REVIEW ARTICLE

Delayed tooth eruption: Pathogenesis, diagnosis, and treatment. A literature review

Lokesh Suri, BDS, DMD, MS,[a] Eleni Gagari, DDS, DMSc,[b] and Heleni Vastardis, DDS, DMSc[c]
Boston, Mass

Delayed tooth eruption (DTE) is the emergence of a tooth into the oral cavity at a time that deviates significantly from norms established for different races, ethnicities, and sexes. This article reviews the local and systemic conditions under which DTE has been reported to occur. The terminology related to disturbances in tooth eruption is also reviewed and clarified. A diagnostic algorithm is proposed to aid the clinician in the diagnosis and treatment planning of DTE. The sequential and timely eruption of teeth is critical to the timing of treatment and the selection of an orthodontic treatment modality. This review addresses the need for a more in-depth understanding of the underlying pathophysiology of DTE and gives the clinician a methodology to approach its diagnosis and treatment. (Am J Orthod Dentofacial Orthop 2004;126:432-45)

Eruption is the axial movement of a tooth from its nonfunctional position in the bone to functional occlusion. However, *eruption* is often used to indicate the moment of emergence of the tooth into the oral cavity. The normal eruption of deciduous and permanent teeth into the oral cavity occurs over a broad chronologic age range. Racial, ethnic, sexual, and individual factors can influence eruption and are usually considered in determining the standards of normal eruption.[1-3] True and significant deviations from accepted norms of eruption time are often observed in clinical practice. Premature eruption has been noted,[4,5] but delayed tooth eruption (DTE) is the most commonly encountered deviation from normal eruption time.

Eruption is a physiologic process that strongly influences the normal development of the craniofacial complex.[2,6] Often, DTE might be the primary or sole manifestation of local or systemic pathology.[7] A delay in eruption can directly affect the accurate diagnosis, overall treatment planning, and timing of treatment for the orthodontic patient. Thus, DTE can have a significant impact on a patient's proper health care.

The importance of DTE as a clinical problem is well reflected by the number of published reports on the subject, but there is considerable controversy regarding the terminology used and the pathogenesis of DTE. Here, we propose to systematically review the literature for reports on the diagnosis and treatment of DTE, to classify the etiology and pathogenesis of DTE, and to clarify the relevant terminology.

From the School of Dental Medicine, Tufts University, Boston, Mass.
[a]Assistant professor, Department of Orthodontics.
[b]Associate professor, Department of Oral and Maxillofacial Pathology.
[c]Research assistant professor, Department of Orthodontics.
Reprint requests to: Dr Lokesh Suri, Tufts University, School of Dental Medicine, Department of Orthodontics–DHS-2, One Kneeland St, Boston, MA 02111; e-mail, lokesh.suri@tufts.edu
Submitted, June 2003; revised and accepted, October 2003.
0889-5406/$30.00

doi:10.1016/j.ajodo.2003.10.031

DEFINITIONS AND TERMINOLOGY

Many terms have been used in the literature to describe disorders of tooth eruption (Table I). There seems to be considerable confusion concerning their usage. *Eruption* is the developmental process responsible for moving a tooth from its crypt position through the alveolar process into the oral cavity to its final position of occlusion with its antagonist. It is a dynamic process that encompasses completion of root development, establishment of the periodontium, and maintenance of a functional occlusion.[2] *Emergence*, on the other hand, should be reserved for describing the moment of appearance of any part of the cusp or crown through the gingiva. Emergence is synonymous with *moment of eruption*, which is often used as a clinical marker for eruption.

Impacted teeth are those prevented from erupting by some physical barrier in their path. Common factors in the etiology of impacted teeth include lack of space due to crowding of the dental arches or premature loss of deciduous teeth. Frequently, rotation or other positional deviation of tooth buds results in teeth that are "aimed" in the wrong direction, leading to impaction. *Primary retention* has been used to describe the cessation of eruption of a normally placed and developed tooth germ before emergence, for which no physical barrier can be identified.[8,9]

Pseudoanodontia is a descriptive term that indicates clinical but not radiographic absence of teeth that

Table I. Delayed tooth eruption: terminology used in the literature

Delayed eruption
Impacted teeth
Primary retention
Embedded teeth
Pseudoanodontia
Late eruption
Retarded eruption
Arrested eruption
Primary failure of eruption
Misplaced teeth
Displaced teeth
Impaired eruption
Depressed eruption
Noneruption
Submerged teeth
Reinclusion/inclusion of teeth
Paradoxical eruption

should normally be present in the oral cavity for the patient's dental and chronologic age. In these cases, radiographic examination discloses the teeth in the jaws. These persons might have retained their deciduous teeth; more commonly, the deciduous teeth have been shed, but the permanent ones failed to erupt.

Controversy exists in the literature about the use of "delayed," "late," "retarded," "depressed," and "impaired" eruption. Root development has been taken as a basis for distinguishing some of these terms. In 1962, Gron[10] showed that, under normal circumstances, tooth eruption begins when 3/4 of its final root length is established. However, at the moment of eruption, mandibular canines and second molars show more advanced root development than the expected 3/4 of the final root length, whereas mandibular central incisors and first molars show root development less than 3/4 of the expected final root length. Becker[11] suggests that root development alone should be the basis for defining the expected time of eruption for different teeth. Thus, if an erupted tooth has less root development than the expected 3/4 of length, its eruption is deemed premature, whereas if the tooth has developed more than the root length expected for eruption and remains unerupted, it should be defined as having delayed eruption.

Rasmussen and Kotsaki,[12] on the other hand, suggest using established norms for mean eruption ages calculated from population studies. According to them, when the emergence of a tooth is more than 2 standard deviations (SDs) from the mean of established norms for eruption times, it should be considered delayed eruption. However, the authors further propose the terms "late" and "retarded" eruption, to be used on the basis of root development. It would then seem that Rasmussen's "retarded eruption" coincides with Becker's and Gron's "delayed eruption," and that "late eruption" is used when a tooth's eruption status is compared with chronologic eruption times defined by population studies.

The terms "depressed" and "impaired" eruption have also been used synonymously with delayed, late, or retarded eruption. However, most of these reports refer to comparisons of observed eruption times with the chronologic standards set by population studies. Thus, "late eruption" used by Rasmussen would describe these conditions best.

Primary or idiopathic failure of eruption is a condition described by Profitt and Vig,[13] whereby nonankylosed teeth fail to erupt fully or partially because of malfunction of the eruption mechanism. This occurs even though there seems to be no barrier to eruption, and the phenomenon is considered to be due to a primary defect in the eruptive process.[13-15] Terms such as arrested eruption and noneruption have been used interchangeably to describe a clinical condition that might have represented ankylosis, impaction, or idiopathic failure of eruption. These terms refer more to the pathogenesis of DTE than to the benchmarks that define DTE.

Embedded teeth are teeth with no obvious physical obstruction in their path; they remain unerupted usually because of a lack of eruptive force.[16,17] *Submerged teeth* and *inclusion/reinclusion of teeth* refer to a clinical condition whereby, after eruption, teeth become ankylosed and lose their ability to maintain the continuous eruptive potential as the jaws grow.[18,19] Such teeth then seem to lose contact with their antagonists and might eventually be more or less "reincluded" in the oral tissues. This condition should not be confused with chronologic delayed eruption, because the eruption was normal according to both chronologic and biologic parameters (root formation), but the process was halted. *Paradoxical eruption* simply has been used to represent abnormal patterns of eruption and can encompass many of the above conditions.[18]

Although many terms are used to characterize DTE, they all refer to 2 fundamental parameters that influence this phenomenon: (1) expected tooth eruption time (chronologic age), as derived from population studies, and (2) biologic eruption, as indicated by progression of root development. Chronologic age has been used quite often to describe DTE. The advantage of using chronologic norms of eruption lies in the ease of use. Although not necessarily representating biologic age, expected time of tooth eruption often helps in forming a baseline for further clinical evaluation of a patient. Eruptive movements are closely related clinically with

tooth development.[1,2,10,20] Tooth eruption begins after root formation has been initiated. During eruption of teeth, many processes take place simultaneously: the dental root lengthens, the alveolar process increases in height, the tooth moves through the bone, and, in cases of succedaneous teeth, there is resorption of the deciduous tooth. These parameters are currently used as clinical markers for orthodontic treatment planning.

We propose a classification scheme (Fig 1) that takes into account these parameters, allowing the clinician to follow a diagnostic algorithm for DTE and its etiology. In this scheme, we sequentially examine several aspects of tooth eruption. First, we examine the patient's age and clinically apparent dentition. We define as *chronologic DTE* the eruption time that is greater than 2 SDs from the mean expected eruption time for a specific tooth (chronologic norm of eruption). A second step includes determining the presence or absence of a factor that adversely affects tooth development. This will prompt the clinician to consider certain diseases that result in defects of tooth structure, size, shape, and color. If tooth development is unaffected by any such factor, the third step is to consider the patient's dental age as evidenced by root formation. *Normal biologic eruption time* is defined as tooth eruption that occurs when the dental root is approximately 2/3 its final length. *Delayed biologic eruption* is defined as tooth eruption that has not occurred despite the formation of 2/3 or more of the dental root. Thus, if a patient has chronologic delayed eruption, he or she might simply be of a dental age that does not fit the norms (root length less than 2/3).

In Tables II and III, we attempt to categorize different conditions associated with DTE on the basis of hypothesized mechanisms. Further division into separate categories is also suggested to help in the classification and diagnostic scheme.

PATHOGENESIS AND DIFFERENTIAL DIAGNOSIS

The process of normal eruption and the source of eruptive forces are still controversial topics. This section reviews reported mechanisms that lead to DTE in some local and systemic conditions.

Local conditions

Local conditions causing DTE are listed in Table II. Physical obstruction is a common local cause of DTE of at least 1 tooth. These obstructions can result from many different of causes, such as supernumerary teeth, mucosal barrier, scar tissue, and tumors. DTE has been reported to occur in 28% to 60% of white people with supernumerary teeth.[21] Supernumerary teeth can cause crowding, displacement, rotation, impaction, or delayed eruption of the associated teeth. The most common supernumerary tooth is the mesiodens, followed by a fourth molar in the maxillary arch.[16,17] Different forms of supernumerary teeth have been associated with different effects on the dentition; the tuberculate type is more common in patients with DTE, whereas the conical form has been associated with displacement.[22] Odontomas and other tumors (in both the deciduous and permanent dentitions) have also been occasionally reported to be responsible for DTE.[23-28] In many of these case reports, the DTE was actually the alerting sign for diagnosing these conditions. Regional odontodysplasia, also called "ghost teeth," is an unusual dental anomaly that might result from a somatic mutation or could be due to a latent virus in the odontogenic epithelium.[17,29] Affected teeth exhibit a delay or total failure in eruption. Their shapes are markedly altered, generally very irregular, often with evidence of defective mineralization. Central incisors, lateral incisors, and canines are the most frequently affected teeth, in either the maxillary or mandibular arch, and deciduous and permanent teeth can be affected.[17] Abnormality in the tooth structure itself might be responsible for the eruptive disorders seen in this condition.

Mucosal barrier has also been suggested as an etiologic factor in DTE.[28,30-32] Any failure of the follicle of an erupting tooth to unite with the mucosa will entail a delay in the breakdown of the mucosa and constitute a barrier to emergence. Histologic studies have shown differences in the submucosa between normal tissues and tissues with a history of trauma or surgery.[30] 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 be an impediment to tooth eruption.[33] Injuries to deciduous teeth have also been implicated as a cause of DTE of the permanent teeth. Smith and Rapp,[34] in a cephalometric study of the developmental relationship between deciduous and permanent maxillary central incisors, found that the bony tissue barrier between the deciduous incisor and its successor has a thickness of less than 3 mm. This intimate relationship between the permanent and deciduous incisors is maintained during the developmental years. Traumatic injuries can lead to ectopic eruption[35,36] or some disruption in normal odontogenesis in the form of dilacerations[37,38] or physical displacement of the permanent germ.[35,36] Cystic transformation of a nonvital deciduous incisor might also cause delay in the eruption of the permanent successor.[39] In some instances, the traumatized deciduous incisor might become ankylosed or delayed in its

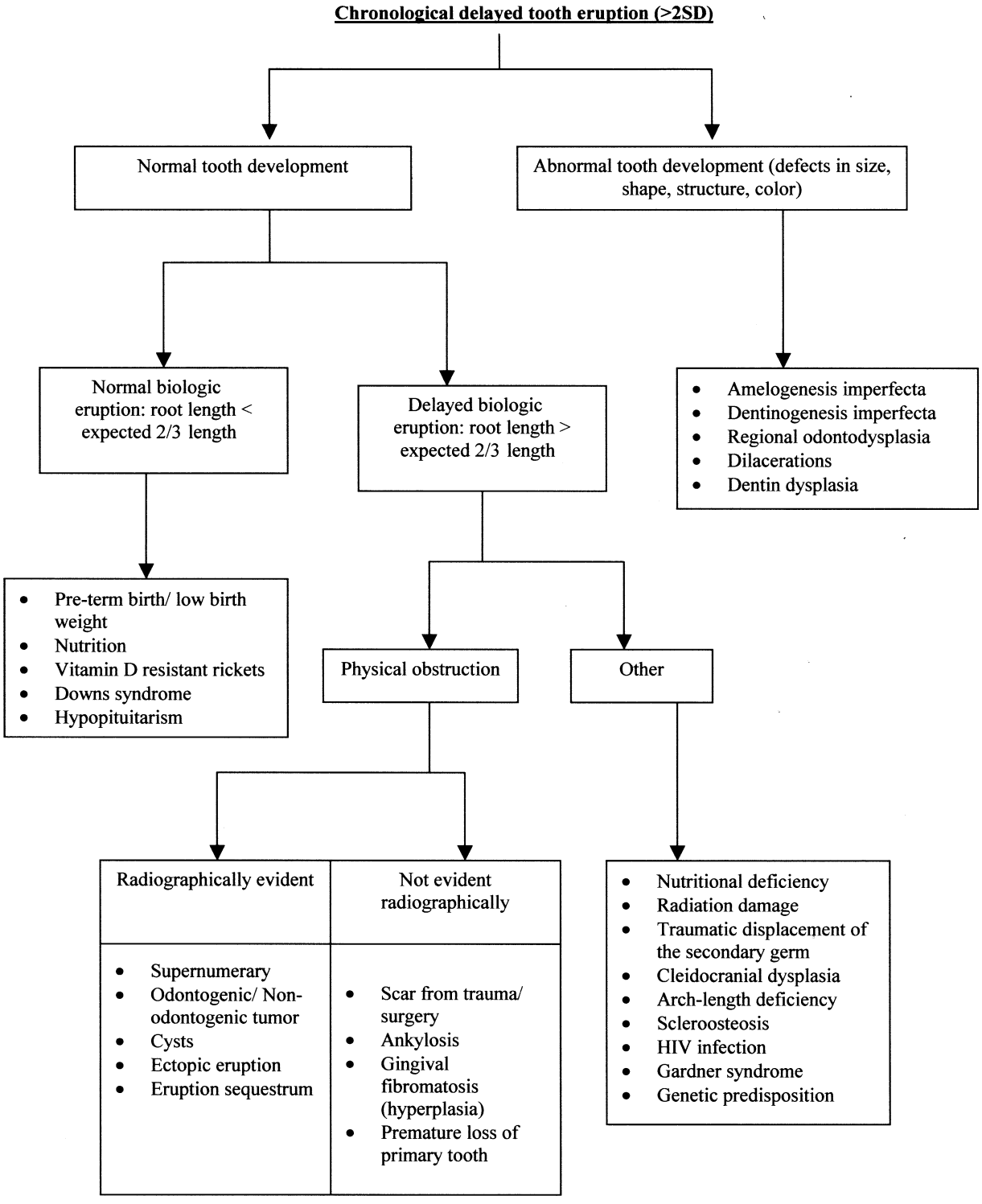

Fig 1. A diagnostic algorithm for DTE.

Table II. Conditions reported in literature to be associated with DTE

Local	*Systemic*
Mucosal barriers-scar tissue: trauma/surgery (28,30-32)	Nutrition (51-53,128)
Gingival fibromatosis/ gingival hyperplasia (33)	Vitamin D-resistant rickets (17,83)
Supernumerary teeth (116-119)	Endocrine disorders (54-58,129)
Odontogenic tumors (eg, adenomatoid odontogenic tumors, odontomas) (23-28,120)	Hypothyroidism (cretinism)
	Hypopituitarism
	Hypoparathyroidism
	Pseudohypoparathyroidism
Nonodontogenic tumors (16,17)	Long-term chemotherapy (130)
Enamel pearls (121)	HIV infection (61,62)
Injuries to primary teeth (122)	Cerebral palsy (63)
Ankylosis of deciduous teeth (38,44,45)	Dysosteosclerosis (131)
Premature loss of primary tooth (36,38,42)	Drugs
Lack of resorption of deciduous tooth (40,41)	Phenytoin (132,133)
Apical periodontitis of deciduous teeth (38,39)	Anemia (65)
Regional odontodysplasia (29)	Celiac disease (134)
Impacted primary tooth (123)	Prematurity/low birth weight (59,60)
Ectopic eruption	Ichthyosis (83)
Arch-length deficiency and skeletal pattern (48,77)	Other systemic conditions: renal failure (66), cobalt/lead or other heavy metal intoxication (135), exposure to hypobaria (64)
Radiation damage (49,124)	Genetic disorders
Oral clefts (125,126)	Familial/inherited (12,136-138)
Segmental odontomaxillary dysplasia (127)	Tobacco smoke (139)
	Idiopathic (140)

Numbers in parentheses are reference citations.

root resorption.[40,41] This also leads to the overretention of the deciduous tooth and disruption in the eruption of its successor. The eruption of the succedaneous teeth is often delayed after the premature loss of deciduous teeth before the beginning of their root resorption. This can be explained by the abnormal changes that might occur in the connective tissue overlying the permanent tooth and the formation of thick, fibrous gingiva.[38,42] 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.[8,9,43-45] Ankylosis occurs commonly in the deciduous dentition, usually affecting the molars, and has been reported in all 4 quadrants, although the mandible is more commonly affected than the maxilla. Ankylosed teeth will remain stationary while adjacent teeth continue to erupt through continued deposition of alveolar bone, giving the clinical impression of infraocclusion.[9,43,46,47]

Arch-length deficiency is often mentioned as an etiologic factor for crowding and impactions.[8,9] In a recent study of the relationship between formation and eruption of the maxillary teeth and the skeletal pattern of the maxilla, a shortened palatal length was found to delay the eruption of the maxillary second molar, although no delay in tooth formation was observed.[48] Arch-length deficiency might lead to DTE, although more frequently the tooth erupts ectopically.

X-radiation has also been shown to impair tooth eruption. Ankylosis of bone to tooth was the most relevant finding in irradiated animals. Root formation impairment, periodontal cell damage, and insufficient mandibular growth also seem to be linked to tooth eruption disturbances due to x-radiation.[49,50] Occasionally, localized DTE might be idiopathic.

Systemic conditions

Systemic conditions causing DTE are listed in Table II. The influence of nutrition on calcification and eruption is less significant compared with other factors, because it is only at the extremes of nutritive deprivation that the effects on tooth eruption have been shown.[51-53] Nevertheless, delayed eruption is often reported in patients who are deficient in some essential nutrient. The high metabolic demand on the growing tissues might influence the eruptive process.[52,53] Disturbance of the endocrine glands usually has a profound effect on the entire body, including the dentition. Hypothyroidism, hypopituitarism, hypoparathyroidism, and pseudohypoparathyroidism are the most common endocrine disorders associated with DTE. In hypothyroidism, failure of thyrotropic function on the part of

Table III. Genetic disorders associated with DTE

- Amelogenesis imperfecta (141) and associated disorders (115)
  - Enamel agenesis and nephrocalcinosis
  - Amelo-onychohypohydrotic dysplasia
  - Tricho dento-osseous syndrome (types I and II)
- Apert syndrome (67,68)
- Carpenter syndrome (81)
- Cherubism (7)
- Chondroectodermal dysplasia (Ellis-van Creveld syndrome) (17)
- Cleidocranial dysplasia (142)
- Congenital hypertrichosis lanuginosa (143)
- Dentin dysplasia (144)
- Mucopolysaccharidosis (MPS)
  - DeLange syndrome (83)
  - Hurler syndrome (MPS I-H) (17,69,145)
  - Hurler Scheie syndrome (MPS I-H/S) (9)
  - Hunter syndrome (MPS II) (9)
  - Pyknodysostosis (Maroteaux-Lamy syndrome) (MPS IV) (17)
- Down syndrome (146)
- Dyskeratosis congenita (147)
- Ectodermal dysplasia (83)
- Ekman-Westborg-Julin syndrome (148)
- Epidermolysis bullosa (149)
- GAPO syndrome (growth retardation, alopecia, pseudoanodontia, and optic atrophy) (69)
- Gardner syndrome (71,74)
- Gaucher disease (150)
- Gingival fibromatosis associated syndromes (9)
- Laband syndrome
- Murray-Puretic-Drescher syndrome
- Rutherford syndrome
- Cross syndrome
  - Ramon syndrome
  - Gingival fibromatoses with sensorineural hearing loss
  - Gingival fibromatoses with growth hormone dificiency
- Gorlin syndrome (151)
- Hallermann-Streiff syndrome (69,152)
- Hyperimmunoglobulinemia E (Buckley syndrome) (83)
- I-cell disease (mucolipidosis II) (153)
- Incontinentia pigmenti (Bloch-Sulzberger syndrome) (154)
- Mc-Cune-Albright syndrome (polyostotic fibrous dysplasia) (17)
- Menkes' kinky hair syndrome (155)
- Neurofibromatoses (156,157)
- Oculoauriculo vertebral spectrum (Goldenhar syndrome/hemifacial microsomia) (69,158)
- Osteoglophonic dyspalsia (69)
- Osteopathia striata with cranial stenosis (69,159)
- Osteopetrosis (marble bone disease) (78,79)
- Osteogenesis imperfecta (160,114)
- Otodental dysplasia (161)
- Parry-Romberg syndrome (progressive hemifacial atrophy) (162)
- Progeria (Hutchinson-Gilford syndrome) (17,69)
- Rothmund-Thompson syndrome (9,69)
- Sclerosteosis (80)
- Shokier syndrome (hereditary anodontia spuria)
- SHORT syndrome (9,69)
- Singleton-Merten syndrome (163)
- VonRecklinghausen neurofibromatosis (164)
- 22q11 deletion syndrome (165)

Numbers in parentheses are reference citations.

the pituitary gland or an atrophy or destruction of the thyroid gland per se leads to cretinism (congenital hypothyroidism) in a growing person. The dentofacial changes in cretinism are related to the degree of thyroid deficiency.[54-56] In hypopituitarism or pituitary dwarfism, the eruption and shedding of the teeth are delayed, as is the growth of the body in general.[54,57,58] The dental arch has been reported to be smaller than normal; thus it cannot accommodate all the teeth, so a malocclusion develops. The roots of the teeth are shorter than normal in dwarfism, and the supporting structures are retarded in growth.

Retardation of dental growth and development in preterm babies has been reviewed by Seow[59] and identified as a cause of DTE. Teething is often delayed, and Seow's results have shown a distinct relationship between birth weight and numbers of erupted teeth. DTE is common in preterm babies with respect to the deciduous dentition, but "catch-up" development occurred in later infancy. The permanent teeth showed a significant mean delay in dental maturation of approximately 3 months in very low birth-weight babies (birth weight of $<$1500 g). In another study, Seow[60] found that children with a birth weight less than 1000 g and gestational ages less than 30 weeks had the greatest lag in dental maturation. A correlation between human immunodeficiency virus (HIV) infection and DTE has also been suggested. A study[61] of dental manifestations in 70 children perinatally infected with HIV indicates that delayed dental eruption (defined as dental age 6 or more months younger than chronologic age) was directly associated with clinical symptoms. DTE did not seem to correlate with CD4 positive T-lymphocyte depletion. The investigators concluded that HIV infection itself is not associated with DTE, but, rather, the onset of the clinical symptoms is. Another study[62] has found that a lower tooth count at different chronologic ages in HIV-infected children might represent a marker for socioeconomic status, reflecting poorer nutrition or health. In a study of children with cerebral palsy, Pope and Curzon[63] found that unerupted deciduous and permanent teeth were more common in them compared with the controls. The first permanent molar erupted significantly later. No etiology or implicated mechanisms were elaborated.

Other systemic conditions associated with impairment of growth, such as anemia (hypoxic hypoxia,[64] histotoxic hypoxia, and anemic hypoxia[65]) and renal failure,[66] have also been correlated with DTE and other abnormalities in dentofacial development.

Genetic disorders

Genetic disorders causing DTE are listed in Table III. DTE has been found to be a feature in many genetic disorders and syndromes. Various mechanisms have been suggested to explain DTE in these conditions. A generalized developmental delay in permanent tooth formation is seen in Apert syndrome.[67,68] Supernumerary teeth have been found to be responsible for DTE in Apert syndrome,[67,69] cleidocranial dysostosis,[70] and Gardner syndrome.[71] There is considerable evidence to implicate the periodontal tissues' development in DTE. Abnormalities in these tissues, as have been found in some syndromes, might be a factor in DTE. Lack of cellular cementum has been found in cleidocranial dysplasia[72,73]; cementum-like proliferations and obliteration of periodontal-ligament space with resultant ankylosis have been noted in Gardner syndrome.[74] Tooth eruption is also regulated by various cytokines, including epidermal growth factor,[75] transforming growth factor-β, interleukin-1, and colony stimulating factor-1.[75-77] Lack of appropriate inflammatory response, an inadequate expression of some cytokines, and increased bone density that impedes resorption have been noted to be factors for DTE in some syndromes. In osteopetrosis,[78,79] sclerosteosis,[80] Carpenter syndrome,[81] Apert syndrome,[67,68] cleidocranial dysplasia,[82] pyknodysostosis,[17] and others, underlying defects in bone resorption and other operating mechanisms might be responsible for DTE. Conversely, bone resorption is enhanced in hyperimmunoglobulin E syndrome, but DTE has been noted as a feature of this condition. This has been suggested to be due to defective root resorption of the deciduous teeth or the presence of a protective factor on the root that resists physiologic resorption.[83] Tumors and cysts in the jaws can also cause interference with tooth eruption. Occasionally, some syndromes or genetic disorders are associated with multiple tumors or cysts in the jaws, and these might lead to generalized DTE. Gorlin syndrome, cherubism, and Gardner syndrome are such disorders, in which DTE might be the result of interference to eruption by these lesions. Occasionally, families are found in which a generalized delay in the eruption of teeth is noted. Patient medical history might be totally unremarkable, with DTE as the only finding. The presence of a gene for tooth eruption has also been suggested, and its "delayed onset" might be responsible for DTE in "inherited retarded eruption."[12] Delayed development of isolated teeth has also been reported. This is most commonly seen in the premolar region. Profitt and Vig[13] hypothesized that a "gradient of eruption" might exist distally along the dental lamina. This could explain the frequency of DTE in posterior teeth. Some patients who have delayed eruption of the second molars alone might fall into the category of mild eruption failure syndrome.[13]

CLINICAL IMPLICATIONS

Accurate diagnosis of DTE is an important but complicated process. When teeth do not erupt at the expected age (mean ± 2 SD), a careful evaluation should be performed to establish the etiology and the treatment plan accordingly. The importance of the patient's medical history cannot be overstated. A wide variety of disorders has been reported in the literature to be associated with DTE (Tables II and III). Family information and information from affected patients about unusual variations in eruption patterns should be investigated. Clinical examination should be done methodically and must begin with the overall physical evaluation of the patient. Although the presence of syndromes is usually obvious, in the mild forms, only a careful examination will reveal the abnormalities. Right-left variations in eruption timings are minimal in most patients, but significant deviations might be associated with (for example) tumors or hemifacial microsomia or macrosomia and should alert the clinician to perform further investigation.

Intraoral examination should include inspection, palpation, percussion, and radiographic examination. The clinician should inspect for gross soft tissue pathology, scars, swellings, and fibrous or dense frenal attachments. Careful observation and palpation of the alveolar ridges buccally and lingually usually shows the characteristic bulge of a tooth in the process of eruption. Palpation producing pain, crackling, or other symptoms should be further evaluated for pathology. In patients in whom a deciduous tooth is overretained, with respect to either the contralateral side or the mean exfoliation age for the patient's sex and ethnicity, the deciduous tooth and the supporting structures should be thoroughly examined.[53] Ankylosed teeth also interfere with the vertical development of the alveolus.[8,9,44] Retention of the deciduous tooth might lead to deflection of the succedaneous tooth and resorptive damage of the adjacent teeth.[84]

Schour and Massler,[1] Nolla,[2] Moorrees et al,[3,85] and Koyoumdjisky-Kaye et al[86] have developed tables and diagrammatic charts of the stages of tooth development, starting from the initiation of the calcification process to the completion of the root apex of each tooth. Norms with the average chronologic ages at which each stage occurs are also provided. Root development, with few exceptions, proceeds in a fairly constant manner.[10,11]

DTE is often seen in the region of the maxillary canines.[87-90] The maxillary canine develops high in the maxilla and is the only tooth that must descend more than its length to reach its position in the dental arch. When pathologic conditions are ruled out, the etiology of DTE of the canines has been suggested to be multifactorial.[9,91,92] Specifically, 3 factors have been proposed for consideration: (1) DTE of the canine might simply reflect ectopic development of the tooth germ that could be genetically determined, (2) there might be a familial association to them, and (3) in a significant number of cases of delayed eruption of the canine, an abnormality of the lateral incisor in the same quadrant is observed.[90,93] According to Becker et al,[94,95] abnormalities of the lateral incisor occur so frequently in cases of delayed canine eruption that the association is not likely to be due to chance.[94,95] A developmental anomaly might exist in this part of the maxilla, which contains one of the embryonic fusion lines, and DTE of the canines in many cases could be part of a hereditary syndrome.[90]

Permanent tooth agenesis (excluding the third molars) in the general population has been noted to range from 1.6% to 9.6%. The incidence of tooth agenesis in the deciduous dentition is in the range of 0.5% to 0.9%.[96] After third molars, the most commonly missing teeth are mandibular second premolars and maxillary lateral incisors, in that order.[16] Thus, congenital absence of a tooth should also be suspected when considering DTE.

A panoramic radiograph is ideal for evaluating the position of teeth and the extent of tooth development, estimating the time of emergence of the tooth into the oral cavity, and screening for pathology. The parallax method (image/tube shift method, Clark's rule, buccal object rule) and 2 radiographs taken at right angles to each other[97,98] are suggested for radiographic localization of tumors, supernumerary teeth, and displaced teeth, which require surgical correction. Computed tomography can be used as the most precise method of radiographic localization, although its additional cost and relatively high radiation dose limit its use.[97,99]

DTE can also have psychological implications for the patient, especially if anterior teeth are affected. The duration of orthodontic treatment might be prolonged while the orthodontist and the patient wait for tooth eruption. In such situations, adequate space should be maintained and a reevaluation for possible systemic influences should be performed. When there are coexisting systemic conditions, factors such as bone quality, bone density, and skeletal maturation also should be considered.

THERAPEUTIC CONSIDERATIONS FOR THE PATIENT WITH DTE

DTE presents a challenge for orthodontic treatment planning. A number of techniques have been suggested for treating DTE. The main considerations for teeth affected by DTE are (1) the decision to remove or retain the tooth or teeth affected by DTE, (2) the use of surgery to remove obstructions, (3) surgical exposure of teeth affected by DTE, (4) the application of orthodontic traction, (5) the need for space creation and maintenance, and (6) diagnosis and treatment of systemic disease that causes DTE.

The treatment flowchart (Fig 2) can serve as a guideline for addressing the most important treatment options in DTE. Once the clinical determination of chronologic DTE (>2 SD) has been established, a panoramic radiograph should be obtained. The screening radiograph can be used to assess the developmental state of the tooth and rule out tooth agenesis.

DTE with defective tooth development

If there is defective tooth formation, the first step should be to assess whether the defect is localized or generalized.

In the deciduous dentition, close observation of the defective deciduous tooth or teeth is the usual course of treatment, and space should be maintained where indicated. Unerupted deciduous teeth with serious defects should be extracted, but the time of extraction should be defined carefully by considering the development of the succedaneous teeth and the space relationships in the permanent dentition. Information in the literature is sparse on this topic, either because the condition is underreported or because defective tooth development is primarily diagnosed in the permanent dentition.

In the permanent dentition, unerupted teeth are normally closely observed until the skeletal growth period necessary for appropriate development and preservation of the surrounding alveolar ridge has been attained. Management has traditionally focused on the restorative challenges of these patients once the teeth have erupted.[16] No systematic approach to accelerate the eruption of malformed retained teeth could be found in the literature. However, Andreasen[9] suggests that in patients in whom the defect is not in the supporting apparatus of the tooth, exposure of the affected teeth might bring about the eruption. Severely malformed teeth usually must be extracted.[16] Often, defective teeth can serve as abutments for restorative care once they have erupted.[16,100]

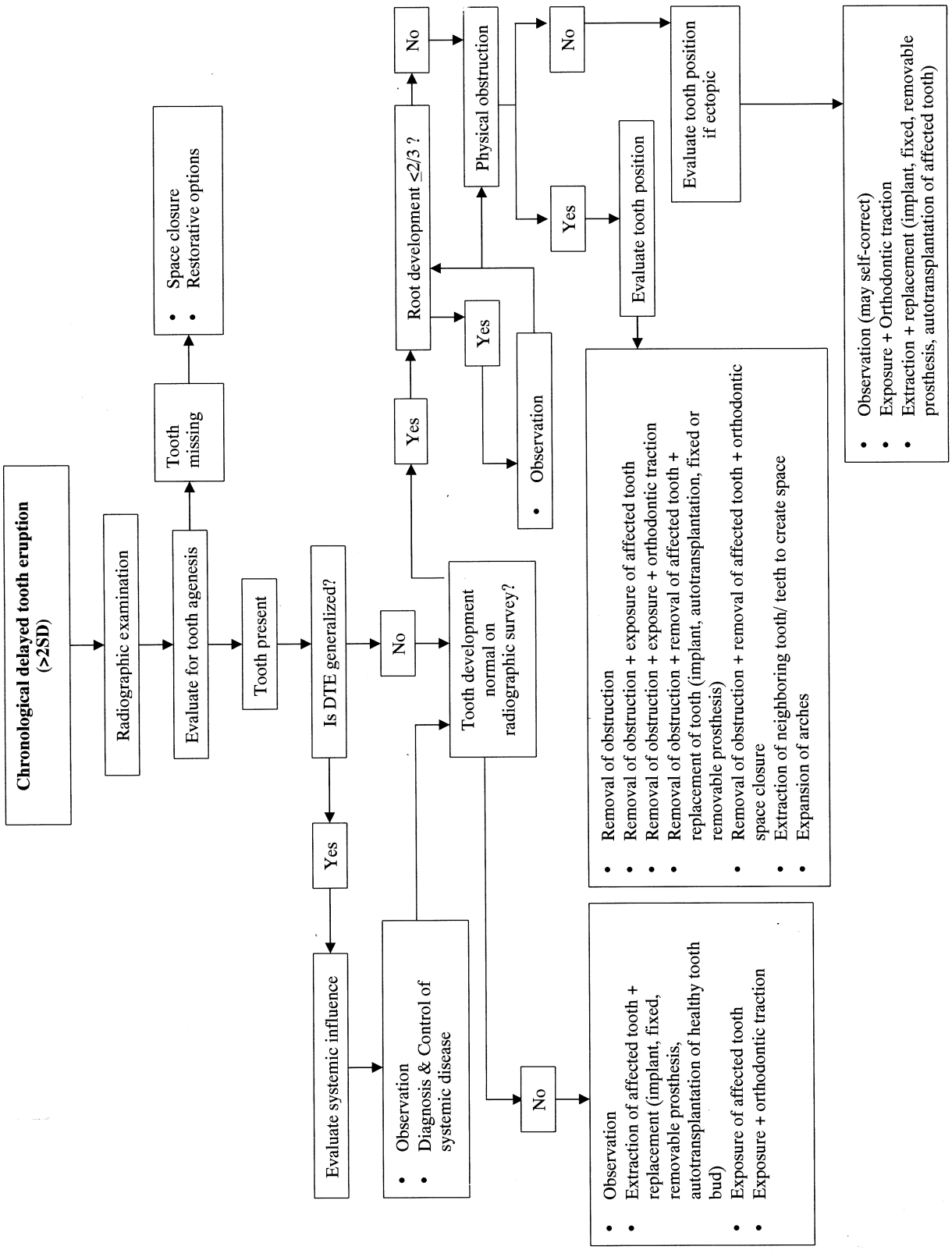

Fig 2. Flow chart of treatment options for DTE affecting permanent dentition.

DTE with no obvious developmental defect in the affected tooth or teeth on the radiograph

In this case, root development (biologic eruption status), tooth position, and physical obstruction (radiographically evident or not) should be evaluated.

In the absence of ectopic tooth position and physical obstruction, and if the biologic eruption status is within normal limits, periodic observation is the recommended course of action. For a succedaneous tooth if root formation is inadequate, extraction of the deciduous tooth or exposure to apply active orthodontic treatment is not justified.[10,11] Root development should be followed by periodic radiographic examination. If the tooth is lagging in its eruption status, active treatment is recommended when more than 2/3 of the root has developed.

Radiographic examination might also show an ectopic position of the developing tooth. Often, some deviations self-correct,[101] but significant migration of the tooth usually requires extraction.[102] If self-correction is not observed over time, active treatment should begin. Exposure accompanied by orthodontic traction has been shown to be successful. In patients in whom the ectopic teeth deviate more than 90° from the normal eruptive path, autotransplantation might be an effective alternative.[9]

An obstruction causing delayed eruption might or might not be obvious on the radiographic survey. A soft tissue barrier to eruption is not discernible on the radiograph, but, regardless of etiology, an obstruction should be treated with an uncovering procedure that includes enamel exposure.[9,30,38] Supernumerary teeth, tumors, cysts, and bony sequestra are examples of physical obstructions visible on the radiographic survey. Their removal usually will permit the affected tooth to erupt.

In the deciduous dentition, DTE due to obstruction is uncommon, but scar tissue (due to trauma) and pericoronal odontogenic cysts or neoplasms are the usual culprits in cases of obstruction. Trauma is more common in the anterior region, but cysts or neoplasms are more likely to result in DTE in the canine and molar regions.[23] Odontomas are reported to be the most common of the odontogenic lesions associated with DTE.[103,104] Treatment options for deciduous DTE range from observation, removal of physical obstruction with and without exposure of the affected tooth, orthodontic traction on rare occasions, and extraction of the involved tooth.[23,103,105]

In the permanent dentition, removal of the physical obstruction from the path of eruption is recommended. When neoplasms (odontogenic or nonodontogenic) cause obstruction, the surgical approach is dictated by the biologic behavior of the lesion. If the affected tooth is deep in the bone, the follicle around it should be left intact. When the affected tooth is in a superficial position, exposure of the enamel is done at tumor removal.[9,16,38] Occasionally, the affected tooth must be removed. Four surgical approaches have been recommended for uncovering impacted teeth.[106-109] These include gingivectomy, apically positioned flap, flap/closed eruption, and the preorthodontic uncovering technique.[106] Two opinions seems to exist regarding management of the tooth delayed in eruption after removing the physical barrier. McDonald and Avery[110] recommend exposure of the tooth delayed in eruption at the surgical removal of the barrier, but Houston and Tulley[111] advocate removing the obstruction and providing sufficient space for the unerupted tooth to erupt spontaneously. Most teeth (54%-75%) erupt spontaneously in the latter situation; however, the eruption rate might be protracted.[112] DiBiase[112] reported that if the tooth is not displaced and its follicle not disturbed during the surgical procedure, the tooth might take an average of 18 months to erupt. DiBiase[112] also stated that sufficient space should be made available for the tooth's eruption. If the tooth is exposed at the time of surgery, it might or might not be subjected to orthodontic traction to accelerate and guide its eruption into the arch.[9,106] The decision to use orthodontic traction in most case reports seems to be a judgment call for the clinician. No conclusive guidelines could be derived from the literature regarding when active force should be used to aid eruption of the exposed tooth. Occasionally, a deciduous tooth can be a physical barrier to the eruption of the succedaneous tooth.[34,38,47] In most cases, removing the deciduous tooth will allow for spontaneous eruption of the successor. When arch-length deficiency creates a physical obstruction, either expansion of the dental arches or extraction might be necessary to obtain the required space. Extraction of either the affected or adjacent teeth can be performed.[9]

Occasionally, several teeth in a quadrant might be unerupted, and this can present an orthodontic challenge because of the lack of adequate anchorage elements. Osseointegrated implants might offer viable alternatives for anchorage in such cases.[113]

DTE associated with systemic disorders

Whenever DTE is generalized, the patient should be examined for systemic diseases affecting eruption, such as endocrine disorders, organ failures, metabolic disorders, drugs, and inherited and genetic disorders. Various methods have been suggested for treating eruption disorders in these conditions. These include no treat-

ment (observation), elimination of obstacles to eruption (eg, cysts, soft tissue overgrowths), exposure of affected teeth with and without orthodontic traction, autotransplantation, and control of the systemic disease.[7,9,12,16,59,61,83,114,115]

CONCLUSIONS

Variation in the normal eruption of teeth is a common finding, but significant deviations from established norms should alert the clinician to further investigate the patient's health and development. Delayed tooth eruption might be a harbinger of a systemic condition or an indication of altered physiology of the craniofacial complex. Orthodontists are often in a sentry position to perform an early evaluation of craniofacial structures, both clinically and radiographically. Proper evaluation of DTE in orthodontic diagnosis and treatment requires a clear definition of the term and its significance. We propose a diagnostic "tree" that would enable the clinician to perform an accurate and thorough orthodontic diagnosis of the patient with DTE.

REFERENCES

1. Schour I, Massler M. The development of the human dentition. J Am Dent Assoc 1941;28:1153-60.
2. Nolla CM. The development of the human dentition. ASDC J Dent Child 1960;27:254-66.
3. Moorrees CFA, Fanning EA, Hunt EEJ. Age variation of formation stages for ten permanent teeth. J Dent Res 1963;42: 1490-502.
4. Manouvrier-Hanu S, Devisme L, Vaast P, Boute-Benejean O, Farriaux JP. Fryns syndrome and erupted teeth in a 24-weeks-old fetus. Genet Couns 1996;7:131-4.
5. Cunha RF, Boer FA, Torriani DD, Frossard WT. Natal and neonatal teeth: review of the literature. Pediatr Dent 2001;23: 158-62.
6. Krogman WM. Biological timing and the dento-facial complex. 3. J Dent Child 1968;35:328-41.
7. Pulse CL, Moses MS, Greenman D, Rosenberg SN, Zegarelli DJ. Cherubism: case reports and literature review. Dent Today 2001;20:100-3.
8. Raghoebar GM, Boering G, Vissink A, Stegenga B. Eruption disturbances of permanent molars: a review. J Oral Pathol Med 1991;20:159-66.
9. Andreasen JO, Petersen JK, Laskin DM. Textbook and color atlas of tooth impactions. St. Louis: Mosby; 1997.
10. Gron AM. Prediction of tooth emergence. J Dent Res 1962;41: 573-85.
11. Becker A. The orthodontic treatment of impacted teeth. London: Martin Dunitz; 1998.
12. Rasmussen P, Kotsaki A. Inherited retarded eruption in the permanent dentition. J Clin Pediatr Dent 1997;21:205-11.
13. Proffit WR, Vig KW. Primary failure of eruption: a possible cause of posterior open-bite. Am J Orthod 1981;80:173-90.
14. O'Connell AC, Torske KR. Primary failure of tooth eruption: a unique case. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87:714-20.
15. Pytlik W. Primary failure of eruption: a case report. Int Dent J 1991;41:274-8.
16. Neville BW, Damm DD, Allen CM. Oral and maxillofacial pathology. Philadelphia: W.B. Saunders; 2002.
17. Shafer WG, Hine MK, Levy BM. Textbook of oral pathology. Philadelphia: W.B. Saunders; 1983.
18. Spieker RD. Submerged permanent teeth: literature review and case report. Gen Dent 2001;49:64-68.
19. Antoniades K, Kavadia S, Milioti K, Antoniades V, Markovitsi E. Submerged teeth. J Clin Pediatr Dent 2002;26:239-42.
20. Moorrees CF, Gron AM, Lebret LM, Yen PK, Frohlich FJ. Growth studies of the dentition: a review. Am J Orthod 1969;55:600-16.
21. Tay F, Pang A, Yuen S. Unerupted maxillary anterior supernumerary teeth: report of 204 cases. ASDC J Dent Child 1984; 51:289-94.
22. Foster TD, Taylor GS. Characteristics of supernumerary teeth in the upper central incisor region. Dent Pract Dent Rec 1969;20:8-12.
23. Flaitz CM, Hicks J. Delayed tooth eruption associated with an ameloblastic fibro-odontoma. Pediatr Dent 2001;23:253-4.
24. Damm DD, Fantasia JE. Failure of eruption. Adenomatoid odontogenic tumor. Gen Dent 2000;48:650, 722.
25. Yassin OM. Delayed eruption of maxillary primary cuspid associated with compound odontoma. J Clin Pediatr Dent 1999;23:147-9.
26. Veis A, Tziafas D, Lambrianidis T. A case report of a compound odontoma causing delayed eruption of a central maxillary incisor: clinical and microscopic evaluation. J Endod 2000;26:477-9.
27. Rad AS, Reid J. Delayed eruption of a permanent molar associated with a complex odontoma: report of case. ASDC J Dent Child 1996;63:299-301.
28. Tomizawa M, Yonemochi H, Kohno M, Noda T. Unilateral delayed eruption of maxillary permanent first molars: four case reports. Pediatr Dent 1998;20:53-6.
29. Damm DD, Fantasia JE. Focal delayed eruption. Regional odontodysplasia. Gen Dent 2001;49:356, 428.
30. Di Biase DD. Mucous membrane and delayed eruption. Dent Pract Dent Rec 1971;21:241-50.
31. Di Biase DD. Mucous membrane and delayed eruption. Trans Br Soc Study Orthod 1969;5:149-58.
32. Goho C. Delayed eruption due to overlying fibrous connective tissue. ASDC J Dent Child 1987;54:359-60.
33. Katz J, Guelmann M, Barak S. Hereditary gingival fibromatosis with distinct dental, skeletal and developmental abnormalities. Pediatr Dent 2002;24:253-6.
34. Smith RJ, Rapp R. A cephalometric study of the developmental relationship between primary and permanent maxillary central incisor teeth. ASDC J Dent Child 1980;47:36-41.
35. Brin I, Ben-Bassat Y, Zilberman Y, Fuks A. Effect of trauma to the primary incisors on the alignment of their permanent successors in Israelis. Community Dent Oral Epidemiol 1988; 16:104-8.
36. Hawes RR. Traumatized primary teeth. Dent Clin North Am 1966:391-404.
37. Andreasen JO, Sundstrom B, Ravn JJ. The effect of traumatic injuries to primary teeth on their permanent successors. I. A clinical and histologic study of 117 injured permanent teeth. Scand J Dent Res 1971;79:219-83.
38. Andreasen JO, Andreasen FM. Textbook and color atlas of traumatic injuries to the teeth. Copenhagen: Munksgaard; 1994.

39. Yawaka Y, Kaga M, Osanai M, Fukui A, Oguchi H. Delayed eruption of premolars with periodontitis of primary predecessors and a cystic lesion: a case report. Int J Paediatr Dent 2002;12:53-60.
40. Wilson CF. Management of trauma to primary and developing teeth. Dent Clin North Am 1995;39:133-67.
41. Ravn JJ. Sequelae of acute mechanical traumata in the primary dentition. A clinical study. J Dent Child 1968;35:281-9.
42. Korf SR. The eruption of permanent central incisors following premature loss of their antecedents. ASDC J Dent Child 1965;32:39-44.
43. Raghoebar GM, Boering G. An unerupted deciduous molar. Oral Surg Oral Med Oral Pathol 1991;71:521-2.
44. Biederman W. The problem of the ankylosed tooth. Dent Clin North Am 1968:409-24.
45. Brearley LJ, McKibben DH Jr. Ankylosis of primary molar teeth. I. Prevalence and characteristics. J Dent Child 1973;40: 54-63.
46. Raghoebar GM, Boering G, Vissink A. Clinical, radiographic and histological characteristics of secondary retention of permanent molars. J Dent 1991;19:164-70.
47. Kurol J. Early treatment of tooth-eruption disturbances. Am J Orthod Dentofacial Orthop 2002;121:588-91.
48. Suda N, Hiyama S, Kuroda T. Relationship between formation/eruption of maxillary teeth and skeletal pattern of maxilla. Am J Orthod Dentofacial Orthop 2002;121:46-52.
49. Piloni MJ, Ubios AM. Impairment of molar tooth eruption caused by x-radiation. Acta Odontol Latinoam 1996;9:87-92.
50. Gowgiel JM. Eruption of irradiation produced rootless teeth in monkeys. J Dent Res 1961;40:540.
51. Alvarez JO. Nutrition, tooth development, and dental caries. Am J Clin Nutr 1995;61:410S-6S.
52. Garn SM, Rohmann CG. Interaction of nutrition and genetics in the timing of growth. Pediatr Clin North Am 1966;13:353-79.
53. Moyers RE. Handbook of orthodontics. Chicago: Year Book Medical Publishers; 1988.
54. Shaw L, Foster TD. Size and development of the dentition in endocrine deficiency. J Pedod 1989;13:155-60.
55. Loevy HT, Aduss H, Rosenthal IM. Tooth eruption and craniofacial development in congenital hypothyroidism: report of case. J Am Dent Assoc 1987;115:429-31.
56. Hinrichs EH Jr. Dental changes in juvenile hypothyroidism. ASDC J Dent Child 1966;33:167-73.
57. Kosowicz J, Rzymski K. Abnormalities of tooth development in pituitary dwarfism. Oral Surg Oral Med Oral Pathol 1977;44: 853-63.
58. Kjellberg H, Beiring M, Albertsson Wikland K. Craniofacial morphology, dental occlusion, tooth eruption, and dental maturity in boys of short stature with or without growth hormone deficiency. Eur J Oral Sci 2000;108:359-67.
59. Seow WK. Effects of preterm birth on oral growth and development. Aust Dent J 1997;42:85-91.
60. Seow WK. A study of the development of the permanent dentition in very low birthweight children. Pediatr Dent 1996; 18:379-84.
61. Hauk MJ, Moss ME, Weinberg GA, Berkowitz RJ. Delayed tooth eruption: association with severity of HIV infection. Pediatr Dent 2001;23:260-2.
62. Ramos-Gomez FJ, Petru A, Hilton JF, Canchola AJ, Wara D, Greenspan JS. Oral manifestations and dental status in paediatric HIV infection. Int J Paediatr Dent 2000;10:3-11.
63. Pope JE, Curzon ME. The dental status of cerebral palsied children. Pediatr Dent 1991;13:156-62.
64. Giglio MJ, Sanz AM, Costanzo A, Bozzini CE. Impeded eruption rate of the rat maxillary incisor during exposure to different simulated altitudes. J Dent Res 1987;66:1490-2.
65. Giglio MJ, Sanz AM, Bozzini CE. Depressed eruption rate of the rat maxillary incisor in a drug-induced uncompensated hemolytic state model. J Dent Res 1990;69:906-8.
66. Giglio MJ, Frid A, Barcat JA, Arrizurieta E. Depressed eruption dental rate in rats with hemodynamically mediated acute renal failure. Acta Odontol Latinoam 1994;8:27-37.
67. Kaloust S, Ishii K, Vargervik K. Dental development in Apert syndrome. Cleft Palate Craniofac J 1997;34:117-21.
68. Kreiborg S, Cohen MM Jr. The oral manifestations of Apert syndrome. J Craniofac Genet Dev Biol 1992;12:41-8.
69. Gorlin RJ, Cohen MMJ, Hennekam RCM. Syndromes of the head and neck. New York: Oxford University Press; 2001.
70. Jensen BL, Kreiborg S. Development of the dentition in cleidocranial dysplasia. J Oral Pathol Med 1990;19:89-93.
71. Buch B, Noffke C, de Kock S. Gardner's syndrome—the importance of early diagnosis: a case report and a review. J S Afr Dent Assoc 2001;56:242-5.
72. Yamamoto H, Sakae T, Davies JE. Cleidocranial dysplasia: a light microscope, electron microscope, and crystallographic study. Oral Surg Oral Med Oral Pathol 1989;68:195-200.
73. Fukuta Y, Totsuka M, Takeda Y, Yoshida Y, Niitsu J, Yamamoto H. Histological and analytical studies of a tooth in a patient with cleidocranial dysostosis. J Oral Sci 2001;43:85-9.
74. Takeda Y. Multiple cemental lesions in the jaw bones of a patient with Gardner's syndrome. Virchows Archiv A Pathol Anat Histopathol 1987;411:253-6.
75. Shroff B, Kashner JE, Keyser JD, Hebert C, Norris K. Epidermal growth factor and epidermal growth factor-receptor expression in the mouse dental follicle during tooth eruption. Arch Oral Biol 1996;41:613-7.
76. Marks SC Jr. The basic and applied biology of tooth eruption. Connect Tissue Res 1995;32:149-57.
77. Shroff B, Siegel SM. Molecular basis for tooth eruption and its clinical implications in orthodontic tooth movement. Semin Orthod 2000;6:155-72.
78. Ida-Yonemochi H, Noda T, Shimokawa H, Saku T. Disturbed tooth eruption in osteopetrotic (op/op) mice: histopathogenesis of tooth malformation and odontomas. J Oral Pathol Med 2002;31:361-73.
79. Droz-Desprez D, Azou C, Bordigoni P, Bonnaure-Mallet M. Infantile osteopetrosis: a case report on dental findings. J Oral Pathol Med 1992;21:422-5.
80. Stephen LX, Hamersma H, Gardner J, Beighton P. Dental and oral manifestations of sclerosteosis. Int Dent J 2001;51:287-90.
81. Blankenstein R, Brook AH, Smith RN, Patrick D, Russell JM. Oral findings in Carpenter syndrome. Int J Paediatr Dent 2001;11:352-60.
82. Hitchin AD. Cementum and other root abnormalities of permanent teeth in cleidocranial dysostosis. Br Dent J 1975;139: 313-8.
83. O'Connell AC, Puck JM, Grimbacher B, Facchetti F, Majorana A, Gallin JI, et al. Delayed eruption of permanent teeth in hyperimmunoglobulinemia E recurrent infection syndrome. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89: 177-85.
84. Ericson S, Kurol J. Resorption of maxillary lateral incisors caused by ectopic eruption of the canines. A clinical and radiographic analysis of predisposing factors. Am J Orthod Dentofacial Orthop 1988;94:503-13.

85. Moorrees CFA, Fanning EA, Gron AM, Lebret L. The timing of orthodontic treatment in relation to tooth formation. Trans Eur Orthod Soc 1962;38:1-14.
86. Koyoumdjisky-Kaye E, Baras M, Grover NB. Stages in the emergence of the dentition: an improved classification and its application to Israeli children. Growth 1977;41:285-96.
87. Kettle MA. Treatment of the unerupted maxillary canine. Trans Br Soc Orthod 1957:74-87.
88. Rozylo TK, Jurkiewicz-Mazurek M, Rozylo-Kalinowska I, Drelich A. Early detection of potential impaction of permanent maxillary canines caused by resorption disorders of deciduous canines. Ann Univ Mariae Curie Sklodowska [Med] 2001;56: 119-24.
89. Ericson S, Kurol J. Longitudinal study and analysis of clinical supervision of maxillary canine eruption. Community Dent Oral Epidemiol 1986;14:172-6.
90. Richardson A, McKay C. Delayed eruption of maxillary canine teeth. Part I. Aetiology and diagnosis. Proc Br Paedod Soc 1982;12:15-25.
91. Ericson S, Kurol J. Radiographic assessment of maxillary canine eruption in children with clinical signs of eruption disturbance. Eur J Orthod 1986;8:133-40.
92. Becker A, Chaushu S. Dental age in maxillary canine ectopia. Am J Orthod Dentofacial Orthop 2000;117:657-62.
93. Jacobs SG. Palatally impacted canines: aetiology of impaction and the scope for interception. Report of cases outside the guidelines for interception. Aust Dent J 1994;39:206-11.
94. Becker A, Smith P, Behar R. The incidence of anomalous maxillary lateral incisors in relation to palatally displaced cuspids. Angle Orthod 1981;51:24-9.
95. Becker A, Sharabi S, Chaushu S. Maxillary tooth size variation in dentitions with palatal canine displacement. Eur J Orthod 2002;24:313-8.
96. Vastardis H. The genetics of human tooth agenesis: new discoveries for understanding dental anomalies. Am J Orthod Dentofacial Orthop 2000;117:650-6.
97. Jacobs SG. Radiographic localization of unerupted teeth: further findings about the vertical tube shift method and other localization techniques. Am J Orthod Dentofacial Orthop 2000; 118:439-47.
98. Southall PJ, Gravely JF. Radiographic localization of unerupted teeth in the anterior part of the maxilla: a survey of methods currently employed. Br J Orthod 1987;14:235-42.
99. Preda L, La Fianza A, Di Maggio EM, Dore R, Schifino MR, Campani R, et al. The use of spiral computed tomography in the localization of impacted maxillary canines. Dentomaxillofac Radiol 1997;26:236-41.
100. Wilson PH, Ali A. Case report: restorative options in regional odontodysplasia. Eur J Prosthodont Restor Dent 2002;10:5-8.
101. Wagner M, Katsaros C, Goldstein T. Spontaneous uprighting of permanent tooth germs after elimination of local eruption obstacles. J Orofac Orthop 1999;60:279-85.
102. Rebellato J, Schabel B. Treatment of a patient with an impacted transmigrant mandibular canine and a palatally impacted maxillary canine. Angle Orthod 2003;73:328-36.
103. Otsuka Y, Mitomi T, Tomizawa M, Noda T. A review of clinical features in 13 cases of impacted primary teeth. Int J Paediatr Dent 2001;11:57-63.
104. Morning P. Impacted teeth in relation to odontomas. Int J Oral Surg 1980;9:81-91.
105. Jarvinen SH. Unerupted second primary molars: report of two cases. ASDC J Dent Child 1994;61:397-400.
106. Kokich VG, Mathews DP. Impacted teeth: orthodontic and surgical considerations. In: McNamara JA, editor. Orthodontics and dentofacial orthopedics. Ann Arbor (Mich): Needham Press; 2001.
107. Vanarsdall RL, Corn H. Soft-tissue management of labially positioned unerupted teeth. Am J Orthod 1977;72:53-64.
108. Vermette ME, Kokich VG, Kennedy DB. Uncovering labially impacted teeth: apically positioned flap and closed-eruption techniques. Angle Orthod 1995;65:23-33.
109. Kokich VG, Mathews DP. Surgical and orthodontic management of impacted teeth. Dent Clin North Am 1993;37:181-204.
110. McDonald RE, Avery DR. Dentistry for the child and adolescent. St. Louis: Mosby; 1999.
111. Houston WJB, Tulley WJ. A textbook of orthodontics. Bristol, United Kingdom: Wright; 1992.
112. Di Biase DD. The effects of variations in tooth morphology and position on eruption. Dent Pract Dent Rec 1971;22:95-108.
113. Janssens F, Swennen G, Dujardin T, Glineur R, Malevez C. Use of an onplant as orthodontic anchorage. Am J Orthod Dentofacial Orthop 2002;122:566-70.
114. Malmgren B, Norgren S. Dental aberrations in children and adolescents with osteogenesis imperfecta. Acta Odontol Scand 2002;60:65-71.
115. Sauk JJ. Genetic disorders involving tooth eruption anomalies. In: Davidovitch Z, editor. The biological mechanisms of tooth eruption and root resorption. Birmingham, Ala: Ebsco Media; 1988. p. 171-9.
116. Mitchell L, Bennett TG. Supernumerary teeth causing delayed eruption—a retrospective study. Br J Orthod 1992;19:41-6.
117. Gallas MM, Garcia A. Retention of permanent incisors by mesiodens: a family affair. Br Dent J 2000;188:63-4.
118. Gregg TA, Kinirons MJ. The effect of the position and orientation of unerupted premaxillary supernumerary teeth on eruption and displacement of permanent incisors. Int J Paediatr Dent 1991;1:3-7.
119. Sekletov GA. [Supercomplect retained tooth is the cause of delayed eruption of the upper central left incisor. Therapy]. Stomatologiia (Sofiia) 2001;80:66-8.
120. Yeung KH, Cheung RC, Tsang MM. Compound odontoma associated with an unerupted and dilacerated maxillary primary central incisor in a young patient. Int J Paediatr Dent 2003;13: 208-12.
121. Acquavella FJ. Delayed eruption. Why? N Y State Dent J 1965;31:448-9.
122. Diab M, elBadrawy HE. Intrusion injuries of primary incisors. Part III: Effects on the permanent successors. Quintessence Int 2000;31:377-84.
123. Adams TW. An impacted primary lateral incisor as a cause of delayed eruption of a permanent tooth: case report. Pediatr Dent 1998;20:121-3.
124. Ubios AM, Piloni MJ, Cabrini RL. Mandibular growth and tooth eruption after localized x-radiation. J Oral Maxillofac Surg 1992;50:153-6.
125. Peterka M, Tvrdek M, Mullerova Z. Tooth eruption in patients with cleft lip and palate. Acta Chir Plast 1993;35:154-8.
126. Palubis JE. Cleft lip and palate with delayed eruption and congenital absence of teeth. Birth Defects Orig Artic Ser 1971;7:265-6.
127. Becktor KB, Reibel J, Vedel B, Kjaer I. Segmental odontomaxillary dysplasia: clinical, radiological and histological aspects of four cases. Oral Dis 2002;8:106-10.
128. Agarwal KN, Narula S, Faridi MM, Kalra N. Deciduous dentition and enamel defects. Indian Pediatr 2003;40:124-9.

129. Trevathan TH. Delayed eruption of teeth in pseudohypoparathyroidism. N Z Dent J 1961;57:20-3.
130. Minicucci EM, Lopes LF, Crocci AJ. Dental abnormalities in children after chemotherapy treatment for acute lymphoid leukemia. Leuk Res 2003;27:45-50.
131. Oncag O, Ozkinay FF, Eronat C. Dysosteosclerosis: a case with unique dental findings and SEM evaluation of a hypoplastic tooth. J Clin Pediatr Dent 1999;23:347-52.
132. Appleton RE, Leach H. Delayed eruption of secondary dentition associated with phenytoin therapy. Dev Med Child Neurol 1991;33:1117-8.
133. Church LF Jr, Brandt SK. Phenytoin-induced gingival overgrowth resulting in delayed eruption of the primary dentition. A case report. J Periodontol 1984;55:19-21.
134. Prati C, Santopadre A, Baroni C. [Delayed eruption, enamel hypoplasia and caries in childhood celiac disease]. Minerva Stomatol 1987;36:749-52.
135. Gerlach RF, Toledo DB, Novaes PD, Merzel J, Line SR. The effect of lead on the eruption rates of incisor teeth in rats. Arch Oral Biol 2000;45:951-5.
136. Rasmussen P, Kotsaki A. Inherited primary failure of eruption in the primary dentition: report of five cases. ASDC J Dent Child 1997;64:43-7.
137. Shokeir MH. Complete failure of eruption of all permanent teeth: an autosomal dominant disorder. Clin Genet 1974;5: 322-6.
138. Pytlik W, Alfter G. Impairment of tooth eruption. Pathogenetic aspects. J Orofac Orthop 1996;57:238-45.
139. Kieser JA, Groeneveld HT, da Silva P. Delayed tooth formation in children exposed to tobacco smoke. J Clin Pediatr Dent 1996;20:97-100.
140. Kaban LB, Needleman HL, Hertzberg J. Idiopathic failure of eruption of permanent molar teeth. Oral Surg Oral Med Oral Pathol 1976;42:155-63.
141. Collins MA, Mauriello SM, Tyndall DA, Wright JT. Dental anomalies associated with amelogenesis imperfecta: a radiographic assessment. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;88:358-64.
142. Smylski PT, Woodside DG, Harnett BE. Surgical and orthodontic treatment of cleidocranial dysostosis. Int J Oral Surg 1974;3:380-5.
143. Franklin DL, Roberts GJ. Delayed tooth eruption in congenital hypertrichosis lanuginosa. Pediatr Dent 1998;20:192-4.
144. Kalk WW, Batenburg RH, Vissink A. Dentin dysplasia type I: five cases within one family. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86:175-8.
145. Cawson RA. The oral changes in gargoylism. Proc R Soc Med 1962;55:1066-70.
146. Ondarza A, Jara L, Munoz P, Blanco R. Sequence of eruption of deciduous dentition in a Chilean sample with Down's syndrome. Arch Oral Biol 1997;42:401-6.
147. Knight S, Vulliamy T, Copplestone A, Gluckman E, Mason P, Dokal I. Dyskeratosis Congenita (DC) Registry: identification of new features of DC. Br J Haematol 1998;103:990-6.
148. Ritzau M, Carlsen O, Kreiborg S, Brinch-Iversen J, Gorlin RJ, Rasmussen NH. The Ekman-Westborg-Julin syndrome: report of case. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;84:293-6.
149. Kostara A, Roberts GJ, Gelbier M. Dental maturity in children with dystrophic epidermolysis bullosa. Pediatr Dent 2000;22: 385-8.
150. Carter LC, Fischman SL, Mann J, Elstein D, Stabholz A, Zimran A. The nature and extent of jaw involvement in Gaucher disease: observations in a series of 28 patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85:233-9.
151. Rosenblum SH. Delayed dental development in a patient with Gorlin syndrome: case report. Pediatr Dent 1998;20:355-8.
152. Hutchinson D. Oral manifestations of oculomandibulodyscephaly with hypotrichosis (Hallermann-Streiff syndrome). Oral Surg Oral Med Oral Pathol 1971;31:234-44.
153. Cipolloni C, Boldrini A, Donti E, Maiorana A, Coppa GV. Neonatal mucolipidosis II (I-cell disease): clinical, radiological and biochemical studies in a case. Helv Paediatr Acta 1980;35: 85-95.
154. Welbury TA, Welbury RR. Incontinentia pigmenti (Bloch-Sulzberger syndrome): report of case. ASDC J Dent Child 1999;66:213-5, 155.
155. Brownstein JN, Primosch RE. Oral manifestations of Menkes' kinky hair syndrome. J Clin Pediatr Dent 2001;25:317-21.
156. Shapiro SD, Abramovitch K, Van Dis ML, Skoczylas LJ, Langlais RP, Jorgenson RJ, et al. Neurofibromatosis: oral and radiographic manifestations. Oral Surg Oral Med Oral Pathol 1984;58:493-8.
157. D'Ambrosio JA, Langlais RP, Young RS. Jaw and skull changes in neurofibromatosis. Oral Surg Oral Med Oral Pathol 1988;66:391-6.
158. Farias M, Vargervik K. Dental development in hemifacial microsomia. I. Eruption and agenesis. Pediatr Dent 1988;10: 140-3.
159. Franklyn PP, Wilkinson D. Two cases of osteopathia striata, deafness and cranial osteopetrosis. Ann Radiol (Paris) 1978;21: 91-3.
160. O'Connell AC, Marini JC. Evaluation of oral problems in an osteogenesis imperfecta population. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87:189-96.
161. Chen RJ, Chen HS, Lin LM, Lin CC, Jorgenson RJ. "Otodental" dysplasia. Oral Surg Oral Med Oral Pathol 1988;66:353-8.
162. Foster TD. The effects of hemifacial atrophy on dental growth. Br Dent J 1979;146:148-50.
163. Singleton EB, Merten DF. An unusual syndrome of widened medullary cavities of the metacarpals and phalanges, aortic calcification and abnormal dentition. Pediatr Radiol 1973;1:2-7.
164. Bekisz O, Darimont F, Rompen EH. Diffuse but unilateral gingival enlargement associated with von Recklinghausen neurofibromatosis: a case report. J Clin Periodontol 2000;27:361-5.
165. Klingberg G, Oskarsdottir S, Johannesson EL, Noren JG. Oral manifestations in 22q11 deletion syndrome. Int J Paediatr Dent 2002;12:14-23.