Republic of Iraq

Ministry of Higher Education
and Scientific Research

University of Baghdad
College of Dentistry

Factors Affecting the Process of Teeth Eruption

A Project submitted to
College of Dentistry, University of Baghdad, Department of Pedodontics & Preventive Dentistry in Partial Fulfillment for the Degree of Bachelor of Dental Surgery (B.D.S.)

By
Mohammed Haider Ibrahim

Supervised by
Assist. Lec. : **Shatha Abdullah Abbas**
B.D.S, MSc, Preventive Dentistry

2022

Supervisor Declaration

I certify that this project entitled " Factors Affecting the Process of Teeth Eruption " was prepared by the fifth-year student Mohammed Haider Ibrahim under my supervision at the College of Dentistry/University of Baghdad in partial fulfilment of the graduation requirements for the Bachelor Degree in Dentistry.

Supervisor name :

Assist. Lec. Shatha Abdullah Abbas

Dedication

To my family, I could never done this without your faith, support, and constant encouragement, thank you for teaching me how to believe in myself, In my life, and in my dreams, especially my little brother Ibrahim.

To my superwisor Dr. Shatha Abdullah who believed in my abilities and was always there for me whenever I needed

To all people who supported and encouraged me Family, Friends, Teachers and Colleagues. Many thanks to all of you , especially Fatima.

To the unknown solder, best partner ever

Acknowledgement

In the name of Allah the most compassionate and most merciful. I am most grateful to God for the presence of health, patience, guidance, and protection through the duration of this study and achieve this study, which I hope it will be useful and objective.

Deep thank to Prof. Dr. Raghad Al-Hashimi , the Dean of the College of Dentistry, the University of Baghdad. Also to Prof. Dr. Ahlam Taha , the Head of the Pedodontic and Preventive Dentistry Department for her kindness and help.

I am gratefully thankful to my supervisor Assist.Lec. Shatha Abdulla Abbas, for her kindness, advice, instructions, valuable unlimited cooperation and continued encouragement throughout work and writing of my study. I have indeed learned a great deal from her.

I am really thankful for my colleagues Bassam Alaa & Mohammed Salim for giving me motivation all the way during the time of writing this project.

I would like to present special thanks for teaching staff for their great ethics and to all colleagues and all members of Pedodontic and Preventive Dentistry Department for their helpful assistance

List of content

List of figures

List of tables

List of abbreviation:

abbreviation	Meaning
SI	Stratum Intermedium
SR	Stellate Reticulum
DEJ	Dentinoenamel Junction
CEJ	cementoenamel Junction
IEE	Inner Enamel Epithelium
OEE	Outer Enamel Epithelium
PDL	Periodontal Ligament

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 , Numerous studies had been performed to well understand the process of tooth eruption. The most common general symptoms during tooth eruption include anxiety (15%), diarrhea (13%), a combination of the two (8%), fever and increased salivation (Almonaitiene et al., 2010).

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, cleidocranialdysostosis, hypothyroidism, hypopituitarism and achondroplastic dwarfism (Boka et al, 2009).

2.Aim of the study :

The aim of this study is to discuss different stages of tooth development, eruption process and the factors affecting it, because:

*Awareness of the fundamental aspects and understanding of this entire process is essential to identify the cause behind any deviations in eruption from the normal and in treating the condition.

*Eruption of deciduous teeth, their exfoliation followed by eruption of permanent dentition is an orderly, sequential, and age-specific event and is considered as an important milestone during child's development.

*Parents consider eruption of first primary teeth as an important developmental milestone that has to be achieved by the child.

*Eruption is a physiologic process that strongly influences the normal development of the craniofacial complex.

Review of literature

3.Teeth development (Odontogenesis)

Tooth development or odontogenesis is the complex process by which teeth form from embryonic cells, grow, and erupt into the mouth. For human to have a healthy dentition, dental tissue must develop during appropriate stages of fetal development. Primary teeth start to form between the sixth and eighth week of intrauterine life, while permanent teeth begin to form in the twentieth week , If teeth do not start to develop at or near these times, they will never develop, resulting in hypodontia or anodontia (nanci, 2013).

3.1. Physiological phases in tooth development

Tooth development can be divided into the following overlapping phases (Kumar, 2015) , which are:

1- Initiation : During this phase, the sites of the future teeth are established with the appearance of tooth germs along an invagination of the oral epithelium called dental lamina.

2- **Proliferation**: During this phase, Proliferative growth causes regular changes in the size and proportions of the growing tooth germ.

3- **Histodifferentiation**: During this phase, differentiation of cell (begun during morphogenesis) proceed to give rise to the fully formed dental tissues, both mineralized (such as enamel, dentin and cementum) and unminaralized (such as pulp and periodontal ligament).

4- Morphodifferentiation : During this phase, the shape of the teeth are determined by a combination of cell proliferation and cell movement.

5- Apposition: During this phase, the deposition of dental hard tissue occur in the tooth like dentin and enamel.

3.2. Morphological stages of tooth development

For descriptive purposes, tooth germs are classified into bud, cap and bell stages according to the degree of morphodifferentiation and histodifferentiation of their epithelial components (enamel organs). Leading up to the late bell stage, the tooth germ changes rapidly both in its size and shape, the cells are dividing and morphogenetic processes are taking place (Peterkova et al., 2014).

3.2.1. Bud stage

This stage is characterized by round or ovoid swellings at ten different points arise from dental lamina in each jaw, corresponding to the future position of deciduous teeth. They are the primordial of enamel organs (the tooth buds), thus the development of the tooth germ is initiated (Muthu & Kumar, 2019)

3.2.2. Cap stage :

As the tooth bud continues to proliferate into the ectomesenchyme, the deeper surface of the enamel organ invaginates to form a cap-shaped structure (Fehrenbach,2020).

The enamel organ at this stage is consisted of three layers, outer enamel epithelium, inner enamel epithelium and stellate reticulum (Ten Cate et al., 2003)

The adjacent ectomesenchymal cells are continue to proliferate and surround the enamel organ. The part of the ectomesenchyme lying beneath the inner enamel epithlial is called dental papilla. The ectomesenchymal tissue surrounding both

enamel organ and dental papilla is called dental sac or dental follicle. During the early stages of tooth development, three transitory structures may be seen, which are enamel knot, enamel cord and enamel niche (Som & Miletich, 2018).

3.2.3. Bell stage

The enamel organ is bell-shaped during this stage, and the majority of its cells are called stellate reticulum because of their star-shaped appearance. Tooth crown assumes its final shape (Morpho-differentiation), and the cells that will make the hard tissues of crown (Ameloblasts and odontoblasts) acquire their distinctive phenotype (Histo-differentiation).The bell stage is divided into the early bell stage and the late bell stage (Hand & Frank, 2014).

A- Early bell stage

High degree of histodifferentiation is achieved in the early bell stage .The enamel organ shows four distinct layer: cuboidal cells on the periphery of the enamel organ are known as outer enamel epithelium (OEE), the columnar cells of the enamel organ adjacent to the enamel papilla are known as inner enamel epithelium (IEE), the cells between the IEE and the stellate reticulum(SR) form a layer known as the stratum intermedium (SI). The function of cells of the IEE layer first is to exert an organizing influence on the undelying ectomesenchymal cells of dental papilla to differentiate into odontoblasts that produce dentin, and then it differentiate into ameloblasts which forms enamel. The rim of the enamel organ where the outer and inner enamel epithelium join is called the cervical loop (Kumar, 2015)

B -Advance (late) bell stage or crown stage:

Hard tissues, including enamel and dentin, develop during advance bell stage. Important cellular changes occur at this time. During this stage the boundary between inner E. epithelium and odontoblasts outlines the future D.E.J. (dentino enamel junction). In addition, the basal margin of the enamel organ (cervical loop) gives rise to the Herwig's epithelial root sheath. The dental papilla ultimately gives rise to dental pulp, once the dentin formation begins at the cuspal tip of the bell stage tooth germ (Boyde & Bromage, 2021) .

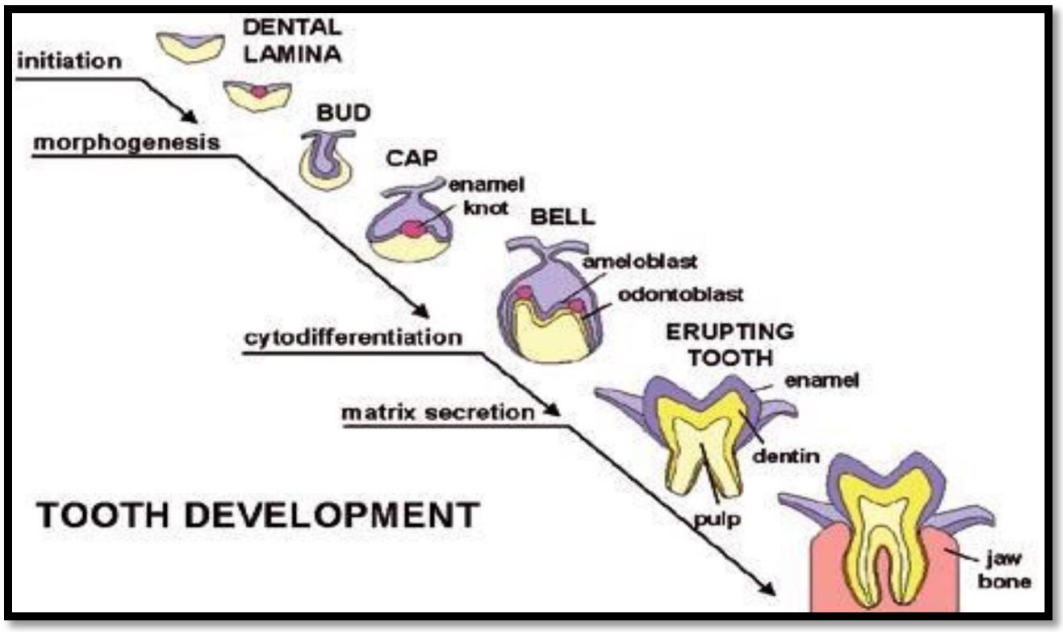

Figure 1: tooth development stages (Thesleff and Mikkola , 2002)

3.3. Root formation :

Root development initiates after the completion of enamel formation. Hertwig's epithelial root sheath (HERS) determines the size and shape of the root or roots of the tooth. As soon as the layer of dentin matrix mineralizes, gaps appear within the HERS-derived Malassez epithelium, allowing mesenchymal cells from the dental sac to move into contact with the newly formed dentin. These mesenchymal stem cells thereafter differentiate into cementoblasts and

deposit cementum matrix on the root dentin.(Liversidge and Molleson, 2004 ; KEIJO & Ellen , 2011).

4.Tooth eruption:

The word erupt has origins in the Latin word eruptus, which mean breakout, it refers to the axial or occlusal movement of the tooth from its developmental position within the jaw to its functional position in the occlusal plane (Bhaskar ,1991).

In common usage, eruption signifies the cutting of the tooth through the gingiva. However, the eruption of even a single tooth has to be understood in the broad context of various changes that occur in the jaws in preparation to, simultaneous with, and subsequent to the eruption of that tooth. These overlapping phases of physiologic tooth movements are studied as:

4.1. Pre-eruptive tooth movement:

It includes all movement of the primary and permanent tooth germs through tissues of the jaw prior to their eruption.

Bony remodeling of crypt wall occurs to facilitate movements of growing tooth germ and its movement. In bodily movement in a mesial direction, bone resorbs on the mesial side and forms on the distal side of the crypt. There is a considerable change in position between the permanent incisor tooth germ and its deciduous predecessor in the first 2 years of life. The permanent molars, which have no deciduous predecessors, also exhibit movement (Nanci , 2009)

The pre-eruptive movements of primary and permanent tooth germs place the teeth in a position within the jaw for eruptive movement (figures 3 &4) . The movements are a mixture of total bodily movement of the tooth germ and a

change in the center of the tooth germ by growth in which a part of the tooth germ remains fixed while the remaining parts continues to grow (Bhashar, 1991; Avery and Steel, 1992).

4.2. Eruptive tooth movement:

During the phase of eruptive tooth movement the tooth moves from its position within the bone of the jaw to its functional position in occlusion, and the principal direction of movement is occlusal or axial. However, as in the case of preeruptive tooth movement, jaw growth is still occurring while most teeth are erupting so that movement in planes other than axial movement is superimposed on eruptive movement (Srinath et al., 2013).

The term 'prefunctional' eruptive tooth movement is used to describe the movement of the tooth after its appearance in the oral cavity till it attains the functional position. The actual eruption of the tooth, when it breaks through the gum, is only one phase of eruption, the final position of tooth in the oral cavity is determined by environmental factors, forces of muscles (the tongue, cheeks, and lips) that acts on the tooth, also the contact with other erupted teeth . Thumb-sucking which is a childhood habit is an example of the environmental influence of tooth position (Avery and Steel,1992) .

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).

Post-eruptive tooth movements are those that (Nanci, 2013):

(1) maintain the position of the erupted tooth while the jaw continues to grow.

(2) compensate for occlusal and proximal wear.

5. Eruption hematoma (eruption cyst)

An eruption hematoma is a bluish- purple elevated area of tissue occasionally develops a few weeks before the eruption of a primary or permanent tooth. It may result from trauma to the area during function and then hemorrhage in the follicle of an erupted tooth, it will subside after eruption following rupture of the soft tissue by the tooth. The blood-filled cyst is most frequently seen in the primary second molar or the first permanent molar region (6 and E).

Because the condition is almost always self-limiting, treatment of an eruption hematoma is rarely necessary (Hayes,2000 ; Şen-Tunç et al,2017).

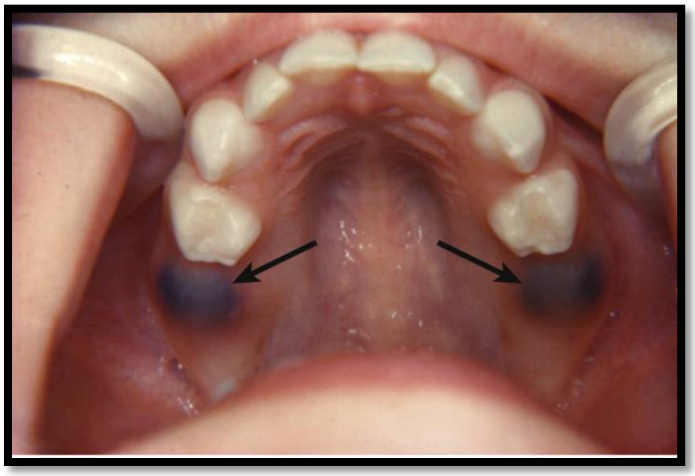

Figure 2: Eruption hematoma (Shanthala, 2019).

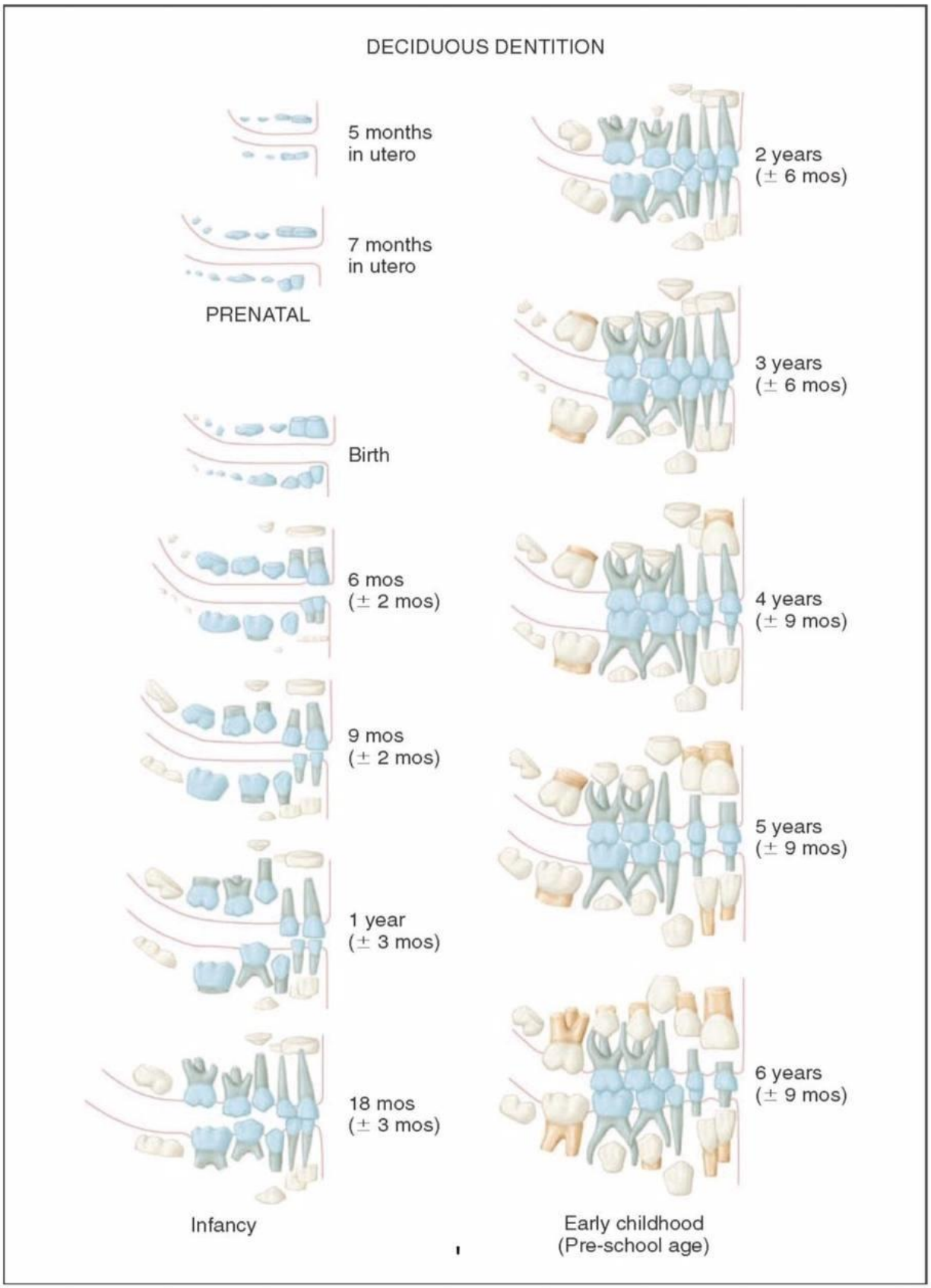

Figure 3 : chronology of development of deciduous dentition (Nelson, 2015)

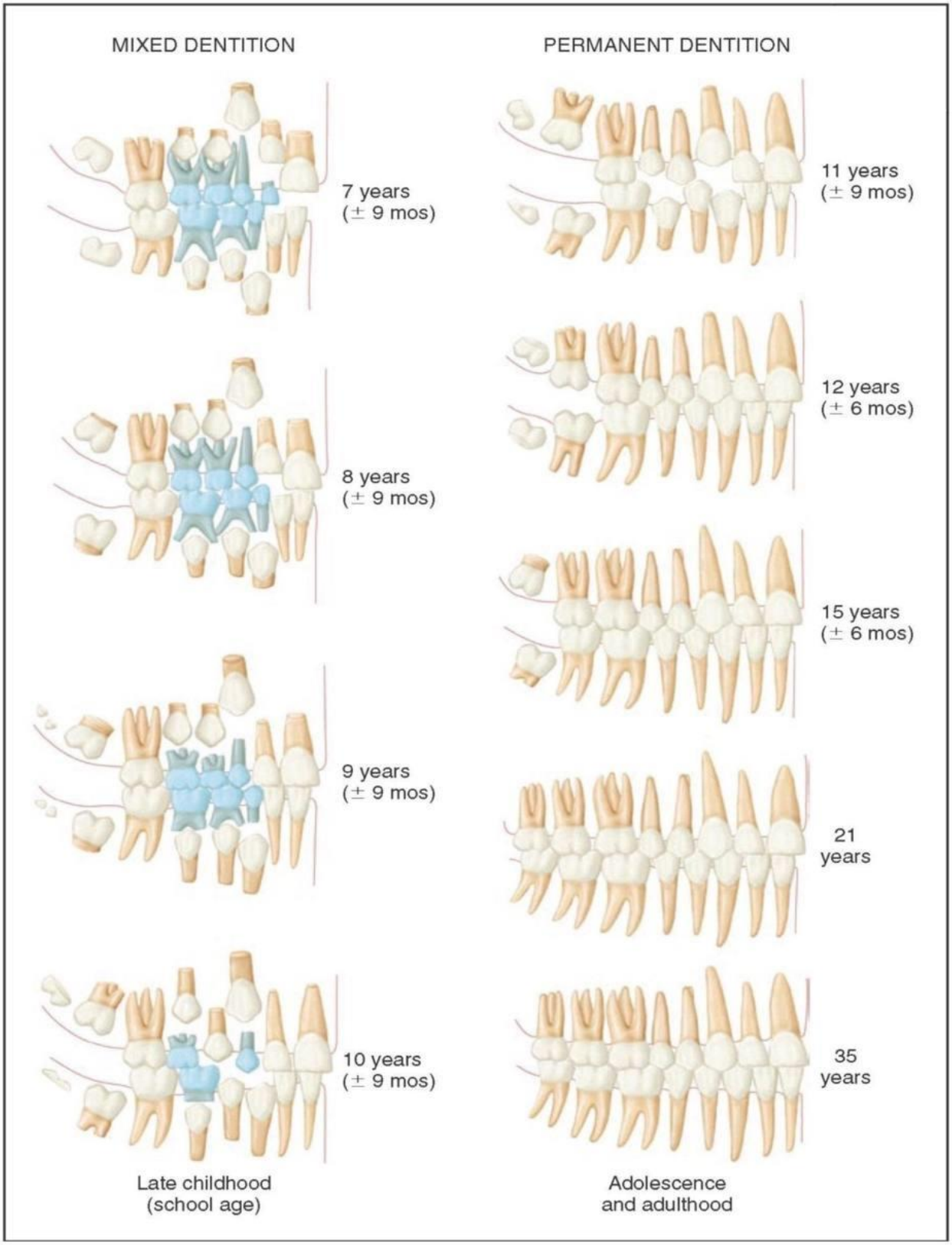

Figure 4: chronology of development of permanent dentition (Nelson, 2015)

6. Theories of tooth eruption:

Tooth eruption had been investigated since the beginning of the 19th century. The eruption process was studied by cross sectional studies in animal periodontal membrane and bone, both histologically and clinically, but this was not possible in human tissues, however, in human, it had been extensively studied by longitudinal studies that focusing on both normal and pathological conditions. Although the mechanism of eruption is not completely understood yet (Marks, 1995; Kuhr et al. ,2004; Kjaer, 2014), the possible explanations for this process could be:

6.1. Vascular Pressure

It had been suggested that the tissue fluid or blood pressure from the tissue that lies between the developing tooth and the jaw in the periapical region is sufficient to move tooth and is responsible for the tooth eruption (States that "the tissue pressure apical to the erupting tooth was greater occlusally, theoretically generating an eruptive force"). It means that the hydrostatic pressure above the tooth is less than that underneath the tooth. This difference in pressure will push the tooth occlusally. However, since surgical excision of the growing root and associated tissues eliminates the periapical vasculature without stopping eruption, this means that the local vessels absolutely are not necessary for tooth eruption. Eruption can also occur independently of the vascular alteration (Kumar, 2015).

6.2. Pulpal Pressure

It was thought that pressure generated by the dental pulp was sufficient to push the tooth occlusally. However pulpless teeth can erupt at the same rate as that of the normal teeth (Marks and Schroeder, 1996).

6.3. Alveolar Bone Remodeling

This theory suggested that bone remodeling achieved by the formation and resorption of bone around a tooth produce a pulling force to the tooth which permit the tooth movement, selective bone deposition and resorption occured around the developing tooth during eruption, however, the opinions against this theory mentioned that bone deposition at the bottom of the crypt during eruptive phase occurs only after the tooth had moved and when eruption is prevented by removing a developing tooth while leaving the dental follicle intact, the eruptive pathway forms in the overlying bone. Thus it is generally accepted that the bone remodeling is a result and not a cause for tooth movement. In addition, the rate of bone growth is slower than the growth of tooth (Kuhr et al., 2004).

6.4. Root Elongation

It was suggested that root elongation is responsible for tooth eruption since it is happened together with tooth eruption, but rootless teeth also erupt which mean that presence of root elongation presence is not required for tooth eruption but it might accelerate it. In addition, tooth eruption completes before completion of root (Wang, 2013).

6.5. Periodotal ligament:

Eruptive force resides in the dental follicle-periodontal ligament complex. Formation and renewal of the PDL has been considered a factor in tooth eruption because of the traction power that fibroblasts have which may pull the tooth out during eruption "The periodontal ligament, which is derived from the dental follicle, provides the force required for eruption mainly by fibroblast contraction" (Keinan and Cohen ,2013). Kimmoto et al. in 1999 stated that periodontal

ligament cell synthesize and secret molecules as autocrine or paracrine factors that affect bone remodeling and root resorption and thus affect the eruption process. However, in vitro tissue studies have limitations concerning this theory. Rootless teeth can erupt while impacted teeth with a well-developed PDL cannot erupt.

6.6. Dental follicle theory:

Dental follicle is a thin, dense connective tissue that surround the crown of the unerupted tooth . This theory has an important role in tooth eruption as it regulates the alveolar bone resorption that is needed for formation of eruption pathway for the tooth and provides a factor called colony stimulating factor-1 (CSF-1), which regulates the influx of monocytes needed to be differentiated into osteoclasts, thus the dental follicle serves as a target to attract the monocytes, whereby these cells fuse to form osteoclasts. When removal of a developing tooth occurred , leaving the follicle intact, the eruptive pathway still formed in the overlying bone, but if the dental follicle was removed, no eruptive pathway forms (Marks, 1995; Nanci , 2013).The coronal aspect of the dental follicle regulates osteoclastogenesis (bone resorption) and the basal aspect of the dental follicle regulates osteogenesis (bone formation) Therefore, the dental follicle is mainly responsible for the process of eruption (Nel et al., 2015).

7. New theories behind the eruption process

7.1. Kjaer in 2014

suggested that the mechanism of eruption depends on the eruption course created by crown follicle, hypothesis behind this theory depend on:

(1) Space in the eruption path

(2) Pressure from apical root membrane

(3) Adaptability in the periodontal membrane, as shown in Figure 5.

The crown follicle destroys the covering bone tissue and therefore creates space in the eruption pathway. The assumption is that the root membrane acts as a glandular membrane and the innervation in the glandular end-cells causes an overpressure that supplants to the root surface, periodontal membrane and pulp tissue (Becktor et al., 2002).

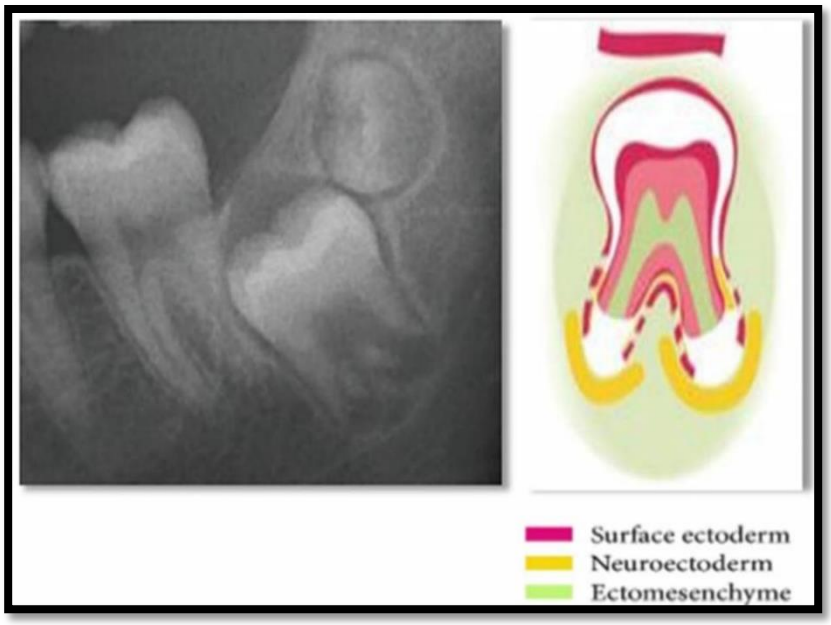

Fig.(5): Schematic drawing and radiograph showing the morphology of a mandibular first molar shortly before eruption, and showing the ectodermal cell layers of the follicle around the crown and the innervation in the root sheet, (Kjaer, 2014).

7.2. Bite forces sensed by soft tissue dental follicles theory:

This theory postulates that follicular soft tissues detect bite-forces and so direct bone remodeling with the effect of enabling tooth eruption .The highest equivalent strains induced by bite forces in the dental follicle and PDL in both erupted and unerupted teeth irrespective of incisive or unilateral molar bite forces .Examination of the soft tissue dental follicles, suggested broad areas of

compression in overlying crowns and wide zones of tension in follicle below root apices. So, these soft tissue dental follicles act as relevant stress sensors (Sarrafpour et al., 2013).

7.3. Neuromuscular theory or unification theory:

The neuromuscular theory or unification theory of tooth eruption stated that the synchronized forces of the orofacial muscles, under the control of the central nervous system, are responsible for the active movements of a tooth and the molecular events prepared a pathway under the control of these forces (Ruta et al., 2010; Bille et al., 2011; Loto, 2017).

The coordinated neuromuscular forces are converted into electrical, electrochemical and biomechanical energies for the stimulation of cellular and molecular activities within and around the dental follicle and enamel organ to prepare a pathway as well as other cellular functions for eruption of a developing tooth (Wise, 2009; Loto,2017)

8.Stages of occlusal development

8.1. 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). In some cases, teeth presents at this stage these are either:

-Natal Teeth which are existing at birth, or

-Neonatal teeth that erupt during the first month, these teeth resemble the deciduous teeth, that have enamel, dentine, pulpal tissue and usually without roots or have very short roots (Tapasyajuneja, 2007). The incidence of natal and neonatal teeth is estimated to be 1:1000 and 1:30000 respectively. These teeth are

almost always mandibular incisors, which frequently display enamel hypoplasia. There is a familial tendency for such teeth. They should not be removed if they are normal but a need for removl in case of being supernumerary or mobile teeth (Tapasy and Gurkeerat, 2007).

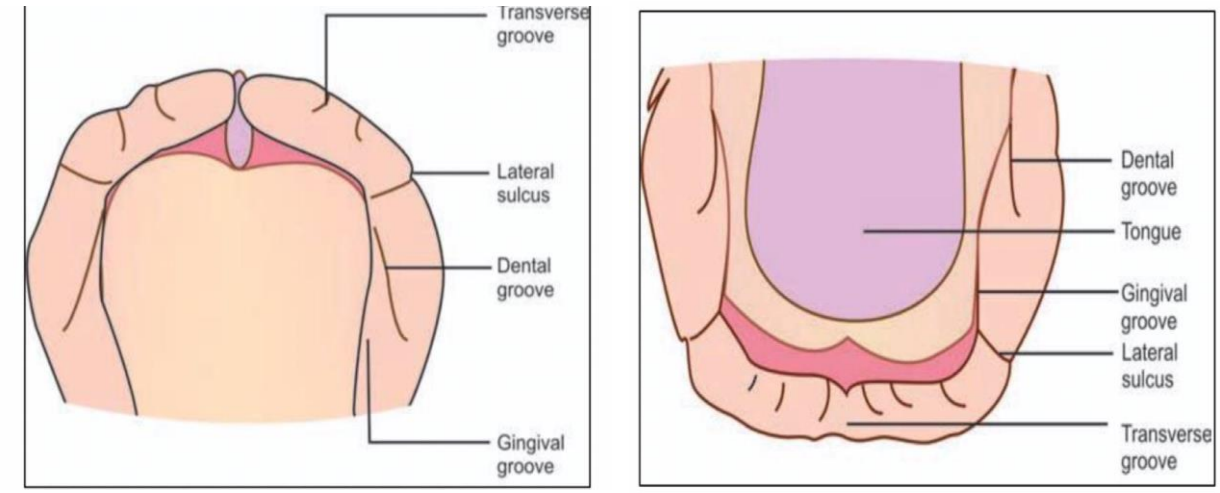

Figure 6: gumpad, maxilla and mandible gumpad, (Tapasy and Gurkeerat, 2007)

8.2.Primary dentition stage

The deciduous dentition stage starts from the eruption of the first deciduous tooth, usually the deciduous mandibular central incisors and ends with the eruption of the first permanent molar, i.e. from 6 months to 6 years of postnatal life. Usually lower teeth erupt before upper teeth. (Tapasy and Gurkeerat,2007). Sequence of eruption is as follow:- **(A-B-D-C-E)**

8.3. Mixed dentition stage

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

8.4. Permanent dentition period

The permanent dentition forms within the jaws soon after birth. Calcification begins at birth with the calcification of the cusps of the first permanent molar and extends as late as the 25th year of life (Tapasy and Gurkeerat, 2007).

Table 1 : the development time line of the primary teeth (Ash et al, 2003).

	Maxillary (upper) teeth				
Primary teeth	**Central incisor**	**Lateral incisor**	**Canine**	**First molar**	**Second molar**
Initial calcification	14 wk I.U.	16 wk I.U.	17 wk I.U.	15.5 wk I.U.	19 wk I.U.
Crown completed	1.5 mo	2.5 mo	9 mo	6 mo	11 mo
Root completed	1.5 yr	2 yr	3.25 yr	2.5 yr	3 yr
	Mandibular (lower) teeth				
Initial calcification	14 wk I.U.	16 wk I.U.	17 wk I.U.	15.5 wk I.U.	18 wk I.U.
Crown completed	2.5 mo	3 mo	9 mo	5.5 mo	10 mo
Root completed	1.5 yr	1.5 yr	3.25 yr	2.5 yr	3 yr

Table 2 : the development time line of the permenant teeth (Ash, 2003).

Permanent teeth	Maxillary (upper) teeth							
	Central incisor	Lateral incisor	Canine	First premolar	Second premolar	First molar	Second molar	Third molar
Initial calcification	3–4 mo	10–12 mo	4–5 mo	1.5–1.75 yr	2–2.25 yr	at birth	2.5–3 yr	7–9 yr
Crown completed	4–5 yr	4–5 yr	6–7 yr	5–6 yr	6–7 yr	2.5–3 yr	7–8 yr	12–16 yr
Root completed	10 yr	11 yr	13–15 yr	12–13 yr	12–14 yr	9–10 yr	14–16 yr	18–25 yr
	Mandibular (lower) teeth							
Initial calcification	3–4 mo	3–4 mo	4–5 mo	1.5–2 yr	2.25–2.5 yr	at birth	2.5–3 yr	8–10 yr
Crown completed	4–5 yr	4–5 yr	6–7 yr	5–6 yr	6–7 yr	2.5–3 yr	7–8 yr	12–16 yr
Root completed	9 yr	10 yr	12–14 yr	12–13 yr	13–14 yr	9–10 yr	14–15 yr	18–25 yr

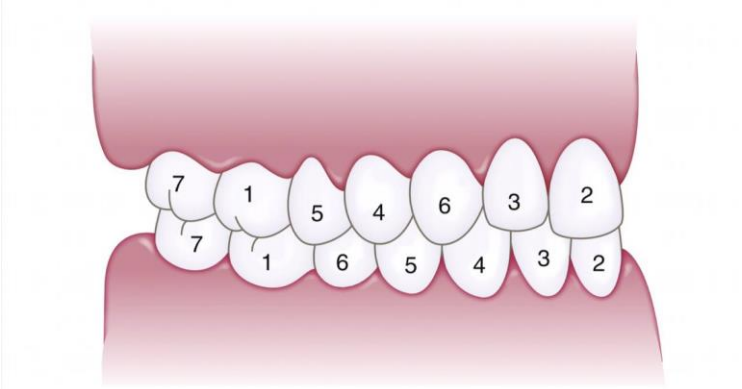

Figure 7 : desirable sequence of eruption of permanent teeth (Shanthala, 2019)

9.Factors affecting tooth eruption:

9.1. Local Factors: include:

a. Mucosal barriers-scar tissue:

It is an etiologic factor of delayed teeth eruption, any failure of the follicle of an erupting tooth to unite with the mucosa will involve a delay in the breakdown of the mucosa and constitute a barrier to emergence. Histological studies had been shown differences in the submucosa between normal tissues and tissues with a

history of trauma or surgery. 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 et al,1994; Ekstrand et al.,2003).

Figure 8 : **Mucosal barriers (Andreason et al, 1997).**

b. Inflammatory Dentigerous Cyst :

Inflammatory dentigerous cyst is the most common type of developmental odontogenic cysts and mostly is found in the mixed dentition (Delbem et al, 2006) . It is usually single, located in the posterior mandible in association with the roots of non-vital deciduous teeth and the crown of their unerupted permanent successors (Kozelj & Sotosek, 1999 ; Slater 2003) .Radiographically, the tooth may be displaced: it is not uncommon to see teeth displaced to the condylar neck, the nasal floor or high in the maxillary sinus approaching the orbit (Delbem et al ,2006).

c. Trauma 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).

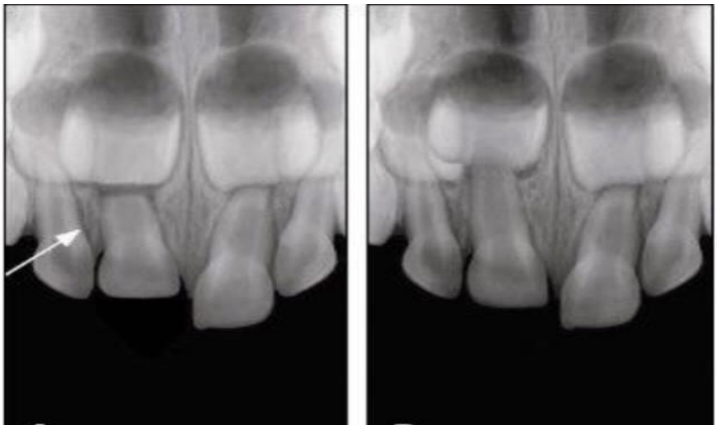

Figure 9 : injury to primary teeth (Diab & Badrawy, 2000)

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 incidence of ankylosis in the deciduous teeth mostly follows a familial pattern and very slow root resorption was observed for most of the ankylosed teeth. 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 remain in a fixed occlusal level 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 10 : Rariographic film for ankylosed seconed primary molar (shanthala, 2019).

Figure 11: Ankyloses (Brin et al, 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 be related with a delay in the eruption of the maxillary second molar, despite 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. Eruption sequestrum

The eruption sequestrum is occasionally seen in children at the time of the eruption of the first permanent molar (6). Clinically it is appear as a tiny spicule of nonviable bone overlying the crown of an erupting permanent molar just before or immediately after the emergence of the tips of the cusps through the oral mucosa. It is composed of dentin and cementum as well as a cementum-like material formed within the follicle (Maki et al ,2005).

h. Concrescence: Cemental union of two teeth that can cause a difficulty or failure in teeth eruption (Romito and Omaha ,2004) .

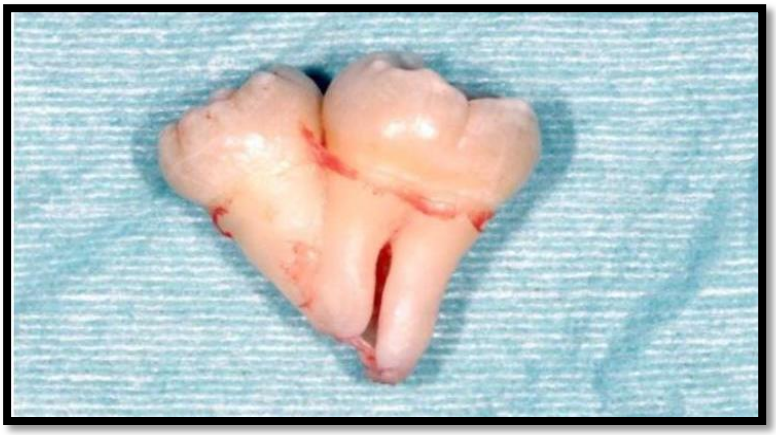

Figure 12: concrescence (Tapasy and Gurkeerat, 2007).

i. 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).

9.2.General factors

Permanent teeth eruption is a continuous development that is affected by a number of factors (Almonaitiene et al. ,2010) .

A. **Gender**

Many studies in different countries found that the permanent teeth erupted earlier in girls than in boys , and the difference between eruption times normally is from 4 to 6 months (Helm and Seidler,1974; Titly, 1984;ElZahid and Hafez,1993;Daood, 2001;Sahib,2008). The earlier eruption of permanent teeth was due to earlier onset of maturation (Ekstrand et al. ,2003), also in group of Iraqi children aged (9-10) years a significant advancement in dental development includes all stages of root formation in girls than boys for maxillary and mandibular canine, first molar and maxillary second premolar (Farah, 1988). Iraqi study by Medha A. in 2019 that examined a group of children aged 8 and 9 years at primary school children in Al-Khalis district /Diyala Governorate, concluded that total count of erupted permanent teeth was higher among girls at both of the age groups, so the eruption of permanent teeth among girls was earlier than that among boy at the same arch and the same age group .

B. Preterm birth

World Health Organization (WHO) defined preterm birth as “birth occurring prior to 37 weeks of gestation or if the birth weight is less than 2500g

”(Goldenberg et al ., 2008). Most of the studies showed delayed primary and permanent teeth eruption among preterm children. Some studies found that the greatest delay was in children aged less than 6 years of age, while for those nine years or older, there was no difference (Harila et al. , 2003). In a study of Al-Sayag et al. in 2008 to evaluate the effect of premature birth on primary dentition at AL- Mosul city , the results showed that the eruption of deciduous teeth was significantly delayed among prematurely born children.

C. Race factor

Many studies reported that the race factor had a greater significant effect on tooth eruption than the environmental factor (Lavelle , 1975). Houpt et al. in 1967 studied the eruption times of permanent teeth in Ghanaian children and they found that permanent teeth erupted approximately one year earlier than in North American Caucasian children and this difference was due to a racial factor. While Garn et al. in 1973 studied the Negro-Caucasian difference in permanent teeth emergence at constant income level and found that the American Negroes had their teeth emerged earlier than the American whites and this racial difference was more significant on eruption time of incisor, molars and the least for canines and premolars .Longitudinal and cross-sectional studies reported differences in teeth emergence time among different races, permanent teeth eruption significantly earlier in African and American-African children than in Asians and Caucasians (Mugonzibwa et al., 2002).

9.3.Genetic Factor

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 cessation of emergence after a tooth erupts 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 normal, with delayed teeth eruption is the only finding, it has been suggested that there is a gene for tooth eruption, and its delayed onset might participate in delayed teeth eruption as in inherited retarded eruption (Blankenstein et al,2001) .

9.4. Environmental Factors: include

1. **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 (Blankenstein et al, (2001); Mugonzibwa et al,2002), however: higher number of erupted permanent teeth were found among the obese children compared to others at the same age group (Medha A, 2019).

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).

There is evidence that chronic malnutrition, which continue after the early childhood is linked with delayed teeth eruption (Psoter et al. ,2008). In Iraq, cross sectional study was done by Al- Obaidi in 1995 to evaluate the oral health and nutritional status among kindergarten children aged 4 and 5 years in Baghdad city, the results showed that malnutrition caused significant delay in permanent first molar eruption time among 5 years old children for both age and weight for height.

In 2005, Gatta evaluate primary teeth emergence in relation to nutritional status among 4-48 months old children in Baghdad city, the results showed that malnutrition caused significant delay in primary teeth eruption .

II. 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).

III. Radiation effect

X-radiation which is a form of electromagnetic radiation had been shown to affect the eruption of the teeth. Impairment of root formation, damaging to the periodontal cell and lacking of mandibular growth also appeared to be related to tooth eruption disorders due to x-radiation (Piloni and Ubios,1996)

9.5.Systemic Factors :

Many diseases reported to be related to delayed eruption of teeth ,but only those with diabetes have earlier teeth eruption, the exact mechanisms responsible for earlier tooth eruption remain uncertain, findings demonstrated enhanced tooth eruption by colony-stimulating factor-1, which up-regulates the immune-

reactivity 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 et al, 2008).

9.5.1.Down's Syndrome :

Down's syndrome (trisomy 21) is one of the congenital pathologic conditions in which delayed eruption of the teeth is frequently observed. Various studies in children with Down's syndrome have shown that delayed tooth eruption is common, but sporadic (Ondarza et al, 1997). The eruption usually follows an abnormal sequence and some of the deciduous teeth may be retained until the age of 15 years. The eruption abnormalities are associated with retardation in the growth of maxilla and mandible. A reduction of the anterior skull base and protrusion of lower incisors is often observed, which is related to a tendency to anterior cross- bite and, to a lesser extent, to diminished overbite (Quintanilla et al, 2002).

9.5.2.Cleidocranial Dysplasia :

Cleidocranial dysplasia is a hereditary disorder characterized by abnormal clavicles, delayed fusion of the bones in the skull, extra teeth and short stature. Other bones, such as the ribs, pelvis and bones of the hands and feet may also be affected. Most patients with cleidocranial dysplasia do not have significant physical or mental disability (Gorlin et al ,1990).

The development of the dentition is delayed and may reach the age of 15 years. Oral radiographs usually show many unerupted and supernumerary permanent teeth. Even after extraction of the deciduous or supernumerary tooth, eruption of the permanent dentition may be delayed without the proper orthodontic intervention (Tanaka et al ,2006).

9.5.3.Achondroplastic Dwarfism ;

Achondroplastic dwarfism is the most common type of dwarfism and is clinically manifested with a characteristic appearance. There are short muscular extremities, brachycephalus, and bowed legs. The oral manifestations include retruded maxilla, disparity in the size of the jaws resulting in malocclusion, and delayed eruption of the teeth (Collins and Choi ,2007).

9.5.4.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).

9.5.5.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).

9.6.Other causes ;

Delayed eruption may be caused by factors including fibromatosisgingivae , Albright hereditary osteodystrophy, Vitamin D-resistant rickets, and Hunter syndrome (McDonald and Avery ,2011).

10.Factors Affecting the Development of the Teeth :

There are many developmental disturbance affecting the teeth (Tapasy and Gurkeerat, 2007).

10.1.Disturbances during initiation of tooth germ :

A. Hypodontia : Hypodontia is the term used to describe the developmental absence of one or more primary or permanent teeth, excluding the third molars . It is the most common developmental dental anomaly . Hypodontia in the primary dentition is less common with reported prevalence rates varying between 0.5% to 2.4% (Cunha, 1999)

Figure (13) : Hypodontia (Tapasy and Gurkeerat, 2007).

B. Anodontia : denotes complete developmental absence of teeth in both dentitions (Tapasy and Gurkeerat, 2007).

C. Hyperdontia : is a condition characterized by the presence of additional teeth, these are either supernumerary or supplemental teeth . The difference between them is that supplemental teeth are similar to the normal teeth while supernumerary teeth have various categories (Tapasy and Gurkeerat, 2007).

Types of supernumerary teeth: (Meighani & Pakdaman ,2010 ; Parolin et al , 2011; Nayak et al ,2012; Amini et al ,2013).

- Mesiodens: located between maxillary central incisors.
- Peridens located buccal to the arch
- Distomolar distal to the third molar.
- Paramolar located buccal or lingual to molars.
- Natal and neonatal teeth: These may be either supernumerary or deciduous teeth.

Fiigure 14 : super numerary teeth (Tapasy and Gurkeerat, 2007).

10.2.Disturbances during histodifferentiation and morphodifferentiation of tooth germ :

A. Microdontia: is a term used to describe teeth which are smaller than normal i.e. outside the usual limits of variation The most frequently affected teeth are maxillary lateral incisor and third molars (Namdar and Atasu , 1999) .

B. Macrodontia: refers to the teeth that are larger than normal (Namdar and Atasu, 1999).

C. Dens Invaginatus: is an embryologic anomaly that results in invagination of an amelodentinal structure, more or less developed, within the pulp (Sauveur et al ,1997).

Figure 15 : Dens in dente (Tapasy and Gurkeerat, 2007).

D. Dens evaginatus: is a rare dental anomaly involving an extra cusp or tubercle hat protrudes from the occlusal surface of the affected tooth also called as occlusal tuberculated premolar (Stecker and Diangells, 2002).

Figure 16 : Dens evaginatus (Tapasy and Gurkeerat, 2007).

E. Talon cusp: is a horn-like protuberance projecting from the lingual surface. In canines and incisors, it originates usually in the palatal cingulum as a tubercle projecting from the palatal surface; however, the anomaly also has affected the labial surface of the tooth. Mellor and Ripa named the accessory cusp talon cusp because of its resemblance in shape to an eagle's talon (Segura et al ,2003).

F. Gemination: is a single tooth germ splits into partially or fully separated crowns but with a common root and root canal (Neville et al , 2002).

G. Fusion: is a two tooth germs unite to form a single large crown with two root canals; commonly seen in incisors (Chipshvili et al , 2011).

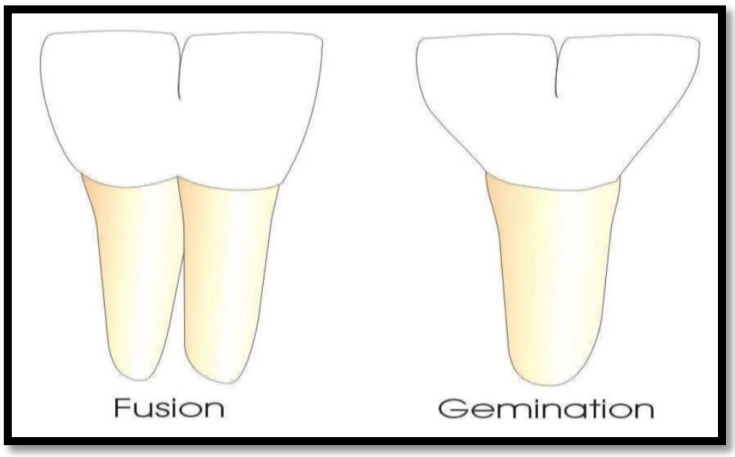

Figure 17 : gemination and fusion (Tapasy and Gurkeerat, 2007).

H. Dilaceration: is defined as an abnormal bend between two parts of a tooth that is nearly always associated with trauma that moves the developing tooth bud.

Another explanation is due to an abnormality in the formation of the tooth whereby the calcified part is displaced in relation to the uncalcified part (Hegde and Munshi, 2001)

Figure 18 : Dilaceration (Tapasy and Gurkeerat, 2007).

10.3.Disturbances during Apposition and Calcification of Hard Tissue: (Tapasy and Gurkeerat, 2007).

A. Enamel hypoplasia: is a reduction in the amount of enamel formed.

B. Amelogenesis imperfect: is a hereditary disorder in which the quality and quantity of enamel formed is altered.

C. Dentinogenesis imperfect: is a hereditary developmental disorder of the dentine. The dentine appears grey to brownish violet, enamel frequently separates from the defective dentine, roots become short, canals get obliterated, and rapid attrition is seen.

D. Odontodysplasia: is rare, but is most likely to occur in the maxillary anterior teeth. The enamel, dentin, and pulp of teeth are affected, so the teeth are very

brittle. On radiographs the teeth appear more radiolucent than normal, so they are often described as "ghost teeth".

E. Cemental hypoplasia: is a reduced rate of cementum formation, as in hypophosphatasia.

F. Enamel hypocalcification: the enamel calcification is abnormal. It may be caused by local, systemic or hereditary factors.

Conclusion

Eruption is a complicated process initiated from the age of 6 months till (18-24) years old which is eruption time of third molar. There are many disturbances which may be occurred during teeth development and eruption that could affect this process. Furthermore, eruption of teeth are influenced by local, systemic, genetic and environmental factors, these factors either cause delay or prevent eruption of teeth. Since eruption of teeth follows a strict pattern and time of eruption, understanding this process can be helpful in some instance for assessing the age both clinically and radiographically through the assessment of extent of crown and root formation.

Reference :

A

- Almonaitiene R, Balciuniene I & Tutkuviene J.2010. Factors influencing permanent teeth eruption. Part one–general factors. *Stomatologija*; 12(3):67-72.
- Al-obaidi W.1995.Oral health status in relation to nutritional status among kindergarten children in Baghdad .Iraq.Master thesis, College of dentistry ,University of Baghdad.
- Al-sayag G DH, & Qassim ,Al-rawi BA.2008. the effect of premature birth on primary dentition,Al –Rafidain Dent J; 8(1):18-22 .
- Amini, F., Rakhshan, V., & Jamalzadeh, S. (2013). Prevalence and Pattern of Accessory Teeth (Hyperdontia) in Permanent Dentition of Iranian Orthodontic Patients. *Iranian journal of public health*, 42(11), 1259–1265.
- Andreasen JO, Petersen JK & Laskin DM.1997. Textbook and color atlas of tooth impactions. 1st ed. Munksgaard.
- Ash, Major M.; Nelson & Stanley J. (2003). Wheeler's dental anatomy, physiology, and occlusion .
- Avery JK, Steel PF. 1992.Essentials of oral histology and embryology. Mosby year book;CH.(5)and(6),PP:51-81.

B

- Becktor K, B, Hansen BF, Nolting D & Jaer I.2002. Spatiotemporal expression of NGFR during prenatal human tooth development. *Orthodontics & craniofacial research*; 5(2), 85-89.
- Bhashar SN.1991. Orban’s histology and emberyology,11th ed.,St.Louis, Mosby;PP:49-405.

- Bille ML, Thomsen B & Kjear I.2011. Apoptosis in the human periodontal membrane evaluated in primary and permanent teeth. *Acta Odontol Scand*;69(6):385–8.
- Blankenstein R, Brook A H, Smith R N, Patrick D , & Russell J M. 2001. Oral findings in Carpenter syndrome. International Journal of Paediatric Dentistry; 11(5), 352-360.
- Blankenstein R, Brook A H, Smith R N, Patrick D , Russell J M. 2001. Oral findings in Carpenter syndrome. *International Journal of Paediatric Dentistry;* 11(5), 352-360.
- Boka V, Markopoulos A &Poulopoulos A. 2009. Tooth eruption :topical and systemic factor. Review part. *Balk J Stom*; 13:11-14
- Boyde, A., & Bromage, T. G. (2021). Histology of Dental Hard Tissues. In Vertebrate Skeletal Histology and Paleohistology (pp. 259-288). Boca Raton and London: CRC Press.
- Brin I, BenBassat, Y, Zilberman, Y & Fuks A.1988. Effect of trauma to the primary incisors on the alignment of their permanent successors. *Community dentistry and oral epidemiology*; 16(2), 104-108.

C

- Camargo LB, Cavalcanti T M, Aldrigui JM, Imparato J CP, RaggioDP.2011.Ankylosis of deciduous molar associated with other dental anomalies: report of three cases. IJD. *International Journal of Dentistry*;10(2), 108-111.
- Chipshvili N, Vadachkoria D & Beshkenadze E. 2011. Gemination or fusion ? – challenge for dental practitioners. (case study). Georgian Med News.;(194):28-33.

- Clements E M B, Davies-Thomas E, & Pickett K G. 2009. Time of eruption of permanent teeth in British children at independent, rural, and urban schools. *British medical journal*; 1(5034), 1511.
- Collins WO, & Choi SS. 2007.Otolaryngologic manifestations of achondroplasia. Arch Otolaryngol Head Neck Surg, 133:237-244.
- Craddock H L, & Youngson C C.2004. Eruptive tooth movement— the current state of knowledge. *British dental journal*; 197(7), 385- 391.
- CUNHA R F, BOER FA C, TORRIANI D D, & FROSSARD W T G .2001. Natal and neonatal teeth: review of the literature. Pediatric dentistry; 23(2): 158- 162.

D

- Daood ZH .2001.Chronology of permanent teeth emergence for Iraqi children /Baghdad city (Across-sectional study). Master Thesis College of dentistry;
- Delbem AC, Cunha RF, Afonso RL, Bianco KG, & Idem AP. 2006. Dentigerous cysts in primary dentition: report of 2 cases. Pediatr Dent, 28:269-272
- Diab M, & elBadrawy HE.2000. Intrusion injuries of primary incisors. Part III: Effects on the permanent successors. *Quintessence Int*;31:377-84.

E

- Ekstrand KR, Christiansen J, & Christiansen ME.2003. Time and duration of eruption of first and second permanent molars: a longitudinal investigation. *Community Dent Oral Epidemiol*;31: 344-50.

- EL-ZAHID H. , & HAFEZ S. 1993.Sequence of eruption of maxillary and mandibular permanent canines and premolars for a group of Saudi children. Egyptian Orthod J; (7):321-327.

F

- FARAH ME.1988. The orthodontic examination of children aged 9 and 10 years from Baghdad, Iraq-A clinical and radiographic study . Master thesis `College of Dentistry ,University of Baghdad.
- Fehrenbach, M. J. & Popowics, T. (2020) Illustrated dental embryology, histology, and anatomy. 5th edition. St. Louis, Missouri: Elsevier.
- Friel S.1954 The development of ideal occlusion of the gum. Pads and the teeth *American journal of orthodontics* , 40(3), 196-227 .

G

- Garn S M, Sandusky ST, Nagy J M, & Trowbridge FL.1973. Negro-Ca ucasoid differences in permanent tooth emergence at a constant income level. *Archives of Oral Biology*; 18(5): 609-615
- Gatta E. 2005. Primary teeth emergence in relation to nutritional status among 448 months old children in Baghdad city/Iraq. A master thesis Department of POP, College of Dentistry, University of Baghdad.
- Goldenberg R L, Culhane J F, Iams J D, & Romero R .2008. Epidemiology and causes of preterm birth. The lancet; 371(9606): 75-84.
- Gorlin RJ, Cohen MM, & Levin LS.1990. Cleidocranial dysplasia. In: Syndromes of the Head and Neck. 3rd ed. New York: Oxford Press, pp 249-253.

H

- Hand, A. R., & Frank, M. E. (2014). Fundamentals of oral histology and physiology. John wiley & sons.
- Harila Kaera V, Hekkineh T, & Alvesalo L .2003. The eruption of permanent incisors and first molars in prematurely born children. *The European Journal of Orthodontics*; 25(3): 293-299.
- Hayes PA. 2006, Hamartomas, eruption cyst, natal tooth and Epstein pearls in a newborn. *J Dent Child*. 67:365-8.
- Hegde S, Munshi AK (2001) Management of an impacted, dilacerated mandibular left permanent first molar: A case report. *Quintessence Int* 32: 235-237].
- Helm S and Seidler B.1974. Timing of permanent tooth emergence in Danish children. Comm. Dent. Oral. Epidemiol; 2: 122-129.
- Houpt MI , AD.U -Aryee S, & Grainer RM. 1967.Eruption times of permanent teeth in Bong Alhafo region .*Am.J. orthod*; 53:95-99.

k

- Kaczmarek M.1994. Variation in eruption sequence of permanent teeth in polish children. *In Auxology'94 Humanbiol. Budapest*;p : 349-58.
- Keijo Luukko , & Ellen Berggreen , 2011. in Cohen's Pathways of the Pulp (Tenth Edition).
- Keinan D,& Cohen RE . 2013.The significance of epithelial rests of Malassez in the periodontal ligament. *Journal of endodontics*; 39(5): 582-587.
- Kimoto S, Matsuzawa M, Matsubara S, Komatsu T, Uchimura N, Kawase T, & Saito S.1999. Cytokine secretion of periodontal ligament fibroblasts derived from human deciduous teeth: effect of mechanical stress on the

secretion of transforming growth factor β1 and macrophage colony stimulating factor. *Journal of periodontal research*; 34(5), 235-243.

- Kjaer I.2014. Mechanism of human tooth eruption: review article including a new theory for future studies on the eruption process. Scientifica .
- Kochhor. R. and Richardson A .1998. The chronology and sequence of eruption of human permanent teeth in Northern Ireland. Int. *J. Paediatr*. Dedt.2014; 8(4): 243-252..
- Kozelj V, &Sotosek B. 1999. Inflammatory dentigerous cysts of children treated by tooth extraction and decompression- report of four cases. Br Dent J, 187:11.
- Kuhr A, Popa Wanger A, Schmoll H, Schwahn C, & Kocher T .2004. Observations on experimental marginal periodontitis in rats. *Journal of periodontal research*; 39(2): 101-106.
- Kumar, G. S. (2015). Orban's Oral Histology & Embryology-E-BOOK. Elsevier Health Sciences.

L

- Lal S, Cheng B, Kaplan S, Softness B, Greenberg E, Goland RS, & Lamster I B.2008. Accelerated tooth eruption in children with diabetes mellitus. *Pediatrics*;121(5), e1139-e1143.
- Lavelle C L B.1975.A note on the variation in the timing of deciduous tooth eruption. *Journal of dentistry*; 3(6), 267-270.
- Liversidge H M, & Molleson T.2004. Variation in crown and root formation and eruption of human deciduous teeth. *American Journal of Physical Anthropology*; 123(2), 172-180.
- Loto AO.2017. Tooth eruption: a neuromuscular theory, part one. *J. Craniomax*. Res;4(1):278–83.

M

- Maki K, Ansai T, Nishida I, Zhang M, Kojima Y, & Takehara T, et al.. 2005. Eruption sequestrum: x-ray microanalysis and microscopic findings. *J Clin Pediatr Dent* 29:245-247.
- Marks S C, Schroeder HE.1996. Tooth eruption: theories and facts. *The Anatomical Record*; 245(2), 374-393.
- Marks S C.1995 The basic and applied biology of tooth eruption. Connective tissue research; 32(1-4): 149-157.
- McDonald R E and Avery D R.2011. Dentistry for the child and adolescent. Mosby/Elsevier,chapter 9; p:190-200.
- Mehda, A. 2019. Maximum Bite Force in Relation to Oral and Nutritional Status Among 8 and 9 years Old Primary School Students (Cross Sectional Study). Master thesis, college of dentistry, university of Baghdad.
- Meighani, G., & Pakdaman, A. (2010). Diagnosis and management of supernumerary (mesiodens): a review of the literature. Journal of dentistry , 7(1), 41–49.
- Mugonzibwa EA, Kuijpers Jagtman A M, Laine Alava M T, & Vanthof M A.2002. Emergence of permanent teeth in Tanzanian children. Community dentistry and oral epidemiology 30(6): 455-462.
- Muthu, M. S., & Kumar, S. (2019). Pediatric dentistry. Elsevier Health Sciences.
- Myllarniemi S,Lenko HL,&PerheentupaJ.1978. Dental maturity in hypopituitarism, and dental response to substitution treatment. *European Journal of Oral Science*; 86(5), 307-312.

N

- Namdar F, & Atasu M (1999) Macrodontia in association with a contrasting character microdontia. *J Clin Pediatr Dent* 23:271-274.
- Nanci A. 2009. Physiologic tooth movement: eruption and shedding. The Ten Cate's Oral Histology: Development, Structure, and Function, Mosby Elsevier; 268-289.
- Nanci,development,structure and function eight edition , 2013, Ten Cate's Oral Histology. Elsevier, p 70-94]
- Nayak, G., Shetty, S., Singh, I., & Pitalia, D. (2012). Paramolar - A supernumerary molar: A case report and an overview. *Dental research journal*, 9(6), 797–803.
- NEL S, Hendrik HD, Boy SC, & Raubenheimer EJ. 2015.Recent perspectives vis-à-vis the biological basis of tooth eruption. SADJ ;70(6):238– 41.
- Nelson S J.2015..Wheeler's dental anatomy, physiology and eruption ,E-book Elsevier Health Science .
- Neville BW, Damm DD, Allen CM, & Bouquot JE. 2002.Oral and Maxillofacial Pathology ed 2. Philadelphia: Saunders, 84-85.
- Nonaka K, Ichiki A, & Miura T.1990. Changes in the eruption order of the first permanent tooth and their relation to season of birth in Japan. *Am J PhysAnthropol*; 82:191-8.
- Nystrom, M. (2001) Reflective Life World Research. Studentlitteratur, Lund.

O

- Ondarza A, Jara L, Munoz P, & Blanco R, 1997, Sequence of eruption of deciduous dentition in a Chilean sample with Down's syndrome. *Arch Oral Biol*, 42:401-406.

P

- Parolia A, Kundabala M, Dahal M, Mohan M, & Thomas M S. (2011). Management of supernumerary teeth. 14(3), 221-224.
- Peedikayil F C.2011. Delayed tooth eruption. J Dent; 1(4), 81-6.
- Peterkova, R., Hovorakova, M., Peterka, M., & Lesot, H. (2014). Three dimensional analysis of the early development of the dentition. *Australian dental journal*, 59, 55-80.
- Piloni M J, Ubios A M .1996. Impairment of molar tooth eruption caused by xradiation. Acta odontologica latinoamericana: AOL; 9(2): 87-92.
- Proffit WR, & Fields HW.2000. Contemporary orthodontics. 3rd ed, St.louis ; Mosby , 142.
- Psoter WJ, Spielman AL, Gebrian B, Jean R, & Katz RV.2008. Effect of childhood malnutrition on salivary flow and PH. *Arch oral Biol*; 53(3):231-237.
- Pytlik W.1991. Primary failure of eruption; a case report. *Int Dent J*; 41(5): 274-278.

Q

- Quintanilla JS, Biedma BM, & Rodriguez MQ, et al , 2002. Cephalometrics in children with Down's syndrome. Pediatr Radiol, 32:635-643.

R

- Raghoebar GM, Boering G, Vissink A, & StegengaB.1991.Eruption disturbances of permanent molars: a review. *J Oral Pathol Med*;20:159-66.
- Romito LM, & Omaha (2004) Concrescence: Report of a rate case. *Oral Med Oral Pathol Oral Radiol Endod* 97: 325-327.
- Ruta A, Irena B, Janina T.2010. Factors influencing permanent teeth eruption. Part one – general factors. Stomatologija. *Baltic Dental and Maxillofacial Journal*;12:67–72.

S

- Sahib WW. 2008. Permanent Teeth Emergence for Iraqi Students in Hilla and Najaf Cities (A Cross-Sectional Study), *Kufa Med. Journal*; Vol. 11. No.1 .
- Sarrafpour B, Swain M, LI Q, & Zoellner H. 2013.Tooth eruption results from bone remodeling driven by bite forces sensed by soft tissue dental follicles: a finite element analysis. PLoS One;8(3):1–18.
- Sauveur G, Sobel M,& BoucherY (1997) Surgical treatment of a lateroradicular lesion on an invaginated lateral incisor. *Oral Surg Oral Med Oral Pathol Oral Radiol endod* 83: 703-706.
- Segura-Egea JJ, Jimenez-Rubio A, Rios-Santos JV, & Velasco-Ortega E (2003). Dens evaginatus of anterior teeth (talon cusp): Report of five cases. *Quintessence Int* 34: 272-277.
- Şen-Tunç E, Açikel H, Şaroğlu-Sönmez I, Bayrak S, & Nuray Tüloğlu N. 2017. Eruption cysts: A series of 66 cases with clinical features. *Med Oral Patol Oral Cir Bucal*.22 (2):e228-32

- Shanthala, B. M. . (2019). McDonald and Avery's Dentistry for the Child and Adolescent--E Book: Second South Asia Edition. Elsevier Health Sciences.
- Slater LJ. 2003. Dentigerous cyst versus paradental cyst versus eruption pocket cyst. *J Oral Maxillofac Surg*, 61:149.
- Som, P. M., & Miletich, I. (2018). Review of the embryology of the teeth *Neurographics*, 8(5), 369-393.
- Srinath SK, Sahana S, Vishwanath SK, Ritu S.2013. Mechanism of tooth eruption & its clinical significance - a systematic review of literature. *Elixir Dentistry*;65:19676–80.
- Stecker S, Diangells AJ (2002) Dens evaginatus: A diagnostic and treatment challenge. JADA 133: 190-193.
- Suda N, Hiyama S, & Kuroda T.2002. Relationship between formation/ eruption of maxillary teeth and skeletal pattern of maxilla. *Am J OrthodDentofacialOrthop*;121: 46-52.
- Suri L, Gagari E, & Vastardis H, 2004. Delayed tooth eruption: pathogenesis, diagnosis, and treatment. A literature review, *Am J Orthod Dentofacial Orthop* 126:435,

T

- Tanaka, J.. Ono, E. M. Filho, J. C. Castilho, L. C. Moraes, and M. E. Moraes, "Cleidocranial dysplasia, , 2006. importance of radiographic images in diagnosis of the condition," *Journal of Oral Science*,. 48, 3. 161–166.
- TapasyaJuneja & Gurkeerat Singh.2007. Textbook of Orthodontics Second Edition, p 39-48:P50-51

- Ten Cate A R, Sharpe P T, Roy S, & Nanci A.2003. Development of tooth and its supporting tissues .Oral Histology,4th ed. P::45-80.
- ThesleffI ,& Mikkola M.2002. The role of growth factors in tooth, Elsevier.
- Tittly K C. 1984.Comparative investigation of permanent tooth emergence timing of Northern Ontario Indians. Canad. *Dent. Assoc. J*; 50(10): 775-778.

W

- WANG XP.2013. Tooth eruption without roots. *Journal of dental research*, 92(3),212-214 .
- Wise GE.2009. Cellular and molecular basis of tooth eruption. *Orthod Craniofac Res*;12(2):67–73.