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Contemporary noninvasive dental caries treatment

A PEER-REVIEWED ARTICLE | by Lisa Mayo, MHA, BSDH, RDH

ABSTRACT

Dental caries is a major global public health crisis affecting almost half the world's population. It is one of the greatest unmet health-care needs of the 21st century and is the most prevalent preventable disease condition as reported by the World Health Organization. Dentistry has undergone a paradigm shift in noncavitated and cavitated carious lesions. This course will present the latest research on noninvasive treatment modalities for carious lesions that will promote healthier lives for patients and can assist in avoiding more invasive restorative procedures..

EDUCATIONAL OBJECTIVES

Upon completion of this course, the dental professional should be able to:

1. Define and identify a noncavitated and cavitated carious lesion, demineralization, and remineralization
2. Perform an accurate carious risk assessment that drives preventive and therapeutic recommendations
3. Explain the mechanism of action of neutral sodium fluoride, silver diamine fluoride, calcium-phosphate products, and peptide P_{11}-4
4. Select the appropriate chemotherapeutic intervention based on patient presentation for the treatment of noncavitated and cavitated carious lesions

Introduction

A dental carious lesion is the "clinical manifestation of caries disease" on an erupted tooth surface. It can be noncavitated or cavitated.[1]

A cavitated carious lesion occurs when there is loss of enamel and the lesion has invaded the underlying dentin (figures 1a-1b).[1] A cavitated carious lesion is irreversible and requires removal of the lesion to arrest its progression because enamel cannot regenerate or repair itself.[1-3]

A noncavitated carious lesion is referred to as an initial or early lesion and occurs when enamel loses minerals with microscopic breakdown. Enamel will discolor, causing white spot lesions (figures 2a-2b).[1] A noncavitated lesion may be reversed with nonsurgical approaches such as biofilm modification, chemotherapeutic (fluoride, calcium-phosphate products, peptide P_{11}-4) interventions, and patient behavior changes.[1,3]

Dental caries is a preventable, multifactorial, and complex disease involving personal (poor oral hygiene habits, systemic diseases, medications), biological (genetic predisposition), behavioral (diet, tobacco), psychosocial (value of oral health), and environmental (low socioeconomic status,

access to dental care) factors.[1,3] A carious lesion is more likely to form when these risk factors outweigh the protective capabilities of the oral microbiome.[3]

An untreated cavitated carious lesion will lead to many adverse sequelae, such as a decreased quality of life due to pain, anxiety associated with the condition, loss of sleep, change in eating habits, adverse growth patterns, and tooth loss. Advanced lesions can lead to infection, abscess, sepsis, aggravation of systemic diseases, and even loss of life (figures 3a-3b).[4,5]

Dental caries statistics

Global outlook: Dental caries is a major public health crisis affecting almost half the world's population and is the most prevalent preventable disease condition as reported by the Global Burden of Disease 2019.[6] Globally, dental caries ranks first for permanent tooth decay (2.3 billion) and 12th (560 million) for deciduous (primary, baby) teeth.[4] The treatment for dental caries makes up 5% to 10% of global health-care budgets ($298 billion) and is the main reason for the hospitalization of children in many high-income countries.[4]

Global policies have traditionally not mentioned dental caries, and "dental caries is not currently visible in global or national noncommunicable disease (NCD) strategies."[3] Dentistry's current preventive interventions appear to be insufficient to meet the health needs of the public because the incidence and prevalence of dental caries has not declined since 1990.[5]

United States outlook: In the United States, one in four adults ages 20-64 currently has at least one cavity, over half (52%) of children ages 6-8 have had a cavity in their primary teeth, and 57% of adolescents ages 12-19 have had a cavity in their permanent teeth.[7] Everyone is at risk for dental caries, but children and adolescents have the highest risk, with more disparity for those raised in low socioeconomic families.[4,7] Children from lower-income families are two times as likely to have untreated caries than those raised in higher-income families.[7] It is evident from these statistics that dental caries is an uncontrolled pandemic requiring a call to action from oral health professionals and governments to provide care for one of this generation's greatest unmet health-care needs.

FIGURE 1: Cavitated carious lesions. Cavitated lesions on the maxillary central incisors (a); cavitated lesion on the maxillary left lateral incisor (b)

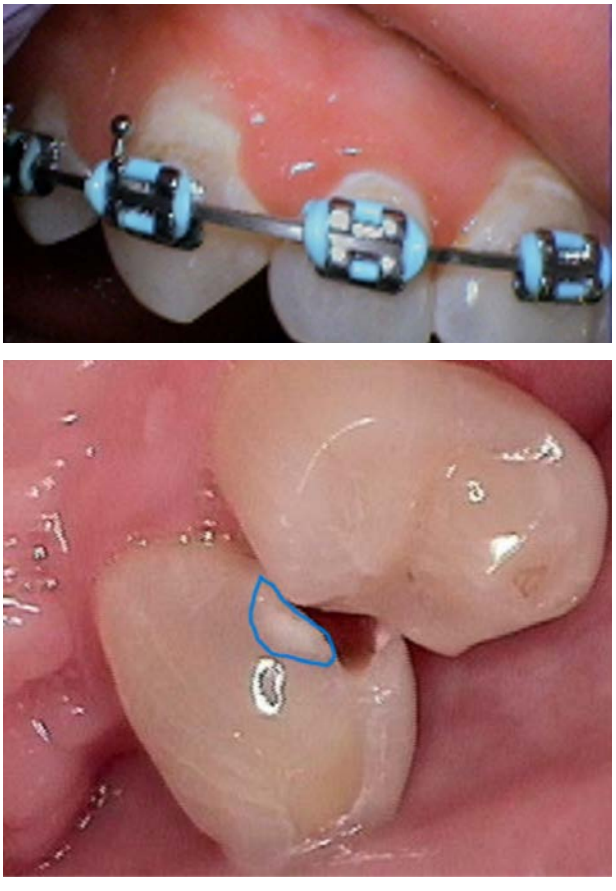

FIGURE 2: Enamel discoloration. White-spot lesions in the cervical third of the tooth above the orthodontic brackets (a); white-spot discoloration in the enamel around the cavitated lesion (b)

Tooth histology and embryology

Enamel, dentin, and cementum are the hard tissues that make up the structure of a tooth. Unlike dentin and cementum, enamel is completely formed before tooth eruption and is unable to repair itself over the course of a person's lifetime due to its lack of regenerative cells and vascularization.[8-10] Histologically, enamel, dentin, and cementum vary in their mineral, protein, and water content. Enamel has the greatest percentage of calcified content (96%) while dentin has 70%, and cementum has only 45% to 50%, making enamel the hardest substance in the vertebrate body.[8,9,11,12] Enamel contains 96% mineral content (hydroxyapatite), 2% organic, and 2% water by weight.[9] Embryologically, hydroxyapatite is formed from calcium, phosphate, and water in the enamel matrix. The hydroxyapatite crystals are hexagonal in shape and are housed inside oval-shaped enamel rods (or prisms) that extend from the dentinoenamel junction (DEJ) to the surface of the tooth.[9]

FIGURE 3: Abscess from untreated dental caries maxillary right central incisor. Radiograph (a); visible abscess (b)

Caries etiology and process

Enamel is coated with an acquired pellicle, which is made up of a variety of proteins, glycoproteins, and mucins that come from saliva, oral bacteria, and gingival crevicular fluid. The proteins create a calcium-rich environment on the tooth surface, and the pellicle provides a barrier to the diffusion of bacterial acids. Unfortunately, the proteins also contain binding sites for oral bacteria to establish biofilm formation (figure 4).

An oral biofilm is a collection of microbial cells that bind together on a surface and form an enclosed matrix held together by polysaccharides that irreversibly bind to a surface. They form highly regulated, complex, organized communities that attach to hard and soft tissues of the mouth. An oral biofilm is composed of many different organisms, and when it grows uncontrolled, a state of oral dysbiosis occurs, and pathogenic (capable of causing disease) bacteria begin to dominate the oral microbiome.

Dental caries is a biofilm-mediated, diet-modulated, multifactorial disease with a broad etiology.[3]

Oral biofilms create acid as a byproduct of their metabolism of carbohydrates, which causes demineralization of hard tissues.[2,3,4,13] Demineralization is the process of mineral ion loss from hydroxyapatite crystals in which calcium ions are removed first, followed by phosphate, resulting in a weakened and porous tooth structure and chalky white discoloration[2]

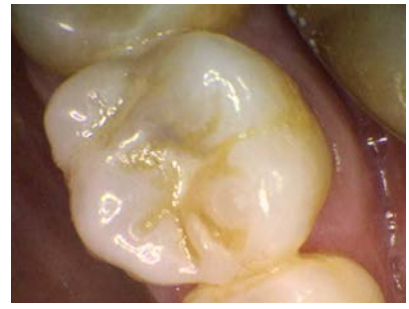

FIGURE 4: Maxillary molar coated with acquired pellicle and biofilm

FIGURE 5: Demineralization on mesial surfaces of the maxillary central incisors

(figure 5). Demineralization of enamel will occur when the pH falls to 5.0-5.5 in the presence of fermented bacterial acids.[9]

A diet high in sugar and acidic foods and drinks contributes to higher rates of dental caries.[4] The World Health Organization recommends limiting free sugar intake to less than 10% of total energy consumption, and when values drop below 5%, this will decrease the risk of dental caries throughout one's lifetime.[4]

Enamel can remineralize (restoration of lost mineral ions back into hydroxyapatite) if biofilm and acids are removed and there are sufficient quantities of calcium, phosphate, and fluoride present in the mouth. When fluoride ions are incorporated into

hydroxyapatite crystals, fluorohydroxyapatite (FAP) is formed. While enamel cannot repair itself since it is fully formed upon eruption, it can go through posteruptive maturation, in which it becomes more resistant to acid attacks through the formation of less soluble fluorohydroxyapatite.[9]

Caries classification

There are many caries classification systems used in the dental field. Carious lesions have different clinical presentations throughout the disease process. It is important to incorporate a classification system into your practice that specifies lesion location, site of origin, extent, and activity to support treatment decisions using the most current nonsurgical and surgical approaches to caries management.

CARIES CLASSIFICATION EXAMPLES

- G. V. Black: This system classifies caries by location and surface involvement. It is commonly used for teeth needing restorative (operative, surgical) interventions and treatment. It does not address noncavitated lesions or distinguish the depth of lesions.[1,14]
- American Dental Association Caries Classification System (CCS): This system includes noncavitated and cavitated carious lesions and describes them by their clinical presentation without reference to a specific treatment approach.[1] Carious lesions are classified as healthy/sound, initial, moderate, or advanced.[1] CCS links the clinical lesion presentation to radiographic findings and provides an approach to identify and track lesion activity over time.[1]
- International Caries Detection and Assessment System (ICDAS): This system includes noncavitated and cavitated carious lesions using visual surface characteristics to measure changes and potential histologic extent and activity of carious lesions.[1] ICDAS-I was released in 2002 and ICDAS-II in 2005. There are six stages to classify the caries process ranging from no disease to extensive lesions with recommendations for treatment approaches.
- International Caries Classification and Management System (ICCMS): This system classifies tooth surfaces from healthy to severe decay and allows for early detection of enamel changes that will benefit from remineralization. ICCMS provides a comprehensive set of clinical protocols that address diagnostic, preventive, and restorative decisions to best manage noncavitated and cavitated carious lesions.[1,14]

Caries risk assessment

The American Dental Association offers a comprehensive caries management by risk assessment (CAMBRA) for pediatric and adult patients that can easily be implemented into clinical practice to help drive patient recommendations with low, moderate, or high ratings. CAMBRA is an evidence-based assessment that compares a patient's caries risk factors, such as lifestyle, diet, systemic health, presence of harmful bacteria, and saliva alterations, and weighs them against their protective factors.

The caries risk assessment forms from the ADA "are not intended to include all possible risk factors. The risk factors selected are intended to provide the patient with information that may help them lower caries risk over time, while also providing a form that can be integrated into a busy practice setting."[15] Dental providers use the information gathered from CAMBRA to determine patient management and treatment strategies that include, but are not limited to, radiographic frequency, recall frequency, nutritional counseling, fluoride selection, oral hygiene aids, and home-care chemotherapeutics.

Remineralization therapies

The goal of remineralization therapy is to arrest the progression of an active noncavitated carious lesion with a chemotherapeutic agent used at home or applied in the office. Professionally applied agents differ in their formulations and concentrations from in-office agents. Many times, a combination of both is needed to decrease a patient's overall CAMBRA risk rating.

Neutral sodium fluoride: Fluoride, in its various delivery and application forms, has been the dominant medicament used for caries prevention as its effect on hard tissues is extensively documented owing to its bacteriostatic and bactericidal properties.[9,13,16,17] Neutral sodium fluoride is available as an at-home product in gels, rinses, and pastes with concentrations of 5,000 ppm and lower.[14] In-office neutral sodium fluoride is available as a 2% gel or foam with 9,050 ppm and as a 5% varnish with 22,600 ppm.[14] These topical applications increase the fluoride concentration that is available in saliva, within a biofilm, and on tooth surfaces.[9] In saliva and on tooth surfaces, fluoride is present as calcium fluoride (CaF_2). In biofilm, fluoride reacts with bacterial calcium to form calcium-fluoride (Ca-F) bonds.

When the pH of the mouth decreases, the calcium and fluoride ions retained in the outer mineral surface layer of the enamel are released and become readily available for enamel remineralization because fluoride has a high affinity for hydroxyapatite. The calcium in hydroxyapatite is displaced by fluoride, forming fluorohydroxyapatite, which is less soluble than the calcium-deficient hydroxyapatite of natural enamel.[8]

Neutral sodium fluoride has limitations in its ability to reverse active noncavitated carious lesions. When topically applied, neutral sodium fluoride will only penetrate approximately 40 µm into the outer surface of the tooth, and active noncavitated lesions are typically much deeper than 1 mm in depth prior to cavitation.[18] When a noncavitated carious lesion progresses deeper than 40 µm, the fluoride ions are unable to penetrate to the depth (subsurface) of the lesion where remineralization is needed.[13]

Calcium fluoride products: To form fluorohydroxyapatite, for every two fluoride ions, 10 calcium ions and six phosphate ions are needed.[8] When calcium and phosphate ions are lacking in the mouth, remineralization is hindered. Calcium-phosphate products were developed under this chemical principle, and there are multiple formulations for the dental professional to choose from.

- **ACP:** Amorphous calcium phosphate was the first product available on the market. Once ACP dissolves in enamel fluids, calcium and phosphate ions precipitate and recrystallize as apatite. The problem with ACP is that it has low substantivity and high solubility, and once rinsed away is no longer bioavailable in the mouth.[18]
- **CPP-ACP (Recaldent):** This chemical formulation combines casein phosphopeptide (CPP) with amorphous calcium phosphate (ACP).[8] CPP are peptides derived from a milk protein, casein. When CPP is added to ACP, it stabilizes and increases its substantivity.[19] CPP-ACP contributes to remineralization by buffering free calcium and phosphate ions in biofilm, antagonizing (opposing) bacterial adhesion to tooth surfaces, and provides a calcium- and phosphate-rich environment for enamel.[19] This product is also available with added neutral sodium fluoride.
- **TCP:** This combines beta (β)-tricalcium phosphate and a surfactant (sodium lauryl sulfate) to form a more functionalized calcium phosphate. TCP provides a slow release of calcium onto tooth surfaces as it contacts saliva, and allows for crystal modification in enamel lattice defects.[20]
- **NovaMin:** This product is a bioactive glass composed of calcium, sodium, phosphorous, and silicon, known as calcium sodium phosphosilicate. NovaMin has antibacterial effects on cariogenic bacteria and initiates remineralization by developing apatite on the surface of demineralized hard tissues.[21] NovaMin releases sodium ions first when it contacts saliva, and the pH of the mouth will rise to a level that is needed for hydroxyapatite formation. Calcium and phosphate ions release second to form calcium hydroxycarbonate that will remineralize the affected area.[21]
- **Nano-hydroxyapatite:** $Ca_{10}(PO_4)_3(OH)_2$/zinc carbonate-hydroxyapatite nanocrystals (nHA) "is a bioactive compatible material with similar chemical composition to the apatite crystals of human enamel" and induces remineralization. It is available in toothpaste formulations, and when present in 10% concentration, has been shown in studies to cause an enamel mineral gain.[22]

Silver diamine fluoride: $Ag(NH_3)_2F$ is a liquid topical medicament composed of 24.4% to 28.8% silver, 5% to 5.9% fluoride, and ammonia.[23,24]

- Silver: antimicrobial agent that disrupts cell membranes, denatures proteins, inhibits DNA replication, and leads to bacterial cell death[23,25]
- Fluoride: promotes remineralization[23,25,26]
- Ammonia: stabilizes the solution[23,25,26]

Silver diamine fluoride is FDA approved in the US for tooth sensitivity owing to its ability to occlude dentinal tubules. It is also used off-label as a noninvasive method for caries arrest of active noncavitated and cavitated lesions.[2,26] For complete arrest, silver diamine may need reapplication and will cause a permanent dark discoloration to the carious lesion (figure 6).[2,24,25,27] When silver diamine is used in the treatment of halting caries progression, the procedure is referred to as atraumatic restorative treatment (ART).[26]

Silver diamine fluoride is commonly used in the treatment of carious lesions in primary teeth, permanent teeth that are not in the smile line, root surface lesions, molar/incisor hypomyelination, and for

FIGURE 6: Silver diamine fluoride discoloration on maxillary left primary molars

pediatric patients who cannot tolerate restorative procedures, have special needs, or are uncooperative.

Peptide P_{11}-4: Amino acids are organic molecules that are the building blocks for protein in the human body. Amino acids are covalently linked to one another via peptide bonds.[28] A polypeptide is a chain of eight or more amino acids. Proteins are made of hundreds of amino acids and are some of the largest macromolecules in a cell.[28] Proteins are responsible for multiple functions in the human body, such as repairing and building body tissues, transporting and storing nutrients, promoting immunity (antibodies are proteins), and maintaining metabolic pH and fluid balance.[28]

- Self-assembling β-sheet-forming peptides have been shown to form three-dimensional fiber networks, supporting tissue regeneration, and can be made synthetically in a lab. These peptides mimic the body's natural properties of self-assembling peptides.[10,29] Dentistry now has a peptide product available that is composed of 11 amino acids: peptide P_{11}-4 (Curodont Repair/Curodont Repair Fluoride Plus Credentis) that is used to regress, repair, and regenerate damaged enamel caused by active noncavitated carious lesions.[2,13,16,30,31]

FIGURE 7: Peptide P_{11}-4 (Curodont Repair Fluoride Plus). Curodont Repair applied to the facial interproximal of maxillary central incisors (a); Curodont Repair applied to the lingual interproximal of maxillary central incisors (b)

CURODONT REPAIR'S MECHANISM OF ACTION:

- When applied, peptide P_{11}-4 (Curodont Repair) diffuses to the subsurface depth of an active noncavitated carious lesion (figures 7a-7b). The chemical penetrates deep into the lesion, unlike topically applied fluoride.
- Peptide monomers self-assemble into a 3-D scaffold that forms a matrix.
- Calcium is attracted from saliva to the binding sites on the matrix.
- The matrix and calcium template for the formation of new (de novo) hydroxyapatite and fluoridated hydroxyapatite crystals, resulting in increased microhardness within the lesion body.[2,10,13,16,30,32]

Microscopically, the scaffold is not arranged in a parallel prismatic structure but forms in a fanlike fashion.[13] This has a minor effect on the optical properties of the regenerated enamel where the discoloration of white-spot lesions is significantly reduced but may not completely disappear, because the opacity of the natural enamel originated from the prismatic arrangement of the hydroxyapatite crystals inside the enamel rods.[13] These optical changes are more pronounced in deeper enamel lesions than shallow ones. If providers intervene earlier in the caries process and apply peptide P_{11}-4, persistent white-spot discolorations can be minimized.

According to a systematic review published in the *Journal of the American Dental Association* in 2023,

peptide P_{11}-4 (Curodont Repair) is recognized as "a noninvasive intervention that regenerates enamel in initial carious lesions."[2] The systematic review found that "73% of all Curodont Repair treated lesions were anticipated to arrest" and of those lesions, "33% would not have arrested without peptide P_{11}-4 intervention."[2]

Curodont Repair Fluoride Plus became registered with the FDA in 2019 under the fluoride monograph.[2] The 2024 CDT book released the code for application: D2991 *Application of hydroxyapatite regeneration medicament – per tooth. Preparation of tooth surfaces and topical application of a scaffold to guide hydroxyapatite regeneration.*[33] Curodont Repair Fluoride Plus is kept as a lyophilized powder (powder made by freeze-drying) and rehydrated before application.[2] Curodont Repair Fluoride Plus contains 500 ppm neutral sodium fluoride. It is tasteless and does not stain tooth surfaces.[2]

Conclusion

Dental professionals have a call to action to increase interventions in response to the dental caries pandemic. Noninvasive chemotherapeutic medicaments such as neutral-sodium fluoride, silver diamine fluoride, calcium-phosphate products, and peptide P_{11}-4 are easily incorporated into patient care plans and can assist patients in avoiding more invasive restorative procedures due to the presence of noncavitated and cavitated carious lesions. In addition, oral health-care providers should perform a caries-risk assessment and provide patients with recommendations to decrease their risks.

Author's disclosure

The author has no affiliations with any company that would have a gained interest in the material published in this course. There was no corporate sponsor in the making of this course, and the author is not employed by a company that would stand to profit by the publication of this course. All research is presented in an unbiased manner.

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1. Which of the following terms refers to a carious lesion with total loss of enamel and exposure of the underlying dentin?
   - A. Cavitation
   - B. Noncavitation
   - C. Initial carious lesion
   - D. Early carious lesion

2. Which of the following is the most prevalent preventable disease as reported by the Global Burden of Disease in 2019?
   - A. High blood pressure
   - B. Diabetes
   - C. Dental caries
   - D. Obesity

3. What percentage of global health-care budgets does the treatment of dental caries make up?
   - A. 1-2%
   - B. 5-10%
   - C. 20-30%
   - D. 40-50%

4. Children from lower socioeconomic families are how many times more likely to have untreated caries than higher-income families?
   - A. Two times
   - B. Three times
   - C. Four times
   - D. Six times

5. In the United States, how many adults currently have at least one cavity?
   - A. 1 in 2
   - B. 1 in 3
   - C. 1 in 4
   - D. 1 in 5

6. What percentage of US children ages 6-8 have had a cavity in their primary teeth?
   - A. 15%
   - B. 25%
   - C. 35%
   - D. 52%

7. What percentage of US adolescents ages 12-19 have had a cavity in their permanent teeth?
   - A. 15%
   - B. 25%
   - C. 35%
   - D. 57%

8. Which of the following hard tissues is completely formed before tooth eruption and is unable to repair itself throughout a person's lifetime?
   - A. Epithelium
   - B. Cementum
   - C. Enamel
   - D. Dentin

9. What percentage of calcified content is present in enamel?
   - A. 25%
   - B. 45%
   - C. 60%
   - D. 96%

10. During demineralization, which ions are removed from hydroxyapatite crystals first?
   - A. Sulfur
   - B. Calcium
   - C. Carbon
   - D. Hydrogen

11. Demineralization of enamel will occur when the pH of the mouth drops to:
   - A. 2.0-3.0
   - B. 3.0-4.0
   - C. 5.0-5.5
   - D. 7.0-8.0

12. What structure is formed when fluoride ions are incorporated into hydroxyapatite crystals?
   - A. Carbonate hydroxyapatite
   - B. Fluorohydroxyapatite
   - C. Sodium hydroxyapatite
   - D. Phosphate hydroxyapatite

13. Which of the following is a risk factor for dental caries?
   - A. Poor oral hygiene
   - B. Diet high in sugar
   - C. Low socioeconomic status
   - D. All of the above

14. Which caries classification system uses the terms "healthy/sound, initial, moderate, and advanced" when describing carious lesions?
   - A. G. V. Black
   - B. American Dental Association Caries Classification System
   - C. International Caries Detection and Assessment System
   - D. International Caries Classification and Management System

15. Which caries classification system classifies caries only by their location and surface involvement?
   - A. G. V. Black
   - B. American Dental Association Caries Classification System
   - C. International Caries Detection and Assessment System
   - D. International Caries Classification and Management System

16. Which of the following is true of fluoride?
   - A. Bacteriostatic
   - B. Bactericidal
   - C. Has a high affinity for hydroxyapatite
   - D. All of the above

17. How many ppm are present in 2% neutral sodium gel/foam applied in-office?
   - A. 1,000 ppm
   - B. 5,000 ppm
   - C. 9,050 ppm
   - D. 22,600 ppm

18. How many ppm are present in 5% neutral sodium varnish applied in-office?
   - A. 1,000 ppm
   - B. 5,000 ppm
   - C. 6,000 ppm
   - D. 22,600 ppm

19. How deep can neutral sodium fluoride penetrate into the outer surface of a tooth?
- A. Approximately 40 µm
- B. Approximately 80 µm
- C. Approximately 100 µm
- D. Approximately 200 µm

20. Which of the following calcium-phosphate products combines casein phosphopeptide with amorphous calcium phosphate?
- A. ACP
- B. CPP-ACP
- C. TCP
- D. NovaMin

21. Which of the following calcium-phosphate products is made of beta-tricalcium phosphate and sodium lauryl sulfate?
- A. Nano-hydroxyapatite
- B. CPP-ACP
- C. TCP
- D. NovaMin

22. Which of the following calcium-phosphate products is a bioactive glass composed of calcium, sodium, phosphorous, and silicon?
- A. Nano-hydroxyapatite
- B. CPP-ACP
- C. TCP
- D. NovaMin

23. Which of the following is found in silver diamine fluoride?
- A. Silver
- B. Fluoride
- C. Ammonia
- D. All of the above

24. Which chemical can be used for the treatment of a cavitated carious lesion?
- A. Neutral sodium fluoride varnish
- B. ACP-CPP
- C. Silver diamine fluoride
- D. Stannous fluoride

25. Which chemical is used for the procedure atraumatic restorative treatment (ART)?
- A. Neutral sodium fluoride varnish
- B. ACP-CPP
- C. Silver diamine fluoride
- D. Peptide P_{11}-4

26. Which of the following are the building blocks for protein synthesis in the human body?
- A. Amino acids
- B. Carbohydrates
- C. Fats
- D. Vitamins

27. How many amino acids combine to form peptide P_{11}-4?
- A. 0
- B. 1
- C. 2
- D. 11

28. Which of the following products can penetrate to the depth of a noncavitated carious lesion and regenerate and repair the damaged enamel?
- E. Peptide P_{11}-4
- F. ACP
- G. Neutral sodium fluoride
- H. CPP-ACP

29. When applied to a carious lesion, which of the following chemicals forms new (de novo) hydroxyapatite and fluoridated hydroxyapatite crystals?
- A. Neutral sodium fluoride varnish
- B. ACP-CPP
- C. Silver diamine fluoride
- D. Peptide P_{11}-4

30. Which of the following chemicals is approved in the US by the FDA as an anticaries drug under the fluoride monograph?
- A. Neutral sodium fluoride varnish
- B. ACP-CPP
- C. Silver diamine fluoride
- D. Peptide P_{11}-4

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Contemporary noninvasive dental caries treatment

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EDUCATIONAL OBJECTIVES

1. Define and identify a noncavitated and cavitated carious lesion, demineralization, and remineralization
2. Perform an accurate carious risk assessment that drives preventive and therapeutic recommendations
3. Explain the mechanism of action of neutral sodium fluoride, silver diamine fluoride, calcium-phosphate products, and peptide P_{11}-4
4. Select the appropriate chemotherapeutic intervention based on patient presentation for the treatment of noncavitated and cavitated carious lesions

COURSE EVALUATION

1. Were the individual course objectives met?
   Objective #1: Yes No Objective #3: Yes No
   Objective #2: Yes No Objective #4: Yes No

Please evaluate this course by responding to the following statements, using a scale of Excellent = 5 to Poor = 0.

2. To what extent were the course objectives accomplished overall?	5	4	3	2	1	0
3. Please rate your personal mastery of the course objectives.	5	4	3	2	1	0
4. How would you rate the objectives and educational methods?	5	4	3	2	1	0
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8. Please rate the usefulness and clinical applicability of this course.	5	4	3	2	1	0
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10. Do you feel that the references were adequate?	Yes	No				
11. Would you take a similar course on a different topic?	Yes	No				

12. If any of the continuing education questions were unclear or ambiguous, please list them.

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14. How long did it take you to complete this course?

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7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	22.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	23.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	24.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	25.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	26.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	27.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	28.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	29.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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