Pediatric Oral Health-
Caries Management & Preventive Therapies
L0118 - 3 credits

Manual and Test developed by:
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COURSE OBJECTIVES

At the completion of this course the dental provider should be able to do the following:

1. Discuss the etiology of dental caries.
2. Identify caries susceptibility factors.
3. Identify the caries experience of various ethnic groups.
4. Recognize the role of carbohydrates in caries production.
5. Define early childhood caries according to the American Academy of Pediatric Dentistry (AADP) and the American Dental Association (ADA).
6. Demonstrate understanding of the role of fluoride as a decay-preventive measure and its available forms.
7. List the clinical manifestations and prevention measures for early childhood caries.
8. Describe periodontal disease in children and appropriate treatment options.
9. Identify ways members of the dental team can educate and advise young patients and their parents about preventing tooth decay.

ACKNOWLEDGMENTS

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INTRODUCTION

Oral diseases, such as dental caries and periodontal disease, can be devastating to both children and adolescents. Dental caries may interfere with a child’s ability to eat, sleep, and learn, and it can have a significant impact on their self-esteem. Because caries affects individuals disproportionately, it is essential that those at the highest risk are identified early so that preventive therapies can be targeted toward those who are most likely to benefit.

EPIDEMIOLOGY AND TRENDS OF PEDIATRIC CARIES

The prevalence of caries varies in developed and undeveloped countries and among socioeconomic groups in developed countries. In the United States, among children and adolescents aged 6 to 19 years, 42% had caries experience in their permanent teeth, and approximately 14% had untreated tooth decay in their permanent teeth. Among children aged 2 through 11, 41% had caries experience in their primary teeth, and approximately 21% had untreated tooth decay in their primary teeth (CDC:2005).

In its 2005 report comparing findings of two National Health and Nutrition Examination Surveys (NHANES) on dental caries, the Centers for Disease Control and Prevention (CDC) indicated that dental caries is trending higher among the nation’s youngest children. Although caries prevalence decreased among permanent teeth of 6- to 19-year-olds from 1988-1994 and

Likewise, the CDC reported a decrease in untreated tooth decay among permanent teeth of children ages 6 to 19, but not among ages two through five (CDC:2005). This suggests that over 4 million children are affected nationwide, a rise of over 600,000 preschoolers over a decade.

Because tooth decay in the primary teeth predicts future tooth decay in permanent teeth, the upturn in caries experience in today’s preschoolers may be expected to continue in their permanent teeth as they grow older. The challenge to the dental care system is significant as U.S. preschoolers have the lowest rates of dental care of all age groups and, therefore, currently miss an important and timely opportunity for effective prevention.

**Oral Health Disparities in Poor Children and Children of Color**

Low-income children have the greatest odds of having tooth decay, have the most severe experience with tooth decay, and are most likely to have untreated cavities (CDC:2005). According to the CDC data, children ages 2 through 11 in families with income under $18,000 are nearly twice as likely to experience decay as children in families with twice that income level (55% versus 31%). In addition, children in poverty are more than twice as likely to have untreated cavities as their higher-income peers (33% versus 13%).
In addition, children of color are more likely to experience tooth decay and have their cavities untreated (CDC:2005). Mexican-American children had higher caries experience (55%), compared with black (43%) or non-Hispanic white children (38%). Non-Hispanic white children had a lower prevalence (18%) of untreated tooth decay than non-Hispanic black (27%) and Mexican-American children (32%). Because children of color are the fastest growing subpopulation of American children, their higher caries experience predicts an upturn in disease prevalence in coming years.

ETIOLOGY OF DENTAL CARIES

Dental caries is a multifactorial, posteruptive destructive disease affecting the mineralized tissues of the teeth. It results from the interaction of three variable factors:

- Fermentable carbohydrates
- Pathogenic oral microorganisms in dental plaque
- Tooth surfaces that are susceptible to acid dissolution

Caries susceptibility of teeth is affected by a number of factors, including:

- Genetics
- Nutritional status during tooth formation
- Fluoride incorporation during post-eruption stages
• Tooth and surface morphology

Occlusal surfaces are the most caries prone, followed by proximal and free, smooth surfaces. The pits and fissures of first and second adult molars rank higher in caries susceptibility than other permanent teeth.

**Microbial Etiology of Dental Caries**


Dental plaque is a biofilm consisting of microorganisms and their products such as adhesive glucans. In dental caries there is an enrichment, induced by dietary carbohydrate, of the plaque microbiota with organisms such as the mutans streptococci and lactobacilli, which causes an increase in the pH-lowering and cariogenic potential of plaque. The shift in the proportions of these organisms appears to be related to their relatively high acid tolerance (van Houte:1994; Kleinberg:2002).

A large body of evidence also supports a major effect of saliva on caries development (van Houte:1994; Kleinberg:2002). Salivary effects appear to have a major role in regulating the exposure of tooth surfaces to carbohydrate and plaque acidity and hence the cariogenic potential of dental plaque.
The first organisms to be implicated as caries-etiologic agents were the acidogenic lactobacilli, but a much stronger case can be made for the mutans streptococci, a group of relatively well-defined species (van Houte:1994; Kleinberg:2002). In many Western European countries and the United States, the mutans streptococci in plaque are predominantly *Streptococcus mutans*, with significant but lesser numbers of *S. sobrinus*. Like the lactobacilli, they rank high among the plaque microbiota with respect to their acid tolerance and their acidogenicity in an acidic plaque milieu.

The most important virulence factor of mutans streptococci is glucosyltransferases, which synthesize adhesive, water-insoluble glucans from sucrose (van Houte:1994; Kleinberg:2002). *S. mutans* and *S. sobrinus* produce three and four glucosyltransferases, respectively, whose cooperative actions is essential for adhesive glucan synthesis. Adhesive glucans mediate attachment of bacteria to the tooth surface as well as to each other.

Glucan synthesis is an important virulence trait of these organisms. This process may increase plaque’s cariogenicity by enhancing plaque mass, by promoting the colonization of mutans streptococci on the teeth, and by changing the diffusion properties of the plaque matrix. Thus, tooth surfaces destined to become carious are often associated with plaque in which mutans streptococci make up a large proportion of the cultivable flora; cavitated lesions also often contain high levels of these bacteria.
Early Childhood Caries

Rampant caries in infants and young children has long been recognized as a clinical syndrome referred to by various names, including nursing caries, nursing bottle syndrome, night bottle mouth, and baby bottle tooth decay (Reisine:1998; Misra:2007; ADA:2000). It represents a serious problem in dentistry as it progresses rapidly and affects young children.

The common theme among these terms is the central role of the baby bottle in the etiology and progression of carious lesions, but use of the baby bottle is not the only factor and may not be the most important one in caries development. The CDC has suggested that this clinical syndrome be called simply early childhood caries or ECC (Reisine:1998; CDC:2005).

There is a wide variation in the case definitions and diagnostic criteria for ECC. However, the American Academy of Pediatric Dentistry (AADP) and American Dental Association (ADA) use decayed/missing/filled score or DMFS, the number of decayed (noncavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces. Early childhood caries is defined as a DMFS of 1 or greater in primary teeth in a preschool-age child between birth and 71 months of age (ADA:2000). The term severe early childhood caries or S-ECC refers to “atypical” or “progressive” or “acute” or “rampant” patterns of dental caries. In children younger than three, any sign of smooth-surface caries is indicative of S-ECC. From ages three through five, S-ECC is defined as DMFS of 1 or greater in primary maxillary anterior teeth, or a total DMFS greater than 4 (for age three), greater than 5 (for age four), or greater than 6 (for age five).
In children with ECC, the plaque microbiota or the carious material itself consists almost entirely of mutans streptococci (van Houte:1994; Kleinberg:2002). Permanent colonization of a child’s oral cavity with mutans streptococci can occur only after tooth eruption because these bacteria require a nonshedding surface for attachment (Tinanoff:2001; Tinanoff:2002). The bacteria typically come from the child’s primary caregiver, usually the mother. Those teeth first exposed to a cariogenic environment generally are the first to show signs of disease. Consequently, children at high risk for ECC may develop lesions on their maxillary anterior teeth soon after eruption.

Early childhood caries can be a particularly virulent form of caries, beginning soon after dental eruption, developing on smooth surfaces, progressing rapidly, and having a lasting detrimental impact on the dentition (AAPD:2007e; AAPD:2007f). Children experiencing caries as infants or toddlers have a much greater probability of subsequent caries in both the primary and permanent dentitions. Not only does ECC affect teeth, but also the consequences of this disease may lead to more widespread health issues. Infants with ECC grow at a slower pace than caries-free infants. Some young children with ECC may be severely underweight because of associated pain and the disinclination to eat. ECC also may be associated with iron deficiency.

**Risk Factors for ECC**

Frequent bottle feeding at night, breastfeeding upon demand, and extended and repetitive use of a no-spill training cup are associated with ECC,
but not consistently implicated (AAPD:2007f). However, the severity of ECC is associated with poor feeding habits. In addition, a consistent association exists between clinical hypoplasia and ECC.

Social, socioeconomic, educational, and cultural considerations are factors associated with ECC. Maternal factors also play an important role in ECC risk (Misra:2007; Spitz:2006). Study results indicate that the mother’s DMFS, education, and feeding habits are strong risk indicators for the colonization of caries-related microorganisms and ECC (Ersin:2006). The DMFS scores of the mothers are an impact factor for the children’s caries experience. The prolonged usage of feeding bottle with sweetened milk, pacifier use, and maternal sharing are strongly associated with the colonization of S. mutans, lactobacilli, and Candida albicans, respectively.

**CLINICAL DECISION-MAKING FOR PEDIATRIC CARIES MANAGEMENT**

Appropriate dental care in a child requires an understanding of the carious process that includes the patient’s age, caries risk, prior therapy outcomes, location, and extent of the lesions. In Tinanoff’s 2001 clinical decision-making model, a child who has been identified as being at low risk for dental caries may need few diagnostic procedures and preventive therapies. Conversely, a child who is caries-active may require frequent diagnostic procedures and preventive therapies.
Currently, decisions for therapy often are based on whether a tooth is diagnosed as cavitated by clinical or radiographic examination (Tinanoff:2001; Tinanoff:2002). In addition to determining whether a tooth is cavitated or not, caries diagnosis should attempt to estimate the more critical issue—whether a lesion is progressing or arrested. Currently, longitudinal evaluation of lesion progression at periodic recall visits is the best method to determine lesion activity and progression.

Along with other information, such as the likelihood of a patient returning for periodic recalls and depth of a lesion, the clinician must decide whether an active carious lesion may require preventive and restorative therapy, whereas nonactive or arrested lesions may require no therapy.

**Caries Risk Assessment**

In young children, the risk indicator—previous caries experience—is not particularly useful because it is important to determine caries risk before disease is manifest (Tinanoff:2001; Tinanoff:2002). However, various markers of caries risk have been identified. For example, low birth weight has been suggested as a caries risk indicator for primary teeth, either directly, because it is associated with enamel hypoplasia and other enamel defects, or indirectly, because it is a marker for low socioeconomic situations.

Other caries risk indicators in preschool children are:

- The age that a child becomes colonized with cariogenic flora
- The child’s mutans streptococci levels
• Baseline caries scores
• Presence of visible plaque on the maxillary anterior teeth
• Sociodemographic factors, such as education and income of parents

**PREVENTIVE THERAPIES**

**Systemic Fluoride Therapy**

Daily fluoride exposure through optimizing the fluoride content of water supplies has been shown to be efficacious in reducing dental caries, with reductions in the range of 40% to 50% for primary teeth (Tinanoff:2001; Tinanoff:2002).

The AAPD treatment guidelines on fluoride therapy recommend that systemically administered fluoride supplements be considered for all children drinking fluoride-deficient (<0.6 ppm) water (AAPD:2007g). After determining the fluoride level of the water supply or supplies, evaluating other dietary sources of fluoride, and assessing the child’s caries risk, the daily fluoride supplement dosage can be determined using the Dietary Fluoride Supplementation Schedule. To optimize benefits of systemic fluoride supplements, the child should be encouraged to maximize topical exposure of the erupted dentition by chewing or sucking fluoride tablets.
Topical Fluoride Treatment

According to the AAPD, professional topical fluoride treatments should be based on caries-risk assessment (AAPD:2007a; AAPD:2007g). Children with moderate caries risk should receive a professional fluoride treatment at least every six months; those with high caries risk should receive them every three to six months. Ideally, this would occur as part of a comprehensive preventive program.

Dentifrice is the most widely used method of applying fluoride topically. Daily or twice-daily fluoride exposure through the controlled use of fluoridated dentifrice is now considered a major approach to the reduction of dental caries. Parents should be counseled on their child’s caries risk and frequency and supervision of tooth-brushing. Additional fluoride therapy should be considered for children at high risk for caries. Home fluoride programs using fluoride mouth rinses or brush-on fluoride gels should be considered for use by school-aged children at high risk for caries.

The concentration of fluoride found in toothpaste is regulated by national regulatory authorities. A level of available fluoride ion in toothpastes between 1,000 and 1,450 parts per million provides a safe and effective fluoride concentration.

Nearly all commercial toothpastes contain some form of fluoride; however, not all fluorides are bioavailable. Only toothpastes having the ADA Seal of Acceptance should be recommended because of the guarantee of bioavailability of fluorides. Fluoridated dentifrices are most effective when
used with a soft-bristle toothbrush, after breakfast and at bedtime. This places fluoride in the oral cavity prior to times of expected low salivary activity, prolonging fluoride availability.

Fluoride toothpastes can contribute to an excessive intake of fluoride and subsequent development of dental fluorosis. Thus, the dental professional must be concerned about the levels of fluoride ingested by children from a fluoridated dentifrice, in addition to their consumption of fluoridated water or use of fluoride supplements during the dental formative years. The current recommendation is that only a pea-sized amount of toothpaste or a “smear layer” of dentifrice should be applied to the toothbrush. Patients should expectorate after brushing to avoid swallowing excess toothpaste or fluoride.

Dental professionals should also be aware of individual patient’s needs for any form of supplemental fluoride, as well as ingredients contained in these products. Some contain sucrose, which can actually contribute to caries. Others contain no dye; one is sweetened with xylitol, which has a noncariogenic effect upon enamel.

**Diet**

The role of sugar in promoting the dental caries process has been established in numerous epidemiological, laboratory, and clinical studies. In preschool children, high-frequency sugar consumption, including its
consumption by means of baby bottles or sippy cups, has been implicated in early childhood caries (Tinanoff:2001; Tinanoff:2002).

Chewing gums with sugar substitutes such as saccharin, aspartame, sorbitol, mannitol, or xylitol should reduce caries risk not only by reducing sugar consumption, but also by stimulating salivary flow and decreasing mutans streptococci colonization (Tinanoff:2001; Tinanoff:2002). The outcomes of several clinical studies show that chewing xylitol-containing gums reduces caries and mutans streptococci levels.

Within the past 20 years, dental researchers have discovered unexpected, beneficial properties of specific foods in preventing tooth decay. These foods include:

• Some cheeses, such as aged cheddar, Monterey Jack, and Swiss
• Peanuts
• Chocolate
• Some cocoa products

Specific types of cheese have been demonstrated to inhibit acid formation when consumed before, during, or after sucrose ingestion. Some cheeses inhibit demineralization and may actually promote remineralization of early enamel lesions. These unique properties have been attributed by researchers to casein contained in the cheese in combination with the stimulation of saliva. Tannins in chocolate and other cocoa products have also been demonstrated to inhibit plaque formation.
Researchers have further found that the physical form of a particular food may affect acid formation in plaque. The drop in plaque pH after consuming unsweetened peanut butter is similar to the pH drop following consumption of a 10% sucrose solution; however, consuming dry-roasted peanuts produces no drop in plaque pH.

**Oral Hygiene**

Poor oral hygiene is widely believed to be a contributor to caries. Thus, tooth brushing, flossing, and professional tooth cleaning have long been considered basic components of programs aimed at preventing caries (Tinanoff:2001; Tinanoff:2002). However, literature reviews on this topic have not found a relationship between dental plaque scores and dental caries prevalence, or between brushing with nonfluoridated toothpaste and dental caries prevalence.

In young children, however, early visible plaque on the labial surfaces of the maxillary incisors is strongly associated with caries development. Furthermore, reductions in caries have been noted in children who receive high-frequency professional prophylaxis combined with some form of fluoride therapy or frequent tooth brushing with fluoridated dentifrice. The specific contribution of the tooth-cleaning procedure as part of these regimens remains unknown. Regular tooth brushing, nevertheless, should be encouraged, at least as a delivery system for the fluoride dentifrice.
According to the AAPD guidelines on dental prophylaxis in pediatric dentistry (AAPD:2007c), a periodic professional prophylaxis should be performed to:

- Instruct the caregiver and child or adolescent in proper oral hygiene techniques
- Remove microbial plaque and calculus
- Polish hard surfaces to minimize the accumulation and retention of plaque
- Remove extrinsic stain
- Facilitate the examination of hard and soft tissues
- Introduce dental procedures to the young child and apprehensive patient

In addition to establishing the need for a prophylaxis, the clinician should determine the most appropriate type of prophylaxis for each patient (AAPD:2007c). To minimize loss of the fluoride-rich layer of enamel during polishing, the least abrasive paste should be used with light pressure. A topical fluoride application is recommended if a rubber cup/pumice prophylaxis is performed.

Caries Risk and Preventive Therapies

Decisions for preventive therapies in primary teeth should be guided by an understanding of risk indicators for the child (Tinanoff:2001;
Tinanoff:2002). Risk-based therapy assumes that there will be little benefit of preventive therapies for those children who are at low risk for dental caries. Conversely, children at high risk require intense prevention to primarily prevent caries initiation and secondarily to arrest caries progression.

**RESTORATIVE THERAPY**

Children at low caries risk may not need any restorative therapy (Tinanoff:2001; Tinanoff:2002). Children at moderate risk may require restoration of progressing and cavitated lesions, while white spot and enamel proximal lesions should be treated by preventive techniques and monitored for progression. However, high-risk children may require earlier restorative intervention of enamel proximal lesions, as well as intervention of progressing and cavitated lesions, to minimize continual caries development. In high-risk cases, treatment of primary teeth with stainless steel crown restorations is better over time than multisurface intracoronal restorations.

**INTERVENTIONS FOR EARLY CHILDHOOD CARIES (ECC)**

The AAPD emphasizes that immediate intervention is required to prevent destruction and more widespread health problems associated with ECC (AAPD:2007e; AAPD:2007f). Prevention of ECC begins with intervention in the
prenatal and perinatal periods. Women should be advised to optimize nutrition during the third trimester and the infant’s first year to avoid enamel hypoplasia. Because cariogenic bacteria may be transmitted to the child, decreasing the mutans streptococci levels in the mother and primary caregivers may decrease the child’s risk of developing ECC (AAPD:2007e; AAPD:2007f).

The ADA urges parents and guardians, as a child’s first tooth erupts, to consult with their dentist regarding scheduling the child’s first dental visit (ADA:2000). The first visit should occur within six months of eruption of the first tooth and no later than 12 months of age. At that time, parents should consult with their dentist about oral health education based on the child’s developmental needs (also known as anticipatory guidance).

The ADA urges its members to educate parents (including expectant parents) and caregivers about reducing the risk for early childhood caries, including the following six points:

- Role of Bacteria. Because decreasing the mother’s mutans levels may decrease the child’s risk of developing ECC, the ADA recommends that parents, including expectant parents, be encouraged to visit a dentist to ensure their own oral health.

- Nutrition. Infants and young children should be provided with a balanced diet in accordance with the Dietary Guidelines for Americans published by the U.S. government. Unrestricted, at-will consumption of beverages and foods containing fermentable carbohydrates can contribute to decay after eruption of the first tooth.
• Bottle Feeding. Unrestricted and at-will intake of sugary liquids during the day or while in bed should be discouraged. Infants should finish their bedtime and nap time bottle before going to bed.

• Breast Feeding. Unrestricted, at-will nocturnal breastfeeding after eruption of the child’s first tooth can lead to an increased risk of caries.

• Use of a Cup. Children should be encouraged to drink from a cup by their first birthday. At-will, frequent use of a training cup should be discouraged.

• Home Care. Proper oral hygiene practices, such as cleaning an infant’s teeth following consumption of foods, liquids, or medication containing fermentable carbohydrates, should be implemented by the time of the eruption of the first tooth. A child’s teeth should be periodically checked at home according to the directions of the dentist.

When very young children develop ECC, therapeutic intervention is warranted (AAPD:2007e; AAPD:2007f). According to the AAPD, the use of anticariogenic agents may reduce the risk of development and progression of caries. Alternative restorative treatment techniques, using materials such as glass ionomers that release fluoride, are efficacious in both preventive and therapeutic approaches. Stainless steel crowns decrease the number of tooth surfaces at risk for new or secondary caries and are less likely than other restorations to require retreatment.
PERIODONTAL DISEASES OF CHILDREN AND ADOLESCENTS

Children and adolescents are subject to several periodontal diseases. Although there is a much lower prevalence of destructive periodontal diseases in children than in adults, children can develop severe forms of periodontitis (Califano:2003). In some cases, this destructive disease is a manifestation of a known underlying systemic disease. In other young patients, the underlying cause for increased susceptibility and early onset of disease is unknown. These diseases are often familial, suggesting a genetic predisposition for aggressive disease.

Gingivitis and Periodontitis

The American Academy of Periodontology position paper on periodontal diseases of children and adolescents, which has been endorsed by the AAPD, notes that gingivitis is common in children (Califano:2003; AAPD:2007d). In addition, children and adolescents can have any of the several forms of periodontitis (aggressive periodontitis, chronic periodontitis, and periodontitis as a manifestation of systemic diseases). However, chronic periodontitis is more common in adults, while aggressive periodontitis may be more common in children and adolescents.

The onset of aggressive periodontitis often occurs around the time of puberty. The primary features of aggressive periodontitis include a history of rapid attachment and bone loss. Aggressive periodontitis is classified as
localized or generalized. Localized aggressive periodontitis patients have interproximal attachment loss on at least two permanent first molars and incisors, with attachment loss on no more than two teeth other than first molars and incisors. Generalized aggressive periodontitis patients exhibit generalized interproximal attachment loss including at least three teeth that are not first molars and incisors.

Successful treatment of aggressive periodontitis depends on early diagnosis, directing therapy against the infecting microorganisms, and providing an environment for healing that is free of infection. A combination of surgical or nonsurgical root debridement and antimicrobial therapy is recommended.

The use of antibiotics (often tetracyclines) is usually beneficial in the treatment of localized aggressive periodontitis. However, generalized aggressive periodontitis does not always respond well to conventional mechanical therapy or to antibiotics commonly used to treat periodontitis. Alternative antibiotics may be required, based upon the character of the pathogenic flora.

Chronic periodontitis is most prevalent in adults, but can occur in children and adolescents. It can be localized (less than 30% of the dentition affected) or generalized (greater than 30% of the dentition affected) and is characterized by a slow to moderate rate of progression that may include periods of rapid destruction.

As with adults, periodontitis associated with systemic diseases occurs in children and adolescents. Such diseases include Papillon-Lefèvre syndrome,
cyclic neutropenia, agranulocytosis, Down syndrome, hypophosphatasia, and leukocyte adherence deficiency. Diabetes is considered a significant modifier of all forms of periodontitis rather than a specific form of periodontitis. Treatment is similar to the treatment of aggressive periodontitis in the permanent dentition and includes debridement and antimicrobial therapy.

**Necrotizing Periodontal Diseases**

Necrotizing periodontal diseases occur with varying but low frequency (less than 1%) in North American and European children. The two most significant findings used in the diagnosis are the presence of interproximal necrosis and ulceration and the rapid onset of gingival pain. Patients can often be febrile. Treatment involves mechanical debridement, oral hygiene instruction, and careful follow-up. Debridement with ultrasonics has been shown to be particularly effective and results in a rapid decrease in symptoms. If the patient is febrile, antibiotics may be an important adjunct to therapy. Metronidazole and penicillin have been suggested as drugs of choice.
REFERENCES


APPENDIX A: COMMONLY AVAILABLE FLUORIDE PRODUCTS

Professional Application

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Self-Applied Mouthrinses/Gels

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<td>Yes</td>
</tr>
<tr>
<td>0.4% SnF₂ gel</td>
<td>968 ppm</td>
<td>Daily</td>
<td>No</td>
</tr>
<tr>
<td>0.05% APF rinse</td>
<td>200 ppm</td>
<td>Daily</td>
<td>No</td>
</tr>
</tbody>
</table>

Note: The 0.63% SnF₂ rinse is diluted to 0.1% before use. Professionally used topical fluorides may be used more frequently in high-risk patients.

Dentifrices

<table>
<thead>
<tr>
<th>Product</th>
<th>Concentration</th>
<th>Frequency</th>
<th>Prescription</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.24% NaF</td>
<td>1,105 ppm</td>
<td>1-3 times daily</td>
<td>No</td>
</tr>
<tr>
<td>0.76% Na₂PO₃F</td>
<td>1,000 ppm</td>
<td>1-3 times daily</td>
<td>No</td>
</tr>
</tbody>
</table>

Varnishes

<table>
<thead>
<tr>
<th>Product</th>
<th>Concentration</th>
<th>Frequency</th>
<th>Prescription</th>
</tr>
</thead>
<tbody>
<tr>
<td>5% NaF</td>
<td>22,600 ppm</td>
<td>2-4 times annually</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Charts adapted from: Recommendations for Fluoride Use in Children.
**FURTHER RESOURCES**

**American Dental Hygienists’ Association**  
444 N. Michigan Ave., Suite 3400  
Chicago, IL 60611  
(312) 440-8949  
“Preventing Baby Bottle Tooth Decay Kit” contains instructional flip chart and two pads of giveaway instructions. (English/Spanish)

**American Society of Dentistry for Children (ASDC)**  
211 E. Chicago Ave., Suite 1430  
Chicago, IL 60611  
(800) 637-2732  
“Baby’s Bright Smile”

**Idaho Department of Health & Welfare**  
Dental Health Section  
Statehouse  
450 W. State Street  
Boise, ID 83720  
(208) 344-4140  
“Keep Your Baby Smiling . . . Prevent Baby Bottle Tooth Decay”

**Los Angeles County Health Services**  
Dental Health Program  
313 N. Figueroa Street, Room 227  
Los Angeles, CA 90012  
(213) 974-8115  
“Nursing Bottle Mouth—WIC Program”

**National Institute of Dental Research (NIDR)**  
National Institutes of Health  
P.O. Box 574-93  
Washington, DC 20032  
(301) 496-4261  
“Prevent Baby Bottle Tooth Decay”
The Test

Now proceed to the test. All questions are constructed using a multiple-choice format.

Take the test by logging in at www.cdabc.org, highlight My Desktop > My Events.

The test will be evaluated immediately after you click Grade Now and upon successful completion, verification of your continuing education credits will be forwarded to you immediately by email. A pass mark of 80% must be achieved to receive continuing education credits. Should you not obtain a passing score, you will be notified immediately and given the opportunity to complete the test again. A maximum of four attempts is provided. Credit/course refunds are not issued for courses not passed within four attempts.
1. What percentage of American children ages two through eleven have untreated tooth decay in their primary dentition?
   A. 77%
   B. 41%
   C. 21%
   D. 15%

   A. 15% decrease
   B. 15% increase
   C. 30% increase
   D. 50% decrease

3. Children in which ethnic group reported a higher caries experience?
   A. Mexican-Americans
   B. African Americans
   C. Caucasians
   D. Asians/Pacific Islanders

4. Which of the following is one of the three variable factors that interact to form caries?
   A. Nonfermentable carbohydrates
   B. Pathogenic oral microorganisms in dental plaque
   C. Oral hygiene
   D. Dietary fluoride

5. Which of the following is a microbial product in dental plaque?
   A. Adhesive glucans
   B. Adhesive streptans
   C. Cohesive aminoglycosides
   D. Phosphodiesterase

6. What is the magnitude of the effect that saliva has on caries development?
   A. None
   B. Minor
   C. Major
   D. Inverse
7. Which microorganism has been implicated as the predominant agent in dental caries?
   A. Streptococcus sobrinus
   B. Lactobacilli
   C. Candida albicans
   D. Mutans streptococcus

8. Which of the following is the most important virulence factor of mutans streptococci?
   A. Amylase
   B. Glucosyltransferase
   C. Phosphodiesterase
   D. Acetylcholinesterase

9. Which of the following is the AADP/ADA definition of early childhood caries?
   A. ≥ 1 DMFS on primary teeth in preschool-age child between birth and 71 months of age
   B. ≥ 1 DMFS on permanent teeth in preschool-age child between birth and 71 months of age
   C. ≥ 2 DMFS on primary teeth in preschool-age child between birth and 71 months of age
   D. ≥ 1 DMFS on primary maxillary anterior teeth in preschool-age child between birth and 71 months of age

10. A child who has been identified as being at low risk for dental caries may need few diagnostic procedures and preventive therapies.
   A. True
   B. False

11. Which of the following is a more critical issue in caries diagnosis after determining whether a tooth is cavitated or not?
    A. The dimensions of the lesion
    B. The microbiota associated with the lesion
    C. Whether a lesion is progressing or arrested
    D. The number of virulence factors

12. Which of the following is one of several other useful caries risk indicators in preschool children?
    A. High birth weight
    B. Previous caries experience
    C. Child’s lactobacilli levels
    D. Baseline caries scores
13. The AAPD treatment guidelines recommend that systemically administered fluoride supplements be considered in which of the following?
   A. In children with ECC drinking fluoride-deficient (<0.6 ppm) water
   B. In all children
   C. In all children drinking fluoride-deficient (<0.6 ppm) water
   D. In all children older than age five

14. Which of the following is the recommended schedule of professional fluoride treatment in children with high caries risk?
   A. Twice daily
   B. Every three to six months
   C. Every six months
   D. Every year

15. How much fluoride dentifrice is recommended as an application on a children’s toothbrush?
   A. As little as possible
   B. Enough to smear over all tooth surfaces
   C. An amount the size of a penny
   D. An amount the size of a pea or a ‘smear layer’

16. Literature reviews have found a relationship between brushing with nonfluoridated toothpaste and dental caries prevalence.
   A. True
   B. False

17. What kind of restorative therapy may be required for children at moderate caries risk?
   A. Restoration of progressing and cavitated lesions
   B. Full crown coverage of all cavitated teeth
   C. Restoration of all white spot and enamel proximal lesions
   D. Only monitoring

18. When does intervention to prevent ECC begin?
   A. Upon discovery of the first plaque
   B. Upon eruption of the first tooth
   C. After the infant’s first year
   D. In the prenatal and perinatal periods
19. For which periodontal disease is the use of antibiotics (often tetracyclines) usually beneficial?
   A. Generalized aggressive periodontitis
   B. Localized aggressive periodontitis
   C. Gingivitis
   D. Papillon-Lefèvre syndrome

20. Which of the following results in rapid decreases in necrotizing periodontal disease symptoms?
   A. Early diagnosis
   B. Alternative antibiotics
   C. Ultrasonic debridement
   D. Extended use of a no-spill training cup