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PRACTICE

Ankylosis of Traumatized Permanent Incisors: Pathogenesis and Current Approaches to Diagnosis and Management

Karen M. Campbell, DDS, MSc, FRCD(C); **Michael J. Casas,** DDS, DPaed, MSc, FRCD(C);
David J. Kenny, BSc, DDS, DPaed, PhD, FRCD(C)

Contact Author

Dr. Campbell
E-mail:
roy.campbell4@sympatico.ca

ABSTRACT

Ankylosis is a known complication of replanted or severely intruded permanent incisors and can be diagnosed by the characteristic sound emitted when the tooth is tapped. The ankylosed incisor demonstrates a lack of physiologic mobility and, later, radiographic evidence of replacement resorption. If the patient is pre-adolescent or adolescent at the time of trauma, infraocclusion relative to adjacent teeth will become apparent during jaw growth. Despite considerable knowledge about the pathogenesis of ankylosis garnered from animal studies and observation of human replanted teeth, there is no known treatment to arrest this condition. Management techniques and rehabilitation options for addressing ankylosis and its consequences are supported by little evidence, do not appear to be widely adopted and do not offer any proven long-term benefit. Avulsion and severe intrusion of permanent incisors are rare injuries. Should the decision be made to intervene by replantation or reduction of the intrusion, the clinician must be prepared to diagnose ankylosis, identify its negative consequences and develop treatment plans accordingly.

Ankylosis is a pathologic fusion of the cementum or dentin of a tooth root to the alveolar bone.[1] It is most likely to affect a replanted avulsed tooth or a severely intruded tooth (i.e., intrusion greater than 6 mm or half the clinical crown length[2] within weeks following trauma.[3–9] Risk of ankylosis is highest in this subset of luxation injuries because of the nature and severity of damage to the root-side periodontal ligament. Ankylosis and replacement resorption are largely responsible for the low 5-year survival of teeth after these injuries.[10,11]

Detection of ankylosis depends on clinical signs and radiographic interpretation (**Fig. 1**). Clinical diagnosis of ankylosis is based on qualitative assessment of the sound produced on percussion and of mobility. Ankylosis of teeth in the pre-adolescent can dramatically alter local growth and development of the alveolus.[12] The time at which these effects become clinically significant depends on patient age and stage of growth and development. Progressive infraocclusion and distortion of the gingiva and underlying bone produce both functional and esthetic deficits with jaw growth (**Fig. 2**). Early detection of ankylosis does not change the inevitable outcome: tooth loss from replacement resorption. In fact, the only benefit of early detection is that the clinician will have earlier warning of growth-associated infraocclusion. If the

Figure 1: Radiographic appearance of advanced replacement resorption in a 17-year-old female. Teeth 11 and 21 were replanted at age 15 years.

Figure 2: Tooth 11, which was avulsed and replanted when the patient was 7.5 years of age, developed subsequent ankylosis. Note the infraocclusion of tooth 11, space loss and distortion of the gingival architecture in the maxillary anterior segment at 10.25 years.

Figure 3a: Clinical appearance of a 10-year-old at 16 weeks following replantation of teeth 11 and 21. Both incisors were replanted after 180 minutes of extraoral exposure and received endodontic treatment within 14 days. Ankylosis was confirmed in both central incisors at 16 weeks post-injury by the characteristic percussion sound and lack of physiologic mobility.

Figure 3b: Clinical appearance of the same patient at 57 weeks post-injury. The esthetic effects of infraocclusion appear minimal despite the well-established ankylosis. Infraocclusion is expected to become increasingly apparent as pubertal growth accelerates.

Figure 3c: Radiographic appearance of the affected teeth at 57 weeks post-injury. Note the lack of periodontal ligament space and replacement resorption in the apical half of tooth 21.

patient is a pre-adolescent or an adolescent, early diagnosis will facilitate the timing of appropriate interventions that may produce less morbidity and are associated with better long-term outcomes.

Pathogenesis of Ankylosis

Current knowledge of the pathogenesis of ankylosis is based largely on findings from animal and in vitro studies and observations from human studies of replanted teeth. In healthy patients, abundant periodontal ligament fibroblasts block osteogenesis within the periodontium by releasing locally acting regulators, such as cytokines and growth factors, thereby maintaining separation of tooth root from alveolar bone.[13] Necrosis of the periodontal ligament's cellular elements by desiccation, crushing or mechanical damage, as in severe luxation injury, disrupts this normal homeostatic mechanism. Ankylosis is established not only via inflammatory-mediated and mechanical alterations in the periodontal ligament,[8] but also because insufficient functional cellular elements survive to suppress osteogenic activity.[14] This disruption allows growth of bone across the periodontal ligament and ankylosis (fusion of the tooth root and alveolar bone).

Ankylosis is most common following delayed replantation or severe intrusion (**Figs. 3** and **4**). These are catastrophic dentoalveolar injuries as they create significant

Figure 4a: Clinical appearance of a 12-year-old at 9 weeks after severe intrusion of teeth 11 and 21. Both incisors were intruded their full crown length (12 mm) and underwent immediate surgical repositioning. Ankylosis was confirmed in both central incisors at 9 weeks by characteristic percussion sound and lack of physiologic mobility.

Figure 4b: Clinical appearance of this patient at 42 weeks post-injury. As in the patient shown in **Figs. 3a** and **3b**, the esthetic effects of infraocclusion are minimal at this time. However, it is expected that infra-occlusion and replacement resorption will progress with the onset of puberty.

Figure 4c: Radiographic appearance of the affected teeth at 42 weeks post-injury. Replacement resorption is present but minimally evident in both teeth 11 and 21. Calcium hydroxide dressings remain in both canals.

damage to the periodontal ligament and pulp. Tooth avulsion can lead to root-side cell necrosis due to desiccation or improper storage. The root surface sustains mechanical damage from the avulsion force, impact or mishandling. In contrast, the periodontal ligament of the severely intruded tooth is crushed as it is driven into the alveolar bone of the socket. The resultant compression produces ischemia in the periodontal ligament, apical vascular bundle and alveolus. The cementum is sheared from the root surface. The most severe intrusions exhibit no mobility and, therefore, are unlikely to be successfully repositioned with orthodontic traction alone.[15] Clinicians either surgically reposition and splint the tooth or provide immediate orthodontic traction after mobilizing the tooth to decompress the tissues and ensure access for prompt pulpal extirpation. Both avulsion and severe intrusion, therefore, can cause massive cell death within the periodontal ligament and mechanical damage to the root cementum. The probability that ankylosis will develop in the replanted tooth approaches 100% as extraoral exposure time increases.[6] The probability that ankylosis will develop in an intruded tooth increases with severity of intrusion.[11,16]

Animal studies have demonstrated that ankylosis is likely to occur if periodontal ligament damage permits endosteal progenitor cells from the adjacent bone marrow to repopulate the defect rather than root-side periodontal ligament progenitor cells.[3,17] As part of the repair process, adjacent endosteal progenitor cells migrate to the defect under the influence of cell-signaling mechanisms within the periodontal ligament. Although these cells are capable of differentiation into all periodontal ligament cell types (i.e., fibroblasts, cementoblasts, osteoblasts), the phenotype that will repopulate the wound is largely determined by interaction between or absence of locally acting regulators.[14] In vitro studies have illustrated the susceptibility of root-side progenitor cells of exarticulated human teeth to desiccation and their fragility when subjected to prolonged extraoral storage or even to storage in chilled tissue culture media.[18–20] Although some root-side progenitor cells retain their vitality after injury, they lose the ability to differentiate into functional fibroblasts. Rather, differentiation preferentially produces cells capable of osteogenesis and osteoclasis[21] favouring ankylosis over periodontal ligament regeneration.

Histologic studies in animals have determined that at least 20% of the root surface must be attached to adjacent bone before a lack of mobility and the characteristic percussion sound can be detected.[1] Ankylosis initially favours the labial and lingual root surfaces,[8] which explains why it is difficult to detect radiographically in its early stages.

Observational studies of replanted human teeth have also contributed to the understanding of ankylosis, the most common periodontal ligament complication following replantation.[6] The single most important factor affecting the prognosis of the replanted tooth is extra-alveolar time (i.e., immediate replantation minimizes negative periodontal ligament outcomes).[6] Inflammatory resorption sustained by bacterial infection of necrotic pulp tissue in the replanted or severely intruded tooth can be effectively arrested by pulpectomy followed by calcium

Figure 5: The Periotest: an instrument for quantification of tooth mobility that has been applied to the study of ankylosis. A low Periotest value, indicating less mobility, is not diagnostic for ankylosis. The reliability of this sophisticated device in this context is considerably less than the accuracy of the human ear in detecting ankylosis by simply tapping the tooth.

hydroxide root canal filling.[22] However, despite the ability to treat inflammatory resorption predictably, its arrest promotes replacement resorption.

With the loss of periodontal ligament homeostasis and subsequent ankylosis, replacement resorption ensues. The root is gradually replaced by bone as part of normal turnover of the body's skeletal mass. In the young child, the combined effect of a higher metabolic rate that promotes replacement resorption and the lack of root mass in the immature tooth produces tooth loss within a few years.[23] Ankylosis diagnosed before the age of 10 years or before pubertal growth carries a high risk of severe infraocclusion. This is accompanied by distortion of the gingiva and supporting alveolar bone due to localized arrest of growth of the alveolar process.[12,23] In contrast, the skeletally mature patient who sustains a similar injury experiences a much slower rate of replacement resorption with minimal infraocclusion and may retain the replanted tooth for decades.[24]

Diagnosis of Ankylosis

It is accepted practice to use assessment of mobility and percussion sound to detect ankylosis early. Tooth mobility can be evaluated by observing the extent of tooth movement during luxation in a labial–lingual direction. The Miller index is most commonly used to measure tooth mobility, but its dependence on the interpretation and experience of the examiner limits its reliability. Over the past century, a variety of quantitative methods of assessing

tooth mobility have been developed.[25] Of these, Mühlemann's macroperiodontometer was used most often for clinical research.[25] Although considered highly reliable, it was too complex and time consuming to be clinically useful.[26] Later, instruments developed to quantify the stability of endosseous implants were recruited to diagnose ankylosis. The Periotest (Siemens/Medizintechnik, Bensheim, Germany) (**Fig. 5**) and the Osstell (Integration Diagnostics AB, Göteborg, Sweden) are both commercially available, but only the Periotest has received clinical attention. Despite problems with error readings, unit malfunction and test–retest reliability,[27–29] the Periotest was presumptively applied as the sole diagnostic criterion for extraction of ankylosed permanent incisors in one clinical investigation.[30] Recently, it has been verified that the Periotest can confirm a diagnosis of ankylosis by comparison with intact incisors, but a low Periotest value alone cannot be considered diagnostic for ankylosis.[31]

An ankylosed tooth produces a characteristic high-pitched sound[1,32] on percussion, compared with adjacent unaffected teeth. Past investigations using sound analysis of tapped teeth have focused on healthy teeth or those affected by malocclusion and periodontal disease.[32,33] Recently, digital sound wave analysis has confirmed that, in an ankylosed incisor, a significantly higher proportion of the sound energy produced by percussion lies in the higher frequency bands, corroborating the characteristic sound.[31] The simplest diagnostic test — subjective assessment of the sound from percussing the tooth with a metal dental mirror handle — is both highly specific and sensitive for the diagnosis of ankylosis.[31]

Radiographic examination is considered to be of limited value in the early detection of ankylosis because of the 2-dimensional nature of the image. The initial location of ankylosis is often on the labial and lingual root surfaces, complicating radiographic detection[34,35] (**Figs. 3c** and **4c**). The observation of progressive infraocclusion during adolescent growth is another late indicator of ankylosis.[6]

Current Management Options for the Ankylosed Incisor

Adults, with their slower rate of replacement resorption,[24] may retain an ankylosed tooth for many years with minimal treatment or minor cosmetic modifications. A number of increasingly invasive interventions have been advocated for growing individuals where ankylosis may produce significant local alveolar distortion. Early extraction followed by a series of transitional prostheses, intentional luxation and surgical repositioning, decoronation (crown amputation), intentional replantation with Emdogain (Biora, Malmö, Sweden), an enamel matrix derivative, alveolar distraction osteogenesis and ridge augmentation with placement of an endosseous implant-retained prosthesis at skeletal maturity have all been

described.[36] However, these interventions appear in single case reports or case series and are not supported by evidence from clinical trials. Intentional or initial replantation with Emdogain will not cure or prevent ankylosis[37]; therefore, the use of Emdogain for these applications cannot be justified. Most of the remaining treatment options, with the exception of endosseous implants, have not been widely adopted. These treatments have variable morbidity and unproven long-term benefit. The decision to extract infraoccluded incisors in adolescents and youths is often based on esthetics or the desire to complete orthodontic treatment. Decoronation (which leaves the ankylosed root in situ to be consumed by replacement resorption) has been proposed to minimize ridge resorption and reduce the need for bone grafting before prosthetic treatment. However, the impact of decoronation on the need for eventual alveolar grafting or on the quality of bone that may receive an implant has not been demonstrated.

The choice of treatment depends on the severity of infraocclusion and replacement resorption, the preference and experience of the clinician and patient expectations. The effects of ankylosis extend well beyond the original dental injury and vary with the age of the patient. Oral rehabilitation entails an ongoing investment of time, money and resources[38] and a number of treatment options with no predictable long-term outcomes.

Conclusions

Permanent incisors that have been replanted or severely intruded have poor 5-year survival rates. The development of ankylosis and the inability to stem its progression produce esthetic and functional concerns through local distortion of the gingival architecture and inevitable loss of affected incisors. Once the clinician commits to treatment of the severe luxation injury, vigilance is required for timely and accurate diagnosis of ankylosis. Ankylosis can be diagnosed simply and reliably by its characteristic percussion sound. Other more sophisticated and expensive methods, such as the Periotest, do not appear to improve the diagnosis of ankylosis and are not recommended.

There is little evidence to support the application of current techniques for management of the ankylosed incisor and its sequelae. Additional research to improve the level of evidence for existing treatment modalities and development of novel approaches for management of the ankylosed incisor is warranted.

Clinicians are justified in advising of the grave prognosis for pre-adolescents who present with avulsed incisors that cannot be replanted immediately or incisors that have sustained the most severe degrees of intrusion.[39] External root resorption and ankylosis ensure tooth loss. If, through the process of informed consent, the decision is made to proceed with treatment, ankylosis should be anticipated within weeks of the original injury. The clinician should maintain a vigilant follow-up to facilitate early diagnosis and allow for the timely extraction of the ankylosed incisor to minimize infraocclusion and subsequent growth-related alveolar distortion and bone loss. ◈

THE AUTHORS

Dr. Campbell is adjunct professor at the Schulich School of Medicine & Dentistry, University of Western Ontario, London, Ontario. She also maintains a specialty practice in pediatric dentistry in London.

Dr. Casas is a staff pediatric dentist and a project director at the Research Institute, The Hospital for Sick Children, and an associate professor at the University of Toronto.

Dr. Kenny is director of dental research and graduate studies and senior associate scientist at the Research Institute, The Hospital for Sick Children, and a professor at the University of Toronto.

Correspondence to: Dr. Karen M. Campbell, Division of Orthodontics & Paediatric Dentistry, Schulich School of Medicine & Dentistry, UWO, Dental Sciences Bldg., Rm 1012, London, ON N6A 5C1. E-mail: roy.campbell4@sympatico.ca.

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