Controversial Issues in Dentistry

HOW TO RECEIVE CREDIT

- Read the enclosed course.
- Complete the questions at the end of the course.
- Return your completed Answer Sheet to NetCE by mail or fax, or complete online at www.NetCE.com. Your postmark or facsimile date will be used as your completion date.
- Receive your Certificate(s) of Completion by mail, fax, or email.

Faculty
Mark J. Szarejko, DDS, FAGD, received his dental degree from the State University of New York at Buffalo in 1985. He received fellowship from the Academy of General Dentistry in 1994.

Faculty Disclosure
Contributing faculty, Mark J. Szarejko, DDS, FAGD, has disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

Director of Development and Academic Affairs
Sarah Campbell

Director Disclosure
The director has disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

Audience
This course is designed for dental professionals in all practice settings.

Accreditations & Approvals
NetCE is an ADA CERP Recognized Provider.

ADA CERP is a service of the American Dental Association to assist dental professionals in identifying quality providers of continuing dental education. ADA CERP does not approve or endorse individual courses or instructors, nor does it imply acceptance of credit hours by boards of dentistry.

Concerns or complaints about a CE provider may be directed to the provider or to ADA CERP at www.ada.org/cerp.

NetCE
Nationally Approved PACE Program Provider for FAGD/MAGD credit.
Approval does not imply acceptance by any regulatory authority or AGD endorsement.
10/1/2021 to 9/30/2027
Provider ID #217994.

NetCE is a Registered Provider with the Dental Board of California. Provider number RP3841. Completion of this course does not constitute authorization for the attendee to perform any services that he or she is not legally authorized to perform based on his or her permit type.

NetCE is approved as a provider of continuing education by the Florida Board of Dentistry, Provider #50-2405.

Designations of Credit
NetCE designates this activity for 5 continuing education credits.

AGD Subject Code 750.

This course meets the Dental Board of California's requirements for 5 units of continuing education.

Dental Board of California course #05-3841-00376.

About the Sponsor
The purpose of NetCE is to provide challenging curricula to assist healthcare professionals to raise their levels of expertise while fulfilling their continuing education requirements, thereby improving the quality of healthcare.

A complete Works Cited list begins on page 23.

Mention of commercial products does not indicate endorsement.

Our contributing faculty members have taken care to ensure that the information and recommendations are accurate and compatible with the standards generally accepted at the time of publication. The publisher disclaims any liability, loss or damage incurred as a consequence, directly or indirectly, of the use and application of any of the contents. Participants are cautioned about the potential risk of using limited knowledge when integrating new techniques into practice.

Disclosure Statement

It is the policy of NetCE not to accept commercial support. Furthermore, commercial interests are prohibited from distributing or providing access to this activity to learners.

Course Objective

The purpose of this course is to provide factual information about controversial topics in dentistry, allowing professionals to objectively assess the issues and discuss them with patients and other professionals.

Learning Objectives

Upon completion of this course, you should be able to:

1. Discuss the relative safety of amalgam restorative material.
2. Outline the issues that surround the use of fluoride in public water supplies.
3. Describe situations that do and do not require the use of antibiotics in dentistry.
4. List dental procedures and medications that are safe for pregnant patients.
5. Evaluate the data that suggest a link between periodontal disease and certain systemic illnesses.
6. Identify dental concerns related to alternatives to smoking, such as e-cigarettes, hookahs, and smokeless tobacco.
7. Evaluate issues related to access to dental care within the United States.

INTRODUCTION

Dentistry is a profession in which exacting measurements can dictate procedural success or failure. The margin of a crown that is open by only 0.1 mm will allow for bacterial ingress and the development of recurrent caries and the ultimate failure of the restoration. Minute errors in the angulation of the placement of implants can compromise prosthetic function and aesthetics. Periodontal pathology and the success of periodontal treatment are measured by millimeters.

However, there are many controversial issues in the dental profession that are more subjective in nature and for which definitive answers are not available. This course will highlight issues that are of current interest and that have differing views among members of the dental profession, members of other allied health professions, and/or the general public. Course participants will be provided with an analysis of the underlying elements of these controversial issues, which should allow them to make an informed decision about each.

DENTAL AMALGAM: IS IT SAFE?

The use of restorative materials to repair carious lesions predates modern times. As early as 1578, Li Shizhen recorded the use of a dental formulation, the composition of which was 100 parts mercury coupled with 45 parts silver and 900 parts tin [1]. In 1826, Taveau mixed melted silver coins and mercury to produce a silver paste that was used as a filling material [2]. G.V. Black, the first dentist to develop the scientific principles of cavity design, advanced the use of an alloy composed of 68.5% silver, 25.5% tin, 5% gold, and 1% zinc that could be combined with mercury to form the premier dental restorative material of its time [3]. While the composition of these alloys may seem rudimentary, they are indeed the predecessors of modern-day dental amalgam alloys.

The current composition of dental amalgam is 40% to 70% silver, 12% to 30% tin, and 12% to 24% copper, with the quantitative ranges reflecting variations in the manufacturing processes. Trace amounts of indium, palladium, and zinc may also be included in the mixture [4]. The combination of these metals is useless as a restorative dental material unless they are combined with mercury, which creates a pliable restorative material that can be condensed into cavity preparations and that hardens soon after its placement. Mercury comprises about 42% to 45% of the total weight of the combined amalgam alloy [5].

Amalgam has been used to restore millions of carious lesions on several generations of patients. It is a versatile material that is easy to place, can withstand the cumulative load-bearing forces of occlusion for an extended duration, and has been the most cost-effective among all dental restorative materials. Dental amalgam restorations have often been called "silver fillings," because when they are polished after placement, the amalgam surface has a luster that is similar to silver. However, oxidation reactions of amalgam cause the amalgam surface to darken over time and acquire a gray-to-black appearance that can be cosmetically displeasing. The retention of an amalgam restoration depends on adherence to the modern conservative principles of cavity design and by internal retention features, such as the judicious placement of slots and grooves. Ignoring these principles will compromise the longevity of an amalgam restoration and will jeopardize the integrity and strength of the remaining tooth structure.

The placement of an amalgam restoration can require the removal of healthy tooth structure beyond the boundaries of the carious lesion, which impairs the strength of the tooth by decreasing its fracture resistance. All restorative dental materials have advantages and disadvantages related to the physical properties of the material, the ease of placement of the material, and cost. However, the controversy surrounding dental amalgam restorations is detached from these issues; rather, it is focused on concerns of the potential for systemic toxicity due to the inclusion of mercury in its formulation.

Mercury is in its elemental form in dental amalgam restorations and should not be confused with methylmercury, which is found in fish and is absorbed through the digestive tract. Elemental mercury is the least potentially toxic form of mercury, unlike the highly toxic methylmercury. The elemental form is minimally absorbed by the body, while methylmercury is readily absorbed and accumulates within the body tissues [6].

At one time, the elemental mercury in dental amalgam was believed to be bound within the restorative matrix and rendered inert. However, research has revealed that minute amounts of mercury vapor are released from dental amalgam restorations when they wear and when old restorations are removed from teeth [7]. It is this release of minute amounts of mercury vapor over time that some groups have proposed may be the cause of several systemic illnesses [8]. Other groups consider the mercury vapor released from amalgam restorations as inconsequential and unrelated to the development of any systemic illness [8].

The debate about the safety of dental amalgam restorations is a global one. Norway, Denmark, and Sweden have all banned the use of dental amalgam due to their concerns about the impact of mercury on patients and have labeled it an environmental toxin [9]. It is unclear if the actions of these countries will set a precedent for other countries (such as the United States) to enact similar legislation and ban the use of dental amalgam as a restorative material.

Numerous independent studies have been conducted over many years to explore the possible relationship between the mercury in dental amalgam and adverse systemic health. These studies have addressed the concerns that mercury vapor from dental amalgam restorations is implicated in the development of systemic illnesses such as Alzheimer disease, autism, multiple sclerosis, Parkinson disease, renal disease, and lupus [10]. However, the results of scientific studies and reviews to date have not supported these claims.

In 1993, the U.S. Public Health Service (USPHS) conducted a review of the available literature about the safety of mercury used in dental amalgam restorations. The conclusion was that the current scientific evidence demonstrated that exposure to mercury released from dental amalgam restorations does not pose a serious health risk [11]. The USPHS updated this report in 1997. The U.S. Food and Drug Administration (FDA) reviewed about 60 studies published in peer-reviewed scientific literature and cited by citizens groups as a basis to petition the FDA for stringent regulations against the use of dental amalgam [10]. Analysis of the data did not support claims that dental amalgam restorations were the cause of adverse neural, renal, or developmental effects.

The independent nonprofit organization Life Sciences Research Organization (LSRO) uses scientists from a broad spectrum of disciplines to investigate issues related to a variety of areas, including health care and biomedicine. In 2004, the LSRO was commissioned by the National Institutes of Health (NIH), the FDA, and the U.S. Department of Health and Human Services (HHS) to review in excess of 950 scientific articles and research studies about the safety of dental amalgam. These studies and articles were derived from many sources, including the USPHS, the World Health Organization, the European Commission, and Health Canada [12]. The LSRO concluded there was inadequate evidence to establish a definitive link between dental amalgam and kidney disease, Parkinson disease, multiple sclerosis, neurodegenerative diseases (e.g., Alzheimer disease), and autoimmune diseases [12].

In 2009, the FDA published a white paper entitled *FDA Update/Review of Potential Adverse Health Risks Associated with Exposure to Mercury in Dental Amalgam* [10]. That same year, the FDA issued a final rule on encapsulated dental amalgam in which the powder alloy and the elemental mercury were classified as a class II medical device [13]. The FDA also reiterated its position that dental amalgam remains a safe restorative material for patient use. Classification as a class II medical device means that the FDA can impose controls on dental amalgam [13]. In 2020, the FDA updated its guidance related to the use of mercury-containing dental amalgam [14]. In these guidelines, the FDA states that certain groups may be at greater risk for potential harmful health effects of mercury vapor released from these amalgams. As a result, it now recommends that certain high-risk groups avoid getting dental amalgam whenever possible and appropriate, including [14]:

- Pregnant women and their developing fetuses
- Women who are planning to become pregnant
- Nursing women and their newborns and infants
- Children, especially those younger than 6 years of age
- People with pre-existing neurologic disease (e.g., multiple sclerosis, Alzheimer disease, Parkinson disease)
- People with impaired kidney function
- People with known heightened sensitivity (allergy) to mercury or other components of dental amalgam

The guideline panel stressed that while the majority of evidence suggests exposure to mercury vapor from dental amalgam fillings does not lead to harmful health effects for most people, there may be some effects in specific subpopulations.

The Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR), one of the European Commission's leading scientific authorities, issued a report in 2008 concluding there was no scientific evidence that dental amalgam caused any adverse systemic effects and that its use did not pose a risk for the development of systemic disease. In 2014, the SCHENIR reaffirmed its position but also suggested that studies about its use should be continued [15]. As recently as 2019, based on a comprehensive literature review, the FDA indicated that there was no change in their position about the use of dental amalgam. The FDA also indicated that they would continue to evaluate the safety of dental amalgam and that further actions would be taken if warranted [16].

There is no doubt that the debate about the safety of dental amalgam will continue. To date, studies have not found scientific evidence demonstrating the use of dental amalgam is a precipitating factor in the development of systemic disease, but research is ongoing. Dentists should be forthright in discussing the use of dental amalgam as a restorative material with their patients. Alternative restorative materials, such as composites, gold, and porcelain, also have their limitations, advantages, and disadvantages. Patients should be informed that these alternatives are more expensive than dental amalgam and that insurance reimbursement is variable. An open exchange of scientifically based information about the safety of dental amalgam among members of the dental profession, the allied health professions, and the public is in the best interest of all.

COMMUNITY WATER FLUORIDATION

In 1945, the city of Grand Rapids, Michigan, began to add low amounts of fluoride to its community water system [17]. This practice began after dentists in some regions of the United States observed that the teeth of many of their patients appeared stained or mottled yet also protected from tooth decay. As of 2018, approximately 73% of people in the United States are served by fluoridated community water systems [18]. The decision of whether or not to fluoridate municipal water systems is not made by the federal government but by local and state governments.

Fluoridated water works both topically and systemically to reduce the potential for teeth to develop carious lesions. Systemic fluorides are ingested directly through the consumption of fluoridated water, foods, or beverages. Fluoride dietary supplements in pill, tablet, or liquid form are also considered systemic fluorides.

Developing enamel is strengthened and is more resistant to the development of carious lesions when it incorporates systemic fluoride. Systemic fluorides also exert a topical effect, as residual amounts of the ingested fluoride ions remain in the saliva, the dental plaque, and the gingival crevicular fluids. These ions inhibit the demineralization of enamel, promote the remineralization of demineralized enamel, and alter the metabolism of cariogenic bacteria [19]. Systemic fluorides are distinct from dental fluorides, which are strictly topical in nature (e.g., fluoride-containing toothpastes, mouth rinses, fluoride applied in a professional setting).

The initial target concentration of fluoride in community water systems was introduced in the 1940s as a range of 0.7–1.2 mg per liter of water [20]. At the time, it was assumed that people who lived in warmer climates would have a higher fluid intake and would consequently ingest fluoride more than those who lived in cooler regions of the country with assumed lower fluid intake and an overall lower consumption of fluoride. Thus, higher levels of fluoride were added to community water systems in cooler regions of the country, while lower levels of fluoride were added to those in warmer regions of the country.

These recommendations were not re-evaluated until 2010, when the HHS reviewed the most current information about fluoride intake from community water and other sources [20]. Variables considered included fluid intake relative to regional temperature differences, sources of fluoride that were unavailable in the 1940s, trends in patterns of the staining or mottling of enamel due to excess fluoride consumption (dental fluorosis), and the prevalence of and trends in dental caries.

As a result of their analysis, the HHS changed its recommendation to a target concentration of 0.7 mg per liter of water for all fluoridated water supplies across the United States [20; 21]. This level of fluoride was deemed sufficient to reduce the incidence of dental caries while minimizing the risk of dental fluorosis.

Fluoridation of community water supplies has been a major factor in the reduction of dental caries in the United States [20]. It is a cost-effective way to provide all members of a community with fluoride. The benefits of fluoridation apply to both deciduous and permanent teeth not only during their formation but for their duration in the oral cavity. However, there are several groups that oppose the fluoridation of community water sources. They believe that when fluoridated community water sources are combined with fluorides in toothpaste, foods, and beverages, adverse systemic health effects, such as bone fractures, skeletal fluorosis, cancer, neurologic effects, and endocrine problems, may occur [20].

It is nearly impossible to determine the amount of fluoride each person ingests daily. Fluoride from community water systems, processed food and beverages, and fluoridated toothpastes or mouth rinses all contribute to daily intake. The national standard of 0.7 mg/liter in community water reflects these additional sources of fluoride ingestion.

As with any ingested substance, there are levels at which chronic fluoride ingestion can be associated with adverse effects. The maximum allowable concentration of fluoride in community water systems, also known as the maximum contaminant level (MCL), is 4.0 mg/liter [20]. This level is nearly six times the recommended level for community water systems. Ingestion of fluoride at the MCL for a long period of time can lead to the development of dental and skeletal fluorosis [22]. The most common adverse effect associated with the long-term ingestion of fluoride in excess of 2 mg/liter is dental fluorosis [20; 23]. Mild forms of dental fluorosis feature small white spots or streaks in the dental enamel (i.e., mottled enamel). Severe forms of dental fluorosis feature brown or black pits in the enamel. These are not carious lesions but may require composite restorations, as surface pitting will enhance the retention of plaque and increase the potential for the development of caries. In more severe cases, all the enamel may be damaged [24]. A study published by the Centers for Disease Control and Prevention found that approximately 23% of the U.S. population between 6 and 49 years of age had some form of dental fluorosis; 16.0% had very mild fluorosis, 4.8% had mild fluorosis, 2.0% had moderate fluorosis, and less than 1% had severe fluorosis [25]. An additional 16.5% were classified as having questionable or possible dental fluorosis. This same study found that the rates of dental fluorosis among adolescents (12 to 15 years of age) significantly increased between 1986–1987 and 1999–2004. Skeletal fluorosis is characterized by pain and stiffness in major joints and an increased risk for fractures. This condition is extremely rare in the United States [20].

The National Research Council did not find any evidence to substantiate a relationship between fluoride concentrations of 2–4 mg/liter and the occurrence of adverse systemic effects [26]. Furthermore, an analysis of peer-reviewed scientific evidence does not support a correlation between community water fluoridation of 0.7–1.2 mg/liter of water and illnesses such as heart disease, cancer, osteoporosis, renal disorders, Alzheimer disease, and bone fractures [27]. Based on this information, the current fluoride concentration of 0.7 mg/liter of water (and the previously recommended range) was considered a safe way reduce the risk of caries. The use of fluoride in its appropriate concentrations in community water systems should be considered an adjunct in achieving and maintaining optimal oral health.

Another possible area of controversy is the link between fluoride exposure and intelligence quotient (IQ). Several studies have explored this relationship, and patients may have questions about the safety of fluoride, especially for children or while pregnant. A 2019 Canadian study of 512 mother-child pairs compared IQ in offspring of women living in areas with fluoridated water to those living in areas with non-fluoridated water. In this study, a 1-mg higher daily intake of fluoride among pregnant women was associated with a 3.66 lower IQ score in offspring at 3 to 4 years of age [28]. These findings were consistent with findings from other studies in India and Mexico [29; 30]. Studies over the last three years have consistently linked fluoride exposure in pregnancy with adverse neurodevelopmental effects in offspring [31; 32; 33].

To date, the American Dental Association and the American Academy of Pediatrics continue to recommend fluoridated water as a means to protect children from caries and tooth decay, based on decades of research supporting the safety of fluoride and the effectiveness in improving oral health [34; 35]. However, studies are ongoing, and changes in recommendations may be considered based on evolving evidence.

ANTIBIOTIC USE IN DENTISTRY

Sir Alexander Fleming's observation in 1928 that the mold *Penicillium notatum* inhibited a circumferential zone of bacterial growth in a petri dish ultimately led to the development and mass production of penicillin for medical use in 1943—the beginning of the wide use of antibiotic therapy [36]. Today, a myriad of antibiotics are used to treat infections of bacterial origin, some of which are odontogenic.

The indiscriminate use of antibiotics among the allied health professions has led to increasing antibiotic resistance and to an increasing difficulty in the eradication of bacterial infections that were once easily eliminated with empiric therapy. This section will highlight the general principles of conservative and efficacious use of antibiotics.

Bacterial ingress from necrotic pulpal tissues and from periodontal pockets is the usual source of odontogenic infections. Empiric antibiotic therapy should be used when a patient with an odontogenic infection develops an elevated body temperature, lymphadenopathy, trismus (i.e., difficulty opening the mouth), dysphagia (i.e., difficulty swallowing), and/or cellulitis (the extension of the infection into the contiguous tissues, the borders of which are ill-defined and the surface texture indurated). Other indications for empiric antibiotic therapy are periodontal abscesses, pericoronitis, and acute necrotizing ulcerative gingivitis [37]. This is not an all-inclusive list, as the patient's overall medical history, immune status, and age should also be considered when making the decision to prescribe an antibiotic.

Perhaps the greatest issue and challenge to the appropriate use of antibiotics in dentistry arises from patient demands and expectations. Inflammation is the basis for most dental pain, and definitive treatment of dental disease (e.g., restorative dentistry, oral surgery, endodontics, periodontal therapy) is usually the proper treatment—not empiric antibiotic therapy. Antibiotics are not a typical part of the treatment of a carious lesion or pulpitis without swelling. Restoring, extracting, or performing endodontic therapy is the treatment of choice in these cases, with nonsteroidal anti-inflammatory drugs (NSAIDs) used for analgesic purposes, if necessary. Dental clinicians should not prescribe antibiotics as a substitute for definitive treatment. Patients may have received antibiotics from dental clinicians for these situations in the past and may expect or demand them when dental problems arise. It is important that antibiotics not be prescribed to assuage a patient. Instead, clinicians should take time to educate patients about the underlying causes of dental conditions and to explain why antibiotics will be of no use.

Gingivitis and periodontal disease are chronic inflammatory conditions treated by definitive therapy, such as increasing the frequency of recall appointments, root planing and curettage, patient education and instructions tailored to the patient's individual periodontal needs, and periodontal surgery. Traditional approaches to periodontal therapy, whether surgical or nonsurgical (i.e., scaling and root planing), have been shown to significantly reduce the bacterial population in treated sites without the use of supplemental antimicrobial agents. In an attempt to enhance the effect due to mechanical therapy, antibiotics such as penicillin, members of the tetracycline family, and metronidazole have all been given systemically. The members of the tetracycline family (e.g., tetracycline, doxycycline, minocycline) are good candidates for use as these drugs concentrate in the gingival crevicular fluid by a factor of four to

eight, depending on the agent employed. Thus, every systemic application of a member of the tetracycline family will produce a topical application of the agent within the periodontal pocket in a concentration several times greater than that produced in the serum. Clinical studies have failed, however, to demonstrate any significant benefit of the routine use of systemic antibiotics alone or in combination with mechanical therapy over mechanical therapy alone for patients diagnosed with adult periodontitis [38; 39; 40; 41; 42]. Additionally, the use of antibiotics to treat periodontitis is controversial due to the wider context of the overprescribing of antibiotics and the rise of antimicrobial resistance [43]. The use of supplemental systemic antibiotics seems indicated, however, in the treatment of patients diagnosed with aggressive manifestations of periodontal disease, such as those associated with juvenile and refractory periodontitis [44; 45; 46; 47].

The use of antibiotics in conjunction with oral surgical procedures has also been a subject of controversy. Some patients may believe that the only way to prevent alveolar osteitis ("dry sockets") or to heal properly is to take antibiotics after oral surgery. Acute alveolar osteitis is an acute inflammatory condition caused by the loss of the protective blood clot that covers the alveolar bone of the extraction site. The intense pain associated with this condition arises from an inflammatory response of the exposed bone and is not the result of an infection. As such, the treatment of choice is gentle irrigation and debridement of the socket and the placement of an obtundent dressing; antibiotics are not appropriate [48].

The prophylactic use of antibiotics prior to the extractions of third molars ("wisdom teeth") also remains controversial. A meta-analysis of 24 studies related to this issue found that the data do not support the use of adjunctive antibiotics in the prevention of infection after third-molar surgery [49].

Antibiotics will always have their place as an adjunct to dental treatment. However, clinicians should prescribe these medications judiciously, in the best interest of the oral and general health of their patients. They should also educate patients that the indiscriminate use of antibiotics will continue to cause the emergence of bacterial strains resistant to all known antibiotics, potentially precipitating a public health crisis.

TREATING TWO PATIENTS AT ONCE: THE PREGNANT DENTAL PATIENT

Perhaps one of the most controversial issues in dentistry is treating the pregnant patient. The concern for harming the developing fetus and/or the mother has led many in the general public to believe that only dental problems of a dire or emergent nature should be treated during pregnancy. One of the ethical foundations of the dental profession is to "do no harm," and this applies to the pregnant patient and the developing fetus. However, treatment modifications during pregnancy can promote optimal oral health in a safe and efficacious manner. Before treating a pregnant patient, dentists should consult with the patient's obstetrician to determine if routine dental treatment is appropriate or if there are comorbidities that mandate the deferral of elective dental treatment until the completion of the pregnancy.

GENERAL CONSIDERATIONS

Nausea

Nausea and vomiting of pregnancy ("morning sickness") occurs in about 75% of pregnancies [50]. It usually begins between the fourth and eighth week of pregnancy and though it usually resolves before 20 weeks, it may continue through the duration of the pregnancy for some women [51]. The long-term regurgitation of acidic gastric contents can lead to the erosion and loss of enamel. Patients who have this problem should be instructed to first rinse their mouths with a neutralizing solution of 1 teaspoon of baking soda to 8 ounces of water to remove any acidic residue from the teeth. Patients who do not rinse before brushing their teeth with toothpaste will essentially be burnishing the acidic residue onto the enamel surface, enhancing the erosive effects. Continued erosion of enamel can lead to exposure of dentin, with subsequent development of tooth sensitivity. Patients may be prone to avoid brushing and flossing sensitive areas, with a consequent accumulation of plaque that can increase the risk of caries and periodontal problems. Desensitizing toothpastes or fluoride varnishes can relieve this sensitivity for minor areas of exposed dentine, but larger areas of erosion will require restoration.

Some patients will develop a heightened gag reflex during pregnancy and may be especially sensitive to the accumulation of intraoral fluids (e.g., from high-speed hand pieces and ultrasonic scaling units). Staff members should use high-speed suction to minimize the accumulation of fluids. Impressions may be deferred until after the pregnancy, as the flow of these materials toward the soft palate can stimulate the gag reflex.

Medication Use

Prescribing and administering medications for pregnant patients should be done with concern for both maternal and fetal safety. The FDA has established pregnancy drug risk categories according to drugs' effects on reproduction and pregnancy [52]. The safest medications that have been tested in pregnant women are category A drugs. However, due to possible risks to maternal and fetal health, drug trials rarely include pregnant women, and there are only a few medications that fall into this category. Category B medications have been found to have no fetal risk in animal studies, or more rarely, have been proven safe in women despite evidence of increased risks in animal studies. Medications for which increased risk of harm to mother or fetus cannot be ruled out are referred to as category C, while medications with evidence of negative effects are category D. Any medications that are contraindicated during pregnancy are considered category X.

The drug risk categories have been in effect since 1979 and were substantially updated in 2006 under the FDA's Physician Labeling Rule (PLR). Effective June 2015, the Pregnancy and Lactation Labeling Rule (PLLR) replaced the product letter categories with three detailed labeling subsections [52; 53; 54; 55; 56]:

- The pregnancy subsection must provide information relevant to use of the drug in pregnant women (e.g., dosing, potential risk to fetus); it must also provide information about whether a registry exists that collects/maintains date on the drug's effect on pregnant women.
- The lactation subsection must provide information about use of the drug during breastfeeding (e.g., amount of drug in breast milk, potential effect/s on the child).
- The females/males of reproductive potential subsection must include information about pregnancy testing, contraception, and infertility as it relates to the drug.

The PLLR is intended to provide a more consistent way to include relevant information about the risks and benefits for the mother, the fetus, and the breastfeeding child. It will apply to all "newly approved" drug and biologic product applications. The labeling of previously approved products that contain the product letter categories will be phased in gradually [56].

When prescribing medications to pregnant women before, during, and after dental treatment, these guidelines should be taken into consideration. In addition, the patient's unique medical history, medication use, and current condition should be considered. If there is any doubt as to which medication, dose, or frequency of administration to use, consultation with the patient's obstetrician is advisable.

FIRST TRIMESTER

Rapid cell division and organ development occurs between the second and eighth week of pregnancy. During this time, the fetus is most susceptible to the effects of stress and teratogens. Approximately 50% to 75% of all spontaneous abortions (miscarriages) occur during this period [57]. Dental treatment should be limited to basic periodontal prophylaxis, if needed, and emergency treatment of traumatic injuries, acute odontogenic infections, and/or pain of dental origin. Radiographs should only be done if required for diagnosis of an emergent dental problem. This is also an excellent time to educate patients about the importance of oral hygiene and the challenges that they may face during pregnancy.

A number of changes in the oral cavity have been associated with pregnancy, including caries, perimylolysis, tooth mobility, xerostomia, pregnancy granuloma, and ptyalism/sialorrhea [58]. Perhaps most commonly, the hormonal changes that occur during pregnancy have been linked with gingivitis. Approximately 60% to 75% of women will develop pregnancy gingivitis [59]. This can range from mild to severe gingival inflammation that can develop despite meticulous oral hygiene and the absence of chronic irritants such as plaque and calculus. Shifts in hormone levels may cause changes in the established microbiota, with overgrowth of certain bacteria species, increases in the ratio of bacterial anaerobes to aerobes, and changes in the proportions of *Prevotella intermedia*, *Prevotella melaninogenicus*, and *Porphyromonas gingivalis* [58; 60; 61]. Pre-existing subclinical gingivitis may become exacerbated during pregnancy to the point that clinical signs become apparent, including swelling, redness, bleeding, and tenderness [62]. These signs may begin to be noticeable in the second trimester and peak around the eighth month. Anterior teeth may be more apparently involved than the posterior. Mouth breathing is a potential exacerbating factor. A woman who has poor oral hygiene runs the risk of even greater gingival problems, although gingivitis can develop in women with no changes in their plaque-management behavior. Postpartum studies have shown that after delivery, the mother's level of gingivitis decreases as the constituency of the microbiota changes back to approximate its prepregnancy status. With the inflammation comes an increase in tooth mobility. Xerostomia is also reported in a high percentage of patients.

In a study published in 2010, researchers evaluated the way in which changing hormone levels influenced the gingival tissues in 48 pregnant and 28 nonpregnant women. In analyses of the subjects clinically and microbiologically, the researchers found that the proportions of the subgingival pathogens did not differ during pregnancy but did differ significantly after delivery. Patients who were *P. gingivalis*-positive presented with increased gingival inflammation that was not related to plaque [61].

Receptors for female sex hormones are located on human gingiva. The presence of progesterone, for example, may lead to greater gingival exudate. The inflammatory response also appears to be triggered as levels of estrogen and progesterone rise [60; 63; 64; 65].

In addition to generalized gingival changes, a solitary, tumor-like growth, frequently referred to as a "pregnancy tumor" or "pregnancy granuloma," may appear. This lesion is often found associated with anterior interdental areas and has a histologic appearance similar to a pyogenic granuloma. Often, the lesion will regress after delivery, so decisions about surgical removal are best delayed until some time postpartum. Also, removal of the lesion during pregnancy may result in a recurrence [58].

SECOND TRIMESTER

By the second trimester, fetal organ formation is complete, so some elective dental procedures may be performed at this time. The increasing fetal size and weight during the second trimester can place pressure on the inferior vena cava and compromise the return of blood to the heart when the patient is in a supine position. This can lead to maternal hypotension, decreased cardiac output, and loss of consciousness. Turning the patient on her left side will relieve the fetal weight against the inferior vena cava and allow the resumption of the appropriate blood flow. Radiographs should only be taken if absolutely necessary.

THIRD TRIMESTER

While dental treatment poses a minimal risk to the fetus in the third trimester, increasing fetal size and weight can make the mother feel uncomfortable in any position for dental treatment. Appointment duration should be kept to a minimum and should address basic oral hygiene procedures and emergent dental issues. Contact in this period presents an opportunity to educate the mother about the oral hygiene needs of young children and the need for the child to see a dentist or pedodontist early in life so a preventive regimen can be instituted to promote optimal oral health. Pregnant patients should be provided with information about the importance of proper brushing techniques of deciduous teeth and about whether or not fluoride supplements are recommended for the child.

PERIODONTAL DISEASE AND SYSTEMIC DISEASE

Since the late 20th century, there has been a resurgence of interest about the association between oral health, specifically periodontal disease, and various systemic diseases. However, this is not a new theory. In 1891, a publication entitled *The Human Mouth as a Focus of Infection* theorized that poor oral health could be detrimental to systemic health, postulating that the extraction of all teeth could be a means of safeguarding overall health [66]. Today, the areas of greatest interest are the potential relationships between periodontal disease and cardiovascular disease and diabetes. Because this is an area of continuing research, there is some disagreement.

PERIODONTAL DISEASE AND CARDIOVASCULAR DISEASE

In 1989, studies by Mattila and colleagues determined that there was a higher rate of caries, periodontitis, periapical lesions, and pericoronitis among patients with recent myocardial infarctions [67]. Chang and colleagues noted that poor oral hygiene was associated with increased risk of stroke [68]. Studies continue to explore the relationship between periodontal disease and cardiovascular health, suggesting factors that may constitute the basis of this relationship (e.g., systemic inflammation, poor oral hygiene) [69; 70; 71].

Researchers have hypothesized both direct and indirect pathways by which the pathogenic bacteria associated with periodontal disease may cause damage to the vascular endothelium, with the subsequent development of atherosclerosis. Atherosclerosis is the primary cause of myocardial infarction, stroke, and peripheral vascular disease. While research in this area continues, and a direct cause-and-effect relationship between periodontal disease and cardiovascular disease has not yet been established, a 2020 consensus report from the European Federation of Periodontology and the American Academy of Periodontology concludes that there is now a significant body of evidence supporting independent associations between severe periodontitis and chronic noncommunicable disease, including cardiovascular disease [70; 71; 72; 73].

Direct Pathways

Periodontal disease is inflammatory in nature, and *Actinobacillus actinomycetemcomitans*, *P. gingivalis*, *Tannerella forsythia*, and *Treponema denticola* are among the most common pathogenic bacteria [74]. Inflammation in the periodontal pockets creates a highly vascular environment, which allows periodontal pathogens direct access to the systemic circulation.

Atheromas are small, patchy thickenings that develop on the inner lining of arteries and subsequently protrude into the arterial lumen. These plaques can impede and ultimately stop the flow of blood. Platelets can collect on these atheromatous lesions and further impede the flow of blood by forming a thrombus (a stationary blood clot). If the thrombus detaches and travels through the bloodstream, it can lodge anywhere in the vascular network. Studies show that 40% of atheromatous lesions contain antigens produced in response to periodontal pathogens [75]. *P. gingivalis* can stimulate platelet aggregation, a component of atheromas and thrombi [76]. It has also been suggested that these periodontal pathogens can infiltrate the vascular endothelium and directly cause dysfunction, inflammation, and atherosclerosis [77]. One study suggests that having a better understanding of the periodontal pathogens coupled with proper examination of the oral cavity may aid in the early diagnosis and treatment of systemic diseases and conditions [78].

Indirect Pathways

Periodontal disease is a chronic inflammatory disease, and in patients with this disease, chemical mediators of inflammation are released systemically. These mediators have been theorized as an indirect pathway by which periodontal disease may influence the development of cardiovascular disease. Circulating inflammatory mediators produced and released in association with periodontal disease include cytokines, such as tumor necrosis factor-alpha (TNF-a) and interleukin-1 (IL-1) [79]. These cytokines can increase the low-density lipoprotein (LDL) level and decrease the high-density lipoprotein (HDL) level while also elevating the triglyceride level, all of which contribute to the development of cardiovascular disease [80].

The soluble blood protein fibrinogen, which is converted to fibrin during the coagulation process, is also elevated in patients with periodontal disease [81]. The release of fibrinogen can lead to localized clot (thrombus) formation, with the potential for a stroke or myocardial infarction. C-reactive protein (CRP), an acute-phase protein, is elevated in patients with periodontal disease. CRP is a strong indicator of cardiovascular risk. Elevated CRP levels coupled with the ratio of total cholesterol to HDL are used to identify persons at high risk for the development of a myocardial infarction [82]. When definitive periodontal treatment is performed, with a subsequent decrease in gingival inflammation, the serum levels of these inflammatory mediators also decrease [83].

PERIODONTAL DISEASE AND DIABETES

Periodontal disease is unequivocally a major complication of diabetes, and poorly controlled diabetes and periodontal disease are closely linked [84; 85; 86]. Susceptibility to periodontitis is increased by approximately threefold in people with diabetes. [86]. Individuals with diabetes frequently complain of oral changes, including diminished salivary flow and/or xerostomia, altered saliva composition, inflammation, loss of sensation, changes in taste perception, numbness, burning mouth syndrome, and lesions of the oral mucosa and tongue [87]. Although these are not all symptoms of periodontitis, the oral changes can predispose an individual for the development of gingivitis and periodontitis. There is evidence of a relationship between periodontal disease severity and diabetes (particularly poorly controlled diabetes), but the exact mechanism by which one might influence the other has not been definitively determined [86]. Emerging evidence suggests the existence of a two-way relationship between diabetes and periodontitis, with diabetes increasing the risk for periodontitis and periodontal inflammation negatively affecting glycemic control [79; 86]. Many believe that the host of oral changes evidenced in patients with diabetes may act synergistically to predispose these individuals for periodontitis [88].

Both diabetes and periodontal disease share a common pathogenesis that involves enhanced inflammatory response at the local and systemic levels. This inflammatory response is mainly caused by the chronic effects of hyperglycemia and specifically the formation of biologically active glycated proteins and lipids [86; 89]. Patients with diabetes, especially uncontrolled diabetes, are at an increased risk for impaired healing, and the periodontal pocket can experience persistent inflammation and bacterial infection in patients with periodontal disease, which can be made worse by this impaired healing [88; 90]. Loss of teeth because of aggressive periodontitis may also occur [90].

The pathophysiology of endothelial dysfunction and inflammation is the root cause of much of the microvascular and macrovascular deterioration associated with diabetes [86; 90]. The normal metabolic response to a glucose load is an increase in free fatty acids and insulin. These changes result in a transient decrease in endothelium-derived nitric oxide production and in endothelium-mediated vasoconstriction. In the presence of normal glucose tolerance, endothelial nitric oxide production and vasodilation return to normal within two hours. However, in patients with diabetes, endothelium-mediated vasoconstriction continues for hours [91]. This impaired blood flow can affect collagen synthesis, maturation, and homeostatic turnover, all of which can result in impaired healing and the development of periodontal disease [88].

In addition to vasoconstriction, endothelial dysfunction is correlated with aggregation of platelets, a proinflammatory state characterized by the accumulation of leukocytes and coagulation products on the endothelium. Fibrinolysis is decreased, and thrombosis is increased. As the secretion of prostacyclin and nitric oxide induce vasoconstriction, plasma cytokine and prothrombin factors levels increase. This makes the plasma markedly procoagulant and antifibrinolytic, promoting atherosclerosis [90]. The Insulin Resistance Atherosclerosis Study also demonstrated that chronic hyperglycemia was positively associated with increased intimal-medial wall thickness [89]. These changes in both the microvascular and macrovascular systems lead to reduced vascular reactivity and increased production of glycation end products [91]. The accumulation of advanced glycation end products in the gingival tissues is generally responsible for the oral complications of diabetes. In fact, individuals with poorly controlled diabetes have a two- to three-fold increase in the prevalence of oral lesions and periodontal disease.

TOBACCO USE: IS THERE A SAFE ALTERNATIVE TO SMOKING?

Tobacco was the first export of the New World and was marketed in Europe as a remedy for stress, ulcers, headaches, asthma, and even rheumatism. In contrast to strict regulations found elsewhere in the world, tobacco was brought to the United States as a cash crop. Though medical concerns were suggested, the first tobacco prohibition movements in the United States were primarily driven by religious and moral motivations [92].

Associations between smoking and cancer were not published in the United States until the 1950s and 1960s. The 1964 publication *Smoking and Health: Report of the Advisory Committee to the Surgeon General* led to immediate political notice of the tobacco issue and the advent of programs and policies to reduce smoking [93]. Anti-tobacco policies have included taxation on tobacco products, increased insurance premiums, warning labels, public health campaigns, and restrictions on tobacco sales to minors, smoking in public areas, and tobacco marketing. Prior to 1964, there were few if any laws regulating involuntary secondhand smoke exposure. Studies revealing the detrimental effects of secondhand smoke to nonsmokers led to new anti-smoking legislation. Today, nearly all 50 states have laws restricting smoking in places such as schools, public transportation, government buildings, elevators, and restaurants. In accordance with federal law, smoking is prohibited on buses, trains, and domestic airline flights. Such laws have decreased cigarette consumption by making smoking less socially acceptable and more inconvenient [92].

Despite the seemingly well-known and highly publicized health consequences of smoking, in 2020, 12.5% (30.8 million) of adults in the U.S. population 18 years of age or older were current cigarette smokers [94]. Approximately 480,000 Americans die each year as a result of active and/or passive smoking-related health consequences [94; 95].

With the adverse effects of smoking known and increasing social pressures to limit or ban smoking in many public places, smokers have begun to seek alternatives to cigarette smoking that can satisfy the physiologic and psychologic cravings for nicotine but without the deleterious health effects and stigma of cigarettes. Although the prevalence of cigarette smoking continues to decline, there is some evidence that this decline is a reflection of a migration to non-cigarette products, especially e-cigarettes [96; 97]. In addition to a rise in the use of smokeless tobacco, people across the United States (especially youth) are using e-cigarettes, cigars, cigarillos (small cigars), hookahs, kreteks, pipes, and bidis (or beedis) [98; 99]. Unfortunately, each of these products is just as dangerous (if not more so) as use of cigarettes, despite public perception.

SMOKELESS TOBACCO PRODUCTS

Smokeless tobacco by definition is non-pyrolytic (not burned or combustible), and there may be an erroneous belief that because it is not smoked, this tobacco is safe. However, this is demonstrably untrue.

Smokeless tobacco is also known as dip, chew, snuff, or chewing tobacco and generally exists in two forms. Snuff is finely ground tobacco that can be moist, dry, or placed in pouches. Moist snuff comprises about 81% of the smokeless tobacco market. With this product, a pinch (or "dip") is placed between the gingival tissues and the cheek or lip [100]. Expectoration is required. More rarely, finely ground snuff may be sniffed or inhaled through the nose. Chewing tobacco exists in the form of loose leaves, as tobacco leaves condensed together as a plug, or as tobacco leaves that are twisted together like a rope. The loose leaf form of chewing tobacco accounts for about 18% of the smokeless tobacco market [101]. In 2020, approximately 2.3% of adults, or about 5.7 million individuals 18 years of age and older, in the United States currently used a smokeless tobacco product [102].

While smokeless tobacco eliminates the risks of secondhand and thirdhand smoke to bystanders, it remains highly addictive and harmful to users. Nicotine from smokeless tobacco is absorbed more slowly through the oral mucosa, compared with the rapid systemic absorption and distribution from cigarette smoke [103]. However, the effects of nicotine absorbed through the oral mucosa are more protracted. Although the route of absorption varies, the potential for addiction remains the same.

Smokeless tobacco contains 28 carcinogens and is a known cause of oral and pancreatic cancers [104]. The extended contact time of smokeless tobacco products on the oral mucosa allows these carcinogens ample time to begin changing the cells of the mouth and esophagus. Smokeless tobacco users may develop squamous cell carcinoma, smokeless tobacco keratosis (also known as snuff pouch lesion or snuff dipper's lesion), and/or verrucous carcinoma. Potential adverse effects to the periodontium include gingival recession, periodontal disease, and necrotizing ulcerative gingivitis [51].

ELECTRONIC CIGARETTES (E-CIGARETTES)

The rise of e-cigarettes in the past decade has introduced new variables in the prevention and treatment of nicotine addiction. Originally marketed as a smoking cessation tool, e-cigarettes are electronic products that typically deliver nicotine in the form of an aerosol [105]. Most e-cigarettes consist of a cartridge (which holds a liquid solution containing varying amounts of nicotine, flavorings, and other chemicals), a heating device (vaporizer), and a power source (usually a battery) [105; 106]. In many e-cigarettes, puffing activates the battery-powered heating device, which vaporizes the liquid in the cartridge. The resulting aerosol or vapor is then inhaled (called "vaping") [106]. It is unclear if this delivery method decreases the risks seen with conventional tobacco smoking; however, it does introduce the risks of toxicity associated with consumption of the potent e-liquid.

In 2020, 3.7% of adults, 3.3% of middle school students, and 14.1% of high school students were current e-cigarette users [105]. In 2019, among current adult e-cigarette users overall, 36.9% also currently smoked cigarettes ("dual users"), 39.5% formerly smoked cigarettes, and 23.6% had never smoked cigarettes. Among current adult e-cigarette users, the percentage of those who have never smoked cigarettes is highest among those 18 to 24 years of age and is lower in older age groups [105].

From 2017 to 2018, adolescent use of e-cigarettes increased from 11.7% to 20.8%, making it the number one form of tobacco used among youth. The rate increased again in 2019 to 27.5%, but decreased in 2020 to 19.6% [107]. In 2018, the FDA issued more than 1,300 warnings and fines to retailers who illegally sold e-cigarette products to minors [108]. In 2020, the FDA banned mint- and fruit-flavored e-cigarette cartridges in an effort to halt uptake among children [109].

Because e-cigarettes have been used in the United States for a relatively short period, the long-term effects on oral and systemic health are not totally clear. Elimination of exposure to tar is a benefit for e-cigarette use. However, the chemicals that are inhaled while using e-cigarettes are associated with known health hazards. Nicotine remains a highly addictive compound regardless of its source. People who use e-cigarettes can experience nicotine withdrawal symptoms if they discontinue the use of the device. One study demonstrated that formaldehyde, a known carcinogen, can be produced during the vaping process, with the potential for the user and bystanders to inhale vapor that contains the carcinogen [110]. In general, e-cigarettes cannot be considered a safe alternative to cigarette smoking.

HOOKAHS

A hookah is a type of waterpipe comprised of a head or bowl, plate, body, jar, hose, and mouthpiece. The body of the hookah fits down into the jar, which is partially filled with water, although any liquid (e.g., alcohol, juice) can be used. Tobacco is placed in the bowl at the head of the body and covered with a filter, such as perforated tin foil, and then burning embers or charcoal is placed above it (and sometimes covered by a cap). The hot air from the charcoal roasts the tobacco and the ensuing smoke is passed down into the liquid in the jar, where it is partially filtered, diluted, and cooled. The smoke then bubbles up and passes through the hose and mouthpiece for inhalation. Repeated inhalation is required to keep the tobacco burning. The plate stores dead coals/embers. The types of tobacco used for hookah are ajami or tumbak, which is a pure, dark tobacco paste; "honeyed" tobamel or maassel, containing 70% honey or molasses and featuring flavors (e.g., apple, mango, banana); or jurak, which may be sweetened or contain fruits or oils. It is commonplace to use 10–20 g at a time, and these tobaccos may be mixed with other drugs [111]. Smoking sessions last up to an hour or longer, and it has been reported that the nicotine content of the tobacco used for hookah is higher than that in cigarettes [112]. Thus, the smoker is exposed to a higher volume of smoke for longer periods (not to mention those in the vicinity). A report from the World Health Organization states that a hookah user may inhale as much smoke in one session as a cigarette smoker would after consuming at least 100 cigarettes [113].

Contrary to popular belief, waterpipe smoking is not safer or less addictive than cigarette smoking [114]. The FDA began regulating the manufacture, import, packaging, labeling, advertising, promotion, sale, and distribution of tobacco mixtures used for hookah in 2016 [115]. Hookah smoke contains higher concentrations of carbon monoxide, nicotine, tar, heavy metals, and carcinogens, likely because of its method of use (i.e., tobacco mixtures heated by quick-burning charcoal or wood embers and inhalation through use of a plastic hose for an hour or longer) [116; 117]. It is also common to share a hookah, so users are also at risk of exposure to infections (e.g., herpes due to sharing of the mouthpiece) [118]. Hookah pipe smoking may be a gateway to cigarette smoking and other drug use. Although policies are in place to ban smoking in many public places, many times, hookah use is exempt because it is done in places which identify themselves as "tobacco bars," waterpipe smoking areas are set up outside, or the smoking is done in places where tobacco is sold.

HALTING MEDICATIONS PRIOR TO INVASIVE DENTAL PROCEDURES

The ability to achieve hemostasis after invasive dental procedures can be challenged by various coagulopathies and by the patient's use of anticoagulant and antiplatelet medications. A literature review of thromboembolic risks upon discontinuance of anticoagulant therapy prior to invasive dental procedures revealed serious embolic complications, including four deaths, among 493 patients [119]. Whether or not it is necessary to temporarily hold these medications before invasive dental treatment has been the source of confusion. In essence, this decision is based on an individualized assessment of risk.

ANTICOAGULANTS

Prior to initiating invasive dental treatment for any patient who is using an anticoagulant medication, the first step is to identify the underlying condition. A variety of conditions require anticoagulation, including venous thrombosis, pulmonary embolism, and prevention of recurrent myocardial infarction or stroke. In many cases, patients with these conditions have additional comorbidities, such as hypertension and diabetes. A patient's entire medical history should be considered before dental treatment is initiated, and consultation with the patient's physician should be sought if there is concern about proceeding with treatment. It is important to note that only a physician should make the decision to withhold anticoagulant therapy.

Anticoagulants reduce vitamin K-dependent synthesis of coagulation factors II, VII, IX, and X in the liver. Warfarin has been the most commonly prescribed anticoagulant in the United States, although other agents (e.g., apixaban, dabigatran) are growing in popularity [120]. The therapeutic effects of warfarin can be monitored using the international normalized ratio (INR), which is a ratio of the patient's prothrombin time against a laboratory control. The INR of a person whose blood clots normally and is not taking an anticoagulant would be 1. Values greater than 1 indicate a lengthened time to coagulation. The therapeutic range of INR depends on the medical problem for which it is prescribed. Laboratory values obtained within the last 48 hours are the most accurate [121]. An INR of 3 or less generally indicates it is safe to perform minor oral surgery without a temporary discontinuance of the warfarin [122]. However, INR values should only serve as a guide, because there is no universally accepted value at which a patient on warfarin is guaranteed to achieve hemostasis after an invasive dental procedure. Other factors that can adversely affect hemostasis include hepatic disease, alcoholism, thrombocytopenia (low platelets), renal failure, and the nature and the extent of the planned surgical procedure.

As noted, while warfarin is the most common anticoagulant, other agents are commonly used, including dabigatran (a potent inhibitor of free thrombin) and the selective factor Xa inhibitors rivaroxaban and apixaban. INR is not useful for these medications, and evidence-based guidelines are lacking for the dental management of patients using these agents, so consultation with the patient's physician is advisable prior to the initiation of any invasive dental procedure [123].

Patients who remain on anticoagulant therapy during invasive dental treatment should not use NSAIDs (e.g., naproxen, ibuprofen), as these medications can inhibit platelet aggregation. When this action is coupled with the actions of an anticoagulant medication, the potential for postprocedural bleeding increases.

ANTIPLATELET MEDICATIONS

Antiplatelet medications act by inhibiting platelet aggregation; aspirin and clopidogrel are popular examples. The most common indications for antiplatelet medications are the prevention of arterial and venous thrombosis in patients with ischemic heart disease, the prevention of platelet aggregation in patients who have cardiac stents or heart valve implants, and prevention of recurrent stroke in patients who have experienced one in the past [124]. These medications have no impact on coagulation factors. In most cases, patients who use aspirin for antiplatelet therapy do not experience more postsurgical bleeding than patients who do not take aspirin [125]. Similarly, studies have demonstrated that the use of clopidogrel is not equated with excessive bleeding during or after minor oral surgery procedures [126].

As with patients who use anticoagulants, the underlying condition necessitating antiplatelet therapy should be documented. Consultation with the patient's physician is advisable to understand the current status and prognosis of the patient's cardiovascular or cerebrovascular problem and to determine if any other co-existing systemic illnesses complicate the ability to provide dental care for the patient.

Platelets are not actually cells but are fragments of a larger progenitor cell called a megakaryocyte. These fragments lack a nucleus but contain a host of chemicals that are essential to the clotting process. Because antiplatelet medications interrupt varied phases of the process by which platelets adhere to a damaged blood vessel and to each other, there is a concern about the ability to achieve hemostasis after an invasive dental procedure. The risk of bleeding during or after an invasive dental procedure if a patient remains on antiplatelet medications should be weighed against the risk of an adverse cardiovascular or cerebrovascular event if these medications are held temporarily.

The American Heart Association, the American College of Cardiology, the Society for Cardiovascular Angiography and Interventions, the American College of Surgeons, and the American Dental Association have published a consensus opinion about drug-eluting stents and antiplatelet therapy [127]. It stresses the importance of maintaining antiplatelet medications to minimize the risk of stent thrombosis and recommends that the patient's cardiologist should be consulted to discuss the patient's antiplatelet regimen and the optimal management of the patient for invasive dental procedures [128]. A decision to temporarily discontinue aspirin, clopidogrel, or any other antiplatelet medication is made by the cardiologist.

While studies indicate that patients who continue antiplatelet therapy during invasive dental procedures are not likely to experience excessive bleeding, it remains a possibility. The nature and extent of the procedure, hepatic disease, alcohol abuse, and/or any coagulopathy should be considered. If the medications are continued and intra-operative or postsurgical bleeding is a concern, the procedure may need to be completed in a hospital or an outpatient treatment center.

The use of NSAIDs for analgesic relief of pain adjunctive to dental treatment is common, and these medications can inhibit platelet aggregation and prolong the bleeding times in some patients [129]. If antiplatelet therapy and NSAIDs are taken concurrently, the risk of bleeding will increase. Therefore, an alternative analgesic, such as acetaminophen, should be considered.

ACCESS TO DENTAL CARE: A MULTIFACTORIAL PROBLEM

Although the link between oral health and general health is well-established, the divide between the two fields is great. Many healthcare professionals have not received formal training in oral health, but collaborative care with dental professionals is an essential aspect of improving dental care. A discussion of all the underlying causes of poor access to dental care for people in the United States is beyond the scope of this course. However, a brief exploration of major contributing factors will be provided.

RURAL AREAS AND THE DENTAL WORKFORCE

As compared to decades ago, dental health has improved across the United States, which is primarily attributed to fluoridation of water and toothpaste and greater awareness of optimal oral hygiene. However, rural areas have a variety of factors that continue to contribute to poorer oral health in this population [130]:

- Geographic isolation
- Lack of adequate transportation
- Higher rate of poverty compared to metro areas
- Large elderly population (with limited insurance coverage of oral health services)
- Acute provider shortages
- State-by-state variability in scope of practice
- Difficulty finding providers willing to treat Medicaid patients
- Lack of fluoridated community water
- Poor oral health education

Dental care is a problem largely because of a lack of practicing dentists and insufficient dental insurance. There are fewer rural dentists (per capita) available to address oral disease than in urban settings, and the majority of dental health professional shortage areas lacking access to dental services are rural [131]. An estimated 8,600 additional dentists will be required nationally to meet care needs by 2025 [131]. To address this problem, areas with fewer dental professionals may qualify for a federal dental health professional shortage area designation. Having this designation can qualify the area to receive financial aid or recruitment aid from the government [132].

It is estimated that 70 million people in the United States live in areas in which access to dental care is limited [133]. Rural areas and areas in which there is a chronic high rate of unemployment can have difficulty in attracting dentists to these areas due to the high cost of maintaining a practice.

A shortage of dental practitioners in rural and micropolitan areas has resulted in emergency departments becoming the alternative for evaluation and treatment of dental conditions [134]. To address this problem, states have passed laws intended to expand the dental care workforce. In Minnesota, for example, additional license types (dental therapists and advanced dental therapists) have been added to help meet the need for dental professionals qualified to provide preventive and restorative dental care, in some cases with less direct supervision [135; 136]. In this case, at least half of a dental therapist's patients must be considered underserved—that is, on public assistance, uninsured, or living in an area with a shortage of dentists [137]. Several states permit new dental profession types to provide dental care under varying levels of supervision by dentists, allowing these providers to meet dental care needs in nontraditional, tribal, school-based, and community settings [135]. In Alaska, where the majority of land is classified as rural, some clinics have sent out dental health aide therapists to distant rural sites to deliver routine restorative care. As a result of this initiative, many recipients were able to have regular access to dental care for the first time [137].

Aside from government actions to increase workforce numbers and to expand the scope of practice, dental care workforce capacity can be built using teledentistry. In California, dental hygienists use teledentistry to improve dental care access for the young and disabled. Dental hygienists go to community settings such as schools, Head Start public programs, and nursing homes, where patients are

screened and data are transmitted digitally back to the dentist, who creates a treatment plan for the hygienist to implement [137]. These solutions have potential to mitigate dental disease in rural underserved communities.

CHILDREN

Dental caries are the most common chronic childhood disease and are five times more common than asthma [138]. However, studies indicate that many children have not received even the most basic of dental care. A study of third-grade children in New York revealed that 54% had dental caries, with 33% lacking treatment [139]. In 2014, more than 18 million low-income children received no dental care [140]. There are many underlying reasons for this disparity of dental care among children, including lack of transportation, lack of insurance, poor understanding of the importance of deciduous teeth, and difficulty finding a pedodontist.

As compared with the general population and other racial/ethnic groups, American Indian/Alaska Native (AI/AN) children are disproportionately affected by dental disease, and oral health for school-aged AI/AN children 6 to 9 years of age did not change significantly between 2010 and 2018–2019 [141]. AI/AN elementary school children have a higher prevalence of both dental caries experience and untreated dental caries than the general U.S. population. Compared with the general U.S. population, AI/AN children are twice as likely to have untreated dental caries in their primary teeth and five times more likely to have untreated dental caries in their permanent teeth [141]. The compromised dental health of this population is believed to be rooted in differences in host, bacterial, behavioral, sociodemographic, and environmental risk factors, as well as historic economic adversity and poor social conditions [141].

The Indian Health Services Early Childhood Caries Initiative provides AI/AN children with oral assessments and interventions to mitigate early childhood dental disease, giving public health nurses the opportunity to be part of a collaborative team effort improving oral health. The Indian Health Services Early Childhood Caries Initiative involves multiple stakeholders, including dental and medical staff and other programs, such as the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC). WIC is a social program for low-income women, infants, and children up to 5 years of age to provide healthy foods and referrals [142].

ELDERLY PATIENTS

Numerous socioeconomic issues can present obstacles for patients of any age who wish to obtain dental care. However, geriatric patients may experience additional barriers in their attempts to maintain dental health.

Financing of dental care is the primary obstacle for many older adults. Most patients older than 65 years of age are retired and therefore no longer have dental insurance as an employee benefit. Without this option and income limited to retirement savings, social security income, and any pension plan benefits, the costs associated with dental treatment may not be easily accommodated. Funding from federal, state, and county sources is often limited, both in available funds and treatment coverage.

Available financial resources among the geriatric population vary considerably. Unfortunately, many older adults live near or even below the poverty level and have difficulty in affording basic preventive dental care.

Medical problems can also present a major obstacle in the provision of dental care for geriatric patients. As discussed, many older adults are afflicted with at least one chronic disease and most have experienced medical problems. Even with Medicare insurance, the cumulative costs of medical treatment and medications can escalate and contribute to budgetary concerns, making it difficult to afford dental care.

Coping with serious medical problems may leave older adults without the motivation and ability to seek dental care. Some medical problems may also lead to one spouse assuming the role of caretaker for the other. If this is the case, both can have difficulties in obtaining dental care. The caregiver spouse may have difficulty setting aside time for a dental appointment, while the morbidity of the medical problem and transport issues make dental appointments difficult for the infirmed. Patients in long-term care facilities may also face obstacles in obtaining dental care [143]. The cost of long-term care is often a strain and may limit patients' ability to afford dental treatment. Difficulties with transportation, especially to an outside dental office, may also be a barrier to seeking dental care. In order to overcome this barrier, some long-term care facilities may contract with a private dentist to provide care within the facility. However, the fees associated with this level of service are prohibitive to many.

While there are other barriers in the provision of dental treatment of geriatric patients, including the availability of clinicians specializing in treating older patients, financial and transportation issues are the most frequently encountered. Because oral health is such an important component to overall health and quality of life, efforts should be made toward the improvement of access to dental care for all within the geriatric population.

CONCLUSION

This course has presented several controversial issues that arise in dental practice. In some cases, these can be divisive issues, but ideally, they can be a means of beginning and maintaining progress toward long-term solutions and improved care and patient education. Clinicians who are well-informed about these multifaceted issues will be in the best position to inform patients and to provide treatment that will enhance their quality of their life.

Works Cited

1. Ring ME. *Dentistry: An Illustrated History*. New York, NY: Abrams Books; 1992.
2. Campbell JM. *Dentistry Then and Now*. Glasgow, UK: Privately printed; 1981.
3. Bharti R. Wadhwani KK, Tikku AP, Chandra A. Dental amalgam: an update. *J Conserv Dent*. 2010;13(4):204-208.
4. Letzel H, van 't Hof MA, Marshall GW, Marshall SJ. The influence of the amalgam alloy on the survival of amalgam restorations: a secondary analysis of multiple controlled clinical trials. *J Dent Res*. 1997;76(11):1787-1798.
5. Ritter AV, Boushell LW, Walter R. *Sturdevant's Art and Science of Operative Dentistry*. 7th ed. St. Louis, MO: Elsevier; 2019.
6. California Dental Association. Dental Amalgam: Public Health and the Environment. Available at https://www.cda.org/Portals/0/pdfs/policy_statements/issue_amalgam.pdf. Last accessed January 10, 2023.
7. Colgate. Dental Amalgam: Is It Safe? Available at https://www.colgate.com/en-us/oral-health/fillings/dental-amalgam-a-health-risk#. Last accessed January 10, 2023.
8. Rathore M, Singh A, Pant VA. The dental amalgam toxicity fear: a myth or actuality. *Toxicol Int*. 2012;19(2):81-88.
9. Designs for Dental Health. Mercury Amalgam Fillings Banned. Available at http://designsfordentalhealth.com/nadler-blog/mercury-amalgam-fillings-banned. Last accessed January 10, 2023.
10. U.S. Food and Drug Administration. White Paper: FDA Update/Review of Potential Adverse Health Risks Associated with Exposure to Mercury in Dental Amalgam. Available at https://www.fda.gov/medical-devices/dental-amalgam/white-paper-fda-updatereview-potential-adverse-health-risks-associated-exposure-mercury-dental. Last accessed January 10, 2023.
11. U.S. Department of Health and Human Services Public Health Service. *Dental Amalgam: A Scientific Review and Recommended Public Health Service Strategy for Research, Education and Regulation*. Rockville, MD: Public Health Service; 1993.
12. Life Sciences Research Organization. Review and Analysis of the Literature on the Potential Health Effects of Dental Amalgams. Available at http://www.lsro.org/amalgam/frames_amalgam_home.html. Last accessed January 10, 2023.
13. Federal Register. Dental Devices: Classification of Dental Amalgam, Reclassification of Dental Mercury, Designation of Special Controls for Dental Amalgam, Mercury, and Amalgam Alloy; Technical Amendment: A Rule by the Food and Drug Administration. Available at https://www.federalregister.gov/documents/2010/06/11/2010-14083/dental-devices-classification-of-dental-amalgam-reclassification-of-dental-mercury-designation-of. Last accessed January 10, 2023.
14. U.S. Food and Drug Administration. FDA Issues Recommendations for Certain High-Risk Groups Regarding Mercury-Containing Dental Amalgam. Available at https://www.fda.gov/news-events/press-announcements/fda-issues-recommendations-certain-high-risk-groups-regarding-mercury-containing-dental-amalgam. Last accessed January 10, 2023.
15. European Commission Scientific Committee on Emerging and Newly Identified Health Risks. The Safety of Dental Amalgam and Alternative Dental Restoration Materials for Patients and Users. Available at https://ec.europa.eu/health/archive/ph_risk/committees/04_scenihr/docs/scenihr_o_016.pdf. Last accessed January 10, 2023.
16. U.S. Food and Drug Administration. Center for Devices and Radiological Health. Epidemiological Evidence on the Adverse Health Effects Reported in Relation to Mercury from Dental Amalgam: Systematic Literature Review (2010-Present). Available at https://www.fda.gov/media/131151/download. Last accessed January 10, 2023.
17. Murthy VH. Community water fluoridation: one of CDC's "10 Great Public Health Achievements of the 20th Century." *Pub Health Rep*. 2015;130(4):296-298.
18. Centers for Disease Control and Prevention. Community Water Fluoridation: 2018 Fluoridation Statistics. Available at https://www.cdc.gov/fluoridation/statistics/2018stats.htm. Last accessed January 10, 2023.
19. American Academy of Pediatric Dentistry. Fluoride Therapy. Available at https://www.aapd.org/globalassets/media/policies_guidelines/bp_fluoridetherapy.pdf. Last accessed January 10, 2023.
20. U.S. Department of Health and Human Services Federal Panel on Community Water Fluoridation. U.S. Public Health Service recommendation for fluoride concentration in drinking water for the prevention of dental caries. *Public Health Rep*. 2015;130(4):318-331.
21. Centers for Disease Control and Prevention. Community Water Fluoridation FAQs. Available at https://www.cdc.gov/fluoridation/faqs/index.htm. Last accessed January 10, 2023.
22. National Research Council, Division on Earth and Life Studies, Board on Environmental Studies and Toxicology, Committee on Fluoride in Drinking Water. *Fluoride in Drinking Water: A Scientific Review of EPA's Standards*. Washington, DC: National Academies Press; 2007.
23. New Hampshire Department of Environmental Services. Environmental Fact Sheet: Fluoride in Drinking Water. Available at https://www.des.nh.gov/sites/g/files/ehbemt341/files/documents/2020-01/dwgb-3-5.pdf. Last accessed January 10, 2023.
24. World Health Organization. Drinking Water. Available at https://www.who.int/news-room/fact-sheets/detail/drinking-water. Last accessed January 10, 2023.
25. Beltrán-Aguilar ED, Barker L, Dye BA. NCHS data brief No. 53: prevalence and severity of dental fluorosis in the United States, 1999–2004. *NCHS Data Brief*. 2010;(53):1-8.

26. Murthy VH. Surgeon general's perspectives. *Public Health Rep*. 2015;130(4):296-298.
27. Centers for Disease Control and Prevention. Statement on the Evidence Supporting the Safety and Effectiveness of Community Water Fluoridation. Available at https://www.cdc.gov/fluoridation/guidelines/cdc-statement-on-community-water-fluoridation.html. Last accessed January 10, 2023.
28. Green R, Lanphear B, Hornung R, et al. Association between maternal fluoride exposure during pregnancy and IQ scores in offspring in Canada. *JAMA Pediatr*. 2019;173(10):940-948.
29. Bashash M, Thomas D, Hu H, et al. Prenatal fluoride exposure and cognitive outcomes in children at 4 and 6–12 years of age in Mexico. *Environ Health Perspect*. 2017;125(9):097017.
30. Das K, Mondal NK. Dental fluorosis and urinary fluoride concentration as a reflection of fluoride exposure and its impact on IQ level and BMI of children of Laxmisagar, Simlapal Block of Bankura District, W.B., India. *Environ Monit Assess*. 2016;188(4):218.
31. Till C, Green R. Controversy: the evolving science of fluoride: when new evidence doesn't conform with existing beliefs. *Pediatr Res*. 2021;90(5):1093-1095.
32. Grandjean P. Developmental fluoride neurotoxicity: an updated review. *Environ Health*. 2019;18(1):110.
33. Till C, Green R, Flora D, et al. Fluoride exposure from infant formula and child IQ in a Canadian birth cohort. *Environ Int*. 2020;134:105315.
34. Jenco M. AAP Continues to Recommend Fluoride Following New Study on Maternal Intake and Child IQ. Available at https://publications.aap.org/aapnews/news/6598?autologincheck=redirected. Last accessed January 10, 2023.
35. American Dental Association. News Releases: ADA Statement on Study in JAMA Pediatrics. Available at https://health.mo.gov/living/families/oralhealth/pdf/ada-study-jama-pediatrics.pdf. Last accessed January 10, 2023.
36. Public Broadcasting Service. Fleming Discovers Penicillin 1928–1945. Available at http://www.pbs.org/wgbh/aso/databank/entries/dm28pe.html. Last accessed January 10, 2023.
37. Dar-Odeh NS, Abu-Hammad OA, Al-Omiri MK, Khraisat AS, Shehabi AA. Antibiotic prescribing practices by dentists: a review. *Ther Clin Risk Manag*. 2010;6:301-306.
38. Hellden LB, Listgarten MA, Lindhe J. The effect of tetracycline and/or scaling on human periodontal disease. *J Clin Periodontol*. 1979;6(4):222-230.
39. Slots J, Mashimo P, Levine MJ, Genco RJ. Periodontal therapy in humans. I. Microbiological and clinical effects of a single course of periodontal scaling and root planing, and of adjunctive tetracycline therapy. *J Periodontol*. 1979;50(10):495-509.
40. Bollen CM, Quirynen M. Microbiological response to mechanical treatment in combination with adjunctive therapy: a review of the literature. *J Periodontol*. 1996;67(11):1143-1158.
41. Silva-Senem MX, Heller D, Varela VM, Torres MCB, Feres-Filho EJ, Colombo APV. Clinical and microbiological effects of systemic antimicrobials combined to an anti-infective mechanical debridement for the management of aggressive periodontitis: a 12-month randomized controlled trial. *J Clin Periodontol*. 2013;40(3):242-251.
42. Hagenfeld D, Koch R, Junemann S, et al. Do we treat our patients or rather periodontal microbes with adjunctive antibiotics in periodontal therapy? A 16S rDNA microbial community analysis. *PLoS One*. 2018;13(4):e0195534.
43. European Federation of Periodontology. Debate: Is it Time for a Rethink on the Use of Antibiotics to Treat Periodontitis? Available at https://www.efp.org/news-events/news/debate-is-it-time-for-a-rethink-on-the-use-of-antibiotics-to-treat-periodontitis-29978/. Last accessed January 10, 2023.
44. Walker C. The supplemental use of antibiotics in periodontal therapy. *Compend Contin Educ Dent*. 1999;20(4):4-12.
45. Kapoor A, Malhotra R, Grover V, Grover D. Systemic antibiotic therapy in periodontics. *Dent Res J (Isfahan)*. 2012;9(5):505-515.
46. Rajendra A, Spivakovsky S. Antibiotics in aggressive periodontitis: is there a clinical benefit? *Evid Based Dent*. 2016;17(4):100.
47. Barca E, Cifcibasi E, Cintan S. Adjunctive use of antibiotics in periodontal therapy. *J Istanb Univ Fac Dent*. 2015;49(3):55-62.
48. Salako NO, Rotimi VO, Adib SM, Al-Mutawa S. Pattern of antibiotic prescription in the management of oral diseases among dentists in Kuwait. *J Dent*. 2004;32(7):503-509.
49. Oomens MA, Forouzanfar T. Antibiotic prophylaxis in third molar surgery: a review. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 2012;114(6):e5-e12.
50. Lacasse A, Rey E, Ferreira E, Morin C, Bérard A. Epidemiology of nausea and vomiting of pregnancy: prevalence, severity, determinants, and the importance of race/ethnicity. *BMC Pregnancy Childbirth*. 2009;9:26.
51. Little JW, Miller C, Rhodus NL. *Little and Falace's Dental Management of the Medically Compromised Patient*. 9th ed. St. Louis, MO: Elsevier; 2018.
52. Merck Manual. Drugs in Pregnancy. Available at https://www.merckmanuals.com/professional/gynecology-and-obstetrics/drugs-in-pregnancy/drugs-in-pregnancy. Last accessed January 10, 2023.
53. Federal Register. Content and Format of Labeling for Human Prescription Drug and Biological Products: Requirements for Pregnancy and Lactation Labeling. Available at https://www.federalregister.gov/articles/2014/12/04/2014-28241/content-and-format-of-labeling-for-human-prescription-drug-and-biological-products-requirements-for. Last accessed January 10, 2023.

54. Gaffney A. FDA Scraps Pregnancy Labeling Classification System in Favor of New Standard. Available at https://www.raps.org/regulatory-focus%e2%84%a2/news-articles/2014/12/fda-scraps-pregnancy-labeling-classification-system-in-favor-of-new-standard. Last accessed January 10, 2023.
55. U.S. Food and Drug Administration. Pregnancy and Lactation Labeling Final Rule. Available at https://www.fda.gov/drugs/labeling-information-drug-products/pregnancy-and-lactation-labeling-drugs-final-rule. Last accessed January 10, 2023.
56. U.S. Food and Drug Administration. FDA Issues Final Rule on Changes to Pregnancy and Lactation Labeling Information for Prescription Drug and Biological Products. Available at https://wayback.archive-it.org/7993/20170111093956/http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm425317.htm. Last accessed January 10, 2023.
57. Kurien S, Kattimani VS, Sriram RR, et al. Management of pregnant patient in dentistry. *J Int Oral Health*. 2013;5(1):88-97.
58. American Dental Association Council on Access, Prevention, and Interprofessional Relations. Women's oral health issues. In: *ADA Oral Health Care Series*. Chicago, IL: American Dental Association; 1995: 9.
59. Patton LL, Glick M. *The ADA Practical Guide to Patients with Medical Conditions*. 2nd ed. Hoboken, NJ: Wiley Blackwell; 2016.
60. Kornman KS, Loesche WJ. The subgingival microbial flora during pregnancy. *J Periodont Res*. 1980;15(2):111-122.
61. Carrillo-de-Albornoz A, Figuero E, Herrera D, Bascones-Martinez A. Gingival changes during pregnancy: II. Influence of hormonal variations on the subgingival biofilm. *J Clin Periodontol*. 2010;37(3):230-240.
62. Markou E, Eleana B, Lazaros T, Antonios K. The influence of sex steroid hormones on gingiva of women. *Open Dent J*. 2009;3:114-119.
63. Lindhe J, Branemark PI. Changes in vascular permeability after local application of sex hormones. *J Periodontol Res*. 1967;2(4):259-265.
64. Mariotti A. Sex steroid hormones and cell dynamics in the periodontium. *Crit Rev Oral Biol Med*. 1994;5(1):27-53.
65. Soory M. Targets for steroid hormone mediated actions of periodontal pathogens, cytokines and therapeutic agents: some implications on tissue turnover in the periodontium. *Curr Drug Targets*. 2000;1(4):309-325.
66. Barnett ML. The oral-systemic disease connection: an update for the practicing dentist. *J Am Dent Assoc*. 2006;137:5S-6S.
67. Mattila KJ, Nieminen MS, Valtonen VV, et al. Association between dental health and acute myocardial infarction. *BMJ*. 1989;298(6676):779-781.
68. Chang Y, Woo HG, Lee JS, Song TJ. Better oral hygiene is associated with lower risk of stroke. *J Periodontol*. 2021;92(1):87-94.
69. Carrizales-Sepúlveda EF, Ordaz-Farías A, Vera-Pineda R, Flores-Ramírez R. Periodontal disease systemic inflammation and the risk of cardiovascular disease. *Heart Lung Circ*. 2018;27(11):1327-1334.
70. Priyamvara A, Dey AK, Bandyopadhyay D, et al. Periodontal inflammation and the risk of cardiovascular disease. *Curr Atheroscler Rep*. 2020;22(7):28.
71. Sanz M, Del Castillo AM, Jepsen S, et al. Periodontitis and cardiovascular diseases: consensus report. *J Clin Periodontol*. 2020;47(3):268-288.
72. Lam OL, Zhang W, Samaranayake LP, Li LS, McGrath C. A systematic review of the effectiveness of oral health promotion activities among patients with cardiovascular disease. *Int J Cardiol*. 2011;151(3):261-267.
73. Bui FQ, Almeida-da-Silva CLC, Huynh B, et al. Association between periodontal pathogens and systemic disease. *Biomed J*. 2019;42(1):27-35.
74. Joshipura K, Zevallos JC, Ritchie CS. Strength of evidence relating to periodontal disease and atherosclerotic disease. *Compend Contin Educ Dent*. 2009;30(7):430-439.
75. Haraszthy VI, Zambon JJ, Trevisan M, Zeid M, Genco RJ. Identification of periodontal pathogens in atheromatous plaques. *J Periodontol*. 2000;71(10):1554-1560.
76. Herzberg MC, Meyer MW. Effects of oral flora on platelets: possible consequences in cardiovascular disease. *J Periodontol*. 1996;67(10):1138-1142.
77. Seymour GJ, Ford PJ, Cullinan MP, Leishman S, Yamazaki K. Relationship between periodontal infections and systemic disease. *Clin Microbiol Infect*. 2007;13(4):3-10.
78. Issrani R, Reddy J, El-Metwally Dabah TH, et al. Exploring the mechanisms and association between oral microflora and systemic diseases. *Diagnostics (Basel)*. 2022;12(11):2800.
79. Sanz M, Ceriello A, Buysschaert M, et al. Scientific evidence on the links between periodontal diseases and diabetes: consensus report and guidelines of the joint workshop on periodontal diseases and diabetes by the International Diabetes Federation and the European Federation of Periodontology. *J Clin Periodontol*. 2018;45(2):138-149.
80. Fong IW. Infections and their role in atherosclerotic vascular disease. *J Am Dent Assoc*. 2002;133:7S-13S.
81. Meisel P, Kohlmann T, Wallaschofski H, Kroemer HK, Kocher T. Cholesterol, c-reactive protein, and periodontitis: HMG-CoA reductase inhibitors (statins) as effect modifiers. *ISRN Dent*. 2011;2011:125168.
82. Ridker PM. Evaluating novel cardiovascular risk factors: can we better predict heart attacks? *Ann Intern Med*. 1999;130(11):933-937.
83. D'Aiuto F, Parkar M, Andreou G, et al. Periodontitis and systemic inflammation: control of the local infection is associated with a reduction in serum inflammatory markers. *J Dent Res*. 2004;83(2):156-160.
84. Lalla E, Cheng B, Lal S, et al. Periodontal changes in children and adolescents with diabetes. Diabetes Care. 2006;29(2):295-299.

85. American Academy of Periodontology. Gum Disease and Other Diseases: Diabetes. Available at https://www.perio.org/for-patients/gum-disease-information/gum-disease-and-other-diseases/. Last accessed January 10, 2023.
86. Preshaw PM, Alba AL, Herrera D, et al. Periodontitis and diabetes: a two-way relationship. *Diabetologia*. 2012;55(1):21-31.
87. Shinkai RS, Hatch JP, Cornell JE, Yeh CK. Intraoral tactile sensitivity in adults with diabetes. *Diabetes Care*. 2004;27(4):869-873.
88. Mealey BL, Oates TW. Diabetes mellitus and periodontal disease. *J Periodontol*. 2006;77(8):1289-303.
89. Southerland JH, Taylor GW, Offenbacher S. Diabetes and periodontal infection: making the connection. *Clin Diabetes*. 2005;23(4):171-178.
90. Westerfield J, Holcomb S, Jensen S (eds). *Current Trends in Diabetes Management: A Guide for the Healthcare Professional*. 7th ed. Nashville, TN: Healthways; 2005.
91. Lorber D. Macrovascular disease in diabetes. In: Mensing C (ed). *The Art and Science of Diabetes Self-Management Education: Desk Reference*. 3rd ed. Chicago, IL: American Association of Diabetes Educators; 2014.
92. Cummings KM. Programs and policies to discourage the use of tobacco products. *Oncogene*. 2002;21(48):7349-7364.
93. U.S. Department of Health, Education, and Welfare. Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service. 1964. Available at https://www.govinfo.gov/content/pkg/GPO-SMOKINGANDHEALTH/pdf/GPO-SMOKINGANDHEALTH.pdf. Last accessed January 10, 2023.
94. Centers for Disease Control and Prevention. Burden of Cigarette Use in the U.S. Available at https://www.cdc.gov/tobacco/campaign/tips/resources/data/cigarette-smoking-in-united-states.html. Last accessed January 10, 2023.
95. Centers for Disease Control and Prevention. Smoking and Tobacco Use: Fast Facts and Fact Sheets. Available at https://www.cdc.gov/tobacco/data_statistics/fact_sheets/fast_facts/. Last accessed January 10, 2023.
96. Centers for Disease Control and Prevention. Current cigarette smoking among adults—United States, 2016. *MMWR*. 2018;67(2):54-59.
97. Wang TW, Asman K, Gentzke AS, et al. Tobacco product use among adults—United States, 2017. *MMWR*. 2018;67(44):1225-1232.
98. Centers for Disease Control and Prevention. Smokeless Tobacco Use in the United States. Available at https://www.cdc.gov/tobacco/data_statistics/fact_sheets/smokeless/use_us/index.htm. Last accessed January 10, 2023.
99. Lipari RN, Van Horn SL. *Trends in Smokeless Tobacco Use and Initiation: 2002 to 2014*. Rockville, MD: Center for Behavioral Health Statistics and Quality, Substance Abuse and Mental Health Services Administration; 2017.
100. Centers for Disease Control and Prevention. Smokeless Tobacco: Products and Marketing. Available at https://www.cdc.gov/tobacco/data_statistics/fact_sheets/smokeless/products_marketing/index.htm. Last accessed January 10, 2023.
101. Maxwell JC. The Maxwell Report: The Smokeless Tobacco Industry in 2011. Richmond, VA: John Maxwell, Jr.; 2011
102. Centers for Disease Control and Prevention. Smokeless Tobacco Product Use in the United States. Available at https://www.cdc.gov/tobacco/data_statistics/fact_sheets/smokeless/use_us/. Last accessed January 10, 2023.
103. Benowitz NL. Neurobiology of nicotine addiction: implications for smoking cessation treatment. *Am J Med*. 2008;121(4 Suppl 1):S3-S10.
104. Johns Hopkins Medicine. Oral Cancer and Tobacco. Available at https://www.hopkinsmedicine.org/health/conditions-and-diseases/oral-cancer-and-tobacco#. Last accessed January 10, 2023.
105. Centers for Disease Control and Prevention. About Electronic Cigarettes (E-Cigarettes). Available at https://www.cdc.gov/tobacco/basic_information/e-cigarettes/about-e-cigarettes.html. Last accessed January 10, 2023.
106. National Institute on Drug Abuse. Vaping Devices (Electronic Cigarettes) DrugFacts. Available at https://nida.nih.gov/publications/drugfacts/vaping-devices-electronic-cigarettes. Last accessed January 10, 2023.
107. Sun R, Mendez D, Warner KE. Trends in nicotine product use among US adolescents, 1999-2020. *JAMA Netw Open*. 2021;4(8):e2118788.
108. U.S. Food and Drug Administration. FDA Takes New Steps to Address Epidemic of Youth E-Cigarette Use, Including a Historic Action Against More Than 1,300 Retailers and 5 Major Manufacturers for Their Roles Perpetuating Youth Access. Available at https://www.fda.gov/news-events/press-announcements/fda-takes-new-steps-address-epidemic-youth-e-cigarette-use-including-historic-action-against-more. Last accessed January 10, 2023.
109. U.S. Food and Drug Administration. FDA Finalized Enforcement Policy on Unauthorized Flavored Cartridge-Based E-Cigarettes that Appeal to Children, Including Fruit and Mint. Available at https://www.fda.gov/news-events/press-announcements/fda-finalizes-enforcement-policy-unauthorized-flavored-cartridge-based-e-cigarettes-appeal-children. Last accessed January 10, 2023.
110. Altschuler C. Dangerous liaisons: the hazardous attraction of e-cigarettes, hookahs, and smokeless tobacco products. *AGD Impact*. 2015;43(5):18-23.
111. Knishkowy B, Amitai Y. Water-pipe (narghile) smoking: an emerging health risk behavior. *Pediatrics*. 2005;116(1):e113-e119.
112. Kiter G, Uçan ES, Ceylan E, Kilinç O. Water-pipe smoking and pulmonary functions. *Respir Med*. 2000;94(9):891-894.
113. WHO Study Group on Tobacco Product Regulation. Advisory Note: Waterpipe Tobacco Smoking: Health Effects, Research Needs and Recommended Actions by Regulators. Available at https://apps.who.int/iris/bitstream/handle/10665/43378/9241593857_eng.pdf?sequence=1. Last accessed January 10, 2023.

114. Ward KD, Eissenberg T, Gray JN, Srinivas V, Wilson N, Maziak W. Characteristics of U.S. waterpipe users: a preliminary report. *Nicotine Tob Res*. 2007;9(12):1339-1346.

115. U.S. Food and Drug Administration. Hookah Tobacco (Shisha or Waterpipe Tobacco). Available at https://www.fda.gov/tobacco-products/products-ingredients-components/hookah-tobacco-shisha-or-waterpipe-tobacco#references. Last accessed January 10, 2023.

116. Sepetdjian E, Shihadeh A, Saliba NA. Measurement of 16 polycyclic aromatic hydrocarbons in narghile waterpipe tobacco smoke. *Food Chem Toxicol*. 2008;46(5):1582-1590.

117. Saleh R, Shihadeh A. Elevated toxicant yields with narghile waterpipes smoked using a plastic hose. *Food Chem Toxicol*. 2008;46(5):1461-1466.

118. Lewin T. Collegians smoking hookahs...filled with tobacco. *The New York Times*. April 19, 2006.

119. Wahl MJ. Myths of dental surgery in patients receiving anticoagulant therapy. *J Am Dent Assoc*. 2000;131(1):77-81.

120. Jiménez Y, Poveda R, Gavaldá C, Margaix M, Sarrión G. An update on the management of anticoagulated patients for dental extractions and surgery. *Med Oral Patol Oral Cir Bucal*. 2008;13(3):E176-E179.

121. Dennis MJ. Anticoagulant therapy and dentistry. *Today's FDA*. 2012;24(6):40-41, 43-47.

122. Hupp JR. Prevention and management of surgical complications. In: Hupp JR, Ellis E, Tucker MR (eds). *Contemporary Oral and Maxillofacial Surgery*. 5th ed. St. Louis, MO: Mosby Elsevier; 2008: 185-200.

123. ADA. Oral Anticoagulant and Antiplatelet Medications and Dental procedures. Available at https://www.ada.org/resources/research/science-and-research-institute/oral-health-topics/oral-anticoagulant-and-antiplatelet-medications-and-dental-procedures. Last accessed January 10, 2023.

124. Mingarro-de-León A, Chaveli-López B, Gavaldá-Esteve C. Dental management of patients receiving anticoagulant and/or antiplatelet treatment. *J Clin Exp Dent*. 2014;6(2):e155-e161.

125. Al-Harkan AM, Al-Ayoub GA. Should antiplatelet and anticoagulant medications be discontinued before minor oral surgery procedures? *J Can Dent Assoc*. 2012;78:c24.

126. Little JW, Miller CS, Henry RG, McIntosh BA. Antithrombotic agents: implications in dentistry. *Oral Surg Oral Med Oral Pathol Radiol Endod*. 2002;93(5):544-551.

127. Grines CL, Bonow RO, Casey DE, et al. Prevention of premature discontinuation of dual antiplatelet therapy in patients with coronary artery stents: a science advisory from the American Heart Association, American College of Cardiology, Society for Cardiovascular Angiography and Interventions, American College of Surgeons, and American Dental Association, with representation from the American College of Physicians. *Circulation*. 2007;115(6):813-818.

128. The American Academy or Oral medicine. Anticoagulant and Antiplatelet Medications and Dental Procedures. Available at https://www.aaom.com/index.php?option=com_content&view=article&id=80:antiplatelet-and-anticoagulant-agents-and-dental-procedures&catid=22:patient-condition-information&Itemid=120. Last accessed January 10, 2023.

129. Wynn RL, Meiller TF, Crossley HL. *Drug Information Handbook for Dentistry*. 23rd ed. Hudson, OH: LexiComp; 2017.

130. National Advisory Committee on Rural Health and Human Services. Improving Oral Health Care Services in Rural America. Available at https://www.hrsa.gov/sites/default/files/hrsa/advisory-committees/rural/2018-oral-health-policy-brief.pdf. Last accessed January 10, 2023.

131. National Rural Health Association. Improving Rural Oral Healthcare Access. Available at https://www.ruralhealthweb.org/NRHA/media/Emerge_NRHA/Advocacy/Policy%20documents/05-11-18-NRHA-Policy-Improving-Rural-Oral-Health-Access.pdf. Last accessed January 10, 2023.

132. Health Resources and Services Administration. What is Shortage Designation? Available at https://bhw.hrsa.gov/workforce-shortage-areas/shortage-designation. Last accessed January 10, 2023.

133. Health Resources and Services Administration. Health Workforce Shortage Areas. Available at https://data.hrsa.gov/topics/health-workforce/shortage-areas. Last accessed January 10, 2023.

134. Agency for Healthcare Research and Quality. National Healthcare Quality and Disparities Report. Available at https://www.ahrq.gov/research/findings/nhqrdr/index.html. Last accessed January 10, 2023.

135. National Conference of State Legislatures. Improving Access to Care in Rural and Underserved Communities: State Workforce Strategies Health. Available at http://www.ncsl.org/research/health/improving-access-to-care-in-rural-and-underserved-communities-state-workforce-strategies.aspx. Last accessed January 10, 2023.

136. Minnesota Department of Health. Emerging Health Professions. Available at https://www.health.state.mn.us/facilities/ruralhealth/emerging/index.html. Last accessed January 10, 2023.

137. PEW Charitable Trusts. Expanding the Dental Team Increasing Access to Care in Public Settings. Available at https://www.pewtrusts.org/en/research-and-analysis/reports/2014/06/30/expanding-the-dental-team. Last accessed January 10, 2023.

138. Garg S, Rubin T, Jasek J, Weinstein J, Helburn L, Kaye K. How willing are dentists to treat young children? A survey of dentists affiliated with Medicaid managed care in New York City, 2010. *J Am Dent Assoc*. 2013;144(4):416-425.

139. New York State Department of Health. Oral Health Plan for New York State. Available at https://www.health.ny.gov/prevention/dental/docs/oral_health_plan.pdf. Last accessed January 10, 2023.

140. PEW. Children's Dental Health Disparities. Available at https://www.pewtrusts.org/en/research-and-analysis/articles/2016/02/16/childrens-dental-health-disparities. Last accessed January 10, 2023.

141. Indian Health Service Data Brief April 2019. The Oral Health of American Indian and Alaska Native Children Aged 1–5 Years: Results of the 2018-19 IHS Oral Health Survey. Available at https://www.ihs.gov/doh/documents/surveillance/2018-19%20Data%20Brief%20of%201-5%20Year-Old%20AI-AN%20Preschool%20Children.pdf. Last accessed January 10, 2023.

142. Indian Health Service. The Public Health Nurse's Role in ECC Prevention and Early Intervention. Available at https://www.ihs.gov/doh/documents/ecc/CommunityDocs/PHNRoleECCPreventionAndEarlyIntervention.pdf. Last accessed January 10, 2023.

143. Stein PS, Henry RG. Poor oral hygiene in long-term care. *Am J Nurs*. 2009;109(6):44-50.