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Factor Affecting Sequence of Eruption of teeth

A Project

Submitted to the college of dentistry, University of Baghdad Department of Pediatric Dentistry in partial fulfillment of the requirement for the degree of B.D.S.

Submitted by

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Supervisor Declaration

This is to certify organization and preparation of this project has been made by the graduate student Alzahraa Abbas Abdullah under my supervision **at the College of Dentistry, University of Baghdad in partial fulfillment of the requirement for the degree of B.D.S.**

Signature:

Dr. Muna Abdullah Saleem

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Dedication

For my mother

For my father

For my brothers

For my friends

Thank you for your support, Love and Help

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1. 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 andddentigerous cysts. Systemic factors include Down's syndrome, cleidocranialdysostosis, hypothyroidism, hypopituitarism and achondroplastic dwarfism (Balk J Stom, 2009).

2. Development and Eruption of Teeth

The development and subsequent eruption of teeth is a complex process that occur at once with the growth of entire facial structures . The development of teeth begins at about 5-6 weeks of intrauterine life, and this development results from an interactions of oral epithelial cells and underlying mesenchymal cells,(20) primary and (32) permanent teeth developed and the developmental processes are similar for all teeth (Arathi, 2012). Development of teeth can be divided based on shape and physiologic changes into figure:

2.1 Growth: Growth can be divided into:

2.1.1 Crown Formation:

A) Bud stage/Initiation: The first epithelial invasion into the mesenchyme of jaws to form a tooth looks like a bud and called enamel organ. lack in initiation leads to absence of tooth development resulting in congenitally missing tooth and on abnormal initiation may lead to abnormal teeth .The initiation processes occur along each jaw which mark the beginning of development of teeth. The timing for anterior teeth is much earlier than posterior teeth (Ash et al, 2003).

B) Cap stage/Proliferation: Regular changes in size and proportion of the growing tooth germ is seen as the growth continues leading to the formation of a cap shaped enamel organ characterized by a shallow invagination on the deep surface of the bud. The enamel organ at this stage consisted of three layers, enamel knot and enamel cord. The three layers are the outer enamel epithelium, inner enamel epithelium and stellate reticulum (Ten Cate *et al*., 2003).

C) **Bell stage/Morphological and Histological Differentiation**: In this stage, cells undergo morphologic as well as functional changes by arrangement of formative cells along the future dento-enamel and dento-cemenal junction; it also outlines the size and the shape of the future crown and root (Gartner, 1999).

D) **Advanced Bell stage/Apposition:** In this stage ,there is apposition of the matrix of the dental hard tissues in a layer like pattern and at a definitive rate (Gartner, 1999).

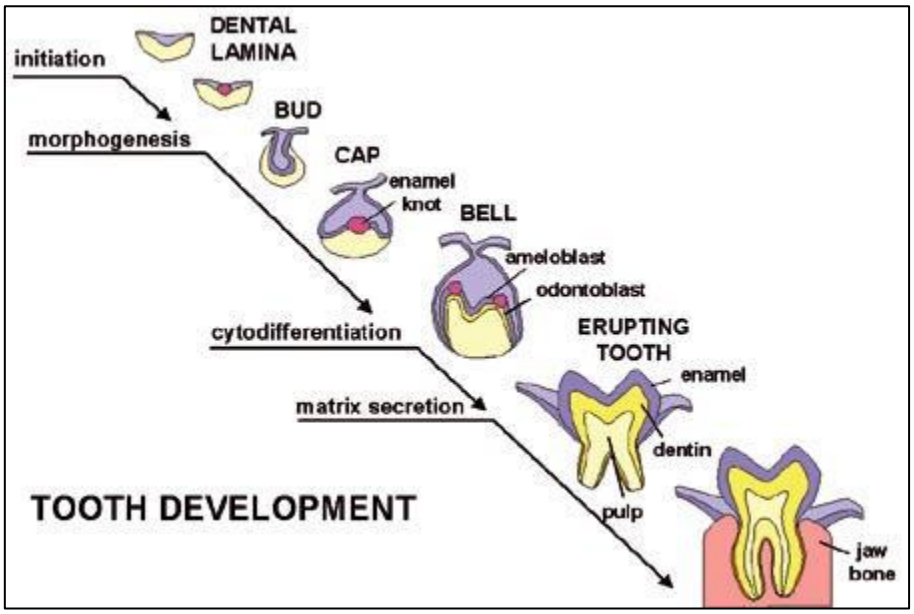

Figure 1:Tooth developmental stages (Thesleff and Mikkola, 2002)

2.1.2 Root Formation:

Root formation begins after enamel and dentin formation has reached the future cemento-enamel junction. Hertwige's epithelial root sheath moulds the shape of root and initiates radicular dentin formation (Liversidge and Molleson, 2004).

2.2 Calcification:

It involve precipitation of inorganic calcium salts within the deposit matrix, the process is clearly not one of simple precipitation; it depends upon the activity of living tissue.(Proudfoot*elal*., 1998).

2.3 Tooth Eruption:

Eruption is defined as movement of the tooth through the jaw tissues into the oral cavity.

Active eruption movements arise when root formation begins and thus it was assumed that eruptive force begins from periodontal ligament, but the mechanisms of tooth eruption are still not fully understood, periodontal ligament provides eruption force after the tooth has pierced gingiva but not during intra-osseous stage (Wise *et al*.,2002).

Nelson (2015) published chart of chronology of development of human dentition of both deciduous and permanent teeth Figure 2 and 3.

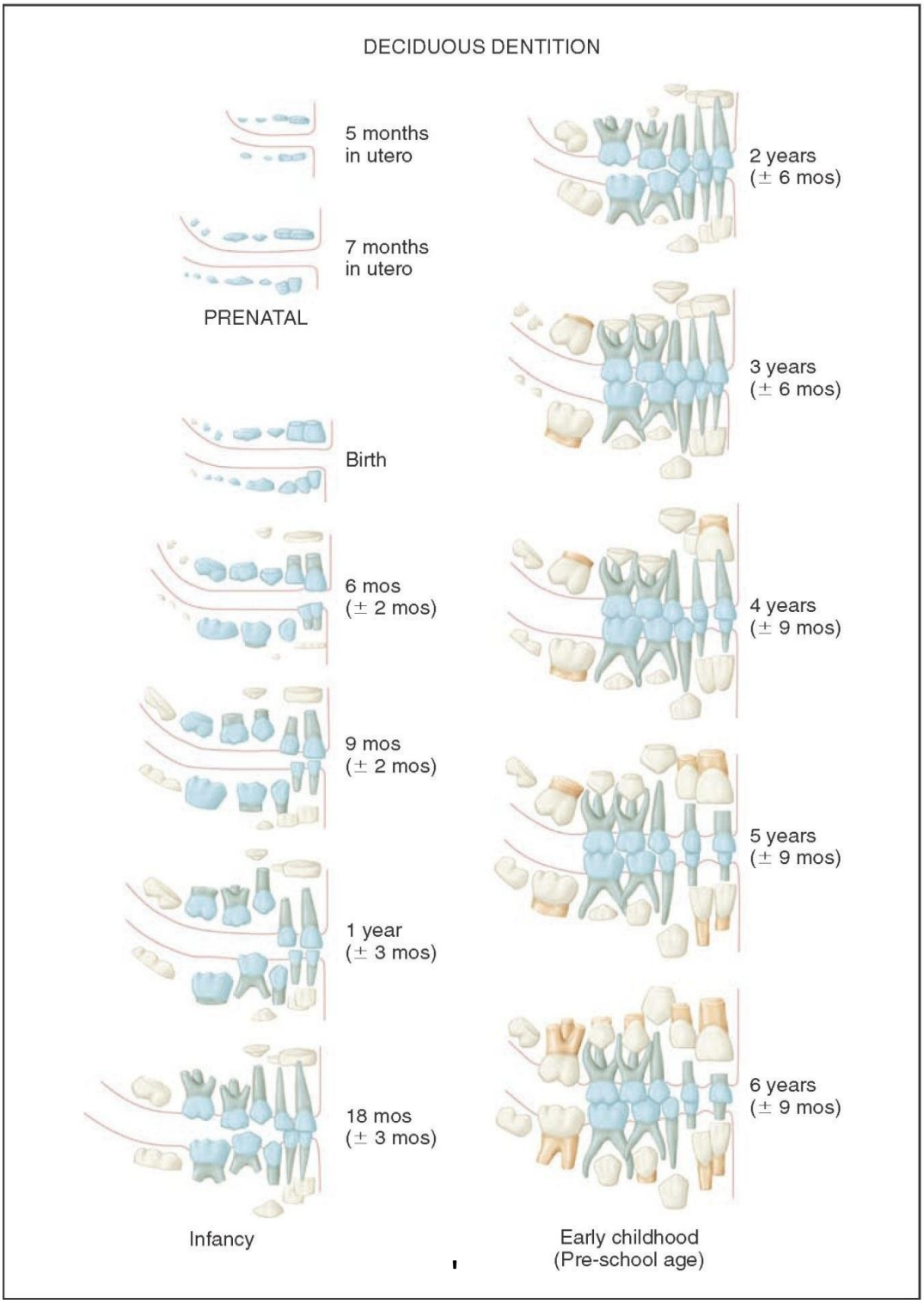

Figure 2: Chronology of development of deciduous dentition

(Nelson, 2015)

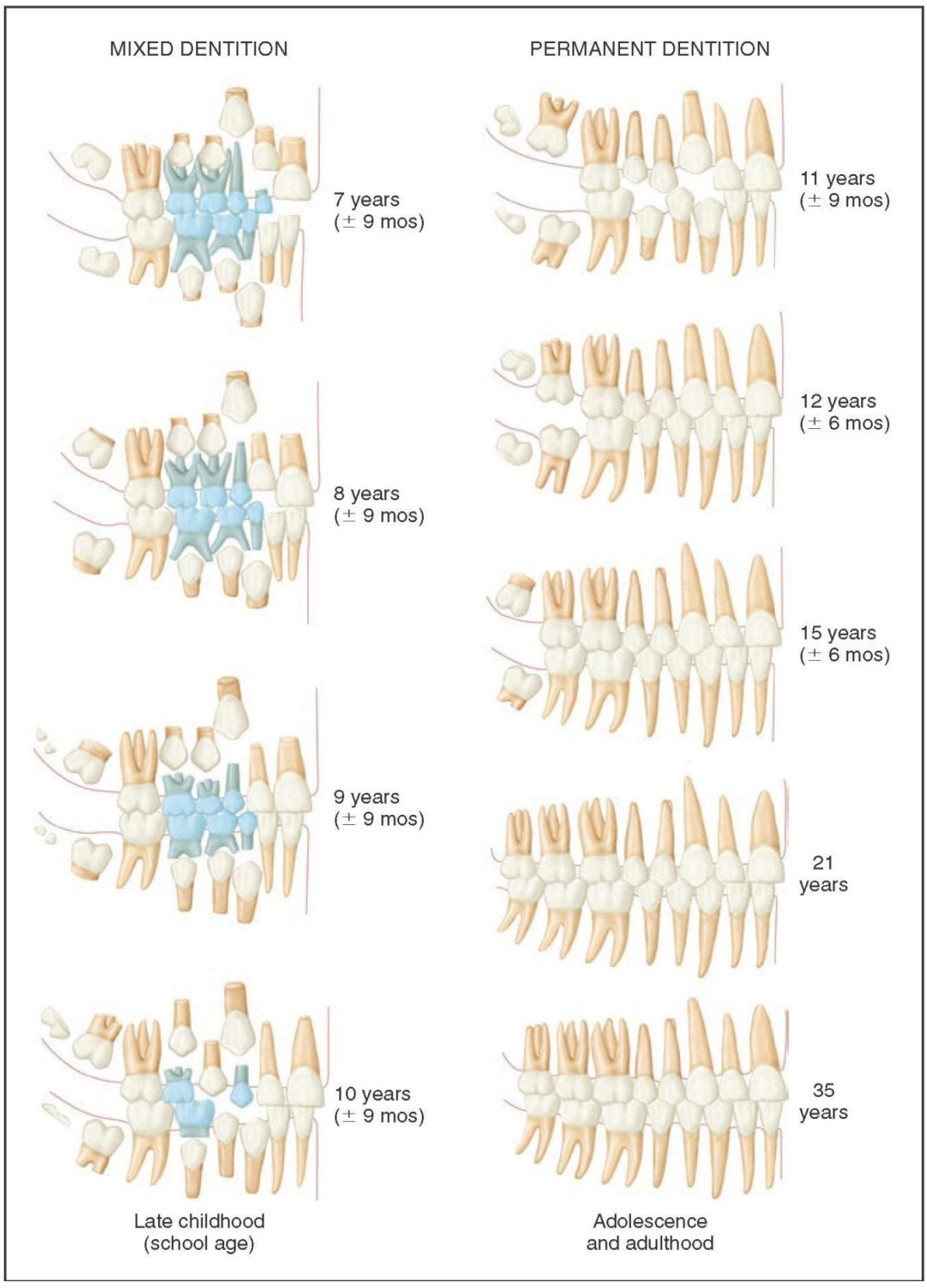

Figure 3: Chronology of development of Permanent dentition (Nelson,2015)

3. Occlusal Development

Stages of occlusal development can divided into the following development stages:

3.1 The Gum Pads (Neo-natal stage)

The alveolar arches of an infant at the time of birth are called Gum Pads. They are pink in color, firm and covered by a dense layer of fibrous periosterum (Friel s, 1954).

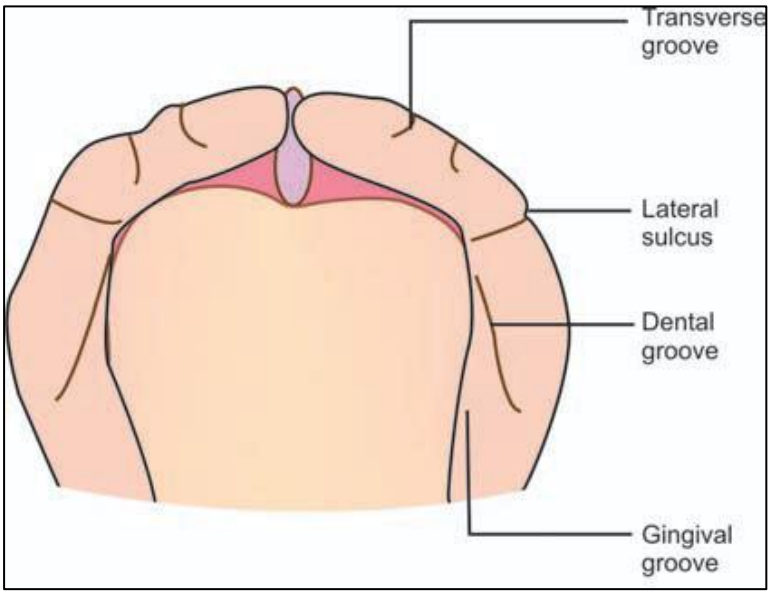

Figure 4:Gumpad, maxilla (Juneja, 2007)

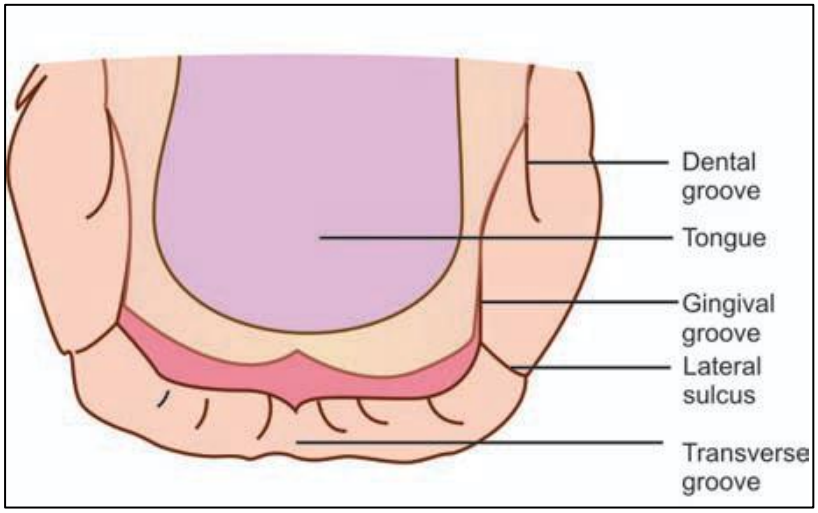

Figure 5: Gum pad, mandibular(Juneja, 2007)

Natal Teeth are Present at Birth

Neonatal teeth erupt during the first month these teeth looklike the deciduous teeth, that are contained enamel, dentine and pulpal tissue and usually without roots or there is very short root with them (Tapasyajuneja, 2007). The incidence of natal and neonatal teeth isestimated to be 1:1000 and 1:30000 respectively. These teeth are almost always mandibular incisors, which frequently display enamel hypoplasia. There are familial tendencies for such teeth. They should not beremoved if normal but removed if supernumerary ormobile (Gurkeeratsingh, 2007).

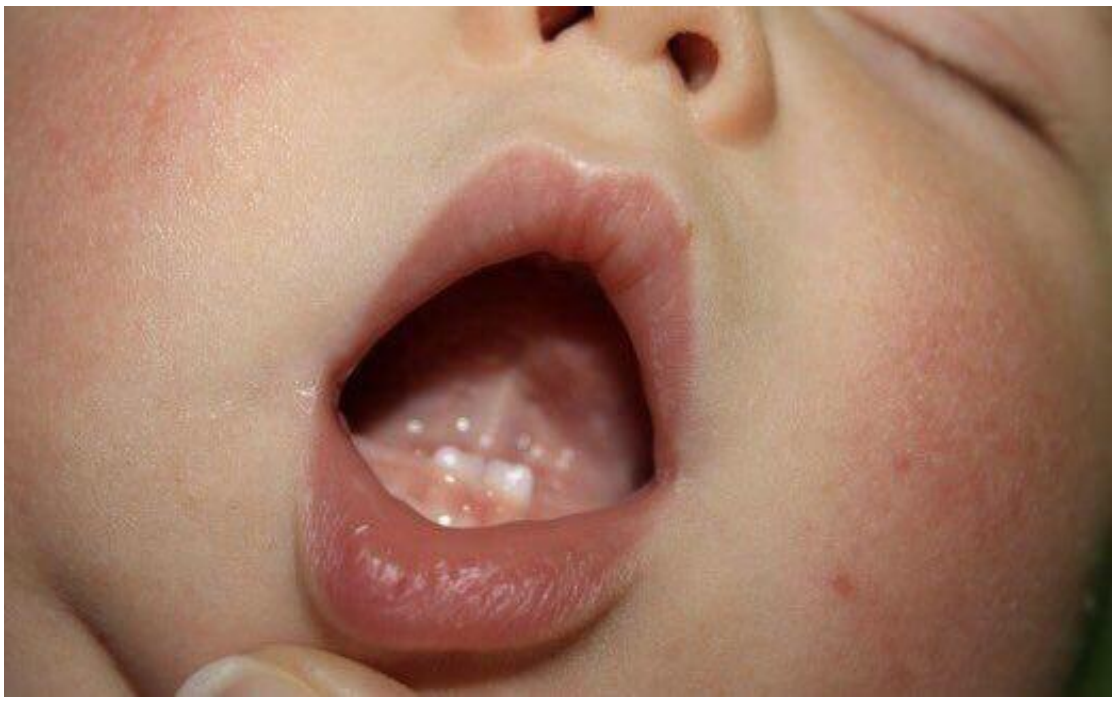

Figure 6: Natal tooth seen in a newborn (Singh, 2007)

3.2 The Primary Dentition Stage

The deciduous dentition stage starts from the eruption of the first deciduous tooth, usually the deciduousmandibular central incisors and ends with the eruptionof the first permanent molar, i.e. from 6 months to 6 years of postnatal life. Usually lower erupt before upper. (Tapasya Juneja; Gurkeerat Singh,2007). Sequence of eruption is as follow:-

A-B-D-C-E

3.3 The Mixed Dentition Stage

This is the period where teeth of both deciduous and permanent dentition are seen. It extends from 6-12 years of age(Gurkeerat Sing, 2007).

3.4 The Permanent Period

The permanent dentition forms within the jaws soonafter birth. Calcification begins at birth with the calcificationof the cusps of the first permanent molar andextends as late as the 25th year of life (Tapasyajuneja, 2007).

4. Physiology of Tooth Movement

For teeth to become functional, significant movement is needed to bring them into the occlusal plane (Nanci, 2009). The movements of the teeth are complicated and may be described as the followings:

4.1 Pre-eruptive tooth movement

Include all movement of the primary and permanent tooth germs through tissues of the jaw prior to their eruption (Ten Cate, 1994).

4.2 Eruptive Tooth Movement

Made by the movement of tooth from its site within the bone of the jaw to its functional position in occlusion (levers and Darling, 1983; Bhashar, 1991).

4.3 Post Eruptive Tooth Movement

This tooth movement is a complicated series of events to move the tooth in three - dimensional space. It is a passive process (unlike the pre eruptive and eruptive movement) that continues throughout the life time of the tooth. It results from attrition of the occlusal/ incisal and proximal surfaces of the tooth which then allows for continued occlusal movement and mesial drift of the teeth(Craddock and Youngson , 2004).

5. Developmental Disturbances Affecting the Teeth

There are many developmental disturbance affecting the teeth.(Junela, 2007). Can be divided in to :

5.1 Disturbances during Initiation of Tooth Germs

a. **Ectodermal dysplasia:** Complete or partial anodontia of both the dentitions along with the presence of malformed teeth Figure 7.

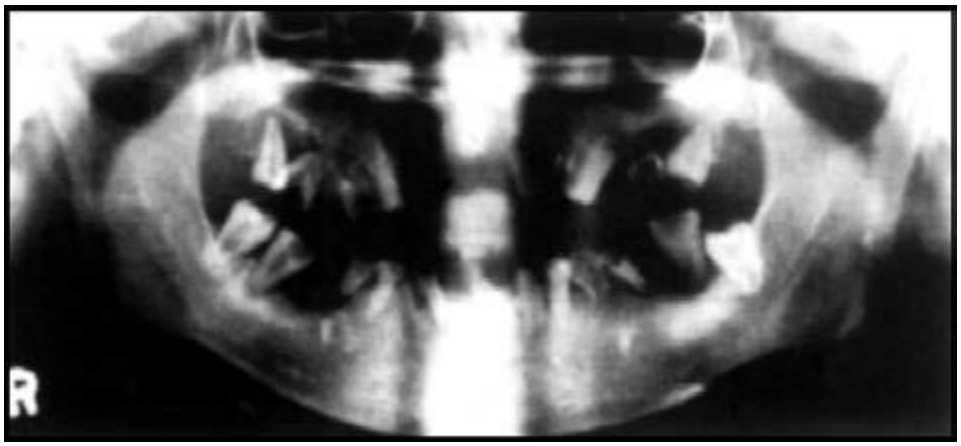

Figure 7: Orthopantomogram of a case with ectodermaldysplasia

b. **Anodontia:** Absence of 1 or more teeth due to failureof tooth bud initiation. Most commonly missing teeth are third molars (16%) followed by mandibular second premolars (4.4%), maxillary lateral incisor (1.7%) and maxillary second premolars (1.6%).
c. **Supernumerary and supplemental teeth:** Teeth in excess of the normal complement of teeth. The difference between the two is that supplemental teeth resemble normal teeth where as supernumerary teeth do not, e.g. of supernumerary teeth:

I- Mesiodens between maxillary central incisors.

II- Peridens located buccal to the arch

III- Distomolar distal to the third molar.

IV- Paramolar located buccal or lingual to molars.

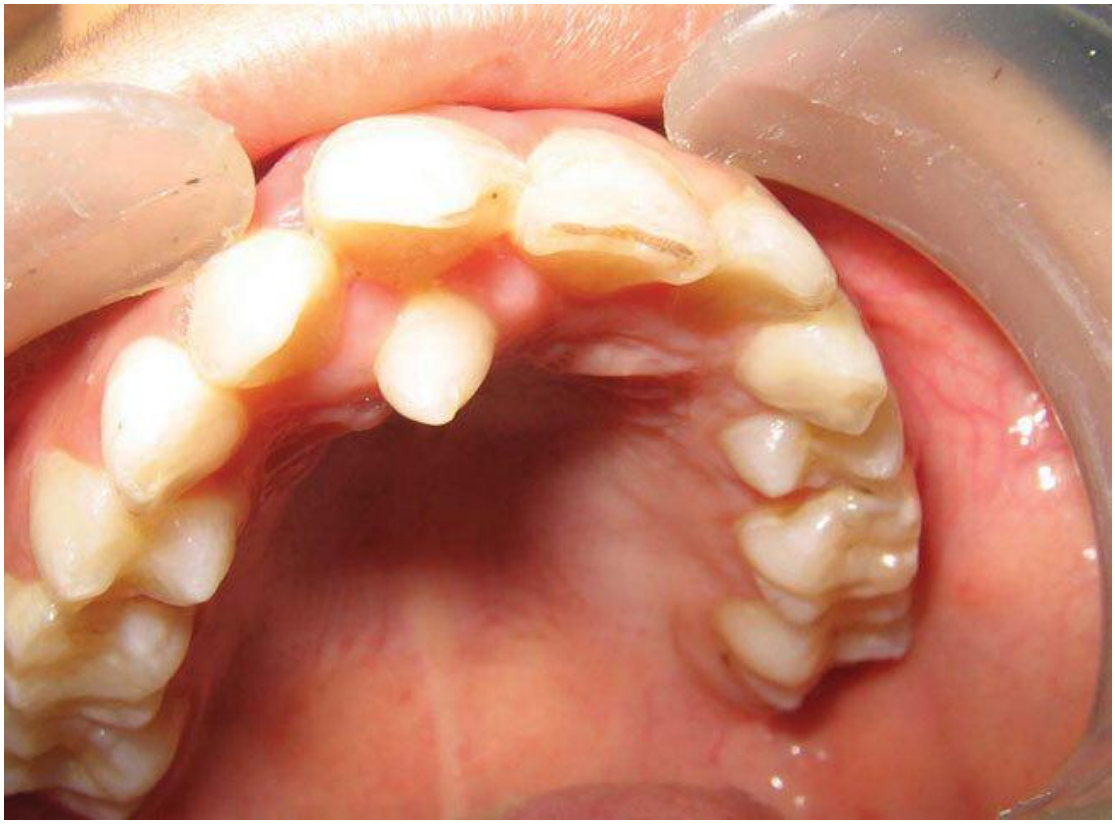

Figure 8: Supernumerary teeth (Singh, 2007)

d. Natal and neonatal teeth: These may be either supernumerary or deciduous teeth.

e. Predeciduous dentition: Aborted structures with capsof enamel and dentine.

f. Post permanent dentition: Teeth erupt after the loss of the permanent dentition, usually impacted accessory teeth.

5.2 Disturbances during Morpho-Differentiation of Tooth Germs

A. Hutchinson's incisors: Screw driver shaped notched incisors, e.g. in congenital syphilis.

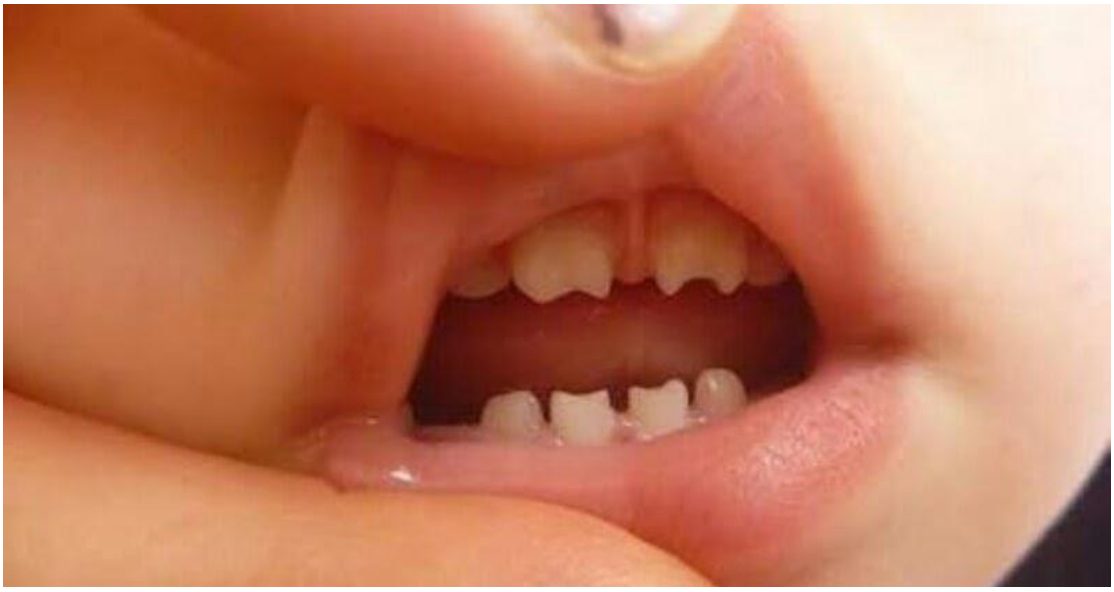

Figure 9:Hutchinsons incisor (singh, 2007)

B. Mulberry molars: Occlusal surface is narrower than the cervical margin and is made up of agglomerate mass of globules; seen in congenital syphilis.

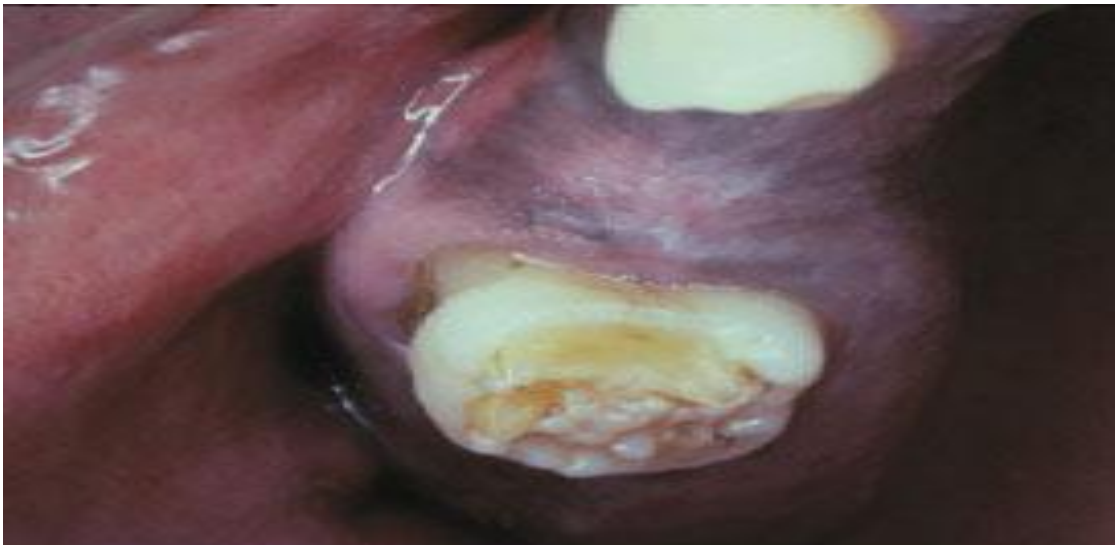

Figure 10: Mulberry molars (singh, 2007)

c. Peg shaped laterals: Proximal surfaces of the crown converge giving the tooth a conical shape.

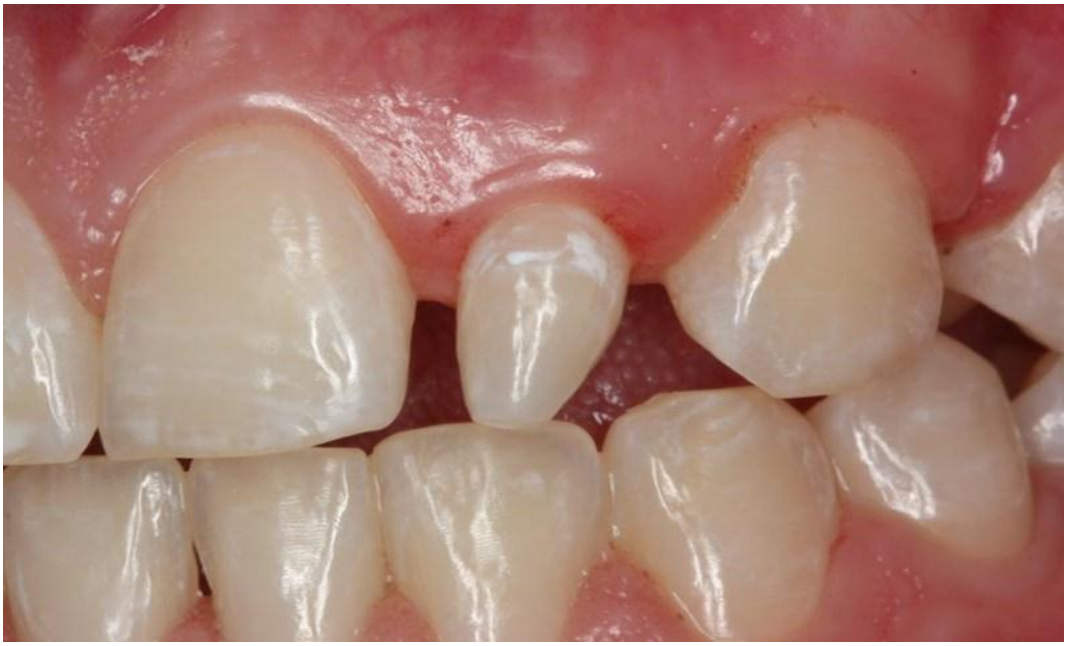

Figure 11: Peg shaped lateral (singh, 2007)

C. Macrodontia: Teeth larger than normal.

D. Microdontia: Teeth smaller than normal; most commonly the lateral incisor and third molars.

E. Dens in dente: Tooth invaginates before calcification,e.g. permanent maxillary lateral incisor.

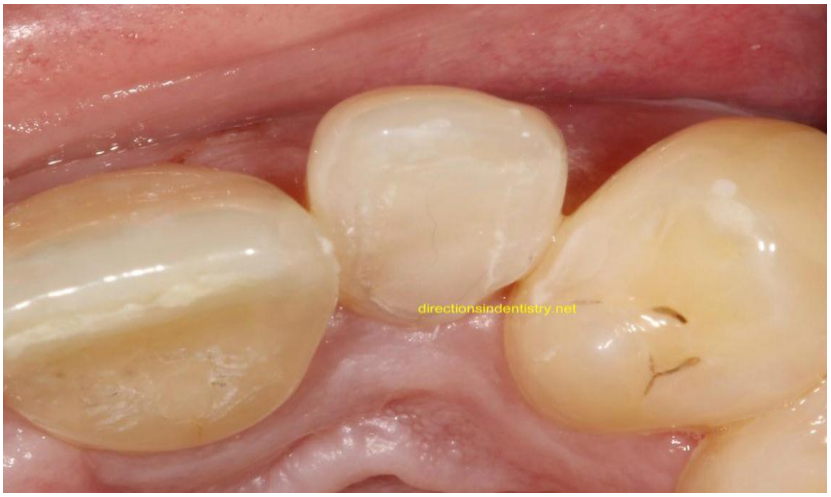

Figure 12: Dens in dente (singh, 2007)

F. Dens evaginatus: A tubercle or protruberance from the involved surface of the affected tooth; occursdue to proliferation or evagination of part of the inner enamel epithelium into the stellate reticulum. Seen in premolars.

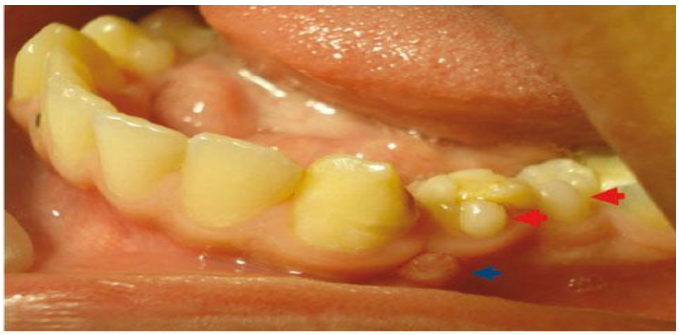

Figure 13: Dens evaginatus (singh, 2007)

G. Gemination: Single tooth germ splits into partiallyor fully separated crowns but with a common root and root canal.

H. Fusion: Two tooth germs unite to form a single large crown with two root canals; seen in incisors.

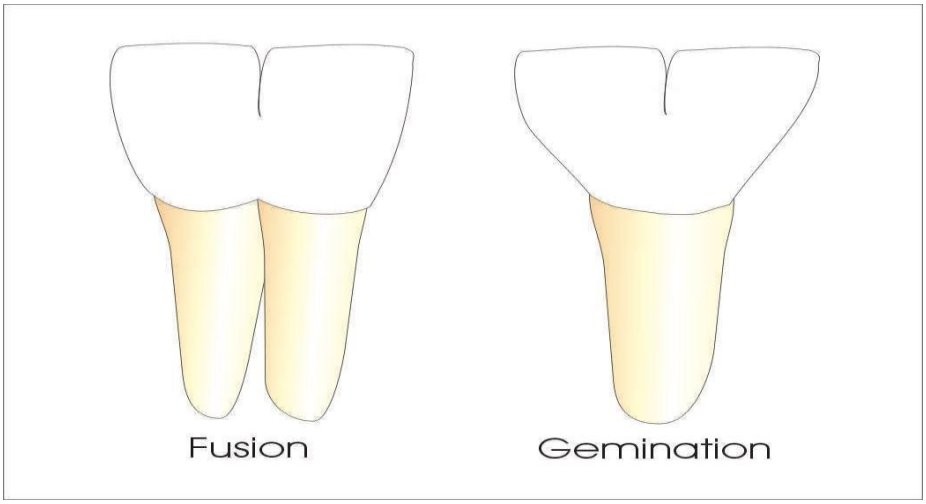

Figure 14: Gemination and fusion (singh, 2007)

I. **Dilaceration:** Twisting, bending or distortion of aroot.

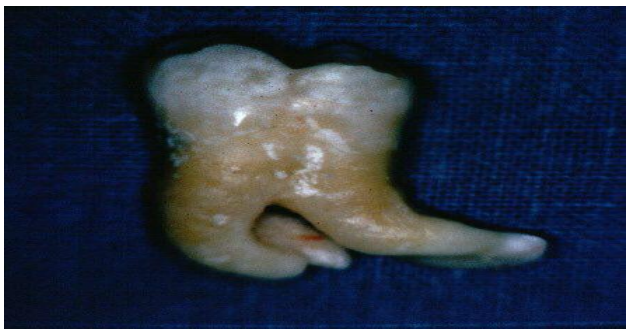

Figure 15: Dilaceration (singh, 2007)

J. **Taurodontism:** Enlargement of the body and pulp chamber of a multi-rooted tooth with apical displacement of the pulpal floor and bifurcation of the roots.

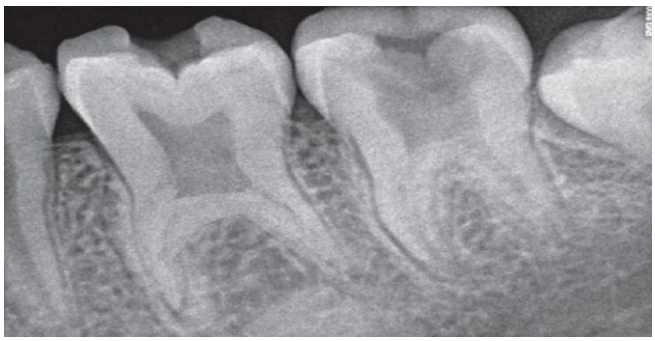

Figure 16: Taurodontism (Singh, 2007)

5.3 Disturbances during Apposition of Hard Tissue

a. Enamel hypoplasia: Reduction in the amount of enamel formed.

b. Amelogenesis imperfect: Hereditary disorder where in the quality and quantity of enamel formed is altered. Three types:

- Hypoplastic Defective matrix formation
- Hypocalcification Defective mineralization of matrix.
- Hypomaturation Immature enamel crystals.

c. **Dentinogenesis imperfect:** Hereditary developmental disorder of the dentine. The dentine appears grey to brownish violet, enamel frequently separatesfrom the defective dentine, roots become short, canals get obliterated, rapid attrition is seen.

d. **Dentinal dysplasia:** Premature loss of teeth, short roots.

e. **Shell teeth:** Roots fail to form, pulp chambers are wide.

f. **Odontodysplasia (Ghost teeth):** Enamel and dentine is defective and very thin.

g. **Pigmentation of enamel and dentine**

I – Erythroblastosis fetalis: enamel is green/blue.

II- Porphyria: red to brownish

III - Tetracyclines: brownish

h. Cemental hypoplasia: Reduced rate of cementum formation, e.g. hypophosphatasia.

i. Enamel pearls: Attached to the furcation area of maxillary molars.

5.4 Disturbances during Calcification of Hard Tissue

a. Enamel hypocalcification: Calcification is subnormal. It may be local, systemic or hereditary.

b. Interglobular dentin Areas of partially calcified dentine.

5.5 Disturbance during Eruption of Teeth

a. Concrescence: Cemental union of two teeth.

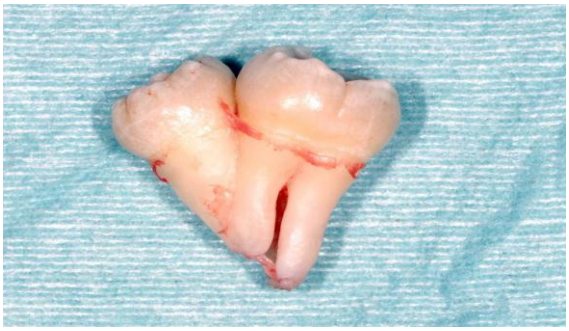

Figure 17: Concrescence (Singh, 2007)

b. Retarded Eruption: Due to endocrine disturbances, vitamin deficiencies, local causes.

c. Ankylosed teeth: Teeth fail to erupt to the occlusal level as they are fused to the bone.

6 Factors Affecting Sequence of Eruption of teeth

6.1 Local Factors: include

a. 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(Andreasen,1994; Ekstrand*et al.*,2003).

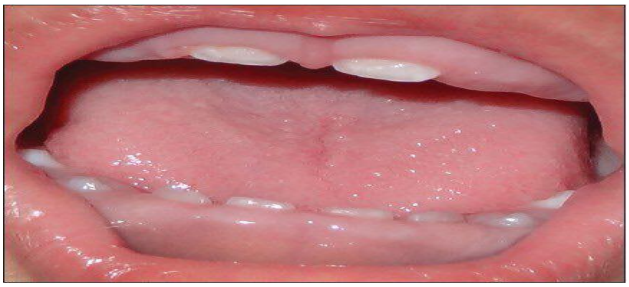

Figure 18: mucosal barrier (Andreason, 1994)

b.Supernumerary Teeth

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

c. Injuries to primary teeth

Injuries to deciduous teeth can be the cause of delay 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).

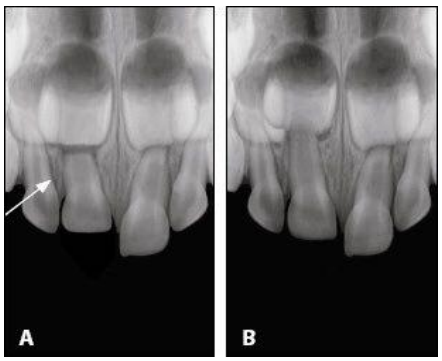

Figure 19: Injury to primary teeth (Diab, 2000)

d. 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).

e. 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 ankylosis of the primary tooth may prevent normal exfoliation, as well as the eruption of the permanent successor (Raghoebar*et al.*,1991)

The cause of ankylosis in the deciduous mostly follows a familial pattern and very slow root resorption was observed for most of the ankylosedteeth. Ankylosis resulting from the fusion of the cementum or dentin with the alveolar bone is the most common local cause of delayed deciduous tooth exfoliation usually affecting the deciduous molars in all 4 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(Brin *et al.*, 1988; Camargo *et al.*, 2011).

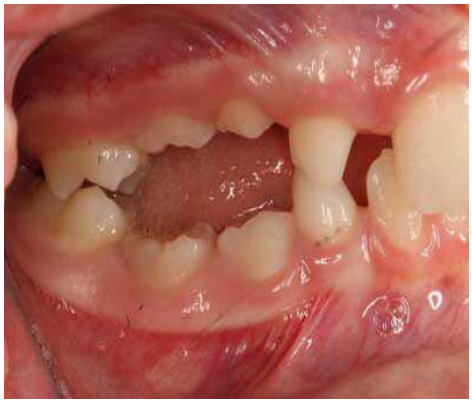

Figure 20: Ankylosis (Brin, 2011)

f. 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).

g. 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).

6.2 Genetic Disorders

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) .

6.3 Environmental Factors: include

a. Nutrition

Nutrition effects on timing of 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 (Nystrom et al, 2001 ; 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 (Kaczmarek, 1994 ; Kochhar and Richardson,1998).

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 low socioeconomic status. Some studies reported that the sequence permanent teeth eruption is different among children from different socioeconomic classes, the first teeth to erupt in child's from high backgrounds oral cavity is mandibular incisor while the mandibular first molar first teeth is first tooth to erupt in children from lower background (Helm and Seidler, 1974; Nonaka*et al.*, 1990; Clements *et al.*, 2009).

6.4 Systemic Conditions

Many diseases reported to be related to delayes eruption of teeth ,but only those with diabetes have earlier teeth eruption, the exact mechanisms responsible for earlier tooth eruption remain unsure, findings demonstrated enhanced tooth eruption by colony-stimulating factor-1, which upregulates the immunoreactivity of bone marrow mononuclear cells to growth hormone receptor and insulin-like growth factor-I ,also the local effect of the disease may play a role as gingival inflammation which exaggerated inflammatory response to bacterial plaque may contribute to localized osseous changes, resulting in reduced quality and quantity of surrounding bone and accelerating tooth eruption.(Andreasen*et al.*,1997; Proffit and Fields,2000; Lal*etal.*,2008).

There are numerous mechanisms for the delay eruption of teeth, it can be linked with primary teeth retaintion, hyperplasia of gingiva, or in hormonal changes which affect bone resorption rate (Wise *et al.*, 2002). the systemic factors described as following:

a. Trisomy 21 syndrome (Down syndrome)

Down syndrome is one of the congenital anomalies which is frequently associated with delayed eruption of the teeth (Jara*et al.*, 1993). A study of 127 males and 128 females with down syndrome by Ondarza et al in 1997 reported that six primary teeth were delayed in down syndrome eruption in boys and eleven primary teeth were delayed in girls and the chronological sequence of eruption in Down's children was not completely different from that of normal individual (Ondarza*etal.*,1997). Moraes et al in 2007 had been studied a sample of 49 syndromicpatientswith their panoramic x-rays and found that taurodontism was the most common dental anomaly in individuals with

Down syndrome and the primary teeth not always had their formation completed before the age of 5 years with delayed permanent teeth eruption.

b. Cleidocranial dysplasia

Cleidocranial dysplasia is rare congenital syndrome that has dental significance with delayed development of teeth; it characterized by delayed resorption of the primary teeth and delayed eruption of the permanent teeth thus complete set of primary dentition at 15 years is not uncommon (Becker *et al.*, 1997).

Delayed dentition and the presence of many supernumerary teeth, removal of supernumerary teeth in the maxillary arch cause irregular and delayed eruption of some of the permanent teeth (Subasioglu, 2015).

c. Hypothyroidism

Hypothyroidism is the result of an absence or under-development of the thyroid gland and inadequate levels of thyroid hormone (Werhun and Hamilton, 2015).

The teeth are normal in size but jaws are smaller than normal and therefore, teeth are crowded , hypothyroidism cause delay stages of tooth eruption as the thyroid hormones interfere with the process of eruption and have a structural impact on developing teeth((McDonald and Avery,2011).

d. Achondroplastic dwarfism

Achondroplasia is a form of skeletal dysplasia/dwarfism that manifests with stunted stature and disproportionate limb shortening,Achondroplasia may be associated with malocclusion and crowding with delayed eruption of the teeth due to altered bone growth (Al-Saleem and Al-Jobair , 2010).

e. Hypopituitarism

A pronounced slowing of the growth of the bones and soft tissues which result from a deficiency in the growth hormone secretion rate, it characterized by delayed eruption of teeth. In severe cases the primary teeth may not undergo the normal physiological resorption but retained all over the life (Myllarniemi*et al.*, 1978; Peedikayil, 2011).

f. Other causes

May cause delayed eruption of teeth including fibromatosisgingivae , Albright hereditary osteodystrophy, Vitamin D-resistant rickets ,Hunter syndrome (McDonald and Avery ,2011).

Conclusion

Eruption is a complex process started from 6 months to the time of eruption of third molar between (18-24) years old. There are many disturbances occur during formation and eruption which affect on sequence of eruption of teeth. Also there are systemic, genetic and environmental factors affecting on eruption of teeth, these factors either cause delay or prevent eruption of teeth.

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