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Maxillary canine anomalies and tooth agenesis

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SUMMARY The aims of the study were to analyse the records of 26 subjects (18 females, eight males) with maxillary canine–first premolar transposition (Mx.C.P1) together with 160 subjects with a palatally displaced canine (PDC) to determine the pattern of tooth agenesis in these cases and to compare them with similar samples reported in the literature.

A strong association between Mx.C.P1, lateral incisor and lower second premolar agenesis was found, with a 20 per cent prevalence of lateral incisor agenesis and a 24 per cent prevalence of lower second premolar agenesis. There was a lesser association with third molar (M.3) agenesis, with a prevalence of 52.2 per cent. Weaker associations were found for a PDC, with a prevalence of 5 per cent for lateral incisor agenesis. The prevalence of lower second premolar (5 per cent) and M.3 (27.5 per cent) agenesis approached reference values. Evidence for the implication of the MSX1 or PAX9 genes in the aetiology of PDC was weak.

Introduction

Tooth transposition is defined as a form of ectopic eruption where a permanent tooth develops and erupts in the position normally occupied by another permanent tooth (Shapira *et al.*, 1989). The commonest form of transposition is between the maxillary canine–first premolar (Mx.C.P1). Tooth transpositions are rare. The prevalence of this particular anomaly is between 0.135 and 0.51 per cent and varies according to the race and region studied (Ruprecht *et al.*, 1984; Chattopadhyay and Srinivas, 1996; Burnett, 1999).

Several authors (Joshi and Bhatt, 1971; Peck *et al.*, 1993; Chattopadhyay and Srinivas, 1996; Plunkett *et al.*, 1998; Shapira *et al.*, 2000; Shapira and Kuftinec, 2001) have studied transposed teeth in an attempt to shed light on the aetiology of the condition. The weight of evidence is that canine transposition is a disturbance of eruption under a measure of genetic control (Feichtinger *et al*., 1977; Peck *et al.*, 1993, 1997, 2002; Shapira *et al*., 2000). Other theories proposed are an interchange in the position at the anlage stage of the involved teeth during odontogenesis (Joshi and Bhatt, 1971; Mader and Konzelman, 1979; Laptook and Silling, 1983) and trauma (Dayal *et al*., 1983; Shah, 1994).

A palatally displaced canine (PDC) is a more common developmental disorder with a prevalence of 0.8–2.8 per cent (Shah *et al*., 1978; Grover and Lorton, 1985). This, too, may have a genetic aetiology (Zilberman *et al*., 1990; Peck *et al*., 1994). Inheritance on an autosomal dominant basis has been proposed (Pirinen *et al*., 1996).

Both Mx.C.P1 and PDC are associated with hypodontia (Svinhufvud *et al*., 1988; Peck *et al*., 1993), another autosomal dominant condition. Hypodontia may be caused by one major gene mutation, but is very often heterogenic (Arte, 2001).

Peck *et al.* (1994) pointed out similarities between PDC and Mx.C.P1 and argued that both conditions are genetic in origin and frequently occur in association with other, genetically interrelated, dental anomalies.

Peck *et al.* (2002) analysed the pattern of hypodontia associated with PDC, Mx.C.P1 and other variations of canine transposition. Third molar (M.3) agenesis was found to be strongly associated with mandibular incisor–canine transposition (Mn.I2.C) and PDC. Mx.C.P1 was associated with lateral incisor agenesis, but not with M.3 agenesis. In view of this, the homeobox genes MSX1 and PAX9, associated with posterior field (molar) hypodontia, have been suggested by Peck *et al.* (2002) as candidate genes for the control of Mn.I2.C and PDC.

The Maltese population has a high prevalence of PDC, over 4 per cent (Camilleri, 1995). The most likely genetic explanation for this is the founder effect, the local population having grown rapidly from less than 20 000 to over 350 000 in the past 500 years (Cassar, 2000). This high prevalence causes a considerable drain on the resources of the School Dental Service. Seventeen per cent of the consultant caseload involves dealing with ectopic canines. Consequently, the aetiology of PDC is of considerable interest.

The aim of this study was to analyse a large sample of subjects with Mx.C.P1 and PDC and to compare the prevalences and patterns of tooth agenesis in these groups with similar samples reported in the literature.

Subjects and method

One hundred and sixty non-syndromic consecutive subjects with PDC were ascertained from the files of the School Dental Clinic, Floriana, and from private practice over the past 3 years. Diagnosis was made by both clinical and radiographic examination and confirmed at the time

of surgery, where appropriate. Patients under 13 years of age were eliminated from the M.3 study. All subjects were Caucasian and resident in the Maltese Islands.

Twenty-six non-syndromic subjects with Mx.C.P1 transposition were gathered from private practice over the past 10 years and from the files of the School Dental Clinic over the last 3 years. A general dental practitioner contributed study models of one further case. Transposition was confirmed clinically or by radiographic or photographic evidence for all cases except one, which was documented with study models only and used solely for assessment of lateral incisor agenesis. Two Mx.C.P1 subjects were under 13 years of age and therefore were excluded from the M.3 assessment, as was the study model case.

Fisher's exact test was used to compare the frequencies of agenesis of specific teeth with earlier published population prevalences (Grahnén, 1956; Bot and Salmon, 1977; Bredy *et al*., 1991). The significance level was set at $P < 0.05$.

Data were also obtained from previously published articles on PDC and Mx.C.P1 transposition. The ratios of tooth agenesis were compared with each other and with the present sample, using chi-squared tests.

Results

Table 1 shows the data for the Mx.C.P1 subjects. The sample comprised 18 females and eight males. Various other anomalies were observed to occur in these subjects, apart from the transposed canines and hypodontia. Six patients presented with ectopic contralateral canines. Of these, five were palatally displaced (Figure 1) and one was buccally impacted. In one subject, the transposed canine had erupted palatally (Figure 2). Other anomalies were submerged primary teeth, ectopically erupting premolars and supernumerary teeth (Table 1). In one patient, a dental panoramic tomogram (DPT) had been taken at 10 years of age, prior to the diagnosis of the transposition. The second DPT was taken at 15 years of age (Figure 3). In another case, the transposition was found to have corrected spontaneously after extraction of the adjacent submerged primary teeth. Following this, the contralateral canine erupted palatally (Figure 4).

Table 2 shows the tooth agenesis frequencies for the PDC and Mx.C.P1 samples for the three studied tooth types, M.3, mandibular second premolar (Mn.P.2), maxillary lateral incisor (Mx.I.2). The number of affected subjects in each category is reported along with the computed relative frequency of agenesis.

Discussion

Mx.C.P1 transposition seems to be strongly associated with incisor agenesis but less so with premolar and M.3 agenesis.

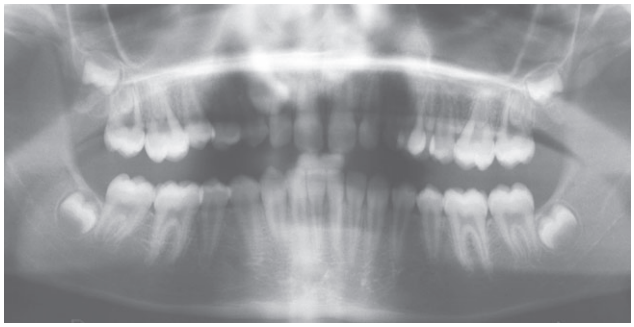

Figure 1 Case 12: transposed right maxillary canine with palatally displaced left maxillary canine.

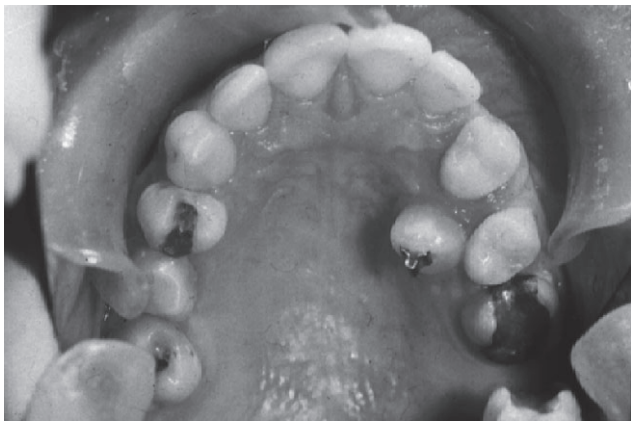

Figure 2 Case 13: transposed maxillary canine erupted palatally. The bracket was bonded post-eruption in an attempt to bring it into the arch.

Comparison of the data reported previously on Mx.C.P1 (Table 3) with the present sample showed remarkable similarity. There was no significant difference between the figures for lateral incisor, premolar and M.3 agenesis ($P = 0.1114$, $P = 0.1977$, $P = 0.0953$, respectively). The prevalence ratios quoted were quite similar in most cases.

The ratios for incisor and premolar agenesis were remarkably consistent for all Mx.C.P1 transposition samples throughout the world.

There was no significant difference between the studies of Plunkett *et al.* (1998) and Shapira and Kuftinec. (2001) for maxillary canine impaction ($P = 0.0636$). The prevalences of canine impaction reported in those studies (1.9 and 2.7 per cent) were well within the quoted range. However, in the present study, the prevalence was markedly higher at 20.8 per cent ($P = 0.0041$). This is an unusual finding, as PDC has not been reported as being associated with Mx.C.P1. Plunkett *et al.* (1998) and Shapira and Kuftinec (2001) documented one case each, while Peck *et al.* (2002) reported no such case in their sample or in a search of the literature.

Table 1 The subject data, recording the position and number of transposed teeth together with associated dental anomalies. Subjects under 13 years of age at the time of the study are listed as '<13 years'.

Case no.	Gender	Teeth (FDI system)												
		13	23	12	22	15	25	35	45	18	28	38	48	Anomalies
1	m		tr											23 palatal
2	f	tr	tr		ab								ab	
3	f	tr	tr							ab				None
4	f	tr	tr							ab		ab	ab	None
5	f	tr	tr	ab	ab									
6	f	tr	tr			ab	ab			?	?	?	?	Midline supernumerary, 45 impacted; <13 years
7	m	tr	tr	ab				?	?	?	?	?	?	Only study models available
8	f	tr						ab	ab	ab	ab	ab		23 impacted buccally
9	f	tr		ab	ab					ab	ab	ab	ab	23 palatal
10	f		tr								ab			13 palatal
11	f		tr						ab			ab		13 palatal and 74 and 75 ankylosed
12	f		tr											13 palatal
13	f	tr								ab	ab	ab		35 impacted, 13 erupted palatally
14	m	tr												None
15	m	tr												None
16	m	tr												None
17	f	tr												None
18	f		tr	ab	ab									15 and 25 ectopic
19	m		tr											22 diminutive
20	m	tr					ab	ab	ab					55, 65 and 76 ankylosed
21	m		tr											None
22	f	tr							ab	ab	ab	ab	ab	None
23	f		tr							ab		ab	ab	None
24	f		tr							ab	ab	ab	ab	None
25	f		tr						ab			ab	ab	45 ectopic, <13 years
26	f		tr											25 ectopic

ab, absent; tr, transposed; ?, presence undetermined.

There were highly significant differences between all samples and the reference values for Mx.I.2 agenesis (Table 4). The same applied for Mn.P.2 agenesis, apart from the findings of Shapira and Kuftinec (2001), where there was no significant difference from the reference value.

The figures for the Maltese Mx.C.P1 group were elevated when compared with the published prevalences for lateral incisor, premolar and M.3 hypodontia ($P = 0.0006$, $P = 0.0002$, $P = 0.0234$, respectively).

The PDC samples (Table 5) also showed no significant differences between each other, although the level of agreement was low for both Mx.I.2 and Mn.P.2 agenesis. The ratios for Mx.I.2 agenesis were also consistent, except for the figure given by Mossey *et al.* (1994). This may be due to ethnic or sampling differences.

There was no significant difference in M.3 agenesis between the Maltese PDC sample and the published population prevalences (Table 6). The figure for lateral incisor agenesis for the control sample was significantly different from the PDC sample ($P = 0.0324$), but not for premolar hypodontia ($P = 0.1246$). A comparison of previous PDC data with the reference values showed that PDC in this sample was also associated with incisor agenesis, while the association with premolar agenesis was variable.

Separation of the Maltese PDC sample into unilateral and bilateral subsamples and comparison of these samples gave interesting results (Table 7). The ratio of tooth agenesis in the bilateral sample was much higher than in the unilateral sample. This is not surprising, as one would expect the bilateral PDC subjects to have more complex problems. However, the ratios of Mn.P.2 and M.3 agenesis in the unilateral sample approached the reference values.

The data do not support the theory that PDC is specifically associated with M.3 agenesis. Rather, it points to an incisor/premolar/M.3 agenesis gradient, more pronounced in bilateral than in unilateral PDC cases, and even stronger in Mx.C.P1 cases. This supports previous work (Bjerklin *et al*., 1992; Pirinen *et al*., 1996; Baccetti, 1998; Arte *et al*., 2001) where a relationship was found between incisor/premolar hypodontia and PDC.

MSX1 mutations have been associated with facial clefting (Lidral *et al*., 1998; van den Boogaard *et al*., 2000) and also premolar/M.3 agenesis (Vastardis *et al*., 1996). However, no association has been shown with MSX1 mutations and

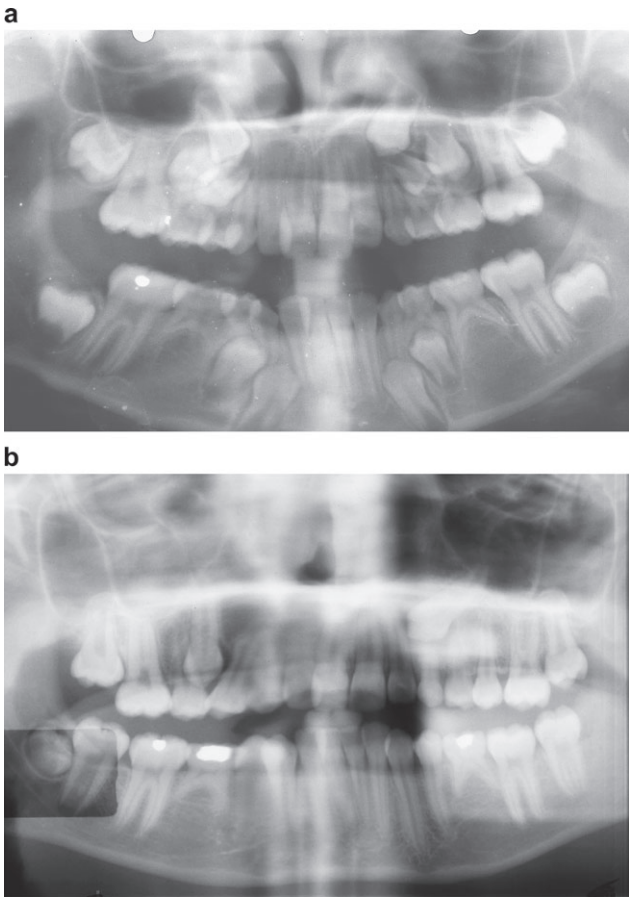

Figure 3 Case 8: (a) dental panoramic tomogram (DPT) taken at 10 years of age. The apex of the upper right canine is much further distal than normal, over the bud of the developing second premolar. Both first premolars are markedly distally inclined. (b) DPT taken at 15 years of age. The upper right canine has erupted into a transposed position, buccal to the first and second premolars. The upper left canine is impacted high and buccal to the arch.

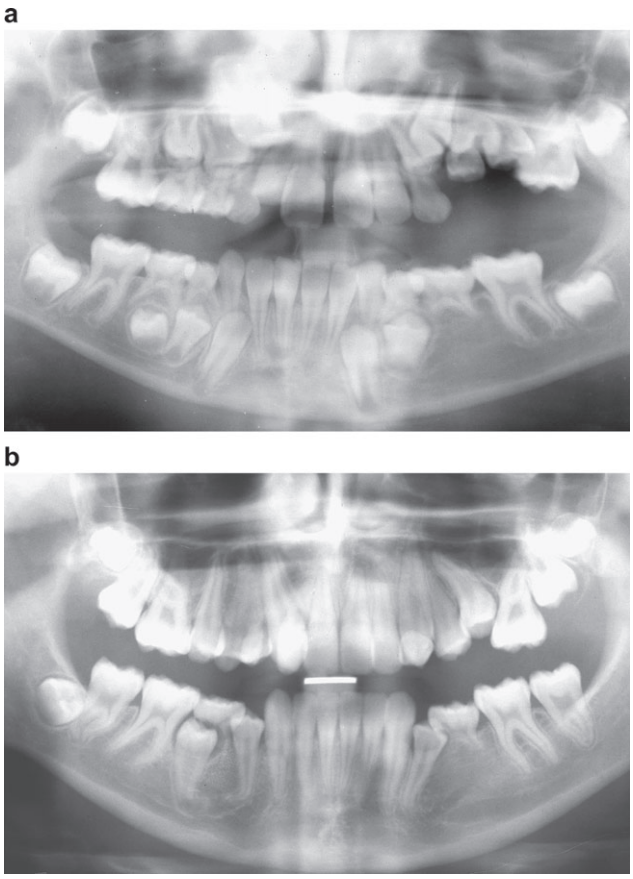

Figure 4 Case 11: (a) the apex of the upper left canine seems to be in the correct position, over the root of the first primary molar. The first and second premolars seem to be ectopic, being mesial to their normal positions. (b) Following extraction of the submerging primary molars, the teeth erupted in the correct sequence, with the upper left canine buccal to the retained primary canine crown, and the second premolar palatal to the line of the arch. Had the primary molars not been extracted, transposition would probably have occurred, due to ectopic eruption of the premolar. The upper right canine had spontaneously erupted palatally.

incisor/premolar hypodontia (Lidral and Reising, 2002). Lidral and Reising (2002) and Arte *et al.* (2001) are of the opinion that, as one of the features of MSX1 hypodontia is multiple missing teeth, MSX1 and PAX9 mutations are unlikely to be the cause of incisor/premolar hypodontia, where one or two teeth are usually missing.

The evidence for an association of PDC with posterior orofacial genetic fields, as proposed by Peck *et al.* (2002), seems weak. It seems unlikely that MSX1 or PAX9 would be candidate genes in the aetiology of PDC.

In all Mx.C.P1 studies where gender was reported (Shapira, 1980; Peck *et al.*, 1993; Plunkett *et al.*, 1998) a gender bias was evident, with the proportion of girls ranging from 60 to 80 per cent. The present sample showed a similarly high proportion of females (69 per cent). This finding cannot simply be explained by the increased ratio of females over males seeking orthodontic treatment and supports the view of Peck *et al.* (1993) that a degree of gender linkage seems to be present.

It is controversial whether the anlages develop in the transposed position or whether the tooth buds develop correctly and subsequently migrate to ectopic positions. The maxillary permanent canine starts to calcify at 1.5 years of age, between the roots of the first primary molar. As the jaws grow, the canine moves apically, away from the first primary molar. The first premolar then develops in the same site as the canine (Broadbent, 1941). As the jaws grow rapidly in depth and width, the teeth move to maintain their correct relationship to each other. As the maxilla grows, the first premolar moves distally relative to the canine, providing space for the canine to erupt. This involves precise co-ordination of the movement of the tooth germs in the growing maxilla. This movement is probably effected by osteoblast–osteoclast interaction, controlled by the dental follicle, as part of the eruption process. A deficiency in the cell signalling process of one tooth or more adjacent teeth, at an early stage, could well cause the tooth buds to move in the wrong direction

Table 2 Agenesis according to tooth type in maxillary canine–first premolar (Mx.C.P1) transposition and palatally displaced canine (PDC) cases. The data are compared with reference values.

Canine tooth malposition	Missing maxillary lateral incisors			Missing mandibular second premolars			Missing third molars		
	No. of cases	%	*P*	No. of cases	%	*P*	No. of cases	%	*P*
PDC (*n* = 160)	8	5.0	0.0324	8	5.0	0.1246	44	27.5	0.4409
Mx.C.P1 (*n* =26*)	5	20.0	0.0006	6	24.0	0.0002	12	52.2	0.0234
Reference	Bot and Salmon (1977)			Grahnén (1956)			Bredy *et al.* (1991)		
Normal prevalence value	109/5738 = 1.9%			25/1064 = 2.3%			427/1061 = 20.7%		
				**n* adjusted for missing data			**n* adjusted for missing data		

Table 3 Comparison of data for tooth agenesis and ectopic maxillary canines in different published samples of maxillary canine–first premolar transposition.

Study	Missing maxillary lateral incisors			Missing mandibular second premolars			Missing third molars			Ectopic maxillary canines		
	Missing	Total	%	Missing	Total	%	Missing	Total	%	Ectopic	Total	%
Peck *et al.* (1993)	12	43	27.9	6	43	14.0						
Plunkett *et al.* (1998)	6	33	18.2	4	33	12.1				1	52	1.9
Shapira and Kuftinec (2001)	9	36	25.0	1	36	2.8				1	36	2.8
Peck *et al.* (2002)				5	43	11.6	8	43	18.6			
Present study	5	26	19.2	6	25	24.0	12	22	54.5	6	26	23.1
χ^2 statistic	7.5068			6.0188		2.7823				10.9850		
P	0.1114			0.1977		0.0953				0.0041		

Table 4 Proportion of congenitally missing teeth in different published samples of maxillary canine–first premolar transposition as compared with reference data.

Study	*n*	Missing maxillary lateral incisors			Missing mandibular second premolars			Missing third molars		
		No.	%	*P*	No.	%	*P*	No.	%	*P*
Peck *et al.* (1993)	43	12	27.9	<0.0001	6	14.0	0.0030			
Plunkett *et al.* (1998)	33	6	18.2	0.0002	4	12.1	0.0260			
Peck *et al.* (2002)	43	11	25.6	<0.0001	5	11.6	1.0000	8	18.6	0.9608
Shapira and Kuftinec (2001)	36	9	25.0	<0.0001	1	2.8	0.0139			
Present study	26*	5	25.0	0.0006	6	23.1	0.0002	12	54.5	0.0234
Reference		Bot and Salmon (1977)			Grahnén (1956)			Bredy *et al.* (1991)		
Normal prevalence value		109/5738 = 1.9%			25/1064 = 2.3%			427/1061 = 20.7%		
					**n* adjusted for missing data			**n* adjusted for missing data		

(or fail to move), leading to transposition of the tooth germ.

The genes identified with the failure of eruption in experimental animals and humans are those associated with osteoclast/osteoblast function, such as cleidocranial dysostosis and osteopetrosis (Walker, 1975; Cooper *et al.*, 2001), although tooth agenesis is not usually a feature.

It is possible that the gene or genes responsible for both PDC and Mx.C.P1 are those involved with the control of tooth eruption. These in turn seem to be linked with the gene or genes causing incisor/premolar hypodontia. The heterogenic nature of tooth agenesis has made it difficult to identify the culpable genes (Arte, 2001).

Conclusion

The weight of available evidence points to a genetic association between PDC and hypodontia. This association is even more marked in the case of Mx.C.P1. Family studies to establish the mode of heredity and the prevalence

Table 5 Comparison of data for congenitally missing teeth in palatally displaced canine cases.

Study	Missing maxillary lateral incisors			Missing mandibular second premolars			Missing third molars		
	Missing	Total	%	Missing	Total	%	Missing	Total	%
Oliver *et al.* (1989)	2	60	3.3						
Bjerklin *et al.* (1992)				5	91	5.5			
Mossey *et al.* (1994)	23	182	12.6						
Peck *et al.* (2002)	2	58	3.4	8	58	13.8	23	58	39.7
Chaushu *et al.* (2002)	4	58	6.9						
Present study	8	160	5.0	8	160	5.0	44	160	27.5
χ^2 statistic	7.5068			4.6106			2.7823		
P	0.1114			0.0998			0.0953		

Table 6 Proportion of congenitally missing teeth in different published samples of palatally displaced canines as compared with reference data.

Study	*n*	Missing maxillary lateral incisors			Missing mandibular second premolars			Missing third molars		
		No.	%	*P*	No.	%	*P*	No.	%	*P*
Oliver *et al.* (1989)	60	5	8.3	0.0161						
Bjerklin *et al.* (1992)	91				5	5.5	0.1769			
Mossey *et al.* (1994)	182	23	12.6	>0.0001						
Chaushu *et al.* (2002)	58	4	6.9	0.3842						
Peck *et al.* (2002)	58	2	3.4	0.6233	8	13.8	0.0007	23	39.7	0.0188
Present study	160	8	5.0	0.0324	8	5.0	0.1246	44	27.5	0.4409
Reference		Bot and Salmon (1977)			Grahnén (1956)			Bredy *et al.* (1991)		
Normal prevalence value		109/5738 = 1.9%			25/1064 = 2.3%			427/1061 = 20.7%		

Table 7 Comparison of congenitally missing teeth in unilateral and bilateral palatally displaced canine cases in the present study with reference values.

	n	Missing maxillary lateral incisors			Missing mandibular second premolars			Missing third molars		
		No.	%	*P*	No.	%	*P*	No	%	*P*
Bilateral	52	3	5.8	0.1717	3	5.8	0.2953	20	38.5	0.0369
Unilateral	108	4	3.7	0.3253	3	2.8	0.9756	27	25.0	0.8804
Reference		Bot and Salmon (1977)			Grahnén (1956)			Bredy *et al.* (1991)		
Normal prevalence value		109/5738 = 1.9%			25/1064 = 2.3%			427/1061 = 20.7%		

of other anomalies are indicated for the latter group. The difference in prevalence of hypodontia in cases of unilateral and bilateral PDC warrants further research.

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