# Radiographic and Histologic Evaluation of Experimentally Induced Severe Intrusive Luxation of Immature Teeth in Rats

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## ABSTRACT

**Introduction:** The aim of this study was to establish an intrusive luxation model in rats and observe the pulpal and periodontal outcomes. **Methods:** The intrusion was experimentally induced by an application of 20-N force on the occlusal surface of maxillary right second molar along the tooth axial using a striking instrument in 3-week-old male Sprague-Dawley rats. Thirty rats were divided into 6 groups ($n = 5$) and were sacrificed after 3, 7, 14, 30, 60, and 90 days of the surgery. The occurrence of pulpal and periodontal complications was observed by micro–computed tomographic scanning and hematoxylin-eosin staining. **Results:** All experimental teeth were fully intruded into the alveolar bone with their occlusal surface located at the cervical level of the adjacent first molar. Spontaneous re-eruption initiated at 7 days. At 14 days, 4 teeth (80%, 4/5) partially re-erupted, whereas 2 (40%), 3 (75%), and 4 (100%) teeth completely re-erupted at 30, 60, and 90 days, respectively. Pulp degeneration and inflammation mainly occurred in 4 teeth at 3 days, 5 at 7 days, and 2 at 14 days; after 14 days, pulp calcification was observed in 8 teeth. Ankylosis and replacement root resorption mainly occurred in 1 tooth at 30 days, 2 teeth at 60 days, and 3 at 90 days. Marginal bone loss was observed in 3 teeth (60%) at 30 days, 3 (75%) at 60 days, and 2 (50%) at 90 days. **Conclusions:** An animal model of intrusive dentoalveolar trauma was successfully established in rats. Pulpal and periodontal complications similar to clinical tooth intrusion were observed, which provided a basis for exploring the mechanisms of complications in the future. *(J Endod 2020;46:1631–1638.)*

## KEY WORDS

Ankylosis; intrusive luxation; pulp calcification; replacement root resorption

## SIGNIFICANCE

The prognosis of severe intrusive luxation of young permanent teeth was poor, and mechanisms of complications were unknown. This study established an intrusive luxation model in rats. Pulp calcification and replacement root resorption with osteoid tissue were observed during the healing process.

Intrusive luxation, which is defined as displacement of the tooth axially into the alveolar bone[1], is one of the most serious injuries in young permanent teeth. Treatment options vary according to the root development and the severity of intrusion[1]. According to the guidelines of the International Association of Dental Traumatology, teeth with incomplete root formation should first be allowed to re-erupt without intervention. If no movement occurs within a few weeks, orthodontic or surgical repositioning should be initiated. Teeth with complete root formation and intrusion of <3 mm are allowed 2–4 weeks for re-eruption followed by orthodontic or surgical repositioning if no movement is observed. If the tooth intruded by more than 7 mm, it should be repositioned surgically[1]. A poor prognosis usually resulted from mature teeth, severe intrusion, or active repositioning in clinical cases[2,3]. Young permanent teeth have the potential to re-erupt. However, damage to the periodontium and pulpal tissues in severe intrusion usually causes serious complications including inflammatory root resorption, replacement root resorption/ankylosis, pulp calcification, and pulp necrosis. Replacement root resorption (12%–22%)[2,4–6] is the worst complication with regard to healing, and no treatment can arrest this type of resorption. Pulp calcification is another common complication in young teeth even though it was considered as a kind of pulp survival. Until now, the microscopic manifestations and mechanisms of pulpal and periodontal complications are still unknown.

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Because of the low incidence of intrusive luxation (0.3%–2%)[7–11] and the difficulty of obtaining human specimens of permanent teeth after dental trauma, it is hard to carry out relevant clinical researches. Therefore, animal models are invaluable aids in gaining insights into the cellular and molecular mechanisms of the condition and developing new therapeutic approaches. Amanda and Tracy[12] used the farm pig, *Sus scrofa*, as an *ex vivo* intrusion model to show changes of mechanical properties of the periodontal ligament. In their research, a 6-kg load could induce root embedding in the alveolar bone with a mean of 3.81 mm. Cunha et al[13,14] used mongrel dogs to create an animal model of 3-mm intrusion by 4-kg force, whereas Gomes et al[15] applied another device that revealed an intrusive impact energy of 13.26 J in dogs. However, because of the high cost and long experimental period of large animals, researches based on intrusive models were limited. The rat is a commonly used model for studying dental trauma because of its shorter experimental period, low cost, and easier specimen handling. Pereira et al[16,17] induced concussion and subluxation in rats by light force (900 cN) in order to avoid bone and tooth fracture. However, appropriate forces in rats for intrusion were rarely mentioned. The aims of this study were to establish an intrusive luxation model in rats and observe the pulpal and periodontal outcomes.

## MATERIALS AND METHODS

The animal test was approved by the Animal Research Ethics Committee of Peking University Health Science Center, Beijing, China (LA2015212).

### Instrument Design

The power of dentoalveolar trauma was induced by a striking instrument (Fig. 1*Aa*), which was modified from an ear piercer combined with a metal force transfer rod (Fig. 1*Ab*). Within the instrument body (Fig. 1*Ac*), the force was generated by stainless steel springs that amounted on the barrel. By replacing the springs with different diameters, different forces were produced (Fig. 1*Ad*). According to the results of different springs, a combination of 7-mm + 9-mm springs was adapted for inducing complete intrusive luxation of the maxillary second molar in rats. The force produced by 7-mm + 9-mm springs was 20 N, which was measured by a materials testing machine (3356; Instron, Norwood, MA) (Fig. 1*Ae*).

A metal force transfer rod (Fig. 1*Ab*) was set between the instrument body and the molar to deliver the force in order to avoid crown fracture. One end of the force transfer rod, which was pressed on the occlusal surface of the molar, was modeled into a round shape with a 2-mm-diameter surface in accordance with the maxillary second molar. The end of the force transfer rod remains perpendicular to the occlusal surface of the second molar when hitting to ensure the impact force is transferred along the long axis of the tooth. A piece of 50-mm-thick sponge was used under the head of animals in order to protect the brain from injury.

### Animals

Thirty 3-week-old male Sprague-Dawley rats with an average weight of 69.28 g obtained from Beijing Vital River Laboratory Animal Technology Co, Ltd, Beijing, China, were used. The animals were housed in plastic cages (5 rats in 1 cage, fed and drunk freely) under climate-controlled conditions (12-hour light/12-hour dark cycle, thermostatically regulated room temperature) and were fed a standard solid chow and water *ad libitum*.

### Experimental Protocol

All experimental procedures were performed under anesthesia with an intraperitoneal injection of 2% sodium pentobarbital (0.25 mL/100 g body weight). The dentoalveolar trauma (intrusion) was experimentally induced by an application of the striking instrument on the occlusal surface of maxillary right second molar (Fig. 1*Ba–d*). The maxillary left second molar was used as the control. Animals were secured on a piece of sponge during surgery to diminish the injury to the brain. The animals were divided into 6 groups ($n = 5$) and were sacrificed after 3, 7, 14, 30, 60, and 90 days of the surgery, respectively.

### Micro-computed Tomographic Analysis

The specimens were scanned using a micro–computed tomographic scanner (GANTRY-STD CT 3121; Siemens, Knoxville, TN) with 80 kV, 500μA, a pixel size of 33.65 μm, a camera exposure time of 1200 milliseconds, and 360° rotation around the vertical axis with a 1° rotation step. The aluminum filter was set at 0 mm during the scans. Raw data obtained in scanning stage were reconstructed using the Inveon Research Workplace 4.2 software (Siemens).

The degree of tooth re-eruption, pulp calcification, ankylosis, replacement root resorption, and marginal bone loss in 2-dimensional and 3-dimensional image constructions were observed. The definitions of the observed outcomes are as follows:

1. Degree of tooth re-eruption: the degree of tooth re-eruption was divided into 3 categories: complete re-eruption, partial re-eruption, and no re-eruption. A completely re-erupted tooth was when the occlusal surface of the intrusive tooth reached the occlusal level of the first molar. A no re-erupted tooth referred to those whose occlusal surface was still at the cervical level of the first molar, and partially re-erupted referred to a state between completely re-erupted and no re-erupted.
2. Pulp calcification: radiopaque in the pulp cavity and root canal
3. Ankylosis: the loss of periodontium space and the integration of acellular cementum and alveolar bone
4. Replacement root resorption: the root resorbed and replaced with bonelike tissue
5. Marginal bone loss: the marginal bone in the buccal side of the intruded tooth healed but recessed; marginal bone loss was evaluated from 30 days after injury.

### Histologic Evaluation

The maxillae of all samples were removed and fixed in 4% paraformaldehyde, decalcified in 10% EDTA solution at 37°C for 6–8 weeks followed by dehydration, embedded in paraffin, and sectioned at 5 μm by a Leica microtome (Leica Instruments GmbH, Hubloch, Germany). Sections were evaluated histologically by hematoxylin-eosin staining and Masson trichrome staining (Solarbio, Beijing, China) according to the manufacturer's protocol. The stained slides were observed by light microscopy (Olympus, Tokyo, Japan). In hematoxylin-eosin–stained images, manifestations of pulp and periodontal tissues were observed at a magnification of 4, 10, 20, and 40×.

## RESULTS

### Establishment of Animal Model for Intrusion

All teeth were fully intruded into the alveolar bone with mobility and the occlusal surface located at the cervical level of the adjacent first molar. Micro–computed tomographic imaging showed that immediately after injury, the occlusal surface of the second molar was lower than the adjacent teeth accompanied by narrowing of the periodontal space and fracture of the buccal alveolar bone (Fig. 1*Be*). Three rats (3/30) died during the recovery process as a result of brain damage, excessive bleeding, or asphyxia. Exophthalmos and cyanosis of limbs were observed in these 3 rats. The rest of the experimental teeth ($n = 27$) were followed up and examined at 3 ($n = 4$), 7

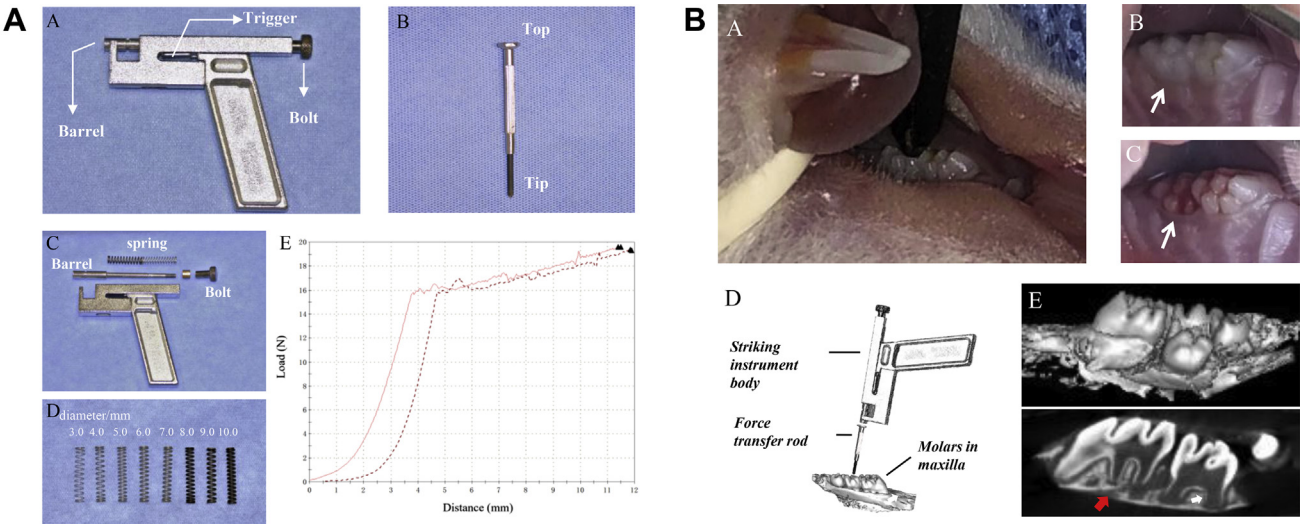


**FIGURE 1 –** The experimental design. (*A*) The striking instrument. (*Aa*) The instrument body. (*Ab*) The metal force transfer rod. (*Ac*) Within the instrument body, the force was generated by stainless steel springs that amounted on the barrel. (*Ad*) Stainless steel springs with different diameters. (*Ae*) The force of 7-mm + 9-mm springs was 20 N measured by the materials testing machine. (*B*) The establishment of the intrusion model in rats. (*Ba*) The rat was secured on a piece of sponge and had the upper right second molar exposed. (*Bb*) Before the hit. (*Bc*) Intruded after the hit. (*Bd*) The scheme of the hitting process. (*Be*) The micro-CT scan showed the position of the second molar was lower than the adjacent teeth accompanied by narrowing of the periodontal space (*white arrow*) and fracture of the buccal alveolar bone (*red arrow*).

($n$ = 5), 14 ($n$ = 5), 30 ($n$ = 5), 60 ($n$ = 4), and 90 days ($n$ = 4). The mean weight (g) at the end of each experimental period was 78.75 ± 4.79, 104.00 ± 11.40, 184.00 ± 29.45, 299.00 ± 30.29, 425.00 ± 28.58, and 505.00 ± 51.96, respectively. All remaining animals were alive with no systemic symptoms.

## Spontaneous Re-eruption and Root Development of Injured Teeth

Tooth re-eruption initiated at 7 days after intrusive luxation. At 14 days, 4 teeth (80%, 4/5) partially re-erupted to the occlusal level of the maxillary right first molar (Fig. 2*A–C*). At 30 days, 2 teeth (40%, 2/5) completely re-erupted (Fig. 2*D–F*), whereas the other 3 did not re-erupt (Fig. 2*G–I*). Data of tooth re-eruption at different time points are shown in Table 1. The second molar of 3-week-old rats had two-thirds root formation and the apex closed at 30 days after injury in teeth with complete or partial re-eruption (Fig. 2*E* and *F*), whereas in teeth that did not re-erupt root development arrested with an open apex and pulp calcified in 30 days (Fig. 2*I*).

## Pulp Complications

Pulp degeneration, inflammation, and calcification were observed after injury (Table 2). Pulp necrosis and inflammatory root resorption were not observed. Dental pulp degeneration and inflammation occurred at the early stage (3rd–14th day) of trauma and mainly manifested as vacuolar degeneration of odontoblasts and fibroblasts, reticular atrophy of pulp, pulp fibrosis, and lymphocyte infiltration (Fig. 3*B–D*). In the control tooth, the pulp chamber and the root canal were mainly composed of stellate cells and a homogenous extracellular matrix (Fig. 3*A*).

From day 14 after injury, the calcification of pulp appeared, and the area of calcification increased gradually on days 30 and 60. Pulp calcification occurred in 4 no re-erupted, 3 partially re-erupted, and 1 completely re-erupted teeth. In addition, calcification was composed of osteoid tissue with trabecular bone, bone lacunae, and the Harvard system (Fig. 3*E*, *F*, *I*, and *J*) which was similar to the bone tissue in furcation area of the control tooth (Fig. 3*G* and *H*).

## Periodontal Complications

From the 3rd to 30th day of trauma, tissue degenerations were observed in the apical region, including squeezed cells, vacuolar degeneration of cells, infiltration of inflammatory cells, and calcification in root canals.

Irregular proliferation of cellular cementum occurred on day 14, which was mainly manifested by an increase of thickness and abnormal morphology of cellular cementum (Fig. 4*Aa–d*). On the 30th day, ankylosis and replacement root resorption were observed. Ankylosis mainly occurred at the root furcation area (Fig. 4*Ae–h*). Replacement root resorption mainly occurred at the apical area or involved the entire root with osteoid tissue deposition (Fig. 4*Ai–l*) and irregularly arranged collagen fibers and fibroblasts (Fig. 4*Ao* and *Ap*) compared with the control tooth (Fig. 4*Am* and *An*). Occurrence of periodontal complications are shown in Table 3.

Bone repair of alveolar fracture initiated on day 7. From days 7 to 30, the marginal alveolar bone in the buccal side of intruded tooth healed and thickened gradually. At 30, 60, and 90 days, marginal bone loss occurred in 3 (60%), 3 (75%), and 2 (50%) teeth, respectively. In those teeth, 4 had no re-eruption, 3 had partial re-eruption, and 1 had complete re-eruption. No re-erupted teeth showed marginal bone loss much severer than those with partial or complete re-eruption manifested as a deep collapse of the buccal alveolar bone of the second molar (Fig. 4*Ba–Bd*).

# DISCUSSION

This study introduced a method to establish intrusive dentoalveolar trauma to molars of rats. The advantage of this method is the standardization of strength and direction of the force applied and root development (two-thirds root formation) so that the incidence, severity, and reproducibility of intrusion are guaranteed. Our results showed that teeth fully intruded into the alveolar bone with their occlusal surface located at the cervical level of the first molar and with alveolar fracture resembled a severe intrusion (>7 mm) in clinics. All teeth were without crown fracture and waited for re-eruption without any intervention to observe the natural healing

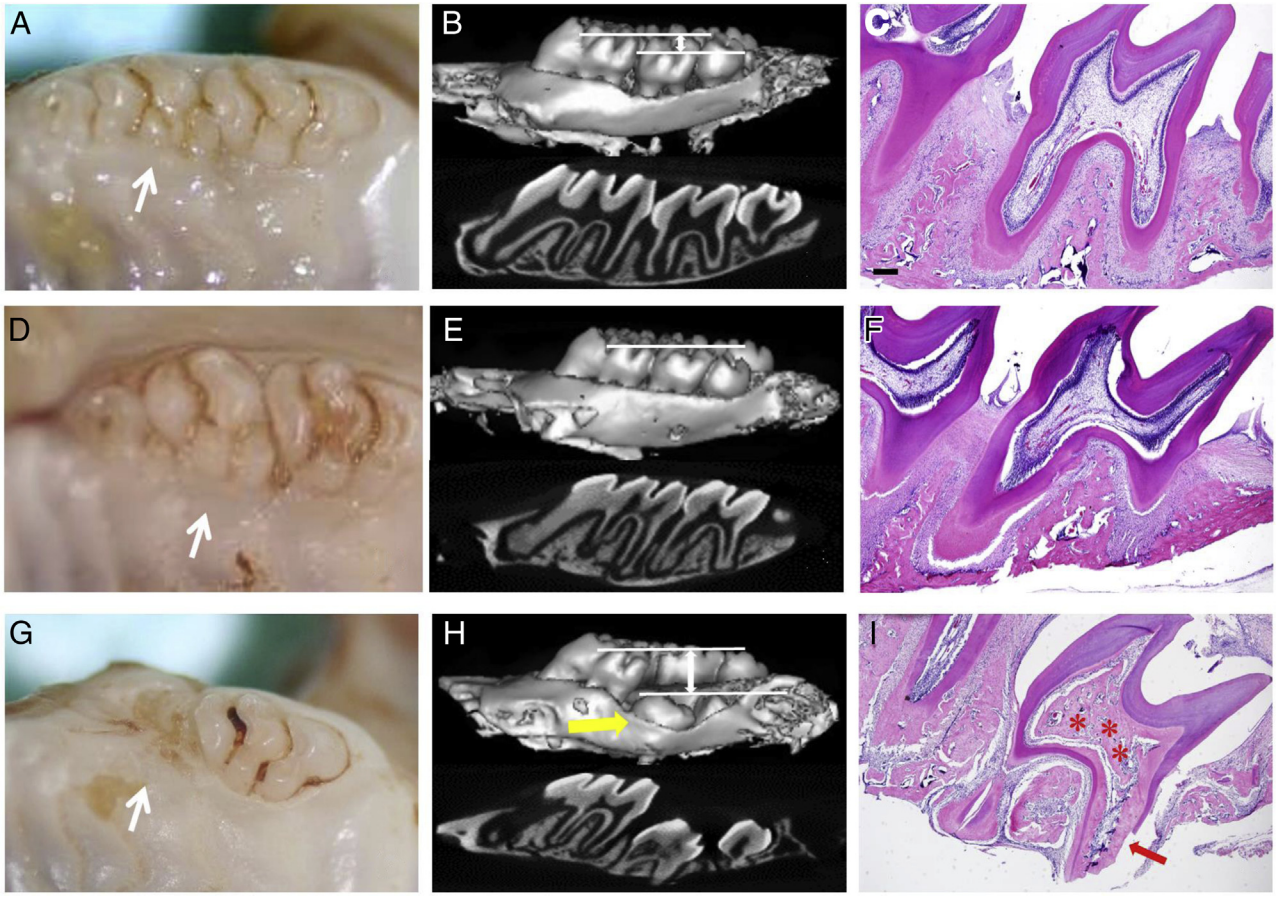


**FIGURE 2 –** The degree of re-eruption of an intruded upper second molar. (*A* and *B*) On the 14th day, intraoral examination and 3-dimensional reconstruction of micro–computed tomographic imaging showed a partially re-erupted tooth. (*C*) Hematoxylin-eosin staining showed a normal pulpal and periodontal structure, (4×). (*D–F*) On the 30th day, the tooth was completely re-erupted with normal pulpal and periodontal tissues. (*G–I*) On the 30th day, the tooth was ankylosed with pulp calcification (*red **), an open apex (*red arrow*), and marginal bone loss (*yellow arrow*). (*C*, *F*, and *I*) Hematoxylin-eosin staining: ×4. (*C*, *F*, and *I*) Scale bar: 200μm.

process. Because of the multiple roots of the second molar and the thin alveolar process, intrusive luxation of rats in this study was mainly combined with buccal alveolar fracture according to micro–computed tomographic results (Fig. 1). This is a limitation of using a rodent model to simulate intrusive dental trauma. The healing process in this animal model involved not only pulpal and periodontal tissue recovery but also bone repair and regeneration. For studying the specific healing process of pulpal or periodontal tissue, the method should be improved in future studies. For instance, the springs could be more specifically designed to adjust the force, and mechanical devices could be applied to modify the direction and movement of the transfer rod. In addition, the metal transfer rod could be replaced by a wooden rod or other materials to exert a blunt force, which might reduce the risk of bone fracture.

In this study, the maxillary second molar was selected to perform intrusive luxation. The reasons are as follows. First of all, intrusive luxation in clinics is more likely to occur in maxillary teeth. The bone density, structure, and blood supply around the roots of molars are different between the maxilla and mandible. The roots of mandibular molars are more likely to fracture because they are very close to the long incisor roots. Injuries in maxillary teeth could better simulate the situation of dental trauma in clinics. Second, the vision of rat's maxillary molars was clear so that it was easier to operate. The limited mouth opening and thinner and delicate alveolar bone made mandible molars difficult to handle. However, compared with lower teeth, the force applied on upper teeth was more likely to cause brain damage or asphyxia. There was 10% loss of animals because of brain damage or asphyxia (blood flowing backward to the lung) in this study. Therefore, head protection using a thick sponge and intraoral hemostasis were necessary preventive measures.

Working with growing young animals (3 weeks old) might bring systemic influences during the long experimental period. In this study, before and after injury, the rats were raised in a specific pathogen-free animal laboratory with appropriate temperature, humidity, and light and fed and drunk freely. Even though 5 rats were raised in 1 cage, the body weight of rats grew evenly with a small standard deviation within each group. Furthermore, all remaining animals were alive with no systemic symptoms. Therefore, the

**TABLE 1 -** The Occurrence of Tooth Re-eruption at Different Time Points

| Group | 3 d ($n = 4$) | 7 d ($n = 5$) | 14 d ($n = 5$) | 30 d ($n = 5$) | 60 d ($n = 4$) | 90 d ($n = 4$) |
|---|---|---|---|---|---|---|
| Complete re-eruption | 0 | 0 | 0 | 2 | 1 | 3 |
| Partial re-eruption | 0 | 1 | 4 | 0 | 2 | 1 |
| No re-eruption | 4 | 4 | 1 | 3 | 1 | 0 |

**TABLE 2 -** The Occurrence of Pulp Complications at Different Time Points

| Group | 3 d ($n = 4$) | 7 d ($n = 5$) | 14 d ($n = 5$) | 30 d ($n = 5$) | 60 d ($n = 4$) | 90 d ($n = 4$) |
|---|---|---|---|---|---|---|
| Pulp degeneration | 3 | 4 | 1 | 0 | 0 | 0 |
| Pulp inflammation | 1 | 1 | 1 | 0 | 0 | 0 |
| Pulp calcification | 0 | 0 | 2 | 3 | 3 | 0 |

differences of body growth within each group could be neglected.

Although the experimental method needs to be improved, we have found some interesting results of pulpal and periodontal tissue changes. In research performed by Cunha et al[13], young permanent intruded incisors of dogs re-erupted to the original position in the dental arch in 30 days when apical closure had occurred. In this study, teeth re-erupted since 7 days after injury and completely re-erupted to the occlusal level of the first molar in 2 teeth (40%) with apical closure at 30 days as well. At this time point, the other 3 teeth (60%) with root canal calcification, arrested root development, and an open apex did not re-erupt. This suggested that the potential of re-eruption reduced when periapical tissue was injured and root development arrested. Pulp necrosis and inflammatory root resorption were not observed in specimens, which indicated that teeth with an immature root and an open apex had greater potential for pulpal and periodontal healing. However, a larger sample size was needed to observe and identify the incidence of spontaneous re-eruption and pulpal and periapical complications.

Dental pulp calcification occurs mainly in young permanent teeth[18,19]. In clinical intrusion studies, Tsilingaridis et al[2] and Wigen et al[4] reported that 20.4%–35.3% of intruded teeth developed pulp obliteration, and the pulp preservation rate of young permanent teeth with intrusion was 43%, 82% of which had pulp calcification during the follow-up period. In this study, the incidence of calcification from 14 to 60 days was 40%–75%, which was similar to the clinical data. The calcification component in this study was not a calcified nodule as denticle or diffused calcification in age-related changes[20] and pathologic manifestations[21]. It was more like an osteogenic structure with bone trabecular, osteoblasts, osteoclasts, lacunae of bone, and the Harvard system. In 2000, Heling et al[22] reported 5 cases of bonelike tissue growing in the root canal of immature permanent teeth after traumatic injuries with absence of infection. However, relevant studies about the exact component and mechanism of pulp

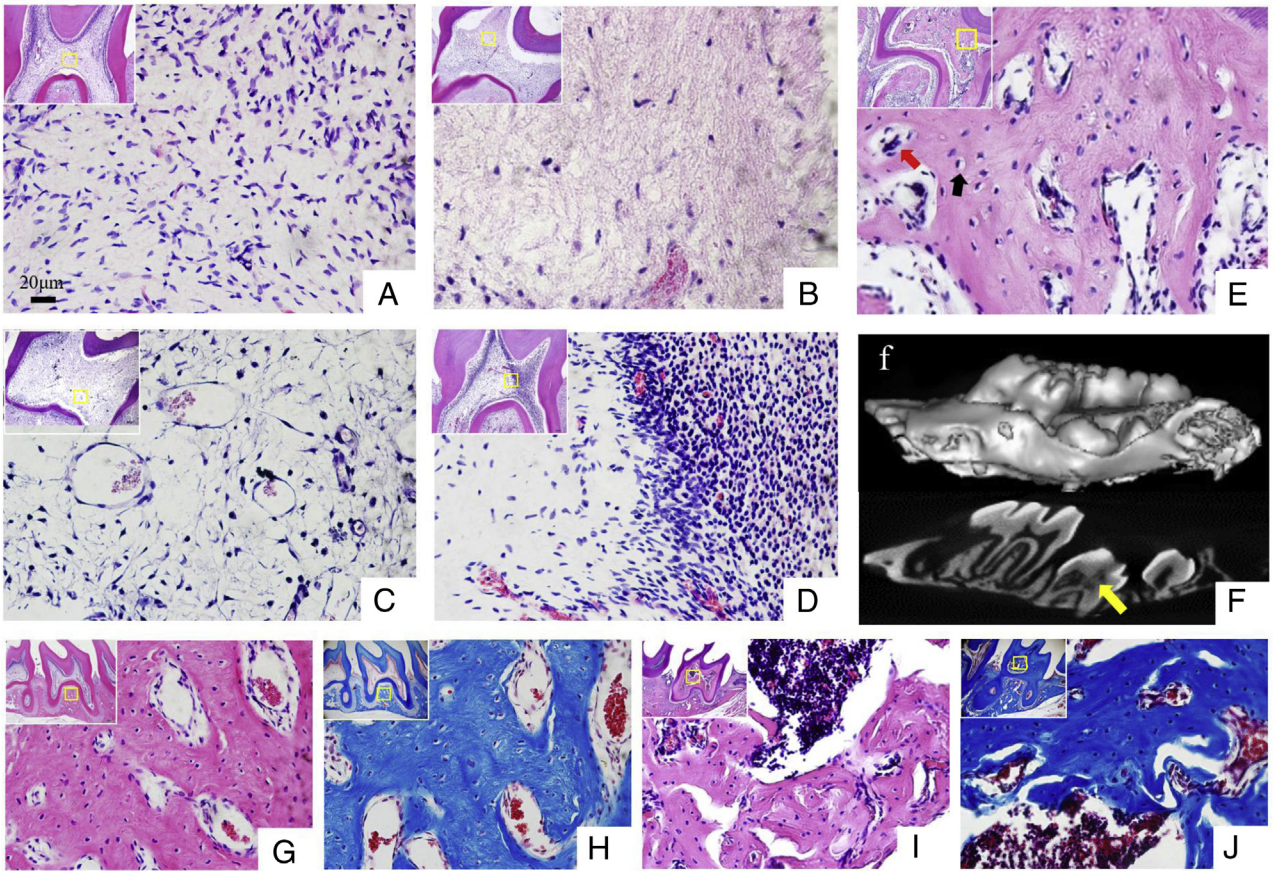


**FIGURE 3 –** Histologic and micro–computed tomographic manifestations of pulp complications after intrusion. (*A*) Normal pulp. (*B*) Pulp fibrosis. (*C*) Reticular atrophy. (*D*) Inflammatory cell infiltration in the pulp cavity and root canals. (*E*) Pulp calcification with osteoblastlike cells (*black arrow*) and osteoclastlike cells (*red arrow*). (*F*) Micro–computed tomographic imaging showed pulp calcification (*yellow arrow*). (*G* and *H*) Bone tissue in the furcation area of a normal tooth. (*I* and *J*) Pulp calcification with osteoid tissue in the pulp cavity of an injured tooth. (*A–E*, *G*, and *I*) Hematoxylin-eosin staining: ×40. (*H* and *J*) Masson trichrome staining ×40. (*A–E*, *G*, *H*, and *I*) Scale bar: 20μm.

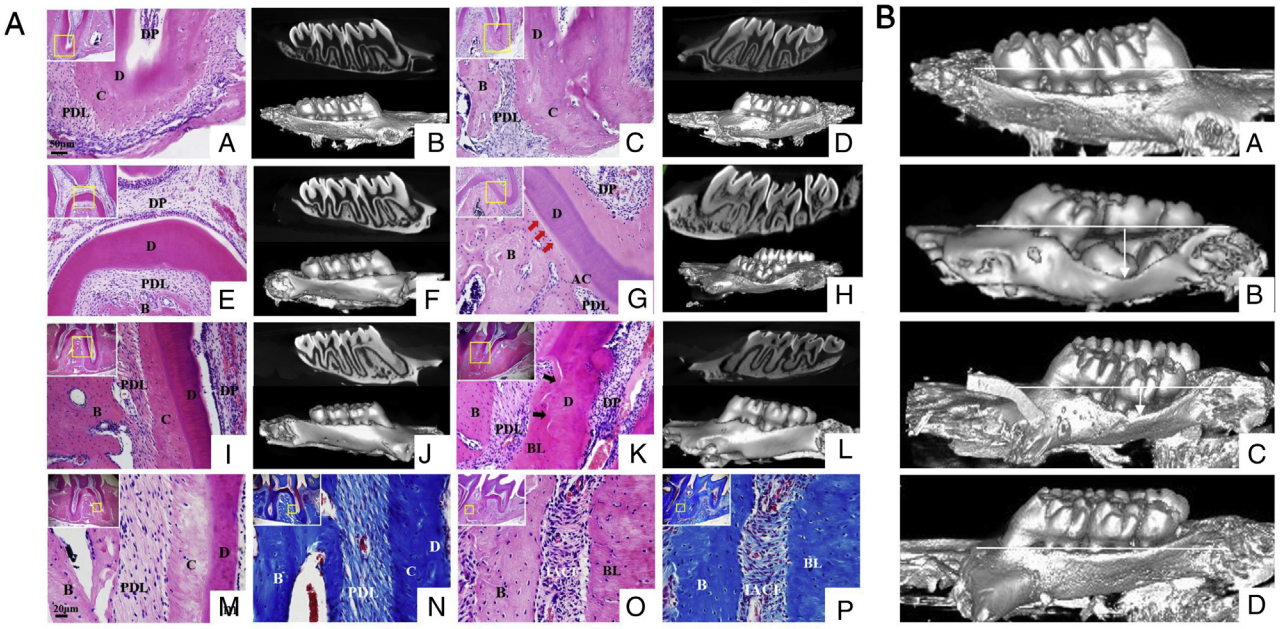


**FIGURE 4 –** (*A*) Histologic and micro–computed tomographic findings of periodontal tissues after intrusion. (*Aa* and *Ab*) Normal cellular cementum in the periapical area. (*Ac* and *Ad*) Irregular proliferation of cellular cementum. (*Ae* and *Af*) Normal tissues at furcation of the upper second molar. (*Ag* and *Ah*) Ankylosis with loss of periodontal ligament space (*red arrows*). (*Ai* and *Aj*) Normal cementum and dentin in the middle area of the root. (*Ak* and *Al*) Replacement root resorption (*black arrows*). (*Am* and *An*) Normal periodontal ligament. (*Ao* and *Ap*) Irregularly arranged collagen fibers (IACF). (*Aa*, *Ac*, *Ae*, *Ag*, *Ai*, and *Ak*) Hematoxylin-eosin staining: ×20, scale bar: 50μm. (*Am* and *Ao*) Hematoxylin-eosin staining: ×40, scale bar: 20μm. (*An* and *Ap*) Masson trichrome staining: ×40, scale bar: 20μm. AC, acellular cementum; B, bone; BL, bonelike tissue; C, cementum; D, dentin; DP, dental pulp; PDL, periodontal ligament. (*B*) Micro–computed tomographic 3-dimensional reconstruction of marginal bone loss at 30 days. (*Ba*) Normal marginal bone level of the control upper left second molar. (*Bb*) Severe marginal bone loss in teeth with no re-eruption (*white arrow*). (*Bc*) Moderate marginal bone loss in teeth with partial re-eruption (*white arrow*). (*Bd*) Mild marginal bone loss in teeth with complete re-eruption.

**TABLE 3 -** The Occurrence of Periodontal Complications at Different Time Points

| Group | 3 d ($n = 4$) | 7 d ($n = 5$) | 14 d ($n = 5$) | 30 d ($n = 5$) | 60 d ($n = 4$) | 90 d ($n = 4$) |
|---|---|---|---|---|---|---|
| Degeneration of periapical tissue | 3 | 2 | 2 | 2 | 0 | 0 |
| Irregular hyperplasia of cementum | 0 | 0 | 3 | 3 | 1 | 2 |
| Ankylosis | 0 | 0 | 0 | 1 | 0 | 1 |
| Replacement root resorption | 0 | 0 | 0 | 0 | 2 | 2 |
| Marginal bone loss | — | — | — | 3 | 3 | 2 |

calcification after dental trauma in young permanent teeth were still unknown and need to be explored. The intrusive luxation model in this study provided a basis for future research.

In the periodontal healing process, after degeneration of periapical tissues and irregular proliferation of cementum, ankylosis and replacement root resorption occurred from 30 days after injury. Ankylosis occurred in teeth with no re-eruption, whereas replacement root resorption occurred in fully re-erupted teeth. In clinics, ankylosis/replacement root resorption occurred in dental trauma with a necrotic periodontal membrane, mainly including intrusion and avulsion. In early research in dogs performed by Cunha et al in 2002[14], replacement root resorption of intrusive luxation developed after loss of periodontal ligament and contact of cementum and alveolar bone. In this study, irregularly arranged collagen fibers and fibroblasts were observed around the replacement root resorption area. This indicated that the periodontal ligament might play an important role in the deposition of calcified tissue during replacement root resorption. Further studies about the mechanisms of this process were needed. During the healing process of alveolar bone fracture, thickness rather than height of the buccal marginal bone increased, resulting in marginal bone loss. Andreasen et al[3] reported that marginal bone loss had a significant relationship to root development and gingival laceration in clinics. In this study, most intruded young teeth were with bone fracture, and the incidence of marginal bone loss was high (60% in 30 days, 75% in 60 days, and 50% in 90 days, respectively), indicating that marginal bone loss might relate to alveolar fracture.

From the occurrence of complications, we have basically established an traumatic intrusive luxation model of young permanent teeth in rats, which can be further studied for the mechanisms of pulp calcification and replacement root resorption and used for randomized controlled study for the treatment of intrusion. In the future, a mice model of intrusive luxation could be attempted by modifying the transfer rod so that knockout animals could be used to analyze the signaling pathways of traumatic healing process.

In conclusion, an animal model using striking instruments to induce severe intrusive trauma in young rat molars was successfully established. Various complications similar to clinical tooth intrusion such as pulp calcification and replacement root resorption were observed, which provided a basis for further exploration of the mechanism of complications.

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*The authors deny any conflicts of interest related to this study.*

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