REVIEW ARTICLE

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Permanent maxillary canines – review of eruption pattern and local etiological factors leading to impaction

Anand K. Sajnani

KIMS Qatar Medical Centre, Wakra, Qatar

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Correspondence
Dr Anand K. Sajnani, Specialist – Paediatric and Preventive Dentistry, KIMS Qatar Medical Centre, Abdulrahman Bin Jassim Al Thani Street, P.O. Box 82125, Wakra, Qatar.
Tel: +974-30181952
Email: aksajnani@gmail.com

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Abstract

The position of the permanent maxillary canine at the angle of the mouth is strategically significant in maintaining the harmony and symmetry of the occlusal relationship. However, the maxillary canine is the second most frequently impacted tooth, with prevalence reported to be between 1% and 2%. Moreover, treatment of this condition is often complex and involves substantial time and financial cost. Hence, it is only prudent to monitor the eruption and identify the etiological factors that lead to impaction of the maxillary canine. Numerous researchers have tried to identify specific and nonspecific etiological factors responsible for displacement of canines. The purpose of this review was to track the development processes of maxillary canines and determine the hindrances that affect the eruption at different ages. Awareness of the eruption process and etiology of noneruption will help to reduce the incidence of impacted canines by allowing for early recognition and interceptive treatment.

Introduction

The human canine tooth has been of particular interest ever since claims of finding evidence of fossil apes was first documented in the 1830s in France and the then British Colonial India. More recently it has been suggested that in modern man, the canine has no special function to perform.[1] Charles Darwin[2] wrote:

> The early male forefathers of man were probably furnished with great canine teeth; but as they gradually acquired the habit of using stones, clubs, or other weapons for fighting with their enemies or rivals, they would use their teeth less and less. In this case, the jaws, together with the teeth would become reduced in size. This tooth no longer serves man as a special weapon for tearing his enemies or prey; it may, therefore as far as its proper function is concerned, be considered as rudimentary (p. 26).

Nevertheless, the location of canines is important to an individual's appearance since the canines play a major role in the support of the facial muscles.[3] Apart from their role in mastication, which is mainly tearing,[4] the canines exhibit the greatest combined crown plus root length in each arch and their root is very firmly anchored in alveolar bone. Because of the thick bony support and the length of the root, the canines are usually the most steadfast teeth in the mouth.[3] Also, many authorities firmly believe that the maxillary canine guides the mandible into centric position so as to prevent the contact of the remaining opposing teeth until they meet in centric occlusion.[5] Functionally, the lack of canine guidance due to noneruption has negative consequences on the dynamics of the temporo-mandibular joint,[6] and the neighboring teeth, which exhibit a high frequency of root resorption.[7,8] In addition, patients without canine protection have a Class III malocclusion five times more frequently than those with canine protection.[9]

Significance of maxillary canines

The presence of the tooth bud of the canine in the dental arch followed by its natural eruption into the oral cavity, provides the basis for its normal structure and periodontal support. However, this may not always be the case and, although rare, congenitally missing canines are a definite possibility.[10] Congenitally missing permanent canines pose a number of treatment planning challenges. Factors to be considered include the condition of the

primary predecessor, the number of missing teeth, the overall alignment and occlusion, and most importantly the patient's and/or parents' preferences. Treatment options may include timely extraction of the primary predecessor to facilitate spontaneous space closure with or without further orthodontic alignment, or to retain the primary canine and replace it with a suitable prosthesis when lost.[11,12]

Between the two extremes of natural eruption and congenital absence lies the aberration of "impaction". Impacted teeth are those with a delayed eruption time, or that are not expected to erupt completely based on clinical and radiographic assessments.[13] Impaction of a permanent canine is said to be a condition in which the tooth is embedded in the alveolus so that eruption is prevented.[14] Both the maxillary and mandibular canines may be impacted, however, mandibular canine impaction is regarded as a much rarer phenomenon.[15]

"There can be no doubt that in the scheme of occlusion Nature intended the canine to be one of its foremost mainstays. Nevertheless, this keystone of the human denture is found in positional abnormalities of the maxillae more often than any other tooth, and its failure to find its normal position in the arch is a calamity to the occlusal mechanism" (p. 678).[16] It follows then that the impaction of the maxillary canine is one of the most perplexing problems a dental practitioner has to face in their practice and there is no other oral anomaly that requires greater ingenuity than the treatment of this condition.[17] The permanent maxillary canine is the second most frequently impacted teeth and the prevalence has been reported to be 1–2% in the general population.[18,19] Also, there is now sufficient evidence in the literature to show that the maxillary canine is the most frequently impacted tooth in childhood.[20] The treatment of an impacted canine usually involves a surgical approach to either remove the tooth or to perform orthodontic movement to correct the position.[21] These procedures offer a high success rate but can involve substantial time and financial cost. They also carry a risk of gingival recession, bone loss, and detachment of the gingiva around the treated canine, especially if care is not taken to ensure that the canine either erupts or is positioned into keratinized mucosa.[22] If the canines have to be moved a considerable distance then ankylosis is a distinct possibility, as well as loss of vascular supply and therefore pulp death.[23] Treatment often takes in excess of 2 years and it is important to maintain a motivated and cooperative patient.[23] It is necessary to create sufficient space for the canine to be aligned and this is usually around 9 mm.[23] Also, it is quite common at the end of treatment to see a slightly darker crown of the permanent canine, this probably results from either a change in vascularity and vitality of the canines, or potential hemoglobin products seeping into the dentine thus changing the color of the overlying enamel.[23] The protracted length of treatment also results in patients abandoning treatment. Thus, impaction of a canine poses a convoluted situation to the clinician, leading to not only loss of function but also compromised esthetics in the maxillofacial region.

Despite all of our improvements in treatment mechanics and diagnosis for impacted canines, the eruption path is often unpredictable. Canines that have a seemingly hopeless prognosis can sometimes correct their position and erupt. Nevertheless, to wait and observe a patient where the canines are clearly in difficulty without referral to a specialist would be difficult to defend legally.[23] Hence, it is only prudent to monitor the eruption process and identify the etiological factors that lead to the impaction of maxillary canines.

Etiology of impaction

Over the years, numerous researchers have focused on trying to identify specific and nonspecific etiological factors responsible for displacement of canines and several theories have been put forward to explain this anomaly. These can be broadly divided into generalized and localized causes. The generalized ones have been attributed to many diseases, syndromes, and systemic factors, including hypopituitarism, hypothyroidism, cleidocranial dysostosis, Down syndrome, achondroplasia, hypovitaminosis (A or D), amelogenesis imperfecta, and osteopetrosis.[24,25]

The most common causes for canine impactions, however, are usually localized and they occur as a result of any one, or combination of, the following factors: tooth size/arch length discrepancy,[26] prolonged retention or early loss of the primary canine,[3,17,27], abnormal position of the tooth bud and the long path of eruption,[3,25,28,29] presence of an alveolar cleft,[30,31] ankylosis,[32] follicular disturbance and cyst or neoplasm formation,[32,33] dilaceration of the root[34,35] or trauma,[36] and idiopathic factors including primary failure of eruption.[37]

Crowding was considered to be one of the major causes of impacted maxillary canine, for both buccal and palatal displacements.[26] Association with certain malocclusions such as an Angle Class II, division 2 relationship has been suggested.[38] However, it is unclear and there is a consensus in the literature that arch length deficiency is associated primarily with buccal canine displacement.[13,29,39,40] Further, a number of studies have shown that the likelihood of palatally displaced canines is lower when crowding is present.[29,41,42]

An etiological influence on maxillary canine displacement has, for a long time, been attributed to the various developmental phases of the tooth germ and the long

eruption path.[3,25,28,29] The permanent canine develops high in the maxilla with calcification commencing 4–12 months post-natally and crown completion at 6–7 years of age. At the age of 2.5 years, the tooth germ of the permanent maxillary canine is lying above the first premolar tooth germ. From this position, the maxillary canine has a long and deviational path to cover.[17] In the three planes of space, the canine travels almost 22 mm from its position at the age of 5 years to its position at the age of 15 years.[43] While the primary dentition is being carried antero-inferiorly in conjunction with normal facial growth, the permanent canine lags behind during the crown formation phase and is closely related to the root of the permanent lateral incisor.[44] It moves down the distal aspect of the lateral incisor during eruption and this will often result in closure of a physiological midline diastema, if present.[45] Displacement from the normal path of eruption most commonly occurs in a palatal direction and this has been suggested to be the cause of the impaction.[13,14,17–19,24–26,28,29,32] Thus, there is a general consensus that buccal and palatal impactions have different etiologies. Although crowding has been considered the primary cause for buccal impactions, a number of causes have been attributed to palatal impactions. Currently the two most popular theories reported in the literature that have gained some degree of consensus worldwide are the guidance theory[18,32,46,47] and the genetic theory.[48]

Guidance theory

Miller[46] and Bass[18] reported that there appeared to be an unusually high prevalence of congenitally missing lateral incisors associated with palatally impacted canines. They suggested that under such circumstances, the permanent canine lacks the guidance normally afforded by the distal aspect of the root of the lateral incisor. Miller,[46] however, assumed that the root of even an abnormally small lateral incisor, such as a peg-shaped lateral incisor, is usually of adequate length to guide the canine along a normal course.

Many researchers have supported the hypothesis of Miller and Bass that the lateral incisor plays a significant role in guiding the normal eruption of the permanent canine.[18,46] Nevertheless, numerous proponents of the guidance theory have also reported a significantly higher incidence of hypoplastic and peg-shaped lateral incisors in patients with palatally displaced canines, when compared with the general population.[39,40,42,47,49,50] These authors considered palatal displacement of a canine to be due to the abnormal adjacent lateral incisor being unable to provide the required guidance for normal canine eruption.

The explanation given by Becker *et al.*[47] was based on a two-phase development of palatal canine displacement. During the first phase, the canine deviates from the physiologic eruption path in the palatal direction. This is often due to retarded development of hypoplastic maxillary lateral incisors, the roots of which are insufficiently formed to take over the guiding function at the critical time in the eruption of the permanent canine. Furthermore, in cases of incomplete root development and congenital aplasia of the maxillary lateral incisor there is an excess of space in the maxillary apical base.[47] This is the precondition for the canine to be able to leave its labial developmental position and migrate to a palatal position across the roots of the incisors and premolars. Jacoby[29] showed that patients with palatal displacement of their canines exhibited excess space.

During the second phase, corrective movements occur, with the canine moving into an upright position to fit into the dental arch. In patients with hypoplastic, or peg-shaped lateral incisors, the completely developed roots of the lateral incisors would prevent this self-correcting movement, whereas it could still take place if the lateral incisors are congenitally missing. Peg-shaped lateral incisors have been recorded approximately three times as often as congenital aplasia of those teeth in patients with palatal canine displacement.[47,48] Peck *et al.*[49] also reported a significant increase in the frequency of peg-shaped lateral incisors, but found no statistical significance in the frequency of agenesis of the maxillary lateral incisors in association with palatally displaced canines. Becker *et al.*[51] even suggest that aplasia was more likely to occur on the contralateral side, whereas hypoplastic and peg-shaped laterals are more likely to cause palatal displacement of the adjacent canine.

Thus, it is evident that the permanent lateral incisors exert a powerful local influence. However, in the majority of the cases, palatally displaced canines are found adjacent to normally developed incisors.[42,47,52] The guidance theory offers no explanation for this, hence recourse to the theory of genetic origin is necessary, which is supported by the increased risk of palatal canine displacement in association with aplasia or impaction of other teeth.

Genetic theory

The theory of "genetic origin" is based on the observation that palatal displacement of a canine rarely occurs as an isolated symptom but is generally accompanied by genetically determined tooth anomalies such as hypoplasia and/or agenesis of the maxillary lateral incisor,[18,39,46,49,50,53] or the aplasia of other teeth.[18,49] According to the literature, this is because the palatal displacement of a canine is due to complex genetically determined tooth anomalies, which

are ultimately aplasia-oriented and are in turn due to disturbances of dental development or of the dental lamina.[49,54]

The possibility of there being an autosomal inherited dominant trait with variable expression and incomplete penetrance is under discussion.[50,55–58] Family studies of patients with hypodontia have revealed mutations in the MSX1/MSX2 homeodomains. These mutations are expressed in dental tissues at the onset of tooth development and are held responsible for the developmental disturbance.[58–60] Besides tooth agenesis, tooth shape anomalies such as hypoplastic or peg-shaped teeth, tooth impactions and retarded tooth mineralization are regarded as covariables of this genetic developmental anomaly.[48,50,55,57] According to Peck *et al.*[48] it is not only the association with genetically determined anomalies but also the frequent bilateral occurrence, significant gender related differences, the cumulation of symptoms among affected families,[56] and significant interpopulation differences[29,39] that suggest a genetic origin for palatal displacement of the maxillary canine.

As a second possibility, the genetic etiology may be due to a disturbance in an ontogenetically critical zone,[56] for example, in the fusion area between the palatal shelves and the median nasal process. However, it is difficult to explain why the most pronounced manifestation of this disturbance, that is, aplasia of the lateral incisor, is not significantly greater adjacent to a palatally displaced canine[49] but rather on the contralateral side, as recorded by Becker *et al.*[51]

Although the genetic theory is an attractive hypothesis, it is difficult to solely subscribe to it as it attempts to justify circumstantial and epidemiological evidence as being genetic.[61] It remains uncertain, however, whether an anomalous lateral incisor is a local causal factor for palatally displaced canines (guidance theory) or if the displacement of the canines occurs as the result of an associated genetic developmental influence as proposed by the genetic theory.

Thus, from the literature it appears that no single theory can completely explain the etiology of impaction of maxillary canines. Also, so far, the scientific community has been treating buccal and palatal canine impactions as separate entities from an etiological perspective. Nevertheless, a recent theory has emerged which suggests that buccal and palatal canine impactions have similar etiological factors.[62]

Sequential theory – culmination of guidance theory and genetic theory

The sequential hypothesis provides a sequence in which the two most commonly accepted theories, that is, the genetic theory and the guidance theory, might act at different stages during the development of the maxillary canine and the surrounding structures.[62] It postulates that both buccally and palatally impacted maxillary canines share similar etiologies. The role of genetics as well as other extrinsic factors, particularly the influence of the lateral incisor, play a critical part at various periods during the development of the maxillary canine and subsequently determine if the canine would erupt into the oral cavity or become impacted.[62]

Sequential theory – factors leading to impaction at various stages of eruption and development

The tooth germ of the maxillary permanent canine starts to develop at the age of 4–5 months, high in the anterior wall of the maxillary sinus, under the floor of the orbit. At about 3 years of age, the intra-bony position of the developing maxillary permanent canine is inferior to the orbit, superior to the floor of the nasal cavity, and between the nasal cavity and the maxillary sinus. The crown of the tooth is directed mesially and lingually with respect to the primary canine and to the developing first premolar – it is also close to the mesial root of the first primary molar.[28,61] With the development of the first premolar, the developing permanent canine and first premolar and the first primary molar are all positioned one above each other. Meanwhile, the developmental position of the lateral incisor is palatal in relation to both the permanent central incisor and the permanent canine.[63]

At approximately 5 years of age, the incisal edge of the permanent maxillary lateral incisor is situated nearer to the occlusal plane than the incisal edge of the permanent central incisor.[61] Also, the disto-incisal corner of the maxillary central permanent incisor is in contact with the mesial surface of the roots of the adjacent primary lateral incisor. The sequential theory suggests that at this age, the maxillary canine begins to lose its potential to move in the vertical plane and would eventually become impacted.[62] While a normally erupting canine would travel approximately 22 mm,[43] a canine that would eventually remain impacted would travel less than 3.5 mm in the next 7 years of its development.[62,64] It is highly unlikely that the developing permanent lateral incisors, which would be positioned away from the permanent canine at this age, could substantially influence the eruption of the permanent canine at this point in time. The most likely cause for this difference is that genetic factors regulate the ectopic position of the canine. This genetic predetermination reduces the eruption potential of the tooth in the vertical plane towards the occlusal level.

At approximately 6 years of age, the canine crown tip is at the level of the nasal floor. It is positioned palatal to the primary canine root and directed mesially. The permanent canine, still in a buccal position though to the permanent lateral incisor, depending on the stage of development of the lateral incisor may occupy a position palatal to it. Deviation of the canine may become possible either because of excess space, agenesis, or microdontia of the lateral incisor,[47] or as suggested by the sequential theory[62] by a lack of vertical movement of the permanent canine in relation to a normally developing lateral incisor. However, the sequential hypothesis also suggests that in cases with both buccally and palatally impacted canines, microdontia was exhibited by the lateral incisors almost twice and thrice times respectively of that reported in the literature, thus offering support to the theory that both buccally and palatally impacted canines may have similar etiologies.[62]

Further, as suggested by the guidance theory, corrective movements may occur after this stage and the canine may move into a more favorable position in the arch. In patients with microdontia of the lateral incisor, this self-correcting movement will, according to Becker *et al.*[47] be prevented by the meanwhile completely developed roots of the maxillary lateral incisors, whereas it can still take place if the lateral incisors are congenitally missing. However, if the canine has lost the potential to move in the vertical plane, this stage may provide an opportunity for the tooth to move in a buccal direction, or to remain in the palatal position.[62]

Between 8 and 9 years of age the maxillary canine gains guidance from the developing root of the adjacent lateral incisor and begins to change from a mesial inclination to a more vertical direction and begins to lie parallel to the facial midline by 9 years of age. As further development takes place, the root of the lateral incisor eventually prevents the canine from occupying an ideal position in the arch.[28] Thus, a strong local influence of the lateral incisors in the ultimate impaction of a maxillary canine cannot be denied. However, both buccal and palatally displaced canines are also found adjacent to normally developed lateral incisors.[47,52] The guidance theory offers no explanation for this phenomenon and recourse has to be taken once again to the genetic theory, which is supported by the increased risk of palatal canine displacement with aplasia, or impaction of other teeth as supported by the sequential theory. Nevertheless, the sequential theory also demonstrates a high proportion of buccally impacted canine presenting with similar anomalies.[62]

While the intrinsic genetic mechanisms form the core of the sequential hypothesis, the influence of environmental factors such as guidance from the lateral incisor (or the lack of it) plays a vital role in the mechanism. It is suggested that genetic mechanisms strongly influence the potential of the maxillary canine to be impacted and the guidance from the lateral incisor and the stage of the development play a vital role in determining the ultimate position of the impacted canine (buccal or palatal).

Conclusions

Impaction of a maxillary canine is a frequent occurrence and requires a multidisciplinary approach for proper management. Awareness of the eruption patterns and etiology of impaction allow for early recognition and implementation of interceptive treatment. Consequently, this reduces the incidence of impacted canines and is beneficial in minimizing the need for active treatment in a patient who might otherwise have an acceptable occlusion.

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