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Failure of Teeth Eruption

A Project Submitted to
The college of Dentistry, University of Baghdad, Department of
Orthodontics in Partial Fulfillment for the Bachelor of Dental Surgery

By

Zahraa' Khalid Mohammed

Supervised by:

Asst. Prof.

Dr. Ammar Salim Kadhum

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Certification of the Supervisor

I certify that this project entitled "**Failure of teeth eruption**" was prepared by the fifth-year student **Zahraa' Khalid Mohammed** under my supervision at the College of Dentistry/University of Baghdad in partial fulfilment of the graduation requirements for the Bachelor Degree in Dentistry.

Asst. Prof. Dr. **Ammar Salim Kadhum**

Date: April 30, 2023

Dedication

To my loving family,

To my loving mother whom without I would have never reached success

Dr. Khalida AlRawi,

To my younger brother who is the joy of my life,

To my dearest friends who never fail to make me smile and finally to the toughest person I have ever known, **myself.**

Acknowledgment

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List of abbreviations

Abbreviation	Meaning
PFE	Primary failure of eruption

Introduction

Tooth eruption is a continuous biological process by which developing teeth emerge through the jaws and the overlying mucosa to enter into the oral cavity (**Almonaitiene et al., 2010**).

Several studies have been performed to understand the process of tooth eruption better. The most common general symptoms during tooth eruption include anxiety (15%), diarrhea (13%), a combination of the two (8%), fever and increased salivation. Apart from general symptoms that end up to normal eruption of the teeth, several local and systemic factors have been reported to influence the eruption of teeth. The exact nature of the factors responsible for tooth eruption is not fully understood. It is believed that these factors influence the matrix formation and the calcification process. The most important local conditions that influence tooth eruption are eruption cysts, eruption sequestra, fibrous developmental malformations and dentigerous cysts. Systemic factors include Down's syndrome, cleidocranial dysostosis, hypothyroidism, hypopituitarism and achondroplastic dwarfism (**Balk J Stom, 2009**).

Most commonly, local factors cause mechanical obstruction to tooth movement into the oral cavity and are responsible for the failure of tooth eruption. Primary Failure of Eruption is defined as a cessation of tooth eruption before emergence that is not due to a physical barrier in the eruption path, does not result from an abnormal position, and has no systemic cause (**Bhuvanesarri and Chandrasekaran, 2018**).

Aims of the study:

To provide an insight about the process of teeth eruption, eruption theories, the mechanism and phases of eruption, failure of teeth eruption and its causative factors, and the primary failure of eruption.

CHAPTER ONE: REVIEW OF LITERATURE

Teeth Eruption

Eruption is the movement of the tooth from its developmental site in alveolar bone to its functional position in the oral cavity. It is a continuous multifactorial process, associated with alveolar bone remodeling, root elongation, cementum apposition and periodontal ligament formation. Eruption has been a matter of long historical debate. Many theories have been suggested in the matter related to the process of teeth eruption. Each theory has a perspective in the mechanism of teeth eruption (**Rabea, 2018**).

Tooth eruption represents a series of precisely regulated cascades of paracrine signaling events between epithelial cells of the enamel organ and ectomesenchymal cells of the dental follicle. These tightly regulated processes which bring about selective alveolar bone resorption in the coronal aspects of the erupting tooth and bone formation in the apical aspects of the tooth, are considered central to the process of tooth eruption (**Hendrik *et al.*, 2015**).

Tooth eruption should be regarded as a stage of tooth development which, through epithelial-ectomesenchymal interactions. It represents the very mechanism that allows the dental follicle to assume its fundamental role in the process of selective bone remodeling required for the movement of a tooth from its developmental position in bone to its functional position in the oral cavity (**Hendrik *et al.*, 2015**).

Theories of teeth eruption

There is no consistent understanding of the cause behind tooth eruption. The etiology behind eruption and explanation of the eruption mechanism seems to be essential to perform etiology-based treatment. Why a tooth begins eruption and what enables it to move eruptively and later to end these eruptive movements is not known. Pathological eruption courses contribute to insight into the etiology behind eruption (**Kjaer, 2014**).

The mechanism of eruption depends on the correlation between space in the eruption course, created by the crown follicle, eruption pressure triggered by innervation in the apical root membrane, and the ability of the periodontal ligament to adapt to eruptive movements. Animal studies and studies on normal and pathological eruption in humans can support and explain different aspects in the different theories. Understanding the etiology of the eruption process is necessary for treating deviant eruption courses (**Kjaer 2014**).

1- Root formation theory

The root formation theory assumes that the proliferating root encounters a fixed structure; and the apically directed force is converted into a reactive occlusal force that causes coronal movement of the erupting tooth (**Figure 1**)(**Nanci, 2013**).

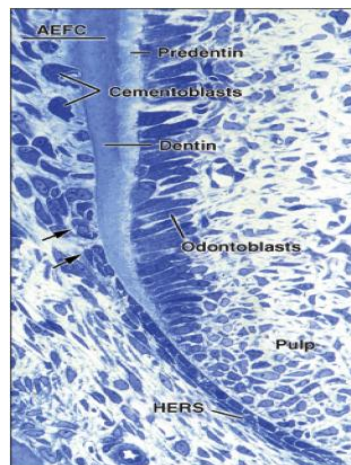


Figure 1: Photomicrograph showing root formation (Nanci, 2013)

However, there are facts refuted this hypothesis such as; rootless teeth can erupt, some teeth erupt greater distance than the total root length; and the teeth erupt after completion of root formation or when the tissue forming the root is removed. In addition, the onset of root formation does not coincide with the eruptive movement. Moreover, newly formed dentin at root apex is unmineralized and can be deformed by trauma (**Topouzelis et al., 2010**).

2- Cushioned hammock theory

It was proposed by Harry Sicher. This theory assumes that ligament (cushioned hammock ligament) below a tooth is responsible for eruption (**Figure 2**) (**Rabea, 2018**).

However, the ligament described by Sicher was an artifact in slide preparation (**Edward, 2002**).

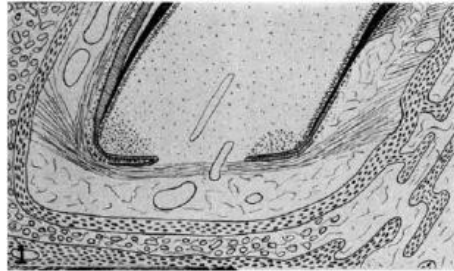


Figure 2: Diagram of basal end of rat incisor showing cushioned hammock ligament (Rabea, 2018)

3- Vascular pressure/blood vessel thrust or hydrostatic pressure theory

This theory suggests that a local increase in tissue fluid pressure in the periapical region is sufficient to move the tooth. This is debatable because root and local vasculature excision, does not prevent tooth eruption (**Yim, 200**It was reported that the hydrostatic pressure theory occurs during postemergent eruption. This is due to that dental follicle secrete mediators, such as vascular endothelial growth factor (VEGF), that cause angiogenesis and so increase in the apical tissue pressure that lead to tooth eruption. Moreover, hydrostatic pressure theory was supported by several studies that confirm tooth eruption after a local injection of vasodilators. Whereas injection of vasoconstrictors caused decrease in the rate of eruption (**Frazier-Bowers and Hendricks, 2015**).

4- Dental follicle theory

The follicular theory postulates that the dental follicle is capable of inducing. Bone resorption above the developing crown and bone apposition below it (**Figure 3**). This enables the formation of an eruptive path to occur through which

the tooth will be passively conducted. In osteopetrotic animal, which lack a factor that stimulates differentiation of osteoclasts, eruption is prevented, because no mechanism for bone removal exists. However, local administration of this factor, colony-stimulating factor 1 (CSF-1), permits the differentiation of osteoclasts and eruption occurs (Nanci, 2013).

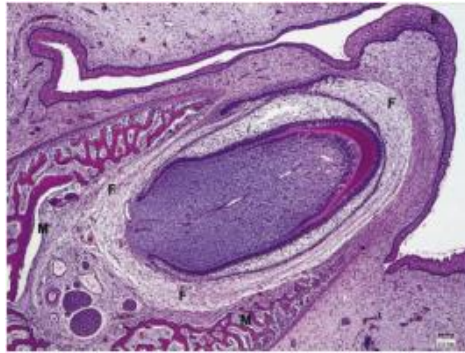


Figure 3: Photomicrograph of a developing dog tooth indicating the position of the dental follicle around the developing tooth, developing mandibular alveolar bone and overlying oral epithelium (Nanci, 2013)

5- Bone remodeling theory

This theory based on bone resorption occurs coronally and bone apposition occurs apically. The dental follicle is the source for osteoblasts and osteoclasts (Nanci, 2009).

The asymmetric bone remodeling around the tooth is thought to be responsible for teeth moving into the oral cavity, and therefore it is the most accepted theory (Wang, 2013).

Whether bone remodeling that occurs around teeth causes or is the effect of tooth movement is not known, and both circumstances may apply. The strongest evidence in support of bone remodeling as a cause of tooth movement comes from a series of experiments in dogs. When the developing premolar is removed without disturbing the dental follicle, or if eruption is prevented by wiring the tooth germ down to the lower border of the mandible, an eruptive pathway still forms within the bone overlying the enucleated tooth as osteoclasts widen the gubernacular canal (Figure 4). If the dental follicle is removed,

however, no eruptive pathway forms. Furthermore, if a metal or silicone replica replaces tooth germ and so as long as the dental follicle is retained, the replica will erupt, with formation of an eruptive pathway. It is concluded that programmed bone remodeling can and does occur (i.e., an eruptive pathway forms in bone without a developing and growing tooth). However, the conclusion cannot be drawn that the demonstration of an eruptive pathway forming within bone means that bone remodeling is responsible for tooth movement unless coincident bone deposition also can be demonstrated at the base of the crypt where its prevention can interfere with tooth eruption (Nanci, 2009).

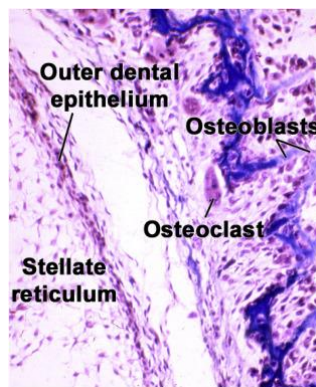


Figure 4: Photomicrograph showing bone remodeling (Srinath et al., 2013).

Mechanism implicated in tooth eruption

Active eruption: the movement of the tooth from its developmental site in alveolar bone to its functional position in the oral cavity, it compensates for the lengthening of clinical crown (Srinath *et al.*, 2013).

Passive eruption: Passive eruption is characterized by the apical shift of the dentogingival junction. As this occurs, the length of the clinical crown increases as the epithelial attachment migrates apically. Although the movement of teeth into function has been the subject of extensive research, there is no consensus regarding the mechanism involved (Yim *et al.*, 2008).

Gubernacular cord (Figure 5) and canal are involved in the tooth eruption process (Goldberg, 2022). The Gubernacular Canal is an anatomical structure

that can be seen on Cone Beam Computed Tomography, connecting the dental lamina of an unerupted tooth the overlying gingivae at the site of normal eruption, The gubernaculum dentis is composed of a channel. The gubernacular canal, which encloses a connective tissue cord, known as the gubernacular cord, they are both involved in tooth eruption: vascular pressure, periodontal ligament maturation, changes at the alveolar bone level. Widening of the gubernacular canal allows the tooth to erupt (**Radia, 2022**).

In addition to maturation of the periodontal ligament, changes in the alveolar bone, collagen fibers maturation, and cementum formation. Transcription factors and growth factors are integral parts of these multifactorial events. Hormones such as of IL-1 and PTHrP are located in the stratum reticulum. Metalloproteinases suggest that tooth eruption is also implicated in these multifactorial processes. Genesis of osteoclasts and osteoblasts, tooth eruption molecules, biological aspects of the periodontal ligament are involved in this process. Other events may also be implicated: such as pulpal pressure, pulpal growth, root lengthening, traction by periodontal fibroblasts acellular and cellular cementum formation, and vascular pressure. Developmental anomalies of tooth are recognized, including flexion, dilacerations, conrescence, fusion, shortened roots, accessory roots, hypercementosis, and enamel pearl (**Goldberg, 2022**).

Therefore, there is no single process responsible for tooth eruption, but complex effects combined shed lights on the different mechanisms involved in tooth eruption (**Goldberg, 2022**).

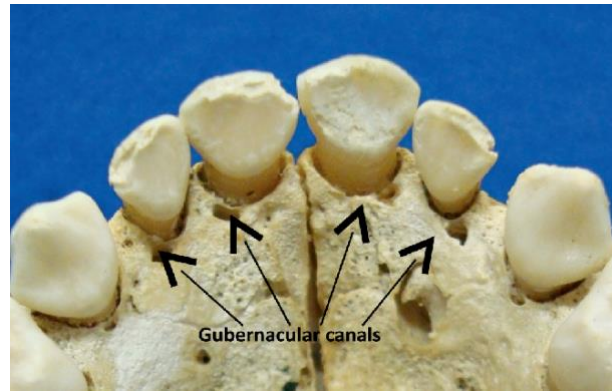


Figure 5: Dry child skull, Gubernacular canals located in the alveolar bone crest, behind the mandibular deciduous incisors (Ferreira *et al.*, 2013)

There is no consensus in the mechanisms involved in teeth eruption, but it includes three successive stages:

- Pre-eruptive movement
- Intra-osseous
- Pre-and post- occlusal eruption

Tooth eruption phases

Pre-eruptive phase:

Also named primary eruption. It is the movement of teeth germs. It is best thought of as the mean by which the teeth are positioned within the jaw for eruptive movement. The Pre-eruptive phase starts from the end of early bell stage until the beginning of root formation (Srinath *et al.*, 2013). In pre-eruptive tooth movement, the posterior teeth move backward, whereas anterior teeth move forward (Goldberg, 2022).

Eruptive phase:

Tooth movements during eruptive phase are subdivided into intraosseous and supraosseous stages. The eruptive phase begins with the onset of root formation and terminates by tooth appearance in the oral cavity, just before function (pre-functional phase) (Nanci, 2013). The eruptive phase includes 4 stages: Root formation, movement, penetration in the oral cavity, and occlusal

contact (**Goldberg, 2022**). The rate of tooth eruption depends on two different phases:

Intraosseous phase: Rate 1-10 $\mu\text{m}/\text{day}$

Extraosseous phase: Rate 75 $\mu\text{m}/\text{day}$ (**Goldberg, 2022**).

Post-eruptive phase:

This movement maintains the tooth position in occlusion by compensation for occlusal and proximal tooth wear. The post-eruptive phase starts when the teeth attain occlusion and continues for as long as each tooth remains in the oral cavity (functional phase) (**Srinath et al., 2013**).

That maintain the teeth contact as the jaws continue to grow, and compensate for occlusal and proximal wear. Two sets occur in human (diphiodont): (deciduous vs permanent dentition). At some moment: the two dentitions are present (occurrence of a mixed dentition) the formation of the furcation dentin, followed by the merge and occurrence of contact between lower and upper molars. This signifies the end of the tooth eruption (**Figure 6**) (**Goldberg, 2022**).

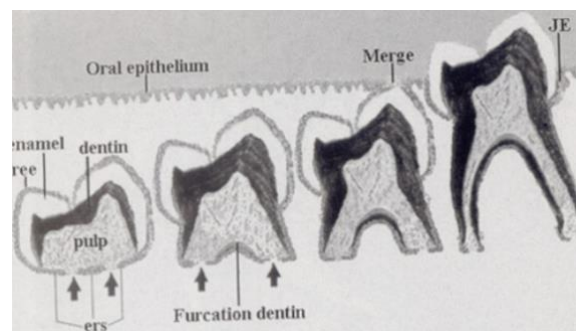


Figure 6: The formation of the furcation dentin (Goldberg, 2022)

Delays in teeth eruption

A delay in tooth eruption of up to 12 months may be of little or no importance in an otherwise healthy child. Delays often result from such local factors as a tooth in the path of eruption, insufficient space in the dental arch, or dental infection. Ectopic positioning and impaction most often affect the third

molars, second premolars, and canines, possibly because these are the last teeth to erupt (**Holt *et al.*, 2001**).

Failure of teeth eruption

The failure of teeth eruption occurs when one or more teeth fail to erupt in the mouth or other mechanical factors (**Bhuvanewarri and Chandrasekaran, 2018**). Several local and systemic factors have been reported to influence the eruption of primary, as well as the permanent teeth. Some of the local factors include eruption cysts, eruption sequestra, fibrous developmental malformations and dentigerous cysts. The systemic factors include Down's syndrome, cleidocranial dysostosis, hypothyroidism, hypopituitarism and achondroplastic dwarfism. Finally, a condition called primary failure of eruption has been found to be associated with the parathyroid hormone 1 receptor mutation (**Boka *et al.*, 2009**).

Several studies have shown variations in the ages at which individual primary teeth erupt as well as variations of eruption pattern between different ethnic and racial groups. Other suggested factors, which affect the eruption time, may include gestational period, diseases, nutritional status, growth and climate. In addition to genetic factors, environmental factors such as maternal smoking, height and weight of a newborn at the time of birth and nutrition status has shown to play a role in the eruption of the first primary tooth. A few reports have focused on the discerning effect of nutrition in early age of a child, including breast milk (**Alshukairi, 2019**).

According to **Suri *et al.* (2004)**, the factors can be classified as:

- Systemic factors:
 - Endocrine diseases :
 - Congenital hypothyroidism “ cretinism “
 - Hypoparathyroidism
 - Hypopituitarism

- Pseudohypoparathyroidism type 1A “Albright hereditary osteodystrophy”
- Chronic drug therapy:
 - Bisphosphonate therapy
 - Medications that inhibit prostaglandins pathway, e.g., antineoplastic chemotherapy
- Malnutrition
- Primary failure of eruption
- Genetic conditions:
 - Amelogenesis imperfecta
 - Apert syndrome
 - Cherubism
 - Chondroectodermal dysplasia (Ellis van Creveld syndrome)
 - Cleidocranial dysplasia
 - Familial adenomatous polyposis
 - Infantile osteopetrosis (Albers–Schonberg disease)
 - McCune–Albright syndrome
 - Mucopolysaccharidosis
 - Trisomy 21 (Down syndrome)
- Local factors:
 - - Causes related to the permanent tooth successor
 - Dilaceration
 - Ectopic eruption
 - Injuries to primary teeth
 - Premature loss of deciduous teeth
 - Ankylosis

- Obstruction

This can happen due to:

- Fibrous gingival tissue and/or gingival enlargement
- Odontogenic and non-odontogenic tumors and cysts
- Odontome
- Supernumerary tooth
- Chronic local infection
- Arch-length deficiency and skeletal pattern
- Malocclusion
- Oral facial cleft
- Environmental factors

Systemic factors

Endocrine diseases:

- Congenital hypothyroidism (cretinism) (**Figure7**): childhood hypothyroidism known as cretinism is characterized by thick lips, large protruding tongue (macroglossia), malocclusion and delayed eruption of teeth (**Dudhia and Dudhia, 2014**).



Figure 7: An infant with cretinism, retention of primary teeth and obstruction of permanent teeth (Garcia, 2014).

- Hypoparathyroidism: Hypoparathyroidism has known adverse effects on the development of tooth and its eruption that may manifest as the retention

of primary teeth, impaired upper and lower jaw growth, alteration of facial dimensions and a shortfall of coordination in lower jaw growth that affects the development of dentition (**Chalapathi *et al.*, 2018**).

In permanent dentition, third molars are the most frequently impacted teeth and fail to erupt followed by the canines. In decreasing order of frequency, impaction is seen with mandibular premolars, mandibular canines, maxillary premolars, maxillary central incisors, maxillary lateral incisors and mandibular second molars. First molars and maxillary second molars are rarely affected. When impaction involves few teeth, the condition is localized but when it involves multiple teeth, the condition becomes generalized. It is not a rarity to find impaction of a single tooth but a rarity to find multiple impacted teeth. Impaction can be owing to physical reasons like insufficient space, early closure of space, crowded arches or, thickened overlying bone or, soft tissues. The factors considered pathological causing impactions, likewise, may be local including overlying cysts or, tumors and trauma or, systemic conditions including hypoparathyroidism etc. Various syndromes, metabolic and hormonal disorders are attributed to impactions (**Chalapathi *et al.*, 2018**).

- Hypopituitarism: In pituitary dwarfism there may be various anomalies of the dental apparatus, from the morphological profile and in terms of development. The clinical picture presents aspects of hypodontia, delayed tooth eruption, abnormalities of tooth shape and size, and double or impacted teeth (**Ferrante *et al.*, 2017**).

Genetic conditions:

Genetics has an significant role in development, genetic factors are included in the multifactorial nature of tooth eruption which may be disturbed at any stage of development and result either in non-emergence or early ceasing of emergence after a tooth appear into the oral cavity (**Pytlik,1991**).

General delay in the eruption of teeth is reported in some families. The medical history of patient might be completely unremarkable, with delayed teeth eruption as the only finding, it has been suggested that there is a gene for tooth eruption, and its —delayed onset|| might be participate in delayed teeth eruption in —inherited retarded eruption (**Blankenstein *et al.*, 2001**).

- Amelogenesis imperfecta (**Figure 8**): Amelogenesis imperfecta can be associated with significant aesthetic concerns, dental sensitivity, loss of vertical dimension with an increased frequency of caries, anterior open bite, delayed eruption, tooth impaction, and associated gingivitis or periodontitis (**Hartsfield, 2016**).



Figure 8:The clinical phenotypes vary marked in people with amelogenesis imperfecta as seen in these cases of generalized hypoplastic AI (A),hypocalcified AI with heavy calculus formation and gingival inflammation (B) and the orange brown coloration often seen in the hypomaturational AI type (C). This panographic radiograph (D) shows the markedly reduced contrast typically seen between the enamel and dentin due to the reduced mineral content seen in hypomaturational AI (Carlson, 2014).

- Apert syndrome (**Figure 9**): In individuals with Apert syndrome, an underdeveloped upper jaw can lead to dental problems, such as missing teeth, irregular tooth enamel, and crowded teeth leading to failure of eruption of teeth due to the lacking space (**Saberi and Shakoopour, 2011**).



Figure 9: Apert syndrome (Hered, 2023).

- Cherubism (**Figure 10**): Enlargement of the cervical lymph nodes that contribute to the patient's full-faced appearance, a V-shaped palate with a high arch, early loss of primary teeth and displaced, impacted, supernumerary and missing teeth are common findings in patients with cherubism (**Papadaki *et al.*, 2012**).



Figure 10: Front view of the oral cavity of a 12-year-old boy with cherubism (Lahfidi *et al.*, 2022)

- Cleidocranial dysplasia (**Figure 11**): mainly characterized by a pathognomonic deformity of the skull (persistently open skull sutures with bulging calvaria), hypoplastic midface, lack of eruption of permanent teeth, supernumerary teeth, hypoplastic or aplastic clavicles, and multiple other malformations. Special problem for the dentists due to a large number of abnormalities: highly arched palate, cleft palate, delayed union of mandibular symphysis, delayed tooth eruption, dental root and crown abnormalities, crypt formation around impacted teeth, and ectopic teeth.

Dental abnormalities are sometimes a sole clinical sign of the mutation (Impellizzeri *et al.*, 2018).



Figure 11: facial and intraoral photographs of patient suffering from Cleidocranial dysplasia (Zhu *et al.*, 2018).

- McCune–Albright syndrome (**Figure 12**): Dental findings observed in a series of fibrous dysplasia included malocclusion, delayed eruption, tooth displacement and tooth anomalies like tooth rotation, oligodontia and taurodontism. They occur due to direct activation in tooth mutations or indirect effect due to proximity to abnormal bone. The dental findings observed were anterior cross bite, spacing between teeth and labial tipping of 41, 42 (Aravinda, 2013).



Figure 12: Images of a person with McCune-Albright syndrome with distinct malformations of the face and the appearance of teeth (Asadi, 2020).

- Trisomy 21 (Down syndrome) (**Figure13**): Malocclusion is found in most people with Down syndrome because of the delayed eruption of permanent

teeth and the underdevelopment of the maxilla. A smaller maxilla contributes to an open bite, leading to poor positioning of teeth and increasing the likelihood of periodontal disease and dental caries. People with Down syndrome may have small teeth, which can cause spacing between the teeth. They also tend to have a small upper jaw. This may cause crowding of the teeth and may result in the permanent teeth being “impacted” because there is no room in the mouth for them to come in (Moraes *et al.*, 2007).

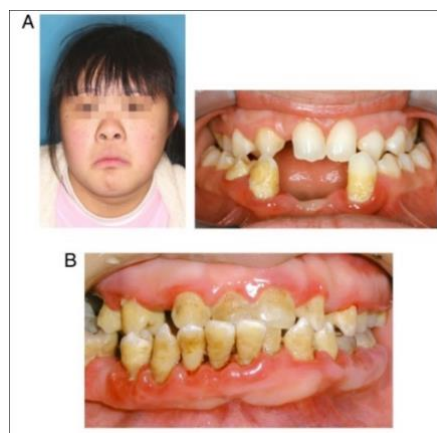


Figure 13: A. Gingival inflammation in lower dental arch, mixed dentition. Subject also has congenitally missing upper lateral incisors and all lower incisors; B. Periodontitis in permanent dentition of an adult Down syndrome individual (Cheng *et al.*, 2011).

Local factors

These are teeth related factors including:

Dilaceration

Dilaceration of a deciduous tooth can alter its resorption and delay eruption of the underlying permanent tooth, occasionally mandating extraction of the bent tooth. Severe dilacerations also can prevent eruption of the affected tooth and create endodontic or extraction difficulties (Neville and Chi, 2017).

Injuries to primary teeth:

Injuries to deciduous teeth can be the cause of delayed tooth eruption of the permanent teeth. Traumatic injuries can result in disruption of the odontogenesis result in dilacerations or physical displacing of the permanent germ **(Diab and elBadrawy, 2000)**.

Premature loss of deciduous teeth:

The eruption of the succedaneous teeth is frequently delayed after the early loss of deciduous teeth before resorption of their roots begins, it can be clarified by the abnormal changes that occur in the connective tissue covering the permanent tooth and the formation of dense, fibrous gingiva **(Suri et al., 2004)**.

Ankylosis of deciduous teeth:

It is a dental situation in which the roots of the tooth lose their normal attachment to the bone and become directly fused to the bone, extensive bony ankyloses of the primary tooth may prevent normal exfoliation, as well as the eruption of the permanent successor **(Raghoobar et al., 1991)**.

Exfoliation usually affecting the deciduous molars in all 4 dentin with the alveolar bone is the most common local cause of delayed quadrants, although the mandible is more commonly affected than the maxilla. Ankylosed teeth will remain standing while adjacent teeth continue to erupt through continued deposition of alveolar bone, giving the clinical impression of infraocclusion **(Figure14) (Camargo et al., 2011)**.



Figure 14: Ankylosis (Brin, 2011)

Obstruction

This can be due to several factors including:

- Mucosal barriers-scar tissue:

Gingival hyperplasia resulting from various causes (hormonal or hereditary causes, vitamin C deficiency, drugs such as phenytoin) might cause an abundance of dense connective tissue or acellular collagen that can cause inhibition of tooth eruption (**Figure 15**) (**Ekstrand et al., 2003**).



Figure 15: mucosal barrier (Andreason, 1994)

- Odontogenic and non odontogenic tumors : Dentigerous cysts are the most common jaw lesion in this category, followed by odontomas, unicystic ameloblastomas, and keratocystic odontogenic tumors. The least commonly encountered lesions are calcifying epithelial odontogenic tumors and calcifying odontogenic cysts. The highest frequency of association with an impacted tooth was seen for unicystic ameloblastomas, ameloblastic fibro-odontomas, adenomatoid odontogenic tumors, odontomas, and dentigerous cysts. In contrast, the lowest prevalence of such an association was observed for calcifying odontogenic cysts and keratocystic odontogenic tumors. The mandibular third molar was found to be the most commonly associated tooth for these lesions, except for adenomatoid odontogenic tumors, calcifying odontogenic cysts, and compound odontomas, for which the anterior maxillary teeth were found to be most commonly involved (**Mortazavi and Baharvand, 2016**).

Most of the jaw lesions associated with impacted tooth appear more commonly in men; however, a significant female predilection has been found for adenomatoid odontogenic tumors and calcifying epithelial odontogenic tumors. In addition, no gender predilection has been detected for calcifying odontogenic cysts, ameloblastic fibro-odontomas, and odontomas. Although most of these lesions tend to appear in the posterior aspect of the mandible, adenomatoid odontogenic tumors and compound odontomas frequently occur in the anterior portion of the maxilla. Moreover, no jaw predilection has been reported for calcifying odontogenic cysts and ameloblastic fibro-odontomas (**Mortazavi and Baharvand, 2016**).

- Odontomas: Odontomas are the most common odontogenic tumor in relation to unerupted teeth and failed teeth to erupt, they are mixed epithelial and ectomesenchymal tumors composed of dental hard and soft tissues. They are generally regarded as a tumor-like malformations or hamartomas, rather than neoplasms. Odontomas are the most common odontogenic tumor. There are two types of odontoma; compound and complex (**Wright and Tekkesin, 2017**).

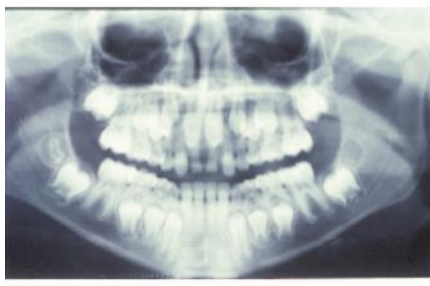
Odontomas are generally asymptomatic, often associated with delayed eruption or impaction of permanent teeth and retained primary teeth. In some occasional cases, pain, infection, regional adenopathies, alveolar bone expansion and tooth displacement may be present (**Kulkarni et al., 2012**).

- Arch-length deficiency and skeletal pattern: Arch-length deficiency is an etiologic factor for crowding and impactions. In a study of the relationship between formation and eruption of the maxillary teeth and the skeletal pattern of the maxilla, a shortened palatal length was found to delay the eruption of the maxillary second molar, although no delay in tooth

formation was observed, arch-length deficiency might lead to delayed teeth eruption, although more frequently the tooth erupts ectopically (Suda *et al.*, 2002).

- Supernumerary teeth: Supernumerary teeth can produce crowding, displacing, rotation, impaction, or delayed eruption of the related teeth. Mesiodens is the most common supernumerary tooth, followed by the fourth molar in the maxillary arch (Cunha *et al.*, 2001).

The presence of a supernumerary tooth is the most common cause for the failure of eruption of a maxillary central incisor. It may also cause retention of the primary incisor. The problem is usually noticed with the eruption of the maxillary lateral incisors together with the Failure of eruption of one or both central incisors Supernumerary teeth in other locations may also cause failure of eruption of adjacent teeth (Figure 16) (Garvey *et al.*, 1999).



A



B

Figure 16: A: Paired tuberculate supernumeraries with associated eruption disturbances, B: Failure of eruption of maxillary central incisors associated with the paired tuberculate supernumeraries illustrated in Figure A (Garvey *et al.*, 1999).

Environmental Factors:

a. Nutrition

Nutrition effects on timing of the teeth emergence can be considered in two different ways: if fatness is taken into account, a positive but only slight relationship can be found between weight and dental development (**Mugonzibwa et al., 2002**).

But if the effects of stunting or wasting considered as a result of a deficient nutrition, it is clear that eruption of both primary and permanent teeth will be delayed, except for earlier eruption of first permanent incisors and first permanent molars, which can show either earlier or later than normal emergence time (**Kochhar and Richardson, 1998**).

Nutrition can affect the weight of children:

- Low birth weight: Seow reviewed and identified retardation of dental growth and development in preterm babies (**Seow, 1997**).
- In another study children with a birth weight less than 1000g and gestational ages less than 30 weeks had the greatest lag in dental maturation (**Seow, 1996**).
- Malnutrition: The extremes of nutritive deprivation that the effects on tooth eruption have been shown (**Alvarez, 1995**).

b. Socioeconomic factors

In a number of studies, it has been shown that children from lower socioeconomic classes show later tooth emergence than children from higher socioeconomic classes (**Nonaka et al., 1990**).

It is suggested that children from high socioeconomic backgrounds gain better health care and better nutrition and therefore have their teeth to

erupt earlier than those with lower socioeconomic status. (Clements *et al.*, 2009).

c. Radiation damage

X-radiation, which is a form of electromagnetic radiation, has been shown to affect the eruption of the teeth. Impairment of root formation, damaging to the periodontal cell, and lacking of mandibular growth also appear to be related to tooth eruption disorders due to x-radiation (Piloni and Ubios,1995).

Primary Failure of Eruption

Primary failure of eruption (PFE) is a rare condition that can lead to posterior open bites, defined as incomplete tooth eruption despite the presence of a clear eruption pathway. Orthodontic extrusion is not feasible in this case because it results in ankylosis of teeth. It is difficult to diagnose and difficult to treat. PFE appears to be a condition that predominantly affects the molar dentition. The increased frequency of hypodontia in affected individuals and common findings of a family history regarding tooth eruption problems suggests a significant genetic component to the etiology of this rare condition (Hanisch *et al.*, 2018).

The term “primary failure of eruption” was first used in 1981 by William Proffit and Katherine Vig. This type of failure of eruption is usually caused by a genetic or familial background precursor. The prevalence of PFE is present in approximately 0.06% of the population. In this type of failure, teeth which are non-ankylosed, fail to erupt. These teeth also do not have a primary tooth that is blocking their path. These teeth have a tendency to erupt partially, but then fail to fully erupt as they would normally with time. Proffit stated that only the posterior teeth are the ones that are affected. The teeth which are distal to the affected tooth, are also impacted. Both the permanent and primary teeth can be

equally affected. This condition often results in a posterior open bite for patients who have primary failure of eruption. This issue can occur within any quadrant of the mouth (**Bhuvaneshwarri and Chandrasekaran, 2018**).

The failure of teeth eruption may be the result of some interference of the process of teeth eruption. Most commonly, local factors causing mechanical obstruction to the tooth movement into the oral cavity (**O'Connell, 1999**).

There are two different types of PFE Type I and II. Type I involves the failure of eruption of teeth distal to the most mesial affected tooth. Type II involves a greater eruption pattern, which is incomplete, among the teeth distal to the most mesial affected tooth. It is often difficult to diagnose between these two types of failure of eruption because the 2nd molar does not erupt for most patients until they are 15-years-old. Plenty of times, a patient will receive orthodontic treatment before they are 15. As a result, in order to properly diagnose between the two types, a patient needs to be over 15 and a definitive proof of un-eruption of the 2nd molar is required (**Bhuvaneshwarri and Chandrasekaran, 2018**).

PFE was further divided by Frazier-Bowers et al. into three different types in Type I, the mesial to distal teeth show a similar or severe lack of eruption potential, and in Type II, the teeth distal to the most mesial affected tooth show greater but still inadequate eruption potential. Patients affected by both Type I and II PFE are diagnosed as having Type III PFE (**Yamaguchi et al., 2022**).

According to Raghoobar et al., failure of teeth eruption can be categorized into the following: (1) primary retention that is defined as an arrest of the eruption process before the crown has penetrated the oral mucosa and (2) secondary retention that involves cessation of further eruption after the tooth has penetrated the oral mucosa (**Hanisch et al., 2018**).

Management

Managing teeth that have primary failure of eruption commonly includes extracting the affected teeth, followed by an orthodontic space closure or the placement of a prosthetic implant with a bone graft. This option can only be used in cases where a single tooth is affected. If multiple teeth are affected, a segmental osteotomy can be performed in order to bring the entire segment into occlusion. However, this procedure has not been found to be highly successful. These teeth not usually responsive to the orthodontic force. Studies have shown that ankylosis of these teeth can also occur when force is applied (**Bhuvanesarri and Chandrasekaran, 2018**).

Some figures showing the intraoral condition of an individual with Primary failure of teeth eruption:



Figure 17: Unerupted permanent teeth occlusal view, Primary Failure of eruption (Bhuvanesarri and Chandrasekaran, 2018).

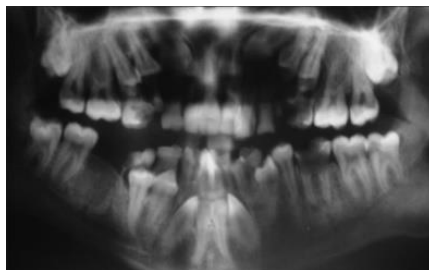


Figure 18: Unerupted permanent teeth occlusal view, Primary Failure of eruption (Bhuvanesarri and Chandrasekaran, 2018).

Discussion

Teeth eruption is one of the most important biological processes that need to be studied more, to focus on the way of how the teeth erupt and the failure of teeth eruption because there is no clear understanding of how it occurs. However, there are explanations and theories supported by animal studies and studies on normal and pathological eruption in humans.

Several theories tried to explain the process of tooth eruption, the most acceptable one is Bone remodeling theory, this theory is based on asymmetric bone remodeling around teeth coronally and apically.

The two essential mechanisms that are implicated in tooth eruption, these are the active and passive eruption. The former is described as the movement from alveolar bone to its functional position while the second is characterized by apical shift of dentogingival junction.

Failure of eruption could occur due to several factors, including systemic, local, and environmental factors. It is important to identify the cause of failure, as it will determine the type of intervention later.

Systemic factors related to hormonal imbalance and hormonal based disease are of great importance in relation to the failure of eruption of teeth. Management of these conditions may lead to the balance of hormones and eventually solve the problem standing in the way of normal eruption. However, some systemic factors are related to underlying syndromes and are could not be treated eventually without the intervention of physician. Local factors like non-odontogenic tumors, obstruction due lack of space etc are one of the most important factors causing the failure of eruption. PFE on the other hand is rare, it may lead to many serious condition like posterior open bite.

The exact cause of disorders of tooth eruption can be difficult to diagnose given the lack of knowledge about the eruptive process. The diagnosis is based

on clinical and radiographic characteristics and sometimes on the response to treatment. The treatment goals are to encourage the teeth to erupt into the oral cavity, usually through surgical exposure.

Managing cases with PFE include extraction of affected teeth and closing spaces by the orthodontist or place a prosthetic implant to establish satisfactory occlusion. In moderately severe cases, small-segment osteotomy and simultaneous elastic traction of the affected teeth will improve the level of the occlusal plane.

Conclusion

1. Eruption is a complex process with many contributing factors. Disturbances at the local, systemic or environmental levels may lead to a delay or failure of eruption.
2. Variation in the normal eruption of teeth is a common finding, but significant deviations from established norms should alert the clinician to further investigate the patient's health and development.
3. Delay or failure of tooth eruption might be indication of a systemic condition or an indication of altered physiology of the craniofacial complex.
4. It is important to identify the cause of failure, as it will determine the type of intervention.
5. The management of cases of failed teeth eruption can be accomplished by a series of procedures and modalities of treatment, from space creation to orthodontic treatment and sometimes prosthetic replacement.

Suggestions

- More studies about the nature of the process of teeth eruption.
- Researching more about the stimulating factors of teeth eruption.
- Understanding and researching more in depth in the theory of bone remodeling.
- Comparing between different races, age groups and gender in the matter of delay and failure of teeth eruption to find the dominance and find common factors.
- Researching more about the causative factors of failure of teeth eruption; systemically, locally and environmentally.
- More studies about the most causative factors.
- Research about PFE and its generation.
- Understanding the genetics behind PFE and the possibility of it being prevented.
- More case management reviews.

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