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Review Article

Etiology of Various Dental Developmental Anomalies -Review of Literature

Introduction

The development of the tooth involves many complex biological processes, including epithelial- mesenchymal interactions, differentiation, morphogenesis, fibrillogenesis and mineralization. After 37 days of development, a continuous band of thickened epithelium forms around the mouth in presumptive upper and lower jaws from the fusion of separate plates of thickened epithelium–primary epithelial band and gives rise to vestibular lamina and dental lamina [1].

A series of factors influence the normal development of the occlusion, interfering with the correct alignment of teeth and harmonic relationship with the adjacent and antagonistic elements [2]. The most spectacular period of development of the human body takes place in utero and during this period various disturbances may occur, producing changes which are congenital but not always inherited. The explanation for the tendency of the person to inherit certain features or characteristics from his parents is based upon the monumental principle observations of Mendel, who gave two principles on which the transmission of characteristics was based [3].

- Principle of dominance
- Principle of segregation

Developmental disturbances of the teeth may manifest by variations in number, position, size, shape, eruption or structure. Such disturbances may occur in association with some more generalized disorder or may occur independently [4]. General as well as local factors may operate to affect the form and structure of the teeth. It may be that only the form is altered or perhaps only the structure. In other instances both are disturbed. Such influences may begin either before or after birth, so that either deciduous or permanent teeth are involved. The treatment plan for the various congenital and hereditary disturbances depends upon the structural, functional and aesthetic requirements and the influencing factors such as the age of the patient, type of dentition, associated abnormalities etc. must be taken into consideration.

Discussion

The primitive oral cavity or stomodaeum is lined by stratified squamous epithelium called the oral ectoderm. The oral ectoderm contacts the endoderm of the foregut to form the buccopharyngeal membrane. At about the 27th day of gestation this membrane ruptures and the primitive oral cavity establishes a connection with the foregut [5]. The primitive oral band gives rise to two subdivisions, the vestibular lamina and dental lamina. The vestibule forms as a result of proliferation of the vestibular lamina into the ectomesenchyme (Figure 1). Within the dental lamina, continued and localized proliferative activity leads to the formation of a series of epithelial ingrowths into the ectomesenchyme at sites corresponding to the position

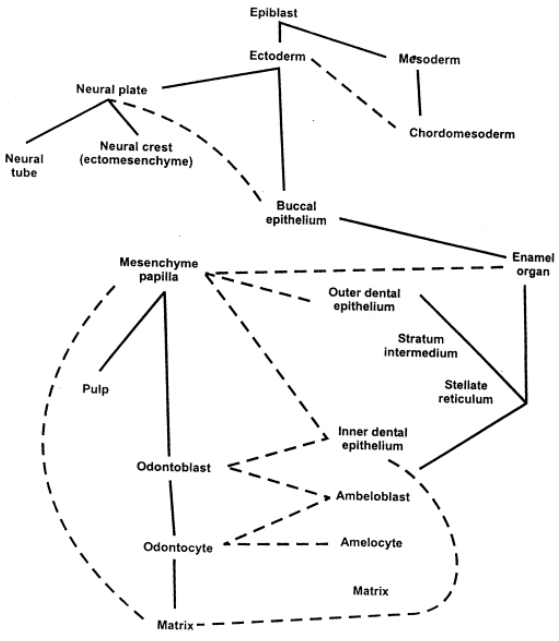

Figure 1: Outline of devolpment of tooth. Broken lines, known or suspected interactions that occur Between tissues.

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of future deciduous teeth [5]. The dental lamina serves as the primordium for the ectodermal portion of the deciduous teeth. Later during the development of the jaws, the permanent molars arise directly from a distal extension of the dental lamina [1].

Because of the complex nature of odontogenesis wherein cells undergo morphodifferentiation and histodifferentiation and where the changes in one group of cells are dependent upon another group of cells, there are many possibilities for disturbances in the development of teeth [5]. Both systemic and local conditions may affect the form and structure of the developing teeth (Table 1). In some instances only the gross appearance of the tooth is affected, the structure remaining normal; in others the structure itself is changed, or both form and structure may be involved [6,7].

Abnormalities of anatomical form and histological structure include multiple teeth, hyperplasia or overdevelopment, and hypoplasia or underdevelopment of the entire tooth and hypoplasia of part of the tooth, the crown or root. Dysplasia of the dental structures includes colour changes, hypo plastic defects which result from deficiencies, traumatic injury, pyogenic or specific infection of the developing teeth. The deciduous teeth which develop in utero and not as frequently affected as are the permanent ones. However, heredity, congenitally transmitted diseases, malnutrition, and diseases affecting the mother during gestation may have their effects on the deciduous teeth [7]. The dental anomalies have been classified according to the stage of development of tooth germ and according to the number, morphology and size and structure (Table 2) [8].

Disturbances during initiation of tooth germ

Hypodontia: Hypodontia is the term used to describe the developmental absence of one or more primary or secondary teeth, excluding the third molars (Figure 2). It is the most common developmental dental anomaly and can be challenging to manage clinically. The term oligodontia is used to define developmental absence of multiple teeth, usually associated with systemic manifestations [9]. Total anodontia denotes complete developmental absence of teeth in both dentitions [10]. The prevalence varies from 2.6% to 11.3% [9]. Hypodontia in the primary dentition is less common with reported prevalence rates varying between 0.5% to 2.4% [11].

Etiology: The etiology of hypodontia may arise as a familial condition; a high proportion of affected individuals are members of families with a previoushistory of the condition. The nature of the inheritance is complex and not well understood [9]. It has been regarded as a multifactorial condition with genetic and environmental influences playing a role. Hypodontia is also a common presenting feature in a number of systemic conditions, such as ectodermal dysplasia, cleft lip and palate, vander wounde syndrome, down syndrome, incontinentia pigmenti, hyalinosis cutis et mucosae, mandibulo-oculo-facial dyscephaly [8,10,12,13].

Brook suggested that in the majority of cases hypodontia has a polygenetic inheritance pattern and the risk of relatives having hypodontia will depend upon a combination of numerous genetic and environmental factors, each with a small effect [11].

Table 1: Experimental and Clinical Causes of Congenital Development Anomalies.

Genetic factors... Inherited Mutagenetic	Polyfunctional alkalating agents NH^2, ThioTEPA CB1348, TEM Traizene Quinine Pilocarpine Thallium Selenium Nicotine Sulfonamides Tetracyclines Chloroquine Thalidomide Salicylates Malachite green
1. Infections - Rubella Influenza A	
2. Physical Injuries..... Pressure Temperature changes Radiation	
3. Hormones ... Diabetes mellitus Hyperthyroidism Hypothyroidism ACTH Cortisone Androgenes Estrogen	
4. Nutrition..... Deficiencies of – Vitamin A Vitamin B complex Vitamin D Vitamin E Vitamin K Proteins Unsaturated fatty acids Potassium Excess of Vitamin A	7. Maternal diseases & Defects Uterine tumors Uterine inflammation Defects in implantation Age Emotional disturbances Stress Multiple prgenancies
5. Respiration. Hypoxia Carbon dioxide excess Carbon monoxide Anesthesia with ether gas-oxygen	8. Embryonic defects Abnormalities of the ovum Abnormalities of semen Antigen – antibody reactions
6. Miscellaneous drugs and chemicals ... Antimetabolites Aminopterin Amethopterin	

Table 2: Classification of developmental dental anomalies.

1. According to number, morophology and size and structure

A) Anomalies of tooth number
- i) Hypodontia
- ii) Hyperdontia

B) Anomalies of tooth size and morphology
- i) Microdontia
- ii) Macrodontia
- iii) Dens invaginatus
- iv) Dens evaginatus
- v) Talon's cusp
- vi) Taurodontism
- vii) Fusion
- viii) Gemination
- ix) Conscrescence
- x) Dilacertioin
- xi) Enamel Pearls
- xii) Supernumerary Cusps and rotos

C) Anomalies of tooth structure
- i) Amelogenesis imperfecta
- ii) Enamel Hypopalasia
- iii) Dentinogenesis imperfecta
- iv) Dentin Dysplasia
- v) Regional Odontodysplasia
- vi) Cemental hypoplasia
- vii) Hypercementoses
- viii) Interglobular dentin

2. According to the stage of development of tooth germ

A) Disturbances during initiation of tooth germ
- i) Hypodontia
- ii) Hyperdontia

B) Disturbances during histodifferentation and morophodifferentation of tooth germ
- i. Microdontia
- ii. Macrodontia
- iii. Dens invaginatus
- iv. Dens evaginatus
- v. Talon's cusp
- vi. Taurodontism
- vii. Fusion
- viii. Gemination
- ix. Concrescence
- x. Dilaceration
- xi. Supernumerary Cusps and roots

C) Disturbances during apposition of hard tissues
- i) Amelogenesis imperfecta
- ii) Enamel hypoplasia
- iii) Dentinogenesis imperfecta
- iv) Dentin Dysplasia
- v) Regional Odontodysplasia
- vi) Cemental hypoplasia
- vii) Hypercementoses
- viii) Interglobular dentin
- ix) Enamel Pearls

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Recent advances in the fields of human genetics and molecular biology are providing us with a greater understanding of tooth development. Those of particular interest in tooth development are the muscle specific homeobox genes Msx1 and Msx2. The proteins encoded by these homeobox genes are known as transcription factors, and can switch other genes on or off, therby controlling gene expression. The Msx1 gene appears to be more important in specification and induction, and Msx2 in further development of the tooth buds [14]. Msx1 gene knocked out had complete failure of tooth development. In humans, genetic linkage analysis of a family with severe hypodontia has demonstrated a mutation in the Msx1 gene, causing selective familial hypodontia [14].

Hyperdontia

Hyperdontia may manifest itself by the production of additional teeth, occurring either in succession as a predeciduous or a postpermanent arrangement or in contemporary arrangement to increase the number of any group of teeth [7] (Figure 3). The prevalence in the permanent dentition is between 0.15 and 1% with predilection of 2:1 for male sex while in the deciduous dentition varies from 0.3% to 0.66% [2].

Etiology: The aetiology of supernumerary teeth remains unclear, but several theories have been suggested for their occurrence. The phylogenetic process of atavism (evolutionary throwback) has been suggested to explain the development of supernumerary teeth [15]. According to the dichotomy theory, Taylor (1972) stated that the tooth bud splits into two equal or different-sized parts, resulting in two teeth of equal size or one normal and one dysmorphic tooth, respectively.

Sedano and Gorlin (1969) indicated the possibility of an autosomal dominant trait with lack of penetrance in some generations and Bruning, et al., (1957) has even suggested the possibility of sex-linked inheritance to explain the existence of a sex predominance of males over females [16]. Cadenat, et al., (1977) pointed out the presence of a recessive gene on an autosome and a gene on the inhibiting X chromosome. Brook (1984) proposed a combination of genetics and environmental factors to explain the occurrence of supernumerary teeth. Another possibility of the origin is that they are derived from clumps of epithelium that remain after the breaking up of the tooth band and become activated to tooth formation [15,17]. According to Dean, et al., (2001), the differentiation phase of the dental germ, will determine the appearance of a supernumerary tooth [2].

Disturbances during histodifferentiation and morpho-differentiation of tooth germ

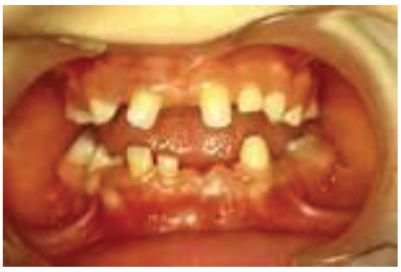
Microdontia: Microdontia is used to describe teeth which are smaller than normal i.e. outside the usual limits of variation [18] (Figure 4). The most frequently affected teeth are maxillary lateral incisor and third molars. It has been classified as True generalized microdontia – All the teeth are smaller than normal. Aside from its occurrence in some cases of pituitary dwarfism, this condition is exceedingly rare, Relative generalized microdontia – Normal or slightly smaller than normal teeth are present in jaws that are somewhat larger than normal and there is an illusion of true microdontia [8].

Etiology: Proportional microdontism is generally associated with dwarfism due to hypofunction of the pituitary gland. Small teeth in normal or large jaws may be due to cross inheritance [6]. Regression or atavism may be the cause of rudimentary development of individual teeth, which take on the cone-shaped or haplodont form of the reptile or fish dentition. This abnormality is frequently inherited and occurs especially in the weakest teeth, the maxillary second incisors [19].

Macrodontia

Macrodontia refers to the teeth that are larger than normal [18] (Figure 5).It has been classified as true generalized macrodontia – this condition in which all the teeth are larger than normal has been associated with pituitary gigantism, but is extremely rare Relative generalized macrodontia – In this normal or slightly larger than normal teeth are present in small jaws, the disparity in size giving the illusion of macrodontia, macrodontia of single teeth – It is relatively uncommon [3,10].

Etiology: Proportional gigantism is usually caused by hyperpituitarism which increases the length of the long bones and teeth. Disproportional dental gigantism, on the other



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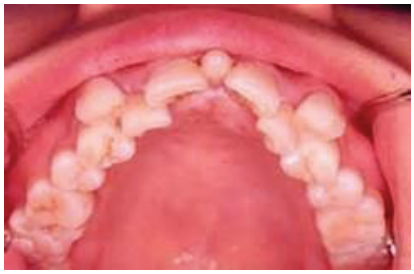

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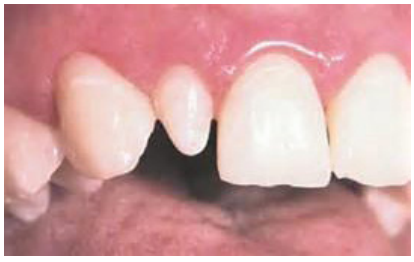

Figure 4:

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hand, is suggestive of cross inheritance – large teeth from one parent, small jaws and skeleton from the other6. Hyde (1938) mentioned that size is markedly influenced by heredity and that the inheritance of large teeth is a dominant character. Hrdiicka (1935) stated that size in teeth presents great variations and is a blend of inheritance rather than a single dominant [7].

Dens Invaginatus

Dens invaginatus is an embryologic anomaly that results in invagination of an amelodentinal structure, more or less developed, within the pulp [20] (Figure 6). Incidence ranges from 0.25% to as high as 10% [21,22].

Oehlers classified dens invaginatus in 3 categories according to the depth of penetration and communication with the periodontal ligament or periapical tissue: Type 1 cases are those in which the invagination ends as a blind sac confined to the crown, In Type 2, the invagination extends apically beyond the external cementoenamel junction, ending as a blind sac and never reaching the periapical tissues. In Type 3, the invagination also extends beyond the cementoenamel junction and a second "apical foramen" is evident in either the periapical tissues or the periodontal ligament [21,22].

Etiology: Several theories have been proposed for this phenomenon, but the etiology of dens invaginatus remains unclear. Kronfeld (1934) proposed that dens invaginatus is caused by a focal failure of growth of the internal enamel epithelium leading to proliferation of the surrounding normal epithelium with eventual engulfment of the static area [23]. Hulsmann (1997) suggested that a part of inner enamel epithelium proliferates faster than adjacent parts and invades the dental papilla [24]. Oehlers (1957) proposed that distortion of the enamel organ occurs during tooth development and results in protrusion of a part of the enamel organ. Other theories include infection (Fischer, 1936) trauma (Gustafson, 1950) and genetics (Hosey, 1996) as possible contributing factors [23]. Radicular invagination was described by Swanson proliferation of epithelial cells causing an apical ingrowth into the dental papilla, Hunter (1950) suggested that it should be classified as dilated odontome [7].

Dens evaginatus

Dens evaginatus is a rare dental anomaly involving an extra cusp or tubercle that protrudes from the occlusal surface of the affected tooth [25], (Figure 7). Also called as Occlusal Tuberculated Premolar, Leong's Premolar, Evaginated Odontome and Occlusal Enamel Pearl. Prevalence of Dens Evaginatus is between 1% and 4% [26].

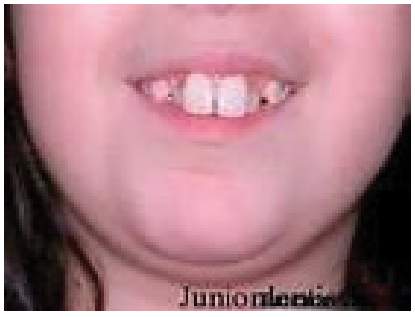
Etiology: It is the result of an abnormal proliferation of the inner enamel epithelium into the stellate reticulum of the enamel organ [25]. Lau (1955) described it as an odontoma of the axial core type. The family involvement and the association of the talon cusp with other dental abnormalities suggest that genetics may be a major causative factor [27]. However, sporadic occurrences of this abnormality probably are induced by trauma or other localized insults affecting the tooth germ.

Talon cusp

As early as 1892, Mitchell reported a maxillary central incisor with a horn-like protuberance projecting from the lingual surface [28]. 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 [27], (Figure 8). Its prevalence varies from less than 1% to approximately 8% [29].

Hattab, et al., classified this developmental anomaly into three types on the basis of cusp formation and extension ; Talon – A morphologically well – delineated additional cusp that prominently projects from the palatal surface of a primary and permanent anterior tooth and extends at least half the distance from the millim enamel junction to the incisal edge, Semi talon – An additional cusp of a millimeter or more extending less than half the distance from the cementoenamel junction to the incisal edge, Trace talon – Enlarged or prominent cingula and their variations, i.e. conical, bifid, or tuberclelike [16,29].

Etiology: It is suggested that this condition has a multifactorial



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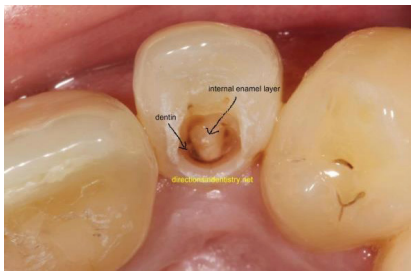

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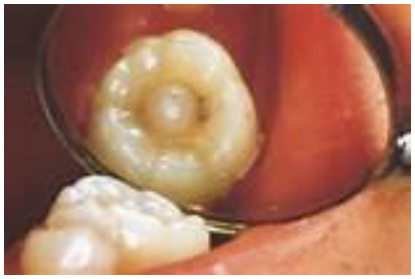

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etiology including both genetic and environmental factors (S M Garn, 1965) [29]. As with other dental abnormalities, talon cusp occurs during the morphodifferentiation stage or odontogenesis, Sicher and Bhaskar (1972) suggest that disturbance during morphodifferentation (such as altered endocrine function) might affect the shape and size of a tooth without impairing the function of the ameloblasts or odontoblasts. Another theory by Hattab, et al. [30], suggests that talon cusp might occur as a result of an outward folding of the inner enamel epithellal cells (preceursors of ameloblasts) and a transient focal hyperplasia or the mesenchymal dental papilla (precursors of odontoblasts) [31]. Genetics may be a major causative factor. This abnormality may also be induced by trauma or other localized insults affecting the tooth germ. Aberrant hyperactivity of the dental lamina may also, be responsible for its occurrence [29].

Taurodontism

Sir Arthur Keith introduced the term in 1913 to describe the "bull-like" condition in teeth (from Latin tauro; "bull" and don't: "tooth" from Greek"), although Gorjanovic-Kramberger, in 1908, was the first to describe this type of tooth 8, [32]. Taurodontism is a dental anomaly characterized by the enlargement of the pulp chamber, which may reach the proximity of the root apex (Figure 9). Most reports indicate a prevalence of 2.5 to 3.2% of the population and as high as 11% in the Middle Eastern population [19].

Etiology: The condition is thought to arise from a delay in transformation of the enamel organ into several sheets of Hertwig, a process which normally starts soon after completion of the crown. The etiology of taurodontism is heterogeneous. The trait appears to be due to by an autosomal dominant gene with variable expressivity. Jaspers and Witkop (1980, Jaspers (1981) and Townset (1990) suggested the association of taurodontism with X, chromosome aneuploidy [32]. A variety of possible causes of taurodontism have been enumerated by Mangion as follows : a specialized or retrograde character, a primitive pattern, a mendelian recessive trait, an atavistic feature, a mutation resulting from odontoblastic deficiency during dentinogensis of the roots.

Fusion

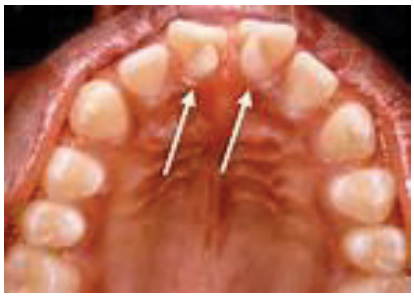
In 1963 Tannenbaum and Alling defined fusion as a union of two separate tooth buds at some stage in their development (Figure 10) Depending on the stage they are united, one tooth may have only one pulp chamber as a gemination, or there may be two pulp chambers, with union only of the dentin [33]. Fusion of teeth is relatively frequent, ranging from 0.5 percent to 2.5 percent [34].

Etiology: Fusion arises through the union of two normally separated tooth germs (Duncum 1987) [35]. Greth (1936) suggested that fused teeth are produced by some physical action, perhaps special pressure forcing young tooth germs into contact, causing necrosis of the intervening tissue and giving the enamel organ and the dental papilla an opportunity to unite. If this occurs very early, the crowns may fuse. At a later stage fusion would affect the roots only because the crowns have been separately developed [7]. If the contact occurs early, that is before calcification begins, the two teeth may be completely united to form a single large tooth. If the contact of teeth occurs later, when a portion of the tooth crown has completed its formation, there may be union of the roots only.

Gemination

In 1963 Tannenbaum and Alling, defined gemination as the formation of the equivalent of two teeth from the same follicle, with evidence of an attempt for the teeth to be completely separate, indicated clinically by a groove or depression which could delineate two teeth (Figure 11) The prevalence of geminated teeth ranges from 0.5% - 2.5%34.

Etiology: Geminated teeth are produced by abnormal odontogeny. Colyer (1926) was the first to show an irregular epithelial invagination in the enamel organ which seemed to be an attempt to divide it and form two teeth. The result is a bifid crown with confluent roots and root canals. Sprawson (1937) showed how symmetrical division will produce a tooth with bifid normal – appearing crowns, while asymmetrical



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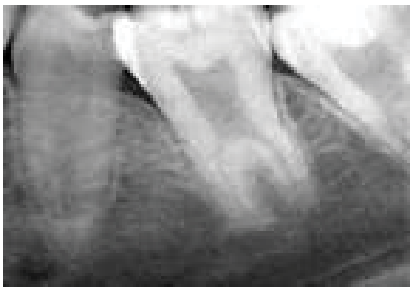

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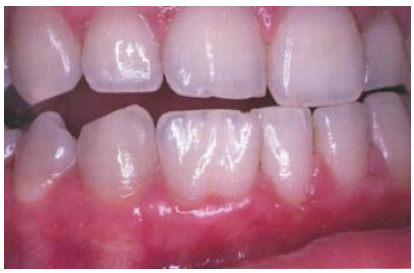

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invagination will produce a component not resembling a normal tooth (accessory tooth) Gemination is the result of either schizodontism, the splitting of a tooth germ during development or synodontism, the fusion of a normal tooth bud with one from a developing supernumerary tooth [8].

Concrescence

Concrescence is a form of fusion in which the union is only in the cementum of adjacent teeth and occurs after the root formation has been completed [3,6]. Concrescence is more frequently noted in maxillary molars (Figure 12). It can occur between normal molars, a normal molar and a supernumerary molar, and in both erupted and impacted teeth [8, 35].

Etiology: Although the exact etiology is unknown, it is thought to result from trauma or from crowding of the adjacent teeth such that the interdental bone resorbs allowing the adjacent tooth roots to become fused by the deposition of cementum between them [3,6,19]. It has also been postulated to result from an inflammatory response, for example, to a carious lesion, which causes cemental deposition and ultimately attachment to the root of the adjacent tooth [36]. Concrescence originates much later than gemination or fusion. It occurs at a time when the roots are established [7].

Dilaceration

Dilaceration is defined as an angular position between the two parts of a tooth [19,37] (Figure 13). This is due to an abnormality in the formation of the tooth whereby the calcified part is displaced in relation to the uncalcified part [37]. When a deciduous tooth is driven apically into the jaw, a displacement of or injury to the germ of the permanent successor may occur; either an angle between the crown and the root or a disturbance in the formation of the hard dental tissues may result.

Etiology: The germs of the permanent incisors are initially situated lingual to the apices of the deciduous teeth. During their further development, the germs gradually get nearer to the resorbing roots of the deciduous teeth. If the already calcified part of the germ is displaced, in relation to the unclarified part, by trauma, the result may be dilacerations or circular hypoplasia [38]. According to Meyer, displacement of the deciduous tooth produces a bone wound which is followed by scar formation, resulting in root curvature. The developing root pushes the calcified part against the scar, thus forcing it into an abnormal direction. It has been emphasized by Van Gool (1973) that dilaceration often follows traumatic injury to the deciduous predecessor, in which the tooth is driven apically into the jaw3 [37].

Conclusion

A series of factors can influence the normal development of the occlusion, interfering in correct alignment of the teeth and harmonic relationship with the adjacent and antagonistic elements. In order to evaluate discrepancies in dentition, it is necessary to be familiar with the normal development of the teeth and the stages involved in it. Early detection and diagnosis of dental anomalies are essential steps in evaluation of the child patient and in treatment planning. In presence of dental anomalies, the dentist should evaluate the moment that they begin to interfere in the normal developmental pattern of occlusion. Then intervention should occur as soon as possible to avoid malocclusion.

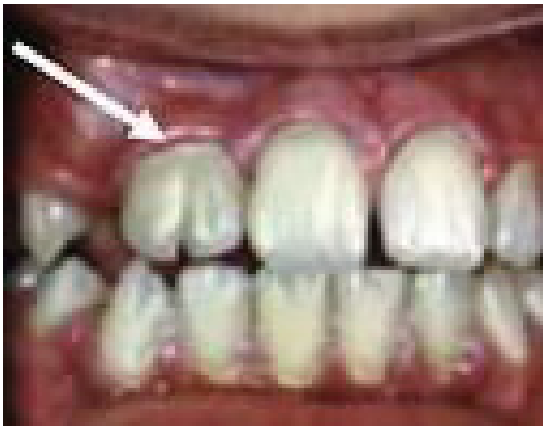
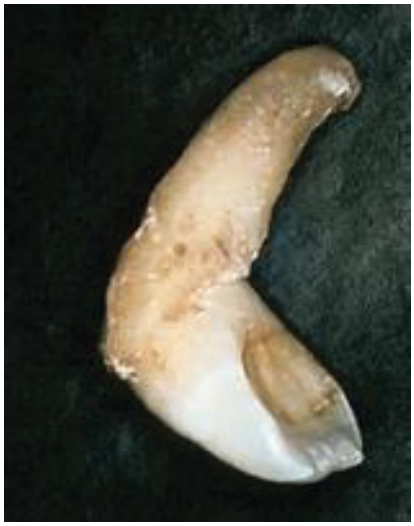


Figure 11:

Figure 12:



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References

1. Tencate AR (1998) Oral histology development, structure and function, 5th Edition, Mosby Publications. Link: https://goo.gl/PC47kp
2. Chevitarese ABA, Tavares CM, Primo L (2003) Clinical complications associated with supernumerary teeth : A case report. J Clin Pediatr Dent 28: 27-32. Link: https://goo.gl/aNwAZL
3. Shafer, Hine, Levy (1983) A textbook of Oral Pathology. 4th Edition, Saunders International. Link: https://goo.gl/H9MFaL
4. Kerr and Ash (1986) An introduction to general and oral pathology for hygienist, 5th edition, Lea and Febiger Philadelphia. Link: https://goo.gl/fmcvdS
5. Orbans (1997) Oral histology and embryology, 11th Edition, CBS publishing and distribution. Link: https://goo.gl/H3114A
6. Bernier JL (1959) The management of oral disease, 2nd Edition, Mosby Publications.
7. Thoma KH (1954) Histological, roentengenological and clinical study of the disease of teeth, jaws and mouth 4th edition, Mosby Publications.
8. Eversole LR (1986) Clinical outline of oral pathology. Diagnosis and treatment, 2nd Edition, Lea and Febiger Philadelphia. Link: https://goo.gl/INSVaF
9. Dhanrajani PJ (2002) Hypodontia: Etiology, clinical features and management. Quintessence Int 33: 294-302. Link: https://goo.gl/CFKwK0
10. Stones HH (1966) Oral pathology 5th Edition.
11. Cunha RF, Delbem ACB, Hirata E, Toyota E (1999) Hypodontia in primary dentition: A case report. J Clin Pediatr Dent 23: 361-363. Link: https://goo.gl/5kQvUT
12. Paschos E, Huth KC, Hickel R (2002) Clinical management of hypohidrotic ecotodermal dysplasia with anodontia: A case report. J Clin Pediatr Dent 27: 5-8. Link: https://goo.gl/MgJUBA
13. Shashikiran ND, Karthik V, Subbareddy VV (2002) Multiple congenitally missing primary teeth: A case report. Pediatr Dent 24: 149-152. Link: https://goo.gl/Y3RzYE
14. Larmour CJ, Mossey PA, Thind BS, Forgie AH, Stirrups DR (2005) Hypodontia – A retrospective review of prevalence and etiology Quintessence Int 36: 263-270. Link: https://goo.gl/fHAJoh
15. Ibricevic H, Al-Mesad S, Mustagrudic D, Al-Zohsjry N (2003) Supernumerary teeth causing impaction of permanent maxillary incisors : consideration of treatment J Clin Pediatr Dent 27: 327-332. Link: https://goo.gl/BwflkJ
16. Nadkarni UM, Munshi A, Damle SG (2002) Unusual presentation of talon cusp: Two case reports. International Journal of Paediatric Dentistry 12: 332-335. Link: https://goo.gl/GZ1Oq2
17. Rajab LD & Hamdan MA (2002) Supernumerary teeth : Review of the literature and a survey of 152 cases. International Journal of Paediatric Dentistry 12: 244-254. Link: https://goo.gl/MytCxZ
18. Namdar F, Atasu M (1999) Macrodontia in association with a contrasting character microdontia. J Clin Pediatr Dent 23:271-274. Link: https://goo.gl/31Q1Ui
19. Cawson RA and Odell EW: Essentials of Oral pathology and Oral Medicine; Churchill Livingstone.
20. 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. Link: https://goo.gl/DJkcsM
21. Chen YH, Tseng CC, Harn WM (1998) Dens invagininatus : Review of formation and morphology with 2 case reports. Oral Surg Oral Med Oral Pathol Oral Radiot Endod 86: 347-352. Link: https://goo.gl/aOeozL
22. Holan G (1998) Dens invaginatus in a primary canine; a case report. International Journal of Paediatric Dentistry 8:61-64. Link: https://goo.gl/rSHgWx
23. Nallapati S (2004) Clinical management of a maxillary lateral incisor with vital pulp and type 3 Dens invaginatus: A case report JOE 30: 726-731. Link: https://goo.gl/yiHBJW
24. Hulsmann M, Hengen G (1996) Severe dens invaginatus malformation: report of 2 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 82: 456-457. Link: https://goo.gl/rwSSUU
25. Stecker S, Diangells AJ (2002) Dens evaginatus: A diagnostic and treatment challenge. JADA 133: 190-193. Link: https://goo.gl/rbO9G9
26. Forgie AH, Thind BS, Larmour CJ, Mossey PA, Stirrups DR (2005) Management of hypodontia: Restorative considerations. Quintessence Int 36: 437-445. Link: https://goo.gl/Z8RHaK
27. Segura-Egea JJ, Jimenez-Rubio A, Rios-Santos JV, Velasco-Ortega E (2003) Eugenio Velasco-Ortega. Dens evaginatus of anterior teeth (talon cusp): Report of five cases. Quintessence Int 34: 272-277. Link: https://goo.gl/4epDRb
28. Dankner E, Harari D, Rotstein I (1996) Dens evaginatus of anterior teeth – literature review and radiographic survey of 15,000 teeth, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 81: 472-476. Link: https://goo.gl/KLcqIX
29. Soares AB, De Araujo, JJ, de Sousa, SMG, et al. (2001) Bilateral talon cusp: Case report. Quintessence Int 32: 283-286. Link: https://goo.gl/DfWN1I
30. Hattab FN, Yassin OM, Al-NImri KS (1995) Talon cusp – clinical significance and management: case reports. Q I 26 : 115-120. Link: https://goo.gl/d9fUvt
31. Gungor HC, Altay N, Kaymazz FF (2000) Pulpal tissue in bilateral talon cusps of primary central incisors: A case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 89: 231-235. Link: https://goo.gl/ahxpIJ
32. Genc A, Namdar F, Goker K, Atasu M (1999) Taurodontism. In association with supernumerary teeth. J Clin Pediatr Dent 23: 151-154. Link: https://goo.gl/TLuRx3
33. Bhaskar. Synopsis of oral pathology 1st Indian Edition, 1990; CBS publishing and distribution.
34. Eidelman E (1981) Fusion of maxillary central and lateral incisors bilaterally. The American Academy of Pedodontics 3: 346-347. Link: https://goo.gl/nuZvVH
35. Gedik R, Cimen M (2000) Multiple taurodontism: A case report. Journal of dentistry for children 216-217.
36. Romito LM, Omaha (2004) Concrescence: Report of a rate case. Oral Med Oral Pathol Oral Radiol Endod 97: 325-327.
37. Van Gool AV (1973) Injury to the permanent tooth germ after trauma to the deciduous predecessor. Oral Surg 35: 2-12. Link: https://goo.gl/VvJFXx
38. Hegde S, Munshi AK (2001) Management of an impacted, dilacerated mandibular left permanent first molar: A case report. Quintessence Int 32: 235-237. Link: https://goo.gl/XZZLSL

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