge to dental abnormalities:

1. The prevalence of sella turcica bridge in adolescents with dental anomalies is increased.
2. Age and gender do not significantly influence ossification of the ICL.
3. The very early appearance in life of sella turcica bridge, as described by Axelsson *et al.* (2004b), should alert clinicians to the possible development of tooth anomalies later in life, as these two phenomenons are to a certain extent associated.

Address for correspondence

Rosalia Leonardi
Istituto di II Clinica Odontoiatrica
Citta' Universitaria
Università di Catania
Via S. Sofia n.78 Policlinico
Catania 95123
Italy
E-mail: rleonard@unict.it

References

Arte S, Nieminen P, Apajalahti S, Haavikko K, Thesleff I, Pirinen S 2001 Characteristics of incisor-premolar hypodontia in families. Journal of Dental Research 80: 1445–1450

Axelsson S, Storhaug K, Kjær I 2004a Post-natal size and morphology of the sella turcica in Williams syndrome. European Journal of Orthodontics 26: 613–621

Axelsson S, Storhaug K, Kjær I 2004b Post-natal size and morphology of the sella turcica. Longitudinal cephalometric standards for Norwegians between 6 and 21 years of age. European Journal of Orthodontics 26: 597–604

Baccetti T 1998 A controlled study of associated dental anomalies. The Angle Orthodontist 68: 267–274

Becker A 1998 The orthodontic treatment of impacted teeth. Martin Dunitz Ltd, London

Becktor J P, Einersen S, Kjær I 2000 A sella turcica bridge in subjects with severe craniofacial deviations. European Journal of Orthodontics 22: 69–74

Bishara S E, Kommer D D, McNeil M H, Montagano L N, Oesterle L J, Youngquist H W 1976 Management of impacted canines. American Journal of Orthodontics 69: 371–387

Bisk S, Lee F A 1976 Abnormalities found on cephalometric radiographs. The Angle Orthodontist 46: 381–386

Cederberg R A, Benson B W, Nunn M, English J D 2003 Calcification of the interclinoid and petroclinoid ligaments of sella turcica: a radiographic study of the prevalence. Orthodontics & Craniofacial Research 4: 227–232

Dahlberg G 1940 Statistical methods for medical and biological students. Allen and Unwin, London

Hochstetter F 1940 Über die Taenia interclinoidea, die Commissura alicochlearis und die Cartilago supracochlearis des menschlichen Primordialkraniums. Gegenbaurs Morphologisches Jahrbuch 84: 220–243

Houston W J B 1983 The analysis of errors in orthodontic measurements. American Journal of Orthodontics 83: 382–390

Hunter S B 1981 The radiographic assessment of the unerupted maxillary canine. British Dental Journal 150: 151–155

Inoue T, Rhoton Jr A L, Theele D, Barry M E 1990 Surgical approaches to the cavernous sinus: a microsurgical study. Neurosurgery 26: 903–932

Jones R M, Faqir A, Millett D T, Moos K F, McHugh S 2005 Bridging and dimensions of sella turcica in subjects treated by surgical-orthodontic means or orthodontics only. The Angle Orthodontist 75: 714–718

Kantor M L, Norton L A 1987 Normal radiographic anatomy and common anomalies seen in cephalometric films. American Journal of Orthodontics and Dentofacial Orthopedics 91: 414–426

Kier E L 1966 Embryology of the normal optic canal and its anomalies. An anatomic and roentgenographic study. Investigative Radiology 1: 346–362

Kimonis V E et al. 1997 Clinical manifestations in 105 persons with nevoid basal cell carcinoma syndrome. American Journal of Medical Genetics 69: 299–308

Kjær I, Becktor K B, Lisson J, Gormsen C, Russell B G 2001 Face, palate, and craniofacial morphology in patients with a solitary median maxillary central incisor. European Journal of Orthodontics 23: 63–73

Kjær I, Hansen B F, Reintoft I, Keeling J W 1999 Pituitary gland and axial skeletal malformations in human fetuses with spina bifida. European Journal of Pediatric Surgery 9: 354–358

Kjær I, Keeling J W, Reintoft I, Nolting D, Fischer Hansen B 1998 Pituitary gland and sella turcica in human trisomy 21 fetuses related to axial skeletal development. American Journal of Medical Genetics 80: 494–500

Lang J 1977 Structure and postnatal organization of heretofore uninvestigated and infrequent ossifications of the sella turcica region. Acta Anatomica 99: 121–139

Miletich I, Sharpe P T 2004 Neural crest contribution to mammalian tooth formation. Birth Defects Research. Part C, Embryo Today: Reviews 72: 200–212

Morotomi T et al. 2005 In vitro differentiation of dental epithelial progenitor cells through epithelial-mesenchymal interactions. Archives of Oral Biology 50: 695–705

Mostowska A, Kobielak A, Trzeciak W H 2003 Molecular basis of non-syndromic tooth agenesis: mutations of MSX1 and PAX9 reflect their role in patterning human dentition. European Journal of Oral Sciences 111: 365–370

Näsman M, Forsberg C M, Dahllöf G 1997 Long-term dental development in children after treatment for malignant disease. European Journal of Orthodontics 19: 151–159

Peck S, Peck L, Kataja M 2002 Concomitant occurrence of canine malposition and tooth agenesis: evidence of orofacial genetic fields. American Journal of Orthodontics and Dentofacial Orthopedics 122: 657–660

Peters H, Balling R 1999 Teeth: where and how to make them. Trends in Genetics 15: 59–65

Peters H, Neubuser A, Kratochwil K, Balling R 1998 Pax9-deficient mice lack pharyngeal pouch derivatives and teeth and exhibit craniofacial and limb abnormalities. Genes and Development 12: 2735–2747

Pirinen S, Arte S, Apajalahti S 1996 Palatal displacement of canine is genetic and related to congenital absence of teeth. Journal of Dental Research 75: 1346–1352

Russell B G, Kjær I 1999 Postnatal structure of the sella turcica in Down syndrome. American Journal of Medical Genetics 87:183–188

Satokata I, Maas R 1994 Msx1 deficient mice exhibit cleft palate and abnormalities of craniofacial and tooth development. Nature Genetics 6: 348–356

Seward G R 1963 Radiology in general dental practice. IX-unerupted maxillary canines, central incisors and supernumeraries. British Dental Journal 115: 85–91

Tetradis S, Kantor M L 1999 Prevalence of skeletal and dental anomalies and normal variants seen in cephalometric and other radiographs of orthodontic patients. American Journal of Orthodontics and Dentofacial Orthopedics 116: 572–577

Thesleff I 1998 The genetic basis of normal and abnormal craniofacial development. Acta Odontologica Scandinavica 56: 321–325

Vastardis H 2000 The genetics of human tooth agenesis: new discoveries for understanding dental anomalies. American Journal of Orthodontics and Dentofacial Orthopedics 117: 650–656