REVIEW ARTICLE

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Mechanism and control of tooth eruption: overview and clinical implications

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Structured Abstract

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Objectives – To review pre- and post-emergent eruption, with particular emphasis on distinguishing isolated molar ankylosis from primary failure of eruption (PFE) and genetic considerations in eruption problems.

Material and Methods – Radiographic review of eruption failure patients; animal and human experiments; high precision observations of movements of erupting teeth.

Results – In pre-emergent tooth eruption, the controlling element is the rate of resorption of overlying structures. A path is cleared, and then the erupting tooth moves along it. This has clinical importance in recognizing the cause of eruption problems, particularly PFE, in which all teeth distal to the most mesial involved tooth do not erupt or respond to orthodontics. In our study of by far the largest sample of PFE cases yet reported, familial cases of PFE accounted for approximately ¼ of all cases examined. Candidate genes now are being evaluated. In post-emergent eruption, control seems to be light forces of long duration that oppose eruption, rather than heavy forces of short duration such as those during mastication. Studies of human premolars in their passage from gingival emergence to the occlusal plane show that in this phase eruption occurs only during a few hours in the early evening. The critical hours for eruption parallel the time that growth hormone levels are highest in a growing child. In this stage intermittent force does not affect the rate of eruption, but changes in periodontal blood flow do affect it.

Key words: Moiré magnification; post-emergent eruption; pre-emergent eruption; primary failure of eruption; tooth eruption

Introduction

Tooth eruption is difficult to study, primarily because it occurs so slowly and the teeth are so inaccessible until they emerge into the mouth. As a result, neither the eruption mechanism nor the controlling factors in eruption are completely understood. This paper reviews studies of tooth eruption, with a focus on clinical application of what is known now about pre-emergent eruption and on human studies of erupting premolars using high-resolution measurement techniques.

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Pre-emergent eruption

During the formation of its crown, a developing tooth remains in the same location in the bone. This can be seen most clearly in cephalometric radiographs of children in whom metallic implants have been placed in the jaws to provide stable reference points (1). Eruptive movements begin when root formation begins, and then the tooth starts to move away from the point at which root development is occurring.

For eruptive movements to occur, two things are necessary: 1) force to move the tooth along the eruption path must be generated; and 2) bone and primary tooth roots (and later gingival tissues) must be removed from the eruption path. It seems reasonable that a force created by cellular proliferation at the root apex would propel the erupting tooth – but after eruptive movements begin, cutting off the root apex does not stop eruption. Cellular activity or differential blood flow/pressure in the periodontal ligament (PDL) seem a possibility, but a tooth with no PDL can erupt. These and other possibilities for generating the propulsive force were reviewed by Marks and Schroeder, with no definite conclusion (2). It is fair to say that prior to emergence into the oral cavity, the mechanism that moves a tooth along its eruptive path remains elusive.

It also seems entirely reasonable that resorption to clear the eruption path would be caused by pressure from the eruptive force, but this is not the case. Instead, an eruption path is cleared and the tooth follows along it. In a series of classic experiments with dog lower premolars, Cahill showed that when eruption was prevented by ligating the tooth bud to the lower border of the mandible, the eruption path was cleared anyway (3). Inadvertent human experiments, in which a tooth bud was ligated during treatment of a jaw fracture, show that this also is true in humans (Fig. 1). The rate and direction of resorption to clear the eruption path, not the force developed to move the tooth, controls pre-emergent eruption. How does the beginning of root formation trigger resorptive activity by clast cells in the dental follicle over the crown? So far, that has not been explained.

Pre-emergent eruption failure as a clinical problem

Posterior open bite, a relatively rare type of malocclusion, occurs when permanent teeth in one or more posterior quadrants fail to erupt. There are two possible causes:

1) Mechanical obstruction of eruption. Prior to emergence, this usually is due to an ankylosed primary tooth or a supernumerary tooth but occasionally is caused by another permanent tooth when space is lacking. When an obstruction is removed, a tooth that was unable to erupt often does so, or if not, it can be moved orthodontically. Ankylosis of an unerupted tooth can be considered a form of mechanical obstruction, but permanent release of ankylosis is impossible, and temporary release, so that a tooth can be moved before it re-ankyloses, is difficult and unpredictable.

2) Failure of the propulsive mechanism that moves the tooth. This is seen in the unusual condition of primary failure of eruption (PFE) (4–6). Affected teeth are not ankylosed but fail to follow the eruption path that has been cleared for them, i.e. there is an uncoupling of resorption and eruption (Fig. 2). The periodontal ligament is abnormal, so not only does an affected tooth fail to erupt, it does not respond to orthodontic force. Typically, posterior but not anterior teeth are affected, and all teeth distal to the most mesial affected tooth fail to erupt.

When a permanent first molar does not erupt even though its eruption path has been cleared, the differential diagnosis between ankylosis and PFE is

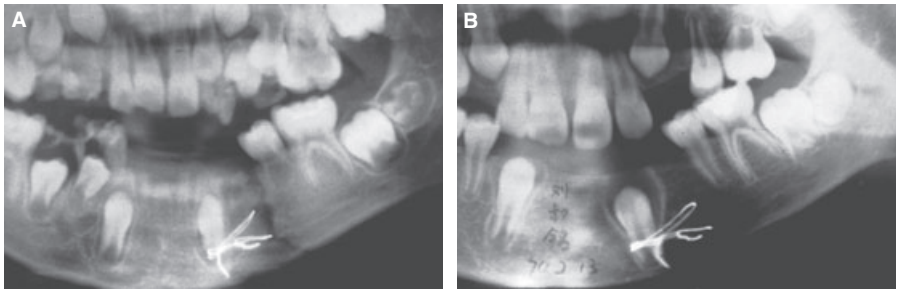

Fig. 1. Effect on eruption of ligating a human tooth bud. A, immediately after mandibular fracture in which one canine was inadvertently ligated; B, 1 year later. Note that the eruption path for the ligated tooth was cleared although it could not erupt, while the canine on the other side erupted normally. (courtesy Dr John Lin).

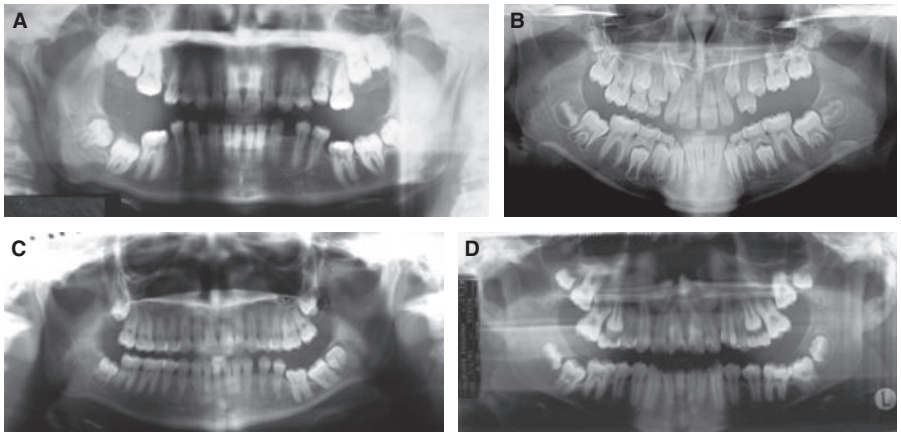

Fig. 2. The classic form of primary failure of eruption (PFE), in which the loss of eruption potential appears to strike affected teeth at about the same chronologic time. Note the uncoupling of resorption and eruption for multiple teeth. (A), All four posterior quadrants affected, with 2nd premolars questionably involved. (B), Maxillary left quadrant involved, including 2nd premolar; other quadrants not affected. (C), All four quadrants involved, including 2nd premolars in three quadrants. (D), All four quadrants involved, molars only. In PFE any or all the posterior quadrants can be affected. First premolars are rarely involved, but second premolars frequently are.

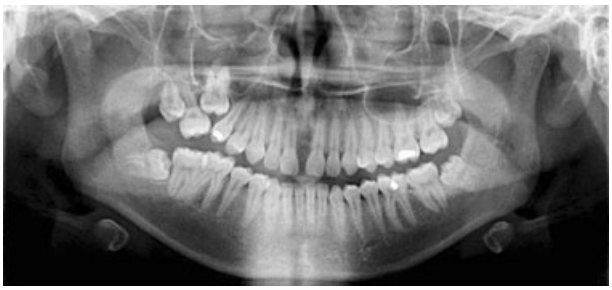

Fig. 3. Isolated ankylosis of the maxillary right first molar. Note the normal eruption of the second molar. A first molar that fails to erupt should be extracted when the eruption failure is first noticed, so that if the second molar is normal, it can move mesially and bring alveolar bone with it. At this late stage extraction of the first molar will leave a major periodontal defect that cannot be corrected.

important. If the problem is isolated ankylosis of the first molar (Fig. 3), second and third molars are likely to be normal and will erupt on their own, and can be moved into the first molar area when the ankylosed tooth is removed. If it's PFE, all the molars and perhaps the premolars are abnormal, will not erupt, and cannot be moved orthodontically. A definitive differential diagnosis cannot be made until the child is old enough to see radiographically whether or not the premolars and second molars are following the eruption path that is being cleared for them. Nevertheless, extraction of a non-erupting first molar is recommended so that a normal second molar can drift mesially, if it is normal. No harm is done by early extraction if the ultimate diagnosis is PFE. The molar would have had to be extracted anyway. A diagnosis of PFE at least prevents attempted orthodontic treatment that would prove futile.

Genetic considerations in eruption failure

In considering the mechanism and control of tooth eruption, genetic etiology is an important consideration and becomes a factor in differential diagnosis of eruption failure. Eruption failure and delayed eruption that does not involve a failure of the propulsive mechanism are associated with craniofacial dysostosis, hypothyroidism, hypopituitarism, and several other recognized genetic and medical syndromes (7–10), but in essentially all these conditions the eruption failure is due to mechanical obstruction (i.e. fibrous gingiva, supernumerary teeth or retained deciduous teeth). The presence of a familial involvement when eruption failure is observed (with an uncoupling of resorption and eruption) is most consistent with PFE. In our study of by far the largest sample of PFE cases yet reported, familial cases of PFE accounted for approximately ¼ of all cases examined (6). Further analysis of these families strongly suggested an autosomal dominant inheritance pattern. However, PFE related to a genetic syndrome also could be non-familial (i.e. as a result of a sporadic mutation). The specific genetic contribution to non-syndromic eruption failure, or PFE, remains unclear. Whether familial or non-familial, the eventual identification of genetic alterations responsible for eruption failure is important, as their discovery will also

contribute to the understanding of the normal eruption process.

For many of the human syndromic conditions that involve disruptions in the eruption process, a corresponding mode of inheritance and causative genetic mutation has been identified. It is important to appreciate the goals of deciphering the genetic contribution to eruption failure from a clinical perspective. Since eruption failure includes a broad spectrum of 'eruption phenotypes', ranging in severity from delayed to complete failure and occurring at various developmental time points, it is logical to hypothesize that specific eruption failure phenotypes may stem from distinct mutations. Hence, a first step toward understanding the mechanism involved in eruption failure is the definitive characterization of eruption failure phenotypes and the concomitant correlation with the causative mutations.

Recent findings from our study that sought to accomplish this clinical characterization showed that there are indeed two distinguishable types of PFE that seem to be related to the timing of onset (6). The first (Type I) represents the classic form described initially (Fig. 2), in which there is a progressive open bite from the anterior toward the posterior. This suggests that the eruption defect 'struck' at the same developmental time. The second (Type II; Fig. 4) presents as a varied expression of eruption failure in more than one quadrant and greater although inadequate eruption of a second molar. In this type, it is hypothesized that the timing of onset might be related to the stage of root development. As is expected in phenotypic studies, we also observed a combination of Type I and Type II in different quadrants in 24% of the subjects with a definite diagnosis of PFE. Rarely, we also observed individuals with PFE in one quadrant coupled with a single ankylosed tooth in a different quadrant. This underscores the theory that PFE and ankylosis might be closely related (11). It is possible that abnormalities in the PDL can lead to either condition.

Finally, to fully consider the genetic contribution to the eruption process it is important to understand the cellular and molecular advances that give rise to a genetic etiology. Specifically, it is well known that normal eruption is the result of a temporally coordinated process in which the dental follicle interacts with both osteoclasts and osteoblasts. Studies in mice have revealed clues about how cell to cell signaling of key receptor-associated factors (i.e. c-fos, RANKL, INF-B) interact in a common pathway to facilitate osteoclastogenesis (12). However, there is clearly a considerable amount of work remaining to completely determine the genetic etiology of eruption defects in humans. As our knowledge of human genetics and molecular biology rapidly advances, it is quite possible that future treatment modalities could include therapeutics that recreate eruption events. Identification of gene(s) involved in PFE and other conditions will not only provide a better understanding the tooth eruption process, but also lead to the development of effective treatment options for these otherwise clinically challenging eruption defects. The continued development of diagnostic tools that can positively distinguish between the various subtypes of eruption failure may offer a way to successfully manage PFE.

Post-emergent eruption

Post-emergent eruption occurs in four stages: first, the *pre-functional spurt*, as the tooth moves from initial emergence into the mouth up to the occlusal plane; and then three stages that more or less parallel vertical facial growth. These are the *juvenile equilibrium* when

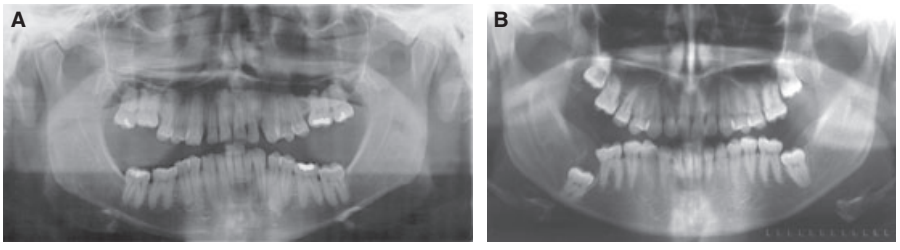

Fig. 4. In Type 2 PFE, the onset appears to be at a certain developmental stage rather than all affected teeth at the same time. (A), Both upper quadrants show 2nd molar eruption after failure of 1st molar eruption, with the 2nd molar affected at about the same developmental stage. On the left side 2nd premolars are involved, on the right they are not. (B), All four quadrants affected, with premolars and 2nd molars showing failure later than the first molar, but at about the same developmental stage.

both jaw growth and eruption are quite slow, the *adolescent eruptive spurt* as growth accelerates and teeth have to erupt to remain in occlusion, and the *adult equilibrium*. To keep up with jaw growth, first permanent molars have to erupt about a centimeter after they first come into occlusion, so the amount of post-emergent eruptive movement is not trivial. Because a tooth can erupt even late in adult life when its antagonist is lost, it is clear that the eruption mechanism remains active during adult life.

After a tooth comes into eruption, collagen fibers in the PDL become oriented to support the tooth against the forces of occlusion. As collagen matures it cross-links and shortens, and this provides a potential propulsive mechanism for eruption, which would be available only after the number and orientation of the fibers changed upon exposure to oral forces. Both animal and inadvertent human experiments with ingestion of lathryogens, which stop collagen maturation and also stop tooth eruption, indicate that this is a primary mechanism for post-emergent eruptive movement (13). Post-emergent control would seem to have something to do with forces that oppose eruption.

Until recently, measurements on sequential dental casts or sequential cephalometric radiographs were the only way to track post-emergent eruption, and it was impossible to resolve less than a millimeter or so of change. In the 1980s it became possible to use miniature variable capacitance displacement transducers (VCDTs) to track the eruption of animal incisors. This provided resolution of 1–2 microns (μ), enough to allow experiments with forces opposing eruption. Experiments in Proffit's laboratory with continuously-erupting rabbit incisors (Fig. 5) showed that continuous application of a force of less than 1 g would stop eruption and a force of 1–2 g would cause intrusion of the tooth (14). If the force was applied intermittently, 10% time had little or no effect; 25% time had a variable effect, as if it might be a threshold; and 50% had the same effect as continuous force (15). Similar results were obtained by Moxham and Berkovitz in VCDT experiments with both rabbit incisors and ferret canines, which are more similar to human teeth (13, 16). These data suggest that control of post-emergent eruption probably is much more from light but long-lasting soft tissue pressures (such as those from tongue posture during sleep) than heavy but short-duration forces during mastication and occlusion.

Fig. 5. Experimental set-up for variable capacitance displacement transducer (VCDT) tracking of a rabbit incisor and its response to intermittent force. The principle of the device is that very small changes in the separation of two plates that form a capacitor can be measured accurately. The VCDT plates can be seen just above the center of the photograph, with one plate attached to the incisor, the other to the jaw. A beam that can be moved into or out of contact, so that force opposing eruption of known magnitude and duration can be applied, is seen contacting the incisal edge.

Extension of VCDT studies to humans was not possible because a VCDT must be attached to the bone of the jaw. The first non-invasive method for tracking the eruption of a human tooth used a video microscope to view the changes in position of an optical ruling on an erupting second premolar relative to another ruling on a bar between the first molar and first premolar (17). This provided the same 1–2 μ resolution as VCDT instrumentation, and was expected to allow tracking of changes over periods of 30 min to an hour. The observation that the premolar erupted very little during the afternoon, but quite rapidly (for a human tooth) in the evening, was unexpected. With a fiber optic cable attached to a distant video microscope and a fixed prism intraorally, it was possible to continuously observe the erupting tooth for a 12-h (overnight) period (Fig. 6). This confirmed that eruption occurred almost entirely during the evening, between 6 or 7 PM and midnight or 1 AM (Fig. 6) (18, 19).

In order to run real-time experiments with an erupting human tooth, it would be necessary to improve the resolution by at least an order of magnitude. This was accomplished by creating an interference pattern between a chevron-shaped ruling attached to the erupting tooth and its inverted reflection, and by taking advantage of Moiré magnification to track the movement of interference bars (Fig. 7) (20). The theoretical resolution of this device was 0.02 μ, and the actual resolution was better than 0.05 μ (21). With

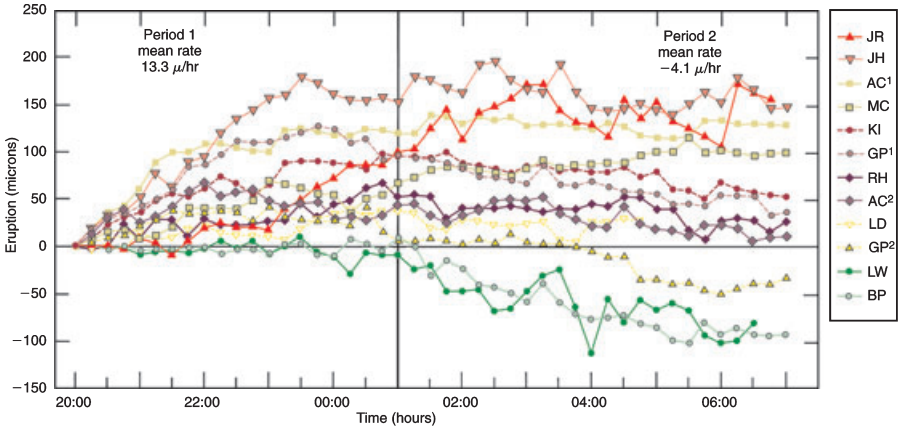

Fig. 6. Eruption plots for 12 human premolars continuously observed via a fiber optic cable to a video microscope from 8 PM (20:00) to 8 AM. Note the consistent pattern of eruption during the early evening, trailing off to no eruption or intrusion after midnight.

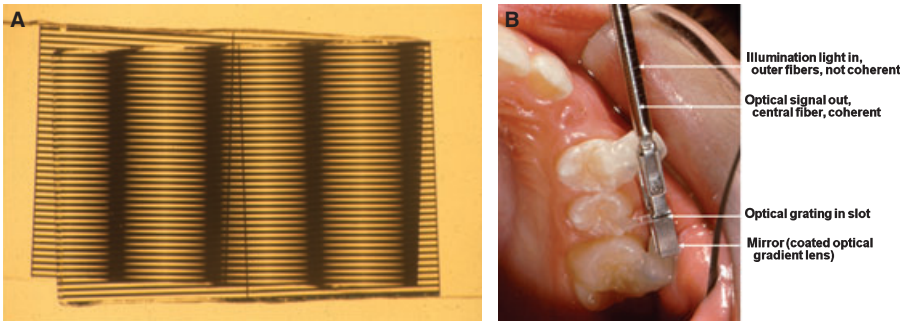

Fig. 7. (A), Moiré magnification occurs when two optical rulings are moved vertically relative to each other, and Moiré interference fringes (the dark vertical bands) move laterally at a greater rate that can be tracked with very high precision. (B), For use in a miniature intraoral device, the interference pattern was created between a chevron-shaped ruling attached to the erupting tooth and its inverted reflection, so that the orientation of the rulings could be known precisely.

this instrumentation, it was possible to run two experiments in children with an erupting premolar: 1) determination of the effect of short-duration applied force that opposed eruption; and 2) the effect of altering blood flow in the PDL.

Effect of short-duration force

Based on the animal experiments, it seemed likely that intermittent force opposing eruption would have little or no effect, but the observation that eruption occurred primarily during evening hours when the teeth were not brought into occlusion offered the possibility that a lengthy period without loading was needed for eruption. In typical human sleep, swallowing that brings the teeth into light contact occurs only about once an hour, and rapid eye movement (REM) sleep with clenching and grinding of the teeth occurs after midnight. The experiments showed that intermittent light force against an erupting tooth did not affect the rate of eruption (Fig. 8) (22). The conclusion was that presence or absence of force opposing eruption did not

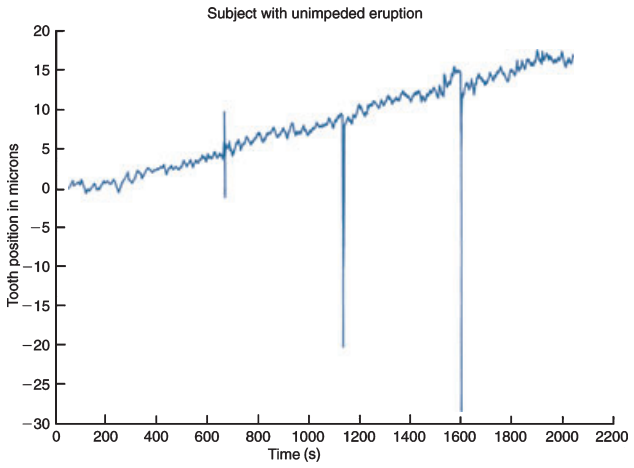

Fig. 8. In this child intermittent force against the erupting tooth displaced it, but did not affect the rate of eruption. Note the resolution of the position of the erupting tooth to a fraction of a micron, using the Moiré magnification technique. Similar results were obtained consistently.

explain the diurnal rhythm in eruption. The evening period of rapid eruption is also the time when major release of growth hormone (HGH) occurs in children. This suggests that during the pre-functional eruptive spurt, HGH levels affect the amount of eruption.

Growth hormone-deficient children now receive HGH injections. It would be possible to evaluate the rate of eruption after a child did not receive HGH for a wash-out period, and compare the rate of eruption after the next HGH injection. Preliminary data from such children suggest that eruption may be faster after HGH replacement, but unfortunately it has not been possible to study enough HGH-deficient children to be sure what the effect on eruption would be.

Effect of altering blood flow

The second experiment was done by examining the effect of injecting a local anesthetic with or without a vasoconstrictor over the root of the erupting tooth. The anesthetic itself is a modest vasodilator, which would increase blood flow in the area; it usually is used with a small quantity of epinephrine as a vasoconstrictor to increase the duration of anesthesia by decreasing blood flow. The experiments showed that injecting the anesthetic without epinephrin increased the rate of eruption if a tooth already was erupting, and could cause a tooth that was not erupting or intruding slightly to begin erupting (Fig. 9). Injecting the anesthetic with a vasoconstrictor stopped eruption or led to intrusion (23).

It appears that blood flow in the PDL does have something to do with the mechanism of eruption, at least in the pre-functional eruptive spurt. Perhaps this can be considered evidence for a blood flow influence on eruption prior to emergence as well.

Conclusions

Tooth eruption begins when formation of the PDL begins, but its control is the rate and direction of resorption to clear the path of eruption, not the rate at which the tooth root lengthens. The eruption path is cleared, and the tooth then follows along it. An uncoupling of resorption and eruption indicates either ankylosis or a problem with the eruption mechanism. Both occur in humans, and there appears to be a genetic link between isolated molar ankylosis and primary failure of eruption, because both can be found in different quadrants of the same mouth.

The major source of force to move an erupting tooth after it reaches occlusal function appears to be maturation, cross-linking and shortening of collagen fibers in the PDL, but human experiments suggest that altering blood flow to an erupting tooth does affect eruption, at least during the pre-functional eruptive spurt. Orthodontic tooth movement is accomplished with light forces of long duration; post-emergent eruptive tooth movement seems to be controlled by similar forces, rather than by the forces of occlusion.

Clinical relevance

Failure of tooth eruption is a frequent and difficult problem in clinical orthodontics. Although mechanical obstruction of an erupting tooth is the most likely cause of eruption failure, primary failure of eruption (PFE) is

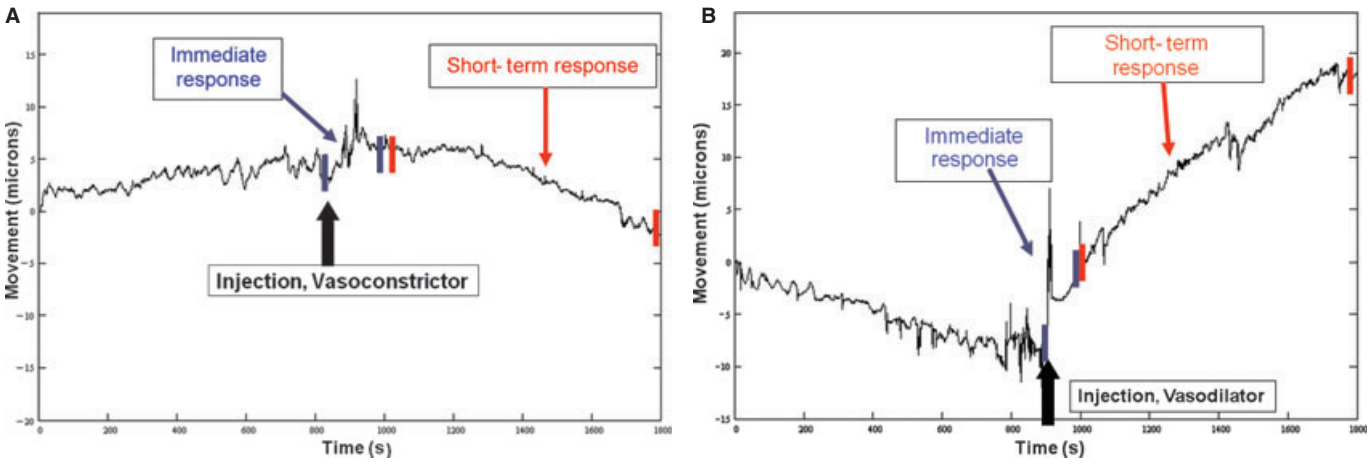

Fig. 9. The effect of altering blood flow around an erupting premolar, observed using Moiré magnification. (A), Vasocontriction from injection of a local anesthetic with epinephrin. The tooth was erupting slowly. The immediate response was displacement of the tooth (on a scale of microns) followed by intrusion. (B), Vasodilation from injection of a local anesthetic without epinephrin. The tooth was intruding slowly (during an afternoon session, a time at which no eruption is expected). After the injection, eruption at a rapid rate began.

an often-overlooked cause of posterior open bite. The diagnostic distinction between mechanical obstruction, isolated ankylosis and PFE is critically important in planning treatment. Establishing the prognosis for patients with eruption failure is likely to be an early application of genetic analysis in orthodontics.

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