TOOTH EROSION

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

Classic ADA North American Standards for Tooth Development

**TABLE 2.3** Chronology of Tooth Eruption, Primary Dentition

|-------|------|-------|---------------|---------|------|-------|------|-------|------|-------|

**TABLE 3.3** Chronology of Tooth Eruption, Permanent Dentition

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Eruption sequence

- Maxillary teeth: 6 1 2 4 5 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?

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

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

Fig. 16 Stage of root formation for the permanent mandibular canine and premolars of females.

Fig. 18 The extent of root formation in females.

Fig. 19 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
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

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

DONALD R. CAHILL AND SANDY C. MARXES, 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 gubernacular dentin, root formation, root eruption, and dental follicle in pre-eruptional eruption of a mandibular germs have been studied in 12 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 emergence of the eruptive pathway and formation of bone at the base of the bone crypt, the radiographic and histologic hallmark of tooth eruption. These data, together with the stereoscopic relationships of the dental follicle to some of the bone crypts 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 1978

Cahill and Marks Famous Experiment

Fig. 7. Radiographs depicting effect of removal of tooth crypt, dental follicle containing it, etc., during surgery. (a) 10–14 postnatal weeks. Arrow (*) shows base of crypt after removal of crown. (b) Operated side 1 week after surgery, showing radiolucent area below base of crypt indicating elimination of crypt pathway (arrows). (c) Operated side 2 weeks after surgery. Note radio-opaque area at base of bone crypt indicating area of bone formation (NB). D, upper side 3 weeks after surgery. Arrows show further elimination of crypt pathway. White line indicates plane of section of Fig.
Nature's Evidence that the dental follicle creates the eruption pathway

Five stages of tooth eruption:

1. Preeruptive movements
2. Intraosseous eruption
3. Mucosal penetration
4. Preocclusal eruption
5. Postocclusal eruption
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-α, EGF, TGF-β, and IL-1 are likely candidates for local molecular regulation
- Bone resorption is the rate-limiting step of this stage

- 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 increased 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 cause 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
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 BWXR5 or PANs useful if not sure
    - Sound not diagnostic (see permanent teeth)
  - Mobility

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
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!!

Conference Paper

Decompression as an Approach to Treat Ankylosis in Growing Children

Abstract: There is no greater treatment challenge for the dental practitioner than the ankylosed, non-vital permanent lower incisor in a young patient. Treatment options vary depending on the age of the patient, the patient's active growth potential, the root fusion status of the ankylosed tooth, and the viability of the underlying follicle. In this article, a case is reported where a decompression procedure was performed for a 11 year old patient with a non-vital, ankylosed, lower left central incisor. The decompression procedure involved the creation of a fenestration in the buccal cortex of the ankylosed tooth, allowing for the creation of a new periodontal ligament space. The patient's incisor was monitored post-operatively, and at 1 year follow-up, the incisor had regained function and vitality.

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 not ankylosed
- 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
**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

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

**ABO READING LIST**

Helmers-Westphal, DDS, DMD

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 controlling 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 defined and well-defined form of tooth agenesis, we have identified a distinct gene that affects the formation of second permanent 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 precise diagnosis and permit improved orthodontic treatment. (J Dent Res 2007;117:2694-8)

Tooth agenesis: some common terms: oligodontia, anodontia, partial anodontia, hypodontia
Unopposed tooth eruption

  - 86% of 155 unopposed teeth overerupted
  - 52% caused occlusal interferences in function