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Histological evaluation of dog permanent teeth after traumatic intrusion of their primary predecessors

Torriani DD, Percinoto C, Cunha RF, Guimarães I. Histological evaluation of dog permanent teeth after traumatic intrusion of their primary predecessors. © Blackwell Munksgaard, 2006.

Abstract – The aims of this study were to analyze the histomorphology of developing permanent teeth whose primary teeth had suffered traumatic intrusion, as well as to compare the influence of immediate extraction of the intruded tooth to passive re-eruption. Nine dogs from 45 to 50 days old were submitted to the intrusion of the maxillary central and lateral primary incisors using a force applicator adapted to the teeth incisal cuspids. The right side intruded teeth were kept in their sockets and the ones on the left side were extracted 30 min later. After a postoperatory periods of 30 and 60 days, four (group 1) and five (group 2) dogs, respectively, were killed by perfusion. The histological evaluations showed that, in group 1, alterations had occurred in the odontoblastic layer and deposition of the enamel matrix had taken place in some specimens while in group 2, a portion of non-mineralized matrix was observed. We concluded that the morphological changes were because of the immediate trauma of intrusion. No differences were found between the groups where the primary tooth was immediately extracted or left to passively re-erupt.

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When a tooth undergoes trauma, a number of injuries occur to different cells present not only in the affected tooth, but also in the surrounding tissues. Epidemiological data show a high occurrence of trauma in primary teeth (1–5), especially in the ages between 1 and 3 years old (3, 6, 7), which is the period when permanent incisors begin their formation (8). Thus complications to the developing permanent teeth are expected after trauma to the primary teeth. During this age period, intrusion is one of the most prevalent types of trauma (1, 9, 10), and it is claimed to be the most important injury associated with the development of complications (11–15).

Damage to the permanent tooth can be more severe in the younger the children (15). Disturbances in the permanent dentition were more frequent in cases when the injury occurred between the ages of 0 and 2 years old (63% of the cases) (1). When the trauma occurs during the three initial stages of odontogenesis, it may impair the normal pattern of dental development and result in clinically and radiographically perceivable defects (16). The authors have described the existence of a connection between the intensity of the traumatizing force with the age and the resulting damage to dental development. Macroscopically, minor color alterations of the permanent teeth can be noticed after the intrusion of the primary teeth. No relationship has been reported between complications with or without extraction of the intruded primary tooth (17).

Despite the frequency of the intrusion of primary teeth and the potential to interfere in the

development of the permanent successors, only a few studies have been performed to analyze the role of the odontogenic cells of the involved developing permanent teeth and the influence of certain treatments on these cell groups. Therefore, this study aimed to: (i) analyze the odontogenesis of dog permanent teeth whose primary teeth had undergone traumatic intrusion; (ii) determine the relationship between immediate extraction or passive re-eruption of the intruded teeth and the odontogenesis of their successors.

Materials and methods

The study protocol was submitted to the evaluation of the Animal Experiment Ethics Commission of the Araçatuba College of Dentistry, UNESP, and was approved. To perform the present study, nine dogs were used. The animals were of non-defined breed and of both genders, aged between 45 and 50 days, weighing 2.5–2.9 kg and in good health. They were pre-anesthetized (ROMPUM intramuscular, 0.2 ml, 1 cc) in order to perform the periapical radiographs of the lateral and central incisor region, using the paralleling device. The teeth selected to undergo the traumatic intrusion were the primary upper lateral and central incisors, and their successors presented with one-third of crown formed. For the experimental procedures, the dogs were anesthetized with a 3% Thionembutal solution, 1 ml kg^{-1} body weight. They were then placed on a surgical table that allowed perfect positioning of the animal, immobilization of the head and a good position of the maxillary region with good support, permitting the application of the force in the axis of the tooth to result in the intrusion. To cause the trauma, the selected upper incisors received a mechanical impact in a longitudinal direction to cause intrusion of the teeth. The force was applied through a device described in a previous report (18). The original tool was modified for this study by elongating the metal bar to enhance the force in order to get a reliable intrusive effect. The impact was directed to the incisal edge of the teeth at 4 kg intensity. The level of the intrusion varied between about two thirds of the crown and total intrusion.

The dogs were divided into two groups: in group 1, four animals were killed 30 days after the experiment; in group 2, five animals killed down 60 days after the experiment. The lateral and central incisors of the right side, after sustaining the intrusion, were preserved in the socket and the same teeth on the left side were extracted 30 min after the trauma, with a spatula and a haemostatic instrument. The animals received, in the first 48 h after the procedure, a semi-liquid diet and, after this period, food and water *ad libitum*. After a 30 and 60-day postoperative interval, the animals were again anesthetized and killed by intravenous perfusion of 10% formol. Then, the laboratory procedures prepared the specimens for optical microscope reading (Jenaval–Carl Zeiss, Jena, Germany), in 40–400× magnification.

Results

Group 1 (30 days postoperative)

From the animals used in this group, eight central permanent incisors were obtained, two in each maxilla. Of these eight teeth, four had their predecessor primary teeth extracted after the traumatic intrusion, and the other four primary teeth were kept in the socket throughout the experiment. Six primary teeth were observed in the arch in the stages of advanced root resorption, and in two specimens they had already exfoliated. In all teeth, it was possible to see the cells of the inner epithelium of the enamel organ, differentiated into adult ameloblasts. The Tomes' processes were visualized between the ameloblasts and the deposited matrix. Near these, blood vessels were observed. In all specimens, the non-mineralized matrix in the most cervical part of the middle crown third was observed. In the areas where mineralization occurred, the ameloblasts presented diminished height and volume. The pulp tissue showed all the characteristics of young connective tissue, with blood vessels, odontoblasts, young fibroblasts, undifferentiated mesenthimal cells and collagen fibers. The odontoblastic layer was present in all the extension of the pulp, with column-shaped cells disposed as a palisade. It was possible to verify the presence of predentin and dentin tubules that were seen in all the extension of the dentin. In the most cervical portion of the crown, the beginning of the formation of the Hertwig's epithelial sheath (Fig. 1) was seen.

Among the animals whose intruded primary teeth were kept in the socket, one specimen presented the ameloblastic layer with an altered outline, interrupted and without preserved characteristics. At this point, ameloblasts presented cuboid shape and, with the cells of the starred reticulum and intermediate layer, they appeared condensed, mixed, making their identification difficult. The adjacent matrix showed very intense coloring with a disorganized and flake-like center (Figs 2 and 3).

In the group of teeth in which intrusion and extraction of the primary teeth were performed, abnormality of matrix deposition (similar to a deflection in its original contour) was observed. The odontoblast layer appeared ruptured (Fig. 4). Therefore, from the eight evaluated specimens of

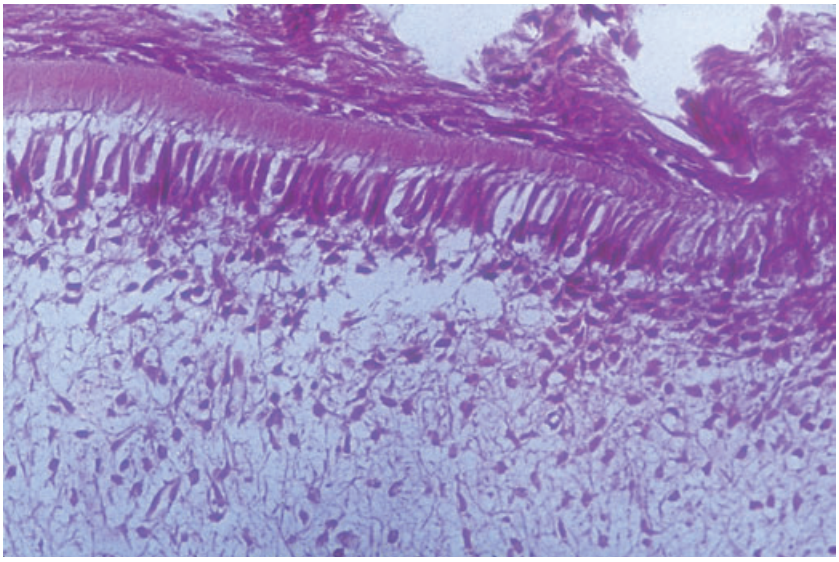

Fig. 1. Ameloblasts, matrix, near these, blood vessels, blood vessels, odontoblasts, young fibroblasts and undifferentiated mesenquimal cells, and also collagen fibers, odontoblastic layer, Hertwig's epithelial shealth (×100).

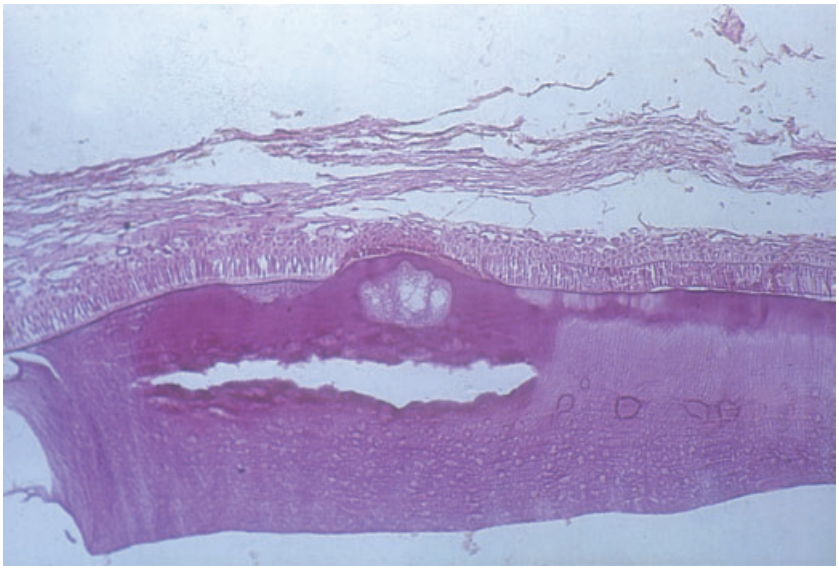

Fig. 2. Ameloblasts, starred reticulum, intermediate stratum and altered matrix (×100).

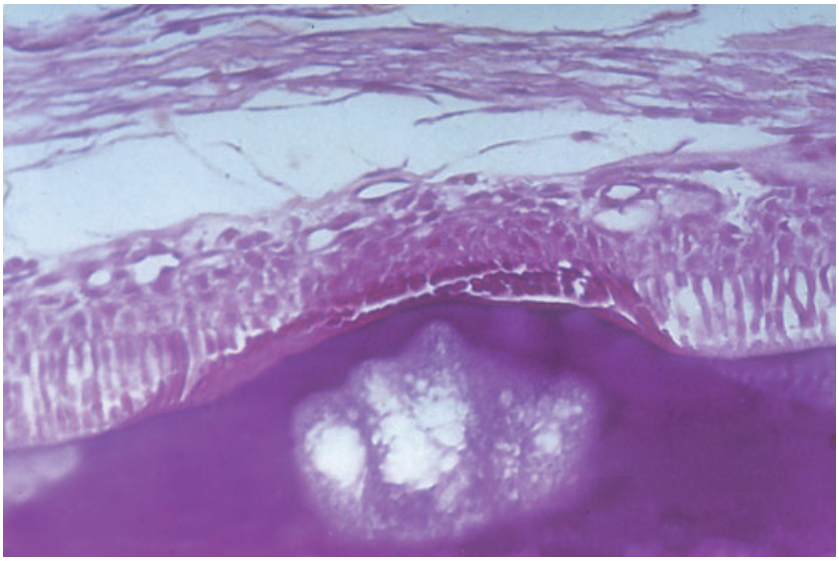

Fig. 3. Ameloblasts, starred reticulum, intermediate stratum and altered matrix (×400).

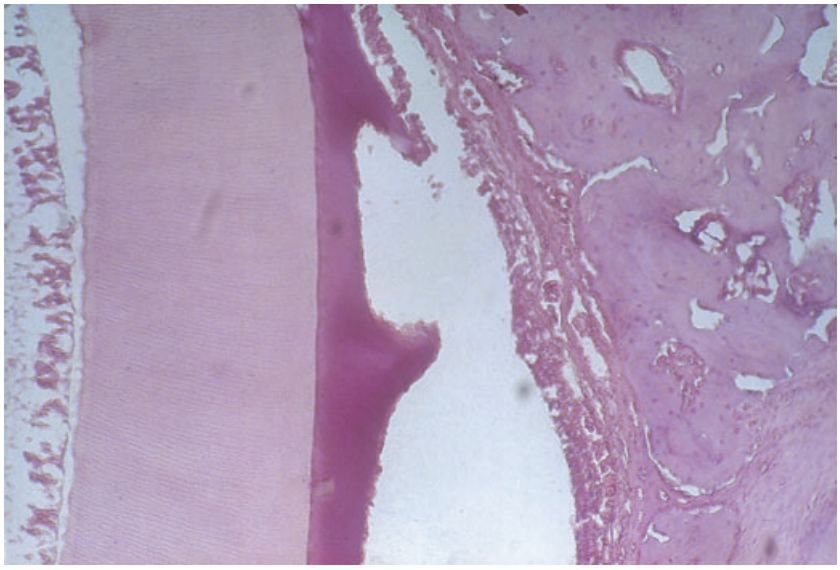

Fig. 4. Damaged ameloblasts and alteration of the matrix contour (×100).

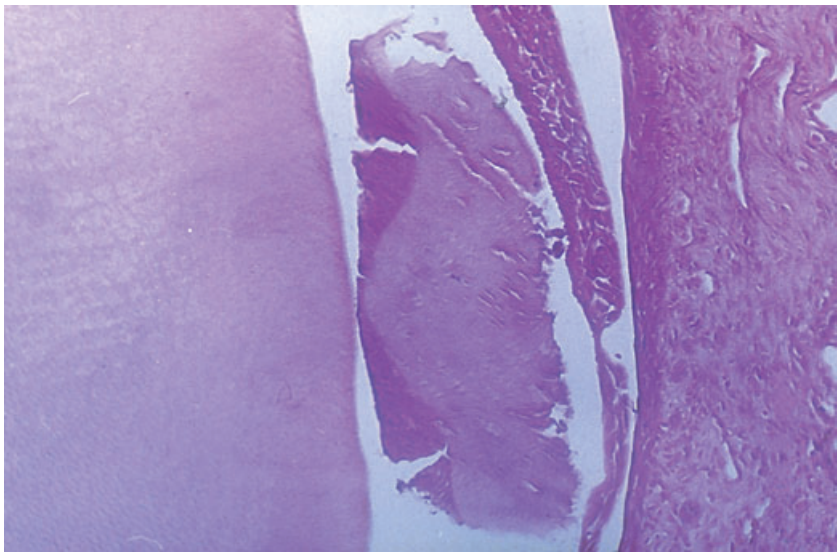

Fig. 5. Portion of non-mineralized matrix (×400).

group 1, 3 showed histological alteration. One was in the group of intrusion without extraction and two in intrusion with extraction of the primary tooth.

Group 2 (60 days postoperative)

Five animals were killed in this group, resulting in 10 permanent central incisors and 10 permanent lateral incisors, with one lost specimen for each type of treatment. In two cases, root remains of the primary teeth were identified. The histological observations were similar to those described in group 1, with the normal development of the tooth bud formation of the teeth in which the primary tooth was intruded and kept in the socket, one specimen showed remains of non-mineralized enamel matrix in the incisal portion of the crown (Fig. 5). In the primary teeth that were extracted, no alteration was evident.

In both groups 1 and 2, all primary teeth reerupted, and there was no case of ankylosis or retention of the intruded or the permanent tooth. The root resorption of the remaining primary teeth appeared physiological. No alteration in the dental papilla or in the permanent teeth root formation was observed.

Discussion

The histological variations described here were considered to be pathological alterations, as they differed in appearance from what the literature shows to be normal in the odontogenesis of dogs

(19–22). They may have occurred as a consequence of the induced traumatic intrusion. We also think that there may be a relationship between the occurrence of the hypoplasic episode and the moment of the trauma, when the deposition and mineralization of the matrix occurred, indicating a strong possibility that the alterations found in this study are a result of the intrusion. As the intrusions were performed in initial stage of odontogenesis, it is natural that complications occur, given that less differentiated cells are more susceptible to alterations.

In the evaluation of group 1, alterations in the inner epithelium of the enamel organ, with morphologically altered ameloblasts and other epithelial cells as well as abnormality in the aspect of the matrix were encountered. This fact might clinically represent, a zone of hypoplasia or hypomaturation of the enamel when the tooth eruption occurs. Facing a trauma that is considered to be intrusive, the buds in which the matrix is thicker and has not begun calcification may yet suffer more intense and extensive injuries to the organization of both enamel and dentin, being more severe in the former. Adjacent to the degenerated ameloblasts, the matrix is also altered, with a granular appearance (23–26). Regarding the deflection of the matrix, it appears that the ameloblasts have been displaced (12, 26).

In studies with monkeys concerning the immediate alterations in the germs of permanent teeth after the intrusion of primary teeth, morphological changes in the enamel matrix seemed possible (12). The pathogenesis is not completely clear, but if a total loss of the ameloblasts occurs there is no regenerative potential. If the damage is partial, then ameloblasts may survive and continue to secrete, and maturation can occur normally. If damage to the ameloblasts occurs during the maturation stage, a hypo-maturated area will develop, in which the thickness of the enamel is normal, but can be perforated under pressure (27). In damage caused in the permanent tooth bud by the displacement of the primary incisor, the root apex traumatizes the tooth follicle of the permanent tooth and causes injury to some of the ameloblasts of the buccal surface of the developing tooth germ (27–29). The already calcified enamel and the enamel-dentin junction do not suffer alteration. If the damage is more severe, there is inflammation and injury to a greater number of ameloblasts and, where the matrix was not mineralized, there will be a hypoplasic area.

In group 2, however, the visible alteration was described as a portion of matrix that would not have been mineralized and would be 'imprisoned' in the most incisal portion of the tooth. Nevertheless, we cannot affirm exactly when such pathology occurred. The histopathogenesis indicates that the trauma would have occurred in the matrix mineralization phase. But as the tooth is already with the cuspid in the mouth, we do not have the epithelial cover of the tooth germ and so we do not have the altered ameloblast layer to support this supposition. The histological condition found in this specimen leads to the occurrence of a deficiency in the mineralization of the matrix in the incisal portion, keeping, therefore, this significant amount of organic material, which was not lost during the histological process. The occurrence of alterations in the final mineralization of the matrix would be because of the disturbances of the ameloblasts, that could not participate in the removal of the organic substances from this tissue (24). The disturbances in the secondary mineralization of the matrix would be the origin of the most frequent sequels (11, 28).

The process of dealing with the intrusion of primary teeth is widely discussed. The first chosen treatment should be to wait for their reeruption, unless the invasion of the developing permanent tooth follicle is detected. In a follow-up of 88 intruded primary teeth, 72 erupted, four were extracted after 14 days because of infection and four did not erupt, being the extraction performed after 12–18 months (29). When the intrusion is of less than half of the crown and there is no evidence of alveolar fracture, it is possible to wait for the reeruption (30). However, if half or more of the crown is intruded, the authors recommend immediate extraction. In a study of intruded primary teeth, 54 reerupted and 20 were removed for complications. In a follow-up of 123 intruded primary incisors, total reeruption occurred in 84% of the completely intrude teeth and in 92% of those that had suffered partial intrusion, showing that the degree of intrusion did not have influence on the tooth's ability to reerupt (31).

We tried to work in the period when the dental age of the dog should correspond to that of the human species when most frequently subjected to trauma. Between 0 and 2 years old is the moment when the incisal third of the crown of the permanent incisors is being formed (8), corresponding to the age of the dogs in days when they were used in the study. Nevertheless, when 50 day old the dogs would have the deposition of enamel initiated up to the middle third of the crown. At the moment of the sacrifice of group 1, the animals were 80 days old and would have all the matrix deposited, except for a small portion in the cervical region, and would not have initiated the root resorption of the primary teeth. According to microscopical analysis, the stages of deposition and mineralization of the enamel and dentin matrices and the aspect of the cells that are responsible for these events agree with the histological literature (18–21). When the

sacrifice of group 2 was performed, the animals were 100 days old, which would correspond to the mineralization of two-thirds of the crown, and the primary teeth would be near to exfoliation. However, in the present study, an acceleration of root resorptin was observed as only a few animals presented with primary teeth. When present, primary teeth showed advanced levels of reabsorption, within physiological levels and without inflammatory reabsorption.

The results of this study in agreement with the literature and our clinical experience, appears suggest no reason to alter what has already been recommended. We feel that extraction should not be the immediate treatment for all intruded incisors, and based on our results there was no perceivable difference between the extraction and the conservative treatment. The indication of waiting for the reeruption, which should occur within 1–6 months, made by the authors (9, 32–34) seems to remain the preferable treatment, based on our results and on the comparison with other authors.

An exception to passive eruption based on the literature would be a radiographic exam showing invasion of the space of the developing tooth bud, or clinical situation such as local infection. Even if the intruded tooth is immediately extracted, the permanent, when erupting, may present some complications, considering that the damage occurs at the moment of impact, which emphasizes the importance of careful diagnosis and periodical follow-up aimed at the prevention of future complications for the developing permanent tooth bud.

Finally, we reaffirm the importance of further studies on the intrusion of primary teeth and its consequences on permanent dentition. The damage may remain subclinical, but still have the same importance, and clinical evaluation should be periodical.

Conclusion

Analyzing the results of the present study, it is concluded that: (i) some dog permanent teeth presented alterations during the formation of the buds, after the traumatic injury of their predecessors; (ii) the alterations did not show direct connection with the maintenance of primary teeth in the socket or their extraction.

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