Intrusion injuries of primary incisors. Part III: Effects on the permanent successors

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Intrusion injuries of the primary dentition are highly associated with developmental disturbances of their successor teeth. The age of the child at the time of injury, the direction and severity of the intrusion, and the presence of alveolar bone fracture are important variables influencing the effect of primary tooth intrusion on the developing permanent germ. The developmental defects of the permanent successor tooth range from mild alteration in enamel mineralization to severe sequestration of the developing germ. This article will review the developmental anomalies of the permanent incisors induced by intrusion injuries of their predecessors. (Quintessence Int 2000;31:377-384)

**Key words:** dilaceration, intrusion, odontogenesis, permanent tooth, primary tooth, sequelae, tooth injury

Different types of traumatic injuries affect the primary dentition. However, there is general agreement that primary incisors are highly liable to luxation (displacement) injuries, which constitute 21% to 81% of traumatic injuries of the primary incisors.1-9

Intrusion is a luxation injury, which usually results from an axially directed impact, displacing the incisor deeper into the alveolar socket. The displacement results in compression of and damage to the periodontal ligament, contusion to the socket walls, and injury to the pulp of the intruded incisor. The reported prevalence of intrusion injuries affecting primary incisors varies among different studies and ranges from 4.4% to 22%.2-4,7-14

The potential for disturbances of the developing germs of the permanent incisors is relatively high following injuries to their predecessors. This can be explained by the close anatomic relationship between the apices of the primary incisors and the germs of the succeeding teeth.1,9,15-17 The percentage of developmental disturbances of the permanent incisors that could be attributed to injuries of their predecessors ranges from 12% to 74%.6,1,14,16-24

The type of injury to the primary teeth influences the type and severity of disturbances of the permanent successor germs. For instance, intrusion injuries of primary incisors are highly related to serious developmental anomalies of their permanent successors3,9,15,19,20-22,24-29, frequencies range between 18% and 69%.6,9,16,20,22,25 This can be explained by the nature of intrusion injuries: The incisor is driven deeply into the alveolar bone, invading the follicle of the permanent germ, which lies palatally or lingually in close proximity to the primary incisor root.15,22,23,29-33 A cephalometric study has shown that the hard tissue barrier between the primary incisor and its successor has a thickness of less than 3 mm,33 and this barrier might simply consist of connective fibrous tissue.15 This intimate relationship between the permanent and primary incisors is maintained during the developmental years and explains the serious disruptive effect severe intrusion injuries can have on permanent germ odontogenesis.33

**FACTORS INFLUENCING THE SEQUELAE OF INTRUSION INJURIES**

Several factors play an important role in inducing developmental anomalies of the permanent successors following intrusion injuries to the primary incisors.

**Age of the child at the time of injury**

The influence of a child's age at the time of injury is very important.1,16,20-23,31 Many reports have stated that the younger the child at the time of intrusion injury, the more severe the induced sequelae on the successor tooth.16,20,22,23,31 For example, the frequency of developmental disturbances following primary tooth injuries that occur before the age of 2 years is 65%.
This frequency is reduced to 53% when injuries occur between the ages of 3 and 4 years and to 24% when injuries occur between the ages of 5 and 6 years. In addition, serious sequelae have been reported subsequent to intrusion of primary incisors before the child is 2 years old.20 This is due to the fact that germs of the permanent teeth are particularly sensitive in the early stages of their development, which occurs between the ages of 4 months and 4 years.16,31 At this critical time, injury or inflammatory changes can interfere with the different levels of odontogenesis, such as morphodifferentiation, organization, mineralization, or the final prerenptive maturation.1,16,25,35

Injuries that occur before the child is aged 3 years may result in a variety of anomalies, ranging from enamel hypoplasia to severe coronal dilaceration or odontoma.1,16,31 Usually, the location of the malformation depends on the quantity of enamel formed before the trauma takes place. Because the formation stage of permanent incisor crowns is usually completed around 3 years of age, the crowns become less sensitive to injuries occurring after this age, and the risk of enamel hypoplasia is reduced.31,32 With the beginning of root development, between the ages of 3 and 4 years, the root becomes the vulnerable part of the tooth and might suffer from different types of deformation, ranging from minor dilaceration to complete arrest in root development.1,16,31,32,36

On the other hand, no relationship has been found between the degree of primary root resorption and mineralization defects in the permanent successors.16,18,33 This can be attributed to findings that show the distance between the resorbed primary root and the developing permanent successor remains relatively consistent between the ages of 5 and 6 years.35

Although the permanent germ can be considered more sensitive to environmental changes in the early stages of its development, the germ can still suffer mineralization disturbances even after complete formation of its crown.15,16,18,27,34,37

**Direction of the intrusion**

The optimum conditions to create a disturbance in the developing germ arise when an intrusive force is applied to the lingual surface, displacing the crown labially and intruding the root palatally. In this case, the root apex of the primary incisor contacts or invades the follicle of the permanent germ.1,14,16,20,21,27,35-40 The immediate outcome of such an injury would be disruption of enamel epithelium, displacement of the hard tissues in relation to the cervical loop, or disruption of Hertwig's epithelial root sheath.1,12 Conversely, an intrusive force applied to the labial surface usually displaces the crown palatally and intrudes the root labially away from the permanent tooth germ, reducing the possibility of future related deformation to the developing germ.34,20,29,30,52,56

**Severity of the intrusion**

A high correlation has been found between the degree of the intrusion of primary incisors and the frequency and severity of developmental disturbances of their successors.28 For example, the percentage of permanent incisor malformation subsequent to moderate or severe intrusion (grade II or III), in which more than 50% of the crown is intruded in the socket, is higher than that following mild, or grade I, intrusion.28 In severe palatal intrusion, the primary incisor can invade the follicle surrounding the germ and destroy wide areas of the developing enamel matrix.27,59,60

In addition, if fracture of the alveolar socket or jaw accompanies the intrusion, the chance that the impact will be transferred to the developing germ and disturbance in morphology or mineralization will be induced is increased.1,16,39,41

**Type of treatment**

There is some controversy among different studies about the relationship between treatment procedures for the intruded primary incisors and the resulting anomalies of their permanent successors. Several studies have found no significant difference in the frequency or extent of developmental disturbances of the permanent successor, whether the intruded tooth was extracted immediately or allowed to erupt spontaneously.16,20,28,42 Macroscopically and histologically, it does not seem that there is a difference between the effects of preservation or immediate removal of recently intruded primary incisors on the resulting damage to their succeeding teeth.42

Ravn21 conducted a study on 78 children, mostly aged between 1 and 3 years, and 100 intruded primary incisors. He reported that 52% of the observed and reerupted intruded incisors induced disturbances of their successors, compared with 72% of the intruded incisors that were extracted after the injury. This indicates that injury to the permanent tooth germ is sustained at the time of the actual intrusion and that subsequent management directed at the primary tooth has a minor influence on the developing successor.15,16,20,42,43 Another study by Selliseth25 reported that developmental disturbances were more severe if the primary incisors were treated and allowed to exfoliate spontaneously than if they were extracted. However, these findings were limited to children older than 3 years, and the differences for other age groups were insignificant.
When a wait-and-watch approach is elected to manage intruded primary incisors, pulpal necrosis and subsequent periapical inflammation are possible complications.\(^1\) Pulpal disintegration in the primary incisor may result in apical inflammatory changes, such as osteitis followed by bone resorption. The infection may spread and surround the follicle of the permanent germ, leading to metaplastic changes in enamel epithelium and sometimes to disturbance in dentin formation.\(^2\) The outcome of such infection would be disruption of odontogenesis, reflected by enamel hypomineralization and/or hypoplasia and, in severe cases, arrest of permanent germ development.\(^3\)

The duration of the unattended insult (the infection) is a key consideration in the resulting malformation of the permanent germ.\(^4\) Experimental studies in monkeys have shown that a periapical inflammation of 6 weeks' duration around primary incisors did not lead to developmental alterations of their successor teeth.\(^5\) Moreover, because of the short exposure time of the insult, no alteration in the formation or the mineralization process was noted when the infected primary incisors were extracted immediately after diagnosis of periapical inflammation.\(^6\) This can be explained by the protection provided to the tooth bud by the follicular tissues and the bony crypt, even when the thickness of the undamaged tissue is less than 1 mm.\(^7\)

Conversely, the longer the infection remains untreated, the greater is the possibility of damage in the permanent germ.\(^8\) Chronic inflammation leads to accumulation of neutrophilic leukocytes in the bony environment surrounding the permanent tooth bud, providing a mechanism to damage the enamel epithelium and creating enamel malformation.\(^9\) In severe cases of chronic periapical infection around the primary tooth, cessation of permanent germ development may be the outcome.\(^10\)

**SEQUELAE AFFECTING CORONAL PORTIONS OF THE PERMANENT SUCCESSORS**

**White or yellow-brown discoloration**

Enamel discoloration is described as internal enamel hypoplasia or internal disturbance of mineralization.\(^11\) This sequela may result from intrusion of primary incisors during the mineralization stage of the permanent incisor crowns between the ages of 2 and 7 years.\(^12\) Ravn\(^13\) reported that 44\% of the permanent incisors suffering from internal white or yellow-brown discoloration followed intrusion injuries to their predecessors before the child was aged 4 years.

The discolorations appear on the labial surface of the crowns as isolated hypocalcified areas, ranging in size from small white spots to large yellow-brown areas (Figs 1 and 2). The discolored areas correspond to the stage of calcification. The incisal third of the permanent crown is usually the main discolored portion following a traumatic injury to the primary incisor.\(^14\)

The whiteness is a manifestation of insufficient calcification during the maturation stage of the developing enamel, corresponding to areas where the traumatic invasion occurred.\(^15\) The enamel epithelium bordering the damaged enamel may show an accelerated deposition of minerals.\(^16\) Microscopic examination will show involvement of the reduced enamel epithelium and destruction of highly specialized ameloblasts, responsible for the maturation process.\(^17\)
If intrusion of the primary incisor is severe, bleeding may occur in the periapical tissues. Consequently, degradation products of hemoglobin enter the mineralizing portion of the enamel, resulting in a yellow-brown discoloration. The discoloration can occur in different stages of crown mineralization, even after cessation of ameloblastic activity and completion of crown formation. This is due to the fact that secondary crown mineralization occurs over an extended period of time, even during root development.

Because disturbances in mineralization cannot be revealed on radiographs, diagnosis of coronal discoloration can only be made clinically, after complete eruption of the tooth.

**White or yellow-brown discoloration associated with enamel hypoplasia**

Enamel hypoplasia implies an imperfect formation, which results from an injury to the coronal portion of the developing germ during the formation stage of the enamel before the age of 2 to 3 years. Intrusion injuries of primary incisors were highly related to enamel hypoplasia of their successors. An intrusion impact may result in displacement in the normal alignment or the synchronized activity of ameloblasts. Moreover, it may induce an irreversible destruction of the active enamel epithelium, leading to its replacement with flattened squamous cell epithelium, and arrest of matrix formation, resulting in deformed pits and grooves. The edge of the hypoplastic enamel area represents the junction between the hard tissue formed before and after the injury. A histologic examination of the altered enamel will reveal abnormal angulation of enamel prisms and deposition of cementlike tissue.

Clinically, the typical appearance of the deformation is hypoplastic enamel with white or yellow-brown discoloration (see Fig 2), sometimes associated with a narrow horizontal groove surrounding the crown cervical to the discolored area. Prior to permanent incisor eruption, diagnosis of the hypoplastic enamel may be established radiographically. Deformed enamel areas would appear radiolucent on the radiograph (Fig 3). Enamel hypoplasia and discoloration can be managed with a variety of restorative techniques, depending on the severity and the extent of the deformation.

**Dilaceration of the crown**

Dilaceration is described as an acute deviation in the long axis of the crown or root, originating from a nonaxial displacement of already formed hard tissue in relation to the developing noncalcified tissue (Fig 4). Crown dilaceration can result from an intrusion of the primary incisor when a child is around the age of 2 years, when half the crown would be formed. Subsequent to invasion of the partially formed follicle by the apex of the intruded incisor, the mineralized portion of the crown twists lingually over the papilla. Sometimes, the displaced enamel epithelium becomes activated in the new position, forming a peculiar shape of deformed enamel projecting into the pulp canal or externally at the crown-root junction (Fig 5).

A radiologic diagnosis of crown dilaceration can be established prior to tooth eruption. The dilacerated crown appears foreshortened on the occlusal radiographic image in comparison with the adjacent crowns (Fig 6). Additionally, a lateral projection helps to establish a diagnosis and determine the direction of the dilaceration.

Teeth with dilacerated crowns may erupt normally or in facial or lingual version (Fig 4). Unfortunately, even without evidence of caries, they may later develop pulpal necrosis, followed by apical periodontitis and chronic abscess. To avoid such complications, the dilacerated portion of the crown should be removed after its eruption, and a provisional crown should be placed until final restoration is made. In other cases, their failure to erupt necessitates surgical exposure and orthodontic alignment followed by an aesthetic restoration (Fig 5).
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Fig 4 Dilacerated crown of the mandibular right central incisor, resulting from severe intrusion of the primary predecessor at age 26 months. The severely angulated crown has erupted horizontally in buccal version.

Fig 5 Dilacerated, deformed crown of the left central incisor, showing a peculiar shape of deformed enamel projecting at the distal side of the crown-root junction. Because the tooth was impacted, it was exposed surgically and moved with orthodontic force.

SEQUELAE AFFECTING ROOT PORTIONS OF THE PERMANENT SUCCESSORS

Intrusion of primary incisors may result in different types of root malformation when it occurs between the ages 2 and 5 years. An intrusion force with considerable magnitude will be transmitted through the bone, reaching the epithelial sheath of Hertwig during root development. Such force can deflect or displace Hertwig's epithelial sheath and consequently induce alterations in the shape or length of the root. The direction and the severity of the traumatic force has a major influence on the observed root deformities. Severe intrusion injuries may generate a distortion or displacement of Hertwig's sheath, leading to root duplication, root dilaceration, or complete cessation of root formation.

Duplication of the root

This rare malformation results from severe intrusion of a primary incisor around the age of 2 years, when less than half the crown is formed. The traumatic impact on the cervical loop results in radicular division to 2 separate mesial and distal portions.

Dilaceration of the root

Root dilaceration is an acute curvature in the long axis of the root, originating from a change in the direction of root development in either a vestibular or lateral direction (Fig 7). This complication may result from intrusion of a primary incisor after completion of permanent crown formation between the ages of 2 and 5 years. The trauma leads to displacement of the formed hard tissues in relation to the developing nonmineralized tissues.

Diagnosis of vestibular root dilaceration can be established with an occlusal radiograph, in which the affected tooth would show a foreshortened image compared with adjacent teeth. Because of the marked vestibular curvature of the root, the permanent incisor may change its path of eruption and become impacted. A lateral radiograph helps to localize the...
position of the dilacerated tooth. If adequate space is available, the tooth can be surgically exposed and aligned with orthodontic treatment.\textsuperscript{1,3,11}

Lateral root dilaceration appears on the radiograph as a mesial or distal curvature.\textsuperscript{1,2,12,25} It can result from traumatic injuries occurring between the ages of 2 and 7 years.\textsuperscript{1} Incisors with lateral dilaceration usually erupt normally.\textsuperscript{1,25}

Partial or complete cessation of root formation

This rare sequela is characterized by cessation of root formation, resulting from injury to primary incisors between the ages of 4 and 7 years.\textsuperscript{1} The injury damages Hertwig's epithelial root sheath and subsequently results in formation of a short root. The tooth may exfoliate prematurely because of inadequate periodontal support.\textsuperscript{1,9,25}

SEQUELAE AFFECTING THE WHOLE SUCCESSOR TOOTH

Odontomalike malformation of the permanent tooth

This rare malformation may result from severe intrusion of the primary incisor and invasion of the developing germ during early phases of odontogenesis, when the child is 1 to 3 years old.\textsuperscript{1,9,25,29,31,40} Cleavage of the tooth germ results in the development of separate tooth elements, characterizing an odontomalike structure. Radiologically, these teeth appear as an opaque mass. They usually do not erupt and require surgical extraction.\textsuperscript{1,9,40}

Sequestration of the permanent tooth germ

Cessation of tooth formation is characterized by an underdeveloped tooth germ and inadequate root formation (Fig 8), which may be regarded as sequestration of the permanent tooth germ.\textsuperscript{1,25} This rare sequela may result from severe, or grade III, intrusion of primary incisors.\textsuperscript{6,40} It has been also related to chronic periradicular infection, leading to bone resorption and inflammatory changes in the follicle of the permanent germ.\textsuperscript{1,35} Radiographically, a wide radiolucency is noticed around the developing germ, and the outline of the dental crypt is not visible.\textsuperscript{1,25,40} Treatment of this sequela would include administration of antibiotics and surgical intervention to enucleate the tooth germ and the surrounding granulation tissues.\textsuperscript{35,40}

Disturbances of permanent successor eruption

The influence of premature loss of primary incisors, such as the extraction of severely intruded primary incisors, on the alignment of their permanent successors is an important variable.\textsuperscript{5,14,35} To study the effect of primary incisor injuries on the alignment of their permanent successors, Brin et al\textsuperscript{55} compared 117 children in the trauma group with 137 children in the control group. Compared with the control group, the trauma group experienced a higher percentage of malpositioned incisors among the successors to primary incisors that suffered injury and early loss.\textsuperscript{55}

Following the early loss of a primary incisor prior to the beginning of its root resorption between the ages of 3 and 4 years, the eruption of the successor tooth is often delayed.\textsuperscript{32,34} This can be explained by
the abnormal changes that may occur in the connective tissue overlying the permanent tooth and the formation of thick, fibrous gingiva, which may result in delayed eruption of the permanent incisor.\(^1,5,6\)

Surgical excision of the thick gingiva usually facilitates eruption of the permanent incisor.\(^1,2,3,29\)

Conversely, if the primary incisor is lost after the child is aged 5 years, eruption of the successor tooth might be accelerated,\(^6,32\) especially in the presence of alveolar bone resorption following an infection of the injured tooth.\(^34\)

Ectopic eruption and an abnormal final position of permanent incisors are other possible outcomes following traumatic injuries to their predecessors.\(^5,34,35,36\) This can be explained by the physical displacement of the permanent germ, with or without dilaceration, at the time of the injury.\(^26,34,52,54,55\)

Another possible explanation is the lack of eruption guidance from the prematurely lost primary incisor, leading to eruption of the permanent incisor in labial or lingual version.\(^31,54-59\)

In addition, when watchful waiting is elected to manage intrusion, the intruded incisor may become ankylosed or delayed in its root resorption.\(^22,26,29,34,36,38\)

This leads to overretention of the primary incisor and disruption of the eruption pathway of the permanent successor tooth.\(^22,26\)

**CONCLUSION**

There is a high probability of damage to the permanent incisor germ following intrusion injury to its primary predecessor. This is due to the nature of intrusion, which displaces the primary incisor deeper into the alveolar bone, where the succeeding germ is lying in close proximity to the lingual surface of the primary incisor root. The extent of the disturbance of the developing germ is related to the stage of germ development (the child’s age at time of injury) and to the severity and direction of the impact.

The type of treatment offered subsequent to intrusion injury of the primary incisor is another important factor. Although no significant difference has been found in the developmental disturbances induced, whether the intruded tooth is extracted immediately or allowed to reerupt spontaneously, a long-term infection of the intruded tooth may lead to developmental alterations of the permanent bud.

When an observation strategy is elected to monitor the spontaneous reeruption of the intruded primary incisor, regular follow-up visits are very important. Periodic recall examinations help in detecting early signs of pulpal disintegration or inflammation of the periradicular tissues. Such infections may surround the permanent germ, leading to alteration in its odontogenesis. Parents should always be informed about the consequences of traumatic injuries to the primary incisors on their developing successors and the importance of follow-up examinations.

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