TOOTH ERUPTION

- Chronology
- Biology
- Ankylosis
- Infraocclusion or submerged teeth
- Primary Failure of Eruption
- Tooth Migration

Classic ADA North American Standards for Tooth Development

TABLE 3-2 *Chronology of Tooth Development, Primary Dentition*

	Calcification begins		Crown completed		Eruption		Root completed	
Tooth	Max.	Mand.	Max.	Mand.	Max.	Mand.	Max.	Mand.
Central	14 wk. in utero	14 wk. in utero	1½ mo.	2½ mo.	10 mo.	8 mo.	1½ yr.	1½ yr.
Lateral	16 wk. in utero	16 wk. in utero	2½ mo.	3 mo.	11 mo.	13 mo.	2 yr.	1½ yr.
Canine	17 wk.	17 wk. in utero	9 mo.	9 mo.	19 mo.	20 mo.	3¼ yr.	3¼ yr.
1st Molar	15 wk. in utero	15 wk. in utero	6 mo.	5½ mo.	16 mo.	16 mo.	2½ yr.	2¼ yr.
2nd Molar	19 wk. in utero	18 wk. in utero	11 mo.	10 mo.	29 mo.	27 mo.	3 yr.	3 yr.

TABLE 3-3 *Chronology of Tooth Development, Permanent Dentition*

	Calcification begins		Crown completed		Eruption		Root completed	
Tooth	Max.	Mand.	Max.	Mand.	Max.	Mand.	Max.	Mand.
Central	3 mo.	3 mo.	4½ yr.	3½ yr.	7¼ yr.	6¼ yr.	10½ yr.	9½ yr.
Lateral	11 mo.	3 mo.	5½ yr.	4 yr.	8¼ yr.	7½ yr.	11 yr.	10 yr.
Canine	4 mo.	4 mo.	6 yr.	5¾ yr.	11½ yr.	10½ yr.	13½ yr.	12¾ yr.
1st Premolar	20 mo.	22 mo.	7 yr.	6¾ yr.	10¼ yr.	10½ yr.	13½ yr.	13½ yr.
2nd Premolar	27 mo.	28 mo.	7¾ yr.	7½ yr.	11 yr.	11¼ yr.	14½ yr.	15 yr.
1st Molar	32 wk. in utero	32 wk. in utero	4¼ yr.	3¾ yr.	6¼ yr.	6 yr.	10½ yr.	10½ yr.
2nd Molar	27 mo.	27 mo.	7¾ yr.	7½ yr.	12½ yr.	12 yr.	15¾ yr.	16 yr.
3rd Molar	8 yr.	9 yr.	14 yr.	14 yr.	20 yr.	20 yr.	22 yr.	22 yr.

Eruption sequence

- Maxillary teeth: **6 1 2 4 5 3 7**
- Mandibular teeth: **6 1 2 3 4 5 7**

- Females develop slightly earlier than males

Standards on based on data several decades old in the US using Caucasian populations of Northern European ancestry

HAVE THERE BEEN ANY CHANGES
REPORTED IN THE LAST FEW DECADES?

Emergence of permanent teeth and dental age in a series of Finns – Nystrom et al. Acta Odontologica Scandinavia April 2001. 68% of children – lower 1s erupted before 6s – shift in emergence order in last 30 years

New standards for emergence of permanent teeth in Australians – Diamanti and Townsend. Australian Dental J. 2008. Eruption rate of all permanent teeth delayed compared to data from previous years.

Expected location of neonatal line

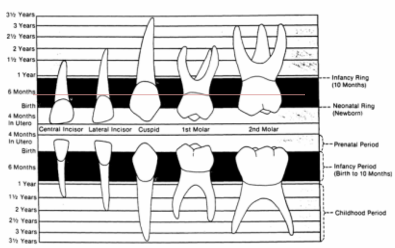

FIGURE 3-13 Primary teeth shown on a developmental scale that indicates the expected location of the neonatal line. From a chart of this type, the timing of illness or traumatic events that led to disturbances of enamel formation can be deduced from the location of enamel lines on various teeth.

The Consideration of Dental Development In Serial extraction
- Moorrees CA, Fanning EA, Gron AM. AJO **1963**. OLD BUT STILL USEFUL

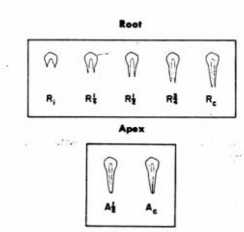

Fig. 4 Stages of root formation for determining dental maturation.

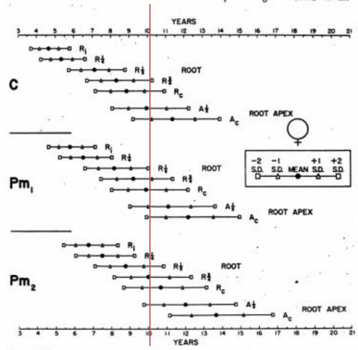

Fig. 10 Norms of root formation for the permanent mandibular canines and premolars of females.

The Consideration of Dental Development In Serial extraction - Moorrees CA, Fanning EA, Gron AM. AJO 1963.

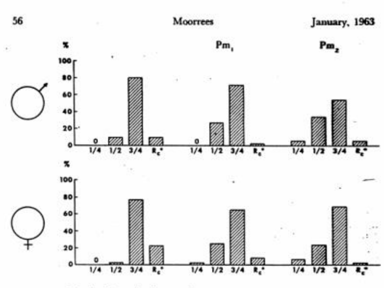

Fig. 12 The extent of root formation at emergence.

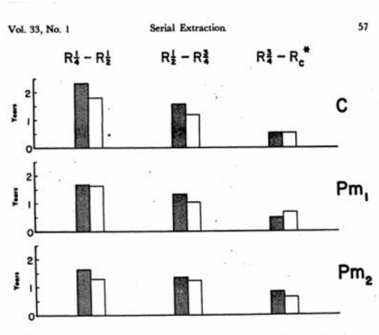

Fig. 13 The average time for the development of quarter stages of root length.

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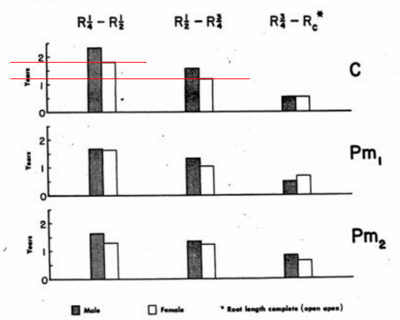


Fig. 13 The average time for the development of quarter stages of root length.

BIOLOGY OF TOOTH ERUPTION

- Definition: movement of a tooth from its site of development within the alveolar process. Research has shown that eruption of teeth continues well into the fourth and fifth decades of life albeit on a smaller scale.

Theories of tooth eruption

- Discounted
  1. Pulpal pressure
  2. Pulpal growth
  3. PDL fibroblast traction
  4. Vascular pressure

- Newer
  1. Root elongation
  2. Alveolar bone remodeling
  3. Periodontal ligament formation
  4. Dental follicle

1. Root elongation theory

- Basis : Not biological
- Does not explain movement in three-dimensional space
- Teeth without roots erupt (Dentin dysplasia Type I)
- May account for eruption acceleration

CLINICIAN'S CORNER AJO-DO

Rootless eruption of a mandibular permanent canine

Yehoshua Shapira[a] and Mladen M. Kuftinec[b]
New York, NY

The purpose of this article was to describe the rootless eruption of a mandibular permanent canine in a 10-year-old boy; his mandible had been fractured in a car accident. The fracture was at the region of the developing canine, resulting in arrested root formation and causing abnormal, rootless eruption. Current theories on tooth eruption and the important role of the dental follicle in the process of eruption are discussed. (Am J Orthod Dentofacial Orthop 2011;139:563-6)

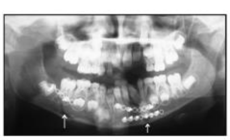


Fig 1. Panoramic radiograph at age 7 years showing postsurgical repair of the double mandibular fractures (*arrows*) with rigid fixation.

Fig 3. Panoramic radiograph 2 years later (age, 9 years) showing the rotated left canine crown with no root formation. The right permanent second molar appears mesially inclined.

Fig 2. Panoramic radiograph 1 year later (age, 8 years) showing repair of the fractures. The mandibular left canine appears rotated, and the left lateral incisor has an obliterated root canal.

Fig 4. Pretreatment panoramic radiograph (age, 10 years) showing the rootless left permanent canine and the mandibular right permanent second molar horizontally impacted.

Fig 6. Panoramic radiograph showing the 2×4 maxillary edgewise appliance. The left canine appears in its eruption process.

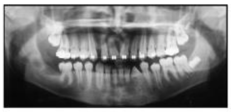


Fig 7. Panoramic radiograph (age, 12 years) showing the left canine erupted and rootless. The impacted mandibular right permanent second molar and the third molar bud were removed.

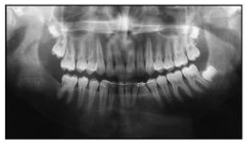


Fig 8. Posttreatment panoramic radiograph showing the complete rootless eruption of the mandibular left canine.

2. Periodontal ligament theory

- Basis : Fibers in PDL
- Presence of PDL does not assure eruption
- Osteopetrotic mutations - PDL present but no eruption
- Dentinal dysplasia - no PDL but teeth erupt
- Previous research was done on rodents with teeth that erupt continuously

3. Alveolar bone remodeling theory

- Basis : Alveolar bone growth, tooth development and tooth eruption are closely related
- Bone formation per se is not sufficient for tooth eruption (cleidocranial dysplasia)

4. Dental Follicle Theory

- Basis : Clastic cells in DF
- Eruption begins only after crown formation is complete
  - Clastic cells surrounding crown not activated until enamel formation is complete (Proffit)
- Root formation occurs initially at the expense of basal bone without movement of the crown
- Most root growth occurs during the stage of preocclusal eruption
- Root completion is at the expense of basal bone
- Tooth eruption and bone formation depend on the dental follicle - Marks, Cahill

Journal of Oral Pathology 1980: **9:** 189–200

Tooth eruption: evidence for the central role of the dental follicle

Donald R. Cahill and Sandy C. Marks, Jr.

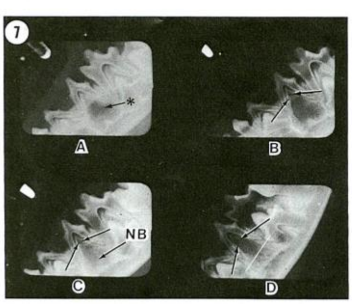
Department of Anatomy, University of Miami School of Medicine, Miami, Florida, and Department of Anatomy, University of Massachusetts Medical School, Worcester, Massachusetts, U.S.A.

Abstract. The roles of the gubernaculum dentis, root formation, tooth crown and dental follicle in prefunctional eruption of a mandibular premolar have been studied in nine beagle dogs by radiographic and histologic evaluations of the effects of surgical ablation or removal of these structures on tooth eruption. The dental follicle was the only one of these structures required for the coordinated enlargement of the eruption pathway and formation of bone in the base of the bony crypt, the radiographic and histologic hallmarks of tooth eruption. These data, together with the topographic relationships of the dental follicle to areas of localized bone resorption and formation, are interpreted to mean that the dental follicle may influence, if not coordinate, these processes in tooth eruption.

Accepted for publication 5 December 1979

Cahill and Marks Famous Experiment

196 CAHILL AND MARKS



Beagle Dog Teeth

Fig. 7. Radiographs depicting effect of removal of tooth crown, dental follicle remaining *in situ*. A, during surgery, 17 postnatal weeks. Arrow (*) shows bony crypt after removal of crown. B, operated side 1 week after surgery showing radiolucency above bony crypt indicating enlargement of eruption pathway (arrows). C, operated side 2 weeks after surgery. Note radio-opacity at base of bony crypt indicative of new bone formation (NB). D, operated side 3 weeks after surgery. Arrows show further enlargement of eruption pathway. White line indicates plane of section of Fig. 8

Nature's Evidence that the dental follicle creates the eruption pathway

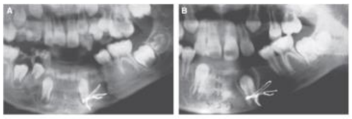


Fig. 1. Effect on eruption of ligating a human tooth bud. A, immediately after mandibular fracture in which one canine was inadvertently ligated; B, 1 year later. Note that the eruption path for the ligated tooth was cleared although it could not erupt, while the canine on the other side erupted normally.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

60 | *Orthod Craniofac Res* 2009:**12**:59–66

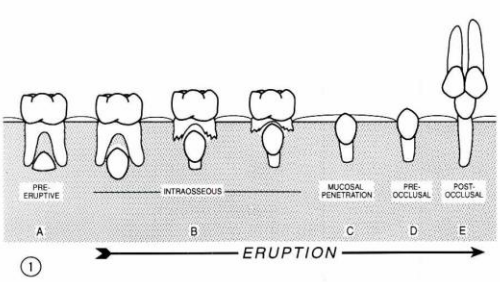
From Proffit's article

Five stages of tooth eruption:

1. Preeruptive movements
2. Intraosseous eruption
3. Mucosal penetration
4. Preocclusal eruption
5. Postocclusal eruption

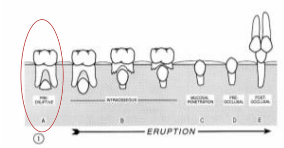
Five stages of tooth eruption:

224 *S.C. Marks et al.*



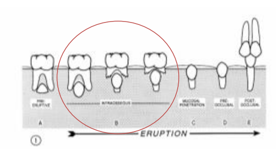
Stage 1: Preeruptive movements

- Random, very short movements
- Cause unknown - development of dental follicle or regional growth of the jaws
- Gubernacular canals - small remnants of the original invagination of oral ectoderm



Stage 2: Intraosseous stage

- Rate limiting step in early eruption is formation of an eruption pathway by osteoclasts - shown by Cahill in dogs
- No osteoclasts - no eruption - by Sundquist 1994
- Bony deposition occurs at apical end of dental follicle
- If crown is removed but not DF eruption still takes place - Marks 1985



Stage 2 continued

- NO DF - no eruption
- Removal of coronal half - no eruption
- Removal of apical half - no eruption
- Removal of crown & replacement with a metal tooth - eruption
- Early experiments removed enamel epithelium
- Enamel organ alone insufficient for eruption - Larson 1995

Stage 2 continued

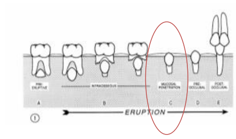
- Fragmentation of a sialoprotein (DF-95) seems to mark the onset of preosseous eruption - Gorski 1994
- Proposed pathway: activation of proteases from the enamel organ at the completion of crown formation initiates eruption by release of metalloproteinases from the dental follicle - Marks 1996

- Root formation is a consequence not a cause of tooth eruption
- Enamel organ is involved
- CSF-a, EGF, TGF-B, and IL-1 are likely candidates for local molecular regulation
- Bone resorption is the rate-limiting step of this stage
- Orientation of follicle – differential gene expression linked to nuclear matrix – intermediate filament proteins – see Bidwell et al, Arch Oral Biology 1995.

- Primate experiments - transplantation failures due to damage follicle
- Root growth is usually fast enough to keep up with eruption
- Marks' summary: Primary determinant of both the direction and rate of tooth eruption is the rate of formation of the eruption pathway and its coordination with bone formation in selected areas of the crypt and alveolar bone

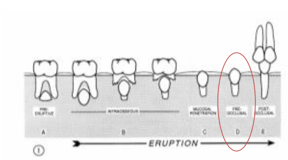
Stage 3: Mucosal penetration

- Enamel epithelium fuses with oral epithelium
- Rate of eruption increases when cusps reach alveolar crest
- Clinical signs of hypersensitivity ("teething") during this stage are thought to be from release of enamel matrix proteins



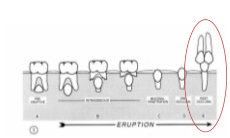
Stage 4: Preocclusal eruption

- Major event: Formation of junctional epithelium- not much known about this - Marks
- Rate of preocclusal eruption : 75 microns per day - Proffit 1991 using a high resolution video microscope custom made



Stage 5: Eruption at the Occlusal Plane

- Tooth eruption slows
- Alveolar bone becomes denser around teeth (lamina dura)
- Maturation/organization of fibers of periodontal ligament
- Proffitt: Shrinkage of collagen fibers; also claims major factor is blood pressure from pulp (Old study showed vasodilator increasesed eruption rate)
- Determinants of final positioning not known - possibly Enlow's drift



Ectopic Eruption

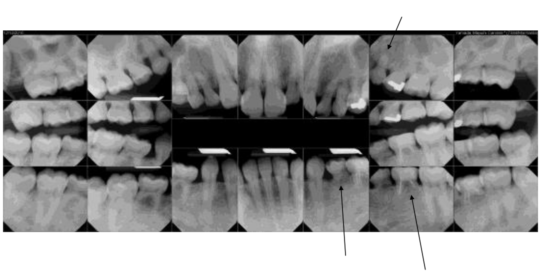
Definition: Eruption occurring in an abnormal position or place

- 1.2% of children in North America
- Unknown causes but probably genetic
- Can cause:
    - A) Resorption of a primary tooth other than the one it is supposed to replace or
    - B) Resorption of an adjacent primary or permanent tooth

Over-retained teeth

- Defined as a primary tooth still present when 3/4 of root of permanent successor has formed
- Possibly some root of primary tooth present
- Should extract if not much mobility
- Also extract if major part of root present (e.g. distal of primary first or second molar)

Note: If appliances are placed on primary teeth and forces applied, the primary tooth root will usually resorb – urban myth??



Delayed eruption

- Children whose primary or permanent teeth erupt six months or later than normal, or who have asymmetric eruption, should be evaluated for abnormal dental eruption or congenitally missing teeth.
- Delays in dental eruption can be familial or can occur with conditions such as:
  - Down syndrome
  - Hypothyroidism, hypopituitarism
  - Achondroplastic dwarfism
  - Osteopetrosis, rickets, or chondroectodermal dysplasia.

ANKYLOSIS of primary teeth

- Causes:
  - Genetics (inherited)
  - Trauma
- Diagnosis:
  - Submergence
    - Shorter tooth than permanent neighbors so watch bone level – could be normal if pdl levels are the same
    - Serial BWXRs or PANs useful if not sure
    - Sound not diagnostic (see permanent teeth)
    - Mobility

KokichAJODOJun2002MissingTeeth.pdf KurolAJODOJun2002.pdf

ANKYLOSIS of primary teeth

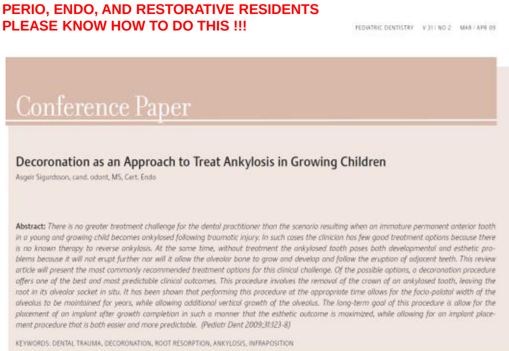
- If succedaneous tooth present:
  - Use as a space maintainer as long as possible
  - Extract when over one half of successor root formed
    - Why? Causes delay in eruption
- If no successor:
  - Extract ASAP
  - Move teeth into space for bone
  - Careful extraction - potential serious periodontal problems - also if wait too long to extract

KokichAJODOJun2002MissingTeeth.pdf KurolAJODOJun2002.pdf

ANKYLOSIS of permanent teeth

- Causes : Genetics or trauma - damage to PDL
- Diagnosis - history, percussion
- Treatment - none or extraction - subluxation *rarely* helps
- Ectopic canines (& other impacted teeth) - occasionally ankylosed - probably iatrogenic in most cases (Becker)

PERIO, ENDO, AND RESTORATIVE RESIDENTS PLEASE KNOW HOW TO DO THIS !!!



PEDIATRIC DENTISTRY V 31 / NO 2 MAR / APR 09

Conference Paper

Decoronation as an Approach to Treat Ankylosis in Growing Children

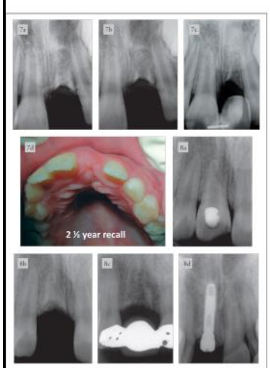
Asgeir Sigurdsson, cand. odont, MS, Cert. Endo

Abstract: *There is no greater treatment challenge for the dental practitioner than the scenario resulting when an immature permanent anterior tooth in a young and growing child becomes ankylosed following traumatic injury. In such cases the clinician has few good treatment options because there is no known therapy to reverse ankylosis. At the same time, without treatment the ankylosed tooth poses both developmental and esthetic problems because it will not erupt further nor will it allow the alveolar bone to grow and develop and follow the eruption of adjacent teeth. This review article will present the most commonly recommended treatment options for this clinical challenge. Of the possible options, a decoronation procedure offers one of the best and most predictable clinical outcomes. This procedure involves the removal of the crown of an ankylosed tooth, leaving the root in its alveolar socket in situ. It has been shown that performing this procedure at the appropriate time allows for the facio-palatal width of the alveolus to be maintained for years, while allowing additional vertical growth of the alveolus. The long-term goal of this procedure is allow for the placement of an implant after growth completion in such a manner that the esthetic outcome is maximized, while allowing for an implant placement procedure that is both easier and more predictable. (Pediatr Dent 2009;31:123-8)*

KEYWORDS: DENTAL TRAUMA, DECORONATION, ROOT RESORPTION, ANKYLOSIS, INFRAPOSITION

11 y.o. boy traumatic injury. Left central incisor reimplanted 12 hours later. One year after trauma – note replacement resorption

“Decoronation “ procedure : crown is removed and tissue sutured over to cover the resorbing root



The goal is to preserve periosteum across the extraction site. Alveolar bone growth will occur under a healthy periosteum.

Beware of : Infraocclusion or submerged teeth

- Primary dentition
- Etiology unknown
- Teeth are <u>not ankylosed</u>
- Kurol - 9% of primary molars so affected
- No treatment required unless teeth are tipped into space or there is no permanent successor

Primary failure of tooth eruption

- Primary and secondary dentitions fail to erupt
- Surgical exposure and orthodontic treatment do not work
- No other systemic problems
- Skeletal/facial growth normal

REVIEW ARTICLE

WR Proffit
SA Frazier-Bowers

Mechanism and control of tooth eruption: overview and clinical implications

Authors' affiliation:
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Structured Abstract
Authors – Proffit WR, Frazier-Bowers SA
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.

PRIMARY FAILURE OF ERUPTION

Proffit and Frazier-Bowers. Mechanism and control of tooth eruption

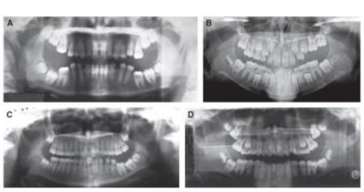

Fig. 2. The classic form of primary failure of eruption (PFE), in which the loss of eruption potential appears to strike affected teeth at about the same chronologic time. Note the uncoupling of resorption and eruption for multiple teeth. (A), All four posterior quadrants affected, with 2nd premolars 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.

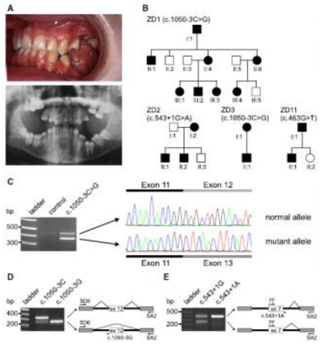

Figure 1. *PTHR1* Mutations in Individuals Affected by Nonsyndromic PFE and Their Effect on Transcript Splicing
(A) Eruption failure of posterior teeth and deficits in alveolar bone growth result in a severe bilateral open bite. Shown are an intraoral view (upper chart) and orthopantomogram (lower chart) of patient ZD1-III:2.
(B) Pedigree drawings showing individuals examined clinically and by mutation analysis. *PTHR1* mutations segregating with the clinical phenotype are presented in brackets.
(C) RT-PCR analysis of splice-site mutation c.1050-3C > G in gingival RNA of patient ZD1-III:3. Sequence traces of the normal allele and the shortened mutant allele demonstrate the excision of exon 12 in the mature transcript of the mutated allele.
(D) In vitro minigene reporter analysis of splice-acceptor sequence mutation c.1050-3C > G. Irregular transcript splicing is observed for the mutant allele such that the entire *PTHR1* exon 12 is skipped.
(E) In vitro minigene reporter analysis of splice-donor sequence mutation c.543+1G > A. Irregular transcript splicing involving a vector-specific cryptic splice is observed for the mutant allele.

PFE Summary

- Rare, familial
- Must distinguish between mechanical obstruction, isolated ankylosis, and PFE
- Occurs at post-emergent stage
- Almost always posterior permanent teeth
- Cannot treat with conventional orthodontic mechanotherapy

The genetics of human tooth agenesis: New discoveries for understanding dental anomalies

ABO READING LIST

Heleni Vastardis, DDS, DMSc[a]
New York, NY

The important role of genetics has been increasingly recognized in recent years with respect to the understanding of dental anomalies, such as tooth agenesis. The lack of any real insight into the cause of this condition has led us to use a human molecular genetics approach to identify the genes perturbing normal dental development. We are reporting a strategy that can be applied to investigate the underlying cause of human tooth agenesis. Starting with a single large family presenting a clearly recognizable and well-defined form of tooth agenesis, we have identified a defective gene that affects the formation of second premolars and third molars. With the use of "the family study" method, evidence is produced showing that other genetic defects also contribute to the wide range of phenotypic variability of tooth agenesis. Identification of genetic mutations in families with tooth agenesis or other dental anomalies will enable preclinical diagnosis and permit improved orthodontic treatment. (Am J Orthod Dentofacial Orthop 2000;117:650-6)

Tooth agenesis: some common terms: oligodontia, anodontia, partial anodontia, hypodontia

Unopposed tooth eruption

- Craddock HL, Youngson CC. A study of the incidence of overeruption and occlusal interferences in unopposed posterior teeth. Br Dent J. 2004 Mar 27;196(6):341-8.
  - 86% of 155 unopposed teeth overerupted
  - 52% caused occusal interferences in function