Transposition of teeth and genetic etiology

Amit Chattopadhyay, BDS (Hons), DcFM, MSASMS, MDS;
K. Srinivas, BDS

Tooth transposition occurs when adjacent teeth switch positions, particularly at the roots, or when a tooth develops or erupts in a position normally occupied by a nonadjacent tooth.[1]

The phenomenon of transposition appears to be limited to cases involving a canine and a lateral incisor or premolar, and it occurs much more commonly in the maxilla than the mandible (unilateral/bilateral). Transposition of central and lateral incisors has also been described.[2-17]

Transposition is said to be complete when the two involved teeth have fully or almost fully exchanged places. It is incomplete when the positions of the teeth are not completely exchanged but show a tendency toward such exchange.[7] One needs to exercise caution when diagnosing transpositions to avoid confusing them with mere ectopic eruptions. In transposition, the teeth exchange places, thereby reversing their sequence in the mouth. Ectopic eruption is a broad category referring to any abnormal or aberrant eruptive position taken by a tooth. Transposition, therefore, must be considered a subdivision of ectopic eruption: All transpositions are examples of ectopic eruption, but few ectopic eruptions are transpositions.[1]

The etiology of transposition remains speculative. Various theories have been proposed to explain the phenomenon, including transposition of dental anlage during development, migration of a tooth during eruption, heredity, and trauma.[5,7,16]

Materials and methods

The sample for the present study consisted of 21 patients exhibiting true transposition of teeth. These patients were drawn from the 4933 patients visiting SDM College of Dental Sciences in Dharwad, India, between August and October, 1993. All the patients where Kannadigas (who lived originally in the state of Karnataka, India) and none were aware of the transpositions.

Information was gathered to differentiate be-

Abstract

Twenty-one cases of transposition are presented showing a crude prevalence of 0.4%. A high rate of bilateral occurrence was seen. A significant number of cases were associated with other dental anomalies, such as peg-shaped lateral incisors and overretained deciduous teeth. Genetic etiology has been stressed and various other theories discussed.

Key Words

Transposition • Genetic etiology

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Table 1
Features of transposition cases

No.	Age	Sex	Transposed teeth and other features	
1	28	M	13 and 12	Over-retained 53
2	25	F	23 and 22	Over-retained 63, missing 31, 41
3	26	M	23 and 24	Peg-shaped 12, 22
4	34	M	23 and 22	—
5*	26	F	13 and 12, 23 and 22	Missing 32, 42; space in normal canine area
6*	36	F	13 and 12, 23 and 22	Missing 24
7	50	M	23 and 24	Missing 22
8	40	M	13 and 12	Over-retained 53, missing 22
9**	25	M	33 and 32	Over-retained 73
10	16	F	23 and 22	Over-retained 63, 23 palatal
11	24	M	23 and 24	Peg-shaped 22; 23 buccally
12	35	F	13 and 12	Over-retained 53, missing 22
13	26	M	13 and 14	Over-retained 53, peg-shaped 12
14	26	M	13 and 12, 23 and 22	Over-retained 53, 63; missing 11, 21
15	19	M	13 and 12, 23 and 22	Over-retained 53, 63
16	22	M	23 and 22	Over-retained 53, 63, 73; missing 13, 14, 33
17	43	M	13 and 12	Over-retained 63, peg-shaped 22
18	36	M	23 and 22	Space distal to canine area
19	33	M	23 and 24	Over-retained 53, 63; peg-shaped 12, 22, 32, 42 in patient and mother
20	22	M	23 and 22	Space distal to canine area
21	26	M	23 and 24	—

*bilateral cases; **rare mandibular transposition

tween teeth missing due to extraction and those missing due to agenesis. The following data were recorded: Age at diagnosis, sex, race/ethnicity, location of transposition, and associated congenital dental anomalies with details. Panoramic and IOPA radiographs were taken at the examination to determine the presence of true or pseudo transpositions and congenitally missing or impacted teeth. A peg-shaped lateral incisor was defined as a severe, conical, crown-sized reduction of the tooth, and narrowing in diameter from the cervix to the incisal edge.[18]

Results

Table 1 summarizes the main features of the subjects with transposed teeth. Some of the subjects are shown in Figures 1 and 2. During the study period, transpositions were found in 21 of 4933 patients, giving a crude prevalence of 4.257/1000 or 0.4%. Transpositions were found more often in males than in females (15/6 or 2.5:1), compared with the total patient population (1.78:1). The age range of the patients with transpositions was from 16 to 50 years, with almost even distribution over the various decades.

For further discussion, only transpositions found in the maxilla are considered; the rare case of mandibular transposition is described separately. Maxillary teeth were involved in the transpositions in 20 subjects (4.054/1000). Four of these cases (20%) had bilateral transposition and 16 had unilateral involvement, of which 11 (69%) were on the left side. The canine was involved in all the cases. In 14 patients (70%) the canine and lateral incisor were involved, and 6 patients (30%) had canine-premolar involvement. Five patients (25%) exhibited peg-shaped lateral incisors. In 8 patients (40%) the transpositions were associated with missing teeth, and 10 patients (50%) had over-retained deciduous canines on the involved side.

Table 2 shows the comparison of the data after separating the transpositions into two groups: canine-lateral incisor, and canine-premolar. Both groups contained more males than females, and the left side was more frequently involved in unilateral cases. A significant number of cases of both groups exhibited other associated dental anomalies, such as an over-retained deciduous canine on the affected side, peg-shaped lateral incisors, or congenitally missing teeth.

Discussion

The transposition of teeth usually involves the canines, along with either incisors or premolars.[1,7,8,12,13,15,16] The transposition of a canine with a first premolar has been reported in both contemporary and prehistoric populations.[8-10] Similarly, the transposition of central and lateral incisors has also been reported.[4]

The present sample shows a high male predilection, which is distinctly different from earlier reports from India and elsewhere,[1,7] although similar to Shapira.[16] We believe that because most of the previous samples were from orthodontic clinics,[1,7,11,16] a high female count could be expected because more females than males seek orthodontic treatment. It is noteworthy, then, that the present study was carried out in a dental school and all the visiting patients were screened. We found a male predominance in the transposition sample (2.33:1) which was higher than the male/female ratio of the population visiting the institution (1.78:1). Many of the transposition cases reported in the literature have also been in males.[4-6,8,9,13,15,24] Unfortunately, sex data is missing from one of the earliest large series.[7] In a study of a prehistoric population,[10] there was

also a high male percentage (44.4%) Among published cases cited by Peck et al.,[1] there is a higher male predilection (22:17). If all the published cases are brought together, the vexing problem of sex predilection in the transposition of teeth can be solved.

Shapira found canine and first premolar transpositions to occur twice as often as canine and lateral incisor transpositions, a result similar to earlier studies.[7] However, our results show the opposite: Canine-lateral incisor transposition was much more frequent than canine-premolar transposition (14:6).

We found a high number of bilateral cases (20%), similar to Peck et al.,[1] but in sharp contrast to the very low number of bilateral cases seen by Joshi and Bhatt (7.5%).[7] Most of the transposition cases in a prehistoric population[10] were also bilateral. Interestingly, earlier studies[1,7,10,13,15] reported bilaterality only in canine-premolar transpositions, while all our bilateral cases involved a canine and a lateral incisor. Again, we have noted that in patients having bilateral transpositions, the same pair of teeth are involved on both sides (canine-lateral incisor, in our sample). No case has been reported where different pairs were transposed on either side.

Among the unilateral cases, we found a left-sided predominance (11:5). This corresponds to earlier results.[1,7] The preponderance of left-sided expression of transposition remains unexplained at present. However, laterality is not uncommon in expression of other orofacial anomalies. For example, left-sided predilection is twice that of right side in unilateral cleft lip patients.[1]

Table 2 compares the data for canine-lateral incisor and canine-premolar transposition cases in the maxilla in the present study. The breakdown was done to find out if there were any major differences in the patterns exhibited by the two types of transpositions. In both types, we found a male predominance, left-sided preponderance, significant association with an over-retained deciduous canine on the involved side, a higher-than-normal occurrence of peg-shaped lateral incisors, and a high number of congenitally missing teeth. However, peg-shaped laterals were more frequently associated with canine-premolar transposition than canine-lateral incisor transposition. Given these obvious similarities and going by the fact that the permanent canine tooth is involved in both transposition types, we have grouped the cases together to represent the phenomenon of transposition of teeth as a whole (i.e. true transposition). We consider transposition of canine-lateral incisor and canine-premolar to be the same basic phenomenon with different expressions (i.e., involving different teeth).

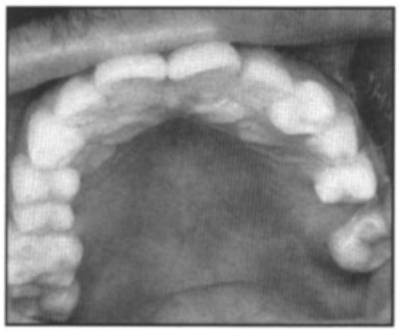

Figure 1

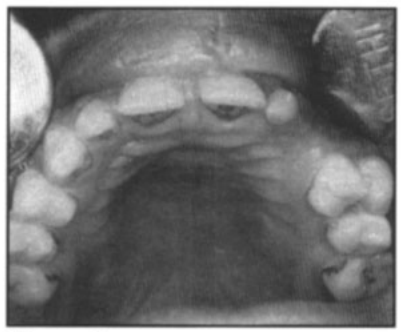

Figure 2

Table 2
Comparison of canine transposition with lateral incisor and premolar (maxillary)

	Canine-lateral incisor	Canine-premolar	Total
No. of cases	14	6	20
Male:Female	8:6 (1.33:1)	6:0	14:6 (2.33:1)
Frequency/1000	3.04/1000 (14/4933)	1.21/1000 (6/4933)	4.05/1000 (20/4933)
Bilateral	28.57% (4/14)	—	20% (4/20)
Left:Right	6:4 (1.5:1)	5:1 (5:1)	11:5 (2.2:1)
Peg-shaped lateral	7.1% (1/14)	67% (4/6)	25% (5/20)
Over-retained deciduous canine	64.3% (9/14)	33% (2/6)	55% (11/20)
Missing teeth	50% (7/14)	16.7% (1/6)	40% (8/20)

Male:Female ratio of the total population: 1.7:1

The etiology of transposition has been an area of controversy. Various theories, such as canine migration drift (due to obstruction) or dental anlage exchange have been proposed.[7,16] However, a genetic etiology has been strongly supported by Nelson[10] from his study of prehistoric material from Santa Cruz Island, Calif, and also by Peck et al.[1] from their study of a present-day sample of 43 canine-first premolar transpositions. The present study proposes to strengthen the argument for a genetic etiology.

All the subjects in the present study belong to the same race and were from the same geographical area (Karnataka, India). The crude prevalence of transposition, 0.4%, is much higher than the 0.16% prevalence seen in a Czechoslovakian population,[19] suggesting a definite racial difference.

Figure 1
Complete transposition of canine with first premolar: left side (case 21).

Figure 2
Incomplete transposition of canine with first premolar. Despite sufficient eruption space in its normal place, the canine erupted distally. Can space shortage/obstruction account for this? Note peg-shaped lateral incisor (case 11).

The prevalence of peg-shaped lateral incisors varies between 1% and 2% in the general population.[1,20] However, 25% of our transposition cases had associated peg-shaped lateral incisors, showing a massive increase in frequency. This is even higher than the 16% occurrence of peg-shaped lateral incisors associated with transposition that Peck et al.[1] reported.

Agenesis of teeth in the general population has a frequency of 3.5% to 8%.[1,21] Peck et al.[1] found a 37% tooth agenesis frequency in transposition cases and we found a 40% occurrence. Missing teeth have frequently been associated with transposition cases.[4,13,16] The high frequency of congenitally missing teeth, 4.5 to 10 times higher than normal, shows a higher correlation with transposition than in general population. Taken together, peg-shaped lateral incisors and missing teeth were seen in 11 out of 20 cases (55%, which is higher than the 49% figure quoted by Peck et al.[1]).

Several factors, taken together, point very strongly toward a gene-based etiology for transposition. These include:

- Racial differences in frequency of transposition
- The high frequency of associated dental abnormalities, such as peg-shaped laterals and congenitally missing teeth
- Frequent bilateral occurrence
- Involvement of the same teeth type in bilateral cases on both sides.

Furthermore, familial cases have been reported.[19,22] Feichtinger et al.[23] described a prehistoric American population that lived on a small island, had intermarried for generations, and had a high frequency of transpositions. These observations further strengthen the case for considering a genetic component in a preeminent position in the etiology of transposition.

Deciduous canines have often been found to be over-retained in transposition cases,[1,5,7,13,16] as also in 55% of our subjects. This has been proposed as the etiology of transposition.[7,8] Under this theory, the over-retained deciduous teeth obstruct the eruption path of the permanent canine, which therefore gets deflected; it then migrates and erupts in a transposed position (migration/deflection/drift theory). Peck et al.[1] clearly assert that an over-retained deciduous canine is a direct outcome of the failure of the permanent canine to erupt below it, and not the cause of the transposition. We considered all the reported cases and agreed that overretention is the effect rather than the cause of transposition.

Indeed, it is intriguing that transpositions are more common in the maxilla than in the mandible (where transmigrations are more common). Maxillary canines develop in a position high above the first premolars, and it has been suggested that they may change position during eruption. Thus, the canine may migrate too far distally and become transposed with the first premolar.[7,16] This sounds plausible, as the eruption times of the canine and premolars are close to each other, but the theory fails to explain transposition of the canine with the earlier-erupting lateral incisor. It is also intriguing that the canine should be able to exchange places with other teeth so precisely at different frequencies in different races, due to abnormally deflected movement. Furthermore, the migration theory cannot explain the high frequency of the associated features discussed above.

Often, in the absence of the adjacent lateral incisor, the canine gets enough space to erupt but still transposes. In our cases #18 and #20 we saw that despite adequate eruption space distal to the lateral incisor, the canine was transposed. The presence of a lateral incisor root with the right length formed at the right time has been implicated as an important variable; the root guides the erupting canine in a favorable distal and incisal direction.[25] Cases where laterals are present and enough space is available for the canine to erupt normally but the canine is still transposed with a premolar become difficult to explain by the migration theory because the transposition occurs despite all conditions being present for normal eruption of the canine.

Eruption patterns of teeth are generally constant. The usual sequence of permanent tooth eruption is first molar (at 6 years), central incisor (7 years), lateral incisors (8 to 9 years), first premolars (11 to 12 years), canine and second premolars (11 to 12 years), second molars (13 years), and third molars (18 years and up).[26] The lateral incisor precedes the canine by 2 to 3 years. If transposition of the canine with the lateral incisor is to be explained by migrational ectopic drift of the canine, then what condition or situation makes the earlier-erupting lateral incisor occupy the canine's position? Also, remember that the lateral incisor is a smaller tooth, with a smaller mesiodistal dimension than the canine. If the canine has to force its way between the central and lateral incisors, pushing the lateral distally, especially with an over-retained deciduous canine present distally (as in a large number of cases), it is bound to find very little space for precise eruption in the lateral incisor space. This would lead to impaction or palatal eruption of

the canine rather than transposition. This provides the antithesis for the migration-deflection theory of transposition. Furthermore, such drifted eruption, even if it occurs, would invariably leave the roots pointing along the trajectory taken by the canine. Such an occurrence will, by definition, rule out the case as a transposition. In transposition, the roots of the transposed canine are directed normally, but in the wrong place.

Nelson explained and measured canine root position and root curvature to find out differences, if any, between transposed canines and controls. He found the roots of the transposed canines to be of normal configuration but placed too far off (all canine-premolar transpositions) from nasal alare. He showed that the differential placement of normal and transposed roots is real, suggesting that the buds for the transposed teeth themselves originated in a transposed position and teeth proceeded to grow and erupt normally, except in the wrong place.[10] Such an occurrence can account for the precise exchange of places by adjacent teeth, as seen in transpositions.

An interesting proposition was made in 1926 by Widdowson,[27] based on Bolk's theory of the evolution of mammalian teeth. An ardent supporter of this theory, Widdowson stated that transpositions are "evidences in support of the view that every human tooth has been evolved by the concentration of the germs of two primitive triconodont reptilian teeth, and that where a sexicuspid tooth does not result, it is a sign of incomplete development." He went on to state that transposition occur when "the deuteromere of the tooth in the canine region has been developed to a greater degree than normal (resulting in a premolar instead of the normal canine) and that of the tooth in the premolar region has been suppressed (resulting in a canine instead of a premolar)." Though tackled from an evolutionary angle, this theory also strongly implicates genetic etiology for, after all, "overdevelopment" or "suppression" of any of the concerned odontomeres can only be the function of genes, especially in light of other observations noted above. Of course, as per this theory, altered suppression or overdevelopment reverses the phenotypic expression of the teeth and no actual exchange of tooth germs occurs.

There remains an important question to answer: At what point does the transposition originate? Nelson[10] suggested that transposition originates during cellular migration following induction of cell groups. He further states that at this time, moving epithelial cells "test" positional information put out by the mesodermal cells over which they move by matching protein chains. When they contact cells at the site for which they are programmed, they stop. The mutation which causes the anomaly may lie within the mesodermal organizer genes in that the cells producing canine and premolar placement proteins arise in reverse order along the developing maxilla, causing the canine and premolar epithelial cells to attach in a transposed position. Once these tooth cells have attached, they are in position and proceed to develop and grow normally.[10] The same may occur for canine-lateral incisor transpositions or any other transposition types.

The permanent tooth originates from the dental lamina as an offshoot of that for the deciduous tooth.[28] If Nelson's[10] concept is correct, then there must exist separate "lines" of cells specific for deciduous and permanent teeth that move in association with each other, depending upon tooth type. This association insures that permanent and deciduous canines develop from the same dental lamina. When this association of cell-line is disturbed, depending on the disturbance, transposition arises. This means, essentially, that the offshoot dental lamina arising from the dental lamina for the deciduous canine gives rise to a premolar, and vice-versa. Similar relationships must also exist when other teeth are involved. This must be so because two teeth from the same lamina grow within the same bony crypt, thereby keeping their relative positions more or less constant.

The interchange of dental anlage during developmental stages has been proposed as a cause of transposition,[29] as has trauma,[5] which presumably causes an "exchange" imposed by an external force. It is important to remember that transpositions affect the permanent teeth but leave the deciduous teeth normal.[19] Since the deciduous tooth/germ and its permanent successor are in the same bony crypt,[28] it is obvious that precise "exchange" of germs of the permanent tooth/germ would not occur when the deciduous teeth have not erupted. In the phase following the eruption of deciduous teeth, such precise exchanges would be precluded due to obvious anatomical, morphological, and physiological reasons. Indeed, it is difficult to see how "exchange of dental anlagen" could cause transposition. If trauma were to play a role, one would expect displacement of the dental elements rather than precise exchanged position, as in transposition.

Peck et al.[1] suggested a polygenic, multifactorial inheritance for transposition of teeth and supported the concept of field influence in the process of odontogenesis. In light of the above discussion, we believe that transposition of teeth has a definite genetic basis. The detailed mechanism of origin of transposition, however, is a matter for further research.

Peck et al.,[1] in their excellent work, stated "one significant failing of previous articles has been the mistaken search for a single unifying etiology to fit all the apparent cases of transposition, maxillary and mandibular..." We presume that the term "apparent" indicates the inclusion of pseudotranspositions within the category of transposition. However, once only true transpositions are discussed, it becomes important to find one unifying theory to describe the etiology of transposition. We see no reason to consider canine-premolar and canine-lateral incisor transpositions to be different phenomena. As explained earlier, we hold that all transpositions (true transpositions) are the varying manifestations (in involvement of different teeth types) of the same phenomenon.

Author Address

Amit Chattopadhyay
253E, DDA Flats (MIG)
Rajouri Garden
New Delhi 110027
INDIA

A. Chattopadhyay, assistant professor, oral pathology, Dept. of Oral Pathology and Oral Medicine-Radiology, SDM College of Dental Sciences & Hospital, Dharwad, India.

K. Srinivas, MDS trainee, Dept. of Oral Pathology and Oral Medicine-Radiology, SDM College of Dental Sciences & Hospital, Dharwad, India.

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