

PREVENTION OF DENTAL DISEASE

The Role of the Pediatrician

Tara E. Schafer, DMD, MS, and Steven M. Adair, DDS, MS

Oral and dental health are integral parts of good overall health, and dental caries is the most common chronic infectious disease of childhood. A child with severe dental caries may experience chronic oral pain and infection, be malnourished, frequently absent from school, and suffer from low self-esteem because of missing or defective teeth. Evidence in the dental literature increasingly suggests that, to be successful in preventing dental disease, clinicians must begin risk-factor determination, preventive counseling, and preventive interventions within the first year of life (infant oral health care is discussed in detail in the article by Nowak and Warren later in this issue). Pediatricians are well positioned to begin this process as they see their patients for well-baby visits and as they provide anticipatory guidance to parents and other caregivers. Pediatricians are also in a good position to see that every child has a dental home in addition to the medical home. This article provides information to pediatricians that will enable them to provide practical, targeted, and effective advice to parents about preventing dental disease. A full review of the pathogenesis of dental caries can be found in the article by Caufield and Griffen earlier in this issue. This article focuses on reviews of etiologic factors, the evidence to support preventive measures for each factor, and recommendations that can be made to parents. Risk factors for dental disease are presented to enable pediatricians to identify infants and toddlers at high risk for caries. The role of fluoride

From the Departments of Pediatric Dentistry (TES, SMA) and Oral Biology and Maxillofacial Pathology (SMA), Medical College of Georgia, Augusta, Georgia

in caries prevention is discussed, with emphasis on dietary fluoride supplements, which are often prescribed by pediatricians. Finally, this information is brought together in a section on anticipatory guidance and other interventions that enable pediatricians to begin the process of dental disease prevention before referral for an initial dental evaluation.

As explained elsewhere in this issue, transmission of the bacterial etiologic agent for caries, mutans streptococci (MS), occurs in infancy. Thus, true prevention can occur only in infants who have not yet been infected. When the transmission of MS has occurred, pediatricians and dentists must consider ways to suppress the caries activity through the medical management of dental caries.¹⁵ Medical, or nonsurgical, management can be used up to and including the point at which tooth cavitation occurs. As illustrated in Table 1, surgical management (i.e., excavation and replacement of carious tooth structure) must be used at some point to deal with the symptoms of dental caries, that is, cavities.¹⁴ This schema also indicates that treatment becomes more expensive as it becomes more invasive. Prevention and early diagnosis are just as important in managing dental caries as in managing any disease.

ETIOLOGIC FACTORS AND PREVENTION OF DENTAL CARIES

Dental caries is a dietary carbohydrate-modified infectious disease. It is the most common chronic infectious disease of childhood, with a prevalence of more than 40% by age 6 years in the primary dentition and more than 85% by age 17 years in the permanent dentition.^{12, 32} Caries typically is described as a multifactorial process, involving specific oral microflora, diet, and a susceptible host. Prevention of dental caries therefore is aimed at (1) reducing cariogenic microbes in the oral cavity; (2) reducing the exposure of these microbes to a cariogenic substrate; and (3) increasing the decay resistance of the tooth. A combi-

Table 1. NATURAL HISTORY OF A CARIOUS LESION

Cost of Intervention	Status of Child/Carious Lesion	Management
Less expensive	Predentate; dentate, not infected; dentate, infected, no active caries	Prevention
	Active caries	Suppressor, medical management
More expensive	Decalcification/white spot lesion	Medical/surgical management
	Cavity present Infection of dental pulp; pain, alveolar or facial infection	Surgical management

Adapted from Edelman BL: Medical management of infant and toddler dental caries. In Pinkham JR (ed): Pediatric Dentistry: Infancy Through Adolescence. Philadelphia, WB Saunders, 1999, p 181.

nation of dietary advice, coupled with mechanical plaque removal (e.g., brushing and flossing) and adequate fluoride exposure, is sufficient to control dental disease in most patients.

Control of Microflora

As detailed in the article by Caufield and Griffen earlier in this issue MS are transmitted vertically from parents (usually the mother) to infants at an early age. The presence of MS is a necessary, but not solely sufficient, condition for dental caries. The primary antimicrobial approach to caries prevention traditionally has been the removal of plaque through oral hygiene practices. The relationship between oral hygiene practices and caries prevention, however, is not strong. Bacteria are harbored in niches where caries often initiates, such as the pits and fissures on the biting surfaces of posterior teeth, the protected environment between teeth, and the microscopic crevices at the margins of dental restorations. These niches may not be cleansible with standard brushing and flossing techniques. Thus, the suppression of MS by chemotherapeutic means is beginning to find favor.⁶²

Chlorhexidine

Topical treatment with chlorhexidine has been shown to reduce the number of MS in the saliva of highly infected patients,⁴⁰ and it is used in dentistry in various forms. The best result has been obtained with the use of chlorhexidine gel (not yet available in the United States) in custom-fitted trays. In younger children, application of a gel by toothbrush may be more easily managed.⁶⁴ A significant decrease in the number of MS in the saliva of preschool-aged children has been reported after daily brushing with chlorhexidine gel, and this decrease was still evident 1 month after the termination of the 2-week study.⁶⁴ In some experiments, chlorhexidine was more effective than was fluoride, whereas other studies demonstrated an additive effect of the two agents.⁵² A 0.12% chlorhexidine mouthrinse (Peridex, PerioGard) is available with prescription in the United States.

Other studies have attempted with some degree of success to delay or prevent the transmission of MS from mothers to their children.^{22, 34} The decrease of maternal salivary MS at the time of tooth emergence may delay or prevent the colonization of MS in the child's primary dentition, with a concomitant decrease in caries prevalence.⁶¹ The influence of the age of infection with MS on the subsequent development of caries has been demonstrated in numerous animal studies⁵⁶ and in humans.⁵ Children who become infected at younger ages are at a higher risk for caries. To date, no specific guidelines exist for prescribing chlorhexidine or other topical antimicrobials, but clinicians may choose to use these agents as adjuncts to standard oral hygiene practices in children who are at high risk for early childhood caries. Also, because

patients with xerostomia are at increased risk for caries, twice-daily chlorhexidine mouthrinse use should be considered for individuals who have received radiotherapy in the region of the salivary glands and for patients who are on long-term medications that suppress salivary flow (see section on host factors).

Oral Hygiene

Young children require assistance with their oral hygiene routine, and parents should integrate oral hygiene into children's daily schedules. Before age 1 year, it is sufficient to clean the child's oral tissues and teeth with a soft cloth or gauze pad. Parents may wish to use a fluoride-free dentifrice, though this may be difficult to find. At least one product, First Teeth (Nu-Tec Health Products, Carlsbad, CA) is marketed as a "baby toothpaste" for children as young as 5 months of age. Fluoride-containing toothpaste typically is not recommended until children gain some control of the swallowing reflex, typically at approximately 3 years of age. Young children swallow as much as 60% of the toothpaste that is dispensed, or up to 0.8 g per brushing^{8, 9} which provides 0.8 mg of fluoride ion. This may lead to systemic overexposure to fluoride and subsequent mild discoloration of the developing teeth. Some practitioners, however, may wish to recommend the use of a fluoridated dentifrice at a young age (18–24 mo) for children at high risk for dental caries. Whenever the use of fluoride-containing toothpaste is begun, parents should use a pea-sized amount of dentifrice on a soft-bristled toothbrush. Parents can adopt various techniques to aid in brushing their children's teeth, including standing behind the child or having the child place his or her head in the parent's lap. Whatever method is selected, parents should initiate oral hygiene measures at least once daily just before bedtime. Gradually, the child can become a more active participant in the process, and parents may elect to alternate brushing with the child, in a my-turn, your-turn fashion.

Recommendations to parents include:

- Clean the oral cavity after feedings. A moist cloth is sufficient for infants.
- Begin toothbrush cleanings at approximately age 1 year, using a moist, soft-bristled toothbrush. A nonfluoridated dentifrice, if available, can be introduced as tolerated by the infant. The teeth should be cleaned at least once daily just before bedtime. Twice-daily cleanings are preferable.
- Introduce fluoride-containing dentifrice when the child can control the swallowing reflex, or earlier for high-risk children. Use a small amount of toothpaste. Children who wish to brush can do so with a moist toothbrush; fluoride dentifrice should be applied to the teeth by parents. The child should be encouraged to expectorate freely at the end of the brushing.

- Older, high-risk patients should be evaluated by their dentists for the possible need for other antimicrobial regimens.

Dietary and Medication Factors of Caries Promotion and Protection

Cariogenic bacteria metabolize fermentable carbohydrates, resulting in periodic production of organic acids and subsequent loss of the mineral components of tooth structure. Dietary interactions with the other factors necessary for caries are complex. Total carbohydrate consumption and the frequency of intake are associated with dental caries.^{11, 21, 35}

More than any other carbohydrate, sucrose has been associated with the formation of carious lesions. The relationship between the sucrose content of the individual food or of the total diet and the resulting caries describes a sigmoid curve, which rises steeply when sucrose-containing foods are eaten on a frequent basis, when newly erupted teeth are at high risk, and when the immune response is immature, as is found in young children. After this initial increase in caries, the curve levels off, indicating that increases in the sucrose content of the diet beyond a certain level do not increase caries development to any significant extent.⁴⁸

Despite extensive research, foods unlikely can be reliably ranked on the basis of cariogenicity. The Toothfriendly labeling system, popular in some European countries, has not found favor in the United States. The US Food and Drug Administration (FDA) allows *Does not promote tooth decay* labels for products containing sugar alcohols known to be noncariogenic, but concerns over testing models have led to resistance to subscribing to more lenient labeling. The following recommendations may be of value: (1) parents should be informed of the known association between frequent consumption of fermentable carbohydrates and dental caries; (2) parents should be encouraged to promote balanced, low-caries-risk diets; (3) parents should be encouraged to limit their children's frequency of exposure to highly cariogenic foods; and (4) parents should have various dentally healthful snacks on hand to promote healthier snacking.

Between-meal snacks should be low in fermentable carbohydrates or contain nonfermentable sweeteners, of which the FDA has approved four: saccharin, aspartame, acesulfame K, and sucralose. The consistency of the food may be equally important in the overall scheme. Sticky or adhesive foods that can maintain high sugar levels in the mouth for a prolonged time are more cariogenic than are foods that are rapidly cleared. The minimal sucrose content required for cariogenicity varies with the stickiness, rate of oral clearance, and the frequency of ingestion. Some foods, such as cheese, have anticariogenic effects.^{13, 23, 31} These effects of cheese may be related to the presence of calcium lactate and various fatty acids. Calcium and phosphates may be retained by salivary

micelles and therefore serve as slow-release units for mineral components needed for tooth-surface remineralization and the prevention of demineralization. Also, the physical form of various cheeses may promote salivary flow, which, in turn, buffers decreases in pH and aids in cleansing and clearance of food particles. Various other foods and food components have been investigated as caries-protective agents, including chocolate, nuts, licorice, and phosphopeptides from milk. Milk has been reported to have some cariostatic properties and is thought to contribute little to the production of caries when ingested under normal dietary conditions.⁵³ Foods with protective actions, those that are hypoacidogenic in plaque pH studies, and those made with nonfermentable carbohydrates, such as xylitol, can be recommended, though conclusive data are lacking in this area.

A child's use of a bottle containing juice, formula, milk, or liquids sweetened with fermentable carbohydrates can increase the risk for early childhood caries (ECC) caused by prolonged contact of sugars in the beverage with cariogenic bacteria present on the teeth.⁵⁷ The contents of the bottle and the frequency and duration of bottle use contribute to potentially dramatic decay. The risk for ECC also exists for at-will breastfed children and is related to frequency of exposure to breast milk that is allowed to pool on the teeth, especially if the child falls asleep while nursing. This exposure, coupled with less-than-effective oral hygiene practices, can have deleterious effects on the teeth, leading to pain, infection, growth retardation,² and early loss of teeth. The American Academy of Pediatric Dentistry offers the following suggestions to aid in the prevention of ECC. First, infants should not be put to sleep with a bottle containing any liquid other than water. Prolonged, at-will breastfeeding should be avoided after eruption of the first tooth. Second, parents should be encouraged to have infants drink from a cup as they approach their first birthday. Weaning from the bottle should occur between 12 and 14 months of age in most cases. Thirdly, oral hygiene measures should be implemented by the time of eruption of the first primary tooth. Lastly, an oral health consultation visit within 6 months of eruption of the first tooth is recommended for evaluation of the child's dental condition and to educate parents and provide anticipatory guidance for prevention of dental disease.⁶

Information released in 1984 from the National Pharmaceutical Association indicated that nearly 100% of pediatric medicine contained sucrose.⁴⁷ Some formulations contain as much as 80% sucrose on a weight-volume basis (Table 2).²⁵ Children receiving sucrose-containing medications demonstrated a higher incidence of caries and gingivitis than did children not receiving medication.⁵⁴ Frequency of administration coupled with the chronic dosing and pH of the sucrose-containing medication contribute to the caries susceptibility of these children. The cariogenic potential of children's medications can be reduced in various ways, perhaps most simply by educating parents about the need for cleaning the teeth after each dose of medication. Bedtime dosages of medications should be given before the child's nightly oral hygiene

Table 2. SUCROSE CONTENT OF SELECTED LIQUID PEDIATRIC PREPARATIONS

Rights were not granted to include this data in electronic media. Please refer to the printed journal.

Adapted from Hill EM, Flaitz CM, Frost GR: Sweetener content of common pediatric oral liquid medications. American Journal of Hospital Pharmacy 45:135, 1988; with permission.

ritual. A more effective strategy would be to substitute sugar-free formulations when available.

Recommendations to parents include:

- Do not put the infant to sleep with a bottle containing any liquid other than water.
- Infants who fall asleep while breastfeeding may be at higher risk for caries.
- Wean the infant from the bottle by 14 months of age.
- Avoid prolonged consumption of sweetened beverages or low-pH fruit juices from a bottle or "tippy" cup.
- Do not dip pacifiers in sweetened solutions or honey (also carries a risk for infant botulism⁶⁷).
- Monitor the child's diet for the amount and frequency of exposure to fermentable carbohydrates. Restrict intake of sweets to meal-times, when salivary flow is greater. Substitute less-cariogenic foods as snacks.
- Clean the child's teeth after the intake of medications flavored with sucrose.

Host Factors

Individuals with teeth are susceptible to some degree to dental caries. This susceptibility can be lowered if the teeth are structurally sound, if salivary flow and composition are normal, and if sufficient ambient fluoride is present in the oral environment. Whole saliva, the mixture of secretions in the oral cavity derived from major and minor salivary glands plus the gingival exudate, is the host's greatest defense against caries. Saliva significantly influences the carious process, as evidenced by a myriad of animal experiments in which the salivary glands are surgically removed¹⁷; however, even in desalivated animals,

a cariogenic substrate is still a necessary factor for caries, underscoring the multifactorial causes of the disease process.

Saliva is supersaturated with respect to calcium and phosphate. The pH at which these ions precipitate is referred to as the critical pH, thought to be approximately 5.5. At any pH less than this value, tooth structure may begin to dissolve.

The effects of saliva can be partly explained by its ability to wash away food debris and bacteria. Saliva also possesses a buffering capacity, and some studies suggest that it has antibacterial properties. Salivary IgA antibodies prevent colonization of streptococci on epithelial cells,⁶⁸ but the role of salivary IgA in regulating colonization of MS on tooth surfaces is controversial.⁴⁴ Secretory IgA constitutes the main specific immune defense mechanism in saliva and may have an important role in the homeostasis of the oral microbiota.⁴¹ Protection against bacterial agents of caries may be conferred by salivary IgA antibodies by stimulation of the common mucosal immune system, which produces protective antibodies on mucosal surfaces, including those in the oral cavity. Serum-derived antibodies of the IgG type also exist in whole saliva and are at detectable levels within the first year of life. Nonimmune factors found in saliva include myeloperoxidase, lysozyme, lactoferrin, and cystatins. These antimicrobial agents may interact with each other and with the immune factors in an additive or synergistic manner.⁶¹ The antimicrobial agents produced by oral peroxidases may be protective in the early phases of dental caries.⁵¹ Dentifrices and mouthrinses that generate the hydrogen peroxide necessary to drive the production of hypochlorite from oral peroxidases are already commercially available, and these products may be beneficial in patients with xerostomia. Further research into the development of products containing nonimmune or immune agents may yield important new ways to battle oral infections, including tooth decay. The use of a sugar-free chewing gum after meals stimulates salivary flow and has documented beneficial effects.¹⁶ Gums sweetened with xylitol may provide additional antimicrobial effects.⁵⁸

Physiologic xerostomia occurs during sleep, when the salivary glands do not secrete spontaneously. With no saliva to buffer pH and wash away fermentation products of plaque during sleep, the most important time for plaque removal is just before bedtime. Also, many medications can produce xerostomia as a side effect, including antispasmodics, antidepressants, antihistaminics, anticonvulsants, and others. Individuals on these medications long term may benefit from increased oral hygiene measures, more frequent fluoride exposure, and saliva substitutes to reduce the damaging effects of reduced salivary flow.

Enamel is the hardest material in the body and, as such, is the first line of defense against decay. Developmental defects that reduce its hardness or alter its morphology can diminish its protective nature. Hypomineralization, a relatively rare phenomenon, results in enamel that is softer than normal and may be easily lost from the tooth. Hypoplastic enamel results from various conditions, including prematurity and very low birthweight. In hypoplasia, the enamel has normal hard-

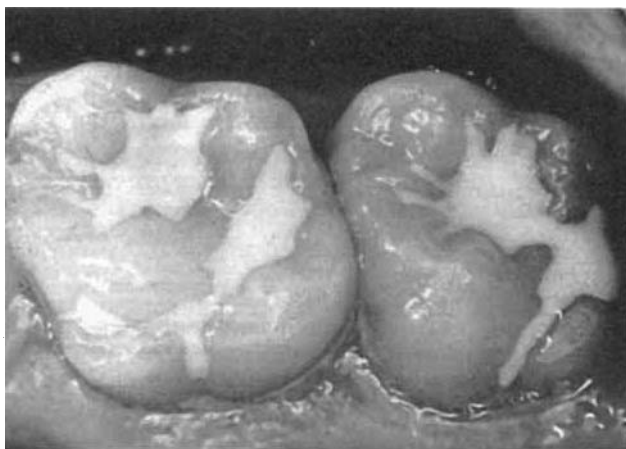


Figure 1. Pit and fissure sealants in place on two permanent upper molars. The sealant is adhesively bonded to the tooth enamel and prevents fermentable carbohydrate from entering the narrow pits and fissures in which cariogenic bacteria reside.

ness but is pitted, appears creased, or lacks normal thickness. Evidence shows that the hypoplastic defects associated with prematurity are sub-clinical but sufficient to allow for the development of ECC.

Ambient fluoride in the oral environment can promote remineralization of early enamel demineralization lesions. The mechanisms of action of fluoride and the appropriate use of fluoride modalities are discussed later.

The occlusal (biting) surfaces of molar and premolar teeth are the most caries-susceptible tooth surfaces. This is true of permanent first (6-y) molars, which can have deep pits and fissures that may be impenetrable by toothbrush bristles. Consequently, pit and fissure sealants have been advocated for teeth that are susceptible to decay, such as those with deep, non-coalesced pits and fissures. A sealant is a clear or shaded resin material that may be bonded to the chewing surfaces of caries-susceptible posterior teeth. The sealant forms a coating or barrier to protect these surfaces from decay (Fig. 1). Numerous studies have confirmed that sealants effectively prevent occlusal caries.²⁸

Recommendations for parents include:

- Prescribe fluoride supplements if needed.
- Establish a dental home for the patient at approximately 1 year of age.
- Ensure that the child's dentist evaluates the teeth for developmental defects and for the need for pit and fissure sealants.

CARIES RISK ASSESSMENT FOR PEDIATRICIANS

Assessing an infant's or toddler's risk for dental caries is an essential component of an oral health program. Disease risk assessment is a

systematic evaluation of the presence and intensity of etiologic and contributory disease factors. This assessment is designed to provide an estimation of an individual's disease susceptibility and to aid in targeting preventive and treatment strategies. In the case of dental caries, risk assessment information can be divided into three categories (Table 3).⁴⁵ Category I comprises markers of disease that are provided by the patient and parent through the history and physical examination. The presence of many of these markers can be determined by the pediatrician. Category II comprises disease markers and the single true risk factor for dental caries, the presence of MS. Some of the items in Category II can be appreciated by pediatricians, but most require dental training or technologies not likely to be present in a pediatric office. Determination of Category III markers requires the use of technologies that are not practical for clinical use at this time.

Table 3. CATEGORIES OF CARIES RISK FACTORS

Rights were not granted to include this data in electronic media. Please refer to the printed journal.

Among the data from Category I, pediatricians can make a general assessment of caries risk for a child through the patient's demographic data. The socioeconomic status of the family is a risk marker because dental caries is increasingly a disease of people of low socioeconomic status. Eighty percent of the dental caries in children in the United States can be found in approximately 25% of the population, and this portion of the population is typically children of poverty.^{19, 32, 66} Caries rates in some communities are also higher among some racial and ethnic groups, in particular African Americans, Hispanics, and Native Americans.^{32, 33} The patient's age indicates whether transmission of MS is likely to have taken place.¹⁰ Lower maternal education levels also have been associated with dental caries risk.²⁰

A child's medical history, well known to the pediatrician, can provide valuable information. Prematurity and very low birthweight are associated with the presence of enamel defects, often subclinical, that can predispose teeth to caries at an early age. An increased risk also exists for children who have taken syrup-based medications on a long-term basis. A child's dental history, which might not be well known to the pediatrician, may provide substantial information. A history of early childhood caries is one of the best indicators of future dental disease, so pediatricians should routinely evaluate the dentition for obvious carious lesions or evidence of dental restorations.

Behavioral factors include oral hygiene habits, feeding practices, and dietary preferences. A diet rich in fermentable carbohydrates, frequent snacking, constant use of a "tippy" cup filled with fruit juice, bottle use at sleeptime, and prolonged at-will breastfeeding have all been associated with early childhood caries.^{20, 27, 30} Parental inattention to oral hygiene practices are another marker for potential dental disease.²⁶ Patients with an impaired ability to maintain oral hygiene are at higher risk; this category of patients includes infants, toddlers, and young children, all of whom must rely on a caregiver for tooth-cleaning procedures. Others in this category include physically challenged and developmentally disabled individuals. Older children undergoing orthodontic treatment or wearing other types of intraoral appliances are also at a higher risk. Such appliances provide difficult-to-clean niches for plaque growth.

Other risk factors in Category I that may not be as readily available to pediatricians include the mother's oral health status. High levels of maternal dental caries⁶³ or a poor gingival condition⁵⁵ have been associated with higher levels of dental disease in the offspring. The child's fluoride exposure, although important in assessing resistance to tooth decay, is not always easily quantified. More detail is provided in the discussion on supplemental fluoride.

Data from Category II are more likely to be obtained in a pediatric dental office. A plaque index measures in a relatively standardized way the amount of plaque present on the teeth. High plaque levels have been associated with an increased risk for dental caries.^{42, 43} Diet histories are used to assess the cariogenicity of the diet and to provide the basis of recommendations for dietary changes. Salivary MS and lactobacilli

assays can be easily done with commercially available kits and incubators (Cultura Incubator, Dentocult-SM, and Dentocult-LB, Ivoclar Vivadent, Amherst, NY), although they are not in wide use at this time. Such tests not only indicate whether transmission of MS has occurred but also provide a quantitative measure of the infection, another factor in caries risk determination.²⁷ Salivary flow rate, although relatively easy to assess, is not routinely done, nor are assays of salivary buffering capacity, which requires special equipment.

Pediatricians interested in gathering data on caries risk in young children can use much of the information in Category I, especially the demographic data, including the parents' dental histories. Additional information about the patient's medical history, previous dental treatment (if any), and dietary or feeding habits may help practitioners to make a general determination about a child's risk for dental disease. Fine tuning the risk determination beyond low risk versus high risk is not easily done.

USE OF FLUORIDE IN CARIES PREVENTIVE PROGRAMS

Water Fluoridation

In the 1900s, McKay, practicing in Colorado, noticed that many of his dental patients exhibited an unusual intrinsic enamel discoloration, termed *Colorado brown stain* by area residents. McKay also noted that individuals with this type of mottled enamel had low levels of dental caries. Subsequent investigations determined that water-borne fluoride was responsible for the mottling and the caries resistance. Epidemiologic studies were conducted in the 1930s in several midwestern communities with differing levels of fluoride in the water supply to determine the relationships between fluoride concentrations in drinking water, dental fluorosis (replacing the term *mottling*), and dental caries. (An example of dental fluorosis is shown in Fig. 11 in article by Wright earlier in this issue). Those studies led to the findings that communities with water fluoride levels of approximately 1 mg/L (part per million) demonstrated the best compromise between caries reductions and community fluorosis levels. At that level, fluorosis in the population was mild in severity and low in prevalence. Subsequent studies determined the relationship between mean annual temperature and mean water consumption in various locales. Based on those data, recommendations were developed for artificially fluoridating water supplies at a fluoride level between 0.7 (warmer climates) to 1.2 (cooler climates) mg/L.

In the 1940s, full-scale prospective trials of artificial water fluoridation began in four pairs of cities in the United States and Canada. Each pair of communities was carefully selected after matching on many demographic, socioeconomic, and health parameters. Before the studies, the fluoride level in each city was negligible. Artificial fluoridation to a

level of 1.0 ppm to 1.2 ppm was begun in one city in each pair in 1945. Sequential cross-sectional surveys were conducted for 13 to 15 years. Among the multitude of variables studied, the only significant difference found was a 50% to 65% decrease in dental caries in the fluoridated communities. The prevalence and severity of fluorosis in each intervention city was comparable with those of communities with naturally occurring fluoride at a level of 1.0 mg/L. These studies ushered in an era of community water fluoridation that continues today and that resulted in sharp decreases in dental caries in the United States over the latter half of the twentieth century. This modality of fluoride delivery is extremely cost-effective, with estimates of per capita cost ranging from a few cents to a few dollars per year, depending on community size.

Contemporary Exposure to Fluoride

At the time of the initial fluoridation studies, an individual's exposure to the ion was limited to naturally occurring fluoride. Except in communities with high natural fluoride levels or artificial fluoridation, these levels were low. In the intervening years, however, the disparity in fluoride exposure has decreased between optimally fluoridated and fluoride-deficient communities. Exposure to fluoride in fluoride-deficient communities has increased through three primary sources: (1) fluoridated dentifrice; (2) foods and beverages processed in optimally fluoridated communities; and (3) fluoride supplements. This increase in fluoride exposure in fluoride-deficient communities has been termed the *halo effect* because of the concomitant reductions in caries in these locales. Today the caries-reduction benefits of optimal fluoridation are approximately 20% to 40% compared with some fluoride-deficient communities in contrast to the 50% reductions reported in the 1950s. This change is caused by an improvement of the dental health in residents of fluoride-deficient communities compared with 50 years ago. At the same time, the prevalence of fluorosis in fluoride-deficient communities has increased to levels approximating the prevalence in optimally fluoridated communities.⁶⁰ Most of this fluorosis is scored as mild and is not considered by most authorities to be cosmetically objectionable or to pose a public health concern. The halo effect does, however, make it difficult to accurately determine an individual's exposure to fluoride by simply assessing the fluoride concentration of the municipal or well water supply.

Fluoride Mechanisms of Action

At the time of the water fluoridation trials, and for some time afterward, investigators assumed that fluoride exerted its anticaries activity by becoming incorporated into developing enamel. The presence of fluoride in enamel results in a crystalline structure that is resistant to acid dissolution. Other studies purported that fluoride exposure during

dental development results in the formation of posterior teeth with shallower pits and fissures that are therefore less susceptible to decay.¹⁸ Some controversy exists on these issues, but most authorities now agree that the systemic effect of fluoride is minor.³⁸

Greater importance is now given to the topical effects of fluoride. Topical contact of fluoride with teeth occurs through the intake of foods and beverages containing the ion, use of fluoridated dentifrice, and perhaps more importantly through salivary secretion of fluoride that has been systemically absorbed. Topical effects are mediated through direct contact with teeth and through the effects of fluoride on bacterial plaque. Dental caries begins with demineralization of enamel through repeated exposure of acid produced by plaque bacteria as they metabolize fermentable carbohydrate. The early lesion begins below the enamel surface and appears clinically as chalky white enamel (Fig. 2). This white spot lesion can be remineralized as long as the surface enamel layer remains intact. The presence of low levels of fluoride in the oral environment promotes remineralization. Cycles of demineralization and remineralization occur constantly. As fluoride is taken up by the lesion during the remineralization process, the lesion is strengthened and becomes more acid resistant than the original intact enamel.

Fluoride in the oral environment also is absorbed by dental plaque and becomes concentrated to levels that can disrupt bacterial enzyme systems, decreasing the acidogenicity and aciduricity of plaque bacteria. Plaque also serves as a reservoir of bound fluoride that is released as free



Figure 2. A *white spot* lesion on the mesial proximal surface of primary lower molar now visible because of the exfoliation of the adjacent primary molar. The white spot represents an area of enamel that has been demineralized by the organic acids produced by plaque bacteria. As long as the surface enamel remains intact, as it is in this example, it is possible to remineralize the lesion.

fluoride during a pH decrease. Free fluoride ions then can participate in the remineralization process.

Supplemental Fluoride

Dietary fluoride supplements were developed as a means of providing fluoride to individuals who resided in fluoride-deficient communities. They represented an attempt to provide a daily dose of fluoride equivalent to the intake of individuals who resided in optimally fluoridated communities. Early supplementation schedules were somewhat awkward, requiring that sodium fluoride tablets be dissolved in water that was used for drinking and cooking. This rationale was also based on the earlier assumption that the anticaries mode of action of fluoride was primarily systemic. Subsequent schedules began the practice of supplementing with a daily dose of fluoride based on the age of the patient and the fluoride content of the drinking water supply. Clinical studies documented caries reductions of 15% to 30% in individuals in fluoride-deficient communities who consumed dietary supplemental fluoride. Dietary fluoride supplements can approximate the caries protection only of water-borne fluoride, given their relatively high-dose, low-frequency nature. By contrast, exposure to fluoridated water is a low-dose, high-frequency regimen.

In several studies over the past 30 years, fluoride supplements have been associated with mild fluorosis.^{1, 50} The severity of fluorosis was low, and it was most often found in children who began supplementation before the age of 6 years. Findings of this sort have prompted reductions in the fluoride-dosage schedules over the years. The current schedule, adopted in 1994, is shown in Table 4. No studies are available to document the fluorosis rates associated with the new schedule, but it is reasonable to assume that the risk for fluorosis is lower.

The benefit of supplemental fluoride can be maximized and the risk can be minimized by appropriate prescribing of supplements, which entails:

- **Determination of the fluoride concentration in the child's primary drinking water source, which might not be tap water in the home.** Many families use bottled water, which usually has low levels of fluoride^{39, 59, 65}; however, some contain significant concentrations of the ion. Households with reverse osmosis water-filtration systems have a reduced level of fluoride in the tap water. Point-of-use filtration systems, such as charcoal filters, typically do not remove fluoride. Most well water in the United States is deficient in fluoride, but in some areas of the country, fluoride occurs naturally in ground water sources. Finally, some children may spend most of their days in a day-care setting or at a relative's house, and the primary source of drinking water may come from that location, not the home. Whatever the case, the primary drink-

ing water source should be assayed for fluoride content. This analysis may be provided by some local or state health departments, schools of dentistry, or through commercial sources (Fluori-Check, Omnii Products, West Palm Beach, FL).

- **Appropriate prescribing.** Fluoride supplements should not be prescribed for children younger than 6 months or whose primary drinking water contains significant levels (> 0.6 mg/L) of fluoride.
- **Education of the parents and caregivers.** Parents who do not understand the potential benefits of supplemental fluoride are less likely to fill and properly use the prescriptions with their children. The trade-off of a risk of mild fluorosis for reduced caries experience must be considered by pediatricians and discussed with the parents. Consideration of a child's risk factors for caries may assist in this decision.

Fluoride supplements are available as drops, tablets, and lozenges that deliver 0.25 mg, 0.5 mg, or 1.0 mg fluoride ion to match the doses indicated in Table 4. Drops should be placed on the infant's tongue once daily between feedings. Tablets and lozenges should be administered after tooth brushing, preferably just before bedtime. They should be sucked or chewed for 1 or 2 minutes before swallowing to maximize contact with the teeth. Supplemental fluoride is also available in combination with vitamins. No evidence suggests that the fluoride in these products is any less effective in caries prevention than is fluoride alone; however, in some cases, the vitamin and fluoride dose requirements of the child may be incompatible with the combinations available. In such cases, it may be necessary to resort to separate supplements to avoid prescribing too much or too little of either component in combination products.

The FDA bans manufacturers of fluoride supplements from claiming that the use of supplemental fluoride by pregnant women will convey a caries-protective effect to their offspring. The only placebo-controlled, double-blind study to investigate this practice in a fluoride-deficient community found no additional benefits to prenatal supplementation

Table 4. FLUORIDE SUPPLEMENTATION SCHEDULE ADOPTED IN 1994 BY THE AMERICAN ACADEMY OF PEDIATRICS, AMERICAN ACADEMY OF PEDIATRIC DENTISTRY, AND AMERICAN DENTAL ASSOCIATION

Age	Fluoride Concentration of Primary Drinking Water Source, mg/L (ppm)		
	<0.3	0.3-0.6	>0.6
Birth to 6 mo	0*	0	0
6 mo to 3 y	0.25	0	0
3 y to 6 y	0.50	0.25	0
6 y to at least 16 y	1.00	0.50	0

*Dose in mg fluoride ion.

when the offspring received postnatal supplements and brushed with a fluoridated dentifrice.³⁶

Fluoride-containing Dentifrices

Fluoride-containing dentifrices, introduced in the 1950s, constitute approximately 95% of the dentifrice market in the United States. Most products sold in the United States contain approximately 1000 to 1100 mg/kg (ppm) fluoride, although some dentifrices with higher concentrations (1500 mg/kg fluoride) have been introduced. Trials of at least 2 years' duration with fluoridated dentifrice have resulted in median caries reductions in the order of 15% to 30%.^{29, 46} Regular use of fluoride-containing toothpaste over a lifetime probably provides decay protection equivalent to that of fluoridated drinking water. The combination of the two provides additional protection.⁴⁹

Because some children enjoy the taste of toothpaste and ingest it deliberately, parents should be cautioned to keep these products out of the reach of young children. Additional vigilance is required with dentifrices that are flavored to appeal to children because they may encourage young patients to use more toothpaste at each brushing, thereby increasing the risk for ingestion.^{3, 37}

Fluoride-containing Mouthrinses

Fluoridated mouthrinses, initially prescription-only products, became available over the counter in the 1980s. These products contain 0.05% sodium fluoride, a concentration equivalent to approximately 1 mg fluoride per teaspoonful. Prescription mouthrinses may be ingested, but over-the-counter products are intended for topical use only. They are unsuitable for use by children whose swallowing reflex is not fully mature. Fluoridated mouthrinses have been shown to be effective in individuals and groups at high risk for dental caries, but the rationale for their routine use by most children is questionable.⁴

Professionally Applied Fluoride Compounds

Dentists routinely apply high-concentration (12,300–22,600 mg/kg) fluoride gel, foam, or varnish to their patient's teeth once or twice yearly to provide protection against decay. Although investigators originally thought that the fluoride from these products entered enamel crystals, research has shown that professionally applied fluoride forms a layer of calcium fluoride-like material on the enamel surface. When the oral pH decreases, fluoride is released and made available to remineralize early developing carious lesions.

Self-Applied Fluoride Compounds

Fluoride gels with a concentration of 0.5% sodium fluoride (5000 mg/kg) or 0.4% stannous fluoride (1000 ppm) are available as prescription agents for patient self-application using custom-made dental trays or simply by toothbrush. Although the evidence supporting the caries-protective benefits of 0.5% NaF is good, no clinical trials have documented the efficacy of 0.4% stannous fluoride, which has the same fluoride concentration as does dentifrice. The stannous ion, however, has been shown to have some antimicrobial effects.²⁴ Self-applied gels are primarily reserved for short-term use in individuals at high risk for caries.

ANTICIPATORY GUIDANCE BY PEDIATRICIANS FOR PREVENTION OF DENTAL DISEASE

Pediatricians can do much to make parents more aware of the importance of preventing dental disease in their children. Pediatricians interested in promoting good oral health should identify demographic and socioeconomic risk factors for dental caries and flag high-risk children as being in need of more intensive preventive counseling.

Pediatricians should include a brief dental screening as part of the routine examination of well children. The mouth should be inspected, noting the number of erupted teeth, their color, spacing, enamel quality, presence of dental restorations, and obvious dental caries. Patients with caries should be referred to a dentist immediately for treatment.

Practitioners should consider including the following educational aspects in the anticipatory guidance that they provide to all parents:

- **Nutritional counseling.** Before the eruption of teeth, parents should be taught basic information about the role of diet in promoting good oral health, and dietary factors that can lead to dental decay. Pediatricians should advise parents about appropriate foods and snacks.
- **Feeding practices.** Inappropriate use of nursing bottles and "tippy" cups as pacifiers should be discussed. Prolonged at-will breastfeeding or use of a nursing bottle at sleeptimes should be discouraged. Parents should be apprised of the dental effects of the prolonged use of high-sugar liquids and foods.
- **Oral and dental cleaning.** The pediatrician or office staff member should demonstrate methods of cleaning the oral cavity and, when erupted, the teeth. Parents should be instructed to clean the infant's mouth routinely after feedings. This practice ingrains in the child and parent the need for regular tooth brushing when the child is older. Parents of toddlers and preschool-aged children should be reminded to perform tooth cleanings for their children, introducing fluoridated toothpaste when the child has some con-

trol of the swallowing reflex, or earlier for high-risk children. Parents should be cautioned to use a pea-sized dab of toothpaste and to guard against swallowing excessive amounts of dentifrice.

- **Review of medications.** High-sugar medications that a child is taking long term should be identified, and parents should be cautioned to clean the child's teeth after ingestion. Physicians should consider sugar-free alternatives, if available.
- **Determination of fluoride status.** Physicians should determine the fluoride content of the primary drinking water source. Dietary supplemental fluoride should be prescribed as appropriate. Physicians should educate parents about the benefits and provide instructions on proper administration.

ESTABLISHING A DENTAL HOME

Pediatricians should assist parents in establishing a dental home for their children by referral to a pediatric dentist or family dentist for an initial evaluation and consultation. The American Academy of Pediatric Dentistry⁶ and the American Public Health Association⁷ recommend that a child's first dental visit occur within 6 months after the eruption of the first tooth, typically at 1 year of age. If a family does not have access to dental care, physicians should refer to a community dental clinic or health department for care and treatment as needed.

Finally, pediatricians and dentists should work together for the benefit of the child. Promoting good oral health is as critical as is any other aspect of pediatric care in promoting good overall health.

References

1. Aasenden R, Peebles TC: Effects of fluoride supplementation from birth on deciduous and permanent teeth. *Arch Oral Biol* 19:321, 1974
2. Acs G, Shulman R, Ng MW, et al: The effect of dental rehabilitation on the body weight of children with early childhood caries. *Pediatr Dent* 21:109, 1999
3. Adair SM, Piscitelli WP, McKnight-Hanes C: Comparison of the use of a child and an adult dentifrice by a sample of preschool children. *Pediatr Dent* 19:99, 1997
4. Adair SM: The role of fluoride mouth rinses in the control of dental caries. *Pediatr Dent* 20:101, 1998
5. Alaluusua S, Renkonen OV: *Streptococcus mutans* establishment and dental caries experience in children 2 to 4 years old. *Scand J Dent Res* 91:453, 1983
6. American Academy of Pediatric Dentistry: Reference Manual 1999-00. *Pediatr Dent* 21:77, 1999
7. American Public Health Association: Policy Statements Adopted by the Governing Council of the American Public Health Association, November 10, 1999 [on-line]. Available: www.apha.org/legislative/policy
8. Barnhart WE, Hiller LK, Leonard GJ, et al: Dentifrice usage and ingestion among four age groups. *J Dent Res* 53:1317, 1974
9. Beltran ED, Szpunar SM: Fluoride in toothpastes for children: Suggestion for change. *Pediatr Dent* 10:185, 1988

10. Berkowitz R: Etiology of nursing caries: A microbiologic perspective. *J Public Health Dent* 56:51, 1996
11. Bibby BG: The cariogenicity of snackfoods and confections. *J Am Dent Assoc* 90:121, 1975
12. Brunelle JA, Carlos JP: Recent trends in dental caries in U.S. children and the effect of water fluoridation. *J Dent Res* 69(spec iss):723, 1990
13. deSilva M, Jenkins G, Gurgess R, et al: Effect of eating cheese on Ca and P concentrations on experimental caries in human subjects. *Caries Res* 20:263, 1986
14. Edelstein BL: Medical management of infant and toddler dental caries. In Pinkham JR (ed): *Pediatric Dentistry: Infancy Through Adolescence*. Philadelphia, WB Saunders, 1999, p 181
15. Edelstein BL: The medical management of dental caries. *J Am Dent Assoc* 125(suppl):31, 1994
16. Edgar WM: A role for sugar-free gum in oral health. *J Clin Dent* 10(spec iss):89, 1999
17. Finn SB, Klapper CE, Volker JF: Intraoral effects upon experimental hamster caries. In Sognnaes RF (ed): *Advances in Experimental Caries Research*. Washington, DC, American Association for the Advancement of Science, 1955, p 152
18. Foreman FJ, Retzlaff AE: Effects of systemic fluoride on the morphology of occlusal grooves of primary and permanent molars. *ASDC J Dent Child* 57:101, 1990
19. Graves RC, Bohannon HM, Disney JA, et al: Recent dental caries and treatment patterns in US children. *J Public Health Dent* 46:23, 1986
20. Grindefford M, Dahllöf G, Nilsson B, et al: Prediction of dental caries development in 1-year-old children. *Caries Res* 29:343, 1995
21. Gustafsson B, Quensel C, Lanke L, et al: The Vipeholm dental caries study: The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. *Acta Odontol Scand* 11:232, 1954
22. Gunay H, Dmoch-Bockhorn K, Gunay Y, et al: Effect on caries experience of a long-term preventive program for mothers and children started during pregnancy. *Clinical Oral Investigations* 2:137, 1998
23. Harper S, Osborn J, Herrermen J, et al: Cariostatic evaluation of cheeses with diverse physical and compositional characteristics. *Caries Res* 20:123, 1986
24. Hastreiter RJ: Is 0.4% stannous fluoride gel an effective agent for the prevention of oral diseases? *J Am Dent Assoc* 118:205, 1989
25. Hill EM, Flaitz CM, Frost GR: Sweetener content of common pediatric oral liquid medications. *American Journal of Hospital Pharmacy* 45:135, 1988
26. Hinds K, Gregory JR: National Diet and Nutrition Survey: Children aged 1.5 to 4.5 years. Report of the Dental Survey, vol 2. London, Her Majesty's Stationary Office, 1995
27. Holbrook WP, de Soet JJ, de Graaff J: Prediction of dental caries in pre-school children. *Caries Res* 27:424, 1993
28. Horowitz HS, Heifetz SB, Poulson S: Retention and effectiveness of a single sealant application of an adhesive sealant in preventing occlusal caries: Final report after five years of a study in Kalispell, Montana. *J Am Dent Assoc* 95:1133, 1971
29. Horowitz HS, Law FE, Thompson MB, et al: Evaluation of stannous fluoride dentifrice for use in dental public health programs: I. Basic findings. *J Am Dent Assoc* 72:408, 1966
30. Ismail AI: The role of early dietary habits in dental caries development. *SCD Special Care in Dentistry* 18:40, 1998
31. Jenkins G, Hargreaves J: Effect of eating cheese on Ca and P concentrations of whole mouth saliva and plaque. *Caries Res* 23:159, 1989
32. Kaste LM, Selwitz RH, Oldakowski RJ, et al: Coronal caries in the primary and permanent dentition of children and adolescents 1-17 years of age: United States, 1988-91. *J Dent Res* 75(spec iss):631, 1996
33. Kaste LM, Marianos D, Chang R, et al: The assessment of nursing caries and its relationship to high caries in the permanent dentition. *J Public Health Dent* 52:64, 1992
34. Kohler B, Brathall D, Krasse B: Preventive measures in mothers influence the establishment of *Streptococcus mutans* in their infants. *Arch Oral Biol* 28:225, 1983
35. König KG, Schmid P, Schmid R: An apparatus for frequency controlled feeding of small rodents and its use in dental caries experiments. *Arch Oral Biol* 13:13, 1968

36. Leverett DH, Adair SM, Vaughan BW, et al: Randomized clinical trial of the effect of prenatal fluoride supplements in preventing dental caries. *Caries Res* 31:174, 1997
37. Levy SM, Maurice TJ, Jakobsen JR: A pilot study of preschoolers' use of regular-flavored dentifrices and those flavored for children. *Pediatr Dent* 144:388, 1992
38. Limeback H: A re-examination of the pre-eruptive and post-eruptive mechanism of the anti-carries effects of fluoride: Is there any anti-carries benefit from swallowing fluoride? *Community Dent Oral Epidemiol* 27:62, 1999
39. MacFadyen EE, McNee SG, Weetman DA: Fluoride content of some bottled spring waters. *Br Dent J* 153:423, 1982
40. Maltz M, Zickert I, Krasse B: Effect of intensive treatment with chlorhexidine on number of *Streptococcus mutans* in saliva. *Scand J Dent Res* 89:445, 1981
41. Marcotte H, Lavoie MC: Oral microbial ecology and the role of salivary immunoglobulin A. *Microbiol Mol Biol Rev* 62:71, 1998
42. Mascarenhas AK: Oral hygiene as a risk indicator of enamel and dentin caries. *Community Dent Oral Epidemiol* 26:331, 1998
43. Mattos-Graner RO, Zelante F, Line RC, et al: Association between caries prevalence and clinical, microbiological and dietary variables in 1.0 to 2.5-year-old Brazilian children. *Caries Res* 32:319, 1998
44. McGhee JR, Michalek SM: Immunology of dental caries: Microbial aspects and local immunity. *Ann Rev Microbiol* 35:595, 1981
45. Moss ME, Zero DT: An overview of caries risk assessment, and its potential utility. *J Dent Educ* 59:932, 1995
46. Muhler JC: Effect of a stannous fluoride dentifrice on caries reduction on children during a three-year study period. *J Am Dent Assoc* 64:216, 1962
47. National Pharmaceutical Association: Sugar Content of Medicines: Notes for Proprietors. St. Albans, Hertfordshire, National Pharmaceutical Association, 1984
48. Newbrun E: *Cariology*, ed 2. Chicago, Quintessence, 1989
49. Newbrun E: Effectiveness of water fluoridation. *J Public Health Dent* 49(spec iss):279, 1990
50. Pendrys GD, Katz RV: Risk of enamel fluorosis associated with fluoride supplementation, infant formula, and fluoride dentifrice use. *Am J Epidemiol* 130:1199, 1989
51. Pruitt KM, Reiter B: Biochemistry of peroxidase system: Antimicrobial effects. In Pruitt KM, Tenovuo J (eds): *The Lactoperoxidase System: Chemistry and Biological Significance*. New York, Marcel Dekker, 1985, p 143
52. Regolati B, Schmid R, Muhlemann HR: Combination of chlorhexidine and fluoride in caries prevention. *Helvetica Odontologica Acta* 10:12, 1974
53. Reynolds E: Anticariogenic complexes of amorphous calcium phosphate stabilized by phosphopeptides: A review. *SCD Special Care in Dentistry* 18:8, 1998
54. Roberts IF, Roberts GJ: Relation between medicines sweetened with sucrose and dental disease. *BMJ* 2:14, 1979
55. Sasahara H, Kawamura M, Kawabata K, et al: Relationship between mothers' gingival condition and caries experience of their 3-year-old children. *Int J Paediatr Dent* 8:261, 1998
56. Schuster GS, Morse PK, Birksen TR: Interaction of microbial challenge and age at inoculation in the production of dental caries in rats. *Caries Res* 12:28, 1978
57. Seow WK: Biological mechanisms of early childhood caries. *Community Dent Oral Epidemiol* 26(suppl 1):8, 1998
58. Söderling E, Mäkinen KK, Chen CY, et al: Effect of sorbitol, xylitol and xylitol/sorbitol chewing gums on dental plaque. *Caries Res* 23:378, 1989
59. Stannard J, Rovero J, Tsamtsouris A, et al: Fluoride content of some bottled waters and recommendations for fluoride supplementation. *Journal of Pedodontics* 14:103, 1990
60. Szpunar SM, Burt BA: Trends in the prevalence of dental fluorosis in the United States: A review. *J Public Health Dent* 47:71, 1987
61. Tenovuo J: Antimicrobial function of human saliva: How important is it for oral health? *Acta Odontol Scand* 56:250, 1998
62. Tinanoff N: Dental caries: Etiology, pathogenesis, clinical manifestations, and management. In Wei SHY (ed): *Pediatric Dentistry: Total Patient Care*. Philadelphia, Lea & Febiger, 1988, p 9

63. Tuutti H, Lahti S, Honkala E, et al: Comparison of dental caries experience of the parents of caries-free and caries-active children. *J Paediatr Dent* 5:93, 1989
64. Twetman S, Grindeford M: Mutans streptococci suppression by chlorhexidine gel in toddlers. *Am J Dent* 12:89, 1999
65. Van Winkle S, Levy SM, Kiritsy MC, et al: Water and formula fluoride concentrations: Significance for infants fed formula. *Pediatr Dent* 17:305, 1995
66. Vargas CM, Crall JJ, Schneider DA: Sociodemographic distribution of pediatric dental caries: NHANES III, 1988-1994. *J Am Dent Assoc* 129:1229, 1998
67. Wigginton JM, Thrill P: Infant botulism: A review of the literature. *Clin Pediatr* 32:669, 1993
68. Williams RC, Gibbons RJ: Inhibition of bacterial adherence by secretory immunoglobulin A: Mechanism of antigen disposal. *Science* 177:697, 1972

Address reprint requests to

Tara E. Schafer, DMD, MS
Department of Pediatric Dentistry
School of Dentistry
1459 Laney Walker Boulevard
Medical College of Georgia
Augusta, GA 30912-1210

e-mail: tschafer@mail.mcg.edu