Site-specificity of tooth agenesis in subjects with maxillary canine malpositions

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

Tooth agenesis (hypodontia) is one of the most commonly observed anomalies of the human dentition. Three types of permanent teeth account for over 95% of cases of tooth agenesis: third molars (M3), second premolars (P2), and lateral incisors (I2).[1,2] Among Europeans and others of European ancestry, reports of tooth agenesis in these three tooth categories show a distinct pattern of site predilection. The frequency ranking of absent-tooth sites in European individuals is as follows[1-4]: M3 > P2 > I2.

An interesting relationship is becoming apparent between the occurrence of tooth agenesis and the presence of certain malpositions of the maxillary canine. Studies indicate significantly elevated prevalence rates for tooth agenesis in association with maxillary canine - first premolar transposition (Mx.C.P1)[5] and with palatal displacement of the canine (PDC).[6-9] Mx.C.P1 transposition is an orthodontic problem involving the positional interchange of maxillary canine with first premolar, usually due to distal ectopic development of the canine. PDC is characterized by developmental dislocation of the maxillary canine to a palatal site, often resulting in tooth impaction requiring surgical and orthodontic treatments. These three related abnormalities (tooth agenesis, Mx.C.P1, PDC) appear to have genetic determinants.[5-11]

This study examines the specific site occurrences of tooth agenesis in individuals with either Mx.C.P1 transposition or the PDC anomaly.

Abstract

Tooth agenesis (hypodontia) was studied in two samples of nonsyndromic subjects possessing either maxillary canine-first premolar transposition (Mx.C.P1; N=43, M9:F34) or palatal displacement of the maxillary canine (PDC; N=58, M21:F37). Agenesis of permanent teeth was identified by x-ray film analysis. Significantly elevated tooth-agenesis frequencies were noted in both samples. Statistically significant differences between the Mx.C.P1 and PDC samples were found in locations of absent teeth, indicating site-specificity of tooth agenesis associated with these canine malpositions. In Mx.C.P1, agenesis of third molars (M3) occurred at a near-normal rate (19%) while maxillary lateral incisor (I2) agenesis showed a thirteen-fold increase (26%). In PDC, the prevalence rate for associated M3 agenesis was 40%, twice the normal rate, while I2 agenesis was 3%, a slight elevation of no statistical significance. These new findings may warrant a hypothesis of anteroposterior site-specific shift in the occurrence of tooth agenesis, associated genetically or epigenetically with distinct anomalies of maxillary canine position and possibly other abnormalities.

Key Words

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

Submitted: May 1996 **Revised and accepted:** August 1996 Angle Orthod 1996;66(6):473-476.

It aims to further an understanding of the biological relationships among these dental disturbances of orthodontic consequence.

Materials and methods

Two samples were studied of nonsyndromic subjects possessing certain positional anomalies of one or both maxillary canine teeth: maxillary canine-first premolar transposition (Mx.C.P1; N=43, M9:F34) and palatal displacement of the maxillary canine (PDC; N=58, M21:F37). All subjects were from northeastern USA, except for 20 in the Mx.C.P1 sample who were from France and one from Finland. In the Mx.C.P1 sample, 40 of the subjects identified socially as white and three as black. The PDC group consisted of 58 white subjects. The mean age of the subjects for the Mx.C.P1 sample was 12.8 years and for the PDC sample, it was 14.2 years. Both samples are further described in other publications.[5,9]

Panoral x-ray films taken at clinical diagnosis of Mx.C.P1 and PDC were used to identify agenesis of permanent teeth including third molars. Any subject under age 14 years at diagnosis and having x-ray findings of one or more absent third molars was reevaluated using later panoral roentgenograms taken at 14 years or older, since 14 years seems to be the "critical age" regarding third molar formation.[10,12]

Results

Table 1 shows tooth agenesis frequencies for the Mx.C.P1 and PDC samples compared with reference values for the prevalence of tooth agenesis.[1,4] Statistical testing of differences was performed using the Chi-square test (χ^2). Strengths of associations were determined using the odds ratio assessment (O.R.; 95% confidence interval).

Agenesis of one or more teeth, including M3, occurred in 44% of the Mx.C.P1 sample and 48% of the PDC sample, of no significant difference in frequency statistically, but significantly elevated statistically when each was compared with the normal prevalence rate of 25% for hypodontia in á European population.

For each of the two samples, highly significant increases were observed generally in the prevalence of tooth agenesis compared with prevalence norms recorded in four other epidemiological categories: (1) tooth agenesis, excluding M3; (2) M3 agenesis; (3) mandibular P2 agenesis; (4) maxillary I2 agenesis. Conspicuously, two of these comparisons showed only slight differences that were not statistically significant: M3 agenesis in the Mx.C.P1 sample (χ^2 test= not significant; O.R.= 0.88) and maxillary I2 agenesis in the PDC sample (χ^2 test= not significant; O.R.= 1.84), as seen in Table 1. In Mx.C.P1, agenesis of M3 occurred at a near-normal rate (19%) while I2 agenesis showed a thirteen-fold increase (26%). In PDC, the prevalance rate for M3 agenesis was 40%, twice the normal rate, while I2 agenesis was 3%, a slight elevation of no statistical significance.

The odds ratio is a proportion of probabilities that in this study determined if the proportion of persons with tooth agenesis in the presence of PDC or Mx.C.P1 is greater than the proportion of persons with tooth agenesis in a normal population. The arithmetic value of the odds ratio is not influenced by sample size and, as such, it can function as a reliable indicator of clinical significance of trait associations, particularly of conditions occurring at low prevalence, such as PDC, Mx.C.P1 and tooth agenesis. Generally, an O.R.<2.0 indicates a questionable association between two variables (in this case, tooth agenesis and canine malposition) and an O.R.>2.0 represents two variables with a strong association. Therefore, the odds ratio assessment in this study added statistical evidence suggesting the lack of clinical association between occurrences of M3 agenesis and Mx.C.P1 and between occurrences of maxillary I2 agenesis and PDC. Conversely, the odds ratio values for the remaining eight agenesis:canine-malposition combinations displayed in Table 1 were all greater than 2.0, indicating the likelihood of strong associations clinically.

Discussion

Statistically significant differences between Mx.C.P1 and PDC samples in site-specificity of associated tooth agenesis are unexplained, important findings. The observed site-specific differential occurrence of tooth agenesis may provide a useful factor in the study of phenotypic variability in multiple dental anomaly patterns. PDC seems to be associated with suppression of tooth formation in the posterior (molar) odontogenic field and Mx.C.P1 seems associated with suppression of tooth formation in the anterior (incisor) odontogenic field. These new findings may warrant a hypothesis of anteroposterior site-specific shift in the occurrence of tooth agenesis, associated genetically or epigenetically with distinct anomalies of maxillary canine position and possibly other abnormalities. Since tooth agenesis is found sometimes occurring concomitantly with other discrete dental abnormalities, such as hyperdontia,[13,14] infraocclusion of deciduous molars,[15,16] ectopic eruption of permanent first

Table 1
Frequency of tooth agenesis in subjects with maxillary canine malpositions, compared with normal prevalence values for tooth agenesis.

Condition	Mx.C.P1 (N=43)				PDC (N=58)				Normal
	No. cases	Percent	O.R.	95% C.I.	No. cases	Percent	O.R.	95% C.I.	Prevalence
Tooth agenesis, including M3	19 **	44%	2.32	(1.28 - 4.20)	28 ****	48%	2.74	(1.65 - 4.54)	524/2061 = 25% Bredy,[1] 1991
Tooth agenesis, excluding M3	16 ****	37%	11.3	(6.49 - 19.7)	10 ****	17%	3.97	(2.00 - 7.88)	53/1064 = 5% Grahnén,[4] 1956
M3 agenesis (1 or more)	8 n.s.	19%	0.88	(0.40 - 1.90)	23 ***	40%	2.52	(1.50 - 4.23)	427/2061 = 21% Bredy,[1] 1991
Mandibular P2 agenesis (1 or 2)	5 ***	12%	5.47	(2.21 - 13.5)	8 ****	14%	6.65	(3.18 - 13.9)	25/1064 = 2% Grahnén,[4] 1956
Maxillary I2 agenesis (1 or 2)	11 ****	26%	17.8	(10.6 - 29.9)	2 n.s.	3%	1.84	(0.45 - 7.49)	18/1064 = 2% Grahnén,[4] 1956

χ^2 test: n.s. not significant; **P<0.01; ***P<0.001; ****P<0.0001
Note: O.R. = odds ratio
95% C.I. = 95% confidence interval of O.R.

molars[15,17] and I2 peg-shape anomaly,[18] site-specificity of hypodontia may be an important factor in future studies of the biological mechanisms underlying those associations also. Current evidence seems to implicate a heterogeneity of genetic controls behind the expression of associated dental anomalies.

The findings of significant variability in occurrence of M3 agenesis associated with canine malpositions should encourage more investigation of M3 presence and absence. Most earlier hypodontia studies have excluded third molars because of the difficulty of assembling a properly aged sample for recording reliable M3 epidemiological data.

The results of this study should condition clinicians to expect common occurrence of some unusual dental problems in the PDC or Mx.C.P1 patient. In PDC cases, the doubled chance of agenesis of one or more third molars would likely necessitate frequent consideration of therapeutic extraction of other third molars to balance the dental occlusion. The absence of one or both maxillary lateral incisors in over one-quarter of Mx.C.P1 cases would lead sometimes to an extraordinary, but clinically workable, orthodontic treatment plan of positioning a maxillary first premolar next to a central incisor.[19]

Author Address

Dr. Sheldon Peck
1615 Beacon Street
Newton, MA 02168 USA

Sheldon Peck, DDS, MScD, Department of Orthodontics, Harvard School of Dental Medicine, Boston, Mass., USA.

Leena Peck, DMD, MSD, Department of Orthodontics, Harvard School of Dental Medicine, Boston, Mass., USA.

Matti Kataja, PhD, National Public Health Institute, Helsinki, Finland.

References

1. Bredy E, Erbring C, Hübenthal B. Häufigkeit der Zahnunterzahl bei Anlage und Nichtanlage von Weisheitszähnen. Dtsch Zahn-Mund-Kieferheilkd 1991;79:357-63.
2. Lynham A. Panoramic radiographic survey of hypodontia in Australian Defence Force recruits. Austral Dent J 1989;35:19-22.
3. Haavikko K. Hypodontia of permanent teeth. An orthopantomographic study. Suom Hammaslääk Toim 1971;67:219-25.
4. Grahnén H. Hypodontia in the permanent dentition. Odontol Revy 1956;7(Suppl 3):1-100.
5. Peck L, Peck S, Attia Y. Maxillary canine - first premolar transposition, associated dental anomalies and genetic basis. Angle Orthod 1993;63:99-109.
6. Racek J, Sottner L. Príspevek k dedicnosti retence spicáku. Cesk Stomat 1977;77:209-13.
7. Svinhufvud E, Myllärniemi S, Norio R. Dominant inheritance of tooth malpositions and their association to hypodontia. Clin Genet 1988;34:373-81.
8. Peck S, Peck L, Kataja M. The palatally displaced canine as a dental anomaly of genetic origin. Angle Orthod 1994;64:249-56.
9. Peck S, Peck L, Kataja M. Prevalence of tooth agenesis and peg-shaped maxillary lateral incisor associated with palatally displaced canine (PDC) anomaly. Am J Orthod Dentofac Orthop 1996;110:441-3.
10. Garn SM. Genetics of dental development. In: McNamara JA Jr, ed. The biology of occlusal development. Craniofacial Growth Series. Monograph 7. Ann Arbor: Center for Human Growth and Development, University of Michigan. 1977:61-88.
11. Vastardis H, Karimbux N, Guthua SW, Seidman JG, Seidman CE. A human MSX1 homeodomain missense mutation causes selective tooth agenesis. Nature Genet 1996;13:417-21.
12. Garn SM, Lewis AB. The relationship between third molar agenesis and reduction in tooth number. Angle Orthod 1962;32:14-8.
13. O'Dowling IB. Hypo-hyperdontia in an Irish population. J Irish Dent Assoc 1989;35:114-7.
14. Spyropoulos ND, Patsakas AJ, Angelopoulos AP. Simultaneous presence of partial anodontia and supernumerary teeth. Oral Surg Oral Med Oral Pathol 1979;48:53-6.
15. Bjerklin K, Kurol J, Valentin J. Ectopic eruption of maxillary first permanent molars and association with other tooth and developmental disturbances. Europ J Orthod 1992;14:369-75.
16. Baccetti T, Tollaro I. Infraocclusion of primary molars as a dental anomaly of genetic origin. In: Radlanski RJ, Renz H, eds. Proceedings of the 10th international symposium on dental morphology. Berlin: "M" Marketing-Services, 1995:428-32.
17. Hoffmeister H. Die unterminierende Resorption der zweiten Milchmolaren durch die 6-Jahr-Molaren als Mikrosymptom der vererbten Störanfälligkeit der Gebissbildung. Schweiz Monatschr Zahnmed 1985;95:151-4.
18. Le Bot P, 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 Anthrop 1977;46:231-44.
19. Nestel E, Walsh JS. Substitution of a transposed premolar for a congenitally absent lateral incisor. Am J Orthod Dentofac Orthop 1988;93:395-9.