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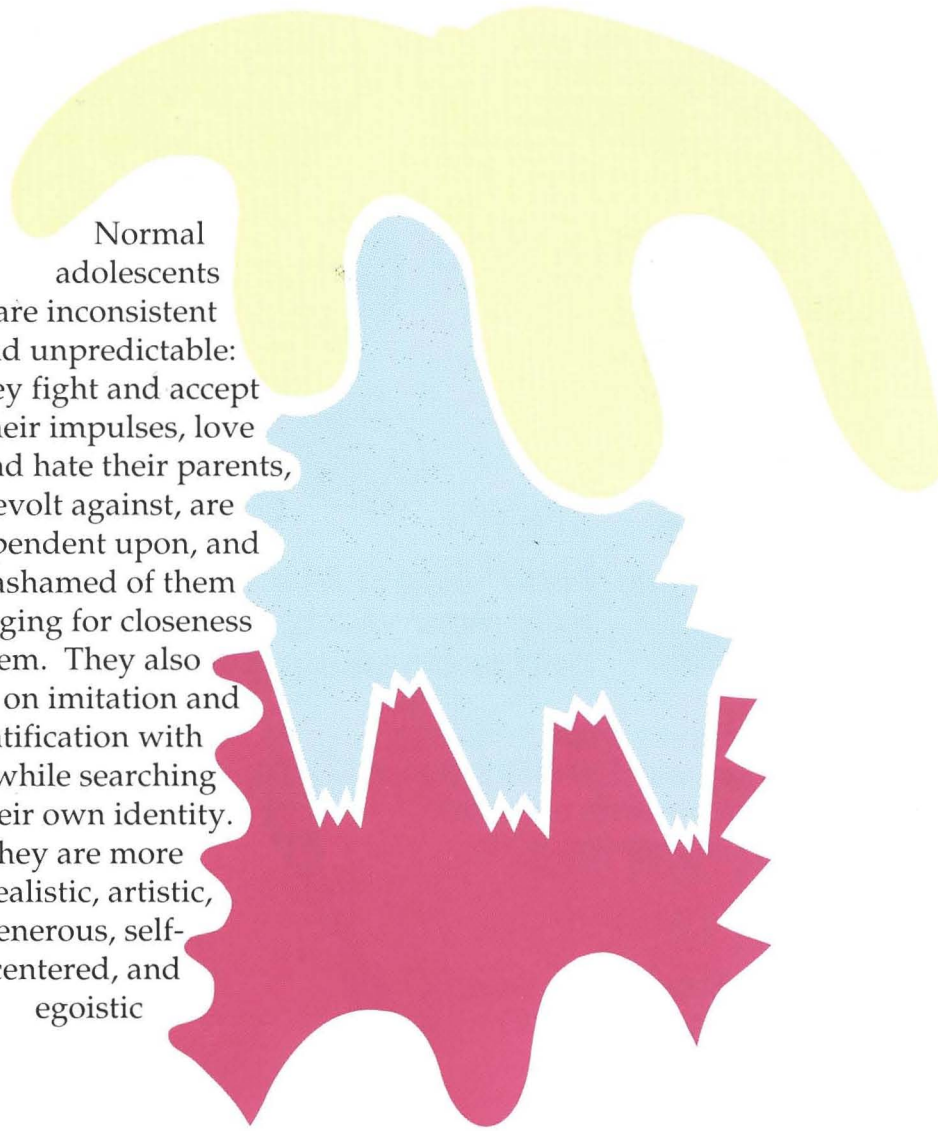
JOURNAL OF DENTISTRY FOR CHILDREN
NOVEMBER–DECEMBER 1991



Normal adolescents are inconsistent and unpredictable: they fight and accept their impulses, love and hate their parents, revolt against, are dependent upon, and are ashamed of them while longing for closeness with them. They also thrive on imitation and identification with others while searching for their own identity. They are more idealistic, artistic, generous, self-centered, and egoistic than at any other time of life.

—*Robert C. Prall, M.D.*

HE THAT WILL HAVE HIS SON HAVE A RESPECT FOR HIM AND HIS ORDERS, MUST HIMSELF HAVE A GREAT REVERENCE FOR HIS SON.

—*John Locke*

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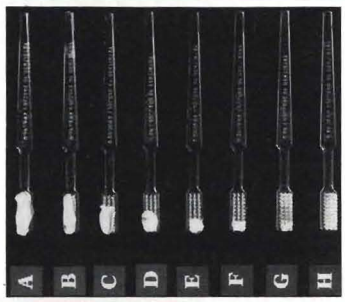
Adolescence is a time for searching for one's own identity.

Cover art and design by Sharlene Nowak-Stellmach.

REVIEW

BEHAVIOR

EPIDEMIOLOGY



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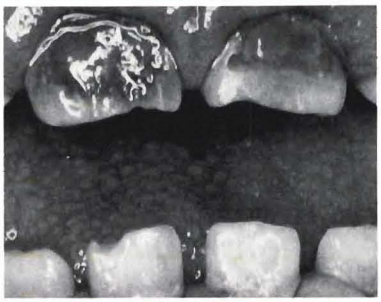

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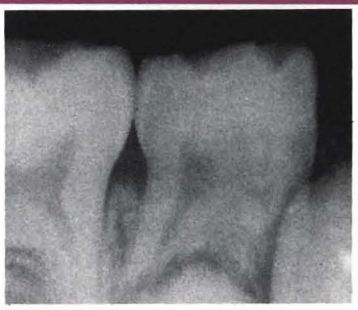

CLINIC

CASE REPORTS

For the busy reader

Enamel hypoplasia in the primary dentition: a review—page 441

This paper provides a critical review of the current concepts of the etiological factors involved in the pathogenesis of this significant clinical entity in the primary dentition. As in the permanent dentition, enamel hypoplasia in the primary dentition may be inherited as genetic diseases involving only the teeth, such as amelogenesis imperfecta, or as oral manifestations of many inherited systemic diseases and dysmorphic syndromes. More commonly, however, enamel hypoplasia in primary teeth results from acquired environmental factors encountered during the period of enamel formation.

Requests for reprints should be directed to Dr. W. Kim Seow, University of Queensland Dental School, Turbot St., Brisbane, Queensland, Australia 4000.

Dental treatment of fearful children, using nitrous oxide. Part I: Treatment times—page 453

Until now, little attention has been paid to the effect of the use of nitrous oxide on working time. The aim of this study was to investigate this effect when it is used as an additional aid to behavioral management in treating highly anxious child dental patients during sequential dental visits. Fifty-six patients, ages six- to eleven-years-old, were selected for this study; all had had previous, negative dental experience. This study shows that the use of nitrous oxide as an additional tool is more time-consuming than using only behavior management to allow the child to get acquainted with dental treatment. The influence on the rest of the treatment varies with the operator.

Requests for reprints should be directed to Dr. J.S.J. Veerkamp, ACTA, Louwesweg 1, 1066 EA Amsterdam, Holland.

An analysis of the phenomenon of increased parental participation during the child's dental experience—page 458

One aspect of behavioral dentistry for children that has captured the attention of the profession during the last decade has been the role of the parent during their child's dental experience. This paper interprets the changing interaction of clinician and child in light of concepts presented by noted anthropologist Margaret Mead and by social biologist Edward O. Wilson. Dr. Mead wrote of the metamorphosis in American parenting strategies since World War II as moving away from *postfigurative* or conventional ones, through *configurative* or modified, to very contemporary or *prefigurative*, with constant adjustments to the needs of the child. Dr. Wilson presented the concept of *social hypertrophy*: a process where latent social tendencies become more pronounced or emerge in civilization, in response to a perceived need — such as to protect one's child, even during the dental appointment. Some parents suppress the desire; others do not.

Requests for reprints should be directed to Dr. Jimmy R. Pinkham, University of Iowa, Department of Pediatric Dentistry, College of Dentistry, Iowa City, IA 52242.

Trends in the prevalence of dental caries in Israel—page 464

This study was part of a nationwide study carried out in 1988 in West Jerusalem. The objective here was to assess the prevalence of dental caries in five-year-olds and twelve-year-olds. In 166 of the younger children, 27.7 percent were caries-free, whereas only 4.8 percent of the children between twelve and thirteen years of age were caries-free. Even in the same country, there are problems trying to compare findings related to the prevalence of dental caries.

Requests for reprints should be directed to Dr. Dan Zadik, Department of Community Dentistry, P.O.B. 12000, Jerusalem, Israel.

Evaluation of fluoride exposures in children—page 467

The prevalence and severity of dental fluorosis in the U.S. may be increasing because of a widespread ingestion of fluoride. The children in this study clearly received fluoride from multiple sources, including: drinking water sources; type of formula consumed; toothpaste ingestion; and dietary fluoride supplements. All of these are complex and variable sources. Care should be taken to avoid unnecessary risks of dental fluorosis.

Requests for reprints should be directed to Dr. Steven M. Levy, N330, DSB, The University of Iowa, Iowa City, IA 52242.

Oral changes associated with end-stage liver disease and liver transplantation: implications for dental management—page 474

This study investigates the pedodontic management of a group of children with end-stage liver disease, with particular reference to the oral manifestations of the disease, as well as the complications associated with liver transplantation. This study confirms and extends the findings of three previous reports that described the oral manifestations of congenital biliary atresia in childhood.

Requests for reprints should be directed to Dr. W. Kim Seow, University of Queensland Dental School, Turbot St., Brisbane, Queensland, Australia 4000.

The etiology, prevalence, and sequelae of infraclusion of primary molars—page 481

Dental infraclusion is a common condition, especially in mandibular primary molars. Most literature references claim infracluded molars do not exfoliate within normal time limits and must be extracted to prevent detrimental sequelae. The few documented studies on the topic suggest, however, that most exfoliate normally within a six-month period. Space loss and molar tipping may be prevented, either by restoring the height of the affected tooth or by using space maintainers without extraction. The successors of infracluded molars reportedly develop normally and have few occlusal abnormalities.

Requests for reprints should be directed to Dr. Joanna Douglass, Fellow, Department of Pediatric Dentistry, University of Connecticut Health Center, Farmington, CT 06030.

Treatment of a pseudo-class III relationship in the primary dentition: a case history—page 484

A modified Quadhelix was fabricated, activated and cemented. In eight weeks, the anterior crossbite was corrected, and four weeks later, the posterior relationship was found to be acceptable. Occlusion has remained stable to date. Correction allows for normal dental base development and subsequent favorable skeletal growth.

Requests for reprints should be directed to Dr. Stephen E. Grimm III, Assistant Professor, Department of Pediatric Dentistry, Howard University College of Dentistry, 600 W. Street, N.W., Washington, D.C. 20059.

Local anesthetic mortality: Report of case—page 489

Requests for reprints should be directed to Dr. Elliot V. Hersh, Director of Pharmacology and Therapeutics, Department of Oral and Maxillofacial Surgery, University of Pennsylvania-School of Dental Medicine, 4001 Spruce Street, Philadelphia, PA 19104-6003.

Intraoral etiology of a life-threatening infection in an immunocompromised patient: Report of case—page 492

Requests for reprints should be directed to Dr. Paul O. Walker, University of Minnesota-School of Dentistry, 6-150 Moos Tower-Pediatric Dentistry, 515 Delaware Street, Minneapolis, MN 55455.

REVIEW

Enamel hypoplasia in the primary dentition: a review

W. Kim Seow, BDS, MDSc, PhD, FRACDS

Enamel hypoplasia may be defined as a deficiency in enamel formation manifesting clinically as grooves or pits, as well as partial or total lack of surface enamel. Clinical significance of enamel hypoplasia includes poor esthetics, tooth sensitivity, malocclusion, as well as predisposition to dental caries. Of further clinical importance is the fact that enamel hypoplasia may provide diagnostic clues related to genetic influences and systemic diseases as well as local insults occurring during the long time span of dental development. The primary dentition may provide further unique information related to *in utero* development as it commences mineralization *in utero*, beginning with the central incisors at fifteen to nineteen weeks postmenstrual age and ending with the second molars at twenty to twenty-two weeks.[1-3] Mineralization of the crowns of the entire primary dentition is not complete until about twelve months postnatally.[3]

As in the permanent dentition, enamel hypoplasia in the primary dentition may be inherited as genetic diseases involving only the teeth, *viz.* amelogenesis imperfecta, or as oral manifestations of many inherited systemic diseases and dysmorphic syndromes. More commonly, however, enamel hypoplasia in primary teeth results from acquired environmental factors encountered during the period of enamel formation.

The objective of this paper is to provide a critical review of the current concepts of the wide spectrum of etiological factors involved in the pathogenesis of this significant clinical entity in the primary dentition.

Dr. Seow is senior lecturer in Pediatric Dentistry, University of Queensland Dental School, Turbot Street, Brisbane, Queensland, Australia 4000.

HEREDITARY ETIOLOGICAL FACTORS

Inherited types of enamel hypoplasia form a relatively small component of all developmental enamel defects in the general population. As shown in Table 1, this type of enamel hypoplasia is observed in genetic abnormalities of enamel formation (amelogenesis imperfecta) or as dental features of many inherited diseases or dysmorphic syndromes.

Amelogenesis Imperfecta

PREVALENCE

The term amelogenesis imperfecta is limited to those inherited defects that primarily affect only enamel formation and not accompanied by systemic effects.[4] It is a heterogeneous group of disorders, with reported prevalence rates ranging from 1 in 718[5], 1 in 4000[5], 1 in 8000[6] and 1 in 14,000[7].

CLINICAL PRESENTATION

Amelogenesis imperfecta has been conveniently classified into hypoplastic, hypocalcification and hypomature types of the enamel, although distinction between these types may have little ultrastructural basis.[4,8-10]

Table 1 ☐ Classification of inherited type of enamel defects.

I.	Inherited defects of enamel		
	Amelogenesis imperfecta	(i)	hypoplastic
		(ii)	hypocalcified
		(iii)	hypomature
II.	Inherited systemic conditions with enamel defects		
1.	Conditions related to ectodermal dysplasia and ectodermal-mesodermal disorders.		
	(i) enamel hypoplasia and terminal onycholysis syndrome		
	(ii) ankyloblepharon syndrome		
	(iii) epidermolysis bullosa		
	(iv) tricho-dento-osseous syndrome		
	(v) oculo-dento-osseous syndrome		
	(vi) incontinentia pigmenti		
	(vii) Ehlers-Danlos syndrome		
	(viii) mucopolysaccharidoses (type IV)		
2.	Multiple systemic malformation/dysmorphic syndromes.		
3.	Inherited disorders of calcium metabolism		
	(i) Parathyroid diseases		
	(ii) Enzyme disorders: hypophosphatasia, vitamin D–dependency rickets		

Several subclassifications have been proposed, based on the mode of inheritance.[4,7-12]

Table 2 shows a recent updated classification of amelogenesis imperfecta proposed by Witkop, which incorporates most clinical subtypes described in literature.[4] Many of these classifications were based, however, on subjective evaluations of clinical material, and often on small family pedigrees. Hence there is a need for continuing revision of such classifications as larger patient studies become available.

The primary dentition appears to be affected in most types of amelogenesis imperfecta.[13-17]

The hypoplastic type is characterized by enamel that does not develop to normal thickness, but contrasts normally from dentin in radiographs.[5,11,12] The clinical presentation of subtypes of the hypoplastic variety may differ significantly. In one of the most common forms (Type IA, Table 2), the hypoplastic enamel presents as pits, often arranged in rows and columns.[4,11] Pitted as well as grooved enamel may also occur in the local type of amelogenesis imperfecta, which may be inherited as autosomal dominant (Type IB) as well as autosomal recessive (Type IC).[6,11,18]

Table 2 ☐ Classification of amelogenesis imperfecta as proposed by Witkop, 1989.

Type I HYPOPLASTIC	
1A	hypoplastic, pitted (AD)
1B	hypoplastic, local (AD)
1C	hypoplastic, local (AR)
1D	hypoplastic, smooth (AD)
1E	hypoplastic, smooth (XD)
1F	hypoplastic, rough (AD)
1G	enamel aagenesis (AR)
Type II HYPOMATURATION	
IIA	hypomaturation, pigmented (AR)
IIB	hypomaturation, (XR)
IIC	snow-capped teeth (X D)
Type III HYPOCALCIFICATION	
IIIA	(AD)
IIIB	(AR)
Type IV HYPOMATURATION-HYPOPLASTIC	
IVA	Hypomaturation-hypoplastic with taurodontism (AD)
IVB	Hypoplastic-hypomaturation with taurodontish (AD)

AD–Autosomal dominant XR–X-linked recessive
AR–Autosomal recessive XD–X-linked dominant

Hypoplastic amelogenesis imperfecta may also be expressed as smooth and thin enamel, resulting in lack of contact between adjacent teeth.[8,11] This form has been reported to be inherited as autosomal dominant (Type ID), as well as X-linked dominant (Type IE).[4,11] In the latter mode of inheritance, carrier females show alternating vertical bands of normally thick and abnormally thin enamel as a result of the Lyonization effect.[11,14,19]

The hypoplastic rough autosomal dominant type (Type IF, Table 2) may be distinguished from the smooth type by the hard granular vitreous surface of the thin enamel observed in the latter.[11]

The extreme form of the hypoplastic type of amelogenesis imperfecta is observed in the autosomal recessive type of enamel agenesis (Type IG), where there is no clinical evidence of enamel formation.[11,20]

In contrast to the hypoplastic variety, hypomaturation enamel has normal thickness, but displays a mottled appearance, breaks off easily and appears approximately the same density as dentin in radiographs. Hypomaturation amelogenesis imperfecta may be inherited as autosomal recessive (Type IIA, Table 3) or X-linked recessive (Type IIB, Table 2), as well as autosomal dominant (Type IIC, Table 2).[11,21-25] The latter is characterized by a "snow-capped" appearance, which may resemble fluorosis, except for the absence of enhanced perichymata characteristic of fluorosis.[4]

The hypocalcified type of enamel may be distin-

Table 3 ☐ Systemic factors associated with enamel hypoplasia.

Birth trauma	
(1)	Breech presentation
(2)	Multiple births
(3)	Prolonged labor
Infections	
(1)	Syphilis
(2)	Rubella
(3)	Cytomegalovirus
(4)	Measles
(5)	Chicken Pox
(6)	Pneumonia
(8)	Gastrointestinal infections
Nutritional disorders	
(1)	General malnutrition
(2)	Vitamin D deficiency
(3)	Vitamin A deficiency
Metabolic diseases	
(1)	Toxemia of pregnancy
(2)	Maternal diabetes
(3)	Hyperbilirubinemia
(4)	Neonatal asphyxia
(5)	Hypocalcemia
(6)	Hypothyroidism
(7)	Hypoparathyroidism
(8)	Cardiac disease
(9)	Gastrointestinal malabsorption
(10)	Nephrotic syndrome
(11)	Chronic renal failure
(12)	Biliary atresia
(13)	Birth prematurity
Chemicals	
(1)	Tetracycline
(2)	Lead
(3)	Fluoride

guished by normal-thickness enamel, which consists of poorly mineralized matrix, resulting in early loss of the surface and a yellow-brown appearance. Radiographically, the enamel is less opaque than dentin and may have a moth-eaten appearance.[26,27] The hypocalcified type of amelogenesis imperfecta may be inherited as autosomal dominant (Type IIIA, Table 2) or as autosomal recessive (Type IIIB, Table 2).[4,11,28] It may also occur in conjunction with enamel maturation defects and taurodontism (Types IVA, IVB).[4,15,29]

Inherited systemic condition with enamel defects

Table 3 lists the broad groups of inherited systemic diseases that have been reported to show enamel hypoplasia.

Conditions related to ectodermal dysplasias and ectodermal-mesodermal disorders

As ameloblasts are derived from ectoderm, and the formation of enamel is dependent upon sound ectodermal-mesodermal interactions, many inherited conditions of generalized ectodermal and related abnormalities are likely to appear in abnormal enamel formation. This fact is demonstrated in some types of ectodermal dysplasias that show enamel hypoplasia as a constant feature. These include the enamel hypoplasia and terminal onycholysis syndrome described by Witkop *et al* (1975), as well as the ankyloblepharon syndrome (ankyloblepharon, ectodermal dysplasia and cleft lip and/or palate).[30,31] Other known conditions of ectodermal origin include epidermolysis bullosa, an X-linked recessive disease, characterized by skin and mucosa bullae and erosions, dysplastic nails as well as other abnormalities, such as anaemia and growth retardation.[32-35] Enamel hypoplasia characterized by pits and thin enamel has been described in this condition, particularly in the more severe forms.[36-40]

Enamel hypoplasia resembling amelogenesis imperfecta is a characteristic feature of two autosomal dominant syndromes involving ectodermal structures: the tricho-dento-osseous (TDO) syndrome and the oculo-dento-osseous syndrome.[41-43] The tricho-dento-osseous syndrome is further characterized by excessively curly hair, radiodense bone and taurodontism, while narrow nose with hypoplastic alae, microcornea with iris anomalies and syndactyly and camptodactyly of the fourth and fifth fingers are additional features of the oculo-dento-osseous (oculo-dento-digital) dysplasia.[41,44-47]

In incontinentia pigmenti, a developmental condition involving many structures of ectodermal and mesodermal origin, such as the skin, hair, nails, eyes and the central nervous system, enamel defects have also been reported.[48-53] Furthermore, in the Ehlers-Danlos syndromes, which is characterized by hyperelastic skin, joint hypermobility, increased bruising tendency and papyraceous scarring, enamel hypoplasia has been described.[54,55]

In some mucopolysaccharidosis such as the Morquio Syndrome, pitted enamel has also been described.[56,57]

Multiple systemic malformation/dysmorphic syndrome

In addition to the above ectodermal-mesodermal disorders, enamel hypoplasia may also be an accompanying trait of many dysmorphic syndromes and multiple system malformation syndromes. These include cleidocranial dysplasia, tuberous sclerosis, Prader-Willi syndrome, pyknodysostosis, acrocephalosyndactyly as well as in the new palmoplantar-hyperkeratosis syndrome reported by Seow (1989).[43,58-66]

Inherited disorders of calcium metabolism

As calcification of teeth is dependent on an intact metabolic pathway, derangements of this complex system

may result in enamel hypoplasia. Thus inherited diseases of the parathyroid endocrine system, vitamin D metabolism, liver and kidney diseases are likely to show enamel defects in affected patients. Enamel hypoplasia is a constant feature of many familial syndromes showing hypoparathyroidism. These include idiopathic and familial hypoparathyroidism, hypoparathyroidism associated with known syndromes, such as DiGeorge syndrome (athymia, hypoparathyroidism, cardiac hypoparathyroidism, adrenal insufficiency, diabetes mellitus, thyroiditis, myasthenia gravis.[43,67-73] The enamel defects associated with hypoparathyroidism are likely the direct result of hypocalcemia but they may also be an expression of ectodermal defects associated with hypoparathyroidism.[72,73,77,78] In addition, a syndrome of nephrocalcinosis, impaired renal concentration, possible abnormality of calcium metabolism and enamel hypoplasia resembling amelogenesis imperfecta was recently described.[20]

Another group of patients with inherited disorders of calcium metabolism and presenting with enamel hypoplasia are those with defects of enzymes linked with vitamin D metabolism and the calcification process.[77] In vitamin D-dependency rickets, an autosomal recessive disease where there is a failure of the kidneys to produce α-1-hydroxylase enzyme, there is impaired activation of vitamin D and resultant hypocalcemia.[79] Enamel hypoplasia has been commonly reported to occur in this condition.[11] Also, in hypophosphatasia, an autosomal recessive disorder where a deficiency of the enzyme alkaline phosphatase results in abnormal bone calcification and increased urinary excretion of phosphoethanolamine, enamel hypoplasia has been reported, although the most significant dental finding is usually premature exfoliation of the teeth associated with cementum aplasia.[80-85]

ACQUIRED TYPES OF ENAMEL HYPOPLASIA

In contrast to the inherited types of enamel hypoplasia, those sustained as a result of environmental factors are more commonly encountered. Developmental enamel defects resulting from these acquired causes tend to affect those teeth that are at developmentally sensitive stages, so that groups of teeth in each quadrant are affected, usually in a symmetrical manner. The term "chronological enamel hypoplasia" has also been used to describe such defects.[9] Local factors, however, tend to produce enamel defects that remain localized to one or a few adjacent teeth.

Many investigators have suggested that frank enamel hypoplasia results from injury to the ameloblasts during matrix formation stage, whereas enamel opacity or hypocalcification is usually related to injury occurring at the later maturation stage of enamel formation.[86] The degree of enamel loss depends on the duration as well as severity of the insult.[87] The most common presentation of acquired enamel hypoplasia is a horizontal groove located at a position of the tooth crown corresponding to its stage of development at the time of injury.[88,89] Less commonly, acquired enamel hypoplasia presents as complete or partial surface-loss of enamel, or surface pits.

Prevalence

Comparatively few studies are available on the prevalence of developmental enamel defects in the primary dentition. The prevalence rates vary considerably from around 4 to 60 percent, depending on the population studied, the teeth examined, and the criteria used to diagnose enamel defects, such as whether enamel opacities were included in the diagnosis.[88-97] Some general prevalence studies in developed countries on nonspecific enamel hypoplasia in the primary dentition have reported frequencies of 4 to 5 percent.[90,94]

Most other studies on the prevalence of enamel hypoplasia, however, have confined the examinations to specific clinical entities such as linear enamel hypoplasia and localized labial enamel hypoplasia of primary canines.[88,92,98-100] High prevalences of linear enamel hypoplasia have been reported in may underdeveloped countries such as 21 percent in Nigeria, 22 to 73 percent in Guatemala, 73 percent in Hawaiian children.[88,91,92,99,101] In addition, the prevalence is also high in disadvantaged groups such as low socioeconomic communities and native American Indians.[91,102] Linear enamel hypoplasia may be quickly attacked by caries resulting in characteristic lesions known as "odontoclasia" or circular caries, crescent-shaped caries or bar-type decay.[89,101-106] This type of caries and related enamel hypoplasia have also been found to be of high prevalence in prehistoric population.[107-109] This archaeological evidence suggests high levels of infant morbidity and malnutrition in the populations studied.

In contrast to the linear type of enamel hypoplasia, some studies have examined the prevalence of labially located enamel hypoplasia of the primary canines. This type of defect was first reported in Jorgenson (1956), who found it in 21 percent of modern Danish and 28 percent of medieval Danish primary canine teeth.[110] Other studies on modern populations have reported widely ranging prevalences of around 37 percent and 45 percent to only about 1 to 3 percent.[98-100,111,112]

Etiologic factors in noninherited enamel hypoplasias

That several diverse conditions have been associated with enamel hypoplasia attests to the fact that amelogenesis is sensitive to a variety of factors, summarized in Table 3. In the primary dentition, these factors may act prenatally, perinatally as well as postnatally, and may be systemic or localized. They are broadly classified into birth trauma, infections, nutritional disorders, metabolic diseases, and chemicals.

Systemic factors

☐ Birth trauma

At birth, even the normal change from intrauterine to extrauterine may have an adverse effect on amelogenesis as evidenced by a line of subclinical enamel hypoplasia also known as the neonatal line observed in ultrastructural studies.[113,114] Any stressful event during birth is likely to accentuate this line, resulting in clinically evident enamel defects.[115] Thus, difficult birth such as breech presentation, prolonged labor, multiple pregnancy, and caesarean section have been associated with enamel hypoplasia.[116,117] In these conditions, the enamel defects probably result from metabolic changes resulting from fetal stress.

☐ Infections

Severe infections occurring during amelogenesis may be associated with enamel hypoplasia. The mechanisms of damage may be related to direct cellular damage by the infecting microorganisms, although secondary systemic insults may arise from malfunctions of the major organs affected. Furthermore, the increase in body temperature observed in many infections may also cause ameloblastic derangements.[118]

Congenital infections with syphilis and rubella have been well documented to result in enamel hypoplasia of both primary and permanent dentition.[119-123] In addition it has also been shown that congenital cytomegalovirus infection is also associated with a high prevalence of enamel defects of the primary dentition.[124]

Postnatal infections with exanthematous diseases such as measles, chicken pox, and scarlet fever, as well as severe respiratory infections have all been associated with enamel hypoplasia.[125-128] In addition, severe gastroenteritis has also been reported to result in enamel defects, although it is unclear whether the cause is directly related to the infection or is secondary to the associated malabsorption.[129-131]

☐ Nutritional disorders

In some underdeveloped countries, the high prevalence of linear enamel hypoplasia has been significantly associated with malnutrition.[88,92,105,130,132,133] Although it is reasonable to assume that ameloblasts are affected by severe general malnutrition, deficiency of certain nutritional elements directly associated with epithelial cell function and the mineralization process must be particularly important. Thus deficiencies of vitamins A and D have been documented to result in enamel hypoplasia.[134-139]

☐ Metabolic diseases

☐ Hyperbilirubinemia

Hypoplastic teeth stained with green discoloration had been associated with jaundice and hemolytic disease of the newborn since 1912 by Thursfield.[142] Before the availability of effective control measures for hemolytic diseases of the newborn, enamel hypoplasia and intrinsic green staining of the teeth by bilirubin pigment were common observations in affected children.[143-146] In addition, Miller demonstrated that the green pigment was located in a band in dentine at the level of neonatal development.[147]

☐ Hypocalcemia

As calcium metabolism is directly involved in dental development, it is not surprising that in conditions that demonstrated disturbed calcium metabolism, enamel hypoplasia is often noted. As early as 1941, Gaunt and Irving demonstrated in animal experiments that hypocalcemia caused severe disturbances of tooth calcification.[148] Later, a clinical study by Grahnen and Selander reported that there was a significant increase in the frequency of enamel hypoplasia in a group of children who suffered hypocalcemic tetany (73 percent) compared with a control group of normal healthy children (3 percent).[149] In addition, Stimmler *et al* showed that all patients in their study who had neonatal hypocalcemia resulting from the feeding of unfortified cow's milk had severe enamel hypoplasia in spite of the short duration hypocalcemia.[150] A microscopic study of such hypoplastic teeth confirmed that the enamel defect occurred during the neonatal period.[151]

Further confirmation of the association of derangements of calcium metabolism and enamel hypoplasia is provided by the study of Seow *et al*, which showed that a group of prematurely born children who suffered neonatal rickets all had enamel hypoplasia.[152] In addition, hypocalcemia resulting from congenital abnormalities of the parathyroid glands have also been associated with severe enamel hypoplasia.[76,77,153]

☐ Other metabolic diseases

Metabolic disease associated with many other organ systems have also been implicated in enamel hypoplasia. These include congenital cardiac diseases and renal

neurological disorders.[154-159] Furthermore, children with kidney diseases such as nephrotic syndrome and chronic renal failure as well as liver conditions such as biliary atresia have also been associated with severe enamel defects and green staining of teeth.[160-162] These enamel changes associated with renal and liver malfunction are likely to result from derangements of vitamin D activation as well as calcium metabolism. In addition, gastrointestinal malabsorption conditions such as celiac disease, cow's milk intolerance as well as galactosemia have been associated with enamel hypoplasia, most likely from a deficiency of supply of calcium and phosphorus.[80,129,163-165]

☐ Birth prematurity and low birthweight

As early as the 1930s, prematurity of birth has been associated with enamel hypoplasia of the primary dentition.[166,167] Later studies of children with birth weights between 2500g and 3000g have shown the prevalence of enamel hypoplasia to be around 20 percent.[117,141,168-171] It has been possible only in recent years, however, to study children with extremely low birth weights, i.e. 1500g and below, because most of these children did not survive previously. Recent studies that included these very low birthweight children have indicated much higher prevalences of enamel hypoplasia ranging from 62-79 percent, 77 percent, 52 percent, 38 percent, and 30 percent.[172-177]

The inverse relationship of birthweight with prevalence of enamel hypoplasia was shown conclusively by Seow and co-workers in a well controlled study, which found that enamel defects was present in 62.3 percent of children with birthweight < 1500, whereas only 27.3 percent of children with birthweight 1500-2000g and 12.7 percent of control children with birthweight >2500g had the defects.[173]

Many systemic factors have been implicated in the pathogenesis of enamel hypoplasia including neonatal asphyxia, respiratory distress syndrome, neonatal rickets, maternal preeclampsia, maternal diabetes, hyperbilirubinemia, and neonatal infection.[116,117,140,152,175,178,179] It is likely that many of these individual factors may in fact, act through a central mechanism, that of mineral deficiency as proposed in a theory by Seow *et al.*[180] Adequate calcium and phosphorus stores are required during tooth and bone mineralization; premature infants, however, often suffer serious deficiencies of bone mineral (osteopenia) as a result of metabolic derangements and inadequate mineral supply.[181] Under these conditions it is likely that enamel hypoplasia may result directly from calcium and phosphorus being diverted from calcification sites as mineral is conserved for other vital body functions. Seow and co-workers were able to substantiate their hypothesis in a controlled study which showed that prematurely-born children with the greatest bone demineralization suffered the highest prevalence of enamel hypoplasia.[180]

In addition to systemic factors, local trauma to the alveolar ridge from the laryngoscope as well as from the orotracheal tube in mechanically-ventilated children also contribute significantly to the prevalence of enamel defects in prematurely-born children.[173,180,182-185]

☐ Chemicals

☐ Tetracyclines

Enamel hypoplasia and tooth discoloration caused by tetracyclines became widely appreciated in the early 1960s.[186] Tetracyclines given after the eighth week of pregnancy may pass through the placenta to affect the developing fetal dentition. Direct toxicity to the ameloblasts and disruption of the mineralization process are possible mechanisms of damage.[187,188]

☐ Fluoride

The effects of excessive fluoride in causing enamel hypoplasia have now been well documented since Dean showed unequivocally that the prevalence and severity of dental fluorosis is determined by the fluoride concentration in the drinking water.[189-193] The mechanisms whereby fluoride produces enamel changes, however, are still unclear.[194,195]

Dental fluorosis in the primary dentition is generally reported to being less severe than in the corresponding permanent dentition.[196-198] This may be attributed to the shorter duration of enamel formation and maturation of primary teeth or to the thinner enamel.[198,199] Severe fluorosis may be observed in the primary dentition, however, where the fluoride content exceeded six parts per million or six times optimal in temperate climates.[200,201]

☐ Antineoplastic therapy

Prolonged chemotherapy for childhood cancers is now recognized as a potential factor related to abnormal enamel formation. It is likely that many of the cytotoxic drugs employed may be directly toxic to the ameloblasts.[202]

☐ Local factors

Since Turner first described a localized type of enamel hypoplasia resulting from infection of primary teeth, many clinical reports have confirmed that local factors are important causes of tooth defects.[96,181,203-206]

☐ Trauma to developing teeth

Trauma to developing teeth may result in derangement of tooth development ranging from minimal morbidity such as an opacity, to severe changes manifested as enamel hypoplasia or even crown and/or root dilacer-

ation. Animal studies have provided direct evidence of these effects of trauma.[86,207] According to Andreasen and Ravn, as high as 10 percent of enamel hypoplasia affecting permanent anterior teeth in school children in Copenhagen were due to trauma to the primary dentition.[204] Other studies showed that the type of trauma sustained determines the degree of developmental disturbance with avulsion and intrusive luxation representing injuries with very high frequencies of developmental disturbances compared with subluxation and extrusion.[204,206,207]

In children who have been intubated during the neonatal period, laryngoscopy has been implicated in the etiology of localized enamel hypoplasia.[178,180-182] Furthermore, the orotracheal tube abutting on the maxillary alveolar ridge may also exert sufficient traumatic pressure to cause disruption of amelogenesis.[183-185.]

Other forms of trauma to developing teeth, which have been previously documented, include jaw fractures, surgical trauma, gunshot injuries and electrical burns.[96,205,208-212] It is of interest to note that animal studies have shown that traumatic forces are most effective in producing enamel hypoplasia in hypocalcemic states.[213] This observation has also been reported in humans in the study of Seow *et al*, which showed that prematurely-born children suffering from undermineralization of bone (osteopenia) were those most susceptible to the traumatic effects of laryngoscopy.[180]

☐ Cleft lip and palate

Teeth in the region of cleft palates are often hypoplastic.[214,215] Reparative surgery for cleft palates, however, may also be contributory factors in enamel hypoplasia.

☐ Local infections

Periapical infections of primary teeth causing enamel hypoplasia of succedaneous permanent teeth are also well known.[216] In addition, acute local osteomyelitis has also been established as a cause of enamel hypoplasia.[96]

☐ Other local causes of enamel hypoplasia

Irradiation has been known to cause enamel hypoplasia although ameloblasts have been reported to be generally resistant to low levels of irradiation.[217-219]

Ankylosis of primary teeth has also been associated with an increased frequency of enamel hypoplasia in the succeeding permanent teeth, although the reason for this association is unknown.[220,221]

Biochemical alterations in enamel hypoplasia

Although the biochemical basis of enamel hypoplasia is not completely understood, it is likely that enamel defects are related to alterations of the enamel matrix proteins during amelogenesis. These proteins consisting of amelogenins, which are present in the early stages of amelogenesis, and enamelins, which are found later in enamel formation, have major roles in the structural organization and mineralization of developing enamel.[222,223] In a recent investigation of teeth obtained from a patient with the hypomaturation-type of amelogenesis imperfecta, excessive amelogenins was detected in the hypoplastic enamel, confirming that the primary defect in this condition was an abnormality in the maturation process.[224] Furthermore, environmental insults such as high levels of fluoride have been shown to inhibit the secretion of enamel matrix proteins, as well as causing abnormal retention of amelogenins during the early maturation stage of enamel formation.[225,226] This, in turn, may interfere with crystallite growth and delineation.

Problems in clinical diagnosis

Although there is a diverse panorama of factors that may cause hypoplasia, the clinical appearance of the lesions is often nonspecific, and difficulties in diagnosis may be encountered. In addition, several systemic etiological factors often occur concurrently so that it is usually difficult to isolate and rank in order of importance, the individual causes involved. Added to this complexity is the synergistic interaction of local and systemic factors.[180,213]

To assist clinicians in the diagnosis of enamel defects, a history and examination check list may be used (Table 4). If amelogenesis imperfecta is suspected, members of the immediate and extended family should be examined to established diagnosis and mode of inheritance. In the acquired types of enamel hypoplasia, specific diagnostic tests may be employed, such as fluorescence under UV illumination in the case of tetra-

Table 4 ☐ Clinical approach to patients with enamel hypoplasia.

1.	Full history (prenatal, neonatal, postnatal)
	– systemic diseases, drug intake
	– fluoride intake
	– familial conditions
2.	Clinical examination
	– which teeth are affected
	– types of enamel hypoplasia
	– location of enamel hypoplasia
	– any pigmentation of teeth
	– any fluorescence under UV illumination
3.	Clinical problems and management
	– esthetics
	– dental caries
	– tooth sensitivity
	– loss of occlusal vertical dimension

cycline staining and enamel biopsy in the case of fluorosis.

FUTURE STUDIES

In spite of significant research advances into dental development and enamel formation in recent years, the pathogenetic mechanisms of developmental enamel defects remain poorly understood. It is likely, however, that advances in cell and molecular biology will rapidly improve our understanding of this complex field in the future. The development of molecular probes for enamel proteins will enable accurate biochemical analysis of abnormal enamel and may aid in the diagnosis of many inherited and acquired types of enamel hypoplasia.[227] Furthermore, controlled animal studies investigating direct and indirect local or systemic causes of enamel hypoplasia may shed further light on the complex interplay of these factors in the pathogenesis of enamel hypoplasia. Finally, properly controlled long-term clinical studies of children with systemic and local enamel anomalies are urgently required to identify the clinical complications associated with this common clinical entity.

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BEHAVIOR

Dental treatment of fearful children, using nitrous oxide Part I: Treatment times

J.S.J. Veerkamp, DDS
W.E. van Amerongen, DDS, PhD
J. Hoogstraten, PhD
H.J. Groen

Nitrous oxide is a well-known and accepted tool for managing children during dental treatment. Sedated with nitrous oxide in concentrations from 20 to 50 percent, children seem to relax more easily in circumstances they generally dislike, and which are considered to be stressful.[1,2]

The use of nitrous oxide in dentistry has been widely investigated, especially in young children.[1,3] Many studies focussed on patients of an age when behavioral management is handicapped by lack of oral communication. The main aim of these studies was to investigate the possible improvement of behavior during dental visits. The results indicate the usefulness of nitrous oxide, but stress the importance of the dentist's behavior.[1,3] Nitrous oxide is known to be an effective aid in treating moderately anxious pediatric patients, but its use as a sole strategy in managing highly anxious children during dental treatment is questionable.[3,4] Recently, behavioral management has regained appreciation: studies pinpoint the effective words and behaviors the dentist can use to lessen dental fear and apprehensiveness. From this point of view, one should see the use of nitrous oxide merely as an additive to behavioral management, to relax the child more easily and make him or her accessible to the words and suggestions of the dentist.

Until now, very little attention has been devoted to the effect of the use of nitrous oxide on working time. One might hypothesize that dental treatment is initially more time-consuming using nitrous oxide, especially if patients are not used to it; but this disadvantage is neutralized, if the more relaxed patient enables the dentist to work more efficiently.

The aim of this study, then, is to investigate the influence of nitrous oxide on treatment time, when it is used as an additional aid to behavioral management in treating highly anxious child dental patients during sequential dental visits. As different parts of the treatment might vary during sequential analysis, we will also consider separate consecutive parts of the treatment in terms of the time they take.

MATERIALS AND METHODS

For this study, fifty-six children were selected. The children had previous (negative) dental experience, in all cases unfinished dental treatment resulting in a referral to our dental fear clinic. The children were selected during a screening visit by a dentist not

Drs. Veerkamp and van Amerongen are with the Department of Pediatric Dentistry, ACTA; and Drs. Hoogstraten and Groen are with the Department of Social Dentistry, ACTA, Louwesweg 1, 1066 EA Amsterdam, Holland.

Time	Starting of the interval	Contents
1	Child sitting in the chair	Becoming familiarized with it
2	Nasal mask put in place	Sedation
3	Dentist reaching for the syringe	Local anaesthesia
4	Dentist reaching for the bur	Actual conservative treatment

Figure. Time intervals during dental treatment.

participating in the actual treatment, using the following criteria:

- ☐ Age between six and eleven, ensuring a sufficient level of mental development and verbal skills, but not yet adolescent, a period characterized by its own rules and regulations of behavioral management.
- ☐ Not attending a special school (for mentally retarded children or emotionally disturbed children or children with educational problems).
- ☐ Dutch native speaker.
- ☐ Scoring high on a special (Likert-type) anxiety-scale.[6]
- ☐ Previous dental experience; but not previous dental experience at our clinic.
- ☐ Sufficient dental disease to require at least two consecutive conservative treatments.

To standardize treatment conditions, a few additional decisions had to be made:

- ☐ Parents were not allowed to be present during treatment (to prevent the child being influenced by the behavior of the parent or their presence).
- ☐ The dental assistant was not allowed to communicate with the patient (to make sure the only verbal or nonverbal contact was between the child and the dentist).
- ☐ Conservative treatment was given, using rubber-dam and anesthesia.

The sedation times differed.

The children were divided randomly into two matching (sex, age) groups: one to be treated with behavioral management only (control group), the other with behavioral management and nitrous oxide sedation (experimental group). Two dentists, both with six years of experience in managing fearful children, took part in the study. Children from both groups were assigned randomly to each of them, each dentist treating the same number of children from each group. If the parents expressed a preference for either treatment condition, the child was not included in the study.

Treatment conditions

Treatment was divided into four consecutive parts: time recording was started when the child sat down in the dental chair, producing the intervals shown in Figure, and ended when the child left the chair after treatment.

The first treatment session was spent introducing and getting used to procedures. It included prophylaxis, instruction in dental hygiene, and, in the experimental group, getting acquainted with the use of nitrous oxide. This consisted of explaining the nose piece, combined with a relaxation exercise, and administering nitrous oxide in a concentration up to a level where the child was sedated, with a maximum of 40 percent. After sedation, the treatment was continued in the same way as in the control group.

The dentist decided whether another introductory visit was needed. Restorative treatment was provided at the earliest moment in the next session. In the sedation group, the nitrous oxide was discontinued during the last minutes of treatment. All sessions were videotaped, using a fixed auto-focusing camera with a built-in clock. The nitrous oxide was used within the limits concerning scavenging and long-term exposure that make it a relatively safe aid nowadays.[7,8]

The dentists were well trained in using behavioral management techniques on children. Behavioral management as used today, is a combination of skills, a way of acting rather than of using any specific technique. The dentists adjust their words to (the age of) the child,

Table 1 ☐ Number of children and total number of sessions needed to complete dental treatment for each group and each dentist.

Sessions	Control	Experimental	Dentist 1	Dentist 2
1	–	–	–	–
2	–	–	–	–
3	4	3	6	1
4	7	8	14	1
5	9	6	4	11
6	4	8	4	8
7	–	1	1	–
8	2	2	1	3
9	1	1	1	1
T	27	29	31	25

Table 2 ☐ Average number of sessions for each dentist and each condition. Standard deviation between brackets.

Condition	Dentist 1	Dentist 2	Mean
Control	4.2(1.0)	5.5(1.1)	4.8(1.2)
Experimental	4.9(1.8)	5.7(1.0)	5.2(1.5)
Mean	4.6(1.5)	5.6(1.0)	5.0(1.4)

F (dentist) = 8.32, df = 1,52, p = 0,006*)
F (condition) = 1.74, df = 1,52, n.s.*)
F (mean) = 4.79, df = 2,52, n.s.*)

*significant, if p≤0.05

Table 3 ☐ Treatment times of both dentists for both treatment conditions (time in minutes, added up during course of treatment).

Interval	Control	Experimental	F	df	P)**
1					
(dentist1)	20	36	9.16	1,29	0.05
(dentist2)	24	41	4.51	1,23	0.04
2					
(dentist1)		27			
(dentist2)		41			
3					
(dentist1)	34	30	0.35	1,29	n.s.
(dentist2)	30	23	3.13	1,23	n.s.
4					
(dentist1)	58	52	0.37	1,29	n.s.
(dentist2)	94	107	1.44	1,23	n.s.
TTT)*					
(dentist1)	116	147	2.46	1,29	n.s.
(dentist2)	158	218	5.91	1,23	0.02

)*total treatment time
)**significant, if p≤0.05

clearly describe the desired behavior, and reward it afterwards. They always explain what they are doing; ignore undesirable behavior to avoid treatment, if possible; and only become angry, if the situation calls for this response.

Statistical analysis

The time intervals were recorded from the clock on the videotape. The results were analyzed using SPSS.[10]

RESULTS

The number of sessions needed to complete a dental treatment is given in Table 1. The children needed at least three sessions for dental rehabilitation and most of them were treated in four to six sessions. Statistical analysis indicates no significant difference in the number of treatment sessions (F = 1.74, Table 2) between the control and the experimental groups. Treating highly anxious children using nitrous oxide did not require a larger number of treatment sessions. Dentist 1 usually completed treatment of most of his patients in three to four sessions. Dentist 2 usually finished treatment in five to six sessions, indicating a different work schedule.

The average number of sessions for each dentist is give in Table 2. It also shows the analysis of variance to check for interaction effects. Table 2 demonstrates a difference in the number of treatment sessions between the two dentists. The difference is statistically significant (p = 0.006). The analysis of variance showed no interaction effect; there was no relation between the treatment group and the operating dentist, either way. Both dentists had their own consistent way of managing their treatment time, regardless of the nature of the treatment.

Treatment can be viewed in terms of the number of treatment sessions, but also in terms of the time needed for each treatment interval. Because of the discrepancy in treatment sessions between the dentists, the times of the treatment intervals, as shown in Figure, are given in Table 3.

Between conditions

For both dentists, interval 1 (getting used to procedures) took significantly more time, if nitrous oxide was used. This, combined with interval 2 (sedation) caused a further significant difference in the TTT (total treatment time) of one of the dentists. Intervals 3 (local anesthesia) and 4 (preparation and filling) did not demonstrate any further significant differences between the conditions. The TTT of the experimental group was longer, therefore, though the difference was only significant for one dentist. The difference, however, was not caused by the use of nitrous oxide itself during conservative treatment, but by the fact that the child had to get acquainted with sedation (interval 1) and the sedation procedure (interval 2).

Between dentists

The sedation time differed considerably between the two dentists, probably due to the difference in the number of treatment sessions (Tables 1 and 2). A considerable difference in restorative treatment time between the dentists was observed, but no interactions were found.

DISCUSSION

Nitrous oxide has proved to be an useful additive in reducing dental fear. As a tool to reduce dental fear in children with previous negative dental experience, it is useful to know whether it can be used efficiently in

The need for the child to become acquainted with sedation procedures increased total treatment time, where nitrous oxide was used for the first time.

the home office. In this study, treatment conditions did not differ considerably from those in the home office; the dentists worked as efficiently as possible, spending no extra time on experimental records. The results thus have direct practical applications.

The results demonstrate that the use of nitrous oxide is not time-saving. Whether the use of nitrous oxide is a time consuming activity depends on the dentist. It causes prolonged sessions of dental treatment, but no extra sessions (Tables 2 and 3), which would have increased the difference in treatment times between the two groups. Both dentists spent significantly more time getting the patients accustomed to being treated with nitrous oxide, resulting in a longer total treatment time for one dentist. For the other dentist, the extra time needed for sedation was balanced by a more efficient total treatment. The treatment intervals, however, did not demonstrate any further time-saving effect: since the dentists only started the actual treatment after the child was fully acquainted with the sedation procedures and the third and fourth interval did not show any difference, using nitrous oxide did not appear to be a time-saving activity.

The assumption of a similar level of dental anxiety and the same amount of dental disease in the two groups as a consequence of the random selection was confirmed by the number of sessions needed (Table 1, first part). One might wonder why it took significantly more time to get used to sedation. One reason might be that the anxious child had trouble adapting to a new treatment situation and even more trouble adapting to a situation where he or she appeared to have less control; the nasal mask seemed to cause a restriction of the children's movements.

Early studies indicate that the dentist is only inclined to use verbal support, if the behavior of the child makes it necessary.[9] In the long run, this might prove to be a time-consuming activity. Moreover, the dentists in this study noted that the use of nitrous oxide hindered verbal communication, mostly because the sedated child was not inclined to participate spontaneously in the communication with his dentist. This finding is supported by Lindsay, though merely based on feelings of the dentists in his survey rather than on statistical findings.[5] If sedation enables the child to distant himself more easily from treatment, it might be that the sedated child has less need for verbal support or verbal control. All this might save a lot of time and effort on the part of the dentist who would have to provide verbal support.

It might seem inappropriate to present a study on working times in treating anxious children. The main aim of the dentists in this study, however, was to reduce dental fear to a level where normal dental treatment was possible and secondly, to perform such treatment. From this point of view, it is to the benefit of the child to make the treatment as efficient as possible.

The patients needed three to nine sessions to solve all the dental problems (including behavioral problems). In the experimental and control groups, the amount of dental disease was the same. As the working times for each treatment session might vary considerably due to the amount of work planned for that ses-

sion, the total amount of time needed to make the child dentally healthy was used for the statistical analysis.

In this study, two dentists treated the children. Their working times differed significantly. No interaction effect was found, however: the difference in time in the experimental group corresponded with the difference in the control group. No significant differences were found between the sedation times of the two dentists or the amounts of time they needed to allow the child to get used to the treatment conditions. In the two matching groups as treated by the two dentists, only a significant difference was found in the actual restorative treatment time, merely indicating a difference in restorative treatment style. Apparently both dentists felt an appointment should not take too much time, and since the assistant always scheduled the same amount of time for the conservative treatments, the first dentist was inclined to work a little harder, if the amount of work was too much, while the second dentist just made another appointment. In some cases, nitrous oxide had to be used at the next appointment, producing the longer sedation time seen in Table 3. In the long run, these differences might influence the children's anxiety levels. This is why further research is mandatory.

The ages of the patients in this study were between six and eleven. At this age, fantasy still exerts a great deal of influence on the child's perseverance, but it is based more on reality than on the fairy tale way of thinking and solving problems we observe in the younger child. At this age, the child still accepts whatever an adult tells him or her. A few years later, the adult (the dentist) might find himself in a more difficult situation trying to convince an adolescent to abandon his ideas (i.e. fears). This implies that the six to eleven age-group is a specific one and the results of this study only refer to this group.

Treating children requires special skills on the part of a dentist, who is apt to have even more trouble, if this child has a history of negative dental experiences: now the dentist is not able to just teach or demonstrate something new to the child; he also has to erase the imprints of the previous dental treatment. Doing so by using nitrous oxide as an additional tool for behavioral management might prove in the long run to be a time-saving technique. Further analysis of time records can be advised, combined with further research into the anxiety levels of this critical age.

CONCLUSIONS

This study shows that:

- ☐ The use of nitrous oxide as an additional tool in treating anxious child dental patients in the six to eleven age-group is more time-consuming than the use of behavioral management as a sole technique for allowing the child to get acquainted with dental treatment. The influence on the rest of the treatment varies with the operator. For one operator, the use of nitrous oxide results in a significantly longer total treatment time.
- ☐ Using nitrous oxide, dental treatment does not require a greater number of sessions.
- ☐ The use of nitrous oxide does not appear to be a time-saving activity.
- ☐ If an operator uses more sessions for a comparable amount of dental work, this results in a longer total sedation time, if nitrous oxide is chosen as an additional tool.

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An analysis of the phenomenon of increased parental participation during the child's dental experience

Jimmy R. Pinkham, BS, DDS, MS

It is easy to present the premise that dentistry for children in its clinical practice is from the onset of the clinician-patient interface a behavioral science. The effective psychosocial abutment of an adult dental care provider with a child dental care receiver is critical for both participants. Its importance outweighs considerably all the other factors that are part of the dental experience for children, such as biological sciences, diagnostic skills, metallurgical techniques, chemistry, physics, and so on. The design of cavity preparations, the mechanism of fluoride's action on hydroxyapatite crystals, or the process of pellicle removal by microabrasives have no realistic meaning, if the dentist behaviorally cannot secure the child in the chair by some practical means that allows for the delivery of the nonpsychological parts of dentistry.

In fact, behavioral pediatric dentistry has been so important for so long that it could be concluded that surely dentistry put issues related to the behaviors of children to rest a long time ago. This conclusion, however, would be unwarranted.

"*All is flux, nothing is stationary. There is nothing permanent except change.*" is a quote attributed to the Greek philosopher, Heraclitus (*c.*540-*c.*470 B.C.), which introduced a 1990 Journal of Dentistry for Children article reviewing behavioral themes in pediatric dentistry as reported in the Journal from 1968-1990.[1] The quote was appropriate, because one of the most substantial conclusions of this essay was that considerable change had happened in this arena of dentistry for children during those twenty-two years. Furthermore, the review also concluded that all evidence would seem to predict even further changes in behavioral pediatric dentistry during the 1990s.

One aspect of behavioral dentistry for children that has certainly captured the attention of the profession during the last decade has been the role of the parent during their child's dental experience. This is particularly true for the young patient, and especially the preschool patient between the ages of three years and six years. There appear to be schisms within the profession regarding the advantages or disadvantages of parental presence in the dental operatory, during the child's dental experience. Included in this matter are issues regarding risk management and practice management for the dentist who treats children.

The purpose of this article is to focus on the issues regarding parental participation in the child's dental appointment. This paper is not a research paper. Instead, it borrows concepts espoused by two very respected scientists: anthropologist, Dr. Margaret Mead; and social biologist, Dr. Edward O. Wilson, and uses their ideas in the context of examining the changing interface of the dental clinician with the American child.

There is no arrogance intended in this paper. Any serious student of Dr. Mead or of Dr. Wilson could draft a similar paper and could, arguably, say that they got to the heart of the matter more cogently, more accurately, and perhaps faster than I. So be it.

Dr. Pinkham is Professor and Department Head, Department of Pediatric Dentistry, College of Dentistry, The University of Iowa, Iowa City, IA 52242.

There is no humility, however, about this offering either. There remains controversy as to the best way for a dentist and a child to meet and the most appropriate role for the parent to play during this meeting. The dental literature discussing the interface of dentists with children must continue to evolve by borrowing from as many collateral sciences as possible. As long as there is controversy, it is not flippant to suggest that the thoughts of this children's dentist or any children's dentist be recorded for the rest of the profession to ponder, question, attack, or even agree with. In fact, the very last sentence in the summary of the 1990 review article on behavioral themes in dentistry for children as recorded in the *Journal of Dentistry for Children* highlighted this theme:

"Lastly, it probably means that dentistry for children is very much a changing behavioral activity and that any journal addressing this activity must by definition continually address the subject and its changes."[1]

BASIS FOR THE CONTROVERSY OF PARENTAL ATTENDANCE IN THE OPERATORY

There is evidence that there have always been dentists who appreciated and, therefore, enlisted routinely the support of parents during their children's dental experiences. There also appears to be, however, from the very beginning of the organized dentistry-for-children movement in the U.S.A., which gathered momentum very rapidly after World War I, that the "party-line" philosophy within dentistry-for-children circles was that parental attendance was not needed; that at least in some instances, it was counterproductive; and that many dentists were uncomfortable attending to the management of the child, when the parent was in attendance. These three facts are echoed in the pediatric dentistry textbooks published over the ensuing decades, in journal articles, in notes taken during professional lectures, and even in intraprofessional conversations. In fact, the American Dental Association itself for years offered attractive posters for display in reception rooms that reminded parents that it was their responsibility to remain in the reception room during their child's visit.

So at some point in the past—let's settle on twenty-five years ago—there was a party-line attitude about parents in the operatory that was so absolutely clear that the major organization representing dentistry felt a responsibility to represent the needs of those dentists who routinely treated children sans their parents by the publication of a placard reminding parents that they needed to stay in the waiting room. Then what happened to account for certain dentists campaigning for greater involvement of the parents at the dental appointment; the conduct of a major behavior management symposium sponsored by the American Academy of Pediatric Dentistry Educational Foundation which addressed among others this important theme; the discussion to this topic by a variety of authors, lecturers, and academicians?[2] Why all this change, discussion, and/or concern?

Every reader of this article knows the answer to this last question. Each answer may vary in content and context, but one fundamental noun resounds in any plausible answer and that noun is *change*. Life in the United States has changed dramatically in the last few decades. It would appear that Toffler's warning in 1970 that the future would "shock" us was reasonable.[3] The American family has evolved through extraordinary external events and the relationships of children to their parents and to other adults with whom they must interface have been defined and redefined, again and again. Children's dentistry and the dentist-to-child-patient encounter was one of those relationships that has been examined and in some instances modified. This process is still going on.

There have been in the United States dramatic changes in parenting strategies, in the years following WWII. Authorities and researchers from a variety of disciplines have delved into these changes and forwarded their thoughts and conclusions, their anticipations and worries, about what is happening in this arena of American life. As a health scientist, perhaps no unifying theory has seemed to me to be more basic to understanding the changing nature of parenting strategies than has been Margaret Mead's description of postfigurative, configurative, and prefigurative paradigms regarding the parenting of children.[4] Mead writes convincingly about how quickly America metamorphosed from a culture that reared children according to conventional techniques (postfigurative) into a transitional stage where modifications of various types and experimental levels were allowed (configurative); and finally, in certain places and for certain people, into parenting strategies that are very contemporary with sometimes day-by-day or even hour-by-hour adjustments to the needs of the child (prefigurative).

This is a theme that has been highlighted very strongly for the pediatric medical community by Shulman and Hanley in their textbook, *Anticipatory Guidance*.[5] Their admonishments to their medical colleagues about being aware of a different parent with a different child being often the rule today and their reliance upon Dr. Mead's conclusions about changing strategies in rearing children were the very heart of the essay describing the

phenomenal twenty-two years of change that pediatric dentistry experienced from 1968-1990 in the area of behavior management of children.

The basis for the controversy then is that parents have changed in certain attitudes about children, and so did some dentists. Had everybody changed, in other words, had everyone lost all postfigurative values and acquired the same prefigurative paradigm, there would be no controversy. That did not happen, however, and ergo, the intraprofessional arguments began.

PARENTAL PARTICIPATION IN THE DENTAL APPOINTMENT

Why do Parents Want to Participate? Why Would A Dentist Want Parental Participation?

Conversations with numerous clinicians who have been in practice for more than two decades often underscore their conclusion that there has been a marked increase in the number of parents desiring to be present in the operatory during their children's appointments for treatment.

It is also this author's conclusion with two decades of experience in working with dental students that there is a much more determined tendency today among dental students and young dentists to want parents in attendance than there was a decade or certainly two decades ago. In fact, some regard the solo visit—i.e. young child without a parent—as unreasonable.

These two findings seem compatible and probably are: parents who want to attend and dentists who are willing or even anticipating parental attendance. They are probably evolved out of the same major societal changes that have taken American parenting from a pre-World War II postfigurative style to a configurative style in early post WWII decades to the post-1980s prefigurative style of today found in so many communities in the country. So something profound happened during this metamorphosis to change at least some parents' and at least some dentists' opinions about the role of parents in their child's dental appointment. What happened? Perhaps a better question is: Why do parents want to be in attendance during their child's dental experience? Why indeed?

Many parents might argue that it is because they believe that their child will behave better. This argument could also be generated by dentists supportive of parental attendance. It is a very, very reasonable answer. It is, however, not without substantial detractors. The dental literature provides abundant anecdotal information, generated over all the decades that dentistry for children has been an organized entity within the American dental community, that many children after the 3rd birthday do behave better, when their parents are not in attendance. In fact, almost every dentist who has practiced parental separation has encountered those circumstances whereby the child exploded in misbehavior at the advent of entering the operatory with his parents and then upon the parent's departure reacted with good behavior and even in fact went on to have a wonderful occasion.

Three- and four-year-olds are remarkably engaging patients.

I have not seen any compelling evidence that supports the notion that normally developed and psychologically stable preschool children cannot have a successful experience in a new environment, without their parents being present, but under the aegis of nonparental, yet child-oriented adults. Most information would lead one to conclude that man is a social creature, a herd animal that very much needs its own kind; and that the desires for social compatibility manifest themselves early in life and are certainly apparent in the preschool child. In fact, many parents in response to safety concerns have talked to their preschoolers about strangers and when, where, and how to avoid them; and many are chagrined that in the eyes of preschoolers, even strange-looking strangers may not seem so very strange. Preschoolers, as a group, are gregarious and adults from a variety of environments routinely approach them successfully without their parents in attendance. There are many authorities who endorse this conclusion. The following quote, from a physician, is one example:

"Three and four-year-olds are remarkably engaging patients. This developmental stage is marked by rapid growth in language and motor skills, which in turn enables the child to take important steps in social and emotional development."[6]

Furthermore, there is a tremendous amount of evidence, at least by the older practicing dental community now and by our forefathers in dentistry earlier, that we have had decades of very satisfactory interfaces with children, without their parents there. It might even be added that a lot of these were encounters in earlier times, when dental equipment was not so fast and fancy, when children had considerably more dental problems than today, when there were not Berenstain Bear books portraying dentistry as a nice experience or Sesame Street and Mister Rogers vignettes highlighting the fact that a dental appointment is a fun thing to do.

I submit that prefigurative parents, because they work with a parenting strategy that makes moment-by-moment adjustments in their guidance of their child especially if their child looks anxious at all on the way to a dental appointment. The following paragraph from the 1990 review article summarizes some of my thoughts on this issue: "The example could be made that postfigurative parents will expect their 3-year-old to go through the dental appointment behaving well and do so without parent accompaniment, because that was always the way it was done. Configurative parents may opt to do it that way, because that was the way it was done for them, and it worked out just fine, or they may have a strong desire to accompany their child, because they remember it did not go so well for them during their first appointments. They will pair their own remembrances with present convictions. The prefigurative parent must solve this one before arriving at the dental office. Likely, if the child looks anxious, they will want to stay with the child. That is not an absolute assumption, but experience supports it."[1]

Some of the thoughts of Dr. Edward O. Wilson as expressed in his Pulitzer Prize-winning book, *On Human Nature*, would seem to support the conclusion that the prefigurative parent could be very emotionally driven to want to be in attendance with their child.[7] Dr. Wilson notes that the emergence of civilization depended to a large degree on the hypertrophy of preexisting social tendencies within humankind. His purely biological analogies are very clear and instructive. He sees the tusks of elephants as a hypertrophy of a tooth and the cranial bones of the forefather of a stag elk hypertrophying into antlers of astonishing size. Socially he argues that the subtle social responses and adaptations of the hunter-gatherers developed into the sociologies of advanced societies in certain cases by the same process of hypertrophy.

In his book, Wilson includes very thorough descriptions of human behavior that depict this phenomenon of hypertrophy. For instance, the !Kung San of the Kahlahari Desert are examined as to how they do not impose sex roles upon their children as a rule, but how certain !Kung bands have in response to taking up an agricultural lifestyle "hypertrophied" subtle male and female tendencies observable in young children into sexual roles far divergent from the more homogeneous sexual roles shared by the !Kung at large. Subtle sociobehavioral tendencies, when allowed to hypertrophy, can become overt and compelling features of human behavior.

Can hypertrophy be useful in understanding prefigurative parents and their expectations of attendance at their child's dental appointment? For hypertrophy to be an issue pertinent to parental attendance at all, it must first be argued effectively that there is deep within each member of humankind a tendency (potential) to feel protective of one's child. This seems to be an undeniable conclusion. In fact, I offer that many parents would say that they would be most capable of a sacrificial altruistic act—i.e., giving a part of their body or even their life to spare someone else's—for their child. In fact, I believe that they would say it would be easier for them to do such for their own child than perhaps for any other conceivable person they have ever known.

Accepting the fact that there has always been a desire to protect one's child from possible harm, then why was this not seen in dental offices or felt by dentists in the early decades of the dentistry-for-children movement before WWII. How did the "party line" of parents not being in attendance get started?

The answer is simple. In a postfigurational society, this tendency was allowed to stay latent, because there was substantial intrasocietal trust, and that parents believed there were certain adults to whom they entrusted their child in a solo fashion. Postfigurative societies are in fact very strong that way. Furthermore, traditional societies often are conducted regarding certain aspects of childhood with the conclusion that "if it was good enough for me then it is good enough for my child." Since dentistry for children had started out for the most part with parents not accompanying children, that finding became the norm and the expected.

Furthermore, when the whole community reacts by trusting dentists to be alone with children, even that parent who is inclined to accompany their child to the operatory will probably find it easy to suppress the desire, because of the substantial feeling in the community that a parent need not or even should not stay with his or her child. Pair this fact with a dentist who

has been convinced that there is no reason for a parent to be there and who openly admits he does not want the parent to be there and who has bought from the American Dental Association a poster telling parents that the American Dental Association does not feel that the parent should be there, and you have what was for generations the reality that the dental experience was not attended by a parent.

The advent of the prefigurative parent living in communities of other prefigurative adults and being treated by younger dentists of prefigurative or configurative persuasion has seen very substantial dissolution, however, of many dental communities consensus conclusion that parents are not needed in attendance during their preschool child's visit. This would seem to be in Dr. Wilson's words the hypertrophy of the tendency parents have to protect their child. It is easy to see how this hypertrophy could occur. Mobile societies living in communities where the names of neighbors three apartments removed or a quarter of a block away are not even known underscore some of the disadvantages that urbanization and mobility have brought to the American parent and the American child. A lack of familiarity with other people and the roles that those people play within the society have hypertrophied fears of outside adults that remained dormant in quieter, more stable, and less forbidding social environments.

So we have a protective parent. Is this the overprotective parent recognized by leaders in dentistry for children, decades ago?[8,9] Perhaps that parent was the vanguard of the much larger group found today. The salient point is that the trend toward increased parental protection is becoming more widespread. Today's dental clinician, therefore, needs to understand the emotional convictions of these parents. Their desire to attend does not mean that they intellectually distrust the dentist or his abilities with their child. It does mean, however, that they are uncomfortable, if they cannot visually verify their child's safety. This desire to attend may extend even to the general anesthesia suite of a hospital.

Dentists are encouraged to understand that, if parents are determined to be in attendance with their child, the decision was made objectively, even though its origin was an emotional one, but also very normal. Any strategy to change this parent's mind must deal with her emotional needs and probably will only be satisfactory when the dentist explains what will happen during the dental experience and to what levels of management the dentist is prepared to go. This is the very heart of risk management for the dentist who wishes to separate the child from the parent.

Another issue is important here. Dentists who wish parents to accompany their child should be encouraged to avoid explaining their strategy as a more sophisticated way of behavior management than separating parent from child. They should not describe their method as the "good one" and the method of separation as a "bad one." Words like good or bad are not appropriate here. What is appropriate is to understand the changing emotional needs of American parents, because of the growth of a latent, but natural sense to be protective of their child, which has been allowed to foster because of the strong prefigurative philosophy about children today. This prefigurative parenting philosophy has been quickly realized in the American scene, because of the shocking changes in many aspects of American life, in the last several decades.

The changing emotional needs of parents are due to a latent but natural sense to be protective of their child: today's strong prefigurative philosophy.

FINAL COMMENTS

At this time it appears that in a growing number of dental offices, parents are expected and welcome to be present in the operatory room during the child's appointments. Whether this will continue, stabilize, or even reverse itself is a point of prognostication well beyond the intent of this paper. Who knows?

There are dentists on both sides of this issue, resulting in a schism that is potentially dangerous. It could become the basis of litigation, professional unrest, stress, and all those other unfortunate ramifications that happen when people disagree. There is evidence that some of these things have already happened. The question of a parent's presence should not cause conflict within the profession, however, as long as we understand why we can so easily be emotional about it.

Instead, we need to be students of change. It is hoped that the terms like social hypertrophy and prefigurative parenting are useful concepts for the dental clinician who has to recognize the sometime shockingly and many times quickly evolving environment in which he lives and conducts his practice. It is assumed that removing the mystery around the expectations of certain parents or the clinical philosophies of peer clinicians may lessen the anxieties of the dentist who is a student of these processes.

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THE ROOTS OF CHILDREN'S MORAL CONFUSION

At least some of children's moral confusion stems from the conduct and attitudes of prominent adults and major social institutions. In recent years, the nation has seen religious leaders and public officials involved in scandals that belie their professed commitment to family values and betray the public's trust. Leading financiers and corporate executives have been prosecuted for enriching themselves at the expense of their clients or shareholders. Rampant materialism among adults fosters shallow ambitions in children and encourages them in empty, reckless, and sometimes dangerous pursuits. The media and entertainment industries glamorize drugs, sex, greed, and violence through movies, television, and music, and in the personal lives of some popular entertainers and athletes.

There are also disturbing indications that a growing number of mothers and fathers lack both the ability and the commitment to be responsible parents. Profound social and economic changes in the past two decades have fundamentally altered the roles and relationships of many clients and children, as well as the routines of family life. Some of these changes have had troubling consequences. More children today grow up without the consistent presence of a father in their lives. Working parents, even in two-parent families, find it difficult to spend as much time with their children as they would like and their children need. A higher percentage of unmarried teenagers give birth today than in decades past, and these young mothers often lack the maturity, economic means, and parenting skills to care for themselves and their children.

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Epidemiology

Trends in the prevalence of dental caries in Israel

Dan Zadik, DMD, MPH
Arkadi Deitsch, DMD
Dov Tamir, MD, MPH
Moshe A. Kelman, BDS, DDH

The WHO, European region, has determined dental goals for the program "Health for all for the year 2000".[1] These goals include: 50 percent of children at the age of five to six to be caries free, and a mean DMF (T) of 3 at the age of twelve to thirteen.[2]

Many studies have been conducted in Israel since 1955, some of which related to the relevant age-groups (Tables 1 and 2). Most of these studies were carried out, however, on selected groups. The only study that was representative for five-year-olds was done in 1971 by Zadik.[7]

The present study could be of particular interest, because it relates to the same age-group in the same city after a period of seventeen years. The findings of this study may help to determine the specific goals, for the year 2000, for the entire population of Israel. The new water-fluoridation plant started operating in Jerusalem at the time of the study, and the data collected may thus serve as a baseline for the commencement of fluoridation.

The objective of this study was to assess the prevalence of dental caries in five-year-olds and twelve-year-olds in Jerusalem.

METHODS AND MATERIALS

This study was part of a nationwide study carried out in 1988 in West Jerusalem. A total of 166 children, five to six year-olds; and 147 children, twelve to thirteen year-olds, were selected by a previously described method using a stratified random cluster sample, to form a representative sample of the relevant age-groups for the city in this year.[15] The examinations took place in the classrooms under natural lighting with the aid of a mouth mirror and a probe. Bitewing radiographs were not taken.[7] Criteria for dental caries were those described by WHO.[16] All examinations were performed by one of the authors (A.D.), after pretest for self-calibration to minimize intraobserver variations. About 25 percent of the children were examined simultaneously and independently by the principal author (D.Z.), who is conducting the nationwide survey, to minimize interobserver variations.

RESULTS

It was found that 27.7 percent of the children at the age of five to six years were caries-free, with a mean

Dr. Zadik is Senior lecturer in pedodontics; Dr. Tamir is Director of the Health Department, Jerusalem municipality; Dr. Kelman is Director of Dental Health, Ministry of Health, Israel, from the Department of Community Dentistry, Hadassah-Hebrew University Faculty of Dental Medicine.

dif(t) value of 3.65 ± 3.58 (Table 3). The major contribution to this value was decayed teeth (d). Thirty-six percent of the carious teeth had been treated. Fifty-five percent of the children with caries had not been treated. No difference was found between males and females (X^2 test). At the age of twelve to thirteen years, only 4.8 percent of the children were caries free (Table 4) with mean DMF (T) value of 4.9 ± 3.09. Fifty-seven and a half percent of the carious permanent teeth were treated. Forty-two percent of the children with caries had not been treated. No difference was found between males and females (X^2 test).

DISCUSSION

There is a problem in trying to compare findings related to the prevalence of dental caries, even in the same country. The quoted studies since 1955 were conducted by different investigators, sometimes using different criteria, and performed under different physical conditions. The selectivity of most of the population groups studied, also limits comparisons. The only meaningful comparison of the findings of this study is to the findings of a previous study conducted by the author.[7] An increase in the percent of caries-free children at the age of five from 16 percent to 27.7 percent, and a decrease of mean dif (t) values from 4.7 to 3.65 was noted. This might be of particular interest, since it is the first decrease in the prevalence of dental caries, reported in Israel.

The year of 1982 was a turning point, when a marked decrease in the prevalence of dental caries was reported in the industrialized countries.[17] Since then, reports on decrease are rather common. The debate on the reasons for that decrease, continues. Most authors, however, attribute it to the use of fluorides in all forms and to the increase of awareness to prevention methods.[18,19]

The addition of fluoride to the Jerusalem water supply was only recently made.

In Jerusalem, fluoridation of the water supply was implemented only recently and the observed reductions cannot be attributed to it. A possible indicator of the increase of awareness in the public is the fact that 36 percent of the decayed teeth were treated; while only 4 percent were treated in the previous Jerusalem study.[7]

The relatively decreased value of caries-rates in pri-

Table 1 ☐ Prevalence of caries in primary teeth in Israel based on various studies.

Investigator	Age	No. of children examined	Percent of caries free	Mean dif(t) values
Rosenzweig and Cohen[3]	5	408	23	3.99
Rosenzweig *et al*[4]	5	242	–	1.79
Kenyon and Young[5]	3–5	108	28.7	3.6
Milgalter *et al*[6]	3–5	12	33.3	2.5
Zadik[7]	5	965	16	4.7
Ran and Anaise[8]	6–7	218	14	5.2
Present study	5–6	166	27.7	3.65

Table 2 ☐ Prevalence of caries in permanent teeth in Israel based on various studies.

Investigator	Age	No. of children examined	Percent of caries free	Mean DMF(T) values
Laufer[9]	13	2724	–	2.55
Rosenzweig[10]	13–14	4500	–	2.43
Rosenzweig and Langer[11]	10–14	94	–	3.41
Kenyon and Young[5]	10–14	157	–	1.8
Anaise *et al*[12]	11–14	531	8	4.64
Anaise *et al*[13]	11–12	111	–	3.9
Anaise[14]	11–14	3672	–	3.73
Present study	12–13	147	4.8	4.88

Table 3 ☐ Prevalence of dental caries in five-year-olds.

	No. of children examined	Percent of caries free	Mean values				
			dif(t)	s.d.	d(t)	i(t)	f(t)
Males	69	29.6	3.20	3.14	1.86	0.17	1.17
Females	97	26.5	3.95	3.84	2.10	0.45	1.40
Total	166	27.7	3.65	3.58	2.00	0.34	1.31

Table 4 ☐ Prevalence of dental caries in twelve-year-olds.

	No. of children examined	Percent of caries free	Mean values				
			DMF(T)	s.d.	D(T)	M(T)	F(T)
Males	75	4.48	4.47	3.05	1.96	0.07	2.44
Females	72	5.17	5.31	3.08	2.15	0.04	3.11
Total	147	4.80	4.88	3.09	2.05	0.05	2.77

mary teeth was not demonstrated in the permanent teeth of the twelve-year-olds. The minor percentage of caries-free (4.8 percent) with mean DMF (T) value of almost 5 indicates that first primary molars are affected in almost all children.

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BREAST-FEEDING TRENDS

In this issue of *Pediatrics* the most recent (1989) survey documents the steady decline in breast-feeding across all age groups surveyed since 1984. The greatest decline was among the most vulnerable high-risk mothers whose infants stand the most to gain from being breast-fed. These include the younger, uneducated, underprivileged, and underserved women. The advantages of breast-feeding for their infants are legion.

While this valuable epidemiologic survey brings grim news, let us not "shoot the messenger" but let us examine the message and its meaning. It is not an indictment of the many individuals working in the lactation vineyard who have labored tirelessly to help women on a one-to-one basis to choose breast-feeding and to nurse their infants successfully. The role of the physician has been equally important in encouraging women to nurse their infants. The support systems provided to women in private offices, clinics, and newborn nurseries have helped women establish and maintain lactation. The impact of the recent decline in breast-feeding with respect to infection protection, immunologic implications, and psychosocial advantages can only be estimated at this time.

The demographic factors associated with the greatest decline in breast-feeding cannot be altered by the government or the health care system and include ethnic background, maternal age, prior education level, income, geographic location (ie, north, south, east, west), parity, or year of survey. However, the authors point out some factors that could be modified. They list employment practices, support by friends, the health care system, community groups, the workplace environment, and lactation education. I would add birth weight to the list because of the impact of prenatal care on preventing prematurity.

Lawrence, Ruth A.: Breast-feeding trends:
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CLINIC

Evaluation of fluoride exposures in children

Steven M. Levy, DDS, MPH
Zavash Zarei-M, MS, DDS

The purposes of this paper are to report the findings of a study looking at children's fluoride exposures in detail beyond the fluoride content of home drinking water, and thus reveal the most clinically-relevant questions to be asked of parents, when conducting histories of fluoride exposure of child patients.

INTRODUCTION

In recent years, dental caries in children has decreased dramatically in the United States and other developed countries.[1-4] Many factors likely are responsible for this decline, but the most important factor probably is the widespread use of fluoride in various forms.[3,5-7] Among these are intentional sources of ingested fluoride such as fluoridated drinking water and dietary fluoride supplements. Unintentional sources of systemic fluoride, however, abound, from use of fluoridated water in food processing, use of infant formula, and the ingestion of topical fluoride from toothpastes, mouthrinses and gels.[5] It is now virtually impossible to find "non-fluoride" communities in the United States, because of these frequent "alternative exposures to fluorides."[8]

The prevalence and severity of dental fluorosis in the U.S. may be increasing, because of this widespread ingestion of fluoride.[5] A review by Szpunar and Burt indicated that there is a trend toward increased prevalence of fluorosis of the permanent teeth in the U.S. population. A recent follow-up study by Heifetz *et al* provides additional evidence of a growing prevalence of fluorosis from 1980 to 1985 among children aged eight to fifteen.[9] Kumar *et al* also found evidence of increased prevalence of fluorosis.[10] A recent study reported 81 percent prevalence of (mostly mild) fluorosis in a city with 0.9 to 1.2 ppm fluoride.[11] In the first national study of dental fluorosis in U.S. school children in 1986-87, 22 percent showed signs of fluorosis: 17 percent, very mild; 4 percent, mild; 1 percent, moderate; and 0.3 percent, severe.[12] There were substantial regional variations in prevalence, ranging from 14 percent to 39 percent. Despite this evidence, the suggested increase in dental fluorosis is not as clear-cut, nor as widely accepted, however, as the recent decline in the prevalence of dental caries.[6] Further study is indicated.

The importance of individualizing fluoride therapy to achieve an acceptable balance between prevention of dental caries and risk of dental fluorosis was underscored recently by two events. The U.S. Department of Health and Human Services' Special "Review of Fluoride: Benefits and Risks" was published in February 1991 and called for increased attention, both in dental practice and research, to "using no more than the amount (of fluoride) necessary to achieve" the desired effect.[13] Also, in April 1991, a special workshop supported by the National Institute of Dental Research

Dr. Levy is Associate Professor and Dr. Zarei-M is graduate student in the Department of Preventive and Community Dentistry, College of Dentistry, University of Iowa, Iowa City, IA 52242.

was held to consider the implications of the increased prevalence of fluorosis and make recommendations for changes in the dosages of dietary fluoride supplement.

Sources of fluoride and ingestion

Substantial amounts of fluoride ingested from a variety of sources other than drinking water may contribute to dental fluorosis. Fluids other than tap water that may contribute fluoride include bottled water, fruit juices and ready-to-feed infant formulas, soft drinks processed in fluoridated areas, and tea.[14-22]

Although changes in the manufacturing process of milk-based infant formulas have reduced their fluoride content, soy-based formulas have significantly higher fluoride content.[19,20] Formula base may have a substantial influence, therefore, on total dietary fluoride.

The amount of fluoride ingested from professional topical applications varied from 1.3 to 31.2 mg in different studies.[6,23-28] Substantial ingestion of fluoride has also been shown with fluoride mouthrinse and with self-applied topical fluoride gel.[29-31]

Fluoride ingestion from dentifrices has been assessed with several analytical techniques and substantial variation in results.[32,33] Fluoride ingested per brushing ranged from 0.12 mg to 0.38 mg for the 1 mg F/g toothpaste formulations and is likely adequate for the development of fluorosis, since almost all the ingested fluoride is absorbed.[29,34-37]

Risk factors for dental fluorosis

Although studies consistently have found greater prevalence and severity of dental fluorosis of the permanent teeth to be associated with greater fluoride content of drinking water, the few North American studies of other risk factors for dental fluorosis have had less consistent results. Pendrys *et al* found the use of dietary fluoride supplements in a 1972-75 birth cohort, using a previous dietary fluoride supplementation schedule, to be associated with mild to moderate fluorosis.[38] Leverett *et al* found the use of fluoride supplements in a nonfluoridated community and continuous use of fluoridated toothpaste in a fluoridated community to be associated with more severe fluorosis.[39] In the fluoridated community, breast-feeding was associated with a significantly lower severity of fluorosis. Kumar *et al*, Woolfolk *et al*, and Ismail *et al* found use of dietary fluoride supplements to be associated with more fluorosis.[10,40,41] Szpunar and Burt found more fluorosis to be associated with the use of a fluoride mouthrinse, but not dietary fluoride supplements.[42] Bohaty *et al* reported prevalence of fluorosis in an optimally fluoridated community to be associated independently with the use of dietary fluoride supplements and gels or rinses; but not with toothbrushing frequency.[43] Osuji *et al* found prolonged use of infant formula beyond age one and toothbrushing before age two with a fluoride dentifrice in a fluoridated community to be risk factors for dental fluorosis among eight- to ten-year olds.[44]

Patterns of fluoride exposures

Knowledge about multiple fluoride exposures during the first several years of life is limited. Approximately 54 percent of the U.S. population receives optimally fluoridated water.[45] Data on other fluoride exposures have been collected less systematically. The Dental Care

Fluoride ingested per brushing ranged from 0.12 mg to 0.38 mg for the 1 mg F/g toothpaste formulations and is probably adequate for the development of fluorosis, because almost all ingested fluoride is absorbed.

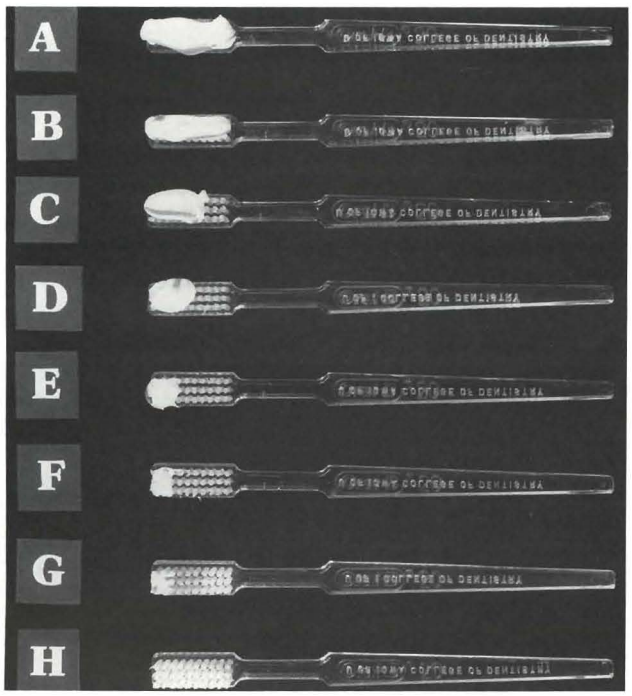

Figure 1. Photograph used by parents to quantify the amount of toothpaste used during the first six years of life.

Supplement of the National Health Interview Survey (NHIS) in 1983 was the first time that this national survey asked about the use of fluoride products.[46] Sixty-eight percent of children under age five were reported to be using fluoride dentifrices, compared with 96 percent of five-to-nine-year-olds and ten-to-fourteen-year-olds. Five percent of those under age five and 17 percent of those age five to seventeen were reportedly using fluoride mouthrinses. Approximately 14 percent of those age three and under, 11 percent of those age four to eleven, and 4 percent of those age twelve to thirteen reported use of fluoride supplements. Whites and those with income above the poverty level were approximately three times more likely to be receiving fluoride supplements. Similar patterns were found in the 1986 NHIS study.[47]

Data from the National Institute of Dental Research's (NIDR) 1986-87 U.S. Children's Survey showed that previous or current use of supplemental or topical fluoride was widespread.[7] Among those children age five to seventeen without access to fluoridated water, 54 percent had used dietary fluoride tablets or drops, 54 percent had received topical fluoride treatment at a dentist's office, and 22 percent had participated in school-based topical fluoride programs. Overall, 71 percent had received at least one form of supplemental or topical fluoride.

Implications of the problem of increasing fluorosis

Because fluorosis may be increasing, we must understand better the relationships between ingestion of fluoride and fluorosis. It is important that our understanding of the role of various fluoride sources in the etiology of dental fluorosis be improved, so that discussions concerning modification of current fluoride use can have a sound scientific base. Otherwise, proven measures for preventing dental caries may be inappropriately discontinued, merely because they may be associated with greater dental fluorosis.

Discussions should have a sound scientific base.

METHODS

After consultation with a number of other investigators who had previously conducted studies on this subject, a fifty-item questionnaire was developed to assess retrospectively the fluoride exposures of children.[7,10,38-40,42,44] The questionnaire was administered to mothers by personal interview at the time of a regularly scheduled dental or medical appointment at the University of Iowa. Sixty-nine children, age five to seventeen years (mean = eight years), from sixty-six different families were studied. The participants were a convenience sample, generally with good health and dental awareness, and higher than average educational levels. Questions addressed exposure to drinking water at home, school, child care site, and other; dietary fluoride supplements; fluoridated dentifrice; fluoride mouthrinse; and professional (dental office) topical fluoride treatments. Test-retest reliability was assessed with mothers of thirty-eight children providing responses about two weeks apart, with overall agreement of 93 percent. Figure 1 shows the photograph used to quantify the amount of toothpaste used during the first six years of life. Parents reported separately the quantities of dentifrice used daily by their child from birth until age two, from two until four, and from four until six. (A full strip of toothpaste would be almost 1.0 gram of toothpaste, which typically contains approximately 1.0 mg of fluoride.)

RESULTS

Water from community water supplies was ingested by 68 percent of the children. Fifty-three percent reported receiving fluoridated water, 35 percent reported using nonfluoridated water, and 11 percent were unsure. Seven percent reported their children drinking primarily bottled water rather than tap water, from birth through age six. Parents reported that 28 percent of the children drank more than 25 percent of their water outside the home, from birth through age six. Among these, the estimated mean percentages of drinking water from birth to six from home, childcare, and school/other were 57 percent, 13 percent, and 30 percent, respectively. Twenty-seven percent reported ever using dietary fluoride supplements with approximately half prescribed by physicians and half by dentists. Forty-seven percent started dietary fluoride supplementation as infants, 18 percent as one- or two-year-olds, 18 percent began when three or four, 12 percent began when five or six, and 6 percent began when seven or older. Nineteen percent were still taking dietary fluoride supplements at the time of the survey, 25 percent has stopped supplementation before age two, 18 percent at ages two or three, and 38 percent from ages four through eight. Children receiving their prescriptions from physicians were more likely to begin and discontinue supplementation at younger ages, compared with those receiving prescriptions from dentists.

During their first year of life, 32 percent were mostly breast-fed; 61 percent, mostly bottle-fed; and 7 percent were equally divided between bottle-fed and breast-fed. Among the bottle-fed infants, more than 90 percent used commercial infant formula. Among those using formula, 48 percent reported use primarily of liquid concentrate, 25 percent used ready-to-feed liquid, 21 percent used powder concentrate, and 7 percent used a combination of types. Twenty-one percent used soy-based formulas and the remainder used milk-based formulas.

Five percent had never visited the dentist, 9 percent first saw the dentist before age two, 59 percent at age two or three, 23 percent at ages four to six, and 5 percent at age seven or older. Fifty-eight percent had received professional (office) topical fluoride treatment(s), with half of those having received four or more treatments. Twenty-two percent reported use (ever) of home fluoride mouthrinses and 21 percent had participated in preschool or school-based mouthrinsing.

Sixty-two percent reported the child's teeth were first brushed before age two; 24 percent were first brushed at age two; and 14 percent, at age three or older. All reported the use of fluoridated dentifrice, with 54 percent beginning such use by age two; 39 percent at age two or three; and only 7 percent, age four or later. Six percent reported that the children brushed their own teeth before age two, while an additional 57 percent reported that the children brushed their own teeth at age two or three. Parents reported that 36 percent of their children brushed twice daily, from age two to six; 54 percent brushed once daily; and 10 percent brushed less than once daily.

Table shows the parents' responses concerning the amount of toothpaste used per brushing by their children until age two, from age two to four, and from four to six. Figure 2 presents the same data as Table 1, using a different format and excluding those who did not know and those with no response. Referring to Figure 2, from birth until age two, 26 percent used a strip of toothpaste a quarter of the length of a toothbrush; 30 percent used half to three-quarters of a strip; and 9 percent used a full strip. When two- and three-year-olds, 44 percent used half to three-quarters of the length

Table ☐ Amount of toothpaste used per brushing* (Percentage of children).

		Age-Groups		
		0-2 yrs	2-4 yrs	4-6 yrs
Toothpaste quantity (using photograph in Figure 1)				
A	Full strip-thick	3	6	9
B	Full strip-thin	6	7	13
C	3/4 strip	9	7	26
D	1/2 strip	20	30	17
E	1/4 strip	25	16	3
F	< 1/4 strip	12	9	4
G	Small amount	14	9	1
H	No paste	7	1	0
	Do not know	4	1	0
	No response	0	13	26

n = 69

*Full strip equals approximately 1.0 g of toothpaste which is typically approximately 1.0 mg of fluoride.

FIGURE 2

Amount of Toothpaste Used by Children at Each Age

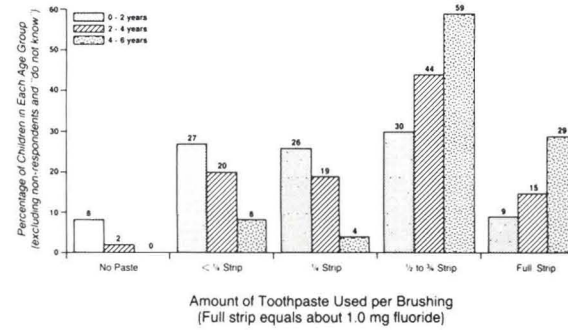

Figure 2. Quantity of toothpaste used by children from birth to 6 years old.

of a toothbrush and 15 percent used a full strip. During ages four and five, almost all children used a quantity of toothpaste equivalent to at least half the toothbrush length.

DISCUSSION

The subjects in this study were a convenience sample and neither randomly selected nor necessarily representative of a larger group. In addition, the retrospective design means that the results are dependent on parents' recall of events that may have happened several years earlier, thus increasing the chance of recall bias. One must be cautious, therefore, in drawing conclusions. As recommended by Szpunar and Burt, personal interviews were conducted in this study, instead of using only self-administered questionnaires, where biases might be greater.[48] This permitted more specific questions to be asked and allowed for verification of the parents' understanding of the meaning of the questions.

Drinking water sources

The children in this study clearly received fluoride from multiple sources, but with substantial variation among individuals. In the current study and in those reviewed previously, almost all children used fluoridated dentifrice, the majority had received professional fluoride treatments, and a minority had used dietary fluoride supplements and fluoride mouthrinses.

Several sources of fluoride were examined in more detail in this study than has been the case in previous studies. One source was drinking water. Thirty-one percent reported that their children drank more than 25 percent of their drinking water outside the home, from birth through age six. Among these 31 percent, sources other than home drinking water provided an average of almost 50 percent of the total water ingested. These findings may be important in determining the need for and dosage of dietary fluoride supplements. Consideration of drinking water sources other than at home is not explicitly part of the current recommendations of the American Academy of Pediatrics and American Dental Association, which focus, respectively, on "the concentration of fluoride in the local water supply" and on "the natural level of fluoride in drinking water where the child lives."[49,50]

With a large proportion of infants and young children in the United States being cared for outside the home, consideration of multiple water sources may be indicated. For example, a two-year-old child living in an area with a low level of fluoride (0.1 ppm) in the water in his home might be spending fifty hours per week at formal or informal daycare served with optimally fluoridated water (1.0 ppm). In that case, provision of a full-dosage supplement likely would place the child at increased risk of dental fluorosis. A weighted averaging of the fluoride levels of multiple water sources would be more appropriate.[51] In the example above, were home and daycare each determined to provide approximately 50 percent of the child's drinking water, the weighted average would be 0.55 ppm and a reduced dosage supplement (0.25 mg instead of 0.50 mg) would be recommended.

Although only 7 percent reported bottled water as the main source of drinking water from birth through age six, there has been a great increase in sales of bottled water in the United States in recent years. Since bottled waters have been found to have variable and sometimes substantial levels of fluoride, it is inappropriate to assume that bottled water has negligible levels of fluoride.[14-16] Fluoride assay of patients' bottled drinking water, therefore, is indicated at this time. In the future perhaps, manufacturers could determine and display on each bottle the fluoride contents of their products.

Another area of interest concerns the use of soy-based infant formulas. More than a fifth used primarily soy-based formulas, which have been shown to have significantly higher fluoride levels than do milk-based formulas.[19,20] This is true because changes in the manufacturing process of milk-based formulas led to a reduction in fluoride content of milk-based products, while soy-based formulas contain components that bind fluoride, thus limiting further fluoride level reductions.[20] The type of formula consumed (soy- vs. milk-based) may become a factor, therefore, in recommending dietary fluoride supplements.[20]

Toothpaste ingestion

In this study, toothbrushing habits and quantities of toothpaste used were investigated in detail. All children were reported to have been using fluoridated dentifrices during the preschool years, while teeth are at risk of dental fluorosis. Eighty-six percent of children were having their teeth brushed (or brushing themselves) before age three. For each two-year period (birth to two, two to four, four to six), children were using substantial quantities of toothpaste, with older children generally using larger quantities than did younger chil-

dren. The most common (and approximate mean and median) quantities used per brushing were one-quarter strip, one-half strip, and three-quarters of a strip for the birth-to-two, two-to-four, and four-to-six age-groups, respectively. Since a quarter-strip of toothpaste would contain almost 0.25 mg of fluoride and with 90 percent brushing at least once daily from age two to six and more than a third brushing at least twice daily, there were substantial quantities of fluoride in the oral cavity. Any child using Extra-Strength Aim® would be exposed to 50 percent more fluoride. Large proportions of the toothpaste may be ingested, especially among young children and those brushing without supervision. In this study, although only 6 percent brushed their own teeth before age two, 63 percent were brushing by themselves before age four. Also, the introduction of "kiddie" toothpastes likely has increased the likelihood of toothpaste ingestion among young children.

Although toothpaste "ingestion" was not quantified in this study, the patterns of toothbrushing, supervision, and toothpaste-use suggest that toothpaste ingestion likely was a substantial source of systemic fluoride for these children, during the years before age six, the years during which there is risk of dental fluorosis. Thus, these data support the conclusions of Beltran and Szpunar that young children may be receiving enough fluoride from ingestion of fluoride toothpaste alone to be at risk of dental fluorosis.[35] Clearly, the risk of fluorosis would be greater for those receiving systemic fluoride from multiple other sources. Beltran and Szpunar recommended the marketing and use of fluoridated toothpaste with lower fluoride content for young children and direct parental instruction and/or supervision of toothbrushing with a very small amount of toothpaste.[35]

Dietary fluoride supplements

Based on the substantial variation in the sources of fluoride identified in this study, it would seem appropriate for dentists and physicians to be advised to be conservative in the prescription of dietary fluoride supplements, to avoid increased risks of dental fluorosis, using them perhaps only with children at high risk of dental caries.[48] Providers probably should inquire about patients' multiple water sources and fluoride levels, including bottled water sources, use of (soy-based) infant formula and method of formulation, and patterns of toothpaste ingestion, before determining need for dietary fluoride supplements. It may be unreasonable, however, to expect this level of detailed inquiry in light of previous studies that documented deficiencies in providers' appropriate use of water fluoride assay with the currently recommended dosage schedule.[52-57]

In addition, it may be appropriate to modify the recommended dosage schedule for dietary fluoride supplementation to reflect the increased availability of systemic fluoride from other sources such as toothpaste. Data from additional studies of fluoride exposures and fluoride ingestion, including longitudinal studies, would be of great value in determining future recommendations.

CONCLUSION

This study has demonstrated the complexity and variability of the sources of fluoride of young children. Although additional longitudinal research is warranted, it is clear that consideration of multiple drinking water sources and fluoride dentifrice usage is indicated before prescription of dietary fluoride supplements, to avoid unnecessary risks of dental fluorosis.

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Oral changes associated with end-stage liver disease and liver transplantation: implications for dental management

W. Kim Seow, BDS, MDSc, DDSc, PhD, FRACDS
R.W. Shepherd, MD, FRACP
T.H. Ong, MBBS, FRACS

In children, chronic liver disease has a variety of causes including extra hepatic biliary atresia, biliary hypoplasia, chronic active hepatitis, postviral hepatitis and some metabolic diseases such as tyrosinemia and α-1 antitrypsin deficiency.[1] Extrahepatic biliary atresia, which affects 1 in 14,000 live births, is a sclerosing process involving the biliary tree. It is the most important cause of death from hepatic failure in children in North America, Europe, Australia, and Japan. The affected children present with neonatal jaundice and although early intervention with portoenterostomy may be successful, failure of the procedure would result in progressive biliary cirrhosis and end-stage liver disease.[2]

Nutrition is affected with general malabsorption of fats and fat soluble Vitamins A, D, E, and K. Other complications include bleeding tendencies due to poor vitamin K absorption and inadequate synthesis of clotting factors and prothrombin in the liver. In addition, osteopenia and rachitic changes may be observed in the skeleton as a result of impaired vitamin D metabolism.[3]

Liver transplantation provides a cure for end-stage liver disease and is now widely accepted as the preferred treatment.[1,2] This is largely due to the fact that the success rates of liver transplantation have improved significantly as a result of advances in surgical techniques, improved postoperative care, as well as the use of cyclosporin as an effective immunosuppressive agent.[4-7] Furthermore, a larger donor pool is now available, as a result of better public education programs, improved preservation technology, as well as development of reduced size donor hepatectomy techniques and selective use of live parent-to-child, partial liver donation.[6,7]

Chronic liver disease in children may have many oral manifestations such as green staining of the teeth and gingiva as well as enamel hypoplasia.[8-10] In addition, the dental management of affected children may be complicated by many factors, such as bleeding tendencies, and inability to metabolize routine anesthetics. Furthermore, patients with liver transplants require life-long immunosuppressive therapy. Although three earlier reports described the oral findings of biliary atresia, to the authors' knowledge there have been no investigations on the effects of liver transplantation on the oral tissues.[8-10] This study investigates the pedodontic management of a group of children with end-stage liver disease with particular reference to the oral manifestations of the disease, as well as the complications associated with liver transplantation.

Dr. Seow is Senior Lecturer in pediatric dentistry, University of Queensland Dental School, Turbot Street, Brisbane, Australia 4000; Dr. Shepherd is Associate Professor, Department of Child Health, University of Queensland; and Dr. Ong is Director of Pediatric Surgery, Royal Children's Hospital, Brisbane.

Table 1 ☐ Oral health of nine patients with end-stage liver disease.

Patient (Sex)	Liver Disease	Age at dental examination	Primary teeth present (n)	Teeth carious (n)	Dental abscesses (n)	Teeth with enamel defects (n)	Dental stain	Dental eruption
(1) MY (F)	EHBA-BC	3y 1mo	20	0	0	20	+ + +	Normal
(2) ROC (F)	EHBA-BC	3y 11mo	20	3	1	20	+	Normal
(3) SY (F)	EHBA-BC	4y 6mo	20	9	6	20	+	Normal
(4) RO (F)	EHBA-BC	3y 11mo	20	20	18	20	+ +	Normal
(5) IT (M)	EHBA-BC	2y 6mo	20	0	0	20	+ +	Normal
(6) MF (F)	EHBA-BC	1y 4mo	4	0	0	0	+	Delayed
(7) KM (F)	EHBA-BC	3y 0mo	20	0	0	20	+	Normal
(8) NO (F)	EHBA-BC	2y 5mo	14	0	0	14	+ + +	Delayed
(9) JS (M)	Alagille syndrome (biliary hypoplasia)	1y 8mo	12	0	0	12	+ + +	Delayed

EHBA-BC: Extrahepatic biliary atresia-biliary cirrhosis

PATIENTS AND METHODS

All patients in the study were referred from the Queensland Liver Transplantation Service at the Royal Children's Hospital, Brisbane. There were seven females and two males. Their mean age at first dental examination was two years, eleven months (range one year, four months to three years, eleven months). Of the nine patients seen, six were dentally examined before, as well as after the liver transplantation, whereas three were examined posttransplantation only.

The dental examinations were performed by one author (WKS) at the University of Queensland Dental School, or at the Royal Children's Hospital, Brisbane. The soft oral tissues were examined to determine green staining and other abnormalities. A mirror and probe were used to detect abnormalities of the teeth. Dental caries was diagnosed using WHO criteria.[11] Enamel hypoplasia was diagnosed, if there was missing enamel or a break in the continuity of the enamel surface.[12,13] Staining of the enamel was noted.

The teeth present were noted and the eruption status compared to normal standards.[14,15] Bitewing radiographs and orthopantographs were taken of children who were cooperative. Extracted teeth from patients SY and RO were photographed. The maxillary left primary canine from patient SY was sectioned longitudinally in a buccolingual direction, using a saw microtome, and hand polished to obtain sections approximately 80 μm thick.

Details of medical and surgical management of the patients were obtained from records kept at the Royal Children's Hospital. All patients were provided with preventive, restorative, and surgical management as needed, at the University Dental School. General anesthesia sessions were performed at the Royal Children's Hospital.

RESULTS

Liver disease

As shown in Table 1, eight patients suffered from biliary cirrhosis resulting from congenital biliary atresia. The ninth patient had biliary hypoplasia as part of the Alagille syndrome, which is characterized by arteriohepatic dysplasia, prominent forehead, hypertelorism and peripheral pulmonary arterial hypoplasia.[16] All patients had previous portoenterostomy procedures that were unsuccessful.[17] Three patients had body weights at or below the third percentile, while two were at the tenth percentile, one at the fiftieth percentile, and one at the ninetieth percentile. With the exception of one patient, all had heights at or below the third percentile.

Oral health

The effects of end-stage liver disease are permanently recorded on the teeth even after successful transplantation. Table 1 shows the oral health of nine patients in the study, illustrating the oral manifestations of end-stage liver disease.

ERUPTION STATUS

The mean age of the patients at dental examination was 2 y, 10 mo (range 1 y, 4 mo to 4 y, 6 mo). Six (66.7 percent) patients had the full primary dentition and normal dental eruption status, whereas three (33.3 per-

The effects of end-stage liver disease are permanently recorded on the teeth.

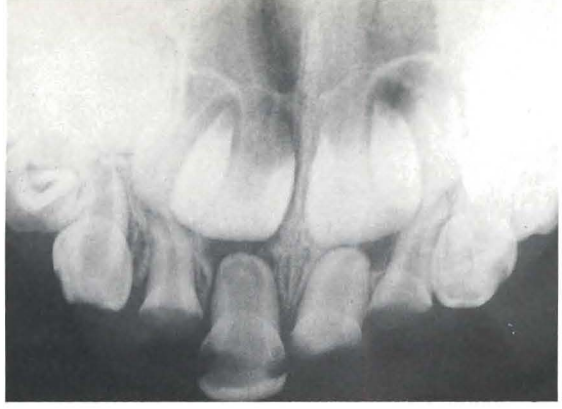

Figure 1. Radiographs of patient RO showing extensive dental decay, as well as large pulp chambers of the teeth.

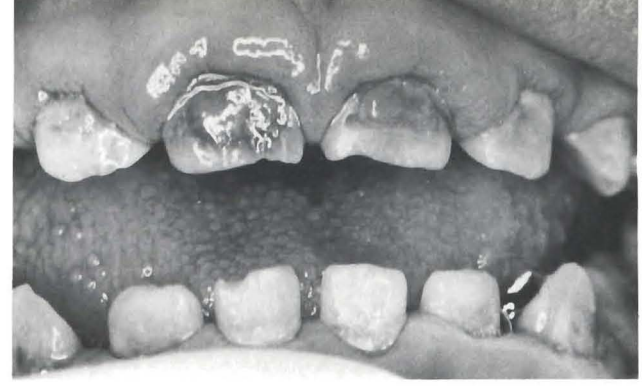

Figure 2. Anterior teeth of patient MY. There was severe enamel hypoplasia which was localized to the cervical half of the teeth. Staining of the teeth was also observed. Gingival hyperplasia associated with cyclosporin intake was clearly evident.

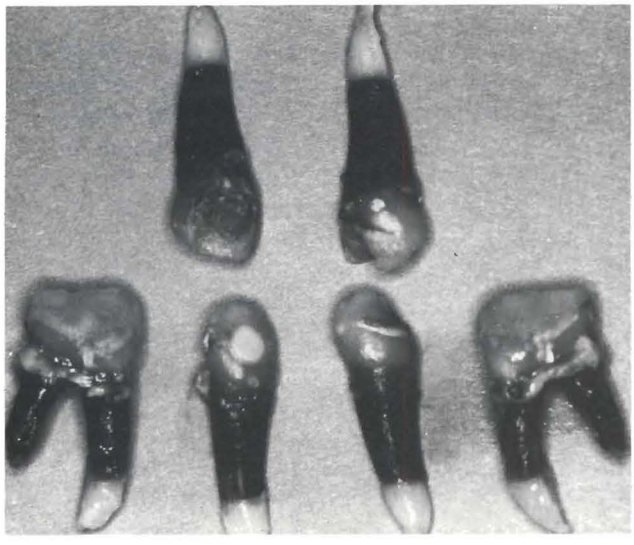

Figure 3. Extracted primary canines and mandibular first molars from patient SY who had her liver transplantation at age 2 y, 6 mo. The deeply stained portions of the roots formed prior to liver transplantation were clearly demarcated from those normally-colored portions formed after transplantation.

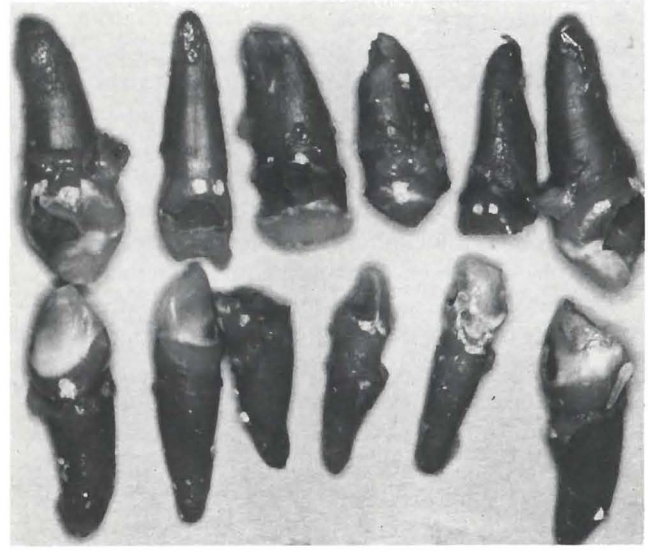

Figure 4. Extracted primary teeth of patient RO showing uniform staining throughout the entire root lengths. Root formation of all the teeth has been completed before her liver transplantation at age 3 y, 10 mo.

cent) showed delayed eruption relative to their respective ages.

DENTAL CARIES

As shown in Table 1, three patients (33.3 percent) had untreated dental decay of at least three teeth. One patient (RO) had all of twenty primary teeth decayed, with eighteen of these associated with dental abscesses. Bitewing radiographs and an occlusograph of this patient (Figure 1) revealed the extent of dental caries, and also demonstrate the large pulp chambers of the teeth.

ENAMEL HYPOPLASIA

In every patient, enamel hypoplasia was noted on all erupted teeth present (Figure 2). The enamel defects ranged from minor breaks in the enamel to large areas of missing enamel, and appeared to be mainly located in those areas of the primary teeth that were formed postnatally.

STAINING OF TEETH

Staining of the crowns of teeth was noted clinically in all the patients. The stains ranged in color from yellowish-brown to deep green, with varying color intensity from patient to patient. Extracted teeth from two patients (RO and SY) provided interesting observations with regard to the dental staining present before and after liver transplantation.

Figure 3 shows four extracted primary canines and the mandibular first primary molars from patient SY, who had her liver transplantation at age 2 y, 6 mo (2 y, 3 mo posttransplant); and Figure 4 shows the extracted primary anterior teeth of patient RO who had the liver transplantation at age 3 y, 10 mo (teeth ex-

It can be expected that dental tissues formed after liver transplantation will be normal.

tracted 2 mo posttransplant). As shown in Figure 3, for patient 3Y the portions of the roots formed prior to transplantation, i.e. the occlusal two-thirds of the canine roots, as well as the occlusal two-thirds of mesial roots of the mandibular first primary molars were deeply stained with green bilirubin pigment. There was a sudden and dramatic change to normal color at the apical thirds of these roots, which were formed after the successful liver transplantation. In contrast, all the roots of the teeth of patient RO (Figure 4) were uniformly stained green, most likely due to the fact that root formation has occurred before liver transplantation.

HISTOLOGICAL APPEARANCE

Figure 5 shows an undecalcified section of the maxillary left canine tooth. A portion of the crown is missing due to removal of a large restoration during processing. Most of the hypoplastic enamel had been removed during the restoration of the crown.

A deeply-stained green band within the dentin that ended exteriorly at the apical third of the root demarcated those parts of the dentin formed pretransplantation from those formed posttransplantation. A few other less distinct bands were also evident within the dentin formed pretransplantation. Furthermore, changes in the number and distribution of dentinal tubules were observed. Dentin formed prior to liver transplantation had a large number of tubules and these were irregularly distributed, whereas that formed after transplantation had fewer tubules that were more regular in distribution. Interglobular dentin, which is often present in rickets, was not observed in this section in spite of the fact that the patient suffered generalized osteoporosis before the liver transplantation.[18,19]

EFFECTS OF LIVER TRANSPLANTATION ON THE GINGIVAL TISSUES

Staining of the gingiva from hyperbilirubinemia is often observed in patients with end-stage liver disease.[8-10] The effects of liver transplantation on gingival staining, however, has not been previously described. Furthermore, although the gingival changes associated with cyclosporin have been described in previous case reports in adults, little information is available on the prevalence and time-course of gingival changes in child patients.

The results of the present study (Table 2) showed that two out of the nine patients (22.2 percent) did not show gingival staining, when they presented for their dental examination at posttransplantation periods of 2 y, 6 mo and 2 y, 1 mo, respectively. In contrast, seven patients showed apparent green staining of the gingival tissues when examined before liver transplantation as well as after transplantation, at posttransplantation pe-

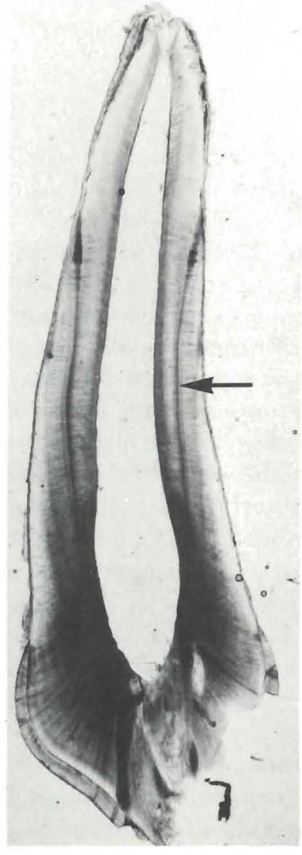

Figure 5. Undecalcified section of the primary maxillary canine tooth from patient SY. A large portion of the crown had been removed during the restoration of the tooth. A deeply stained band within the dentin (arrow), which ended exteriorly at the apical third of the root, demarcated those parts of the dentin formed pretransplantation from those formed posttransplantation. A few other less distinct bands were also evident within the dentin formed pretransplantation.

Table 2 ☐ Effects of liver transplantation on the gingiva.

Patient	Posttransplant period	Serum bilirubin μmol/L		Gingival stain		Gingival hypertrophy	
		Pre	Post*	Pre	Post	Pre	Post
(1) MY	2y 6mo	522	33	NE	–	–	+++
(2) ROC	3mo	249	101	+	+	–	–
(3) SY	2y 1mo	940	23	NE	–	–	++
(4) RO	3mo	234	16	+	+	–	+
(5) IT	1y 3mo	520	16	NE	+	–	++
(6) NO	2mo	334	10	+	+	–	+
(7) JS	1mo	235	16	+	+	–	+

*Standardized or two months posttransplantation except for patients 8 and 9 whose values were taken at one month postoperative.
NE: Not examined

riods ranging from 1 mo to 1 y, 3 mo, despite substantially reduced bilirubin levels.

Table 2 also shows the effects of cyclosporin therapy on the gingival tissues.[20-29] Eight (88.9 percent) patients presented with gingival hypertrophy ranging from mild to severe. The gingival enlargement appeared confined to the free gingival margin and attached gingiva (Figure 2). The degree of gingival enlargement appeared to be directly related to length of posttransplantation period; mild enlargement was observed in patients three months or less after transplantation, whereas moderate to severe enlargement was seen in patients at one or more years after transplantation.

DISCUSSION

The present study confirms and extends the findings of three previous reports that described the oral manifestations of congenital biliary atresia in childhood.[8-10] Green or brown staining of the teeth and gingival tissues by bilirubin pigments is the result of hyperbilirubinemia associated with chronic liver failure. Enamel hypoplasia, often manifested as areas of missing enamel is another common finding. These enamel defects may have been caused by changes in the organic matrix of developing enamel resulting from metabolic disturbances, but are more likely to have resulted from the effects of osteopenia and other disturbances of calcium and phosphate metabolism encountered in chronic liver disease.[3,23] Similarly, enlarged pulp chambers, which is a manifestation of dentin hypoplasia, is the likely result of hepatic rickets.[18,19] Delayed dental eruption most probably reflects the general retardation of growth and development observed in chronic liver disease.

The present study described the oral changes in the gingiva as well in the hard dental tissues of seven children who underwent liver transplantation. Gingival staining was persistent in five of these patients up to a posttransplant period of 1 y, 3 mo, even though the bilirubin levels were substantially reduced after transplantation. In contrast, in two patients, gingival staining was not observed after approximately two years posttransplantation, indicating a significant turnover time of gingival tissues in young children of approximately two years.

The present study has also demonstrated clinically and micromorphologically that while end-stage hepatic disease severely affect dental development, the dental tissues that form after the transplantation is made may be normal.

More importantly, the present study has shown that cyclosporin therapy is significantly associated with gingival hypertrophy of the primary dentition. While previous studies have described the effects of cyclosporin in adult gingival tissues, there have been few reports regarding the primary dentition.[29] In the present study, it was noted that the highest degree of gingival enlargement was observed in the children who had been on cyclosporin for the greatest length of time, although it may appear as early as one month posttreatment. The mechanism of action of cyclosporin is not well understood, but it is thought to interfere with the production and release of interleukin 2 from helper T cells.[24] Histologic studies of affected adult gingiva indicate that it may be a dental plaque-induced lesion characterized by large aggregations of plasma cells, macrophages and helper T lymphocytes; these cells are replaced by noninflamed fibrotic tissue at a later stage.[22]

The medical and surgical management of patients with end-stage liver disease and undergoing transplantation require a well-coordinated team of medical experts. It is now recognized that dentists should also form part of this team to monitor dental health of the patients, because poor oral status may adversely affect the general health of the patients.

While awaiting the liver transplantation, the general health and nutritional status of the patients are carefully monitored.[32] Patients are usually given vitamin supplements and some require total parenteral nutrition in the hospital. After transplantation, the patients are placed on immunosuppressive therapy, usually consisting of corticosteroids, azathioprine and cyclosporin.[1] The side effects of these drugs include increased susceptibility of infections as well as growth disturbances in children, osteoporosis, and moon facies in the case of corticosteroids, and hirsutism and gingival hypertrophy in the case of cyclosporin.[20-29,33]

The systemic complications of the disease as well as transplantation therapy dictate special considerations in the dental management of these patients. Firstly,

oral foci of infection must be eliminated as suppression of the immune system may result in serious systemic spread of oral infections. In this regard, it is disturbing to note the significant amount of untreated dental decay and dental abscesses in the study patients, when they first present for dental examination. The high predisposition to dental caries in these young children may be associated with the frequent feeding that is necessary to compensate for low intestinal absorption of nutrients.[9] Furthermore, enamel hypoplasia predisposes affected teeth to dental decay.[27] Also, oral hygiene levels tend to be low in chronically sick children due to parental preoccupation with other more difficult aspects of care. In the present study, one patient (RO) had complete clearance of her primary dentition, every tooth of which was abscessed. Endodontic treatment of primary teeth was not considered due to the possibilities of chronic infection, even after careful treatment. The patient was supplied with full dentures, which she tolerated well.

In addition, the prevention of oral infection cannot be overemphasized. Expedient restoration of dental caries, as well as the prevention of new lesions through dietary counselling and improved oral hygiene should be instituted as soon as possible. The use of topical applications of high concentrations of fluoride solutions is not advised routinely, because of the compromised liver function. Furthermore, the prevention of periodontal disease through good personal oral hygiene and professional prophylaxis should also be instituted as soon as possible. Good oral hygiene is less likely to lead to gingival hypertrophy associated with cyclosporin therapy.

Secondly, the increased susceptibility of these patients to infection dictates a need for postoperative antibiotic cover for surgical dental procedures. Penicillin is usually the antibiotic of choice for these purposes.

Thirdly, patients with chronic liver disease usually suffer from bleeding diatheses, due to inadequate levels of prothrombin and clotting factors that are produced in the liver, as well as from low levels of vitamin K, which are poorly absorbed from the gut.[2] These deficiencies would need to be corrected before and during all dental surgical procedures, usually through the intravenous infusion of fresh frozen plasma and vitamin K.

Fourthly, the compromise liver function of the patients requires the use of special anesthetic techniques. Isoflurane is a general anesthetic of choice.[1,2]

In conclusion, increased knowledge of the oral complications of chronic liver disease and liver transplantation, as well as their implications in dental management may contribute greatly to the dental care of affected children. To this end, longitudinal studies are necessary to elucidate further the long-term management problems of these special care patients.

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CARIES PREDICTION

The marked decline in total caries prevalence in many industrialized countries may relate to the more general availability of fluoride, predominantly in toothpastes and to some degree in drinking water. It has been suggested by Klock & Krasse (1987) that one of the reasons for the lower caries prevalence may be the lower number of mutans streptococci. They observed a reduced prevalence of caries in 9- to 12-year-old Swedish schoolchildren and a concurrent reduction in the numbers of 'mutans' in their saliva: as the prevalence of dental caries declined from a mean number of open lesions and fillings of 12.8 in 1973 to 3.0 in 1984, so the mean number of mutans per ml of saliva declined from 2,800,000 to 270,000. Increased use of fluorides, they postulate, might at least partly explain these changes in the microflora.

However, in industrialized countries there remains a minority of the population, perhaps some 15-20 percent, who have the majority, some 60 percent or more, of the dental caries. Moreover, members of this high risk group, as individual patients, are often difficult to treat successfully; they are those in whom restorative care alone, or even those basic preventive measures widely used in the community, have not been enough to control the disease. These individuals have a high caries experience in spite of the general availability of fluoride toothpaste and, indeed, some live in areas where there is an optimal concentration of fluoride in the water supply. As Koch (1970) comments, the treatment of children with a high caries activity is often more complicated, and therefore, more time-consuming.

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The etiology, prevalence, and sequelae of infraclusion of primary molars

Joanna Douglass, BDS
Norman Tinanoff, DDS, MS

Dental infraclusion, teeth below the occlusal plane, is a condition frequently found in primary teeth; its etiology and treatment, however, are still controversial. The terms "ankylosed" and "submerged" teeth are synonymous with infraclusion. The most frequently stated cause of infraclusion is ankylosis, the fusion of a tooth root to the surrounding bone. Since ankylosis inhibits normal vertical tooth eruption, affected teeth may appear to "submerge" below the occlusal plane. Other stated etiologies for infraclusion include disturbed local metabolism, gaps in the periodontal membrane, local mechanical trauma, localized infection, chemical or thermal irritation, local failure of bone growth, and abnormal pressure from the tongue.[1-4]

PREVALENCE AND PROGRESSION

The reported prevalence of infracluded primary molars ranges from 1.3 -8.9 percent (Table 1). Such large variation may be due to different diagnostic criteria, ages of children examined, and different study populations. A significantly higher incidence of infraclusion has been reported in siblings of children diagnosed with infraclusion. One study showed that 18.1 percent of children with affected siblings had infraclusion compared with a control prevalence of 8.9 percent.[9] Another showed that the prevalence in siblings was 44 percent compared with controls of 1.3 percent.

Most reports show that mandibular first primary molars are the most frequently affected, followed by second mandibular molars, first maxillary molars and second maxillary molars.[3,6,9] An earlier study, however, reported that second mandibular primary molars were the most commonly affected teeth (Table 2).[7]

Several studies have shown that the peak prevalence of primary molar infraclusion occurs around ages eight to nine years (Figure).[3,6,9] This peak may be artificially low because many infracluded teeth are extracted. If such teeth were left *in situ*, it might be expected that the prevalence would continue to increase until the point of natural exfoliation.

The majority of subjects with infraclusion show progression of tooth submergence with time.[1,2,4,5,10] The progression has been correlated with age in some subjects but not in others.[1,5,10] In some cases infraclusion remains static or even decreases.[1,4] Maxillary molar infraclusion reportedly progresses faster than mandibular molar infraclusion.[1,2]

SEQUELAE OF INFRACLUSION

Conventional dental wisdom asserts that infraclusion delays exfoliation of primary teeth and consequently necessitates extraction to prevent detrimental sequelae. Specific data on this subject, however, are only found in a few papers.[1,2,5,6,10,11]

Exfoliation of infracluded teeth has been reported to be within normal age limits, when compared with either

Dr. Douglass is Fellow and Dr. Tinanoff is Professor, Department of Pediatric Dentistry, University of Connecticut Health Center, Farmington, CT 06030.

Table 1 ☐ Reported prevalence of infracluded primary molars.

Author	Year	Country	Race	Number	Age	Prevalence	Diagnostic criteria
Kurol et al[5]	1985	Sweden	Caucasian	1059	3-12yrs	8.9	Tooth >1mm below occlusal plane.
Krakowiak[3]	1978	USA	Mixed	2234	6-12yrs	3.7	Tooth apical to occlusal plane. Tooth not in occlusal contact.
Brearly et al[6]	1973	USA	Caucasian	1641	3-15yrs	6.9	Tooth >1mm below occlusal plane. Tooth immobile.
Lamb et al[7]	1968	USA	Unspecified	2105	8-12yrs	3.2	Unspecified.
Via[5]	1964	USA	Unspecified	2342	Unspecified	1.3	Tooth >1mm below occlusal plane. Tooth not in occlusal contact.

Table 2 ☐ Reported distribution of infracluded primary molars.

				Percentage of total infracluded molars			
Author	Year	Number of subjects	Number of affected molars	1st Mand.	2nd Mand.	1st Max.	2nd Max.
Kurol[9]	1981	94	172	61%	34%	3%	2%
Krakowiak[3]	1978	82	133	65%	23%	6%	5%
Brearly et al[6]	1973	113	191	69%	19%	8%	4%
Lamb et al[7]	1968	68	130	32%	59%	5%	5%

a contralateral control or normal population values.[1,5] There is a tendency toward a delay of six months, however, on the infracluded side (Table 3). The degree of infraclusion does not appear to correlate with exfoliation time. Of 194 infracluded teeth observed in one study, only five teeth (all maxillary molars) in three subjects were extracted due to unusually severe infraclusion. The remaining infracluded teeth reportedly exfoliated normally.[1]

Another study of 116 teeth in forty-six subjects reported that infracluded mandibular first molars generally exfoliated on time, while mandibular second molars did not. These findings are weak; 37 percent of second molars were extracted by private dentists, 12 percent had no successors, 39 percent exfoliated on time, and only 12 percent were counted as delayed. Furthermore, teeth that were diagnosed as delayed in exfoliation were immediately extracted, so the actual delay in exfoliation was not determined.[2]

Radiographic studies have shown comparable root resorption between infracluded and unaffected teeth, and also have shown no difference in root formation in successors of infracluded and unaffected teeth.[1,5,6,11]

The reported evidence thus suggests that there may be a short delay in exfoliation of infracluded primary teeth, even though they will exfoliate naturally. In contrast, infracluded primary teeth with no successors reportedly do not exfoliate.[2,10]

Retained roots are reported as being a sequela of infracluded primary molars.[1,2,5] In a study of twenty-three pairs of infracluded teeth, Kurol and Koch found ten retained roots on the extraction side and ten on the contralateral side that were left to exfoliate naturally.[5] Another study showed the same number of root fragments remained after infracluded or unaffected teeth exfoliated naturally. No occlusal abnormalities have been reported as a result of these unresorbed roots.[1] There is no evidence in the dental literature that retained roots, therefore, are a detrimental side effect of infraclusion.

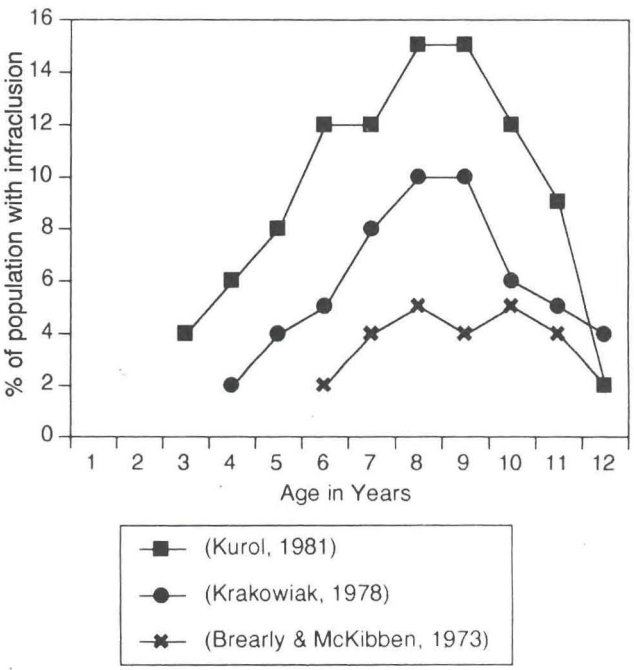

Figure. Prevalence of infracluded primary molars by age.

Infracluded molars have been associated with decreases and increases in arch-length. Decreased arch-

Table 3 □ Exfoliation age (years) of infracluded and unaffected primary molars.

Tooth Type	Normal teeth	Unilaterally infra-cluded teeth	Bilaterally infra-cluded teeth
1st. max. molar	11.2 (3)	11.9 (3)	11.1 (8)
2nd. max. molar	12.1 (10)	12.5 (10)	12.8 (14)
1st. mand. molar	10.9 (4)	11.4 (4)	11.0 (44)
2nd. mand. molar	12.1 (9)	12.5 (9)	12.2 (56)

( ) = Number of teeth in the sample.

length has been shown to occur in 28 to 43 percent of subjects with infracluded mandibular molars.[1,2,5,6,7,10] Mandibular second molars are most commonly associated with the space loss.[2,6] Increasing severity of infraclusion, with loss of marginal ridge contact may increase the likelihood of space loss and subsequent molar tipping.[1,6,10] One comprehensive report states that the space loss is temporary and will self-correct with eruption of the premolars.[1]

Abnormal position and abnormal development of the permanent successors of infracluded teeth have been cited as additional detrimental sequelae. One study found that twenty-five successors from 149 infracluded teeth were tipped "slightly" according to radiographic criteria. These twenty-five teeth, however, were the successors of more severely infracluded teeth.[1] Another study found five successors of 131 infracluded first mandibular molars and seven successors of thirty-seven infracluded second mandibular molars exhibited radiographic tipping.[6] Studies have also reported increased premolar rotations with affected first mandibular molars compared with controls.[2] The successors of infracluded teeth have been shown to have normal vertical relationships and normal alveolar bone height.[1,2,5]

CONCLUSION

Dental infraclusion is a common condition, especially in mandibular primary molars, with more than seventy references in the literature. Most claim infracluded molars do not exfoliate within normal time limits and must be extracted to prevent detrimental sequelae. The few documented studies on this topic suggest, however, that most infracluded teeth exfoliate normally, albeit with a six-month delay. The frequent sequelae of severely infracluded teeth, space loss and molar tipping, may be prevented by either restoring the height of the affected tooth or by using space maintainers without extraction. The successors of infracluded molars reportedly develop normally and have few occlusal abnormalities.

The peak prevalence of primary molar infraclusion occurs at age eight to nine years.

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Treatment of a pseudo-class III relationship in the primary dentition: a case history

Stephen E. Grimm III, DDS

When crossbites or other occlusal discrepancies exist in the primary dentition, should interceptive therapy be contemplated? Or should it be deferred until the mixed dentition, when the permanent first molars and several succedaneous teeth have erupted? This question of optimal timing has been addressed by many authors directly or indirectly.[1-8] Posterior crossbites and pseudo-class III relationships of the primary dentition should be corrected once noted clinically, if the child is cooperative. The foundation of the occlusion is the normal development of the primary dentition.[1,2,7,8] If dentoalveolar deviations are not rectified at an early age, they may develop into skeletal discrepancies.[1,2,7,8] Crossbites will not improve with the eruption of the succedaneous teeth and the crossbite relationship of the primary teeth will be mimicked in the permanent dentition.[7] Correction of the primary dentition can be accomplished by removable or fixed appliances. Removable acrylic appliances, such as Schwartz plates, usually incorporated a hyrax expander to modify actively the denture base width by splitting the midpalatal suture.[1,5,6] Crozats can be used either as a fixed or removable appliance, to expand the dental arch width, similar to the popular Porter or "W" appliance, or the Quadhelix expander; the latter two being banded and fixed.[1,5,6] With varying forces, these orthodontic appliances can cause sutural expansion and concurrent mandibular arch "growth".[4] Frey and Full illustrated the use of edgewise appliances on the primary anterior teeth to correct a pseudo-class III relationship after correcting the existing posterior crossbite with "W" arch.[3] In similar circumstances, Kutin and Hawes feel that the anterior relationship should be corrected before the posterior discrepancy, to prevent overcorrection.[7] Whatever pathway is chosen, a practitioner must thoroughly examine all diagnostic records before proceeding with his treatment. Especially with pseudo-class III relationships, a cephalometric radiograph is essential for revealing any skeletal problems. Extraoral photographs can demonstrate subtle changes in the facial profile. Study models can show arch size, symmetry, and can be sectioned, if duplicated, through the posterior molar region to demonstrate the axial inclination of the molars (i.e. Curve of Wilson). Radiographs, such as the panoral, can highlight the chronological development of teeth, and provide an opportunity to survey the maxillary complex. Some elements of the orthodontic workup may not be obtainable, when working with a preschooler, and may be a determent to cooperation later; nor may they be practical, if the problem is a localized discrepancy. This case report is intended to illustrate a clinical technique and to stimulate thoughts on appropriate documentation and diagnosis.

CASE REPORT

In October 1984, the comprehensive examination of a 3.5-year-old Black female revealed an anterior and pos-

Dr. Grimm is Assistant Professor, Department of Pediatric Dentistry, Howard University College of Dentistry.

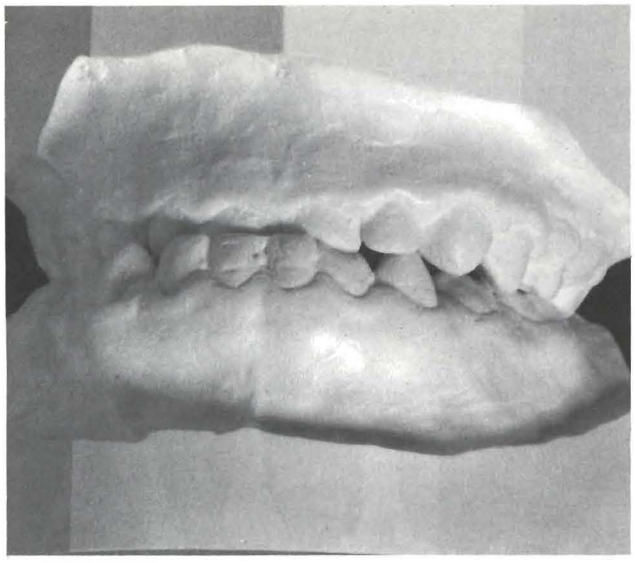

Figure 1. Frontal view in occlusion.

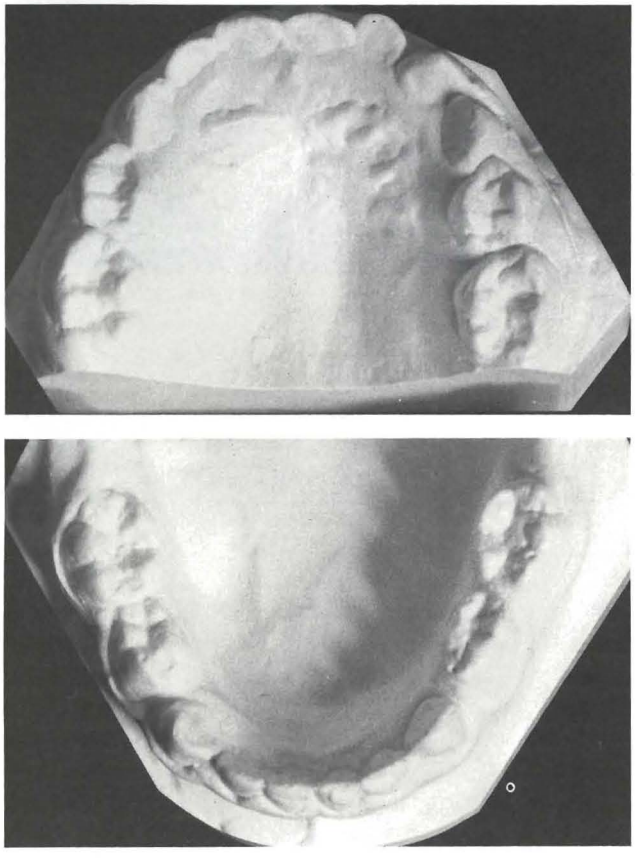

Figure 2. Maxillary and mandibular occlusal views.

terior crossbite that included the maxillary left primary lateral incisor to the maxillary right primary second molar (Figure 1). An edge-to-edge incisal relationship with premature eruption of the canines could be demonstrated upon closure in the terminal hinge axis (centric relation). The count of the primary teeth revealed the presence of a supernumerary left maxillary canine (Figure 2). An occlusal radiograph of the maxillary anterior region showed a crypt of a developing permanent supernumerary left canine, later confirmed on the pan-

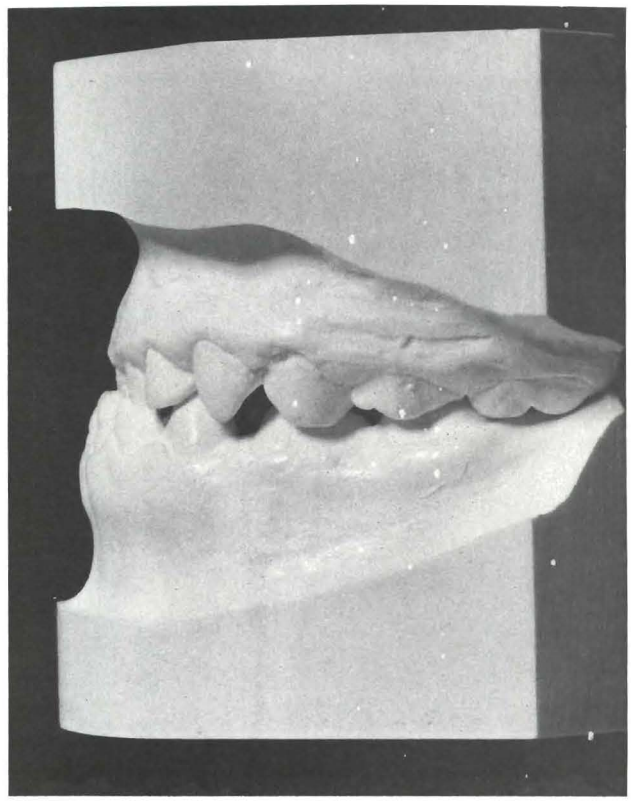

Figure 3. Left buccal view.

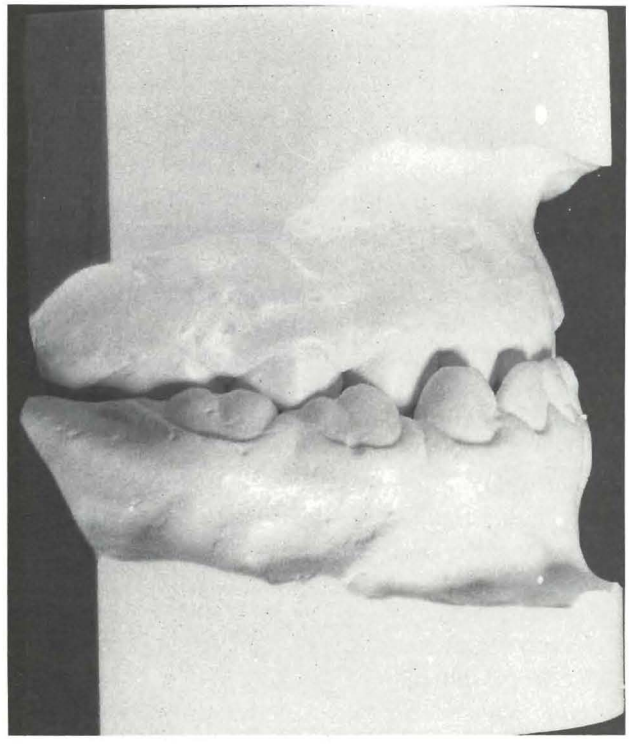

Figure 4. Right buccal view.

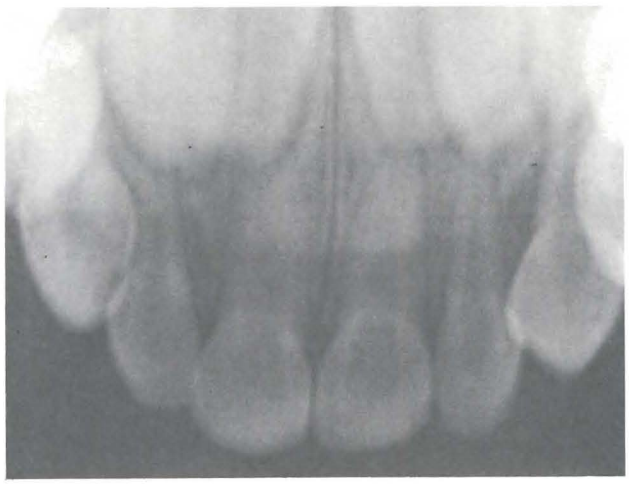

Figure 5. Occlusal radiograph of maxillary anterior segment.

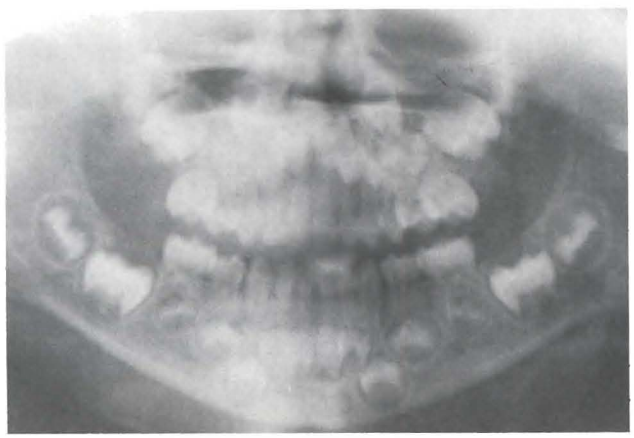

Figure 6. Panoral radiograph.

oramic radiograph (Figures 5,6). In addition, it showed that there was no physiologic or pathologic root resorption of the primary incisors. The initial recommendation was to correct the crossbites after the child had been acclimated to the dental environment by performing routine preventive and restorative procedures, coincidental with the four-month recall.

In March 1985, panoramic and cephalometric radiographs with study models were taken as case documentation (Figures 1-7). A compilation of the findings follows:

Molar relationship Mesial step (noting that the presence of a supernumerary primary left canine within the arch circumference caused an aberrant relationship on the affected side.

Crossbite A to G

Overjet Negative

Overbite Minimal

Centric Relation Accommodated centric occlusion due to canine prematurity and an edge-to-edge, incisal relationship.

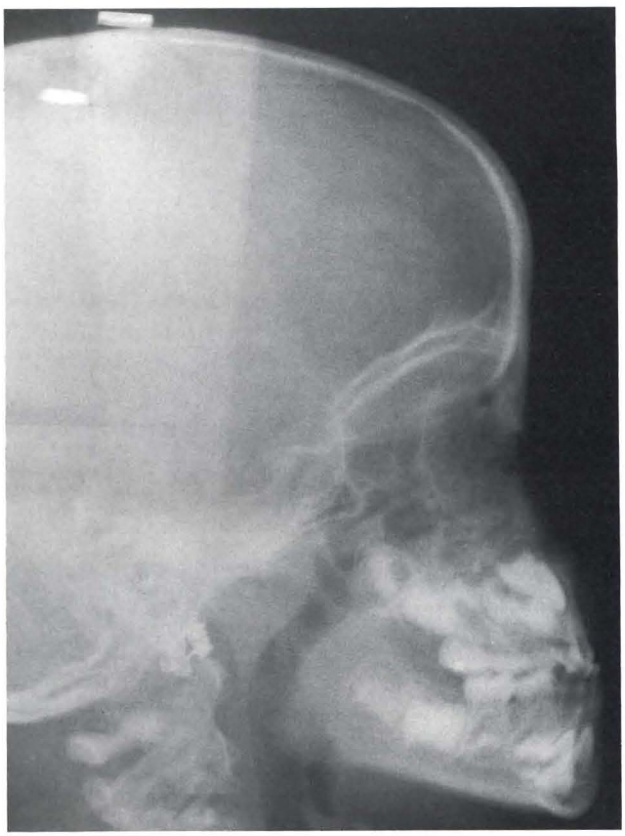

Figure 7. Cephalometric radiograph.

CEPHALOMETRIC ANALYSIS (IN MAXIMUM CUSPATION-Co)[10]

	PATIENT'S ACTUAL	NORM. (PERM.) -BLACK	NORM. (PRIM.) -BROADBENT*	NORM. (PRIM.) -VANN/FEMALE*
SNA	84.5	85.5	83.1	83.0
SNB	84.25	81.0	77.3	78.2
ANB	+.25	4.5	4.0	4.8
⊥ to NA	6°	23°	—	—
⊥ to NA	-.5mm	7mm	—	—
T to NB	17°	34°	—	—
T to NB	+4mm	+10mm	—	—
⊥ to T	154°	119°	152.2°	151.5°
OCC/SN	20°	16°	18.9°	20.0°
MP/SN	32.5°	32.5°	—	35.2°
Y-AXIS	63°	—	57.9°	58.5°
T to MP	82.5°	—	85.5°	84.2°

*Patient population primarily Caucasian

Radiographic Analyses

☐ Panoramic: presence of supernumerary left maxillary primary and permanent canines.

☐ Cephalometric: see Chart above.

Our treatment goal was to produce a normal anterior overbite and overjet relationship with an acceptable posterior occlusion. After banding the maxillary primary second molars, a modified Quadhelix with re-

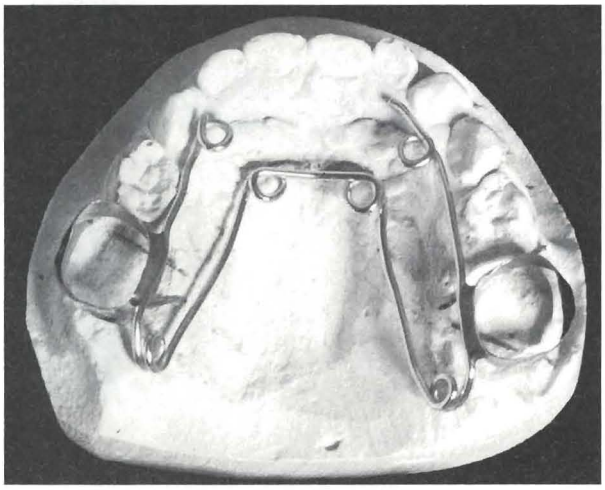

Figure 8. Top: Second generation used in report.

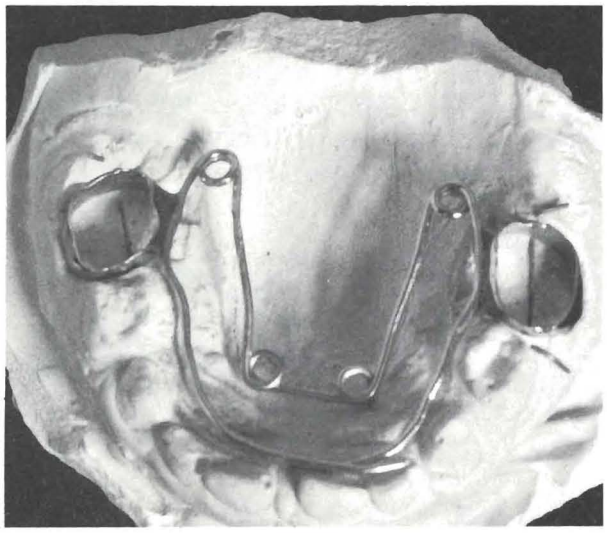

Figure 8. Bottom: First generation used in prior patients.

curved anterior extensions was fabricated, using .036 wire (Figure 8, top). The appliance was activated and cemented with durelon. Oral hygiene and dietary instructions were given to the parent and patient. Every three weeks, the expander was checked for irritation, and activated. In eight weeks, the anterior crossbite was corrected. On the following appointment, four weeks later, the posterior relationship was found to be acceptable. The anterior extensions were removed from the appliance and the posterior Quadhelix was recemented with zinc phosphate for three months, serving as passive retention of the posterior correction. Post-treatment study models were made at this time (Figures 9-12). The occlusion has been stable to present.

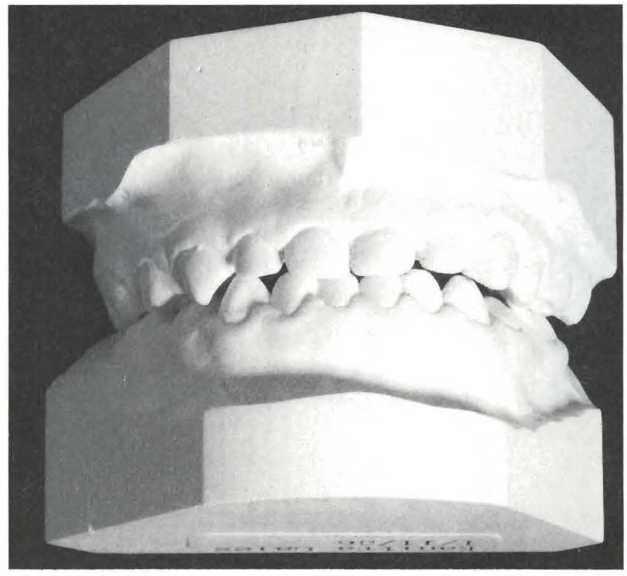

Figure 9. Frontal view in occlusion.

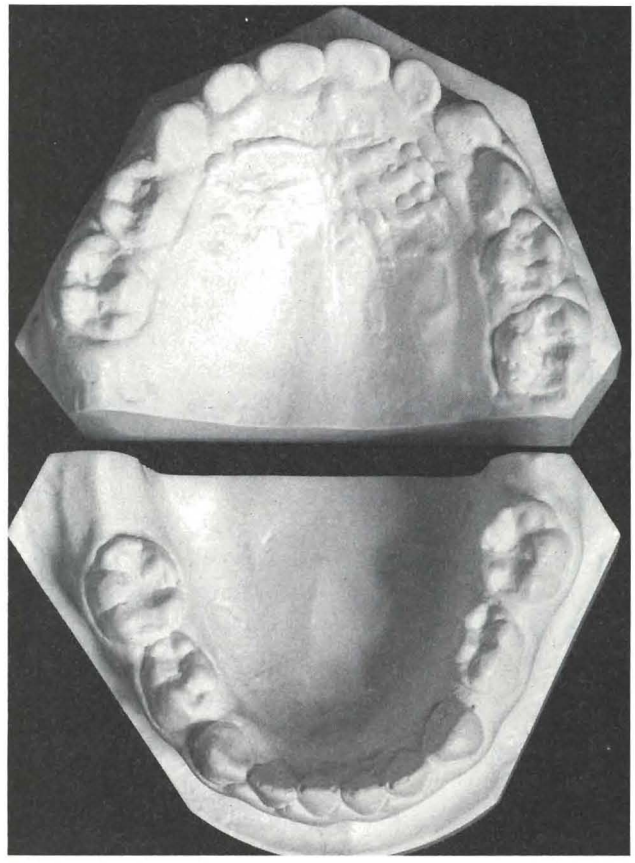

Figure 10. Maxillary and mandibular occlusal views.

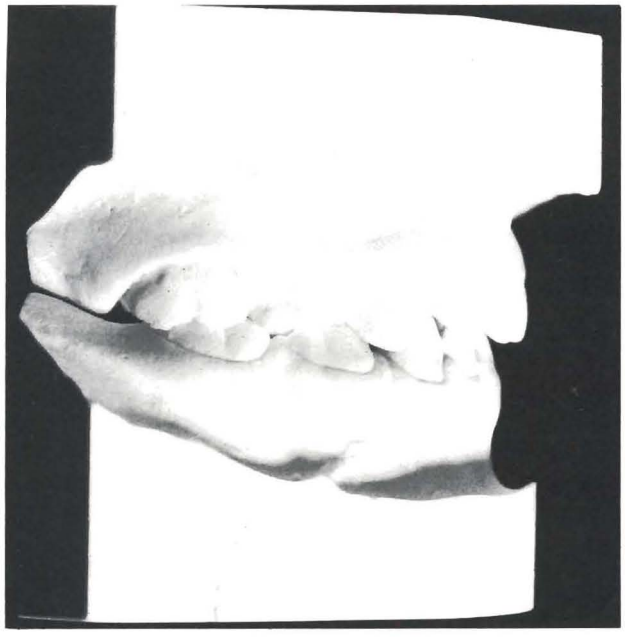

Figure 11. Right buccal view.

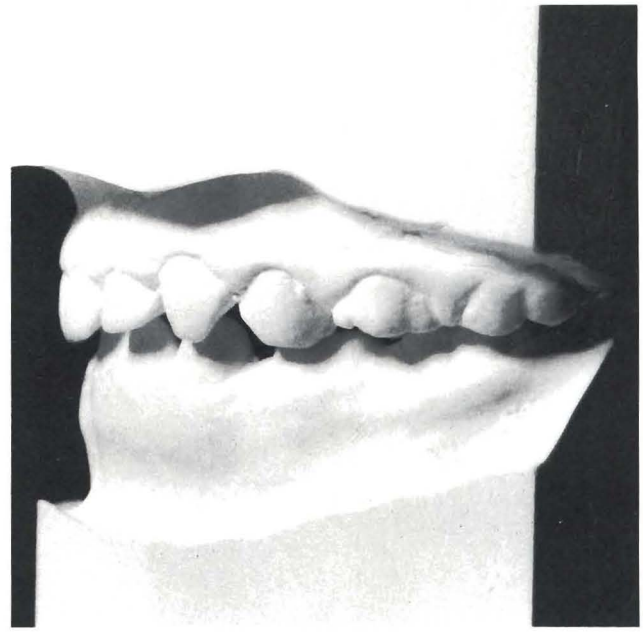

Figure 12. Left buccal view.

DISCUSSION

Before active treatment of posterior crossbites begins, the operant etiology of denture base or dental arch constriction must be eliminated; especially when attributable to digital or pacifier habits. Hypothetically, with the eruption of the primary canines into a tip-to-tip prematurity, selective equilibration may redirect the eruptive path of the canines, if the discrepancy is minor. Other than serving as diagnostic records, study models can be duplicated to be used as working casts, to determine whether equilibration may lessen the effects of the prematurity. Sectioning the models through the posterior heel region and observing the palatal inclination of the molars can indicate whether a Porter appliance is sufficient to correct the abnormal axial tilt of the molars or whether the apparent constriction requires sutural expansion to correct the denture base width, not tipping when the axial inclination is normal. Appliances for the latter would include the Hyrax or Quadhelix expanders. Porter or "W" appliances invite relapse, when tipping is not indicated. When treating anterior discrepancies of the primary dentition and advanced root resorption of the primary incisors is denoted radiographically, the practitioner should delay treatment until the eruption of the permanent successors.

SUMMARY

In conclusion, posterior and anterior crossbites of the primary dentition should be corrected, once recognized, to allow for normal dental base development and subsequent favorable skeletal growth, provided the analysis of the case reveals no gross boney discrepancies. To quiet any archaic ideas of delaying posterior crossbite correction until the eruption of the permanent first molars, a pilot is being undertaken to determine whether there is a statistical difference in the need for retreatment of patients, when the permanent first molars erupt with the use of either the Porter, Quadhelix, or R.P.E.-type appliances. It is hoped that this will support the low retreatment frequency of 7.6 percent reported by Kutin and Hawes.

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CASE REPORTS

Local anesthetic mortality: Report of case

Elliot V. Hersh, DMD, MS, PhD
Mark L. Helpin, DMD
Owen B. Evans, MD

Local anesthetic morbidity and mortality are rare events in modern dental practice. However, case reports of pediatric dental patients receiving fatal or near fatal overdoses of local anesthetic drugs still appear in the dental literature.[1-3]

CASE REPORT

A healthy five-year-old-female patient, weighing thirty-six pounds (16.4 kg), was scheduled for multiple extractions. The child received nitrous oxide-oxygen analgesia (concentration unknown) via a nasal mask at 11:05 AM, followed by maxillary and mandibular injections of five cartridges (9 ml) of 3 percent mepivacaine (Carbocaine) at 11:10 AM. According to the operator, the child appeared "sleepy" after the injections, yet she was able to open up her eyes when the extractions were begun five minutes later. At 11:20 AM the child experienced a "stiffening and shaking" of all extremities that lasted approximately ten seconds. Shortly thereafter she exhibited another convulsive episode, lasting approximately thirty seconds. The nitrous oxide was then discontinued and the patient was placed on 100 percent oxygen. Following a third convulsive episode the child was unresponsive to verbal commands, but the operator reported the patient was still breathing spontaneously. At 11:30 AM the patient was transported to a nearby physician's office and upon arrival was found to be in full cardiopulmonary arrest. Cardiopulmonary resuscitation was initiated and the child was transported to a local hospital by ambulance. She was converted to a regular sinus rhythm with sodium bicarbonate, epinephrine, calcium gluconate and atropine in appropriate intravenous doses at approximately 12:05 PM. The patient experienced another seizure shortly thereafter and subsequently received intravenous phenobarbital, 130 mg; phenytoin, 350 mg; and diazepam, 4 mg, to control the convulsions. Dexamethasone, 8 mg, to reduce intracranial pressure and ampicillin, 500 mg, to prevent pneumonia were also given.

The patient was transferred to a University Hospital by ambulance, where she was intubated due to a lack of spontaneous respirations. Approximately four and a half hours after the dental injections, a urine toxicology screen was positive only for mepivacaine and blood levels of mepivacaine measured 1.35 μg/ml at this time.

Intracranial pressure was monitored and remained elevated for four days, despite treatment with intravenous dexamethasone and mannitol. Although aggressive intravenous antibiotic therapy was instituted, body temperature rose to 104°F, consistent with the

Dr. Hersh is Director, Pharmacology and Therapeutics and Dr. Helpin is Director, Pediatric Dentistry, University of Pennsylvania, School of Dental Medicine, 4001 Spruce Street, Philadelphia, PA 19104-6003. Dr. Evans is Chairman, Department of Pediatrics, University of Mississippi Medical Center, Jackson, Mississippi 39216.

The predominant toxic reactions to local anesthetics appear in the central nervous system and cardiovascular system.

development of pneumonia. A brain flow study performed in the afternoon of the fourth hospital day revealed an absence of cerebral blood flow. Brain death was declared, and the heart stopped beating four hours later. Death was judged to be caused by anoxic brain injury, secondary to cardiopulmonary arrest following an overdose of mepivacaine.

DISCUSSION

This case report describes the tragic sequelae that occurred in a five-year-old-female dental patient, following the administration of five cartridges of 3 percent mepivacaine. With a 3 percent solution there are 30 mg/ml or 54 mg of local anesthetic in each cartridge. Thus a total dose of 270 mg was injected into this patient over approximately a five-minute span. According to Malamed, the maximum safe dose of either lidocaine or mepivacaine is 2 mg/lb body weight.[4] For this particular thirty-six-pound child, this would translate into a maximum safe dose of only 72 mg, or the amount of mepivacaine contained in slightly less than 1.5 cartridges of a 3 percent solution. Thus in this case, the maximum safe dose was exceeded by more than three fold. A retrospective morbidity and mortality study by Goodson and Moore concerning pediatric dental patients who had received local anesthetic and narcotic sedative agents revealed that when the total dose of local anesthetic alone, or the combined doses of local anesthetic plus narcotic analgesic exceeded the maximum safe dose by three fold (300 percent) or more, permanent brain-damage or death always resulted.[2]

Some clinicians, when treating young children, prefer to employ 3 percent mepivacaine or 4 percent prilocaine (Citanest) solutions without vasoconstrictors instead of 2 percent lidocaine with 1:100,000 epinephrine, based on their assumption that the two former will result in significantly less lip, cheek and tongue mutilation due to their much shorter durations of action. In fact, while pulpal anesthesia is relatively short-lived with 3 percent mepivacaine or 4 percent prilocaine, soft tissue anesthesia may still last three to four hours after mandibular block injection with these agents (Table).[3,5,6]

Maximum dosage limits are quickly reached with mepivacaine or prilocaine in young patients. While the maximum safe dose based on mg/lb of lidocaine and mepivacaine are the same, one cartridge of 3 percent mepivacaine (54 mg) contains 1.5 times the dose of local anesthetic than 2 percent lidocaine (36 mg). While prilocaine's maximum safe dose is 33 percent greater than that of mepivacaine, because of its higher concentration (4 percent), the amount of local anesthetic in the cartridge (72 mg) is also increased by 33 percent, thus offering no safety advantage.[4,5] Two cartridges of 3 percent mepivacaine (108 mg) or 4 percent prilocaine (144 mg) exceed the maximum recommended doses in a thirty-six-pound child. Inattention to such limits when using mepivacaine has led to a number of previous cases of morbidity and mortality.[7-9]

Table □ Comparison of 3 percent mepivacaine and 4 percent prilocaine with 2 percent lidocaine plus 1:100,000 epinephrine.

Local anesthetic solution	Duration (min)[a]		Dose per cartridge (mg)	Maximum safe dose (mg/lb)
	Pulpal	Soft tissue		
3% Mepivacaine	20–40	120–180	54	2.0
4% Prilocaine	10–60	90–240	72	2.7
2% Lidocaine with 1:100,000 epi.	60–90	120–240	36	2.0

[a]Longest durations are generally obtained after block anesthesia

Pharmacokinetic studies in adults have shown that average peak blood levels of 3 percent mepivacaine are three times greater than that of 2 percent lidocaine with 1:100,000 epinephrine, after maxillary infiltration anesthesia.[10-12] This further reduces the therapeutic index of mepivacaine on an injection volume basis, especially in children.

The predominant toxic reactions to local anesthetics appear in the central nervous system (CNS) and cardiovascular system.[4,5] Early toxicity from a mild overdose or inadvertent intravascular injection is usually self-limiting and generally consists of a variety of CNS symptoms such as dizziness, drowsiness, and disorientation. Higher blood levels may produce convulsions due to an initial blockade of inhibitory neurons in the CNS, thus leaving the excitatory neurons firing unopposed. Still further increases in blood levels lead to unconsciousness and respiratory depression. The cardiovascular effects of a local anesthetic overdosage include vasodilation, which in turn can lead to a drop in systemic blood pressure. There is also a direct depressant effect on the myocardial cell membrane, which can cause a progressive bradycardia and full cardiac arrest. Death can occur due to either respiratory depression or cardiac arrest. It is also important to realize the narcotic sedative regimens lower the convulsive threshold of local anesthetics and increase the likelihood of respiratory depression.[2,3]

Signs of early local anesthetic-induced toxicity in man generally appear at blood levels of greater than 4.5 μg/ml of lidocaine or mepivacaine. Myocardial depression and peripheral vasodilation begin to appear at levels above 5.0 μg/ml. Tonic-clonic seizures and respiratory depression usually manifest themselves at anesthetic levels above 7.5 μg/ml. Massive vasodilation and full cardiac arrest usually do not occur until anesthetic levels exceed 10 μg/ml.[4] Individual variation in response to local anesthetic drugs may produce, however, adverse clinical signs and symptoms at blood concentrations lower than those discussed. In the present case, the peak blood levels of mepivacaine that may have existed can be estimated by considering the pharmacokinetics of the drug. After infiltration injection, peak blood levels of 3 percent mepivacaine plain usually appear within fifteen to thirty minutes.[12] The plasma half life of mepivacaine has been reported to average between 90 and 120 minutes.[4,13] Since this patient had a mepivacaine blood level of 1.35 μg/ml 4.5 hours after injection, it can be projected that peak blood levels in this child were at least 5.4 μg/ml to 9.9 μg/ml, clearly above the average threshold for local anesthetic toxicity.

CONCLUSIONS

This report illustrates the relatively low safety margin when employing local anesthetics in young children. On an injection volume basis, maximum safe dosages are more rapidly reached with 3 percent mepivacaine or 4 percent prilocaine than with 2 percent lidocaine plus epinephrine. In addition, there is currently no evidence to support the assumption that the administration of 3 percent mepivacaine or 4 percent prilocaine results in less lip and cheek biting than the administration of 2 percent lidocaine with 1:100,000 epinephrine. We thus agree with Yagiella's premise that, "in light of the low safety margin for local anesthetics in children, it is advisable to employ a preparation containing a vasoconstrictor, if not doing so would result in more total drug being administered."[6]

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Intraoral etiology of a life-threatening infection in an immunocompromised patient: Report of case

Ana Paula Faria Marques, DDS, MS
Paul O. Walker, DDS, MS

Acute leukemias are characterized by neoplastic proliferation of cell types of the hematopoietic population. With chemotherapy a high percentage of patients will remain in complete remission. In cases of patient relapse or at risk for relapse, bone marrow transplantation may be employed.[1]

Oral infections are one of the major causes of morbidity and mortality in patients with hematologic malignancies.[2,3] The increased susceptibility to oral infections is related to the degree and duration of granulocytopenia, preexisting oral disease, drug-induced disruption of mucosal integrity, and changes in the composition of the oral flora resulting in more virulent pathogens. Total body irradiation and chemotherapy suppress the ability of the immune system to respond.[4]

CASE REPORT

An eight-year-six-month-old Caucasian female, previously diagnosed with acute monocytic leukemia was admitted to the University of Minnesota Hospital and Clinics, with a rapidly enlarging swelling of the right submandibular area, and a temperature of 102° F. The child was on the 5th day of her fourth round of chemotherapy (daunorubicin and dexamethasone) and had a white blood cell count of 900 mm^3 with no neutrophils and a platelet count of 2000 mm^3. She presented no stridor or respiratory distress, but significant hard brawny edema of her right submandibular region and neck, with significant erythema, some pitting upon palpation, increased temperature, and no fluctuation.

Computerized tomography of the affected region revealed a phlegmon-type process involving the right submandibular space and neck. There were inflamed fat-soft tissue interfaces, but no signs of abscess and no deviation of the airway (Figure 1).

The patient was placed on an intravenous, broad-spectrum-antibiotic regimen (ceftazidime, metronidazole, penicillin and vancomycin), and white blood cell infusion.

When the patient was examined two hours later, the inflammation had increased and was crossing the midline into the submental area, and seemed to be progressing superiorly into her face. Immediate intubation was recommended, due to concerns about airway compromise. Nasotracheal intubation was performed in the operating room, after which the patient returned to the Pediatric Intensive Care Unit for continuous observation.

After twenty-four hours of intravenous antibiotic therapy the patient showed no improvement and the infection appeared to have increased. The edema extended from the level of the mastoid tip to the submandibular area, passing the midline on the submental

Dr. Marques is with the Division of Pediatric Dentistry, Department of Preventive Sciences, University of Minnesota, Minneapolis, MN. Dr. Walker is Director, Advanced Education Program, Division of Pediatric Dentistry, Department of Preventive Sciences, University of Minnesota, Minneapolis, and Director, Hospital Dental Clinic of Minnesota Hospital and Clinics.

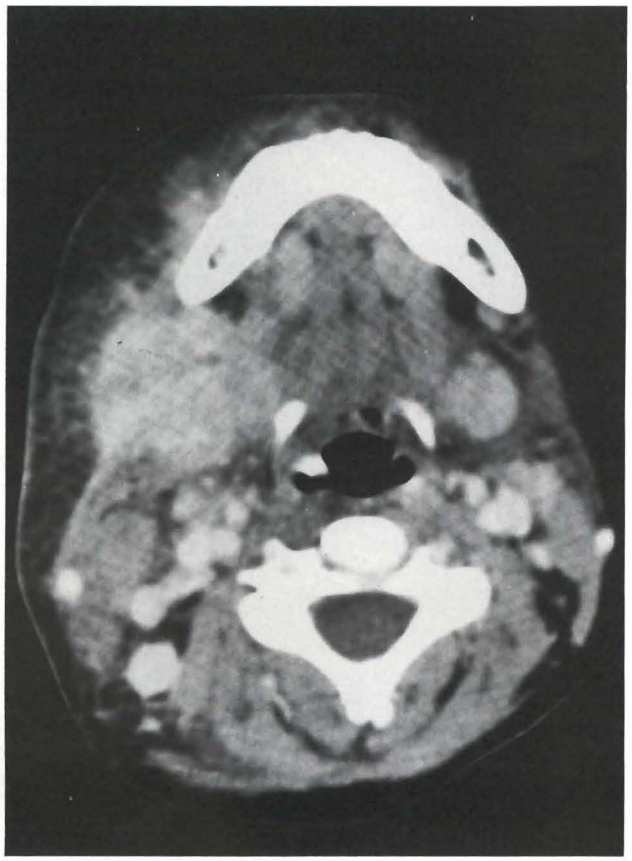

Figure 1. Initial CT scan showing marked swelling of the right submandibular space. Note the patency of the airway.

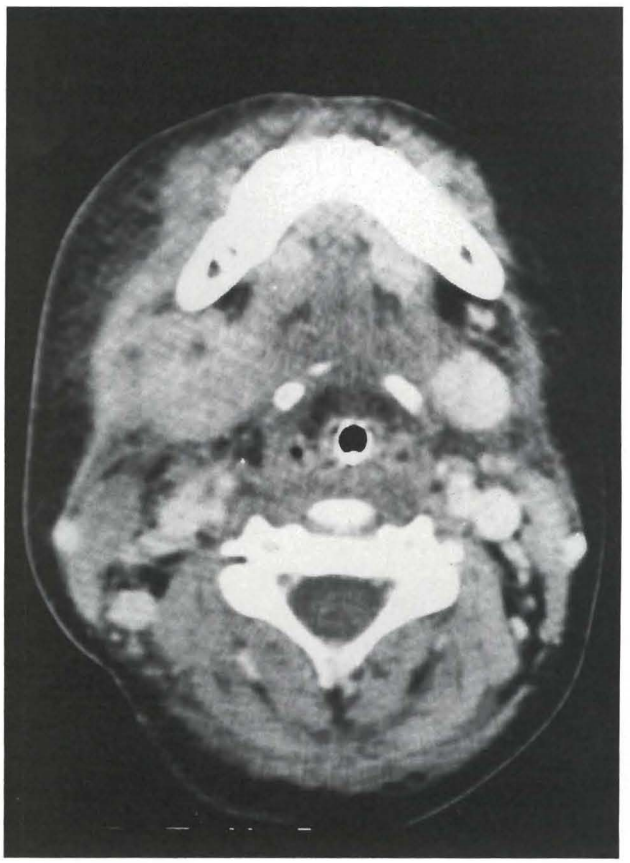

Figure 2. Preoperative CT scan showing swelling of the right submandibular space and complete airway obstruction, which was kept patent only by the endotracheal tube.

area. The erythema extended up to the level of the zygoma and down her neck. At this time there was concern for a developing necrotizing fasciitis. She had not signs of sepsis; her temperature, however, had risen to 105° F. Incision and drainage of the neck was recommended.

Preoperative computerized tomography was performed to help direct the incision and drainage procedure. It revealed complete airway obstruction from massive edema, which was kept patent only by means of the endotracheal tube (Figure 2). Dissection of the neck and drainage of any possible abscess was undertaken to prevent extension of the disease into the chest.

Using general anesthesia, a wide incision was made, approximately two inches below the mandible, from the mastoid tip to the midline of the neck (Figure 3). Blunt dissection was carried down to the level of the submandibular gland, and the submandibular space was entered. From this route the submental, submasseteric, and temporal spaces were entered and clear fluid was drained from each of these spaces. No pus was found. Large Penrose drains were sutured into each space, and the neck was wrapped (Figure 4). The fluid was cultured for aerobic and anaerobic bacteria, fungi, AFB, TB, and a viral serology. A biopsy was also performed. Laboratory results revealed a fragment of unremarkable skeletal muscle. No organisms could be identified.

A consultation has been requested with the Pediatric Dentistry service to determine whether an oral condition could be responsible for the swelling. An intraoral examination revealed normal dentition with a mobile mandibular right primary molar, no swelling of the buccal plate, lack of clinically detectable dental caries, periodontal morbidity, or significant oral morbidity. The patient's overall oral health was good. Panoramic

This work was undertaken at the Hospital Dental Clinic of Minnesota Hospital and Clinics.

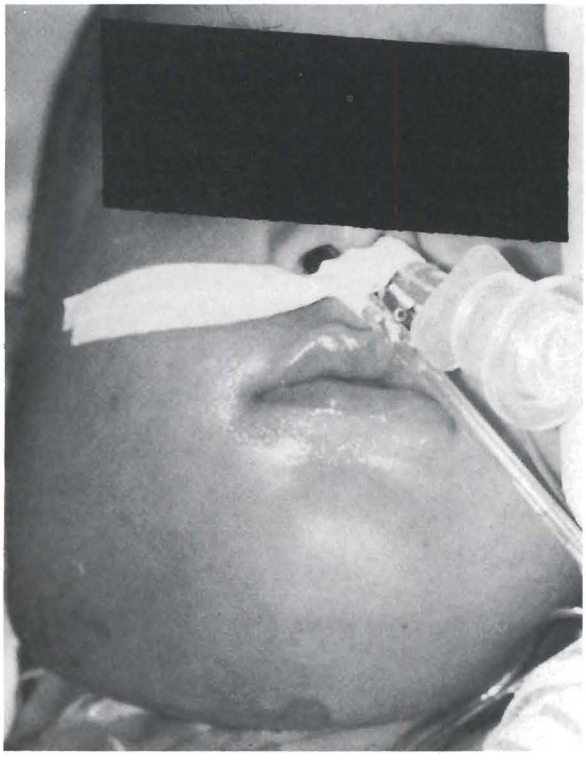

Figure 3. Extraoral view of the incision and swelling.

and periapical radiographs were taken (Figures 5 and 6). The right mandibular film showed an erupting permanent canine causing resorption of the mesial root of the mandibular right primary molar, resulting in the clinically observed mobility (Figure 6). Although a conclusive etiology for the infection was not determined, the erupting canine and resorbing primary molar clearly provided a communication of the intraoral environment with the submandibular space. Strict oral hygiene procedures and regular irrigation with 0.12 percent chlorhexidine gluconate were prescribed.

Over the next ten days, the patient showed increasing signs of progressive healing. Her swelling and fever decreased dramatically and she was then extubated.

Twenty days later, the swelling had completely resolved, the incision site was healing well, the patient's condition was stable, and she was discharged from the hospital.

CONCLUSION

This case illustrates the importance of early discussion on managing preexisting dental conditions in patients with hematologic malignancies. Controversy exists over

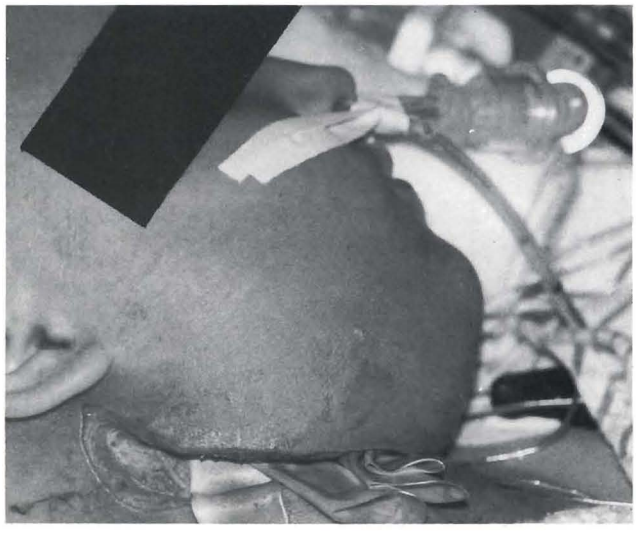

Figure 4. Extraoral view of the incision and drains.

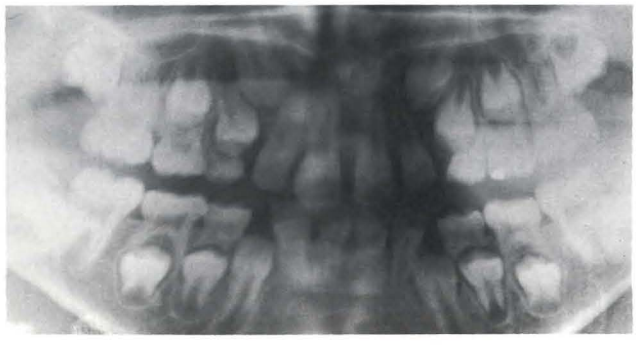

Figure 5. Panoramic radiograph of patient at the time of infection.

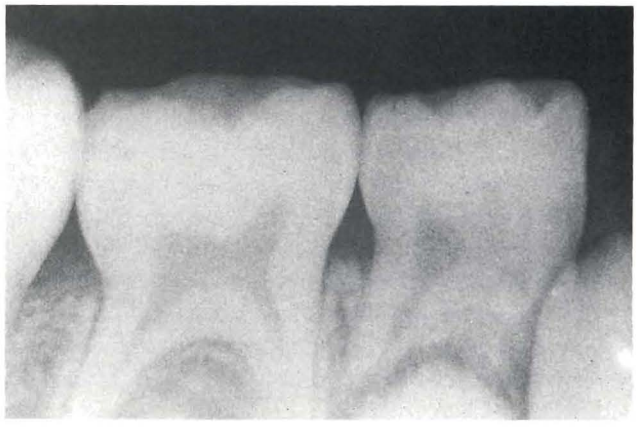

Figure 6. Intraoral periapical radiograph of right mandibular area, showing erupting permanent canine causing resorption of the mesial root of the first primary molar.

Computerized tomography revealed complete airway obstruction from massive edema.

the issue of whether or not mobile teeth should be extracted in patients with suppressed immune system.[5]

In pediatric patients it is essential to account for developmental changes such as tooth eruption and exfoliation, which may provide a setting for other oral complications such as secondary infections which can have life-threatening consequences.

Note: One year after this episode the patient successfully received a autologous bone marrow transplant and was doing well.

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EFFECTS OF ANTICALCULUS DENTIFRICE ON REMINERALIZATION

The ability of anticalculus dentifrices containing 3.3% pyrophosphate ion to inhibit calculus formation has been demonstrated repeatedly [Lobene and Volpe, 1987; Schiff, 1987; Rosling and Lindhe, 1987; Mallat, 1985; Zacherl *et al*, 1985]. Because pyrophosphate functions to inhibit calculus formation by interfering with the formation and growth of crystal nuclei in plaque [McGaughey, 1983], it has the potential of interfering with the natural anticaries mechanism of remineralization. Previous studies, however, have shown that 3.3% pyrophosphate in fluoride dentifrice formulations does not interfere with remineralization in situ [Mellberg *et al*, 1987] nor does 1% pyrophosphate in a mouthrinse [Mellberg *et al*, 1988]. Similarly, pyrophosphate did not adversely affect the anticaries effect of fluoride in a rat caries model [Spak *et al*, 1988]. These findings are also in agreement with human clinical caries studies involving anticalculus dentifrices [Lu *et al*, 1985; Triol *et al*, 1988]. In spite of these favorable data, the use of lower concentrations of any anticalculus agent might be considered desirable, not only to improve the taste of the dentifrice, but also to minimize potential interference with remineralization.

Mellberg, J.R. *et al.*: Evaluation of the effects of a pyrophosphate-fluoride anti calculus dentifrice on remineralization and fluoride uptake in situ.
Caries Res, 25:65-69, January-February 1991.

ABSTRACTS

Seow, W. Kim: Enamel hypoplasia in the primary dentition: a review. J Dent Child, 58:441-452, November-December 1991.

Clinical significance of enamel hypoplasia includes poor esthetics, tooth sensitivity, malocclusion and predisposition to dental caries. It may provide diagnostic clues as to genetic influences and systemic diseases, as well as to any trauma during the span of dental development. These systemic factors include birth trauma, infections, nutritional disorders, metabolic diseases, and exposures to chemicals such as tetracycline, lead, and fluoride. Inherited types form a relatively small component overall, including genetic abnormalities of enamel formation, or dental features of many inherited diseases or dysmorphic syndromes. Developmental enamel defects range in prevalence from 4 percent to 60 percent, depending on the criteria and the population studied. It is likely that many of these individual factors may in fact act through a central mechanism: mineral deficiency.

Hypoplasia, enamel; Biology, molecular; Deficiency, mineral; Development, dental

Veerkamp, J.S.J.; van Amerongen, W.E.; Hoogstraten, J.; Groen, H.J.: Dental treatment of fearful children, using nitrous oxide. Part I: Treatment times. J Dent Child, 58:453-457, November-December 1991.

This study investigated the influence of nitrous oxide on treatment time when it is used as an additional aid to behavior management. Different parts of the treatment of highly anxious child dental patients may vary during sequential dental visits, and separate consecutive parts of the treatment were considered in terms of their duration. Fifty-six children between the ages of six and eleven, each with a previous negative dental experience, were selected. They were randomly assigned into one of two matching groups: one with behavioral management only (control); the other also having nitrous oxide sedation (experimental). Interval 1, involved getting used to the procedures; interval 2 was sedation; interval 3, local anesthesia; and interval 4, preparation and filling. Most children required between four and six sessions. Results show that in terms of total treatment time (TTT), the use of nitrous oxide does not save time, although dental treatment did not require a greater number of sessions. For the dentist to erase the negative imprint of a previous dental experience, special skills are required. Further research is advised concerning the anxiety levels of this age-group.

Nitrous oxide; Behavior management; Pediatric dentistry

Pinkham, Jimmy R.: An analysis of the phenomenon of increased parental participation during the child's dental experience. J Dent Child, 58:458-463, November-December 1991.

The role of the parent during their child's dental experience, in contemporary society, has changed during the past decade and should be addressed by pediatric dentists. Included in this matter are issues regarding risk management and practice management. Behavioral pediatric dentistry is in flux, much like the world that it serves; there appear to be schisms within the profession regarding one aspect of this: the presence of parents in the dental operatory. Presented here is an analysis of the paradigm-shifts in society since World War II, when a *postfigurative* parenting strategy was in vogue. An explication of noted anthropologist Margaret Mead's descriptions of this and two subsequent parenting styles, including *configurative* (or transitional) and *prefigurative* (contemporary) are presented. They are analyzed in terms of parental attitudes concerning trusting their child to authority figures, including the dental clinician. This emerging protective instinct is termed "social hypertrophy", based on social biologist Edward O. Wilson's work. Dentists are encouraged to understand parental attitudes and avoid unnecessary conflict.

Sociobehavioral tendencies; Pediatric dentistry; Attitudes, parental; Childrearing strategies (postfigurative, configurative [and] prefigurative); Social hypertrophy

Zadik, Dan; Deitsch, Arkadi; Tamir, Dov; Kelman, Moshe A.: Trends in the prevalence of dental caries in Israel. J Dent Child, 58:464-466, November-December 1991.

This study's objective was to assess the prevalence of dental caries in five-year-olds and twelve-year-olds in Jerusalem, Israel. A total of 166 children comprised the younger group; and 147 children represented the twelve- to thirteen-year-olds. Examinations took place in the classrooms under natural lighting, using a mouth mirror and a probe. It was found that 27.7 percent of the younger children were caries-free, with a mean dif (t) value of 3.65 ± 3.58. At the age of twelve years, only 4.8 percent were caries-free, with a mean DMF (T) value of 4.9 ± 3.09. Nearly 58 percent of these carious permanent teeth had been treated. In Jerusalem, fluoridation of the water supply was implemented only recently. There are signs of increased public awareness of prevention methods.

Caries; Prevention; Epidemiology; Israel

Levy, Steven M. and Zarei-M, Zavash: Evaluation of fluoride exposures in children. J Dent Child, 58:467-473, November-December 1991.

This paper reports the findings of a study looking at children's fluoride exposures in detail beyond the fluoride content of

home drinking water. What emerges are the most clinically relevant questions to ask parents when conducting histories of fluoride exposure of child patients. Dental caries in children has decreased dramatically in the U.S. and other developed countries. At the same time, the prevalence and severity of dental fluorosis in the U.S. may be increasing because of the widespread ingestion of fluoride. Children are vulnerable to fluorosis, according to the results of a fifty-item questionnaire administered to mothers through the University of Iowa. Patients have reported multiple water sources and fluoride levels, use of soy-based formulas, toothpaste ingestion, and dietary fluoride supplements.

Fluorosis; Pediatric dentistry; Exposure, fluoride; Ingestion, toothpaste; Supplementation, fluoride

Seow, W. Kim; Shepherd, R.W.; Ong, T.H.: Oral changes associated with end-stage liver disease and liver transplantation: implications for dental management. J Dent Child, 58:474-480, November-December 1991.

In children, chronic liver disease has a variety of causes; its effects include malnutrition, bleeding tendencies, osteopenia, and rachitic changes in the skeleton. Chronic liver disease in children may have many oral manifestations, such as green staining of the teeth and gingiva, as well as enamel hypoplasia. Dental management may be complicated by many factors, like bleeding tendencies and inability to metabolize routine anesthetics. Patients who undergo increasingly successful liver transplantation require lifelong immunosuppressive therapy, which has side-effects. Results of this study of nine patients, seven females and two males, show that the effects of end-stage liver disease are permanently recorded on the teeth. Six were examined both before and after liver transplantation; the other three, only afterwards. This study confirms and extends the findings of three previous reports describing the oral manifestations of congenital biliary atresia in childhood.

Liver disease, chronic; Children; Liver transplantation; Pediatric dentistry

Douglass, Joanna and Tinanoff, Norman: The etiology, prevalence, and sequelae of infraclusion of primary molars. J Dent Child, 58:481-483, November-December 1991.

Dental infraclusion, teeth below the occlusal plane, is a common condition, especially in mandibular primary molars, with more than seventy references in the literature. Most claim infracluded molars do not exfoliate within normal time limits and must be extracted to prevent detrimental sequelae. The few documented studies on this topic suggest, however, that most infracluded teeth exfoliate normally; however, there is a tendency toward a delay of six months. The degree of infraclusion does not appear to correspond with exfoliation time. The frequent sequelae of severely infracluded teeth, space loss and molar tipping, may be prevented by either restoring the height of the affected tooth or by using space maintainers without extraction. The successors of infracluded molars reportedly develop normally and have few occlusal abnormalities.

Infraclusion; Molars, primary; Exfoliation; Sequelae

Grimm, Stephen E.: Treatment of a pseudo-class III relationship in the primary dentition: a case history. J Dent Child, 58:484-488, November-December 1991.

Posterior crossbites and pseudo-class III relationships of the primary dentition should be corrected once noted clinically, if the child is cooperative. Crossbites will not improve with the eruption of the succedaneous teeth and eventually the crossbite relationship of the primary teeth will be mimicked in the permanent dentition. Correction of the primary dentition can be accomplished by removable or fixed appliances. The case report of a 3.5-year-old girl is described.

Crossbite; Dentition, primary; Relationship, Class III, pseudo-

Hersh, Elliot V.; Helpin, Mark L.; Evans, Owen B.: Local anesthetic mortality: Report of case. J Dent Child, 58:489-491, November-December 1991.

The case of a healthy five-year-old, thirty-six pound female patient scheduled for multiple extractions is reported. The child received a total dose of 270 mg of mepivacaine, instead of the correct dose of 72 mg, which resulted in multiple seizures, hospital admission, pneumonia, and death caused by anoxic brain injury secondary to cardiopulmonary arrest following the overdose.

Mepivacaine; Recommended dosages; Anesthetic, local; Morbidity; Mortality

Marques, Ana Paula Faria and Walker, Paul O.: Intraoral etiology of a life-threatening infection in an immunocompromised patient: Report of case. J Dent Child, 58:492-495, November-December 1991.

Oral infections are one of the major causes of morbidity and mortality in patients with hematologic malignancies. Total body irradiation and chemotherapy suppress the ability of the immune system to respond to the more virulent pathogens that can attack the compromised patient. The case of an 8.5-year-old girl is reported, in which an oral infection and a clinically observed motility resulted in a communication with the submandibular space; a significant hard, brawny edema of her right submandibular area resulted. Surgery, strict oral hygiene, and regular irrigation with a chlorhexidine gluconate solution resulted in successful healing.

Teeth, mobile; Immunosuppression; Malignancy, hematologic