Etiology of maxillary canine impaction: A review


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This article is a review that enumerates the causes of impaction of the maxillary permanent canines, including hard tissue obstructions, soft tissue lesions, and anomalies of neighboring teeth, and discusses the much-argued relationship between environmental and genetic factors. These phenomena have been shown in many investigations to accompany the diagnosis of canine impaction and have been presented as unrelated anomalous features, each of which is etiologically construed as genetic, including the aberrant canine itself. While in general the influence of genetics pervades the wider picture, a guidance theory proposes an alternative etiologic line of reasoning and interpretation of these studies, in which the same genetically determined anomalous features provide an abnormal milieu in which the canine is reared and from which it is guided in its misdirected and often abortive path of eruption. (Am J Orthod Dentofacial Orthop 2015;148:557-67)

With the exception of the third molars, impaction of the maxillary permanent canines is the most common form of tooth impaction. Relatively recent studies into the frequency with which maxillary canine impaction occurs in the general population have indicated a prevalence from 0.27% in a Japanese population[1] to as much as 2.4% among Italians,[2] with the condition affecting female patients 2.3 to 3 times more frequently than males.[2-5]

Notwithstanding the opinions of some respected researchers in favor of an exclusively genetic etiology for its occurrence, there are many and varied reasons for impaction of the maxillary canines.[6-9] The causes can be classified into 4 distinct groupings: local hard tissue obstruction, local pathology, departure from or disturbance of the normal development of the incisors, and hereditary or genetic factors.

LOCAL OBSTRUCTION

Clinical and radiographic assessment of a number of impacted canine cases led Lappin[10] to observe that the deciduous canines were frequently overretained, often with a long and unresorbed root (Fig 1). He speculated that the nonresorption of the deciduous canine was the cause of the anomaly. However, this was not a controlled study; he drew the conclusions solely from what he had observed. Although the mechanism for root resorption of a deciduous tooth is unknown, it is recognized that it occurs when the dental follicle is near an unerupted permanent tooth. Thus, it is equally plausible to argue the reverse: that resorption has not occurred because of the distance of the permanent tooth; therefore, the unresorbed root of the deciduous canine is not the cause of the displacement, but rather its result.

On the other hand, Lappin's speculation[10] might be justified, since several studies have shown that prophylactic extraction of the deciduous canines when there is the potential for maxillary permanent canine impaction appears to encourage spontaneous eruption of a majority of displaced permanent canines.[11-13]

From parallel work relating to impacted incisors, we know that hard tissue pathology in the immediate area can cause displacement of a developing tooth. The first entities that come to mind are the supernumerary tooth and the odontoma. These diagnoses are highly definitive, and they present an etiologic role that is easily understood. Although this is a potent cause of impaction and is frequently seen in relation to impacted central incisors, supernumerary teeth and odontomata in the canine area are relatively rare (Fig 2).[14]

Perhaps a little more surprising was the finding in a recent study that in unilateral cases of central incisor

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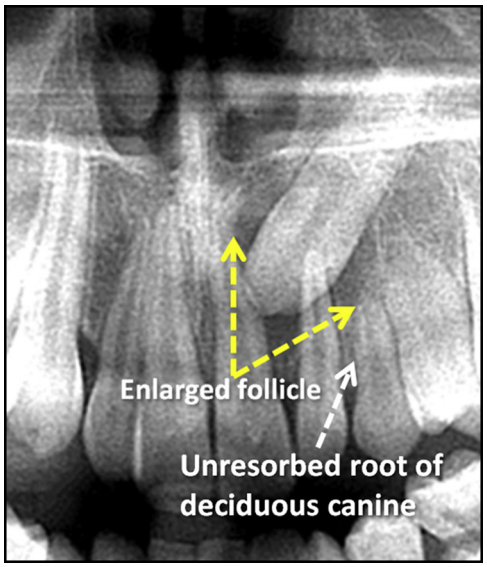

Fig 1. Panoramic view of a 12-year-old girl with a palatally impacted maxillary left canine. There is an enlarged dental follicle surrounding its crown (*yellow arrows*), and the deciduous canine has a long unresorbed root (*white arrows*).

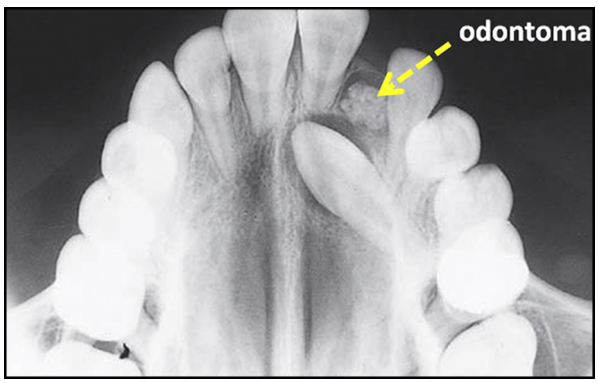

Fig 2. Odontoma (*arrow*) preventing eruption of the canine. (Reprinted from Becker A. Orthodontic treatment of impacted teeth. 3rd ed. Oxford, United Kingdom: Wiley Blackwell; 2012.[14])

impaction, whether because of obstruction by a supernumerary tooth or an odontoma or because of dilaceration or recent trauma, there is a high frequency of eruption disturbance of the canine on the same side.[15] This investigation showed a significant increase in the prevalence and severity of displaced canines (41.3%): buccal displacement was seen in 30.2%, palatal displacement occurred in 9.5%, and canine-lateral incisor transposition in 1.6% of the patients. Half of the buccally displaced canines on the ipsilateral side were pseudotransposed with the adjacent lateral incisor.

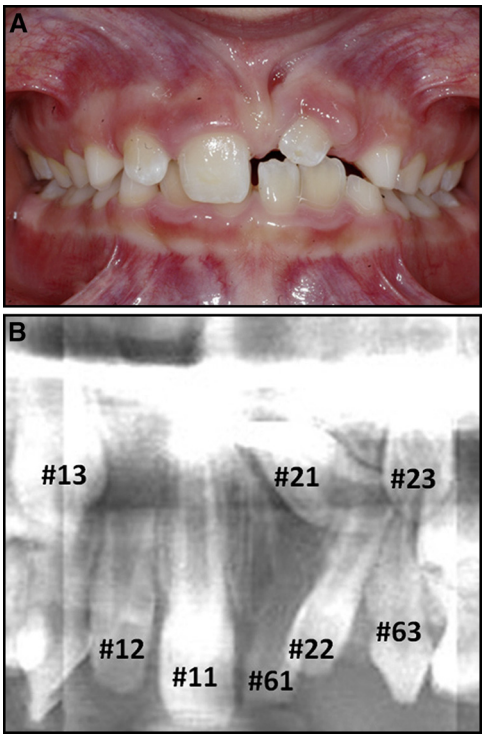

Fig 3. A, Intraoral view of an 8-year-old child with an unerupted left central incisor. The adjacent lateral incisor has erupted and is strongly tipped mesially, encroaching on the space for the missing tooth. **B,** Panoramic film taken before extraction of the deciduous central incisor (*#61*). The permanent central incisor (*#21*) is dilacerated with its crown in the area of the anterior nasal spine. The long axis of the lateral incisor (*#22*) is strongly tipped, displacing the root end distally and into a close relationship with the canine crown (*#23*).

These figures compared with a total of 4.7% on the contralateral side.

These abnormal features may be explained by the fact that the lateral incisor tips mesially and encroaches on the space of the unerupted central incisor to a considerable degree (Fig 3, *A*). The corollary of this is that the root apex tips distally and into a position where it interferes with the eruption path of the unerupted canine.

In the immediate neighborhood of the canine, the sequence of eruption of teeth dictates that the maxillary lateral incisor and the first premolar precede the canine by 3 years and 1 year, respectively. As long as the canine is in its normal eruptive position (ie, slightly buccal to the line of the dental arch) and has adequate space, its path of eruption will permit it to erupt unhindered. However, if the premolar has erupted with a mesiobuccal rotation, then its palatal root will be rotated directly into the path of the canine (Fig 4). In this way, the abnormal

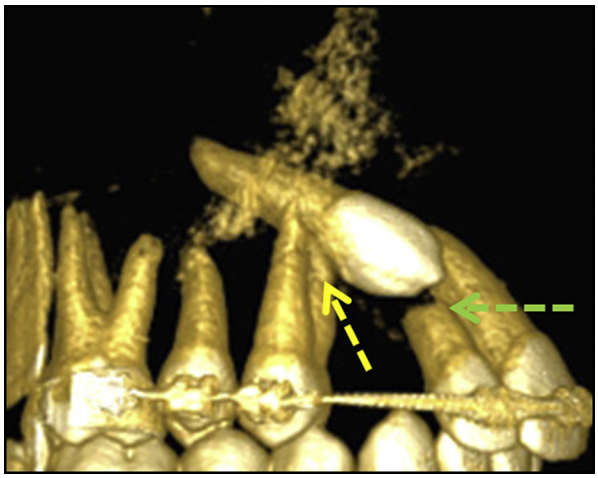

Fig 4. Cone-beam computed tomography 3-dimensional screen shot of an impacted maxillary right canine shows how the orientation of the palatal root of a first premolar can cause canine impaction (*yellow arrow*). Note the resorption of the lateral incisor root (*green arrow*). (Reprinted from Becker A. Orthodontic treatment of impacted teeth. 3rd ed. Oxford, United Kingdom: Wiley Blackwell; 2012.[14])

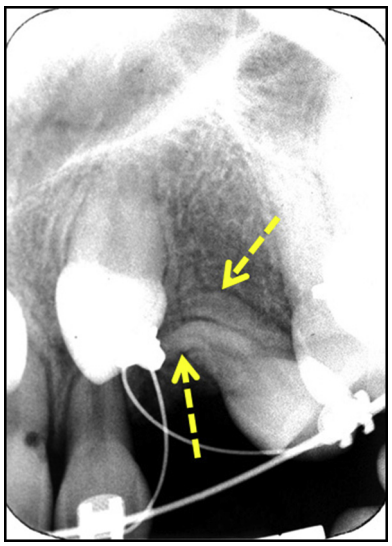

Fig 5. This periapical view of another patient was taken to check why there was no progress in the attempted resolution of the canine impaction. Both roots of the first premolar can be seen to turn mesially in their apical third (*arrows*) and lie in the direct path of the impacted canine.

orientation or abnormal root form of the adjacent first premolar (Fig 5) can be the impediment that causes the impaction of the maxillary permanent canine, despite its normal development and location.

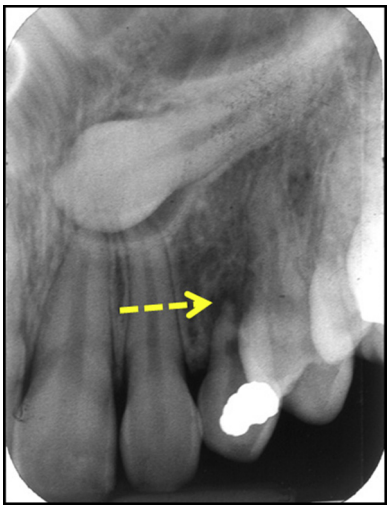

Fig 6. Periapical view of a palatal canine. The deciduous canine has a distal restoration, is nonvital, and can be seen to exhibit periapical pathology (*arrow*).

LOCAL PATHOLOGY

Overretained deciduous canines are commonly nonvital by the age of 12 years because of caries, trauma, or extreme attrition. The resulting chronic periapical granuloma, by itself, is a soft tissue inflammatory lesion that will have a potent effect on deflecting or arresting the eruption of the permanent canine (Fig 6). Extraction of a diseased deciduous canine usually and concurrently eliminates the granuloma, which is the displacing factor for the permanent tooth.

Investigations into the efficacy of prophylactic extractions of deciduous canines have been referred to above. Those articles do not mention whether patients with nonvital deciduous canines were included in the study samples. One may be permitted to question just how many deciduous canines in these study samples were nonvital. A high percentage of the permanent canines had later erupted spontaneously in what was claimed to be the apparent sequel to the extraction of the deciduous predecessor. There are grounds to argue that their successful eruption can also be attributable to the concurrent elimination of a periapical lesion.

In rare instances, a granuloma develops into a radicular cyst by adversely stimulating the rests of Malassez in the area, and this expanding, space-occupying, fluid-filled, turgid, and epithelium-lined balloon will displace adjacent unerupted teeth. It is more likely, however, that a long-standing granuloma at the apex of a deciduous canine may induce cystic change in the follicular sac of the adjacent unerupted permanent canine, which begins as a benign enlargement of the sac surrounding the permanent canine and increases to become

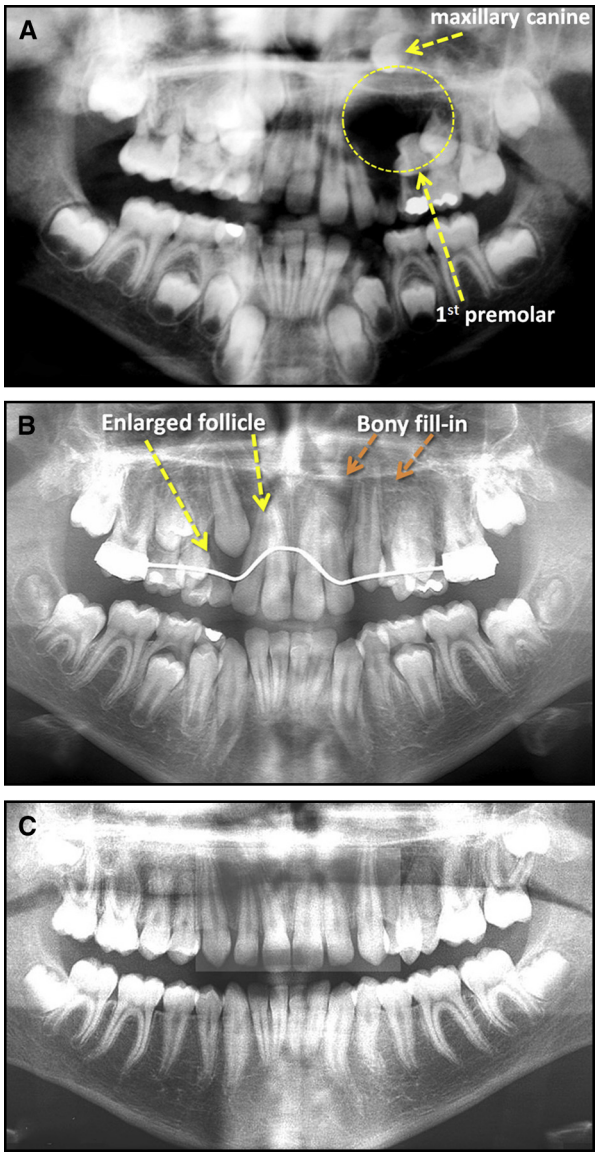

Fig 7. A, September 2008. A large cyst occupies much of the left side of the maxilla (approximately demarcated by the *yellow ring*). The lateral incisor root has been tipped mesially into contact with the central incisor root. The first premolar lies horizontally in the floor of the cyst, and the canine has been pushed upward and tipped almost horizontally. This appears to be a radicular cyst resulting from the nonvital deciduous first molar. (Reprinted from Becker A. Orthodontic treatment of impacted teeth. 3rd ed. Oxford, United Kingdom: Wiley Blackwell; 2012.[14]) **B,** December 2010. After marsupialization of the cyst, the canine has progressed rapidly with excellent fill-in of alveolar bone behind it to eliminate the former cyst cavity (*orange arrows*). The premolar has uprighted. Note the large eruptive cyst encompassing the crown of the maxillary right canine (*yellow arrows*). A palatal arch space maintainer was placed immediately after the surgery. No other treatment was provided. **C,** February 2013. After extraction of the right deciduous canine, the eruptive cyst dispersed, and there was good bony fill-in after the autonomous eruption of both canines.

a dentigerous cyst. The hydrostatic pressure in the cyst overcomes the innate force of eruption of the tooth, arresting its downward progress and even causing the tooth to "back up" in more advanced cases. The cyst may continue to enlarge by initiating pressure resorption of the adjacent bone, until the cyst lining comes into contact with the roots of adjacent teeth, which will be displaced into an adjacent area of potentially resorbable bone.

One method of treatment of a dentigerous cyst involves opening it to the exterior—marsupialization—allowing for drainage and effectively defusing the displacing factor. The area previously occupied by the cyst remains lined with cystic/follicular epithelium. When the increased hydrostatic pressure is released, bone begins again to fill in behind this epithelial lining, which undergoes metaplasia as its healing cut edges become contiguous with the oral epithelium. The residual cyst cavity slowly shrinks, and teeth that had previously been located in the cyst wall begin to migrate with the returning bone toward more accessible positions. In this way, prophylactic extraction of a deciduous canine to resolve potential palatal canine impaction may be successful in producing spontaneous eruption, as the result of the simultaneous and inadvertent rupture and evacuation of an associated and enlarged follicular sac or early dentigerous cyst (Fig 7). This has also been beautifully illustrated in several case reports.[14,16,17]

Trauma to the face can cause laceration of the soft tissues of the lips and cheek, and its force may be transmitted to the maxilla to cause displacement of the unerupted canine or dilaceration of its developing root, particularly in younger children. Pursuant to incidents of this kind, the tooth may become impacted.[18]

It is clear that local anatomic obstructions and hard and soft tissue pathologic entities can cause deflection in the normal eruption path of the canine. When these etiologic factors are eliminated, there is a degree of autonomous correction in the eruption path that may lead to spontaneous eruption of the canine.

DISTURBANCE OF NORMAL DEVELOPMENT

As the starting point to understanding abnormal development that creates canine ectopy, it is crucial to first understand how normal development occurs and how, in this scenario, the canine maneuvers its way in relation to the roots of adjacent teeth. As it does so, it

influences the alignment of the adjacent teeth, and at the same time, its own eruption and alignment are influenced by them, until it erupts into the mouth and its final position.

The mechanism of normal eruption and normal alignment of the maxillary anterior teeth was first described by Broadbent[19] over 70 years ago. He outlined how the eruption of the 2 central incisors produces an initial temporary arrangement that is quite different from the final alignment just 4 or 5 years later. He called this temporary arrangement the "ugly duckling" stage. He described the orientation of the 2 newly erupted maxillary central incisors and the initial wide intercoronal space between them—the midline diastema—and how this space spontaneously closes in the fullness of time with the eruption of the canines. At the outset, he considered that the diastema was caused by the early developmental location of the unerupted lateral incisors, high up on the distal side of the roots of the central incisors. With one lateral incisor on each side in the narrow apical area, the roots of the central incisors are pushed together to cause distal flaring of their crowns. A hypothetic apically directed extension of their long axes converges somewhere above their developing apices.

In the ensuing months of normal development, the lateral incisors migrate downward on the distal aspect of the central incisors, surrendering their constricting effect on the apices of the central incisors. As they move down past the cementoenamel junction, they eventually erupt, distally flared, into interproximal contact with the crowns of the central incisors. The outcome of this is that their relationship to the incisors becomes reversed. The central incisor crowns are influenced to tip mesially, reducing the wide diastema and partially uprighting the long axes of these teeth.

At the age of 8 years, normally developing unerupted canines may be seen on a periapical radiograph to be mesially angulated, high on the distal side of the apical third of the roots of the lateral incisors, in much the same relationship that existed between the lateral and central incisors a year or so earlier. The canines constrict the 4 developing incisor apices into a small space, and their crown-to-root long axes converge to a virtual point high above their apices (Fig 8, *A*).

During the ensuing 2 to 3 years, the downward eruptive movements of the canines are guided along the distal aspect of the roots of the lateral incisors; as they move down, they release their "stranglehold" on the incisor apices and generate a progressive mesial uprighting of all 4 incisor crowns as they go. This results in closing off what remains of the midline diastema and an integral chain of interproximal contacts between the crowns of the 6 anterior teeth (Fig 8, *B*).

This account of the natural dynamics of eruption and alignment of the maxillary anterior teeth, described by Broadbent[19] so long ago, has become a well-recognized and established cornerstone of our orthodontic literature and has withstood the test of time.[1] It is widely quoted in the literature and is accepted as axiomatic to the narrative of normal growth and development. This is the guidance theory of eruption of the maxillary anterior teeth.

From this description, it is clear that much can go wrong in this complex scheme of events and have an effect on the eruption path of the canines. Indeed, Broadbent[19] speculated that because of the long path of eruption taken by the maxillary canine from close to the floor of the orbit to its final destination—a distance of 22 mm—it had a greater chance of going off course. It clearly requires a relatively small discrepancy in direction or degree of influence of one factor to malfunction and to undermine this fragile scheme. His view was that this was why the canine occasionally became palatally displaced.

About 50 years ago, Miller[20] and Bass[21] independently observed that the prevalence of palatal displacement was greater when lateral incisors were congenitally missing. They concluded that the absence of the lateral incisor denied the canine its guidance, permitting it to migrate palatally. These conclusions were based on clinical impressions from viewing a number of patients in the clinic and not from a disciplined study of a large sample of affected patients vs an appropriate random control group.

In a study conducted by our group in Jerusalem on a large sample of palatal canine subjects, we found normal lateral incisors in only half of the patients, whereas the other half had missing, peg-shaped, or small lateral incisors.[4] By comparison in random studies in the same geographic area, normal lateral incisors were present in 93% of the general population sample. We subsequently initiated a study of the parents and siblings of our palatal canine patients and found an exceptionally high incidence of hereditary lateral incisor anomalies.[22] We also found a high incidence of palatal canine displacement.[23] These results have been confirmed in many subsequent studies in other countries.[3,6-9,24]

With such a compelling association, the obvious, facile, and simplistic conclusion is that palatal impaction of the canine is also genetically determined and linked to anomalous or missing lateral incisors—a view that is held by the authors of most of these studies.

THE GUIDANCE THEORY OF CANINE IMPACTION

It is nevertheless possible to view these associated phenomena from a different perspective. Maxillary

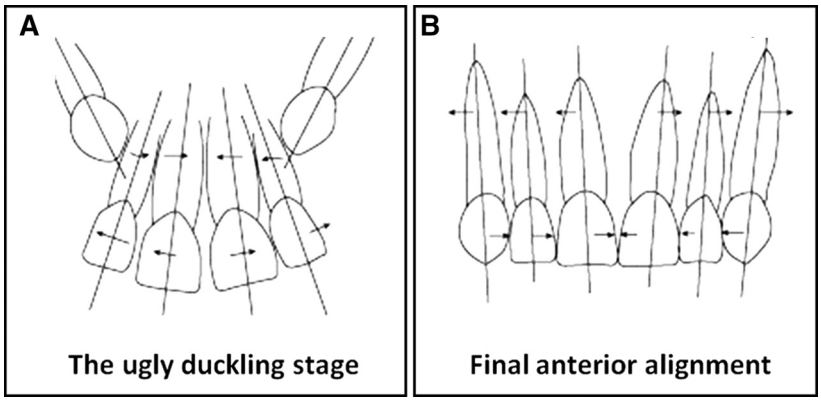

Fig 8. A, Diagrammatic representation of the relationships between the maxillary incisors, and between them and the unerupted canines in normal development in a 9- to 10-year-old patient. The canines restricted the roots into a narrowed apical area, causing lateral flaring of the incisor crowns. **B,** Diagrammatic representation of the final alignment and long axis reorientation after eruption of the canines. (Reprinted from Becker A. The orthodontic treatment of impacted teeth. 2nd ed. Abingdon, United Kingdom: Informa Healthcare Publishers; 2007. Reproduced with permission).

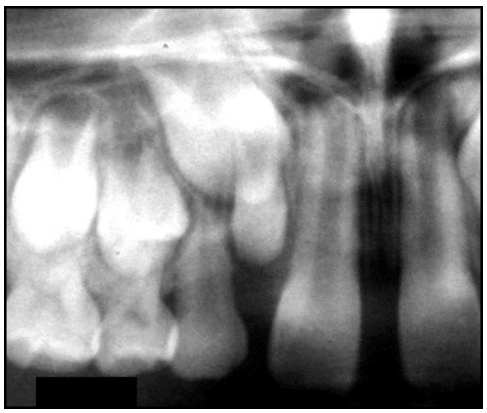

Fig 9. Anterior section of a panoramic view of a 10-year-old boy with delayed dental development conforming to age 8 overall. The crown of the unerupted lateral incisor is reduced in size and mildly peg shaped, whereas its developing root has a length normally seen at age 4 to 5 years.

lateral incisors normally erupt at age 7½ to 8 years, when their root development is between two thirds and three quarters complete. However, these teeth are notoriously variable in their development and are among the most likely to be congenitally absent from the dentition.[25-36] Maxillary lateral incisors are also among the most frequent with a deficient form, with small and peg-shaped crowns, and have been confirmed as representing a microform or a lesser degree of severity of agenesis.[26,27,35] In these circumstances, their development is often as much as 3 or 4 years later than for normal lateral incisors. Thus, a 10-year-old child may have an unerupted lateral incisor; from the radiograph, it will be determined that its crown is peg-shaped, and typically there is only a third or less of the expected root development and an open apex (Fig 9). These features are well recognized as genetically determined traits.[6-9,25-36]

If we now refer back to the above description of the normal development of the anterior teeth, we are reminded that at the age of 9 to 10 years, the unerupted canine is normally found at the distal aspect of the root of the lateral incisor. In contrast to the maxillary lateral incisors, maxillary canines are ontogenically stable teeth in terms of shape, size, and developmental timing. If the lateral incisor is absent or late developing, peg shaped, or small with only the earliest degree of root development, it will be clear that the canine will not find the guidance that would enable it to descend along its normal eruption path (Fig 10). Thus, the tooth may move down in a more palatal path into the downward converging, V-shaped alveolar ridge until it comes close to the periosteum of the medial aspect of the alveolar process. This process acts as a secondary guide, encouraging the canine to descend farther in the ensuing year or two; if the lateral incisor is absent, the canine can erupt autonomously in the line of the arch about age 11 to 12 years (Fig 11). However, in the presence of a late-developing and now-erupted anomalous lateral incisor, this self-correcting mechanism is not available, and the incisor then takes on the role of an obstruction that impacts the canine on its palatal side.

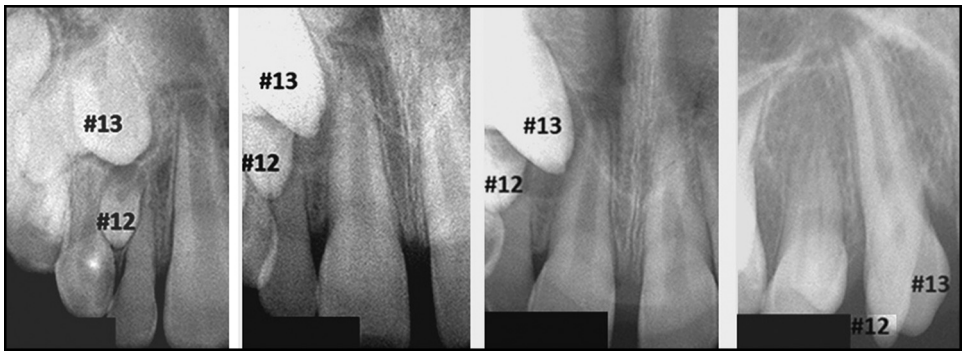

Fig 10. Periapical radiographs of an untreated girl, taken between ages 8 and 15 years. The lateral incisor (*#12*) is peg shaped and extremely late developing. It provides no guidance for the canine (*#13*), which progressively moves to the mesial aspect, passing the lateral incisor, to finally erupt palatally and mesially to it.

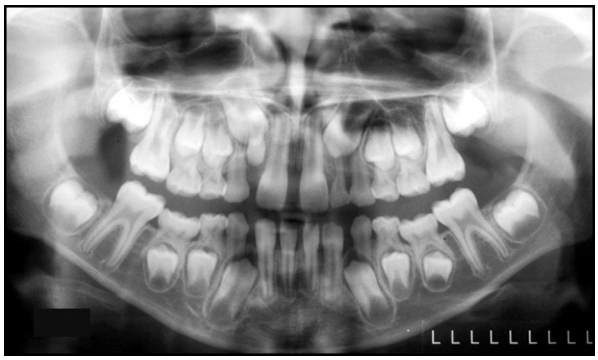

Fig 11. Full panoramic view of Figure 9 and shows the congenital absence of the left lateral incisor. The left canine appears to be migrating mesially into a position where it could cause resorption of the root of the deciduous lateral incisor and, probably, the canine.

This description is the essence of what has been termed the guidance theory of canine impaction. A fuller description of it can be found elsewhere.[14] Its salient keys are the following.

1. The immediate environment surrounding the unerupted canine is governed by genetically controlled factors (late development of small lateral and peg-shaped incisors or by congenitally absent incisors).[6-9,25-36]
2. The direction and progress of canine eruption are strongly influenced by environmental factors. This is particularly relevant when there is a lack of chronologic coordination between normal canine eruption, on the one hand, and growth of a developed and guiding incisor root of adequate length, on the other.[4]

INCONSISTENCIES OF THE GENETIC THEORY OF CANINE IMPACTION

The right side of any patient is genetically identical to the left side; thus, any genetic condition of one side will also affect the other. One cannot find a patient with cystic fibrosis or Marfan's syndrome or cleidocranial dysplasia affecting just one side of the body. Notwithstanding, in several hereditary conditions, the degree of penetrance can affect one side more, resulting in varying degrees expressions of the characteristics of that condition. Cleidocranial dysplasia is an autosomal dominant inherited disease, for which the *RUNX2* gene is specifically responsible. An affected patient may show variations in gene expression and have more supernumerary teeth on one side than the other, but the left and right sides are undoubtedly affected. As noted above, missing, small, and peg-shaped lateral incisors are 3 varieties of expression of 1 genetic factor. Therefore, one may frequently see a peg-shaped or small lateral incisor on one side of the mouth and a missing antimere. In genetics, bilateralism is the rule rather than the exception.

Therefore, if canine impaction were under hereditary control, it is reasonable to expect bilateral canine impaction in most patients, with a small percentage of patients showing variations in gene expression and lesser degrees of impaction on one side than on the other. From the epidemiologic information gleaned from the many studies in the orthodontic literature, the findings indicate a 60% to 75% preponderance of unilateral canine impaction.[13,37,38]

If we assume that canine impaction is genetic, then it is logical to expect to find monozygous (identical) twins with more impacted canines than dizygous (fraternal) twins. The outcome of a study testing this hypothesis refuted this assumption; the authors found similar degrees of concordance for ectopic canines in both groups, suggesting a nongenetic etiology.[39]

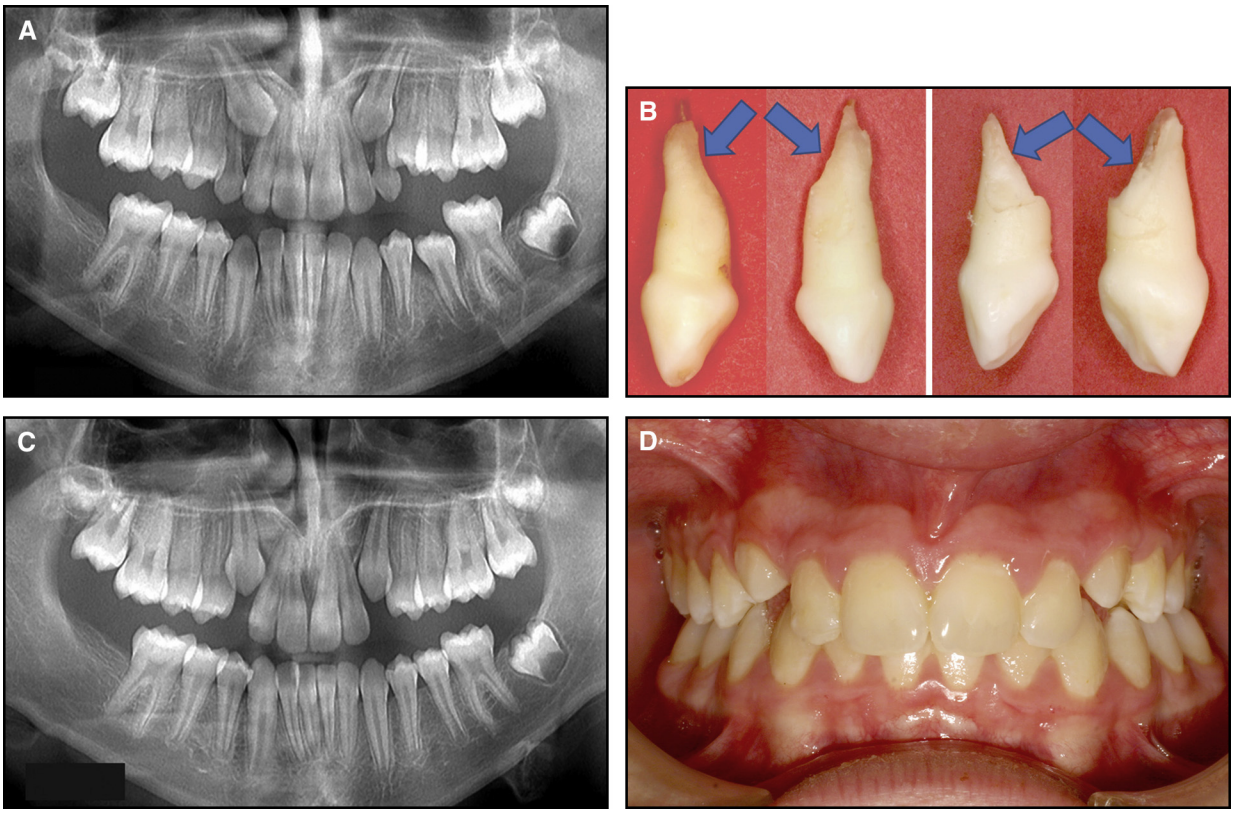

Fig 12. A, Panoramic view of the dentition of an 11-year-old boy, showing mesially angulated unerupted maxillary canines with enlarged follicles. Both canines are mildly palatally displaced (confirmed with supplementary views). Note the overretained and long-rooted deciduous canines, the congenital absence of the mandibular right second molar, and the late-developing mandibular left second molar. **B,** The extracted deciduous teeth seen from the mesial and distal aspects show oblique resorption of the roots on their palatal sides (*arrows*), indicating the location of the permanent canines. **C,** A panoramic view taken 1 year after extraction of the deciduous canines shows a favorable alteration in the orientation of the canines, whose normal eruption appears to be imminent. **D,** Clinical anterior view of the dentition shows the buccal migration of the erupting permanent canines. This illustrates spontaneous secondary correction of previously palatal canines.

In our earlier studies, we had found that small and peg-shaped incisors appeared to be more frequent in association with a displaced canine than with congenital absence.[4,24]

To confirm this, we undertook the study of a highly selected group of patients, each of whom exhibited (1) unspecified unilateral maxillary canine impaction, (2) unspecified unilateral congenital absence of the maxillary lateral incisor, and (3) an anomalous lateral incisor (small or peg shaped) on the other side.

The aim was to see whether the side with the congenital absence of the incisor or the side with the anomalous lateral incisor had a greater affinity to be associated with the impacted canine. Our findings were that the overwhelming majority of canine impactions were associated with the anomalous lateral incisor: 7 times more than on the side of congenital absence.[40]

If the behavior of the canines is truly governed by genetics, it is to be expected that the stronger genetic trait (congenital absence) would be associated with a greater frequency of canine impaction. However, canine impaction was shown to be more frequent with the weaker genetic pattern—anomalous incisors that represent a microform (less severe, weak, or partial expression, or incomplete penetrance)—of total absence.[26,27,34,35] This finding appears to contradict the genetic theory.

CANINE ERUPTIVE BEHAVIOR CHANGES WITH PROACTIVE ENVIRONMENTAL ALTERATION

A genetically determined anomaly of the lateral incisor creates an alteration in the local environment that encourages an uncontrolled and deflected path of eruption of the canine. Many studies have investigated the efficacy

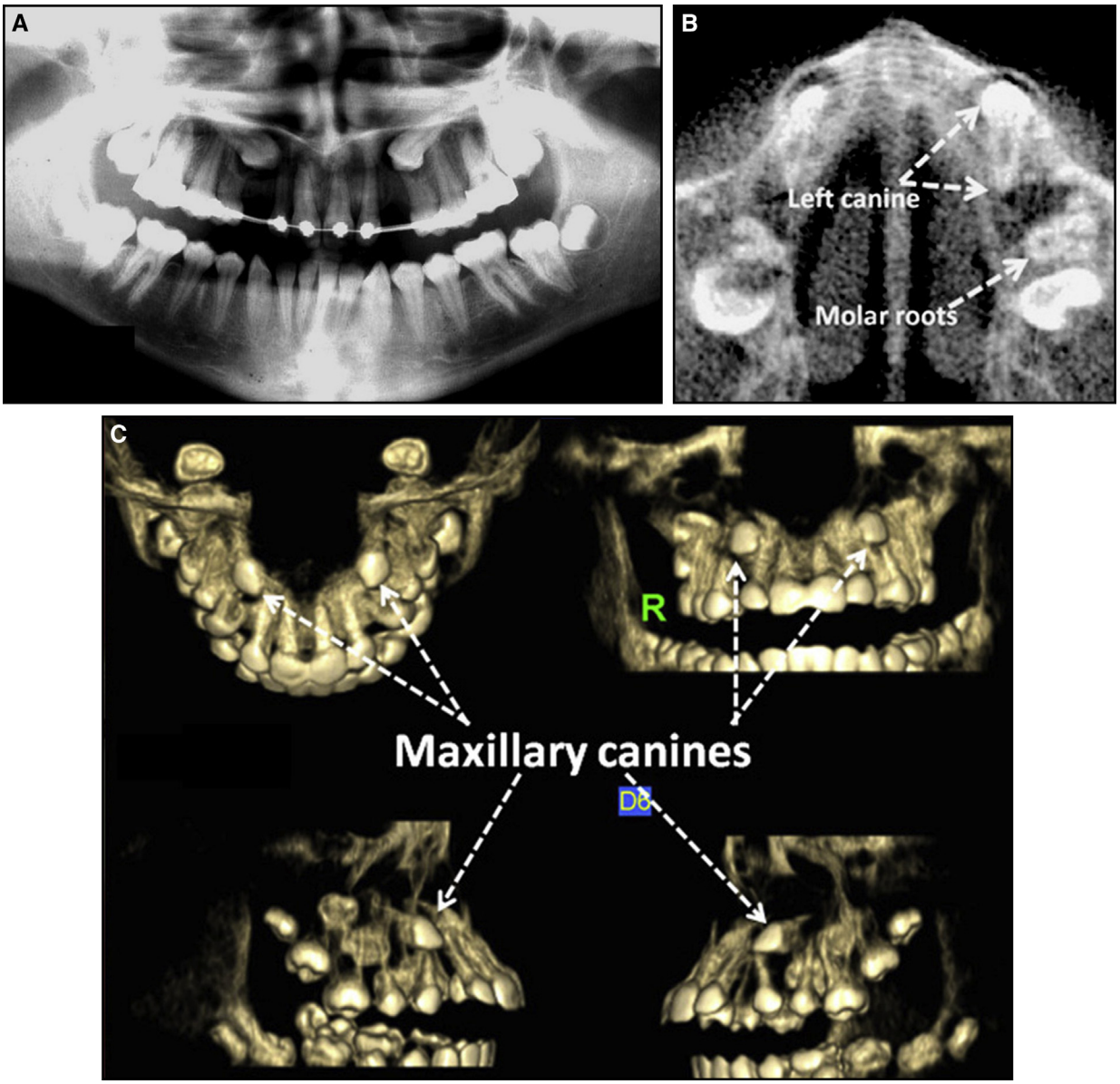

Fig 13. A, Panoramic view taken after space was created in the arch for the bilaterally impacted maxillary canines. Note the ectopic location of their apices in the first and second molar region. There is an obvious abnormal orientation of the long axes of these teeth. **B,** The axial (*horizontal*) cut from the cone-beam computed tomography image shows the apices of the canines to be both posteriorly and medially displaced from their normal locations and the teeth to be orientated horizontally with buccally directed long axes. **C,** 3-dimensional reconstruction images viewed from the anterior, right, left, and above confirm the location of the root apices and the orientation of the teeth in space (*arrows*).

of various indirect modalities aimed at influencing a corrective redirection. These conditions include (1) extraction of the deciduous canines (Fig 12)[11,13,41-43]; (2) extraction of the deciduous canines and first molars[44]; (3) increasing the space in the arch in the immediate area with routine orthodontic treatment[41-43]; (4) extraction of the deciduous canines with or without the use of cervical headgear[41-43]; (5) rapid maxillary expansion[45]; (6) extraction of the first premolars in a serial extraction procedure,[14] with the entire justification for this to alleviate anterior crowding by encouraging the canine to adopt a more distal eruption path; and (7)

extraction of a peg-shaped lateral incisor,[14] also to increase the chances of spontaneous eruption of an adjacent impacted canine.

There is ample evidence that a spontaneous change of the eruption path of the canine occurs because of an alteration of environmental conditions.

The guidance theory and the genetic theory share the belief that certain genetic features occur in association with the cause of palatal displacement of the maxillary canine. These include small, peg-shaped, and missing lateral incisors, spaced dentitions, and late-developing dentitions. The core issue over which the 2 theories differ is as follows.

According to the genetic theory, palatal displacement of the canine is just another associated genetic characteristic.

According to the guidance theory of canine impaction, these factors create a genetically determined environment in which the developing canine is deprived of its guidance, thus influencing it to adopt an abnormal eruption path.

Based on the evidence quoted above, it seems clear that determination of the eruption path of the palatal canine is, for the most part, not under genetic control.

CANINE IMPACTION THAT IS EXCLUSIVELY GENETIC

Notwithstanding the above argument, there are specific forms of canine impaction that are exclusively hereditary.[46]

In normal dentofacial development, the teeth are arranged in sequence along the dental arch—incisors, canines, premolars, and molars—each in its place, because each has originated from a specific point in the embryonic dental lamina in that order.

For the vast majority of impacted maxillary canines, the root is long, and its apex is correctly located in the mesiodistal line of the dental arch and in its appropriate buccolingual location, high above the apices of the roots of the adjacent teeth. An abnormal orientation of the long axis of these canines, therefore, will have displaced the crown to an abnormal location. But the root apex of the canine indicates the original location of the tooth germ; as with other teeth, apical mislocation is exceptional. Such mislocation, when it occurs, is dictated by genetic factors and is largely seen bilaterally, because the patient's left side is genetically identical to the right. There is undoubtedly room for variations of expression, but this will usually take the form of minor right-left differences in the orientation of the long axes of the teeth, caused by local factors, but apex location is most likely to be similar on each side.

There will be a relatively high degree of bilateral occurrence of canines whose root apices are displaced distally or palatally to the premolars (Fig 13). Similarly, transposition of the canine with the premolar is also due to apex displacement.

These situations have nothing to do with guidance and cannot be satisfactorily treated by extraction of the deciduous canines or other interceptive modalities, insofar as they exhibit what has been termed "primary displacement of the tooth bud."[11] The etiology of these teeth with mislocated root apices is exclusively heredity. The location of the apex is the diagnostic key to distinguishing it, and this requires sophisticated imaging, best provided by cone-beam computerized tomography.

Twenty years ago, Kokich and Mathews[47] declared that the etiology of impacted maxillary canines is unknown. Insofar as there is no single and exclusive cause, they were correct. But that does not mean that nothing is known about agents that are etiologically associated with its occurrence. On the contrary, the scientific community is manifestly confused by the wealth of information about the etiology of impacted canines. The only problem is interpreting it.

CONCLUSIONS

Alteration of the immediate environment of the unerupted maxillary canine by hard tissue bodies, soft tissue lesions, or developmental pathologic entities can cause the tooth to become impacted, whereas their elimination often results in partial or complete resolution. On the other hand, creating space, by anteroposterior or lateral expansion, by extraction of teeth, and by uprighting premolar or incisor roots, is effective in favorably redirecting the eruption path of an errant canine. The timing discrepancy that is seen in the normal development of anomalous adjacent teeth has been shown to be implicitly linked with canine impaction.

Anomalies in the anatomic form of the lateral incisor are considered to be a partial expression or a genetic microform of its absence, yet canine impaction is less likely to occur with a missing adjacent lateral incisor. Right-left equivalence is the rule in genetics; yet unilateral canine impaction outnumbers bilateral occurrence by 2 or 3 to 1. The parity of its prevalence in monozygous vs dizygous twins is difficult to explain in genetic terms.

The evidence presented here endorses the assertion that eruption of the canine is strongly influenced by environmental factors. The current tendency to glibly and roundly blame genetics as the fundamental cause of canine impaction appears to be unwarranted.

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