The palatally displaced canine as a dental anomaly of genetic origin

Sheldon Peck, DDS, MScD; Leena Peck, DMD, MSD; Matti Kataja, PhD

The unerupted maxillary canine tooth in man is sometimes dislocated from its usual position within the dentoalveolar process, disturbing its normal eruption and often resulting in nonemergence (impaction). From existing data, the prevalence rate of maxillary canine impaction varies from less than 1% to 3%.[1-7]

Most canine impactions diverge from the normal eruptive site in either of two directions: palatal or facial. In European samples, the impacted maxillary canine has been found palatally at least two to three times more frequently than facially.[1,8-13] These two canine malpositions — facial and palatal displacement — are actually very different phenomena, although they seldom have received separate consideration in studies of impacted teeth. *Facial* displacement of the maxillary canine is usually due to inadequate arch space and it eventually results in eruption in most cases. In contrast, *palatal* displacement of the maxillary canine is a positional anomaly that generally occurs despite adequate arch space and which characteristically leads to impaction of the tooth, unless measures like deciduous canine extraction, surgical exposure and orthodontic treatment are implemented at appropriate times.

The palatally displaced canine — the focus of this article — has been identified in human skulls dating from prehistory[14] through medieval times[15] to the eighteenth century,[16] and continuing to the present. Varied theories and hypotheses have been offered regarding the reasons for palatal displacement of the maxillary canine. The cause-and-effect associations most often advanced suggest palatal canine displacement as happening because of a number of local conditions and factors: (1) retained

Abstract

Palatal displacement of the maxillary canine tooth is a positional variation thought generally to develop as a result of local factors, such as retained deciduous canines, anomalous permanent lateral incisors, or dental crowding. This article contributes biologic evidence pointing to genetic factors as the primary origin of most palatal displacements and subsequent impactions of maxillary canine teeth. Data gathered from multiple sources are integrated to support a genetic etiology for the palatally displaced canine (PDC) on the basis of five evidential categories: 1. Occurrence of other dental anomalies concomitant with PDC; 2. Bilateral occurrence of PDC; 3. Sex differences in PDC occurrence; 4. Familial occurrence of PDC; 5. Population differences in PDC occurrence. From analysis of available evidence, the PDC positional anomaly appears to be a product of polygenic, multifactorial inheritance.

Key Words

Tooth eruption, ectopic • Canine, impacted • Tooth abnormalities • Genetics

Submitted: January 1994 **Accepted for publication:** March 1994 Angle Orthod 1994;64(4)249-256

deciduous canines;[17,18,20-23] (2) anomalous or missing lateral incisors;[11,22-32] (3) crowded or delayed eruptive pathways;[18-20,26,33] and (4) other local conditions and circumstances.[11,20-3,32,34-36]

A few clinicians have referred to heredity as an etiologic factor.[21,35,37-39] More significantly, family studies report palatal canine displacement as an inherited trait: Racek and Sottner[40-43] studying Czech families, and Svinhufvud, Myllärniemi and Norio[44] examining Finnish families.

This article is written to bring together and contribute biologic evidence pointing to genetic factors as the primary origin of most palatal displacements and subsequent impactions of maxillary canine teeth.

In the text that follows, the palatally displaced canine will be referred to as PDC.

Genetic control of the palatally displaced canine (PDC): the evidence

Data gathered from multiple sources will be cited to provide evidence supporting a genetic etiology for PDC. Five evidential categories are addressed:

1. Occurrence of other dental anomalies concomitant with PDC.
2. Bilateral occurrence of PDC.
3. Sex differences in PDC occurrence.
4. Familial occurrence of PDC.
5. Population differences in PDC occurrence.

Occurrence of other dental anomalies concomitant with PDC

Several published studies of PDC have recorded increased frequency of associated dental anomalies and variations.

Bass[36] in 1967 and Weise and Anbuhl[45] in 1969 were perhaps the earliest to include certain variations in tooth position as associated phenomena in what seemed to be a pattern of concomitantly occurring dental anomalies. They noted an interrelationship among observed anomalies of tooth position, number, and size and suggested a shared etiology, without actually recognizing heredity as the basis. They reported that orthodontic patients with characteristic displacements of the maxillary canine, such as palatal impaction, or transposition with the first premolar, showed increased frequency of absence or malformation of other teeth, especially the maxillary lateral incisors.

In 1977, Racek and Sottner[40] described a sample of 92 dental patients with PDC. Their data indicated that 33% of their PDC sample also exhibited congenital absence of various teeth (excluding third molars), along with tooth-size reductions. Normal prevalence rates of tooth agenesis — an anomaly under genetic control — range from 3.5% to 8%,[46-49] so this 33% represents a four to nine times increase in occurrence over the general population. From this and subsequent findings, Racek and Sottner[40-43] recognized that PDC was likely an inherited trait. Thus, they proposed that PDC was one of the anomalies in a complex of genetically related dental disturbances often occurring in combination, which included tooth agenesis, tooth size reductions, supernumerary teeth, and other ectopically positioned teeth.

Later investigations[25,27-32,44] of the PDC phenomenon have also reported clear associations with tooth agenesis and tooth malformations, particularly involving maxillary lateral incisors. In PDC cases, increased congenital absence of one or both lateral incisors was observed. Furthermore, the existing maxillary lateral incisors in instances of PDC often exhibited significant size reductions, including shortened roots and conical "peg-shaped" crowns.

Crown-size reductions affecting all the teeth are commonly present in people with PDC.[42] In orthodontic terms, this variation is manifested by the infrequency of dental crowding, giving the preponderance of PDC-patient treatments an obvious "nonextraction" diagnostic appearance.

A delayed pattern of development and eruption throughout the dentition is frequently seen concomitant with the PDC trait. Some early articles[18,33] interpreted the slow eruptive pace of the maxillary canines in particular as a primary reason for its palatal displacement and impaction, rather than as a developmental covariable.

Garn, Lewis and co-workers[50-52] found that a genetic interrelationship existed among tooth agenesis, systematic tooth-size reduction and generalized retardation of tooth development — three of the abnormalities that appear associated with the PDC phenomenon. Moreover, another positional anomaly of the maxillary canine, transposition with the first premolar (Mx.C.P1 transposition), also occurs with remarkable frequency in combination with these specific anomalies and, like them, it strongly appears to be of polygenic origin.[53] The evidence, then, points clearly to the idea that PDC is not an isolated phenomenon, but instead an anomaly like Mx.C.P1 transposition, frequently occurring in association with several genetically interrelated dental anomalies.

Bilateral occurrence of PDC

Data regarding bilateral occurrence of PDC is often obscured by the failure of studies to distinguish palatal impactions from the other types. Table 1 displays results gathered from samples in ten investigations which have made this distinction, identifying PDC by roentgenographic analy-

Table 1
Published samples with palatally impacted/displaced maxillary canines

Study	Nationality Subjects	Number Subjects	Mean Age	Location R	Location L	Location Bilateral	Sex Ratio
Paatero, Kiminki[8], 1962	Finnish	238	—	106 44%	88 37%	44 19%	M1 : F2.4
Nordenram, Stromberg[9], 1966	Swedish	375	19.3	135 36%	115 31%	125 33%	M1 : F1.8
McKay[12], 1978	Irish	878	—	360 41%	367 42%	151 17%	M1 : F2.7
Fleury, Deboets, et al.[13], 1985	French	188	—	78 41%	74 39%	36 20%	M1 : F1.3
Becker, Smith, Behar[27], 1981	Israeli	88	—	R+L =	48 55%	40 45%	M1 : F2.4
Zilberman, Cohen, Becker[31], 1990	Israeli	25	—	10 40%	8 32%	7 28%	M1 : F2.1
Racek, Sottner[40], 1977	Czech	92	18.5	39 42%	32 35%	21 23%	M1 : F3.2
Racek, Sottner[42], 1984	Czech	179	18.0	—	—	—	M1 : F2.3
Ericson, Kurol[54], 1988	Swedish	35	11.4	R+L =	24 69%	11 31%	M1 : F1.5
Power, Short[55], 1993.	British	39	11.2	R+L =	31 79%	8 21%	M1 : F2.3

sis.[8,9,12,13,27,31,40,42,54,55] Nine have reported bilateral occurrences of PDC ranging from 17% to 45% of the total number of PDC cases in each study (Table 1). This rate of bilateralism is elevated beyond chance occurrence, reasonably pointing to an intrinsic etiology such as a genetic mechanism. (Note: *Facial* displacement of the maxillary canines also occurs bilaterally, but this is a totally different phenomenon, occurring for reasons not directly related to genetics. The maxillary canines express bilateral facial displacement — their ectopic path of least resistance — usually as a result of generalized crowding or space deficiency within the dental arch, in the same manner mechanical causes are responsible for the high frequency of bilateral impactions found among mandibular third molars.)

Prevalence rates of bilateralism for specific dental anomalies under genetic control appear remarkably similar to that of PDC, giving support for a comparable genetic origin in the instance of PDC. In four definitive studies of maxillary lateral incisor agenesis, bilateral occurrence comprised 29% to 46% of cases of this anomaly.[47,48,56,57] Furthermore, maxillary canine - first premolar transposition was found to occur bilaterally in 23% to 43% of cases studied.[53]

Sex differences in PDC occurrence

Sex ratios, concisely reporting patterns of male-female differences in occurrence, are useful in identifying biological phenomena with genetic links involving the sex chromosomes. Table 1 indicates dominance of female occurrence in all of the sex ratios calculated from 10 samples of PDC cases. These male-to-female prevalence rate ratios ranged from M1:F1.3 to M1:F3.2.

These data compare favorably with sex ratios recorded for other dental anomalies of genetic origin. Hypodontia, a polygenic trait, has been found with sex ratios ranging from M1:F1.3 to M1:F1.6.[47-49,57-59] A sample of maxillary canine - first premolar transposition cases showed a sex ratio of M1:F3.8, amplifying this observed trend.[53]

Familial occurrence of PDC

Most orthodontists can recall treating palatally impacted canines of patients who have had family members (siblings, parents, grandparents) with the same anomaly. This observation is found occasionally in clinical articles on the diagnosis and orthodontic treatment of PDC.[35,38,60]

Results of family studies of probands with the PDC anomaly have been reported by three groups of investigators: Racek and Sottner;[41,42] Svinhufvud, Myllärniemi and Norio;[44] Zilberman, Cohen and Becker.[31] From examining the pedigrees of PDC patients, all three groups found evidence of elevated occurrence rates of PDC and various related dental anomalies among other family members. Based on quantitative analysis of sibling and three-generational data on PDC trait occurrence in families, Sottner and Racek[41-42] have suggested a polygenic mode of transmission for the PDC anomaly. Svinhufvud, Myllärniemi and Norio[44] proposed an autosomal dominant inheritance pattern for PDC, and Zilberman, Cohen and Becker[31] held to a mechanical chain-reaction etiology for PDC, dependent on reduced or absent lateral incisors.

Population differences in PDC occurrence

A dichotomy in PDC prevalence seems to exist between people of European ancestry and those of Asian or African ancestry. The preponderance of published cases of canine palatal displacement/impaction are of European origin. Reports and studies of PDC in the dentitions of Africans or Asians are quite rare.

Kramer and Williams[4] found 1.2% of a predominantly (95%) African-American sample showing maxillary canine impactions of all types, including palatal and facial impactions. Being at the low end of the 1%-to-3% range of prevalence established by epidemiologic studies of impacted maxillary canines,[3,5-7] this may suggest the likelihood of a lower prevalence of PDC in African blacks than European whites. Moreover, the classic monographs on the dentitions of blacks in Africa fail to uncover occurrences of PDC.[61-63]

Montelius[2] in 1932 investigated the frequency of impacted teeth observed by roentgenography in Chinese and Caucasians living in China, and he noted "the relative infrequency of impacted cuspids [in Chinese] as compared with the number in Caucasian peoples." Calculations from his data show the prevalence rate ratio for impacted maxillary canines to be 2:1, Caucasian to Chinese. Montelius did not distinguish between palatal and facial impactions, and neither have most other reports of cases of impacted maxillary canines among eastern Asians.[64-67] In the Asian case reports with published diagnostic films permitting localization of the impacted canines, rarely is palatal displacement seen; most often, the canine impaction appears caused by space deficiency in the maxillary dental arch, trapping the unerupted canine in the mid-alveolus or displacing it facially.[68-72]

Oliver, Mannion and Robinson[30] studied a sample of 29 Hong Kong Asians selected for unilaterally impacted maxillary canines. Their results indeed showed evidence helping to confirm the relative infrequency of PDC among Asians. They found palatal displacement in only 28% of the subjects, with the remaining 72% exhibiting canine displacement to the facial. In contrast, studies of maxillary canine displacement among Europeans have shown the reverse proportions, with PDC occurring in approximately 70% of cases.[1,8-13] If these European/Asian palatal:facial data are integrated into the Montelius-derived prevalence rate ratio, a numerical estimate may be calculated of the relative prevalence of PDC between Europeans and Asians:

Maxillary canine impaction, all types:

Prevalence rate ratio

European : Asian[2]

2 : 1

Maxillary canine impaction, palatal only (PDC):

Prevalence rate ratio

European : Asian[1,2,8-13,30]

0.7 [70%] x 2 : 0.3 [30%] x 1 =

1.4 : 0.3 =

European 5 : 1 Asian

This mathematical estimate, based on available data, indicates that Europeans may be expected to have palatally displaced canines at approximately five times the frequency expected among Asians.

Additional signs that the PDC anomaly is infrequent among Asians show up in the published details of the few case reports of Asians with maxillary canine impactions. The features of the reported malocclusions and their subsequent orthodontic treatments appear highly atypical for cases of the PDC anomaly: dental crowding;[68,70,71] Angle Class III malocclusion;[68] surgical canine exposures performed on the facial side of the alveolus;[68-70,72] and extraction orthodontic treatment.[68,70,71] As another indication of the low prevalence of PDC among Asians, no Asian family studies of PDC have been found in the literature.

All the above factors point to significantly different frequencies of PDC occurrence in different populations, concordant with racial groupings, evidence supportive of genetic involvement in the etiology of the PDC anomaly. Palatal displacement of the canine seems to be a predominantly European trait. Other orofacial anomalies showing racial predilections, such as cleft lip with or without cleft palate[73] and maxillary canine - first premolar trans-

position,[53] are thought to be products of polygenic, multifactorial inheritance. Thus, the PDC positional anomaly has a strong likelihood of being under the same kind of genetic control.

Critique of earlier hypotheses on PDC etiology

Mechanical causes — such as blockage from retained deciduous canines or from dental arch space inadequacy — were implicated in the earliest attempts to explain palatal displacement of canines, coming to us from the era before roentgenology. These mechanical explanations persist today, perhaps as a natural extension of common clinical experience with third molars. The mandibular third molars in modern man are the teeth most frequently involved in tooth impaction, or failure to erupt. These impactions are almost always a visible result of space inadequacy and mechanical obstruction by adjacent molar teeth, directly causing the noneruption. In contrast to the circumstances of mandibular third molar impaction, cases of palatally displaced canines routinely have adequate arch space for the affected canine; PDC orthodontic treatments are largely of the nonextraction type. Moreover, a retained deciduous tooth is a consequence of the canine dislocation palatally, not its cause. So, clearly, mechanical theories of etiology appear simplistic and inadequate in light of details of the associated features of the PDC anomaly.

Several investigations[22-32] have constructed arguments implicating lateral incisor variations in the genesis of palatal development of canines: they claim that peg-shaped, short-rooted or absent maxillary lateral incisors are causal factors in the development of PDC, based upon an empirical notion that the lateral incisor provides an essential guiding function for normal canine eruption. It appears true that palatal canine displacement and anatomical variations of the maxillary lateral incisor are often associated anomalies, but this implies nothing about causality. The questions should be, "Is PDC a dependent variable, as these studies would lead us to believe, or are the associated canine and lateral incisor phenomena covariables within a larger pattern of multiple anomalies?

Help in answering these questions comes from our recent study of maxillary canine - first premolar (Mx.C.P1) transposition, another disturbance of maxillary canine position associated with increased frequency of lateral incisor anomalies.[53] Mx.C.P1 transposition, an anomaly found to be of genetic origin, is the interchange of position of the maxillary canine and first premolar, with the canine displaced distally and often facially. As such, the canine is dislocated at a significant distance from the lateral incisor, reasonably precluding any chances of direct or mechanical interactions between the two teeth. In our published sample[53] of Mx.C.P1 transposition cases, 37% of the subjects showed absence or severe size reduction of the maxillary lateral incisors, a percentage comparable with frequencies reported by others[27-30] for the same kinds of lateral incisor anomalies associated with PDC. Therefore, there seems to be no reason to believe these coexistent conditions are causally related. They are simply covariables: coincident traits appearing within the context of a larger, biological mechanism of control, namely, genetics.

Svinhufvud, Myllärniemi and Norio[44] suggest an autosomal dominant inheritance pattern for ectopic eruption (including palatal *and* facial displacements) of the maxillary canine, since its closely related condition of hypodontia is accepted in their view as an autosomal dominant characteristic. Actually, the preponderance of evidence points to a polygenic origin, rather than a single-gene locus, for congenital tooth absence.[53,74-79] The significant differences in occurrence of PDC between the sexes and among racial groupings, as reported in this article, further support complex genetic influence consistent with polygenic control of the PDC trait. With the added likelihood of low penetrance[42] and increased phenotypic variability,[53] a multifactorial pattern of inheritance appears reasonable for the PDC anomaly.

Clinical implications

As with many other inherited anatomical conditions, early recognition and interception are worthwhile strategies in conservative clinical management of the PDC anomaly. Roentgenographic examinations and family histories are the basic tools for early detection. At present, the best interceptive procedure for a palatally developing, unerupted canine involves early extraction of the associated maxillary deciduous canine tooth at the time of diagnosis in the mixed dentition.[54,55,80,81]

The high probability of occurrence of other dental abnormalities in combination with PDC, such as congenital tooth absence and delayed eruption, should alert the orthodontist to approach treatment planning with special care and circumspection. Typically, orthodontic planning might begin early with extraction of the deciduous canine to help ameliorate the PDC position, and might finish late with orthodontic appliances to align the developmentally slowed permanent teeth.

The genetic basis for palatal displacement of the maxillary canine does not rule out the occasional influence of environmental and adventitious factors in the genesis of this positional anomaly. For ex-

ample, early dentofacial trauma has been identified as an apparently predisposing event for some canine dislocations, such as certain types of impactions and transpositions.[34,82] Therefore, early accidents must be included as a subject of pretreatment inquiry in collecting a germane history for the PDC patient.

For the future, intensive study should be accorded PDC and the other dental disturbances regularly seen with it, to perhaps unlock more understanding of the role of genetics in various conditions of malocclusion and to elucidate relationships among coincident dental anomalies. Quantitative family studies of PDC should be initiated to characterize further the underlying genetic mechanisms discussed in this analytical study.

Note

This work has developed in part as an outgrowth of an unpublished study entitled, "Clinical characteristics of patients with palatally impacted canines," presented by the first author before the Edward H. Angle Society of Orthodontists, Eastern Component, in Washington, D.C. on March 21, 1980.

Author address:

Dr. Sheldon Peck
1615 Beacon Street
Newton, MA 02168

S. Peck is an Assistant Clinical Professor of Orthodontics, Harvard School of Dental Medicine, Boston, MA.

L. Peck is an Assistant Clinical Professor of Orthodontics, Harvard School of Dental Medicine, Boston, MA.

M. Kataja is with the National Public Health Institute in Helsinki, Finland.

References

1. Röhrer A. Displaced and impacted canines. Int J Orthod Oral Surg Radiogr 1929;15:1003-20.
2. Montelius GA. Impacted teeth, a comparative study of Chinese and Caucasian dentitions. J Dent Res 1932;12:931-8.
3. Dachi SF, Howell FV. A survey of 3874 routine full-mouth radiographs II. A study of impacted teeth. Oral Surg Oral Med Oral Pathol 1961;14:1165-9.
4. Kramer RM, Williams AC. The incidence of impacted teeth. Oral Surg Oral Med Oral Pathol 1970;29:237-41.
5. Shah RM, Boyd MA, Vakil TF. Studies of permanent tooth anomalies in 7886 Canadian individuals, I. Impacted teeth. J Canad Dent Assoc 1978;44:262-4
6. Grover PS, Lorton L. The incidence of unerupted permanent teeth and related clinical cases. Oral Surg Oral Med Oral Pathol 1985;59:420-5.
7. Hirschfelder U, Petschelt A. Retention von Zähnen aus kieferorthopädischer Sicht. Dtsch Zahnärztl Z 1986;41:164- 70.
8. Paatero YV, Kiminki A. Jatkotutkimus retinoituneen yläkulmahampaan palato-labiaalisesta sijainnista. Suom Hammaslääk Toim 1962;58:294-300.
9. Nordenram A, Stromberg C. Positional variations of the impacted upper canine. Oral Surg Oral Med Oral Pathol 1966;22:711-4.
10. Herren P. Die Elongationsbehandlung oberer retinierter Eckzähne mit dem Drahtum-schlingungsverfahren. Fortschr Kieferorthop 1969;30:244-90.
11. Jacoby H. The etiology of maxillary canine impactions. Am J Orthod 1983;84:125-32.
12. McKay C. The unerupted maxillary canine, an assessment of the role of surgery in 2500 treated cases. Br Dent J 1978;145:207-10.
13. Fleury JE, Deboets D, Assaad-Auclair C, Maffre N, Sultan P. La canine incluse. Rev Stomatol Chir Maxillofac 1985;86:122- 31.
14. Iseri H, Uzel I. Impaction of maxillary canines and congenitally missing third molars. Description of an ancient skull (7250-6700 BC). Europ J Orthod 1993;15:1-5.
15. Twiesselmann F, Brabant H. Nouvelle observations sur les dents et les maxillaires d'une population ancienne d'Age Franc de Coxyde (Belgique). Bull Group Int Rech Sci Stomat 1967;10:5-180.
16. Colyer F. The teeth of Londoners of the 17th and 18th centuries. Dent Record 1922;42:237-43.
17. Angle EH. Treatment of malocclusion of the teeth. Angle's system. 7th ed. Philadelphia: SS White Dental Manufacturing Co, 1907:98.
18. Newcomb MR. Recognition and interception of aberrant canine eruption. Angle Orthod 1959;29: 161-8.
19. Mew JRC. Re Jacoby: etiology of maxillary canine impactions [letter]. Am J Orthod 1984;85:440-1.
20. Thilander B, Jakobsson SO. Local factors in impaction of maxillary canines. Acta Odontol Scand 1968;26:145-68.
21. Fastlicht S. Treatment of impacted canines. Am J Orthod 1954;40:891-905.
22. Hitchin AD. The impacted maxillary canine. Br Dent J 1956;100:1-14.
23. Kettle MA. Treatment of the unerupted maxillary canine. Dent Practit 1958;8:245-55.
24. Miller BH. The influence of congenitally missing teeth on the eruption of the upper canine. Dent Practit 1963;13:497- 504.
25. Becker A. Etiology of maxillary canine impactions [letter]. Am J Orthod 1984;86:437-8.
26. Moss JP. The unerupted canine. Dent Practit 1972;22:241-8.
27. Becker A, Smith P, Behar R. The incidence of anomalous maxillary lateral incisors in relation to palatally-displaced cuspids. Angle Orthod 1981;51: 24-9.
28. Becker A, Zilberman Y, Tsur B. Root length of lateral incisors adjacent to palatally-displaced maxillary cuspids. Angle Orthod 1984;54:218-25.
29. Brin I, Becker A, Shalhav M. Position of the maxillary permanent canine in relation to anomalous or missing lateral incisors: a population study. Europ J Orthod 1986;8:12-6.
30. Oliver RG, Mannion JE, Robinson JM. Morphology of the lateral incisor in cases of unilateral impaction of maxillary canine. Br J Orthod 1989;19:9-16.
31. Zilberman Y, Cohen B, Becker A. Familial trends in palatal canines, anomalous lateral incisors, and related phenomena. Europ J Orthod 1990;12:135-9.
32. Brin I, Becker A, Zilberman Y. Resorbed lateral incisors adjacent to impacted canines have normal crown size. Am J Orthod Dentofac Orthop 1993;104:60-6.
33. Dewel BF. The upper cuspid: its development and impaction. Angle Orthod 1949;19:79-90.
34. Brin I, Solomon I, Zilberman Y. Trauma as a possible etiologic factor in maxillary canine impaction. Am J Orthod Dentofac Orthop 1993;104:132-7.
35. Lewis PD. Preorthodontic surgery in the treatment of impacted canines. Am J Orthod 1971;60:382-97.
36. Bass TB. Observations on the misplaced upper canine tooth. Dent Practit 1967;18:25-33.
37. Takahama Y, Aiyama Y. Maxillary canine impaction as a possible microform of cleft lip and palate. Europ J Orthod 1982;4:275-7.
38. Richardson A, McKay C. Delayed eruption of maxillary canine teeth, Part I - aetiology and diagnosis. Proc Br Paedodont Soc 1982;12:15-25.
39. Chimenti C, Colangelo P, Accivile E. Classificazione ed etiopatogenesi dell'inclusione del canino superiore. Minerva Ortognat 1990;8:59-69.
40. Racek J, Sottner L. Prispevek k dedicnost retence spicáku. Cesk Stomat 1977;77:209-13.
41. Sottner L, Racek J. Stanoveni dedivosti. Model: retence spicáku. Cas Lék Cesk 1978;117:1060-2.
42. Racek J, Sottner L. Nase názory na dedicnost retence spicáku. Sborn Lék 1984;86:355-60.
43. Sottner L, Racek J, Marková M, Sládková M. Genetika v ortodoncii. Sborn Lék 1987;89:15-9.
44. Svinhufvud E, Myllärniemi S, Norio R. Dominant inheritance of tooth malpositions and their association to hypodontia. Clin Genetics 1988;34:373-81.
45. Weise W, Anbuhl B. Beitrag zur Keimverlagerung des Eckzahnes. Dtsch Zahnärztl Z 1969;24:803-8.
46. Grahnèn H. Hypodontia in the permanent dentition. Odontol Revy 1956;7(Suppl 3):1-100.
47. Muller TP, Hill IN, Peterson AL, Blayney JR. A survey of congenitally missing permanent teeth. J Am Dent Assoc 1970;81:101-7.

48. Davis PJ. Hypodontia and hyperdontia of permanent teeth in Hong Kong schoolchildren. Community Dent Oral Epidemiol 1987;15:218-20.
49. Haavikko K. Hypodontia of permanent teeth. An orthopantomographic study. Suom Hammaslääk Toim 1971;67:219-25.
50. Garn SM, Lewis AB, Vicinus JH. Third molar polymorphism and its significance to dental genetics. J Dent Res 1963;42:1344- 63.
51. Garn SM, Lewis AB, Kerewsky RS. Third molar agenesis and variation in size of the remaining teeth. Nature 1964;201:839.
52. Garn SM, Lewis AB. The gradient and the pattern of crown-size reduction in simple hypodontia. Angle Orthod 1970;40:51-8.
53. Peck L, Peck S, Attia Y. Maxillary canine - first premolar transposition, associated dental anomalies and genetic basis. Angle Orthod 1993;63:99-109.
54. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. Europ J Orthod 1988;10:283-95.
55. Power SM, Short MBE. An investigation into the response of palatally displaced canines to the removal of deciduous canines and an assessment of factors contributing to favourable eruption. Br J Orthod 1993;20:215-23.
56. Meskin LH, Gorlin RJ. Agenesis and peg-shaped permanent maxillary lateral incisors. J Dent Res 1963;42:1476-9.
57. Ringqvist M, Thilander B. The frequency of hypodontia in an orthodontic material. Svensk Tandläk Tidskr 1969;62:535-41.
58. Niswander JD, Sujaku C. Congenital anomalies of teeth in Japanese children. Am J Phys Anthrop 1963;21:569-74.
59. Rose JS. A survey of congenitally missing teeth, excluding third molars, in 6000 orthodontic patients. Dent Practit 1966;17:107-14.
60. Rayne J. The unerupted maxillary canine. Dent Practit 1969;19:194-204
61. Drennan MR. The dentition of a Bushman tribe. Annals So Afr Museum 1929;24:61-87.
62. Shaw JCM. The teeth, the bony palate and the mandible in Bantu races of South Africa. London: John Bale, Sons, and Danielsson. 1931:134p.
63. Jacobson A. The dentition of the South African Negro. Anniston, Alabama: Higginbotham. 1982:365p.
64. Kotoku K, Matsumoto Y, Yamaguchi H, Okamoto S. Two cases of impacted upper central incisor and canine [in Japanese]. Shikwa Gakuho 1979;79:607-10.
65. Nakano K, Tanaka S, Fukuda M, Okada N, Osawa K, Fujino E, Komine K, Awazawa I, Hondo T, Masuda T. Statistical analyses of impacted teeth in the last three years of department of oral diagnosis, Josai Dental University Hospital [in Japanese]. Bull Josai Dent Univ 1984;13:611-5.
66. Ozaki Y, Nakano Y, Marumori M, Sato K, Shigematsu T. Clinical statistical observation of impacted teeth by orthopantomograph [in Japanese]. Shikwa Gakuho 1986;86:1873-8.
67. Harada K, Arita K, Nishino M. Severe root resorption of maxillary incisors and ectopic eruption of maxillary canine [in Japanese]. Shoni Shikagaku Zasshi 1989;27:692-9.
68. Takeshita K, Shimoyama K, Matsuura A, Ohtsuka E, Taki H, Niwa M. Four cases of Class III malocclusion with impacted maxillary canine [in Japanese]. J Japan Orthod Soc 1972;31:247-65.
69. Sasakura H, Yoshida T, Murayama S, Hanada K, Nakajima T. Root resorption of upper permanent incisor caused by impacted canine. An analysis of 23 cases. Int J Oral Surg 1984;13:299-306.
70. Ito M, Nozaka K, Moriguchi O, et al. The clinical observation of exposure and traction of impacted teeth [in Japanese]. Shoni Shikagaku Zasshi 1986;24:643-52.
71. Suhr CH. Orthodontic considerations in maxillary impacted canines [in Korean]. Taehan Chikkwa Uisa Hyophoe Chi 1987;25:59-70.
72. Tang ELK. Multispecialty team management of a case with impacted maxillary permanent canines. ASDC J Dent Child 1992;59:190-5.
73. Gorlin RJ, Cohen Jr MM, Levin LS. Syndromes of the head and neck. 3rd ed. New York:Oxford University Press, 1990:695.
74. Woolf CM. Missing maxillary lateral incisors: a genetic study. Am J Hum Genet 1971;23:289-96.
75. Chosack A, Eidelman E, Cohen T. Hypodontia: a polygenic trait - a family study among Israeli Jews. J Dent Res 1975;54:16-9.
76. Suarez BK, Spence A. The genetics of hypodontia. J Dent Res 1974;53:781-5.
77. Brook AH. A unifying aetiological explanation for anomalies of human tooth number and size. Arch Oral Biol 1984;29:373-8.
78. Sofaer JA. Dental morphologic variation and the Hardy- Weinberg law. J Dent Res 1970;49:1505-8.
79. Sládková M. Prispevek k dedicnost hypodoncie. Sborn Lék 1982;84:355-7.
80. Jacobs SG. Reducing the incidence of palatally impacted maxillary canines by extraction of deciduous canines: a useful preventive/interceptive orthodontic procedure. Case reports. Aust Dent J 1992;37:6-11.
81. Lindauer SJ, Rubenstein LK, Hang WM, Andersen WC, Isaacson RJ. Canine impaction identified early with panoramic radiographs. J Am Dent Assoc 1992;123:91-7.
82. Peck S, Peck L. Classification of maxillary tooth transpositions. Am J Orthod Dentofac Orthop 1994(in press).